INTRODUCTION:
Hypertension is highly prevalent among aging adults and can lead to coronary artery disease, diastolic and systolic heart failure and other cardiovascular conditions. Hypertension is also known to worsen clinical heart failure since high afterload increases the work of the heart, prompting a maladaptive state that is reflected by congestion, fatigue and edema.

CASE PRESENTATION:
• **Background:** Mr. S is an 80 year old African American with a history of smoking (current), ETOH (claims he no longer drinks), epileptic seizures from a right fronto-temporal region (for approximately 4 years) and a history of heart failure with preserved ejection fraction. Ejection fraction (EF) by echocardiography was 50% when diagnosed in January 2003; presumably from hypertension. Echocardiogram on this visit revealed left ventricular hypertrophy and abnormal relaxation [stage 1], left ventricular end-systolic and end-diastolic diameters of 2.6 cm and 3.6 cm, respectively (normal), EF 50%, 3+ tricuspid regurgitation (TR), and right ventricular systolic pressure (RVSP) 38 mm Hg (mild pulmonary hypertension). He has “hip problems”, so he uses a wheelchair to get from the parking garage to the clinic area. He is being seen in the outpatient department today by a Clinical Nurse Specialist. He was last seen 6 weeks ago and has not been hospitalized or treated in an emergency department setting since his last visit. He states that his breathing ability and energy level are similar to his last visit. He can climb one flight (14 steps) before becoming dyspneic. He has 2 pillow orthopnea but no paroxysmal nocturnal dyspnea. Mr. S. has fatigue with activities of daily living. He denies chest pain, palpitations, lightheadedness, dizziness, nausea, edema, or difficulties with eating (states his appetite is fair). He follows a low sodium diet. Mr. S. has no known allergies. He is accompanied by a family member to his visit.

• **Physical exam:** alert, cooperative, no jugular venous distension, no hepatojugular reflux, no peripheral edema. Lungs clear, abdomen is soft and nontender. Heart rate and rhythm are regular, has a 2/6 systolic murmur at the left lower sternal border.

• BP, 142/64 mm Hg; pulse, 64 bpm; weight, 132 lbs (60 kg); NYHA FC II-III.

• Most recent echocardiogram (6 months later): EF, 55%, 2+ TR, right ventricular systolic pressure (RVSP) 42 mm Hg (mild pulmonary hypertension). Left ventricular hypertrophy and abnormal relaxation (Stage 1). Left ventricular end-systolic and end-diastolic diameters are 2.5 cm and 3.6 cm, respectively (normal).
Case Study: Hypertension

• Medications:
  - metoprolol succinate (Toprol XL) 150 mg/daily
  - paroxetine (Paxil) 10 mg/daily
  - levetiracetam (Keppra) 500 mg/ twice daily
  - amlodipine (Norvasc) 10 mg/daily
  - ramipril (Altace) 10 mg/ twice daily
  - aspirin 325 mg/daily
  - Magnesium oxide (Mag-Ox) 400 mg/ twice daily
  - gabapentin (Neurotin) 300 mg/ twice daily

• Serum laboratory history:
  - Sodium, 136 mmol/L
  - Potassium, 4.7 mmol/L
  - BUN, 22 mg/dL
  - creatinine, 1.3 mg/dL
  - total cholesterol, 174 mg/dL; HDL, 62 mg/dL; LDL, 93 mg/dL; triglyceride, 96 mg/dL

• Plan: Blood pressure is suboptimal. Will add hydrochlorothiazide 25 mg/daily to reduce systolic blood pressure.

• Outcome at 4 weeks: At next visit, NYHA FC was reduced to II; less fatigue with activities and now sleeping on one pillow. BP, 102/50 mm Hg. Eating better; has gained 1 kg (now 61 kg). Current echocardiogram with this visit shows RVSP down to 38 mm Hg. No complaints of dizziness or lightheadedness. Treatment regime maintained.

• Serum laboratory at 4 weeks:
  - Sodium, 143 mmol/L
  - Potassium, 4.3 mmol/L
  - BUN, 22 mg/dL
  - creatinine, 1.3 mg/dL
SUMMARY:

Even though Mr. S’s EF is normal, he has clinical heart failure based on echocardiogram history and symptoms of dyspnea and fatigue. Additionally, his serum sodium level is reduced, reflecting hypervolemia and neurohormonal activation. Mr. S benefited from tweaking of his medications to bring his blood pressure into better control. While his diastolic blood pressure was acceptable, his elevated systolic blood pressure was increasing afterload and backflow pressure, leading to mild pulmonary hypertension secondary to his heart failure. Hydrochlorothiazide is a mild distal tubule diuretic that has shown to be effective in decreasing blood pressure in hypertension. As Mr. S did not have evidence of hypervolemia and does not have systolic left ventricular dysfunction, there was no reason to initiate drug therapy with a loop diuretic agent. Oftentimes, patients with hypertension require more than 1 agent to decrease blood pressure adequately. Mr. S is an example of the need for multiple agents to control his co-morbid condition of heart failure and hypertension. He is receiving an angiotensin converting enzyme, calcium channel blocker, distal tubule diuretic and a beta-blocker. This combination led to better control of hypertension and a reduction in clinical symptoms of heart failure. Note: when adding an antihypertensive agent to a patient’s medical regimen, the systolic blood pressure generally decreases by < 10 mm Hg. In this case study, Mr. S’s systolic blood pressure went from 142 mm Hg to 102 mm Hg reflecting the benefit of thiazide diuretic in improving blood pressure control. His serum sodium level returned to within normal limits (from 136 to 143 mmol/L), reflecting better control of intravascular volume. Preload (including a drop in tricuspid regurgitation from 3+ to 2+) and afterload were markedly reduced and his systolic and diastolic pressures dropped to a greater extent than with vasodilator therapies alone (even though he was receiving different classes of antihypertensives).