INTRODUCTION:
Sleep disturbances are common patient complaints, and are associated with increased morbidity in Heart Failure (HF). Sleep disturbances have been documented in up to 60% of HF patients, and are often overlooked or unnoticed by clinicians. Thus detection of sleep disturbances should be an important component of clinical assessment, which can lead to appropriate treatment and dramatic improvements in HF patient outcome and quality of life.

PRESENTATION OF THE CASE:

Background Information:

▲ 49-year old, White male
▲ returns to the clinic for his regularly scheduled HF evaluation
▲ history of:
  • idiopathic, dilated cardiomyopathy
  • LVEF = 0.30 (no change from a year ago)
  • BMI = 29.4 (2 pound weight gain from 6 months ago)
  • no smoking history
  • sinus rhythm with rare to occasional PVCs
  • medications include ACE-inhibitor, digoxin, beta-blocker, diuretic, and a potassium supplement
  • employed as a night guard at a local shopping mall
▲ denies any new or increasing HF symptoms, sleep complaints, or changes in physical activity
▲ continues to sleep flat (1-pillow) at night
▲ physical exam findings:
  • S4 and grade III/VI mitral regurgitation murmur (not new)
  • Faint inspiratory crackles in the lung bases bilaterally
  • Patient exhibiting Cheyne-Stokes respiratory pattern while awake and listening to the clinician
  • Slightly irregular pulse rhythm
▲ Beck Depression Inventory (symptoms of depression) = 14 (depressed classified as ≥ 10)
**Differential Diagnoses:**

- Sleep disturbance – probably central sleep apnea (Cheyne-Stokes respiratory pattern, symptoms of depression) secondary to increased heart failure severity
- Sleep disturbance – possibly obstructive sleep apnea or mixed (both central and obstructive sleep apneas)
- Cardiac dysrhythmia – (irregular pulse rhythm and symptoms of depression)

**Labs/Confirmatory Evaluations:**

- EKG – sinus rhythm with occasional, unifocal PVCs, rare ventricular couplets
- Serum electrolytes – within normal limits
- Asked his wife about this patient’s sleep, she states that he “stops breathing a lot when he’s asleep.”
- Overnight polysomnography ordered – results are as follows:
  - Respiratory disturbance index (RDI) = 68 (normal < 5)
  - SaO2 nadir (lowest level of SaO2) = 73% (average SaO2 was 91%)
  - 22-70 second apneic periods (average of 59 apneic periods/hour on this 8-hour sleep study)
  - Bradycardia (heart rate down to 39/minute) with longer apneic periods (average heart rate while asleep during non-apneic periods was 56/minute)
  - Sleep study interpretation: mixed sleep apnea (both central and obstructive – Figure 1.)

**Review of Interventions/Treatments:**

- Bilevel Positive Airway Pressure (BiPAP) tried during polysomnography – started at lowest level, but patient could not tolerate mask and stated that his sleeping was better without the BiPAP device
- Increased diuretic dose – patient lost 11 pounds in 2 days
- Returned to clinic for evaluation in 5 days
  - inspiratory crackles gone
  - patient denies any change in his sleep status
  - clinician did not observe any Cheyne-Stokes respiratory pattern breathing by patient while in clinic
  - patient’s wife states that now patient does not stop breathing during sleep, although he occasionally snorts or gasps at night
Despite diuresis, repeat overnight polysomnography still shows evidence of obstructive sleep apnea (RDI = 18), with SaO2 nadir at 88%.

Further treatment with increased emphasis on sodium and fluid restrictions and weight loss (to a BMI of 27), resulted in a repeat overnight polysomnography within normal limits (RDI = 4), with 11 obstructive apneic events and an improvement of depressive symptoms (Beck Depression Inventory score = 8) after 6 months. Patient continued to deny any changes in his sleep quality, but did admit that his energy levels at work were much better.

**SUMMARY**

Sleep disorders can occur in the presence of stable or equivocal levels of heart failure. Clinical information on sleep disorders and sleep-disordered breathing should be gathered not only from the patient, but also from the patient’s sleeping partner and from direct clinician observation and assessment. While aggressive treatment of heart failure can be very effective at decreasing the incidence of central sleep apneas, it should not be assumed that obstructive sleep apneas will not persist. Therefore, evaluation of sleep-disordered breathing should be a part of routine clinical evaluation of this high-risk patient population.

**Panel A. Central Sleep Apnea**

Example of one of the patient’s central sleep apnea episodes. In the highlighted area, the top line is air flow (nasa), second line is chest movement, third line is abdominal movement, and the bottom line is SaO2. Note that there is minimal/no movement of the chest or abdomen in the highlighted area, and the SaO2 drops precipitously (to 98% to 63%). Apnea associated with minimal/no chest or abdominal wall movement is a hallmark of central sleep apnea.

**Panel B. Obstructive Sleep Apnea**

Example of one of the patient’s obstructive sleep apnea episodes. In the highlighted area, the top line is air flow (nasa), second line is chest movement, third line is abdominal movement, and the bottom line is SaO2. Note that there are attempts to breathe by the chest and abdomen in the highlighted area, with minimal airflow, and the SaO2 has dropped to 64% during this episode. Apneas associated with attempted movements of the chest and/or abdomen are hallmarks of obstructive sleep apnea.