QUESTION ANSWERS

Question #1:

- 1. What is the spirometric diagnosis demonstrated here?
 - a. Airway obstruction with significant but not complete reversibility
- 2. What conclusion can be drawn by the lack of complete responsiveness to bronchodilator?
 - a. The patient has ACO (Asthma-COPD overlap syndrome), not asthma
 - b. She likely had adult-onset asthma initially but developed asthma remodeling (change from eosinophilic inflammatory cascade to a neutrophilic inflammatory cascade over time)
- 3. How would the severity of her lung disease best be classified?
 - a. By the GOLD criteria, with "COPD" being confirmed with the FEV1/FVC ratio < 70% and "moderate" being determined by the FEV1 of 58% predicted
- 4. What are the mortality implications to be considered in this case?
 - a. She is female and as such has increased mortality in COPD. Careful review of potential comorbidities, particularly cardiovascular disease, is advised.

Question #2:

- 1. What is the spirometric diagnosis?
 - a. Restriction
- 2. Is further lung function testing indicated?
 - **a.** Yes measurements of lung volumes and lung diffusion
- 3. What diagnosis would these findings suggest?
 - a. Interstitial Lung Disease
- 4. What radiographic imaging would be suggested?
 - a. HRCT (high-resolution CT) Lung
- 5. Would your diagnosis change if the TLC remained at 75% but the FRC was normal (95% predicted)?
 - **a.** The TLC is the addition of the *measured* FRC and then the *added* IC (Inspiratory Capacity which may be sub-optimal or poor effort).

Question #3:

- 1. What findings might allow underwriting to proceed with a potentially favorable rating?
 - a. Nodule calcification (with exception of eccentric calcification)
 - b. Smooth nodule border
 - c. History of known-positive skin or serum testing (+) for pulmonary tuberculosis
- 2. Would a negative PET-CT be helpful?
 - a. The lower limit of resolution of current PET imaging is 8 mm, so failure to "see" the lesion for uptake of isotope would be expected
- 3. What if fiberoptic bronchoscopy with transbronchial lung biopsy + brushing + washings were negative would that help?
 - a. These studies are valuable only if they reveal malignancy or some other diagnosis such as granulomatous disease

- 4. And what if, following negative fiberoptic bronchoscopy the patient had undergone a VATS (video-assisted thorascopy) or open lung sub-segmental or wedge resection, with all findings negative for any diagnosis other than "normal lung"?
 - a. Again, the only significant finding would be malignancy or some other specific diagnosis, such as granulomatous disease or a scar or benign lesion. A nodule this small cannot be felt by a surgeon. At bronchoscopy, injection of dye near the lesion is often done to help the surgeon decide where to remove lung.
- 5. Assuming the case is postponed, when might a decision be made (assuming correct surveillance follow-up of the nodule is done)?
 - a. Usually two years of no change in the nodule size or appearance allows assumption that nodule is not cancerous. See Fleischner Rules for Lung Nodules.
- 6. Would any features of the nodule call for a longer observation period?
 - a. If the nodule was associated with ground-glass change on CT imaging, suggesting a diagnosis of bronchioalveolar carcinoma (now called AIS – adenocarcinoma in situ).

Question #4:

- 1. How severe is his OSA?
 - a. An AHI of 44 indicates severe OSA
- 2. What is his phenotypic subtype?
 - a. Subtype B (Oldest, comorbid), or Subtype E (Severe, hypoxemic)
- 3. What impairments are influencing his mortality?
 - a. Severe OSA; cardiovascular risk from HTN, possible LVH, hyperlipidemia; polypharmacy with narcotics, muscle relaxers, trazadone; possible cognitive impairment from OSA or meds or vascular disease or some combination of these? The strongest mortality driver is his cardiovascular risk, and his polypharmacy and cognitive decline contribute to this mortality risk. His hypoxemia is an additive concern and his AHI is a secondary concern.
- 4. What do you want to know to complete the mortality risk assessment?
 - a. What is his cardiovascular risk? (Needs a cardiac evaluation.) Is his PAP treatment effective? (Needs to see the sleep specialist and have a laboratory-based sleep study.) Does he have MCI? (May need a neurological evaluation, medication adjustments, better pain management.) What does his MVR show?

Question #5:

- 1. How severe is his OSA?
 - a. An AHI of 88 indicates severe OSA, even if there is no hypoxemia and even if his PAP study shows improvement.
- 2. What is his phenotypic subtype?
 - a. Subtype A (Classic) or Subtype F (Severe, non-hypoxemic)
- 3. What impairments are influencing his mortality?

a. Severe OSA, obesity, HTN, daytime sleepiness (risk of accidents), PVCs while sleeping. The strongest mortality driver is his severe OSA with elevated AHI without hypoxemia.

4. What do you want to know to complete the mortality risk assessment?

a. How will he improve compliance with PAP? Has he been fitted with a new mask with new pressure settings? Will he exercise and lose weight? What does his MVR show?

Question #6:

- 1. How severe is her OSA?
 - a. An AHI of 13 indicates mild OSA
- 2. What is her phenotypic subtype?
 - a. Subtype C (Female, insomnia)
- 3. What impairments are influencing her mortality?
 - a. Mild OSA, life stressors, insomnia, overweight. She has multiple common mild drives of mortality.
- 4. What do you want to know to complete the mortality risk assessment?
 - a. What does her MVR show? Routine age/amount requirements.