

SOCA

PRESENTATION, PROCESS AND ASSESSMENT

“Being a premed never prepared me for public speaking. I mastered the Henderson–Hasselbach equation and the physics of laminar flow, but in hindsight, I wish I had also joined Toastmasters.” Rachel Sobel, a fourth year medical student at UCSF contemplating an upcoming presentation, from the article: *Medicine as a Second Language*, NEJM 352:1945-1946, 2005

I. OVERVIEW

Oral case presentations will be a vital component of your medical career. Over time, you will find yourself presenting to peers, housestaff, attending physicians AND you will be called upon to explain medical problems to your patients and their families. At MUSM we begin early to provide a unique means for our students to gain the skills and confidence necessary to present patient cases comfortably.

The Student Oral Case Analysis (or SOCA) is an opportunity for students to demonstrate medical knowledge, effective oral communication skills and appropriate professional interactions with faculty. This is an educational opportunity that is both anxiety producing and exhilarating. First and foremost, it is a skill that can be learned, improved upon and eventually mastered. This will not happen overnight and it will not happen passively. Like all skills, it must be first modeled, then practiced and finally experienced.

This monograph provides a basic introduction to the SOCA including an outline of the required presentation model and a step-by-step approach to using that model with two specific cases. The first is a case that would be presented early in your academic career, during Phase A. The second is a case that would be presented much later, during the Pulmonology phase of year II. You will notice that there are obviously different expectations of first-year students versus the second-year students when it comes to SOCA performance. A tremendous amount of growth occurs over those 14 months and the expectations of the faculty increase over time. The increased expectations are well illustrated with these examples.

Details about the mechanics of the SOCA exam (for example: who are the examiners, how is it graded and what happens if you are unsatisfactory) should be carefully reviewed in the BMP policy manual. But basically, this is how it works. A SOCA is given at the end of each phase. Each student presents the case to a single faculty member. While the exam is formative in nature, students are evaluated against an exam standard and are assessed as being either satisfactory or unsatisfactory. Students who are unsatisfactory on a given exam are required to undergo a remediation process. After each exam, students are provided with detailed written and verbal feedback from their examiner. This is a learning process and students are expected to use this feedback to improve their presentation skills.

II. GENERAL SUGGESTIONS

Before we get too far into the details, let's start with some basic general information. You are given ~20 minutes for each SOCA presentation. It would be a good idea to become familiar with how much information can be conveyed in 20 minutes time beforehand. Each SOCA case will be representative of the cases in the Phase. Therefore, practice with a classmate using one of your BMP cases and time yourself.

Note how each case illustrates several essential basic science concepts from the phase. These will be apparent if you think through the case. Learning to identify these key concepts will be critical for effective case analysis.

Practice discussing the science to a depth that is relevant to the case. Be careful not to get bogged down in details. (For instance, in the Phase A example, there would be no reason to discuss every enzyme involved in gluconeogenesis!). One way to keep from getting unnecessarily immersed in minutiae is to pretend you are explaining the patient to a person who has scientific knowledge but is not medically trained.

Practice using the board... a simple diagram or pathway can give you a reference point for your discussion. It also serves to slow you down and helps to focus your presentation. However, effective use of the board is also a learned skill; do not spend too much time drawing something complicated.

Make eye contact with your examiners (or your practice partner). Remember you are presenting a patient to a faculty member, good communication skills require that you connect with your audience.

Take your hands out of your pockets. Be careful of engaging in distracting behaviors (paper shuffling, pen clicking, change rattling). Remember, this is a professional presentation, so refrain from using slang and lay terminology. Regulate the volume and inflection of your voice. If you suffer from "cotton mouth," it is acceptable to bring a bottle of water with you and take periodic sips. A successful SOCA must be heard and a confident and enthusiastic style enhances the effectiveness of the presentation.

Never forget that the case, albeit on paper, represents YOUR patient.

III. INTRODUCTION TO THE PROCESS

In Clinical Skills, medical students are introduced to the SOAP note format. This is a standard approach to writing progress notes for both most in-patients and out-patients. This format is not used by Psychiatry, however, and a special format (bio-psycho-social method) will be provided for your case analyses in the Brain and Behavior phase.

SOAP stands for

S: subjective...what the patient complains of and admits to during the history taking and review of systems.

O: objective...findings that the physician notes upon physical exam and the results of laboratory tests, imaging studies, biopsies or surgical resections.

A: assessment...the diagnosis, presumptive diagnosis, or differential diagnosis

P: plan...this includes further assessment treatment, prognosis, patient education

Thus the traditional, written SOAP note consists of four areas. At MUSM, we have adapted this four part SOAP note format to provide an organization scheme for the oral presentation of a paper case. The SOCA should consist of:

- 1) **an opening case summary based on the SOAP format .**
- 2) **identification of 3-4 issues that will be developed**
- 3) **a scientific case analysis based on the SOAP format that includes discussion of the identified issues.**
- 4) **at the end of the presentation, a brief concluding summary. This can be similar to the opening summary but emphasizes the assessment and plan parts a bit more.**

While in the BMP program, it is hoped that this format will provide a scaffold for reproducible SOCA analyses. It also provides a guide whereby groups may *close* BMP cases.

THE OPENING SUMMARY: This should be a brief, several sentence summary of who the patient is and why the patient came to the doctor (S), what findings the doctor noted (O), your conclusion as to what the diagnosis is or is likely to be (A), and what will be done for the patient (P). (This is the kind of summary that is given on “work rounds” to explain briefly each new admission from the night before. You will use a similar summary style to present a clinic patient that you have evaluated to your attending physician before taking him/her in to see the patient.)

In-depth explanations of mechanisms of disease are not part of the summary. It’s simply: why the patient is here, what findings are noted, what the diagnosis is or presumed to be, what is going to be done. The length of the opening summary may vary a bit with the complexity of the case, but should be no more than a few lines.

CHOICE OF ISSUES: just like during group process, key concepts derived from the case are made into issues that will be discussed as part of the scientific analysis of the case. You should write them on the board and be sure to include them in your presentation.

THE ACTUAL SCIENTIFIC ANALYSIS OF THE CASE:

During this part of the SOCA, the basic medical sciences and pathology from the phase should be used to explain, in a mechanistic way (that is, how things work or are not working), the patient's complaints, the findings, justify the diagnosis or differential diagnosis, the prognosis and the basis for therapy. Through the identification of specific case related issues, discuss the S, O, A, and P portions of the case. The case dictates which issues are most important. In one case, a specific subjective finding may be the basis of an issue; in another, the issues may all relate to the objective and plan/prognosis categories. The issues focus your analysis and description, but all four elements of the format must be addressed in some fashion in order for the presentation to be minimally satisfactory. Your presentation, including choices of issues, will be evaluated against a standard developed by the tutors from the phase.

Subjective: Using information from the phase, explain why the patient developed the symptoms and complaints that brought him/her to the doctor. You can add what kind of conditions the complaint(s) and/symptoms are making you think of, and you offer what they argue against. Explain (when given) if and how the past medical history, social history, family history contributed to the current problem (or not.) Depending on the case, this could include behaviors, exposures, drugs, occupational risks, genetics, other diseases that contribute to the patient's current problem. Again, use the scientific information from the phase in your explanation.

Objective: Use the phase related science to explain the physical findings, the lab data, any other imaging or test results, biopsy or surgical pathology reports. You should try to incorporate and integrate some or all of the disciplines into your explanations.

Assessment: Here you explain what the diagnosis is and offer any additional justification that is necessary. You can explain why the subjective information and objective data support it. You may even, as you become more comfortable with the process, explain why other diagnoses are not supported by the data.

Plan/prognosis: At this point you can explain the therapy in terms of mechanism of action of any drugs, etc. used and mention possible side effects. You can discuss the outlook for the patient in terms of prognosis and can offer any education that you think appropriate for the patient and family.

CONCLUDING SUMMARY: Here you summarize the patient case again in a few lines. You can emphasize the prognosis, treatment and any appropriate patient education. This summary provides a formal ending to the presentation.

Your SOCA skills are a “work in progress.” With each SOCA, you will be expected to give more in-depth analysis, demonstrate greater clinical and scientific reasoning and develop an increasingly professional “style.”

The following are two examples of SOCAs with their summaries, issues and analyses. The first has been used in Phase A ; the second is the 2005 Pulmonology SOCA.

IV. YEAR 1, PHASE A SOCA EXAMPLE

Herbert Smart is a 50-year-old man who was admitted through the Emergency Room with

Chief Complaint: Comatose state.

History of Present Illness: Mr. Smart had been to see his family physician 2 days ago because of recurring episodes of rapid heart beat, sweating and feeling faint. These symptoms were relieved by eating. He also stated that he had a near-constant voracious appetite and had gained 10 lbs. At that time, a random blood glucose level was 35 mg/dL (normal fasting glucose: 60-100 mg/dL). An insulin level obtained at the same time was within the normal range, but the ratio of insulin to glucose was 1.0 (normal < 0.4). Further laboratory studies revealed elevated levels of pre-proinsulin and proinsulin as well as C-peptide. A glucose tolerance test was planned for today, but the patient was found unresponsive by his wife this morning and brought into the ER.

Physical examination in the ER was remarkable for a well developed, slightly obese man who was unresponsive to deep pain.

Blood was drawn and an intravenous (IV) bolus of 50 mls of 50% glucose was rapidly administered.

Lab results on the pre-bolus blood sample:

Serum analyte	Patient value	Normal range
Glucose	22 mg /dL	60-100 (fasting)
Potassium (K ⁺)	3.1 mEq/L	3.5-5.0
Sodium (Na ⁺)	140 mEq/L	136-145
HCO ₃ ⁻	24 mEq/L	21-28
Chloride (Cl ⁻)	103 mEq/L	98-106

The patient awoke and was able to give a urine sample which was negative for glucose and ketones. He became unresponsive again after 20 minutes. He was quickly stabilized on continuous IV administration of glucose and admitted for further evaluation. Imaging studies revealed the presence of a small mass in the tail of the pancreas. He underwent surgical resection.

Surgical pathology report: pancreatic endocrine tumor. Special stains revealed the tumor cells were positive for insulin.

The patient recovered uneventfully without recurrence of symptoms. His physician plans to follow him with periodic checks of serum insulin levels to rule out recurrence of the tumor.

THE FOLLOWING IS AN EXAMPLE OF HOW YOU WOULD APPROACH THIS CASE USING THE 4-STEP PRESCRIBED FORMAT

1) Opening summary: This patient was a 50-year-old man with a history of episodes of rapid heart rate, sweating and feeling faint. He was found to be hypoglycemic with evidence of increased insulin secretion. Before additional tests could be completed, he presented to the ER in hypoglycemic coma. He responded to IV glucose and fluids. Subsequent evaluation followed by surgical resection revealed a pancreatic insulin producing tumor. His post-operative course was uneventful. He will be followed to rule-out recurrence of the tumor.

SOAP components of the **opening summary**:

S: This patient was a 50 year old man with a history of **episodes of rapid heart rate, sweating and feeling faint.** (what he complains of)

O: He was found to be **hypoglycemic with evidence of increased insulin secretion.** Before additional tests could be completed, he presented to the ER in hypoglycemic coma. He responded to IV glucose and fluids. (data collected and observations made by the doctors)

A: Subsequent evaluation followed by surgical resection revealed a **pancreatic insulin producing tumor.**(a diagnosis and the reason for his episodes)

P: His post-operative course was uneventful. He **will followed to rule-out recurrence of the tumor.** (no other treatment necessary right now; he may be cured but will need follow-up)

2) Key concepts in this case included normal glucose homeostasis; the effects of excess insulin in the patient; overall anabolic function of insulin.

Examples of possible issues would be:

- 1) What was the underlying cause of his “episodes,” increased appetite and weight gain?
- 2) How is serum glucose normally regulated and why were his levels abnormal?
- 3) What is the normal function of insulin and how was this exaggerated in our patient?
- 4) How were the lab data related to insulin levels in our patient?

These issues will be brought out in your analysis and discussion that follow.

3) Scientific Analysis of the Case:

Subjective: This patient is a middle-aged man who complained of episodes of rapid heart rate, sweating and feeling faint. These were relieved by eating, suggesting that they were associated with a low serum glucose. Here you could discuss how ingested glucose gets into the blood and then from the blood into the tissues that utilize it. The interplay of glucose with insulin and the insulin dependent tissues could be brought out here. You might go ahead and say that the brain generally depends on a steady supply of glucose and when deprived, symptoms of dizziness and feeling faint occur; if the deprivation is severe enough, frank coma can result as we see later on. (The rapid heart rate and sweating were triggered by epinephrine release; the mechanisms, etc of this hormone are studied in a later phase.)

Objective: The low serum glucose levels were confirmed. There was also laboratory evidence of increased insulin secretion. He complained of weight gain, but it is also something that the physician can document. His weight gain was due to the anabolic effects of insulin in combination with the ingestion of foodstuffs in excess of his caloric needs. Here you could talk about the influence of insulin on glycogen synthesis, triglyceride synthesis and protein synthesis. It would be fine to mention the role that glucagon plays in the normal person as well. Another objective finding is his response when comatose to the IV bolus of glucose. His low K^+ level is in keeping with excess insulin as K^+ enters cells along with glucose under the influence of insulin. His normal HCO_3^- , low glucose, and the absence of ketones and glucose from the urine help rule out another cause of coma that you have learned about, diabetes mellitus.

Imaging studies showed a mass in the tail of the pancreas. Obviously the source of insulin is the beta cell population of the pancreatic islets. The presence of a mass along with his symptoms and findings suggested that he has an insulin-producing tumor.

Assessment: Examination of the surgical resection specimen revealed a tumor with cells positive for the presence of insulin. The diagnosis is accordingly an islet cell tumor or a pancreatic endocrine tumor. Either term is fine.

Plan: The patient did well after surgery, indicating that the tumor was removed completely. He was told that he will need follow-up as there is the possibility of recurrence. Any return of his previous symptoms should prompt his notifying his physician.

4) Concluding summary: This was the case of a 50-year-old man who suffered from worsening episodes of hypoglycemia and finally presented to the ER in a coma. An insulin producing pancreatic tumor was identified as the cause of his problem. Following successful surgical removal, he was counseled about the nature of his tumor and the importance of follow-up.

V. YEAR 2 SOCA EXAMPLE

(obviously this is more complex than the Year 1 example, in keeping with the level of training)

Mrs. Kathy L. is a 29-year-old woman who presents to her family physician with

Chief complaint: chronic cough and shortness of breath.

History of Present Illness: Mrs. L has noticed the gradual onset of a worsening non-productive cough over the last few months. The cough troubles her throughout the day, regardless of activity or where she is and sometimes keeps her awake at night. She states that she now feels tired much of the time and has shortness of breath on exertion such that she can no longer participate in her aerobics class. Despite giving up aerobics, she has lost 10 lbs without trying to. She has not taken her temperature, but says that she has felt “feverish” on some afternoons. She has no allergies, has never smoked cigarettes and her medications are limited to ibuprofen as needed and oral contraceptive pills. Aside from the occasional viral upper respiratory tract infection, she has not had any previous lung problems. She has not traveled recently and denies exposure to individuals with tuberculosis or other known infectious diseases, bird or bat droppings, or fibrogenic dusts.

PMH: No major illnesses or surgeries.

Social History: The patient works as a bank teller in a relatively new building. She admits to moderate social alcohol intake. She has been married for two years; her husband is in good health. They live in a recently built condominium near downtown.

Family History: The patient is of African American descent. Her parents and two younger brothers are alive and well. To her knowledge, there is no family history of pulmonary diseases.

Physical Examination: Well developed thin young woman who appears her stated age.

T: 37.5 C; P 80/min; R 20/min; BP 120/80 mmHg; O₂ sat 93% by pulse oximetry; BMI 19

HEENT: PERRLA; neck supple without masses; trachea midline
Chest: quiet breath sounds with occasional inspiratory crackles
Cardiac: regular rate and rhythm without murmur rub or gallop
Abdomen: soft, non-tender without masses or hepatosplenomegaly
GU: normal female, no masses; no discharge.
Extremities: full ROM at all joints, pulses 2+ throughout
Neurologic: AAOx3; reflexes 2+ and symmetric

Laboratory Data:

	Patient	Normal ref values
Hgb g/dL	11.2	12.7-14.7
Hct %	34	37.9-43.9
RBC X10 ⁶ /μL	4.45	4.1-4.9
WBC / μL	3700	4000-10000
Monocytes %	10	0-10
Lymphocytes %	10	15-40
Neutrophils %	75	40-80

CXR: mediastinal and hilar lymphadenopathy with bilateral hazy interstitial infiltrates; no cardiomegaly; no pleural changes.

Arterial blood gases		Normal 30 y.o. woman, room air
pH	7.46	7.38-7.44
pO ₂ mmHg	66 (room air)	90-100
pCO ₂ mmHg	30	40
HCO ₃ ⁻ mEq/L	15	21-28

Pulmonary function tests	Patient	% predicted
Forced Vital Capacity (FVC)	1.65 L	44%
Forced Expiratory Volume at 1 second (FEV ₁)	1.46 L	52%
FEV ₁ / FVC	89%	(Normal 80%)
Functional Residual Capacity (FRC)	1.31 L	42%
Residual Volume (RV)	0.9 L	56%
Total Lung Capacity (TLC)	2.55 L	46%
Diffusing Capacity for Carbon monoxide (DL _{CO})	16.92 mL/min/mmHg	60%

A 5 TU PPD skin test was applied to the volar surface her left arm and a panel of standardized antigens (known to elicit a DTH response) was applied to her right arm. After 48 hours all sites were reported as non-reactive.

Bronchial lavage and a transbronchial lung biopsy were performed. Surgical pathology report on biopsy material: multiple well circumscribed noncaseating granulomas; all special stains for organisms were negative.

Routine bacterial, mycobacterial and fungal cultures of the biopsy material and lavage fluid subsequently reported as no growth.

Flow cytometric analysis of bronchoalveolar lavage fluid revealed a lymphocytosis with CD4/CD8 T-cell ratio 5:1.

The patient was started on 30 mg of prednisone daily with a plan to continue that dose for 8 to 12 weeks whereafter she will be re-evaluated.

THE FOLLOWING IS AN EXAMPLE OF HOW YOU WOULD APPROACH THIS CASE USING THE 4-STEP PRESCRIBED FORMAT

1) Opening summary: The patient is a 29-year-old African-American woman who has experienced persistent cough and increasing shortness of breath over a period of several months. Findings included quiet breath sounds bilaterally, interstitial infiltrates on chest X-ray and a restrictive pattern of pulmonary function studies. A bronchoscopic biopsy revealed non-caseating granulomas with negative special stains and cultures. A presumptive diagnosis of sarcoidosis was made and prednisone therapy has been initiated.

SOAP components of the **opening summary:**

S: The patient is a 30-year-old African-American woman who has experienced **persistent cough and increasing shortness of breath over a period of several months.**

O: Findings included **quiet breath sounds bilaterally, interstitial infiltrates on chest X-ray and a restrictive pattern of pulmonary function studies.** A bronchoscopic biopsy revealed **non-caseating granulomas with negative special stains and cultures.**

A: A presumptive (Note: at this point you are expected to arrive at a diagnosis on your own) diagnosis of **sarcoidosis** was made and

P: **prednisone therapy** has been initiated

2) Key concepts include mechanism of cough, compromise of pulmonary function in restrictive lung diseases, diseases that typically cause restrictive changes, granulomatous lung diseases, the role of immunosuppressive drugs in treating the patient. In addition, at this level of training you should be able to think your way through the possible diagnoses in this case, choose one and justify it.

Examples of possible issues would be:

- 1) What changes of pulmonary function are characteristic of restrictive lung diseases and why.
- 2) Does the patient have any predisposing factors to the development of restrictive lung disease?
- 3) How does the patient's combination of symptoms, findings and test results support or rule out a particular diagnosis?

- 4) What is the long term outlook for this patient in terms of likelihood of disease progression, efficacy of treatment and side effects of treatment?

3) Scientific Analysis of the Case:

Subjective: The patient described a gradual development of pulmonary problems rather than an abrupt and relatively precipitous onset that might be expected in acute infectious pneumonia. Her cough is non-productive, unlike what would be expected in a bacterial pneumonia. The characterization of the cough as occurring anywhere, everywhere and all the time makes allergic processes less likely. The persistence of the cough but lack of sputum production might point to a process that stimulates J receptors. She does not smoke and has no obvious workplace exposures, so dust related diseases, COPD and even Idiopathic Pulmonary Fibrosis (which has a smoking association) become unlikely. The history of weight loss and fever suggests a systemic illness where IL1 and TNF elaboration occur. Those symptoms might even make us wonder about TB or other deep-seated granulomatous infection or cancer. Since the SOCA is phase specific, you are probably not going to suggest lymphoma, but someone might mention lung cancer. Her lack of a smoking history, while not guaranteeing “no cancer”, makes it less likely. You might bring up bronchioloalveolar carcinoma as it has no clear association with smoking, occurs at any age, can be bilateral, and can be situated so peripherally that it may not result in much sputum production (although some tumors actually are responsible for a lot).

She has no preexisting acquired or inherited conditions that would predispose to pulmonary disease, and no obvious workplace or environmental exposures. She does not have socioeconomic risks for TB. Although you have not systematically studied HIV yet, you might still know that she has no obvious risk factors for HIV. African Americans have a higher incidence of sarcoidosis. They also are more likely to develop primary progressive TB than are whites (might not be in primary reading).

Objective: On physical examination, she appeared chronically ill, but not in acute distress. She was afebrile at this time with respirations increased and O₂ saturation decreased. The remainder of the abnormal findings were limited to the lungs with no signs of consolidation. Inspiratory crackles are often heard in interstitial (fibrotic) processes. Her routine lab data revealed a mild M/H anemia. Young women are often iron deficient, but the findings are also c/w anemia of chronic disease where IL1 and TNF inhibit the release of iron from storage sites. She was borderline leukopenic with an absolute lymphopenia and a relative monocytosis. Lymphopenia can occur in a number of settings, including sarcoidosis. Extrinsic Allergic Alveolitis (EAA or hypersensitivity pneumonitis) often has an associated neutrophilia during attacks. Her WBC was not consistent with an acute bacterial infection but could be consistent with TB or a pathogenic fungal infection.

Her chest X-ray revealed bilateral interstitial infiltrates; these are seen in many infiltrative lung diseases: Idiopathic Pulmonary Fibrosis, Desquamative Interstitial Pneumonitis (another smoking related interstitial lung disease), EAA, Collagen Vascular Diseases, pneumoconioses, viral and other “atypical” infectious pneumonias. Hilar

lymphadenopathy, however, is not generally seen in those conditions. Of the pathologic conditions you should have encountered this phase, this constellation of chest X-ray findings is characteristic of sarcoidosis but could also be consistent with TB or histoplasmosis. Also, physical exam and cardiac shadow do not suggest that she has developed RVH or cor pulmonale. Her arterial blood gases show that she is hypoxemic with a respiratory alkalosis and partial metabolic compensation. The drive for hyperventilation is likely due to some extent by hypoxic stimulus of peripheral chemoreceptors and due to stimulation of J receptors in the lung (as is the cough) by the granulomatous and subsequently fibrotic infiltrates.

Her pulmonary function studies show a pure restrictive pattern. The infiltrative process visible on chest X-ray is interfering with normal expansion of the lung, helping us see why she has developed exercise intolerance and quiet breath sounds. In sarcoidosis, the granulomatous and then fibrotic infiltrates disrupt, distort and even obliterate alveolar surface area and capillary beds. There will be areas that are ventilated better than they are perfused and other areas that are perfused better than they are ventilated (with the latter probably being the dominant finding). This combination leads to a V/Q mismatch.

The anergy panel results indicate that the patient is anergic: typical in sarcoidosis, not part of EAA, not part of berylliosis; new onset of anergy is associated with fulminant miliary TB but she is not that acutely sick. The differential diagnosis on the biopsy material includes all granulomatous infectious diseases: TB, other mycobacteriosis, histoplasmosis, blastomycosis, coccidioidomycosis as well as non-infectious granulomatous diseases like sarcoidosis, EAA and berylliosis. She has no exposure history for berylliosis; the pattern of symptoms is not characteristic of EAA (nor is the hilar adenopathy). Infectious granulomas eventually undergo caseous necrosis, but we might have sampled some relatively “fresh ones.” All special stains and cultures are negative; while not absolute, the findings are very suggestive of a non-infectious granulomatous disease. In addition, the lavage findings are characteristic of sarcoidosis. (EAA usually has increased #s of CD8s relative to CD4s).

A: The patient’s history, physical exam, chest X-ray, PFTs, lab data and biopsy findings are all consistent with a diagnosis of **sarcoidosis**. Sarcoidosis is a systemic disease that can involve a number of organs and anatomic sites including the heart, lymph nodes and lungs. Histologically, it is characterized by infiltrates of granulomatous inflammation and the formation of non-caseating granulomas; unlike infectious granulomas, these typically do not undergo caseous necrosis, but become surrounded and replaced by fibrosis. In the lungs, it proceeds as a bilateral interstitial process that leads to restrictive changes. Lymphocytes of the CD4 TH1 phenotype are prominent members of the infiltrates as well. Since there has not been convincing evidence for an infectious etiology in sarcoidosis, the formation of granulomas followed by fibrosis has been attributed to an aberrant immune response

P: The patient was started on 30mg of prednisone daily with a plan to continue that dose for 8 to 12 weeks whereafter she will be re-evaluated. The etiology of sarcoidosis is unknown and treatment is empiric. Since there has not been convincing evidence for an infectious etiology in sarcoid, the formation of granulomas followed by fibrosis has been attributed to an aberrant immune response and therapy has been directed at immune

suppression with the use of glucocorticoids. The nature of the inflammation indicates a role for CD4 lymphocytes. Glucocorticoids have a number of actions. In this case, they most likely act to reduce the expression of IL2 by CD4 cells and ultimately impact CD4 cell number and function. Steroids also interfere with collagen synthesis. She should be advised that there is no cure for her illness, but that steroids do help reduce the symptoms and the long term changes in the lungs. She should be advised that the immunosuppressive therapy will make her more vulnerable to infections.

4). Concluding summary: This patient is a young African American woman with symptoms and findings of restrictive lung disease. Laboratory studies and biopsy results are consistent with a diagnosis of sarcoidosis. She has begun prednisone therapy and has been advised that she will need long-term follow-up. While sarcoidosis generally responds favorably to prednisone therapy, she should be advised of the possibility that she may develop pulmonary fibrosis and even multi-organ involvement. The side effects of immunosuppressive therapy have been explained and she knows she is more vulnerable to infections.