For my first site evaluation, I presented a case of a 42 y/o M with no significant vascular risk factors who came in as a stroke code for acute onset left-sided weakness and slurred speech. The history was obtained mainly from his mother after she noticed a sudden change in speech and responsiveness while speaking with him over the phone. On exam, he had significant neurologic deficits including left hemiplegia, hemisensory loss, left facial droop, dysarthria, right gaze deviation, and left homonymous hemianopia with an NIHSS of 19. Imaging ultimately revealed a right MCA occlusion consistent with an acute ischemic stroke secondary to LVO. I chose this case because it really stood out to me, especially since it was the first time I had actually seen a stroke code unfold in real time. Seeing how quickly everything moved and how structured the response was made the whole process feel very different from just reading about it. It also made me realize how important it is to stay organized and focused in situations where every minute matters, even when the presentation doesn't fit the typical risk profile you expect. I also liked thinking through the patient education portion of this case. Even though I wasn't actually delivering it to the patient or family, I found myself having to slow down and figure out how I would explain something as serious as a stroke in a way that makes sense without using too much medical language. My evaluator felt that my presentation was organized and my differential was appropriate.

For my second site evaluation, I presented a case of a 78 y/o F with a history of COPD and HTN who presented with progressively worsening shortness of breath and productive cough with yellow sputum. On arrival, she was hypoxic and required BiPAP support, with labs showing leukocytosis, elevated lactate, and significantly elevated BNP, along with imaging demonstrating a moderate left pleural effusion with concern for possible underlying pneumonia. Given her presentation, the differential included COPD exacerbation, pneumonia, and possible CHF exacerbation contributing to acute hypoxic respiratory failure. I chose this case because it showed how complex respiratory presentations can be, especially in elderly patients with multiple overlapping comorbidities where it is rarely just one isolated diagnosis. My evaluator felt that my assessment and plan were appropriate and well-structured, particularly in how I addressed multiple possible etiologies while initiating broad but targeted management.

For my journal article, I focused on the role of natriuretic peptides in elderly patients with COPD and how BNP and NT-proBNP can be interpreted in this population. I chose this article because during my rotation I noticed how often BNP levels were used to help sort out whether a patient's shortness of breath was coming more from the heart or the lungs. The article pointed out that while BNP and NT-proBNP are helpful in supporting the diagnosis and prognosis of heart failure, things get more complicated in COPD. Levels can be elevated for a number of reasons even when left-sided heart failure is not present, including chronic hypoxia, pulmonary hypertension, right heart strain, and acute exacerbations. It also showed that BNP levels tend to rise with worsening COPD severity and can be associated with poorer outcomes like increased need for ventilatory support and higher mortality. At the same time, there isn't a clear cutoff that can reliably separate COPD from heart failure in every case, especially in older patients with multiple comorbidities. Reading this made me more cautious about how much weight I put on a single lab value when I'm working through dyspnea cases, and it pushed me to think more about the overall clinical picture rather than relying on a lab value in isolation.