1. Breakdown of motor control following failure of sensory of motor systems leads to what?
   a. Deafferentation [peripheral]-- Can you define deafferentation vs afferentation?
   b. Parkinson's disease [central]
   c. What is the distinction between central and peripheral?
2. What are some sensory systems?
3. What is the sixth sense?
4. What is proprioception?
5. Are motor systems organized hierarchically? What other system is organized this way?
6. How many levels or hierarchies are there in the motor system?
7. What structures are at each level?
8. What is the role of spinal cord neurons in movement?
9. How else is the motor system organized in addition to hierarchically? What is this an important feature of the motor system? What does it essentially protect against?
10. What structure is damaged in Parkinson's Disorder (PD)? How does this structure influence movement since it does not have a direct projection to the brain stem?
11. Is the basal ganglia (BG) located deep or superficially in the cortex?
12. As stated by one of your fellow in students in lecture, what is the role of dopamine? Two examples were given.
13. What happens to dopamine neurons in PD?
14. What are the cardinal signs of PD?
   a. Resting Tremor-- What ameliorates the tremor? When does it pick back up?
   b. Akinesia-- This is also characteristic of a lack of swinging of the arms. What is the gait like in an individual with PD? How can this characteristic gait be alleviated? (Hint: something can be added to the floor to help the individual walk more normally.)
   i. Masked facies- please define.
   c. Rigidity- Know the definition
   d. Loss of postural reflexes- How did they demonstate this characteristic in the video?
15. What is bradykinesia?
16. What structure is important for the internal guidance of movement?
17. Describe the Pointing Paradigm.
18. In the study, were the subjects ON or OFF their medication?
19. How did the controls perform in the task? Did they still show errors?
20. What about the PD subjects' performance? How did they compare to the controls?
21. What are the conclusions of the study?
22. What were the 3 conditions in the second experiment?
i. No-vision- requires proprioceptive cues
ii. Finger-vision- bypasses the use of proprioception
iii. Target-vision- bypasses memory requirement
23. How did the controls compare to the PD patients in this paradigm?
24. What was the worst condition for PD patients? Target vision was harder than finger vision, so what helped them most was giving them visual information about the position of their arm.
25. What condition did not show significant differences between the two groups?
26. What were the conclusions from this experiment?
27. So, why do chalk marks on the ground help these patients? These external cues act as a substitute for which ability? [Hint: Something about knowing where something is in space. Also, the 6th sense described earlier.]
28. What two conditions force subjects to rely on proprioceptive cues?
29. Why were virtual realities used? What could the researchers do in this type of environment? [Hint: Something about distortion...]
30. False visual feedback
31. In what condition was there a HUGE difference (initial learning, baseline, reversal learning, or aftereffect)?
32. What were the conclusions of the virtual reality study?
33. Why are the deafferented subjects rare? [Hint: Think specificity!]
34. What is still intact in these patients?
35. How is Ian(?), the subject from the video, different from other individuals with deafferentation? [Hint: Think preserved abilities.]
36. How are large nerve fiber tracks lost as the result of a virus in these cases? [Think about the direct effects of the virus vs the body's defenses.]
37. In general, think about how proprioceptive cues are used/transformed in the basal ganglia. Note that there is a cross-modal interaction (combining information gained from one modality with information from another modality).
38. Did visual cues help deafferented subjects perform the reaching task? [Think of the vision vs no vision comparison].