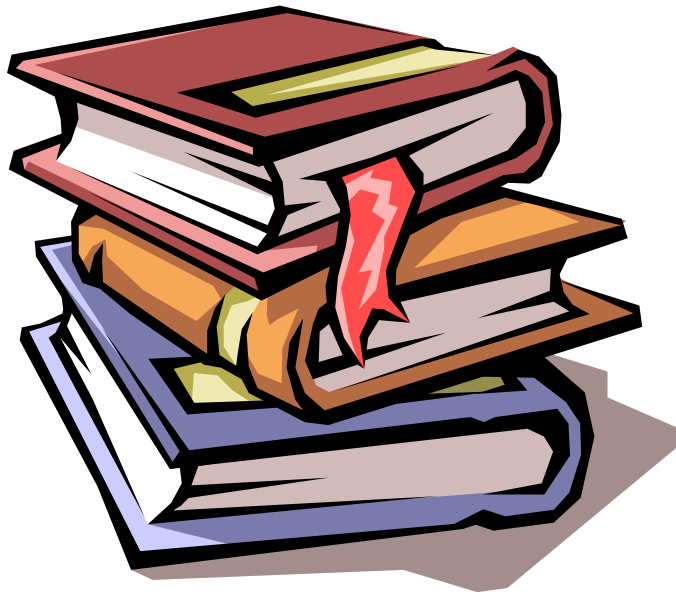


University of Virginia School of Medicine

Clinical Problem Sets

**Neurology Clerkship
2009 - 2010**



Dr. David Geldmacher, Clerkship Director
KarenMarie Vale, Clerkship Coordinator
434-924-5548

Revised September 2009

This book contains nine (9) clinical neurology cases, each with a set of problems or questions. You are expected to study each case, and to research and answer all questions. You are expected to master this material since a final examination at the end of the clerkship will be based on this exact same material.

These problem sets have been carefully constructed. The cases, together with the questions, end up covering a standardized set of topics. This way, all students have the same chance to master the same core neurology concepts. *The cases have been written so that you will learn best by studying and answering all questions on one page before going on to the next page.*

We trust you to take ownership of your own learning. Mastering this material is an expectation of the clerkship and the responsibility of the student. You can decide how that works best for you. We are here to help, if you wish. You are free to use any reference material you want. You may talk with each other and with whomever else you like. You ***are not*** required to turn in any answers or write responses to the questions in this book. You ***are*** expected to attend the weekly Friday problem set review sessions, led by Department of Neurology attendings. ***Please arrive prepared to discuss your thoughts and ask questions about the problem sets assigned for that day (see below).***

The case schedule is as follows:

Week #1: Problem sets 1, 2, 3

Week #2: Problem sets 4, 5, 6

Week #3: Problem sets 7, 8, 9

Problem 1 - Page 1

A 26 year-old right-handed nurse stops you in the hall to ask about hand numbness. For two months, she has had numbness and tingling in both hands. When typing or adjusting IVs she feels clumsy. One time she dropped a cup of coffee for no apparent reason. She does not feel that her hands are weak, and has no other complaints or significant past medical history.

She takes an oral contraceptive but no other medication.

She smokes one pack of cigarettes daily, drinks about six beers on weekends, and has a nightly glass of wine with dinner. She used marijuana, cocaine and LSD in college five years ago. She is sexually active in a serially monogamous fashion; she has been with the same man for three years and is contemplating marriage. She denies exposure to solvents or other toxins.

Questions:

- 1. List three potential neurologic localizations for her hand numbness, and discuss how you would differentiate them with neurologic examination and diagnostic tests.**
- 2. What syndromes or diseases are most likely for each localization?**
- 3. What are the neurological complications associated with oral contraceptive agents, and their relationship to tobacco use? How might they be relevant in this case?**

Problem 1 - Page 2

Physical examination:

Unremarkable full general exam.

Neurologic exam:

Mental status: Looks concerned. She correctly pantomimes the use of a hammer and scissors with either hand.

Cranial nerves: Pupils 4mm, round, and briskly reactive. Fundi with pale disks. Extraocular movements intact. Visual acuity and fields normal.

Motor: Normal strength, tone, bulk, and movement .

Reflexes: Brisk, symmetric without clonus or pathologic reflexes. Toes downgoing.

Sensory: Symmetric and full temperature, position, pinprick, and vibration; no abnormal sensations associated with neck movement

Coordination: Accurate finger-to-nose bilaterally

Gait: Full stride, narrow base, symmetric arm swing, good tandem.

Question:

- 4. What additional sensory examinations could be conducted? What information might they provide beyond the normal primary sensory modalities?**
- 5. She asks what you think the diagnosis is. What will you tell her? Would you perform any investigations at this point? If so, which ones? Why?**

Problem 1 - Page 3

She marries the next summer. She sees you after coming back from a Hawaii honeymoon. She says that at the beach she had double vision when she looked to the right. She thought it was due to fatigue from travel and all the stress of wedding planning.

Her exam is now remarkable for the following changes (the rest of the exam is unchanged):

Cranial nerves: Both pupils constrict to 3 mm diameter in response to light in the left eye. But, when the same light is shone in the right eye, both pupils constrict further to about 2 mm. Corrected visual acuity using a Snellen card held at 14 inches is accurate to the 20/200 equivalent OS, 20/20 OD. On fundoscopic exam, the left optic disc is swollen and gray. The eyes at rest and on left lateral gaze are conjugate. On right lateral gaze, the left eye does not adduct past midline; the right eye abducts fully but develops horizontal nystagmus with the fast phase to the right.

Reflexes: Clonus at both ankles. Bilateral Babinski signs.

Questions:

6. **What are the pathways subserving the pupillary light reflex? Draw them, and indicate the point at which you think this patient has a lesion.**
7. **Evaluate the relative merit of performing the pupillary light reflex examination in this patient in a dark environment versus a bright environment.**
8. **What are the pathways coordinating conjugate lateral eye movement? Draw them, and indicate the point at which you think this patient has a lesion. How do the neuroanatomic pathways control for vertical gaze, horizontal gaze, and ocular convergence differ?**
9. **What is your differential diagnosis at this point, and how would you work up the patient at this point?**

Problem 1 - Page 4

Laboratory studies:

- CBC, Chemistries, UA, RPR, ANA, ESR, and complement levels are normal.
- CSF: opening pressure 12 cm H₂O
protein 40 mg dL
glucose 70 mg dL (serum 95 mg dL)
cell counts: 0 RBCs, 7 WBCs (all lymphocytes)
IgG albumin ratio: 0.24 in CSF, 0.11 in serum
High resolution CSF electrophoresis: 4 oligoclonal bands
Positive antibody to myelin basic protein

Studies:

- MRI brain and spinal cord show six areas of increased T2 signal in the cerebral white matter. Several hemispheric lesions are oriented perpendicular to the lateral ventricles. No hemispheric lesions enhance with administration of contrast material.
- Visual evoked responses yield normal conduction on stimulation of the right eye and delayed conduction on stimulation of the left eye.
- Brainstem auditory evoked responses reveal a delay between waves III and V on right ear stimulation.
- Somatosensory evoked responses show normal latencies from each median nerve to the brachial plexus and cervical spine, but bilaterally delayed conduction from the cervical spine to the cortex.

Questions:

9. **What is the neuro-immunologic basis of this condition? How do similar conditions differ in pathophysiology?**
10. **What effect does corticosteroid treatment have on this condition?**

Problem 1 - Page 5

You admit the patient to the hospital for corticosteroid treatment . Two weeks later, her left eye acuity is 20/100 and remains stable thereafter. Her diplopia gradually resolves; her left eye adducts normally on right lateral gaze, but the right eye continues to demonstrate some nystagmus.

Questions:

- 11. What are contemporary treatments that may improve the natural history of this condition? Which categories of patients may not benefit from these treatments?**

- 12. Discuss the natural history of this condition. What types of courses does this disease exhibit? What is the effect of pregnancy on the natural history of this condition?**

- 13. In light of her subsequent course, what might have been done differently at initial presentation? What information from imaging would have been most informative?**

Problem 2 - Page 1

A 40 year-old right-handed Asian-American man was admitted to the hospital for progressive leg tingling and weakness.

Four weeks earlier he had a flu-like illness. A throat culture for group A *Streptococcus* was negative. He was treated conservatively with rest and acetaminophen, and was back to normal in five days.

Three days prior to admission, he noticed “pins and needles” in his legs. The next day, he couldn't climb stairs because his legs felt “tired,” hard to lift, and “numb.” Yesterday, he had trouble walking on flat surfaces. He has also noticed a weak grip, difficulty raising his arms, and difficulty swallowing.

Questions:

- 1. What possible localizations could account for his symptoms? Describe why.**
- 2. What is your differential diagnosis at this point?**

Problem 2 - Page 2

He has no history of diabetes mellitus, hypertension, TB, hepatitis, arthritis, or seizures.

Pertinent Review of Systems: He had momentary lightheadedness on rising from a sitting position. He denied bladder or bowel symptoms, or abdominal pains. He had not traveled out of Virginia in the last five years, and has never traveled overseas.

Social and Family History: He is a local delivery driver with no exposures to industrial chemicals, heavy metals or pesticides. He occasionally drinks alcohol, but denied other drugs. He quit smoking two years ago. He had no family history of muscle weakness or a rheumatologic condition.

Questions:

- 3. What is the importance of the absence of bowel and bladder symptoms in a patient with progressive weakness?**
- 4. What nutritional, toxic, or infectious processes could trigger a syndrome like this?**

Problem 2 - Page 3

Physical examination:

General: Appeared tired.
Vital Signs: BP 140/80, P 100 supine; BP 90/60, P 98 sitting; T 98.4° F; RR 24/min Became mildly dizzy on sitting
Lungs: Clear to auscultation; using accessory muscles of respiration
CV: Loud S1, S2; RRR; no S3, S4, murmur
Genitorectal: Normal anal sphincter tone

Neurological examination:

Mental Status: Mildly anxious.
Cranial Nerves: Bilateral facial weakness. Extraocular movements full.
Motor: Flaccid tone in all extremities; normal bulk; strength 4 - /5 lower extremity and trunk muscle groups, 4/5 upper extremity muscle groups.
Sensory: Decreased position sense in hands and feet.
Reflexes: Absent
Coordination: Slow heel to shin.
Gait: Patient unable to stand due to weakness and dizziness/wooziness.

Laboratory evaluation

CBC: normal
Serum chemistry panel: normal; Creatinine Kinase: normal
Pulmonary Function Test: vital capacity 1200 cc
CSF
Opening pressure: 18 cm water
Protein: 250 mg/dl
Cells: 2 lymphocytes per mm³
Glucose: 60 mg/dl

Questions:

5. How would you synthesize/formulate the patient at this point (including neurological localization) if you were presenting him on hospital rounds? (*Be prepared to be called on to present your formulation*).
6. What is the significance of the changes in blood pressure and pulse as he changed posture? How do they contribute to the differential diagnosis?
7. In testing the power of the wrist extensor muscles, two strategies may be available. One, start with the wrist fully flexed and have the patient try to extend the wrist against the power of the examiner. Two, start with the wrist fully extended and have the examiner overcome the power of the patient. Evaluate the optimal method to test muscle power for a meaningful conclusion.

Problem 2 - Page 4

Hospital Course:

On hospital day #1, his vital capacity dropped to 900 cc. He was endotracheally intubated and put on a ventilator. Heart rhythm and blood pressure were monitored. A nasogastric tube was placed for definitive enteral access. Plasmapheresis was performed every other day for 10 days, with good improvement in muscle strength and vital capacity. Physical therapy emphasizing passive range of motion was started early to avoid joint contractures.

On hospital day #3, he developed severe hypotension that resolved after less than one minute. During the next few weeks his blood pressure fluctuated wildly. This problem resolved after two weeks.

Questions:

8. **What role do immunological treatments (e.g., IVIG, steroids, Plasma Exchange) have in the management of this and related conditions? In what situations are they most or least useful?**

9. **What is the likely pathophysiology associated with this condition? At what point in the motor system are the lesions, (e.g. brain, spinal cord, nerve roots, nerves)? What are the key parts of the clinical presentation that lead us to that localization?**

Problem 2 - Page 5

Additional Studies:

Chest X-ray: normal
MRI C-spine: normal
24-hour urine for porphobilinogen: negative
Erythrocyte sedimentation rate: 11 mm/h
HIV: negative

EMG: Consistent with an acute, acquired demyelinating polyradiculoneuropathy.

Disposition

He was extubated on hospital day #15. Because of depressed mood and tearfulness, he began paroxetine. Motor and sensory function improved to close to his premorbid baseline. He was discharged home to continue outpatient physical therapy.

Questions:

- 10. How do the above tests help you to select from your earlier differential diagnosis?**
- 11. How would normal CSF or EMG findings have influenced your decisions differently? How often are they normal in acute neuromuscular disease of this type ?**
- 12. What prognostic variables are associated with the good recovery in patients with this condition? What factors predict poor recovery?**

Problem 3 - Page 1

A 67 year-old right-handed woman presents to you for evaluation of tremor. For 6 months she has had a slight bilateral hand tremor that goes away if she holds things. Her husband says it is absent during sleep. During these six months, he thinks her walking has slowed and she has fallen a few times while going down stairs. She voices surprise at his report of slowing, and states the falls were due to slippery shoes. She does not note stiffness.

Her husband also volunteers that for about one year prior to emergence of the tremor and falls, she has been more forgetful and disengaged in conversations. He states that she has been having difficulty using silverware, kitchen tools, and writing instruments. She interjects that her tremor is causing the problem, but he feels she seems to have “forgotten” how to use them. These changes are also slowly getting worse.

Questions:

- 1. Discuss the classification and differential diagnosis of tremor.**
- 2. Other than tremor, what other hyperkinetic movement disorders are there? Define and describe their clinical features.**

Problem 3 - Page 2

Past medical history:

- Restless leg syndrome diagnosed three months ago, well controlled by gabapentin.
- She had an uneventful appendectomy last year, but acted “crazy” one night in the hospital after getting “something for nerves.”
- Urinary incontinence.

Medications: gabapentin, aspirin, oxybutynin, hydrochlorothiazide, metoclopramide

Social history:

- She was asked to retire from being a legal secretary last year.
- She lives with her 75 year-old husband in a large rural house.
- She drives into town to do the shopping weekly. (She becomes angry when her husband states that she once became lost driving home from the store when she was forced to take a detour).

Family history:

- A cousin was hospitalized for a “nervous breakdown” at age 25 and later died from cirrhosis. The patient's family thinks he was an alcoholic.
- Both parents died in their late 80s of “old age,” and were “forgetful” in their later years.

Review of systems:

- Because of poor appetite, which she attributed to constipation, her PCP prescribed metoclopramide about 6 months ago.
- She is usually calm, though not very talkative, and describes her mood is “good.” She has poor sleep onset.
- Her husband is concerned that a few times during the day, she has seemed suddenly disengaged and sleepy, with her “thinking” very much worse. Then she would quickly be back to her usual baseline.
- He reports that she frequently sees small animals around the house that no one else sees. These perceptions occur especially often in the evening. They do not provoke fear, but she seems anxious about them and “fusses” about them.

Questions:

3. **What is dementia? What is delirium? What are key differences between them?**
4. **What conditions should be considered in patients with cognitive loss and gait/movement difficulty?**

Problem 3 - Page 3

Physical examination:

Vital Signs: BP 143/78, P 82 supine, BP 138/72, P 80 standing

Skin: seborrheic dermatitis on forehead; superficial bruises on arms and shins.

Neurological examination:

Mental Status:

- Alert, pleasant, few facial expressions.
- She often breaks off conversations to stare into space.
- She can spell WORLD forwards, but backwards spells it DLORLD.
- She is oriented to person, but cannot name the year, season, city, or state.
- She forgets three words in 10 minutes but recognizes them with multiple choices.
- She cannot name parts of objects very well (e.g. the point of a pen).
- When pretending to use key, she seems confused about how to make the proper movements

Cranial nerves:

- reduced blink rate
- saccadic intrusion on horizontal smooth pursuit
- voluntarily moves her eyes up, down, left, and right
- activates her face symmetrically to command

Motor:

- Minimally increased arm tone after contralateral activation; no cogwheeling
- 5/5 power in all extremities
- faint 3-5 Hz bilateral hand tremor at rest, no change when she walks.

Sensory: Normal

Reflexes:

- symmetric 1+ DTRs
- positive grasp and Myerson sign.

Coordination:

- Slow but accurate finger-to-nose.

Gait:

- Feet clear ground well, but initiation is slow, stride is short, and base slightly wide
- withstands a strong retropulsive challenge.

Questions:

5. **Formulate the important points of the history and examination. What is your differential diagnosis?**
6. **What work-up would you propose for this patient's neurologic condition? Why?**

Problem 3 - Page 4

Laboratory data:

CBC: within normal limits

Chem 20: within normal limits

RPR: nonreactive

TSH: within normal limits

Vitamin B12 within normal limits

Ceruloplasmin, serum copper, 24 hour urine copper: Within normal limits

Studies:

Brain CT: moderate generalized cortical atrophy with hydrocephalus ex vacuo.

Questions:

7. **How do these data affect your differential diagnosis? How often are tests for reversible sources of dementia informative? What does it imply if one of them is abnormal?**

8. **What treatments, tests, and/or medication adjustments would you like to recommend?**

Problem 3 - Page 5

Based on the CT report and the family's report of something they saw on a TV commercial, a lumbar puncture performed.

CSF: WBC 3, RBC 0, glucose 60, protein 28 ; 14-3-3 protein - negative.

No change in walking or mentation after 25 CC removed.

You diagnose dementia with Lewy Bodies (DLB) and begin therapy with transdermal rivastigmine, increasing to the 9.6mg/24 hour patch after one month. The family begins administering Vitamin E 1000 IU daily, and Vitamin C 500 mg po daily. A week later, her husband calls to say that she is more alert and less forgetful, seems to hold her own in conversations better, and has stopped having visual hallucinations.

Questions:

- 9. How does Dementia with Lewy Bodies differ from Alzheimer's disease clinically? How does it differ from other dementia syndromes with movement disorders?**
- 10. What will you tell the patient and her husband about the natural history of her condition, and what changes should you expect to manage over time? How might the nutritional supplements alter the expression of the syndrome?**

Problem 4 - Page 1

A 19 year-old female college student presents to an urgent care center complaining of “the worst headache of my life.” It began about 45 minutes ago, and consists of severe throbbing over the right side of her head, more marked anteriorly. She has vomited twice. She tried to lie down in a dark room to relieve the pain, but after a half an hour she decided to walk to the clinic.

Question:

- 1. What is your differential diagnosis at this point? What is the most urgent diagnosis to consider? What is most common?**
- 2. How would you organize the possible headache types by category?**

Problem 4 - Page 2

Review of systems: Her general health has been good.

Past medical history: She has had headaches in the past, often near her menses. Some of these have been one-sided and throbbing, but others have been circumferential and non-throbbing. She has never vomited with them before, and they always went away with acetaminophen or aspirin (and rest in a dark quiet room).

Medications: Two months ago she began taking oral contraceptives. She takes over the counter herbal supplements and occasional over-the-counter appetite suppressants.

Social history: She smokes a pack of cigarettes per day. She drinks no alcohol but uses energy drinks, like Red Bull, 4-5 times per week.

Family history: Her father died suddenly of an unknown cause at age 47. A maternal aunt has "migraine" headaches.

Questions:

- 3. Discuss the contributions of her various medications and habits to her presenting problem.**
- 4. How does her father's unexpectedly early death influence your thinking about her risks?**

Problem 4 - Page 3

Physical examination:

General: looks uncomfortable

Vital Signs: BP 113/68, P 72, T 98.9° F

Neck: supple

MM-skeletal: no restriction on passive movement

Skin: no rashes or lesions.

Neurological examination:

Normal

Following the visual field examination, she recalls that she saw some unusual flickering in the left visual field lights for about 20 minutes before the headache started.

Questions:

5. How does this information affect your differential diagnosis?
6. What investigations and/or management do you propose at this point?

Problem 4 - Page 4

Her nausea and headache have improved somewhat. You give her a tablet of oral acetaminophen + codeine, and suggest that she lie down and rest. Fifteen minutes later, she is sleeping comfortably, and two hours later awakens and feels well.

Questions:

- 7. What advice do you give her regarding this headache, her medications, and her habits?**
- 8. What advice should you provide regarding use of codeine and acetaminophen for recurrences of her headache?**
- 9. What other medications are available for her to abort her headaches if they recur? How are they best used? What risks do they carry?**

Problem 4 - Page 5

She stops smoking, changes to a different form of contraception, and stops using diet pills. She is unable to maintain reduced caffeine/energy drink intake. She has no similar recurrences for two years, and enters graduate school. She comes to see you after her first semester because her headaches have recurred, which she attributes to sleep deprivation. Despite attempts at improving her schedule, she now has 2-3 headaches each week, at least one of which is incapacitating for most of the day. She gets some relief from ibuprofen 600-800mg up to 4 times per day, but is afraid of taking it too frequently. She inquires about sumatriptan, which one of her friends uses, and also about preventive treatment.

Questions:

- 10. Discuss the mechanisms of action of sumatriptan.**

- 11. Is she is a candidate for preventive therapy for her headaches? Why? If so, what are the choices and what would you recommend?**

Problem 5 - Page 1

A 57 year-old left-handed woman comes to the emergency department after a seizure. She was in her usual state of good health until that afternoon when she began to speak “gibberish.” Her husband saw that she did not appear to understand what he said to her. After about 30 seconds, the left side of her face began twitching, then her left arm, then she became unresponsive and her entire body stiffened for five seconds, followed by 45 seconds of bilateral shaking. She then appeared asleep and groggy.

Past medical history: Headaches for the past two months, relieved with aspirin.

Social history: She smokes two packs of cigarettes per day. She drinks alcohol rarely, but had been to a party the night before admission where she had consumed enough to become intoxicated and did not go to sleep until 3 a.m.

Questions:

1. **Classify this acute event according to the International Classification of Epileptic Seizures.**
2. **What is the relationship of left versus right-handedness, and the degree to which language is left- versus right-lateralized?**
3. **To what cortical region would you localize the onset of this seizure? How could this explain her language problem? Characterize the type of language dysfunction that occurred at the start of the seizure.**
4. **What roles can sleep deprivation play in changing the seizure threshold? What about the temporal relationship between alcohol use and seizures?**

Problem 5 - Page 2

Physical examination reveals a lethargic woman who does not respond to verbal commands, but mimics movements shown to her. Over the next 10 minutes she begins to respond to some commands. The initial exam revealed that she did not move her left arm as well as her right, but this resolved quickly. She then began to exhibit left facial twitching, and repeated the sequence described on the previous page. Following this, she was unresponsive to noxious stimuli and had bilateral Babinski signs.

Laboratory data obtained at the end of the second seizure were remarkable for:

ABG: pH 7.11, pCO₂ 62, pO₂ 40 (room air)

Serum chemistry: AST 50, ALT 22, LDH 300, Na⁺ 140, K 5.5, Cl⁻ 108, HCO₃⁻ 10

CK 1,940

The patient gets intravenous phenytoin @ 20 mg/kg.

Questions:

- 5. Why was her arm transiently weak? What is the eponym for this condition?**
- 6. Discuss the abnormal laboratory values with reference to their causes and consequences.**
- 7. Discuss the pharmacologic properties of phenytoin, and comment on the risks and technique of its intravenous administration. How would fosphenytoin be administered here, and why might it be preferable?**

Problem 5 - Page 3

Over the next 30 minutes she begins to awaken. She was again briefly weak on the left, but this cleared quickly. As she became more alert, she spoke easily and effortlessly, but her words were nonsensical, and she did not follow commands. These language problems resolved over about two hours.

Her physical examination was then repeated:

Neurologic examination:

General: a fatigued woman with a small tongue laceration

- On simultaneous stimulation of both inferior visual fields, she failed to acknowledge the left-sided stimulus, even though she could see normally in the left visual field when it was stimulated alone.
- Left toe upgoing, right was downgoing
- Remaining exam normal

Questions:

8. **Name and discuss the etiology of her visual field testing results.**

Problem 5 - Page 4

Studies:

- Brain CT reveals a hypodense area in the superficial convexity of the right parietal region. It is a brightly enhancing mass with surrounding edema on the contrasted CT images.
- EEG revealed “focal slowing, and sharp waves” in the right parietal and posterior temporal regions.

Questions:

- 9. What is your differential diagnosis for this lesion?**
- 10. What does the EEG abnormality mean?**

Problem 5 - Page 5

After discussing the options for diagnosis and treatment with the patient, she agrees to an attempted resection of the lesion. A craniotomy is performed, and a meningioma is resected. She continued oral phenytoin as seizure prophylaxis.

One month postoperatively, she reports no further seizures or headaches. She now has a normal neurologic examination. She asks you how long she needs to continue taking phenytoin.

Question:

- 11. For patients with seizures as a group, what prognostic factors increase the risk for recurrent seizures upon discontinuation on antiepileptic agents? What action would you recommend for this patient?**

Problem 6 - Page 1

A 64 year-old right-handed man was sitting in his living room at home when he had sudden-onset problems talking, and right arm and leg weakness. His wife witnessed this, and called 911. He arrived in the Emergency Department 30 minutes later. The ED recorded his blood pressure as 170/90 on arrival, and reported that he had difficulty speaking, with weakness of the right face, arm, and leg.

Past medical history: insulin dependent diabetes mellitus and hypertension

Medications: He takes atenolol and insulin.

Questions:

- 1. What is the difference between dysarthria and dysphasia/aphasia? How will you distinguish between these on exam? What is the significance of this distinction in helping you localize the lesion?**
- 2. What tests should be done immediately, and what specifically are you looking for?**
- 3. What therapies are available for treatment and what is their mechanism of action?**

Problem 6 - Page 2

Physical exam:

Vital Signs: BP 169/85, P 90; RR 12; T 98.6° F
Neck: supple; right carotid bruit
CV: S1, S2, RRR; no S3, S4, or murmur; asymmetric wrist pulses
Skin: old healing ulcer in left heel.

Neurologic exam:

Mental Status:

- alert, frustrated
- spontaneous speech is halting, effortful; he can't name objects or follow commands; occasionally says "yes" or "I don't know"; cannot repeat what others say. Reads but cannot write formed letters, even with left hand.

Cranial nerves:

- patchy right visual field loss in each eye
- right lower facial droop
- decreased corneal on right

Motor:

- right arm and leg with less than antigravity power

Sensory:

- decreased modalities on the right arm and leg throughout, except also had poor sensation in left leg as exam moved more distally

Reflexes:

- deep tendon reflexes are brisk in the right arm and knee, absent at both ankles, and diminished in the left arm and knee.
- plantar response is extensor on the right.

Coordination:

- good left finger-to-nose, cannot test on the right due to weakness

Gait:

- he is unable to stand

Studies/labs:

- Serum glucose: 131
- brain CT: no blood or acute lesion

Questions:

4. Discuss the sensory pathways likely involved and the location of involvement for his sensory loss on the right body versus his left leg.
5. What treatment could you give him for causes of his weakness, and what side-effects will you discuss with the patient and his family?

Problem 6 - Page 3

He is treated with tPA at one hour and 20 minutes, and admitted to the Neuro-ICU for close monitoring of vital signs and neurologic status.

At six hours, his exam showed BP 145/85, normal level of consciousness, fluent speech, normal comprehension, and normal ability to name, repeat, read, and to write. He has a slight pronator drift and 4/5 power in the right leg.

Questions:

- 6. What are the possible causes of thromboembolism in this man?**
- 7. What is the vascular anatomy of his problem, and specifically, why was he weak, numb, and aphasic?**
- 8. What tests would you like to get to evaluate the etiology of his stroke, and what are you looking for?**

Problem 6 - Page 4

A cerebral arteriogram demonstrates a 75% left internal carotid artery stenosis just above the carotid bifurcation. His blood pressure is still elevated at 150/85.

Questions:

- 9. What treatment would you suggest for the vascular lesion, and what evidence exists to support your recommendation?**

- 10. Why is it important to avoid aggressively treating his blood pressure in this acute setting? What are the current recommendations regarding treatment of blood pressure in acute stroke.**

Problem 7 - Page 1

A 67 year old retired man was in generally good health when he developed achy, midline, low back pain while playing golf. The pain was different than the muscle fatigue and strain he had previously experienced while golfing; despite the pain, he was able finish his planned 18 holes for the day. After arriving home he took 400mg of over-the counter ibuprofen and used a hot pad on the area, obtaining partial relief. The next day, the pain persisted. It again responded partially to ibuprofen and did not limit his activities.

Later, while twisting to lift groceries out of the back seat of his car he experienced a sudden increase in the pain with a severe electrical or burning character that radiated from his upper buttocks down the side of his right leg. The radiating pain resolved within minutes after taking more ibuprofen, applying the hot pad, and lying down.

Over the next week, his back pain varied in intensity but was never completely gone. When he tried to play golf again, the buttock and leg pain re-occurred.

Upset over his inability to play golf, he scheduled an appointment with his primary care doctor.

Questions:

- 1. What is your differential diagnosis of this patient's problems at this point?**
- 2. In the absence of focal or debilitating neurologic dysfunction, should bed rest be recommended to this patient? How do topical treatments like heat or cold work in situations like this?**

Problem 7 - Page 2

PMH:

Hypertension, stable on lisinopril 10mg daily

Hyperlipidemia, stable on simvastation 40mg daily

“Skin Cancer” removed from left ear 2 years ago; patient unaware of type

SH:

Nonsmoker x 30 years, except for 1 cigar monthly “on poker night;”

1 glass of wine with dinner daily, occasional beer or whiskey with friends (1-2/month)

FH:

Father died with cerebral hemorrhage; Mother alive age 87 with “bad arthritis”

ROS:

Arthritic knee pain; Nocturia x 2 most nights; all other ROS negative

Physical examination:

General: A well-appearing older man

Vital Signs: BP 148/84, P 72, T 37.4° C

HEENT: 2mm nontender ulcerated superficial lesion on the right scalp near hairline

Back: Marked tenderness to midline spine palpation at L3 through L5 levels; paraspinal muscle spasm evident bilaterally through lumbar area

Neurological examination:

Motor: Normal muscle mass; 5/5 power throughout, except possibly 4+/5 right ankle dorsiflexion

Sensory: Mild, symmetric decreased position/vibration sense in both feet

Reflexes: Upper limbs normal; Knees 2+ symmetrically; left ankle 1+, right ankle absent.

Remainder of exam normal

Questions:

3. Based on the physical examination, where would you localize this lesion?
4. Would you institute any therapy at this point? If so, what? Why?
5. What neuroradiologic studies would be most useful in clarifying the diagnosis?

Problem 7 - Page 3

The patient is advised to continue NSAIDs and accepts a referral to physical therapy for “back school” and an exercise program designed to facilitate spine health.

Plain films of the lumbar spine showed multilevel disk space narrowing and prominent osteophytes, but no fractures or spondylolisthesis.

After two weeks of therapy, the patient calls to say the pain is still present, and maybe worse. He is disappointed that he still can't play golf without considerable discomfort and radiating pain into his leg.

Questions:

- 6. How do the X-ray results help narrow the differential diagnosis? What information do they provide that augments the exam findings? What other tests might further clarify the disease process?**
- 7. What is the role for narcotics in managing the pain at this point? What other alternatives might be offered? Compare and contrast acute pain and chronic pain. How do treatment approaches differ?**

Problem 7 - Page 4

A few days later the patient requests an urgent appointment because he is dragging his right foot when he walks. The pain is worsening and now radiates into his leg most of the time.

On exam, he has 2/5 strength on ankle dorsiflexion; inversion and eversion are 3/5; there is a loss of sensation to pin and light touch that extends from the lateral right calf down to the big toe.

Questions:

- 8. What treatment options might be suggested now? What criteria predict successful or unsuccessful surgical treatment of back pain?**

- 9. If he had developed neurologic dysfunction bilaterally, or at multiple spinal levels, or was found to have acute urinary retention, how might your differential diagnosis change?**

- 10. What are the key features that help distinguish between the syndromes arising from the cauda equina, the conus medullaris, and the lumbar spinal cord ?**

Problem 8 - Page 1

A 70 year-old white man is found unresponsive by his son. The last time he was seen, three days earlier, he felt well in his usual state of health.

He arrives at your hospital's Emergency Department. On initial exam, he is an elderly, unresponsive man in a cervical collar with the following vital signs: BP 190/100, HR 100, 28 snoring respirations per min and axillary temperature 39.1 C. The patient's son states that his father has been 'depressed' for several months about his numerous health problems which include coronary heart disease, hypertension, diabetes mellitus, 'kidney problems', and a prior 'stroke'. The patient has a 70 pack-year smoking history and drinks 3-4 shots of liquor a day with occasional binges.

Questions:

- 1. What are your immediate concerns and plans for diagnosis and management?**

Problem 8 - Page 2

On further exam, you determine the following: his eyes remain closed to even painful stimuli, and when opened by you he does not appear to regard you, nor does he follow commands. Brain stem reflexes appear intact, but he tends to gaze and turn his head to the left. Occasionally, he moans and weakly pulls his left arm or leg away from painful stimuli, but he does not move his flaccid right extremities. Deep tendon reflexes are increased on the left, absent on the right. He has bilateral Babinski signs.

General examination is notable for abrasions on the right side of the patient's head and face but no Battle's sign, raccoon's eyes or hemotympanum. There are hypertensive changes and no venous pulsations on funduscopy. He is tachycardic and the precordium is hyperdynamic with prominent PMI but no murmurs, rubs or gallops. You hear crackles in the lung fields posteriorly on both sides. There are a number of nevi on the patient's skin, two of which look 'suspicious'. The remainder of the general examination adds nothing else useful.

Questions:

- 2. What constitutes coma? Is this patient in a coma? What are the neuroanatomic bases for coma?**
- 3. What features above help to localize the cause of this patient's unresponsiveness? Where do you localize the cause?**

Problem 8 - Page 3

The initial management and work-up for your patient's unresponsiveness is well under way. You look at the patient's CT with the radiology resident and then return to see your patient back in the ED. You notice that now his left pupil is significantly larger than the right and is not reacting to changes in light intensity.

Questions:

- 5. What structural causes for unresponsiveness are particularly likely in this patient?**
- 6. What is/are the mechanism(s) by which they produce changes in level of consciousness?**
- 7. What is the significance of the pupillary changes?**
- 8. What management options pertinent to the pupillary changes might you consider at this point?**

Problem 8 - Page 4

You speak with the patient's son about the acute change in his father's condition and the results of the CT, and agree to pursue aggressive treatment, but to no avail. The patient loses pupillary light reactivity bilaterally, followed by loss of oculo-cephalic and oculo-vestibular reflexes, corneal reflexes, and then gag and cough reflexes. At about the same time that gag and cough reflexes were lost, the patient became markedly hypertensive and bradycardic and then markedly hypotensive.

Questions:

9. **Briefly describe the neuroanatomy of the reflexes referred to above. What is the pathophysiologic correlate of this sequence of events?**
10. **Define "brain death." What terms might be better used in place of "brain death"? What else has to be performed/documentated in this case if brain death is to be diagnosed? Is brain death required for withdrawal of medical care?**