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AN EVIDENCED-BASED CURRICULUM FOR CLINICAL REASONING EDUCATION IN NOVICE STAGE LEARNERS: A NEUROLOGY STROKE CURRICULUM FOR PRECLINICAL PHYSICIAN ASSISTANT EDUCATION

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AN EVIDENCED-BASED CURRICULUM FOR CLINICAL REASONING EDUCATION IN NOVICE STAGE LEARNERS: A NEUROLOGY STROKE CURRICULUM FOR PRECLINICAL PHYSICIAN ASSISTANT EDUCATION

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Abstract

Introduction

Preventable medical error represents a top 10 cause of mortality in the United States and misdiagnosis is the most common, costly and dangerous form of medical error. An estimated 80,000 people die every year because of misdiagnosis related harms. Although various approaches to the problem of misdiagnosis have been explored, research and expert consensus suggest that educational interventions are most likely to improve the problem of misdiagnosis. Specifically, improved educational models in clinical reasoning are thought to be the most effective approach to improving diagnostic capacity among healthcare teams. The majority of data and research on the problem of misdiagnosis focuses on physician performance. Other healthcare providers, like physician assistants, play a significant role in the modern healthcare system but less is known about the diagnostic performance of physician assistants. Although existing data suggest that physician assistants, like their physician counterparts, may be associated with diagnostic error, there is virtually no mention of diagnostic performance or preventable medical error in the literature.

Methods

Working from the premise that physician assistants would benefit from improvements in clinical reasoning training, the study set out to design a curriculum in diagnostic proficiency. Using the literature on best practices and evidence-based principles, an experimental curriculum in diagnostic proficiency was developed and delivered at a Midwestern physician assistant training program. The experimental curriculum focused on neurology with an emphasis on diagnosis of stroke. The rationale for this content emphasis is that neurology represents a significant source of misdiagnosis with stroke being a frequent cause of misdiagnosis related harm. The form of the study was a within-subjects comparison between trainee performance on material covered by the experimental curriculum (neurology, stroke) versus

trainee performance on content covered by the institution's conventional curricula. The effect of the experimental curriculum on the diagnostic capacity of the trainees was measured using case studies and illness script exercises. The study compared the trainee's performance on conditions covered by the experimental curriculum (anterior stroke and posterior stroke) against conditions covered by the institution's existing curriculum (Henoch-Schönlein purpura and acute adrenal insufficiency). Additionally, student attitudes toward the curriculum of diagnostic proficiency were assessed using a series of survey questions.

Results

Trainee performance on the conditions covered by the experimental curriculum demonstrated a diagnostic accuracy of 88% (posterior circulation stroke case study) and 62% illness script repleteness (cardiogenic anterior stroke). Trainee performance on conditions not covered by the experimental curriculum (taught using the institutions current practices) demonstrated a diagnostic accuracy of 46% (Henoch-Schönlein purpura case study) and an illness script repleteness of 16% (acute adrenal crisis). Student attitudes toward the experimental curriculum were positive with qualitative analysis showing favorable student comments correlating with the evidence-based principles of the experimental curriculum.

Discussion

This study highlighted the importance of educational approaches to the problem of misdiagnosis among physician assistant trainees. Although confounding factors are present, the study provides a training model for diagnostic proficiency that may serve as a starting point in improving the clinical reasoning education for physician assistant programs.

Chapter I. Introduction

Misdiagnosis: A Leading Cause of Mortality

Over two decades ago, The Institute of Medicine Committee on Quality of Health Care in America issued a report "To Err Is Human: Building A Safer Health System"[1]. To the shock of both the public and the medical profession, the report detailed that an alarming 98,000 people die every year from medical errors. Other causes of death, such as motor vehicle accidents, breast cancer or AIDS, received much more attention in the media but for many, medical error did not register as a significant source of mortality. Subsequent studies confirmed the findings of the IOM report. Preventable medical errors represent one of the top 10 causes of mortality every year in the United States[2]. Taking more lives than diabetes or septicemia, fatalities from preventable medical errors range between 70,000 and 100,000 annually.[3] To mitigate the impact of preventable medical errors, researchers initially sought to describe the root causes of medical error. Multiple sources of medical error were described. Categories of error included procedural errors such as wrong site surgeries, clerical errors such as misplaced abnormal lab values and errors in clinical reasoning that included clinicians arriving at false judgments about a patient's medical problem[2]. Researchers found that among all sources of medical error the most common, costly and dangerous kind of medical error was a failure of clinical reasoning in the form of misdiagnosis.[2]

In a more recent study Newman-Toker and other researchers corroborated previous mortality estimates of mortality associated with misdiagnosis citing up to "40,000 to 80,000 misdiagnosis related deaths in U.S. hospitals annually."[3] This study further identified three pathophysiologic categories (the "big three") of serious misdiagnosis related harms: vascular events, infections, and cancers. This study analyzed 11,592 diagnostic error cases and demonstrated that the "big three" diseases represent 74.1%

of the high severity cases.[3] Stroke or cerebrovascular accident was the most common of all causes of serious misdiagnosis related harms. [3]

These findings are consistent with another systematic review published in 2022: "Diagnostic Errors in The Emergency Department: A Systematic Review."[4] This systematic review deployed a similar methodology and sought to identify causal factors associated with misdiagnosis in the emergency department setting. This study reported a crude misdiagnosis rate of 5.7%, low by comparison with other studies, but when the associated harms are extrapolated on an annual basis, the toll is considerable.[4] With "approximately 130 million emergency department visits per year in the United States, 7.4 million (5.7%) are misdiagnosed, 2.6 million (2.0%) suffer an adverse event as a result and about 370,000 (0.3%) suffer serious harms from diagnostic error."[4] The study also identified salient correlations in the identified cases of misdiagnosis. First, five conditions accounted for 39% of serious misdiagnosis related harms: #1 stroke, #2 myocardial infarction, #3 aortic aneurysm/dissection, #4 spinal cord compression/ injury, #5 venous thromboembolism. Stroke, the top serious harm-producing disease, is misdiagnosed an estimated 17% of the time. Unsurprisingly, the researchers also found that "for a given disease, nonspecific or atypical symptoms increased the likelihood of error."[4] A statistically significant example of this problem is once again stroke where the presence of dizziness or vertigo "increases the odds of misdiagnosis 14-fold over motor symptoms (those with dizziness and vertigo are missed initially 40% of the time)."[4] The most compelling causal factor identified by the researchers in the prevalence of misdiagnosis is cognitive errors. In their root analysis of emergency department diagnostic errors, the researchers found "errors were mostly cognitive errors linked to the process of bedside diagnosis." The researchers' review of both medical malpractice claims and incident report data indicate that "serious misdiagnosis- related harms involved failures of clinical assessment, reasoning or decision making in about 90% of cases."[4,5] Failures in clinical reasoning is a recurrent theme in the analysis of preventable medical error. The next question posed by researchers is what causes failures in clinical

reasoning? When a patient presents to a clinician they typically do so with a problem. The problem may be some sort of pain e.g. sore throat, chest pain or a disturbance of normal physiologic functioning like insomnia or depressed mood. Through the process of clinical reasoning a clinician will hopefully connect the patient's initial problem with an accurate diagnosis. Diagnosis, from the Greek prefix dia (δια) "through" and suffix gnosis (γνοσισ) "knowledge," is a fundamental naming of the underlying cause of the patient's problem. This naming or explanation of the problem's cause drives all subsequent clinical decision making[6,17]. If the given diagnosis is incorrect, all subsequent clinical decision-making including treatment decisions and prognosis will be misguided. Broadly speaking, misdiagnosis may be thought of as a failure to find a timely and actionable explanation for a patient's problem. The growing consensus among researchers is that the most impactful approach to the problem is through improved training in clinical reasoning[7]. In an article from *Diagnosis*, Andrew Olson and other researchers attempted to achieve a consensus regarding an educational approach in their "Competencies for Improving Diagnosis: An Interprofessional Framework for Education and Training in Healthcare." With diagnostic error contributing to such significant morbidity and mortality annually in the United States, Olson and colleagues asserted "outcomes of our current education programs with respect to diagnosis are not enough; that is, the products of our current education programs-healthcare professionals in practice-are not achieving desirable levels of diagnostic performance."[7] Moreover, current clinical educators have a "moral obligation to equip tomorrow's healthcare professionals to be effective members of diagnostic teams." Olson and his colleagues identified the most urgent area of improvement for educational interventions is individual competencies for diagnosis. The research group identified several areas for improvement: gathering data, formulating an accurate problem representation, formulating an appropriate differential diagnosis, hypothesis refinement and cognitive debiasing.

Misdiagnosis Among Physician Assistants: An ill-defined Problem

The harms associated with misdiagnosis are significant and researchers have narrowed the causal focus of this problem to deficits in clinical reasoning. Furthermore, it has been suggested that improvements in our training models in clinical reasoning are the strategies most likely to be effective in mitigating the problem of misdiagnosis. However, these judgments have been informed by research that predominantly addresses physicians. This research does not address the prevalence of misdiagnosis among non-physician healthcare providers. A growing segment of the United States healthcare delivery system includes "advanced practice providers."[8] These providers include advanced practice registered nurses (APRNs), nurse practitioners (NPs) and physician assistants or physician associates (PAs). A recent study published in the British Medical Journal estimated that up to 25% of healthcare encounters are provided by advanced practice providers like NPs and PAs.[9] Despite the growing presence of physician assistants in the US health care system, little research has been conducted into the prevalence of diagnostic errors among PAs. One study, conducted by Brock and other researchers performed an analysis of data from the national practitioner data Bank in 2013. This study used medical malpractice claims as an index for medical errors. Brock et al found that physician assistants and nurse practitioners were less likely to make a medical malpractice payment than their physician counterparts.[10] Several factors may contribute to this statistic. Physicians tend to see larger patient volumes with higher acuities.[11] This is the case in some emergency department models where physicians see higher acuity patients and the advanced practice providers the lower acuity patients. Additionally, as a result of the "doctrine of respondent superior," physicians, rather than the APP, may be the listed provider for a medical malpractice payment but the fault may belong to an advanced practice provider acting as the

physician's employee[10]. An important finding in this study was the discrepancy between medical malpractice claims regarding diagnostic errors. A disproportionately higher number of medical malpractice cases for physician assistants belong to the category of misdiagnosis. For physicians, the claims related to diagnostic errors represented about 32% of total physician claims. For physician assistants diagnostic error represented over half of their malpractice claims at 52.8%.[10] The disproportionate percentage of claims associated with diagnostic error raises the question could physician assistants be vulnerable to diagnostic error?

Misdiagnosis Among Physician Assistants: Beyond Medical Malpractice Data

Although specific cases of diagnostic failure involving PAs are available for review, physician assistants' aggregate rates of misdiagnosis and associated harms are obscured by current reporting and billing practices. Until reporting mechanisms allow for more detailed data on misdiagnosis among PAs, other data sources may provide insight. Two slightly tangential sources of data that may provide insight into the status of misdiagnosis among physician assistants are 1. Evaluating high-stakes examinations as a reflection of PA training models and 2. Evaluating the status of misdiagnosis as a problem represented in the literature of physician assistants.

Epidemiologic studies have demonstrated that several conditions are overrepresented in the problem of misdiagnosis. The correlating question is do physician assistant training programs effectively incorporate high risk conditions into their curricula? One approach to these data, surveying the curricula of programs, would require correspondence with program officials to determine the amount of time high-risk conditions are allocated in the program curriculum. However, response rates and sample issues make this approach unrealistic. Another data source is the construction of and performance on Physician Assistant National Certification Exam (PANCE). The PANCE is a summative exam that all PA students are required to pass before they are allowed to practice as a physician assistant. The high-

stakes nature of this exam compels PA programs to allocate time to content that is proportional to the amount of material on the national certification exam. The content and task areas evaluated by the exam are published by the National Commission on the certification of Physician Assistants (NCCPA) in a document called the "PANCE blueprint" (https://www.nccpa.net/become-certified/pance-blueprint/). The PANCE blueprint specifies the conditions (diseases, illness, and injuries) as well as the task areas (performing an exam, interpreting lab values) that are covered on the exam. The specific disease areas (e.g. cardiology) are listed by percentages showing how much of that disease area is represented on the exam. With an average program length of 27 months, physician assistant training must be judicious in terms of time allocated to content. Consequently, programs spend a greater amount of time covering diagnoses associated with cardiology that represent 12% of the exam than diagnoses associated with nephrology that represent only 5% of the exam. Neurology is the body system shown to be overrepresented in the problem of misdiagnosis with stroke being the number one cause of serious harms[4]. In the Physician Assistant National Certification Exam, neurology represents 7% of the total exam. Importantly, neurology is the content area in which most students frequently underperform on the national certification exam (Appendixes 1-2). Most recently, the 2022 PANCE cohort (11,260 first-time test-takers nationwide) scored 74% on neurology (Appendix 1). This was the lowest score of all content areas even though neurology, at 7% of the exam, is weighted more heavily than six other content areas against which neurology underperformed: dermatology 5%, genitourinary system 5%, hematologic system 5%, infectious disease 6%, psychiatry/behavioral science 6% and nephrology 5%. Summative evaluations are not thought to be direct indicators of diagnostic performance. However, it is noteworthy that neurology - and possibly strokes - consistently underperform on the Physician Assistant National Certification Exam (Appendices -2).

Another point of interest is the methodology by which the PANCE blueprint is constructed. The specific details and procedures are not widely known but the National Commission on the Certification

of Physician Assistants website describes the formation of the content blueprint in this way: “The content blueprint is a set of specifications that describes the relative proportion of questions from each content category and task area that should be included on a test form. This blueprint is the output of a practice analysis, which is an analysis of the various diseases and disorders PAs encounter and the skills they use in practice. The last practice analysis was conducted in 2015. A committee of PAs used survey data gathered as part of the 2015 practice analysis as well as their own personal experience to set the blueprint that became live in 2019.”[12] The formation of the blueprint relies upon the voluntary completion of surveys among currently practicing physician assistants in the United States. These surveys are then collated and studied by a committee of PA's who make recommendations also factoring in “their own personal experience.” This process is problematic for several reasons. First, is the problem of selection bias. Only PAs completing the surveys will have their opinions and experience incorporated into the blueprint. Consequently, there may be a disproportionate number of respondents that represent specialties or segments of the PA workforce that do not represent the workforce as a whole. Importantly, there is no mention of how crucial epidemiologic trends such as preventable medical errors are used to inform the construction of the blueprint. While the opinions of practicing clinicians are an important source of data, the epidemiology of misdiagnosis-related harms has been shown to be a public health problem.

One additional consideration regarding misdiagnosis among physician assistants is the extent to which preventable medical error and misdiagnosis are recognized as potential problems within the profession. An indicator of this data point is the representation of preventable medical errors and misdiagnosis among physician assistants in the literature. Studies that describe the phenomenon, the prevalence or that even mention misdiagnosis among physician assistants are virtually absent from the current literature. Moreover, the leading association journals and educational journals of physician assistants have yet to even mention the words “misdiagnosis, preventable medical error” as a central

theme in any of the publications (using the search terms physician assistant along with modifiers of misdiagnosis and medical error). There is minimal recognition of the problem of misdiagnosis in the professional communities of physician assistants. This gap in the literature and apparent lack of stakeholder interest regarding misdiagnosis among physician assistants stands in contradistinction to physician professional communities. Physicians have responded to the problem of misdiagnosis with a rich development of literature, clinical and public health initiatives, as well strong institutional guidance by The Society for Improvement of Diagnosis in Medicine.[13]

Problem Summary

A review of the literature thus far has adduced the following: 1. Misdiagnosis is a central cause of medical harm and is frequently the result of a failure in clinical reasoning 2. Scholarly consensus suggests that improvements in clinical reasoning education would improve diagnostic performance 3. Diagnostic performance among physician assistants is not well documented but if similar to their physician counterparts, is in need of improvement 4. Physician assistant training models do not factor in the epidemiology of misdiagnosis and public harms in the formation of their curricula. 5. Definitive data are currently unavailable but preliminary studies suggest that PAs may contribute disproportionately to the high percentage of diagnostic errors[9] 6. Strokes represent the number one cause of significant misdiagnosis related harms 7. Strokes belong to the field of neurology that is overrepresented in the causes of misdiagnosis related harms and represents only 7% of the PANCE blueprint content areas (Appendixes 1-2).

Purpose of the study

The purpose of this study is to determine if an evidence-based approach to clinical reasoning education will improve diagnostic capacity in novice stage learners as measured by performance in illness script repleteness and diagnostic accuracy[14-15]. The curriculum was developed using best practices and

evidence-based principles from the literature on clinical reasoning education. These principles include: 1. The structuring of clinical knowledge through the deliberate and ongoing use of frameworks and illness scripts 2. Evidence-based integration of physical examination and diagnostic evaluations in clinical reasoning 3. Titration of cognitive load and contextual elements in the training process to optimize learning 4. Intensive coverage of high-risk conditions and problems through spaced-repetition and high-risk condition highlights. These principles were used to design a curriculum that includes interactive lectures, problem-based learning (small group case studies and web-based PBLs), low stakes assessments (quizzes), summative assessments (MCQ examinations) and assessments of entrustable professional activities (OSCEs - objective structured clinical encounters). The scope of this curriculum is limited to neurological problems with an emphasis on strokes as they are overrepresented in epidemiological studies of misdiagnosis.[3] Based on the implementation of this curriculum, this study aims to answer the following research questions:

1. Does exposure to the proposed curriculum of diagnostic proficiency improve illness script repleteness[67] in novice stage learners?
2. Using the key features assessment method[79], does exposure to the curriculum enhance diagnostic capacity in novice stage learners?
3. What are the students' impressions of the curriculum?

Assumptions and Limitations

This study sought to describe the effects of an improved training model on clinical reasoning in preclinical physician assistants. Due to the paucity of data on the diagnostic performance among PAs, the study assumes that there is a problem with misdiagnosis among PAs. Moreover, the study assumes that the problem may be corrected through improvements in a training model as there is also a lack of literature regarding the kinds of training models guiding physician assistant education. Proceeding with

the current study based on these assumptions rather than data-based judgments, warrants consideration of several critical questions. Why not direct research efforts toward more adequately defining the problem before proposing and studying solutions? That is, why not study the mechanisms of medical error reporting among PA's and study solutions to improve those mechanisms? Similarly, why not map out the training models and curricula in PA education in clinical reasoning before suggesting improvements of those models? The response to these questions and the impetus of the current study arises from the urgency of the problem. Waiting for the problem of misdiagnosis among PAs to be fully described may take years to accomplish and require significant changes to the medico-legal system. Pragmatically, there is sufficient data to suggest that preventable medical error, in the form of misdiagnosis, does occur among physician assistants. The prevalence of physician assistants in the current US health care system will increase rapidly over the next few years and the Bureau of Labor Statistics estimates that "From 2019 to 2031 … the number of physician assistants will increase from 120,000 to 178,000 (48 percent growth)."[9] The significant annual mortality and morbidity associated with misdiagnosis combined with the projected increase in physician assistants in the health care system warrants urgent evaluation of strategies to reduce harm.

Chapter II. Review of the Literature

Definition of terms: A Narrative Review of Diagnosis and Clinical Reasoning

In addition to defining terms specific to this study and its related domains, this section provides a narrative review of the process of diagnosis and clinical reasoning. The purpose of this review is to provide a normative description of the model of clinical reasoning that reflects not only the author's understanding of the diagnostic process but also the prevailing view in the contemporary practice of medicine.

Diagnosis is part of a broad form of thinking and decision-making that may be referred to as clinical reasoning. Clinical reasoning arose within the background of Western enlightenment empiricism and has seen refinements in the post-enlightenment period.[6] The practice of most clinicians deploys a pragmatic or "abductive" form of reasoning with the following salient features: gathering of data, hypothesis generation, hypothesis testing, hypothesis refinement and hypothesis verification.[6,16]

Gathering data, the first component of the diagnostic process, is embedded within the procedural activities of acquiring a medical history and performing a physical examination. Patients present to a clinician some sort of problem or complaint which represents the reason for which they are seeking care. Acknowledging that patients may have multiple complaints, the phrase "chief complaint" has been used to denote the problem about which the patient is most concerned. Arriving at an accurate identification of the chief complaint will set the trajectory for the diagnostic inquiry. Importantly, the type of problem thought to be under investigation dictates a range of methodological considerations.[13] Psychiatric problems have a method of evaluation that is proper to those problems and orthopedic problems have a method of evaluation that is proper to those. Confusing the entire class of chief complaint is not common except in exceptional cases but more nuanced difficulties in defining the chief complaint are common. If the patient reports being "fatigued" when they are short of breath or complains of a "sprained ankle" when in fact they have pain at the base of the fourth and fifth metatarsal, the patient's unintentional misreport can set a false direction for the diagnostic process. Throughout the history taking process it is crucial for the clinician to identify any linguistic ambiguities or potential misunderstandings that could mislead the diagnostic process. Locating a complaint within a range of diagnostic possibilities relies upon clinician having an adequate "storehouse" of conditions to choose from or an adequate base of knowledge. If the clinician is unable to consider diagnostic possibilities because of knowledge gaps, a correct diagnosis is essentially impossible.[6,16]

Having arrived at an accurate chief complaint, a clinician can be reasonably confident that the complaint represents a problem in a class of conditions - joint pain an orthopedic problem, unilateral weakness a likely neurological problem. By taking a history the clinician will use a series of questions designed to elicit the nature of the underlying complaint. If a patient complains of pain for example, the clinician will ask a series of questions to characterize the pain: site, onset, character, radiation, associations, time course, exacerbating/relieving factors and severity. In many cases, the patient's answers to these questions will already bring into consideration several diagnostic possibilities. If a 22-year-old female complains of right frontal throbbing headache associated with nausea and photophobia, migraine headache will no doubt be on the list of possibilities. Indeed, to novice clinician the diagnostic task is already complete-the patient clearly has migraine headaches. However, the clinician must consider worst case possibilities for various complaints. In the case of new onset of severe headache, a clinician would certainly consider intracranial neoplasm and intracranial hemorrhage. In addition to the characteristics of the complaint the clinician will also consider the patient's medical, family medical and social history as possible clues to the patient's underlying problem. All the data gathered in the history begins to create a range of probable and possible explanations for the patient's problem. To further refine or expand this range of possible explanations the clinician will perform a physical examination.

Although the process and techniques of physical examination have evolved in the past few decades, all physical examinations begin with vital signs that are an index of the patient's most basic cardiovascular functioning. Markedly abnormal vital signs warrant a prompt initial investigation before proceeding with other areas of examination. What comes next in the examination process is largely a function of the underlying complaint. Most exams will include a "general" component but the specific techniques that follow will be tailored to the patient's chief complaint. Increasingly, clinicians are beginning to home in on techniques and exam practices that have the greatest return in terms of accuracy.[6]

Throughout the history and physical examination clinicians are usually well on their way to developing a differential diagnosis. Many patient safety authorities have described the importance of a well-formulated differential diagnosis.[16] Although categories vary, the components of a robust differential are thought to include the following components: probabilistic diagnosis (the most likely diagnosis), prognostic diagnosis (possible conditions representing worst case scenarios) and pragmatic diagnosis (conditions that respond to safe and reasonable treatments)[6,16]. Probabilistic conditions are the most common and likely conditions to explain a patient's underlying problem. Understanding the epidemiology of a particular condition informs the background judgments of probabilistic thinking. Prognostic possibilities include those conditions that represent worst case scenarios and could have dire prognostic implications for patients if they were to go undiagnosed. This principle is illustrated by consideration of subarachnoid hemorrhage or intracranial neoplasm in the case of new onset headaches. The pragmatic component of differential diagnoses includes conditions known to respond well to treatment. An example of this type of diagnosis would be a pediatric patient with nausea and vomiting is given intravenous fluids in lieu of extensive laboratory workup and upon seeing clinical improvement a diagnosis of dehydration is clinically established and the patient has been appropriately treated.[6,16]

After a clinician has formulated a sound differential diagnosis it will be necessary to test each of the items on the differential as a kind of hypothesis. Hypothesis number one is a probabilistic diagnostic possibility: "The patient is experiencing a migraine headache." Hypothesis number two is a prognostic possibility or a "can't miss" diagnosis: "The patient could be having a subarachnoid hemorrhage." Prognostic possibilities include worst case scenarios that would have a deleterious prognostic effect on the patient's outcome. These prognostic or worst-case scenarios must be "ruled out." That is to say, the clinician must become reasonably certain that a "can't miss" condition has been taken out of consideration. In the case of new onset of headache where there is concern to rule out subarachnoid

hemorrhage, the clinician must determine what kind of diagnostic test would have the adequate sensitivity such that if the test were negative, we could say that subarachnoid hemorrhage is no longer a consideration. The clinician proceeds to order a noncontrast computerized tomography (CT) scan of the head knowing that the sensitivity for detecting a subarachnoid hemorrhage is 97% in the first 12 hours of presentation with headache.[23]

The 22-year-old female with the new onset of severe headache undergoes a CT of the head which is read as "normal" and no subarachnoid hemorrhage is found. This datum combined with the fact that the patient's headache is almost completely relieved with treatment strongly implies that the prognostic diagnosis (subarachnoid hemorrhage) has been excluded and that the pragmatic, clinical diagnosis (migraine headache) has assumed the leading position in the differential. Because there are no physical exam findings, lab tests, or diagnostic imaging that "disclose" the presence of a migraine, the clinician will have to rely on clinical criteria and pragmatic response to therapy to establish this diagnosis. The clinician recalls the "POUND" mnemonic criteria: Pulsatile (throbbing), One day in duration (4-24 hours without medications), Unilateral, Nausea and Disabling intensity.[16] If the patient's headache, the clinician recalls, satisfies four out of the 5 pound positive criteria, the positive likelihood ratio for a migraine headache is 24. With the worst-case scenario ruled out, a favorable response to treatment and a strong evidentiary basis for a positive diagnosis, the patient is diagnosed with migraine headache.

The process of hypothesis verification can be complicated. If the patient in the case study was not responding to treatment and she presented 24 hours after the headache's onset, a negative CT scan would not have been sufficient to rule out a subarachnoid hemorrhage. In cases like these, it is recommended that patients undergo a lumbar puncture to assess for the presence of blood products in the cerebrospinal fluid. If an abundance of red blood cells persists across collected tubes of cerebrospinal fluid or there is evidence of blood products such as xanthochromia this would be

suggestive of a subarachnoid hemorrhage[18]. In this case, cerebral angiography will be required as it is the gold standard test for detecting cerebral arterial defects leading to subarachnoid hemorrhage.

The Importance of Diagnosis in The Clinical Process

A review of the diagnostic process reveals that diagnosis is not just a mere component of medical reasoning but represents a kind of lodestar for the clinical encounter. A correct diagnosis is the fundamental identification of the patient's underlying medical or surgical problem that illuminates all other elements of the clinical process. If correctly identified, then everything that is important is well-known-natural history of the underlying problem, prospects for management, and reasonable expectations for prognosis. In the case of misdiagnosis, everything important is hidden. A malpractice case illustrates the possible impact of misdiagnosis on a patient's clinical course. A 37-year-old women presented with a new onset of severe headaches; she described them as the "worst headache" of her life headache and was misdiagnosed with migraines.[19] The patient's family physician performed a CT of the brain which was read as normal but was performed eight days after the onset of headaches when the sensitivity for subarachnoid hemorrhage is known to be unacceptably low.[23] The patient's headaches persisted and a neurologist was consulted and diagnosed the patient with viral meningitis. The patient died of subarachnoid hemorrhage two days after being evaluated by the neurologist. Effective and timely treatment opportunities for subarachnoid hemorrhage were missed resulting in patient death. Misdiagnosis is a kind of totalizing error that gives rise to further misunderstandings and further misguided actions. Without correct diagnosis successful treatment of underlying disorders is impossible except in the most benign self-limiting conditions. The totalizing nature of diagnostic error gives us an indication of why misdiagnosis is so dangerous in terms of morbidity and mortality and so costly in terms of medical malpractice expenditure. It does not, however, give us any indication of why misdiagnosis is so common.

Obstacles to Diagnosis

Analyses of the problem of misdiagnosis have revealed numerous potential obstacles to effective diagnosis. These obstacles are best described by organizing them into the foci in which they commonly arise: complex nature of disease, difficult patient encounters, healthcare system dynamics and factors affecting individual clinician performance.[6]

Diseases arise within complex biological systems and consequently have protean manifestations. As clinicians attempt to compress this vast complexity into recognizable patterns, difficulties are inevitable. The complexity of disease processes produces a great deal of overlap in presentations. Two or more disease processes may have very similar presentations and it is only through a careful diagnostic process that clinicians arrive at the correct diagnosis. Consider the case of atypical hemiplegic migraines versus stroke. Although not common, hemiplegic migraines will present with the sudden onset of unilateral weakness that is clinically indistinguishable from a thromboembolic stroke. It is only after a patient has been fully evaluated with the appropriate imaging modalities that these two conditions can be distinguished from one another. Moreover, advances in imaging and molecular technology have brought about an exponential increase in the number of disease states that warrant clinical recognition. Using the most recent ICD codes as a rough index for the number of diseases, the Centers for Medicare and Medicaid Services listed over 69,000 disease codes in ICD-10.[34] The sheer volume and complexity of disease data can easily outstrip the cognitive capacity of even the best trained clinicians.

Another important source of obstacles to effective diagnosis is the dynamics of healthcare systems. Many citizens of the United States experience segmented, fragmentary healthcare if they receive any healthcare at all. The lack of widespread healthcare coverage results in an un-even distribution of healthcare resources that eventually converts into emergency department overcrowding.

Overcrowded and understaffed emergency departments are a kind of perfect storm for medical errors, particularly misdiagnosis.[20] In addition to healthcare system mismanagement, we may also consider the kind of dysfunctional social ecologies frequently found in medical organizations. Physicians and other healthcare providers that maintain a highly autonomous "captain of the ship" mentality are much less likely to rely on team efforts in healthcare delivery[21]. A lack of team concept has been shown to be associated with healthcare delivery failures including errors in diagnosis[21].

Lastly are the obstacles to effective diagnosis that result from the performance of individual clinicians. Effective medical reasoning, particularly in the form of diagnostic capacity, requires not only a great deal of training but effective functioning of a clinician's mental capacity. Cognitive preventable medical errors such as misdiagnosis arise from two general sources-cognitive deficits and cognitive biases[6]. Cognitive deficits are transient impairments of cognitive functioning that may generally be prevented or corrected.[6] Arising from some alteration in normal cognitive functioning, states causing cognitive deficits include fatigue, stress, poor mood and acute illness. More persistent but potentially correctable are affective dispositions such as burnout and reduced pleasure of practice. Included in cognitive deficits are gaps in knowledge and deficiencies in training.

Current Models of Clinical Reasoning and The Diagnostic Process: Theoretical Frameworks

Anyone who practices clinical reasoning has been trained in one (or more) models of clinical reasoning. Though not always explicit, these models can be analyzed to discern the conceptual foundations and background judgments that fund the ways of acting and thinking by clinicians trained by those models. Evidence regarding the relative strengths and weaknesses of various models can be adduced and used to construct a proposed model of clinical reasoning for physician assistants. A review of the evidence suggests that four overlapping theoretical frameworks provide the bases for a strong

model of clinical reasoning. These frameworks include cognitive load theory, dual-mode cognition, situated learning and knowledge organization or the structured knowledge approach.

Cognitive Load Theory

Cognitive load theory, first advanced in 1988, describes learning and thinking in terms of three components of cognitive architecture: memory systems, learning processes and types of cognitive load imposed on working memory.[24-25] Memory systems include the sensory system, working memory and long-term memory. Learners initially encounter the sights and sounds of phenomenon that they intend to learn. These data may be the words spoken by a lecturer (or patient) or the images of a slide deck (or a patient's physical exam). The sounds and images are held in the dual components of the sensory system – iconic and echoic memory. The two channels of the sensory data system receive and briefly hold enormous amounts of data. While only held in the sensory system for 0.25 seconds to two seconds, only some of this sensory data will reach conscious awareness. When a learner attends to the information in sensory memory, the data – images and sounds – are moved into working memory. Working memory organizes and reorganizes the information "so that it may be efficiently stored as packages in long-term memory. Theoretically, LTM has a limitless capacity but "a roadmap is required to find the information…working memory encodes information with this route map to enable retrieval when the information is needed in the future."[24] Unlike long-term memory and sensory memory, working memory is not only finite it is constrained to generally only seven elements (± two)[24]. Working memory can "actively process (i.e. organize, compare and contrast) no more than two to four elements at any given moment."[21] As information can only be held in working memory for a few seconds, learners may maintain its position by rehearsing the data like repeating the digits of a phone number. The limited nature of working memory poses challenges to anyone attempting to manage larger volumes of data.[24] To work within these restraints "all of the information elements must be combined and organized into a few meaningful units called 'chunks'." [24-25] This chunking of information subsumes multiple elements

into one element thereby freeing up space in working memory. More complex forms of chunking include the use of schema. In working memory, the mind mentally rearranges words and images into a coherent cognitive representation or schema. These new knowledge structures or schemas connect with relevant prior knowledge activated from long-term memory. [24-25] For example, one may have a schema for going to a restaurant. One knows according to the restaurant schema that one waits for the maître d' to be seated, handles the napkin in a particular manner, listens to the specials, inspects the wine list and so on.[14] These various elements – background understandings, procedures and cues - have been packed into the schema which importantly can be activated as a single cognitive element[14,24]. Schemas are not universal knowledge structures like syllogisms or logic tables. Rather they are domain specific and formulated in a way that is rooted in the context in which they are learned. This contextual learning allows for the organization of "multiple elements of information according to how those elements relate to each other and or will be used."[24] Additionally, schemas may be so well constructed and highly complex that they are said to act "as a central processor: with extensive practice, a schema can become fully automated and can act as a central processor, organizing information and knowledge without conscious effort…"[24] The development of cognitive structures like schemas is thought to be an evolutionary adaptation to the burdens of cognitive load.[6]

Cognitive load, according to cognitive load theory, may be described in three forms: intrinsic load, extrinsic load and germane load. Intrinsic cognitive load is the load or work associated with a given task.[24-25] At first, this designation may seem subjective as a task may be difficult for some but not for others. However, in addition to proficiency of the learner, intrinsic cognitive load also accounts for the "number of information elements and the extent to which the elements interact with each other referred to as element interactivity."[24] Consequently recalling and recognizing the clinical criteria to diagnose streptococcal pharyngitis will require lower intrinsic load than recalling and recognizing the clinical criteria to diagnose systemic lupus erythematosus.

Extraneous cognitive load refers to the load forced into the trainee's working memory but not necessary for learning the task at hand, i.e. for schemas construction or automation. A classroom example of extraneous cognitive load is when a teacher produces "visual overload when he shows full text slides but allows too little time for the learners to read them; if, in addition, he gives simultaneous verbal information that does not align with the visual slides, distracting (extraneous) cognitive load is introduced that will impair both channels of information."[24]

Germane cognitive load is the load imposed by the mental processes "necessary for learning such as schema formation and automation."[24] In novice learners, germane load will be relatively high as they lacked pre-existing knowledge structures to construct new schemata where novices will require noticeably greater work in learning new don't domain material, experts will leverage existing schemas to incorporate new domain material more easily.

Cognitive load theory provides important applications for clinical reasoning and curricular design. First, from a global perspective, the only way that most medical learners will be able to manage the cognitive load associated with the information volume is through some type of knowledge organization. From a curricular standpoint, this will include sequencing "context" in a way that scaffolds pre-existing knowledge structures. More recent models of medical education and curriculum development have begun to emphasize efficiency, prioritized content, and organized knowledge structures.

Cognitive load theory provides important considerations for curriculum development as well as the future of medical education. On the level of curriculum development, it is important for educators to titrate cognitive load to developmental stages of learning. Titration may take the form of content sequencing that scaffolds on to previous knowledge as well as a gradual progression from partial tasks to whole tasks and eventually progression to high fidelity simulation. On a more macro scale, medical

educators and curriculum designers must come to terms with the global cognitive load associated with the exponential rise in medical knowledge. One frequently cited estimate suggests that while in 1950 the doubling time of medical knowledge was 50 years, by 2020 medical knowledge doubles every 72 days.[35] Although these figures must be regarded as dubious as they were provided without specific evidence, the point remains the exponential growth of medical knowledge warrants a move away from an encyclopedist views of medical knowledge and toward efficient and streamlined modes of learning and cognition.

Dual System Cognition

Another theoretical framework that is central to current understandings of clinical reasoning is dual system thinking.[28] Conceived in the seminal work of Daniel Kahneman and Amos Tversky, dual system thinking posits that thinking occurs in two modes: Type I or fast thinking and Type II or slow thinking.[28] Type I thinking may be understood as driven by a series of acquired intuitions. Our ability to think fast is predicated upon acquiring a series of mental shortcuts or heuristics that allow us to make sense of our complex world.[28,30-32] Heuristics include cognitive structures like the schemas mentioned above and pattern recognition mechanisms that allow us to interpret the diverse data we encounter. For medical practitioners, medical training is the process of acquiring effective heuristics that will allow them to evaluate complex clinical scenarios efficiently and effectively. [28,30-32] An example of medical heuristics are illness scripts where clinicians first learn didactically about an illness's signs and symptoms. The student then moves on to guided clinical education where these illness scripts are confirmed and made more effective through modifications acquired by the data of additional experience and research. There are many types of heuristics that clinicians deploy daily and although this type of thinking is efficient there are ultimately trade-offs. [28,30-32] Dual system cognition is the ability to move between rapid heuristic pattern recognition Type I and more plotting analytical Type II thinking. A paradigm of Type I thinking is rapid visual diagnosis of a patient with shingles. The patient presents with a complaint of

burning, tingling thoracic pain on the unilateral torso followed by a vesicular rash that follows the boundaries of a thoracic dermatome. In the absence of unusual factors, the diagnosis will be certain based simply on the patient's report of symptoms and physical examination. Developing an elaborate differential diagnosis in a case like this would be a waste of time and if pursued with any seriousness would likely generate unnecessary testing and costs. There are some conditions which by virtue of their very nature, shingles being an excellent example, that are best identified using type I pattern recognition. That same type of thinking can be effectively extended into other more complex cases if a clinician recognizes data that does not fit their heuristic expectations. However, the relationship between heuristic thinking and the ability to recognize new data is precisely the problem. Although most cases can be effectively managed using heuristic pattern recognition other cases, such as atypical presentations, will require a more analytical approach. Type II thinking is a slow, plodding, and methodical process of describing and analyzing data that do not fit pre-existing patterns. When experienced clinicians encounter a novel presentation or atypical case, the well calibrated clinician will switch to Type II thinking to better assess the problem. Revisiting the headache case in the narrative review, when the clinician noticed that the patient's pain was not responding to appropriate therapy, that should've prompted a reconsideration of the migraine diagnosis. For the most part, migraines will respond to a particular treatment regimen for abortive treatment. When a clinician is evaluating a particular case and recognizes that some data do not fit their prevailing diagnostic hypothesis, the clinician must consider other possibilities. To consider other diagnostic possibilities the clinician may consider gaps or omissions in their own history and physical exam and reevaluate the patient to fill in those gaps. Perhaps a clinician forgot to inquire about a family medical history of aneurysm and having revisited the matter has discovered that the patient does have a family history of cerebral aneurysms. Perhaps the evaluating clinician, a new trainee, has never seen a case of nontraumatic subarachnoid hemorrhage and this diagnostic possibility has not emerged with sufficient clarity in the clinician's

thinking. The clinician may discuss the case with a trusted colleague or an appropriate specialist to bring some clarity to the patient's differential diagnosis. After reevaluation of the patient and consulting a trusted colleague, it occurs to the clinician that a lumbar puncture to further assess for subarachnoid hemorrhage would be appropriate. Having done so the patient will still be on track for appropriate diagnosis despite the initial misdirection. Were the clinician to have persisted in their initial diagnostic impression of migraine despite the new data of unremitting pain and family history of cerebral aneurysms, diagnostic failure would've been certain.

When a clinician evaluates the patient considering their training-acquired heuristic they have in mind a particular pattern that drives their understanding of a patient's case. This pattern could be an illness script acquired in training or a clinical vignette from previous experience with similar patients. Regardless of the source, these heuristic patterns will shape the clinician's intentionality in such a way that they will only recognize the significance of data that conform to their currently held pattern. Data that do not fit their heuristic pattern will be regarded less significant or dismissed altogether.

Dual System Cognition and Cognitive Biases

Cognitive bias is the tendency to make false judgments about an objective state of affairs despite having access to all of the phenomena regarding those objective states of affairs. [28,30-32] That is, cognitive bias is the tendency to ignore data that does not fit our heuristic patterns. Research over the last two decades indicates that there are many cognitive biases that can affect clinical thinking. [28,30-32]

The framing bias occurs whenever a patient presents in a context, setting or manner that frames the way the clinician thinks about the patient's case. [28,30-32] Consider how the clinician's thinking might be affected if the patient were presented by the nursing staff in the following manner; "the gal in room five with a headache is here all the time looking for pain medication." The clinician searches electronic medical record and finds that the patient has been evaluated in the same emergency department for

five pain related complaints in the last 18 months. The patient's presentation has now been framed in terms of possible drug seeking behavior and this background judgment will begin to influence how the clinician views the rest of the patient encounter. Another example can demonstrate how framing bias can be more cumulative. A 12-year-old male is brought to an urgent care by his mother with a chief complaint of "strep throat." The patient's mother is quite emphatic that last time her child contracted streptococcal pharyngitis he exhibited the symptoms of abdominal pain and vomiting. Note the two sources of framing in this instance. First there is the setting. Common assumptions and background judgments regarding appropriate care settings suggests that people with strep throat should go to an urgent care and people with worrisome abdominal pain should go to the emergency department. Secondly, there is the emphatic mother with a strong healthcare belief that her son has streptococcal pharyngitis. Although it is true that streptococcal toxin can precipitate nausea and vomiting, any patient with abdominal pain requires a properly developed differential diagnosis. The mother's stern eye contact, slightly raised voice and no-nonsense demeanor make an impression on the clinician. The patient's mother strongly prefers to forgo any testing and for the patient be to be treated empirically with antibiotics. Despite the absence of any typical findings of pharyngitis the clinician yields and provides a prescription for penicillin. The next day the 12-year-old boy is taken to the local hospital and is found to have a perforated appendix.

Another type of cognitive bias is the anchoring effect. [28,30-32] In anchoring, clinicians will anchor upon an initial datum that is discovered early in the patient's presentation and allow that datum to characterize the remaining evaluation. A common scenario for anchoring bias is when a patient with COPD (chronic obstructive pulmonary disease) presents with a chief complaint of shortness of breath. Patients with COPD may have acute and chronic dyspnea and so any complaint of shortness of breath is attributed to their underlying lung disease. Unfortunately, patients with COPD are at increased risk for other cardiopulmonary conditions and so patients with COPD will frequently be misdiagnosed with

"acute exacerbation of COPD" when they are experiencing pulmonary embolism, heart failure, myocardial infarction or critical aortic stenosis.

Another bias that can occur when well respected senior clinicians are involved in patient care is the authority bias. [28,30-32] Returning to the headache case study, the clinician had reassessed the patient to be at increased risk for cerebral aneurysm and had identified an abundance of red blood cells in the patient's cerebrospinal fluid. The clinician contacts the neurosurgeon on call and provides the neurosurgeon with the details of his evaluation. The neurosurgeon is "unimpressed." Rather than being concerned about a sub tomographic cerebral aneurysm the neurosurgeon attributes RBCs in the cerebral spinal fluid to the trauma of lumbar puncture and dismisses the patient's ongoing report of severe headache as "histrionics." The emergency department clinician is advised to discharge the patient and have them follow up with their primary care provider. The ED clinician objects that the patient has persistent pain and that the lumbar puncture was easily acquired and without complication. The neurosurgeon persists in his assessment of the case and declines to admit the patient. The ED clinician is vexed by this apparent dilemma. On the one hand he has a patient with signs of a potential subarachnoid hemorrhage and on the other hand he has a well-regarded neurosurgeon summarily refuting the diagnosis of subarachnoid hemorrhage. Here the clinician has two options: follow the advice of the specialist or seek a second opinion for consultation. The first option seems reasonable given the status and authority of the consultant; he is, after all, a neurosurgeon. However, when the ED clinician considers that the neurosurgeon too is a mere mortal and subject to the same potential cognitive deficits and cognitive biases as every other well trained medical professional, a second opinion is warranted. The three common cognitive biases described above are just a few representatives of a large number of cognitive biases that have been described. A recent survey identified no less than 47 described cognitive biases that may come to bear on clinical reasoning.[6]

Cognitive biases have been estimated to be associated in 50 – 83% of cases of misdiagnosis.[30] What can be done to mitigate the impact of cognitive biases in clinical reasoning? Recall that cognitive biases arise as a result of heuristics deployed to manage cognitive load and to navigate the terrain of various domains. If heuristics or mental shortcuts give rise to cognitive biases and their associated diagnostic failures, wouldn't clinicians be better off adopting models of clinical reasoning that are based on the ideals formal logic and probability?[29] That is, shouldn't training "alert decision-makers to generic human biases (e.g. availability and representativeness), warn them about potential for error associated with these biases and increase knowledge and appreciation of less biased strategies?"[29] Training could, on this model, "focus on circumventing heuristics in order to reduce opportunities for errors that occur in the form of deviations from logical rationality."[29] From the perspective of cognitive load theory, a shift into a predominantly type II system of thinking would be paralyzing. In the absence of heuristic knowledge structures, intrinsic load of various tasks would be unachievable.[24-25] Not only is a purely analytical form of reasoning untenable from a cognitive load standpoint, but pure rationality does also not really occur in human thinking. Investigations of expertise in multiple domains indicate that rather than "universal principles" of rationality, experts "actually use heuristics as smart adaptations to the complexities of a specific domain…Training should "not aimed to circumvent the use of heuristics or to nudge decision-makers towards logical decisions… Instead, training should increase the perspicacity of the student with regard to heuristic decision-making; that is, tune the (recognition) processes that underlie the adaptive selection of heuristics and management of errors in the domain of interest."[29]

Dual system thinking and the problem of cognitive bias has assumed a central place in our understanding of clinical reasoning. Although the extent that cognitive bias plays a factor in the problem of misdiagnosis is not known with certainty, the effect is thought to be quite significant.[20,31-32] So much so, that the scholarly work group producing "Competencies for improving diagnosis: An interprofessional framework for education and training in health care" included cognitive debiasing as

an essential competency in their framework. The question for PA training models is when is it appropriate to introduce cognitive debiasing into the curriculum? When misdiagnosis occurs because of cognitive bias, it usually does so in more advanced stage learners and experienced practitioners. That is generally, cognitive bias occurs in learners and clinicians who have acquired sufficient knowledge and skills to engage in varying levels of clinical reasoning. What renders more advanced learners (including intermediates) and clinicians "eligible" for cognitive bias, is their preliminary development of clinical reasoning skills. That is, clinicians begin to tacitly endorse the efficacy of their own knowledge and skills and fail to consider how blind spots affect their practice. Novice stage learners, for the most part, are not adequately equipped with sufficient knowledge structures and practice patterns to develop a clinical perspective. Novices are not yet prone to the blind spots of cognitive biases because they have yet to acquire eyes that see, metaphorically speaking. Consequently, introducing cognitive debiasing strategies into novice stage learning is likely to promote confusion and misunderstanding. However, cognitive debiasing strategies will be crucial to more advanced stages of training.

Situated cognition

Another theoretical perspective that has garnered enormous attention by medical educators is situativity or context. Situativity and context refers to the broad range of contextual factors that could impact a clinician's decision-making at the point of care[39-45]. Context is mediated through the domain of the clinical encounter. The clinical encounter may be thought of as consisting of various elements: the clinician, the patient, the problem being presented and the setting in which the patient presents[39-45]. These elements may be thought of as constituting the domain that is the clinical encounter. The nature, complexity and, importantly, the interaction of these elements, may be further understood as rendering the domain of the clinical encounter as more or less well defined. Clinical encounters in which the elements and their interaction are stable and simple are well-defined. An example of the clinical encounter that may be thought of as a well-defined domain could be a 32-year-old previously healthy

female presenting to her primary care provider for an appointment regarding a painful blister rash that follows a dermatomal distribution. Clinical encounters that are constituted by increasingly complex and unstable elements will produce ill-defined domains. An example of such an encounter could be a 76-year-old diabetic female presenting to a busy emergency department where she is evaluated by a junior clinician for a complaint of “weakness.”

The significance of context in clinical reasoning stems from a series of same-subject studies where a subject's performance in one context changes in another.[39] That is, in one context, a subject will be given the data of a patient with a particular problem and arrive at a correct diagnosis. Those same data and clinical problems will then be presented to the same clinician in a different context and the clinician arrives at a wrong diagnosis. Eva and other researchers have proposed that clinical reasoning and diagnostic capacity are not stable dispositions (traits developed in the practitioner) so much as context-bound states. These context-specific variations in reasoning have given rise to a perspective that regards clinical reasoning as “situated cognition.” That is, clinical reasoning is always bound or shaped, in an important way, by the situation or context in which that reasoning occurs.[39-45]The various elements and dynamics that may give rise to unique, contingent circumstances are sometimes referred to by the substantive term - “situativity.”[43] The various proposed elements that constitute “context” or “situation” are wide-ranging and there is no agreement on what can be regarded as the stuff of context. Medical researchers studied the impact of various inputs and factors attempting to determine which of the factors impacted patient outcomes; the factors studied included professionalism, clinical skills, communication skills and overall competence.[37-39] However these factors were studied in isolation. The emergent theme was that "selected inputs tend to explain little variation in outcome."[37] Unable to identify isolated factors that led to variations in diagnostic performance and cognizant of the potential impact of context on performance researchers turned their attention to context and situativity as a possible source of variation in clinical reasoning. The problem with the previous approach was that

factors – disease, patient complexity, clinical setting – were considered if at all, in isolation and not as elements interacting. Context theorists suggest that it is not only the number of elements in a patient encounter that increase the complexity in an additive way but the way in which the elements interact with each other thereby increasing the complexity of the encounter in a multiplicative way. In recent treatments, context has become so complex in the thinking of medical educators that the very idea of context has emerged as a kind of metaphysical force. Context, in one recent article described context "as a fabric…an interplay of dynamically interacting patterns, and emergent product, activity or an element surrounding the individual."[42] In another article, "Mapping the Dark Matter of Context: A Scoping Conceptual Review," the authors summarize the possible elements of context and arrive at 50 or so descriptive categories.[41] Context is apparently so conceptually fecund that an attempt to even broadly outline context still warrants 50 categories of descriptors. Moreover, the authors assert that context is both ubiquitous and somehow strangely hidden from us. That is context becomes "invisible" to those within a context.[41] How do clinicians interact with context if it cannot be seen, defined or in any way managed? Situated cognition is characterized, says one author, as an activity of creating meaning "on the fly, rather than reading it back from something (representation or schematic) stored in the head."[42] They go on to suggest that "knowledge and cognition emerge from the particularities of the given situation."[42] The notion of creating meaning "on the fly" or instantaneously without reference to any sort of existing knowledge is problematic. All cognition requires a store of knowledge to enact the cognitive process. The suggestion that cognition arises de novo "from the particularities of the given situation" is completely unintelligible without a rational agent in possession of some capacity for interpreting and interacting. This extreme form of situated cognition seems to suggest that the "situation" impresses itself upon the subject; every subject is a kind of naïve tabula rasa and every novel situation renders a new mind. A more modest and parsimonious account of contextual factors describes three categories of contextual elements: clinician factors (knowledge, skill, professionalism, fatigue,

expertise etc.), patient factors (acuity of illness, complexity of problems, anxiety, communication skills, common versus rare presentation) and encounter or practice factors (appointment length, ambulatory or inpatient setting, support systems and staffing).[37]

Knowledge organization and illness script theory

Another pivotal theoretical framework that informs clinical reasoning is the field of knowledge organization and illness script theory. In attempting to describe "how medical expertise matures," Schmidt and Rikers proposed a "theory that considers the development of expertise as progressing through a number of transitory stages, each characterized by knowledge structures underlying diagnostic performance that are qualitatively different from the other stages."[46] The dynamic driving the learner from one stage to the next is knowledge organization: "development of expertise in medicine can only be properly understood by assuming certain kinds of knowledge shifts or knowledge restructuring."[46]

The first phase in the acquisition of clinical expertise is the novice stage of learning. In this stage "students rapidly develop structures that can be described as rich, elaborate causal networks that explain the causes and consequences of disease in terms of general underlying biological or pathophysiological processes."[46] These learners will grow into a more advanced stage of learning through a process called encapsulation. Through "extensive and repeated application of knowledge acquired and particularly through exposure to patient problems" the learner's knowledge undergoes a restructuring. That is their "networks of detailed causal pathophysiologic knowledge become encapsulated into diagnostic labels or high-level simplified causal models that explain signs and symptoms."

As learners practice extensively with patients a second shift occurs in which the knowledge encapsulated is reorganized into narrative structures called illness scripts. In this model, illness scripts

are a kind of schema or cognitive entity "containing relatively little knowledge about pathophysiological causes of symptoms and complaints (because of encapsulation), but a wealth of clinically relevant information about the enabling conditions of the disease, as a product of a growing experience with how disease manifests in daily life."[46] In illness script theory, the script schematizes three main components: enabling conditions, fault and consequences. Enabling conditions include the contextual and risk factors that set the stage for a particular condition or disease. The enabling conditions are typically revealed in the stem of a clinical vignette: "a 76-year-old male with long-standing hypertension complains of sudden onset of severe chest pain. The pain is retrosternal and radiates through to the back and is described by the patient as tearing." Upon hearing "76-year-old male with long-standing hypertension" an experience clinician has in store of number of conditions that could occur as the sequela of long-standing hypertension: hypertensive heart disease, nephropathy, retinal hemorrhage, coronary artery disease and aortic aneurysm (non-exhaustive list). Upon hearing the complaint "chest pain" that is typical for aortic dissection, a decisive association is made and aortic aneurysm appears in the differential diagnosis. The patient's signs and symptoms as well as diagnostic findings are listed under the category of consequences. The fault in illness script theory is the pathophysiologic mechanism. In the case of aortic aneurysm, the fault is a separation of the tunic intima from the tunica externa. The internal logic of the illness script schema reads in this way: because of the presence of enabling conditions (age greater than 55 and long-standing hypertension), a pathophysiologic fault occurred (separation of the aortic wall layers) that resulted in the clinical presentation (severe chest pain, tachycardia, cool pale and diaphoretic) and subsequent sequelae, depending upon appropriate treatment in the patient's response.

Researchers have described differences in how intermediates and experts recognize illness scripts. Experts tend to focus on enabling conditions and clinical manifestations to arrive at accurate diagnoses more rapidly than their intermediate counterparts.[46-48] Intermediates in contrast tend to

focus on pathophysiologic data in the category of fault. The differences in how experts and intermediates utilize illness scripts is explained by the restructuring process: "novices…will rely more on their knowledge of the fault, the underlying pathophysiologic mechanism, in understanding disease in diagnosing patients." [46-48] A frequently cited analogy to illuminate the differences between novices and experts is mastery in chess.[46] Expert chess players have been shown to have analytic problem solving that is comparable to novices. What distinguishes expert chess players is that, by studying the games of other experts, they have developed a mental repertoire of schemas that reduce cognitive load and improve efficiency. In chess, these schemas include learning a patterned series of moves that constitute, for example, a specific kind of opening game that is denoted by a single label like the “Ruiz-Lopez” opening[47]. Without the availability of schemas (illness scripts), novices resort to slow, analytical, system two thinking. As novices gain clinical experience "this reasoning through" a case quickly become superfluous as the steps in reasoning chains become compiled."[46] Like encapsulation, this compilation of reasoning chains allows the clinician to gain efficiency by focusing on enabling conditions and chief complaint (consequences). Importantly, the compilation process does not obviate the need or utility of pathophysiologic knowledge. Rather it, it is compiled or encapsulated into the new knowledge structures: “pathophysiological knowledge relating to causes and consequences of disease does not decay with experience, but rather forms a coherent structure of knowledge that can easily be accessed when needed.”[46] Moreover, the restructuring process is not static or irreversible. If an expert practitioner encounters an unfamiliar problem, illness scripts can be "decapsulated" that is "when necessary, illness scripts in the embedded biomedical knowledge can be used separately." [25, 46]

Schemas, in the form of illness scripts, provide a widely accepted and validated understanding of knowledge organization and have demonstrated the importance in the development of clinical expertise. With knowledge organization so central to expertise development, medical educators have

begun to consider whether preclinical knowledge may be organized or structured in such a way that it optimizes learning.

One proposal is the use of frameworks to complement the formation of schema.[49-51] In attempting to sort out the language of medical education, Dreiser and colleagues make an important distinction between schemas and frameworks.[49] Schemas are cognitive structures *in the minds* of student clinicians of which illness scripts are an important example. Frameworks on the other hand are *external* "representations of conscious elements of schema that are developed to clarify and or simplify relationships between concepts in a specific knowledge domain."[49] Frameworks "provide an organized and simplified structure of complicated, often very detailed, medical knowledge codified outside an individual's mind."[49] Usually represented in a written or pictorial form, frameworks are geared towards a specific educational purpose. Generally, frameworks have two basic elements: a thematic emphasis and a mode of representation. A common mode of representation is the algorithm which consists of a series of branching decision points. The decision points can be a query about the presence of the diagnostic feature. For example, in a solitary skin lesion algorithm the first decision point may be size, then color, then border and so on until the differential diagnosis has been sufficiently narrowed. The other element of frameworks is the thematic emphasis. For diagnostic frameworks, conditions may be framed according to anatomy and pathophysiology to sub categorize a differential diagnosis or focus on common and deadly (can't miss) conditions.[49] An example of such a diagnostic framework is the mnemonic "GOLDMARK" (glycolic acid, five Oxo-proline, L lactic acid, D lactic acid, methanol, aspirin, renal failure, ketoacidosis) used to frame diagnostic possibilities of an ion gap metabolic acidosis.[49] Far from being a mere gimmick or simple mnemonic, frameworks provide educational value "by organizing a differential diagnosis into subcategories or limiting the number of diseases to be memorized."[49] By "pre-organizing" the data within the frame "frameworks can reduce the load on working memory allowing easier recall of the information while also providing a scaffold to organize new information (that will

ideally be integrated into the learner's existing schema)."[49] Note that the putative benefits of using frameworks is not merely recall but a qualitative enhancement of knowledge organization: “explicitly teaching frameworks (and thus making clear the features that discriminate between anatomical/pathophysiological “buckets” and or diseases) will aid in the development of better schema in future learning and practice.”[49] Precisely how the use of frameworks will qualitatively improve knowledge organization is not known but the authors suggest that the use of frameworks “likely leads to changes in neural networks and thus alters an individual’s schema development.” Moreover, there is evidence to suggest that teaching a structured knowledge approach to diagnostic problems can improve diagnostic accuracy for early learners. [46-48]

Problems with the current training models: Context misalignment

A significant problem with current models of clinical reasoning stem from their tendency to defer or bracket clinical context from the preclinical phase of education[78]. On the traditional model, pre-clinical education functions only to provide adequate bulk of theoretical knowledge for later phases of learning. A substantial amount of time will be spent in the accretion phase of learning and without this preparatory or preclinical phase, later stages of learning are thought to be impossible[55]. Consequently, many traditional curricula defer clinical reasoning education to the clinical phase of training. An important example of the strictly separated model of training is the current widespread use of the physical diagnosis course. The physical diagnosis course, common in PA programs and medical schools, is a preclinical or didactic course that tends to focus on appropriate performance of history taking and physical examination maneuvers. One midwestern PA program’s syllabus stated the aim of the physical diagnosis course in this way: “The purpose of the Physical Diagnosis Course is to learn the skills and techniques that enable you to obtain, document, and perform a complete and timely history and physical examination” (private document). The emphasis is on the exam “skills and techniques” rather than their application as part of the clinical reasoning process. On this model, students “are first taught

the physical exam as a comprehensive battery of maneuvers."[78] These physical exam maneuvers that students are required to learn can range to above 100.[78] For assessments, "students are typically assessed on their ability to perform a comprehensive exam that often includes both commonly and infrequently performed maneuvers."[78] This process of decontextualized accretion of techniques is reinforced by evaluation strategies such as having students regurgitate a number of review of system items and correctly perform exam maneuvers without chief complaint or clinical context.[77-78] Later, in the clinical setting, students find themselves "confused and anxious about which maneuvers should be routinely performed on each patient and which maneuvers should be performed only when clinically indicated."[78]

Problems with the current training models: Cognitive load mismanagement

The problem with traditional models that strictly separate preclinical from clinical learning is that they defer the cognitive load associated with clinical reasoning to the clinical phase of education. Consider a physician assistant student beginning the clinical phase of their education and being required to not only recall salient basic science, clinical medicine, and physical diagnosis information but to synthesize that information into a coherent and rational process of clinical reasoning. This must all be done while at the same time encountering "live" patients and navigating the complexities of various practice settings. The misguided emphasis on components like physical exam maneuvers rather than on clinical reasoning process tends to produce students that struggle with clinical reasoning.

Problems with the current training models: Knowledge fragmentation

Another problem with the traditional models is a lack of knowledge integration. This issue is highlighted by the problems of transfer: "a major problem that particularly vocational and professional education struggles with is transfer. Too often employers or supervisors and students themselves complain that they, as graduates are not sufficiently prepared to act in the workplace, despite

educational programs that have covered all relevant topics. Learners are said to be not able to transfer what has been ‘‘learned’’ in school to what they must ‘‘do’’ at work.[24] One reason for this is that the educational programs decompose the real-life tasks into fragments that are taught at different moments in the curriculum.[24] Medical training programs have attempted to deal with the problem of context deferral and by “vertical and horizontal integration” strategies.[36]

A comparison of medical training with aviation training may help illuminate the challenge of context in education and training[49]. Pilots undergo a significant amount of classroom and laboratory training before being given an opportunity to take control in the cockpit. Leading up to this context rich experience of being in the cockpit, the novice pilot must acquire a significant amount of background knowledge that is necessary to understand the various phenomena experienced within the cockpit. Understanding how weather trends, physics and other elements come to bear on the performance of the aircraft are just some of the background knowledge that is essential to understanding the readings of the altimeter or other instrumentation. For a novice pilot, an instructor may choose to give the student control of the aircraft but only if the conditions were suitable. Weather conditions, aircraft conditions, student conditions would all be evaluated by the instructor to ensure that the experience of piloting the aircraft and all associated context provided a stable and well-defined domain. One would expect that the novice pilot, in time, would be able to fly with increasingly less input from the instructor until they were able to pilot the plane in a solo flight. Importantly, a novice pilot’s solo flight would only be permitted if the context (weather conditions, aircraft conditions and student conditions) were suitable for the pilot’s stage of learning. Inclement weather, increased air traffic or student fatigue are all potential factors that would increase the complexity of the learning domain. In time, however, pilots are expected to fly in a range of weather conditions and manage a range of other contextual factors. As pilots progress toward expert stages of learning, their training allows them to adjust to contextual factors and account for nonlinear modifications of the flight plan. Where novice pilots can be expected

to execute a linear flight plan under ideal conditions, expert pilots can adjust for increasingly complex contextual factors and execute nonlinear modifications to a flight plan.

For both medical and aviation trainees, ultimate success in their vocational goals requires formative training in their respective contexts: the clinic and the cockpit. The question in both fields is how much and what type of pre-context learning is required to prepare them for these contexts? By analogy, if pilot training were based on the traditional model, a fairly sharp distinction would be made between preflight and flight learning. The novice pilot trained in this model would be very familiar with the background knowledge associated with aviation and would have some superficial procedural knowledge about piloting an airplane. However, because of the strict division between preflight and cockpit phases of learning, the cognitive load associated with this transition may be unmanageable. The sensory data of instrumentation, the physical sensation of small aircraft flight and the anxiety associated with an intense performance situation would raise the extrinsic cognitive load to a potentially crippling level. In aviation training, the problems of cognitive load and knowledge transfer are mitigated through increasingly complex forms of simulation training. Preflight training is scaffolded onto flight training through a series of simulation-based exposures that allow for cognitive load titration and preliminary forms of knowledge transfer.

Summary of literature

A review of the literature on prevailing theoretical frameworks in clinical reasoning provides several useful insights. An analysis of cognitive load theory demonstrates how knowledge organization occurs as a result of the demands of memory architecture. Schemas, like illness scripts, are constructed to improve cognition despite the limitations of working memory. Cognitive load theory also provides insight into pedagogical technique and curricular elements. Learning experiences should be managed in a way that titrate cognitive load for optimal learning; the level of intrinsic load should be formulated in a

way that parallels the developmental stage of learning. Lastly, global cognitive load - the ever-growing knowledge burden associated with contemporary medicine - warrants heuristic streamlining of complex content and modern approaches to information management.

An analysis of dual system cognition theory revealed the impact of cognitive biases on clinical reasoning. A review of the literature indicates that, at this time, there is current evidence to suggest that clinical reasoning should both deploy type I and type II system thinking. However, our baseline mode of cognition generally will take the form of heuristically driven type I thinking. Training and metacognition and other debiasing strategies should be reserved for intermediate and advanced stage learners. Deploying cognitive bias training in novice stage learners could be confusing as their initial charge is to acquire the logic inherent in well-defined domains.

The field of situativity and context has provided powerful insight into the variables of the clinical encounter and their interaction. The extent to which context affects clinical decision-making and approaches to improve clinical reasoning as situated cognition will be the subject of many future inquiries. For novice stage learners, the cognitive load associated with context should be incrementally titrated to learning experiences and the learner stage of development. Encouraging novice stage learners to attend to the context, particularly in its robust formulations, would distract students away from their primary goal of acquiring the logic of clinical medicine.

Knowledge organization and its emphasis on efficient and effective knowledge structures offers promise for educating novice stage learners. Frameworks, like those described above, have been used with some success in promoting diagnostic capacity in early-stage learners. Any contemporary model of clinical reasoning will make full use of frameworks and leverage their ability to promote rich and effective schemas.

Curriculum for diagnostic proficiency: Content focus - Stroke Neurology

Stroke neurology represents a unique opportunity for curriculum development. As noted above, strokes are overrepresented in the epidemiology of misdiagnosis related harms. Additionally, neurology is widely regarded by PA and medical students to be among the more difficult body systems and topics in clinical medicine.[66] Data consistent with this perception is that neurology is the lowest performing organ system on the PANCE for at least the last several years (Appendix 2). Given the urgency of the problem and the pedagogical difficulty associated with stroke neurology, neurology provides a rigorous test case for a curriculum in diagnostic proficiency.

Why emphasize stroke diagnosis rather than prevention?

It has been established that strokes are a common cause of mortality in the US[59]. The total healthcare costs associated with strokes including rehabilitation and long-term care are estimated to be approximately 50 billion dollars annually. These data combined with the knowledge that strokes are potentially preventable raises the question; shouldn't most of our time, educational efforts and healthcare dollars be spent on stroke prevention? This question is analogous to the historical trend in pulmonary causes of morbidity and mortality. In the early 1900s, tuberculosis was a leading cause of pulmonary morbidity and by the 1970's tuberculosis was replaced by chronic obstructive pulmonary disease (COPD) as a leading cause of morbidity[61]. In the case of tuberculosis, the advent of anti-tubercular agents provided an effective treatment that could be administered across a narrow window of time and required no long-term lifestyle modifications. COPD continues to be a significant source of morbidity and mortality because of ongoing and widespread cigarette smoking. Similarly, strokes are associated with the nexus of risk factors that are embedded in chronic lifestyle choices. These lifestyle choices are in turn embedded in the nexus of social determinants that are complex, structurally intransigent and will require years of macro social changes to see any improvement. Consequently,

strokes will continue to be overrepresented in causes of morbidity and mortality for the foreseeable future. Public health and educational efforts, in the near term, must not only address preventative measures but also improve clinicians' ability to effectively identify stroke syndromes and improve outcomes.

Studies estimate that somewhere between 9 and 17% of all stroke patients are misdiagnosed in the emergency department setting.[62-65] A review of the literature on stroke misdiagnosis indicates that there are several factors associated with diagnostic failure[2-4, 62-65]. One study reviewed multiple databases regarding stroke misdiagnosis and identified risk factors clustered under the headings of patient factors, system factors and disease factors[62]. The first factor that increased the risk of diagnosis was age less than 40. Clinicians tend to appropriately identify stroke as more prevalent in the elderly with multiple risk factors. However, younger adults may experience stroke as a result of vertebral artery dissections, patent foramen ovale and cardioembolic strokes[62]. Female stroke patients are more likely to present in an atypical fashion. An increased risk of stroke misdiagnosis among racial and ethnic minorities is consistent with unfortunate trends in recent studies with the correlation between misdiagnosis and race or ethnicity. Under system factors, the authors noted that healthcare centers without ready access to a neurologist were more prone to misdiagnosis[62]. It is not clear whether telehealth initiatives will improve diagnosis rates at centers without neurologists. Next, the authors analyzed disease or presentation factors or how stroke patients appear clinically when they are misdiagnosed. With stroke being the fourth most common disease that is misdiagnosed, a great deal of attention has been paid to the considerable variations in patient presentations[62]. This variation is so significant that researchers have produced a rubric for stroke misdiagnosis[62]. "Stroke mimics" are conditions that appear to be a stroke but are actually other conditions. The term "stroke chameleons" is used to describe when a patient actually has a stroke but it is diagnosed as another condition.

Stroke mimics are conditions that are frequently misdiagnosed as strokes but are other

conditions, such as hemiplegic migraine, Guillain-Barre or factitious disorder. Stroke mimics or false positive diagnoses of stroke, incur the risk of inappropriate treatment[62]. A notorious but avoidable stroke mimic is a patient that presents with Bell's palsy that is diagnosed with anterior circulation stroke[62]. Stroke chameleons, on the other hand, are actual strokes that are confused with and misdiagnosed as other conditions. Stroke chameleons are frequently embedded in atypical presentations of strokes such as dizziness, vertigo, headache, altered mental status and seizure.[62] This finding was corroborated where the presence of dizziness or vertigo "increases the odds of misdiagnosis 14-fold over motor symptoms (those with dizziness and vertigo are missed initially 40% of the time)."[5]

Analysis of practitioner factors in the misdiagnosis of stroke suggested that "cognitive factors may play a leading role in misdiagnosis in the emergency department setting with failure and judgment, knowledge or competence and vigilance (i.e. failing to keep watch for possible dangers) or flawed memory identified as dominant contributing factors."[62]

Curriculum for diagnostic proficiency: Heuristic streamlining and scaffolded sequencing

Evaluating neurological problems such as strokes requires clinicians to evaluate nuanced and subtle clinical phenomenology that is only recognized through the matrix of neuroanatomy. However, the complexity of neuroanatomy can be daunting and medical trainees frequently struggle with this area of medical science. Students in advanced practice training programs are required to learn anatomy but there are no specified curricular requirements for neuroanatomy. Consequently, neuroanatomy education in advanced practice provider (APP) programs may not receive depth of content and sustained treatment that are required to form a foundation in the subject.

Although currently there is no data on the amount of neuroanatomy training in physician assistant programs there are, potentially, several obstacles to a robust neuroanatomy curriculum. First, is the lack of faculty with sufficient training to teach neuroanatomy. One factor inhibiting neuroanatomy

implementation is the sheer complexity of the subject matter. The ability to accurately describe the nervous system and identify the specific location of anatomical lesions requires a great deal of intensive study, review, and clinical confirmation. Lastly, accelerated training programs such as physician assistant training require enormous efficiency as there is less time and curricular space to teach complex subjects. However, a review of medical school neuroanatomy education may reveal the kinds of challenges and successes that PA programs face if neuroanatomy is fully implemented into their programs.

While neuroanatomy is a common component of medical school training, educators report problems with neuroanatomy education. These problems were described in 1994 with the term "Neurophobia" or "the fear of neural sciences and clinical neurology that originates from the students' inability to apply their basic science knowledge to clinical practice leading to paralysis of thinking or action."[66] Modifiable "risk factors" for neurophobia: poor teaching, complex terminology, separation of basic science teaching and clinical application, and a lack of simulations and patient encounters.[68]

A more effective neuroanatomy curriculum will avoid low efficiency pedagogical techniques such as passive learning formats and instead deploy methods that promote active learning and student engagement. Medical education in general has been stifled by a lack of coordination between basic science faculty and medical/clinical faculty. Collaboration between the two would improve understanding on how best pedagogical practices could complement capacity in clinical reasoning.

A review of the literature on stroke misdiagnosis has yielded several principles that have shaped the curriculum. First are insights from cognitive load theory. The state of neuroanatomy is so replete that it is virtually impossible for individual clinicians to comprehend and apply all nuances of the domain. Clinicians may, however, be expected to master a streamlined and heuristically crafted version of the content. Some elements of neuroanatomy are particularly complex and historically have been problematic for students to understand and master[68]. Two elements that would benefit from

streamlined and heuristically crafted treatment are brainstem/cranial nerves and vascular territories. In two neuroanatomy introductory lectures (Appendices 6 and 7), brain vascular territories are taught using a pictorial analogy ("Willis"). This technique leverages the spatial nature of long-term memory and the ability to use well recognized forms to understand new forms. Pictorial analogy and similar techniques are increasingly being deployed in heuristic modes of education.[67] Lecture three (Appendix 8) provides a scaffolded approach to the brainstem and cranial nerves. Following Berkowitz brainstem and cranial nerves were discussed with reference to a basic top to bottom framework that addresses how cranial nerves arise on the ventral brainstem (with exceptions) but have their nuclei beneath the surface.[69] Onto this basic framework, additional instruction on the brainstem will cover the autonomic functions and organization of somatosensory and motor pathways within the brainstem. This approach differs from traditional treatments of brainstem neuroanatomy that provide linear descriptions of the brainstem without an organizational schema.

Another principle recommended in the literature is early and robust clinical correlation.[68] For advanced practice providers, all anatomy and physiology must be applied within a clinical context. Trainees in longer programs have sufficient time to develop more complete background knowledge but physician assistants are trained on an accelerated curriculum which must emphasize the most salient clinical correlations. Initially, clinical correlations will be general and cover very little material regarding topics like treatment. It is important to remember that neuroanatomy must only provide a sufficient basis for further treatment that will come later in the curriculum in the form of clinical medicine and physical diagnosis. Subsequent treatment in other courses will provide spaced repetition and multi-perspective treatment that is effective in improving long-term retention and mastery. This curriculum provided robust clinical correlation through case studies (Appendix 12) as well as spaced repetition in the clinical assessment component of the curriculum (Appendix 15).

In terms of scope and sequence, it is best for neurology to be positioned toward the middle or

beginning of the last third of the didactic curriculum. Students need background knowledge in anatomy, physiology (with some neurology in both courses). It is also helpful for students have some background of general pathophysiologic mechanisms. In terms of organ system sequencing, it is helpful for students to have received ophthalmology and ENT prior to neurology as they will have an opportunity to review the cranial nerves thereby decompressing the cognitive load in neurology. Also, compared to other organ systems that may rely almost exclusively on laboratory evaluations (hematology) or simple pattern recognition (E-ENT), neurology requires eliciting and interpreting clinical phenomenology. Prior exposure to clinical reasoning would help to scaffold the learner's ability to evaluate neurological conditions.

Curriculum for diagnostic proficiency: Novice-stage learning and cased-based clinical reasoning

The learners for the experimental curriculum were physician assistant students at a Midwestern PA program. The learners had completed anatomy, physiology and a medical ethics course prior to starting the didactic curriculum where the experimental curriculum was deployed. Like their physician counterparts, physician assistants are trained on the medical model of clinical practice. In that regard there is a great deal of overlap between the two professions in their approach to clinical reasoning. However, there are two unique features of the PA profession. First, due to the limited formal training timeframe, physician assistants are trained in an accelerated curriculum. With the average program length being 27 months, physician assistants must be trained in a curriculum that is fast-paced and streamlined. The other unique feature of the PA profession is that PAs are prepared to practice medicine as part of the team. PAs work in a variety of practice settings and configurations. Some PAs work side-by-side with their physician counterparts, particularly in surgical specialty settings. Other PAs work completely autonomously such as those who provide emergency coverage in remote settings. The way in which a PA fits into a particular practice, is in part shaped by the practice laws in their respective states but generally, these laws provide minimal boundaries for practice configuration. PAs can work

with varying degrees of autonomy, with some working more proximal to their collaborating physicians than others. The variability of autonomy in practice settings will require a more flexible model of expertise development than those suggested for physicians.

The model advanced here is developmental in nature and can be described in four overlapping stages: novice, supervised intermediates, entrustable intermediate and expert in collaboration. Novice stage learners include pre-PA learners who seek exposure in various medical settings as well as physician assistant students in the "didiactic" phase of learning. When PA students have been adequately prepared for clinical training, their learning setting transitions to the clinical phase where they learn as supervised intermediates. Here, training requires the PA student to begin tuning their knowledge structures to the replete context of various clinical settings but is done so under the supervision of a competent and qualified clinician. After completing the clinical phase of their education, the PA student will graduate and practice in the entrustable intermediate stage. Training programs attest, by the graduation of the learner, that the learner has acquired mastery of entrustable professional activities. Employers and collaborating physicians also play an important role in the entrustment process. They too have evaluated the PA graduate and deem them entrustable to perform the professional activities circumscribed by state law, the agreed upon practice configuration and any relevant rules or guidelines imposed by various practice environments (hospital or clinic bylaws etc.). After years of calibrated practice, the PA may grow into the expert stage of practice. Being an expert in clinical medicine does not imply perfection or practice beyond error. Rather expertise implies a high level of professional competence.

The experimental curriculum proposed here focuses on the novice stage of physician assistant learning. In addition to being accelerated in nature, the curriculum must be highly integrated with clinical knowledge requiring the early introduction of elements like case-based reasoning. In reviewing the evidence from strong models of clinical reasoning, a recurrent theme emerged across multiple

theoretical frameworks – the need for early introduction of clinical problems: "Allow students to work with patient problems early in the curriculum and allow them to see many and varied patients. This would certainly encourage processes of encapsulation and illness script formation."[46] From the standpoint of diagnostic proficiency, all student development in clinical reasoning has its goal in expertise. However, before a student can acquire expertise, they must undergo the novice and intermediate stages of learning.

Recall the formulation of a patient encounter as a domain. The domain has several elements: the patient, the problem, the clinician, and the setting in which the encounter occurs. These elements, their complexity and interaction will render the domain well-defined or ill-defined. A patient encounter that is well-defined will have lower levels of complexity. Novice learners must begin with clinical cases that represent well defined domains.[55] These cases allow students to confirm their preliminary application of knowledge to clinical encounters. The apparent lack of “authenticity” of the novice stage of learning is by design. The managed contexts of novice learning allow for the titration of cognitive load and the development of knowledge structures necessary for progression in clinical reasoning. Recalling that misdiagnosis frequently occurs in the setting of ill-defined problems such as atypical manifestations of the disease, educators may be tempted to incorporate such atypical presentations in novice stage case-based clinical reasoning. An example of this early exposure to misdiagnosis-prone complexity would be asking novice students to work through a case of jaw pain that turns out to be a myocardial infarction or right shoulder pain that turns out to be biliary colic (the classic presentation myocardial infarction is chest pain and patients with biliary colic will classically present with postprandial right upper quadrant pain.) Although such presentations do exist and will eventually be important to expose more advanced leaners to such cases, attempting to teach ambiguous presentations too early in the curriculum could lead to confusion. The issue highlights an important aspect of case-based clinical reasoning. Learning to reason clinically through cases - vignettes, OSCE's inpatient simulations - does not

merely convey information, they shape and confirm knowledge in ways previously described in illness script theory. Early introduction to more typical cases helps to confirm knowledge of disease states and organize the learners' categories of understanding.[27,36,47-48,55] Concepts such as disease atypicality, or when a known disease presents in an uncommon way, is a category of understanding that can only emerge in the mind of the learner after sustained and confirmed experience with typicality. Novice learners should be instructed in the concepts of atypicality, look-alike conditions and diagnostic difficulty. This can occur in the form of lecture or reading but this is distinct from the kind of learning that occurs through case-based instruction. [27,36,47-48,55] Incorporating atypical presentations into novice stage learning before the students have some consolidated basis for clinical reasoning could result in significant destabilization of the students' knowledge structuring.[55] Introducing novice stage learners to a wide range of ill-defined domains such as atypicality or complex contexts could lead to students developing disjointed clinical perspectives that are not rooted in epidemiologic realities. Ironically, radical context theorists have suggested that novice stage learners should be exposed to a wide variety of ambiguous presentations and complex contexts. Doing so they suggest will provide opportunities "challenge existing frameworks."[42] Importantly, students must first have frameworks for them to be challenged.

Though it is necessary for students to effectively progress through stages of learning, novice stage of learning must not be merely traversed, it must be transcended: "These discrepancies in aims and tactics (along with others that have been observed) raise the possibility that introductory learning, even when it is "successful" lays foundations in knowledge and in an approach to learning that interfere with advanced acquisition"[55]. Having acquired a foundation through engaging well-defined domains, it becomes necessary to engage with increasingly complex and ill-structured domains. It is only with this sort of incremental engagement with complexity that allows students to progress. Where context radicalists see learning as heavily context bound through all stages, a developmental model views the

early stages of learning as requiring managed learning settings. One first masters clinical vignettes, navigates objective structured clinical encounter (OSCEs) and standardized patient encounters; then learners progress to stable, well-defined "live" patient encounters.[27,36,47-48,55] As learners progress from the novice to intermediate stage, they move into advanced knowledge acquisition. Although the stages of development occur in a progression there is a paradoxical discrepancy between the nature of introductory and advanced learning: "The methods of education in introductory and advanced learning seem, in many ways, to be at odds. For example, compartmentalizing knowledge, presenting clear instances (and not the many pertinent exceptions), and employing reproductive memory criteria are often in conflict with the realities of advanced learning-- knowledge which is intertwined and dependent, has significant context dependent variations, and requires the ability to respond flexibly to "messy" application situations"[55]

As learners engage progressively ill structured domains (complex clinical encounters) it is important for learners to have multiple representations: "...single representations (e.g., a single schema organizational logic, line of argument, prototype, analogy etc.) will miss important facets of complex concepts. Cognitive flexibility is dependent upon having a diversified repertoire of thinking about a conceptual topic. Knowledge that will have to be used in many ways has to be learned represented and tried out (in application) in many ways."[55] As learners progress through more advanced stages of learning, frameworks will need to be modified to account for ill structured problems and novel conditions. An example of this is the seventh category in the neurology framework labeled "ill-defined presentations." All organ systems that are frequently associated with ill-defined complaints (dizziness, cardiology and syncope, weakness) can be modified to contain a category of ill-defined problems. The seventh category serves a kind of "junk drawer" for ill-defined presentations which can then be schematized into an overall framework for ill-defined presentations.

Eventually, learners must be tasked with cases of ever-increasing complexity as biology and biomedicine are intrinsically complex: "...fields characterized by increasing complexity (biology and biomedical science) represent a kind of landscape of ill structured domains."[55] Consequently, Spiro suggests, "...deep understanding of a complex landscape will not be obtained from a single traversal… rather the landscape must be crisscrossed in many directions to master its complexity and to avoid having the fullness of the domain attenuated."[55] Cognitive flexibility theorists register and attend to context in many effective ways but do so in ways that are appropriate to the stage of learning. Moreover, cognitive flexibility theory accounts for the salient elements of interaction between the learner and the environment. As learners progress into the clinical phase of PA education ("supervised intermediates") learners will begin to traverse numerous clinical domains and acquire the experiential knowledge that will allow the learners to expand and tune their knowledge structures. Keeping in mind that not all students will be able to "traverse all domains," that is not all students will have an opportunity to experience, for example, a patient with an atypical presentation of myocardial infarction in a very challenging context. Such domains can be approximately re-created through high fidelity simulation and other components of case-based reasoning.

The physician assistant training model is driven, in part, by ensuring success on the PANCE. A review of the PANCE blueprint indicates that the topics required for study and subject to examination on the PANCE are almost exclusively conditions. That is, with few exceptions such as diarrhea and amenorrhea, the PANCE requires students to focus on disease states (e.g. coronary artery disease) rather than problems or clinical presentations (chest pain). Consequently, there is no formal mechanism to ensure that programs are allocating enough curricular resources to the evaluation of problems. With a limited timeframe to complete training, programs may be tempted to defer problem-based evaluation to the clinical phase of training. An important concept in a curriculum of diagnostic proficiency is incorporating the epidemiology of misdiagnosis into the curriculum. By emphasizing conditions such as

stroke, medical educators have an opportunity to provide more curricular resources to conditions responsible for the greatest harms. Understandably, educators will worry about distributing adequate curricular resources across other problems and conditions. However, based on the significance of misdiagnosis related harms it seems prudent to spend more time discussing high-risk conditions like stroke than common and frequently self-limiting conditions such as allergic rhinitis. Other conditions that are overrepresented in the epidemiology of misdiagnosis related harms include aortic dissection, sepsis, etc. etc. in addition to stroke, incorporating more curricular resources to these conditions seems warranted.

Knowledge organization and integration in neurology

Teaching clinical medicine as structured knowledge will require a shift away from the learning of isolated facts and toward integrative knowledge structures. Students will build a basis for understanding clinical medicine through the formation of frameworks. Frameworks will be formulated in terms of clinical presentation categories that are proper to the body system. Clinical problems can be common complaints and cardinal manifestations of disease as well as laboratory abnormalities. The framework for ophthalmology, for example, may be formulated in clinical presentation categories that most efficiently allows the student clinician to locate a patient's chief complaint within a range of possibilities most likely to lead to a correct diagnosis. Ophthalmic conditions are frequently confined to a narrow range of presentation possibilities: visual changes, pain, trauma and lesions or tissue changes of the eye and associated structures. Consequently, a framework for ophthalmic conditions will be framed in terms of these common presentation categories. Encoded within the framework is an organizational structure that guides the preliminary problem representation of a patient's ophthalmic complaint. By correctly locating the complaint within the framework, the clinician has already defined a range of reasonable possibilities for the cause of the complaint. frameworks must be adapted to clinical problems that are proper to the particular body system. In dermatology, for example effective diagnosis is largely a

function of visual pattern recognition that can be achieved through an algorithmic description of cutaneous conditions.

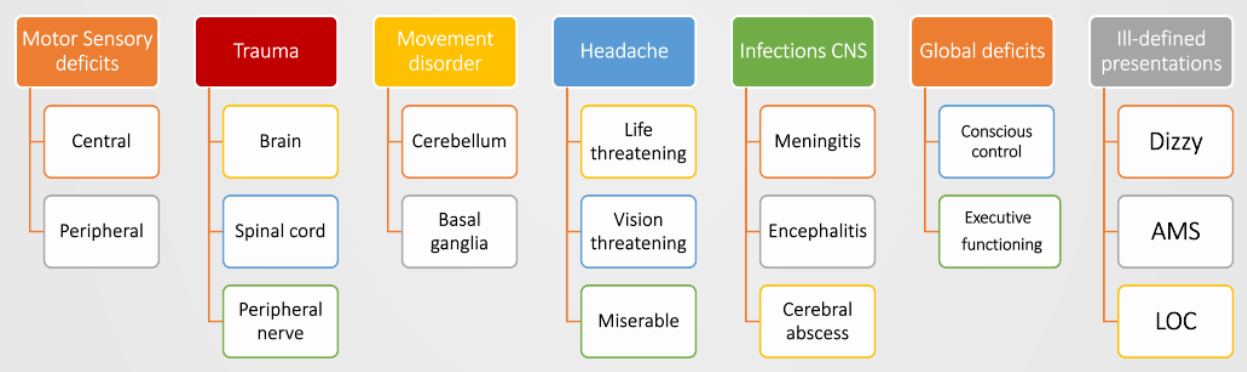
Neurological problems in contrast require the clinician to analyze the "phenomenology" of presentations and consider possible causes in a more analytical and stepwise fashion. Consequently, frameworks for neurological problems will be more extensive and will contain categories appropriate to the neurological system. Hematologic and kidney problems have no clinical presentation that is specific to those organ system problems. They are best framed through laboratory definitions and pathophysiology. Initially students should develop frameworks that are more straightforward and rely on only preliminary supporting courses like anatomy. For example, frameworks in ENT require a foundation mostly in anatomy and the presentation categories are comparatively straight forward. As the students' knowledge base grows with additional supporting courses such as neuroanatomy, pathophysiology and laboratory medicine more complex disease states and schemas can be incorporated. It is also recommended that frameworks are initially formed with a limited number of categories. Research suggests that a limited number of items in the schema provides sufficient germane cognitive load for knowledge acquisition but allows for further development (number seven). With clinical experience students will need to expand their framework as they encounter novel and unlearned conditions. As frameworks for locating patient complaints within an appropriate range of diagnostic possibilities, schemas will be operationalized in the data collection phase of the clinical process beginning with the patient history. The effective use of frameworks is largely predicated upon the successful identification of the patient's chief complaint. That is, chief complaints must be sufficiently clarified to understand what schema of clinical presentation categories the complaint belongs (Appendices 4 and 5 Clinical reasoning lectures I & II). In some cases, the exact problem category may not be fully recognized until the history of present illness sufficiently elaborates the elements of the presentation. Emphasis on early clarification of problem category allows for a more even redistribution

of the cognitive load of hypothesis formation from a later point in the clinical encounter (differential diagnosis) (Appendices 4 and 5 Clinical reasoning lectures I & II). Framing chief complaints in this way also marks a shift away from history taking as a passive recording of information and toward history as a discursive, hypothesis-seeking activity. Having correctly located a complaint in the appropriate clinical presentation category schema the student will begin gathering additional data through the history of present illness (HPI). As the student collects data through the history of present illness diagnostic possibilities will begin to emerge and take priority. The ability to recognize the distinguishing features of various clinical conditions will require not only the inchoate knowledge of the schema but also a more replete understanding of disease states. This replete understanding of clinical conditions will require formation from supporting coursework such as clinical medicine, pathophysiology and laboratory medicine. After taking a history, the student should have some - even if only fragmentary - ideas for diagnostic possibilities. These preliminary ideas can be further explored or excluded, in some cases, by the performance of an appropriate physical exam.

Neurology framework

A framework was developed for clinical problems or presentation categories that are commonly associated with neurology (Appendix 11). The thematic emphasis of the framework is based upon the well-known neurological diagnostic questions: 1. Where is it? (Neuroanatomical location) and 2. What is it? (what type of pathophysiologic insult is giving rise to the problem).[74-76] The categories included motor and sensory deficits, movement disturbance, global deficits, neuro trauma, headaches, neuro infection

and ill-defined presentations (Appendix 11).



Motor and sensory deficits (weakness and dysthesias) were sub categorized as being anatomically central (the brain and/or spinal cord) and then sub categorized by pathophysiologic mechanism (stroke, multiple sclerosis, amyotrophic lateral sclerosis). Peripheral motor and sensory deficits - those arising on the nerve root, nerve, neuromuscular junction, muscle – were sub categorized according to pathophysiologic mechanism. Movement disturbances (tremor, ataxia and involuntary movements) were sub categorized from their anatomical location - the cerebellum or the basal ganglia. Cerebellar insults could include the pathophysiologic mechanisms of tumors, bleeds or chronic degenerative processes. Movement disorders associated with dysfunction of basal ganglia could be described in terms of the pathophysiologic mechanisms of well-known diseases (e.g. Parkinson's disease). Under the category of global deficits, seizures are listed as a problem with "conscious control" as seizures can be experienced as partial (a part of the body exhibiting movement without control) as well as generalized seizures, in which the patient has no conscious control over their body. Seizures are distinct from acute alterations in level of consciousness which include the continuum of delirium and coma. Lastly under global deficits included dementias which represent chronic declines in executive mental functioning (the cardinal feature) with Alzheimer's disease being an important etiology. The category of trauma is itself a pathophysiologic mechanism which can then be broken into neuroanatomical location nerve (neuropraxia), nerve root (disc herniation), spinal cord (complete and

partial insults), brain (TBI and hemorrhages). Under the heading of headache learners are encouraged to parse out headaches as being potentially life-threatening (subarachnoid hemorrhage, brain tumor and infection), potentially associated with blindness (temporal arteritis and benign intracranial hypertension) and then medically significant headaches which include those that will have significant impact on the patient's quality of life. These headaches included migraine headaches, cluster headaches and muscle tension headaches. Another pathophysiologic category in the neurological framework included infections of the CNS: meningitis, encephalitis and cerebral abscess. The last problem category included in the neurology framework is a section labeled "ill-defined conditions." As noted above, there are a number of medical conditions which by their very nature are so ill-defined that it is not immediately clear what organ system or pathophysiologic mechanism could be responsible for the patient's presentation. These ill-defined problems include presentations like weakness, altered mental status, dizziness and fatigue. These problems, as ill-defined presentations, act as a sort of placeholder until more information has been collected to better define the problem.

Evidence-based integration of physical examination and diagnostic evaluations in clinical reasoning

To improve the utility of physical diagnosis in the clinical process it is recommended that the physical examination be reformulated in terms of the core and clusters paradigm.[78] A “core” physical exam refers to a set of physical exam maneuvers that would be performed routinely on any patient presenting for preventative health examination. The core exam would be tailored to the patient’s gender, age and past medical history but would generally contain a set of core elements that are supported by evidence. Additionally, patients with specific complaints would be evaluated using “clusters” of physical exam maneuvers that are appropriate to that complaint.[78] Students will be trained to recognize that patients with sudden onset of unilateral weakness should undergo testing of strength, reflexes and special maneuvers like pronator drift. In this way, physical examination will be seen as an essential component of the clinical process. The clusters of physical exam maneuvers will be taught in

association with their clinical presentation categories so that students understand not only how to perform the maneuvers but what the maneuvers mean in terms of diagnostic information. Formative instruction and summative assessments must be geared towards not only the appropriate performance of the maneuver but testing the students' ability to select appropriate physical exam items for a given clinical presentation category and accurately interpret the results of those maneuvers.

Hypothesis formation-differential diagnosis

Another important component of this process is the prioritized differential diagnosis. Students will be instructed to formulate probabilistic, prognostic and pragmatic components of the differential diagnosis and will be required to know all "can't miss" diagnoses associated with cardinal presentations. Included in the formation of differential diagnosis will be instruction in high-yield symptoms/presentations and their associated evidentiary base such as likelihood ratios for particular physical findings (Appendices 4, 5 and 12). To effectively engage in diagnostic hypothesis evaluation, students will be instructed in the best practices of diagnostic testing. These practices will include choosing the appropriate modality for purpose and accuracy as well as understanding the scientific basis for various testing modalities. Such testing modalities include radiologic imaging, electrocardiography, as well as hematologic and biochemical assessments.

Chapter III. Methodology

This study began with the problem of significant morbidity and mortality associated with preventable medical errors. It has been shown that the most common, costly and dangerous of all preventable medical errors is the problem of misdiagnosis. Although the exact rate of misdiagnosis among physician assistants is not well documented, the growth of the physician assistant profession warrants careful consideration of how PAs can improve their diagnostic capacity. Scholarly consensus suggests that the most effective solutions to the problem of misdiagnosis are those associated with

improving training models in clinical reasoning[7]. This study sought to determine if a curriculum in diagnostic proficiency could improve the clinical reasoning skills and diagnostic capacity in preclinical physician assistant trainees. In "Competencies for improving diagnosis: an interprofessional framework for education and training in health care," outcomes of our current education programs with respect to diagnosis are not enough; that is, the products of our current education programs – health care professionals in practice – are not achieving desirable levels of diagnostic performance[7]. In an attempt to move toward desirable levels of diagnostic performance an experimental curriculum in diagnostic proficiency was developed. As discussed above, the curriculum was devised using evidence-based principles from a literature review. Those principles were then used to formulate specific curricular elements with a particular emphasis on stroke neurology. A summary of the problems with current training models, experimental curricular approaches, evidence-based principles, curricular elements and specific location in this document are given in the table below.

Figure 1 Evidence-based Curricular Elements

Problems with current training models	Experimental curricular approaches	Evidence based principle	Curricular elements	Dissertation & Appendices
Condition oriented (Atherosclerotic cerebrovascular disease)	Problem oriented (Dizziness with dysarthria and ataxia)	Structured knowledge approach (Frameworks & schemas)	Diagnostic schema for neurological conditions	Dissertation pp. 53 – 58 Appendices 3,4, 11
No input from epidemiology of misdiagnosis related harms	Allocates additional time and resources for high-risk conditions	Epidemiologically informed training model (more time on strokes than allergic rhinitis)	High risk conditions featured in OSCE's, case studies and simulations	Dissertation pp. 8 – 11 Appendix 3,4,10
Physical diagnosis checklist- rote memorization and maneuver techniques	Physical examination as data gathering process	Hypothesis driven inquiry; knowledge integration	Use of problem-based scenarios rather than checklists, verbalization of target condition for all physical exam maneuvers	Dissertation pp. 15,16, 38, 56 – 58 Appendices 4,5, 12
Emphasis on accretion in didactic education, defers clinical reasoning to clinical phase of education	Integration of clinical reasoning in didactic education	Clinical reasoning, knowledge integration, cognitive load management	Case-based clinical reasoning with spaced repetition	Dissertation pp. 38-40 Appendices 4,5,11-18
Deferral of clinical reasoning and cognitive load mismanagement	Integration of clinical reasoning in didactic curriculum with germane cognitive load management	Germane cognitive load management and learning optimization	Titration of cognitive load to incrementally increase the complexity of clinical reasoning (begin with E – ENT, paper case studies initially, partial task before whole task)	Dissertation pp. 38-40 Appendices 4,5
Neuroanatomy taught with traditional teaching methods	Streamlined and heuristically crafted neuroanatomy lectures	Structured knowledge approach	Interactive neuroanatomy lectures and PBLs	Dissertation pp. 15,16, 56 – 58 Appendices 5-12

One problem with current training models, especially physician assistant training models which rely heavily on the PANCE blueprint is that time on content is condition oriented. That is curricula tend to focus on conditions or diagnoses rather than problems or presentations. The curriculum devised and studied here is problem oriented focusing on presentations. The problem orientation of this curriculum was supported by the evidence-based principle of structured knowledge. As discussed above, structured

knowledge provides the benefit of enhancing trainees' ability to master knowledge by improving knowledge organization. The experimental curriculum used a diagnostic schema for neurological conditions to improve the trainee's ability to categorize patient presentations in neurology. Another problem associated with current training models is the lack of input from the epidemiology of misdiagnosis related harms when forming content areas in the curriculum. By focusing on stroke neurology, this curriculum allocated greater time and additional resources on the number one condition associated with misdiagnosis related harms. The curriculum achieved this increase in time on content by featuring strokes and OSCE's, case studies and PBL's.

Paralleling the paradigm shift toward clinical reasoning, training programs are beginning to move away from physical diagnosis checklists as rote memorization and performance of techniques. Included in this curriculum, is the use of physical examination as a data gathering process. Not only must trainees demonstrate appropriate use of physical exam maneuvers but understand the diagnostic value these maneuvers bring to the evaluation of potential diagnoses. This shift toward hypothesis driven physical examination was achieved by using problem-based scenarios rather than checklists and requiring the trainees to verbalize the target conditions for all physical exam maneuvers. Reflected in the experimental curriculum and consistent with the changing paradigm toward clinical reasoning is the integration of clinical reasoning in the didactic component of education. The experimental curriculum achieved this integration by providing case-based clinical reasoning in a manner that was consistent with the trainees' level of education. Where previous training models deferred clinical reasoning to the clinical phase of training, the experimental curriculum introduced case-based clinical reasoning and titrated those exposures the appropriate phase in the didactic curriculum. Care was taken to titrate the cognitive load by moving from partial to whole task, lower fidelity cases to higher fidelity cases and increasing the complexity of organ systems throughout the curriculum. Lastly, conventional training models struggled to deliver effective treatments of neuroanatomy which are essential to effective

performance in neurology. The experimental curriculum delivered a streamlined and heuristically crafted neuroanatomy curriculum. This approach emphasized key clinical features and was delivered using interactive neuroanatomy lectures and problem-based learning exercises. The evidence-based principles and the specific curricular elements were deployed in a physician assistant curriculum which spanned clinical reasoning and the body system of neurology. While the experimental curriculum addressed the whole of neurology, the specific problems and conditions (strokes) were covered by or taught within the experimental curriculum and will be referred to as "covered conditions." The experimental curriculum did not extend beyond neurology and so all other problems and conditions were covered by or taught within the institution's current conventional curriculum. Consequently, any condition not covered in neurology (the experimental curriculum) will be referred to as "an uncovered condition." A comparison will then be performed between problems/conditions covered by the experimental curriculum (stroke neurology) and those not covered by the experimental curriculum.

Measuring the effect of the experimental curriculum on pre-clinical trainees warranted several considerations. As novice stage learners in the preclinical phase of their training, the assessment process required the use of modalities that were both appropriate to their stage of learning and effective in discriminating the impact of the experimental curriculum. Described in greater detail below, the modalities of illness script repleteness and key features assessment were chosen to evaluate the effects of the experimental curriculum. Consequently, the overarching goal of the study was guided by the following research questions:

4. Does exposure to the proposed curriculum of diagnostic proficiency improve illness script repleteness[67] in novice stage learners?
5. Using the key features assessment method[79], does exposure to the curriculum enhance diagnostic capacity in novice stage learners?
6. What are the students' impressions of the curriculum?

To evaluate the curriculum's impact on the clinical reasoning skills of participants, a within-subjects study was conducted using computerized case study evaluations that assessed both illness script repleteness as well as diagnostic capacity using the key features approach.

Exposure to the experimental curriculum

The participants in this study included nineteen preclinical physician assistant students in the didactic phase of training at a Midwestern PA program. After IRB approval and full disclosure of the study, the students were included, only with their informed consent. Care was taken to ensure that students understood that their participation in the study was completely voluntary. Declining to participate would in no way impact their performance or standing in the program. The instructional setting for the experimental curriculum included classrooms and laboratories. At the time of exposure to the experimental curriculum, the nineteen participants in this study had completed two semesters of the didactic (preclinical) curriculum. The experimental curriculum instruction that was delivered across three courses “Clinical Assessment and Counseling” and “Laboratory medicine and radiologic principles” and “Clinical medicine.” The experimental curriculum consisted of eight lectures, interactive case studies and objective structured clinical encounters. In addition to these materials, other lectures were delivered as part of the neurology module. Lectures and other elements of the experimental curriculum are listed in Appendix 15 Experimental Curriculum Timetable along with the date of their delivery, time interval and the evidence-based principles deployed in the curricular element. The principal investigator was responsible for the development and delivery of all curricular elements including lectures and case studies. The neurology objective structured clinical encounters (OSCEs) were delivered with the assistance of principal faculty at the principal investigator’s institution. The enacted curriculum began in January 2023 with four hours of lecture introducing the students to the clinical reasoning process. In these lectures, students were introduced to the concepts of schemas in clinical medicine (frameworks and illness scripts) as well as the vignette as clinical encounter. Students were also provided with a

heuristic method for organizing and recalling the elements of the clinical process. Between January and the execution of the neurology module June, students underwent training in the clinical medicine modules that included ophthalmology and otorhinolaryngology. In these models, the students reviewed the cranial nerves as part of the content in those modules. Subsequently, students did not receive neurology content until June 2023 when the students received the streamlined curriculum in neuroanatomy, neuroradiology and diagnostics, neurology physical diagnosis, and neurology cases (OSCEs). Between the experimental curriculum and other lectures in clinical medicine, students received a total of 31 hours of content in neurology. The conditions highlighted for evaluation purposes in this study were 1) posterior circulation stroke (dizziness and dysarthria) and anterior circulation stroke (cardiogenic thromboembolic stroke). In the data collection phase of the study, posterior circulation stroke was presented as a case study and students were evaluated for diagnostic accuracy. The cardiogenic thromboembolic CVA was given as a condition and students were required to provide an illness script for that condition.

The uncovered conditions and problems included palpable purpura with hematuria (Henoch-Schönlein purpura) and acute adrenal insufficiency. Palpable purpura and hematuria were presented as a condition for students to evaluate in case study format. The ultimate diagnosis, Henoch-Schönlein purpura, was covered in the nephrology module and covered again, in contrast to non-palpable purpura, in pediatric hematology. In the nephrology clinical module where this condition was covered, students received a total of 30 hours of instruction. This instruction was not provided by the principal investigator and included both principal faculty and guest lecturers. Acute adrenal insufficiency was covered in the endocrinology module during which the students received a total of 20 hours of instruction. Acute adrenal insufficiency was covered a second time in the emergency medicine module (adrenal crisis and distributive shock). In Appendix 16 “A Comparison of Covered Versus Uncovered Conditions” the time lag and organ system coverage intervals are summarized. All organ systems - both those covered by the

experimental curriculum and those not covered by the experimental curriculum - received a similar number of instructional hours in the curriculum. Importantly, elapsed time between primary coverage and additional iterations was much shorter for the uncovered conditions. Henoch-Schönlein purpura received its initial treatment two months and three weeks prior to the evaluation and received an additional iteration five weeks prior to the evaluation. Adrenal insufficiency received its primary treatment three months and three weeks prior to the evaluation and an additional treatment three weeks prior to the evaluation.

In contrast, posterior circulation stroke received its primary treatment four months and three weeks prior to the evaluation and was covered tangentially (central versus peripheral vertigo) 10 months prior in the otorhinolaryngology module. The primary treatment for cardioembolic cerebrovascular accident (anterior CVA) was conducted four months and three weeks prior to the evaluation and received an initial treatment eight months prior as part of the instruction regarding atrial fibrillation.

In summary, the uncovered conditions were treated in time frames closer to the date of the evaluation and were taught using principal faculty and guest lecturers in traditional lecture format. The covered conditions were taught at time intervals greater than the uncovered conditions but were taught by the principal investigator and included high-yield elements including case studies highlighting the conditions. A comparison of conventional and experimental curricula is provided in the table below.

Instruments used to collect data

The rubrics and evaluation process stem from two established theoretical frameworks for the assessment of clinical reasoning. First, is the key features approach that was used in the development of the case study component of the study. The key features approach to clinical reasoning assessment was initially developed in the 1980s and has been validated extensively across multiple domains[79]. Key

features assessments have been used in the evaluation of medical students, residents, pharmacists and veteranarians.[79] In 2018, Bordage and Page published "The key-features approach to assess clinical decisions: validity evidence to date." In this article, the authors assessed publications from 1984 to 2017 that used the key features approach to assessment of clinical reasoning. Bordage and Page evaluated the articles for five sources of validity evidence according to the Standards for Educational and Psychological Testing: content, response process, internal structure, relations to other variables and consequences.[79] The researchers found that "The accumulated validity evidence for the KFs approach since its inception supports the decision-making construct measured and its use to assess clinical decision-making skills at all levels of training and practice and with various types of exam formats."[79]

As the name suggests, the key features approach formulates assessments in ways that require examinees to identify crucial or key features within a clinical vignette. These key features drive pivotal decision-making and if a key feature is unrecognized or misinterpreted, subsequent decision-making is prone to error. An example would be a vignette in which a 23-year-old prima gravida presents with a third trimester vaginal bleed. The key feature in such a case is consideration of placenta previa and the subsequent clinical decision is to avoid performing a vaginal exam which may precipitate catastrophic hemorrhage. By assessing the nexus of relationships, examiners are able to assess the reasoning process itself rather than loose association or guessing the correct answer. In key feature examinations, examinees must exhibit a chain of reasoning to achieve the ultimate diagnosis or component in the clinical reasoning process. Two cases were presented using a web-based platform (Nearpod). One case was covered using the experimental curriculum and the other case was not covered by the experimental curriculum.

The case covered by the experimental curriculum was a posterior circulation stroke (CVA – cerebrovascular accident). After logging into the system, the students received instructions on how to navigate the case materials. Participants were given an initial stem (preliminary information of the

clinical vignette) and then they were required to respond in a subsequent slide to a free response question (FRQs). The free response questions were designed to elicit the ability to identify key features within the clinical case. In some slides, the participant was required to choose from an extended panel of choices. Extended choice panels (more than one would be chosen) were provided at pivotal points in the clinical process such as the choice of physical exam maneuvers and diagnostic tests. The extended range of the multiple-choice panels (up to thirteen items) required greater discrimination than five item MCQ and parallels order panels common in contemporary electronic medical records. The combination of free response questions and extended multiple-choice panels provided a greater range response. This configuration of the assessment tool is consistent with the historic use of the key features approach. All items were scored in ways that were appropriate to the case being presented. In the case of posterior circulation CVA, the categories and choices were formulated using key features that are specific to those cases. Points were awarded in ways that equal to a maximum of five points per case study.

Case Study Rubric for Covered Condition: Posterior circulation stroke

The key features assessed in the covered case of posterior circulation stroke included appropriate physical examination, differential diagnosis, diagnostic evaluation plan and final diagnosis. Points were awarded for the appropriate choice and interpretation of physical exam maneuvers. The results were either provided in written descriptions or in images. The differential diagnosis panel, also a free response question, gave credit for any variation on posterior stroke circulation disturbance (brainstem stroke, stroke, cerebellar deficit). If a participant suggested an inappropriate peripheral cause for the patient's symptoms (benign paroxysmal positional vertigo or labyrinthitis), a point was deducted. The next key feature evaluation was to provide an "evaluation plan formulated in a manner appropriate for the patient's presentation." In this section participants were graded on their choice, sequence and interpretation of diagnostic tests. The last section in the case study was “differential diagnosis and/or final diagnosis that is supported by clinical reasoning." Points were awarded in the

manner specified in the rubric.

Case Study Rubric for uncovered condition: Henoch-Schönlein purpura

In a manner similar to the covered case, the case involving Henoch-Schönlein purpura the participants were given a stem (leading portion of the and yet). The first key feature assessed was "selection and interpretation of physical exam findings." Points were awarded for both choosing appropriate physical exam techniques as well as interpreting a description or image of any exam findings. The next key feature was differential diagnosis supported by clinical reasoning. In this case a wider range of possibilities was permitted as the history and physical exam was less suggestive of the underlying condition than in the case of a posterior circulation CVA. The next key feature assessed was evaluation and appropriate tests. The last key feature to be assessed "differential diagnosis or final diagnosis supported by clinical reasoning." The specific points structure is available on appendix 14.

The other theoretical framework used in the development of the studies assessment is illness script theory. Illness script theory has also received widespread utilization since the 1990s. As noted above, illness scripts are schemas that allow clinicians to effectively process clinical information into patterns or scripts that can be used to drive clinical reasoning process. Some studies have focused on the illness script "maturity" or the extent to which an illness script matches those of expert clinicians. For the purpose of this study, illness script repleteness was used instead as the critical principle. For the rubric, illness script repleteness refers to sufficient critical mass of interconnected judgments that drives the clinical decision-making process.

To assess illness script repleteness and utility participants were asked to construct illness scripts upon being given the name of a particular condition. As an example, a participant may be given the condition “coronary artery disease.” The participant then wrote an illness script using only information from their long-term memory. Adapting the work from the Keemink et al, rubrics for repleteness and

utility were tied to the principal components of illness script theory: the fault (pathophysiological mechanism), the enabling condition (patient features and contextual factors) and the consequences (signs and symptoms)[14]. Learners participated in exercises for conditions covered by the experimental curriculum as well as an equally complex condition that was taught using the institution's existing conventional curriculum. A comparison and statistical analysis were conducted between conditions covered by the experimental curriculum and those conditions covered by the existing conventional curriculum.

In the illness script portion of the study, participants respond to the condition prompt by telling the story or illness script of the condition. Repleteness assesses not only the long-term memory of the disease state but how the knowledge is structured in the mind of the participant. Points were awarded if the participant with illness script features that were sufficient to warrant action. That is, if the data were sufficient in detail and organization that a reasonable plan could be made from the three elements of the script – enabling conditions, fault, and consequences. To receive credit for data supplied in enabling conditions, the participant was required to provide sufficient information in the other two categories of the illness script. This would exclude guessing or categorical error. All categories were numerically weighted in a way that parallels the contribution to the repleteness of the illness script.

Rubric for illness script repleteness: Cardiogenic Thromboembolic stroke (covered condition)

Under the category of enabling condition participants were awarded a maximum of points for documenting enabling condition which includes patient features and context. In the case of cardiogenic stroke, the participant received credit (0.25 points) for listing age greater than 65, history of hypertension, history of CHF, history of CVA/TIA or coronary or peripheral arterial disease. Additionally, one point was awarded for including atrial fibrillation in the patient's history. A thromboembolic cardiogenic stroke would receive credit for risk factors if they included atrial fibrillation in either the

fault or the consequences as there is overlap with these two components. The participant only received credit for atrial fibrillation if the fault/pathophysiology of atrial fibrillation was identified by an electrocardiogram and either a vessel test or a transesophageal echo showing a left atrial thrombus. Under consequences (signs and symptoms) one point was awarded for hemiparesis and dysthesias that occurred in a pattern or altered mental status with aphasia. Alternatively, one point was awarded if the patient exhibited any signs of posterior circulation disturbance.

Rubric for illness script repleteness acute adrenal insufficiency (uncovered condition)

Under enabling conditions, students were awarded 1 point for including any history of immune disorders (type I diabetes, vitiligo) or including history of chronic corticosteroid use. For consequences, participants were awarded 1 point for any acute nonspecific syndrome (abdominal pain, nausea/vomiting/diarrhea, generalized weakness, altered mental status). Additionally, participants were awarded 0.5 points for including hypotension and 0.5 points for hyperpigmentation. Under the category of fault, participants were awarded one point if they ordered adrenocorticotropic hormone stimulation test and 0.5 points for hyponatremia as well as hyperkalemia.

Data Collection

Participants underwent a series of web-based exercises that evaluated illness scripts and diagnostic accuracy. The web-based exercises included case studies in which participants analyze clinical data and record their thoughts about the different components of the clinical process. Each of the participants underwent evaluation of conditions and problems covered by the curriculum and then another set of exercises that evaluate performance on conditions not covered by the curriculum. The content regarding conditions and problems not covered by the experimental curriculum was delivered using traditional methods of the institution's existing curriculum.

Qualitative and Quantitative Measures

Quantitative measures for the first research question (Does exposure to the proposed curriculum of diagnostic proficiency improve illness script repleteness in novice stage learners?) and the second research question (Using the key features approach, does exposure to the curriculum enhance diagnostic capacity in novice stage learners?) included scores from the case study exercises. For the third research question (What are the students' impressions of a curriculum in diagnostic proficiency?), quantitative data from student surveys was collected. Responses were analyzed using a an open coding approach. When participants responded with a sufficiently specific comment, those responses were organized under categories if there were two or more responses (<10% of cohort). After initially reviewing the comments, six broad categories were established: structured knowledge (schemas), high risk condition highlights, use of spaced repetition, use of cognitive load titration, use of case-based clinical reasoning and experimental curriculum (general). When participants responded with a generalized favorable remark (e.g. "the use of cases was very helpful to my education"), those responses were recorded under a general heading of "favorable."

Data collection occurred through the administration of case studies that were conducted in a computer lab. Students self-assorted to computers within a university computer lab and no identifying information or IP addresses were collected. The students were given an envelope that contained one of nineteen randomly generated seven-digit numbers and a null number (7777777). Any student who chose to decline participating in the study and having their data collected could enter the null number (7777777). However, all nineteen students entered a randomly generated number. The random numbers were entered into a name panel using the Nearpod web-based platform. Case studies and

illness script exercises were all conducted using this platform.

Chapter IV. Results

Data from student surveys and case study exercise scores were compiled and analyzed using SPSS version 29. A comparison was made between results on curriculum-covered conditions versus conditions not covered by the curriculum. The topics covered by the experimental curriculum significantly outperformed the uncovered topics. For the case study exercises, covered topic 1 (posterior circulation stroke) scored an average mean of 88% versus uncovered topic 1 (Henoch Schönlein purpura) which scored 46% (Figure 3 below). For illness script repleteness, covered topic 2 (cardiogenic thromboembolic stroke) scored a mean of 62% where uncovered topic 2 (acute adrenal insufficiency) scored 16% correct (Figure 2 below). Descriptive statistics and normality were tested for both the case studies and illness script repleteness. For the case studies portion of the study, the difference between cases covered by the experimental curriculum and performance on those cases uncovered produced a case difference (diff_case) mean of 2.04. The median for case differences was 2.7, the variance 3.1 and standard deviation of 1.78. Skewness was measured at -.306 with a standard error of .524. Kurtosis was listed at -1.252 with a standard error of 1.014. The mean difference between the covered illness script scores and the uncovered illness script scores (diff_illscript) was 1.29. The median for illness script difference was 2.00, variance 2.86 and standard deviation 1.69. Skewness was measured at -0.400. kurtosis measured at -.199 with a standard error of 1.014. The condition of normality was met for both data sets.

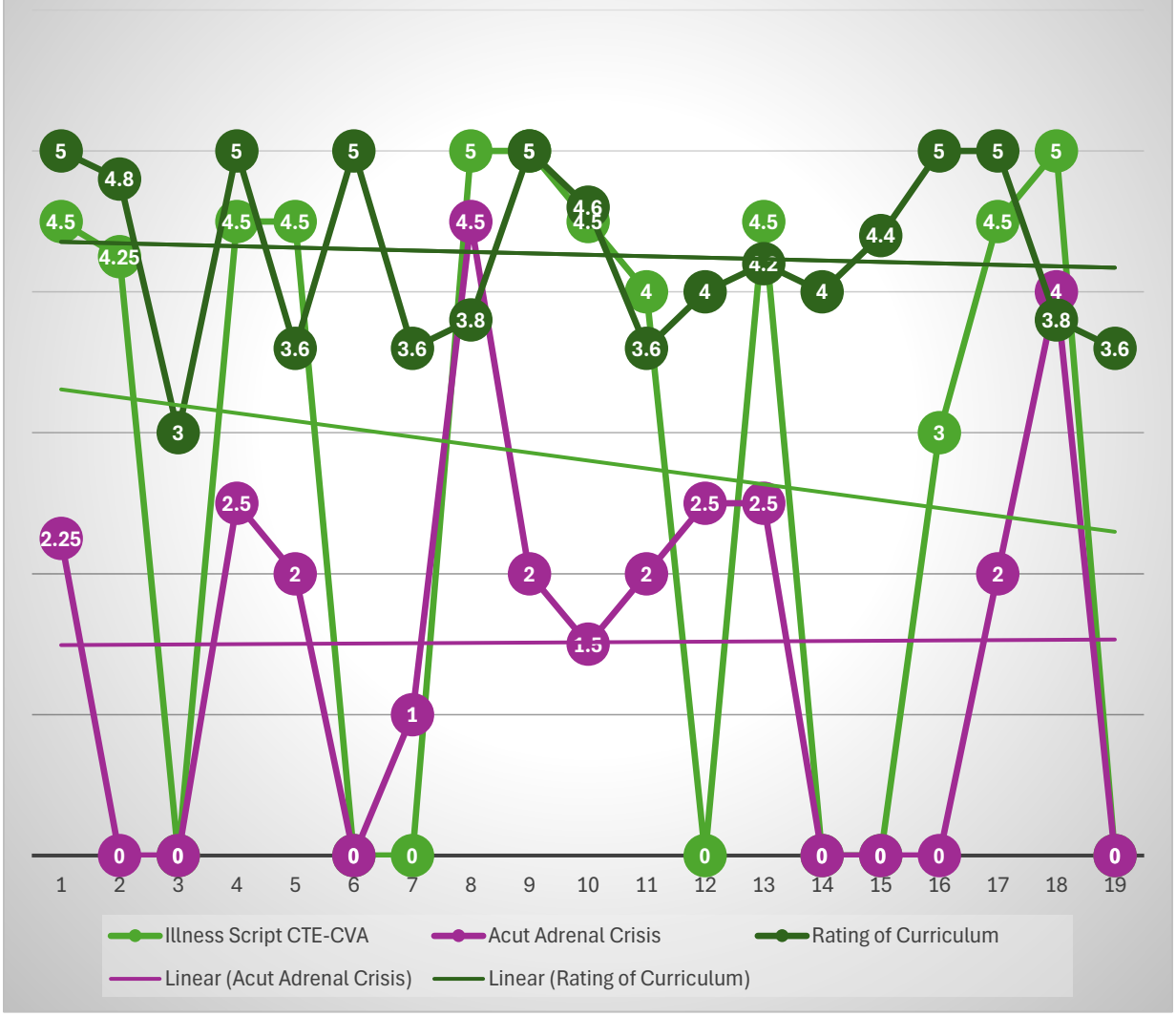
Paired samples t-test showed that there was a statistically significant difference between performance on the case studies associated with the experimental curriculum versus conditions not covered the experimental curriculum: t= 5.005, degrees of freedom 19, p<.0001 and Hedges G of 1.1. A statistically significant difference was also observed in the difference between illness script performance

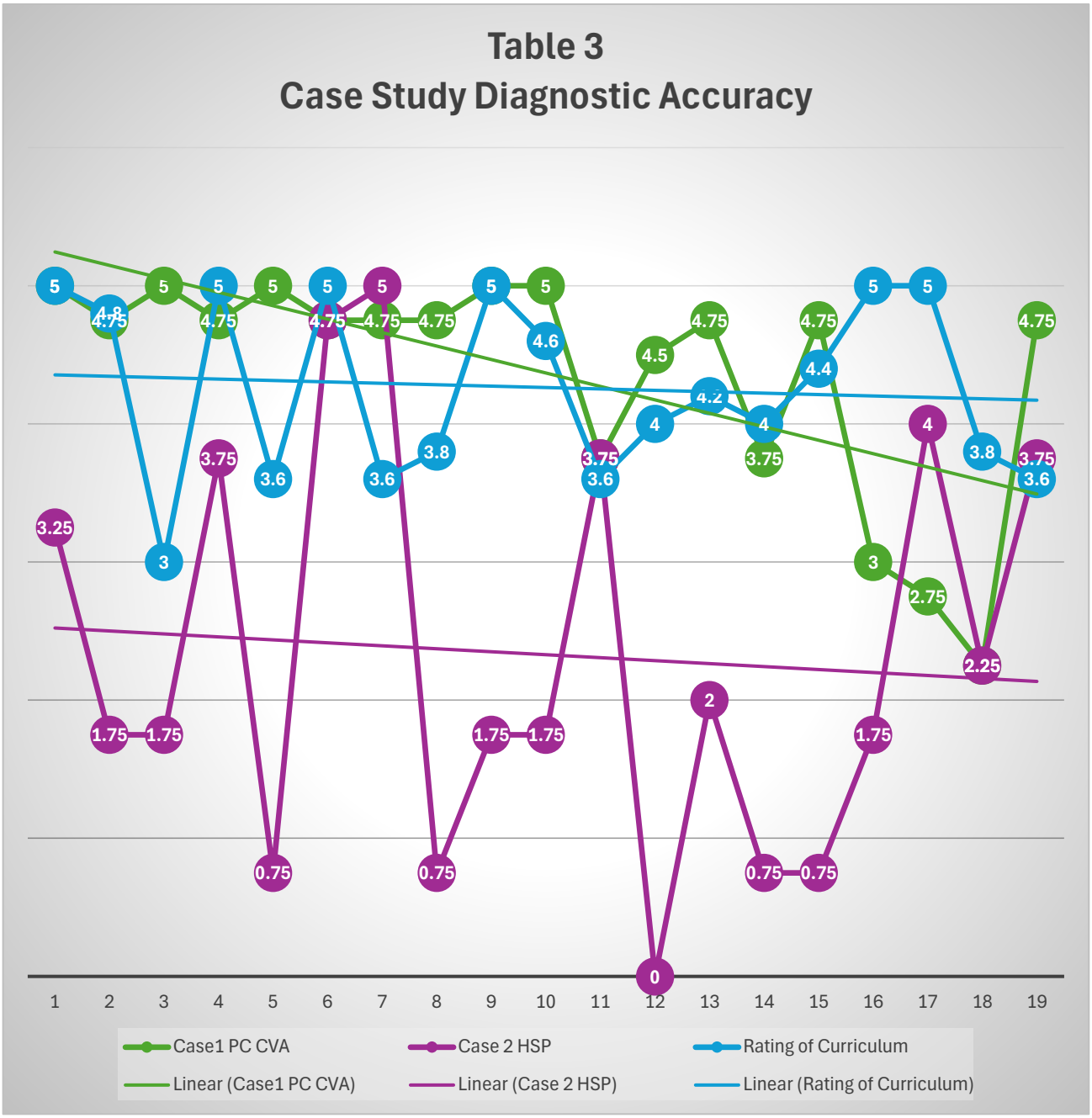
on conditions covered by the experimental curriculum versus those not covered by the experimental curriculum: t=4.470, degrees of freedom 18 , p<.0001 and a Hedges G of 0.982.

As a curriculum implementation project, an important component of this study was assessing participants' attitudes toward the experimental curriculum. This assessment took the form of five questions that were listed at the end of the data collection exercises as well as an open-ended portion for the students to provide their thoughts regarding the experimental curriculum. The first question (Student Question 1 = SQ 1) assessed the participants' attitudes regarding the structured knowledge component of the curriculum with the question "rate the effect that the structured knowledge approach had in improving your ability to reason clinically." SQ 1 had a mean response of 3.8 out of five and was the lowest rated component of the experimental curriculum. Only 32% of the participants rated it a five out of five, 16% of participants rated it a 4/5 and 53% of the participants rated it a 3/5. SQ2 Assessed the participants attitudes regarding high-risk condition highlights with the question "rate the effect that high-risk condition highlights approach had in improving your ability to reason clinically." SQ2 had a mean response of 4.2 out of five. 42% of respondents rated this aspect of five, 32% rated this aspect of four and 26% rated this aspect a three. Student question 3 (SQ 3) assessed the participants' attitudes regarding the spaced repetition component of the curriculum with the question "rate the effect that the spaced-repetition approach had in improving your ability to reason clinically." Participants rated this component of the curriculum with a mean response of 4.4 out of five. 58% of the respondents rated this element of five, 32% rated the element a four and 11% of respondents rated this element a 3/5. SQ 4 assessed the participants' attitudes towards the use of cognitive load titration in the experimental curriculum with the question "rate the effect that the cognitive load titration had in improving your ability to reason clinically." SQ 4 scored an average mean of 4.4 out of five. 53% of respondents rated the item 5, 42% of the respondents rated the item a 4, and 5% of the respondents rated the item a 3. Student question 5 (SQ 5) assessed the participants' attitudes toward the use of case-based clinical

reasoning with the question “rate the effect that the case-based clinical reasoning approach had in improving your ability to reason clinically.” SQ 5 scored a mean response of 4.4 out of five. 53% of participants rated this element a five, 37% rated this element a four and 11% of the participants rated this element a 3/5. The overall mean for responses to all questions regarding the experimental curriculum was 4.2 out of 5.

Table 2
Illness Script Repleteness





Qualitative data

Following the five quantitative questions, students were provided to space to provide free text feedback with the prompt "in the space below, please provide any thoughts or recommendations you have for improving the clinical reasoning component of the PA curriculum." Responses were analyzed using a an

open coding approach. When participants responded with a sufficiently specific comment, those responses were organized into categories noted above if there were two or more responses (>10% of cohort).

Students provided favorable responses to high-risk condition highlights (3), use of spaced repetition (3) use of case-based clinical reasoning (3) and two participants responded favorably about the experimental curriculum in general. Under the heading of experimental curriculum three subcategories emerged. The first was “active learning.” Three students reported that the experimental curriculum promoted active learning in their acquisition of clinical reasoning knowledge and skills. Two students reported that the use of objective structured clinical encounters (OSCEs) helped the students integrate the knowledge and skills that they had acquired in two a more realistic clinical setting. Lastly, four participants expressed a need for more case studies in the curriculum.

Chapter V. Summary, Conclusions and Recommendations

Summary

Analysis of the quantitative data regarding the key features case studies and illness script exercises suggests a significant performance difference between conditions covered by the experimental curriculum and those not covered by the curriculum: CS 1 (posterior circulation stroke) - 88%, CS 2 (Henoch-Schönlein purpura) – 46%, IS1 (cardiogenic thromboembolic stroke) – 62%, IS2 (acute adrenal crisis) – 16%. The performance comparisons clearly favor the benefits of the experimental curriculum and this is supported by effect sizes. Also, illness script exercises demonstrated a moderate correlation that was statistically significant. However, there was no statistically significant correlation when comparing the case study means.

Statistical analysis reveals a mixed picture between performance and effect sizes versus statistical significance (in the case study mean comparisons). There are several possible confounding

factors in this study. The first issue is the small sample size of n=19. Additional data points from larger sample sizes may provide some correction to analyses that are limited by a small sample size. Other factors to consider are structural issues within the study. For example, the principal investigator provided not only the design of the curriculum but also provided the classroom and laboratory instruction for the experimental curriculum in stroke neurology. The uncovered conditions were taught by other faculty and/or guest lecturers. Another factor to consider is that the experimental curriculum used case studies as formative experiences whereas the uncovered conditions were not associated with case studies. The effectiveness of case-based clinical reasoning is well-established in the literature. Another structural component to consider is case similarity in the covered conditions. To evaluate stroke neurology, this core topic of the curriculum, stroke syndromes were featured in both types of assessments (key feature cases and illness script repleteness). The presence of a posterior circulation stroke in the first exercise may raise the concern of content priming so that that students could transfer stroke content to the second case study on anterior circulation strokes. Interestingly, this does not seem to have been the case. Approximately 30% of the respondents erroneously produced an illness script for acute coronary syndromes. One cannot be certain about the cause of this irregularity, but the most likely explanation is a mere terminological error in which the respondents equated “cardiogenic” as acute coronary syndrome. A similar effect was seen in the uncovered illness script exercise in which students were asked to produce an illness script for acute adrenal insufficiency and several respondents produced illness scripts Cushing’s disease or hyperaldosteronism.

Student attitudes toward the experimental curriculum

The participants’ overall quantitative rating of the curriculum is 4.2 (Likert 1 – 5) scale which is overall net positive. This high rating was supported by favorable qualitative remarks by at least 25% of the population. The specific themes that emerged from the qualitative analysis of the remarks were 1. Active learning, 2. The use of case-based clinical reasoning and 3. The structured delivery of OSCE's

(objective structured clinical encounters). Under the theme of active learning, students specifically commented on how the curriculum facilitated active learning: "I thoroughly enjoyed the clinical reasoning introduced to the curriculum. I felt as though it enforced active learning… I truly feel as though I'm being well prepared in the subjects that were taught this way [experimental curriculum]." Another qualitative theme was the structure and delivery of objective structured clinical encounters. Multiple students commented on how the OSC's contributed to their learning of clinical reasoning: "… having OSCE's that give us a proposed stem and require us to formulate a narrowed examination technique based on the complaint more closely mirrors practical applications of the knowledge and allows for a deeper clinical understanding of the examination and diagnostic approach." The third specific theme registered in qualitative analysis was the use of case-based clinical reasoning. Several students expressed their perceived value of cases in the clinical reasoning training and recommended additional cases be distributed throughout the curriculum: "I really valued the case studies and following scenario step-by-step because it allowed me to slow down and really think about what I would do if it was real life." Lastly, the student perceptions high overall net rating and support from qualitative remarks suggests student confidence and value in the experimental curriculum.

Conclusions

The purpose of this study was to determine if an evidence-based approach to clinical reasoning education could improve diagnostic capacity in novice stage learners. The curriculum was developed using best practices and evidence-based principles from the literature on clinical reasoning education. These principles were used to design a curriculum that included interactive lectures, problem-based learning (small group case studies and web-based PBLs), low stakes assessments (quizzes), summative assessments (MCQ examinations) and assessments of entrustable professional activities (OSCEs - objective structured clinical encounters). The scope of this curriculum was limited to neurological problems with an emphasis on strokes as they are overrepresented in epidemiological studies of

misdiagnosis. This study demonstrated that, based on the implementation of the experimental curriculum, clinical reasoning could be improved in novice stage learners as measured by key features case studies and illness script repleteness.

Recommendations

Like all research, this study was embedded in a specific social and historical context that shaped the study's scope and limitations. Specifically, the study was conducted in a Midwestern PA program that needed curriculum modifications across several disciplines. A need for such broad curricular modifications coincided with the implementation of the study and consequently the implementations, though necessary for the program's improvement, were multiple and their effects cannot be certain. Although the experimental intervention of the study was less focused than other projects, this study may be thought of as framing a program of research through which several additional studies may be conducted. Recommendations for future directions in this area of study will be offered in two sections. The first section will include recommendations for improving subsequent iterations of the current study. The second section will include possible approaches to the problem of misdiagnosis and preventable medical error among physician assistants.

Modifications to the current study

1. Focus on structured knowledge approach

Further study regarding the structured knowledge approach to clinical reasoning training is recommended. Additional research into the instructional techniques using frameworks and schemas in the formative process. Among future studies there should be a restricted focus on the structured knowledge approach to clinical reasoning education. Although the current study deployed frameworks and schemas, assessment of the specific intervention is bound up with the overall effect of the curriculum. In future studies, specific elements of the structured knowledge approach such as the use of

problem schemas in clinical reasoning versus non-schematized problems in an uncovered portion of a curriculum. Additionally, researchers may benefit from studying the specific effect of the use of illness script diagrams in the retention of clinical information and later illness script development. There are numerous approaches to representing illness scripts but there has been no attempt to standardize the representation of illness scripts or to formulate them in ways that are accessible to educators. A proposed model for standardizing illness script diagrams is provided in Appendix 17.

2. Use of simulation

To assess the effectiveness of curricular elements, some form of evaluation will be required. Historically, diagnostic failures have been exposed in post hoc evaluations like morbidity and mortality (M&M) meetings. The M&M session allows medical trainees to report their diagnostic errors but only after patients have experienced harm. Although such retrospective assessments are indispensable for ongoing quality improvement, educational programs need to assess trainees' diagnostic capacity in ways that do not expose patients to harm. A more safe and effective method is the use of high fidelity simulation. Although medical simulation has been well studied cornerstone of medical education a focus on misdiagnosis is a recent development. The web-based cases and illness scripts used in this study were adequate for assessing novice stage learners but as learners advance within the curriculum, high fidelity training and assessment modalities will be necessary to titrate cognitive load and increase contextual acuity in the ways described above.

3. Multidisciplinary approach to CR curriculum

As the structured knowledge approach to clinical reasoning becomes confirmed by additional research, a more programmatic approach could be taken in the development of clinical reasoning education. This could take the form of a multidisciplinary team studying schemas (for chest pain, dizziness, weakness) that are most effective in clinical reasoning education. Along with the studies in the use of schemas,

there is also an opportunity to provide calibrated and valid rubrics to evaluate novice stage learners. Although the effect size in overall performance suggested some utility of the experimental curriculum, problems with statistically significant correlation in the case study group warrant further evaluation of rubrics.

Approaches to the problem of misdiagnosis and preventable medical error among physician assistants

1. Enhanced reporting of preventable medical error among physician assistants

As described above, preventable medical error among physician assistants is difficult to tract because of the current reporting system. The preponderance of data regarding medical error is extracted from medical malpractice databases. In these databases, the physician assistant or other advanced practitioner may be named secondarily or not named due to the doctrine of higher respondent. An important project would be to provide a comprehensive review of all available databases, determine if physician assistant involvement can be determined and to search for alternative databases regarding physician assistant preventable medical error.

2. Scholarly work to raise awareness of preventable medical error among PAs

As previously described in this study, the terms misdiagnosis or any variation on preventable medical error have yet to be mentioned as a theme of any publication in the profession's leading journals (Journal of the American Academy of physician Associates and Journal of Physician Assistant Education). An important project would be a descriptive article that outlines the ill-defined nature of the problem in the historic opportunity to be proactive in formulating preventative responses to reduce preventable medical error among physician assistants.

3. Model of clinical expertise for PAs

In physician training, models of expertise acquisition have been well described and entail stages of

development and recommendations for calibration. Although there is a great deal of overlap in the training and practice patterns between physicians and physician assistants, a worthwhile project would be to develop a model of clinical expertise acquisition among physician assistants. This model would account for program training models, practice patterns, changes in specialty and other features that are unique to physician assistants.

4. High-risk condition highlights in PA curricula

Multiple interventions used in the study make it difficult to draw precise correlations, but it is noteworthy that the experimental curriculum produced a significant over performance in the high-risk condition of posterior circulation stroke. Posterior circulation stroke is overrepresented in the conditions responsible for misdiagnosis. Although this over performance by the participants could be attributed to mere bulk effect in which more time and content regarding the specific topic equates to improved performance. Improving performance in these high-risk conditions is precisely what programs should be

achieving.

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Appendices

Appendix 1 Physician Assistant National Certification Exam National Data

Appendix 2 Physician Assistant National Certification Exam National Performance Data (Neurology)

Appendix 3 Lecture PowerPoint on Clinical Reasoning I

Appendix 4 Lecture PowerPoint on Clinical Reasoning II

Appendix 5 Lecture PowerPoint on Neuroanatomy Overview

Appendix 6 Lecture PowerPoint on CNS Environs, Vasculature And Pathophysiology

Appendix 7 Lecture PowerPoint on Movement And Sensation (Neuroanatomy)

Appendix 8 Lecture PowerPoint on Cranial Nerves

Appendix 9 Lecture PowerPoint on Movement as Volition And Coordination

Appendix 10 Lecture PowerPoint on Stroke Pathophysiology and Clinical Phenomenology

Appendix 11 Lecture PowerPoint Approach to The Neuro Patient (Clinical Assessment and Counseling)

Appendix 12 Neurology Case Studies (Clinical Assessment and Counseling)

Appendix 13 OSCE #2 with rubric (Clinical Assessment and Counseling)

Appendix 14 Rubric for Evaluating Student Key Feature Case Studies and Illness Script Repleteness

Appendix 15 Experimental Curriculum: Content and Exposure Times

Appendix 16 Covered conditions and organ systems versus uncovered conditions and organ systems

Appendix 17 Illness script diagrams

Appendix 1

	ALL PROGRAMS (2022) Percent Correct Score			
	First-Time Exams		All Exams	
CONTENT AREA	Mean	s.d.	Mean	s.d.
ORGAN SYSTEMS				
Cardiovascular System	82	9	82	10
Dermatologic System	80	13	79	13
Endocrine System	78	12	77	12
Eyes, Ears, Nose, and Throat	79	11	79	11
Gastrointestinal System/Nutrition	77	10	76	11
Genitourinary System (Male and Female)	79	13	78	13
Hematologic System	78	14	77	14
Infectious Diseases	76	12	75	13
Musculoskeletal System	76	11	75	12
Neurologic System	74	11	74	12
Psychiatric/Behavioral	82	11	82	11
Pulmonary System	75	11	74	11
Renal System	79	13	78	13
Reproductive System (Male and Female)	75	12	75	12

Appendix 2

	2017 National	2018 National
ORGAN SYSTEMS		
Cardiovascular	81	81
Dermatologic	76	81
Endocrine	79	78
Eyes, Ears, Nose, and Throat	81	80
Gastrointestinal/Nutrition	78	79
Genitourinary	76	77
Hematologic	82	76
Infectious Diseases	79	81
Musculoskeletal	80	78
Neurological	79	79
Psychiatric/Behavioral	80	80
Pulmonary	79	79
Renal System	NA	NA
Reproductive	78	79
TASKS		
Applying Scientific Concepts	77	77
Clinical Intervention	78	75
Formulating Most Likely Diagnosis	82	82
Health Maintenance/Patient Education	77	77
History Taking and Performing Physical Exams	80	82
PANCE Professional Practice	NA	NA
Pharmaceutical Therapeutics	78	78
Using Lab and Diagnostic Studies	80	80

Appendix 3

From complaint to diagnosis

Clinical Reasoning and the Process of Diagnosis

Perdue

Appendix 3

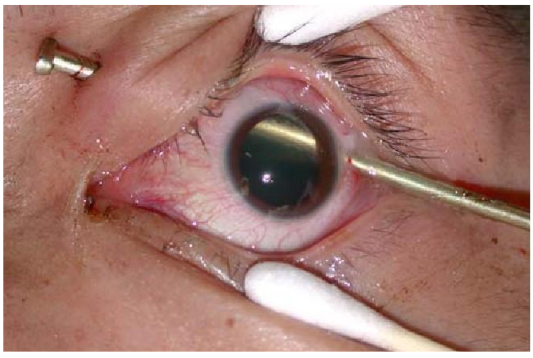
Clinical Encounter

Problem

Prevention

Problems

30 y/o with Eye Pain



72 y/o with weakness

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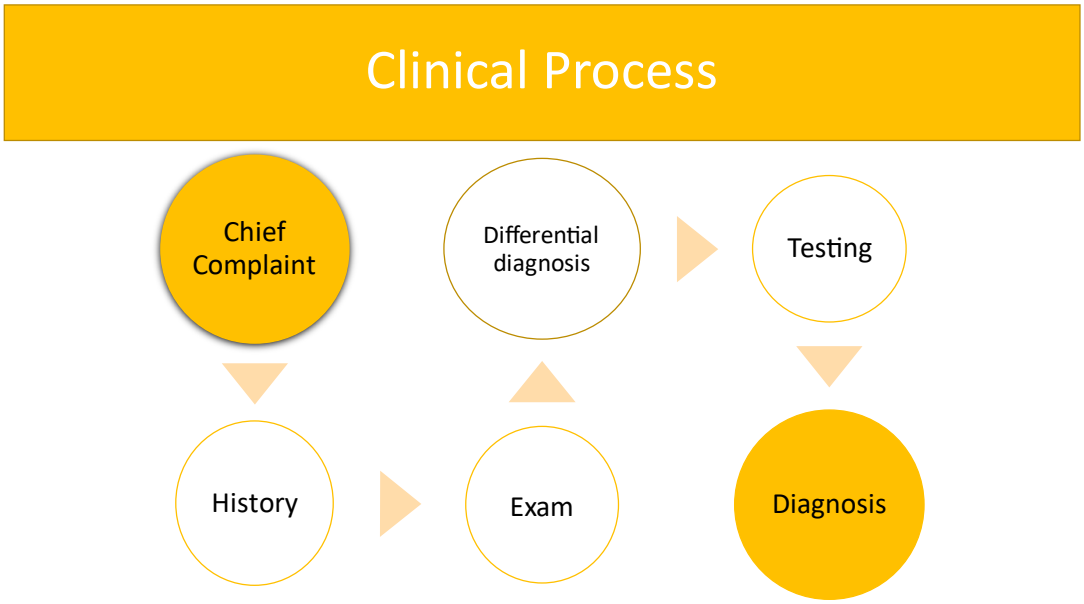
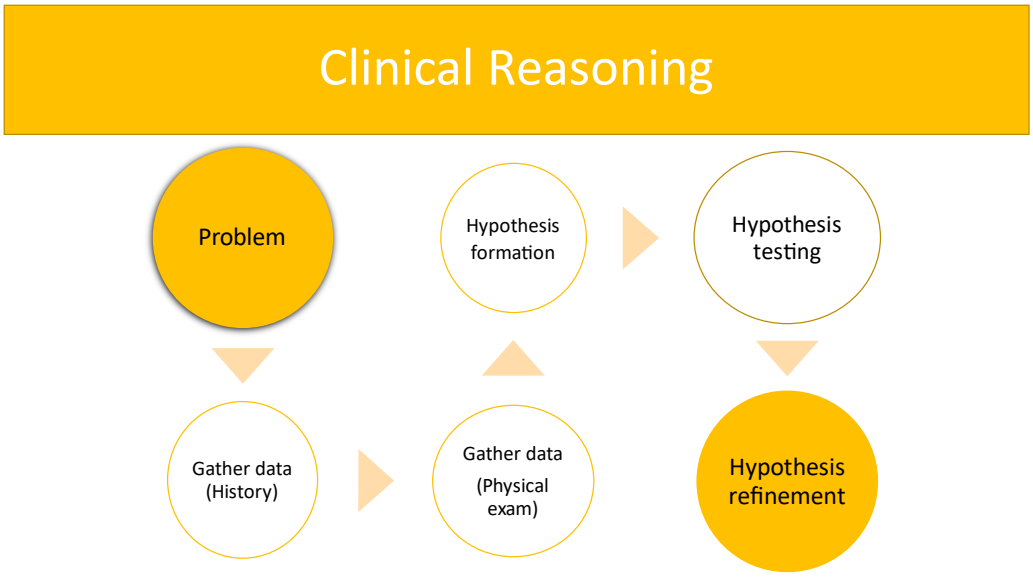
Appendix 3


Clinical Process
Chief Complaint
Differential diagnosis
Testing
History
Exam
Diagnosis


Clinical Reasoning
Problem
Hypothesis formation
Hypothesis testing
Gather data (History)
Gather data (Physical exam)
Hypothesis refinement

Appendix 3

Problem Representation

History

- Subjective
- Chief complaint
- History of present illness
- Medical & Surgical history
- Family history
- Social history
- Review of systems

Appendix 3

Chief Complaint

- Complaints - Chief among many
- Clarified chief complaint
- Clinical problem category
- Initial problem representation
- Proper diagnostic trajectory

- "Ankle pain" - pain at base of fourth and fifth metatarsals
- “Tired" - dyspnea on exertion
- "Dizzy"-near-syncope

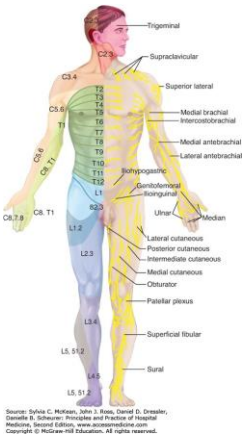
History of Present Illness

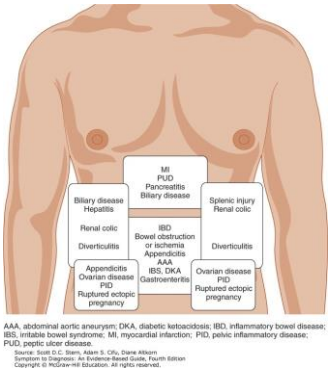
SOCRATES

- **S**ite
- **O**nset
- **C**haracter
- **R**adiation
- **A**ssociated signs and symptoms
- **T**iming
- **E**xacerbating, alleviating factors
- **S**everity

Appendix 3

History of Present Illness

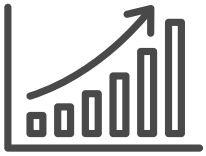




AAA, abdominal aortic aneurysm; DKA, diabetic ketoacidosis; IBD, inflammatory bowel disease; IBS, irritable bowel syndrome; MI, myocardial infarction; PID, pelvic inflammatory disease; PUD, peptic ulcer disease.

- SOCRATES
- Site
- Point to specific area if possible
- Focal versus Diffuse
- Anatomical distribution?

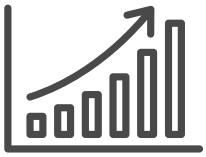
History of Present Illness



- SOCRATES
  - Onset
    - Sudden
    - Subacute
    - Chronic

Appendix 3

History of Present Illness



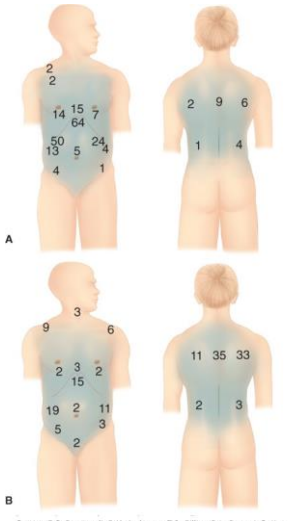
- SOCRATES
  - Onset
    - Sudden
    - Subacute
    - Chronic

History of Present Illness

- SOCRATES
  - Character
    - Verbal descriptors
      - “Sharp”
      - “Dull”
      - “Pressure”
      - “Squeezing”

Appendix 3

History of Present Illness



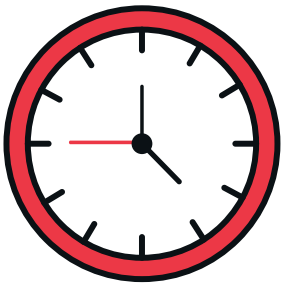
- SOCRATES
  - Radiation
    - Does it move to another area?
    - Radiculopathy
    - Myocardial infarction
    - Muscle tension headache
    - Nephrolithiasis
    - Biliary Colic

History of Present Illness

- SOCRATES
  - Associated signs and symptoms
    - Syndromes
    - Pertinent negatives and positives
      - M. I.
        - N/V, diaphoresis, SOB
    - Doesn't fit?

Appendix 3

History of Present Illness



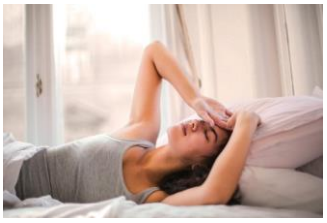
- SOCRATES
  - Timing
  - How long?
    - Time interval
    - Happened before?

History of Present Illness

- SOCRATES
  - Exacerbating, alleviating factors

Appendix 3

History of Present Illness



- SOCRATES
  - **S**everity
  - 1 – 10 Scale
  - Congruity with general presentation?

History of Present Illness

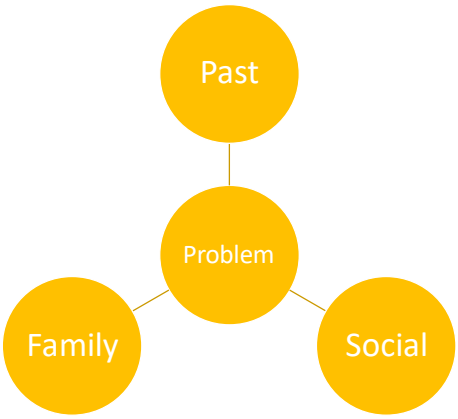
- Depression
- Fatigue
- Difficulty concentrating
- Fever
- Generalized weakness

- Site
- **O**nset
- **C**haracter
- Radiation
- **A**ssociated signs and symptoms
- **T**iming
- **E**xacerbating, alleviating factors
- **S**everity

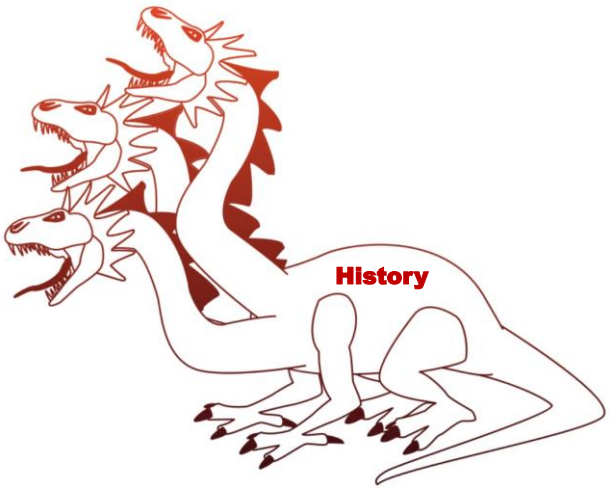
Appendix 3

History

- Chief complaint
- History of present illness
- Medical & Surgical history
- Family history
- Social history
- Review of systems



Past Medical & Surgical history



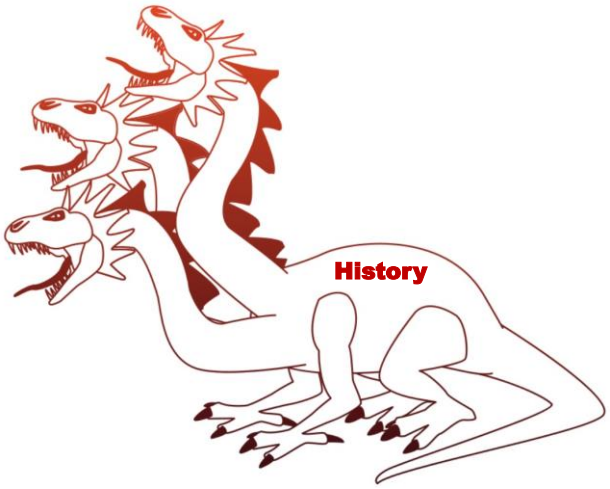
- The patient's past history
- Medical history
  - Diagnoses
  - Conditions
  - Hospitalizations
  - Medications & Vaccinations
  - Allergies
- Surgical history
  - Total abdominal hysterectomy
  - Appendectomy
  - Splenectomy

Appendix 3

Past Medical & Surgical history

- Childhood illness: measles at 4 y/o, chickenpox at 7 y/o both with no treatment or complications
- Adult illness:
  - HTN for 10 years treated with Lisinopril 10mg daily
  - DM type 2 for 3 years treated with metformin 1000mg BID for 3 years
    - Eye exam 2013 and annually no significant findings
    - Self foot exams weekly no changes
  - Hospitalization at St Francis for Chest pain 12/26/2011 with negative stress test and benign origin of pain (? GERD)
- Surgical History:
  - Tonsillectomy age 6
  - Appendectomy age 13
- OB/GYN: G3P3A0L3, menarche at age 11, LMP 4 years ago, sexually active with husband no history of STD, 2 total partners, no current contraception
- Health Maintenance:
  - Immunizations: tetanus 2011, shingles vaccine 2013, pneumonia vaccine 12/2011 with hospitalization
  - Pap smear: last 2012- negative, no history of abnormal
  - Colonoscopy: 2016- negative
- Medications
  - Lisinopril 10 mg daily
  - Metformin 1000mg bid
- Allergies – NKDA

Family history



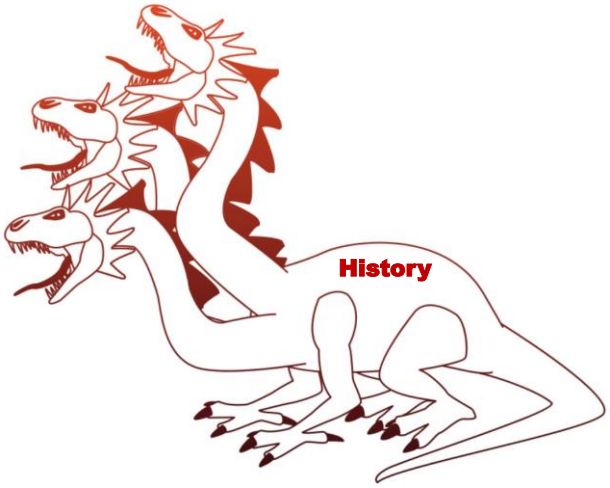
- Parents
- Siblings
- Grandparents OR children

Appendix 3

Family history

- Father died at age 43 from heart attack, had HTN, DM, high cholesterol
- Mother living age 73, with Lupus, HTN, chronic kidney disease, asthma since childhood, and seasonal allergies
- Grandfather deceased heart failure at 51
- Grandmother deceased colon cancer at 62
- Brother died at age 32 from heart attack, obese, alcoholic, smoked
- Son with polycystic kidney disease at age 19, HTN, migraines

Social history



Appendix 3

Social history

- Marital status:
  - Married x 30 years, divorced, 3rd marriage, single, same sex partner…
- Occupation:
  - Works as a welder and uses a respirator mask routinely, work stress is minimal, but works long hours including nights and weekends
- Substance abuse: Patients can be reluctant to divulge this and takes practice to elicit this many times
  - Drinks Vodka 2 pints daily for 6 years
  - Smokes 1 PPD for 20 years but quit 10 years ago
  - Smokes Methamphetamines daily for the last 7 years
  - IV heroin 3 x/wk for 10 years and shared needles
  - Caffeine (what and how much)
- Diet:
  - Poor, fair, good, eats routine fast foods, low salt, high fat, Adkins, low salt
- Physical activity:
  - Exercises 3x/wk for 1 hour each treadmill, none, rare basketball
- Functional status:
  - Ambulates with cane only, can perform most of her ADL's, has difficulty with transfers from bed to standing or to wheel chair.
- Safety:
  - Wears seat belts, helmets, no firearms in the house, reports no abuse at home (domestic or other, sexual, verbal, or physical)
- Exposures:
  - Recent travel to Australia 3 weeks ago for business, has 2 dogs/ 3 cats/ 1 guinea pig, goes to day care or has 2 kids in day care, works at a virology lab, exposure to second hand smoke.
- Religion:
  - Jehovah's witness and uses no blood products, Catholic and would like the priest to come by
- Hobbies:
  - Paints routinely, sports, needle work
- Stress/psychosocial
  - Home, family, work, spouse, other…
- Living situation:
  - Lives with parents, uncle, grandparent. Lives in an apartment with no heat
- Level of education:
  - Finished 3rd grade, Doctorate degree in Medicine at OU Tulsa
- Sexual history (if not addressed in past medical history)
  - 8 sexual partners prior
  - No routine condom use
  - No birth control
  - Last sexual encounter 1 week ago (include when pertinent)

History

- Chief complaint
- History of present illness
- Medical & Surgical history
- Family history
- Social history
- Review of systems
  - Anything we are missing?

Appendix 3

ROS

- *Urinary*
  - Frequency; polyuria; hesitancy; nocturia; urgency; dysuria; hematuria; incontinence; Δ in urine stream; kidney/flank pain
    - *MALES*: Δ in urinary stream; hesitancy; dribbling; prostate problems
- *G/U*
- *(General)*
  - Sexual habits; interest; function; satisfaction; use of birth control methods; HIV exposure
- *Male G/U*
  - Discharge from or sores on penis; testicular pain/masses
- *Female G/U*
  - Menarche; frequency/duration of menses; dysmenorrhea; PMS symptoms: bleeding between menses or after intercourse; LMP
  - Vaginal discharge; itching; sores; lumps; menopause; hot flashes; post-menopausal bleeding
- *Peripheral Vascular*
  - Claudication; leg cramps; varicose veins; hx of blood clots; swelling of the calves, legs, feet, or hands; Δ in color of fingers/toes with cold weather; problems with cold hands/feet; cold, numbness, pallor in the legs or loss of hair
- *Musculo-skeletal*
  - Muscle or joint pain; joint stiffness; problem opening/closing mouth; muscle weakness or spasm; limitation in joint mobility; redness, swelling, tenderness of a joint; scoliosis; popping/grinding of a joint when moved; broken/injured bones/joints; surgeries
- *Neuro*
  - Syncope; dizziness; blackouts; seizures; weakness; paralysis; numbness/tingling; tremors; involuntary movements; Δ speech, memory, mood, or thinking;
- *Heme*
  - Hx of anemia; easy bruising or bleeding; blood transfusions
- *Endo*
  - Heat or cold intolerance; excessive sweating; polydipsia; polyphagia; polyuria; thyroid problems or diabetes; excessive thirst
- *Psych*
  - Nervousness/anxiety; depression; memory changes; homicidal/suicide thoughts, ideas, or attempts; Δ in sleep or stress

ROS

- *General*
  - Wgt Δ; weakness; fatigue; fevers
- *Skin*
  - Skin Δ's; rashes; bumps/lumps; sores; itching; dryness; color change; Δ in hair/nails; mole changes in shape, size, color
- *Head*
  - Headache; head injury; trauma
- *Eyes*
  - Vision Δ; corrective lenses; last eye exam; pain; redness; excessive tearing; double vision; blurred vision; scotoma; dryness of the eyes
- *Ears*
  - Hearing loss or Δ; tinnitus; earaches; infections; discharge
- *Nose/Sinuses*
  - Frequent colds; congestion; runny nose; discharge; itching; hay fever; nosebleeds; Δ in sense of smell
- *Mouth/Throat*
  - Dentures; bleeding gums/mouth/teeth; last dental exam; sore tongue; dry mouth; sore throats; hoarse; problems swallowing; Δ in sense of taste
- *Neck*
  - Lumps; swollen glands; goiter; pain; neck stiffness
- *Breasts*
  - Lumps; pain; discomfort; nipple discharge; rashes; asymmetry
- *Pulmonary*
  - Cough—productive/non-productive; hemoptysis; dyspnea/shortness of breath; wheezing; chest pain; pleuritic pains; frequent lung infections, night sweats
- *Cardiac*
  - Chest pain or discomfort; shortness of breath; palpitations; dyspnea; orthopnea; PND; peripheral edema or swelling; exertional chest pain or SOB; cardiovascular testing;
- *G/I*
  - Appetite/diet Δ; jaundice, liver/gallbladder/hepatitis problems; nausea/emesis; dysphagia; heartburn/indigestion; pain; hematemesis; excessive belching/flatulence; Δ in bowel habits-color, size, consistency; hematochezia; melena; hemorrhoids; constipation; diarrhea; food intolerance; pain with defecation

Appendix 3

History

- Chief complaint
- History of present illness
- Medical & Surgical history
- Family history
- Social history
- Review of systems
  - Anything we are missing?

History: Review of Systems

- Chief complaint
- History of present illness
- Medical & Surgical history
- Family history
- Social history
- Review of systems

GI

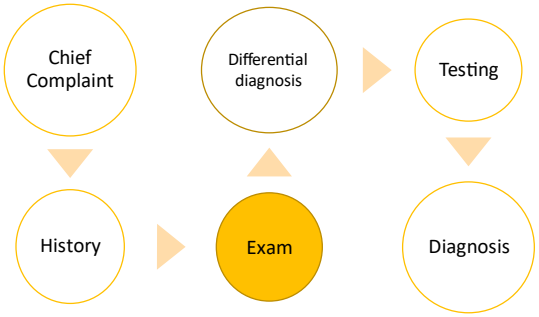
Unexplained weight loss, change in stool caliber, red blood in stool

CV

Chest pain with exertion, dyspnea on exertion, feeling dizzy/passing out

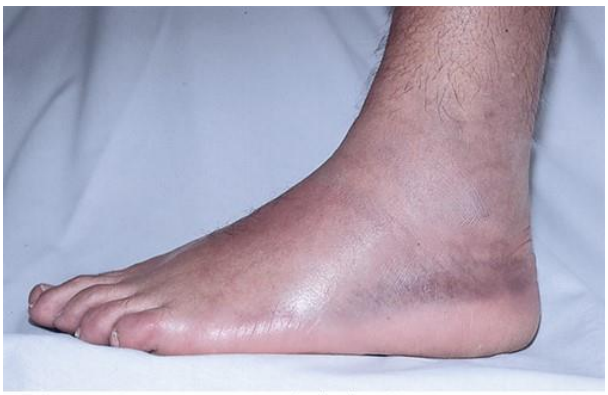
Appendix 3

Physical Examination

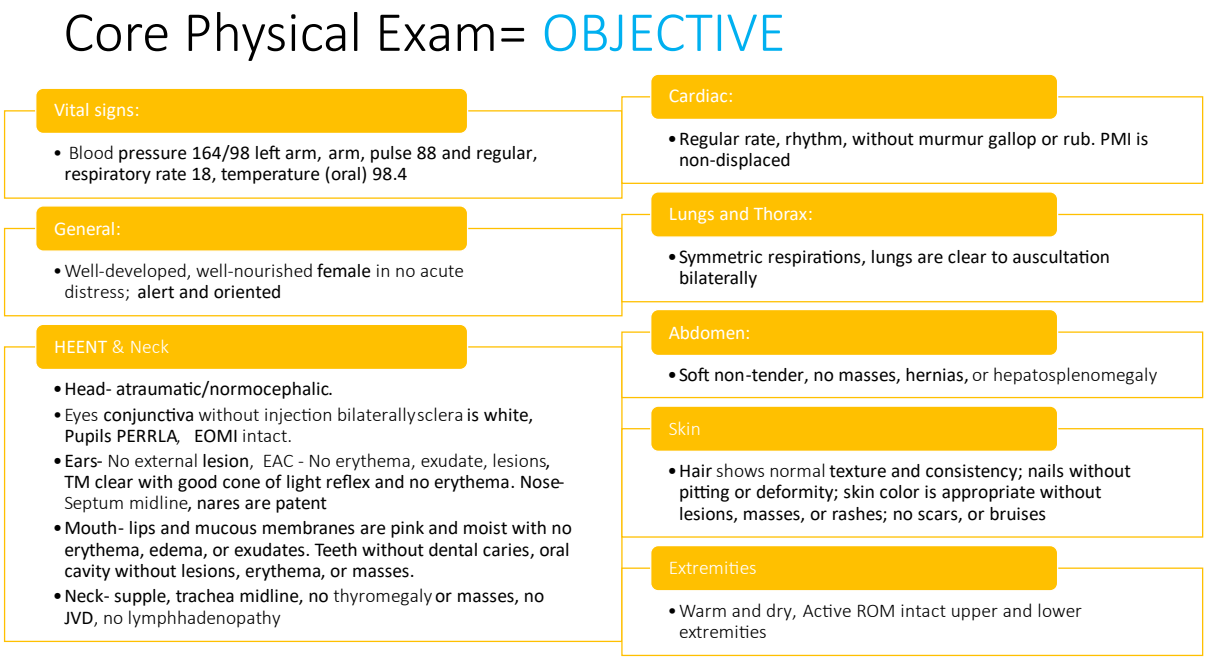


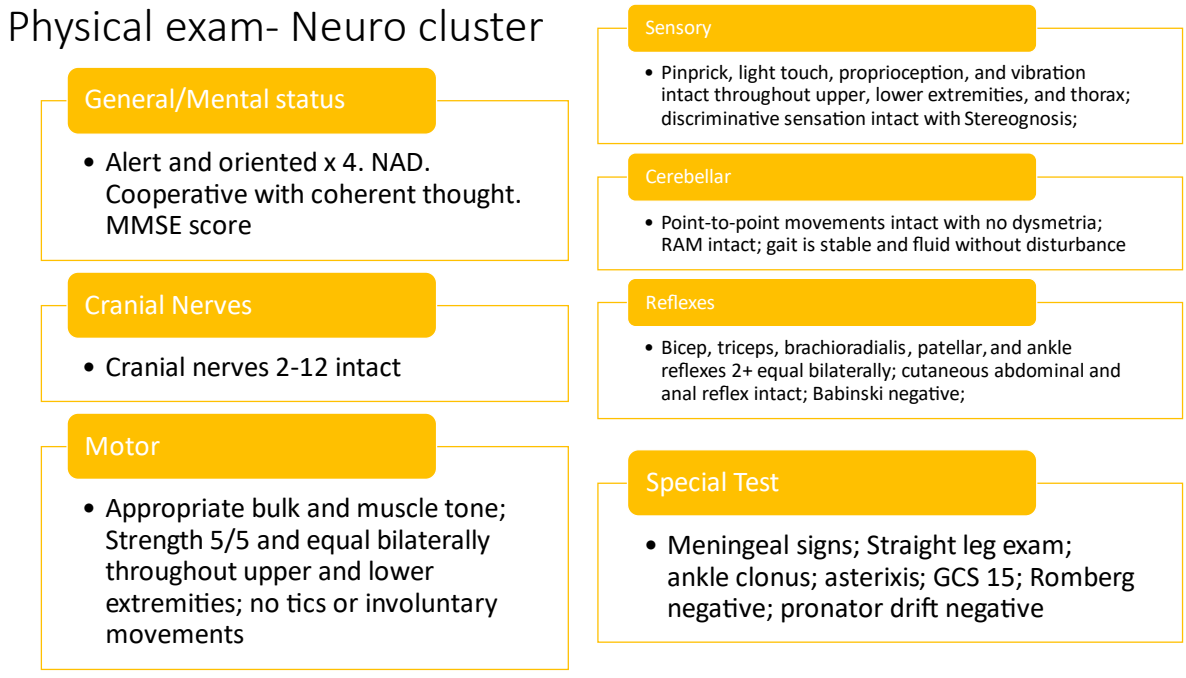
Physical Examination

- Objective
- Core
  - Vitals
  - General
  - Skin
  - Heart and Lungs
- Clusters
  - MSK
    - Ankle, foot and leg
      - Appearance
      - Tenderness to palpation
      - ROM
  - Neurovascular

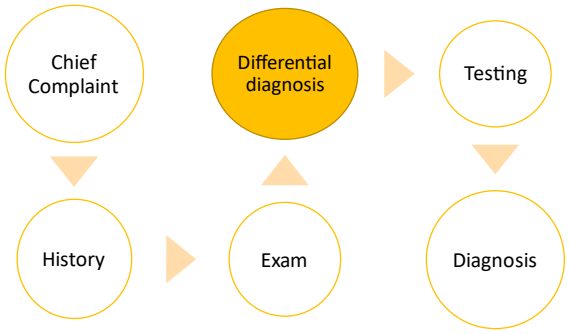


Appendix 3


Core Physical Exam= OBJECTIVE
Vital signs:
• Blood pressure 164/98 left arm, arm, pulse 88 and regular, respiratory rate 18, temperature (oral) 98.4
General:
• Well-developed, well-nourished female in no acute distress; alert and oriented
HEENT & Neck
• Head- atraumatic/normocephalic.
• Eyes conjunctiva without injection bilaterallysclera is white, Pupils PERRLA, EOMI intact.
• Ears- No external lesion, EAC - No erythema, exudate, lesions, TM clear with good cone of light reflex and no erythema. Nose- Septum midline, nares are patent
• Mouth- lips and mucous membranes are pink and moist with no erythema, edema, or exudates. Teeth without dental caries, oral cavity without lesions, erythema, or masses.
• Neck- supple, trachea midline, no thyromegaly or masses, no JVD, no lymphhadenopathy
Cardiac:
• Regular rate, rhythm, without murmur gallop or rub. PMI is non-displaced
Lungs and Thorax:
• Symmetric respirations, lungs are clear to auscultation bilaterally
Abdomen:
• Soft non-tender, no masses, hernias, or hepatosplenomegaly
Skin
• Hair shows normal texture and consistency; nails without pitting or deformity; skin color is appropriate without lesions, masses, or rashes; no scars, or bruises
Extremities
• Warm and dry, Active ROM intact upper and lower extremities


Physical exam- Neuro cluster
General/Mental status
• Alert and oriented x 4. NAD. Cooperative with coherent thought. MMSE score
Cranial Nerves
• Cranial nerves 2-12 intact
Motor
• Appropriate bulk and muscle tone; Strength 5/5 and equal bilaterally throughout upper and lower extremities; no tics or involuntary movements
Sensory
• Pinprick, light touch, proprioception, and vibration intact throughout upper, lower extremities, and thorax; discriminative sensation intact with Stereognosis;
Cerebellar
• Point-to-point movements intact with no dysmetria; RAM intact; gait is stable and fluid without disturbance
Reflexes
• Bicep, triceps, brachioradialis, patellar, and ankle reflexes 2+ equal bilaterally; cutaneous abdominal and anal reflex intact; Babinski negative;
Special Test
• Meningeal signs; Straight leg exam; ankle clonus; asterixis; GCS 15; Romberg negative; pronator drift negative

Differential diagnosis



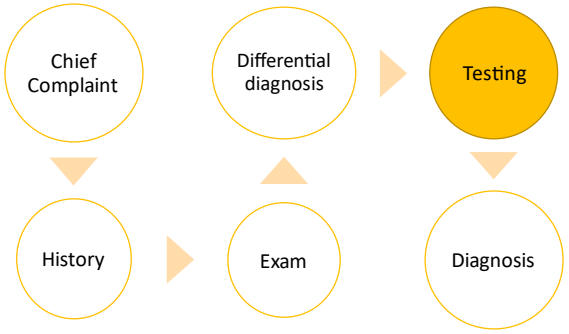
- Hypotheses
- List of possible causes
- Grows out of History and Physical exam
- Probabilistic
- Prognostic
- Pragmatic

Differential diagnosis

- Probabilistic - Common things are common = URI
- Prognostic – Can't miss diagnoses = Pneumonia, cancer
- Pragmatic – Diagnosis of exclusion, response to treatment

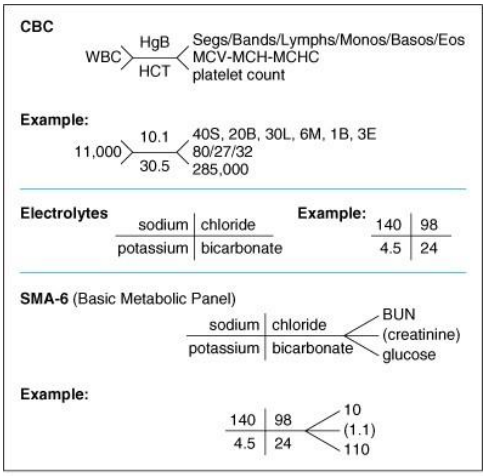
Appendix 3

Testing: Diagnostic Evaluations



Testing: Diagnostic Evaluations

- Lab data,
- X-rays, CT, MRI
- EKG's or other results
- Format of lab data



Appendix 3

Documenting the process

Documentation

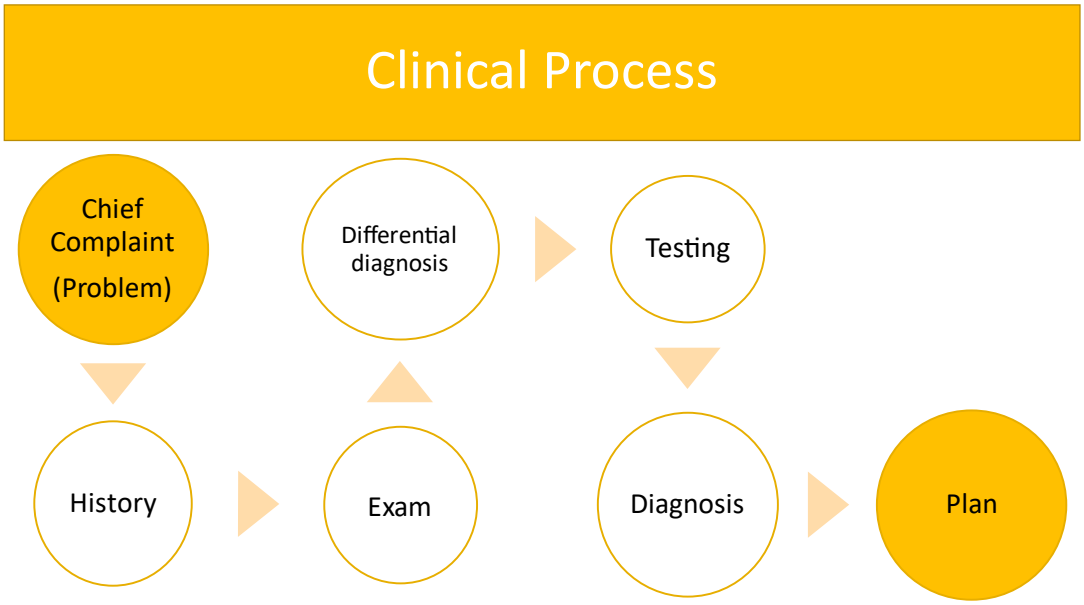
- The first rule of documentation: if it isn’t documented it didn't happen.
- The 2nd rule of documentation: if it isn’t documented it didn't happen.

Documenting the process

SOAP NOTE

- Subjective
  - What the patient (others) (chart) tells you
- Objective
  - What you can see, observe or measure
- Assessment
  - Diagnosis – working or final
- Plan
  - Treatment or further evaluation

Appendix 3



"Whip Chief Socrate's 3-Headed History with Rose Pe*d*als"

Appendix 3

Clinical Process and Developing a Relationship

- Wash hands
- Introduce (identify) {inquire}
- Provider (patient) {permission}

Clinical Process and Developing a Relationship

- Wash hands
- Introduce (identify) {inquire}
- Provider (patient - {permission}

Appendix 3

Clinical Process and Developing a Relationship

- Wash hands
- Introduce (identify) {inquire}
- Provider (patient - people) {permission}

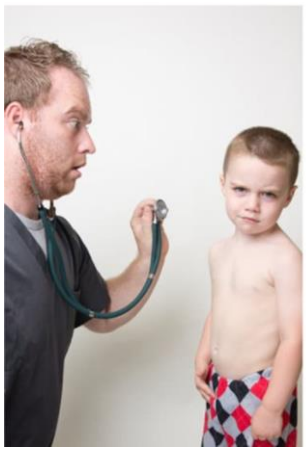
Clinical Process and Developing a Relationship

- Wash hands
- Introduce (identify) {inquire}
- Provider (patient) {permission}

- Playful

Appendix 3

Clinical Process and Developing a Relationship



- Wash hands
- Introduce (identify) {inquire}
- Provider (patient) {permission}

- Playful or not

Clinical Process and Developing a Relationship

- Listen
- Subtle verbal and nonverbal

Appendix 3

"Whip Chief Socrate's 3-Headed History with Rose Pedals"

- Wash hands
- Introduce (identify) {inquire}
- Provider (patient) {permission}

"Whip Chief Socrate's 3-Headed History with Rose Pedals"

- Chief Complaint
  - Clarified

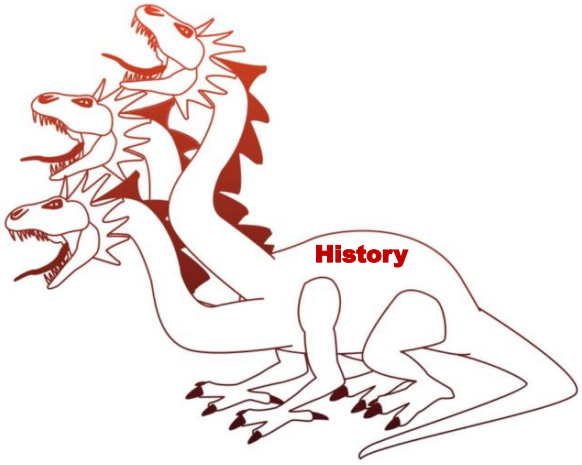
Appendix 3

"Whip Chief Socrate's 3-Headed History with Rose Pedals"

- History of present illness

- **S**ite
- **O**nset
- **C**haracter
- **R**adiation
- **A**ssociated signs and symptoms
- **T**iming
- **E**xacerbating, alleviating factors
- **S**everity

"Whip Chief Socrate's 3-Headed History with Rose Pedals"



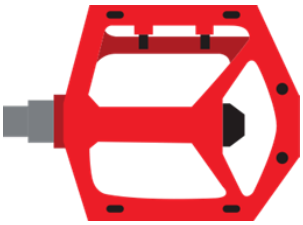
- Past Medical & Surgical history
- Family history
- Social history

Appendix 3

“Whip Chief Socrate’s 3-Headed History with Rose Pedals”

- Rose
- Review of Systems

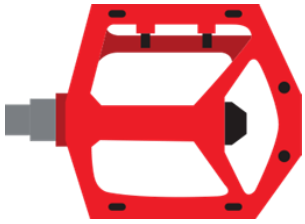
Whip Chief Socrate’s 3-Headed History with Rose Pedals



- **P**hysical **e**xamination

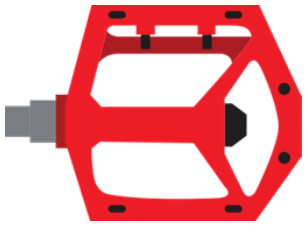
Appendix 3

Whip Chief Socrate's 3-Headed History with Rose Pedals



- **Differential Diagnosis**

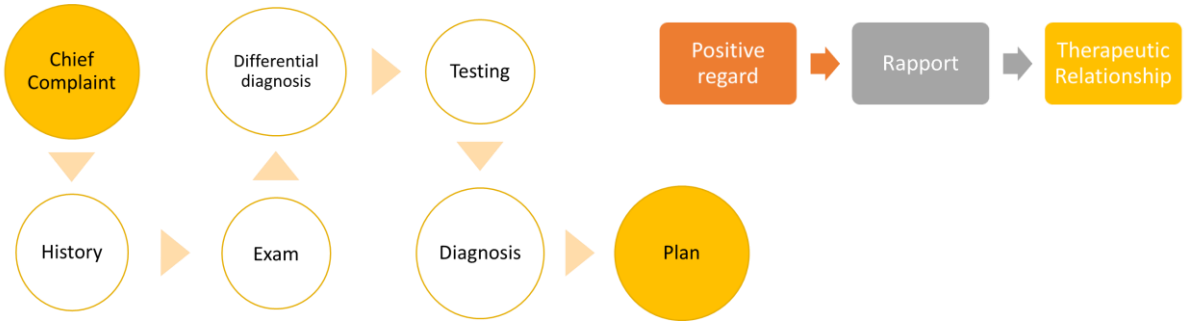
Whip Chief Socrate's 3-Headed History with Rose Pedals



- **A list of stuff**
- **Assessment**
  - Working or clinical diagnosis
- **Plan**
  - Treatments
  - Testing

Appendix 4

Practice of Medicine



Sore throat

- 29 y/o female c/o sore throat

Appendix 4

SOAP: Subjective

- **CC**: Throat pain
- **HPI**: 29-year-old female complains of "sore throat" for two days. Patient describes the throat pain as "it feels like I'm swallowing razor blades." The throat pain has been associated with fever up to 101.4 F (oral) but the patient denies any rash, neck pain, neck stiffness, cough, post-nasal drainage, reflux sxs, headache or shortness of breath. The patient notes that it is worse with swallowing and improves moderately with ibuprofen and saltwater gargles. The patient reports she had similar symptoms six months ago and was diagnosed with "strep throat." The patient rates her pain and "6" out of 10 but states it worsens with swallowing.

SOAP: Subjective

- **PMHx** – Negative; NKDA, Meds - OCP
- **PSHx** – Appendectomy age 7
- **FMHx** – Father – alive and well with Hx of HTN, Mother and two siblings are alive and well without chronic disease.
- **SocHx** - Single, works as grade school teacher, denies use of alcohol, tobacco or illicit substances.

Appendix 4

SOAP: Subjective

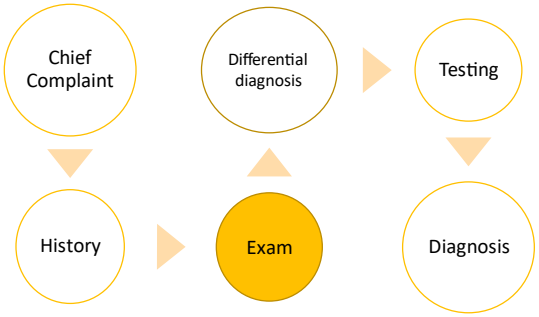
- **ROS**
- General – Denies Wgt Δ; weakness; fatigue; Notes fevers as above
- Skin – Denies Skin Δ's; rashes; bumps/lumps; sores; itching; dryness; color change; Δ in hair/nails;
- Head – Denies Headache; head injury; trauma
- Eyes – Denies Vision Δ; corrective lenses; last eye exam; pain; redness; excessive tearing; double vision; blurred vision; scotoma; dryness of the eyes
- Ears – Denies Hearing loss or Δ; tinnitus; earaches; infections; discharge
- Nose/Sinuses – Denies Frequent colds; congestion; runny nose; discharge; itching; hay fever; nosebleeds; Δ in sense of smell
- Mouth/Throat - sore throat as noted above ; Denies hoarse; problems swallowing; Δ in sense of taste
- Neck Lumps - Denies swollen glands; goiter; pain; neck stiffness
- Pulmonary - Denies Cough; hemoptysis; dyspnea/shortness of breath; wheezing; chest pain; pleuritic pains; frequent lung infections, night sweats

SOAP: Objective

- **PE**
- Vital signs – T 99.8 F Oral, Pulse-88, Respirations 14, Pulse Oximetry Equals 99% Room Air, blood pressure 124/70
- General – Well-developed well-nourished female in no acute distress
- Skin - No rashes or cutaneous lesions noted
- HEENT – Head-normocephalic, atraumatic; Eyes -conjunctivae clear , ears -tympanic membranes intact bilaterally, external auditory canals are clear, nose-nasal mucosa patent, moist; Throat - diffuse moderate erythema to the posterior oropharynx with minimal white exudate, tonsils surgically absent, no mass or apparent abscess, uvula midline
- Neck - supple, full range of motion, bilateral anterior cervical adenopathy less than 1 cm
- Heart - regular rate and rhythm without murmur rubber gallop
- Lungs – clear to auscultation bilaterally with good air movement
- Abdomen - soft, nontender, no hepatic splenomegaly

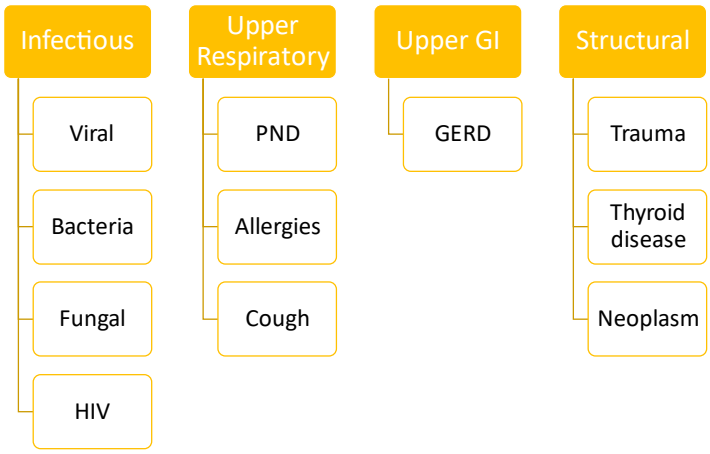
Appendix 4

Physical Examination

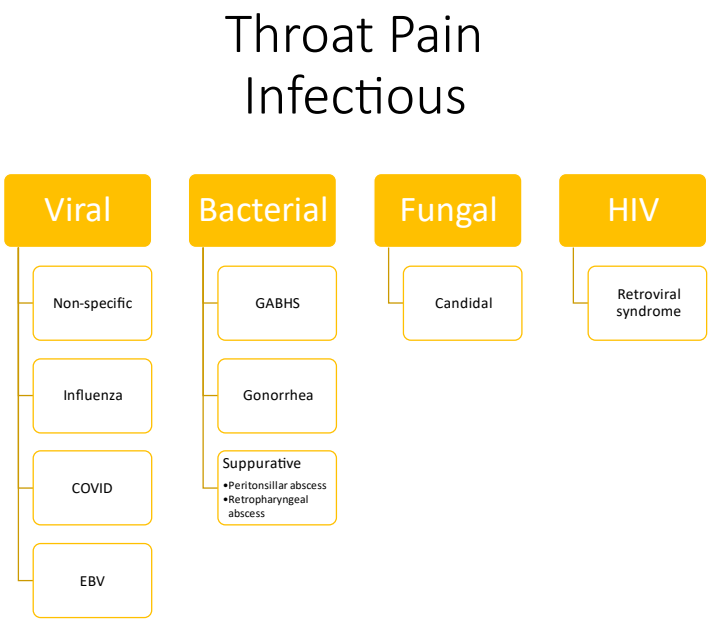


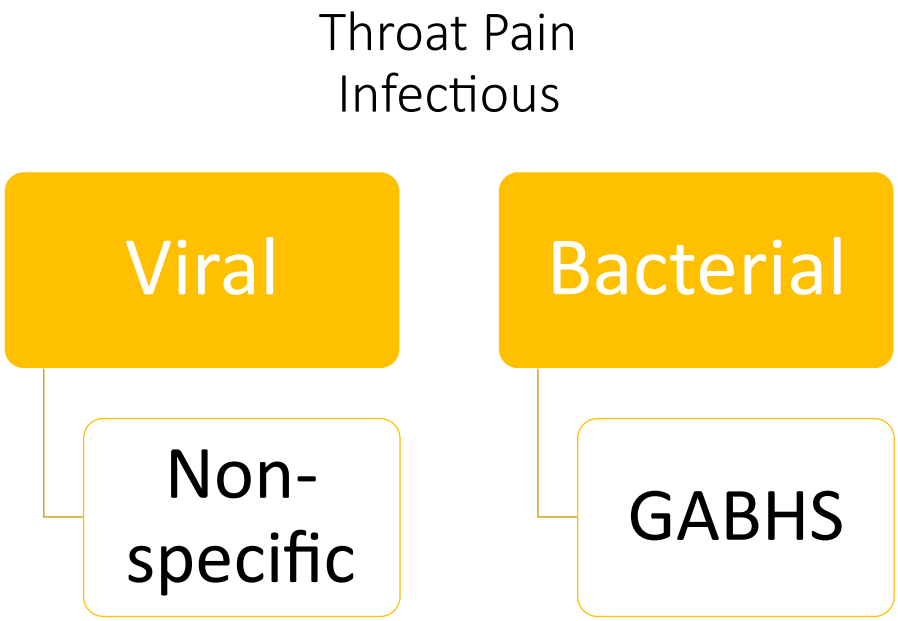
- Sore throat
  - Vital signs
  - General
  - Skin
  - HEENT
  - Neck
  - Heart
  - Lungs
  - Abdomen

Throat Pain



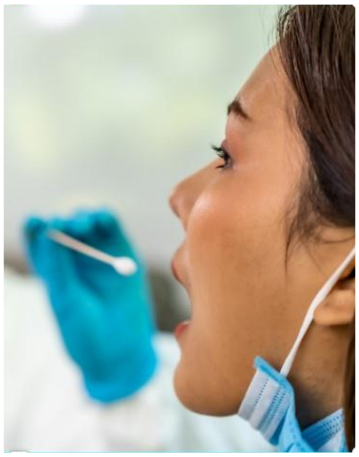
Appendix 4


Throat Pain
Infectious
Viral
Non-specific
Influenza
COVID
EBV
Bacterial
GABHS
Gonorrhea
Suppurative
•Peritonsillar abscess
•Retropharyngeal abscess
Fungal
Candidal
HIV
Retroviral syndrome


Throat Pain
Infectious
Viral
Non-specific
Bacterial
GABHS

Appendix 4

Testing: Diagnostic Evaluations



- Strep Pharyngitis?
  - Rapid strep
    - Sensitivity 70%
    - Specificity 97%
  - Throat culture
    - Sensitivity 98%
    - Specificity 100%

Throat Pain
Infectious

Viral

- Viral pharyngitis
- ***Diagnosis of exclusion***

Non-specific

Appendix 4

SOAP: Assessment & Plan

- **Assessment:** Pharyngitis, rule out streptococcal pharyngitis
- **Plan:**
- 1. Throat culture 2. Saltwater gargles and ibuprofen for discomfort 3. Call or return to clinic for any rapid worsening of symptoms or attend emergency room 4. Information on pharyngitis given to patient.
- Addendum: throat culture returned positive for Streptococcus Pyogenes, contacted patient and prescribed Penicillin VK 500 mg one PO twice daily for 10 days (patient verifies no known drug allergies), prescription called in to patient's pharmacy.

SOAP: Assessment & Plan

Diagnosis

- 1. YOUR ACTUAL diagnosis not the ddx
- 2. Must list diagnoses in order of importance

Plan

- 1. Use shared decision making with provider and the patient
- 2. Document patient education
- 3. Acknowledge patient's support or agreement with plan of care

Appendix 4

Clinical Process and Developing a Relationship

Appendix 5

Neuroanatomy overview

Perdue

Objectives

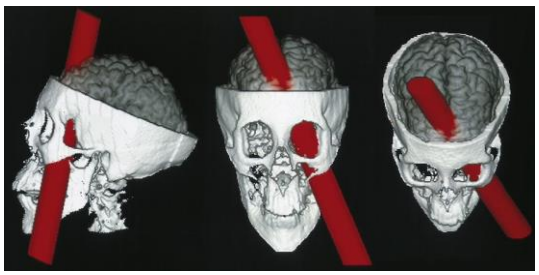
1. Describe the function of the central nervous system and its subcomponents
   A. Overview of Neuroanatomy - messenger system
   B. Neurohistology - cells that make the pathways
   C. Parts of the brain and what they do
   D. Somatic
   E. Autonomic
2. Apply the anatomical planes of orientation as you look at the CNS specimens and cross-sectional imaging studies.
3. Describe the major surface markings of the forebrain and brainstem.
4. Identify major sulci and gyri on the surface of the brain.
5. Identify the five cerebral lobes and define their boundaries in general terms.
6. Describe the location/organization of the gray matter and the white matter in the CNS in general terms.

Appendix 5

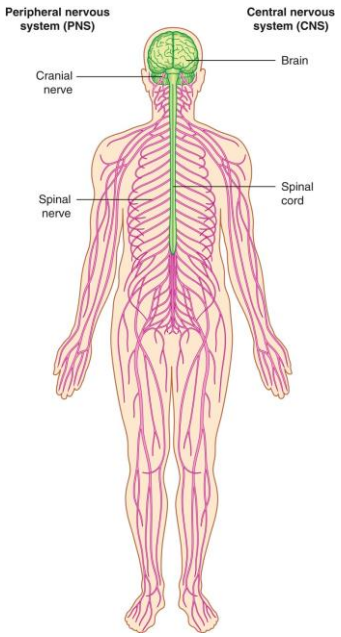
Functions of the Nervous system

- **Perception** - Translation of the outer world into electrochemical signals that can be interpreted by the brain
- **Action** – Brain coordinates organism in interacting with the environment by moving the body and vocalizations
- **Cognition** - Interpreting perceptual input to understand the environment and plan to respond to the environment through action

"No longer Gage"



Appendix 5

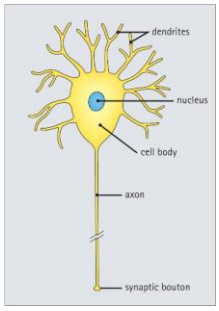


Structures of the nervous system

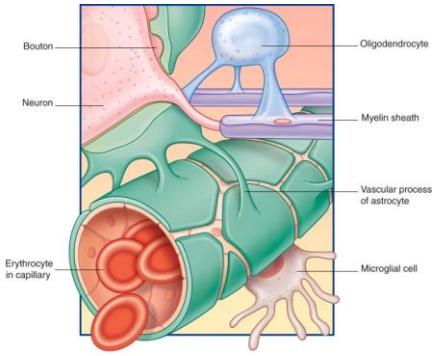
- CNS
  - Cortical brain
  - Subcortical brain
  - Brainstem
  - Spinal cord
- PNS
  - Nerve root
  - Nerve
  - NMJ
  - Muscle

Neurohistology

Neurons

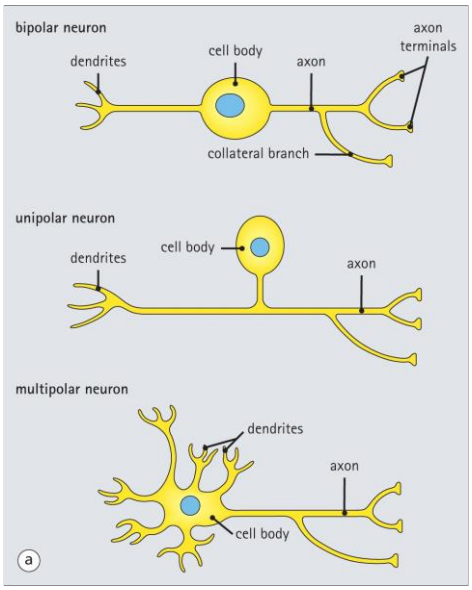


Neuroglia



Appendix 5

Neuron



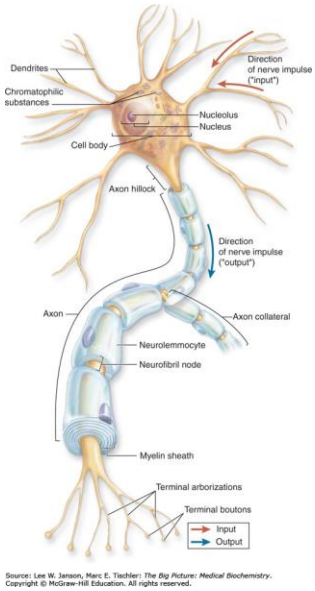
- Dendrites
- Cell body
- Axon
- Excitability

Neuron

- Action potential

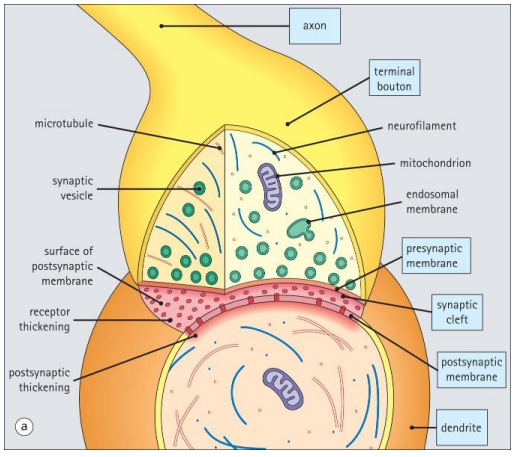
Appendix 5

Neuron



- Action potential
- Impulses received at the cell body or dendrites meet a certain threshold
- Electrochemical process initiated in the axon hillock

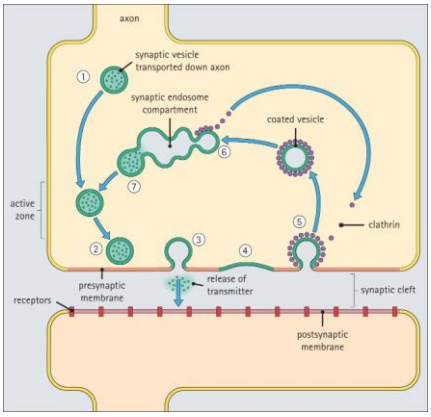
Synaptic communication



- **Synapses** - sites where nerve impulses are transmitted from one nerve to another.
- Presynaptic axon terminal (terminal bouton) contains mitochondria and numerous synaptic vesicles from which neurotransmitter is released by exocytosis
- Postsynaptic cell membrane-contains receptors for the neurotransmitter and ion channels or other mechanisms to initiating impulse
- Synaptic cleft-20 - 30 nm wide intracellular space separates these presynaptic and postsynaptic membranes

Appendix 5

Synaptic communication



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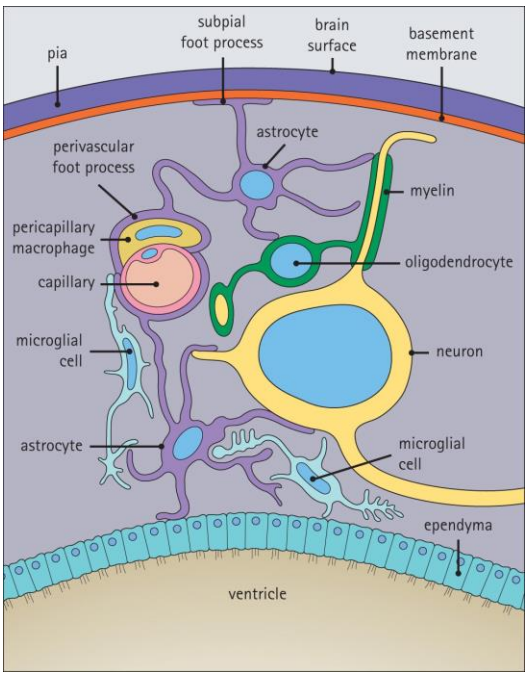
Neurotransmitter	Areas of Concentration
Acetylcholine (ACh)	Neuromuscular junction, autonomic ganglia, parasympathetic neurons, motor nuclei of cranial nerves, caudate nucleus and putamen, portions of the limbic system
Norepinephrine (NE)	Sympathetic nervous system, locus ceruleus, lateral tegmentum
Dopamine (DA)	Hypothalamus, midbrain nigrostriatal system
Serotonin (5-HT)	Parasympathetic neurons in gut, pineal gland, nucleus raphe magnus of pons
Gamma-aminobutyric acid (GABA)	Cerebellum, hippocampus, cerebral cortex, striatonigral system
Glycine	Spinal cord
Glutamic acid	Spinal cord, brain stem, cerebellum, hippocampus, cerebral cortex

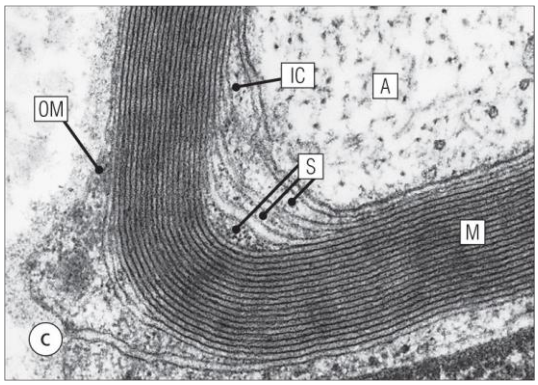
Appendix 5

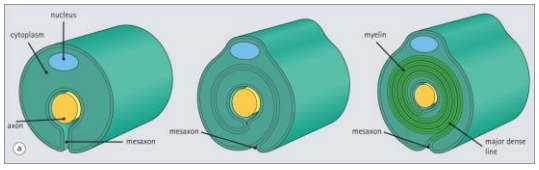


Neuroglia

- **Oligodendrocytes** wrap processes around portions of axons in the **CNS**, forming myelin sheaths that insulate the axons and facilitate nerve impulses.
- **Astrocytes** - the most numerous cell of the CNS, all produce hundreds of processes to cover and provide regulated microenvironments for neuronal perikarya, synapses, and capillaries.
- **Ependymal cells** are epithelial-like cells, lacking basement membranes, which line the fluid-filled cerebral ventricles and central canal of the spinal cord.
- **Microglia** - they mediate immune defense activity within the CNS.
- **Satellite cells** are located within PNS ganglia, aggregated sensory or autonomic neuronal cell bodies, where they enclose each perikaryon and regulate its microenvironment.
- **Schwann cells** (neurolemocytes) enclose all axons in nerves of the **PNS,** producing myelin sheaths around large-diameter axons, whose impulse conductivity is augmented at the nodes of Ranvier between successive Schwann cells.

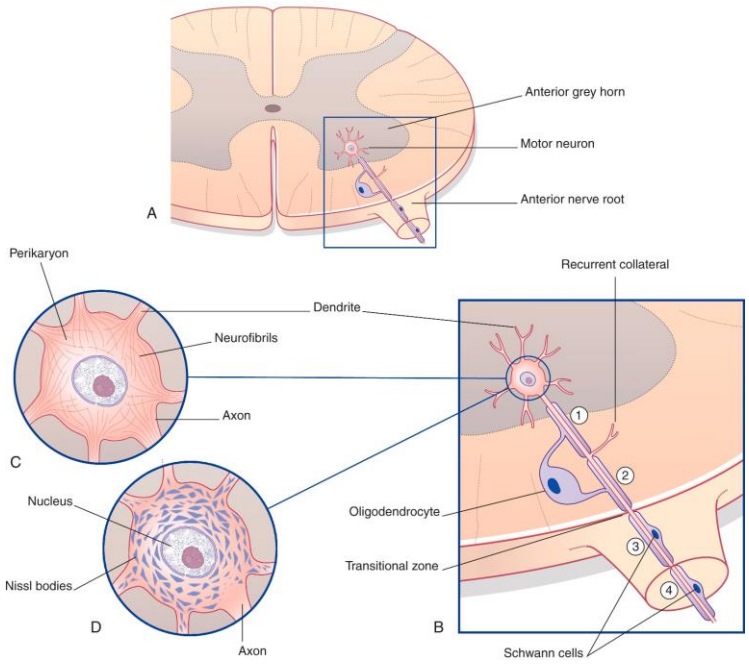
Myelin

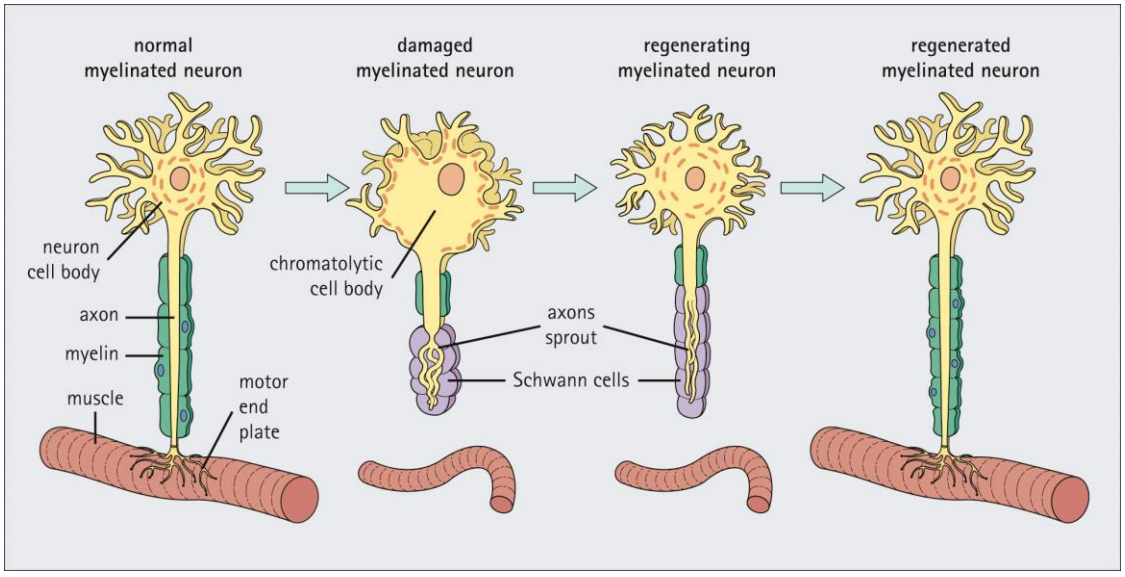




Appendix 5

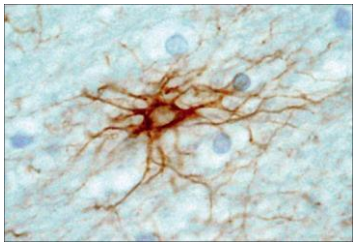
Neuron

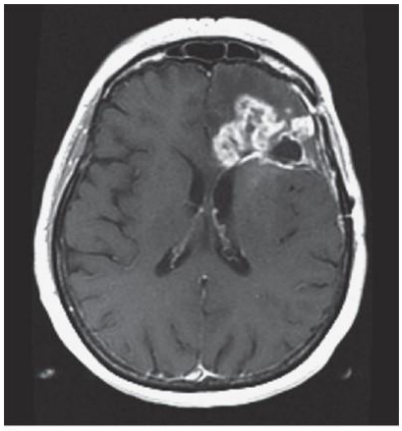




Astrocytoma

- GRADE IV ASTROCYTOMA (GLIOBLASTOMA)





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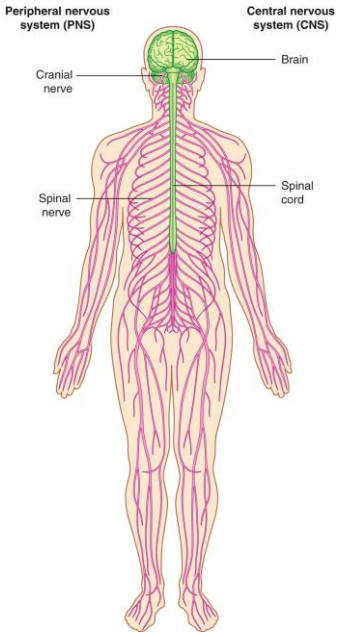
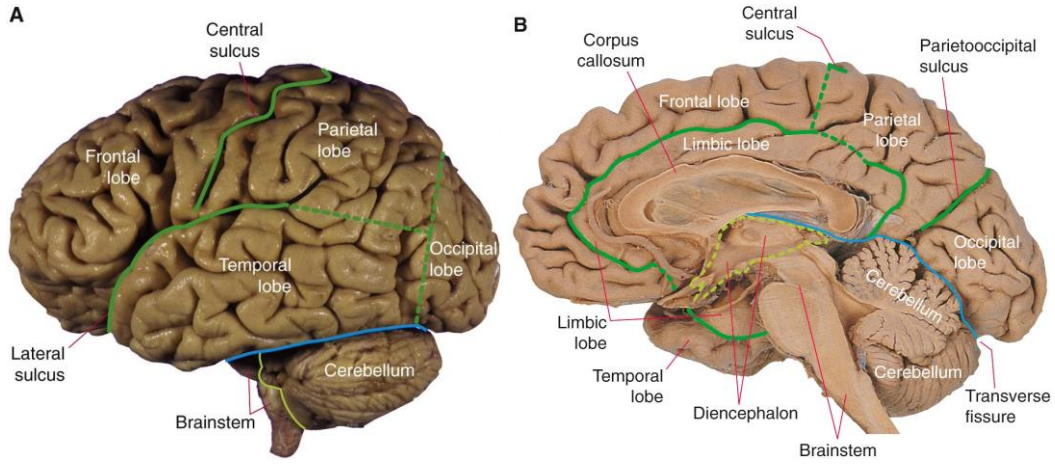


Structures of the nervous system

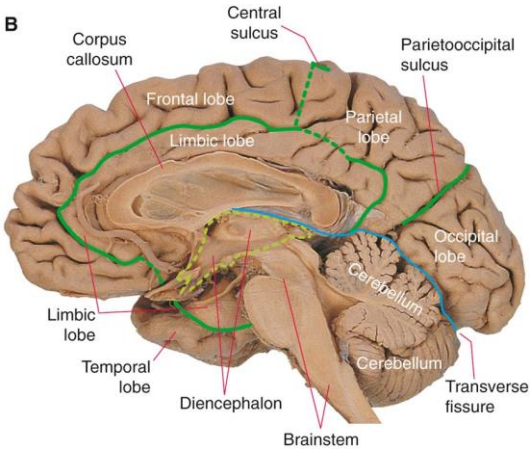
- CNS
  - Cortical brain
  - Subcortical brain
  - Brainstem
  - Spinal cord
- PNS
  - Nerve root
  - Nerve
  - Muscle

Appendix 5

Brain



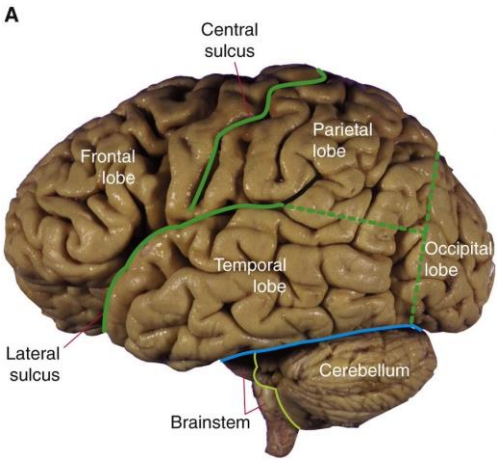
Brain



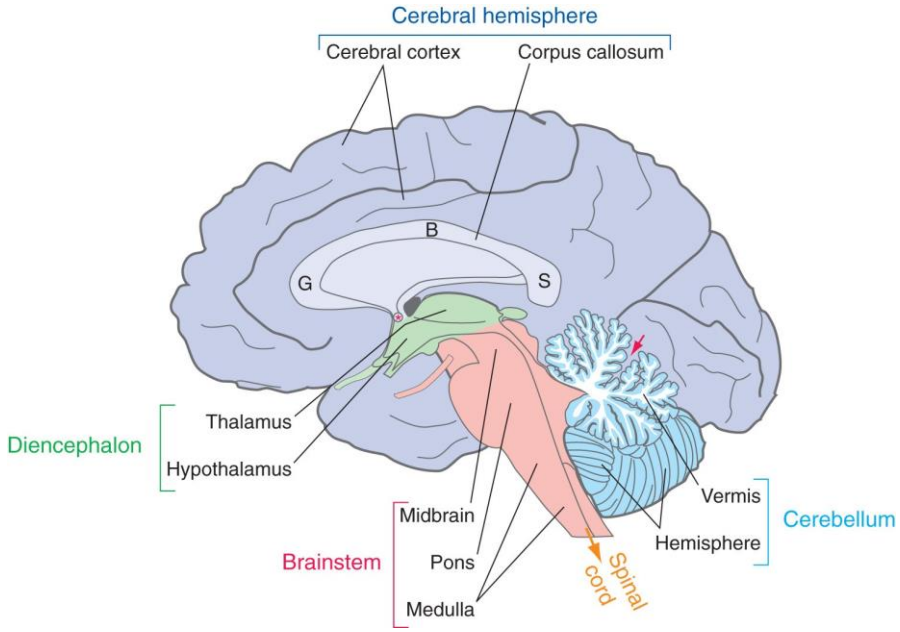
- Maximize surface area
- Gyri - folds
- Sulci – spaces between
- Fissures -Large divisions between the hemispheres or lobes

Appendix 5

Brain

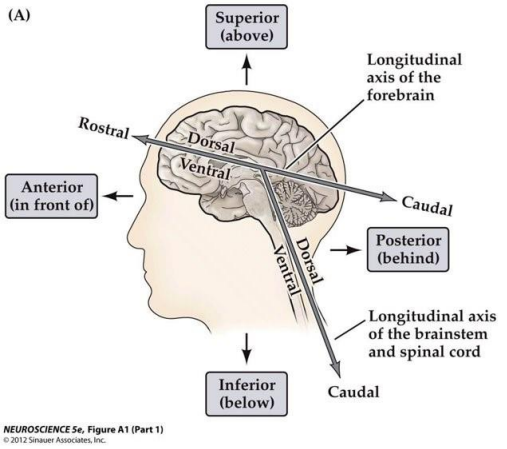


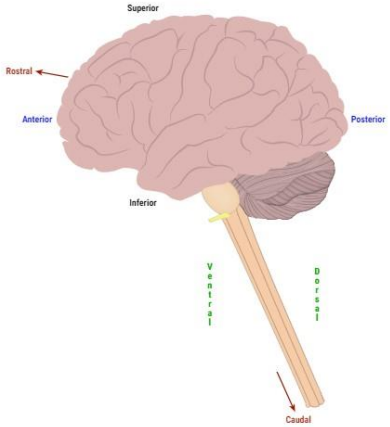
- Two Hemispheres
  - Left & Right
- Four Lobes
  - Frontal
  - Temporal
  - Parietal
  - Occipital



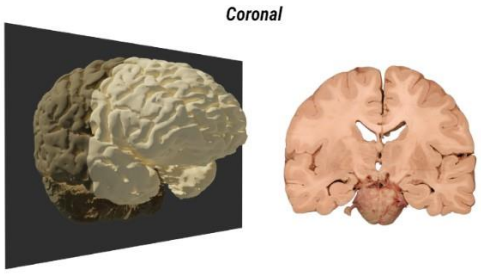
Appendix 5

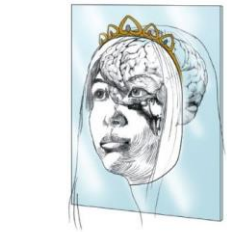
Axial orientation





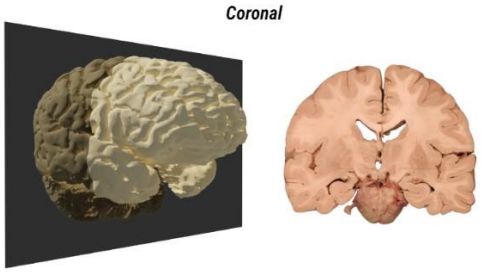
Coronal section

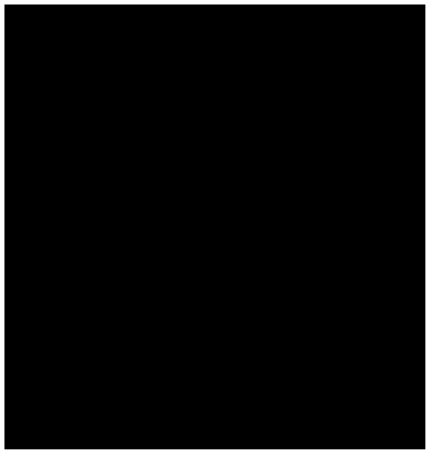




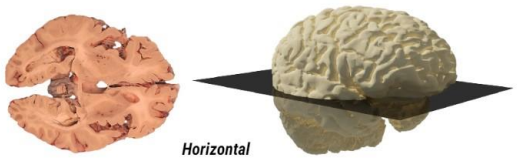
Appendix 5

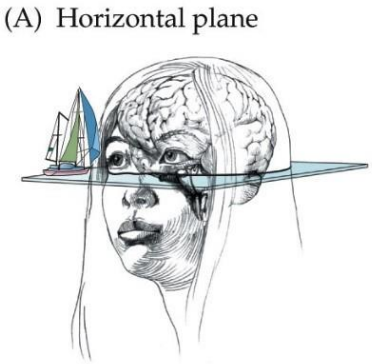
Coronal section





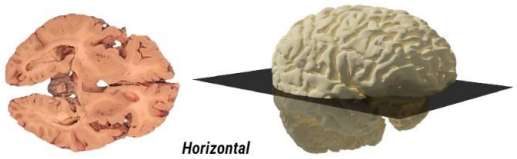
Horizontal section

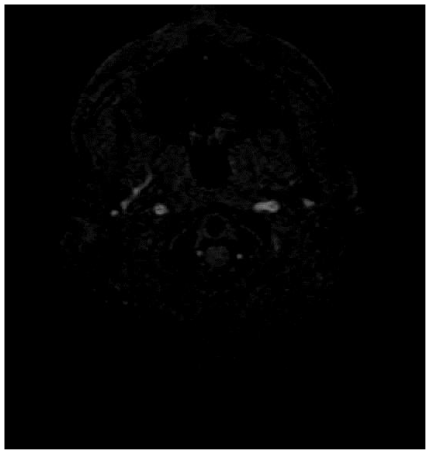




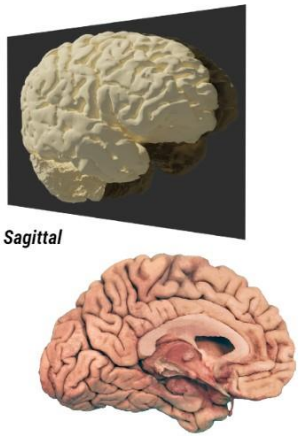
Appendix 5

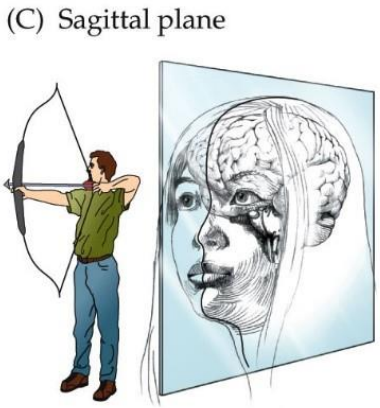
Horizontal section





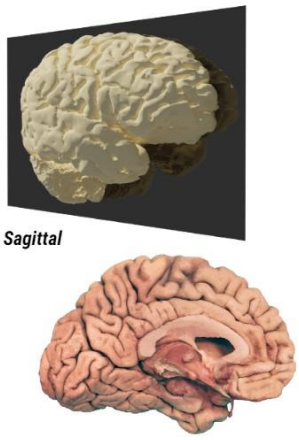
Sagittal section

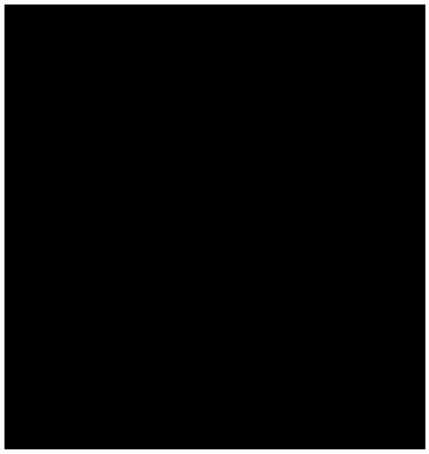




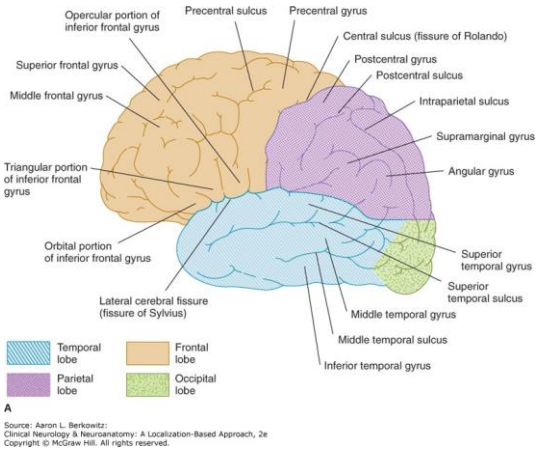
Appendix 5

Sagittal section





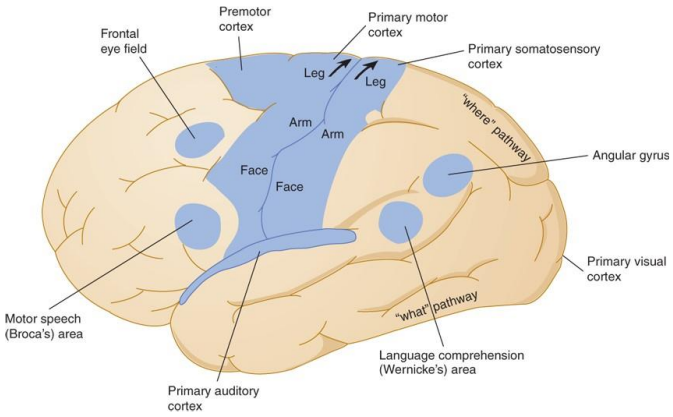
Brain

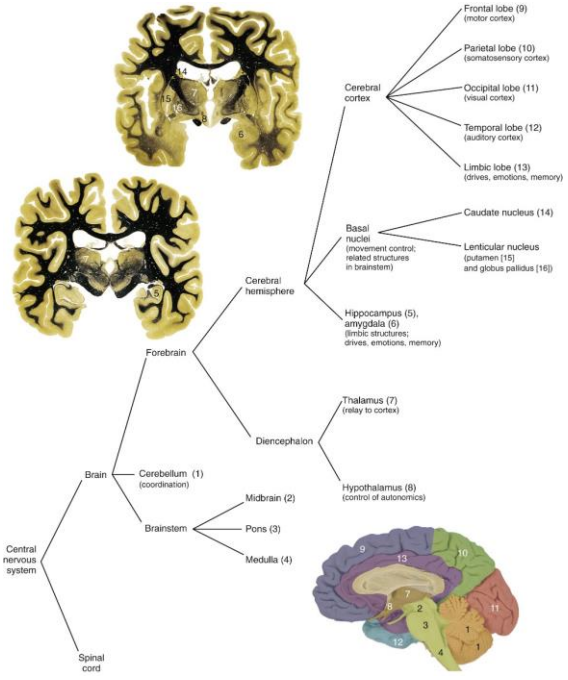


- Frontal lobe – Precentral gyrus and forward
- Parietal lobe – Post central gyrus back to parietooccipital sulcus
- Occipital lobe - parietooccipital sulcus and back (occiput)
- Temporal lobe – inferior to other lobes, lateral cerebral fissure

Appendix 5

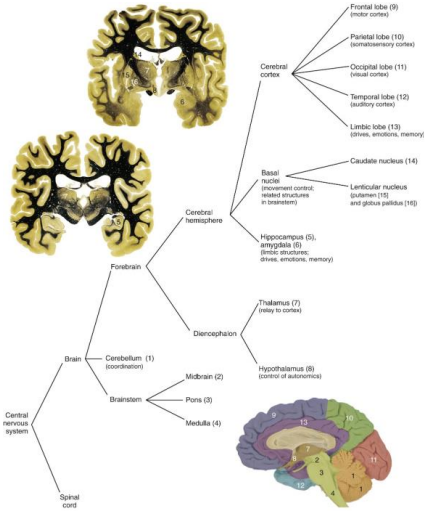
Cognition: Interpreting environment & planning to act





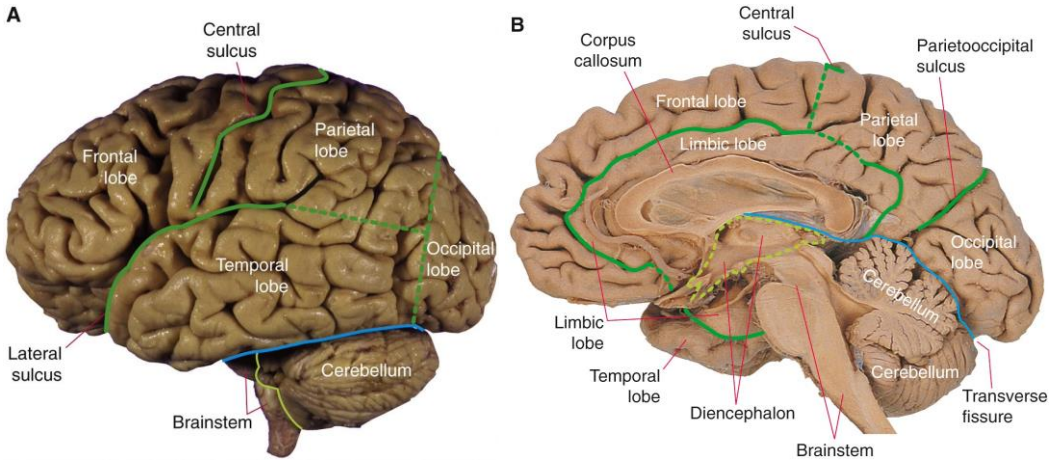
Appendix 5

Brain and function – highly simplified



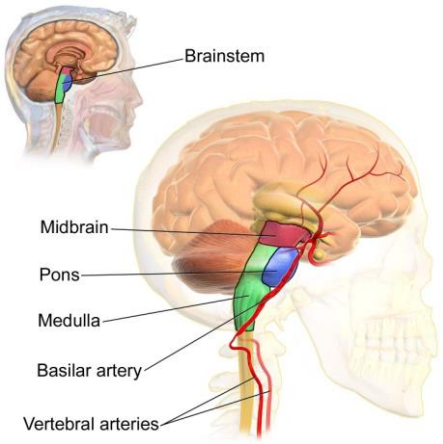
a. Somatic: Pre/Post central gyrus (and other areas) for movement and sensation
b. Autonomic: Hypothalamus and brainstem
c. Higher (and lower) functioning
    a. Higher – Language processing and executive functioning, morality, identity (Diffuse cerebral cortex)
    b. Lower – Shared with animals, limbic system: Feeding (hunger and thirst), Fighting/Defense (fear), "Fornication" reproduction (sexual desire).

Brain



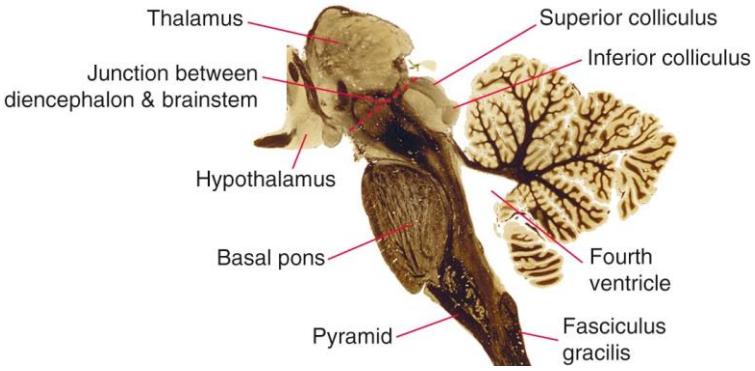
Brainstem

- **Major long tracts**
- **Cranial nerves**
- **Reticular formation**
- **Autonomics**



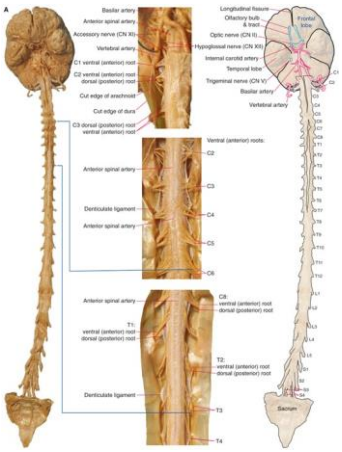
Brainstem

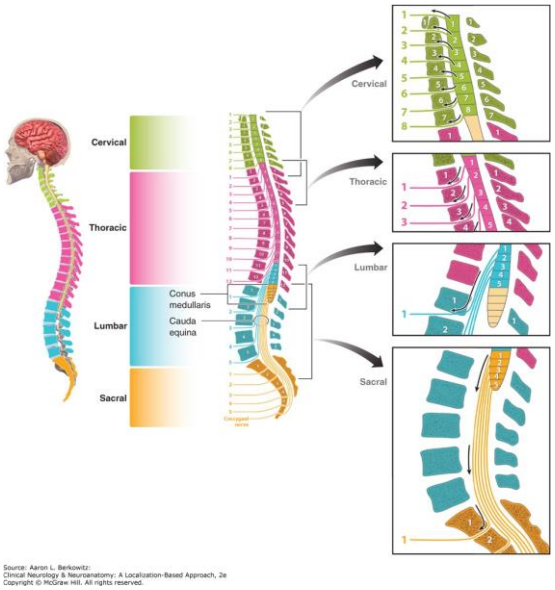
- **Major long tracts**
- **Cranial nerves**
- **Reticular formation**
- **Autonomics**



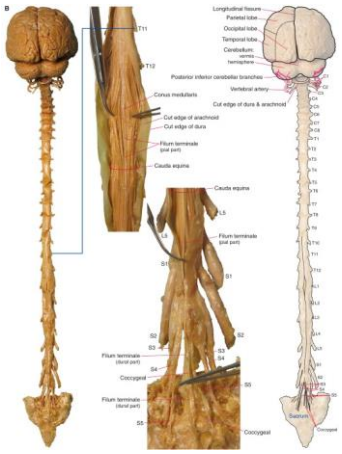
Appendix 5

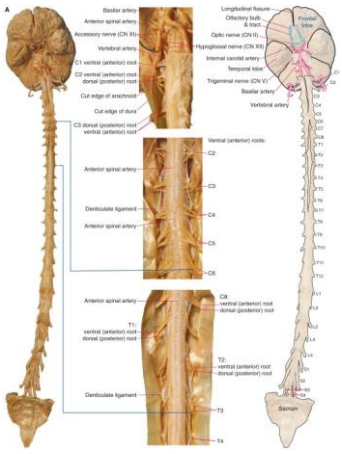
Spinal cord





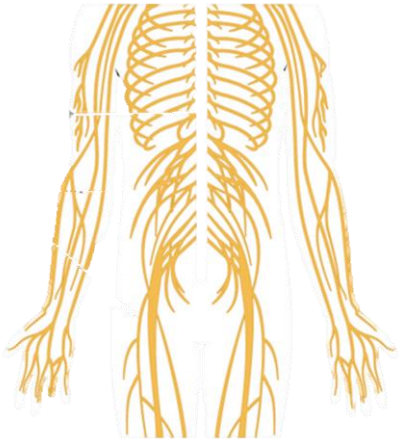
Spinal cord





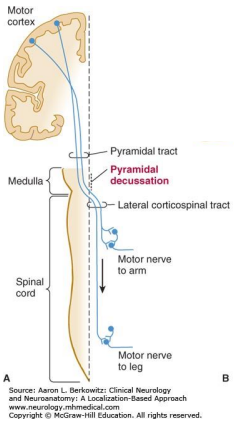
Appendix 5

Peripheral Nervous system



- Nerve Root
- Nerve
- Neuromuscular junction
- Muscle or effector organ

Somatic: CNS & PNS



Cortical spinal tract (Descending motor pathway)

i. Motor cortex of precentral gyrus
ii. Somatotopically arranged

i. Motor neuron #1 from cortex (UMN)

i. Internal capsule
ii. Cerebral peduncle
iii. Pons
iv. Pyramid of medulla
v. Decussate at cervicomedullary junction
vi. Descend in lateral corticospinal tract

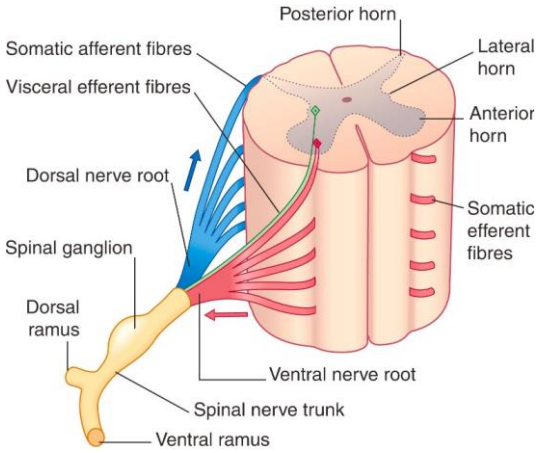
ii. Motor neuron #2

i. Anterior horn of SC (LMN)
ii. Ventral root
iii. Peripheral nerve

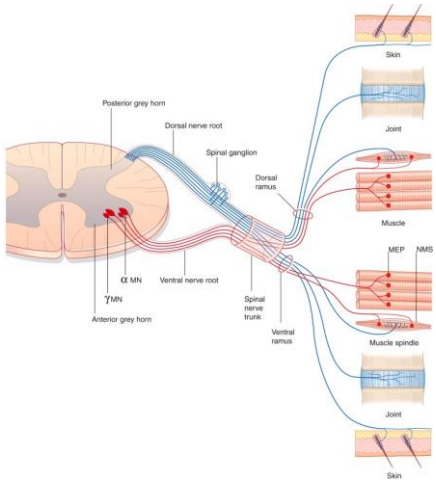
Appendix 5

SOMATIC: PNS

i. Peripheral somatic nervous system
   i. Dorsal roots (sensory)
   ii. Ventral root (motor)
   iii. Peripheral nerve (mixed)
   iv. Dorsal rami – (mixed) 1. sensory input from dorsal midline (back) and 2. erector muscles (core)
   v. Ventral rami - Emerge to become mixed somatic nerves of extremities and torso



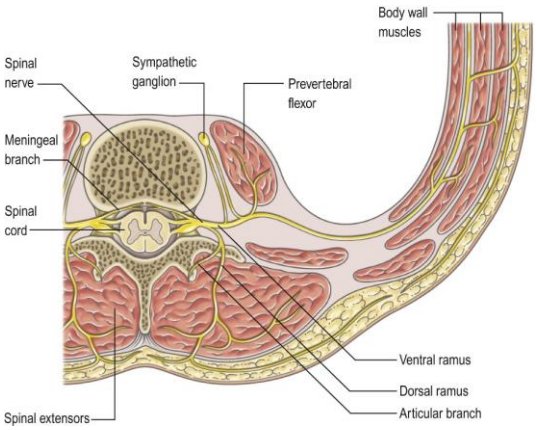
SOMATIC: PNS

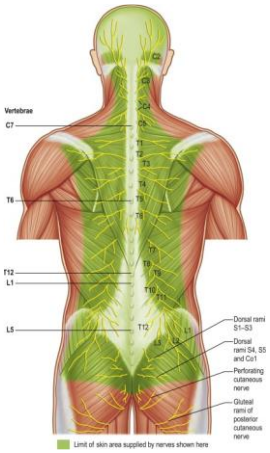


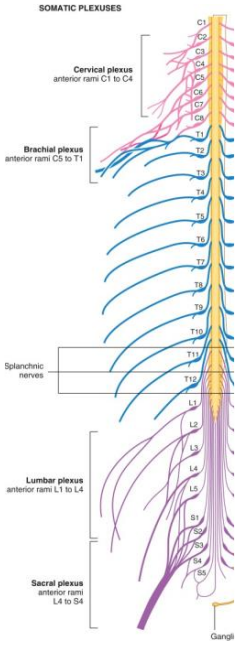
i. Dorsal rami – (mixed) 1. sensory input from dorsal midline (back) and 2. erector muscles (core)
ii. Ventral rami - Emerge to become mixed somatic nerves of extremities and torso

Appendix 5

Dorsal primary rami







Peripheral nerves

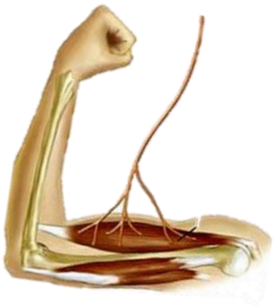
i. Cervical plexus
   i. Cervical roots are ventral rami of cervical spinal nerves
   ii. Roots combine and recombine into plexuses
   iii. From plexuses emerge peripheral nerves

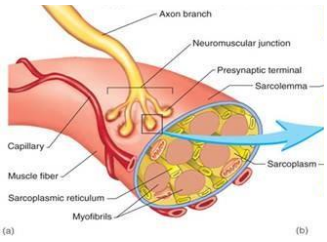
ii. Brachial plexus
   i. C5 - T1 roots are ventral rami of cervical and thoracic spinal nerves
   ii. Roots combine into trunks, trunks into divisions and divisions into “branches” which are peripheral nerves proper

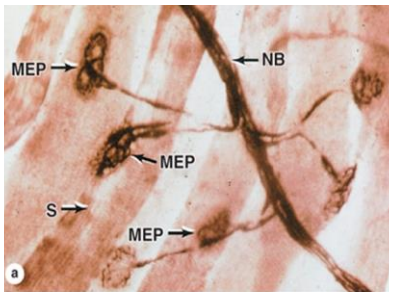
iii. Sacral plexus

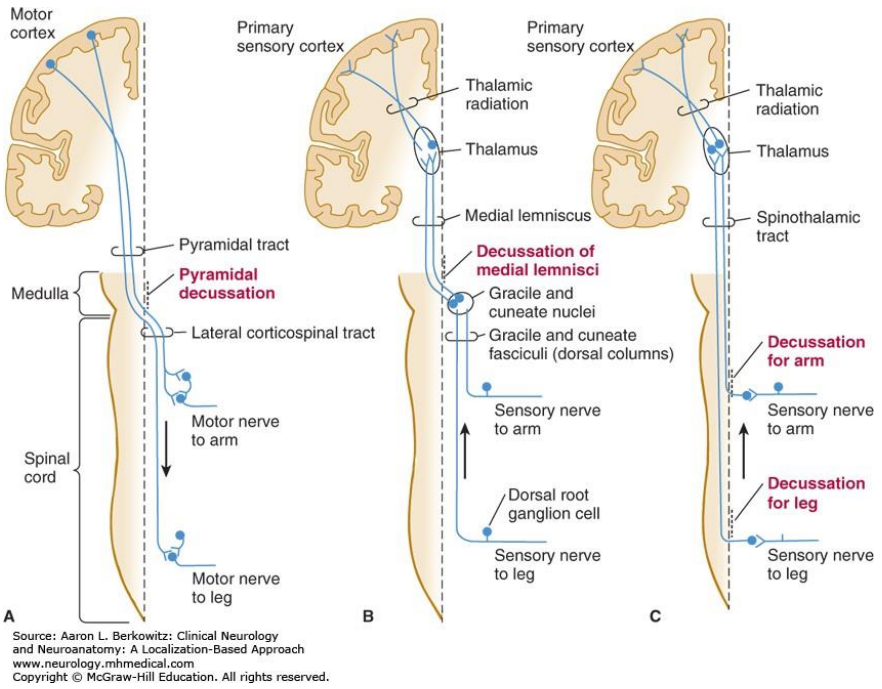
Appendix 5

Neuromuscular Junction & Muscle



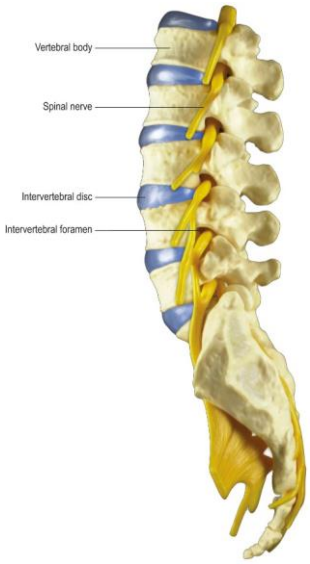


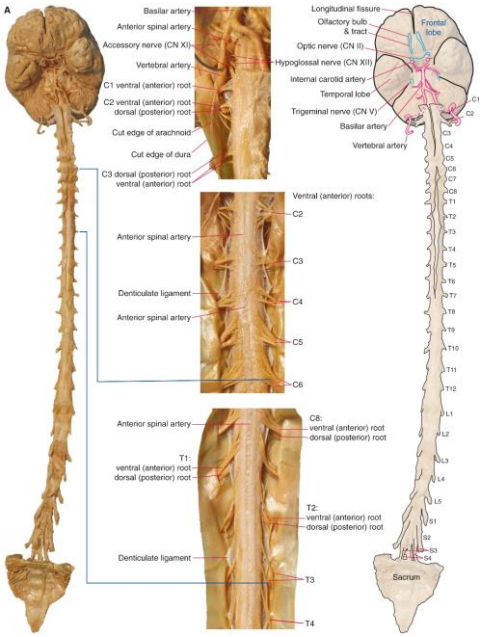




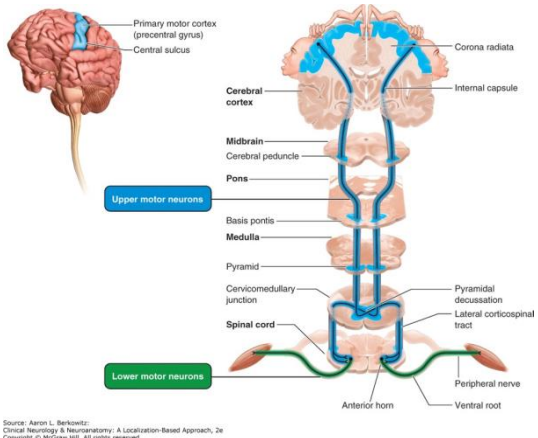
Appendix 5

Nerve root





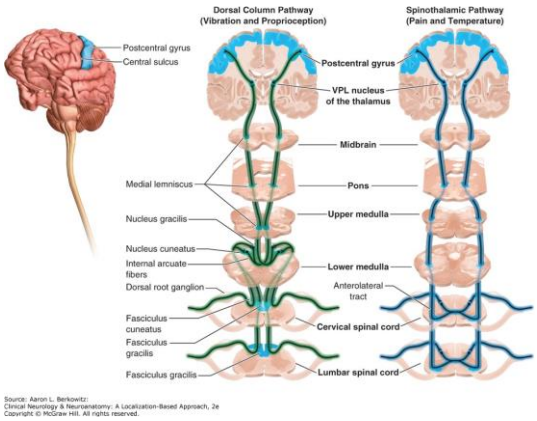
Action: Movement as strength



- **Cortical spinal tract**
  - Motor cortex of precentral gyrus
  - Somatotopically arranged
- Motor neuron #1 from cortex
  - Internal capsule
  - Cerebral peduncle
  - Pons
  - Pyramid of medulla
  - Decussate at cervicomedullary junction
  - Descend in lateral corticospinal tract
- Motor neuron #2
  - Anterior horn of SC
  - Ventral root
  - Peripheral nerve

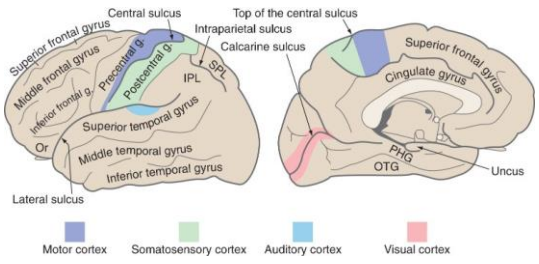
Appendix 5

Perception: Somatic sensation



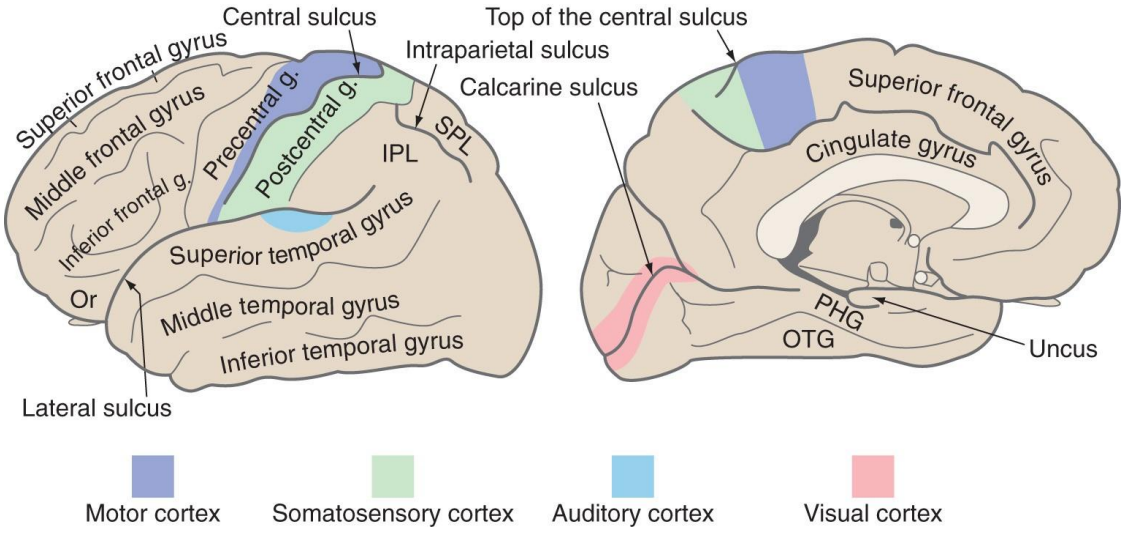
- **Dorsal column pathway** (vibration and proprioception)
- **Spinothalamic pathway** (pain and temperature)

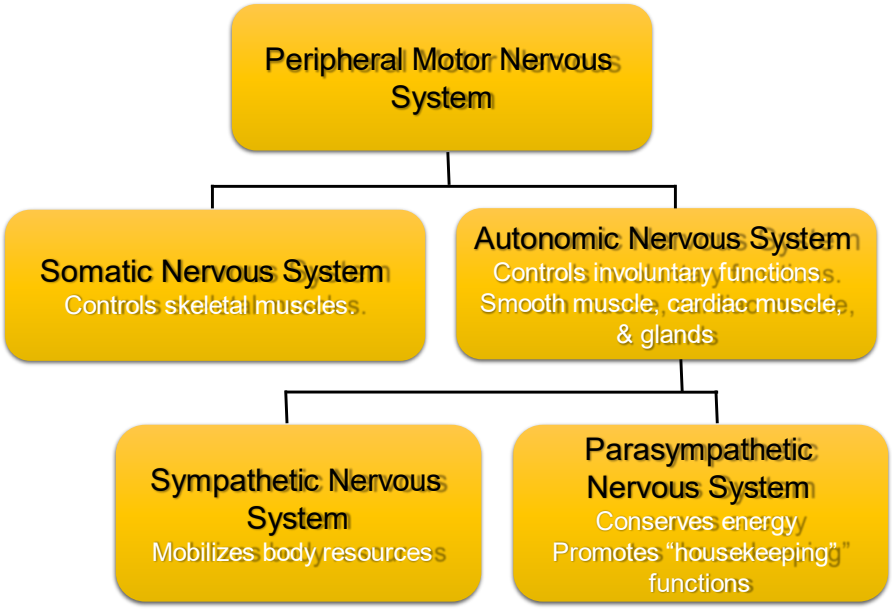
Cognition: Interpreting environment & planning to act



- The frontal lobe-motor areas
- The parietal lobe-somatosensory areas
- The temporal lobe-auditory areas.
- The occipital lobe -visual areas.
- The limbic lobe -interconnected with other limbic structures- in the temporal lobe and plays a role in emotional behavior

Appendix 5

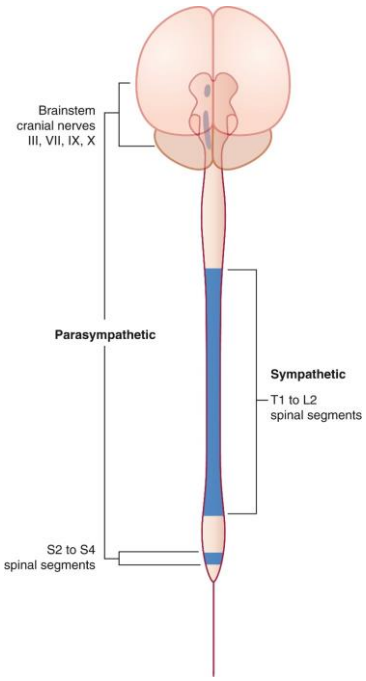

Central sulcus
Top of the central sulcus
Intraparietal sulcus
Calcarine sulcus
Superior frontal gyrus
Middle frontal gyrus
Inferior frontal g.
Precentral g.
Postcentral g.
SPL
IPL
Superior temporal gyrus
Middle temporal gyrus
Inferior temporal gyrus
Or
Lateral sulcus
Superior frontal gyrus
Cingulate gyrus
PHG
OTG
Uncus
Motor cortex
Somatosensory cortex
Auditory cortex
Visual cortex

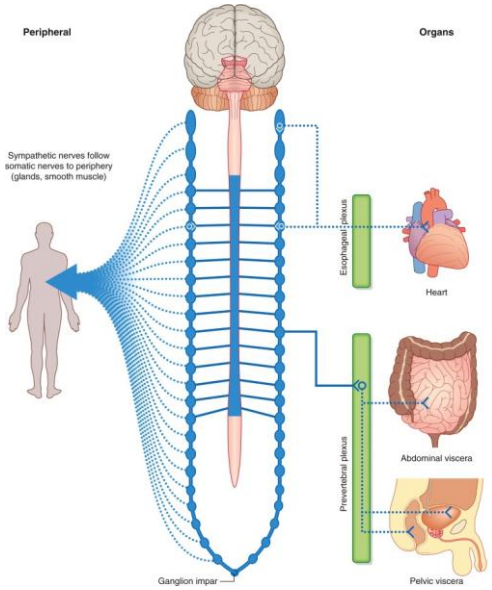

Peripheral Motor Nervous System
Somatic Nervous System
Controls skeletal muscles
Autonomic Nervous System
Controls involuntary functions
Smooth muscle, cardiac muscle, & glands
Sympathetic Nervous System
Mobilizes body resources
Parasympathetic Nervous System
Conserves energy
Promotes "housekeeping" functions

Appendix 5

Autonomic Nervous System

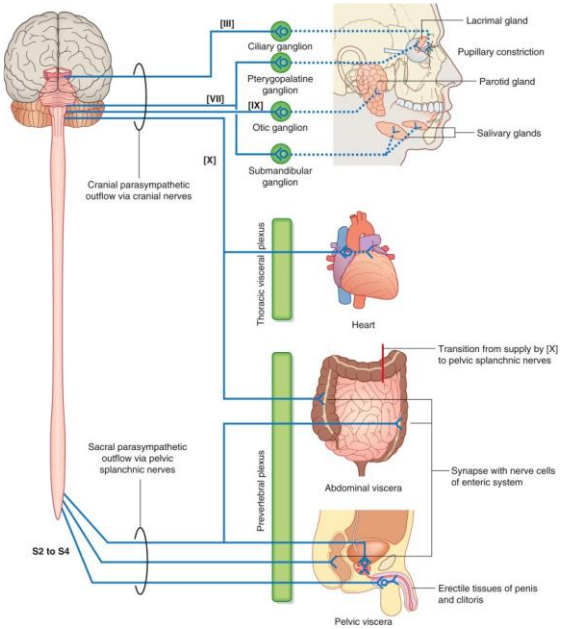
- Craniosacral (Parasympathetic)
- Thoracolumbar (Sympathetic)



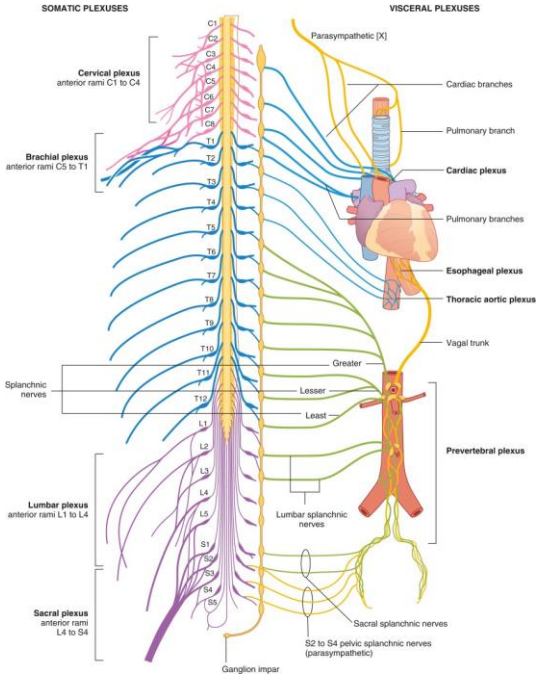


Thoracolumbar

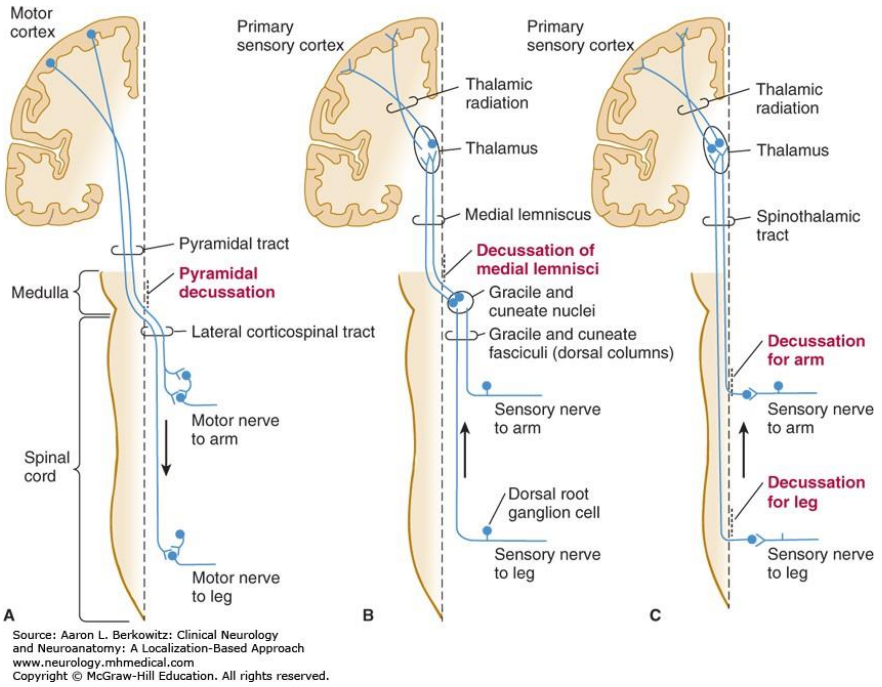
Appendix 5



Craniosacral



Appendix 5


Motor cortex
Pyramidal tract
Pyramidal decussation
Medulla
Lateral corticospinal tract
Motor nerve to arm
Spinal cord
Motor nerve to leg
A
Primary sensory cortex
Thalamic radiation
Thalamus
Medial lemniscus
Decussation of medial lemnisci
Gracile and cuneate nuclei
Gracile and cuneate fasciculi (dorsal columns)
Sensory nerve to arm
Dorsal root ganglion cell
Sensory nerve to leg
B
Primary sensory cortex
Thalamic radiation
Thalamus
Spinothalamic tract
Decussation for arm
Sensory nerve to arm
Decussation for leg
Sensory nerve to leg
C
Source: Aaron L. Berkowitz: Clinical Neurology and Neuroanatomy: A Localization-Based Approach
www.neurology.mhmedical.com
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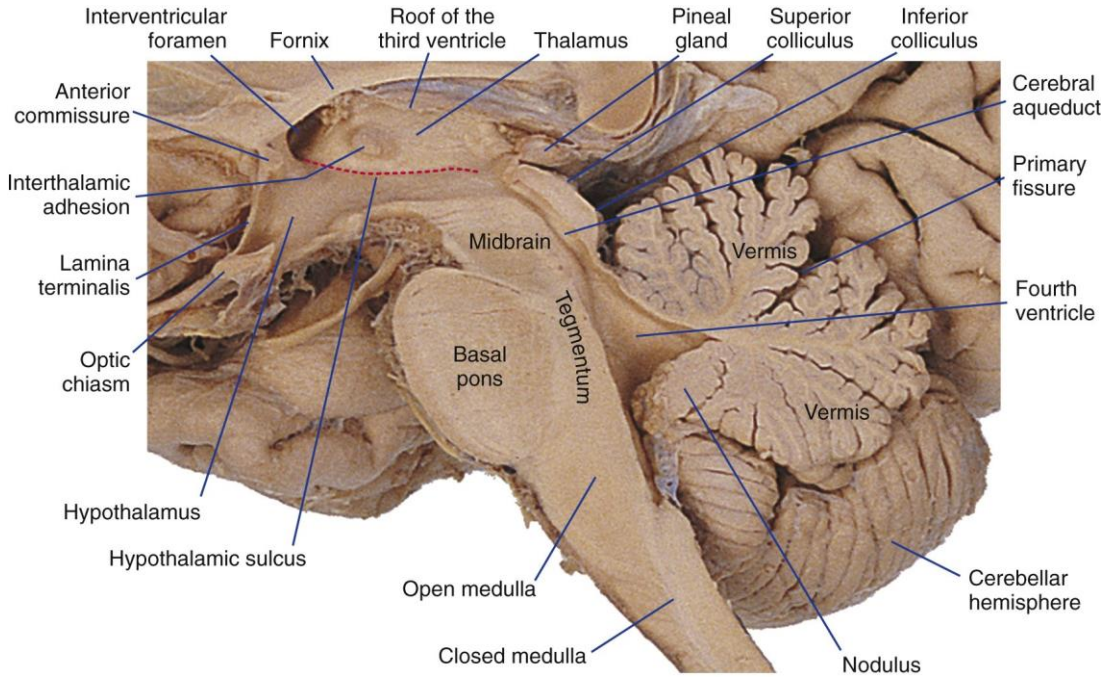
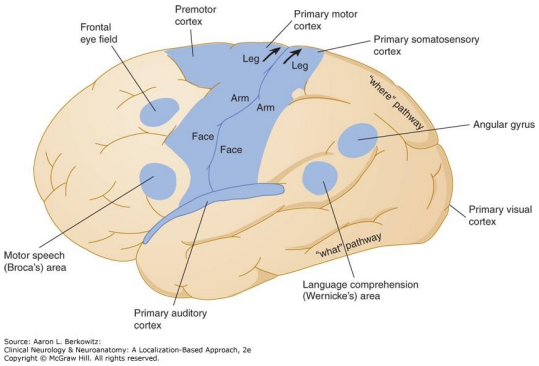
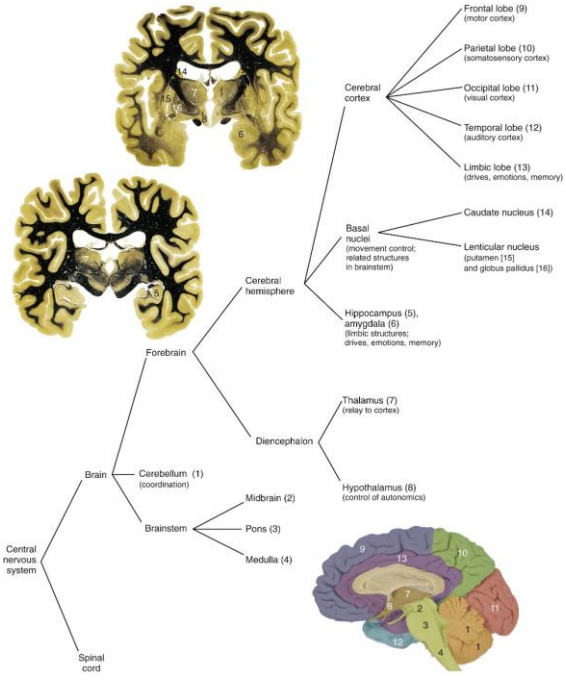

Interventricular foramen
Fornix
Roof of the third ventricle
Thalamus
Pineal gland
Superior colliculus
Inferior colliculus
Anterior commissure
Interthalamic adhesion
Lamina terminalis
Optic chiasm
Hypothalamus
Hypothalamic sulcus
Midbrain
Basal pons
Tegmentum
Open medulla
Closed medulla
Cerebral aqueduct
Primary fissure
Vermis
Fourth ventricle
Vermis
Cerebellar hemisphere
Nodulus

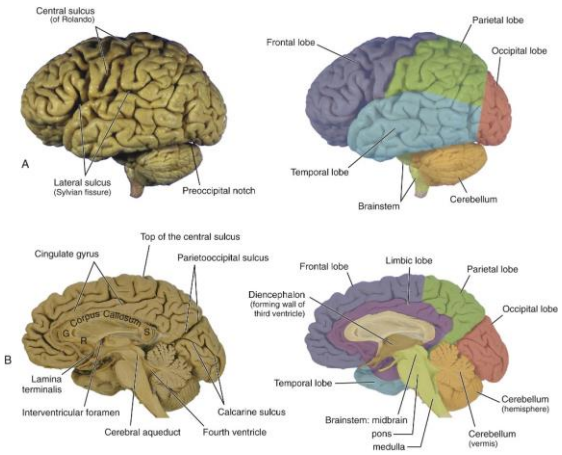
Appendix 5

Cerebral Cortex Areas





Appendix 5



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- https://en.wikiversity.org/wiki/WikiJournal_of_Medicine/Medical_gallery_of_Blausen_Medical_2014

Appendix 6

CNS Environs, vasculature & pathophysiology

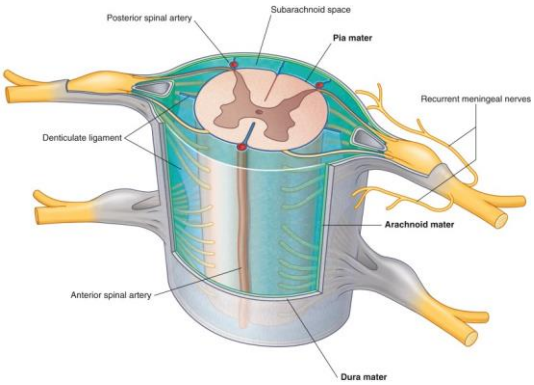
Perdue

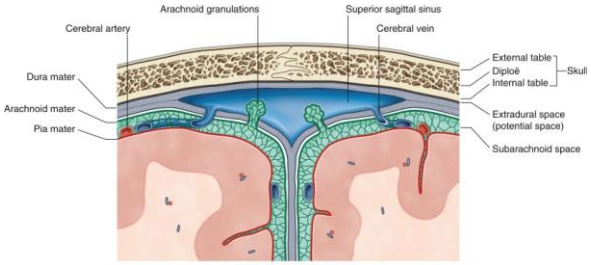
Objectives

- Describe the meninges and their function in the nervous system
- Describe the structure and function of cerebral vasculature

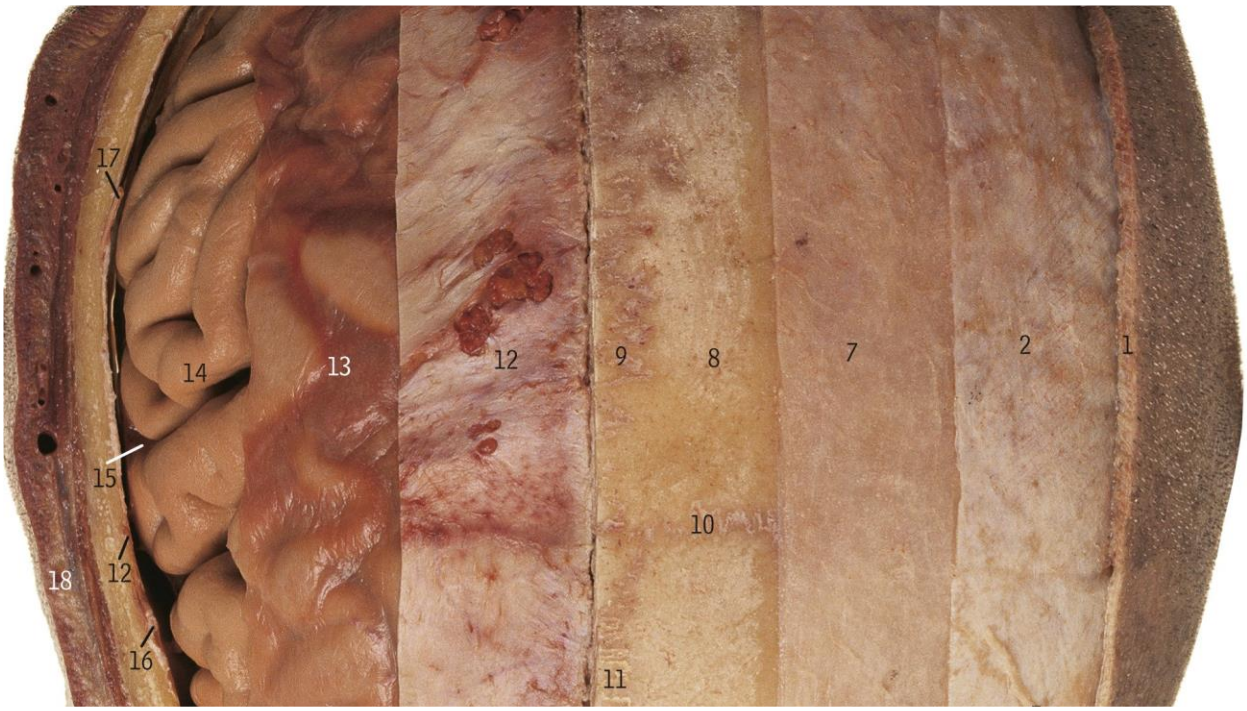
Appendix 6

Meninges



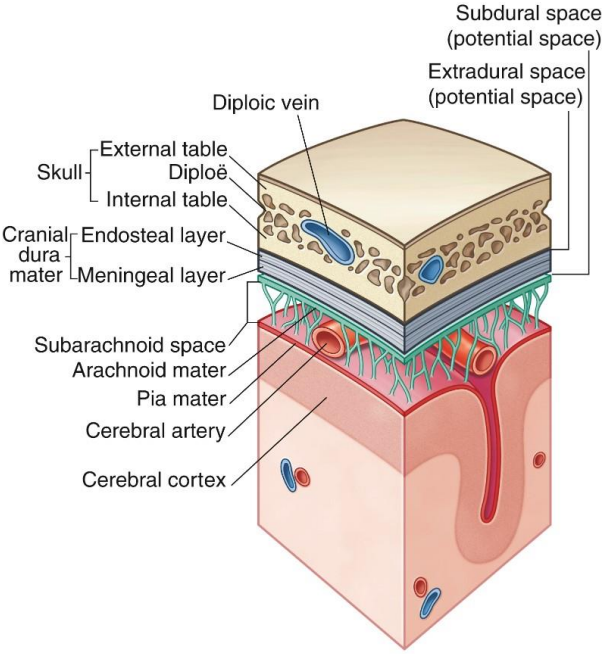


Appendix 6



Meninges

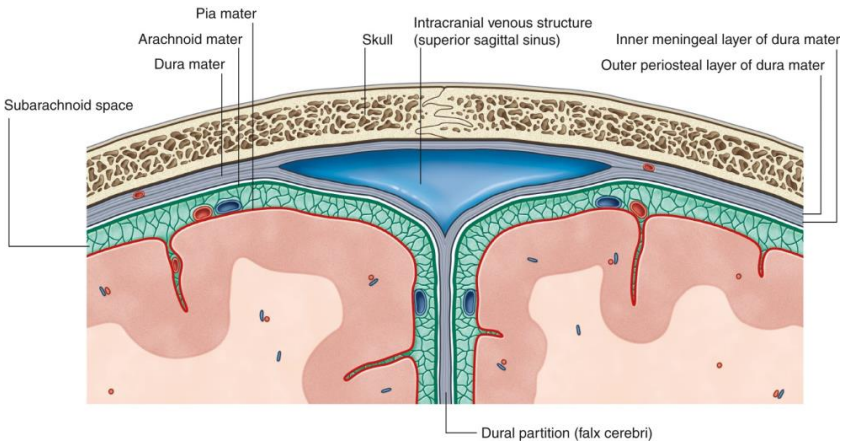
- Skull
- Dura mater
- Arachnoid mater
- Pia mater



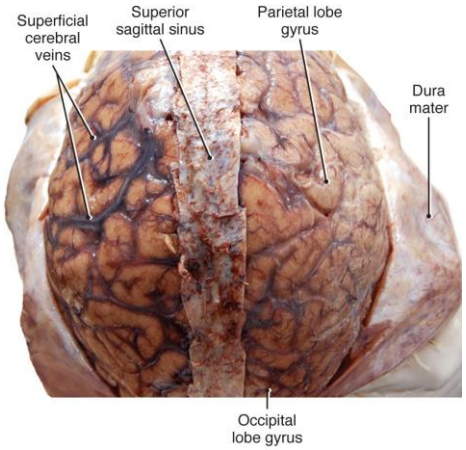
Appendix 6

Meninges

- Skull
- Dura mater
- Arachnoid mater
- Pia mater



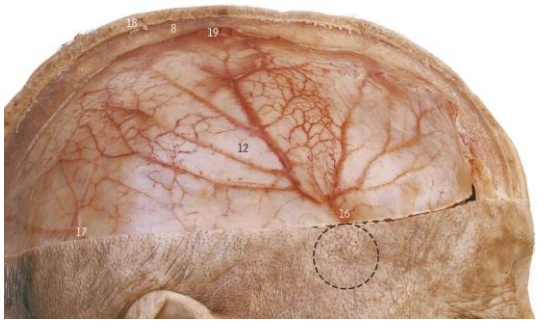
Dura mater



- Literally “tough mother” - tough outer “skin”
- Covers the entire brain, with folds that extend between the hemispheres (falx cerebri) and between the cerebral hemispheres and cerebellum (tentorium cerebelli)
- Surrounds the brain but does not invaginate into the sulci.
- Outer surface of the dura mater is tightly affixed to the skull

Appendix 6

Dura mater



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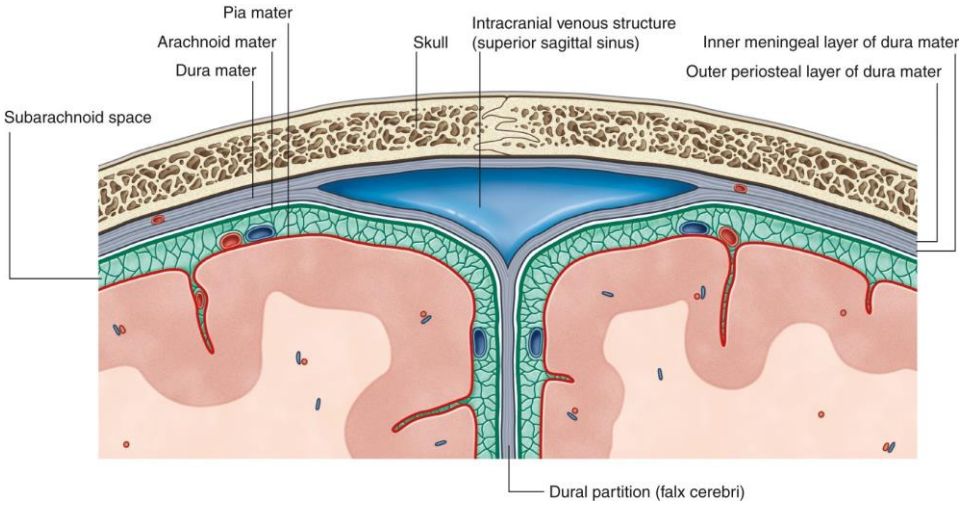
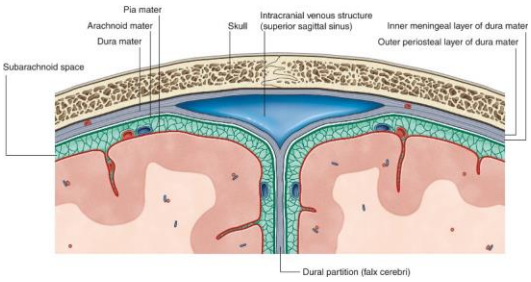
- Outer surface of the dura mater is tightly affixed to the skull

Arachnoid mater



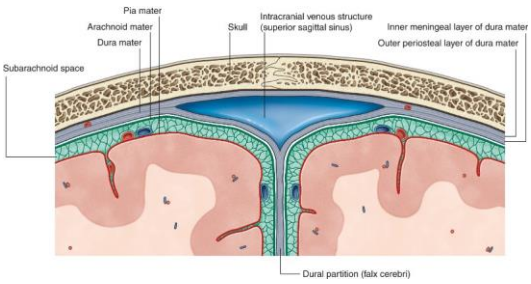
Appendix 6

Arachnoid mater



- Thin membrane beneath the dura mater
- Does not invaginate into the sulci.
- Orange analogy

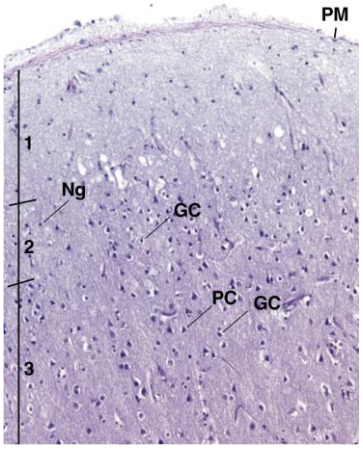
Pia mater



- Invaginates into the sulci and therefore contacts the entire surface area of the brain

Appendix 6

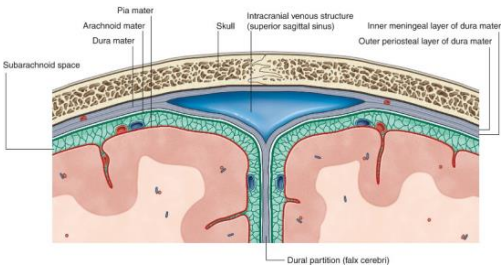
Pia mater



- Invaginates into the sulci and therefore contacts the entire surface area of the brain

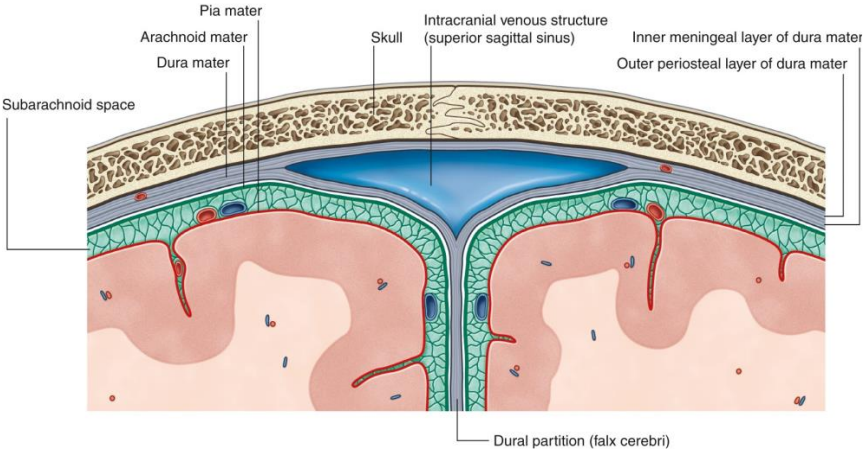
Meninges

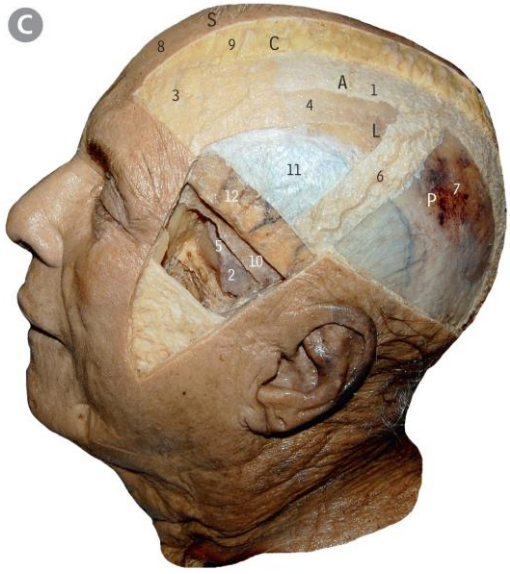
- **Leptomeninges** - Pia and Arachnoid
- **Pachymeninges** - Dura mater
- Infectious meningitis predominantly affects the leptomeninges
- Inflammatory meningitis predominantly affects the pachymeninges
  - Exceptions
    - Neurosarcoidosis can affect the leptomeninges
    - Tuberculosis and fungal infections can affect the pachymeninges
- Metastatic cancer can affect the leptomeninges or the pachymeninges



Appendix 6

Epidural space





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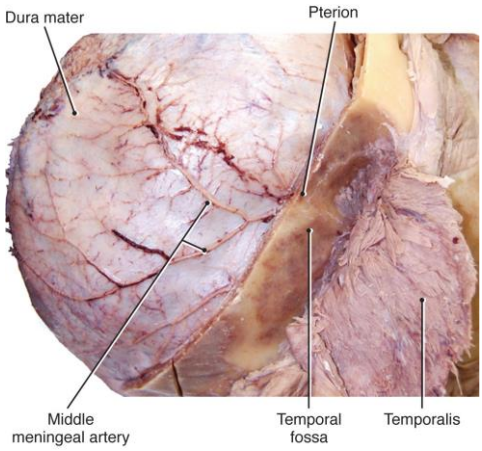
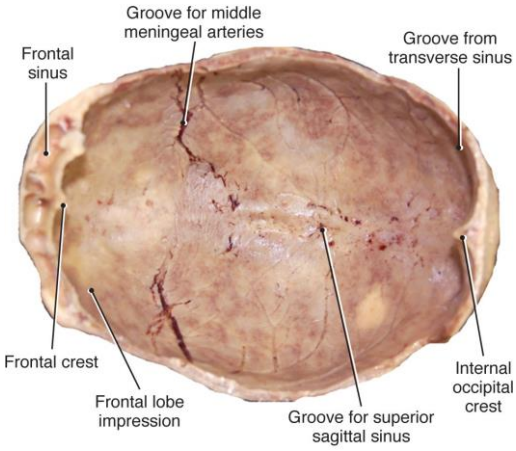
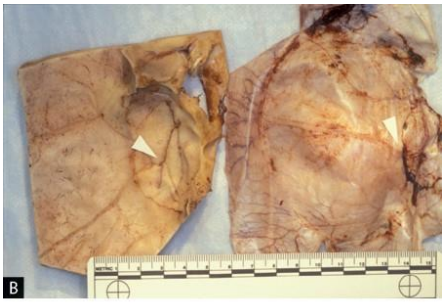
Epidural space

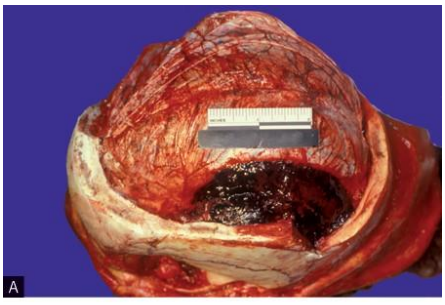




Epidural hematoma



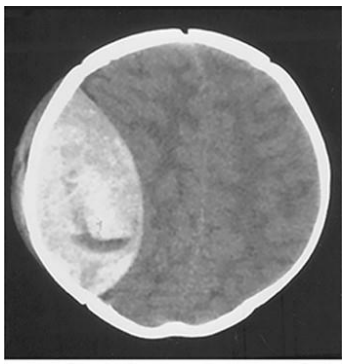
Source: Kemp WL, Burns DK, Brown TG: *Pathology: The Big Picture*: www.accessmedicine.com



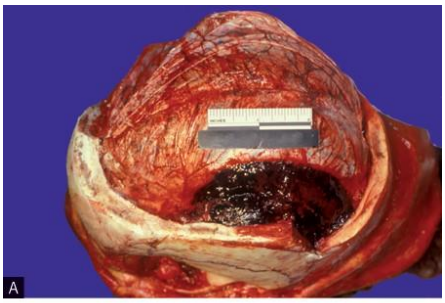
Source: Kemp WL, Burns DK, Brown TG: *Pathology: The Big Picture*: www.accessmedicine.com

Appendix 6

Epidural hematoma

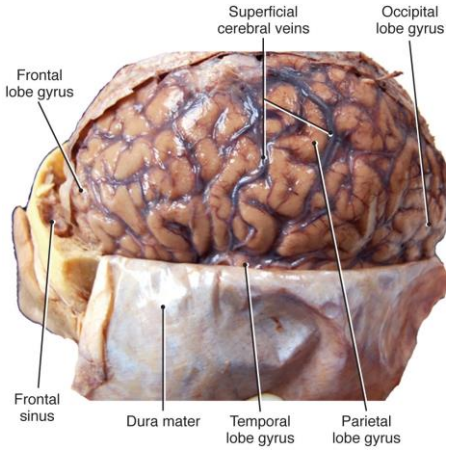


Source: Julia I. Reading: *Test-Taking Strategies for the USMLE STEP 2 Exam: Proven Methods to Succeed*



Source: Kemp WL, Burns DK, Brown TG: *Pathology: The Big Picture*: www.accessmedicine.com

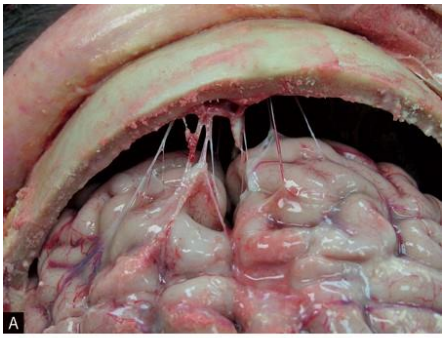
Subdural space



- The space between the dura and arachnoid
- …back to the orange

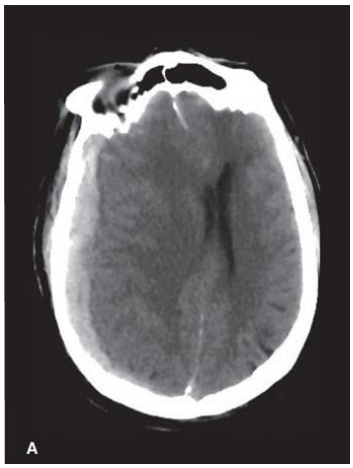
Appendix 6

Subdural hematoma



- Tearing of bridging veins that connect the cerebral hemispheres to the dural vessels and the superior sagittal sinus.
- Elderly and alcoholics, cerebral atrophy leads to elongation of the distance the bridging veins must cross between the cortical surface and the dural sinuses

Subdural hematoma

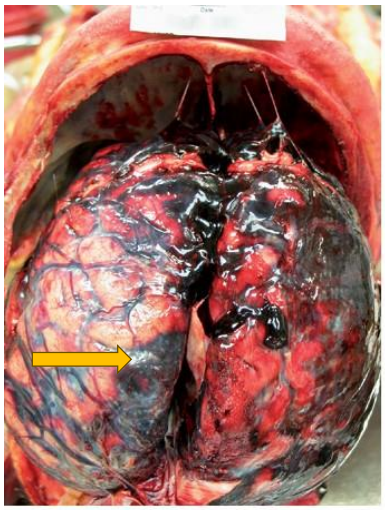


- Crescent shaped hemorrhage
- More diffuse in surface area than epidural hemorrhage

Appendix 6

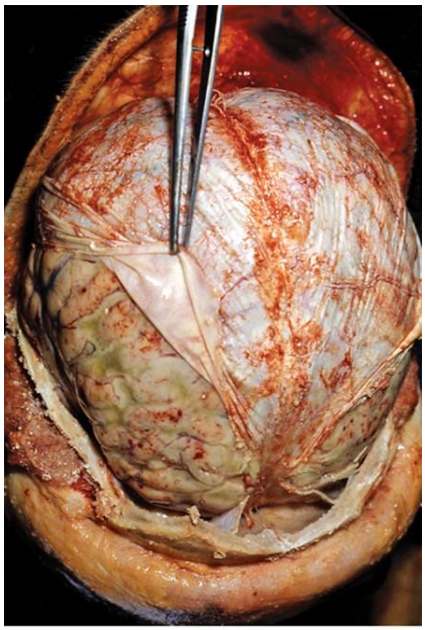
Subarachnoid hemorrhage

- Bleeding into subarachnoid space
- **Circle of Willis** arteries
  - Trauma
  - Aneurysms



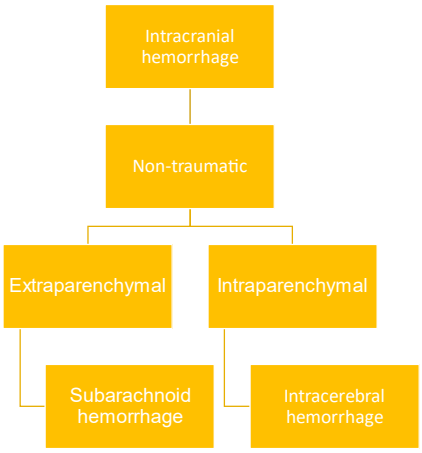
Meningitis

- Infectious
  - Leptomeninges & Subarachnoid space

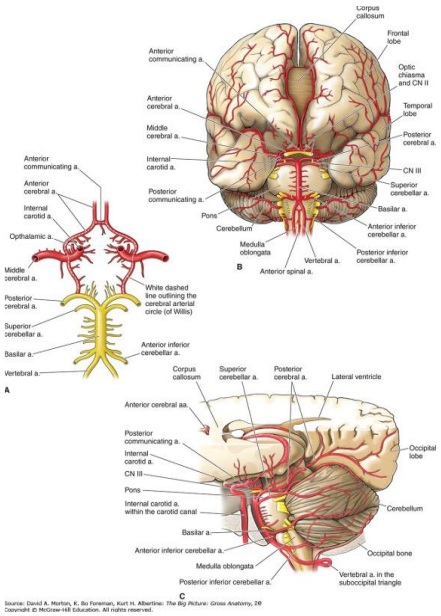


Appendix 6

Hemorrhagic strokes



Cerebral Plumbing



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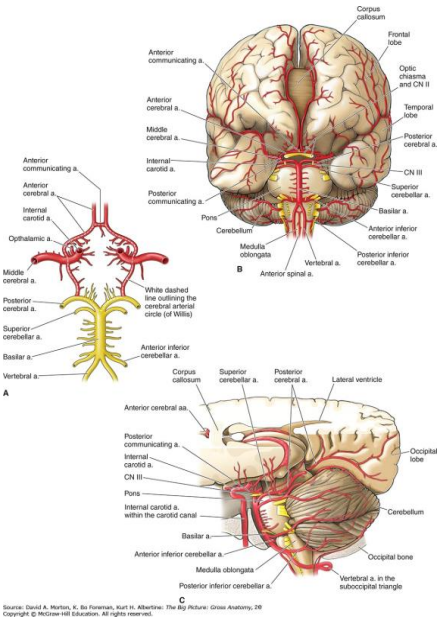
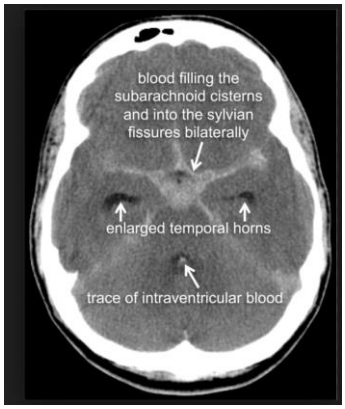


Cerebral Plumbing

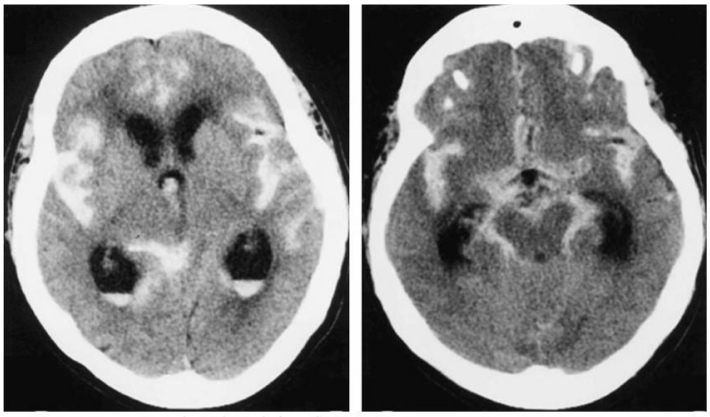
Subarachnoid hemorrhage

- Blood in the subarachnoid space, i.e. between the arachnoid membrane and the pia mater.



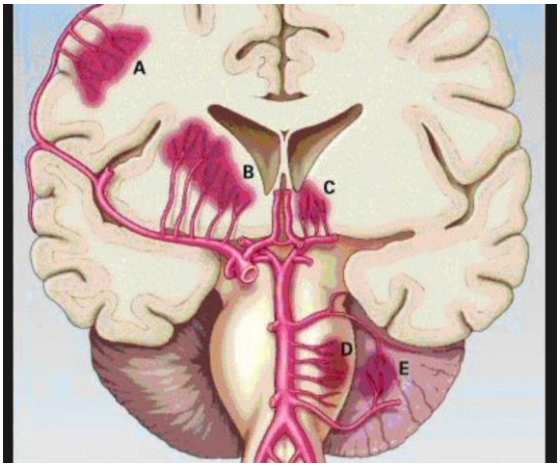
Appendix 6

Subarachnoid hemorrhage



Intracerebral Hemorrhage

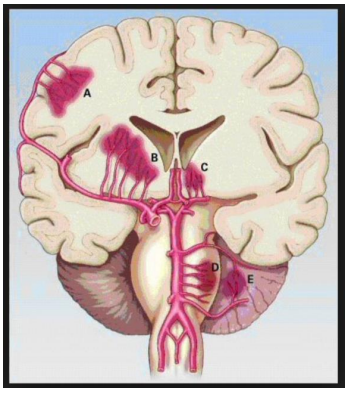
- Hemorrhage within the brain parenchyma
- Second most common form of STROKE
  - More deadly than ischemic



Appendix 6

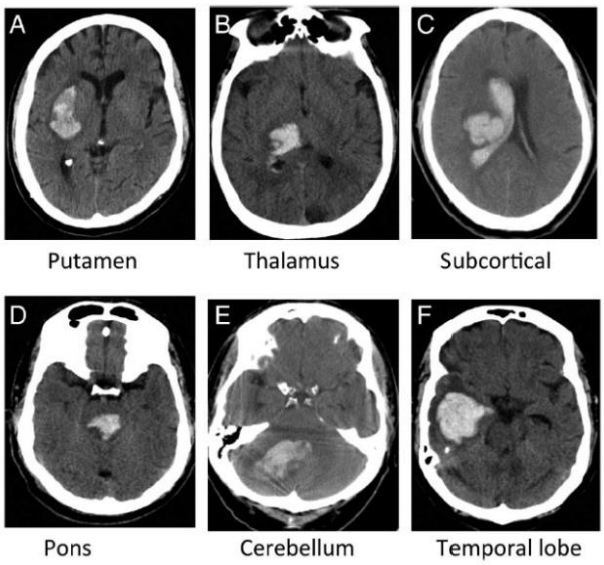
Pathogenesis

- Bursting of small intracerebral arteries
  - Long term hypertension can cause weakening of blood vessels deep in the brain



Locations

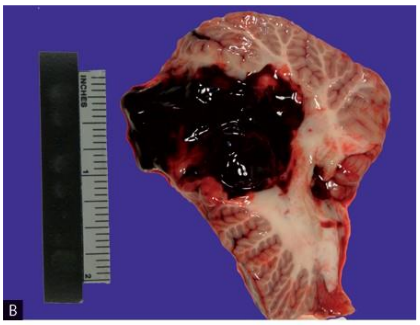
- Basal Ganglia – 50%
- Thalamus – 15%
- Pons – 10-15%
- Cerebellum – 10%



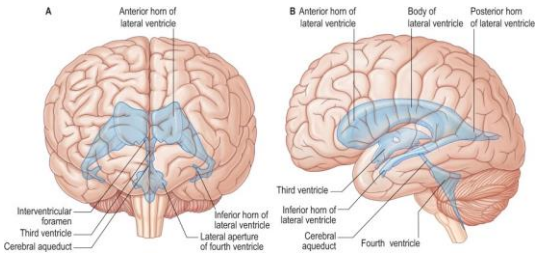
Appendix 6

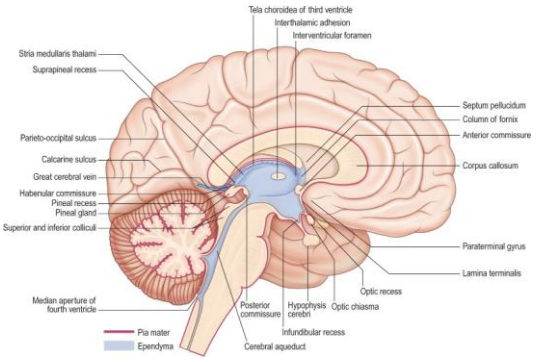
Cerebellar hemorrhage

- Acute headache with associated vestibular symptoms (vertigo or ataxia) should be considered a **cerebellar hemorrhage** until proven otherwise.
- Requires prompt surgical evacuation of the hematomain order to prevent the rapid progression to severe disability or death



Cerebrospinal fluid flow

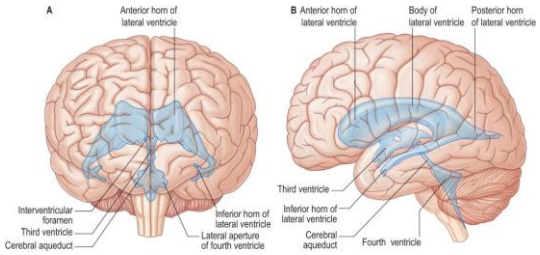




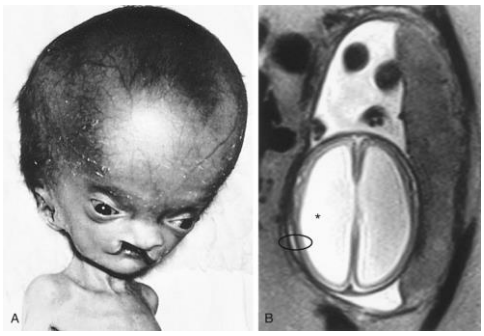
Appendix 6

Cerebrospinal fluid flow

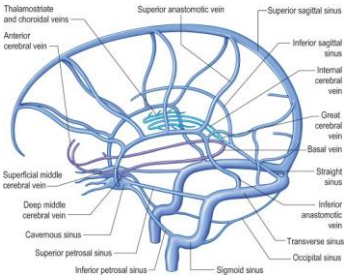
I. The CSF is produced in the **choroid plexus (**which lines the ventricles)
II. The two **lateral ventricles** drain into the **third ventricle** via the **foramina of Monro**
III. The CSF then flows from the third ventricle to the **fourth ventricle** (between the brainstem and cerebellum) by way of the **cerebral aqueduct** in the midbrain.
IV. The fourth ventricle is continuous with the central canal of the spinal cord.
V. From the fourth ventricle, CSF can exit the ventricular system into the subarachnoid space via the **foramen of Magendie** (midline) and the **foramina of Luschka** (lateral) to bathe the outer surface of the brain and spinal cord.
VI. CSF is then reabsorbed via the **arachnoid granulations** into the venous sinuses.
VII. CSF is produced at a rate of approximately half a liter per day (approximately 20 mL per hour).

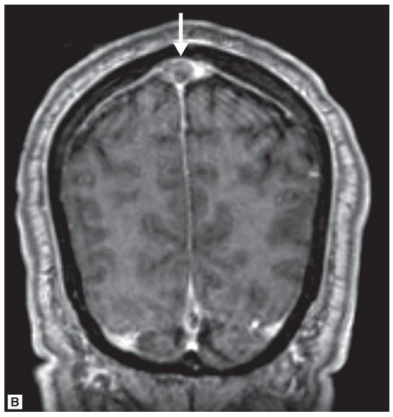


Hydrocephalus

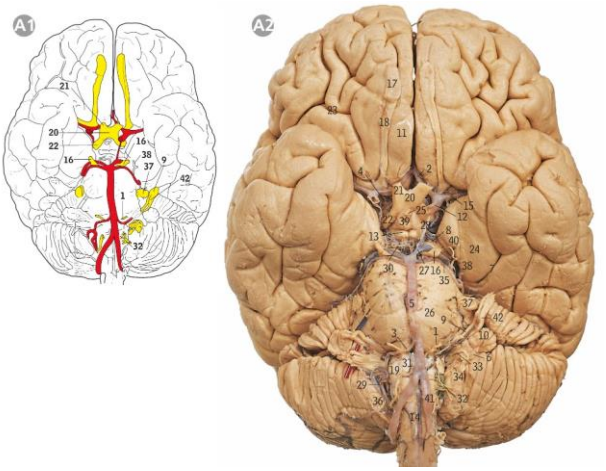


Cerebral venous system





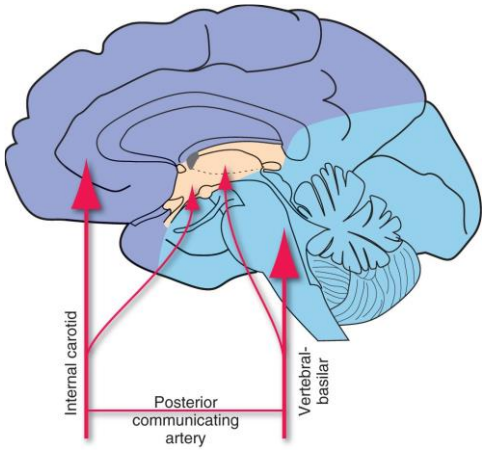
Blood supply of brain



- (CNS) is tremendously active metabolically—relative to its weight, it uses much more than its share of the available oxygen and glucose
- Abundant and closely regulated arterial supply and a large venous drainage system
- Complicated

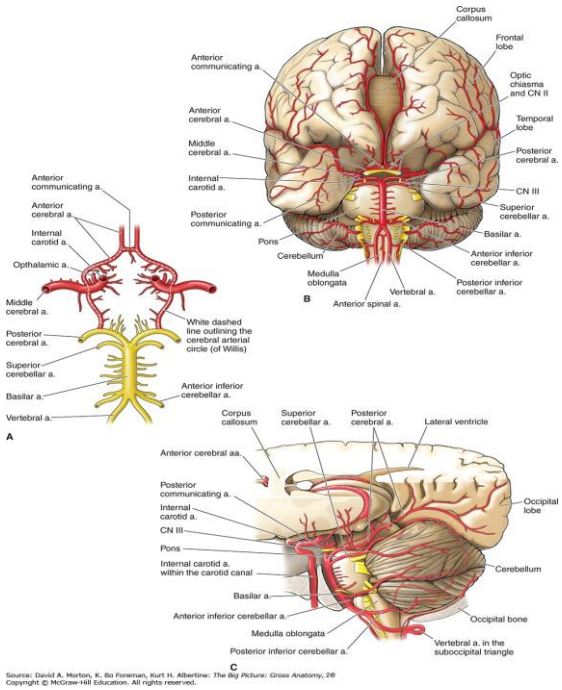
Appendix 6

Blood supply of brain



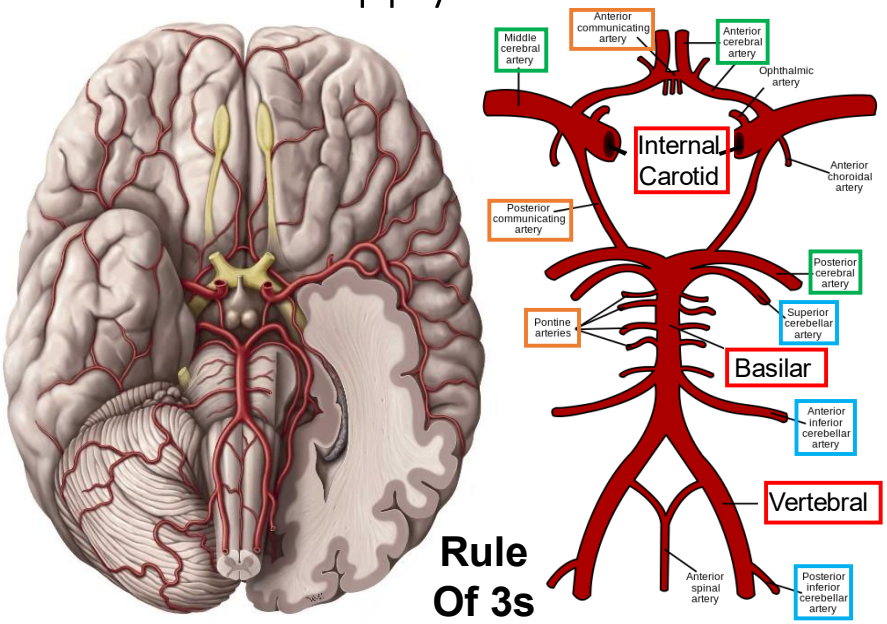
- **Internal carotid** system of each side supplies the ipsilateral cerebral hemisphere
  - Except for the medial surface of the occipital lobe and the medial and inferior surfaces of the temporal lobe
  - **Anterior circulation**
- **Vertebral-basilar system** supplies those parts of the occipital and temporal lobes, as well as the brainstem and cerebellum
  - **Posterior circulation**
- Diencephalon
  - **Vertebral/basilar** system supplies most of the **thalamus**
  - **Internal carotid** system supplies most of the **hypothalamus**

Vascular supply

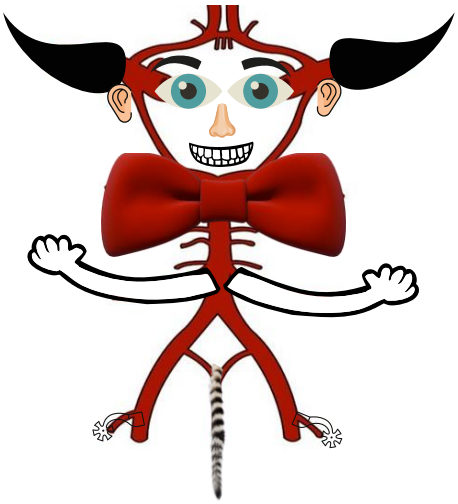


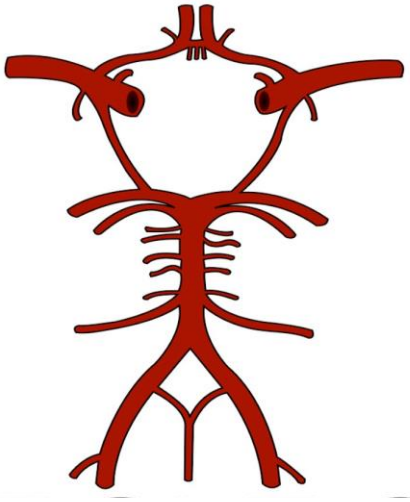
Appendix 6

Blood Supply to the Brain



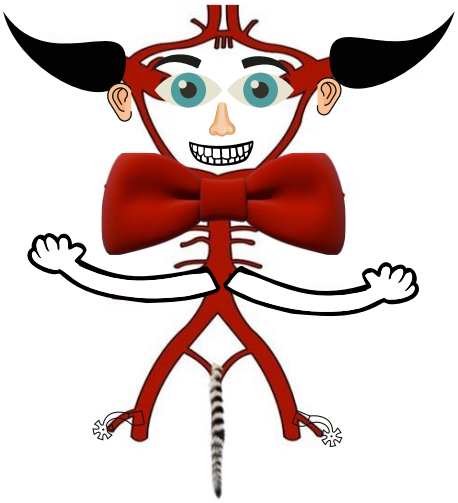
Circle of Willis

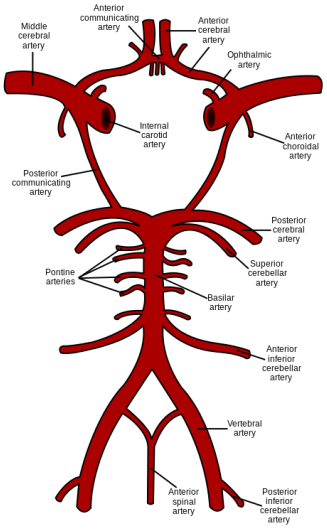




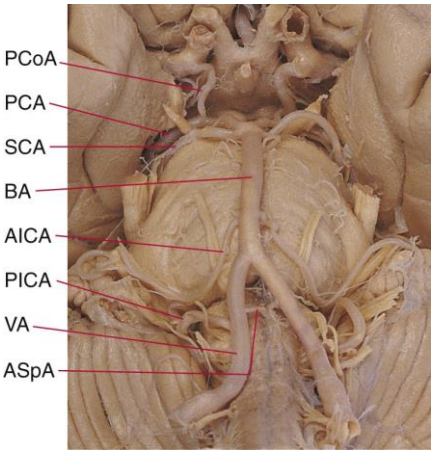
Appendix 6

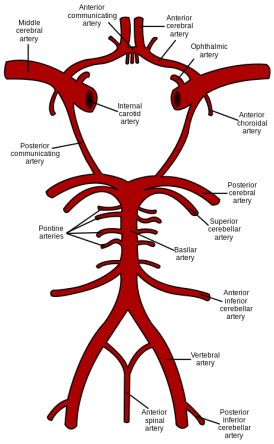
Circle of Willis



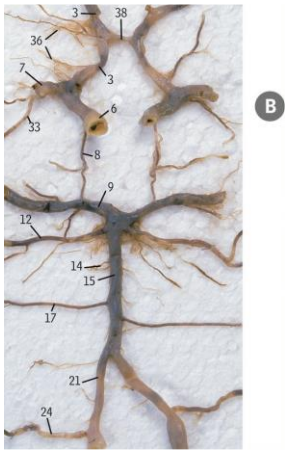


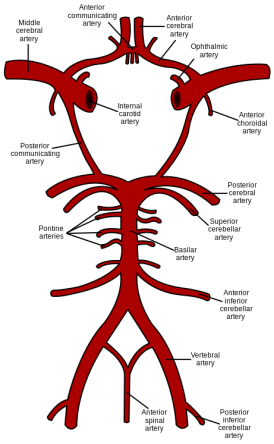
Blood Supply to the Brain

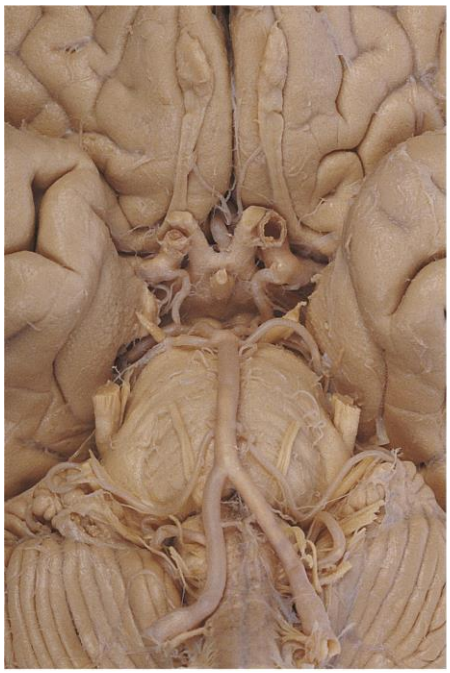




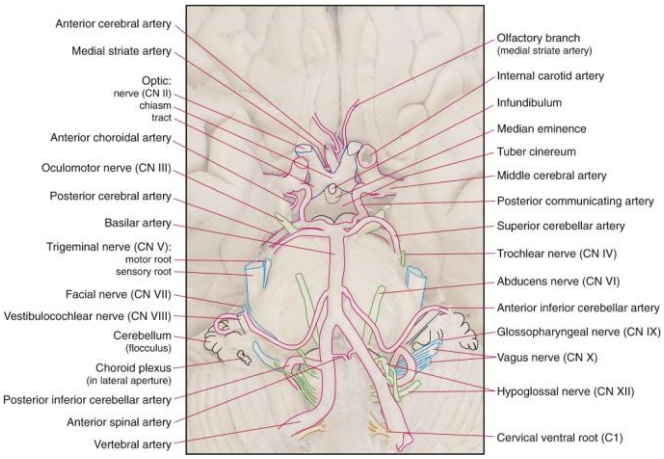
Blood Supply to the Brain







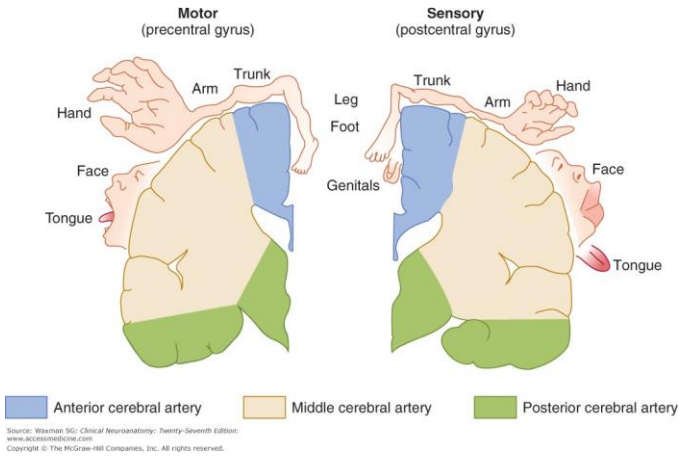
Blood Supply to the Brain



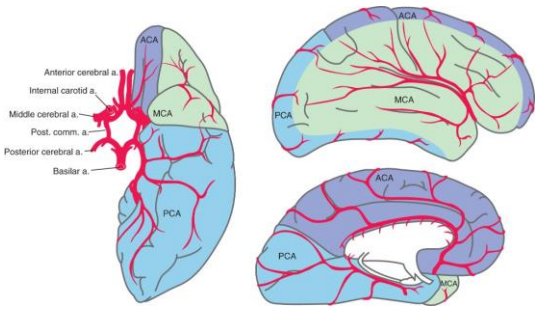
Appendix 6

Blood Supply to the Brain

- Anterior cerebral artery
- Middle cerebral artery
- Posterior cerebral artery



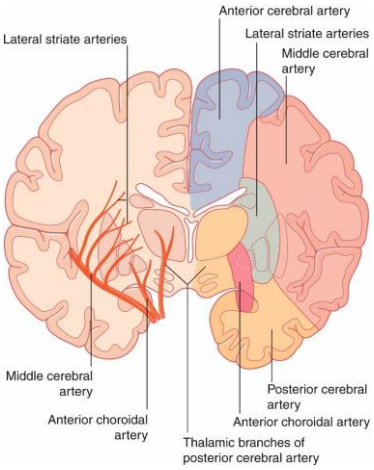
Blood supply of brain



- Internal carotid artery has two major terminal branches
  - Anterior (ACA)
  - Middle cerebral arteries (MCA)
- Both give rise to many small perforating arteries
  - hypothalamus, basal ganglia, and internal capsule

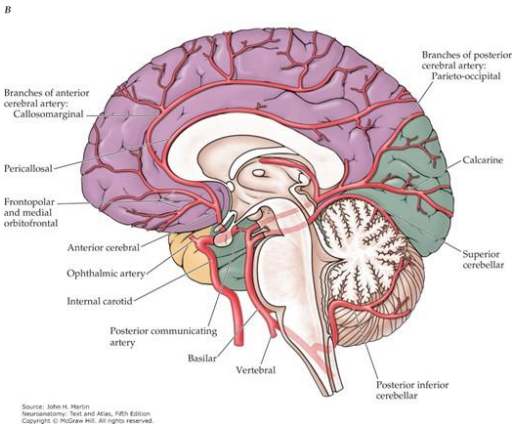
Appendix 6

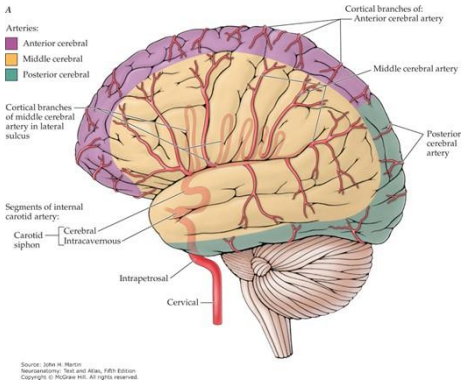
Blood supply of brain



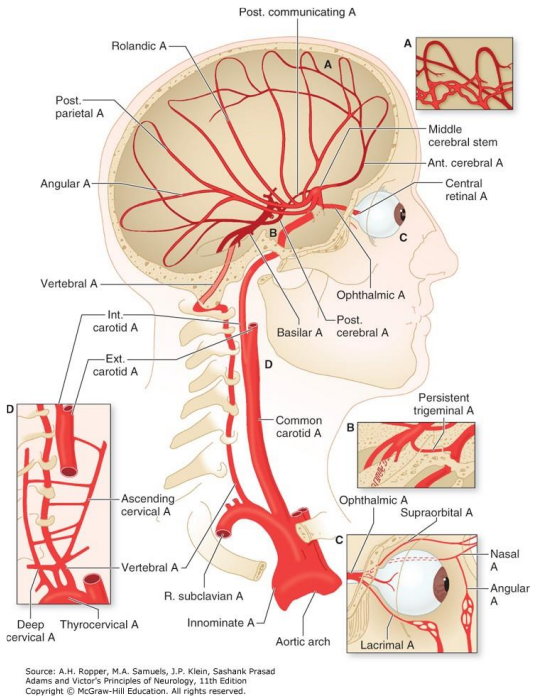
Artery	Territory
Anterior circulation	
Internal carotid artery branches	
Anterior choroidal	Hippocampus, globus pallidus, lower internal capsule
Anterior cerebral	Medial frontal and parietal cortex and subjacent white matter, anterior corpus callosum
Middle cerebral	Lateral frontal, parietal, occipital, and temporal cortex and subjacent white matter
Lenticulostriate branches	Caudate nucleus, putamen, upper internal capsule

Cerebral Cortex

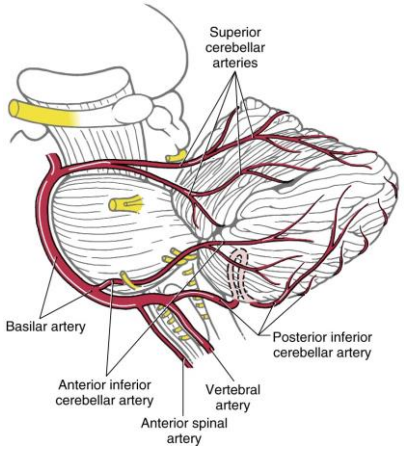




Appendix 6



Posterior circulation



Posterior circulation	
Vertebral artery branches	
Posterior inferior cerebellar	Medulla, lower cerebellum
Basilar artery branches	
Anterior inferior cerebellar	Lower and middle pons, anterior cerebellum
Superior cerebellar	Upper pons, lower midbrain, upper cerebellum
Posterior cerebral	Medial occipital and temporal cortex and subjacent white matter, posterior corpus callosum, upper midbrain
Thalamoperforate branches	Thalamus
Thalamogeniculate branches	Thalamus

Appendix 6

Sources

Thanks to Dr. Rob Jackson, PhD for the use of his Circle of Willis slide.

Netter Collection of Medical Illustrations: Nervous System, Part I - Brain. JonesNeuroanatomy Text and Atlas, 5th Edition

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- Pathology Big Picture, Access Medicine, McGraw-Hill

Appendix 7

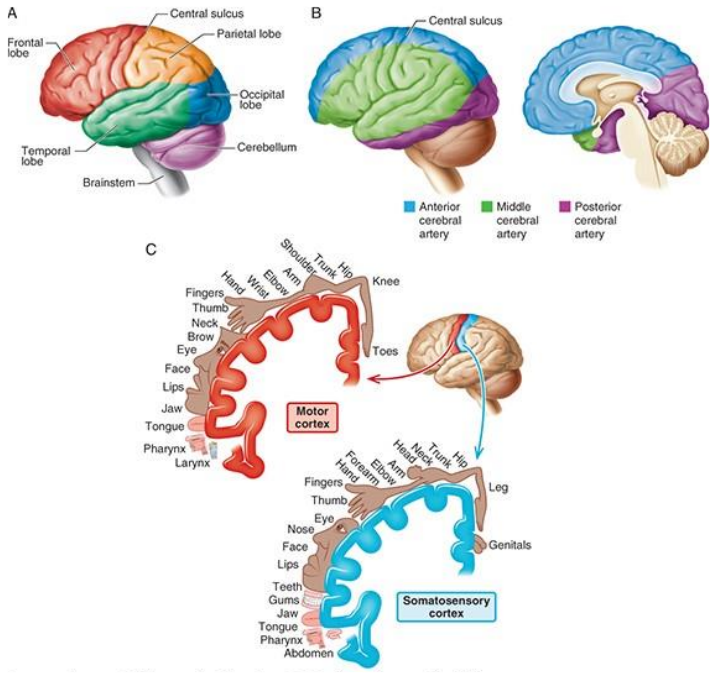
Movement & Sensation

Perdue

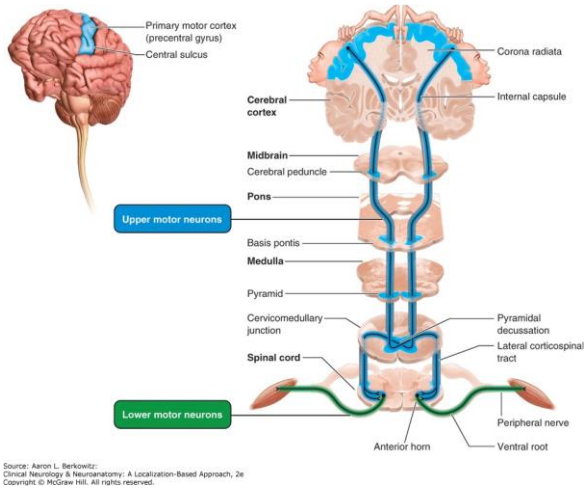
Movement & Sensation

- Learning objectives
- Localizing “lesions” that disrupt movement and sensation
    - Movement (Strength)– corticospinal tract
    - Sensation - dorsal column (ML) and spinothalamic tract
    - Movement (Reflex)
    - Weakness: UMN versus LMN lesions
        - UMN
            - Brain
            - Spinal cord
        - LMN:
            - Anterior horn cells
            - Root
            - Peripheral nerves
            - NMJ
            - Muscles

Appendix 7



Movement as strength

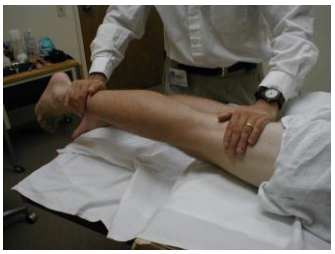


- **Cortical spinal tract**
  - Motor cortex of precentral gyrus
  - Somatotopically arranged
- **Motor neuron #1 from cortex**
  - Internal capsule
  - Cerebral peduncle
  - Pons
  - Pyramid of medulla
  - **Decussate at cervicomedullary** junction
  - Descend in lateral corticospinal tract
- **Motor neuron #2**
  - Anterior horn of SC
  - Ventral root
  - Peripheral nerve

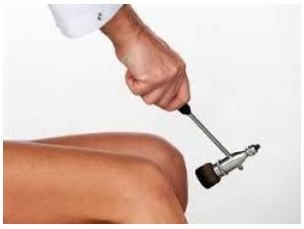
Appendix 7

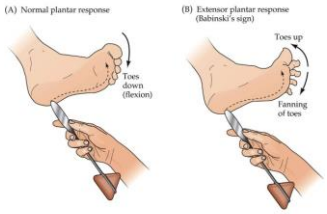
Motor Exam
Corticospinal tract function

Muscle strength



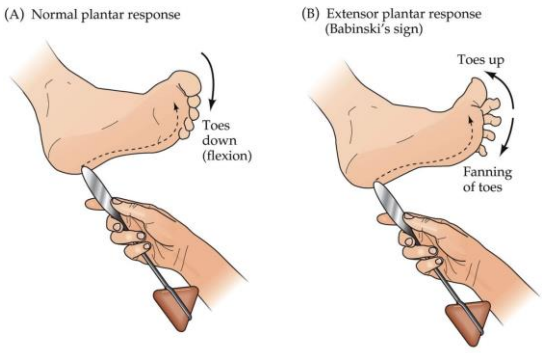
Reflexes





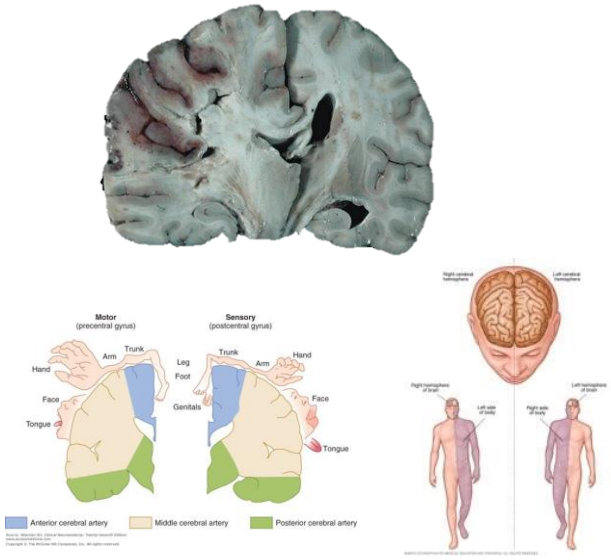
UMN Injury

Babinski sign (extensor plantar response) is an UMN hyperreflexia. This response is never normal (unless you are an infant and you're not fully myelinated) and is a sign of damage the **corticospinal tract (UMN)**.



Appendix 7

Weakness: Abnormality of movement as strength



UMN damage produces **spastic paralysis or weakness**

- Increased reflexes and tone (hyperreflexia, hypertonicity)
- Upward going Babinski's sign
- No atrophy
- No fasciculations (twitching).
- Brain and Spinal cord*
- Stroke (Cerebrovascular accident)

Weakness: Abnormality of movement as strength

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Weakness: Abnormality of movement as strength

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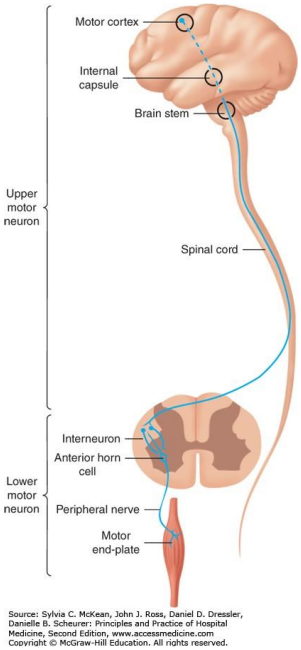


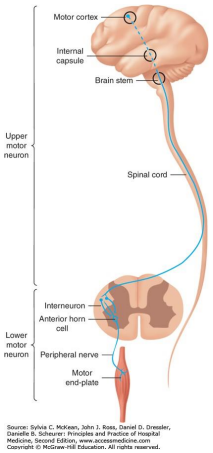
Motor Neurons

Neuron #1: **Upper motor neuron (UMN)**: cell body and dendrites of the upper motor neuron is found in the primary motor cortex in the precentral gyrus.

Neuron #2: **Lower motor neuron (LMN)**
The **cell body and dendrites** of lower motor neurons are found in the nuclei of brainstem and spinal cord. The axon of the lower motor neuron exits the CNS to form either cranial nerves (face) or spinal nerves (body).

Appendix 7

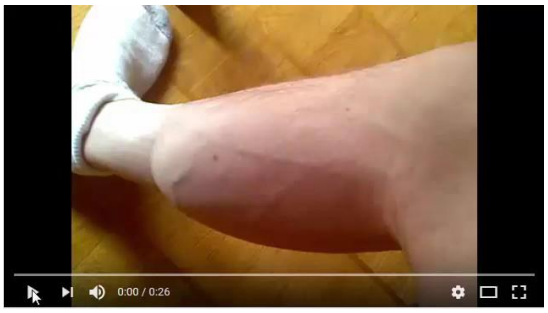
Weakness: Abnormality of movement as strength



- Damage to lower motor neurons (**LMN**s) produces **flaccid paralysis** or weakness
  - Decreased reflexes and tone (hyporeflexia, hypotonicity)
  - + Atrophy
  - + Fasciculations (twitching)
  - LMN:
    - Anterior horn cells
    - Ventral root
    - Peripheral nerves
    - NMJ
    - Muscle

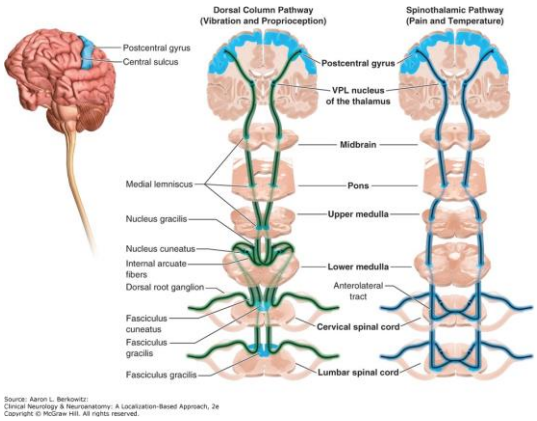
Weakness: Abnormality of movement as strength

- Damage to lower motor neurons (**LMN**s) produces **flaccid paralysis** or weakness
  - Decreased reflexes and tone (hyporeflexia, hypotonicity)
  - + Atrophy
  - + Fasciculations (twitching)

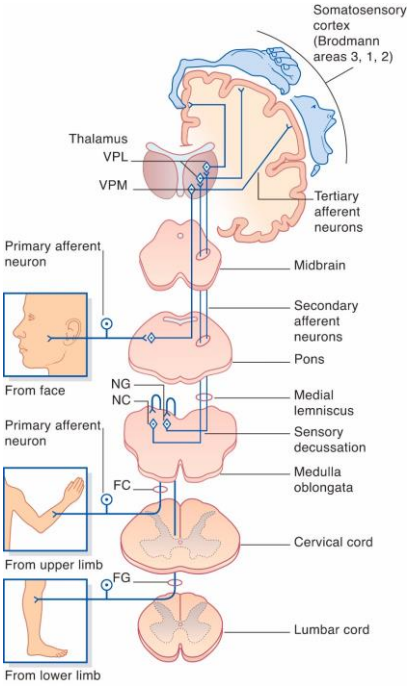


Appendix 7

Perception: Somatic sensation



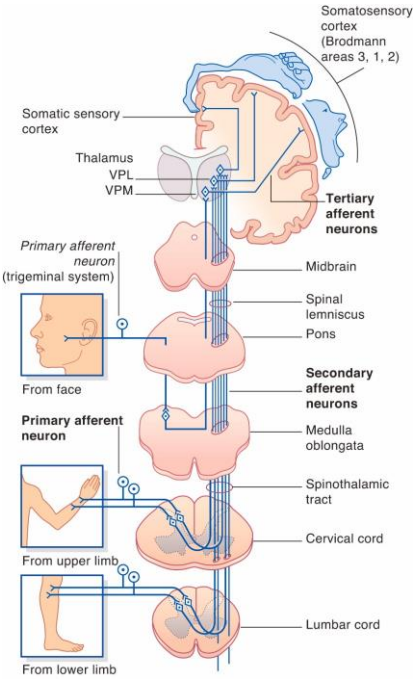
- **Dorsal column pathway** fine touch (vibration, proprioception)
- **Spinothalamic pathway** pain and temperature
- 3 neuron *ascending* pathway!!!



Dorsal column pathway
FT: Vibration, Proprioception

- Neuron #1
  - Peripheral nerves
  - Dorsal roots and DRG
  - Fasciculus gracilis & cuneatus
- Neuron #2
  - Cell bodies in nucleus gracilis and cuneatus
  - Internal arcuate fibers *decussate* in **medulla**
  - Ascend in contralateral brainstem as fibers of medial lemniscus
- Neuron #3
  - Cell bodies in thalamus that sends fibers to the somatosensory cortex

Appendix 7

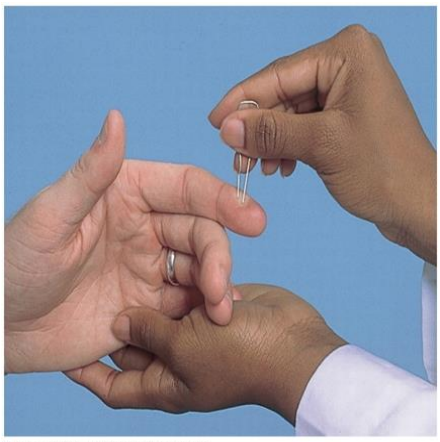


Spinothalamic pathway

Pain and Temperature

- Neuron #1
  - Peripheral nerves
  - Dorsal roots and DRG
  - Dorsal horn
- Neuron #2
  - Cross in the anterior commissure
  - Ascend in contralateral spinal cord as spinothalamic tract
- Neuron #3
  - Cell bodies in thalamus
  - To somatosensory cortex

Physical

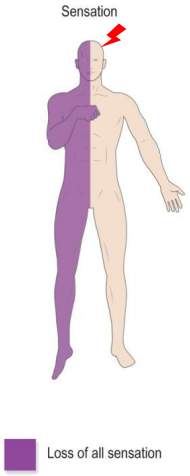


Sensory Exam

- **Sensory tract function**
  - Loss of fine touch & proprioception vs.
  - Loss of pain & temperature sensation
  - Complete anesthesia

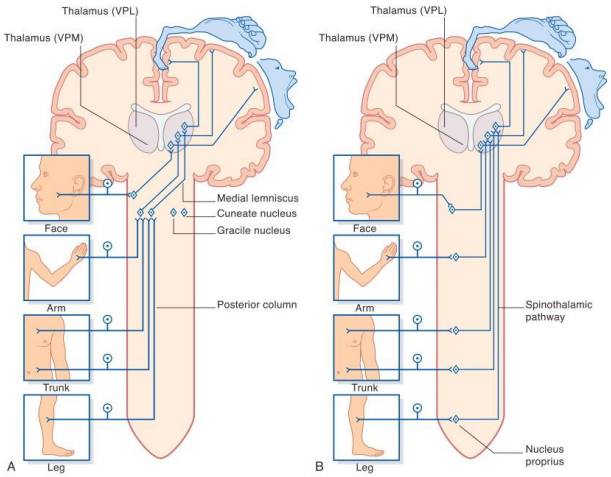
Appendix 7

Abnormalities of sensation



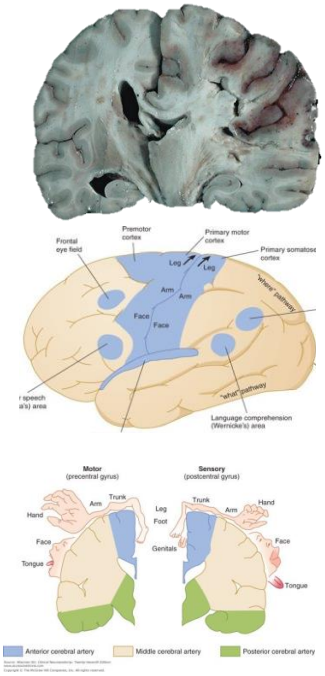
- Damage to **postcentral gyrus**
- Contralateral loss of sensation
  - No pain or temperature
  - No vibration of proprioception

Abnormalities of sensation



- Damage to **postcentral gyrus**
- Contralateral loss of sensation
  - No pain or temperature
  - No vibration of proprioception

Appendix 7

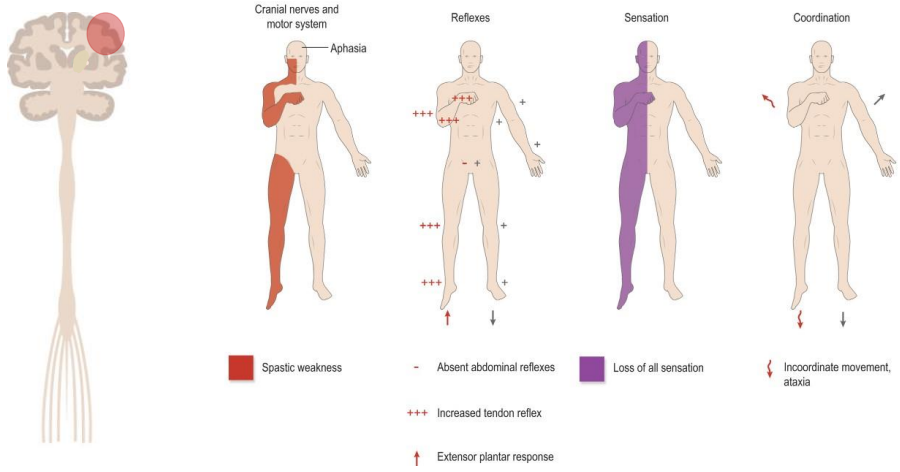


Cortex lesion: Stroke

Contralateral UMN weakness (spastic paralysis)

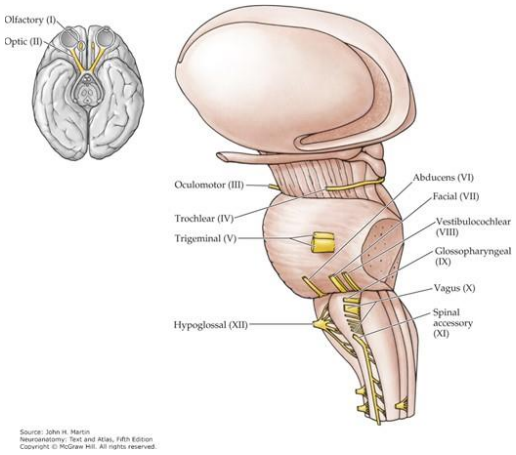
Contralateral loss of sensation in all modalities

Cortex lesion: Stroke

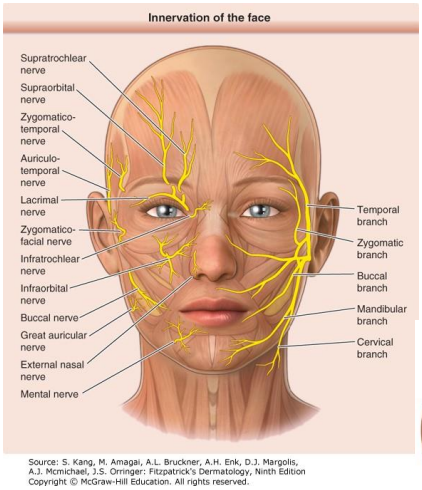


Appendix 7

Corticobulbar tracts



- Midbrain 1* 2 3 4
- Pons 5* 6 7* 8*
- Medulla 9 10 11* 12



Source: S. Kang, M. Amagai, A.L. Bruckner, A.H. Enk, D.J. Margolis, A.J. Mcmichael, J.S. Orringer: Fitzpatrick's Dermatology, Ninth Edition
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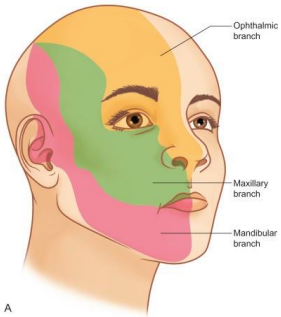
Corticobulbar tracts

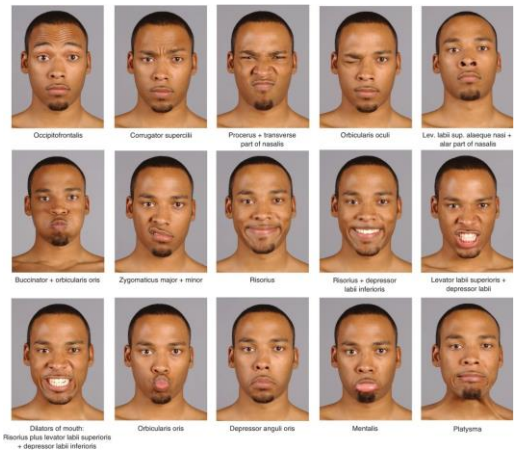
- CN VII Facial nerve
  - Facial expression muscles



- CN V Trigeminal nerve
  - Facial sensation

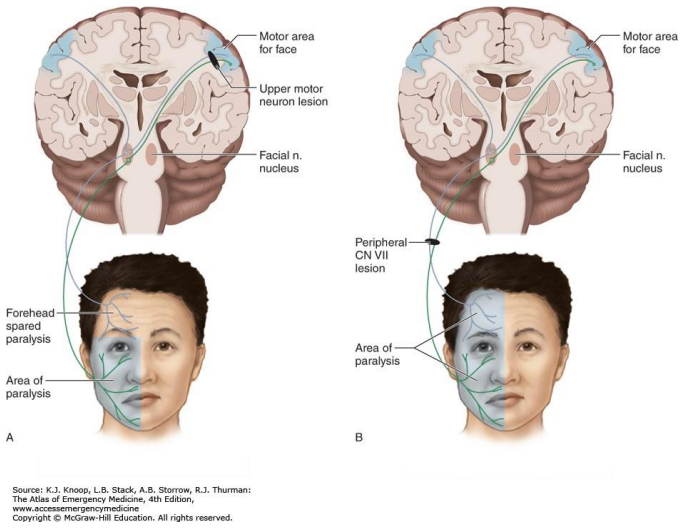
Appendix 7

Corticobulbar tracts



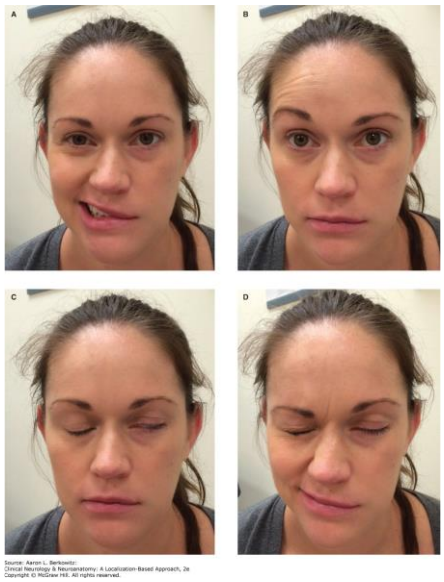
- CN VII
- Facial expression muscles
  - To – temporal
  - Zanzibar – Zygomatic
  - By – Buccal
  - Motor – Mandibular
  - Car - Cervical

LMN V. UMN



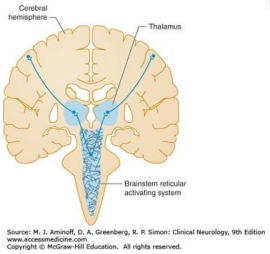
Appendix 7

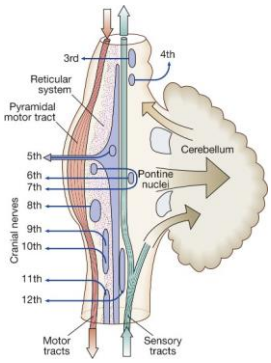
Bells Palsy



- Inflammation of CN VII (Facial nerve)
- Unilateral weakness of the facial muscles of expression unilateral
- Change in taste sensation
- Hyperacusis (sensitivity to sound)
- Corneal dryness

Brainstem



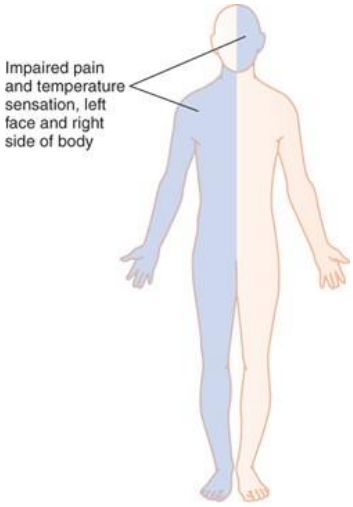


- Nuclei of some cranial nerves
- Long tracts
  - Somatosensory
  - Motor
- Reticular system
- Autonomics

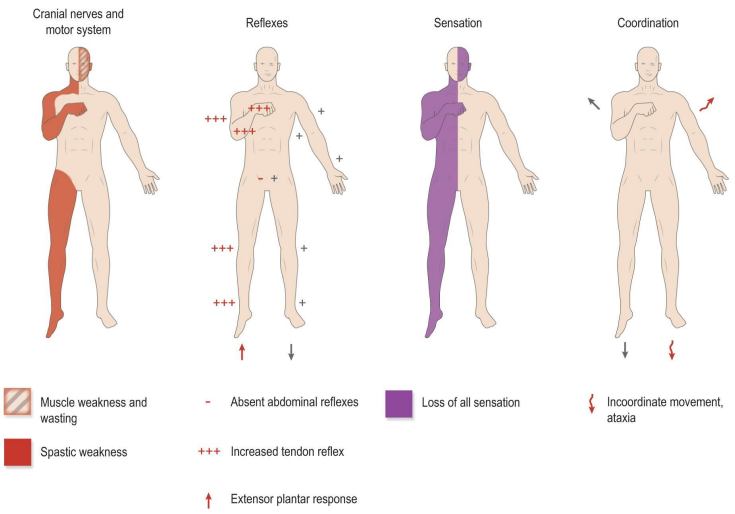
Appendix 7

Brainstem

- **Alternating or Crossed** signs:
- Long tracts cross
- Cranial nerve generally do not
- Brainstem lesions often produce symptoms on one side of the face and the opposite side of the body

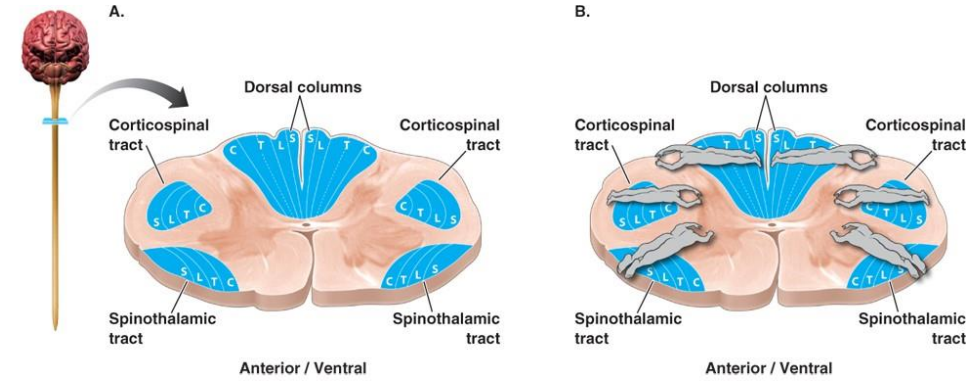


Brainstem lesion



Appendix 7

Spinal cord lesions



Spinal cord lesions

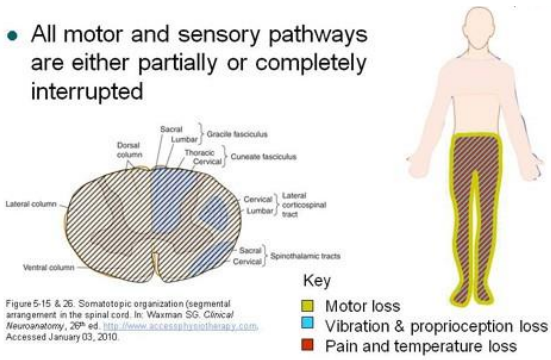
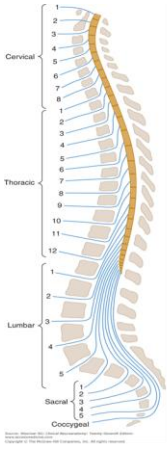


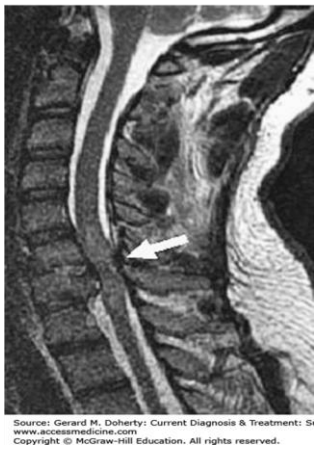
Figure 5-15 & 26. Somatotopic organization (segmental arrangement in the spinal cord. In: Waxman SG. *Clinical Neuroanatomy*, 26th ed. http://www.accessphysiotherapy.com. Accessed January 03, 2010.

- **Weakness** – Spastic paralysis
  - Distal, symmetric
  - Except Brown-Sequard type lesions
- **Dysthesias**
  - **Sensory level**
    - Sharp line or band (on the trunk or abdomen) below which there is a decrease in sensation.
    - **Pathognomonic** for spinal cord disease.
  - Bladder and bowel problems

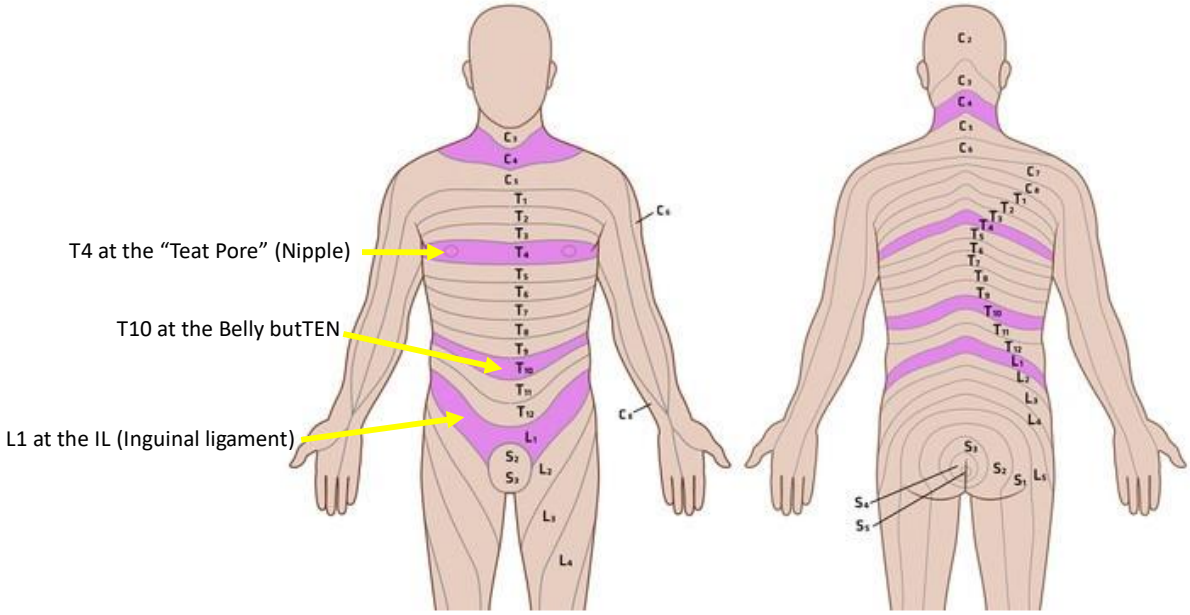
Appendix 7

Spinal Cord Injuries



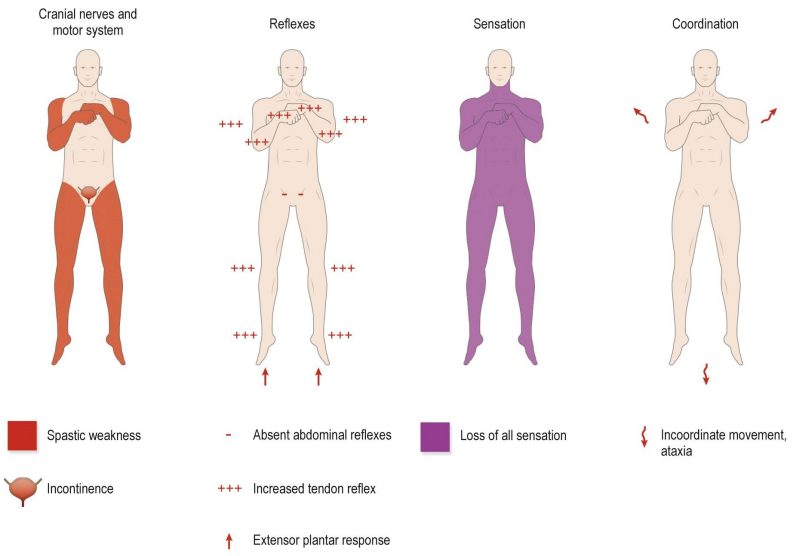


- **<u>Complete</u>**
- Complete loss of function below the level of injury
  - Anesthesia
  - Paralysis
- **<u>Level of injury</u>**
- ↑C6 (C3,4,5) = Respiratory dysfunction
- C6 and below= Quadriplegia
- T1 or below = Paraplegia

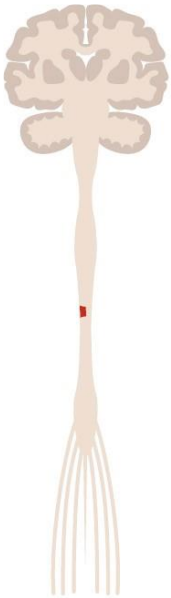


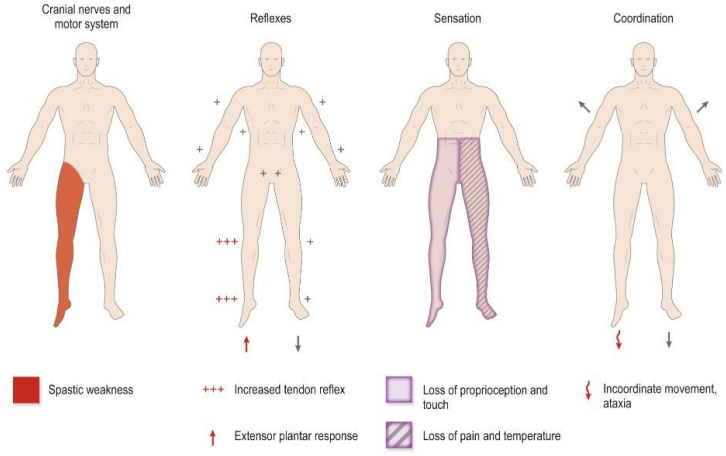
Appendix 7

High cervical cord lesion



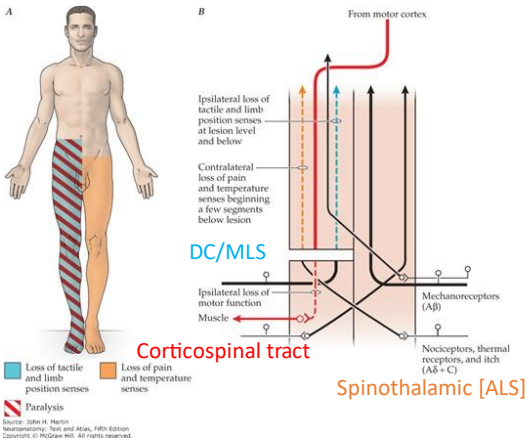
Hemilesion of the thoracic spinal cord





Appendix 7

Hemilesion of the thoracic spinal cord



- Brown–Séquard syndrome

Central Nervous System Deficit Summary

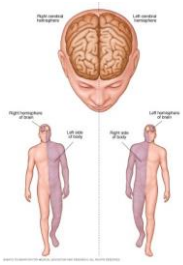
Cortical Brain

Same side
- Contralateral **Weakness** (Spastic paralysis)
- Contralateral **Dysthesias** (PCML/ST)

Face = Arm > Leg or Leg

+/- higher function deficits

+/- visual field deficits



Brain stem

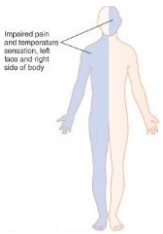
Weakness (Spastic paralysis)

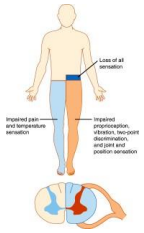
Dysthesias

Crossed (Alternating) deficits

Cranial nerve deficits

Coma & Cardiopulmonary dysfunction ?





Spinal Cord

Weakness (Spastic paralysis*)

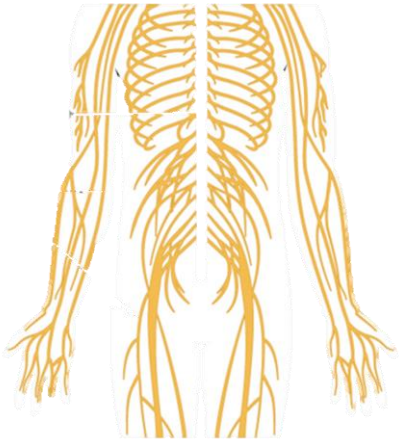
Dysthesias/Anesthesia

Sensory level

Bladder/bowel dysfunction

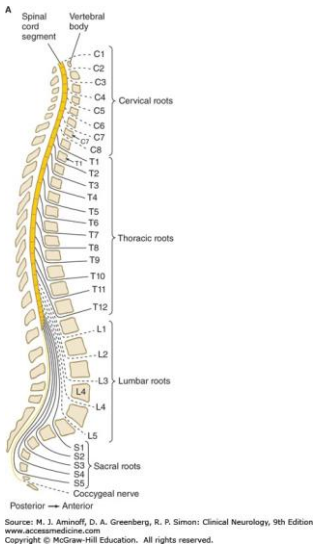
Appendix 7

Motor and Sensory Deficits



- Peripheral
  - Radiculopathy
  - Neuropathy
  - NMJ disease
  - Muscular disease

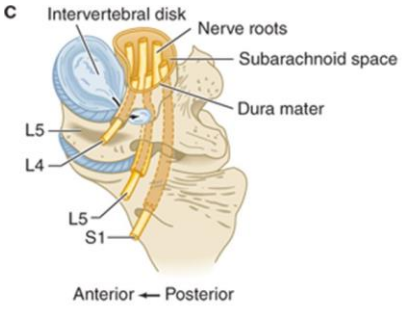
Nerve root



- **Radicular pain**
- Asymmetric weakness, denervation (atrophy and fasciculations)
- Sensory loss (Like PN except radiating pain).
- Weakness is distal or proximal depending on which nerve root is involved.

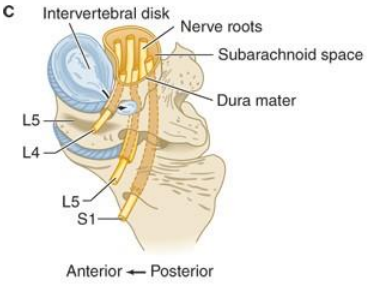
Appendix 7

Nerve root



- Pain – Radicular pain may worsen with stretch of nerve root – rotating neck in C6, or straight leg raise in L5
- Asymmetric weakness - confined to **myotomal** group – e.g C6 or L5
- Sensory loss occurs in **dermatomal** distribution
- Denervation signs – Atrophy and Fasciculations
- Reflexes may be diminished or absent

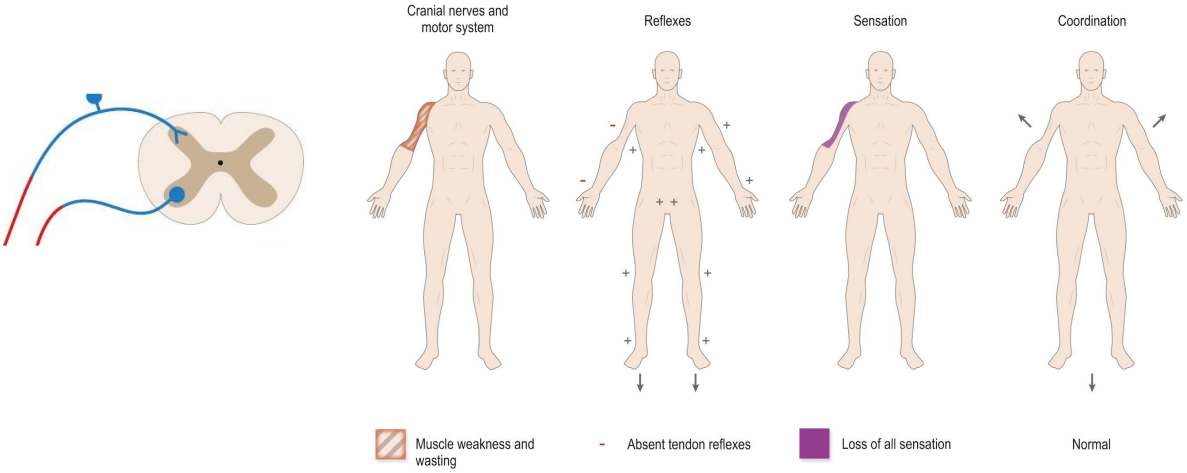
Nerve root



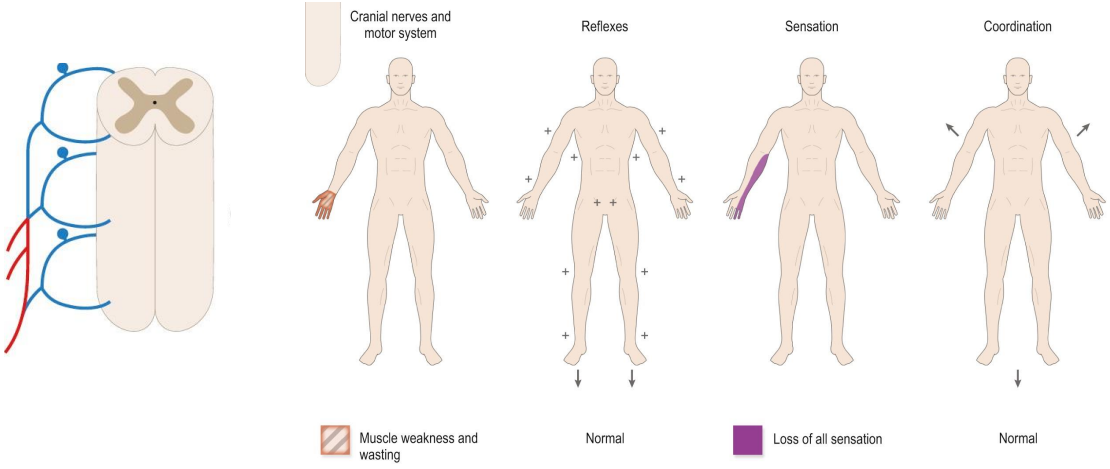
- Lumbar – **L5-S1** nerve root **most common** radiculopathy in legs causing distal weakness
- Cervical – **C5-C6** nerve root **most common** radiculopathy in arms which innervate proximal weakness

Appendix 7

Spinal nerve root lesion at the level of C5

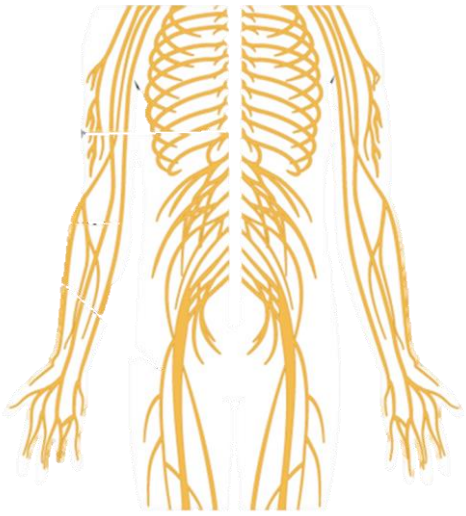


Lesion of the brachial plexus (lower cord C–T)



Appendix 7

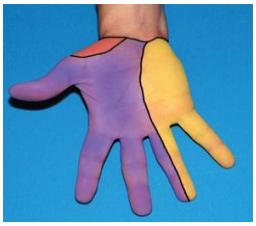
Peripheral Nerve



- Asymmetric
- Symmetric
  - Metabolic (EtOH, DM)
- Accompanied by atrophy, fasciculations
- Almost always associated with sensory changes.

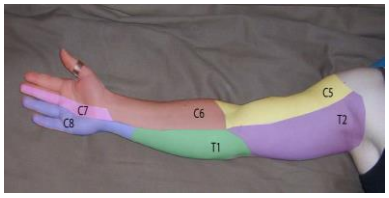
Median Nerve Versus C6 Radiculopathy

Median Nerve



Anatomically distinct area
Distal
Correlates with overuse injury of wrist
No neck pain
Reflexes intact
EMG & NCS = Peripheral Neuropathy

C6 Radiculopathy

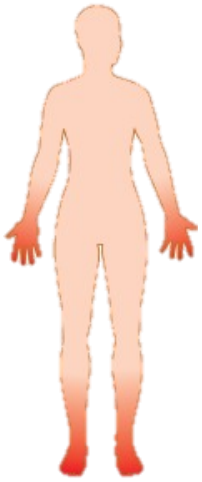


Myotome and Dermatome
Neck to index finger and thumb
Symptoms worse with movement of neck
Radicular Neck pain
Reflexes may be diminished
EMG & NCS = Radiculopathy

Appendix 7

Peripheral Nerve

- Symmetric
  - Metabolic (EtOH, DM)
  - Toxic
  - Infectious



Peripheral Nerve Disease
"Dang Therapist"!

DANG

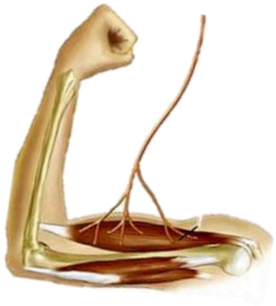
- **D**iabetes
- **A**lcohol
- **N**utritional
- **G**uillain-Barre

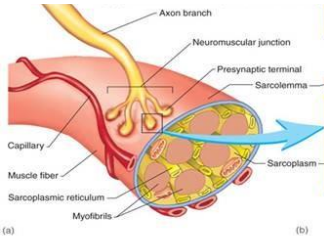
THERAPIST

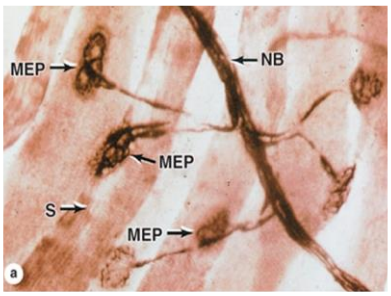
Trauma
Hereditary
Environmental toxins and drugs
Rheumatic (Collagen vascular)
Amyloid
Paraneoplastic
Infections
Systemic disease
Tumors

Appendix 7

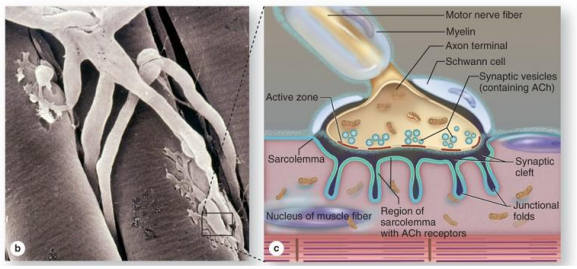
Neuromuscular Junction Disease







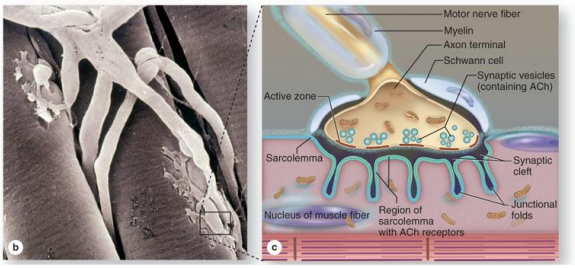
Neuromuscular Junction Disease



- **Fatigability**
- Weakness of muscles with repetitive movements
- Proximal muscles
  - Muscles of face, eyes (ptosis), jaw
- Improves or resolves with rest
- Myasthenia Gravis
- LEMS

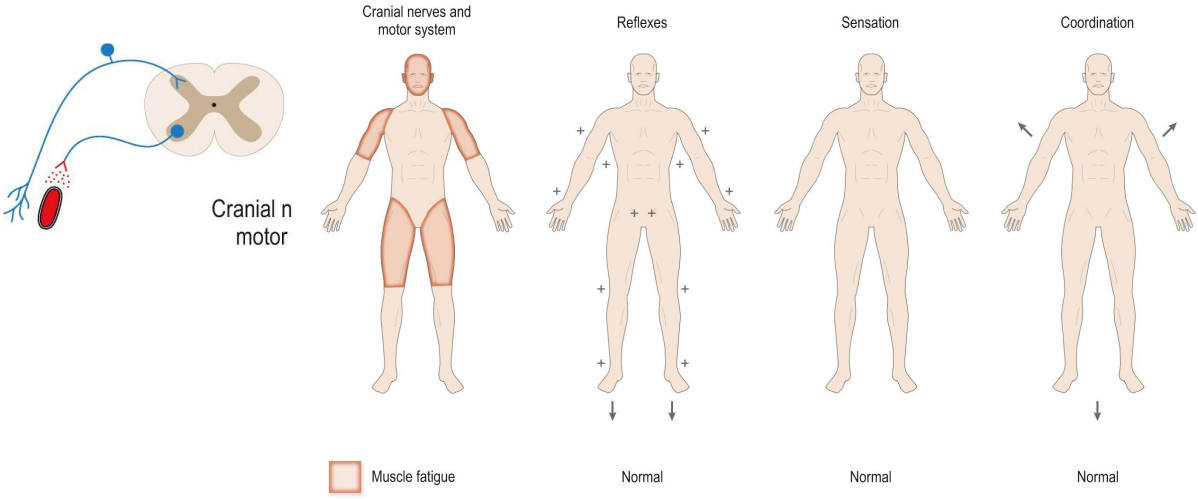
Appendix 7

Neuromuscular Junction Disease



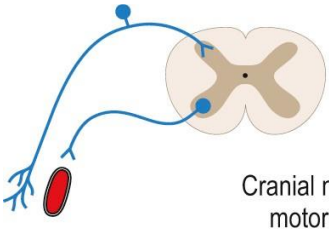
- Exam
- Weakness of muscles with repetitive movements.
- Muscular size normal without atrophy, fasciculations, sensory loss
- Muscle tone and reflexes are normal

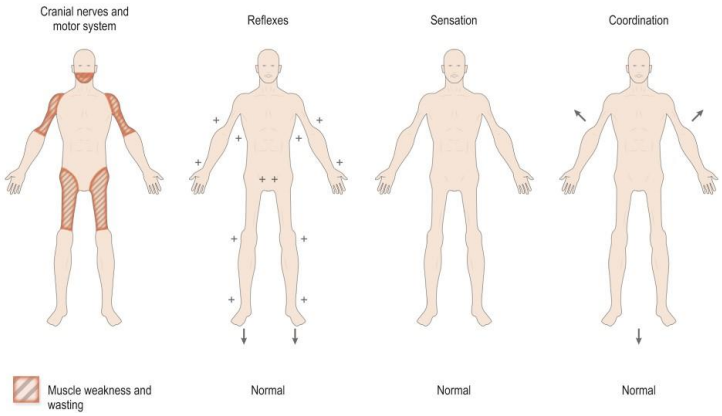
Neuromuscular junction syndrome



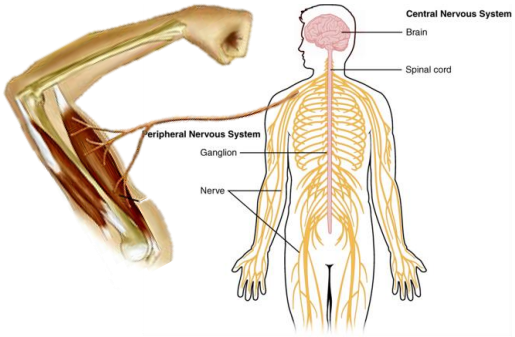
Appendix 7

Myopathy





Muscle Disease (Myopathy)



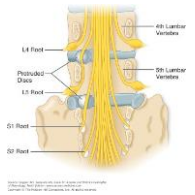
- **Weakness**
  - **Proximal > distal**
  - **NO sensory changes**
- **Muscular dystrophy**
- Myopathy
- Can pt. carry objects like grocery/garbage bags, small children, books?
- Can pt. get out of car, off toilet, up from chair without using hands?

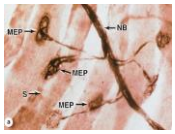
Appendix 7

Peripheral Nervous System Deficit Summary

Nerve Root

Segmental Weakness (myotome)
+Segmental dysthesias (dermatome)
+**Pain (radicular)**





Neuromuscular Junction

Weakness
Diffuse & **Fatigable**
NO sensory loss

Peripheral Nerve

Weakness
Distal > Proximal
+ **Dysthesias**

Muscular disease

Weakness
Proximal >Distal
No sensory loss

Sources

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Appendix 8

Cranial Nerves & Brainstem

Perdue

Session Objectives

1. Identify the 12 cranial nerves by name, Roman numeral, and their anatomical relationships.

2. Trace the course of each nerve as it passes from the brain/brainstem through foramina of the skull.

3. Identify the different regions of the brainstem and locate the cranial nerves and their nuclei associated with each.

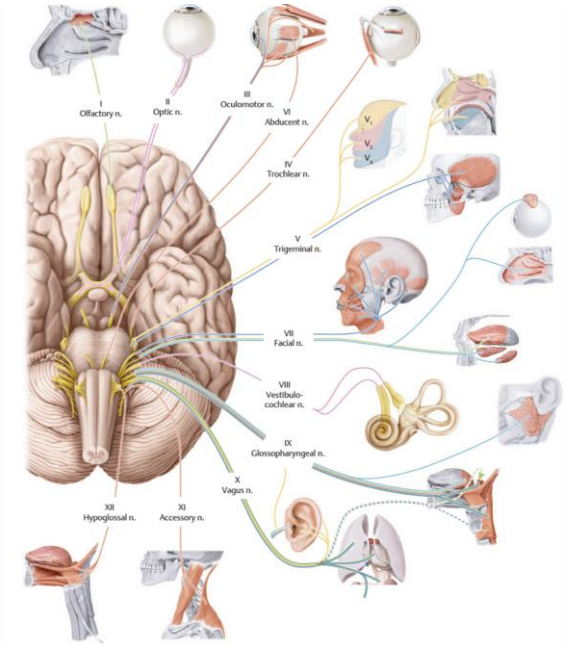
4. Describe the various functions and general distribution of each of the cranial nerves.

5. Describe the clinical deficits associated with lesions of each of the cranial nerves.

Appendix 8

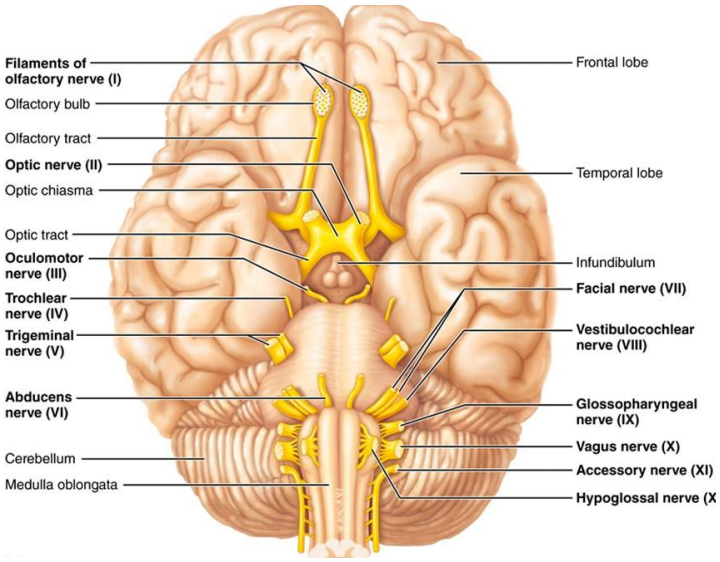
Cranial nerves

- Nerves that emerge from brain or brainstem
- One to many functions
- UMN v. LMN
- Stroke Localization
  - Cortex
  - Brainstem



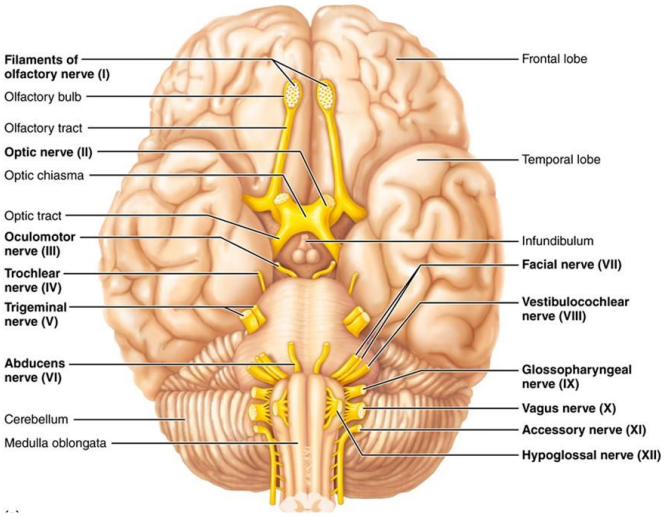
Cranial nerves

Cranial nerves are numbered in the order they have to be cut to get the brain out!



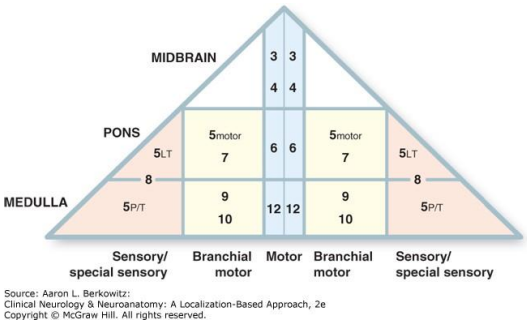
Appendix 8

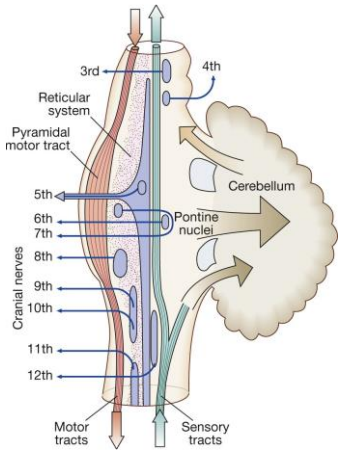
Surface location on brainstem



- Midbrain ~~1~~ 2 3 4
- Pons 5 6 7 8
- Medulla 9 10 ~~11~~ 12

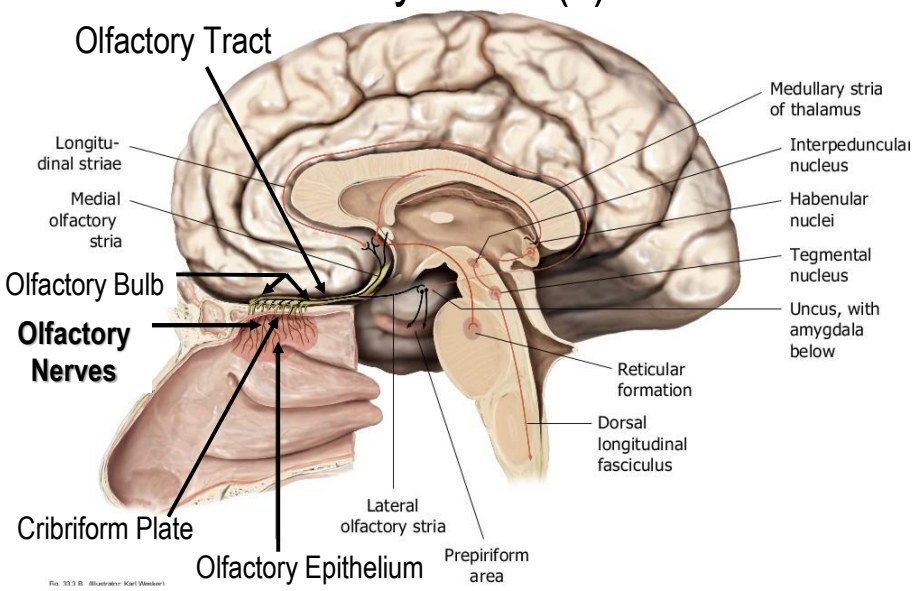
Location of the **nuclei** in brainstem





Appendix 8

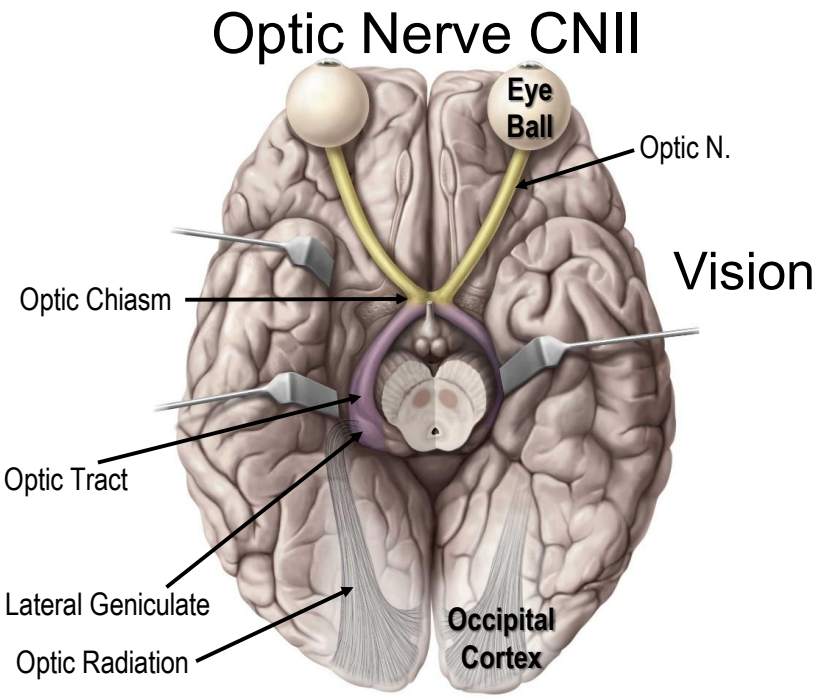
Olfactory Nerve(s) - CNI

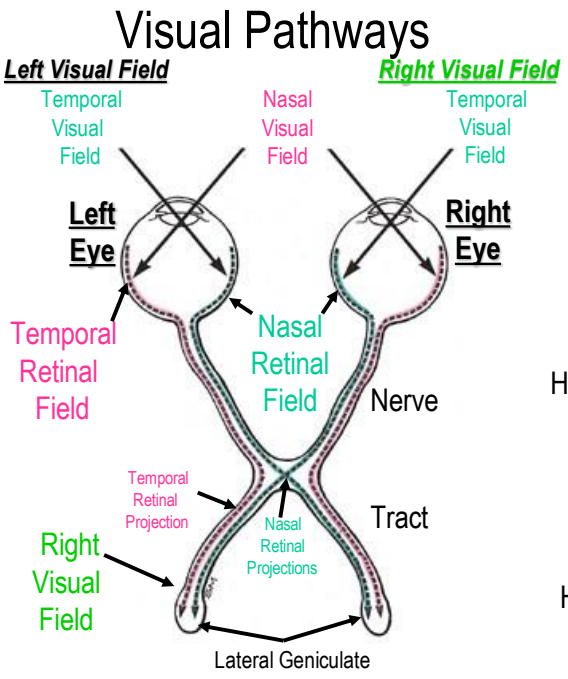


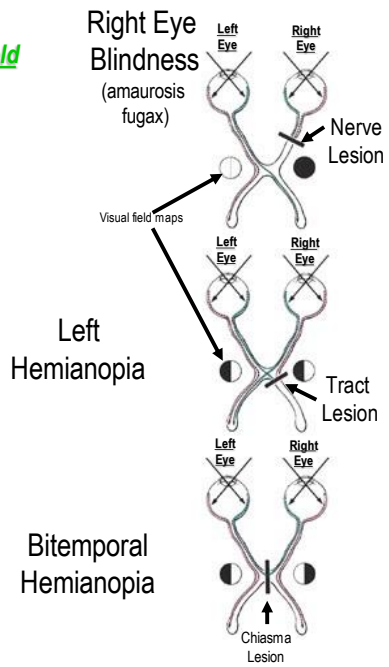
Lesion of the Olfactory Nerves

- Viral infection
  - Loss of Smell
  - Food Tastes funny
  - COVID
- History of trauma?
  - Nose is running (CSF?)

Appendix 8


Optic Nerve CNII
Eye Ball
Optic N.
Vision
Optic Chiasm
Optic Tract
Lateral Geniculate
Optic Radiation
Occipital Cortex

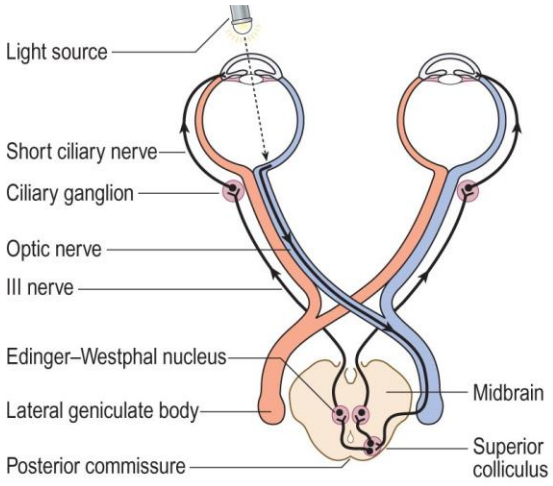

Visual Pathways
Left Visual Field
Right Visual Field
Temporal Visual Field
Nasal Visual Field
Temporal Visual Field
Left Eye
Right Eye
Temporal Retinal Field
Nasal Retinal Field
Nerve
Temporal Retinal Projection
Nasal Retinal Projections
Tract
Right Visual Field
Lateral Geniculate


Right Eye Blindness
(amaurosis fugax)
Left Eye
Right Eye
Nerve Lesion
Visual field maps
Left Hemianopia
Tract Lesion
Bitemporal Hemianopia
Chiasma Lesion

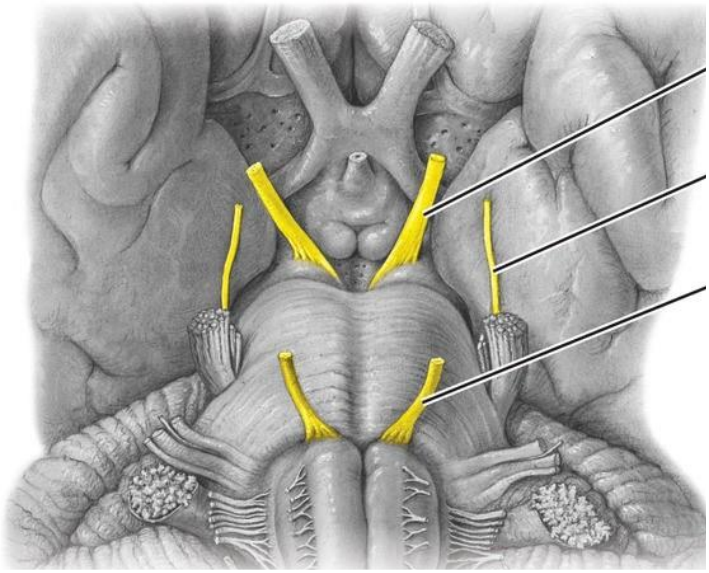
Appendix 8

CN II Afferent

- Pupillary Reaction: Size changes in response to light and effort of focusing on a near object
  - Pupil Light reaction:
    - Cranial nerve II afferent limb of the reflex (sensory)
    - Cranial nerve III efferent limb of the reflex (motor)
    - A light shining into one eye causes bilateral pupil constriction.
      - Direct reaction for the eye the light was shined in and
      - Consensual reaction for the opposite eye.
  - Miosis – pupil constriction (parasympathetic)
  - Mydriasis – pupil dilation (sympathetic)
    - D for dilation because of fear of the MY BIG DOG! (or danger)



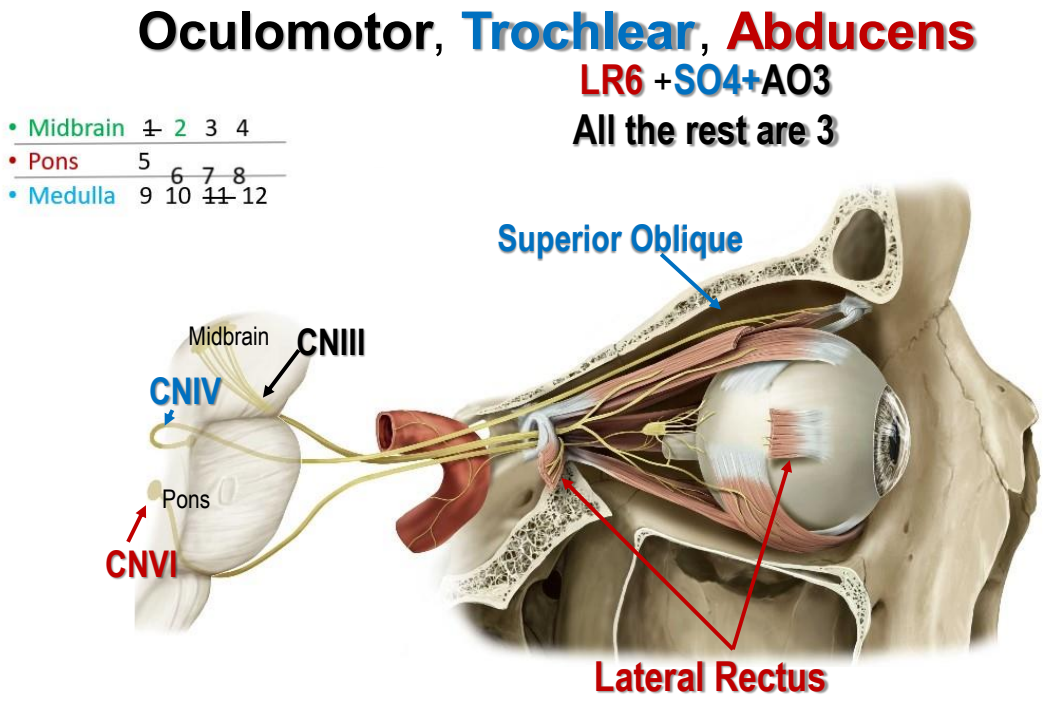
CNIII, CNIV, and CNVI



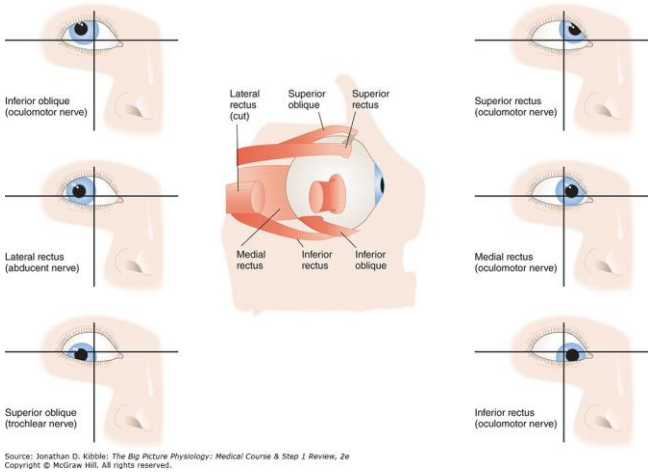
LR6 – SO4
All the rest are 3

Extraocular (eye) movements

Appendix 8

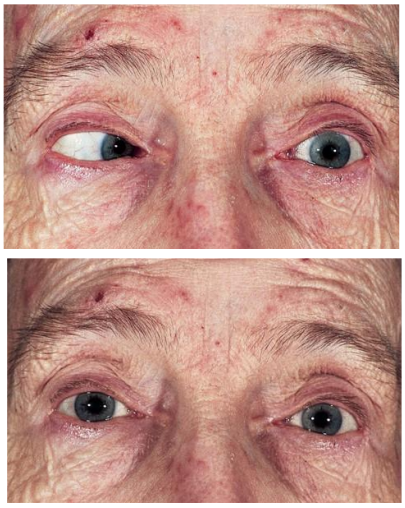


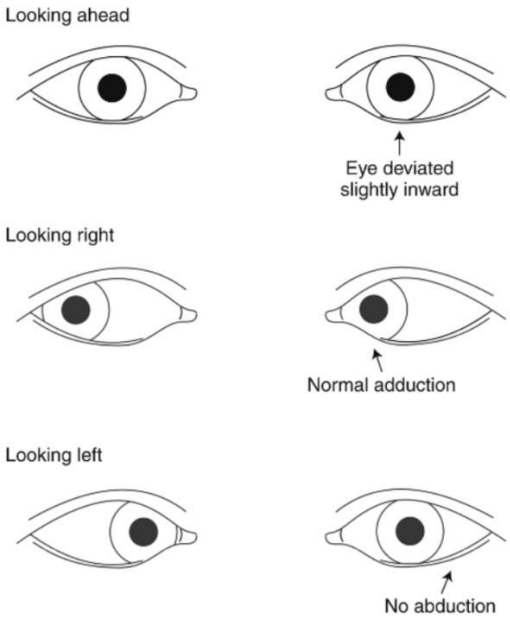
Extraocular Muscles



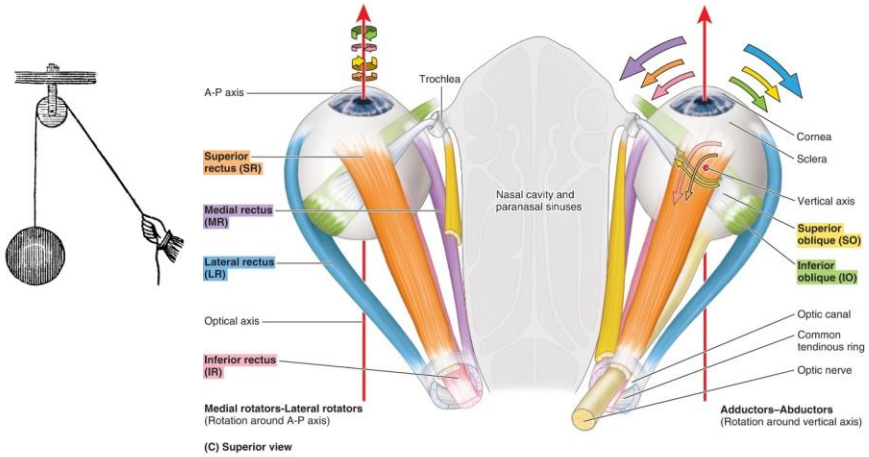
Appendix 8

CN VI
Abducens Palsy





CN IV
Trochlear Nerve Palsy

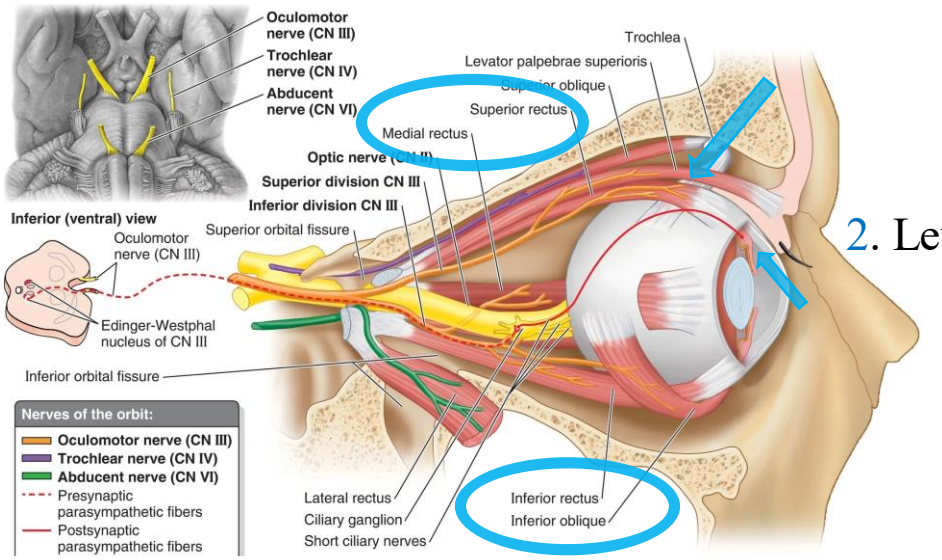


Appendix 8

A

B

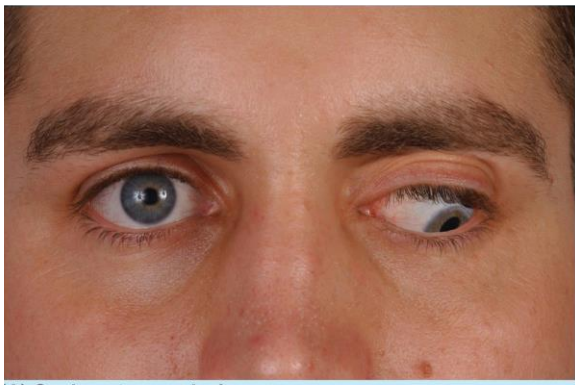
Oculomotor Nerve CN III → 3 Things Nerve

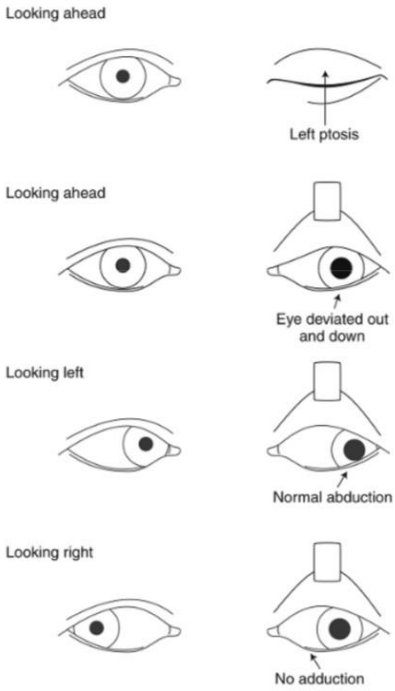


1. "ATR 3"
2. Levator palpebrae
3. Pupil constrictors

Appendix 8

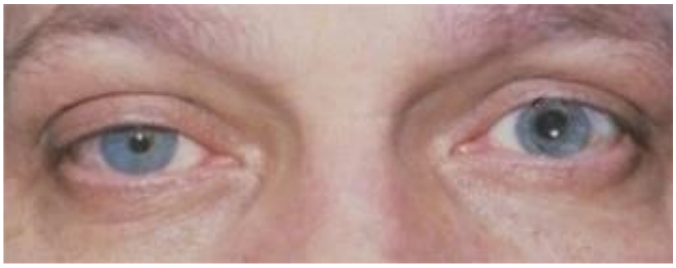
CN III Oculomotor Nerve Palsy

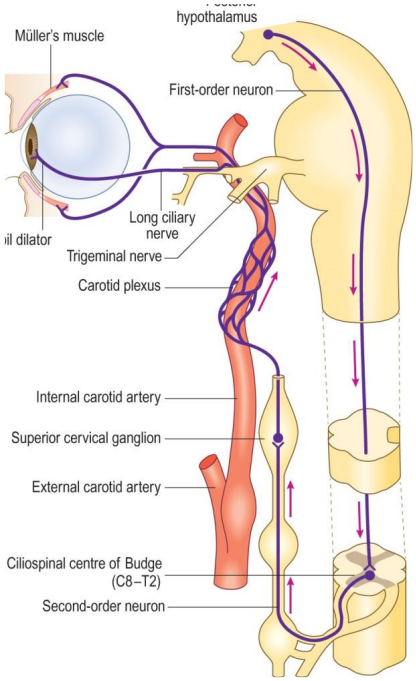




Horner's Syndrome

P	**Ptosis**
A	**Anhidrosis**
M	**Miosis**

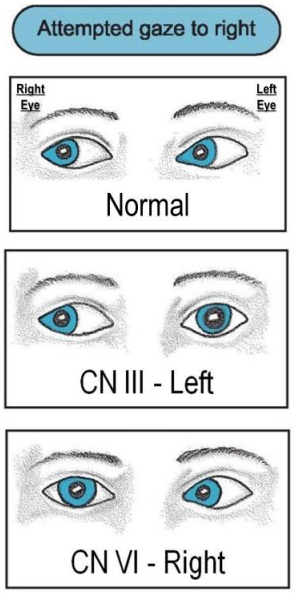


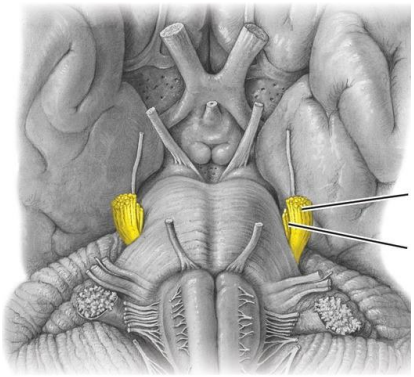


Appendix 8

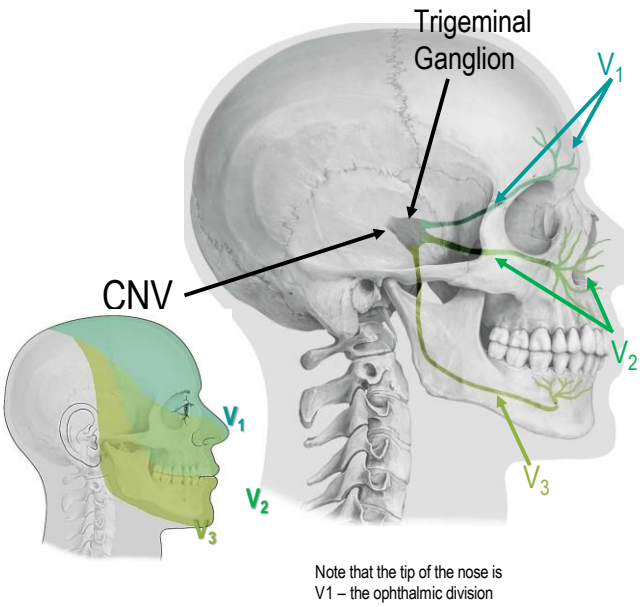
Lesions of CN III, IV and VI

- There may be diplopia with certain eye movements
- Patients may say they are dizzy
- If the patient cannot look in and the pupil is dilated, **think III**
- If they get diplopia with out and down, **think IV**
- If they cannot look out, **think VI**





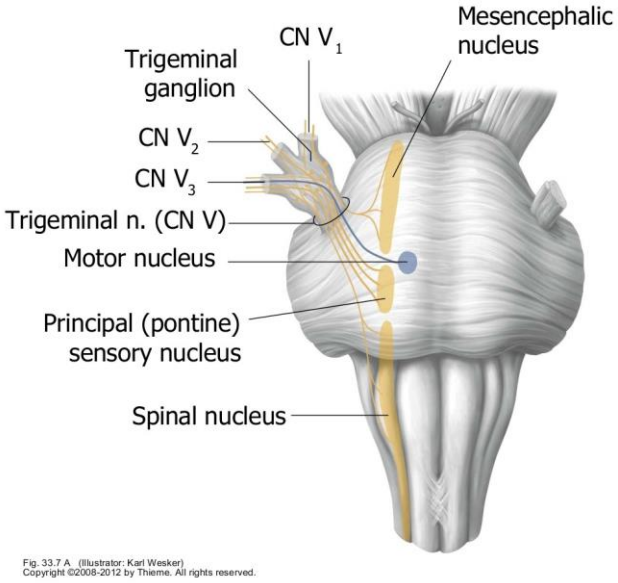
Trigeminal Nerve CN V



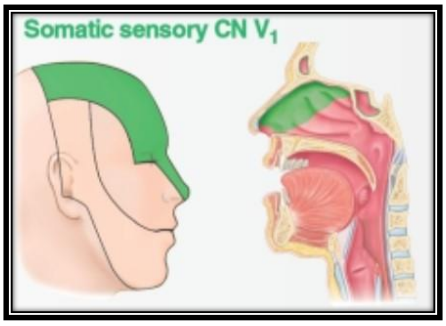
Note that the tip of the nose is V1 – the ophthalmic division

Appendix 8

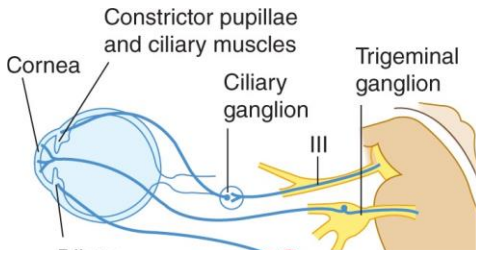
Trigeminal Nerve CN V

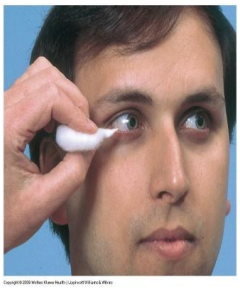


Trigeminal Nerve CN V



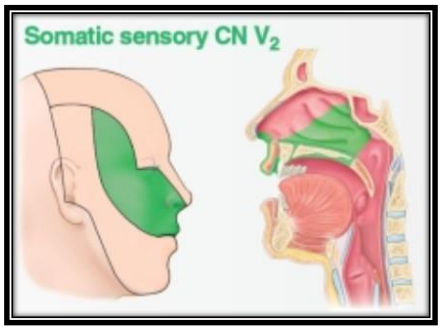
- **CN V 3 + 2 = 5**
  - **(3) Facial somatic sensation**
    - **Ophthalmic**
      - » Corneal blink reflex





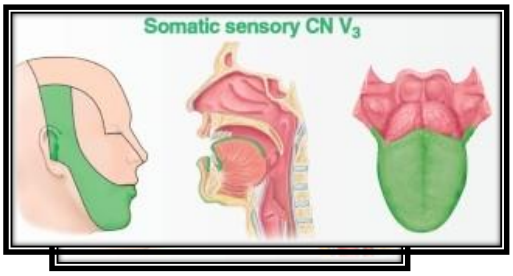
Appendix 8

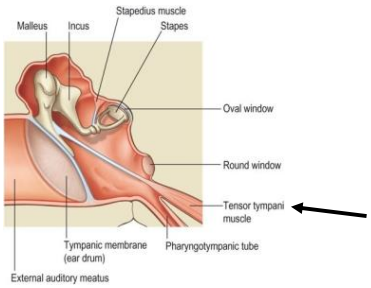
Trigeminal Nerve CN V



- **CN V 3 + 2 = 5**
  - **(3) Facial somatic sensation**
    - –Ophthalmic
    - –**Maxillary**
    - –Mandibular

Trigeminal Nerve CN V



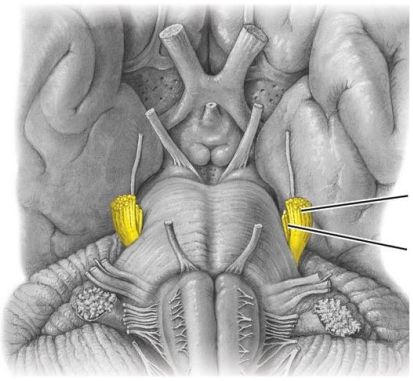


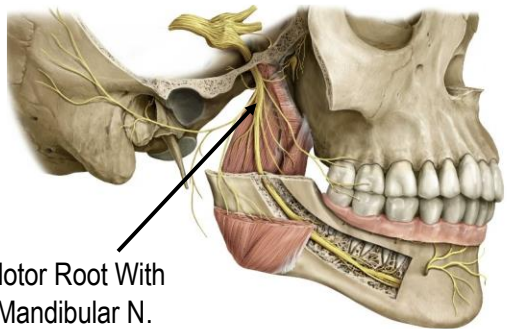
- **CN V 3 + 2 = 5**
  - (3) Facial somatic sensation
    - – Ophthalmic – including sensation in corneal blink reflex
    - – Maxillary
    - – Mandibular
  - **Muscles of mastication**
  - **Special sense organs: Tongue and Ear**
    - – ***Somatic*** sensation of anterior 2/3rds
    - – Tensor tympani – activation dampens loud sound

Appendix 8

Trigeminal Nerve, Motor Component

CN V





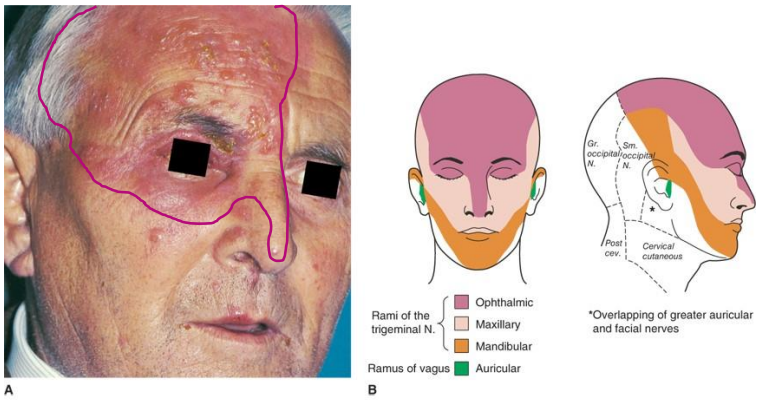
Weakness in muscles
Of Mastication – jaw clench

Problems with the Trigeminal

- Loss of muscle tone on the side of the lesion
- Wasting of the Muscles of Mastication
- When asked to open the jaw, the mandible deviates to the side of the lesion
- Anesthesia over head – distribution depends on part of the nerve that is interrupted
- Trigeminal Neuralgia (Tic de le Rue)
- Herpes Zoster Ophthalmicus

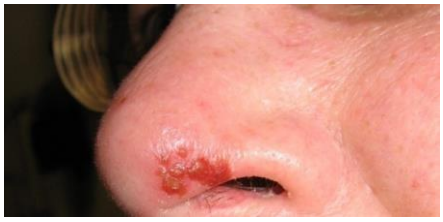
Appendix 8

Herpes Zoster Ophthalmicus

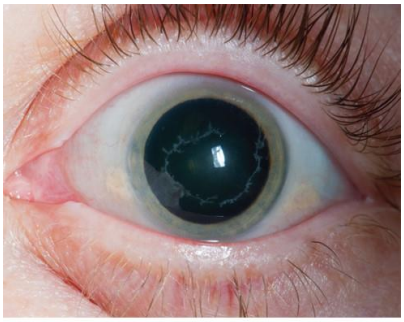


Herpes Zoster Ophthalmicus

Hutchinson's sign

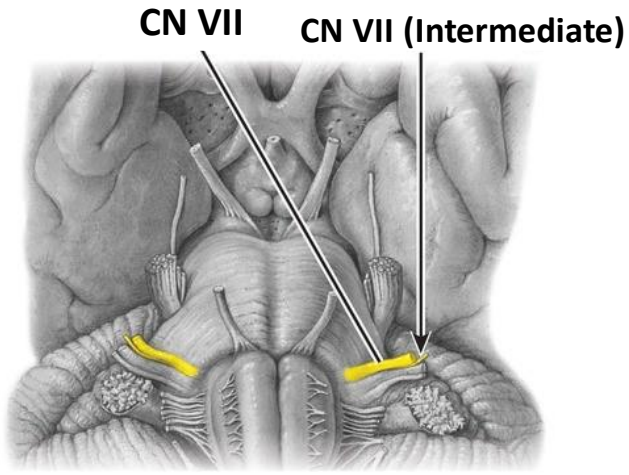


Dendrite

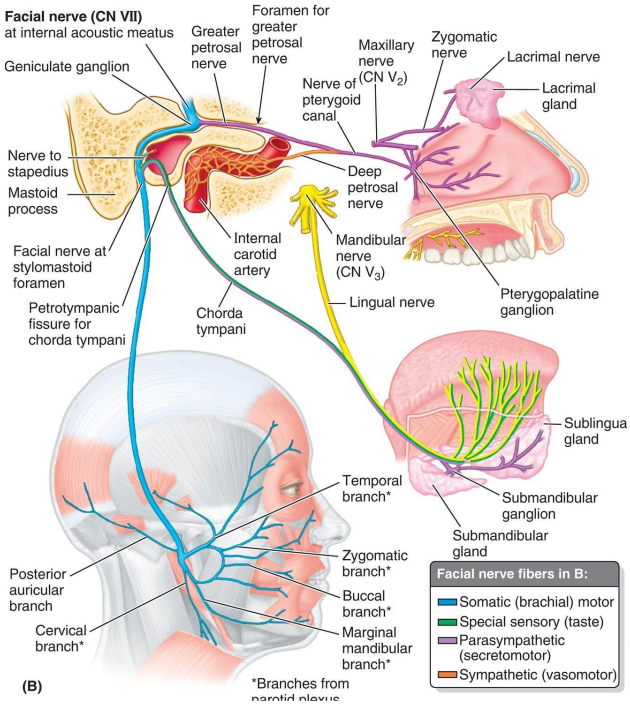


Appendix 8

Facial Nerve CN VII



- Emerges from the junction of the pons and medulla as two divisions:
- Primary root
  - Larger primary root (facial nerve proper) innervates the muscles of facial expression
- Intermediate nerve
  - Smaller nerve carries taste, parasympathetic, and somatic sensory fibers.

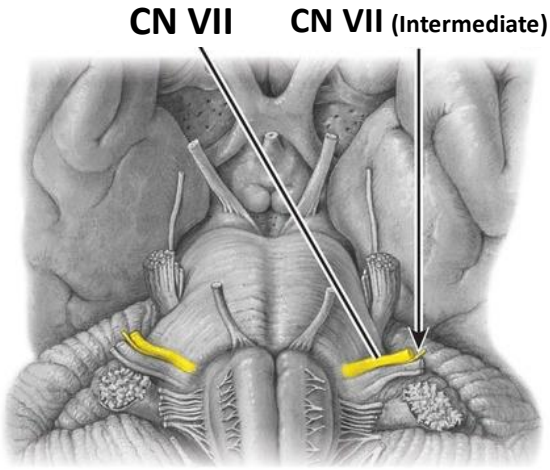


Facial Nerve CN VII

- Course
  - Internal acoustic meatus
  - Temporal bone
  - Stylomastoid foramen
  - Parotid gland

Appendix 8

Facial Nerve CN VII



CN VII 5 + 2 = 7

- **+ 5 motor branches** facial expression
- **+ Autonomic "moisture makers" of the face**
- **+ Special sense organs: Tongue and Ear**

Facial Nerve CN VII

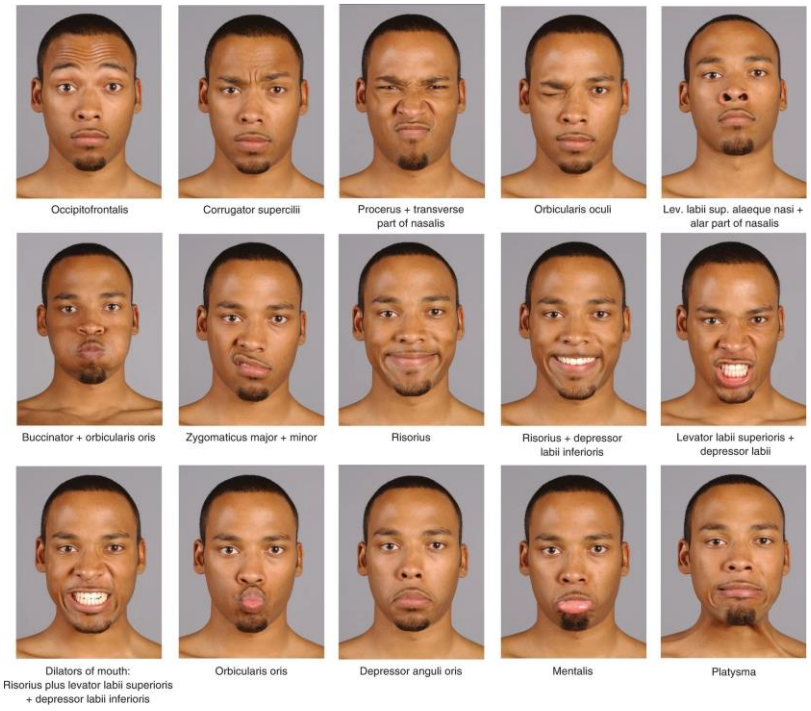
CN VII 5 + 2 = 7

- **+ 5 motor branches** - muscles of facial expression

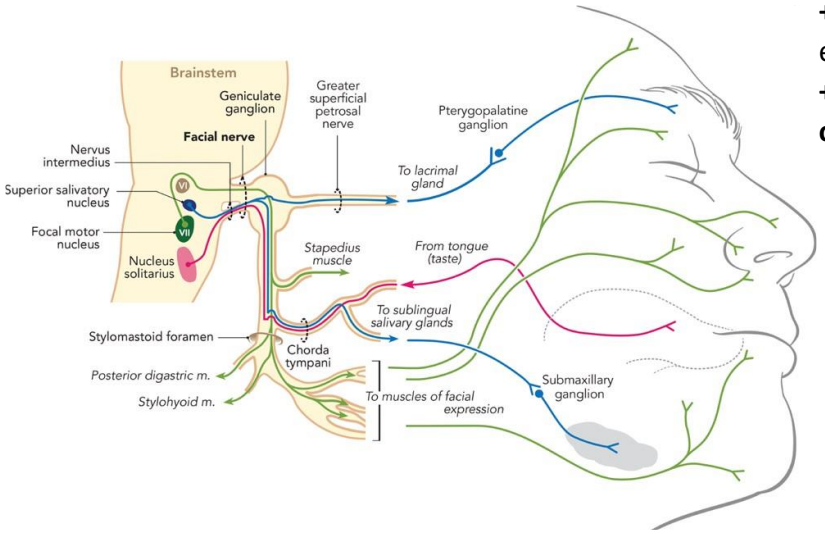
To – temporal
Zanzibar – Zygomatic
By – Buccal
Motor – Mandibular
Car - Cervical

Distribution of terminal branches of CN VII

Appendix 8



Facial Nerve CN VII



CN VII 5 + 2 = 7

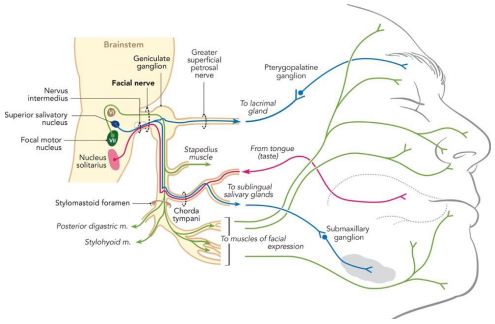
+ 5 motor branches facial expression

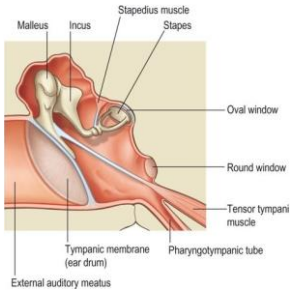
+ Autonomic "moisture makers" of the face

- Lacrimal gland
- Nasal gland
- Sublingual & Submandibular glands
- (NOT parotid →CN IX)

Appendix 8

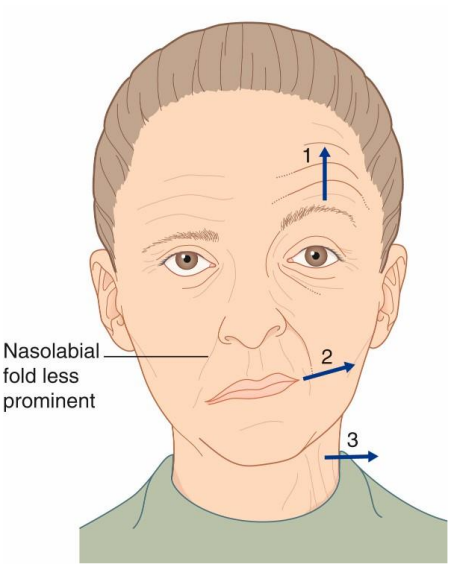
Facial Nerve CN VII





CN VII 5 + 2 = 7

- + 5 motor branches facial expression
- + Autonomic "moisture makers" of the face
  - Lacrimal gland
  - Nasal gland
  - Sublingual & Submandibular glands
- **+ Special sense organs: Tongue and Ear**
  - Taste anterior 2/3rd of tongue
  - Stapedius – dampens loud sound - hyperacusis

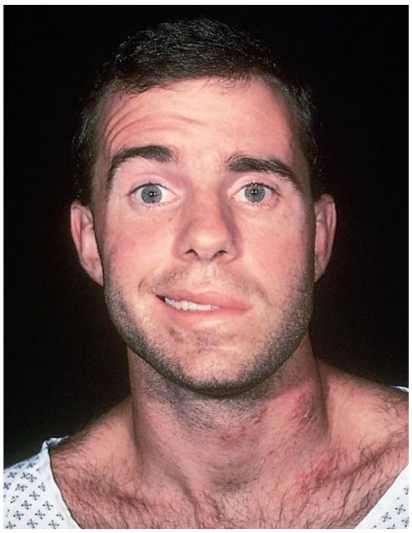


Bells Palsy

- Inflammation of CN VII (Facial nerve)
- Unilateral weakness of the facial muscles of expression unilateral
- Change in taste sensation
- Hyperacusis (sensitivity to sound)
- Corneal dryness

Appendix 8

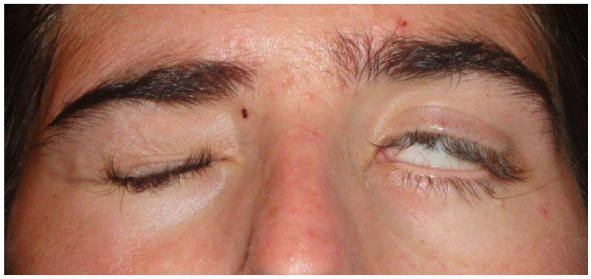
Bells Palsy



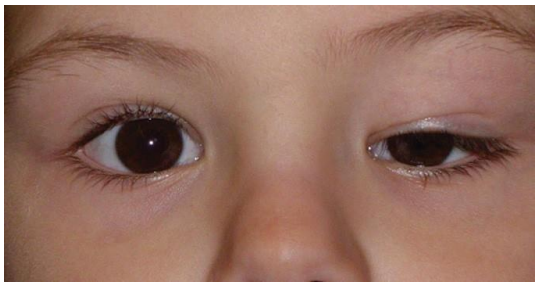
- Inflammation of CN VII (Facial nerve)
- Unilateral weakness of the facial muscles of expression unilateral
- Change in taste sensation
- Hyperacusis (sensitivity to sound)
- Corneal dryness

Problems with eye lid function

Lagophthalmos - can't close
Orbicularis Oculi (CN VII)

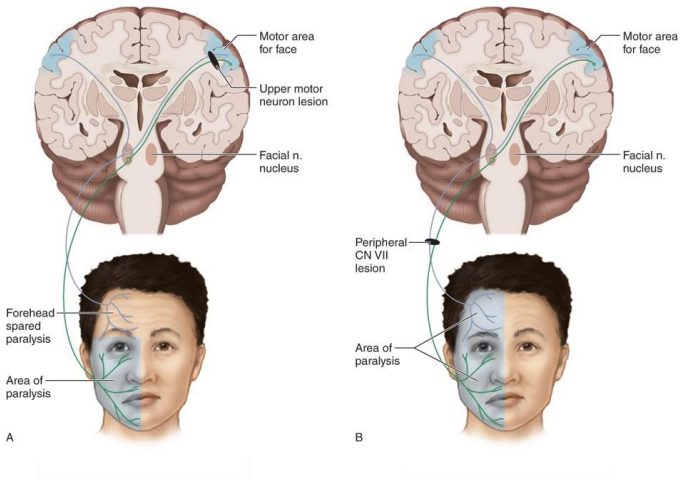


Ptosis – can't open
Levator Palpebra (CN III)



Appendix 8

LMN V. UMN

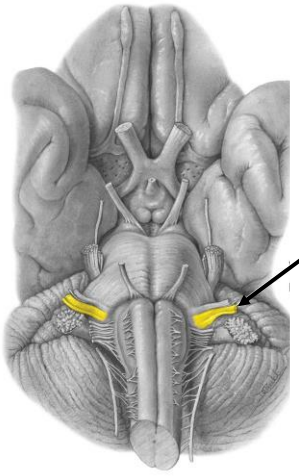


Lesions of CN VII

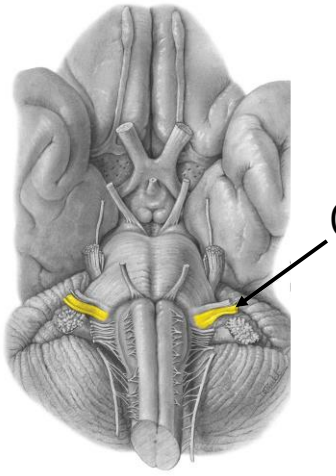
- Symptoms depend on the location of the insult
- Facial nerve palsy with decreased hearing is bad!
- Facial nerve palsy with increase in sound – good!
- Major Parasympathetic Nerve of the head
- Loss of taste to anterior 2/3rd
- Preservation of upper part of facial muscles – think CNS!

Appendix 8

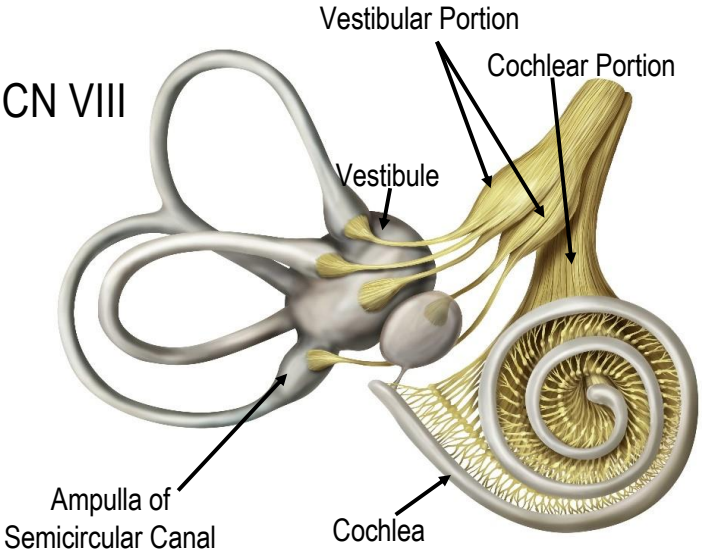
CN VIII
Vestibulocochlear Nuclei



- Double nerve that receives auditory and vestibular input from the ear
- Exits skull through the internal acoustic meatus

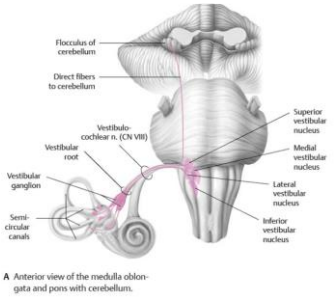


Vestibulocochlear Nerve

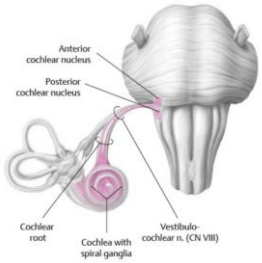


Appendix 8

CN VIII
Vestibulocochlear Nuclei



A Anterior view of the medulla oblongata and pons with cerebellum.



A Anterior view of the medulla oblongata and pons.

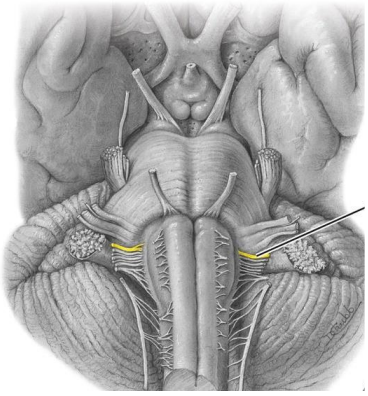
- Midbrain ~~1~~ 2 3 4
- Pons 5 ~~6~~ ~~7~~ ~~8~~
- Medulla 9 10 ~~11~~ 12

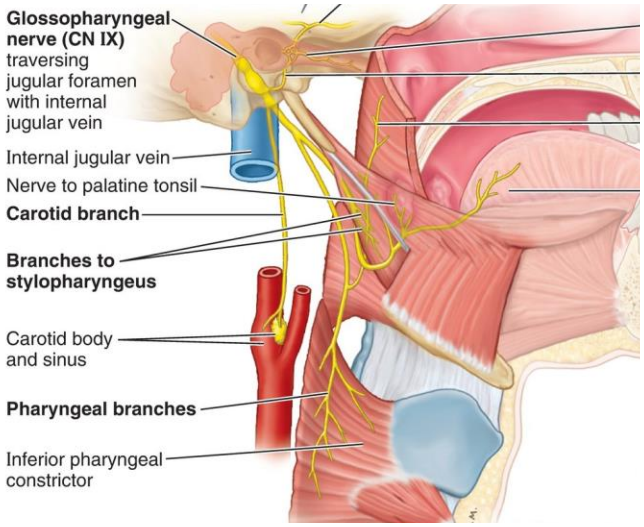
Problems with CN VIII

- Vertigo
- Meniere's disease
- Nystagmus
- Hearing loss

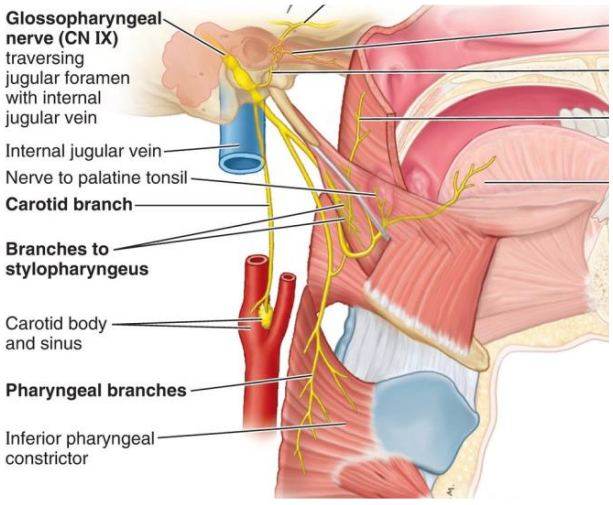
Appendix 8

Glossopharyngeal Nerve – CN IX





Glossopharyngeal Nerve – CN IX



"Glosso" –taste and somatic sensation from the posterior 1/3 of the **tongue**

"Pharyngeal" - somatic sensation of posterior oro**pharynx** and motor innervation of gag reflex (stylopharyngeus muscle)

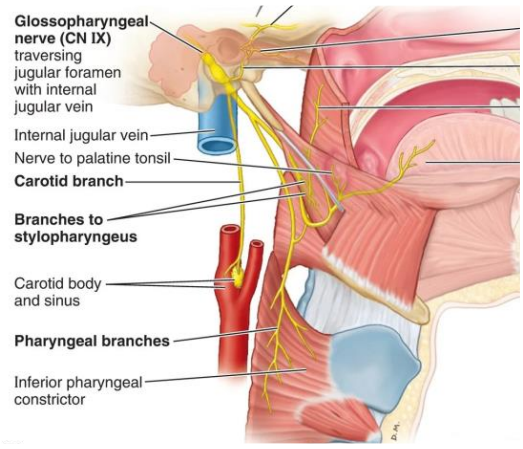
+ Autonomic side -hustle

Carotid & Parotid

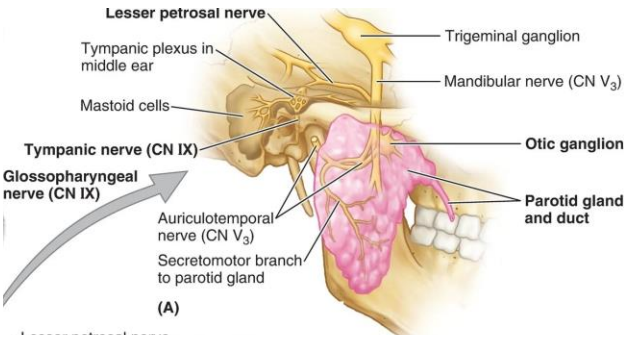
Appendix 8

Glossopharyngeal Autonomics

Carotid



Parotid

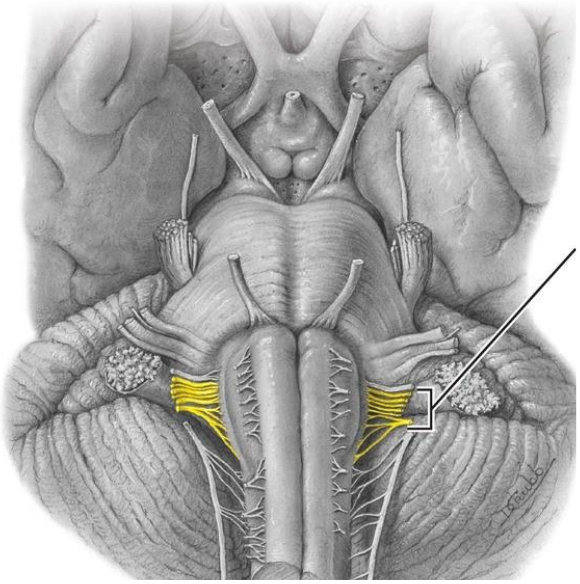


Problems with CN IX

- Loss of gag reflex
- Glossopharyngeal neuralgia

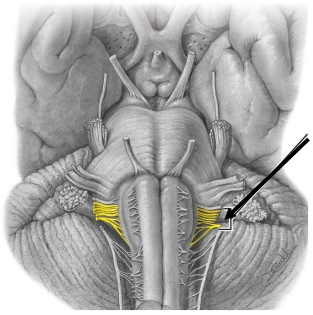
Appendix 8

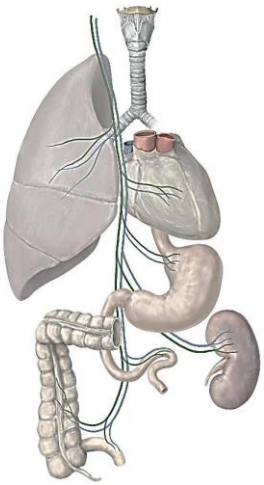
Vagus Nerve – CN X

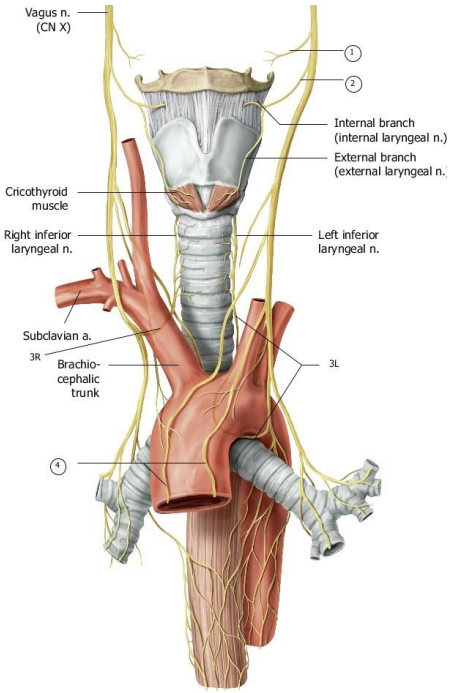


- Arises from ventral lateral medulla just below CN IX
- Exits the cranial cavity via the jugular foramen.

Vagus Nerve







Motor Nerves
Pharyngeal branches
Superior laryngeal nerve
R and L recurrent laryngeal nerve
Cervical cardiac branches

Visceral Afferents from Carotid and foregut

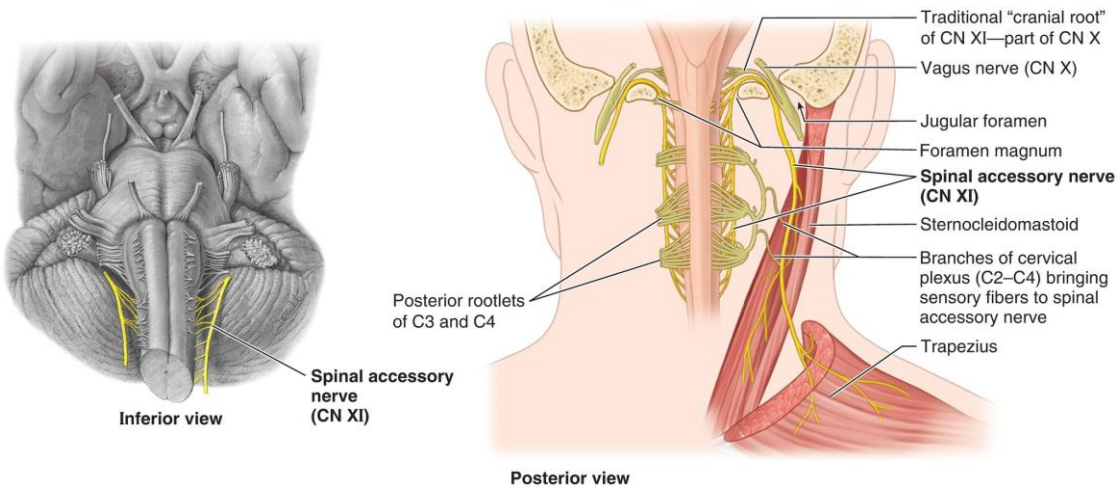
Parasympathetic to the foregut

Appendix 8

Problems with CN X

- Uvula and palate do not elevate

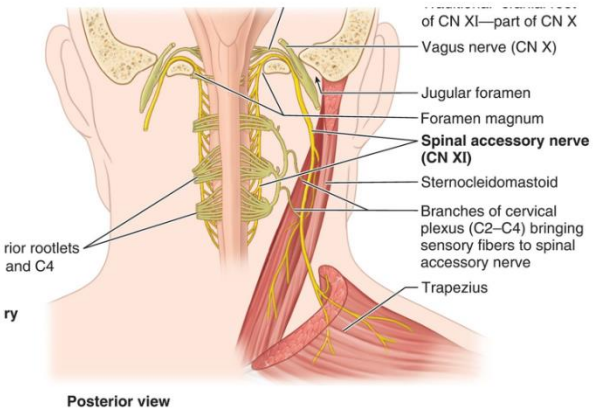
Accessory Nerve – CN XI



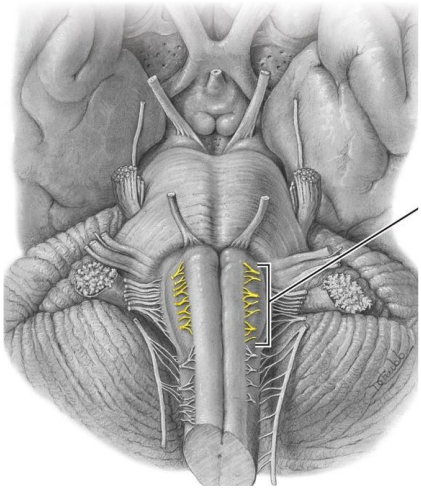
Appendix 8

Lesion of CN XI

- Loss of shoulder shrug



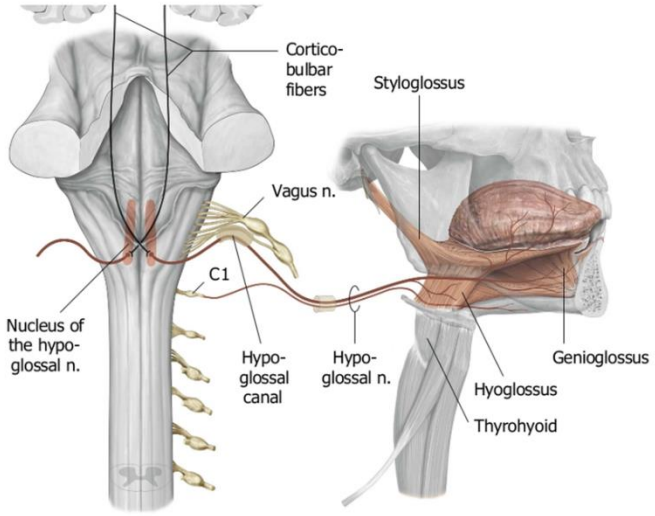
Hypoglossal Nerve – CN XII



- Arises from medial medulla
- Innervation of tongue muscles

Appendix 8

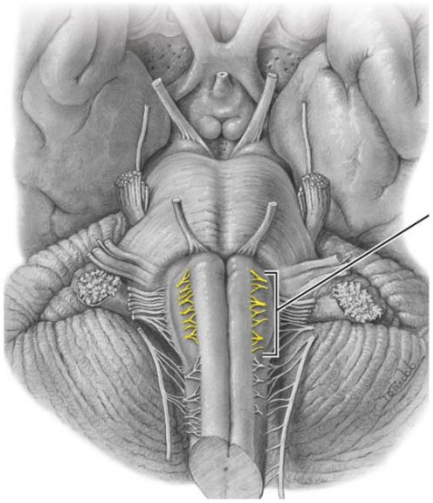
Hypoglossal Nerve – CN XII



- Nuclei in medulla
- Arises from medulla medially and ventrally
- Exits skull through the hypoglossal foramen
- Inserts into the hypoglossus, genioglossus and styloglossus muscles

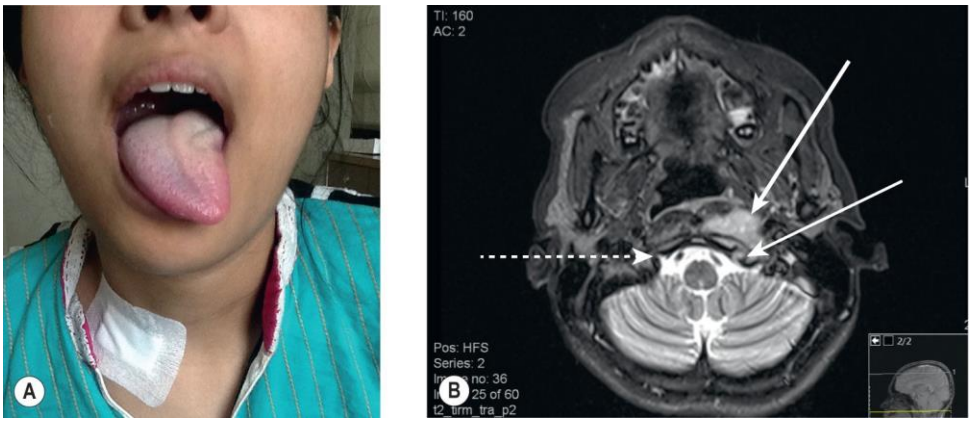
Lesions of the CN XII

- Tongue points toward the side of the lesion



Appendix 8

Lesions of the CN XII



Sources

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Evidence-based Physical Diagnosis Steven McGee MD

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Some slides courtesy of Dr. K. Klump MD, PhD– any errors are my own.

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Appendix 9

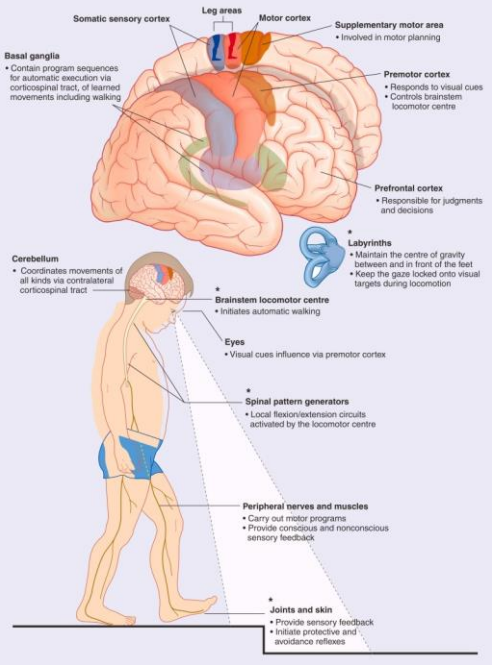
Movement (Volition and Coordination)

Perdue

Learning objectives

- Describe motor function as volition and coordination
- Identify the components of the nervous system involved in movement as volition and coordination
- Discuss the following movement disturbances in the context of neuroanatomical location or system:
  - Ataxia, tremor, bradykinesias, dyskinesias

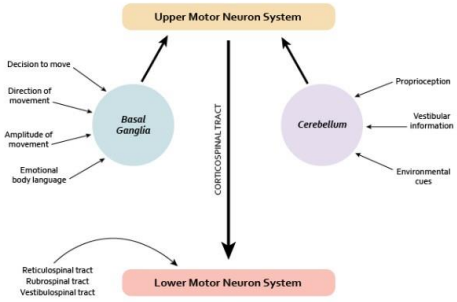
Appendix 9



Movement & Spatial Orientation

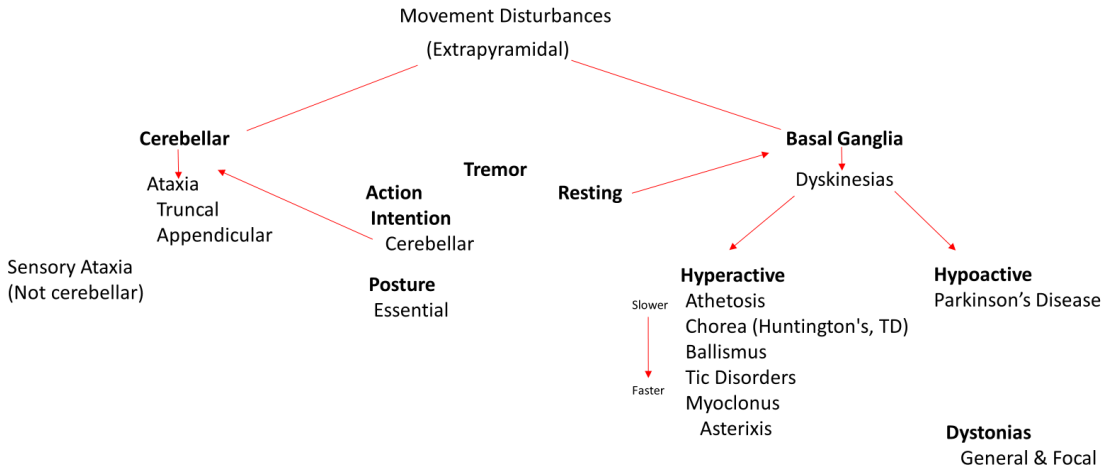
- Spatial Orientation
  - Proprioception
  - Vestibulum
  - Vision
- Volitional component of movement
  - Basal ganglia - the initiation and control of movements
- Coordination component
  - Cerebellum

Motor System

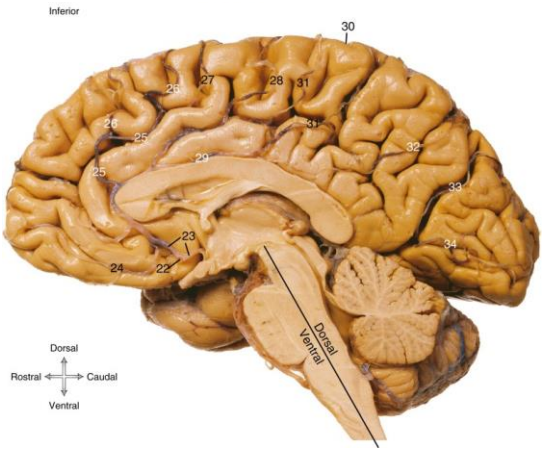


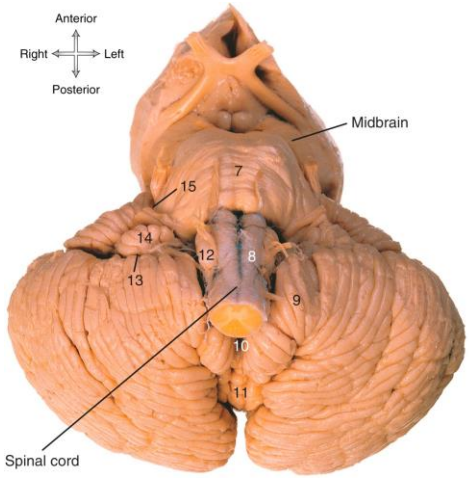
- Cerebral cortex
  - Intent of the movement
  - Concept of an action
  - Output from the cortex projects to the basal ganglia, brainstem and spinal cord
- Brain stem and spinal cord
  - Decode the intent to move into muscle movements, activating specific motor neurons and receiving feedback on the success of the intended movements.
- Cerebellum
  - Receives input on movement and provides information used to correct movement
- Basal ganglia
  - Receive input regarding success in movement have major projections of their output *ultimately back to the cortex* – this modifies its output in accordance with the information received

Appendix 9



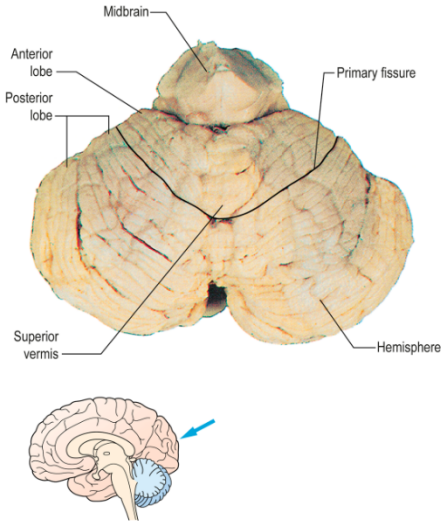
Cerebellum





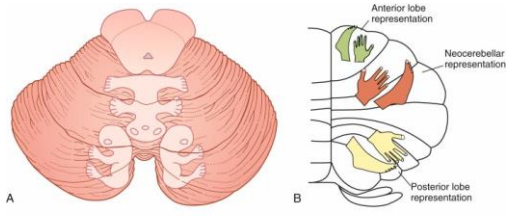
Appendix 9

Cerebellum



a. Two hemispheres
    a. Cortex - deep white matter, and deep gray matter
b. Vermis - midline structure lies between the hemispheres.

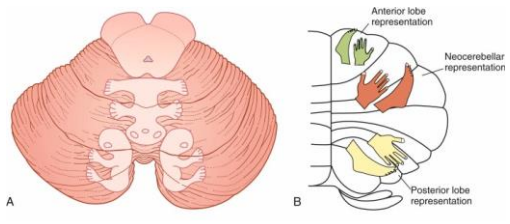
Cerebellum



a. Vermis - midline structure lies between the hemispheres.
    a. Controls coordination of the middle of the body, so pathology of the vermis leads to **truncal and gait instability** .

Appendix 9

Cerebellum

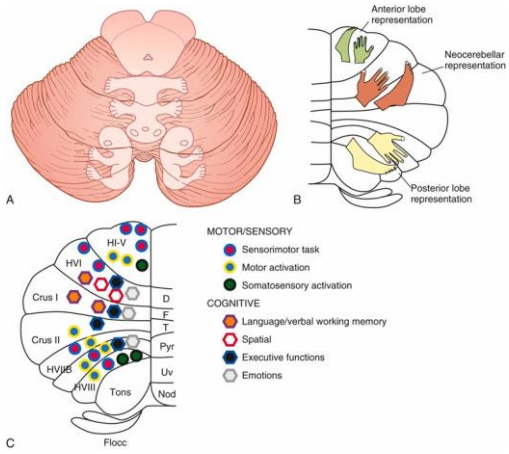


I. **Lateral** cerebellar hemispheres control the lateral parts of the body: the **limbs**.

II. Lesions of one or both cerebellar hemispheres can cause limb ataxia.

III. Lesions in the cerebellar hemispheres cause deficits in the arm and/or leg **ipsilateral** to the affected hemisphere

Cerebellum

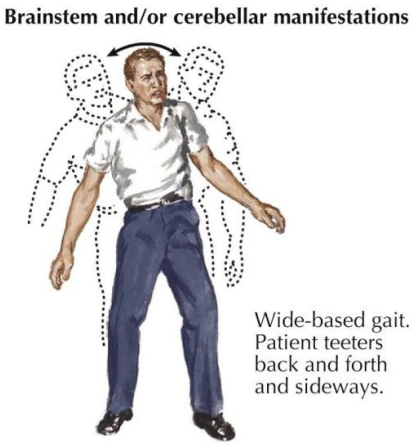


- The left and right **flocculi** and the **midline nodulus** (together referred to as the **flocculonodular lobe**) are anterior cerebellar structures involved in vestibular function and eye movements.

Appendix 9

Movement Disturbances
Cerebellar

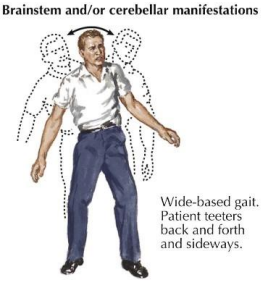
- **S**tance and gait
- **T**remor – intention
- **A**taxia – coordination problems
- **N**ystagmus
- **D**ysarthria



Movement Disturbances
Cerebellar

- Limb ataxia
  - Finger-to-nose
  - Heel-shin
  - Rapid alternating movement

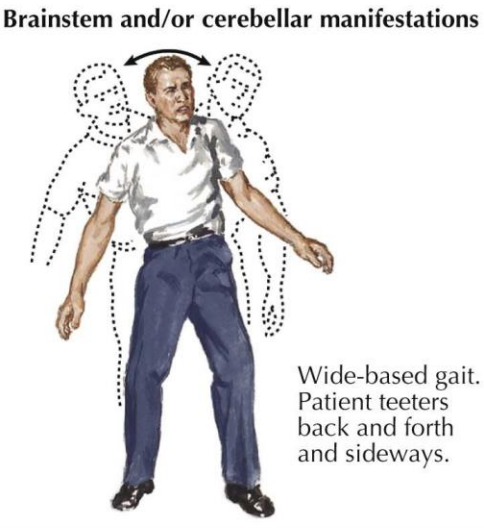
- Truncal and gait ataxia
  - Heel-to-toe walk



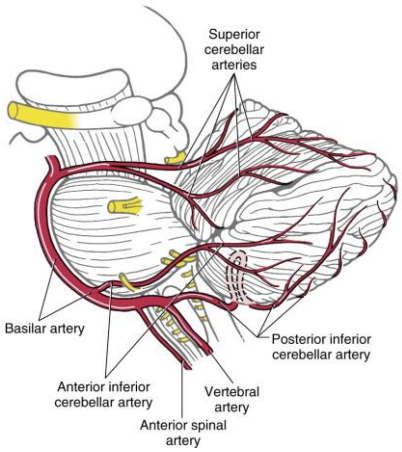
Appendix 9

Movement Disturbances
Cerebellar Diseases

- Cerebellar tumor
- Bleed/Stroke
- Abscess
- Chronic diseases



Cerebellar Circulation



Appendix 9

Ataxia

Cerebellar

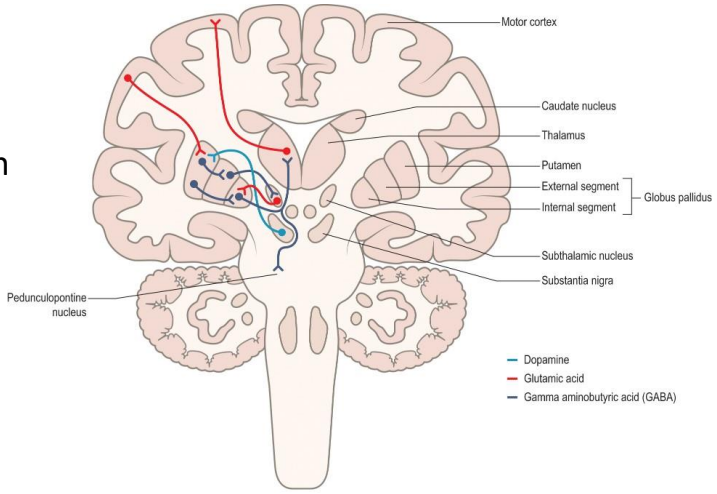
- Nystagmus
- Titubation (oscillation of the head and/or trunk at rest)
- Dysarthria
- Romberg's?

Sensory

- No nystagmus or dysarthria
- Proprioception and vibration sense
- Romberg's sign positive
- Pseudoathetosis – mini Romberg's

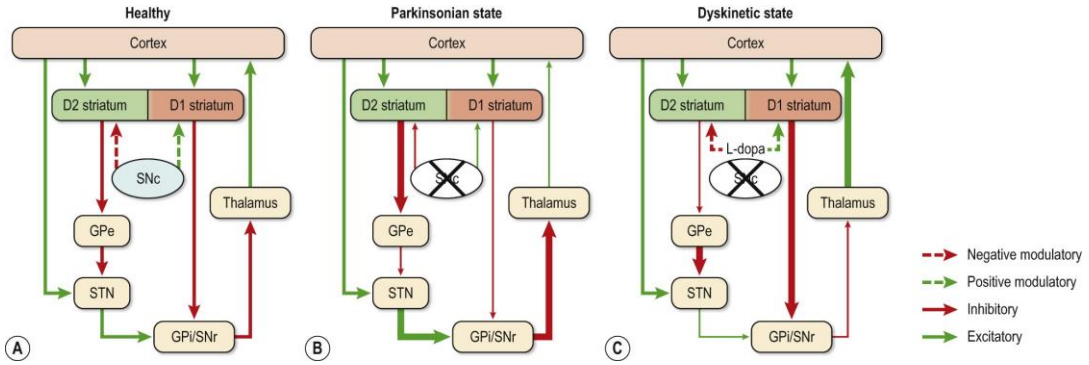
Basal ganglia

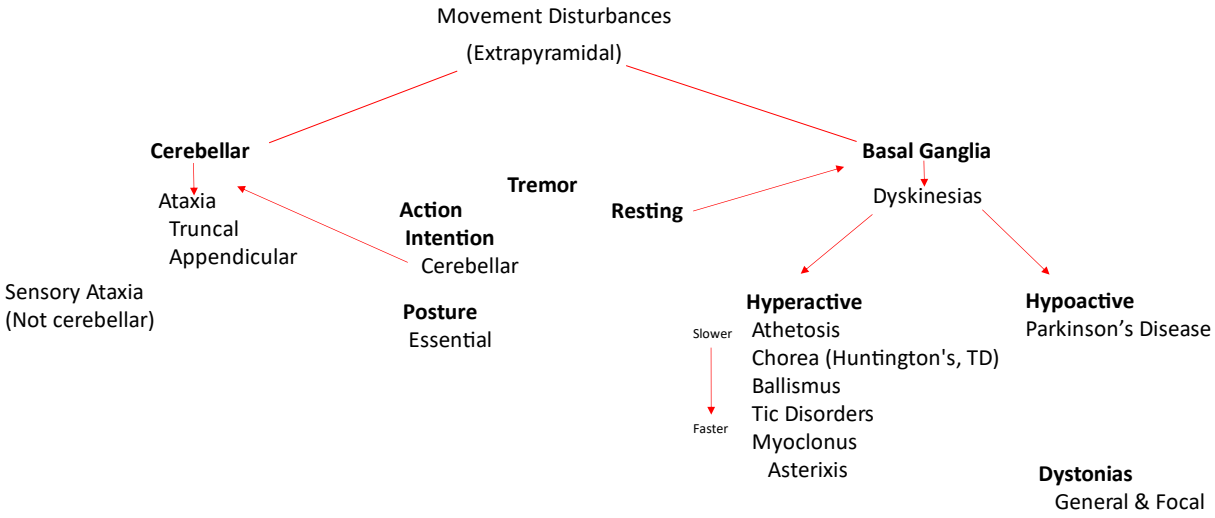
- "…function is to facilitate behavior and movements that are required and appropriate in any particular context and to inhibit unwanted or inappropriate movements."
- Fitzgerald's Neuroscience



Appendix 9

Basal Ganglia





Appendix 9

Movement Disturbances
Hyperkinetic dyskinesia

- Chorea
  - "Dance-like"
  - Involuntary
  - Irregular
  - Purposeless
  - Abrupt
  - Rapid
  - Unsustained movements that seem to move unpredictably from one body part to another

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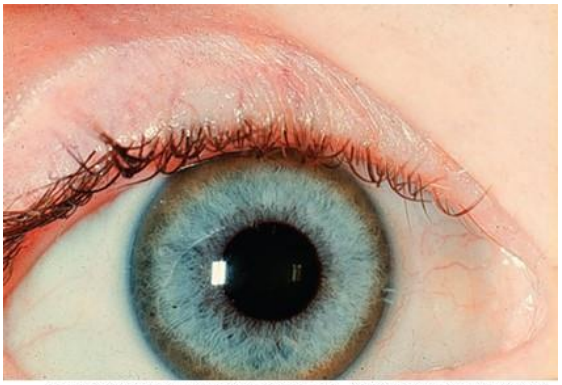
Wilson's disease

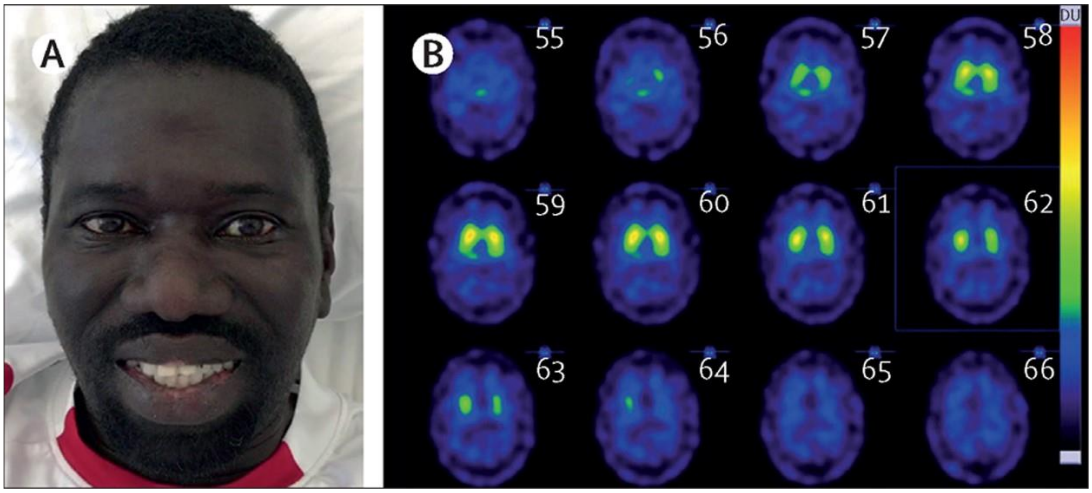


Source: Maxine A. Papadakis, Stephen J. McPhee, Michael W. Rabow, Kenneth R. McQuaid: Current Medical Diagnosis & Treatment 2022

- An autosomal recessive disorder of biliary copper excretion the causes progressive degeneration of the **liver** and **basal ganglia**.
- **Dysarthria, dystonia, rigidity, tremor, choreoathetosis and prominent psychiatric disturbances**
- Wing beating tremor, arms are abducted and elbows flexed
- Facial dystonia called the *risus sardonicus* , wry smile
- Liver disease

Appendix 9

Wilson's disease



Hyperkinetic dyskinesias

- Tic disorders
  - Brief, stereotyped abnormal movements or sounds
  - Paroxysmally out of a background of normal motor behavior and usually associated with an urge
  - Can be stopped on command, but there is ensuing buildup of tension to do it

NEUROLOGY VIDEO TEXTBOOK (2013)

Appendix 9

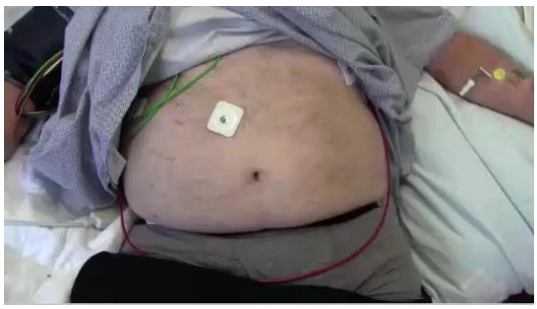
Hyperkinetic dyskinesias

- Tic disorders
  - Tourette's syndrome

NEUROLOGY VIDEO TEXTBOOK (2013)

Movement Disturbances
Hyperkinetic dyskinesias

- Myoclonus
  - Sudden, lightening-like movement
  - Can be repetitive, but not rhythmic
  - Similar to tremor, but non-rhythmic



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Appendix 9

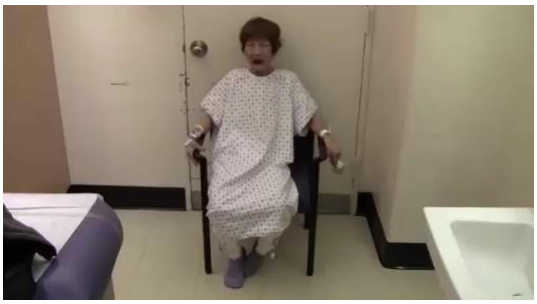
Movement Disorders

- Dystonias
- Sustained muscle contractions cause abnormal postures or repetitive movements
- Frequently initiated or worsened by voluntary movements
- Frequently repetitive in the same group of muscles
  - Unlike chorea
- Can be focal, segmental, or generalized

NEUROLOGY VIDEO TEXTBOOK (2013)

Movement Disorders

- Drug-induced movement disorders
  - Acute dystonic reaction



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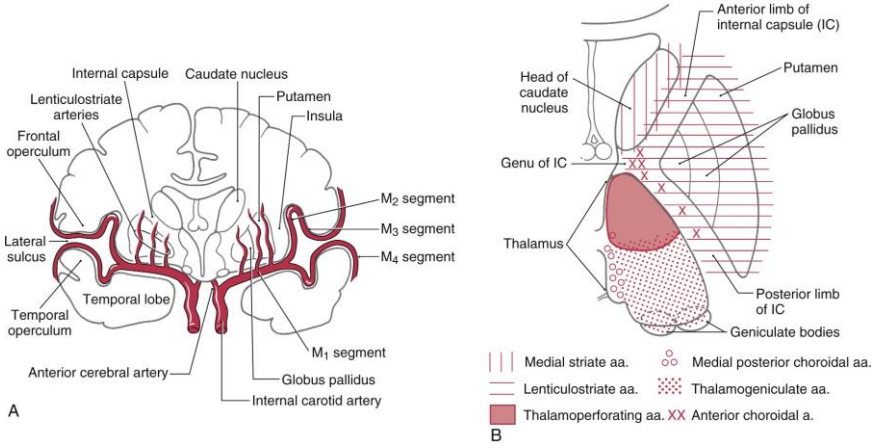
Appendix 9

Basal Ganglia



Movement Disturbances
(Extrapyramidal)

Cerebellar

Ataxia
Truncal
Appendicular

Sensory Ataxia
(Not cerebellar)

Tremor

Action
Intention
Cerebellar

Posture
Essential

Resting

Basal Ganglia

Dyskinesias

Hyperactive
Athetosis
Chorea (Huntington's, TD)
Ballismus
Tic Disorders
Myoclonus
Asterixis

Slower
Faster

Hypoactive
Parkinson's Disease

Dystonias
General & Focal

Appendix 9

Sources

Netter Collection of Medical Illustrations: Nervous System, Part I - Brain.

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Appendix 10

Strokes: Pathophysiology and Clinical Phenomenology

Perdue

Objectives

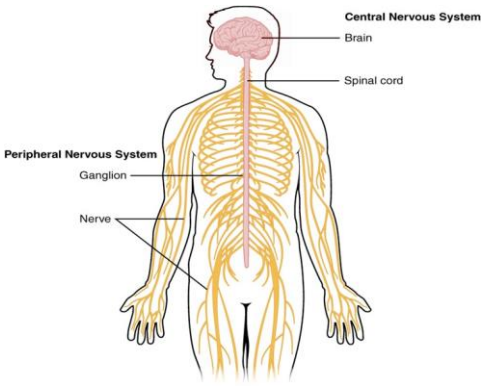
- Describe the pathophysiologic mechanisms of ischemic stroke
- Relate these mechanisms to anatomical structures
- Describe the localization method of CNS ischemic lesions
- Identify the signs and symptoms of neurological dysfunction associated with lesions of the
  - Cortex, subcortical brain, brainstem
- Distinguish deficits associated with anterior circulation problems with posterior circulation problems

Appendix 10

Morbidity and mortality of strokes

- In 2020, 1 in 6 deaths from cardiovascular disease was due to stroke.
- Every 40 seconds, someone in the United States has a stroke.
- Every year, more than 795,000 people in the United States have a stroke.
- About 87% of all strokes are ischemic strokes, in which blood flow to the brain is blocked.
- Stroke-related costs in the United States came to nearly $53 billion between 2017 and 2018.
  - This total includes the cost of health care services, medicines to treat stroke, and missed days of work.
- Stroke is a leading cause of serious long-term disability.
- Stroke reduces mobility in more than half of stroke survivors age 65 and older.

Where is it?



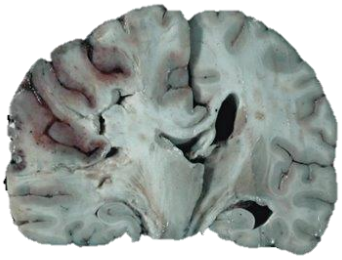
- CNS
  - Cortical brain
  - Subcortical brain
  - Brainstem
  - Spinal cord
- PNS
  - Nerve root
  - Nerve
  - Muscle

Appendix 10

What is it?

• **V**ascular	• Strokes (Ischemic)/Bleeds
• **I**nfection	• Encephalitis, meningitis
• **N**eoplasm	• Brain tumors
• **D**egeneration & **D**rugs	• Degen –ALS Drugs - EPS
• **I**nflammation, **I**diopathic	• Seizure (idio)?
• **C**ongenital	• Malformations, CP
• **A**utoimmune	• Multiple sclerosis, GBS
• **T**rauma	• TBI, SC injury, nerve damage
• **E**ndo-Metab	• Diabetic neuropathy

Ischemic stroke: Cerebrovascular accident



Mechanisms

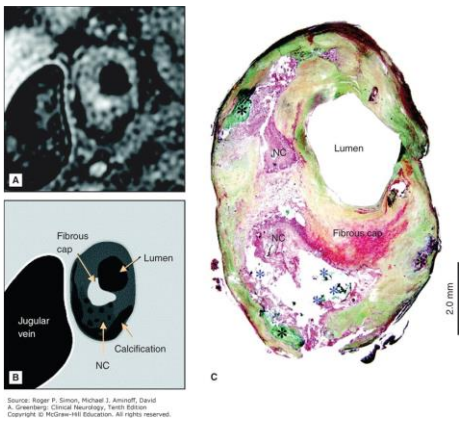
I. Thrombotic stroke

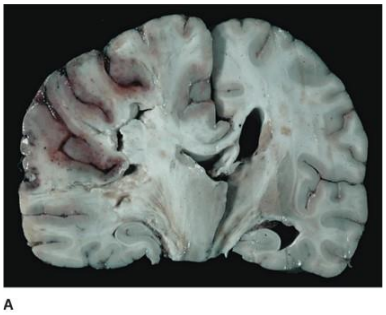
II. Embolic stroke

III. Systemic hypoprofusion

Appendix 10

Atherosclerotic cerebrovascular disease





A

Source: Roger P. Simon, Michael J. Aminoff, David A. Greenberg: Clinical Neurology, Tenth Edition
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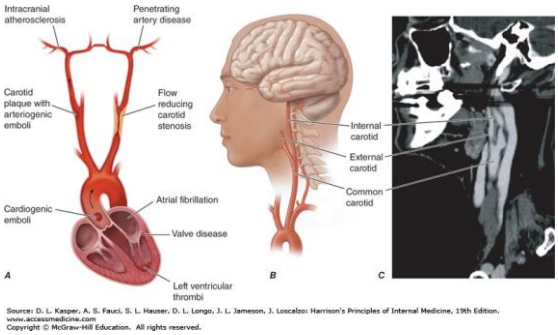
Atherosclerotic cerebrovascular disease



Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition.
www.accessmedicine.com
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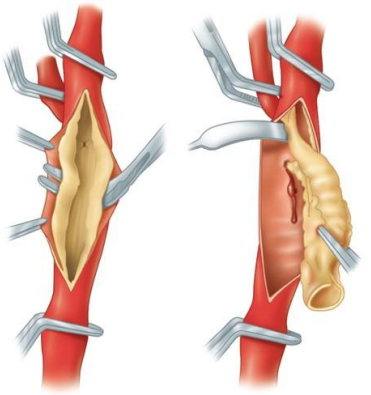
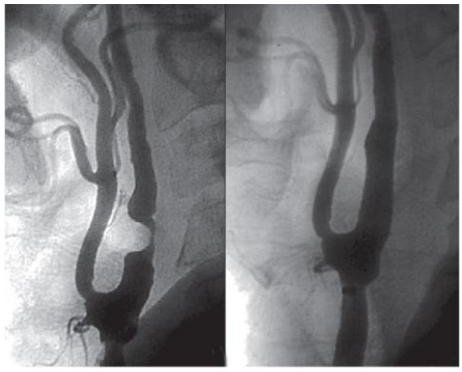
- Atherosclerotic plaques within arterial vessels of the brain's blood supply can cause occlusive disease
- Thrombotic disease from rupture of plaque and subsequent occlusion of vessel
- Embolism – plaque breaks off and circulates to distal site
- Risk factors "SAD CHF"

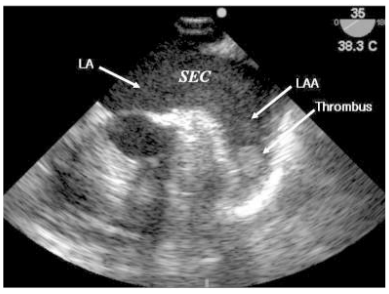
Appendix 10

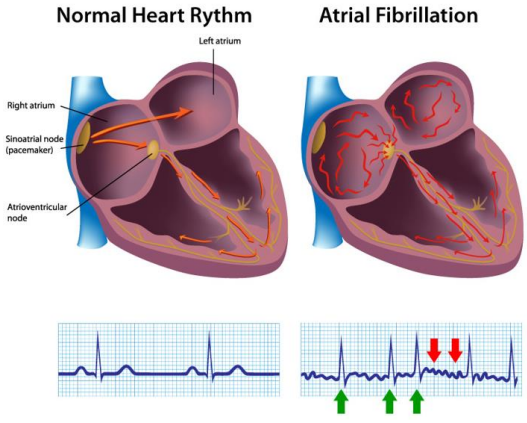
Carotid disease





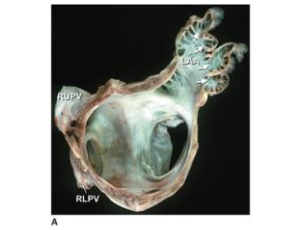
Cardioembolic strokes





Appendix 10

Left atrial appendage



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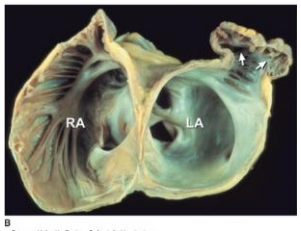
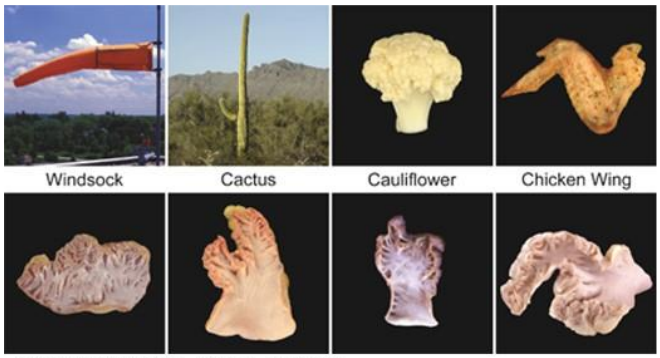
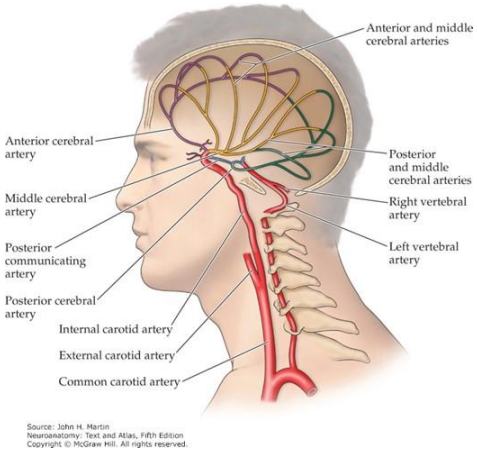


B



Review: Anterior v. Posterior Circulation



Appendix 10

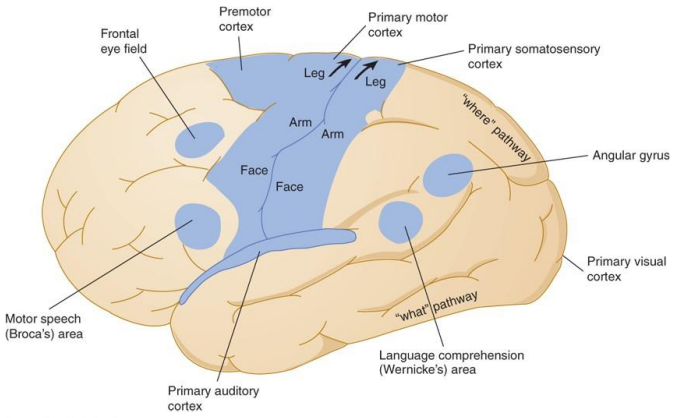
Where is it? the stroke?

- Are there cortical findings?
  - Aphasia, visuospatial neglect, gaze deviation, loss of vision, mental status change are indicators of stroke involving the **cortex** of the brain
- Is there equal face/arm/leg involvement **without** cortical signs?
  - **Subcortical** lesion
- Are there **crossed** findings?
  - Ipsilateral face, contralateral body, eg. think brainstem/medullary lesion

- Cortical brain
- Subcortical brain
- Brainstem

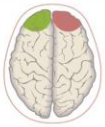
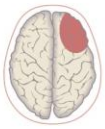
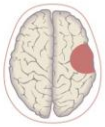
Weakness and sensation abnormalities of body?

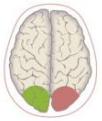
- Cortex?
  - Frontal lobe
  - Parietal
  - Temporal lobe



Appendix 10

Cortical lesions

Site of lesion	Disorder	R	L
Frontal, either	Intellectual impairment Personality change Urinary incontinence Monoparesis or hemiparesis		
Frontal, left	Broca's aphasia		
Temporo-parietal, left	Acalculia Alexia Agraphia Wernicke's aphasia Right-left disorientation Homonymous field defect		

Site of lesion	Disorder	R	L
Temporal, right	Confusional states Failure to recognize faces Homonymous field defect		
Parietal, either	Contralateral sensory loss or neglect Agraphaesthesia Homonymous field defect		
Parietal, right	Dressing apraxia Failure to recognize faces		
Parietal, left	Limb apraxia		
Occipital/ occipitoparietal	Visual field defects Visuospatial defects Disturbances of visual recognition		

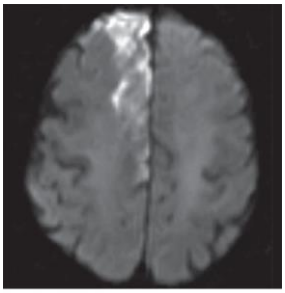
Weakness and sensation abnormalities of body?

- Cortex?
  - Frontal lobe
  - Parietal
  - Temporal lobe
- Vascular Territory:
  - ACA & MCA off of ICA
- Contralateral somatosensory and motor deficits
- Cognitive impairment
  - Aphasias
  - Neglects

Appendix 10

Anterior Cerebral Artery (Frontal Lobe)

- Aphasia (dominant left hemisphere)
- Contralateral Weakness –**Leg > Arm**
- Gaze preference (destructive lesion affecting frontal eye fields)
- Abulia (lack of will)
- Impaired Judgment, disinhibition, loss of abstract thought if bi-frontal lobe involvement



Source: Aaron L. Berkowitz:
Clinical Neurology & Neuroanatomy: A Localization-Based Approach, 2e
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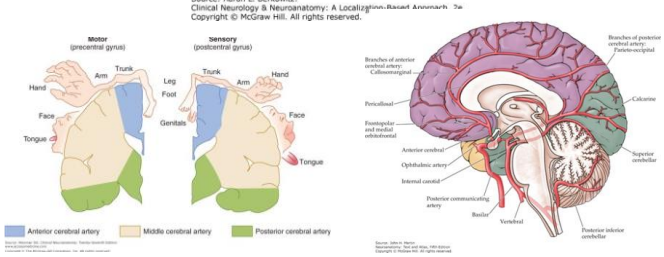
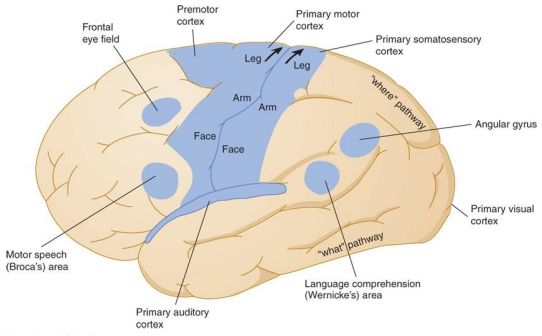


Middle Cerebral Artery (Parietal Lobe)

- Left Parietal Lobe
  - Aphasia
  - Right sensory loss
  - Right Side Weakness
    - **Face & Arm > Leg**
  - Right-Left Confusion
  - Agraphesthesia
  - Right Lower quadrantopia



Source: Aaron L. Berkowitz:
Clinical Neurology & Neuroanatomy: A Localization-Based Approach, 2e
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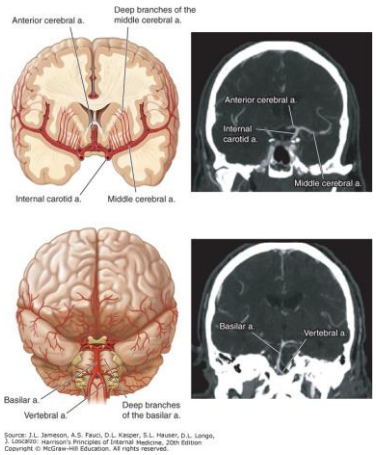
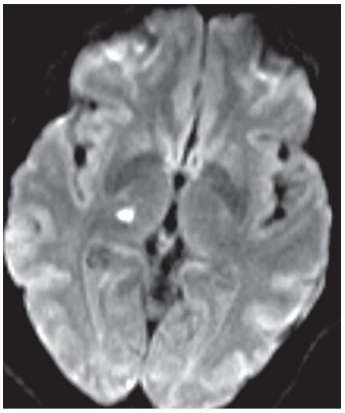
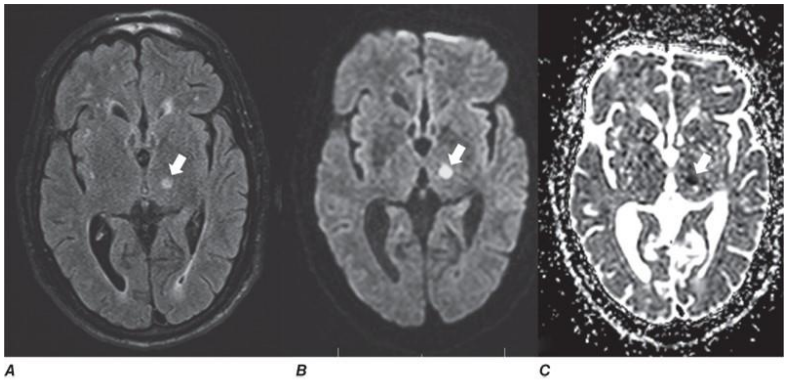
Appendix 10

Lacunar stroke



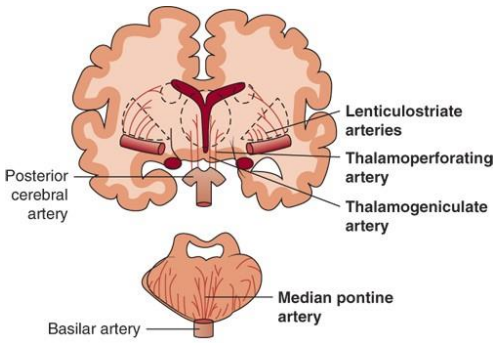


Lacunar Infarction



Appendix 10

Lacunar strokes



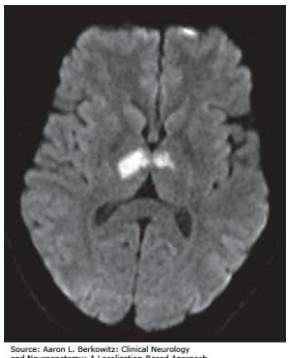
Source: V. Fuster, J. Narula, P. Vaishnava, M.B. Leon, D.J. Callans, J. Rumsfeld, A. Poppas: Fuster and Hurst's The Heart, 15e

- Vascular Supply:
  - Distal PCA branches:
    - Thalamogeniculate arteries
    - Thalamoperforating Arteries

Thalamus

- Sensory Relay Station
- Multiple Small Nuclei
- **Loss of Sensation on one side of body**
- Ataxia (if internal capsule involved)
- Coma (if bilateral)
- Aphasia (if in dominant hemisphere)

- Vascular Supply:
  - Distal PCA branches:
    - Thalamogeniculate arteries
    - Thalamoperforating Arteries

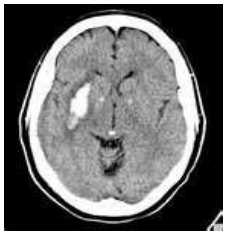


Source: Aaron L. Berkowitz: Clinical Neurology and Neuroanatomy: A Localization-Based Approach
www.neurology.mhmedical.com

Basal Ganglia

- Consists of Nuclei: Sub-Thalamus, Caudate, Putamen and Globus Pallidus
- Role in smooth movement

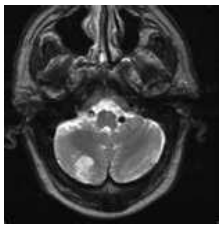
- Vascular Supply:
  - Anterior Choroidal
  - Anterior Cerebral
  - Middle Cerebral Artery



Cerebellum

- Wide Based Gait
- Decreased Tone
- **Ipsilateral** Dysmetria
- Ataxia
- Nystagmus

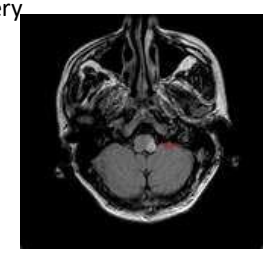
- Vascular Supply
  - Vertebral-Basilar System
    - PICA
    - AICA
    - SCA



Brainstem

- **Midbrain**: pupil abnormalities, decreased vertical eye movements, Decreased LOC
- **Pons**: Dysarthria, contralateral weakness, horizontal gaze problems, pinpoint pupils, locked in syndrome, decreased LOC
- **Medulla**: autonomic nervous system, Wallenberg syndrome, Decreased LOC

- Vascular Supply
  - Mid Brain: Posterior Cerebral Artery from Basilar Artery
  - Pons: Pontine Arteries from Basilar Artery
  - Medulla:
    - Vertebral Artery, PICA
    - Anterior Spinal Artery

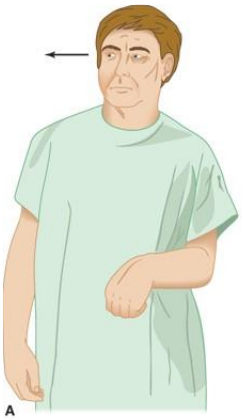


Focal deficits– Cortical Brain

- History - Sudden onset
- Contralateral
  - **Paralysis** (Spastic - UMN)
  - **Dysthesias** (Complete - PCML & ALS/ST)
- ***Either*** Face = Arm > leg (MCA)

  Or

  Leg (ACA)
- Motor and sensory sxs on same side
- Higher cortical function deficits

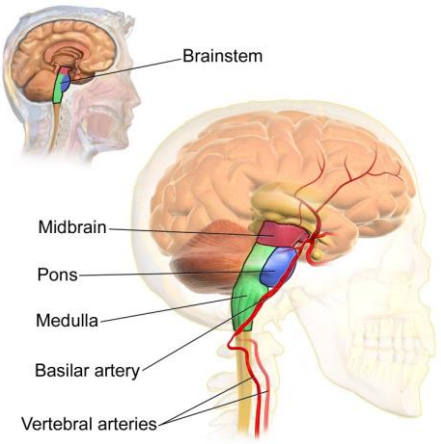


A

Appendix 10

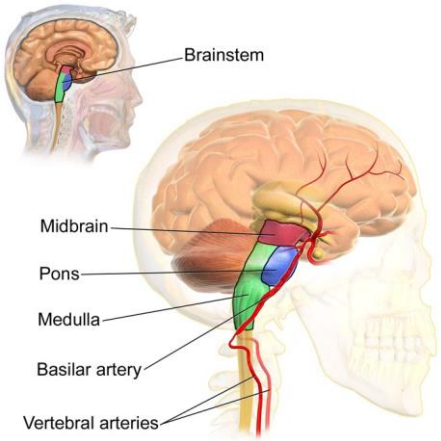
Brainstem

- **Major long tracts**
- **Cranial nerves**
- **Reticular formation**



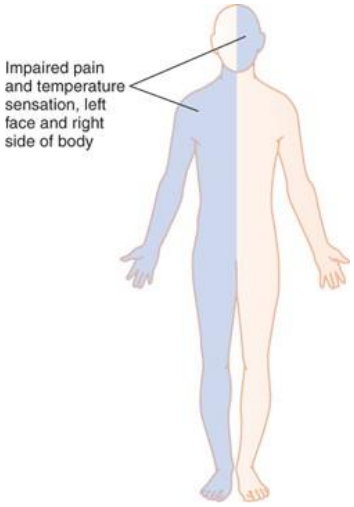
Brainstem Lesions

- **C**rossed or **alternating** long tract signs
- **C**ranial Nerve deficits
- **C**oma
- **C**ardiorespiratory dysfunctions



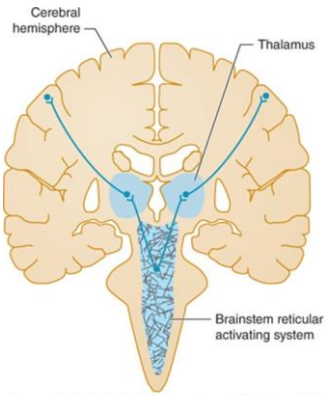
Brainstem

- **Alternating or Crossed** signs:
- Long tracts cross
- Cranial nerve generally do not
- Brainstem lesions often produce symptoms on one side of the face and the opposite side of the body



Brainstem Lesions

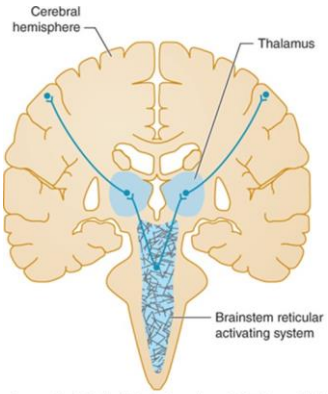
- Coma
  - Reticular activating system
  - Responsiveness



Appendix 10

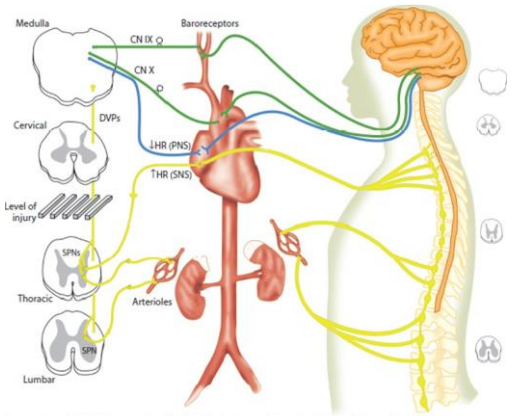
Brainstem Lesions

- Coma
  - Reticular activating system
  - Responsiveness



Brainstem Lesions

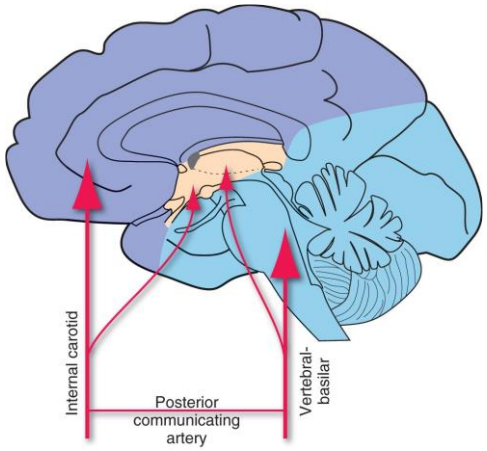
- Cardiorespiratory dysfunction



Appendix 10

Anterior vs. Posterior circulation

- **Anterior circulation**
  - Anterior cerebral arteries: contralateral leg>arm weakness
  - Middle cerebral arteries: Face and arm > leg, loss of speech, contralateral weakness/sensory changes, visuospatial neglect, etc.
- **Posterior circulation**
  - Vertebrobasilar system: nausea, vomiting, ataxia, gait disturbance, double vision, vertigo, headache, crossed findings
  - Posterior cerebral arteries: loss of vision (visual field cut), sensory loss if thalamic involvement)



Brain Deficit Summary

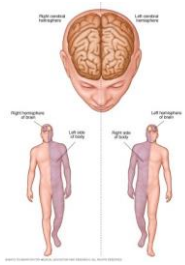
Cortical Brain

Same side
- Contralateral **Weakness** (Spastic paralysis)
- Contralateral **Dysthesias** (PCML/ST)

Face = Arm > Leg or Leg

+/- higher function deficits

+/- visual field deficits



Subcortical Brain

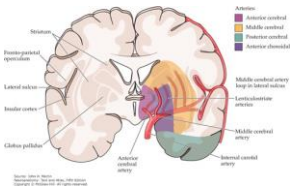
Weakness (Pure Motor)

Dysthesias (Pure sensory)

Mixed

No higher cortical deficits

No visual field abnormalities



Brain stem

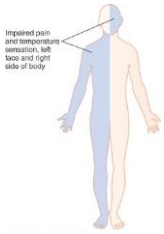
Weakness (Spastic paralysis)

Dysthesias

Crossed (Alternating) deficits

Cranial nerve deficits

Coma & Cardiopulmonary dysfunction ?



Appendix 10

Sources

Netter Collection of Medical Illustrations: Nervous System, Part I- Brain.

Neuroanatomy Text and Atlas, 5th Edition Jones, Martin

Neuroanatomy: An Illustrated Colour Text. Crossman, Alan R, PhD DSc; Neary, David, MD FRCP

Nolte's Essentials of the Human Brain. Vanderah, Todd W., PhD

Clinical Neurology & Neuroanatomy: A Localization-Based Approach, Aaron L. Berkowitz

Nolte's The Human Brain in Photographs and Diagrams. Vanderah, Todd W., PhD.

The Big Picture: Gross Anatomy, David A. Morton, K. Bo Foreman, Kurt H.Albertine

Fuster V, Narula J, Vaishnava P, Leon MB,Callans DJ, Rumsfeld JS, Poppas A. eds. *Fuster and Hurst's The Heart, 15e*. McGraw-Hill Education; 2022. https://accessmedicine-mhmedical-com.ezproxy.nsuok.edu/content.aspx?bookid=3134&sectionid=262105936

Principles of Critical Care, 4e McGraw-Hill

Clinical Neurology, Aminoff and Green Access Medicine McGraw-Hill

Appendix 11

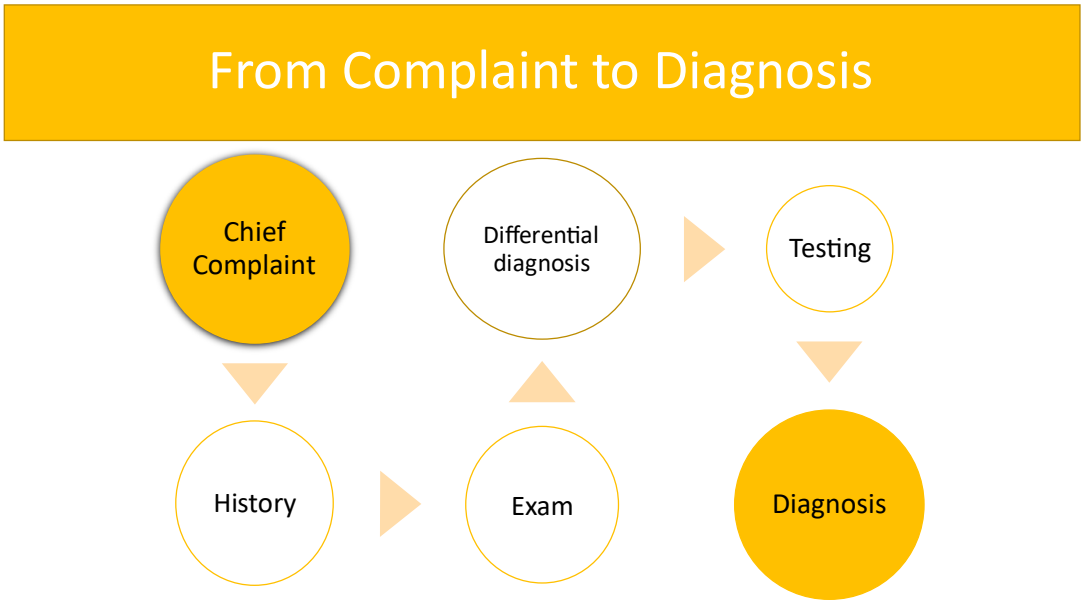
Approach to the Neuro Patient

Perdue

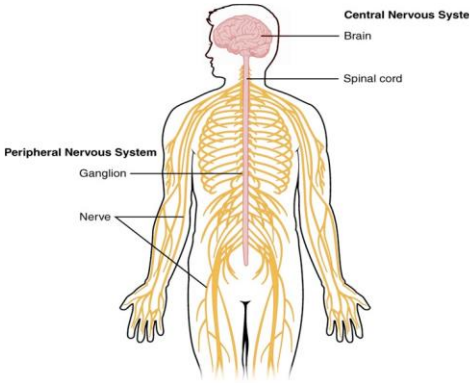
Learning Objectives

- What are the presentation categories for neuro conditions?
- Describe the clinical features of conditions in each of the diagnosis categories.
- Demonstrate the physical examination maneuvers used to diagnose neurological disorders

Appendix 11



Where is it?



- CNS
  - Cortical brain
  - Subcortical brain
  - Brainstem
  - Spinal cord
- PNS
  - Nerve root
  - Nerve
  - NMJ
  - Muscle

Appendix 11

What is it?

• **V**ascular	• Strokes/Bleeds
• **I**nfection	• Encephalitis, meningitis
• **N**eoplasm	• Brain tumors
• **D**egeneration & **D**rugs	• Degen –ALS Drugs - EPS
• **I**nflammation, **I**diopathic	• Seizure (idio)?
• **C**ongenital	• Malformations, CP
• **A**utoimmune	• Multiple sclerosis, GBS
• **T**rauma	• TBI, SC injury, nerve damage
• **E**ndo-Metab	• Diabetic neuropathy

History

What kind of complaint?

Motor and Sensory Deficits

Focal Weakness
Central - Stroke (CVA), MS, ALS
Peripheral – Injury, Neuropathy

Generalized Weakness
Electrolytes
Anemia…

Appendix 11

History

- What kind of complaint?
- Time course of the complaint?
- Does it fit an illness script?

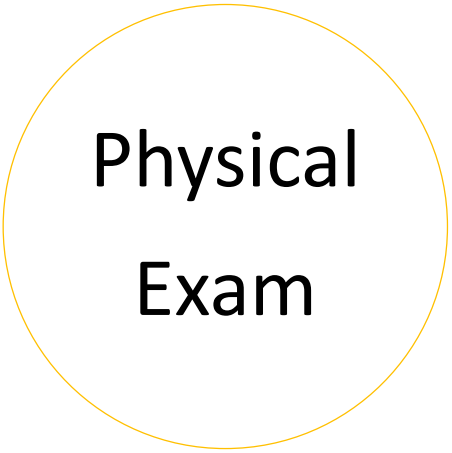
- **Vascular accident** - sudden onset, cardiovascular risk factors
- **Trauma** - sudden onset, MVA, disc herniation
- **Infection** - sudden onset, other signs of infection (fever)
- **Neurodegenerative disease**- chronic and often progressive; often have recognizable set of symptoms
- **Tumor** - onset can be fairly sudden (days) to more gradual (weeks to months); symptoms depend on location of tumor
- **Congenital disorders**– Born with malformations of the nervous system

History

- What kind of complaint?
- Time course of the complaint?
- Does it fit syndrome or an illness script?

Appendix 11

From Complaint to Diagnosis



- Mental status
- Cranial nerves
- Motor
- Reflexes
- Sensation
- Coordination
- Special tests

How do we get from a neurological complaint to a diagnosis?

Physical

Lethargic or Unresponsive?

Glasgow Coma Scale

Normal interaction and conversation?

Mental status

- **Alertness** - brainstem & diffuse cortical function
- **Memory** recent - temporal lobe, remote - diffuse cortical areas
- **Language** Wernicke's area - comprehension; Broca's area - articulation
- **Spatial attention** right parietal and/or frontal lobe; damage causes neglect
- **Executive function** "restraint, control & initiative", frontal lobe

Appendix 11

How do we get from a neurological complaint to a diagnosis?

Physical

Recent – 3 Objects

Remote – Who is president?

Now, when in high school?

Mental status

- **Alertness** - brainstem & diffuse cortical function
- **Memory** recent - temporal lobe, remote - diffuse cortical areas
- **Language** Wernicke's area - comprehension; Broca's area - articulation
- **Spatial attention** right parietal and/or frontal lobe; damage causes neglect
- **Executive function** "restraint, control & initiative", frontal lobe

How do we get from a neurological complaint to a diagnosis?

Physical

Language

Mental status

- **Alertness** - brainstem & diffuse cortical function
- **Memory** recent - temporal lobe, remote - diffuse cortical areas
- **Language** Wernicke's area - comprehension; Broca's area - articulation
- **Spatial attention** right parietal and/or frontal lobe; damage causes neglect
- **Executive function** "restraint, control & initiative", frontal lobe

Appendix 11

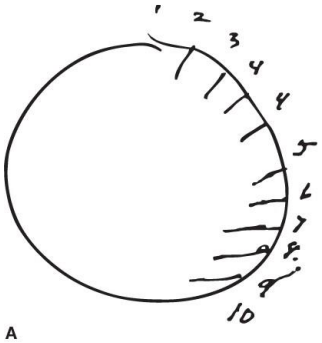
Language Disorders

Wernicke's Aphasia

Broca's Aphasia

How do we get from a neurological complaint to a diagnosis?

Physical



A

B

Mental status

- **Alertness** - brainstem & diffuse cortical function
- **Memory** recent - temporal lobe, remote - diffuse cortical areas
- **Language** Wernicke's area - comprehension; Broca's area - articulation
- **Spatial attention** right parietal and/or frontal lobe; damage causes neglect
- **Executive function** "restraint, control & initiative", frontal lobe

Appendix 11

How do we get from a neurological complaint to a diagnosis?

Physical

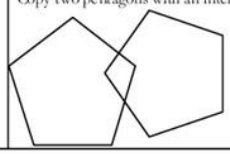
Changes in behavior?

MMSE

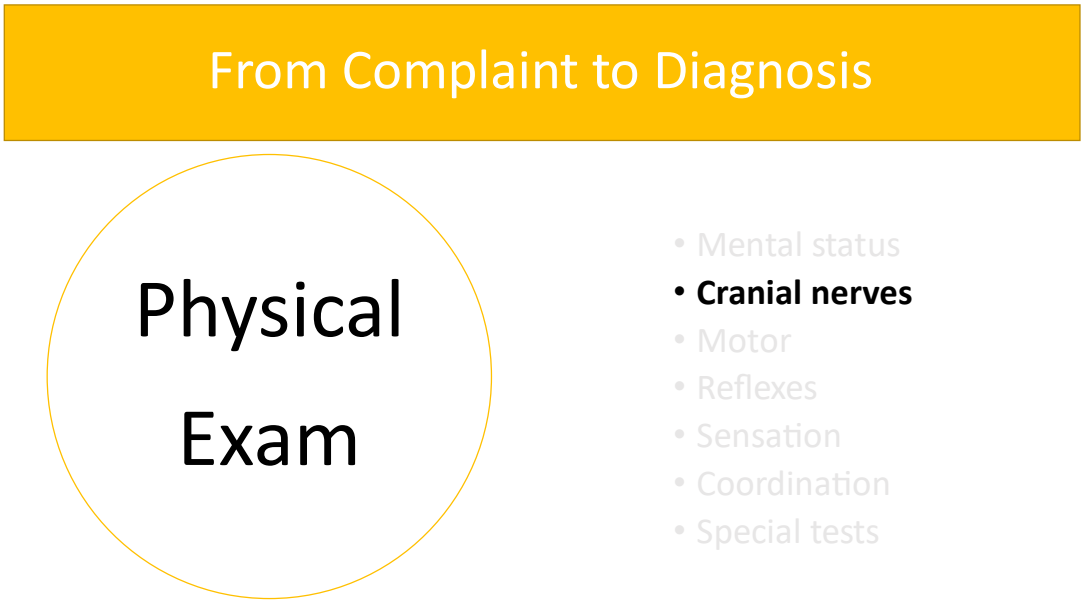
Mental status

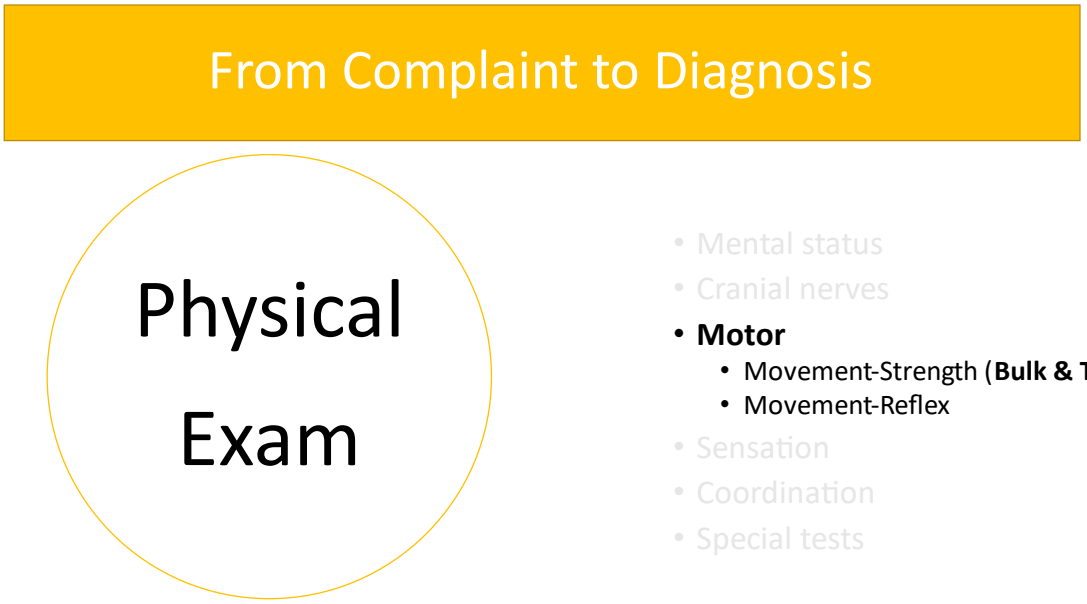
- **Alertness** - brainstem & diffuse cortical function
- **Memory** recent - temporal lobe, remote - diffuse cortical areas
- **Language** Wernicke's area - comprehension; Broca's area - articulation
- **Spatial attention** right parietal and/or frontal lobe; damage causes neglect
- **Executive function** "restraint, control & initiative", frontal lobe

MMSE

Temporal orientation (5 points)	What is the approximate time?
	What day of the week is it?
	What is the date today?
	What is the month?
	What is the year?
Spatial orientation (5 points)	Where are we now?
	What is this place?
	In what district are we or what is the address here?
	In which town are we?
	In which state are we?
Registration (3 points)	Repeat the following words: CAR, VASE, BRICK
Attention and calculation (5 points)	Subtract: 100-7 = 93-7 = 86-7 = 79-7 = 72-7 = 65
Remote memory (3 points)	Can you remember the 3 words you have just said?
Naming 2 objects (2 points)	Watch and pen
REPEAT (1 point)	"NO IFS, ANDS OR BUTS"
Stage command (3 points)	"Take this piece of paper with your right hand, fold it in half, and put it on the floor"
Writing a complete sentence (1 point)	Write a sentence that makes sense
Reading and obey (1 point)	Close your eyes
Copy the diagram (1 point)	Copy two pentagons with an intersection 

Appendix 11

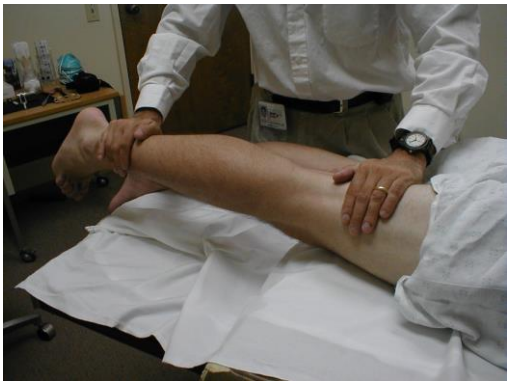

From Complaint to Diagnosis
Physical Exam
• Mental status
• Cranial nerves
• Motor
• Reflexes
• Sensation
• Coordination
• Special tests


From Complaint to Diagnosis
Physical Exam
• Mental status
• Cranial nerves
• Motor
• Movement-Strength (Bulk & Tone)
• Movement-Reflex
• Sensation
• Coordination
• Special tests

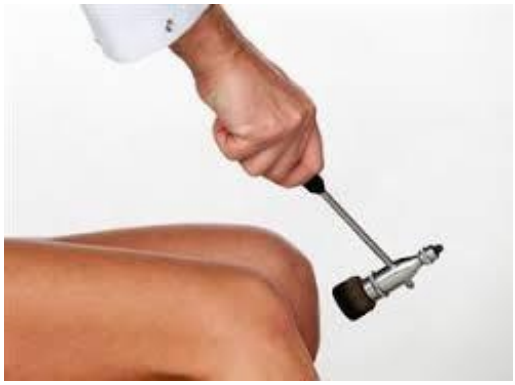
Appendix 11

Motor Exam

Movement as strength



Reflexes



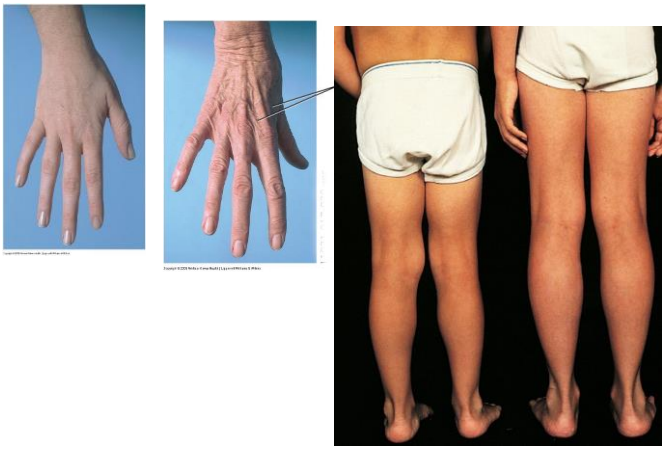
Motor-Movement-as-strength

- **Motor**
  - Movement-Strength (**Bulk & Tone**)
  - Movement-Reflex
- Inspect:
  - Atrophy, muscle tone and bulk
  - Involuntary movements (tremors, tics, fasciculations – further explore in movement-as-volition/coordination)

Appendix 11

Muscle Bulk

- Inspect:
  - Inspect the size and contours of muscles.
  - Do the muscles look flat or concave, suggesting **atrophy**? unilateral or bilateral? proximal or distal?
  - When looking for atrophy, pay particular attention to the hands, shoulders, and thighs.
  - **Pseudo hypertrophy:** as seen with Duchenne muscular dystrophy; increased bulk without increased strength



Muscle tone

- Exam:
- This can be assessed best by feeling the muscle's resistance to passive stretch during **Passive ROM**.
- Take one hand with yours and, while supporting the elbow, flex and extend the wrist, and elbow, and put the shoulder through a moderate range of motion.
- To assess muscle tone in the legs, support the patient's thigh with one hand, grasp the foot with the other, and flex and extend the patient's knee and ankle on each side. Note the resistance to your movements
- On each side, note muscle tone—the resistance offered to your movements.

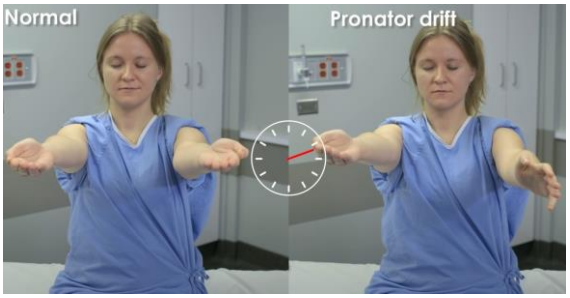
- Spasticity - hypertonia that is rate dependent (tone greater with rapid ROM)
  - Causes: CVA
- Rigidity – hypertonia not dependent on rate (resistance throughout ROM)
  - "Lead pipe rigidity". Causes: Parkinson's
- Flaccidity – hypotonic or floppy. Causes: Guillain- Barre', spinal shock

Appendix 11

Movement as Strength

Pronator drift

- The patient should stand for 15 seconds with both arms straight forward, palms up, and with eyes closed.
  - A person who cannot stand may be tested for a pronator drift in the sitting position.
  - In either case, a normal person can hold this arm position well.
- POSITIVE drift is the pronation of one forearm and drift laterally and inferiorly.
- It is both sensitive and specific for a corticospinal tract lesion originating in the contralateral hemisphere as in CVA.
- Weakness from UMN lesion



https://neuroanatomy.ca/

Muscle Strength

- Normal people vary widely in their strength
  - Your standard of normal should allow for such variables as age, sex, and muscular training.
  - A person's dominant side is usually slightly stronger than the other side.
- Test muscle strength by asking the patient to move actively against your resistance or to resist your movement.
  - Remember that a muscle is strongest when shortest, and weakest when longest
- Impairment: weak is called: paresis, absence of strength paralysis
  - Hemiparesis – weakness on one half of the body
  - Hemiplegia – paralysis of one half of the body
  - Paraplegia – paralysis of the legs
  - Quadriplegia – paralysis of all 4 limbs

Appendix 11

Documentation for grading muscle strength

- Muscle strength is graded on a 0 to 5 scale:
  - 0—No muscular contraction detected
  - 1—A barely detectable flicker or trace of contraction
  - 2—Active movement of the body part with partial gravity eliminated
  - 3—Active movement against gravity
  - 4—Active movement against gravity and some resistance
  - 5—Active movement against full resistance without evident fatigue. This is normal muscle strength.

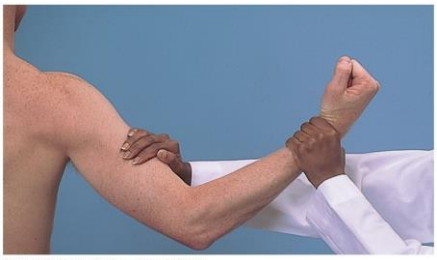
Neurologic- Testing major muscle groups

- Test the following **muscle groups and movements:**
  - **Biceps and triceps, wrist** – flexion and extension at the elbow and wrist extension
  - **Handgrip, finger** – abduction and adduction, **thumb** opposition
  - **Trunk** – flexion, extension, lateral bending
  - **Thorax** – expansion, diaphragmatic excursion during respiration
  - **Hip** – flexion, extension, abduction, and adduction
  - **Knee and ankle** – flexion, extension

Appendix 11

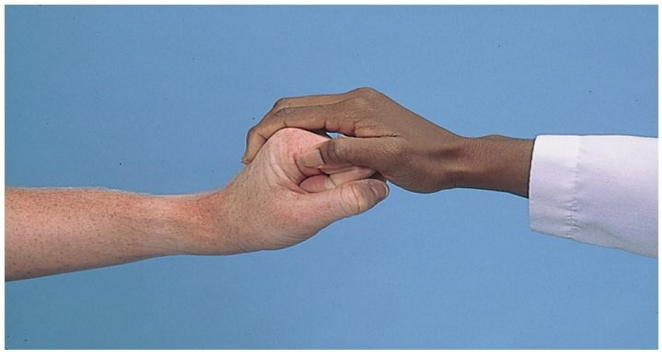
Bicep and Triceps

- Flexion (C5, C6—biceps) and extension (C6, C7, C8—triceps) at the **elbow**
  - Have the patient pull and push against your hand.



Wrist

- Extension at the **wrist** (C6, C7, C8, radial nerve—extensor carpi radialis longus and brevis) and flexion
  - ask the patient to make a fist and resist your pulling it down.



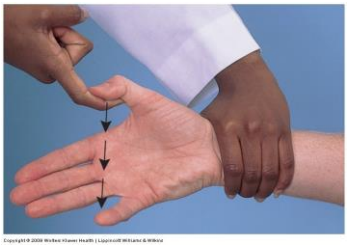
Appendix 11

Grip

- **Grip** (C7, C8, T1).
  - Ask the patient to squeeze two of your fingers as hard as possible and not let them go.
  - Testing both grips simultaneously with arms extended or in the lap facilitates comparison.
    - Weak grip in cervical radiculopathy, de Quervain's tenosynovitis, carpal tunnel, epicondylitis, arthritis

Hands

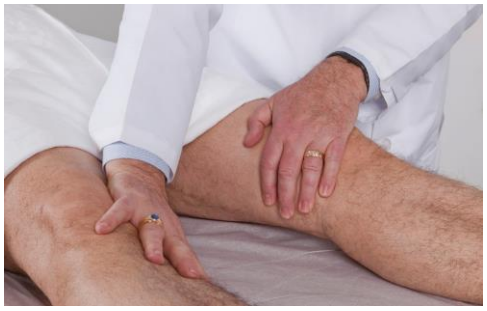
- **Finger abduction** (C8, T1, ulnar nerve).
  - Position the patient's hand with palm down and fingers spread. Instructing the patient not to let you move the fingers, try to force them together.
    - Deficit in ulnar nerve disorders
- Adduction- tested by placing examiners fingers between patients and having the patient squeeze.
- **Opposition of the thumb** (C8, T1, median nerve).
  - The patient should try to touch the tip of the little finger with the thumb, against your resistance
    - Deficit in median nerve disorders as carpal tunnel

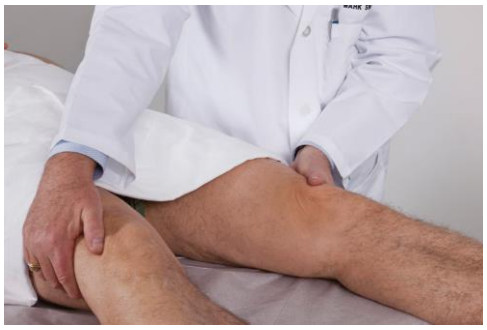


Appendix 11

HIP

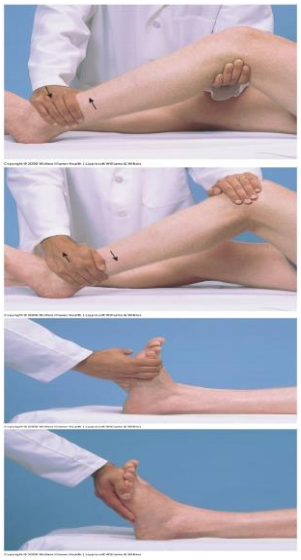
- Flexion at the **hip** (L2, L3, L4—iliopsoas)
  - Placing your hand on the patient's thigh and asking the patient to raise the leg against your hand.
- Extension at the **hips** (S1—gluteus max).
  - Have the patient push the posterior thigh down against your hand.
- Adduction at the **hips** (L2, L3, L4—adductors).
  - Place your hands firmly on the bed between the patient's knees. Ask the patient to bring both legs together
- Abduction at the **hips** (L4, L5, S1—gluteus med and min).
  - Place your hands firmly on the bed outside the patient's knees. Ask the patient to spread both legs against your hands.





Knee and ankle

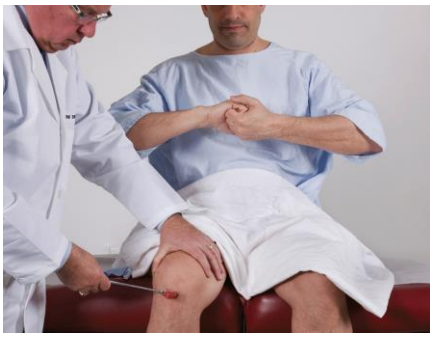
- Extension at the **knee** (L2, L3, L4—quadriceps).
  - Support the knee in flexion and ask the patient to straighten the leg against your hand. The quadriceps is the strongest muscle in the body, so expect a forceful response.
- Flexion at the **knee** (L4, L5, S1, S2—hamstrings)
  - Place the patient's leg so that the knee is flexed with the foot resting on the bed. Tell the patient to keep the foot down as you try to straighten the leg.
- Dorsiflexion (mainly L4, L5—tibialis anterior) and plantar flexion (mainly S1—gastrocnemius, soleus) at the **ankle**
  - Ask the patient to pull up and push down against your hand



Appendix 11

Deep Tendon Reflexes (DTR's)

- Reflex is an involuntary stereotypical response that involves both sensory and motor neurons
- **Encourage the patient to relax;** position the limbs properly and symmetrically
  - Reinforcement – if reflexes are symmetrically diminished or absent, this is a technique involving isometric contraction of other muscles that may increase reflex activity.
- **Hold the reflex hammer loosely** between your thumb and index finger so that is swings freely in an arc
- Strike the tendon with a **brisk direct movement**; use the minimum force needed to obtain a response
  - Use the broad portion of the hammer for larger broad tendons and the narrow portion for the small tendons
- Use **reinforcement** when needed
- Grade the response



DTR Grading

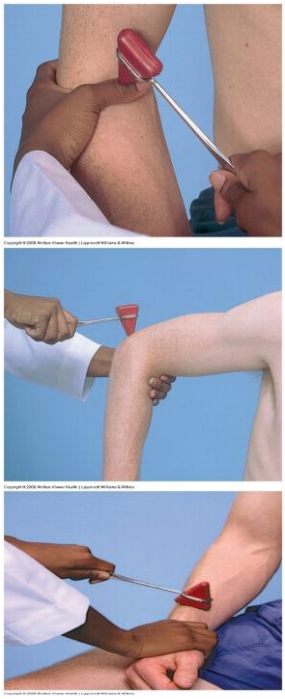
Reflexes are usually graded on a 0 to 4+ scale

- 4+ Very brisk, hyperactive, with *clonus*
- 3+ Brisker than average; possibly but not necessarily indicative of disease
- 2+ Average; normal
- 1+ Somewhat diminished; low normal
- 0 No response

Appendix 11

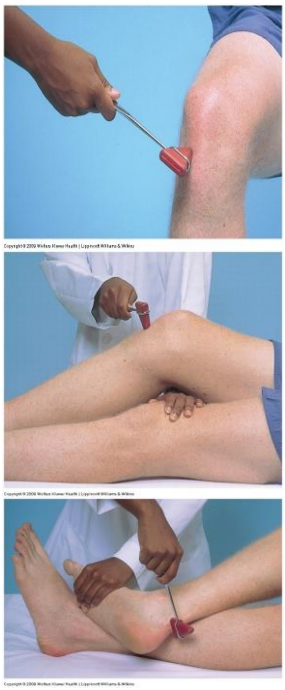
DTR's Arm

- **Deep tendon reflexes** with record levels for each response helps localize any abnormalities
  - **Biceps reflex** (C5-6)
    - Thumb on biceps tendon
  - **Triceps reflex** (C6-7)
    - Strike the triceps tendon itself
  - **Supinator** or **brachioradialis** (C5-6)
    - Forearm slightly pronated strike radius 1 -2 inches proximal to the wrist



DTR's Leg

- **Knee reflex** (L2-4)
  - Strike the patellar tendon
- **Ankle reflex** (primarily S1)
  - Dorsiflex ankle and strike Achilles tendon
- **Clonus:** if the reflexes seem hyperactive (4+), Ankle clonus should be tested by repeatedly dorsi- and plantar flexing the foot, and then sharply dorsiflex and maintain. Rhythmic oscillations between dorsiflexion and plantar flexion during exam.
  - Seen in CNS disease if sustained



Appendix 11

DTR's Abnormalities

- Hyperactive reflexes (hyperreflexia) in central nervous system lesions along the descending corticospinal tract.
  - Look for associated upper motor neuron findings of weakness, spasticity, or a **positive Babinski** sign.

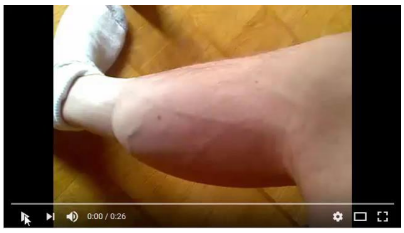
DTR's Abnormalities

- Hypoactive or absent reflexes (hyporeflexia) in diseases of spinal nerve roots, spinal nerves, plexuses, or peripheral nerves.
  - Look for associated findings of lower motor unit disease, namely weakness, atrophy, and fasciculations.

Appendix 11

DTR's Abnormalities

- Hypoactive or absent reflexes (hyporeflexia) in diseases of spinal nerve roots, spinal nerves, plexuses, or peripheral nerves.
  - Look for associated findings of **lower motor unit disease**: weakness, atrophy, and fasciculations.



Cutaneous stimulation reflexes

- **Babinski or Plantar response (L5,S1)**
- With an object stroke the lateral aspect of the sole from the heel to the ball of the foot, curving medially across the ball.
  - Note movement of the big toe
    - Normal = plantar flexion (down).
  - Dorsiflexion of the big toe is a positive **Babinski** response from a central nervous system lesion in the corticospinal tract; also seen in unconscious states from drug or alcohol intoxication or the postictal period following a seizure.
- Some patients withdraw from this stimulus by flexing the hip and the knee.
  - Hold the ankle, if necessary, to complete your observation.
  - It is sometimes difficult to distinguish withdrawal from a Babinski response.

Cutaneous stimulation reflexes

- Anal Reflex (S2-4) "Anal Wink"
  - Using a dull object, such as a cotton swab, stroke outward in the four quadrants from the anus.
    - Watch for reflex contraction of the anal musculature.
- Loss of the anal reflex suggests a lesion in the S2-4 reflex arc, as in a cauda equina lesion, spinal cord injury, spina bifida, or tumor at the lower sacral segment

Weakness: Abnormality of movement as strength

UMN damage produces **spastic paralysis or weakness**

- Increased reflexes and tone (hyperreflexia, hypertonicity)
- Upward going Babinski's sign
- No atrophy
- No fasciculations (twitching).
- Stroke (Cerebrovascular accident)

Weakness: Abnormality of movement as strength

UMN damage produces **spastic paralysis or weakness**

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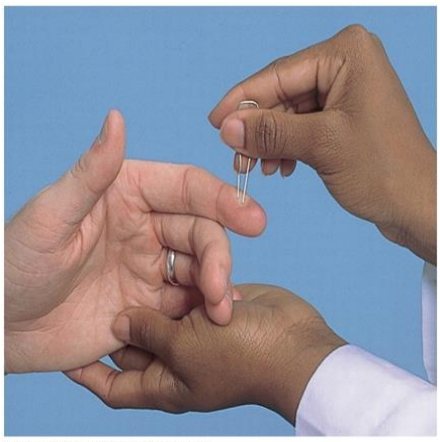
Physical



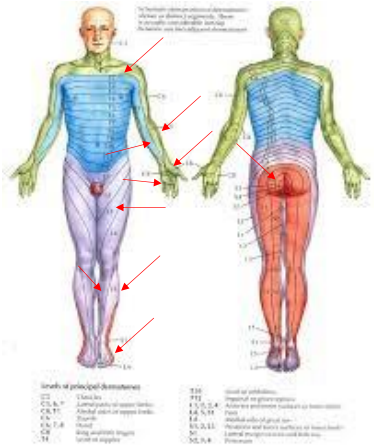
Sensory Exam

- **Sensory tract function**
  - Loss of fine touch & proprioception vs.
  - Loss of pain & temperature sensation
  - Complete anesthesia

Appendix 11

Sensory System-Test

- **Pain, temperature,** and **touch, compare distal with proximal areas** of the extremities :
  - 1. Pain and temperature (**Spinothalamic tracts/ALS**)
  - 2. Position and vibration (**Posterior columns/PCML**)
  - One **suggested pattern** includes
    - Shoulders (C4)
    - Inner and outer aspects of the forearms (C6 and T1)
    - Thumbs and little fingers (C6 and C8)
    - Fronts of both thighs (L2)
    - Medial and lateral aspects of both calves (L4 and L5)
    - Little toes (S1)
    - Medial aspect of each buttock (S3).
- Compare **symmetric areas** on both sides of the body
  - **Map out the boundaries** of any area of sensory loss or hypersensitivity
- When you detect abnormal findings, **correlate them with motor and reflex activity**.
- Assess the patient carefully as you consider the following Questions
  - Is the underlying lesion central or peripheral?
  - Is the sensory loss bilateral or unilateral?
  - Does it have a pattern suggesting a dermatomal distribution, a polyneuropathy, or a spinal cord syndrome with a loss of pain and temperature sensation but intact touch and vibration?



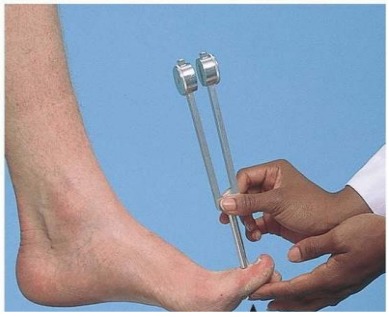
Pain and Temperature

- Inform the patient what you are about to perform
- The patient's eyes should be closed during test
- **Pain**: use a **disposable object** such as a broken cotton swab or pin and discard after each use.
  - Ask if prick is **sharp or dull?**
- One suggested pattern includes
  - shoulders (C4)
  - inner and outer aspects of the forearms (C6 and T1)
  - thumbs and little fingers (C6 and C8)
  - fronts of both thighs (L2)
  - medial and lateral aspects of both calves (L4 and L5)
  - little toes (S1)
  - medial aspect of each buttock (S3).
- If pain exam is normal temperature testing if often omitted
- **Temperature:** use warm and cold objects

Appendix 11

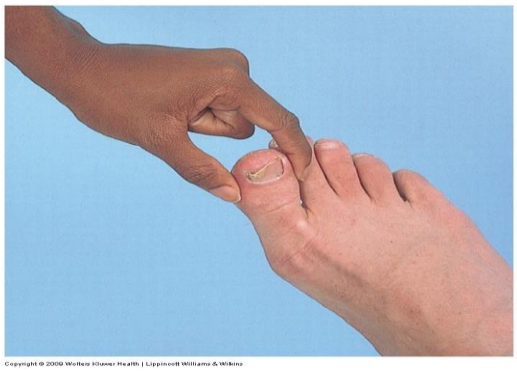
Light Touch and Vibration

- **Light touch**, Examiners fingers: ask the patient to **compare 2 sensations:** "Does this feel the same on both sides?"
  - Test over the suggested pattern
    - Anesthesia
    - Hyperesthesia
    - Hypoesthesia
- **Vibration**: tap a 128-Hz tuning fork on your hand, then place it on the **DIP joint of the patient's finger**. Ask the patient, "Do you feel a buzz? Tell me when it stops." Likewise test over the **joint of the big toe**.
  - If vibration sense is impaired, proceed to more proximal bony prominences (e.g., wrist, elbow, medial malleolus, patella, spinous processes, and clavicles).
  - If felt distally it is safe to assume proximal are normal
  - Vibration sense is often the first sensation to be lost in peripheral neuropathy.
    - Causes: Diabetes and alcoholism



Proprioception

- **Proprioception**: hold the big toe by its sides between your thumb and index finger, pull it away from the other toes, and move it up then down. Ask the patient to identify the direction of movement. (Repeat with patient's eyes closed)
  - Loss of position sense due to:
    - Posterior column disease – tabes dorsalis, Multiple Sclerosis, or Vitamin B12 deficiency
    - Peripheral neuropathy - diabetes



Application 11

Discriminative Sensations

- **Two-point discrimination**: using two ends of an opened paper clip, or two pins, touch the finger pad in two places simultaneously; ask the patient to identify 1 touch or 2
  - Normally the minimal distance discrimination is 5 mm or less on the finger pads
  - Digital nerve - laceration

Discriminative Sensations Exams

- **Stereognosis**: place a key or familiar object in the patient's hand and ask the patient to identify it (limited by motor impairment, arthritis)
  - Normally a patient will manipulate the object and identify it within 5 seconds
  - Astereognosis– refers to the inability to recognize objects place in the hand
- Number identification (**graphesthesia**): outline a large number in the patient's palm and ask the patient to identify the number
  - Normally a patient can identify most numbers
  - Abnormalities suggest a lesion in the sensory cortex
- **Point localization**: lightly touch a point on the patient's skin and ask the patient to point to that spot. Especially useful on trunk and legs
  - Normally this can be done
- **Extinction**: touch an area on both sides of the body at the same time and ask if the patient feels 1 spot or 2
  - Normally both stimuli are felt.
  - A sensory cortex lesion the stimulus opposite the damaged cortex is extinguished (lost)

Appendix 11

Extinction

- **Extinction**: touch an area on both sides of the body at the same time and ask if the patient feels 1 spot or 2
  - Normally both stimuli are felt.
  - A sensory cortex lesion the stimulus opposite the damaged cortex is extinguished (lost)

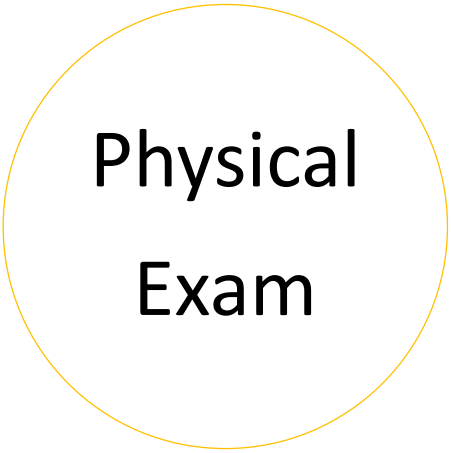
Stereognosis

Graphesthesia

2 pointdiscrimination

Appendix 11

From Complaint to Diagnosis



- Mental status
- Cranial nerves
- **Motor**
  - Reflexes
- Sensation
- **Coordination**
  - **Movement-volition & coordination**
- Special tests

Motor-Movement as Volition and Coordination

- **Inspect**: -atrophy, involuntary movements, muscle tone and bulk
  - Body position
    - At rest and with movement– weakness or paralysis
  - Involuntary movements (tremors, tics, fasciculations)
    - 1. **Tremor** – rhythmic oscillatory movements
      - A. Resting (static): occur at rest and disappear with voluntary movement. Example: Parkinson's disease
      - B. Postural: appear when the affected part is actively maintaining a posture. Example: benign essential tremor, anxiety.
      - C. Intention: absent at rest and appear with voluntary movement and worsen as the target of movement is neared: cerebellar disorders
    - 2. **Tics** – brief, repetitive, stereotyped, coordinated movements occurring at irregular intervals.
      - Example: repetitive blinking, grimacing, shoulder shrugging. Causes: Tourette's, drugs, other
    - 3. **Dystonia** – involve larger portions of the body. Grotesque, twisted postures.
      - Causes: drugs, spasmodic torticollis
    - 4. **Athetosis** – slower and more twisting and writhing than chorea with larger amplitude. Typically involve the face and distal extremities.
      - Causes: include Cerebral Palsy
    - 5. **Chorea** – brief, rapid, jerking, irregular, and unpredictable. At rest and interrupt normal movement. Seldom repeat unlike Tics.
      - Causes: Sydenham's chorea, Hunington's Disease

Appendix 11

Motor- Coordination

- Coordination of muscle movement requires that four areas of the nervous system function in an integrated way:
  1 The motor system, for muscle strength
  2 The cerebellar system (also part of the motor system), for rhythmic movement and steady posture
  3 The vestibular system, for balance and for coordinating eye, head, and body movements
  4 The sensory system, for position sense

Coordination Exam- **Cerebellar**

- **Rapid alternating movements**:
  - Arms:
    - 1. patient turns hand rapidly over and back on thigh
    - 2. taps tip of index finger rapidly on distal thumb
  - Legs:
    - 3. taps ball of foot rapidly on your hand
  - **Dysdiadochokinesis** – found in cerebellar disease when one movement cannot be followed quickly by its opposite, and movements are slow. (Abnormal rapidly alternating movements)

- **Point-to-point movements**
  - **1. Finger-to-Nose test -** patient touches nose then your index finger as you move it to different positions
  - 2. **Heel-to-Shin** - patient moves heel from opposite knee down the shin to the big toe
  - **Dysmetria** – found in cerebellar disease with difficulty coordinating movements by overshooting or undershooting its mark. (Abnormal Finger-to-Nose or past pointing with finger to nose exam)

Appendix 11

Coordination-Gait

- **Gait** – assess gait as patient:
  - Walks **across room**
  - Walks **heel-to-toe**
  - Walks on **toes then heels**
    - May reveal distal muscle weakness in the legs and is a sensitive test for Corticospinal tract damage
  - **the patient is in danger of falling.**
- Ataxia – a gait that lacks coordination and is unsteady.
  - May be seen in cerebellar disease, loss of position sense, intoxication, and other

Romberg test

- This is mainly a test of position sense.
- The patient should first stand with feet together and eyes open and then close both eyes for 30 to 60 seconds without support.
  - Loss of balance when eyes closed is a **positive test**
- In ataxia from dorsal column disease and loss of position sense, vision compensates for this sensory loss when vison is remove the abnormality is evident
- In cerebellar ataxia, the patient has difficulty standing with feet together whether the eyes are open or closed.

https://neuroanatomy.ca/

Appendix 11

Romberg test

- This is mainly a test of position sense.
- The patient should first stand with feet together and eyes open and then close both eyes for 15 seconds without support.
  - Loss of balance when eyes closed is a **positive test**
- In ataxia from dorsal column disease and loss of position sense, vision compensates for this sensory loss when vison is remove the abnormality is evident
- In cerebellar ataxia, the patient has difficulty standing with feet together whether the eyes are open or closed.

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From Complaint to Diagnosis

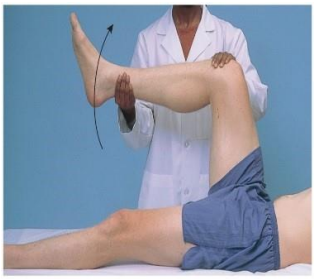
Physical Exam

- Mental status
- Cranial nerves
- **Motor**
- Sensation
- Coordination
- **Special tests**

Appendix 11

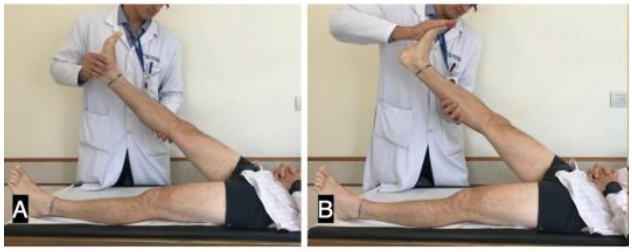
Special Tests and Diseases

- Meningitis:
  - An inflammation of the brain tissue (meninges) due to subarachnoid hemorrhage or infection with bacteria, virus, fungal, or other. Associated with HA, fever, myalgias
  - **Meningeal Signs:**
    - **1. Nuchal rigidity –** check for resistance to mobility of the neck. With patient supine flex the neck forward until chin touches chest or as far as possible.
      - Normal neck is supple
      - Abnormal– resistance to flexion in 90% of patients with acute bacterial meningitis.
    - **2. Brudzinski's sign** – while flexing the neck watch for hip or knee flexion.
      - **Positive** Brudzinski is flexion of the hips and or knees suggesting inflammation
    - **3. Kernig's sign** – flex the patient's leg at the **k**nee and hip and then straighten.
      - **Positive** Kernig's is pain and increased resistance to extending the knee



Special Tests and Diseases

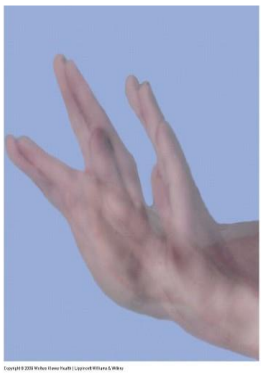
- Lumbosacral Radiculopathy:
  - If the patient has low back pain with nerve pain that radiates down the leg
  - Test straight-leg raising on each side
    - While the patient is supine raise the patient's relaxed and straightened leg, flexing the leg at the hip, then dorsiflex the foot
    - **Positive sign** – is pain radiating into the ipsilateral leg
    - **Crossed straight leg positive** – is seen when the contralateral (healthy) leg is raised and causes increased pain
      - Note the degree of elevation at which pain occurs **(**e: 30 degrees)
    - More than 95% of disc herniation's occur at L5-S1, where the spine angles is sharply posterior.



Appendix 11

Special Tests and Diseases

- Asterixis:
  - Helps identify metabolic encephalopathy in patients when mental functions are impaired.
  - Ask the patient to "stop traffic" by extending both arms, with hands cocked up and fingers spread.
  - Watch for 1 to 2 minutes, coaxing the patient as necessary to maintain this position.
  - Sudden, brief, nonrhythmic flexion of the hands and fingers indicates asterixis
  - Seen in liver disease, uremia, and hypercapnia.

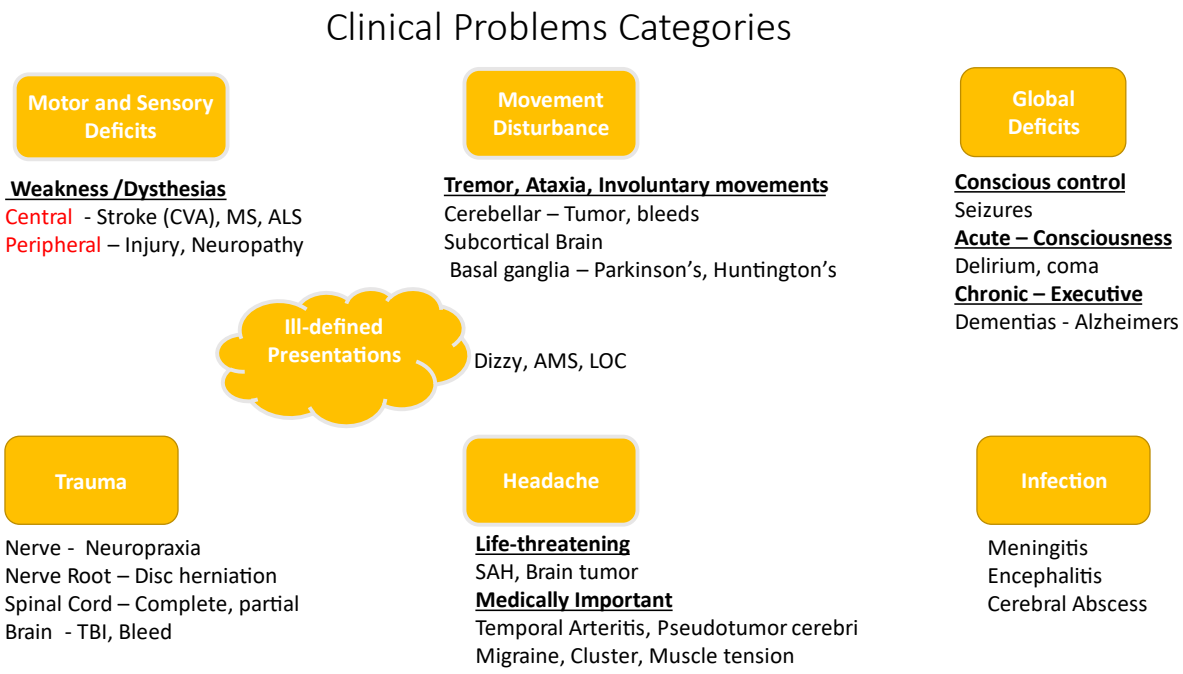


From Complaint to Diagnosis

Differential Diagnosis

- Most common and most likely
- Can't miss dxs
- All other reasonable possibilities

Appendix 11



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Appendix 12

Neuro Case studies

1a. Chief complaint: Sudden onset of weakness

- **History of present illness** 64-year-old male complains of sudden onset of weakness to the right side of his right face and arm. The patient's wife also says he is also not speaking normally. The patient denies any headache, trauma, fever or other complaints of time of examination.
- Sxs began about 45 min. prior to arrival.

Appendix 12

1a. Chief complaint: Sudden onset of weakness

- **Past medical history** – Type II diabetes currently taking metformin, insulin; hypertension, 20 pack year history of tobacco use (discontinued three years ago), coronary artery disease with stent x1 three years ago.
- **Past surgical history** - Appendectomy age 14
- **Social history** - Retired truck driver, No EtOH or tobacco use
- **Family medical history** - father died of mi at rage 52, the patient's brother had "small stroke" at age 60
- **Medications** - Lipitor, enalapril, Viagra, multivitamin, ASA
- **Allergies** - No known drug allergies

1a. Chief complaint: Sudden onset of weakness

- **Physical examination**
- **Vital signs:** T 98.6, Pulse 112, BP 166/90, Resp 18 SaO2 98% Room
- **General exam**-well-developed well-nourished male in moderate acute distress
- Alert and aphasic
- **HEEENT**-Head atraumatic, eyes pupils- perrl, extraocular muscles intact; Fundi benign
- Face-

Appendix 12

1a. Chief complaint: Sudden onset of weakness

- **Physical examination**
- **Neck** supple
- **Chest** - clear to auscultation bilaterally, heart-irregularly irregular rhythm, No Murmur, rub or gallop; rate of 114
- **Abd** soft and NT
- **Extremities**- Strength right arm 1/5 all muscle groups, pronator drift– falls immediately with gravity, Right leg strength 4/5 all muscle groups, Decreased sensation in all modalities to right arm & face

Appendix 12

1a. Chief complaint: Sudden onset of weakness

- **Physical examination**
- **Neck** supple
- **Chest** - clear to auscultation bilaterally, heart-irregularly irregular rhythm, No Murmur, rub or gallop; rate of 114
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1a. Chief complaint: Sudden onset of weakness

- Differential diagnosis?

Appendix 12

1a. Chief complaint: Sudden onset of weakness

- Differential diagnosis?

1. Ischemic stroke
2. Hemorrhagic stroke
3. Transient ischemic attack
4. Brain tumor
5. Cerebral abscess
6. Seizure, atypical
7. Hypoglycemia
8. Atypical migraine
9. Delirious tremens – etoh
10. WK syndrome

1a. Chief complaint: Sudden onset of weakness

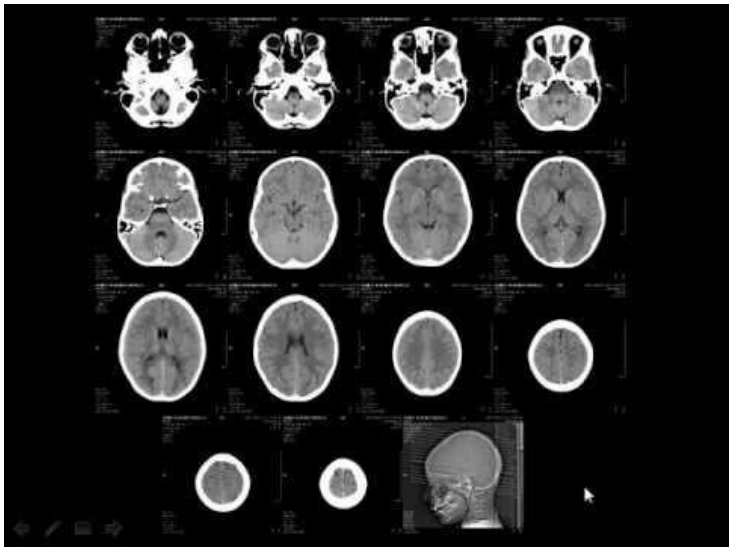
- Emergency Department workup?

Appendix 12

1a. Chief complaint: Sudden onset of weakness

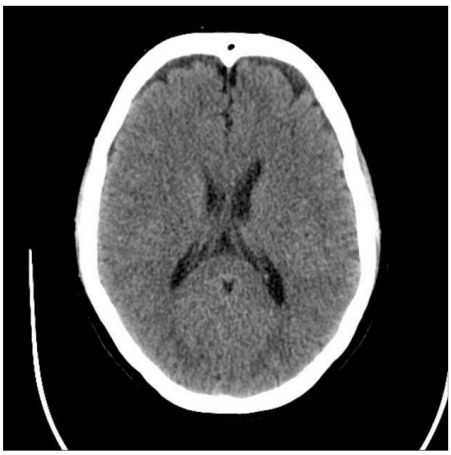
- Emergency Department workup?
- CT Head without contrast
- CBC, Chem 8, PT/INR, EKG
- FS Glucose – 133
- EKG – Afib rate 122

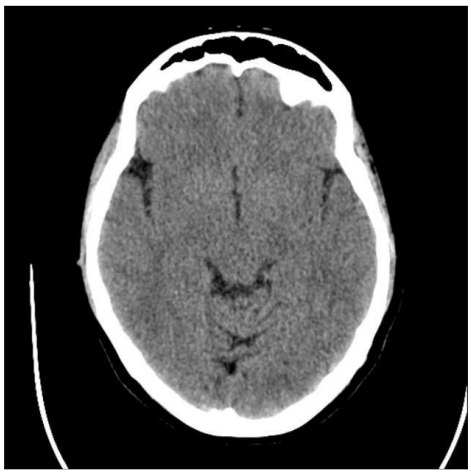
1a. Chief complaint: Sudden onset of weakness



Appendix 12

1a. Chief complaint: Sudden onset of weakness



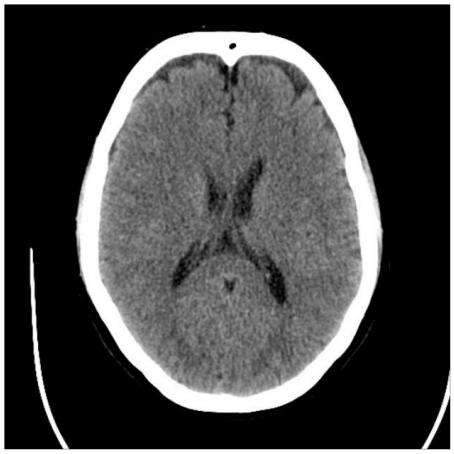


1a. Chief complaint: Sudden onset of weakness

- Emergency Department workup?
- CBC
  - WBC – 10.4
  - Hgb 11.2
  - HCT 40%
  - PLT – 167,000
- Chem 8
  - Gluc 134
  - BUN – 17
  - CO2 – 22
  - CL-106
  - K+ - 4.0
  - Na+ - 136
- PT- 11 seconds
- INR – 1.0

Appendix 12

1a. Chief complaint: Sudden onset of weakness



- Report:
- Negative CT Head

1a. Sudden onset of weakness
NIHSS Stroke Scale

- 1a Level of consciousness-alert, keenly responsive
- 1b Month-and age-aphasic
- 1c - Performs one task
- 2-Normal horizontal extraocular movements
- 3 Test visual fields-partial hemianopia
- 4: Flat nasolabial fold, smile asymmetry
- 5 a. Left arm motor drift-no drift for 10 seconds
- 5B-Test right arm motor drift-no effort against gravity
- 6a-Test left leg motor drift-no drift for five seconds
- 6 b-Test right leg for motor drift-drift but doesn't hit bed
- 7 a Test limb ataxia using f in f/heal-shin) no ataxia to left limb and right upper extremity not testable due to paralysis, right lower extremity normal
- 8 Test sensation-mild to moderate loss have sharp and dull, can tell being touched
- 9 Test language/aphasia-using characters in print on standard text describe the scene and asked patient name the words and read the sentences. Global aphasia
- 10 Test for dysarthria-anarthric
- 11: Test extinction/inattention-No abnormality

Appendix 12

IVtPA (Alteplase) Criteria

Inclusion

Age> 18 yo

Stroke Symptom onset< 3 hour

Clinical diagnosis of ischemic stroke causing a measurable neurological deficit

> 4 qualifies for IVtPA and > 25 too severe for IV tPA

Exclusion

Stroke or serious head trauma < 3 months
Major surgery < 14 days
History of ICH
SBP > 185mm Hg, DBP > 110mm Hg or aggressive treatment required
Rapidly improving or minor neurological symptoms
Symptoms suggestive of SAH
GI or urinary tract hemorrhage in the last 21 days
Arterial puncture < 7 days at a non-compressible site
Seizure at onset (though not an absolute exclusion criteria)
INR > 1.7 or PT > 15 seconds
Platelet count < 100, 000/mm3
Glucose < 50 mg/dl or > 400 mg/dl
Current use of oral anticoagulant (direct thrombin or factor Xa inhibitor with last use within 48 hours)

1a. Chief complaint: Sudden onset of weakness

- IVtPA given
  - Symptoms resolved within 1.5 hours
  - NO headache or mental status changes
  - Now what?
- Inpatient workup
- Treat Afib
- Find exact source of stroke
  - Thrombotic
    - Atherosclerotic plaque with thrombus
  - Embolic
    - Thrombus travels from orginal site
    - Cardioembolic

Appendix 12

Inpatient Stroke Work-Up

Most all patients will get:

- Imaging to confirm presence of infarction:

-MRI brain (without contrast)

-if not MRI candidate, repeat CT scan

- A Blood Vessel Study

CTA Head and Neck

MRA Head and Neck

Carotid Ultrasound*

- 2D Echocardiogram with Bubble study
- Lipid profile, LFTs, hemoglobin A1C

- Some patients may get additional testing:

-TEE (trans-esophageal echo)

-Hypercoagulable Panel (very expensive)

-Cerebral arteriogram

If vessel anatomy needs clarification

-MRI brain with contrast (tumor? MS? abscess?)

Inpatient Stroke Work-Up

-MRI brain (without contrast)

Negative except for age associated cerebral atrophy

- A Blood Vessel Study

CTA Head and Neck

Negative except for diffuse atherosclerotic changes, no critical stenosis

- **2D Echocardiogram with Bubble study**
  - EF 50%, otherwise negative
- **Lipid profile, LFTs, hemoglobin A1C**
  - Consistent with history of diabetes

- Some patients may get additional testing:

-TEE (trans-esophageal echo)

Small thrombus on atrial appendage

Appendix 12

1a. Chief complaint: Sudden onset of weakness

- DDX
  - Ischemic stroke
    - S/P IVtPA with no residual deficit
  - Atrial fibrillation
- What was the vascular distribution of the patient's initial symptoms?
- What is the difference between thrombolysis, anticoagulation and antiplatelet therapy?
- What is the patient's and primary care provider's responsibility upon discharge?

Appendix 12

2a. Chief complaint: Sudden change in mental

- **History of present illness**
- 77-year-old Hmong Female was brought in by ambulance. By her family's report the patient was weeping loudly after being told that her son had recently passed away when she experienced a sudden onset of mental status change. Paramedics report that The patient is unresponsive to verbal stimulus her eyes are partially opened . Paramedics have placed the patient on nasal cannula oxygen, vital signs are stable with the exception of blood pressure which is 202/110. The patient has a long-standing history of hypertension.

2a. Chief complaint: Sudden change in mental

- **History of present illness**
- 77-year-old Hmong Female
- No preceding s/s - No fever, no chest pain, no focal weakness, no palpitations, no convulsions or sensorium changes prior to unresponsiveness, no trauma or mechanism of injury, no known substance abuse, no other medical history other than HTN, in US for 3 years

Appendix 12

2a. Chief complaint: Sudden change in mental

- **History of present illness**
- 77-year-old Hmong Female was brought in by ambulance. By her family's report the patient was weeping loudly after being told that her son had recently passed away when she experienced a sudden onset of mental status change. Paramedics report that The patient is unresponsive to verbal stimulus her eyes are partially opened . Paramedics have placed the patient on nasal cannula oxygen, vital signs are stable with the exception of blood pressure which is 202/110. The patient has a long-standing history of hypertension.

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- 77-year-old Hmong Female
- No preceding s/s - No fever, no chest pain, no focal weakness, no palpitations, no convulsions or sensorium changes prior to unresponsiveness, no trauma or mechanism of injury, no known substance abuse, no other medical history other than HTN, in US for 3 years

2a. Chief complaint: Sudden change in mental

- **History of present illness**
- 77-year-old Hmong Female
- Vital signs
  - Pulse 84
  - Resp 18
  - Sao2 95% @l O2 NC
  - BP: 198/92
  - Temp 98.4
- ABC's
  - Pt. unresponsive but breathing spontaneously and maintaining oxygenation
- "DONT Forget"
  - FS Glucose – 144
  - Oxygen – 95%
  - Narcan – 0.4mg IV – no response

2a. Chief complaint: Sudden AMS

GCS

Eyes –Open to pain

Talks – Incomprehensible sounds

Motors – Flexion to pain

- Score and Significance?

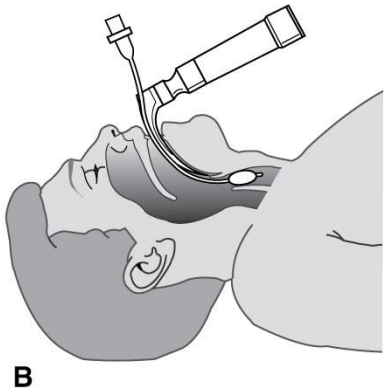
2a. Chief complaint: Sudden AMS

GCS = 7 Cannot protect airway

Endotracheal intubation

Rice in posterior oropharynx and around glottis

Intubation without problem



B

2a. Chief complaint: Sudden AMS

Repeat physical exam and order emergent testing

12 lead EKG

CT Head without contrast

CMP

CBC

PT/PTT

Cardiac enzymes

Tox panel

HEENT – NCAT

EYES – Pupils unresponsive bilaterally in mid fixed position

Fundoscopic – no SVP seen, questionable early papilledema bilaterally, no other acute findings

Neck supple

Chest CTAB

CV – rrr without m/r/g

NO signs of trauma

Appendix 12

2a. Chief complaint: Sudden AMS

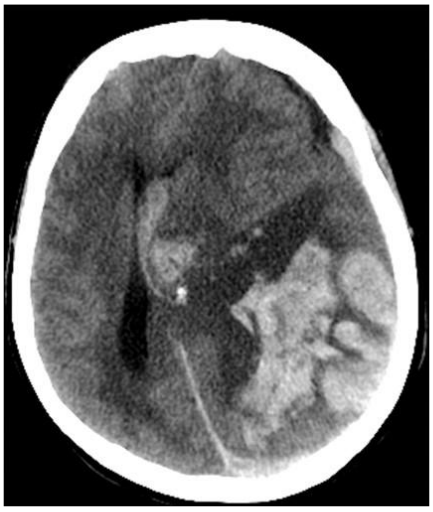
What is your ddx?

COMA
Etiologies

• **V**ascular	• Strokes/**Bleed**, CV collapse
• **I**nfection	• CNS infection, Sepsis
• **N**eoplasm	• Mass with herniation
• **D**rugs	• Opiates, Benzos, toxic effects
• **I**diopathic	• Seizures (prolonged)
• **C**ongenital	
• **A**utoimmune	
• **T**rauma	• TBI, traumatic bleeds
• **E**nvironment/**E**ndocrine-Metab	• Hypothermia, Gluc, electrolyes

Appendix 12

#2a Chief complaint:LOC



2a. Chief complaint: Sudden AMS

Emergent testing

12 lead EKG - nsr, no acute changes

CT Head without contrast - image

CMP - wnl

CBC - wnl

PT/PTT - wnl

Cardiac enzymes - wnl

Tox panel – all negative

CXR – WNL ET tube midline with proper placement

Appendix 12

COMA
Etiologies

• **V**ascular	~~• Strokes/**Bleed**, CV collapse~~
• **I**nfection	~~• CNS infection, Sepsis~~
• **N**eoplasm	~~• Mass with herniation~~
• **D**rugs	~~• Opiates, Benzos, toxic effects~~
• **I**diopathic	~~• Seizures (prolonged)~~
• **C**ongenital	
• **A**utoimmune	~~–~~
• **T**rauma	~~• TBI, traumatic bleeds~~
• **E**nvironment/**E**ndocrine-Metab	~~• Hypothermia, Gluc, electrolyes~~
	~~–~~

2a. Chief complaint: Sudden AMS

What is your Dx?

What is your plan?

Neurosurgery consult
ICU Neuro exam

Appendix 12

2a. Chief complaint: Sudden AMS

- Pupillary reflex – Both pupils dilated midfixed position and unresponsive to light
- Corneal reflex – No blinking when either eye is dropped with sterile saline
- Doll's eyes test –Eyes stay fixed with axis of face
- Caloric test – 50cc of ice water in each ear provokes no movements of eyes

2a. Chief complaint: Sudden AMS

- Cerebral Flow Study – Negative for flow
- May consider EEG (electroencephalogram)
  - No activity
- Ventilator respiratory rate and volume set to allow PaCO2 of 60mmHg
- NO intrinsic respiratory drive

Appendix 12

2a. Chief complaint: Sudden AMS

- What is the patient's neurological status?
- What is the prognosis?
- What is a reasonable course of action for this patient?
- Process for compassionate withdrawal of care
- Family agrees
- Family Disagrees

Appendix 13

OSCE Neuro (Headache Patient form)

Clarified Chief complaint "I woke up about 4 o'clock this morning with this horrible pounding headache."

HPI (8)

- Site "Both sides of the front of the head"
- Onset "I woke up at 4am with the headache…suddenly I guess."
- Character "Throbbing or pounding"
- Radiation "No"
- Associated signs and symptoms "I threw up once when I first woke up and I feel nauseated now."
    - Photophobia, phonophobia
    - Visual changes, speech changes, difficulty walking
    - Fever
- Timing (#minutes, hours, day, weeks) "12 hours - I have never had headaches like this before."
- Exacerbating & Alleviating factors "I took ibuprofen and Tylenol-none of that helped. The light seems to make it worse."
- Severity (1-10) "10" "Worst headache i have ever had."

- PMHx (3)
    - Illnesses, diseases or conditions "No"
    - Current medication "Birth control pills"
    - Medication allergies "No drug allergies"
- FMHx (2)
    - Mother or Father Father – "Dad is healthy but he has "cysts on his kidneys"; Mother and sisters are alive and healthy
    - Sister or brother
- SocHx (4)
    - Occupation Bar tender
    - Hobbies Yoga
    - Tobacco or alcohol "2 to 3 drinks at a time, and drinks 1 to 2 times per week"; the patient vapes nicotine products as well as cannabinoids but denies other illicit substances.
    - Drugs of abuse - Last exposure to cannabinoids was eight hours before onset of headache.

- ROS: (7)
    - General – Denies Wgt Δ; weakness; fatigue; no fevers
    - Skin – Denies Skin Δ'es; rashes; bumps/lumps; sores; itching; dryness; color change; Δ in hair/nails;
    - Head –Headache noted above; Denies head injury; trauma

- Eyes – Denies Vision Δ; corrective lenses; last eye exam; pain; redness; excessive tearing; double vision; blurred vision; scotoma; dryness of the eyes
- Ears – Denies Hearing loss or Δ; tinnitus; earaches; infections; discharge
- Nose/Sinuses – Denies Frequent colds; congestion; runny nose; discharge; itching; hay fever; nosebleeds; Δ in sense of smell
- Mouth/Throat –Denies sore throat hoarse; problems swallowing; Δ in sense of taste
- Neck Lumps - Denies swollen glands; goiter; pain; neck stiffness
- Pulmonary - Denies Cough; hemoptysis; dyspnea/shortness of breath; wheezing; chest pain; pleuritic pains; frequent lung infections, night sweats
- Cardiac – Denies Chest pain or discomfort; shortness of breath; palpitations; dyspnea; orthopnea; PND; peripheral edema or swelling; exertional chest pain or SOB; cardiovascular testing;
- G/I – Denies Appetite/diet Δ; jaundice, liver/gallbladder/hepatitis problems; nausea/emesis; dysphagia; heartburn/indigestion; pain; hematemesis
- Neuro – Denies Syncope; dizziness; blackouts; seizures; weakness; paralysis; numbness/tingling; tremors; involuntary movements; Δ speech, memory, mood, or thinking; denies previous headaches

Vital signs – T 99.8 F Oral, Pulse-98, Respirations 14, Pulse Oximetry Equals 99% Room Air, blood pressure 124/70

OSCE #2 CORE Exam with Expanded Neuro & BH CA&C II

Date	Student	Instructor

30 MINUTE TIME LIMIT: 1 point deduction for every 2 minutes over this limit

Hands sanitizer (1)
Introduce self and identify patient (1)

HPI (8)
Site
Onset (Sudden, gradual)
Character (Sharp, dull, stabbing)
Radiation
Associated signs and symptoms
Timing (#minutes, hours, day, weeks)
Exacerbating & Alleviating factors
Severity (1-10)

PMHx (3)
Illnesses, diseases or conditions
Current medication
Medication allergies

FMHx (2)

Mother or Father
Sister or brother

SocHx (4)

Occupation
Hobbies
Tobacco or alcohol
Drugs of abuse

ROS: (10)

Head: headache, head injury, head trauma
Eyes: use of glasses/contacts; vision changes, blurry, or double vision; excessive redness, tearing, or dry eyes
Ears: hearing loss, hearing changes, ringing in the ears; ear pain or discharge
Nose: frequent colds, runny nose, congestion of the nose or sinuses; nose bleeds; change in sensation of smell
Throat/mouth: problems with teeth, gums, mouth, or throat; use of dentures; bleeding gums, mouth or teeth; problems with swallowing or sense of taste
Lungs: cough, shortness of breath, chest pain; wheezing, sputum production, hemoptysis; frequent lung infections or problems; night sweats
Mental Health: nervousness, anxiety, depression, suicidal/homicidal thoughts or ideas, suicide attempts and when, problems dealing with stress, change in sleep, or changes in memory
Cardiovascular: Chest pain or discomfort; shortness of breath; palpitations; dyspnea; orthopnea; PND; peripheral edema or swelling; exertional chest pain or SOB; cardiovascular testing; sores that won't heal
Neurologic: changes in speech, memory, mood, or thinking; dizziness, fainting, seizures, or blackouts; weakness, numbness, or loss of sensation; unusual movements or tremors
Student provides differential diagnosis for positive items

Exam: (verbalize inspected items during the inspection but verbalizing what you are palpating, or testing may also help)

Vital Signs: (5)

Sao2
Temperature
Blood pressure
Pulse
Respirations

General (3)

Observed development
Nourishment
Level of distress

Head: (1)

Inspect and palpate skull for symmetry, shape, deformities, and trauma

Eyes: (9)

- Test visual acuity
- Test visual fields
- Inspect position, shape, and symmetry of eyes, conjunctiva, sclerae, and pupils
- Inspect brows, eyelids, and lacrimal system
- Check extraocular eye movements
- Check Cover Test
- Ophthalmoscope exam: Inspect iris, and cornea at 90 degrees
    - Inspect pupil and light reaction & accommodation
    - Inspect optic disc and retina

Ears: (3)

Inspect the auricle shape, size, symmetry, and ear placement
Otoscope exam: Inspect external canal and TM (bilaterally)
Check Auditory Acuity (Finger rub)

Nose: (1)

Inspect position, symmetry, and skin changes

Throat and Mouth: (4)

Inspect lips and mouth for symmetry, color, lesions, and moisture
Inspect teeth, gums, tongue, soft/hard palate, pharynx, and mucosa
Inspect floor of mouth
Ask patient to say "Ah" and assess swallowing mechanism

Neck (3)

Inspect neck for symmetry, contour, masses
Auscultate for bruits
Palpate for masses, lymph nodes, thyroid, trachea

Chest and Lung Exam: (1)

Auscultate for heart and lung sounds

Abdomen: (1)

Inspect, auscultate, and palpate the abdomen in all four quadrants

<u>Neurologic Exam: (13)</u>

- Mental status: Perform Mini-mental status exam (MMSE)

- Perform Cranial nerve test I-XII:
  - I - Smell (Verbalize)
  - II - Visual acuity
  - III - EOM, light reflex, accommodation (object moved from far to near)
  - IV - EOM
  - V
    - Motor: clench teeth, and moves side/side
    - Sensory: light touch and sharp/dull (verbalize)
  - VI- EOM
  - VII- Raises brow, frowns, closes eyes with resistance, shows teeth, puffs cheeks, smile
  - VIII- Auditory acuity (finger rub if not done on ear)
  - IX/X- Clear speech, swallows, palate rises, ask to say "Ah"
  - XI- Shrugs shoulders, turns head each side with resistance
  - XII- Speech is clear, tongue protrudes without deviation

Sensory: (7)

Assess pain sensation by asking "sharp" or "dull"- in suggested pattern (face, extremities and thorax)
Assess light touch using a cotton wisp and compare symmetry- in suggested pattern (facial, extremities, and thorax)
Assess vibratory sense using tuning fork at IP of the hand or foot
Assess proprioception using the patient's big toe
Test for stereognosis using familiar object
Test for graphesthesia by drawing a number in patient's palm
Demonstrate two point discrimination (finger pads)

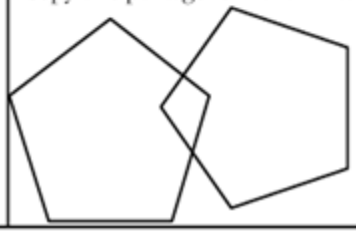
Motor: (Movement-as-strength) (15)

Inspect for atrophy, involuntary movements, muscle tone and bulk
Pronator drift
Assess patient muscle strength in the upper extremity:
Elbow- flexion/extension
Wrist- flexion/extension
Finger abduction/adduction, opposition of thumb, and grip
Reflexes
Biceps or Brachioradialis
Triceps
Patellar
Ankle
Assess patient muscle strength in the lower extremity:
Hip-flexion/extension, abduction, adduction
Knee- flexion/extension
Ankle- flexion/extension
While recumbent

Ankle for clonus
Babinski (Plantar response reflex for UMN lesion)
Assess for Brudzinski, Kernig's sign (meningeal irritation)

Motor: (Movement-as-volition & coordination)

Assess for asterixis (Hand flop from hepatic encephalopathy)
Rapid alternating movements (Dysdiadochokinesia)
Point-to-point movements: either finger-to-nose or heel-to-shin (Dysmetria and ataxia)
Romberg test (Sensory ataxia)
Observe gait (Truncal and gait ataxia)
Have patient walk heel-to-toe, walk on toes then heels (Truncal and gait ataxia)

Temporal orientation (5 points)	What is the approximate time?
	What day of the week is it?
	What is the date today?
	What is the month?
	What is the year?
Spatial orientation (5 points)	Where are we now?
	What is this place?
	In what district are we or what is the address here?
	In which town are we?
	In which state are we?
Registration (3 points)	Repeat the following words: CAR, VASE, BRICK
Attention and calculation (5 points)	Subtract: 100-7 = 93-7 = 86-7 = 79-7 = 72-7 = 65
Remote memory (3 points)	Can you remember the 3 words you have just said?
Naming 2 objects (2 points)	Watch and pen
REPEAT (1 point)	"NO IFS, ANDS OR BUTS"
Stage command (3 points)	"Take this piece of paper with your right hand, fold it in half, and put it on the floor"
Writing a complete sentence (1 point)	Write a sentence that makes sense
Reading and obey (1 point)	Close your eyes
Copy the diagram (1 point)	Copy two pentagons with an intersection 

Presentation of findings

Vital signs: Report as documented, comment on any abnormalities
General: Well-developed well-nourished white female (male,?) in no acute distress
Head: Normocephalic atraumatic
Eyes: Conjunctiva and sclera are clear bilaterally; pupils are equal round and reactive to light and accommodation
Ears: External ears are intact without lesions, external auditory canals are patent bilaterally, tympanic membranes are intact bilaterally with an intact light reflex in normal mobility
Nose: Nares are patent bilaterally, septum is midline
Throat: Oropharynx is clear of any lesions or exudate; the uvula elevates midline, dentition and oral cavity is without lesion
Neck: Supple, full range of motion, no mass or tenderness to palpation, no lymphadenopathy, no thyromegaly
Chest – Normal respiratory effort and chest wall excursion; clear to auscultation bilaterally with good airflow; heart – regular rate and rhythm without murmur rub or gallop
Abdomen – Normal active bowel sounds, soft, nontender without masses or hepatosplenomegaly
Extremities – Warm well perfused upper and lower extremities

Neuro - The patient is oriented to person, time, and place. Cranial nerves II to XII are intact. Motor exam reveals normal strength bilaterally. Reflexes are equal bilaterally and are within normal limits. The sensory examination results are normal, with pain, light touch, and stereognosis intact. Cerebellar function is normal, revealing normal gait, normal heel-to-toe movement (point-to-point) and no indication of ataxia.

Total points:_____/100 - time deduction: = _____ Passing must have 90/100 = 90%

OSCE #2

Instructions

In the following slides you will be given pertinent results of various diagnostic evaluations (X-rays etc.).

After you have evaluated the data in the following slides, you will then use this platform to record the elements of your history and physical exam soap note. Slides for the soap note elements will follow.

After you have finished, you may leave.

Slide #1 In this slide radiologic images are provided. What type of image is presented and what is your

interpretation of the image?

Slide #2 Use this space to record your chief complaint and history of present illness.

Slide #3 Use this space to record your patient's past medical history, family medical history and social history.

Slide #4 Use this space to record your patient's review of systems.

Slide #5 Use this space to record your patient's physical examination.

Slide #6 Use this space to record a differential diagnosis for your patient's clinical presentation.

Slide #7 Use this space to record an assessment (most likely diagnosis) and plan including additional investigations as well as therapeutic measures (medications, disposition etc.).

Slide #8 Recheck! Testing: STAT CT head non contrast, CBC, BMP, ESR, PT/INR, urine preg

Tx: Prochlorperazine, Diphenhydramine, 1 Liter NS IV for headache.

Recheck 39 minutes

The patient reports that her headache is a"7" on a 1 to 10 scale. Laboratory studies were normal including an erythrocyte sedimentation rate of 12. The CT scan was read as negative for any acute abnormality. You discussed with the patient the normal CT scan and laboratory values.

Use this space to record any additional investigations as well as therapeutic measures (medications, disposition etc.).

Slide #9 Recheck The patient's headache persists. An image of the patient's CSF is in the associated image. Use this space to record any additional investigations as well as therapeutic measures

(medications, disposition etc.).

Appendix 14

Rubric for Web-based Case Study (Posterior circulation stroke) Participant:

1. Physical examination elements were chosen in a hypothesis seeking manner (The choice of body systems and physical exam maneuvers was appropriate for the patient's presentation.)

Vital signs (0.25 points)

Cardiopulmonary (0.25 points)

Neurology detailed (cranial nerve deficits documented in physical exam) (0.25 points)

Ophthalmology detailed (0.25 points) Section points

2. Differential diagnosis and/or final diagnosis supported by clinical reasoning

Brainstem stroke or similar (lateral medullary syndrome, vertebral artery dissection, posterior circulation stroke (1 point)

Stroke, NOS (1 point)

Cerebellar lesion (1 point)

Minus: BPPV, labyrinthitis (-1 point) Section points

3. Evaluation and management plan was formulated in a manner appropriate for the patient's presentation.

Ordered Interpreted [Read as normal] CT head without contrast (0.25 points)

Ordered Fingerstick glucose and ECG (0.25 points)

Ordered MRI of brain (0.25 points)

Ordered CT angiogram or another vessel test after initial workup (0.25 points)

Minus: Initially ordered CT head with contrast, MRI, Plain film cranial Xray (-0.25 points)

Section points

4. Differential diagnosis and/or final diagnosis supported by clinical reasoning

Brainstem stroke or similar (lateral medullary syndrome, vertebral artery dissection, posterior circulation stroke (2 points)

Section points

Total points _____ (5 points possible)

Rubric for Web-based Case Study (HSP)

1. Selection and interpretation of exam findings

   ENT exam selected (normal findings given) (0.25 points)

   Skin palpable (0.25 points) purpura (0.25)

   Abdominal exam selected [Normal findings given] (0.25 points) Section points

2. Differential diagnosis supported by clinical reasoning

   (1 point) Hematuria with palpable purpura, Nephritis, Nephritic syndrome, UTI, Kidney stone, platelet disorder, infectious disease

3. Evaluation plan was formulated in a manner appropriate for the patient's presentation

   Ordered CMP or BMP, CBC, UA, Complement, Antibodies, PT/PTT/INR (0.25 points)

   Interpreted urine sediment (0.25 points)

   Noted hematuria (0.25 points)

   Noted elevated creatinine (0.25 points)

   Minus: ordered Abd X-ray or CT Abd (-0.25 points) Section points_____

4. Differential diagnosis and/or final diagnosis supported by clinical reasoning

   (2 points) Henoch-Schonlein purpura, IgA nephropathy, Immune mediated nephropathy

   (1 point) Hematuria with palpable purpura, Nephritis, Nephritic syndrome,

Section points_____ Total points _____ (5 points possible)

Rubric for illness script repleteness :

Cardiogenic thromboembolic stroke

Enabling conditions (patient features and context)

>65 Female or Male (0.25)

HTN (0.25)

CHF (0.25)

CVA/TIA (0.25) or CAD or PAD (0.25)

Atrial fibrillation (1) Total: out of 2

Consequences (signs and symptoms)

Anterior

Hemiparesis and dysthesias (patterned) (1)

Or

AMS with aphasia (1)

Fault (pathophysiological mechanism)

EKG (Afib)

+

TEE or TTE – thrombus in atrial appendage (2)

OR

EKG (Afib)

+

Vessel study (MRA/CT angio) (2)

Total:

Rubric for illness script repleteness:

Acute adrenal insufficiency

Enabling conditions (patient features and content)

>Age, Female or Male

Hx of immune disorder **(1)**

Diabetes, Type I

Vitiligo

OR

Chronic corticosteroid use **(1)**

Consequences (signs and symptoms)

Any acute, non-specific syndrome: Abdominal pain, N/V/D, Weakness, AMS **(1)**

Hypotension **(0.50)**

Hyperpigmentation **(0.50)**

Fault (pathophysiological mechanism)

ACTH test **(1)**

Hyponatremia **(0.50)**

Hyperkalemia **(0.50)**

Total:

Appendix 15 **Experimental Curriculum Timetable**

Curricular element	**Date and time on task**	**Structured Knowledge Approach** (Frameworks, Schemas and heuristic streamlining)	**Case-based clinical reasoning** (CR through cases, integrated use of physical exam and diagnostics)
Clinical reasoning lecture I	January 10, 2023 2 hours	Schemas Heuristic for elements of clinical process	Vignette as clinical encounter
Clinical reasoning lecture II	January 13, 2023 2 hours	Frameworks and illness scripts	Vignette as clinical encounter
Neuroanatomy overview lecture	June 15, 2023 1.5 hours	CNS, PNS and Pathway framework High-yield and clinically oriented content selection	Clinical vignette
Movement and sensation lecture	June 16, 2023 1.5 hours	Pathways deficit summary Neural pathways simplified for lesion localization	Lesion graphics as vignettes
Brainstem and cranial nerve lecture	June 20, 2023 1.5 hours	Scaffolded three-tier approach	Clinical correlations
Neurovascular and meninges lecture	June 21, 2023 1.5 hours	Anatomic schematization of neurovascular insults Graphic to anatomic spatial arrangement; pictorial representation	Correlation of location and nature of deficit
Movement as volition and coordination lecture	June 22, 2023 1.5 hours	Schema for movement disturbances Simplified model of extrapyramidal system	Clinical correlations, video vignettes
Cortical functions and cerebrovascular accident	June 23, 2023 2 hours	Schema for pathophysiology Schema for anterior versus posterior circulation strokes Location and phenomenology repetition (“Where is it, what is it?”)	Clinical correlations (examples of prevalent stroke syndromes)
Neuroradiology and diagnostics lecture	June 27, 2023 1.5 hours		Integrated use of physical exam and diagnostics
Neurology history and physical exam lecture and lab	June 29, 2023 2 hours	Schemata for pathophysiologic mechanisms Repetition of the location and phenomenology approach	Clinical correlations with examples
Neurology case studies	June 29, 2023 2 hours		Case-based clinical reasoning
Neurology OSCE	June 30, 2023 2 hours		Case-based clinical reasoning

Appendix 16

A Comparison of Covered Versus Uncovered Conditions

Condition or problem	Body system	Number of hours covered in body system	Time from coverage until assessment	Additional coverage in curriculum	Cases used in content delivery
Dizziness and dysarthria (brainstem stroke) [Covered]	Neurology	31 hours	Four months three weeks	ENT (Central versus peripheral vertigo)	Yes
Cardioembolic cerebrovascular accident [Covered]	Neurology	31 hours	Four months three weeks	Cardiology (atrial fibrillation)	Yes
Palpable purpura and hematuria (Henoch-Schönlein purpura) [Uncovered]	Nephrology	30 hours	Two months three weeks	Pediatric hematology	No
Acute adrenal insufficiency [Uncovered]	Endocrine	20 hours	Three months three weeks	Emergency medicine (adrenal crisis and distributive shock)	No

Data collection using cases studies and illness script exercises – November 20,2023

Neurology 31 hours (Completed June 27) Time lapse before data collection: 4 months and 3 weeks

Endocrinology 20 hours (Completed August 2) Time lapse before data collection: 3 months and 3 weeks

Nephrology 30 hours (Completed Sept 1) Time lapse before data collection: 2 months and three weeks

Additional treatments of conditions and problems

Endocrine – Adrenal insufficiency (Adrenal insufficiency covered again Nov 15 shock in EM Module)

Nephrology – Iga nephropathy and purpura (August 27) (ITP covered in peds Oct 30)

Appendix 17

Illness Script Diagrams

Medical education seeks strategies that will improve clinical reasoning capacity prior to the clinical stage of education.

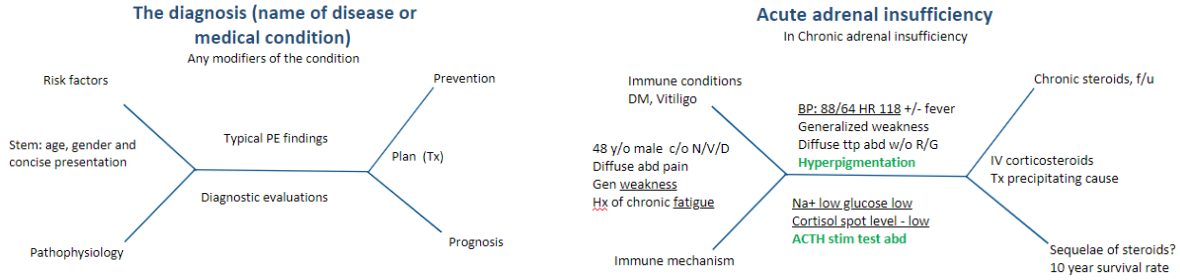
One proposed pedagogical technique is the use of illness script diagrams. Using a simple horizontal bracket diagram (like those used to diagram the findings of the CBC) student clinicians can learn to record typical illness scripts in a timely manner.

Overview of terminology: Frameworks, schemas and illness scripts

Frameworks assist in structuring a learner's knowledge of diseases and conditions. Frameworks have different anchoring principles depending on the class or set of diseases/conditions being studied. The principles may begin with organ system (cardiology – ACS, arrhythmias etc) or type of presentation (Chest pain, palpitations). The framework may also be based on distinctions within a particular disease state or condition (vision loss [monocular, binocular], acute kidney injury [pre, renal, post]. Frameworks provide a kind of logic model for understanding related and distinct diseases. By parsing out the distinctions between disease states and depicting those states in a diagram or concept map, the conceptual relationships defining the distinctions and overlap between disease states are made readily evident. Contrast this display of the relationships among diseases and conditions (say the cardiomyopathies in concept map form) versus the description of the relationships in prose form. In prose form, the relationships between disease states are tacit and it is this reader's task to more efficiently organize the relationships among the various diseases. Some may say that this discursive work by the reader aids in the mastery of content but at what cost? The sheer volume and complexity of medical knowledge requires a more structured and efficient approach. The discursive burden, and the educational benefit of that work, will not be lost but rather shifted to a more productive level of cognition. Frameworks then, are what is used in the teaching of relationships between diseases and conditions. Frameworks are forms of structured knowledge that, hopefully, promote the organization of the knowledge in the mind of the learner.

When encountering a patient with a possible disease state, knowledge of the possible disease state is understood not as a series of isolated facts that are analytically recombined during the clinical encounter. The learner is already primed with a pattern that they deploy in to make sense of the various data of the encounter. Within the mind of the learner, disease states and conditions begin to organize into patterns that will be readily recognizable when the learner sees patients in the clinic. These patterned ways of seeing are called schemas and have enormous importance for understanding the development of clinic reasoning. Schemas are cognitive structures in the minds of student clinicians that efficiently interpret the data of clinical encounters. Previously it was thought that clinical reasoning developed only in the clinic and that the role of didactic or preclinical education was to arm the learner with sufficient facts and declarative knowledge to fund clinical learning. My suggestion here is that we begin to use illness script diagrams (ISDs) to facilitate knowledge organization in the preclinical phases of learning. An illness script diagram uses a very basic diagram structure to organize the various components of a disease or condition in a way that can be captured in a single visual frame. The components of the diagram are shown in the images below: the left closing bracket provides a middle space for recording the stem (demographic data and concise presentation), the upper spoke of the left

open bracket provides a space for risk factors in context, the middle bar of the diagram divides upper from lower. In the upper space, typical physical exam findings are recorded. In the lower space of the middle section key findings of diagnostic studies are recorded. The bracket to the far right, the opening bracket, provides a middle space to record concise treatment information. The upper spoke of the opening bracket provides a space to record preventative measures and the lower spoke of the opening bracket provides a space to record a brief prognosis.



The left to right nature and closing bracket / opening bracket parallels the logic of illness scripts. In the left bracket, enabling conditions (risk factors, context) and fault (pathophysiology) along with the epidemiologic clues provided by the stem symbolically suggests these causal features are “compressing” into the middle line that will set the stage for consequences (signs and symptoms). The movement from left to right and top to bottom (in the middle line) provides sufficient data for a diagnosis to be placed at the top of the diagram in the middle section. The opening bracket at the right of the diagram suggests possible sequelae of the illness script beginning with appropriate treatment in the middle of the opening bracket. The upper spoke of the open bracket provides a space to annotate secondary or tertiary prevention measures; the bottom spoke of the open bracket provides a space for a concise prognosis.

A simple color code is also used in the diagrams. If a particular feature of an illness script is pathognomonic or essential for the diagnosis, that feature is listed in a green bold font. Components of the illness script that are not pathognomonic of a condition but are highly suggestive or frequently associated with the disease or condition are listed in underlined font. The stem provides a classic presentation of a particular illness or condition. The age, gender and classic presenting signs and symptoms are concisely recorded in the middle of the opening bracket. If a particular age range is essential to a condition (ACS, temporal arteritis) the age may be listed with underlined font to suggest the epidemiological importance of the age. The same can be said of stem elements such as sex, ethnicity, race. An example would be a 23 y/o Ashkenazi Jew with Tay Sachs disease. In the RF/context area, other elements may be highlighted in green – occupation (ship yard worker = asbestos exposure). In the case of conditions that are usually diagnosed clinically (e.g. dermatologic conditions), no diagnostic testing will be listed in the bottom middle panel. Red font is used to denote elements of the illness script that may present confusion in the diagnostic process or common pitfalls in diagnosing a particular condition. An example would be ST segment elevations in a patient with an aortic dissection that causes the clinician to mistake the presentation of chest pain as a STEMI.

The utility of the ISD relies upon their initial simplicity. The simple form allows anyone to draw the diagrams without software or any special equipment. A chalk board or dry erase board will do. There may be a temptation to overly elaborate the diagrams with images or video clips. In later stages of learning this may be helpful but initially the diagrams should be simple, concise and rely heavily on text.

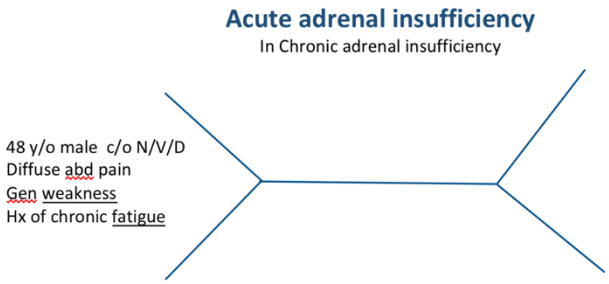
It is important for novice stage learners to know the medical terminology and linguistic modifiers that are commonly used in medical literature and daily practice.

The use of illness script diagrams and formative learning experiences

As part of the “structured knowledge approach" to clinical reasoning, we believe that illness scripts can be used effectively in conjunction with frameworks. As described above, frameworks provide an organized way of thinking about a particular category of diagnoses or medical conditions. An illness script diagram is an explicit depiction of a particular type of schema (illness script). Illness script diagrams can be used to present the information in a concise manner for introductory clinical medicine lectures. When first discussing a particular condition (COPD for example) the illness script diagram can be used to provide a concise and cohesive depiction of that particular disease state. The form of the diagram will help novice stage learners organize information in a way that is consistent with the logical flow of the clinical process. That is, the simple nature of the diagram will be helpful in teaching novice stage learners the various components of an illness script in a way that manages cognitive load. In a lecture format, the diagrams will need to be supplemented with high-yield images and additional content but the diagrams act as a kind of central illustration that guides and organizes the learning process. As a central illustration, the illness script diagrams can also be used for clinical education “chalk talks” in which each of the components of the diagram can be highly elaborated and discussed in an interactive format. For example, the diagram for “acute adrenal insufficiency" may simply say “immune mechanism” under pathophysiology section. Although the initial form of the diagram is initially truncated for simplicity sake, the presenter could use the pathophysiology gloss as an opportunity to present or quiz the learners and further elaborate the pathophysiology of a particular disease state. As the discussion proceeds and components of the illness script become elaborated through discussion, students can take notes and make additions to subsequent versions of the diagram. That is, the diagrams can be expanded by the student in ways that parallel the development of their knowledge structures.

The illness script diagram may be used in several ways for formative learning experiences. In an illness script challenge, an ISD may be listed completely blank and the learner given the name of the disease or condition. The learner is required to fill out the entirety of the diagram. Alternatively, the diagram could be listed with all information but the diagnosis (diagnosis challenge) and the learner is required to provide the diagnosis. An important variation of the diagnostic challenge is the DDX challenge or differential diagnosis challenge in which a partial ISD given (Stem, RF and PE findings) and the learner is tasked with developing a differential diagnosis for the ISD. The learner would then be queried about the appropriate plan to rule-in or out various diagnoses. Any of the other components could be omitted and the learner is required to provide the missing data.

Another application of the illness script diagram for more advanced stage learners is diagnostic reasoning. When students are given a case scenario and tasked with providing the diagnosis, students may find it useful to diagram the data in their case study. The diagram will help to organize information in the appropriate categories. As students consider a differential diagnosis for the case scenario,



students can compare the data in their diagram with the data of most likely conditions. Any discrepancy would prompt reconsideration of another diagnosis. Comparing the ISDs of two conditions that present in very similar ways may help to draw out the distinctions between the two conditions in their appropriate diagnosis. In glomerulonephritis, for example, the palpable purpura manifesting distinguishes Henoch-Schönlein purpura from IGA nephropathy. Whether for clinical readiness or high-stakes exams, the single visual frame and organized nature of the illness script diagram may be helpful to more advanced stage learners.

Illness Script challenge

The learner is provided an ISD with only the name of the condition. The learner would then provide the stem, risk factors, middle section (upper – physical exam findings, lower diagnostic evaluations) and treatment. The learner would then go back and fill in the pathophysiology, prevention and prognosis. When the learner is able to recall all elements of the ISD, the learner would answer some probing questions about the illness script.

1. Describe the relationship between the risk factors and the pathophysiology of the condition?
2. How do the clinical findings (exam and diagnostics) relate to the pathophysiology?
3. Are there any diagnostic pitfalls for this condition?
    a. The script presented in this diagram depicted a classic presentation. Describe two possible atypical presentations for this illness.
    b. Are there any Dopplegangers or other conditions that bear semblance to this condition? Briefly diagram both conditions and highlight the diagram elements that would allow you to distinguish one from the other.
    c. Red-herrings?
4. Disease specific conditions: In idiopathic dilated cardiomyopathy, the title also specifies “with compensated heart failure.” How would the script change if the diagnosis line read “with acute decompensated heart failure”?

5. List the other pathophysiologic mechanism for dilated cardiomyopathy.
6. Describe the other structural types of CM.