CURRICULUM

Novel Emergency Medicine Curriculum Utilizing Self-Directed Learning and the Flipped Classroom Method: Neurologic Emergencies Small Group Module

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ABSTRACT:

Audience: This is a group discussion, case-based curriculum to cover emergency medicine neurology core content. It was created and implemented at The Ohio State University Emergency Medicine Residency program and was designed to educate our emergency medicine (EM) residents, PGY-1 to PGY-3, as well as medical students and attending physicians.

Length of Curriculum: The neurologic emergencies module consists of eight 45-60-minute small group sessions for a total of six to eight hours of content. This curriculum block is part of an overall 18-month emergency medicine residency curriculum.

Introduction: In 2015, approximately 7 million emergency department visits were related to diseases of the nervous system. Headache alone represented 2.8% of all emergency department visits, making it the fifth most common chief complaint.[1] Residents must be proficient in the differential diagnosis and management of the wide variety of neurological emergencies. To address this specific curricular need, we developed a flipped classroom case-based small group discussion series for emergency medicine learners that emphasizes self-directed learning activities, followed by facilitated small group discussions pertaining to the topic reviewed. The active learning fostered by this curriculum increases faculty and learner engagement and interaction time typically absent in traditional lecture-based formats.[2-4] The application of knowledge through case studies, personal interaction with content experts, and integrated questions are effective learning strategies for emergency medicine residents.[4-6]

Educational Goals: We aim to teach the presentation and management of neurologic emergencies through the creation of a flipped classroom design. This unique, innovative curriculum utilizes resources chosen by

education faculty and resident learners, study questions, real-life experiences, and small group discussions in place of traditional lectures. In doing so, a goal of the curriculum is to encourage self-directed learning, improve understanding and knowledge retention, and improve the educational experience of our residents.

Educational Methods: The educational strategies used in this curriculum include: small group modules authored by education faculty and content experts based on the core emergency medicine content as outlined in the ABEM model curriculum.[7] This program also includes resident-submitted questions that were developed during review of the content. The use of free open access medical education (FOAM) resources allows learners to work at their own pace and maximize autonomy.

The modules and associated learning resources are posted on a digital learning management system for faculty and residents to review at least one week before the session. On the day of the sessions, learners divide into small groups of 10-15 participants with a mix of junior and senior residents. Each group is led by both a faculty leader and an optional designated senior resident who has spent extra time preparing to lead the discussion.

Research Methods: Learners evaluated the content using end-of-session feedback forms. Learners were also surveyed after the curriculum implementation and asked to evaluate the effectiveness of the small group curriculum. Faculty small-group facilitators assessed learner participation.

Results: Overall the neurology small group curriculum was well received by residents and faculty educators. A majority of residents (63%) reported that the small group discussions were good or excellent, compared to only 24% of residents that felt that grand rounds lectures during the same time were good or excellent. Residents evaluated the neurology block teaching methods as effective, with an average rating of each small group session receiving more than 4.3 out of 5 (4 being agree, 5 being strongly agree).

Discussion: Learners and educators were enthusiastic about the conference structure and expressed a preference for it rather than the previous, lecture-based didactics. This implementation showed that case-based, flipped classroom small group modules are a preferred format for teaching core content in emergency neurology. Significant faculty time was required to create and update the small modules, which may limit widespread adoption of this format for resident education. Future developments could help guide learners with different baseline knowledge.

Topics: Emergency medicine, flipped classroom, medical education, neurologic emergencies, pedagogy, teaching.

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List of Resources:

Learner Audience:
Medical students, interns, junior residents, senior residents, attending physicians and faculty members

Length of Curriculum:
The neurologic emergencies module consists of eight 45-60-minute small group sessions for a total of six to eight hours of content. Two small group sessions have replaced two hours of traditional lecture each week during our resident education conference. Therefore, this curriculum block comprises 4 weeks of an overall 18-month emergency medicine residency curriculum.

Topics:
Emergency medicine, flipped classroom, medical education, neurologic emergencies, pedagogy, teaching.

Objectives:
After completing the neurologic emergencies module, resident learners will exhibit mastery within this content area and will critically discuss the pathophysiology, diagnosis, and treatment of various pediatric and adult neurologic emergencies including:

1. Acute Headache
    a. Discuss the differential diagnosis for patients presenting to the ED with acute headache.
    b. Review the causes of primary and secondary headaches.
    c. Discuss the diagnostic workup and indications for imaging in patients with acute headache.
    d. Discuss the approach to management of three types of acute headache in the ED.
2. Subarachnoid Hemorrhage
    a. Describe the epidemiology and clinical presentation of subarachnoid hemorrhage (SAH).
    b. Discuss the diagnostic workup of SAH.
    c. Discuss the approach to management of the patient with SAH.
3. Intracerebral Hemorrhage
    a. Describe epidemiology, pathophysiology and risk factors for development of intracerebral hemorrhage (ICH).
    b. Describe clinical presentation of ICH. Discuss how the clinical presentation differs from ischemic stroke.
    c. Discuss the diagnostic workup for ICH.
    d. Discuss the approach to initial management of the patient with ICH.
4. Seizure
    a. Understand the different types of possible seizures including status epilepticus.
    b. Discuss management of a patient presenting with a first-time seizure vs a patient with a previous history of seizures.
    c. Discuss management of status epilepticus.
    d. Discuss the differential for non-epileptic causes of seizures.
    e. Differentiate between the definition and management of simple febrile seizures and complex febrile seizures.
5. Acute Ischemic Stroke and Transient Ischemic Attack
    a. Describe epidemiology, pathophysiology, and risk factors for development of acute ischemic stroke (AIS).
    b. Describe clinical presentation of AIS and transient ischemic attack (TIA). Discuss the other clinical entities which form the differential diagnosis.
    c. Discuss the diagnostic workup for AIS including the NIH Stroke Scale.
    d. Discuss the approach to initial management of the patient with AIS in the ED.
    e. Discuss options for the management of TIA.
6. Vertigo
    a. Discuss features of the patient history that can help distinguish between true vertigo

and other non-specific descriptions of dizziness.
   b. Demonstrate physical exam techniques to distinguish between central and peripheral forms of vertigo.
   c. Define acute vestibular syndrome, spontaneous episodic vestibular syndrome, and triggered episodic vestibular syndrome.
   d. Describe an algorithmic approach to isolated vertigo.
   e. Discuss the utility of advanced imaging in the work-up of vertigo.
   f. Identify the common causes of peripheral vertigo and their associated treatment.
7. Infectious and Inflammatory Neurologic Disorders
   a. Discuss the clinical historical features and exam findings suggestive of meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess.
   b. Discuss the emergency department workup for viral and bacterial meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess.
   c. Identify the appropriate initial treatments for viral and bacterial meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess.
8. Demyelinating, Neuromuscular, and Movement Disorders
   a. Discuss the underlying pathophysiology, distinguishing clinical features, diagnostic workup, treatment and expected complications of common demyelinating disorders: multiple sclerosis (MS), and Guillain-Barré Syndrome (GBS).
   b. Distinguish clinical presentation and management of myasthenia gravis (MG) from demyelinating disorders.

Brief introduction:

In 2015, approximately 7 million emergency department visits were related to diseases of the nervous system. Headache alone represented 2.8% of all emergency department visits, making it the fifth most common chief complaint.[1] Neurologic conditions such as ischemic stroke and intracerebral hemorrhage are major causes of severe long-term disability and death.[8] In these, as well as other emergent neurologic conditions, the amount of injury to the central nervous system is critically dependent on the duration of the underlying pathology. Rapid recognition and treatment are vitally important in limiting morbidity and mortality associated with these conditions.[9] Therefore, emergency medicine residents must be proficient in the differential diagnosis and management of a wide variety of neurological emergencies. To address this specific curricular need, we developed a flipped classroom, case-based small group discussion series for emergency medicine learners.

The flipped classroom learning approach is becoming more commonly recognized as a preferred curricular model for mature learners, specifically those in medical education. This particular model is a natural fit for the hands-on, experiential emergency medicine learner.[4] The active learning fostered by this curriculum increases faculty and learner engagement and interaction time, which is typically absent in traditional lecture-based formats.[5,10] Education literature shows that resident learners prefer learning activities that involve small group discussion, are case- or skill-based, and emphasize the application of newly obtained knowledge.[3,4] This educational model also provides a clear channel for the incorporation of evidence-based medicine and increases opportunities for educator-learner conversations. A successful flipped classroom curriculum fosters learner accountability and provides robust opportunities for formal assessment in various emergency medicine milestones.[4, 10, 11] Accordingly, we have transitioned our traditional lecture-based didactics to a flipped classroom curriculum at The Ohio State University Emergency Medicine Residency. This neurologic emergencies curriculum is one of several topics in our overall didactic curriculum.[12-20]

Problem identification, general and targeted needs assessment:

Neurologic emergencies are identified as an important aspect of an emergency medicine curriculum as outlined in the ABEM model EM curriculum.[7] This topic makes up a significant portion of the content that will appear on the emergency medicine in-training exam. Consequently, we decided that neurologic emergencies warranted a dedicated content block within our curriculum.

Prior to the implementation of this curriculum, education faculty leadership identified the topic areas to cover based on the neurologic emergencies identified in the ABEM model EM curriculum.[7]. The topics covered represent the disease processes considered most critical in the EM model of practice that are not covered elsewhere in our didactic series. These topics were then assigned to core faculty educators who developed the small group modules. Education faculty leadership edited the modules for content and consistency. Dissemination of the modules and associated learning resources was accomplished by posting on a digital learning management system for faculty and resident review prior to the sessions.

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Goals of the curriculum:
We aim to teach the presentation and management of neurologic emergencies through the creation of a flipped classroom design. This unique, innovative curriculum utilizes resources chosen by education faculty and resident learners, study questions, real-life experiences, and small group discussions in place of traditional lectures. In doing so, a goal of the curriculum is to encourage self-directed learning, improve understanding and knowledge retention, and improve the educational experience of our residents.

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    a. Discuss the underlying pathophysiology, distinguishing clinical features, diagnostic workup, treatment and expected complications of common demyelinating

disorders: multiple sclerosis (MS), and Guillain-Barré Syndrome (GBS).
b. Distinguish clinical presentation and management of myasthenia gravis (MG) from demyelinating disorders.
c. Discuss the underlying pathophysiology, distinguishing clinical features, and diagnostic workup of Parkinson's Disease.
d. Discuss diagnosis and management of extrapyramidal symptoms and akathisia.

Educational Strategies: (See curriculum chart)
Please refer to the curriculum chart of linked objectives and educational strategies.

Evaluation and Feedback:
This innovative curriculum was literature-based and specifically designed to maximize active learning using the flipped classroom learning model. We overcame initial challenges and skepticism from both educators and learners to execute a successful, novel curricular model. Both resident learners and faculty educators have provided an overwhelming amount of positive feedback. Additionally, residents were surveyed on their perceived quality of instruction of the various program components each year. In the year prior to implementation of this curricular innovation, only 52% of residents completing the survey (n=28) rated the quality of small group discussion as good or excellent, whereas 68% of respondents (n=19) rated the quality as good or excellent after the conclusion of the first year. Learners and educators were enthusiastic about the conference structure and expressed a preference for it rather than the previous, lecture-based didactics. More recently during the second 18-month cycle of the flipped classroom curriculum, 63% of residents responding to the survey (n=38) reported that the small group discussions were good or excellent, compared to only 24% of residents that felt that our grand rounds sessions during the same time were good or excellent. This curriculum has been delivered to two cohorts of learners, having delivered the content twice in three years with about 50 residents per cycle. On the most recent iteration, residents evaluated the teaching methods as effective, with an average rating of more than 4.3 out of 5 (4 being agree, 5 being strongly agree). The curriculum is critically evaluated and updated by education faculty members in order to ensure educational material remains current and consistent with the emergency medicine core content. Future research will be needed to investigate the potential effects of this curriculum on in-training exam (ITE) scores and clinical performance.

Significant faculty time was required to develop and update these small group modules. This time commitment may limit the wide-spread adoption of the flipped classroom model for residency education. But we hope that this content will help jump start a similar implementation at other emergency medicine residency programs. Future work could focus readings and materials on different levels of learners, so that it is more specifically tailored to both the junior and senior resident.

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Additional Resources:

Educational resources are available within each individual chapter of this neurologic emergencies curricular module; however, a complete list of resources and educational materials are listed below.

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Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Headache	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of primary headache syndromes.	1. Discuss the differential diagnosis for patients presenting to the ED with acute headache. 2. Review the causes of primary and secondary headaches. 3. Discuss the diagnostic workup and indications for imaging in patients with acute headache. 4. Discuss the approach to management of three different types of acute headache in the ED.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Subarachnoid Hemorrhage (SAH)	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of subarachnoid hemorrhage.	1. Describe the epidemiology and clinical presentation of subarachnoid hemorrhage (SAH). 2. Discuss the diagnostic workup of SAH. 3. Discuss the approach to management of the patient with SAH.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Intracerebral Hemorrhage (ICH)	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of intracerebral hemorrhage.	1. Describe epidemiology, pathophysiology and risk factors for development of ICH. 2. Describe clinical presentation of ICH. Discuss how the clinical presentation differs from ischemic stroke. 3. Discuss the diagnostic workup for ICH. 4. Discuss the approach to initial management of the patient with ICH.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Seizures	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of seizures.	1. Understand the different types of possible seizures including status epilepticus. 2. Discuss management of a patient presenting with a first-time seizure vs a patient with a previous history of seizures. 3. Discuss management of status epilepticus. 4. Discuss the differential for non-epileptic causes of seizures. 5. Differentiate between the definition and management of simple febrile seizures and complex febrile seizures.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Acute Ischemic Stroke (AIS) and Transient Ischemic Attack (TIA)	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of seizures.	1. Describe epidemiology, pathophysiology, and risk factors for development of AIS. 2. Describe clinical presentation of AIS and TIA. Discuss the other clinical entities which form the differential diagnosis. 3. Discuss the diagnostic workup for AIS including the NIH Stroke Scale. 4. Discuss the approach to initial management of the patient with AIS in the ED. 5. Discuss options for the management of TIA	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Vertigo	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of vertigo.	1. Discuss features of the patient history that can help distinguish between true vertigo and other non-specific descriptions of dizziness. 2. Demonstrate physical exam techniques to distinguish between central and peripheral forms of vertigo. 3. Define acute vestibular syndrome, spontaneous episodic vestibular syndrome, and triggered episodic vestibular syndrome. 4. Describe an algorithmic approach to isolated vertigo. 5. Discuss the utility of advanced imaging in the work-up of vertigo. 6. Identify the common causes of peripheral vertigo and their associated treatment.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

DIDACTICS AND HANDS-ON CURRICULUM

Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Neurologic Infectious and Inflammatory Conditions	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of neurologic infections such as meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess.	1. Discuss the clinical historical features and exam findings suggestive of meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess. 2. Discuss the emergency department workup for viral and bacterial meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess. 3. Identify the appropriate initial treatments for viral and bacterial meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

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Topic	Recommended Educational Strategy	Educational Content	Objectives	Learners	Timing, resources Needed (Space, Instructors, Equipment, citations of JETem pubs or other literature)	Recommended Assessment, Milestones Addressed
Demyelinating Disorders, Neuromuscular Disorders and Movement Disorders	"Flipped" classroom discussion of pre-reading material, case discussions, and discussion questions. Encourage participants to share clinical experiences to enhance discussion. 45 minutes for case and content discussion.	Pathophysiology, diagnosis, and management of multiple sclerosis, Guillain-Barre syndrome, and Parkinson's disease. Discussion of management of extrapyramidal symptoms.	1. Discuss the underlying pathophysiology, distinguishing clinical features, diagnostic workup, treatment and expected complications of common demyelinating disorders: multiple sclerosis (MS), and Guillain-Barré Syndrome (GBS). 2. Distinguish clinical presentation and management of myasthenia gravis (MG). from demyelinating disorders. 3. Discuss the underlying pathophysiology, distinguishing clinical features, and diagnostic workup of Parkinson's Disease. 4. Discuss diagnosis and management of extrapyramidal symptoms and akathisia.	PGY-1 PGY-2 PGY-3 Medical Students Faculty	Equipment: projector and screen preferable (instructor can pull up web images during session). Tables and space promoting small group discussion. Instructors: 1-2 faculty members or content experts. Predetermined senior resident discussion leader (optional). Timing: small group discussions involve no more than 15 learners and last about 45 minutes.	Milestone: Emergency stabilization (PC1), diagnostic studies (PC3), differential diagnosis (PC4), pharmacology (PC5), medical knowledge (MK). Assessment: Faculty evaluation of resident participation during small group activities. Evaluation: Resident evaluation of small group session content and facilitators. Yearly program evaluation of overall small group component.

Appendix A:
Acute Headaches

Objectives

1. Discuss the differential diagnosis for patients presenting to the ED with acute headache.
2. Review the causes of primary and secondary headaches.
3. Discuss the diagnostic workup and indications for imaging in patients with acute headache.
4. Discuss the approach to management of three common types of acute headache in the ED.

Case Studies

Case 1: A 28-year-old female with history of morbid obesity presents with daily headaches that started two months ago and have become constant for the past week. She has never had this type of headache before. The pain is diffuse and bilateral and associated with mild nausea and occasional blurred vision. She notices improvement with ibuprofen but the headache never goes away completely. She feels that the headache worsens when lying down.

Vital signs: Temperature (T) 99.0°F, heart rate (HR) 92, blood pressure (BP) 106/78, respiratory rate (RR) 18, oxygen saturation (O_2sat) 97% on room air

Physical exam: The patient appears comfortable. She has no nuchal rigidity or focal neurologic deficits on exam. Fundoscopic exam reveals mild papilledema bilaterally.

Question Prompts:

1. What is the differential diagnosis for acute headache?
    a. The differential diagnosis includes both primary and secondary types of headache.
        i. Primary headaches include migraine (with or without aura), tension headache, and the trigeminal autonomic cephalgias such as cluster headache and paroxysmal hemicrania.
        ii. Secondary headaches arise from numerous different etiologies, ranging from benign causes to potentially life-threatening causes. Common benign causes of headache include viral syndromes, herpes zoster, sinusitis, referred dental pain, substance use and substance withdrawal. The cause can also sometimes be iatrogenic, such as post-LP headaches, which are generally benign in nature.
        iii. Potentially serious causes of secondary headache include subarachnoid or other intracranial hemorrhage, cerebral sinus thrombosis, giant cell arteritis, carotid or vertebral artery dissection, central nervous system infections (meningitis, encephalitis, and cerebral abscess), acute angle closure glaucoma, iritis, optic

neuritis, mass lesions, idiopathic intracranial hypertension (IIH), carbon monoxide poisoning, hypertensive encephalopathy, preeclampsia, and pheochromocytoma.

   b. Based on the presented history, idiopathic intracranial hypertension (IIH) seems most likely but would need to include a broad differential. Given this patient's findings of papilledema, would consider secondary causes of headache including dural sinus thrombosis, tumor/mass, Chiari malformation to name a few.

2. What historical and physical exam features are important to obtain for patients presenting with headache?
   a. The headache history should include the number and type(s) of headaches present, age and circumstances of onset, family history of headache, characteristics of the pain (location, severity, onset, duration, and frequency), prodromal symptoms or aura, associated symptoms, exacerbating/alleviating factors (*eg,* position change), medications and prior workup.
   b. The physical exam should include a complete neurological exam; ears, eyes, nose and throat; scalp and skin exams looking for signs of trauma or infection; funduscopic looking for papilledema; and neck exam with attention to meningeal signs.
3. What are the indications for imaging in the diagnosis of acute headache?
   a. Neuroimaging is indicated for *all* patients with a headache and new focal neurologic deficit on physical exam.
   b. In addition, there are certain historical "red flags" suggestive of a potentially dangerous cause of secondary headache which may indicate a need for diagnostic workup. These include new onset headache after the age of 50, worsening frequency or severity of headache over short time course, headaches made worse by Valsalva or laying down, history of head trauma, history of cancer or immunosuppression, systemic symptoms such as fever or neck pain, alterations in level of consciousness, or focal neurologic symptoms.
4. What diagnosis does the patient's symptom complex suggest?
   a. The patient's symptoms are most consistent with elevated intracranial pressure. While this could be from more serious intracranial processes such as mass lesion, dural sinus thrombosis, or hemorrhage, idiopathic intracranial hypertension (IIH) is most likely with the patient's history of morbid obesity. Patients with IIH typically present with subacute or chronic headache in combination with nausea and vomiting, cognitive difficulty, and dizziness as well as a variety of visual symptoms including blurry vision, transient visual obscurations, visual field defects and 6th nerve palsy (a marker of elevated intracranial pressures since it has longest intracranial course).
5. Describe the diagnostic workup for this patient.
   a. Neuroimaging (non-contrast head computed tomography [CT] or magnetic resonance imaging [MRI]) should be performed to exclude mass lesion prior to lumbar puncture. Concerning red flags are progressive symptoms over time, worsening symptoms when lying flat, and neurologic changes (blurry vision).
   b. In the absence of secondary causes of increased intracranial pressure, the diagnosis of IIH is made by lumbar puncture (LP) with cerebral spinal fluid (CSF) opening pressure greater than 25cm H_2O when measured in the lateral decubitus position. Although there is no clear

evidence regarding the time course of diagnostic testing, consensus of experts suggests that LP should be performed urgently (within 24 hours) for most patients with headache and signs of increased intracranial pressure.[3] Consider emergent LP in patients with rapidly progressive visual symptoms.

c. Both the opening and closing pressures, as well as amount of CSF removed should be measured. Cerebral spinal fluid should be sent for routine analysis to exclude central nervous system (CNS) infection.
d. Magnetic resonance imaging with contrast or MR venogram (MRV) is recommended to exclude cerebral venous sinus thrombosis before the diagnosis of IIH can be made. Although there is no clear evidence regarding the time course of diagnostic testing, consensus of experts suggests that MRV, or CT venography (CTV) in patients unable to have MRI, should be performed urgently (within 24 hours) for most patients with headache and signs of increased intracranial pressure. [3] Consider emergent MRV or CTV in patients with rapidly progressive visual symptoms.

6. What are treatment options for this patient?
    a. In the acute setting, LP provides symptom relief in most patients; however, closing pressure should be reduced to no more than 50% of opening pressure to avoid low pressure headache and the theoretical risk of tonsillar herniation.
    b. Long-term symptom control may be attained through weight reduction, medications (acetazolamide, furosemide, topiramate), optic nerve fenestration, CSF diversion, or dural sinus stenting. Acetazolamide and topiramate both have significant side effects and require dose titration. Therefore, it is reasonable to defer starting these medications until the patient receives outpatient follow-up.

Case 2: An 18-year-old female presents with episodic left-sided, throbbing headaches that occur three to four times per month. The headaches started five years ago, but seem to have increased in severity. They are associated with nausea and she sees waves of light about 30 minutes before the pain begins. Light and noise make the headache worse. Ibuprofen and rest usually provide relief. Her mom and sister have a history of similar headaches.

Vital signs: T 98.8°F, HR 95, BP 120/80, RR 18, sPO_2 97% on room air.

Physical Exam: She is uncomfortable and sits with the blanket covering her eyes. Heart and lung exam are normal. There is no abdominal tenderness. Cranial nerve exam is normal, with no focal weakness or sensory changes. Normal coordination.

Question Prompts:

1. What is the patient's diagnosis?
    a. The patient's presentation is most consistent with a migraine.
    b. According to the International Headache Society, the diagnosis of migraine requires:
        i. at least five attacks lasting 4-72 hours

ii. Two of four pain features including:
    1. unilateral
    2. pulsating
    3. moderate to severe intensity
    4. aggravation by routine activity
iii. at least one of two associated symptoms
    1. nausea and vomiting
    2. photophobia and phonophobia

2. What diagnostic evaluation for headache should be pursued?
    a. A thorough history and physical exam is the most important part of the diagnostic evaluation for headache. In the absence of any concerning symptoms or signs, no specific diagnostic testing is indicated.
3. Discuss treatment options for this patient and their potential side effects.
    a. The main options for treatment of migraine can be classified into prophylactic and abortive measures.
    b. Prophylactic measures often involve headache "hygiene"
        i. regular sleeping/eating habits
        ii. stress reduction
        iii. avoidance of triggers
        iv. medications (triptans, amitryptyline, depakote, topiramate, and propranolol).
    c. Abortive measures used in the ED usually consists of a migraine "cocktail"
        i. Neuroleptic agents – prochlorperazine, haloperidol, metoclopramide, promethazine, chlorpromazine.
        ii. Nonsteroidal anti-inflammatory drugs (NSAIDs) – 15mg intravenous (IV) ketorolac, with lower doses many times having similar efficacy. NSAIDs alone without neuroleptic agents have a relatively low rate of success in ED patients with severe migraine symptoms.
    d. Other adjuncts (second line) - magnesium, depakote, and IV fluids.
    e. Sphenopalatine ganglion blocks are safe and easy to perform in the ED and may provide significant symptom relief in the setting of acute headache.[4]
    f. Dihydroergotamine (DHE) is another medication that may be considered, but is generally not used in the ED setting due to its potential side effect profile. Although extremely rare, DHE can potentially cause vasospasm leading to cerebral, cardiac or peripheral ischemia and is therefore contraindicated in patients with a history of cardiovascular disease or poorly controlled hypertension. However, DHE is well tolerated by most patients, with the most common adverse effects being nausea, flushing, dizziness, and rebound headache.
    g. Diphenhydramine is commonly co-administered to prevent extrapyramidal side effects of the neuroleptic agents such as akathisia and dystonic reactions. However, there is currently no evidence that diphenhydramine decreases this rate of akathisia.[5]
    h. Corticosteroids should be offered for prevention of headache recurrence in patients with a classic migraine history/status migrainosus. However, caution should be taken to avoid

steroid use in patients with poor glycemic control or recurrently in patients with frequent headaches.

i. Opioids are not recommended for regular use due to concerns about potential long-term effects and potential rebound symptoms.

Case 3: A 36-year-old male presents to the ED during your night shift. He complains of a severe headache that woke him up shortly after falling asleep. He reports similar headaches three other nights this week that resolved after an hour without treatment, and he is concerned because they are continuing and very uncomfortable. He reports sharp, severe frontal/periorbital headache on the right side with associated rhinorrhea and sweating when he has the pain. The patient has no past medical history and takes no medications.

Vital signs: T 98.8°F, HR 95, BP 120/80, RR 18, sPO_2 97% on room air.

Physical Exam: The patient appears slightly uncomfortable. His vital signs are normal. His head exam is atraumatic and unremarkable except for scleral injection and tearing of the right eye. He has no nuchal rigidity and his neurologic exam is normal.

Question Prompts:

1. What is the patient's diagnosis?
    a. The patient's presentation is most consistent with acute cluster headache. Cluster headaches manifest as attacks of severe, strictly unilateral pain which is orbital, supraorbital, or temporal, lasting 15-180 minutes and occurring from once every other day up to eight times per day.
    b. The attacks are associated with one or more of the following:
        i. conjunctival injection
        ii. lacrimation
        iii. nasal congestion
        iv. rhinorrhea
        v. facial sweating
        vi. ocular symptoms - miosis, ptosis, or eyelid edema, all of which are ipsilateral to the headache
    c. Most patients are restless or agitated during an attack. Attacks usually occur in series (cluster periods) lasting for weeks or months separated by remission periods usually lasting months or years. Attacks may be provoked by alcohol, histamine or nitroglycerin. About 10%-15% of patients have chronic symptoms without remissions.
    d. Prevalence is three to four times higher in men than in women. Age at onset is usually between 20 and 40 years.
2. Describe options for treatment and for prophylaxis of this condition.
    a. High flow oxygen therapy and injectable or nasal triptans are considered to be first line agents for treatment of cluster headache.[6] There is limited evidence that IV corticosteroids

may also be effective to acutely decrease frequency of attacks and transition patient to prophylactic therapy.

b. Due to the sporadic nature of symptoms, prophylaxis may be considered the most important aspect of treatment in cluster headache. Agents that have been successfully used for suppression of cluster headache include verapamil, lithium, topiramate, valproate, and baclofen.

3. Discuss the role of regional anesthesia for treatment of this condition.
    a. A variety of cranial nerve blocks have been used to treat trigeminal autonomic cephalgias. These include supraorbital, auriculotemporal, and supratrochlear blocks. However, the best evidence for efficacy has been found with greater occipital nerve blocks. Occipital nerve block has been shown to decrease number of attacks and reduce time to remission in cluster headache.
    b. While these may be most effective in patients with trigeminal autonomic cephalgia- type headaches, many other primary headache syndromes may benefit from regional anesthesia.
    c. While outside the scope of this small group session, facilitators may want to review these potential procedures and highlight key steps during the small group session as time allows.

Suggested Reading:

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Appendix B:
Subarachnoid Hemorrhage

Objectives

1. Review the epidemiology and clinical presentation of subarachnoid hemorrhage (SAH).
2. Discuss the diagnostic workup of SAH.
3. Discuss the approach to management of the patient with SAH.

Case Studies

Case 1: A 24-year-old female is brought to the emergency department (ED) by ambulance for severe headache. She has a history of migraine headaches but states that this is more intense than any prior headache. When taking her history, you discover that her typical headache is unilateral and associated with an aura and that this headache was more sudden in onset and more diffusely painful. She was evaluated by a midlevel provider and medicated with ketorolac, prochlorperazine, and diphenhydramine intravenously (IV) while her case was being staffed. She tells you that these medications nearly completely resolved her headache.

Vital signs: Temperature (T) 99.4°F, heart rate (HR) 95, respiratory rate (RR) 18, blood pressure (BP) 148/92, and oxygen saturation (O_2 sat) 99% on room air.

Physical Exam: The patient appears uncomfortable but is alert and oriented. Neurologic exam is normal. There is no meningismus or rash. Remaining physical exam is normal.

Question Prompts:

1. Describe the epidemiology, risk factors, and pathophysiology of this condition.
    a. The incidence of SAH varies widely in different regions of the world, which may be related to genetic differences between populations. Estimated incidence in the US is approximately 30,000 per year. In general, the incidence and prevalence of SAH increases with rising age. Women are more frequently affected than men.
    b. Non-traumatic SAH is most commonly caused by the rupture of a cerebral aneurysm (75%) or brain arteriovenous malformation (AVM, 10%). Accordingly, personal or family history of cerebral aneurysm (or associated disorders such as polycystic kidney disease or certain connective tissue disorders) confers increased risk of SAH.
    c. Hypertension, tobacco and alcohol abuse, and use of sympathomimetic drugs also increase risk of SAH.
2. Should diagnostic evaluation of headache be pursued in this case presentation?

a. Diagnostic evaluation should be pursued because this patient exhibits two symptoms which may be considered potential "red flags" for serious secondary headache. She reports that this is the worst headache of life, and that this is a significant change in character from her typical migraine. These symptoms are concerning for SAH.
b. Others features suggestive of SAH include maximal headache within a few minutes of onset, onset with exertion, onset associated with syncope, neck pain and history of a "sentinel" headache in the days to weeks prior.
c. Response to treatment is *not* indicative of benign headache as approximately one third of patients with SAH have resolution of headache with medication.

3. What are the potential strategies for diagnostic evaluation of SAH?
    a. The sensitivity of non-contrast head computed tomography (CT) for detecting SAH approaches 100% when read by an experienced neuroradiologist within the first six hours after symptom onset. However, sensitivity falls to 50% by 1 week. Further testing should be done beyond non-contrast CT such as lumbar puncture or CT angiogram if the headache symptoms started more than 6 hours prior.[3]
    b. Therefore, in other cases with clinical suspicion of SAH, additional testing to rule out SAH must be performed. Either lumbar puncture (LP) or CT angiography (CTA) may be performed as the next step in diagnostic workup for SAH. The combination of non-contrast head CT + LP has 100% sensitivity for detecting SAH, but specificity of only about 65%.[4] Because the majority of SAH is caused by cerebral aneurysms, CTA can be useful to rule out presence of bleeding aneurysm as well as guide therapy for aneurysm repair. CTA is 98% sensitive and 100% specific at detecting aneurysms greater than 3mm in the setting of SAH. The risk of SAH with negative CT + CTA is estimated to be less than 1%.[5] However, the discovery of incidental non-ruptured aneurysms may lead to potentially unnecessary downstream testing; therefore, CTA should be reserved only for select patients with a reasonable suspicion of SAH.
4. How do you interpret spinal fluid analysis in the context of potential SAH?
    a. Cerebrospinal fluids (CSF) analysis is typically accomplished by performing cell counts on four sequentially collected tubes of CSF. The likelihood of SAH is inferred from the number or percent change in RBCs between the first tube of CSF and the last tube of CSF collected.
    b. Unfortunately, it is difficult to determine if CSF RBCs are due to true SAH or incidental to perforation of epidural vessels during lumbar puncture. Therefore, algorithms using CSF RBC count tend to overestimate the likelihood of SAH. One small study showed that RBCs <100 in the final tube were not associated with SAH, whereas RBCs >10,000 increased odds of SAH by 6-fold. In addition, a percent change of less than 63% between the first tube and the final tube is more likely to represent true SAH.[6] Given the limited sample size, however, there are still no definite guidelines to suggest a number or percentage of RBCs that can distinguish true SAH. If the results of CSF analysis are ambiguous, neurosurgery should be consulted to guide decisions about further diagnostic testing.
    c. The presence of xanthochromia, a byproduct of heme breakdown, is thought to be a more specific marker of SAH. However, it may take up to 12 hours to develop xanthochromia after SAH, and some hospital laboratories utilize manual (visual) detection of

xanthochromia which is not considered to be as sensitive as spectrophotometry. Therefore, detection of xanthochromia may not be a reliable indicator of SAH in all settings.

Case 2: A 49-year-old male with no known past medical history presents to the ED via ambulance complaining of a severe headache while sitting at his desk at work about one hour ago. It started suddenly and reached maximal intensity within a few seconds. He said it felt like something hit him out of nowhere. After the pain started, he had a near-syncopal episode that prompted colleagues to call emergency medical services. On exam, patient is alert and oriented, well developed, and well nourished. He appears slightly uncomfortable.

Vital signs: T 99.4°F, HR 91, RR 18, BP 168/110, and O_2sat 99% on room air.

Physical Exam: His head is atraumatic, cervical spine is nontender, and heart and lungs are normal. There are no focal deficits noted on neurologic exam.

A non-contrast CT of the head is performed and shown below:

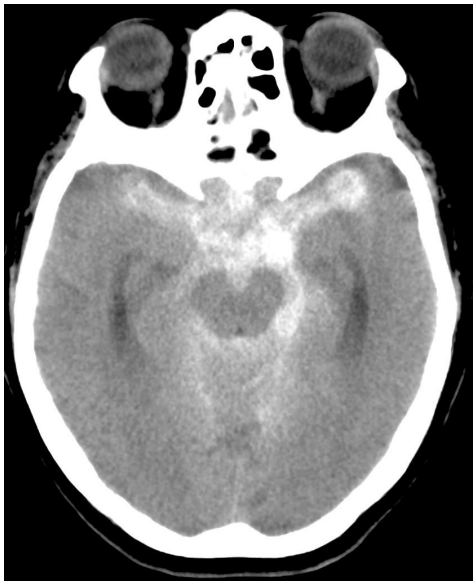

Image source: Gaillard F. Subarachnoid hemorrhage. In: Radiopaedia. rID: 2995. https://radiopaedia.org/cases/sah?lang=us. CC BY-NC-SA 3.0.

Question Prompts:

1. How would you approach the management of this patient? Describe potential interventions that may benefit this patient.
    a. The goal of management is to prevent re-bleeding which is associated with increased morbidity and mortality.
    b. Surgical clipping or endovascular coiling of the ruptured aneurysm is the definitive therapy for SAH and should be performed as soon as possible to reduce risk of re-bleeding.

c. Tight blood pressure control is recommended to reduce the risk of hypertension-related re-bleeding until the patient has received definitive therapy. There is a paucity of data to support specific blood pressure parameters; however, a goal of systolic blood pressure (SBP) less than 140-160mm hg is usually recommended. Hypotension should be avoided because the resulting decrease in cerebral perfusion pressures may lead to cerebral ischemia and worsen outcomes. Mental status can be used as a marker for adequate cerebral perfusion pressure, and thus in awake/alert patients aggressive blood pressure goal of less than 140mm hg can be obtained. In comatose or intubated patients, be cautious in aggressively lowering blood pressure because it is unclear if they are maintaining adequate cerebral perfusion pressure, and such decisions should be made in consultation with a neurosurgeon.
d. Potential anti-hypertensive agents include labetalol, nicardipine, or enalapril.

2. What complications may be expected as a result of this condition? What therapies should be initiated to prevent and treat these complications?
    a. Early complications of SAH include hydrocephalus and seizure, while the major late complication is delayed cerebral ischemia from vasospasm.
    b. Acute hydrocephalus occurs in a substantial proportion of patients with SAH. Management consists of CSF diversion via external ventricular drain or lumbar drain. Aggressive blood pressure management should be delayed for these patients until the hydrocephalus is treated; however, there are no clear guidelines for blood pressure goals in this patient population.
    c. Up to 25% of patients with SAH experience seizures. Risk factors for developing seizures in the setting of SAH include middle cerebral artery (MCA) aneurysm, associated intracerebral hematoma, re-bleeding, infarction, and poorly controlled hypertension. Antiepileptics should be initiated for patients with active seizure and may be considered in patients with known risk factors.
    d. Delayed cerebral ischemia (DCI) associated with cerebral artery vasospasm is a major cause of death and disability in patients with SAH. Oral nimodipine 60mg every 4 hours for 21 days has been shown to improve neurologic outcomes and should be administered to all patients with SAH within 96 hours of symptom onset. Maintenance of euvolemia is recommended to prevent DCI, and hemodynamic augmentation (via hypervolemia and hypertensive agents) is recommended to treat DCI.

Suggested Readings:

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Appendix C:
Intracerebral Hemorrhage

Objectives

1. Review epidemiology, pathophysiology and risk factors for development of intracerebral hemorrhage (ICH).
2. Describe clinical presentation of ICH. Discuss how the clinical presentation differs from ischemic stroke.
3. Discuss the diagnostic workup for ICH.
4. Discuss the approach to initial management of the patient with ICH.

Case Studies

Case 1: A 56-year-old female with a past medical history of hypertension and diabetes presents to the emergency department (ED) via emergency medical services (EMS) as a stroke alert with complaints of acute right-sided weakness while at work approximately 45 minutes ago. Emergency medical services reports that the patient was alert and oriented with clear speech on their arrival, but that she has decompensated when pulling into the ambulance bay. On arrival in the ED, the patient is actively vomiting.

Vital Signs: Heart rate (HR) 45, Blood pressure (BP) 210/100, respiratory rate (RR) 18, oxygen saturation (O_2sat) 86%.

Physical exam: A quick bedside assessment reveals that she has moderate confusion, garbled speech and R hemiparesis.

You direct EMS to bypass the computed tomography (CT) scanner and place the patient in a critical care bay.

Question Prompts:

1. What is the suspected diagnosis? How does the clinical presentation differ from ischemic stroke?
    a. The suspected diagnosis is ICH. Symptoms of ICH overlap with those of ischemic stroke and often include focal neurologic symptoms. However, symptoms of increased intracranial pressure (ICP) such as headache, nausea, vomiting, seizures, and decreased level of consciousness often dominate the clinical picture in ICH. The presentation also differs from that of ischemic stroke in that symptoms typically progress over minutes to hours.
    b. Additionally, as in our case, brain herniation is much more common with ICH and often develops within 24 hours, sometimes prior to ED arrival. Herniation can be identified by an acute change in mental status, or an abrupt onset of Cushing's reflex (hypertension, bradycardia and irregular breathing). On the other hand, in ischemic stroke, herniation is less common and often due to large territory infarcts, and typically develops with a delayed

onset (greater than 24 hours). In ischemic stroke, intracranial pressure increases more gradually as a result of developing brain edema.

2. Why was the patient diverted from the CT scanner? What special considerations must be taken in the management of airway, breathing and circulation (ABCs)?
    a. The patient presented with a rapid decline in mental status which carries a high risk of airway compromise due to depressed level of consciousness. Patients with ICH require close monitoring and frequent re-assessment of neurologic status because 20% of patients decline by two or more Glasgow Coma Scale (GCS) points from the time of initial evaluation by EMS to the time of arrival in the ED. An additional 20% show further decline in GCS over the first few hours after arrival. Therefore, you must maintain readiness to intervene on ICH patients for airway protection.
    b. Patients with ICH may also have respiratory insufficiency due to neurogenic pulmonary edema or comorbid conditions such as hypertensive heart failure that can produce hypoxemia and worsen stroke symptoms. A quick assessment of oxygenation, respiratory rate and respiratory effort will help determine if the patient may require intubation for primary ventilatory support even if the airway is intact.
    c. Finally, patients with confirmed ICH must have blood pressure tightly controlled to avoid further neurologic deterioration. Rapidly decreasing systolic blood pressure (SBP) to goal of 140 is safe and improves neurologic outcomes. However, hypotension must be strictly avoided to prevent further decreases in cerebral blood flow to at-risk tissues.
3. Describe the neuroprotective strategy for airway management in the patient with ICH.
    a. The goal is to blunt the reflex-mediated rise in ICP associated with laryngoscopy and prevent further neurologic deterioration. As such, it is important to ensure that the patient has adequate sedation and analgesia prior to intubation. Fentanyl (3-5 mcg/kg) and esmolol (1.5-2mg/kg) have been shown to blunt the hemodynamic response to laryngoscopy and may be useful in blunting reflex rise in ICP. However, there is no clear evidence that either of these agents improves outcomes.
    b. If used, these agents should be administered 3 minutes before induction of anesthesia. Pre-oxygenation of the patient should also occur during this time.
    c. Blood pressure control should ideally be initiated before intubation. However, push-dose vasopressors should also be available at the bedside because the agents used for pretreatment may cause transient hypotension.
    d. The patient should remain upright (head of bed 30 degrees or higher) until conditions have been optimized and all equipment is ready to intubate the patient.

Case 2: A 78-year-old male with a history of hypertension and atrial fibrillation presents to the ED via EMS after family found him lying on the floor confused and unable to stand up. In the ED, the patient is lethargic, opening eyes only to stimulation.

Vital signs: HR 63 (irregular), BP 183/99, RR 14, O_2sat 94%.

Physical exam: He is confused, but follows simple commands with all four extremities.

A CT is obtained immediately that demonstrates the following:

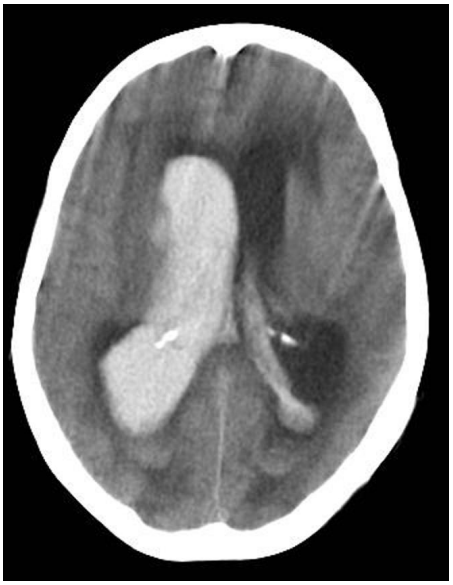

Image source: Jones J. Intracerebral hemorrhage with ventricular extension. In: Radiopaedia. rID: 6223. https://radiopaedia.org/cases/intracerebral-haemorrhage-with-ventricular-extension?lang=us. CC BY-NC-SA 3.0.

Question Prompts:

1. Describe epidemiology, pathophysiology and risk factors for development of ICH.
    a. Approximately 115,000 Americans present with spontaneous ICH per year. Intracranial hemorrhage carries the highest morbidity and mortality amongst all stroke subtypes, with 30-day mortality approaching 45%-55% in most populations despite medical care.
    b. Hypertensive and amyloid angiopathy are the leading causes of spontaneous ICH. Uncontrolled hypertension leads to accelerated atherosclerosis and arteriosclerosis of medium and small penetrating vessels within the deep structures of the brain, while amyloid angiopathy leads to fibrinoid necrosis of intraparenchymal cortical vessels. Both types of angiopathy may predispose to microaneurysm formation and make the vessel wall brittle and vulnerable to shear stress caused by elevated blood pressure.
    c. The most common secondary cause of ICH is anticoagulation. Other less frequent etiologies include arteriovenous malformation (AVM), cavernous malformation, aneurysm, conversion of an ischemic stroke, tumor, and sinus thrombosis.
    d. Regardless of etiology, an enlarging hematoma is associated with increased morbidity and mortality. Hematoma expansion leads to local mass effect, intraventricular extension and hydrocephalus (independent predictor of mortality), and eventual herniation. Secondary brain injury may also be due to toxic effects of hemoglobin breakdown and other inflammatory mediators.
2. Discuss the diagnostic workup for ICH.

a. The diagnostic workup for ICH should include non-contrast head CT and point-of-care glucose to exclude alternate diagnoses. Complete blood count (CBC), metabolic panels and coagulation studies should also be obtained to screen for potential bleeding diatheses and guide medical therapy.
b. Computed tomography angiogram may be useful when available to rule out vascular abnormalities and discern a "spot sign," an area of contrast extravasation in the hematoma which may represent the site of initial vessel breakdown.
c. The spot sign is associated with greater hematoma expansion and poor outcome; therefore, patients with this sign may need more aggressive medical management, as well as more frequent and earlier repeat scans to ascertain stability.

3. What is the approach to initial management of the patient?
    a. Hematoma growth is associated with higher blood pressures[2]. Rapidly decreasing SBP to goal of less than 140 mmHg is safe and improves neurologic outcomes compared to conservative control.[3] There is no consensus on best agents to use. Nicardipine or labetalol drips are commonly used, but also consider bolus dosing of labetalol, hydralazine or enalapril. Avoid hypotension because it may lead to reduced cerebral blood flow and worsening symptoms. If patients have refractory hypertension despite treatment, consider better pain control.
    b. Reversal of endogenous or medication-induced bleeding diatheses is important to limiting hematoma expansion. Patients on anticoagulant therapy should be reversed with the appropriate reversal agent [fresh frozen plasma (FFP) + vitamin K or prothrombin complex concentrate (PCC)] for most oral anticoagulants, protamine sulfate for heparin-based anticoagulants). Replacement of factor or platelets is indicated for patients with known factor deficiencies or platelet disorders.
    c. Control ICP.
        i. Maintain the head of the bed at 30 degrees and ensure adequate pain control and/or sedation.
        ii. Optimize ventilation to titrate RR to $paCO_2$ of 35-38 mmHg.
        iii. Osmotic agents such as mannitol or hypertonic saline can be used for patients with symptoms of increased ICP.
        iv. Some patients may benefit from early surgical intervention – early consultation with a neurosurgeon is always indicated.
    d. Other considerations in the initial management of ICH include antiepileptics (levetiracetam preferred) for treatment of seizures, early neurosurgical consultation because patient may eventually need ventricular drain or decompressive craniotomy, and early admission to a neurocritical care ICU.

Suggested Readings:

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Appendix D:
Seizures

Objectives

1. Review the different types of possible seizures including status epilepticus.
2. Discuss management of a patient presenting with a first-time seizure versus a patient with a previous history of seizures.
3. Discuss management of status epilepticus.
4. Discuss the differential for non-epileptic causes of seizures.
5. Differentiate between the definition and management of simple febrile seizures and complex febrile seizures.

Case Studies

Case 1: A 27-year-old male presents by emergency medical services (EMS) after he was reportedly found down with jerking movements. The duration of the event is unknown. He was not shaking and generally unresponsive when the ambulance arrived. He was breathing spontaneously and started moaning as they unloaded him into the emergency department (ED). He was still not following commands and responding to voice.

Past medical history, medications and social history are unknown at this point.

Vital signs: Heart rate (HR) 96, Respiratory rate (RR) 12, blood pressure (BP) 144/82, Temperature (T) 99.0°F, oxygen saturation (O_2sat) 97%.

Physical exam: Patient is alert but confused. Moving all extremities equally. No evidence of head trauma. Oropharynx is normal.

Question Prompts:

1. What physical exam findings would be suggestive of seizure?
   Check for:
   a. Tongue/mouth lacerations
      i. Sides of tongue more often bitten than tip of tongue.
      ii. Tongue biting has sensitivity of ~25% and approaches 100% specificity in lateral tongue biting.
   b. Urinary incontinence
   c. Posterior shoulder dislocation

d. Focal motor deficit, known as Todd's paralysis (but keep ischemic stroke and intercranial hemorrhage on the differential in patients with unilateral weakness).
e. While not suggestive of seizure, it is also important to address potential traumatic injuries to the head and cervical spine, or other injuries such as lacerations.

2. What diagnostic tests are useful in the evaluation of a first-time seizure? Recurrent seizure?
    a. In new onset seizure OR recurrent seizures with change in baseline frequency consider:
        i. Electrocardiogram (ECG) to evaluate for long QT, torsades de pointes or other arrhythmia.
        ii. Pregnancy test if female.
        iii. Glucose (evaluate for hypoglycemic cause of seizure), electrolytes (primarily to evaluate for hyponatremia).
        iv. Other recommended tests if warranted by history or exam – liver function tests especially if on antiepileptic medications, urinalysis, urine drug screen.
        v. Brain imaging - non-contrast computed tomography (CT) should be obtained on most adult patients presenting with first-time seizure. However, it may be reasonable to obtain outpatient magnetic resonance imaging (MRI) in patients at low risk of malignancy (younger age) so long as physical exam and history in the ED are otherwise reassuring.
        vi. Neurology evaluation – in most cases this can be done as an outpatient.
        vii. Electroencephalogram (EEG) in patients who fail to return to baseline within 60 minutes, otherwise can arrange for outpatient EEG.
        viii. Lumbar puncture (LP) in patients suggestive of infectious etiology such as meningismus, persistent altered mental status, and/or fever.
    b. In patients with recurrent seizures without change in baseline frequency consider:
        i. Anticonvulsant drug concentration.
        ii. Point-of-care glucose.
        iii. Check for signs of trauma and cervical spine tenderness.
        iv. Otherwise these patients can be managed with little ED testing.
3. What is your initial management in this patient? What is the disposition?
    a. Initial management would likely be first-time seizure work-up as described above. Ideally providers could reach out to family/friends to obtain additional history to see if the patient has a previous history of seizures.
    b. Disposition would be based on diagnostic work-up findings, underlying etiology if found, and return to baseline.
4. What would you include with your discharge instructions for a first-time seizure patient who can be discharged from the ED?
    a. Instructions not to drive, swim, bathe or participate in other potentially dangerous activities, especially alone, are important.
        i. States vary in driver licensing requirements regarding episodes of loss of consciousness, including the responsibilities of clinicians to notify state authorities.
        ii. One suggested resource to see laws in your practice location can be found at the epilepsy foundation website - https://www.epilepsy.com/driving-laws.

b. First-time seizure patients should be aware of common seizure triggers or precipitating factors, including sleep deprivation, alcohol, substance use, medications (isoniazid), caffeine, and infection or systemic illness.

5. Besides a primary seizure disorder such as epilepsy, what other diagnoses should be on the differential for first time seizure?
    a. Acute symptomatic seizure refers to a seizure that occurs at the time of a systemic insult or in close temporal association with a documented brain insult, such as metabolic derangements, drug or alcohol withdrawal, and acute neurologic disorders such as stroke, encephalitis, or acute head injury. These account for 25%-30% of first-time seizures.
        i. Acute ischemic or hemorrhagic stroke, particularly lobar hemorrhage.
        ii. Subdural hematoma.
        iii. Subarachnoid hemorrhage.
        iv. Traumatic brain injury.
        v. Hypoxic-ischemic injury.
        vi. Brain abscess.
        vii. Meningitis or encephalitis.
        viii. Hypoglycemia – common in diabetic patients who take excessive amounts of insulin or oral hypoglycemic agents. Islet cell tumors are much less common, but seizures may be the initial presentation.
        ix. Hyperglycemia – non-ketotic hyperglycemia most commonly occurs in diabetic older adults and can cause focal motor seizures.
        x. Hyponatremia – precipitous falls in serum sodium concentrations can trigger generalized tonic-clonic seizures, usually in association with a prodrome of confusion and depressed level of consciousness.
        xi. Hypocalcaemia – most often occurs in neonates. In adults, hypocalcaemia may occur after thyroid or parathyroid surgery or in association with renal failure, hypoparathyroidism, or pancreatitis.
        xii. Hypomagnesaemia – magnesium levels below 0.8 mEq/L may result in irritability, agitation, confusion, myoclonus, tetany, and convulsions, and may be accompanied by hypocalcemia.
        xiii. Uremia – renal failure and uremia are often associated with seizures, particularly myoclonic seizures. Generalized tonic-clonic seizures occur in approximately 10% of patients with chronic renal failure. Seizures may also occur in patients undergoing dialysis as part of the dialysis disequilibrium syndrome.
        xiv. Hyperthyroidism.
        xv. Disorders of porphyrin metabolism – acute intermittent porphyria.
        xvi. Eclampsia – during pregnancy or the early post-partum period.
        xvii. Withdrawal states – particularly alcohol and benzodiazepine withdrawal, is associated with seizures.
        xviii. Drug intoxication, poisoning, and overdose – cocaine, amphetamines, phencyclidine (PCP) and other illicit substances. Isoniazid, tricyclic antidepressants, and sodium channel blockers.

6. What is the management of status epilepticus?
    a. Goals of status epilepticus management:
        i. Resuscitation (see below) to prevent secondary brain injury and maintain cerebral perfusion pressure.
        ii. Terminate seizure.
        iii. Monitor for ongoing seizure.
        iv. Diagnose and treat cause.
        v. Treat complications.
        vi. Obtain appropriate disposition.
    b. Resuscitation
        i. Attend to airway, breathing and circulation (ABCs) and address life threats.
        ii. Manage airway with recovery position, airway adjuncts, and intubation if required.
        iii. Optimize oxygenation and provide ventilatory support as needed (prone to hypercapnia).
        iv. Continuous EEG monitoring may be necessary following neuromuscular blockade for intubation to detect ongoing subclinical seizures.
        v. Early intravenous or intraosseous access.
        vi. Optimize cerebral perfusion pressure.
        vii. Treat hypoglycemia and life-threatening electrolyte disturbance if present.
        viii. Maintain normothermia.
        ix. Give relevant antidote if due to toxic agent (*eg,* pyridoxine for isoniazid).
    c. Terminate seizure
        i. First-line therapies: bolus dose benzodiazepines
            1. Midazolam 0.1mg/kg IV – also buccal or intramuscular (IM). IM is not inferior to IV lorazepam. Intranasal midazolam at dose of 0.2mg/kg may also be used, often reserved for small children because of the lower doses required.
            2. Lorazepam 0.1mg/kg IV. Onset in 3-5 minutes and lasts hours. Preferred for longer acting effects.
            3. Diazepam 0.15mg IV/rectal (PR) (avoid IM as painful) up to 10 mg. Onset in approximately 1 minute but lasts only about 20 min for anti-seizure activity.
            4. Clonazepam 0.15 mg/kg (common outside of US).
        ii. Second-line therapies. These may require intubation and mechanical ventilation.
            1. Fosphenytoin 15-20 mg/kg IV over 30 minutes or longer.
                a. Avoid rapidly pushing this medication.
                b. Contraindicated in patients with 2nd and 3rd degree heart block.
                c. Fosphenytoin is not available in Australia.
            2. Valproic acid 40 mg/kg IV over 10 min. May give additional 20 mg/kg over 5 min if still seizing.
            3. Leviteracetam 60 mg/kg IV, max dose 4.5 g.
        iii. Third-line therapies for refractory status epilepticus. These nearly always require intubation and ventilation and continuous EEG monitoring.
            1. Propofol 2-5mg/kg, then infusion of 2-10mg/kg/hr.

2. Midazolam 0.2mg/kg, then infusion of 0.05-2mg/kg/hr.
3. Ketamine loading dose 0.5 to 3 mg/kg, followed by infusion of 0.3 to 4 mg/kg/hr.
4. Lacosamide IV 400 mg IV loading dose over 15 min, then maintenance dose of 200 mg q12hrs oral (PO) or IV.
5. Phenobarbital IV 15-20 mg/kg at 50-75 mg/min, then continuous infusion at 0.5-4.0 mg/kg/hr with dose adjusted to suppression-burst pattern on continuous EEG.
6. Consider consulting anesthesia for inhaled anesthetics.

iv. Monitor for ongoing seizure
1. Avoid neuromuscular blockade.
2. Observe for muscular activity.
3. Continuous EEG to exclude non-convulsive status epilepticus in patients who do not return to normal conscious state.
4. A period of 24–48 hours of electrographic control is recommended prior to slow withdrawal of continuous infusion.

v. Treat underlying cause
1. Antibiotics – bacterial infection.
2. Antivirals – viral infection.
3. Abscess – surgery.
4. Increased intracranial pressure – neurosurgical decompression.
5. Eclampsia – magnesium and early delivery of baby and placenta.
6. Isoniazid overdose – pyridoxine.
7. Cholinergic syndrome – atropine, pralidoxime if organophosphate poisoning.
8. Sodium channel blocker overdose – sodium bicarbonate, intralipid.

vi. Prevent and treat complications
1. Aspiration.
2. Neurogenic pulmonary edema.
3. Rhabdomyolysis.
4. Hyperthermia.
5. Trauma (*eg,* head injury due to fall, posterior shoulder dislocation).
6. Todd's paralysis (may last 24 hours).

vii. Disposition
1. Patients with status epilepticus generally require intensive care admission.
2. Neurology consult.

Case 2: An 11-month-old female presents by EMS after reported seizure. Parents noted that one hour prior to arrival, her eyes rolled back in her head and she demonstrated jerking movements in both arms and legs. She subsequently developed central cyanosis. The event lasted for approximately three minutes and she returned to being somnolent, but interactive by the time EMS arrived. Parents have noted recent nasal discharge and cough. The child is unvaccinated.

Vital signs: T101.3°F, HR 144, RR 25, O_2sat 99% on room air.

Physical exam: Fussy appearing baby. Nasal discharge present. Breath sounds with transmitted upper airway sounds but clear after coughing. No murmur. Extremities pink, capillary refill less than two seconds. Moving all extremities. Anterior fontanelle flat. Making tears when crying.

Question Prompts:

1. What are the criteria for a febrile seizure and how is a simple febrile seizure different from a complex seizure?
    a. Simple febrile seizure
        i. Associated with temperature greater than 38°C (100.4°F). Fever does not need to be present at the time of the seizure and may occur hours later.
        ii. Six to 60 months of age.
        iii. Less than 15 minutes duration.
        iv. Generalized (without a focal component).
        v. Occur once in a 24-hour period.
    b. Complex febrile seizures
        i. Prolonged (greater than 15 minutes duration) or
        ii. Focal or
        iii. Occur more than once in 24 hours.
2. What diagnostic tests are useful in the evaluation of a simple febrile seizure? Complex febrile seizure?
    a. Simple febrile seizure
        i. Detailed history and physical.
        ii. Point-of-care glucose.
        iii. Evaluation and treatment of obvious/common sources of infection/fever (acute otitis media, streptococcal pharyngitis, urinary tract infection).
        iv. Follow up with primary care provider within 48 hours.
        v. Not supported by evidence:[5]
            1. Immediate neurology evaluation.
            2. Immediate brain imaging.
            3. Extensive laboratory workup, including LP.
                a. Lumbar puncture should be performed when there are meningeal signs or symptoms or other clinical features that suggest a possible meningitis or intracranial infection.
                b. Lumbar puncture should be considered in infants between 6 and 12 months if the immunization status for *Haemophilus influenzae type b* (Hib) or *Streptococcus pneumoniae* is deficient or undetermined.
                c. Lumbar puncture should be considered when the patient is on antibiotics because antibiotic treatment can mask the signs and symptoms of meningitis.

b. Complex febrile seizure
    i. Detailed history and physical.
    ii. Evaluation and treatment of obvious/common sources of infection (acute otitis media, streptococcal pharyngitis, urinary tract infection).
    iii. Detailed lab evaluation (*eg,* LP) if exam/history warrants.
    iv. Consider CT if:
        1. Persistently abnormal neuro exam (especially with focality).
        2. Signs/symptoms of increased intracranial pressure such as altered mental status, nausea, vomiting, papilledema, and lateral gaze palsy.
        3. Patient has a ventriculoperitoneal (VP) shunt.
    v. Consider ECG if family history of long QT, Brugada, or sudden unexplained death/drowning.
    vi. Routine EEG not indicated - consider only if developmental delay or for focal symptoms.
    vii. Consider causes amenable to specific treatment
        1. Hypoglycemia.
        2. Hyponatremia (water intoxication, dilution of formula).
        3. Hypocalcemia.
        4. Hypomagnesemia.
        5. Isoniazid ingestion.
    viii. Consider consultation with neurology/infectious disease.
    ix. Consider admission for observation vs. 24-hour primary care follow-up.
    x. Consider discharge with abortive seizure medication.

3. What is your initial management of this patient? What is the disposition?
    i. Detailed history and physical.
    ii. Point-of-care glucose.
    iii. Evaluation and treatment of obvious/common sources of infection/fever as above.
    iv. Lumbar puncture should be considered because patient is between 6 and 12 months with deficient Hib and *Streptococcus pneumoniae* immunizations.
    v. Shared decision making: Follow up PCP in 24 hours vs. observation/admission due to immunization status, pending workup.
4. How do you counsel the parents of a patient who had a febrile seizure?
    a. Anticipatory guidance:
        i. Febrile seizure likely to recur until age of 5 years.
        ii. Anticonvulsants are not indicated due to the self-limited nature of febrile seizure side-effect profile.
        iii. Risk of developing epilepsy very low.
        iv. Use antipyretics but know they will not prevent febrile seizure.
    b. Seizure first aid:
        i. Lay patient on floor on the patient's side. This will help maintain airway and help prevent aspiration if patient vomits.
        ii. Do not place anything in a seizing patient's mouth.

iii. Patients will not swallow their tongue.

c. Return if:
   i. Multiple recurrence of seizure in 24 hours, especially without return to baseline between events.
   ii. Change in behavior or mental status.
   iii. Does not return to baseline after seizure.
   iv. If a seizure lasts more than 5-10 minutes.
   v. Underlying cause of fever becomes worse.
   vi. Any other parental concerns.

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Appendix E:
Acute Ischemic Stroke and Transient Ischemic Attack

Objectives

1. Describe epidemiology, pathophysiology, and risk factors for development of acute ischemic stroke (AIS).
2. Describe clinical presentation of AIS and transient ischemic attack (TIA). Discuss the other clinical entities which form the differential diagnosis.
3. Discuss the diagnostic workup for AIS including the National Institutes of Health Stroke Scale (NIHSS).
4. Discuss the approach to initial management of the patient with AIS in the emergency department (ED).
5. Discuss options for the management of TIA.

Case Studies

Case 1: A 56-year-old female with a past medical history significant for hypertension presents to the ED complaining of an episode of left-hand weakness which caused her to drop her phone. A family member also reported that the patient's left face was droopy and she appeared to be drooling from the left side of her mouth. The episode lasted for two to three minutes before spontaneously resolving. She denies any other symptoms and now feels back to baseline.

Vital signs: Temperature (T) 99.0°F, heart rate (HR) 92, blood pressure (BP) 106/78, respiratory rate (RR) 18, oxygen saturation (O_2sat) 97% on room air.

Physical exam: Well appearing, normal heart and lung sounds, no abdominal tenderness, normal cranial nerve exam, no focal weakness or sensory deficits.

Question Prompts:

1. What is the patient's diagnosis? How is it defined?
    a. The patient likely had a TIA. Transient ischemic attack is currently defined as a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction (no evidence of permanent tissue damage on neuroimaging). Typical duration of symptoms is less than two hours but longer duration is possible.
    b. Transient ischemic attack was classically defined as brief episodes of neurological dysfunction resulting from focal cerebral ischemia which resolve within 24 hours.
2. What are the incidence of and risk factors for AIS and TIA?
    a. Approximately 240,000 people experience a TIA and 795,000 people experience a new or recurrent stroke each year. Over the past 10 years, the death rate from stroke has fallen by

34%. However, stroke is still the 5th leading cause of death in the United States, killing nearly 130,000 people a year.

b. The incidence and prevalence of stroke and TIA increase greatly with advancing age. Men have a greater risk of stroke in most age groups. However, women have a greater lifetime risk of stroke, primarily due to longer lifespan. African-Americans, Hispanics, and Native Americans carry the highest risk of new stroke or TIA.

c. Primary risk factors for AIS and TIA include hypertension, smoking, obesity, hyperlipidemia, diabetes, carotid artery atherosclerosis/stenosis, and atrial fibrillation.

3. What is the strategy for management of the patient with TIA in the ED?

a. The estimated risk of stroke after TIA ranges from 3%-10% at two days and 9%-17% at 90 days. Therefore, the primary focus of ED management is to establish the patient's near-term risk of stroke to determine whether the patient is appropriate for inpatient or expedited outpatient workup.

b. One commonly used clinical decision tool is the $ABCD^2$ score, which is a risk assessment tool optimized to predict the short-term risk of stroke after a TIA. Current guidelines recommend hospitalization for workup and observation if the $ABCD^2$ score is greater than 3. However, recent studies have shown that the $ABCD^2$ score performs poorly in the ED and may not add significant value to the ability to risk stratify patients with TIA compared to an ED workup which includes neuroimaging.

i. $ABCD^2$ Score:

1. **A**ge greater than 59 (+1pt),
2. **B**lood pressure - initial SBP >139 or DBP>89 (+1),
3. **C**linical Features:
   a. Symptoms were unilateral weakness (+2),
   b. Symptoms only included speech disturbance without weakness (+1).
4. **D**uration:
   a. Symptoms were 10-59min (+1)
   b. Symptoms were >59 min (+1)
5. **D**iabetes history (+1)

ii. The risk for stroke can be estimated from the $ABCD^2$ score as follows:

1. Score 1-3 (low)
   a. 2 day risk = 1.0%
   b. 7 day risk = 1.2%
2. Score 4-5 (moderate)
   a. 2 day risk = 4.1%
   b. 7 day risk = 5.9%
3. Score 6–7 (high)
   a. 2 day risk = 8.1%
   b. 7 day risk = 11.7%

iii. Patients with $ABCD^2$ score 0-3 are low risk and may benefit from expedited outpatient evaluation.

c. Other considerations for management include treatment with antiplatelet agents, treatment with anticoagulation for suspected cardio-embolic source such as atrial fibrillation, and optimization of modifiable risk factors including hypertension, hyperlipidemia and smoking cessation.
d. Many centers may have observation units for TIA protocols to obtain magnetic resonance imaging (MRI) brain, potential carotid dopplers, potential transthoracic echo, and potential neurology consult.

Case 2: A 75-year-old male with a history of hyperlipidemia and hypertension presents to the ED after acute onset of speech difficulty, impaired comprehension, and right-sided weakness. His last known normal was two hours ago.

Vital signs: T 98.7°F, HR 92, BP 106/78, RR 18, O_2sat 97% on room air.

Physical exam: The patient was found to have global aphasia, right homonymous hemianopsia, right hemiplegia, and right hemi-sensory loss.

Question Prompts:

1. What is the pathophysiology of acute ischemic stroke (AIS)?
    a. Acute ischemic stroke is caused when poor cerebral blood flow to a localized area of the brain initiates an ischemic cascade resulting in acid-base imbalance and free radical production that trigger cell death in the affected tissue.
    b. The primary mechanisms of disruption to cerebral blood flow in ischemic stroke are *in situ* thrombosis from atherosclerosis of the carotid or cerebral arteries or embolization of thrombus from the heart.
2. What are some of the other ways that AIS can present?
    a. Acute ischemic stroke may present as any form of acute neurologic deficit. In this case, the patient presented with acute aphasia, hemiplegia and hemi-sensory loss among other deficits. However, patients may also present with symptoms such as acute vertigo or ataxia/dysmetria in cerebellar strokes. A pontine stroke may also present as locked-in syndrome.
    b. Regardless of the neurologic deficits, however, the overall pattern of the deficits should usually follow a vascular distribution, except in the rare case of showered emboli. A good resource for reviewing stroke syndromes by vascular territory may be found at strokecenter.org. http://www.strokecenter.org/professionals/stroke-diagnosis/stroke-syndromes/.
3. What other clinical entities form the differential diagnosis for AIS?
    a. The differential diagnosis includes: intracranial hemorrhage, structural brain lesions such as tumor or aneurysm, central nervous system (CNS) infection, complex migraine, post-seizure neurologic deficit, peripheral neuropathies as well as non-neurologic conditions which may mimic the symptoms of stroke including hypoglycemia and conversion disorder.

4. What are the initial steps of management in AIS?
    a. The primary survey should include assessment of the patient's airway, breathing and circulation. After stabilization of any critical conditions found on primary survey, a focused history and physical (H&P) is performed to assess level of neurologic dysfunction, exclude alternate diagnoses, and determine the patient's eligibility for therapy.
    b. The H&P should include calculation of the National Institutes of Health Stroke Scale (NIHSS), a systematic assessment tool that provides a quantitative measure of stroke-related neurologic deficit. The NIHSS is a 15-item neurologic examination used to evaluate the effect of acute cerebral infarction on the level of consciousness, language, neglect, visual-field loss, extraocular movement, motor strength, ataxia, dysarthria, and sensory loss. The NIHSS can serve as a measure of stroke severity and has been shown to be a predictor of both short- and long-term outcomes of stroke patients.
        i. While providers who routinely administer the stroke scale may have its different steps memorized, it is important to use a checklist to avoid forgetting items. There are many electronic versions and applications that can help guide the NIHSS.
        ii. One suggested app for iOS is NIH stroke scale from StatCoder - https://itunes.apple.com/us/app/nih-stroke-scale-from-statcoder/id408788598?mt=8.
        iii. For android devices – NIH stroke scale from ETZ - https://play.google.com/store/apps/details?id=com.soft.etz.nihstrokescale&hl=en_US
        iv. It is recommended that facilitators highlight the NIHSS, but it is out of the scope of this small group module to fully teach the administration of the NIHSS. More information about the NIHSS can be found on the National Institutes of Health website: https://www.stroke.nih.gov/resources/scale.htm.
    c. Blood glucose, non-contrast head CT, and electrocardiogram (ECG) should be rapidly performed to exclude alternate diagnoses which would exclude the patient from intervention for AIS.
5. What are the options for intervention for ischemic stroke?
    a. The American Heart Association (AHA)/American Stroke Association (ASA) currently recommends two forms of treatment for eligible patients with AIS: Intravenous (IV) thrombolytic agents and mechanical thrombectomy.
    b. Intravenous recombinant tissue plasminogen activator (tPA) is the only Food and Drug Administration (FDA) approved treatment for ischemic stroke. Tissue plasminogen activator works by dissolving the thrombus and restoring blood flow to the affected part of the brain. Intravenous tPA is most effective if administered within three hours of onset of symptoms. Earlier administration of tPA is associated with significantly reduced in-hospital mortality, decreased rate of intracerebral hemorrhage (ICH), more frequent independent ambulation at discharge, and a higher rate of discharge to home.
    c. Thrombectomy is recommended for adults with AIS caused by large vessel occlusion [internal carotid artery (ICA), M1 or M2 segments of the middle cerebral artery (MCA)] within six hours of symptom onset and is reasonable up to 16 hours for patients meeting

additional eligibility requirements (perfusion mismatch on CT perfusion or MRI). This recommendation was based on pooled analysis of five studies which showed benefit of thrombectomy over standard care.

d. Decompressive craniectomy may be considered for selected patients with severe cerebral edema related to infarct.

6. What are the indications and contraindications of thrombolysis?
    a. Thrombolysis is indicated for adults 18 years or older with measurable neurologic deficit attributable to ischemic stroke and symptom onset less than 180 minutes (three hours) before treatment begins. It is also recommended (but not FDA approved) for a select subgroup of patients meeting additional criteria within 4.5 hours.
    b. Absolute contraindications:
        i. Acute intracranial hemorrhage (the reason for stat head CT)
        ii. Factors which may increase likelihood of ICH –
            1. Uncontrolled hypertension – systolic BP remains greater than185 mmHg or diastolic BP remains >110 mmHg despite treatment. Aggressively treat elevated BP when patients present with stroke symptoms.
            2. Bleeding diatheses
                a. Platelet count <100,000/mm3.
                b. Heparin received in prior 48 hours with elevated partial thromboplastin time (PTT).
                c. International normalized ratio (INR) >1.7.
            3. Recent intracranial or spinal surgery.
            4. Head trauma or previous stroke in last three months.
            5. Arterial puncture at non-compressible site within last seven days.
    c. Relative/controversial contraindications:
        i. Central nervous system structural lesions - known arteriovenous malformation, brain neoplasm, brain aneurysm (contraindication per AHA guidelines and drug label.)
        ii. Pregnancy – these patients were excluded from the studies. However, there is some evidence that supports that it is safe to use in this population.
        iii. Advanced age (older than 75).
        iv. Mild or improving stroke symptoms.
        v. Severe stroke or coma.
        vi. Recent major surgery.
        vii. Recent gastrointestinal or genitourinary hemorrhage.
        viii. Seizure at onset – presentation may be Todd's paralysis, or weakness due to recent seizure and not ischemic event.
        ix. Recent myocardial infarction (within seven weeks) – possible increased risk of free wall rupture.

Suggested Readings:

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Appendix F:
Vertigo

Objectives

1. Discuss features of the patient history that can help distinguish between true vertigo and other non-specific descriptions of dizziness.
2. Demonstrate physical exam techniques to distinguish between central and peripheral forms of vertigo.
3. Define acute vestibular syndrome, spontaneous episodic vestibular syndrome and triggered episodic vestibular syndrome.
4. Describe an algorithmic approach to isolated vertigo.
5. Discuss the utility of advanced imaging in the work-up of vertigo.

Case Studies

Case 1: A 45-year-old female presents to the emergency department (ED) from work by private vehicle after she developed an episode of "dizziness" while working at her computer. She complained to her coworkers who insisted that she be seen in the ED for evaluation. She described the sensation as wanting to fall off of her chair. Further clarification reveals that she feels like either she or the room is spinning around like a merry-go-round. The feeling began gradually over about two minutes and lasted about 15 minutes. It then recurred and is now persistent. She was nauseated. She improved slightly when she went to lie down on the couch, but the feeling still persisted. Nothing else that she did was able to significantly improve the symptoms. The patient also describes the sensation of "weakness" in her right arm and leg.

At the time of ED presentation, most of the symptoms had improved but the vertigo was still present.

Past medical history: Diabetes, depression, fibromyalgia.
Medications: Gabapentin, paroxetine, metformin, glimepiride, hydrocodone/acetaminophen.
Allergies: Nonsteroidal anti-inflammatory drugs (NSAIDs).
Social history: Social use only of alcohol, 1 pack per day smoker; denies recreational drug use.
Family history: Diabetes, hypertension, coronary artery disease.

Vital signs: Temperature (T) 98.5°F, blood pressure (BP) 198/112, heart rate (HR) 92, respiratory rate (RR) 16, oxygen saturation (O_2sat) 99% on room air (RA).

Physical Exam:

General:	Alert and oriented to person, place and time; white female
Head:	Atraumatic
Ears:	Within normal limits

Eyes: Pupils equally round and reactive to light, multi-direction horizontal nystagmus noted on exam with head movements
Nose: Within normal limits
Throat: Asymmetric palate rise, tongue protruding to midline
Neck: Non-tender, trachea midline; thyroid is normal
Chest: Lung sounds clear, heart sounds regular with 3+ systolic murmur
Abdomen: Soft, non-tender
Skin: Warm and dry with no rashes

Neuro:

Cranial nerve (CN) Exam:

III, IV, VI (eye movements) – Can look in all directions on command
V (facial sensory) – normal.
VII (facial motor) – normal.
VIII (auditory) – normal.
XI (glossopharyngeal) – asymmetrical palate movement with left side not elevating the same as the right.
XI (accessory) – normal.
XII (hypoglossal) – tongue protrudes to midline.

Speech: Mild slurring of speech.
Reflexes: 2+ bilaterally, Babinski is equivocal.
Sensory: Grossly intact symmetrically to light touch.
Motor: Upper extremity strength is 5/5 and symmetric; lower extremity strength is 5/5 and symmetric.

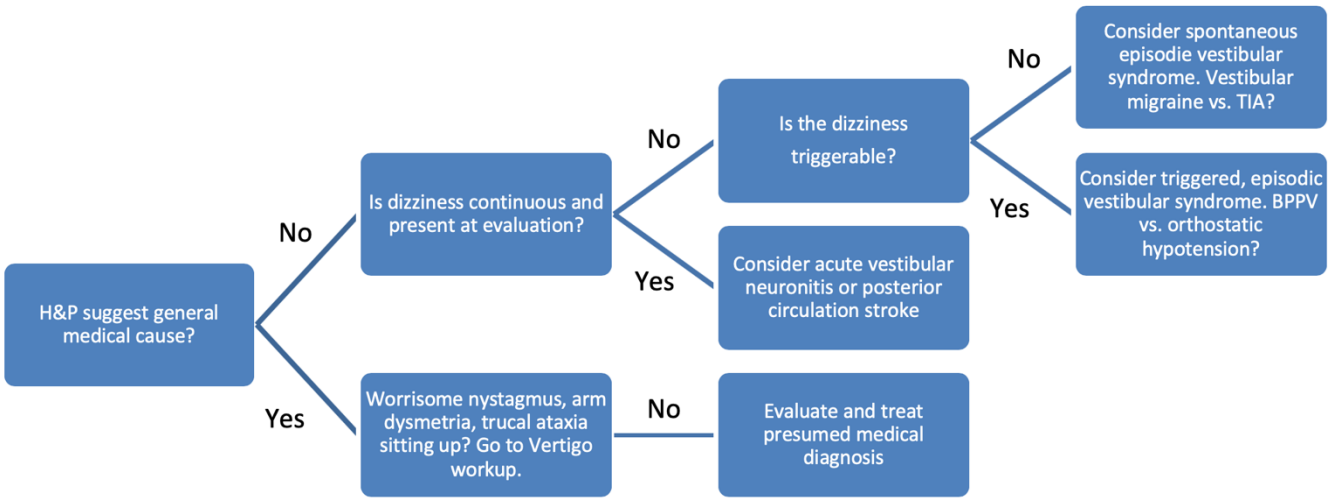
Provocative Testing:

Dix-Hallpike: Nystagmus regardless of position.
Pronator Drift: Problems when the patient tries to stand and loses balance. Test is non-diagnostic.
Tandem Gait: Wide-based gait with difficulty walking in a straight line.
Romberg: Inability to maintain balance even with eyes open.
Finger to Nose/Heel to shin: Mild right sided abnormality compared to the left.
Rapid Alternating Movements: No appreciable difference between sides.
Nystagmus: Nystagmus is horizontal and bidirectional.
Head Impulse Test: Normal.
Test of Skew: Skew deviation of the right eye.

Question Prompts:

1. What specific questions and verbiage can you use to distinguish between true vertigo and other non-specific descriptions of dizziness?

a. Pre-syncope: The feeling of passing out. "Lightheadedness, "going to pass out," "swimmy-headed," "stood up too fast" are all used to describe this feeling.
b. True balance problems: Often described as falling to one side or not being able to coordinate movements. This can be a true ataxia representing posterior fossa disease.
c. Vertigo: The sensation of spinning.
d. Nonspecific vague complaints:
    i. In a 2007 study, ED patients with dizziness were asked a series of questions aimed at determining the reliability and consistency of eliciting "symptom quality" and timing and triggers of their dizziness. When the "symptom quality" question was re-asked an average of 6 min later, half of the patients changed their primary dizziness type.[2]
    ii. More than 60% of the patients with dizziness endorse more than one dizziness type.
e. Alternative approach: ATTEST
    i. Associated symptoms, timing and triggers, bedside examination signs, and additional testing.



Adapted from Figure 1. Diagnostic approach to the acutely dizzy patient. In: Edlow JA. A new approach to the diagnosis of acute dizziness in adult patients. *Emergency Medicine Clinics of North America.* 2016 Nov; 34(4):717-742.

2. How does *acute vestibular syndrome* differ from *spontaneous episodic* vestibular syndrome? Contrast this with a *triggered* episodic vestibular syndrome. What are the causes of each?
    a. Acute vestibular syndrome (AVS): Acute onset of *persistent* dizziness associated with nausea or vomiting, gait instability, nystagmus, and head motion intolerance lasting days to weeks.
        i. Symptoms are exacerbated (dizzy at baseline, worse with movement) vs. triggered (not dizzy at baseline, dizziness develops with movement).
        ii. Causes include vestibular neuritis (dizziness only) or labyrinthitis (dizziness plus hearing loss or tinnitus), posterior circulation ischemic stroke, multiple sclerosis, cerebellar hemorrhage, thiamine deficiency and other various autoimmune, infectious or metabolic conditions.

b. Triggered episodic vestibular syndrome (t-EVS): Brief episodes of dizziness lasting seconds to minutes with an "obligate" trigger that consistently causes dizziness. Causes include benign paroxysmal positional vertigo (BPPV) and orthostatic hypotension.
c. Spontaneous episodic vestibular syndrome (s-EVS): Recurrent, spontaneous episodes of dizziness that range in duration from seconds to days, the majority lasting minutes to hours. The dizziness cannot be triggered at the bedside by head movements.
    i. If patients are still symptomatic at presentation, use the ATTEST approach. If asymptomatic at presentation, the evaluation usually relies entirely on the history.
    ii. Causes include panic attacks, hypoglycemia, cardiac arrhythmias, pheochromocytoma, vestibular migraine, and posterior circulation transient ischemic attack (TIA).
        1. Isolated spontaneous dizziness is the most common presentation of vertebrobasilar TIA.
        2. In transient symptoms preceding vertebrobasilar stroke, 9 of 10 who sought medical attention are initially missed.[3]

Syndrome	Description	Common Causes
AVS	Rapid onset of *persistent* acute dizziness that lasts days, often associated with nausea, vomiting, and head motion intolerance	Benign: vestibular neuritis and labyrinthitis Serious: cerebellar stroke
t-EVS	Episodic dizzy episodes triggered [b]by some specific obligate event, usually head movement or standing up and usually last <1 min	Benign: Benign paroxysmal positional vertigo (BPPV) Serious: serious causes of orthostatic hypotension and positional vertigo
s-EVS	Episodic dizzy episodes that occur spontaneously, are not triggered, and usually last minutes to hours	Benign: vestibular migraine, Meniere disease Serious: TIA
Chronic vestibular syndrome (CVS)	Chronic dizziness lasting weeks to months (or longer)	Benign: medication side effects, anxiety and depression Serious: posterior fossa mass

3. What are the components of the HINTS exam and how is it used?
    a. HINTS stand for head-impulse, nystagmus, test-of-skew. https://www.youtube.com/watch?v=84waYROlI4U.
    b. Nystagmus:
        i. Have the patient look straight ahead and observe for eye movements.
        ii. The direction of nystagmus is named by the direction of the fast component.

iii. Look for "gaze-evoked" nystagmus by having the patient look right and left. Observe for the presence of nystagmus and not the direction of the fast movement.
    1. Horizontal nystagmus on extreme lateral gaze that is very low amplitude, extinguishes quickly, beats in the direction of gaze and is symmetric to the two sides is a normal finding in many individuals.
iv. Two patterns suggest stroke:
    1. Dominantly vertical or torsional nystagmus in any gaze position.
    2. Bidirectional, gaze-evoked nystagmus - dominantly horizontal nystagmus where the fast beat of the nystagmus **changes direction** in different gaze positions.
    3. If one of these are observed, presume vertebrobasilar stroke as cause. In other words, nystagmus for peripheral vertigo (benign) should be **horizontal** and **not change** direction.
v. However, the most common pattern in stroke is direction-fixed horizontal nystagmus, the same as acute vestibular neuritis. Which is why the remainder of HINTS is required.

c. Skew deviation: vertical misalignment of the eyes due to imbalance in gravity-sensing vestibular pathways.
    i. Elicited using the "alternate cover" test.
        1. The patient looks directly at the examiner's nose or eyes and the physician covers one eye, then the other, and continues alternating back and forth, roughly every 1-2 s. The amplitude of correction is small (1–2 mm) and appears at the moment the eye is uncovered.
        2. A skew deviation is present if a slight vertical correction occurs (one side corrects upward and the other corrects downward);
        3. If no vertical movement occurs, there is no skew. ***NOTE: horizontal movements do not count***.

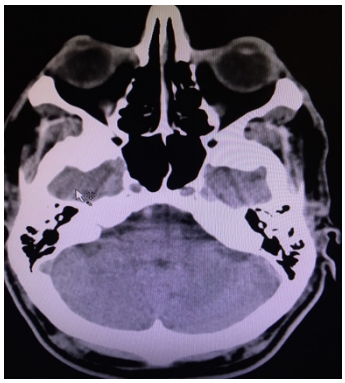
d. Head Impulse Test: A test of the vestibulo-ocular reflex (VOR). This should only be used in patients in AVS with nystagmus. A head impulse test done in a patient without nystagmus will be normal and, therefore, misleading.
    i. The patient looks directly at the examiner's nose while the examiner holds the patient's head in his or her hands. It is important to keep the patient chin down, so as not to rapidly move the neck while extended, theoretically putting the patient at risk for vestibular artery injury during manipulations with the head in extension (looking up). The physician displaces the patient's head about 10–20 degrees from the midline to one side. The examiner quickly moves head back toward the center position and stops at the midline.
    ii. In a normal response, the patient's gaze remains locked on the examiner's nose.
    iii. A corrective saccade occurs when the gaze does not remain locked on the examiner's nose, but instead follows the rotation of the head and then quickly saccades back to the examiner's nose once the head rotation is complete. The

presence of a corrective saccade is a "positive" test, which generally indicates a peripheral process, usually vestibular neuritis.

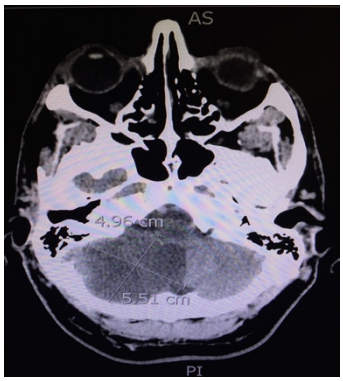
iv. The absence of a corrective saccade on ***both sides*** suggests a possible stroke in AVS.

e. For the HINTs exam to support peripheral vertigo, the patient must have ALL three findings – reassuring nystagmus (horizontal, unidirectional), negative test of skew, and positive head impulse testing. If patient does not have all testing reassuring and risk factors for a central process, advanced imaging should be strongly considered.

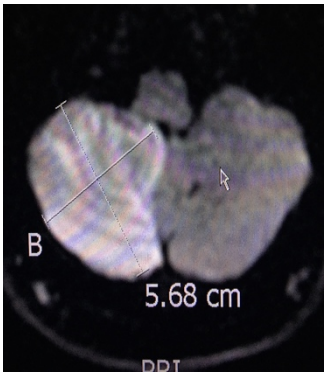
4. What is the Dix-Hallpike maneuver and how is it used? What about the supine roll test?
   a. Dix-Hallpike: Patients are lowered quickly to a supine position with the neck extended by the clinician and head is rotated to one side.
      i. The test is positive when the patient experiences reproduction of vertigo, and the clinician observes nystagmus.
      ii. The test is repeated on both sides.
      iii. Benign paroxysmal positional vertigo is suggested when the test demonstrates unilateral, fatigable nystagmus.
         1. If BPPV is suggested by a positive Dix-Hallpike maneuver, the Epley maneuver can be performed as a therapeutic intervention. This process starts in the ending position of the Dix-Hallpike with patient laying on the back with head extended and rotated to the symptomatic side. Then after symptoms resolve, the provider rotates the head 90° to other side. Many times, patients will re-experience symptoms. Wait again a few minutes for symptoms to resolve. Then continue to rotate the head 90 in the same direction, so patient is now looking towards the floor. Again, wait a few minutes for symptoms to resolve. Then rotate the head more while having the patient sit up. This can be repeated multiple times to provide additional relief.
   b. Supine roll test: A test for lateral canal BPPV in the setting of a negative Dix-Hallpike
      i. The patient is positioned supine with the head in a neutral position.
      ii. The head is quickly rotated 90° to one side with the clinician observing the eyes for nystagmus. After any elicited nystagmus has resided, the head is returned to neutral and the eyes are once again observed for nystagmus.
      iii. A positive supine roll test can evoke two different patterns of nystagmus, geotropic and apogeotropic, which reflect the two different types of lateral canal BPPV.
         1. Geotropic nystagmus refers to a nystagmus pattern where the fast beat is directed towards the ground. Apogeotropic nystagmus is where the fast beat is directed to the sky. Both are forms of vertical nystagmus.
5. The patient has the below findings on computed tomography (CT) imaging. What is the clinical utility of CT and magnetic resonance imaging (MRI) in acute vestibular syndrome? What are the sensitivities of each for posterior circulation stroke?



Initial CT at time 2 hours post onset



Repeat CT 24 hours later



Initial MRI at time 2.5 hours post onset

Author's own images.

a. The overall sensitivity of CT for acute posterior ischemic stroke is approximately 26%.
b. MRI will miss 19%-27% of strokes presenting with an acute vestibular syndrome during the first 12 hours after onset.
c. Delayed MRI (>12-24 hours days post symptom onset) may be required to confirm the presence of new infarcts. MRI more than 12 hours after onset of symptoms has a sensitivity of 92% for acute ischemic stroke.[4]

6. Who can you send home with no additional testing and who needs an MRI/neurology consult?
    a. Two types of patients can be discharged from the ED with no additional testing:
        i. Patients who have triggered episodic vestibular syndrome that have a Dix-Hallpike or supine roll test demonstrating fatigable unidirectional nystagmus.
        ii. Patients with a reassuring history and HINTS exam consistent with a peripheral process (positive unidirectional horizontal nystagmus, positive head impulse test, negative test of skew).
    b. Patients that need MRI:
        i. Have continuous acute vestibular syndrome with a HINTS exam concerning for a central process (any deviation from above. This includes lacking nystagmus).
        ii. Dizziness with or without vertigo and additional neurologic deficit.
        iii. Have spontaneous episodic vestibular syndrome concerning for TIA.

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Appendix G:
Infectious and Inflammatory Neurologic Disorders

Objectives

1. Discuss the clinical historical features and exam findings suggestive of meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess.
2. Discuss the emergency department workup for viral and bacterial meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess.
3. Identify the appropriate initial treatments for viral and bacterial meningitis, encephalitis, transverse myelitis, spinal epidural abscess and intracranial abscess.

Case Studies

Case 1: A 27-year-old incarcerated male presents with seizures. Per the corrections officers, he has no known medical illnesses and had a witnessed generalized tonic-clonic seizure lasting one to two minutes less than one hour prior to arrival. The officers note he has visited the infirmary several times in the last week for headaches. The patient is unable to provide additional history due to altered mental status.

Vitals: Temperature (T) 102.1°F, heart rate (HR) 113, respiratory rate (RR) 22, blood pressure (BP) 120/70, oxygen saturation (O_2sat) 99% on room air.

Physical Exam:
General: Somnolent and difficult to arouse. Disoriented.
Chest: Tachypnea, lungs clear to auscultation bilaterally. Heart sounds normal.
Abdomen: Soft, nontender and nondistended.
Neurologic: Opens eyes to noxious stimulus, localizes pain but does not follow commands, incomprehensible speech, moving all four extremities spontaneously, pupils are reactive to light. Uncomfortable appearing and groans with head movements, but Brudzinski and Kernig signs negative.
Skin: No rash.

Question Prompts:

1. What clinical features distinguish encephalitis from meningitis? What is meningoencephalitis?
    a. The presence of an acute change in brain function distinguishes encephalitis from meningitis
        i. With meningitis, patients may be uncomfortable, lethargic, or distracted by headache, but their cerebral function remains at baseline. Seizures and subsequent postictal altered mental status can be seen with meningitis. Thus, patients who

return to baseline mental status eventually after a seizure are less likely to have encephalitis.

ii. In encephalitis, abnormalities in brain function are present, and may present as confusion, motor or sensory deficits, altered behavior, personality changes, speech or movement disorders, hemiparesis, flaccid paralysis, and paresthesia.

b. Meningoencephalitis is also a common term that recognizes the overlap between the two entities because frequently patients can be presented with both parenchymal and meningeal processes with clinical features of both.

2. What exam findings are consistent with meningitis? How frequently are these findings seen in cases of bacterial meningitis?
    a. The typical symptoms of acute bacterial meningitis consist of fever, nuchal rigidity, headache, and altered mentation.
        i. Fever is present in 77%%-95% of patients at presentation.
        ii. Upwards of 99% will demonstrate fever within 24 hours.
        iii. Nuchal rigidity is present in 83%-94% of patients on presentation.
            1. The Brudzinski sign refers to spontaneous flexion of the hips during attempted passive flexion of the neck. Sensitivity 2% and specificity 98%.
            2. The Kernig sign refers to the inability or reluctance to allow full extension of the knee when the hip is flexed 90 degrees. Sensitivity 2% and specificity 97%.
            3. The Jolt accentuation of headache test consists of accentuation of headache by horizontal rotation of the head at a frequency of two to three times per second. Sensitivity 97%-100% and specificity 60%.
        iv. Change in mental status (acute delirium) is present in 78%-94% at presentation.
        v. Headache is present in 79%-94% at presentation.
    b. Almost all patients (99%-100%) will have at least one of these symptoms and 95% present with at least two of four symptoms (ie, headache, fever, stiff neck, and altered mental status).
    c. Other common symptoms include seizure, rash and arthritis.
        i. Seizures have been described in 15 to 30 percent of patients and focal neurologic deficits in 10 to 35 percent.
        ii. *N. meningitidis* can cause characteristic skin manifestations, such as petechiae and palpable purpura.
            1. Palpable purpura or petechiae are present in 11 and 26 percent of bacterial meningitis cases, respectively. 75%-92% are associated with meningococcal meningitis.
        iii. Arthritis is seen in 7% of meningitis cases; *N. meningitidis* is the etiologic agent in two-thirds of these joint infections.
            1. Cultures from joint aspiration are positive in 23% of cases.
3. What are the indications for head computed tomography (CT) prior to lumbar puncture (LP)?
    a. Immunocompromised state [*eg,* human immunodeficiency virus (HIV) infection, immunosuppressive therapy, solid organ or hematopoietic stem cell transplantation].

b. History of central nervous system (CNS) disease (mass lesion, stroke, or focal infection).
c. New onset seizure.
d. Papilledema.
e. Abnormal level of consciousness.
f. Focal neurologic deficit.

4. What workup should be performed on this patient? What LP findings are suggestive of bacterial meningitis? Viral meningitis? Fungal Meningitis?
    a. Complete blood count (CBC), basic metabolic panel (BMP), blood cultures, urinalysis (UA), CT head, chest X-ray (CXR) (50% of patients with pneumococcal meningitis have evidence of pneumonia on CXR), LP.
    b. See chart below:

	Appearance	Opening Pressure (cm H_2O)	White Blood Cells (WBC/mcl)	Percent polymorpho-nuclear neutrophils (PMNs)	Glucose	Protein (mg/dl)	Gram Stain
Normal	Clear	10-20	0-5	n/a	>60% Serum glucose	<45	Negative
Bacterial	Clear, Cloudy or purulent	>25	>100	80%-90%	Low	Elevated	Positive
Viral	Clear	Variable	5-1000	1%-50%	Normal	Elevated	Negative
Fungal	Clear or opaque	>25	<500	1%-50%	Low	Elevated	India Ink

Adapted from lumbar puncture diagnosis. In: Badawy A, Fields A, Eggeman D, et al. Meningitis. WikiEM. https://wikem.org/wiki/Meningitis. Updated February 22, 2018. Accessed May 25, 2019.

5. What is the treatment for bacterial meningitis? Viral meningitis? Cryptococcosis? What are the indications and treatment regimens for prophylaxis?
    a. Bacterial
        i. Neonates (up to 1 month of age):
            1. Ampicillin 50mg/kg intravenous (IV) q6 hours +.
            2. Cefotaxime 50mg/kg IV q6 hours OR gentamicin 2.5mg/kg IV q8hrs.
            3. If suspecting S. pneumoniae or methicillin-resistant *Staphylococcus aureus* (MRSA), add vancomycin.
            4. Consider acyclovir for herpes simplex virus (HSV), especially if mother is HSV positive.

ii. Infant > 1 month and Adults < 50 years old:
    1. Ceftriaxone 2gm (50mg/kg) IV twice daily (BID) daily +. Vancomycin 15-20 mg/kg IV BID daily.
iii. Adult > 50 years old or immunocompromised:
    1. Ceftriaxone 2gm (50mg/kg) IV BID daily + Vancomycin 15-20 mg/kg IV BID daily + Ampicillin 2gm IV q4h (hourly if listeria suspected).
iv. Post-procedure or penetrating trauma:
    1. Vancomycin 15-20mg/kg IV BID daily + Cefepime 2g (50mg/kg) IV q8 hours daily OR ceftazidime 2g (50mg/kg) IV q8 hours daily OR meropenem 2gm (40mg/kg) IV q8 hours daily.
v. Severe PNC allergy:
    1. Chloramphenicol 1g IV q6 hours + vancomycin 15mg/kg q8-12 hours.
vi. Ventriculoperitoneal (VP) shunt: Coverage for skin contaminants (*S. epidermis*, *S. aureus*).
    1. Vancomycin + ceftriaxone + shunt removal.
vii. Steroids:
    1. Dexamethasone in adults:
        a. Only give 15 minutes prior to or with first dose of antibiotics.
        b. 10mg IV q6 hours x4 days.
    2. Dexamethasone in children and infants:
        a. There has been no mortality benefit found with steroid use in children.
        b. Neurologic sequelae were reduced only in high income countries.
    3. Hydrocortisone for adrenal failure:

    Waterhouse–Friderichsen syndrome, bilateral adrenal hemorrhage causing adrenal failure – seen in meningococcemia.

b. Cryptococcosis
    i. Amphotericin B 1-5 mg/kg IV once daily and 5-fluorocytosine (5-FC) 25mg/kg orally (PO) q6 hours daily.
c. Viral
    i. Supportive care.
    ii. Admit for empiric antibiotics until culture results return vs. discharge with 24hr follow up.
    iii. Consider acyclovir for patients with suspected viral meningitis who present with neurologic deficits.
        1. 10mg/kg IV q8 hour (obese patients should be dosed using ideal body weight).
d. Prophylaxis
    i. Indications for prophylaxis:
        1. Household contacts.
        2. School or day care contacts in previous seven days.
        3. Direct exposure to patient secretions (kissing, shared utensils or toothbrush).

4. Intubation without facemask.

ii. Prophylaxis regimen
1. Meningococcus exposure:
a. Rifampin 600mg PO BID x 2 days.
i. 5mg/kg PO if < 1 month old.
ii. 10mg/kg PO ≥ 1 month old.
b. Ceftriaxone 250mg IM once.
i. 125mg IM if ≤ 15 years old.
ii. Ceftriaxone should be used for pregnant patients.
c. Ciprofloxacin 500mg PO once.

6. The non-contrast CT head demonstrates a "focal area of hypodensity." How does this change the likely diagnosis, emergency department (ED) workup, and treatment? What are the possible etiologies of this condition?
a. A focal area of hypodensity on non-contrast CT head in a patient with fever, seizure and encephalopathy should be concerning for an intracranial abscess.
i. The triad of headache, fever, AND focal neuro deficit is present in <33%.
1. Headache is the most common symptom (present in almost all cases).
2. Fever (50% of patients).
3. Focal neuro symptoms or seizure (~3% of patients).
4. Neck stiffness (<50% of patients).
5. Signs of increased intracranial pressure (ICP): Papilledema, vomiting, confusion, obtundation (50% of patients).
ii. Intracranial abscess is caused by one of three methods:
1. Hematogenous spread (33%).
2. Contiguous infection from middle ear, sinus, teeth (33%).
3. Direct implantation by surgery or penetrating trauma (10%).
a. Microbiology:
i. *Streptococcus* in 50% of cases.
ii. Anaerobes and gram-negative rods are other typical pathogens.
iii. *Staphylococcus* is nearly always involved with direct implantation cases.
b. The patient should undergo additional imaging, either contrast enhanced CT head or magnetic resonance imaging (MRI) brain.
i. Findings would demonstrate a ring-enhancing lesion surrounding low-density center surrounded by white matter edema.
c. Lumbar puncture is contraindicated in confirmed or high suspicion cases of brain abscesses.
i. Results are often variable. If results are consistent with bacterial meningitis, this usually indicates an abscess rupture into the ventricle system. In patients with meningitis who respond poorly to antibiotics, MRI should be performed for evaluation of abscess.

d. Neurosurgery consult, blood cultures, and admission for IV antibiotics are important in early treatment of brain abscesses. Antibiotics include:
    i. Otogenic, sinogenic or odontogenic source:
        1. Cefotaxime 2gm IV q6 hours + metronidazole 500mg IV q6hr.
    ii. Penetrating trauma or neurosurgical procedures:
        1. Vancomycin 15mg/kg IV q12hr + ceftazidime 2gm IV q8 hours
    iii. Hematogenous source or no obvious source:
        1. Cefotaxime 2gm IV q6 hours + metronidazole 500mg IV q6 hours.

Case 2: A 27-year-old female presents to the emergency department with weakness. She reports approximately three days of progressively worsening symmetric bilateral lower extremity weakness. Over that time, she has also been experiencing midline thoracic back pain. She is now unable to walk. She also reports subjective fevers, diminished sensation to light touch in the lower extremities and urinary incontinence. Per her medical record, she has a history of IV drug use but she denies continued use. On review of systems, she has had recent viral upper respiratory infection symptoms including cough, sore throat and rhinorrhea, which resolved weeks ago.

Vital Signs: T 99.9°F, HR 103, RR 20, BP 120/70, O_2sat 99% on room air.

Physical Exam:
General: Alert, uncomfortable appearing but not in distress.
Chest: Regular tachycardia, 3/6 systolic murmur, reports murmur present since childhood. Mild tachypnea, lungs clear to auscultation bilaterally.
Abdomen: Soft and non-tender.
Neuro: Cranial nerves (CN) 2-12 intact, upper extremity strength and sensation intact, diminished sensation to light touch below the level of the nipple line, 3/5 strength of hip, knee and ankle flexion/extension, absent patellar and Achilles reflexes. Post-void residual bladder volume is 600 ml.

Question Prompts:

1. What is the differential diagnosis in this case?
    a. Transverse myelitis.
    b. Compression myelopathy: Disc herniations, epidural masses, abscesses or hematomas, vertebral body compression fractures, and spondylosis.
    c. Vascular myelopathies: anterior spinal artery infarction, spinal-dural arteriovenous fistula, fibrocartilaginous embolism.
    d. Metabolic, toxic, and nutritional myelopathies: vitamin B12 deficiency, vitamin D deficiency, vitamin E deficiency, copper deficiency, nitrous oxide toxicity, neurolathyrism and neurocassavism.
    e. Neoplasm: Intramedullary primary spinal cord tumor, primary central nervous system lymphoma, intravascular lymphoma, metastatic lesion.
    f. Radiation myelitis.

   g. Demyelinating disorders (multiple sclerosis, Guillain-Barré syndrome).
2. What is the ED workup in this patient?
   a. Basic labs, inflammatory markers, erythrocyte sedimentation rate (ESR). The sensitivity of an elevated ESR >100 in patients with epidural abscess risk factors approaches 100%.
   b. When myelopathy is suspected, emergent spinal imaging is warranted to exclude a compressive etiology.
      i. An MRI of the spine is the preferred diagnostic study.
      ii. Computed tomography myelogram can be used if an MRI cannot be obtained immediately.
   c. Brain MRI with and without contrast to evaluate for the presence of brain lesions suggestive of multiple sclerosis
   d. In the absence of compression findings or contrast enhancement on MRI, cerebrospinal fluid analysis should be obtained, including cell count and differential, protein, glucose, venereal disease research laboratory (VDRL) test, oligoclonal bands, immunoglobulin G index, and cytology.
3. If the MRI findings are concerning for epidural abscess, what are next steps in management?
   a. Blood cultures for identification of source organism.
   b. Neurosurgery consult for early surgical decompression and drainage.
   c. Antibiotics targeted for *Staphylococcus*, *Streptococcus*, and gram-negative bacilli.
      i. Vancomycin 15-20mg/kg BID + metronidazole 500g (7.5mg/kg) q6 hours + cefotaxime (2 g IV every six hours) or ceftriaxone (2 g IV every 12 hours) or cefepime (2 g IV every 8 hours) or ceftazidime (2 g IV every 8 hours). Cefepime or ceftazidime is preferable when Pseudomonas aeruginosa is considered a possible or likely pathogen.
4. In the absence compressive findings on MRI, what are the diagnostic criteria for transverse myelitis?
   a. Sensory, motor, or autonomic dysfunction attributable to the spinal cord.
   b. Bilateral signs and/or symptoms.
   c. Clearly defined sensory level.
   d. No evidence of compressive cord lesion.
   e. Inflammation defined by cerebrospinal fluid pleocytosis, elevated immunoglobulin G (IgG) index, or gadolinium enhancement on MRI.
   f. Progression to nadir between four hours and 21 days.
5. What is the treatment of transverse myelitis?
   a. High dose steroids are mainstay of treatment.
      i. Methylprednisolone 1000 mg daily or dexamethasone 200 mg daily for three to five days.
   b. In patients with acute transverse myelitis complicated by motor impairment, treatment with plasma exchange is also indicated.

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Appendix H:
Demyelinating Disorders, Neuromuscular Disorders and Movement Disorders

Objectives

1. Discuss the underlying pathophysiology, distinguishing clinical features, diagnostic workup, treatment and expected complications of common demyelinating disorders: multiple sclerosis (MS), and Guillain-Barré syndrome (GBS).
2. Distinguish clinical presentation and management of myasthenia gravis (MG) from demyelinating disorders.
3. Discuss the underlying pathophysiology, distinguishing clinical features, and diagnostic workup of Parkinson's disease.
4. Discuss diagnosis and management of extrapyramidal symptoms and akathisia.

Case Studies

Case 1: A 27-year-old female with no significant medical history presents with complaint of weakness. Symptoms have been present for the past three days. She describes initial onset of bilateral leg tingling and weakness that has progressed since onset. She awoke today and had difficulty walking so presented to the emergency department (ED). She has never had these symptoms before. She recovered from a severe diarrheal illness 2 weeks ago but denies any current gastrointestinal symptoms.

Vital signs: Temperature (T) 99.0°F, heart rate (HR) 92, blood pressure (BP) 106/78, respiratory rate (RR) 18, oxygen saturation O_2sat 97% on room air.

Physical exam: The patient appears fatigued but is not in any distress. Heart is regular. Breath sounds slightly diminished but otherwise clear. Lower extremity strength is 3/5 bilaterally and patellar reflexes are absent.

Question Prompts:

1. What is the underlying pathophysiologic mechanism of the most likely underlying diagnosis?
    a. Guillain-Barré syndrome (GBS) is a heterogeneous group of acute immune-mediated polyneuropathies.
    b. It is thought to result from an immune response to a preceding infection that becomes directed toward myelin of peripheral nerves resulting in demyelination.
2. What are the distinguishing clinical features of Guillain-Barré syndrome?

   a. Clinical diagnosis of GBS is made in the presence of progressive, symmetric muscle weakness and absent or depressed deep tendon reflexes. Weakness usually (90%) starts in the legs and is often described as ascending.
   b. Deep tendon reflexes are depressed or absent in 90% of patients at presentation.
   c. Facial nerve palsies, oropharyngeal weakness and occulomotor weakness are not uncommon. Paresthesias accompanying weakness occur in 80% of cases. Dysautonomia may occur in 70% of patients.
3. What diagnostic testing can be used to support the diagnosis of Guillain-Barré syndrome?
   a. Guillain-Barré syndrome is a clinical diagnosis, but can be supported by cerebral spinal fluid (CSF) findings.
      i. Albuminocytologic dissociation - Normal to mildly elevated white blood cell (WBC) count, usually with lymphocytic predominance with disproportionately elevated protein.
   b. Nerve conduction studies and electromyelography are confirmatory.
4. What treatments are available for Guillain-Barré syndrome?
   a. Guillain-Barré syndrome treatment is primarily supportive, and addresses complications.
      i. Respiratory failure, autonomic dysfunction, arrhythmias, electrolyte abnormalities, etc.
      ii. Intravenous immunoglobulin (IVIG) or plasmapheresis is indicated in non-ambulatory patients within four weeks of onset of symptoms and in ambulatory patients who are not improving within four weeks of symptoms.
5. What diagnostic evaluation should be pursued in this case?
   a. Diagnostic studies should include basic labs and electrolytes, electrocardiogram (ECG) for arrhythmia and neurology consultation.
   b. Neuroimaging and lumbar puncture may be used to exclude alternative diagnosis, including MS and spinal cord compression/cauda equina.
   c. Respiratory parameters, maximal inspiratory pressure (MIP) also known as negative inspiratory force (NIF) may be helpful in determining potential for respiratory decompensation and need for intubation.
      i. Normal MIP/NIF is greater than 60cm of water.
      ii. If MIP/NIF is dropping or less than 30cm of water, consider respiratory support with positive pressure ventilation and likely intubation.[5]
      iii. Use these values in the context of the clinical situation. There are no hard cut offs that demand intervention; instead they should be used to predict impending respiratory failure.
6. Describe potential complications associated with this patient's diagnosis.
   a. Respiratory failure and need for ventilation, autonomic instability, arrhythmia, electrolyte abnormalities (especially hyponatremia) and pain.
7. Discuss treatment options and appropriate disposition for this patient.
   a. Intravenous immunoglobulin or plasmapheresis in conjunction with consultants as indicated and supportive care.
   b. Disposition is admission due to inability to ambulate and progressive symptoms.

c. Intensive care unit (ICU) if evidence of airway or respiratory compromise or significant autonomic dysfunction.

Case 2: A 39-year-old female with no significant medical history presents with complaint of weakness. Symptoms have been present for the past three weeks. She describes difficulty climbing stairs and frequently has to take breaks before continuing. She reports that over the past 24 hours, she has been experiencing double vision when watching television and difficulty chewing.

Vital signs: T 98.8°F, HR 72, BP 136/78, RR 22, O_2sat 97% on room air.

Physical Exam: The patient appears fatigued but is not in any distress. Breath sounds slightly diminished but otherwise clear. Upper and lower extremity strength is 3/5 bilaterally and weakness seems to worsen as the exam continues. Reflexes and sensation are normal. She has left eye ptosis and a deconjugate gaze.

Question Prompts:

1. What is the underlying pathophysiologic mechanism of the most likely diagnosis.
    a. Myasthenia gravis (MG) is an autoimmune disease due to acetylcholine receptor antibodies impairing muscle function at the neuromuscular junction.
2. What are the distinguishing clinical features of myasthenia gravis?
    a. Clinical diagnosis of MG is suggested in patients with ocular disturbances and/or proximal limb weakness not associated with other systemic causes of weakness.
    b. Most patients have generalized weakness, especially of the proximal muscle groups, neck extensors, and facial muscles without changes in sensation, reflexes, or cerebellar functioning.
    c. Symptoms worsen with prolonged use and improve with rest. Emergency department presentation usually consists of worsening weakness +/- respiratory failure due to infection, recent surgery or rapid tapering of immunosuppression.
    d. Myasthenia crisis occurs due to extreme weakness in respiratory muscles and can be seen in undiagnosed patients or those with inadequate drug therapy or drug tolerance.
3. What diagnostic testing can be used to support the diagnosis of myasthenia gravis?
    a. Myasthenia gravis is diagnosed with edrophonium administration resulting in improvement of symptoms. Ice pack test can be used in the absence of edrophonium.
    b. Electromyelography and acetylcholine receptor (AChR) antibody testing are also supportive.
4. What treatments are available for myasthenia gravis?
    a. Myasthenia gravis is treated with acetylcholinesterase inhibitors (pyridostigmine or neostigmine), thymectomy, and chronic immune suppression.
    b. Acutely, patients may need plasma exchange or IVIG as well as supportive ventilation. If intubation is required, patients will be resistant to depolarizing paralytics and sensitive to nondepolarizing paralytics. Some suggested dosing is to half the dose of rocuronium 0.6mg/kg. Some recommend against the use of succinylcholine.

Case 3: A 35-year-old female presents with multiple complaints. She reports heat intolerance, a stumbling gait and a tendency to fall. These symptoms have been present for several years. Her visual acuity also seemed to change periodically during this period. Several months ago, she experienced a viral upper respiratory infection, but also experienced decreased grip strength, significant tremors and severe exhaustion. She is here today because over the last 24 hours, she abruptly developed right upper and lower extremity numbness.

Vital signs: T 98.4°F, HR 87, BP 122/71, RR 14, O_2sat 97%.

Physical Exam: She is noted to have a left eye relative afferent pupillary defect (RAPD) with an otherwise intact cranial nerve exam. Sensory exam reveals paresthesia on the right to touch and decreased pin sensation on the right diffusely. Upper extremity reflexes 2+, lower extremity reflexes 3+. Babinki positive bilateral. Upper extremity strength 5/5, lower extremity strength 4/5. Rapid alternating movements are difficult in both upper extremities and the patient has dysdiadochokinesia in the left hand.

Question Prompts:

1. What are the underlying pathophysiologic mechanisms of the most likely diagnosis?
    a. This presentation would have a broad differential given the multiple neurologic complaints. But multiple sclerosis (MS) should be strongly considered.
    b. Multiple sclerosis is the result of central nervous system (CNS) myelin destruction with variable motor, sensory, visual, and cerebellar dysfunction. The exact cause is unknown but it causes inflammation leading to neuron demylination.
2. What are the distinguishing clinical features of multiple sclerosis?
    a. Common symptoms of MS include parasthesias, gait difficulty, extremity weakness, poor coordination, and visual disturbances. MS is often characterized by discrete symptomatic episodes “separated in time and space.”
    b. On further history, patients may often recall history of previous episodes of focal neurologic deficits that may have self-resolved and do not seem to follow a pattern consistent with a single CNS lesion.
    c. Optic neuritis may be the initial presenting sign in up to 30% of people with MS and causes an acute or subacute central vision loss.
    d. Physical exam findings may include decreased strength, increased tone, hyperreflexia, clonus, positive Babinski, decreased vibratory sense, proprioception, and pain and temperature sensation. Multiple sclerosis often has a relapsing/remitting course (~90%) vs. relapsing/progressive or chronically progressive.
3. What diagnostic testing can be used to support the diagnosis of multiple sclerosis?
    a. Multiple sclerosis is diagnosed by suggestive history and clinical features.
    b. Magnetic resonance imaging demonstrating dissemination of CNS lesions in time and space – in other words having lesions in different locations of the central nervous system that occur at different times. A T2 weighted MRI cannot distinguish between acute and chronic lesions, but a single gadolinium-enhanced MRI can potentially provide evidence for dissemination in space and time.

   c. Cerebral spinal fluid analysis with oligoclonal bands can be supportive of the diagnosis.
4. What treatments are available for multiple sclerosis?
   a. Multiple sclerosis treatment includes steroids, immune suppression and immunomodulation (interferon beta-1a, glatiramer, natalizumab, and mitoxantrone).

Case 4: A 64-year-old male with history of Parkinson's disease is brought to the emergency department by his family due to agitation. Over the last three days, the patient has felt restless and has had odd, involuntary movements of his limbs and face. Per family, he gets in and out of bed frequently because he cannot get comfortable and he has had multiple falls as a result. He denies any head injury, weakness or pain to the neck or back. He denies headaches or vision changes. He has had no fevers or urinary symptoms. His only medications are levadopa-carbidopa and bromocriptine.

Vital signs: T 98.7°F, HR 72, BP 124/71, RR 16, O_2sat 99%.

Physical Exam: He answers questions and is oriented appropriately while writhing in bed, but is not cooperative with additional exam due to agitation.

Question Prompts:

1. What is the underlying pathophysiology of Parkinson's disease?
   a. Parkinson's disease is an extrapyramidal movement disorder associated with decreased number of functional dopaminergic receptors in the substantia nigra.
2. What are some of the common features of Parkinson's disease? How is it diagnosed?
   a. Patients will have one or more of the following: Resting tremor, cogwheel rigidity, bradykinesias or akinesias, or impaired posture/equilibrium. Patients may also have facial and voice changes, depression, impulse control difficulties, and muscle fatigue.
   b. Tremor often improves with purposeful movement and returns with rest. Typically diagnosed clinically based on symptoms. However, Parkinsonism can also occur due to drugs, toxins, neuroleptic drugs, trauma, infections and other neurologic disorders.
3. What is the treatment for Parkinson's disease?
   a. Treatment is symptomatic and includes anticholingeric agents like trihexyphenidyl and benzatropine, drugs that increase central dopamine levels like amantadine, levodopa/carbidopa, and dopamine receptor agonists like bromocriptine and pergolide.
4. What are the side effects of treatment? What is the management of these side effects? What medications can cause similar side effects?
   a. Dopamine therapy toxicities include cardiac dysrhythmias, orthostatic hypotension, dyskinesias (involuntary movements) and dystonias (painful, prolonged muscle contractions), and akathisia (inner restlessness).
   b. Management of dyskinesia often begins with adjusting the levodopa regimen and/or the use of adjunctive medications including dopamine agonists. Amantadine and clonidine can also be useful for suppressing dyskinesia.

c. This patient is likely suffereing from akathisia. In Parkinson's disease, this can be managed with beta-blockers or clonidine. Benzodiazepines are second-line treatment. Antihistamines (such as benzatropine or diphenhydramoine) are recommended when there are co-occuring extrapyramidal symptoms but are not ideal for long-term use given their side effect profile.

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Small Group Resident Assessment

Session:		
Facilitator (s):		
DATE:		
Small Group 3	Contributes to group discussion	
	BE/ME/EE	Comments
Resident 1		
Resident 2		
Resident 3		
Resident 4		
Resident 5		
Resident 6		
Resident 7		
Resident 8		
Resident 9		
Resident 10		
Resident 11		
Resident 12		

BE—Below Expectations
Minimal discussion during the session
No discussion on the site discussion board
Comments not contributory to discussion or distracting to discussion
Has minimal knowledge of topic as expected of PGY year

ME—Meets Expectations
Contributes to group discussion in a meaningful way
Incorporate textbook/website/podcast reading into discussion
Has knowledge of topic appropriate to level of training

EE—Exceeds Expectations
Contributes to group discussion in a meaningful way
Incorporate literature into discussion
Has advanced knowledge of topic

Small Group Evaluation

The moderator demonstrated adequate knowledge of subject.

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

The moderator's facilitation of the conference facilitated my learning.

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

The overall discussion was relevant to the stated topic(s).

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

The faculty/resident's teaching methods (slides, handouts, videos, etc.) were effective.

5) Strongly Agree 4) Agree 3) Slightly Agree 2) Disagree 1) Strongly Disagree

Faculty Facilitator Evaluation

1. **Preparation – was faculty well prepared?**

Needs Improvement Effective Exemplary

2. **Engaged residents - Encouraged discussion and actively participated, demonstrated enthusiasm?**

Needs Improvement Effective Exemplary

Strengths:

Areas for Improvement:

Reviewer Recommendations:

Resident Facilitator Evaluation

1. **Preparation – was the resident facilitator well prepared?**

Needs Improvement Effective Exemplary

2. **Engaged residents – Controlled and led the session and encouraged discussion, active involvement, and demonstrated enthusiasm?**

Needs Improvement Effective Exemplary

Strengths:

Areas for Improvement:

Reviewer Recommendations:

Evaluation of the Teaching materials

1. **Were the objectives appropriate for the topic?**

Needs Improvement Effective Exemplary

2. **Was the amount of material appropriate?**

Too Short Appropriate Too Long

Strengths:

Areas for Improvement:

Reviewer Recommendations: