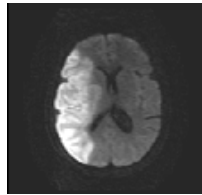
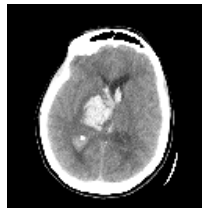


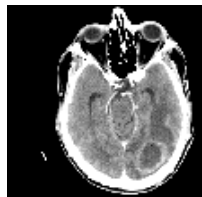
HMC Neurobook



Diffusion weighted MRI sequence of a 62-year-old woman with sudden left sided weakness and dysarthria



Non-contrast head CT of a 47-year-old woman found down with history of hypertension and stimulant drug use



Contrast-enhanced head CT of a 49-year-old man with confusion and aphasia

**The Harborview Medical Center Neurology Service
University of Washington
Seattle, WA**

Version 1.7, last revised 10/15

Assembled by Dr. Sandeep Khot with input from Neurology residents and attendings

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Introduction

Welcome to the neurology service at Harborview Medical Center. This rotation provides an opportunity to learn about the evaluation and care of patients with a variety of neurologic conditions. As with many patients seen throughout Harborview, our patients often present a challenging mixture of neurologic, medical, psychiatric and social problems. During this rotation, you will not learn all of neurology but will gain experience on handling acute inpatient neurological problems. Key among the skills you will practice is the neurologic examination. Since every patient you examine will also be examined by a neurology resident and an attending, you will have ample opportunity to check your findings against the findings of others.

This booklet provides an initial guide to neurology in general but especially at Harborview. We hope it will help you get the most out of this rotation. The booklet is not meant to be comprehensive. You will have many questions that are not addressed here. If you do not understand something, others likely have the same questions—please ask. We hope that by caring for patients on the neurology service you will start to develop the skills necessary to identify patients with acute neurologic problems and to acquire the knowledge needed to initiate treatment for these patients.

Good luck and welcome to the service!

A Day in the Life

6:30 AM Pre-rounding

R1s cannot begin pre-rounding before 6:30 AM.

7:30 AM Morning Report

Team meets in 3 West Hospital Neurology Team Room (3WH-22) every morning. Post-call residents briefly review cross-cover issues on other team and new admissions to their team.

7:30-9:30 AM Work Rounds

The expectation is that work rounds will finish by 9:30 AM before imaging rounds. At 8:05 the neurology teams meet with the neuro-critical care teams in the NICU to co-manage the ICU patients on the service.

9:30-10:30 AM Imaging Rounds

Review imaging tests with neuro-radiologist in the Radiology Nelson Library on the 1st Floor.

11:00 AM R1 Lectures

As noted on the team-room calendar and as announced in morning report, R1 Lectures occur at 11 AM on Fridays and some Tuesdays. Other conferences include Wednesday lunch time conference, Tuesday afternoon conferences (4-5 PM) and Thursday afternoon Neurology Grand Rounds.

Cores sign-in and sign-out

Communication is key to providing excellent patient care. Please use CORES to achieve this communication and make sure CORES always accurately reflects the primary contact for the patient.

The Neurological Examination

Besides a detailed history, clinicians must use a structured neurological examination to localize the nervous system lesion(s) that explains the patient's condition. Localization is essential to deciding the evaluation, diagnosis, and treatment. The examination allows you to distinguish diseases of the central and peripheral nervous system and points you towards the mechanism of disease. The examination is presented in a particular order to help others digest the information and to ensure that parts are not forgotten. The following is merely an outline for the structure of what is to be included in the examination. Usually a more focused examination is needed based on the patient's history and presenting symptoms.

In doing your examination, it is important to document your observation in a way that can be repeated and understood by others. For example, "Cranial nerves II-XII" normal can mean different things to different people. On the other hand "pupils 4 mm, equal and reactive" is much more precise. Also, make use of your observations such as "patient resists my attempts to open their eyes," or "looks toward examiner entering from the left" or "holds arms up against gravity spontaneously." These are descriptive and repeatable observations.

You will have plenty of opportunities on this rotation to perform detailed neurologic exams and we encourage you to seek feedback on your exam from the neurology residents and attendings.

Mental Status:

- Level of Consciousness: Alertness, interaction, responsiveness to command, orientation. Consider noting a Glasgow Coma Scale score in comatose patients and Mini-Mental State Examination score (MMSE) or Montreal Cognitive Assessment (MoCA) in patients with cognitive problems.
- Speech: check for fluency, comprehension, repetition, and naming.
- Mentation and Attentiveness: Tests of attention, memory and higher cognitive processes. Consider MMSE or MoCA score in patients with cognitive problems. (see website: <http://www.mocatest.org>)

Cranial Nerves:

- I: Ability to smell (rarely tested)
- II: Visual fields, visual acuity, fundoscopic examination
- II, III: Pupillary response to light
- III, IV, VI: Extraocular movements in horizontal and vertical planes; note any nystagmus
- V: Facial sensation, muscles of mastication, jaw jerk
- V, VII: Corneal reflexes

VII: Muscles of facial expression, taste over the anterior 2/3 of tongue

VIII: Hearing, vestibular function

IX, X: Elevation and sensation of soft palate, gag reflex

XI: Sternocleidomastoid and trapezius strength

XII: Tongue protrusion

**** In unresponsive pts without cervical spine problems, can test Doll's eyes or oculocephalic reflex to test eye movements (CN III,IV,VI),**

Motor:

- Observe muscle bulk, fasciculations. Test tone and strength (graded 0-5) as well as functional tests (pronator drift).
- Examine dexterity in a cooperative patient.

Reflexes:

Deep tendon reflexes (biceps, triceps, knee, ankle), plantar responses (Babinski's sign). May include jaw jerk. Graded 0 (no response), trace (reinforcement required), 1 (diminished), 2 (normal, average), 3 (brisker than average), 4 (clonus).

Other reflexes: Abdominal, Hoffman, glabellar, snout, palmomental, cremasteric, bulbocavernosis

Sensory:

- Primary Modalities: light touch, pinprick, temperature, vibration, and joint position sense
- Cortical Sensation: two point discrimination, graphesthesia, stereognosis, and extinction
- Romberg's test

Coordination:

- Finger-nose, heel-shin, rapid alternating movements,
- Fine finger movements and toe tapping

Gait and station:

Normal, tandem, heel and toe walking, note base or stance and arm swing.

Movements:

Abnormal movements and speed of movement

Meningeal and mechanical signs:

Neck stiffness, Kernig's and Brudzinski's sign
straight leg raising

Vascular status:

Auscultation of head and neck, heart, extremity vessels

*** For further details on the neurological examination including examples of both normal and abnormal exams, please see the appendix from the following website:*

<http://courses.washington.edu/neural/pdfs/pocetsyllabus.pdf>

Brain Vascular Disease

The UW Medicine Comprehensive Stroke Center is located at HMC; you will have ample opportunities to care for stroke patients. Acute treatments follow the principles outlined in the Acute Stroke Algorithm, which is found a few pages ahead and online at www.stroke.washington.edu, where you can also find printable documents such as the NIH Stroke Scale (see below), Coagulopathy Reversal guidelines, CPOE ischemic and hemorrhagic stroke admission orders, and published guidelines summarizing the evidence upon which these recommendations are based. Decisions about diagnosis and treatment are typically based on conversations between the neurology resident, the service attending, and the stroke attending carrying the stroke phone (206-744-6789). You will learn about the main types and subtypes of stroke, clinical management during the acute phase, and the initial work-up for stroke patients.

Key Facts about Stroke in the U.S.

American Heart Association Annual Heart Disease and Stroke Statistics (www.americanheart.org/statistics/index.html)

- 4th leading cause of death and leading cause of disability
- Someone suffers a stroke every 40 seconds and dies from a stroke every 4 minutes, approximately 1 of every 18 deaths

- Women account for about 60% of all stroke deaths; stroke kills more women than breast cancer. Women also have greater disability than men after stroke.
- 1 in 5 persons require institutional care at 3 months; about 1/3 of persons are permanently disabled by their stroke

Ten Things You Should Know about Stroke

Time is brain

Acute interventions are most successful when they are provided as soon as possible after the onset of symptoms. Every minute counts. Harborview has a goal of door to IV tPA treatment time of 45 minutes in appropriate ischemic stroke patients. Some treatments are still available for patients with ischemic strokes who present within 6 hours of symptom onset, maybe even longer if it is a posterior circulation (basilar artery) syndrome (see Acute Stroke Algorithm below and at www.stroke.washington.edu). Treatments include intravenous tPA, for which the best evidence of efficacy exists, and endovascular approaches.

Timing is also of importance among patients with transient ischemic attack (TIA). The 3 month risk of stroke after a TIA is about 10%, with the greatest risk in the first week. The ABCD2 score is a simple prognostic tool for short-term stroke risk

after a TIA and is based on 5 factors: age, blood pressure, clinical features (including weakness and speech impairment), duration and diabetes. A higher score on this validated risk assessment tool predicts a greater risk of stroke within 2, 7, 30 and 90 days after a TIA. At HMC, the majority of patients with TIA are admitted to the hospital for an expedited stroke workup.

The initial imaging study of choice in stroke is the non-contrast head CT

The head CT is the most reliable way to differentiate ischemic from hemorrhagic strokes. All head CTs must be read IMMEDIATELY with the neurology residents. A CT angiogram tailored to the stroke type (ischemic vs. hemorrhagic) can also be useful early in the patient's course. Ischemic stroke patients should almost always have a CTA of both the neck and brain.

Ischemic stroke treatment varies based on subtype

Ischemic strokes are caused by a blockage or clot within an artery supplying the brain. They can be divided into small vessel, large vessel, embolic and other. Embolic strokes result from clots that can arise from arteries, cardiac structures, or veins, assuming a right-to-left shunt that circumvents the pulmonary circulation. Embolic strokes can involve multiple emboli and vascular territories. The clinical evaluation for ischemic stroke should help

identify the subtype. The treatment options may differ based on these findings.

Hemorrhagic stroke represents 1/5 strokes in the population but 2/3 of strokes at Harborview due to the large number of referrals

Spontaneous intracranial hemorrhages are caused by the rupture of an artery within the brain or in the subarachnoid space. Deep intraparenchymal hemorrhages (IPH) are most commonly caused by hypertension, and subarachnoid hemorrhages, by a ruptured berry aneurysm. Amyloid angiopathy is a cause of superficial intraparenchymal hemorrhages in elderly patients.

Blood pressure treatment varies by stroke type:

In patients with ischemic stroke who have not received tPA, allow for permissive hypertension.

Lowering blood pressure in the face of an acute ischemic stroke may worsen neurologic outcome. Often some outpatient anti-hypertensive medications will need to be held. Unless some other specific reason exists to lower the blood pressure (such as ongoing cardiac ischemia), elevated blood pressure is not treated in a patient with an acute ischemic stroke unless the systolic exceeds 220 mm Hg or the diastolic exceeds 120 mm Hg. Typically the blood pressure falls without treatment in the first few days after presentation. In the long term, hypertension is the major risk factor for re-

current stroke and needs to be treated. When treatment for persistently elevated blood pressure should begin after an acute ischemic stroke is unknown, but probably not before one week from stroke onset.

In patients with ischemic stroke who receive tPA, blood pressure control is needed before and after the drug is given.

To be eligible for IV tPA a patient's blood pressure must be less than 185 mm Hg systolic and 110 mm Hg diastolic. Regimens such as those in the table below from a guideline paper are used and rely on labetalol and nicardipine (see links on Stroke Algorithm). For the 24 hours after tPA is given the goal is to keep the systolic blood pressure less than 180 mm Hg and the diastolic less than 105 mm Hg, using similar regimens.

In patients with intraparenchymal hemorrhages, blood pressure control and correction of coagulopathies are needed

Both hypertension and coagulopathies can contribute to hematoma expansion, which is associated with worse neurologic outcomes in patients with intraparenchymal hemorrhages. The ideal blood pressure in these patients is unknown. We try to keep the systolic blood pressure less than 160 mm Hg and the diastolic less than 105 mm Hg, using regimens as for ischemic stroke patients who have received tPA (see below). A group of UW hema-

tologists and neurologists have established guidelines for reversing coagulopathies, and these are available through the stroke algorithm. They should be reviewed in every patient with an intraparenchymal hemorrhage.

The ICH score is a reliable grading scale that allows for risk stratification on presentation for patients with intraparenchymal hemorrhage (see below). The score involves a measurement of IPH volume based on the formula $ABC/2$ where A and B are perpendicular sizes in cm on the cut with biggest hemorrhage and C is vertical size (number of involved slices x slice thickness).

Brain edema and herniation can occur in ischemic and hemorrhagic stroke

Avoid sedating drugs and watch closely for signs of increase intracranial pressure (e.g. headache, vomiting, anisocoria, decreased LOC, and worsening or new focal neurological signs). NEVER give standing orders for hypotonic fluids (such as half-normal saline). For increased intracranial pressure and herniation, patients should be intubated for airway control and the head of the bed elevated (typically to 30 degrees). Other options include osmolar therapy (including mannitol or hypertonic saline- 2%, 3% or 23.4%) and hypothermia. If these interventions are not effective, we may ask the neurosurgeons to perform a decompressive hemicraniectomy, typically sooner rather than later.

Stroke work-up will vary between patients

The quick look with CT is often complemented by MRI with special sequences to see acute ischemia (DWI), chronic ischemia (FLAIR), and chronic bleeds (GRE). Vascular imaging is needed in many patients with both ischemic and hemorrhagic strokes and can include CT or MR angiography, carotid and vertebral Duplex, transcranial Doppler (TCD) with emboli monitoring, catheter angiography, or some combination of these tests. Other studies in patients with ischemic strokes include cardiac rhythm monitoring, a transthoracic echocardiography with bubble study to evaluate for PFO. Transesophageal echocardiography and a hypercoagulable work-up are usually reserved for young patients without an obvious explanation for their ischemic stroke.

Antithrombotic therapy is used for secondary prevention after ischemic stroke

If the patient was previously not on anti-platelet therapy, aspirin 325 mg PO QD or 300 mg PR QD can be initiated in the acute setting. In patients treated with tPA, no antithrombotic therapy is given for 24 hours and until a follow-up head CT is obtained. The combination of aspirin and clopidogrel can be used in the acute phase in patients not receiving IV tPA. In general, full anticoagulation with heparin or low molecular weight heparin is NOT used in the acute setting. Even in patients with atrial fibrillation, immediate anticoagulation does not provide a clear benefit and increases the risk of conversion of a bland to a hemorrhagic infarct.

Stroke Quality of Care

It is measured for all patients on nationally established performance measures. Help us achieve these quality measures.

All Stroke (Ischemic, IPH, SAH)

- **Venous Thromboembolism (VTE) Prophylaxis:** All stroke pts have VTE prophylaxis (SCD's OR Pharmacologic therapy) or documentation reason why not by end of hospital day 2.
- **Stroke Education:** Documented that all stroke patients (or caregivers) are given educational materials addressing: activation of EMS, need for follow-up after d/c, medications prescribed at discharge, risk factors for stroke, & warning signs & symptoms of stroke.
- **Assessed for Rehabilitation:** All stroke patients need documented assessment for rehabilitation

Ischemic Stroke

- **Thrombolytic Therapy:** Acute ischemic stroke patients who arrive within 2 hours of time last known well, given tPA within 3 hours of time last known well OR documented reason why they were not a tPA candidate.
- **Antithrombotic Therapy By End of Hospital Day 2:** Ischemic stroke patients administered antithrombotic therapy by the end of hospital day 2 OR have a clearly documented reason why not.

- **Discharged on Statin Medication:** Ischemic stroke patients with LDL >100 mg/dL, or not measured, or who were on a lipid-lowering medication prior to arrival prescribed statin at discharge. Must be outlined in discharge summary.
- **Discharged on Antithrombotic Therapy:** Ischemic stroke patients prescribed antithrombotic therapy at hospital discharge as documented in discharge summary or documented reason why not.
- **NIHSS score documented:** required on admission

Ischemic stroke with atrial fibrillation

- **Anticoagulation Therapy for Atrial Fibrillation/Flutter:** Ischemic stroke patients with atrial fibrillation/flutter discharged on anticoagulation therapy as outlined in the discharge summary or reason why not.

Intraparenchymal hemorrhage

- **Severity measurement performed on admission:** ICH score for intraparenchymal hemorrhage on admission, prior to surgery
- **Procoagulant Reversal Agent:** For intraparenchymal hemorrhage if INR >1.4

Questions? Contact: Vicki Johnson, DNP, MHSEd, ARNP; vlj@uw.edu; office: (206) 744-2403

NIH Stroke Scale (printable link at www.stroke.washington.edu)

NIH Stroke Scale Item	Function	Scores
1a. Level of Consciousness (Alert, Drowsy, etc.)	Alert	0
	Drowsy	1
	Stuporous (requires repeated stimuli)	2
	Comatose (reflex responses only)	3
1b. LOC Questions (Ask month, age)	Both correct	0
	One Correct	1
	Both Incorrect, unable to answer*	2*
1c. LOC Commands (Open, close eyes, make fist, let go)	Obeys both correctly	0
	Obeys one correctly	1
	Both Incorrect, unable to perform*	2*
2. Best Gaze (Eyes open-patient follows examiner's finger or face)	Normal	0
	Partial gaze palsy (for coma see Note 2 below)	1
	Forced deviation	2
3. Visual (Introduce visual stimulus/threat to patient's visual field quadrants)	No loss	0
	Partial hemianopia (for coma see Note 3 below)	1
	Complete hemianopia	2
	Bilateral hemianopia	3
4. Facial Palsy (Show teeth, raise eyebrows and squeeze eyes shut)	Normal	0
	Minor asymmetry	1
	Partial (lower face paralysis)	2
	Complete*	3*
5a. Motor Arm-Left (Elevate extremity 90 degrees for 10 sec when sitting and score drift/movement)	No drift	0
	Drift	1
	Some effort against gravity	2
	No effort against gravity	3
	No movement*	4*
5b. Motor Leg-Left (Elevate extremity 30 degrees for 5 sec when lying down and score drift/movement)	No drift	0
	Drift	1
	Some effort against gravity	2
	No effort against gravity	3
	No movement*	4*
6a. Motor Arm-Right (Elevate extremity 90 degrees for 10 sec when sitting and score drift/movement)	No drift	0
	Drift	1
	Some effort against gravity	2
	No effort against gravity	3
	No movement*	4*
6b. Motor Leg-Right (Elevate extremity 30 degrees for 5 sec when lying down and score drift/movement)	No drift	0
	Drift	1
	Some effort against gravity	2
	No effort against gravity	3
	No movement*	4*
7. Limb Ataxia (Finger-Nose, heel down shin)	Absent*	0*
	Present in upper or lower	1
	Present in both	2
8. Sensory (Pin prick to face, arm, trunk, and leg compare side to side)	Normal	0
	Partial Loss	1
	Dense Loss*	2*
9. Best Language (Name items, describe picture and read sentences)	No aphasia	0
	Mild-moderate aphasia	1
	Severe aphasia (see Intubated and Alert notes)	2
	Mute*	3*
10. Dysarthria (Evaluate speech clarity by patient repeating listed words)	Normal articulation (if appropriate)	0
	Mild-moderate slurring	1
	Severe, nearly unintelligible or worse*	2*
11. Extinction and Inattention (Use information from prior testing to identify neglect or double simultaneous stimuli testing)	No neglect	0
	Partial neglect	1
	Profound Neglect*	2*
NIH Stroke Scale Total		

*assign this score if patient comatose or unresponsive

Note 2: perform oculocephalic testing to determine score if comatose, absent oculocephalic reflex leads to score of 2

Note 3: determine via visual threat if comatose, no response leads to score of 3

Intubated and Alert: for item 9, make best guess and score; for item 10, untestable so score 0

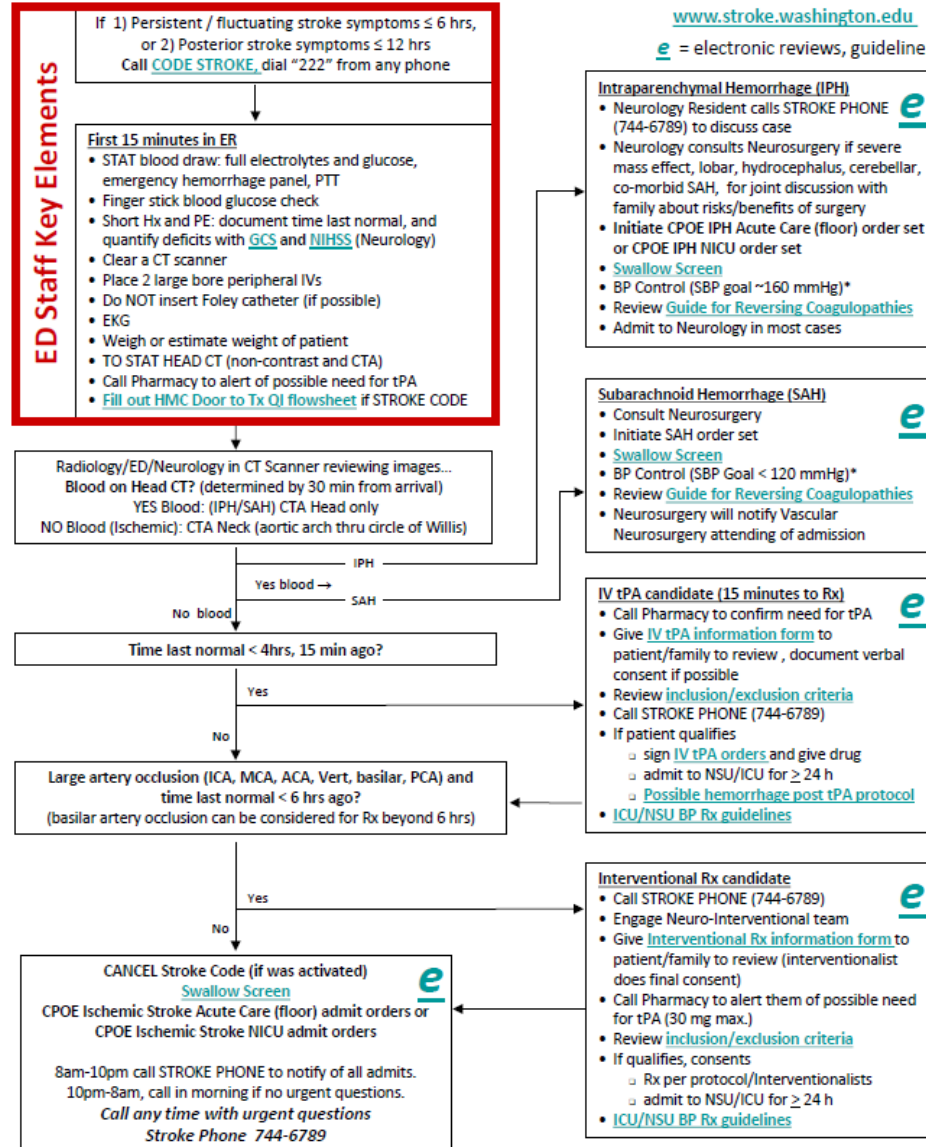
Acute Stroke Evaluation and Treatment

(updated April 2013)

UW Medicine Comprehensive Stroke Center at Harborview

Online with links at www.stroke.washington.edu

e = electronic reviews, guidelines



* IV labetalol then nicardipine drip preferred BP agents

Approach to Arterial Hypertension in Acute Ischemic Stroke After tPA (*Stroke* 2007; 38: 1671)

Indication that patient is eligible for treatment with intravenous rtPA or other acute reperfusion intervention

Blood pressure level

Systolic >185 mm Hg or diastolic >110 mm Hg

Labetalol 10 to 20 mg IV over 1 to 2 minutes, may repeat $\times 1$;

or

Nitropaste 1 to 2 inches;

or

Nicardipine infusion, 5 mg/h, titrate up by 0.25 mg/h at 5- to 15-minute intervals, maximum dose 15 mg/h; when desired blood pressure attained, reduce to 3 mg/h

If blood pressure does not decline and remains >185/110 mm Hg, do not administer rtPA

Management of blood pressure during and after treatment with rtPA or other acute reperfusion intervention

Monitor blood pressure every 15 minutes during treatment and then for another 2 hours, then every 30 minutes for 6 hours, and then every hour for 16 hours

Blood pressure level

Systolic 180 to 230 mm Hg or diastolic 105 to 120 mm Hg

Labetalol 10 mg IV over 1 to 2 minutes, may repeat every 10 to 20 minutes, maximum dose of 300 mg;

or

Labetalol 10 mg IV followed by an infusion at 2 to 8 mg/min

Systolic >230 mm Hg or diastolic 121 to 140 mm Hg

Labetalol 10 mg IV over 1 to 2 minutes, may repeat every 10 to 20 minutes, maximum dose of 300 mg;

or

Labetalol 10 mg IV followed by an infusion at 2 to 8 mg/min;

or

Nicardipine infusion, 5 mg/h, titrate up to desired effect by increasing 2.5 mg/h every 5 minutes to maximum of 15 mg/h

If blood pressure not controlled, consider sodium nitroprusside

The ICH Score (*Stroke* 2001; 32: 893)

TABLE 3. Determination of the ICH Score

Component	ICH Score Points
GCS score	
3–4	2
5–12	1
13–15	0
ICH volume, cm ³	
≥30	1
<30	0
IVH	
Yes	1
No	0
Infratentorial origin of ICH	
Yes	1
No	0
Age, y	
≥80	1
<80	0
Total ICH Score	0–6

The ABCD2 Score (*Stroke* 2009; 40: 750)

Table 1. ABCD2 Scoring Criteria*

A	Age	≥60 years	1 point
B	Blood pressure	≥140/90 mm Hg	1 point
C	Clinical features	Unilateral weakness	2 points
		Speech impairment without weakness	1 point
D	Duration	≥60 minutes	2 points
		10–59 minutes	1 point
D	Diabetes	Presence of diabetes mellitus	1 point

Seizures and Epilepsy

Seizures and epilepsy are some of the most common neurological disorders. Learning to care for patients with these is an integral part of this rotation. Differentiating a first time epileptic seizure from seizure-like symptoms such as syncope or psychogenic non-epileptic events is essential but can be challenging. Clinically isolated seizures are fairly common with a 10% life time risk. A high proportion of seizures are provoked by metabolic or electrolyte derangements, drug intoxication or drug withdrawal. Seizures can also present in acute setting of other conditions affecting the brain such as CNS infection or trauma. The challenge is to make sure clinically significant seizures have stopped and that the underlying provoking factors (such as infection or drug withdrawal) have been addressed. A large portion of these patients are at risk of recurrent unprovoked seizures (i.e. epilepsy). The long term goal is to assess the risk of seizure recurrence, which will guide use of anti-epileptic medications.

Ten Things You Should Know about Seizures and Epilepsy

(Adapted from Poolos, et al. 2006)

The history is key to the diagnosis of seizures

Although you may occasionally witness a seizure in a patient, more commonly you will need an eyewitness who can describe the details about it. Ask

about auras, unusual behavior, type and description of the movements, urinary incontinence, tongue biting, and post-ictal neurologic deficits (e.g. confusion, aphasia or focal weakness). Other important points in history are prior head trauma or CNS infection, and precipitating factors such as sleep deprivation, medications, drugs, and systemic illness. In patients with recurrent events, history helps identify the precipitating factors and whether the events are stereotyped (as should be in epilepsy). A phone call to a family member can prove quite valuable.

A single seizure may not mean epilepsy

Epilepsy is defined as a tendency towards recurrent unprovoked seizures. The risk of recurrence after a single unprovoked seizure is only 40% if no other risk factors, but rises if risk factors exist. For example, it is above 80% with a history of moderately severe traumatic brain injury. Risk of recurrence is increased by the presence of a structural brain lesion (as noted on history, MRI or neurological exam), epileptiform changes on EEG, partial onset of seizures, and other factors such as history of status epilepticus, post-ictal Todd's paralysis, or family history.

MRI and EEG are useful for making a diagnosis and identifying underlying causes.

After an acute seizure clinical circumstances may indicate tests of electrolyte studies, a head CT, lumbar puncture for CSF studies, drug screening, AED

levels, or other tests to help identify acute provoking factors needing urgent attention. Subtle lesions such as low grade neoplasms or vascular malformations are often missed on head CT. An MRI with and without contrast should be included in the initial workup for all patients with epilepsy of unknown cause. An EEG, may help in making the correct diagnosis or identifying risk of recurrence. An initial EEG is normal in half the patients with epilepsy.

Anti-Epileptic Drugs (AEDs) are the Main Tool for Treating Epilepsy.

AEDs should be chosen by efficacy, tolerability, and other patient factors such as compliance, need for rapid titration, drug interactions, etc. The type of epilepsy, as determined by history and EEG, makes a difference for AED selection. Some AEDs have a narrow therapy spectrum (only work for certain types of epilepsy) such as ethosuxamide which is used for absence epilepsy. Sometimes AEDs can also paradoxically exacerbate seizures such as carbamazepine and gabapentin exacerbating absence epilepsy. If selected appropriately AEDs generally reduce the burden of epilepsy. Initial monotherapy is effective in controlling seizures in half the patients. An alternative AED or poly-therapy may be indicated if the first drug fails.

A few AEDs can be given IV or acutely

IV loading is possible with fosphenytoin, valproic acid, phenobarbital, levetiracetam and lacosamide. Administration of phenobarbital and phenytoin requires close monitoring of cardiovascular and respiratory functions due to risk of arrhythmias, cardiac and respiratory depression.

The typical loading dose of IV phenytoin (Dilantin) is 20 mg/kg given at a rate not to exceed 50 mg/min and requires a large vein. Oral loading of phenytoin is possible, but requires the loading dose to be divided over several hours to allow absorption (e.g. 500 mg every 4-6 hours for 3 doses).

Epilepsy in adults is typically a chronic and often a disabling condition.

While children sometimes outgrow their epilepsy, in adults its spontaneous remission is uncommon. Need for AEDs is generally long term. Direct risk of death from brief acute seizures is low, particularly when an effective AED regimen is established. Even then, acute discontinuation of AEDs can result in life threatening status epilepticus.

About two-thirds of those with epilepsy have seizures fully controlled with AEDs. Patients with uncontrolled seizures (e.g. one complex partial seizure every 6 months) are required to follow seizure precautions (see discharge section).

The disability of epilepsy is due to loss of control due to unpredictable nature of seizures which place limitations on work and transportation, affecting independence, employability, and income. Other major causes of disability are drug related side effects and comorbidities, which include depression.

For chronic treatment, start low, go slow

AEDs are better tolerated when tapered up slowly. Elderly patients may require lower drug doses than are typically used in younger patients.

Treat the patient, not the drug levels

In general, make dosing decisions based on the presence of side effects and the level of seizure control, both of which can be determined clinically.

Drugs levels may be useful in some inpatients where the dose needs to be adjusted to maximize the benefit without causing toxicity, or when drug interactions are anticipated. Phenytoin is highly protein bound (about 90%) with the free portion being what is available to the brain. In inpatients with low protein or abnormal protein binding (such as dialysis), the free phenytoin fraction may be higher than the typical 10% of the total. Thus free levels of 1.0 to 2.0 ug/mL are a common goal.

AEDs can have side effects, sometimes serious

Risks and intolerable side effects are associated with various AEDs and include: life threatening allergic reactions, exacerbation of epilepsy, somnolence, cognitive and depressive symptoms. Other rare reactions for individual AEDs include liver and bone marrow toxicity, pancreatitis, kidney stones, as well as adverse drug interactions. In general, risks of life threatening reactions are *lower* than risks from recurrent seizures. In women of childbearing age, teratogenicity is an additional concern, particularly for valproate. For these reasons, AED selection should be based on effectiveness as well as tolerability/toxicity. AEDs are generally better tolerated when used in monotherapy. In patients of Asian descent HLA B*1502 testing is advised before starting carbamazepine since this haplotype is highly associated with risk of Steven-Johnson Syndrome.

Status epilepticus is a medical emergency

Status is defined as a continuous seizure or repetitive discrete seizures with impaired consciousness in between events lasting 5 to 30 minutes or longer. Causes include anticonvulsant withdrawal, medical noncompliance, metabolic disturbance, drug toxicity, CNS infection or tumors, refractory epilepsy and head trauma. A common initial treatment includes an intravenous benzodiazepine and phenytoin. See one commonly used status treatment algorithm be-

low. Treatment of status epilepticus is most successful if provided rapidly.

Treatment of status epilepticus

- ACLS - attention to airway and cardiorespiratory function
- Treat hyperthermia, hypoglycemia, hyponatremia and other metabolic derangements
- Stop seizures with antiepileptic drug therapy

CURRENT CONCEPTS

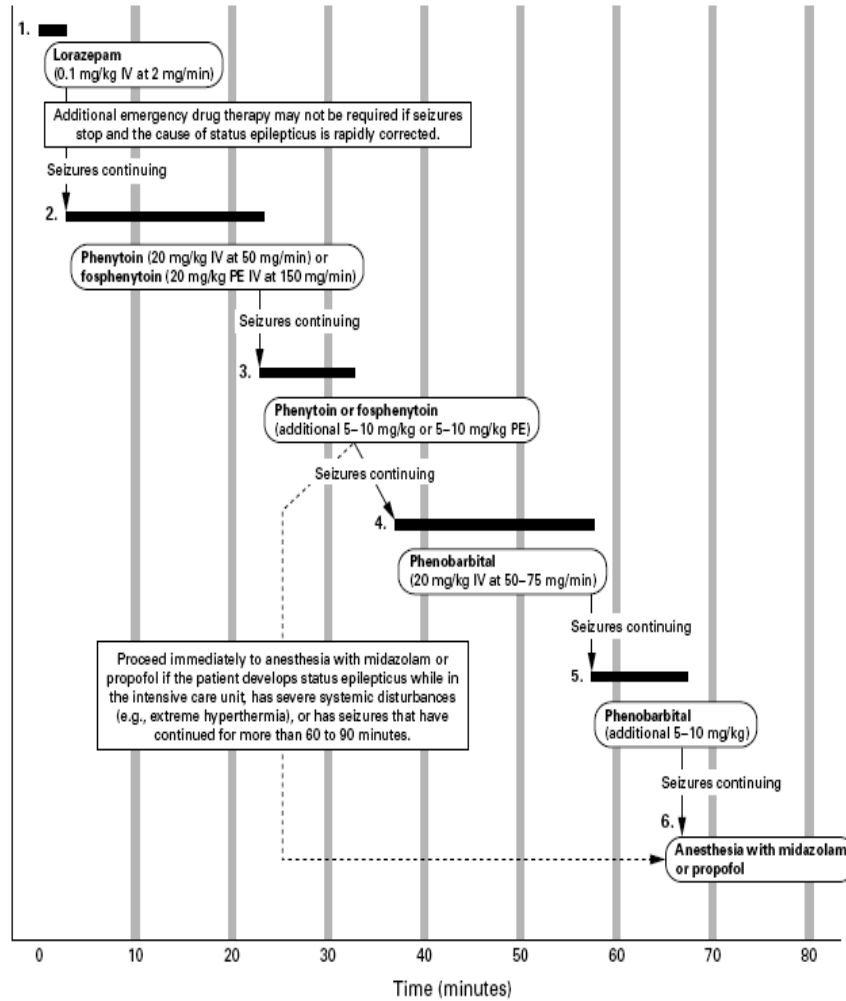


Figure 2. Antiepileptic-Drug Therapy for Status Epilepticus. IV denotes intravenous, and PE phenytoin equivalents. The horizontal bars indicate the approximate duration of drug infusions.

For status epilepticus, if no allergy or specific contraindications:

1. Begin with lorazepam (Ativan) 0.1 mg/kg as a single dose. Diazepam (Valium) 5-10 mg can be given instead of lorazepam.
2. Also, load fosphenytoin 20 mg Phenytoin equivalent/kg at a rate no faster than 150 mg PE/min (slower in patients with cardiac issues). Some newer algorithms suggest using Valproic acid 40-60 mg/kg administered at 3 mg/kg/minute before or after phenytoin.
3. If seizures continue, obtain a stat phenytoin level and load additional fosphenytoin 5 - 10 mg PE/kg (or valproic acid). Intubation is usually required at this point.
4. If seizures continue, a phenobarbital load or continuous infusion with midazolam should be considered. General anesthesia with propofol or pentobarbital are also options.
5. If you opt to use phenobarbital, load with 20 mg/kg IV no faster than 50 - 75 mg/min. If seizures persist, load with additional 5-10 mg/kg.
6. If you opt to use midazolam, begin the infusion with bolus of 0.15 - 0.2 mg/kg then 0.1-0.4 mg/kg/hr IV

7. If you opt for anesthesia, initiate continuous infusion of propofol or pentobarbital. Anesthesia with drug induced coma requires continuous EEG monitoring.

Stupor and Coma

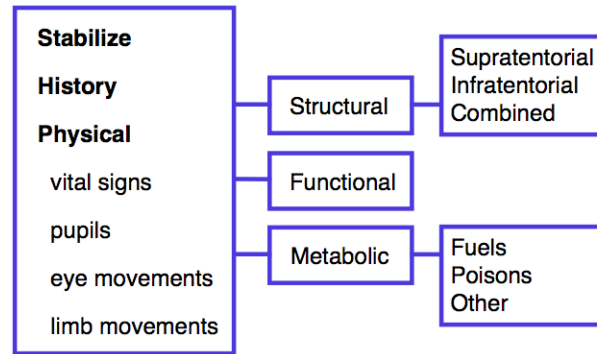
One of the most common presentations to the HMC neurology service is the patient with altered level of consciousness. The purpose of this section is to familiarize you with the general approach to the unresponsive patient and with the key features of the examination of these patients.

In an unresponsive patient you need rapidly to establish whether the cause is: metabolic, such as hypoglycemia; structural, such as with a stroke; or psychogenic, such as with hysteria. The examination is key in distinguishing among these mechanisms of unresponsiveness.

Spectrum of acute alterations of consciousness:

Alert	Normal
Delirious	Increased sleep, disorientation, fear, agitation, often visual hallucinations
Obtunded	Moderate reduction in consciousness, decreased interest in environment
Stupor	Arousable only by vigorous and repeated stimulation
Coma	Total absence of awareness of self and environment

Evaluation of the Unresponsive Patient



Key points of the coma exam

PUPILS: Pupillary size is dependent on sympathetic and parasympathetic input. Reactivity is usually resistant to metabolic insults although exceptions exist for hypothermia and severe barbiturate toxicity. Pupils are usually small and reactive in metabolic causes. Unilateral papillary dilation may indicate uncal herniation.

EYE MOVEMENTS: While full and conjugate eye movements can be seen in either metabolic or structural disease, asymmetry suggests structural disease. The oculocephalic reflex or “Dolls eyes” is inhibited by an intact cortex (suppression of eye deviation). In the oculovestibular reflex or “calorics”, the cortex is responsible for the fast component of

nystagmus and the brain stem for the slow component. In patients with intact brain stem reflexes but cortical damage, the reflex will not elicit a fast component of the cold calorics response. Rather, the eyes will become tonically deviated toward the ear being injected with cold water.

LIMB MOVEMENTS: The motor responses are as indicated on the Glasgow Coma Scale: obeying (6), localizing (5), withdrawing (4), abnormal flexor posturing (3), extensor posturing (2), and lacking any response (1). All motor responses can be seen in the same patient over time in cases of central (rostral-caudal) herniation. While symmetry in motor responses can be seen in either metabolic or structural disease, asymmetry suggests structural disease. Tremor, asterixis, and myoclonus may be seen in metabolic causes of coma.

Brain Imaging and Coma

A mass causing coma is not subtle on brain imaging.

If the CT is negative and you suspect structural problem, do MRI and MRA, CTA, or TCD to make sure the problem is not in the basilar artery.

If the initial metabolic screens and CT are negative and you suspect metabolic problem, consider an LP to rule out infection and EEG to rule out sub-clinical status epilepticus.

What to do if the LP suggests infection:

- **Bacterial Empiric coverage:** ceftriaxone 2g IV q12hr and IV vancomycin 20mg/kg IV load (max 2.5gm) then 15mg/kg q12hr. Immediately start dexamethasone 10mg q6h for 4 days. The first dose should be given prior to antibiotics, if possible. Consider ampicillin 2g IV q4h to cover for *Listeria* (especially in elderly, nursing home patients and children).
- **Viral Empiric coverage:** acyclovir 10mg/kg IV q8h for Herpes Simplex Virus until negative PCR (with IVF if possible to avoid kidney injury).
- If *N. meningitidis* is a possible causative organisms, consult with ID about respiratory isolation and treatment for exposed family members and health care workers.

At the Time of Discharge

Appointments

Most neurology inpatients should have outpatient follow up, but not all of that follow up needs to be in the Neurology Clinic. Please send CPOE referrals to schedule the patients prior to discharge. The PCC for the general neurology clinic can also be reached at 744-3422 and for the Stroke clinic at 744-6285.

Acute stroke patients

In patients who are discharged home, follow-up in 2-4 weeks after discharge in HMC Stroke Clinic with ARNP and 2-3 months with stroke attending.

Patients without need for neurologic follow up

These patients may be followed up in the Adult Medicine clinic or with an outside PCP.

Non-stroke patients with need for neurological follow up

- 2-4 weeks with their PCP. If without a PCP, attempt to establish one.
- 2-4 months in the Adult Neurology clinic.
- Health care specialists can see discharged patients in their clinic soon and serve as a bridge to the Adult Neurology Clinic.

Neuro-HIV patients

Make sure these patients are seen in the Madison clinic with their PCP and/or Dr. Marra in the Madison clinic or Dr. Zunt in the general neurology clinic.

Medications, studies and tests

Be sure to refill medications until the next scheduled appointment time, especially in stroke and epilepsy patients. The usual is a **3 month supply**. Some patients will fill their medication on the outside and will require written prescriptions. Try to arrange studies and tests by faxing requests and forms to the PCC prior to patient's departure.

Seizure Precautions

State law bans driving for 6 months for "unexplained loss of awareness", and that includes seizures (but may apply to other conditions as well). Any situation where a temporary lapse in awareness puts the patient or others at risk of injury should be avoided. These include no swimming (unless there is a lifeguard around and with a vest), no bathing in a tub of water (due to risk of drowning), no climbing heights, no working with heavy or dangerous machinery such as fork lifts or chain saws, and no working near fire or water (e.g. swimming pool) or near other dangers. It is important to list these precautions on discharge activities for patient with suspected seizures.

Useful Websites – UW Net ID required

Stroke Algorithm:

www.stroke.washington.edu

Neuroradiology Basics tutorial (please review this brief presentation to better understand CT and MRI scans during daily radiology rounds):

www.depts.washington.edu/neurolog/seminars/index.php

Neuroanatomy reviews

<http://depts.washington.edu/neurolog/seminars/neuroanatomy-lectures.html>

Neurological Examination:

<http://courses.washington.edu/neural/pdfs/poektsyllabus.pdf>

Neurology Archived Lectures:

<http://depts.washington.edu/neurolog/seminars/wednesday/index.php>

OTHER USEFUL WEBSITES:

American Stroke Association Stroke Statements and Guidelines

http://my.americanheart.org/professional/StatementsGuidelines/ByTopic/TopicsQ-Z/Stroke-Statements-Guidelines_UCM_320600_Article.jsp

Epilepsy website

<http://professionals.epilepsy.com/homepage/index.html>

Washington University Neuromuscular Home Page

<http://neuromuscular.wustl.edu>

American Academy of Neurology Practice Guidelines

www.aan.com/go/practice/guidelines

UW Department of Pharmacy Anticoagulation Services

www.uwmcacc.org

UW Neurology Residency Website Selected Papers

<https://depts.washington.edu/resneuro/wordpress/>

Useful Phone Numbers

Stroke Phone

Stroke Phone 744-6789 (cell phone)

Offices

Neurology office 744-3251 (fax 744-8787)

Neurology clinic

General Clinic PCC (Chris Davis) 744-3422

Stroke Clinic PCC (Tracy Ezell) 744-6285

Attendings	office	pager
Kyra Becker	744-8028	541-4561
Claire Creutzfeldt	744-3570	540-4447
Arielle Davis	744-6061	680-1997
Shahin Hakimian	744-4340	559-3108
Gene Hu	744-3569	540-1665
Suman Jayadev	221-2930	680-2741
Sandeep Khot	744-2891	540-9174
Eric Kraus	598-0216	989-3553
Will Longstreth	744-5860	560-0257
Christina Marra	897-5400	645-3470
Sara Schepp	744-8029	680-2074
David Tirschwell	744-5929	405-5291
Melanie Walker	744-3251	340-3162
Jon Weinstein	744-2223	663-0829
Joe Zunt	744-3715	680-3991

Neurology residents

Copass Senior: 663-0650 (pager) 940-5929 (cell)

Sumi Senior: 663-0178 (pager) 714-3883 (cell)

Consult Senior: 663-0650 (pager) 708-3618 (cell)

Junior (cell): Copass 402-8438; Sumi 402-8437

Health Care Specialist: 714-2558

On-call resident after hours 663-0651 (pager)

Resident room -2144, -4506, -2979, -5732,
-4520, -4520, -4522, -4553, -4547, -4556

Cell phones 375-3792, 300-1053, 714-2576

NPs/PAs

Anna Krumpe 986-1911 (pager)

Mary Lou Willis 314-3002 (pager)

Lynn Smith 540-1319 (pager)

Pharmacists

Tom McPharlin 986-4700 (pager)

Debra Page 540-9648 (pager)

Kelly Pinson-Hinerth 540-9648 (pager)

3W Therapists

Physical Therapy 994-3526 (pager)

Occupational Therapy 994-4583 (pager)

Speech Therapy 993-5041 (pager)

NOTES:

Services and Contacts

3WA 4-5561 3WB 4-5562 3WC 4-5563

3W fax 4-8576

3E 4-3351

4E 4-3331

5E 4-3551

6E 4-3550

ED-OBS 4-8575

NICU: 2WA 4-5361 2WB 4-5362 2WC 4-5363

9EA 4-3127

9EB 4-5666

MCICU: 2EA 4-5371 2EB 4-5372 2EC 4-5373

TSICU: 9MB 4-4628

9MA 4-3510

Inpatient Laboratory 4-3451

Inpatient pharmacy 4-3220

TCD Office 4-3905

MRI 4-2460

CT 4-6937

Neurorad 4-6143

Angio suite 4-6505

Stroke Center 4-3975 or stroke@uw.edu