

ENLS Protocols

ENLS Protocol	Version	Date
Acute Non-Traumatic Weakness	1.0	5/23/2013
Acute Stroke	1.0	5/23/2013
Airway Ventilation and Sedation	1.0	5/23/2013
Coma	1.0	5/23/2013
Elevated ICP and Herniation	1.0	5/23/2013
Intracerebral Hemorrhage	1.0	5/23/2013
Ischemic Stroke	1.1	6/5/2013
Meningitis and Encephalitis	1.0	5/23/2013
Resuscitation following Cardiac Arrest	1.0	5/23/2013
Spinal Cord Compression	1.0	5/23/2013
Status Epilepticus	1.0	5/23/2013
Subarachnoid Hemorrhage	1.0	5/23/2013
Traumatic Brain Injury	1.0	5/23/2013
Traumatic Spine Injury	1.0	5/23/2013

Tools

Glasgow Coma Scale (GCS)
Hunt Hess Classification of SAH
World Federation Neurological Scale (WFNS)
of SAH

About ENLS

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ENLS Training and Certification

ENLS training and certification can be accomplished online at <http://www.neurocriticalcare.org> and clicking on the ENLS tab in the top menu. This online course is available for fee and CME credit to those interested in learning about ENLS and obtaining a certificate of training good for 2 years. More details can be found on the neurocritical care website.



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Feedback

The quality and integrity of ENLS is based in large part on user feedback. Feedback on each protocol or the overall concept of ENLS is reviewed in detail allowing us to update the protocols as needed. Feedback about the applicability of these protocols in different countries, new evidence that has not been integrated into ENLS protocol recommendation, and issues of clarity, etc. are all welcomed.

You may provide feedback by emailing enlsfeedback@neurocriticalcare.org, or by clicking on the feedback button on any of the online protocols.



ENLS Protocol Updates

Protocol	Version Change Date	Explanation
Acute Ischemic Stroke	1.0 -> 1.1 6/5/2013	Dose of PCC changed from mg/kg to mcg/kg; note this is errata in the manuscript

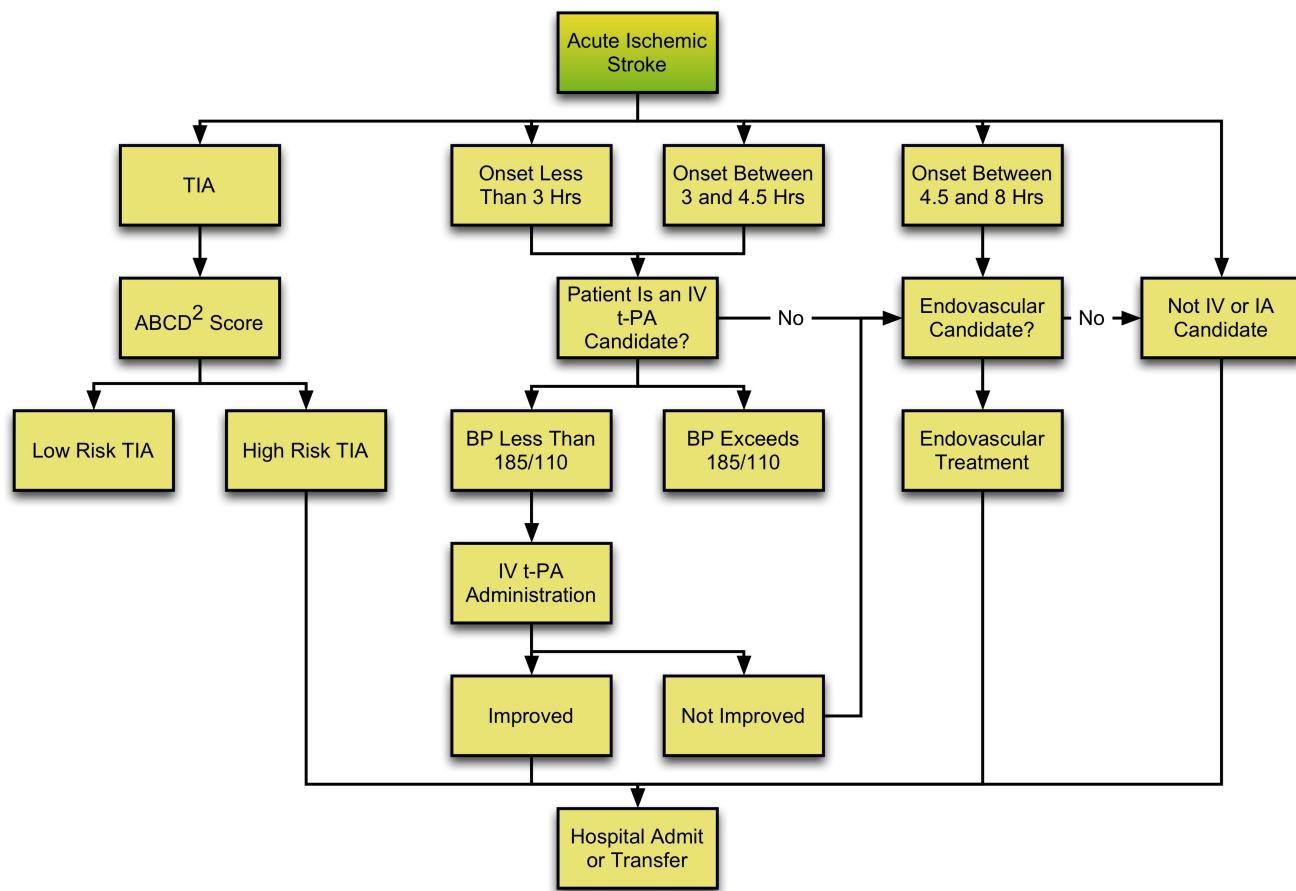
Emergency Neurological Life Support

All Protocols

Ischemic Stroke

Version: 1.1

Last Updated: 6/5/2013



[Checklist & Communication](#)



Checklist

- Labs: capillary glucose, CBC with platelets, PT/PTT, EKG, and beta-HCG for women
- IV access
- Supplemental oxygen to maintain saturation > 94%
- Activate stroke code system (if available)
- Determine NIHSS score

Communication

- Age
- Airway status
- Time of symptom onset
- NIHSS
- CT or MRI results



Acute Ischemic Stroke

Based on imaging and symptoms

The diagnosis of acute ischemic stroke is based on new onset focal neurological findings with an imaging study (CT or MRI of the brain) that shows no hemorrhage, or shows evidence of ischemic infarction.

In some centers, patients may be screened at the door when EMS arrives and then is taken directly to CT (or MRI) based on symptoms of facial droop, dysarthria, gaze preference, motor weakness or other focal findings.

If not completed already:

- STAT vital signs, capillary glucose, CBC with platelets, PT/PTT, EKG, and beta-HCG for women
- IV access
- Supplemental oxygen to maintain saturation >94% (hyperoxia may be detrimental in stroke, so no need for high flow oxygen)
- Activate stroke code system (if available)
- Stroke MD/team to evaluate patient with 5 minutes
- Determine NIHSS score

Topic Co-Chairs: Hartmut Gross, MD Gene Sung, MD



Administer IV t-PA

Start IV t-PA infusion

After placing 2 peripheral IV lines:

- Weight the patient; do not estimate body weight.
- Mix (do not shake) 0.9 mg/kg t-PA, total dose not to exceed 90 mg.
- Give 10% of the total dose of t-PA by bolus, then infuse the remaining dose over 1 hour.

Footnote:

As t-PA is dispensed in 50 and 100 mg bottles, it is suggested to draw off and discard any excess tPA to avoid accidental infusion of excess t-PA.

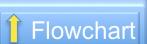


Endovascular treatment

Consider IA thrombolysis or thrombectomy

If the patient has a large vessel occlusion (MCA, intracranial ICA, basilar or vertebral artery) and is within an 8-hour time window, IA treatment may be helpful. Large vessel occlusion can be suspected by seeing a hyperdense sign (clot within the vessel) on non-contrast CT imaging but this sign is insensitive. CTA or MRA is diagnostic, as is conventional angiography.

- Contact the neurointerventional physician on call; if the treating hospital does not have this capability, consider transfer to a comprehensive stroke center
- Some hospitals use CT perfusion or MR perfusion techniques to select appropriate patients for intervention (ischemic penumbra)



Hospital Admission & Transfer

While waiting for ICU bed

After IV, IA or no specific treatment consider the following initial admission orders:

- Neuro check q30 min x 6 hrs., then q1 hr.
- Oxygenation to keep O₂ sat > 94%
- BP check q 15 min x 2 hrs., then q 30 min x 6 h, then q 1h x 16 h
- BP-after reperfusion treatment keep <180/105 (Note: this is lower than pretreatment values); if no t-PA given, keep BP <220/120
- Bedside swallow test (30 mL water PO) before anything else PO
- Keep glucose <140, consider insulin drip
- IVF (NS) to keep euvoolemia
- Monitor for A-fib
- Treat fever sources with antipyretics

If t-PA was administered:

- avoid indwelling urinary catheter, nasogastric tubes, intra-arterial catheters for 4 hours; do not give anticoagulant/antiplatelet therapy for 24 hours; repeat head CT or MRI at 24 hrs. before starting anticoagulant/antiplatelet meds

Watch for complications of t-PA, including

- Airway obstruction due to angioedema- consider rapid intubation
- Hemorrhage- stop t-PA
- Sudden deterioration in mental status- see below
- Severe hypertension or hypotension- may be signs of ICH or systemic hemorrhage

A sudden decline in neurological exam during or following t-PA administration may be due to an intracranial hemorrhage. This is often accompanied by a marked rise in blood pressure; however, a marked rise or fall in blood pressure alone may signal an ICH. Do the following immediately:

- STOP t-PA infusion
- Obtain STAT head CT scan
- Notify your neurosurgeon on call; if not available begin the process to transfer the patient to a facility with neurosurgical capability once the CT scan results are available
- Stat labs: PT, PTT, Platelets, fibrinogen, type and cross 2-4 unit PRBCs
- Give the following:
 - 6-8 units of cryoprecipitate
 - 6-8 units of platelets
 - Consider 40-80 mcg/kg of recombinant Factor VIIa while waiting for platelets and cryoprecipitate



Consider patient transfer

- if the treating hospital cannot provide the level of care for the patient (no ICU for example). Patient outcomes have been shown to improve if the patient is treated in a stroke center.
- if there is evidence of large vessel occlusion (CTA/MRA, hyperdense vessel sign on imaging; or clinical findings consistent with an MCA stroke) and the patient can arrive and be treated at the receiving hospital within 8 hours of symptom onset.



Low Risk TIA

ABCD² Score 0-3

Outpatient workup:

- Start on antithrombotic agent (ASA, clopidogrel 75 mg/day, or ASA/ extended release dipyridamole)
- Carotid imaging: ultrasound, CTA or MRA
- Consider echocardiography
- Consider long-term cardiac monitor*
- Smoking cessation
- Initiate statin

*- if ECG or rhythm strip shows atrial fibrillation consider starting anticoagulation (oral anticoagulant or low molecular weight heparin) or ASA depending on CHADS₂ score.



No

Blood pressure exceeds 185 over 110 mm Hg

- This is too high for IV t-PA administration and *requires* gentle reduction prior to initiating t-PA.
- Labetalol 10 mg IV every 10 minutes (consider doubling dose (i.e. 20, 40, 80) to max total dose of 150 mg. Start maintenance infusion.
- Nicardipine IV- start 5 mg/h, titrate up by 2.5 mg/h at 5- to 15-minute intervals, maximum dose 15 mg/h; when desired blood pressure attained, reduce to 3 mg/h

If BP falls below 185/110 mmHg, proceed to IV t-PA administration.

If BP proves refractory to the above, the patient is considered too high risk for intracerebral hemorrhage and should not be treated with t-PA. Continue to treat BP to keep less than 220/120 mmHg however.

Footnote:

While nitroglycerin paste (for patients with no IV access), labetalol, and nicardipine are recommended by the American Stroke association, other new drugs are available, but not yet studied in acute stroke management, including clevidipine. Be sure to initiate a drip as boluses will wear off and BP will likely return to its previous high levels.

Permissive hypertension is allowed for TIA, as it is for non-t-PA treated patients, up to 220/120 mmHg.



No improvement following t-PA

Within 1 hour no change in exam?

Often this is defined as no change in the NIHSS score.



Not low risk

TIA risk moderate or high, or unable to arrange timely outpatient work-up and follow-up

Admit for observation:

- Permissive hypertension (not to exceed 220/120 mm Hg)
- Start ASA, clopidogrel or ASA/extended release dipyridamole
- Carotid imaging: ultrasound, CTA or MRA
- Consider echocardiography
- Telemetry*
- Smoking cessation
- Initiate statin
- Consider keeping patient flat to improve brain perfusion (controversial)

*if ECG or rhythm strip shows atrial fibrillation consider starting anticoagulation (oral anticoagulant or low molecular weight heparin) or ASA depending on CHADS2 score.



Onset less than 3 hours

Time from stroke symptom onset is less than 3 hours

Time of onset is when the patient was last seen normal.

- If they can say when the first symptoms began, use that time
- If an observer can say when they saw the symptoms begin (excluding wake up), use that time
- If a patient awakens with a stroke, the time of onset is when they last went to bed

The time of onset is critical for using t-PA as the risk of intracerebral bleeding increases with increased time from stroke onset. If you cannot establish the time with certainty, most physicians will not treat with t-PA.

Check eligibility for on-label (US and elsewhere) use of IV t-PA:

- Diagnosis of ischemic stroke causing measurable neurological deficit.
- The neurological signs should not be clearing spontaneously.
- The neurological signs should not be minor and isolated.
- Caution should be exercised in treating a patient with major deficits.
- The symptoms of stroke should not be suggestive of subarachnoid hemorrhage.
- No head trauma or prior stroke in previous 3 months.
- No myocardial infarction in the previous 3 months.
- No gastrointestinal or urinary tract hemorrhage in previous 21 days.
- No major surgery in the previous 14 days.
- No arterial puncture at a noncompressible site in the previous 7 days.
- No history of previous intracranial hemorrhage.
- Blood pressure not elevated (systolic < 185 mm Hg and diastolic < 110 mm Hg).
- No evidence of active bleeding or acute trauma (fracture) on examination.
- Not taking an oral anticoagulant or, if anticoagulant being taken, INR < 1.7.
- If receiving heparin in previous 48 hours, aPTT must be in normal range.
- Platelet count <100 000 mm³.
- Blood glucose concentration < 50 mg/dL (2.7 mmol/L).
- No seizure with postictal residual neurological impairments.
- CT does not show a multilobar infarction (hypodensity >1/3 cerebral hemisphere).
- The patient or family members understand the potential risks and benefits from treatment.



Onset between 3 and 4.5 hours

Time from stroke onset is between 3 and 4.5 hours

Time of onset is when the patient was last seen normal.

- If they can say when the first symptoms began, use that time
- If an observer can say when they saw the symptoms begin (excluding wake up), use that time
- If a patient awakens with a stroke, the time of onset is when they last went to bed

The time of onset is critical for using t-PA as the risk of intracerebral bleeding increases with increased time from stroke onset. If you cannot establish the time with certainty, most physicians will not treat with t-PA.

In the US, t-PA is not yet approved for 3-4.5 use, although it is approved in Europe and Canada. The inclusion criteria are similar to those of < 3 hours, but are modified as follows:

- Age < 80 years
- No anticoagulant use, regardless of INR
- NIHSS < = 25
- No combined history of prior stroke and diabetes



Patient improves following t-PA

Measurable improvement within 1 hour?

Often this is defined as a lowering of the NIHSS score, and there is no clear consensus as to how much.



Patient is an IV t-PA Candidate

Is BP less than 185/110 mm Hg?

After reviewing the inclusion/exclusion criteria for IV t-PA use, the patient is eligible to receive the drug. Current blood pressure is the last inclusion criteria. If it is too high, the risk of ICH from t-PA is increased. Steps can be taken to lower blood pressure so as to make the patient eligible for t-PA.



Patient is not an IV t-PA or IA treatment candidate

Neither IV t-PA or IA intervention is appropriate

Common exclusions for IV t-PA are time (duration > 4.5 hours), and contraindications to t-PA (recent surgery, current bleeding at a non-compressible site, etc.), and large area of infarction already present on the brain imaging study (> 1/3 of the MCA territory).

IA exclusions include lack of large vessel occlusion on CTA or MRA, lack of consent from the patient or surrogate, or large area of infarction already present on the brain imaging study. If IA intervention is not available at the treating hospital, but there is clinical or radiographic evidence of a large vessel occlusion, consider rapid transfer to a facility with this capability.



Symptom onset between 4.5 and 8 hours

Outside IV t-PA window

Beyond 4.5 hours, IV t-PA is associated with intracerebral hemorrhage. IA therapies may be helpful in this time window (and earlier as well).



The ABCD² Score

What is the predicted risk for stroke?

The ABCD² score is an ordinal scale that provides risk prediction of stroke following the TIA. It is scored as follows:

ABCD ² Element	Points
Age > 60 years	1
Blood Pressure ≥ 140/90 on initial evaluation	1
Clinical Features	
Speech disturbance without weakness	1
Unilateral weakness	2
Duration of symptoms	
10-59 minutes	1
60 minutes or greater	2
Diabetes mellitus in patient's history	1
Total Score	0 - 7

The following is the estimated risk (%) of a stroke occurring within various time ranges:

Total Risk	ABCD ² Score	2 day	7 day	90 day
Low	0-3	1.0	1.2	3.1
Moderate	4-5	4.1	5.9	9.8
High	6-7	8.1	12	18

Ref:Cucchlara B et al, Ann Emerg Med 2008, 52:S27-39

Based on this risk stratification some physicians choose to admit high-risk patients and discharge low risk, and controversy exists about moderate risk patients.



TIA

Symptoms have completely resolved

Diagnosis of TIA (transient ischemic attack) is based on new onset of focal neurological symptoms that are explainable by a vascular cause (i.e. arterial occlusion of a single or group of arteries adequately explain the patient's signs and symptoms) and these signs and symptoms resolve within 24 hours.



Yes

BP is less than 185/110 mmHg

The patient is eligible for IV t-PA.
Place two peripheral IV lines.

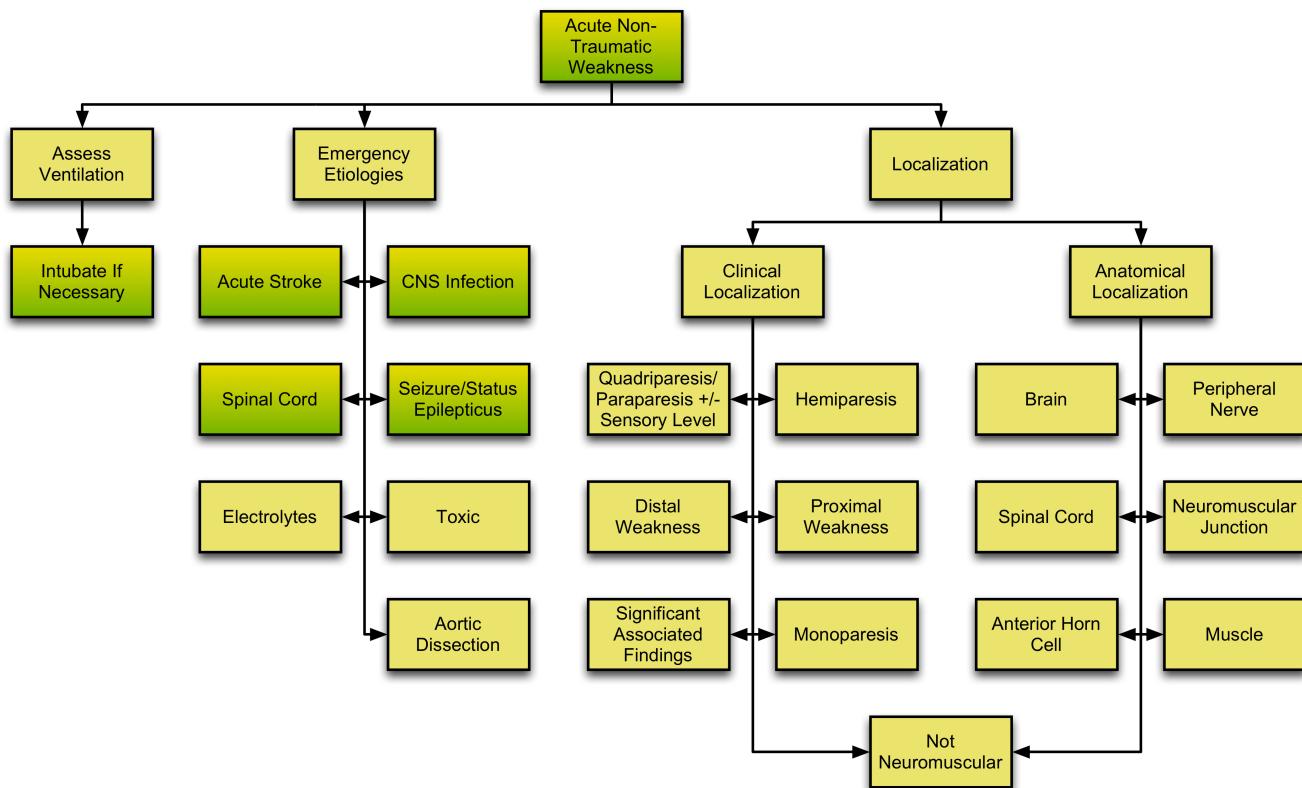
Acute Non-Traumatic Weakness

All Protocols

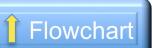
Emergency Neurological Life Support

Version: 1.1

Last Updated: 5/23/2013



[Checklist & Communication](#)

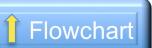


Checklist

- Assess airway, breathing, and circulation
- Characterize the weakness by detailed exam
- Build an initial differential diagnosis
- Consider emergency causes
- Initial labs: glucose, electrolytes, Ca, Mg, PO4, Bun, Cr, LFTs, PT,PTT, Platelets
- Special labs: TFTs, CPK, ESR
- Relevant imaging

Communication

- Cause of weakness if known; differential diagnosis if not known
- Airway status and any respiratory issues
- Salient history and exam findings
- Relevant labs and imaging (if done)
- Treatments provided



Acute Stroke

Within the time window?

If the patient has signs and symptoms consistent with acute stroke, especially if the patient is within the time window for thrombolysis, see the emergency evaluation of [Acute Stroke](#).



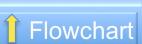
Acute Weakness

Patients presenting with any form of new weakness

This topic provides an organized approach to the patient with new weakness not associated with or caused by trauma. If the patient has experienced trauma follow the links to (Hyperlink to ENLS protocols [Traumatic Brain Injury](#) and [Traumatic Spine Injury](#) where appropriate.

Based on the pattern of weakness one can decide the degree of urgency, the need for ventilatory support and whether there are time-sensitive treatments necessary to consider.

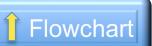
Topic Co-Chairs: Michael Cadogan, MD Eelco Wijdicks, MD Contributors: Chris Nickson, MD Oliver Flower, MD



Anatomical Localization

Based on the location of nervous system pathology

Understanding the cause of weakness can be aided by localizing anatomically, since diseases are often specific for each anatomic region. The neurological examination greatly aids the localization of weakness by anatomic means. The protocol figure at the beginning of this tutorial breaks down anatomic regions into the brain and spinal cord, the anterior horn cell, the peripheral nerve, neuromuscular junction (NMJ), and muscle. Diseases of the brain and spinal cord produce "UMN weakness," meaning disruption of descending motor axons or cell bodies that innervate the LMN (the anterior horn cell, peripheral nerve, and NMJ). After performing a neurological examination, refer to the table below to ascribe the appropriate anatomic localization. The key features to focus on are the presence or the absence of sensory signs (loss of sensory modality) or symptoms (complaints of numbness or tingling). If sensory signs/symptoms are absent, peripheral nerve is eliminated, and central nervous system processes are reduced in likelihood. Anterior horn cell causes are principally Lou-Gehrig's disease (ALS) and polio, neither of which have acute treatments. Reflexes are helpful to determine among the remaining causes that are most likely to occur. In general, lesions of the brain and spinal cord and the NMJ are the most emergent causes to consider, as there are specific treatments for some of these diseases (acute stroke and spinal cord compression) or public health concerns (botulism).



Anterior Horn Cell

Alpha motoneuron

Pattern of Weakness

- Proximal and distal, fasciculations are prominent
- Sensory Loss
- Absent Reflexes
- Decreased if muscle bulk is severely decreased; increased in ALS
- Acute Etiologies
- ALS, polio



Brain

Cerebral cortex, brainstem and spinal cord

Pattern of Weakness

- Distal > proximal, extensors > flexors, hemiparesis or single limb

Sensory Loss

- May be present depending on whether sensory tracts or cortex are involved
- Elevated during acute brain insult; however, reflexes may be decreased but later increase

Acute Etiologies

- See ENLS protocols [Acute stroke](#), [Subarachnoid Hemorrhage](#), and [Status Epilepticus](#)
- Hypertensive encephalopathy



Clinical Localization

Based on the neurological examination

After performing the above neurological examination, consider the pattern of weakness. Specifically, are all 4 limbs weak and there is sensory loss (quadripareisis), half the body weak (hemiparesis), one limb weak (monoparesis), the distal extremities only are weak (distal weakness), or the proximal muscles are weak (proximal weakness)? Also, are there any significant associated findings?

Accurately defining the presenting complaint helps generate a focused differential diagnosis. A good clinical history is essential, as the examination may be difficult or unreliable in the obtunded or confused patient. However, it should be possible to elicit whether the deficit is unilateral or bilateral, which anatomical region is affected, and whether there is a sensory deficit. With a cooperative patient, it should also be possible to establish whether the deficit is symmetrical or asymmetrical, and proximal or distal. Note that it is important to attempt to differentiate between upper motor neuron (UMN) and lower motor neuron (LMN) lesions in the acute setting, though this may be difficult in some situations. In well-established UMN lesions, hyperreflexia (brain and spinal cord), increased tone, and a positive Babinski sign are seen. In comparison, LMN lesions (from the anterior horn cells to the muscles) cause a flaccid, areflexic weakness and, with time, atrophy and fasciculations. However, in the acute phase, UMN lesions may mimic an LMN lesion: flaccid paralysis, normal or reduced tone, and unreliable reflexes. There is often not enough time for atrophy to be evident, and fasciculations are rarely seen.



CNS Infection

Consider meningitis or encephalitis as a cause. See ENLS protocol [Meningitis and Encephalitis](#).



Distal Weakness

- Vasculitic neuropathy
- Toxin induced peripheral neuropathy
- Nerve compression syndromes

Distal weakness is weakness mainly affecting the extremities. It is typically caused by peripheral neuropathies that often present along with sensory symptoms. Distal weakness affects the hands and feet, causing the patient to drop objects or develop gait disturbance due to foot drop. The pattern of weakness and history are of great significance. Of the many types of peripheral neuropathy, vasculitic and toxin induced are the most likely to produce an acute weakness. It may also be produced by local nerve compression syndromes (e.g., carpal tunnel syndrome that predominantly affects peripheries, causing both sensory and motor symptoms).



Electrolyte Disturbance

Glucose, K, PO4

Acute hypoglycemia, hypokalemia, hypophosphatemia or other electrolyte disturbances suggesting other organ dysfunction should be attended to now.



Emergency Etiologies

Exclude these time-sensitive emergency causes first

There are several time-sensitive causes of acute weakness that should be excluded quickly before moving on to a more comprehensive localization of the cause of weakness. Consider each of the causes to the right before proceeding to localization.

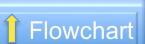


Hemiparesis

Half body weakness

- See ENLS protocols [Acute stroke](#), [Intracerebral Hemorrhage](#), or [Subarachnoid Hemorrhage](#)
- Intracranial mass
- See ENLS protocol [Meningitis and Encephalitis](#)
- Hypoglycemia/hyperglycemia
- Postictal Todd's paresis
- Hemiplegic migraine
- Brown-Sequard syndrome

Hemiparesis is acute weakness involving only one side of the body. While acute hemiplegia is most commonly due to an ischemic stroke , other differentials must be considered, as management of these differentials vary. The history and demographic of the patient is likely to narrow the diagnosis, and examination findings provide further clues. A blood glucose level and a non-contrast head computed tomography are part of the initial workup.



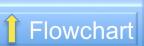
Localizing the Cause of Acute Weakness

This will help determine the cause

Perform a neurological examination on the patient that includes:

- Deep tendon reflexes
- Power testing of proximal versus distal muscles and flexors versus extensor muscles, noting symmetry between sides
- Judge diaphragmatic and chest wall muscle strength to determine if there is any respiratory insufficiency (count to 20 [external intercostal muscles], and negative inspiratory force [diaphragm])

After performing this focused neurological examination, determine the pattern of weakness.

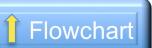


Monoparesis

Weakness of a single limb

- See ENLS protocol [Acute Stroke](#)
- Intracranial mass
- Postictal Todd's paresis
- Nerve compression syndromes
- Diabetic lumbosacral radiculoplexus neuropathy
- Acute poliomyelitis

Monoparesis refers to paralysis of a single muscle, muscle group, or limb. Acute paralysis involving a single limb may be caused by a central or a peripheral lesion. Historical and examination factors may help to localize the lesion. For example, sudden onset right arm weakness with an associated dysphasia is most likely to result from a central lesion, whereas wrist drop in the right hand, with hypoesthesia on the back of the hand following falling asleep with the arm over the back of a chair, results from a peripheral nerve compression syndrome. Poliomyelitis is rare, but can occur in the unvaccinated.



Muscle

Pattern of Weakness

- Proximal

Sensory Loss

- Absent Reflexes
- Normal unless muscle severely weak

Acute Etiologies

- Rhabdomyolysis



Need for Assisted Ventilation

Do you need to intubate this patient?

Assess the patient's airway and potential need for assisted ventilation. If any of the following general, subjective or objective findings are present, consider intubation.

General:

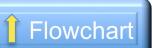
- Increasing generalized weakness?
- Dysphagia?
- Dysphonia?
- Dyspnea on exertion and at rest?

Subjective:

- Rapid shallow breathing
- Tachycardia
- Weak cough
- Staccato speech
- Use of accessory muscles
- Abdominal paradox
- Orthopnea
- Weakness of trapezius and neck muscles- inability to lift head
- Single-breath count: count to 20 in single exhalation (FVC 1.0 L is roughly equal to counting from 1 to 10)
- Cough after swallow

Objective:

- Vital capacity < 1 L or 15 mL/kg, or 50% drop in VC over course of evaluation
- Negative inspiratory force < 15 mmHg
- Maximum inspiratory pressure < 30 cm H₂O
- Maximum expiratory pressure < 40 cm H₂O
- Nocturnal desaturation
- Rising P_aCO₂ (note- this is a late finding)
- Hypoxemia
- Unconscious state



Neuromuscular Junction

Pattern of Weakness

- First in eye muscles, neck extensors, pharynx, diaphragm, followed by more generalized weakness
- Sensory Loss Absent
- Reflexes normal, decreased if muscle is paralyzed

Acute Etiologies

- Botulism, tick bite, organophosphate



Not Neuromuscular Weakness

Consider psychiatric cause

Some disease states may produce symptoms of generalized weakness or fatigue that does not have a neuromuscular basis. These may be medical emergencies in their own right meriting urgent specific treatment.

Consider:

- Any severe medical illness can have weakness as a symptom, but generally these will become clinically obvious during the patient's evaluation

Diagnoses of exclusion:

- Malingering
- Conversion disorder
- Chronic fatigue syndrome
- Anxiety disorders
- Fibromyalgia



Other Urgent Causes

Consider:

- Shock
- Myocardial infarct
- Addisonian crisis



Paraplegia from Aortic Dissection

Spinal cord infarct

Acute aortic dissection can close the artery of Adamkiewicz that supplies the anterior spinal artery to the mid thoracic and lumbar spinal cord. The patient will have an anterior spinal artery syndrome (paraplegia with loss of pain and temperature sensation below the lesion but preservation of light touch). Assess distal lower extremity pulses and consider CTA, ultrasound or other techniques to rule out aortic dissection if the patient has an anterior spinal artery syndrome.



Peripheral Nerve

Pattern of Weakness

- In the distribution of the nerve, or diffusely present as stocking/glove weakness
- Sensory Loss Present
- Reflexes Decreased

Acute Etiologies

- Guillain-Barre syndrome, vasculitis



Proximal Weakness

- Acute myopathy
- Guillain-Barre syndrome
- Acute diabetic lumbosacral radiculoplexus neuropathy(DLRN)
- Myasthenia gravis
- Acute West Nile virus associated paralysis
- Lambert-Eaton myasthenic syndrome (LEMS)

Proximal weakness is weakness predominantly affecting the hip or shoulder girdle musculature. Acute proximal weakness classically presents with difficulty rising from a chair or brushing hair. The most common cause is myopathy. Less common causes include LEMS and myasthenia gravis. DLRN may be the presenting feature of diabetes mellitus and is also important to consider. While poliomyelitis is very rare in western countries, it remains endemic elsewhere. West Nile virus, with similar semiology as acute poliomyelitis, is more common in the United States and Europe.



Quadripareisis or Paraparesis with or without Sensory Level

Suggests spinal cord lesion

Quadripareisis/Paraparesis ± Sensory Level

- See ENLS protocol [Spinal Cord Compression](#)
- Spinal cord infarction

Transverse myelitis Quadripareisis/paraparesis is symmetrical weakness of either all four limbs (quadripareisis) or legs (paraparesis), characteristically with a sensory level. Non-traumatic spinal cord injury may occur from compression (e.g., epidural abscess, hematoma, expanding tumor, prolapsed intervertebral disc), ischemia (spinal cord infarction), or inflammation (transverse myelitis). In the acute phase, a flaccid paralysis below the level of cord injury is typically seen, with an accompanying corresponding sensory level, although there is considerable variation. Neurological examination should localize the lesion in patients with acute paraplegia or quadriplegia. Sensory abnormalities localize in the vertical plane (cervical, lumbar, or sacral) and, when combined with other long tract signs, point to localization in the horizontal plane (extradural, intradural, or intramedullary). Key sensory levels (T4 nipple, T10 navel) should be used.



Significant Associated Findings

Other finding that may make the diagnosis clear

Certain constellations of symptoms and signs can make specific, often unusual diagnoses more likely. The Table below lists some of these, and each is elaborated further in separate tables. Stroke syndromes may also have characteristic patterns which are too numerous and varied to discuss here. However, findings such as aphasia, agnosia, apraxia, and neglect with acute weakness or sensory signs should prompt consideration of acute stroke.

Acute tetraplegia, facial muscles paralyzed except eyes, clear sensorium

- Locked-in syndrome (also consider residual neuromuscular blockade) Fatigable weakness in eyelids and extra-ocular muscles with variable weakness elsewhere and no sensory symptoms
- Myasthenia gravis

History of animal bite, descending paralysis, and possible coagulopathy, rhabdomyolysis, and shock

- Envenomation

Severe, refractory hypertension with headache and transient, migratory neurological non-focal deficits

- Hypertensive encephalopathy

Ascending paralysis following upper respiratory mild viral illness/infection

- Guillain-Barre syndrome

Descending symmetrical paralysis with a clear sensorium and no fever

- Botulism

Weakness with prominent cholinergic signs and symptoms

- Organophosphate toxicity

Heavy metal exposure, prominent gastrointestinal symptoms, then multi-organ failure

- Heavy metal toxicity

Episodic proximal weakness with family history

- Periodic paralysis

Heliotrope rash with proximal weakness

- Dermatomyositis

Abdominal pain, proximal weakness, psychiatric symptoms, red urine

- Acute intermittent porphyria

Tick bite followed by ascending paralysis

- Tick paralysis



Special considerations for Intubation

Consider

Special consideration for Intubation:

- One should use a rapid sequence intubation, however avoid use of succinylcholine if there is evidence of underlying progressive neuromuscular disease (precipitates acute hyperkalemia)
- Consider non-invasive assisted ventilation as a temporizing measure
- Prepare atropine/glycopyrrolate, fluids and vasopressors if there is evidence of autonomic instability

See ENLS protocol [Airway, Ventilation and Sedation](#).



Spinal Cord Compression

Non traumatic cause

Pattern of Weakness

- Distal > proximal, extensors > flexors, paraparesis, quadriplegia, rarely hemiparesisSensory Loss
- May be present depending on whether sensory tracts or cortex are involved; loss of sensation below a certain spinal level is diagnostic

Reflexes

- Elevated during acute brain insult; however, reflexes may be decreased but later increaseAcute Etiologies
- Epidural abscess, tumor, spinal cord infarct

Acute spinal cord compression of non-traumatic cause should be suspected if the patient has weakness of both legs or both arms and legs with intact mental status and cranial nerves, especially if they have a history of cancer or complaint of back or neck pain. See ENLS protocol [Spinal Cord Compression](#). If there is any sign of trauma, see ENLS protocol [Traumatic Spine Injury](#).



Status Epilepticus or Seizure

Postictal or non-convulsive status or Todd's Paresis

Patients who are comatose, or encephalopathic, may be post-ictal. A patient with focal neurological findings (typically hemiparesis) may have a Todd's paralysis caused by a generalized seizure in a brain with prior injury. Also, a patient may be having non-convulsive status epilepticus. See the ENLS protocol [Status Epilepticus](#).



Toxic Cause

Any toxin exposure?

Consider organophosphate or carbon monoxide exposure among others.

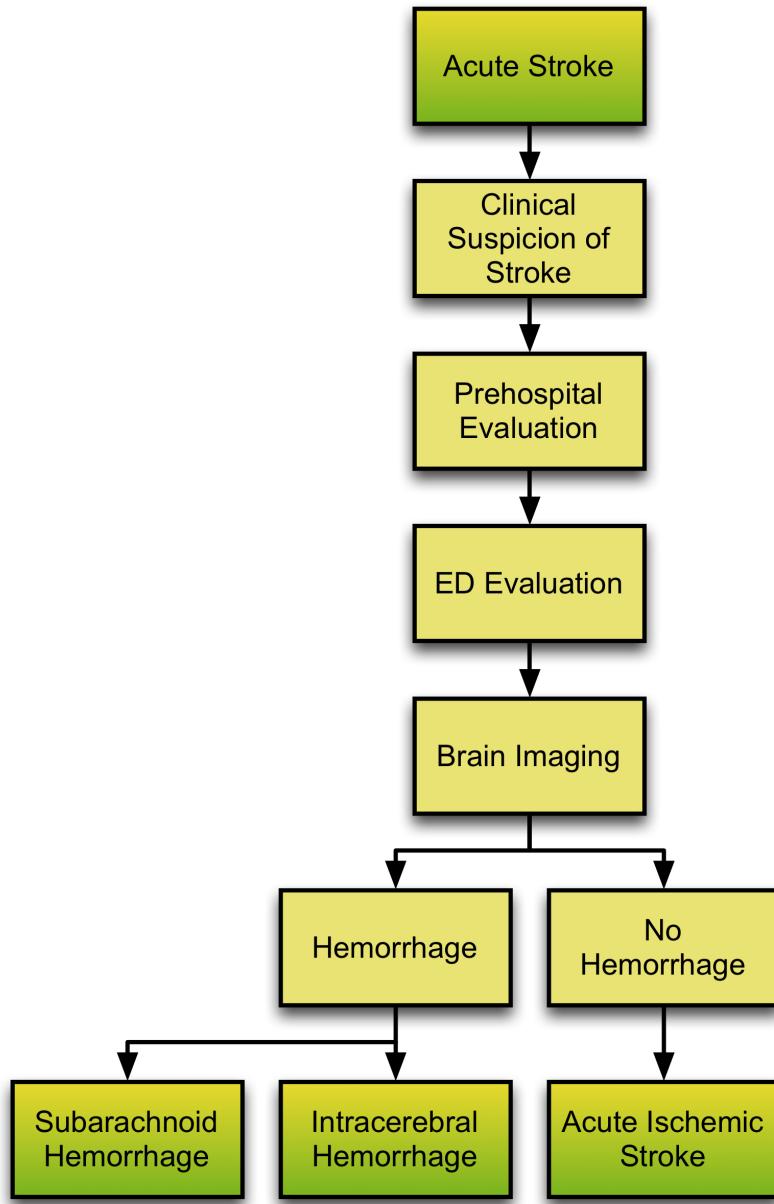
Emergency Neurological Life Support

All Protocols

Acute Stroke Initial Assessment

Version: 1.0

Last Updated: 5/23/2013



[Checklist & Communication](#)



Checklist:

- Establish time of onset (time last seen normal)
- Vital Signs
- Imaging
- NIHSS
- GCS
- Labs: CBC, Platelets, Chemistries, PT/PTT, glucose

Communication:

- Age
- Time of Onset
- NIHSS
- Imaging findings: primary hemorrhage, ischemia, or normal scan



Imaging does not show hemorrhage

CT or MRI imaging are either normal, or show an ischemic infarct

CT or MRI imaging is either normal, or shows an ischemic infarct.
See ENLS protocol [Ischemic Stroke](#).



Imaging Shows Hemorrhage

CT or MR imaging show a hemorrhage

CT or MRI imaging reveal hemorrhage in the brain accounting for their neurological findings. Now determine whether the blood is in the subarachnoid space or within the brain itself (including ventricle).



Intracerebral Hemorrhage

Most of the blood is within the brain parenchyma

If there was clear evidence of head trauma, the blood may be simply due to the trauma alone. If so, refer to ENLS protocol [Traumatic Brain Injury](#).

If there is no evidence of head trauma, refer to the ENLS protocol [Intracerebral Hemorrhage](#).



Step 1

Clinical suspicion of stroke

Out of hospital:

- Acute onset focal neurologic symptoms
- 911 EMS services alerted



Step 2

Prehospital evaluation

Prehospital Evaluation:

- ABCs
- Stroke screening tool
- Time last known normal
- Medication list
- Consider triage to stroke center



Step 3

Primary Emergency Department Assessment

Emergency department evaluation:

- ABCs
- Focused neurologic exam (5 minutes): GCS, NIHSS
- History: medications, atrial fibrillation
- Labs: CBC, PT/PTT, glucose, chemistry panel



Step 4

Cerebrovascular Imaging

Imaging:

- CT or MRI - CT is usually faster
- Consider "Stroke CT" that includes non-contrast head CT, CTA (angiography) of the neck and brain, and CT perfusion of the brain
- Consider MRI that includes MRA of head and neck, DWI and MR perfusion of the brain

Note: imaging inclusions and exclusions regarding t-PA administration are typically based on a non-contrast CT of the head alone.



Subarachnoid Hemorrhage

CT or MRI shows blood in the subarachnoid space

The predominance of blood is in the subarachnoid space. If there was clear evidence of head trauma, the blood may be simply due to the trauma alone. If so, refer to the ENLS protocol [Traumatic Brain Injury](#).

If the predominance of blood is in the subarachnoid space and there is no evidence of head trauma, the hemorrhage is likely due to a ruptured cerebral aneurysm. Refer to the ENLS protocol [Subarachnoid Hemorrhage](#).

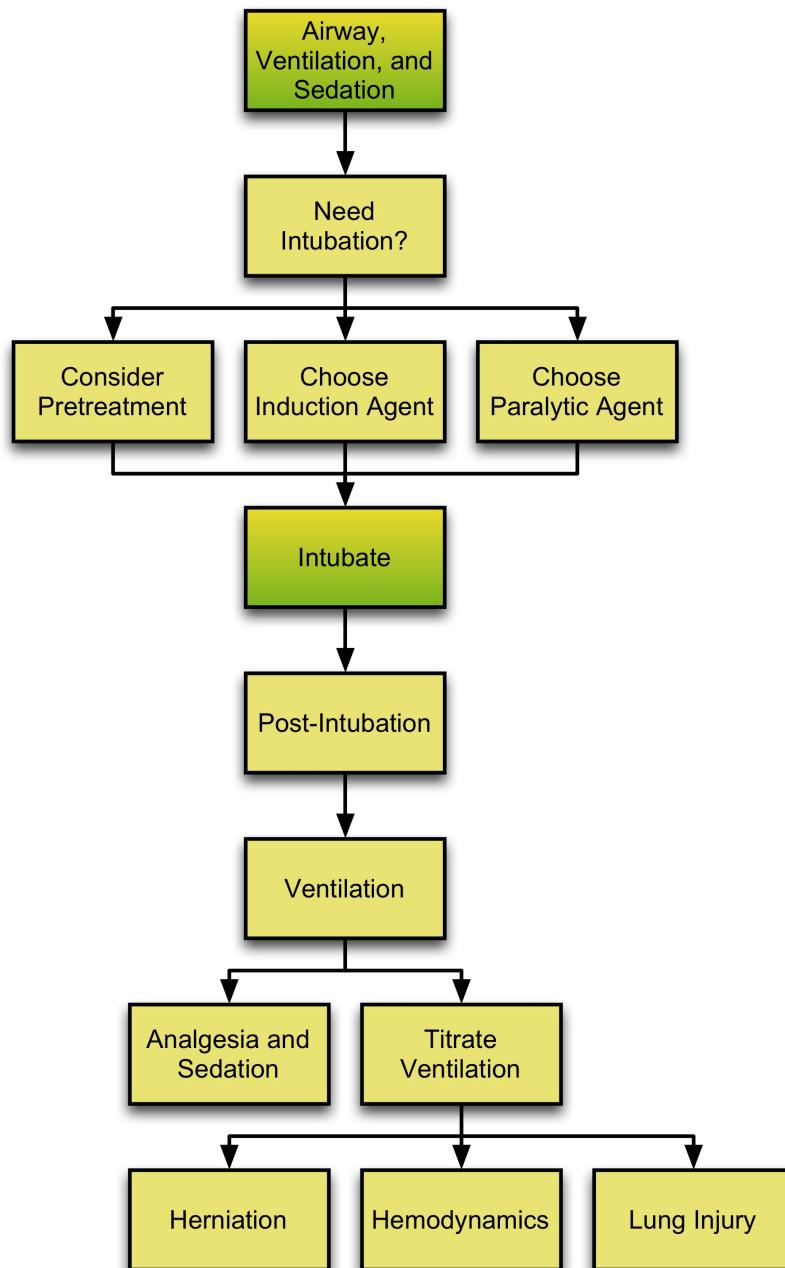
Emergency Neurological Life Support

All Protocols

Airway, Ventilation and Sedation

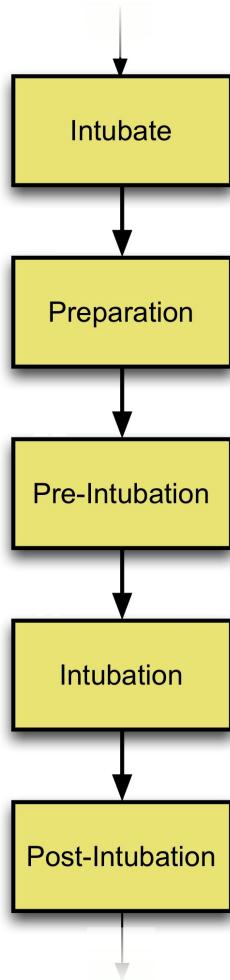
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[Checklist & Communication](#)

Intubation Sequence



Communication

- Mental status and exam prior to intubation
- Vitals pre and post intubation
- Ease of intubation
- ETT position confirmed?

Checklist

- Assess the need for intubation or non-invasive positive pressure ventilation
- Assess the endotracheal tube position
- Assess the need for analgesia and/or sedation in mechanically ventilated patients



Acute lung injury

Does the patient have ARDS?

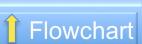
Patients with acute lung injury or ARDS should be ventilated with low tidal volumes (6 cc/kg IBW), adequate PEEP, and plateau pressure < 30 mmHg.

* Although, "Permissive hypercarbia" and tolerance of low SpO₂ or P_aO₂ targets are part of ARDSnet, in acute brain injury hypoxia and/or hypercarbia may be deleterious so is discouraged.

Oxygenation Goal: P_aO₂ 55-80 mmHg, or SpO₂ 88-95%.

Use minimum PEEP of 5 cm H₂O. Consider use of incremental F_iO₂/PEEP combinations such as shown below:

F _i O ₂	PEEP (cm H ₂ O)
0.3	5
0.4	5
0.4	8
0.5	8
0.5	10
0.6	10
0.7	10
0.7	12
0.7	14
0.8	14
0.9	14
0.9	16
0.9	18
1.0	18-24



Airway Ventilation and Sedation

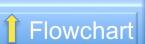
In patients with known or suspected neurological injury

Management of the airway, how to ventilate and if needed sedate a patient with suspected or known neurological injury requires skill and understanding of the underlying issues of cerebral herniation, elevated ICP and neuromuscular status surrounding chemical paralysis. This topic will provide an organized approach to assessing and establishing an airway, the initial ventilator settings and suggest methods to sedate an agitated patient in whom you may not fully know the cause of their neurological condition.

In cases of suspected elevated ICP (coma, subarachnoid hemorrhage, TBI, hydrocephalus, etc.):

- Rapid sequence intubation is the preferred method of securing the airway in patients with suspected elevated ICP since it provides protection against the reflex responses to laryngoscopy and rises in ICP. The presence of coma should not be interpreted as an indication to proceed without pharmacological agents, or to administer only a neuromuscular blocking agent without a sedative/induction drug. Although the patient may seem unresponsive, laryngoscopy and intubation will provoke reflexes that elevate ICP unless appropriate pretreatment and induction agents are used.
- Outcomes in patients with intracranial catastrophes are related to the maintenance of both brain perfusion and oxygenation; consequently, close assessment and management of these two parameters is critical. Cerebral perfusion pressure (CPP) is the driving force for blood flow to the brain, and is measured by the difference between the mean arterial blood pressure (MAP) and the ICP: CPP = MAP - ICP. Clinical evidence of increased ICP include altered mental status plus unilaterally dilated pupil, bilaterally dilated and fixed pupils, and decerebrate posturing (N.B. decorticate posturing is not predictive of elevated ICP). It is generally recommended that the ICP be maintained below 20 mmHg, the MAP between 100-110 mmHg, and the CPP near 70 mmHg. It is generally recommended that the ICP be maintained below 20 mmHg, the MAP between 100-110 mmHg, and the CPP near 70 mmHg.
- Problems associated with elevated ICP may be compounded by many of the techniques and drugs used in airway management since they may cause further elevations of intracranial pressure. In addition, victims of multiple trauma may present with hypotension, thus limiting the choice of agents and techniques available.

Topic Co-Chairs: Andy Jagoda, MD David Seder, MD

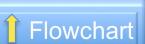


Analgesia and Sedation

Sedation and analgesia principles

- Assure patient comfort with analgesics
- Heavy sedation is employed ONLY for control of ICP, safety concerns including transport, or cerebral metabolic failure requiring control of CMRO₂
- Light sedation, using short acting agents only
- Minimal or NO sedation is preferred when above concerns are absent
- DO NOT allow medication-induced hypotension

The goal of analgesia and sedation in the neurocritically ill is typically to maintain comfort with minimal disruption of the neurological examination, ventilator weaning, and rehabilitation activity. Many short-acting sedatives have prolonged activity when continuously infused, and neurointensivists often choose short acting analgesic infusions that can be discontinued for neurological examination, supplemented with boluses of short or intermediate acting sedatives. Occasionally, ICP crisis, status epilepticus, or other neurological crises require a state of deep, continuous sedation. This is often achieved by high dose infusions of benzodiazepines or barbiturates. High dose propofol (>80 µg/kg/min) must be avoided due to the dose-dependent risk of Propofol Infusion Syndrome (PRIS).



Assess Airway

LEMON and MOANS

Critically ill patients are at risk of airway compromise; consequently, early management of these patients must include an assessment of their airway regarding ease of intubation and ease of bag mask ventilation. This assessment enables strategic planning and efficient resource utilization.

The "LEMON" mnemonic helps to predict the difficult airway:

L = Look

E = Evaluate the mouth opening and airway position

M = Mallenpati score

O = Obstruction

N = Neck mobility.

The "MOANS" mnemonic predicts ease of bag mask ventilation.

M = Mask seal not good, e.g. beard, facial deformity, etc.

O = Obesity (difficult ventilate), 3rd trimester pregnancy, or obstruction e.g. neck swelling, angioedema, hematomas, cancer, etc.

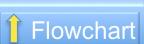
A = Age, elderly, loss of muscle tone to support the upper airway

N = No teeth (no teeth causing caved in face)

S = Stiff lungs - upper airway obstruction - exacerbation of asthma, COPD, etc.

Patients who are deemed to be difficult should alert the clinician to the need for appropriate back-up either in terms of skills (call anaesthesia) or devices (set up the flexible fiberoptic intubating device and set-up for a cricothyrodiotomy).

Assessment of ease of bag mask ventilation is critical and if deemed difficult mandates access to an intrapharyngeal ventilation device, e.g., LMA.



At the Time of Intubation

Induction and paralysis

Give:

- etomidate 0.3 mg/kg or ketamine 0.5-1 mg/kg
- Succinylcholine 1.5 mg/kg or rocuronium 1.2 mg/kg

Induction should be performed using an agent that will not adversely affect CPP. Etomidate is a short-acting imidazole derivative that is the most hemodynamically stable of all commonly used induction agents. Its ability to decrease CMRO₂ and ICP in a manner analogous to that of sodium thiopental and its remarkable hemodynamic stability make it the drug of choice for patients with elevated ICP. Propofol is an alternative.

Thiopental confers some cerebroprotective effect via decreasing the basal metabolic rate of oxygen utilization of the brain (CMRO₂); it also decreases cerebral blood flow, thus decreasing ICP. However, thiopental is a potent venodilator and negative inotrope thus it has a tendency to cause hypotension and reduce CPP, even in relatively hemodynamically stable patients.

In the past, Ketamine was generally avoided in patients with known elevations in ICP as it was believed it could elevate the ICP further. Recent evidence has disputed this viewpoint. However, in hypotensive patients, ketamine's superior hemodynamic stability may argue for its use.

Either a depolarizing or a nondepolarizing agent can be used to paralyze the patient after induction. Succinylcholine remains the drug of choice for management of patients with elevated ICP because of its rapid onset and short duration. Though it has been associated with transient increases in ICP, this is not considered significant and therefore not a contraindication to its use. Premedicating with a defasiculating dose of a nondepolarizing agent is no longer considered worthwhile. An alternative would be to use a competitive neuromuscular blocking agent. Rocuronium is the competitive agent most suited to this strategy due to its rapid onset and consistent achievement of intubating conditions.



BP and Cardiac Output

Positive pressure ventilation can cause hypotension

Ventilation and (mean) airway pressure may affect blood pressure and cardiac output, especially in a hypovolemic patient:

- The effect is minimized if you adequately replete intravascular volume
- Apply PEEP, increased minute ventilation, and increased respiratory rate while carefully monitoring BP and CO/CI

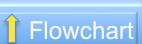


Brain Herniation or High ICP

Should you hyperventilate?

In the setting of clinical or radiographic brain herniation:

- Minimize the duration of hyperventilation as much as possible by employing other means of ICP control
- Prolonged hyperventilation requires the use of CNS metabolic monitoring to verify the adequacy of cerebral blood flow: jugular venous oximetry ($S_{jv}O_2$), Brain tissue oxygen ($P_{bt}O_2$) monitoring, or cerebral microdialysis
- See ENLS protocol [Elevated ICP](#) for additional details.
- Routine hyperventilation for control of elevated ICP is discouraged as it may exacerbate cerebral ischemia:
- If hyperventilation is used, do not lower P_aCO_2 below 35 mm Hg, and [consider other methods for control of ICP](#).

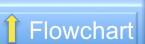


Choose Induction Agent

For rapid sequence induction

Induction is performed using an agent that will not adversely affect cerebral perfusion pressure (CPP)

- Fentanyl: At doses of 2-3 µg/Kg, attenuates the reflex sympathetic response (RSR) associated with intubation, and is administered as a single pretreatment dose over 30-60 seconds in order to reduce chances of apnea or hypoventilation before induction and paralysis. Is generally not used in patients with incipient or actual hypotension, or those who are dependent on sympathetic drive to maintain an adequate blood pressure for cerebral perfusion.
- Etomidate: Short-acting imidazole derivative that provides sedation and muscle relaxation with minimal hemodynamic effect. Considered the most hemodynamically neutral of all commonly used induction agents and a drug of choice for patients with elevated ICP.
- Propofol: At a dose of 2 mg/kg intravenous (IV) push, is an alternative, but is also a potent vasodilator that routinely causes hypotension and often requires concurrent vasopressor administration to maintain CPP.
- Thiopental: At a dose of 3mg/kg IV push, confers cerebroprotective effect by decreasing the basal metabolic rate of oxygen utilization of the brain (CMRO₂) and CBF, thus decreasing ICP. However, is a potent venodilator and negative inotrope with a strong tendency to cause hypotension and reduce CPP, even in relatively hemodynamically stable patients.
- Ketamine: Hemodynamically neutral dissociative agent administered at 2 mg/kg IV push. In the past, was generally avoided as an agent believed to raise ICP. However, recent evidence suggests when sedation is provided concurrently, may be safe in patients with elevated ICP, and its hemodynamic profile argues for more widespread use.



Choose Paralytic Agent

Succinylcholine vs. Rocuronium

For rapid sequence intubation, it is desirable to have rapid onset of muscle paralysis. Succinylcholine is the drug of choice unless there are contraindications where rocuronium is useful.

- Succinylcholine (SCh): Depolarizing agent that remains the neuromuscular blockade agent of choice for intubation of acutely ill neurological patients with elevated ICP, due to its rapid onset and short duration of action. Although it has been associated with transient increases in ICP, the effect is not considered clinically significant. However, the neurologically ill are at higher risk for succinylcholine-induced hyperkalemia, and clinicians should consider that patients with disuse atrophy may have severe hyperkalemia following administration of a depolarizing agent. This includes patients with prior brain or spinal cord injury but also those with as little as 24-72 hours of immobility, and patients with upper or lower motor neuron defects. Risk may be averted by instead using a non-depolarizing agent, such as rocuronium (at 1.2-1.4 mg/kg IV push) or the longer acting agents pancuronium and vecuronium (at 0.2 mg/kg IV push).



Consider Pretreatment

Pretreatment medication to blunt the sympathetic response

For patients who are normotensive, or hypertensive (SBP > 90 mmHg): At time intubation minus 3 minutes give:

- Lidocaine 1.5 mg/kg AND Fentanyl/remifentanyl 3 µg/kg over 30 sec

OR

- Lidocaine 1.5 mg/kg AND esmolol 1-2 mg/kg

For patients who are hypotensive (SBP ≤ 90 mmHg): At time intubation minus 3 minutes give:

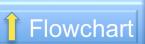
- Fluids
- Blood products
- Inotropes
- Pressors

Rationale: Hypotension may lead to cerebral hypoperfusion. A MAP of > 65 mm Hg should be maintained at all times throughout the peri-intubation. This may require small aliquots of vasopressors until definitive volume loading or blood product replacement is achieved.

When the airway is manipulated there are two responses that result in increased ICP: there is a reflex sympathetic response (RSR) which results in increased heart rate, increased blood pressure, and consequent increased ICP; in addition, there is a direct laryngeal reflex that stimulates an increase in ICP independent of the RSR. In the management of patients who are suspected of having an increased ICP, elevations in the ICP should be mitigated by minimizing airway manipulation, i.e., the most experienced person should perform the intubation, and pharmacologically using medications. The three commonly used pre-medications are lidocaine, fentanyl, and esmolol.

- Lidocaine's primary benefit is on attenuating the direct laryngeal reflex. There is mixed evidence that it actually mitigates the RSR.
- The short acting beta blocker, esmolol does control both heart rate and blood pressure responses to intubation. A dose of 2 mg/kg given three minutes before intubation has been shown to be effective. Unfortunately, in the emergency situation, the administration of a beta-blocking agent, even one that is short acting, may be problematic. Although esmolol is consistent and reliable for mitigation of RSRL in elective anesthesia, it is generally not used for this purpose in the ED.
- Fentanyl at doses of 2 - 3 µg/kg attenuates the RSR associated with intubation. Although a full sympathetic blocking dose of fentanyl is 9-13 µg/kg, the recommended dose of fentanyl for RSI in emergency patients is 2-3 µg/kg and should be administered as a single pretreatment dose over 30 to 60 seconds in order to reduce chances of apnea or hypoventilation before induction and paralysis.

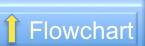
Fentanyl should not be administered to patients with incipient or actual hypotension, or those who are dependent on sympathetic drive to maintain an adequate blood pressure for cerebral perfusion.



Decision made to intubate

What is the patient's blood pressure?

Rapid sequence intubation is the preferred method of securing the airway in patients with suspected elevated intracranial pressure (ICP), since it provides protection against the reflex responses to laryngoscopy and rises in ICP. Keep the Cerebral Perfusion Pressure (Mean arterial pressure - ICP) between 80-100 mmHg throughout the process. If ICP is not known, assume an ICP of 20 mmHg.



Does the patient need to be intubated?

Failure to oxygenate, ventilate, protect airway or anticipate deterioration

There are four commonly accepted indications to intubate a patient:

- Failure to oxygenate is generally driven by pulse oximetry though the clinician is reminded of its limitations, e.g., hypoperfusion, severe anemia, opaque nail polish.
- Capnometry provides a monitor for ventilation though it too has its limitations in trauma and does not always correlate with PCO₂, however it does provide a valuable tool for monitoring trends in the patient's ventilatory status.
- Ability to protect the airway is fundamental to minimizing risk of aspiration and its complications; this is best assessed by observing the patient's ability to spontaneously swallow or to swallow after suctioning.
- A gag reflex is an inaccurate method of assessing airway protection, however, if this is used it is best done with a suction catheter and not a tongue blade.

Anticipated course of the patient's airway status is the most inexact of the indicators to intubate a patient and requires that multiple factors be taken into consideration including location of the patient and resources available.



Initiate Volume Cycled Ventilation

Initial parameters

Initial volume cycled ventilation parameters:

- Tidal Volume 8 cc/kg (ideal body weight)
- Respiratory rate: match pre-sedation spontaneous respiratory rate, or set to 12 breaths per minute
- Inspired fraction of oxygen: 1.0; titrate rapidly to lowest F_iO_2 that will maintain S_pO_2 at 95-99%
- Start capnometry monitoring (ETCO₂)
- Pulse oximetry

This applies to several neurological emergencies, including:

- Traumatic brain injury
- Subarachnoid hemorrhage
- Large intracerebral hemorrhage
- Ischemic stroke with airway compromise or coma
- Hydrocephalus
- Intracranial tumor with depressed mental status
- Suspected high ICP and depressed mental status
- Diffuse cerebral edema
- Status epilepticus

Goals of mechanical ventilation:

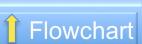
- Mechanical ventilation must be carefully titrated to maintain physiological homeostasis. Because PCO₂ is the most potent acute mediator of cerebral vascular tone and cerebral blood flow, great caution must be used when performing ventilation. Over ventilation to a low PCO₂ and high pH may cause decreased cerebral blood flow, worsening brain ischemia, and sometimes causing seizures. Under ventilation to a high PCO₂ may cause cerebral vasodilation and lead to ICP crisis. Both very low ($PO_2 < 60$ mmHg) and very high ($PO_2 > 300$ mmHg) have been strongly linked to poor outcome in TBI and in hypoxic-ischemic encephalopathy after cardiac arrest.
- Rarely, clinicians are forced to employ ventilatory techniques to manage intracranial catastrophes, such as purposeful hyperventilation to acutely decrease intracranial pressure in a patient with acute brain herniation. Such techniques should be performed for the minimum possible time, however, with the goals of substituting more durable means of decreasing ICP, and restoring ventilatory homeostasis as soon as possible.



Just before Tracheal Intubation

In an orderly and efficient manner

- Preoxygenate with 100% O₂
- Administer any pretreatment medications
- Consider osmotic agents (mannitol or hypertonic saline) if ICP is elevated, or is believed to be elevated
- Administer induction agent and paralytic simultaneously (if using thiopental, flush line prior to giving succinylcholine)<Allow for full muscle relaxation (45 seconds for succinylcholine, 60 seconds for rocuronium)
- Consider administering 6-8 low volume manual ventilations during apnea



Patient is normotensive or hypertensive

Consider these induction medications

At time intubation minus 3 minutes give:

- Lidocaine 1.5 mg/kg AND Fentanyl/remifentanyl 3 ug/kg over 30 sec

OR

- Lidocaine 1.5 mg/kg AND Esmolol 1-2 mg/kg

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Place patient on monitors

ECG, Pulse oximeter, capnometer

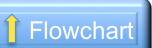
Management of the critically ill patient is generally provided by a team of physicians, nurses, and assistants with each team member doing their intervention simultaneously with the other team members. Placing the patients on monitors provides the baseline physiologic information needed in decision making. In addition, from the onset, any patient who may require intubation should receive 100% oxygen via non-rebreather face mask in order to maximize nitrogen washout and maximize oxygenation; this will minimize desaturation during intubation.



Post-Intubation

Check location of ETT

- Keep HOB elevated to 30 degrees
- Keep MAP > 80 mmHg, or CPP > 60-70 mmHg if ICP is monitored
- Do not hypoventilate
- Keep SpO₂ > 94%
- Follow pupil exam
- Secure endotracheal tube and obtain post-intubation chest film
- Move on to ventilator management



Prepare for Intubation

Have equipment ready

- Elevate the head of bed (HOB)
- IV access X 2
- Infuse isotonic crystalloid
- Have vasopressor available
- Have intubation medications ready
- NPO, consider NG decompression



Set Up Equipment

Include failed airway equipment

The affordability and proven benefit new generation intubation devices have established a new standard in airway management. To have only direct laryngoscopy fails to meet the expect standard of care in managing critical patients. The success of direct laryngoscopy is dependent on patient anatomy, the ability to hyperextend the patient's neck and create an alignment of the visual axis. In trauma, direct laryngoscopy may be impeded by cervical immobilization, bleeding, and or anatomic distortion. Consequently, airway management in critical environments requires that an assortment of airway devices be available; at a minimum, a optical enhancement device, and an intrapharyngeal ventilation system, e.g., LMA, should be available.



The Patient is Hypotensive

Consider these interventions

At time intubation minus 3 minutes give:

- Fluids
- Blood products
- Inotropes
- Pressors

Hypotension may lead to cerebral hypoperfusion. A MAP of > 65 mmHg should be maintained at all times throughout the peri-intubation. This may require small aliquots of vasopressors until definitive volume loading or blood product replacement is achieved.



Titrating Ventilation

Adjusting the ventilator

- P_aO_2 target is 35-40 mm Hg
- ETCO₂ target is 35-45 mmHg; adjust respirator rate to achieve this target
- Obtain arterial blood gas analysis; initial target is $P_aO_2 > 110$ mmHg; Calibrate ETCO₂ measurement with P_aCO_2
- Quantitative capnography is recommend in patient receiving neuromuscular blockade
- All oxygenation and ventilation goals should be adjusted as necessary to maintain cerebral metabolic homeostasis; F_iO₂ may be increased in response to low P_{bt}O₂, but hyperoxygenation with $P_aO_2 > 470$ mmHg is discouraged.



Tracheal Intubation

- Ensure HOB elevated to 30 degrees
- Avoid hypotension (keep MAP > 80 mmHg)
- Avoid hypoventilation
- Bag-mask ventilate immediately if desaturation

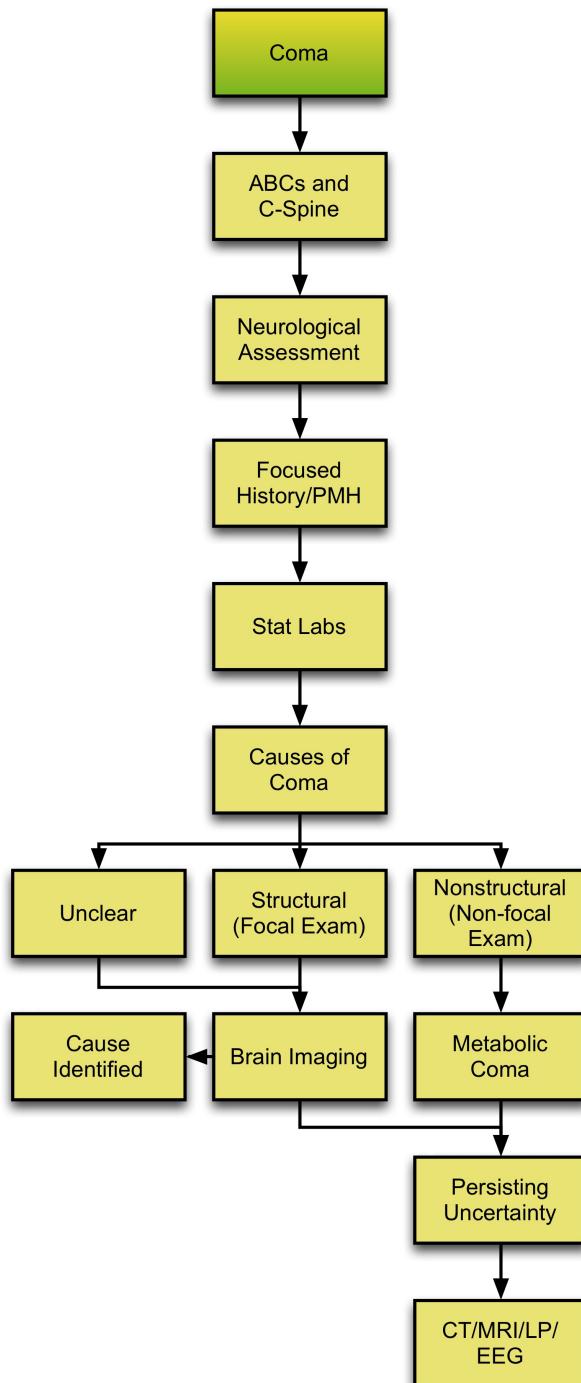
Emergency Neurological Life Support

All Protocols

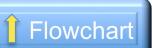
Coma

Version: 1.0

Last Updated: 5/23/2013



[Checklist & Communication](#)

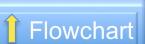


Checklist

- Evaluate/treat circulation, airway, breathing and c-spine
- Exclude/treat hypoglycemia or opioid overdose
- Serum chemistries, arterial blood gas, urine toxicology screen
- Emergent cranial CT if structural or uncertain etiology

Communication

- Current clinical presentation
- Relevant past medical/surgical history
- Findings on neurological examination
- Relevant labs
- Cranial CT, MRI, LP and/or EEG results if available
- Treatments instituted thus far



Assess ABCs and C-Spine

Immobilize C-Spine

- Airway, breathing and circulation are assessed and concurrently treated as detailed in ENLS protocol [Airway, Ventilation and Sedation](#)
- Rapid survey of head and neck, chest, abdomen, and extremities. Cervical spine is immobilized if there is any likelihood of trauma.
- Bedside glucose testing is performed on all unconscious patients. If blood glucose is < 70 mg/dl administer 25 ml of 50% dextrose. Thiamine 100 mg IV should be given with dextrose in patients at risk for nutritional deficiency (e.g. chronic alcohol users, bariatric surgery, malabsorptive states)
- If there is suspicion of opioid toxicodrome (history of drug use, coma, bradypnea, pupillary constriction), administer naloxone 0.4-0.8 mg IV and repeat as needed



Brain Imaging

Head CT

Noncontrast cranial CT should be obtained emergently in unconscious patients with a presumed structural cause and in patients with an unclear cause of coma.

If an acute ischemic stroke is being considered, cranial CT angiography and CT perfusion may be considered as an alternative to MRI (see ENLS protocol [Acute Ischemic Stroke](#)).

When a CNS infection is being considered, cranial CT with and without contrast should be obtained to evaluate for abscess, extra-axial fluid collections, hydrocephalus, hemorrhagic transformation, and vasculitic infarcts.



Causes of Coma

Three possibilities

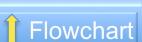
Information accrued so far is used to establish a preliminary impression of either a structural cause, a nonstructural cause, or an unclear cause. Structural and nonstructural causes of coma may coexist.



HPI/PMH

Focused history

Patient history is obtained concurrently with resuscitative measures. Potential causes of unconsciousness are sought from witnesses, friends, family, or EMS personnel. Medical and surgical history, medications, alcohol and illicit drug use, and environmental exposures should be systematically queried.



Metabolic Coma

Global or metabolic causes

Nonstructural causes of coma include anoxic-ischemic encephalopathy, metabolic alterations, endocrinopathies, systemic infections, over dosage of medications, alcohol and illicit drug use, exposure to nonpharmacologic neurotoxic compounds.

Treatment is guided by the underlying etiology. Where appropriate, specific antagonists/antidotes should be administered for example

- Opioid overdose: naloxone
- Acetaminophen overdose: N-acetylcysteine
- In selected cases, such as acute liver failure, an initially metabolic encephalopathy may evolve towards a structural one (cerebral edema, herniation)
- Seizures and [status epilepticus](#) commonly are not associated with any detectable lesion on CT. However, in patients with new seizures or a change in seizure pattern a structural cause must be excluded. [CNS infections](#) (e.g. bacterial meningitis) may have no structural correlate on noncontrast CT, however this study should be obtained to exclude brain abscess. Remember to initiate antibiotics and dexamethasone prior to the head CT if you suspect bacterial meningitis.



Neurological Assessment

Focused neuro exam

Neurologic assessment of the unconscious patient has 3 parts:

- Level of consciousness: [Glasgow Coma Scale](#). Assess additional potential signs of arousal including visual fixation, visual pursuit (tracking), forced eye closure resisting the examiner
- Brainstem examination:
 - Pupillary size, reactivity, and symmetry
 - Corneal reflex
 - Threat response
 - Oculocephalic reflex (Doll's eyes - only if no suspicion of trauma)
 - Vestibulo-oculocephalic reflex (cold calorics)
 - Corneal reflex
 - Gag reflex
 - Cough reflex
- Motor function: spontaneous muscle position/posture, spontaneous movements, response to verbal command, response to noxious stimulus. Examiner should distinguish purposeful from reflexive activity. Examples of purposeful activity include following commands, pushing examiner away, reaching for endotracheal tube, localizing to noxious stimulus. Examples of reflexive activity include withdrawal, flexion, or extension to noxious stimulus
- The breathing pattern may have localizing value in comatose patients with brainstem lesions.
 - Central neurogenic hyperventilation: lesions of the pons or midbrain
 - Cluster breathing: lesions of the pons
 - Absence of spontaneous breathing, ataxic breathing, cluster breathing: lesions involving the medulla



Persisting Uncertainty

Next steps

When diagnostic uncertainty persists after the work-up so far, additional test measures include:

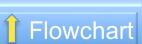
- Noncontrast CT is obtained in all comatose patients with an undiagnosed etiology if not done already
- Consider basilar artery thrombosis (hyperdense basilar artery sign); CTA or MRA is definitive
- EEG obtained to evaluate for nonconvulsive seizures or status epilepticus, burst suppression, or patterns consistent with metabolic encephalopathy
- Lumbar puncture is obtained if there is suspicion of CNS infection, inflammation, infiltration with lymphoma or malignant cells, or to substantiate a suspicion of aneurysmal subarachnoid hemorrhage in patients with negative CT findings. Prior to LP, space occupying lesions should be ruled out with noncontrast head CT
- MRI is obtained if there is a presumption of hyperacute ischemic stroke or when the cause of coma is not explained by other tests
- Consultation with a specialist



Presumed Nonstructural Metabolic Causes

A nonstructural cause of coma is suggested by

- Progressive, gradual onset of symptoms
- History of medication, alcohol, or illicit drug use, or environmental toxic exposure
- Symmetric cranial nerve and motor findings



Presumed Structural Focal pathology

A structural etiology is suggested by

- History: trauma, acute onset of symptoms, AIDS, malignancy
- Physical examination: asymmetric cranial nerve findings, asymmetric motor responses
- Absence of an obvious toxic-metabolic etiology

Until/unless proven otherwise, coma is presumed to be structural in origin and should be immediately assessed with a noncontrast cranial CT, since emergent neurosurgical management may be needed.

Patients with a new onset of seizures, a change in seizure pattern, or status epilepticus should be evaluated for a possible structural substrate. See ENLS protocol [Status Epilepticus](#).

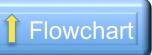


Stat Labs

Serum chemistries, CBC, PT/PTT, ABG, urine toxicology, blood EtOH

Unless a readily reversible cause of unresponsiveness has been discovered and corrected, additional laboratory work is obtained emergently.

- Serum chemistries at a minimum Na, K, creatinine, BUN, transaminases
- Hematological panel at a minimum hemoglobin/hematocrit, platelets, white blood cells
- Arterial blood gas
- Blood alcohol level; urine toxicology screen for opioids, benzodiazepines, illicit drugs. (Note: Some toxins that cause unconsciousness are not detectable in common toxicologic screens)
- Urinalysis; cultures of blood, urine



Structural Cause

CT finding reveal cause

Structural causes of coma include [trauma](#), [ischemic stroke](#), [hemorrhagic stroke](#), brain tumor, and [CNS infections](#).

Management should be initiated in consultation with Neurology and/or Neurosurgery.



Unclear Etiology

In many patients, the etiology of coma cannot be identified with information collected so far. These patients should undergo emergent noncontrast cranial CT and further testing if CT is negative.



Unconscious Patient

Eyes closed, unresponsive

A patient who has eyes closed and is unresponsive is comatose.

Determine unresponsiveness:

- Observation: eyes closed, immobility, lack of facial expression, obliviousness to environmental stimuli

Examiner evaluates response to graded stimulus

- Verbal stimulus ("are you OK?" or "what is your name?")
- Tactile stimulus (to body parts with large cortical representation: face, hands)
- Noxious stimulus. Noxious stimulus should be intense but not cause tissue injury. Recommended maneuvers include sternal rub, nail-bed pressure, pressure on supraorbital ridge or on posterior aspect of mandibular ramus.

Topic Co-Chairs: J. Stephen Huff, MD Robert Stevens, MD

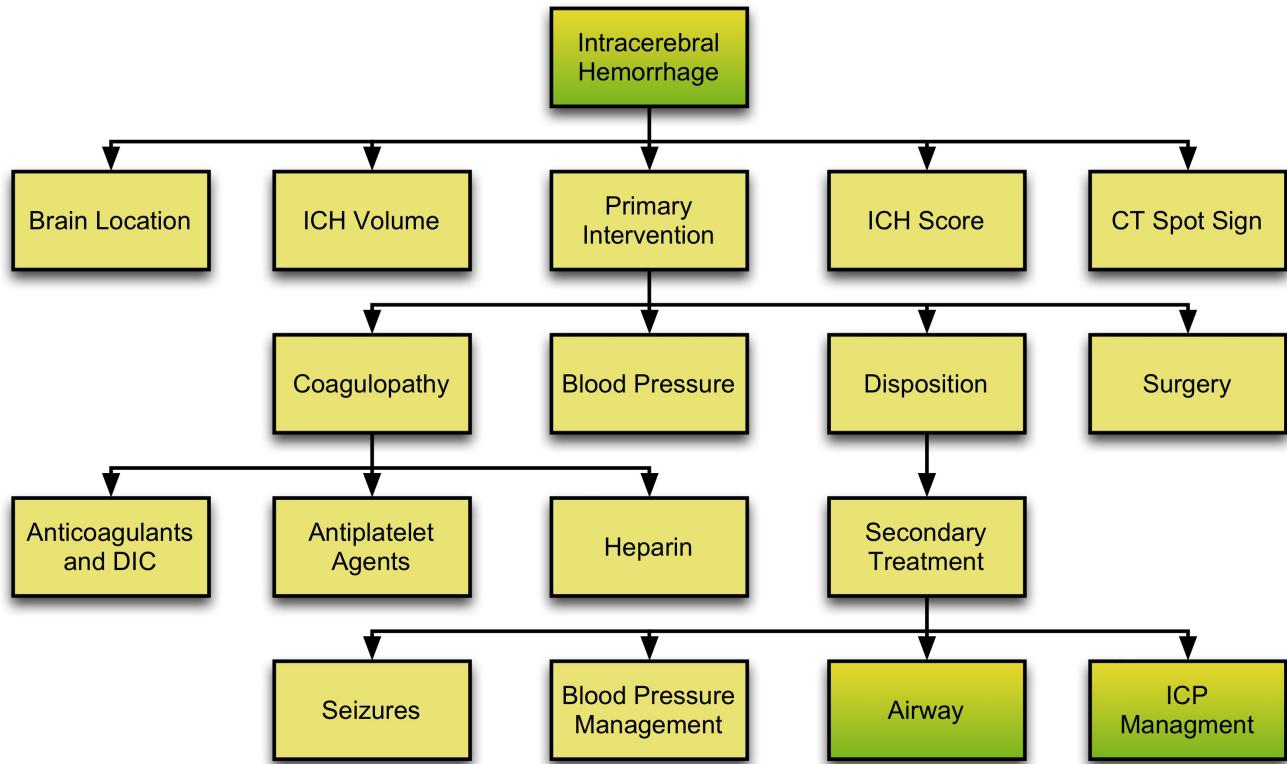
Emergency Neurological Life Support

All Protocols

Intracerebral Hemorrhage

Version: 1.0

Last Updated: 5/23/2013



[Checklist & Communication](#)



Checklist

- Check PT, PTT, INR
- Head Imaging Results: Size of bleed
- GCS
- Calculate ICH Score

Communication

- Age
- ICH Volume
- GCS
- ICH Score

- Hydrocephalus present?



Admit

ICU admission

NeuroICU admission is preferable



Airway

Is the patient's airway stable?

ICH may continue to expand and the patient's mental status and airway may become compromised. Continued vigilance to airway is critical especially in posterior fossae hemorrhages.



Anticoagulants and DIC

INR > 1.4

Consider vitamin K antagonist reversal with purified factor concentrates or FFP if warfarin or other vitamin K antagonists have been prescribed, followed by Vitamin K 10 mg IV. To calculate the volume of plasma or IU of prothrombin complex concentrate:

1. Decide on target INR
2. Convert INR to percent (%) functional prothrombin complex:

INR Range	Percent function prothrombin complex
> 5	5%
4.0 – 4.9	10%
2.6 – 3.9	15%
2.2 – 2.5	20%
1.9 – 2.1	25%
1.7 – 1.8	30%
1.4 – 1.6	40%
1.0	100%

3. Calculate dose:

(Target in %PC - Current level in %PC) X weight (kg) = mL of FFP or IU of prothrombin-complex concentrate (PCC) needed

Example: a patient with INR on arrival = 7.5, target INR 1.5, body weight = 80 kg:

$$(40-5) \times 80 = 2,800$$

Therefore, the needed dose is 2,800 mL of FFP or 2,800 IU of PCC.

Reference: Schulman, S. Care of patients receiving long-term anticoagulant therapy. NEJM (2003) 349:675



Antiplatelet Agents

Aspirin, clopidogrel, prasugrel, etc.

If the patient has been taking antiplatelet drugs, transfuse with platelets and administer DDAVP 0.3 mcg/kg IV.



Blood Pressure

Should BP be lowered?

Keep SBP below 160 mm Hg or MAP below 110 mmHg; consider using IV nicardipine with and without IV labetalol



Brain Location

Brain location of ICH

Determine where the bleeding has occurred.

Options include:

- lobar
- basal ganglia
- thalamus
- cerebellum
- midbrain
- pons
- intraventricular



Coagulopathy

Is there an underlying coagulopathy?

Consider use of vitamin K antagonists, antiplatelet agents, DIC



Control BP

Continue to control blood pressure

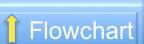
Keep SBP below 160 mm Hg or MAP below 110 mmHg; consider using IV nicardipine with and without IV labetalol



Heparin

Recent heparin administration

Administer protamine sulfate 1 mg per 100 U heparin received in last 2 hours; maximum dose 50 mg



ICH Score

Calculate the ICH score

The ICH score can be calculated as follows:

Component	Criteria	Points
GCS	3-4	2
	5-12	1
	13-15	0
ICH Volume (cc)	≥ 30 cc	1
	< 30 cc	0
Intraventricular Hemorrhage	Yes	1
	No	0
Infratentorial Origin	Yes	1
	No	0
Age	≥ 80 y	1
	< 80 y	0
Total		0-6



ICH Volume

Measure the amount of blood

If the blood is within the brain parenchyma, use the ABC/2 method.

Formula for Estimating ICH Hematoma Volume



A red crosshair is drawn through the center of the hemorrhage, with point A marking the longest axis and point B marking the longest axis perpendicular to A.

$$\frac{A \times B \times C}{2}$$

Select CT slice with largest ICH
A = longest axis (cm)
B = longest axis perpendicular to A (cm)
C = # of slices x slice thickness (cm)

Estimated volume of spheroid
Correlates well w/ planimetric CT analysis

Kothari et al. Stroke 27:1304-1305, 1996

Image courtesy of J. Claude Hemphill III, MD, MAS



ICP Elevated

Is the patient developing high ICP?

Consider ICP monitoring if GCS <8 or the patient has symptomatic hydrocephalus.
See ENLS protocol [Elevated ICP and Herniation](#) for management recommendations.



Intracerebral Hemorrhage (ICH) Diagnosis

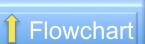
ICH diagnosis confirmed

Intracerebral Hemorrhage (ICH): ICH typically produces a new headache followed by progressive neurological signs. The onset is usually sudden and many patients progress over a few hours likely due to continuing intracerebral bleeding. It is not possible to be certain whether the stroke is due to hemorrhage or ischemia based on signs and symptoms alone, so some form of brain imaging is necessary.

Topic Co-Chairs: J. Claude Hemphill, MD, MAS

Ed Jauch, MD

Contributors: Mary Kay Bader, Jon Edlow, Kevin Sheth, Luis Computaro, Susan Yeager, Oliver Flower



Other findings

CT spot sign

If IV contrast was administered during the CT scan, extravasation of contrast can be seen in hemorrhages that are still accumulating. This is called the spot sign as shown in the figure:

CT “Spot Sign” seen on CTA or post-contrast image

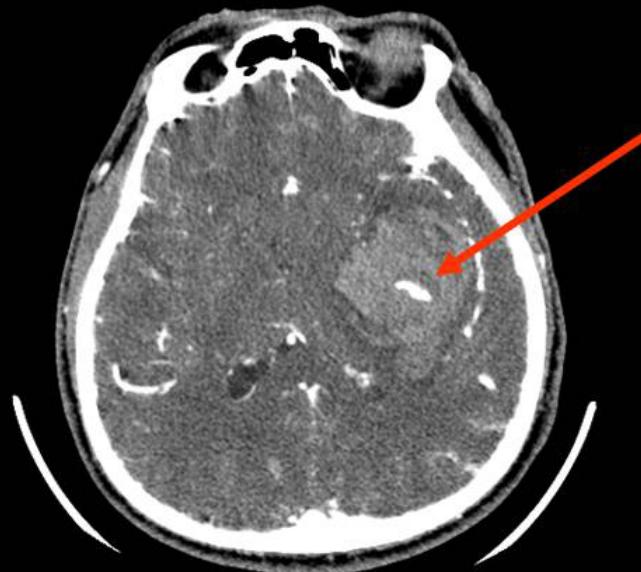


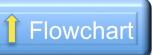
Image courtesy of Joshua Goldstein, MD



Primary Intervention

First steps for intervention

Intervention for ICH is classified as "primary" meaning what can be done to impact the patient right now, and "secondary" once these primary interventions are addressed. Certainly, one can consider the secondary interventions of blood pressure control, declining neurological exam requiring airway protection, concurrently.



Secondary Treatment

Begin secondary interventions

Intervention for ICH is classified as "primary" meaning what can be done to impact the patient right now, and "secondary" once these primary interventions are addressed.



Seizures

Seizure prophylaxis and treatment

- Do not administer prophylactic anticonvulsants.
- Treat clinical seizures with benzodiazepines then anticonvulsants.
- Consider EEG monitoring if the patient's level of consciousness is worse than is likely explained by the size and location of the hemorrhage.



Surgery

Is the patient a surgical candidate?

Cerebellar ICH should be considered for surgery urgently depending on size.
Lobar ICH with mass effect should also be considered for surgery.

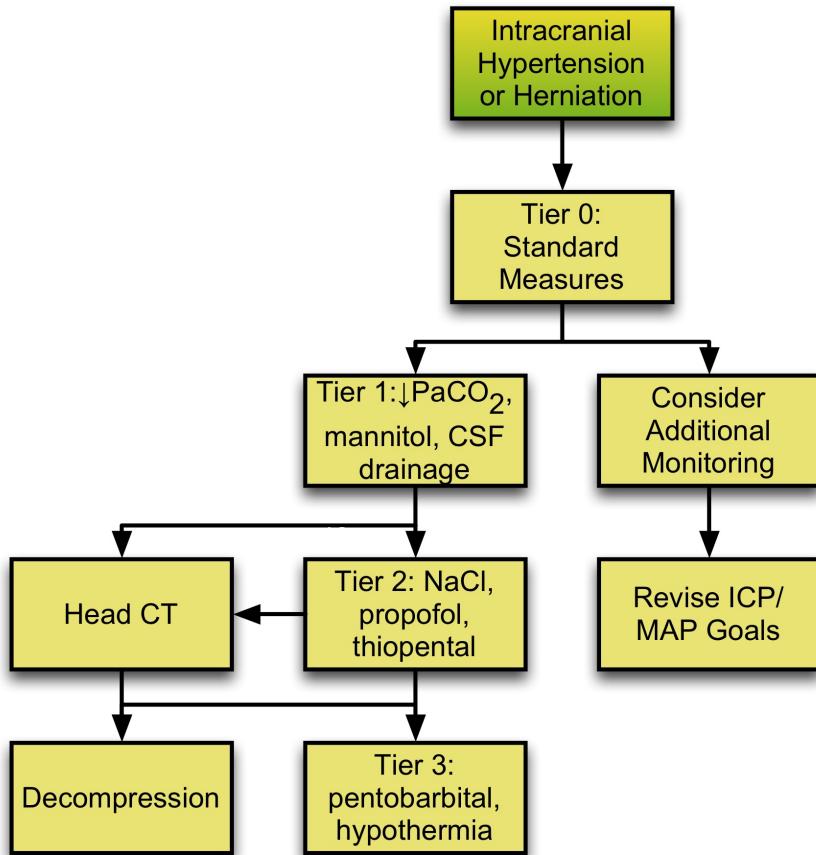
Emergency Neurological Life Support

All Protocols

Elevated ICP or Herniation

Version: 1.0

Last Updated: 5/23/2013



[Checklist & Communication](#)



Checklist

- Tier Zero: HOB > 30 degrees; ensure adequate sedation; correct hyponatremia, hyperthermia, and vasogenic edema; correct hyperthermia; keep CPP > 60-70 mm Hg
- Tier One: secure airway; mannitol 0.5-1 gm/kg IV bolus; CSF drainage; start 3% saline 10-20 cc/hr
- Tier Two: hypertonic saline bolus (3%-23.4%); consider propofol bolus and infusion; consider decompressive craniotomy
- Tier Three: pentobarbital bolus and infusion titrated for ICP goal; induce hypothermia; hyperventilation if used with cerebral oxygen monitor; raise MAP to improve CPP



Consider Additional Monitoring

Consider additional neuromonitoring, including:

- Brain tissue oxygen or jugular venous oximetry
- Cerebral microdialysis
- Cerebral pressure autoregulation indices

Treatment based on ICP and CPP may overlook significant information on the physiologic and metabolic state of the brain. Moreover, assumptions regarding CPP may not hold if cerebral pressure autoregulation is impaired (see Box I). Complementary neuromonitoring techniques should be considered to optimize medical management in selected patients with severe brain injury.

- Monitors of cerebral oxygenation: brain tissue oxygen sensors, jugular venous oximetry.
- Cerebral microdialysis. Brain interstitial lactate, lactate/pyruvate ratio, and glutamate are indicative of cerebral ischemia in the region of the microdialysis probe.
- Dynamic indices of cerebral autoregulation. These indices express the correlation between a systemic hemodynamic parameter (arterial blood pressure or CPP) and an intracranial physiological parameter, e.g. ICP (PRx), transcranial Doppler-derived CBF velocity (M_x), or brain tissue PO₂ (Orx). High degrees of correlation suggest failure of autoregulation.



Decompressive Surgery

For mass lesion or hemicraniectomy

The decision to proceed with surgical decompression is made in consultation with neurosurgery and prioritizes patients in whom there is a significant likelihood of meaningful recovery.

- Surgical mass lesion. Surgical evacuation to be considered in selected patients with rapid neurologic deterioration from space-occupying lesions, e.g. brain tumors, brain abscesses, ischemic stroke, traumatic and nontraumatic intraparenchymal hemorrhages.
- Decompressive craniectomy may also be considered in the absence of a focal lesion, i.e. diffuse brain edema associated with aneurysmal subarachnoid hemorrhage, traumatic brain injury, and meningoencephalitis



Intracranial Hypertension or Herniation

ICP > 20 mm Hg or Clinical Signs

Intracranial hypertension (elevated ICP) or clinical brain herniation are a "brain code" and must be addressed urgently.

- Intracranial hypertension is defined as ICP > 20 mm Hg sustained for more than 5 minutes.
- Cardinal signs of transtentorial herniation are the acute onset of unilateral or bilateral pupillary dilation with loss of light reactivity, and loss of consciousness
- Other clinical changes that indicate herniation include extensor posturing, hypertension, bradycardia and changes in respiratory pattern (Cushing's triad).

Subtopic written by: Robert Stevens, MD Josh Duckworth, MD Alexander Papangelou, MD J. Stephen Huff, MD



Maintain CPP 60-70 mm Hg

Using fluids and/or vasopressors

CPP (MAP - ICP) should be maintained in the 60-70 mmHg range to prevent cerebral ischemia.

- This can be done by lowering ICP (see above) or can be accomplished by raising MAP with fluids, vasopressors, or inotropes.



Non-contrast Head CT

Head imaging should be performed to exclude bleeding, hydrocephalus, cytotoxic edema or other sources of mass effect causing an acute elevation in ICP.

- CT is the preferred method of imaging because of availability and speed of imaging. The patient needs to lay flat for this study so it is wise to make sure that the patient can tolerate lowering the head of the bed prior to transporting.
- CT results will inform decisions to place or revise an intraventricular drain (hydrocephalus), perform decompressive craniectomy, or remove a mass lesion.



Rescue Surgery

If tier 1 and 2 fail

Consider rescue surgery, i.e. performing procedures described in decompressive surgery above (evacuation of mass lesions or decompressive craniectomy in the absence of mass lesions) in consultation with the neurosurgery.

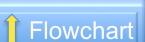
If the patient is ineligible for further surgery, proceed to Tier Three.



Revise CPP Targets

Consider modifying CPP targets:

- Cerebral hypoxia (brain tissue hypoxia, jugular venous oxygen desaturation) or ischemia (increased lactate/pyruvate) can be treated by controlled increases in CPP, or by other measures that increase cerebral oxygen delivery (transfusion, inotropic agents, increased FIO₂).
- In patients with preserved cerebral autoregulation and CPP 60-70 mmHg, controlled increases in CPP may reduce ICP through increases in cerebrovascular resistance leading to reductions in CBV
- In patients with impaired cerebral pressure autoregulation and CPP > 60 mmHg, controlled reductions in CPP to the 40-60 mmHg range can effectively reduce ICP (by decreasing CBF and CBV), however such manipulations should only be accomplished with simultaneous cerebral oxygenation/ischemia monitoring.



Tier One

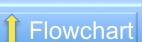
Airway, hyperventilation, mannitol, CSF drainage

The first interventions should include:

- Ensure airway (endotracheally intubate or use tracheostomy if present)
- Short-term hyperventilation may be instituted either with manual bag-mask technique or mechanically.
- Mannitol is administered as 0.5-1 g/kg IV bolus
- CSF drainage: If acute obstructive hydrocephalus is contributing to clinical deterioration, place EVD emergently. If external ventricular drainage system is in place, drain 5-10 ml of CSF.
- Begin 2% or 3% IV saline to keep serum sodium between 140 and 150 meq/L. Check serum electrolytes Q 2-4 hours.

If ICP is controlled and/or clinical signs of herniation resolve with Tier One interventions, obtain head imaging studies.

If not, move to Tier Two interventions first.



Tier Three

No longer a surgical candidate

Tier Three measures represent the most aggressive level of medical management and carry the highest risk of adverse effects.

- Pentobarbital dosing: bolus 10 mg/kg IV over 30 min, then 5 mg/kg/hr x 3 hrs.; maintenance 1 - 4 mg/kg/hr, titrated to ICP goal. Infusion is continued for 24 - 96 hrs. while underlying process driving ICP is treated or begins to resolve. Treatment is associated with respiratory depression, circulatory instability, immune suppression, and paralytic ileus.
- Moderate hypothermia (target core temperature, 32 - 34 degrees C) is induced with external cooling devices or with intravenous infusion of cooled fluids. Treatment is associated with shivering, cardiac arrhythmia, sepsis, coagulopathy, and electrolytes disturbances.
- Hyperventilation to moderate hypocapnia (P_aCO_2 25-35 mm Hg) may be considered in selected patients who have failed Tiers One and Two. Hyperventilation should be accomplished in conjunction with a cerebral oxygenation monitor (jugular venous oximetry, brain tissue oxygen probe), in order to minimize the risk of cerebral ischemia. Prolonging hyperventilation for > 6 hours is unlikely to be beneficial and may cause harm.
- CPP manipulation



Tier Two

Hypertonic saline, propofol, thiopental

- Hypertonic saline bolus may be administered in concentrations ranging from 3% to 23.4% with goal of serum sodium between 140 and 150 meq/L. Evidence supports rapid infusion of hypertonic saline bolus to reverse transtentorial herniation or decrease ICP. Concentrations > 2% must be given through a central venous catheter.
- If ICP is not responsive to sodium infusion, consider propofol 1-3 mg/kg to reduce CMRO₂, CBF, and ICP. Administration of propofol may be associated with circulatory depression that should be corrected with a iv fluids or a vasopressor infusion to maintain CPP goal. Propofol may be continued as an infusion 200 micrograms/kg/min.

If ICP is responsive to Tier Two therapies, and the patient has not been imaged yet, obtain brain imaging.

If the patient is unresponsive to Tier One and Tier Two interventions, consider rescue surgery, i.e. performing procedures described in decompressive surgery (evacuation of mass lesions or decompressive craniectomy in the absence of mass lesions) in consultation with the neurosurgery.

If the patient is ineligible for surgery or too unstable to obtain brain imaging, proceed to Tier Three.



Tier Zero

Standard issues to prevent herniation

For sustained ICP elevation or clinical signs of herniation:

- Make sure the head has not been lowered below 30 degrees
- Be sure that the ICP elevation is not associated with tracheal suctioning or other noxious stimulus.
- Is hyponatremia present? If so begin correction
- Is hyperthermia present? If so begin measures to lower body/brain temperature
- Is ICP elevation associated with agitation? If so, treat pain and consider short acting sedation.
- Is vasogenic edema present? If so, high dose corticosteroids should be given when brain code is driven by a brain tumor (primary or metastatic), brain abscess, or the in presence of a progressive neuroinflammatory process (e.g., acute disseminated encephalomyelitis). For brain tumor, typical regimens are dexamethasone 0.1 mg/kg q 6 hrs. or methylprednisolone 0.5 mg/kg q 6hrs

CPP (MAP-ICP) should be maintained in the 60-70 mmHg range to prevent cerebral ischemia.

- This can be done by lowing ICP or raising MAP with fluids, vasopressors or inotropes.

If elevated ICP persists or clinical signs of herniation are not rapidly mitigated by the above, proceed to Tier One treatments.

Additional monitoring may be useful now based on the specific disease process.

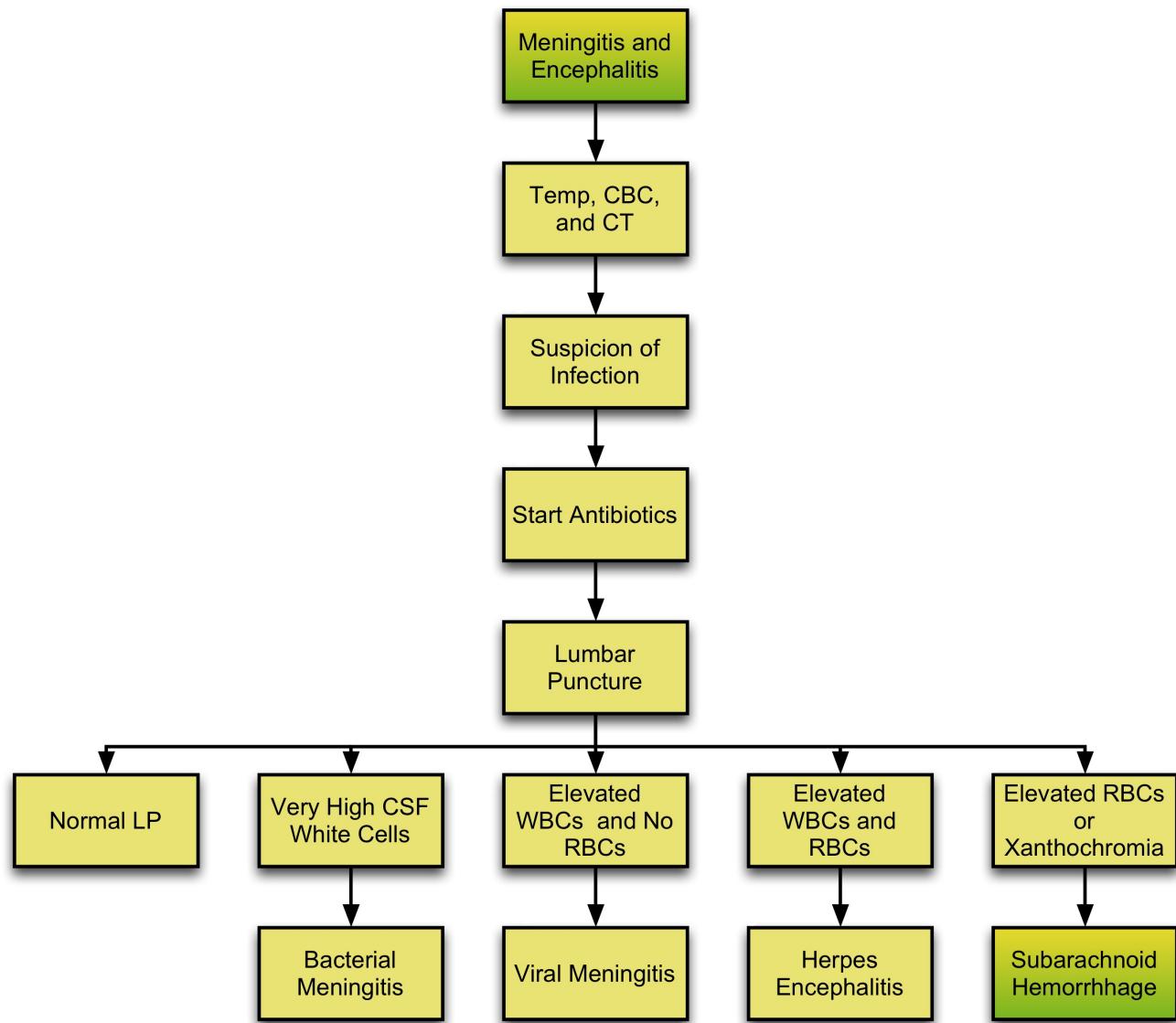
Emergency Neurological Life Support

All Protocols

Meningitis and Encephalitis

Version: 1.0

Last Updated: 5/23/2013



[Checklist & Communication](#)

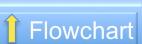


Checklist

- Vital signs, history, examination
- IV access, draw labs, blood cultures and lactate
- IV fluids, treat shock
- Immediate administration of dexamethasone followed by appropriate antibiotics to treat suspected meningitis.
- Consider acyclovir
- Obtain head CT if altered mental status or focal neurological findings.
- Perform lumbar puncture (after Head CT results available, if CT necessary)
- If meningococcus remember exposure prophylaxis

Communication

- Presenting signs, symptoms, vital signs on arrival
- Pertinent past medical history and history of the present illness
- Relevant laboratory results including white blood cell count, bicarbonate level, lactate level, and renal function
- Whether head CT was obtained and results if obtained
- Antibiotics given
- IV fluid given, input/output
- Results of LP
- Current vital signs
- Ongoing concerns, active issues, outstanding studies/tests
- Last physical and neurological exam finding prior to transfer



Bacterial Meningitis

Likely bacterial meningitis

- Continue antibiotics
- Stop acyclovir
- Continue dexamethasone
- Adjust antibiotics based on finalized gram stain and culture results and sensitivities

In addition to antibiotics and dexamethasone, supportive care and management of other systems is important in patients with bacterial meningitis. Some patients may have a concomitant bloodstream infection with the offending pathogen and may require early goal directed therapy for sepsis. If the lumbar puncture demonstrates elevated intracranial pressure when the opening pressure is measured, the patients should be monitored closely for signs of increased ICP. There is no evidence that intracranial pressure monitoring devices are safe or helpful in this patient population and the risks, including the potential of a superinfection with the foreign body, must be weighed with the potential benefits. Likewise, no evidence exists as to the appropriate treatment of increased ICP. Hyperventilation should probably be avoided as these patients already may suffer from some degree of decrease vessel diameter due to vasculopathy. Mannitol or hypertonic saline may be reasonable alternatives.

Age factors:

- Children and young adults with suspected bacterial meningitis are at risk for *Haemophilus influenzae* (if not vaccinated), *Neisseria meningitidis*, and *Streptococcus pneumoniae*. As such they should be started on a 3rd generation cephalosporin and vancomycin at doses appropriate for CNS penetration
- Middle aged adults are at highest risk for *Streptococcus pneumoniae*. As such they should be started on a 3rd generation cephalosporin and vancomycin at doses appropriate for CNS penetration. Vancomycin can be used alone in patients with a severe penicillin allergy.
- The elderly and immunosuppressed are at risk for *Streptococcus pneumoniae* and *Listeria monocytogenes*. As such they should be started on a Ampicillin, a 3rd generation cephalosporin and vancomycin at doses appropriate for CNS penetration. Vancomycin and trimethaprim-sulfamethoxazole can be used in patients with a severe penicillin allergy.



Elevated RBCs and WBCs

Consider herpes encephalitis

If the following is true:

- Elevated RBC
- WBCs in the hundreds
- Glucose > two-thirds serum glucose, or sometimes lower
- Protein < 50 mg/dL or elevated
- No organisms on gram stain

Then, the patient may have herpes encephalitis. The presence of seizures is also compatible with this diagnosis.



Elevated RBCs no WBCs

Likely SAH

If the following is true:

- Elevated RBC
- WBC < 5
- Glucose > two-thirds serum glucose
- Protein < 50 mg/dL
- No organisms on gram stain
- Xanthochromia

Then, the patient likely has a subarachnoid hemorrhage that was not detected on the CT scan. Xanthochromia may be absent if the LP was done within the first few hours of headache onset (and so one typically only sees RBCs).



Elevated WBC no RBCs

Probably viral meningitis

Mild elevation in WBCs without RBCs is suggestive of viral meningitis or viral (not herpes) encephalitis. So, if the following is true:

- Normal RBC
- WBCs 10-100s
- Glucose > two-thirds serum glucose
- Protein < 50 mg/dL
- No organisms seen on gram stain

Then the patient likely has a viral meningitis or viral (not herpes) encephalitis. Seroconversion of HIV is also a consideration here.



Herpes Encephalitis

Empirical treatment and diagnosis

- Continue acyclovir 10 mg/kg every 8 hours IV
- Continue other antibiotics until MRI/PCR negative
- Send CSF for HSV PCR
- MRI of the brain
- Achieve and maintain euvoolemia to prevent acyclovir associated renal failure



Immunocompromised Patient

Confirmed or suspected

Immunocompromised patients, or patients suspected of being Immunocompromised, may present with less classic signs of meningitis or encephalitis.

- For such patients, lower your pretest probability for these diagnoses and error on the side of a more complete work-up including LP and brain imaging.



Lumbar Puncture

Rapid assessment of spinal fluid

An LP is essential for both establishing a diagnosis and tailoring therapy.

The opening pressure should be measured with a manometer prior to the collection of CSF. CSF should be collected in (at least) 4 tubes.

- Send tube 1 and tube 4 for red and white cell counts
- Send tube 2 for protein, glucose and lactic acid
- Send tube 3 for gram stain and culture (and India ink if fungal infection is suspected).

If there is a suspicion for Herpes encephalitis, a small amount of CSF from tube 2 or 3 should be sent for Herpes PCR. Some laboratories perform bacterial antigen assays, which may be useful in some circumstances. Additional laboratory tests that may be performed by some centers include bacterial PCR (particularly for Mycobacterium), enterovirus PCR, fungal antigens and viral culture.



Normal LP

Rules out meningitis and encephalitis

An LP is considered normal if

- No RBCs
- WBCs < 5
- Glucose > two-thirds serum glucose
- Protein < 50 mg/dL
- No organisms seen on gram stain

If all of the above are true, meningitis is ruled out as is encephalitis (in most cases). Work-up the patient from the perspective of fever, elevated WBC count and a normal CT scan without evidence of meningitis or encephalitis.



Start Antibiotics

Empirical treatment

Anti-infectives should be started as soon as possible after the patient with a suspected CNS infection presents for medical care.

Empiric anti-infectives are based on the:

- Course of the suspected CNS infection
- Age of the patient
- Other infectious risk factors

For suspected CNS infections that evolve over hours, consider bacterial meningitis or viral meningitis.

- Children and young adults with suspected bacterial meningitis are at risk for *Haemophilus influenzae* (if not vaccinated), *Neisseria meningitidis*, and *Streptococcus pneumoniae*. As such they should be started on a 3rd generation cephalosporin and vancomycin at doses appropriate for CNS penetration.

Middle aged adults are at highest risk for *Streptococcus pneumoniae*. As such they should be started on a 3rd generation cephalosporin and vancomycin at doses appropriate for CNS penetration. Vancomycin can be used alone in patients with a severe penicillin allergy.

The elderly and immunosuppressed are at risk for *Streptococcus pneumoniae* and *Listeria monocytogenes*. As such they should be started on a Ampicillin, a 3rd generation cephalosporin and vancomycin at doses appropriate for CNS penetration. Vancomycin and trimethaprim-sulfamethoxazole can be used in patients with a severe penicillin allergy.

For suspected CNS infections that evolve over days consider viral encephalitis, particularly Herpes simplex encephalitis: Treatment should begin with Acyclovir at 10mg/kg every 8 hours. Hydration should be sufficient to achieve normovolemia. This avoids the complication of acyclovir associated renal failure.

For suspected CNS infections that evolve over days in an immunosuppressed patient, consider fungal meningitis. If there is a high index of suspicion for fungal meningitis such as prior history of the disease or systemic fungal infections, and the patient is progressing rapidly, empiric Amphotericin B, can be considered. Otherwise, starting anti-fungals after LP is typically prudent.



Subarachnoid Hemorrhage

Management of SAH

Re-review the head CT to look for subarachnoid blood (this can be negative approximately 5% of the time).

See the ENLS protocol [Subarachnoid Hemorrhage](#).



Suspected Meningitis or Encephalitis

Headache and altered mental status

Meningitis and Encephalitis: Patients that have a hyper-acute (hours) and acute (hours to days) onset of headache and altered mental status, should be considered as potentially having meningitis or encephalitis. Additional symptoms including stiff neck (to flex/extend), fever, new rash, focal neurological finds or new onset seizures, should significantly increase the clinical suspicion of CNS infection.

As with all acute medical and neurological events, the basics of ABC (airway, breathing and circulation) should be evaluated early in the Emergency Department course. Patients with altered mental status are at high risk for losing a patent airway and should be monitored closely for the potential of needing intubation. Likewise, patients with bacterial meningitis are at risk for lung or bloodstream infections with the same pathogen, and as such, vital signs and hemodynamics need to be monitored closely.

Meningitis is defined as inflammation of the meninges while encephalitis is defined as inflammation of the brain. If both are inflamed, the patient has meningoencephalitis. Meningitis causes fever, meningismus, and pain (headache, neck, etc.) but other than depressing a patient's mental status, does not affect any cortical function. Encephalitis on the other hand causes typically cortical disturbances (seizures, aphasia, hemiparesis, etc.). In pure encephalitis, the spinal fluid is free of white cells but protein may be elevated. Once white cells are found in the spinal fluid, some form of meningitis is present.

The two conditions that are most important to recognize in the first hour are bacterial meningitis and herpes encephalitis as these diseases have specific treatments that can improve patient outcome if administered quickly.

Topic Co-Chairs: David Gaieski, MD Bart Nathan, MD



Suspicion for CNS Infection

Moderate to high suspicion

Based on the head CT scan being negative (if performed), and the presence of fever and white count, along with headache and altered mental status, one should have moderate to high suspicion for meningitis or encephalitis.

There is evidence for the use of dexamethasone in bacterial meningitis, particularly in *Streptococcus pneumoniae* meningitis. Unless there is clear clinical evidence that the cause is NOT *Streptococcus pneumoniae*, dexamethasone is recommended.

Give:

- dexamethasone 10 mg now and q 6 hours IV. Ideally the steroid should be given prior to or at the start of antibiotic therapy.



Temperature, WBC, CT results

Fever, leukocytosis, normal head CT?

Assess body temperature, peripheral white count, and head CT results (if there is time).

Temperature

Oral temperature is adequate. Both fever (temperature $> 38^{\circ}\text{C}$) or hypothermia (temperature $< 35^{\circ}\text{C}$) are compatible with CNS infection. If the patient is euthermic, the pretest probability of bacterial meningitis or HIV encephalitis is decreased. However, newly Immunocompromised patients, patients with viral meningitis, and even a rare patient with bacterial meningitis may present euthermic. Depending on other signs and symptoms, it may be appropriate to stop here and work-up other causes of headache.

Peripheral white count

If the white count is not elevated, then bacterial meningitis is unlikely; depending on body temperature and results of the head CT scan, you may stop here and work up non-infectious causes of headache and altered mental status with the same caveats mentioned in "Temperature" above. For example, the patient may still have a viral meningitis without a leukocytosis so LP may still be indicated to establish a diagnosis.

Head CT

In patients where there is a moderate to high suspicion of CNS infection and the lumbar puncture has not yet been done, parenteral anti-infectives should not be delayed while waiting for a CT scan. CSF sterilization occurs only after 4-6 hours in the most sensitive organisms.

A head CT prior to the LP should always be done in the patient with suspected CNS infection when the presentation includes papilledema/loss of venous pulsations or focal neurological signs. Other definite indications include patients with known mass lesions. A head CT is NOT always required prior to an LP, however, in most patients who have a clinical presentation consistent with meningitis or encephalitis, there will be enough uncertainty as to the exact intracranial process, that it is incumbent on the examiner to perform a CT prior to the LP. A normal head CT does not protect the patient from a herniation syndrome after the LP.

If the head CT shows a mass lesion or other condition that adequately explains the patient's mental status, then stop here and work up that process.



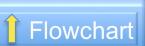
Very High WBCs

WBCs > 100-1000

Marked elevation in WBCs without RBCs is highly suggestive of bacterial meningitis. So, if the following is true:

- Normal RBC
- WBCs 100-1000 or higher
- Glucose < two-thirds serum glucose, but rarely normal
- Protein > 50 mg/dL
- Organisms seen on gram stain

Then, the patient has bacterial meningitis.



Viral meningitis or Viral (not Herpes) Encephalitis

Treatment

Treatment of viral meningitis or viral (not herpes) encephalitis:

- Discontinue acyclovir and antibiotics
- Discontinue dexamethasone
- Treat headache
- For West Nile Virus, there is risk of respiratory decompensation from spinal cord involvement so admission to the ICU for observation may be beneficial

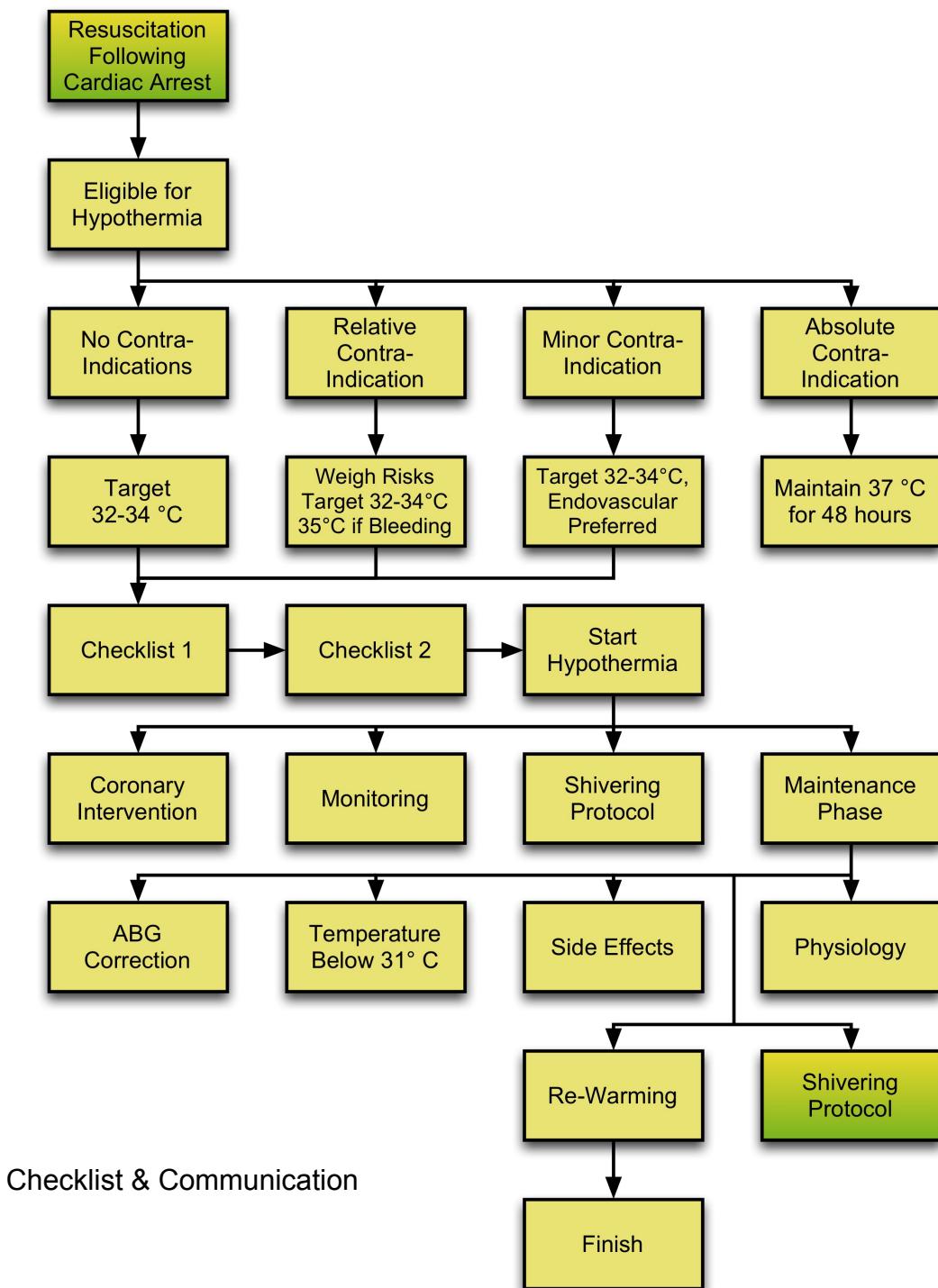
Emergency Neurological Life Support

All Protocols

Resuscitation following Cardiac Arrest Protocol

Version: 1.0

Last Updated: 5/23/2013





Checklist

- Eligibility for hypothermia assessed
- Target temperature decided
- Complete checklists 1 and 2
- Induction of hypothermia started
- Anti-shivering plan in place

Communication

- Duration of cardiac arrest
- Neurological examination on first assessment in the Emergency Department
- When hypothermia was induced
- Any relative or minor contraindications to hypothermia
- Current core temperature



Absolute contraindication

One or more absolute contraindication is present

Maintain 37°C for 48 hours.



Absolute Contraindications

Any absolute contraindications?

Do not induce hypothermia if any of the following is true:

- Rapid neurologic recovery (patient is following commands; squeezes fingers/lets go, wiggles toes on command)
- Illness that precludes meaningful recovery
- Prior advanced directive or do-not-resuscitate wishes
- Other preclusion to ICU admission



Checklist 1- Preinduction

Make sure of the following before hypothermia induction

Checklist 1:

- Patient is intubated
- Patient is comatose and/or sedated.
- Patient does not meet any exclusion criteria
- A probe for core temperature measurement is in place (in order of preference): endovascular, esophageal, bladder, rectal. Peripheral temperature measurements during hypothermia are unreliable).



Checklist 2- Preinduction

Make sure of the following before hypothermia induction

Checklist 2:

- Sedation: propofol if patient is hemodynamically stable; midazolam if hemodynamically unstable
- Analgesia: fentanyl or remifentanil infusion
- Consider 4 gms magnesium over 15 minutes IV
- Avoid continuous paralysis unless EEG is in place



Coronary Intervention

Does the patient need coronary intervention?

Coronary angiography can be safely performed during mild hypothermia. Hypothermia is not a contraindication for anticoagulants or anti-platelet agents. Mild to moderate hypothermia (32-34°C) does not increase the risk of arrhythmias.



Correction of ABG

Correct ABG for temperature

Correction of blood gas values:

- **pO₂**: for every °C below 37°C: subtract 5 mm Hg from the value as measured in the lab. Example: Lab value pO₂ 90 mmHg; patient core temp = 32°C; corrected pO₂ level = 65 mmHg
- **PCO₂**: for every °C below 37°C subtract 2 mm Hg from the value as measured in the lab. Example: Lab value pCO₂ 35 mmHg; patient core temp = 32°C; corrected PCO₂ level = 25 mmHg.
- **pH**: for every °C below 37°C add 0.012 units to the value as determined by the lab. Example: Lab pH 7.20, patient core temp = 32°C, corrected pH value = 7.26.



Eligibility for Induced Hypothermia- Relative

Any relative contraindications?

Consider the following conditions relative contraindications to the institution of hypothermia:

- Active bleeding with the cause not (yet) under control
- Greatly increased risk of bleeding (e.g. injury of the spleen or liver)
- Cardiac arrest more than 12 hours ago (consider fever prevention rather than hypothermia)

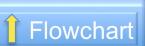


Eligibility for Induced Hypothermia- Minor

Is there a minor contraindication?

Consider the following conditions minor contraindications to the institution of hypothermia:

- Known presence of cold agglutinins (usually only if temp < 31°C)



Eligible for Hypothermia?

Establish Target Temperature and Method

Eligible patients should be comatose (eyes closed, unresponsive), have spontaneous circulation restored, and not have any contraindications. These contraindications range from minor to absolute.

If no absolute contraindications, consider the following when determining what Target Temperature to which you should cool the patient:

Relative contraindications to the institution of hypothermia:

- Active bleeding with the cause not (yet) under control
- Greatly increased risk of bleeding (e.g. injury of the spleen or liver)
- Cardiac arrest more than 12 hours ago (consider fever prevention rather than hypothermia)
- Weigh Risks and set Target Temperature to 32-34°C, or to 35°C if bleeding or bleeding risk

Minor contraindications:

- Known presence of cold agglutinins (usually only if temp < 31°C)
- Set Target Temperature to 32-34°C, and consider using endovascular cooling as the preferred method

No contraindications

- Set Target Temperature to 32-33°C



End of Hypothermia

Finished rewarming at 36.5°C

Rewarming is completed when core temperature reaches 36.5°C.

- Begin controlled euthermia
- Switch off cooling device; if temperature increases to >37.5°C re-start cooling, set target temperature at 36.5°C. If temp >37.8°C infuse 500-1000 ml of cold fluids.
- Combat shivering as described above.



Maintain Euthermia

Patient is contraindicated from cooling



Maintenance Phase

Temp is now < 34°C

- Duration of maintenance phase: usually 24 hours
- Keep target temperature within narrow range (32.0 °C, 33.0 °C or 34.0 °C)
- With paralysis, temperature may overshoot target temperature immediately following induction phase by about 1.0°C
- Use cooling device with controlled feedback system, set at target temperature
- Temperature should never decrease below 30°C
- If temperature increases to 1 degree or more above target temperature: cause is usually shivering, carefully screen patient, give (extra) anti-shivering medication
- In general: target MAP = 80 mm Hg, heart rate 36-100 BPM

Checklist:

- Continuous monitoring of blood pressure and heart rhythm
- Lab: Glucose (conform insulin protocol); ABG, K, Mg, Phos, lactate every 6 hours; PT, CBC every 12 hours
- Target electrolyte levels: (normal/high normal) K > 4.0 mmol, Mg > 2.0 mg/dl (1.0 mmol/l), P > 3.0 mg/dl



Minor Contraindication Exists

Patient has history of cold agglutinins

Consider cooling to 3G-3I °C

- If possible avoid surface cooling, especially of the extremities (as local blood temperature may drop below 31°C). Apply core cooling and skin counter-warming especially of the extremities.



Monitoring

Consider these monitors

- Continuous EEG
- Esophageal Doppler
- CVP monitoring



Physiological Parameters

Maintain

Maintain the following physiological parameters:

- MAP > 80 mm Hg
- HR 36-100 BPM
- Sedation: Ramsay score 4-5; Sedation-agitation scale 2-3; Motor activity assessment scale 0-1
- PO₂ corrected for temperature: > 65 mmHg
- PCO₂ corrected for temperature: 32-40 mmHg
- K >4.0; Mg > 2.0; P >3.0; Glucose 80-200 mg/dL; Hb>9.0; Platelets > 30



Proceed with Therapeutic Hypothermia

Start standard protocol for therapeutic hypothermia.

- Perform checklists 1 and 2, then proceed to hypothermia induction.



Relative Contraindication

One or more relative contraindications exist

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Resuscitation following Cardiac Arrest

Institution of hypothermia for comatose survivors

Resuscitation following Cardiac Arrest:

A patient who remains comatose following return of spontaneous circulation may benefit from induced hypothermia. This protocol addresses the initiation of hypothermia for such patients. This protocol does not address the standard ACLS protocols for cardiac resuscitation.

Topic Co-Chairs: Jon Rittenberger, MD Kees Polderman, MD



Rewarming

Warming the patient to euthermia

After 24 hours of cooling, begin re-warming.

- Duration of re-warming phase usually 12-24 hours.
- Warming speed $0.1\text{-}0.3^{\circ}\text{C}/\text{hour}$. Absolute maximum $0.5^{\circ}\text{C}/\text{hour}$; avoid more rapid warming.
- Perform controlled re-warming using a cooling device with a feedback mechanism.
- Points of attention: beware of hyperkalemia (in particular in case of rapid warming); hypoglycemia (due to increase in insulin sensitivity during re-warming).
- Hypotension may occur during re-warming, usually due to hypovolemia

Checklist during rewarming:

- Monitor blood pressure and heart rhythm
- Lab: ABG, K, glucose every 3 hours; Mg, P every 6 hours
- Target electrolyte levels: (normal/high normal) K > 4.0 mmol, Mg > 2.0 mg/dl (1.0 mmol/l), Phos > 3.0 mg/dl



Shivering Protocol

Methods to stop shivering

Shivering can be suppressed by several techniques:

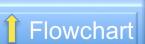
During Induction:

- Check ventilator settings such that sedation or chemical paralysis will not worsen P_aCO_2
- Propofol infusion 20-50 $\mu g/kg/min$ IV (as BP tolerates)
- Then add Fentanyl infusion 25-100 $\mu g /hr$.
- If not successful, add Diazepam 10-20 mg IVP
- Consider magnesium sulfate 4 gm IV over 15 minutes and single dose vecuronium 0.1 mg/kg IVP for induction

Maintenance:

- See above and consider midazolam 2-6 mg/hr

Avoid continuous paralysis unless continuous EEG in place



Side Effects

Hypothermia induced complications

Most important side effects:

- Bradycardia: usually no treatment necessary. Normal heart rate at a core temperature of 32°C is 34-40 BPM. If treatment is deemed necessary use Isoproterenol or dopamine infusion. Atropine is INEFFECTIVE for hypothermia-induced bradycardia.
- Shivering: Fentanyl 50-100 µg; Mg 2-4 grams; Skin counter warming, especially of hands, feet and face.
- Cold diuresis: replace lost fluids.
- Electrolyte disorders: replace, target normal levels, high Mg levels.
- Arrhythmias. Arrhythmias due to hypothermia occur ONLY if core temperature decreases below 30°C. If this occurs re-warm rapidly to temp > 30°C, and then slowly to target temp. If core temp is > 30°C arrhythmias do NOT require any change in cooling therapy. Treat arrhythmias with standard antiarrhythmic medications. Beware of possible decrease in clearance of amiodarone during hypothermia. Beware of decubiti due to skin vasoconstriction and immobilization.



Start Cooling Protocol

No contraindications were found

Start standard protocol for therapeutic hypothermia.

- Cool to 32-33°C for 24 hours.
- Perform checklists 1 and 2, then proceed to hypothermia induction.

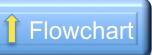


Start Hypothermia

Induction Phase

Start hypothermia induction; normal duration of induction phase is 60-120 minutes.

- Start infusion of cold fluids (4°C) WITH A PRESSURE BAG as rapidly as possible.
Type of fluid: saline 0.9%. Volume required usually 2,000-2,500 ml, but may require up to 4,000 ml.
- In case of cardiogenic shock/left ventricle failure: reduce bolus infusion to 1,000 ml per hour.
- Options to control shivering: fentanyl 1 µg /kg/hr IV; remifentanil continuous infusion; midazolam 2-5 mg IV; propofol infusion; diazepam 10-20mg IV; magnesium 2-4 grams IV (up to a serum level of 6 mg/dl (3 mmol/l); consider single-dose paralysis in case of refractory shivering.
- In general: avoid hypotension during hypothermia treatment. Target MAP 80 \geq mmHg



Temperature below 31°C

Did the temperature fall below target?

In cases of over-cooling:

- Temporarily increase the target temperature of the cooling device to 34.0°C until core temp > 31.5°C.

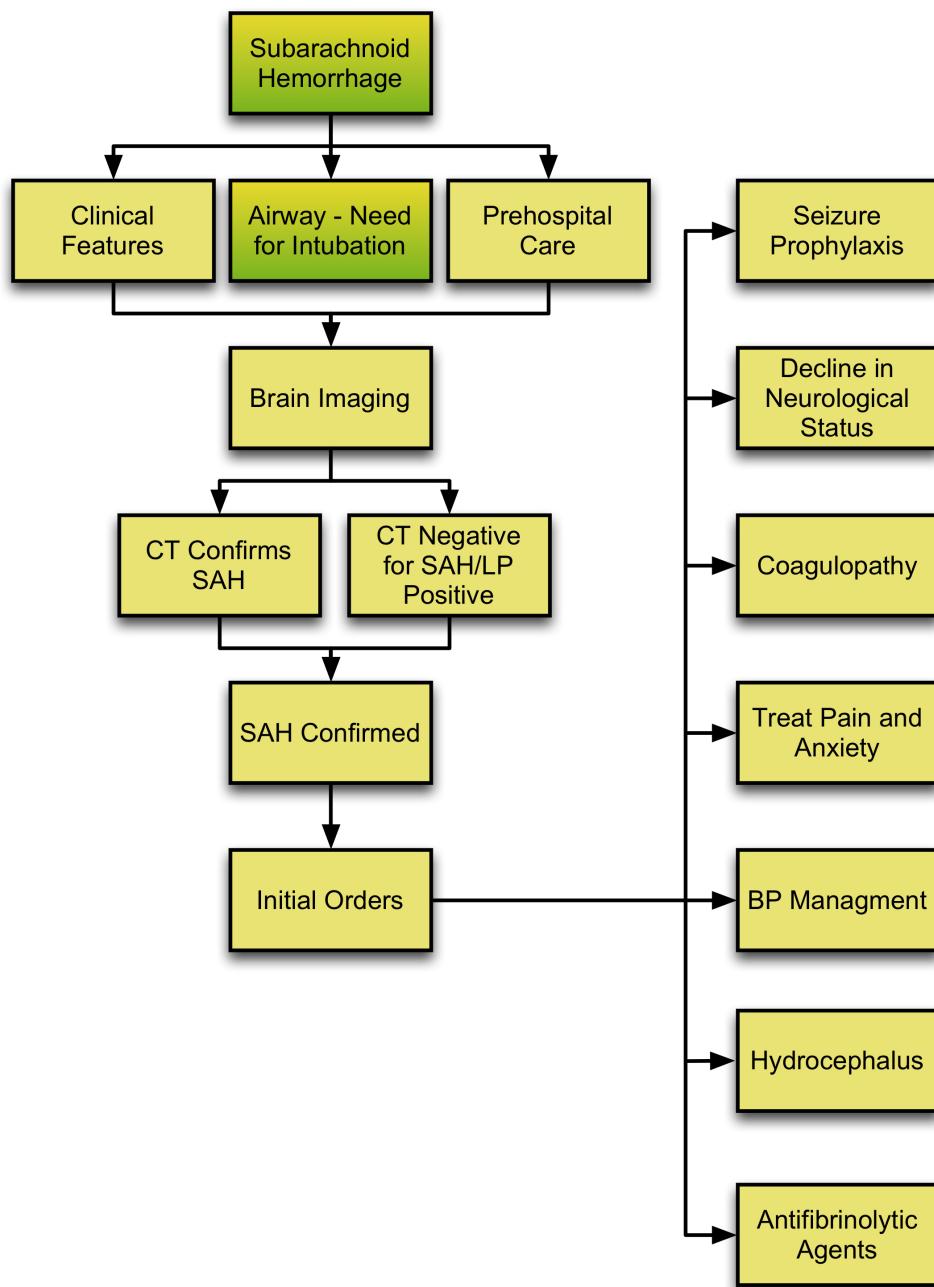
Emergency Neurological Life Support

All Protocols

Subarachnoid Hemorrhage

Version: 1.0

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[Checklist & Communication](#)

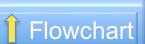


Checklist

- Brain Imaging
- Labs: PT/PTT, CBC, electrolytes, BUN, Cr, troponin
- 12 lead ECG

Communication

- Clinical presentation (level of consciousness, motor exam, pupil exam)
- WFNS score and Hunt-Hess Grade
- Imaging/LP results
- Hydrocephalus present?
- Airway status
- Sedation and other meds given
- Coordination of other vascular imaging



Antifibrinolytic Agents

Stop leak?

Preventing re-rupture of the aneurysm is a major goal of initial therapy.

- Antifibrinolytic agents such as amicar and tranexamic acid can reduce aneurysmal re-rupture. However, these agents also raise the risk of DVT, PE, and ischemic stroke if they are continued. If the patient is free of recent MI, DVT/PE or any known hypercoagulable state, many centers administer antifibrinolytic agents until the aneurysm can be secured; this strategy appears safe (Hillman et al, J Neursurg (2002) 97:771).

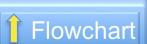


BP Management

Avoid hypertension to prevent re-rupture

General principles:

- Precise guidelines for BP management do not exist (see Bederson et al, Guidelines for the management of aneurysmal SAH; Stroke (2009) 40:994)
- Many specialists recommend SBP < 140 in a patient with no history of hypertension. SBP > 150 has been associated with aneurysmal re-rupture, and over treatment of BP can lead to brain ischemia (especially if hydrocephalus is present).
- Use short acting, titratable medications such as labetalol or nicardipine
- Avoid long-term nitroprusside due to concern of raising ICP



Brain Imaging for SAH

If you suspect SAH by history head imaging is the next step

Non-contrast CT imaging of the brain is the gold-standard for identifying SAH (Class1, LOE B).

- However, CT imaging is more sensitive in the first hours following a SAH and becomes progressively less sensitive with the passage of time (so that by 3 days, it is approximately 85% sensitive). Besides time, other reasons for a false negative CT include anemia, low volume SAH and a technically poor scan.
- Some physicians advocate a CTA at the time of the CT scan to look for an intracranial aneurysm. Although this is helpful if an aneurysm is seen, the negative predictive value is less clear. One should not use a negative CTA alone to rule out SAH.
- MRI is useful in patients who are imaged a few days following the SAH; specific sequences can be used to image subarachnoid blood even several days later.

A CT image of a SAH is shown below





Clinical Diagnosis of SAH

Clinical features

The diagnosis of traumatic SAH is based on history and brain imaging. The protocol for management of traumatic SAH can be found under the ENLS protocol [Traumatic Brain Injury](#).

Aneurysmal SAH has a classic and not so classic presentation.

Classically:

- Abrupt onset of a sudden, severe headache; onset is typically less than 1 second
- The headache is a NEW, QUALITATIVELY DIFFERENT headache for the patient
- May have neck pain, nausea and vomiting
- The patient may transiently lose consciousness, or present in coma
- The nature and onset of the headache is the key distinguishing feature from other forms of stroke, syncope, and seizure. A not-so-classic presentation:
- Headache is not reported as abrupt (the patient may not remember the event well)
- Headache responds well to non-narcotic analgesics
- Headache goes away on its own within hours
- Approximately 40% of patients with SAH will have a normal neurological examination. They may but may not have meningismus. They do not necessarily appear acutely ill.

Key Examination Features:

- [Glasgow Coma Scale](#) (GCS)
- Pupil exam
- Fundoscopic exam for retinal hemorrhages
- Neck exam for meningismus

Determine the clinical severity of the subarachnoid hemorrhage using one of the scales below:

World Federation Neurological Scale (WFNS):

Grade 1: GCS 15

Grade 2: GCS 13-15 without neurological deficit

Grade 3: GCS 13-15 with neurological deficit

Grade 4: GCS 7-12

Grade 5: GCS 3-6

Hunt-Hess Scale:

- Grade 1. Asymptomatic, mild headache, slight nuchal rigidity
- Grade 2. Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy
- Grade 3. Drowsiness / confusion, mild focal neurologic deficit
- Grade 4. Stupor, moderate-severe hemiparesis
- Grade 5. Coma, decerebrate posturing



Coagulopathy

Elevated INR or low platelets?

For platelet count < 50,000, administer 6-pack platelets.

Consider vitamin K antagonist reversal with purified factor concentrates or FFP if warfarin or other vitamin K antagonists have been prescribed, followed by Vitamin K 10 mg IV. To calculate the volume of plasma or IU of prothrombin complex concentrate:

1. Decide on target INR
2. Convert INR to percent (%) functional prothrombin complex:

INR Range	Percent function prothrombin complex
> 5	5%
4.0 – 4.9	10%
2.6 – 3.9	15%
2.2 – 2.5	20%
1.9 – 2.1	25%
1.7 – 1.8	30%
1.4 – 1.6	40%
1.0	100%

3. Calculate dose:

(Target in %PC - Current level in %PC) X weight (kg) = mL of FFP or IU of prothrombin-complex concentrate (PCC) needed

Example: a patient with INR on arrival = 7.5, target INR 1.5, body weight = 80 kg:
 $(40-5) \times 80 = 2,800$

Therefore, the needed dose is 2,800 mL of FFP or 2,800 IU of PCC.

Reference: Schulman, S. Care of patients receiving long-term anticoagulant therapy. NEJM (2003) 349:675



CT Scan Confirms SAH

Blood is seen on the CT scan

The diagnosis of SAH is confirmed and spinal fluid analysis is not necessary.



CT Scan is Negative - Do LP Next

Must do an LP if the CT is negative

The sensitivity of CT for recent SAH is nearly 95%. However, that means that you can miss 5 out of 100 patients with CT alone. If the patient's complaints suggest SAH, you are obligated to perform a lumbar puncture to look for evidence of subarachnoid blood products (Class 1, LOE B).

The LP is done to look for xanthochromia. Xanthochromia is the staining of CSF by heme breakdown products (chiefly bilirubin) by ependymal xanthene oxidase. It takes several hours for blood in the subarachnoid space to break down, so the presence or absence of xanthochromia is time dependent.

- If the CSF shows xanthochromia, the diagnosis of SAH is confirmed (be careful if the CSF protein exceeds 100 mg/dL as this can be a false positive).
- If the CSF is clear of RBCs and xanthochromia is absent, it is highly unlikely that the patient had a subarachnoid hemorrhage. However, a rapidly expanding aneurysm without subarachnoid rupture can present with a classic headache, so if you still suspect an aneurysm on clinical grounds, emergent consultation is suggested.
- If the CSF shows RBCs in the 1st and 4th tube of equal amounts, and the LP was done in the first few hours of the onset of headache, SAH is likely.

Stated otherwise, the typical findings of SAH on spinal fluid analysis are:

- RBCs
- < 5 WBCs
- WBC:RBC ratio 1:700
- Xanthochromia is present
- Minimal clearing of RBCs between tubes 1 and 4.

Atypical or inconclusive findings:

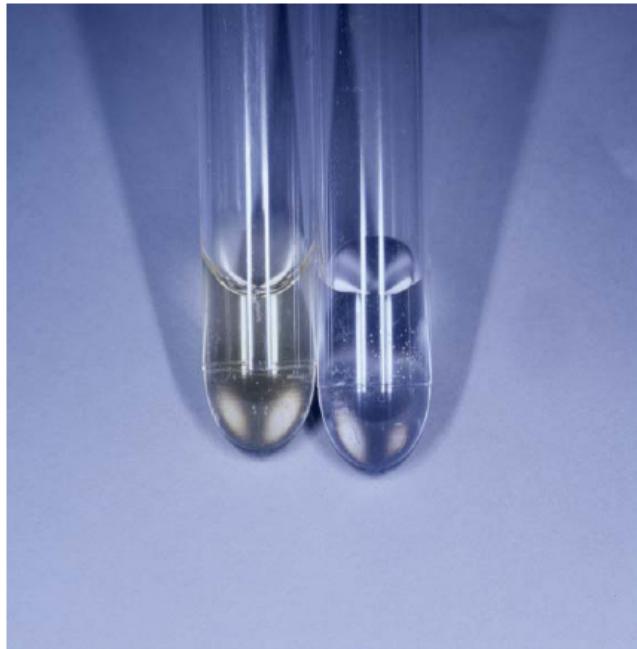
- Clearing of RBCs from tube 1 to 4 (perhaps because the spinal needle caused venous bleeding on the way in: traumatic tap)
- RBCs present in similar number in Tubes 1 and 4 but the LP was done within the first 4 hours of the headache (could be SAH or traumatic tap)
- Xanthochromia is absent, and the LP was done more than 12 hours following the onset of headache (bleed was too small to produce much xanthochromia)
- Excessive WBCs (ratio WBC:RBC > 1:700) suggesting meningitis or encephalitis

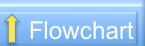
Note:

- The sensitivity of all tests for SAH are dependent upon the time from the bleed. CT is more sensitive early and less so with time. RBCs in the spinal fluid is also more

likely to be seen early and they will clear with time. Xanthochromia is absent early and nearly always present by 12 hours after the bleed.

Typical appearance of xanthochromia (CSF is spun first to take any RBCs out of solution):





Hydrocephalus

Are the ventricles dilated?

Hydrocephalus is caused by blockage of CSF absorption and is diagnosed by interpreting the head CT scan. If a patient is obtunded or comatose, it is important to provide ventricular drainage by having an external ventricular drain (EVD) placed by a neurosurgeon or neurointensivist. This both treats the hydrocephalus and provides a monitor of ICP.

- If you do not have a neurosurgeon and hydrocephalus is present, consider treating the patient with mannitol 1 gm/kg and expediting transfer to a facility with neurosurgical capability within the next hour.



Initial Orders

First steps

Once SAH is diagnosed, take these first steps:

- Bed rest (Class 2B, LOE B)
- Obtain pre-operative labs: CBC, Platelets, PT/PTT, electrolytes, BUN, Cr, cardiac enzymes
- 12-lead ECG
- Cardiac telemetry
- Nimodipine 60 mg po/ng (watch for hypotension)



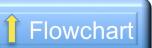
Intubation

Assess need for intubation

Factors that should be considered when deciding to intubate include:

- Not protecting airway
- Hypoventilation
- Hypoxemia
- Expected decompensation during transport within hospital or to another hospital

See ENLS protocol [Airway, Ventilation and Sedation](#).

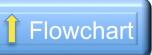


Neurological Exam has Declined

Worsening neurological examination?

There are several immediate causes of early (within the first hour) neurological decompensation.

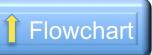
- Re-rupture of the aneurysm; repeat head CT is diagnostic
- Worsening hydrocephalus; repeat head CT is diagnostic; need for external ventricular drain is now paramount; give mannitol while arranging for EVD placement
- Seizure- treat with phenytoin load
- Cardiopulmonary cause- neurogenic pulmonary edema, catecholamine cardiomyopathy; worsening hypoxia (get CXR), falling BP consider urgent echocardiogram; cardiovascular collapse may also be a sign of cerebral herniation from re-rupture or untreated hydrocephalus.



Prehospital Issues Regarding SAH

Prior to hospitalization

See ENLS protocol [Acute Stroke](#) for a prehospital protocol pertaining to SAH and other forms of stroke.



SAH is confirmed

CT or LP evidence of SAH

Diagnosis of SAH is confirmed. The goal is to reduce the chance of aneurysm re-rupture and expedite treatment of the aneurysm while preventing any medical complications.



Seizure Prophylaxis

Should one prescribe AEDs now?

Use of prophylactic anticonvulsants is controversial.

- Pro: seizures following SAH and before definitive aneurysm treatment have been associated with re-rupture, and can raise ICP.
- Con: Phenytoin use has been associated with worse cognitive outcomes

One strategy is to administer a loading dose of phenytoin in the ED, and continue it until the aneurysm is secured, then stop the medication unless seizures have occurred (Class 2B, LOE B).



Subarachnoid Hemorrhage (SAH)

Blood within the subarachnoid space

Subarachnoid Hemorrhage (SAH) is most commonly produced by trauma and next most common by a ruptured intracranial aneurysm. For the latter, it is imperative that a timely diagnosis is made because the prevention of aneurysm re-rupture can be life saving.

Topic Co-Chairs: Jonathan A. Edlow, MD Owen Samuels, MD



Treat Pain and Anxiety

An uncomfortable patient can re-rupture their aneurysm

It is important to avoid straining, Valsalva, and writhing as this can cause re-rupture of a tenuous aneurysm. One must also be careful to not over-sedate the patient as one could mask the symptoms of hydrocephalus (obtundation).

- Use IV medication with short half-lives (fentanyl for example)
- Liberal use of anti-emetics is justified especially if vomiting occurs
- BP control is enhanced with adequate analgesia.
- If anxiety seems to be the major issue, consider small doses of an anxiolytic such as lorazepam.

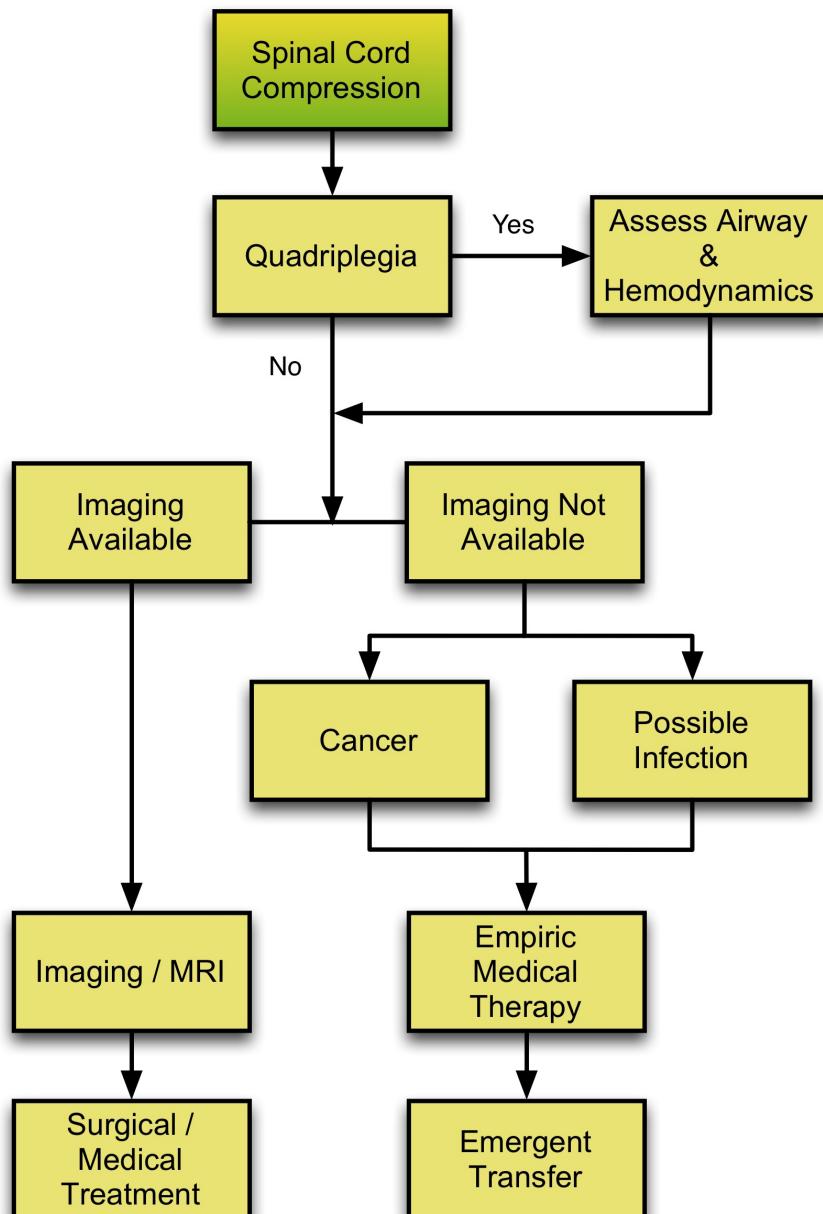
Emergency Neurological Life Support

All Protocols

Spinal Cord Compression

Version: 1.0

Last Updated: 5/23/2013



[Checklist & Communication](#)



Checklist

- Quadriplegia? Ensure proper ventilation
- Attain emergent spine imaging (MRI unless contraindicated)
- Alert spine surgeon if indicated
- Labs: CBC, platelets, PT,PTT
- NPO if expected to go to OR
- Suspected metastasis: contact radiation oncology; give steroids if spinal metastasis and cord compression confirmed
- Suspect epidural infections: ESR, start antibiotics

Communication

- Airway status
- Any significantly abnormal vital signs
- Onset and duration of weakness or numbness, and last neurological exam
- Bowel or bladder involvement
- Suspected spinal level
- Results of spine imaging if available yet
- Systemic illness like malignancy or infection
- Any medications started
- Ask if there is any other therapy they would like started immediately

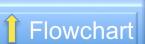


Arrange for Emergent Transfer

To a facility that has spine imaging available

Arrangement of expedited transfer to a facility with spine imaging may help expedite spinal decompression.

- Clearly communicate the urgency to the receiving physician
- The receiving physician should pre-arrange the imaging study so that valuable time is not lost

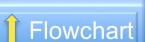


Assess Airway and Hemodynamics

Cervical myelopathy may affect diaphragm

Assess ventilatory functions (ABG, simple inspection, EtCO₂) and consider airway protection and mechanical ventilation. A bedside Forced Vital Capacity is helpful if available (intubate for FVC < 1 L); having the patient count out loud as fast as possible is also a good screen (normals should be able to exceed 20-30 count).

- Patients with total body weakness (unable to move the face and arms and legs) either has a generalized neuromuscular disorder, or perhaps a stroke of the brainstem (locked in). Secure the airway first then pursue the ENLS protocol [Acute Weakness](#), or ENLS protocol [Acute Ischemic Stroke](#). Once ventilation has been assessed, move on to acute imaging, but for those patients who are not intubated, anticipate progression of weakness and ensure continuous monitoring of ventilation as the work-up continues.



Empiric Medical Therapy

Steroids, Antibiotics

If suspicion for an epidural abscess is present then consider starting empiric antibiotics.

- recommended antibiotics

Administration of steroids (dose) may rapidly shrink the tumor preventing spinal cord damage for several hours.

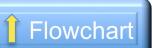
- Consider solumedrol 1 gm IV now

Empirical Treatment for Presumed Infectious Cause

Patients with evidence of infection such as fever, leukocytosis, intravenous (IV) drug use, or a known infectious source should be started on empiric antibiotics after blood cultures are drawn. Anti-microbial coverage should include staphylococcus, streptococcus, and methicillin resistant staphylococcus aureus (MRSA). If there is a history of a recent neurosurgical procedure, coverage for gram negative organisms should be added. These empiric therapies may be coordinated with the accepting facility's physicians.

Empirical Treatment if Cancer is Suspected

Steroids are often given to rapidly shrink edema and reduce the chance of cord venous infarction. Methylprednisolone may be given at 30 mg/kg IV bolus followed by 5.4 mg/kg/hr by 23 hours.



Evidence of Infection

Consider epidural abscess

Suspicion for an infectious cause (epidural abscess) rises if the following are present:

- Fever
- Elevated WBC count
- History of intravenous drug use
- Known infectious source- current or past endocarditis, sepsis, chronic infection like osteomyelitis
- Any of the above with focal spine tenderness elicited by percussion (reflex hammer striking your finger placed over the vertebral spinous process)



Hemorrhage

Epidural hemorrhage or other

Bleeding in the epidural space may be spontaneous or from underlying coagulopathy.

- Contact a spine surgeon immediately and present to key features listed in communication
- Rule out coagulopathy
- Labs: PT/PTT, platelets, consider DIC screen and blood smear for red cell analysis
- Reverse warfarin associated coagulopathy (see ENLS protocol [Intracerebral Hemorrhage](#) discussion on reversal of coagulopathy)
- Intramedullary bleeding (bleeding into the spinal cord) may be due to an underlying vascular malformation and will likely require additional imaging studies if the etiology is not otherwise apparent (repeat MRI, spinal angiography)



History of Cancer

Possible metastasis

Consider spinal metastasis with spinal cord compression if there is a history of cancer, or new suspicion of cancer.



Imaging is Available

MRI or CT

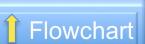
Emergent MRI with gadolinium is preferred in most cases.

- CT with contrast and or CT myelogram is an alternative if MRI is contraindicated or not available.



Imaging Negative

Likely intrinsic cord pathology

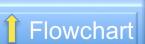


Imaging Not Available

No MRI or CT

Without imaging, one needs to consider presumptive treatments that can be put in place to temporize until imaging can be made available.

- If there is a history suggesting infection so that epidural abscess is a possibility then one should consider empiric antibiotics
- If there is a history of cancer so that spinal metastasis and cord compression is a possibility, one should consider empirical steroids
- If neither is present, an expedited transfer to a facility with imaging capability is warranted.

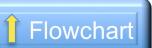


Imaging with MRI or CT

Imaging is used to rule out any compressive etiology of the spinal cord like tumor, infection, or intervertebral disc herniation. It is important to communicate the neurological findings to your radiologist so that the proper location(s) of relevance are imaged.

- Quadriplegic patients should have the C-Spine imaged. Entire spine imaging (including the conus) may also be appropriate especially if the patient has known cancer.
- Paraplegic patients (if there are no symptoms in the arms) should have both the T-spine and LS spine imaged. Reflexes are likely unreliable in this context in guiding whether to include T-spine imaging; i.e. rapid compression of the T-spine can cause hyporeflexia in the lower extremities acutely, so an areflexic paraplegia is not necessarily a cauda equina syndrome (which localizes to the LS spine on imaging). A discussion with the radiologist is important to image the proper level, and to expedite the imaging so that treatments can be provided efficiently and quickly.

It is also important to notify a spine surgeon at this point to alert them that your patient may have a myelopathy that will need surgical decompression, and when their spine imaging will be completed.



Infection

Epidural abscess

Imaging reveals a likely abscess:

- Contact a spine surgeon immediately and present to key features listed in communication
- Start empirical antibiotics based on the patient's risk factors; discuss this with your consultant as this may reduce the likelihood to get positive culture results
- Labs: ESR, blood cultures, UA, urine culture (prior to any antibiotics)
- 12- lead ECG looking for PR prolongation (could indicated endocardial abscess); if prolonged consider cardiology consult
- Consider echocardiogram looking for valvular vegetations
- Use of steroids is controversial; discuss with your consultant
- Spinal cord decompression is possible but often not done emergently to give time to observe a response to antibiotics. Document the neurological exam (primarily strength testing) well to establish a good baseline from which to make this decision.

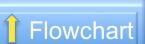


Medical and Surgical Treatment

Based on what is seen on MRI

Imaging may reveal no evidence of cord compression. If so, other causes of myelopathy need to be considered including transverse myelitis, spinal cord infarction, viral myelitis (West Nile, CMV, HIV, HTLV-1), dural AV fistula, and others. Refer to the ENLS protocol [Acute Non-Traumatic Weakness](#) for a discussion of these entities, and especially consider aortic dissection. Spine imaging may reveal several compressive etiologies, including:

- Metastatic disease: metastatic disease to the spine is not an uncommon presentation of some cancers. There is usually spinal tenderness over the regions involved. Treatment with high dose corticosteroids (methylprednisolone 30 mg/kg IV bolus followed by 5.4 mg/kg/hr by 23 hours) can help shrink tumor edema and maintain cord health until surgical decompression is available. STAT involvement of a spine surgeon and radiation oncology is imperative, and facilitation of transfer elsewhere if none is available.
- Spinal hemorrhage: spontaneous epidural bleeding is uncommon but may present in patients without coagulopathy and no other predisposing conditions. Treatment may involve surgical decompression. STAT involvement of a spine surgeon is imperative, and facilitation of transfer elsewhere if none is available. If there is a coagulopathy present, following the ENLS protocol on [reversal of coagulopathy](#).
- Acute disk herniation: disk herniation that compresses the spinal cord or the cauda equina may represent a neurosurgical emergency. Treat with methylprednisolone 30 mg/kg IV bolus followed by 5.4 mg/kg/hr by 23 hours, and STAT involvement of a spine surgeon is imperative, and facilitation of transfer elsewhere if none is available.
- Epidural abscess: pus in the epidural space likely causes myelopathy by venous infarction rather than actual cord compression but the clinical signs and symptoms are identical. STAT involvement of a spine surgeon is imperative, and facilitation of transfer elsewhere if none is available. Draw blood cultures, look for signs of endocarditis, perform a 12-lead ECG (to look for PR prolongation), and consider starting empirical antibiotics in consultation with the surgeon and perhaps infectious disease consultant. Anti-microbial coverage should include staphylococcus, streptococcus, and methicillin resistant staphylococcus aureus (MRSA). If there is a history of a recent neurosurgical procedure, coverage for gram negative organisms should be added.



Neoplasm

Spinal neoplasm

The MRI or CT imaging shows cord compression from tumor

- Emergent decompression may be helpful to this patient so rapid involvement of specialists is key
- Contact a spine surgeon immediately and present to key features listed in communication
- Consult radiation oncology to consider emergent spinal irradiation
- Give glucocorticoids (solumedrol 10-30 mg/kg IV times one); clear this with your consultant first
- Pain management: short acting narcotics, consider airway issues if the process is cervical
- DVT prophylaxis: no heparin yet until surgical decision is complete; pneumatic compression stockings are appropriate
- If the neoplastic process is leptomeningeal (i.e. not directly compressing the cord but encasing the cord), decompression is likely not necessary but spinal fluid assessment is the next step. Consult oncology and consider LP

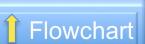


Quadriplegia

Special airway issues

In the event of sudden or progressive quadripareisis or quadriplegia, the cause may be a cervical cord pathology. This may lead to hypoventilation because of both chest wall and diaphragmatic weakness.

If the patient has paraplegia/paraparesis ventilatory issues are uncommon, so move on to imaging.



Spinal Cord Compression

Suspected myelopathy

Acute signs and symptoms of myelopathy

- Bilateral numbness or weakness that is present at a specific dermatomal level and continues caudally
- Weakness is of upper motor neuron variety (spastic, extensors effected more than flexors, up-going toes)
- Urine retention or spastic bladder
- May have focal back pain identified via percussion of the spine

Acute spinal cord compression is an emergency so work-up and intervention should begin immediately.

- If there is severe back pain and leg weakness consider aortic dissection as a cause; typically such patients will have intact joint position sense in the toes but loss of temperature sensation along with marked weakness (anterior spinal artery syndrome).

If the patient is taking anticoagulants, and emergent workup for coagulopathy is warranted. See ENLS protocol on [reversal of coagulopathy](#). See ENLS protocol [Approach to Non-Traumatic Weakness](#) for a more formal evaluation of the cause of weakness.

Topic Co-Chairs: Kristine O'Phelan, MD Brad Bunney, MD



Spinal Stenosis

Disk or bone encroachment on cord

Imaging reveals compression from disk herniation or from bone/vertebral body encroachment (spinal stenosis)

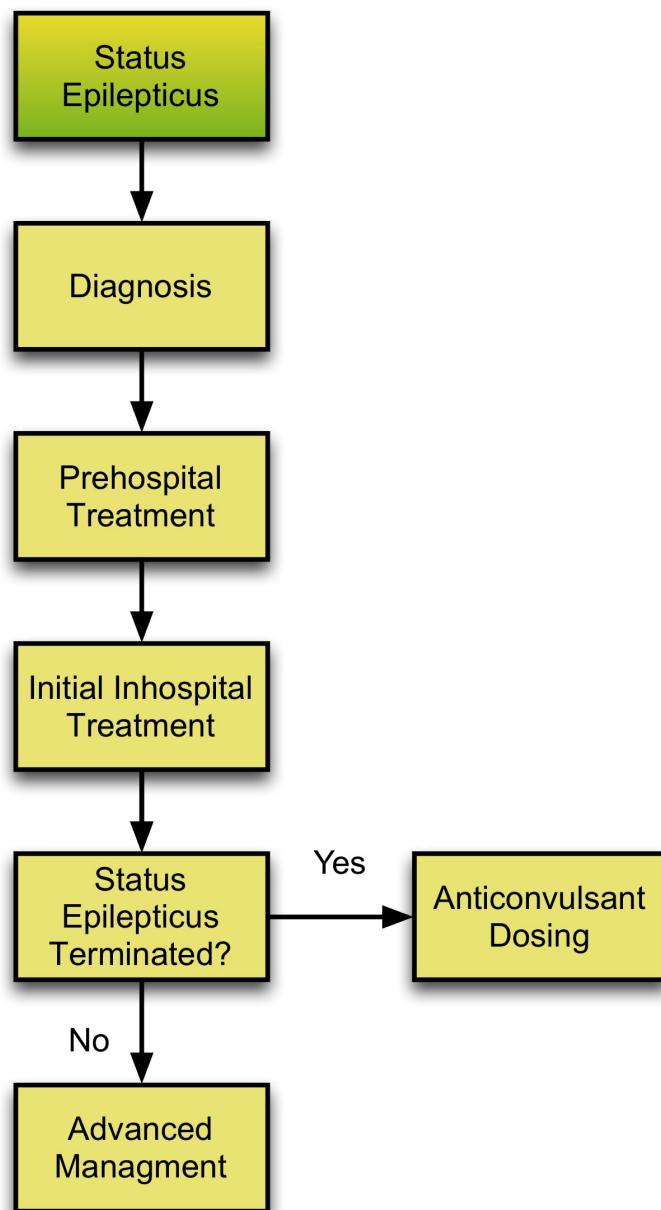
- Contact a spine surgeon immediately and present to key features listed in communication
- For sudden onset disk herniation with myelopathy, urgent decompression may be necessary; more chronic myelopathy may take a less urgent course
- Discuss use of glucocorticoids with your consultant

Emergency Neurological Life Support

Status Epilepticus Protocol

All Protocols

Version: 1.0
Last Updated: 5/23/2013



[Checklist & Communication](#)

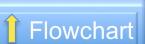


Checklist

- Fingerstick glucose
- Obtain IV access
- Monitor pulse oximetry, BP, cardiac; supplemental O₂ and fluid as needed
- Order labs: CBC, BMP, Ca, Mg, AED levels
- Head CT (appropriate for most cases)
- cEEG monitoring - Notify EEG tech if available (as soon as available unless patient returns to pre-status epilepticus baseline)

Communication

- Clinical presentation
- Duration of status epilepticus
- Relevant PMH/PSH
- Prior medications, medication given so far, AED levels if drawn
- Neurological examination
- Brain imaging/LP results (if available)



Advanced management

If status epilepticus has still not halted

If the patient is still convulsing despite benzodiazepines, phenytoin or valproate loading, and then third line agents described in the prior step, the patient will need more aggressive intervention. The patient should at least be intubated and transferring to an ICU at this point. Once in the ICU, establish good blood pressure monitoring, then begin pentobarbital.

Give:

- Pentobarbital: Load: 5 mg/kg IV up to 50 mg/min; repeat 5 mg/kg boluses until seizures stop; Initial rate: 1 mg/kg/hour; Maintenance: 0.5-10 mg/kg/hour traditionally titrated to suppression-burst on EEG but titrating to seizure suppression is reasonable as well
- Continuous EEG monitoring is essential; if not available in your center consider transfer to a regional center with this capability.

Comments:

- Hypotension is frequently encountered as a side effects of pentobarbital and pressors should be readily available. Other side effects include gastric stasis, myocardial suppression, thrombocytopenia, metabolic acidosis (68-75% propylene glycol).
- Often this step will be done in ICU setting but at times with patients that are highly refractory pentobarbital infusions may need to be started while in the ER and within the first hour of status epilepticus onset.
- The duration of continuous IV antiepileptic medications used for Boxes #5 and #6 is unclear. Once seizures are controlled, many physicians continue treatment for at least 24 hours prior to consideration of wean. The rapidity of weaning is also controversial but should not be done too abruptly unless pentobarbital is used where this is less of an issue. The goal of pentobarbital therapy is unclear (seizure control, burst suppression, or completely suppressed background)?
- The antiepileptic agent is controversial. Choices include but are not limited to ketamine (which should be used in combination with a benzodiazepine), lacosamide, levetiracetam, and hypothermia to 33 degrees Celsius.

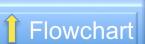


Diagnosis

The diagnosis of status epilepticus

The clinical definition of status epilepticus is five minutes or more of convulsions or two or more convulsions in a 5-minute interval without return to preconvulsive neurological baseline. However, a patient may be seen to seize, then brought into the hospital and not regain consciousness. This too may be status epilepticus and usually requires EEG measurement to diagnose.

Traditional definition of status epilepticus required 30 minutes to have passed. Do not wait for 30 minutes to pass before starting antiepileptic medications since permanent brain injury may occur before 30 minutes have elapsed and most seizures that do not progress to status will be shorter than 5 minutes.



Initial in-hospital management

If not already done pre-hospital

- ABCs, including supportive care if needed (O₂, airway, BP)
- Monitors: ECG, BP, O₂Sat
- Obtain IV access
- Draw labs: CBC, BMP, CA, Mg, AED levels. Additional orders for specific circumstances: Labs: PO₄, LFTS, Troponin, Toxicology screen (urine and blood), ABG, type and hold, coagulation studies
- Diagnose hypoglycemia: if hypoglycemic give D₅₀W 50 ml IV and thiamine 100 mg IV (may be given empirically if suspected in the absence of a definitive diagnosis)
- Give lorazepam 4 mg IV over 2 minutes or diazepam 5 mg IV
- Alternatives include: diazepam 20 mg PR (may use diastat or IV solution of diazepam), or midazolam 10 mg IN/buccal/IM/IV

Comments:

- First line benzodiazepines are frequently under dosed.
- Initiate a complete workup of the underlying etiology for status epilepticus. Seizures will be difficult to control with antiepileptic medications if they are caused by an underlying uncorrected metabolic problem.
- Wide availability and reliability of blood sugar testing does not support administration of D₅₀W to all patients that may worsen outcome in a number of acute brain injuries. However, hypoglycemia needs to be treated promptly if this is the underlying cause of status epilepticus.
- ECG, Chest X-ray
- Consider toxins that can cause seizures: INH (treat with lorazepam followed by pyridoxine 70 mg/kg; max dose 5 gm); tricyclics (look for QRS widening on the EKG, treat with sodium bicarbonate); theophylline; cocaine / sympathomimetics; alcohol withdrawal (rarely causes SE, treat with accelerating doses of a benzodiazepine); Organophosphates (treat with atropine, midazolam, and pralidoxime)
- Almost any agent in overdose may cause a seizure indirectly if they cause hypoxia, hypotension, or electrolyte (including hypoglycemia) abnormalities



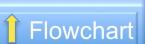
Pre-hospital treatment of status epilepticus

Prior to admission

- ABCs, including supportive care if needed (O₂, airway, BP)
- Obtain IV access
- Diagnose hypoglycemia: if hypoglycemic give D₅₀W 50 ml IV and thiamine 100 mg IV (may be given empirically if suspected in the absence of a definitive diagnosis)
- Give: lorazepam 4 mg/ IV over 2 minutes or diazepam 5 mg IV
- Alternatives include: diazepam 20 mg PR (may use diastat or IV solution of diazepam), or midazolam 10 mg IN/buccal/IM/IV

Comments:

- Time is control. Most important factor in predicting ease to control seizures is time elapsed prior to initiating AEDs. If unable to get intravenous access give benzodiazepines via alternate route.
- Respiratory decompensation is more commonly encountered in untreated status epilepticus than in status epilepticus treated with benzodiazepines.
- Weight based benzodiazepine administration may be appropriate in certain circumstances but is an off-label use, more prone to dose calculation error, and there are no data showing that it is superior
- Lorazepam needs to be refrigerated or restocked every 60 days. For this reason it is usually impractical for EMS use and diazepam or midazolam are used as alternatives.



Seizures have not stopped

Or they stopped but the patient will not awaken

Start second line anticonvulsant. Choose one of the following:

- Fosphenytoin: load 20 mg/kg IV at up to 150 mg/min
- OR -
- Valproic acid: load: 40 mg/kg IV over 10 min (may give additional 20 mg/kg over 5 min if still seizing)

Arrange for EEG monitoring if available.

If status epilepticus persists despite starting second line anticonvulsants, [intubate the patient](#), then choose one of the following:

- Continuous infusions of midazolam: load: 0.2 mg/kg IV over 2-5 min; repeat 0.2-0.4 mg/kg boluses every 5 minutes until seizures stop, up to a maximum loading dose of 2 mg/kg. Initial rate: 0.1 mg/kg/h. Bolus and increase rate until seizure control; maintenance: 0.05-2.9 mg/kg/hour
- Continuous infusions of propofol: Load: 1-2 mg/kg IV over 3-5 min; repeat boluses every 3-5 minutes until seizures stop, up to maximum total loading dose of 10 mg/kg. Initial rate: 33 µg/kg/min (2 mg/kg/hr). Bolus and increase rate until seizure control; maintenance: 17 - 250 µg/kg/min (1-15 mg/kg/hour)
- Valproic acid (if not chosen already as second line agent): 40 mg/kg IV over 10 min (may give additional 20 mg/kg over 5 min if still seizing)
- Phenobarbital: Load: 20 mg/kg IV up to 60 mg/min; maintenance: 1-3 mg/kg/day in 2-3 divided doses

Comments:

- Titrate AEDs to therapeutic levels. When checking post-load drug levels, wait at least 2 hours post infusion for fosphenytoin and phenytoin, or immediately post infusion of valproate
- Continue second line antiepileptic medication when starting treatment of refractory status epilepticus.
- Phenobarbital has been used traditionally for status epilepticus refractory to first and second line therapy but recently experts recommend more rapid advancement to continuous IV antiepileptic medications.
- What should the target for continuous drips be: seizure control, burst suppression, or completely suppressed background?
- Definition of refractory status epilepticus is unclear. Some controversy regarding duration of time and number of agents that patients have to have failed exists.



Seizures have stopped

And the patient is following commands

The half-life of benzodiazepines is brief and therefore a longer-lasting anticonvulsant needs to be administered to prevent subsequent seizures.

Give:

- Fosphenytoin: load 20 mg/kg IV at up to 150 mg/min
 - OR -
- Valproic acid: load: 40 mg/kg IV over 10 min (may give additional 20 mg/kg over 5 min if still seizing)

If possible connect to EEG unless patient wakes up or returns to pre-convulsive baseline. Determine the cause of the seizure (prior history of seizures and medication non-compliance, new onset seizure, etc.) Serum levels of anticonvulsants are useful to determine what threshold the patient with epilepsy has for developing seizures. Urine toxicology screen may be helpful for recreational drug-associated seizures.



Status Epilepticus

Unremitting seizures

Status Epilepticus: Ongoing seizure activity is injurious to the brain and can cause other organ system problems like pneumonia and sudden death. Making an accurate diagnosis is essential as is the orderly institution of anticonvulsant drugs to terminate the seizure activity. This protocol gives a practical, step-by-step guide to how status epilepticus can be terminated.

Topic Co-Chairs: Jan Claassen, MD Robert Silbergleit, MD



Status epilepticus terminated

Have the seizures stopped or the patient began following commands

Status epilepticus is terminated when the patient begins to follow simple commands. Even if the convulsions have stopped the patient may still be seizing. If the patient does not rapidly awaken following the administration of the first line anticonvulsants, one should consider the patient to still be seizing.

Emergency Neurological Life Support

All Protocols

Glasgow Coma Scale

Version: 1.0

Last Updated: 5/23/2013

Response	Score
Eye Opening	
Opens eyes spontaneously	4
Opens eyes in response to speech	3
Opens eyes in response to pain	2
Does not open eyes in response to pain	1
Motor Response	
Follows commands	6
Localizes pain	5
Moves to pain but not purposefully	4
Flexes upper extremities to pain	3
Extends all extremities to pain	2
No motor response to pain	1
Verbal Response	
Oriented to person, place and time	5
Confused speech	4
Replies with inappropriate words	3
Incomprehensible sounds	2
No verbal response	1
Total	1-15

Emergency Neurological Life Support

All Protocols

Hunt Hess Classification for SAH

Version: 1.0

Last Updated: 5/23/2013

Hunt Hess Grade	Criteria
1	Asymptomatic, mild headache, slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy
3	Drowsiness / confusion, mild focal neurologic deficit
4	Stupor, moderate-severe hemiparesis
5	Coma, decerebrate posturing

An alternative scale is the [World Federation Neurological Scale](#).

Fisher Group of SAH

It is common to report the Fisher Group (amount and location of subarachnoid blood) when discussing the severity of the SAH.

Fisher Group	Criteria based on CT Imaging
1	No subarachnoid blood seen
2	Diffuse or vertical layer of subarachnoid blood < 1 mm thick
3	Localized clot and/or vertical layer within the subarachnoid space > 1 mm thick
4	Intracerebral hemorrhage, or IVH with diffuse or now subarachnoid blood

Emergency Neurological Life Support

World Federation Neurological Scale

All Protocols

Version: 1.0
Last Updated: 5/23/2013

The World Federation Neurological Scale (WFNS) incorporates both the GCS score and features of the neurological exam.

WFNS Grade	Criteria
1	GCS 15
2	GCS 13-15 without neurological deficit
3	GCS 13-15 with neurological deficit
4	GCS 7-12
5	GCS 3-6

Although this score correlates better with clinical outcomes, most people still use the [Hunt-Hess Scale](#).

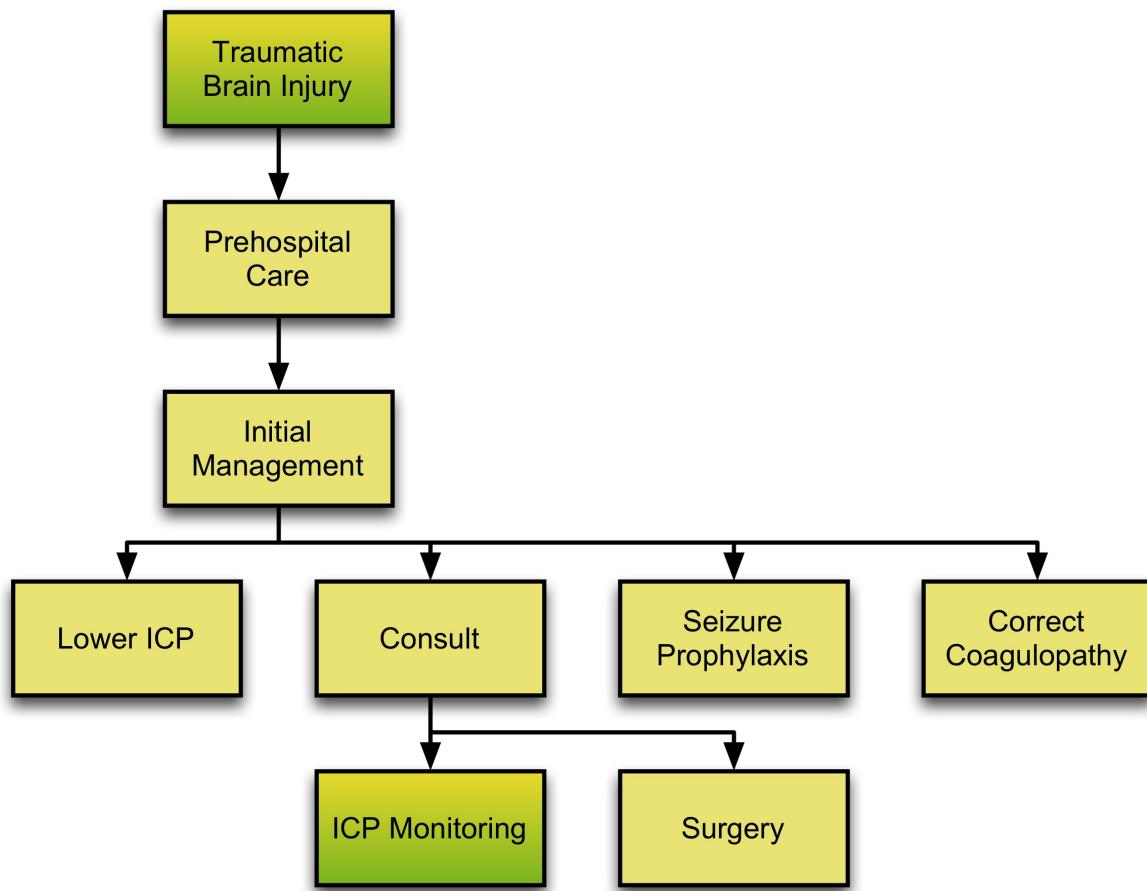
Emergency Neurological Life Support

All Protocols

Traumatic Brain Injury

Version: 1.0

Last Updated: 5/23/2013



[Checklist & Communication](#)

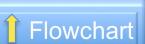


Checklist

- Airway
- SBP > 90 mmHg
- C-spine precautions
- Head CT
- Reverse herniation

Communication

- Patient age
- Pre-injury health, if the information is available
- Mechanism of Injury
- Post resuscitation GCS
- Pupil size, reaction, and symmetry
- Focal motor findings
- Coagulation status
- Other injuries
- State of cervical spine - cleared, not cleared, injury
- CT scan findings



Coagulopathy

Recognition and treatment

Indicated if known or suspected coagulopathy:

- recent elevated PT/INR/PTT
- low platelets
- history or physical examination consistent with end-stage hepatic or renal disease
- on anticoagulant therapy
- on antiplatelet therapy

Consider the following:

- Plasma and vitamin K - for patients on warfarin or with end-stage liver disease
- Platelets - for patients with conditions with low or malfunctioning platelets
- DDAVP - for patients with end-stage renal disease or on certain anti-platelet agents

Sidebar common pitfalls

- In most cases, reversal can begin immediately according to empiric guidelines and does not require laboratory values or confirmation.
- Reversal of anticoagulation is a complex subject, and in some cases, such as in patients with hemophilia and other bleeding dyscrasias, it may be necessary to obtain specialist consultation from a hematologist.
- Reversal of antiplatelet agents such as ASA, clopidogrel and ticlopidine is controversial, with some authors recommending the use of DDAVP.



Consults

Neurosurgery

Neurosurgical consultation may be necessary depending on the severity of the injury and the patient's clinical status. Findings that should prompt neurosurgical consultation include:

- GCS < 13
- Lateralizing findings on neurological examination, including unequal pupils or focal weakness
- Abnormal head CT scan
- CSF leak, or signs of basal skull fracture
- Penetrating skull injury



Diagnosis

What constitutes TBI?

Traumatic Brain Injury (TBI):

- Severe TBI: Mechanism consistent with TBI and/or physical signs of trauma in unconscious patient, with a Glasgow Coma Scale < 9.
- It is important to consider other treatable causes of decreased level of consciousness. Every attempt should be made to identify and reverse vascular, metabolic, infectious, environmental, toxicological and other nontraumatic causes. These causes may co-exist with TBI.
- The GCS should be obtained through interaction with the patient (e.g. by giving verbal commands or if those unable to respond by applying a painful stimulus)
- The GCS should be assessed after appropriate resuscitation and before the administration of sedative or neuromuscular blocking agents

Diagnosis of TBI - recognition of TBI depends on consideration of:

- Physiology (e.g. GCS)
- Anatomy (scalp laceration, depressed skull fracture)
- Mechanism of injury (e.g. fall > 20 feet, MVA > 30 mph)

Topic Co-Chairs: Stuart Swadron, MD Peter Le Roux, MD



ICP Management

Strategies to lower ICP

Indicated if signs of herniation develop in the unconscious patient. These include:

- dilated and nonreactive pupils
- asymmetric pupils
- motor exam that demonstrates extensor posturing or no response
- progressive decline in neurologic condition (decrease in GCS > 2 points) that are not associated with non-TBI causes
- Cushing's response (increased BP, decreased pulse and irregular respirations)

If signs of herniation are present, the patient should be treated presumptively for high ICP while simultaneously facilitating the placement of an ICP monitor (see next section). Initial treatment may include:

- Mannitol or hypertonic saline: Administer 20% mannitol 0.25-1 g/kg IV as a rapid (5 minutes) IV infusion; If BP (systolic) < 90 mmHg in adults, hypertonic saline rather than mannitol should be used - administer 3% NaCl 250ml IV over minutes
- Hyperventilation: Target a PCO₂ of 28-35 mmHg (20 breaths a minute in adult)
- Goal CPP (MAP-ICP) is ≥ 60 mmHg
- Additional crystalloid if ICP allows
- If lactate not elevated, norepinephrine 10-100 mcg/min once adequate volume is achieved, and add other pressors as needed
- Transfuse RBCs if active bleeding or Hgb < 7 gm/dl

Sidebar common pitfalls

- Hypotension (systolic BP< 90 mmHg) should prompt rapid discontinuation of mannitol.
- To administer hypertonic saline (5% NaCl), serum Na should be < 160 mEq/L



ICP Monitoring

Indications and treatment algorithm

Placement of an ICP monitor is indicated in the following clinical scenarios:

- GCS 3-8 and abnormal CT scan
- GCS 3 - 8 with normal CT & 2 or more of the following: a) age > 40 years, b) motor posturing, and c) SBP < 90 mmHg
- GCS 9-15 and CT scan showing mass lesion (extra-axial blood > 1 cm thick, temporal contusion, or ICH > 3 cm), effaced cisterns, or brain shift > 5 mm
- Following craniotomy
- Neurological examination cannot be followed e.g. requires another surgical procedure or deep sedation

Elevated ICP treatment (ICP > 20 mmHg for more than 2 min):

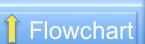
- Elevate HOB 30 degrees (as tolerated by MAP, ICP, $P_{bt}O_2$)
- Drain CSF (if available)

Open drainage until:

- ICP drops below 20 mmHg, or
- 5 ml CSF drains, or
- Drainage stops
- Repeat as needed; do not actively withdraw CSF

Meds: Continuous

- Analgesic - Fentanyl or Morphine
- Sedation - Propofol for 24 to 48 hours, then lorazepam
- Neuromuscular blockers only if shivering or bucking ventilator
- Control body temperature; avoid fever. Consider normothermia protocol
- Osmotherapy - in sequence - mannitol boluses, then 5% hypertonic saline boluses, then 3% NaCl infusion titrated according to Na level
- Hyperventilate to decrease P_aCO_2 as tolerated by $P_{bt}O_2$ & $S_{jv}O_2$ If ICP remains elevated despite these measures
- Additional propofol to EEG burst suppression
- Decompressive craniectomy
- Pentobarbital bolus then continuous infusion



Initial Hospital Management

If not done prehospital

- Spinal precautions to be maintained at all times
- Advanced airway management to ensure: a) airway protection to maintain oxygen saturation > 90%, b) control of ventilation (if inadequate or inappropriate)
- Continuous monitoring of oxygenation, blood pressure, cardiac rhythm and PCO₂
- Obtain parenteral access (IV or IO)
- Diagnose hypoglycemia: if hypoglycemic give D_{50%} 50 ml IV
- Obtain CT Head without contrast

Sidebar common pitfalls

- Although a Glasgow Coma Scale of 8 or less during the initial evaluation is an indication for endotracheal intubation; severe extracranial injuries or a rapidly declining mental status may also be indications.
- Patient can be ventilated with 100% O₂ until ABG values are available. Any adjustments should maintain S_aO₂ > 90%.



Prehospital

Evaluation and management in the field

- Spinal precautions to be maintained at all time
- Basic and advanced airway management as indicated to maintain oxygen saturation greater than 90%
- Normal breathing should be maintained ($E_{T}CO_2$ 35-40 mmHg) and hyperventilation avoided ($E_{T}CO_2 < 35$ mmHg) unless there are signs of herniation (see below) when hyperventilation is indicated (20 breaths per minute in the adult can be used as temporary measure until signs of herniation resolve)
- Continuous monitoring of oxygenation (pulse oximetry) and blood pressure
- In the adult, systolic BP should be > 90 mmHg
- Hypotensive patients should be treated with isotonic fluids
- Hypertonic resuscitation is an option
- Obtain IV access
- Diagnose hypoglycemia: if hypoglycemic give D_{50%} 50 ml IV
- Assess Glasgow Coma Score and pupils

Common pitfalls

- The use of neuromuscular blocking medications to facilitate intubation (rapid sequence intubation) in the field worsened outcomes in one large study. In the spontaneously breathing individual who maintains an $S_aO_2 > 90\%$ on supplemental oxygen, endotracheal intubation is not indicated. If it is performed for other indications, monitoring of oxygenation, blood pressure and end-tidal CO_2 should take place.
- Hypo- and hyperventilation should both be avoided. If $E_{T}CO_2$ measurement is available, this should be in the range of 35 to 40 mmHg.
- Hyperventilation to decrease PCO_2 to between 28-35 mm Hg is only indicated for patients with signs of herniation (rapidly decreasing LOC, particularly with changes in pupil reactivity)
- Intravenous fluid boluses (500cc to 1L of crystalloid or smaller volumes of hypertonic saline solutions) may be given in adult trauma victims with systolic BP < 90 mmHg or with signs of hypoperfusion (e.g. poor capillary refill)
- Pupils should be measured after resuscitation and evidence of orbital trauma noted
- Pupil asymmetry is defined as > 1 mm difference in diameter
- A fixed pupil is defined as < 1 mm response to bright light
- Signs of herniation include: dilated and nonreactive pupils, asymmetric pupils, motor exam that demonstrates extensor posturing or no response or progressive decline in neurologic condition (decrease in GCS > 2 points)

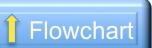


Seizures

Control and prevention

If seizure activity was witnessed, or the patient has a depressed level of consciousness, or the head CT is abnormal, it is recommended to treat with IV phenytoin unless there is a known allergy.

- Phenytoin 18 mg/kg IV no faster than 50 mg/minute



Surgery

Indications for operative intervention

General indications for surgical intervention after trauma

- Extra-axial mass lesion > 1 cm thick
- Midline shift > 5 mm
- ICH > 3 cm
- Midline shift < 5mm but ICP > 20 mmHg
- Penetrating injury
- Compound depressed skull fracture
- Intracranial hypertension refractory to medical management

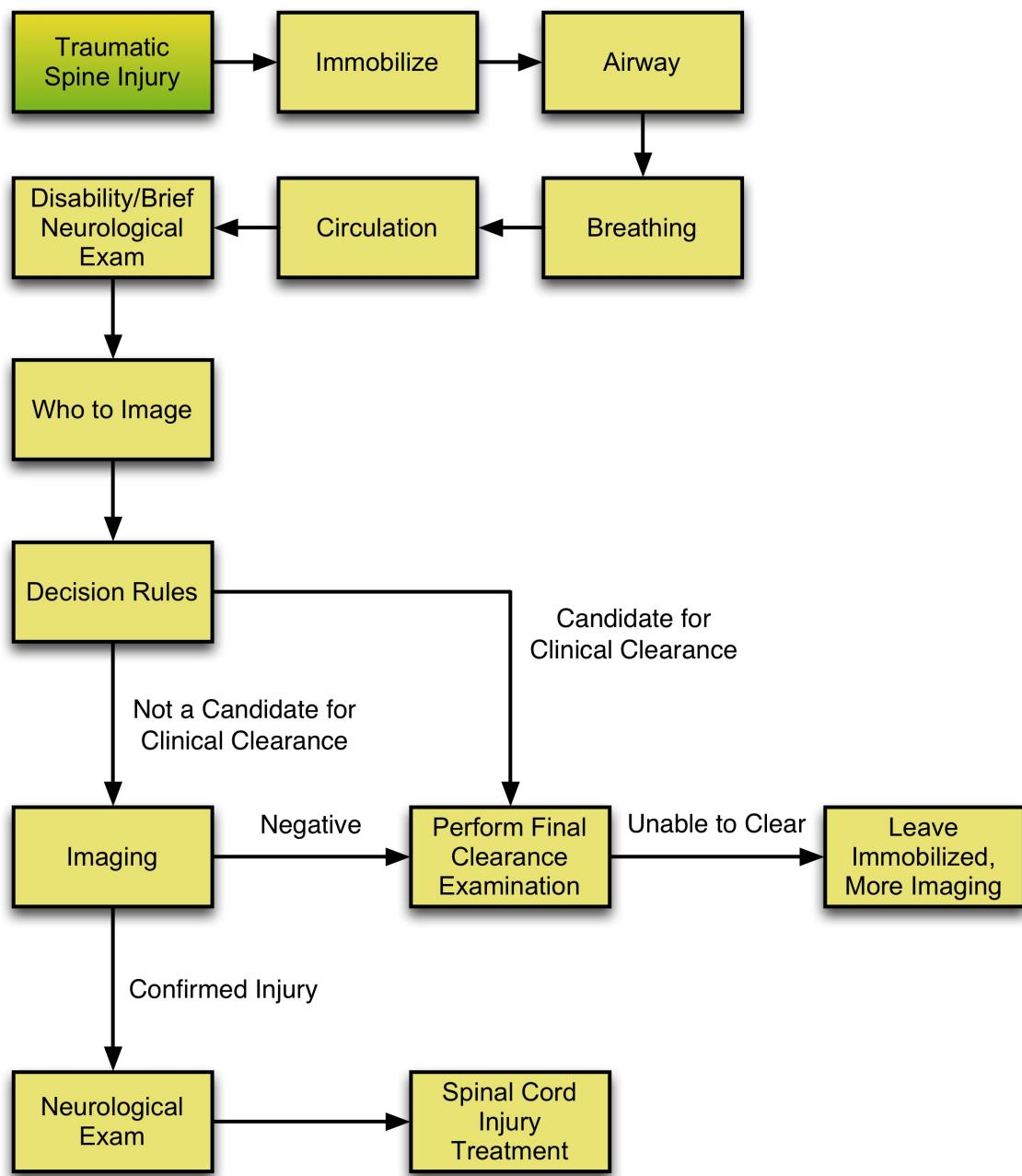
Emergency Neurological Life Support

All Protocols

Traumatic Spine Injury Protocol

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Last Updated: 5/23/2013



Checklist & Communication

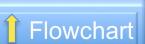


Checklist

- Immobilize C-Spine until cleared
- Keep SBP > 90 mmHg
- C-Spine precautions (definitively declare if the spine is cleared or not when handing off the patient)
- Head CT
- Treat herniation

Communication

- Age
- Mechanism of injury
- GCS
- Coagulation status
- Other injuries
- Last neuro exam
- Is the spine cleared? If so, by which criteria. If not, why?
- CT scan results



Airway

Who to Intubate

Patients with TSI can be at exceptionally high risk of loss of airway due to a combination of:

- Airway edema
- Loss of diaphragmatic innervation (C3, C4, and C5 innervate the diaphragm)
- Loss of chest and abdominal wall strength

All patients with a complete cervical TSI above C5 should be intubated as soon as possible.

Patients with incomplete or lower injuries will have a high degree of variability in their ability to maintain adequate oxygenation and ventilation. General parameters for urgent intubation:

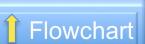
- Complaint of "shortness of breath", inability to "catch my breath", or breathlessness
- Vital Capacity < 10 mL/kg or decreasing vital capacity
- Appearance of "belly breathing" or "quad breathing" (abdomen goes out sharply with inspiration) When in doubt, it is better to intubate a patient with a cervical TSI electively rather than wait until it needs to be done emergently. Patients will typically develop worsening of their primary injury shortly after admission due to cord edema and progressive loss of muscle strength. Patients with very high (above C3) complete TSI will almost invariably suffer a respiratory arrest in the field and, if not intubated by pre hospital providers, typically present in cardiac arrest.

How to Intubate

Generally, patients with cervical TSI who require intubation should be intubated using an awake fiber optic approach by an experienced provider. Patients who require urgent or emergent intubation, should be intubated using rapid sequence intubation (see ENLS protocol [Airway, Ventilation and Sedation](#)).

Special issues related to intubation in TSI:

- Aspiration precaution should always be taken as for any emergent intubation.
- The cervical collar can be removed ONLY IF in-line stabilization is carefully maintained.
- Care MUST be taken not to hyperextend the neck.
- No particular RSI regimen is preferred, but these patients will already have loss of vasomotor tone and therefore medications that diminish the catecholamine surge may result in hypotension and bradycardia.



Breathing

Patients with TSI are at high risk of inadequate oxygenation and ventilation. This is due to a combination of factors:

- Loss of diaphragmatic function
- Loss of ability to cough and deep breathe due to loss of chest wall and abdominal musculature function
- Aspiration
- Retention of secretions
- Atelectasis
- Concomitant injuries (pulmonary contusions, pneumothorax) Supplemental oxygen should be supplied to all patients with cervical TSI. Hypoxia is extremely detrimental to patients with neurological injury. Noninvasive methods of ventilation should be used with caution as the inability to cough and clear secretions may lead to an increased risk of aspiration.



Brief Neurological Examination

As part of initial trauma survey

In trauma patients during the primary survey, this can be abbreviated to the patient's Glasgow Coma Scale (GCS), pupil size and reactivity, and ability to move all four extremities.



Circulation

Patients with TSI (above T4) often develop neurogenic shock. The patient suffers a "sympathectomy" resulting in unopposed vagal tone. This leads to a distributive shock with hypotension and bradycardia.

- Patients are generally hypotensive with warm, dry skin. This is due to the loss of sympathetic tone resulting in an inability to redirect blood flow from the periphery to the core circulation.
- Bradycardia is a characteristic finding of neurogenic shock and can help to differentiate from other forms of shock.

Care should be taken to "assume" that a patient has neurogenic shock because of a lack of tachycardia as young healthy people and patients on premorbid beta-blockers, etc. will often not manifest tachycardia in the setting of hemorrhage.

- As a general rule, the higher and more complete the injury, the more severe and refractory the neurogenic shock.
- These signs can be expected to last from one to three weeks.
- Patients may develop manifestations of neurogenic shock hours to days following injury due to progressive edema and ischemia of the spinal cord resulting in "ascension" of their injury.
- In the patient with traumatic injury, other sources of hypotension (hemorrhage, TBI) MUST be sought and ruled out. Pitfall: "Spinal shock" has nothing to do with hemodynamics, but rather refers to the loss of deep tendon stretch reflexes because of the spinal injury.

Management of Hypotension: maintain MAP 85-90 mm Hg for the first 7 days

First line treatment of neurogenic shock is always fluid resuscitation to maintain euvoolemia.

- The loss of sympathetic tone leads to vasodilation and the need for an increase in the circulating blood volume ("filling the tank")
- CVP monitoring may be helpful in establishing fluid resuscitation goals. Second line therapy once euvoolemia is established is pressors and/or inotropes.
- Norepinephrine - has both alpha and some beta activity thereby improving both blood pressure and bradycardia. Probably the preferred agent.
- Phenylephrine - pure alpha agonist. Is very commonly used and easily titratable. Lacks beta activity so does not treat bradycardia and may actually worsen it through reflexive mechanisms.
- Dopamine - need high doses (> 10 microgram/kg/min) for alpha effect, but does have significant beta effects at lower doses. May lead to inadvertent diuresis at lower doses exacerbating relative hypovolemia.

- Epinephrine - An alpha and beta agonist. Causes vasoconstriction and increased cardiac output. High doses often required leading to inadvertent mucosal ischemia. Rarely used or needed.
- Dobutamine - Beta agonist that can be useful if the loss of sympathetic tone is causing cardiac dysfunction. Caution should be used in patient who is not adequately volume loaded as may cause hypotension.

Some institutions utilize a protocol based on the American Association of Neurological Surgeons and the Congress of Neurological Surgeons' Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries which recommend as an option "Maintenance of mean arterial blood pressure at 85 - 90 mm Hg for the first seven days following acute TSI to improve spinal cord perfusion . . ."



Clinical clearance

Can the spine be cleared clinically?

The patient has had either a negative CT scan, or did not meet NEXUS or Canadian Rules criteria that recommend CT imaging. Depending on the patient's level of consciousness, one may be able to clear the patient clinically.

- Repeat the neurological examination and if normal and the patient is alert and without pain, clear the cervical spine. This is done by removing the neck collar and have the patient rotate their head 45 degrees to each side. If they are able to do this without significant pain, the C-Spine can be cleared. If there is significant pain or the patient cannot perform the entire movement, replace the neck collar
- If the patient has altered mental status that is expected to be transient (e.g. alcohol or drug intoxication), maintain cervical spine immobilization until reliable examination is possible (NEXUS or Canadian C-Spine Rules) and proceed through this algorithm from the beginning.

Unable to Clear Spine Clinically: May be ligamentous injury

If the action of self-imposed neck rotation 45 degrees to either side proves too painful to complete, ligamentous injury is a possibility; therefore, the C-collar should be left in place and imaging pursued. See section on Imaging. If the patient has already had a CT of the spine revealing no fracture, MRI may be of value to investigate ligamentous injury.



Decision Rules

Canadian and NEXUS Rules

The decision to perform a CT of the spine to image potential bony injury is as follows:

Significant associated injuries:

- Multiple trauma patient needs CT of head, chest, or abdomen/pelvis
- Intubated
- Depressed level of consciousness
- Neurological deficit referable to the spine, or complaints of bilateral paresthesias
- Strong clinical suspicion of any spinal fracture
- Multiple fractures
- Pelvis fracture
- Significant head or facial trauma

Significant mechanism of injury:

- Motor vehicle collision with speed exceeding 35 mph
- Ejection from vehicle
- Pedestrian, bicyclist, or motorcyclist struck and thrown
- Axial load injury (vehicle roll-over or diving injury)
- Fall in excess of 10 ft.
- Death at accident scene

Patient Factors

- Age > 65 yr.
- DJD, ankylosing spondylitis, rheumatoid arthritis
- Depressed level of consciousness
- Known cervical spine injury

If the mechanism is worrisome (clear history of neck injury or circumstances that have a reasonable likelihood of causing spinal trauma) one can consider using two validated clinical scales. These are the NEXUS Rules and the Canadian C-spine Rules. Each of these systems allows you to either move toward clinical clearance of the C-spine or escalate evaluation to spine imaging.

Canadian C-Spine Rules

These rules help one decide if you need spinal imaging. First consider any high-risk features; if none, examine any low risk features. If after considering all of the features, and none apply, the patient can be cleared clinically and the cervical spine immobilization can be discontinued.

Canadian High-risk Features

Are there any high-risk factor that mandates radiography?

- Age > 65 yrs. or dangerous mechanism (fall from elevation over 3 ft. or 5 stairs)?
- An axial load to the head (e.g. diving)?
- A motor vehicle collision exceeding 100 km/hr. or with roll-over or ejection, or a collision involving a motorized recreational vehicle, or a bicycle collision?

IF YES to ANY of the above, consider CT criteria for imaging next.

If NO to ALL of the above, move on to Canadian Low Risk features below.

Canadian Low-risk Features

Do any of the following low risk features exist?

- Simple rear-end motor vehicle collision
- Sitting position in the emergency department
- Ambulatory at any time
- Delayed (not immediate) onset of neck pain
- Absence of midline cervical-spine tenderness

If YES to ANY of the above, then proceed to testing of neck rotation.

If NO to ALL of the above, then consider CT criteria for clearance.

NEXUS Rules:

5 questions

NEXUS Rules: These "rules" apply 5 criteria that used alone can help you clinically clear the cervical spine. These include the presence of spinal tenderness and presence of focal neurological deficit among other things. Use of the NEXUS rules is a reasonable protocol to clear the cervical spine; we encourage you to look at the Canadian Rules as well.

The NEXUS rules are:

- No posterior midline cervical-spine tenderness.
- No evidence of intoxication.
- A normal level of alertness.
- No focal neurological deficit.
- No painful distracting injuries.

If all of the above are true then you can clinically clear the cervical spine and remove the immobilization device. If any one or more is true, move on to the next step regarding spine imaging.

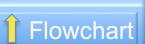


Image the C-Spine with CT

CT is most sensitive for bony injury

The patient meets criteria for CT imaging of the spine. Maintain C-spine immobilization throughout the imaging and transportation.

CT Imaging Positive: Reveals a fracture or suspicious Injury

The CT reveals a finding that is definitive (vertebral fracture) or suspicious (soft tissue swelling).

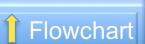
- Maintain C-spine immobilization
- Consult Neurosurgery

Obtain MRI as indicated

- If neurological examination is compatible with spinal cord injury (myelopathy) or the patient complains of bilateral paresthesias
- If the patient is alert with continued midline cervical spine tenderness or if the patient is expected to require prolonged cervical spinal immobilization (e.g. severe closed head injury), consider MRI for the possibility of anterior -posterior spinal ligamentous injury
- Perform MRI of the known or suspected areas of spinal cord injury

CT Shows No Fracture: No fracture or soft tissue swelling

The CT shows intact vertebrae and no evidence for soft tissue swelling around the spine. At this point it is okay to move toward clinical clearance of the cervical spine if possible.

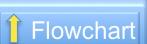


Initial Treatment of TSI

The question of steroids

The mainstay of treatment for SCIs is

- decompression of the spinal cord to minimize additional injury from cord compression
- surgical stabilization of unstable ligamentous and bony injury
- minimizing the effect of secondary complications, such as venous thromboembolic disease, pressure ulcer prevention, respiratory failure, and infections.
- Place Foley catheter
- GI prophylaxis
- Steroids: The use of steroids following acute traumatic cervical spinal injury is highly controversial and is both institution and practitioner specific. If steroids are to be used, the recommended regimen is methylprednisolone 30 mg/kg as a bolus, then 5.4 mg/kg/hr for 23 or 47 hours.



Maintain Spine Immobilization

Focus on stabilization until injury is confirmed absent

Appropriate care must be taken to provide spinal immobilization on scene. The spinal column should be immobilized until an unstable injury can be excluded. In the prehospital setting, patients are typically fitted with a cervical collar to provide cervical spinal column immobilization, and patients are subsequently transferred to the hospital on a backboard. If the patient is intoxicated and uncooperative with medical evaluation, chemical sedation may be indicated to assure proper protection of the spinal column and, more importantly, the spinal cord.

As a general rule, the diagnosis and treatment of the majority of spine injuries can be deferred to address other life-threatening injuries, such as hemorrhage or intracranial mass lesions, as long as spine immobilization is maintained.



Neurological Examination

Focus on signs related to spinal cord injury

The neurological examination should focus on motor, sensory and rectal tone findings. If the patient has abnormality in any of these, the goal is to localize the lesion to the highest spinal level where you see dysfunction.

Neurological Signs Present? Clinical finding supporting spine injury:

Based on a neurological examination, there are findings consistent with a spinal cord injury. These include:

- weakness below the level of the spine injury
- sensory loss below the level of spine injury
- loss of anal tone
- hyperreflexia or areflexia

If present, one should image the spine with CT and maintain spine immobilization.

Detailed Examination

Here are a few motor and sensory "levels" as a guide (these refer to the myotome and dermatome respectively for these regions of dysfunction):

Ten key muscles that should be tested and documented (grade each as grade 0-5*):

- C5- biceps
- C6 - Extensor carpi radialis longus
- C7 - Triceps
- C8 - Flexor digitorum profundus (#3)
- T1 - Adductor pollicis
- L2 - Iliopsoas
- L3 - Quadriceps
- L4 - Tibialis anterior
- L5 - Extensor hallucis longus
- S1 - Gastrocnemius, soleus
- Sacral: voluntary anal contraction (present/absent)

*Motor Strength Grading:

- 0 = no active movement
- 1 = muscle contraction
- 2 = movement thru ROM w/o gravity
- 3 = movement thru ROM against gravity

- 4 = movement against some resistance
- 5 = movement against full resistance

Detailed examinations recommended by the American Spinal Injury Association can be found on their [website](#).

Sensory:

- C4 - deltoid
- T4 - nipple
- T10 - umbilicus

Decreased rectal tone:

- may be the only sign of a spinal cord injury.

Sensory Examination: Is there a sensory level?

With light touch and/or pin, touch each dermatome beginning with C1 (posterior scalp) and move caudally to see if the patient has normal, diminished or absent sensory function at a particular level and below. Light touch and pain may be separated by a dermatome or 2; select the highest (cephalad) level as the sensory level. Test sacral sensory function with a pin; score it as normal, diminished or absent. Score deep anal sensation as present or absent.

Specific Syndromes: Depending on the level and nature of injury

There are several spinal cord injury syndromes that if present help indicate the extent and nature of the injury.

- Anterior Cord Syndrome - Loss of pain/temperature and motor but not light touch; due to contusion of the anterior cord or occlusion of the anterior spinal artery. It is associated with burst fractures of the spinal column with fragment retropulsion by the axial compression.
- Central Cord Syndrome - loss of cervical motor function with relative sparing of lower extremity strength. This is typically due to hyperextension injury in elderly patients with cervical stenosis. It is often not associated with a fracture; rather, buckling of the ligamentum flavum contuses the cord causing bleeding with the center of the cord. The amount of damage to the corticospinal tracts (which lie laterally) is variable and determines the amount of lower extremity weakness.
- Brown-Sequard Syndrome - hemiplegia, loss of ipsilateral light touch, and loss of contralateral pain/temperature sensation due to hemisection of the cord. Indicates a penetrating cord injury often from missile or knife, or a lateral mass fracture of the spine.

ASIA Impairment Scale: Important for prognosis

The American Spinal Injury Association (ASIA) defined a 5-element scale that is prognostic of neurological recovery:

- A - Complete: No motor or sensory function in the lowest sacral segment.
- B - Incomplete: Sensory but not motor function is preserved in the lowest sacral segment.
- C - Incomplete: Less than one-half of the key muscles below the neurological spinal level have grade 3 or better strength.
- D - Incomplete: at least one-half of the key muscles below the neurological level have grade 3 or better strength.
- E - Sensory and motor function are normal.

Complete injury (no sensory or motor function below a spinal level) has a worse prognosis; however, spinal shock can present this way. Incomplete injury is better prognostically.



Traumatic Spine Injury

Cervical Spine Injury

Traumatic Spine Injury (TSI): This topic covers TSI as it relates to the cervical spine; many of the concepts apply to less common thoracic or lumbar spine trauma. One should suspect cervical spine injury when there is

- A worrisome mechanism
- Midline cervical spine tenderness
- Neurological findings consistent with acute spinal cord injury

Initial management should include:

- Airway
- Breathing
- Circulation
- Immobilization
- Detailed examination
- Imaging, if necessary
- Treatment

Notes:

- You may put the patient in reverse Trendelenburg if at risk for aspiration.
- Back board may, and should, be removed and used for transport only because of the risk of skin breakdown

Topic Co-Chairs: John Marx, MD Deborah Stein, MD



Who should be imaged

How to "clear the cervical spine"

Point tenderness over a spinous process may indicate instability of the respective vertebral bone. Examine the entire spine by palpation or percussion; focal spine injury often produces highly focal tenderness.

- If focal tenderness is present, the patient may need a CT of the spine

There are two recommended systems that help you determine who you can "clinically clear" from significant spine injury without imaging, and who you should perform CT/MRI to detect fractures or spine misalignment. These are the NEXUS and Canadian Rules.