This pocket-sized guide distills complicated neurological conditions to deliver the essentials of best care for the neurocritical patient. Often missing from acute care courses, neurocritical care is a growing field, with more patients than ever admitted to the ICU for neurocritical conditions. This specialty requires specificity and precision, but as this practical resource demonstrates, the intricacies of neurocritical care should not be an insurmountable obstacle for any APP.

Written in an easy-access style, Fast Facts About Neurocritical Care covers the defining characteristics, clinical presentation, diagnostics, treatment, and nursing considerations of common neurological disorders seen in acute care settings. Chapters review the assessment and diagnosis of common and not-so-common neurological conditions that can often be difficult to recognize and manage. With learning objectives, illustrations, and Fast Facts boxes highlighting critical content, this reference is an invaluable resource for orientation into this often-challenging specialty.

- Useful pocket resource for difficult-to-master neurological conditions presenting in ICU
- Addresses a growing area of healthcare—a rapidly expanding specialty requiring well-versed nurses, nurse practitioners, and physician assistants
- Reviews the basic neurological exam, as well as exam of the comatose patient
- Explains pertinent diagnostics including CSF interpretation and different imaging modalities
- Discusses commonly used treatments and medications
- Presents an orientation resource to this challenging specialty

“This practical and common-sense approach is an excellent companion to the care you provide to your patient.”

—Grace H. Bryan
President, Association of Neurosurgical Physician Assistants
[From the Foreword]
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Diane McLaughlin, DNP, AGACNP-BC, CCRN, is a critical care nurse practitioner who works in the departments of neurosurgery and neurocritical care at MetroHealth Medical Center in Cleveland, Ohio, and in critical care at Mayo Clinic in Jacksonville, Florida. Dr. McLaughlin has worked in critical care for 15 years, first as a nurse and then as a nurse practitioner. She received her master of science in nursing from the University of Florida in 2013 and her doctorate of nursing practice from the University of Florida in 2017. Her research interests include neurosurveillance, sleep in critical care, and advanced practice provider training and education.

Dr. McLaughlin is active within the Society of Critical Care Medicine, serving 3-year appointments to both the Adult Ultrasound Committee and the Advanced Practice Provider Resource Committee. She has also served as faculty for the SCCM Ultrasound Fundamentals Course. Dr. McLaughlin is also active within the Neurocritical Care Society, having served as a reviewer and currently serving on a guideline writing committee. Dr. McLaughlin is also a member of the American Association of Critical Care Nurses and American Association of Nurse Practitioners. She has spoken at multiple local, national, and international conferences on topics in neurocritical care and has published regarding topics in critical care, neurocritical care, and advanced practice provider use in critical care.
This book is dedicated to Dr. William David Freeman, who woke up at 4 a.m. on Saturday mornings just to teach me. His mentorship and encouragement continue to inspire me to explore the unknown, teach the known, and always strive to reach higher.
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Foreword

If you are an advanced practice provider (APP), you should obtain this book. If you are working in neurology, neurosurgery, or critical care, you need this book. As a practicing physician assistant for over 22 years, I have seen a dramatic change in the acceptance of APPs as integral partners in healthcare. The demand on our healthcare system has put an ever-increasing need for our patients and loved ones to rely on an advocate and mediator to care for them. There are very few resources that are specific to neurology critical care and neurosurgery APPs. This book, authored by Diane McLaughlin, meets those expectations.

Starting with the basic neurology exam and then thoroughly walking you through the different types of strokes, trauma, infectious diseases, seizures, and brain death criteria, this practical and commonsense approach is an excellent companion to the care you provide to your patient.

I have had the good fortune of working directly with Dr. McLaughlin at Mayo Clinic since 2013, sharing patients and exchanging ideas. Her vast experience in critical care and expertise in clinical trials and studies places her at the top of her field in patient care and research. I am honored to work with her and care for the critical needs of our patients and their families.

Grace H. Bryan, PA-C
Mayo Clinic Jacksonville Neurosurgery
President, Association of Neurosurgical Physician Assistants

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Welcome to Fast Facts About Neurocritical Care: A Quick Reference for the Advanced Practice Provider. This book is a very nonexclusive resource for anyone who works in neurocritical care, including physician assistants, nurse practitioners, clinical nurse specialists, and bedside nurses. I would not even be surprised to find it in the hands of a medical student, intern, or resident.

If you are reading this book, then you probably already take care of neurology patients. This also means that you already realize that neurology is a challenging specialty. Lack of knowledge regarding how to perform an adequate neurological examination, how to diagnose specific conditions, and, perhaps most importantly, how to treat them, can be dangerous for both the patient and provider.

This book will not tell a story. This book will not provide in-depth anatomy, pathophysiology, or pharmacology. Instead, this book will give you exactly what the title portrays—a quick reference book to give you “fast facts” about commonly seen neurological conditions in the adult critical care setting. You will also receive some pearls of wisdom, some useful tables, and even some scoring guides to help you assess your patients and classify their pathology. This book is best suited for a work bag or office desk to reference when you forget whether seizure prophylaxis is indicated, cannot find your stroke scale booklet, or are unsure which tests you should order during a meningitis workup. I hope it serves you well and that you use it often.

Diane McLaughlin
Intracranial Hypertension

It is essential to understand the concept of intracranial hypertension to realize the consequences of many of the other disorders detailed within this book. Intracranial pressure (ICP) can rise transiently without becoming pathologic (e.g., a sneeze). In sustained increased ICP, brain herniation can occur, which can lead to severe morbidity and death. This chapter will explain the concept of ICP, herniation, and the ways to manage acute rise in ICP.

In this chapter, you will learn how to:

- Explain the Monro–Kellie doctrine and its application to ICP.
- Describe brain herniation syndromes.
- Detail measurement and treatment strategies to decrease ICP.

PATHOPHYSIOLOGY

The Monro–Kellie doctrine describes the skull as being a fixed box containing brain tissue, cerebrospinal fluid (CSF), and blood. If there is an increase in any one of these components, there must be a compensatory change in another in order to prevent an increase in pressure. These components are very limited in the amount that they can compensate; therefore, any space-occupying lesion (hemorrhage, tumor), edema, or increase in CSF can result in increased...
pressure, which may subsequently shift structures into an opening or into an adjacent space that they do not typically occupy (herniation).

**INTRACRANIAL HYPERTENSION**

- Normal ICP
  - 5 to 15 mmHg, but anything less than 20 mmHg is nonpathologic.
- ICP greater than 20 mmHg
  - Reduces cerebral perfusion pressure (CPP)
  - Can cause ischemic brain injury
- CPP
  - CPP is the difference between mean arterial pressure (MAP) and ICP
  - Autoregulation refers to the brain's ability to maintain constant cerebral blood flow (CBF) over a range of CPP (60–150 mmHg)
    - May be impaired or absent in the injured brain
  - CPP less than 60 mmHg decreases CBF and can lead to ischemic brain injury
  - CPP greater than 150 mmHg can lead to cerebral edema
  - CPP may be more important than ICP measurement in outcomes

**Causes**

- Space-occupying lesion
  - Intracranial blood
    - Epidural
    - Subdural
    - Subarachnoid
    - Intraparenchymal
  - Brain tumor
  - Brain abscess
- Cerebral edema (Table 4.1)
  - Cytotoxic
    - Intracellular edema
    - Intact blood–brain barrier
  - Vasogenic
    - Extracellular edema
    - Loss of intact blood–brain barrier
    - Associated with brain tumor or inflammation disorder
- CSF accumulation
Approximately 20 mL of CSF is produced every hour.

- **Hydrocephalus**
  - Accumulation of CSF causing widening of the ventricles
  - **Types of hydrocephalus**
    - **Communicating**
      - CSF is blocked after exiting ventricles but can still flow between ventricles
      - Causes
        - Congenital
    - **Noncommunicating/obstructive**
      - CSF is blocked along the path connecting the ventricles
      - Causes
        - Subarachnoid hemorrhage
        - Aqueductal stenosis
    - **Hydrocephalus ex vacuo**
      - Compensatory enlargement of the ventricles
      - Brain tissue shrinks following stroke or traumatic brain injury
    - **Normal pressure hydrocephalus**
      - Hydrocephalus not associated with increased ICP

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**NS**, normal saline.
Hygroma
- CSF collection without blood
- Most commonly subdural
- Often seen in elderly after trauma or subdural hematoma or in children after infection

Pneumocephalus
- Air trapped in the intracranial vault
- Can cause displacement and compression of brain tissue
- Causes
  - Following neurosurgery
  - Following ear, nose, and throat (ENT) surgery
  - Rarely: spontaneously
- Symptoms
  - Headache
  - Symptoms of increased ICP
  - Altered level of consciousness
  - Focal findings based upon mass effect
- CT findings (Figure 4.1)
  - Anechoic area with adjacent compression or flattening of sulci
  - Mount Fuji sign in tension pneumocephalus
- Treatment
  - Conservative: allow to reabsorb over time

Figure 4.1 Pneumocephalus. (The dark area in the left upper portion of the intracranial vault depicts pneumocephalus.)
Oxygen therapy to wash out nitrogen
  - Most protocols provide 100% supplemental oxygen
    - Alternating or continuous
    - 8 to 24 hours
    - Ventilator, nonrebreather, or high-flow nasal cannula
  - Surgical decompression in extreme cases

- Valsalva maneuver
- Coughing
- Sneezing

**BRAIN HERNIATION**

- Supratentorial
  - Uncal transtentorial herniation
    - Uncinate process of the temporal lobe herniates into the anterior opening of the tentorium cerebelli
    - Compresses the posterior cerebral artery as it crosses the tentorium and causes posterior cerebral artery (PCA) territory infarct
  - Central tentorial herniation
    - Thalamic region herniates downward through the opening of the tentorium cerebelli
  - Subfalcine herniation
    - Cingulate gyrus displaces across the midline and beneath the falx
  - Transcalvarial herniation
    - External herniation
    - Brain displaces through skull defect (fracture or postcraniectomy)

- Infratentorial
  - Upward transtentorial herniation
    - Reverse coning
    - Posterior fossa mass displaces upward
    - Most commonly occurs when external ventricular drain (EVD) is placed supratentorial for ICP monitoring or hydrocephalus and posterior fossa contents are pulled upward
  - Tonsillar or foraminal herniation
    - Cerebellar tonsils displace downward through the foramen magnum
Signs and Symptoms

- Physical exam findings
  - Headache
    - Continuous
    - May be worse in the morning (after lying flat all night)
  - Emesis
    - Often not preceded by nausea
    - Projectile
  - Altered mental status
    - Confusion
    - Loss of consciousness
    - Coma
  - Seizures
  - Vision changes
  - Pupil changes

- Vital signs abnormalities
  - Cushing’s triad: widened pulse pressure, bradycardia, irregular respirations
  - Hypertension
  - Bradycardia
  - Respiratory pattern changes
  - Shallow breathing

- CT features of increased ICP
  - Effacement of basal cisterns
  - Loss of sulci
  - Loss of gray–white differentiation
  - Midline shift
  - Herniation syndromes

DIAGNOSIS OF INCREASED ICP

- ICP monitors
  - EVD
    - EVD is the gold standard ICP monitor because it is the only device that can both monitor and treat elevations of ICP
    - Typically leveled at tragus
      - Many institutions will also level arterial lines at the tragus to calculate CPP when an EVD is in place
    - ICP waveform can indicate cerebral compliance (Figure 4.2)
      - P1—percussion wave—arterial pulsation
Normal ICP waveform

Abnormal noncompliant ICP waveform

**Figure 4.2** Demonstration of a normal ICP waveform and an ICP waveform with P2 prominence, suggestive of poor intracranial compliance.

ICP, intracranial pressure.

Illustration: Nicholas McLaughlin.

- **P2**—tidal wave—cerebral compliance
- **P3**—dicrotic wave—venous pulsation

- Normal ICP is less than 20 mmHg
- ICP may increase during times of high pressure, such as sneezing or coughing, but is not a concern unless it does not return to less than 20 mmHg
- ICP sustaining greater than 20 mmHg for 5 or more minutes should be addressed

**Fast Facts**

A normal ICP waveform follows a step-like progression, in which the amplitude of P1 is greater than P2, which is greater than P3 (Figure 4.2).

- **Fiberoptic catheter**
  - Sensor located within the catheter tip
  - May be placed intraparenchymally or in the ventricle
- **Bolt**
  - Typically placed between the arachnoid membrane and cerebral cortex
- **Transcranial Doppler**
  - Measures CBF
  - Can evaluate cerebral compliance
  - Noninvasive
  - Posterior circulation can be obtained; however it is more difficult and not commonly done
Optic nerve sheath diameter (Figure 4.3)

Ultrasound
- Dilation of the optic nerve sheath beyond 0.5 cm has been associated with an ICP greater than 20 mmHg
- Quick, noninvasive
- Unlikely to correlate with changes in pressure in the infratentorium

**NURSING INTERVENTIONS**

- Positioning: Assure optimal venous return from the brain
  - Head of bed at 30 degrees
  - Midline neutral positioning
  - Avoid any venous compression
- Observe for signs of sleep apnea
  - If CO$_2$ increases during apneic periods, it is often accompanied by increased ICP
MEDICAL INTERVENTIONS

- Optimize oxygen delivery
  - PaO₂
  - Treat anemia
- Maintain CPP greater than 60 mmHg
  - Fluids
  - Vasopressors

Fast Facts

*Do not administer dextrose solutions.* They decrease plasma osmolality and increase water content within brain tissue.

- Cerebral vasoconstriction avoidance
  - Goal PaCO₂ of 35 to 45 mmHg

Fast Facts

CO₂ dilates cerebral vasculature, which increases intracranial blood volume and increases ICP. Acutely, hyperventilation can be used to help bring down ICP; however long-term use of hyperventilation should be avoided, as the vasoconstriction that occurs could reduce CBF to the point of ischemia.

- Decrease cerebral metabolic rate
  - Pain control
  - Sedation
  - Neuromuscular blockade
  - Treat seizures
  - Hyperthermia avoidance
- Medication—osmotherapy
  - Mannitol (0.25–1 g/kg)
    - Target: serum osmolality of 300 to 320 mOsm/kg
    - Stop administration for any serum osmolality greater than 330 mOsm/kg
    - Can be given peripherally
    - Standard dose is 100 g of 20% mannitol
Hypertonic saline (23.4%)
- Often given in volumes of 15 to 30 mL at a time
- Target: serum Na of 145 to 155 mEq
- Must be given via a central line
- Also called “hot salts”

Hypertonic saline (3%)
- Can be given carefully through a peripheral line at a low rate
  - Monitor for phlebitis
  - Cannot run with any other medications
  - Do not exceed rate of 30 mL/hr
  - If extending infusions or giving at an increased rate, place central venous access
- Used to slowly drive sodium to goal range

Consider repeat imaging
- Worsening edema
- Increasing hematoma, lesion size
- Accumulating hydrocephalus

NEUROSURGICAL INTERVENTIONS

- Reduction of intracranial volume
  - EVD placement
  - Hematoma evacuation
  - Tumor debulking
- Opening the cranial vault to allow expansion
  - Decompressive craniectomy
    - Controversial, as it may not reduce morbidity or avoid death

Bibliography

Meningitis is defined as inflammation of the meninges. There are many reasons that this may occur, and although the initial presentation may be similar, the treatment is very different dependent upon the cause. This chapter will help the reader identify meningitis as a differential diagnosis and discuss workup, cerebrospinal fluid (CSF) interpretation, and treatment.

In this chapter, you will learn how to:

- Recognize signs and symptoms of possible meningitis.
- Interpret CSF to confirm diagnosis.
- Discuss treatment strategies for different types of meningitis.

**PRESENTATION**

- Most common symptoms
  - Altered mental status
  - Fever
  - Meningismus (neck stiffness)
  - Headache
In one study, less than half of patients had the once-called “classic” triad of altered mental status, neck stiffness, and fever, but almost all patients had at least two symptoms when headache was added.

- Additional common findings
  - Seizures
  - Vomiting
  - Cranial nerve palsies
  - Kernig’s sign
    - Unable to straighten leg greater than 135 degrees without pain
  - Brudzinski’s sign
    - Severe neck stiffness causes hips and knees to flex when neck is flexed
  - Jolt accentuation test
    - Can only be done with cooperative patients
    - Patient quickly moves head side to side in a horizontal plane; if headache worsens, then this is considered a positive finding
    - Positive jolt in combination with fever may be a more sensitive test than Kernig’s or Brudzinski’s sign.
  - Papilledema

The sensitivity and specificity of Kernig’s and Brudzinski’s signs have not been adequately studied. Absence of these signs does not rule out meningitis.

- Atypical presentations
  - Elderly
    - Obtundation without fever or meningismus
  - Immunocompromised host
    - Altered mental status
    - No fever due to inability to mount inflammatory response
DIAGNOSIS

- Neuroimaging
  - Noncontrast CT
    - Do not delay antibiotic administration to obtain CT or lumbar puncture (LP)
    - Excludes alternative diagnosis, subarachnoid hemorrhage (SAH)
    - Evaluates mass effect
    - Obtain CT prior to LP in the following patients
      - Immunocompromised patients
      - History of central nervous system (CNS) disease
      - Altered mental status
      - Focal neurological deficits
      - Papilledema
      - New-onset seizure(s)
    - In some cases LP may be pursued prior to CT scan or without CT scan

Fast Facts

A high suspicion for meningitis should be maintained in patients with fever, headache, altered mental status, and a normal CT.

- LP
  - A diagnosis of meningitis cannot be made without an LP
  - Always obtain an opening pressure
  - Results will help to guide therapy
  - CSF testing (Table 14.1)
    - Ideally obtain CSF in all four tubes
    - The following tests should be ordered for all LPs
      - Red blood cell (RBC) count
      - White blood cell (WBC) count
      - Protein
      - Glucose
      - Gram stain
      - Culture
    - Consider these additional tests
      - Lactic acid (CSF)
      - India ink if suspicion for fungal infection
      - Herpes polymerase chain reaction (PCR)
      - Antigens
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<td>Appearance</td>
<td>Clear</td>
<td>Cloudy, turbid</td>
<td>Clear</td>
<td>Clear or cloudy</td>
<td>Opaque; forms fibrin web if left to settle</td>
<td>Sanguineous initially, xanthochromia &gt;12 hr later</td>
<td>Clear or xanthochromia</td>
<td>Clear</td>
</tr>
<tr>
<td>Opening pressure</td>
<td>&lt;20 cmH₂O</td>
<td>Elevated; &gt;25 cmH₂O</td>
<td>Normal or elevated</td>
<td>Elevated</td>
<td>Elevated</td>
<td>Elevated</td>
<td>Normal or elevated</td>
<td>Normal</td>
</tr>
<tr>
<td>WBC count</td>
<td>&lt;5 cells/μL</td>
<td>Elevated; &gt;100 cells/μL; primarily PMNs (&gt;90%)</td>
<td>Elevated; 50–1,000 cells/μL; primarily lymphocytes, can be PMNs early on</td>
<td>Elevated; 10–500 cells/μL</td>
<td>Elevated; 10–1,000 cells/μL; early PMNs then mononuclear cells</td>
<td>Elevated; WBC to RBC ratio of 1:1,000</td>
<td>Normal</td>
<td>0–20 cells/μL; primarily lymphocytes</td>
</tr>
<tr>
<td>Glucose</td>
<td>CSF glucose/serum glucose ratio</td>
<td>Serum glucose</td>
<td></td>
<td></td>
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<tr>
<td>-------------------------</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low; &lt;40%</td>
<td>Normal; &gt;60% of serum glucose</td>
<td>Low</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low; &lt;40%</td>
<td>Serum glucose; may be low in HSV</td>
<td>Low</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal; &gt;60% of serum glucose; may be low in HSV</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal; &gt;60% of serum glucose; may be low in HSV</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal; &gt;60% of serum glucose; may be low in HSV</td>
<td>Normal</td>
<td>Normal</td>
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</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Protein</th>
<th>&lt;50 mg/dL</th>
<th>Elevated; &gt;50 mg/dL</th>
<th>Elevated; 1–5 g/L</th>
<th>Elevated; &gt;5.5 g/L</th>
<th>Mildly elevated; 0.45–0.75 g/L</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Causes</th>
<th>Normal Adults: Neisseria meningitides, Streptococcus pneumoniae, Listeria monocytogenes</th>
<th>HSV (HSV 2 more common than HSV 1); enterovirus, varicella zoster virus, mumps, HIV, adenovirus</th>
<th>Cryptococcus neoformans, Candida</th>
<th>Tuberculosis</th>
<th>Trauma, vascular malformation</th>
<th>Campylobacter jejuni, CMV, EBV, Mycoplasma pneumoniae, varicella zoster virus</th>
</tr>
</thead>
</table>

CMV, cytomegalovirus; CSF, cerebrospinal fluid; EBV, Epstein–Barr virus; GBS, Guillain–Barré syndrome; HSV, herpes simplex virus; MS, multiple sclerosis; PMN, polymorphonuclear leukocyte; RBC, red blood cell; SAH, subarachnoid hemorrhage; TB, tuberculosis; WBC, white blood cell.
Patients with a normal CT scan are still at risk for herniation in cases of fulminant meningitis due to disease progression. This risk should be discussed at the time of procedure consent.

**TREATMENT**

- Treat every patient with suspicion for meningitis until ruled out
- Administer antimicrobials immediately (Tables 14.2 & 14.3)
  - CT scan, LP, or blood cultures should not delay antibiotic administration
  - Any delay over an hour increases mortality rate by greater than 10%
- Empiric dexamethasone if unclear source
  - Ideally given 10 to 20 minutes prior to or concurrently with first dose of antibiotics; do not give if patients have already received antibiotics
  - Dose: dexamethasone 10 mg IV × 1; then q6h for a duration of 2 to 4 days
  - Not necessary if organism is unlikely to be *Streptococcus pneumoniae*
- Septic shock
  - Fluid bolus of 30 mL/kg IV over 1 hour
  - Goal mean arterial pressure (MAP) greater than 65 mmHg; the patient may require vasopressors to sustain after volume resuscitation
  - Trend lactic acid

**TYPES OF MENINGITIS**

- Bacterial
  - Epidemiology
    - In United States: approximately 3 cases per 100,000 people
    - Worldwide: ~500,000 cases per year
  - Treatment
    - Dependent upon organism
    - Initially broad coverage per Table 14.2
### Table 14.2

<table>
<thead>
<tr>
<th>Early Antibiotics Recommendations for Suspected Meningitis by Age</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute Symptom Onset (hours)</strong></td>
</tr>
<tr>
<td>---------------------------------</td>
</tr>
<tr>
<td>Decadron + 3rd-generation cephalosporin + vancomycin</td>
</tr>
<tr>
<td>Decadron + 3rd-generation cephalosporin + vancomycin</td>
</tr>
<tr>
<td>Decadron + 3rd-generation cephalosporin + vancomycin + ampicillin</td>
</tr>
</tbody>
</table>

### Table 14.3

<table>
<thead>
<tr>
<th>Suggested Antibiotic Dosing in Adults With Normal Renal Function</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Third-Generation Cephalosporin</strong></td>
</tr>
<tr>
<td>-----------------------------------</td>
</tr>
<tr>
<td>Ceftriaxone 2 g IV q12h</td>
</tr>
<tr>
<td>Trough goal: 15–20 mcg/mL</td>
</tr>
</tbody>
</table>

IV, intravenous.

- **Prognosis**
  - *Fatal* if not treated
  - High morbidity and mortality rates
Hospitalized patients should be placed on droplet precautions.

- **Viral**
  - **Treatment**
    - Primarily supportive
    - Patients with West Nile virus meningitis—high risk of respiratory failure
    - Discontinue antibiotics and steroids, if started for bacterial meningitis, once it has been ruled out
  - **Prognosis**
    - Most cases improve within 7 to 10 days
    - Some cases, such as West Nile, can take weeks to months

- **Fungal**
  - **Epidemiology**
    - *Cryptococcus* is one of most common causes of adult meningitis in Africa
    - *Histoplasma* in environments with bird or bat feces contamination, more common near Ohio and Mississippi rivers
    - Consider in immunocompromised patients
  - **Treatment**
    - Amphotericin B
    - Typically requires long courses of antifungals due to immunosuppressed state

**Bibliography**


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