The Emergent Management of Neuromuscular Disease in Intensive Care Unit (ICU)

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Neuromuscular Disorders in ICU

- **Admissions**: *Below 0.5% of ICU entries*, creating unique challenges in management
- **Prognosis**: New diagnoses lead to longer ICU stays but often have better outcomes compared to other neurological disorders
- **Goal**: Aim to restore prior function
- **Research Gap**: Need more studies and expert collaboration for enhanced patient care

Anterior Horn Cells and Peripheral Nerves

- **Anterior Horn Cells (Acute Flaccid Myelitis):**
  - Enteroviruses: Poliomyelitis, Enterovirus D68, Enterovirus D71
  - Arboviruses: West Nile Virus
  - Paraneoplastic Motor Neuron Disease
  - HIV
- **Peripheral Neuropathy and/or Polyneuropathy:**
  - ***Guillain-Barre Syndrome (GBS)***
  - Lyme Disease
  - Viral Infections: CMV, HIV, EBV, VZV
  - Endocrine: Hypothyroidism
  - Nutritional: Vitamin Deficiency (Thiamine, B12 or Nitrous Oxide Poisoning)
  - Vasculitic Neuropathy: Rheumatoid Arthritis, Polyarteritis Nodosa
  - Metabolic: Acute Intermittent Porphyria
  - Toxins: Heavy Metals, Ethylene Glycol, Methanol, n-Hexane
  - ***Critical Illness Polyneuropathy***
  - Malignancy: Leptomeningeal Malignancy

Neuromuscular Junction Disorders and Myopathies

- **NMJ (Neuromuscular Junction):**
  - ***Myasthenia Gravis***
  - Botulism
  - Tetanus
  - Tick Paralysis (Toxin interferes with Acetylcholine release)
  - Organophosphate Poisoning, Overdose of Anticholinesterases
  - Hypermagnesemia
  - Paralytic Medications (Effects may be prolonged by renal failure, hepatic disease, hypermagnesemia, metabolic acidosis)
- **Myopathy:**
  - Metabolic: Hypokalemia (Periodic Paralysis), Hypomagnesemia, Hypophosphatemia, Thyroid Disorders
  - Mitochondrial Disease
  - Inflammatory Myositis: Polymyositis, Dermatomyositis, Lupus, Scleroderma
  - Infectious Myopathies: Influenza, Coxsackievirus, HIV, Legionella Pneumophila, Borrelia Burgdorferi
  - Drug-induced Myopathy: Amiodarone, Cyclosporine, Hydroxychloroquine, Labetalol, Statins, Steroids, Zidovudine
  - Hereditary Myopathies: Late-onset Pompe Disease (most likely to present to ICU with undiagnosed respiratory muscle weakness)
  - ***Critical Illness Myopathy***
  - Mitochondrial Disease
  - Rhabdomyolysis

Key Points from Today's Presentation

- Guillain Barré syndrome (GBS) in the ICU
- Myasthenia gravis (MG) in the ICU
- Neuromuscular disease arising during treatment in the ICU (Critical illness polyneuropathy, Critical illness myopathy)

- Plasma exchange
- Intravenous immunoglobulin (IVIG)

Neuromuscular Emergencies

- Rapidly worsening weakness
- Respiratory failure and infection
- Oropharyngeal weakness and aspiration
- Cardiac failure or dysrhythmia
- Dysautonomia
- Acute rhabdomyolysis

Neck Flexion (Sternocleidomastoid Muscle) Weakness

- **Manifestation**: Can't lift head off pillow due to bilateral weakness
- **Causes**: Myasthenia Gravis, Myopathy, Guillain-Barre Syndrome
- **Correlation**: *Neck weakness often mirrors respiratory muscle weakness*
- **Implication**: Checking neck strength helps assess respiratory muscles, aiding patient care

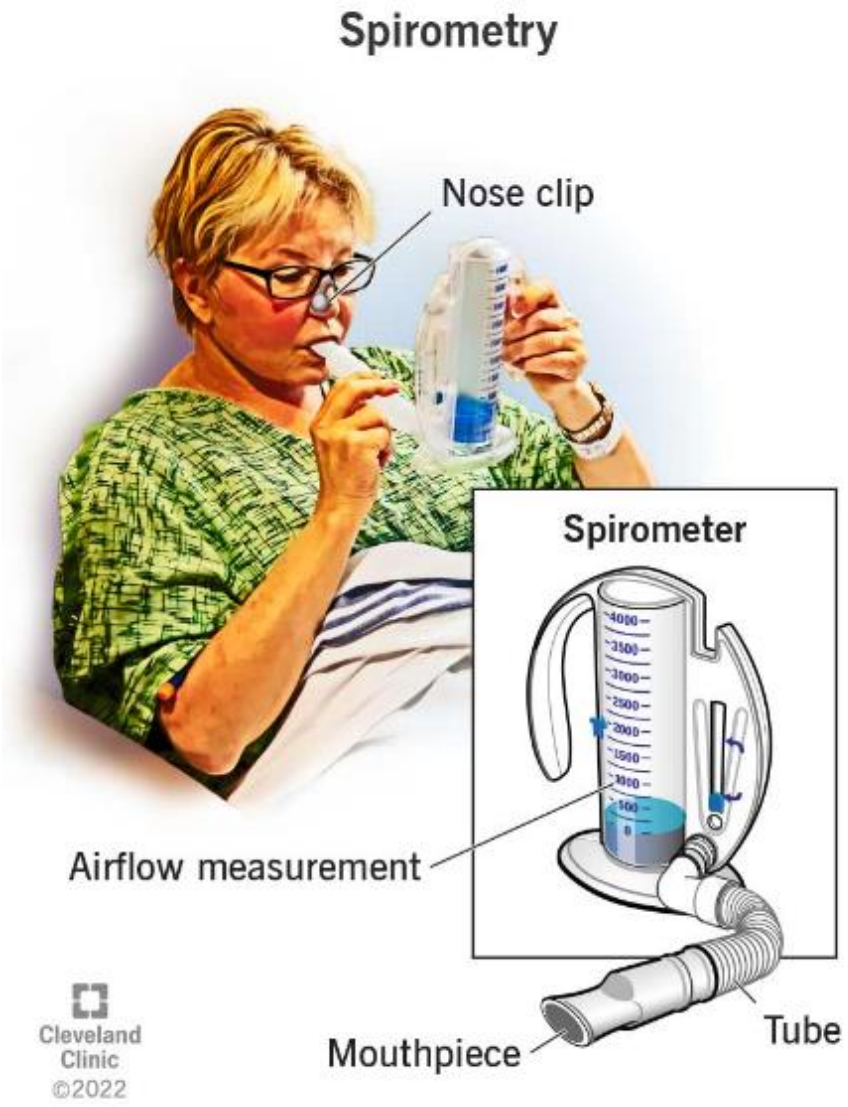
Pract Neurol. 2022 Jul 21:pn-2022-003422. doi: 10.1136/pn-2022-003422.

Laboratory Evaluation

- **Blood Tests:**
- **Electrolytes:** Including *Calcium, Magnesium, Phosphorus*
- **Creatine Kinase:** Elevation may indicate myopathy
- **HIV Screening:** If applicable
- **TSH:** Thyroid-Stimulating Hormone assessment.
- **Lumbar Puncture:**
- **CSF Normality:**
  - Myopathy.
  - Neuromuscular Junction Disorders.
  - Peripheral Neuropathies (Though abnormalities may occur in neuropathies *involving nerve roots*, e.g., Guillain-Barre Syndrome, CMV, HIV).
- **CSF Utility:**
  - **Guillain-Barre Syndrome:** Classic *Albuminocytologic Dissociation* (elevated protein with normal cell count). Protein elevation may take time to develop.
  - **Myelitis.**
  - **Pleocytosis:** Often indicative of infections (e.g., Encephalitis, Enterovirus, West Nile Virus, HIV, CMV) or inflammatory conditions (e.g., Sarcoidosis). Mild pleocytosis of *5-50 cells* may be seen in Guillain-Barre Syndrome.

Bedside Pulmonary Function Tests Forced Vital Capacity (FVC)

- **Definition**: FVC is the largest breath volume a patient can take
- **Reflection**: Covers Inspiratory Strength, Expiratory Strength, *Lung Compliance
- **Vs NIF**: FVC measures *more than diaphragmatic strength*, potentially predicting respiratory failure better
- **Benefits**: More consistent, less discomfort than NIF; *better for tracking* respiratory progress over time



- In obesity, the weight should probably be the ideal body weight

For men:
IBW=50*kg*+2.3*kg*×(height in inches−60)
For women:
IBW=45.5*kg*+2.3*kg*×(height in inches−60)

Rough Interpretation	cc/kg*
Normal	60 cc/kg
Concerningly low (Consider ICU monitoring)	<30 cc/kg
Very worrisome (At risk for intubation)	<15 cc/kg

Negative Inspiratory Force (NIF)

- **Definition**: max negative pressure during inhalation
- **Characteristics**:
  - More uncomfortable and effort-reliant than FVC
- **Use in Neuromuscular Disorders**:
  - *Limited for tracking progress in known cases* (e.g., Myasthenia Gravis)
- **Advantage**:
  - Better at measuring muscle strength with other lung issues (e.g., Obstructive Lung Disease)
- **Interpretation**:
  - Values between 0 and -30 cm water show severe respiratory muscle weakness

Utilizing Pulmonary Function Tests

- **Initial Diagnosis**:

  Identifies neuromuscular weakness in unclear respiratory failure cases

- **Evaluation & Triage**:

  Normal FVC with dyspnea suggests other issues (e.g., pulmonary embolism)

  Low admission FVC (e.g., <30 cc/kg) requires intensive respiratory monitoring

- **Tracking Progress**:

  Intermittent FVC measurements assess therapy response

  Thrice daily FVC measurement usually sufficient

  Look for *consistent trend* over multiple measurements for significance

Single Breath Count Test as a Surrogate for FVC

- **Role:**

  Alternative to Forced Vital Capacity (FVC) in resource-limited settings

- **Procedure:**

  Patient takes the deepest possible breath

  Counts upwards from one, as high as possible, at a rate of about two counts per second (120 per minute)

  Utilize a *metronome* to keep the beat

- **Attempts:**

  Generally, two attempts are allowed

  Higher count is considered the most accurate value

Interpreting Single Breath Count Test

- **Correlation:**
  - Roughly correlates with Forced Vital Capacity (FVC)
- **Count Interpretation:**
  - Count ***>25*** may indicate reasonably preserved FVC (over ~2 liters)
- **Limitation:**
  - Not validated in patients with concurrent respiratory issues (e.g., *pneumonia plus neuromuscular weakness*), unclear efficacy in such situations

Blood Gas Monitoring in Acute Neuromuscular Weakness

- **Worthless in patients without chronic respiratory dysfunction:**

-Normal respiratory drive should prevent hypercapnia until near exhaustion

-*Hypercapnia is an extremely late finding* unless medications like opioids are blunting respiratory drive

-Generally, not useful in most patients with acute neuromuscular weakness

Utility in Chronic Hypercapnia

- **Somewhat useful in patients with chronic hypercapnia (e.g., severe COPD):**

-Abnormal respiratory drive may lead to insidious hypercapnia without distress signs

-Instead of respiratory extremis, patients may accumulate CO2 quietly, leading to CO2 narcosis

-Useful in assessing altered mental status in such patients

Guillain-Barre Syndrome (GBS)

Treatment Indications in New-Onset Guillain-Barre Syndrome (GBS)

- **When to Treat:**

  -Initiate treatment with clinical diagnosis of GBS, no need to await lumbar puncture or nerve conduction study confirmations

  -Particularly crucial for critically ill or hospitalized patients with new-onset GBS

IVIG vs Plasma Exchange
&
Treatment-Related Fluctuations

- **Treatment Selection:**
  - Both treatments *equally effective*; *combination not superior*
  - IVIG preferred due to safety and ease: 0.4 grams/kg/day for five days
  - Plasma exchange as an alternative if IVIG contraindicated
- **Treatment-Related Fluctuations:**
  - Occur in up to *10%* of patients; common practice is to retreat with original treatment
  - More than three relapses or relapse after 8 weeks suggests Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)

Dysautonomia in Guillain-Barre Syndrome (GBS)

- More common in severe weakness cases, occurring in different GBS types: Demyelination or Axonal disease
- Patients may experience *alternating episodes* of sympathetic and parasympathetic hyperactivity, posing serious risks like bradyasystole
- Sustained hypertension is easier to manage compared to hemodynamic fluctuations
- Persistent hypertension can lead to target organ damage (e.g., Takotsubo cardiomyopathy or posterior reversible encephalopathy syndrome)

Hemodynamic Management Principles

- Blood pressure swings *often short-lived*; avoid treatment unless necessary to prevent worsening hemodynamic fluctuations
- *Avoid beta-blockers* to reduce bradycardia risk
- For persistent/critical hypotension: Administer fluids for true hypovolemia, or use vasopressors at the lowest effective dose

Addressing Persistent or Critical Hypertension

- Avoid treatment unless there's target-organ damage or severe ongoing hypertension
- 1st line: Address underlying causes (e.g., pain, agitation)
- 2nd line: Use *short-acting vasodilators* (nicardipine infusion), transitioning to *oral calcium-channel blockers* if stable.

Managing Other Autonomic Issues

- Urinary retention/incontinence: Employ Foley catheter or frequent bladder scanning
- Gastroparesis: Use pro-motility agents or a post-pyloric feeding tube
- Intestinal ileus/colonic pseudo-obstruction: Implement aggressive bowel regimen to prevent complications like bowel perforation
- Extreme caution with neostigmine due to autonomic swings risk; pre-treat with glycopyrrolate to reduce bradycardia risk

Noninvasive Respiratory Support

- **Early Initiation:**

  Lack of high-quality evidence; theoretically, *early noninvasive support* may prevent respiratory exhaustion and intubation
- **Utilization of BiPAP:**

  Beneficial in off-loading respiratory muscles at night, facilitating rest

  Ideal for nocturnal use; if patient is completely dependent, intubation is advisable
- **Alternatives:**

  High-Flow Nasal Cannula (HFNC) for patients with respiratory weakness not requiring intubation
- **Titration & Selection:**

  Largely based on patient tolerance; aimed at ensuring comfort while providing adequate support
- **Key Takeaway:**

  Balance between *early support* and *timely intubation* is crucial to prevent further respiratory deterioration

Pulmonary Function (Forced Vital Capacity)

- Provides a *piece of the overall clinical puzzle*
- Decision to intubate should never solely rely on respiratory mechanics
- Aberrantly low values may result from poor patient effort
- Beware of unvalidated “rules” for intubation based on specific cutoff values; they lack evidence basis

Tracking Weakness of Other Muscle Groups

- Respiratory weakness often correlates with:
  i) Neck flexion weakness (e.g., lifting head off the pillow relates to diaphragmatic weakness due to shared cervical nerve roots innervation)
  ii) Facial or bulbar weakness
  iii) Limb weakness (*exception: myasthenia gravis)

- Progressive *weakness across multiple muscle groups* is concerning and warrants closer monitoring

Clinical Evaluation & Decision to Intubate

- Numerous criteria for intubation exist, yet lack high-quality evidence backing
- Intubation decision is *clinical*, integrating multiple information sources:
  (1) Increased work of breathing signs (e.g., accessory muscle use, subjective dyspnea)
  (2) Difficulty in controlling secretions
  (3) Cough strength assessment
  (4) Overall muscle weakness progression
  (5) Trends in forced vital capacity
- *A comprehensive evaluation*

Intubation Procedure and Ventilation Management

- **Intubation**:
  - Be ready for *vagal episode* with push-dose epinephrine; avoid if possible
  - Check volume status; may need fluid resuscitation
  - *Skip Succinylcholine* due to potassium release risk from denervated muscles
- **Ventilation**:
  - Standard ICU protocols apply
  - *No preference for SIMV or other modes* in Guillain Barre Syndrome
- **Weaning**:
  - Follow usual steps; strength gain could allow extubation
  - Measure Forced Vital Capacity on ventilator during spontaneous breathing trials
  - *Diaphragm may recover before extremity muscles*; extubation viable with ongoing extremity weakness

Myasthenic Crisis

Definition of Myasthenic Crisis

- **Definition Variability:**
  - Some denote it as MG exacerbation needing intubation or noninvasive ventilation
  - Others refer to any MG exacerbation causing/threatening respiratory failure
- **Usage in this Lecture:**
  - Refers to respiratory dysfunction due to MG *necessitating ICU admission*
- **Initial Manifestation:**
  - Myasthenic crisis can be the first indication of MG in about *20%* of patients
- **Misconceptions:**
  - *Not all dyspnea in MG patients is myasthenic crisis*; other cardiopulmonary diseases like pneumonia, heart failure, or PE should be considered

Cholinergic Crisis

- **Definition:**

  Caused by excessive doses of acetylcholinesterase inhibitors like pyridostigmine, leading to elevated acetylcholine levels acting as a depolarizing paralytic *(akin to succinylcholine)*

- **Incidence:**

  Rare in modern practice due to standard pharmacologic dosing *(<120 mg every 3 hours).* More common historically with higher pyridostigmine doses. *Can occur with self-medication* exceeding recommended doses

- **Clinical Features:**

  1. **Escalated acetylcholinesterase inhibitor use:** Particularly doses *above 120 mg q3hr*
  2. **Skeletal muscle fasciculation**
  3. **Autonomic symptoms from acetylcholine excess:** Nausea, vomiting, diarrhea, salivation, lacrimation, diaphoresis. Miosis and bradycardia

- **Management:**

  1. **Cease acetylcholinesterase inhibitor administration**
  2. **Supportive Therapy:** Intubation if necessary
  3. **Post-recovery:** Reintroduction of lower acetylcholinesterase inhibitor doses

Triggers and Causes of Myasthenic Crisis

- **General:**

  Myasthenic crisis may be the initial manifestation of myasthenia in ~20% of patients
- **Common Triggers:**

  Infection (~40% of crises, notably pneumonia)

  Medications:

  Exacerbating medications
  Mismanagement of myasthenia gravis meds (e.g., pyridostigmine)
  Alteration in immunosuppressive or steroid regimen

  Electrolyte Abnormalities (Ca/Phos/Mg)

  Thyroid Disease (both hypo- and hyperthyroidism); associated with autoimmune thyroid disease

  Surgical Procedures/Trauma

  Pregnancy and Delivery
- **Undetermined Cause:** in about *one-third* of patients

Non-Invasive Respiratory Support (HFNC or BiPAP)

- *Initiate early* in mild respiratory distress to prevent exhaustion
- Failures occur when initiated in extremis
- HFNC: Safe, reduces work of breathing, improves ventilatory efficiency
- BiPAP: First-line for mild-moderate dyspnea, helps in oropharyngeal bulbar weakness by providing pneumatic stent
- Requires ICU supervision for effectiveness monitoring (e.g., minute ventilation and tidal volume on BiPAP)

Bedside Respiratory Assessment for Intubation Decision

- *Not solely based on pulmonary function tests or ABG/VBG trends*
- Assess:
  - Respiratory rate, work of breathing, distress signs
  - Cough efficacy, secretion clearance, airway protection
  - Neck flexion weakness trends
  - FVC values and trends
- Concerns: Worsening hypoxemia, atelectasis, aspiration, alternative overlooked diagnosis
- Chest radiograph for lobar collapse or aspiration signs

Intubation Procedure

- Use non-depolarizing paralytic (e.g., rocuronium, *~50% dose reduction*)
- *Avoid succinylcholine* due to reduced acetylcholine receptor density

Extubation & Tracheostomy

- *Extubation within 1-2 weeks, ~20% require tracheostomy*
- Aggressive MG management (e.g., plasma exchange) to enhance muscle strength for early extubation
- Standard extubation procedure, with planned BiPAP support to reduce reintubation risk, followed by nocturnal BiPAP until full recovery
- Risk factors for extubation failure:
  Age >50
  Pre-intubation serum bicarbonate >30 mEq/L
  Vital capacity <25 ml/kg in first week
  Pneumonia
  MuSK antibody positivity

Management of Steroids in Myasthenic Crisis

- **Existing Steroid Users**:
  - Maintain initial dosage.
  - Post substantial improvement with plasmapheresis/IVIG, consider dose escalation if initial dose was low
- **New Steroid Users**:
  - **Option 1**: Delay steroid initiation until post-plasmapheresis/IVIG and notable improvement
  - **Option 2**: Initiate with low dose (e.g., 20 mg prednisone), gradually up-titrate. *Low initial dose may prevent MG deterioration*

Plasma Exchange and IVIG in Myasthenic Crisis

- **Plasma Exchange - First-line for Severe Exacerbation**

  Directly extracts anti-acetylcholine receptor antibody, usually improving symptoms within *a few days*

  Advantage: *Rapid response*, beneficial for severe cases at risk of or already intubated, reducing ventilator-associated complications

  Especially effective for *MuSK+* patients

  Improvement often observed by 2nd or 3rd session

  Faster action compared to IVIG, crucial in severe exacerbation

- **Intravenous Immunoglobulin (IVIG) - Suitable for Less Severe Exacerbations**

  Takes *2-3 weeks* to manifest improvements, offering possibly more sustained efficacy

  Dosage: 2 grams/kg, typically spread over 5 days

  Alternative when PLEX is unavailable or contraindicated

Neuromuscular disease arising during treatment in the ICU

(Critical illness polyneuropathy, Critical illness myopathy)

There are 15 more slides……

Critical Illness Polyneuropathy

- **Definition**:
  - length-dependent, symmetric, *axonal sensorimotor* polyneuropathy
  - It *can develop rapidly* among critically ill patients, potentially *within three days*
- **Clinical Manifestations**:
  - Generalized weakness varying from moderate paresis with hyporeflexia to complete quadriparesis and areflexia
  - Symmetric, distal-to-proximal distribution of weakness
  - Respiratory function often affected, typically presenting as failure to wean from mechanical ventilation
  - *Usually, cranial nerves are spared*
  - *Sensory impairment* may be *subclinical*, though observed in about half of the patients
  - Initially preserved but eventually reduced or absent deep tendon reflexes; a distinguishing factor from critical illness myopathy

Critical Illness Polyneuropathy

- **Diagnostic Measures**:
  - **Nerve Conduction Studies**:
    - Axonal polyneuropathy evidenced by reduced amplitudes or absence of compound muscle action potentials (CMAPs) and sensory nerve action potentials (SNAPs)
    - Expect *normal latencies and conduction velocities*, indicating absence of a demyelinating process
  - **Cerebrospinal Fluid (CSF)**:
    - Typically, *CSF findings are normal* in CIP
- **Management**:
  - Predominantly *supportive*, aimed at alleviating symptoms and ensuring patient comfort

Critical Illness Myopathy

- **Occurrence**:
  - May manifest either *solely or alongside* Critical Illness Polyneuropathy
  - Associated with extended therapeutic paralysis, high-dose steroids, severe pulmonary disease, and sepsis
- **Clinical Symptoms**:
  - Proximal diffuse muscle weakness, occasionally *extending to facial muscles* (*excluding extraocular muscles*)
  - Preserved sensation
  - Relatively intact reflexes
  - Potential muscle wasting and challenges in weaning from mechanical ventilation

Critical Illness Myopathy

- **Lab Tests**:
  - *Creatinine kinase* generally remains *normal or mildly elevated*. Significantly elevated levels may indicate other conditions like rhabdomyolysis or inflammatory myopathy
- **Electromyography**:
  - Compound Motor Action Potentials (CMAPs) exhibit reduced amplitude and prolonged duration
  - Needle examination unveils fibrillation potentials and small, myopathic motor units
- **Treatment**:
  - Primarily *supportive*, including avoidance of known risk factors
  - Compared to Critical Illness Polyneuropathy, Critical Illness Myopathy presents a *more favorable prognosis* with patients often achieving complete recovery

Plasma exchange & Intravenous immunoglobulin (IVIG)

There are 10 more slides……

Basics of Plasma Exchange

- Process:
  - Insert a large-bore hemodialysis catheter
  - Exchange patient's plasma with albumin or fresh frozen plasma (usually albumin; fresh frozen plasma for patients with coagulopathy)
- Purpose:
  - Remove a wide range of proteins from blood
  - For example, to quickly get rid of auto-antibodies
- Note:
  - Plasma exchange can also take away *endogenous anti-inflammatory cytokines* from the body

Contraindications for Plasma Exchange

- Unable to get large-bore vascular access
- Severe hemodynamic instability (including severe dysautonomia)
- Serious coagulopathy (especially low levels of fibrinogen)
- Untreatable low calcium levels
- ACE inhibitors could raise the risk of severe swelling (angioedema)
- Pregnancy (relative contraindication): Hormonal changes from plasmapheresis could trigger early labor

Potential Complications

- Catheter-Related:
  - Pneumothorax, infection, hematoma
  - Catheter usually stays in for around 10 days, raising infection risk
- Medication Removal:
  - Some drugs get removed as they are either *highly bound to proteins* or are *proteins*.
  - Examples: Certain blood thinners, IVIG, rituximab, natalizumab, some antiseizure meds
- Other Complications:
  - Low calcium due to anticoagulant used in the outside-the-body circuit
  - Transfusion reaction (if using fresh frozen plasma)
  - Coagulopathy (removal of clotting factors)
  - Weakened immune system (goal is to remove immunoglobulins but this could affect response to infections)
  - Severe swelling (angioedema) as exchange can *boost bradykinin levels*

Basics of IVIG (Intravenous Immunoglobulin)

- Composition:
  - Made from pooled immunoglobulin from blood donors
  - Contains a variety of antibodies reacting to common antigens (e.g., infections)
- Mechanism:
  - *Unclear* how IVIG helps suppress autoimmune disorders
  - Might change immunoglobulin metabolism or compete for binding to Fc-receptors on immune cells

Contraindications for IVIG Administration

- Refractory volume overload (due to large volume of fluid in IVIG)
- Renal failure
- Known allergic reaction to IgA
- IgA deficiency:
  - Higher risk of severe allergic reaction (anaphylaxis)
  - Ideally, measure IgA levels before IVIG, *but in emergencies, IVIG is given without this check*

Potential Complications and Dosage

- Complications:
  - Drug-induced aseptic meningitis (DIAM)
  - Acute kidney injury, volume overload
  - Infusion-related side effects: fatigue, fever, nausea, headache, flushing (up to 24 hours)
  - Allergic reactions (especially with IgA deficiency), *thrombotic complications*, antibody-mediated cytopenias
- Dosage:
  - Typical dose: 0.4 grams/kg/day for five days.
  - Dosage based on ideal body weight in case of morbid obesity

Plasma Exchange vs. IVIG

- **General Controversy**:
  - Choice between Plasma Exchange & IVIG varies across centers
  - Decision influenced by local norms & logistics
- **Cost & Availability**:
  - IVIG: widely available but expensive
  - Plasma Exchange: limited to large centers, sometimes unavailable during nights/weekends, may be cost-effective
- **Disease-Specific Considerations**:
  - Guillain-Barre Syndrome: Equal efficacy
  - Myasthenic Crisis:
    - Plasma exchange preferred for faster action
    - More effective in MuSK+ patients
- **Individual Patient Considerations**:
  - Previous slides list contraindications/complications

Pitfalls in GBS Management in ICU

Autonomic Swings: Avoid over-aggressive treatment

Forced Vital Capacity: Guard against excessive focus and measurement

Negative Inspiratory Force: Judicious use

GBS Diagnosis: Consider in ICU patients with post-viral weakness

Respiratory Failure: Differentiate causes, not just neuromuscular failure

Pitfalls in Myasthenia Gravis Management in ICU

Differential Diagnosis in Dyspnea: Do not solely attribute dyspnea to myasthenic crisis. Consider other causes like pneumonia or heart failure

Pulmonary Mechanics Monitoring: *Avoid over-frequent measurements*, which can be distressing for patients

Intubation Criteria: Refrain from intubating solely based on arbitrary pulmonary mechanic thresholds; lack of data supporting this practice

Medication Caution: When introducing new medications, be aware of contraindicated drugs for myasthenia gravis patients

Thanks for your attention