Practical points of EMG in the intensive care unit

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Weakness in ICU patients

- Primary neuromuscular disorder
- Neuromuscular disorders arising in the ICU
- Systemic illness
- Medication
- Encephalopathy – hypoxia, trauma, metabolic
- Spinal cord damage


- 92 patients over a 4.5 year period
- 26 (28%) had neuromuscular disorders
  - GBS
  - Myopathy
  - Motor neuron disease
- 39 (42%) developed CIM
- 12 (13%) developed CIP
- Patients with CIM and CIP had similar outcomes.

Multiple organ failure (MOF)

- Two or more of the following
  - Lung – require mechanical ventilation
  - Kidney – oliguric renal failure, creatinine >200 umol/l
  - Liver – s-bilirubin >35 umol/l, GOT, LD elevated
  - Cardiovascular system – hypotension
  - Brain – depressed consciousness, abnormal EEG
  - Hematologic system - lymphocyte count ↓, platlet count ↓
  - Gastrointestinal – blood from nasogastric tube, melena
- Mortality 50-60%

SIRS – Systemic Inflammation Response Syndrome

- Response to severe infection or trauma
- Cytokines and free radicals released
- Affects the microcirculation
- Many patients that have been >1 week in the ICU have SIRS, especially if they have serious infection
- Mortality 50%

Disorders in the ICU where EMG is useful

- Primary neuromuscular disorders leading to ICU
  - Polyneuropathies
    - Polyradiculitis
    - Porphyria
  - Myopathies
  - Neuromuscular transmission disorders
- Critical illness polyneuropathy (CIP)
- Critical illness myopathy (CIM)
- Prolonged neuromuscular block (PNMB)
- Focal neuromuscular
Cascade of events leading to CIW

Treatment in ICU
- Trauma, infection, etc

Sepsis

Status asthmaticus
- Organ transplant

SIRS

Steroids
- NMB agents
- Immobilization

MOF

CIP
- Prolonged NM block

CIM

Causes of CIW?

CAUSES OF NEUROMUSCULAR WEAKNESS IN THE INTENSIVE CARE UNIT: A STUDY OF NINETY-TWO PATIENTS

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Onset of CIW?

Early development of critical illness myopathy and neuropathy in patients with severe sepsis

Jeffry Knox, MD, Tyler B. Harrison, MD, Mark M. Bick, MD, PhD, and Marc Been, MD

Abstract: Objective: To characterize the prevalence, time of onset, and course of neuromuscular dysfunction in patients with severe sepsis. Methods: We conducted a prospective cohort study in which consecutive patients with severe sepsis underwent weekly neurologic examination and nerve conduction studies (NCS) within 24 hours of developing severe sepsis and during their hospitalization. Results: Neurologic examination revealed abnormalities at admission in 18 of 19 patients (95%) on day 1. Among patients examined on day 7, 9 of 10 (90%) were abnormal. NCS were abnormal in 48% of patients on day 7, 12 patients on day 14, and 10 of 20 (45%) on day 21. Twenty percent patients examined on day 21 were normal. Conclusions: The cumulative incidence of neuromuscular dysfunction in this population was significant. The finding that the cumulative incidence of neuromuscular dysfunction was not different between day 7 and day 14 suggests that neuromuscular dysfunction is a potent marker of severe sepsis and may have clinical utility in determining outcome. The clinical importance of the findings of this study is limited by the small number of patients examined. The results of this study highlight the need for further research to better understand the pathophysiology of neuromuscular dysfunction in critical illness.

Time to onset of weakness

Figure 2. The cumulative incidence of neuromuscular dysfunction while in the intensive care unit.

Proportional rate of findings

Figure 3. Classification of neuromuscular dysfunction.

Prognostic significance of ENMG

Baseline Nerve Conduction Studies

Figure 1. Mortality rates based on initial electrophysiological abnormalities.
**Prolonged NM block - features**

- Generalized weakness
- Ptosis, external ophthalmoplegia
- Normal sensation
- Areflexia
- Transient reversal of weakness with Tensilon

**Special aspects of EMG in the ICU**

- Electrically hostile environment
- Catheters, bandages, etc limit access to limbs
- Edema of the limbs
  - Unreliable sensory and motor nerve action potentials
  - In the legs it may be necessary to use near-nerve needle electrodes
- Early investigation
  - Fibrillations may not yet be present
  - No or poor patient co-operation

**50 Hz alternating current artifacts**

1. Check your ground electrode
2. Disconnect electric motors on the bed
3. Switch the electric plug of EMG equipment
4. Disconnect non-vital electric devices
5. Not always possible to have perfect recordings

**Phrenic nerve neurography**

Phrenic nerve neurography

- High stimulus intensities required
  - 70-100mA, 0.2 ms
  - Painful
- Difficult or impossible to obtain responses in conscious obese subjects with short neck
- M-wave amplitude varies with respiration
  - Study during expiration if possible
- EKG artifact may be considerable
- Avoid stimulation of the brachial plexus

Phrenic nerve – reference values

- Latency 6.6 ± 1.4 ms
- Amplitude >200 uV
- Amplitude side difference large (>100%) even in healthy subjects

EMG of the diaphragm

EMG - diaphragm

Inferior view of the diaphragm
EMG of the diaphragm

EMG of the diaphragm

Bursts of activity during inspiration
Amplitude 200-500 μV
Individual MUPs difficult to identify
- in partial old neuropathies large MUPs may
- reduced amplitude in myopathies
Fibrillation potentials
- It may be useful to study the trapezius muscle (C3-4 myotome)

Complications
- Pneumothorax!!
- 2/1000 in a survey by Bolton (1996)
  - patients with COPD on ventilator
- In outpatients follow the patient for three hours
  - Chest X-ray
  - Re-examine
  - Instructions to contact the hospital if shortness of breath

EMG of limb muscles
- Lack of central activation
- Anterior tibial muscle can be activated by pressing the sole of the foot with a blunt object
Activation of m. tibialis anterior

Direct muscle stimulation

Direct stimulation of muscle fibers
- In patients with little spontaneous activity and voluntary activity
- Record with concentric electrode and surface electrode
- Stimulate with monopolar neurography electrodes distally close to the tendon
- Stim intensity 30-90 mA

Direct muscle fibre stimulation (DMS)

DMS

Direct muscle stimulation
Planning the examination

"Asking the right questions will provide the right answers"

Successful EMG in the ICU

- Understanding of the clinical problems
- Limitations due to the patient and the environment
- Proper technical skills
- Good collaboration with the ICU personnel

Strategy

- Is there a peripheral neuromuscular problem?
- Is it a pre-existing neuromuscular disorder?
- Is there an acute neuromuscular disorder?
- Is the weakness related with critical illness?
- Are there focal nerve lesions?
  - Ulnar nerve

Background information

- Diseases prior to admission to the ICU
- Causes that led to treatment in the ICU
- SIRS and MOF
- Drug treatment (vancuromium and steroids)
- CK?
- Ventilation?
- Level of consciousness
  - Co-operation
  - How much pain can you cause?

Clinical assessment

- Patient difficult to assess due to depressed consciousness or medication
- Muscle strength
- Reflexes
  - Tendon reflexes
  - Babinski

Neurophysiological methods

- EEG
- SEP
- MEP
- Blink reflex, masseter reflex
- EMG
- Neurography
- Decrement
Protocol - Neurography

- Sensory neurography
  - Radial and sural nerve unilaterally, No response - check the other side
  - Motor neurography
  - Median and peroneal nerve unilaterally, No response - check the other side
- Respiratory muscle weakness not a part of generalized muscle weakness, check phrenic nerve bilaterally

Protocol - Motor neurography

- Median and peroneal nerve unilaterally, No response - check the other side

Protocol - EMG

- One proximal and distal muscle in upper and lower extremities
- If patient needs a respirator and respiratory weakness is out of proportion compared to generalized weakness, diaphragm bilaterally
- Direct muscle fiber stimulation

Protocol - Decrement

- One distal and one proximal muscle
  - Trapezius
  - Hypothenar muscles

CIP diagnostic criteria

- Neurography
  - Reduced M wave amplitude in > 2 nerves
  - Reduced sensory nerve action potentials in > 2 nerves, Reduced amplitude not explained by edema
- Abnormal F waves
  - Anesthetics abolish F-waves (Propofol!)
- Normal or near normal conduction velocities
- EMG
  - Fibrillation potentials after 10-14 days

CIM - diagnostic criteria

- Normal sensory responses
- Reduced M wave amplitude
- Increased M wave duration
- Abnormal direct muscle fiber stimulation
- Motor units recruited early
- Small and polyphasic MUPs
- CK abnormal or normal
- Biopsy done only if another myopathy is suspected (polymyositis or other)

Problems

- Sensory nerve amplitudes may be reduced due to edema
- Fibrillations and low M wave amplitudes may either be a polyneuropathy or myopathy
- Patient has diabetes or other disorder causing PNP
- MUP analysis, both qualitative and quantitative, requires co-operation
Summary of strategy

- Try to examine the patient early when still some co-operation can be obtained
- Exclude neuromuscular disorders that started before admission to ICU
- Characterize disorder
  - Patients with SIRS are more likely to develop CIP
  - Patients treated with high doses of steroids and NMB are more likely to develop CIM
  - Some patients have prolonged NMB
  - Many patients have a combination of all three

Game over