Plenary I: How Do We Measure Up?
Quantitation in EDX and Clinical Practice

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David R. Cornblath, MD
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Table of Contents

Course Committees & Course Objectives 4

Faculty 5

What Is “Normal” in Nerve Conduction Studies?
Lawrence R. Robinson, MD 7

Using Nerve Conductions to Distinguish Axonal and Demyelinating Neuropathies
David R. Cornblath, MD 13

Quantifying Normal and Abnormal Needle Electromyography
Erik V. Stålberg, MD, PhD 17

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Chair: Holli A. Horak, MD

The ideas and opinions expressed in this publication are solely those of the specific authors and do not necessarily represent those of the AANEM.
Objectives

Objectives - Participants will acquire the skills to (1) explain the process for establishing normal values for NCS and identifying the variables that influence these, (2) discuss the patterns of abnormality found in NCSs and how these patterns relate to underlying lesions of the neuron, axon, and myelin sheath, and (3) describe the techniques for quantifying the results of the needle EMG and the relation between those results and the underlying lesions of the motor neuron, axon, myelin sheath, NMJ, and muscle cell.

Target Audience:
• Neurologists, physical medicine and rehabilitation and other physicians interested in neuromuscular and electrodiagnostic medicine
• Health care professionals involved in the management of patients with neuromuscular diseases
• Researchers who are actively involved in the neuromuscular and/or electrodiagnostic research

Accreditation Statement - The AANEM is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education (CME) for physicians.

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Plenary I: How Do We Measure Up?  
Quantitation in EDX and Clinical Practice

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Dr. Stålberg is an emeritus professor in the Department of Clinical Neurophysiology at Uppsala University in Uppsala, Sweden. He is a member of numerous professional organizations, including the Swedish Association of Clinical Neurophysiology, International Federation of Clinical Neurophysiologists, Deutsche EEG-Gesellschaft, British Society for Clinical Neurophysiology, and Canadian Society of Clinical Neurophysiologists. Dr. Stålberg was awarded AANEM’s Distinguished Researcher Award in 1994 and Lifetime Achievement Award in 1999. He has published about 450 scientific papers and a number of book chapters in the field of electrodiagnosis, mainly on electromyography (EMG). Dr. Stålberg has developed a number of EMG methods, such as single fiber, macro, and scanning EMG and methods for quantitation of conventional EMG. He has studied the microphysiology in normal and diseased muscle.

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What Is "Normal" in Nerve Conduction Studies?
What Is “Normal” in Nerve Conduction Studies?

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PURPOSE OF “NORMAL” VALUES

Electrodiagnostic (EDX) physicians commonly use nerve conduction studies (NCSs) to assist in the formation of a diagnostic impression. Part of this process is to examine the results of NCS testing and decide whether the results are more likely to come from a healthy individual or a person with disease. Based upon this analysis, one can put results in the clinical context and develop an overall diagnostic impression. This discussion describes important factors to consider in the interpretation of NCS results when deciding whether or not they are “normal”.

TERMINOLOGY

There are several problems with using the term “normal values.” First, other than this author, one cannot be sure that anyone else is truly “normal.” It is generally more helpful to think about the concepts of healthy individuals (those without symptoms or known disease) and those with disease. One cannot determine if someone is truly normal or not with NCSs, but it is the job of EDX physicians to determine which group each patient likely comes from with regard to a specific disease process: the healthy or the disease group.

Another problem with the term “normal value” is that, for the great majority of medical testing procedures, there is no clear boundary in measurements such that all people with a disease fall on one side of the measurement and all healthy individuals fall on the other (Figure 1). There is usually some overlap, such that some healthy people can have the same measures as some people with disease (Figures 2, 3). Because of this, NCSs cannot provide the assurance that everyone within a range is “normal” or “abnormal.” Rather, they indicate a level of probability that the measure came from a healthy person or someone with disease. Having a value outside the reference range indicates it is unlikely the value came from a healthy person (but not impossible).
However, with larger ranges of correction, these errors are magnified.

unknown "errors" in correction factors do not make much difference. 

nerves are delayed and small. If the limb is not very cold, then amplitudes are large, this generally indicates cold, whereas diseased proper reactions for large responses because when latencies are delayed, but response is large and if the limb is not very cold. This is more appropriate for large responses because when latencies are delayed, but amplitudes are large, this generally indicates cold, whereas diseased nerves are delayed and small. If the limb is not very cold, then unknown "errors" in correction factors do not make much difference. However, with larger ranges of correction, these errors are magnified and the use of correction factors introduces more error.

A second approach is to warm the limb. This is generally preferable if the limb is very cold. It is unclear which technique is most effective at limb warming, but infrared lamps, heating pads, warm water, convection heating (hair dryers), and other techniques are commonly used. This author’s personal preference is the hair dryer because of the ease of use, which means there is a low threshold for using it (the author also uses it for hair styling during a long day in the laboratory).

A third commonly used approach, particularly for focal neuropathies, is to compare results with another nearby nerve, which is presumably at about the same temperature. This works particularly well for studies of entrapment neuropathies where one nerve in the limb is affected and an adjacent nerve is not. This approach does not, however, work well in the setting of polyneuropathy where all nerves are affected.

**Age and Height**

Age and height represent different challenges than temperature, since these cannot be altered in the EMG laboratory. There are only a couple of choices to address the influences of height and age. One can examine a control group of healthy individuals comparable to the patient being measured (e.g., taller younger people). Alternatively, one can use adjustment factors to estimate what the result would have been if the patient had been at the mean of the control group for age and height. The former is preferable because it does not assume a linear relationship between age or height and the measurement, but it does require a large control group.

**The EMG Machine**

A couple of important EMG machine settings, including display sensitivity and filters, can materially influence the measurement of NCS results. When measuring peak latencies, there is little room for disagreement around marker placement: it is at the highest point of the waveform. However, whenever departure from a baseline (e.g., onset or duration) is measured, that measurement is markedly influenced by display sensitivity. It is important to remember that there is no "true" onset latency; rather, the onset is where the waveform departs from the baseline. When the sensitivity is higher and the waveform is larger, an earlier departure from the baseline is noted than when the sensitivity is lower. For median motor responses, for example, one notes a 0.3 ms difference in onset latencies, on average, between displays of 500 μV/div and 5 mV/div. For this reason, the display sensitivity should be the same for all studies and the same as that used in the collection of the reference values.

Although a less common issue with today’s EMG instruments, filters can also influence NCS measures. These, again, should be the same as those used in the collection of reference values.

**The Electrodiagnostic Physician's Brain**

How the EDX physician considers the NCS measures can also influence whether appropriate reference values are used. The level of training and experience will, for example, allow one to recognize the median pre-motor potential and measure the compound muscle action potential (CMAP) after this response rather than measure the

![Figure 3. Marked overlap between healthy and diseased groups. Testing would be of little help.](image)
CNAP in error. The experienced EDX physician will also use the right reference value for the right study, using it only when performing the same study for which the reference values were developed. An EDX physician with insufficient training may, for example, apply the reference values for long segment ulnar conduction velocity (e.g., 50 m/s) to short segment inching; this would result in many false-positive errors.

The experienced EDX physician will also select a reference value that appropriately balances sensitivity and specificity. This includes recognizing that there will unavoidably be some false-positive and false-negative results.

**STATISTICAL ANALYSIS OF CONTROL SUBJECTS**

Usually, at least for NCSs, one sets reference values by collecting measurements on a number of healthy control individuals, preferable similar in many respects (e.g., age and height) to patients seen in the laboratory. Based upon this control group data, one then determines the level at which one will start to think that a patient result does not come from a similar healthy group.

Although this method is the most commonly used, it is not the only way to develop reference values. One could, for example, measure the NCSs in a group of people with a known disease and compare them with a healthy control group and find the cutoff point that optimizes sensitivity and specificity. However, the former rather than the latter is usually used since the EMG laboratory is concerned with a number of different potential diseases.

There are two broad approaches to determine reference values based upon analysis of control data: parametric and nonparametric statistics. Parametric statistics originally come from repeated measures of the same physical observation. An example is repeatedly measuring the length of a hallway in the EMG laboratory over a year; one would not always obtain the exact same measurement, but one would more likely get a bell-shaped (Gaussian) curve, which has well described statistical features. For instance, 95% of the measures are contained within the mean ± 2 SD, and 99% are contained within the mean ± 3 SD (Figure 4).

This theory is often applied to the analysis of NCS data or other biologic measures. However, there is nothing that says NCS measures (a biologic phenomenon) across different subjects (not the same subject repeatedly) should follow a bell-shaped curve. In fact, they usually do not. Latencies are usually skewed such that many people have average latencies, but a few people have longer latencies (tail to the right, Figure 5). This may be in part because there is a boundary or limit to how short a latency can be, but less of a limit to how long it can be. Amplitude distributions are skewed as well; there is a definite limit to how small an amplitude can be (0) but not a firm limit to how large it can be.

Variables measured in a control group can be analyzed by parametric techniques using statistical software and a mean and standard deviation calculated, whether or not such analysis is meaningful. However, when the assumptions of a bell-shaped curve are not true, parametric analysis produces erroneous reference values that do not accurately represent the healthy control group. Sometimes this is especially evident for amplitude measures, when the mean −2 SD will even result in a negative reference value (which of course is physically impossible).

There are two ways to tackle this problem of skewness. First, one can transform the data, by taking the square root or logarithm, for example, of each value. For distributions with a tail to the right, this will often produce a bell-shaped curve. If so, then one may apply parametric statistics to the resulting distribution and determine reference values (e.g., mean ± 2 SD); reverse transformation is then required to obtain the reference values in their original units.

The other method is to use nonparametric statistics (i.e., those statistics that do not rely on the distribution of the control group). An example of a nonparametric statistic is the range (i.e., the lowest or highest value) observed in the control group. The range, while initially appealing, is problematic in that it is susceptible to error made in that single subject at the end of the range. Thus, percentile values are usually used near the ends of the range. For example, if one collected latencies on 100 people, the 97th percentile value (comparable to mean ± 2 SD) would be the value observed in the person 97 people up from the lowest number, or three down from the top number. This method does not depend upon the distribution, but it
does require a larger number of subjects. To get an accurate value at extreme ends of the range (e.g., 97th percentile) likely requires more than 120 subjects.

**THE PROBLEM OF MULTIPLE COMPARISONS**

Since reference values are typically set by collection of measurements in healthy subjects (as above), a small number of these healthy subjects should be expected to have observations outside the reference range. For example, if a mean ± 2 SD is used (assuming the distribution is a bell-shaped curve), then 2.5% of measures (one end of the bell curve) would be expected to have too long a latency, too small an amplitude, or too slow a conduction velocity. If one performs two separate studies in a patient or subject, this number is roughly additive. So, assuming results are independent of each other, there is an approximately 5% chance that one of the two studies will have results outside the reference range (or be “abnormal”). As stated above, these numbers are roughly additive; three studies produce a nearly 7.5% change of one abnormal result and four studies yield a nearly 10% chance (Table 1). As one performs more studies in a single individual the chance of a false-positive result by chance alone goes up quickly.

**Table 1. Probability of abnormal results by chance alone**

<table>
<thead>
<tr>
<th>Number of variables examined</th>
<th>Number of abnormalities</th>
<th>1 (%)</th>
<th>2 (%)</th>
<th>3 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>≤ 1</td>
<td>2.5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>≥ 2</td>
<td>0.1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>≥ 3</td>
<td>0.2</td>
<td>&lt;0.1</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>0.6</td>
<td>&lt;0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>11.9</td>
<td>0.6</td>
<td>&lt;0.1</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>14</td>
<td>0.9</td>
<td>&lt;0.1</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>16.2</td>
<td>1.2</td>
<td>&lt;0.1</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>18.3</td>
<td>1.6</td>
<td>&lt;0.1</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>20.4</td>
<td>2</td>
<td>&lt;0.1</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>22.4</td>
<td>2.5</td>
<td>0.2</td>
<td></td>
</tr>
</tbody>
</table>

This shows the probability of finding an abnormal result by chance alone according to the number of parameters studied. These calculations assume that 2.5% of an asymptomatic control population fall into the “abnormal” range for each parameter studied, and that each parameter is independent.

There are generally two ways to handle this problem of multiple comparisons. First, one could require that multiple results be abnormal to make a diagnosis. If one still performs three tests, there is about a 7.5% chance that at least one will be abnormal, but less than 1% probability that two or three of the three will be abnormal by chance alone.

Another approach is to combine the results of multiple measures into one summary variable and only compare this one summary variable to the reference group. An example of this is the combined sensory index (CSI). Following this approach brings the false-positive rate down to 2.5% (only one value is being compared to the reference range) but enhances sensitivity and reliability at the same time (Table 2). There are a number of special circumstances required to do this easily, however. First, the measures must be in the same units (i.e., ms or mV). Otherwise, they could be converted into number of standard deviations from the mean, which gets operationally more challenging. Second, they should measure the same concept. For instance, combining latencies and velocities may not make sense conceptually. Third, it should be easy logistically. This author has learned, for example, that EDX physicians enjoy doing addition and subtraction in the EMG laboratory. However, when it gets to higher level math, such as multiplication or division (required for weighted averages) adoption is difficult.

**Table 2. Reference values, sensitivity, specificity, and reliability (Spearman rho) for four different approaches to carpal tunnel syndrome**

<table>
<thead>
<tr>
<th>Test</th>
<th>Reference value</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mid-palm</td>
<td>≤ 0.3</td>
<td>70%</td>
<td>97%</td>
<td>0.74</td>
</tr>
<tr>
<td>Ring</td>
<td>≤ 0.4</td>
<td>74%</td>
<td>97%</td>
<td>0.67</td>
</tr>
<tr>
<td>Thumb</td>
<td>≤ 0.5</td>
<td>76%</td>
<td>97%</td>
<td>0.75</td>
</tr>
<tr>
<td>CSI</td>
<td>≤ 0.9</td>
<td>83%</td>
<td>95%</td>
<td>0.95</td>
</tr>
</tbody>
</table>

CSI = combined sensory index

**SUMMARY**

This was a nice discussion, but what should be done with this information upon returning home?

- Select reference values (whether from book or other source) carefully.
  - Make sure the methods used on patients are as similar as possible to those used in the collection of the reference population.
  - Filters, sensitivity, and methods should be the same.
  - Determine whether the patients being studied bare any similarity (i.e., age) to the control group studied.
- Look at how the reference values were analyzed.
  - Were nonparametric statistics used? If so, are 3rd or 97th percentile levels being used?
  - If using mean ± 2 SD, were the data skewed? Were they transformed?
- Expect false-positives results.
  - Even if the data were perfectly analyzed, about 3% (1/33) of NCS results should be outside the reference range in healthy subjects. If performing multiple tests per subject, as is common, occasional “abnormal” results among patients will be seen frequently.
  - If an “abnormal” result is found that is just outside the reference range, it is okay to discount it if it does not fit the clinical picture. That should happen in about 1/30-40 test results.
• Avoid the problem of multiple comparisons.

• Use a summary index, such as the CSI, when available.

• When performing multiple tests, require more than one abnormality to make a diagnosis. A single extreme value may, however, carry more weight than a value just outside the reference range.\textsuperscript{15}

• Do not hunt for abnormalities by performing one test after another if the prior one is normal. This greatly increases the chance of false-positives. Think out the entire strategy before starting.

• Use a healthy degree of skepticism in the interpretation.

• Do not over interpret small deviations from the reference range.

• Accurately express the level of certainty or uncertainty in the diagnostic impression: “It is an essentially normal study,” or “It is possibly suggestive of, but not diagnostic.”

REFERENCES

Using Nerve Conductions to Distinguish Axonal and Demyelinating Neuropathies
For polyneuropathies both symmetrical and asymmetrical (multifocal), traditionally the differential diagnosis was divided into axonal and demyelinating neuropathies. This is partly because the differential diagnosis of human demyelinating neuropathy is limited, and many of the demyelinating neuropathies are treatable (Table 1).

**Table 1. Demyelinating neuropathies**

<table>
<thead>
<tr>
<th>Hereditary</th>
<th>Acquired</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital hypomyelination</td>
<td>Guillain-Barré syndrome</td>
</tr>
<tr>
<td>Charcot-Marie-Tooth family</td>
<td>Chronic inflammatory demyelinating polyradiculoneuropathy</td>
</tr>
<tr>
<td>Refsum disease</td>
<td>Chronic acquired demyelinating polyneuropathies</td>
</tr>
<tr>
<td>Adrenoleukodystrophy and adrenomyeloneuropathy</td>
<td>Multifocal motor neuropathy</td>
</tr>
<tr>
<td>Metachromatic leukodystrophy</td>
<td>Diphtheria</td>
</tr>
<tr>
<td>Krabbe disease</td>
<td>Hexacarbon</td>
</tr>
<tr>
<td>Cockayne syndrome</td>
<td>Buckthorn intoxication</td>
</tr>
<tr>
<td>Cerebrotendinous xanthomatosis</td>
<td>Perhexilene</td>
</tr>
</tbody>
</table>

Using this definition of demyelination, there are a number of physiological consequences: conduction block, reduced conduction velocity, differential conduction velocities leading to abnormal temporal dispersion, prolonged relative refractory period, reduced maximum transmission frequency, altered response to temperature, ectopic impulses, and ephaptic transmission.

Here, the focus is on the clinical recognition of peripheral nervous system (PNS) demyelination, not at the more basic and experimental level. Therefore, only certain of these abnormalities can be detected with standard NCSs and includes reduced conduction velocity (CV), conduction block, and abnormal temporal dispersion. These are usually identified using motor NCSs. Kimura has written about the difficulty of detecting demyelination with sensory NCSs.

What is the best method of detecting PNS demyelination? One method is to look for illnesses without PNS demyelination and characterize the motor NCS. The prototype is amyotrophic lateral sclerosis (ALS). Another method is to look for illnesses of clear demyelinating neuropathy and characterize the motor NCS. The prototypes are Guillain-Barré syndrome (GBS) (acute inflammatory demyelinating polyneuropathy) and chronic inflammatory demyelinating polyneuropathy (CIDP). These however suffer from circular reasoning and are complicated by secondary axonal loss. Therefore, the focus here will be restricted to chronic conditions, not acute situations like GBS.

In 1962 and 1967, Lambert wrote two important articles on the electrodiagnosis of ALS. Concentrating on the NCS alone, he suggested that in ALS there would be normal sensory NCSs, normal motor NCSs with “CV allowed down to 70% of the average
normal value when muscles are severely affected,” and reduced motor evoked amplitudes. Lambert and others (see his articles) recognized that as motor amplitudes fell there are changes in distal latency (DL) and CV. His superb graph used mean values for the conduction parameters while most electrodiagnostic physicians now use the lower limit of normal (LLN). His LLN from the graph was −2.5 SD (Fig. 1).

The author and colleagues decided to relook at this question using the LLN, the value used in most laboratories, and for more nerves than just the ulnar nerve, and for the motor CV, DL, and F latency (Cornblath and colleagues). First, 61 patients with well-defined ALS were examined and then the data were validated in another 34 ALS patients. The examiners evaluated motor conduction parameters, correlated these parameters with amplitude, developed regression equations, developed confidence limits, and then tested the equations in 34 other ALS patients.

Using the peroneal nerve as representative, it was found that the range in distal amplitude is large, from 2% to 500% of the LLN, while the range of values of the conduction parameters is smaller (Fig. 2).

However, the data are not “normal” in a statistical sense requiring transformation so that parametric statistics could be performed. The best fit was transformation using the square root. When transformed, these equations describe the best fit for the data. Below is an example of the date from the peroneal nerve.

Regression Equations from the Peroneal Nerve

- \((\text{Sqrt Latency}) = 10.391 - 0.116(\text{SqrtAmp}) \pm 0.9794\)
- \((\text{Sqrt Velocity}) = 9.868 - 0.0551(\text{SqrtAmp}) \pm 0.5435\)
- \((\text{Sqrt F Lat}) = 10.179 - 0.0551(\text{SqrtAmp}) \pm 0.5435\)

Since the equations are awkward to use in practice, the data were eye-balled per the LLN raw data slides for the peroneal, median, and ulnar nerves.

In nerves with reduced amplitude:

- CV rarely fell to less than 80% of the LLN, and
- Distal and F-wave latency rarely exceeded \(1.25 \times \text{upper limit of normal (ULN)}\).

Why is it that motor conduction parameters such as CV and distal and F-wave latency change with loss of axons? This is because CV is proportional to nerve fiber diameter as was shown by Erlanger and Gasser for which they won the Nobel Prize. The smallest diameter motor nerve fibers (5-6 μm) distally in the legs conduct between 25 and 30 m/s. The smallest diameter motor nerve fibers (6-7 μm) in the arms conduct at 30 m/s. Thus, when there is a loss of large fibers from axon loss, one measures the conduction of the remaining fibers.

The notion that conduction values changed as amplitude fell were later incorporated into the ALS Criteria by El Escorial Revisited, 1999: motor conduction velocities > 70% LLN, distal motor latency < 130% ULN, and F and H latencies < 130% ULN.

Thus, these ALS studies provide “limits” for NCS parameters for axon loss. Values outside those “limits” are therefore possibly due to demyelination.

However, these numbers cannot be used blindly. It is important to remember to use common sense and clinical correlation. If there are concerns about the data, try to confirm findings in other nerves. Repeat the study later, if possible. Check sensory conduction across the same segments. Most important is not to overcall a study as demyelinating.

Practically, ask yourself several questions. First, is this person likely to have a demyelinating polyneuropathy? Remember there is not likely to be CIDP hiding inside every person, to paraphrase Sen. Joseph McCarthy. Second, motor NCSs with data are much more valuable than motor NCSs without data (no response nerves are a waste of time; see Koski and colleagues). Third, perform the study simply but technically well. Avoid techniques that are likely to create problems (e.g., proximal stimulation).

USING NERVE CONDUCTIONS TO DISTINGUISH AXONAL AND DEMYELINATING NEUROPATHIES
### Table 2. The nerve conduction study score card

<table>
<thead>
<tr>
<th>Parameter</th>
<th># nerves studied</th>
<th># nerves with response</th>
<th># with demyelinating values</th>
<th>% with demyelinating values</th>
</tr>
</thead>
<tbody>
<tr>
<td>DML</td>
<td></td>
<td></td>
<td>(DML &gt; 125-130% of ULN)</td>
<td></td>
</tr>
<tr>
<td>CV</td>
<td></td>
<td></td>
<td>(CV &gt; 70-80% of LLN)</td>
<td></td>
</tr>
<tr>
<td>F latency</td>
<td></td>
<td></td>
<td>(DML &gt; 125-130% of ULN)</td>
<td></td>
</tr>
<tr>
<td>H latency</td>
<td></td>
<td></td>
<td>(DML &gt; 125-130% of ULN)</td>
<td></td>
</tr>
<tr>
<td>dCMAP</td>
<td></td>
<td># nerves with values &lt; LLN</td>
<td>—</td>
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CV = conduction velocity, dCMAP = distal compound muscle action potential, DML = distal motor latency, LLN = lower limit of normal, ULN = upper limit of normal

The Nerve Conduction Study Score Card

- For each of the major parameters of the motor NCS, calculate the number of times the parameter was studied and then the number of values in the demyelinating range for the conduction parameters.

- The parameters are DL, distal compound muscle action potential (dCMAP), motor nerve CV, and F latency.

- Studies with no response count only for the dCMAP.

In 2011, Bromberg reviewed the criteria for CIDP and concluded findings similar to the author’s. He also suggested a Score Card (Table 2) adding distal duration and partial motor conduction block criteria:

- DL: > 125% of ULN
- CV: < 70% of LLN
- F-wave latency: > 125% of ULN
- CMAP negative peak duration (measured as return to baseline of last negative peak):
  - Median nerve > 6.6 ms
  - Ulnar nerve > 6.7 ms
  - Fibular nerve > 7.6 ms
  - Tibial nerve > 8.8 ms
- Prolonged proximal-to-distal negative CMAP peak duration: > 30%

In using such a Score Card, look over the totality of the study to decide if the major features are axon loss (usually a length-dependent loss of amplitudes, sensory more involved than motor, majority of conduction parameters not in demyelinating range) or demyelination (relative preservation of amplitudes and majority of conduction parameters in the demyelinating range). Do not forget to correlate clinically!

### REFERENCES

Quantifying Normal and Abnormal Needle Electromyography

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INTRODUCTION

Needle electromyography (EMG) was first used in physiology studies of firing patterns of the motor unit (MU). For that purpose, Adrian and Bronk (1929) invented the concentric needle EMG electrode with dimension that is still used. Jasper and Ballem developed the monopolar electrode and described denervation activity in 1949. In the late 1940s Kugelberg described the needle EMG pattern in myopathies and in the 1950s Buchthal presented the detailed pattern typical of neurogenic conditions. He was also the first to advocate quantitative analysis of needle EMG signals, at that time with time-consuming manual methods; the complete study of one muscle could take a few hours. Lambert’s first publication on needle EMG was in 1954, on “Electrical Activity in Myositis.” At the same time nerve conduction studies came into clinical use. Lambert was early to adopt this method and published an article on the use of neurography in motor nerves.

This overview will focus on the development of the different types of EMG and the way they have been quantified.

Buchthal and colleagues made thorough studies of the needle EMG technique itself and the generation of the signals and in particular defined ways for quantitation. The motor unit potential (MUP) parameters were mainly amplitude, duration, and number of phases. Since measurements were made from paper with a ruler, the display gain, paper speed, and thickness of the ink jet had to be defined. To be accepted as a unique MUP, and not an occasional superimposition of many MUPs, the signal had to be repeated with the same shape five times and have a short rise time.

NEEDLE ELECTROMYOGRAPHY PARAMETERS TO BE QUANTIFIED

Later, with a greater understanding of the relationship between the needle EMG signal and its generators, the concept of analysis has changed a bit, from signal description to concepts that attempt to interpret the signals in biological terms. The signal-generator relationship has been better understood from many simulation studies.

Quantification can be made visually, likely still the most commonly used approach, or with computer-supported methods. Both ways will be mentioned here.

The needle EMG investigation is typically performed to assess the electrical stability of the muscle fiber membrane; the size, number, and distribution of muscle fibers; neuromuscular transmission; recruitment; and the number of activated motor units (MUs).

Spontaneous Activity

The most common spontaneous activities with the muscle at rest are fibrillation potentials and positive sharp waves. These signals indicate membrane instability, and they are seen in denervation, in muscular dystrophies (likely due to loss of innervation in segments of muscle fibers after focal necrosis), and in critical illness myopathies, where ion channel disturbances may produce this activity.

Other types of spontaneous activity include complex repetitive discharges (CRDs) generated by ephaptic activation of adjacent hyperexcitable usually denervated muscle fibers; myotonic discharges seen as waxing and waning single fiber action potentials; myokymic
discharges, either from muscle but more often bursts of activity from individual axons; and fasciculations, which are usually generated in the distal arborization of the axons. No automatic method for quantification of spontaneous activity is yet available and the amount of this activity is assessed visually as 0 (no activity) to 4+ (abundant) for each defined type of spontaneous activity, or as the number of sites out of 10 recording sites with this activity. Fasciculations or CRDs may be described as being “present” or “absent.”

**Needle Electromyography at Slight Voluntary Contraction (Motor Unit Potential)**

In the second step (after spontaneous activity), MUPs are studied at slight voluntary effort. This means that the first recruited type I MUs are studied. This is quite acceptable, since it is rare for muscle pathology to be restricted to only one type of MU. The MUP is the temporal and spatial summation of individual single fiber potentials within the uptake area of the electrode (Figure 1).

**MUP** = motor unit potential, **p** = phase, **t** = turn

This has a radius of about 2 mm which should be compared to the average diameter of the MU territory in a limb muscle, which is 5-15 mm. The anatomical correlates of the MUP are roughly as follows: the duration parameter is mainly dependent on the number of muscle fibers that lie within the uptake area of the electrode. The low amplitude signals surrounding the depolarization event in each muscle fiber summate to produce the initial and late parts of the MUP that define the onset and end of duration. The greater the number of muscle fibers, the longer the distance over which the signal can be detected, thus producing a longer MUP duration. To a lesser degree, the duration of a normal MUP is affected by other factors such as variable conduction in nerve terminals or aberrant position of the end plates. The amplitude parameter represents the number of muscle fibers close to the tip of the recording electrode. Usually the spiky component of a normal MUP is generated by two to five fibers that lie within 0.5 mm of the electrode tip, more in cases of reinnervation. The normal MUP is usually rather homogenous in shape, more so when recorded close to the end-plate region. Towards the tendon the normal difference in conduction velocity among muscle fibers in the MU may cause temporal dispersion, seen as separate peaks or phases in the signal. In pathology with increased fiber diameter variation, the temporal dispersion can be very pronounced giving rise to serrated (separate peaks not crossing the baseline) or polyphasic signals (signals that cross the baseline many times). Another important parameter assessed both visually and automatically is shape variability at consecutive discharges: the MUP instability, or “jiggle.” This reflects the neuromuscular jitter for each contributing muscle fiber and is increased in reinnervation and in myasthenic disorders.

The MUP is traditionally classified from its peak-to-peak amplitude, duration, number of phases (zero crossings +1), or number of peaks. A MUP is called polyphasic if it has more than four phases, and serrated or complex if it has more than five peaks.

**Analysis**

Visual analysis can be performed as a semiquantitative process just by watching and listening to the signals. However, visual analysis can also include some quantitation. It can be performed by using a trigger and delay line, by which the signal can be frozen on the screen and measured by setting manual cursors. This is time consuming, and it typically gives a bias towards the highest MUPs, since the so-called “amplitude trigger” is the most common type of triggering. Other methods such as a “window trigger” are implemented in some equipment but are not used in routine needle EMG studies.

Automatic MUP quantitation is usually made by a template method. These “decomposition-based” methods identify sharp signals (individual or summed MUPs) and save them as “templates.” A template-matching algorithm is used to identify identical signals that appear more than four times and are thus unlikely to be the summation of different MUPs. All the discharges from a given MU are averaged to obtain the MUP (Figure 2). For analysis, the parameters are mathematically defined, and they are therefore independent of amplifier gain. With good algorithms for this extraction, very little editing is necessary; therefore, these methods are relatively user independent. With the MUPs available in digital form, other parameters can also be analyzed, such as.
Figure 2. Example of MUP analysis. Top: Numerical data with frequency histograms of amplitudes and durations and a scatter plot of duration versus amplitude (log amplitude) (outlier and mean value limits are shown as boxes). X is the mean value of duration and amplitudes. Bottom: Averaged MUPs.

MUP = motor unit potential

Area Integrated positive and negative area under the signal within the duration cursors.

Thickness Area/ amplitude, a synthetic duration measure.

Size Index Thickness normalized for peak-to-peak amplitude.

Number of turns A measure with similar physiological meaning as polyphasicity.

Jiggle The instability of the MUP shape at repeated discharges, seen best with a triggered sweep and enhanced by a high pass filter set at 500 or 1,000 Hz. It is usually assessed visually, although automatic methods have been developed, but not implemented. This parameter corresponds to the neuromuscular jitter of the individual motor end plates that contribute to the MUP.

Complexity The length of the signal normalized for amplitude. It has not been implemented in any routine equipment yet, but it represents a parameter that awaits clinical testing.

Other methods for analysis have been tried, such as the Wavelet method. This method does not include cursor setting for duration, the most uncertain parameter. Recent results indicate that this approach is promising and needs further testing.

Reference Values

The need for reference values for any form of quantitation is obvious. For visual assessment (eye-balling), the reference is mainly the experience of the operator, a subjective description, which is difficult to transfer over time and to other investigators. For quantitative analysis methods, values obtained from single laboratories or from multicenter studies for the various muscles with reference data including age, height, and other parameters of interest describe reference limits for mean, SD, and percentiles. In this way, results obtained from a given study can be described in terms of deviation from mean or upper normal limits for matched control subjects. Another way to express abnormality is by using “outliers.” In the outlier-based approach, reference values are defined for individual MUP measurements. A study of 20 (or fewer) MUPs is considered abnormal when more than two outlier MUPs are recorded. In some situations of homogenous pathology among MUs, these two ways of expressing abnormality may be equally good. When there is heterogeneous involvement of MUs, outliers may be more sensitive in detecting pathology.
Typical Motor Unit Potential Findings in Pathology

The typical MUP shape in muscular dystrophy is polyphasic and of short duration, except for those that are extremely polyphasic and low amplitude. In some situations the duration may be long due to many late components; in others the duration is normal. Likewise, the amplitude may be normal (Figure 3).

Figure 3. MUP analysis in a patient with myositis. Note the increased polyphasicity which makes this study abnormal. Some amplitudes are low, some are high, and the mean values for these parameters are within normal limits. The IP analysis shows a myopathic pattern.

amp = amplitude, dur = duration, IP = interference pattern, MUP = motor unit potential, tib ant = tibialis anterior

The polyphasicity in myopathy is mainly due to the increased variation in fiber diameter with the understanding that propagation velocity along a muscle fiber is correlated to fiber diameter. The low amplitude is due to loss of muscle fibers in the uptake area, and to temporal dispersion among contributing spikes, which decreases the chance of signal summation. Occasionally, some MUPs have high amplitude and short duration, being generated by hypertrophic muscle fibers close to the electrode or even by locally-increased fiber diameter along the fiber (e.g., at the point of longitudinal splitting). This should not be misinterpreted as an evidence of a neurogenic process.

In patients with neuropathy there is also an increased incidence of polyphasic MUPs and this abnormal parameter is thus nonspecific. Here also the cause is the variation in fiber diameter, but it is sometimes due to aberrant motor end plates from reinnervation. Differentiation between myopathy and neuropathy is determined from a combination of these along with other parameters. The amplitude and duration of MUPs are increased because of an increased number of muscle fibers in the uptake area of the electrode due to reinnervation. Jiggle is an important parameter in neurogenic conditions. Increased instability is seen during ongoing reinnervation (e.g., in the early phase after nerve injury or in progressive neurogenic conditions Figure 4, while the MUPs are more stable in chronic neurogenic conditions.

Figure 4. Two MUPs in the biceps brachii muscle in a patient with chronic inflammatory demyelinating polyradiculoneuropathy. A: A MUP in raster mode, filters 5 Hz-10 kHz. C: The same filters, superimposed signals from MUP A. B: The same MUP (not exactly same time epoch) in raster mode, filters 500 Hz-10 KHz to better demonstrate the sharp components. D: A MUP in superimposed mode. The amplitudes are increased and the signals are long and polyphasic. There is increased jiggle.

MUP = motor unit potential

The patterns of abnormality are similar for concentric and monopolar needle electrode recordings, but the absolute values of MUP measurements differ between them. The monopolar electrode records MUPs with slightly higher amplitude and complexity. The duration measurements are based on slow components that are recorded similarly by both needle types and are therefore similar within the two electrodes. The MUP area, thickness, and size index are low in myopathy and high in neuropathy. It should be noted that the different parameters change with pathology to different degrees, depending on the muscle and the pathologic condition. In muscles with normally long duration MUPs, this parameter tends to increase more in neuropathy than in muscles with normally short duration. In facial muscles, the amplitude does not increase as much as the number of phases in reinnervation, due to the general shape of the MUP in normal facial muscles.

Needle Electromyography During Increasing and Strong Effort

In the third step, increasingly strong activity is analyzed. Force increases by increasing the firing rate of individual already active motor neurons and the orderly recruitment (from small to large) of MUs. Different types of recruitment abnormalities are seen in central, myopathic, and neurogenic disorders.

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The needle EMG signal obtained during moderate and strong effort is sometimes called the “interference” pattern (IP), or “pattern at full effort.” At full effort the baseline should be completely obscured. In myopathies, MUs are recruited early; there is disproportionally more needle EMG activity than the developed muscle force, but the amplitude is typically low. With neurogenic loss of MUs there are gaps in the signal due to reduced numbers of MUs that can be activated.
(Figure 5). In central disorders, the firing rate is irregular and lower than normal, and this also produces a reduced fullness. The amplitude of the IP signal increases with recruitment of new MUs. Some of the later recruited MUs will have higher amplitude MUPs, but the main reason for the impression of increasing amplitude is likely that the newly recruited fibers happen to be closer to the electrode tip than those initially seen at slight contraction. The size principle cannot be seen with conventional needle EMG.7

![Figure 5](image)

**Figure 5.** Interference pattern at full effort in myopathy (A), normal (B), and in status postpolio (C). In A there is a dense pattern of low amplitude and in C a reduced interference pattern with individual MUPs of high amplitude. Horizontal lines indicate roughly what the eye would assess as the envelope amplitude.

Modified with permission from Stålberg and Daube.22

**Analysis**

With standard gain and sweep speed settings, the experienced electrodagnostic medicine (EDX) physician can classify the obtained pattern by visual assessment.22 The sound of the needle EMG signals is also useful in the subjective description. Long duration MUPs tend to have a dull sound while polyphasic potentials with short duration give a high pitched sound. Even the jiggle can be heard.

The recruitment can be quantitated with careful analysis and with slight muscle activation. The technique is conceptually simple but quite challenging and therefore is not implemented as an automatic method for two reasons. Firstly, it requires significant patient cooperation to activate single MUs, which is often not possible in myopathy. Secondly, and more importantly, there is no formal definition for which MUs should be accepted for analysis. The onset frequency is the lowest regular firing rate of a MUP. Recruitment frequency is the firing rate of the last recruited MU at the point when an additional MU starts to fire. In this case the recruitment frequency is 23 Hz during a transient increase in firing rate of the first MU. Just before and after this period it is around 12 Hz.

![Figure 6](image)

**Figure 6.** Recruitment frequency. The interdischarge interval (reciprocal of the firing rate) of the first MU (with mainly positive going components) is measured (between arrows) at the time when the second MU (*) starts to fire. The presence of high amplitude long duration MUPs in neurogenic conditions and the reverse in myopathies was used by Willison26 in his method of measuring the number of turns/second and amplitude between turns. The absolute values of these parameters depend upon the force of contraction and, hence, early studies had to standardize the force level. Stålberg and colleagues described the turns and amplitude (TA) method in which the IP was recorded at force levels ranging from slight to maximum effort.21 A plot of turns/second versus amplitude is constructed for the individual patient and compared with data from control subjects, a so-called “normal cloud.” Limits were defined so that more than 90% of data points should be inside this cloud. Contrasting patterns of data distribution in pathology (Figure 7) make it useful in differentiating neuropathy and myopathy in a quantitative way. The relationship between turns and amplitudes can also be expressed as a ratio.5 Nandedkar and colleagues proposed a further development of the TA method with a new set of measurements that mimic visual assessment (Figure 8).15 This measures the “fullness” of the IP as “activity.” Amplitude is measured as the “envelope” of peaks after excluding some isolated very high negative and positive amplitude peaks similar to intuitive eye-balling. The number of small segments (NSS) measures low amplitude and short duration spikes that give the high-pitched sound to the needle EMG. The plots and statistics of these parameters are used to assess the IP. In patients with neuropathy the activity is decreased indicating an incomplete or reduced pattern (i.e., the baseline is not completely obscured by MUPs), and the amplitude is increased. The NSS is decreased, indicating the presence of mainly low frequency signal (i.e., dull sound). In contrast, patients with myopathy have normal activity values (a full pattern where the baseline is obscured by MUPs), decreased amplitude, and increased NSS (high-pitched sound). A comparison between methods is seen in Figure 9.
OTHER ELECTROMYOGRAPHY METHODS

Other special EMG methods have been developed to obtain more quantitative information about the MU.

Single Fiber Electromyography

Jitter is a quantitative measure of neuromuscular transmission, and it has been used mainly in the diagnosis of myasthenia gravis. Modules for this analysis are implemented in most new EMG equipment. Fiber density is a measure of the organization of muscle fibers in the motor unit.

Macro Electromyography

Macro EMG records activity from the cannula of a single fiber EMG (SFEMG) or concentric electrode, providing a nonselective measure of activity from MUs and giving information about the size and number of their muscle fibers. This has been used, for example, to follow patients with a history of polio and in patients with amyotrophic lateral sclerosis.

Scanning Electromyography

In scanning EMG an electrode is moved in small and defined steps through the MU and records its electrical cross section. This method has helped in the understanding of the distribution of fibers in pathological conditions and also of motor end plates.

Motor Unit Estimation, MUNE

Motor unit number estimation (MUNE) is a group of methods by which the number of MUs in a muscle can be estimated by comparing the size of the compound muscle potential at electrical stimulation and the average amplitudes of individual surface-recorded MUPs. Similar information is partly obtained from interference pattern analysis, but the MUNE methods provide better quantitative information of this important parameter.

SHOULD ALL ELECTROMYOGRAPHY METHODS BE APPLIED IN A ROUTINE STUDY?

As seen above, many parameters and methods are available for quantitative analysis of the EMG. It is not feasible to use all of them at all times. Each measurement provides a different piece of the puzzle (Figure 10). At each phase of the ongoing EMG study, one has to carefully choose the test that will best help in making the diagnosis, prognosis, and followup. This does not always imply that one should choose a method or parameter that has the highest sensitivity and specificity for a given disease. Not infrequently, alternative diagnoses need to be ruled out. For example, in a patient with a wrist drop that may have a radial palsy, distal myopathy should be ruled out. Here, one should use a method that is good at detecting myopathy rather than neurogenic changes. A good history and physical examination will reduce the number of diseases in the differential diagnosis and, thus, reduce the number of tests necessary to perform, patient discomfort, and the cost of study.
The parameters used for followup are not necessarily the same as those used initially to make the diagnosis. In a prospective EMG study in patients with inclusion body myositis, concentric MUP analysis was demonstrated to be sensitive for diagnosis but macro EMG was better to follow the change in MU size when the patients became weaker.\(^{1}\) In a question of myasthenia gravis, SFEMG is the most sensitive parameter. It is invasive, somewhat time consuming, and indicates the status only in the studied muscle. One may prefer to monitor vital muscles once the diagnosis is settled. This can be made with quantitative strength measurements or with clinical methods.

Quantitative analysis of the EMG is superior to visual subjective assessment in terms of sensitivity and ability to follow changes over time. Many laboratories have been slow to take advantage of this, partly for historical reasons when quantitation was made manually and therefore remembered as time consuming. Today, MUP quantitation takes about 3 minutes for one muscle, compared to a few hours with old manual methods. The analysis itself is reproducible between experienced and nonexperienced physicians and over time. Unfortunately, different EMG equipment use slightly different algorithms for the definition of signal selection and of some parameters, particularly the MUP duration. Therefore, each equipment type needs its own reference values. Interference pattern is analyzed in even less time. This is an important part of a complete EMG investigation. It gives information both about the individual MUPs and about the number of firing MUs and their behavior.

Particularly in situations with borderline visual impressions, it is important to perform quantitative analysis for optimal quality, a service EDX physicians should be able to give referring physicians and patients. Modern EMG quantitation does not take more time, effort, or patient discomfort than subjective assessment.

Quantitative analysis of needle EMG can be quite challenging in pediatric patients but is still possible.\(^{10}\) Stimulation SFEMG can be quite feasible in children (e.g., in the evaluation of congenital myasthenia).\(^{11}\) Recent reports have also shown that it may be possible to use surface EMG electrodes to differentiate between myopathy and neuropathy.\(^{26}\) Further studies are needed to introduce this method into general use.

The use of automatic quantitative methods are also very useful in teaching. Importance of good signal quality becomes obvious, otherwise the computer may give alarm. The measurements made by the computer system are consistent and, therefore, guide the user how to obtain standardized results. It can also be used to test the inter- and intra-operator reliability and so improve quality. It gives the less experienced users a valuable feedback on their subjective assessment.

CONCLUSIONS

With improved knowledge about the relationship between the EMG signal and its generators, the MUs, EMG quantitation rests on scientifically and clinically firm ground. The methods used in quantitative analysis have different sensitivity and specificity, and vary with the disease, the skill of the EDX physician, and the ease by which the final analysis can be performed. Of utmost importance is also the availability of reference values, linked to specific equipment and the algorithms that are used. Through quantitative analysis, the sensitivity to detect subtle abnormalities in EMG increases. It is possible to assess severity, monitor the progression of disease, and provide prognosis more precisely. Needless to say, this ultimately benefits the patient.

FUTURE PERSPECTIVE

Quantitation of EMG is still used routinely only in some laboratories. This may be due to insufficient software capacity in small EMG equipment and lack of reference values, especially for monopolar electrode recordings, and the fear that the procedure time will increase. Once implemented in a laboratory, however, this way of working will likely be appreciated and maintained since it improves the quality, sensitivity, and standardization, and it permits monitoring changes over time quantitatively.

Gross recruitment abnormalities are recognized quite easily on subjective examination but future analysis should continue to develop sensitive sets of measurements that quantify what EDX physicians hear and see.

With the large number of parameters that can be measured in an EMG pattern, improved interpretation likely can be obtained in different statistical ways, such as multidimensional analysis of all measurements. The algorithms must be robust, and evidence-based rules for interpretations must be further developed. These must be supported by administrative facilities such as help databases (anatomy, electrode placement), strategy suggestions based on guidelines, and evidence-based data.

Surface EMG has been tried in various applications. One is multichannel recordings which can be mapped and the conduction of surface recorded MUPs can be studied. Methods for single channel recordings which are suggested to differentiate pathology from normal pattern will be tested more intensely.
If use of quantitative EMG is pursued to improve sensitivity and reproducibility, electrodiagnosis will remain competitive and an important tool among many new complementary modalities to study nerve and muscle diseases such as biochemistry, genetics, and imaging techniques.

REFERENCES