neurosciences india group

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Electrophysiology Workshop

Venue:

Ramanujam Auditorium, III Floor, Madras Institute of Neurology,
Madras Medical College &
Government General Hospital, Chennai

Date : 12th - 14th Aug. 2005

Madras Institute of Neurology,
Madras Medical College, Chennai

12TH AUGUST 2005

EEG

Lead Faculty: **Prof. MBM Sundaram, Professor of Neurology, University of Jackson, Mississippi, USA**

0700-0800 Hrs: Registration & Breakfast

0800-1030 Hrs: Basics of EEG

- Technical Aspects
- Normal awake and sleep patterns in adults
- Recognizing artifacts
- Normal Variants
- Focal and generalized slowing
- Patterns in encephalopathy and coma
- Uses and limitations of EEG
- How to write a report

1030-1100 Hrs: Coffee Break

1100-1300 Hrs: Pediatric EEG

- EEG in full-term neonates
- Evolution of EEG through childhood
- Unique patterns in childhood EEG
- Seizures in childhood

1300-1400 Hrs: Lunch

1400-1630 Hrs: Advanced EEG

- Uses of Video EEG Telemetry
- Recognition of various partial and generalized seizures
- Non-epileptic spells
- Invasive Monitoring
- Surgical issues

1630- 1700 Hrs: Tea

1700-1800 Hrs: How to do it! A live interactive demonstration of a gold-standard EEG recording

13TH AUGUST 2005

ENMG

Lead Faculty: **Prof. V. Vedanarayanan,**
Professor of Pediatric Neurology,
University of Jackson, Mississippi, USA

0700-0800 Hrs:	Registration & Breakfast
0800-0915 Hrs:	Basics of NCS and EMG: trouble shooting & pitfalls
0915-1030 Hrs:	Mononeuropathies in upper and lower limb: Addressing diagnostic criteria, techniques, issues of anatomical variations; evaluating time of injury and prognosis
1030-1100 Hrs:	Coffee Break
1100-1400 Hrs:	Seminar Hall, Madras Medical College
the	♦ Shri. 5th Appa Rao Lecture - An Approach to Patient with Intractable Epilepsy - Prof. MBM Sundaram
-	♦ Shri. 7th ES Krishnamoorthy Memorial Lecture Recent Advances in Neurogenetics - Prof. V Vedanarayanan
	♦ Lunch
1400-1600 Hrs:	Polyneuropathies, Mononeuropathy Multiplex, Brachial Plexopathy, Lumbosacral Plexopathy: Addressing diagnostic criteria, techniques, issues of anatomical variations; evaluating time of injury and prognosis
1600-1630 Hrs:	Tea Break
1630-1800 hrs:	♦ NCS in Upper and Lower Limbs- Basics
	♦ NCS in Upper and Lower Limbs- Nerves Tested Less Frequently
1800 Hrs:	Close

14TH AUGUST 2005

ENMG

Lead Faculty: \	**Prof. V. Vedanarayanan,** **Professor of Pediatric Neurology,** **University of Jackson, Mississippi, USA**
0700-0800 Hrs:	Registration & Breakfast
0800-1000 Hrs:	Problem Solving in Nerve Conduction Studies: A Live Interactive Demonstration
1000-1030 Hrs:	Coffee Break
1030-1130 Hrs:	Myopathies and Motor Neuron Diseases
1130-1230 Hrs:	Disorders of Neuromuscular Transmission
1230-1330 Hrs:	Lunch
1330-1430 Hrs:	Disorders of nerve and muscle membrane hyperexcitability
1430-1530 Hrs:	Applying electrodiagnostic studies for intraoperative monitoring of nerve function
1530-1600 Hrs:	Tea Break
1600-1800 Hrs:	Practical Skills Workshop ♦ Repetitive nerve stimulation studies ♦ Blink Reflex, H Reflex, Silent Period ♦ Problem Solving ♦ EMG Pattern Recognition- Video
1800 Hrs:	Close

PROF. M.B.M. SUNDARAM

Prof. M. B. M. Sundaram, M.R.C.P. (London), F.R.C.P.(Canada), Diplomate, American Board of Neurology and Diplomate, American Board of EEG, is the Professor of Neurology and Director of the Neurophysiology Laboratory and Epilepsy Unit at the University of Missssissippi Medical Center, Jackson, Mississippi, USA.

After medical graduation from Madras University, Prof. Sundaram did a period as Senior House Officer with Prof. K. Srinivas at the K. Gopalakrishna Dept. of Neurology, VHS Medical Center in 1973, where Prof. Srinivas, in his inimitable manner, guided him on the possibilities of further training in neurology in the West.

Prof. Sundaram did his postgraduate training in internal medicine at Newcastle General Hospital, UK and later was Registrar in Neurology at the General Infirmary, Leeds.While in Canada, he consolidated his area of interest as a clinical neurologist with special interest in epilepsy and the research aspects of EEG. This involved residencies at University Hospital, Saskatoon and at Regina in Canada and an EEG – Epilepsy fellowship under Dr. W.T. Blume, at the University of Western Ontario in Canada. He has several significant peer-reviewed publications to his credit. He has received several awards, including 'Best Teacher Award' in Neurology for 1995-96, 2001-2002, 2003-2004, and 'Among the 'Best Doctors in America'– year 2000 onwards.

Yet another 'Made in India' and exported to the USA, Prof. M. B. M. Sundaram brings to us the state of art EEG technology from the USA, with, as clinician, the added value of its clinical correlation.

EEG WORKSHOP

Prof. M.B.M. Sundaram

Objectives:

1) Assist in obtaining competency in recognizing various normal and abnormal EEG patterns in children and adults.
2) Learn the clinical uses and limitations of EEG.
3) Learn how to write an EEG report.
4) Review the role of long-term video-EEG monitoring in epilepsy.
5) Discuss the role of EEG in epilepsy surgery.
6) Become familiar with recommended guidelines for EEG technology to ensure the recordings are of highest quality.

BASIC EEG

1.0 Normal EEG and rhythms.

1.1 Alpha activity:

Alpha rhythm is the hallmark of normal EEG in healthy awake adults and older children. Alpha activity consists of 8 to 13 Hz frequencies, expressed in the posterior head regions. Alpha activity attenuates with eye opening and reappears with eye closure. Up to 50% amplitude difference between left and right is considered normal. Persistent frequency difference of 1 Hz or more between sides is considered abnormal.

Alpha activity should appear with eye closure by age 2 or 3. Even in the very elderly, alpha frequencies should not fall below 8 Hz when fully awake in normal individuals.

Sometimes, 2 alpha waves fuse and the rhythm may fall to the 4 to 6 Hz range. This rhythmic posterior rhythm is referred to as "slow alpha variant". These records do contain, at other times, regular alpha frequencies.

If the individual is drowsy, alpha rhythm may appear with eye opening and this is called "paradoxical alpha".

Failure of alpha blocking with eye opening is called "Bancaud's phenomena". This is often due to a pathology in the occipital/parietal regions.

Rhythmic activation of thalamic pacemaker cells in the specific nuclei driving the cortical neurons, followed by inhibition, is thought to produce the rhythmicity of alpha activity. Some researchers believe that the cortical association fibers are responsible for the rhythmicity.

Mu rhythm may appear somewhat similar to alpha rhythm, but differs in that it is seen in the central regions, is more sharply contoured and disappears to finger movements rather than to eye opening.

1.2 Beta rhythm:

Beta activity ranges from 14 to 30 Hz. Normal individuals have some amount of intermittent beta activity, often in the anterior head regions. The quantity of beta increases during drowsiness and sleep, especially in children. Excessive, persistent beta activity could result from medications such as diazepam, lorazepam, midazolam and phenobarbital. Pathologically excessive and prominent beta activity without medications may also be seen in some children with severe mental retardation and lissencephaly.

1.3 Sleep:

Sleep is divided into 2 stages - non-REM and REM (rapid eye movement). Normal healthy adults spend approximately 7-8 hours in sleep with 4-6 cycles of non-REM sleep, and REM sleep.

Non-REM sleep consists of stages I-IV. EEG during stage I non-REM sleep shows attenuation of alpha activity, appearance of theta requencies, slow eye movements and vertex waves. Note that vertex waves may be asymmetrical, and sometimes, sharply contoured.

Stage II non-REM sleep is characterized by the appearance of sleep spindles in the central and parietal regions. The frequency of sleep spindles is 10-14 Hz. Sleep spindles may be asymmetrical and maybe associated with vertex eaves ("K-complexes").

The quantity of spindles decreases in stage III non-REM sleep.

Stage III and IV non-REM sleep contain abundant delta activity.

REM sleep usually follows a cycle of non-REM sleep. The duration of REM sleep increases in later sleep cycles, often in the early morning hours. EEG in REM sleep is characterized by lower amplitude but faster background frequencies in the theta and alpha range. Axial EMG activity decreases during REM sleep. Heart rate and respiration tend to be irregular. In normal healthy adults, REM sleep comprises approximately 25% of total sleep time. However, full term newborns spend 50% of their sleep time in REM (active) sleep. In normal adults, REM appears 80-90 minutes after sleep onset. The REM latency is considerably shortened in patients with narcolepsy and in those with REM sleep deprivation due to prolonged lack of sleep, sleep apnea or REM-suppressing medications such amphetamines and phenobarbital.

1.4 <u>Hyperventilation</u>:

Hyperventilation (HV) is routinely done in children and adults as an activation procedure for detecting generalized epileptiform discharges and absence seizures. Normal response during HV consists of diffuse theta and delta activity. Delta activity may be seena s frontally predominant rhythmic bursts (FIRDA). Such delta activation in children may be prominent in the occipital regions rather than frontal areas (OIRDA). Exaggerated delta activity has been described in subjects with tendency for hypoglycemia. Note that HV may not elicit any slow wave build up at all, especially in uncooperative subjects.

Normal changes described above are thought to be due to cerebral vasoconstriction mediated by hypocapnia. This view, however, remains somewhat controversial.

HV is contraindicated in patients with tendency for severe vasoconstriction and these conditions include sickle cell trait or disease, subarachnoid hemorrhage and malignant hypertension.

1.5 <u>Photic Stimulation</u>:

Photic Stimulation (PS) is done using light flashes with frequencies of 1 Hz to 30 Hz. Normal response consists of occipital, rhythmic, sharply contoured activity referred to as "photic driving".

Photic driving response may be symmetrical or asymmetrical or sometimes prominent, mimicking occipital spikes. Asymmetrical photic driving in the absence of other EEG abnormalities in considered non-pathological.

2.0 <u>Pathological slow waves.</u>

Theta (4-7 Hz) and delta (1-3 Hz) activity could be focal or generalized. Pathological slow waves should be carefully differentiated from the physiological slow wave activity listed below. Physiological posterior delta of youth is intermittent and reacts to alerting and eye opening. Temporal delta in normal awake elderly subjects, also referred to as "delta of old age" should occupy approximately 1% or less of the awake EEG.

2.1 Diffuse theta:

Several subacute or chronic conditions produce diffuse theta activity and these include toxic/metabolic/septic encephalopathy, Alzheimer's disease, static encephalopathy with mental retardation, chronic CNS infections etc.

2.2 Focal theta:

Focal or hemisphere disorders such as trauma, stroke, tumors (especially more benign tumors), underlying seizure focus, may result in focal theta activity.

2.3 Diffuse polymorphic delta:

This is seen in toxic/metabolic/septic encephalopathy, viral encephalitis, bacterial meningitis, diffuse trauma, diffuse dementias, subarachnoid hemorrhage, leukodystrophies, meningeal cancer, lupus cerebritis and neuroleptic malignant syndrome. Such diffuse delta activity results from bilateral subcortical white matter or thalamic dysfunction with or without cerebral grey matter dysfunction.

Polymorphic persistent unreactive delta in the posterior head region in children may be from recent seizures, posterior fossa tumors, leukodystrophies, head injury andhypothermia.

Unilateral or focal polymorphic delta is seen in hemispheric stroke, tumor, abscess and trauma. This results from underlying white matter or thalamic dysfunction.

2.4 Rhythmic delta:

Rhythmic bursts of delta, if frontally predominant, are referred to as "FIRDA" (frontal intermittent rhythmic delta activity). Such activity may be expressed in the posterior head region in children and is referred to as "OIRDA". Causes of FIRDA or OIRDA include hyperventilation, drowsiness, metabolic/septic/toxic encephalopathy, increased intracranial pressure, medial thalamic lesions and postictal state. Rhythmic paroxysmal delta activity denotes diffuse cerebral or diencephalic grey matter dysfunction. Occasionally, rhythmic delta bursts are seen intermittently in the temporal region ("TIRDA") and this signifies an underlying seizure focus.

3.0 Normal Variants.

These are sharply contoured isolated or sequential waveforms which mimic epileptiform discharges or electrographic seizures. Their unique morphology, distribution and lack of evolution should help in recognizing them. These waveforms are considered to be of no clinical significance.

3.1 Posterior:

Slow alpha variant:

This rhythmic "fused" activity is often half of alpha, i.e. 4 to 6 Hz and mixed with regular alpha activity. Note that rhythmic theta of drowsiness will be more diffusely distributed whereas slow alpha is confined to the posterior head region - especially occipital. Also, theta of drowsiness will be associated with other drowsy patterns such as slow eye movements, POSTS and vertex waves.

Lambda:

These sharply contoured mono- or diphasic potentials are seen in the occipital regions, often when looking or scanning at objects of interest. This activity is electro-positive in the occipital regions. Lambda activity needs to be differentiated from occipital spikes which are considerably narrower and often associated with slow waves. Occipital spikes are usually electro-negative in polarity and will persist during drowsiness and sleep. They are seen even when the eyes are closed.

POSTS:

Positive Occipital Sharp Transients of Sleep are similar in morphology and polarity to lambda, but occur in drowsiness and early non-REM sleep. POSTS are sometimes referred to as "lambdoid" rhythm. POSTS may be isolated or sometimes recur in close succession. These also need to be differentiated from the occipital spikes described above.

3.2 Central:

Mu:

Mu is characterized by sharply contoured, rhythmic activity in alpha frequencies in the central and parietal regions. Mu is often unilateral but may sometimes be simultaneous bilateral. Movement of ipsi- or contralateral fingers (or even thinking of movements) often attenuates mu activity. Mu occurs when awake and may be best seen when the eyes are open, thereby blocking posteriorly distributed alpha activity. Mu becomes prominent when there is an underlying skull defect.

Breach Rhythm:

Breach rhythm is seen when there is underlying skull defect (burr hole or craniotomy). Breach rhythm is a mixture of prominent mu activity, sharply contoured fast theta frequencies and beta activity. Breach rhythm is seen during wakefulness and sleep.

3.3 **Temporal**:

RMTD:

Rhythmic Mid Temporal Discharges are also referred to as "psychomotor variant". RMTD is seen in the temporal regions during drowsiness and early sleep in older children and young adults. This sharply contoured rhythmic activity is in the theta frequencies and often "notched". These discharges may be seen in either temporal region or at times simultaneously in both temporal areas and their duration is usually several seconds. RMTD needs to be differentiated from temporal electrographic seizures which often last last longer and undergo changes in the frequency, amplitude and distribution; whereas RMTD remains monomorphic. Recordings with temporal electrographic seizures also show focal epileptiform discharges at other times.

Fourteen & six positive spikes:

This pattern consists of low amplitude comb-like rhythm in the temporal and occipital regions during drowsiness and sleep in older children, adolescents and young adults. This activity may be unilateral or simultaneous bilateral and monomorphic without metamorphic evolution. These discharges are often approximately 14 Hz in frequency. 6 Hz variant is uncommon.

Wicket spikes:

Wicket spikes are sharply contoured activity occurring in the temporal regions or less often in other regions during drowsiness of older individuals and are not associated with slow waves. These discharges may be unilateral or bilateral and do not clearly stand out from the ongoing background activity. Wicket spikes simply represent a combination of different frequencies producing sharply contoured peak to the waveform. Note that temporal spikes are much narrower, associated with slow waves and often have a clear field of distribution.

BETS/SSS:

Benign epileptiform transients of sleep, also referred to as small sharp spikes, are seen during drowsiness and early non-REM sleep in adults. These discharges, as the name implies are low in amplitude (often less than 50 µV) and brief in duration (less than 50 ms). The morphology often resembles the letter “N”. These discharges are often diffusely seen in the frontal and temporal regions uni- or bilaterally and lack a clear field of distribution, which characterizes pathological focal epileptiform discharges. If unilateral, BETS often shift from side to side.

3.4 Miscellaneous:

SREDA:

Subclinical rhythmic electrographic discharge of older adults are monomorphic and often in the theta range; these discharges may be somewhat sharply contoured but do not undergo metamorphic evolution. SREDA is bilaterally distributed in the parietal, central and temporal regions and may last from several seconds up to 1 or 2 minutes. This rhythm occurs during wakefulness and is not associated with any impairment of response or any other signs of seizures.

4.0 Focal epileptiform discharges.

4.1 Interictal epileptiform discharges:

Epileptiform discharges (ED) are the hallmark of epilepsy. They stand out from the ongoing background activity, are quite sharp in contour, if focal, have a defined field and are often followed by slow waves. If the duration of ED is less than 70 msec, the discharge is referred to as a spike; if the duration is between 70 and 200 msec, the term “sharp wave” is used. The clinical significance of spikes and sharp waves is the same.

Focal ED in scalp recordings are usually electro negative, but surface positive spikes may be seen in patients with skull defect, in neonatal recordings, and as a part of dipole activity in recordings with rolandic spikes.

Although the presence of focal ED generally support partial onset seizure disorder, approximately 2% of EEGs in subjects without epilepsy may show incidental ED. These incidental ED include: rolandic spikes, occipital spikes, generalized 6 Hz rapid spike/wave discharges, generalized ED during photic stimulation. The frequency of incidental spikes may reach 8-9% in children, especially when prolonged recordings are done. Hence, the physician should carefully consider other clinical information when interpreting the clinical significance of spikes.

Approximately 20% of patients with temporal lobe epilepsy may show ED in either temporal region, yet the clinical seizures still originate from one side. This is thought to be due to kindling of the other side. Such patients will require continuous monitoring when surgery is contemplated, to establish the exact side of clinical seizures.

Sleep recordings and sleep deprivation are useful for activation of focal as well as generalized ED. The quantity of ED increases in non-REM sleep and returns to awake quantities in REM sleep. The field of focal ED tends to expand in non-REM sleep.

4.2 Focal electrographic seizures:

Repeated spikes or sharp waves or any sinusoidal rhythm lasting up to several seconds are referred to as electro-graphic or subclinical seizures. The amplitude and frequency often change during the electro-graphic seizure. These changes are referred to as "evolution" of spikes.

5.0 Generalized epileptiform discharges.

Generalized epileptiform discharges (GED) are often described according to the most common frequency.

5.1 3 Hz GED:

3 Hz GED is the characteristic inter ictal finding in subjects with typical absence seizures. Some impairment of clinical response occurs if 3 Hz GED lasts more than one second. During non-REM sleep, the frequency of 3 Hz GED often slows down and the spikes may fragment or become somewhat periodic.

5.2 Slow spike and wave:

GEDs with a frequency 2½ Hz or less are common in patients with atypical absence, atonic seizures and tonic/clonic seizures, especially in patients with Lennox-Gastaut syndrome. These discharges are often referred to as generalized slow spike and wave discharges or generalized sharp and slow wave complexes.

5.3 Generalized polyspikes:

Typical interictal finding in patients with myoclonic seizures is generalized polyspike and wave discharges or slow spike wave discharges.

5.4 Spike and wave with PS:

Generalized spike and wave discharges may occur in patients with generalized epilepsies. These discharges, if confined to the duration of PS (i.e. not outlasting PS), may be an incidental finding in subjects without epilepsy. Rhythmic frontal EMG discharges time-locked with photic stimuli are referred to as "photomyogenic response", and need to be carefully differentiated from spikes.

6.0 Ictal Rhythms and Seizures.

6.1 Generalized Seizures:

Absence:

Typical absence seizures are associated with 3 Hz generalized spike wave discharges. These seizures usually last up to 10 or 15 seconds although briefer episodes are not uncommon. The frequency of spike wave discharges may slow down below 3 Hz during the later part of the seizure.

Atypical absence seizures are associated with generalized spike wave discharges slower than 3 Hz and last longer than typical absence attacks. These seizures occur in mentally retarded subjects and are more difficult to treat than typical absence seizures.

Note that interictal generalized "slow" spike wave discharges in Lennox Gastaut syndrome may become frequent or even continuous in sleep without overt clinical signs. Such continuous spike wave discharges in sleep may also occur in an uncommon syndrome referred to as "electrical status epilepticus during sleep".

Tonic Clonic Seizures:

The tonic phase is characterized by repetitive generalized spikes with fast frequencies and the clonic phase by interrupted spike wave activity. Sometimes, rhythmic diffuse activity precedes the tonic phase and this is referred to as "recruiting rhythm". Postictal recording after a tonic clonic seizure often shows diffuse delta, sometimes preceded by generalized suppression. The duration of these postictal changes is variable and may be a few seconds to several minutes. Note that postictal delta in children is often prominent in the posterior head region and may last several days.

Myoclonic Seizures:

Myoclonic seizures are associated with brief generalized polyspikes or spike wave discharges.

Atonic Seizures:
These often show brief generalized spike wave discharges of variable frequency. Sometimes, these seizures are not associated with any scalp EEG changes.

Tonic Seizures:
These seizures typically show diffuse attenuation with superimposed rhythmic low voltage beta-like activity. Sometimes, brief generalized spike wave discharges precede the attenuation. Also note that ictal EEG during a tonic seizure may be difficult to interpret because of prominent EMG activity.

6.2 Partial Seizures:

Ictal changes during partial seizures are often metamorphic, i.e. they show two or more distinct phases. This is also referred to as "evolution" of seizures. The ictal patterns are rhythmic and may consist of spikes, sharp waves or sinusoidal waveforms. The amplitude and frequency typically change during the evolution of the seizure. The ictal discharge also gradually spreads involving other areas. The initial frequency of partial onset seizures in scalp recordings may be in the alpha, theta, delta or beta range. Frontal seizures tend to start with the beta or alpha range, whereas temporal seizures tend to start in the theta or alpha range. Focal attenuation is also seen at the onset of some partial seizures. During the post ictal period, EEG may show lateralized or localized delta or suppression.

Note that the scalp EEG may not show any change during some simple partial seizures, especially in those with exclusive sensory symptoms. Also, scalp EEG occasionally may not show any ictal change during frontal seizures with deep focus.

7.0 Periodic Lateralized Epileptiform Discharges.

7.1 PLEDs:

PLEDs show periodic sharp wave discharges in one hemisphere or in a region of one hemisphere. Underlying cerebral lesion(s) are common. Patient are often unresponsive or semi-responsive.

Causes of PLEDs include: stroke, tumor, hematoma, abscess, herpes simplex encephalitis and LaCrosse encephalitis. PLEDs may also be seen in patients with metabolic encephalopathy due to hypoxia or hypoglycemia, as well as in those patients with recent partial seizures and mitochondrial encephalopathy.

Prolonged EEG monitoring in patients with PLEDs may reveal frequent electrographic seizures which may explain the mental status change.

Simple PLEDs without electrographic seizures in patients with already improving level of consciousness is considered "inter ictal" and the patient may not require additional anticonvulsants. However, if the patient with PLEDs remains obtunded or if the level of consciousness deteriorates without any other obvious cause, a prolonged recording should be considered, looking for electrographic/subclinical seizures.

Disconnection between grey and white matter in experimental animal models, results in periodic discharges.

7.2 PLEDs plus:

The morphology of the periodic complexes in PLEDs plus pattern is more complex than simple PLEDs. Sharp waves of PLEDs plus are followed by brief rhythmic fast rhythm.

PLEDs plus pattern is considered more "epileptogenic" and often follows or precedes clinical seizure.

7.3 BiPLEDs:

BiPLEDs affect each hemisphere independently. In other words, periodic sharp waves occur at different times in either hemisphere.

Causes of BiPLEDs include: hypoxia, herpes simplex encephalitis and independent cerebral pathologies.

8.0 Artifacts.

8.1 Physiological artifacts:

Eye movements:

Anterior cornea is positively charged and moves towards Fp1/Fp2 or F7/F8 during eye blinks and lateral eye movements respectively. Eye movement artifacts are produced when these electrodes pick up the positive potential from the cornea. Note that eye movements are out of phase in the channels monitoring eye movements whereas frontal lobe activity will be "in phase" in the channels monitoring eye movements.

Rectus spikes:

Rectus spikes resulting from brief bursts of EMG activity from one of the rectus muscles may be seen preceding eye movements. Fronto-polar electrodes, F7/F8 are commonly affected. Rectus spikes need to be carefully differentiated from epileptiform discharges.

Glossokinetic artifact:

Movements of the tongue toward the recording electrodes produce a unique artifact consisting of diffuse or fronto-temporal delta with superimposed EMG burst from pharyngeal and scalp muscles. The slow delta potential results from negative activity from the tip of the tongue contaminating the scalp electrodes.

Chewing and sucking artifacts:

These are characterized by rhythmic bursts of EMG activity.

Pulse artifact:

Pulse artifact results when a recording electrode is on or near a pulsating scalp artery. This rhythmic activity corresponds to the pulse rate and can be easily diagnosed with the EKG channel.

Respiration artifacts:

Electrode or lead movements sometimes produce a rhythmic artifact with the rate corresponding to respiration. This is not uncommon during hyperventilation. A similar artifact may be seen in the intensive care setting resulting from rhythmic vibrations in respirator equipment.

Sweat artifact:

This is characterized by very slow undulating waveforms affecting several electrodes. This is usually seen in CCU/ICU units. The frequency of sweat artifact is considerably slower than physiological delta activity. Cerebral potentials may be suppressed if perspiration is profuse and links two or more electrodes (creating the so-called “salt bridging”).

Tremor artifact:

This may affect one or several scalp electrodes and may be seen in patients with essential tremor and Parkinson’s disease. The rhythmic artifact is at the same rate as the tremor. A tremor artifact is often associated with rhythmic bursts of EMG as well.

Patting artifact:

This rhythmic artifact is seen in newborns and infants, and may affect several electrodes.

8.2 Non-physiological artifacts:

Electrode artifacts:

Poor electrode contact may produce "electrode pops" due to disturbance of the electronic double layer. High impedence and impedence mismatch between electrodes will result in 60 Hz artifact. 60 Hz artifact may also be seen in "electrical hostile environment" where other equipment are near the EEG machine as in ICU and operating room.

Static artifact:

Any disturbance near the EEG recording machine and patient bed in the ICU and OR may produce a static discharge affecting the EEG electrodes. Sometimes, rhythmic static artifact may be caused by intravenous drips. Static artifacts may also result from cell phones and lightning.

9.0 Miscellaneous.

9.1 Triphasic waves:

Triphasic waves (TW) are seen in metabolic and septic encephalopathy as well as in Alzheimer's disease. TW typically have initial surface negative phase followed by a prominent surface positive second phase and then a surface negative third phase. The amplitude of the second phase often exceeds the other two phases. The first phase may be sharply contoured and these TW need to be carefully differentiated from epileptiform sharp and slow wave complexes. TW are often maximally expressed in the frontal regions but may be prominent in the occipital region. The peak of the prominent surface positive phase two is seen slightly earlier in the anterior channels compared to the more posterior regions ("frontal-occipital delay"). TW sometimes are periodic. TW are infrequent in Alzheimer's disease but are often abundant in metabolic encephalopathy. TW are rare in children.

9.2 Periodic Epileptiform Discharges:

Periodic Epileptiform Discharges (PEDs). These are sharply contoured periodic discharges occurring simultaneously in both hemispheres. These need to be differentiated from PLEDs which are lateralized to one region or one hemisphere. Causes of PEDs include: hypoxic encephalopathy, drug overdose especially with sedatives, herpes simplex encephalitis, Creutzfeldt-Jacob disease (CJD) and subacute sclerosing panencephalitis (SSPE). The periodic complexes in CJD tend to be lower in amplitude, occurring every 1-2 seconds whereas the complexes in SSPE tend to be higher in amplitude, more complex in morphology and recur only every several seconds. PEDs may mimic BS pattern.

9.3 Burst-suppression:

Burst suppression pattern (BS) typically shows high voltage bursts of theta, delta and spikes lasting a few seconds separated by diffuse flat periods lasting many seconds. Causes of BS include hypoxic encephalopathy, severe head injury, sedative drug overdose or iatrogenic medication administration (midazolam, propofol). Spontaneous BS pattern without medication use or overdose generally carries poor prognosis.

Brief periods of diffuse attenuation with more continuous activity are referred to as "electro decremental events"; these are not uncommon in patients with severe encephalopathy and those receiving sedative medications.

9.4 Alpha Coma:

Alpha coma pattern consists of diffuse, anteriorly prominent, unreactive alpha activity in a comatose pattern. This pattern is seen in severe hypoxia, sedative overdose and head injury. This EEG pattern may evolve into other patterns and generally carries a poor prognosis.

9.5 Other coma patterns:

Other patterns seen in comatose patients consists of monomorphic diffuse theta activity and is referred to as "theta coma"; "spindle coma" pattern consists of prominent sleep spindles and vertex waves in comatose patients. Theta coma and spindle coma patterns may be seen in patients with hypoxia, head injury or sedative overdose and generally carry a better prognosis than alpha coma.

10.0 Technical Aspects.

10.1 **The international 10-20 system** of electrode placement ensures uniformity and is recommended by national and international EEG societies.

10.2 Additional electrodes:

We recommend routine use of surface sphenoidal electrodes. Additional electrodes may be necessary as the clinical situation dictates - SO_1 and SO_2, F_1 and F_2, CP_z, etc.

10.3 Electrodes:

Gold-plated silver cup electrodes are commonly used. Electrode impedence should be reduced to < 5 K ohms using a commercially available abrasive skin prepping gel; low impedence ensures clean, artifact free recordings. Good contact is achieved by using collodion which can be dried by compressed air with insertion of an electrode gel to provide adequate conduction. Adhesive EEG conducting paste is also commercially available and provides good electrode contact.

10.4 Activation procedures:

Hyperventilation (HV) should be for at least 3 minutes. Longer periods may be necessary if one suspects absence seizures. Contra-indications for HV include potential vasospastic disorders such as sickle cell disease, subarachnoid hemorrhage and malignant hypertension.

Photic stimulation is done using frequencies upto 30 Hz. The strobe light is kept at a distance of 20-30 cm from the eyes. Stimulation is started with eyes open and followed by eye closure midway during a given frequency.

10.5 Recording duration is at least 20 minutes. Sleep deprived recording should be for approximately 60 minutes.

11.0 Uses and limitations of EEG.

11.1 Uses:

EEG is useful in a) supporting the clinical diagnosis of seizure disorder, b) classification of seizures and epileptic syndromes, c) predicting recurrence following first seizure and during anticonvulsant withdrawal, d) the diagnosis of non-convulsive status epilepticus, e) helping with prognosis in coma and f) diagnosing metabolic/septic/toxic encephalopathies, various types of dementias and infections such as herpes simplex encephalitis, bacterial meningitis and SSPE.

11.2 Limitations:

Note that the first of random EEG in patients with epilepsy may not show epileptiform discharges in approximately 50%. The yield however is higher in absence epilepsy and if the EEG is done within 1-2 days of a seizure. Serial EEGs improve the yield upto 90% or so. If repeated EEGs remain negative, - including sleep-deprived recordings -, continuous EEG monitoring should be considered for the diagnosis.

Approximately 1-2% of healthy adults without epilepsy may show epileptiform discharges in their EEG. The frequency of “false positive” recordings may be higher, - as high as 8-9% in children, especially if prolonged recordings are performed. These incidental epileptiform discharges include rolandic spikes, occipital spikes and generalized spikes, especially if occurring during photic stimulation and 4-5 Hz generalized spikes.

12.0 Pattern Recognition and how to write a report.

12.1 Pattern Recognition:

This is made easy by following a systematic approach the author refers to as:

"The rule of W":

Where is the finding? - occipital, anterior head, central, temporal, hemisphere or diffuse.

When? - is the patient awake or drowsy, are eyes open or closed.

What morphology? - is the frequency in alpha, theta or delta range, is the finding isolated or sequential (rhythmic), is there anything unique about the morphology.

12.2 Report:

A typical report should contain patient identification information, indication, the body of the report describing major frequencies/findings, and interpretation explaining the clinical significance of the findings. Additional recommendations should also be mentioned such as repeating with sleep deprivation or obtaining long-term EEG monitoring.

PEDIATRIC EEG

1.1 Evolution of EEG through childhood:

EEG during infancy contains abundant theta and delta, even when awake. In early infancy the quantity of theta activity should exceed the amount of delta in awake recordings. Central rhythm in infancy frequently reaches 7 or 8 Hz. The background activity and overall frequencies gradually increase with age. At the same time, the quantity of delta decreases.

Rhythmic posterior activity of 4 Hz reacting to eye closure is seen by age 6 months; this reaches 6 Hz by 12 months. By age 2 or 3, rhythmic alpha activity should appear in the occipital regions. In older children and during the teenage years, diphasic posterior delta rhythm with super-added alpha frequencies is a common finding and this is referred to as "posterior delta of youth"; this normal delta rhythm disappears with eye opening and thus differs from pathological delta activity which persists with eye opening or alerting. The EEG assumes adult patterns in most normal subjects by age 14 to 18 years of age.

Normal sleep potentials such as vertex waves and sleep spindles begin to appear by 3-4 months, following post term in healthy infants. These sleep potentials are often asymmetric but should be seen in either hemisphere. Vertex waves and sleep spindles, if consistently suppressed in one hemisphere, is considered abnormal. Also note, that vertex waves in children may be sharply contoured but they lack the slow wave activity associated with pathological midline sagittal spikes. Moreover, sagittal spikes are present also during wakefulness.

1.2 <u>Unique patterns</u>:

A common pattern seen in drowsiness and during arousal in late infancy and early childhood is referred to as hypnagogic hypersynchrony. This pattern consists of high voltage bursts of rhythmic theta and sometimes delta. These bursts sometimes contain sharply contoured transients and one needs to be careful to avoid over-interpretation of these discharges as epileptiform activity.

Another normal finding during sleep in early childhood is the "frequency amplitude gradient". This pattern occurs during non-REM sleep and consists of prominent runs of high voltage delta activity in the posterior head regions while lower and faster frequencies are seen anteriorly. Isolated diphasic, high amplitude waveforms are also common in the occipital regions during sleep in infants and these are referred to as "cone waves".

1.3 <u>EEG in the full term neonate</u>:

EEG in the full term neonate (38 to 42 weeks GA) is unique in many respects. Awake-sleep cycles are short and these infants usually fall asleep in REM sleep. Moreover, REM sleep occupies approximately 50% of total sleep. Non-REM sleep -also referred to as quiet sleep- may consist of tracé alternant pattern or irregular high voltage delta activity. Some amount of frontal, temporal and central sharp waves are normal up to 2 months of age, especially in quiet sleep. Presence of premature patterns, excess pathological epileptiform discharges, lack of sleep-wake cycles, electro-positive central or temporal sharp waves, diffuse suppression, persistent asymmetry, burst-suppression patterns and neonatal seizures represent commonly seen abnormalities at this age.

1.4 <u>EEG in premature infants</u>:

In the very premature newborns, aged 26-29 weeks, EEG is discontinuous with flat periods lasting up to 30 seconds (tracé discontinu) and no cyclical awake-sleep organization is seen. At this age, EEG during active periods, shows diffuse, predominantly theta frequencies between flat periods. The EEG becomes more continuous with development of sleep-wake cycles by 30 weeks GA. Multifocal sharp transients and rhythmic temporal theta bursts (“saw tooth waves”) are common at 30-32 weeks GA. Delta brushes are also common at 30-34 weeks GA. State differentiation is complete by 35-37 weeks GA. Symmetry between hemispheres reaches almost 100% by 38 weeks.

1.5 <u>Neonatal seizures</u>:

Neonatal seizures consist of rhythmic spikes or sinusoidal waves lasting several seconds with or without clinical changes. These discharges may be of any frequency and often involve multiple foci.

<u>ADVANCED EEG</u>

Long term Video-EEG monitoring (LTM) is now widely available for differentiating seizures from non-epileptic events such as pseudoseizures, movement disorders, sleep disorders and syncope. LTM may be done on an outpatient basis or as an inpatient. Amubulatory monitoring is subject to several artifacts, and most available systems do not have video capability.

For surgical localization, one needs to record several seizures to show consistency of focus; the number of seizures one needs to record depends on interictal spike distribution, MRI/PET findings and clinical features. Video analysis of seizures is important when surgery is under consideration. Also, postictal slowing, when present, provides useful lateralizing or even localizing information. When scalp recordings fail to reveal a clearcut focus and if the clinical features suggest a single focus, subdural or depth recordings are performed. Subdural electrodes provide the additional advantage of being able to perform language and motor mapping.

Electro-corticography assists in fine tuning the area of resection, although one should avoid “spike chasing”.

Commonly performed surgical procedures include temporal lobectomy, extratemporal corticectomy, corpus callosum section, multiple subpial resection and hemispherectomy.

Vagus nerve stimulator is used in those who are not otherwise surgical candidates.

PROF. VETTA VEDANARAYANAN

Prof. Vetta Vedanarayanan M.D., F.R.C.P.(Canada) and Diplomate, American Board of Neurology with Special Competence in Child Neurology, is the Professor of Pediatric Neurology at University of Mississippi Medical Center, Jackson, Mississippi, USA.

After medical graduation from JIPMER, Pondicherry, Prof. Vedanarayanan joined AIIMS, Delhi for an MD in Pediatrics before leaving for the USA. He did his residency in pediatrics at the New York Medical College, and channelised his energies towards the specialty areas of pediatric neurology at Duke University Medical Center, and later at Johns Hopkins, USA, where he received a fellowship in neuromuscular diseases.

Prof. Vedanarayanan has several peer-reviewed publications to his credit and has received awards and honours for his significant contribution to the neurosciences. His remarkable ability to integrate the clinical and electrophysiological aspects of neuromuscular disorders and diseases is bound to hold our individual attention for the next couple of days.

EMG Workshop

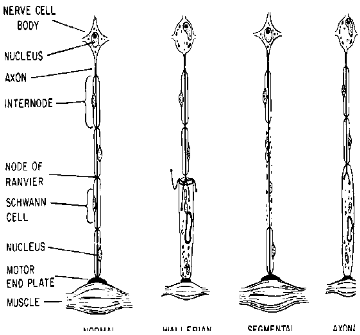
Prof. V. Vedanarayanan

Polyneuropathies, Mononeuropathy Multiplex and Plexopathies

Polyneuropathies

- Generalized involvement of nerves.
- Longest ones are involved first
- Classified based on:

  Clinical presentation: sensory, sensorimotor, autonomic
  Pathological lesion: axonal degeneration, demyelinating or mixed lesions
- Etiology varied: Metabolic diseases, Inherited, collagen vascular disorders, infections and parainfectious, paraneoplastic, medications, environmental factors – heavy metals, toxins



Pathological Lesions

axonal degeneration

demyelination

mixed lesions

Axonal Degeneration

CAUSES:

Diabetes mellitus
Uremia
Porphyria
Vitamin deficiency: B12, folate, vitamin E, thiamine
Infections: HIV, diptheria
Collagen vascular: SLE, Sjogrens syndrome, scleroderma, small vessel vasculitis

Medications: chemotherapy, isoniazid, hydrochlorthiazide, DDI, suramin, taxol, nitrofurantoin, amiodrone
Heavy metals
Paraneoplastic
Solvents, adhesives

INHERITED – HSMN type 2, neuraxonal dystrophy, giant axonal neuropathy

AXONAL POLYNEUROPATHY

- Sensory – motor polyneuropathy
- Sensory polyneuropathy
- Autonomic polyneuropathy (may be associated with dysfunction of small nerve fibers)
- Pure motor neuropathy - rare

Sensory Neuropathies

- Large Fiber:
  Vitamin deficiency- B12, folate
  Diabetes mellitus
  Paraneoplastic associated with anti Hu antibodies
  Sjogrens syndrome
  Syphillis – tabes dorsalis
  Leprosy
  Hereditary sensory polyneuropathy
  Idiopathic
- Small fiber
  Diabetes mellitus
  Amyloidosis
  Heavy metals – thallium, mercury, antimony
  Small vessel vasculitis
  Nutritional – beri beri

AXONAL NEUROPATHY

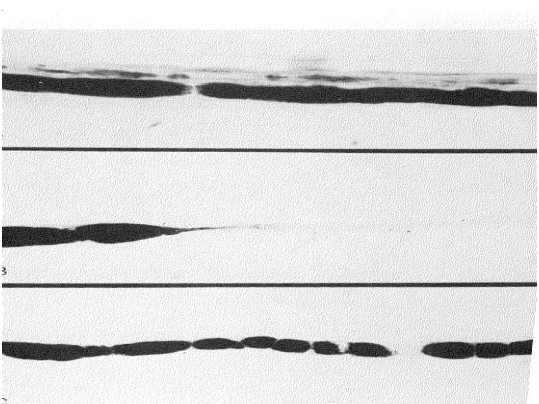
- Sensory latency usually normal.
- SNAP amplitude reduced or absent in a distal to proximal gradient
- Distal motor latency usually normal.
- Motor conduction velocity normal. The velocity may be slower by less than 25 % even when CMAP is reduced below 10%.
- CMAP reduced.

- F wave latency normal.
- H reflex may be absent.
- Needle EMG - denervation, acute, chronic or mixed with a distal to proximal gradient
- Entrapment neuropathies and radiculopathies can cause more severe and asymmetric patterns due to phenomenon of double crush.

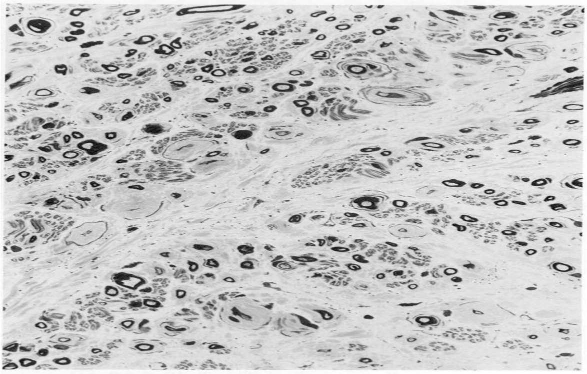
Sensory Polyneuropathy

- Sensory nerve conduction studies are abnormal, pattern consistent with axonal degeneration. The motor nerve fibers are relatively spared !!!
- In small fiber polyneuropathy there is paucity of abnormalities in routine nerve conduction studies and needle EMG examination.
- The autonomic nerve function may be abnormal.
- Quantitative sweat measurement after an axonal reflex will be abnormal and show a distal to proximal gradient.
- Measurement of epidermal nerve fiber density on skin biopsies are useful when it can be performed.
- MANY TIMES THE DIAGNOSIS OF SMALL FIBER POLYNEUROPATHY IS MADE ON THE BASIS OF OTHER CLINICAL DATA.

Demyelination and remyelination



Chronic demyelination and remyelination



ABNORMAL FINDINGS - DEMYELINATING NEUROPATHY

- Latency of sensory response is prolonged
- Conduction velocity is slowed,
- SNAP amplitude is reduced.
- Distal motor latency is prolonged.
- CMAP is reduced.
- **<u>Conduction block.</u>**
- F wave latency prolonged or absent.
- H reflex absent.
- Needle EMG – Reduced motor unit recruitment, normal motor unit morphology and sometimes increase in polyphasia

Conduction Block

- Reduction in the compound motor/ sensory amplitude with proximal site of stimulation compared to size of response from stimulation close to the recording electrodes.
- Size of reduction variable -30 to 50 %. Proximal / distal
- Represents partial or total failure of conduction of electrical impulse across segment of nerve.
- Reduction in motor CMAP or sensory SNAP or both across a nerve.
- Proximal conduction block can be difficult to detect.
- Discrepancy between the size of CMAP and the strength in muscle can be a clue for conduction block.
- Motor unit recruitment reduced.

- Focal demyelination, ischemia, cooling, local anesthetic

Demyelinating Polyneuropathy

- GBS,
- CIDP,
- MMN with CB,
- Associated with monoclonal gammopathy
- Associated with myeloma,
- Focal compressive neuropathies,
- HSMN type 1.

Mononeuritis multiplex

- Collagen vascular disorders
  vasculitis – polyarteritis nodosa, Churg - Strauss syndrome
  Rheumatoid arthritis

  SLE
  Wegener's granulomatosis
- Diabetes mellitus
- Infections: leprosy, diphtheria
- Lymphomatous infiltration of the nerves
- Hereditary neuropathy with susceptibility to pressure palsies
- Others

ELECTRODIAGNOSTIC STUDIES IN MONONEUROPATHY MULTIPLEX

- Evidence of axonal injury to multiple nerves
- Evidence of acute demyelination can be seen rarely, mixed pattern of axonal and demyelinating injury can be seen.
- Very advanced stages of mononeuropathy multiplex can be similar to polyneuropathy and careful evaluation of asymmetry in changes will be required to differentiate from polyneuropathy.
- HNPP changes will be predominantly demyelination – acute or chronic changes. Axonal loss seen late in the course.

Plexopathies

- Lesion in the plexus , brachial or lumbosacral
- Lesions can be axonal injury , demyelinating, or a mixed pattern
- Extent of lesion can be variable, single or at multiple sites
- ELECTRODIAGNOSTIC FINDINGS

  reduced or absent SNAP in the appropriate distribution; reduced or absent motor CMAP; absent F waves, H waves; denervation acute or chronic in muscles innervated by the portion of the plexus.
- Extent of the lesion and pattern of lesion help determine the nature of injury.
- Varied lesions involve the brachial and lumbosacral plexus.

BRACHIAL PLEXOPATHY- CLASSIFICATION

- **CLOSED LESIONS**
- Traction injury
- Obstetric paralysis
- Radiation induced

  Neoplastic:

  primary&secondary

  Post median sternotomy

  Orthopedic related

  Burner syndrome

  TOS

  Pack palsy
- NEURALGIC AMYOTROPHY
- **OPEN LESIONS**

  Neurovascular

  Gunshot wounds

  Lacerations

  Needles and cannula insertion

  Others

Brachial plexopathy - Anatomical

- **UPPER TRUNK**

  Traction injury

  Obstetrical

  Gunshot and stab wounds

  Neuralgic amyotrophy

- **LOWER TRUNK**

  Thoracic outlet syndrome

  Apical lung lesions

  Median sternotomy

Lumbosacral Plexopathy

- Causes:
  Trauma – most common

  Neoplastic lumbosacral plexopathies

  Hemorrhagic Compartment syndrome

  Radiation-induced lumbosacral plexopathy

  Maternal lumbosacral plexopathy

  Retroperitoneal infection

  Diabetic amyotrophy

  Idiopathic

  ELECTRODIAGNOSTIC FINDINGS
  Absent or reduced sural , saphenous, peroneal SNAP

  Reduced CMAP in lower limb motor nerves

  Denervation of muscles in proximal and distal portion of the lower limbs

Thoracic Outlet Syndrome

- Compression injury to the lower trunk of the brachial plexus by cervical rib or band.
- Weakness, wasting of thenar muscles(especially lateral thenar) muscles with numbness in the ulnar distribution
- ELECTRODIAGNOSTIC FINDINGS:

  Normal median nerve SNAP with reduced ulnar SNAP. Medial cutaneous nerve of forearm.

Reduced thenar CMAP and to lesser degree in hypothenar muscle

Denervation of thenar muscles and to a lesser extent in muscles innervated by the ulnar nerves and lower trunk

Sciatic Neuropathy

- Combination of weakness in distribution of peroneal and tibial nerve
- Causes: Trauma; hip dislocation, fracture of hip and as complication of hip replacement procedure

  Intramuscular injections

  Prolonged pressure in the area of ischial tuberosity

  Tumours – schwanomma
  Intrapelvic hematoma – anticoagulant overdose
- Electrodiagnostic Studies:
  Reduced or absent response from sural and superficial peroneal nerves.
- Reduced CMAP from peroneal and tibial nerves.
- EMG ; denervation of peroneal, tibial and hamstring muscles.
- Differential diagnosis: Lumbosacral plexopathy

Diabetic Motor Neuropathies

- Proximal symmetric
- Proximal asymmetric (Garland's syndrome)
- Distal motor neuropathy
- Focal and multifocal mononeuropathy
- AUTONOMIC NEUROPATHY

Diabetic Proximal Asymmetric Motor Neuropathy

Garland at al, 1955

Middle aged or old diabetics

Excruciating pain in thighs usually asymmetric

Pain followed by rapid weakness in thigh muscles

Followed by slow gradual recovery of strength

Diabetic Polyneuropathies

- Nervous system frequently involved in diabetes mellitus.
- Peripheral nerves are frequently affected.
- Prevalence of peripheral neuropathies increases with duration of diabetes, age, height and more often in males.
- Prevalence of peripheral neuropathies is less frequent in childhood.

Diabetic Amyotrophy

- Rare and dramatic complication of diabetes mellitus
- Seen early in the course of DM and with recent weight loss and poor control of blood sugar.
- Pain in thighs , severe, asymmetric, followed by muscle wasting and weakness predominantly involving the upper part of the lumbosacral plexus.
- Absent knee jerk with preserved ankle jerks.
- Normal sural SNAP, saphenous SNAP may be absent

  Reduced femoral CMAP, peroneal and tibial CMAP may be reduced

  Denervation , prominent in the quadriceps muscles, adductors and often the hamstrings are involved.

  Lumbosacral paraspinals may be involved.

  Multi-focal and patchy lesions are common

Neuralgic Amyotrophy (Parsonnage -Turner syndrome)

- Unique clinical syndrome of undetermined cause.
- Associations: infections, immunizations, trauma, surgery
- CLINICAL SYNDROME:
  severe and intractable shoulder and arm pain

  As the pain resolves weakness sets in Weakness mainly in distribution of C5 and C6 root distribution

  Involvement is probably multifocal

  Absent biceps and sometimes triceps jerk

  Evidence of axonal injury to the involved muscles
- Recovery of motor function seen in majority of patients. Greater than 75% improve in strength. Rate of recovery is slow and can take several months.

- Reduced/ absent SNAP, reduced CMAP and patchy denervation in muscles innervated by various branches. Upper trunk muscles more commonly involved,
- Serratus anterior and muscles innervated by the anterior interosseous nerve may be involved in isolation

EMG and Nerve Conduction Studies with Clinical Correlation

Prof. V. Vedanarayanan

Case 1

A 37-year-old farmer has had progressive difficulty gripping with his right hand for the past 6 months. His hand "goes to sleep" when driving his tractor for more than 30 minutes, but feels better if he shakes it. Examination shows mild loss of pain and light touch sensation over the palmar side of his first three digits on the right. Findings on NCS and EMG are:

Nerves	Amplitude	Velocity	Latency	F-latency
Median motor, rt.	5.2	54	4.9*	28
Median motor, lt.	5.8	55	3.8	27
Median sensory, rt.	5*	51	4.6*	—
Median sensory, lt.	25	56	3.2	—

*= outside the normal range; hand temperature 32.0

Needle electromyography was normal in the right hand and arm muscles tested, especially C6 innervated muscles. Testing the opposite arm was normal.

REPORT

Summary. Motor and sensory nerve conduction studies showed only a moderate prolongation of median distal latencies on the right with a low amplitude sensory nerve action potential. Needle examination was normal.

Clinical Interpretation. The findings are those of a moderate, median neuropathy at the wrist, of the type seen with carpal tunnel syndrome.

Comment. The clinical picture of a carpal tunnel syndrome is confirmed with standard studies that demonstrate prolonged median motor and sensory latencies with a low-amplitude median sensory response on the right. If standard median motor and antidromic sensory responses had been normal, palmar testing over a shorter segment or comparison measurements with radial nerve to the thumb, ulnar nerve to digit IV, or transcarpal and distal median segments might demonstrate the defect; lumbrical recordings are also occasionally more sensitive.

Case 2

A 19-year-old college student dislocated his right shoulder in a motorcycle accident 4 months earlier. Since then he has had persistent weakness of his right shoulder. On examination there is deltoid muscle atrophy with no voluntary movement of that muscle. Other muscles appear to have normal strength, although the presence of shoulder pain made full testing difficult. No sensory loss was found. Reflexes were normal except for a slightly reduced right biceps reflex as compared with the left.

Nerves, right arm	Amplitude	Velocity	Distal latency	F-latency
Median motor	8.5	56	3.3	25
Median sensory, antidromic	18	58	2.9	—
Ulnar motor	9.0	55	3.1	25
Ulnar sensory, antidromic	24	57	2.7	—
Lateral antibrachial cutaneous	0*	No response	No response	—
Deltoid	0*	No response	No response	—
Musculocutaneous/biceps	6.5	67	4.3	—

*= outside the normal range; hand temperature 33.5

Other Conduction Studies

Left median sensory amplitude was 34 µV; left lateral antibrachial cutaneous sensory amplitude was 18 µV.

Needle electromyography

On needle examination, there were prominent fibrillation potentials in the left deltoid muscle with only two voluntary motor unit potentials; both were of very low amplitude, normal duration, polyphasic, and varying in configuration. Mild motor unit potential changes, with very few scattered fibrillation potentials were also seen in the biceps, infraspinatus, and brachioradialis muscles. No abnormalities were found in the cervical paraspinal muscles.

REPORT

Summary. Motor and sensory nerve conduction studies are normal except for absent left axillary motor (deltoid) and lateral antibrachial cutaneous responses. On needle examination, there is a severe loss of motor unit potentials with two nascent potentials in left deltoid. Some other left upper trunk muscle show similar, milder changes.

Clinical Interpretation. The findings are those of a severe left axillary neuropathy and mild upper trunk plexopathy. The axillary nerve is intact with some evidence of early, ongoing reinnervation.

Comment. The needle EMG findings confirm the presence of severe axillary nerve damage, but the small (nascent) motor unit potentials indicate that reinnervation is occurring and may be expected to continue. The median sensory asymmetry and antibrachial cutaneous loss, along with the EMG changes in other upper trunk muscles, indicate that there was also damage to the upper trunk of the brachial plexus. Improvement in the needle EMG and in the strength of those muscles is likely due to collateral sprouting (rather than to reinnervation from the site of damage) that is required for deltoid recovery after the more severe axillary nerve damage.

Case 3

A 45-year-old former football player with an old elbow injury has had intermittent tingling and numbness of his left hand for 1 year. The left hand feels "weaker." The symptoms are increasing in severity and frequency, but are not related to specific positions or activities. He has no neck pain or limitation of motion. Examination demonstrates a loss of pain and touch sensation in digits IV and V on the left with mild weakness in intrinsic hand muscles. A Tinel's sign is present over the ulnar nerve at the elbow. No reflex changes are found.

Nerves, left arm	Amplitude	Velocity	Distal latency	F-latency
Median motor	8.5	56	3.8	25
Median sensory, antidromic	32	58	2.9	—
Ulnar motor	3.8*	44*	3.9*	31
Ulnary sensory, antidromic	0*	No response	No response	—

* = outside the normal range; hand temperature 32.5

Other Nerve Conduction Studies

Ulnar nerve stimulation distal to the elbow showed a normal amplitude and forearm conduction velocity. "Inching" the stimulation across the elbow segment of the ulnar nerve showed a localization conduction block and slowing at the level of the medial epicondyle. Needle EMG documented reduced recruitment with some large motor unit potentials and fibrillation potentials in ulnar innervated hand muscles. Flexor carpi ulnaris exhibited similar, but less severe abnormalities. Other arm muscles, especially those innervated by C8, were normal. Right arm conduction studies and EMG were normal.

REPORT

Summary. Motor and sensory nerve conduction studies show localized slowing of conduction with a 40% conduction block in the right ulnar nerve at the medial epicondyle. On needle examination there were moderate motor unit potential changes with fibrillation potentials in right ulnar innervated muscles.

Clinical Interpretation. The findings are those of long-standing, moderately severe, ulnar neuropathy at the elbow, localized at the medial epicondyle with a 40% conduction block. The conduction block suggests that significant improvements in function could occur, if the local compression could be relieved.

Comment. The clinical impression of an ulnar neuropathy at the elbow is confirmed with specific localization of the damage. The large motor unit potentials and fibrillation potentials are evidence of both a long-standing and an ongoing process. The presence of a conduction block indicates a good prognosis for improvement if the local compression can be relieved. If these standard tests had not demonstrated an abnormality, motor conduction to the first dorsal interosseous muscle, or sensory conduction across the elbow might have done so. At a later stage in this disorder, the localized abnormality might be replaced by slowing and amplitude loss with distal stimulation as well, making it more difficult to precisely localize the site of damage.

Case 4

A 59-year-old hog farmer with mild diabetes and chronic neck and back pain from degenerative osteoarthritis has had numbness of the left fourth and fifth digits for the past 6 weeks. On examination there is limitation of all spine motion with mild to moderate pain, but no radicular symptoms. There is mild weakness of intrinsic hand and foot muscles bilaterally, absent Achilles reflexes, and mild distal sensory loss, especially in the feet. The sensory loss is more prominent in the left hand and extends only one third of the way up the medial forearm.

Nerves, left arm	Amplitude	Velocity	Distal latency	F-latency
Median motor	4.9	50	4.2	33*
Ulnar motor	5.7*	48	3.9*	33*
Median sensory	11*	49*	3.9*	—
Ulnar sensory	8*	51*	3.3*	—
Peroneal motor	2.5	38*	5.3	No response
Sural	0*	No response	No response	—

* = outside the normal range; hand temperature 32.9

Other Nerve Conduction Studies

Ulnar conduction studies of the right arm were similar to the left. Motor unit number estimate of the peroneal nerve was reduced to 25, while ulnar nerve was only borderline at 94.

Needle Electromyography

EMG showed widespread, large motor unit potentials with reduced recruitment in distal muscles; fibrillation potentials were seen in the intrinsic foot muscles. Motor unit potential changes were more prominent in the left C8 muscles, where scattered fibrillation potentials, including the low cervical paraspinal muscles, were found.

REPORT

Summary. Motor and sensory nerve conduction studies show a mild, generalized slowing of conduction with some low-amplitude responses. The changes may be slightly greater in the ulnar nerves, but with no evidence of localized abnormality. On needle examination there is a loss of motor unit potentials with large potentials in left C8 and other distal muscles.

Clinical Interpretation. The findings are those of an active left C8 radiculopathy superimposed on a mild peripheral neuropathy. The latter could be related to the patient's diabetes.

Comment. The nerve conduction and needle EMG findings confirm the presence of a mild peripheral neuropathy, likely related to his diabetes. The more prominent needle EMG changes in the left C8 muscles indicate the presence of an additional, superimposed C* radiculopathy, likely due to foraminal stenosis. More prominent slowing of motor conduction in the left ulnar nerve, without concomitant change in the ulnar sensory responses, is more likely due to a loss of C8 axons in the ulnar nerve than to an additional ulnar neuropathy at the elbow or a more proximal involvement of the plexus.

Case 5

A 55-year-old attorney, who has smoked heavily for 35 years, was recently found to have carcinoma of the lung. During medical evaluation prior to surgical resection, he was noted to have weakness of his right leg. No sensory loss or reflex change was recorded.

Nerves,right leg F latency	**Amplitude**	**Velocity**	**Distal latency**	**F-latency**
Peroneal motor	0.5*	34*	5.6	No response
Peroneal sensory	0	No response*	No response*	No response
Tibial motor	10.5	46	4.9	54
Sural	14	43	3.9	—

* = outside the normal range; foot temperature 29.5

Other Nerve Conduction Studies

A 75% decrease in CMAP amplitude was found at the head of the fibula by "inching" (stimulation at 2.0-cm. intervals) along the nerve with a 2.0-mV response below the head of the fibula. No abnormalities were seen with repetitive stimulation of the peroneal nerve.

Needle Electromyography

EMG showed marked reduction in recruitment in the anterior compartment muscles. No fibrillation potentials or motor unit potential changes were seen. Other leg muscles, including L5 innervated muscles and the short head of the biceps femoris, were normal.

Report

Summary. Motor and sensory nerve conduction studies show only a localized conduction block of the peroneal motor fibers of the head of the fibula, and absent peroneal sensory response. Needle examination show only a loss of motor unit potentials in distribution of the right peroneal nerve distal to the knee.

Clinical Interpretation. The findings are those of a moderately severe, recent onset, left peroneal neuropathy with a 75% conduction block localized at the head of the fibula. No evidence of a more proximal lesion was found. These findings indicate that there could be a rapid, good recovery if the local compression could be relieved.

Comment. An unsuspected peroneal neuropathy localized to the head of the fibula was demonstrated by nerve conduction studies, likely related to recent weight loss with or without compression from leg crossing. The lack of changes in the motor unit potential or of the fibrillation potential confirm that the process is primarily a conduction block with little axonal disruption. A good recovery can be anticipated if the local compression is relieved.

Case 6

A 38-year-old logger awoke 2 weeks prior to this study, with acute, severe, back and right leg pain, exacerbated by coughing and straining. The back pain had subsided with bed rest, but the leg pain persisted, and he had developed sensory symptoms on his leg and foot. He had had similar symptoms 3 years earlier that had resolved with rest. Examination documented no weakness, but there was mild sensory loss on the dorsum of the foot, and the right ankle reflex was reduced. An MRI of the lumbosacral spine was normal. Findings on NCS and EMG are:

Nerve, right leg	Amplitude	Velocity	Distal latency	F latency
Peroneal motor	3.8	45	4.2	No response
Peroneal sensory	18	—	3.7	—
Tibial motor	15.4	48	4.7	52
Sural	22	44	3.6	—

* = outside the normal range; foot temperature 29.5

Other Nerve Conduction Studies

Peroneal and tibial motor conduction studies were normal. No peroneal F waves could be elicited from either leg, but tibial F waves were normal. The extensor digitorum brevis amplitude was higher on the left than the right. Sural and superficial peroneal sensory responses were normal and symmetrical.

Needle Electromyography

EMG showed reduced recruitment with minimal motor unit potential change in the peroneus longus, extensor hallucis, flexor digitorum, tensor fascia lata, and gluteus medius muscles. Fibrillation potentials were seen only in the right low lumbar paraspinal, gluteus medius, and tensor fascia lata muscles. Large motor unit potentials with no fibrillation potentials were seen in right S1 innervated muscles distally.

REPORT

Summary. Motor and sensory nerve conduction studies were normal. On needle examination, there were fibrillation potentials with reduced recruitment in left L5 muscles. More prominent motor unit potential changes were seen in distal S1 muscles.

Clinical Interpretation. The findings are those of a recent-onset, right L5 radiculopathy superimposed on the residuals of an old right S1 radiculopathy.

Comment. The motor conduction studies suggest the presence of a proximal L5 radiculopathy with CMAP asymmetry and normal sensory response. This is confirmed by the observation of L5 fibrillation potentials. The proximal distribution, with no motor unit potential configuration changes, indicates that the L5 radiculopathy is of very recent onset. In contrast, the S1 muscle changes are those of an old radiculopathy and are consistent with the clinical reduction in the ankle reflex.

Case 7

A healthy, 27-year-old nurse awoke with a “numb” right face, and inability to smile or pucker on the right, with no preceding illness or injury. There was a history of “cold sores” in the mouth. Neurologic examination 4 days later was normal except for total absence of right facial movement. Findings on NCS and EMG are:

Nerve	Amplitude	Distal latency
Facial motor, right	3.2	3.3
Facial motor, left	3.4	3.2

	R1 Latency	R2 Latency, right	R2 Latency, left
Blink reflex, right	No response*	No response*	34.5
Blink reflex, left	10.5	No response*	33.9

* = outside the normal range.

Needle Electromyography

EMG of right facial muscles showed marked reduction in recruitment, with only a single, normal-appearing MUP in each of the muscles tested. The masseter and left facial muscle were normal. No fibrillation potentials were found.

REPORT

Summary. No blink responses were recorded on the right. On needle examination, there was reduced recruitment, but with some residual voluntary potentials in right facial muscles.

Clinical Interpretation. The findings are those of an acute, severe, right facial neuropathy of the type seen with Bell's palsy. The normal right facial response indicates that the facial nerve axons are still intact and could make an excellent recovery.

Comment. Absent ipsilateral and contralateral blink reflex responses on the right are consistent with a severe right facial neuropathy. The normal contralateral R2 with stimulation on the right indicates that the right trigeminal nerve is intact. Wallerian degeneration, if present, causes CMAP reduction at 3-5 days. The intact facial CMAP at 4 days indicates that the axons have not undergone Wallerian degeneration. This is most likely because the process is due to a conduction block proximal to the site of stimulation that has blocked the blink reflex, but leaves the peripheral facial response intact. A conduction block can recover fully. Acyclovir therapy may not be effective at 4 days, but with normal facial CMAP, is worth using to minimize the likelihood of axonal damage.

Case 8

A 33-year-old homemaker, with two children, developed progressive difficulty climbing stairs and arising from low chairs, with distal tingling, over the course of 2 weeks, 10 days following an acute diarrheal illness. She also had moderately severe mid and low back pain. Over the last 3 days, some weakness of the arms and drooling had also developed. On neurologic examination, there was generalized weakness, including facial weakness, with absent tendon reflexes. No sensory loss was found, but there was paraspinal muscle tenderness. The CSF was normal. Findings on NCS and EMG are:

Nerve, right	Amplitude	Velocity	Distal latency	F latency
Median motor	3.2*	51	5.6*	41*
Ulnar motor	4.5*	49*	3.8*	38*
Median sensory	8*	54*	3.8*	—
Peroneal motor	0.6*	39*	6.8*	No response
Tibial	2.5*	36*	7.3*	No response
Sural	28	44	3.9	—

R1 Latency		R2 Latency, right	R2 Latency, left
Blink reflex, right	14.2*	44*	45*
Blink reflex, left	14.8*	46*	47*

*= outside the normal range; foot temperature 30.4

Additional Nerve Conduction Studies

Dispersion of CMAP with no conduction block was seen in each motor nerve. Motor unit number estimates were normal.

Needle Electromyography

EMG was performed in proximal and distal muscles on the right side, including paraspinal and facial muscles.There was a marked reduction in recruitment in all muscles tested with scattered fibrillation potentials in paraspinal muscles. No motor unit changes were seen, but there were myokymic discharges in facial muscles.

REPORT

Summary. Motor and sensory nerve conduction studies show marked prolongation of distal latencies, F wave latencies, and blink reflexes with low-amplitude, dispersed CMAP. On needle examination, there was a marked loss of motor unit potentials, with fibrillation potentials only in proximal muscles.

Clinical Interpretation. The findings are those of a moderately severe inflammatory demyelinating polyradiculoneuropathy with some evidence of axonal loss. This pattern is typical of Guillain-Barré syndrome (GBS). The signs of axonal loss suggest that recovery may be slow.

Comment. The clinical impression of GBS is confirmed by the marked distal latency and F wave latency prolongation, and the CMAP dispersion. The distribution of slowing is often most prominent in distal latencies and proximally with F wave latencies, but may have any distribution and may be asymmetrical. The low-amplitude CMAP, the fibrillation potentials, and some reduction in arm SNAP, provide evidence of axonal loss as well. More severe involvement with axonal loss is more common in patients whose disease follows *Campylobacter* infection. Sparing of the sural response is typical of GBS. Back pain is more common with involvement of the spinal nerves. Myokymic discharges are seen occasionally in GBS during the acute phase, especially in facial muscles.

Case 9

A 35-year-old woman employed as a state environmental safety engineer became acutely ill with fever, malaise, and flu-like symptoms 2 months prior to this study. She recovered gradually over 2 weeks, but was left with numbness of her hands and feet, imbalance while walking, and tingling of her lower face. Neurologic examination showed moderate distal sensory loss for all modalities, pseudoathetosis, gait ataxia, and reduced or absent tendon reflexes. Muscle strength and cranial nerve functions were normal. Extensive laboratory testing in search of an underlying cause of neuropathy had been unrevealing. Findings on NCS and EMG are:

Nerve, right	Amplitude	Velocity	Distal latency	F latency
Median motor	6.9	52	3.2	28
Ulnar motor	7.9	53	2.9	27
Median sensory	0*	No response	No response	—
Ulnar sensory	0*	No response	No response	—
Radial sensory	0*	No response	No response	—
Peroneal motor	4.4	44	5.1	No response
Tibial motor	10.6	44	4.8	5.6
Sural	0*	No response	No response	—

R1 latency		R2 latency, right	R2 latency, left
Blink reflex, right	No response	—	—
Blink reflex, left	No response	—	—

* = outside the normal range; foot temperature 31.5

Additional Nerve Conduction Studies

Masseter inhibitory reflex was normal.

Needle Electromyography

EMG of arm, left, paraspinal and cranial muscles was normal.

Summary. Motor nerve conduction studies were normal, but sensory studies showed an absence of all SNAP. Masseter inhibitory reflex was normal. No abnormalities were seen on needle examination.

erpretation. The findings are those of a sensory neuropathy. The clinical picture in combination with intact cranial reflexes suggests that this is an acute inflammatory sensory ganglionopathy.

Comment. The history of subacute onset and no further progression is that of an inflammatory process. The prominent sensory deficit, including gait ataxia and cranial symptoms suggests that this is likely a proximal process. Unlike GBS, the NCS and EMG here confirm that this is a pure sensory disorder. The absent sensory potentials in all extremities is consistent with this, and the absent blink reflexes with a normal masseter inhibitory reflex confirm it. The sensory ganglion cells of the masseter inhibitory reflex are in the brainstem, while the other sensory ganglion cells are in the periphery. The former location serves to protect those cells from the immune-mediated attack.

Case 10

A 65-year-old homemaker has had generalized weakness for 18 months, manifested as difficulty climbing stairs, arising from chairs, and lifting her arms over her head. She has had no rash or other systemic signs or symptoms. There is no family history of muscle disease. A previous EMG and muscle biopsy were said to have identified her disease as "myositis." Treatment with prednisone and azathioprine for a number of months resulted in no measurable improvement in symptoms or signs.

On examination, she has moderate symmetrical proximal weakness that does not fatigue. There is also some weakness of finger flexors. Reflexes are absent at the ankles and mildly reduced elsewhere. Findings on NCS and EMG are:

Nerve, right	Amplitude	Velocity	Distal latency	F latency
Median Motor	4.5	51	3.8	28
Ulnar motor	5.9*	49*	3.5	29
Median sensory	13*	52*	3.5	—
Peroneal motor	2.3	39*	4.9	58
Sural	0	No response	No response	—

* = outside the normal range; foot temperature 29.8

Additional Nerve Conduction Studies

Repetitive stimulation of the median and ulnar nerves showed no abnormalities.

Needle Electromyography

Abnormalities were seen in all muscles with many short duration, low amplitude, polyphasic MUP. Scattered fibrillation potentials were also seen, particularly in proximal, paraspinal, and forearm muscles. No myotonic discharges were found. A few of the polyphasic motor unit potentials varied. Intermingled with the small potentials were scattered long-duration, high-amplitude, polyphasic motor unit potentials.

REPORT

Summary. Motor and sensory nerve conduction studies show borderline or mild slowing of conduction with some borderline low-amplitude responses. Repetitive stimulation was normal. On needle examination, there were widespread short duration, polyphasic muscles with fibrillation potentials. Some intermingled large potentials were also seen.

Clinical Interpretation. The findings are those of an active, inflammatory myopathy of moderate severity with findings of an additional mild peripheral neuropathy. Some large potentials may be seen in chronic myositis but also raise the possibility of inclusion body myositis. The distal arm weakness and signs of a peripheral neuropathy are consistent with this diagnosis. However, some collagen-vascular diseases may demonstrate a combination of inflammatory myopathy and peripheral neuropathy. These changes are more than would be expected with a steroid myopathy.

Comment. The clinical history and findings are those of a myopathy, but testing for a defect of neuromuscular transmission is always appropriate in patients with proximal weakness. The mild or borderline changes on nerve conduction studies are evidence of a mild peripheral neuropathy but do not identify a specific type of neuropathy. While the pattern of abnormalities seen in this patient could result from more than one cause, a comment in the report about less well-known disorders is appropriate to assist the clinician in sorting out the problems. A careful search for slowly firing, small fibrillations in proximal muscles, especially paraspinal, is particularly important in patients suspected of having a myopathy in whom fibrillation potentials are not readily found.

Case 11

A 68-year-old, generally healthy, retired family doctor developed intermittent mild drooping of his right eye 4 months prior to this examination. In the last 2 weeks, he notices some fatigue when climbing stairs, but otherwise feels well. Neurologic examination demonstrates mild weakness in the neck muscles, with additional, but mild weakness after prolonged effort in other proximal muscles. Reflexes and sensations are normal. Findings on NCS and EMG are:

Nerve, right	Amplitude	Velocity	Distal latency	F latency
Median motor	7.9	54	2.9	25
Ulnar motor	8.4	56	2.7	24
Median sensory	22	58	3.4	—
Peroneal motor	3.8	45	4.2	52
Sural	22	44	3.6	—

* = outside the normal range; foot temperature 30.5

Repetitive Stimulation Decrement Before and After 1 Minute Exercise

Nerve, right	minutes	Rest	Immediate	1 minute
Median/thenar	0%	0%	0%	0%
Ulnar/hypothenar	2%	0%	1%	3%
Accessory/trapezius	7%	2%	8%	14%
Facial/nasalis	11%	4%	13%	18%

Needle Electromyography

EMG of hand and forearm muscles was normal. Proximal arm muscles had scattered, varying MUP with a few localized areas of irritability. Infraspinatus exhibited more prominent MUP variation with an excess of short-duration potentials. Cervical paraspinal and masseter muscles had similar MUP changes with scattered areas of fibrillation potentials.

REPORT

Summary. Motor and sensory nerve conduction studies are normal, but repetitive stimulation shows a decrement, especially in proximal muscles. On needle examination, there are both varying and short-duration motor unit potentials, especially in the paraspinal muscles, where some fibrillation potentials were also seen.

Clinical Interpretation. The findings are those of a defect of neuromuscular transmission of the type seen in myasthenia gravis. Short-duration motor unit potentials with fibrillation potential can be seen in myasthenia gravis, particularly in paraspinal and masseter muscles. Pharmacologic therapy is often less effective in this setting.

Comment. The pattern of decrement in a number of muscles provides convincing evidence of a defect of neuromuscular transmission, in this patient most likely due to myasthenia gravis. Decrements of less than 10% may be seen in muscle, with little clinical weakness, but are not sufficiently reliable to ascertain the abnormality. It is particularly important to be sure that an apparent decrement is not due to movement, especially in proximal muscles. Testing should therefore begin in the more reliable distal muscles. If a clear decrement is not found, more proximal testing is needed. If repetitive stimulation had been entirely normal in a patient like this, the EMG would still usually show MUP variation. Only in rare cases is it necessary to perform SFEMG to demonstrate the abnormality. Short-duration MUP are the result of the presence of a sufficient number of blocked muscle fibers to reduce the number of fibers contributing to the MUP. Fibrillation potentials occur when the damage to the end-plate zone is sufficiently severe to denervate the muscle fiber and do not imply the presence of another disease.

Notes