Technical Note

Single-fiber EMG: A review

V. Arul Selvan
Walton Center for Neurology and Neurosurgery, Lower Lane, Fazakerley, L9 7LJ, Liverpool UK.

For correspondence:
Dr. V Arul Selvan, Consultant, Walton Center for Neurology and Neurosurgery, Lower Lane, Fazakerley, L9 7LJ, Liverpool, UK, E-mail: arulselvan@doctors.org.uk

Ann Indian Acad Neurol 2011;14:64-7

Introduction

Single fiber electromyography (SFEMG) was established by Stalberg and Eskedt in the 1960s, and is of proven value in the diagnosis of neuromuscular disorders, especially myasthenia gravis. It has proved to be the most sensitive technique in detecting a neuromuscular transmission defect in comparison with the tension test, repetitive stimulation, and acetyl choline receptor antibody estimation.

Single fiber electromyography typically requires the use of a specially contracted single fiber EMG needle electrode [Figure 1] or facial concentric needle electrode [Figure 2] with a small recording surface (25 micrometers), which is exposed at a port on the side of the electrode, 3 mm from the tip.

The validity of the technique has been proven by examining a large number of myasthenia patients with a sensitivity of up to 99% in detecting a neuromuscular transmission defect. In generalized myasthenia gravis has been reported. When a motor axon is depolarized the action potentials travel distally and excite the muscle fiber more or less at the same time. The variation in the time interval between the two action potentials of the same motor unit is called as ‘jitter’. SFEMG measures the variation of this inter potential interval (jitter) [Figure 3].

Technical considerations

Most of the modern nerve conduction and EMG machines have a software to perform and analyze the SFEMG examination. There are two methods to perform this. One is stimulated and the other is under a volitional effort. However, most physicians, including the author, prefer to perform SFEMG under the volitional effort. The goal of SFEMG is to study the adjacent action potentials from the same motor unit, known as ‘pairs’. This is achieved by using a specially constructed single fiber needle electrode or facial concentric needle electrode. This identifies and selectively records the action potentials from individual muscle fibers. The selectivity of the recording is further strengthened by adjusting the filter settings. Low frequencies filter 500 HZ — high frequencies filter 10 KHZ. This filter setting selectively abolishes low frequency components from distant muscle fibers. Action potentials should be greater than 200 microwatts in amplitude and the rise time should be less than 300 microseconds. Around 20 potential pairs are collected from the same muscle by three-to-four insertions. The subject is asked to maintain a steady contraction if volitional SFEMG is undertaken, until 100 consecutive discharges are recorded from each pair.

Stimulation SFEMG is particularly useful in children, uncooperative, comatose patients, and those who have tremors.

Access this article online
Quick Response Code:
Website: www.annalsofian.org
DOI: 10.4103/0972-2327.78058

Figure 1: Single fibre needle electrode
Figure 2: Concentric facial needle electrode
A branch of the motor nerve is stimulated by using a monopolar needle electrode and recording is made by SFEMG or a concentric needle electrode. Stimulation is delivered at 2 – 10 HZ and the stimulus intensity is adjusted accordingly.

**Jitter Analysis:**
Jitter is the measurement of variation of the inter-potential interval. This is calculated between the triggered potential and the time-locked, second single muscle fiber action potential [Figures 4-7]. This is expressed as a mean consecutive difference (MCD). Most modern EMG machines have a program that automatically performs the MCD calculation. Mean MCD is calculated using the following formula:

\[ \text{MCD} = \frac{(IP_1 - IP_2) + (IP_2-IP_3) + \ldots + (IP_n - IP_n)}{n - 1} \]

When neuromuscular transmission is sufficiently impaired, nerve impulses fail to elicit an action potential and this is called ‘blocking’ [Figure 8]. This usually happens when the jitter value is markedly prolonged, usually when MCD is more than 100 microseconds.[5]

**Normal values**
The normal jitter values have been determined for many muscles in a multicenter collaborative study[6,7] [Figure 9]. The study is considered abnormal if one of the following criteria is met,

- Mean jitter value exceeds the upper limit of the normal value
- More than 10% of the pairs have increased jitter (two out of twenty pairs)

Normal values apply only if the inter-spike interval is up to four microseconds. Errors may be encountered if this is higher and may produce a false jitter.

**Discussion**
In patients with myasthenia gravis, jitters are greater in the weak muscles, but they are also increased in muscles with normal strength. Jitter is abnormal in the orbicular oculi > 95%, followed by frontalis and extensor digitorum communis in 85% of the patients. Jitter is abnormal even when patients take anticholinesterase inhibitors. [8] It is our practice to not recommend stopping this, except when the study is normal and also when there is a strong clinical suspicion of myasthenia gravis. Extensor digitorum communis is usually tested first, unless symptoms or signs are limited to extraocular muscles.
Table 1: Reference values for Jitter measurement in healthy subjects during voluntary muscle activation (microseconds): 95% confidence limits for upper limit of mean, MCD / 95% confidence limits for MCD values of individual fibers

| Muscle           | 10yr | 20yr | 30yr | 40yr | 50yr | 60yr | 70yr | 80yr | 90yr |
|------------------|------|------|------|------|------|------|------|------|------|
| Frontalis        | 33.6| 33.9| 34.4| 35.5| 37.3| 40.0| 43.8|     |      |
| Obicularis oculi | 39.8| 39.8| 40.0| 40.4| 40.9| 41.8| 43.0|     |      |
| Obicularis oris  | 34.7| 34.7| 34.9| 35.3| 36.0| 37.0| 38.3|     |      |
| Tongue           | 32.8| 33.0| 33.6| 34.8| 36.8| 39.8| 44.0|     |      |
| Stern Cleido mas | 29.1| 29.3| 29.8| 30.8| 32.5| 34.9| 38.4|     |      |
| Deltoid          | 32.9| 32.9| 32.9| 32.9| 33.0| 33.0| 33.1|     |      |
| Biceps           | 29.5| 29.6| 29.6| 30.1| 30.5| 31.0|     |      |      |
| Ext dig comm     | 34.9| 34.9| 35.1| 35.4| 35.9| 36.6| 37.7| 39.1| 40.9|
| Abd digit V      | 44.4| 44.7| 45.2| 46.4| 48.2| 51.0| 54.8|     |      |
| Quadriceps       | 35.9| 36.0| 36.5| 37.5| 39.0| 41.3| 44.6|     |      |
| Ant tibialis     | 49.4| 49.3| 49.2| 48.9| 48.5| 47.9| 47.0| 45.8| 44.3|

Figure 7: Abnormal jitter values in a sequential plot

Figure 8: Single fiber EMG recordings: (a) Normal; (b) Increased; Jitter (c) Blocking both increased jitters, and blocking is seen in the neuromuscular disorders. (With permission from EMG and Neuromuscular Disorders, Preston and Shapiro; Butterworth - Heinemann)

when orbiculorius oculi or frontalis are tested.

Jitter is increased in myasthenia gravis, but it does not correlate well with disease severity. However, in serial SFEMG studies, the mean jitter values increase by at least 10% in the tested muscles in two-thirds of the patients who become worse, and the converse is also true. In a few cases, SFEMG was performed before and after remission of myasthenia. Although the mean jitter values had decreased, some pairs still showed abnormalities, indicating that SFEMG did not normalize completely.

A comparison was made between the diagnostic yield of repetitive stimulation, antibody titers, and SFEMG [Figure 10]. The SFEMG was highly sensitive (99%), followed by the acetyl choline receptor antibody, and the least sensitive was repetitive stimulation (76%), if the proximal muscles were tested. Repetitive stimulation was technically difficult and mild decremental response was well-recognized in motor neurone disease and peripheral neuropathies. In myastenia...
Gravis, the decremental response was less pronounced in the distal muscles than in the proximal muscles. Acetyl cholinereceptor antibody was detected in only 50% of ocular myasthenia and 85% of generalized myasthenia patients. The remaining patients were treated as 'seronegative', but a proportion of such patients have antibodies to MUSK (muscle specific tyrosine kinase).

Single fiber electromyography is highly sensitive, but not specific to the diagnosis of myasthenia and myasthenic syndromes. It must be emphasised that increased jitter values are not pathognomonic for myasthenia, but indicate disturbed neuromuscular transmission. Increased jitter values are seen during the early stages of reinnervation, when motor unit remodeling occurs. Such changes can be seen in motor neurone disease, polyneuropathies, polymyositis, and Facioscapulohumeral dystrophy. However, it is also true that if SFEMG is normal in a weak muscle, it almost completely excludes the diagnosis of myasthenia.

**Conclusion**

Single fiber electromyography is the most sensitive test to demonstrate an impaired neuromuscular transmission like myasthenia gravis. However, it must be emphasized that it is not specific, as SFEMG can be abnormal in other myopathic and neuropathic disorders. The test is safe, but technically demanding for both the patient and the neurologist performing it. It needs considerable experience and technical expertise.

**References**

1. Sanders DB, Stalberg EV. AAEM minimonograph # single fiber EMG, Don sanders and Erik V stalberg. Muscle nerve 1996;19:1069-83.
2. Sarrigiannis PG, Kennett RP, Read S, Farrugia ME. Single fiber EMG with a concentric needle electrode validation in myasthenia gravis. Muscle Nerve 2006;33:61-5.
3. Padua L, Stalberg E, LoMonaco M, Evoli A, Batocchi A, Tonali P. Single fiber EMG in ocular myasthenia. Clin Neurophysiol 2000;111:1203-7.
4. Stalberg E. Clinical and electrophysiology in myasthenia gravis. J Neurol Neurosurg Psychiatry 1980;43:622-33.
5. Single fiber EMG reference values: a collaborative effort. Ad Hoc Committee of the AAEM Special Interest Group on Single Fiber EMG. Muscle Nerve 1992;15:151-61.
6. Bromberg MB, Scott DM. Adhoc committee of AAEM special interest group on single fiber EMG:SF EMG reference values reformatted in tabular form. Muscle Nerve 1994;17:820-1.
7. Massey JM, Sanders DB, Howard JF Jr. Serial single fiber EMG studies in myasthenic patients treated with corticosteroids plasma exchange therapy. Muscle Nerve 1981;4:254.
8. Massey JM, Sanders DB, Howard JF Jr. The effect of cholinesterase inhibitors on single fiber EMG in myasthenia gravis. Muscle Nerve 1989;12:154-5.
9. Preston DC, Schapiro B. EMG and Neuromuscular disorders. Oxford: Butterworth-Heinemann; 1998.
10. Farrugia M, Jacob S, Sarriaganpis P, Kennett RP. Correlating extent of neuromuscular instability with acetyl choline receptor antibodies. Muscle Nerve 2009;39:489-93.
11. Milne M, Monaco ML, Evoli A, Serviloi S, Toneli P. Ocular Myasthenia: diagnostic value of single fiber EMG in orbiculares oculi muscle. J Neurol Neurosurg Psychiatry 1993;56:720-3.
12. Stalberg E. Single fiber EMG J V Trontlej Old woking. Surrey: Mirrale Press; 1979.
13. Farrugia ME, Weir A, Cleary M, Cooper S, Metcalfan R, Malik A. Concentric and single fiber EMG needle electrodes yield comparable jitter results in myasthenia gravis. Muscle Nerve 2009;39:579-85.
14. Benetar M, Hamad M, Doss Riney H. Concentric needle single fiber EMG for the diagnosis of myasthenia gravis. Muscle Nerve 2006;34:163-8.

Received: 22-06-10, Revised: 01-02-11 Accepted: 05-02-11

Source of Support: Nil, Conflict of Interest: Nil