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CLINICAL UTILITY OF SURFACE EMG

Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology

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This report reviews the clinical uses of surface electromyography (SEMG) as a diagnostic tool for neurologic disorders. SEMG is assessed with regard to the evaluation of patients with neuromuscular diseases, low back pain, and disorders of motor control. This broadens the scope of a previous assessment of SEMG in neurologic practice by the American Association of Electrodiagnostic Medicine[1] in which its utility was examined with regard to neuromuscular diseases only.

Needle electromyographic evaluation (NEMG), in combination with nerve conduction studies, is the gold standard methodology for assessing the neurophysiologic characteristics of neuromuscular diseases. Moreover, fine-wire EMG (FWEMG) often has been used in the evaluation of gait disorders, kinesiologic studies, and research and is also considered a standard. Nevertheless, NEMG and FWEMG are both invasive and painful, and this limits their use when activity from several muscles needs to be monitored simultaneously.

SEMG is a technique to measure muscle activity noninvasively using surface electrodes placed on the skin overlying the muscle. SEMG differs from NEMG and FWEMG with respect to technical requirements and electrical properties. Unlike NEMG, SEMG electrodes record from a wide area of muscle territory, have a relatively narrow frequency band (range, 20 to 500 Hz), have low-signal resolution, and are highly susceptible to movement artifact. SEMG electrodes typically are approximately 10 mm in diameter and usually are passive (i.e., they are simple conductive surfaces requiring low skin resistance). They can, however, be active, incorporating preamplifier electronics that lessen the need for low skin resistance and improve the signal-to-noise ratio. SEMG can record both voluntary and involuntary muscle activity in addition to externally stimulated muscle action potentials such as motor evoked potentials after central or peripheral nerve stimulation.[2] SEMG has also been used in several non-neurologic settings such as obstetric monitoring and animal research, but these potential applications are beyond the scope of this review.

More than 2500 original articles, reviews, and books were examined to determine the scope of SEMG utility, its benefits and risks, and the extent to which SEMG techniques vary, and to assess SEMG's strengths and weaknesses for specific clinical applications. Manual and computerized literature searches from the National Library of Medicine were used to obtain the articles. Key words used included SEMG, spontaneous activity, fasciculation, myopathy, muscle fiber conduction, motor unit estimation, fatigue, low-back pain, tremor, movement disorders, reaction time, and psychophysics. Representative articles are cited and listed at the end of this article. Other key words relating to neuromuscular diseases (other than when cross-referenced with SEMG) were not searched for specifically because this topic was the focus of the earlier AAEM assessment[1] and was not the main focus of the current paper.

Neuromuscular diseases. No original article or review article has suggested that SEMG is better or even equivalent to NEMG in providing evidence of denervation at rest. This is because of the limited spatial resolution of SEMG that results in poor fidelity recordings of high-frequency signals such as polyphasic potentials, fibrillation potentials, and positive sharp waves.[3] In addition, because of electrical cross-talk, SEMG cannot identify the origin of the electrical signal when two or more muscles, which lie in close proximity to each other, are active simultaneously. Furthermore, the electrical signals in SEMG recordings are often attenuated by intervening soft tissue, particularly when the active muscle is 10 mm or more below the skin surface.[4] Insertional activity, another important measure in the evaluation of neuromuscular disease,[2,5] cannot be evaluated by SEMG for the self-evident reason that SEMG is noninvasive.

Some studies have proposed that SEMG may be a useful adjunct in the evaluation of fasciculation, particularly in the assessment of patients with neuromuscular disease. In a review of 116 patients with a variety of neuromuscular conditions, including, among others, motor neuron disease (n = 43), neuropathy (n = 14), myelopathy or radiculopathy (n

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= 9), spinal muscular atrophy (n = 6), benign cramps and fasciculation (n = 7), and postpolio syndrome (n = 5), SEMG was compared to NEMG and clinical observation in detecting fasciculation.[6] Pairs of plate electrodes were placed in a total of eight sites per patient. SEMG was reported to detect fasciculation in 284 (82%) of the 344 sites studied compared with clinical observation (38.4%) and NEMG (73.6%). This study, however, lacks a gold standard and does not assess the specificity of the SEMG findings. Further, the study was retrospective, so there was no standard for the method for either clinical observation or conventional NEMG recording. Similarly, patients with motor neuron disease were studied using SEMG, and fasciculations were detected in 107 (95.5%) of 112 sites, whereas clinical examination alone revealed fasciculations in 69 (61.1%) of these sites.[7] Again, a gold standard is lacking and specificity is not assessed. As a result, these studies represent Class III evidence for a clinical role of SEMG in the evaluation.

Two studies reported a relationship between electromechanical coupling and myopathies. Using the ratio of SEMG electrical amplitude to acoustic amplitude, children with Duchenne, Becker, and myotonic dystrophy had significantly higher ratios than normal control subjects.[8] Similarly, using the ratio of the root mean square SEMG amplitude to the mechanomyogram amplitude, the electromechanical coupling efficiency of some muscles was statistically different in patients with myotonic dystrophy (all with CTG expansion) compared with that of age-matched control subjects.[9] In a brief report,[10] SEMG activity in one patient with myotonia congenita and transient weakness showed a decline in mean root SEMG voltage compared with that of a patient without transient weakness and a single control. Based on this observation, the authors suggested that SEMG could be a reliable and painless method to investigate and quantify transient weakness in myotonia congenita. Using a principal component analysis of SEMG activity, patients with Duchenne could be separated from control subjects when recording from the biceps but not the brachioradialis muscle in severely affected patients.[11] These studies, however, used nonstandard and dissimilar methods, did not replicate the findings of other authors, and generally used small sample sizes. Thus, these reports do not allow an assessment of the sensitivity and specificity of the procedures and constitute, at best, only weak and inconsistent Class III evidence in favor of a clinical role for SEMG in the evaluation of myopathies.

Using turns frequency, zero crossing frequency, and median power frequency analyses, SEMG was compared to NEMG in the study of interference patterns of maximal voluntary contraction from various muscles.[12] In the tibialis anterior, but not in the rectus femoris, there was similarity between SEMG and NEMG parameters. However, data from these two techniques correlated only when the needle was within 0.5 mm of the muscle surface. To evaluate the sensitivity, specificity, and positive predictive value of SEMG as a diagnostic test, 61 control subjects and 72 patients were studied using "high-spatial resolution" SEMG. The results were compared with those of available recognition rates by NEMG and incorporated into a computerized evaluation procedure that combined and weighted different parameters to optimize the recognition rate of diagnosis.[13] Again, NEMG was superior to SEMG by these measures.

A few studies directly compared SEMG to NEMG to examine muscle fiber conduction velocity (MFCV), but in none were attempts made to ascertain the best methodology using unbiased approaches or double-blind crossover techniques. Thus, patients with ALS showed significantly reduced mean NEMG MFCV compared with that of control subjects.[14] SEMG, by contrast, showed significantly higher mean MFCV compared with that of control subjects. Comparing NEMG to SEMG determination of MFCV in carriers of hypokalemic periodic paralysis, asymptomatic first-degree relatives, and control subjects, the mean SEMG MFCV in carriers was significantly lower than in control subjects.[15] However, in 7 of 22 carriers with attacks and in 3 of 11 carriers without attacks, SEMG MFCV values were in the low normal range. The NEMG method showed MFCV disturbance in all proved carriers, implying a greater sensitivity than SEMG.

Thus, these studies, in general, provide clear Class II evidence in favor of NEMG in preference to SEMG in the evaluation of patients with specific disturbances of neuromuscular function.

We conclude that SEMG is substantially inferior to NEMG for the evaluation of patients with neuromuscular disorders. Some of the most important diagnostic NEMG measurements such as insertional activity, spontaneous activity, motor unit size and shape, and interference pattern have not been or cannot be reliably measured with SEMG. Furthermore, SEMG has limited spatial resolution, is more susceptible to mechanical artifact, and is more likely to show cross-talk between adjacent muscles than NEMG. Therefore, based on Class II data, SEMG is considered unacceptable as a clinical tool in the diagnosis of neuromuscular disease at this time (Type E recommendation).

Low back pain assessment. The presumed association between low back pain and muscle fatigue provides the rationale for studying pain with SEMG. Nevertheless, the actual association between pain and fatigue has been difficult to establish. Moreover, the mechanisms of low back pain are not clearly understood, although excessive fatigue due to muscle deconditioning, inhibition of muscle activation secondary to pain, and pain-related action have been suggested as possible causes that might be addressed using SEMG.[16]

During sustained muscle contraction, SEMG signals undergo a spectral shift to lower frequencies. This spectral shift has been suggested, but not convincingly established, to provide an index of muscle fatigue.[17-24] It is not known whether there are other causes of similar spectral shifts.[25,26] Some spectral parameters such as the mean, mode, and median frequency are known to decrease continuously after the onset of muscle contraction. It may, therefore, be possible to monitor the fatiguing process early in contraction before the point of mechanical failure has been reached.

Twelve patients with a history of chronic low back pain (average duration, 15.2 years) were compared to 12 control subjects in a two-group, stepwise discriminant analysis using the median spectral frequency of muscle contraction.[27] Spectral frequency was determined at three contraction force levels: 40%, 60%, and 80% of maximal voluntary contraction (MVC). At 40% MVC, the discriminant analysis correctly classified 92% of the low back pain group and 82% of the control group. At 80% MVC, analysis correctly classified 84% of the low back pain group and 91% of control subjects. At 60% MVC, however, classification was poor (67% in the control group, 75% in the low back pain group). In addition, this particular discriminant function was not verified on an independent sample of patients and controls. Such inconclusive or incomplete findings are characteristic of most studies in this area, possibly related to external factors such as motivation bias, body movement, and electrode placement, each of which can adversely affect these measurements.

In an attempt to address some of these issues, 27 patients with chronic low back pain of more than 6 months were divided into "avoiders" (i.e., those who reduced physical and social activities to cope with the pain) and "confronters" (i.e., those who remained active despite their back pain) and compared to 22 control subjects.[28] Discriminant analysis correctly classified 88.9% of the avoiders but did less well with confronters. The relevance of these findings to clinical issues, however, is unclear and, again, the findings were not replicated in an independent sample.

Among rowers with and without low back pain, discriminant analysis was able to correctly classify all 6 patients and 14 (93%) of 15 without low back pain.[29] Similarly, when the same methodology is applied, 25 rowers were examined: 8 with and 17 without low back pain.[30] The percentage of recovery in the median spectral frequency at 1 minute and at 2 minutes after a 30-second contraction (80% MVC) was applied to a discriminant analysis, which correctly classified from 88% to 100% of both groups. More important, however, is that the similarity of the discriminant functions used in these two studies is not known, so these two studies cannot be considered replications of each other.

In an attempt to correlate pain with changes in SEMG spectral frequency, 403 nurses without any serious low back pain history were prospectively evaluated.[31] At baseline, spectral parameters were measured during a 28-second muscle contraction at 80% MVC. A decline in the median SEMG spectral frequency was associated with a greater probability of subjects having low back pain develop in the future. In a related study,[32] mixed results were found regarding the reliability of SEMG spectral parameters. In this study, muscle function of the multifidus and iliocostalis was evaluated in the prone position (trunk holding test) in 12 normal subjects. Two trials were performed in two testing sessions over 3 days. Pearson's product moment correlation coefficients, *t*-tests for paired data, analysis of variance of intrasubject coefficient of variation, and intraclass coefficient correlation were used as reliability measures of the initial median frequency and median frequency slope. Within-day reliability and between-days reliability of the initial median frequency recorded in the multifidus and iliocostalis were good (Pearson's $r = 0.74$ to 0.94), but median frequency slope reliability measurements were less stable compared with the initial median frequency (Pearson's $r = 0.39$ to 0.55). These findings imply that the basis for SEMG determination of low back pain may not be reliable.

In addition, several considerations make the reported SEMG findings in low back pain of doubtful clinical value. First, although muscle fatigue is thought to be related to the development of low back pain and is associated with changes in SEMG spectral frequency, the relationship between the two is uncertain. Second, it is unclear what other factors may influence spectral frequency, making the specificity of the SEMG findings in this clinical setting unclear. Third, many of the reports use discriminant functions based on case-control studies, which have not been verified on independent samples of patients and control subjects. Fourth, the actual discriminant functions used have differed between reports. Fifth and finally, even if the reports are accepted at face value, the findings suggest only that SEMG can identify patients who have low back pain. Presumably, the gold standard is the clinical history and, in this circumstance, it would be easier and cheaper simply to ask the patient whether his or her back hurts (unless the patients are malingering and one wishes to determine whether the patient truly has low back pain). A more useful clinical application of this technique would be to distinguish patients with nerve root compression syndromes from those with back pain due to other causes, but this question has not been addressed by the studies to date.

In summary, based on Class III and inconclusive or inadequate Class II data, SEMG is considered unacceptable as a clinical tool in the evaluation of patients with low back pain at this time (Type E recommendation).

Kinesiology and disorders of motor control. There are several applications of SEMG in which this technique is considered standard. For example, the use of SEMG recordings is routinely used to measure nerve conduction velocities after electrical stimulation of a peripheral nerve.[2] Similarly, SEMG is the standard for recording compound muscle action potentials after magnetic stimulation either transcranially or peripherally. SEMG has been used for decades as a technique for studying human motion,[22,33-43] for recording EMG signals from multiple muscles in other clinical settings, and for monitoring response times in experimental circumstances.[38,44-53] Indeed, because of the noninvasive and painless nature of the method, this should be considered a standard application of SEMG (often superior to either NEMG or FWEMG), although the precise clinical utility of such recordings in these latter circumstances remains to be defined.[22,35] Few articles in this area critically compare SEMG with other methods of recording muscle activity and rarely is a gold standard (e.g., NEMG, imaging studies, or muscle biopsies) identified. The reason for this is twofold. First, there is no adequate gold

standard for movement analyses. Second, the technique of SEMG is not usually in question but merely used as a tool within the scope of a larger testing goal.[33,35,53-56]

The neurophysiologic analysis of movement disorders, particularly tremor, myoclonus, dystonia, and dyskinesia, typically is studied using SEMG rather than NEMG or FWEMG. An important reason for this is that the mean rectified SEMG signal, as opposed to the NEMG or FWEMG signal, varies linearly with the force generated at constant length[57-59] as well as during constant velocity contractions.[60] This linear relationship remains true even in fatigued or diseased muscle,[61,62] thus facilitating interpretation of SEMG data as they relate to muscle force generation. Another important advantage to SEMG in this setting is that it allows prolonged recordings of muscle activity from multiple sites simultaneously.

Surface electromyography may be used to classify movement disorders through measurement of frequency and amplitude of muscle activity, and its relationship to separately recorded limb or truncal movement or force. This is based on Class III evidence as most reports are formulated from expert opinion, nonrandomized historical control subjects, and observations from case series. These Class III studies show that many tremor disorders reveal distinct muscle activity patterns (e.g., orthostatic tremor[56,63]) such that SEMG data can be helpful diagnostically. SEMG can provide information about motor unit recruitment and synchronization with the tremor activity[64,65] and can also determine the relationship of involved muscles to tremor movements and reveal whether antagonists (such as wrist flexors and extensors) discharge simultaneously or alternately to produce the tremor. Differentiating tremor from myoclonus,[56] spasmodic torticollis from other head tremors,[66] and primary writing tremor from writer's cramp[67] and identifying speed of spread of muscle activity[68] and origin of muscle activity in propriospinal myoclonus[68] are other potentially important clinical applications of SEMG. SEMG is also useful in the analyses of movement disorders in which prolonged recordings must be pain-free and interfere minimally with the clinical phenomenology.[69]

Rhythmic EMG signals containing bursts of activity, as in chewing,[70-72] walking,[22,73-75] and breathing,[76-81] can be analyzed using SEMG and automated burst detection methods. These have an advantage in that large amounts of SEMG data can be processed easily and objectively.[71] Multiple cycles of movement may be recorded and averaged patterns of muscle activation and joint movements determined. Psychophysical measurements, such as movement and reaction time analysis,[38,44-48] requiring precise timing of muscle contraction onset benefit from SEMG as a noninvasive tool for this purpose. Without SEMG, painful intramuscular insertion of an NEMG or FWEMG electrode would be required to determine the onset of movement, adversely interfering with the psychophysical measurements under analysis.

In summary, based on Class III evidence, SEMG is considered an acceptable tool for kinesiologic analysis of movement disorders because it is a method for recording and quantifying clinically important muscle-related activity with the least interference on the clinical picture. SEMG may also be useful in differentiating the many types of tremors, myoclonus, and dystonia; for evaluating gait and posture; and for evaluating psychophysical measurements of reaction and movement time (Type C recommendation).

Conclusions

1. Based on Class II data, SEMG is considered unacceptable as a clinical tool in the diagnosis of neuromuscular disease at this time (Type E recommendation).
2. Based on Class III data and inconclusive or inadequate Class II data, SEMG is considered unacceptable as a clinical tool in the diagnosis of low back pain at this time (Type E recommendation).
3. Based on Class III data, SEMG is considered an acceptable tool for kinesiologic analysis of movement disorders; for differentiating types of tremors, myoclonus, and dystonia; for evaluating gait and posture disturbances; and for evaluating psychophysical measures of reaction and movement time (Type C recommendation).

Further studies comparing specificity and sensitivity of FWEMG with SEMG are to be encouraged.

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Geriatric Neurology Section, Government Service Section, Neurogenetics Section, Neuromuscular Section, Pain Medicine Section, and the Section on Women's Issues.

Appendix 1

American Academy of Neurology Therapeutics and Technology Assessment Subcommittee members: Douglas S. Goodin, MD (Chair); Elliot Mark Frohman, MD, PhD; Robert Goldman, MD; John Ferguson, MD; Philip B. Gorelick, MD, MPH; Chung Hsu, MD, PhD; Andres Kanner, MD; Anne Marini, MD, PhD; Carmel Armon, MD; David Hammond, MD; David Lefkowitz, MD; and Edward Westbrook, MD.

Appendix 2

Quality of evidence ratings

Class I. Evidence provided by one or more well-designed clinical studies of a diverse population using a "gold standard" reference test in a blinded evaluation appropriate for the proposed diagnostic application.

Class II. Evidence provided by one or more clinical studies of a restricted population using a reference test in a blinded evaluation of diagnostic accuracy.

Class III. Evidence provided by expert opinion, nonrandomized historical controls, or observation(s) from case series.

Definitions

Safe. A judgment of the acceptability of risk in a specified situation, e.g., for a given medical problem, by a provider with specified training, at a specified type of facility.

Effective. Producing a desired effect under conditions of actual use.

Established. Accepted as appropriate by the practicing medical community for the given indication in the specified patient population.

Possibly useful. Given current knowledge, this technology appears to be appropriate for the given indication in the specified patient population. If more experience and long-term follow-up are accumulated, this interim rating may change.

Investigational. Evidence insufficient to determine appropriateness, warrants further study. Use of this technology for given indication in the specified patient population should be confined largely to research protocols.

Doubtful. Given current knowledge, this technology appears to be inappropriate for the given indication in the specified patient population. If more experience and long-term follow-up are accumulated, this interim rating may change.

Unacceptable. Regarded by the practicing medical community as inappropriate for the given indication in the specified patient population.

Suggested strength of recommendations

Type A. Strong positive recommendations, based on Class I evidence, or overwhelming Class II evidence when circumstances preclude randomized clinical trials.

Type B. Positive recommendation, based on Class II evidence.

Type C. Positive recommendation, based on strong consensus of Class III evidence.

Type D. Negative recommendation, based on inconclusive or conflicting Class II evidence.

Type E. Negative recommendation, based on evidence of ineffectiveness or lack of efficacy, based on Class II or Class I evidence.

Type O. Insufficient data to make a recommendation.

References

1. Haig AJ, Gelblum JB, Rechtien JJ, Gitter AJ. Technology assessment: the use of surface EMG in the diagnosis and treatment of nerve and muscle disorders. Muscle Nerve 1996;19:392–395.
2. Kimura J. Electrodiagnosis in diseases of nerve and muscle: principles and practice. Philadelphia: F.A. Davis, 1990.
3. Turker KS. Electromyography: some methodological problems and issues. Phys Ther 1993;73:698–710.
4. Fuglevand AJ, Winter DA, Patla AE, Stashuk D. Detection of motor unit action potentials with surface electrodes: influence of electrode size and spacing. Biol Cybern 1992;67:143–153.
5. Wilbourn AJ, Aminoff MJ. AAEE Minimonograph #32: the electrophysiologic examination in patients with radiculopathies. Muscle Nerve 1988;11:1099–1114.
6. Howard RS, Murray NM. Surface EMG in the recording of fasciculations. Muscle Nerve 1992;15:1240–1245.
7. Hjorth RJ, Walsh JC, Willison RG. The distribution and frequency of spontaneous fasciculations in motor neuron disease. J Neurol Sci 1973;18:469–474.
8. Barry DT, Gordon KE, Hinton GG. Acoustic and surface EMG diagnosis of pediatric muscle disease. Muscle Nerve 1990;13:286–290.
9. Orizio C, Esposito F, Sansone V, Parrinello G, Meola G, Veicsteinas A. Muscle surface mechanical and electrical activities in myotonic dystrophy. Electromyogr Clin Neurophysiol 1997;37:231–239.
10. Lagueny A, Marthan R, Schuermans P, Le Collen P, Ferrer X, Julien J. Single fiber EMG and spectral analysis of surface EMG in myotonia congenita with or without transient weakness. Muscle Nerve 1994;17:248–250.
11. Priez A, Duchene J, Goubel F. Duchenne muscular dystrophy quantification: a multivariate analysis of surface EMG. Med Biol Eng Comput 1992;30:283–291.
12. Preece AW, Wimalaratna HS, Green JL, Churchill E, Morgan HM. Non-invasive quantitative EMG. Electromyogr Clin Neurophysiol 1994;34:81–86.
13. Huppertz HJ, Disselhorst-Klug C, Silny J, Rau G, Heimann G. Diagnostic yield of noninvasive high spatial resolution electromyography in neuromuscular diseases. Muscle Nerve 1997;20:1360–1370.

14. van der Hoeven JH, Zwarts MJ, Van Weerden TW. Muscle fiber conduction velocity in amyotrophic lateral sclerosis and traumatic lesions of the plexus brachialis. Electroencephalogr Clin Neurophysiol 1993;89:304–310.
15. van der Hoeven JH, Links TP, Zwarts MJ, van Weerden TW. Muscle fiber conduction velocity in the diagnosis of familial hypokalemic periodic paralysis—invasive versus surface determination. Muscle Nerve 1994;17:898–905.
16. Roy SH, Oddsson LI. Classification of paraspinal muscle impairments by surface electromyography. Phys Ther 1998;78:838–851.
17. Bazzy AR, Korten JB, Haddad GG. Increase in electromyogram low-frequency power in nonfatigued contracting skeletal muscle. J Appl Physiol 1986;61:1012–1017.
18. Krivickas LS, Taylor A, Maniar RM, Mascha E, Reisman SS. Is spectral analysis of the surface electromyographic signal a clinically useful tool for evaluation of skeletal muscle fatigue? J Clin Neurophysiol 1998;15:138–145.
19. Bigland-Ritchie B, Donovan EF, Roussos CS. Conduction velocity and EMG power spectrum changes in fatigue of sustained maximal efforts. J Appl Physiol 1981;51:1300–1305.
20. Bigland-Ritchie B, Cafarelli E, Vollestad NK. Fatigue of submaximal static contractions. Acta Physiol Scand Suppl 1986;556:137–148.
21. Arendt-Nielsen L, Mills KR. The relationship between mean power frequency of the EMG spectrum and muscle fibre conduction velocity. Electroencephalogr Clin Neurophysiol 1985;60:130–134.
22. Basmajian JV. Electromyographic analyses of basic movement patterns. Exerc Sport Sci Rev 1973;1:259–284.
23. Capodaglio P, Nilsson J. Functional correlates in the rehabilitation of occupational low back pain. G Ital Med Lav 1996;18:35–39.
24. Lind AR, Petrofsky JS. Amplitude of the surface electromyogram during fatiguing isometric contractions. Muscle Nerve 1979;2:257–264.
25. Hof AL. Errors in frequency parameters of EMG power spectra. IEEE Trans Biomed Eng 1991;38:1077–1088.
26. Braakhekke JP, Stegeman DF, Joosten EM. Increase in median power frequency of the myoelectric signal in pathological fatigue. Electroencephalogr Clin Neurophysiol 1989;73:151–156.
27. Roy SH, De Luca CJ, Casavant DA. Lumbar muscle fatigue and chronic lower back pain. Spine 1989;14:992–1001.
28. Biedermann HJ, Shanks GL, Forrest WJ, Inglis J. Power spectrum analyses of electromyographic activity. Discriminators in the differential assessment of patients with chronic low-back pain. Spine 1991;16:1179–1184.
29. Roy SH, De Luca CJ, Snyder-Mackler L, Emley MS, Crenshaw RL, Lyons JP. Fatigue, recovery, and low back pain in varsity rowers. Med Sci Sports Exerc 1990;22:463–469.
30. Klein AB. Comparison of spinal mobility and isometric trunk extensor forces with electromyographic spectral analysis in identifying low back pain. Phys Ther 1991;1:445–454.
31. Mannion AF, Connolly B, Wood K, Dolan P. The use of surface EMG power spectral analysis in the evaluation of back muscle function. J Rehabil Res Dev 1997;34:427–439.
32. Ng JK, Richardson CA, Jull GA. Electromyographic amplitude and frequency changes in the iliocostalis lumborum and multifidus muscles during a trunk holding test. Phys Ther 1997;77:954–961.
33. Hallett M, Berardelli A, Delwaide P, et al. Central EMG and tests of motor control. Report of an IFCN committee. Electroencephalogr Clin Neurophysiol 1994;90:404–432.
34. Hermens HJ, Boon KL, Zilvold G. The clinical use of surface EMG. Acta Belge Med Phys 1986;9:119–130.
35. Hallett M. Analysis of abnormal voluntary and involuntary movements with surface electromyography. Adv Neurol 1983;39:907–914.
36. Giroux C, Maton B. Surface EMG and motor unit activity of partially denervated human muscle during fatiguing submaximal isometric contraction. Electromyogr Clin Neurophysiol 1990;30:283–291.
37. Cohen LG, Hallett M. Hand cramps: clinical features and electromyographic patterns in a focal dystonia. Neurology 1988;38:1005–1012.
38. Benecke R, Rothwell JC, Dick JP, Day BL, Marsden CD. Disturbance of sequential movements in patients with Parkinson's disease. Brain 1987;110:361–379.
39. De Luca CJ. Use of the surface EMG signal for performance evaluation of back muscles. Muscle Nerve 1993;16:210–216.
40. Young RR, Cracco RQ. Clinical neurophysiology of conduction in central motor pathways. Ann Neurol 1985;18:606–610.
41. Valls-Sole J, Hallett M. Modulation of electromyographic activity of wrist flexor and extensor muscles in patients with writer's cramp. Mov Disord 1995;10:741–748.
42. Shahani BT, Fang J, Dhand UK. A new approach to motor unit estimation with surface EMG triggered averaging technique. Muscle Nerve 1995;18:1088–1092.
43. Narabayashi H, Nagahata M, Nagao T, Shimazu H. A new classification of cerebral palsy based upon neurophysiologic considerations. Confin Neurol 1965;25:378–392.
44. Goodin DS, Aminoff MJ. The relationship between the evoked potential and brain events in sensory discrimination and motor response. Brain 1984;107:241–251.
45. Sanes JN. Information processing deficits in Parkinson's disease during movement. Neuropsychologia 1985;23:381–392.
46. Rafal RD, Inhoff AW, Friedman JH, Bernstein E. Programming and execution of sequential movements in Parkinson's disease. J Neurol Neurosurg Psychiatry 1987;50:1267–1273.
47. Pullman SL, Watts RL, Juncos JL, Chase TN, Sanes JN. Dopaminergic effects on simple and choice reaction time performance in Parkinson's disease. Neurology 1988;38:249–254.
48. Goodin DS, Aminoff MJ. Event-related potentials and their relationship to discrimination and response in simple and choice reaction tasks. J Clin Neurophysiol 1998;15:34–43.
49. Botzel K, Mayer M, Oertel WH, Paulus W. Frontal and parietal premovement slow brain potentials in Parkinson's disease and aging. Mov Disord 1995;10:85–91.
50. Daum I, Quinn N. Reaction times and visuospatial processing in Parkinson's disease. J Clin Exp Neuropsychol 1991;13:972–982.

51. Evarts EV, Teravainen H, Calne DB. Reaction time in Parkinson's disease. Brain 1981;104:167–186.
52. Jordan N, Sagar HJ, Cooper JA. Cognitive components of reaction time in Parkinson's disease. J Neurol Neurosurg Psychiatry 1992;55:658–664.
53. Heilman KM, Bowers D, Watson RT, Greer M. Reaction times in Parkinsonís disease. Arch Neurol 1976;33:139–140.
54. Tolosa E, Berciano J. Choreas, hereditary and other ataxias, tics, myoclonus, and other movement disorders. Curr Opin Neurol Neurosurg 1993;6:358–368.
55. Elble RJ, Randall JE. Motor-unit activity responsible for 8- to 12-Hz component of human physiological finger tremor. J Neurophysiol 1976;39:370–383.
56. Deuschl G, Krack P, Lauk M, Timmer J. Clinical neurophysiology of tremor. J Clin Neurophysiol 1996;13:110–121.
57. Milner-Brown HS, Stein RB. The relation between the surface electromyogram and muscular force. J Physiol (Lond) 1975;246:549–569.
58. Inman VT, Ralston HJ, Saunders JB, Feinstein B, Wright EWJ. Relation of human electromyogram to muscular tension. EEG Clin Neurophysiol 1952;4:187–194.
59. Lippold OCJ. The relation between integrated action potentials in a human muscle and its isometric tension. J Physiol 1952;1:117.1:492–499.
60. Bigland B, Lippold OCJ. The relation between force, velocity, and integrated electrical activity in human muscles. J Physiol 1954;123:214–224.
61. Lenman JAR. A clinical and experimental study of the effects of exercise on motor weakness in neurological disease. J Neurol Neurosurg Psychiatry 1959;22:192–194.
62. Edwards RG, Lippold OCJ. The relationship between force and integrated electrical activity in fatigued muscle. J Physiol 1956;132:677–681.
63. McManis PG, Sharbrough FW. Orthostatic tremor: clinical and electrophysiologic characteristics. Muscle Nerve 1993;16:1254–1260.
64. Elble RJ, Koller WC. Tremor. Baltimore: The Johns Hopkins University Press, 1990.
65. Elble RJ. Physiologic and essential tremor. Neurology 1986;36:225–231.
66. Deuschl G, Heinen F, Kleedorfer B, Wagner M, Lucking CH, Poewe W. Clinical and polymyographic investigation of spasmodic torticollis. J Neurol 1992;239:9–15.
67. Bain PG, Findley LJ, Britton TC, et al. Primary writing tremor. Brain 1995;118:1461–1472.
68. Fouillet N, Wiart L, Arne P, Alaoui P, Petit H, Barat M. Propriospinal myoclonus in tetraplegic patients: clinical, electrophysiological and therapeutic aspects. Paraplegia 1995;33:678–681.
69. Watts RL, Pullman SL, Glatt SL, Koller WC. Quantitative techniques of assessing motor disability. In: Koller WC, Paulson G, eds. Therapy of Parkinson's disease. New York: Marcel Dekker, Inc., 1994:47–75.
70. Ottenhoff FA, van der Bilt A, van der Glas HW, Bosman F, Abbink JH. The relationship between jaw elevator muscle surface electromyogram and simulated food resistance during dynamic condition in humans. J Oral Rehabil 1996;23:270–279.
71. Abbink JH, van der Bilt A, van der Glas HW. Detection of onset and termination of muscle activity in surface electromyograms. J Oral Rehabil 1998;25:365–369.
72. Owall B, Elmqvist D. Motor pauses in EMG activity during chewing and biting. Odontol Revy 1975;26:17–38.
73. Ericson MO, Nisell R, Ekholm J. Quantified electromyography of lower-limb muscles during level walking. Scand J Rehabil Med 1986;18:159–163.
74. Malinauskas M, Krouskop TA. Gait analysis measurement techniques. Phys Rehabil Med 1989;1:23–36.
75. Shiavi R, Bugle HJ, Limbird T. Electromyographic gait assessment, Part 1: adult EMG profiles and walking speed. J Rehabil Res Dev 1987;24:13–23.
76. Bolton CF. AAEM minimonograph #40: clinical neurophysiology of the respiratory system. Muscle Nerve 1993;16:809–818.
77. De Troyer A, Heilporn A. Respiratory mechanics in quadriplegia. The respiratory function of the intercostal muscles. Am Rev Respir Dis 1980;122:591–600.
78. Gross D, Grassino A, Ross WR, Macklem PT. Electromyogram pattern of diaphragmatic fatigue. J Appl Physiol 1979;46:1–7.
79. Jeffries B, Brouillette RT, Hunt CE. Electromyographic study of some accessory muscles of respiration in children with obstructive sleep apnea. Am Rev Respir Dis 1984;129:696–702.
80. Schloon H, O'Brien MJ, Scholten CA, Prechtl HF. Muscle activity and postural behaviour in newborn infants. A polymyographic study. Neuropediatrie 1976;7:384–415.
81. White JE, Drinnan MJ, Smithson AJ, Griffiths CJ, Gibson GJ. Respiratory muscle activity during rapid eye movement (REM) sleep in patients with chronic obstructive pulmonary disease. Thorax 1995;50:376–382.

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