AANEM PRACTICE TOPIC

ABSTRACT: Surface electromyography (sEMG) measures myoelectrical signals recorded from sensors placed on the skin surface. The non-invasive nature of sEMG makes it a potentially useful technology for studying diseases of muscle and nerve. Reviews published by the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) and the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology (AAN), covering 1964–1994 and 1952–1998, respectively, concluded that sEMG adds no clinical utility over conventional needle EMG (nEMG) for the diagnosis of neuromuscular disease. The AANEM sEMG task force reevaluated the diagnostic utility and added value of this technology for the study of neuromuscular disease based on a contemporary review of relevant literature published between January 1994 and February 2006. The present review concludes that sEMG may be useful to detect the presence of neuromuscular disease (level C rating, class III data), but there are insufficient data to support its utility for distinguishing between neuropathic and myopathic conditions or for the diagnosis of specific neuromuscular diseases. sEMG may be useful for additional study of fatigue associated with post-poliomyelitis syndrome and electromechanical function in myotonic dystrophy (level C rating, class III data).
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AMERICAN ASSOCIATION OF NEUROMUSCULAR & ELECTRODIAGNOSTIC MEDICINE EVIDENCED-BASED REVIEW: USE OF SURFACE ELECTROMYOGRAPHY IN THE DIAGNOSIS AND STUDY OF NEUROMUSCULAR DISORDERS

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In the last two decades, novel surface electromyography (sEMG) technologies have increasingly been studied as a complement or potential alternative to needle electromyography (nEMG) and nerve conduction studies (NCS) for the investigation of neuromuscular disorders. The sEMG recording techniques vary significantly, but all involve analysis of myoelectrical signals using sensors positioned on the skin surface. In each case, a sensor or sensors are placed on the muscle of interest, and the voltage differences between the electrodes are measured during muscle activation. Compared with conventional nEMG, sEMG is non-invasive and has the theoretical advantage of a larger recording area, making possible the collection of data over a wider region of muscle or even over an entire muscle.

The most basic sEMG recordings include single-channel monopolar and bipolar montages. Single-channel monopolar recordings measure the voltage difference between a recording electrode over muscle and a remote area, whereas bipolar montages measure between two recording electrodes in close proximity over an individual muscle. In both types, myoelectrical activity is recorded, and waveforms are typically analyzed with the aid of specialized com-

Abbreviations: ADP, acquired demyelinating polyneuropathy; ALS, amyotrophic lateral sclerosis; CMT, Charcot–Marie–Tooth; DM, myotonic dystrophy; HD, high-density; HOPP, hypokalemic periodic paralysis; MC, multichannel; MPF, median power frequency; NCS, nerve conduction studies; nEMG, needle electromyography; PPS, post-poliomyelitis syndrome; RMS, root mean square; sEMG, surface electromyography; TCR, trigemino-cervical response; V/E, voluntary/elicited

Key words: electromyography; hypokalemic periodic paralysis; myopathy; myotonia; neuronopathy; neuropathy; surface EMG

Disclosure: This statement is provided as an educational service of the American Association of Neuromuscular & Electrodiagnostic Medicine (AANEM). It is based on an assessment of current scientific and clinical information. It is not intended to include all possible proper methods of care for a particular neurologic problem or all legitimate criteria for choosing to use specific procedures. Neither is it intended to exclude any reasonable alternative methodologies. The AANEM recognizes that specific patient care decisions are the prerogative of the patient and the physician caring for the patient, based on all the circumstances involved.

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puter software. The sEMG data derived in this manner may include waveform measurements of voltage, amplitude, frequency, numbers of turns or baseline crossings, and the relationship of these parameters to force and duration of muscle contraction.

Multichannel sEMG electrodes in more complex arrangements allow for signal analysis from multiple spatial perspectives. As the number of electrodes increases, the overall area of muscle under study may increase, while interelectrode distance and recording area may decrease. This result is suppression of far-field activity and enhanced resolution of individual motor unit or myofiber potentials. Examples of multichannel sEMG arrangements include double-differential montage of three linearly arranged recording electrodes, Laplacian montages of five recording electrodes arranged in a cross pattern, and high-density sEMG (HD-sEMG) involving multiple recording electrodes in a grid pattern. An analysis of muscle fiber conduction velocity, dwell time over root mean square, autocorrelation function, chi-value characteristics, and other parameters can be used to derive information about nerve and muscle function.

The complex nature of sEMG signals and the many variables that influence their quality have resulted in questions regarding the utility of sEMG as a tool to assess neuromuscular disease and its current applicability in routine clinical practice. A literature review conducted by the AANEM found no clinical indication for the use of sEMG in the diagnosis and treatment of neuromuscular disease.[10] A review by the American Academy of Neurology (AAN) also concluded that sEMG is substantially inferior to nEMG for evaluation of patients with neuromuscular disorders.[15] To evaluate further the current clinical utility of sEMG in the diagnosis and study of neuromuscular disorders, the AANEM Board of Directors charged an AANEM task force to conduct a new search to determine whether further research had been published since the last review. This evidence-based report discusses the application of sEMG for the diagnosis and study of nerve and muscle disorders, specifically for neuronopathies, radiculopathies, peripheral neuropathies, plexopathies, neuromuscular junction disease, and myopathies. Fatigue and pain syndromes unrelated to nerve or muscle damage and other neurological disorders involving central nervous system dysfunction were not reviewed.

METHODS

Using Medline and PubMed electronic databases, the members of the task force conducted a systematic literature search covering the period from January 1994 to February 2006. This period spanned the interval since the previous AANEM review of this topic.[10] Studies were chosen for review based on their relevance to sEMG in the diagnosis and treatment of nerve and muscle disorders. Searches were conducted using the key word "surface EMG," cross-referenced with the following terms: electromyography, needle electromyography, neuronopathy, radiculopathy, plexopathy, peripheral neuropathy, neuromuscular junction disorder, myopathy, nervous system, frequency analysis, and spectral analysis. No language or study design limitations were placed on the searches.

There was overlap among the searches conducted, which resulted in the identification of 5682 abstracts. Each abstract was reviewed by at least two task force members. Of these, 53 references were retrieved in their entirety. These articles were reviewed in detail by each member of the task force. Twenty-one of the 53 articles involved novel research pertinent to this review and were evaluated for information addressing the clinical diagnostic utility and the additional value of sEMG in the study of nerve and muscle disorders.

The review of the diagnostic utility of sEMG encompassed the following five questions:

1. Can sEMG distinguish between normal individuals and those with neuromuscular disease?
2. Can sEMG distinguish between patients with nerve disease and those with muscle disease?
3. Can sEMG diagnose specific neuromuscular disorders?
4. What is the threshold of neuromuscular disease severity detectable by sEMG?
5. How does sEMG compare with NCS, nEMG, and other tests in its ability to detect and distinguish between nerve and muscle disease?

A review of the additional value of sEMG focused on whether sEMG provides information about neuromuscular disease progression or pathophysiology that is not usually obtained by standard NCS and nEMG techniques. This evidenced-based review does not address the use of sEMG to distinguish between central and peripheral nervous system disorders.

The strength of evidence for each article was rated according to the AAN classification for diagnostic articles (Table 1).[9] No class I or class II studies were identified in the articles identified and reviewed by the task force members. Studies were considered for a class III level of evidence if the collected sEMG data were analyzed objectively off-line. Studies addressing the diagnostic utility of sEMG in

Table 1. Classification of level of evidence.

Class I:
- Prospective study in broad spectrum of persons with suspected disease
- All patients undergoing diagnostic test have presence or absence of disease determined by blinded examiner
- Gold standard comparison

Class II:
- Prospective study of narrow spectrum of persons with suspected condition OR
- Well-designed retrospective study of a broad spectrum of persons with an established condition
- Blinded examiners
- Gold standard comparison

Class III:
- Retrospective study with narrow spectrum either of persons with established condition or of controls
- If diagnostic test and reference standard not objective, independent examiners must apply diagnostic test and reference standard

Class IV:
- Any design where test is not applied in an independent evaluation, OR
- Evidence provided by expert opinion alone, OR
- Descriptive case series without controls

Adapted from the American Academy of Neurology Clinical Practice Guideline Process Manual (2004).

neuromuscular disorders and providing class III evidence or higher were included in formulation of consensus statements (Table 2). Class IV diagnostic utility studies were reviewed but they do not add significantly to formulation of the evidence-based conclusions and therefore are not discussed in this review.[2,4,16] All classes of evidence from studies that demonstrated added value of sEMG in the investigation of neuromuscular pathophysiology were included in this review. These articles may be of interest to those physicians looking at the potential applications of sEMG to collect neuromuscular clinical and research data not routinely obtained by conventional NCS and nEMG.

RESULTS

Clinical Utility of sEMG in Diagnosis of Neuromuscular Disorders. Three class III studies evaluated the utility of sEMG for detection of primary neuropathic and myopathic diseases. Monopolar sEMG was reported to have a lower sensitivity and specificity for detection of pathological fasciculations (57% and 90%, respectively) than muscle sonography (63% and 93%, respectively) in a neuromuscular disease cohort (inflammatory muscle disease, lower motor neuron disease, hereditary motor and sensory neuropathy, and adrenomyeloneuropathy) compared with healthy control subjects.[21] A bipolar recording montage was able to separate healthy control subjects from patients with myopathy and axonal peripheral neuropathy through observable changes in median power frequency (MPF) and the number of baseline crossings. The bipolar recording montage was unable to differentiate neurogenic from myopathic disorders in the disease group, whereas concomitant nEMG distinguished between control subjects and patients with neuromuscular disease as well as between neurogenic and myopathic conditions.[22] Multielectrode array sEMG was able to distinguish between myopathic and neuropathic disorders (Duchenne and Becker muscular dystrophy, spinal muscular atrophy, and hereditary motor and sensory neuropathy) and healthy control subjects with sensitivities and specificities of detecting neuromuscular disease (82% and 97%, respectively), primary myopathic disorders (85% and 97%, respectively), and primary neuropathic disorders (68% and 98%, respectively), comparable to historical results for conventional nEMG.[11]

Four class III studies evaluated the utility of sEMG for detection of myoelectric signal abnormalities in the more specific neuromuscular disorders of acquired demyelinating peripheral neuropathy (ADP), amyotrophic lateral sclerosis (ALS), hypokalemic periodic paralysis (HOPP), and post-poliomyelitis syndrome (PPS). Single-channel monopolar sEMG study detected proximal conduction block in ADP through comparison of maximal voluntarily contracted muscle sEMG amplitude with electrically stimulated distal compound muscle action potential amplitude (V/E ratio). The mean V/E ratio in the ADP group was statistically reduced compared with the diseased (ALS) and healthy control groups.[1] Monopolar recording of trigemino-cervical responses (TCR) identified 78% of patients with ALS by absent, delayed, or abnormally asymmetrical TCR latencies. Healthy control subjects had significantly shorter TCR latencies.[23]

Multiple channel studies included a double-differential technique to measure muscle fiber conduction velocity in a family with HOPP. The mean muscle fiber conduction velocity was slowed in carriers compared with healthy control subjects, as measured by both surface and needle techniques (sensitivity 70% and 100%, respectively).[20] The HD-sEMG study to detect neurogenic changes in PPS compared with control subjects showed an increase in the averaged motor unit action potential size (Table 2).[6]

Table 2. Evidence of diagnostic utility of sEMG in neuromuscular disease.

Reference (sEMG technique)	Number of subjects		Diagnostic reference standards	Outcome measures	Conclusion
	Disease	Control			
Can sEMG distinguish between patients with neuromuscular disease and control subjects?					
11 (MC)	72	61	nEMG, muscle biopsy, DNA analysis	Combination: dwell time over RMS, MCV, chi-value of frequency–amplitude distribution, ACF	Sensitivity 82%, specificity 97%
22 (BP)	20	10	nEMG, clinical examination, muscle biopsy	MPF, ZX	MPF: disease < control (P = 0.002); ZX: disease < control (P = 0.003)
Can sEMG distinguish between patients with nerve (SMA, HMSN) and muscle disease (DMD, BMD)?					
11 (MC)	72	61	nEMG, muscle biopsy, DNA analysis	Combination: dwell time over RMS, MCV, chi-value of frequency–amplitude distribution, ACF	Nerve disease: sensitivity 68%, specificity 98% Muscle disease: sensitivity 85%, specificity 97%
Can sEMG distinguish between patients with PPS and control subjects?					
6 (MC)	9	9	Halstead criteria	MU mean area	PPS > control (P = 0.0001); sensitivity 100%, specificity 100%
Can sEMG distinguish between patients with ADP and control subjects?					
1 (MP)	25	14 disease (stroke, ALS) 11 healthy	NCS and nEMG	V/E CMAP ratio	ADP < healthy controls, stroke or unaffected limb in ADP, ALS (P < 0.05)
Can sEMG distinguish between ALS and control subjects?					
23 (MP)	45	100	Arlie House criteria (35 definite, 10 probable ALS)	P20, N30 TCR latency	ALS > control (P < 0.05); sensitivity 78%, specificity 100%
Can sEMG distinguish between patients with HOPP and control subjects?					
20 (MC)	33	46	Clinical attacks, vacuolar myopathy, children with attacks	sMFCV	HOPP < control (P < 0.0001); sensitivity 70%

All studies included were class III. None were blinded but all were considered to apply objective anaylsis of sEMG data. Sensitivity and specificity provided where available. ACF, autocorrelation function; ADP, acquired demyelinating polyneuropathy; ALS, amyotrophic lateral sclerosis; BMD, Becker muscular dystrophy; BP, bipolar; CMAP, compound muscle action potential; DMD, Duchenne muscular dystrophy; HMSN, hereditary motor and sensory neuropathy; HOPP, hypokalemic periodic paralysis; MC, multichannel; MCV, muscular conduction velocity; MP, monopolar; MPF, median power frequency; MU, motor unit; N30, latency of negative wave; nEMG, needle electromyography; P20, latency of positive wave; PPS, post-poliomyelitis syndrome; RMS, root mean square; sEMG, surface electromyography; SMA, spinal muscular atrophy; sMFCV, surface mean muscle fiber conduction velocity; TCR, trigemino-cervical response; V/E, voluntary/elicited; ZX, zero crossings.

Additional Value of sEMG for Study of Neuromuscular Disorders. The additional value of sEMG for the study of neuromuscular disorders was evaluated by looking at both bipolar sEMG recording and HD-sEMG. Both were found to add some additional information beyond that obtained from NCS and nEMG studies. Bipolar sEMG recordings provided added information in the study of: (1) fatigue in neuromuscular diseases, with reported changes in waveform amplitude in association with muscle contraction over time [i.e. root mean square (RMS)] in subjects with PPS, torque–RMS ratio in Charcot–Marie–Tooth (CMT) disease and myotonic dystrophy (DM), amplitude–slope reduction in long-thoracic nerve palsy (all class III), and median frequency shift in ALS (class IV)[8,13,17,18]; (2) electromechanical function and energy utilization, with changes in average rectified value of amplitude and RMS, suggesting altered sarcolemmal excitability and electromechanical coupling efficiency in subjects with DM and increasing electrical activity in contracting muscle in subjects with McArdle's disease (all class III)[3,14,24]; and (3) muscle potential propagation, with evidence of voltage and frequency decay, suggesting abnormal muscle fiber depolarization over the course of muscle contraction in myotonia congenita (MC) (class IV).[12]

HD-sEMG provided additional information regarding: (1) muscle action potential propagation, identifying decreased motor unit action potential amplitude and propagation abnormalities in subjects with MC (class III)[5,7]; and (2) energy utilization, with electrophysiologic recovery unchanged as mea-

sured by bipolar RMS and frequency values in muscle of subjects with autosomal-recessive generalized myotonia infused with the Na^+–K^+–ATPase inhibitor, oubain (class IV).[19]

CONCLUSIONS

The following levels were used for the classification of recommendations discussed[9]:

A = Established as effective, ineffective, or harmful for the given condition in the specified population. (Level A rating requires at least two consistent class I studies.)

B = Probably effective, ineffective, or harmful for the given condition in the specified population. (Level B rating requires at least one class I study or at least two consistent class II studies.)

C = Possibly effective, ineffective, or harmful for the given condition in the specified population. (Level C rating requires at least one class II study or two consistent class III studies.)

U = Data inadequate or conflicting; given current knowledge, treatment is unproven.

Utility of sEMG for the Detection of Neuromuscular Disease. Based on a review of the literature, the following conclusions can be drawn regarding the utility of sEMG for the detection of neuromuscular disease:

1. On the basis of two class III studies, sEMG may be useful to detect the presence of neuromuscular disease (level C).
2. The data are insufficient to determine the clinical utility of sEMG for distinguishing between neuropathic and myopathic conditions or for detecting the more specific neuromuscular conditions of post-poliomyelitis syndrome, pathologic fasciculations, acquired demyelinating peripheral neuropathy, amyotrophic lateral sclerosis, myotonic dystrophy, and hypokalemic periodic paralysis (level U).
3. The data are insufficient to address the question of disease severity detectable by sEMG (level U).
4. The data are insufficient to compare diagnostic utility of sEMG with the conventional technologies of nEMG, NCS, and muscle ultrasonography (level U).

The aforementioned conclusions do not differ significantly from those of previous sEMG technology reviews that found no added clinical utility of sEMG over conventional nEMG as a diagnostic tool for detection and differentiation of myopathic from neuropathic neuromuscular diseases.

Added Value of sEMG in the Study of Neuromuscular Disease. Based on a review of the literature the following conclusions can be drawn regarding the added value of sEMG in the study of neuromuscular disease:

1. sEMG may be useful in adding information in the study of fatigue in post-poliomyelitis syndrome and electromechanical coupling dysfunction in myotonic dystrophy on the basis of two class III studies each (level C rating).
2. The data are insufficient to determine the added value of sEMG myoelectric signal changes in the study of fatigue in myophosphorylase deficiency, muscle fiber and motor unit propagation in myotonia congenita and hypokalemic periodic paralysis, or in evaluation of disease progression in myotonic dystrophy and Charcot–Marie–Tooth disease (level U rating).

RECOMMENDATIONS FOR FUTURE RESEARCH

Further research is necessary to determine the clinical utility of sEMG in the diagnosis of neuromuscular diseases and in the differentiation of primary myopathic and neuropathic conditions:

1. Future studies should study patients with neuromuscular diseases defined by a carefully chosen reference (gold) standard. The technique should also be applied to a broad spectrum of subjects including healthy controls and patients with non-neuromuscular diseases. There should be adequate blinding, and the size of the cohorts should be sufficient to detect meaningful differences.
2. The current lack of standardization of sEMG protocols among investigative groups and the variable documentation of methodology makes comparisons between studies problematic. Future studies should include comprehensive methodological and data presentation to facilitate duplication and corroboration of results and to provide clarity for clinical applications.

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