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Recording characteristics of electrical impedance-electromyography needle electrodes

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Abstract

Objective—Needle EMG remains the standard clinical test for neuromuscular disease (NMD) assessment, but it only characterizes myofiber membrane depolarization. On the other hand, electrical impedance provides non-electrically active structural and compositional data of tissues. Here, we designed a prototype of needle electrode integrating electrical impedance and EMG measurement capabilities, the so-called I-EMG needle electrode.

Approach—We use finite element method models to study the impedance recording characteristics of I-EMG needle electrodes. The simulated electrical and mechanical design specifications are then manufactured to create a prototype of an I-EMG needle electrode. We pilot these new needle electrodes by conducting *in vivo* impedance measurements with muscle at rest on healthy wild-type (wt, $n = 5$) and muscular dystrophy (mdx, $n = 5$) mice. Comparisons between wt and mdx mice are performed using Mann–Whitney test, two-tailed, $p < 0.05$. The electrical characterization of the EMG electrode in the developed I-EMG needles was performed *in vitro* on saline solution and through EMG detection in wt animal at rest and during voluntary contractions.

Results—Muscle impedance demonstrate good repeatability ($p < 0.05$ and $p < 0.005$ for resistance and reactance at 50 kHz, respectively) and agreement between different I-EMG needles. Impedance data allows us to discriminate between mdx and wt muscle ($p < 0.05$ and $p < 0.005$ for resistance and reactance at 10 kHz, respectively). EMG broadband noise power and peak amplitude using the I-EMG needle were similar to that of a commercial monopolar EMG needle. EMG recordings using the I-EMG needle measured electrical activity similar to a standard monopolar needle with muscle at rest and during voluntary contraction.

Supplementary material for this article is available online

Conflict of interest

Dr Rutkove has equity in, and serves as a consultant and scientific advisor to, Skulpt, Inc., a company that designs impedance devices for clinical and research use; he is also a member of the company's Board of Directors. The company also has an option to license patented impedance technology of which Dr Rutkove is named as an inventor. Dr Sanchez is named as an inventor on several patent applications in the field of electrical impedance. Dr Sanchez serves as a consultant to Maxim Integrated, Inc., and Impedimed, Inc., two companies that develop impedance devices for consumer electronics, clinical and research use. This study, however, did not employ any relevant company or patented technology.

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Significance—Needle I-EMG technology may offer the opportunity to enhance the diagnostic capability and quantification of NMD beyond that possible with either impedance or EMG techniques separately. Ultimately, needle I-EMG could serve as a new bedside tool to assess NMD without increasing the complexity or duration of the EMG test.

Keywords

needle electrical impedance-electromyography; needle electrodes; four-electrode technique; muscular dystrophy muscle

1. Introduction

Weakness, disability and death remain as the ultimate clinical consequences of neuromuscular disorders (NMD). Although there have been major inroads in the treatment of such diseases, most still remain without effective therapy (see table 1). The increasing number of patients with NMD and the high cost for the healthcare system is the ultimate proof that there is a great need for improved technologies to assess muscle health. For example, the estimated cost in United States alone for amyotrophic lateral sclerosis, Duchenne and myotonic muscular dystrophies combined is >\$1 billion per year (Larkindale *et al* 2014).

Needle EMG is the standard office-based test to assess NMD (Daube and Rubin 2009). Over 1 million EMG studies are performed annually in the United States (Dillingham *et al* 2004). In EMG, a needle is inserted through the skin and subcutaneous fat into a muscle of interest using anatomical landmarks. Once placed in the muscle, the needle is gently maneuvered through the tissue to intentionally irritate and injure individual myocyte membranes (Rubin 2012). The examiner seeks to identify abnormal spontaneous potentials (Preston and Shapiro 2002), including repetitive discharges from single muscle fibers (e.g. fibrillation potentials and myotonic potentials) or from groups of muscle fibers (e.g. fasciculation potentials and myokymic potentials) (Paganoni and Amato 2013). Once this part of the procedure is completed, the patient is then asked to contract the muscle and the electrical characteristics of the voluntary motor unit potentials are evaluated as well as their recruitment patterns. During this procedure, the examiner monitors the patient to ensure that the muscle is being correctly activated while simultaneously scrutinizing the screen and evaluating the EMG signal. Once the test is completed, the examiner makes subjective decisions regarding the status of the muscle, grading abnormalities on a 0–3 ordinal scale (normal, mild, moderate and severe) for both ‘spontaneous activity’ (the part of the exam with muscle at rest) and ‘voluntary motor unit analysis’ (when the patient intentionally contracts the muscle).

Needle EMG has three important limitations that have hampered the technology since its inception (Wilbourn 1993). First, EMG can only assess the *active* electrical activity of the muscle fiber membrane. While this is clearly valuable, we do not learn anything about the non-electrically active structural and compositional (i.e. *passive*) properties of the muscle. However, most NMD impact the muscle in complex ways, causing major alterations in its histology. For example, myopathies may have large numbers of inflammatory cells, edema, and fat and connective tissue deposition. Neurogenic diseases are associated with myofiber

atrophy, fiber type grouping, and intracellular edema. Needle EMG data is essentially ‘blind’ to these underlying alterations, the data being based on the secondary effects of these changes on the *active* electrical properties of the fibers membrane. Second, needle EMG fails to provide information on the contractile apparatus of the muscle itself, since it only measures up to the point of muscle fiber depolarization. Sarcomeric disruption, extracellular connective tissue deposition, and other abnormalities can all impact muscle force, but still leave the EMG signal unaffected. Finally, as commonly practiced, EMG is subjective with the examiner rating abnormalities on a coarse subjective scale. Studies have shown remarkably poor repeatability even among expert electromyographers, with only 60% inter-rater agreement rates (Kendall and Werner 2006) and a specificity for disease of only 70% (Narayanaswami *et al* 2016). Whereas quantitative EMG methods exist, they are rarely used in practice given their very time consuming nature (Farkas *et al* 2010).

Impedance technology embedded into an EMG needle electrode has the potential for improving EMG technology by providing a *complete* (i.e. *passive*—impedance—and *active*—EMG—) electrical characterization of muscle. To achieve this goal, we first focused our previous research efforts on proving the feasibility of recording muscle impedance using a single tetrapolar needle electrode (see Kwon *et al* (2017b)). Here, we build upon our work to construct and pilot *in vivo* a prototype of impedance-EMG (I-EMG) needle electrode in healthy and diseased muscle at rest and during activity.

2. Materials and methods

2.1. Finite element method modeling

We adapted the finite element model (FEM) developed in Kwon *et al* (2017b) using Comsol Multiphysics (Comsol, Inc., Burlington, MA, USA) to study the impedance recording characteristics of I-EMG needle electrodes (sketched in figure 1). For brevity, the design parameters of the I-EMG needle electrodes and the simulation settings are available in the supplementary information (stacks.iop.org/PM/39/055005/mmedia).

2.2. Needle I-EMG electrode manufacturing

The I-EMG needle electrode prototype tested (see in figure 2) was manufactured by FHC, Inc., (Bowdoin, ME, USA) with the same manufacturing process to that described in Kwon *et al* (2017b) (detailed in the supplementary information for convenience).

2.3. Animals

All studies were approved by the Institutional Animal Care and Use Committee at Beth Israel Deaconess Medical Center (BIDMC). Five DBA/2-congenic dmd-mdx mice (weight 18.2 ± 1.6 g, mean and std) (Jackson Laboratory, Bar Harbor, ME, USA, strain #013141) were compared to five wild-type (wt) control DBA/2J mice (weight 21.2 ± 1.8 g, mean and std) (Jackson Laboratory, strain #000671) at age 6 weeks. Animals were acclimatized at the Animal Research Facility for a period of 48 h before experimentation and fed a regular diet ad libitum. During the experimentation, the animals were anesthetized with isoflurane delivered by nosecone, while the body and muscle temperature were maintained by a heating pad (37 °C). Fur was removed with clippers prior to the measurements. We then conducted

needle I-EMG measurements on the quadriceps muscle with the animals first fully and then mildly sedated as described below in sections 2.6 and 2.7, respectively. The animals were euthanized at the completion of the studies by inhalation of CO₂.

2.4. Impedance measurements with muscle at rest

Impedance was measured with the animals fully sedated (isoflurane 1% to 2%) between 3 kHz and 1 MHz (256 frequencies) using SFB7 (Impedimed, Inc., Brisbane, Australia). The impedance device was connected to the impedance current—drive—and voltage—sense—electrodes available in the needle I-EMG connector (see electrodes 1 to 4 in red and blue color in figure 2 and schematic in figure 3(A)). Impedance reproducibility using the same needle was assessed in all animals with two consecutive measurements 5 min apart. In all measurements, the I-EMG needle was inserted into the muscle and then removed after completing the measurement. We also repeated impedance measurements using two different I-EMG needle electrodes to determine any systematic differences such as bias or trends between I-EMG needle electrodes.

2.5. EMG broadband noise measurements (*in vitro* characterization)

Sequential broadband baseline noise measurements were made using a 50 mm × 26G subdermal monopolar needle electrode (#902-DMG50S, Natus Neurology, Inc., Middleton, WI, USA) and the I-EMG needle (see electrode 5 in green color in figure 2) connected to the positive input of a commercial EMG monitoring system (TECA Synergy T2, Oxford Instruments Medical, Ltd., Concord, MA, USA). The needles' tip were immersed to a depth of 2 cm in physiologic saline solution at room temperature. The needles were held in the grounded saline bath by a grounded micromanipulator. The negative and reference inputs of the EMG monitoring system were connected to a surface gel-adhesive electrode (#70010-K/C/12, Ambu, Denmark) immersed in the saline solution. Sixty-hertz line interference was minimized by turning off all electrical equipment in the vicinity of the bath. The noise was then amplified (gain 100) through a bandpass filter from 10 Hz to 10 kHz, and digitized at the rate of 19.2 kHz for 1 s. With both needles, peak and power (root mean square, RMS) of the recorded noise signal were measured. For average peak noise, 10 segments of 100 ms were created and the peak noise in each segment was measured and averaged. Noise power was measured over the entire 1 s. This procedure was performed twice and the mean value and the standard deviation on the noise RMS and peak voltage was calculated.

2.6. EMG measurements with muscle at rest

We connected the EMG electrode in the needle I-EMG (see electrode 5 in green color in figure 2 and schematic in figure 3(B)) to the positive input of the EMG monitoring system (TECA Synergy T2, Oxford Instruments Medical, Ltd.). To record insertional EMG activity, we connected the negative and reference inputs of the EMG monitoring system to a subdermal monopolar needle electrode (#902-DMG50S, Natus Neurology) and to a surface gel-adhesive electrode (#70010-K/C/12, Ambu), respectively (see figure 3(B)). Recordings were made on wt animals while intentionally performing small insertional movements with the needle I-EMG into the muscle. Upon completion, we replaced the I-EMG needle electrode with a subdermal monopolar needle electrode (#902-DMG50S, Natus Neurology)

and repeated the process to register EMG spontaneous activity. The EMG instrumentation settings were the same as in section 2.5.

2.7. EMG measurements during voluntary muscle contraction

EMG recordings were made on wt animals mildly sedated (isoflurane 0.5% to 1%) while providing gentle stimuli to the hind limbs such that the animals repeatedly withdraw the limb. We followed the same methods described in the previous section 2.6 to record EMG and to compare the developed electrode with standard EMG electrode.

2.8. Data analysis

We normalized the apparent impedance measured (resistance and reactance in Ω) with muscle at rest into apparent impedivity (resistivity and reactivity in Ωm) by means of calibration measurements in saline solution. We used the same experimental setup as the *in vivo* measurements described in section 2.6. Calibration measurements were taken at room temperature immediately after immersing the I-EMG needle electrode into saline solution.

2.9. Statistical analysis

Statistical differences in impedance between wt and mdx muscle at rest were determined using Mann–Whitney test, two-tailed, and significance $p < 0.05$. Test-retest measurements were taken using the same I-EMG needle electrode by performing repeated measurements on the same animal five minutes apart. Bland–Altman analysis was performed to evaluate the agreement among two different I-EMG needle electrodes. A single examiner performed the impedance measurements doing repeated measurements on the same animal.

3. Simulation results

3.1. Impedance sensitivity distribution

Figure 4 shows the comparative three-dimensional sensitivity distribution plots around the I-EMG needle when simulating the impedance electrodes in the subcutaneous fat, at shallow depth in the muscle, and deep in the muscle. The sensitivity distribution changes with the impedivity of skin, subcutaneous fat, and muscle tissues. A more detailed view of the sensitivity distribution in the longitudinal and transverse plane is shown in figures 5(A) and (B) with two different separations between voltage—sense—electrodes: $b = 0.5$ and $b = 1$ mm. In figure 5, the sensitivity scale is arbitrarily normalized so that the sensitivity values are appreciable to the naked eye. The apparent impedivity measured in each case is quantified in figure 5(B) from 10 kHz to 1 MHz. Both I-EMG needle electrode designs give similar apparent impedivity when the electrodes are positioned deep in the muscle.

3.2. Specific tissue contribution to the apparent impedivity

From the sensitivity, we calculate the percentage contribution of each tissue to the measured apparent impedance. The values are summarized in table 2 at 10 kHz and in supplementary table 1 at 331 kHz. The presence of the I-EMG needle metal tip does not deteriorate the ability of impedance electrodes to measure the impedance of the muscle, yielding a contribution of muscle equal to or greater than 97% to the apparent impedivity measured.

4. Experimental results

4.1. Impedance repeatability and agreement between I-EMG needle electrodes

Figure 6(A) shows impedance values combining wt and mdx mice, comparing 50 kHz data measured five minutes apart using the same prototype I-EMG needle electrode.

The mean impedance difference values shown in figure 6(B) show no significant systematic bias between two different prototype I-EMG needle electrodes.

4.2. Wild-type and muscular dystrophy muscle impedance

Figure 7 shows the apparent impedivity in the complex plane of healthy and dystrophic (mdx) muscle. The reduction in resistivity detected with needle I-EMG is consistent with the lack of dystrophin expression in young mdx mice. Disease progression in mdx mice is characterized by early muscle edema followed by connective tissue replacement of atrophied myofibers. The representation of the data against the frequency measured including the standard error of the mean is available in supplementary figure 3. Dystrophic apparent resistivity and apparent reactivity values at 10 kHz were lower than those in healthy wt muscle ($p < 0.05$ and $p < 0.005$, respectively).

4.3. EMG broadband noise (*in vitro* characterization)

Table 3 shows the average RMS and peak noise values for a standard commercial 26G monopolar EMG needle (nominal outer diameter 0.46 mm) and the prototype of I-EMG needle tested (19G, nominal outer diameter 1.067 mm).

4.4. EMG recordings with muscle at rest and during voluntary contraction

Figure 8 shows an example of EMG recordings on a healthy wt mouse. Electrical activity was recorded sequentially first using a standard monopolar EMG needle electrode (A) and then using an I-EMG (B) needle electrode.

5. Discussion

Needle EMG examination has remained the standard approach to NMD assessment for a variety of reasons including (1) the ability to sample a variety of muscles; (2) the high sensitivity to the presence of disease; and (3) the capability to identify the acuity/chronicity of a problem. This set of advantages have made EMG the standard, 'go-to' test used to evaluate nerve and muscle disorders (Wilbourn 1993).

Recently, other technologies also have been employed in the evaluation of NMD to assist EMG in the diagnostic process. Standard metrics based on force can detect deterioration of muscle performance; however, they usually require the use of cumbersome equipment and patient cooperation, the latter difficult to achieve if there is associated pain. Muscle edema, fatty infiltration and mass lesion can cause abnormal signal intensity on magnetic resonance imaging (MRI) and computerized tomography (CT); however, the use of such technologies has a high associated cost and, perhaps more importantly, they cannot be employed where they are needed the most: at the bedside to help physicians while evaluating a patient face-

to-face. Ultrasound (US) is another imaging technology available that can be used at the bedside. In fact, Natus Medical, Inc. (Pleasanton, CA, USA), a major manufacturer of clinical EMG systems, has recently commercialized a system that combines both EMG and ultrasound (Vista Ultrasound). However, the signal quality has limited reproducibility and interpretation largely depends on the examiner's expertise. To date, despite the important limitations, no other technology has been able to replace EMG as primary diagnostic tool.

Electrical impedance, also known as electrical impedance myography (EIM) (Sanchez and Rutkove 2017), is a fourth alternative technology available for muscle evaluation. Impedance's main value has been to assess NMD status, although it can also provide diagnostic information, being able to discriminate myopathic from neurogenic disease (Garmirian *et al* 2009). Unlike the technologies mentioned above, impedance is a technique that lends itself naturally to being combined with EMG. First, impedance measures muscles' passive electrical properties, thus complementing the active electrical information provided by EMG. Second, impedance can provide quantitative data both at rest and with muscle contraction. Finally, and unlike MRI, CT and US, this study proves impedance can be integrated within the EMG needle itself. Perhaps more importantly, needle I-EMG may enable in the future simultaneous impedance and EMG recordings without affecting the complexity or duration of the EMG test.

Here, for first time we have integrated impedance recording capabilities into an EMG needle electrode. Compared to our previous EIM needle design (Kwon *et al* 2017b), the I-EMG needle described here is longer (45 mm versus 25 mm) to facilitate its manipulation. Here also, the connection to the impedance and EMG measuring device(s) is made using a polarized 2.54 mm pin male header connector.

The simulation results summarized in table 2 show the contribution of muscle to the measured impedance is above 97% at 10 kHz and consistent across the frequency range (see 331 kHz data in supplementary table 1) once the I-EMG needle electrode is inserted into the muscle. This excellent sensitivity to muscle impedance was also found simulating a thinner 23G and sharper 15 °I-EMG needle electrode (see supplementary figure 1 and supplementary tables 1 and 2). These results are very similar to the previously developed EIM needles electrodes in Kwon *et al* (2017b), indicating that the additional EMG electrode does not deteriorate the performance to measure muscle impedance. When measuring muscle at rest, impedance data (shown in figure 6) was found to be reproducible. Impedance data recorded with two different I-EMG needle electrodes was also in good agreement. Differences were within the mean $\pm 95\%$ thus suggesting that the process of manufacturing I-EMG needles is reproducible and different I-EMG needles may be used interchangeably. Furthermore, it was possible to discriminate between healthy and dystrophic muscle (shown in figure 7). The EMG broadband noise power and peak measured with the I-EMG needle was similar to that of a thinner commercial monopolar EMG needle and consistent with values reported in the literature (Dorfman *et al* 1985). Qualitatively, figure 8 shows that the I-EMG needle is capable of detecting electrical activity with muscle at rest and during voluntary contraction.

Among the limitations of this study is that, compared to *in vitro* experiments, determining *in vivo* the relative orientation measured with respect to the orientation of the myofibers (i.e. anisotropy) was not possible. We are currently working to extend the methods presented in Kwon *et al* (2017a) to obtain the direction information through the use of multiple I-EMG needles (unpublished results). Compared to commercially available monopolar and concentric EMG needle electrodes, further quantitative work assessing robustness to line interference, capacitance between electrodes, harmonic distortion and common-mode conversion is required to validate the ability of I-EMG needle electrodes to record EMG signals (Nandedkar *et al* 1990, Nandedkar and Sanders 1991). Of course, a larger number of needles need to be tested before culminating in the clinical application. Finally, care should be taken when inserting the I-EMG needle to avoid bleeding in the channel of the needle. Like any (minimally) invasive impedance measurement, the presence of biofluids near the electrodes could alter the data.

Despite this being the first study integrating impedance and EMG modalities in a single needle electrode, needle I-EMG technology has the potential to greatly extend the diagnostic capabilities of standard needle EMG. In short, needle I-EMG allows the examiner to obtain a *complete* (i.e. *passive*—impedance—and *active*—EMG—) electrical characterization of muscle. For example, chronic myopathic and neurogenic conditions are challenging to distinguish on EMG; however, they may be easily differentiated with impedance. Fat deposition in myopathies will increase muscle resistivity, whereas the increased intracellular water in neurogenic conditions will decrease muscle resistivity. Reductions in resistivity data will provide quantitative values on muscle condition in mild neurogenic conditions where subtle alterations in motor unit potential recruitment can be difficult to identify on EMG. Ultimately, needle I-EMG has the potential to become a ‘smarter’ clinical tool for assessment of muscle and nerve condition and as a biomarker of therapy efficacy.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Schematic illustrating an impedance-electromyography (I-EMG) needle inserted through the skin and subcutaneous fat into the muscle. The I-EMG needle consists of a monopolar active EMG electrode located at the tip of the needle (in green), and ring-shaped tetrapolar impedance electrodes disposed along the needle's shaft (current—drive—and voltage—sense—impedance electrodes are shown in red and blue, respectively). The recording characteristics of I-EMG needles are determined by the distance between current and voltage electrodes a , the distance between voltage electrodes b , impedance electrodes' dimension width w and height h , EMG electrode area A , needle gauge g , tip angle η , insertion angle θ , orientation angle ϕ , and depth of penetration d . All these parameters determine the spatial resolution being sensed by the impedance electrodes, illustrated in the figure by an ellipsoid.

Figure 2.

Dimensions of the impedance-electromyography (I-EMG) needle electrodes simulated in section 3 and tested in section 4. Electrode nomenclature: current—drive—(in red), source and sink current electrodes 1 and 4; voltage—sense—(in blue), high and low voltage electrodes 2 and 3. The EMG recording electrode 5 is located at the tip of needle (in green).

Figure 3. Schematic illustrating the experimental setup for sequential impedance (I) (A) and electromyography (EMG) (B) measurements with muscle at rest using an I-EMG needle electrode. The pins in the needles' connector denote the use of the electrodes available on the needles.

Figure 4.

Illustrative finite element modeling simulations of a 19G impedance-electromyography (I-EMG) needle electrode (nominal outer diameter 1.067 mm). Three-dimensional impedance sensitivity distribution simulations plots at 10 kHz with equidistant impedance electrodes separation $a = b = 0.5$ mm. The needle I-EMG electrode depth of penetration is $d = -6$ mm (left), $d = 0$ mm (center), and $d = 6$ mm (right); defined as the distance between the impedance current—drive—electrode furthest from the tip of the needle and the interface between the subcutaneous fat and muscle tissues. The reader is referred to the figure online for an interactive version and figure 5 for further details.

Figure 5.

Finite element modeling simulations of a 19G impedance-electromyography (I-EMG) needle electrode in figure 4. Examples of sensitivity distributions simulations at 10 kHz in transverse and longitudinal directions shown in figure 4 for two impedance voltage electrodes separations, $b = 0.5$ mm (A) and $b = 1$ mm (B). The needle I-EMG electrode depth of penetration is $d = -6$ mm (i), $d = 0$ mm (ii), and $d = 6$ mm (iii); defined as the distance between the impedance current electrode furthest from the tip of the needle and the interface between the subcutaneous fat and muscle tissues. Scale bar: 3.125 mm. (C) Apparent resistivity ρ_a and apparent reactivity τ_a measured in (A) and (B) from 10 kHz to 1 MHz (51 frequencies). The apparent impedivity of muscle is shown for each case in circles for comparison purposes. The specific percentage contributions of skin, subcutaneous fat, and muscle tissues to ρ_a and τ_a in (C) are summarized in table 2.

Figure 6.

(A) Test-retest reliability impedance experiment using the same prototype of impedance-electromyography (I-EMG) needle electrode. (B) Bland–Altman plot shows no significant systematic disagreement between two different I-EMG needle electrodes tested.

Figure 7.

Comparison of the averaged apparent resistivity (A) and apparent reactivity (B) measured using needle impedance-electromyography (I-EMG) in a cohort of muscular dystrophy (mdx, $n = 5$) and healthy wild-type (wt, $n = 5$) mice. Representative frequencies from 3 kHz to 1 MHz are shown in circles, increasing in counterclockwise direction.

Figure 8.

Example of electrical activity recorded on healthy muscle during needle insertion and voluntary contraction using a standard monopolar electromyography (EMG) needle electrode (A) and with the prototype of impedance-EMG needle electrode developed (B). EMG activity and sporadic occurrences of movement artefacts can be observed in all traces.

Table 1Epidemiology of some more common neuromuscular disorders^a.

Disease	People affected ^b	Disease	People affected ^b
Amyotrophic lateral sclerosis	30 000	Statin induced myopathy	0.2 million
Spinal muscular atrophy	25 000	Carpal tunnel syndrome	5–10 million
Peripheral neuropathy	20 million	Lumbosacral radiculopathy	9–15 million
Muscular dystrophies	60 000	Cervical radiculopathy	0.8–1 million
Inflammatory myopathy	24 000		

^aThe ALS Association (www.alsa.org), Muscular Dystrophy Association (www.mda.org), Prize4Life (www.prize4life.org).^bIn the United States only.

Table 2

Specific contribution of skin, subcutaneous fat, and muscle tissues to the apparent resistivity ρ_a and apparent reactivity τ_a measured at 10 kHz in figure 4 (the percentage contribution is shown in parenthesis). Data shown correspond to two different separations between the inner voltage—sense—electrodes, $b = 0.5$ mm and $b = 1$ mm in figures 4(A) and (B), respectively. The reader is referred to supplementary table 1 for the specific tissues' contribution at 331 kHz.

Needle electrode depth of penetration d									
b (mm)		−6 mm	−4 mm	−2 mm	0 mm	2 mm	4 mm	6 mm	
ρ_a (Ω cm)	Skin	0.5	0.7 (0%)	0.4 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
		1	0.9 (0%)	0.4 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
	Sub. Fat	0.5	3780.7 (98.7%)	3158.7 (98.8%)	7.9 (2.6%)	4.4 (1.7%)	0.7 (0.3%)	0.2 (0.1%)	0.1 (0%)
		1	2536.9 (88.6%)	2804.1 (96.1%)	9 (2.9%)	4.6 (1.8%)	0.7 (0.3%)	0.2 (0.1%)	0.1 (0%)
	Muscle	0.5	47.7 (1.2%)	39.2 (1.2%)	299.9 (97.4%)	256.6 (98.3%)	225.1 (99.7%)	221.7 (99.9%)	220.7 (100%)
		1	325.9 (11.4%)	114.2 (3.9%)	305.6 (97.1%)	256.1 (98.2%)	223.9 (99.7%)	220.1 (99.9%)	219.1 (100%)
	Total	0.5	3829 (100%)	3198.3 (100%)	307.9 (100%)	261 (100%)	225.8 (100%)	221.9 (100%)	220.8 (100%)
	1	2863.6 (100%)	2918.7 (100%)	314.7 (100%)	260.6 (100%)	224.7 (100%)	220.4 (100%)	219.2 (100%)	
$-\tau_a$ (Ω cm)	Skin	0.5	−1.6 (−1.6%)	−0.9 (−1.1%)	−0 (−0.2%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
		1	−2.1 (−2.7%)	−0.9 (−1.2%)	−0 (−0.2%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
	Sub. Fat	0.5	99.2 (100.2%)	81.5 (101.8%)	0.3 (3.3%)	0.1 (2.2%)	0 (0.3%)	0 (0.1%)	0 (0%)
		1	69.4 (90.8%)	72.8 (98.6%)	0.3 (3.7%)	0.1 (2.3%)	0 (0.4%)	0 (0.1%)	0 (0.1%)
	Muscle	0.5	1.4 (1.4%)	−0.6 (−0.7%)	9 (96.8%)	6.4 (97.8%)	5.7 (99.7%)	5.6 (99.9%)	5.6 (100%)
		1	9.1 (11.9%)	1.9 (2.6%)	9.1 (96.5%)	6.3 (97.7%)	5.6 (99.6%)	5.5 (99.9%)	5.5 (100%)
	Total	0.5	98.9 (100%)	80.1 (100%)	9.3 (100%)	6.5 (100%)	5.7 (100%)	5.6 (100%)	5.6 (100%)
	1	76.4 (100%)	73.9 (100%)	9.4 (100%)	6.4 (100%)	5.6 (100%)	5.5 (100%)	5.5 (100%)	

Table 3

Mean $\pm$ standard deviation of broadband noise measurements. All units are μV .

Needle	RMS	Peak
Monopolar EMG 50mm $\times$ 26G (Natus #902-DMG50-S)	1.7 $\pm$ 0.1	5.8 $\pm$ 0.9
I-EMG 45mm $\times$ 19G	1.7 $\pm$ 0.3	4.9 $\pm$ 1.3

Abbreviations: RMS, root mean square; EMG, electromyography; I-EMG, impedance-electromyography.