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Design and evaluation of a chronic EMG multichannel detection system for long-term recordings of hindlimb muscles in behaving mice

Vicki M. Tysseling¹, Lindsay Janes², Rebecca Imhoff¹, Katharina A. Quinlan¹, Brad Lookabaugh¹, Shyma Ramalingam¹, C.J. Heckman^{1,3,4}, and Matthew C. Tresch^{1,2,3}

¹Physiology Department, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

²McCormick Biomedical Engineering Department, Northwestern University, Evanston, IL, USA

³Department of Physical Medicine and Rehabilitation, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

⁴Department of Physical Therapy and Human Movement Sciences, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA

Abstract

Mouse models are commonly used for identifying the behavioral consequences of genetic modifications, progression or recovery from disease or trauma models, and understanding spinal circuitry. Electromyographic recordings (EMGs) are recognized as providing information not possible from standard behavioral analyses involving gross behavioral or kinematic assessments. We describe here a method for recording from relatively large numbers of muscles in behaving mice. We demonstrate the use of this approach for recording from hindlimb muscles bilaterally in intact animals, following spinal cord injury, and during the progression of ALS. This design can be used in a variety of applications in order to characterize the coordination strategies of mice in health and disease.

Keywords

EMG; spinal cord injury; ALS; locomotion; mouse; chronic; behavioral analysis; electrodes

Introduction

Mice have become a standard model for the characterization of motor control, both in intact animals and in animals with injury (SCI studies with mice), disease (ALS, Alzheimer's, Parkinson's model), or genetic manipulation (Bonnot et al., 2002, Ebadi et al., 2005, Jessell and Sanes, 2000, Kiehn and Kullander, 2004, Steward et al., 1999, Wong et al., 2002).

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Corresponding author: Vicki M. Tysseling, PhD, PT, 303 E. Chicago, Ward Bldg 5-281, Northwestern University, Chicago, IL 60610, Telephone: 312.503.1791, Facsimile: 312.503.5101, vmtysseling@u.northwestern.edu.

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Although analyses involving gross behavioral observation or limb kinematics of mice provide valuable information about motor performance, electromyographic recordings (EMGs) are generally considered to provide critical information for characterizing motor function and disease progression (Leblond et al., 2003). For instance, although one of the major consequences of spinal cord injury in humans is the development of spasticity, such spasticity is difficult to assess from simple observations of limb movements. Subtle deficits of muscle coordination within a limb can be difficult to discern without recording from EMGs, because of the widely recognized difficulties in measuring kinematics in rodents (Bauman and Chang, 2010, Leblond, L'Esperance, 2003). And for motor neuron degenerative diseases such as ALS, EMG characterization is often a critical diagnostic tool to evaluate disease progression (Benatar, 2006, de Carvalho et al., 2008, England et al., 2009).

Acquisition of EMGs in behaving mice presents obvious challenges, primarily due to their small size. Several groups have developed methods to record EMGs in behaving mice but, generally, these studies are limited to a few (1-4) muscles at a time and most have only recorded up to 2 weeks (Akay et al., 2006, Courtine et al., 2008, Fortier et al., 1987, Leblond, L'Esperance, 2003, Masuki and Nose, 2003, Milner et al., 1995, Pearson et al., 2005, Wernig et al., 1989). We describe here our design and evaluation of a chronic EMG system capable of measuring the activity in a larger number of muscles (up to 8) in behaving mice. The array is an extension of previously used techniques for recording EMGs but adapted for the small size of mice (Loeb and Gans, 1986). We evaluate the capability of this design to measure EMGs chronically, over a period of several weeks to months and demonstrate its application to the study of spinal cord injury and ALS. The methods described here can potentially be used in a variety of applications to characterize motor function in mouse models of degenerative diseases, genetic mutations, and basic behavioral performance.

Materials and Methods

Northwestern University Animal Care and Usage Committee approved all animal procedures. We used adult female CD-1 mice and B6SJL SOD1^{G93A} and SOD1^{wt} transgenic mice of either sex in these experiments.

3.1 Construction of EMG electrodes

Omnetics connectors (Nano series) are the basis for the design used here. These connectors can be obtained with the EMG electrode wire (A/M systems, multistranded stainless steel, Teflon insulated, #793200) preassembled so no soldering or crimping is required (Figure 1). The total length of the wire from the base of the connector is 14cm. The wires are threaded through a nylon mesh (Industrial Netting, #ET8120; Figure 1a, Figure 2a) and epoxied in place, leaving approximately 4mm between the base of the connector and the mesh. This was found to be an appropriate distance from the skin to allow manipulation of the connector during recording sessions without compromising the lifetime of the device. A small brass ring approximately 1cm in diameter is placed around the connector and secured with epoxy, leaving the 4mm underneath. This brass ring serves to prevent animals from chewing on the connector. We have used two different designs of these connectors, with and without an extra ground electrode embedded in the epoxy (Figure 1, Figure 2a). In our experimental configuration, grounding the animal did not result in any substantial improvement in recording quality, but in our current design the embedded ground electrode is standard. Small holes are drilled into the epoxy near the brass ring. These holes are used to secure wound margins to the implanted connector (Figure 1c, Figure 2a). Lastly a minimal amount of epoxy is used to secure the wires to the underside of the mesh to lead them in the caudal direction and keep them flat. Securing the wires flat against the mesh

increased the lifetime of the device and prevented excess irritation around the connector implantation site.

At the distal end of the electrodes three to five small overlapping knots are made in each wire at the same point, approximately 6cm from the distal end (Figure 2b). We found that a single knot often slipped through an implanted muscle and at least three knots were necessary to secure the electrode. Approximately 0.5mm of the insulation is then stripped at a distance of 0.5 - 1mm distal to the knots, using a scalpel under a microscope. The distance between the knot and the stripped insulation is varied according to the muscle being implanted. The knots must be tight to prevent movement of the exposure relative to the knot both during and post implantation. Pairs of electrodes that would be implanted in the same muscle are then threaded through colored beads and tied together at their distal end. The colored beads allow each channel to be identified during the implantation procedure. The electrode pairs that will be implanted in each leg (4 muscles for each leg) are bundled together and tied loosely with silk.

The assembly is tested at several steps during the construction using an impedance meter to verify that all exposures are clear and that channels are isolated from one another. The completed assembly is shown in Figure 1D. Once completed, the entire assembly is gas sterilized through a Sterrad® low temperature, hydrogen peroxide gas plasma sterilizer in preparation for implantation.

3.2 Implantation of EMG electrodes

Mice are anesthetized with isoflurane (1-3%) and prepared for aseptic surgery. An incision (~1cm) is made over the lumbar spine and over the lateral aspect of each knee. The skin is separated from the underlying fascia via a blunt scissors and the bundle of wires for each leg is tunneled subcutaneously from the back to each leg incision. Four 6-0 silk sutures (Covidien soft silk suture # S1174) are tied to four corners of the mesh and then sutured to the subdermal tissue of the back. The mesh is then passed through the incision so that the mesh is underneath the skin and the connector sitting above the skin surface. The subdermal sutures are then drawn tight and tied to the mesh. These sutures act to secure the assembly to the skin on the back and the mesh allows the connective tissue to grow around the implanted connector, helping maintain its position over time. The skin is pulled tight around the connector is then sutured to the connector using the holes drilled in the epoxy. If necessary, rostral and caudal skin margins can be closed with 5-0 nylon sutures and wound clips (ETHILON Nylon Sutures, Johnson & Johnson #661H; Reflex 7 Wound Clips, Fine Science Tools #12032-07).

After securing the connector on the back, the electrodes are then implanted in the muscles. Each wire is threaded through a small surgical needle (Kalt suture needles size 3, Fine Science Tools #12050-03). The needle is then threaded through the intended muscle perpendicular to the muscle fibers until the stripped region of the electrode is within the muscle tissue and the proximal knot pressed against one side of the muscle. Two electrodes are placed in each muscle at a distance of approximately 1mm (twice the exposure distance, (Loeb and Gans, 1986)). The two distal ends of the electrode are then tied together so that their common knot lies flush to the muscle, taking care to secure the electrode firmly within the muscle but without compressing the muscle tissue. We typically implant vastus lateralis (VL), posterior biceps femoris (BFp), tibialis anterior (TA), and lateral gastrocnemius (LG) in both legs. Each set of electrodes were placed in the thick portion of the muscle with these specifications; distal muscle belly of VL, mid-muscle belly of BFp and TA, and distal muscle belly of LG to best avoid interference from BFp. Extra wire is looped and pushed rostrally underneath the skin away from the leg incision and the leg incisions are closed with nylon sutures and wound clips. Because most of the assembly of the electrodes is done prior

to the surgery, these surgical procedures for the connector implantation and for the 8 muscles take only approximately 90 to 120 minutes to complete.

Approximately 15 minutes prior to the end of the surgical procedures, mice are given Meloxicam (1 mg/ml). Meloxicam is given daily for 48 hours after the surgery, and all animals recover from the procedures very quickly. Animals are housed in groups or alone with minimal firm obstructions within the cage in order to protect the connector. The mice are also provided consistent enrichment such as Shepherd Shacks®, Ancare Nestlets™, and food treats.

A significant issue in these experiments, especially for the chronic recordings performed here, was the tendency of all mice to remove the sutures on their hindlimbs. This tendency was observed for both strains of mice examined here and appeared to be independent of the suture material or surgical knots used to close the wound. Similarly, prolonging administration of Meloxicam did not prevent this behavior. In contrast, animals were relatively tolerant of the back connector over the first several weeks following implantation. We found that wound clips (Reflex 7 Wound Clips, Fine Science Tools #12032-07) were the most reliable way to ensure that wounds stayed closed, but even so we had to check animals daily for open wounds and make surgical repairs as necessary. Even with this oversight, reopening of the hindlimb surgical wounds was the most common failure mode for electrode recordings, particularly in TA and LG, in these experiments.

3.3 Data collection during treadmill locomotion

After recovery from the implantation (usually 1 week), animals are brought to the lab and placed on a motorized treadmill (Columbus Instruments). The back connector is attached to a cable and the signals amplified (X5000, A-M Systems Model 1700, Differential amplifier, Input impedance <1.5 MΩ, CMRR >80 dB) and filtered (30-1000Hz) prior to being sampled (2500Hz, National Instruments PCI-6229, 16 bit) using custom routines created in Labview (National Instruments). The cable is constructed of the female Omnetics connector soldered directly to the sixteen wires connected to the amplifier. Animals ambulated on the treadmill at speeds between 6m/min and 18m/min. All data is saved to disk for subsequent offline analysis. For general behavioral analysis, video recordings are made of the ambulating animal and synchronized to the collected EMGs using a digital pulse train collected along with the EMGs and used to increment a LED digital counter in the field of view of the video. Video is collected at 60Hz and saved on tape and captured to disk for later analysis. EMGs underwent full rectification for analysis, but recordings were not otherwise processed. Analyses are performed in Matlab (Mathworks, Natick, MA) using customized routines. Cross-talk appeared to be minimal in these recordings. Each channel showed the expected activation patterns from the implanted muscle and showed periods of independent activity. All recordings are shown after high pass digital filtering (2nd order Butterworth, 50Hz cutoff) to minimize movement artifact.

3.4 Kinematic analysis

In 4 animals we examined the consequences of the EMG implantation on the kinematics of treadmill locomotion. Following an initial session to acclimatize animals to the treadmill, mice were lightly anesthetized (isoflurane 2%) prior to the application of retroreflective markers (3mm) placed on the rostral tip of the pelvis, at the hip center of rotation, knee, ankle, and first metatarsal joint. The anesthesia was necessary to ensure consistent placement of the markers. The animal was then allowed to recover from anesthesia and placed on the motorized treadmill. During treadmill locomotion, the position of markers was tracked using a 4 camera Vicon motion capture system (100 Hz). Kinematics were tracked both prior to the EMG implantation and two weeks following the EMG implantation.

Kinematic data was analyzed off line. Periods in which markers were consistently tracked and the animal produced steady walking (cycle period less than 500ms) were then identified. Locomotor cycles were aligned at the onset of the swing phase. At least 8 cycles were measured for each animal. We quantified the global limb angle as the angle formed between the pelvis and the line connecting the hip and metatarsal markers. The global limb length was measured as the distance between the hip marker and the metatarsal marker. Step length and step height were quantified as the horizontal and vertical distances travelled by the metatarsal marker. T-tests were then used to compare these kinematic measures before and after EMG implantation.

3.5 Spinal cord injury

In addition to examining EMGs in intact animals, we evaluated the use of this design for recording EMG activity in mice with spinal cord injury. Adult female CD-1 mice (18-28g) were anesthetized using an inhalation anesthetic (2-3% isoflurane) and prepared for aseptic surgery. An incision was made to expose the T10 vertebral column and a laminectomy was performed at T10 to expose the T12-13 spinal segments. The clip compression injury model was used to create the spinal cord injury at spinal segment T13 (Joshi and Fehlings, 2002). This method uses calibrated clips to compress the spinal cord, with the amount of force applied by the clips allowing for different degrees of spinal cord injury. We used the moderate clip in order to produce an incomplete spinal injury. Following spinal injury, the wound was closed with wound clips. Animals were given Meloxicam (0.3 mg/kg, SQ) for 2 days following the spinal cord injury and bladders were expressed manually until bladder function recovered (~1 week). Following recovery from spinal cord injury for a period of at least 1 month, mice were implanted with EMG electrodes using the procedures described above.

3.6 Transgenic SOD1 mice

We examined the hindlimb EMG activity in a commonly used animal model of ALS (Gurney et al., 1994). These mice highly overexpress a point-mutated form of the human SOD1 gene (single amino acid substitution of glycine to alanine at codon 93 in the superoxide dismutase gene) driven by the endogenous human promoter. We compared the EMG activity in these symptomatic transgenic mice (SOD1^{G93A}) to a control group that overexpress the wildtype form of the human SOD1 gene (SOD1^{wt}). Recordings were made chronically for a period of 4-5 weeks overlapping the onset of symptoms in these animals.

Results

Figure 3A shows an example of the activation patterns recorded from 8 hindlimb muscles in an intact mouse during treadmill locomotion. The activity patterns seen in the figure show good modulation over baseline levels and the cyclic modulation patterns are as expected for the implanted muscles. Figure 3B shows the averaged muscle activity across multiple cycles of treadmill locomotion, again showing the good modulation of these muscles. These results show that the methods described here are able to measure EMG activity with good quality.

In 4 SOD1^{wt} B6SJL mice, we examined our ability to record the activity in muscles chronically over a 5-week period. Such chronic recordings are necessary to track disease progression in mouse models such as ALS, Parkinson's, or SMA and to evaluate the efficacy of potential interventions for these diseases. In the EMG recordings shown in Figure 4, the electrodes in the hindlimb muscles were maintained over 5 weeks following implantation. The figure shows the activity of these muscles from the first day of recording taken 1 week following the implantation surgery, then on the last day of recording taken 5 weeks following the surgery. As can be seen in the figure, the recordings on these muscles

remained very good and the general activity patterns on the muscles were very similar. Figure 5 shows the averaged activity in muscles from this same animal tracked across all days of recordings. The general activity pattern in each muscle remained consistent across days, although amplitudes of some muscles could vary. Similar results were observed in the 3 other animals examined. It should be noted that although 3 animals maintained good recordings in at least 5 muscles (6,6, and 5 muscles), in the fourth animal only 2 muscles remained viable. As mentioned above (see Methods), this failure was a consequence of the animal opening its hindlimb wound and severing implanted electrodes.

In an additional 4 CD-1 mice, we examined the effects of the EMG implantation on the kinematics of locomotion. As seen in Figure 6, several kinematic measures were altered following EMG implantation. In particular, the step length and the limb angle range of motion were both significantly reduced following EMG implantation by 22% and by 19%, respectively ($p < 0.05$). The step height and limb length were slightly reduced as well, although this reduction did not reach significance ($p > 0.05$). Similar changes have been observed with other techniques of EMG implantation which reported a decreased knee range of motion by 16% in their most successful electrode configuration which had only 4 pairs of wires (Pearson, Acharya, 2005).

Figure 7 illustrates the application of these techniques to characterizing muscle activations in animals following spinal cord injury, showing an example of EMG recordings taken 12 weeks following a moderate spinal cord injury (Figure 7A). EMGs were implanted 9 weeks following the initial spinal cord injury and the recordings were taken here after 3 weeks. Six muscles gave viable recordings at this time point. This animal had a high level of functional recovery (BBB score of 14 after 8 weeks) (Joshi and Fehlings, 2002). As seen in the figure, the activity of muscles within a limb was partially coordinated in this animal, with clear alternation between flexors and extensors. However, comparison with the patterns in Figure 3 shows clearly that the animal was still considerably impaired, with slow and irregular cycles and periods of coactivation between muscles. Further, although there was coordination between muscles within a limb, there was little coordination between muscles in different limbs, with muscles in the two limbs changing from in phase to out of phase activation regularly. This lack of interlimb coordination is illustrated in the averages shown in Figure 7B. These averages were aligned to the onset of the right TA muscle. As seen in the figure, although there was good coordination between the muscles in the right hindlimb, the activity in the left limb showed no consistent relationship. These recordings show that the methods described here can potentially allow us to better characterize the level of functional recovery in animals following spinal cord injury and how this recovery might be affected by rehabilitation strategies or pharmacological manipulations.

Figure 8 illustrates the utility of these methods for evaluating motor function in mice with degenerative motor neuron disease caused by mutation in the *SOD1^{G93A}* gene. In these mice, ALS symptom onset is usually considered to be at P90 (postnatal day 90) (Chiu et al., 1995). The figure shows an example of the muscle activation patterns recorded in the hindlimb muscles of a *SOD1^{G93A}* mouse, both prior to symptom onset (P69) and following symptom onset (P100). As illustrated in the figure, the EMG recordings were profoundly altered after the onset of ALS symptoms. In particular, in the later dates there were many large amplitude motor unit potentials that were absent in the presymptomatic recordings (note that the gains for each muscle are the same in the early and late recordings). Further, the dense interference patterns observed at P69 are absent in the EMGs recorded at P100 and many muscles have clear oscillatory tremors. These alterations are typical of the changes observed in ALS in humans (de Carvalho, Dengler, 2008). The chronic recordings used here allow us to track the development of these symptoms and their potential amelioration by pharmacological manipulations.

Discussion

We have described the design of a chronic EMG system suitable for the recording of a large number of muscles in the mouse hindlimb over extended periods of time. We demonstrated the ability of this array to record EMG activity in intact animals and in animals following spinal cord injury or with degenerative motor neuron disease. We documented that these recordings can be maintained for periods of at least one month, allowing for their use in characterizing disease progression or recovery of function following injury. These techniques should thus be useful in providing considerable information about the state of motor function in mouse models.

The design used in this study is an extension of techniques used in EMG recordings that have also been used in mice (Akay, Acharya, 2006, Courtine, Song, 2008, Fortier, Smith, 1987, Leblond, L'Esperance, 2003, Loeb and Gans, 1986, Masuki and Nose, 2003, Milner, Cadoret, 1995, Pearson, Acharya, 2005, Wernig, Irintchev, 1989). The novelty of the present study lies in, first, our ability to record from large numbers of muscles in the mouse and, second, our demonstrated ability to record from these muscles over extended periods or at least one month. As described in the introduction, most studies of mouse EMG patterns have recorded from at most 4 hindlimb muscles, in contrast to the recordings of 6-8 muscles that we demonstrated in this study. Also, most other studies show more acute recordings of up to around two weeks post implantation whereas we can record up to several months post implantation (Pearson, Acharya, 2005). Studies that have followed EMG recordings more chronically only published results from up to two muscles, whereas in this study, we show chronic EMG recordings of up to 6 muscles (Leblond, L'Esperance, 2003). Clearly, information from more muscles can provide invaluable information about motor function in normal and impaired animals, such as provided by the phase diagrams shown in Figure 3B. Similarly, information from different muscles can help better characterize the progression of degenerative diseases through the limb, for example assessing whether there are consistent spatial or temporal gradients of degeneration (Daube, 1985). The fact that most of the connector can be assembled prior to the implantation surgery makes it feasible to implant the large number of muscles examined here, minimizing the time necessary for surgical implantation for each animal.

The chronic stability of the recordings demonstrated here shows that this design can be used successfully to track the progression of diseases in mouse models, such as ALS or SMA (Cifuentes-Diaz et al., 2001, Gurney, Pu, 1994). This information can be important not only to better characterize the progression of the disease but can also be used potentially to develop methods for earlier diagnosis or to evaluate the efficacy of potential treatments. However, as illustrated in Figure 4, even in intact animals there is some variation in the EMG recordings over days. Although some of this variation might be due to differences in locomotor strategy over time rather than changes in recording configurations, it is possible that electrode position could change over time due to tissue healing and changes in inflammation. These observations suggest that comparison of EMG amplitudes over multiple days should be interpreted cautiously. Changes in the timing of muscles, however, will be less affected by changes in amplitude.

We did observe that the EMG implantation can affect the hindlimb kinematics produced by animals. In particular, the overall range of motion of the limb was reduced in the animals we examined in this study. Significant changes in hindlimb kinematics could have an effect on EMG timing and amplitude and, therefore, changes in hindlimb kinematics must be considered. The change in kinematics reported in this study is similar to what has already been published in comparable approaches (Pearson, Acharya, 2005). We find it notable that our approach, which affected kinematics similarly to past studies, involved implanting and

recording from twice as many muscles as the other study. It was clear that restricted limb motion resulted primarily from the wound healing based on observations of animals during experiments and from video recording. As described above, animals often removed hindlimb sutures and the process of reclosing the wound or replacing sutures often resulted in stretching of the skin over the thigh and a consequent reduction in the range of limb motion. Although we did not examine the issue systematically, it appeared that animals requiring less surgical repairs generally had less restricted range of motion. Although we attempted to use different suture material or suture types, this did not reduce the incidence of wounds being reopened. Note also that the primary failure mode of our electrodes was their damage by the animals when opening their incision. Additional approaches, such as using Elizabethan collars might be more successful at reducing the incidence of wound reopening and, therefore, lessen kinematic changes and increase numbers of recordable channels over time.

Taken together, the results shown here demonstrate the utility of our electrode design for characterizing muscle activation patterns in mice. However, there are several features of the design that might be improved in the future. First, our current design requires two needle penetrations for each implanted muscle, one for each wire. A recently described electrode design involving a single needle penetration could be integrated into our design in order to minimize potential muscle damage caused by the implantation procedure, although such a design does not allow easy customization of electrode characteristics (exposure size or interelectrode distance) for the implanted muscle (Pearson, Acharya, 2005). Second, there was no preamplification used in the recordings described here and signals were passed to the amplifier by ribbon cables to the differential amplifier. Adding a head stage close to the animal will likely improve the signal to noise of these recordings. Similarly, in the current recording configuration described here, the animal is tethered to the cabled system and an experimenter standing above the animal releases any torsion in the cable. Standard commutators (DragonFly) for the number of channels used in these studies cannot be used in mice because of their high effective inertia. However, the use of multiplexed systems (e.g. from Triangle Biosystems) that reduce the number of wires tethered to the animal could be used with low inertia commutators to allow for free motion of mice.

Although these considerations suggest ways that our electrode design can be improved in the future, the present study demonstrates that the current design is suitable for obtaining good quality, chronic EMG recordings in behaving mice. The assembly procedures described here are easy to implement and can be scaled to the large numbers of animals used in mouse studies. We believe that these techniques should be useful for many laboratories studying mouse motor function in both health and disease.

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Biography

Vicki M. Tysseling, PT, PhD is currently a Research Assistant Professor in the Physiology Department at Northwestern University. She graduated from the University of Iowa with a Masters in Physical Therapy in 1998 and began working as a physical therapist at the

Rehabilitation Institute of Chicago specializing in treating persons with spinal cord injury (SCI). In 2002, she returned to graduate school at Northwestern University to study molecular and cellular mechanisms of and treatments for SCI. After receiving her PhD in Neuroscience in 2008, she began as a Postdoctoral Fellow and then continued as a Research Assistant Professor to develop corresponding *in vivo* and *in vitro* methodologies of examining behavioral and spinal circuitry changes that occur after SCI and after treatment.

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Figure 1.

Assembly of EMG electrodes (A) Example of a fully assembled connector with ground just prior to sterilization. The left portion of the connector is implanted in the skin covering the lumbosacral area of the mouse. One bundle of wires each pass under the skin toward a leg. (B) Image showing the Omnetics portion fixed to a brass ring through epoxy and showing how a ground can be implemented. (C) Image showing the implanted connector in the skin of a CD-1 strain of mouse. (D) Image showing 3 mice post connector implantation.

Figure 2.

Illustration of the electrode (A) Sagittal (left) and top-down (right) illustrations of the proximal portion of the electrode. The pins and casing are embedded in epoxy with a separate ground pin and are all protected by a brass ring around the edge. Holes drilled in the epoxy allow the proximal connector to be attached to the skin. Distal to the ring, the wires are protected and secured to the mesh by additional epoxy. The mesh is placed under the skin and acts as an anchor for the connector. (B) Illustrations of the electrode before (left) and after (right) implantation into the muscle. Insulation is cleared 0.5 - 1mm distal to the knot during construction. The wires are threaded with a needle and separately implanted through the muscle. The wire ends are then tied in a knot and the superfluous wire is trimmed.

Figure 3.

Example of EMG recording during treadmill locomotion in intact CD-1 mouse. (A) EMG showing an example of several cycles of activity in the eight implanted muscles. See text for abbreviations. (B) EMG showing the averaged activity for the same animal over multiple cycles. Each cycle was aligned to the onset of the right TA, normalized to a constant duration and then averaged. EMG amplitude was normalized to vary between 0 and 1 for each muscle.

Figure 4.

Chronic stability of EMG recording. EMGs recorded in a control SOD1^{wt} B6SJL mouse 1 week (left) and 5 weeks (right) following implantation. The 6 muscles that were consistently recorded across the month of recordings are shown here. Gains for each muscle are the same both time points.

Figure 5.

The average cyclic muscle activity for animal shown in Figure 3. Each cycle of locomotion (at least 19 cycles for each day) was aligned to the onset of the right TA, normalized and averaged for each day over the 4-week period of recording. Note that although the phase of activation of each muscle was relatively similar across days, there were some variations in the amplitude of muscle activation.

Figure 6.

Influence of implanted connector on gait characteristics. Several kinematic measures were measured following EMG implantation in an intact CD-1 mouse. Step length and the limb angle range of motion were both significantly reduced following EMG implantation ($p < 0.05$ indicated by asterisks). The step height and limb length were slightly reduced as well, although this reduction did not reach significance ($p > 0.05$).

Figure 7.

An example of an EMG recording in a chronic spinal cord injured mouse during treadmill locomotion 12 weeks following a moderate spinal cord injury. The activity of muscles within a limb was partially coordinated in this animal, with clear alternation between flexors and extensors, however; also evident are slow and irregular cycles with periods of coactivation between muscles.

Figure 8.

An example of an EMG recording in a *SOD1*^{G93A} mouse before (left) and after (right) the development of ALS-like symptoms. Display gains are matched for each muscle for the two days of recording in order to illustrate the emergence of very large motor unit potentials in the recording at P100. Note also that some events in left VL recordings were clipped. Black bar below RLG burst notes example of oscillatory tremors.