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Contemporary linkages between EMG, kinetics and stroke rehabilitation

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Abstract

EMG and kinetic measures have been primary tools in the study of movement and have provided the foundation for much of the work presented in this journal. Recently, novel ways of combining these tools have provided opportunities to examine elements of motor learning and brain plasticity. This presentation reviews the quantification of EMG within the context of transcranial magnetic stimulation. This vehicle permits acquisition of measures that are fundamental to examining prospects for cortical reorganization among patients with stroke and employs a therapeutic approach called “constraint induced therapy” as a model to demonstrate the interpretation of changes in EMG measures among patients with stroke. Moreover, interfacing novel uses of kinetic measurements during functional task performances is highlighted to illustrate how EMG and kinetics can provide further insight into mechanisms related to reacquisition of movement and concomitant changes in plasticity. Clinicians and researchers interested in expanding their use of these measurement tools are encouraged to learn more about application possibilities.

1. Introduction

Throughout its distinguished history, the \textit{Journal of Electromyography and Kinesiology} has been an innovative forerunner in publishing original studies on topics as diversified as EMG–force interactions \cite{62}, EMG power spectral analyses \cite{22,42}, electrode placements \cite{37,45}, modeling \cite{25,69}, motor control \cite{15,29}, stretch reflex modulation \cite{89}, co-activation \cite{27,28,35}, fatigue \cite{47}, response to injury \cite{60}, spasticity \cite{33}, or a host of other intriguing topics. The approaches used in these studies all share in common a need to understand the processing a surface EMG signal typically recorded with electrodes over a muscle belly at prescribed distances and oriented to an end plate region \cite{3}. Indeed, much has been learned about the properties and function of muscle under normal and pathological conditions.

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1.1. EMG and stroke

A vast literature exists regarding the use of EMG to assess movement capabilities among patients with stroke (see for example [6,46,80]). Our group has had a particular interest in movement control of the upper extremity among patients with stroke [83]. We have used EMG feedback to identify physiological attributes among those chronic patients who would respond best to this form of training [81,84,88]. In fact, this identification led to the first inclusion criteria for the provision of “forced use,” [85] a precursor to “CI” (constraint induced movement) therapy applied to the more impaired upper extremities of patients with stroke [64,75].

1.2. Kinetics and stroke

A complementary approach to understanding movement can be found in the study of kinetics. The measurement of ground reaction forces during standing and gait in healthy subjects has led to a greater understanding of how the CNS maintains postural equilibrium [21] and allows humans to successfully maintain this equilibrium under all but the most extreme changes in the environment [4,24,36,78]. The analyses of fingertip forces during the performance of prehensile actions has facilitated our understanding of the highly efficient method of controlling grasping forces [30–32,73] during object manipulation.

The early studies investigating the control and coordination of grasping forces during dexterous actions indicated that healthy controls use primarily a feed forward mode of control to regulate the coupling of grasping forces [19,20,32]. The relatively rapid formation of the internal model of the dynamics of the upper extremity and the object being manipulated and the intended manipulation result in grip force being scaled to object weight, frictional characteristics, shape and changes in inertial forces on the object due to movement [17,18]. The elegant control of grasping forces requires an intact sensorimotor system. Basal ganglia dysfunction, as exhibited in Parkinson’s disease, has been shown to disrupt grip force scaling [16] and the coupling of grasping forces [1]. Similar results have been reported in patients with cerebellar degeneration [61].

Few systematic investigations of the effects of stroke on the control and coordination of grasping forces exist. Our most recent study [2] and those of Hermsdorfer [23,50] illustrate the importance of analyzing grasping forces throughout the performance of functional object manipulations. During simple unimanual lifts and manipulations patients with stroke can produce an excessive amount of grip force; however, most patients maintain the coupling between grip and load forces during these actions [23,50]. More recently, we developed a methodology to precisely quantify the grasping forces and torques produced during the performance of a functional upper extremity task (e.g., turning a key in a lock). Data were collected from two groups of sub-acute (3–9 months) patients with stroke, one group participating in an intensive two week constraint-induced therapy program while the other group served as controls and did not receive any structured therapy during this same two-week period. Patients, in both groups, produced highly irregular and inconsistent force and torque profiles across trials [2]. Post-test data indicated that the control group did not exhibit any change in the consistency of force or torque profiles during task performance. Four of five patients undergoing CI therapy exhibited a dramatic improvement in the control of grasping forces and torques and two of these patients exhibited grasping force modulation similar to that produced by healthy controls [2].

1.3. Transcranial magnetic stimulation introduced to JEK

In 2003 Mano et al. [43], introduced the readership of the Journal of Electromyography and Kinesiology to another application of electromyography that addresses a comparatively new territory, the interface between EMG and assessments of cortical reorganization. This
approach places EMG assessment and kinetic interfaces in a very new and exciting light by
now exploiting these parameters within the context of studying neuroplasticity. Accordingly,
the purposes of this presentation are to review the role of transcranial magnetic stimulation
(TMS) as a vehicle to study EMG evoked responses to brain stimulation, review the
importance of electrode placement to the interpretation of cortical mapping of movement,
and show how kinetics and EMG can be linked in this evolving area of study. As a result,
those scientists and clinicians with an interest in EMG and kinesiology will gain insights
into approaches that complement those investigative uses to which we have become
accustomed.

2. Cortical plasticity

To date there probably is no generally accepted definition of ‘plasticity’, although it may be
thought of as the brain’s capacity to change or as an intrinsic property of the human nervous
system that persists throughout the lifespan [57]. In most experimental situations, plasticity
is usually defined neurophysiologically by changes in the stimulus–response characteristics
(‘excitability’). Classen and Ziemann, in discussing stimulation-induced plasticity in the
human motor cortex [10], observe that ‘neuronal plasticity may be defined as any functional
change within the nervous system outlasting an (experimental) manipulation’. They also
made clear that there is no general agreement on how long an effect needs to outlast the
intervention, but reasonably assert that “plasticity is usually only applied when neuronal
changes outlast the manipulation by more that a few seconds” [10].

If plasticity does characterize the brain’s capacity to change, and is an intrinsic but persistent
property of the nervous system, one example might be housed in the acquisition of new
skills, seen especially in response to changes in the environment. This mechanism may
underlie growth, development and learning. Additionally, plastic changes may represent the
mechanism by which recovery of function occurs after central or peripheral nervous system
injury [7,34].

One manifestation of cortical plasticity can be observed in what might happen to the motor
cortex following interventions [13,49]. Typically these measures can be assessed with fMRI,
PET, EEG or magnetoencephalography. Another approach uses transcranial magnetic
stimulation (TMS). The responses evoked through this approach are particularly intriguing
to electromyographers and kinesiologists, because they are recorded using surface EMG. A
series of conversions can be made from the evoked potentials and the interpretation of these
responses has implications for suggesting the presence of cortical plasticity.

2.1. The cortical plasticity – EMG interface

Even prior to the publication of the article by Mano et al. [43], neuroscientists recognized
the role that EMG can play in assessing central nervous system plasticity. TMS had become
appreciated as an important treatment modality or evaluative tool [54]. For the latter, evoked
responses, or motor evoked potentials (MEP) are recorded from electrodes placed
strategically over a muscle of interest as maps related to specific movements were charted.
Eliciting MEPs is accomplished by providing a time-varying current passed through a
circuit, or coil, that induces an electric field in the underlying brain when placed over the
appropriate cortical location, such as the motor cortex. This electrical field is mediated by a
magnetic field that easily passes through cortical bone. The conical shaped electrical field
thus generated could activate axo-axonal connections that subsequently are thought to
engage the corticospinal tract.

Single cell studies show that the organization of the primate motor cortex is much less
discrete than that observed in the primary sensory areas. Individual cortico-spinal neurons
project to several muscles and the projections to any one muscle may be spread through a wide area of cortex and intermix with projection to neighboring muscles [56]. The result is a mosaic in which a general pattern of hand, arm and shoulder projections can be distinguished, but where true boundaries are imprecise.

2.2. TMS and EMG measurements

Single pulse TMS cannot provide this level of detail, but can give at least a gross idea of the somatotopy of the human motor cortex and distinguish between the position of the best points to hold the coil to activate muscles in shoulder, arm or hand as well as between face, arm and leg [72]. This process of finding the best point to activate various muscles can be standardize by marking a matrix of points on the scalp and then plotting the amplitude of EMG responses in various muscles obtained at each point (see Fig. 1).

Such maps provide three pieces of information; the optimal position to obtain the largest response, the center of gravity (COG) of the area, and the area of the scalp from which response can be obtained. The optimal position or hotspot presumably corresponds to the location of the most excitable population of neurons that project to the target muscles. The COG is the center of this representation and is often the same as or very close to, the hot spot (Fig. 2). The COG X coordinate is calculated by \[ \frac{\sum_{i=n} nMEP}{nMV} \] and COG Y coordinate is calculated by \[ \frac{\sum_{i=n} nMEP}{nMV} \] [72]. Where, (nMEP) represents the normalization of mean amplitudes for all coordinates for each participant divided by the mean amplitudes by the maximum mean amplitude. The normalized map volume (nMV) is calculated by adding all of the nMEPs and multiplying by the area. The X and Y coordinates for each active site are then multiplied by the normalized MEP amplitude \( (X \times nMEP \text{ and } Y \times nMEP) \), and the sum of all the values are calculated respectively.

The area of the representation is more complex and depends on two factors: the true area on the cortex where neurons are located that project to the target muscle and the stimulus intensity used to project the map. If stimulus intensity is too low, the total extent of map may be underestimated because less excitable elements will not be recruited. If the stimulus intensity is too high, the area will be overestimated because the stimulus current will spread beyond the point of stimulation [66]. These two opposing factors are difficult to reconcile with TMS, and the area of the map that is recorded is virtually meaningless [59].

Nearly all mapping studies recognize these inherent limitations in technique, and thus they focus not on the absolute size of a map, but on change in the maps resulting, for example, from a stroke or intervention. With the exception of a few studies, most interventions change the size of the map without affecting its hot spot [5,11,12,53,67]. In such cases, changes in the maps size are best observed with the subjects at complete rest.

2.3. Cortical plasticity – kinetics interface

While an understanding of TMS measurements made in patients with stroke requires the quantification and delineation of EMG responses manifest as changes in MEP threshold and amplitude along with increases in the size of motor maps and the shifting of the COG within these maps, additional data can be gleaned from analyses of kinetic studies. Recently, we sought to understand how CIT impacted cortical plasticity and dexterous function. Fig. 3A provides an illustration of the experimental paradigm used. A key was instrumented with a six degree-of-freedom Force/Torque transducer (FT axes shown in Fig. 3B). The locking mechanism within the lock was replaced with a spring in order to achieve a variable resistance throughout the action of turning the key.
Our results indicated that after a two-week CIT intervention the contralateral MEP amplitude increased as did the motor map area of that same cortical area [52]. Grasping forces changed as a result of the CIT intervention as well. Pre- and Post-test grasping force and torque data from a representative patient undergoing CIT is shown in the lower plots of Fig. 3. Prior to CIT the grasping forces and torques were highly irregular and were characterized by multiple oscillations. However, after CIT (plot D), the patient(s) improved the control of grasping forces and torques. Inspection of the grip force produced after CIT (solid line; plot D) indicates a bimodal profile. This bimodal profile suggests the patient is modulating grip force to take advantage of the unloading of the spring while turning it back to the original position. The use of TMS and kinetics, in this case, provided us with a better understanding of the potential mechanisms underlying enhanced motor recovery after stroke. Furthermore, this multi-faceted approach provided insight into specific movement parameters that change as a result of CIT. Future studies are planned in which the magnitude of change in MEP and motor maps and movement kinetics are monitored. These studies will enhance our understanding of the evolution of cortical plasticity and at what point do changes in motor performance (learning) occur.

3. Importance of electrode placements to specify meaning of MEP characteristics

Based upon where the stimulus intensity of stimulation is centered, the specificity of an evoked movement can be elicited from surface EMG electrodes. Typically, the latency to activation in upper extremity distal limb muscles is 20–25 ms. While TMS undoubtedly results in concurrent responses from other muscles [58], the response recorded between the reference electrodes is a function of the quality of the signal and the inter-electrode distances. With respect to the latter, most studies that have mapped responses to TMS have used a montage arrangement; that is, a reference electrode placed over the muscle belly and a second electrode over a distal point, usually a bony prominence. While this procedure might be standard for determining muscle response latencies to peripheral nerve stimulation [71], this array is non-specific for recording underlying muscle responses to cortical stimulation. Indeed, the montage arrangement would result in volume conducted activity, often from surrounding or deeper muscles that may be innervated by the same nerve but possess different functions.

Volume conduction, characterized by recording of potentials some distance from the muscle targeted by electrode placements has been observed in both reflex solicitation [26] and in occupational based activities [9] even in the presence of selective filtering. In addition to defining the frequency spectrum for which muscle potentials will be processed, maintaining inter-electrode distances of 2 cm or less [3,44] reduces the recording surface area.

Therefore, relatively close spaced electrodes promote a greater specificity of pick-up from underlying muscle while sacrificing the volume of muscle seen and hence the overall amplitude of an MEP evoked through cortical stimulation. Nonetheless, the recorded EMG data permits the delineation of several quantitative parameters. Fig. 4 highlights these concerns. Surface electrodes (recording area: 4 × 7 mm) were aligned along the right extensor digitorum communis (EDC) muscle and separated by approximately 1.5 cm. The muscle was isolated through palpation as the subject gently extended the fingers against mild resistance with the wrist fixed in flexion. Recordings were made as the subject was asked to flex and extend the fingers or wrist. For each effort, recordings were made from different electrode arrays. As can be seen from the analog recordings in Fig. 4 closer electrode arrays (2, 3) yielded more precise EMG which was even more specific to finger than wrist movements, while wider electrode arrays (1, 4 or 1, 5) produced more activity.
The 1, 5 electrode array was chosen because these electrode placements had been selected in one study used to create a cortical map from MEP responses in EDC [79].

To further expand the relationship between electrode placements and evoked MEPs from cortical stimulation over the motor cortex corresponding to the hot spot for EDC, the study shown in Fig. 5 was undertaken. EMG responses during voluntary movements seen in Fig. 5 are shown on the left for close (2, 3) and wider (1, 4, and 1, 5) electrode placements as well as the evoked MEPs from the cortical stimulus site at threshold (1.0 T) and 10 per cent above threshold (1.1 T) found when recording from the electrode 2, 3 location. Using those stimulation intensities, MEPs were now recorded from electrode sites 1, 4 and 1, 5. Clearly the amplitudes and wave forms are considerably larger and more complex. These enlargements are invariably due to volume conducted responses from surrounding musculature and the asynchronous nature in which motor units contributing to the MEP were engaged as spinal motor neurons were excited from activation of the corticospinal tract.

3.1. Mapping and cortical reorganization

These analog signals are often quantified in the determination of spatial and volumetric measurements of motor cortical areas representing a specific muscle or movement. In fact, the notion of cortical reorganization has been linked to changes in TMS maps [8,40,68]. The presumption is that changes in one or more of the parameters derived from TMS mapping procedures and defined earlier may be related to cortical plasticity. However, clinical and scientific electromyographers should realize that changes often referred to as “mass practice cortical reorganization”, that is, a change at the macro or microcellular level in cortical structure or activation resulting from one or more forms of repetitive task practice [38,39], as an example, may be more artificial than real, since the change could be due to contributions to MEP measurements from muscles that are extraneous to the intervention. For example, the abductor pollicis brevis is commonly mapped using TMS procedures [48] and the montage arrangement for recording is usually employed. When studying patients with stroke and claims are made that an expanded cortical map is the result of an intervention, then one must be assured that geometrically approximated muscles, such as the flexor pollicis brevis, adductor pollicis or interossei, are not contributing to this map since their heightened activation may be counterproductive.

Specifically more cortical activation of these muscles contributing to the MEP could be inappropriate functionally by suggesting an enhancement of flexor (hand closure) activity in patients who already have disinhibited flexor activity in the wrist and fingers.

4. Contemporary use of EMG and kinetics to assess cortical reorganization in stroke rehabilitation

There is mounting evidence that various forms of functional training, including repetitive task practice may induce improvement in movement capabilities. For example, knowledge of results through EMG or force feedback [74,77,87] can augment motor control, even when the EMG source of feedback is shaping of reflex responses [82,86]. The behavioral components that contribute to regaining upper extremity movement capabilities [65] also represent powerful influences on reacquisition of movement among patients with stroke.

More recent data support the belief that strengthening exercises imposed upon repetitive training can have an additive effect on functional return in the upper extremities of patients with acute stroke [76].

Constraint induced movement (CI) therapy shares elements of repetitive task practice and behavioral retraining to enhance function in the upper limbs of patients with chronic stroke.
This approach requires that patients with stroke undertake repetitive activities with the more impaired upper extremity while the contra-lateral limb is immobilized for most waking hours. This treatment approach can be performed in the absence of specific clinical supervision and training, that is, forced use [41,55], with formalized training, CI therapy [14,70], or with periodic, modified CI therapy [51].

The data shown in Fig. 1 reveal the amplitude of MEPs recorded from the EDC using an electrode placement comparable to the 2, 3 inter-electrode distance and location shown in Fig. 4. Responses were elicited by stimulating the hot spot over each cortex at 110% of the motor threshold. Threshold is defined as a response that exceeds 50 µV in at least 5 of 10 stimuli delivered at 0.2 Hz. MEP amplitude plots from the active sites for which responses greater than 50 µV are imposed on a two dimensional (flattened) view of the brain. Here the patient had sustained a stroke six months previously that impaired movement in his more affected, right arm and hand. At the time of initial mapping, there were more sites with greater amplitude in his left EDC evoked from stimulating the hot spot over his right motor cortex than recorded when stimulating the hot spot over his affected left motor cortex before a two week intervention (compare Fig. 1 upper left and right panels). Thereafter, the pattern had reversed (Fig. 1, lower panels) following constraint induced therapy treatment in which the better (left) arm was immobilized during most waking hours as the patient was provided with structured practice using his impaired right arm and hand. Now there were more active sites from which MEPs were evoked when stimulating at 110% motor threshold over the affected cortex.

5. Challenges

The expansion of possibilities in developing interfaces between EMG, kinetics and cortical mapping using TMS offers unique opportunities for clinicians and researchers to augment their knowledge about cortical plasticity while facilitating linkages between the art and science of neurorehabilitation. Those scientists with expertise in EMG can make use of these interfaces to better interpret responses that lend insight into changes in volume of brain active during selective movements. The inclusion of multiple recording sites allows the simultaneous acquisition of data from several muscles during single cortical site stimulation. This arrangement offers a chance to map multiple cortical sites by the numbers of responsive muscles [58] thereby yielding more information about cortical stimulation locations evoking muscle synergistic activation.

The challenge lies in training clinicians and scientists interested in learning this technology and in expanding their appreciation for the use of EMG. In addition, the cost of acquiring a TMS unit and ancillary equipment might be prohibitive for some individuals. Last, even with the availability of equipment and training, the ability to interpret the data in a meaningful fashion requires patience and perseverance. On the other hand, concurrent processing of kinetic information in the context of rehabilitation applications has taken on new dimension.

Currently, a user-friendly “turn-key” system to record simultaneously measures kinetics and EMG is unavailable for clinical use. However, as advances in force sensing and EMG recording continue, coupled with the development of hand-held computing technology, one could envision multi-functional systems that provide objective quantification of motor performance to emerge in clinical settings. The rehabilitation community should embrace these technological and methodological advances that will allow for the nearly instantaneous objective quantification of motor performance. These data will allow clinicians to continuously track the effectiveness of the intervention and shape the treatment of individual patients.
6. Summary

Mounting evidence suggests that quantified EMG can be used to compute characteristics of maps that define movement or muscle representation in the motor cortex using transcranial magnetic stimulation. Students of electromyography are at a distinct advantage because they understand EMG recording properties and the value of relating electrode placements to the interpretation of processed signals. These behaviors are essential to accurately assessing changes in maps and, as such, afford unique opportunities for clinicians and scientists to broaden their scope of interest. At the same time, assessments of kinetics can be blended with mapping efforts to provide a more comprehensive picture of motor (re)learning through the association of changes in force generation with changes in cortical representation of movement during functional activities. These relationships are highlighted in examples of EMG and kinetic applications among patients with stroke who are striving to regain skills in manipulation of objects in their environment.

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References


Biographies

Steven L. Wolf, Ph.D., PT, FAPTA, received his AB in Biology from Clark University in Worcester MA, his physical therapy certificate from Columbia University, an MS in physical therapy from Boston University, an MS in Anatomy University and his Ph.D. in neurophysiology from Emory University. He undertook a two year MDAA post-doctoral Fellowship at the Karolinska Institute in Stockholm studying microneurographic recording techniques. He has defined the selection criteria for the application of EMG biofeedback to successfully restore upper extremity function among chronic patients with stroke. His interests in feedback led to the comparison of center of pressure biofeedback with Tai Chi for falls reduction in older adults. Interests in EMG have led to a series of studies assessing
anatomical and physiological properties of human muscle compartments and the relationship of cortically induced evoked muscle potentials and motor cortex mapping of movement.

Andrew Butler received a B.Sc. degree in Biology in 1987 and a Ph.D. degree in Exercise Science (motor control) in 1995 from the University of Iowa. He obtained his M.S. in Physical Therapy from Texas Woman’s University in 1998. He was a post-doctoral fellow in the Department of Neurology at Heinrich-Heine University, Duesseldorf, Germany until 2001. He has been an Assistant Professor in the Department of Rehabilitation Medicine, Emory University School of Medicine since 2001. His research focuses primarily on how volitional movement, motor learning, and organized motor behavior are represented in the human brain. His current research interests include the study of novel therapeutic intervention on cortical motor reorganization following stroke using transcranial magnetic stimulation (TMS) and functional magnetic resonance imaging (fMRI).

Jay Alberts received a M.S. and Ph.D. in Kinesiology from Arizona State University. He is the Director of the Human Motor Control Laboratory at Georgia Institute of Technology, where he is an Assistant Professor in the School of Applied Physiology. His research focuses on the mechanisms underlying the control and coordination of grasping forces during prehensile actions with specific interests in the effects of stroke and Parkinson’s disease on the control of grasping forces.

Min-Wook Kim received a MD (1991) and a PhD (2003) from Seoul National University in Korea. He is currently an assistant professor in the Department of Physical Medicine and Rehabilitation at the Catholic University in Korea. He is a visiting scholar in the Department of Rehabilitation at the Emory University, Atlanta in USA (2004–2005). His research interests include transcranial magnetic stimulation and brain mapping.
Fig. 1.
A three-dimensional map of the representation of the extensor digitorum communis after stimulation of a patient who had sustained an infarct in the left posterior limb of the internal capsule six months previously resulting in right upper limb paresis. Maps were made either before (top row) or after (bottom row) two weeks of constraint induced movement therapy. The x-y grid represents the surface of the contralateral scalp, marked into 1 cm squares. The height of the bar on each square indicates the mean peak to peak EMG (µV) evoked at each point on the scalp for 10 stimuli delivered at 0.2 Hz. Before intervention EMG responses on the side of the stroke (stimulation of the left hemisphere, right upper panel) were smaller than those on the intact side (left upper panel). However following CIT, MEPs were larger in the impaired muscle (right lower panel) and were obtained from a wider area of scalp than those of the less impaired left hand (left lower panel). Stimulation was set to be 10% above resting threshold. A response required that at least 5/10 stimuli exceeded a peak-to-peak amplitude of 50 µV.
Fig. 2.
Contour maps of MEPs of the same data depicted in Fig. 1. The star represents the COG for the map. Contours of different shades of gray show amplitude of EMG. As above, prior to intervention EMG responses on the side of the stroke (upper left) were smaller than those on the intact side (upper right). However following CIT, MEPs were larger in the impaired muscle (lower right) and were obtained from a wider area of scalp than those of the less impaired left hand (lower left). In the drawings, the head is seen from above. All values are in microvolts.
Fig. 3.
Illustration of participant grasping a 6 DOF force/torque transducer with the key in the neutral position (Plot A). The cross-hatch arrow represents initial position of the key, solid arrow represents the clockwise turn of the key to the 90-degree position and the open arrow represents the counter-clockwise turn to the 90-degree position. Plot B depicts the forces and torques about each axis that were measured. Plot C contains representative grasping force and torque profiles for a patient with stroke prior to undergoing CIT. Plot D contains force and torque data from the same patient after two-weeks of CIT.
Fig. 4.
Electrode placements for extensor digitorum communis recording. EMG is recorded by comparing closely spaced (2, 3) and wider spaced (1, 4 or 1, 5) electrode arrays during finger (left) and wrist (right) flexion and extension movements. The beginning and end of each movement is designated by arrows. Note comparatively greater activity during extension than flexion movement and during wrist rather than finger movement, indicative of volume conduction during the extension phase even with closely (2, 3) placed electrodes.
Fig. 5.
EMG recording arrays (as in Fig. 4), during finger flexion and extension movements (left) and evoked MEPs at motor threshold (1.0 T) and 10% above (1.1 T). Note stimulus artifact and onset of response (arrow). Amplitudes are larger at higher stimulus strength and duration of MEPS longer as a function of inter-electrode distance.