Muscle Motor Unit Conduction Velocity

- Measurements & Modelling –

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Summary

Electromyography (EMG) can be used to monitor the smallest units in a muscle that can be controlled by the central nervous system: the motor units. In the past most studies focusing on these motor units used invasive techniques to record the EMG. Using modern array surface EMG it was shown that this level of detail can also be monitored non-invasively.

In this masters thesis a set-up, using conventional electrodes, for non-invasive detection of single motor unit activity has been developed. We introduced a method for the non-invasive stimulation of single motor units in the muscle flexor digitorum superficialis. This muscle we found to be excellent for these stimulated EMG recordings.

The measured signals of motor unit activity have been analysed using a singular value decomposition. This way we found that a clear separation between active motor units in threshold stimulated EMG can be made. The number of active motor units has been accurately estimated and the average response for every active single motor unit has been reconstructed.

Using the averaged responses obtained in this way, we were able to determine the single motor unit conduction velocity and proved the repeatability of single motor unit stimulation.

Furthermore an abstract model describing the lower arm has been taken from literature and improved. The new model is used to estimate parameters describing the characteristics of single motor units. The model generates a look-up-table for different possible sets of parameters for a MU. Using a least squares method we showed that the model is able to obtain values for these parameters that are within the physical range given for these parameters.

The developed techniques enable the non-invasive stimulation, detection, and characterisation of single motor units. The new techniques can be of great value in future studies in the effects of e.g. aging starvation, exercise, or disease on the electro physiology of muscles.
Samenvatting

Elektromyografie (EMG) kan worden gebruikt voor het in beeld brengen van activiteit van de kleinste eenheden in een spier die door het centrale zenuwstelsel kunnen worden aangestuurd: de motor units. In het verleden hebben de meeste studies met betrekking tot deze motor units gebruik gemaakt van invasieve technieken. Met behulp van modernere array-oppervlakte EMG is echter aangetoond dat activiteit van deze motor units ook niet-invasief gemeten kan worden.

In dit afstudeerproject is een opstelling ontworpen, die gebruik maakt van standaard electrodes, voor niet-invasieve registratie van de activiteit van een enkele motor unit. Er is een methode ontwikkeld voor de niet-invasieve stimulatie van een enkele motor unit in de flexor digitorum superficialis spier. Deze spier bleek uitstekende eigenschappen te hebben voor het mogelijk maken van deze stimulaties. De bij deze stimulatie gemeten signalen, afkomstig van enkele motor units, zijn geanalyseerd met behulp van een singular value decomposition. Zo was het mogelijk een duidelijk onderscheid te maken tussen de verschillende motor units die actief zijn bij de contractie. Het aantal motor units dat is betrokken bij een bepaalde contractie kan nu nauwkeurig worden bepaald en de gemiddelde respons van iedere motor unit kan worden gereconstrueerd.

Met behulp van deze reconstructies kan de geleidingssnelheid van een enkele motor unit worden bepaald en kan de reproduceerbaarheid van het stimuleren van een enkele motor unit worden aangetoond.

Ook is een model van de onderarm uit de literatuur verbeterd. Het zo verkregen model kan worden gebruikt voor het schatten van parameters die een motor unit karakteriseren. Het model vormt een ‘look-up-table’ voor alle mogelijke combinaties van parameters. Met behulp van een kleinste-kwadraten-methode worden de karakteristieke parameters van de waargenomen motor units bepaald. De hiermee verkregen waarden voor deze parameters liggen binnen de anatomische en fysiologische waarden voor deze parameters.

De ontwikkelde technieken maken het mogelijk om een enkele motor unit niet-invasief te stimuleren, meten en karakteriseren. Deze technieken kunnen van grote waarde zijn in toekomstige studies naar de gevolgen van bijvoorbeeld veroudering, voedseltekort, inspanning of ziekte op de elektrofysiologie van spieren.
Introduction

The use of EMG in the clinic has proven to be of great importance in diagnosing neuronal and muscular diseases. Surface EMG (SEMG), as opposed to needle EMG, has the advantage of being non-invasive and is able to provide the clinician with information on the state of the underlying muscles. For a long time, one of the drawbacks of surface EMG has been the lack of specificity, for example the inability to study single motor units.

Motor units (MU’s) are the smallest units in a muscle that can be controlled by the central nervous system. Motor Unit Conduction Velocity (MUCV), motor unit depth, and motor unit size are parameters that characterize a Motor Unit (MU). Changes in some of these (e.g. MUCV) are thought to play a role in force, function and coordination losses in muscle diseases, in metabolic diseases (e.g. diabetes) and during aging or fatigue.

EMG reflects the signals originating of such MU’s. However, the relation between the initial MU-signal and the EMG is a complex one, many factors deform the signal while it travels from the MU to the EMG-electrode on the skin. When only a small number (or one) of MU’s is active during contraction, a reliable estimate of the parameters of this MU should be possible.

The aim of this project is twofold:

- A set-up needs to be developed for the non-invasive detection of single MU activity. For this a method to electrically stimulate a single MU (non-invasive) is also required. The set-up, in contrast to array EMG, is kept as basic as possible by using only a small number of (conventional) electrodes.
- Parameters characterizing a MU (like MUCV, MU depth, and MU size) need to be estimated. Using an abstract model of the tissues surrounding a MU, the inverse problem needs to be solved. This implies comparing the recorded surface EMG to artificially simulated EMG.

Firstly the physiological anatomy of skeletal muscle will be described to get an understanding of the processes which need to be modelled. Then an overview of previous research will lead to the state at which the current measuring and modelling techniques are. In chapter three the specific set-ups required to register voluntary and stimulated SEMG will be described. With these set-ups SEMG signals are measured in healthy subjects and evaluated for parameters by a model which is described in chapter four. The evaluation and parameter extraction is described in chapter five.
Chapter 1:  
Physiological anatomy of skeletal muscle

Skeletal muscle is the most abundant tissue in the human body. Skeletal muscles can perform contractions and by doing so apply forces to bones. This enables the body to move. The forces that can be applied depend on the contractile properties of the muscle and the muscle size. In this masters thesis one of the contractile properties, being the conduction velocity, will be investigated. This chapter describes the structure of skeletal muscle to a level detailed enough for later modelling of electrical phenomena associated with muscle contraction (Chapter 4).

A muscle is made up of several fascicles, each consisting of multiple muscle fibers (Fig 1.1.).

![Figure 1.1. Muscle anatomy (1)](image)

The innervation of muscles occurs at the single fiber level. Impulses from the central nervous system pass through one or more nerves to finally activate a motor neuron. This motor neuron is the connection of the nervous system to the muscular system. Once the motor neuron fires, the resulting depolarisation branches many times and stimulates from three to several hundred skeletal muscle fibers. The connection between the motor neuron and the muscle fiber is called the motor-end-plate. From this plate, located approximately in the middle of the fiber, the depolarisation spreads in both directions over the fiber membrane.

The depolarisation is the result of a changing ion-conductivity of the membrane causing ion-concentration changes in the muscle fiber. The depolarisation does not only involve the outer layer of the fiber, but is also transferred deeper into the fiber by the T-tubuli. Because of the depolarisation, large quantities of calcium are released from the sarcoplasmic reticulum into the sarcoplasm surrounding the myofibrils. This calcium initiates attractive forces between the actin and myosin filaments causing them to slide and by doing so contract the muscle.

If a muscle fiber is not stimulated, there is a constant potential difference between the inside and the outside of the fiber. (app. –90 mV) This potential difference is the Rest Membrane Potential (RMP). The RMP results from a slightly larger number of negative charges than positive charges inside the muscle, and slightly more positive charges outside. The main reasons for this different number of charges are:

- The difference in ion concentration of the intra- and extracellular fluid
- The relative permeability of the fiber membrane for these ions
- The \(\text{Na}^+/\text{K}^+\) pump.

Sodium (\(\text{Na}^+\)) and potassium (\(\text{K}^+\)) are the ions present in the highest concentration. Therefore they have the biggest contribution to the RMP. For every ion there is a balance of two forces acting upon it. On one hand there is the concentration gradient moving the ions in the direction of the lowest ion-concentration. On the other hand, there is the electric force that tries to equal the potential difference over the membrane.
The membrane potential at which the electrical force is equal in magnitude but opposite in direction to the concentration force is called the equilibrium potential. This equilibrium potential is of course dependent on the permeability of the membrane to the ion. This balance exists for all ions in and around the muscle fiber. The net potential difference of all ions contributing to the equilibrium is then enlarged by the sodium/potassium pump, actively increasing the electric gradient. Finally, the resulting equilibrium of all ions and this pump together determine the RMP.

When a muscle fiber is depolarised, the normal RMP is suddenly changed to approximately +10 mV. The spread of this depolarisation (in time) is called the single fiber action potential (SFAP). The main reason for the depolarisation is a sudden change in membrane conductivity for Na$^+$ and K$^+$ ions. At the motor end plate a large quantity of Na$^+$ enters the muscle fiber and causes the membrane conductivity (for Na$^+$) to change and let even more Na$^+$ flow in. This exchange of ions between the extracellular and intracellular cancels the normal resting potential difference between them. After depolarisation the Na$^+$ conductivity of the membrane is restored locally and the membrane returns to the resting state, but an adjacent part of the membrane is now being depolarised in the same way. The term for the velocity at which this depolarisation moves over the membrane is the muscle fiber conduction velocity (MFCV).

This velocity depends on a number of factors, e.g. fiber diameter and fiber type, but is typically in the order of 3-5 m/s. (Guyton, 1996)

Since one motor neuron branches to several muscle fibers, there is a unit of simultaneously activated fibers. The motor neuron, the motor-end-plate and all muscle fibers that can be activated by this motor neuron together are called a motor unit (MU).

![Figure 1.2. Spatial distribution of muscle fibers within one MU.](image)

*Figure 1.2. Spatial distribution of a muscle fibers within one MU. One motor neuron connects the central nervous system (a) to a motor end plate located on the muscle (b). Here the motor neuron activates several muscle fibers (one MU). The fibers belonging to one MU can overlap in a cross-section of the muscle (c) with fibers belonging to a different MU. (Roeleveld, 1997)*

One motor nerve branches and activates several muscle fibers. In a cross-section it can be seen (Fig 1.2.) that the fibers are not necessarily spatially located together but can also overlap with other MU’s.
Chapter 2: Previous Research

During the depolarisation and repolarisation of the membrane, there is an outward and an inward flow of ions (mainly Na\(^{+}\) and K\(^{+}\)) over the membrane resulting in a current through the membrane. This current causes extracellular potential fields in the medium surrounding the fiber. The quantitative description of these extracellular potential-fields is of obvious interest in electrophysiology. These field make it possible to detect muscle activity without actually having to puncture the muscle. The technique that measures the potential fields on the skin is called surface electromyography (SEMG). A lot of research has been done on how to accurately measure and model the signals obtained by SEMG. A brief overview of topics relevant to this study are summarized in this chapter.

§2.1. Modelling and Measurement-techniques history

Already in the 19\(^{th}\) century, Hermann introduced the core-conductor model. This model, which is still used today, is a one-dimensional model of the relationship between intra- and extra-cellular potential in nerve fibers. His theory can also be applied to muscle fibers (Hermann, 1879).

In 1969, Rosenfalck showed in his thesis that information of the intracellular potential was also sufficient to calculate extracellular potential fields (This can also be done by using the tripole model that will be described in paragraph 4.1). He also introduced anisotropy of the muscle tissue in his volume conductor model. This anisotropy is caused by other fibers lying parallel to the active fiber, which complicates the conduction in the radial direction. This reduces the conductivity in this radial direction. Another important improvement to Hermann's model was made when Rosenfalck changed from an infinite conductor to a radially bounded cylinder (Rosenfalck, 1969). Hereby incorporating that a finite conductor shows 'potential reflections' at boundaries.

In 1974 Plonsey applied the intracellular action potential measured in a crayfish axon using Rosenfalck's equations. This resulted in a plot of the extracellular potential field (Fig 2.1.).

![Figure 2.1. Extracellular potential field around a crayfish axon (Plonsey, 1974). The figure shows a 2 dimensional plot of the extracellular potential at one point in time. On the Y-axis is the radial distance to the center of the simulated fiber (Note that the fiber has a radius of 0.006 cm). For values smaller than this, the intracellular potential is indicated by the vertical lines. On the X-axis is the longitudinal coordinate (cm). Measured parallel to the fiber, Equipotential lines (µV) are shown for Y>0.006 cm. The intracellular potential ranges from 50 µV at X=0.1cm to 113800 µV for X=0.6 cm and resembles figure 4.1C.](image)

One of the first papers on the potential fields caused by motor units instead of single muscle fibers was that of Griep (1982). His model was based on a superposition of several muscle fiber potentials. He determined the locations of these fibers (belonging to one MU) by histochemical labelling. This enabled him to calculate the summation of the potential fields of all fibers accurately (Griep, 1982).
Apart from ways to model the EMG, the technique to accurately measure EMG has also been investigated. One of the drawbacks of SEMG is that the detection area of every electrode depends on its size. Electrodes having diameters of 15-30 mm and later even 5-10 mm still are very large compared to the size of a motor unit. This makes it almost impossible to detect single MU activity during voluntary contractions. Reucher in 1987 described the possibility of using closely located electrode configurations to create a spatial filter capable of reducing the pick up area. Results showed that using this technique (one of the first electrode grids) made detection of single MU activity possible at low voluntary contraction levels (Reucher, 1987).

**Figure 2.2. Electrode grids used by Reucher**

In 1990 Gootzen described in his thesis an expansion to the radially bounded volume conductor model of Rosenfalck. He proposed a more realistic model of a two-compartment volume. Instead of the previously used homogeneous volume conductor, his model used specific properties of muscle and fat tissue. For the first time the muscle fibers were modelled with a finite length. This model showed that some previously unexplained surface EMG recordings could be explained by the finite length of the muscle. (Gootzen, 1990)

Fuglevand (1992) constructed a model to investigate the effect of electrode size and spacing on the detection of a motor unit action potential with surface electrodes. He concluded that (Fuglevand, 1992)

1. Only those muscle fibers located closer than 12 mm to the electrode will contribute significantly to the measured surface EMG.
2. Variation in electrode size has almost no influence on the detection depth of motor units.
3. Increased electrode spacing increases detection depth.
4. The frequency content of action potentials decreases for more distant motor units.

Disselhorst-Klug (in 1997) expanded the work of Reucher on the effects of different spatial filters on the spatial resolution of the recording set-up. She suggested the use of an electrode grid in combination with a special IB$^2$ filter for a higher spatial resolution (Fig 2.3).

**Figure 2.3. The IB$^2$ filter.**

Rau and Disselhorst-Klug summarized the techniques for spatial filtering and grid-use. They introduced a new term for it; High Spatial Resolution (HSR) EMG (Rau, 1997).

Also in 1997, part of Roeleveld’s thesis contained an evaluation of different volume conductor models. She found that an expansion of the previous mentioned model by Gootzen, by adding a third layer of skin, gave the best resemblance to measured MU action potentials (Roeleveld, 1997).

Saitou (1999) published an article on the inverse analysis of surface EMG. He used a very simple model for the forward analysis. This model runs many calculations for slightly different MU parameters (size, depth, source strength) and the resulting potentials are matched on a measured MU EMG signal. The match with the highest correlation is said to be the best estimate for the actual parameters of this MU (Saitou, 1999).
Using the models that have been developed in the past, this study derives a complex model to describe extracellular potential fields. Our model is based mainly on the three layered model of Roeleveld (Roeleveld, 1997), with the difference that previously used obsolete terms are left out. Saitou’s method of performing an inverse analysis is then used to determine the parameters of the MU under investigation (Saitou, 1999).

§2.2. Clinical history

Simultaneously with the development of these new filters and EMG analysis techniques, clinical studies tried to link diseases or physiological phenomena to recorded parameter differences between patients and healthy volunteers.

In 1971, Cunningham performed a study on critically ill patients and measured their resting transmembrane potential. He found that in the critically ill patients this potential was significantly lower than in a control group. Muscle biopsies showed that this might be because of increased intracellular sodium and chloride concentrations (Cunningham, 1971). Several years later, in 1987, Bolton found, using needle EMG, that abnormal patterns in EMG recordings might be related to an accelerated breakdown of skeletal muscle protein. He suggested a combination of electrophysiological, morphological and biochemical techniques to get a better understanding of this relation (Bolton, 1987). Needle EMG has also been used to study which motor units are successively recruited if force is slowly increased. This is called a MU recruitment pattern (Søgaard, 1995).

Carsten (1988) related changes in ion-gradients and pH to changes in muscle fiber conduction velocity (measured with intramuscular electrodes) and found that intracellular pH and extracellular potassium change conduction velocity (Carsten, 1988). An overview of techniques for measuring conduction velocity was published by Arendt-Nielsen. He claimed that with surface EMG an estimate of the average muscle fiber conduction velocity (MFCV) of many motor units can be acquired. This could be done at all contraction levels, but did not yet have the High Spatial Resolution technique (§2.1) to distinct between different MU’s (Arendt-Nielsen, 1989).

In 1999 the American Association of Electrodiagnostic Medicine (AAEM) published a technology review concluding that there was almost no literature to support the use of surface EMG in the clinical diagnosis of muscle or nerve disease. This review contained all literature found for the years 1964 to 1994 on this topic, excluding the conduction velocity related studies. Rau and Disselhorst-Klug (1997) used their newly developed HSR-EMG technique in diagnosing specific neuronal and muscular disorders, classifying 97% correctly. Using a model capable of simulating certain neuronal and muscular disorders in 1998 they were able to accurately simulate these disorders (Disselhorst-Klug, 1998).

Zwarts in 2000 commented on the review of the AAEM and claimed that estimation of MFCV was already proven to be of diagnostic value in several myopathies. He stated that surface EMG arrays are currently capable of obtaining information on MU parameters that were previously only accessible by Needle EMG (Zwarts, 2000).

In conclusion this means there is evidence that parameters derived from the surface EMG signal contain information on physiological processes underlying the EMG. Obtaining these parameters could help diagnosing changes in the underlying processes.
Chapter 3: EMG measurements

During voluntary muscle contraction it is possible to record the potential fluctuations on the skin that cause this contraction. These fluctuations, the EMG signal, are a summation of all active electric signals produced by the body. However, this summation is weighted by the distance between the signals source and the recording electrode. Therefore only signals of tissues lying close to the electrode contribute significantly to the EMG signal. Some general characteristics of a surface-EMG signal are a peak to peak amplitude of 0 to 10 mV and a bandwidth of the signal that is almost entirely limited to the 0 to 150 Hz frequency range.

In order to register or visualise EMG signals, they are picked up by electrodes, amplified and filtered. The resulting signal can then either be visualised or be converted to a digital signal that can be used for further processing.

Recording single MU activity in a muscle with a set-up consisting of only a small number of electrodes with a diameter that is relatively large compared to state of the art electrodes (such as used in arrays), asks for special attention in the selection of the muscle. There are several conditions the muscle needs to comply with:

- The muscle fiber orientation needs to be known, in order to place the electrodes directly above the muscle.
- The muscle is located not too deep underneath the skin, with a motor-end-plate that is also close to the skin. This way electrical pulses can stimulate the nerve fibers and activate the MU’s.
- The length of the muscle should be large enough to allow for at least three electrodes being placed in between the motor-end-plate and the muscle/tendon transition.

All these conditions are met in the muscle flexor digitorum superficialis, proving to be excellent for the study of this single MU activity. In this study, all real-life measurements have been performed on the flexor digitorum superficialis muscle (Appendix B). This muscle lies close to the skin-surface and has fibers that run almost parallel with the lower arm. The muscle-tendon transition is located approximately 10 cm from the wrist and the motor-end-plate approximately 15 cm. Electrodes that need to pick up the signal of this muscle can therefore best be placed in the 5 cm in between these points. A bipolar electrode configuration is used to reduce ambient noise. In this configuration two electrodes are used to obtain one differential EMG signal. The signal of electrode at the greatest distance from the motor-end-plate is subtracted from the other electrodes signal. Measurements are performed using circular Ag/AgCl electrodes (Meditrace ²200, Tyco healthcare Canada).

§3.1.: Voluntary contraction

During a voluntary contraction of the flexor digitorum superficialis the EMG can be recorded. (Fig 3.1.)

During the experiment the subject (age 22, male) is asked to bend and stretch his ring finger, during each movement the flexor digitorum superficialis muscle contracts. Each contraction is picked up by the electrodes.
The signal is differentially amplified with a gain of 2340 (de Bekker, 2002). The analog signal is then sampled at 32 kHz, first order low-pass filtered (550 Hz cut-off frequency) and stored. The amplified maximum voluntary contraction (MVC) has a peak to peak voltage value of approximately 14 V. (2340 * 6 mV) Random contractions will therefore not have a maximum value of more than 7 V. Signal to noise ratio at MVC is approximately 100:1.

§3.2: Conduction velocity measurements

When the conduction velocity (CV) is assumed constant over the length of the muscle, the conduction velocity can be determined by simultaneously recording the EMG at two positions above the muscle. Finding the maximum correlation, at different time-shifts, between the two measured signals results in the time the muscle needs to depolarize over the inter-electrode distance. Conduction velocity is then determined by dividing the inter-electrode distance by the measured time delay.

For measuring two EMG signals, three electrodes above the muscle and one distant reference electrode are needed. In order to reduce the inter-electrode distance the electrodes have been modified to a bar-shape (22*11 mm) (Except the reference electrode). This has a small negative effect on the signal to noise ratio, however this effect is negligible. All three electrodes should not only be located above the muscle, but also in between the motor end plate and the muscle to tendon transition. How to locate these points will be described in §3.3.

![Figure 3.2. Conduction velocity.](image)

For the example shown in figure 3.2., the inter electrode distance is 12 mm center-to-center. The recorded time delay is 3.5 ms, and thus the CV is 3.43 m/s.

After repeatedly performing these contractions several times (n=20) a mean CV of 3.78 m/s was calculated (σ = 0.23) for this subject. Since not all contractions are identical, the number of MU’s as well as the location of the MU’s involved in the contraction can not be considered constant. This explains for the large variation in recorded CV. Moreover, there is no information on the activation times of the MU’s, since the motor neuron is stimulated by pulses coming from the central nervous system.

In order to control the number of active MU and the activation time, stimulated contractions can be used.

§3.3: Stimulated contraction

Excitable tissue (e.g. nerves) can be activated by applying an electric pulse to the skin. This is done by depolarizing the membrane to a threshold voltage level at which the mechanisms of the action potential take over. In the most simple way this is done by placing two electrodes very close to the fiber.
During the stimulus a current will travel through the connecting wires (Fig 3.3.). Stimulation of the fiber (muscle or nerve) will occur when the transmembrane potential at some point of the fiber reaches the depolarisation threshold. (Purves, 2001)

The same mechanism also causes depolarisation when the electrodes are placed further away from the fiber (field stimulation, table 3.1). If the electrodes are placed on the skin it is also possible to depolarise fibres. This needs a larger current (Table 3.1).  

<table>
<thead>
<tr>
<th>Location</th>
<th>Current Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracellular</td>
<td>1 nA – 10 nA</td>
</tr>
<tr>
<td>Grease gap, sucrose gap</td>
<td>0.01 μA – 1 μA</td>
</tr>
<tr>
<td>Suction electrode</td>
<td>10 μA – 1 mA</td>
</tr>
<tr>
<td>Monopolar with small cathode pushed amongst the nerve fibres</td>
<td>50 μA – 1 mA</td>
</tr>
<tr>
<td>Bipolar stimulation under paraffin oil</td>
<td>50 μA – 2 mA</td>
</tr>
<tr>
<td>Bipolar stimulation in volume conductor (saline or tissue)</td>
<td>1 mA – 20 mA</td>
</tr>
<tr>
<td>Transcutaneous stimulation</td>
<td>2 mA – 20 mA</td>
</tr>
<tr>
<td>Field stimulation</td>
<td>50 mA – 500 mA</td>
</tr>
</tbody>
</table>

Table 3.1. Current required for stimulation, depending on location of the stimulus electrodes and the nerve, a different current is needed to activate the nerve. The table shows values ranging from electrodes placed inside the fiber (intracellular) to more distant placed electrodes (field stimulation) (Purves, 2001)

By placing an electrode on the skin above the motor-end-plate of the flexor digitorum superficialis and another electrode at the back of the arm, the nerve activating the motor-end-plate can be depolarized. This will cause the flexor digitorum superficialis to contract (Fig 3.4).

In this study the OMRON HV-F116 stimulator is used (Fig 3.4.). This is a common nerve stimulator that is available on the market.

If the stimulus electrode is not positioned above the motor end plate, the muscle will not contract. Therefore this can be used to determine the location of the motor end plate. After positioning the stimulus electrodes, a strong pulse is applied. If no contraction can be seen the electrodes are slightly moved until a contraction appears.

After this, the muscle tendon transition can also be determined. If two electrodes are placed close to the stimulus electrode, both electrodes show a similar but delayed signal. If one of the electrodes is now moved away from the stimulus electrode (in small steps), at one point the signal will start to deform. This is the muscle tendon transition point. The signal deforms because tendons do not depolarize and the muscle contraction stops at this point.
For now, the assumption is made that if the stimulus amplitude is held constant during measurements the effect on the tissue under the electrodes is the same for every subsequent pulse, resulting in a constant number of MU’s being activated. The timing of the onset of the stimulus determines the activation time of the MU.

The effect of a constant amplitude stimulus on the sub lying nerves depends highly on the positioning of the electrodes, and the composition and position of the arm. Therefore no absolute values for the used stimuli are given. Reproducibility at these exact values is very low. Instead slight changes (increase/decrease) to threshold nerve activation levels will be used to describe the used stimulus changes.

![Image](image.png)

*Figure 3.4. Stimulated contractions can be measured using the same electrode configuration but with an extra nerve-stimulator.*

In the resulting EMG recording, the stimulus is superimposed on the EMG signal (Fig 3.5.). When recording at two positions to determine the CV, the stimulus is seen simultaneously at both positions. This is because the stimulus does not need to ‘travel’ over the body by depolarising tissues, it is simply conducted by tissues. The stimulus artefact can thus be used as a trigger for the nerve enervation time.

![Graph](graph.png)

*Figure 3.5. Example of a signal recorded prior to, during and after a stimulus.*

Comparing the response of a stimulated contraction (Fig 3.5.) to a voluntary contraction (Fig 3.2.), it can be seen that the stimulated contraction is much more structured. The stimulated contraction is also easier to reproduce since during voluntary contraction the timing of muscle activation is much less determined. Therefore, CV measurements are preferably performed on stimulated contractions.

As mentioned before, the number of MU’s activated by a stimulus depends on the stimulus amplitude. If the stimulus is very low no response of the muscle can be seen in the EMG, i.e. no MU are triggered. For a slightly increased stimulus a response starts to appear in the EMG (Fig 3.6.). This is the result of one or a few MU’s becoming activated by the stimulus. Increasing the stimulus more and more causes the response to increase as well, up to a point were all MU’s are activated by the stimulus. However this is not included in this experiment since the amplifiers are overloaded in this case by the stimulus and can not register the response curve.
Knowing this, it is possible to look at the contributions that every additionally activated MU has to the EMG. By subtracting two EMG signals recorded with slightly different stimuli, the contribution of the extra activated MU is the only response in the signal that remains.

§3.4. : Detecting single motor unit activity

Detection of single motor unit activity, with a small number of electrodes, is only possible at a very low number of active motor units. This can be achieved by applying a low stimulus amplitude. If the same stimulus is applied twice, the resulting responses are not always identical. Each nerve fiber has a sigmoidal probability (Blok, 2001) of being activated with respect to stimulus strength. This is a small alteration to the simplified description of §3.3. At very low stimuli, no fibers will be innervated (innervation probability \( \approx 0 \)), At very high stimulus strength the probability of a nerve being innervated has changed to \( \approx 1 \). If a low stimulus is applied, there will either be no response at all or a response of one or a few MU’s firing. This is the stimulus level were single MU response can be distinguished. To be able to obtain this level, the muscle response is measured while increasing the stimulus amplitude. Once the response shows a clear waveform, the amplitude is slightly decreased. Now for a period of 4 minutes the EMG is recorded, with a stimulus frequency of 10 Hz. After most stimuli no response is detected, but after some a response can be observed. The signals immediately after the stimuli (approximately 2300 responses) are all aligned (Fig 3.7.A).
All signals with no muscle response contribute to the large ‘noise belt’ in the middle. These are removed and now the remaining signals (approximately 350) appear to be separable in three groups with different maximum response and maximum response time (Fig 3.7.B). To make a more precise separation between the responses, all responses are placed in a matrix U. This matrix is subjected to a singular value decomposition. This results in three matrices E, S and F.

$$\text{svd} \begin{bmatrix} U \\ \end{bmatrix} = \begin{bmatrix} E \\ S \\ F \\ \end{bmatrix}$$

Figure 3.8. Singular Value Decomposition.

Matrix F consists of a set of orthonormal basic-vectors, being time signals present in each of the responses.

Matrix S only has diagonal elements, the other elements are zero. The diagonal elements represent the importance of the different basic vectors in reconstructing the original responses, sorted in decreasing order. Matrix E is what distinguishes the responses. The elements of E determine how strong the different basic vectors are present while reconstructing the original response.

The values of the elements in matrix S stabilise after several steps at a noise level. The number of points not belonging to this noise determine the number of basic vectors necessary for reconstructing all responses without their noise (Fig 3.9.A).

For the previously mentioned EMG recording, only two basic vectors appear to be necessary to reconstruct all responses. The weights of these vectors for each response, found in matrix E, are best displayed in an N-dimensional plot. N being the number of necessary basic vectors, in this case a two-dimensional plot (Fig 3.9.B).

In this plot three clusters of points are clearly separated. This indicates that not two but three different types of responses have been recorded. For each cluster the mean value of both weighting numbers is determined. These two mean numbers are used to reconstruct the average response signal of the cluster. Also in this plot, an indication to the number of active motor units can be obtained. This will now be described.

The mean values of the clusters, together with the origin of the figure form a trapezoid (Fig 3.10.A).
The two clusters directly connected to the origin (1 and 2) by this trapezoid have a response that represents a one motor unit response (Fig 3.10B). An activation of a motor unit can thus be seen as a change in location of the cluster by a constant vector. The third cluster (3) is reached by two consecutive vectors, meaning that in this response both the motor units that characterise cluster one and cluster two are stimulated (Compare fig 3.10B to fig 3.7B).

To verify this, the averaged response of the third cluster is compared to the sum of the averaged responses of the other two clusters (Fig 3.11.). In this figure can be seen that the superposed signal is (almost) identical to the one-MU signal, indicating that cluster three is indeed an activation of the motor units of clusters one and two.

The number of activated MU’s in figure 3.9A is thus equal to the number of clusters that can not be reached by adding other vectors.

Note that the maximum amplitude of a 2-MU signal is not larger than the 1-MU signals amplitude (Fig 3.11.). This means that an increase in number of active motor units does not necessarily increase EMG amplitude and that increasing stimulus amplitude will not always result in a stronger response.

By looking more closely to figure 3.10A a fourth cluster can be seen, looking in the direction of the vector connecting the origin to cluster one. This fourth cluster can be caused by three reasons:

- It is a different MU and has a smaller innervation probability than clusters one and two.
- It is in fact belonging to cluster one, but because of a slight electrode- or arm movement the EMG signals amplitude is suddenly increased. An increase in amplitude, but not in shape, would enlarge the vector connecting the origin to cluster one.
- It is a combination of two MU’s both located in cluster one. This means that cluster one does not belong to one single motor unit, but to two motor units having almost identical responses. This would also explain why the distance of the origin to cluster four is almost equal to twice the distance to cluster one.
(Drawing a vector from this fourth cluster parallel to the activation line of cluster two also leads to one measured response.)

If the same measurements are repeated, but now at an increased voltage level, more active motor units (clusters) will appear (Fig 3.12.A). The number of basic vectors necessary to describe the response also increases and for more than three basic vectors visualisation of the clusters becomes difficult (Fig 3.12.B).

![Figure 3.12. A: Three different MU’s detected at slightly increased amplitude B: No cluster structure anymore for too large stimulus amplitudes.](image)

The single motor unit detection and averaging that is described in this paragraph is finally performed on both electrode positions. By aligning the averaged response of the MU detected at both positions, the method described in §3.2. can be used to determine this MU’s conduction velocity. This will be applied in chapter 5.
Chapter 4: 
Single Motor Unit Modelling

The signals measured by SEMG are determined by two important factors:
1. What is the source of the signal?
2. What happens to the signal as it travels from the source to the electrode?
In the model that is used to simulate the SEMG signals, these two factors will be treated separately.

§4.1. : Source modelling

The depolarisation wave inside a fiber is caused by a change in ion-conductance of the membrane (Chapter 1). In figure 4.1. the change in time of these ion permeabilities (d) and the resulting intracellular potential (c) and transmembrane current (b) can be seen.
Here the direction of the transmembrane current (+) is out the fiber.

Figure 4.1. X-axis: position along the fiber.
Y-axis: (B) transmembrane current [A], (C) Intracellular potential [V], (D) \( \text{Na}^+ \) and \( \text{K}^+ \) membrane permeability (passing ions/cm\(^2\)).
Note that the X-axis can also be seen as a time scale for one position on the fiber.

It might appear strange that the first change in transmembrane current is outside (+) since depolarising starts with the influx (-) of \( \text{Na}^+ \) ions. This can be explained by looking at a view of a part of the fiber at a specific moment in time (Fig 4.2.).

Figure 4.2. Currents in and around a fiber at a moment in time.

In this figure can be seen that in front of the great influx area there is a region of the not yet depolarised fiber where the excessive positive-ions inside the fiber are driven outside (either by pumps or osmosis). Note also that the current lines are perpendicular to the equipotential lines in figure 2.1.
The relation between the intracellular action potential and the transmembrane current is described in the core-conductor model. This model uses a chain of elements that together form a model of the entire fiber. All elements have the same structure, this structure can be seen in figure 4.3.

![Figure 4.3. Element of the two dimensional core conductor model, the z-axis is from left to right. (Plonsey, 1969). Shown here is a small part of the fiber with length ΔZ [m].](image)

The lower half of figure 4.3. represents the intracellular, whilst the upper half represents the extracellular. \( r_1 \) is the specific intracellular resistance \([Ω/m]\), \( r_2 \) is the specific extracellular resistance \([Ω/m]\) , \( I_l^o \) is the intracellular longitudinal current \([A]\), \( I_l^i \) is the longitudinal extracellular current \([A]\). \( i_m \) is the current density \([A/m]\) leaving the intracellular through the membrane with thickness \( Z_m [m] \).

For this model the following equations (Ohm’s law) hold:

\[
\frac{\partial V^o}{\partial z} = -I_l^o \ast r_2 \\
\frac{\partial V^i}{\partial z} = -I_l^i \ast r_i \\
\frac{\partial V}{\partial z} \text{ is the difference in potential of 2 points, in this case separated by a distance } \partial z. \\
\text{Here } V^o \text{ is the extracellular potential } [V], \text{ and } V^i \text{ is the intracellular potential } [V].
\]

Since no charges accumulate:

\[
\frac{\partial I^o}{\partial z} = \ast \frac{\partial I^i}{\partial z} \\
\frac{\partial I^o}{\partial z} \text{ and } \frac{\partial I^i}{\partial z}
\]

Substituting 4.2 in 4.3 yields 4.4:

\[
i_m = \frac{1}{r_i} \frac{\partial^2 V^i}{\partial z^2} \quad \text{and} \quad \frac{\partial I^o}{\partial z} = \frac{\partial I^i}{\partial z}
\]

Equation 4.4 shows a linear relation between the current density \( i_m \) and the second derivative of intracellular potential \( V^i \).

The same can be done for the extracellular potential and results in 4.5.

The shape of the intracellular action potential (Fig 4.1.) can be approximated by a triangular shape. The second derivative of such a shape is a summation of three dirac pulses (Fig 4.4B). These three pulses will be used as a representation of the action potential at a point in time instead of the full second derivative of the real action potential (Fig 4.4A).

Note that the model mentioned above is a two dimensional representation of a fiber. In the rest of this chapter potential fields in a three dimensional space will be used. For this we change equation 4.4a. to equation 4.4b.

\[
2 \ast \pi \ast r_f \ast i_m = \frac{1}{r_i} \frac{\partial^2 V^i}{\partial z^2}
\]

In 4.4b. \( r_f \) is the fiber radius.

Note also that \( i_m \) in 4.4b is \([A/m^2]\).
§4.2. : The Volume Conductor

Physical models to describe the extracellular potential have been used since the first one was developed by Lorente de No (Lorente de No, 1947) The current generated by the source, described in the previous paragraph, travels via a passive conductor to the electrode. The theory involved in modelling such a conductor is described below.

In an isotropic, source-free volume the Laplace equation (4.6) holds (Rosenfalck, 1969):

\[ \nabla^2 \Phi = 0 \]  

\( \Phi \) is the potential field around the fiber. This means that no current sources or sinks are present in the conductor. This can be elaborated using cylindrical coordinates (Fig 4.5.):

\[ \nabla^2 \Phi = \frac{\partial^2 \Phi}{\partial r^2} + \frac{1}{r} \frac{\partial \Phi}{\partial r} + \frac{1}{r^2} \frac{\partial^2 \Phi}{\partial \theta^2} + \frac{\partial^2 \Phi}{\partial z^2} = 0 \]

\( \nabla^2 \Phi = \frac{\partial^2 \Phi}{\partial r^2} + \frac{1}{r} \frac{\partial \Phi}{\partial r} + \frac{\partial^2 \Phi}{\partial z^2} = 0 \)

separation of variables 4.9 results in 4.10:
Dividing 4.10 by \( \Phi(r,z) \) yields:

\[
\nabla^2 \Phi = \left[ \frac{1}{R} \frac{\partial^2 R}{\partial r^2} + \frac{1}{r} \frac{\partial R}{\partial r} + \frac{\partial^2 Z}{\partial z^2} \right] = \left[ k^2 \right] + \left[ -k^2 \right] = 0
\]

This means the following two equations should be satisfied

\[
\frac{d^2 Z}{dz^2} + k^2 Z = 0
\]

\[
\frac{d^2 R}{dr^2} + \frac{1}{r} \frac{dR}{dr} - k^2 R = 0
\]

With \( k \) is real.

The first equation (4.12) is solved by a transformation of function \( Z \) to the Laplace domain.

\[(4.12a) \quad s^2 Z + k^2 Z = 0\]

resulting in the following complex numbers of \( s \)

\[(4.12b) \quad s_1 = i^* k \quad s_2 = -i^* k\]

with \( i^2 = -1 \).

Since \( k \) is the frequency, the spatial Fourier transform in this case is:

\[(4.14) \quad Z(k) = \int_{-\infty}^{\infty} Z(z) e^{ikz} dz\]

By defining \( p = k r \) equation 4.13 changes to 4.13a

\[(4.13a) \quad k^2 \frac{d^2 R}{dp^2} + k^2 \frac{dR}{dp} - k^2 R = 0\]

For this equation the general solution can be found in textbooks (Moon, 1971):

\[(4.15) \quad R(p) = R(r^* k) = \alpha^* I_0(r^* k) + \beta^* K_0(r | k |)\]

with \( \alpha \) and \( \beta \) being constants and \( I_0 \) and \( K_0 \) modified Bessel functions (Appendix A)

Now equations 4.14 and 4.15 are entered in 4.9 leading to the final solution 4.16

\[(4.16) \quad \Phi(r,k) = \left[ \alpha^* Z(k) \right] * I_0(r^* k) + \left[ \beta^* Z(k) \right] * K_0(r | k |)\]

This means that the Fourier transform of the potential (anywhere in the volume) can be written as a summation of 2 modified Bessel functions multiplied by either a function \( A(k) \) or \( B(K) \).

\[(4.16b) \quad \Phi(r,k) = \left[ A(k) \right] * I_0(r^* k) + \left[ B(k) \right] * K_0(r | k |)\]

§4.3. : Fields around a fiber

The properties of the source will now be used to determine the potential field \( \Phi \) for increasingly complex volume conductors, starting with a uniform medium and finally leading to a 3-layered bounded an-isotropic conductor.

§4.3.1. : Uniform medium with fiber at \( r=0 \)

In this case the fiber is assumed to lie in the centre of a coordinate system parallel to the z-axis. The fiber has an infinite length and a radius \( r_f \). The current density \( i_m \) is on the fiber’s surface (so at \( r=r_f \)) but is equal for all \( \theta \). This results in a field that is completely circular symmetric. For a positive \( i_m \) this means that the potential decreases with increasing \( r \).
\( I_n \) approaches infinity for \( r \to \infty \). So if (for any \( k \)) the term \( A(k) \) in 4.16b is non-zero the potential for \( r \to \infty \) can not be zero. In reality for \( r \to \infty \) the potential will be zero (for a point source at \( r=0, z=0 \) and \( \theta=0 \)). This implies that all \( \alpha \) are zero and therefore equation 4.16b changes to:

\[
(4.17) \quad \Phi(r, k) = B(k) * K_0(r | k |)
\]

\[
(4.18) \quad \Phi(r, z) = \frac{1}{2\pi} \int_{-\infty}^{\infty} B(k) * K_0(r | k |) * e^{-ikz} \, dk \quad \text{(inverse Fourier)}
\]

In an unbounded area surrounding a fiber the potential can now be calculated. The only unknown variable \( B(k) \) is found by using the boundary condition.

\[
(4.19) \quad i_m(z) = -\sigma_e \frac{\partial \Phi(r, z)}{\partial r} \bigg|_{r=r_f}
\]

with \( i_m [A/m^2] \) the current density (§4.1), \( \sigma_e [(\Omega*m)^{-1}] \) the specific conductivity of the tissue surrounding the fiber, and \( r_f \) the fiber’s radius.

Substitution of 4.18 in 4.19 leads to:

\[
\frac{\partial \Phi(r, z)}{\partial r} \bigg|_{r=r_f} = -\frac{1}{2\pi} \int_{-\infty}^{\infty} k \left| B(k) * K_1(r_f | k |) * e^{-ikz} \right| \, dk = -\frac{i_m(z)}{\sigma_e}
\]

Since

\[
\frac{dK_0(x)}{dx} = -K_1(x) \quad \text{(Rosenfalck)}
\]

Using the inverse Fourier transform:

\[
\sigma_e \left| k \right| B(k) * K_1(r_f | k |) = \int_{-\infty}^{\infty} i_m(z) * e^{ikz} \, dz = G(k)
\]

The right part of 4.21 is renamed \( G(k) \) and is the Fourier transform of the current density described by the tripole model. This \( G(k) \) is, for a given source, fully known.

Therefore now \( B(k) \) is also fully known:

\[
(4.22) \quad B(k) = \frac{1}{\sigma_e \left| k \right|} \frac{G(k)}{K_1(r_f | k |)}
\]

now we have determined

\[
(4.23) \quad \Phi(r, z) = \frac{1}{2\pi\sigma_e} \int_{-\infty}^{\infty} G(k) \left| k \right| K_1(r_f | k |) * K_0(r | k |) * e^{-ikz} \, dk
\]

\[\text{§4.3.2. : Non-centrally located fiber}\]

If the fiber is not located at \( r=0 \) as can be seen in figure 4.6, the term \( K_0(r | k |) \) will have to be expanded.

\[
\text{Figure 4.6. Non centrally located fiber. } R \text{ is the center-fiber distance, } \rho \text{ is the center-observation point distance and } \theta \text{ is the angle between fiber and observation point.}
\]

An expansion for this term is given by Gray and Mathews (Gray, 1952):

(For \( \rho>R \))

\[
(4.24) \quad K_0(r | k |) = \sum_{n=-\infty}^{\infty} e^{-i\rho \theta} I_n(R * k) K_n(\rho | k |)
\]
This changes 4.23 into 4.25

\[
\Phi(r, \theta, z) = \frac{1}{2\pi \sigma} \int_{-\infty}^{\infty} G(k) \frac{k}{|k|} \sum_{n=-\infty}^{\infty} \left[ e^{-i\omega} I_n(R \cdot k) K_n(\rho \cdot k) \right] \cdot e^{-ikz} dk
\]

§4.3.3: Fiber in a 2 layered bounded isotropic medium

Now the model is changed to a more accurate description of reality. Instead of a fiber in an unbounded medium we now look at a fiber in a medium consisting of two tissues (e.g. muscle and fat) bounded by a non-conductive medium (e.g. air).

Figure 4.7. A two layered conductor. The two layers represent muscle (I) and fat (II)

In this situation the equation describing the potential field depends on the coordinates of the observation point.

If the observation point is in layer I (Fig 4.7.), there is an additional term needed to calculate the potential. This term is due to reflections occurring at the boundary of the two media (\(\rho = a\)). The reflections have a maximal amplitude for \(\rho = a\) and diminish for smaller values of \(\rho\). Therefore their shape is a modified Bessel function of the first kind (I).

The potential equation (4.25) now changes to 4.26. (Note that 4.26 shows the Fourier transform of the potential only).

\[
\Phi_I(\rho, \theta, k) = \frac{G(k)}{\sigma_1} \left[ \sum_{n=-\infty}^{\infty} e^{-i\omega} I_n(R \cdot k) K_n(\rho \cdot k) \right] + \sum_{n=-\infty}^{\infty} \left[ e^{-i\omega} A_n(k) I_n(\rho \cdot k) \right]
\]

If the observation point is in layer II (Fig 4.7), there are two terms needed to calculate the potential. One is due to reflections of the medium/air transition (also I shaped) and the other is due to the transition between the two media. These reflections have a maximal amplitude for \(\rho = a\) and diminish for larger values of \(\rho\). Therefore their shape is a modified Bessel function of the second kind (K).

\[
\Phi_{II}(\rho, \theta, k) = \sum_{n=-\infty}^{\infty} \left[ e^{-i\omega} C_n(k) J_n(\rho \cdot k) \right] + \sum_{n=-\infty}^{\infty} \left[ e^{-i\omega} D_n(k) K_n(\rho \cdot k) \right]
\]

In equations 4.26 and 4.27 \(A_n(k), C_n(k)\) and \(D_n(k)\) are unknown variables that can be determined by using the proper boundary conditions.

These boundary conditions need to be solved for every possible combination of \(n\) and \(k\).

The three boundary conditions for this problem are:

\[
\Phi_I(\rho, \theta, k) \bigg|_{\rho = a} = \Phi_{II}(\rho, \theta, k) \bigg|_{\rho = a}
\]

\[
\frac{\partial \Phi_I}{\partial \rho} \bigg|_{\rho = a} = \frac{\partial \Phi_{II}}{\partial \rho} \bigg|_{\rho = a}
\]

\[
\frac{\partial \Phi_{II}}{\partial \rho} \bigg|_{\rho = b} = 0
\]
Substitution of 4.26 and 4.27 in 4.28 leads to:

\[
I_n(a^*k)A_n(k) - I_n(a^*k)C_n(a|k|)D_n(k) = -1 \frac{I_n(R^*k)K_n(a^*|k|)G(k)}{|k|K_1(r_f|k|)}
\]

For substitution of 4.26 and 4.27 in 4.29 the following equations are needed:

\[
\sigma_1 \frac{\delta \Phi}{\delta \rho} = \sigma_1 e^{-ik\theta} \left[ \frac{G(k)}{\sigma_1} * K_1(r_f|k|) * I_n (R^*k) * \left( \frac{\partial K_n(\rho^*|k|)}{\partial \rho} \right) + A_n(k) * \left( \frac{\partial I_n(\rho^*k)}{\partial \rho} \right) \right]
\]

\[
\sigma_2 \frac{\delta \Phi}{\delta \rho} = \sigma_2 e^{-ik\theta} \left[ C_n(k) * \left( \frac{\partial I_n(\rho^*k)}{\partial \rho} \right) + D_n(k) * \left( \frac{\partial K_n(\rho^*|k|)}{\partial \rho} \right) \right]
\]

In equations 4.32 and 4.33 the derivatives of the modified Bessel functions can be calculated by:

\[
\frac{\partial I_n(\rho)}{\partial \rho} = \frac{I_{n-1}(\rho) + I_{n+1}(\rho)}{2}
\]

\[
\frac{\partial K_n(\rho)}{\partial \rho} = -\frac{K_{n-1}(\rho) + K_{n+1}(\rho)}{2}
\]

This results in:

\[
\sigma_1 \frac{\partial I_n(\rho)}{\partial \rho} = \sigma_1 \left( \frac{I_{n-1}(a^*k) + I_{n+1}(a^*k)}{2} \right) * A_n(k) - \sigma_2 \frac{\partial I_n(\rho)}{\partial \rho} \left( \frac{I_{n-1}(a^*k) + I_{n+1}(a^*k)}{2} \right) * C_n(k)
\]

\[
-\sigma_2 |k| * \left( \frac{K_{n-1}(a^*|k|) + K_{n+1}(a^*|k|)}{2} \right) * D_n(k) =
\]

\[
- \frac{G(k)}{|k|K_1(r_f |k|)} * \frac{I_n(R^*k)|k|}{|k|K_1(r_f |k|)} * \left( \frac{K_{n-1}(a^*|k|) + K_{n+1}(a^*|k|)}{2} \right)
\]

Substitution of 4.33 in the third boundary condition (4.30) gives:

\[
0 * A_n(k) - \sigma_2 |k| * \left( \frac{I_{n-1}(b^*k) + I_{n+1}(b^*k)}{2} \right) * C_n(k)
\]

\[
- \sigma_2 |k| * \left( \frac{K_{n-1}(b^*|k|) + K_{n+1}(b^*|k|)}{2} \right) * D_n(k) = 0
\]

Now there is a set of three equations and three unknown variables for each combination of n and k. After solving these equations (summarized in matrix shape in 4.38) the potential can be calculated.
§4.3.4. : Fiber in a 3 layered bounded isotropic medium

In this case the solution is almost identical to that of a 2 layered conductor. But now the equations need to be solved for three regions with different conductivities.

\[ (4.39) \]

\[
\begin{bmatrix}
I_x(a^*k) \\
\sigma_1^*k\left(\frac{I_x(a^*k)+I_m(a^*k)}{2}\right) \\
0 \\
\sigma_2^*k\left(\frac{I_x(a^*k)+I_m(a^*k)}{2}\right) \\
-1 \\
\frac{L_x(R^*k)K_x(a^*k)}{\sigma_1}
\end{bmatrix}
\begin{bmatrix}
L_x(a^*k) \\
-\sigma_2^*k\left(\frac{L_x(a^*k)+L_m(a^*k)}{2}\right) \\
-\sigma_2^*k\left(\frac{L_x(a^*k)+L_m(a^*k)}{2}\right) \\
-1 \\
\frac{\sum_{n=-\infty}^{\infty} e^{-\beta n} E_n(k) I_n(p^*k) + \sum_{n=-\infty}^{\infty} e^{-\beta n} F_n(k) K_n(p \mid k)}{\sigma_2}
\end{bmatrix}
\begin{bmatrix}
A_2(k) \\
C_2(k)
\end{bmatrix}
\]

\[ \begin{bmatrix}
\sigma_2^*k & -\sigma_2^*k \\
-1 & 0
\end{bmatrix}
\begin{bmatrix}
L_x(a^*k) \\
-\sigma_2^*k\left(\frac{L_x(a^*k)+L_m(a^*k)}{2}\right) \\
-\sigma_2^*k\left(\frac{L_x(a^*k)+L_m(a^*k)}{2}\right) \\
-1 \\
\frac{\sum_{n=-\infty}^{\infty} e^{-\beta n} E_n(k) I_n(p^*k) + \sum_{n=-\infty}^{\infty} e^{-\beta n} F_n(k) K_n(p \mid k)}{\sigma_2}
\end{bmatrix}
\begin{bmatrix}
A_2(k) \\
C_2(k)
\end{bmatrix}
\]

\[ (4.40) \]

\[
\Phi_H(p, \theta, k) = \sum_{n=-\infty}^{\infty} e^{-\beta n} E_n(k) I_n(c^*k) + \sum_{n=-\infty}^{\infty} e^{-\beta n} F_n(k) K_n(c \mid k)
\]

Since this project deals with surface EMG, the electrodes will always be positioned on the outer layer \( \rho = c \) and the only equation of interest is that of the outer layer. (Although all equations must be known to calculate variables \( E_n(k) \) and \( F_n(k) \).)

Equation 4.39 changes to

\[ (4.40) \]

\[
\Phi_H(p, \theta, k) = \sum_{n=-\infty}^{\infty} e^{-\beta n} E_n(k) I_n(p^*k) + \sum_{n=-\infty}^{\infty} e^{-\beta n} F_n(k) K_n(p \mid k)
\]

Substitution of the equations in the boundary conditions is similar to the 2 layered model and will not be repeated here.

§4.3.5. : Fiber in a 3 layered bounded medium with an-isotropy

So far the different layers have all been assumed isotropic. For most body tissues this assumption is adequate. Skeletal muscle fibers however are long and thin, resembling bundles of poorly conducting tubes filled with electrolytes. It is therefore not surprising that the resistivity measured transverse to the
fiber direction is higher than that measured in the direction of the fibers. For this reason the inner medium of the model will now be adapted to an-isotropic with the conductivity of skeletal muscle. Values for the specific conductivity of biological material can be found in table 4.1.

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Specific conductivity $\sigma$ [(Ω·m)$^{-1}$]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>0.346</td>
</tr>
<tr>
<td>Fat</td>
<td>0.040</td>
</tr>
<tr>
<td>Skeletal muscle (longitudinal)</td>
<td>0.416</td>
</tr>
<tr>
<td>Skeletal muscle (transverse)</td>
<td>0.148</td>
</tr>
</tbody>
</table>

Table 4.1. Specific conductivities (Geddes, 1967)

The an-isotropy can be introduced in the model by multiplying factors $R$ and $\rho$ by $\sqrt{\sigma_z/\sigma_r}$. (Rosenfalck, 1969).

So finally we have:

$$
\Phi_j(\rho, \theta, k) = \frac{G(k)}{\sigma_{l_j}} \sum_{i=\pm} e^{-i\theta} \frac{I_k(r_j)}{R^*k} K_n\left(\frac{\sqrt{\sigma_{l_j}}}{\sqrt{\sigma_{l_i}}} \rho^* |k|\right)
$$

$$
\Phi_n(\rho, \theta, k) = \sum_{i=\pm} e^{-i\theta} C_n(k) I_n\left(\frac{\sqrt{\sigma_{l_j}}}{\sqrt{\sigma_{l_i}}} \rho^* |k|\right)
$$

$$
\Phi_m(\rho, \theta, k) = \sum_{i=\pm} e^{-i\theta} E_n(k) I_n\left(\frac{\sqrt{\sigma_{l_j}}}{\sqrt{\sigma_{l_i}}} \rho^* |k|\right)
$$

Resulting in a matrix like 4.38:

$$
\begin{bmatrix}
\vdots & \vdots & \vdots & 0 & 0 \\
\vdots & \vdots & \vdots & 0 & 0 \\
0 & \cdots & \cdots & \cdots & \cdots \\
0 & \cdots & \cdots & \cdots & \cdots \\
0 & 0 & 0 & \cdots & \cdots \\
\end{bmatrix}
\begin{bmatrix}
A_n(k) \\
C_n(k) \\
D_n(k) \\
E_n(k) \\
F_n(k) \\
\end{bmatrix}
= \begin{bmatrix}
\cdots \\
\cdots \\
0 \\
0 \\
0 \\
\end{bmatrix}
\begin{bmatrix}
G(k) \\
\cdots \\
\cdots \\
\cdots \\
\cdots \\
\end{bmatrix}
$$

The full matrices can be seen in appendix C.

§4.3.6.: Time variance

Assuming that the propagation velocity (v) is constant over the fiber’s length, the z position of the current tripole can be expressed as a function of time (t).

This means that the current density $i_m$ can be described by 2 tripole models, propagating in opposite directions, as a function of time.

If the motor end plate is at z=0, then the two leading monopoles are at $z(t)=v^*t$ and $z(t)=-v^*t$. The other two monopoles are at constant distances D1 and D2 lagging these leading monopoles (Fig 4.9.).
Typical values for this can be found in table 4.2.

<table>
<thead>
<tr>
<th>parameter</th>
<th>value</th>
<th>From:</th>
</tr>
</thead>
<tbody>
<tr>
<td>D1</td>
<td>1.6*10^{-3} [m]</td>
<td>Roeleveld 1997</td>
</tr>
<tr>
<td>D2</td>
<td>4.8*10^{-1} [m]</td>
<td>Roeleveld 1997</td>
</tr>
<tr>
<td>v</td>
<td>4 [m/s]</td>
<td>Fuglevand 1992</td>
</tr>
</tbody>
</table>

Table 4.2. tripole source parameter values.

In table 4.2. I [A] is given instead of $i_m$ [A/m²]. The relation between these is given in equation 4.45.

\[
(4.45) \quad i_m = \frac{I}{2 \pi r_f \Delta Z}
\]

$\Delta Z$ [m] is the length of a small part of the fiber. This parameter is set to $5 \times 10^{-4}$ m.

For those values of $t$ were the first monopole has not yet travelled over a distance $D_2$, the source is considered as not fully developed. In this case $i_m$ is taken zero for all $z$.

For those values of $t$ were the first monopole has reached the muscle/tendon transition ($z =$ muscle length), $i_m$ is also taken zero for all $z$.

However, it is not known whether the parameters $D_1$, $D_2$ are exact constants or whether they depend on the conduction velocity. Combining real measurements to simulated EMG responses will be used in chapter five to investigate this.

§4.3.7. : Discussion

In this last section the an-isotropy is included for all three layers. This is however not necessary since the outer two layers are skin and fat. These two tissues can be treated as isotropic. As a result of this all terms $\sqrt{\frac{\sigma_{2z}}{\sigma_{2z}^2}}$ and $\sqrt{\frac{\sigma_{3s}}{\sigma_{3s}^2}}$ are 1.

Furthermore it can be argued that the resulting three layered model is not an accurate representation of a real limb used for measurements. Bone tissue e.g. is neglected.

If this bone tissue was included, it would have to be in the centre of the arm and therefore in the centre of the model. However since the muscle layer is reasonably large (cm) any reflections coming from the bone tissue will be almost entirely extinguished over the distance they travel towards the bone and back.
Chapter 5: 
Parameter extraction

This chapter will start with a summary of the consecutive steps taken in the processing and analysis of the recorded surface EMG signal. An experiment has been set up to see whether the signals acquired with the single MU detection protocol described in §3.4. are reproducible. For this, reproducibility is checked intra-personal and inter-personal. Once reproducibility is proven, the measured response is compared to several artificial responses created by the model described in chapter 4. All these artificial responses have slightly different parameters. In this evaluation variations in MU depth, MU size, and tripole structure have been evaluated. A best fit, using the sum of squares difference, of the measured response with the artificial responses is determined. The parameters, belonging to this best fit, best describe actual parameters of the activated MU.

The continuously stored EMG-data is loaded and scanned for stimulus pulses. If a pulse is detected, the EMG signal recorded in the next 25 ms is separately stored. Assuming a velocity of 2 m/s and a motor-end-plate/tendon distance of 5 cm, this is the time needed to travel over the entire length of the fiber. After storing the first pulse, searching for the next stimulus pulse goes on. Once all the stimuli have been detected this way, the stored responses are aligned. After removal of the responses that do not show muscle activity, the remaining responses are clustered using the singular value decomposition. The average signal of the first selected cluster (MU) is later used for determining MU parameters. All operations described above are performed for both electrode positions, resulting in 2 averaged MU signals, one signal lagging the other. Using this time lag and the known distance between the two electrode positions, the MU conduction velocity for this MU is determined (Chapter 3).

The MU conduction velocity is used as the conduction velocity of the fiber in the model of chapter four. Other parameters needed in the model such as muscle-, fat- and skin-radius vary between people and need to be measured before simulation starts. In this study we did not obtain these values for every test subject, but averaged values found in literature (Roeleveld, 1997) have been used. The model determines what the signal at the skin surface would have looked like for MU’s located at various depths underneath the skin, for MU’s of different size, and for MU’s with different tripole structures. Comparing these simulated responses to the averaged response of the recorded MU signal shows which set of MU parameters is most likely for the averaged response.

§5.1. : Intra-personal variation

Measurements have been performed on one subject. Seated in a chair, with the lower arm resting on a table in front of the subject (male, 22), the elbow is at an angle of approximately 120 degrees. Both wrist and ring finger are in a fixed position to prevent movement. After positioning of the electrodes the inter-electrode distance is measured. Because all velocities are calculated by using the same inter-electrode distance, inaccuracies in this distance do not influence the accuracy of changes in the conduction velocity. The protocol involved threshold stimuli being applied for 20 seconds at a frequency of 10 Hz, after this there is a period of 20 seconds rest, then again a period of 20 seconds with threshold stimuli. Then all wires connecting the electrodes to the recorder were removed for a short period of rest (one min). After this period the wires were connected again and the same protocol was repeated. The most frequently activated MU in each recording is averaged using the singular value decomposition. The MU conduction velocity is determined. To study the effect of the placement of the electrodes, this protocol is repeated five times, each time using a new set of electrodes. Results are shown in table 5.1:
Table 5.1. Single MU conduction velocities [m/s] measured in one subject. Each new electrode-set is a new row. Percentages indicate the change with respect to the previous measurement. The accuracy of the velocities reported here, depends on the accuracy in selecting the center of a cluster (§ 3.4.).

For the measurements performed with only 20 seconds rest in between, the responses should be almost identical. The percentages in table 5.1. show that the average change in velocity of the responses after this period of 20 seconds rest is 3.3 %.

The change in recorded conduction velocity during one minute rest, determined between the average of the two measurements before and the two measurements after the rest period, is 2.7 %.

Comparing the averages of the five experiments, shows that replacing electrodes causes much greater changes in conduction velocity. This is probably because repositioning of the stimulus electrode places the stimulus electrode on a slightly different position and thus above different nerves.

These results indicate that repeated stimulation of the same MU occurs if measurements are performed without replacing the electrodes. However caution has to be taken to prevent the electrodes from moving during the stimulations.

To verify that to same MU is stimulated in two consecutive recordings, the responses (in time) for the first electrode position are also compared. (Fig 5.1.).

![Diagram](image)

Figure 5.1. Responses at the first electrode position for the first and second recording in the first row of table 5.1.

The small difference between these responses, together with the small change in conduction velocity, indicates that the same MU is causing the responses in both recordings.
§5.2. : Inter-personal variation

The same protocol is repeated in three other subjects.

<table>
<thead>
<tr>
<th></th>
<th>First 20 seconds</th>
<th>20 seconds rest</th>
<th>Second 20 seconds</th>
<th>1 min rest</th>
<th>Third 20 seconds</th>
<th>20 seconds rest</th>
<th>Fourth 20 seconds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject A</td>
<td>5.10</td>
<td>5.10 (0.0 %)</td>
<td>3.56</td>
<td></td>
<td>3.63 (2.0 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(male 26)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subject B</td>
<td>2.84</td>
<td>2.75 (-3.2 %)</td>
<td>2.92</td>
<td></td>
<td>2.98 (2.1 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(male 20)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subject C</td>
<td>XXX</td>
<td>XXX</td>
<td>2.73</td>
<td></td>
<td>2.63 (3.7 %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(male 25)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 5.2. Single MU conduction velocities [m/s]. Each row is a different subject.

Again, the change in conduction velocity of the responses separated by 20 seconds is small (2.2 %). Note that for subject C no results are shown for the first two recordings. The reason for this is that due to large background muscle activity, no clear MU response could be determined by the singular value decomposition.

For subject A, the change in conduction velocity over the one minute rest period is relatively large (29.5 %). This is probably because stimulus electrodes have slightly moved and recordings show different MU’s. To verify this, the responses belonging to these measurements are shown in figure 5.2.

![Figure 5.2](image)

Figure 5.2. Responses at the first electrode position for the first and fourth recording in the first row of table 5.2.

In figure 5.2, the responses are not similar (compare Fig 5.2. to Fig 5.1.) This indicates that either not the same MU is active or that the electrodes have moved and are no longer positioned directly above the muscle.

§5.3. : Parameter extraction

Now that the MU conduction velocity has been determined it is possible to estimate other MU parameters as well. For this the model of chapter 4 will be used. The extracellular potential field described by the model is calculated multiple times, every time with different values for the MU depth, MU size, and tripole structure parameters. This way a look-up-table is constructed with for each set of parameters the expected response.
The parameter for MU depth gives an average value for distance between skin and all the fibers belonging to the MU. The range of this parameter is chosen by using the physical location of the muscle digitorum superficialis. The muscle has a thickness of approximately one centimeter and is located directly under skin and fat tissue. Therefore the parameter (R in figure 4.8.) is ranged from a-0.01 to a, where a represents the muscle radius [m] (Fig 4.8.). In this range steps of 1 mm are evaluated, resulting in 11 possible values for R.

The MU size is the number of muscle fibers belonging to one MU. The model in chapter 4 calculates the extracellular potential field of a single fiber. But if we assume that all fibers are located in the center of the MU and all have the same properties, the superposition principle holds and the model can also be used to model a MU.

In §4.3.6. transmembrane current for a single fiber is given. (I = 388*10^{-9} A) (Fuglevand, 1992). Now we assume that the MU consists of X fibers and thus net current of this MU is X*I.

The range for X, being the number of fibers in the MU, can range from as little as three for very small MU’s to several hundreds for the larger MU’s. Here a range of 10 to 310 will be evaluated with steps of 50. This results in 7 possible values for X.

The tripole structure is the parameter that determines the spacing of the three current monopoles in §4.3.6. The values for D1 and D2 given in this paragraph might vary for different MU’s and therefore are also evaluated. The parameter representing tripole structure is D. We assume that D1 = 1/3*D2 and D2 = D for every value of D.

Since the range for this parameter varies in literature (Roeleveld, 1997)(Rosenfalck, 1969)(Saitou, 1999), the evaluated parameters are D=D2, D=0.9*D2, or D=1.1*D2.

Other parameters needed in the model are:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Represents</th>
<th>Source</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>a</td>
<td>Radius of the muscle tissue (Fig 4.8.)</td>
<td>Averaged (Roeleveld, 1997)</td>
<td>3,0*10^{-6} m</td>
</tr>
<tr>
<td>b</td>
<td>Radius of the fat tissue (Fig 4.8.)</td>
<td>Averaged (Roeleveld, 1997)</td>
<td>3,2*10^{-2} m</td>
</tr>
<tr>
<td>c</td>
<td>Radius of the skin tissue (Fig 4.8.)</td>
<td>Averaged (Roeleveld, 1997)</td>
<td>3,3*10^{-2} m</td>
</tr>
<tr>
<td>r_f</td>
<td>Radius of a single fiber (Eq. 4.19)</td>
<td>Averaged (Guyton, 1996).</td>
<td>25*10^{-6} m</td>
</tr>
<tr>
<td>ΔZ</td>
<td>Spatial resolution for longitudinal direction</td>
<td>Optional.</td>
<td>5*10^{-4} m</td>
</tr>
</tbody>
</table>

*Table 5.3. Constants used in the model.*

This results in 231 (11*7*3) possible combinations for the three parameters and in 231 calculated response curves (look-up-table) (Fig 5.3.).

*Figure 5.3. Responses for every possible combination of parameters shown in one figure.*
In order to compare a calculated response with the measured response, the time scales of the measured and the simulated responses need to be synchronised. (Fig 5.4.) For this purpose the cross correlation of the two signals is used. For all time samples in the response the square of the difference between measured and simulated response is calculated. The sum of these squared values is used as a measure to compare the two responses.

![Figure 5.4. Alignment of all the simulated responses (green) to the measured response (red).](image)

This sum of squares method is performed on all 231 responses. Every set of parameters now has a value for how well the shape of its response curve resembles the measured response (Fig 5.5). The parameters belonging to the minimum of these values are the best estimate for the actual real parameters of the MU (Fig 5.6.) since this response most resembles the measured response.

![Figure 5.5. Sum of squares for every parameter combination.](image)
The two curves shown in figure 5.6. show resemblance, but there are also a lot of differences between the two. These might be eliminated by choosing a more precise range of values for the three parameters.

The most profound difference between the two curves is the difference in width of the main peaks. One of the most probable explanations for this is that the averaged values for a, b, and c are not fitting the subject. A thicker layer of fat would for instance lead to a widening of the peaks. Another possibility might be that some assumptions entered in the model are simplified too much. For example, the tripole source could have been described in more detail.

For the ranges describing MU size and MU depth, the best parameter is located well within the maximum and minimum of the range. This indicates that the best value is found within the parameters that are physically possible. The best parameter for tripole shape is found at the largest value of D incorporated in the evaluation. This could indicate that the value for D is actually even larger than the estimated value. However, the effect of having a larger value for D is comparable to the effect of having a thicker fat layer (widening of the peaks). Thus if the constant b, for thickness of the fat layer, was chosen too small, a larger value for D will be favoured by the least squares method.

Parameter extraction is performed for only two measurements (The first two recordings of table 5.1.). The purpose of these two extractions is to investigate whether responses that are caused by the same MU also show similar MU parameters. This checks the convergence of the algorithm simulating the responses. The best set of parameters for these two recording is given in table 5.4.

<table>
<thead>
<tr>
<th>R (MU-to-center radius) [m]</th>
<th>X (number of fibers) [1]</th>
<th>D (tripole structure) [%]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recording 1: 0.024</td>
<td>260</td>
<td>110</td>
</tr>
<tr>
<td>Recording 2: 0.023</td>
<td>260</td>
<td>110</td>
</tr>
</tbody>
</table>

Table 5.4. Best set of parameters for two responses caused by the same MU

Table 5.4. indicates that estimation of parameters for responses caused by the same MU results in approximately the same parameters (even though there might be a mistake in model constants).

In conclusion, this means that the single MU responses, recorded on the skin can be used to determine the characteristics of this MU. In the future a muscle biopsy can help to verify the outcome of the parameter estimation.
Discussion & Recommendations

The use of a very basic EMG recording set-up has the advantages of low costs and ease of reproducibility. However, using a basic set-up also involves certain assumptions. First of all in this thesis the assumption is made that conduction velocity is constant over the entire muscle fibers length. Also the positioning of the electrodes is assumed to be directly above the muscle. Misplacement of the electrodes (or using an asymmetrical set-up) results in false estimates of the conduction velocity since the EMG response recorded at the two electrodes is not only different in time but also in shape (Fig D.1.).

![Figure D.1. Misplacement of electrodes leads to different shapes of the recorded EMG responses.](image)

Positioning of the stimulation electrodes can be difficult. To stimulate the desired muscle (flexor digitorum superficialis) one of the two stimulus electrodes needs to be placed directly above the motor-end-plate on this muscle. If this electrode is placed only above the muscle but not above the motor-end-plate, no stimulation will occur. Therefore, repositioning the stimulus electrode might be needed several times before the motor-end plate is located. The other stimulus electrode is placed at the other side of the arm, exact positioning of this electrode is not necessary. Placement of the recording electrodes starts once the stimulus electrodes are in place. While electrically stimulating the muscle, the exact location of the muscle is determined by palpation. The three electrodes are now placed above this muscle as close to the stimulus electrode as possible. Care needs to be taken that none of the electrodes are in contact with each other because this will short circuit the set-up.

The method described in chapter three to extract single motor unit activity can be further elaborated to allow for detection of more motor units. Using singular value decomposition the basic vectors determined for the first few active motor units (as described in this study) can be extracted from responses showing a larger number of active motor units. Then performing a second singular value decomposition on the remaining responses will show the next few active motor units.

In chapter five has been indicated that an accurate measure for the composition of the arm will probably lead to a better model-to-measurement fit. Obtaining these model constants can for instance be done by analysing an MRI slice of the lower arm. For validation of the estimated parameters, a muscle biopsy should prove whether or not the estimated parameters are correct. Furthermore the model should be checked for sensitivity to the parameters. Parameter extraction as described in this thesis can prove to be a useful method in studies of starvation, exertion or certain muscle diseases.

The measured motor unit conduction velocity can be related to the rest membrane potential and the ion concentrations near the membrane. An additional model describing this relation will help in the investigation of muscle disease caused by abnormalities in this rest membrane potential. In the future a change in motor unit conduction velocity can be a measure for a patients state. Changes in this conduction velocity, and thus in rest membrane potential, can then be countered by a nutritional intervention.
Conclusions

Recording single MU activity in a muscle with a set-up consisting of only a small number of electrodes with a diameter that is relatively large compared to state of the art electrodes, asks for special attention in the selection of the muscle. All the conditions a muscle should comply with are met in the muscle flexor digitorum superficialis.

By applying low voltage stimuli to the motor-end-plate of this muscle (using a common nerve stimulator available on the market) it was possible to elicit very small muscle contractions. These contractions are so small they are not noticed by the subject, but can be recorded with the developed set-up, with electrodes placed on the skin above the muscle.

Using singular value decomposition, a new way to accurately determine the number of motor units contributing to this response was shown in chapter three. Results for up to three motor units have been presented.

With the two signals recorded simultaneously by the set-up, the single motor unit conduction velocity was determined non-invasively. Reproducibility of this motor unit conduction velocity within one subject was studied, proving that it is possible to stimulate the same MU repeatedly. This reproducibility is only possible if the stimulus electrodes are not moved during the test. This leads to the conclusion that comparison of single motor unit conduction velocity is not possible between subjects. However, the set-up can be used in studies in relative conduction velocity changes in individuals, such as exertion or starvation.

A short review of literature on the modelling of motor unit potential fields is given. A model has been derived (guided by one of the most recent models), simulating a three layered (muscle, fat, and skin) volume surrounding the MU. This model describes a moving current source in anisotropic and isotropic tissues. Parameter extraction with the use of this model leads to a characterisation of the active motor unit. Parameters for motor unit size, motor unit depth, and tripole spacing are found to be well within a physical possible range, and are shown to be reproducible if the same motor unit is stimulated repeatedly.
Acknowledgment

I am grateful to all the people who have contributed in different ways to my work. My special thanks go to my supervisor Natal van Riel, to Ad Damen, and to Paul van den Bosch for their comments, guidance and suggestions. Also I would like to thank Ton Wagenmakers, Hans Savelberg, and Kenneth Meijer from Maastricht University for their comments and guidance concerning the medical and physiological issues. It has been a great pleasure working with the students and staff at the group of biosignals & regulation as well as the group of control systems of the electrical engineering department.

Finally I would like to thank my parents, my brothers, my girlfriend, my relatives, my friends, and everybody who I might have been forgotten to mention, for their support over the past five years.
Frequently used symbols and abbreviations:

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Definition</th>
<th>Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \rho )</td>
<td>Electrode to center distance</td>
<td>[m]</td>
</tr>
<tr>
<td>( \sigma )</td>
<td>Specific conductivity</td>
<td>[(( \Omega \cdot \text{m} ))(^{-1} )]</td>
</tr>
<tr>
<td>( \theta )</td>
<td>Angle between R and ( \rho )</td>
<td>[rad]</td>
</tr>
<tr>
<td>( \Phi )</td>
<td>Potential</td>
<td>[V]</td>
</tr>
<tr>
<td>a</td>
<td>Muscle radius</td>
<td>[m]</td>
</tr>
<tr>
<td>b</td>
<td>Fat radius</td>
<td>[m]</td>
</tr>
<tr>
<td>c</td>
<td>Skin radius</td>
<td>[m]</td>
</tr>
<tr>
<td>CV</td>
<td>Conduction velocity (text)</td>
<td>[m/s]</td>
</tr>
<tr>
<td>D1</td>
<td>Tripole parameter</td>
<td>[m]</td>
</tr>
<tr>
<td>D2</td>
<td>Tripole parameter</td>
<td>[m]</td>
</tr>
<tr>
<td>EMG</td>
<td>Electromyography</td>
<td></td>
</tr>
<tr>
<td>( l_0 )</td>
<td>Bessel function (1\textsuperscript{st} kind)</td>
<td></td>
</tr>
<tr>
<td>( i_m )</td>
<td>Current density</td>
<td>[A/m(^2)]</td>
</tr>
<tr>
<td>k</td>
<td>Spatial frequency</td>
<td>[m(^{-1})]</td>
</tr>
<tr>
<td>K</td>
<td>Bessel function (2\textsuperscript{nd} kind)</td>
<td></td>
</tr>
<tr>
<td>MF</td>
<td>Muscle fiber</td>
<td></td>
</tr>
<tr>
<td>MU</td>
<td>Motor unit</td>
<td></td>
</tr>
<tr>
<td>r</td>
<td>Radial coordinate</td>
<td>[m]</td>
</tr>
<tr>
<td>R</td>
<td>Fiber to center distance</td>
<td>[m]</td>
</tr>
<tr>
<td>( r_f )</td>
<td>Fiber radius</td>
<td>[m]</td>
</tr>
<tr>
<td>SEMG</td>
<td>Surface electromyography</td>
<td></td>
</tr>
<tr>
<td>v</td>
<td>Conduction velocity</td>
<td>[m/s]</td>
</tr>
<tr>
<td>X</td>
<td>Number of fibers/MU</td>
<td>[1]</td>
</tr>
<tr>
<td>z</td>
<td>Longitudinal coordinate</td>
<td>[m]</td>
</tr>
</tbody>
</table>
References


Hermann, L. 1879, ‘Handbuch der physiologie’ Leipzig Germany


Appendix A: Bessel Functions

$I_0(x)$ is the Modified Bessel function of the first kind. The zero in the subscript indicates that this is the zero'th order of the first kind.

$K_0(x)$ is the Modified Bessel function of the second kind. The zero in the subscript indicates that this is the zero'th order of the second kind.

These functions are defined as:

$$I_\nu(x) = \frac{x^\nu}{2^\nu \Gamma(\nu + 1)} \sum_{k=0}^{\infty} \frac{x^{2k}}{k! \Gamma(\nu + k + 1)}$$

$$K_\nu(x) = \frac{\pi}{2} \frac{I_{-\nu}(x) - I_\nu(x)}{\sin(\nu \pi)}$$

with

$$\Gamma(y) = \int_0^\infty e^{-t} t^{y-1} dt$$

These functions look like figure A.1.

Figure A.1. Typical Bessel function shapes
Appendix B :
Flexor Digitorum Superficialis

Note: Stimulation is applied to the red coloured part of the flexor digitorum superficialis.
(Putz, 1994)
Appendix C:
Full Model Matrix

\[
\begin{bmatrix}
k1 & k2 & k3 & 0 & 0 \\
k4 & k5 & k6 & 0 & 0 \\
0 & k7 & k8 & k9 & k10 \\
0 & k11 & k12 & k13 & k14 \\
0 & 0 & 0 & k15 & k16 \\
\end{bmatrix}
\begin{bmatrix}
A_n(k) \\
C_n(k) \\
D_n(k) \\
E_n(k) \\
F_n(k) \\
\end{bmatrix} =
\begin{bmatrix}
K1 \\
K2 \\
0 \\
0 \\
0 \\
\end{bmatrix}
\]

with:

\[k1 = I_n\left(\frac{\sigma_{1r}}{\sigma_{1r}} * a * k\right)\]

\[k2 = -I_n\left(\frac{\sigma_{2r}}{\sigma_{2r}} * a * k\right)\]

\[k3 = -K_n\left(\frac{\sigma_{2r}}{\sigma_{2r}} * a | k \right)\]

\[k4 = \sqrt{\sigma_{1r} * \sigma_{1r} * k} \left\{ I_{n-1}\left(\frac{\sigma_{1r}}{\sigma_{1r}} * a * k\right) + I_{n+1}\left(\frac{\sigma_{1r}}{\sigma_{1r}} * a * k\right) \right\} \]

\[k5 = - \sqrt{\sigma_{2r} * \sigma_{2r} * k} \left\{ I_{n-1}\left(\frac{\sigma_{2r}}{\sigma_{2r}} * a * k\right) + I_{n+1}\left(\frac{\sigma_{2r}}{\sigma_{2r}} * a * k\right) \right\} \]

\[k6 = - \sqrt{\sigma_{2r} * \sigma_{2r} * | k \} * \left\{ K_{n-1}\left(\frac{\sigma_{2r}}{\sigma_{2r}} * a * | k \right) + K_{n+1}\left(\frac{\sigma_{2r}}{\sigma_{2r}} * a * | k \right) \right\} \]

\[k7 = I_n\left(\frac{\sigma_{2r}}{\sigma_{2r}} * b * k\right)\]

\[k8 = K_n\left(\frac{\sigma_{2r}}{\sigma_{2r}} * b | k \right)\]

\[k9 = -I_n\left(\frac{\sigma_{3r}}{\sigma_{3r}} * b * k\right)\]
\[ k_{10} = -K_n\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right) \]

\[ k_{11} = \sqrt{\sigma_{3_y} |b| k} \left( I_n^{-1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right) + I_{n+1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right)\right) \]

\[ k_{12} = \sqrt{\sigma_{3_y} |b| k} \left( I_n^{-1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right) + I_{n+1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right)\right) \]

\[ k_{13} = -\sqrt{\sigma_{3_y} |b| k} \left( I_n^{-1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right) + I_{n+1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right)\right) \]

\[ k_{14} = -\sqrt{\sigma_{3_y} |b| k} \left( I_n^{-1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right) + I_{n+1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right)\right) \]

\[ k_{15} = \sqrt{\sigma_{3_y} |b| k} \left( I_n^{-1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right) + I_{n+1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right)\right) \]

\[ k_{16} = \sqrt{\sigma_{3_y} |b| k} \left( I_n^{-1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right) + I_{n+1}\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |b| k\right)\right) \]

\[ K_1 = \frac{I_n\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} R|k\right)K_n\left(\frac{\sigma_{3_y}}{\sqrt{\sigma_{3_y}}} |a| |k\right)}{\sigma_{3_y}} \]
\[ K_2 = \frac{-1}{|k|^*} K_1(r_j | k |) * I_n(\sqrt{\frac{\sigma_{l_i}}{\sigma_{l_j}}} * R * k)^* \frac{\sigma_{l_i}}{\sigma_{l_j}} | k |)^* - \left( \frac{K_{n-1}(\sqrt{\frac{\sigma_{l_i}}{\sigma_{l_j}}} * d^* | k |) + K_{n-1}(\sqrt{\frac{\sigma_{l_i}}{\sigma_{l_j}}} * d^* | k |)}{2} \right) \]