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Motor Unit Action Potentials and the Influence of Skin Conductivity

Modelling and Experiments

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Preface

This master's thesis forms, together with the simulation program Anvolcon, the physical remainder of my research project. This project took place at the Department of Clinical Neurophysiology of the Institute of Neurology during the period from January 1997 until January 1998. By means of this thesis and other, previous, efforts, I will (hopefully) receive the Master of Science Degree in Technical Physics at the University of Technology in Eindhoven (TUE). This would not have been possible without the advise, encouragements and help I got. Therefore, I want to use this opportunity to thank all the people who supported me during my research. Several people should be mentioned in particular: Dick Stegeman, the pioneer of research in EMG at the Department of Clinical Neurophysiology and in many other areas (his name appears everywhere), Joleen Blok, my adviser in practical and theoretical problems (in return, I watered her plants), Pieter Wijn (my mentor in Eindhoven) and for electrotechnical and computertechnical matters, Hans van Dijk, Carlo Berenstein and Leo Haegens. I would also like to thank the persons (subject I,II,III,IV,V,VI,VII) who enabled me to do my measurements. Special thanks go out to my parents who always kept their confidence in me and supported me when necessary. At the end, I would like to thank my sister; playing 'little school' with her in my childhood did pay off at the end.

Theo Arends.
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Clinical electromyography (EMG) plays an important role in the diagnosis and follow-up studies of neuromuscular disorders. Of fundamental importance to this technique is the information that the EMG may offer on the function and structure of motor units, the fundamental units of a muscle. Until now, in clinical diagnosis most often needle-EMG is used. This invasive procedure uses intramuscular needles of a variety of types to closely approach the motor units with the recording tip of the needle. Their main disadvantage is the stress and pain they cause the patient; besides, they are a potential source of infections. Moreover, many diagnostic issues relate to children, for whom this type of investigation is especially cumbersome. Consequently, each opportunity to obtain methods of EMG that are less invasive should be given serious consideration.

Two years ago, a project, supported by SLW/STW, has started with the aim to obtain an easily applicable method to quantify motor units, using a topographical analysis of the surface-EMG. The results of the method to be developed will have to comply both in contents and in form to the present clinical EMG results to facilitate its introduction. It is conceivable however, that this new surface-EMG topography will yield information that goes beyond the present possibilities. Finally, a surface electromyographic technique has greater reproducibility than needle-EMG, which forms a great advantage for follow-up studies.

This master’s thesis forms a part of this project.
Summary

In clinical ElectroMyoGraphy (EMG) neuromuscular diseases are generally diagnosed by needle EMG. Needle EMG is an invasive, local signal acquisition and analysis method. Its aim is to visualise action potentials from motor units, the functional units of muscles. In recent research, the focus has been shifted to surface EMG, in which electrodes are placed on the human skin. In this thesis, volume conduction theory is dealt with for an anisotropic inhomogeneous volume conductor. This theory is applied in a model, Anvolcon. With this model it is possible to simulate motor units in a cylindrical layer model with the specific properties of muscle, fat, skin and bone tissue. An important parameter in the volume conduction model is the conductivity of the human skin layer. Measurements showed that the conductivity of the human skin is high ($0.95 \pm 0.35 \ \Omega^{-1} m^{-1}$) relative to that of the underlying layers. Furthermore, this conductivity increases with temperature ($0.015 \ \Omega^{-1} m^{-1}$ per 1 °C). Simulations showed that this high conductivity value is essential to understand the low-amplitude and distributed potentials at the surface of the skin that were found in earlier surface EMG measurements.
Chapter 1

Introduction to electromyography

In this chapter general aspects of electromyography\(^1\) will be reviewed. The history of electromyography (EMG) is treated in § 1.1, while the building stones of EMG, the muscle fibres, are the subject in § 1.2. An introduction on EMG signals is presented in § 1.3. Because research concerning the human skin is described in chapters 4 and 5 some anatomical aspects of the skin are described in § 1.4. In § 1.5, finally, the survey and the goals of this masters thesis can be found.

1.1. History of electromyography

Muscles have been the object of scientific interest for many centuries. Already in the 17th century the power of some fishes to generate 'electric' shocks was associated with muscle activity in the inner bodies of the fishes. However, until the start of the present century only the anatomical properties of muscles were studied. It took thus three centuries before people obtained information about muscle activity itself.

In the mean time, Galvani reported as early as 1791 that muscular contraction produces electrical activity. In the second half of the 19th century, in 1851, Dubois-Reymond was able to record an action current from a contracting arm muscle of a human subject. He used liquid as electrodes and became by this the first person to demonstrate a human myogram. Duchenne (in 1867) embroidered on this experiment and used electrical stimulation to determine the dynamics of intact muscle tissue. It took however more than 70 years (1929) before Adrian and Bronk measured successfully the electrical product of muscle activity by placing a needle electrode in a muscle. It was the first example of needle electromyography in history, it gave a view of a so-called intracellular action potential (§ 1.3). Until then, attempts to obtain satisfactory recordings with surface electrodes had consistently failed.

At the end of the second world war, after improvements of techniques to measure very low voltages, mostly the phenomenological aspects of the electromyogram were studied. It was first necessary to unravel the functions of individual muscles and muscle groups. Besides that a characterisation and description of the components of the muscle potential had to be made before fruitful clinical applications could be introduced. Important progress on the development of mathematical conduction models was made by researchers like Lorente de Nó (model of a nerve fibre, [Nó64]), while Clark and Plonsey made a model of a cylinder shaped volume conductor in 1968 [Cla68]. In 1969 Rosenfalck described the intracellular and extracellular action potential mathematically [Ros69], while Wallinga-de Jonge (amongst others) in 1985 [Wal85] and Ludin in 1968 [Lud68] measured the action potential in rat respectively human muscles, all important additions to the fundamental knowledge of EMG signals.

In clinical practice nowadays needle and surface electromyograms contribute to the diagnosis of diseases of the neuro-muscular system. The investigation basically consists of the extracellular recording of action potentials generated by muscles. Several needle EMG recording techniques, developed in the past mostly by Buchthal and Stålberg [Stå80,96], and 20 years of application in a

---

\(^1\) electromyography, writing (graphy) of electrical (electro) signals from muscles (myo).
clinical situation, accomplished a large experience and literature on the relations between disorders and deviations of the motor unit action potential (§ 1.2). Needle EMG has however a few disadvantages; it is because of the needle an invasive technique and the provided information is very local (take-up surface between 0.01 to 0.1 mm²).

Surface EMG however, provides an overview of the muscle and is often used for that reason in kinesiological studies. During surface EMG, developed basically in the seventies and eighties e.g. by de Luca and co-workers [deL84], electrodes are placed on the skin of the human body. Surface EMG (SEMG) is thus a non-invasive technique, another significant advantage above needle EMG. SEMG is usually done with a small number of large electrodes (10 mm, 2-10 electrodes) and the spatial resolution is therefore not high. During recent research, however, smaller electrodes (1 mm, 128 electrodes) are used in a larger amount ([Roe97], [Mas91]). Interpretation of the surface profiles on the outside of the human body is much more difficult then needle EMG signals. The electrical signals from muscles have to pass a fat and skin layer and are thus spatially ‘filtered’ (transformed) during their journey in fat and skin tissue. Besides, action potentials from the muscle fibres in motor units interfere with each other which makes it more difficult to isolate specific components of a potential. To obtain a better diagnostic technique, analysis tools are used (and developed) to isolate components from action potentials to visualise differences between SEMG signals of healthy persons and persons with neuromuscular disorder (still in progress). Another way to a better understand SEMG is simultaneously use of surface recordings and triggering with a needle electrode. In the last years, the interest in using surface EMG to obtain information about muscle fibres has increased, not in the least because recent computer facilities allowed multi-channel surface EMG to be used for this purpose [Roe97]. However, the knowledge about surface EMG is still not sufficient enough to allow a widely used application. Hopefully this thesis contributes to the future steps to better comprehend surface electromyograms.

1.2. Muscle fibres and motor units

Interpretation of EMG signals is impossible without the knowledge about the morphology and physiology of a muscle. A description of a muscle fibre is therefore necessary to understand not only needle EMG but also SEMG (§ 1.1). A muscle consists out of long (up to tenths of cms) thin (up to tenths of μms) cells, the muscle fibres. Both ends are connected by tendons to bones. When muscle fibres are activated, they tend to shorten causing a muscle force and a possible displacement of a body segment. In the human body, three types of muscles are present (fig. 1.1):

- cross striated muscles,
- smooth muscles, and
- cardiac muscles.

Each of these show a different microscope pattern. In clinical neurophysiology voluntary activated striated muscles are investigated. Only these striated muscles will be considered in greater detail in this work.
How muscle fibres are orientated in the muscle and with respect to their tendons varies. In some muscles, fibres run parallel to each other and in line with the tendons. However, in the majority of the muscles, fibres are not in line with the tendons (and with each other). Bundles of muscle fibres are surrounded by connective tissue, containing blood vessels and nerve fibres. A muscle as a whole is also surrounded with the muscle facia, which allows movement of the muscle relative to other muscles or to the skin. Between the skin and the muscle, there is usually a layer of subcutaneous fat and in rare cases a small bone layer.

The activation of a muscle finds its origin in the central nervous system. This system generates, when necessary, motor neurons. Each of these motor neurons innervate a group of muscle fibres. A term for such a muscle group was introduced in 1929 by Sherrington. He did some stimulation experiments on a muscle group from an animal and discovered that the contraction of these muscles increased saltatorial with a gradual increment of the stimulus. From these experiments Sherrington concluded that the muscle is built up of units which he called motor units (MUs), the functional units of movement.

The number of muscle fibres innervated by one motor neuron, the innervation ratio, varies considerably between muscles. The smallest innervation ratios of less than 10 are found in the eye and the largest of more than 2000 are found in the large leg muscles. These innervation ratios vary within one muscle; the largest MUs of a muscle contain 10 times more fibres then the smallest MUs within that muscle.

The spatial distribution of muscle fibres belonging to one motor unit can be thought as randomly scattered in a circular shaped area. This is illustrated in fig. 1.2.b, a cross-section of a muscle.

The cross-section of a MU is certainly smaller than the cross-section of a whole muscle, but larger than the smallest cross-section necessary to contain all muscle fibres belonging to one. The actual cross-diameter of muscle fibres belonging to one MU varies between 2 and 15 mm [Buc57]. The diameter of a muscle fibre equals approximately 50 µm.

Chapter 1. Introduction to electromyography
The innervation of muscle fibres usually takes place midway the fibres at the so called motor endplates (fig. 1.2.a). However, because not all muscle fibres run parallel to each other or in direct line with the tendons, the innervation is not necessarily in the middle of the muscle, distributed endplates along scattered zones are possible [Mas91].

The muscle fibres, or muscle cells, are built up of a sacroplasma in which thin contracted elements are positioned, the so-called myofibrils. The muscle fibres themselves are surrounded by a membrane. Tissue outside the muscle fibre and its membrane is called the extracellular tissue, consisting of other fibres, nerves, fat, blood and skin. The muscle fibres (the intracellular tissue with the membrane) are attached with tendons to bones at the proximal (further away from the anatomical centre) and distal (closer to the anatomical centre) side of the body (fig. 1.3).

The myofibrils in the muscle fibre, with a cross-section of approximately 1 µm, each consist of 1000 to 2000 myofilaments. These myofilaments exist of thick myosin-elements and thin actin-elements. A contraction of a muscle is established, by the fact that the myosin elements and the actin elements are able to slide along each other and vice versa (fig. 1.4). Though essential for the muscle function, this process is beyond the scope of this study (see [Not81]). In this thesis only the bioelectrical processes on muscle fibre level are important.

The muscle fibres are activated in the motor endplate by a motor neuron. This implies that the muscle fibres are excited more or less simultaneously. The innervation generates an action potential in the muscle fibre, an intracellular action potential or single fibre action potential (SFAP). An observed signal belonging to a motor unit, called a motor unit action potential (MUAP), is in fact the sum of all potentials from the constituent fibres and shows some similarities with the SFAP. The motor unit action potential is measured extracellular with needles or surface electrodes. In § 1.3 the statement action potential will be further explained and illustrated.
1.3. EMG signals

The action potentials in muscle fibres are sources of activity on which needle EMG and surface EMG are based. The action potentials can be described by several lines of approach (mathematical, electrophysiological, physical, measurements). These lines of approach are revealed in this section.

1.3.1. Shape of an action potential

The shape of an intracellular potential against time is drawn in fig. 1.5. It represents schematically an approximated intracellular action potential in a muscle fibre. Before $T_0$ the muscle fibre is in rest and the potential in the muscle fibre is given by the so-called rest membrane potential, $R_{mp}$ (-90 to -75 mV). This is the potential with respect to the potential of the extracellular tissue (the potential exist in fact over the membrane, therefore it also called the transmembrane rest potential). The current flow through the membrane is in the rest state negligible because of the effectively high impedance of the surrounding membrane (sacrolemma).
At time $T_0$, the muscle is innervated at the motor end plate. This causes a potential change at the motor end plate. The potential at the motor end plate rises quickly where after it falls down, back to its resting state. This all happens in a time of 1 to 2 milliseconds depending on subject and muscle type (intracellular potential in fig. 1.5 is relatively short).

The potential change is not only occurring in the motor endplate region. When the muscle fibre is activated, the innervation at the motor end plate induces a travelling intracellular action potential along the muscle fibre. This happens as a result of electronic coupling from excited to not yet excited membrane areas over the muscle fibre membrane. The intracellular action potential travels thus to the distal respectively proximal side of the fibre, away from the motor end plate. That happens with a velocity, called the conduction velocity of muscle fibres. The conduction velocity depends on the type of muscle (fast and slow) and varies between 3 and 5 m/s [Are89].

1.3.2. Electrophysiology behind an intracellular action potential

The background of the transient positive-going changes of the intracellular action potential finds its origin in changes in the fibre membrane’s potassium ($K^+$) and sodium ($Na^+$) permeability [Sch83], initiated by the innervation (potential disruption) of the motor neuron. The basis of the excitation is an increase in the membrane conductance for sodium. This causes an increase of inflow of sodium whereafter the membrane is depolarised over a certain threshold. The inflow of sodium is followed by an outflow of Potassium which contributes to the repolarisation of the membrane and the return to its resting state (fig. 1.5.b).

1.3.3. Description of the action potential : a physical approach

The ionic current that will flow through the membrane is proportional to the second spatial derivative of the intracellular potential (Poisson equation, see chapter 2). The ionic current will mainly flow nearby the muscle fibre, however some part of it flows through a greater area of the volume conductor (compare with magnetic fields lines around a small magnet). This causes potential changes in the extracellular tissue, even in the skin. These potential changes are now the potentials we are interested in during SEMG.
The current flow through the membrane can be approximated by an electric current *tripole* ([Ros69], [Gri82]) after we simplify the intracellular potential. This is shown in fig. 1.6.

When the muscle fibre is in rest, the three monopoles can be thought as being at the motor endplate. During the innervation the first monopole starts travelling (to the proximal and distal side of the muscle) leaving the others behind (fig. 1.7, I). As the remaining ionic currents stay at the motor end plate we now have a net dipole strength with increasing magnitude.

Next, a second monopole, with a strength that is equal but opposite to the strengths of the other monopoles (one already travelling and the other at the motor endplate), starts. The first dipole (leading dipole) has its maximum moment, while the second trailing dipole (formed by the second monopole and the third monopole which still stays at the motor endplate region) has a moment that, with time, increasingly cancels the first moment (fig. 1.7, II).
When both moments exactly cancel each other, the third monopole begins its journey to the other end of the fibre. There is now no resulting dipole moment (fig. 1.7, III) and the tripole travels (fig. 1.7, IV) along the muscle fibre. At the other end of the fibre, exactly the opposite occurs, the end effect. The leading dipole moment gradually decreases (fig. 1.7, V) in strength while it runs up against the muscle-tendon transition (end of the fibre). Again, a net dipole moment results, which first increases (fig. 1.7, VI), then decreases in amplitude until at last the third monopole is absorbed and the muscle fibre returns to his rest state (fig. 1.7, VII). In fig. 1.8 we see an approximation of the potentials, belonging to these stages (the negative upward sign of the potentials in fig. 1.8 is opposite to the sign you might expect. In EMG however, this is the common way of presentation).

![Graph of potential along the muscle fibre for the stages in fig. 1.7.](image)

**Fig. 1.8. Potential along the muscle fibre for the stages in fig. 1.7.**

### 1.3.4. MUAPs at the skin due to the intracellular potentials at muscle fibre level

After the impulse transfer on the muscle fibres by the motor neuron two action potentials will propagate along the muscle fibre in opposite direction. The summed activity of all individual muscle fibres belonging to one MU is the motor unit action potential (MUAP). The amplitude as well as the spatial and temporal behaviour of these MUAPs at the skin surface are determined by three groups of parameters,

- the geometrical parameters of the MU in the muscle (orientation, depth, place of motor endplate, shape of MU territory),
- the properties of the muscle fibre (length, membrane, thickness, type of muscle, firing frequency, the intracellular action potential, conduction velocity), and
- the active and passive conduction properties of the tissue between the active fibres and the recording electrodes on the skin (bone, fat, muscle, skin, inactive fibres).

These three groups of parameters are of particular interest in the studies for the application of SEMG. The parameters determine the frequency range of the surface MUAP signals. In opposite to the frequency content of the intracellular action potential (500-2000 Hz, see fig. 1.5), the SEMG signals mostly consist of frequency components between 50 and 200 Hz (fig. 1.9). An experimental set-up for
surface EMG focuses in that area. This low frequency range enables investigators to use multi-channel surface EMG up to 128 channels, because of the relatively low sample frequency (2000 Hz), necessary to measure the low frequent SEMG signals precisely. For the interpretation of the results in chapter 5, this is an important figure.

![Power density (arbitrary units)](image)

**Fig. 1.9. Schematic power spectrum of SEMG.**

### 1.4. Brief anatomy of the human skin

The human skin is one of the conducting tissues between the muscle fibres and the recording electrodes on the skin. It consists of two main layers, the *epidermis* (‘upper skin’) and the *dermis* (‘leather skin’), the latter consists again of different layers (fig. 1.10).

The epidermis has four layers. The deepest layer, the *stratum basale*, produces new cells. These cells undergo slowly keratinization until they reach the surface of the skin at which stage the cells are completely keratinized (*stratum corneum*). This process takes three to four weeks and is continuously going on. The dermis itself consists sense-organs, blood vessels and binding tissue that gives the skin its firmness. This layer (*stratum papillare*) is connected with bulges to the epidermis.

![Layers of the human skin](image)

**Fig. 1.10. Layers of the human skin.**

The *subcutis* is a layer directly under the dermis. This layer contains also some binding tissue and without real interruption it shades off into the binding tissue of the *corium* where a great part of the fat tissue is situated, the heat-isolation of the human body.
1.5. Survey and goals of this study

This thesis can be subdivided into three different parts, each with its own emphasis:

- **Theoretical background of volume conduction**

  Volume conduction can be described by the solution of the Poisson equation. This can be done with finite element modelling [Spa97], the finite difference method or it can be done by solving the Poisson equation in an analytical way. This analytical solution has been a subject in several studies [Goo90, Blo95]. Still, it was not complete. The total solution for a three layer - muscle, fat and skin tissue - volume conductor was not available, a bone layer was not incorporated into the model and the outcome of the simulations were in arbitrary units. Hardly any attention was paid to the amplitude and duration of intracellular action potentials, the sources of the volume conduction. The theoretical background of volume conduction is treated in chapter 2.

- **Modelling of volume conductors with a LabWindows ® program**

  Simulations of EMG signals are helpful in analysing the results of measurements. However, simulations were possible only after understanding the theory discussed in chapter 2. The efficiency of performing these simulations was increased considerably by software that implements the formulas in chapter 2. This software also contains facilities to change volume conductor configurations and bioelectric source geometry. The program has been developed in LabWindows ® and makes the model a convenient tool for studying relations between simulations and measurements as well as the effects from several volume conductor configurations on the surface MUAPs. The program is also used in chapters 4 and 5 as a support of the measurements of the skin conductivity. The set-up and possibilities of the LabWindows ® program are dealt with in chapter 3. Chapter 3 also includes a calibration of the model, simulations with dynamic sources and the influence of a bone layer in the volume conductor.

- **Measurements to determine the conductivity of the skin under several conditions**

  Earlier research [Roe97] suggested that the passive conductivity of the skin had to be larger than the conductivity of fat tissue. To verify this, an experimental set-up was chosen which, in co-operation with theory, would lead to a value for the conductivity of the human skin. The theory is described in chapter 4, while in chapter 5 the results of these measurements are shown. Relations between current, skin conductivity and temperature are also investigated in chapter 5.

Of course, measurements are intrinsically more important than modelling. However, real understanding is achieved with a combination. The main goal of this thesis thus is a better understanding of surface electromyography with help of theory, modelling and measurements of the conductivity of the skin. The conclusions of this thesis are finally presented in chapter 6.
Chapter 2

Theory of volume conduction in an inhomogeneous and anisotropic cylinder

In this chapter, the theoretical background of volume conduction in a cylinder is treated. To describe volume conduction in a cylinder we have to solve the Poisson equation. This is done in § 2.1 for an anisotropic cylindrical volume conductor, consisting of four cylindrical layers with the properties of bone, muscle, fat and skin tissue, a reasonable approximation of some parts of the human body. In § 2.2 the formulas for volume conduction are made discrete (computer modelling demands these discrete terms). In § 2.3 measured and simulated intracellular potentials are mathematically described in terms of a current strength, representing the muscle fibre activity. If this represents the muscle fibre activity well, simulations in 'real' μV can be obtained.

2.1. Solving the Poisson equation

2.1.1. Poisson equation

To describe volume conduction, the relationship between current flow and potential field is essential. In electromagnetic theory this relation is formed by the Poisson equation. For a homogeneous and isotropic volume conductor the Poisson equation [Jac75] in cylindrical co-ordinates is given by

\[ \nabla^2 \Phi = \frac{1}{\sigma} \left( \frac{\partial}{\partial \rho} (\rho J_\rho) + \frac{\partial}{\partial \varphi} J_\varphi + \frac{\partial}{\partial z} J_z \right) \]

(2.1.1)

\( \rho, \varphi \) and \( z \) are cylindrical co-ordinates, orientated according to fig. 2.1. \( \Phi \) represents the (extracellular, § 1.2) potential field on or in the volume conductor, \( J(\rho,\varphi,z) \) the current source density (A/m²) in the volume conductor caused by active muscle fibres and \( \sigma \) the conductivity of the cylindrical volume conductor with respect to current, (Ωm)⁻¹.

\( \nabla \cdot J = -i_c \) is the volume current source density (A·m⁻³) and forms an analogue of charge density in electrostatics. Capacitive and inductive effects are ignored in (2.1.1). For the frequencies contained in biological tissue during surface electromyography (EMG), the conditions are supposed to be met [God90, Gab96] for this assumption. However, it is still an assumption which remains to be verified to confirm this so-called quasi-stationarity.
The Poisson equation is in the following sections analytically solved. That means that the tissue layers of the volume conductor must have a cylindrical configuration, symmetrically disposed around the centre of the cylinder.

The total solution of the Poisson equation is now a superposition of the solutions of the homogeneous and the inhomogeneous part of the Poisson equation. The solutions of these two equations are calculated in § 2.1.2 and § 2.1.3.

2.1.2. Homogeneous solution

First we derive the homogeneous solution by solving the homogeneous part of (2.1.1), better known as the Laplace equation, (2.1.2). The Laplace equation is determined by the geometry or configuration of the cylinder and the current source density at the boundaries (§ 2.1.5). In an infinite cylinder would the homogeneous solution be negligible, therefore it is also called the finite solution.

\[ \nabla^2 \Phi(\rho, \varphi, z) = \frac{1}{\rho} \frac{\partial}{\partial \rho} \left( \rho \frac{\partial \Phi}{\partial \rho} \right) + \frac{1}{\rho^2} \frac{\partial^2 \Phi}{\partial \varphi^2} + \frac{\partial^2 \Phi}{\partial z^2} = 0 \]  

(2.1.2)

When we apply Fourier transformation in the z-direction on (2.1.2) (Appendix A.1.1) - in order to get a mathematically better solvable equation - we obtain (2.1.3),

\[ \frac{1}{\rho} \frac{\partial}{\partial \rho} \left( \rho \frac{\partial \Phi^*}{\partial \rho} \right) + \frac{1}{\rho^2} \frac{\partial^2 \Phi^*}{\partial \varphi^2} - k^2 \Phi^* = 0. \]  

(2.1.3)

The derivatives in \( \rho \) and \( \varphi \)-direction are unchanged in (2.1.3). In \( z \)-direction, however, a new variable is introduced, \( k^* \). \( k^* \), called the spatial frequency, is a consequence of the Fourier transformation and represents the repetency of \( \Phi \) in \( z \)-direction (\( \cos 2\pi k^*z, \sin 2\pi k^*z \)). In (2.1.3) \( k \) is used, which is the product of \( 2\pi \) and \( k^* \), this to simplify (2.1.3).
The potential in (2.1.3), $\Phi^*$, is the potential with co-ordinates $\rho$, $k$ and $\varphi$. With separation of variables (step-by-step done in Appendix A.1.2), we arrive at the solution of (2.1.3),

$$
\Phi^*(\rho,k,\varphi) = \sum_{n=-\infty}^{\infty} e^{-in\varphi} (A_n I_n(|k|\rho) + B_n K_n(|k|\rho))
$$

(2.1.4)

and after inverse Fourier transformation in $z$-direction the solutions of (2.1.2) become,

$$
\Phi(\rho,z,\varphi) = \sum_{n=-\infty}^{\infty} e^{-in\varphi} F_{\text{inverse}}^{-1} (A_n I_n(|k|\rho) + B_n K_n(|k|\rho)).
$$

(2.1.5)

The variables $A_n$ and $B_n$ in (2.1.4) and (2.1.5) are determined by boundary conditions which will be a subject later again in § 2.1.5. $I_n(k\rho)$ and $K_n(k\rho)$ are modified Bessel functions which means that they are real-valued for real arguments, here the spatial frequency $k$ and the radius $\rho$. $n$ in (2.1.4) and (2.1.5) is an integer, a result of physical periodicity in angular direction (Appendix A.1.2).

### 2.1.3. Inhomogeneous solution

The next part we have to solve is the inhomogeneous differential equation (the particular equation), presented by equation (2.1.1) itself,

$$
\nabla^2 \Phi = \frac{1}{\rho} \frac{\partial}{\partial \rho} (\rho J_\rho) + \frac{1}{\rho} \frac{\partial}{\partial \varphi} J_\varphi + \frac{\partial}{\partial z} J_z
$$

(2.1.1), (2.1.6)

The solution of this equation depends on the source and not on the boundaries of the cylinder. It is therefore called the infinite solution or source-dependent solution. The inhomogeneous solution plays the dominant role in the total solution of the Poisson equation. The finite solutions, the solutions of the Laplace equation, however, are in most finite cylinder configurations not negligible.

For the simplest configuration, in which the muscle fibre is assumed to be placed in an infinite, isotropic medium, the potential from a point-shaped source $i$ measured at a distance $R$ can be calculated [Jac75] with the Coulomb equation,

$$
\Phi = \frac{1}{4\pi \sigma} \frac{i}{R}.
$$

(2.1.7)

In EMG muscle fibres are responsible for the action potential. So these muscle fibres are responsible for the potentials in the volume conductor. When we handle the line-source approximation we assume the fibre diameter to have diameter zero, with respect to the distance between the source point and the observation point, which is reasonable if you compare the diameter of a muscle fibre with the diameter of a volume conductor representing e.g. a human upper arm (50 $\mu$m vs. 8 cm). The extracellular potential from a muscle fibre in infinite homogeneous tissue can then be expressed as

---

*Chapter 2. Theory of volume conduction in an inhomogeneous and anisotropic cylinder*
\[ \Phi(\rho, z) = \frac{1}{4\pi\sigma} \int_{-L}^{L} \frac{2\pi a J(z_s) \, dz}{\sqrt{\rho^2 + (z - z_s)^2}} \] (2.1.8.a)

with \( a \) as the radius of the fibre, \( 2L \) its length, \( z_s \) the locations of the point sources and \( J(z_s) \) the current source density along the membrane \((A/m^2)\). Calculations are performed, like with the homogeneous equation, in the spatial frequency domain expressed by the spatial frequency \( k \). Because of the line source approximation it is logical to replace \( 2\pi a J(z) \) by \( I(z) \), which represents the current strength in \( A/m \) along the fibre

\[ I(z) = 2\pi a J(z) \] (2.1.8.b)

In combination with the use of modified Bessel functions we derive for an infinite cylinder and a muscle fibre on the centre axis of a cylindrical volume conductor [Bat54]

\[ \Phi'(\rho, k) = \frac{1}{2\pi\sigma} K_0(k\rho) I(k). \] (2.1.9)

(2.1.9) is thus the inhomogeneous solution of (2.1.6). When the muscle fibre lays eccentrically in the cylinder it changes into (2.1.10),

\[ \Phi'(r, k) = \frac{1}{2\pi\sigma} K_0(kr) I(k) \] (2.1.10)

with \( r \) as the distance between the point sources and the observation point (placement of electrode). The drawing in fig. 2.2 illustrates this co-ordinate \( r \) together with the cylinder co-ordinates in fig. 2.1. With the cosine-rule a relationship is derived (2.1.11) between \( \rho_s \) (radius source), \( \rho_{obs} \) (radius observation point), \( \phi_s \) (angle source), \( \phi_{obs} \) (angle observation point), \( z_s \) (axial co-ordinate source), \( z_{obs} \) (axial co-ordinate observation point) and \( r \). Fig. 2.2 gives also an impression of a cylindrical volume conductor consisting of centrically positioned layers with the properties of a bone, muscle, fat and skin layer.

\[ r = \sqrt{(\rho_{obs} - \rho_s)^2 + (z_{obs} - z_s)^2 - 2(\rho_{obs} - \rho_s)(z_{obs} - z_s)\cos(\phi_{obs} - \phi_s)}. \] (2.1.11)
2.1.4. Cylindrical symmetry

Due to the cylindrical symmetry in the analytical method, the muscle fibres have to be positioned parallel to the centre of the cylinder. The solutions in angular direction in (2.1.4) and (2.1.10) are consequently symmetrical. Thus, when the muscle fibres, are positioned parallel with the z-axis under an angle $\varphi = 0^\circ$, the solutions for $\varphi = 0...180^\circ$ equal the solutions for $360^\circ - \varphi$. In theory simulations of oblique fibres are of course possible, if one calculates for every source point again, the solution of the Poisson equation in the finite volume conductor. A tricky summation of these solutions after these ‘time-consuming’ calculations would eventually lead to a simulated oblique fibre (the source points form together the oblique fibre).

2.1.5. Anisotropic volume conductor

The conductivities of the tissue layers are, besides the dimensions of the layers, very important properties of the volume conductor during simulations. The conductivities from the tissue layers do not only differ from each other, there could also be a difference between the radial and axial conductivity within the same tissue layer.

The solutions (2.1.4) and (2.1.10) do account for an isotropic cylindrical volume conductor. It is however possible to simulate an anisotropy in the volume conductor concerning the conductivity between the radial and axial direction. By this, (2.1.1) changes into

$$
\nabla^2 \Phi(\rho, \varphi, z) = \frac{1}{\rho} \frac{\partial}{\partial \varphi} \left( \rho J_\rho \right) + \frac{1}{\rho} \frac{\partial}{\partial \varphi} \left( J_\varphi \right) + \frac{\partial}{\partial z} \left( J_z \right) .
$$

(2.1.12)

$\sigma_\rho$ represents the conductivity in axial direction $(\Omega m)^{-1}$ and $\sigma_\rho$ that in radial direction $(\Omega m)^{-1}$. This form of anisotropy is definitely present in muscle tissue. Parallel to the muscle fibres the conductivity is normally higher than perpendicular to the muscle fibres because of the high membrane impedance of inactive surrounding muscle fibres (§ 1.3).

The solutions of the homogeneous and inhomogeneous equation change, but not drastically. From (2.1.4) we obtain (2.1.13) and (2.1.10) is ‘transformed’ into (2.1.14),

$$
\Phi^* (\rho, k, \varphi) = \sum_{n=-\infty}^{\infty} e^{-in\varphi} \left( A_n I_n (\rho \sqrt{\frac{\sigma_\rho}{\sigma_\varphi}} k) + B_n K_n (\rho \sqrt{\frac{\sigma_\rho}{\sigma_\varphi}} k) \right),
$$

(2.1.13)

$$
\Phi^* (\rho, k) = \frac{1}{2\pi \sigma_\rho} K_0 \left( k r \sqrt{\frac{\sigma_\rho}{\sigma_\varphi}} \right) I(k).
$$

(2.1.14)

The square root terms in (2.1.13) and (2.1.14) more or less represent virtual longer distances for current to travel when the conductivity is lower for a specific direction.
2.1.6. Total solution

With the derivations of the homogeneous solution (2.1.13) and the inhomogeneous solution (2.1.14) in §2.1.5, consider now a cylindrical volume conductor with four concentric boundaries. One may think e.g. of an upper arm consisting of bone tissue in the inner compartment (radius $a$), muscle tissue in a layer around it (radius $b$), a next layer of fat tissue (radius $c$) and skin tissue as the outer compartment (radius $d$). The volume conductor is finally surrounded by air (with conductivity zero), see fig. 2.2.

The general solution of the Poisson’s equation in cylindrical co-ordinates with spatial frequency $k$, (2.1.3), then reads for the described volume conductor at a certain observation point $(\rho_{\text{obs}}, k, \varphi_{\text{obs}})$ lying in the bone, muscle, fat or skin layer,

$$\Phi_{\text{bone}}(\rho_{\text{obs}}, k, \varphi_{\text{obs}}) = \frac{1}{2\pi \sigma_{1,\rho}} K_0(r \sqrt{\frac{\sigma_{1,\rho}}{\sigma_{1,\rho}}} |k|) I(k) + \sum_{n=\infty}^{n=\infty} e^{-i\varphi_{\text{obs}}} [A_n(k)I_n(\rho_{\text{obs}} \sqrt{\frac{\sigma_{1,\rho}}{\sigma_{1,\rho}}} k)]$$

(2.1.15.a)

$$\Phi_{\text{muscle}}(\rho_{\text{obs}}, k, \varphi_{\text{obs}}) = \frac{1}{2\pi \sigma_{2,\rho}} K_0(r \sqrt{\frac{\sigma_{2,\rho}}{\sigma_{2,\rho}}} |k|) I(k) + \sum_{n=\infty}^{n=\infty} e^{-i\varphi_{\text{obs}}} [C_n(k)I_n(\rho_{\text{obs}} \sqrt{\frac{\sigma_{2,\rho}}{\sigma_{2,\rho}}} k)]$$

(2.1.15.b)

$$\Phi_{\text{fat}}(\rho_{\text{obs}}, k, \varphi_{\text{obs}}) = \frac{1}{2\pi \sigma_{3,\rho}} K_0(r \sqrt{\frac{\sigma_{3,\rho}}{\sigma_{3,\rho}}} |k|) I(k) + \sum_{n=\infty}^{n=\infty} e^{-i\varphi_{\text{obs}}} [E_n(k)I_n(\rho_{\text{obs}} \sqrt{\frac{\sigma_{3,\rho}}{\sigma_{3,\rho}}} k)]$$

(2.1.15.c)

$$\Phi_{\text{skin}}(\rho_{\text{obs}}, k, \varphi_{\text{obs}}) = \frac{1}{2\pi \sigma_{4,\rho}} K_0(r \sqrt{\frac{\sigma_{4,\rho}}{\sigma_{4,\rho}}} |k|) I(k) + \sum_{n=\infty}^{n=\infty} e^{-i\varphi_{\text{obs}}} [G_n(k)I_n(\rho_{\text{obs}} \sqrt{\frac{\sigma_{4,\rho}}{\sigma_{4,\rho}}} k)]$$

(2.1.15.d)

The solutions of the Poisson equation (2.1.15.a,b,c,d) account for the potentials in the various tissues: $\Phi_1$ represents the potential field in the inner compartment (bone tissue), $\Phi_2$ the potential in the surrounding muscle tissue layer, $\Phi_3$ the potential in the next layer (fat layer) and $\Phi_4$ as the potential in the outer skin layer. They are all expressed in the spatial frequency domain ($\Phi$ is in V·m). $\sigma_{1,\rho}, \sigma_{2,\rho}, \sigma_{3,\rho}, \sigma_{4,\rho}$ and $\sigma_{1,\rho,\rho}, \sigma_{2,\rho,\rho}, \sigma_{3,\rho,\rho}, \sigma_{4,\rho,\rho}$ are the conductivities ($\Omega$m)$^{-1}$ in the axial respectively radial direction for the bone ($\sigma_{1,\rho}$), muscle ($\sigma_{2,\rho}$), fat ($\sigma_{3,\rho}$), and skin tissue ($\sigma_{4,\rho}$). Furthermore, a potential of zero is defined at infinity. No source would thus result in a zero-potential in the whole volume conductor.
A_n(k), C_n(k), D_n(k), E_n(k), F_n(k), G_n(k) and H_n(k) are variables of k, the spatial frequency. One would also expect B_n(k) in (2.1.15.a). However, B_n(k) cannot exist. \( \Phi_i \), the potential of the bone tissue belongs to the inner compartment of the cylinder. So, the radius of observation, \( \rho_{ob} \), can equal zero. \( K_n(0) \) is however infinite, so, \( B_n(k) \) must equal zero to exclude infinite potentials.

In case of an eccentrically located fibre and when \( \rho_{ob} > \rho \) (2.1.14) is expandable, following the addition theorem for Bessel-functions [Gra66], to

\[
K_0 \left( r \frac{\sigma_{z1, z2, z3, z4}}{\sigma_{p1, p2, p3, p4}} \right) = \sum_{n=-\infty}^{\infty} e^{-i\rho_{ob} \rho_n} \sum_{\rho_{ob}} \frac{\sigma_{z1, z2, z3, z4}}{\sigma_{p1, p2, p3, p4}} k_n(\rho, \frac{\sigma_{z}}{\sigma_{\rho}}). \tag{2.1.16}
\]

with \( \sigma_{p1} \) and \( \sigma_{z1} \) in case of the first term of (2.1.15.a), \( \sigma_{p2} \) and \( \sigma_{z2} \) in case of the first term of (2.1.15.b) and so on.

The total solution of the Poisson equation in the cylinder is determined by the boundary conditions between the various layers of the volume conductor. These boundary conditions account for the potential fields and the current flow. The boundary conditions are the missing equations to determine the variables \( A_n(k), C_n(k), D_n(k), E_n(k), F_n(k), G_n(k) \) and \( H_n(k) \).

\[
\Phi_1 \bigg|_{\rho=a} = \Phi_2 \bigg|_{\rho=a} \tag{2.1.17.a}
\]
\[
\Phi_2 \bigg|_{\rho=b} = \Phi_3 \bigg|_{\rho=b} \tag{2.1.17.b}
\]
\[
\Phi_3 \bigg|_{\rho=c} = \Phi_4 \bigg|_{\rho=c} \tag{2.1.17.c}
\]
\[
\sigma_1 \frac{\partial \Phi_1}{\partial \rho} \bigg|_{\rho=a} = \sigma_2 \frac{\partial \Phi_2}{\partial \rho} \bigg|_{\rho=a} \tag{2.1.17.d}
\]
\[
\sigma_2 \frac{\partial \Phi_2}{\partial \rho} \bigg|_{\rho=b} = \sigma_3 \frac{\partial \Phi_3}{\partial \rho} \bigg|_{\rho=b} \tag{2.1.17.e}
\]
\[
\sigma_3 \frac{\partial \Phi_3}{\partial \rho} \bigg|_{\rho=c} = \sigma_4 \frac{\partial \Phi_4}{\partial \rho} \bigg|_{\rho=c} \tag{2.1.17.f}
\]
\[
\sigma_4 \frac{\partial \Phi_4}{\partial \rho} \bigg|_{\rho=a} = 0 \tag{2.1.17.g}
\]

The first three equations are continuity relationships for the potential, valid at the interface between bone and muscle tissue (2.1.17.a), respectively muscle and fat tissue (2.1.17.b) and fat and skin tissue (2.1.17.c). The next three equations stand for continuity of current at the interfaces between bone and muscle tissue (2.1.17.d), muscle and fat tissue (2.1.17.e) respectively fat and skin tissue (2.1.17.f). The last equation (2.1.17.g) illustrates the fact that no current can cross the interface between the skin tissue and the isolating outer layer (normally air).
With these equations and the four solutions, $\Phi \ell, \Phi \ell, \Phi \ell, \text{ and } \Phi \ell$, we obtain after substitution 7 equations and 7 unknown variables $A_n(k), C_n(k), D_n(k), E_n(k), F_n(k), G_n(k), H_n(k)$ for every integer value of $n \text{ and } k$, (2.1.18). There are a few abbreviations in (2.1.18). These need a further explanation and are thereby enumerated in (2.1.19) and (2.1.20). Solving (2.1.18) will eventually lead to the total solution of the Poisson equation.

\[
\begin{pmatrix}
I_n(a_k) & -I_n(a_k) & -K_n(a_k) & 0 & 0 & 0 & 0 \\
0 & I_n(b_k) & K_n(b_k) & -I_n(b_k) & -K_n(b_k) & 0 & 0 \\
0 & 0 & 0 & I_n(c_k) & K_n(c_k) & -I_n(c_k) & -K_n(c_k) \\
\sigma_1I_n(a_k) & -\sigma_2I_n(a_k) & \sigma_3K_n(a_k) & 0 & 0 & 0 & 0 \\
0 & \sigma_1I_n(b_k) & \sigma_2K_n(b_k) & -\sigma_1I_n(b_k) & -\sigma_2K_n(b_k) & 0 & 0 \\
0 & 0 & 0 & \sigma_1I_n(c_k) & \sigma_2K_n(c_k) & -\sigma_1I_n(c_k) & -\sigma_2K_n(c_k) \\
0 & 0 & 0 & 0 & 0 & \sigma_1I_n(d_k) & \sigma_2K_n(d_k) \\
\end{pmatrix}
\begin{pmatrix}
A_n \\
C_n \\
D_n \\
E_n \\
F_n \\
G_n \\
H_n
\end{pmatrix}
=
\frac{I(k)}{2\pi}
\begin{pmatrix}
I_n(R_k)K_n(a_k) & -I_n(R_k)K_n(a_k) \\
\sigma_1 & \sigma_2 \\
I_n(R_k)K_n(b_k) & -I_n(R_k)K_n(b_k) \\
\sigma_2 & \sigma_3 \\
I_n(R_k)K_n(c_k) & -I_n(R_k)K_n(c_k) \\
\sigma_3 & \sigma_4 \\
-I_n(R_k)K_n(d_k) & \sigma_4 \\
\end{pmatrix}

(2.1.18)

\[
\sigma_1 = \sqrt{\sigma_1, \sigma_2, \sigma_3, \sigma_4}, \quad \sigma_2 = \sqrt{\sigma_1, \sigma_2, \sigma_3, \sigma_4}, \\
\sigma_3 = \sqrt{\sigma_1, \sigma_2, \sigma_3, \sigma_4}, \quad \sigma_4 = \sqrt{\sigma_1, \sigma_2, \sigma_3, \sigma_4},
\]

(2.1.19)

\[
x_1 = \sqrt{\frac{\sigma_1}{\sigma_1}}x, \quad x_2 = \sqrt{\frac{\sigma_2}{\sigma_2}}x, \\
I''_{\ell}(x) \equiv \frac{\partial I_{\ell}(x)}{\partial x} = \frac{I_{\ell+1}(x) + I_{\ell-1}(x)}{2},
\]

(2.1.20)

\[
K''_{\ell}(x) \equiv \frac{K_{\ell+1}(x) + K_{\ell-1}(x)}{2} = \frac{\partial K_{\ell}(x)}{\partial x}.
\]
2.2. Discrete model

It is most unfeasible to solve the matrix in § 2.1, (2.1.18), without the help of a computer. To be able to do the calculations a windows-based program is developed which simplifies simulations (chapter 3). In this section and in § 2.3 we look at the consequences of the implementation of the volume conductor model in a computer program and especially at the accuracy of the solutions and the resolution of the volume conductor model.

With computer modelling we are not able to solve the problem (Poisson equation and boundary conditions) on a continuous basis. Because of limitations of the computer (and time), a calculation of infinite terms in (2.1.15.a.b.c.d) and (2.1.16) for the angular frequency \( n \) and the spatial frequency \( k \) is practically impossible and thus we have to cope with a discrete problem. Even if we could calculate infinite terms, \( I(x) \) is determined by measurements (at the intracellular potential) with a limited sample frequency. Discrete modelling does not have a great impact if we are able to take many discrete steps. Again, there are however limitations on the amount of discrete steps, so one should be aware of the mathematical consequences.

A continuous function \( f(x) \) can be made discrete by means of a sequence \( \{ f(x_0), f(x_0 + \Delta x), f(x_0 + 2\Delta x), ..., f(x_0 + (N-1)\Delta x) \} \) with sampling in \( N \) points and interdistance \( \Delta x \). We obtain a discrete \( f(z) \) which is defined by the following notation,

\[
f(z) = f(z_0 + z\Delta z),
\]

with \( z = 0, 1, 2, ..., N-1 \), as the discrete count variable. The sequence \( \{ f(0), f(1), f(2), ..., f(N-1) \} \) will be used to express from a corresponding function \( f(z) \) a sequence of \( N \) equidistant samples. \( z_0 \) is the offset of the discrete description, and has the value zero in most cases.

With this notation the discrete Fourier transformation terms can be defined,

\[
F(u) = \frac{1}{N} \sum_{z=0}^{N-1} f(z) e^{-j2\pi uz/N}
\]

for \( u = 0, 1, ..., N-1 \) and the inverse discrete Fourier transformation,

\[
f(z) = \sum_{u=0}^{N-1} F(u) e^{j2\pi uz/N}
\]

for \( z = 0, 1, ..., N-1 \).

The values \( u = 0, 1, ..., N-1 \) correspond with samples of the continuous transformation for the values \( u = 0, \Delta u, 2\Delta u, ..., (N-1)\Delta u \). We represent \( F(u\Delta u) \) thus by \( F(u) \).
We have to apply this discrete Fourier transformation in axial direction,

\[
\Phi^*(\rho, \varphi, k) = \frac{1}{N_z} \sum_{z=0}^{N_z-1} \Phi(\rho, \varphi, z_0 + z\Delta z) e^{-j2\pi k z/N_z}.
\]  

(2.2.4)

Equation (2.2.4) implies that the resolution of the calculated potential is determined by \( k \) in the Fourier transformed domain. Thus, the larger the maximal spatial frequency \( k \), so much the better is the resolution of \( \Phi^* \) in axial direction. In \( z \)-domain this implies that \( \Delta z \) becomes smaller and that the amounts of sampling points in the \( z \)-direction, \( N_z \), becomes larger.

For the angular direction \( n \) is an indication for the resolution in angular direction. \( \Phi^* \) is 'restricted' in \( \varphi \) direction as in axial direction, caused by the need of periodicity in angular direction. We are thus able to say that the larger the amount of sampling points in angular direction, \( N_\varphi \), the better the resolution of \( \Phi^* \) in angular direction.

Equation (2.1.4), the homogeneous solution of the Poisson equation, becomes in discrete terms

\[
\Phi^*(\rho, k, \varphi) = \sum_{n=-N_\varphi}^{N_\varphi} e^{-j n \varphi} \sum_{k=0}^{N_z} \frac{1}{N_z} \left( A_n I_n(\rho z) + B_n K_n(\rho z) \right) e^{-j2\pi k z/N_z}.
\]  

(2.2.5)

while the first part of (2.1.15.a.b.c.d), the inhomogeneous solution, becomes,

\[
\Phi(\rho_{\text{ion}}, k, \varphi_{\text{ion}}) = \frac{1}{2\pi \sigma_{1,2,3,4,p}} \sum_{z=2}^{N_z} K_0(r) \sqrt{\frac{\sigma_{1,2,3,4,p}}{\sigma_{1,2,3,4,p}(z)}} I(z) e^{-j2\pi k z/N_z}.
\]  

(2.2.6)

We obtain the total discrete solution of the Poisson equation if we add (2.2.5) and (2.2.6) together.

2.3. Simulations in \( \mu \text{V} \)

2.3.1. Discrete current strength

An important improvement of the volume conductor model is made when the volume conductor model yields results in 'real' voltages (during SEMG normally between 1-500 \( \mu \text{V} \)). The ability to simulate muscle fibres in a quantitative way - thus the possibility to simulate real strength of the muscle fibres and calculate real potentials at the skin surface - could for example be a helpful tool for quantifying the amount of motor units in a muscle group. First, however, we have to determine the real strength of the sources, the active muscle fibres, for a simulation of surface MUAPs.
\( I(z) \), the current strength, is linked to the intracellular potential of the muscle fibres when these are active. The intracellular action potential is in this section represented by a mathematical approximation of muscle fibre activity. The basics of this section do however also account for other (measured) intracellular potentials.

\( \Phi_j(z) \), the intracellular potential in active muscle fibres can thus mathematically be described by [Ros69]

\[
\Phi_j(z) = A z^3 e^{-xz} - B, \quad z \geq 0.
\]  (2.3.1)

The parameters of the description of the intracellular action potential, \( A, B \), and, \( \lambda \) are listed in table 2.1. The intracellular potential is shown in fig. 2.3.a, its first and second derivative in fig. 2.3.b and fig. 2.3.c.

The second derivative of the intracellular potential is important, if we again look back at the Poisson equation, (2.1.1). The intracellular potential, \( \Phi_j \) and the current source density \( J(z) \) in the muscle fibre, in this section in fact the volume conductor, are related by this equation,

\[
\nabla^2 \Phi_j = \frac{\nabla \cdot J}{\sigma_{\text{intra}}},
\]  (2.3.2)

with \( \sigma_{\text{intra}} \) as the conductivity of the intracellular tissue of the muscle fibres.
When the line-source approximation (§ 2.1) is applied on this equation we derive

\[ I(z) = \pi a^2 \sigma_{\text{intra}} \frac{d^2 \Phi_i(z)}{dz^2} \]  

(2.3.3)

wherein \( a \) represents the radius of the muscle fibre and \( I(z) \) is thus the current strength of the active muscle fibres.

The last term in (2.3.3) is the second derivative of the potential (fig. 2.3.c),

\[ \frac{d^2 \Phi_i(z)}{dz^2} = Az(6 - 6\lambda z + \lambda^2 z^2) e^{-\lambda z} \]  

(2.3.4)

In discrete terms (2.3.3) will become

\[ I(z) = \pi a^2 \sigma_{\text{intra}} \cdot A(z_s + z\Delta z)(6 - 6\lambda(z_s + z\Delta z) + \lambda^2(z_s + z\Delta z)^2) e^{-\lambda(z_s + z\Delta z)} \]  

(2.3.5)

with \( z \) as the discrete count variable, \( z = 0, 1, 2, ..., N-1 \) (\( N \) points) and \( \Delta z \) is the interdistance between the sampling points. \((N-1)\Delta z\) equals the total length of the volume conductor, the cylinder. The Fourier transformation of \( I(z) \) equals with (2.2.2)

\[ I(k) = \frac{1}{N} \sum_{z=0}^{N-1} I(z\Delta z) e^{-j2\pi k z \Delta z} \]  

(2.3.6)

2.3.2. Resolution of the volume conductor model

Discrete calculations of the Bessel terms are in computer modelling limited by machine accuracy and time. However, calculations could be done with a spatial frequency up to 1024 \( m^{-1} \). This means that in a cylinder with a length of 15 cm the calculated potential has a resolution of 0.14 mm (15 cm/1024) in axial direction. That is sufficient for simulations of MUAPs. Significant frequency components of the intracellular potentials do namely not exceed 2000 Hz (fig. 1.5). With a conduction velocity between 3 and 5 m/s in the muscle fibres we notice that the width of an single fibre action potential during excitation equals 3 mm or more. With a resolution of 0.14 mm this intracellular potential could already be described with 22 points (3 mm/0.14 mm) in the worst case (duration action potential 1 ms, conduction velocity 3 m/s).

Chapter 2. Theory of volume conduction in an inhomogeneous and anisotropic cylinder
If we take now a measured intracellular potential as an input for the current strength for the simulation model (§ 2.3.1), it would of course be the best if the sampling frequency of this intracellular potential during the measurement is as much or better than the resolution of the simulation model. For a potential travelling along the muscle fibre with a maximal conduction velocity of 5 m/s and a width of the action potential of 1 ms the intracellular potential itself has a axial width of approximately 5 mm. To obtain a value for the intracellular potential every 0.14 mm in the intracellular potential a sample frequency of at least 28 kHz \((5 \text{mm/0.14mm*/1/1ms})\) is necessary.

This sample frequency is easily obtained during the measurements of intracellular action potentials. The restraining resolution factor is thus the resolution of the simulated volume conductor and not the resolution of the measured intracellular potential. Sometimes this induces that the intracellular action potential needs to be interpolated to obtain points with an interdistance equally to the resolution of the cylindrical volume conductor (from fig. 2.4.b to fig. 2.4.a). Decreasing the length of the cylinder - with the intention to improve the resolution of the simulated volume conductor - is of course possible, however, if we want to simulate realistic muscle fibres with a length of 8 cm, the simulated volume conductor has to have at least that length.

In the past, the intracellular potential was approximated by a triangular shape and the current strength was consequently simulated by a tripole (fig. 1.6, fig. 1.7). By simulating this tripole one applied two conditions, this to ascertain that there is no net intracellular voltage change after the passing of the action potential (i.e. no net resulting dipole moment),

\[
\sum_{i=1}^{N} I_i = 0, 
\]

\[
I_1 D_1 = I_2 D_2. 
\]

Fig. 2.4. Discrete \(I(z)\) of the Rosenfalck potential for a. \(dz = 0.78 \text{ mm}\) b. \(dz = 0.39 \text{ mm}\).
Apart from these conditions, the current strengths of the simulated tripole were chosen in arbitrary units. With the theory in this section and § 2.3.1, one is however able to simulate motor units by a real current strength (of course (2.3.7) and (2.3.8) also count for the real current strength in fig. 2.4.a. and b with $i=1..12$ and $i=1..24$). Prudence is in order during simulations with one and the same source in two or more different (concerning the length !) volume conductor configurations, because the current strength is in $A/m$; the total current of $1\ A/m$ over $1\ mm$ (e.g. the resolution of a volume conductor) does not equal a current of $1\ A/m$ over $2\ mm$ (e.g. the resolution of another volume conductor). This is unfortunately a disadvantage of discrete calculations.
Chapter 3

Implementation in a user interface model

The solutions of the analytic volume conductor model in chapter 2 give us the opportunity to simulate several volume conductor configurations and the related surface MUAP signals from intracellular potentials. Several reasons (efficiency, simulation possibilities, view angles) caused us to present the analytic model into an user friendly interface with options to calculate potentials for different kind of cylinders, tissue layers, sources, orientation of the sources and more. A short set-up of this windows-based program is made in § 3.1. In § 3.2, the model is 'calibrated'. Furthermore, the matrix terms in (2.2.18) need to be scaled to obtain a solution. This scaling was satisfactory for a two and three layer model, a fourth layer (bone tissue) however complicated the calculations and the limitations of the Bessel terms in the analytical solution were visible (§ 3.3). The simulations in chapter 4 and other parts of this thesis were done with this program.

3.1. A user interface model for the analytic volume conductor

The theory in chapter 2 has been implemented during this research in a windows-based user interface program, developed with LabWindows/ CVI®, based on C, C++ and windows application tools. The total program is available under the name Anvolcon and can be considered as the final product of the group concerning the analytical approach of volume conduction in EMG in the past years.

The user interface is divided in three panels (i.e. sections):

- a panel for source width, source orientation, source strength (the intracellular potential) and the amount of fibres,
- a panel for the properties of the volume conductor, and
- a panel for the final calculations of the MUAPs on the volume conductor.

The results of the calculations can also be transported to Matlab for Windows® for further implementation. A schematic scheme of the program is drawn in fig. 3.1.

3.1.1. Source parameters

The panel for source parameters allows the user to set the type of source by inputting a measured intracellular action potential in the volume conductor along the muscle fibre (the duration from the potential change can be changed by an input of a sample frequency and conduction velocity). Other imaginary sources can also be taken as source (Rosenfalck potential, tripole approximation). Changes in depth for the location of the muscle fibres are easy to make. It is possible to let the action potential travel along the muscle fibre. In that way the source becomes a dynamic source. Motor units (MUs) can be simulated by variations in activation time and variation in tendon positions of the muscle fibres belonging to the MU.
3.1.2. Volume conductor properties

The properties of the volume conductor are determined by the length of the cylindrical volume conductor, the radii of the tissue layers in the volume conductor, and the conductivities of the layers in axial and radial direction. These are the parameters in this panel which can be modified.

3.1.3. Calculation of potentials

In this section the final calculations are done by solving the equations in chapter 2. Calculations can be saved and the observation points, the sites where the electrodes are virtually placed can be changed in angular and axial direction. It is possible to calculate surface potentials for different kind of conductivities for the skin layer, fat layer or muscle layer and visualise them immediately. In case of moving intracellular potentials, visualisations of MUAPs versus time at different axial and angular positions are possible.

Finally, (own-written) applications in Matlab for Windows ® allows the user to manipulate excellently with these matrices of potentials. Mesh grids can for example be created which are visualisations of potentials against angular direction (fig. 3.2, remember, the potentials are symmetrical around \( \pi \)), § 2.1.4) or time (MU with muscle fibres which vary somewhat in activation time and tendon positions, fig. 3.3). From the latter figure the potentials for \( z = 9, 11 \), and 13 are enlarged in fig. 3.4. Besides the positive potential change for \( t = 5 \) (\( z = 13 \)), \( t = 8 \) (\( z = 11 \)) and \( t = 11 \) (\( z = 9 \)), an end-effect can be noticed for \( t > 45 \). The extinction at the (proximal) tendon does have an influence on the progress of the simulated surface MUAPs between the motor endplate and the tendon positions.
Fig. 3.2. Mesh grid from a potential at the surface of the skin in axial and angular direction.

Fig. 3.3. Mesh from potentials on the surface of the cylindrical volume conductor, due to MU, parallel with the axial axis of the cylinder against time.

Fig. 3.4. Potentials for \( z=9,11,13 \) against time, enlarged from fig. 3.3.
3.2. Calibration of the model

The formulas in § 2.1 and § 2.2 are incorporated in the computer model. To test the model on its integrity, limits of the solution could be checked. When a source is located close to the centre of the cylinder (distance $r_1$) and the observation points are positioned with radius $r_2$ far away from the centre of the cylinder ($r_2 \gg r_1$) the potential in the observation points are mainly determined by the inhomogeneous solution. When, however, the source is located close under the surface of the cylinder the infinite and finite solution should be equal. This is due to the so-called mirror effect, known in electrostatics (when a charge is placed in front of a large flat conductor, the potential field is formed by the charge and that of a virtual charge at the same distance from but at the other side of the flat conductor). These limits of the solution where indeed present.

Checking on amplitude and signal content does however not happen with these limit situations. Another calibration of the model is possible when the output from a simulation can be predicted in shape and amplitude according to a certain input. This can be done by configuring the volume conductor as a muscle fibre and inputting a current strength $I(z)$ into the model, calculated from this active muscle fibre. Thus, from an intracellular potential of a muscle fibre in fig. 3.5.a we determine the current strength ($A/m$) according to (2.3.3)

$$I(z) = \pi a^2 \sigma_{\text{intra}} \frac{d^2 \Phi_i(z)}{dz^2}.$$  \hspace{1cm} (3.2.1)

(radius of the muscle fibre $a$, the intracellular conductivity $\sigma_{\text{intra}}$ and intracellular potential $\Phi_i(z)$).

If we place $I(z)$ on the centre axis of the cylinder and the volume conductor is configured according to a muscle fibre (layers have intracellular conductivity, 0.55 $\Omega^{-1} m^{-1}$ and the radius of the volume conductor equals $a$) the potential on the surface of the volume conductor should be the same in amplitude and shape when the volume conductor simulation model is functioning well. Fig. 3.5.b shows the outcome of the simulation on the surface of the cylinder. The shape and the width of the signal (8 mm) are conserved. The amplitude of the potential in fig. 3.5.a differences maximal 131 mV, about the same as in fig. 3.5.b, 133 mV. The difference in resting values of the potential ($V_{rm} = 0$ mV and $V_{rp} = -90$ mV) is caused by the fact that in fig. 3.5.a, an intracellular potential, and fig. 3.5.b, an extracellular potential, the references are both positioned extracellularly (-90 mV is caused by $R_{mp}$, § 1.3). The model is thereby calibrated and further simulations are possible.
3.3. Limits of the analytical solution and an extra bone layer

It was explained in § 2.2 that for calculations of the solution with a computer, the terms of the total solution have to be discrete. The terms \( A_n(k), C_n(k), D_n(k), E_n(k), F_n(k), G_n(k), \) and \( H_n(k) \) are thus determined for each combination of \( n \) and \( k \) with the Bessel functions in the matrix and the second vector. For increasing values of \( n \) (up to 64) and \( k \) (up to 1024 m\(^{-1}\)) the terms in the 5*5 matrix (§ 2.1), in case of a three layer volume conductor model, become very ill conditioned due to the behaviour of the \( I \) and \( K \) functions. The factors differ tremendously (10\(^{25}\)) and the condition of the matrix will exceed the limiting value based on the machine accuracy. It is thus necessary to condition the linear set of equations. This can be achieved by rewriting the matrix. The terms in the second vector, \( A_n(k), C_n(k), D_n(k), E_n(k), F_n(k), G_n(k), \) and \( H_n(k) \) are multiplied by the corresponding \( I \) and \( K \) Bessel functions, \( I_n(\rho; k) \) and \( K_n(\rho; k) \) while the terms in the matrix are divided by the terms. As a result of this multiplication, combinations like \( I_n(a)/I_n(\rho) \) and \( A_n(k) * I_n(\rho; k) \) do form. Mathematically the problem increases, however these terms are well-dimensioned as long as the fraction \( a/\rho \) is in a region around 1 for higher orders of \( n \), and when \( |m-n| \) is not too large [Goo90]. Changing the terms in the matrix by this scaling will thus improve the condition of the matrix.

When an extra layer is introduced in the model, the matrix increases from 5*5 to 7*7 terms. This fourth layer, representing bone tissue (fig. 2.2), would be the inner layer with a low conductivity [Gra96], 0.005 \( \Omega \cdot m^{-1} \). This bone layer has a reflective character on the current flow in the volume conductor and the potential distribution around the cylinder. A procedure was written to solve the seven linear equations with seven unknown variables. However, due to a doubling of the terms in the matrix and the position of the fibre, which is located now in the second layer, it had an impact on the possibility to solve the problem. This happens especially for higher spatial frequencies \( k \) (2000 m\(^{-1}\)) and when the radii of the bone and the muscle layer differ to much. Another (complicated) scaling will be necessary to suppress this accuracy problem. Future developments will make this scaling however superfluous because other prosperous solution methods, finite element methods [Spa97] or finite difference methods do not use Bessel functions. An indication (the solution is in this case stable for \( k < 1500 \) m\(^{-1}\)) what an extra bone layer does to the total solution is demonstrated in fig. 3.6 for an active fibre at 5.5 mm depth. The configuration for both cylinders is found in table 3.1.

---

Fig. 3.6.a. Potential distribution for cylinder I (no bone). b. Potential distribution for cylinder II (with bone).

Chapter 3. Implementation in a user interface model
Table 3.1. Configuration for cylinder I and II.

<table>
<thead>
<tr>
<th>Cylinder I</th>
<th>Radius (m)</th>
<th>Conductivity (in Ω m⁻¹)</th>
<th>Cylinder II</th>
<th>Radius (m)</th>
<th>Conductivity (in Ω m⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>axial, radial</td>
<td></td>
<td></td>
<td>axial, radial</td>
</tr>
<tr>
<td>muscle</td>
<td>0.0280</td>
<td>0.50, 0.10</td>
<td>bone</td>
<td>0.0280</td>
<td>0.005, 0.005</td>
</tr>
<tr>
<td>muscle</td>
<td>0.0325</td>
<td>0.50, 0.10</td>
<td>muscle</td>
<td>0.0325</td>
<td>0.50, 0.10</td>
</tr>
<tr>
<td>fat</td>
<td>0.0340</td>
<td>0.05, 0.05</td>
<td>fat</td>
<td>0.0340</td>
<td>0.05, 0.05</td>
</tr>
<tr>
<td>skin</td>
<td>0.0360</td>
<td>0.75, 0.75</td>
<td>skin</td>
<td>0.0360</td>
<td>0.75, 0.75</td>
</tr>
</tbody>
</table>

In case of a bone layer, fig. 3.6.b, the amplitudes are higher, probably caused by the reflective character of the bone layer. Also, calculations do show that the potential in n-direction in fig. 3.6.b does not decay as fast as in fig. 3.6.a, this difference is however marginally present. The influence of the bone layer will moreover in most configurations not be really significant, because of a much smaller radius of the bone layer (1.5 cm or less) in anatomical parts of the human body.
Chapter 4

Skin conductivity and EMG signals
-modelling, theory and experimental set-up-

For a further use of the volume conduction model (chapter 2) and the interpretations of the outcome, it is very helpful to know the conductivity of the skin. So far there are no measurements done at the conductivity of the skin. Only Burger [Ged67] did some measurements in the past. Burger determined a conductivity for the skin between 0.25 and 0.50 $\Omega^{-1} m^{-1}$. However, this was an indirect result from another experiment and there was no correction made for underlying fat and muscle layers. The conductivity of the skin influences not only the shape of the signals at the skin, but also the amplitudes. We will see this in § 4.1. The theory behind the measurements of the conductivity of the human skin will be explained in § 4.2 and the experimental set-up of this measurement is treated in § 4.3. Finally, in § 4.4 the development of a self-made current stimulator is described, which enabled measurements with lower currents through the skin and no supply from the electricity grid.

4.1. Relationship between MUAPs and the conductivity of the human skin

The influence of the conductivity of the human skin on the surface MUAPs can be investigated with the volume conductor model, treated in chapter 2 and 3. This is done in this section by varying the value of the human skin conductivity between $0.01 \Omega^{-1} m^{-1}$ and $1.25 \Omega^{-1} m^{-1}$ in steps of a multiplying factor of 5.

We assume a cylindrical volume conductor with the specifications in table 4.1 (conductivities of fat and muscle tissue are known from literature [Gie86, Ged67]. The conductivity of the fat layer is relative small, $0.05 \Omega^{-1} m^{-1}$ while the conductivity of the muscle differs between axial direction, $0.50 \Omega^{-1} m^{-1}$ and radial direction, $0.10 \Omega^{-1} m^{-1}$). In this volume conductor a motor unit (MU) is situated at a depth of 6 mm. The MU is simulated by 300 muscle fibres. Each fibre has a 'realistic' current strength, simulated by a mathematically description of the intracellular potential (chapter 2). Fig. 4.1 represents a point of time of the muscle excitation. We will take this arbitrary situation to illustrate the dependence of surface EMG signals on the conductivity of the human skin.

In fig. 4.2, the simulated MUAPs at the upper side of the cylindrical volume conductor are shown. We see a clear change from a tri-phasic (fig. 4.2.a, radial skin, axial skin equals $0.01 \Omega^{-1} m^{-1}$) to a more-like mono-phasic signal (fig. 4.2.d, radial skin, axial skin equals $1.25 \Omega^{-1} m^{-1}$). We can also notice a difference in the maxima of the MUAPs (in $\mu V$). The differences between the signals for the several skin conductivities look at first sight intuitively correct, although, they need an explanation when trying to understand the differences of the MUAPs.

<table>
<thead>
<tr>
<th>Layer cylindrical volume conductor</th>
<th>Radius (in cm)</th>
<th>Conductivity, Axial direction (in $\Omega^{-1} m^{-1}$)</th>
<th>Conductivity, Radial direction (in $\Omega^{-1} m^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle layer</td>
<td>3.2 cm</td>
<td>0.50</td>
<td>0.10</td>
</tr>
<tr>
<td>Fat layer</td>
<td>0.1 cm</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>Skin layer</td>
<td>0.2 cm</td>
<td>0.01, 0.05, 0.25, 1.25</td>
<td>0.01, 0.05, 0.25, 1.25</td>
</tr>
</tbody>
</table>

Chapter 4. Skin conductivity and EMG signals
Fig. 4.1. Global representation of the simulation set-up.

Fig. 4.2. Calculated SEMG potentials for different conductivities of the skin layer (for observation line, see fig. 4.1).

Chapter 4. Skin conductivity and EMG signals
The MUAP we derive with the cylindrical volume conductor model at the surface does exist because of a certain current flow through the volume conductor, induced by the ‘simulated’ MU. The current flow in the skin layer of course depends on the conductivity of that layer. High conductivity means a low resistance layer while a low conductivity indicates a high resistance layer.

If the current now passes through the fat layer it enters the skin layer. Depending on the conductivity of the skin the current prefers to flow through the skin or fat tissue. If the conductivity of the skin is lower than the conductivity of the fat layer, current will not be spread over the surface of the skin and flow through the fat tissue (and muscle tissue). Only at those places where the current has passed the fat layer, which is merely nearby the places where the current strength is relatively large (emphasized by the minus and plusses in fig. 4.3), the current will enter the surface of the skin, causing the steep peaks in fig. 4.2. In case of a high skin conductivity the current flow is more spread over the skin surface. That causes lower surface EMG potentials but also a more spread potential distribution. The situations for the different skin conductivities are roughly illustrated in fig. 4.3. Remember, fig. 4.3 is an indication, complete calculated current flows for the whole volume conductor would give a better picture.

By these simulations it is made clear that surface EMG signals depend strongly on the conductivity of the skin. This is visible not only in amplitude but also in shape. The simulations can be combined with real measurements on a MUAP of a motor unit. The amplitude of these signals can indicate a value for the conductivity of the skin with the simulations in fig. 4.2. However, we have to assume then that the other parameters of the system are correct, precious and sufficient to describe the volume conduction. Therefore, measurements are necessary to determine this skin conductivity.

![Current flow lines](image)

Fig. 4.3. Schematic current flow for different conductivities of the skin. a. $\sigma_{\text{skin}} << 0.10 \text{ Ohm}^{-1} \text{ m}^{-1}$, b. $\sigma_{\text{skin}} >> 0.10 \text{ Ohm}^{-1} \text{ m}^{-1}$.

### 4.2. Theory behind the measurement of skin conductivity

In the following section, field solutions and measurement equations are derived for determining the electrical conductivities of isotropic conducting media. The outcome, expressed in formulas, can be applied to the measurement of the conductivities of the skin tissue. The formulas are reported in § 4.2.1. The basis for the field solutions in § 4.2.1 is an infinite, homogeneous, isotropic conducting medium. Corrections for the finiteness of skin thickness are made in § 4.2.2. The theory in § 4.2.1 and § 4.2.2 is applied in § 4.2.3 to obtain the formula for the measurement of the human skin conductivity.
4.2.1. The Poisson equation and an isotropic infinite conductor

For isotropic media the solution of the electrostatic equation of continuity can be obtained. A similar problem, but then specialised on muscles, has been treated by S. Rush [Rus62, Smy50].

The basis of many electrostatical problems is the electrostatic equation of continuity, the Poisson equation, which can be written in rectangular co-ordinates as

\[ -\nabla \cdot J = \sigma_x \frac{\partial^2 V}{\partial x^2} + \sigma_y \frac{\partial^2 V}{\partial y^2} + \sigma_z \frac{\partial^2 V}{\partial z^2} \quad (4.2.1) \]

in which \( J \) (a vector) is the current density \( (\text{A/m}^2) \), \( V \) the potential in the media and \( \sigma_x, \sigma_y, \) and \( \sigma_z \) the conductivities in the \( x, y \) respectively \( z \)-direction. The co-ordinate relations are drawn in fig. 4.4.

\[ V = \frac{I}{4\pi \sigma \rho} \quad (4.2.2) \]

\( \rho \) is the distance to the origin. For a semi-infinite medium, the conductivity is zero for the area above the \( x, y \) plane (usually consisting of air). In the latter case, the potential becomes

\[ V = \frac{I}{2\pi \sigma \rho} \quad (4.2.3) \]
4.2.2. Finite thickness of the human skin

It may happen that the isotropic material of interest, fig. 4.5, has a limited extension in the negative and positive z-direction, $z < -h$ and $z > 0$; the shape being referred to as a 'slab'. When slab thickness, $h$, is less than twice the electrode spacing $a_{ed}$, the effects of the boundaries make (4.2.2) and (4.2.3) weak approximations. The electrode spacing is the distance between the electrodes in the electrode arrangement in fig. 4.5. This arrangement will be used in chapter 5 to determine the skin conductivity (see also § 4.2.3). The electrode spacing $a_{ed}$ equals 6 mm in the experimental set-up (§ 4.3), the human skin thickness is at most places certainly thinner then 12 mm, so equation (4.2.2) needs to be modified.

Consider the actual geometry in fig. 4.5, a subdivision of space into three plane-parallel sections, each of these being homogeneous. $\sigma_c$, is the conductivity of the layer above the material of interest, $z > 0$, $\sigma_0$, the conductivity of the material of interest, $-h < z < 0$, and $\sigma_s$ the conductivity of the layer under the material of interest, $z < -h$.

The potential distribution in the three divisions of space can be worked out with the principle of electrical images. This principle deals with the discontinuities in conductivity at both sides of the boundaries and is known from classical electrostatics [Pan62, Oos78].

This principle is applied on the problem. The potential inside the material of interest due to the source, to the first order left- and right image and all subsequent images can be expressed as

$$ V(x) = V_0(x) + \sum_{n=1}^{\infty} \{L_n(x) + R_n(x)\} $$

(4.2.4.a)
with \( V_\omega(x) \) as \( (4.2.2) \)

\( x \) a point in space with co-ordinates \( x, y, z \)

\( L_k(x) \) image in the layer \((\sigma_a)\) below the material of interest =

\[
= \left\{ \frac{\kappa_1 - 1}{\kappa_1 + 1} \right\}^n \left\{ \frac{\kappa_2 - 1}{\kappa_2 + 1} \right\}^n V_\omega(x - n_h) \quad \text{(4.2.4.b)}
\]

\( R_k(x) \) image in the layer \((\sigma_c)\) above material of interest =

\[
= \left\{ \frac{\kappa_1 - 1}{\kappa_1 + 1} \right\}^n \left\{ \frac{\kappa_2 - 1}{\kappa_2 + 1} \right\}^n V_\omega(x + n_h) \quad \text{(4.2.4.c)}
\]

\( n = 1, 2, 3, \ldots \), the number of electrical images

\( n_1 = \text{entire } (n/2), \)

\( n_2 = \text{entire } (n+1)/2, \)

\( n_3 = 2n_1, \)

\( n_4 = 2n_2, \)

\( \kappa_1 = \sigma_a/\sigma_b; \quad \kappa_2 = \sigma_c/\sigma_b, \)

\( x - n_3 h = (x, y, z - n_3 h) \) and likewise \( x + n_4 h = (x, y, z + n_4 h) \).

The image is directed to below for odd values of \( n \) and to above for even values of \( n \).

When one knows the source and the conductivities of all the three layers, the potential distribution could be calculated. Reverse this is not valid, although one might measure the potential distribution one could not calculate the conductivity of the slab even if one knows the conductivities of the layer above the material of interest \((\sigma_c)\) and the layer below the material of interest \((\sigma_a)\). One has, by the summation over \( n \), to deal with \( n^{th} \) degree equations which are not mathematically solvable \((n \) has to be certainly larger than 2 to use principle of electrical images).

However, exact mathematical expressions for the conductivity of the slab can be derived when the boundaries of the slab are either a 'perfect' conductor or an insulator, that means that \( \sigma_a \) and \( \sigma_c \) are 0 or \( \infty \). Consider a situation with \( \sigma_a = 0 \) and \( \sigma_c = 0 \). \( \kappa_1 \) and \( \kappa_2 \) are consequently zero and disappear out of \( (4.2.4.b) \) and \( (4.2.4.c) \). We obtain then

\[
\sigma_b = \frac{I}{2\pi V} \left[ (x^1 + y^1)^{-1} + 2 \sum_{n=1}^{\infty} \frac{1}{(n+1)^2} [x^1 + y^1 + (2nh)^1]^{-1} \right] \quad \text{(4.2.5)}
\]

The situation in fig. 4.6.a is thus described with a system of images in fig. 4.6.b. The electrical images extend to infinity in the plus and minus \( z \)-directions, spaced a distance \( 2h \) apart. The images are equal in strength to the source plus its image in the \( z = 0 \) plane, \( 2I \), and are of the same sign when \( \sigma_a = 0 \) and \( \sigma_c = 0 \). The images create an artificial boundary in the shape of current \( 2I \). The extension in infinity is necessary to counteract each other. The potential on the surface can now be 'calculated' using the principle of superposition, that is superimposing the potential fields from \( n \) sources with strength \( 2I \). The resulting potential distribution at the slab surface \((z=0)\) of the medium can now be calculated with \( (4.2.5) \).
4.2.3. The measurement equation

The measurement of the conductivity of the material of interest (from now on, σ) with two layers of zero conductivity above and below, can be made by applying equation (4.2.5) to a simple electrode arrangement on the regarding piece of tissue, drawn in fig. 4.5. The electrodes consist of four, equally spaced, approximately spherical, conductors of small size. The dimensions of the electrodes must be such as to make (4.2.2) a good approximation at a distance \( r_{\text{max}} \) from the origin; and to eliminate for practical purposes, perturbations of the potential field by the remaining conductors, the source at position 1 and the sink at position 4. Electrodes at position 2 and 3 are connected with an amplifier (§ 4.3) and measure the potential difference, \( \Delta V_{23} \), between position 2 and 3. The potential difference due to the current source electrode at position 1 can be found from (4.2.3). Noting that the sink electrode at position 4 doubles this potential but moreover the finite thickness of the skin (§ 4.2.2), the potential difference between position 2 and 3, measured on this isotropic piece of tissue, equals

\[ \Delta V_{23} = \frac{I}{2\pi a_{\text{wd}}} \sigma \left[ 1 + 2 \sum_{n=1}^{\infty} \left( 2 \left( \frac{1 + (2nh/a_{\text{wd}})^2}{1 + (nh/a_{\text{wd}})^2} \right)^{1/2} - 1 \right) \right] \]  

for \( \phi = 0...\pi/2 \).

Fig. 4.8. Value between bracket terms in (4.2.6) vs. skin thickness.

Chapter 4. Skin conductivity and EMG signals
For the terms between the brackets in (4.2.6), the values of $a_{ied}$ (electrode spacing) and $h$ (skin thickness) are of great importance. That is illustrated in fig. 4.8 for experimental parameters (§ 4.3); $a_{ied}$ equals 6 mm and the skin thickness varies between 2 and 5 mm. The converging series are summed until $n = 500$ (for increasing values of $n$, the value between the bracket terms changes barely, a factor $10^{10}$).

The terms between the brackets will eventually converge to 1 if $h$ extends to infinity and (4.2.3) remains ($z=0$). The conductivity of the skin, $\sigma$ in (4.2.6) can thus conveniently be found from the measurement of $\Delta V_{23}$, a known current $I$, the electrode spacing $a_{ied}$ and the skin thickness $h$.

4.3. Experimental set-up of the skin conductivity measurements

The best way to illustrate the experimental set-up is a description of the measurement equipment according to fig. 4.9 and fig. 4.10 below.

An important part of the set-up is the electrode 'grid'. The grid consists of 63 electrodes, placed into perspex holders which are connected to each other with plastic fibres. The configuration of the electrodes is shown in fig. 4.10 (drawn according to real dimensions). The electrodes - diameter of 1 mm, gold-coated, cylindrical shaped with a robust round shape at the downside - are placed on the skin and measure the surface EMG potentials on the positions where they are settled. The potentials measured on the electrode grid are sampled with 2000 or 4000 Hz and are then amplified with a 64 channels amplifier. The reference (REF), the common sense, (CMS) and the driven right leg (DRL) are placed on the human skin at certain positions. The CMS is responsible for a stable potential of the human skin and works together with the DRL. This DRL is an output to counteract signal disturbances (potential changes) of the human skin (read CMS) which occur during measurements. The REF is subtracted from the potentials measured at the electrodes. The electronical configuration of the amplifier, concerning the DRL, CMS, REF and the channels 2,3...64 is schematically drawn in Appendix B. The amplifier has a bandwidth from 3 to 800 Hz and is developed by Vision Research®.

The current $I$ is provided with a physiological current stimulator which is able to produce a output current between 5 and 300 $\mu$A. The maximum impedance for the current stimulator is 250 $k\Omega$. The current stimulator gets its input from a function generator so the current stimulator is able to produce several signal forms in shape and frequency.

![Diagram of experimental set-up](Fig. 4.9. Schematic presentation of the experimental set-up.)
The signals are then transported to the buffer with an optical glass fibre. The buffer collects the signals and sends them per channel to the data acquisition card of the computer. The signals are then displayed on the screen. The signals can be saved and undergo a further operation if necessary. The data acquisition program is written in CVI/LabWindows® and further calculations (chapter 5) are done with Matlab for Windows®.

4.4. A second current stimulator

The experimental set-up in fig. 4.9 (§ 4.3) exists among other things of a current stimulator, fed by the function generator. This current stimulator was used during experiments we will describe in chapter 5 (§ 5.2). To insert a lower current into the human skin another ‘current’ stimulator has been developed which enables lower currents (< 1 μA) and has no supply from the electricity grid. The stimulator, drawn in fig. 4.11, delivers not a completely stable current because the impedance from the electrode to the skin, $z_e$, does change between measurements.

The stimulator is fed by a battery of 9 Volt at the $310XB$. This is a divider which splits the 9 V from the battery into 0 V, a virtual ground and +9 V. The $TLC555$ is a timer. It takes care of the frequency of the signal, determined by the values of the resistances and the capacitor. In this situation these are $10 \text{ kΩ}$, $0.1 \mu F$ and $2.2 \text{ MΩ}$ which induces an frequency of 34.19 Hz (and a equal duty cycle). The signal, originating from the $TLC555$ has a formation of a bloc.

The $CD4053 BE$ combines now the input from the $310 XB$ and the $TLC555$ (signal with a formation of a bloc) in two opposite block signals with an amplitude of 4.5 V originating from 14 and 15 of the

Chapter 4. Skin conductivity and EMG signals
CD4053 BE. The potential difference between 1 (fig. 4.5) and 4 (fig. 4.5), and thus the current through the skin, depends now on the chosen R in fig. 4.11.

Evaluation of the electronic scheme learns that, when R equals 370 kΩ, the current through the skin varies between 0.5 and 1.1 µA. This depends on Z_e, the impedance of the skin-electrode transition with respect to the current source at position 1 and the current sink at position 4 (Z_e varies normally between 200 and 500 kΩ). The capacitors in the stimulator filter low frequencies and take care of sudden current shortages.

![Circuit Diagram]

*Fig. 4.11. Current stimulator developed for low currents (< 1 µA) and a battery supply.*
Chapter 5

The conductivity of the human skin

-measurements-

With the experimental set-up and the theory in chapter 4 we are able to estimate the conductivity of the human skin. In § 5.1 the experimental set-up in chapter 4 is calibrated. In § 5.2 the results of the skin conductivity measurements are shown. Changes in the experimental parameters are made in § 5.3 (current) and § 5.4 (temperature) and the consequences of these changes on the measured skin conductivities are presented in the accompanying sections. Finally, in § 5.5, the measurements in the previous sections are discussed.

5.1. Calibration of the experimental set-up

The experimental set-up in chapter 4 needs to be calibrated. This calibration consists of three parts,

- Investigation of the synchrony of the channels from the amplifier,
- Investigation of the amplitude distribution of the signals, and
- A comparison between measured and calculated conductivities with literature values of a known solution.

The first and second part of the calibration were done with a 10 mV sinusoidal signal, generated with the function generator. This signal was directly connected with the 63 channels in the amplifier (not with the electrode grid). The REF, DRL and CMS were connected to the earth of the function generator. The signals we eventually would receive in the computer should have the same phase and amplitude as induced by the stimulus. Besides, during the calibration of the amplitudes of the signals one could also look at the decay at the boundaries of the bandwidth of the amplifier (3-800 Hz). For this purpose the frequency of the sinusoidal signal was simply changed.

5.1.1. Phases

The phases of the measured signals (at the 63 channel electrodes) are quite similar with a standard deviation of 0.5° when varying the frequency in a range between 10 to 500 Hz. In fig. 5.1, the phases of the measured signals are plotted against the channel numbers for a 100 Hz sinusoidal signal (phase-axis between 20 and 30°). The average phase in fig. 5.1 differs between every measurement, it depends namely on the phase of the sinusoidal signal when the measurement starts respectively ends.
5.1.2. Amplitude

For lower frequencies (3-100 Hz) a decay of the signals in amplitude can be noticed when we apply the same input from the function generator (10 mV sinusoidal signal) on the 63 channels. In fig. 5.2, the results of the amplitude calibration are worked out. The dotted line represents the target value (10 mV). The bandwidth of the amplifier is thus clearly present. The circles at each frequency show the standard deviation of the measured values for the channels at that frequency. We see a decay of 3.2 dB from 10 to 3 Hz (specification is 3 dB) and there is still a small decay of about 0.5 dB between 10 and 100 Hz.
5.1.3. Conductivity of a known aqueous solution

The third calibration step was a comparison between measured conductivities of an aqueous solution with the experimental set-up and known literature values. A piece of oasis material drenched in a salt water solution with a concentration of approximately 0.08 M NaCl was used as calibration solution. This solution is purely resistive and homogeneous and after correction for the above named calibration factors, it should therefore give a constant conductivity for all frequencies. The measured signals showed after calculation with (4.2.4) (correction for finite thickness was not necessary) conductivity around $0.7 \text{ Ohm}^{-1}\text{m}^{-1}$ for frequencies between 10 and 200 Hz. That conductivity is reasonably close to a literature value for a salt water solution with a concentration of 0.08 M NaCl [Gab96], $0.8 \text{ Ohm}^{-1}\text{m}^{-1}$.

5.2. Results of skin conductivity measurements

Like already mentioned in § 4.2.2 and § 4.2.3 we have to measure at a spot where the tissue layer under the skin layer has a conductivity which is expect to be much smaller than that of the skin. The ideal situation is of course a layer with zero conductivity. This is however not feasible on an anatomical part of the human body, but if the layers fulfil the relationship ($\sigma_{\text{layer}} << < \sigma_{\text{skin}}$) (4.2.6) is a good approximation of the potential difference. If the layers do not, we won't be able to use (4.2.6). Modifications of equation (4.2.4) by the changed anatomy are in that case giving us equations of $n^{th}$ degree (4.2.5) which are not solvable mathematically (§ 4.2.2).

Parts of the body with a bone layer close to the skin surface fulfil the relationship ($\sigma_{\text{layer}} << < \sigma_{\text{skin}}$) very sufficient. The conductivity of bone tissue is basically lying between 0.005 and 0.01 ($\Omega\text{m}^{-1}$) [Gab96]. The skin over the head at some places and also the skin over the tibia ('shine bone') are good sites. Furthermore, the smaller the subcutaneous fat layer between skin and bone, the better is (4.2.6) an approximation of the conductivity of the skin. In this thesis measurements are focused on the skin above the tibia. A cross-section in the lower limb midway the tibia shows us the anatomy in that area for a person with a relatively large amount of fat tissue (fig. 5.3).

---

Fig. 5.3. Cross-section of a human body midway the lower leg. The tibia circumference is indicated by the white line.
The skin layer above midway the tibia has a thickness between 2 and 3 mm in most subjects. There is also a small fat layer with a thickness between 0.5 (male) and 1.0 mm (female). With the help of echography and/or a sliding calliper we could determine the thickness of the layer above the tibia.

The electrode grid is thus placed on the skin above the tibia (fig. 5.4, fig. 5.5). Only hair was removed from the measurement area, no other preparation took place (in clinical surface EMG, the skin is mostly rubbed with gel to remove dead skin cells and sometimes cleaned with alcohol, this to reduce the impedance between the electrodes and the skin). The electrodes 34 and 37 in fig. 4.10 were connected with the current stimulator and are thus consequently not connected with the amplifier. Electrode 35 and 36 were used to measure the potentials $V_2$ and $V_3$ (see fig. 4.5). The grid was positioned vertically (distal, proximal) on the tibia. The CMS and the REF are placed on the patella (knee cap), the DRL on the ankle (fig 5.5).
The width of the tibia directly under the skin varied between 3.5 and 4.5 cm, a factor 6 to 8 times larger than the electrode spacing $a_{\text{ped}}$, 0.6 cm. The measurements in this chapter were done at 7 different healthy subjects (5 males, 2 females). In Table 5.1 some characteristics of the subjects are summarised.

The current through the skin was a sinusoidal signal with a strength of 25 $\mu$A at the frequencies 5, 10, 20, 50, 100, and 200 Hz for each subject. The current was supplied by the current stimulator in fig. 4.9 with the input from the function generator.

A measurement result is shown in fig. 5.6 below. The current during this measurement was sinusoidal and had a frequency of 20 Hz. We are, consequently, able to see this 20 Hz component in the Fourier transformation of the potential signals ($10000 \pm 5 \text{ mV}$) from channel 35 and channel 36 (fig. 5.6.a and b). In fig. 5.6.c the potential signals at channel 35 and channel 36 are plotted against time ($2000 \pm 1 \text{ sec}$). We can also notice a 50 Hz component - and higher harmonics - in the Fourier transformed potential signals from channel 35 and channel 36. This is a problem of the current stimulator (it is not that perfectly isolated from the electricity grid as it should be).

**Table 5.1. Some characteristics of the healthy subjects.**

<table>
<thead>
<tr>
<th>Subject name</th>
<th>Age</th>
<th>Temperature skin in °C</th>
<th>Thickness skin in mm</th>
<th>Subject name</th>
<th>Age</th>
<th>Temperature skin in °C</th>
<th>Thickness skin in mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>I m</td>
<td>23</td>
<td>31 ± 1</td>
<td>2.5 ± 0.5</td>
<td>V f</td>
<td>35</td>
<td>29 ± 1</td>
<td>2.5 ± 0.5</td>
</tr>
<tr>
<td>II m</td>
<td>23</td>
<td>31</td>
<td>2.5</td>
<td>VI m</td>
<td>24</td>
<td>28</td>
<td>3.0</td>
</tr>
<tr>
<td>III m</td>
<td>51</td>
<td>31</td>
<td>2.0</td>
<td>VII f</td>
<td>25</td>
<td>31</td>
<td>2.5</td>
</tr>
<tr>
<td>IV m</td>
<td>27</td>
<td>30</td>
<td>2.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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However, the 50 Hz component disappears when the signal at channel 35 is subtracted with the potential signal at channel 36, fig. 5.6.d. In fig. 5.6.e, the phases of the Fourier terms for 20 and 50 Hz are shown for three electrodes pairs: 43 and 44, 4 and 5, and 35 and 36 (see fig. 4.10). Electrodes 43, 4, and 5 lay close to electrode 34 while 44, 5, and 36 lay close to electrode 37. The phases of 4, 35, and 43 are mutual almost the same just like the phases of 5, 36, and 44. The phases between the ‘upper group (4,35,43)’ and the ‘down group (5,36,44)’ differ 180° as you would expect (the virtual ground of the skin is positioned between electrode 33 and 36). Fig. 5.6.f shows the Fourier transformed from fig. 5.6.d. The value corresponding to the 20 Hz component in fig. 5.6.f is the value representing $\Delta V_{23}$ in (4.2.6) (one could also take the root mean square (RMS) value out of fig. 5.6.d).

The total result of the measurements at the conductivity of the skin is displayed in fig. 5.7 (subject I-VI). The six histograms per subject illustrate the conductivities for the frequencies 5, 10, 20, 50, 100, and 200 Hz. The measured conductivity varies between 0.83 and 1.10 $\Omega^{-1}m^{-1}$ and there is no significant evidence for a relationship between the frequency of the current and the skin conductivity in the range between 5 and 200 Hz. The temperatures of the skin during the measurements at the subjects are also shown in fig. 5.7 (see for temperature relationship, § 5.4).

![Conductivity of the human skin against frequency in vertical and horizontal direction.](image)

Another measurement at the conductivity of the skin in 'horizontal direction' was done by using the electrodes in horizontal direction (from medial to lateral direction). The electrodes 11 and 42 (see fig. 4.10) were used as point sources and electrodes 3 and 34 measured the potential $V_2$ and $V_3$ (fig. 5.5). The conductivities were consequently 10 to 15% lower (see Appendix A.3 for formulas in case of a different conductivity between the horizontal and vertical direction). In fig. 5.7 the results are shown for IIh (horizontal), IIIh and Vh. The measurement situations, however are not the same. During the measurement of the conductivity from medial to lateral direction the source electrodes are more positioned along the sides of the tibia.

Another set-up was tried to reconfirm this statement. The electrode grid was placed over the calf. Directly under the skin there is no bone tissue, but basically isotropic fat tissue. A few measurements were done on that position, horizontally and vertically. Vertically on the calf showed somewhat higher conductivity (0 to 5 %) than in horizontal direction, not enough however, to speak about a significant difference between the conductivity in horizontal and vertical direction.

Chapter 5. The conductivity of the human skin
5.3. Relation between skin conductivity and current flow through the skin

The measurements in § 5.2 were done with a current of 25 μA. During EMG experiments the current flow through the skin is, however, considerably lower. Measurements with a lower current are thus necessary to find out if there is a relationship between current and skin conductivity in a range of 0.5 up to 25 μA.

To determine a possible relationship between the skin conductivity and the current through the skin layer, the current 'stimulator' in § 4.4 (fig. 4.11) was used. With this stimulator it is possible to lower the current through the skin to a value of 500 nA, with the formation of bloc. Lower currents than 500 nA (already a factor 50 smaller than in § 5.2) can be produced too with this stimulator, however, the potential changes in the skin layer would be to small to amplify and the signals disappear in the noise of the amplifier (50 μV).

Measurements were done on subjects I, II, IV and VI. A result of a measurement is shown in fig. 5.8 below.

There are several differences between fig. 5.6 and fig. 5.8. The current in fig. 5.8 has the formation of a bloc. Thus the Fourier transformed of this block signal is composed of frequency $f_{\text{basic}}$ (34.9 Hz) with amplitude $A_0$, frequency $3f_{\text{basic}}$ with amplitude $1/3A_0$, frequency $5f_{\text{basic}}$ with amplitude $1/5A_0$ and so on. This can be noticed in fig. 5.8.a, 5.8.b and fig. 5.8.f.
Another difference appears between fig. 5.6.d and fig. 5.8.d. Not only is the signal in fig. 5.8.d a block signal, it also decreases when the potential has switched over while it should be constant. This is caused by a so-called double layer which occurs between the current electrodes (34 and 37) and the skin. This double layer between skin and electrode (Boe78) is built up automatically and hampers the penetration of the current into the skin. This double layer - an infinitesimally thin interface between the electrode and the tissue due to complicated biochemical reactions (Gre81) - is electrically represented by a parallel combination of a resistance and a capacitor. When the block signal switches, the double layer (at least the capacitor component) is practically transparent for this high frequency behaviour.

Before the block signal switches reverse, the constant potential difference at electrode 34 and 37 has a DC behaviour. The impedance of the double layer increases because of the capacitor, the current through the skin decreases and thus, also the potential difference between channel 35 and channel 36.

The value of $\Delta V_{23}$ is determined by calculating the RMS value of fig. 5.8.d. Taking the potential value of the frequency component 34.9 Hz in fig. 5.8.f would in this case be inaccurate.

The measured conductivities of the subjects I, II, IV, VI are shown in fig. 5.9. On the y-axis stand the conductivities for the self-developed current stimulator while on the x-axis the skin conductivities from fig. 5.7 are placed. The temperatures of the skin were practically the same in both situations. The uncertainty margins in fig. 5.9 represent the variations in skin conductivity during recurring measurements under the same conditions.

![Fig. 5.9. Skin conductivities during a current of 25 $\mu$A through the skin against skin conductivities with a current between 0.5 and 1.1 $\mu$A.](image)

From the measurements in fig. 5.9, it seems that a slightly lower conductivity belongs to a lower current flow through the skin. However, there is no indication for a really significant relationship between the current flow through the skin and skin conductivity in the range of 0.5 and 25 $\mu$A.
5.4. Relation between skin conductivity and temperature

Considering the results in fig. 5.7, it seems that a lower temperature consistently belongs to a lower conductivity. If the temperature of the skin really influences the conductivity of the skin - which is something one intuitively would expect - is investigated in this section.

Possible temperature relationships are determined by a three-step protocol. Firstly, the conductivity of the skin is measured at normal skin temperature, depending on subject, between 28 ± 1°C and 31 ± 1°C. After this, the temperature of the skin is lowered with a cold ‘ColdHot Pack’ to 17 ± 1°C. The ‘ColdHot Pack’ is removed from the skin and the skin conductivity is measured during a slow temperature increment from 17 ± 1°C to 26 ± 1°C. This temperature increment has a natural cause. The final measurements at the conductivity of the skin are done after the temperature is raised up to 32 ± 1°C by means of a warm ‘ColdHot Pack’.

The temperature measurements are done in subjects I,II,IV,VI and VII. The current through the skin was 0.8 to 1.1 μA. Electrode 35, 36 (§ 5.2) are the measuring electrodes while electrodes 34 and 37 supply the current. The electrode grid is placed like in fig. 5.4 and fig. 5.5. The results of the measurements are shown in the underlying figure.

The measurements indicate a positive linear relationship between temperature and the skin conductivity \( \sigma_{\text{skin}} \). A linear fitting was done with the least squares method. This method calculated the slopes of the linear equation between temperature and skin conductivity. The average slope is approximately 0.015 ± 0.002 \( \Omega^{-1}\text{m}^{-1}\text{/°C} \) (see for individual slopes fig. 5.10). The relationship is thus linear in the temperature area of the measurements according to (5.4.1)

\[
\sigma_{\text{skin}} = \sigma_{0} + 0.015 \times (T - T_{0}) \quad \text{for } 18 \leq T \leq 33\text{°C}. \tag{5.4.1}
\]
5.5. Discussion

The simulation in § 4.1, chapter 4, showed a significant difference in surface potentials when using different kind of conductivities for the skin ($\sigma_{\text{pskin}} = \sigma_{\text{saxis}} = 0.01, 0.05, 0.25, 1.25 \ \Omega^{-1} m^{-1}$). When the conductivity of the skin is increased, the simulations show that the potentials are more spread over the surface (a smaller decay along the axial direction) and that the amplitude of the surface potentials is getting considerably lower when the conductivity of the skin is increasing (see fig. 4.2).

Measurements and simulations of [Roe97] showed that the conductivity of the skin layer should be considerably higher than the conductivity of the fat layer ($0.05 \ \Omega^{-1} m^{-1}$). Measurements of the conductivity of the skin in § 5.2 and § 5.3 support this. The measured conductivities vary from $0.83 \pm 0.30$ to $1.05 \pm 0.38 \ \Omega^{-1} m^{-1}$, and are not related to the frequency of the current in the range of 5 to 500 Hz.

The spread in the measured skin conductivity during recurring measurements (5 %) under the same conditions (temperature of the skin, position electrode grid, frequency signal) is far much lower than the uncertainty of the conductivities of the skin (40 %). In the latter uncertainty, four extra parameters do play a role:

<table>
<thead>
<tr>
<th>Table 5.2. Uncertainties of skin conductivity.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of error</td>
</tr>
<tr>
<td>The current through the skin ($Z_e$ changes)</td>
</tr>
<tr>
<td>The electrode spacing $a_{se}$ between the measuring electrodes (bulging of the skin, contact surface between electrode and skin).</td>
</tr>
<tr>
<td>The conductivity of the layer under the skin layer is not zero (§ 4.2)</td>
</tr>
<tr>
<td>The thickness of the skin layer</td>
</tr>
</tbody>
</table>

The conductivity we measured and calculated with (4.2.6) is determined by assuming that the conductivity of the skin layer is isotropic (§ 4.2). To confirm this assumption the electrodes were placed in a horizontal configuration. The measured conductivities in horizontal direction (lateral medial) were for subject II $0.80 \pm 0.26 \ \Omega^{-1} m^{-1}$, for subject III $0.82 \pm 0.25 \ \Omega^{-1} m^{-1}$ and for subject V $0.75 \pm 0.23 \ \Omega^{-1} m^{-1}$. These conductivities are lower than the conductivities in vertical (proximal distal) direction, indicating that the skin slightly differs concerning the conductivity between the x- and y-direction. However, the current flow during the horizontal conductivity measurement is more located on the sides of the tibia so that $10-15\%$ is an approximation of the situation. Measurements at the calf muscle do also not support a difference of conductivity between the horizontal and vertical direction.

A second anisotropy could be hidden in the conductivity of the skin layer between the axial direction and the radial direction of the volume conductor. The conductivity in the z-direction, from inside the body to the outside of the body, is however impossible to measure (with the present experimental setup). Later on in this discussion this subject will be brought up again.

The current we used during the conductivity measurements in y-direction in § 5.2 was approximately $25 \ \mu A$. Since the skin has a cell structure (§ 1.4) it is plausible that the conductivity of the skin is related with the current through the skin. Therefore a current stimulator was developed which could produce a current of $0.5-1.1 \ \mu A$ with a frequency of $34.9 \ Hz$, and has the formation of a bloc. This current approaches the current flow that exists during normal surface EMG which lays between 0.01 and $1 \ \mu A$ if we observe the actual maximal amplitudes of real SEMG potentials ($10-1000 \ \mu V$).

With a lower current, the conductivities were somewhat lower than the conductivities of § 5.2 (fig. 5.9), however, not enough to determine a significant relation between current and skin conductivity in the range of $0.5$ to $25 \ \mu A$. Lower currents were practically possible, because the potential signals at the electrodes would disappear in the noise of the amplifier ($\approx 50 \ \mu V$).
In § 5.2 one could notice that the skin conductivity is possibly related with the temperature of the skin. This relationship should be present if you think that blood vessels ‘shrink’ or ‘close’ during lower temperature and that skin is more open, producing sweat, when the body temperature is rising during exercise or warm weather. That should have, logically spoken, some effect on the conductivity of the skin. There is indeed a relationship, obtained out of measurements, (5.4.1), that implicates that the conductivity of the skin increases with \( 0.015 \pm 0.002 \ \Omega^{-1} \text{m}^{-1} \) per increment of skin temperature with \( 1 ^\circ \text{C} \). If we look back at the simulation in fig. 4.2 this would indicate that a lower skin temperature improves amplitude and shape of the signal. It also induces that the conductivities in § 5.2 will come more close to each other if they are compared for the same skin temperature. Still, a difference exists between the subjects. The obtained results are however, not enormously spread (maximum difference 25 %) in opposite to the subjects which varied in sex, age and skin colour (subject IV).

With the measurements in this chapter we obtained a value for the conductivity of the skin, \( 0.95 \pm 0.35 \ \Omega^{-1} \text{m}^{-1} \) (temperature \( 30 \pm 1 ^\circ \text{C} \)). Earlier remarks were made about measurements and simulations of Roeleveld [Roe97]. Roeleveld measured the relationship between the depth of a motor unit (in the upper arm, biceps branchii) and the maximal amplitude of the negative peak \( Nu \) (fig. 4.2) of the signal. For a MU at a depth of \( 5 \text{ mm} \) Roeleveld obtained fig. 5.11 within a volume conductor referring to § 4.1 and where in the skin conductivity equals \( 1.0 \ \Omega^{-1} \text{m}^{-1} \).

![Motor Unit measured/simulated](image)

**Fig. 5.11. Relation between measured and the modelled MUAP parameters.**

\( r \) is the radial observation distance which increases along the angular direction in fig. 5.11 (MU depth stays at 5 mm), III the three layer model, IIb a two layer model consisting of muscle tissue and fat tissue and IIa is a two layer model consisting of muscle tissue and skin tissue. A value of 1 for all values of \( r \) (dashed line) would prevail a perfect match between simulated and measured data. IIb is certainly an improper way of representing the volume conductor, IIa is considerably better (the high conductivity takes care of smaller decay of the signals along angular direction), but III shows the best results. This was an significant indication that the conductivity of the skin should be larger than the conductivity of the fat layer.

Fig. 5.11 does say something about the ratio of the measured and the simulated signals, however, the measured signals were scaled to the simulated signals. There was no comparison made between the absolute amplitudes of the measured signals and simulated signals. With the theory in § 2.2 and § 2.3 (the coupling of intracellular potentials in the model), it is possible to compare the amplitudes of the measured and simulated signals.
If simulations are made with a conductivity for the skin of 0.95 Ohm\(^{-1}\)m\(^{-1}\) and the motor unit depth \(d\) is varied between 3 and 30 mm we can calculate a relationship between \(N_u\) and \(d\) with the simulation model for a volume conductor with the specification of table 4.1. That relationship is drawn in fig. 5.12. This is compared with measurements of \([\text{Roe97}]\) at MUAPs at the surface of the skin above the biceps branchii.

![Diagram of Motor Unit with d and Qd-factor](image)

Fig. 5.12. Relationship between \(d\) and \(N_u\) for several MUs in the biceps branchii \((\text{[Roe97]})\) and the volume conductor model.

In fig. 5.12 a \(Q_d\)-factor is introduced, which represents the decay of the MUAP with increasing motor unit depth. The decay is represented in (5.5.1),

\[
N_u(i) = k_z \cdot \left( \frac{d(i)}{d_k} \right)^{-Q_d}.
\]  

(5.5.1)

\(Q_d\) describes for a specific position \(z\) along the surface of the volume conductor, how fast the MUAP , the negative peak \(N_u\) from the MUAP, decreases with increasing \(d\), the depth of a motor unit. \(k_z\) is a constant and equals the amplitude estimate of \(N_u\) at a distance where \(d_k\) is set to 1 mm. \(d(i)/d_k\) is the ratio between the depth of MU \(i\), \(d(i)\), and \(d_k\).

The \(Q_d\)-factor from the measurements (\(Q_{d\text{meas}}\)) equalled 1.85, the \(Q_d\)-factor of the volume conductor model (\(Q_{d\text{model}}\)) 2.00. The amplitudes also match remarkably well (to simulate the exact number of fibres is of course impossible). The \(Q_d\) of the model will probably further approach the \(Q_d\)-factor of the measurement when we assume a ‘reflective’ bone layer in the volume conductor (it reflects current back).

It was mentioned that the conductivity of the skin layer in the radial direction could not be measured and that is was assumed to be equal to the conductivity in axial direction and angular direction. The influence of this conductivity on the calculation of the conductivity of the skin layer with (4.2.6) is small because the skin extends more in \(x\) and \(y\) direction than in \(z\) direction. But referring back to the anatomy of the skin it is likely that the conductivity in radial direction is somewhat higher than in axial direction (‘sweating’ happens in radial direction) and certainly not smaller ; simulations with small radial conductivities of the skin will increase the \(Q_d\)-factor of the model, bringing it further away from the experiments.
Chapter 6

Conclusions and recommendations

6.1. Conclusions

The analytical solution has been derived for volume conduction in a four layer anisotropic finite cylindrical volume conductor (chapter 2).

Measured intracellular potentials represent the muscle fibre activity. By describing these measured intracellular potentials in terms of a current source strength, simulations of Motor Unit Action Potentials in 'real' \( \mu V \) can be obtained (chapter 2; § 2.3).

Volume conduction simulation possibilities are enhanced by means of a user interface of the Anvolcon program. Volume conductor properties, source position and orientation, dynamic sources, source strengths, and visualisation possibilities can be changed with simple clicks on the mouse buttons (chapter 3).

The Bessel terms and products used to solve (2.1.18) become extremely large in the case of an extra bone layer and a source in the second muscle layer (§ 3.3). Although theoretically possible, in practice the introduction of a fourth bone layer in the volume conductor model results in an ill-conditioned linear system which the computer cannot solve. Solution of this problem is thus impossible without any further scaling (chapter 3; § 3.3).

Simulations show that a high conductivity of the skin layer (\( \approx 0.10 \, \Omega^{-1} m^{-1} \)) causes smaller amplitudes and a more distributed potential distribution opposite to higher amplitudes and a less distributed potential in the case of a low conductivity of the skin layer (chapter 4).

Measurements showed that the conductivity of the skin equals 0.95 \( \pm 0.35 \, Ohm^{-1} m^{-1} \). There is an indication that the conductivity from lateral to medial direction ('horizontal') is lower (10-15 \%) than the conductivity from proximal to distal direction ('vertical'). The measuring situations did however slightly differ according to the underlying tissue layers and measurements at the calf muscle did not support the anisotropy in conductivity. The conductivity of the skin in radial direction could not be measured; however, simulations showed (fig. 5.11, fig. 5.12) that the conductivity in radial direction has to approach the conductivities in angular and axial direction (chapter 5).

There is no significant relationship between the current flow in the human body and the skin conductivity in a range of 0.5 to 25 \( \mu A \) (chapter 5).

Temperature and skin conductivity are related to each other. Lower temperatures imply lower conductivity. A lower temperature will thus improve the original signal content of muscle fibre activity, but unfortunately with a slow pace (conductivity decreases approximately with 0.015 \( Ohm^{-1} m^{-1} \) with a decline of temperature of 1°C) (chapter 5).
6.2. Recommendations

The exact, three layer analytic model described in chapter 2 may be compared with the previously developed finite element model of volume conduction [Spa97]. This model does not have an analytically obtained, exact solution, but allows non-symmetrical tissue configurations. The theory about the relation between the intracellular potential and the current strength (§ 2.3) could also be implemented in the finite element model.

Further measurements on subjects concerning the skin conductivity may eventually lead to a 'literature value' of the skin conductivity. Smaller current electrodes, a larger bandwidth of the amplifier, a better isolated current stimulator with the possibility to induce high and low current will decrease the uncertainty in the measured skin conductivities. However, the largest source of errors, the uncertainty in the skin thickness (table 5.2), is difficult to reduce since better skin thickness measurements are unfeasible unless incisions are allowed.
Bibliography


Bibliography


### Abbreviations

- $a_{ied}$: Interelectrode distance [m]
- $a$: Muscle fibre radius [m]
- $d$: Motor Unit Depth [m]
- $f_{basic}$: Basic frequency component [s]$^{-1}$
- $h$: Thickness of slab [m]
- $k$: Spatial frequency [m]$^{-1}$
- $n$: Angular frequency [rad]$^{-1}$
- $R_{mp}$: Rest membrane potential [V]
- $\sigma$: Conductivity [Ω$^{-1}$m$^{-1}$]
- $\sigma_{intra}$: Intracellular conductivity [Ω$^{-1}$m$^{-1}$]
- $\rho_s$: Radius source [m]
- $Q_d$: Decay of MUAP with increasing $d$ [m]$^{-1}$
- $\phi_s$: Angle source [rad]
- $\rho_{obs}$: Radius observation point [m]
- $\phi_{obs}$: Angle observation point [rad]
- $\Phi$: Potential [V]
- $\phi_i$: Intracellular potential [V]
- $I(z)$: Current strength [A][m]$^{-1}$
- $J(z)$: Current source density [A][m]$^{-2}$
- $i_v$: Volume current source density [A][m]$^{-3}$
- $a,b,c,d$: Radii volume conductor (§ 2.1.6) [m]

- medial: positioned towards the middle
- lateral: positioned towards the side
- proximal: closer to the anatomical centre
- distal: further away from the anatomical centre

- EMG: ElectroMyoGraphy
- MUAP: Motor Unit Action Potential
- SFAP: Single Fibre Action Potential
- MU: Motor Unit
- DRL: Driven Right Leg
- CMS: Common sense
- REF: Reference
- RMS: Root mean square
Appendix A. Derivation of formulas (§ 2.1, § 5.2).

A1. Fourier transformation of the Laplace equation in the z-direction

The continuous Fourier transformation in the z-direction is defined by (A.1.1)

\[ \Phi^*(\rho, \varphi, k) = \int_{-\infty}^{\infty} e^{-i2\pi k^*z} \Phi(\rho, \varphi, z) \, dz. \tag{A.1.1} \]

The repetency in the z-direction is in (A.1.1) represented by \( k^* \). In chapter 2, \( 2\pi k^* \) is replaced by \( k \). By using the Fourier transformation, we obtain a description of \( \Phi \) as a function of \( \rho, \varphi \) and \( k \). If \( \Phi \) is an even function, the exponential term in (A.1.1) can even be replaced by a \( \cos 2\pi k^*z \) term. \( \Phi^* \) has a dimension of \( \{m \cdot \text{dim}(\Phi) \}, \) if \( \Phi \) has 'undergone' a continuous Fourier transformation.

For a Fourier transformation of the second derivative of the potential \( \Phi \) in cylinder co-ordinates, we derive,

\[ F\left( \frac{1}{\rho} \frac{\partial}{\partial \rho} \left( \rho \frac{\partial \Phi}{\partial \rho} \right) \right) = \frac{1}{\rho} \frac{\partial}{\partial \rho} \left( \int_{-\infty}^{\infty} \Phi e^{-i2\pi k^*z} \, dz \right) + \frac{\partial^2}{\partial \rho^2} \left( \int_{-\infty}^{\infty} \Phi e^{-i2\pi k^*z} \, dz \right) = \frac{1}{\rho} \frac{\partial}{\partial \rho} \left( \rho \frac{\partial \Phi^*}{\partial \rho} \right), \tag{A.1.2.a} \]

\[ F\left( \frac{1}{\rho^2} \frac{\partial^2 \Phi}{\partial \varphi^2} \right) = \frac{1}{\rho^2} \frac{\partial^2}{\partial \varphi^2} \left( \int_{-\infty}^{\infty} \Phi e^{-i2\pi k^*z} \, dz \right) = \frac{1}{\rho^2} \frac{\partial^2 \Phi^*}{\partial \varphi^2}, \tag{A.1.2.b} \]

\[ F\left( \frac{\partial^2 \Phi(\rho, \varphi, z)}{\partial z^2} \right) = \int_{-\infty}^{\infty} \frac{\partial^2 \Phi(\rho, \varphi, z)}{\partial z^2} e^{-i2\pi k^*z} \, dz = \left[ \frac{\partial \Phi(\rho, \varphi, z)}{\partial z} e^{-i2\pi k^*z} \right]_{-\infty}^{\infty} - 2\pi ik^* \int_{-\infty}^{\infty} \frac{\partial \Phi(\rho, \varphi, z)}{\partial z} e^{-i2\pi k^*z} = \]

\[ \left[ \frac{\partial \Phi(\rho, \varphi, z)}{\partial z} e^{-i2\pi k^*z} \right]_{-\infty}^{\infty} - 2\pi ik^* \left[ \Phi e^{-i2\pi k^*z} \right]_{-\infty}^{\infty} - 4\pi^2 k^2 \Phi^* = -k^2 \Phi^*. \tag{A.1.2.c} \]

\( \Phi \) is assumed to be zero for infinite \( z \), so \( \partial \Phi(z)/\partial z \) in (A.1.2.c) is also zero for infinite \( z \) and \( -k^2 \Phi^* \) remains.

We obtain then (2.1.3),

\[ \frac{1}{\rho} \frac{\partial}{\partial \rho} \left( \rho \frac{\partial \Phi^*}{\partial \rho} \right) + \frac{1}{\rho^2} \frac{\partial^2 \Phi^*}{\partial \varphi^2} - k^2 \Phi^* = 0. \tag{A.1.3} \]
A2. Separation of variables

If we write \( \Phi^* (\rho, k, \varphi) \) as \( \Phi^* (\rho, k) \cdot \Phi^* (\varphi) \) (A.1.3) changes into

\[
\frac{\Phi^* (\varphi)}{\rho} \frac{\partial}{\partial \rho} \left( \rho \frac{\partial \Phi^* (\rho, k)}{\partial \rho} \right) + \frac{1}{\rho^2} \Phi^* (\rho, k) \frac{\partial^2 \Phi^* (\varphi)}{\partial \varphi^2} - k^2 \Phi^* (\rho, k) \Phi^* (\varphi) = 0. \tag{A.2.1.1}
\]

Dividing by \( \Phi^* (\varphi) \) and \( \Phi^* (\rho, k) \) and multiplication with \( \rho^2 \) yields

\[
\frac{1}{\Phi^* (\varphi)} \frac{\partial^2 \Phi^* (\varphi)}{\partial \varphi^2} = \rho^2 k^2 - \frac{1}{\Phi^* (\rho, k)} \rho \frac{\partial}{\partial \rho} \left( \rho \frac{\partial \Phi^* (\rho, k)}{\partial \rho} \right) = -\nu^2. \tag{A.2.1.2}
\]

\(-\nu^2\) is in A(2.1.2) the separation constant. With that separation constant we obtain

\[
\frac{\partial^2 \Phi^* (\varphi)}{\partial \varphi^2} + \nu^2 \Phi^* (\varphi) = 0 \tag{A.2.1.3}
\]

and

\[
x^2 \frac{\partial^2 \Phi^* (x)}{\partial x^2} + x \frac{\partial \Phi^* (x)}{\partial x} - \left( x^2 + \nu^2 \right) \Phi^* (x) = 0 \tag{A.2.1.4}
\]

In (A.2.1.4) \( x \) equals \( k \rho \).

The solution of (A.2.1.3) is an exponential equation,

\[
\Phi^* (\varphi) = e^{\nu \rho \varphi}, \tag{A.2.1.5}
\]

in which \( \nu \) necessarily is a natural number because of the periodicity of \( \Phi^* (\varphi), \Phi^* (\varphi) = \Phi^* (\varphi + n2\pi) \). Consequently, \( \nu \) in (A.2.1.5, A.2.1.3, and A.2.1.4) also has to be a natural number.

Equation (A.2.1.4) -with natural numbers \( n \) (replacing \( \nu \))- has as solutions the modified Bessel-functions \( I_n (x) \) and \( K_n (x) \) which are real-valued Bessel functions of real arguments. These modified functions are defined by

\[
I_n (x) = (i)^{-n} J_n (ix) \tag{A.2.1.6}
\]

\[
K_n (x) = \frac{\pi}{2} i^{n+1} [J_n (ix) + iY_n (ix)], \tag{A.2.1.7}
\]

with \( J_n (ix) \) as Bessel functions of the first kind and \( Y_n (ix) \) as Bessel functions of the second kind.

The homogeneous solutions are thereby given by (2.1.4) and (2.1.5).
A.3. Anisotropic media

If we assume that the skin is anisotropic between the x and y-direction of the tissue, (4.2.6) has to be modified. With $\beta = \sigma_y / \sigma_x$ we derive equations which now explicitly belong to angle $\phi$.

\begin{equation}
V_{2,3; y=0, \phi=0} = \frac{I}{2\pi a (\sigma_x \sigma_y)} \left[ 1 + 2 \sum_{n=1}^{\infty} \left( 2 \left[ 1 + \left( \frac{2nh}{a} \right)^2 \right]^{-1/2} - \left[ 1 + \left( \frac{nh}{a} \right)^2 \right]^{-1/2} \right) \right] \tag{A.3.1.a}
\end{equation}

\begin{equation}
V_{2,3; x=0, \phi=\pi/2} = \frac{I}{2\pi a \sigma_y} \left[ 1 + 2 \sum_{n=1}^{\infty} \left( 2 \left[ 1 + \left( \frac{2nh}{\beta a} \right)^2 \right]^{-1/2} - \left[ 1 + \left( \frac{nh}{\beta a} \right)^2 \right]^{-1/2} \right) \right] \tag{A.3.1.b}
\end{equation}

If we take the ratio of the equations (A.3.1.a and b) we derive

\begin{equation}
\frac{V_{23; y=0}}{V_{23; x=0}} = \frac{\beta B}{B(\beta)} \tag{A.3.2}
\end{equation}

in which $B$ and $B(\beta)$ are the terms between the brackets belonging to (A.3.1.a) respectively (A.3.1.b). Filling in the measured potentials we are able to obtain with the terms between the brackets the conductivities in x and y-direction (if $\sigma_x = \sigma_y$, the ratio in (A.3.2) equals one).
Appendix B. Electronic configuration of the CMS, DRL, REF and the channels from the amplifier (§ 4.3)

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