Decrease in skeletal muscle fiber conduction velocity in relation to potassium accumulation during fatigue

Modelling and surface EMG

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Abstract

The possibility to use surface electromyography (SEMG) to identify changes in extracellular potassium concentration inside the skeletal muscle was studied. The goal was to develop a method to relate the measured change in muscle fiber conduction velocity (MFCV) to a change in extracellular potassium concentration ([K$^+$]$_o$) of the skeletal muscle fiber. The advantage of this method is the possibility to analyse in vivo properties of the muscle without using invasive measurements. This technique might be used in future studies to investigate the decrease of the resting membrane potential in critically ill patients.

To be able to relate the MFCV to properties of the muscle fiber, a model of the action potential propagation (MFCV-model) in a muscle fiber was developed. This model is based on the classical action potential model for nerve fibers developed by Hodgkin and Huxley. To improve the model Hodgkin and Huxley’s leakage channel was replaced by a more specific chloride channel.

Experimental MFCV data was obtained from subjects who performed static contractions at 30% of the maximum voluntary contraction (MVC) of the tibialis anterior until exhaustion occurred. This MFCV data shows a decrease in MFCV, that was caused by fatigue. Fatigue in skeletal muscle causes an increase of [K$^+$]$_o$, which leads to a decrease of the resting membrane potential and subsequently results in a reduced MFCV. The changes in MFCV have been used to identify changes in [K$^+$]$_o$. By matching the MFCV-model to the measured change in MFCV, the change in [K$^+$]$_o$ was obtained. The model showed that the decrease in MFCV, from 3.4 to 2.6 m/s, could be explained by an increase in [K$^+$]$_o$, from 4.0 to 7.7 mM. The results indicate that it might be possible to use SEMG to estimate changes in [K$^+$]$_o$ in skeletal muscle fibers.
1 Introduction

Over the past ten years there has been a lot of development in the area of surface EMG (SEMG), which is used to measure the electrical activity of skeletal muscles in a non-invasive way. With new techniques that use electrode arrays, it is possible to determine anatomical properties of single motor units [?, ?, ?] and diagnose muscular disorders [?, ?]. Another property of the skeletal muscle that can be estimated with the electrode array is the muscle fiber conduction velocity (MFCV) [?, ?, ?, ?], which changes during muscle fatigue.

Muscle fatigue is defined as the failure to maintain a required or expected force. During fatigue, potassium accumulates in the extracellular space [?, ?]. The increase in extracellular potassium concentration ([\(K^+\)\text{e}]\text{)} causes the resting membrane potential (RMP) to decrease, this results in a decrease in excitability of the muscle fiber [?] and, finally, this leads to a decrease of MFCV. The decrease of MFCV during fatigue can be measured with SEMG [?, ?, ?]. These measurements are generally performed on healthy subjects. For patients suffering from muscular disorders, the decrease of MFCV during fatigue could provide useful information about the disease. Besides during fatigue, the change in MFCV during recovery from fatigue could also be used for the research into the disease.

For instance critically ill intensive care unit patients suffer from skeletal muscle weakness and a loss of contractility, which can result in respiratory failure [?]. This could be related to a decrease in RMP in critically ill patients [?]. To research the cause of the decrease in RMP, the estimation of the increase in [\(K^+\)\text{e}]\text{)} during fatigue can be a very valuable parameter, because [\(K^+\)\text{e}] is related to the RMP and the MFCV as explained above.

Unfortunately [\(K^+\)\text{e}] during fatigue can not be measured in vivo easily. Therefore it is not possible to relate the changes in MFCV to the changes in [\(K^+\)\text{e}] experimentally. The use of a computer model of the action potential propagation (MFCV-model) is a useful alternative. The MFCV-model was based on the action potential model of a piece of membrane developed by Hodgkin and Huxley (HH-model) [?]. To model the propagation of the action potential in several pieces of muscle fiber, the model made by Adrian and Marshall [?] was used.

The HH-model is based on data that was obtained from the giant nerves of squids. The main properties of the potassium and sodium channels in skeletal muscles and nerve fibers are identical [?]. The chloride channel is not separately modelled in the HH-model, it is incorporated into the leakage channel. The chloride channel plays an important role in stabilizing the RMP in skeletal muscle fibers [?, ?]. Different chloride channels have been identified, of which ClC-1 is the most important in skeletal muscles [?]. A model of this channel was used by Wallinga et al. [?]. It seems likely that a model with a chloride channel instead of a leakage channel, should provide better results, because it was based on skeletal muscle properties.

The first aim of the paper was to investigate how to adapt the electrophysiological model for skeletal muscle fibers to improve MFCV modelling. The MFCV-model had to be able to relate a measured decrease in MFCV to a modelled increase of [\(K^+\)\text{e}]\text{). Therefore it was investigated whether this result could be obtained by a MFCV-model with a ClC-1 channel.

Skeletal muscles have different fiber types, which are divided in type I and type II fibers [?, ?]. In general type I fibers have a smaller radius [?] and a lower MFCV [?], compared to
type II fibers. The type of fibers in a muscle mainly depends on the function of the muscle. In muscles that have to perform short lasting, fast contractions, for instance the extensor digitorum longus muscle, type II fibers dominate. Type I fibers dominate in muscles that have to perform long lasting, slow contractions, for instance the soleus muscle.

With the developed MFCV-model the next goal was to estimate changes in $[K^+]_o$ from changes in MFCV. The SEMG measurements can be obtained from different muscles, with different types of fibers, which results in different MFCV’s. Therefore the radius of the modelled fiber was adapted to match the measured MFCV. To test the MFCV-model, data from Houtman et al. [?] was used. This new technique should provide the possibility to identify physiological changes of $[K^+]_o$ from non-invasive SEMG measurements.
2 Model

2.1 Model description

The basis of the MFCV-model is the HH-model. This model was expanded with the model developed by Adrian and Marshall [?] in order to describe the propagation of the action potential in the muscle fiber. Pieces of fiber were connected with each other to form a longer piece of muscle fiber in which the propagation was modelled. This is schematically presented in figure ???. The transverse tubular system that was used in [?] was not applied to the MFCV-model. This was done because the transverse tubular system makes the model more complex and does not have influence on the propagation of the action potential over the fiber membrane.

Figure 1: Electrical circuit that was used to model the propagation of the action potential between the segments of the fiber membrane.

The leakage channel used in the HH-model not only models the currents of $Cl^-$, but also other ions, like calcium, which is needed to perform a contraction. The MFCV-model does not need to model this kind of properties and therefore they can be neglected when modelling the MFCV. Wallinga et al. [?] used a description of the ClC-1 channel for the chloride channel. One of the known types of chloride channels is the ClC-1 channel, which is assumed to be the main chloride channel in skeletal muscle [?]. The description of the leakage channel was based on measurements on nerve fibers [?]. The chloride channel was based on measurements performed on skeletal muscles [?]. The best results are likely obtained from a channel description of measurements, performed on skeletal muscles. To improve the skeletal muscle model, the leakage channel was replaced by a chloride channel.

The fiber segments were connected to each other by intracellular resistances. The intracellular resistance ($R_i$) was set at 125 Ω cm [?]. At rest $[K^+]_o$ has a value of 4.0 mM [?, ?, ?]. The MFCV has a value of about 3 to 5 m/s [?, ?]. For a muscle fiber at rest, an average MFCV of 4 m/s was chosen. To match the MFCV-model to a MFCV of 4 m/s in the resting state the muscle fiber radius ($r$) was set at 60 µm, which is within the physiological range [?] and gives good model results. The length of a segment ($\Delta x$) was set at 100 µm [?].

To initiate an action potential in the model, an external current ($i_{ext}$ in μA) is applied to the first segment. This current caused the first segment to depolarize, resulting in an action potential, which was conducted to the other segments. The membrane currents ($i_m$ in μA) are calculated from the membrane voltages ($V(s)$) as given by the next formulas.
s is the segment index and has a value between 1 and N. Where N is the number of modelled segments.

\[
i_m(s) = \begin{cases} 
\frac{[V(2) - V(1)]\pi r^2}{\Delta x R_i} + i_{ext}, & s \in \{1\} \\
\frac{[V(s+1) - 2V(s) + V(s-1)]\pi r^2}{\Delta x R_i}, & s \in \{2, ..., N - 1\} \\
\frac{[-V(N) + V(N-1)]\pi r^2}{\Delta x R_i}, & s \in \{N\}
\end{cases}
\]

Per cm² of membrane surface the membrane current (\(I_m\) in \(\mu A/cm^2\)) and external current (\(I_{ext}\) in \(\mu A/cm^2\)) are calculated with the following equations:

\[
I_m(s) = \frac{i_m(s)}{2\pi r \Delta x}, \quad s \in \{1, ..., N\}
\]

\[
I_{ext} = \frac{i_{ext}}{2\pi r \Delta x}
\]

Figure 2: Electrical circuit that was used to model the currents through a segment of the fiber membrane.

A small piece of membrane of the muscle fiber was modelled by a membrane capacity, ionic conductances and voltage sources for the equilibrium potentials of the ions \([\ldots]\). The model can be represented as an electrical circuit, see figure ???. The isolating property between the intra- and extracellular fluid of the membrane was modelled by a capacity. The membrane voltage dependant conductances model the voltage gated sodium, potassium and chloride channels, with the voltage sources to model the equilibrium potential. The parameters for the membrane segment properties were obtained from \([\ldots]\), because these parameters were adapted to match skeletal muscle fiber properties. The formulas for the currents in a membrane segment are given in equations ??, ?? and ?? \([\ldots, \ldots, \ldots]\). The segment index s is omitted in the following equations.

The total membrane current is the sum of the capacitive and ionic currents:

\[
I_m = I_C + I_{ionic}
\]
Where the capacitive current $I_C$ ($\mu A/cm^2$) and ionic current $I_{ionic}$ ($\mu A/cm^2$) are given by:

$$I_C = C_m \frac{dV}{dt}$$

$$I_{ionic} = g_K(V - E_K) + g_{Na}(V - E_{Na}) + g_{Cl}(V - E_{Cl})$$

Where $E_K$, $E_{Na}$ and $E_{Cl}$ are the equilibrium potentials of the potassium, sodium and chloride channel, respectively in mV. The conductances of the potassium ($g_K$ in mS/cm$^2$), sodium ($g_{Na}$ in mS/cm$^2$) and chloride ($g_{Cl}$ in mS/cm$^2$) channel are calculated as follows:

$$g_K = \bar{g}_K n^4$$

$$g_{Na} = \bar{g}_{Na} m^3 h$$

$$g_{Cl} = \bar{g}_{Cl} a^4$$

Where $\bar{g}_K$, $\bar{g}_{Na}$ and $\bar{g}_{Cl}$ are the maximum potassium, sodium and chloride conductances, respectively in mS/cm$^2$. The Hodgkin-Huxley conductance parameters $n$, $m$ and $h$ are used to introduce membrane voltage dependency into the conductances of the potassium and sodium channels. They are calculated with the following set of equations:

$$\frac{dy}{dt} = \alpha_y(1 - y) - \beta_y y; \quad y \in \{m, n, h\}$$

$$\alpha_n = \frac{\bar{\alpha}_n(V - \bar{V}_n)}{1 - e^{-(V-\bar{V}_n)/K_{an}}}; \quad \beta_n = \bar{\beta}_n e^{-(V-\bar{V}_n)/K_{bn}}$$

$$\alpha_m = \frac{\bar{\alpha}_m(V - \bar{V}_m)}{1 - e^{-(V-\bar{V}_m)/K_{am}}}; \quad \beta_m = \bar{\beta}_m e^{-(V-\bar{V}_m)/K_{bm}}$$

$$\alpha_h = \bar{\alpha}_h e^{-(V-\bar{V}_h)/K_{ah}}; \quad \beta_h = \frac{\bar{\beta}_h}{1 + e^{-(V-\bar{V}_h)/K_{bh}}}$$

The voltage dependency of the chloride channel is calculated with a Boltzmann distribution function given below.

$$a = \frac{1}{1 + e^{\frac{-V-\bar{V}_a}{\Delta a}}}$$

The exact description of the constants used in equations is described in appendix [?].

The equilibrium potentials ($E_{Na}$, $E_K$, $E_{Cl}$) depend on the intra- and extracellular concentrations and the conductance of the membrane for an ion. The equilibrium potential of an ion is calculated with the Nernst equation.

$$E_K = 10^3 \frac{RT}{F} \ln \frac{[K^+]_o}{[K^+]_i}$$

$$E_{Na} = 10^3 \frac{RT}{F} \ln \frac{[Na^+]_o}{[Na^+]_i}$$

$$E_{Cl} = 10^3 \frac{RT}{F} \ln \frac{[K^+]_o + 0.01[Na^+]_o}{[K^+]_i}$$

Where $[Na^+]_i$ and $[Na^+]_o$ are the intra- and extracellular sodium concentrations, respectively, and $[K^+]_i$ is the intracellular potassium concentration, all in mM. The parameters
$R$, $T$ and $F$ are the gas constant in J/K/mol, the temperature in K and Faraday’s constant in C/mol, respectively.

For the calculation of $E_{Cl}$ a combination of the potassium and sodium concentrations was used [?] to introduce a dependency of the RMP on $[K^+]_o$. The idea behind the relation between $E_{Cl}$ and $[K^+]_o$ is explained below. The RMP is calculated with the Goldman-Hodgkin-Katz equation.

$$RMP = \frac{RT}{F} \ln \frac{P_K[K^+]_o + P_{Na}[Na^+]_o + P_{Cl}[Cl^-]_i}{P_K[K^+]_i + P_{Na}[Na^+]_i + P_{Cl}[Cl^-]_o}$$  \hspace{1cm} (18)$$

Where $P_K$, $P_{Na}$ and $P_{Cl}$ are the potassium, sodium and chloride permeability of the membrane in the resting state, respectively. $[Cl^-]_i$ and $[Cl^-]_o$ are the intra- and extracellular chloride concentrations. $P_{Na}$ is small compared to $P_K$ and $P_{Cl}$, and therefore, the influence of the sodium concentrations on RMP is little. The chloride ions ($Cl^-$) and the potassium ions ($K^+$) form a Donnan equilibrium [? , ?]:

$$\frac{[K^+]_o}{[K^+]_i} = \frac{[Cl^-]_i}{[Cl^-]_o}$$  \hspace{1cm} (19)$$

If the chloride concentration is changed, diffusion of $Cl^-$, $K^+$ and water occurs until the previous concentration gradient for $Cl^-$ is restored [?]. The result is a redistribution of $Cl^-$ to restore the Donnan equilibrium. If the potassium concentration is changed, a shift in RMP occurs. In this case, the $K^+$ concentration gradient will remain at its new value and the $Cl^-$ concentration gradient will change in order to restore the Donnan equilibrium [?]. Thus alterations in $[K^+]_o$ will not only result in changes of $E_K$, but of $E_{Cl}$ as well.

Experiments with the model show that the modelled RMP is almost totally determined by $E_{Cl}$. In reality, however, the RMP does not change if the chloride concentrations in the muscle fiber change, due to restoring of the Donnan equilibrium. However changing $[K^+]_o$ does not only result in a change in RMP, but also in a change in the $Cl^-$ concentrations. Therefore it is a good way to introduce the dependency of RMP on $[K^+]_o$ in the model by adapting $E_{Cl}$.

The exact description and values of the model constants is given in appendix ??.

### 2.2 Model input/output

Increasing $[K^+]_o$ results in a lower RMP. With a lower RMP the current required to generate an action potential decreases, due to the decreased excitability. When the applied current is unchanged at increased $[K^+]_o$, an action potential was generated with an overshoot. To prevent the overshoot effect $i_{ext}$ is adapted to $[K^+]_o$. For a table with the values of $i_{ext}$ see appendix ??.

The ion concentrations were kept constant during one simulation. The influence of changes in $[K^+]_o$ on MFCV were investigated by running the model for different values of $[K^+]_o$ in the range from 4.0 to 8.0 mM. This range was chosen, because 8.0 mM is the maximum value that can be used with the described model. Higher values of $[K^+]_o$ resulted in spontaneous action potentials, which made it impossible to use the model for a determination of MFCV.

The output of the model for one segment is the membrane voltage in time (example in figure ??). The first 40 and last 30 segments were not used for the MFCV calculation.
This was done because of starting and ending effects that occurred at the segments at the edges of the modelled fiber, as can be seen in figure 3.

![Figure 3: An example of the modelled action potentials for $[K^+]_o$ is 4.0 mM (A) and $[K^+]_o$ is 8.0 mM (B). Not all segment membrane voltages are depicted. The depicted segments are: s=1,10,20,...,90,100. The starting and ending effect are indicated by arrows. $r=60 \, \mu m$.](image)

To determine the MFCV the time the action potential needs to travel from segment to segment has to be calculated. Of each segment, the time at which the membrane voltage reaches its maximum ($t_{max}$ in ms) was determined. Now the time the action potential takes to travel from one segment to another can be calculated. To increase the accuracy of the calculated MFCV, the time the action potential takes to travel from segment $s$ to segment $s+10$ was used. Together with the distance between the segments, the MFCV in m/s can be calculated.

![Figure 4: The modelled MFCV as a function of the number of segments used in the model. The MFCV is calculated by averaging $n$ velocities. $n$ is the number of segments minus 79, the first 40 and last 30 segments are not used because of starting and ending effects. $[K^+]_o$ = 8.0 mM, $r=60 \, \mu m$.](image)
as shown in figure ??). This resulted in the following equation for the MFCV:

\[
MFCV = \frac{1}{2 \Delta x} \sum_{s=40}^{60} \frac{10 \Delta x 10^{-2}}{t_{max}(s + 10) - t_{max}(s)} 10^{-3}
\]  

(20)

Where \( \Delta x \) is multiplied by \( 10^{-2} \), because \( \Delta x \) was defined in \( cm \) for the model calculations. The time difference is multiplied by \( 10^{-3} \), because the model used \( ms \) as a time unit.

### 2.3 Modelling methods

The differential equations (Eq. ??, ??) in the model description were solved by an ordinary differential equation (ODE) solver in Matlab 6.5 (The Mathworks, Inc.). A stiff 'ode15s' solver was used with a relative error tolerance of \( 10^{-9} \). This low error tolerance was necessary to guarantee a smooth curve at the top of the modelled action potential. This smooth curve was needed to get an accurate determination of \( t_{max} \), because this time was used to calculate the MFCV. Figure ?? shows the calculated MFCV for different values of the relative error tolerance. The relative error tolerance is the most important of the relative and absolute error tolerance parameters. Because the ode-solver uses the maximum error tolerance, the absolute error tolerance was set to \( 10^{-20} \) to be sure that only the relative error tolerance was of importance. In figure ?? it can be seen that the calculated MFCV was constant enough from a relative error tolerance of \( 10^{-9} \).

![Figure 5: The modelled MFCV as a function of the relative error tolerance. \([K^+]_o=4.0 \text{ mM}, r=60 \text{ \mu m}\).](image)

For each segment four differential equations needed to be solved. For 100 segments this resulted in a model with a total of 400 states. Despite this high number of states, the model was calculated within 70 \( s \) for one value of \([K^+]_o\) on a 2.2 GHz Pentium-4 PC.
3 Results

3.1 Model

The developed model was suited to model changes of MFCV caused by changes in $[K^+]_o$ in the range from 4.0 to 8.0 mM, which is likely to occur during exercise [1]. Higher concentrations of $[K^+]_o$ resulted in spontaneous action potentials, but higher concentrations during fatigue have been reported in literature [2]. The MFCV-model was compared to a model that incorporated the leakage channel, based on the description used by Cannon et al. [3]. The exact description of the leakage channel used can be found in appendix ?? A comparison between the MFCV-model and the model with the leakage channel is given in figure ?? A and B. There is an obvious difference in the modelled MFCV values (Fig. ??A). It is difficult to determine which one is the best. The most important property is how the MFCV decreases with increasing $[K^+]_o$. This is shown in figure ??B, in which the MFCV is expressed as a percentage of the initial MFCV (MFCV%). The initial MFCV is the one modelled for $[K^+]_o$ equal to 4.0 mM, which is assumed to be the $[K^+]_o$ at rest. Figure ??B shows that the decrease of MFCV% in the two models is almost identical. Therefore it is concluded that both models are suited to model the MFCV. However the model with the chloride channel is preferred, because it can model the MFCV over a wider range of $[K^+]_o$ values.

![Figure 6](image)

Figure 6: The MFCV calculated with the leakage channel (dashed) and with the chloride channel (solid) for different values of $[K^+]_o$. $[K^+]_o$ was increased with steps of 0.02 mM from 4.0 to 6.0 mM. In A the MFCV is given in m/s, in B the MFCV is given as a percentage of the initial MFCV.

For $[K^+]_o$ higher than 8.0 mM the model could not be used, because of the occurrence of spontaneous action potentials. For even higher values of $[K^+]_o$, the RMP should decrease that much that it becomes inexcitable [2]. Obviously this did not occur in the MFCV-model. A possible explanation could be the lacking of slow inactivation of the sodium conductance in the MFCV-model. Namely sodium channels become inactivated when RMP is decreased [2]. Moreover too many inactivated sodium channels make it impossible to
generate action potentials in the muscle fiber [?].

To use the MFCV-model for higher values of $[K^+]_o$, incorporating the slow inactivation of sodium conductance might solve the problem of the spontaneous action potentials. Therefore the slow inactivation of sodium conductance was implemented from the model developed by Wallinga et al. [?]. The description of the slow inactivation of sodium is given in appendix ???. The result, given in figure ???, shows that the MFCV decreases much faster with an increase in $[K^+]_o$ compared to the MFCV-model without slow inactivation of sodium conductance.

Figure 7: MFCV’s calculated with the MFCV-model with slow inactivation of sodium conductance from Wallinga’s model [?]. $[K^+]_o$ is in the range from 4.0 to 6.0 mM, calculated in steps of 0.02 mM. $r = 60 \mu m$.

For $[K^+]_o$ higher than 6.5 mM no action potentials could be elicited in the model with slow inactivation of sodium conductance. However according to Sejersted et al. [?] higher $[K^+]_o$ are needed to reach depolarization block due to inactivation of sodium conductance. The results of the MFCV-model without slow inactivation of sodium conductance seemed to provide a better match with the measurements performed by Juel [?]. Juel performed in vitro measurements of the MFCV, on isolated mouse soleus and extensor digitorum longus muscles, at different $[K^+]_o$. The problem of the spontaneous action potentials was solved by implementing the slow inactivation of sodium conductance. But the relation between $[K^+]_o$ and MFCV from the MFCV-model seemed to provide better results.

Running the MFCV model for values of $[K^+]_o$ from 4.0 to 8.0 mM resulted in a look-up-table. The look-up-table gives the relation between $[K^+]_o$ and the MFCV, graphically this is depicted in figure ??A. With this look-up-table it is also possible to relate measured MFCV’s to the corresponding $[K^+]_o$, figure ??B.

An important parameter for the MFCV calculated by the model is the muscle fiber radius ($r$). The intracellular resistance of the muscle fiber depends on $r$ (Eq. ??). When $r$ increases, the current between two segments increases, due to a smaller intracellular resistance. This results in a higher MFCV, because the current of the action potential is conducted better. This is in correspondence with the physiological properties of muscle fibers, namely larger fibers have a higher MFCV [?, ?]. The dependence of MFCV on $r$ was used to match the model with the MFCV data for rest. The MFCV was calculated as a function of $r$, the result is depicted in figure ??.
Figure 8: Results of the MFCV-model for $r = 60 \ \mu m$. $[K^+]_o$ was increased with steps of 0.02 mM from 4.0 to 8.0 mM. A shows the MFCV as a function of $[K^+]_o$. B shows the relation that can be derived from A, $[K^+]_o$ as a function of MFCV. This figure shows the relation between changes in MFCV and changes in $[K^+]_o$.

Figure 9: The MFCV as a function of the muscle fiber radius for $[K^+]_o=4.0 \ \text{mM}$.

3.2 MFCV data

To test our model data from Houtman et al. [?] was used. Houtman et al. performed MFCV measurements during static contractions. Their interest was to measure changes in MFCV and relate these to changes in motor unit recruitment. The measurements were done on healthy persons who performed static contractions with the tibialis anterior at 30 % of maximal voluntary contraction.

During the first part of Houtman’s measurements, there is no change in the recruited motor units [?] and the measured MFCV decreases. It is important that no change in recruited motor units occurs for two reasons. First a newly recruited motor unit starts without accumulation of $[K^+]_o$. Second another motor unit can have fibers with a different radius resulting in a difference in MFCV. The first part of their measurements was used to test the MFCV-model. The data from Houtman et al. is given in figure ??.
MFCV drops from about 3.4 to 2.6 m/s during the first 190 s.

Figure 10: Used MFCV data measured by Houtman et al.. The first part of the measurement shows a roughly linear decrease of the MFCV, this data was used to test our model. Copied from [?].

3.3 Combining model and data

Matching measured MFCV data with the modelled MFCV in rest was done by adapting the fiber radius in the model. The fiber radius needed for the model was obtained from figure ???. For the data from Houtman et al., with a starting value of about 3.4 m/s, this resulted in a fiber radius of 45 µm. During the measurements there are a number of active muscle fibers, which do not have the same radius. Therefore the radius of 45 µm represents the average fiber radius. When the fiber radius is reduced, \( i_{ext} \) needs to be reduced to prevent overshoot. The exact values of \( i_{ext} \) for a fiber radius of 45 µm are given in appendix ??.

Applying the method mentioned above on the data from Houtman et al. resulted in figure ??A. Because the MFCV decreased roughly linear in time, the relation between time and the MFCV is known. Using this knowledge the increase of \( [K^+]_o \) was plotted as a function of time (Fig. ??B).
Figure 11: A: Results of the increase of $[K^+]_o$, caused by fatigue, as a function of MFCV. B: Results of the increase of $[K^+]_o$, caused by fatigue, as a function of time. The results were based on the MFCV data from Houtman et al. where a roughly linear decrease in MFCV from 3.4 to 2.6 m/s was measured during a time of 190 s. $r=45 \mu m$. 
4 Discussion

The goal of the research was to develop a model of skeletal muscles that describes the relation between accumulation of \([K^+]_o\) during fatigue and the decrease in MFCV. The developed MFCV-model made it possible to relate changes in MFCV during fatigue to changes in \([K^+]_o\). Where \([K^+]_o\) was changed in the range from 4.0 to 8.0 mM, which is likely to occur during exercise [?]. By replacing the leakage channel of the HH-model by a chloride channel, the modelling range of \([K^+]_o\) was doubled. This increase of \([K^+]_o\) was necessary to model the decrease in MFCV during fatigue measured by Houtman et al. [?]. This was not possible using the model with the leakage channel, because the range in which \([K^+]_o\) could be changed was too small.

Spontaneous action potentials occurred in the MFCV-model for \([K^+]_o\) higher than 8.0 mM. It was clear that implementing Wallinga’s slow inactivation of sodium conductance, solved this problem. But the resulting relation between \([K^+]_o\) and MFCV seemed unlikely. Therefore further research into the slow inactivation of sodium conductance is needed, for improvement of the MFCV-model.

The decrease in RMP, caused by a doubling in \([K^+]_o\) from 4.0 to 8.0 mM in the MFCV-model was about 17 mV (Fig. ??). This was in accordance with the recorded depolarization in mouse and rat muscles, of somewhat less than 18 mV [?]. This indicates that the modelled decrease in RMP due to an increase in \([K^+]_o\) was correct.

From the above mentioned facts, it was concluded that the MFCV-model with chloride channel and without slow inactivation of sodium conductance, was better suited to model the relation between the decrease in MFCV and the increase in \([K^+]_o\) than the model with the leakage channel.

In the MFCV-model it was assumed that only \([K^+]_o\) changes during fatigue. This was done for practical reasons. The only data was the measured MFCV, therefore only one parameter could be estimated. \([K^+]_o\) was chosen as output parameter, because it is affected most by fatigue [?]. But the other concentrations, \([K^+]_i\), \([Na^+]_o\) and \([Na^+]_i\), in the muscle fiber also change during fatigue [?]. Changes in these concentrations can also be related to a change in MFCV. A parameter sensitivity study revealed that the other concentrations in the model had a negligible or small influence on MFCV compared to the sensitivity of \([K^+]_o\). \([Na^+]_i\) had the most influence on MFCV. Increasing \([Na^+]_o\) from 24 to 30 mM, resulted in a decrease of MFCV from 4.0 to 3.8 m/s. Which was small compared to the decrease of MFCV from 4.0 to 2.8 m/s as a result of an increase in \([K^+]_o\) from 4.0 to 8.0 mM.

Recent studies concluded that the changes in action potentials are probably the result of ion shifts that depolarize the membrane during exercise [?]. From this and the sensitivity study, it was a valid assumption that \([K^+]_o\) was the most important parameter of changes in MFCV during fatigue.

With the MFCV-model, it is possible to indicate how \([K^+]_o\) increases in time during fatigue. Certain muscular disorders could cause a deviation in the change of \([K^+]_o\) in time during fatigue or recovery from fatigue. In this case the MFCV-model could be suited to detect this deviation. This information might be useful to diagnose certain muscular disorders, to do research in this field and develop therapeutic interventions.

A combination of SEMG and the MFCV-model might for instance be used for the research
into the cause of muscle weakness and loss of contractility in critically ill patients \cite{?}. One of the changes in critically ill patients is a decrease of RMP \cite{?}. The decrease in RMP might be directly related to muscle atrophy \cite{?}, which leads to muscle weakness. The MFCV-model can help to detect changes in RMP, because of its relation with $[K^+]_o$.

The measured MFCV was matched to the modelled MFCV in rest by changing the muscle fiber radius. This property might be used to detect changes in muscle fiber radius in certain muscular disorders.

For example, in diabetics suffering from polyneuropathy, reinnervation of muscle fibers occurs, due to nerve fiber loss \cite{?}. The reinnervation can cause muscle fibers to change of fiber type, because the type of fiber is determined by the nerve supplying the muscle fiber \cite{?}. The change in muscle fiber type, results in a different fiber radius \cite{?}, which leads to a change in MFCV \cite{?}. Due to the reinnervation no loss of muscle strength occurs, so the diabetic does not notice the neuropathy at the muscle. But the change in muscle fiber type and radius might be detected with the use of the MFCV-model. In this case SEMG in combination with the MFCV-model, might be used to diagnose the neuropathy at the muscle in diabetics.

Besides accumulation of extracellular potassium, the MFCV-model was also run for a decrease in $[K^+]_o$. The results are given in figure 12. For lower $[K^+]_o$ the modelled MFCV decreased and the RMP increased. However a decrease in RMP at lower $[K^+]_o$ has been reported for skeletal muscles \cite{?}. It remains unclear whether the modelled decrease of MFCV at lower $[K^+]_o$ is physiologically correct.

![Figure 12: MFCV’s calculated with the MFCV-model for $[K^+]_o$ in the range from 2.0 to 6.0 mM, calculated in steps of 0.02 mM. $r$=60 µm.](image)

The model developed by Wallinga et al. \cite{?} used additional channels to model the action potentials in skeletal muscle. The purpose of Wallinga’s model was to describe the accumulation of potassium. The model incorporated the inward rectifier conductance of potassium ($g_{K,IR}$) and the Na-K pump. These two parts played an important role in the prevention of accumulation of potassium in Wallinga’s model. The MFCV-model however, was used to describe the MFCV at different $[K^+]_o$, which was assumed to increase. The increase of $[K^+]_o$ itself did not have to be modelled. The contribution of the Na-K pump and $g_{K,IR}$, to the action potential itself was negligible. Their activity plays a more important role in between action potentials in Wallinga’s model. Therefore it was not necessary
to incorporate the Na-K pump and $g_{K_{ir}}$ into the MFCV-model.

In summary, the combination of SEMG and the MFCV-model was able to relate the measured MFCV to an accumulation of extracellular potassium. This technique might provide useful information for the research into muscular disorders.
A Model parameters

A.1 Explanation of used parameters

- $\alpha_y, \beta_y$: rate constants in $y \in \{h, m, n\}$ ($ms^{-1}$)
- $\bar{\alpha}_y, \bar{\beta}_y$: maximum rate constant in $y \in \{h, m, n\}$ ($ms^{-1}mV^{-1}$)
- $K_{\alpha_y}, K_{\beta_y}$: gating variable in $y \in \{h, m, n\}$ ($mV$)
- $\bar{V}_y$: voltage at which $y \in \{h, m, n\}$ is at half its maximum value ($mV$)
- $a$: Boltzmann distribution function
- $A_a$: variable determining steepness of $a$
- $V_a$: voltage at which $a$ is at half its maximum value ($mV$)

A.2 Values of used parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Units</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>$K_{\alpha_n}$</td>
<td>7</td>
<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$K_{\beta_n}$</td>
<td>40</td>
<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$\bar{V}_n$</td>
<td>-40</td>
<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$\bar{\alpha}_n$</td>
<td>0.0131</td>
<td>$ms^{-1}$</td>
<td>[?]</td>
</tr>
<tr>
<td>$\bar{\beta}_n$</td>
<td>0.067</td>
<td>$ms^{-1}$</td>
<td>[?]</td>
</tr>
<tr>
<td>$K_{\alpha_m}$</td>
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<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$K_{\beta_m}$</td>
<td>18</td>
<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$\bar{V}_m$</td>
<td>-46</td>
<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$\bar{\alpha}_m$</td>
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<td>$ms^{-1}$</td>
<td>[?]</td>
</tr>
<tr>
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<td>1.38</td>
<td>$ms^{-1}$</td>
<td>[?]</td>
</tr>
<tr>
<td>$K_{\alpha_h}$</td>
<td>14.7</td>
<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$K_{\beta_h}$</td>
<td>9</td>
<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$\bar{V}_h$</td>
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<td>mV</td>
<td>[?]</td>
</tr>
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<td>[?]</td>
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<tr>
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<td>150</td>
<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$V_a$</td>
<td>70</td>
<td>mV</td>
<td>[?]</td>
</tr>
<tr>
<td>$\bar{g}_K$</td>
<td>21.6</td>
<td>$mS/cm^2$</td>
<td>[?]</td>
</tr>
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<td>$\bar{g}_{Na}$</td>
<td>150</td>
<td>$mS/cm^2$</td>
<td>[?]</td>
</tr>
<tr>
<td>$\bar{g}_{Cl}$</td>
<td>6.55</td>
<td>$mS/cm^2$</td>
<td>[?]</td>
</tr>
<tr>
<td>$[K^+]_i$</td>
<td>156</td>
<td>mM</td>
<td>[?]</td>
</tr>
<tr>
<td>$[Na^+]_o$</td>
<td>150</td>
<td>mM</td>
<td>[?]</td>
</tr>
<tr>
<td>$[Na^+]_i$</td>
<td>24</td>
<td>mM</td>
<td>[?]</td>
</tr>
</tbody>
</table>

Concentrations

Physical and other parameters

- $C_m$: 1 $\mu F/cm^2$ [?] 
- $R_i$: 0.125 $k\Omega cm$ [?] 
- $F$: 96485 $C/mol$ 
- $R$: 8.31441 $J/(Kmol)$ 
- $T$: 295 $K$ [?] 
- $\Delta x$: 0.01 cm [?]
A.3 Stimulus current

Stimulus current for \( r = 60 \mu m \)

<table>
<thead>
<tr>
<th>([K^+]_o) in mM</th>
<th>(i_{ext}) in (\mu A)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 &lt;= ([K^+]_o) &lt; 2.75</td>
<td>0.3</td>
</tr>
<tr>
<td>2.75 &lt;= ([K^+]_o) &lt; 3.5</td>
<td>0.25</td>
</tr>
<tr>
<td>3.5 &lt;= ([K^+]_o) &lt; 5</td>
<td>0.2</td>
</tr>
<tr>
<td>5 &lt;= ([K^+]_o) &lt; 6.5</td>
<td>0.15</td>
</tr>
<tr>
<td>6.5 &lt;= ([K^+]_o) &lt; 7.5</td>
<td>0.1</td>
</tr>
<tr>
<td>7.5 &lt;= ([K^+]_o) &lt;= 8</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Stimulus current for \( r = 45 \mu m \)

<table>
<thead>
<tr>
<th>([K^+]_o) in mM</th>
<th>(i_{ext}) in (\mu A)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 &lt;= ([K^+]_o) &lt; 5</td>
<td>0.13</td>
</tr>
<tr>
<td>5 &lt;= ([K^+]_o) &lt; 7</td>
<td>0.1</td>
</tr>
<tr>
<td>7 &lt;= ([K^+]_o) &lt;= 8</td>
<td>0.05</td>
</tr>
</tbody>
</table>

B Leakage channel

The leakage channel replaces the chloride channel of the MFCV-model described in the article. This means that the equation of the chloride current \((g_{Cl}(V - E_{Cl}))\) in equation ?? needs to be replaced by the leakage current \((I_L)\) equation given below [?].

\[
I_L = \bar{g}_L (V - E_L)
\]

\[
\bar{g}_L = 0.75
\]

\[
E_L = \frac{RT}{F} \ln \left( \frac{[K^+]_o + 0.01[Na^+]_o}{[K^+]_i} \right)
\]

C Slow inactivation of sodium

To implement the slow inactivation of sodium, equation ?? was replaced by the equation below [?].

\[
g_{Na} = \bar{g}_{Na} m^3 h S
\]

\[
\frac{dS}{dt} = \frac{S_\infty - S}{\tau_S}
\]

\[
\tau_S = \frac{60}{0.2 + 5.65(V+90)^2}
\]

\[
S_\infty = \frac{1}{1 + e^{\frac{V+78}{5.8}}}
\]

Where \(m\) and \(h\) are calculated by equations ??, ?? and ??.
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Recent progress in the diagnostic use of surface EMG for neurological diseases.
SEMG setup for measurements of MFCV from stimulated contractions

by W.G.J. van Vlimmeren
1 Introduction

During previous work a SEMG setup was developed by Kierkels [8] and De Bekker [1] to perform surface EMG (SEMG) measurements of the flexor digitorum superficialis. With this original setup it was possible to perform measurements of the muscle fiber conduction velocity (MFCV) from stimulated contractions. However the practical use of the setup was rather bad. Therefore it was investigated how the setup could be improved and how the determination of the MFCV could be optimized.

The modified SEMG setup should be used to perform MFCV measurements for the research into extracellular potassium accumulation during fatigue. For this purpose a model was developed that estimates extracellular potassium concentration from changes in MFCV [14].

The original setup had standard Ag/AgCl electrodes. These electrodes were not practical in use. Therefore other types of electrodes were investigated. For the new setup silver bar electrodes in an array were tested. It was investigated whether the silver bar electrodes could be used for SEMG.

The silver bar electrodes have other conducting properties than the Ag/AgCl electrodes. This caused an increased offset between the electrodes. The amplifier had to be adapted to be able to remove the increased offset.

The MFCV had to be calculated from the SEMG. This was done using the cross-correlation technique [4]. Some improvements were made to the MFCV calculation. The main improvement concerns the selection of the data on which the cross-correlation technique was applied.

With the modified SEMG setup good results were obtained. MFCV calculations were performed on data from SEMG measurements, which were performed during stimulated contractions of the flexor digitorum superficialis.
2 Electrodes

The used standard Ag/AgCl-electrodes in the original setup had to be modified to be able to place them close enough to each other. The result was that the gel of the electrodes could easily get in touch with each other, making it impossible to perform good measurements. Furthermore the electrodes could easily move with respect to each other. This resulted in a varying inter electrode distance (IED), which made it hard to determine a good MFCV.

Literature showed that silver electrodes can be used for SEMG [10]. The biggest advantage of the silver electrodes is that they are smaller in size, for instance 10 mm by 1 mm, and can be varied in size. This makes it easy to place them close to each other at a fixed IED. Another advantage of the small size is that it is possible to place an array of electrodes over the muscle [2, 10], because the electrodes can be placed closer to each other. With the array more signals can be measured, which improves the determination of the MFCV [5] and other characteristics of the muscle can be investigated [10], like the locations of the innervation zone and the muscle tendon transitions.

There are many different electrode configurations. The most commonly used one is a differential setup, which was also used in the original setup. A differential setup is preferred over a monopolar setup, because 50 Hz net interference is less. However it is possible to perform monopolar measurements, the problem of 50 Hz interference is solved by using the driven right leg technique [11, 12]. But this was not possible with the available amplifier.

The electrodes can also be used to form spatial filters [3, 13]. The advantage of these electrode setups is that they are better suited to measure the electromyographic activity of one or only several motor units. The disadvantage is that this results in a low SEMG signal amplitude, due to the selectivity of the filter. Another disadvantage is that the spatial filter setup can only be used for superficial muscles with a small fat layer. The SEMG setup was used to measure the SEMG of stimulated contractions. In this case the motor units are activated simultaneously. In that case there is no need to measure the electromyographic activity of only one motor unit.

For future research it might be necessary to be able to measure one motor unit. This can be done using spatial filters. Another option is to further investigate the technique of threshold stimulation combined with detection of single motor units by singular value decomposition developed by Kierkels [8]. Or maybe a combination of these two techniques.

The available amplifier was constructed to measure a set of three electrodes. This amplifier was used to test whether it was possible to measure good SEMG signals with the silver electrodes. For this purpose the amplifier had to be improved, see section 3.

The test setup contained three silver electrodes of 1 mm by 10 mm. The electrodes were placed at a fixed IED of 10 mm in a piece of plastic. This made it easy to place the
electrodes properly and they were unable to shift. The signals from the electrodes were amplified differentially. The used electrode setup is shown in figure 1.

Figure 1: Differential silver electrode setup.
3 Amplifier

The original amplifier developed by De Bekker [1] was designed for the measurements with the Ag/AgCl-electrodes. To perform measurements with the silver bar electrodes some modifications were necessary. The exact changes made in the amplifier are described in appendix B.

The conducting properties of the Ag/AgCl electrodes are much better than of the silver electrodes. This had a negative effect on the offset between the silver electrodes. The offset compensation of the amplifier had to be adapted. The original amplifier was able to remove an offset of 12 mV. The modified amplifier could remove an offset of 57 mV.

Another way is to reduce the offset by using conducting gel. However this makes the effective surface of the electrode bigger which in some cases is undesired.

The amplifier used an isolation amplifier (ISO122, Burr-Brown) to isolate the patient from the net power supply. A property of the isolation amplifier used was that it produced a disturbance of 20 mV at 500 kHz. This disturbance was not removed in the original amplifier. To be sure that this does not lead to aliasing, the modified amplifier had a RC-lowpass filter to remove the disturbance.


4 MFCV determination

The MFCV was calculated from the SEMG data. This was done by determining the time delay between the two SEMG signals of an action potential, which occurred after stimulation of the muscle. Some improvements were made to the MFCV calculation with respect to the calculation used by Kierkels [8]. The implementation is shortly described below. The Matlab implementation of the MFCV calculation is given in appendix A.

The used stimulus was large enough to be sure that an action potential would be elicited. Therefore the action potential signals could be detected by detecting the peaks of the stimulus. A stimulus was detected when a sample had an absolute value higher than 3 V.

When a stimulus was found, it was known that the action potential would follow in the next 100 to 1000 samples. The first 100 samples were not used, because they contained the stimulus signal. On the selected data set from 100 to 1000 samples first a constant detrend was applied. This was done on the selected data set, because this provided better results than performing a detrend on the whole measured data set, as was done in the original MFCV calculation.

A cross-correlation was calculated from the data set to determine which of the two signals was the one where the action potential occurred first, the first signal. In this way it was no problem if the electrodes were placed in another order, because the calculation detects what is the order of the two signals.

The next step was to detect the minimum of the first signal and the maximum of the second signal. Then a cross-correlation was performed on the data from 100 samples before the minimum to 100 samples after the maximum. The maximum of this cross-correlation gave the delay between the samples. It was shown that using this selected data for the cross-correlation provided better results than using the data from 100 to 1000 samples after the detection of the stimulus. This was so because the selected data set contained the most important characteristics of the measured action potential. The data from 100 to 1000 was more likely to contain disturbances, which influence the cross-correlation.

With the delay the signals were matched by shifting them with the calculated time delay. Then the correlation coefficients were calculated. The correlation coefficient is a value for the amount of similarity of the signals. To do a good calculation of the MFCV the two signals have to be very similar. Therefore the correlation coefficient had to be higher than 0.9, on a scale of 0 to 1. If the correlation coefficient was high enough, the MFCV was calculated. If the correlation coefficient was too low, no MFCV was calculated and the correlation coefficient error was increased by one. The correlation coefficient error was used to see how many action potentials were rejected.
5 Results

In figure 2 an example of a measured SEMG is shown. In the beginning the stimulus artifact is visible.

Figure 3 shows the calculated MFCV’s from a SEMG. The calculated MFCV’s seem to show a certain periodicity. The origin of this periodicity was unknown. The measurements were performed with a medium stimulus at 3 Hz.

The SEMG signals were sampled with at 32 kHz. For a speed of 4 m/s and an electrode distance of 10 mm, this resulted in a resolution of 0.05 m/s.

Figure 2: Example of measured SEMG signals.
Figure 3: Differential silver electrode setup.
6 Discussion

With the developed setup it was possible to perform good SEMG measurements from stimulated contractions of the flexor digitorum superficialis. From the SEMG the MFCV’s could be calculated with the provided m-file.

The calculated MFCV’s show a periodicity in time. Maybe this is caused by the stimulus device. In the future it has to be investigated whether the cause of the periodicity is the stimulus device or physiological. This can be done by using another stimulus device or by measuring the applied stimulus.

For future research SEMG measurements will be performed on voluntary isometric contractions, like in [7]. One of the main differences with the stimulated contractions is the fact that the motor units are not innervated simultaneously. This results in a smaller amplitude of the action potentials in the SEMG. Furthermore the action potentials will interfere with each other. Therefore more advanced methods will have to be applied to provide a good estimation of the MFCV.

When electrode arrays with more than three electrodes are used in the future, it might be possible that a sample rate of 32 kHz can not be used anymore. To obtain a good resolution of the MFCV at lower sample rates, other methods will have to be applied to the calculation of the MFCV.

To obtain a good resolution at lower sample rates the SEMG signal can be transformed to the frequency domain [4]. Where different techniques can be applied to determine the time delay. When using two SEMG signals the maximum likelihood estimator [9] is preferred, because of its low variance and high resolution of estimation [10]. When arrays with more electrodes are used, other techniques can provide better results. A comparison of two techniques for electrode arrays, the modified beamforming algorithm and the maximum likelihood estimator, is given in [5].

For future research that is done with stimulated contractions the developed SEMG setup is good enough. To provide more data to calculate the MFCV it might be useful to extend the amount of used electrodes. When this is done a new amplifier with more channels must be developed, this can be done with the techniques used in the current amplifier. It can be useful to investigate the possibilities of a monopolar setup at that moment.
function [velocity,stimtime,vel_mean,vel_std] = veldat(file,first,last,plot_data)

% Function to calculate velocitys from measured data sets with stimulated contraction
%
% [velocity,stimtime,vel_mean,vel_std] = veldat(file,first,last,plot_data)
% 'file' is the rootname of the .dat files that are loaded.
% 'first' is the number in the name of the first .dat file
% 'last' is the number in the name of the last .dat file
% 'plot_data' is optional. If plot_data='n' then the data signals will not be plotted. Else the data will be plotted.
%
% Example:
% veldat('data',1,4,'n'); Calculates the velocities from the data files 'data001.dat' till 'data004.dat' and does not plot the measured data signals from the data files.
%
% 'velocity' is a matrix that contains the calculated velocities for the contractions where a good velocity could be calculated
% 'stimtime' contains the times at which the stimulus is detected
% 'vel_mean' is the mean value of the calculated velocities
% 'vel_sd' is the standard deviation of the calculated velocities
%
% Requirements of used data-input:
% It is assumed that the measured action potential signal first has a minimum and then a maximum voltage. This should be verified with an oscilloscope. When this is not the case the polarities of the electrodes have to be switched.

% Ward van Vlimmeren
% august 2003
% TU/e; CS; Dept. Electrical Engineering

if nargin<4
    plot_data='y';
if nargin<3
    error('Not enough input arguments. See veldat.');
end

signal1=[];
signal2=[];

for i=first:last
    number=num2str(i);
s    s=size(number);
    if s(1,2)==1
        number=strcat('00',number);
    elseif s(1,2)==2
        number=strcat('0',number);
    end
    name=strcat(file,number);

    fid=fopen([name,'.dat'],'r','b');
signal=fread(fid,[2,inf],'float32');
signal1=[signal1(:); signal(2,:)'];
signal2=[signal2(:); signal(1,:)'];
end

% Determine fs (sample frequency) and ied (inter electrode distance)
fs = 32258.06; % [Hz] This is the actual sample frequency used by LabView
ied = 0.01; % [m]

time=(1/fs:1/fs:length(signal1)/fs);

% Some variables
ccerror = 0; % Correlation coefficient errors
dferror = 0; % Determine first signal errors
deleted = 0; % Ammount of manually deleted velocities
k = 0; % Index of calculated velocity
j = 1; % Index of sample of signal1
stimtime=[];
velocity=[];
% Search the action potentials
while j < length(signal1)-1100

...
if abs(signal1(j)) > 3 % Detect stimulation peak can be positive or negative
    data1 = detrend(signal1(j+100 : j+1000),’constant’);
    % Select datawindow 100-1000 samples after stimulation signal
    data2 = detrend(signal2(j+100 : j+1000),’constant’);
    % and remove offset by constant detrend
    [a1,b1] = max(xcorr(data1, data2));
    % Calculate the time delay (in samples) with maximal correlation
    sample_delay = length(data1) - b1;

% Calculate the velocity using the knowledge of which signal is % 'delayed'. The velocity is calculated from a data subset that is % selected by detecting the minimum of the first signal and the % maximum of the delayed signal. The data subset is from 100 samples % before the minimum till 100 samples after the maximum.
if sample_delay > 0 % data1 is first signal and data2 is delayed signal
    [temp,t_min]=min(data1);
    [temp,t_max]=max(data2(201:end));
    % Skip first part there can be a maximum caused by the stimulus signal
    t_max=t_max+200; % Correct index for skipping first part.
    if t_min<101
        t_min = 101;
    end
    if t_max>size(data2,1)-100
        t_max=size(data2,1)-100;
    end
    data1=data1(t_min-100:t_max+100);
    data2=data2(t_min-100:t_max+100);
    [a1,b1] = max(xcorr(data1, data2));
    % Calculate the time delay (in samples) with maximal correlation
    sample_delay = length(data1) - b1;
    if sample_delay > 0 % Check sample delay should be positive
        time_delay = sample_delay*(1/fs); % Calculate the time delay in seconds
        corc = corrcoef(data1(1:end-sample_delay),data2(sample_delay+1:end));
        if size(corc,1)==2
            if corc(1,2)>0.9
                k=k+1;
                velocity(k) = ied/time_delay; % Calculate the velocity
                stimtime(k)=time(j);
            else
                ccerror = ccerror + 1;
            end
        end
    end
% The cc’s are too small --> increase ammount of ccerrors
else
    cccerror = cccerror + 1;
    % The cc’s are too small --> increase ammount of ccerrors
end
else
    dferror=dferror+1; % Error in determination of which signal comes first
end

else  % data2 is first signal and data1 is delayed signal
    [temp,t_min]=min(data2);
    [temp,t_max]=max(data1(201:end));
    % Skip first part there can be a maximum caused by the stimulus signal
    t_max=t_max+200;  % Correct index for skipping first part.
    if t_min<101
        t_min = 101;
    end
    if t_max>size(data1,1)-100
        t_max=size(data1,1)-100;
    end
    data1=data1(t_min-100:t_max+100);
    data2=data2(t_min-100:t_max+100);
    [a1,b1] = max(xcorr(data2, data1));
    % Calculate the time delay (in samples) with maximal correlation
    sample_delay = length(data1) - b1;
    if sample_delay > 0    % Check sample delay should be positive
        time_delay = sample_delay*(1/fs); % Calculate the time delay in seconds
        corc = corrcoef(data2(1:end-sample_delay),data1(sample_delay+1:end));
        if size(corc,1)==2
            if corc(1,2)>0.9
                k=k+1;
                velocity(k) = ied/time_delay;  % Calculate the velocity
                stimtime(k)=time(j);
            else
                cccerror = cccerror + 1;
                % The cc’s are too small --> increase ammount of ccerrors
            end
        else
            cccerror = cccerror + 1;
        end
    end
end
% The cc’s are too small --> increase amount of ccerors
    end
else
    dferror=dferror+1;  % Error in determination of which signal comes first
    end
end
j = j + 1099;   Skip next 1100 samples for peak detection
end
j = j+1;
end

if isempty(velocity)
    disp('---------------------------------------------------------');
    disp('There are no velocities calculated');
    disp(['The number of correlation coefficient errors: ' num2str(ccerror)]);
    disp(['The number of determine first errors: ' num2str(dferror)]);
    disp('---------------------------------------------------------');
else
    figure(100);
    plot(velocity,’x’);
    hold on;
    plot(velocity,’g’);
    hold off;
    title(['Calculated velocitys from ' file]);
    xlabel('sample#');
    ylabel('Velocity (m/s)');
    while input('Do you want to delete any calculated velocities? (y/n): ’,’s’)==’y’;
        deleted=deleted+1;
        j=input('Which velocity should be deleted? Enter sample number: ’);
        velocity=[velocity(1:j-1) velocity(j+1:end)];
        stimtime=[stimtime(1:j-1) stimtime(j+1:end)];
        figure(100);
        plot(velocity,’x’);
        hold on;
        plot(velocity,’g’);
        hold off;
        title(['Calculated velocitys from ' file]);
        xlabel('sample#');
        ylabel('Velocity (m/s)');
end
end

close(figure(100));

disp('---------------------------------------------------------');
disp(['The number of calculated velocities: ' num2str(k-deleted)]);
disp(['The number of deleted velocities: ' num2str(deleted)]);
disp(['The number of correlation coefficient errors: ' num2str(ccerror)]);
disp(['The number of determine first errors: ' num2str(dferror)]);
disp('---------------------------------------------------------');

vel_mean=mean(velocity);
vel_std=std(velocity);

figure;
plot(stimtime,velocity,'x');
hold on;
plot(stimtime,velocity,'g');
hold off;
title(['Calculated velocitys from ' file]);
xlabel('Time (s)');
ylabel('Velocity (m/s)');

disp('---------------------------------------------------------');
disp(['The avarage calculated velocitys: ' num2str(vel_mean)]);
disp(['with standard deviation: ' num2str(vel_std)]);
disp('---------------------------------------------------------');

end

if plot_data=='y'
    figure;
    hold on;
    plot(time,signal1);
    plot(time,signal2,'r');
    hold off;
end

%EOF
B Aanpassingen oppervlakte EMG versterker

Hieronder wordt een beschrijving gegeven van de aanpassingen die gedaan zijn aan de oppervlakte EMG versterker. Het originele ontwerp staat beschreven in: Design & development of an amplifier for surface EMG measurements; F. de Bekker; Stageverslag TU/e, faculteit Elektrotechniek, Control Systems; 2002 (Verslag nummer: 02s/05).

B.1 Ontkoppel condensatoren

Bij alle voedingen van de IC’s zijn ontkoppel condensatoren geplaatst. Deze zijn aangesloten tussen de positieve of negatieve voeding van het IC en aarde. De waarde van de condensatoren is 100 nF. Ze zijn zo dicht mogelijk bij de voedingsaansluiting van het IC geplaatst.

B.2 Laagdoorlaatfilter

Op de uitgang van de isolatieversterker (ISO122) zit een verstoring van 20 mV met een frequentie van 500 kHz. Waarschijnlijk levert dit geen grote problemen op. Om er zeker van te zijn dat hierdoor geen aliasing problemen optreden, wordt de uitgang gefilterd met een laagdoorlaatfilter.

Het gebruikte filter bestaat uit een eenvoudige schakeling van een in serie geschakelde weerstand en een condensator parallel met de aarde. De gebruikte waarden zijn: $R = 10 \, k\Omega$ en $C = 1,5 \, nF$. Dit levert een eerste orde laagdoorlaatfilter met een afsnijfrequentie van 10,6 kHz. Met deze afsnijfrequentie wordt de verstoring goed onderdrukt en de frequenties van het signaal dat gemeten moet worden, worden niet onderdrukt.

B.3 Offset

In de versterker is een schakeling opgenomen die de offset tussen de meetelektroden moet verwijderen. Het signaal dat dit doet wordt via een weerstandsdeling teruggekoppeld. Door de weerstandsdeling wordt het teruggekoppeld signaal verzwakt. Bij de oude schakeling wordt het signaal ongeveer vijf keer verzwakt.

De maximale offset aan de ingang die verwijderd kan worden is als volgt te berekenen:

- De spanning aan de uitgang van de opamp die de offset verwijdert is maximaal gelijk aan de voedingsspanning: 9 V.
- De maximaal teruggekoppelde spanning is: $9 \times \frac{22k}{(22k + 100k)} = 1,62 \, V$. 
1,62 V is de offset die maximaal aan de uitgang van de INA118 verwijderd kan worden. De offset die aan de ingang van de oppervlakte EMG versterker (is tevens ingang van de INA118) verwijderd kan worden is: $1,62 / Gain_{INA118} = 1,62 / 130 = 12 \text{ mV}$.

Uit metingen is gebleken dat de offset meer dan 12 mV is. De offset regeling moet dus aangepast worden. Dit is gedaan door de weerstandsdeling aan te passen, de weerstanden zijn met elkaar verwisseld. De verzwakking wordt dan ongeveer 5/4 keer.

Het nadeel van het aanpassen van de weerstandsdeling is dat de tijdconstante van de offset regeling groter wordt. Het gevolg hiervan is dat de offset minder snel verwijderd wordt. Om dit effect te beperken wordt de weerstand aan de ingang van de opamp die de offset regeling doet veranderd. De weerstand van 1 MΩ is vervangen door een weerstand van 10 MΩ. Voor een berekening van de tijdconstante zie het verslag van F. de Bekker. De offset regeling is nu wel iets trager dan de oude regeling, maar de offset die verwijderd kan worden is groter:

$\frac{9 \times 100k}{(22k + 100k)} / 130 = 57 \text{ mV}$

**B.4 LabView**

Tot nu toe werden de metingen gedaan met DSpace. Om flexibeler te zijn wat betreft de plaats waar de metingen gedaan worden, is ervoor gekozen om de metingen met behulp van een laptop met een NiDaq data-aquisitie kaart te doen. Voor de metingen wordt dan gebruik gemaakt van het softwarepakket LabView.

DSpace kan spanningen meten met een bereik van ±10 V. Labview kan spanningen meten met een bereik van ±5 V. De versterker is gemaakt voor DSpace en heeft daarom een uitgangsspanning met een bereik van ±10 V. Deze spanning moet met behulp van een weerstandsdeling verzwakt worden. Deze weerstandsdeling wordt gecombineerd met het laagdoorlaatfilter. Dit wordt gedaan door een weerstand van 10 kΩ parallel te zetten met de condensator van 1,5 nF. Deze weerstand is niet aangebracht in de versterker zelf, maar op het connectiebordje dat voor de meting met LabView gemaakt is. Op deze manier is de versterker ook nog geschikt om metingen met DSpace te doen.

In LabView zijn een aantal programma’s geschreven om de metingen te kunnen doen:

- Scope programma (Cont Acq&Chart (buffered).vi)
- Meet programma (ContAcqToFile.llb / ContAcqToFile.vi)
- Data omzet programma (ContAcqToFile.llb / ReadFileFromContinuousAq.vi)

Het scope programma kan gebruikt worden om de gemeten SEMG signalen op het scherm van de laptop af te beelden. Dit is handig als er geen losse oscilloscoop aanwezig is. Een
<table>
<thead>
<tr>
<th>Kleur</th>
<th>Pin#</th>
<th>Omschrijving</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rood</td>
<td>1</td>
<td>Vin1+</td>
</tr>
<tr>
<td>Oranje</td>
<td>2</td>
<td>Vin-</td>
</tr>
<tr>
<td>Geel</td>
<td>3</td>
<td>Vin2+</td>
</tr>
<tr>
<td>Groen</td>
<td>4</td>
<td>Vin2-</td>
</tr>
<tr>
<td>Blauw</td>
<td>5</td>
<td>Vin3+</td>
</tr>
<tr>
<td>Paars</td>
<td>6</td>
<td>Vin3-</td>
</tr>
<tr>
<td>Grijs</td>
<td>7</td>
<td>Vin4+</td>
</tr>
<tr>
<td>Wit</td>
<td>8</td>
<td>Vin4-</td>
</tr>
<tr>
<td>Zwart</td>
<td>11</td>
<td>Ground</td>
</tr>
</tbody>
</table>

Table 1: Aansluitingen DaqCard

Losse oscilloscoop met geheugen functie heeft echter de voorkeur omdat hiermee de SEMG signalen goed getriggerd kunnen worden en stilstaand worden weergegeven.

Het meet programma wordt gebruikt om de SEMG signalen op te slaan op de harde schijf. Er kunnen vier kanalen differentieel gemeten worden. De kanalen worden gesampled met een frequentie van ongeveer 32 kHz. De exacte frequentie is in het programma af te lezen. De kanalen worden continu gemeten en opgeslagen. De data wordt opgeslagen in het binary data formaat. De data wordt gesplitst in meerdere bestanden, de grootte van de bestanden kan in het programma ingesteld worden.

Het data omzet programma zet de binary data bestanden om naar een tekstbestanden. Deze tekstbestanden kunnen vervolgens in Matlab ingelezen worden om de data verder te verwerken. Matlab gebruikt een punt als decimaalteken en LabView gebruikt standaard een komma. Om te zorgen dat dit geen problemen oplevert moet in LabView de volgende instelling gedaan worden:
Ga naar 'Tools' 'Options';
Kies dan de 'Front panel' tab;
Zet het vinkje voor 'Use localized decimal point*' uit.

B.5 Aansluiting versterker op DaqCard

In tabel 1 staat hoe de versterker op het connectiebord van de DaqCard aangesloten moet worden. Let erop dat de weerstand van 10 kΩ tussen V_out en gnd_src zich niet in de versterker bevindt, maar op het aansluitingsbordje voor de DaqCard.
B.6  Uiteindelijke schakeling

De uiteindelijk schakeling staat in figuur 4.

![Diagram](image-url)  

Figure 4: Nieuw schema
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