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Model for arm movements during myoclonic seizures

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Abstract— **A model is formulated for arm movements during myoclonic (epileptic) seizures. The system described in the model, consists of a mechanical and an electrophysiological part. The model output is compared to real patient accelerometry (ACM)-data from six epilepsy patients. Eight out of ten myoclonic seizures have a good fit to the model. The values of the model parameters tuned to the real seizures are physiologically feasible. Using mean parameter values leads to agreeable fits in six out of ten myoclonic seizures. Two of the four parameters seem to be robust for variation in patient and seizure. The presented model approach leads to a better understanding of patterns in ACM-recordings that are associated with myoclonic seizures and in the future can contribute to automated detection of these patterns.**

I. INTRODUCTION

In this paper an analytical model is formulated for accelerometric output associated with myoclonic seizures. A myoclonic seizure consists of one single muscle jerk, with a spike-wave as an EEG-correlate. The electrical activation of the muscle lasts less than 50 milliseconds [1]. The agonists and antagonists in the muscle groups involved contract and relax synchronously. A myoclonus can occur synchronously throughout the body but often only one limb is involved. A myoclonus can occur isolated but can also occur in series or can be followed by other seizure types.

The clinical manifestation of myoclonic seizures is very subtle hence they are often missed by current available detection systems. Detecting these subtle seizures is of clinical importance. A patient can experience many myoclonic seizures during the night that can disturb sleep rhythm. Counting myoclonic seizures may also be an important measure for successful medical treatment. Furthermore, severe motor seizures are often preceded by myoclonic seizures, thus detection of myoclonic seizures could be used for early warning.

Myoclonic seizures are associated with clearly visible stereotypical patterns in accelerometry (ACM) signals [2]. In choosing suitable features for automated detection of these seizures from the ACM-signal, knowledge about these patterns is important. The suggested model can be used to study important characteristics of accelerometric waveforms associated with myoclonic seizures. It can contribute to a better understanding of the patterns that are observed in the ACM-recordings. Hence, the model makes it also possible to derive parameters from the ACM-signal that have a physiological meaning. Therefore this model-based approach can contribute to a robust detection of myoclonic seizures.

The system described in the model consists of a mechanical and an electrophysiological part. The electrophysiological part contains the definition of stimuli and a muscle response to these stimuli during a myoclonic seizure. The mechanical part is based on kinematic and kinetic relations for the lower arm, modeled as a rigid body system. The model output is compared to real patient accelerometry data. The sensitivity of the parameter settings is studied in order to get an indication whether the model is robust across patients and seizures.

II. METHODS

A. Model overview

During a seizure the distal segments of the limb are more affected than the proximal ones, and arm movements are dominant over leg movements. Video observations confirm that myoclonic seizures very often manifest themselves as short abrupt flexions involving only the lower arm. It is also frequently observed that a seizure starts with myoclonic jerking in one arm followed by a spread of tonic, clonic or tonic-clonic contractions towards the other arm, the trunk and the legs. Based on the above statements and observations, it was decided to include only the forearm in the model for myoclonic seizures. Figure 1 is a schematic overview of all

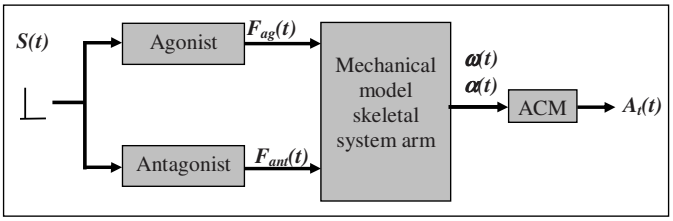

Fig. 1. Schematic representation of model.

the systems incorporated in the model. The central nervous systems sends signals to the muscles. The innervation patterns or stimuli $S(t)$ are for a myoclonic seizure modeled as a pulse. The muscles react to the innervation patterns of the central nervous systems and apply force on the skeletal system. For evaluation of the movement model, the choice was made to registrate movements with accelerometers, thus in the model also the accelerometer output corresponding to the movements is calculated.

B. Innervation patterns during myoclonic seizures

During a myoclonic seizure a spike-wave (or a poly spike-wave) pattern is visible in the EEG signal. It is believed

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that during the spike part of the spike-wave discharge, trains of action potentials are sent down to the motor units and the muscles contract in reaction to this stimulation [3]. The agonists and antagonists in the muscle groups involved contract synchronously. During the wave part there is an inhibition, and no action potentials are descending to the motor units. This results in complete relaxation of all the muscles involved. The EMG activation during an epileptic myoclonus is < 50 ms [1]. Based on the above observations it was decided to model the innervation pattern during a myoclonic seizure as a pulse.

C. Muscle contraction during seizures

In literature [4], [5], many complicated muscle models are described. Most of these models go deeply into the muscle's structure and physiology. To use such an extensive model would go far beyond the scope of this paper. The choice was made to use a simple approximation for muscle force in time, based on the response of a motor unit to one single stimulation pulse [6]:

$$F(t) = F_0 \frac{t}{\tau_0} e^{\frac{-t}{\tau_0}} \ . \tag{1}$$

This simplification is based on the fact that during a seizure the muscles are much more stimulated than under normal circumstances. One burst of epileptic activity can cause a sudden jerk of a limb. The entire muscle consists of many motor units. During the myoclonus they are all activated synchronously. Thus, the impulse responses of all the motor units should be added together. For simplicity it is assumed that during a myoclonic seizure the impulse response of all the activated motor units together can then be approximated by: $F(t) = F_{sum} \frac{t}{\tau_{sum}} e^{\frac{-t}{\tau_{sum}}}$, with F_{sum} the weighted sum of all the F_0's of all the different motor units, and τ_{sum} a general time constant for al the units together. An advantage of the use of this impulse response is that it is possible to simulate physiological-like muscle responses to different types of stimuli.

In the model one agonistic muscle pair is included that is synchronously innervated during the seizure [1]. The muscle force of the agonist muscle $F_{ag}(t)$ is modeled as:

$$F_{ag}(t) = F_{sum} \frac{t}{\tau_{sum}} e^{\frac{-t}{\tau_{sum}}} \ . \tag{2}$$

It is necessary to create an alternating positive and negative netto muscle movement, to generate the typical myoclonic 'shock-like' pattern. Therefore for modeling the antagonist muscle force $F_{ant}(t)$ a similar equation would yield, but with different values for F_{sum} and τ_{sum}:

$$F_{ant}(t) = F_{sum_{ant}} \frac{t}{\tau_{sum_{ant}}} e^{\frac{-t}{\tau_{sum_{ant}}}} \ . \tag{3}$$

Equation 3 can be expressed in F_{sum} and τ_{sum} by:

$$F_{ant}(t) = \frac{1}{A} F_{sum} \frac{t}{\tau_{sum}} e^{\frac{-t}{B\tau_{sum}}} \ . \tag{4}$$

where A and B –as we shall see in section III– are dimensionless constants approximately equal to 1.

D. Mechanical model of the skeletal system of the arm

The most dominant element in a myoclonic seizure is the flexion of the elbow. The elbow is modeled as a hinge joint that is fixed at its position. This means that the movement of the wrist is a pure rotation around the elbow axis, a two-dimensional planar movement. Figure 2 shows the rigid body that represents the lower arm and the hand. The box is the fixed accelerometer that measures acceleration components in the tangential (A_t) direction. The forces that act on the rigid body are the agonist muscle force (F_{ag}), the antagonist muscle force (F_{ant}), and the joint reaction force (F_j). The length of the rigid rod is represented by L.

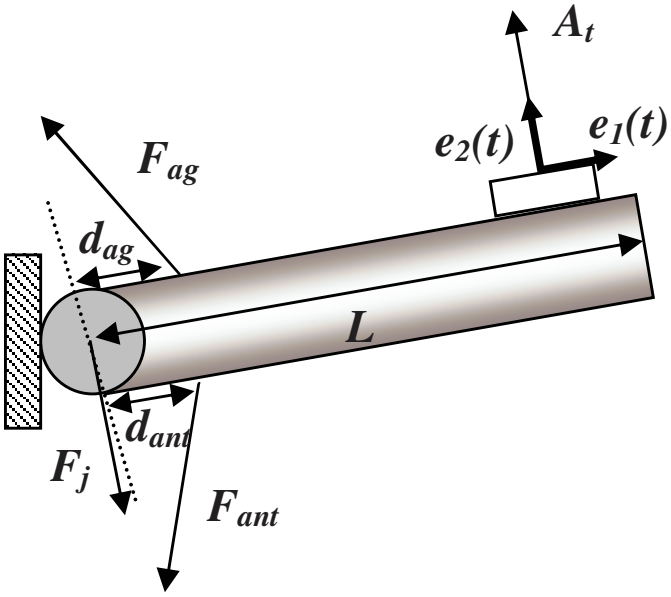

Fig. 2. Schematic overview of the mechanical part of the model.

1) Kinematic relations for the rigid body system: The kinematic relation for position $x(t)$, during a 2-D rotation in a time-dependent, moving frame of reference $\{\mathbf{e}_1(t), \mathbf{e}_2(t)\}$, is:

$$\mathbf{x}(t) = R\mathbf{e}_1(t) \ , \tag{5}$$

with R, the distance of the elbow to the accelerometer. The corresponding velocity $v(t)$ is:

$$\mathbf{v}(t) = \omega R\mathbf{e}_2(t) \ , \tag{6}$$

where $\omega(t)$ is the angular velocity of the moving frame. The corresponding acceleration $a(t)$ is:

$$\mathbf{a}(t) = \alpha R\mathbf{e}_2(t) - \omega^2 R\mathbf{e}_1(t) \ , \tag{7}$$

where $\alpha(t)$ is the angular acceleration of the moving frame. This means that during a pure rotation, acceleration A_t (at the position of the accelerometer) in the tangential direction $\mathbf{e}_1(t)$ equals αR. The acceleration A_n in the normal direction $\mathbf{e}_2(t)$ equals $-\omega^2 R$.

2) Kinetic relations for the rigid body system: When there is an input force, the sum of all moments can be calculated by multiplying all the tangential components of the acting forces with their moment arms. The muscle forces described in section II-C are linked to Eq. 7 by Eq. 8:

$$\Sigma \mathbf{M} = \mathbf{I}\alpha \ , \tag{8}$$

where I is the mass moment of inertia about a parallel z-axis through the fixed rotating point. For the model in this paper this means that:

$$\Sigma \mathbf{M} = F_{ag}\perp \cdot d_{ag} - F_{ant}\perp \cdot d_{ant} = I\alpha \ . \tag{9}$$

The joint reaction force F_j, acts on the fulcrum and does not contribute to the sum of moments.

Since the output of interest is α, Eq. (9) is rewritten:

$$\alpha(t) = \frac{F_{ag}\perp \cdot d_{ag} - F_{ant}\perp \cdot d_{ant}}{I} \ . \tag{10}$$

For a rigid rod, of length L, rotating around one end, the moment of inertia is constant and equal to $\frac{1}{3}mL^2$, with m the mass of the lower arm. The length L and the mass m of the lower arm, can be expressed in terms of full body length (BL) and full body mass (BM) using anthropometric data [6]. Using average values from literature [7] for d_{ag} and d_{ant} Eq. 10 can be rewritten as:

$$\alpha(t) = \frac{4.5}{BM\ BL^2}(F_{ag} - F_{ant}) \ . \tag{11}$$

The measured output of the ACM-sensor caused by the myoclonic seizure equals αR. The actual accelerometer output measures also an acceleration component caused by gravity. During such a subtle movement, the displacement of the arm is very small and thus the rotation with respect to the gravity field can be neglected. Therefore for comparing simulated movements with real accelerometer output, Eq. 11 needs to be multiplied with R, the distance from the elbow to the wrist, that is equal to $0.146BL$ [6]. Thus the ACM-pattern observed during a myoclonic seizure is of the shape:

$$A_t(t) = K\left(te^{\frac{-t}{\tau_{sum}}} - \frac{t}{A}e^{\frac{-t}{B\tau_{sum}}}\right) \ , \tag{12}$$

where constant $K = \frac{0.66}{BM\ BL}\frac{F_{sum}}{\tau_{sum}}$.

E. Comparison of model to real data

Equation 12 was fitted to real patient data with an optimization algorithm in MATLAB.

ACM-data are used from six mentally retarded patients who suffer from severe epilepsy. This data is described in [8]. In order to get an indication whether the model needs to be tuned for every patient or seizure, the sensitivity of the parameter settings is studied.

III. Results

Figure 3 shows real accelerometer output compared to the modeled myoclonic seizures. Table I shows for every myoclonic seizure the values of K, τ_{sum}, A, B, F_{max}, and the correlation coefficient R^2 between the modeled myoclonic seizure and the corresponding real myoclonic seizure. F_{max} is determined by filling out $t = \tau_{sum}$ in Eq. 2. F_{max} represents the maximal muscle force applied to the arm by the agonist muscle. Eight out of ten myoclonic seizures have a correlation coefficient > 0.7, with a p-value $<< 0.01$. Myoclonus no. 6 and 9 have a poor fit. No. 6 is not a single muscle twitch, but there is a more repetitive movement visible in the ACM-signal. This is also the case for No. 4 although in this case the fit to the first part of the repetitive pattern is good and the correlation coefficient is 0.73. The shape of No. 9 seems to consist of two subsequent twitches. The values of τ_{sum} are physiologically meaningful. The contraction time τ for motor units in the arm are in the range of 16–68 ms for the triceps and in the range of 16–85 ms for the biceps [6]. Also the values of F_{max} are physiological feasible forces. The values of A and B are all > 1. A needs to be > 1 since the arm first flexes during a myoclonic seizure. This means that when a seizure starts the force delivered by the agonist is larger than the force delivered by the antagonist. This phenomenon can possibly be explained by the fact that the projection area of the motor cortex through the pyramidal tract that belongs to the agonist muscles is larger [9]. For generating an alternating positive and negative netto muscle movement $A = B^2$. Therefore also $B > 1$. From the simulation

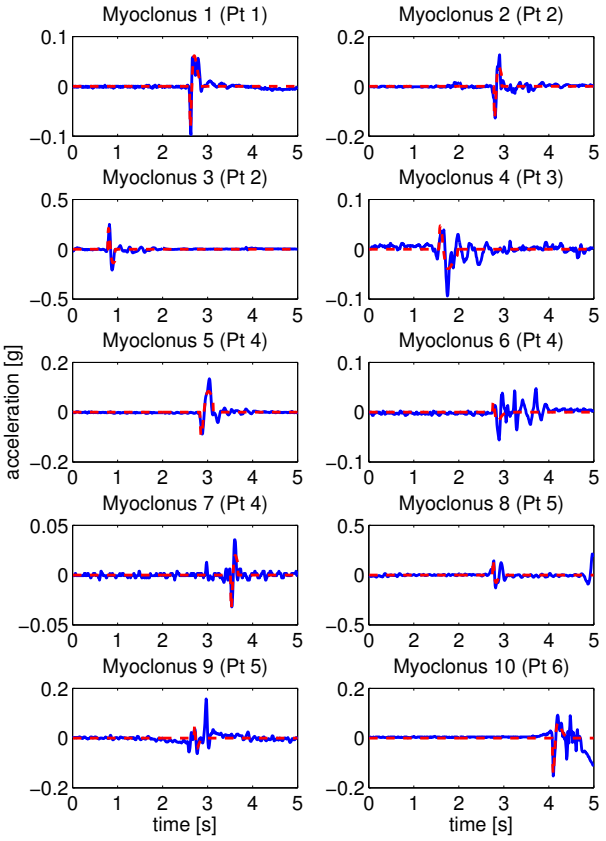

Fig. 3. Simulation results (red dashed line) compared to real ACM-data (blue solid line). 'Pt #' indicates the patient where the data is from.

results we can also observe that $A \approx B^2$.

TABLE I
PARAMETERS OF MODEL FITTED TO REAL DATA

Myocl.	K [N/(kg s)]	τ_{sum} [s]	A	B	$F_{max}[N]$	R^{2*}
1	47.8	0.035	1.025	1.017	96	0.88
2	49.9	0.035	1.036	1.022	131	0.88
3	67.8	0.040	1.038	1.024	203	0.85
4	30.7	0.070	1.011	1.008	116	0.73
5	32.3	0.065	1.024	1.017	92	0.88
6	34.5	0.038	1.109	1.006	58	0.34
7	32.9	0.034	1.015	1.009	49	0.83
8	81.6	0.024	1.038	1.025	56	0.71
9	11.6	0.022	1.114	1.076	7	0.51
10	36.9	0.039	1.046	1.025	74	0.91
Mean	42.6	0.040	1.045	1.023	88	0.75

* with a p-value $<< 0.01$

To study the robustness of the model and the sensitivity of the parameters, the correlation coefficients between the real data and a simulated myoclonus with the mean values of Table I filled in for the parameter settings are calculated. This leads to correlation coefficients > 0.8 in 6 myoclonic seizures. The fit was poor for myoclonus No.'s 4, 6, 8, and 9. No.'s 6 and 9 already had a bad fit. In Table I No. 4 has a relatively large value for τ_{sum} compared to the other waveforms, therefore the correlation to the mean parameters is poor. In Table I No. 8 has a relatively large value for K compared to the other waveforms, hence the correlation to the mean parameters is also poor. Keeping all the parameters fixed at the mean value and only the value of K to be optimized leads to similar results. Table I for both the correlation coefficient and K. Keeping all the parameters fixed at the mean value and only the value of τ_{sum} to be optimized leads to poor fits with erratic values of τ_{sum} in 5 cases. Keeping only A and B fixed, and the values of K and τ_{sum} to be optimized leads to similar results as those presented in Table I. Therefore A and B might be fixed across patients, and appear to be typical for myoclonic seizure movements.

IV. DISCUSSION AND CONCLUSION

This paper presents an analytical model for arm movements during myoclonic seizures. The model output is compared to real patient accelerometer data from six patients. The values of the model parameters tuned to real seizures are physiologically feasible. Eight out of ten myoclonic seizures have a good fit to the model (correlation coefficient $>$ 0.7). The ACM-pattern associated with myoclonic seizures is typical [2], although some myoclonic seizures are somewhat longer in duration and have a more repetitive pattern (No. 4, 6, and 9). Maybe in these cases the neural input modeled as a pulse does not yield, but the input should be represented by a series of pulses. This corresponds with the fact that a myoclonic seizure can have either a spike and wave correlate in the EEG or a poly-spike and wave correlate [1].
Using mean parameter values leads to agreeable fits in six out of ten myoclonic seizures. The results imply that some of the parameters might be robust for patient and seizure variation. Further research will be done, with larger amounts of data to refine and optimize these findings.
By choosing other types of stimuli for an input, the model has the possibility to be extended for other types of simple motor seizures (clonic and tonic seizures). Clonic seizures consist of repeated myoclonic contractions that regularly recur at intervals between 0.2 and 5 times per second [10]. They have series of (poly)spike-wave patterns as an EEG-correlate. The ACM-pattern is rhythmic and the limbs show repetitive jerking. Tonic seizures consist of sustained muscle contractions, and the EEG shows fast frequency activity. The ACM-pattern has a typical block-like shape.
The presented analytical model can be helpful for feature extraction for detection of myoclonic waveforms from accelerometer signals. The model can be used to study important characteristics of accelerometric waveforms during myoclonic seizures. It contributes to a better understanding of the patterns that are observed in the ACM-recordings, and the model makes it possible to derive parameters from the ACM-signal that have a physiological meaning. Therefore this model-based approach can contribute to a solid detection of myoclonic seizures.

REFERENCES

[1] M. Hallett, Myoclonus: Relation to Epilepsy, *Epilepsia*, vol. 26, Suppl. 1, 1998, pp. S67–S77.
[2] T. M. E. Nijsen, J. B. A. M. Arends, P. A. M. Griep, P. J. M. Cluitmans,The potential value of 3-D Accelerometry for detection of motor seizures in severe epilepsy, *Epilepsy and Behavior*, vol. 7, pp. 74-84, 2005.
[3] H. M. Hamer, H. O. Lüders, S. Knake, B. Fritsch, W. H. Oertel and F. Rosenow, Electrophysiology of focal clonic seizures in humans: a study using subdural and depth electrodes, *Brain*, vol. 126, 2003, pp. 547–555.
[4] M. Epstein and W. Herzog, Theoretical models of skeletal muscles: Biological and mathematical considerations, Wiley, Chichester, 1998.
[5] J. M. Winters, Multiple muscle systems : biomechanics and movement organization, Springer, 1990.
[6] D. A. Winter, Biomechanics and motor control of human movement,Wiley-Interscience, Chichester, 1990.
[7] H. Veeger, B. Yu, K. N. An, R. H. Rozendal, Parameters for modelling the upper extremity, *J. Biomechanics* vol. 30, 1997, pp. 647–652.
[8] T. M. E. Nijsen, R. M. Aarts, P. J. M. Cluitmans, P. A. M. Griep, Time-frequency analysis of accelerometry data for detection of myoclonic seizures, submitted to IEEE TBME, 2007.
[9] P. E. Voorhoeve, Physiologie van het centrale zenuwstelsel en de zintuigen, N.V. Noord-Hollandsche Uitgeversmaatschappij, Amsterdam, 1968.
[10] H. O. Lüders and, S. N. Noachtar, Epileptic Seizures, Pathophysiology and Clinical Semiology, Churchill Livingstone, New York, 2000.