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**EXPERIMENTAL OBSERVATION OF THE  
PROPORTIONAL EFFECT HYPOTHESIS OF THE  
KINEMATIC THEORY : PRELIMINARY REPORT**

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# **Experimental Observation of the Proportional Effect Hypothesis of the Kinematic Theory: Preliminary Report**

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*Experimental Observation of the Proportional Effect Hypothesis of the Kinematic Theory:  
Preliminary Report*

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**Abstract**—For more than ten years, the Kinematic Theory has been exploited successfully in applications dealing with handwriting processing. The theory uses Delta-Lognormal profiles to model the behaviors of the motor control and the neuromuscular system involved in the production of rapid movements. Its main underlying assumption is that the cumulative time delays of the propagated impulse responses, recorded along different points on a neuromuscular system are linked by proportional relationships. The study reported in this paper aims at using the physiological knowledge of the upper arm to deeply investigate this assumption. First, the concept of a subsystem is clarified and justified. Then, to observe the proportionality hypothesis, surface electromyographic signals (SEMG) recorded from the upper limb, during the execution of rapid handwriting movements, are analyzed. The results reveal proportional regressions, with strong correlation, between the cumulative time delays of the muscle activities, calculated from SEMG envelopes. This finding highlights the relationships that exist between the macro-delays observed on the SEMG bursts and constitutes an observation proof of the proportionality hypothesis. These developments add further support to the usefulness of the Kinematic Theory as a tool for the study and understanding of human movement.

**Index Terms**— Kinematic Theory, rapid hand movements, proportional effect, EMG, Delta-Lognormal model, Weber's law.

## 1. Introduction

For many years, our interest has been devoted to the knowledge of how humans control their complex movements; particularly, how the motor control and the neuromuscular system are involved when someone is producing a point-to-point rapid movement of his upper limb end-

effector. This interest aims at studying the most physiological paradigms observed through experimental data and offering simple models which provide both the analysis and synthesis of specific movements, and the prediction of others. It has long been known that the velocity profile of rapid movements presents a bell-shaped pattern (vg. [34],[36],[43],[45],[46],[48] ) and to study this stereotyped profile, many theories have been proposed (vg. [35],[37-42],[44],[47],[49-51]). The Kinematic Theory is one of them which describe the stereotyped velocity profile with a Delta-Lognormal model that is based on a few fundamental concepts and assumptions [22],[23],[25],[26].

According to this paradigm, the rapid movement of an end-effector mainly results from the interaction between the actions of an agonist and antagonist neuromuscular systems controlled by the central nervous system (CNS). In a functioning point of view, each of these systems is considered to be activated by a Dirac-Impulse  $U_0(t-t_0)$  shifted in time by  $t_0$  and weighted by a command  $D_i$ , where the subscript  $i = 1, 2$  stands for the agonist and antagonist systems respectively. The propagation of these commands from the brain to the end-effector is characterized by transient responses that are time delayed and governed by a proportional effect, expressed by:

$$T_j = (1 + \varepsilon_j) T_{j-1} \quad (1)$$

where  $T_j$  and  $T_{j-1}$  are respectively the cumulative time delays of the responses measured at two subsystem outputs, and  $\varepsilon_j$ , a proportionality factor. Applying the Central Limit Theorem (CLT) to the convolution of an infinite number of coupled linear subsystems, under the proportionality hypothesis, the impulse responses of the agonist and the antagonist system converge toward lognormal profiles characterized by their logtime delays  $\mu_i$  and their logresponse times  $\sigma_i$

where  $i = 1, 2$ . The stereotypical velocity profile is thus described by a Delta-Lognormal equation [22]. The distance covered by the end-effector during a movement is controlled by  $D_1 - D_2$  and the corresponding movement time  $MT$  is a function of  $D_2/D_1$ . Since this ratio is linked to the relative spatial accuracy  $\frac{\Delta D}{D}$  requested for the movement, various speed-accuracy tradeoffs are possible [22-25].

Through curve fitting, statistical analysis and mathematical demonstrations [1-2],[9][10], [21-22], it was shown that most of the models aimed at modeling the velocity profile of a rapid movement were less adequate than the Delta-Lognormal one. Some of these models (i.e. Beta, Gamma, Minimum-Jerk and Minimum-Time models) can even be considered as analytical approximations of the Delta-Lognormal equation [7][8]. The Delta-Lognormal model is based on a representation scheme and a main hypothesis both inspired from physiological observations: the notion of system and subsystem currently used in the modeling of biological systems and the proportionality effect, also known as a Weber's law. This study aims at deeply investigating these two features. In the following text, an overview of rapid movement genesis is presented in section 2. The concept of a subsystem is treated in section 3 while the methods are described in section 4. The pretreatments and the analysis of data are reported in section 5. The discussion and the conclusion are presented in section 6.

## 2. Rapid Movement Genesis

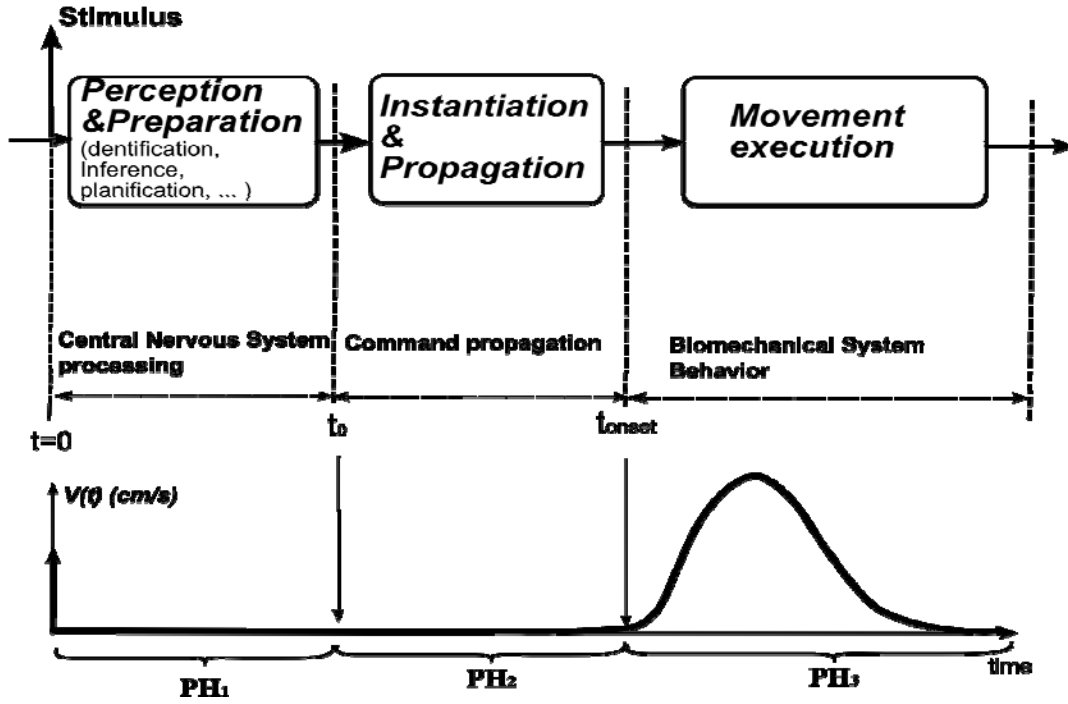
Large handwriting strokes have been used in this study as typical instances of rapid movements. Indeed, when a writer is asked by an external visual or audio stimulus to produce a rapid stroke between two points on a digitizer surface with a stylus in his hand, several processes



are occurring in his central nervous system to generate an appropriate set of commands which will propagate in the peripheral neuromuscular system (PNS) to produce the planned motor task. The movement of the end-effector can be seen as the response of the PNS to neural commands, planned and launched in from the CNS.

Indeed, the patterns appearing during the execution of particular movements can be interpreted as special behaviors of the CNS in the motor control of the PNS and then the functioning of such biological living systems can be modeled. According to the Kinematic Theory, the time-based description of the movement genesis is made up of three phases [26]. The first one  $PH_1$  corresponds to the perception of the stimulus by the subject and the preparation of the adequate neuromotor commands in the CNS level, the second phase  $PH_2$  depicts the instantiation and propagation of the commands along the PNS and the third phase  $PH_3$  refers to the effective movement execution. As one can see in Figure 1, where upward arrows indicate the stimulus onset, the rapid movement, represented by its velocity profile, occurred in the two last phases, while the first phase introduces an only pure time shift in the velocity.

Thus, a stimulus which occurred at  $t=0$  and is processed in  $PH_1$ , leads to the generation, after a time occurrence  $t_0$ , of appropriate impulse neural commands launched at  $t_0$  to the neuromuscular network. A rapid movement may thus be seen as resulting from shifted impulse commands  $U_0(t-t_0)$  propagating along the neuromuscular system toward the end-effector. Thus, the PNS can be considered as a biological communication channel which transmits, transduces (into mechanical movement) and decodes (from pulse-frequency modulated action potentials to muscle bursts) the neuronal input command, hereafter producing as output a biomechanical response. From the generation of the commands in the brain to the end-effector displacement, numerous concerned populations of neurons and many muscles are then involved.



**Figure 1.** The three phases of a rapid movement generation from the stimulus onset to the end of the movement. The time origin is at  $t = 0$ , the command occurrence at  $t_0$  and the movement onset at  $t_{onset}$ .

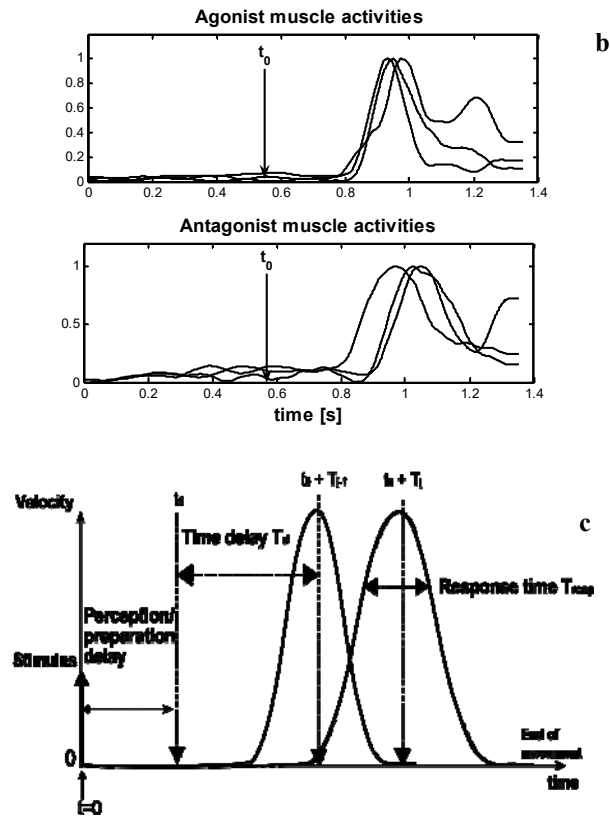
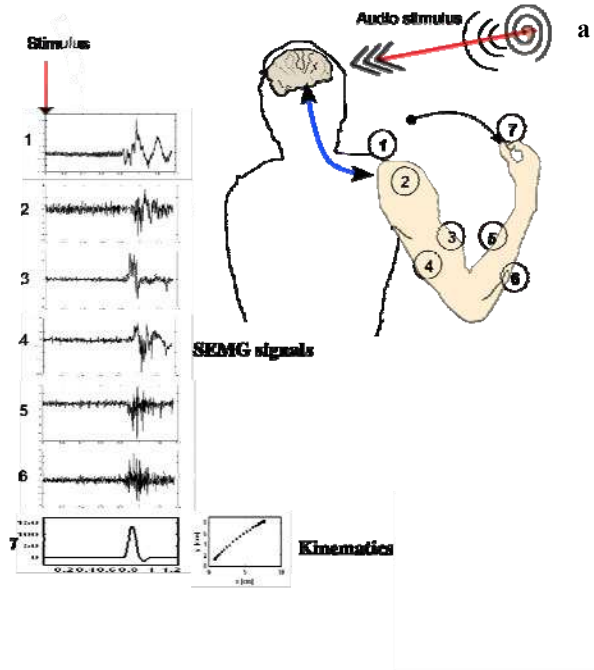
According to the physiological descriptions of neural and muscular networks, the recruited components can thus be represented as a hierarchical and parallel architecture [4],[28],[30].

The first physiological behavior, considered by the Theory, concerns the agonist/antagonist synergy. Indeed, the displacement of the end-effector is controlled by the superposition of various muscle contractions occurring at different delays and in different directions. The superposition of these contractions leads to the agonist and antagonist effects. In rapid movements, accomplished in straight-line or with a slight curved trajectory, the agonist muscles initiate and produce a movement in the desired direction while the antagonist muscles are later put in action to stop the movement. Thus, the system involved in a rapid movement of the upper arm can be modeled by an agonist and antagonist neuromuscular system. While this latter system has non-

linear components, it can be described by the convolution product of a large number of subsystems working around their linearity operating points [24].

The second physiological behavior concerns a sequential functioning of a hierarchical and parallel neuromuscular system. When the force produced by a muscle has to be increased, additional motoneurons have to be recruited to meet the demand. The recruitment is done from small motoneurons units producing low-level force to large motoneurons units producing large forces. That feature of the CNS which has been termed the “size principle” [12-13], introduces micro-delays in the recruitment of motor units. At the muscles level, the accumulation of these micro-delays leads to macro-delays as can be observed in surface electromyography (SEMG) particularly when activities of agonist and antagonist muscles are considered [3],[11],[14].

In the Delta-Lognormal model, basic kernel of the Kinematic Theory, these macro-delays are taken into account by considering the cumulative time delays of the impulse responses recorded at the output of the coupled subsystems. Particularly, the asymmetry observed in the stereotyped velocity patterns of rapid movements can be explained by the existence of a proportional relationship between the cumulative time delays of adjacent subsystems (the output of one being the input of the other) [22],[26]. As illustrated in Fig.2a, the propagation of the impulse command along the neuromuscular system can be observed through SEMG signals recorded at different points on the upper limb. A typical sample of delays between agonist and antagonist muscles during a rapid movement is depicted in Fig.2b while the notion of cumulative time delay of adjacent subsystems impulse responses is illustrated in Fig.2c. Such a setup will be used in the investigation of the notion of a subsystem and the hypothesis of a proportionality effect.



**Figure 2.** (a). Illustration of the generation of a rapid movement of the upper limb characterized by kinematic data. From the brain, effects of the propagation of the command producing the movement can be observed along the upper limb through surface electromyographic (SEMG) signals. (b) Sample of linear envelopes of such signals grouped as agonist and antagonists (c) Illustration of the time delay and response time and the concept of the proportionality effect of two adjacent subsystem impulse responses.

### 3. Concept of a subsystem

In the Kinematic Theory, the concept of a subsystem is used among other considerations to quantify the micro-delays introduced by the neuromuscular components when the nerve impulse relating to the neural command of motor tasks propagates along a complex pathway, made up of different synapses and muscles. Indeed, various mechanisms are occurring during this propagation such as a reflex mechanism of antagonist muscles, etc. and each of them create micro-delays which shift the biomechanical effect on the end-effector of the muscle contraction. The effect is located both in time (time occurrence of muscle activation) and in space (muscle position in the limb). Through a learning process, the control of these micro-delays leads to an organized pattern of the PNS, which allows the anticipation and the production of smooth and skilled movements. This theory, which deals with the macroscopic behavior of the motor system, links the motor control of a rapid movement to a high-level recruitment of motor units needed for the corresponding motor tasks. In this process, when no movement is observed on the end-effector, the time used to prepare the neural commands is represented by a pure delay  $t_0$ , a time occurrence when simultaneous activations, modeled by a shifted Dirac-Impulse  $U_0(t - t_0)$ , of the agonist and the antagonist neuromuscular networks are taking place. The low level organization of the pathway, where the recruited components are connected to propagate the nerve impulse, represents a specific configuration of a neuromuscular network. From this consideration, the relative micro-delays are not embedded in a motor control level but constitute the intrinsic behavior of a neuromuscular system. In this context, the Kinematic Theory has then characterized the functioning of a PNS by the time-based parameters ( $\mu_i$  and  $\sigma_i$ ).

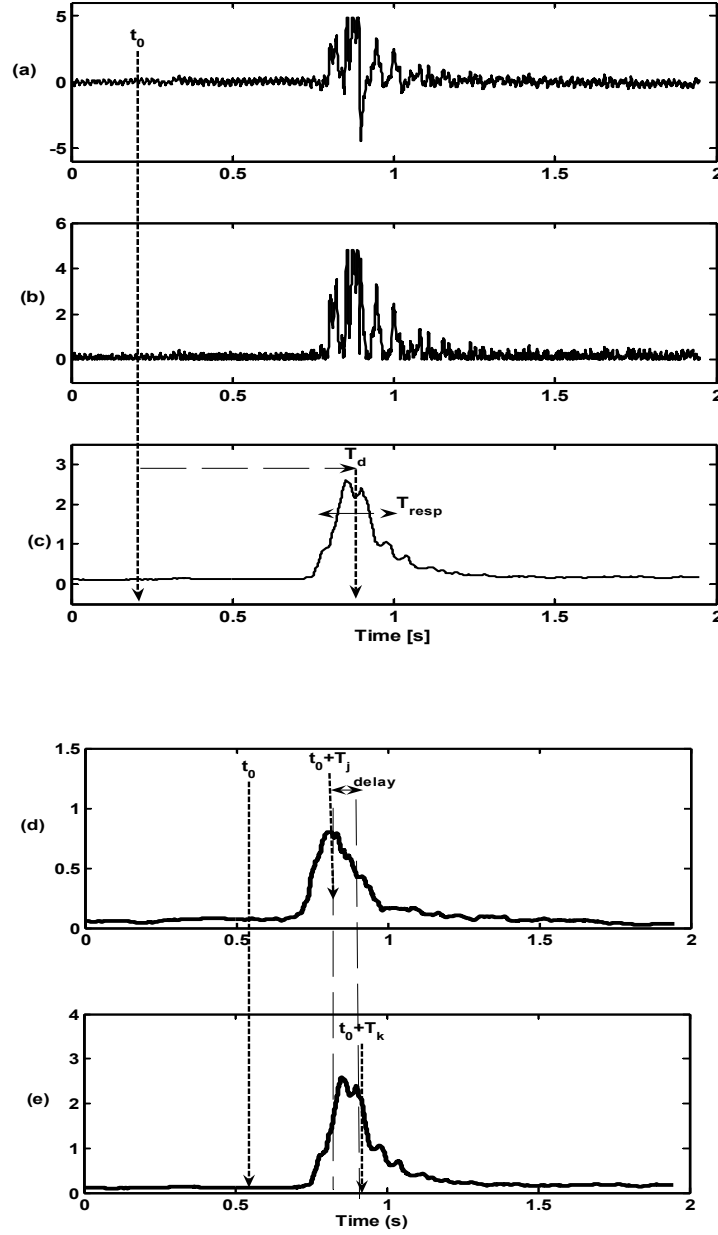
To illustrate this, let consider a case when a muscle produces an elementary movement on the end-effector, its activity can be observed through SEMG (Fig 3a-c). The bell-shaped burst of the signal results from the summation of the action potentials propagating in the muscle fibers that are set in action to produce the movement (Fig. 3c). This bell-shaped response can be characterized by its time delay  $T_d$  and its response time  $T_{resp}$ , which correspond respectively to the average time taken by the recruited muscle fibers population to respond to the CNS command, and to the duration of this response.

The delay between the biceps and the triceps activities is easily observed in Fig.3d-e from the corresponding bursts [3],[4],[11],[17]. From the time occurrence  $t_0$ , the stimulus effect occurring in  $PH_2$  can be modeled by a shifted Dirac impulse command  $U_0(t-t_0)$  propagating from the cortical cells to the descending fibers connected to the motoneurons and to the muscles fibers leading to a muscular contraction. The bursts in Fig.3d-e can be seen as the impulse responses of two neuromuscular pathways. One can thus interpret the relative time shift between SEMG bursts as a delay resulting from the accumulation of many shorter delays associated with the various neuromuscular elements.

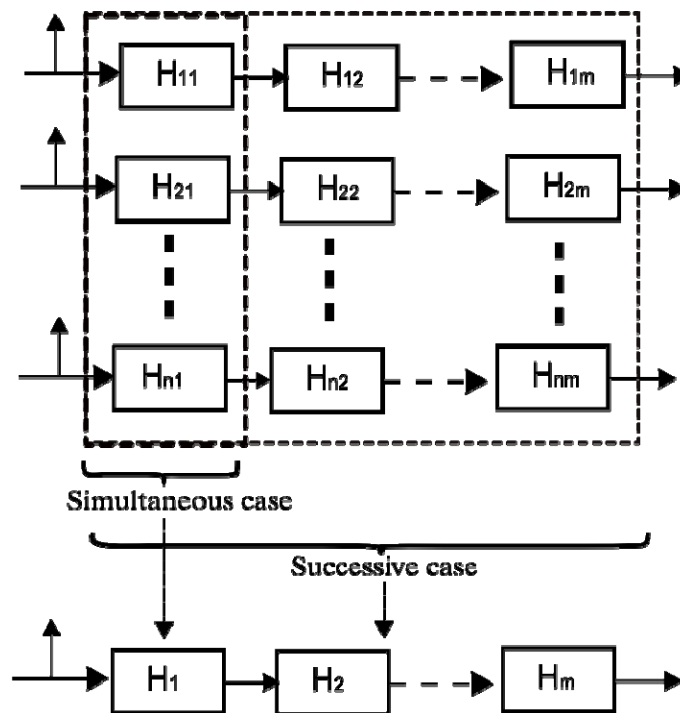
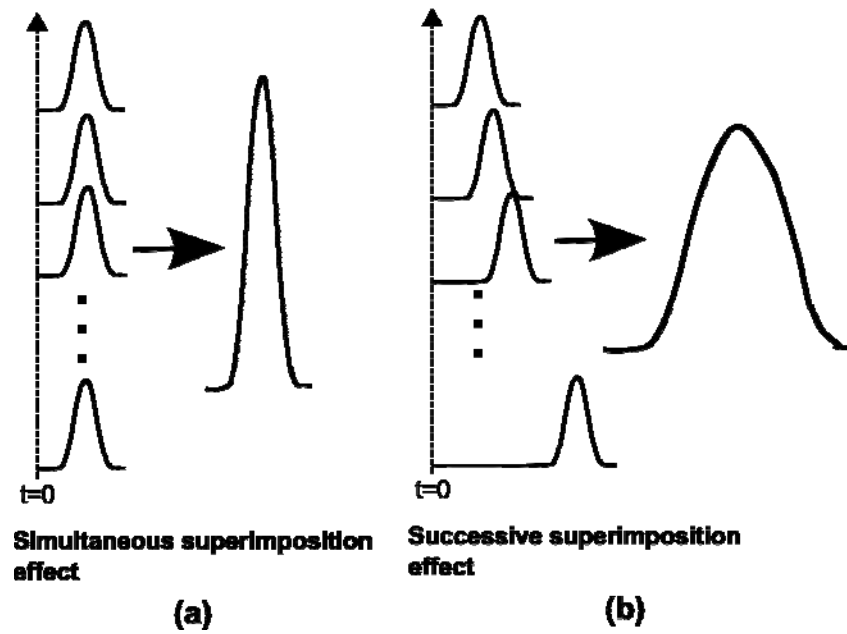
If  $n$  similar ensembles connected in an hierarchical and parallel architecture are considered, the activity of the whole population can be modeled by the activity of a single ensemble weighted by its contribution to the amplitude on the recorded signal and the time delay of the response of such an ensemble can be associated to a single delay when all the ensembles are *simultaneously* activated (Fig.4a).

When similar muscles ensembles are *successively* activated, their total activity sum up in time and a large burst can appear in the SEMG (Fig.4b). In this population, the change of the time

scale can be viewed as a translation and a dilation of an individual pattern response which can be modeled by the convolution product of a large number of subsystem impulse responses.



**Figure 3:** Typical example of the apparition of a bell-shaped burst of SEMG signal. (a) Original SEMG signal recorded on a Biceps brachii muscle, (b) its rectified version and (c) its envelope as calculated by a Savitsky-Golay low-pass filter [29]. Associated with a rapid end-effector movement, typical example of EMG envelopes recorded on (d) the Triceps (d) and on the Biceps (e). The command to initiate the movement was at  $t_0$ .



**Figure 4:** Illustration of (a) a superposition where simultaneously active processes have the same time delay and response time (b) a superposition of processes presenting an increased time delay (c) both the simultaneous superposition effects (column wise: individual magnitudes are summed up as in (a)) and, the successive superposition (row wise) where time delay, response time and magnitude are changing.

When both simultaneous and successive recruitment of muscle fibers occurs to produce a rapid movement (Fig.4c), the total effect can be modeled by the convolution product of subsystem im-



pulse responses, where the simultaneous active muscle fiber ensembles is represented by the command amplitude and where the successive recruitment of muscles ensembles affects both the time delay and the response time of the global response. At the output, all the parallel subsystems working agonistically and those working antagonistically can be represented by a single system embedding all the subsystems.

Thus, the subsystem concept used in the Kinematic Theory to model the behavior of a neuromuscular system during the execution of a rapid movement can be seen as describing the functioning of motoneuron populations and muscular fiber ensembles, characterized by a bell-shaped impulse response, which contributes to a global movement. The effect of a stimulus, as recorded at a given location on the upper limb (SEMG signals), can be seen as the output response of a given system made up of  $n$  subsystems whose time delay corresponds to the cumulative time delays caused by these  $n$  subsystems. Even if two subsystems are not adjacent (i.e. considered at two arbitrary points of a network), the proportionality effect still governs the coupled ensemble [7]. In such case, Eq. 1 is rewritten as follow:

$$T_j \square (1 + \varepsilon_j + \varepsilon_{j-1} + \dots + \varepsilon_k) T_k = (1 + \alpha_{jk}) T_k, \quad j > k \quad (2)$$

where  $T_j$  and  $T_k$  are respectively the cumulative time delays of the two impulse responses as measured from two arbitrary subsystem outputs, and  $\alpha_{jk} = \varepsilon_j + \varepsilon_{j-1} + \dots + \varepsilon_k$  a proportionality factor. Experimental data were collected along the upper limb to verify if cumulative time delays met the proportional regression of Eq. 2.

## 4. Methods

### 4.1 Experimental procedures

Following the approval of the local ethical committee, experiments were performed on 10 right-handed subjects of both genders, aged between 22 and 45, in good health i.e. without declared history of neurological and physiological diseases. They were asked to grasp firmly a stylus in their dominant right hand and, following an audio signal, to displace the stylus as rapidly as possible on a digitizer tablet (Wacom Intuos II, 22x32cm, resolution of 100 points per mm) between a departure point located in the left bottom corner and a target zone located in the upper right corner. A handwriting 2D digitizer system (*Sign@medic*) was used to collect the stylus pressure and the X-Y trajectory at 200 Hz. From that trajectory, the velocity profile was calculated by using a derivative filter with cutoff frequency  $F_c = 60$  Hz, and a Cheby II low-pass filter with  $F_c = 16$  Hz and attenuation  $Att = -81$  dB. During the execution of the motor task, SEMG of six muscles of the upper limb (Deltoid, Triceps, Extensor Digitorum, Pectoral, Biceps Brachii, Flexor Carpi Ulnaris) were recorded with surface Ag/AgCl electrodes and a reference electrode was placed at a wrist bone. These signals were passed through *Grass*<sup>TM</sup> amplifiers model 15 (gain: 2000, band-pass filter 3-1000 Hz) before being digitized online with a *National Instrument*<sup>TM</sup> data acquisition card (A/D: 2000 Hz) controlled by a *Labview*<sup>TM</sup> user interface. The wires linking the electrodes to the amplifiers were fixed in a way to allow fluent movements while preventing motion artifacts. Acquisition of the stylus displacement was synchronized with the SEMG through a TTL SYNC signal. The experimental set-up is illustrated in Fig. 5a.

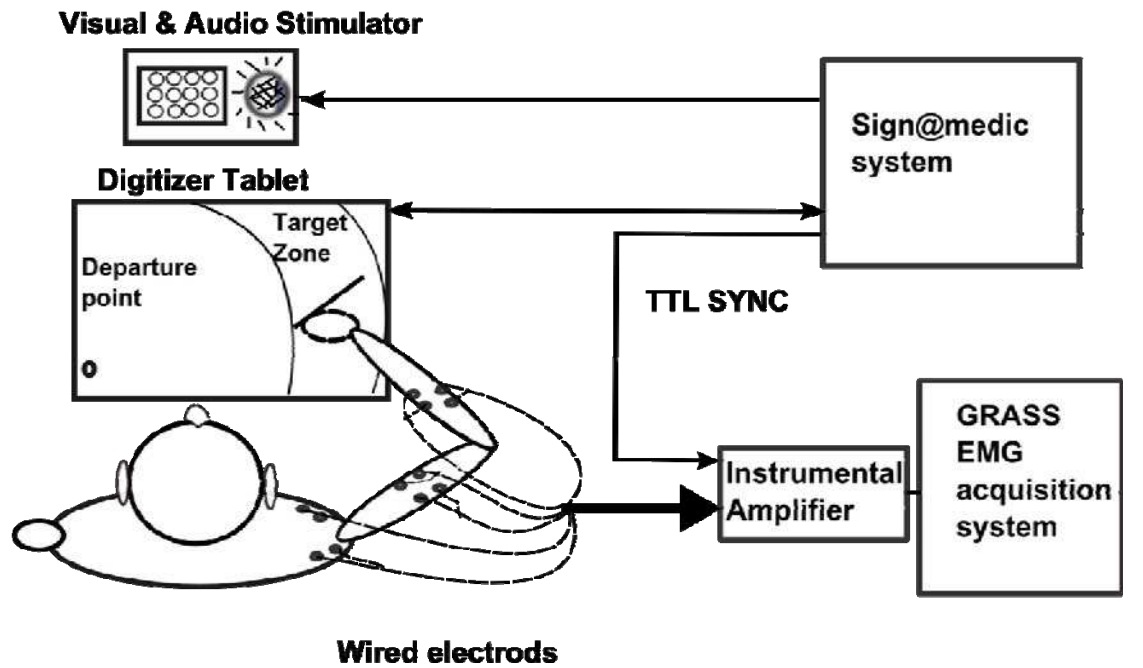
Similarly to reaction time experiments, speed of the movement was privileged over the precision of the stylus positioning. The protocol contained three steps (Fig. 5b): 1) a “WARNING” period

during which the stimulus generator emits a blinking red light indicating that the system is ready to record the trajectory when the subject puts the stylus on the departure point. 2) a «READY» period during which the stimulator switches off the red light after a random pause governed by an exponential hazard distribution to prevent any anticipation of the subject [15]. 3) a «GO» signal consisting in a short beep of 500 ms emitted by the stimulator following which the subject has to execute the motor task as fast as possible. When the target is reached, the subject pulls off the stylus outside the sensitive digitizer surface. This stops the recording process and prepares the system for a new acquisition.

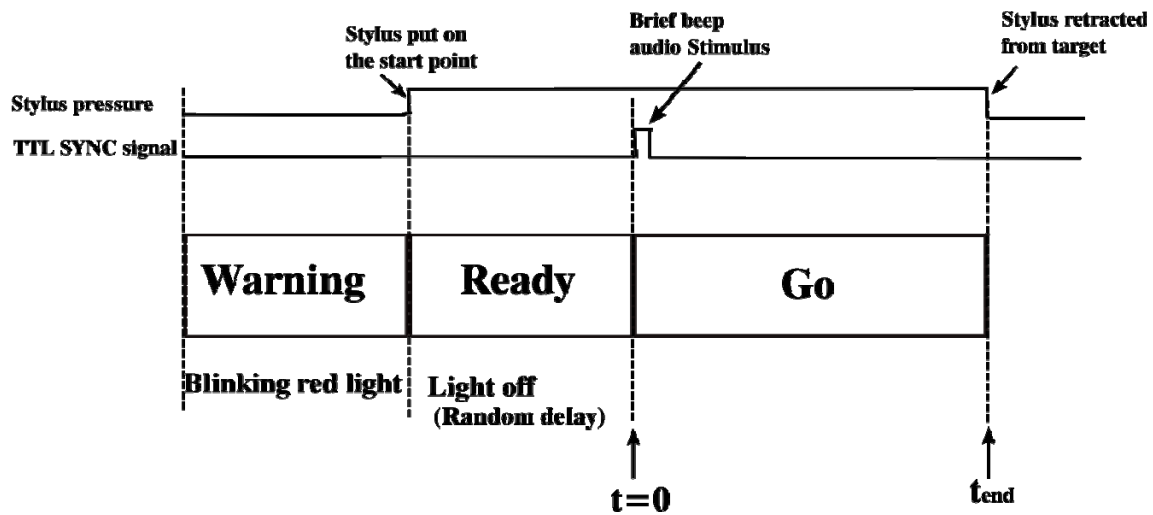
Before the execution of a motor task, the subjects were asked to maintain their shoulder in a comfortable rest posture. Initiation and maintenance of the movement was accomplished by the Triceps, the Deltoid and the Extensor digitorum which are considered as agonist muscles, whereas the Biceps brachii, the Pectoral and the Flexor carpi ulnaris are considered as antagonist muscles. In Table 1, odd numbers refer to the antagonist muscles and the even ones to agonists.

Muscles	Type of muscle	Label
Pectoral (Pectoralis Major)	Antagonist	1
Deltoid (Medial Deltoid)	Agonist	2
Biceps brachii	Antagonist	3
Triceps	Agonist	4
Flexor carpi ulnaris	Antagonist	5
Extensor digitorum	Agonist	6

**Table 1:** Nomenclature of the muscles studied presented as agonist/antagonist pairs



(a)



(b)

**Figure 5:** (a) Illustration of the experimental set-up; (b) steps of the experimental protocol, as adapted from a reaction time protocol [20].

## 4.2. Databases

As illustrated in (Fig. 6a-b), portions of the SEMG associated with a rapid movement were isolated and used to build databases  $DB_1$  and  $DB_2$ . The first database contains the cumulative time delays  $T_j^{(m)}$  calculated from the specific envelopes of the muscle  $j$ , as illustrated in Fig 3d-e, of each trial  $m$  and for each subject, while the second is made up of  $T_j$  calculated from average envelopes, as depicted in Fig 6d-e, obtained by adopting  $t_0$  as a time reference in the superposition of repetitive trials of a same motor task executed by each subject [11]. In both databases, the cumulative time delay of each muscle was computed using the following equation.

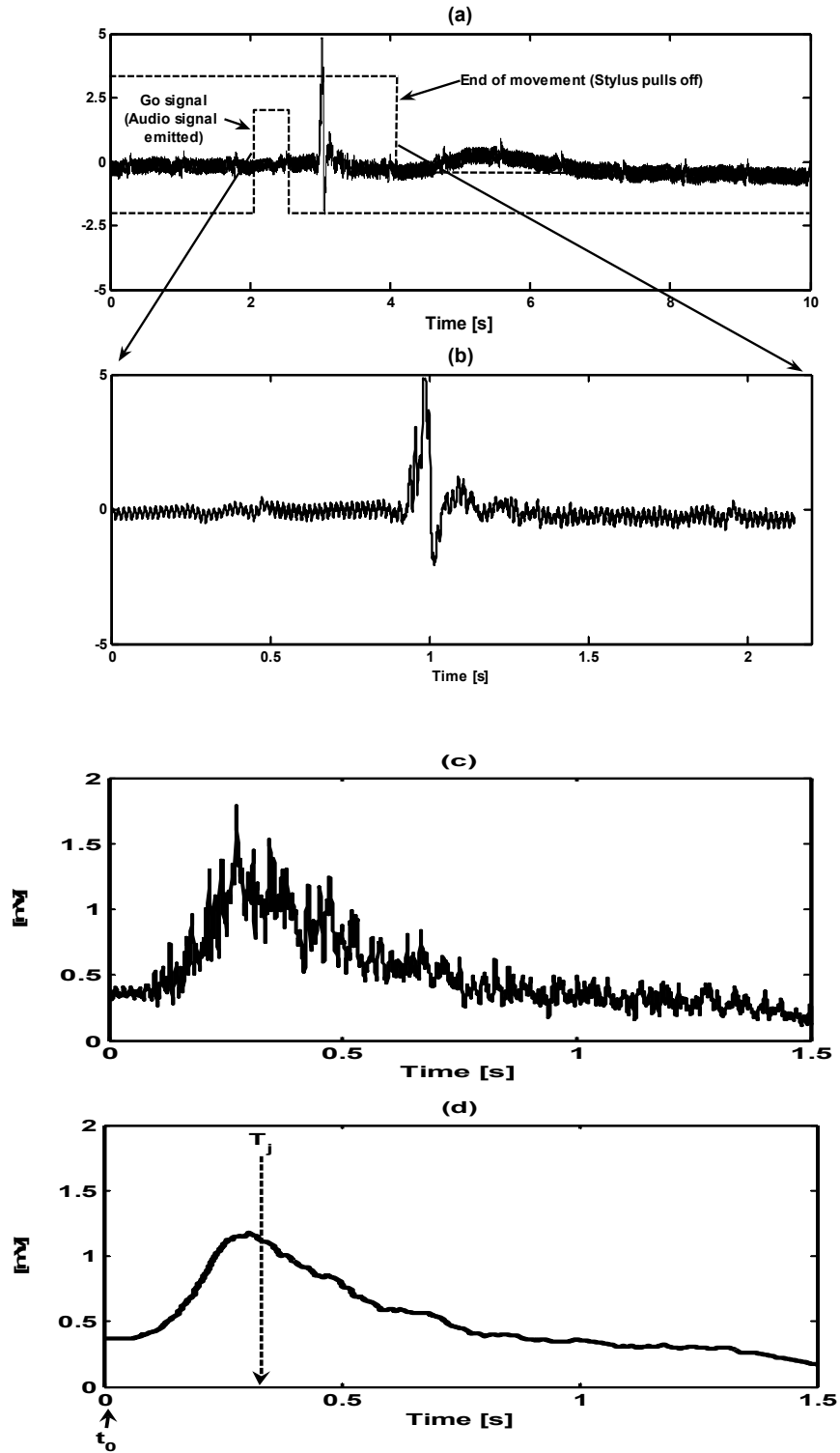
$$T_j = \frac{\int_0^{t_{end}} t |E_j(t)| dt}{\int_0^{t_{end}} |E_j(t)| dt} \quad (3)$$

where  $E_j(t)$  is the SEMG envelope  $j$ , and  $j = 1, \dots, 6$  is the muscle number indicated in Table 1.

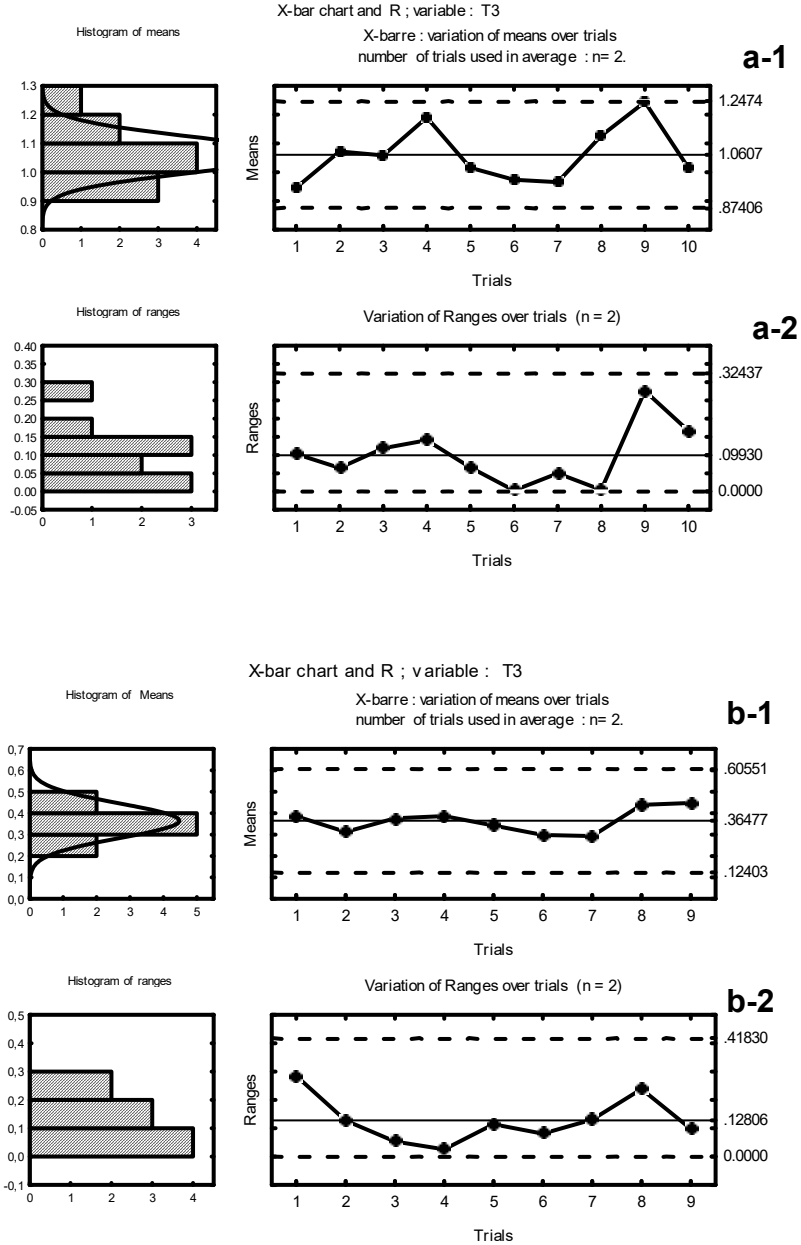
## 5. RESULTS

### 5.1. Databases validation

To check if the experiments were statistically controlled, the Shewhart control charts [31] were used. In this test, each subject is considered as an entity whose neuromuscular system produces the temporal impulse responses observed through the SEMG signals from which  $T_j$  are measured.



**Figure 6:** (a) Use of the SYNC signal and of the stylus pressure signal to localize (b) a portion of the SEMG corresponding to a rapid movement: (c) Superposition relative to  $t_0$  of all SEMG envelopes of the Biceps brachii of subject #6; (d) its filtered version.



**Figure 7:** Examples of Shewhart charts (X-bar and Range) depicting the variability of the cumulative time delays  $T_j$ . (a) The maximum was observed on the Biceps muscle activity of subject #7 and (b) the minimum on the Biceps muscle activity of subject #4. For all the subjects, the variability patterns were within these two extreme cases and the experiment was considered as statistically controlled.

For a reliable statistical analysis, the data should have a variability which is constrained within the limits determined by the test. Figures 7 depict the maximum (panels a) and the minimum (panels b) variations observed on the mean values (panels a-1 and b-1) and the ranges (pa-

nels a-2 and b-2) of  $T_j$  in this experiment. For all the subjects, the variations remained inside the realistic limits (dotted lines) defined by the test and the  $DB_1$  data was considered as statistically valid.

The time occurrence  $t_0$ , initially included in the data of  $T_j$  (see Fig 2c), introduces a bias by shifting the SEMG envelopes [5]. To correct this effect, each velocity profile was fitted with a Delta-Lognormal equation. The reconstruction error was minimized using a Delta-Lognormal parameter extraction tool [6],[27]. The extracted parameter  $t_0^{(m)}$  was subtracted from  $T_j^{(m)}$  for each trial  $m$  of the  $DB_1$  database.

## 5.2. $DB_1$ result analysis

The database  $DB_1$  was subdivided in two subsets  $S_1$  and  $S_2$  where each of them containing the data of 5 randomly chosen subjects. The search for the proportional effect was made through proportional regressions between the different  $T_j$ . A square correlation coefficient  $r^2 \geq 0.7$  was interpreted as reflecting a confident correlation between samples [33]. This threshold was used as the decision criterion for the observation of the proportional effect. The analysis was first conducted on  $S_1$ . The data processing consisted of the construction of proportional regression curves  $T_j$  vs.  $T_k$  ( $j, k = 1, \dots, 6$  and  $j \neq k$ ) using:

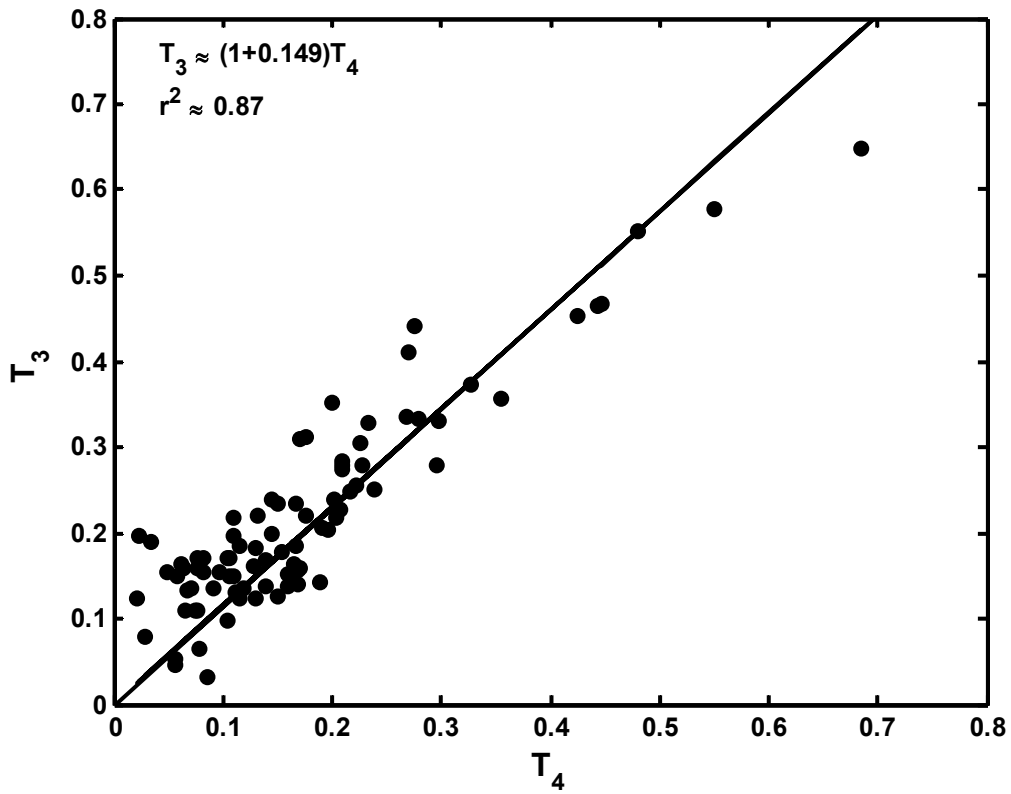
$$T_j = (1 + \alpha_{jk}) T_k = \beta_{jk} T_k \quad (4)$$

where  $\beta_{jk}$  are the regression slopes. Figure 8 illustrates a typical result. Each proportional regression was accompanied by its square correlation  $r^2$  coefficient. The global results are summarized in Table 2.. As one can see, the proportional regressions were obtained with  $r^2 \geq 0.74$ ,



which effectively confirm the existence of proportional effects between the different cumulative time delays.

To confirm, with another data set, the conclusions concerning the observation of the proportionality on the  $S_1$  subset, the regression analysis was repeated on the five subjects of the  $S_2$  subset. Here again the presence of the proportional effect was also confirmed with  $r^2 \geq 0.85$  (see Fig. 9 and Table 3).

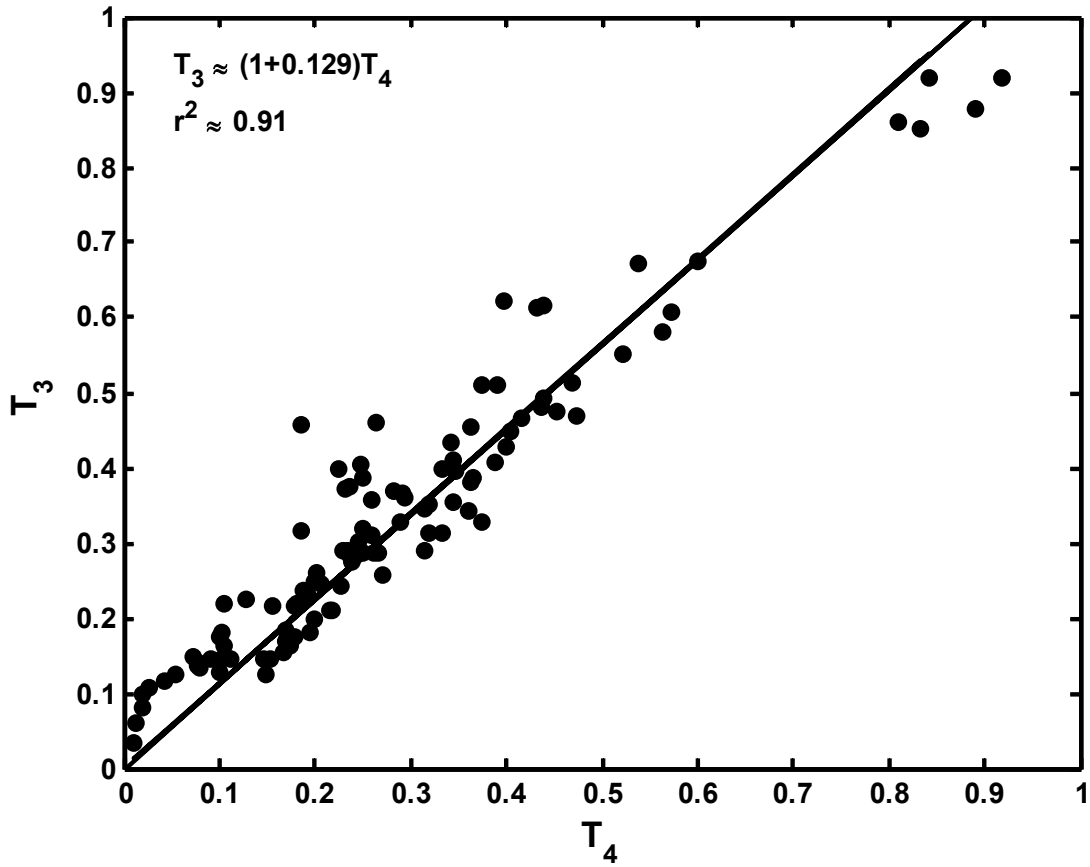


**Figure 8:** Typical proportional regression between cumulative time delays of  $T_3$  (Biceps) and of  $T_4$  (Triceps) calculated from  $S_1$  subset data.

Thus, by processing two independent subsets  $S_1$  and  $S_2$  of individual trials, the proportional effect hypothesis was statistically verified.

$\begin{matrix} Y \\ \backslash \\ X \end{matrix}$	$T_1$	$T_2$	$T_3$	$T_4$	$T_5$	$T_6$
$T_1$	1.00	0.76	0.85	0.76	0.74	0.81
$T_2$	0.76	1.00	0.8	0.82	0.74	0.75
$T_3$	0.85	0.80	1.00	0.87	0.79	0.81
$T_4$	0.75	0.82	0.87	1.00	0.85	0.89
$T_5$	0.74	0.74	0.79	0.85	1.00	0.79
$T_6$	0.81	0.75	0.81	0.89	0.79	1.00

**Table 2:** Correlation coefficients  $r^2$  of the proportional regression results (defined by  $Y=aX$ ) between cumulative time delays of  $S_1$  subset



**Figure 9:** Proportional regression between cumulative time delays of  $T_3$  (Biceps) and of  $T_4$  (Triceps) calculated from  $S_2$  subset data.

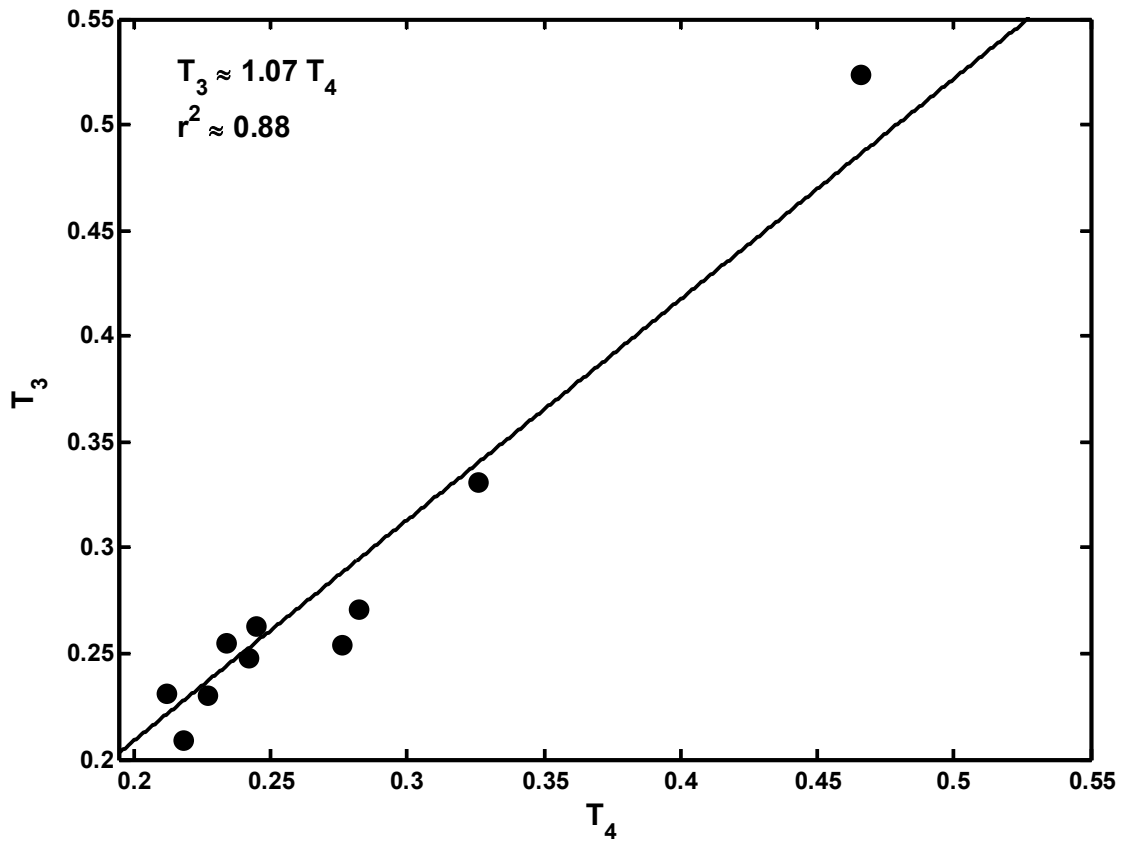
$\begin{matrix} Y \\ X \end{matrix}$	$T_1$	$T_2$	$T_3$	$T_4$	$T_5$	$T_6$
$T_1$	1.00	0.89	0.93	0.90	0.90	0.91
$T_2$	0.89	1.00	0.88	0.91	0.85	0.93
$T_3$	0.93	0.88	1.00	0.91	0.92	0.91
$T_4$	0.89	0.92	0.91	1.00	0.88	0.93
$T_5$	0.91	0.86	0.92	0.88	1.00	0.90
$T_6$	0.91	0.93	0.92	0.93	0.90	1.00

**Table 3:** Correlation coefficients  $r^2$  of the proportional regression results between the cumulative time delays of the  $S_2$  subset.

### 5.3. $DB_2$ result analysis: group averages

To corroborate the results obtained with  $DB_1$ , a second analysis was conducted using the cumulative time delays  $T_j$  of  $DB_2$ . This database contains  $T_j$  calculated from the average envelopes constructed by superposing the SEMG trials. As for individual results, the Shewhart control charts were used and confirmed that the data of  $DB_2$  were statistically controlled. A regression analysis was carried out (Fig. 10) and the proportional effect emerged again with a minimal  $r^2$  of 0.82, as can be seen in Table 4.

Thus, by using another method in the computation of  $T_j$ , the proportionality effect was still observed through the regression results of the all cumulative time delays.



**Figure 10:** Regression curves of the cumulative time delays  $T_3$  vs.  $T_4$ , as calculated from group averages.

Y \ X	$T_1$	$T_2$	$T_3$	$T_4$	$T_5$	$T_6$
$T_1$	1.00	0.92	0.95	0.89	0.92	0.89
$T_2$	0.93	1.00	0.94	0.90	0.84	0.86
$T_3$	0.96	0.95	1.00	0.95	0.94	0.90
$T_4$	0.89	0.89	0.88	1.00	0.93	0.88
$T_5$	0.93	0.85	0.93	0.94	1.00	0.92
$T_6$	0.87	0.82	0.85	0.86	0.89	1.00

**Table 4:** Correlation coefficients  $r^2$  of the proportional regressions between the cumulative time delays of the  $DB_2$  database.

## 6. Discussion and Conclusion

In this paper, two important assumptions of the Kinematic Theory were investigated: the subsystem concept and the proportionality hypothesis. The notion of subsystem was associated, from the brain to the hand, to the presence of different populations of neurons and to the numerous motor units required in the production of a rapid movement. The SEMG collected during such movement constitute a sample of the responses of these biological units to the stimulus signaling the subject to produce a fast displacement of his hand on a digitizer surface. The SEMG signals are characterized by bell-shaped bursts of activity which suggests that the activity of a population of muscular fibers can be represented by the impulse response of a linear subsystem. While the notion of system and subsystem is currently used in physiological modeling of biological systems [16],[18-19], the Kinematic Theory models the behavior of a neuromuscular system involved in the generation of rapid movements by a network of coupled subsystems. Even if these subsystems are non-linear, the theory assumes that their normal functioning is occurring in linear zones of their characteristics [26]. Thus, the impulse response of the coupled network can be modeled by a convolution product of a large number (theoretically infinite) of linear subsystems.

Notice that a subsystem cannot be analyzed individually but such an analysis has a sense when it is considered in a network. For example, the output of a subsystem of rank  $n$  corresponds to its own response and of all  $n-1$  subsystems which precede it. In a rapid hand displacement, the SEMG bell-shaped burst recorded on the upper limb can be considered as the impulse response to a neural command of a finite number of subsystems located upstream of this recording site. Consequently, its time delay, relative to the stimulus onset, corresponds to the accumulation of the upstream subsystems time delays. Since the end-effector rapid movement has also a bell-shaped

velocity profile, the theory considers that the velocity pattern results from the propagation of the neural impulses along the neuromuscular system and can thus be considered as the impulse response of the convolution product. Using the CLT theorem under a proportionality hypothesis of the cumulative time delays, the convolution product converges towards a limit response which has a lognormal profile; considering the agonist/antagonist synergy, the velocity profile of the end-effector rapid movement can then be associated to a Delta-Lognormal profile [22].

As predicted by the Kinematic Theory, the proportional regression of the cumulative time delays reveals that the  $T_j$  are governed by a proportional effect, i.e. the difference between  $T_j$  and  $T_k$  (i.e.  $\Delta T_j$ ) of two subsystem impulse responses is proportional to  $T_j$  ( $\Delta T_j = T_j - T_{j-1} = \alpha_j T_j$ ). This effect known as the Weber's law is applied in a wide variety of sensory systems where the sensation of the stimulus is linked to its intensity [12],[32]. Hatze [12] has pointed out that this law can be viewed as the realization of a general teleological principle of minimum transentropy. Hatze also showed that the 'size principle' of motor units recruitment advanced by Henneman [13] can be seen as the manifestation of a Weber's law, when a motor system is considered as a biological channel which minimizes the entropy in the production of motor tasks. This means that the individual delays, induced by a sequential recruitment of motor units, can be governed by a Weber equation. Thus, it seems that the proportional effect, predicted by the Kinematic Theory, can reasonably be explained by the 'size principle'.

In this perspective, the observation of proportional relationships between cumulative time delays of the different upper arm muscle activities acting in the production of rapid movements can then be considered as a support to a basic hypothesis of the Kinematic Theory.

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