ROLE OF MULTIUNIT ACTIVITY IN RYTHMOGENESIS: INSIGHTS FROM DELETIONS

A Thesis
Submitted to
the Temple University Graduate Board

In Partial Fulfillment
of the Requirements for the Degree
MASTER OF SCIENCE
(BIOENGINEERING DEPARTMENT)

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(December 2015)

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ABSTRACT

The rhythmic activity of locomotion is most frequently modeled as a periodic oscillation coordinated by a spinal Central Pattern Generator (CPG) controlling reciprocal activation of flexor and extensor muscles. Expression of locomotion errors in the form of spontaneous deletions in the motor output has been critical in formulating models of CPG network structure governing locomotion in mammals (Lafreniere-Roula et al 2005, Duysens 2006). Deletions are defined as the disappearance of either antagonist or agonist muscles' activity along with the simultaneous tonic/rhythmic activity of the corresponding agonist or antagonist muscles. The formulation of a two-layer model of the CPG (Rhythm Generator (RG) layer & Pattern Formation (PF) layer) by Rybak et al (2006) stems from observations of such deletions in the fictive locomotion of the decerebrated cat. The RG functions as a clock controlling the temporal activity of the PF layer which controls the firing pattern of motor neuron pools that activate muscles. The deletion episodes are said to be "resetting" if the EMG activity after the deletion does not return after an integer value of the pre-deletion average period. If the motoneuron activity returns in phase with the pre-deletion "clock", the deletion period is considered to be "non-resetting". Multiunit Activity (MUA) recorded from a spinalised air-stepping cat was analyzed against its corresponding EMG activity to investigate the role of MUA in *rhythmogenicity*, specifically whether or not MUA activity may represent the RG layer of the Central Pattern Generator (CPG) model. This hypothesis would predict that MUA activity should be disrupted in phase or amplitude when and only when deletions episodes are re-setting. Alternatively, MUA activity may reflect PF layer activity. In this case MUA activity should be disrupted in phase or amplitude during each of the deletions

episodes. MUA's spatio-temporal characteristics were compared to that of the EMG activity during the deletion periods for analysis. From the analysis performed, there was a significant proportion (average more than 25%) of the MUA (collected from the lumbar region of the spinal cord of spinalized cat) that were disrupted in phase or amplitude during non-resetting deletions or undisrupted during resetting episodes, indicating that MUA activity is unlikely to represent the RG layer activity during . In addition, MUA oscillation during the period of deletions was unchanged (amplitude or phase) for more than 25% of the deletion episodes, ruling out the possibility that MUA represents the activity of the PF layer. So although MUA has been found to be highly synchronized throughout the lumbar extent during locomotor activity, it does not appear to act as a "clocking" mechanism for the locomotor rhythm.

To my awesome, ever supporting parents (Lakshmanan Nagaswamy, Kumari Lakshmanan) and sister (Saishini Lakshmanan) without whom none of this (pursuit for education and science) might have happened for me. Dr Michel A Lemay, Dr Vallorie Peridier and my undergrad head of the department - Dr Kantharaj -> All for their support and encouragement.

ACKNOWLEDGMENTS

I would like to thank my advisor Dr Michel A Lemay for his guidance on thesis and for providing working freedom in the laboratory that helped me to learn various aspects of research. Thanks to Alexander Krupka for helping to shape the analysis better. I would also like to thank my committee members Dr Andrew Spence and Dr Iyad Obeid for having accepted to be a part of the committee and for providing feedback. I would also like to thank my roommate and friend (Devika Sil) for her encouragement and support. Thanks to all the members of Dr Lemay's lab for having helped directly or indirectly with the thesis work.

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CHAPTER 1

INTRODUCTION

In quadrupeds and bipeds, locomotion is exhibited as a periodic progression consisting of a series of alternating stance and swing phases. Explanation for the control and initiation of each phase of a step has been attributed to the interactions between neurons within a network and the sensory feedback from innervated muscles and cutaneous receptors. This locomotor network has been studied with muscle electromyograph (EMG) and spinal neuronal recordings (Multi Unit Analysis (MUA), Single Unit Analysis (SUA), etc.), considering neuronal signals as input and the mechanical movement as output.

The spinal central pattern generator (CPG) is the innate spinal circuitry that is responsible for the generation of rhythmic locomotion and that controls the sequence of each step phase. There have been several models postulated to describe the functioning of the CPG, and its independence from the supraspinal neural circuits in generating the locomotor rhythmic motor output. In order to analyze the spinal circuits' function as locomotion generator, data collected from spinal transected cats (i.e. spinal circuitry deprived of descending control) was used for my thesis. The functional output of the spinal cord was studied in terms of limb muscles' activation during air-stepping (Giuliani, 1985), while the functioning of the spinal circuitry was explored by recording the electrical activity within the grey matter of the lumbar cord during bouts of air-stepping. EMGs provided a measure of the motoneuronal output to the hind limb muscles, while extracellular neuronal recordings provided a measure of the interneuronal activity driving the

motoneurons. The aim of this thesis was to explore the relationship between the two sets of recordings and their implications for current models of the spinal locomotor circuitry.

In all models, motoneurons exciting the flexors and the extensors are alternatively activated by the CPG to generate the oscillation of limbs, resulting in propulsion of the body. This mutual inhibitory coupling between the synergist and antagonist motoneuron pools is one of the major characteristics of the various CPG models. Recordings of motoneuron activity in the fictive locomotion preparation (paralyzed (no afferent feedback), decerebrated (no cortical input) animal) show spontaneous silencing of a muscle group (flexors or extensors) (output of the respective motor neuron pool) with the simultaneous antagonist muscle group being either tonically or rhythmically active during certain periods of an on-going locomotion cycle (Lafreniere-Roula and McCrea 2005, McCrea and Rybak 2007). These episodes are termed 'deletions'. It was observed that after the period of deletion, the post-deletion EMG onset (output of motoneurons) could occur in phase with the pre-deletion period (i.e. the first post-deletion onset was an integer times the average of the pre-deletion cycle period). This preservation of the motoneuronal output phase after a period of deactivation was termed a 'non-resetting' (NR) deletion while the term 'resetting' (R) was used to describe deletions where the motoneuronal output phase shifted (did not restart on the pre-deletion cycle period) after the deletion episode. In order to account for the non-resetting type of deletion, Rybak et al. (2006) added a layer called rhythm generator (RG) to the one-layer CPG model of Graham-Brown (1911). This RG layer acts as a clock and drives the output of a pattern formation layer (PF) that controls the patterns of motoneuron firing. The separate RG layer encodes rhythmic activity separately from the muscle activation layer, explaining

how NR deletions may occur through disruption of the PF layer without concurrent disruption of the RG layer activity.

The objective of this thesis was to investigate whether the MUA obtained from the lumbar segment of the spinalized cat might represent the output of a clock (RG) or pattern formation (PF) layer. The correlations between the muscle EMGs and the MUA during the deletion periods were computed and analyzed.

1.1 Hypothesis

Multiunit Activity recorded from a spinalized air-stepping cat was analyzed against its corresponding EMG activity to investigate the role of MUA in *rhythmogenicity*, specifically whether or not MUA activity may represent the output of the rhythm generation layer of the central pattern generator (CPG) model formulated by Rybak et al (2009). The investigation was conducted by analyzing the correlations between EMG and MUA activity during deletions episodes.

We hypothesized that the MUA might play the role of the RG since it has been observed to oscillate at the frequency of walking and be synchronized throughout the lumbar extent in spinalized decerebrated air-stepping cats (AuYong et al. 2011). Our hypothesis predicts that if MUA oscillation reduces or stops during an EMG deletion period, then the EMG would reset after deletion. It also predicts that if MUA keeps oscillating, without disruption in its phase or amplitude, during a deletion period then the EMG deletions should be non-resetting. Other outcomes, EMG resetting if MUA activity does not reset, or EMG non-resetting if MUA resets, would suggest that the MUA activity does not represent the RG layer output.

An alternative hypothesis is that MUA activity acts as the Pattern Function layer. In that case, EMG deletion episodes (whether resetting or not) should always coincide with a simultaneous disruption in the MUA activity, either MUA amplitude or phase.

Table 1: Summary of the possible outcomes related to the Hypotheses:

MUA activity during EMG deletion period	EMG deletion episode classification	MUA potential role
Oscillation disrupted	Resetting	RG or PF
Oscillation disrupted	Non-Resetting	PF
Oscillation undisrupted	Resetting	Neither RG nor PF
Oscillation undisrupted	Non – Resetting	RG but not PF

CHAPTER 2

BACKGROUND

2.1 Conceptualization of CPG

Efforts to understand the control of motor activity of an organism in relation to its spinal cord activity started to gain momentum in the early 20th century with the advent of Sherrington's notable take on the reflex circuitry. Sherrington suggested that locomotion was a result of the activation of a series of sequential reflex pathways. After conducting experiments on decerebrated cats, Sherrington (Sherrington 1910) claimed that locomotion could be achieved even after the loss of communication with the supra-spinal efferent and thus the spinal cord was equipped with a locomotor circuitry that could be controlled by the reflex pathways activated by the sensory afferents.

2.2 Single Layered CPG

Thomas Graham Brown refuted the involvement of reflex pathways in generating the rhythmic motor pattern by conducting an experiment on deafferented, decerebrated spinal cats (Brown 1911). He proposed that the spinal cord has an innate neural system (which was later termed the CPG) controlling the rhythmic activity of flexors and extensors resulting in the progressive action of locomotion. Brown postulated a half center model representing the mutual inhibition between the flexors and the extensors. A layer of interneurons passed on their output to the population of motoneurons that innervates the flexors and the extensors (Brown 1916, Stuart and Hultborn 2008). This theory was in contrast to Sherrington's which claimed that the peripheral feedback was the main driver of the extensor-flexor alternating activity. The half center model suggests that when the agonist muscle undergoes contraction, the corresponding antagonist muscles would be

simultaneously inhibited (agonist muscles inhibited through fatigue turning off, and thus releasing the inhibition to the antagonist muscles) and this could be modulated by the proprioceptive feedback (Brown 1916). This was later supported by the results obtained through electrophysiological experiments carried out in cats paralyzed with curare (Jankowska, Jukes et al. 1967). The reciprocal inhibition between the flexors and extensors was noticed at the interneuronal level when locomotion was induced with L-DOPA.

2.2.1 Limitations of the Single Layered Pure Half Center Model

The single layered CPG model had motoneurons divided distinctively into a group controlling the activity of the flexor muscles and another controlling the extensor muscles. Certain uniarticular (for example Vastus Lateralis) or biarticular (for example Rectus Femoris) muscles had activity bursts during both the flexor and the extensor phase making them double-burst muscles (Edgerton et al. 1974; de Leon et al. 1994; C. M. Chanaud et al. 1991) These double-burst muscles do not fit within the half-center model of Brown which predicts that muscles are only active during the flexor or extensor portion of the step cycle, due to the mutual inhibition between the agonist - antagonist motoneuron pools. As stated by McCrea and Rybak (2007) and others, the half-center model did not account for the observation of excitation in some of the agonist motorneurons when the corresponding synergist motorneurons were active. Neither can it explain the asymmetric activity of flexor-extensor motorneurons in terms of the onset time during increase in the speed of fictive locomotion caused by the increased MLR drive (Brown 1916, Markin, Klishko et al. 2010). This asymmetricity is attributed to the

decrease in the stance phase as the speed of fictive locomotion increases while the swing phase is constant and hence the extensor time duration in reduced. The group of motoneurons of each population (flexor, extensor) that gets activated differs depending upon the task and different muscles are employed at different time instances for generating a motor activity. Following are the two types of CPG models that have been proposed to overcome the shortcomings of the single layered CPG model.

2.2.1.1 Flexor Burst Model

The flexor burst model suggested by Pearson et al. (1976) was initially an asymmetrical model (which tried to account for the asymmetrical problem that was considered then to be a drawback of half – centre model) that claimed that the flexor is a burst generator that inhibits the extensor (the extensor gets activated when the flexor drive is silenced). The asymmetry in the coupling between flexors and extensors is attributed to the excitation drive that is found within a synergist group of motor neurons; in the case of flexors, the motoneurons receives a rhythmic drive from the flexor burst generator while in the case of the extensors, it receives a tonic drive (Brown 1916, Orlovskiĭ, Severin et al. 1966, Markin, Klishko et al. 2010). The group of motoneurons of each population (flexor, extensor) that gets activated differs depending upon the task and different muscles are employed at different time instances for generating a motor activity.

2.2.1.2 Unit Burst Generator (UBG) Model

The Unit Burst Generator model postulated by Grillner (1981) is made of separate modules that acts as unit burst generators acting on the flexor and extensors of the

individual joints. It was designed to explain various complicated locomotion scenarios where the flexors and extensors do not behave as mutually synchronized inhibiting network. But, the model does poorly at explaining the deletions observed during fictive locomotion as it does not have a separate layer to control the clock function of the CPG. The preservation of phase after the deletion period cannot be accounted by the UBG model (McCrea and Rybak 2007).

2.3 Double Layered CPG Model

2.3.1 Insight from the deletions

As noted in the Introduction, the occurrence of NR type of deletions in fictive locomotion of cats led to the development of the two layered CPG model with the separation of the rhythm generator (RG) from the pattern formation (PF) layer. The RG serves as a clock that controls the output of the PF which in turn commands the pool of motoneurons that excites either the flexor or the extensor rhythmically producing the periodic step cycles (Rybak, Shevtsova et al. 2006).

2.3.2 Structure of the Rybak/McCrea two-layer CPG Model

Neurons were modeled based on the Hodgkin and Huxley model (McCrea and Rybak 2007; Rybak, Shevtsova et al. 2006; Rybak, Stecina et al. 2006) and grouped as Extensor controlling or Flexor controlling neurons of two layers. A single CPG controlled the activity of the flexors/extensors of one hindlimb and can be coupled to another CPG controlling the contralateral hindlimb. Interneurons controlling the rhythmic behavior of the extensors and flexors were connected in mutual reciprocal inhibition according to the

half center model. RG, PF and motoneuron layers each had two populations of neurons with one activating the Flexors (RG-F, PF-F, Mn-F respectively) and the other extensors (RG-E, PF-E, Mn-E respectively). The PF–E (extensor population) and PF-F (flexor population) were also connected in reciprocal inhibition fashion through Inpf-E and Inpf-F interneurons (Figure 1), respectively. Ia inhibitory interneuron reciprocally inhibits the motorneuron population (extensor-flexor connection). PF excites both its associated motoneuron population that activates the agonist muscle and also the Ia inhibitory interneuron that inhibits the activity of the antagonist muscle (Figure 1).

The effects of stimulated afferents (Table 2) on fictive locomotion was incorporated in the two level CPG network model to account for the phase changes and modulation (in terms of both the amplitude and the time period) when certain afferents are activated. The Renshaw interneurons, Ia interneurons and the Ib interneurons are reciprocally inhibited while the Ib interneuron has additional mutual inhibition. By adding a secondary RG layer, the modulation of phase has been separated from that of its amplitude, thus explaining the preservation of the overall step cycle period after premature phase transition (by stimulating appropriate afferent) or preservation of time period after deletion. The afferents when modulating the RG layer, produce a delay/premature change or no change in the phase which would either reset or non-reset the step cycle phase respectively. Afferent induced changes in the PF layer changes the duration or amplitude of a phase but do not affect the overall timing of the locomotor cycle.

Table 2: Synopsis of afferent effect (from Figure 1)

Activated afferent	Type of excitation/inhibition	Target

Group Ia through Ia interneurons	Monosynaptic excitation	Synergist motorneurons
	Disynaptic inhibition	Antagonist motorneurons
Group Ia, Ib through non-reciprocal group I inhibition (non-locomotion)	Short latency inhibition	Synergist motorneurons
Renshaw cells	Inhibition	Agonist/synergist motorneurons and Ia inhibitory interneurons
Iab	Disynaptic reflex excitation	Extensor motoneurons

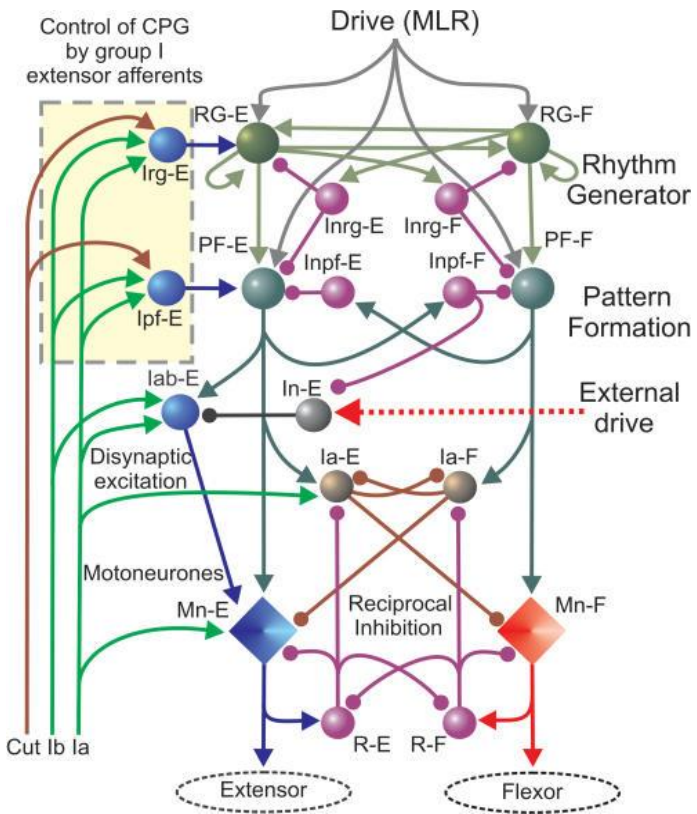

Figure 1: Afferent integration to the extensor activity modulation

Rybak and McCrea's CPG model for locomotion with the extensor afferent effect integrated. Inrg- E and Inpf – E (RG and PF respectively) are the reciprocally inhibiting interneurons of the extensor activating neuron population that helps in achieving the half center model while Inrg – F and Inrg – F are related to the flexor group. Iab-E facilitates the non-locomotion extensor activation (prolonging stance or switching to stance) which will be activated by the PF-E excitation, group-1 non reciprocal excitation and Inpf-F inhibition. Ia acts as a reciprocally inhibiting interneuron that could get excited by the active synergist PF population or Group Ia disynaptic excitation and gets inhibited by the antagonist PF exciting antagonist Ia interneuron. The Renshaw cells along with the Ia interneurons form the reciprocal inhibition connection at the lower motor neuron level.

CHAPTER 3

METHODS

3. 1 Classifying Deletion Episodes

Previously recorded data on muscle and interneuronal activity recorded during air-stepping in spinalized cats was examined to determine the presence of deletion episodes during the locomotor bouts (McMahon 2014). The EMG onset times recorded from 5 cats were used to determine the presence of deletion episodes in a given bout of air-stepping (duration of an air-stepping trial was around 1 s). Each air-stepping trial was checked for the presence of deletion episodes with consistent rhythmic activity pre and post-deletion (criteria used was to have three cycles preceding the deletion and three following). In order to find the deletion episodes in the air-stepping trials, an algorithm for finding the periods during which stepping stopped was developed. The algorithm calculated the time differences between consecutive EMG onsets for each trial; trials where the time differences jumped to a level significantly higher than the average time difference during certain periods were further explored. We found 15 different trials where 27 deletion episodes were observed (some trials had more than one deletion episodes). The deletion episodes selected showed a significant drop in the EMG amplitude (levels dropped to less than half of the pre-deletion levels) for some or all of the hindlimb flexors/extensors during a period lasting longer than a step cycle.

The deletion episodes were classified based on the muscles that went silent or tonic during the deletion period. The muscle EMG activity was said to be tonic for a given period of time if the amplitude of the EMG did not get less than half of the average amplitude for more than the average time period of one EMG burst (i.e. EMG showed non-rhythmic continuous activity). EMG activity could also be classified as silent if its

amplitude was less than half the average amplitude (most of the silent activity had amplitude equivalent to zero) for at least the average time period of a burst or rhythmic if EMG had a consistent behavior in terms of both its amplitude and time period. A typical deletion consisted of the extensor or flexor muscle group going silent while the antagonist muscle group showed tonic activity. Further classification was based on whether the deletion involved a single limb, i.e. unilateral response where flexor/extensor activity in the contralateral limb continued to be rhythmic, or both limbs.

These deletion episodes were then checked for the change in onset time after deletion (phase change) and were classified as either non-resetting (NR) or resetting (R) deletions. In the case of non-resetting deletions, the initial EMG onset time following deletion was an integer multiple of the average cycle period (averaged over three periods pre- and three periods post-deletion). The remainder of the time period between the muscle onsets preceding and terminating the deletion, divided by the average cycle period was compared with the 95% confidence interval of the average cycle period. If that remainder was within the 95% confidence interval the deletion was considered non-resetting, if it was outside the interval it was considered resetting (Lafreniere-Roula and McCrea 2005).

3.2 MUA Analysis

3.2.1 Signal Pre - Processing

Two microelectrode arrays with 64 sites each (over 8 shanks) (model A8x8-5mm-200-200-177, Neuronexus, Ann Arbor, MI) were inserted at about 3000μm in depth in two lumbar segments along the rostrocaudal axis (McMahon 2014). Data collected from the first (dorsal) and the last (ventral) row of sites (8 sites per row) from the rostral and caudal electrodes were used for this analysis. The raw MUA signal was clipped off at

about 2 times the standard deviation (Auyong, Ollivier-Lanvin et al. 2011) and was further band-pass filtered from 300Hz to 4000Hz. The average root mean square of the bandpass filtered MUA signals obtained from the electrode sites in the top and bottom row was further filtered at 100Hz. Fast Fourier Transform was performed on the resulting signal to identify the frequency component with the higher power density. For the majority of the trials, the highest power was at around 1 Hz and 6 – 8Hz. The signal was thus filtered at both 1Hz low-pass and 6 – 8 Hz bandpass for separate analyses of those two bands.

3.2.2 Data Processing

Both the 1Hz filtered signal and the 6-8Hz filtered signals from the first and last row of sites of the caudal and rostral electrodes were analyzed to determine the changes in MUA activity during the deletion periods. Local maximum peaks (maxpeak) and local minimum peaks (minpeak) were used to examine the change in the phase of MUA during the deletion episodes. The following calculations were done for the 1Hz and 6-8Hz MUA filtered signals:

1) MUA Amplitude: The peaks were analyzed to ensure that modulation of the MUA continued during the deletion period. The differences between the minima and maxima was compared to the average minima-maxima of the 5 cycles preceding the deletion episodes and a peak was considered valid if its amplitude difference was more than the lower value of the 95% confidence interval of the average pre-deletion amplitude differences. The label ‘R’ is used to indicate a significant drop in amplitude during the deletion period, indicating a resetting of MUA during the deletion period; while ‘NR’

indicates no significant change in amplitude during the deletion period (i.e. non-resetting behavior).

2) MUA Phase: The period between MUA peaks during the deletions was compared to the average period for the 5 cycle periods preceding the deletions to establish if the phase of MUA activity changed during deletions. The MUA signal was considered to have changed in phase or resetted if the largest MUA cycle period (period only got longer during deletions, never shorter) during the deletion was outside the 95% confidence interval of the pre-deletion average period. If the MUA had undergone modulation/disturbance during the deletion period when compared to its pre-deletion time period, it was said to be resetting ('R'), if not it was considered non-resetting ('NR').

3) MUA Oscillation State: MUA oscillation state was defined as non-resetting ('NR') when both amplitude and time period were unchanged from pre-deletions values during the deletion episode. If either changed (amplitude dropped, or phase re-setted), the MUA was considered non-oscillatory or resetting ('R') during the deletion period.

3.2.3 Analysis:

Correlations between the EMG deletion episodes and MUA were examined. MUA behavior during the deletion period was analyzed based on the changes in phase (significant changes in period), amplitude (significant change in amplitude) or amplitude and phase (significant changes in both period and amplitude – oscillation state). Correlations between the EMG and MUA responses during the deletion episodes were examined. We performed two series of analyses: one that included all the deletions

found, and one that excluded the deletions that occurred in the left hindlimb since the MUA was always collected from the right hemicord.

The contingency tables (for chi-square testing) of the resetting/non-resetting cases for EMG and MUA (classification based on either phase, amplitude or phase & amplitude) are presented in Table 17 and 18. Separate tables were constructed for the MUA collected from the rostral and caudal segments of the spinal cord. In addition, MUA were separately analyzed based on their dorsoventral position using the first and the last row of the electrode array – MUAtr and MUAbr respectively. The L3/L4 segment activity was analyzed separately using the MUA that were collected from those regions of the spinal cord. In order to analyze the deviations of the diagonal and column chi- square distributions against homogeneous distributions, independence chi-square statistical tests were conducted for three different parameters of the MUA (MUA Phase vs EMG phase – analysis A, MUA amplitude vs EMG amplitude – analysis B and Oscillation state of MUA vs EMG phase – analysis C). The null hypothesis of a homogenous distribution for the EMG versus MUA resetting/non-resetting combinations was tested for the diagonal and column distributions. The contingency table was subjected to chi-square test analysis (element based analysis, diagonal and column analysis) to obtain the statistical difference between the expected and the observed frequency of parameters.

3.2.3.1 EMG Phase *versus* MUA Phase

The change in MUA activity during the deletion period in terms of its phase was compared with the EMG change in phase (resetting/non-resetting) using the chi-square test. We use a chi-square homogeneity test to determine whether the various

combinations (EMG R-MUA R, EMG NR – MUA- R, etc.) were equally distributed, with p set at 0.05. We further examined the sums of the diagonals and columns of the distribution to probe if these sums were even or if they matched the predicted distributions based on our hypotheses (chi-square test between sums of the columns or sums of the diagonal).

3.2.3.2 EMG Phase *versus* MUA Amplitude

MUA amplitude during deletion episodes was compared against the EMG phase resetting following the deletions. Similar chi-square testing was used to examine the relation between MUA amplitude changes and phase of EMG following the deletion episodes.

3.2.3.3 State *versus* Phase of EMG

MUA oscillation state was compared against the EMG phase resetting following the deletions. Similar chi-square testing was used to examine the relation between MUA oscillation state changes and phase of EMG following the deletion episodes.

CHAPTER 4

RESULTS AND ANALYSIS

4.1 EMG Deletion Patterns

From 191 air-stepping trials that were recorded from 7 different cats, 27 deletion episodes were identified from 15 trials in 5 cats. The deletion episodes were classified into 9 categories depending upon the hindlimb muscles' EMG activity during the deletion period. In 12 of the 27 recorded episodes, the EMG deletions occurred in only one leg (unilateral response) and muscle activity in the contralateral hindlimb continued uninterrupted. In the remaining 15 episodes, deletions affected both hindlimb and were classified as bilateral (See Table 2). Each deletion was further characterized as resetting or non-resetting. Out of the 27 episodes, 20 were resetting and 7 cases were non-resetting. Most of the deletion episodes were classified into tonic/rhythmic flexors – silent extensors (12 cases) or silent flexors – rhythmic/tonic extensors (13 cases). The remaining 2 cases were bilateral responses with tonic extensors – silent flexors in one limb and tonic/rhythmic flexors – silent/rhythmic extensors in the other, or tonic extensors in both hindlimbs but with rhythmic flexors in one hindlimb and silent in the other.

Correlations between the EMG and MUA responses during the deletion episodes found were examined. We performed two series of analyses: one that included all the deletions found, and one that excluded deletions that occurred in the left hindlimb since MUA were only collected from the right hemicord.

4.2 EMG – MUA Correlation:

The statistical null hypothesis of homogenous chi square distribution for the EMG *versus* MUA resetting/non-resetting combinations was tested for the both the sums of the diagonals and columns. The individual cell based homogeneity tests yielded statistically

significant chi-square values for both dorsal and ventral region MUAs along rostrocaudal segments and L3/L4 segments compared with the EMG phase (for analyses involving MUA phase, amplitude or oscillation state). This implies that the observed frequencies of each EMG category of phase resetting in relation to the MUA phase resetting category are not homogeneously distributed. Further column and diagonal based analyses were conducted to test the scientific hypothesis concerning the possibility that the MUA represents an RG or PF signal.

The contingency tables were subjected to chi-square test to obtain the statistical difference between the expected and the observed sums of columns or sums of diagonals. The column analysis involved the comparison of two groups called the left and the right which were obtained by the summation of two parameter sets (left column = R (EMG) – R (MUA) + NR (EMG) – R (MUA) *versus* right column = R (EMG) – NR (MUA) + NR (EMG) – NR (MUA). Diagonal analysis was conducted by comparing two diagonal sums called no-sync and sync where sync is the summation of R (MUA) – R (EMG) cell and NR (MUA) – NR (EMG) cell while no-sync is the summation of NR (MUA) – R (EMG) cell and R (MUA) – NR (EMG) cell. The following results were obtained for the 1Hz filter MUA signal (Tables 3, 4, 5).

4.2.1 1Hz MUA Signal Analysis

4.2.1.1 EMG Phase *v*ersus MUA Phase (Analysis A)

The change in MUA activity during the deletion period in terms of its phase was compared with the EMG change in phase (resetting/non-resetting) (Table 3, Table 17). There was no significant difference between the sums of the diagonals, rejecting the

research hypothesis that MUA acts as the RG layer. The sums of the columns were significantly different for the MUAs collected from the dorsocaudal region of the spinal cord (n: 27, χ^2: 6.26, p: 0.0124) indicating a non-homogeneous distribution. Since the right column had a significantly lower frequency count than that of the left side, the data contradicts the hypothesis that the MUA acts as the PF layers as this hypothesis predicts that the left column should ideally have a frequency count of zero.

Analysis for the MUA signals from the L3/L4 segments did not show any statistically significant difference for the sums of the diagonals or columns.

When we excluded the unilateral left hindlimb deletion episodes (Table 4), we found no statistical differences for either the diagonal or column distributions. Thus, the MUA from that region does not behave as RG or PG.

4.2.1.2 EMG Phase *versus* MUA Amplitude (Analysis B)

When comparing the MUA amplitude behavior against the EMG phase during deletion episodes (using the chi-square distribution test), the sums of the diagonal tests didn't show any significant differences (Table 3). There were two significantly different column distribution and they were for the MUAs of the dorsal caudal and ventral caudal segment (n: 27, χ^2: 4.48, p: 0.034 and χ^2: 6.26, p: 0.012 respectively). In both these cases, the left column sum was more than that of the right side but the right column sum was still over 25% of the total number of deletions, suggesting that the MUA did not represent a PF layer.

Exclusion of the unilateral left side deletion episodes showed no significant differences for any of the diagonal distributions and only one significant one for the column

distribution (n: 18, χ^2: 5.56, p: 0.0184). There was a difference of about 55% between the right and the left columns with the left column having higher frequency, which is contrary to the hypothesis that the MUA represents the PF layer output.

For the MUA signals from the L3/L4 segments there was no significant difference between either the diagonal or column distributions with and without the inclusion of the unilateral left deletion episodes.

4.2.1.3 MUA Oscillation State *v*ersus Phase of EMG (Analysis C)

MUA signals from all the analyses done on different spinal segments data showed statistically significant differences between the sums of the columns. In these cases, the right column sum was greater than the left and so it does not support the research hypothesis that MUA represents the PF output. Analysis of the sums of the diagonals showed a heterogeneous distribution for the caudal, rostral and L3/L4 data that were collected along the dorsal and ventral regions. But, no-sync summation value was greater than that of the sync summation value suggesting that the MUA could not be RG (Table 17).

When we excluded the unilateral left hindlimb deletion episodes, both the dorsal, and ventral data of the rostral, caudal and L3/L4 data had significantly different sums for the diagonals and columns of the contingency table (except for the diagonal relation for dorsal MUA along the L3/L4 region which was not significantly different). But neither the MUA as RG nor as PF hypotheses can be supported since the counts were higher for the diagonal or column expected to have a count of zero under the hypothesis of the MUA representing the RG or PF layer output. (P value for comparison of the diagonal

sums of dorsorostral, ventrorostral and dorsocaudal: 0.0184, for ventrocaudal: 0.0047; for the column sums of dorsorostral and ventrorostral: 0.0047, dorsocaudal: 0.000016, ventrocaudal: 0.00005)

4.2.2 6-8 Hz MUA Signal Analysis

Following are the results obtained from the analysis of 6-8 Hz MUA signal (Tables 6, 7, 8) with respect to the resetting/non-resetting of the EMG signals following deletions *versus* changes in MUA. For all the analyses (A, B and C) the distributions were homogeneous (similar to the results for the 1Hz MUA signal analysis). Analysis of the sums of the columns and diagonals revealed very few significant differences.

4.2.2.1 EMG Phase *versus* MUA Phase (Analysis A)

Comparison of EMG and MUA phase using chi-square tests resulted in no statistically significant differences between the sums of the diagonals or columns of the MUA *versus* EMG phase resetting distribution. Even after having excluded the unilateral left deletion episodes there was no change in the results.

4.2.2.2 EMG Phase *versus* MUA Amplitude (Analysis B)

The summation of columns for the ventral caudal MUA data were significantly different (n: 27, χ^2: 7.296, p: 0.0069); column sums for MUA data from the other regions and the diagonal summation were not significantly different. The sum of the left column was higher than the right column for the MUA data collected from the ventral caudal region but the right column still included 22.2% of the cases, invalidating the hypothesis that the MUA represents the RG output.

In the case of the L3/L4 data, the ventral region had significantly different (n: 19, χ^2 : 4.333, p: 0.0373) frequency count for the columns and the left column was higher than right column but the right columns still included a significant number of deletions (31.6%) invalidating the hypothesis that MUA acted as PF.

Exclusion of the unilateral left deletion episodes resulted in a significantly different (n: 18, χ^2: 5.556, p: 0.0184) column summation for the MUAs from the ventral caudal region with a higher sum for the right column which contradicts the overall hypothesis that MUA represent the RF layer output.

4.2.2.3 MUA Oscillation State *versus* Phase of EMG (Analysis C)

MUA signals from the ventrorostral region had significantly different sums of diagonals. The no-sync values were higher than the sync summation values which does not support the hypothesis that the MUAs represent the RG layer output. The no-sync values were 70.3% of the total number of deletions as opposed to being zero. For MUAs from the L3/L4 segments, all the analyses had significantly different diagonal and column summations except for the diagonal sums of the dorsal data. But the column sums using dorsal and ventral data were larger for the right column, invalidating the research hypothesis. The diagonal analysis was significantly different and its no-sync summation was not zero (it was 78.9% of the total number of deletions with χ^2: 9.133 and p: 0.0025). By excluding the unilateral left deletion episodes, both the diagonal and the column sums were significantly different but the frequency count was lesser in left column and sync diagonal invalidating the two research hypotheses.

Table 3: Classification of deletion episodes

Side	Classification type	phase (EMG)	No. of cases	
Unilateral	R Tonic Flexor-> Silent Extensor	R	1	1
		NR	0	
	L Tonic/Rhythmic Flexor -> Silent Extensor	R	2	5
		NR	3	
	R Tonic Extensor -> Silent Flexor	R	2	3
		NR	1	
	L Tonic/Rhythmic Extensor -> Silent Flexor	R	2	3
		NR	1	
Bilateral	R Tonic Flexor ->Silent Extensor L Tonic/Rhythmic Flexor -> Silent Extensor	R	4	5
		NR	1	
	R Tonic/Rhythmic Flexor -> Silent Extensor L Tonic Flexor -> Silent Extensor	R	1	1
		NR	0	
	R Tonic Extensor -> Rhythmic Flexor L Tonic Extensor -> Silent Flexor	R	1	1
		NR	0	
	R Tonic Extensor -> Silent Flexor L Tonic/Rhythmic Flexor ->Silent/Rhythmic Extensor	R	1	1
		NR	0	
	R Tonic Extensor -> Silent Flexor L Tonic/Rhythmic Extensor ->Silent Flexor	R	6	7
		NR	1	

Table 4: Chi-Square Table - 1 Hz, All Data					
Rostral		Caudal		L3/L4	
A. EMG Phase vs MUA Phase					
A1. Dorsal					
	MUA Phase		MUA Phase		MUA Phase

EMG Phase		R	NR	EMG Phase		R	NR	EMG Phase		R	NR
	R	12	8		**R**	14	6		**R**	10	6
	NR	5	2		**NR**	6	1		**NR**	2	1
A2. Ventral											
	MUA Phase				MUA Phase				MUA Phase		
EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**
	R	9	11		**R**	14	6		**R**	6	10
	NR	3	4		**NR**	4	3		**NR**	2	1
B. EMG Phase vs MUA Amplitude											
B1. Dorsal											
	MUA Amplitude				MUA Amplitude				MUA Amplitude		
EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**
	R	8	12		**R**	8	12		**R**	7	9
	NR	2	5		**NR**	0	7		**NR**	0	3
B2. Ventral											
	MUA Amplitude				MUA Amplitude				MUA Amplitude		
EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**
	R	8	12		**R**	5	15		**R**	6	10
	NR	3	4		**NR**	2	5		**NR**	2	1
C. EMG Phase vs MUA Oscillation State											
C1. Dorsal											
	Oscillation State				Oscillation State				Oscillation State		
EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**
	R	2	18		**R**	2	18		**R**	3	13
	NR	1	6		**NR**	0	7		**NR**	1	2
C2. Ventral											
	Oscillation State				Oscillation State				Oscillation State		
EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**
	R	4	16		**R**	0	20		**R**	2	14
	NR	2	5		**NR**	0	7		**NR**	0	3

Table 5: Chi-Square Table - 1 Hz, Excluding unilateral left deletion episode					
Rostral		**Caudal**		**L3/L4**	
A. EMG Phase vs MUA Phase					
A1. Dorsal					
	MUA Phase		MUA Phase		MUA Phase

EMG Phase		R	NR	EMG Phase		R	NR	EMG Phase		R	NR
	R	8	7		**R**	11	4		**R**	8	6
	NR	1	2		**NR**	2	1		**NR**	1	0
A2. Ventral											
	MUA Phase				MUA Phase				MUA Phase		
EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**
	R	6	9		**R**	10	5		**R**	6	8
	NR	1	2		**NR**	2	1		**NR**	0	1
B. EMG Phase vs MUA Amplitude -Exclude Unilateral Left											
B1. Dorsal											
	MUA Amplitude				MUA Amplitude				MUA Amplitude		
EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**
	R	7	8		**R**	7	8		**R**	6	7
	NR	1	2		**NR**	0	3		**NR**	0	2
B2. Ventral											
	MUA Amplitude				MUA Amplitude				MUA Amplitude		
EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**
	R	5	10		**R**	4	11		**R**	3	10
	NR	1	2		**NR**	0	3		**NR**	1	1
C. EMG Phase vs MUA Oscillation State											
C1. Dorsal											
	Oscillation State				Oscillation State				Oscillation State		
EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**
	R	2	13		**R**	1	14		**R**	2	11
	NR	1	2		**NR**	0	3		**NR**	0	2
C2. Ventral											
	Oscillation State				Oscillation State				Oscillation State		
EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**
	R	2	13		**R**	0	15		**R**	1	11
	NR	1	2		**NR**	0	3		**NR**	1	2

Table 6: Summary of Chi square test analysis for 1Hz MUA signal, All data						
Analysis	Segment level		All Data			
			Diagonal		Column	
			No of Significant cases	summation group that varies	No of Sig cases	summation group that varies
A. EMG Phase vs MUA Phase	A1. Rostral	Dorsal	0	Not different	1	Not different
		Ventral				Not different
	A2. Caudal	Dorsal				**left > right (x2 - 6.26, p - 0.0124)**
		Ventral				Not different
B.EMG Phase vs MUA Amplitude	B1. Rostral	Dorsal	0	Not different	2	Not different
		Ventral				Not different
	B2. Caudal	Dorsal				**left > right (x2 - 4.482, p - 0.0343)**
		Ventral				**left > right (x2 - 6.259, p - 0.0124)**
C. EMG Phase vs MUA Oscilation state	C1. Rostral	Dorsal	2	Sync < No sync (x2 - 4.482, p - 0.0342)	4	left < right (x2 - 16.33, p - 0.000053)
		Ventral		Not different		left < right (x2 - 8.333, p - 0.0039)
	C2. Caudal	Dorsal		Not different		left < right (x2 - 19.59, p - 0.000009)
		Ventral		Sync < No sync (x2 - 6.26, p - 0.0124)		left < right (x2 - 27, p - 0.0000002)
A. EMG Phase vs MUA Phase	A1. L3/L4	Dorsal	0	Not different	0	Not different
		Ventral				
B.EMG Phase vs MUA Amplitude	B1. L3/L4	Dorsal	0	Not different	0	Not different
		Ventral				
C. EMG Phase vs MUA Oscilation	C1. L3/L4	Dorsal	2	Sync < No sync (x2 - 6.467, p - 0.0109)	2	left < right (x2 - 9.133, p - 0.0025)
		Ventral		Sync < No sync (x2 - 6.467, p - 0.0109)		left < right (x2 - 16.07, p - 0.000061)

Note:
left = R (EMG) – R (MUA) + NR (EMG) – R (MUA)
right = R (EMG) – NR (MUA) + NR (EMG) – NR (MUA)
sync = R (MUA) – R (EMG) + NR (MUA) – NR (EMG)
no-sync = NR (MUA) – R (EMG) + R (MUA) – NR (EMG).
$\chi 2$ = chi square value and p = significant level

Table 7: Summary of Chi square test analysis for 1Hz MUA signal, Excluded unilateral left deletion episodes

Analysis	Segment level		Excluded unilateral left deletion episodes			
			Diagonal		Column	
			No of Significant cases	Summation group that varies	No of Significant cases	Summation group that varies
A. EMG Phase vs MUA Phase	A1. Rostral	Dorsal	0	Not different	0	Not different
		Ventral				
	A2. Caudal	Dorsal				
		Ventral				
B.EMG Phase vs MUA Amplitude	B1. Rostral	Dorsal	0	Not different	1	Not different
		Ventral				Not different
	B2. Caudal	Dorsal				Not different
		Ventral				left < right (x2 - 5.556, p- 0.0184)
C. EMG Phase vs MUA Oscilation state	C1. Rostral	Dorsal	4	Sync < No sync (x2 - 5.556, p - 0.0184)	4	left < right (x2 - 8, p - 0.0047)
		Ventral		Sync < No sync (x2 - 5.556, p - 0.0184)		left < right (x2 - 8, p - 0.0047)
	C2. Caudal	Dorsal		Sync < No sync (x2 - 5.556, p - 0.0184)		left < right (x2 - 14.2, p - 0.0002)
		Ventral		Sync < No sync (x2 - 8, p - 0.0047)		left < right (x2 - 18, p - 0.00002)
A. EMG Phase vs MUA Phase	A1. L3/L4	Dorsal	0	Not different	0	Not different
		Ventral				
B.EMG Phase vs MUA Amplitude	B1. L3/L4	Dorsal	0	Not different	0	Not different
		Ventral				
C. EMG Phase vs MUA Oscilation state	C1. L3/L4	Dorsal	1	Not different	2	left < right (x2 - 8.1, p - 0.0045)
		Ventral		Sync < No sync (x2 - 5.4, p - 0.02)		left < right (x2 - 8.1, p - 0.0045)

Note:
left = R (EMG) – R (MUA) + NR (EMG) – R (MUA)
right = R (EMG) – NR (MUA) + NR (EMG) – NR (MUA)
sync = R (MUA) – R (EMG) + NR (MUA) – NR (EMG)
no-sync = NR (MUA) – R (EMG) + R (MUA) – NR (EMG).
$\chi 2$ = chi square value and p = significant level

Table 8: Chi-Square Table - 6-8 Hz, All Data												
Rostral				**Caudal**				**L3/L4**				
A. EMG Phase vs MUA Phase												
A1. Dorsal												
	MUA Phase				MUA Phase				MUA Phase			
EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	
	R	10	10		**R**	9	11		**R**	9	7	
	NR	6	1		**NR**	5	2		**NR**	3	0	
A2. Ventral												
	MUA Phase				MUA Phase				MUA Phase			
EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	
	R	12	8		**R**	13	7		**R**	9	7	
	NR	5	2		**NR**	5	2		**NR**	2	1	
B. EMG Phase vs MUA Amplitude												
B1. Dorsal												
	MUA Amplitude				MUA Amplitude				MUA Amplitude			
EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	
	R	11	9		**R**	13	7		**R**	8	8	
	NR	7	0		**NR**	4	3		**NR**	3	0	
B2. Ventral												
	MUA Amplitude				MUA Amplitude				MUA Amplitude			
EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	
	R	12	8		**R**	16	4		**R**	10	6	
	NR	6	1		**NR**	5	2		**NR**	3	0	
C. EMG Phase vs MUA Oscillation State												
C1. Dorsal												
	Oscillation State				Oscillation State				Oscillation State			
EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	
	R	6	14		**R**	3	17		**R**	5	11	
	NR	0	7		**NR**	0	7		**NR**	0	3	
C2. Ventral												
	Oscillation State				Oscillation State				Oscillation State			
EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	
	R	1	19		**R**	0	20		**R**	1	15	
	NR	0	7		**NR**	0	7		**NR**	0	3	

Table 9: Chi-Square Table - 6-8 Hz, Excluding unilateral left deletion episode

Rostral				Caudal				L3/L4			
A. EMG Phase vs MUA Phase											
A1. Dorsal											
	MUA Phase				MUA Phase				MUA Phase		
EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**
	R	9	6		**R**	7	8		**R**	8	5
	NR	3	0		**NR**	2	1		**NR**	2	0
A2. Ventral											
	MUA Phase				MUA Phase				MUA Phase		
EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**	EMG Phase		**R**	**NR**
	R	9	6		**R**	10	5		**R**	7	7
	NR	2	0		**NR**	2	1		**NR**	1	0
B. EMG Phase vs MUA Amplitude -Exclude Unilateral Left											
B1. Dorsal											
	MUA Amplitude				MUA Amplitude				MUA Amplitude		
EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**
	R	8	7		**R**	7	8		**R**	8	5
	NR	3	0		**NR**	1	2		**NR**	2	0
B2. Ventral											
	MUA Amplitude				MUA Amplitude				MUA Amplitude		
EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**	EMG Phase		**NR**	**R**
	R	10	5		**R**	3	12		**R**	4	9
	NR	3	0		**NR**	1	2		**NR**	0	2
C. EMG Phase vs MUA Oscillation State											
C1. Dorsal											
	Oscillation State				Oscillation State				Oscillation State		
EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**
	R	4	11		**R**	0	15		**R**	4	9
	NR	0	3		**NR**	0	3		**NR**	0	2
C2. Ventral											
	Oscillation State				Oscillation State				Oscillation State		
EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**	EMG Phase		**Os**	**Nos**
	R	3	12		**R**	0	15		**R**	0	13
	NR	0	3		**NR**	0	3		**NR**	0	2

Table 10: Summary of Chi square test analysis for 6-8Hz MUA signal, All data

Analysis	Segment level		All Data			
			Diagonal		Column	
			No of Significant cases	summation group that varies	No of Sig cases	summation group that varies
A. EMG Phase vs MUA Phase	A1. Rostral	Dorsal	0	Not different	0	Not different
		Ventral				
	A2. Caudal	Dorsal				
		Ventral				
B.EMG Phase vs MUA Amplitude	B1. Rostral	Dorsal	0	Not different	1	Not different
		Ventral				Not different
	B2. Caudal	Dorsal				Not different
		Ventral				**left > right (x2 - 7.3, p - 0.0069)**
C. EMG Phase vs MUA Oscilation state	C1. Rostral	Dorsal	2	Not different	4	left < right
		Ventral		Sync < No sync (x2 - 4.5, p - 0.034)		
	C2. Caudal	Dorsal		Not different		
		Ventral		Sync < No sync (x2 - 6.259, p - 0.0123)		

Note:
left = R (EMG) – R (MUA) + NR (EMG) – R (MUA)
right = R (EMG) – NR (MUA) + NR (EMG) – NR (MUA)
sync = R (MUA) – R (EMG) + NR (MUA) – NR (EMG)
no-sync = NR (MUA) – R (EMG) + R (MUA) – NR (EMG).
$\chi 2$ = chi square value and p = significant level

Table 11: Summary of Chi square test analysis for 6-8Hz MUA signal, Excluded unilateral left deletion episodes

Analysis	Segment level		Excluded unilateral left deletion episodes			
			Diagonal		Column	
			No of Significant cases	summation group that varies	No of Significant cases	summation group that varies
A. EMG Phase vs MUA Phase	A1. Rostral	Dorsal	0	Not different	0	Not different
		Ventral				
	A2. Caudal	Dorsal				
		Ventral				
B.EMG Phase vs MUA Amplitude	B1. Rostral	Dorsal	0	Not different	1	Not different
		Ventral				Not different
	B2. Caudal	Dorsal				Not different
		Ventral				left < right (x2 - 5.556, p - 0.018)
C. EMG Phase vs MUA Oscilation state	C1. Rostral	Dorsal	2	Not different	4	left < right (x2 - 5.556, p - 0.018)
		Ventral		Not different		left < right
	C2. Caudal	Dorsal		Sync < No sync (x2 - 8, p - 0.0047)		left < right (x2 - 8, p - 0.0047)
		Ventral		Sync < No sync (x2 - 8, p - 0.0047)		left < right
A. EMG Phase vs MUA Phase	A1. L3/L4	Dorsal	0	Not different	0	Not different
		Ventral				
B.EMG Phase vs MUA Amplitude	B1. L3/L4	Dorsal	0	Not different	0	Not different
		Ventral				
C. EMG Phase vs MUA Oscilation state	C1. L3/L4	Dorsal	1	Not different	1	Not different
		Ventral		Sync < No sync (x2 - 8.1, p - 0.0045)		left < right (x2 - 15, p - 0.0001)

Note:
left = R (EMG) – R (MUA) + NR (EMG) – R (MUA); righ = R (EMG) – NR (MUA) + NR (EMG) – NR (MUA)
sync = R (MUA) – R (EMG) + NR (MUA) – NR (EMG);no-sync = NR (MUA) – R (EMG) + R (MUA) – NR (EMG).; $\chi 2$ = chi square value and p = significant level

CHAPTER 5

DISCUSSION

On analyzing the MUA signals for both the 1Hz and 6-8 Hz bands, 45 chi-square analyses (from all the possible chi-test that were conducted –> column and diagonal analyses) showed significantly different chi square distribution. Out of these 45, 20 involved MUA signals that were filtered at 6-8 Hz and 25 were obtained by using the MUA signal that was filtered at 1Hz. The frequency count of 40 analyses cases did not favor either the hypothesis that MUA represents the PF layer output or the RG layer output. The remaining 5 analyses (having comparatively more frequency count of variable relation which supports the research hypothesis/alternate hypothesis) were the result of the column analysis and had their left side relation frequency count (left summation) to be comparatively more than that of the right side column summation. Three of those five analyses involved the MUA collected from caudal segments. The other two analyses were based on the MUA collected from the ventral region of L3/L4 segment. Even though the analyses had higher left column summation, the right column summation with the lowest count still included 25% of the cases, contradicting the hypothesis which predicts a count of zero if MUA represents RG (diagonal) or PF (column) layer output. Therefore we conclude that the MUA signals recorded do not represent the output of the RG or PF layer of the McCrea and Rybak model.

Both the 1Hz and 6-8Hz MUA signal analyses, after excluding the unilateral left deletion episodes, showed no significantly heterogenous chi-square distribution for the MUA – EMG table. This further supports our conclusions that MUA is neither acting as the RG

or PF layer of the Rybak and McCrea CPG model. Following are the limitations with suggestions to overcome them for future implementation.

Langlet et al (2005) suggested that the rhythmicity of spinal locomotion is concentrated at the mid-lumbar segments (L3/L4), thereby implying the location of CPG to be around that region. Statistical analysis of the MUA data collected at L3/L4 segments did not support the research hypotheses (of MUA acting as either RG or PF) and this goes against what would be expected from the work of Langlet et al on anatomical distribution of CPG network. This contradiction could be further exploited in terms of the synchronization between the MUA signals collected along the rostrocaudal region of the spinal cord. Desynchronization between the segmental outputs could account for the phase difference of the output (EMG) with respect to the input (MUA).

If we assume that the MUA represents the PF layer output, studying the spinal modules (that defines the muscle synergy patterns which could provide the pattern of neural correlation with its EMG signal) (Bizzi and Cheung, 2013) and their phase correlations with the muscle EMGs could provide more clarity on the pattern of interneurons that gets activated for specific tasks.

If MUA represents the clock output, the observation of NR EMG with R MUA (or R EMG with NR MUA) contradicts this hypothesis. This contradiction could be accounted for by the inclusion of a master clock with a different frequency (different from the ones analyzed – we chose the ones that had high signal energy) connected to various other networks and that controls the temporal pattern of motoneuron firing. They could be connected based on the weight distribution that controls the activation of the combined temporal activity.

Although the MUAs are synchronized throughout the cord during air-stepping (AuYong et al, 2011; McMahon, 2014), suggesting that the MUA might be a clocking signal, the evidence during deletions that has been analyzed for my thesis contradicts the hypothesis. With this thesis we establish that the behavior of the MUA signal during deletions is not what would be expected from the output of the RG or PF layer, it doesn't go silent, it doesn't go tonic, and it sometimes stays in phase with the EMG and sometimes not, pretty much at random.

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APPENDIX A

FIGURE

Graphs of EMG activity and the processed MUA data of selected deletion type (Bilateral – R, Bilateral – NR, Unilateral – R and Unilateral – NR) are included in this section. Figure A.1, A.3 and A.4 involves tonic/rhythmic extensors and silent flexor while Figure A.2 involves tonic/rhythmic flexors and silent extensors.

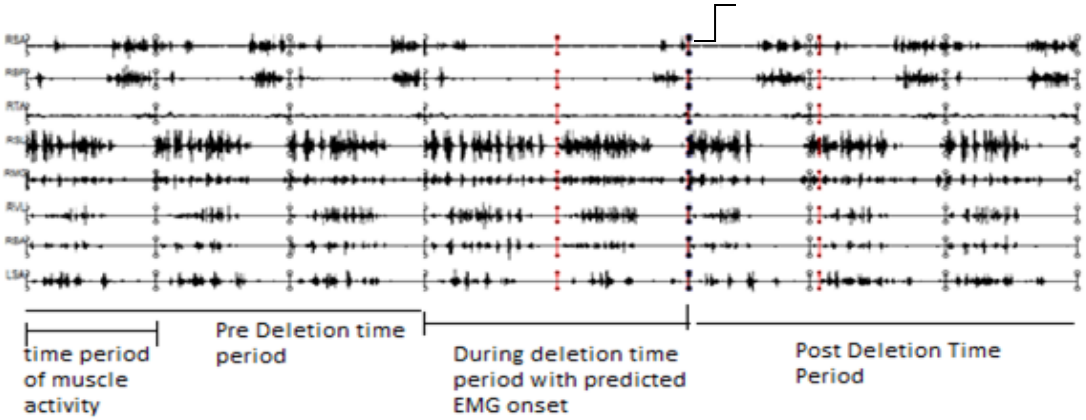

Figure A.1 Example of EMG activity during deletion

EMG output of the extensor, flexor during the deletion time period with three period post and pre deletion periods. Red lines indicate predicted onset time during deletion period that was calculated based on the pre and the post deletion time period.

Muscles have been plotted in the following order (Figure A.3 to A.5): Right Sartorius, Right Biceps Posterior, Right Tibialis Anterior, Left Soleus, Left Medial Gastrocnemius, Left Vastus Lateralis, Left Biceps Anterior, Left Sartorius, Left Biceps Posterior, Left Tibialis Anterior, Right Soleus, Right Medial Gastrocnemius, Right Vastus Lateralis, Right Biceps Anterior. (with Flexors plotted in red and Extensors in blue).

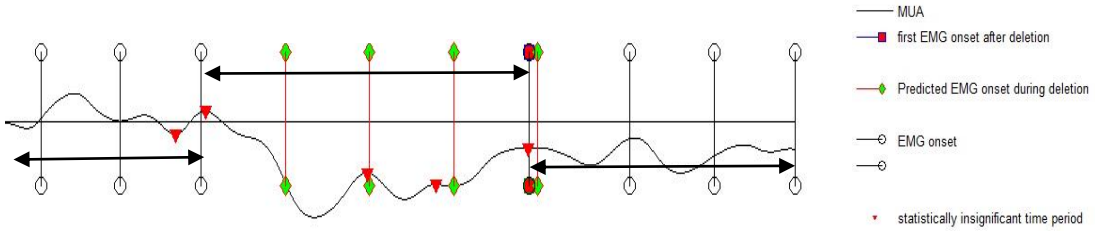

Figure A.2 Example of MUA activity during deletion

Corresponding MUA activity marked with its EMG activity.

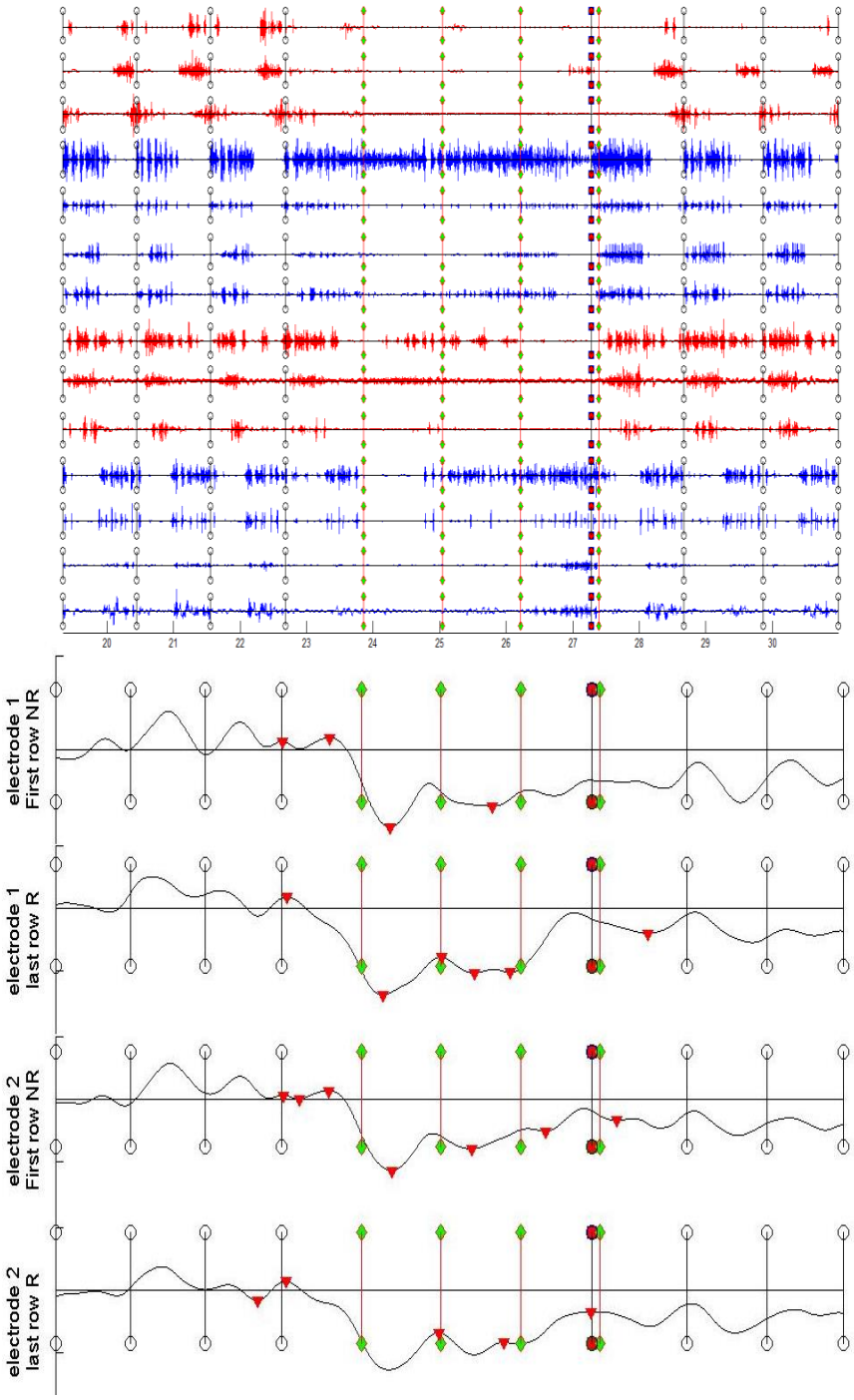

Figure A.3 Bilateral, Resetting deletion episode with Tonic/Rhythmic Extensor and silent flexor -> Cat ID 011 – Trial 013; deletion number 2

(A) EMG (B) MUA collected from electrode 1and MUA collected from electrode 2

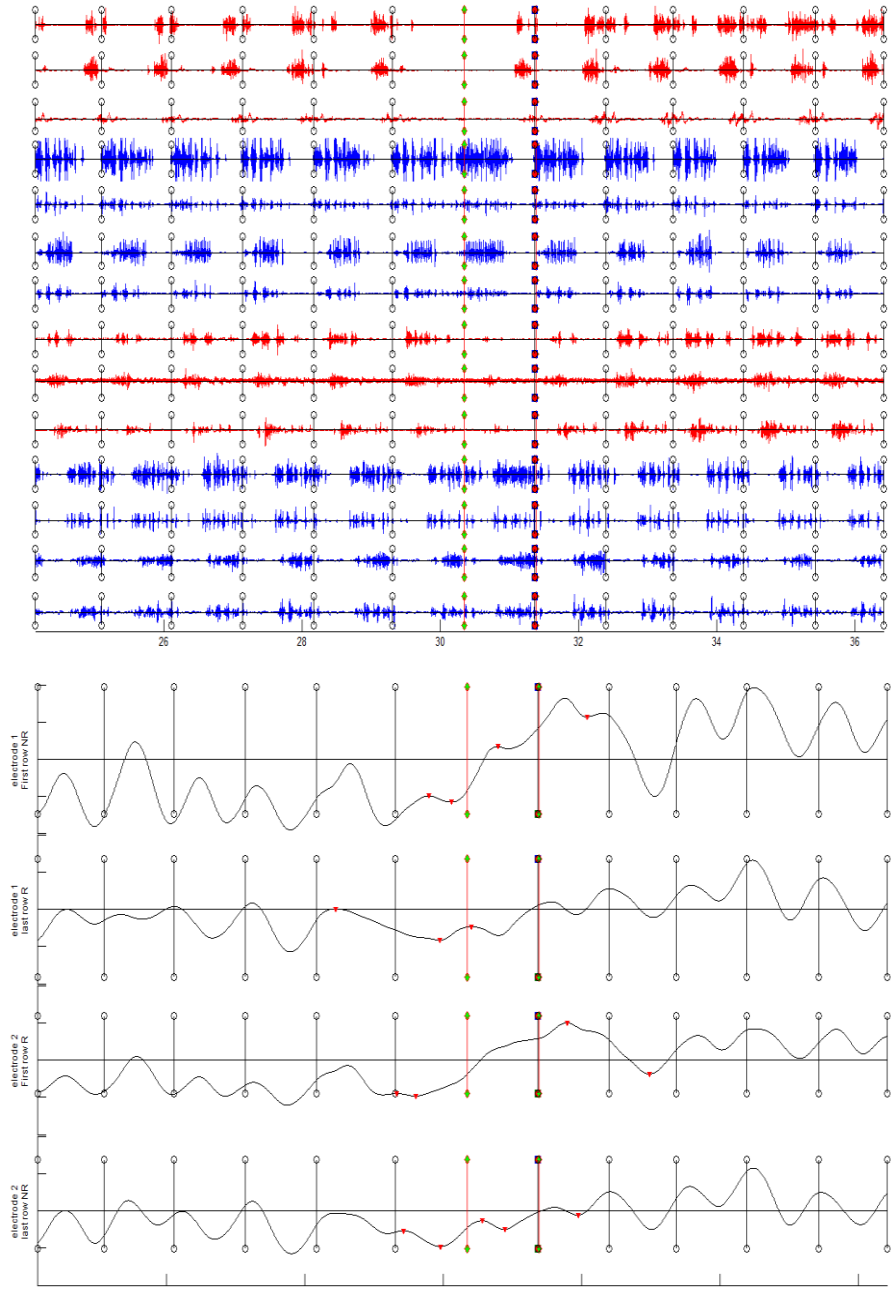

Figure A.4 Bilateral, Non-Resetting deletion episode with Tonic/Rhythmic Flexor and Silent Extensor -> cat ID 05 – Trial 044; deletion number 1

(A) EMG (B) MUA collected from electrode 1 and electrode 2

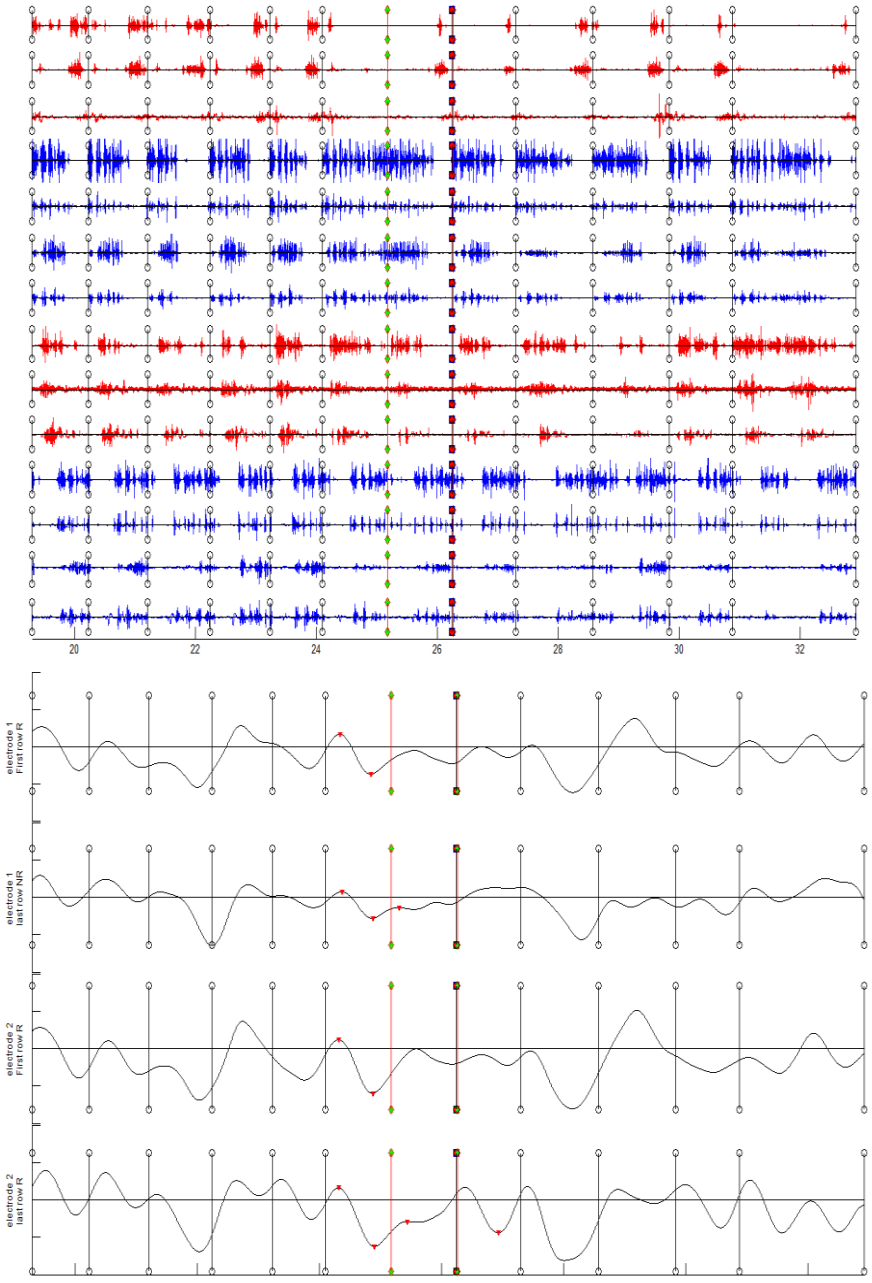

Figure A.5 Unilateral, Resetting Deletion Episode with Tonic Extensor and Silent Flexor -> Cat ID 011 – Trial 035; deletion number 1

(A) EMG (B) MUA collected from electrode 1 and electrode 2

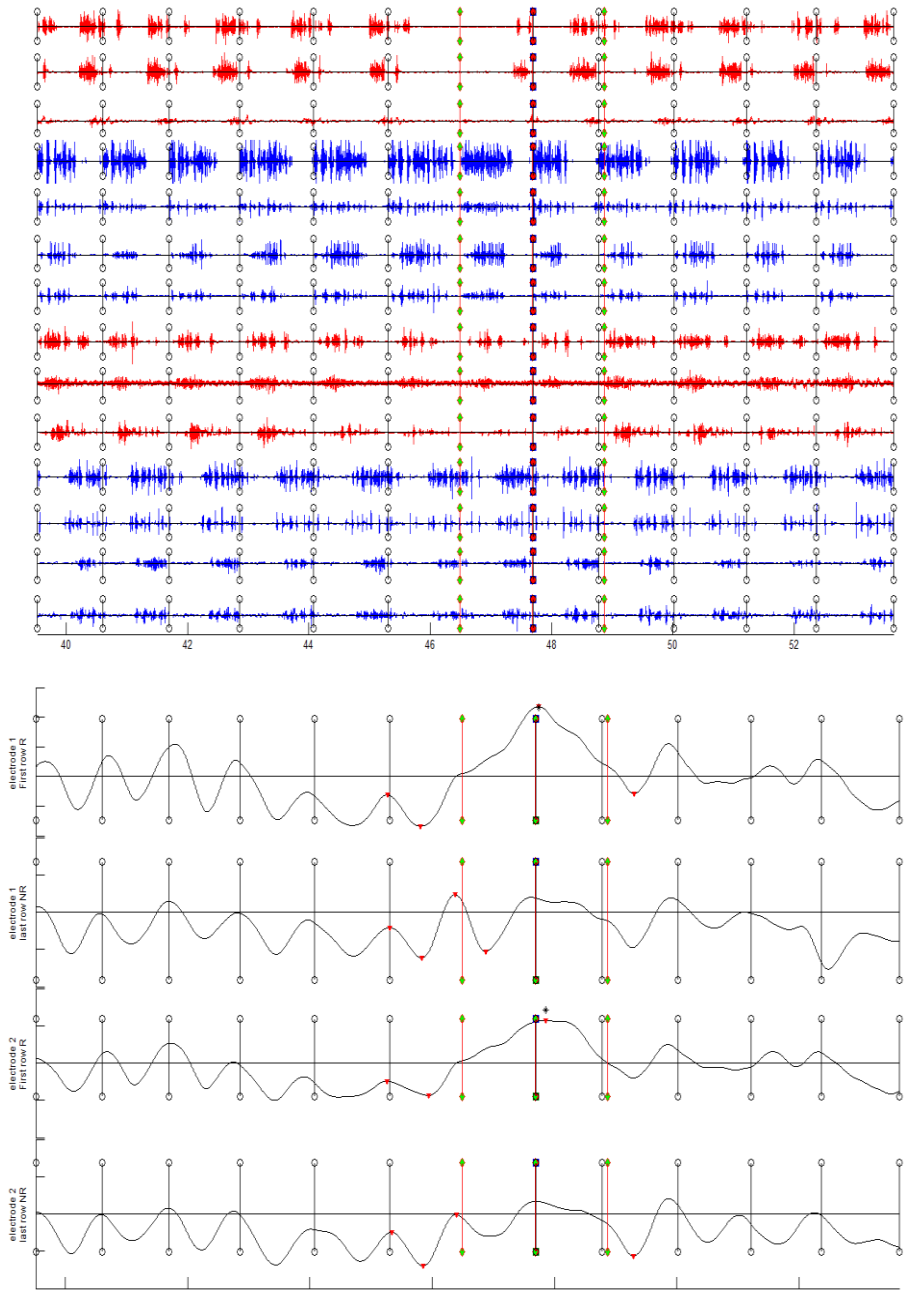

Figure A.6 Unilateral, Non-Resetting Deletion Episode with Tonic/Rhythmic Extensor and Silent Flexor -> Cat ID 011 – Trial 028; deletion number 2

(A) EMG (B) MUA collected from electrode 1 and MUA collected from electrode 2

APPENDIX B

TABLE

Table B.1: Different classifications of deletion episodes

S.No	Muscle type that had Tonic/rythmic EMG activity	Muscle type that had silent/rythmic EMG activity	Side coordination		Phase	Number of deletion episode classified	Phase for each broader groups
1	Extensor	Flexor	Bilateral	Left	Resetting	8	8 Resetting 1 Non-Resetting
					Non Resetting	**1**	
				Right	Resetting	8	
					Non Resetting	**1**	
2	Flexor	Extensor	Bilateral	Left	Resetting	5	5 Resetting 1 Non-Resetting
					Non Resetting	**1**	
				Right	Resetting	5	
					Non Resetting	**1**	
3	Extensor	Flexor	Unilateral	Left	Resetting	2	4 Resetting 2 Non-Resetting
					Non Resetting	**1**	
				Right	Resetting	2	
					Non Resetting	**1**	
4	Flexor	Extensor	Unilateral	Left	Resetting	2	3 Resetting 3 Non-Resetting
					Non Resetting	**3**	
				Right	Resetting	1	
					Non Resetting	**0**	
Resetting -> 20; **Non - Resetting** -> 7							

Table B.2 : Tabulation for Comparing the Amplitude and Phase of MUA (for Spatio-Temporal Analysis) against each other and to Compare Amplitude of MUA with the Phase of EMG

Subject ID - Trial Number	Deletion number	EMG	Electrode 1				Electrode 2				Electrode 1		Electrode 2	
			MUAbr		MUAtr		MUAbr		MUAtr					
			Amplitude	Phase	Amplitude	Phase	Amplitude	Phase	Amplitude	Phase	Depth	RC	Depth	RC
10-010	1	R	R	NR	R	NR	*R*	*R*	*R*	*R*	3000	3	3000	6
	2	R	R	NR	NR	NR	*R*	*R,NA*	*R*	*NR*				
10-013	1	R	NR	NR	R	R	*R*	*R*	*R*	*R*	3000	3	3000	5
10-056	1	R	NR	NR	NR	R	*R*	*NR*	*NR*	*R*	2000	3	2000	4
06-028	1	NR	*R*	*NR*	*R*	*NR*	R	R	R	NR	2700	7	3000	4
06-051	1	R	*NR*	*R*	*R*	*R*	NR	R	R	R	3000	7	2700	4
05-022	1	NR	*NR*	*R*	*R*	*R*	R	NR	NR	R	3500	7	2800	5
	2	R	*R*	*R*	*R*	*R*	R	R	R	R				
05-038	1	NR	*R*	*NR*	*R*	*R*	R	NR	R	R	3500	7	2800	5
	2	NR	*R*	*R*	*R*	*R*	NR	NR	R	R				
	3	R	*R*	*R*	*R*	*NR*	R	R	R	NR				
05-044	1	NR	*R*	*R*	*R*	*R*	R	R	NR	NR	3500	7	2800	5
	2	R	*NR*	*R*	*R*	*NR*	NR	NR	NR	R				
	3	R	*R*	*NR*	*NR*	*R*	NR	R	R	NR				
05-049	1	R	*R*	*NR*	*NR*	*R*	R	NR	NR	R				
11-001	1	R	NR	NR	NR	R	*R*	*NR*	*NR*	*NR*	3000	3	3000	6
11-013	2	R	R	R	R	R	*R*	*R*	*R*	*R*	3000	3	3000	6
	3	R	NR	R	R	NR	*NR*	*R*	*R*	*NR*				
11-023	1	R	NR	R	NR	R	*NR*	*R*	*NR*	*R*	3000	3	3000	6
	2	R	R	NR	NR	NR	*R*	*NR*	*NR*	*NR*				
11-024	1	R	R	R	NR	R	*R*	*R*	*R*	*R*	3000	3	3000	6
	2	NR	NR	R	R	R	*NR*	*R*	*R*	*R*				
11-028	1	R	R	R	R	NR	*R*	*NR*	*NR*	*R*	3000	3	3000	5
	2	NR	NR	NR	R	R	*R*	*NR*	*R*	*R*				
11-035	1	R	R	NR	R	R	*NR*	*R*	*R*	*R*	3000	3	3000	5
	2	R	R	NR	R	NR	*R*	*R*	*NR*	*R*				
	3	R	R	NR	R	R	*R*	*R*	*R*	*R*				

Key:

R - Significant amplitude change
NR - Not significant change in amplitude
R - Reset in Time period, phase
NR - Non-Reset in time period/phase
RC - Lumbar segment

Color code:

For EMG vs MUA Amplitude

EMG	MUA	Color
R	NR	orange
NR	R	Green
R	R	Grey
NR	NR	blue

For MUA Amplitude vs MUA Phase

Amp	Phase	Color
NR	R	orange
R	NR	Green
R	R	Grey
NR	NR	blue

MUAtr - MUA at top row site of the electrode
MUAbr - MUA at bottom row site of the electrode

font in italics - Raustral , normal font - Caudal

Deletion episodes of EMG are marked in different shades of yellow to indicate the crresponding MUA activity to be oscillating (Os) i.e having MUA amp as NR and MUA phase as NR, all the other senarios are considered Not Oscillating (NOs).

Color code for EMG:

- Electrode 1, MUAbr
- Electrode 1, MUAtr
- Electrode 2, MUAbr
- Electrode 2, MUAtr
- Electrode 1, MUAbr & Electrode 2, MUAtr
- Electrode 1, MUAtr & Electrode 2, MUAtr

Electrode 1

MUAtr			MUAbr		
MUA state	EMG	Cases	MUA state	EMG	Cases
Os	R	2	Os	R	3
NOs	R / NR	25	Os	NR	1
			Nos	R / NR	23

Electrode 1

MUAtr			MUAbr		
MUA state	EMG	Cases	MUA state	EMG	Cases
Os	R	2	Os	R	1
Os	NR	1	Os	NR	1
Nos	R / NR	22	Nos	R / NR	25

NS - NR -> MUA Oscillates (Os) during deletion in EMG
NS - R, NS - NR, S - R -> MUA does not Oscillate (Nos) during deletion in EMG

Table B.3: 6-8 Hz MUA Analysis (1 Hz MUA signals)

	EMG	Electrode 1				Electrode 2				Electrode 1 (phase)		Electrode 2 (phase)	
		MUAtr		MUAbr		MUAtr		MUAbr					
		Phase	Amplitude	Phase	Amplitude	Phase	Amplitude	Phase	Amplitude	Muatr	MUAbr	MUAtr	MUAbr
10-010-1	R	R	R	R	R	NR	R	R	R	R	R	NR	R
10-010-2	R	**NR**	**NR**	R	NR	R	NR	R	NR	NR	R	R	R
10-013-1	R	**NR**	**NR**	R	NR	NR	R	R	NR	NR	R	NR	R
10-056-1	R	R	R	NR	R	R	NR	NR	R	R	NR	R	NR
06-028-1	NR	R	NR	R	R	R	R	R	R	R	R	R	R
06-051-1	R	R	R	R	R	R	R	**NR**	**NR**	R	R	R	NR
05-022-1	NR	R	R	R	R	R	R	R	R	R	R	R	R
05-022-2	R	NR	R	NR	R	NR	R	R	R	NR	NR	NR	R
05-038-1	NR	R	NR	R	NR	NR	R	NR	R	R	R	NR	NR
05-038-2	NR	NR	R	NR	R	R	R	R	NR	NR	NR	R	R
05-038-3	R	R	R	NR	R	**NR**	**NR**	R	NR	R	NR	NR	R
05-044-1	NR	R	R	R	NR	R	R	R	R	R	R	R	R
05-044-2	R	R	R	R	NR	R	R	NR	R	R	R	R	NR
05-044-3	R	NR	R	R	R	R	NR	NR	R	NR	R	R	NR
05-049-1	R	R	NR	R	R	NR	R	R	NR	R	R	NR	R
011 -001-1	R	NR	R	NR	R	NR	R	R	R	NR	NR	NR	R
011-013-1	R	R	R	R	R	R	R	R	NR	R	R	R	R
011-013-2	R	NR	R	NR	R	NR	R	R	R	NR	NR	NR	R
011-023-1	R	**NR**	**NR**	NR	R	**NR**	**NR**	NR	R	NR	NR	NR	NR
011-023-2	R	**NR**	**NR**	R	R	**NR**	**NR**	NR	R	NR	R	NR	NR
011-024-1	R	R	R	NR	R	NR	R	NR	R	R	NR	NR	NR
011-024-2	NR	R	R	R	R	R	NR	R	R	R	R	R	R
011-028-1	R	**NR**	**NR**	R	R	NR	R	R	R	NR	R	NR	R
011-028-2	NR	R	R	NR	R	NR	R	NR	R	R	NR	NR	NR
011-035-1	R	R	R	R	NR	R	NR	R	R	R	R	R	R
011-035-2	R	R	NR	R	NR	**NR**	**NR**	NR	R	R	R	NR	NR
011-035-3	R	R	NR	R	NR	R	R	R	R	R	R	R	R

Note:

For MUA Amplitude vs <u>MUA Phase</u>

EMG	MUA	Color
R	NS	blue
NR	R	Green
R	R	Grey
NR	NR	orange

For EMG vs MUA phase

EMG	Phase	Color
R	NR	blue
NR	R	Green
R	R	Grey
NR	NR	orange

Table B.4: Number of cases observed for each combination of EMG (phase) – MUA (phase) activity during the deletion time period for 1Hz MUA signal.

Electrode 1				Electrode 2			
MUAtr				MUAtr			
EMG	MUA	No. of cases	Total no. of cases	EMG	MUA	No. of cases	Total no. of cases
R	R	12	13	R	R	14	16
NR	NR	1		NR	NR	2	
R	NR	8	14	R	NR	6	11
NR	R	6		NR	R	5	
MUAbr				MUAbr			
EMG	MUA	No. of cases	Total no. of cases	EMG	MUA	No. of cases	Total no. of cases
R	R	9	12	R	R	13	17
NR	NR	3		NR	NR	4	
R	NR	11	15	R	NR	6	9
NR	R	4		NR	R	3	

Table B.5: Oscillation state of MUA vs. EMG phase during the deletion time period for 1Hz MUA signal

Electrode 1									
MUAtr					MuAbr				
MUA activity during deletion period of EMG	Phase of deletion episode, EMG	MUA potential role	No of cases	Probability in %	MUA activity during deletion period of EMG	Phase of deletion episode, EMG	MUA potential role	No of cases	Probability in %
No Oscillation	Resetting	RG or PF	18	66.6	No Oscillation	Resetting	RG or PF	17	62.926
No Oscillation	Non-Resetting	PF	7	25.925	No Oscillation	Non-Resetting	PF	6	22.2
Oscillation	Resetting	Neither RG nor PF	2	7.407	Oscillation	Resetting	Neither RG nor PF	3	11.1
Oscillation	Non – Resetting	RG but not PF	0	0	Oscillation	Non – Resetting	RG but not PF	1	3.703
Electrode 1									
MUAtr					MuAbr				
MUA activity during deletion period of EMG	Phase of deletion episode, EMG	MUA potential role	No of cases	Probability in %	MUA activity during deletion period of EMG	Phase of deletion episode, EMG	MUA potential role	No of cases	Probability in %
No Oscillation	Resetting	RG or PF	18	66.6	No Oscillation	Resetting	RG or PF	19	70.37
No Oscillation	Non-Resetting	PF	6	22.2	No Oscillation	Non-Resetting	PF	6	22.2
Oscillation	Resetting	Neither RG nor PF	2	7.407	Oscillation	Resetting	Neither RG nor PF	1	3.703
Oscillation	Non – Resetting	RG but not PF	1	3.703	Oscillation	Non – Resetting	RG but not PF	1	3.703

Table B.6: Location of the Electrodes

Subject ID - Trial Number	Electrode 1	Electrode 2
	Lumbar Segments	Lumbar Segments
10-010	6	3
10-013	5	3
10-023	5	3
10-056	4	3
06-028	4	7
06-051	4	7
11-001	6	3
11-013	6	3
11-023	6	3
11-024	6	3
11-028	5	3
11-035	5	3
05-022	5	7
05-038	5	7
05-044	5	7

Table B.7: Number of cases observed according to the rostrocaudal region as listed in Table B.2 (1Hz signal)

Phase											
Electrode 1						Electrode 2					
Raustral			Caudal			Raustral			Caudal		
EMG	MUA	No	EMG	MUA	No of cases	EMG	MUA	No	EMG	MUA	No of cases
R	R	8	R	R	4	R	R	4	R	R	10
NR	NR	0	NR	NR	1	NR	NR	2	NR	NR	0
R	NR	6	R	NR	2	R	NR	2	R	NR	4
NR	R	2	NR	R	4	NR	R	2	NR	R	2
Amplitude											
Electrode 1						Electrode 2					
Raustral			Caudal			Raustral			Caudal		
EMG	MUA	No	EMG	MUA	No of cases	EMG	MUA	No	EMG	MUA	No of cases
R	NR	6	R	NR	2	R	NR	2	R	NR	6
NR	R	2	NR	R	5	NR	R	3	NR	R	2
R	R	8	R	R	4	R	R	4	R	R	8
NR	NR	0	NR	NR	0	NR	NR	2	NR	NR	0

APPENDIX C

CODE

Function difference_mua

Input: MUA signal and its peak values (both maxpeak and minpeak) along with the desired time period intervals pre and post deletion are fed as the input to the function.

Algorithm

The last pre-deletion time instance and the first post-deletion time instance of the EMG that corresponds to the maxpeak (tmua_h) of the MUA was used to get 5 other time instances of pre and post deletion (predel_h, postdel_h). Similarly, the pre deletion and post deletion periods were determined with respect to the minpeak values. The Peak values during the deletion periods were used for implementing the comparative statistical analysis against the spatio-temporal nature of the pre and post deletion periods. Criteria for selecting good wave period during the deletion were implemented using the Confidence Interval over the average of pre deletion period (tmua_h (predel_h)) activity. Statistical means were adopted to determine MUA's state of oscillation during the deletion period. This was implemented by comparing the amplitude difference found between minpeak and maxpeak during the deletion period with that of the 95% mean confidence interval obtained from the amplitude difference (maxpeak - minpeak) of the pre-deletion period. The peaks that correspond to the amplitude difference that is comparatively greater than that of the mean CI was considered as good peaks (presence of well-defined oscillation).

CI_amp_n = avg_amp - 1.96 * (std_amp / (sqrt (n_amp)))

Similarly, a criteria was set for determining the statistical significance of the time period during the deletion (deletion_time) when compared to the time period of pre-deletion period that is being considered. A peak was considered to be statistically valid in terms of its temporal activity if it was greater than the higher value of 95% mean confidence (CI_time_p) interval of the pre- deletion time periods.

CI_time_p = avg_deltcri+ (1.96 * (std_time / (sqrt (n))))

fin_del_th_ind_t = del_th (deletion_time > CI_time_p)

Where deletion_time (i) = abs (tmua_h (deletion_time_h (i))-tmua_h (deletion_time_h (i+1))), std_time – standard deviation of pre – deletion time period, avg_deltcri – average of pre deletion time period, n – number of samples, del_th, deletion_time_h – time at which maxpeak during deletion occurs, fin_del_th_ind_t – index of statistically significant time instance, tmua_h- MUA time of maxpeak.

Maximum time period of MUA wave (maxpeak – maxpeak) during the deletion of EMG activity was used to compare with the mean confidence interval obtained with the pre deletion time period. If the maximum value of time period (stat_deldiff) during deletion was greater than the higher value of mean CI or lesser than the lower value of mean CI, the deletion episode was said to be Resetting (R).

```
if stat_deldiff > pos_CI || stat_deldiff < neg_CI
  {
        Phase = 'R'
        else phase = 'NR'
  }
```

Function spikephase_method_mua

Algorithm

Similar to the function spikephase_method2 which was implemented to create the required pre - post deletion periods and compute the desired statistical analysis through the function stat, spikephase_method_mua computes the MUA statistical computations through the function stat. Phase difference was calculated by using similar method that was used for EMG for reference.

A. Function used for running the analysis for the chose deletion period trials

```
choose =2;  %1 -> plotwaves, 2-> coherence 3-> emg or any other
numelectrode = 2;
name = {'bev','bobo','pip','woody'}; %set 2 - 1 hz bev,bobo,pip,woody
name1 = {'munch'};

w = {'001','013','023','024','028','035'}; b = {'028','051'};
p = {'022','038','044','049'}; m = {'048'}; be = {'010', '013','056'};
NumOfFreqFilt = 2; % 1Hz filtered and 6-8Hz filtered
wn = 2;
wn2 = [12 16];
range = 'B';
chk_counter = 2;
for freq_num = 1:1: NumOfFreqFilt

   if freq_num == 1
      freq = wn;
   else
      freq = wn2;
   end

   int = 6; % n-1 time interval(for plotting EMG)
   int_mua = 6; % n-1 time intervals (for MUA analysis)
   time_int = 4;% 3 time intervals (for EMG analysis)
   num_plot = 7;
   for i = 1:1:length(name)
      if strcmp(name(i),'woody') == 1
         Trial = w;
      else if strcmp(name(i),'pip') == 1
            Trial = p;
         else if strcmp(name(i), 'bobo') == 1
               Trial = b;
            else if strcmp(name(i), 'munch')==1
                  Trial = m;
               else
                  Trial = be;
               end
```

```
            end
        end
    end

    for j = 1:1:length(Trial)
        name_fin = name(i);
        Trial_fin = Trial(j);
        trialname = strcat(cell2mat(name_fin),cell2mat(Trial_fin));
        display(trialname);
        del_ep = deletion(trialname);%function that has the information about the deletion time for each trial

        for numdel = 1:1:length(del_ep)
            emg = EMGselection(trialname, numdel); %function that has the EMG to be selected for plotting
            [Side, Muscle] = muscle_side(trialname, numdel); %function that has information about the muscle to be marked with.

            if strcmp(trialname, 'woody024')==1
                int = 3;
            else if strcmp(trialname, 'woody013')==1  && numdel == 2
                    int = 2;
                else if strcmp(trialname, 'woody013')==1  && numdel == 1
                        int = 5;
                    else if strcmp(trialname, 'bev010')==1
                            int = 4;
                        else if strcmp(trialname, 'pip044')==1
                                int = 4;
                            else if strcmp(trialname, 'woody023')==1 && numdel ==2
                                    num_plot = 6;
                                else if strcmp(trialname, 'woody035')==1 && numdel ==3
                                        num_plot = 6; int_mua = 5; int = 5;
                                    end
                                end
                            end
                        end
                    end
                end
            end

            plotwaves1(name_fin,Trial_fin,Side,Muscle,emg,numelectrode,del_ep(numdel), int,numdel,time_int, int_mua,choose,freq,num_plot,range);


            if chk_counter > 26 || chk_counter == 26

                range = sprintf('%s%s','A','A'+ abs(chk_counter-26));
            else
                range = sprintf('%s', range+1);
            end
            chk_counter = chk_counter+1;
```

```
        end

      end

   end
range = 'B';
chk_counter = 2;
end
```

B. MUA **pre-processing**
For preprocessing the MUA signal as described previously

1. Function **filteredMUA** (reference chantal's thesis)

```
function filteredMUA (name,Trial,Start,Stop)
trialname = strcat(cell2mat(name),cell2mat(Trial));

data = tdl(trialname);
Fs = data.fsmua;

Hd = butterbp(300,4000,Fs);
points = round(Start*Fs:Stop*Fs);
if points(1)<1, points = points(2:end);
end

RMSres = zeros(2,length(points),2);
size(RMSres);
%% Find all of the channels of a row and average them
for j = 1:2 %Electrode 1 or electrode 2
   dattype = ['MUA' num2str(j)];
   RR = [1 8]; % Row of electrode
   for l = 1:length(RR)
      CC = 0; % Column of electrode
      chans = ElectrodeFinder(RR(l),CC);
      Ecount = length(chans); % Number of electrode sites you would like to average
      mua = NaN(Ecount,length(points));
      for k = 1:Ecount
         dat = data.chan(dattype,chans(k));

         mua(k,:) = rfilt(Hd,comb2(dat(points),60,Fs,15, 0.5));%filter 300-4000Hz
      end
      Mdata = mean(mua);

      S = 2*std(Mdata);  MP = mean(Mdata)+S; MM = mean(Mdata)-S;
      Mdata(Mdata>MP)=MP;  Mdata(Mdata<MM)=MM;%Clip extreme values +-2STD from
mean

      Mdata = RootMeanSquare(Mdata,Fs);%Square, LPF @ 100Hz, SQRT (Stark)
      Mdata = Mdata - mean(Mdata);% Normalize signal by subtracting the mean

      RMSres(j,:,l) = Mdata; %electrode j, row l
```

```
    end

end

filename = [cell2mat(name),cell2mat(Trial),'_MUA_electrode_new.mat'];

filept = [cell2mat(name),cell2mat(Trial),'points_new.mat'];
rmse1 = RMSres(:,:,2);
rmse2 = RMSres(:,:,1);
save(filename,'rmse1','rmse2');
clear rmse1;
clear rmse2;
save(filept,'points');
clear points;
```

2. Function **secondary_filter**

```
%For filtering the data
function [toprow, bottomrow] = secondary_filter(Fs, e1, e2, wn)
      [z,p,k] = butter(4,wn/Fs);
      [sos_var,g] = zp2sos(z, p, k);
      Hd        = dfilt.df2sos(sos_var, g);
      toprow = filter(Hd,e1);
      bottomrow = filter(Hd,e2);
```

C. MUA Analysis:

1. Function difference_mua

```
function [time_cons_h, predel2_tmua, postdel2_tmua, predel2_amp, postdel2_amp, del_critera_ampl, del_critera_amph, del_critera_tl, del_critera_th, predel_amp, postdel_amp, fin_del_th_t,max_del_time,diff_mua,dtmua] = difference_mua(dmua,tmua,tmua_h, tmua_l,peakh,peakl,num_int, tmua_fincoh, decimua_coh,trial_det,varargin)
'difference_function'
fs = 24414;

x_hypothesis = trial_det.x_hypothesis;
req_dat = trial_det.req_dat;

[~, predel_h] = min(abs(tmua_h(:) - req_dat));
[~, postdel_h] = min(abs(tmua_h(:) - x_hypothesis));

predel_l = predel_h;
postdel_l = postdel_h;

% index for coherence calculation
[~, predel_coh] = min(abs(tmua_fincoh(:) - req_dat));
[~, postdel_coh] = min(abs(tmua_fincoh(:) - x_hypothesis));

%index for correlation calculation
[~, predel_coh2] = min(abs(tmua(:) - req_dat));
```

```
[~, postdel_coh2] = min(abs(tmua(:) - x_hypothesis));

%setting the other num_int number of pre deletion maxpk instances
fperiod_h = predel_h  - num_int + 1;
lperiod_h = postdel_h + num_int - 1;
fperiod_l = predel_l  - num_int + 1;
lperiod_l = postdel_l + num_int - 1;
fperiod_coh = predel_coh  - (num_int * fs) + (1 * fs);
lperiod_coh = postdel_coh + (num_int * fs) - (1 * fs);
fperiod_coh2 = predel_coh2  - (num_int * fs) + (1 * fs);
lperiod_coh2 = postdel_coh2 + (num_int * fs) - (1 * fs);

if predel_l == postdel_l
   postdel_l = postdel_l+1;
end

if predel_h == postdel_h
   postdel_h = postdel_h +1;
end

if predel_coh == postdel_coh
   postdel_coh = postdel_coh+1;
end

if predel_coh2 == postdel_coh2
   postdel_coh2 = postdel_coh2 + 1;
end

time_cons_h = [(fperiod_h:predel_h),(postdel_h:lperiod_h)];
time_cons_l = [(fperiod_l:predel_l),(postdel_l:lperiod_l)];
time_cons_numpre  = length(fperiod_h:predel_h);

deletion_time_h =  predel_h: postdel_h;
deletion_time_l = predel_l:postdel_l;
tmua_h(deletion_time_h)
tmua_l(deletion_time_l)
%coherence calculation
predel = fperiod_coh:predel_coh;
postdel = postdel_coh:lperiod_coh;

predel2 = fperiod_coh2:predel_coh2;
postdel2 = postdel_coh2:lperiod_coh2;

sub_del = abs(max(postdel) - length(tmua_fincoh));
if max(postdel)> length(tmua_fincoh)
   post = length(postdel) - sub_del;
   pre = length(predel) - sub_del;
   postdel = postdel(:,1:(post));
   predel = predel (:,1:(pre));
end
```

```
sub_del2 = abs(max(postdel2) - length(tmua_fincoh));
if max(postdel2)> length(tmua_fincoh)
   post2 = length(postdel2) - sub_del2;
   pre2 = length(predel2) - sub_del2;
   postdel2 = postdel2(:,1:(post2));
   predel2 = predel2 (:,1:(pre2));
end

predel_amp = decimua_coh (predel);
postdel_amp = decimua_coh (postdel);
predel2_amp = dmua (predel2);
postdel2_amp = dmua (postdel2);
predel2_tmua = tmua_fincoh (predel2);
postdel2_tmua = tmua_fincoh (postdel2);

tmua_h(:,fperiod_h:lperiod_h);
tmua_l(:,fperiod_l:lperiod_l);

%---- this is used for the output argument difft_muat can be used if required --
mua_cal = horzcat(tmua_h(:,fperiod_h:lperiod_h), tmua_l(:,fperiod_l:lperiod_l));
mua_cal = sort(mua_cal);

for numdiff = 1:1:length(mua_cal)-1
   diff_mua(numdiff) = abs(mua_cal(numdiff) - mua_cal(numdiff+1));
   dtmua(numdiff)              =                          {strcat(num2str(round(mua_cal(numdiff))),'-
',num2str(round(mua_cal(numdiff+1))))};
end

for i=1:1:num_int
   difft_muat(i) = abs(tmua_h(time_cons_h(i)) - tmua_l(time_cons_l(i)));
end

for i= num_int+1:1:length(time_cons_h)
   difft_muat(i) = abs(tmua_h(time_cons_h(i)) - tmua_l(time_cons_l(i)));
end
%-----------------------------------------------------------------

% checking for the criterea using significance level
for i=1:1:length(time_cons_h)
   diff_amp(i) = abs(peakh(time_cons_h(i)) - peakl(time_cons_l(i)));
end
'diff_amp_critera'
diff_amp_pre = diff_amp(1:num_int);
std_amp = std(diff_amp_pre);
n_amp = length(diff_amp_pre);
avg_amp = mean(diff_amp_pre);
% deletion_amp = abs(peakh(deletion_time_h) - peakl(deletion_time_l))
CI  =  1.96 * (std_amp / (sqrt(n_amp)));
CI_amp_n  = avg_amp - CI;

if tmua_h(deletion_time_h(1))> tmua_l(deletion_time_l(1))
```

```
    for i = 1:length(deletion_time_l)-1
        del_right_w = abs(peakh(deletion_time_h(i)) - peakl(deletion_time_l(i+1)));
    end
    del_left_w = abs(peakh(deletion_time_h) - peakl(deletion_time_l));
    del_left_w
    del_right_w
    fin_del_tl_ind(1) = deletion_time_l(1);
    deletion_tl = deletion_time_l(2:end);

    fin_del_tl_indtemp = deletion_tl(del_right_w > CI_amp_n);
    fin_del_tl_ind = [fin_del_tl_ind, fin_del_tl_indtemp];
    fin_del_th_ind = deletion_time_h (del_left_w  > CI_amp_n);

else
    for ii = 1:length(deletion_time_h)-1
        del_left_w = abs(peakh(deletion_time_h(ii+1)) - peakl(deletion_time_l(ii)));
    end
    del_right_w = abs(peakh(deletion_time_h) - peakl(deletion_time_l));
    del_left_w
    del_right_w

    fin_del_th_ind(1) = deletion_time_h(1);
    deletion_th = deletion_time_h(2:end);
    fin_del_th_indtemp = deletion_th(del_left_w > CI_amp_n);
    fin_del_th_ind = [fin_del_th_ind, fin_del_th_indtemp];
    fin_del_tl_ind = deletion_time_l (del_right_w > CI_amp_n);
end

% fin_del_tl_ind = deletion_time_l (del_left_w > CI_amp_n & del_left_w > (avg_amp)/2);
% fin_del_th_ind = deletion_time_h (del_right_w  > CI_amp_n & del_right_w  > (avg_amp)/2);
del_critera_tl = tmua_l(fin_del_tl_ind);
del_critera_th = tmua_h(fin_del_th_ind);
del_critera_ampl = peakl(fin_del_tl_ind);
del_critera_amph = peakh(fin_del_th_ind);
del_critera_indl = find (del_critera_tl >= req_dat & del_critera_tl <= x_hypothesis);
del_critera_indh = find (del_critera_th >= req_dat & del_critera_th <= x_hypothesis);
% del_critera_ampl = peakl(fin_del_tl_ind(del_critera_indl));
% del_critera_amph = peakh(fin_del_th_ind(del_critera_indh));

% critera for time period - deletion oscillation selection
for i=1:1:length(time_cons_h)-1
    diff_time(i) = abs(tmua_h(time_cons_h(i)) - tmua_h(time_cons_h(i+1)));
end
'diff_time time period critera'
std_time = std(diff_time(1:num_int));
n_time = length(diff_time(1:num_int));
avg_deltcri = mean(diff_time(1:num_int));
CI_time_p  = avg_deltcri+ (1.96 * (std_time / (sqrt(n_time)) ));

for i = 1:1:length(deletion_time_h)- 1
    deletion_time(i) = abs(tmua_h(deletion_time_h(i)) - tmua_h(deletion_time_h(i+1)));
```

```
end
'deletion_time'
deletion_time
del_th = deletion_time_h(2:end);
fin_del_th_ind_t = del_th (deletion_time > CI_time_p);
fin_del_th_t = tmua_h(fin_del_th_ind_t);

% ------------------for statistical analysis ----------------------
% if length(del_critera_ampl) == 1
%     max_del_time = 'ND';
%     avg_del_time = 'ND';

% del_critera_th = tmua_h(fin_del_tl_ind(del_critera_indh))
if isempty(del_critera_th) == 1
   max_del_time = [];
   avg_del_time = [];
else if length(del_critera_th) == 1
       max_del_time = del_critera_th;
       avg_del_time = del_critera_th;
   else
       for i = 1:1:length(del_critera_th)- 1
          stat_deletion_time(i) = abs(del_critera_th(i) - del_critera_th(i+1));
       end
       max_del_time = max(stat_deletion_time);
       avg_del_time = mean(stat_deletion_time);

   end
end

if length(varargin) >= 1
   component = trial_det.compt;
   range = trial_det.range;
   trialname = trial_det.trialname;
   freq = trial_det.freq;
   numelec= trial_det.numelec;
   table_label = {'trialname', 'del_critera_th'};
   if isempty(del_critera_th) == 1
       stat_deletion_time = sprintf('%s','null');

   else if length(del_critera_th) == 1
           stat_deletion_time = sprintf('%s','NA');
       else
           stat_deletion_time = stat_deletion_time';
       end
   end
   del_critera_th = num2str(del_critera_th')

   table_val          =          {trialname;num2str(max_del_time);          num2str(avg_del_time);
num2str(CI_time_p)};
   table_val = vertcat( table_val, str2num(del_critera_th), stat_deletion_time)
   filename = ['difference_mua','_',num2str(freq/2),component,'.xls'];
```

```
    range2 = sprintf('%s%d', range,1);
    xlswrite(filename, table_val, numelec, range2);
    xlswrite(filename, table_label', numelec, 'A1');
end
del_critera_th = str2num(del_critera_th);
if nargout  < 14
    clear diff_mua;
    clear dtmua;
end
```

2. Function **spikephase_method_mua**

```
function [phase_diff, Range_CI, expt_int, avg_t, diff_wave] = spikephase_method_mua(trial_det,
time_cons_mua, tmua_h, int_mua, del_ep, avg_deldiff)
'spikephase_method_mua'
trialname = trial_det.trialname;
numdel = trial_det.numdel;        %deletion number
compt = trial_det.compt;          %whether top row or bottom row
range = trial_det.range;          %for saving results in excel file
numelec = trial_det.numelec;      %electrode number
freq = trial_det.freq;            %filtering frequency
diff_wave = zeros(1,(int_mua-1)); %difference between successive time of maxpk

    for next_t=1:1:int_mua-1 %int_mua no. of maxpk time before predeletion maxpk time
        diff_wave(next_t)              =              abs(tmua_h(time_cons_mua(next_t+1))              -
tmua_h(time_cons_mua(next_t)));
    end

    for next_t= int_mua+1:1:(length(time_cons_mua)-1) %int_mua no. of maxpk time before
postdeletion maxpk time
        diff_wave(next_t-1)            =              abs(tmua_h(time_cons_mua(next_t+1))              -
tmua_h(time_cons_mua(next_t)));
    end

    diff_wave_l  = diff_wave(1:int_mua);              %predeletion , left side of the deletion period
    diff_wave_r  = diff_wave(int_mua:length(diff_wave));%postdeletion

    avg_l  = mean(diff_wave_l);
    avg_r  = mean(diff_wave_r);
    std_l  = std(diff_wave_l);
    std_r  = std(diff_wave_r);

    ttest_2  = ttest2(diff_wave_l,diff_wave_r);
    diff_int  =  abs(tmua_h(time_cons_mua(int_mua))  -  tmua_h(time_cons_mua(int_mua+1)));
%difference between postdeletion maxpk time and predeletion maxpk time

    avg_t  = avg_l;  %averge of int_mua-1 no. of post and pre deletion time period, in this case
only pre-deletion is used
```

```
   std_t  = std_l;  %Std dev of int_mua-1 no. of post and pre deletion time period, in this case only pre-deletion is used
   n = int_mua;
   [phase_diff, phase_diff_av, expt_int, CI, Upper_CI, Lower_CI, Range_CI, phase] = stat(diff_int,std_t,avg_t,n, compt, avg_deldiff);
   table_label = {'name','numdel','del_ep', 'avg_l', 'avg_r', 'std_l', 'std_r', 'avg_t', 'std_t', 'expt_int', 'ttest_2','phase_diff', 'phase_diff_av', 'diff_int', 'CI', 'Phase', 'Upper_CI', 'Lower_CI'};
   table_obs = {[trialname,'-',num2str(numdel)], num2str(del_ep), num2str(avg_l), num2str(avg_r), num2str(std_l), num2str(std_r), num2str(avg_t), num2str(std_t), num2str(expt_int), num2str(ttest_2), num2str(phase_diff), num2str(phase_diff_av), num2str(diff_int), num2str(CI), phase, num2str(Upper_CI), num2str(Lower_CI)};

   if strcmp(compt, 'MUAtr') == 1
      component = 'MUAtr';
   else
         component = 'MUAbr';
   end

   filename = ['Observation_spikephase_method2','_',num2str(freq/2),component,'.xls'];
   range2 = sprintf('%s%d', range,1);
   xlswrite(filename, table_obs', numelec, range2);
   xlswrite(filename, table_label', numelec, 'A1');
end
```

4. Function **expected_val_mua**

```
function [x_expton_fin, x_hypothesis, y_expt, predel_mua, ttest_another] = expected_val_mua(tdata, expt_int, time_cons,avg_t,time_int, diff_wave)
'expected_val_mua' %To determine the hypothesized maximum peak time period based on the average of pre and post peak time period.

del_ubound = time_cons(time_int);     % index of maximum peak corresponding to pre-deletion EMG onse value
del_lbound = time_cons(time_int+1);  % index of maximum peak corresponding to post-deletion EMG onse value

aa = expt_int * avg_t; % aa is difference btw pre deletion maxpk time and post deletion maxpk
%expt_int and avg_t is obtained from the function spikephase_method_mua
if aa == abs(tdata(time_cons(time_int)) - tdata(time_cons(time_int+1))) %to verify deletion time period obtained from previous steps
   display('matched');
else
   display('no match');
end
inter_exp = ceil(expt_int);            %hypothesized time period of maxpk during deletions
x_expton_0 = tdata(time_cons(time_int)); %dummy variable for swapping
x_expton_fin = zeros(inter_exp,1);      %time of hypothesized maxpk
y_expt = ones(size(x_expton_fin));      %amplitude at x_expton_fin
```

```
for e = 1:1:inter_exp
   x_expton_c = avg_t + x_expton_0;
   x_expton_0 = x_expton_c;
   x_expton_fin(e) = x_expton_0;

end
diff_last_int = abs(x_expton_fin(end-1)- del_lbound);
ttest_another = ttest2(diff_last_int, diff_wave);    %to verify the above calculations
predel_mua = tdata(del_ubound);   %pre-deletion maxpk time
x_hypothesis = (avg_t * expt_int) + tdata(time_cons(time_int)); %calculating the post-deletion time to verify the entire caculation
```

5. Function **req_time_mua**

```
function [time_cons_mua_h, first_part, last_part] = req_time_mua(x_hypothesis,tmua_h, del_ep,num_int)
'req_mua'

[~, predel_h] = min(abs(tmua_h(:) - del_ep));
[~, postdel_h] = min(abs(tmua_h(:) - x_hypothesis));

fperiod_h = predel_h  - num_int+1;
lperiod_h = postdel_h + num_int-1;

time_cons_h = [(fperiod_h:predel_h),(postdel_h:lperiod_h)];
time_cons_mua_h = time_cons_h;


   first_part = fperiod_h;
   last_part = lperiod_h;
```

6. Function **stat**

```
function [phase_diff, phase_diff_av, expt_int, CI, neg_CI, pos_CI, Range_CI,phase] = stat(diff_int,std_t,avg_t,n, compt, stat_deldiff)
%output from spikephase_method2 or spikephase_method_mua
%std_t - std deviation of the post and the pre deletion time period (from
%either spikephase_method_2 for EMG analysis or spikephase_method_mua
%avg_t - avg of the post and the pre deletion time period
%n - no. of samples
%compt - top row or bottom row
%diff_int - difference between immediate predel time and post del time
%stat_deldiff - measure of deletion time period to compare with CI(difference_mua function)

if nargin ==5
   stat_deldiff = 0;
end

expt_int    = max (diff_int, avg_t)/min (diff_int, avg_t); %expected interval or hypothesized onset/maxpk time during deletion
```

```
phase_diff = mod(expt_int, floor(expt_int));
phase_diff_av  = phase_diff *avg_t;
CI = 1.96* ((std_t/sqrt(n)));
neg_CI = avg_t - CI;
pos_CI = avg_t + CI;6
Range_CI = [neg_CI,' - ',pos_CI];

if strcmp(compt,'MUAtr') ==1 || strcmp(compt, 'MUAbr')==1
   %     if strcmp(stat_deldiff,'ND') == 1
   %         phase = 'ND';
   %     else
   if isempty(stat_deldiff) ==1
      phase = 'R,NA';
   else if stat_deldiff > pos_CI || stat_deldiff < neg_CI
      phase = 'R';
   else
      phase = 'NR';
      end
   end
   %     end
else
   if phase_diff > CI
      phase = 'R';
   else
      phase = 'NR';
   end
end
```

7. Function **coherence_eval**

```
function coherence_eval(trial_det,sig1, sig2, tmua1, tmua2, predel_amp, postdel_amp,
mua_label)
 numdel = trial_det.numdel;
 numelec = trial_det.numelec;
 choose = trial_det.choose;
 filt_freq = trial_det.freq;
 range = trial_det.range;
 trialname = trial_det.trialname;

if length(filt_freq) ==1
myfolder = [num2str(filt_freq/2),'Hz_atlast'];
else
   myfolder = [num2str(filt_freq(1)/2),'-',num2str(filt_freq(2)/2),'Hz_atlast'];
end
subfolder = [cell2mat(trial_det.name)];
sub_subfolder = [cell2mat(trial_det.trial)];


% edited using the matlab example
Fs = 40000;
Fsampling = 24414;
```

```
T = 1/Fsampling;

sig1 = sig1';
sig2 = sig2';

[Cxy,f] = mscohere(predel_amp,postdel_amp,[],[],[],Fs);
[Pxy,F]     = cpsd(predel_amp,postdel_amp,[],[],[],Fs);
phase   = -angle(Pxy)/pi*180;

for numfreq = 1:1:length(filt_freq)
[Cval(numfreq), Cind(numfreq)] = min(abs(f(:)-filt_freq(numfreq)));
coherence_estimate = Cxy(Cind);
[Pval(numfreq), Pind(numfreq)] = min(abs(F(:)-filt_freq(numfreq)));
coherence_phase = phase(Pind);
end
%[gxx,gxxc,f1] = pwelch(predel_amp,[],[],Fs,'ConfidenceLevel',.95);

% gyy = pwelch(postdel_amp,[],[],Fs,'ConfidenceLevel',.95);
% [gxy,gxyc,f2] = csd(predel_amp,postdel_amp,[],Fs,[],[],.95);
% gxxyy = gxx.*gyy;
% length(f1)
% length(f2)
% coh= abs(gxy).^2 / gxxyy;
% phase1 = atan2(-imag(gxy),real(gxy)) * 180/pi;

[sig_lev, sig_lev_f, df, freq ] = cohere_bootstrap_signif_level(predel_amp, postdel_amp, .95);

figure
subplot(411);
plot(f,Cxy, '-o');
title('Coherence Estimate');
grid on;
axis([0 20 0 1])
subplot(412);
plot(F,phase);
title('Cross Spectrum Phase (deg)');
grid on;
xlabel('Frequency (Hz)');
axis([0 20 -180 180])

suptitle(['coherence calculation at ',num2str(filt_freq/2), '  ', mua_label, '  ', trialname,' electrode ',
num2str(numelec),' deletion ', num2str(numdel)])
subplot (413);
plot(tmua1,sig1,tmua2, sig2);
legend('predeletion', 'postdeletion');
if length(sig1) < length(sig2)
   lrr = length(sig1)-1;
else
   lrr = length(sig2) -1;
end
subplot(414);
```

```
[Xcf, lag, bounds] = crosscorr(sig1,sig2, lrr);
[max_corr, points_lag] = max(Xcf);
points_sec = T*lag(points_lag);
if length(filt_freq) ==1
   del_f = filt_freq/2;
else
   [locpkh,pkh] = peakfinder(sig1,0.001,[],1);
   time_period = abs(tmua1(locpkh(1)) - tmua1(locpkh(2)));
   del_f = 1/time_period;
end
lag_deg = 360*(del_f)*points_sec;
deg_pts = lag_deg/100;
crosscorr(sig1,sig2, lrr)

title(['Cross function ', num2str(filt_freq/2), ' by correlation amp ',num2str( max_corr), ' for ',
num2str(points_sec), 'deg',num2str(deg_pts)]);
filenameimg                                                                                    =
[trialname,'_',num2str(filt_freq/2),'Hz','_','electrode',num2str(numelec),mua_label,'_coherence_cal
c_del_',num2str(numdel),'.pdf']
 saveas(gcf,['D:\',fullfile(myfolder,subfolder,sub_subfolder,filenameimg)]);

if choose == 1
   img_pr_3 = gcf;
   set(img_pr_3,'PaperPositionMode','auto');
   set(img_pr_3,'PaperOrientation','landscape');
   set(img_pr_3,'Position',[50 50 1200 800]);
   print(gcf)
   clf;
else
   display('mua_plot is not written in xls neither is it printed');
end

figure
plot(freq,sig_lev_f);
ylabel('coherence')
xlabel('frequency')

title(['correlation calculation at ', num2str(filt_freq/2) ,'   ', mua_label, '   ', trialname,' electrode ',
num2str(numelec),' deletion ', num2str(numdel)]);
filenameimg2                                                                                   =
[trialname,'_',num2str(filt_freq/2),'Hz','_','electrode',num2str(numelec),mua_label,'_correlation_c
alc_del_',num2str(numdel),'.pdf'];
 saveas(gcf,['D:\',fullfile(myfolder,subfolder,sub_subfolder,filenameimg2)]);

 if choose == 1
    img_pr_4 = gcf;
    set(img_pr_4,'PaperPositionMode','auto');
    set(img_pr_4,'PaperOrientation','landscape');
    set(img_pr_4,'Position',[50 50 1200 800]);
    print(gcf)
```

```
    clf;
 else
    display('mua_plot is not written in xls neither is it printed');
 end
filenamestore = ['Coherence_calculation',num2str(filt_freq/2),mua_label,'.xls'];
range2 = sprintf('%s%d', range,1);
if length(filt_freq)==1
coh_var = {'coherence_est', 'cohere_phase', 'cross_func', 'points_sec', 'degree'};
coh_data = {trialname, num2str(coherence_estimate), num2str(coherence_phase),
num2str(max_corr), num2str(points_sec), num2str(deg_pts)};
else
   coh_var = {'trialname','cohere_est(1)', 'cohere_phase(1)','cohere_est(2)',
'cohere_phase(2)','cross_func', 'points_sec', 'degree'};
   coh_data = {trialname,num2str(coherence_estimate(1)), num2str(coherence_phase(1)),
num2str(coherence_estimate(2)), num2str(coherence_phase(2)),num2str(max_corr),
num2str(points_sec), num2str(deg_pts)};
end

xlswrite (filenamestore, coh_var', numelec, 'A1');
xlswrite (filenamestore, coh_data', numelec, range2);
```

D. for plotting the required results: function **Plotwaves**

```
%% Plotting function for correlating the MUA with its EMG
%%
function
plotwaves1_better_version(name,Trial,side,Side_mark,muscle,emgvar,numelectrode,del_ep,
num_int,numdel,time_int,int_mua,choose,freq, num_plot,range)

numrow = 2;
%% _____ EMG plotting with their predicted onset _____
if Side_mark == 1
   side_markreq = {'L'};
else
   side_markreq = {'R'};
end
lside = length(side);
lemg = length(emgvar);
SubplotInd= lemg*lside;

for numside = 1:1:lside
   if side(numside) == 1
      side_req = {'L'};
   else if side(numside) == 0
         side_req = {'R'};
      end
   end
   if numside == 1
      sub = 0;
   else if numside == 2
         sub = length(emgvar);
```

```
    end
  end

  if length(freq) ==1
    myfolder = [num2str(freq/2),'Hz_atlast'];
  else
    myfolder = [num2str(freq(1)/2),'-',num2str(freq(2)/2),'Hz_atlast'];
  end

  subfolder = cell2mat(name);
  sub_subfolder = cell2mat(Trial);
  trialname = [cell2mat(name),cell2mat(Trial)];

  trial_det.trialname = trialname;
  trial_det.numdel = numdel;
  trial_det.range = range;
  trial_det.freq = freq;
  trial_det.choose = choose;
  trial_det.name = name;
  trial_det.trial = Trial;

  [Start, Stop, steponset] = datareq(name,Trial, Side_mark, muscle);
  compt = 'EMG';
  trial_det.compt = compt;

  [time_cons, phase_diff, phase, CI, no_int_expt, avg_t, req_dat, label, extra_pre,
diff_wave_emg] = spikephase_method2(steponset, time_int, del_ep, trial_det);
  [x_expton_fin, x_hypothesis, y_expt,~] = expected_val (steponset, no_int_expt, time_cons,
avg_t,time_int, extra_pre, diff_wave_emg);

  [steponsetp_plot, f_period, l_period] = req_time_per(steponset, num_plot, del_ep);


  lstep = length(steponsetp_plot);
  steponsetp  = steponsetp_plot(1:(lstep),:);

  for numemg = 1:1:lemg

    if strcmp(emgvar(numemg),'SA')==1 ||
strcmp(emgvar(numemg),'BP')==1||strcmp(emgvar(numemg),'TA')==1
      co = 'r';
    else
      co = 'b';
    end

    data = tdl(trialname);
    emg_req = [cell2mat(side_req),cell2mat(emgvar(numemg))];
    demg = data.chan('EMGs',emg_req);
    temg = data.t;
    [~,indexstart] = min(abs(temg - Start));
```

```
        [~,indexstop] = min(abs(temg - Stop));
        temg_fin = temg(indexstart:indexstop);
        demg = demg(indexstart:indexstop);

        figure(1);
        hold on;

        h(numemg+sub) = subaxis(SubplotInd,1,numemg+sub, 'Spacing', 0.0001, 'Padding', .001,
'Margin', 0.13);
        plot(temg_fin,demg,co);
        ylabel(emg_req,'FontSize',7, 'Rotation',360,'Color','w');
        mxdemg = max(demg);
        mndemg = min(demg);
        ylim ([mndemg mxdemg]);
        mtit(['EMG of ',trialname, ' deletion episode ',num2str(numdel),'=', num2str(req_dat),'  ',
cell2mat(label),' with ',phase,' marked with ',cell2mat(side_markreq),muscle])
        hold on;

        minp = min(demg)*ones(size(steponsetp))/1.5;
        maxp = max(demg)*ones(size(steponsetp))/1.5;
        y_exptonu = min(demg)*y_expt/1.5;
        y_exptonl = max(demg)*y_expt/1.5;
        y_hypothesisu = max(demg)*ones(size(x_hypothesis))/1.5;
        y_hypothesisl = min(demg)*ones(size(x_hypothesis))/1.5;

        %___ plotting for each emg _____
        stem(x_expton_fin, y_exptonu, 'diamondr','MarkerFaceColor','green',
'MarkerEdgeColor','red');
        stem(x_expton_fin, y_exptonl, 'diamondr','MarkerFaceColor','green',
'MarkerEdgeColor','red');
        stem(x_hypothesis, y_hypothesisu, 'MarkerFaceColor','red','marker','square');
        stem(x_hypothesis, y_hypothesisl, 'MarkerFaceColor','red','marker','square');
        stem(steponsetp,minp,'k');
        stem(steponsetp,maxp,'k');
        xlim ([steponset(f_period) steponset(l_period)]);
    end
end
    linkaxes(h,'x');
    set(h(1:SubplotInd),'yticklabel',[]);
    set(h(1:SubplotInd),'ycolor','w');
    set(h(1:SubplotInd),'box','off');
    set(gcf,'color','w');
    set(h(1:SubplotInd-1),'xticklabel',[]);
    set(h(1:SubplotInd-1),'xcolor','w');
    filenameemg =
['EMG_predicted_onset_',trialname,'_',num2str(freq/2),'Hz','_del',num2str(numdel),'.jpg',];
    saveas(gcf,['D:\',fullfile(myfolder,subfolder,sub_subfolder,filenameemg)]);
    clf;

%%      _____MUA_____
```

```
for numelec = 1:1:numelectrode
   % location_det = Location_dat(name,Trial,numelec);

      if strcmp(trialname,'munch048') == 1 || strcmp(trialname,'woody001') == 1 ||
strcmp(trialname,'woody028') == 1 || strcmp(trialname,'pip038') == 1 || strcmp(trialname,'pip044')
== 1 ...
            || strcmp(trialname,'bobo028') == 1 || strcmp(trialname,'bobo051') == 1
         filename = [trialname,'_MUA_electrode.mat'];
         filep = [trialname,'points.mat'];
      else
         filename = [trialname,'_MUA_electrode_new.mat'];
         filep = [trialname,'points_new.mat'];
      end
      x = load(filename);
      xp = load(filep);
      points = xp(1).points;
      if numelec == 1,
         e1 = x(1).rmse1(1,:);
         e2 = x(1).rmse1(2,:);
      else
         e1 = x(1).rmse2(1,:);
         e2 = x(1).rmse2(2,:);
      end

      tmua = data.tmua(points);
      Fs = data.fsmua;
      wnn = 100;

      [toprow, bottomrow] = secondary_filter(Fs, e1, e2, freq);
      [tmua_fin, decimuatr, decimuabr] = mua_fin(temg_fin,tmua, toprow, bottomrow);

      [toprow_coh, bottomrow_coh] = secondary_filter(Fs, e1, e2, wnn);
      [tmua_fincoh,decimuatr_coh, decimuabr_coh] = mua_fin(temg_fin,tmua, toprow_coh,
bottomrow_coh);

      [loctrh,pktrh] = peakfinder(decimuatr,0.001,[],1);
      [loctrl,pktrl] = peakfinder(decimuatr,0.001,[],-1);
      tmua_trh = tmua_fin(loctrh);
      tmua_trl = tmua_fin(loctrl);

      [locbrh,pkbrh] = peakfinder(decimuabr,0.001,[],1);
      [locbrl,pkbrl] = peakfinder(decimuabr,0.001,[],-1);
      tmua_brh = tmua_fin(locbrh);
      tmua_brl = tmua_fin(locbrl);

      dtmua_h = abs(size(tmua_trh)-size(tmua_brh));
      dpk_h = abs(size(pktrh)-size(pkbrh));
      dtmua_l = abs(size(tmua_trl)-size(tmua_brl));
      dpk_l = abs(size(pktrl)-size(pkbrl));

      diff_det.dtmua_h = dtmua_h;
```

```
    diff_det.dtmua_l = dtmua_l;
    diff_det.dpk_h   = dpk_h;
    diff_det.dpk_l   = dpk_l;

    if length(tmua_trh)> length(tmua_brh)
       tmua_brh = padarray(tmua_brh,dtmua_h,'post');
       pkbrh = padarray(pkbrh,dpk_h,'post');
    else
       tmua_trh = padarray(tmua_trh,dtmua_h,'post');
       pktrh = padarray(pktrh,dpk_h,'post');
    end

    if length(tmua_trl)> length(tmua_brl)
       tmua_brl = padarray(tmua_brl,dtmua_l,'post');
       pkbrl = padarray(pkbrl,dpk_l,'post');
    else
       tmua_trl = padarray(tmua_trl,dtmua_l,'post');
       pktrl = padarray(pktrl,dpk_l,'post');
    end

    MUA = vertcat(decimuatr,decimuabr);
    MUA_hipk_time = vertcat(tmua_trh,tmua_brh);
    MUA_lopk_time = vertcat(tmua_trl,tmua_brl);
    MUA_hipk_amp = vertcat(pktrh,pkbrh);
    MUA_lopk_amp = vertcat(pktrl,pkbrl);
    MUA_coh = vertcat(decimuatr_coh,decimuabr_coh);

%% Computing the wave characteristics with respect to the MUA
trial_det.x_hypothesis = x_hypothesis;
trial_det.req_dat = req_dat;
trial_det.numelec = numelec;

%          _____ Computation for the first row _______
for row = 1:1:numrow

if numelec == 1
    nume = 0;
else
    nume = numrow;
end
    minp = min(MUA(row,:))*ones(size(steponsetp))/1.5;
    maxp = max(MUA(row,:))*ones(size(steponsetp))/1.5;
    y_exptonu = min(MUA(row,:))*y_expt/1.5;
    y_exptonl = max(MUA(row,:))*y_expt/1.5;
    y_hypothesisu = max(MUA(row,:))*ones(size(x_hypothesis))/1.5;
    y_hypothesisl = min(MUA(row,:))*ones(size(x_hypothesis))/1.5;

    if row ==1
       compt = 'MUAtr';
    else
       compt = 'MUAbr';
```

```
        end
        trial_det.compt = compt;

        [tmua_cons_mua,fpart_tmua, lpart_tmua, fpart_amp, lpart_amp, fin_del_pl, fin_del_ph,
fin_del_tl, fin_del_th, predel_amp, postdel_amp, ...
            time_h,avg_amp, avg_amp_post, avg_amp_del, stat_deldiff] = ...
            difference_mua(MUA(row,:),tmua_fin,MUA_hipk_time(row,:), MUA_lopk_time(row,:),
MUA_hipk_amp(row,:),MUA_lopk_amp(row,:),num_int,tmua_fincoh, MUA_coh(row,:),
trial_det,diff_det);

        [ph_diff_mua,phase, CI_mua, expt_int_mua,  avg_t_mua, diff_mua] =
spikephase_method_mua(trial_det,tmua_cons_mua, MUA_hipk_time(row,:),
num_int,del_ep,stat_deldiff);
        mua_det = [avg_amp, avg_amp_del,avg_amp_post];
        [~,first_half, second_half] = req_time_mua(x_hypothesis,MUA_hipk_time(row,:), del_ep,
int_mua);
        tmuaf_period = first_half;
        tmual_period = second_half;
        %[x_expton_fin_mua_tr, x_hypo_mua_tr, y_expt_mua_tr, predel_muaplottr, ttest_muatr] =
expected_val_mua (tmua_trh, expt_int_mua_tr, tmua_cons_mua_tr, avg_t_mua_tr,num_int,
diff_muatr);


    %%      ---------- peak analysis visulation ----------

    %       _____________________ MUA plot _______________

    if row ==1
        label = 'First row';
    else
        label = 'last row';
    end

    figure(2);

    g(nume+row) = subaxis(4,1,nume+row, 'Spacing', 0.0001, 'Padding', .001, 'Margin', 0.13);
    set(g(nume+row),'ycolor','w')
    title(['MUA',' & CI val = ', CI_mua]);
    plot(tmua_fin, MUA(row,:),'k')
    ylabel ({['electrode ',num2str(numelec)]; [label,' ' , phase]},'FontSize',9,'Color','k');
    hold on;
        hold all;
        stem(x_hypothesis, y_hypothesisu, 'MarkerFaceColor','red','marker','square');
        stem(x_hypothesis, y_hypothesisl, 'MarkerFaceColor','red','marker', 'square');
        stem(x_expton_fin, y_exptonu, 'diamondr',
'MarkerFaceColor','green','MarkerEdgeColor','red');
        stem(x_expton_fin, y_exptonl,
'diamondr','MarkerFaceColor','green','MarkerEdgeColor','red');
        stem(steponsetp,minp, 'k');
        stem(steponsetp,maxp, 'k');
        plot(fin_del_th, fin_del_ph,'vg','MarkerFaceColor','r','MarkerSize',5,'MarkerEdgeColor','r')
```

```
        plot(fin_del_tl, fin_del_pl,'vg','MarkerFaceColor','r','MarkerSize',5,'MarkerEdgeColor','r')
        amph = max(MUA(row,:))/1.25*ones(length(time_h));
        plot(time_h, amph, 'k*');

%stem(predel_muaplottr,(max(decimuabr)*ones(size(predel_muaplotbr))),'rs','MarkerFaceColor',
'r');

%         Xlimp = MUA_hipk_time(row,:);
%         if freq/2 == 1
%            xlim ([Xlimp(tmuaf_period+1) Xlimp(tmual_period)]);
%         else
          xlim ([steponset(f_period) steponset(l_period)]);
%         end

          %     minp = min(MUA(row,:))*ones(size(mua_p));
          %     maxp = max(MUA(row,:))*ones(size(mua_p));
          %     y_exptonu_mua = min(MUA(row,:))*y_expt_mua;
          %     y_exptonl_mua = max(MUA(row,:))*y_expt_mua;
          %     y_hypou_mua = max(MUA(row,:))*ones(size(x_hypo_mua));
          %     y_hypol_mua = min(MUA(row,:))*ones(size(x_hypo_mua));

          %     stem(x_hypo_mua, y_hypou_mua, 'MarkerFaceColor','red','marker','square');
          %     stem(x_hypo_mua, y_hypol_mua, 'MarkerFaceColor','red','marker','square');
          %     stem(x_expton_fin_mua, y_exptonu_mua, 'diamondr',
'MarkerFaceColor','green','MarkerEdgeColor','r');
          %     stem(x_expton_fin_mua, y_exptonl_mua,
'diamondr','MarkerFaceColor','green','MarkerEdgeColor','r');
          %     stem(mua_p,minp);
          %     stem(mua_p,maxp);



    if nume+row == numelec+numrow;
        linkaxes([g,h],'x');
        set(g(1:4),'yticklabel',[]);
        set(g(1:4),'box','off');
        %set(h(SubplotInd+1:SubplotInd+nume+row),'xticklabel',[]);
        set(g(1:4),'xticklabel',[]);
        set(gcf,'color','w');
        set(g(1:3),'xcolor','w');
        suptitle(['MUA plotted ',num2str(freq/2),'Hz','
',cell2mat(name),cell2mat(Trial),'del',num2str(numdel)]);
        filenamemua =
['MUA_plotted_with_emg_onset',trialname,'_',num2str(freq/2),'Hz','_del_',num2str(numdel),'_Pe
akAnalysis.jpg'];
        saveas(gcf,['D:\',fullfile(myfolder,subfolder,sub_subfolder,filenamemua)]);
        clf;
          if choose == 1
             img_pr_3 = gcf;
             set(img_pr_3,'PaperPositionMode','auto');
             set(img_pr_3,'PaperOrientation','landscape');
```

```
            set(img_pr_3,'Position',[50 50 1200 800]);
            print(gcf)

        else
        display('mua_plot is not written in xls neither is it printed');
        end
    end

    %%     ------------ Time difference Bar chart ----------------
    figure(3);
    subplot (numrow+numelectrode,1,nume+1);
    bar (mua_det);
    title('MUA for top row')
    subplot(numrow+numelectrode,1,nume+2);
    bar (mua_det);
    title('MUA for bottom row')
    suptitle (['time differece ',num2str(freq/2),'Hz','  ',cell2mat(name),cell2mat(Trial),...
       'del',num2str(numdel)]);

    filenamehist = [trialname,'_',num2str(freq/2),'Hz','_del',num2str(numdel),'_PeakAnalysis".pdf'];
    saveas(gcf,['D:\',fullfile(myfolder,subfolder,sub_subfolder,filenamehist)]);

    %% -------------------- Coherence Analysis ---------------------------
    coherence_eval(trial_det,fpart_amp, lpart_amp, fpart_tmua, lpart_tmua,
predel_amp,postdel_amp, row);


    end
end
```

E Function mua_fin

```
function [tmua_fin,decimuatr, decimuabr] = mua_fin(temg_fin, tmua,
toprow, bottomrow)
        %Normalising the MUA dimension with respect to EMG dimension
        %temg_fin -> emg time value, tmua - MUA time value, toprow and
        %bottomrow - MUA value first and last rows respectively
        decifactor = length(tmua)/length(temg_fin);
        decimuatr = decimate(toprow,ceil(decifactor));
        decimuabr = decimate(bottomrow,ceil(decifactor));
        tmua_fin = decimate(tmua,ceil(decifactor));
```