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Tendon reflex protocol for exploring the mechanisms associated with whole body vibration

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ABSTRACT

TENDON REFLEX PROTOCOL FOR EXPLORING THE MECHANISMS ASSOCIATED WITH WHOLE BODY VIBRATION

By

Ikechukwu Okeke

Whole body vibration (WBV) machines have in recent years been widely used as rehabilitation equipment. Whole body vibration has been shown to have positive effects on muscle response. Cerebral palsy (CP) is a neurodevelopmental condition that includes a group of non-progressive, but often changing, motor impairment syndromes arising in the early stages of human development. The aim of this project was to design a protocol and related instruments to assess the tendon reflex in response to WBV. Both a press pedal used for measuring the force exerted by the foot and a hammer used for measuring the stimulating force were designed in this study. In addition to the design of the instruments, a protocol for their use was developed in this study. The protocol was tested on a group of 7 subjects not afflicted with CP. The results show no significant change in reflex latency, electromechanical delay, EMG magnitude, nor force output from the foot. These results agree with other published studies [8]. The methods and instrumentation introduced in this project will be used in more comprehensive studies to assess the effect of WBV in CP patients.

TENDON REFLEX PROTOCOL FOR EXPLORING THE MECHANISMS ASSOCIATED WITH WHOLE BODY VIBRATION

by
Ikechukwu Okeke

A Thesis
Submitted to the Faculty of
New Jersey Institute of Technology
in Partial Fulfillment of the Requirements for the Degree of
Masters of Science in Biomedical Engineering

Department of Biomedical Engineering

May 2015

APPROVAL PAGE

TENDON REFLEX PROTOCOL FOR EXPLORING THE MECHANISMS ASSOCIATED WITH WHOLE BODY VIBRATION

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I dedicate this work to the inspiring people of Ladacin Network and Singer House.

ACKNOWLEDGMENT

First and foremost, I must thank my thesis advisor, Dr. Richard Foulds, for all of his guidance and support. I am truly grateful for all of his patience, enthusiasm, and wisdom. I would also like to thank Dr. William Hunter and Dr. Sergei Adamovich for their invaluable input serving on my committee. I am indebted to Peter Michael of the Neuromuscular and Rehabilitation Engineering Lab for his mentorship and support. Thanks is also due to John Hoinowski of the Biomedical Engineering Department for both his advice and his assistance with all technical aspects of machining. I thank all of the students in the Neuromuscular and Rehabilitation Engineering Lab for their assistance. This work would not have been possible without the individuals mentioned here. I also acknowledge the National Science Foundation (NSF) Bridge to Doctorate Fellowship for financial support during my study. I want to thank Mr. Victor Okeke, my dad, and Mrs. Eunice Okeke, my mom, for their prayers and support. Finally thanks to my brothers and sisters: Ijeoma, Nwamaka, Emeka, Chika, Ebele, and Chukwudum for their encouragements.

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CHAPTER 1

INTRODUCTION

1.1 Background Information

Whole body vibration (WBV) machines have in recent years been widely used as rehabilitation equipment. While comprehensive research on the specific targets of WBV is still underway, demonstrated benefits of WBV treatment include: a greater range of motion in targeted limbs, and increased muscle strength and muscle tone [4,6]. These are indicative of reduced threshold in the muscle spindles. The assessment of tendon reflexes is an important part of examining and diagnosing neurological and neuromuscular disorders such as cerebral palsy (CP) [1]. Cerebral palsy is a well-recognized neurodevelopmental condition that includes a group of non-progressive, but often changing, motor impairment syndromes arising in the early stages of human development [3]. Currently, it affects approximately 2 out of every 1000 live births in the United States [2]. There is no known cure for the condition, but physical and occupational therapy may help. This study provides insight into the effects of whole body vibration as a possible treatment for cerebral palsy. Some CP individuals experience spasticity, which is characterized by hypersensitivity to velocity [7], dystonia, which is characterized by an uncontrolled or an involuntary co-contraction of both the agonist and antagonist muscle [9], or a combination of both dystonia and spasticity. In the CP population, vestibular stimulation was shown to reduce spasticity [7] but not dystonia. However, a pilot study from a neuromuscular engineering lab at NJIT (unpublished data, Michael and Foulds) show that dystonia was greatly reduced

for longer than 60 seconds. In this study, the researchers vibrated the subject, sat them down, and performed a pendulum knee drop (PKD), which showed considerably effect after at least five minutes. After the initial stimulation, they performed vestibular stimulation lasting 15 minutes followed by another PKD. The effect of the WBV was evident after the 15 minute stimulation. The effect of reducing dystonia after one session of 4 one-minute WBV treatments was at least 30 minutes (unpublished data, Michael and Foulds).

These results reported above are consistent with work of Ness and Field-Fote [11] who showed lasting effects (on the order of months) of multiple sessions of WBV on subjects with what they defined as spasticity due to spinal cord injury. However, based on the reported PKD data, we believe this form of spasticity is actually dystonia.

Other research in our lab has shown that vestibular simulation has a minor but significant effect on non-disabled subjects. If WBV engaged the same mechanism as vestibular stimulation, we would expect to see an effect on non-disabled subjects. If WBV engages a different mechanism related to enhancing reciprocal inhibition, we would not expect to see much change since non-disabled people have functioning reciprocal inhibition.

In summary, our lab has shown that vestibular stimulation reduces classical CP spasticity through hypersensitivity of the stretch reflex of velocity dependent stretching. The possible mechanism, which is still being investigated, is a reduction in the sensitivity of the agonist stretch reflex. Our lab has also shown that WBV reduces dystonia in CP via excessive co-contraction of agonist and antagonist muscles. The possible mechanism for this is the enhancement of reciprocal inhibition.

1.2 Objective

This project was the first step in designing and testing the protocol and equipment for the studies on the effect of WBV on CP subjects. The specific aim of this project was to design a protocol for data collection, build an apparatus, and test the apparatus and protocol on able-bodied individuals.

To assess the effect of WBV on agonist muscles (the soleus), a group of individuals without CP was used as subjects. The activity of their soleus muscle upon stimulation of the Achilles tendon was measured before and after WBV using a Delsys EMG system (Figure B.1). The tapping force and the planter flexion force due to Achilles tendon tap before and after WBV were collected using an instrumented hammer and a press pedal with force sensors. We expected to see no effect on the non-disabled subjects since they will have function reciprocal inhibition.

This data will be used in a future study wherein individuals with CP will be subjected to the same analysis. It is our hope that this information can be used to better understand the physiological basis of WBV induced reduction in dystonia and thereby leading to the development of treatments for patients suffering from cerebral palsy.

CHAPTER 2

METHODS

2.1 Hammer Modification and Calibration

A study from Lai Kuan et al. stated that reflex stimuli (stretching) can be affected by numerous factors such as tapping force and position of hammer impact [5]. For this reason, the instrumented hammer was developed to ensure that a consistent force was used for every given tapping stimulation. To make this, a Taylor percussion hammer was cut vertically and a Phidget sensor was inserted in between the handle and the hammer head as shown in Figure 2.1. The modified Taylor percussion hammer was calibrated and then validated using various weights and a Phidget GUI. Validation was performed by plotting the output (measured mass) against the input (known mass). Calibration was accepted if results were consisted as seen in Figure 2.2.

Figure 2.1 figure showing the instrumented hammer.

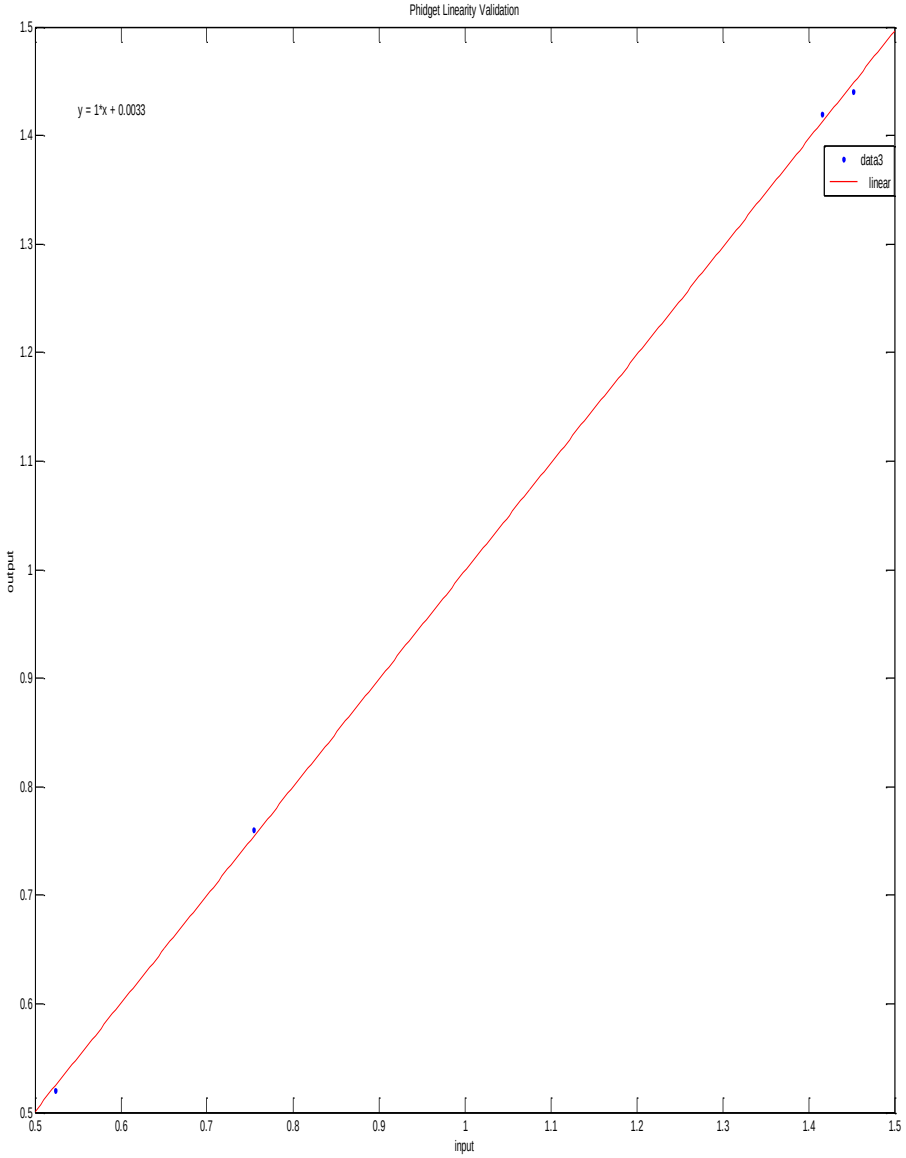

Figure 2.2 Plot showing that the force output from the instrumented hammer is linear.

2.2 Delsys-Hammer Synchronization

The Delsys EMG system was synchronized with the instrumented Taylor percussion reflex hammer using MATLAB software. This was done so that data can be collected by MATLAB from the Delsys system and the instrumented hammer simultaneously. The code used for the synchronization is shown in Appendix C.

2.3 Press Pedal Design

A press pedal was made out of a Phidget, plastic, and 80/20 t-slotted aluminum, as shown in Figure 2.3. The press pedal was calibrated and then validated using various weights and a Phidget GUI. Validation was performed by plotting the output (measured mass) against the input (known mass). Calibration was accepted as the results were consisted as shown in Figure 2.4. This press pedal was necessary to ensure that the amount of force applied on the foot pre and post stimulation was the same.

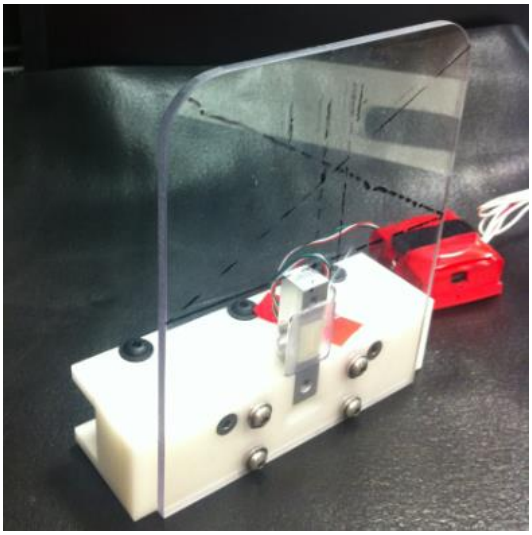

Figure 2.3 figure showing the Press Pedal.

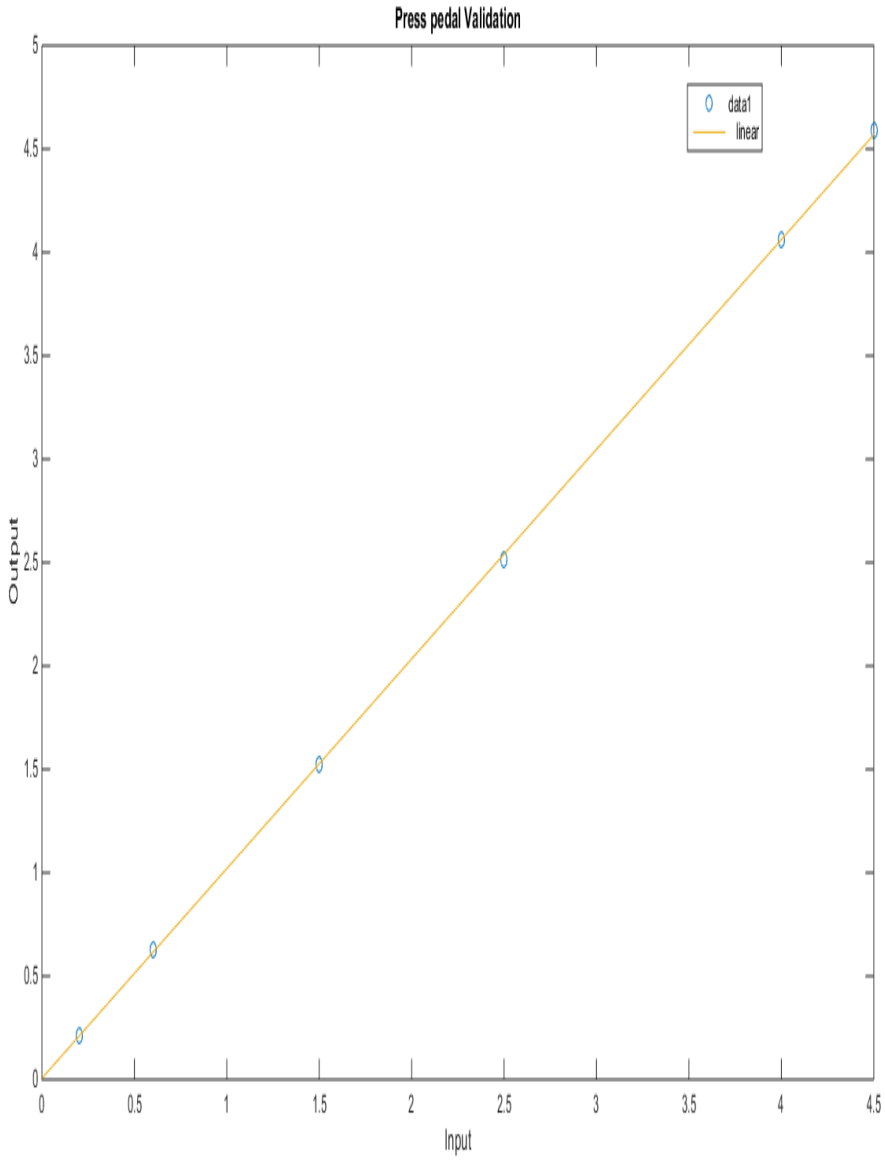

Figure 2.4 Plot showing that the force output from the press pedal is linear.

2.4 Participants

A total of [7] subjects (27yrs ± 3) were recruited to join in this study. Six men and 1 woman were recruited after obtaining approval from the Institutional Review Board

(IRB) and their physicians. Before the session began, the purpose of the study was explained to each subject. A demonstration followed before asking for their consent to participate. They were also given the choice to opt out at any point in the studies.

2.5 Procedure

First, the total body weights and segment lengths of each subject's feet and legs were recorded. One Delsys electrode with a disposable skin to electrode interface was placed on the subject's soleus and one reference electrode directly on the lateral malleolus. The electrode was then connected to a channel on the Delsys system. Subjects laid in a prone position with their feet hanging off the table (Figure 2.6). To access the Achilles' tendon, the foot was dorsiflexed using the press pedal to put a constant tension on the tendon. When comfortable, the Achilles' tendon was tapped 5-8 times with approximately 5 second intervals with the modified Taylor percussion hammer (Figure 2.7). Force and EMG signals were measured for each tap. After this, subjects stood on the XG10 Vibrating platform (Figure B.2). This provided vertical oscillations at 35 Hz. The subjects were stimulated on the platform for one minute, for a total of (4) times with one minute of seated rest in between. After vibration, the tapping of the soleus muscle was repeated, as described above. Figure 2.5 shows the three stages of the study. In the first stage, the Achilles tendon is tapped and the EMG activity and planter flexion force were recorded from the soleus muscle and the press pedal respectively. In stage two, the subject is exposed to WBV, and lastly in stage three the EMG activity of the soleus muscle was once again recorded.

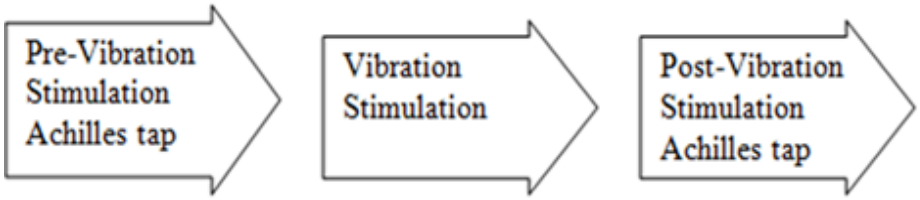

Figure 2.5 The three stages of stimulation in this study.

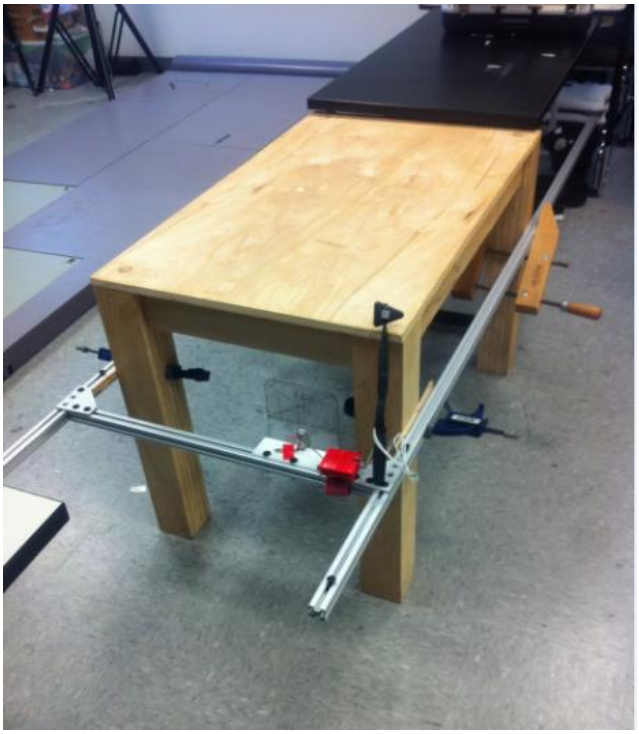

Figure 2.6 Experimental setup.

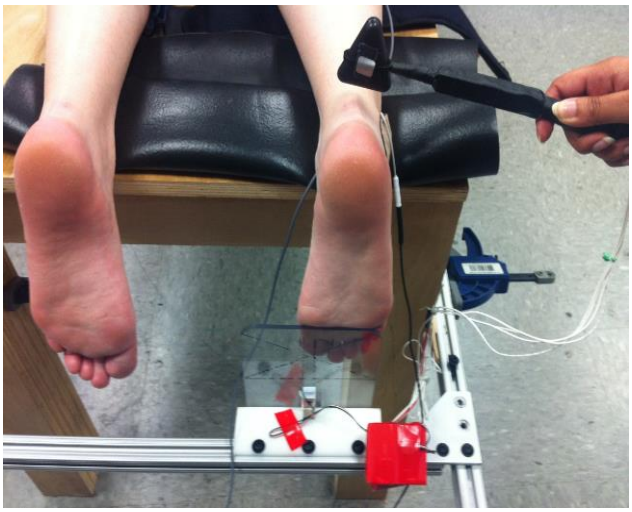

Figure 2.7 Tapping procedure.

2.6 Data Collection

Data from the instrumented hammer and EMG system (Delsys) were recorded to a desktop PC through MATLAB. The electromyography (EMG) system was used to read the voltages generated during muscle activation, while the instrumented hammer and press pedal were used to measure applied forces.

CHAPTER 3

3.1 RESULTS

3.1.1 Data Analysis

A sample plot of a session in the study is shown in Figure 3.1. Figure 3.2 is a zoomed-in version showing one trial from the session. From this, Reflex latency, electromechanical delay, EMG amplitude and force output were calculated. Reflex latency was measured by subtracting the onset or activation of EMG from the onset of the hammer tap. Electromechanical delay was measured by subtracting the onset of the force output from the onset of the EMG activation as shown in Figure 3.1. The EMG amplitude and force output were directly measured by taking the respective maximum values pre and post stimulation.

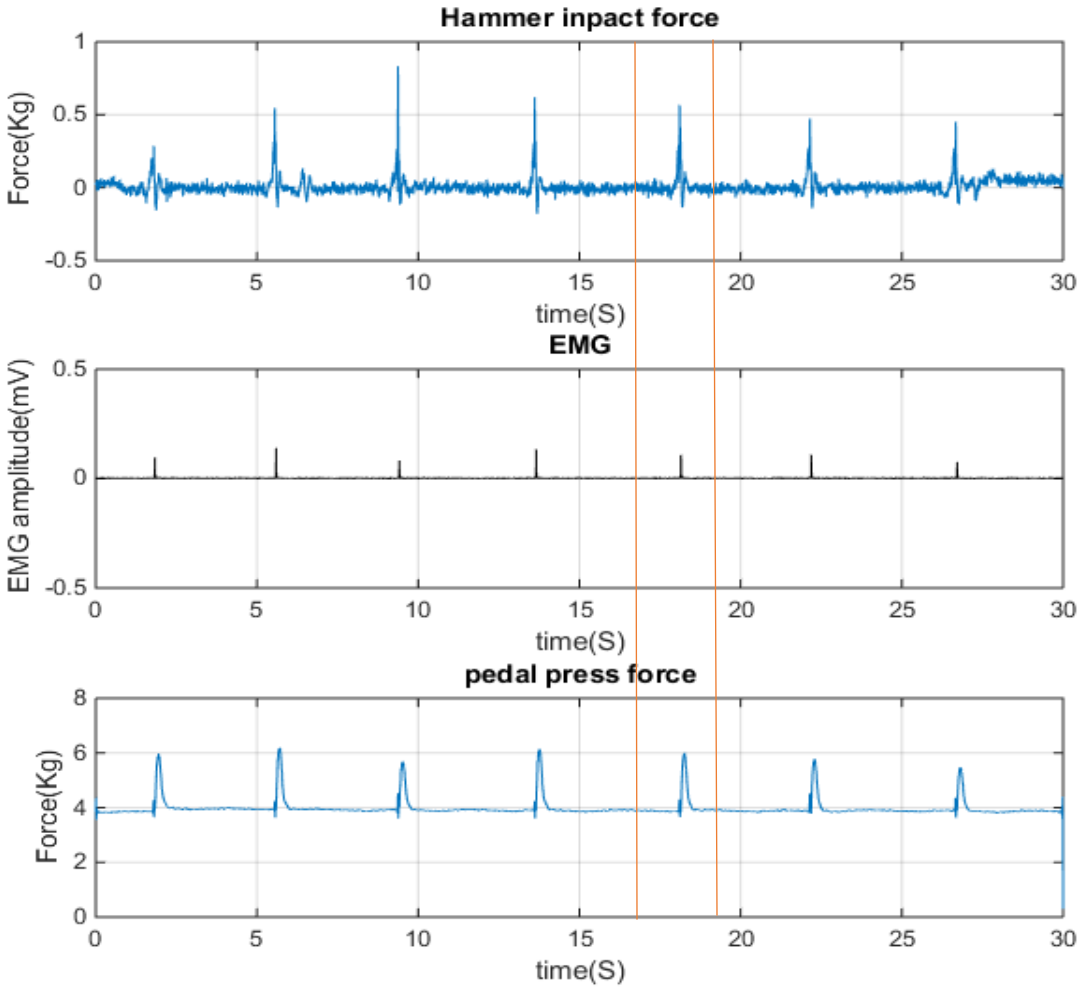

Figure 3.1 Trial plot showing tapping force, EMG signal and Reflex force from top to bottom respectively.

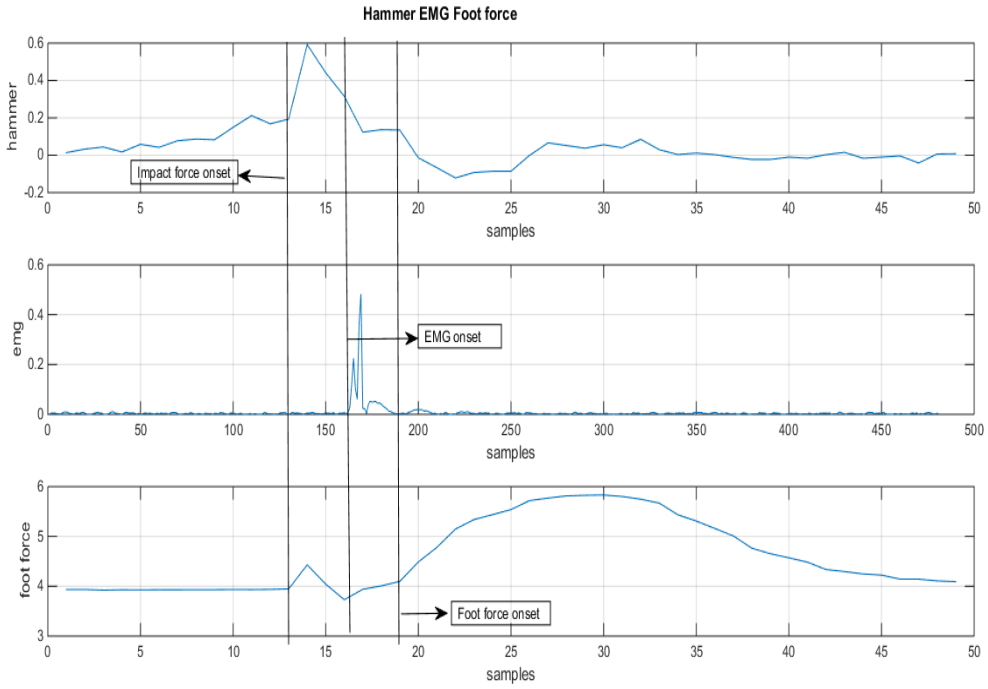

Figure 3.2 Plot showing sample trace of the hammer onset, EMG onset aswell as reflex onset.

Data in Table 3.1 depicts the mean of the pre and post stimulation of the measured factors (EMG amplidude, Reflex latency, Force output and Electromechanical delay).

Table 3.1 Summarized Data Showing the Effect of the Whole Body Vibration.

Subject	EMG Amplitude		Reflex Latency (mS)		Force Output (Kg)		Electromechanical Delay (mS)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	0.514	0.5227	31.5714	27.5000	1.9907	1.7372	21.2857	34.1667
2	0.6851	0.7358	22.0000	44.60	1.1867	1.3956	44.0000	11.4000
3	0.3964	0.4466	34.8333	31.4286	1.4843	1.6724	25.1667	18.5714
4	0.514	0.3481	17.6000	46.6000	3.5265	3.4519	17.6000	25.700
5	0.4089	0.6271	24.2000	45.7143	3.5265	2.875	33.8000	33.833
6	0.5683	0.5545	38.6667	23.2222	0.7436	0.8972	24.6667	27.4444
7	0.4755	0.5333	31.1667	40.3333	1.5547	1.0027	22.1667	19.6667

3.1.2 Statistical Analysis.

To analyze the data collected from the 7 subjects, we used a Bonferonni correction. The Bonferroni correction is used for assessing the probability of at least one significant result in multiple hypotheses being tested simultaneously. The Bonferonni correction was used in the calculation of the p and T values shown in table 3.2.

Table 3.2 Summarized Data Showing the Effect of the Whole Body Vibration

Subject	EMG Amplitude		Reflex Latency (mS)		Force Output (Kg)		Electromechanical Delay (mS)	
	T-val	p-val	T-val	p-val	T-val	p-val	T-val	p-val
1	3.532	0.165	2.173	0.264	3.037	0.307	-3.823	0.594
2	-1.374	0.162	-4.995	0.497	-1.680	0.757	5.245	0.896
3	-1.312	0.707	2.050	0.096	-4.614	0.607	1.545	0.054
4	-0.824	0.022	-4.924	0.708	0.265	0.041	-2.991	0.148
5	-1.626	0.82	-13.85	0.528	-0.112	0.797	-0.091	0.842
6	-0.688	0.492	4.150	0.107	-0.332	0.711	-2.569	0.278
7	3.551	0.484	-0.738	0.538	2.745	0.06694	-3.823	0.594

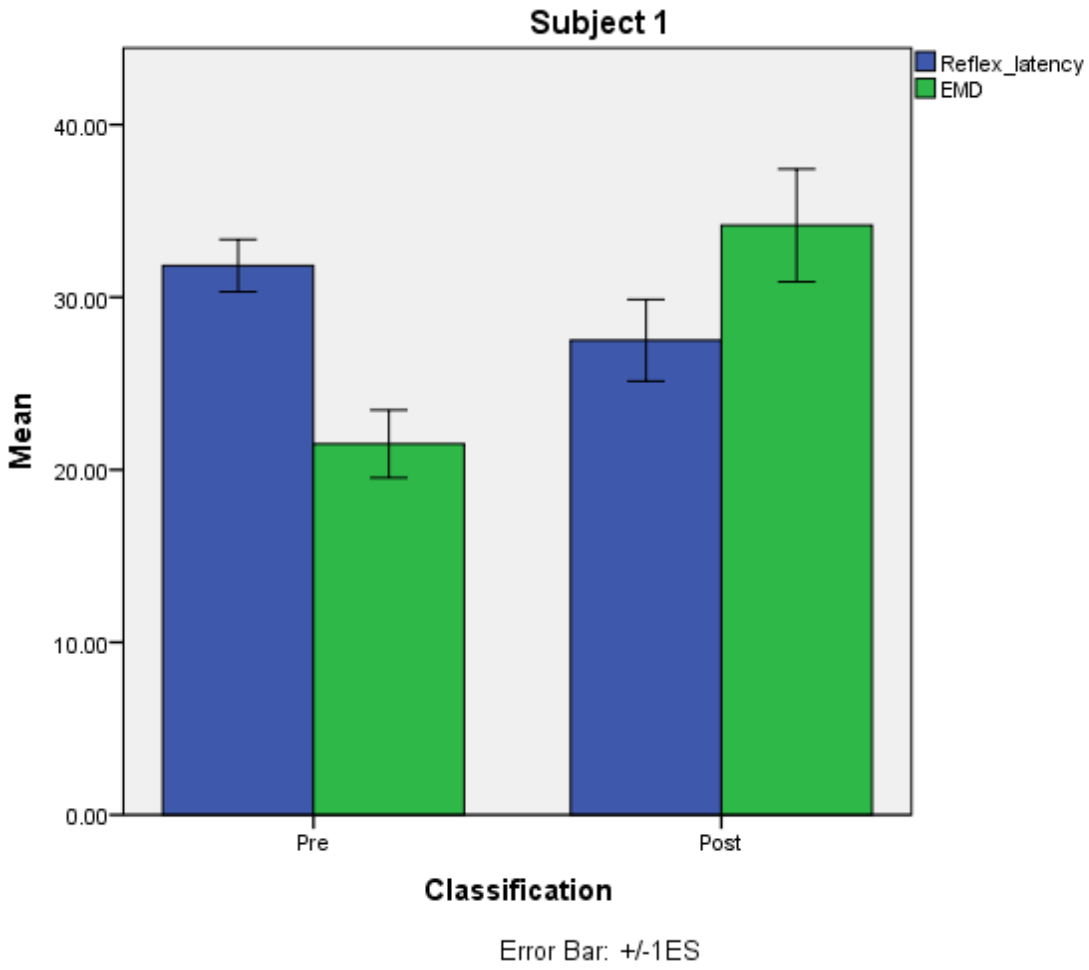

Figure 3.3 Mean reflex latency and EMD in subject 1.

For this particular subject there was a significant decrease in reflex latency and a significant increase in EMD in post simulation. Other subjects, shown in Appendix A, showed varied results. Error bars indicate +/- 1 standard error.

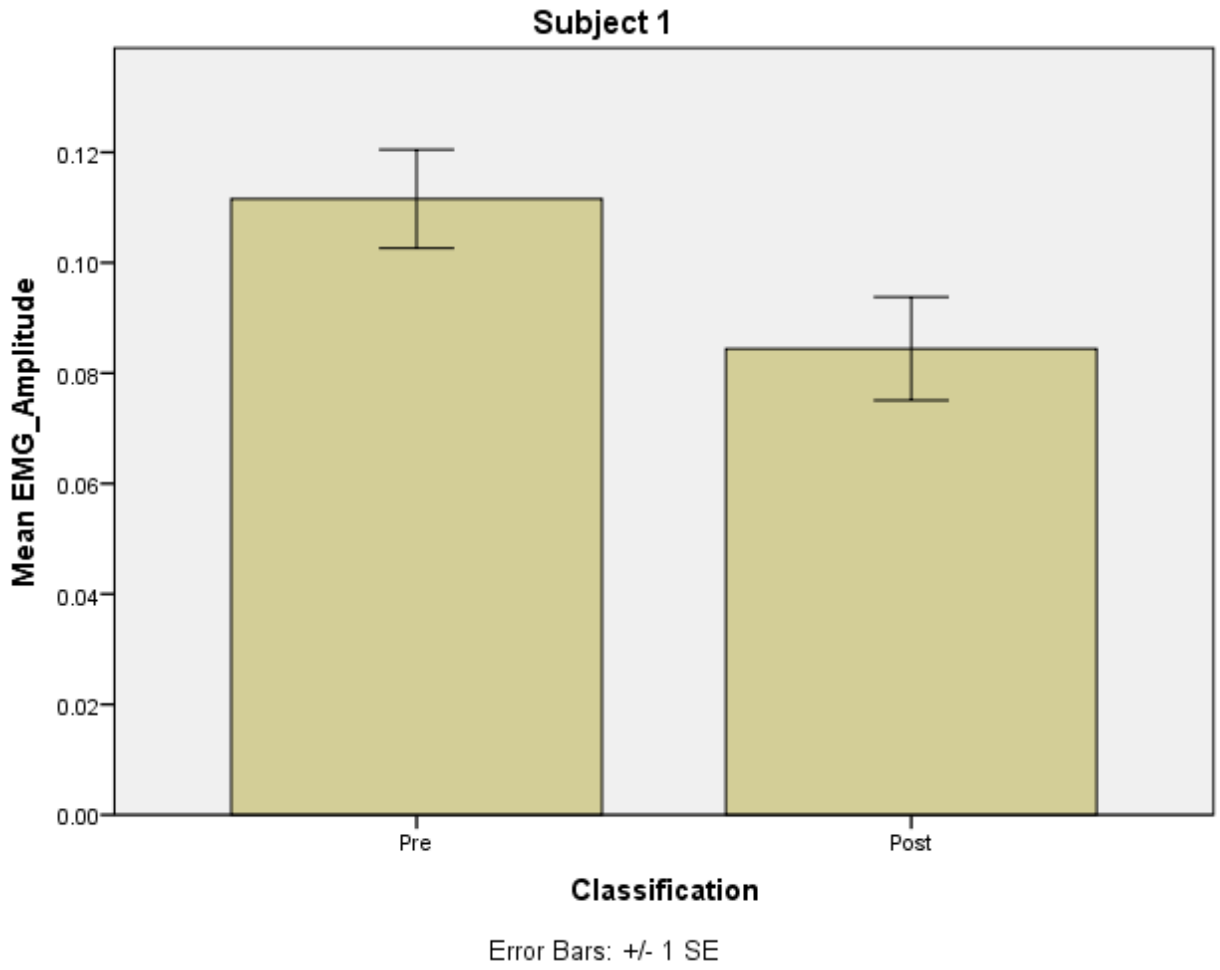

Figure 3.4 Mean EMG amplitude for subject 1.

For this subject, EMG amplitude decreased post stimulation. Other subjects, shown in Appendix A, showed varied results. The error bars indicate the decrease in EMG amplitude is significant. Error bars indicate +/- 1 standard error.

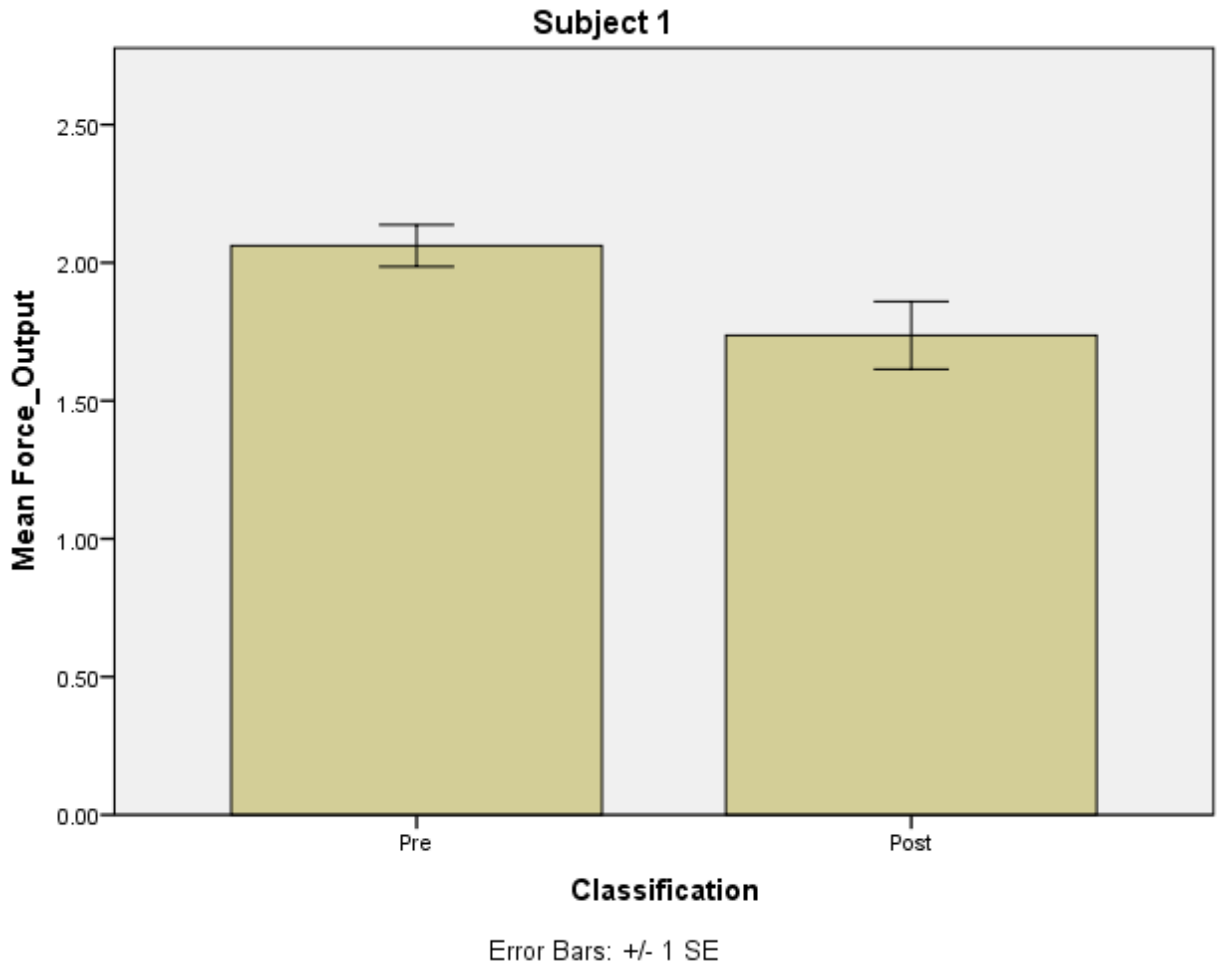

Figure 3.5 Mean force output in subject 1.

The small error bars indicate little deviation from the mean. For this subject, mean force output decreased after stimulation. Other subjects, shown in Appendix A, showed varied results.

CHAPTER 4

4.1 DISCUSSION

For this project, the effect of WBV on lower extremities of able-bodied individuals was studied. The only variable that was consistently affected by WBV was EMG amplitude. Table 3.1 shows that five out of the seven subjects had an increased EMG amplitude post-WBV, however they did not achieve significance.

The results reveal that WBV did not have an effect on reflex latency, electromechanical delay, nor force output from the foot. Almost half of the subjects showed increased delay while the rest showed decreased delay. These results agree with findings reported by Hopkins et al. [8]. The results shown in Tables 3.1 and 3.2 suggest no conclusive difference among the seven subjects in these three variables.

There are several explanations as to why our results may show no changes. First, all of the subjects used in this study are able-bodied and not afflicted with CP or any other disorder. Therefore, since they do not experience co-contractions in their agonist and antagonist muscles, we did not expect to see significant changes. Secondly, according to Sayenko et al., [10], the effects of WBV diminish after 60 seconds. In this study, post-WBV data collection was performed well after 60 seconds had passed. This was due to the time required to ensure that the force used to dosiflex the foot pre and post stimulation were equal.

Thirdly, it is possible that some error may have been introduced in the act of tapping to stimulate the Achilles tendon. It is important to tap at a consistent point with a consistent force at all times, and to achieve this we physically marked the target on the

subject. However, due to the inherent error in manual tapping, it was close to impossible to tap at the exact location with the same magnitude of force with every tap delivered. This may explain why significant changes were not observed. Lastly, it is possible that the subjects were not fully relaxed during testing. This would lead to involuntary contraction of their muscles. Yet, Ness and Field-Fot [11] and our lab both reported significant changes in pendulum knee drop trajectories which indicates a reduction in Dystonia for up to 30 minutes. Therefore, another mechanism not reflected by these variables may be involved. Studies [8, 12, 13, 14] mention that WBV could enhance reciprocal inhibition in the antagonist muscle, which would be consistent with a reduction in co-contraction.

There are several ways to improve this system. In a technical aspect, instead of using a Phidget with 100 frames per second, a more sensitive device i.e. an optoforce sensor can be used to obtain more accurate measurements. The optoforce sensor has a sampling rate of 1000 frames per second. This would allow us to accurately measure the electromechanical delay and reflex latency. On another note, we could additionally measure the EMG of the antagonist muscle in future studies. This will allow us to see if antagonist co-contraction is reflected in reduced agonist EMG.

CHAPTER 5

CONCLUSION

The aim of this study was to design a protocol and instrumentation to study the effects of WBV on tendon reflex response. A protocol was successfully designed and implemented. A foot pedal pad for measuring the force of the foot was designed as well as a hammer for tapping of the Achilles tendon. These two devices were designed, synchronized with a Delsys EMG system, calibrated, and used in the collection of data from 7 subjects. The results of the data collection show that there was no significant difference in the effect of WBV on tendon reflex response in the able-bodied subjects. These methods and instrumentation with some modification can be used in future studies to assess the effect of WBV as a treatment for cerebral palsy.

APPENDIX A

FIGURES FOR SUBJECTS 2 – 7

Figure A.1 to A.18 show bar graphs with error bars of EMD, reflex latency, force output and EMG amplitude for subjects 2-7. The error bars represent +/- 1 standard error.

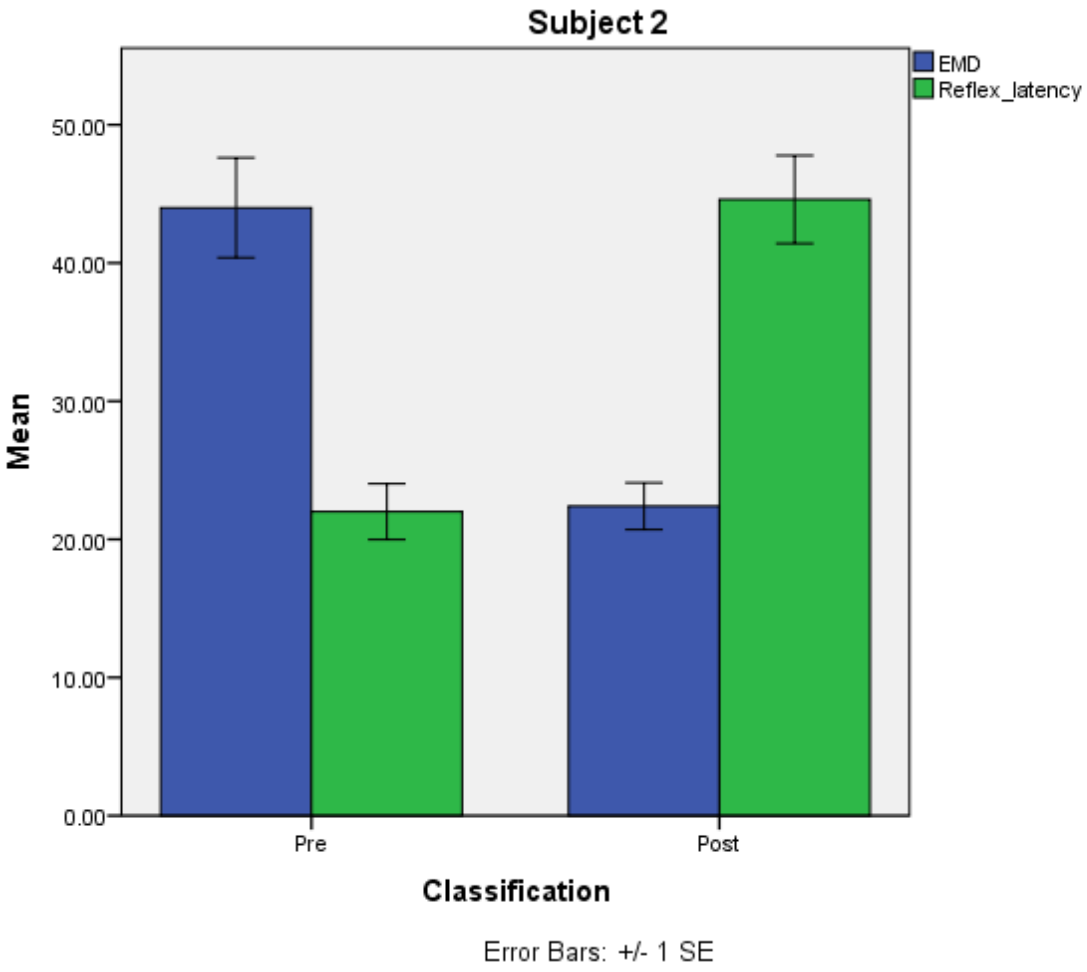

Figure A.1 Mean electromechanical delay and reflex latency for Subject 2.

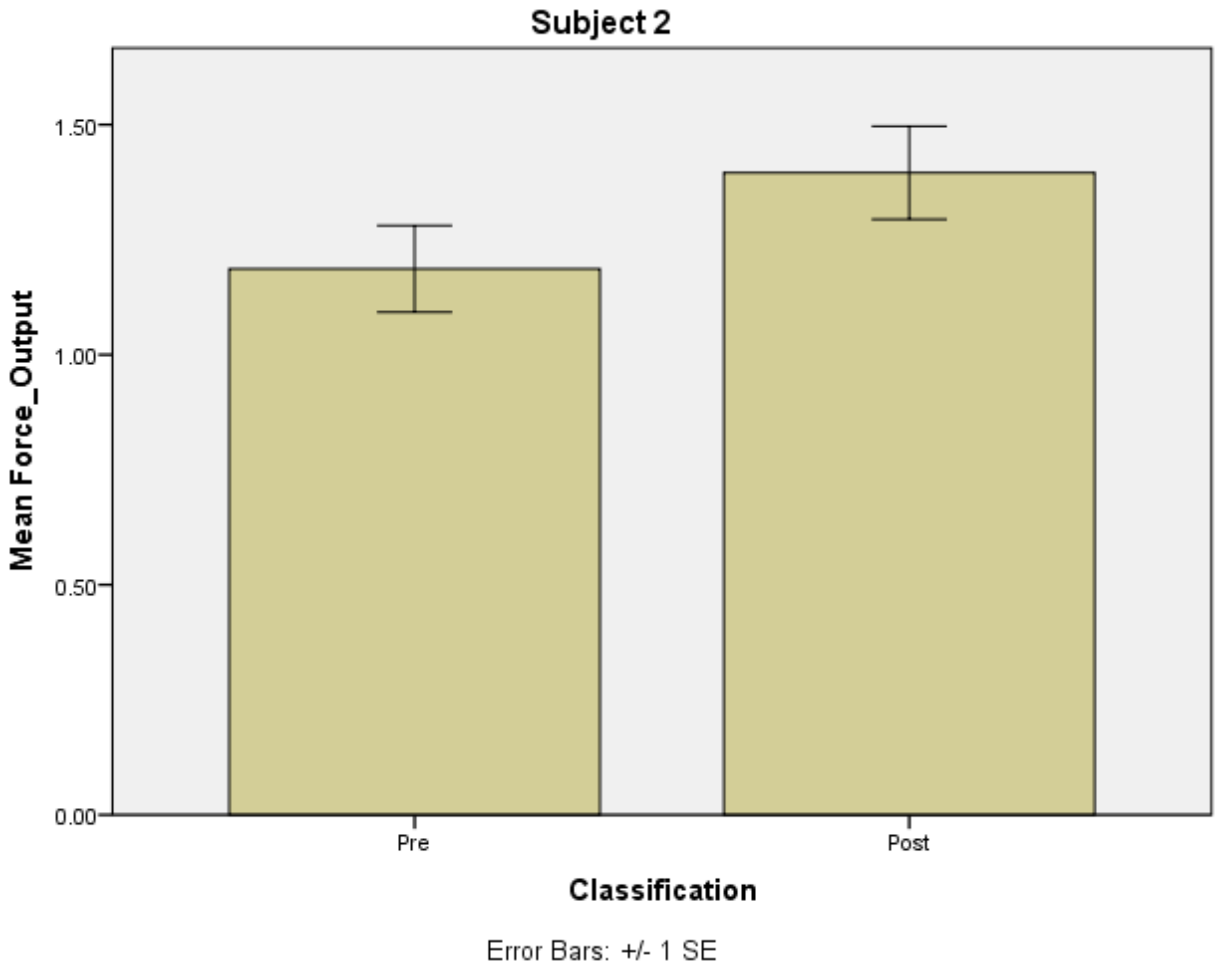

Figure A.2 Mean force output for Subject 2.

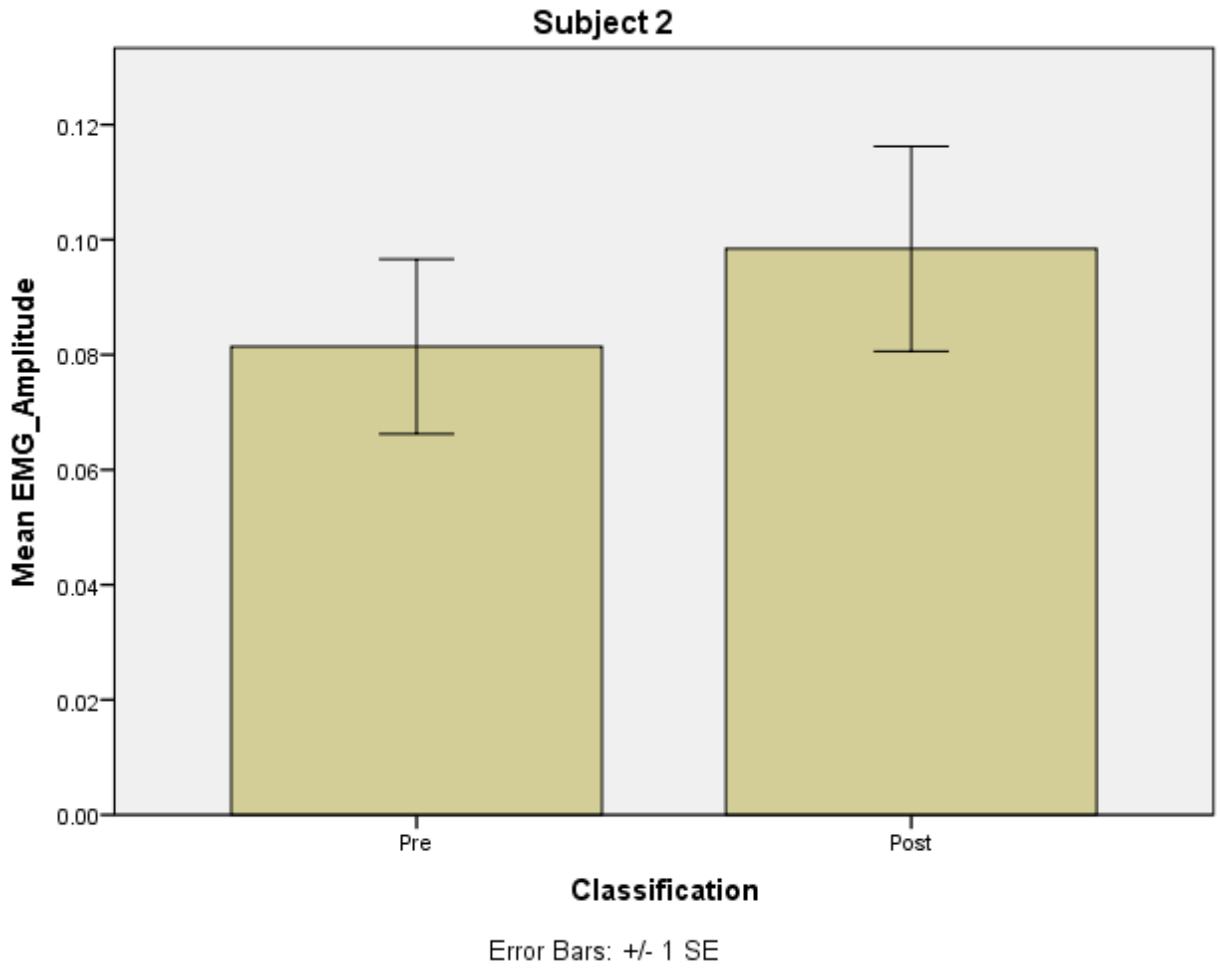

Figure A.3 Mean EMG amplitude for Subject 2.

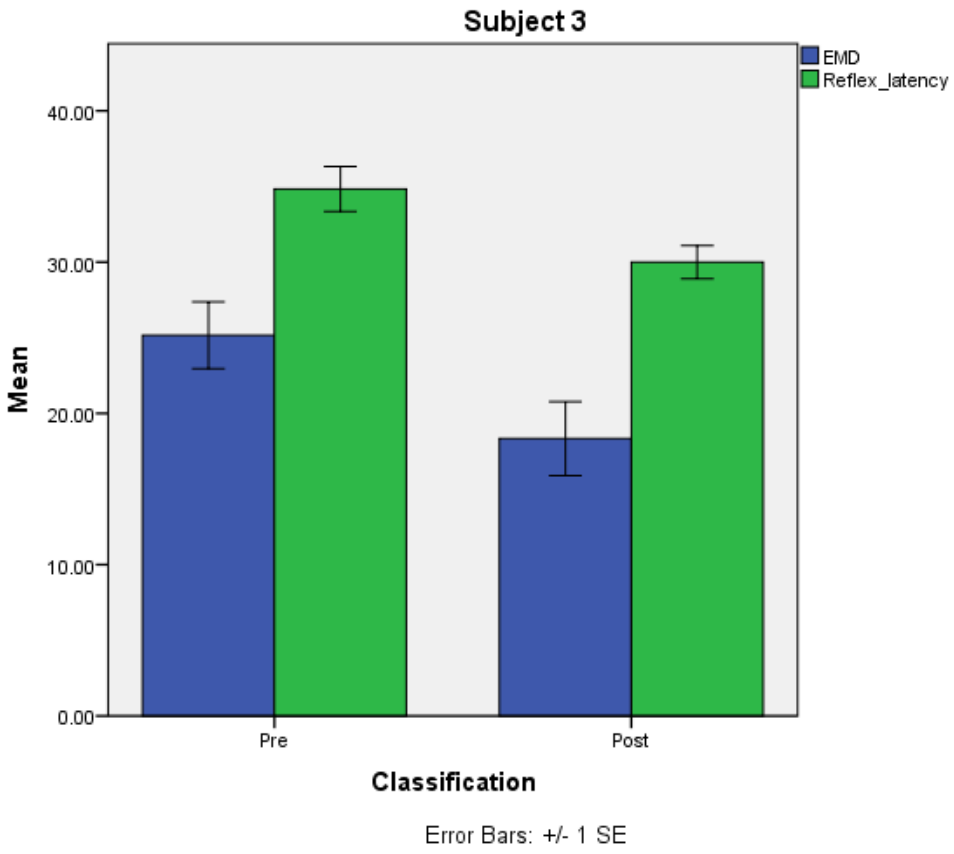

Figure A.4 Mean electromechanical delay and reflex latency for Subject 3.

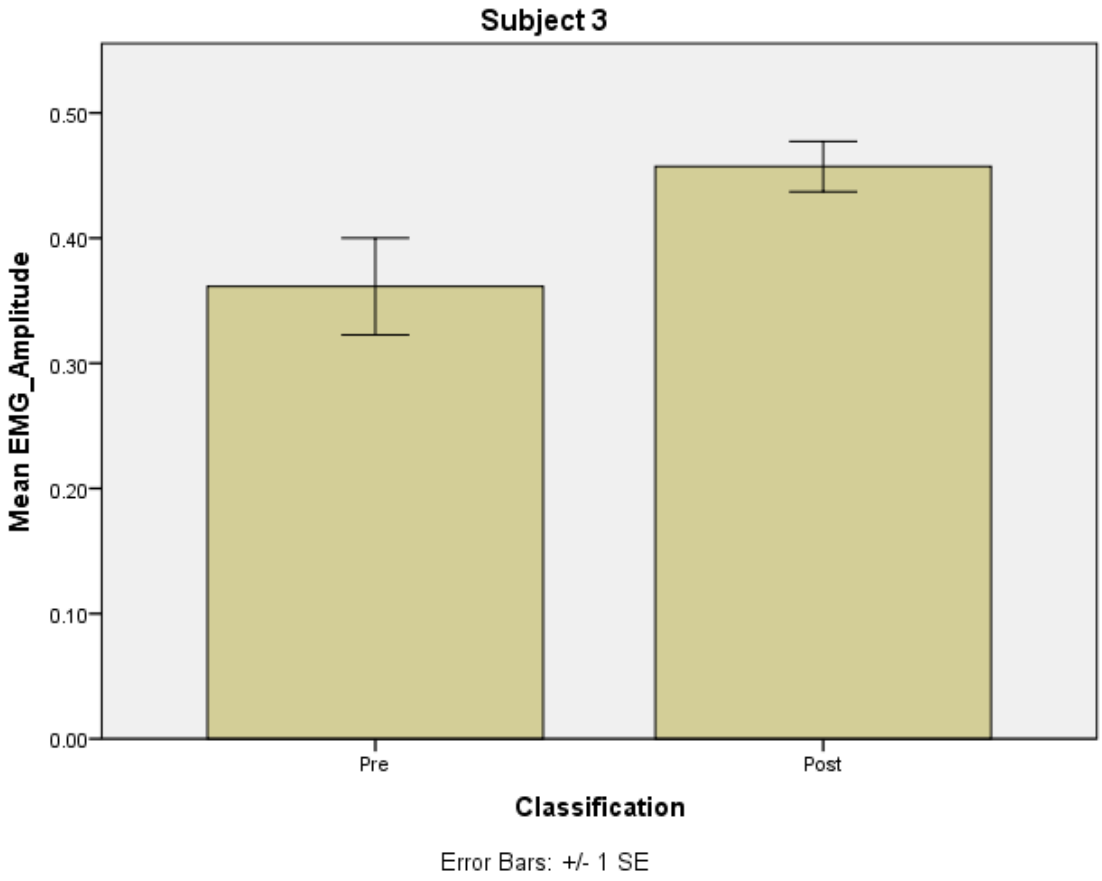

Figure A.5 Mean EMG amplitude for Subject 3.

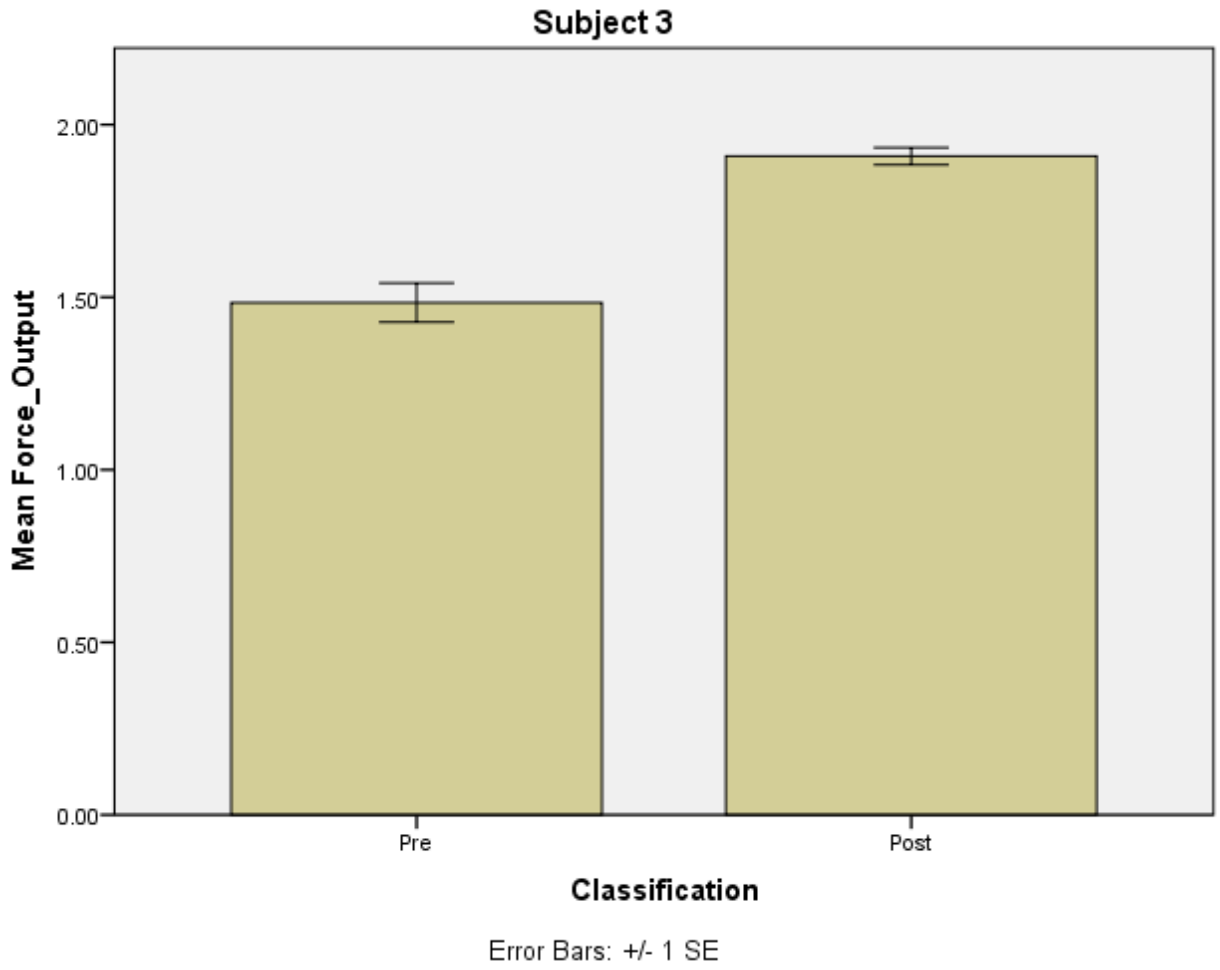

Figure A.6 Mean force output for Subject 3.

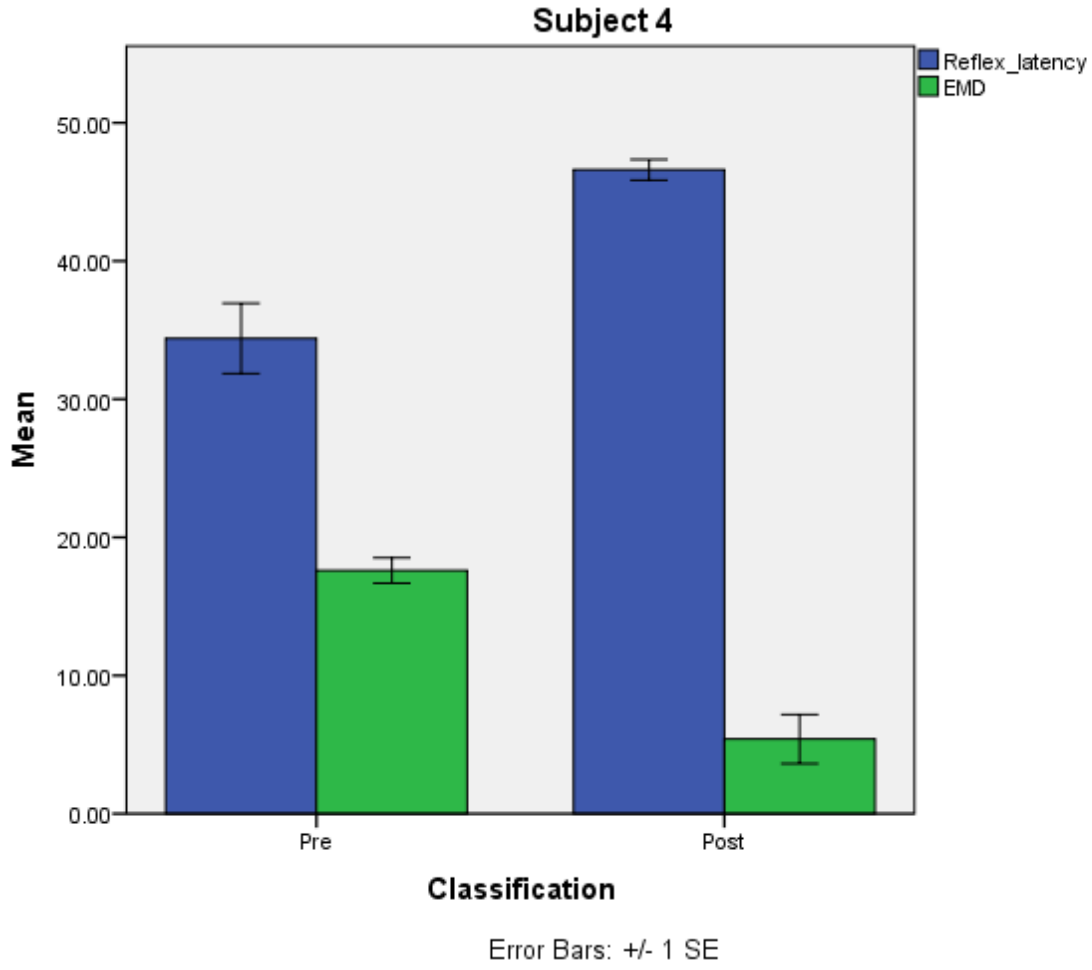

Figure A.7 Mean electromechanical delay and reflex latency for Subject 4.

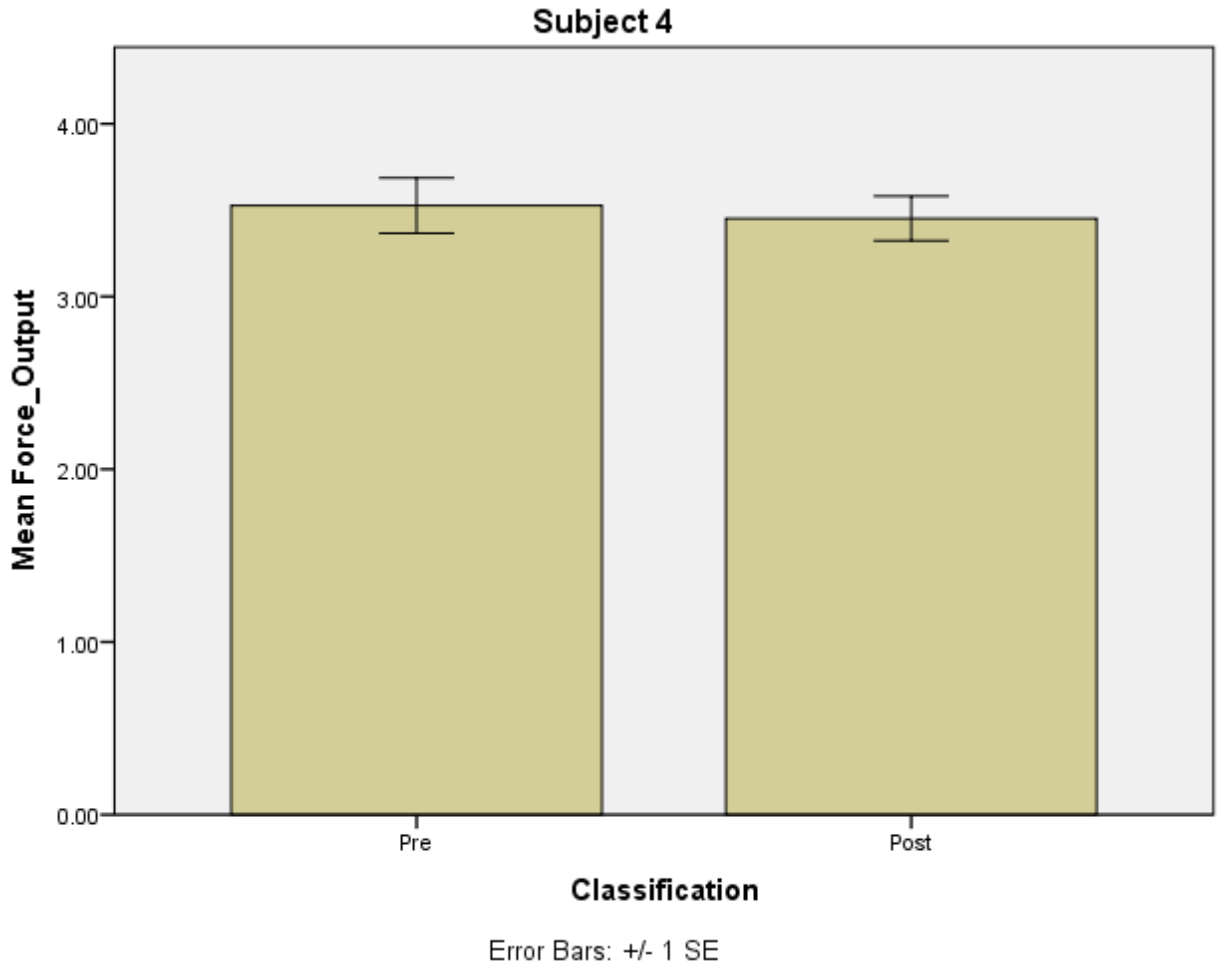

Figure A.8 Mean force output amplitude for Subject 4.

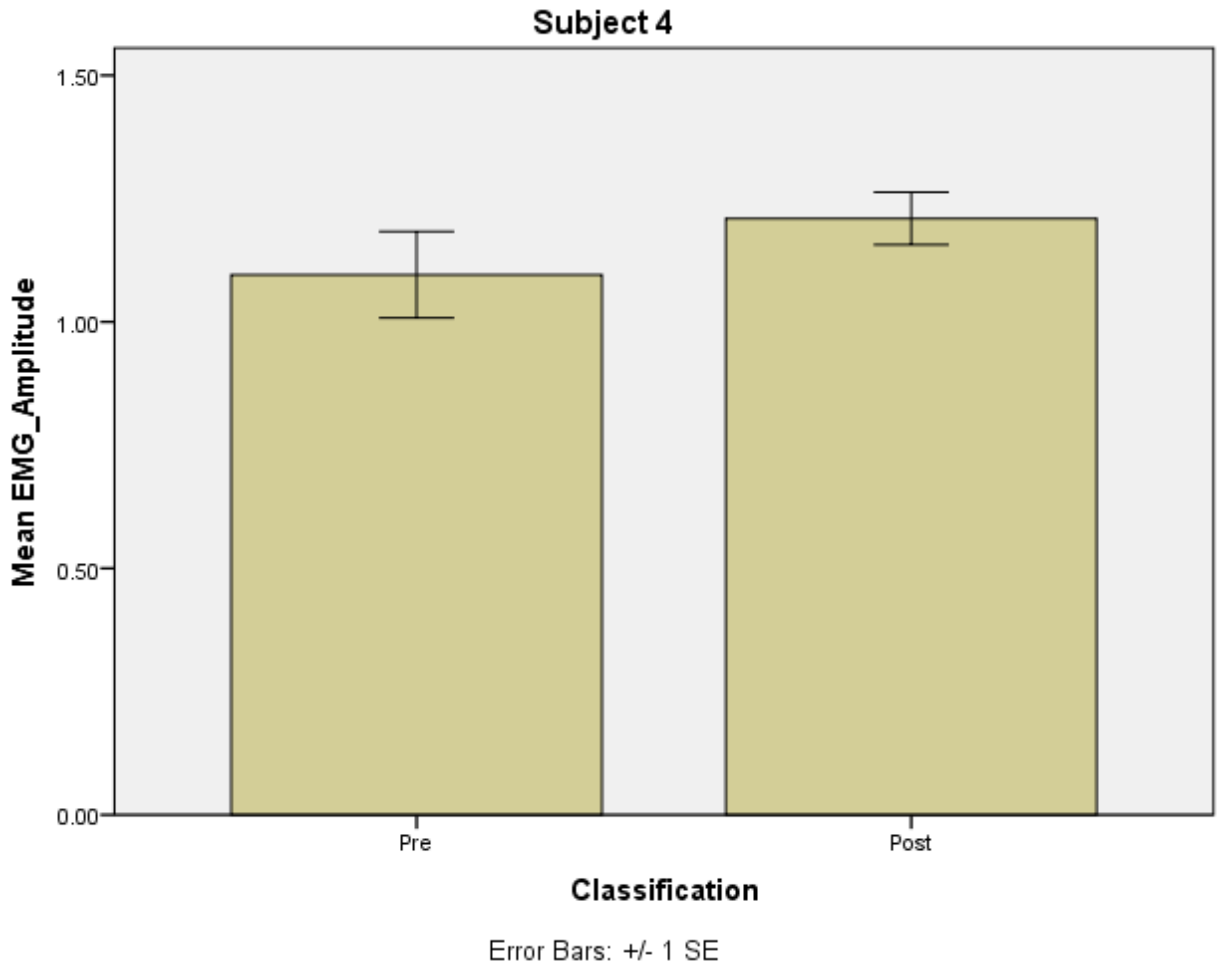

Figure A.9 Mean EMG amplitude for Subject 4.

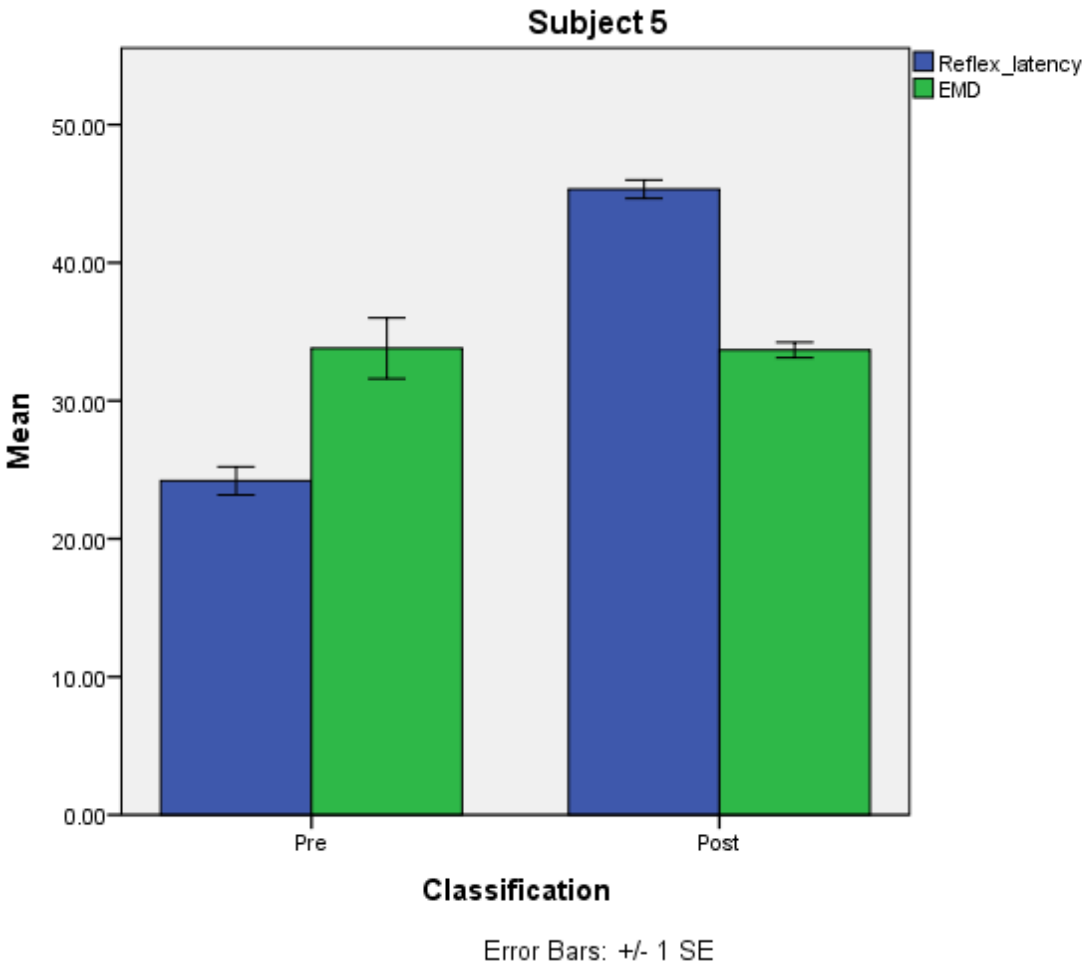

Figure A.10 Mean electromechanical delay and reflex latency for Subject 5.

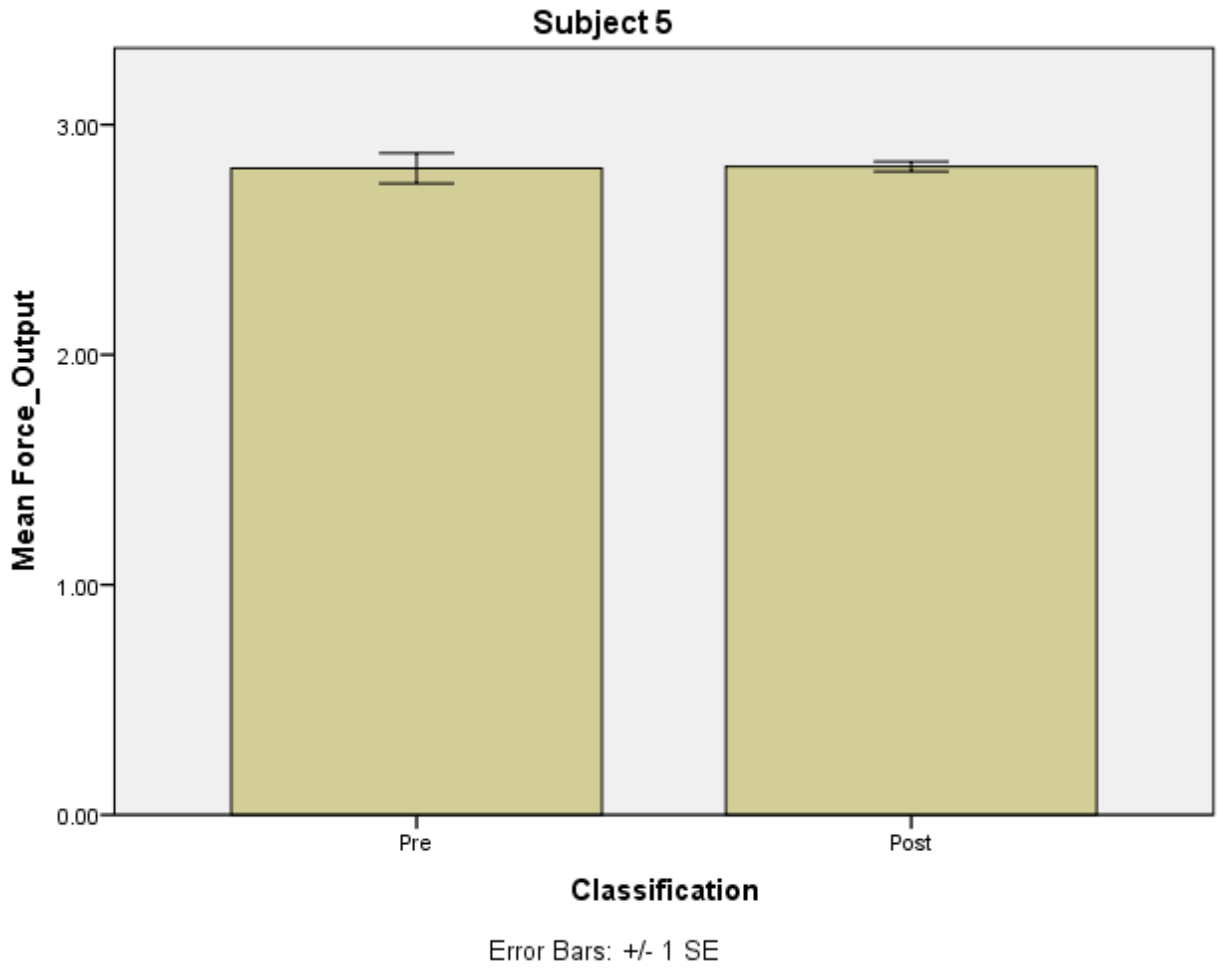

Figure A.11 Mean force output amplitude for Subject 5.

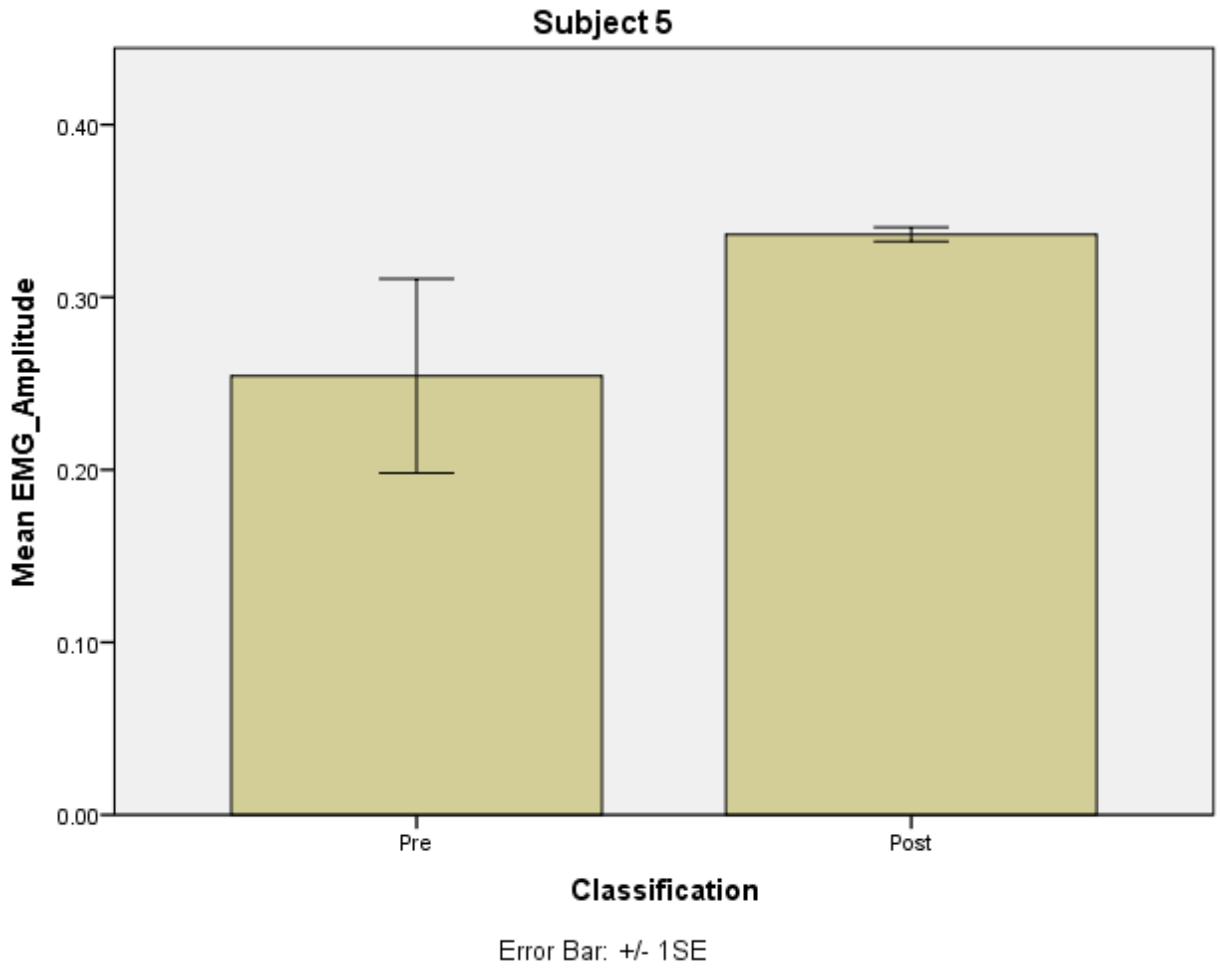

Figure A.12 Mean EMG amplitude for Subject 5.

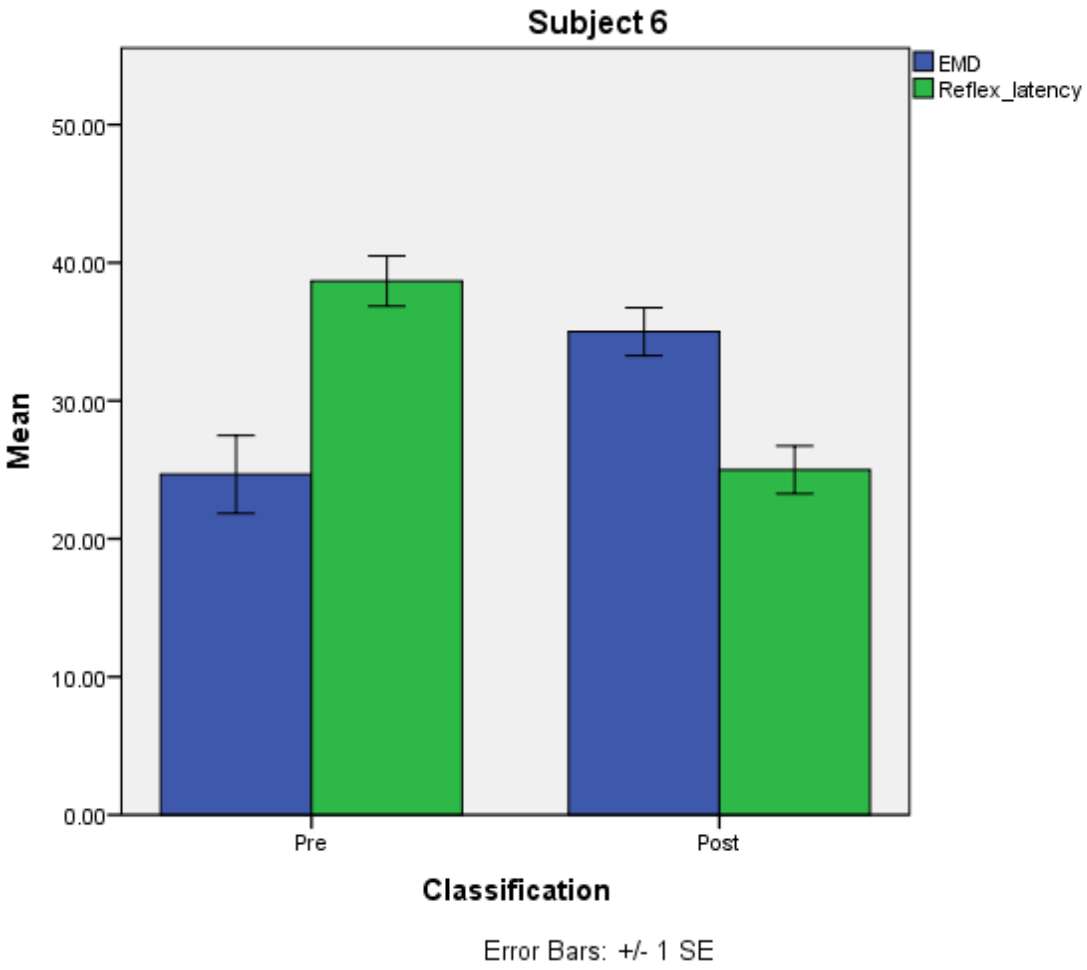

Figure A.13 Mean EMD and reflex latency for Subject 6.

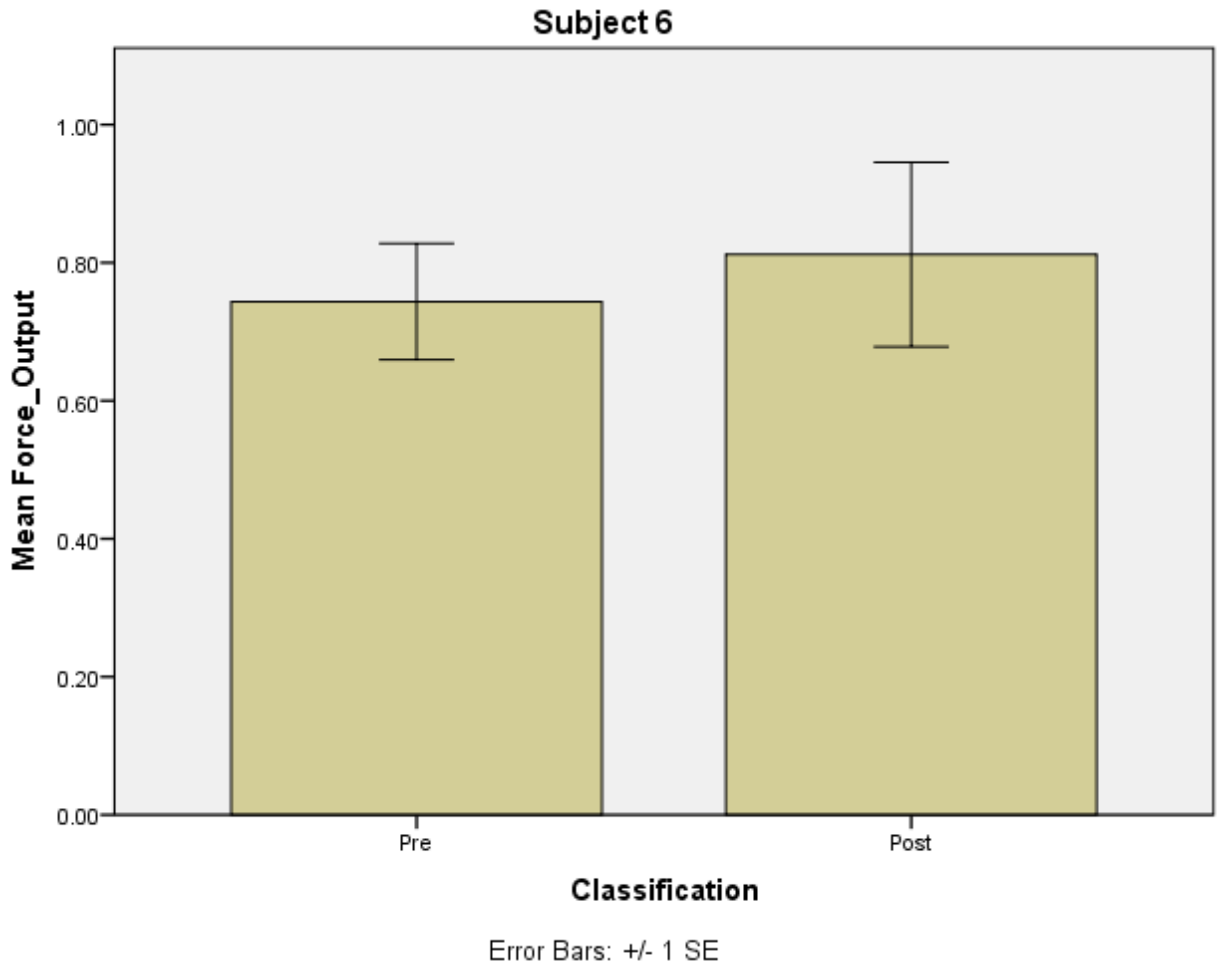

Figure A.14 Mean force output for Subject 6.

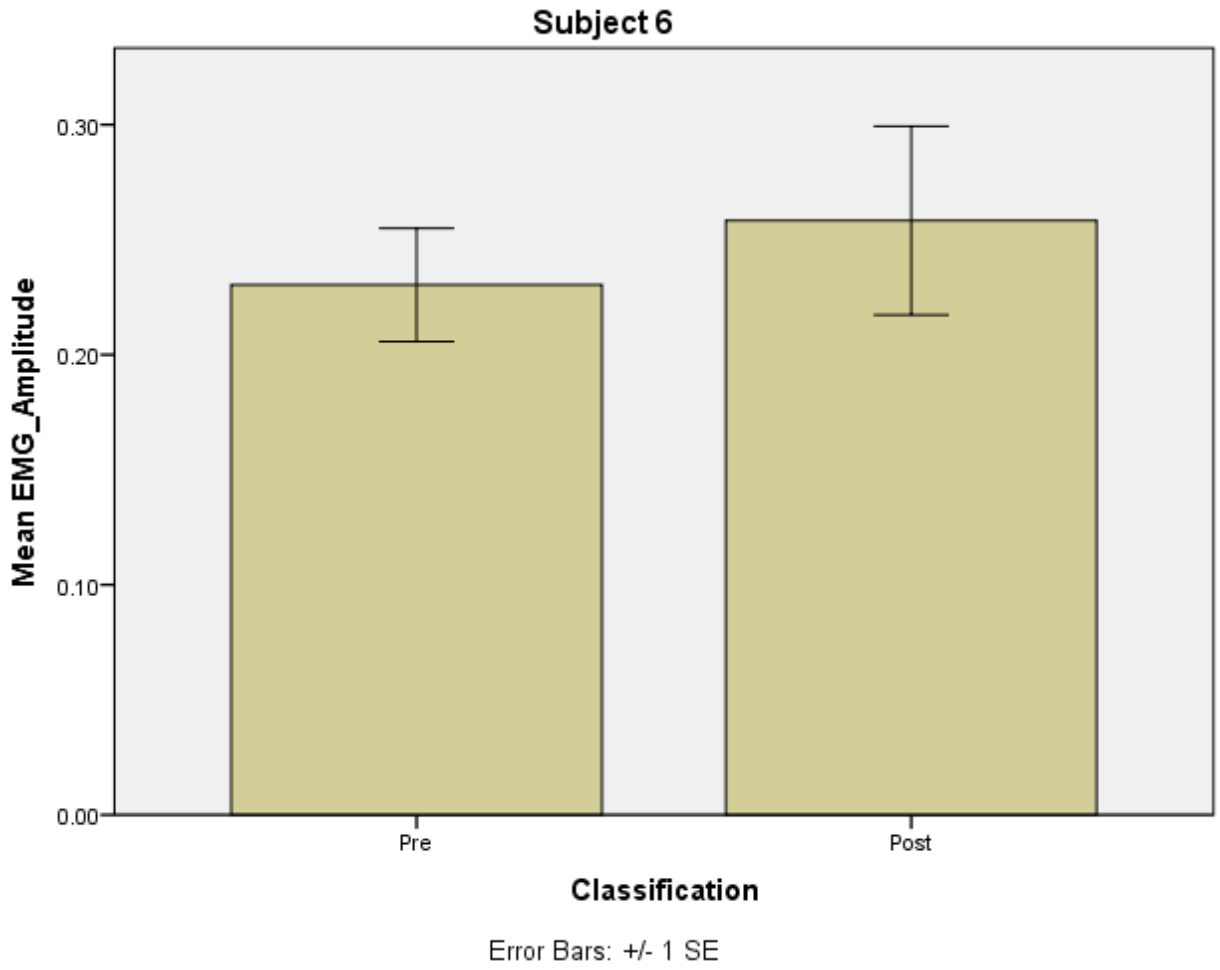

Figure A.15 Mean EMG amplitude for Subject 6.

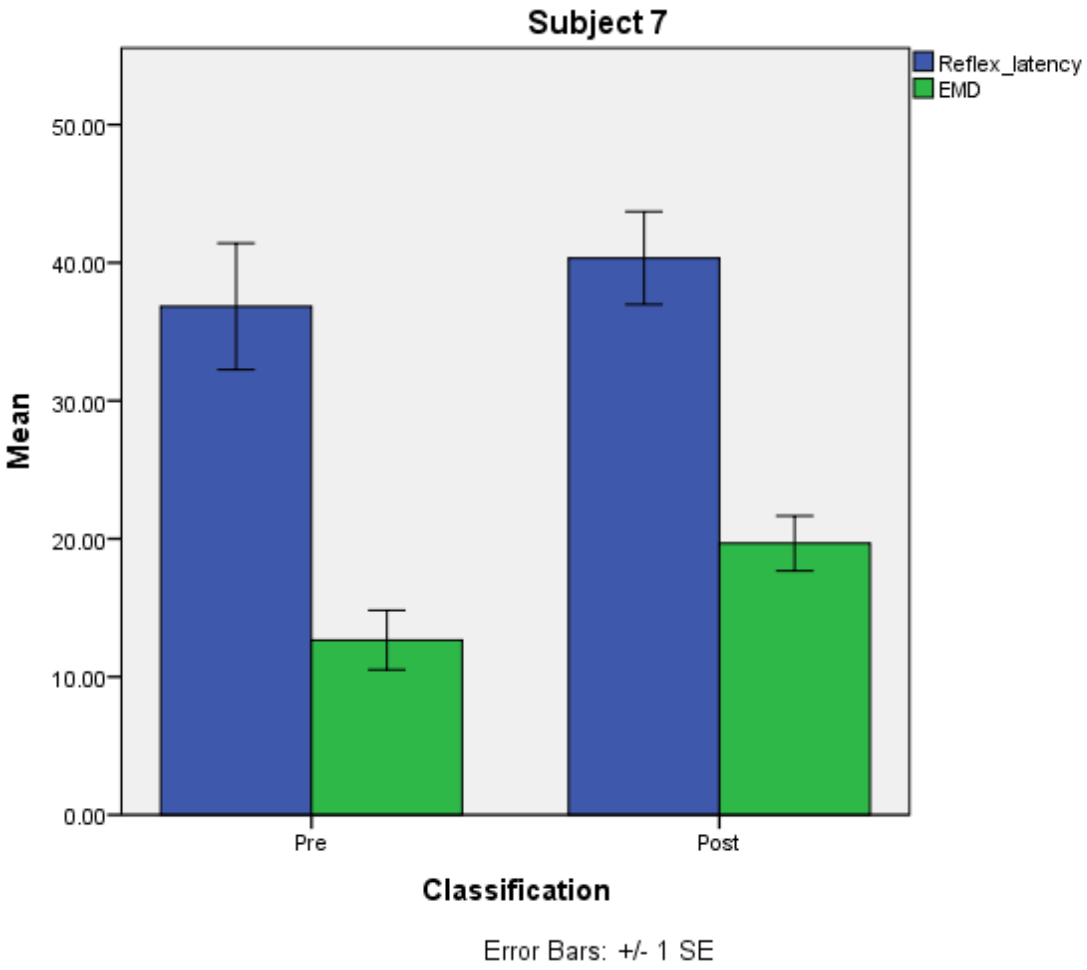

Figure A.16 Mean EMD and reflex latency for Subject 7.

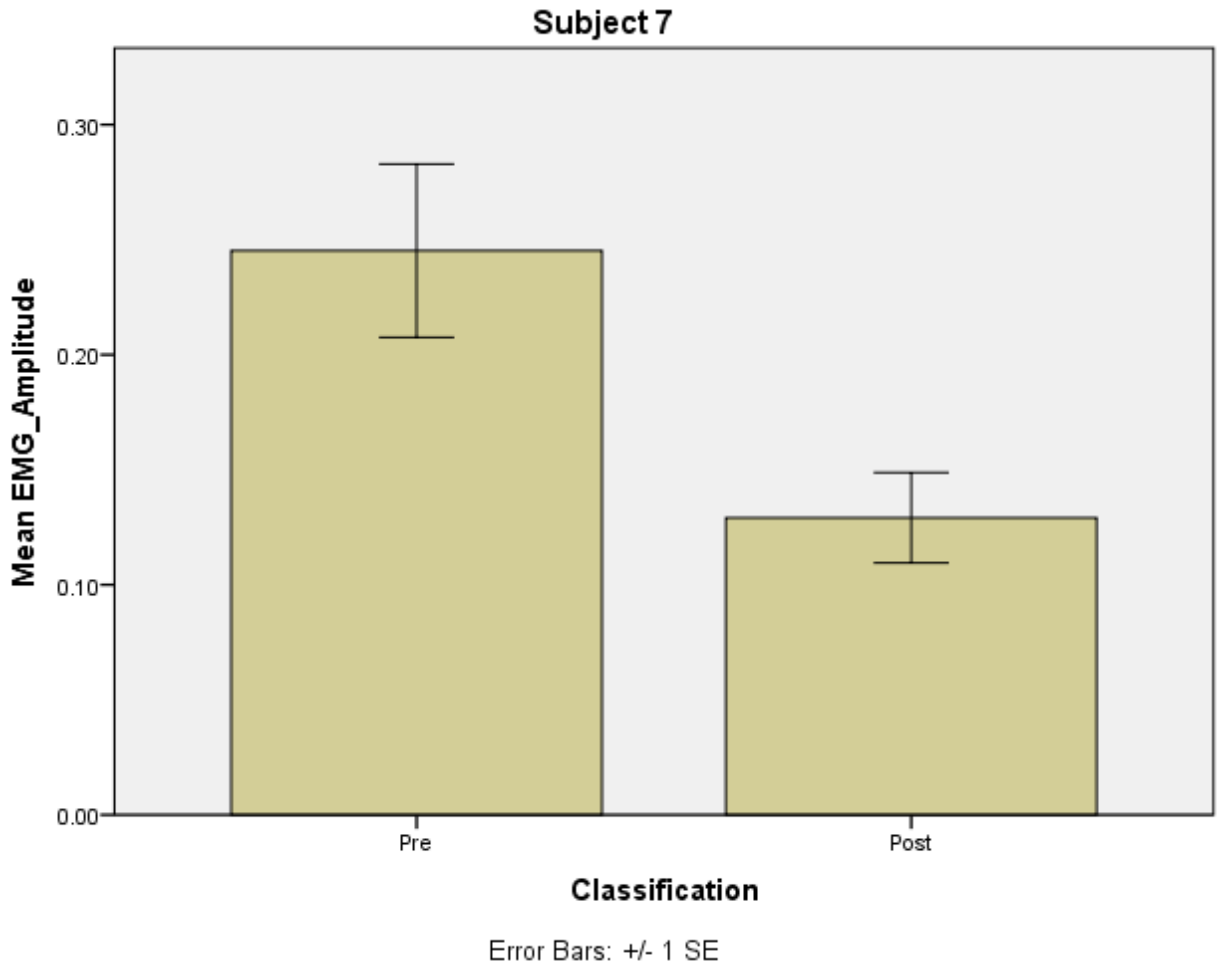

Figure A.17 Mean EMG amplitude for Subject 7.

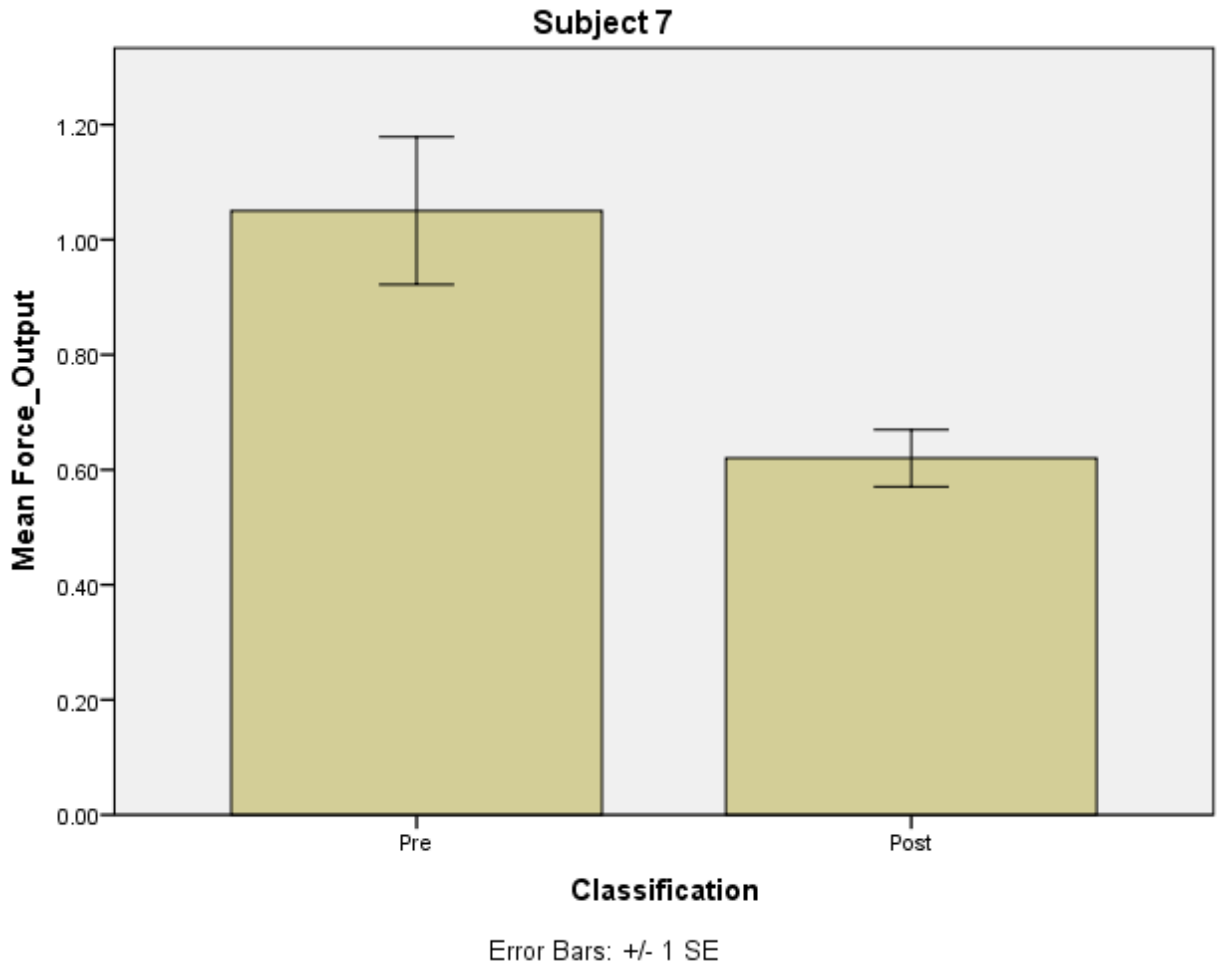

Figure A.18 Mean force output for Subject 7.

APPENDIX B

EQUIPMENTS UTILIZED

In this Appendix, you images of the Delsys Bagnoli EMG system and DKN Technologies – XG10 Vibrating Platform systems. Both pieces of equipment were provided by the neuromuscular lab at New Jersey Institute of Technology.

Figure B.1 figure showing the Delsys-Bagnoli EMG system.

Source: http://www.delsys.com/products/

Figure B.2 Figure showing the XG10 (DKN Technologies) vibrating platform.

Source: http://www.dkn-usa.com/product_xg10.php

APPENDIX C

MATLAB CODE FOR EMG DATA COLLECTION AND SYNCRONIZATION

```
  % %%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%% used high gated for syncing
                                %%%%%%%%%%%%%%%%%%%
                          %% Modification date 3/31/15

%% Data aquisition program for EMG

%%%%%%%%%%%%%%%%%%%%%%%%%%%
% CLOSE PORTS IF OPEN
%%%%%%%%%%%%%%%%%%%%%%%%%%%

if exist ('AI','var')==1
stop(AI)
delete(AI)
clear AI
end

clear all
warning off all;
%% SET DURATION
%%%%%%%%%%%%%%%%%%%%%%%%%%%
Duration = input('Enter Trial Duration in seconds=');
%%%%%
%% SETTING UP PHIDGET
%%%%%%%%%%%%%%%%%%%%%%%%%%%
loadphidget21
bridgePtr = libpointer ('int32Ptr',0);
dataptr = libpointer('doublePtr', 0); %set up double pointer for double
answers
enptr= libpointer('int32Ptr', 0);
calllib ('phidget21', 'CPhidgetBridge_create',bridgePtr);
calllib('phidget21', 'CPhidget_open', bridgePtr, -1); % Open bridge
if calllib('phidget21', 'CPhidget_waitForAttachment', bridgePtr, 2500)
== 0
disp('Opened Bridge')
end
serial = libpointer('int32Ptr',0);
calllib('phidget21', 'CPhidget_getSerialNumber', bridgePtr, serial);
%get serial number
calllib('phidget21', 'CPhidgetBridge_getDataRate', bridgePtr, enptr);
%get the current data rate
dataRate= enptr.Value % output the current data rate
calllib('phidget21', 'CPhidgetBridge_setEnabled', bridgePtr, 0, 2); %
enable bridge 0

%% SETUP EMG
%%%%%%%%%%%%%%%%%%%%%%%%%%%
```

```
AI = analoginput('nidaq','Dev2');
set(AI,'SampleRate',1000); %EMG Sampling Rate
rate=get(AI,'SampleRate');
totalsample=rate*Duration;
set(AI,'TriggerType','Manual');
set(AI,'SamplesPerTrigger',inf);
set(AI,'InputType','SingleEnded');
chan=addchannel(AI,6);
dio = digitalio('nidaq','Dev2');
addline(dio,0:7,'out');
bvdata = logical([0 0 0 0 0 0 0 0]); % this ensures that the gate is
closed
putvalue(dio,bvdata)
start(AI);


%% WAIT TO START DATA COLLECTION
%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
bvdata = logical([1 1 1 1 1 1 1 1]); % This opens the gate
display('Initialization complete.  Press any key to start data
collection')
pause
display('Data Collection has begun........')
putvalue(dio,bvdata)
trigger(AI); % Trigger starts EMG data aquisition
%% COLLECTING BOTH EMG AND PHIGET DATA

% for j=1:100*Duration
%     tic
%  calllib('phidget21', 'CPhidgetBridge_getBridgeValue', bridgePtr, 0,
dataptr);
%  data(j) = -5.0008*(dataptr.Value) -0.0537;
%
% while toc<0.01;
% end
% end

for i=1:100*Duration
tic
% getting foot pedal force
calllib('phidget21', 'CPhidgetBridge_getBridgeValue', bridgePtr, 0,
dataptr);
data(1,i)=-2.0065*(dataptr.Value)-0.1191; % foot pedal
% getting Hammer impact force
calllib('phidget21', 'CPhidgetBridge_getBridgeValue', bridgePtr, 2,
dataptr);
data(2,i)= -5.0008*(dataptr.Value) -0.0537; % Hammer impact force

while toc<0.01;
end
end



stop(AI)% stops the EMG
```

```
bvdata = logical([0 0 0 0 0 0 0 0]); % this stops Trio
putvalue(dio,bvdata)

%Retieve Data
Phiddata=data;
EMGdata = getdata(AI,AI.SamplesAvailable); %Read EMG

%% CLOSE PORTS
%%%%%%%%%%%%%%%%%%%%%%%%%%

% EMG
delete(AI)
clear AI
% PHIDGETS
calllib('phidget21', 'CPhidget_close', bridgePtr); %read value of
bridge 0
calllib('phidget21', 'CPhidget_delete', bridgePtr); %read value of
bridge 0
display('Data Collection has ended')
%% PLOTS
% figure(1)
% plot(Phiddata,'r')
% title('phid')
figure(1)
plot(Phiddata)
title ('Hammer inpact force')
% figure(2)
% plot(-1*(Phiddata(2,:))) %getiing an inverse of the hammer force
% title ('Hammer inpact force')

figure(3)
plot(EMGdata,'k')
title('emg')
```

APPENDIX D

MATLAB CODE FOR DATA ANALYSIS

```
clear all
close all
clc

uiload
warning off


%% Part 1. Hammer Amplitude

hammer_data=-Phiddata(2,:);

[hammAmp,hammLoc]=findpeaks(hammer_data,'MINPEAKHEIGHT',0.25,'MINPEAK
DISTANCE',50);

figure(1)
plot(hammer_data)
hold on
plot(hammLoc,hammAmp,'.r')

meanHam=mean(hammAmp)


%% Part 2. EMG Amplitude

EMG=abs(EMGdata-mean(EMGdata));

[EMGamp,EMGLoc]=findpeaks(EMG,'MINPEAKHEIGHT',1.5*mean(EMGdata),'MIN
PEAKDISTANCE',50);
EMGamp=EMGamp';

figure(2)
plot(EMG)
hold on
plot(EMGLoc,EMGamp,'.r')

meanEMGamp=mean(EMGamp)


%% Part 3. Hammer Amplitude vs. EMG Amplitude
```

```
figure(3)
EMGamp
hammAmp
% plot(EMGamp,hammAmp,'.')
% axis([0 max(EMGamp) 0 max(hammAmp)])
% xlabel('EMG')
% ylabel('Hammer Force')



%% Part 4. Generated Force

foot_data=Phiddata(1,:);

[P2,L2]=findpeaks(foot_data,'MINPEAKHEIGHT',1.04*mean(foot_data),'MINPEAKDI
STANCE',50);

L1=L2-20;

for i=1:length(L1)
   P1(i)=foot_data(L1(i));
end

figure(4)
plot(foot_data,'r')
hold on
plot(L2,P2,'b.','MarkerSize',25)
plot(L1,P1,'k.','MarkerSize',25)

for i=1:length(P2)
   force(i)=P2(i)-P1(i);
end

foot_force=mean(force)

force

%%
%%
EMG_sig=EMGdata; %isolating EMG signal
Fs=1000;     %sample frequency
EMG_Len=length(EMG_sig);
T_emg=(1/Fs):(1/Fs):(EMG_Len/Fs);%getting time for EMG
```

```
Hphid=resample(Phiddata(1,:),100,10); % resampling Force data to be thesame as EMG
data
Pphid=resample(Phiddata(2,:),100,10); % resampling Force data to be thesame as EMG
data
phid_len=length(Pphid);
T_phid=(1/Fs):(1/Fs):(phid_len/Fs); %getting time for force

hammer=-1*Pphid;
hammer=hammer;
Press_pedal=Hphid;

figure(5)
subplot (3,1,1)
plot(T_phid,hammer) %getiing an inverse of the hammer force
title ('Hammer inpact force')
xlabel('time(S)')
ylabel('Force(Kg)')
grid on

subplot (3,1,2)
plot(T_emg,abs(EMG_sig-mean(EMG_sig)),'k');
title('EMG')
xlabel('time(S)')
ylabel('EMG amplitude(mV)')
axis([0 30 -.5 .5])
grid on

subplot (3,1,3)
plot(T_phid,Press_pedal)
title ('pedal press force')
xlabel('time(S)')
ylabel('Force(Kg)')
grid on


 %% Part 5. Timing of Hammer vs. EMG vs. Foot Force

view_window1=13;
view_window2=35;

for i=1:length(hammLoc)
figure(i+5)
subplot(3,1,1)
plot(hammer_data(hammLoc(i)-view_window1:hammLoc(i)+view_window2))
ylabel('hammer')
xlabel('Time(S)')
```

```
grid on
subplot(3,1,2)
plot(EMG(10*(hammLoc(i)-view_window1):10*(hammLoc(i)+view_window2)))
ylabel('EMG (mV)')
xlabel('Time(S)')
grid on
subplot(3,1,3)
plot(foot_data(hammLoc(i)-view_window1:hammLoc(i)+view_window2))
grid on
ylabel('foot force')
xlabel('Time(S)')
end
```

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