Vital Signals Lab

Experiment 4 (EMG)

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This file contains the report and results of the simulations conducted.

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Abstract

A skeletal muscle fiber is innervated by a branch of a motor axon. Under normal circumstances, a neuronal action potential activates all of the muscles innervated by the motor neuron. This activation process involves an action potential and a contraction of the muscle fibers. During a contraction, therefore, there is synchronous activity in a number of fibers in the same muscle. The electrical signal recorded from a contracting muscle is called an electromyogram or EMG. Like the electrocardiogram (ECG), this activity can be detected by electrodes placed on the skin. A voluntary muscle contraction is produced by one or more action potentials in many fibers. The EMG activity is not a regular series of waves like the ECG, but a chaotic burst of overlapping spike-like signals.

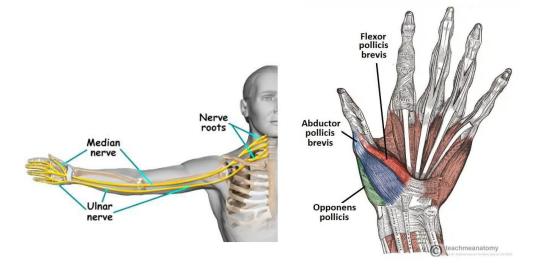


Figure 1 (muscle fibers and neuron)

Note that from the beginning of the first section to the end of the third section, the signal channels are in the following order:

Biceps signal

Triceps signal

Integrated biceps signal

Integrated triceps signal

Data notebook & Results

Voluntary change in contractile force

In this section, we aim to examine EMG signals from the biceps and triceps. To verify the connections, we asked the subject to open and close their hand once, with another person providing resistance to this movement.

The result is:

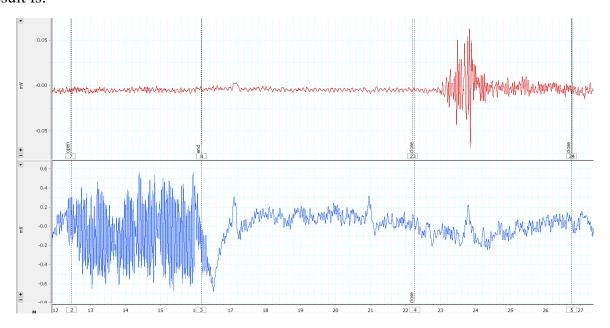


Figure 2 (subject1 EMG signal when open and close hand from elbow with resistance)

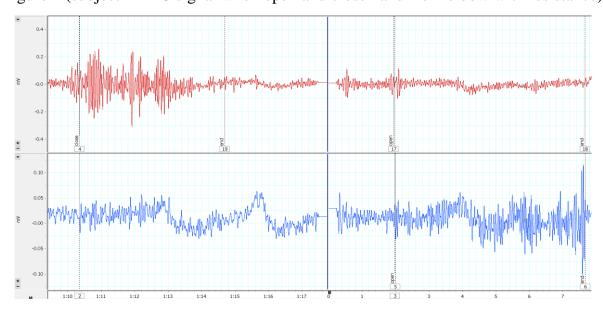


Figure 3 (subject2 EMG signal when open and close hand from elbow with resistance)

As we know, when the arm is extended from the elbow, the biceps are engaged, and when it is flexed, the triceps are engaged. That is clearly confirmed by the signals (see the comments in the image).

Now, the subject should bring their arm to a 90-degree angle and open their palm. Then, another person places weights on their hand.

The results are as follows:

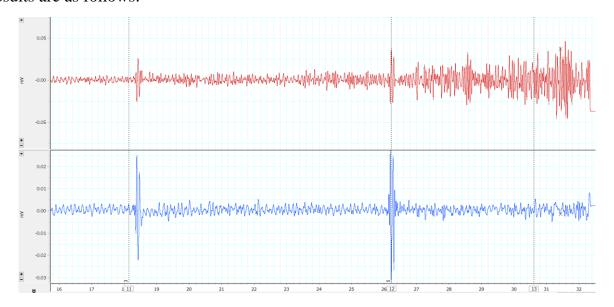


Figure 4 (subject1 EMG signal when placing weights)

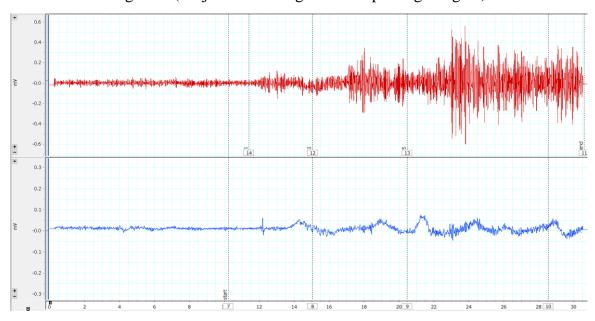


Figure 5 (subject2 EMG signal when placing weights)

As mentioned in the first section, when the hand resists opening, the biceps are engaged, resulting in high peaks in the EMG signal. Additionally, subject two has more triceps engagement during this action compared to subject one. However, subject one experiences a spike in their signal when weights are placed on their hand.

Weight	Biceps EMG amplitude (mv)	Triceps EMG amplitude (mv)	
1Kg	0.014	0.018	
3Kg	0.025	0.025	
5Kg	0.046	0.033	

Table 1 (subject1 EMG signal when placing weights)

Weight	Biceps EMG amplitude (mv)	Triceps EMG amplitude (mv)	
1Kg	0.12	0.07	
3Kg	0.34	0.055	
5Kg	0.56	0.063	

Table 2 (subject2 EMG signal when placing weights)

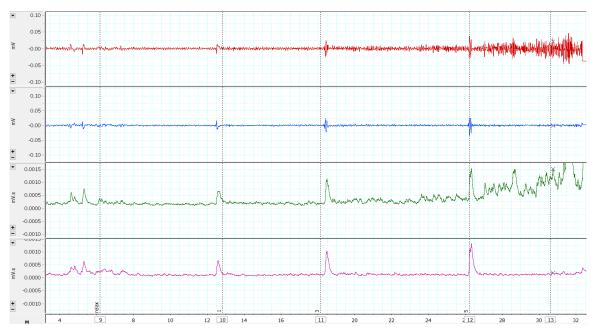


Figure 6 (subject1 placing weights)

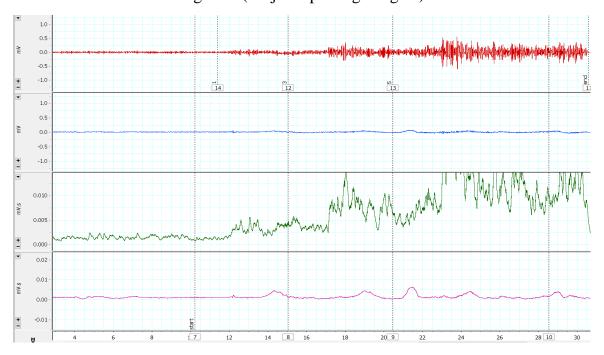


Figure 7 (subject2 placing weights)

The height of the integrated trace reflects the overall activity of the raw EMG signal, and gives a simpler view of the muscle's electrical activity. You can see the integrated signal in Result part.

Alternating activity and co-activation

In this part of the laboratory, the activity of antagonist muscles and the phenomenon of co-activation will be examined.

Antagonist Muscles: Antagonist muscles are those that perform the opposite action to the agonist muscles (the main muscles responsible for movement) during a movement. For example, in the flexion of the forearm (like bending the elbow), the biceps muscle acts as the agonist, while the triceps muscle acts as the antagonist.

Co-activation Phenomenon: Co-activation occurs when both the agonist and antagonist muscles are activated simultaneously. This co-activation can help increase joint stability, allow for more precise movement control, and prevent unwanted joint displacements.

In this section, the subject creates resistance against their hand movement using their other hand, with the following results:

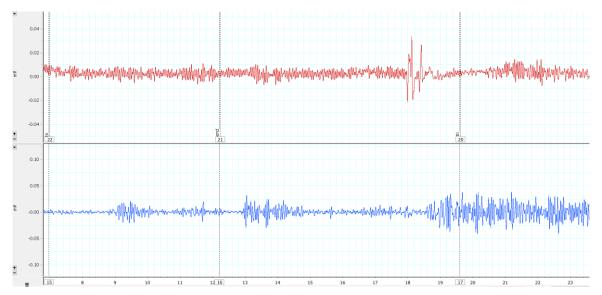


Figure 8 (subject1 Antagonist and co-activation)

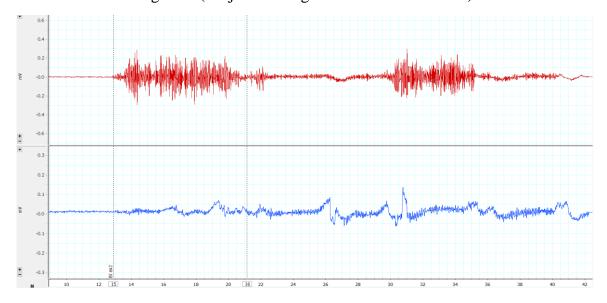


Figure 9 (subject2 Antagonist and co-activation)

As we can see, when the biceps muscles are engaged, the triceps muscles show little to no activity, and vice versa. This illustrates the concept of antagonist muscles and the phenomenon of coactivation.

Active muscle	Active muscle EMG amplitude in "active" muscle	
Biceps	0.04	0.03
Triceps	0.01	0.06

Table 3 (subject1)

	Active muscle	EMG amplitude in "active" muscle	EMG amplitude in opposite muscle	
Biceps		0.2	0.3	
	Triceps	0.06	0.13	

Table 4 (subject2)

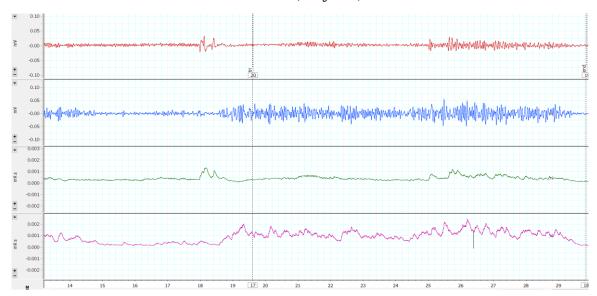


Figure 10 (subject1 placing weights with integrated signals)

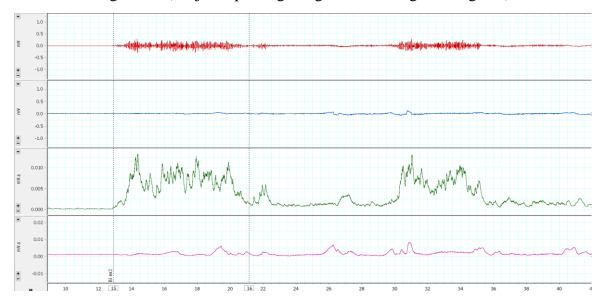


Figure 11 (subject2 placing weights with integrated signals)

Evoked EMG

In this section, we examine nerve conduction time response. We apply an electrical shock to one area of the lower forearm and wait for a response in the form of hand movement to capture the EMG signal.

The time difference between the shock application and the response gives us the time delay for the shock to reach the muscle. We then repeat this procedure on areas higher up on the forearm.

The results are as follows:

Stimulus Location	Latency (ms)	
Wrist	25	
Elbow	40	

Table 5 (subject1 nerve latency)

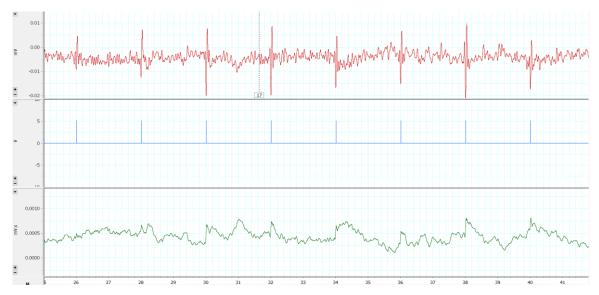


Figure 12 (subject1 Stimulus latency from wrist)

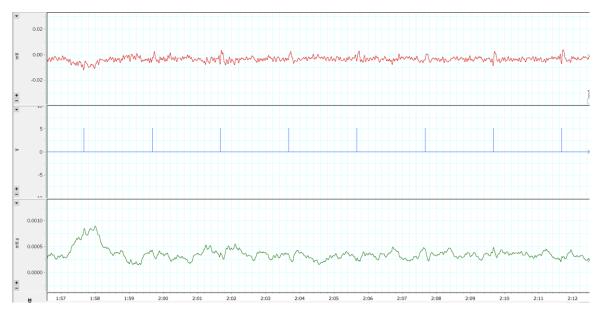


Figure 13 (subject1 Stimulus latency from elbow)

Stimulus Location	Latency (ms)	
Wrist	44	
Elbow	53	

Table 6 (subject2 nerve latency)



Figure 14 (subject2 Stimulus latency from wrist)

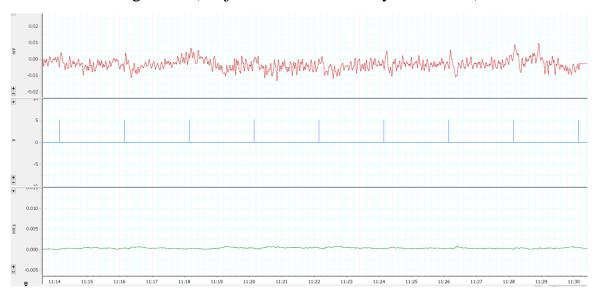


Figure 15 (subject2 Stimulus latency from elbow)

Nerve conduction velocity

Now we can calculate the velocity of the nerve conduction from

$$V = \frac{dx}{dt}$$

So, we have:

Distance between stimulation	Time difference between	Nerve conduction velocity	Nerve conduction velocity
sites (mm)	latencies (milliseconds)	(mm/milliseconds)	(m/sec)
120	40 - 25 = 15	120	8
		${15} = 8$	

Table 7 (subject1 conduction velocity)

Distance between stimulation	Time difference between	Nerve conduction velocity	Nerve conduction velocity
sites (mm)	latencies (milliseconds)	(mm/milliseconds)	(m/sec)
120	53 – 44 = 9	$\frac{120}{9} = 13.33$	13.33

Table 8 (subject1 conduction velocity)

Conclusion

1) Unlike the discrete waveform from an electrocardiogram, the electromyogram waveform is irregular. Why do you suppose this is?

The irregularity of the electromyogram (EMG) waveform, as compared to the more distinct waveform of an electrocardiogram (ECG), arises from several key differences:

The heart's contractions are regulated by its conduction system, producing consistent electrical patterns. Skeletal muscles, on the other hand, lack this level of synchronization, as different motor units are activated at varying times. EMG captures activity from skeletal muscles, which involves voluntary and asynchronous contractions of numerous muscle fibers, creating a complex and variable waveform. In contrast, ECG measures the heart's electrical activity, which is highly organized and rhythmic, resulting in a more regular waveform.

2) How did the EMG trace change when you added weights to your arm? What do your results indicate?

After adding weight, the EMG trace of the biceps increased in voltage, after adding more weights the EMG trace of the triceps also increased. but as shown in figure the increase of the amplitude in the biceps was greater than the triceps.

3) Describe co-activation. Why do you think this phenomenon occurs?

As said before, Co-activation occurs when both the agonist and antagonist muscles are activated simultaneously. This co-activation can help increase joint stability, allow for more precise movement control, and prevent unwanted joint displacements.

4) Describe the meaning of the latent period in your trace from evoked EMG activity. Did latency change with increasing stimulus amplitude?

In an evoked EMG trace, the latent period is the time interval between the application of a stimulus and the beginning of the muscle response. This period includes the time needed for the electrical signal to travel along the nerve, reach the neuromuscular junction, release neurotransmitters, and ultimately depolarize muscle fibers to produce a contraction.

Increasing the amplitude of the stimulus did not significantly affect the latency.

5) Based on your results and calculations for nerve conduction velocity, how long would it take for a nerve impulse to travel from the spinal cord to the big toe? Assume that the distance traveled is one meter.

It will take about 0.093 second for a nerve impulse to travel from the spinal cord to the big toe

$$V = \frac{dx}{dt}$$

$$dx = 1m$$

$$V = \frac{8 + 13.33}{2} = 10.66$$

$$t = \frac{1}{10.66} = 0.093 \text{ sec}$$