

Human nerve conduction velocity



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Medsci 206 Laboratory Report 6: Human Nerve Conduction Velocity

Aim:

- To record the EMG and observe the changes in the recording with different levels of voluntary muscle contraction
- velocities of both the ulnar and the median nerves that are located in the human forearm.
- To analyze and contrast examples of normal and abnormal nerve conduction measurements

Introduction:

The currents that courses through our motor neurons, during the voluntary activation of our muscles, generates an electrical signal that can be detected outside the activated muscle itself (Kandel, Schwartz & Jessell, 2000).

Through observation of electromyographic activity, we are able to determine the physiological activity that underlies muscle contraction. To do this, we use an electromyogram; which uses simple electrodes that records the complex pattern of electrical potentials when placed over the surface of the overlying skin (Kandel, Schwartz & Jessell, 2000).

The currents that are noticed in our nerves upon stimulation are known as action potentials (Totara & Derrickson, 2006). The determining factor of whether an action potential occurs is the opening of voltage-gated sodium channels, which itself is, in turn, dependant on the initial depolarization of cell; usually a specific ‘ threshold’ stimulus is required before opening of the sodium channels can occur (Totara & Derrickson, 2006). Once these channels are opened, an abundance of sodium ions will flood into the cell.

The occurrence of this severe depolarization that results from the influx of sodium ions is synonymous with the occurrence of an action potential. However, we must note that there is a clear difference between an action potential in a single neuron and the compound nerve action potential. In the action potential of a single neuron, the action potential recorded is representative of the depolarization of a single neuron alone. However, as a compound nerve consists of multiple neurons, the resulting action potential recorded represents the number of neurons that are active when stimulation was applied which is, essentially, the accumulated result of multiple action potentials (Kandel, Schwartz & Jessell, 2000). The larger the action potential, the more neurons were depolarized sufficiently to threshold value for an action potential to occur (Kandel, Schwartz & Jessell, 2000). In our experiments, as we did not isolate one neuron but observed the muscle as a whole, the action potentials recorded represent the compound nerve action potential.

The two nerves that will be subject to our experiment are the ulnar nerve and the median nerve. The ulnar nerve can be found on the arm just anterior of the tricep, branching off the medial chord of the brachial plexus (Elizaga 1998). Also branching off the brachial plexus is the median nerve which, down the radial part of the forearm and via the carpal tunnel of the wrist, innervates the lateral muscles of the palm of the hand (Totara & Derrickson, 2006).

The obtained the compound muscle action potential, combined with the knowledge of the physiology of neuromuscular activity, can provide insight on the abnormalities of nerve function. The main use of nerve conduction

studies is to determine the presence in neuropathies in patients (Oh, 2002). The many variables, such as latency or conduction velocity, that are identified in nerve conduction studies allow specific insight in the exact fault with the nerve depending on which variables is or is not abnormal. This further allows the identification of the pathophysiological nature of the neuropathy (Oh, 2002).

Method:**Measurement of the Voluntary EMG:**

1. The skin was prepared for adhesion with electrodes by cleaning with alcohol and lightly scouring with sandpaper.
2. The recording electrodes were then applied. The active electrode (negative) was placed over the hypothenar muscles, which are located at the lateral-dorsal border. We were careful that the electrode was placed over the area where there is maximum concentration of endplates as it was there that the initial potential change is negative and the amplitude of the muscle action potential is optimum.
3. The reference electrode (positive) was placed over a tendon that is located just distal to the fifth metacarpal-phalangeal joint; on the lateral surface of the fifth digit.
4. The ground electrode was firmly attached to the back of the hand at the dorsum of the wrist.
5. Once the electrodes are in place, recording, using the 'Scope version 5' software, may begin. The first recording was used simply to determine the accuracy of which the electrodes were attached. This, thus, represented the muscle in its resting state.

6. Step 5 was then repeated for different intensities of muscle contraction. The levels of intensity are small, medium and maximal force. Small force can be applied by using the pinky finger to resist against a weight, medium force is to be generated against a heavier weight and maximal force can be generated by attempting to lift against a weight that is pushing down against the finger. We were careful that the contraction is sustained for a certain period of time.

Measurement of Nerve Conduction Velocity:

1. Ensure that the setup of the recording and ground electrodes are as were used in the measurement of the voluntary EMG.
2. The stimulating electrodes were then placed over the ulnar nerve or the median nerve, depending on which we wished to experiment on. The anode was placed proximal to the cathode which was placed directly over the nerve.
3. Beginning with a weak stimulus (10 mA current) we delivered a brief pulse. We increased the current until a CMAP was recorded. If no CMAP was recorded beyond a current of 40 mA, we made the assumption that the stimulating electrodes were not correctly placed. If so, we repositioned the stimulating electrode and repeated this step. We made sure that the subject's arm was relaxed and the handheld stimulating electrode was pressed firmly against the nerve.
4. With a CMAP recorded, we have established the threshold stimulus. We continued increasing the current in a stepwise fashion until the amplitude of the CMAP did not increase which, thus, indicated that the supramaximal stimulus has been reached

5. We repeated steps 3 and 4 for the remaining nerve (the nerve that was not stimulated in step 2)
6. We repeated steps 3 and 4 for each nerve at a different site of stimulation; at a different distance from the recording electrodes.

Calculation of Velocities:

$V = \text{distance} / \text{time} =$

$((\text{distance at site 1} - \text{distance of site 2}) \times 10^{-2}) / ((\text{average latency at site 1} / \text{average latency at site 2}) \times 10^{-3})$

Velocity of Ulnar nerve = $((26.5 - 10.7) \times 10^{-2}) / ((7.28 - 3.1) \times 10^{-3}) = 50.97 \text{ ms}^{-1}$

Velocity of Median nerve = $((24.5 - 10.5) \times 10^{-2}) / ((9.7 - 6.7) \times 10^{-3}) = 35.00 \text{ ms}^{-1}$

Discussion:

The results obtained from Part A of our experiments (Graph 1), where we collated data for different intensities of sustained muscular contraction, are an expected result; accurately representing the underlying physiology behind the contraction of such muscles.

A muscle is composed of hundreds to thousands of cells known as muscle fibres (Totara & Derrickson, 2006) and a motor unit consists of a motor neuron and the muscle fibres innervated by that neuron (Kandel, Schwartz & Jessell, 2000). Knowing this, it becomes clear that the amount of force generated by a muscle is dependent on the number of motor units recruited and active at any one time (Purves et al, 2008). As, in Part A of our

experiment, we are measuring the voltage through a muscle, the number of motor units active during the utilisation of this muscle would directly affect the voltage output measured by the recording electrodes. With more motor units utilised, more nerve impulses from somatic motor neurons would be expected to occur, resulting in an increase in the number of muscle fibres stimulated in a muscle (Totara & Derrickson, 2006). Our results followed this described function (Graph 2) as there is an observable correlation between the intensity of contractile force applied by the experimental subject and the size of the voltage measured by the recording electrodes. At maximal sustained contraction, we observed the largest voltage output which exceeded 2 millivolts whereas, when the muscle was in its relaxed state, the lowest voltage output was obtained; barely any voltage was recorded as no motor units were active. As we experimented with sustained contraction, the frequency remained constant throughout all intensities of muscular contraction.

The CMAP obtained from Part B of our experiments also represented the physiological mechanisms involved in muscle contraction. Firstly, we can see that muscle action potentials display an ‘all-or-none’ response. This is clearly seen in our results (Tables 1, 2, 3 & 4), where stimulation at certain, lower, amperages provoked no response from a muscle whereas stimulation at higher, specific amperages was able to induce a response. This is because a certain level of depolarization (known as a threshold) must be reached before the voltage gated sodium channels will open (Totara & Derrickson, 2006). Some of the lower currents did not cause sufficient depolarisation and

(Tables 1, 2, 3 & 4), as the threshold was not reached, no action potential occurred.

It was also noticed that, upon stimulation with larger currents, the voltage output recorded from the muscle increased. This result can be attributed to the all-or-none response in combination with the variable size of motor neurons. As smaller motor neurons have a smaller surface area, it therefore has a higher overall resistance (Kandel, Schwartz & Jessell, 2000). And, according to Ohm's law ($V = IR$), with a higher resistance, a specific current that is applied to a smaller motor neuron would generate a larger voltage than if that same current was applied to a larger motor neuron with a smaller surface area (which, consequently, has a lower overall resistance). Knowing this, it becomes clear that, at some levels of stimulation, smaller neurons would be recruited whilst larger neurons would remain inactive. At Supramaximal stimulation, the current applied is sufficient to allow all neurons of all sizes to depolarize to threshold stimulation and become activated. Therefore, at currents larger than Supramaximal stimulation, the amplitude of CMAP is no larger than those resulting from Supramaximal stimulation as there are no more neurons, of any size, to be activated. The physiology described above applies to both the median nerve and the ulnar nerve; both nerves displayed similar results with regard to both the 'all-or-none' response and the concepts of threshold and supramaximal stimulation.

The accuracy of our experiment and the data resulting is, however, questionable. With regards to our experimental procedure, it was not the most precise of methods and the use of such methods could hamper the accuracy of our results. For example, the positioning of the stimulating

electrodes is crucial. If the stimulating electrode is not directly over the nerve, the current produced by the electrode will not be transferred completely to the nerve. Thus, though the device indicates that, for example, 30 milliamps (Table 3) was applied; if only 10 of the 30 milliamps reached the nerve due to poor positioning, we would wrongly assume that 30 milliamps was applied instead of 10 milliamps.

This could explain why, despite experimenting on the same nerve, the threshold and supramaximal stimulus differs between the different sites (Tables 3 & 4). It is entirely possible that 10 milliamps was all that was required for threshold to be reached but we falsely believed that 30 milliamps was required due to faulty positioning (Tables 3 & 4). This theory can be further supported by our results which show that, in both the median and the ulnar nerve, the threshold stimulus required was at a lower current at the 2nd site of stimulation, near the wrist, compared to the 1st site of stimulation, near the elbow (Tables 1, 2, 3 & 4). As the area near the wrist is smaller than near the elbow, the chances of poorly positioning the stimulating electrode is less likely and, thus, more likely that most of the current applied reaches the nerve which, consequently results in an apparently lower current required for threshold at the 2nd site relative to the 1st site. The only result that should differ between the two sites is the latency. With a greater distance to the recording electrodes the latency would, logically, be longer. The conduction velocity through the nerve and the stimulus strength required for both threshold and supramaximal should be the same for the same nerve (Totara & Derrickson, 2006). Major

differences within/between these results are indicative of faulty or inaccurate experimental procedure.

If we are to look at the class data (Table 5) the majority of the obtained results lie within the normal values for the conduction velocities of both the ulnar ($50\text{-}80\text{ms}^{-1}$) (Schubert, 1964) and median nerve ($45\text{-}70\text{ms}^{-1}$) (Kandel, Schwartz & Jessell, 2000). Between subjects, there are differences present in the resulting conduction velocities, but not to an alarming degree. The differences in obtained results can be attributed to the expected differences between individuals. For example, the differences between the body temperatures of experimental subjects could result in 1 to 2 meters per second difference per degree of temperature (Kandel, Schwartz & Jessell, 2000). Furthermore, not all subjects used their dominant hand in the experiment; it is known that the conduction velocities in the nerves of the dominant hand differ to that of the non-dominant hand (Kandel, Schwartz & Jessell, 2000). A few more factors to be considered is age (Norris, Shock & Wagman, 1953), usage of temperamental equipment in experimental procedures and possibly even simply errors in measurement.

Analysis of Clinical Motor Study:

1. Ulnar Motor Study - Normal

$$1. ((25.5 - 5.5) \times 10^{-2}) / ((5.2 - 2.4) \times 10^{-3}) = 71.4286 \text{ms}^{-1}$$

$$2. ((36.8 - 5.5) \times 10^{-2}) / ((6.9 - 2.4) \times 10^{-3}) = 69.5556 \text{ms}^{-1}$$

Median Motor Study - Normal

$$3. ((32.5 - 6.0) \times 10^{-2}) / ((7.3 - 2.8) \times 10^{-3}) = 58.8889 \text{ms}^{-1}$$

Ulnar Motor Study - Abnormal

1. The time base for this recording is 50 milliseconds, which differs from the time base of the previous Ulnar Nerve recording of 20 milliseconds.
2. The morphology of this CMAP is quite jagged as oppose to the usual smooth curve observed in normal patients. Furthermore, though not obvious visually, if we consider the difference in scale, we can see that the duration of this action potential is far longer than that of a normal patient.
3. Conduction Velocity = $\frac{(26.2 - 5.2) \times 10^{-2}}{(17.5 - 5.7) \times 10^{-3}} = 18.050 \text{ ms}^{-1}$. This conduction velocity is far slower than the expected conduction velocity of a normal patient. A likely cause for this is that damage has occurred to the myelin sheaths of the nerve. By acting as an electric insulator, the myelination of neurons can greatly increase conduction velocity (Purves et al, 2008) thus; it logically follows that conduction velocities will be decreased in the absence or damage of these myelin sheaths. It has been observed in mice that have deficits in the gene expression of the protein proteolipids that make myelin sheaths, the conduction velocities of nerves that contain these faulty sheaths are decreased (Tanaka et al, 2009).

Median Nerve Motor Study - Abnormal

1. The latency to segment 1 is 9 milliseconds. This is about 3 times longer than that of a normal study.
2. Conduction Velocity = $\frac{(29.5 - 6.5) \times 10^{-2}}{(12.7 - 8.6) \times 10^{-3}} = 56.09756 \text{ ms}^{-1}$

3. As the conduction velocity lies within the expected value, we can assume that there is no fault with the conducting ability of the neuron. The abnormality seems to reside in the significantly long latency period. As mentioned earlier, a key component that contributes to the latency period is the time taken for the neuromuscular transmission of chemicals. Thus, a longer latency period resulting from two motor studies of equal distances, given that conduction velocity is more or less the same, indicates that perhaps there is an error in the neuromuscular transmission of chemicals. Thus, a possible cause of this abnormality might be Lambert-Eaton myasthenic syndrome, where acetylcholine release is hampered due the inhibition of the voltage gated calcium channels in the pre-synaptic membrane (Newson-Davis J, 2004). With less acetylcholine released, sufficient depolarization to threshold would take longer if it even occurs at all and, thus, we will see a significantly larger latency period.
4. Furthermore, we notice that the amplitude of the abnormal median nerve is almost only a third of that seen in a normal median nerve. This provides more weight to the possibility of Lambert-Eaton Myasthenic syndrome as, with a deficit in acetylcholine release, neurons with a higher depolarization threshold would not be depolarized sufficiently to be stimulated. With less neurons active when stimulation is applied, and, since we're measuring compound nerve action potential, the amplitude would be expected to be lower.

Conclusion:

Through our experiments, we were able to accurately record an EMG during voluntary muscle contraction and also accurately determine the conduction

velocity of the ulnar and median nerves in the forearm of a human subject. Overall, despite the variations in obtained results, this experiment possessed sufficient accuracy as it reflected the result expected from the physiological mechanisms of muscle contraction and the actions of nerves. The all-or-none response and the specificity of a threshold and a supramaximal stimulus are all characteristics of neuromuscular action and were all observed in our results.

References:

- Tanaka H, Ma JM, Tanaka KF, Takao K , Komada M, Tanda K , Suzuki A , Ishibashi T, Baba H, Isa T, Shigemoto R, Ono K, Miyakawa T , Ikenaka K (July 2009) Mice with Altered Myelin Proteolipid Protein Gene Expression Display Cognitive Deficits Accompanied by Abnormal Neuron-Glia Interactions and Decreased Conduction Velocities, Journal of Neuroscience Volume 29, Issue 26, Pages 8363-8371

Newson-Davis J (February 2004) Lambert-Eaton Myasthenic syndrome Rev. Neurol. (Paris) Volume: 160, Issue 2, Pages 177-180

Norris, A. H., Shock, N. W., Wagman I. H. (1953) Age Changes in the Maximum Conduction Velocity of Motor Fibers of Human Ulnar Nerves. Journal of Applied Physiology, Volume: 5, Issue 10, Pages 589-593.

Elizaga, A., M. (1998). Illustrated Notes in Regional Anesthesia (October 1998)

Oh, S., J. (2002). Clinical Electromyography: Nerve Conduction Studies, pp. 1-20

Derrickson, Bryan. Tortara, Gerard J. (2006) Principles of Anatomy and Physiology 11th Edition

Kandel, Eric R. Schwartz, James H. Jessell, Thomas M. (2000) Principles of Neuroscience 4th Edition

Purves, Dale et al (2008) Neuroscience 4th Edition