Action Potentials

Case study

You are working for a pharmaceutical company attempting to develop new treatments for sufferers of multiple sclerosis. In order to gain a better understanding of the condition you have been asked to design and conduct an experiment against which your treatments can be compared.

Hypothesis 1

Increasing stimulus strength will increase the peak of the compound action potential.

Prediction of results 1

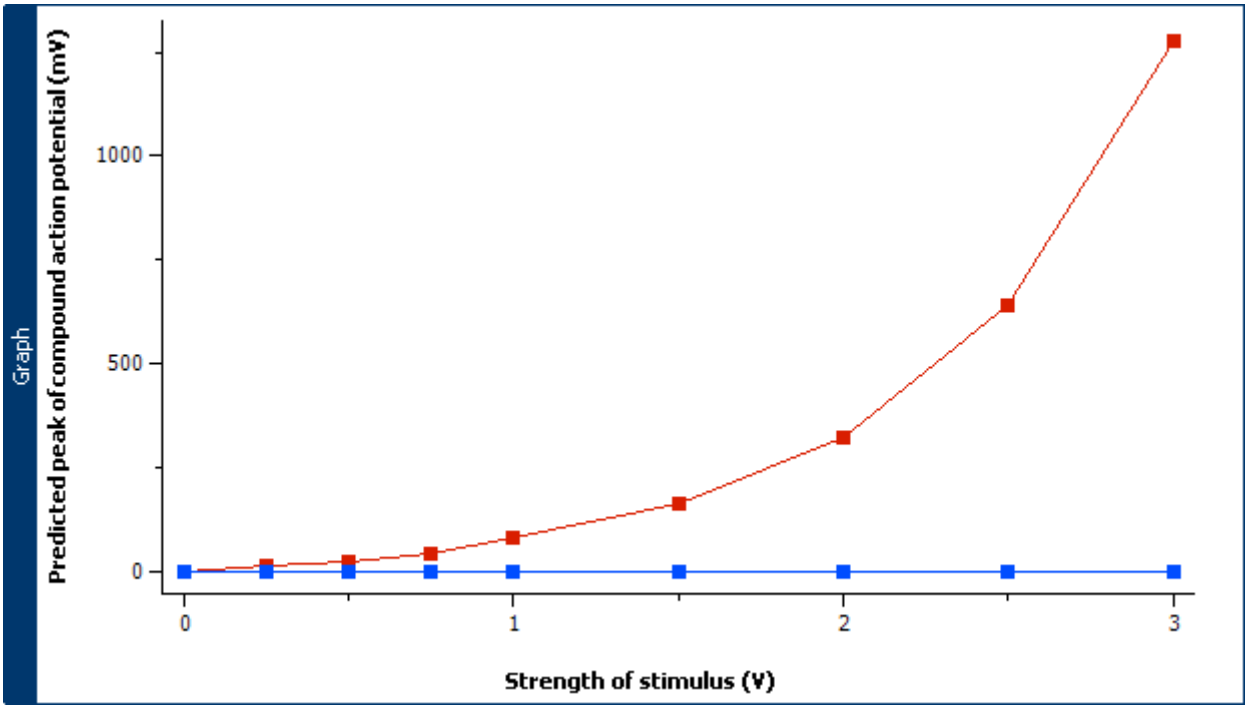

Figure 1: The graphed possible results of increased stimulus strength (V) on compound action potential peak (mV) in Bufo Marinus (Toad) sciatic nerve. Plots represent theoretical data if hypothesis is confirmed (red) and if the response is unaffected (blue).

Results 1

Table 2. Peak of compound action potential (mV) of Bufo Marinus Sciatic Nerve				
Strength of stimulus (V)	Replicate 1	Replicate 2	Replicate 3	Mean
0.00	0	0	0	0
0.25	0.069	0.055	0.051	0.058333333333333
0.50	0.143	0.182	0.639	0.321333333333333
0.75	3.493	4.564	5.469	4.508666666666667
1.00	5.681	6.147	6.698	6.175333333333333
1.50	7.858	7.186	7.5	7.514666666666667
2.00	7.536	7.454	8.446	7.812
2.50	8.018	7.916	8.592	8.175333333333333
3.00	8.177	8.834	8.692	8.567666666666667

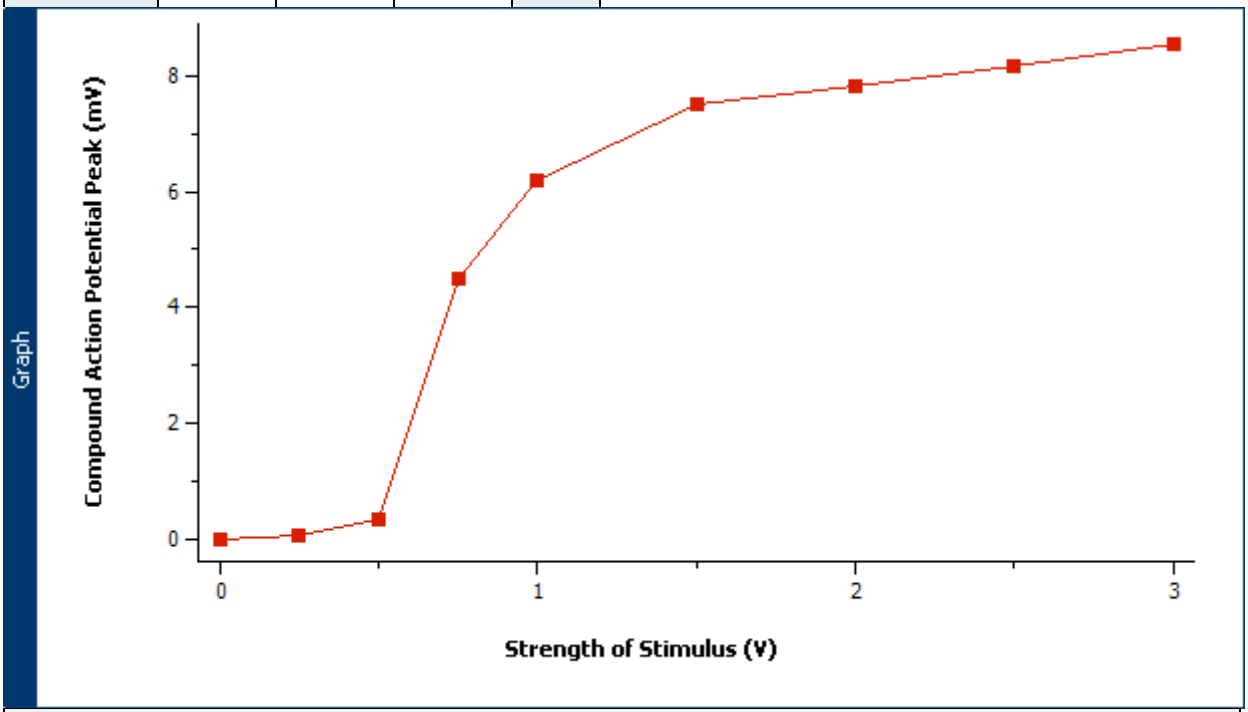

Figure 2:

The relationship between Compound Action Potential Peaks in a Sciatic Nerve of Bufo Marinus (Toad), relative to varied strengths of stimulus (V).

Comparative analysis

Table 3:

The comparison of Maximum Peak Compound Action Potential (mV) and Latency period of that compound action potential (msec) between three different Bufo Marinus' (Toads) Sciatic nerves.

Group	Maximum peak compound action potential (mV)	Latency period of that compound action potential (msec)
Your own	8.56766(mV)	1.01(msec)
Alternate 1	18.864(mV)	1.01(msec)
Alternate 2	13.789(mV)	0.19(msec)

Hypothesis 2

When applied to a Bufo Marinus Sciatic nerve; local anaesthetic blocks voltage gated sodium channels by inhibiting the gates from opening, hence preventing the influx of sodium ions into the cell membrane. Due to this, the longer the time after exposure to local anaesthetic the lower the compound action potential peak (mV) will be.

Materials & methods 2

The experiment was conducted according to the following procedure. The first step was to dissect the sciatic nerve of the Bufo Marinus. The Powerlab was set up with the recording electrodes attached at correct locations. The interval of thirty seconds between each stimulation was determined. This was a control variable. The voltage applied to the sciatic nerve was determined to be 0.75V (thus also a control variable). The initial compound action potential peak (mV) was measured with no anaesthetic applied, using the Powerlab. This value was to be the negative control as no stimulus would affect the result. Enough local anaesthetic (Lignocaine 20mg/mL) was applied to the nerve so that it was completely covered (approximately five drops). Frog Ringer solution was continuously applied to the sciatic nerve to simulate extracellular fluid. Nerve was stimulated using stimulating electrodes and left for 30 seconds intervals. This step was repeated for 240seconds, measuring the compound action potential peak.

Prediction of results 2

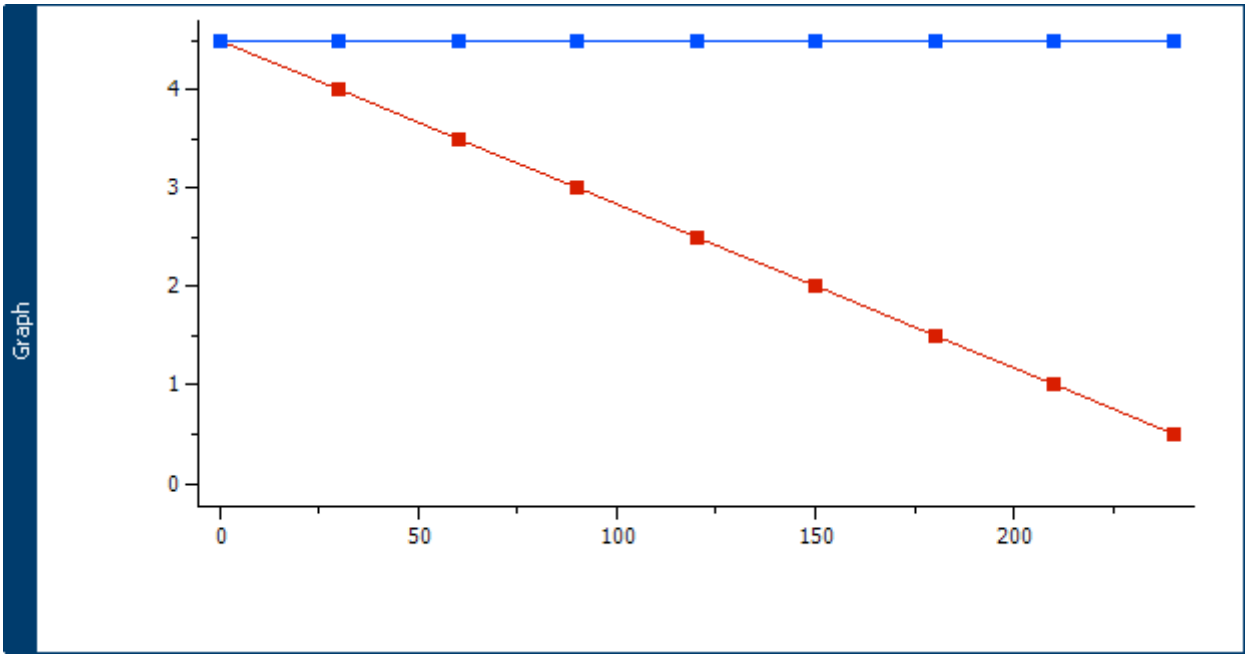

Figure 3.
The predicted relationship between the Compound Action Potential Peak (mV) by applying Anaesthetic (Lignocaine 20mg/mL) to the Sciatic Nerve of the Bufo Marinus and the varied (30 second interval) time.

Results 2

Write a paragraph of text in the box below, describing the important trends and relationships for all data presented from experiment 2:

Figure 3 represents the theoretical results of the experiment between compound action potential peak and time after anaesthetic is induced in a sciatic nerve of a Bufo Marinus. The significan trend in this figure is a decreasing linear relationship between the controlled increased time after anaesthetic and the compound action potential peak. This prediction was relatively supported by the data graphed in figure 4 and table 5, showing that with increased time the compound action potential peak decreased, although not linearly (as represented in the predictions). The mean results in table 5 show results for only one replicate as the sciatic nerve would not produce sufficient compound action potential for replicates two and three. Although replicate one produced great results and thus the others could be disregarded. This could be because the nerve has died or fatigued due to large amounts of stimulation. The results of table 5 and figure 4 show compatibility with predicted results. The implied relationship from this data is that Compound action potential mV in relation to that of increased time after anaesthetic is administered decreases due to the anaesthetic inhibiting voltage gated sodium channels. Therefore further influx of sodium into the cell membrane.

Table 5.
Gathered Values of the Compound Action Potential Peak, from conducted experiment of applying Anaesthetics (Lignocaine 20mg/mL) to the Sciatic Nerve of the Bufo Marinus

Independent Variable	Replicate 1	Replicate 2	Replicate 3	Mean
0.00	7.173			7.173
30.00	3.211			3.211
60.00	1.909			1.909
90.00	1.222			1.222
120.00	0.683			0.683

150.00	0.353			0.353
180.00	0.264			0.264
210.00	0.128			0.128
240.00	0.064			0.064

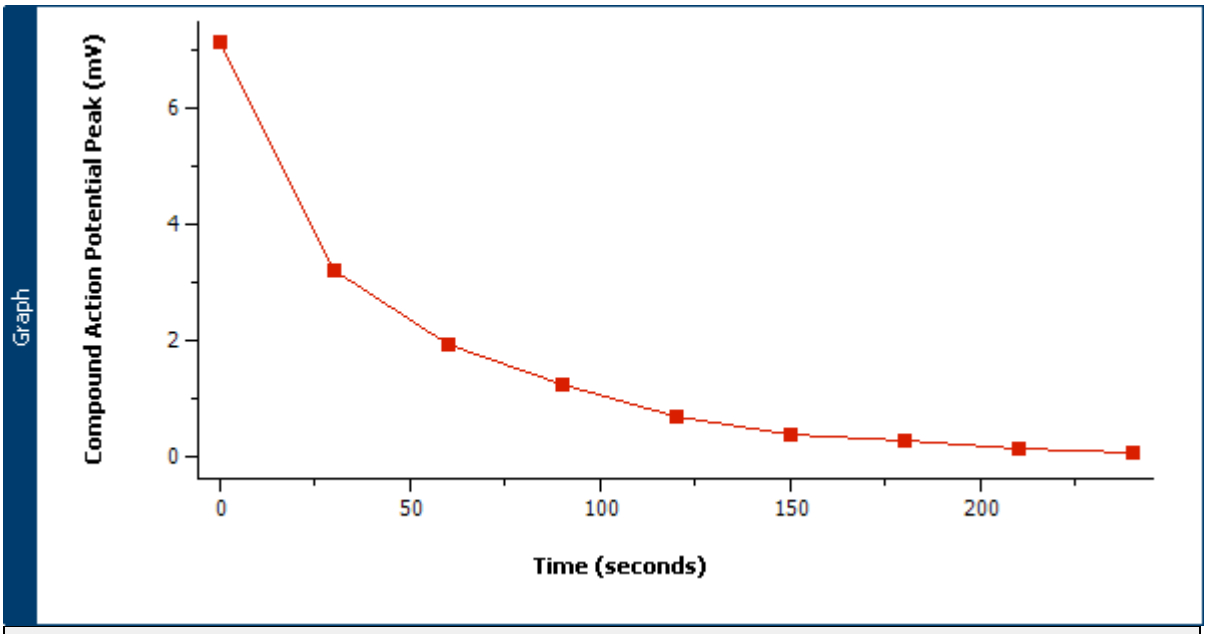

Figure 4.

Relationship between Time (second) since the Anaesthetic (find experimental name) was applied and the varying of the Compound Action Potential Peak (mV) value of the Bufo Marinus's Sciatic Nerve.

Discussion

Remember to treat these questions like a short answer question in the final exam: be specific, clear, and concise.

1. Briefly describe (in complete sentences) whether the results of your first experiment confirm or disconfirm your hypothesis, and any errors in your data.

The hypothesis that increasing stimulus strength will increase the peak of the compound action potential as represented in figure 1 was supported by data graphed in figure 2 and table 1. This was represented by the increase in values of compound action potential peak mV for increased stimulus strength.

2. Why do you think the peak of the compound action potential changes with different strength stimuli?

The peak of the compound action potential could increase as a result of increased stimulus strength. This could be due to the action potentials from the axons involved, becoming excited by this stimulation. The more axons that become excited the more excited the nerve becomes and thus the greater the peak of the compound action potential. Action potential has an “all or nothing” nature and occurs as a result of extracellular stimulation.

3. What reasons can you give for the differences in peak and latency between nerves in your comparative analysis?

Possible explanations for the difference in peak and latency between nerves could be a result of differing amounts of stimulus applied to the nerve, creating tiredness or fatigue in the nerve. The amount of frog ringer applied to the sciatic nerve. This solution produces a simulated extracellular fluid around the nerve, helping it to not dehydrate. Also action potentials are generated as a result of stimulation of the extracellular membrane, thus if this fluid is not maintained the peak of action potential will not be as great. The latency period between differing nerves could be due to nerve damage or death. For example if the nerve was not dissected properly or stretched too far. Increased stimulation creates increased compound action potential, however if the stimulation is too great or the latency too long or short this could severely affect results between differing Bufo Marinus sciatic nerves.

4. Briefly describe (in complete sentences) whether the results of your second experiment confirm or disconfirm your hypothesis, and any errors in your data.

The hypothesis predicted was that when applied to a Bufo Marinus Sciatic nerve; local anaesthetic blocks voltage gated sodium channels by inhibiting the gates from opening, hence preventing the influx of sodium ions into the cell membrane. Due to this, the longer the time after exposure to local anaesthetic the lower the compound action potential peak (mV) will be. This is supported by the resulting relationship in figure 4 and data recorded in table 5. This data displayed evidence to this prediction in the decreasing compound action potential peak values, relative to the longer time intervals proceeding local anaesthetic application. Error in the data could be attributed to the nerve dying after the first replication, making it hard to produce sufficient comparable data.

5. What biological processes do you think have caused the trends your results illustrate?

The possible biological processes of this experiment could be that the induced stimulus in the extracellular membrane of the cell created an action potential in the nerve due to excitation of axons. The applied Local anaesthetic inhibits voltage gated sodium channels and thus no more sodium can flow through membranes, no influx of sodium ions. Thus with increased time in seconds this anaesthetic becomes more active or begins to work. Thus the action potential peak of the compound will be lowered as no influx of sodium ions will be produced.

6. In the case study you are working on multiple sclerosis, how do you think decreasing body temperature would affect patients with multiple sclerosis?

Heat in demylenated nerve fibres cause action potential to decrease or even stop. Decreasing the body temperature of a person with Multiple Sclerosis would increase action potential peak of the compound as a result of this. Multiple sclerosis is a disease that attacks the myelin of an axon therefore disrupting its ability to excite neurones and create an action potential. Thus if a person with this disease were to experience decreased body temperature compound action potential peaks would increase with increased myelination of axons.

7. You have a friend who has been diagnosed with multiple sclerosis who does not have a strong science background. Explain to them (using complete sentences) what causes their symptoms using language which they can understand.

For people with multiple sclerosis, myelin is removed from the axon causing it to slow down the conduction speed at which the action potential can move down the axon. Symptoms they experience are loss of muscle control, vision impairment, balance problems and numbness throughout body. These could be attributed to loss of myelin that helps to transmit nerve impulses through the body. Therefore axons cannot excite the neurons in the body and ultimately action potential cannot be created because of lack of stimulation from excitation of axons.

References

List any references you have used in the panel below:

Campbell, Biology, 8th Edition, MasteringBiology, viewed 5/4/11

DuBois, Marc L. Action Potential: Biophysical and Cellular Context, Initiation, Phases, and Propagation. New York: Nova Science, 2010.

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