13 Excitation and Nerve Conduction

W.F.H.M. Mommaerts, D. Junge, and M.B. Jackson

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13.1 Introduction

Living cells are generally distinguished by the maintenance of an ionic composition in their cytoplasm which differs from that in the tissue fluid bathing them. The cytoplasm is rich in K+ ions and low in Na+, while in the surrounding milieu the reverse holds, and this imbalance is held with considerable stability by homeostatic mechanisms (see Chap. 11). This differential ionic distribution creates the possibility of developing a property called excitability which is found in many different biological systems, including unicellular animals and plants and the nervous systems of higher animals. A simple form of excitability is seen when a touch to the exterior of the animal gives rise to a resulting muscular movement, and indeed until the invention of the electrophysicist in the nineteenth century this was the only way in which the occurrence of excitation could be detected. Subsequently the production of action potentials, impulses of electrical activity, could be seen independent of muscular contraction. It was also learned that muscular contraction and excitation could be electrically elicited, so that their experimental study was no longer dependent upon the biological generation of a functionally normal stimulus delivered to a receptor. Thus the development of the physiology of excitation, conduction, transmission, and response became largely identical with the development of electrophysiology. A brief summary of this classical phase follows, then a discussion of the striking developments which led to the molecular interpretation of the electrical events.

13.2 The Action Potential

13.2.1 Single Nerve Fiber

Recording Technique. A section of nerve may be placed in a moist chamber, over a series of silver or platinum electrodes, and excited by shocks delivered to one pair of electrodes. The resulting activity may then be monitored at other locations along the nerve. The source of the recorded signals may be understood by considering the activity of a single axon in the nerve, as shown in Fig. 13.1. One end of the nerve has been crushed, so that only the activity under the recording electrode is observed – this is called a monophasic recording. The activity is recorded with an Ac amplifier, so the initial potential is zero (trace 1). When the nerve is excited by a shock, after a short interval the potential under the recording electrode becomes negative by a small amount, perhaps 1 mV (trace 2). This is caused by the reversal of potential inside the axon, in the active region. After the impulse has passed by, the potential returns to zero again (trace 3). If two electrodes are placed outside normal regions of the nerve, then a negative-positive sequence is seen, known as the biphasic action potential. Thus, the same basic phenomenon, the action potential, can have different experimental manifestations as a result of differences in the recording technique used.

13.2.2 Compound Action Potential

When the activity of a whole nerve is recorded by the above method, a compound action potential such as that in Fig.
Fig. 13.1. Monophasic recording of extracellular action potential. 
*Left:* External potential recorded with respect to crushed end of nerve at three different times as action potential passes by external electrode. Increasing negativity of recording electrode plotted as upwards. 
*Right:* Explanation based on activity of a single axon

13.2 is seen. In part a of the figure a shock (stimulus) $S_1$ is applied to the nerve and a few milliseconds later the large $\alpha_1$ elevation is seen, as the activity propagates along the nerve and reaches the recording electrode. In part b the stimulus intensity is increased slightly and two elevations are seen, $\alpha_2$ and $\beta_2$. The $\beta_2$ component results from the activity of axons with a slower conduction velocity (about 22 m/s) than those giving rise to the $\alpha_2$ component (about 41 m/s), so their signal takes longer to reach the recording site. The $\beta_2$ elevation was seen when the stimulus exceeded the threshold level for that group of axons. This figure also illustrates a general point that nerve fibers with slower conduction velocities generally have higher thresholds for excitation than more rapidly conducting fibers.

### 13.2.3 Conduction Velocity and Fiber Diameter

By making cross-sections of nerves and staining them with osmium, it is possible to see that the fiber diameters are not distributed continuously but fall into distinct classes. Combining such histological studies with electrophysiological recordings from whole nerves allows us to compare the conduction velocities of fiber groups with various diameters.

![Fig. 13.2a,b. Distinct elevations in compound action potential of frog sciatic nerve [10]. Stimulus $S_1$ is larger than $S_2$](image)

A selection of this type of data in mammalian afferent nerves is shown in Table 13.1. Afferent fibers in muscle nerves have historically been divided into groups I–IV, and in cutaneous nerves into groups A–C as shown. There is a clear suggestion that the nerve fibers with the largest diameters have the fastest conduction velocities. In other studies [10], a monotonic relation between conduction velocity and fiber diameter has been found.

The action potential in a single nerve axon is all-or-none, that is, it either occurs in response to a stimulus or it does not, and the amplitude of the action potential is independent of the stimulus strength. After an impulse has passed, excitability is temporarily lost. During the absolute refractory period, no stimulus of any strength can cause an action potential. Excitability then returns during the relative refractory period, when the threshold is higher than normal. For the quantitative correlation between diameter and conduction velocity, see Sect. 13.4.

### Table 13.1. Fiber diameters and conduction velocities of mammalian nerve fibers [3,32]

<table>
<thead>
<tr>
<th>Group</th>
<th>Function</th>
<th>Diameter (µm)</th>
<th>Conduction velocity (m/s)</th>
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</thead>
<tbody>
<tr>
<td>I</td>
<td>$\alpha$: Afferents from muscle spindles and Ib afferents from Golgi tendon organs, motor efferents to skeletal muscles</td>
<td>ca. 12–20</td>
<td>ca. 70–120</td>
</tr>
<tr>
<td>II</td>
<td>$\beta$: Afferents from cutaneous mechanoreceptors, afferents from secondary muscle spindle endings</td>
<td>ca. 6–12</td>
<td>ca. 30–70</td>
</tr>
<tr>
<td>III</td>
<td>$\gamma$: Efferents to muscle spindles, afferents from deep pressure receptors of muscle, cutaneous afferents for temperature and nociception, preganglionic sympathetic efferents around 3</td>
<td>ca. 2–5</td>
<td>ca. 10–30</td>
</tr>
<tr>
<td>IV</td>
<td>$\delta$: Cutaneous afferents for temperature and nociception, preganglionic sympathetic efferents around 1</td>
<td>ca. 0.5–2</td>
<td></td>
</tr>
</tbody>
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