WE ARE BEINGS OF ENERGY AND OUR MUSCLES WORK MAGNETICALLY

We will start to teach the lesson from fifth grade where we were all taught our bodies are made of atoms. The atoms have electrons in their outer shell and electrons do not touch, they repel each other. So no two atoms can touch. You cannot touch the chair you sit in. The atoms in your body are held in place by many different energy fields. It is the balance of these fields that makes health possible. So we are beings of energy.

Muscles contract by using Electro-magnetic-static attraction forces to grab and slide over each other and provide energy from ATP etc to make muscles work. It is obvious that if the body electric is balanced and the voltage and amperage maximized, muscles will work better.
Standardization of facilitation of compound muscle action potentials evoked by magnetic stimulation of the cortex. Results in healthy volunteers and in patients with multiple sclerosis.

Abstract

Cybermagnetic Chair

Muscles contract by using Electro-magnetic-static attraction forces to grab and slide over each other and provide energy from ATP etc to make muscles work. It is obvious that if the body electric is balanced and the voltage and amperage maximized, muscles will work better.

The action area of this electro-magnetic-static grabbing in the muscles is so small (molecular) it is not affected by macro magnetic action.

As new ATP attaches to the myosin head, the link between myosin and actin weakens, and the cross bridge detaches.
The action area of this magnetic static grabbing in the muscles is so small (molecular) it is not affected by macro magnetic action but:

Here is proof that muscles make magnetic action


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PMCID: PMC1280763

Abstract

We present the first measurements of the magnetic field from a single muscle fiber of the frog gastrocnemius, obtained by using a toroidal pickup coil coupled to a room-temperature, low-noise amplifier. The axial currents associated with the magnetic fields of single fibers were biphasic and had peak-to-peak amplitudes ranging between 50 and 100 nA, depending primarily on the fiber radius. With an intracellular microelectrode, we measured the action potential of the same fiber, which allowed us to determine that the intracellular conductivity of the muscle fiber in the core conductor approximation was 0.20 +/- 0.09 S/m. Similarly, we found that the effective membrane capacitance was 0.030 +/- 0.011 F/m2. These results were not significantly affected by the anisotropic conductivity of the muscle bundle. We demonstrate how our magnetic technique can be used to determine the transmembrane action potential without penetrating the membrane with a microelectrode, thereby offering a reliable, stable, and atraumatic method for studying contracting muscle fibers.

Full text
A strong constant magnetic field affects muscle tension development in bullfrog neuromuscular preparations.

Satow Y, Matsunami K, Kawashima T, Satake H, Huda K.

Abstract
Effects of a constant magnetic field (CMF) of 0.65 T on muscle tension over 9 h were studied in the neuromuscular preparation of the bullfrog sartorius muscle. Tension was developed every 30 min by stimulation of the sciatic nerve (nerve stimulation) or of the sartorius muscle itself (muscle stimulation). In sciatic nerve stimulation, tension decreased rapidly for the first 3-4 h at a similar rate in both test (exposed to CMF) and control muscles. However, the rate of decrease became smaller and almost leveled off after 3-4 h in the test muscles, whereas tension continued to decrease monotonically in control muscles. The slope of the decrease for these later periods was significantly different between the test and the control conditions. Accordingly, tension was larger in test than in control muscles. In muscle stimulation, tension decreased monotonically from the start of experiments in control muscles, while tension in test muscles maintained their initial values for almost 3 h. Thereafter they started to decrease with a similar rate to the control. Hence, tension was always larger in test than in control muscles. A similar pattern of temporal change was observed for the rate of rise of the maximum tension for nerve or muscle stimulation. However, a significant difference was detected only in the case of muscle stimulation. The present results showed that a strong CMF of 0.65 T had biological effects on tension development of the bullfrog sartorius muscle by stimulation of the sciatic nerve as well as by stimulation of muscle itself. The presence of a small AC magnetic field component leaves open the possibility of an AC, rather than a CMF effect.

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OBJECTIVE:
To study the effect of a magnetic field on measurements of urethral function in women with stress urinary incontinence.

PATIENTS OR OTHER PARTICIPANTS:
Twenty-six consecutive women with diagnosis of stress urinary incontinence (SUI).

EVALUATION:
History and physical examination, neurologic exam, urethrocystoscopy, urodynamic testing with water-filling cystometry, urethral profilometry at rest, during coughing, and during coughing while performing a levator ani contraction (knack maneuver).

INTERVENTION:
The same urodynamic procedures were performed again after the subjects were asked to step on specifically designed magnets (magnetic cushion device).

STATISTICAL ANALYSIS:
Two-tailed student t test.

MAIN OUTCOME MEASURES:
Urethral pressure at rest, during coughing, and during coughing while performing a levator ani contraction.

RESULTS:
Mean age was 58.3 years (range: 36-81), mean parity 2.8 (range: 0-8). The urodynamic parameters measured without and with the use of the magnetic cushion device were not found to be different except for the knack maneuver. The pressure in the urethra during the knack maneuver while the subjects were stepping on the magnetic device was significantly higher than the 1 obtained without the magnetic field.

CONCLUSION:
In our patient population, a magnetic field increases the efficacy of voluntary levator ani contractions.
All Chemicals have Atoms with Electrons in their Outer Shell.

Electrons Repel Each other and never touch. Chemicals + Atoms NEVER EVER TOUCH

All Chemical Reactions are a Result of Energetic, Electro-Magnetis-Static-Quantic Field Interactions

All Chemistry is Electro-Chemistry

IMUNE
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Evidence Based Natural Energetic Medicine Education
Muscles are one of those things that most of us take completely for granted, but they are incredibly important for two key reasons:

- Muscles are the "engine" that your body uses to propel itself. Although they work differently than a car engine or an electric motor, muscles do the same thing -- they turn energy into motion.

- It would be impossible for you to do anything without your muscles. Absolutely everything that you conceive of with your brain is expressed as muscular motion. The only ways for you to express an idea are with the muscles of your larynx, mouth and tongue (spoken words), with the muscles of your fingers (written words or "talking with your hands") or with the skeletal muscles (body language, dancing, running, building or fighting, to name a few).

Because muscles are so crucial to any animal, they are incredibly sophisticated. They are efficient at turning fuel into motion, they are long-lasting, they are self-healing and they are able to grow stronger with practice. They do everything from allowing you to walk to keeping your blood flowing!

When most people think of "muscles," they think about the muscles that we can see. For example, most of us know about the biceps muscles in our arms. But there are three unique kinds of muscle in any mammal's body:

- **Skeletal muscle** is the type of muscle that we can see and feel. When a body builder works out to increase muscle mass, skeletal muscle is what is being exercised. Skeletal muscles attach to the skeleton and come in pairs -- one muscle to move the bone in one direction and another to move it back the other way. These muscles usually contract voluntarily, meaning that you think about contracting them and your nervous system tells them to do so. They can do a short, single contraction (twitch) or a long, sustained contraction (tetanus).

- **Smooth muscle** is found in your digestive system, blood vessels, bladder, airways and, in a female, the uterus. Smooth muscle has the ability to stretch and maintain tension for long periods of time. It contracts involuntarily, meaning that you do not have to think about contracting it because your nervous system controls it automatically. For example, your stomach and intestines do their muscular thing all day long, and, for the most part, you never know what's going on in there.

- **Cardiac muscle** is found only in your heart, and its big features are endurance and consistency. It can stretch in a limited way, like smooth muscle, and contract with the force of a skeletal muscle. It is a twitch muscle only and contracts involuntarily.
Skeletal-muscle Basics

Skeletal muscle is also called striated muscle, because when it is viewed under polarized light or stained with an indicator, you can see alternating stripes of light and dark.

Skeletal muscle has a complex structure that is essential to how it contracts. We will tease apart a skeletal muscle, starting with the largest structures and working our way to the smaller ones.

The basic action of any muscle is contraction. For example, when you think about moving your arm using your biceps muscle, your brain sends a signal down a nerve cell telling your biceps muscle to contract. The amount of force that the muscle creates varies -- the muscle can contract a little or a lot depending on the signal that the nerve sends. All that any muscle can do is create contraction force.

A muscle is a bundle of many cells called fibers. You can think of muscle fibers as long cylinders, and compared to other cells in your body, muscle fibers are quite big. They are from about 1 to 40 microns long and 10 to 100 microns in diameter. For comparison, a strand of hair is about 100 microns in diameter, and a typical cell in your body is about 10 microns in diameter.

A muscle fiber contains many myofibrils, which are cylinders of muscle proteins. These proteins allow a muscle cell to contract. Myofibrils contain two types of filaments that run along the long axis of the fiber, and these filaments are arranged in hexagonal patterns. There are thick and thin filaments. Each thick filament is surrounded by six thin filaments.

Thick and thin filaments are attached to another structure called the Z-disk or Z-line, which runs perpendicular to the long axis of the fiber (the myofibril that runs from one Z-line to another is called a sarcomere). Running vertically down the Z-line is a small tube called the transverse or T-tubule, which is actually part of the cell membrane that extends deep inside the fiber. Inside the fiber, stretching along
the long axis between T-tubules, is a membrane system called the **sarcoplasmic reticulum**, which stores and releases the calcium ions that trigger muscle contraction.
Contracting a Muscle

During contraction, the thin filaments slide past the thick filaments, shortening the sarcomere.

The thick and thin filaments do the actual work of a muscle, and the way they do this is pretty cool. Thick filaments are made of a protein called myosin. At the molecular level, a thick filament is a shaft of myosin molecules arranged in a cylinder. Thin filaments are made of another protein called actin. The thin filaments look like two strands of pearls twisted around each other.

During contraction, the myosin thick filaments grab on to the actin thin filaments by forming crossbridges. The thick filaments pull the thin filaments past them, making the sarcomere shorter. In a muscle fiber, the signal for contraction is synchronized over the entire fiber so that all of the myofibrils that make up the sarcomere shorten simultaneously.

There are two structures in the grooves of each thin filament that enable the thin filaments to slide along the thick ones: a long, rod-like protein called tropomyosin and a shorter, bead-like protein complex called troponin. Troponin and tropomyosin are the molecular switches that control the interaction of actin and myosin during contraction.

While the sliding of filaments explains how the muscle shortens, it does not explain how the muscle creates the force required for shortening. To understand how this force is created, let’s think about how you pull something up with a rope:

1. Grab the rope with both hands, arms extended.
2. Loosen your grip with one hand, let’s say the left hand, and maintain your grip with the right.
3. With your right hand holding the rope, change your right arm’s shape to shorten its reach and pull the rope toward you.

4. Grab the rope with your extended left hand and release your right hand's grip.

5. Change your left arm's shape to shorten it and pull the rope, returning your right arm to its original extended position so it can grab the rope.

6. Repeat steps 2 through 5, alternating arms, until you finish.

Muscles create force by cycling myosin electro-magnetic-static cross bridges.

To understand how muscle creates force, let's apply the rope example.

Myosin molecules are golf-club shaped. For our example, the myosin clubhead (along with the crossbridge it forms) is your arm, and the actin filament is the rope:

1. During contraction, the myosin molecule forms a electro-magnetic-static bond with an actin molecule on the thin filament (gripping the rope). This electro-magnetic-static bond is the crossbridge. For clarity, only one cross-bridge is shown in the figure above (focusing on one arm).

2. Initially, the crossbridge is extended (your arm extending) with adenosine diphosphate (ADP) and inorganic phosphate (P\(_i\)) attached to the myosin.

3. As soon as the crossbridge is formed, the myosin head bends (your arm shortening), thereby creating force and sliding the actin filament past the myosin (pulling the rope). This process is called the power stroke. During the power stroke, myosin releases the ADP and P\(_i\).

4. Once ADP and P\(_i\) are released, a molecule of adenosine triphosphate (ATP) electro-magnetic-static binds to the myosin. When the ATP binds, the myosin releases the actin molecule (letting go of the rope).

5. When the actin is released, the ATP molecule gets split into ADP and P\(_i\) by the myosin. The energy from the ATP resets the myosin head to its original position (re-extending your arm).

6. The process is repeated. The actions of the myosin molecules are not synchronized -- at any given moment, some myosins are electro-magnetic-static attaching to the actin filament (gripping the rope), others are creating force (pulling the rope) and others are electro-magnetic-static releasing the actin filament (releasing the rope).

The contractions of all muscles are triggered by electrical impulses, whether transmitted by nerve cells, created internally (as with a pacemaker) or applied externally (as with an electrical-shock stimulus).
Triggering and Reversing Contraction

The coupling process leading from electrical signal (excitation) to electro-magnetic-static contraction in skeletal muscle.

The trigger for a muscle contraction is an electrical impulse. The electrical signal sets off a series of events that lead to crossbridge cycling between myosin and actin, which generates force. The series of events is slightly different between skeletal, smooth and cardiac muscle.

Let’s take a look at what occurs within a skeletal muscle, from excitation to contraction to relaxation:

1. An electrical signal (action potential) travels down a nerve cell, causing it to release a chemical message (neurotransmitter) into a small gap between the nerve cell and muscle cell. This gap is called the synapse.
2. The neurotransmitter crosses the gap, binds to a protein (receptor) on the muscle-cell membrane and causes an action potential in the muscle cell.
3. The action potential rapidly spreads along the muscle cell and enters the cell through the T-tubule.
4. The action potential opens gates in the muscle’s calcium store (sarcoplasmic reticulum).
5. Calcium ions flow into the cytoplasm, which is where the actin and myosin filaments are.
6. Calcium ions electro-magnetic-statically bind to troponin-tropomyosin molecules located in the grooves of the actin filaments. Normally, the rod-like tropomyosin molecule covers the sites on actin where myosin can form crossbridges.
7. Upon binding calcium ions, troponin changes shape and slides tropomyosin out of the groove, exposing the actin-myosin binding sites.
8. Myosin electro-magnetic-statically interacts with actin by cycling electro-magnetic-static crossbridges, as described previously. The muscle thereby creates force, and shortens.
9. After the action potential has passed, the calcium gates close, and calcium pumps located on the sarcoplasmic reticulum remove calcium from the cytoplasm.
10. As the calcium gets pumped back into the sarcoplasmic reticulum, calcium ions come off the troponin.
11. The troponin returns to its normal shape and allows tropomyosin to cover the actin-myosin binding sites on the actin filament.
12. Because no binding sites are available now, no crossbridges can form, and the muscle relaxes.

As you can see, muscle contraction is regulated by the level of calcium ions in the cytoplasm. In skeletal muscle, calcium ions work at the level of actin (actin-regulated contraction). They move the troponin-tropomyosin complex off the binding sites, allowing actin and myosin to interact.

All of this activity requires energy. Muscles use energy in the form of ATP. The energy from ATP is used to reset the myosin crossbridge head and release the actin filament. To make ATP, the muscle does the following:

1. Breaks down creatine phosphate, adding the phosphate to ADP to create ATP
2. Carries out **anaerobic respiration**, by which glucose is broken down to lactic acid and ATP is formed.

3. Carries out **aerobic respiration**, by which glucose, glycogen, fats and amino acids are broken down in the presence of oxygen to produce ATP (see How Exercise Works for details).

Muscles have a mixture of two basic types of fibers: fast twitch and slow twitch. **Fast-twitch fibers** are capable of developing greater forces, contracting faster and have greater anaerobic capacity. In contrast, **slow-twitch fibers** develop force slowly, can maintain contractions longer and have higher aerobic capacity. **Training** can increase muscle mass, probably by changing the size and number of muscle fibers rather than the types of fibers. Some athletes also use performance-enhancing drugs, specifically **anabolic steroids**, to build muscle, although this practice is dangerous and is banned in most athletic competitions.

Cardiac and Smooth Muscle

While most of the processes are similar, there are some notable differences between the actions of skeletal, cardiac and smooth muscle.

Cardiac-muscle cells are **striated**, and are a lot like skeletal-muscle cells except that in cardiac muscle, the fibers are **interconnected**. The **sarcoplasmic reticulum** of cardiac-muscle cells is not as well-developed as that of skeletal-muscle cells. Cardiac-muscle contraction is **actin-regulated**, meaning that the calcium ions come both from the sarcoplasmic reticulum (as in skeletal muscle) and from outside the cell (as in smooth muscle). Otherwise, the chain of events that occurs in cardiac-muscle contraction is similar to that of skeletal muscle.

Compared to skeletal muscle, **smooth-muscle cells** are small. They are spindle-shaped, about 50 to 200 microns long and only 2 to 10 microns in diameter. They have no striations or sarcomeres. Instead, they have bundles of thin and thick filaments (as opposed to well-developed bands) that correspond to
myofibrils. In smooth-muscle cells, **intermediate filaments** are interlaced through the cell much like the threads in a pair of "fish-net" stockings. The intermediate filaments anchor the thin filaments and correspond to the Z-disks of skeletal muscle. Unlike skeletal-muscle cells, smooth-muscle cells have no troponin, tropomyosin or organized sarcoplasmic reticulum.

As in skeletal-muscle cells, **contraction** in a smooth-muscle cell involves the forming of electro-magnetic-static crossbridges and thin filaments sliding past thick filaments. However, because smooth muscle is not as organized as skeletal muscle, **shortening occurs in all directions**. During contraction, the smooth-muscle cell's intermediate filaments help to electro-magnetic-statically draw the cell up, like closing a drawstring purse.

Calcium ions regulate contraction in smooth muscle, but they do it in a slightly different way than in skeletal muscle:

1. **Calcium ions** come from outside of the cell.
2. Calcium ions electro-magnetic-statically bind to an enzyme complex on myosin, called **calmodulin-myosin light chain kinase**.
3. The enzyme complex breaks up ATP into ADP and transfers the P, directly to myosin.
4. This P, transfer activates myosin.
5. Myosin forms electro-magnetic-static crossbridges with actin (as occurs in skeletal muscle).
6. When calcium is pumped out of the cell, the P, gets removed from myosin by another enzyme.
7. The myosin becomes inactive, and the muscle relaxes.

This process is called **myosin-regulated contraction**.

---

**End Plate Potential And Excitation Of The Skeletal Muscle Fiber**

- sudden insurgence of sodium ions into the muscle fiber when the acetylcholine channels open causes the internal membrane potential in the local area of the end plate to increase in the positive direction as much as 50 to 75 millivolts, creating a local potential called the end plate potential.
- end plate potential created by the acetylcholine stimulation is normally far greater than enough to initiate an action potential in the muscle fiber.

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**HOW STRONG ARE YOU?**

You’d probably be amazed to learn how much force your body’s joints and muscles actually support on a daily basis. This interactive segment from Discovery takes you inside the body and explains how much strength your bones and muscles really possess. Other activities let you explore even more of your body's systems to see exactly how they move you through your daily life.
Events in a Skeletal Muscle Action Potential

1. Unlike neurons – the skeletal muscle has only one type voltage dependent sodium gate. When threshold voltage is reached it quickly opens and sodium rushes in causing the depolarization.

2. The sodium voltage dependent gate later closes and depolarization stops.

3. The same voltage (Threshold voltage) that causes the opening of the Na gates is the same Voltage that opens the potassium gates but they are slower - opening about the same time that the Na gates are Closing – K rushes out causing Repolarization

- Na/K pump and larger molecular anions

Return membrane to Resting State (RMP)
2. NERVE + MUSCLES
WORK ELECTRO-
MAGNETIC-STATICALLY

2.1 INTRODUCTION In this chapter we consider the structure of nerve and muscle tissue and in particular their membranes, which are excitable. A qualitative description of the activation process follows. Many new terms and concepts are mentioned only briefly in this chapter but in more detail in the next two chapters, where the same material is dealt with from a quantitative rather than a qualitative point of view.

The first documented reference to the nervous system is found in ancient Egyptian records. The Edwin Smith Surgical Papyrus, a copy (dated 1700 B.C.) of a manuscript composed about 3500 B.C., contains the first use of the word "brain", along with a description of the coverings of the brain which was likened to the film and corrugations that are seen on the surface of molten copper as it cooled (Elsberg, 1931; Kandel and Schwartz, 1985).

The basic unit of living tissue is the cell. Cells are specialized in their anatomy and physiology to perform different tasks. All cells exhibit a voltage difference across the cell membrane. Nerve cells and muscle cells are excitable. Their cell membrane can produce electrochemical impulses and conduct them along the membrane. In muscle cells, this electric phenomenon is also associated with the contraction of the cell. In other cells, such as gland cells and ciliated cells, it is believed that the membrane voltage is important to the execution of cell function.

The origin of the membrane voltage is the same in nerve cells as in muscle cells. In both cell types, the membrane generates an impulse as a consequence of
excitation. This impulse propagates in both cell types in the same manner. What follows is a short introduction to the anatomy and physiology of nerve cells. The reader can find more detailed information about these questions in other sources such as Berne and Levy (1988), Ganong (1991), Guyton (1992), Patton et al. (1989) and Ruch and Patton (1982).

2.2 NERVE CELL

2.2.1 The Main Parts of the Nerve Cell

The nerve cell may be divided on the basis of its structure and function into three main parts:

(1) the cell body, also called the soma;

(2) numerous short processes of the soma, called the dendrites; and,

(3) the single long nerve fiber, the axon.

These are described in Figure 2.1.

The body of a nerve cell (see also (Schadé and Ford, 1973)) is similar to that of all other cells. The cell body generally includes the nucleus, mitochondria, endoplasmic reticulum, ribosomes, and other organelles. Since these are not unique to the nerve cell, they are not discussed further here. Nerve cells are about 70 - 80% water; the dry material is about 80% protein and 20% lipid. The cell volume varies between 600 and 70,000 µm³. (Schadé and Ford, 1973)

The short processes of the cell body, the dendrites, receive impulses from other cells and transfer them to the cell body (afferent signals). The effect of these impulses may be excitatory or inhibitory. A cortical neuron (shown in Figure 2.2) may receive impulses from tens or even hundreds of thousands of neurons (Nunez, 1981).

The long nerve fiber, the axon, transfers the signal from the cell body to another nerve or to a muscle cell. Mammalian axons are usually about 1 - 20 µm in diameter. Some axons in larger animals may be several meters in length. The axon may be covered with an insulating layer called the myelin sheath, which is formed
by *Schwann cells* (named for the German physiologist Theodor Schwann, 1810-1882, who first observed the myelin sheath in 1838). The myelin sheath is not continuous but divided into sections, separated at regular intervals by the *nodes of Ranvier* (named for the French anatomist Louis Antoine Ranvier, 1834-1922, who observed them in 1878).

**Fig. 2.1.** The major components of a neuron.

**Fig. 2.2.** Cortical nerve cell and nerve endings connected to it.
2.2.2 The Cell Membrane

The cell is enclosed by a cell membrane whose thickness is about 7.5 - 10.0 nm. Its structure and composition resemble a soap-bubble film (Thompson, 1985), since one of its major constituents, fatty acids, has that appearance. The fatty acids that constitute most of the cell membrane are called \textit{phosphoglycerides}. A phosphoglyceride consists of phosphoric acid and fatty acids called \textit{glycerides} (see Figure 2.3). The head of this molecule, the phosphoglyceride, is \textit{hydrophilic} (attracted to water). The fatty acids have tails consisting of hydrocarbon chains which are \textit{hydrophobic} (repelled by water).

If fatty acid molecules are placed in water, they form little clumps, with the acid heads that are attracted to water on the outside, and the hydrocarbon tails that are repelled by water on the inside. If these molecules are very carefully placed on a water surface, they orient themselves so that all acid heads are in the water and all hydrocarbon tails protrude from it. If another layer of molecules were added and more water put on top, the hydrocarbon tails would line up with those from the first layer, to form a double (two molecules thick) layer. The acid heads would protrude into the water on each side and the hydrocarbons would fill the space between. This bilayer is the basic structure of the cell membrane.

From the bioelectric viewpoint, the \textit{ionic channels} constitute an important part of the cell membrane. These are macromolecular pores through which sodium, potassium, and chloride ions flow through the membrane. The flow of these ions forms the basis of bioelectric phenomena. Figure 2.4 illustrates the construction of a cell membrane.
Fig. 2.3. A sketch illustrating how the phosphoglyceride (or phospholipid) molecules behave in water. See text for discussion.

Fig. 2.4. The construction of a cell membrane. The main constituents are two lipid layers, with the hydrophobic tails pointing inside the membrane (away from the aqueous intracellular and interstitial mediums). The macromolecular pores in the cell membrane form the ionic channels through which sodium, potassium, and
chloride molecules flow through the membrane and generate the bioelectric phenomena.

2.2.3 The Synapse

The junction between an axon and the next cell with which it communicates is called the synapse. Information proceeds from the cell body unidirectionally over the synapse, first along the axon and then across the synapse to the next nerve or muscle cell. The part of the synapse that is on the side of the axon is called the presynaptic terminal; that part on the side of the adjacent cell is called the postsynaptic terminal. Between these terminals, there exists a gap, the synaptic cleft, with a thickness of 10 - 50 nm. The fact that the impulse transfers across the synapse only in one direction, from the presynaptic terminal to the postsynaptic terminal, is due to the release of a chemical transmitter by the presynaptic cell. This transmitter, when released, activates the postsynaptic terminal, as shown in Figure 2.5. The synapse between a motor nerve and the muscle it innervates is called the neuromuscular junction. Information transfer in the synapse is discussed in more detail in Chapter 5.

![Fig. 2.5. Simplified illustration of the anatomy of the synapse.](image-url)
A) The synaptic vesicles contain a chemical transmitter.  
B) When the activation reaches the presynaptic terminal the transmitter is released and it diffuses across the synaptic cleft to activate the postsynaptic membrane.

2.3 MUSCLE CELL

There are three types of muscles in the body:

- smooth muscle,
- striated muscle (skeletal muscle), and
- cardiac muscle.

Smooth muscles are involuntary (i.e., they cannot be controlled voluntarily). Their cells have a variable length but are in the order of 0.1 mm. Smooth muscles exist, for example, in the digestive tract, in the wall of the trachea, uterus, and bladder. The contraction of smooth muscle is controlled from the brain through the autonomic nervous system.

Striated muscles, are also called skeletal muscles because of their anatomical location, are formed from a large number of muscle fibers, that range in length from 1 to 40 mm and in diameter from 0.01 to 0.1 mm. Each fiber forms a (muscle) cell and is distinguished by the presence of alternating dark and light bands. This is the origin of the description "striated," as an alternate terminology of skeletal muscle (see Figure 2.6).

The striated muscle fiber corresponds to an (unmyelinated) nerve fiber but is distinguished electrophysiologically from nerve by the presence of a periodic transverse tubular system (TTS), a complex structure that, in effect, continues the surface membrane into the interior of the muscle. Propagation of the impulse over the surface membrane continues radially into the fiber via the TTS, and forms the trigger of myofibrillar contraction. The presence of the TTS affects conduction of the muscle fiber so that it differs (although only slightly) from propagation on an (unmyelinated) nerve fiber. Striated muscles are connected to the bones via tendons. Such muscles are voluntary and form an essential part of the organ of support and motion.
Cardiac muscle is also striated, but differs in other ways from skeletal muscle: Not only is it involuntary, but also when excited, it generates a much longer electric impulse than does skeletal muscle, lasting about 300 ms. Correspondingly, the mechanical contraction also lasts longer. Furthermore, cardiac muscle has a special property: The electric activity of one muscle cell spreads to all other surrounding muscle cells, owing to an elaborate system of intercellular junctions.

Fig. 2.6. Anatomy of striated muscle. The fundamental physiological unit is the fiber.

2.4 BIOELECTRIC FUNCTION OF THE NERVE CELL

The membrane voltage (transmembrane voltage) \( V_m \) of an excitable cell is defined as the potential at the inner surface \( \Phi_i \) relative to that at the outer \( \Phi_o \) surface of the membrane, i.e. \( V_m = (\Phi_i) - (\Phi_o) \). This definition is independent of the cause of the potential, and whether the membrane voltage is constant, periodic, or nonperiodic in behavior. Fluctuations in the membrane potential may be classified according to their character in many different ways. Figure 2.7 shows the classification for nerve cells developed by Theodore Holmes Bullock (1959).
According to Bullock, these transmembrane potentials may be resolved into a resting potential and potential changes due to activity. The latter may be classified into three different types:

1. **Pacemaker potentials**: the intrinsic activity of the cell which occurs without external excitation.

2. **Transducer potentials** across the membrane, due to external events. These include **generator potentials** caused by receptors or **synaptic potential** changes arising at synapses. Both subtypes can be inhibitory or excitatory.

3. As a consequence of transducer potentials, further response will arise. If the magnitude does not exceed the threshold, the response will be **nonpropagating** (**electrotonic**). If the response is great enough, a **nerve impulse** (**action potential impulse**) will be produced which obeys the all-or-nothing law (see below) and proceeds unattenuated along the axon or fiber.

![Fig. 2.7. Transmembrane potentials according to Theodore H. Bullock.](image-url)
2.5 EXCITABILITY OF NERVE CELL

If a nerve cell is stimulated, the transmembrane voltage necessarily changes. The stimulation may be

excitatory (i.e., depolarizing; characterized by a change of the potential inside the cell relative to the outside in the positive direction, and hence by a decrease in the normally negative resting voltage) or inhibitory (i.e., hyperpolarizing, characterized by a change in the potential inside the cell relative to the outside in the negative direction, and hence by an increase in the magnitude of the membrane voltage).

After stimulation the membrane voltage returns to its original resting value.

If the membrane stimulus is insufficient to cause the transmembrane potential to reach the threshold, then the membrane will not activate. The response of the membrane to this kind of stimulus is essentially passive. Notable research on membrane behavior under subthreshold conditions has been performed by Lorente de Nó (1947) and Davis and Lorente de Nó (1947).

If the excitatory stimulus is strong enough, the transmembrane potential reaches the threshold, and the membrane produces a characteristic electric impulse, the nerve impulse. This potential response follows a characteristic form regardless of the strength of the transthreshold stimulus. It is said that the action impulse of an activated membrane follows an all-or-nothing law. An inhibitory stimulus increases the amount of concurrent excitatory stimulus necessary for achieving the threshold (see Figure 2.8). (The electric recording of the nerve impulse is called the action potential. If the nerve impulse is recorded magnetically, it may be called an action current. The terminology is further explicated in Section 2.8 and in Figure 2.11, below.)
2.6 THE GENERATION OF THE ACTIVATION

The mechanism of the activation is discussed in detail in Chapter 4 in connection with the Hodgkin-Huxley membrane model. Here the generation of the activation is discussed only in general terms.

The concentration of sodium ions (Na\(^+\)) is about 10 times higher outside the membrane than inside, whereas the concentration of the potassium (K\(^+\)) ions is about 30 times higher inside as compared to outside. When the membrane is stimulated so that the transmembrane potential rises about 20 mV and reaches the threshold - that is, when the membrane voltage changes from -70 mV to about -50 mV (these are illustrative and common numerical values) - the sodium and potassium ionic permeabilities of the membrane change. The sodium ion...
permeability increases very rapidly at first, allowing sodium ions to flow from outside to inside, making the inside more positive. The inside reaches a potential of about +20 mV. After that, the more slowly increasing potassium ion permeability allows potassium ions to flow from inside to outside, thus returning the intracellular potential to its resting value. The maximum excursion of the membrane voltage during activation is about 100 mV; the duration of the nerve impulse is around 1 ms, as illustrated in Figure 2.9. While at rest, following activation, the Na-K pump restores the ion concentrations inside and outside the membrane to their original values.

\[ V_m [\text{mV}] \]

\[ 0 \]

\[ -50 \]

\[ -100 \]

\[ 0 \quad 0.5 \quad 1.0 \quad \text{Time [ms]} \]

**Fig. 2.9.** Nerve impulse recorded from a cat motoneuron following a transthreshold stimulus. The stimulus artifact may be seen at \( t = 0 \).

### 2.7 CONCEPTS ASSOCIATED WITH THE ACTIVATION PROCESS

Some basic concepts associated with the activation process are briefly defined in this section. Whether an excitatory cell is activated depends largely on the strength and duration of the stimulus. The membrane potential may reach the
threshold by a short, strong stimulus or a longer, weaker stimulus. The curve illustrating this dependence is called the strength-duration curve; a typical relationship between these variables is illustrated in Figure 2.10. The smallest current adequate to initiate activation is called the rheobasic current or rheobase. Theoretically, the rheobasic current needs an infinite duration to trigger activation. The time needed to excite the cell with twice rheobase current is called chronaxy.

Accommodation and habituation denote the adaptation of the cell to a continuing or repetitive stimulus. This is characterized by a rise in the excitation threshold. Facilitation denotes an increase in the excitability of the cell; correspondingly, there is a decrease in the threshold. Latency denotes the delay between two events. In the present context, it refers to the time between application of a stimulus pulse and the beginning of the activation. Once activation has been initiated, the membrane is insensitive to new stimuli, no matter how large the magnitude. This phase is called the absolute refractory period. Near the end of the activation impulse, the cell may be activated, but only with a stimulus stronger than normal. This phase is called the relative refractory period.

The activation process encompasses certain specifics such as currents, potentials, conductivities, concentrations, ion flows, and so on. The term action impulse describes the whole process. When activation occurs in a nerve cell, it is called a nerve impulse; correspondingly, in a muscle cell, it is called a muscle impulse. The bioelectric measurements focus on the electric potential difference across the membrane; thus the electric measurement of the action impulse is called the action potential that describes the behavior of the membrane potential during the activation. Consequently, we speak, for instance, of excitatory postsynaptic potentials (EPSP) and inhibitory postsynaptic potentials (IPSP). In biomagnetic measurements, it is the electric current that is the source of the magnetic field. Therefore, it is logical to use the term action current to refer to the source of the biomagnetic signal during the action impulse. These terms are further illustrated in Figure 2.11.
Fig. 2.10. (A) The response of the membrane to various stimuli of changing strength (B), the strength-duration curve. The level of current strength which will just elicit activation after a very long stimulus is called rheobase. The minimum time required for a stimulus pulse twice the rheobase in strength to trigger
activation is called chronaxy. (For simplicity, here, threshold is shown to be independent on stimulus duration.)

Fig. 2.11. Clarification of the terminology used in connection with the action impulse:
A) The source of the action impulse may be nerve or muscle cell. Correspondingly it is called a nerve impulse or a muscle impulse.
B) The electric quantity measured from the action impulse may be potential or current. Correspondingly the recording is called an action potential or an action current.

2.8 CONDUCTION OF THE NERVE IMPULSE IN AN AXON

Ludvig Hermann (1872, 1905) correctly proposed that the activation propagates in an axon as an unattenuated nerve impulse. He suggested that the potential difference between excited and unexcited regions of an axon would cause small currents, now called local circuit currents, to flow between them in such a direction that they stimulate the unexcited region.

Although excitatory inputs may be seen in the dendrites and/or soma, activation originates normally only in the soma. Activation in the form of the nerve impulse (action potential) is first seen in the root of the axon - the initial segment of the axon, often called the axon hillock. From there it propagates
along the axon. If excitation is initiated artificially somewhere along the axon, propagation then takes place in both directions from the stimulus site. The conduction velocity depends on the electric properties and the geometry of the axon.

An important physical property of the membrane is the change in sodium conductance due to activation. The higher the maximum value achieved by the sodium conductance, the higher the maximum value of the sodium ion current and the higher the rate of change in the membrane voltage. The result is a higher gradient of voltage, increased local currents, faster excitation, and increased conduction velocity. The decrease in the threshold potential facilitates the triggering of the activation process.

The capacitance of the membrane per unit length determines the amount of charge required to achieve a certain potential and therefore affects the time needed to reach the threshold. Large capacitance values, with other parameters remaining the same, mean a slower conduction velocity.

The velocity also depends on the resistivity of the medium inside and outside the membrane since these also affect the depolarization time constant. The smaller the resistance, the smaller the time constant and the faster the conduction velocity. The temperature greatly affects the time constant of the sodium conductance; a decrease in temperature decreases the conduction velocity.

The above effects are reflected in an expression derived by Muler and Markin (1978) using an idealized nonlinear ionic current function. For the velocity of the propagating nerve impulse in unmyelinated axon, they obtained

$$v = \sqrt{\frac{i_{Na\ max}}{\sigma C \frac{2}{\rho R} V_{th}}}$$

(2.1)

where

$v$ = velocity of the nerve impulse [m/s]

$i_{Na\ max}$ = maximum sodium current per unit length [A/m]

$V_{th}$ = threshold voltage [V]
A myelinated axon (surrounded by the myelin sheath) can produce a nerve impulse only at the nodes of Ranvier. In these axons the nerve impulse propagates from one node to another, as illustrated in Figure 2.12. Such a propagation is called *saltatory conduction* (*saltare*, "to dance" in Latin).

The membrane capacitance per unit length of a myelinated axon is much smaller than in an unmyelinated axon. Therefore, the myelin sheath increases the conduction velocity. The resistance of the axoplasm per unit length is inversely proportional to the cross-sectional area of the axon and thus to the square of the diameter. The membrane capacitance per unit length is directly proportional to the diameter. Because the time constant formed from the product controls the nodal transmembrane potential, it is reasonable to suppose that the velocity would be inversely proportional to the time constant. On this basis the conduction velocity of the myelinated axon should be directly proportional to the diameter of the axon. This is confirmed in Figure 2.13, which shows the conduction velocity in mammalian myelinated axons as linearly dependent on the diameter. The conduction velocity in myelinated axon has the approximate value shown:

\[
v = 6d
\]

where \(v\) = velocity [m/s]

\(d\) = axon diameter [\(\mu\)m]
Fig. 2.12. Conduction of a nerve impulse in a nerve axon. (A) continuous conduction in an unmyelinated axon; (B) saltatory conduction in a myelinated axon.

Fig. 2.13. Experimentally determined conduction velocity of a nerve impulse in a mammalian myelinated axon as a function of the diameter. (Adapted from Ruch and Patton, 1982.)
REFERENCES

Magnetic field of a single muscle fiber. First measurements and a core conductor model.

J M van Egeraat, R N Friedman, and J P Wikswo, Jr
Abstract

We present the first measurements of the magnetic field from a single muscle fiber of the frog gastrocnemius, obtained by using a toroidal pickup coil coupled to a room-temperature, low-noise amplifier. The axial currents associated with the magnetic fields of single fibers were biphasic and had peak-to-peak amplitudes ranging between 50 and 100 nA, depending primarily on the fiber radius. With an intracellular microelectrode, we measured the action potential of the same fiber, which allowed us to determine that the intracellular conductivity of the muscle fiber in the core conductor approximation was 0.20 +/- 0.09 S/m. Similarly, we found that the effective membrane capacitance was 0.030 +/- 0.011 F/m2. These results were not significantly affected by the anisotropic conductivity of the muscle bundle. We demonstrate how our magnetic technique can be used to determine the transmembrane action potential without penetrating the membrane with a microelectrode, thereby offering a reliable, stable, and atraumatic method for studying contracting muscle fibers.

Full text

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brief communication

Magnetic field of a single muscle fiber
First measurements and a core conductor model

J. M. van Egertat, R. N. Friedman, and J. P. Wikawo, Jr.
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ABSTRACT We present the first measurements of the magnetic field from a single muscle fiber of the frog gastrocnemius, obtained by using a toroidal pickup coil coupled to a room-temperature, low-noise amplifier. The axial currents associated with magnetic fields of single fibers were biphasic and had peak-to-peak amplitudes ranging between 50 and 100 nA, depending primarily on the fiber radius. With an intracellular microelectrode, we measured the action potential of the same fiber, which allowed us to determine that the intracellular conductivity of the muscle fiber in the core conductor approximation was \(0.20 \pm 0.09\) S/m. Similarly, we found that the effective membrane capacitance was \(0.030 \pm 0.011\) F/m². These results were not significantly affected by the anisotropic conductivity of the muscle bundle. We demonstrate how our magnetic technique can be used to determine the transmembrane action potential without penetrating the membrane with a microelectrode, thereby offering a reliable, stable, and atraumatic method for studying contracting muscle fibers.

INTRODUCTION

For a deeper understanding of the electric and magnetic fields produced by biological tissue, it is important to characterize the behavior of a single cell. Almost a decade ago, we reported the first measurements of the magnetic field of a nerve impulse (1). In this paper we present the first recordings of the magnetic field produced by action currents propagating in a single skeletal muscle fiber.

Electrical activity of biological tissue can be detected by a limited number of methods: electrical recording of action potentials, optical methods (e.g., birefringence, voltage sensitive dyes), or magnetic recording of action currents. The first method has the advantage of a relatively high signal-to-noise ratio for typical biological signals, but requires the penetration of the cell with a microelectrode. The optical methods, while avoiding the adverse effects of penetration, have other limitations such as an uncertain origin of the signal (2) and long-term instability (photobleaching, dye washout (3)). In magnetic recording, the signal-to-noise ratio is usually not as good as that which is attainable with electrical measurements using intracellular microelectrodes; but, this disadvantage can be overcome by signal averaging. The magnetic technique is proving to be especially useful in situations where electric recordings are difficult or impossible, such as measurements of the axial variation of the membrane potential near the site of nerve crush injury, requiring multiple electrode penetrations (manuscript in preparation), in vivo recordings of action signals in human nerves (4), or studies of single muscle fibers whose contractions preclude stable microelectrode implantation.

Combined electric and magnetic measurements can be used to obtain information about the intracellular conductivity (\(\sigma\)) and the membrane capacitance (\(C_m\)) of the fiber. Alternatively, the magnetic recordings can be combined with known values of \(\sigma\) and \(C_m\) to determine the membrane potential without microelectrodes (5, 6).

Recordings from single muscle fibers form a necessary element in the analysis of compound action signals from bundles of simultaneously firing fibers. Wijesinghe (7) presents a model to decompose a magnetically recorded compound action current into the contributions of the individual fibers. Important parameters in such models are the intracellular conductivity and the shapes and magnitudes of the single fiber action currents. Once these data are known, it may be possible to use totally noninvasive, in vivo measurements with high-resolution SQUID magnetometers (8) to distinguish between motor units and to obtain data about the fiber composition of each motor unit, without the need for invasive, electrical measurements.

MATERIALS AND METHODS

In our experiments, we used the gastrocnemius muscle of the bullfrog (Rana catesbiana). The muscle was dissected free and submerged in frog Ringer's solution at 21°C. The connective tissue layers around the muscle were opened and a small bundle of 20–40 muscle fibers was carefully dissected from the muscle. The bundle was at least 20 mm
in length. One side of the bundle remained attached to the muscle and the other side was threaded through two toroidal pickup cores (9) connected to a pair of low-noise, current-to-voltage amplifiers (10, 11). The bundle was stretched to approximate its original length and fixed in the bath. The ferrite cores for detecting the magnetic field (type OW 40502; Magnetics, Butler, PA) each had inner and outer radii of 1.08 and 1.95 mm, respectively, and a width of 1.25 mm. The core was wound with 66 turns of insulated copper wire (40 gauge) and a single turn calibration winding. A thin layer of epoxy insulated the core from the bath. We used a microcomputer-based data acquisition system to average 512 recordings of the magnetic signal, thereby improving the signal-to-noise ratio by more than a factor of 20.

Electrical recordings were made with a micropipette, filled with 3 M KCl, coupled to an electrometer (WPI S-7071A; World Precision Instruments Inc., New Haven, CT). Typically, this technique would allow no more than 10 consecutive recordings of an action potential, because the contraction of the fiber would either eject the micropipette or damage the membrane seal around the pipette tip. Therefore, the electrical recordings were made only after recording the magnetic data. The magnetic recording is much less liable to motion artifacts, as there is no physical contact between the probe and the contracting fiber. Also, the magnetic recording will not be distorted by small axial displacements of the fiber relative to the probe, because the spatial extent of the action potential is at least one order of magnitude greater than the contractile movement of the fiber.

Although the toroid was threaded by a bundle, we measured the signal of only one fiber by selective stimulation. A single fiber was partly separated from the bundle so that it could be stimulated extracellularly, without firing any of the other fibers. Threshold was reached with a 60-μ, 0.7-mA stimulus pulse. A precise determination of the conduction velocity of the action potential was made by measuring the propagation delay between the two toroids of known separation.

We used a core conductor model to analyze our data (5). Ignoring volume conductor effects is valid as long as the effective radius (r_eff) of the toroid is smaller than the spatial length of the rising edge of the action potential (12). In our experiments, the length of the depolarization phase of the action potential was always >2.5 mm, whereas r_eff was 1.47 mm, as determined by the toroid dimensions (9).

In the core conductor approximation, the relationship between the axial current (I_a), measured with the toroid, and the intracellularly-recorded membrane potential (V_m) can be expressed as

\[ I_a(t) = \frac{1}{R}(dV_m/dx) = \frac{u}{R}(dV_m/dx), \]  

where \( R \) is the resistance per unit length (Ω/m) of the fiber, \( u \) is the conduction velocity (m/s) and \( x \) is the axial coordinate. If \( u \) is known, \( R \) can be determined from the measured action potential and current. If we know \( R \) and \( u \) beforehand, it is possible to calculate \( V_m \) from \( I_a \).

Eq. 1 assumes that we measure \( I_a \) at a single location along the fiber. In reality, however, the toroid is measuring the axial current, averaged over the width of the toroid. As a consequence, the measured signal is slightly broadened and has a lower amplitude than the actual \( I_a \) in the muscle fiber. The toroid acts as a moving average filter with a corresponding low-pass filter characteristic. The z-transform of this filter as a function of the complex variable \( z \) is given by (13)

\[ H(z) = (1/N) \sum_{k=-\infty}^{\infty} z^{-k} = (1/N)(1 - z^{-N})/(1 - z^{-1}). \]  

(2)

\( N \) is an integer that determines the length of the summation, and is related to the width \( (d) \) of the toroid, the conduction velocity, \( u \), and the sampling interval \( (t_s) \) by

\[ N = d/(ut_s). \]  

(3)

In our case, \( t_s \) was 0.15 ms and \( u \) was 2.0 m/s, which leads to \( N \approx 4 \). Given this, we can correct for the systematic error introduced by the toroid by inverse filtering with a deconvolution filter \( H^{-1}(z) \)

\[ H^{-1}(z) = \frac{(1 - z^{-N})}{(1 - z^{-1})}. \]  

(4)

\( N - 1 \) leads to no correction because \( H^{-1}(z) \) is equal to unity in that case.

The filter \( H^{-1}(z) \) can reconstruct any signal that is distorted by the moving average filtering of the toroid, provided that the signal is free of noise. If this is not the case, the filter may resonate with a fundamental frequency \( 1/(NT) \). We suppressed this resonance in two ways: first, we smoothed the measured signal by replacing it by a fourth order B-spline with 12 control points (14). The B-spline was fitted to the measured signal with a least squares algorithm and then deconvoluted with the filter given by Eq. 4. As expected, the resulting signal contained some resonance artifacts, evoked by the steep transition between the first and second phase of the magnetic signal.

Therefore, the second step of resonance suppression consisted of a simple subtraction of a phase-shifted segment of the tail of the action current waveform (resonating around \( I_a = 0 \)) from the second phase of the action potential that contained the same resonance.

Finally, the deconvoluted spline function, which represents \( I_a \), was integrated to obtain the membrane potential. In this way, it was possible to reconstruct the action potential from the measured action current. Even if the second step of resonance suppression is omitted, the same
results is obtained, because of the smoothing effect of the integration.

RESULTS

Fig. 1 shows the recorded single fiber action current (a) and action potential (b) as solid lines. The action potential does not immediately return to the initial resting potential. This may be due to a long depolarizing afterpotential in the frog muscle (15), changes in the electrode impedance, or other electrode artifacts following the contraction. The prolonged afterpotential was not consistently observed in all fibers. The control points of the B-spline that was fitted to the action current are given in Table 1. A plot of the spline function was barely distinguishable from the recorded signal and was omitted from Fig. 1 a. The dotted line in Fig. 1 a is the deconvoluted action current. The measured current is slightly wider and lower in amplitude than the deconvoluted signal because of the spatial averaging effect of the toroid.

The conduction velocity, $u$, was measured to be 2.0 ± 0.1 m/s. The measured action potential had a maximum slope of 92 V/s, occurring at 0.55 ms before the peak of the action potential. At this time, the deconvoluted action current also reached its maximum of 71 nA. We estimate the error in both the current and the time derivative of the voltage to be 10% or less. With these data, the resistance per unit length, $R$, was found to be 0.65 ± 0.18 GΩ/m. An extensive treatment of all sources of error is given in reference 12.

Based on these numbers, we could perform the integration to calculate the single muscle fiber action potential, shown as the dotted line in Fig. 1 b. Except for the tail of the intracellularly recorded action potential, the measured and reconstructed data match very well. The prolonged afterpotential may be physiological but undetected by the toroid as a result of the high-pass filter characteristics of a current transformer. Alternatively, the afterpotential may be an electrode artifact, as mentioned earlier.

The membrane capacity, $C_m$, per unit fiber length can be calculated from $V_m$ and $I_m$, with the assumption that the radial membrane conduction current is negligible during the subthreshold phase of the action potential (5). We make the rough approximation that the capacity of the T-tubular system is included in the membrane capacity, or also that the so called access resistance of the T-tubular system is equal to zero (16–19). Valdiosera (19) found time constants for the T-tubular system in the range of 0.5–1.0 ms, which means that the approximation is acceptable for the relatively slow subthreshold phase of the action potential. The capacitive membrane current ($J_c$) per unit fiber length is given by

$$J_c = -C_m \frac{dV_m}{dt}.$$  

Continuity of current requires that

$$J_c = (1/a) \frac{dI_m}{dt}.$$  

These two equations can be solved for $C_m$. At 1.4 ms before the peak of the action potential, the rates of change

<table>
<thead>
<tr>
<th>Control point no.</th>
<th>Time</th>
<th>Action current</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ms</td>
<td>nA</td>
</tr>
<tr>
<td>1</td>
<td>1.000</td>
<td>0.6</td>
</tr>
<tr>
<td>2</td>
<td>2.250</td>
<td>-6.7</td>
</tr>
<tr>
<td>3</td>
<td>3.000</td>
<td>23.9</td>
</tr>
<tr>
<td>4</td>
<td>3.375</td>
<td>46.9</td>
</tr>
<tr>
<td>5</td>
<td>3.750</td>
<td>77.7</td>
</tr>
<tr>
<td>6</td>
<td>4.125</td>
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</tr>
<tr>
<td>10</td>
<td>6.000</td>
<td>-37.6</td>
</tr>
<tr>
<td>11</td>
<td>7.500</td>
<td>4.3</td>
</tr>
<tr>
<td>12</td>
<td>10.000</td>
<td>-0.1</td>
</tr>
</tbody>
</table>

For a reconstruction of the B-spline, see reference 12.
of $V_0$ and $I$, are $3.4 \pm 0.3 \text{ V/s}$ and $64 \pm 6 \text{ \mu A/s}$, respectively. Using the value for $u$ as above, we found that membrane capacity per unit fiber length, $C_m$, was $9.4 \pm 2.4 \text{ \mu F/m}$.  

The fiber radius, $a$, was found by optical microscopy to be $50 \pm 10 \text{ \mu m}$. Now we can convert $C_m$ to the specific membrane capacitance $c_m (\text{F/m}^2)$ by dividing by $2\pi a$. Also, we can convert $R$ to the specific axial resistivity $r$ (Ohm) by multiplying by $\pi a^2$. For $c_m$, we find $0.030 \pm 0.011 \text{ F/m}^2$ and for $r$, $5.1 \pm 2.3 \Omega \text{m}$. The corresponding axial conductivity $\sigma$ is the reciprocal of $r$ and is $0.20 \pm 0.09 \text{ S/m}$.

Finally, we checked the validity of the core conductor approximation. The results can be affected by the finite dimensions of the toroid and by the fact that the active fiber is surrounded by an anisotropic bundle of inactive fibers instead of by a grounded, zero-resistance bath. We corrected for the finite toroid size by deconvoluting the action current. To take into account the effects of the anisotropic conductivity, we used the volume conductor model presented by Roth and Wikswo (6). This model calculates the magnetic field produced by a given axial current in the presence of an anisotropic medium around the fiber. Using a four-electrode method (20), we determined that the transverse conductivity of the bundle was approximately one half the longitudinal conductivity. From the volume conductor model, it followed that the anisotropy reduced the magnetic field at $r_0$ by <15%.

This error is less than the uncertainty in the other parameters and is ignored in this analysis, although it would be possible to correct for this systematic error.

**DISCUSSION**

Fig. 1 shows that it is possible to use a toroidal coil and a low-noise amplifier to record 50 nA action currents from a single muscle fiber. After correcting for the toroid effects, it is possible to accurately reconstruct the action potential from the measured action current. It is conceivable that a thinner toroid of higher permeability would eliminate the need for the deconvolution of the action current (9).

The intracellular conductivity of $0.20 \pm 0.09 \text{ S/m}$, calculated from our magnetic and electric measurements, is significantly lower than that of nerve fibers. Proposing a structure or mechanism accounting for this difference would be speculative at this time; however, our results do not differ markedly from the findings of other investigators (15, 19, 21). The largest source of uncertainty is the fiber radius. Our value for the fiber resistance per unit length ($0.65 \pm 0.18 \text{ G\Omega/m}$) is within the range of error of the value $0.46 \pm 0.07 \text{ G\Omega/m}$, found by Valdiosera et al. (19). Valdiosera did not measure the fiber radius inde-

pendently, so that a comparison of the intracellular conductivity cannot be made. Adrian and Peachey (15) and Hodgkin and Nakajima (21) used higher values ($0.25-0.60 \text{ S/m}$) for muscle fiber intracellular conductivity than the value found in our studies. Based on our earlier analysis of a single nerve axon (12), the combined electric/magnetic measurement of the conductivity may have less than half the error of values derived from cable-constant measurements made with multiple micro-electrode penetrations. Our value for the membrane capacitance is in agreement with values from experiments reported by others (18, 19). The fiber radius is again the largest source of error.

Recordings of the magnetic signature of a single muscle fiber, such as presented here, will form the basis for future analyses of magnetically recorded signals from fiber bundles. A decomposition of compound action currents into the contributions of the individual fibers is only possible if we know some of the basic characteristics of the single fiber.

With new, high-resolution SQUID magnetometers (4, 22), it is now feasible to analyze the magnetic signals from single motor units in vivo without the need for invasive measurements with needle electrodes. This may provide the clinician with data on the number and size of fibers in a motor unit or muscle. Furthermore, the values for the intracellular conductivity and membrane capacitance are important parameters in the study of muscle plasticity and in numerical models that simulate the propagation of action potentials. Such studies and models should improve our ability to interpret clinically measured magnetic signals and advance their use in diagnosis.

After correcting for toroid effects, it is possible to accurately reconstruct the action potential from the measured action current, eliminating the need for penetration of the membrane and thereby offering a reliable, stable, and atraumatic method for studying contracting muscle fibers.

We wish to thank R. S. Wiesenfeld for his assistance in the anisotropic bundle calculations.

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These references are in PubMed. This may not be the complete list of references from this article.

Diaphragm compound muscle action potential measured with magnetic stimulation and chest wall surface electrodes

Y.M Luo, N Mustfa, R.A Lyall, W.D.C Man, J.C Glérant, M.I Polkey, J Moxham

Abstract

To seek a method to reliably measure phrenic nerve conduction time (PNCT) with magnetic stimulation we investigated two stimulus sites, placing the magnetic coil at the cricoid cartilage (high position) or close to the clavicle (low position). We also compared compound muscle action potential (CMAP) recorded from three different sites: in the sixth to eighth intercostal spaces in the anterior axillary line (Ant-a); in the 8th intercostal space close to the midclavicular line; and with one electrode at the lower sternum and the other at the costal margin. Fourteen normal subjects were studied. The PNCT measured by magnetic stimulation in the high position recorded from (Ant-a) was 7.6±0.6 on the left side and 8.4±0.7 on the right. The PNCT recorded from all three sites become much shorter when the magnetic coil was moved from the high to the low position. Our results show that PNCT can be accurately measured with magnetic stimulation when care is taken to avoid coactivation of the brachial plexus.
Keywords

- Mammals, humans;
- Methods, nerve conduction time, magnetic stimulation;
- Muscle, diaphragm, compound action potential;
- Nerve, phrenic, conduction time


Standardization of facilitation of compound muscle action potentials evoked by magnetic stimulation of the cortex. Results in healthy volunteers and in patients with multiple sclerosis.

Ravnborg M¹, Blinkenberg M, Dahl K.

Author information

Abstract

To establish the importance of standardization of the facilitation of central motor conduction measured by magnetic stimulation we studied the effect of increasing voluntary muscle contraction on the central motor conduction time (CMCT) and motor evoked potential (MEP) amplitudes for 3 upper and 2 lower limb muscles. MEPs were elicited by magnetic stimulation of the cortex and the spinal roots. Muscle force was indirectly assessed from the integrated electrical muscle activity and expressed as the root mean square (RMS) and was varied from 0 to 40% of maximal activity. The central motor conduction time (CMCT) decreased during increasing muscle contraction, reaching constant values at approximately 10-20% RMSmax. Similarly, the increases of MEP amplitude tapered off at about the same RMS level. For each muscle an optimal RMS level was defined. The shortening of the CMCTs at the optimal RMS levels were: the brachial biceps, 3.4 msec; the radial carpal flexor of the wrist, 2.7 msec; the first dorsal interosseus muscle of the hand, 2.9 msec; the anterior tibial, 4.2 msec; and the abductor hallucis, 2.4 msec. The standardizing procedure was applied to 10 patients with multiple sclerosis. The stimulus thresholds were higher in these patients compared with those of the normals. Only the CMCT reduction of the BB was significantly larger (8.1 msec) than in the controls. Using standardized facilitation the diagnostic value of the amplitudes seems to be only a little less than that of the CMCTs.

PMID:

1710968

[PubMed - indexed for MEDLINE]
The Gentle Magnetic Stimulation of the Chakra Gradually Penetrates and Permeates through the Waters of the Body Till the Body Magnetically Vibrates and Resonates with the Music to Stimulate Health and Vitality.

Water Makes the Human Body Have Magnetic Actions

**Water is a Para-Magnetic Substance**

A Constant Stimulation of a Pulsing Magnetic Field will slowly cause a resonating cascade effect that will allow the Magnetic Field to slowly gather strength and expand and permeate into the body. The CyberMagnetic music will expand throughout the body. Thus the music will slowly permeate all of the cells.
"Every Little Bit of Help is Valuable"
"The Cybermagnetic Chair was a lot more than a bit"

Cybermagnetics Can Open Close Epigenetic Genes

The Future of Genetics is Cybermagnetics

Water molecule implies dialectical interface

Applied Magnetic Field implies oriented spin control

$\uparrow \downarrow = \text{magnetic moment of proton, spin dependent}$

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Cybermagnetics Unlocks Epigenes
Cybermagnetic

Using the computers headphone and microphone jacks we can first analyze the patient’s voice patterns for energetic disturbance and then chose sound files for relaxation, healing or energy. The music is sent into the body thru the headphones and a magnetic field generator. A magnetic field detector then receives the signals from the body establishing a cybermagnetic loop. The computer can then change the music to help the patient’s body electric.

The Cybermagnetic Chair can be purchased with the zero gravity chair you see for 1200 extra, or with the simple back cybermagnetic pads to put on your own chair for 5,000 euro with the QT software included. This sytem can operate indedendently or inter face with your QXCI, SCIO, Indigo or Eductor.

The Revolution in Energetic Medicine Continues

Cybermagnetic

" relating to the cybernetic loop control process of magnetic vibration stimulation and bio-magnetic field measuring resulting in autofocusing and perfecting the non-verbal body electric bio-magnetic field"
Just Imagine Floating on a Zero Gravity Chair. the Waters in your Body Magnetically Start to Align + all Your Cells Vibrate to the MUSIC.

If You Have Never had the Experience of Magnetic Music Expanding and Cascading through your Cells, Try it for an Hour.

Sound Vibration is converted to Magnetic Energy and is Sent into the Heart Chakra.

The Energy Penetrates into the Whole Body Thru the Magnetic Waters of the Body.

Then We Measure the Magnetic Energy at the Stomach Chakra and use a CyberMagNetic Loop to AutoFocus the Therapy.
Cybermagnetic $6,000

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DESIRE DUBOUNET: "THE MAN WHO LEFT AMERICA TO FIND FREEDOM WHO IS NO LONGER A MAN"

THE MIND CAN DO ANYTHING...

...IT JUST TAKES TIME

Pharmacology Fact: To Use a SYNTHETIC anything is an Insult to the Body

Sexual Identity is from What is between your ears Not What is between your legs

"What we Know about Biology Fills a Library, What Nature Knows Fills a World, Maybe a Universe"

Desire Dubounet

There is No Such Thing as Noise, It is Only Information We Do Not Understand

Desire Dubounet

The Cybermagnetic Chair uses the Headphone and Microphone Jack for inputs and outputs

So it blends in perfectly with your SCIO, INDIGO, or Energy to add a New Exciting Dimension to your Therapy

Magnetic measures of reaction into the Computer (in Red)

Cybermagnetic autofocusing of the Body Magnetic and Electric

Sound and Magnetic Vibration into the body (in Black)

"Every Little Bit of Help is Valuable"

"The Cybermagnetic Chair was a lot more than a bit"

IMUNE

International Medical University for Natural Education

Evidence Based Natural Energetic Medicine Education

www.imune.net