Electrodiagnostics; Chronaxymetry

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Introduction

Electrodiagnostics is a part of medicine encompassing methods of studying nerve and muscle stimulation with the use of direct and impulse currents.

Skeletal striated muscles are attached to bones. They have the ability to contract under the influence of stimuli from the central nervous system (impulses from the brain or the spinal cord). That makes performance of mechanical work possible. Skeletal muscles are stimulated by motor fibers of spinal nerves. Location where the nerve enters the muscle is called the *motor endplate*. One nerve fiber from a motor neuron of the anterior horn of the spinal cord ends on several muscle cells. Acetylcholine works as a synaptic transmitter of the end-plate. The receptor of this transmitter is found in the membrane of the muscle cell. Functionally, motor neuron and muscle fibers it innervates create the so-called motor unit. It is the basic functional unit of muscle contraction.

The state of the musculoskeletal system can be assessed qualitatively and quantitatively.

Qualitative methods – the observation of the strength of the muscle contraction in a response to a given electric impulse.

Quantitative methods – determining values of the physical quantities which constitute measures of the muscle stimulation.

On the basis of obtained results it is possible to establish the type of injury (neurogenic, myogenic or psychogenic changes), its localization (centers, neural pathways or muscle), the degree of injury (partial or total denervation), and also to make a prognosis concerning the further treatment and to control the course of recovery.

Depending on the histological process found in examined nerves, the *neuropathy*[1] can be divided into: axonal neuropathy, where the damage of axon occurs (*axonotmesis*), and demyelinating neuropathy, where mainly myelin is damaged (*neurapraxia*). However, in most cases the neuropathy has a mixed character where damaged myelin coincides with the damage of the axon.

The *neurapraxia* (conduction block) (Fig.1) is a temporary blockage of the nerve conduction due to compression or damage of the myelin sheath of the nerve fiber. Above and below the block, the conduction is unimpeded, however stimulating the motor fiber with a proper electric impulse above the block will not cause the muscle contraction. The more nerves are affected by neurapraxia, the less active the nerves will be during stimulation. The greater damage to the nerve the more intense current is needed to activated it. Neurapraxia can be caused, for example, by temporary ischemia of certain part of the nerve due to improper position during sleep and compression of the extremity, injuries or hematomas. Excessive cooling can also result in the blockage of nerve conduction at any given location. Myelin sheath can be damaged by toxins (e.g. of bacterial origin) or ingested poisons. Depending on the type of myelin sheath damage, conduction block can cease after a few minutes (compression of extremity during sleep), weeks or even months.

[1] *neuropathy – is a clinical syndrome caused by functional and structural changes in the peripheral and/or cranial nerves brought about by non-infectious agents*

A more serious damage is the disruption of nerve cell axon or primary axon injury – *axonotmesis*[2], (Fig. 1) leading to a total disruption of conduction. The nerve malfunction is potentially, if at all, restored after full regeneration of the axon. Distal end of the disrupted axon, along with myelin sheath separated from the nerve cell keeps the ability to conduct impulses for approximately 3-4 days, and then in a couple of days undergoes *Wallerian degeneration*[3], becomes inactive and finally ceases to conduct impulses entirely.

The regeneration of the axon is possible, but it is very slow process. On average, axon can extend 1-2 mm per day. This, however, depends on many factors, including the localization and the length of disrupted axon. It is also important whether or not the connective tissue sheath of nerve fibers and Schwann cells remained intact as they are able to create a canal for regenerating and extending axons. If all fibers of a muscle are enervated the muscle only reacts with contraction to direct stimulation with electric current 10 to 20 times higher than that need to cause the contraction of the same muscle on the opposite side of the body. If there are no signs of reinnervation after 6-8 weeks of the injury, the surgery has to be performed to establish whether total disruption of the nerve has occurred.

In the case of total disruption of the axon and its sheaths (*neurotmesis*), the consequences are serious and the prognosis is poor. In situations like this surgery is necessary, because the distal section of the disrupted nerve fiber degenerates completely and very fast, and the lack of "pathways" from the connective tissue kept in axonotmesis inhibits or even precludes the regeneration of the axon.

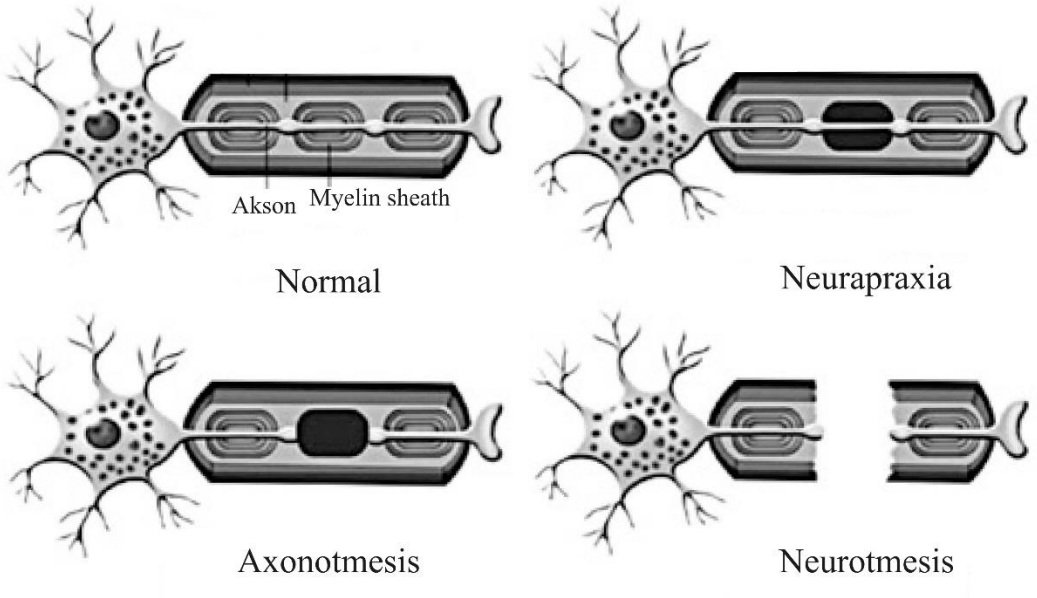

Fig. 1. Schematic drawing of a normal nerve fiber and the three grades of nerve injury according to Seddon's classification.

[2] *axonotmesis - nerve injury characterized by disruption of the axon but with preservation of the connective tissue fragments, resulting in degeneration of the axon distal to the injury site*

[3] *Wallerian degeneration (primary axonal degeneration) - neuropathologic change, occurring as a result of disruption of nerve fibers. Discontinuity of nerve fibers causes changes in axon and myelin sheath. Cut off part of the axon degenerates in the first place. Then the myelin sheath disorders appear as ventricular ischemic, lipid deposits, macrophage infiltration. The disintegration of the cytoskeleton and organelles accumulation is observed microscopically.*

One of clinical symptoms of upper motor neuron damage *is spasticity*[4], whereas the damage of lower motor neuron results in *flaccid paralysis*[5]. The damage to peripheral nerves leads to the atrophy of muscles innervated by the given nerve.

Qualitative methods

Reactions of the neuromuscular system to direct current stimulation.

According to **Du Bois-Reymond's** law (the law of excitation):

> *the direct current flowing through a striated muscle does not cause a contraction. Muscle contraction can occur only during the opening and/or closing of the electric circuit.*

There are many types of contraction:

- anodal closing contraction (ACC): contraction of muscle at the anode upon closure of the electric circuit.
- anodal opening contraction (AOC): contraction of muscle at the anode upon opening of the electric circuit.
- cathodal closing contraction (CCC): contraction of muscle at the cathode upon closure of the electric circuit.
- cathodal opening contraction (COC): contraction of muscle at the cathode upon opening of the electric circuit.

On the basis of experience it is known that the application of DC current of low intensity evokes the muscle contraction only upon closure of the electric circuit if the active electrode[6] is the cathode (CCC). To evoke contraction of a muscle upon opening or closure of the DC electric circuit, if the active electrode is the anode (AOC, ACC), higher current intensity have to be used. The highest intensity of DC current has to be used upon opening of the circuit if the active electrode is the cathode (COC).
The above relations between the strength of a contraction and the type of contraction was given by *W. H. Erb* in the following way (**the Erb law**):

a) upon <u>closure</u> of the electric circuit:

- the strongest contraction takes place if the active electrode is the cathode (CCC)
- the contraction is weaker if the active electrode is the anode (ACC)

$$CCC > ACC$$

b) upon <u>opening</u> of the electric circuit:

- the contraction is stronger if the active electrode is the anode (AOC)
- the contraction is weaker if the active electrode is the cathode (COC)

$$AOC > COC$$

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[4] *spasticity - a state of increased tone of a muscle (and an increase in the deep tendon reflexes).*

[5] *flaccid paralysis - an abnormal condition characterized by the weakening or the loss of muscle tone. It may be caused by disease or by trauma affecting the nerves associated with the involved muscles*

[6] called also *therapeutic electrode* – a small electrode, smaller than an indifferent electrode, with an exciting effect, producing electrical stimulation in a concentrated area; *indifferent (passive) electrode* - one larger than the therapeutic electrode, dispersing electrical stimulation over a larger area.

Nerve and muscle reactions to an impulse of the electric current are different in pathological states and in physiological states. The differences concern the excitability of a nerve and muscle and the deviations from the law of contraction. For instance, if current of 0.5 mA is sufficient to cause muscle contraction, the muscle excitability is increased, whereas the necessity of using higher currents (up to 20 mA) means that the excitability is reduced. One of important symptoms of disruption of the neuronal system in electrodiagnostics is so-called *galvanotonus*. This is when an impulse in the form of direct current causes *tetanic contraction*[7] which is maintained even during breaks of current flow. This phenomenon, suggesting muscle hyper-excitability, occurs in the inflammation of motoneurons and in *tetany*[8]. It can also take place if too high intensity is used. The deviations from the *Erb relations*:

$$CCC < ACC \text{ or } CCC = ACC$$

can be signs of the nerve and/or muscle pathology. Another sign of the pathology of the peripheral nervous system is the presence of the *vermicular contraction* (sluggish, lingering contraction). The presence of the vermicular contraction might be a sign of injury of the peripheral nervous system.

Reactions of the neuromuscular system to faradic and non-faradic current

It is worth mentioning that we distinguish few types of the electric current used in medicine. For example: galvanic current (DC), pulsating current, DC intermittent, alternating current (AC), faradic or non-faradic current (Fig. 2).

Reaction to faradic current is an important element of evaluating the state of the muscle excitability. This type of current elicits a continuous tetanic contraction in normal muscles. This excitability test is used to establish the *reaction of degeneration*[9] (Table 1). Also neofaradic current is used in electrodiagnostics. The advantage of this current is the ability to control time intervals and peak values, unlike in the case of faradic current.

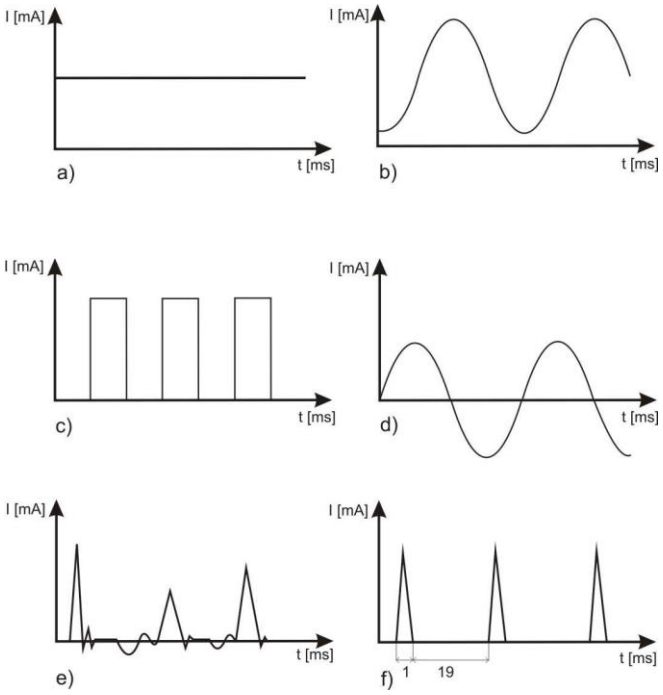

Fig. 2. Few types of electric current: a) galvanic current (DC), b) pulsating current, c) DC intermittent, d) alternating current (AC), e) faradic current f) neofaradic current.

[7] *Tetanic contraction - occurs when the nerve impulses reach the muscle in shorter time than the relaxation occurs – the skeletal muscle stays full contracted under voluntary control.*

[8] *Tetany - pathophysiological condition, manifested by excessive muscle contraction. Occurs at hypocalcaemia.*

[9] *Reaction of degeneration occurs as a result of changes in a muscle after denervation, where the muscle loses the connection with motor neurons. It takes several days after the nerve injury to develop. Depending on the scale of the muscle injury, partial and total reactions of degeneration can be distinguished.*

Table 1**.** Changes in the excitability occurring in the reaction of degeneration.

The type of impulse	Partial reaction of degeneration	Total reaction of degeneration
Faradic or neofaradic current -indirectly	reduced excitability	lack of excitability
Faradic or neofaradic current - directly	reduced excitability	lack of excitability
Intermittent direct current - indirectly	reduced excitability	lack of excitability
Intermittent direct current - directly	reduced excitability	vermicular contraction

Direct excitation – is performed at a motor point of the muscle corresponding to the nerve entry into the muscle (motor endplate of the skeletal muscle). Muscle contraction derived from the motor endplate is stronger than the excitation of the nerve. Indirect excitation - is performed at a motor point of the nerve, i.e. the place where the nerve is placed the most superficially under the skin. It gives the contraction of muscle groups innervated by the nerve.

Quantitative methods - Chronaxymetry

Quantitative methods of the electrodiagnostics involve determination of *chronaxie*, *rheobase* and calculation of the *accommodation coefficient* and the *accommodation quotient*.

The *rheobase* is a measure of tissue excitability represented by the minimum amplitude of the rectangular current impulse, of 1000 ms duration, eliciting the minimal contraction (threshold) of an excitable tissue. The value of rheobase is given in milliamperes. High values of the rheobase mean that the excitability of tissue is low, whereas low values of the rheobase indicate high excitability.

The examination procedure

The first phase of the examination is measurement of the rheobase of a muscle. To do this, the active electrode with the surface area of about 2-3 cm^2 connected to the negative pole of a chronaximeter (the cathode) is applied to the skin in a place corresponding to the *direct motor point*[10] of the examined muscle. A passive electrode (the anode - connected to the positive pole of a chronaximeter), a much bigger one, is placed on the skin surface in a distance from the active electrode. The muscle is stimulated by a rectangular current impulse of 1000 ms duration. The amplitude of the impulse is increased until minimal contraction occurs. This value is the rheobase of the examined muscle.
The second step of the examination is the measurement of chronaxie. To do this the impulse amplitude is set as doubled value of the rheobase, and held constant. The duration of the impulse is increased until minimum contraction occurs. Obtained in this way stimulus duration is the chronaxie.

The *chronaxie* is another measure of the tissue excitability. As already mentioned, the chronaxie is the duration of an impulse heaving the amplitude of doubled rheobase, required to stimulate a tissue, i.e. to evoke minimum noticeable muscle contraction and/or creation of an impulse in a nerve cell (the action potential). The value of chronaxie is given in milliseconds. High values of the chronaxie mean low excitability of a tissue whereas low values of the chronaxie (short duration) indicate high tissue excitability. Values of the chronaxie and rheobase are affected by such factors as:

- conditions of measurement,
- localization of the examined muscle,

[10] *We distinguish two kinds of motor points. Motor point of the nerve (indirect motor point) refers to a place on the skin where the nerve is placed close to its surface. Muscle motor point (direct motor point) is a place where nerve branch enters the muscle. Large muscles can have a few direct motor points.*

- thickness of tissues surrounding the muscle,
- level of blood supply,
- the state of the autonomic nervous system and others.

Despite its limitations the chronaxymetry is often used in the electrodiagnostics.

I/t curve

To elicit the nerve or muscle response the electric impulse must be of rapid onset, amplitude high enough and of long enough duration. The nerve excitability is directly related to the depolarization of the nerve cell membrane. It in turn depends on the amount of electric charge *Q* delivered. The electric charge is the product of the electric current and the time of its flow:

$$Q = i \cdot t$$

where: Q – charge necessary to evoke excitation,
i – intensity of the current (amplitude of the impulse),
t – time of the impulse duration.

The above equation tells that the amount of charge needed to elicit response can be delivered by an impulse of high amplitude and short duration or by an impulse of small amplitude and long duration.
The relationship between the duration of the impulse and its amplitude is given by the ***Hoorweg-Weiss equation***:

$$i = \frac{a}{t} + b$$

where: i – amplitude of the impulse,
a, b – constants, depending on the type of a tissue and its excitability,
t – duration of the impulse.

As the duration of the impulse increases, the value of the factor *a/t* in the above equation decreases and for very long duration it reaches zero. Then the equation takes the following form:

$$i \simeq b$$

what means that the constant *b* is dependent on the value of the threshold current necessary to elicit muscle contraction, and thus it is related to the rheobase. The Hoorweg-Weiss equation implies that the shortening of the electric impulse duration requires an increase of its amplitude if the excitation is to be elicited. This is illustrated in Fig. 3 where the *i/t* curve (solid line) is plotted using values of duration and amplitude of the impulses eliciting minimal contraction (usually of duration from 0.1 to 1000 ms). The *i*/*t* curve constitutes a full picture of the muscle excitability.

The i/t curve can be also obtained by stimulation with triangular impulses. The method of the examination is similar to that with rectangular impulses. Comparision of curves obtained by stimulation with the recangular and triangular impulses provides some information about the excitability of the exmained muscle (in other words about the state of the neuro-muscular system). If, for example, a muscle contracts only in the response to rectangular impulses but not to traingular impulses of the same strength and duration (especially long ones), then this muscle can be regarded as normally innervated.

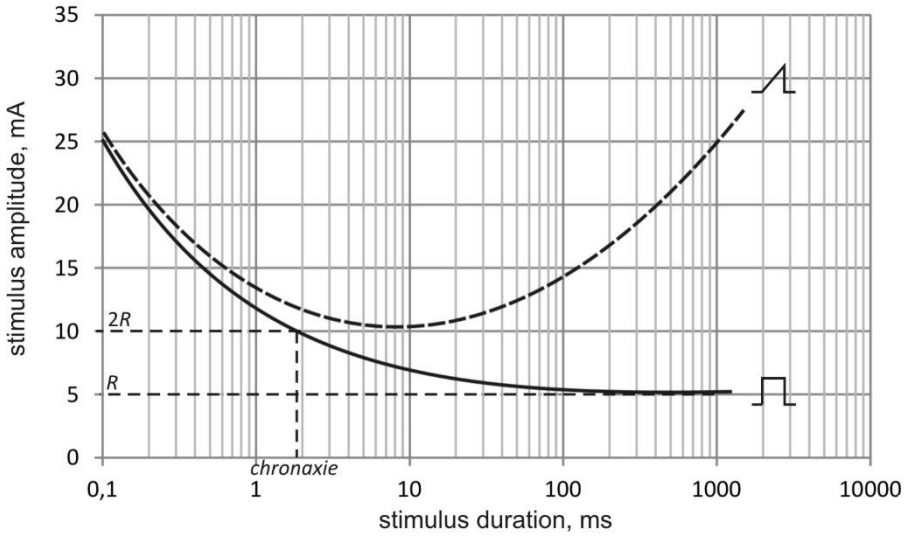

Fig. 3. I/t curve of a healthy muscle obtained by stimulation with rectangular impulses (solid line) and by stimulation with triangular impulses (broken line). The way of determination of the rheobase and chronaxie of the examines muscle is shown.

On the other hand, if the muscle reacts to triangular impulses in a similar way as to rectangular impulses of similar parameters, it indicates denervation and extensive damage. There are many intermediate states of muscle damage between these two extreme situations (see Fig 4).

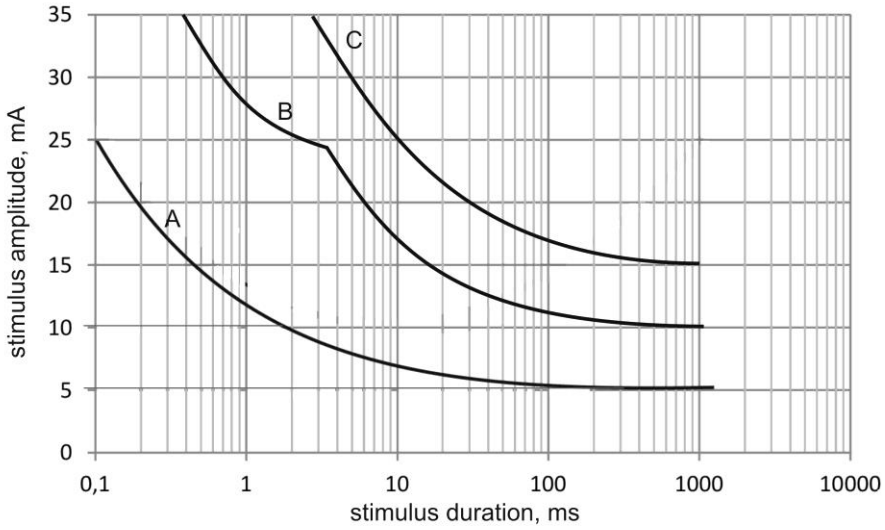

Fig. 4. Examples of i/t curves in various stages of denervation. I/t curves: A – normal, B - partial reaction of degeneration, C – total reaction of degeneration.

Accommodation coefficient and accommodation quotient

By definition the *accommodation coefficient* is the ratio of the *threshold of accommodation*, i.e. the smallest amplitude of a triangular impulse (Fig. 5) of 1000 ms duration, necessary to elicit the minimum noticeable contraction and the value of rheobase:

$$accomodation\,coefficient = \frac{threshold\ of\ \ accomodation}{rheobase}$$

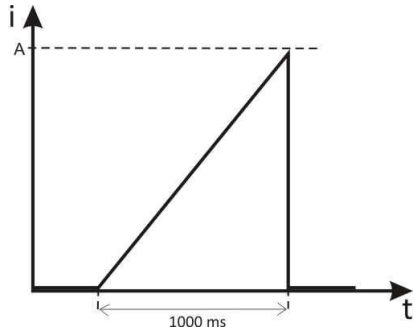

Fig. 5. Triangular impulse of electric current. A- amplitude of the triangular impulse.

This coefficient shows differences in the muscle reaction to rectangular and triangular impulses and reflects the muscle ability to accommodate to slowly increasing intensity in the triangular impulse. It shows how much more intense the current should be in order to achieve a minimum muscle contraction caused by the triangular impulse in comparison to that of the rectangular shape. Values of the accomodation coefficient take the following values and indicate:

- 1-3 – total (1) or partial denervation of the muscle,
- 3-6 – normal neuromuscular excitability,
- more than 6 – *vegetative neurosis*[11].

Accommodation coefficient is very useful in assessment of the excitability of the neuromuscular system, especially in detecting early neuromotor diseases (e.g. early stage *flaccid paralysis* i.e the state in which a muscle shows a significant decrease of the accommodation coefficient as one of the first symptoms). However, the value of the accommodation coefficient is not a sufficient determinant in the diagnosis of the neuro-muscular system degeneration.

The ability of a muscle to accommodate can also be expressed by the *accommodation quotient*.
The accommodation quotient is the ratio of the amplitude of the triangular impulse of 500 ms duration and the amplitude of the rectangular impulse of the same 500 ms duration, both eliciting the minimal contraction of the examined muscle. Values of the accommodation quotient, reflecting the ability of muscles to accommodate, are as follows:

- 1 – total loss of the ability to accommodate,
- 1,1-1,5 – decreased ability to accommodate,
- 1,6-2,5 – normal ability to accommodate,
- 3-4 – increased ability to accommodate.

[11] Vegetative neurosis - Vegetative neurosis (synonym: autonomic dysfunction, vegetate, vegetative dystonia) is a group of diseases, developing as a result of violation of the functions of the upper autonomic centers.

This method is especially useful in the diagnostics of muscles with minor injuries resulting from toxic or mechanical factors. It is also used in the examination of muscles in regions too sensitive to electrical current, such as neck and face.

Experimental procedure

1. Prepare your patient for measurement: clean the places where electrodes are to be attached, ensure comfortable placement of the forearm (muscles should be relaxed).
2. Place the passive electrode (anode, red) outside the examined muscle.
3. With the use of properly prepared active point-electrode (cathode, black) find the motor point of the muscle, for instance the flexor of the thumb. The duration of impulse is 50 ms, which is repeated every 1 s.
4. Prepare the patient: attach the disposable (active) electrode at the motor point, and the passive electrode outside the examined muscle.
5. Make the i/t curve for rectangular impulse stimulation. Start the examination with the impulse of the longest duration i.e. 1000 ms. Continue the examination with impulses of decreasing duration. Plot a graph of the minimum amplitude of the impulses *i* versus their duration *t*: $i = f(t)$. Read the values of the rheobase and chronaxie from the graph.
6. Chronaxymetry. Using the chronaximeter find the value of the rheobase for the examined muscle Next, for the impulse of the doubled rheobase, determine its shortest duration needed to elicit the same contraction as in the case of rheobase – i.e. the chronaxie.
7. Measurement of the accommodation coefficient. Elicit the threshold muscle contraction (i.e. the minimum noticeable contraction) by the triangular impulse of 1000 ms duration. Calculate the accommodation coefficient.
8. On the basis of obtained results assess the state of the examined neuromuscular system.

Person preparation for the purpose of the chronaxymetry experiment – the motor point location

Before electrodes are placed you need to inspect the skin thoroughly to be certain that there are no small abrasion or openings. You should clean the skin of any lotion, oils, dead skin. It can be done with water or with alcohol. Make sure that the skin is dry before the electrodes are placed. Place passive (bigger) electrode outside of the tested muscles and the smaller one (active) in the predetermined locationon the motor point.

The text in part is based upon „Fizykoterapia”, T. Mika, Podręcznik dla wydziałów fizjoterapii medycznych studiów zawodowych, PZWL Warszawa 1996, wydanie II and „Fizjoterapia”, Anna Straburzyńska-Lupa, Gerard Straburzyński, Warszawa PZWL 2007.