

## ORIGINAL RESEARCH PAPER

# INFLUENCE OF EXCHANGED NEUROMUSCULAR REGULATION OF M.GASTROCNEMIUS ON LEG BLOODFLOW REGULATION DURING STATIC VOLUNTARY CONTRACTION OF KNEE FLEXORS

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## Abstract

*In literature from 1970th are described experimentaly proved concept of muscle local blood flow redistribution to capillaries feeding active muscle fibres, and loss of this synhrone blood flow redistribution with changes of recruited motor units, caused by mechanical, metabolic or neural regulatory disturbances. On the other hand, from theory of applied kinesiology (AK) it is known that different external or internal challenges coud exchange neuromuscular regulation and muscle could become weak-testing or become hypertonic. The physiological background of these changes is not jet known. The aim of this study is to find out connections between muscle`s neuromuscular regulation and its local blood flow regulation. We investigate blood flow regulation in the leg during static voluntary contraction with contraction force 5% of the knee flexor muscle maximal static voluntary contraction (MVC) in position with knee flexed till 60 deg. In this experiment participate 8 LASE students in age 22-24 with normotonic m. gastrocnemius and 8 LASE students in age 22-26 with weak-tested m. gastrocnemius. Muscle tests were performed according AK testing methode (Frost, 2002.). Leg blood flow was measured with a mercury-in-silastic strain-gauge plethismograph, using the venous-occlusion plethismography Hokanson AI6 (Hokanson, Sumner and Strandness, 1975). Cycle of leg blood flow measurement was 15 seconds repeated twice in every controled minute. Student paired t-test was used to evaluate diferences in the results of blood flow measurements from strong tested and weak tested leg muscles. We find statistically significant ( $P>95$ ) differences*

*of volume blood flow regulation in muscles triceps surae during static contraction with contracting force of knee flexors 5% MVC. Volume blood flow in leg with exchanged neuromuscular regulation of knee flexors where statistically higher neither blood flow in leg with normal neuromuscular regulation. These differences was observed all the time of the static contraction. Changes of neuromuscular regulation could cause muscles local blood flow regulatory disturbances. Character of muscle local blood flow could be a quantitatively measurable parameter which reflects changes of neuromuscular regulatory processes of studied muscle.*

**Key words:** *Weak-tested muscle, prolonged static contraction, muscle local blood flow.*

### **Introduction**

In literature it is known that different muscle cells have different activity of oxidative phosphorylation and it is known that during light till sub maximal muscle contractions there are active only definite part of all muscle motor units. Simultaneous activity of all motor units is described in literature during maximal voluntary contractions (Skards, Dzerve, 1971) In addition, it is known that during muscle contraction increases intramuscular pressure which reduce muscle's blood supply (Skards, Dzerve, 1973<sup>a</sup>). It means that metabolic activity of muscle cells during static voluntary contraction even in one separate muscle differs. If  $\text{VO}_2$  rate differs between muscle's motor units, then blood flow through capillaries supplying muscle cells must be regulated according to their metabolic activity. In literature from 1970th are described experimentally proved concept of muscle local blood flow redistribution to capillaries feeding active muscle fibres (Паэглитис, 1986; Skards, Paeglitis, Dzerve, Eglitis, Matisone, 1992; Matisone, Skards, Paeglitis, Dzerve 1996) For example during 10%MVC it was shown that  $\text{I}$ ,  $\text{VO}_2$ ,  $\text{La}$  and CFC till the cessation of exercise caused by exhaustion which takes  $42 \pm 1.1$  min stabilizes on appropriate level not reaching maximal possible values. Tacking in account results of the first part of experiment it could be stated that during prolonged contraction with 10%MVC where contraction force was maintained by appropriate amount and loss of this synchronic blood flow redistribution with changes of recruited motor units, of motor units which during contraction changes recruiting new not fatigued motor units blood flow also was distributed not through all the muscle, but through capillaries feeding active muscle fibres. It is obvious also from dynamics of CFC which characterises the number of opened capillaries and which level during

prolonged contraction stays on steady state level not reaching maximal values..

It is also shown that loss of this synchronic blood flow redistribution could be caused with changes of number of recruited motor units (Паэглитис, 1986; Paeglitis, Kukulis, Eglitis, Galeja, 2014). Increasing static voluntary contraction force only for 5% it is to 15%MVC all examined parameters during exercise till exhaustion which takes  $12 \pm 0.8$  min, increases and at moment of cessation of exercise reaches their maximal values. It means that during fatiguing one portion of motor units are switched off and recruits next portion of motor units and the blood flow through capillaries feeding this part of motor unit's remains opened and in addition there are opened new capillaries feeding newly recruited motor units. In the end of contraction there are opened all capillaries and blood flow through the muscle reaches its maximal values. In spite of increasing  $VO_2$  there is activated anaerobic glycolysis which characterises with increasing delivery of La. Increasing of blood supply and  $VO_2$  during 15% MVC did not provide prolonged forearm contraction and exhaustion occurs more than 3 times quicker neither during 10%MVC. Loss of this synchronic blood flow redistribution with continuously increasing of local blood flow during contraction could be observed in situations linked with mechanical, metabolic or neural regulatory disturbances (Skards, Paeglitis, 1985; Паэглитис, 1986). On the other hand, from theory of applied kinesiology (AK) (Walther, 2000; Frost, 2002; Rosner, Cuthbert, 2012) it is known that different external or internal challenges could exchange neuromuscular regulation and muscle could become weak-testing or become hypertonic. The physiological background of these changes is not yet known. We consider that any exchanges of neuromuscular regulation would be connected with changes of functional state of any organ or system of the body. The aim of this study is to find out connections between muscle's neuromuscular regulation and its local blood flow regulation. It gives the opportunity to approve the qualitatively results of AK tests with quantitatively measured values.

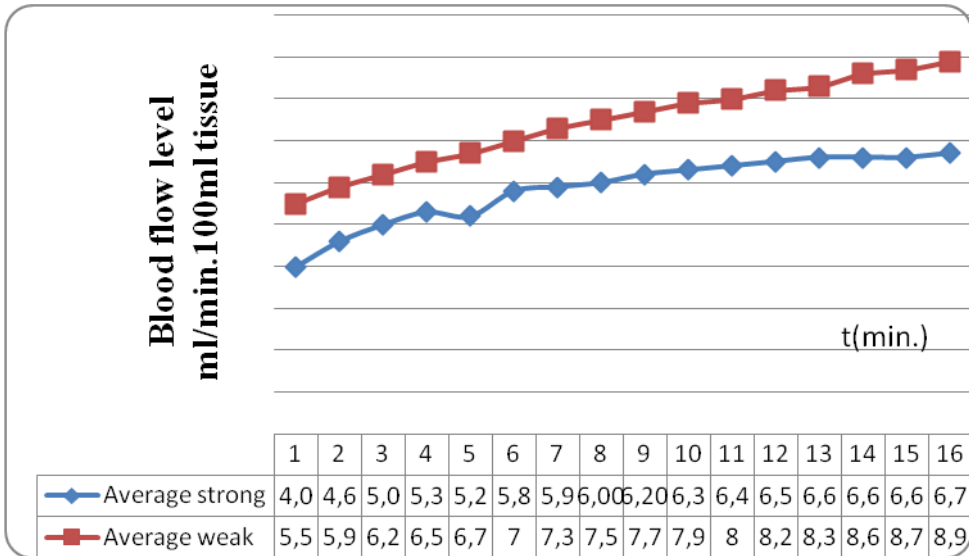
## **Material and methods**

We investigate blood flow regulation in the leg during static voluntary contraction with contraction force 5% of the knee flexor muscle maximal static voluntary contraction (MVC) in position with knee flexed till 60 deg. All participants were examined in prone position. In this experiment participate 8 LASE students in age 22-24 with normotonic m. gastrocnemius and 8 LASE students in age 22-26 with weak-tested m. gastrocnemius. Muscle tests were performed according AK testing method (Frost 2002;

Ramšak, Gerz, 2005) in prone position. MVC was measured with electrical dynamometer Lafayette Instrument (model 01165 Manual Muscle Tester). Angle of knee flexion was measured by goniometer Basoline AcuAngle Inclinator Japan. Leg blood flow was measured with a mercury-in-silastic strain-gauge plethysmograph, using the venous-occlusion plethysmography Hokanson AI6 (Hokanson, Sumner, Strandness, 1975). Leg blood flow (ml/min per 100ml of leg volume) was calculated from the rate of the increase in leg volume, whereas venous return from the leg was prevented by inflation of a cuff on the thigh. The pressure in the venous – occlusion or congesting cuff was 40mmHg. Circulation to the foot was arrested by inflating to the 160mmHg a cuff placed around the lower part of the leg. A mercury-in-silastic strain-gauge was placed around the widest part of the leg around the belly of gastrocnemius to measure changes in leg volume resulting from changes in blood flow. Cycle of leg blood flow measurement was 15s repeated twice in every controlled minute. Student paired t-test was used to evaluate differences in the results of blood flow measurements from strong tested and weak tested leg muscles. A level of  $\alpha \leq 0.05$  was selected to indicate statistical significance.

## Results

We find statistically significant ( $P > 95$ ) differences of volume blood flow regulation in muscles triceps surae with and without exchanged neuromuscular regulation during static contraction with contracting force of knee flexors 5%MVC. Volume blood flow in leg with exchanged neuromuscular regulation of knee flexors were statistically higher neither blood flow in leg with normal neuromuscular regulation. These statistically significant differences were observed all the time of the static contraction. Analysing character of blood flow curves it is obvious that dynamics of blood flow changes in functionally normal triceps surae muscles during static voluntary contraction of knee flexors shows steady-state on the blood flow level 6.5ml/min.100ml tissue, but in triceps surae muscles with exchanged neuromuscular regulation blood flow dynamics during static voluntary contraction of knee flexors increases continuously during all the time of static contraction.



**Figure 1.** Dynamics of volume blood flow in m. triceps surae during 16min static contraction of knee flexors with (■) and without (◆) exchanged neuromuscular regulation

**Discussion**

Comparing obtained results with such published in literature the values of volume blood flow are similar (Skards, Dzerve, 1973<sup>b</sup>; Паэглилис, 1986). It means that blood flow realises through small part of all capillaries because all capillary dilatation gives volume blood flow approximately 30ml/min/100ml. In functionally normal triceps surae stabilisation of volume blood flow during prolonged static contraction is in conformity with theory of blood flow redistribution through capillaries feeding active muscle fibres (Паэглилис, 1986). In literature is discussed role of vegetative nervous system providing local muscle blood flow regulation (Saito, Kagaya, Ogita Shinohara, 1992). Inability to stabilise blood flow during prolonged static contraction in literature is explained as uncoordination between regulatory processes of somatic and vegetative nervous systems (Skards, Paeglitis, 1985; Паэглилис, 1986). Anyway, exchanged neuromuscular regulation is connected with disturbances in regulatory possibilities of vegetative nervous system and as it is so, then such a situation is also connected with inability to stabilise muscles local blood flow.

## Conclusions

Changes of neuromuscular regulation could cause muscles local blood flow regulatory disturbances. Character of muscle local blood flow could be a quantitatively measurable parameter which reflects changes of neuromuscular regulatory processes of studied muscle.

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