# EFFECT OF DIFFERENT OCCLUSION PRESSURE ON PECULIARITIES OF MUSCLE BLOOD FLOW

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## ABSTRACT

*Background*. Occlusion pressure intensity influences the blood flow intensity. Immediately after the cuff pressure is released, reactive hyperaemia occurs. Increased blood flow and nutritive delivery are critical for an anabolic stimulus, such as insulin. The aim of study was to find which occlusion pressure was optimal to increase the highest level of post occlusion reactive hyperaemia.

*Methods*. Participants were randomly assigned into one of the four conditions ($n$ = 12 per group): control group without blood flow restriction, experimental groups with 120; 200 or 300 mmHg occlusion pressure. We used venous occlusion plethysmography and arterial blood pressure measurements.

*Results*. After the onset of 120 and 200 mm Hg pressure occlusion, the blood flow intensity significantly decreased. Occlusion induced hyperaemia increased arterial blood flow intensity 134 ± 11.2% ($p < .05$) in the group with 120 mmHg, in the group with 200 mmHg it increased 267 ± 10.5% ($p < .05$), in the group with 300 mmHg it increased 233 ± 10.9% ($p < .05$)**.** Applied 300 mmHg occlusion from the 12 minute diastolic and systolic arterial blood pressure decreased statistically significantly.

*Conclusions*. Occlusion manoeuvre impacted the vascular vasodilatation, but the peak blood flow registered after occlusion did not relate to applied occlusion pressure. The pressure of 200 mmHg is optimal to impact the high level of vasodilatation. Longer than 12 min 300 mmHg could not be recommended due to the steep decrease of systolic and diastolic blood pressures.

**Keywords:** blood flow restriction, hyperaemia, blood flow intensity, arterial blood pressure.

## INTRODUCTION

Athletes and coaches are looking for the most efficient training method so as to achieve maximum results and to maintain these results for the longest possible period. One of a non-traditional training methods has been developed that uses occlusion, in some literature known as the "Kaatsu" methodology (Ozawa, Koto, Shinoda, & Tsubota, 2015; Sato, 2005). The key point of occlusion training is that blood flow restriction in combination with low intensity (20–50% of maximal voluntary contraction) exercise training has consistently been shown to increase muscle size and enhance function (Dankel et al., 2016; Gundermann et al., 2012; Patterson & Ferguson 2010; Yasuda, Loenneke, Thiebaud, & Abe, 2015). Restriction of blood flow by occlusion applied during the exercise diminishes time or repetitions to task failure (Loenneke et al., 2012) and thus reduces the mechanical loading. The combination of low-intensity resistance training with restricted venous blood flow to the working muscle may provide an alternative training method to the traditional high-intensity resistance training that is used (Shinohara, Kouzaki, Yoshihisa, & Fukunaga, 1998).

Blood flow is one of the important parts in the chain of delivery oxygen and energy substrates to working muscles. Blood flow intensity in muscles varies depending on their functional activity. There are great differences in the data provided by various authors. When comparing the effects of different training exercises on muscle blood flow, it has been observed that endurance training

reduces the intensity of blood flow at rest (Delp & Laughlin, 1998; Villar & Hughson, 2017). Exercise is one of the most powerful non-pharmacological methods of affecting cells and organs in the body (Shalaby, Saad, Akar, Reda, & Shalgham, 2012). Regular aerobic and resistance exercise training has a positive long-term impact on the cardiovascular system, which is a biologically complex adaptive system that is characterized by a variety of complex reactions to different training loads (Alex et al., 2013; Ellison, Waring, Vicinanza, & Torella, 2012; Gibala, Little, Macdonald, & Hawley, 2012).

Applied acute occlusion or occlusion training, its cuff pressure, length, and width are varied, the compressive pressure varies between studies, but typically, the cuff is inflated to a pressure greater than brachial diastolic blood pressure and upward of pressures exceeding systolic blood pressure (Horiuchi & Okita, 2012; Manini & Clark, 2009). Occlusion pressure intensity influences the blood flow intensity. Immediately after the cuff pressure is released, reactive hyperaemia occurs, increased blood flow and nutritive delivery are critical for an anabolic stimulus, such as insulin (Gundermann et al., 2012). After cuff release, nitric oxide and nitric oxide bioavailability increases, the endothelium of blood vessels use nitric oxide to signal the surrounding smooth muscle to relax, thus resulting in vasodilation and increasing blood flow (Horiuchi & Okita, 2012).

The aim of study was to find-out which occlusion pressure was optimal to increase the highest level of post occlusion reactive hyperaemia.

# METHODS

**Participants.** The participants in the study were amateur male middle and long distance track and field runners with 4–6 years of training experience. The study was performed when athletes had the rest period without active training; they had only muscle flexibility exercises, except for calf muscles. None of the participants exercised for at least 12 hours and before the test or ate for at least 3 hours before the test. The weight and body mass index (BMI) (TBF-300 body composition scale; Tanita, UK Ltd., West Drayton, UK) of the participants were estimated while they were semi-nude (shorts and T-shirts). This study was approved by the Regional Biomedical Research Ethics Committee. Participants were randomly assigned into one of the four conditions ($n$ = 12 per group): control group without blood flow restriction, experimental groups with 120; 200 or 300 mmHg pressure occlusion (Table 1).

**Study design.** The participants were seated for 15 min on a chair, with the leg fixed at an angle of 90°, and the ankle at an angle of 70°. In the experimental groups, the participants underwent circulatory restriction with a 40-mm-wide cuff on the groin, the belt air pressure was respectively set at 120, 200 or 300 mmHg (respectively the approximate below, resting and more than resting systolic blood pressure in calf muscle). In the control group, participants seated on a chair without occlusion (Table 2).

Table 1. **Characteristics of the experimental and control groups**

| Group | Occlusion pressure (mmHg) | Occlusion duration (min) | Age (years); Body mass index (kg·m$^{-2}$) |
|---|---|---|---|
| Experimental group 1 ($n$ = 12) | 120 | 15 | 22.7 ± 0.7; 21.3 ± 0.4 |
| Experimental group 2 ($n$ = 12) | 200 | 15 | 22.6 ± 0.6; 21.4 ± 0.3 |
| Experimental group 3 ($n$ = 12) | 300 | 15 | 21.9 ± 0.3; 20.8 ± 0.5 |
| Control group ($n$ = 12) | 0 | 0 | 22.1 ± 0.4; 22.5 ± 0.7 |

Table 2. **Organization of the study**

| Rest 30 min | Registration of Initial values 3 min | Occlusion or rest | Registration of recovery 5 min |
|---|---|---|---|

**Measurements. Venous occlusion plethysmography.** The arterial blood flow intensity in the calf muscles was measured using venous occlusion plethysmography. Changes in the calf volume were determined with a modified Dohn's plethysmograph. An air-filled measuring cuff of latex rubber (width, 5 cm; pressure, 4 cm $H_2O$) was fitted around the thickest portion of the calf. Venous occlusion on the distal part of the thigh was achieved by fast inflating the cuff to a pressure of 70 mmHg. The third cuff was fixed at the ankle, and closed off the circulation to the foot during the measurements. The examinations were conducted in the room temperature (22–23°C) with the subject in a sitting position.

**Arterial blood pressure.** Arterial blood pressure is an important cardiovascular functional parameter. It was measured using the cuff method and by listening to the "Korotkoff" tones (American Diagnostic Corporation Prosphyg™ 770 Aneroid Sphygmomanometer). Arterial blood pressure was measured before and after each set.

**Statistical analysis.** The data were tested for normal distribution using the Kolmogorov–Smirnov test, and all data were found to be normally distributed. Mixed analysis ANOVA (General Linear Model) with one between-subjects factor (control group and experimental group), and two conditional factors (exercise and recovery dynamics) as within-subjects factors of different levels was used. If significant effects were found, Sidak's post hoc adjustment was used for multiple comparisons within each repeated-measure ANOVA. The level of significance was set at $p < .05$. Herewith the calculations for observed power (OP) were performed and the partial eta squared (ŋ2) was estimated as a measure of the test-retest effect size. All statistical analyses were performed using IBM SPSS Statistics 22 (IBM Corporation, Armonk, New York). Data are presented as mean ± SE.

## RESULTS

While the 15-min occlusion applied in the groups with 120 mmHg ($p < .05$, $\eta^2 = 0.879$, $OP = 1.0$), with 200 mmHg ($p < .05$, $\eta^2 = 0.761$, $OP = 1.0$) and with 300 mmHg ($p < .05$, $\eta^2 = 0.779$, $OP = 1.0$) pressure significantly decreased the blood flow intensity after onset of occlusion from the first minute compared to the initial blood flow before occlusion. However at the end of 15-min period, arterial blood flow intensity differed statistically significantly between all groups ($p < .05$). In experimental group with 120 mmHg, arterial blood flow decreased 41 ± 9.9 % ($p < .05$), with 200 mmHg decreased 58 ± 8.6% ($p < .05$) and with 300 mmHg occlusion pressure decreased 71 ± 8.9%. In the control group, while participants were at rest and sat without occlusion, arterial blood flow decreased only 9 ± 9.7% ($p < .05$) (Figure 1).

Applied 300 mmHg pressure occlusion ($p < .05$, $\eta^2 = 0.174$, $OP = 0.893$) from the 13 minute statistically decreased diastolic and systolic arterial blood pressure compared to initial values before occlusion. In other experimental groups,

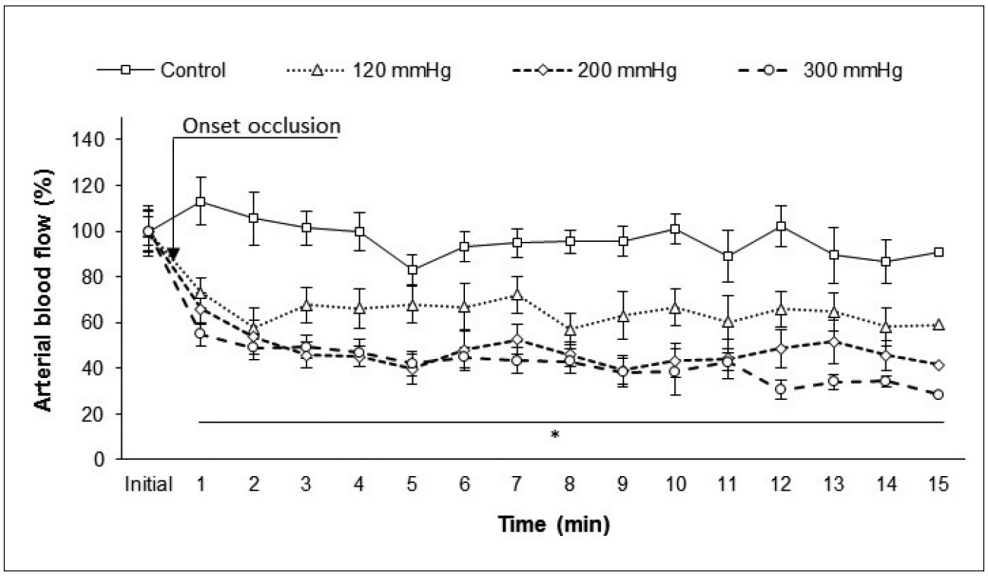


Figure 1. **Changes of arterial blood flow intensity in experimental groups during 120; 200 or 300 mmHg pressure occlusion and in control group without occlusion at rest**

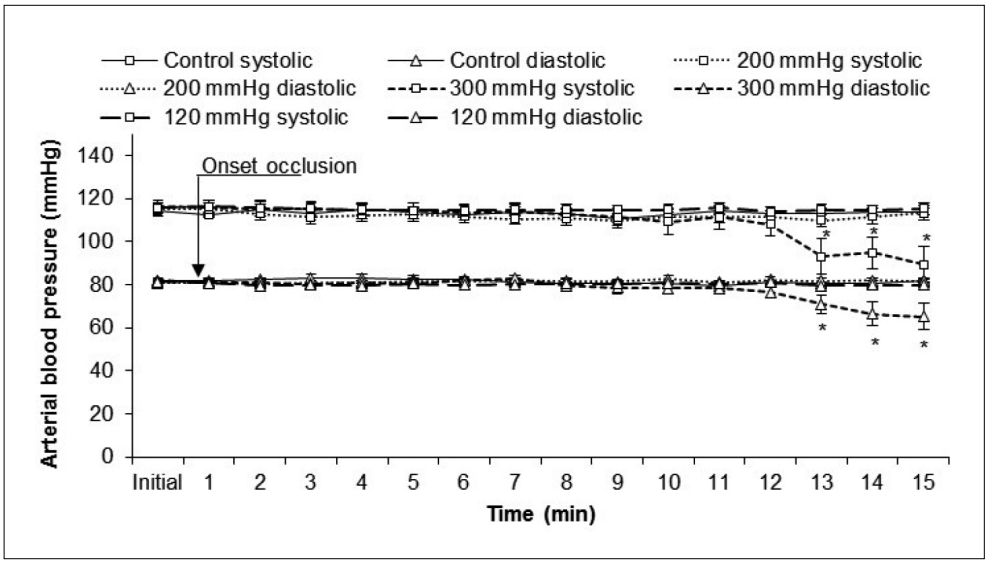


Figure 2. **Changes of arterial blood pressure in experimental groups during 120; 200 or 300 mmHg pressure occlusion and in control group without occlusion at rest**

120 and 200 mmHg occlusion pressure had no significant effect on systolic and diastolic blood pressure, there was also no change in the control group (Figure 2).

Immediately after occlusion removal in group with 120 mmHg ($p < .05$, $\eta^2 = 0.855$, $OP = 0.1$), 200 mmHg ($p < .05$, $\eta^2 = 0.924$, $OP = 1.0$) and 300 mmHg occlusion pressure ($p < .05$, $\eta^2 = 0.742$, $OP = 0.1$), arterial blood flow intensity increased statistically significantly compared to values at 15 min of occlusion. Occlusion induced hyperaemia increased arterial blood flow intensity 134 ± 11.2% ($p < .05$) in experimental group with 120 mmHg, in the group with 200 mmHg it increased 267 ± 10.5% ($p < .05$), and in the group with 300 mmHg occlusion pressure it increased 233 ± 10.9% ($p < .05$). In the control group after rest without occlusion, arterial blood flow intensity increased only 17 ± 9.9% ($p > .05$). After 3 min recovery, blood flow intensity returned to the baseline level (Figure 3).

Applied 300 mmHg occlusion ($p < .05$, $\eta^2 = 0.695$, $OP = 0.1$) immediately after occlusion removal, diastolic and systolic arterial blood pressure was decreased statistically significantly till 4 min of recovery ($p < .05$), compared to initial values before occlusion. In the other experimental groups with 120, 200 mmHg occlusion pressure, it had no significant effect on systolic and diastolic blood pressure ($p > .05$), there was also no change in the control group (Figure 4).

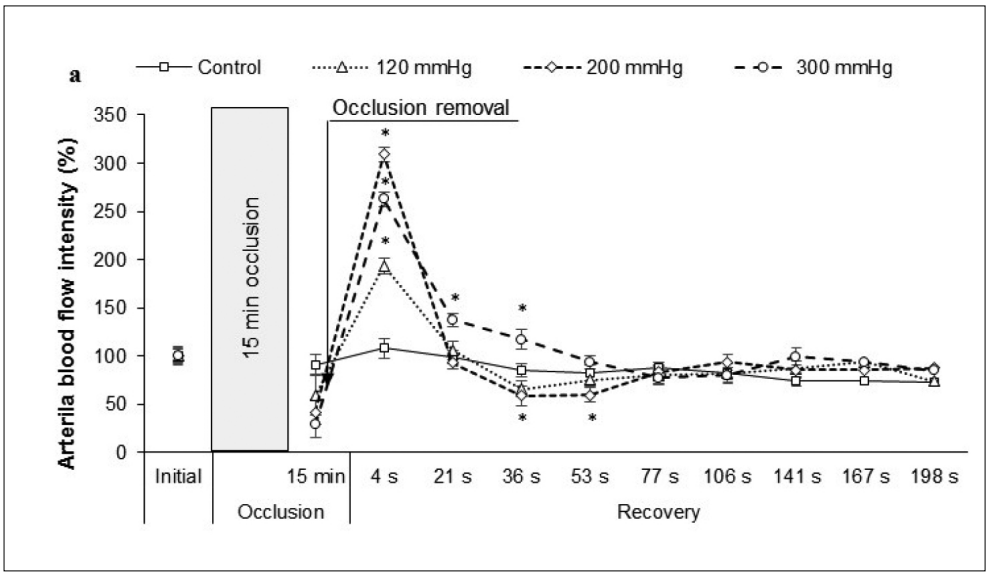


Figure 3. **Changes of arterial blood flow intensity in experimental groups after removal of 120; 200 or 300 mmHg pressure occlusion and in control group at rest without occlusion**

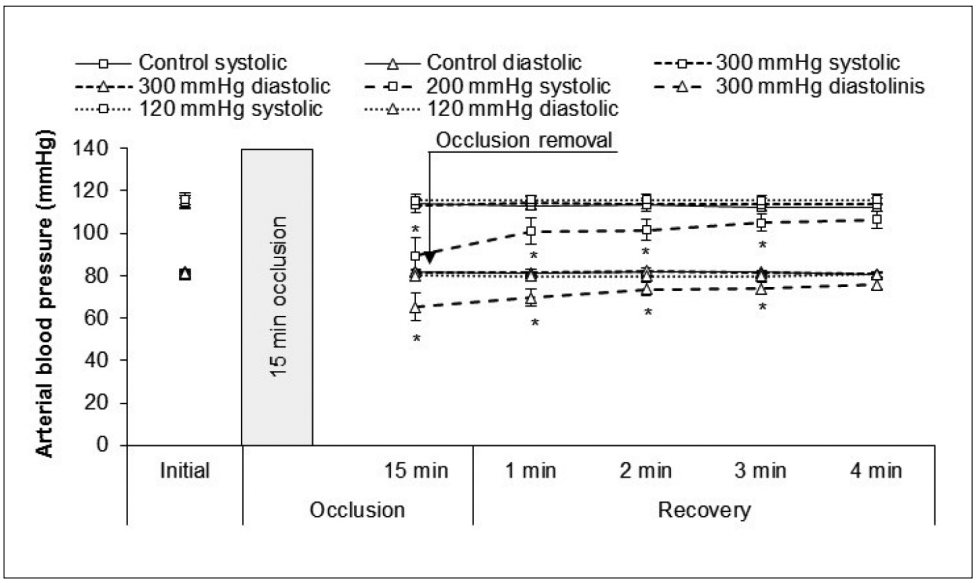


Figure 4. **Changes of arterial blood pressure in experimental groups after removal of 120; 200 or 300 mmHg pressure occlusion and in control group at rest without occlusion**

## DISCUSSION

Rhythmic muscle contractions may affect the cardiovascular system in two ways: to reduce the blood flow within inactive muscles and increase arterial blood flow intensity in active musculature (Behringer & Segal, 2012; Fan et al., 2008; Holwerda, Restaino, & Fadel, 2015). Referring to the findings of various authors we can find sources about sufficiently high variations in arterial blood flow intensity at rest. Raitakari et al. (1996) found from 1.1 to 7.5 mL/100 mL/min of arterial blood flow intensity in the human muscles at rest. In our study, in the sample of muscle endurance representatives, arterial blood flow intensity ranged from 1.6 ± 0.2 mL/100 mL/min to 2.1 ± 0.3 mL/100 mL/min at rest (Raitakari et al., 1996). Blood flow intensity levels at rest were within the same limits as presented in the literature data. Muscle arterial blood flow intensity varies depending on the functional condition as well as on internal and external stimuli. As it was shown, short-term adaptation to endurance exercises does not have a significant effect on skeletal muscle arterial blood flow intensity at rest, while long-term adaptation to endurance exercises significantly effects the intensity of circulation, i.e. reduces the intensity of arterial blood flow in the muscles at rest (Delp & Laughlin 1998).

During passive recovery, occlusion influences muscle arterial blood flow significantly reducing the intensity of it. Results obtained in our study showed that immediately after the 15 min occlusion, arterial blood flow intensity significantly increased. Thus, the occlusion manoeuvres decreased arterial blood flow intensity, and after occlusion there was a short-term reactive hyperaemia. The most interesting fact was that the peak blood flow after occlusion was not related to applied occlusion pressure. The response of blood vessels to pressure of 300 and 120 mmHg was less than to the influence of 200 mmHg. The applied occlusion pressures in restricting blood flow varies between studies, but typically the cuff is inflated to a pressure greater than brachial diastolic blood pressure and upward of pressures exceeding systolic blood pressure (Manini & Clark, 2009).

Was observed arterial blood pressure changes at rest during occlusion, during the first three minutes, the systolic arterial blood pressure increased significantly. During ischemic manoeuvres arterial blood pressure increases, and when the air is released from the cuff, it decreases, but it can be vice versa, i.e., the systolic and diastolic arterial blood pressure can gradually increase (Horiuchi & Okita, 2012). In our study, applying 15 min 120 and 200 mmHg occlusion during passive recovery, we observed that systolic and diastolic arterial blood pressure was almost unaffected, while longer than 12 min 300 mmHg pressure occlusion extremely decreased systolic and diastolic blood pressures.

Dynamic hyperaemia due to the dilation of small blood vessels after the cuff release may act by increasing muscle performance because of the

maximised oxygen supply via reduced lag in oxygen uptake ($VO_2$ transient) and diminished oxygen debt; it may also increase the early by-product wash-out. However, the metabolite build-up during the blood flow restriction could hinder/counteract the achieved benefits in augmented early circulation and in fact may reduce working capacity. The balance of ergogenic to ergolytic effects depends on the degree and duration of the occlusion applied. Among athletes of certain sporting events such as track running, there is a popular belief and practice based on some evidence (Salvador et al., 2016) that working capacity might be increased by raising the legs above the heart level for few minutes prior to the race. The proxy of this practice, also termed ischemic preconditioning, is repetitive blood flow restriction and relief cycles performed prior to exercise. Concomitant occlusion and elevation of the limbs is another variation of this practice. All of these practices are performed with an aim to reduce the blood flow during the procedure, and then induce reactive hyperaemia, oxygen delivery and then (maintenance of) intensity during subsequent competitive exercise. Aside from the effect on the ability to perform subsequent post-release exercise, blood flow restriction deserves investigation as a possible additive training stimulus.

## CONCLUSIONS

Occlusion manoeuvre impacted the vascular vasodilatation, but the peak blood flow registered after occlusion did not relate to the applied occlusion pressure. The pressure of 200 mmHg is optimal to impact the high level of vasodilatation. Longer than 12 min 300 mmHg could not be recommended due to the steep decrease of systolic and diastolic blood pressures.

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