



Blood Flow Restriction (BFR) Therapy in Musculoskeletal Rehabilitation

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Abstract

Blood flow restriction (BFR) therapy has emerged as a novel approach to improve outcomes in musculoskeletal rehabilitation. It was previously thought that heavy-load therapy at 60-70% of one's maximum effort (1RM) was the primary way to increase strength and improve function. However, current evidence suggests that venous occlusion proximal to a muscle body may confer the same results with low-load therapy at 20-30% of 1RM. Due to deconditioning, fragility, and injury, heavy-load training is not always possible. Therefore, low-load BFR may prove to be as a safe and effective method of rehabilitation in certain populations. Ongoing research is dedicated to determine the exact mechanism and optimal approach to applying BFR in the clinical setting.

Keywords

Blood flow restriction, Rehabilitation, Hypertrophy

Low-load blood flow restriction (BFR) therapy may represent a novel approach to increase muscle size and optimize function. First developed in Japan in the late 1960's as Kaatsu training, BFR has since been refined and utilized throughout the world in not only rehabilitation, but also in strength and endurance training [1]. The technique entails applying a tourniquet-style cuff to the proximal aspect of the limb prior to exercise and inflating the cuff as to occlude venous return yet allow arterial flow.

Traditionally, heavy-load strength training, which involves repetitions of 60-70% of one's maximum effort (1RM), was thought to be the most effective way to increase strength and function. However, in individuals with musculoskeletal impairment, including those rehabilitating from injury or surgery, heavy-load training is not always feasible or safe due to fragility and deconditioning. BFR, when combined with multiple-repetition light-load training at 20-30% 1RM, has been shown to produce significant gains in muscle strength and hypertrophy in healthy populations [2].

Despite ongoing biochemical analysis, the exact mechanism of BFR remains unclear. Several hypotheses exist attempting to explain physiologic improvements related to BFR. One theory is that low intensity exercise combined with BFR produces a "metabolic overload", thus

effectively reproducing metabolic changes (i.e. depletion of phosphocreatine stores and lactic acidosis) normally associated with high intensity resistance exercise. These changes would then result in increased fiber recruitment and growth hormone release [3]. The "hormesis" hypothesis, proposed by Loenneke, et al. in 2013, suggests that BFR occlusive pressures may be beneficial up to a certain point after which higher pressures produce harmful biologic effects. In keeping with the "metabolic overload" hypothesis, metabolic stress with BFR likely increases with increasing pressure to a point where the response is no longer augmented. Preliminary studies using electromyography to measure muscle fiber recruitment conclude that low to moderate pressure BFR stimulates muscle activation, whereas pressures closer to arterial occlusion may be inhibitory [4].

Alternatively, another theory is that BFR may alter

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gene expression by downregulating MURF-1, atrogin, and myostatin-genes and proteins that are involved in the degradation of muscle fiber [3]. More recent studies have indicated positive results from the application of BFR in the absence of exercise. This suggests that the mechanism by which BFR improves strength and function may be independent of exercise physiology. Loenneke, et al. hypothesizes that occlusion causing cellular dehydration in muscle tissue may stimulate the mTOR pathway resulting in hypertrophy [3].

The biological effects of low resistance training with BFR have recently been studied in animal models. One study by Pour, et al. in 2017 examined the effects of BFR on muscle hypertrophy and neuromuscular signaling in aging rats. In this study, 48 rats were divided and BFR was compared to sham treatment with exercise. After 10 weeks, the soleus, a slow-twitch postural muscle, and the extensor digitorum longus (EDL), a fast-twitch anaerobic muscle, were removed from all rats and examined for hypertrophy and ACh receptor alterations. The results indicated significant increases in hypertrophy in the EDL of the BFR + exercise group, as well as increased acetylcholine (ACh) receptors in the EDL and soleus of the BFR + exercise groups. The authors concluded that BFR along with low-intensity exercise conferred beneficial effects on muscle mass and ACh receptor clustering at the neuromuscular junction in old rats, suggesting that BFR may represent a safe and effective method of increasing muscle mass and motor skills in aging individuals [5].

Additionally, clinical application of BFR is currently under investigation in human subjects. One 2017 pilot study by Gaunder, et al. reviewed the benefit of BFR in postoperative lower extremity rehabilitation after total knee arthroplasty (TKA). Quadriceps weakness after TKA is negatively correlated with long-term outcome. In this trial, 3 individuals who had previously failed traditional rehabilitation for quadriceps strengthening following TKA were subjected to 8 weeks of BFR 3 times weekly. All 3 individuals experienced an increase in peak torque with flexion and extension, suggesting progress where traditional rehabilitation had failed [6]. However, this study was limited by its size and absence of a control population.

Another 2017 systemic review and meta-analysis by Hughes, et al. examined the outcome of low-load BFR in musculoskeletal rehabilitation toward anterior crucial ligament (ACL) reconstruction, knee osteoarthritis, sarcopenia, and sporadic inclusion body myositis. The aim of this review was to compare the efficacy of low-grade BFR to both low-load and heavy-load training without BFR and to provide recommendations regarding safe and effective application of BFR in the rehabilitation set-

ting. Parameters varied widely, including load, occlusion pressure, frequency of therapy, and duration of therapy. From their review, the authors concluded that compared to low-load training alone, supplemental BFR produced greater responses in muscular strength in nearly 70% of the population studied. While strength gains were inferior to those achieved by heavy-load training, low-load BFR produced a comparable increase in muscle hypertrophy as measured by muscle cross-sectional area. Additionally, augmentation with BFR showed potential to prevent muscle atrophy in early immobilization as determined by functional outcome measures [7]. This study was limited by lack of uniformity in BFR application. Regardless, the authors concluded that low-load BFR is more effective than low-load therapy alone and more tolerable than heavy-load therapy, making it an intriguing approach to musculoskeletal rehabilitation in certain populations.

One ongoing randomized clinical trial by Ladlow, et al. continues to provide insight on BFR prescription. This study follows 28 individuals aged 18-50 participating in rehabilitation for lower extremity injury with a goal of investigating the clinical outcomes of BFR versus conventional resistance training in active military personnel. Outcomes under consideration include hypertrophic and strength response, balance, compliance, pain response, and adverse events. All outcomes will be assessed at baseline and after 3 weeks of inpatient rehabilitation [8]. This study, and likely more to come subsequently, will not only provide additional insight into the efficacy of BFR, but will also help standardize occlusion pressure and exercise prescription to allow reproducible protocols in clinical rehabilitation.

That being said, existing recommendations are unclear as to the exact protocol for implementation of BFR. Current evidence shows 40-80% of arterial occlusion pressure is likely safe when supervised by experienced practitioners [1]. However, primary outcomes suggest thigh circumference and cuff width may influence actual occlusive pressure. Therefore, individualized approach to BFR is essential. The greatest benefit with low-intensity BFR resistance training has been observed with 20-30% of 1RM 2-3 days per week. Ideal training involves 75 repetitions over 4 sets: 30, 15, 15, and 15 with 30-second rest periods between sets. Similarly, low-intensity BFR aerobic training involves walking in 10-15-minute intervals 2-3 times per week to achieve 30% of heart reserve, which has been linked to significant cardiorespiratory endurance following 6-week trials [1].

Despite general tolerability in current literature, several potential adverse reactions exist with the use of BFR. The first of these, albeit rare, is thrombus forma-

tion. Data from two surveys of nearly 13,000 individuals found the incidence of DVT was $< 0.06\%$ and PE $< 0.01\%$ with utilization of BFR [1]. The risk of clot formation increases with occlusion pressures $> 130\%$ of systolic blood pressure [4]. The next is paresthesias from nerve compression. The incidence is again low ($< 2\%$) and typically transient. Similarly, it can usually be avoided by appropriate cuff selection and reduced occlusion pressure. Additionally, autonomic dysreflexia has been reported in high-risk populations including individuals with heart failure, hypertension, and peripheral arterial disease [1]. Lastly, muscle damage is also a theoretical concern. However, the incidence of rhabdomyolysis is $< 0.01\%$ and typically seen in untrained individuals, rates similar to those observed with exercise alone in the same population [1].

Overall, BFR training is a novel method to help achieve improvements in strength and function in individuals unable to tolerate heavy-load training. However, future research is needed to explore the scope of its benefit in rehabilitative medicine. While preliminary data shows its role in optimizing muscle hypertrophy and function, early data suggests it may also help promote bone formation [4]. Additionally, establishing an individualized regimen for occlusion pressure and cuff width to maximize outcome and minimize adverse events is essential in fully utilizing BFR in the clinical setting.

In conclusion, BFR when applied to low-load resistance and aerobic training may represent a safe and effective treatment modality in musculoskeletal rehabilitation. Although its exact mechanism remains unclear, its effects are likely secondary to a combination of biochemical cascades, gene modification, neuromuscular changes. Although neurovascular events are possible with BFR, adverse reactions are rare when appropriately supervised. However, future studies are necessary

to better understand the benefits and risks for BFR in clinical and non-clinical settings. Regardless, it remains clear that BFR is likely emerging as a cost-effective, practical, and safe rehabilitation modality with a potential for widespread clinical use.

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