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Blood Flow Restriction Training in Rehabilitation: A Narrative Review of Musculoskeletal and Neurological Applications, Mechanisms, and Safety

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Abstract

Background. Blood flow restriction (BFR) training combines low-load exercise with a proximal cuff that partially restricts arterial inflow and more substantially impedes venous outflow. Because it produces strength- and hypertrophy-related adaptations at loads too low to threaten healing tissue, BFR has become a prominent rehabilitation adjunct.

Aim. To synthesise, from a clinically oriented and critical perspective, the physiological rationale, methodology of application, condition-specific efficacy, systemic effects, and safety of BFR across musculoskeletal and neurological rehabilitation.

Material and methods. Narrative review of peer-reviewed English-language systematic reviews, meta-analyses, randomised controlled trials, scoping reviews, and clinically oriented reviews addressing BFR in rehabilitation, with emphasis on medical applications and safety.

Results. Low-load BFR consistently outperforms load-matched exercise without occlusion and approaches, without reliably surpassing, high-load training for hypertrophy; maximal-strength gains remain smaller. Evidence is most developed after anterior cruciate ligament reconstruction, in knee osteoarthritis, after knee arthroplasty, in tendon rehabilitation, and increasingly in upper-limb and neurological populations. Benefits for muscle mass, strength, and patient-reported function are frequent; effects on pain and balance are inconsistent. Protocol heterogeneity, small samples, and incomplete adverse-event reporting limit the evidence.

Conclusions. With individualised occlusion pressure, screening, and monitoring, the short-term safety profile is favourable. BFR is a low-risk adjunct for load-compromised patients when higher loads are temporarily contraindicated, to be integrated into progressive rehabilitation rather than used as a universal substitute for conventional training.

Keywords: blood flow restriction; occlusion training; KAATSU; musculoskeletal rehabilitation; neurological rehabilitation; limb occlusion pressure; muscle atrophy; exercise therapy; safety

1. Introduction

Muscle weakness and atrophy are among the most consistent and functionally consequential problems encountered across rehabilitation. They accompany acute injury, surgery, chronic musculoskeletal disease, immobilisation, ageing, and neurological impairment, and they persist as independent barriers to recovery long after the primary tissue insult has healed [1,2]. The conventional remedy is progressive resistance training, and long-standing recommendations hold that external loads of at least 65–70% of one repetition maximum (1RM) are required to reliably increase muscle strength and hypertrophy [1,2]. Yet precisely those patients who most need to arrest muscle loss are frequently unable to tolerate heavy loading. Pain, joint effusion, graft and tissue-healing considerations, arthrogenic inhibition, cardiovascular risk, and general frailty all narrow the therapeutic window in which high loads can be applied [2,3]. This mismatch — atrophy that demands a robust anabolic stimulus in patients who cannot tolerate the loads that classically deliver it — is the clinical problem that blood flow restriction (BFR) training was, in effect, positioned to solve.

BFR, historically termed occlusion or KAATSU training, involves applying an external pneumatic cuff or elastic band to the most proximal region of a limb so that arterial inflow is partially restricted and venous outflow is more substantially impeded during exercise [1]. The technique was pioneered by Yoshiaki Sato in Japan from the 1960s and refined over subsequent decades; it is now practised worldwide and is most commonly delivered with a pneumatic tourniquet system that permits controlled, quantifiable pressures [1]. The defining and clinically attractive feature of BFR is that, when combined with low external loads of approximately 20–40% 1RM, it elicits increases in muscle size and strength that with conventional training would ordinarily require loads near 70% 1RM [1,2]. Because the mechanical stress imposed on joints, grafts, and healing tissue is correspondingly low, BFR offers a means of providing a meaningful

anabolic and neuromuscular stimulus during phases of rehabilitation in which heavy loading is unsafe or intolerable [2,3].

Interest in BFR as a rehabilitation tool has expanded rapidly. What began as a physiological curiosity and a strength-and-conditioning method for healthy and athletic populations has become a subject of systematic reviews, meta-analyses, and randomised controlled trials spanning anterior cruciate ligament (ACL) reconstruction, knee osteoarthritis, knee arthroplasty, tendinopathy and tendon rupture, upper-limb pathology, sarcopenia, and neurological conditions including stroke, multiple sclerosis, spinal cord injury, cerebral palsy, and Parkinson's disease [2,3,4]. This proliferation has been accompanied by a growing appreciation that the promise of the technique is inseparable from questions of methodology and safety: the degree of restriction, the manner in which pressure is prescribed, the choice of cuff, and the screening of patients all determine whether BFR is applied effectively and safely [1,3]. A recurring theme across the literature is that enthusiasm has, at times, outpaced standardisation, and that heterogeneous protocols and inconsistent reporting temper the strength of the conclusions that can currently be drawn [3,5].

The purpose of this narrative review is to provide a clinically oriented and critical synthesis of the use of BFR in rehabilitation, with deliberate emphasis on medical rather than performance considerations. The review addresses, in turn, the physiological rationale and proposed mechanisms of BFR; the methodology of its application, including pressure prescription, cuff characteristics, and exercise dosing; the condition-specific evidence across musculoskeletal and neurological rehabilitation; the systemic effects of the technique beyond the exercising limb; its role in recovery and adjacent applications; and, importantly, its safety, contraindications, and risk stratification. Rather than treating BFR as a uniformly beneficial intervention, the review adopts a comparative stance — weighing BFR against both low-load and high-load conventional training, contrasting findings across conditions, and foregrounding the methodological limitations that qualify the evidence.

2. Materials and Methods

This article was conceived as a narrative review intended to offer a clinically oriented synthesis rather than a systematic review or meta-analysis; accordingly, no review protocol was registered and no formal risk-of-bias appraisal or quantitative pooling was undertaken. The literature considered comprised peer-reviewed, English-language publications addressing the application of BFR in rehabilitation, prioritising position statements, systematic reviews, meta-analyses, randomised controlled trials, scoping reviews, and clinically oriented narrative reviews. Sources were selected for their relevance to musculoskeletal, orthopaedic, sports-medicine, and neurological rehabilitation, for their methodological standing, and for their alignment with the medical focus of this review. Foundational physiological and methodological papers were retained where they provided the mechanistic or technical basis for clinical practice. Evidence was grouped thematically — mechanisms; methodology of application; condition-specific efficacy (general musculoskeletal rehabilitation, ACL reconstruction, knee osteoarthritis and non-traumatic knee pain, knee arthroplasty, tendon rehabilitation, upper-limb conditions, and neurological rehabilitation); systemic effects; recovery and adjacent applications; and safety, risk stratification, and contraindications. Because selection and interpretation were, by design,

narrative, the synthesis may be subject to selection and emphasis bias, and its conclusions should be read as a clinically reasoned overview rather than an exhaustive or formally graded account of all available evidence.

3. Physiological Rationale and Proposed Mechanisms

The central paradox that BFR resolves — substantial muscular adaptation at loads well below the conventional hypertrophic threshold — is explained by a constellation of mechanisms that converge on the same anabolic pathways engaged by heavy resistance exercise, but that are triggered by metabolic rather than predominantly mechanical means [1,4]. Inflation of a proximal cuff produces partial arterial restriction and more pronounced venous occlusion, generating a localised hypoxic, metabolite-rich environment with blood pooling distal to the cuff [1]. Within this environment, the accumulation of hydrogen ions, inorganic phosphate, lactate, and other by-products of anaerobic glycolysis creates the metabolic stress that is now widely regarded as the dominant driver of BFR-induced adaptation when mechanical tension is low [4,6].

Several downstream consequences of this metabolic stress have been proposed. First, the hypoxic and fatiguing intramuscular milieu promotes earlier and more extensive recruitment of higher-threshold, fast-twitch motor units that would ordinarily be reserved for high-intensity effort; these fibres are also disproportionately susceptible to disuse atrophy, which makes their recruitment particularly valuable in rehabilitation [1,4,6]. Second, metabolic stress and hypoxia stimulate anabolic intracellular signalling, notably activation of the mammalian target of rapamycin complex 1 (mTORC1) pathway and increased muscle protein synthesis, together with attenuation of proteolytic pathways [4,6]. Third, cell swelling secondary to venous pooling and osmotic shifts is thought to contribute an additional anabolic signal [1,4]. Fourth, systemic endocrine responses — including acute elevations in growth hormone and, in some studies, insulin-like growth factor 1 and catecholamines — accompany BFR exercise, although their causal contribution to hypertrophy is debated and recent work suggests the hormonal response is not strictly necessary for gains in size and strength [4,7].

A vascular and angiogenic dimension complements these anabolic mechanisms and is especially relevant to rehabilitation. The hypoxic, shear-stress environment created by restriction and reperfusion stimulates endothelial nitric oxide synthase, nitric oxide, and vascular endothelial growth factor, promoting angiogenesis and improving post-occlusive blood flow and microvascular function; these adaptations underpin reported gains in muscle perfusion, endurance, and, in some populations, endothelial and cardiovascular health [4,8]. Such vascular remodelling may be as clinically valuable as hypertrophy in patients whose functional limitation is partly circulatory, and it provides a plausible link between the local stimulus of BFR and its documented systemic effects [7,8]. It should be stressed, however, that the relative weighting of metabolic, hormonal, vascular, neural, and immune contributions remains uncertain, that they are likely to interact and to vary with the exercise mode, restriction pressure, and population, and that mechanistic findings derived from healthy young participants cannot be transferred uncritically to postoperative or medically complex patients whose baseline vascular health, medication use, and exercise tolerance differ substantially [3,4].

A distinctive and clinically relevant body of work has examined the inflammatory and immune dimension of BFR adaptation. Rather than acting through the muscle damage that characterises unaccustomed heavy or eccentric exercise, BFR appears to recruit immune cells largely through metabolic and hypoxic signalling in the relative absence of measurable structural damage [4]. Hypoxia-inducible factor-1α is activated by the ischaemic environment and contributes to increased expression of angiogenic factors, vascular endothelial growth factor, and pro-inflammatory cytokines such as interleukin-6, which in turn participate in the recruitment of neutrophils and macrophages that facilitate tissue remodelling, satellite-cell proliferation, and myofibre repair [4]. Studies consistently report elevated lactate and interleukin-6 after BFR without the increases in creatine kinase or delayed-onset soreness that would signal frank damage, supporting the interpretation that BFR modulates the immune response predominantly through metabolic stress and hypoxia rather than through mechanical injury [4]. This mechanistic profile is one reason BFR is attractive in the early post-injury and postoperative period, when the capacity to tolerate muscle damage is limited.

A further mechanistic observation with clinical resonance concerns the time course of adaptation. In conventional training, early strength gains are attributed largely to neural adaptation, with hypertrophy emerging later; several BFR studies, including work on the elbow flexors and extensors, suggest that low-load BFR may partly invert this sequence, with early gains in muscle size preceding neural adaptation and with limited change in surface electromyographic amplitude, implying that early absolute-strength gains derive substantially from hypertrophy rather than from enhanced neural drive [9]. BFR has also been shown to stimulate the proliferation of myogenic satellite cells and the addition of myonuclei, providing a cellular basis for durable hypertrophy achieved at low mechanical load [4]. These findings matter clinically because they support the use of BFR to rebuild muscle mass rapidly in the early rehabilitation window, when high-load neural and mechanical stimuli are unavailable, and they align with the observation that meaningful hypertrophy can appear within one to three weeks of high-frequency BFR training [1,9].

The neuromuscular dimension of BFR is equally pertinent to rehabilitation. The metabolite-mediated activation of group III and IV muscle afferents that accompanies BFR increases neural drive and augments motor-unit recruitment during low-load contractions, producing greater muscle excitation than low-load exercise without occlusion [3,6]. This property is of particular interest where atrophy coexists with impaired voluntary activation, as after knee surgery or in neurological disease, because it offers a route to engaging musculature that patients cannot otherwise load effectively [3]. Beyond the exercising limb, both endocrine mediators and afferent-driven central effects have been invoked to explain contralateral and systemic adaptations, a theme developed further in the sections on systemic effects and neurological rehabilitation [4,7]. It should be emphasised that the relative contribution of these mechanisms remains incompletely resolved, that much of the mechanistic evidence derives from healthy or young participants, and that mechanistic plausibility supports BFR as a load-management option rather than as proof that it should displace higher-load training once such training becomes appropriate [3].

4. Methodology of Application

Because the physiological stimulus of BFR is produced by the interaction of restriction and exercise, the methodology of application is not a technical afterthought but a determinant of both efficacy and safety [1,3]. The literature distinguishes several modes of delivery: BFR combined with resistance exercise (the most studied), BFR combined with aerobic exercise, passive BFR without concurrent exercise, and combinations of BFR with non-traditional modalities such as neuromuscular electrical stimulation [1]. This review concentrates on the resistance and, secondarily, aerobic and passive applications most relevant to rehabilitation.

4.1. Prescription of occlusion pressure

The single most important methodological principle to have emerged is that occlusion pressure should be individualised rather than fixed [1,3]. The pressure required to arrest arterial flow to a limb — the arterial or limb occlusion pressure (AOP/LOP) — depends on the individual, the limb, its circumference, blood pressure, posture, and the dimensions and material of the cuff [1,3]. A larger limb requires a greater absolute pressure to achieve the same degree of restriction, so applying a single fixed pressure across a clinical cohort produces widely different physiological stimuli and may explain some discrepancies between studies [1]. Contemporary practice therefore favours inflating the exercise cuff to the point of complete arterial occlusion (100% AOP) and then prescribing exercise at a percentage of that value; pressures of approximately 40–80% AOP have evidence supporting their efficacy, and lower, more tolerable pressures within this range often achieve adaptations comparable to higher pressures while reducing discomfort and cardiovascular strain [1,3]. Setting pressure relative to brachial systolic blood pressure is discouraged because systolic pressure correlates poorly with measured AOP and does not translate reliably from arm to leg [1]. AOP and LOP are related but not fully interchangeable reference measures, and their values are specific to the device, cuff, limb, posture, and detection method used; consequently, values obtained with one system should not be transferred uncritically to another, and reassessment is advisable when limb swelling, position, or clinical status changes materially [3].

4.2. Cuff width, material, and placement

Cuff width is a principal determinant of the pressure needed for occlusion: wider cuffs distribute force over a greater surface area and occlude at lower absolute pressures, so two cuffs of different width inflated to the same absolute pressure may produce entirely different degrees of restriction [1,3]. When pressure is prescribed as a percentage of AOP specific to the cuff used, differences attributable to cuff width and material are largely corrected, and both elastic and nylon cuffs have produced beneficial adaptations [1]. Cuffs are applied to the most proximal aspect of the limb — the proximal thigh for the lower limb and the axillary region of the upper arm for the upper limb — to maximise venous restriction at the lowest effective pressure [1,3]. A recognised caveat is that muscle growth may be attenuated directly beneath the cuff, although prescribing pressure as a percentage of AOP appears to mitigate this effect [1]. For safety and reproducibility, clinical records should document the device, cuff width and material, limb and posture assessed, method of occlusion detection, the measured individualised occlusion pressure, and the exercise pressure applied [3].

4.3. Load, volume, rest, frequency, and duration

For resistance exercise, loads of 20–40% 1RM combined with BFR consistently produce muscular adaptation; at the lower end of this range (around 20% 1RM), higher restriction pressures (approaching 80% AOP) may be needed to elicit hypertrophy [1]. A widely used volume scheme comprises 75 repetitions across four sets in a 30-15-15-15 pattern, with short inter-set rest of 30–60 seconds during which pressure is commonly maintained; alternatively, sets may be performed to concentric failure, although repetitions to failure are frequently unnecessary in clinical settings [1]. Conventional programming employs 2–3 sessions per week, while high-frequency approaches of 1–2 sessions per day may be used for short periods (1–3 weeks) to accelerate early rehabilitation [1]. Adaptations in strength and, notably, muscle mass can appear within 1–3 weeks — earlier than is typical for high-load training in the case of mass — in part because the low mechanical demand permits higher training frequency [1]. Aerobic BFR is generally applied at low intensities (below approximately 50% of maximal oxygen uptake or heart-rate reserve) during walking or cycling and can improve aerobic capacity, strength, and function, particularly in older and clinically compromised populations [1].

4.4. Passive BFR and combined modalities

Passive BFR — application of the cuff without concurrent exercise — offers a strategy for the earliest phases of rehabilitation, when the patient is bedbound or unable to perform loaded exercise [1,10]. A standard protocol applies approximately 5 minutes of restriction followed by 3 minutes of reperfusion for three to four cycles, once or twice daily, and has been used to attenuate the decline in muscle mass and strength during immobilisation and after orthopaedic surgery [1,10]. Passive application characteristically employs higher, often near-complete, occlusion pressures than exercise BFR, reflecting the absence of a concurrent contractile stimulus [1,10]. BFR has also been combined with neuromuscular electrical stimulation to activate muscle in patients unable to generate voluntary contractions, an approach of particular interest in neurological rehabilitation [1]. Across all modes, the essential clinical message is that progression — through load, pressure, and exercise complexity — should be tied to the patient's tolerance and to the clinical reason for restricting load, and that BFR should be reduced or discontinued as tolerance to conventional loading returns [3].

Table 1. Commonly reported parameters for the principal modes of blood flow restriction (BFR) training in rehabilitation, synthesised from the reviewed literature [1,3,10].

Parameter	**BFR with resistance exercise**	**BFR with aerobic exercise**	**Passive BFR (no exercise)**
External load / intensity	20–40% 1RM	< 50% VO2max or heart-rate reserve (walking, cycling)	None
Occlusion pressure	40–80% AOP (individualised)	40–80% AOP (individualised)	70–100% AOP / near-complete

Parameter	BFR with resistance exercise	BFR with aerobic exercise	Passive BFR (no exercise)
Cuff placement	Most proximal limb segment	Most proximal limb segment	Most proximal limb segment
Volume	75 reps (30-15-15-15) or sets to failure	Continuous or intervals, 5–20 min	3–4 (–5) cycles
Restriction time	~5–10 min per exercise	5–20 min	5 min per cycle
Rest between sets	30–60 s (pressure often maintained)	Continuous or intervals	3 min reperfusion between cycles
Frequency	2–3×/week; 1–2×/day for 1–3 weeks	2–3×/week; 1–2×/day for 1–3 weeks	1–2×/day during immobilisation
Typical duration	6–12 weeks (adaptation from 1–3 weeks)	≥ 6 weeks	Duration of immobilisation/bed rest

1RM — one repetition maximum; AOP — arterial occlusion pressure; VO2max — maximal oxygen uptake.

4.5. Patient selection, monitoring, and progression in practice

Translating these parameters into safe clinical practice requires a structured process of selection, application, monitoring, and progression [1,3]. Before treatment, the clinician should review the diagnosis, the phase of rehabilitation and the specific reason for restricting load, tissue-loading precautions, symptoms, and a focused medical history covering cardiovascular and thrombotic risk, vascular status, recent surgery or immobilisation, and relevant medications, using this screen to decide whether BFR may proceed, requires modification or enhanced monitoring, warrants medical consultation, or should be deferred [3]. Patients should be informed what to expect — the sensations under the cuff, the planned duration of restriction, and the symptoms that require immediate reporting — and baseline pain, swelling, neurological findings, and relevant vascular signs should be documented so that changes during treatment can be interpreted against a known starting point [3].

The cuff should be applied proximally, with its width, material, and the device kept consistent across sessions, and the exercise pressure prescribed relative to an individualised occlusion pressure rather than as an arbitrary absolute value, with reassessment when the cuff, device, limb position, or clinically relevant patient factors change [1,3]. Exercise selection and dose should reflect the rehabilitation phase and tolerance; a conservative first session using lower volume or pressure allows tolerance to be gauged in deconditioned, postoperative, or anxious patients [3]. During treatment the clinician should monitor local discomfort, pain, numbness,

tingling, unusual fatigue, dizziness or presyncope, cardiopulmonary or neurological symptoms, swelling, and signs of vascular compromise, with blood-pressure and cardiovascular monitoring in higher-risk patients, and should respond to unusual, worsening, or persistent symptoms by reducing pressure, correcting cuff position, decreasing load or volume, lengthening rest, changing the exercise, discontinuing the session, deferring further treatment, or seeking medical evaluation [3]. Progression should be driven by symptoms, tissue-healing considerations, functional goals, and increasing tolerance of conventional loading rather than by a fixed BFR duration; as higher-load resistance exercise becomes appropriate, BFR should be reduced, reserved for selected exercises, or discontinued — reinforcing that it is a phase-specific adjunct within a progressive plan rather than a permanent substitute for conventional training [3,10].

5. Efficacy in Musculoskeletal Rehabilitation: General Considerations

The clearest and most reproducible finding across the rehabilitation literature is comparative rather than absolute: low-load BFR is more effective than the same low-load exercise performed without occlusion, but it does not reliably surpass high-load resistance training [1,2]. The landmark systematic review and meta-analysis by Hughes and colleagues, examining BFR in clinical musculoskeletal rehabilitation, quantified this position [2]. Pooling data from clinical populations, low-load BFR training produced a moderate effect on muscle strength relative to low-load training alone (Hedges' g = 0.523), while heavy-load training retained an advantage over low-load BFR (Hedges' g = 0.674) [2]. The practical interpretation offered by the authors — and echoed subsequently — is that BFR is best understood as a more effective alternative to low-load training and a more tolerable surrogate for heavy loading, positioning it as a progressive tool used when heavy loads are contraindicated and abandoned in favour of conventional training once they can be tolerated [2,3].

More recent syntheses that pool across surgical and orthopaedic populations reinforce, and partially refine, this picture. A systematic review and meta-analysis of eleven randomised controlled trials involving 293 patients undergoing postoperative orthopaedic rehabilitation found that BFR was superior to standard rehabilitation for muscle strength (standardised mean difference [SMD] 0.90; 95% confidence interval [CI] 0.44–1.35) and muscle size (SMD 0.74; 95% CI 0.34–1.14), with moderate-to-high heterogeneity for strength and moderate heterogeneity for size [11]. In the same analysis, however, effects on pain (SMD 0.33; 95% CI −1.16 to 1.82) and balance (SMD −0.07; 95% CI −0.77 to 0.63) were not significant, and the certainty of evidence was rated moderate for strength and size but very low for pain and balance [11]. This divergence — robust and consistent benefits for muscle mass and strength alongside inconsistent effects on pain and balance — recurs throughout the condition-specific literature and is one of the more clinically important nuances of the evidence base [11].

Contemporary narrative and clinically oriented reviews converge on a shared framing. BFR is presented as an optional, adjunctive, or bridging low-load strategy for selected patients when higher-load strengthening is temporarily limited, to be integrated into progressive rehabilitation rather than deployed as a universal replacement for conventional resistance training [3,12]. A narrative review of BFR in postoperative musculoskeletal rehabilitation summarised the mechanistic case — metabolic stress, mTORC1 activation, hormonal responses, and, in

preclinical models, regenerative intercellular mitochondrial transfer from fibro-adipogenic progenitor cells to myocytes — while cautioning that protocol heterogeneity and the paucity of human biopsy data qualify the mechanistic claims and that individualised limb occlusion pressure is central to safe application [12]. Broader syntheses extending beyond the operative setting, including a systematic review of BFR for functionality, quality of life, and pain in neuromusculoskeletal pathologies, similarly report favourable effects on function alongside heterogeneous methodology that limits firm, condition-wide recommendations [13]. The consistent conclusion is that BFR's clinical value is contingent on matching the intervention to a defined limitation in the rehabilitation programme, and on selecting a clinically relevant comparator, rather than on any assumption of general superiority [3,11].

Two practical corollaries follow from this general position and recur throughout the condition-specific sections. First, because BFR's demonstrated strengths lie in muscle mass and strength rather than in pain or balance, it should be prescribed where preserving or rebuilding muscle is the explicit aim, and other modalities should be considered where analgesia or balance retraining is the priority [11]. Second, because the apparent efficacy of BFR depends so heavily on the comparator, the clinically meaningful question is rarely whether BFR works in the abstract but whether it adds value over the specific alternative available to a given patient at a given phase — standard care, low-load exercise, or heavy loading [2,3]. Framing the decision in these terms disciplines expectations and guards against both uncritical enthusiasm and premature dismissal; it also explains why syntheses reach seemingly divergent conclusions that are, on closer inspection, consistent once the comparator and outcome domain are specified [11,14]. The remainder of this review applies this comparative, domain-specific lens to each major clinical setting [3].

6. Anterior Cruciate Ligament Reconstruction

Rehabilitation after ACL reconstruction (ACLR) is the single most thoroughly investigated application of BFR in orthopaedics, and it exemplifies both the promise and the limitations of the field [15,14]. The clinical rationale is compelling: quadriceps atrophy and strength deficits of up to 40% relative to the contralateral limb are common after ACLR, are associated with re-injury and post-traumatic osteoarthritis, and are frequently unresponsive to conventional early rehabilitation because pain, effusion, graft-protection considerations, and arthrogenic inhibition preclude heavy loading in precisely the period when atrophy develops most rapidly [15,14]. BFR offers a means of loading the quadriceps at intensities that do not jeopardise the healing graft.

The evidence, however, is mixed and constrained by methodological weakness. A systematic review and meta-analysis of eight randomised controlled trials found that substantial heterogeneity in protocols and outcome measurement prevented meta-analysis of the primary outcomes of thigh muscle mass and strength [15]. Of five studies measuring muscle mass, three reported statistically significant findings favouring BFR, indicating that BFR was more effective than standard rehabilitation at preserving or increasing muscle mass, while two found no significant difference [15]. For strength, BFR was generally favoured, with its effectiveness appearing to depend on longer rehabilitation periods and higher occlusion pressures, but results were inconsistent [15]. Where meta-analysis of secondary, patient-reported outcomes was

possible, it favoured BFR: the International Knee Documentation Committee (IKDC) score showed a pooled mean difference of 5.90 favouring the intervention ($p = 0.01$, moderate heterogeneity, $I^2 = 49\%$), and the Lysholm score a mean difference of 6.75 [15]. Crucially, the authors judged the overall risk of bias to be moderate to high across all included studies — five of eight at high risk — with concerns spanning unclear randomisation, inability to blind participants, and selective reporting, such that any significant effects must be interpreted with caution [15].

A parallel systematic review restricted to low-load BFR after ACLR reached compatible conclusions from six randomised controlled trials involving 152 patients [14]. Two studies assessing quadriceps strength demonstrated significantly greater strength after low-load BFR than after non-BFR training; of four studies assessing quadriceps mass, two found significantly greater mass with BFR and two found no difference; three studies reported significantly less knee joint pain with BFR; and, reassuringly from a safety standpoint, the two studies that measured ACL graft laxity found no detrimental effect of BFR on graft integrity [14]. The last point is clinically important, because the theoretical concern that low-load BFR might be preferable to heavy loading precisely because it minimises mechanical stress on the maturing graft is supported, at least preliminarily, by the absence of any measurable increase in graft laxity [14]. The authors again emphasised heterogeneity of protocols and comparators and the need for standardised trials before firm recommendations [14].

Several landmark trials illustrate both the evidence and its limits. A United Kingdom National Health Service randomised controlled trial comparing low-load BFR (30% 1RM) with heavy-load resistance training (70% 1RM), both added to standard rehabilitation after ACLR over eight weeks, reported comparable gains in strength and muscle mass together with greater improvements in knee range of motion, higher physical-function scores, and lower pain in the BFR group — a pattern that captures the tolerability advantage of BFR at broadly equivalent efficacy [14,8]. The oldest and most frequently cited trial, by Ohta and colleagues, randomised 44 patients after ACLR to low-load training with or without moderate blood-flow restriction over sixteen weeks and found significantly smaller strength deficits and greater quadriceps cross-sectional area with BFR, without any adverse effect on graft laxity [15,14,8]. Work by Jack and colleagues demonstrated, using dual-energy X-ray absorptiometry, that BFR preserved lower-extremity bone and muscle mass after ACLR, while Kacin and colleagues documented significant gains in quadriceps size with a meaningful occlusion pressure relative to a low-pressure sham [15]. Countervailing results exist: an intermittent, largely passive two-week protocol reported by Iversen and colleagues did not reduce atrophy, a negative finding the authors attributed to sub-therapeutic training duration and non-individualised pressure [15,14]. Trials reporting patient-reported and functional benefits — improved Lysholm, IKDC, and KOOS scores, greater thigh circumference and isokinetic strength, and even shorter return-to-sport times — further support a role for BFR, but the inability to blind participants to a sensation-producing cuff, small samples, and heterogeneous pressures continue to temper confidence [15,8].

Comparisons across reviews highlight genuine inconsistency rather than mere imprecision. Reviews contrasting BFR with non-occlusive training in ACLR and knee osteoarthritis populations have reported conflicting evidence regarding superiority for strength and size [16],

and syntheses examining BFR both before and after ACLR describe possible benefits for muscle mass and patient-reported function without establishing a consistent additional effect on quadriceps strength [17,18]. Two considerations help reconcile these findings. First, timing matters: early postoperative protocols are constrained by pain, swelling, graft protection, and limited exercise selection, whereas later protocols increasingly compete with tolerable conventional strengthening, so the incremental benefit of BFR may be greatest in the early phase and attenuate over time [15,17]. Second, the comparator matters: BFR compared with low-load training or standard care will tend to appear favourable, whereas BFR compared with heavy-load training may show equivalence for mass but inferiority for maximal strength [14,16]. The prudent synthesis is that BFR is a defensible, low-risk adjunct after ACLR — with the strongest signal for preservation of quadriceps cross-sectional area, earlier strength recovery, and improved patient-reported function in the early postoperative weeks — but that it has not been established as a replacement for progressive conventional rehabilitation, and that decisions should be based on the patient's current loading capacity rather than on the diagnosis alone [15,14,18].

7. Knee Osteoarthritis and Non-traumatic Knee Pain

Knee osteoarthritis and non-traumatic knee pain constitute a second major application, driven by the same logic: pain and joint loading frequently reduce tolerance of higher-load exercise in a population for whom quadriceps strengthening is nonetheless a cornerstone of management [8]. Here the evidence is again promising but not uniform. A current narrative review of BFR in clinical knee problems synthesised the mechanistic and clinical literature and concluded that low-load BFR can improve muscle strength, induce hypertrophy, and enhance knee function with outcomes comparable to high-intensity resistance training when appropriate protocols are used, while safety appears acceptable under supervision [8]. The mechanistic account emphasised metabolic stress and mTOR-mediated protein synthesis, growth-factor and angiogenic responses including vascular endothelial growth factor, and analgesic effects, alongside contralateral and systemic adaptations [8].

The clinical evidence in osteoarthritis illustrates both efficacy and its boundaries. Trials of low-load BFR over programmes of six weeks or more have frequently reported gains in maximal strength and muscle mass comparable to high-intensity training and superior to low-intensity training without occlusion, together with improvements in functional performance and reductions in pain and stiffness [8]. In one representative twelve-week trial in women with radiographic knee osteoarthritis, low-load BFR produced increases in strength and mass comparable to high-intensity non-BFR training, whereas a quarter of participants in the high-intensity group withdrew because of exercise-induced pain — a finding that captures the central clinical argument for BFR in this population, namely improved tolerability at comparable efficacy [8]. Studies of shorter duration and analyses stratified by sex have, however, produced discrepant short-term results, and the analgesic effect of BFR has been variable across trials, being clearly demonstrable in some osteoarthritis cohorts yet inconsistent in broader knee-pain populations [8]. The reasonable position is therefore that BFR should be considered principally as a better-tolerated, lower-load alternative when conventional higher-load exercise is poorly

tolerated, rather than because superior effects on pain or function over conventional resistance training have been reliably established [3,8].

Individual trials give texture to this position. In a twelve-week trial by Ferraz and colleagues in women with Kellgren–Lawrence grade 2–3 knee osteoarthritis, low-load BFR produced increases in maximal strength and muscle mass comparable to high-intensity training and superior to low-intensity training without occlusion, with significant gains in functional performance and a reduction in clinical stiffness seen only in the BFR group, while a quarter of the high-intensity group withdrew because of exercise-induced pain [8]. Studies by Segal and colleagues in men and women at risk of symptomatic knee osteoarthritis produced sex-divergent short-term results — significant strength gains in women but not men after a four-week programme — a discrepancy plausibly attributable to the larger limb circumference of men combined with the use of a fixed rather than individualised pressure, which would have delivered a weaker relative stimulus [8]. Trials such as those of Bryk and colleagues and Harper and colleagues reported reduced anterior knee discomfort and improved strength, function, and pain with low-load BFR relative to comparators, reinforcing the tolerability argument, although effects on some functional and quality-of-life measures were not uniformly superior to conventional training [8]. The osteoarthritis literature therefore mirrors the general pattern: consistent strength and mass benefits, more tolerable exercise, and variable analgesic effects, with individualised pressure prescription emerging as a probable determinant of whether a given trial detects benefit [3,8].

The meniscus provides a further, instructive example. A randomised controlled trial of BFR after arthroscopic partial meniscectomy reported that adding BFR to routine rehabilitation improved quadriceps strength and thickness, thigh circumference, pain, knee function, and balance relative to routine rehabilitation, with an eight-week programme begun early after surgery [19]. This positive single-trial result should nonetheless be read against the pooled evidence, in which effects of BFR on pain and balance across postoperative populations were not statistically significant [11] — a reminder that individual trials with favourable results coexist with heterogeneous pooled estimates, and that outcome domains behave differently: gains in strength and size are more consistently demonstrable than gains in pain and balance [11,19].

8. Total Knee Arthroplasty

Total knee arthroplasty (TKA) presents an extreme version of the rehabilitation problem BFR is intended to address: quadriceps strength can fall by more than half in the first postoperative month, high-load training is frequently impossible because of pain, swelling, and surgical precautions, and the population is typically older with reduced physiological reserve [10,20]. A clinically oriented narrative review of BFR in TKA rehabilitation framed the intervention across the perioperative continuum, describing passive BFR for the bedbound early phase to attenuate atrophy and reduce pain and inflammation, aerobic BFR by walking or cycling to develop cardiorespiratory capacity when heavy loading is not yet possible, and low- or high-load BFR resistance training as tolerance improves, with progression tailored to the patient [10]. The review also foregrounded the systemic dimension particularly relevant to older TKA patients

— cardiopulmonary, endocrine, and osteoblastic effects — and stressed careful screening for contraindications given the age and comorbidity profile of this group [10].

The most developed evidence in TKA concerns prehabilitation. Preoperative BFR has been proposed and tested as a strategy to build muscular reserve before surgery so as to attenuate the inevitable postoperative decline [20,21]. A six-week preoperative BFR programme has been reported to influence pre- and postoperative skeletal muscle mass and strength in patients receiving primary TKA, supporting the mechanistic rationale that entering surgery stronger yields better postoperative trajectories [20]. Evidence for BFR initiated after arthroplasty remains more limited and protocol-dependent, and includes work examining passive BFR to counter early postoperative atrophy after total knee replacement [21]. Systematic appraisal of the TKA literature concludes that BFR — as both prehabilitation and postoperative therapy — appears safe and can improve early functional outcomes such as sit-to-stand and walking performance and attenuate loss of muscle mass, without increasing cardiovascular or wound-healing adverse events, but that the evidence base is still small, heterogeneous, and insufficient to support routine, undifferentiated use in all arthroplasty patients [10,20,21]. As elsewhere, decisions for individual patients should account for surgical status, thrombotic risk, wound and limb condition, comorbidity, and the capacity to supervise treatment [10].

9. Tendon Rehabilitation

Tendon is a comparatively recent and instructive frontier for BFR, because the classical view has been that tendon adaptation requires high mechanical loads, which are precisely what BFR avoids [22]. Conservative management of tendinopathy and rehabilitation after tendon rupture have traditionally relied on high-load resistance modalities, yet some patients tolerate high loading poorly, creating the same therapeutic gap that motivates BFR elsewhere [22,23]. The central question is therefore whether a predominantly metabolic stimulus can drive tendon adaptation, and the honest answer from the current literature is that it is unresolved.

A scoping review of nineteen studies — encompassing healthy participants and individuals with tendinopathy or tendon rupture across the Achilles, patellar, hamstring, gluteal, rotator-cuff, biceps, and lateral-elbow tendons — found contradictory results for tendon-specific structural outcomes such as tendon cross-sectional area, thickness, and stiffness [22]. In healthy participants, some studies demonstrated tendon adaptation under BFR while others did not; importantly, where adaptation occurred it generally did not differ significantly from that produced by high-load or low-load training, and the studies with positive structural findings tended to be of longer duration (around 14 weeks), consistent with the slow time course of tendon remodelling [22]. In rehabilitation settings, the structural picture was likewise mixed, but the clinical outcomes were more encouraging: across case reports, feasibility studies, and a small number of randomised trials, BFR was associated with reduced pain, increased strength, enhanced performance, and improved diagnosis-specific function, with high adherence and acceptability [22]. A randomised controlled trial in lateral elbow tendinopathy reported outcomes superior to sham low-load training for pain, function, and strength, providing one of the stronger signals in the tendon literature [22].

Two caveats temper enthusiasm. First, most tendon-rehabilitation studies are non-controlled, limiting causal inference, and structural adaptation does not map neatly onto clinical improvement [22,23]. Second, and importantly for safety, one feasibility study of BFR after non-surgically treated Achilles tendon rupture recorded serious adverse events — two Achilles re-ruptures and one deep vein thrombosis — a reminder that the favourable general safety profile of BFR does not license uncritical application in populations with fragile healing tissue [22]. A companion scoping review focused specifically on intervention parameters, physiological effects, and outcomes reached compatible conclusions, highlighting the heterogeneity of loads, pressures, and application sites and the need for standardisation before firm recommendations can be made [23]. The prudent interpretation is that BFR is a plausible adjunct in tendon rehabilitation — perhaps most useful in the early phase to permit loading at high relative intensity but low absolute load, thereby limiting load-related pain — but that its capacity to drive tendon structural adaptation, and its safety in specific ruptured-tendon populations, require further controlled investigation [22,23].

The mechanistic uncertainty is itself instructive. Tendon adapts slowly, over months rather than weeks, and the studies most likely to detect BFR-induced structural change were those of around fourteen weeks' duration, whereas many rehabilitation trials are shorter; this mismatch between intervention length and the biology of tendon remodelling may explain much of the apparent inconsistency [22]. Acute studies add a further nuance: both high-load exercise and BFR transiently reduce tendon thickness immediately and up to twenty-four hours after loading, a change attributed to fluid movement out of the tendon matrix that some regard as a marker of healthy adaptation, and BFR appears to reach fatigue with less total workload than low-load exercise, which may be advantageous when minimising cumulative tendon load is the goal [22]. Clinically, the most defensible current role for BFR in tendinopathy is in the early, irritable phase, where it permits loading at high relative intensity but low absolute load and thereby reduces load-related pain, functioning as a bridge towards the heavy, slow resistance or eccentric loading that remains the evidence-based mainstay once tolerated [22,23]. The recorded serious adverse events in a ruptured-Achilles cohort, however, mean that enthusiasm must be tempered by careful patient selection and vigilance in populations with fragile healing tissue [22].

10. Upper-limb Conditions

The upper limb has received far less attention than the knee, and the available evidence, though favourable, is sparse [24]. A systematic review of randomised controlled trials of BFR for orthopaedic conditions of the upper extremity identified only four eligible trials involving 133 patients, addressing distal radius fractures, hand osteoarthritis, and lateral elbow tendinopathy [24]. Patients treated with BFR demonstrated marginally greater strength, improved pain control, and improved patient-reported outcome measures compared with non-BFR counterparts; the benefit was most pronounced and most consistent for pain, where all four trials favoured BFR, while strength and patient-reported outcomes favoured BFR less decisively [24]. Range-of-motion outcomes did not differ between groups, and — reassuringly — no complications were reported across the included trials, all of which were judged at low risk of bias [24]. The authors appropriately cautioned that with only 133 pooled patients across

heterogeneous conditions, and with several benefits reaching statistical but uncertain clinical significance, large pathology-specific trials with standardised protocols are needed [24].

The specific conditions studied illustrate where upper-limb BFR may fit. After closed treatment of distal radius fracture, BFR added to standard therapy has been associated with greater reductions in activity-related pain and improved patient-rated wrist evaluation scores, and after operative fixation of distal radius fracture, with improved wrist flexion and extension strength, while range-of-motion recovery was similar to controls [24]. In lateral elbow tendinopathy, low-load BFR resistance training improved elbow flexor and pain-free grip strength and patient-rated tennis-elbow evaluation scores relative to a sham intervention, with the strength advantage most evident at longer follow-up [24]. In hand osteoarthritis, resistance training with BFR reduced pain but did not clearly outperform advice-plus-exercise for function [24]. These heterogeneous conditions share the same logic that motivates lower-limb BFR — a means of strengthening when pain or healing constraints limit loading — and the consistent, complication-free reduction in pain across the upper-limb trials is arguably the most reproducible finding in this small literature, even where strength and functional advantages are marginal [24].

The experimental and mechanistic literature on the upper limb is more developed than the clinical rehabilitation literature. A systematic review of BFR resistance training for the elbow, drawing on eleven higher-quality studies, concluded that low-load BFR significantly increased elbow flexor and extensor strength and muscle size; that it was superior to low-load resistance training alone over longer programmes; and that it achieved gains similar to conventional high-load training, applicable to both isometric and isotonic exercise, in healthy adults and in patients unable to perform conventional strength training such as those with HIV/AIDS or older adults [9]. This review also emphasised two mechanistically interesting observations: that early strength gains appeared attributable to early hypertrophy rather than to neural adaptation, potentially inverting the conventional sequence of training adaptation, and that contralateral cross-transfer effects were observed, whereby the unoccluded limb gained strength — a phenomenon with obvious appeal when a limb cannot be directly exercised [9]. A broader systematic review and meta-analysis of upper-extremity BFR for strength and hypertrophy similarly reported meaningful gains [25]. Taken together, the upper-limb literature indicates genuine promise, particularly for pain control and for strengthening when conventional loading is not feasible, but the clinical rehabilitation evidence remains too limited to support broad, condition-specific recommendations [24,9,25].

11. Neurological Rehabilitation

Perhaps the most striking recent expansion of BFR has been into neurological rehabilitation, a setting in which the rationale differs subtly from the orthopaedic case [26,27]. Neurological patients — after stroke or spinal cord injury, or with multiple sclerosis, cerebral palsy, or Parkinson's disease — frequently present with profound weakness, muscle atrophy, low fatigue tolerance, spasticity, and impaired motor control that make conventional moderate-to-high-load training impractical or unsafe [26,27]. BFR offers a low-load route to a robust neuromuscular stimulus, and there is mechanistic reason to expect central as well as peripheral effects, because

the intense afferent barrage generated by ischaemic, metabolite-rich muscle activity can increase corticomotor excitability and may promote neuroplastic change [27].

The most quantitatively developed evidence concerns stroke. A systematic review and meta-analysis of eleven randomised controlled trials involving 522 patients found that adding BFR to conventional rehabilitation significantly improved balance, with a mean difference in Berg Balance Scale score of 4.52 (95% CI 0.78–8.26; $p = 0.02$), and walking endurance, with a mean difference in six-minute walk test distance of 9.73 m (95% CI 7.26–12.21; $p < 0.00001$) [26]. Subgroup analyses revealed a clear time-dependence: when the intervention exceeded four weeks, BFR additionally improved lower-limb motor function, with a mean difference in the lower-extremity Fugl-Meyer Assessment of 4.38 points (95% CI 2.00–6.77), and activities of daily living, with a mean difference in the Modified Barthel Index of 10.50 points (95% CI 6.32–14.68) [26]. Motor improvements were more pronounced in patients in the subacute phase, consistent with a window of heightened neuroplasticity [26]. Importantly, functional mobility as measured by the Timed Up-and-Go test did not improve significantly, suggesting that BFR influences endurance and strength-related dimensions of walking more than agility and complex motor sequencing [26]. The authors appropriately qualified these findings by noting small samples, marked protocol heterogeneity — with compression sites, pressures ranging from 70 to 250 mmHg, and diverse co-interventions — and methodological limitations including absent blinding, such that the conclusions require confirmation in larger, higher-quality trials; a sensitivity analysis excluding two high-risk studies rendered the balance effect non-significant [26]. A further nuance is that, in almost all included trials, BFR was delivered as an adjunct to another active intervention rather than in isolation, so the current evidence chiefly reflects the added value of BFR on top of other therapy [26].

A comprehensive review of BFR across neurological conditions synthesised evidence from stroke, multiple sclerosis, spinal cord injury, cerebral palsy, and Parkinson's disease, reporting consistent gains in muscle strength and hypertrophy, improvements in gait parameters in multiple sclerosis, and, in a controlled trial in Parkinson's disease, cardiovascular and endothelial benefits alongside strength gains comparable to high-load training [27]. Mechanistically, the review emphasised metabolic stress at the muscular level and increased corticomotor excitability at the cortical level, with sustained increases in motor-evoked potential amplitude after BFR exercise, and it highlighted crossover effects to the contralateral limb [27]. No serious adverse events were reported across the neurological studies reviewed, and BFR was generally well tolerated, though the authors stressed small samples, short durations, restricted generalisability to more severely affected patients, and mechanistic gaps, calling for larger randomised trials and neuroimaging and electrophysiological work to substantiate the proposed neuroplastic effects [27]. A further systematic review and meta-analysis of BFR in neurological rehabilitation reinforces the emerging conclusion that the technique is safe, well tolerated, and capable of enhancing muscle size, strength, and function in these populations, while echoing the call for methodological standardisation [28]. The overall assessment is that neurological rehabilitation is a genuinely promising but still preliminary application, in which the combination of favourable early efficacy signals, biological plausibility for central adaptation, and an apparently benign short-term safety profile justifies further rigorous investigation rather than routine adoption [26,27,28].

The condition-specific literature synthesised in these reviews adds useful clinical detail. In multiple sclerosis, low-load and aerobic BFR programmes have improved walking speed and mobility while being well tolerated: a slow-motion, low-load resistance protocol with thigh occlusion produced markedly greater gains in gait speed than the same programme without occlusion, achieved at a lower heart-rate and perceived-exertion cost — an important consideration in a population prone to fatigue and heat sensitivity [27]. In spinal cord injury, BFR combined with neuromuscular or functional electrical stimulation has increased muscle cross-sectional area and improved fine-motor hand function in tetraplegia and quadriceps thickness in paraplegia, demonstrating that a robust hypertrophic stimulus can be delivered even where voluntary contraction is absent [27]. These applications exemplify the distinctive value of BFR in neurology: it can be coupled with passive or electrically evoked contraction to load muscle that patients cannot activate voluntarily [1,27].

In cerebral palsy, small controlled studies of trunk and lower-limb BFR training in ambulant young people reported significantly greater increases in muscle thickness than matched training without occlusion, with good tolerance and no adverse effects, suggesting a role in building the muscle bulk that is frequently deficient in this population [27]. In Parkinson's disease, a controlled trial of low-load BFR resistance training reported strength gains comparable to high-load training alongside favourable vascular and autonomic changes — improved endothelial function and reductions in blood pressure and cardiovascular risk markers not seen with high-load training — hinting that BFR may confer cardiovascular as well as neuromuscular benefit in older neurological patients [27]. Across all these conditions the same caveats recur: samples are small, interventions short, follow-up limited, more severely affected patients under-represented, and the proposed neuroplastic mechanisms not yet directly demonstrated, so the appropriate posture is cautious optimism and continued controlled investigation rather than routine clinical adoption [27,28].

12. Older Adults, Sarcopenia, and Age-related Decline

Ageing produces a progressive loss of muscle mass, strength, and function — sarcopenia — that carries substantial risks of falls, disability, and mortality, and that is compounded by the reduced sensitivity of ageing muscle to anabolic stimuli and by the frequent inability of older adults to tolerate high-load training [29,30]. This makes older populations a natural target for BFR, and the evidence here is comparatively mature, if still qualified. A systematic review and meta-analysis established that BFR training improves muscular strength and hypertrophy in older individuals, providing an early evidentiary basis for its use in this group [30]. Subsequent work has refined the picture and probed its mechanisms and limits.

A meta-analysis focused on the combination of BFR with low-intensity resistance training in post-middle-aged adults found that BFR significantly improved lower-limb muscle strength (SMD 0.76; 95% CI 0.48–1.05) but did not significantly increase muscle mass, and it identified training frequency as a key moderator: strength gains were seen with a frequency of three sessions per week but not two, and BFR produced greater strength gains than low-intensity training or normal activity while achieving gains comparable to high-intensity resistance training [31]. A scoping review of BFR protocols for elderly populations concluded that low-load BFR at 20–40% 1RM, performed two to four times weekly for six to twelve weeks at

pressures of 50–80% AOP for the lower limbs, produced meaningful improvements in strength (of the order of 15–35% in leg-press 1RM), hypertrophy (5–8% increase in quadriceps cross-sectional area), and functional performance (9–21% improvement in Timed Up-and-Go and six-minute walk tests), with outcomes comparable to, or in some cases superior to, conventional high-load training and with minimal reported adverse effects [32]. Network meta-analyses examining different BFR regimens combined with low-intensity training in older adults, and syntheses addressing health promotion in middle-aged and older women, extend these conclusions while foregrounding cardiovascular safety and the importance of individualised prescription [33,34].

A particularly candid contribution is a systematic review and meta-analysis explicitly framed as a "call to action," which searched for studies of BFR in patients who actually met contemporary diagnostic criteria for sarcopenia and found none [29]. No study of BFR training could be located in individuals classified as sarcopenic by established consensus criteria, and the only functional cut-point that could be applied — the Timed Up-and-Go — was not approached by participants in the available trials, who were healthier than the target population [29]. Nonetheless, in the four studies of BFR in older adults that could be analysed, BFR significantly improved the Timed Up-and-Go (mean difference −0.46 s), the 30-second chair-stand test (mean difference 2.78 repetitions), and knee-extension strength (SMD 0.5) relative to matched exercise without occlusion [29]. The review also drew attention to circulating biomarkers of neuromuscular junction remodelling: BFR exercise was associated with a significant decrease in C-terminal Agrin Fragment, suggesting less neuromuscular junction degradation and muscle wasting — a mechanistically encouraging finding for a population defined partly by neuromuscular deterioration [29]. The unavoidable conclusion is that although BFR demonstrably improves functional performance and strength in older adults, and although its mechanistic profile is well suited to counteracting sarcopenic pathophysiology, direct evidence in diagnosed sarcopenia is absent, and the field urgently needs studies in patients below established functional cut-points [29].

13. Systemic Effects Beyond the Exercising Limb

A defining feature of BFR that distinguishes it from most local rehabilitation modalities is that its effects are not confined to the muscle beneath the cuff. A systematic review of the systemic effects of BFR training synthesised thirty-five studies across cardiopulmonary, musculoskeletal, endocrine, and psychosocial domains and concluded that, when appropriately dosed, BFR produces favourable or non-detrimental systemic effects without adverse outcomes attributable to the technique itself [7]. This systemic reach is a double-edged property: it broadens the potential clinical value of BFR while simultaneously demanding attention to safety in vulnerable systems.

On the cardiopulmonary system, BFR at occlusion pressures below approximately 200 mmHg does not produce detrimental blood-pressure responses in healthy adults; rather, blood-pressure responses resemble those of conventional exercise when prescribed appropriately [7]. Cardiac output is generally maintained during BFR because a rise in heart rate offsets a reduction in stroke volume, and maximal oxygen uptake can improve — sometimes with lower training volume than conventional exercise — which is clinically attractive for patients unable to sustain

higher intensities [7]. Notably, appropriately dosed BFR combined with aerobic exercise improved functional exercise capacity in medically complex populations, including significantly greater improvement in six-minute walk distance in patients with end-stage renal disease undergoing haemodialysis and improvements in anaerobic threshold in patients with chronic heart failure, in both cases without reported adverse effects — early but encouraging signals that BFR can be applied safely in selected high-risk groups under supervision [7].

The osteoblastic dimension deserves emphasis in a rehabilitation context that frequently involves older and immobilised patients at risk of bone loss. Low-load BFR combined with resistance exercise has been associated with increases in bone alkaline phosphatase and a shift in bone-turnover markers towards formation, comparable to high-intensity training, indicating that osteoblastic activity can be stimulated without the heavy mechanical loading conventionally deemed necessary for bone health [7]. In the arthroplasty setting this is of particular interest, since enhanced osteoblastic activity could theoretically support bone–prosthesis consolidation and mitigate periprosthetic bone loss, although direct evidence remains lacking [10]. Systemic metabolic effects extend the potential reach of BFR still further: beyond musculoskeletal outcomes, acute BFR resistance exercise has been shown to lower circulating free fatty acids in obese individuals more than matched non-occluded exercise, an effect linked to localised hypoxia and to vasodilatory and angiogenic signalling, positioning BFR as a candidate adjunct for metabolic as well as musculoskeletal rehabilitation [35]. Taken together with its cardiopulmonary and endocrine effects, this systemic profile suggests that BFR may deliver ancillary benefits — to bone, vasculature, and metabolism — that are especially valuable in the multimorbid, deconditioned patients who most often require load-sparing rehabilitation, provided that the same systemic potency is respected in screening and monitoring [7,10,35].

Musculoskeletal effects extend beyond the occluded limb through contralateral and distal "cross-transfer" phenomena, whereby occlusion of one limb is associated with strength or hypertrophy adaptations in the unoccluded limb, attributed variously to neuromuscular adaptation and to circulating hormonal and metabolic mediators [7]. Endocrine responses include acute elevations in growth hormone and, in some studies, testosterone, together with a decrease in myostatin gene expression and increases in inhibitors of myostatin such as GASP-1 and SMAD-7, all of which favour muscle growth; however, other studies have found no significant change in interleukin-6, insulin-like growth factor 1, or free testosterone, so the hormonal contribution to adaptation remains debated and is unlikely to be the sole mechanism [7]. BFR combined with low-intensity training has also been associated with favourable shifts in bone turnover markers, including increased bone alkaline phosphatase, suggesting an osteoblastic effect achievable without the mechanical loading conventionally thought necessary for bone health — a potentially valuable property in osteopenic and elderly populations [7]. Metabolic stress, reflected in elevated blood lactate and noradrenaline, is consistently greater with BFR than with load-matched exercise and is central to the anabolic response, and it aligns with mechanistic accounts of immune-cell recruitment through hypoxia and metabolite accumulation rather than muscle damage [4,7].

The psychosocial dimension is more equivocal. Ratings of perceived exertion are typically elevated when BFR is introduced, which may initially deter patients, but this heightened

perceived effort tends to diminish over weeks of training, and intermittent BFR is better tolerated than continuous BFR [7]. Some studies report acute negative effects on mood state and increased fatigue immediately after BFR, comparable in magnitude to high-intensity exercise, which argues against its use immediately before performance but is of limited concern in a rehabilitation context [7]. Pain and discomfort ratings vary with cuff width, pressure, and body region. The overall conclusion of the systemic-effects literature is that BFR has wide-reaching but generally favourable or neutral effects across body systems, with no adverse outcomes directly attributable to the technique when dosed properly, although the authors appropriately noted that dosing heterogeneity, the predominance of young healthy samples, and potential conflicts of interest in parts of the foundational literature warrant caution and further study [7].

14. Recovery, Athletic, and Adjacent Metabolic Applications

Although this review is oriented towards rehabilitation, several adjacent applications illuminate the versatility and the limits of BFR. The use of BFR as a post-exercise recovery strategy has attracted interest but rests on a thin and inconsistent evidence base. A systematic review identified only eleven studies of BFR as a post-exercise recovery intervention, most applying passive BFR in a supine position to the proximal thigh, with cuffs typically inflated to around 220 mmHg or 50 mmHg above systolic blood pressure in protocols of three cycles of five minutes' occlusion and reperfusion [36]. Some studies reported beneficial effects on markers of muscle damage, perceived recovery, and subsequent performance, while others found no positive or even detrimental effects, and there was a pervasive lack of standardisation in the number of cycles and occlusion-reperfusion durations [36]. The measured conclusion is that BFR may be a potential post-exercise recovery strategy but that practitioners should apply it with caution given the scarcity and inconsistency of evidence, particularly the near-absence of data in team and individual sports and in female populations [36].

In competitive athletes, technical reviews emphasise that BFR is most defensibly used as a complement to, rather than a replacement for, conventional resistance training [37]. The available evidence indicates that combining conventional heavy-load training with low-load BFR can optimise adaptations, that occlusion pressure should be individualised as a percentage of complete arterial restriction (commonly 50–80%), and that cuff width and material influence the required pressure; it also flags a specific caveat that muscle growth may be attenuated directly beneath the cuff, reinforcing the argument that BFR should not be the sole training stimulus [37]. Applied studies in athletic populations support a role for BFR as an adjunct: an eight-week programme of BFR combined with moderate-intensity resistance training in male collegiate footballers improved isokinetic knee flexor and extensor strength, vertical jump, sprint, and change-of-direction performance more than traditional high-intensity training, and favourably modulated circulating vascular factors, increasing nitric oxide and vascular endothelial growth factor and decreasing endothelin — evidence of vascular as well as neuromuscular adaptation [38]. These vascular and metabolic effects hint at applications beyond musculoskeletal rehabilitation: acute BFR resistance exercise has been shown to reduce plasma free fatty acids in obese individuals more than matched resistance exercise, an effect linked to the localised hypoxia of BFR and to vasodilatory and angiogenic responses,

positioning BFR as a candidate hypoxic-training modality for metabolic health [35]. Quantitatively, a meta-analysis of twenty-five studies confirmed that BFR training exerts a positive, medium-sized effect on muscular strength (Cohen's d 0.558), with exploratory moderator analyses suggesting larger effect sizes with longer programmes (beyond eight weeks), higher relative loads, lower-body exercise, automated cuffs, and higher pressures, although formal between-subgroup differences were not statistically significant [39].

The athletic literature also clarifies what BFR is not. It is distinct from ischaemic preconditioning — brief cycles of complete occlusion and reperfusion applied before exercise to enhance subsequent performance — which shares the tourniquet but differs in aim and physiology, and which has been examined chiefly as an ergogenic rather than a rehabilitative tool [5]. In trained athletes, BFR combined with sprint or endurance work has been used to intensify the hypoxic stimulus without compromising training load, for example by applying restriction during the rest periods of repeated-sprint exercise, and evolving protocols increasingly individualise pressure and combine BFR with systemic hypoxia to deepen the muscular stimulus [6]. These developments, while oriented towards performance, feed back into rehabilitation by refining the understanding of dose, timing, and individualisation that clinical application also requires [6,5]. For the rehabilitation practitioner, the salient message from the athletic and recovery literature is one of measured caution: BFR is a versatile tool whose recovery and performance applications remain less well substantiated than its rehabilitative use, and its adoption should track the strength of evidence for each specific purpose rather than its general popularity [36,37].

BFR sits within a broader family of low-load and alternative modalities employed when high-intensity exercise is contraindicated, and the boundaries of the recovery toolkit are relevant to clinical decision-making. Manual sports massage, for example, is an established recovery adjunct that reduces perceived muscle soreness after strenuous or eccentric exercise, although it does not appear to restore muscle function, illustrating the general principle that recovery interventions often modulate symptoms more than performance [40]. Similarly, isometric training has emerged as a low-barrier modality with a favourable safety profile for patients who cannot tolerate high dynamic loads, with evidence that it lowers resting blood pressure and may modestly modulate cardiac autonomic balance — a reminder that BFR is one option among several low-load strategies, and that its selection should be guided by the specific therapeutic goal and the patient's tolerance rather than by novelty alone [41]. The comparative framing matters because it discourages the reflexive substitution of BFR for well-established, lower-technology approaches when the latter are equally suited to the clinical aim [3,41].

15. Safety, Risk Stratification, and Contraindications

Safety is the pivot on which the clinical acceptability of BFR turns, and the literature supports a nuanced rather than a reassuring-or-alarming verdict. Correctly implemented, BFR appears to present no greater risk than traditional exercise, and large surveys and the accumulated trial literature record a low incidence of serious events [1,7]. Nonetheless, adverse-event reporting across trials is frequently incomplete or absent, medically complex patients are under-represented, and the technique should not be described as risk-free even where serious events are uncommon [3]. Commonly reported symptoms are usually transient or low-grade and

include discomfort or a dull ache, acute muscle fatigue, delayed-onset soreness, tingling or paraesthesia, numbness, dizziness or presyncope, bruising, and subcutaneous haemorrhage [1,3]. Potentially serious complications described in the literature — although rare — include venous thromboembolism, pulmonary embolism, syncope, persistent neurological symptoms, excessive muscle damage, and rhabdomyolysis, and, as noted in the tendon literature, a feasibility study recorded two Achilles re-ruptures and one deep vein thrombosis, underscoring that population and healing-tissue context modulate risk [3,22].

Cardiovascular and haemodynamic considerations are central because, despite the low external loads, BFR alters heart rate, blood pressure, cardiac output, stroke volume, and related variables [1,7]. Continuous BFR, in which pressure is maintained through rest intervals, tends to increase heart rate and systolic and diastolic pressure relative to the same exercise in free-flow conditions, whereas intermittent application, with deflation between sets, mitigates these differences; cardiac output is generally preserved through a compensatory rise in heart rate as stroke volume falls [1,7]. Higher relative pressures produce larger cardiovascular responses and may increase risk, which is one of the practical arguments for using the lowest effective pressure and for individualising to AOP [1]. Consequently, patients with poorly controlled hypertension, heart failure, vascular disease, or uncertain cardiovascular status may require medical consultation, closer supervision, blood-pressure monitoring during initial sessions, or an alternative intervention [3,7]. Cardiovascular disease should not, however, be treated as a single uniform absolute contraindication, since selected cardiovascular populations have derived strength, functional, and even cardiovascular benefit from carefully applied low-load BFR [7].

Thrombotic risk warrants particular scrutiny because BFR involves deliberate venous occlusion, yet the empirical picture is reassuring within its limits. Acute studies have generally found no significant increase in coagulation markers such as D-dimer after BFR, and chronic studies, including one using duplex ultrasonography after knee surgery, have found no evidence of thrombus formation; a large Japanese survey reported an incidence of deep vein thrombosis of 0.055% and of pulmonary embolism of 0.008%, and BFR has been shown to upregulate the fibrinolytic system [1]. These data suggest that the thrombotic risk of BFR per se is low; however, the baseline risk of venous thromboembolism may already be elevated after surgery or prolonged immobilisation, so a history of venous thromboembolism, impaired coagulation, marked unilateral swelling, or uncertain thrombotic risk may justify physician consultation before BFR is considered [1,3]. Rhabdomyolysis has been reported in isolated case reports, but analysis of the published literature places its incidence very low (of the order of 0.07–0.2%, and as low as 0.008% in the large Japanese survey), comparable to or not clearly exceeding that of traditional exercise; predisposing factors such as recent inactivity, infection, or unaccustomed exertion should be excluded, and easing patients into training while monitoring indirect markers of muscle damage is prudent [1]. Concerns about oxidative stress and muscle damage are largely allayed by evidence that the addition of BFR to low-load exercise does not augment oxidative stress or muscle damage beyond that of the low-load exercise itself, with such responses appearing load-dependent rather than BFR-dependent [1].

A recurring safety theme is that how pressure is applied matters as much as whether BFR is used at all. Because a wider cuff occludes at a lower absolute pressure and a narrow cuff requires a higher one, applying a single fixed absolute pressure across patients delivers an inconsistent

and sometimes excessive stimulus, and higher relative pressures are associated with greater cardiovascular responses and more discomfort without proportionate gains in adaptation [1]. Prescribing pressure as a percentage of individually measured arterial occlusion pressure — and preferring the lowest effective pressure and, where practicable, wider cuffs — therefore serves both efficacy and safety, reducing the risk of complete arterial occlusion, undue haemodynamic stress, and the numbness and paraesthesia that follow nerve compression [1,3]. Device variability compounds this: different BFR systems and cuffs yield different occlusion-pressure values and should not be treated as interchangeable, and measured values are further influenced by posture and limb position, so reassessment is warranted whenever the device, cuff, limb, or the patient's swelling or status changes [3]. These considerations reframe several apparent risks of BFR as risks of poor technique rather than of the modality itself, and they explain why individualisation and documentation are presented across the literature not merely as best practice but as the principal means by which the physiological potency of BFR is rendered safe [1,3].

Screening and risk stratification are therefore the operative safeguards. Pre-participation screening should review medical history, current symptoms, comorbidities, recent surgery or immobilisation, cardiovascular and vascular status, thrombotic history, and medications affecting cardiovascular or coagulation risk, and should determine whether BFR can proceed, requires modification or enhanced monitoring, needs medical consultation, or should be deferred [3]. A range of conditions has been proposed as warranting caution or deferral, including cardiovascular disease, poorly controlled hypertension, peripheral vascular disease, impaired circulation, diabetes mellitus, chronic kidney disease, clinically relevant inflammatory disease, coagulation disorders and previous venous thromboembolism, pregnancy or the postpartum period, recent surgery, prolonged immobilisation, lymphoedema or marked swelling, and medication- or substance-related prothrombotic factors, together with sickle-cell disease, active local infection, open wounds under or near the cuff, and malignancy [3]. It is important to recognise, however, that these are not a validated list of absolute contraindications: published risk-stratification frameworks offer a practical structure for weighing baseline health status and condition-specific risk, but they support rather than replace clinical judgement, shared decision-making, and reassessment when the patient's status changes [3]. A pragmatic decision pathway distinguishes patients who may proceed with routine monitoring, those requiring a modified protocol or enhanced observation, those who should obtain medical clearance, and those in whom treatment should be deferred [3]. Finally, clear stopping criteria — unusual or escalating pain, persistent or worsening numbness or paraesthesia, dizziness or presyncope, cardiopulmonary or new neurological symptoms, excessive swelling, or signs of vascular compromise — should complement pre-participation assessment, and treatment should be delivered under appropriate supervision with individualised occlusion pressure, documented parameters, and reassessment when swelling, position, or clinical status changes [1,3].

16. Summary of Representative Evidence

Table 2 summarises representative studies across the principal domains, illustrating both the breadth of the evidence and the recurring dependence of conclusions on design, comparator, and outcome domain.

Table 2. Representative studies of blood flow restriction (BFR) training in rehabilitation.

Source [ref]	Design	Population / focus	Principal findings
Patterson et al. [1]	Expert review	Methodology and safety	Individualised pressure (40–80% AOP); loads 20–40% 1RM; favourable safety with appropriate dosing
Hughes et al. [2]	Systematic review + meta-analysis	Clinical musculoskeletal rehabilitation	LL-BFR > LL alone for strength (g = 0.523); HL > LL-BFR (g = 0.674)
Miller et al. [7]	Systematic review (35 studies)	Systemic effects	Favourable/neutral cardiovascular, endocrine, musculoskeletal, psychosocial effects; no adverse effects attributable to BFR when dosed properly
Alamri et al. [11]	Systematic review + meta-analysis (11 RCTs, 293 patients)	Postoperative orthopaedic	Strength SMD 0.90 and size SMD 0.74 favouring BFR; pain and balance non-significant
Butt and Ahmed [15]	Systematic review + meta-analysis (8 RCTs)	ACL reconstruction	Muscle mass significant in 3/5 studies; IKDC +5.90, Lysholm +6.75 favouring BFR; moderate-to-high risk of bias

Source [ref]	Design	Population / focus	Principal findings
Koc et al. [14]	Systematic review (6 RCTs, 152 patients)	Low-load BFR after ACLR	Greater quadriceps strength, mass, and less pain in several studies; no adverse effect on graft laxity
Kwon et al. [8]	Narrative review	Clinical knee problems (ACL, osteoarthritis)	LL-BFR improves strength, hypertrophy, function comparable to high-intensity training; variable analgesia
Ke et al. [19]	Randomised controlled trial	Arthroscopic partial meniscectomy	BFR improved quadriceps strength, thickness, pain, knee function, and balance vs routine rehabilitation
Öberg et al. [22]	Scoping review (19 studies)	Tendon adaptation and rehabilitation	Contradictory tendon-structure findings; reduced pain, increased strength/function; three serious adverse events in one study
Johnson et al. [24]	Systematic review (4 RCTs, 133 patients)	Upper-extremity orthopaedic conditions	Greater strength, better pain control and PROMs favouring BFR; no complications
Zhang et al. [26]	Systematic review + meta-analysis (11 RCTs, 522 patients)	Stroke, lower-limb dysfunction	Berg Balance +4.52; six-minute walk +9.73 m; motor and ADL gains when intervention > 4 weeks

Source [ref]	Design	Population / focus	Principal findings
Darwish et al. [27]	Comprehensive review	Neurological rehabilitation	Gains in strength/hypertrophy; increased corticomotor excitability; no serious adverse events
Cahalin et al. [29]	Systematic review + meta-analysis	Older adults / sarcopenia	Improved Timed Up-and-Go, chair stand, knee-extension strength; no studies in diagnosed sarcopenia
Chang et al. [31]	Meta-analysis (11 RCTs, 325 participants)	Post-middle-aged adults	Lower-limb strength SMD 0.76; frequency (3×/week) a key moderator; mass not significantly increased
Gear et al. [39]	Systematic review + meta-analysis (25 studies)	Muscular strength	Positive medium effect on strength (d = 0.558); moderators explored but not statistically distinct

ACL — anterior cruciate ligament; ACLR — ACL reconstruction; ADL — activities of daily living; AOP — arterial occlusion pressure; HL — high-load; IKDC — International Knee Documentation Committee; LL — low-load; PROMs — patient-reported outcome measures; RCT — randomised controlled trial; SMD — standardised mean difference.

17. Discussion

Read as a whole, the literature supports a consistent and clinically usable position while resisting the stronger claims that sometimes accompany enthusiasm for BFR. The most reproducible conclusion, spanning meta-analyses in mixed clinical populations, postoperative orthopaedic cohorts, older adults, and specific conditions, is comparative: low-load BFR reliably outperforms load-matched exercise without occlusion, approaches high-load resistance training for hypertrophy, and generally remains inferior to heavy loading for maximal strength [1,2,11,31,39]. This triangulation has a direct clinical corollary. Where the goal is to preserve

or rebuild muscle mass and function under a load ceiling imposed by pain, surgery, graft protection, or frailty, BFR is a rational and evidence-supported choice; where heavy loading is tolerable and maximal strength is the priority, it offers no advantage and should yield to conventional training [2,3]. BFR is thus best conceived not as a superior form of exercise but as a load-management tool with a defined therapeutic niche.

A second, more subtle pattern is the divergence between outcome domains. Across the strongest syntheses, gains in muscle strength and size are demonstrated with reasonable consistency, whereas effects on pain and balance are inconsistent or non-significant when pooled [11,26]. The postoperative orthopaedic meta-analysis rated certainty as moderate for strength and size but very low for pain and balance [11]; the stroke meta-analysis found robust improvements in walking endurance but no significant effect on the agility-dependent Timed Up-and-Go [26]; and individual trials reporting pain relief coexist with heterogeneous pooled estimates [11,8,19]. Clinicians should therefore calibrate expectations by domain: BFR is a muscle-mass and strength intervention first, with analgesic and balance benefits that are plausible but variable and condition-dependent. This distinction also cautions against selecting BFR primarily for pain relief in the absence of a strengthening rationale.

Third, the reviewed evidence repeatedly demonstrates that conclusions depend on the comparator and on timing, which reconciles much of the apparent inconsistency between reviews. In ACL reconstruction, syntheses reach conflicting verdicts partly because some compare BFR with standard care or low-load exercise, against which it looks favourable, while others compare it with heavy loading, against which it appears equivalent for mass but weaker for strength [15,14,16]. Timing compounds this: the incremental value of BFR is greatest early after injury or surgery, when heavy loading is impossible, and attenuates as conventional strengthening becomes tolerable [15,17]. The neurological literature adds a temporal dimension of its own — benefits for motor function and activities of daily living emerged only when interventions exceeded four weeks, and were greatest in the subacute phase — implying that dose and timing are not incidental but decisive [26]. These observations argue for individualised prescription anchored to the patient's current loading capacity and phase of recovery rather than to diagnosis alone [3,15].

Fourth, the mechanistic coherence of BFR strengthens confidence in its clinical effects while leaving important gaps. The convergence of metabolic stress, fast-twitch recruitment, mTORC1 signalling, cell swelling, and hypoxia-driven immune and angiogenic responses provides a plausible account of adaptation without frank muscle damage, which is precisely why BFR is attractive in early rehabilitation [1,4,6,7]. The neuromuscular and central effects — increased motor-unit recruitment, corticomotor excitability, and cross-transfer to the unoccluded limb — extend the rationale into neurological rehabilitation and into situations where a limb cannot be directly exercised [9,27]. Yet much mechanistic evidence derives from healthy, young participants; human tendon-biopsy data are lacking; and the proposed neuroplastic effects in neurological populations remain to be substantiated with imaging and electrophysiology [12,22,27]. Mechanistic plausibility, in short, justifies BFR as a load-management strategy but does not by itself establish superiority or generalisability to medically complex patients.

It is worth making explicit how the musculoskeletal and neurological rationales differ, because the distinction shapes expectations. In orthopaedic rehabilitation, BFR is principally a load-substitution strategy: it supplies an anabolic and neuromuscular stimulus when mechanical loading is unsafe, and it is expected to be superseded by heavy loading once tissue tolerance permits [2,3]. In neurological rehabilitation, by contrast, the constraint is often not the fragility of tissue but the patient's inability to generate or tolerate high voluntary loads at all, so BFR — particularly when combined with electrical stimulation or aerobic work — functions less as a temporary bridge and more as an enabling modality, and its proposed central, corticomotor effects raise the possibility of benefits that conventional high-load training could not deliver [26,27]. This difference explains why the neurological evidence emphasises dose and timing — benefits emerging only beyond four weeks and greatest in the subacute phase — whereas the orthopaedic evidence emphasises comparator and phase of healing [15,26]. In both settings, however, the unifying lesson is that BFR is not a monolithic intervention with a single expected effect, but a parameterised technique whose value depends on matching pressure, load, mode, dose, and timing to a clearly defined clinical goal [3,26].

The safety literature invites a similarly balanced reading. The weight of evidence — low rates of thromboembolism, no augmentation of muscle damage or oxidative stress beyond that of low-load exercise, rare rhabdomyolysis, and favourable or neutral systemic effects — supports the view that correctly applied BFR is no more hazardous than conventional exercise, and can even be applied to selected high-risk cardiovascular and renal patients under supervision [1,7]. At the same time, serious events are documented, adverse-event reporting is frequently inadequate, and medically complex patients are under-represented, so BFR cannot honestly be called risk-free [3,22]. The reconciliation is procedural rather than categorical: individualised occlusion pressure, structured screening and risk stratification, appropriate supervision, and clear stopping criteria convert a technique with genuine physiological potency into one with an acceptable risk profile for its intended populations [1,3].

A further point of synthesis concerns the population in whom BFR is most likely to justify its added complexity. The technique demands equipment, individualised pressure assessment, screening, and monitoring, and it introduces sensations and, occasionally, discomfort that conventional low-load exercise does not. This overhead is most clearly warranted where a genuine load ceiling exists — the early postoperative knee, the painful arthritic joint, the frail or sarcopenic older adult, the neurologically impaired limb — and where the alternative is either ineffective low-load exercise or no loading at all [2,3]. Conversely, in patients who can already tolerate progressive conventional loading, the marginal benefit of BFR narrows and the added complexity becomes harder to justify, which is why the literature repeatedly frames BFR as a bridging or adjunctive strategy rather than a default [3]. Seen this way, debates over whether BFR is superior to heavy loading are somewhat beside the point: its distinctive clinical value lies precisely in the window where heavy loading is unavailable, and its success should be measured by how well it fills that window rather than by whether it can replace a modality that is, in its proper context, already effective [2,3].

Finally, the field's principal weakness is not a lack of positive findings but a lack of standardisation. Protocol heterogeneity — in pressure prescription, cuff characteristics, load, volume, frequency, duration, and comparator — recurs as the limiting factor in virtually every

synthesis, frequently preventing meta-analysis of primary outcomes and inflating heterogeneity when pooling is attempted [15,14,26]. The corollary is that the most valuable contributions to the field at present may be methodological: consensus on individualised pressure prescription and reporting, standardisation of protocols for specific conditions, and adequately powered, well-reported randomised trials with meaningful comparators and longer follow-up [3,12]. This is also the point at which the clinically oriented reviews converge with the primary-trial literature, and it is where the practical guidance for practitioners is clearest — namely, to individualise, to document, to monitor, and to integrate BFR into a progressive plan rather than to treat it as a standalone solution [3].

18. Limitations of the Evidence Base

Several limitations qualify the conclusions of this review and of the literature it synthesises. First, protocol heterogeneity is pervasive: differences in occlusion pressure and its method of prescription, cuff width and material, exercise mode, load, volume, frequency, duration, and comparator complicate direct comparison and frequently preclude meta-analysis of primary outcomes [15,14,26]. Second, many primary studies are small, and randomised trials in this field are commonly rated at moderate-to-high risk of bias, chiefly because participants cannot be blinded to an intervention involving a visible, sensation-producing cuff, and because randomisation and allocation procedures are often unclear [15,14]. Third, adverse-event reporting is inconsistent and frequently absent, so the true incidence of uncommon but serious events — particularly in postoperative, older, or medically complex patients — cannot be estimated with confidence [3,22]. Fourth, medically complex patients, including those with significant cardiovascular, renal, or coagulation disorders, are under-represented, and there is a conspicuous absence of studies in patients who meet contemporary diagnostic criteria for sarcopenia, despite the mechanistic suitability of BFR for that population [29]. Fifth, mechanistic evidence derives largely from healthy or young participants, human tendon-biopsy data are lacking, and the neuroplastic mechanisms proposed in neurological rehabilitation await direct confirmation [12,22,27]. Sixth, long-term follow-up is scarce, so the durability of gains and the persistence or emergence of adverse effects over extended periods remain poorly characterised [3]. Finally, this article is a narrative review: source selection and interpretation were not systematic, no formal risk-of-bias appraisal or quantitative synthesis was undertaken, and the review is therefore subject to selection and emphasis bias; its conclusions should be read as a clinically reasoned overview rather than a formally graded account of all available evidence.

18.1. Priorities for future research

Several priorities emerge from this synthesis. Methodologically, the field needs consensus on the prescription and reporting of occlusion pressure — including the device, cuff dimensions, limb, posture, detection method, and the exercise pressure applied — so that studies become comparable and clinicians can reproduce effective protocols [3]. Condition-specific standardisation of load, volume, frequency, duration, and comparator would reduce the heterogeneity that currently prevents meta-analysis of primary outcomes and inflates statistical heterogeneity when pooling is attempted [15,26]. Clinically, adequately powered randomised trials with meaningful comparators, blinded outcome assessment where feasible, standardised

and consistently reported adverse-event surveillance, and follow-up beyond the immediate training period are required to establish durability and to quantify uncommon serious events [3,12]. Populations currently under-represented — patients meeting diagnostic criteria for sarcopenia, and those with significant cardiovascular, renal, or coagulation disorders — should be studied directly rather than by extrapolation, given the strong mechanistic case for benefit in exactly these groups [29]. Mechanistically, human tissue studies, including tendon biopsy, and neurophysiological and neuroimaging work in neurological populations are needed to substantiate the pathways inferred from preclinical and healthy-participant data [12,22,27]. Finally, formal health-economic evaluation is almost entirely absent, yet it will be decisive for the routine integration of BFR into rehabilitation services [12].

19. Conclusions

Blood flow restriction training is a mechanistically grounded, low-load intervention that provides a meaningful anabolic and neuromuscular stimulus at external loads too low to threaten healing or vulnerable tissue, and it has become a valuable adjunct across musculoskeletal and neurological rehabilitation. On the basis of the evidence reviewed, the following principal conclusions are offered.

First, BFR reliably outperforms load-matched exercise without occlusion and approaches high-load resistance training for hypertrophy, but does not consistently match heavy loading for maximal strength; it should therefore be used as a load-management adjunct or bridging strategy when higher loads are temporarily contraindicated, and progressively replaced by conventional training as tolerance returns.

Second, the benefits of BFR are domain-specific: gains in muscle mass and strength are demonstrated with reasonable consistency, whereas effects on pain and balance are variable and condition-dependent, so the technique should be selected primarily for a strengthening or mass-preservation rationale and not chiefly for analgesia.

Third, the evidence is most developed for ACL reconstruction, knee osteoarthritis and non-traumatic knee pain, knee arthroplasty, tendon rehabilitation, upper-limb conditions, older adults, and, increasingly, neurological rehabilitation; across these settings, conclusions depend heavily on the comparator and on the timing and dose of the intervention, with the greatest incremental value early after injury or surgery and, in stroke, with programmes exceeding four weeks.

Fourth, BFR exerts favourable or neutral systemic effects — cardiopulmonary, endocrine, osteoblastic, and neuromuscular — that extend its potential value but also require attention to safety in vulnerable systems.

Fifth, when delivered with individualised occlusion pressure, appropriate screening and risk stratification, supervision, documentation, and clear stopping criteria, the short-term safety profile of BFR is favourable and comparable to that of conventional exercise, although serious events are documented, adverse-event reporting is incomplete, and medically complex patients remain under-represented; BFR should not be described as risk-free.

Sixth, the principal barrier to stronger conclusions is not a shortage of positive findings but a shortage of standardisation; the field's most pressing needs are consensus on pressure prescription and reporting, standardised condition-specific protocols, adequately powered and well-reported randomised trials with meaningful comparators and longer follow-up, studies in medically complex and sarcopenic patients, and mechanistic work — including human tissue and neurophysiological studies — to substantiate the proposed pathways. Until these needs are met, BFR is best integrated into progressive, individualised rehabilitation as an optional component whose prescription reflects patient risk, tolerance, rehabilitation goals, and the certainty of the available evidence.

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References

[1] Patterson SD, Hughes L, Warmington S, Burr J, Scott BR, Owens J, Abe T, Nielsen JL, Libardi CA, Laurentino G, Neto GR, Brandner C, Martin-Hernandez J, Loenneke J. Blood flow

restriction exercise: considerations of methodology, application, and safety. Front Physiol. 2019;10:533. https://doi.org/10.3389/fphys.2019.00533

[2] Hughes L, Paton B, Rosenblatt B, Gissane C, Patterson SD. Blood flow restriction training in clinical musculoskeletal rehabilitation: a systematic review and meta-analysis. Br J Sports Med. 2017;51(13):1003–1011. https://doi.org/10.1136/bjsports-2016-097071

[3] Ziębka W, Dziewięcka O, Błażuk M, Zając D, Broda D, Mikulska W, Sak N, Tasior K, Sikora K, Wilanowska J, Broda A. Blood flow restriction training in musculoskeletal rehabilitation: clinical applications, safety, risk stratification, and practical implementation – a narrative review. Qual Sport. 2026;65:73916. https://doi.org/10.12775/QS.2026.65.73916

[4] Rossi FE, de Freitas MC, Zanchi NE, Lira FS, Cholewa JM. The role of inflammation and immune cells in blood flow restriction training adaptation: a review. Front Physiol. 2018;9:1376. https://doi.org/10.3389/fphys.2018.01376

[5] Patterson SD, Burr JF, Warmington S. Editorial: blood flow restriction: rehabilitation to performance. Front Physiol. 2021;12:566421. https://doi.org/10.3389/fphys.2021.566421

[6] Hurr C. Evolving blood flow restriction training. Exp Physiol. 2025;110(6):781–783. https://doi.org/10.1113/EP092255

[7] Miller BC, Tirko AW, Shipe JM, Sumeriski OR, Moran K. The systemic effects of blood flow restriction training: a systematic review. Int J Sports Phys Ther. 2021;16(4):978–990. https://doi.org/10.26603/001c.25791

[8] Kwon S, Bae KC, Yon CJ, Kim DH. Current narrative review—application of blood flow restriction exercise in clinical knee problems. Medicina (Kaunas). 2025;61(8):1377. https://doi.org/10.3390/medicina61081377

[9] Xu X, Liu Q, Peng L. Effect of blood flow restriction resistance training on elbow joint muscle strength. Qual Sport. 2024;28:56999. https://doi.org/10.12775/QS.2024.28.56999

[10] De Renty C, Forelli F, Mazeas J, Kakavas G, Hewett TE, Korakakis V. Knee loading with blood flow restriction can enhance recovery after total knee arthroplasty. Cureus. 2023;15(4):e37895. https://doi.org/10.7759/cureus.37895

[11] Alamri AA, Aljuhani WS, Alamri SA, Aloraini AA, Alamri SA. Blood flow restriction training in post-operative orthopedic rehabilitation: a systematic review and meta-analysis of randomized controlled trials. J Musculoskelet Surg Res. 2026. https://doi.org/10.25259/JMSR_365_2025

[12] Rodziewicz B, Fidut M, Kacperski M. Blood flow restriction training in postoperative musculoskeletal rehabilitation. J Educ Health Sport. 2026;91:70722. https://doi.org/10.12775/JEHS.2026.91.70722

[13] Reina-Ruiz ÁJ, et al. Effectiveness of blood flow restriction on functionality, quality of life and pain in patients with neuromusculoskeletal pathologies: a systematic review. Int J Environ Res Public Health. 2023;20(2):1401. https://doi.org/10.3390/ijerph20021401

[14] Koc BB, Truyens A, Heymans MJLF, Jansen EJP, Schotanus MGM. Effect of low-load blood flow restriction training after anterior cruciate ligament reconstruction: a systematic review. Int J Sports Phys Ther. 2022;17(3):334–346. https://doi.org/10.26603/001c.33151

[15] Butt J, Ahmed Z. Blood flow restriction training and its use in rehabilitation after anterior cruciate ligament reconstruction: a systematic review and meta-analysis. J Clin Med. 2024;13(20):6265. https://doi.org/10.3390/jcm13206265

[16] Bobes Álvarez C, Issa-Khozouz Santamaría P, Fernández-Matías R, Pecos-Martín D, Achalandabaso-Ochoa A, Fernández-Carnero S, Martínez-Amat A, Gallego-Izquierdo T. Comparison of blood flow restriction training versus non-occlusive training in patients with anterior cruciate ligament reconstruction or knee osteoarthritis: a systematic review. J Clin Med. 2020;10(1):68. https://doi.org/10.3390/jcm10010068

[17] Fraca-Fernández E, et al. Effects of blood flow restriction training in patients before and after anterior cruciate ligament reconstruction: a systematic review and meta-analysis. Healthcare (Basel). 2024;12(12):1231. https://doi.org/10.3390/healthcare12121231

[18] Fontanet R, et al. Blood flow restriction training prior to and after anterior cruciate ligament reconstruction: a scoping review. J Funct Morphol Kinesiol. 2025;10(4):450. https://doi.org/10.3390/jfmk10040450

[19] Ke J, Zhou X, Yang Y, Shen H, Luo X, Liu H, et al. Blood flow restriction training promotes functional recovery of knee joint in patients after arthroscopic partial meniscectomy: a randomized clinical trial. Front Physiol. 2022;13:1015853. https://doi.org/10.3389/fphys.2022.1015853

[20] Franz A, Ji S, Bittersohl B, Zilkens C, Behringer M. Impact of a six-week prehabilitation with blood-flow restriction training on pre- and postoperative skeletal muscle mass and strength in patients receiving primary total knee arthroplasty. Front Physiol. 2022;13:881484. https://doi.org/10.3389/fphys.2022.881484

[21] Franz A, et al. Passive blood-flow-restriction exercise's impact on muscle atrophy post-total knee replacement: a randomized trial. J Clin Med. 2025;14(15):5218. https://doi.org/10.3390/jcm14155218

[22] Öberg S, von Schewelov L, Tengman E. The impact of blood flow restriction training on tendon adaptation and tendon rehabilitation – a scoping review. BMC Musculoskelet Disord. 2025;26(1):503. https://doi.org/10.1186/s12891-025-08734-5

[23] Burton I, McCormack A. Blood flow restriction resistance training in tendon rehabilitation: a scoping review on intervention parameters, physiological effects, and outcomes. Front Sports Act Living. 2022;4:879860. https://doi.org/10.3389/fspor.2022.879860

[24] Johnson SR, Ndjonko L, Hou D, England P, Tjong VK, Sheth U. The impact of blood flow restriction therapy on orthopaedic conditions of the upper extremity: a systematic review of randomized controlled trials. Ann Joint. 2026;11:11. https://doi.org/10.21037/aoj-25-31

[25] Jing J, Zheng Q, Dong H, et al. Effects of upper extremity blood flow restriction training on muscle strength and hypertrophy: a systematic review and meta-analysis. Front Physiol. 2024;15:1488305. https://doi.org/10.3389/fphys.2024.1488305

[26] Zhang J, Xiao Y, Qiu L, Zhang G, Wang R, Zhu X, Wang C, Xu M. Effects of blood flow restriction therapy for lower limb dysfunction in stroke patients: a systematic review and meta-analysis. Front Neurol. 2026;17:1814592. https://doi.org/10.3389/fneur.2026.1814592

[27] Darwish MH, El-Tamawy MS, Soubhy HZ, Khalifa H, Marzouk MH. Blood flow restriction training in neurological rehabilitation: a comprehensive review. Health Sport Rehabil. 2026;12(3). https://doi.org/10.58962/hsr.1314

[28] Wang H, et al. Effectiveness of blood flow restriction training in neurological rehabilitation: a systematic review and meta-analysis. Sports Med Open. 2026;12. https://doi.org/10.1186/s40798-026-01022-z

[29] Cahalin LP, Formiga MF, Anderson B, Cipriano G Jr, Hernandez ED, Owens J, Hughes L. A call to action for blood flow restriction training in older adults with or susceptible to sarcopenia: a systematic review and meta-analysis. Front Physiol. 2022;13:924614. https://doi.org/10.3389/fphys.2022.924614

[30] Centner C, Wiegel P, Gollhofer A, König D. Effects of blood flow restriction training on muscular strength and hypertrophy in older individuals: a systematic review and meta-analysis. Sports Med. 2019;49(1):95–108. https://doi.org/10.1007/s40279-018-0994-1

[31] Chang H, Yao M, Chen B, Qi Y, Zhang J. Effects of blood flow restriction combined with low-intensity resistance training on lower-limb muscle strength and mass in post-middle-aged adults: a systematic review and meta-analysis. Int J Environ Res Public Health. 2022;19(23):15691. https://doi.org/10.3390/ijerph192315691

[32] Emam MA, Elsayed A, Hortobágyi T, Amin WM, Malik S, Ali OI. Exploring blood flow restriction exercise protocols for elderly populations: a scoping review of cuff pressure, frequency, and duration for muscle strength, hypertrophy, and functional abilities outcomes. J Clin Med. 2025;14(12):4185. https://doi.org/10.3390/jcm14124185

[33] Ren M, et al. Effect of different blood flow restriction training regimens combined with low-intensity training on muscle strength and cardiovascular safety in older adults: a systematic review and network meta-analysis. Front Physiol. 2025;16:1587876. https://doi.org/10.3389/fphys.2025.1587876

[34] Feng M, et al. Effect of blood flow restriction training on health promotion in middle-aged and elderly women: a systematic review and meta-analysis. Front Physiol. 2024;15:1392483. https://doi.org/10.3389/fphys.2024.1392483

[35] Tian H, Xiang Q, Huang L, Liu H, Yu H, Wu J, Li H, Kuang J, Yan X, Peng L. How does acute blood flow restriction resistance training influence free fatty acids in obese individuals? Qual Sport. 2025;38:58841. https://doi.org/10.12775/QS.2025.38.58841

[36] Oliva-Lozano JM, Patterson SD, Chiampas G, Maybury E, Cost R. Blood flow restriction as a post-exercise recovery strategy: a systematic review of the current status of the literature. Biol Sport. 2024;41(3):191–200. https://doi.org/10.5114/biolsport.2024.133664

[37] Wilk M, Krzysztofik M, Gepfert M, Poprzecki S, Gołaś A, Maszczyk A. Technical and training related aspects of resistance training using blood flow restriction in competitive sport – a review. J Hum Kinet. 2018;65:249–260. https://doi.org/10.2478/hukin-2018-0101

[38] Chen Y. Influence of blood flow restriction training combined with resistance training on physical function of college football players. Qual Sport. 2025;38:58447. https://doi.org/10.12775/QS.2025.38.58447

[39] Gear KM, Kim K, Lee S. Effects of training with blood flow restriction on muscular strength: a systematic review and meta-analysis. Int J Exerc Sci. 2022;15(3):1563–1577. https://doi.org/10.70252/TLFI8466

[40] Choroszewicz P, Dobosiewicz AM, Badiuk N. Sports massage as a method of preventing delayed onset muscle soreness. Pedagogy Psychol Sport. 2020;6(2):104–112. https://doi.org/10.12775/PPS.2020.06.02.010

[41] Xu X, Peng L. Effects of isometric training on heart rate variability: a systematic review and meta-analysis. Pedagogy Psychol Sport. 2025;28:67675. https://doi.org/10.12775/PPS.2025.28.67675