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Review

Arthrogenic Muscle Inhibition and Quadriceps Dysfunction After Primary and Revision Total Knee Arthroplasty: A Narrative Review

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Abstract

Total knee arthroplasty (TKA) reliably relieves pain in end-stage knee osteoarthritis, but quadriceps dysfunction may continue to limit walking, stair climbing, balance, and confidence. This narrative review examines arthrogenic muscle inhibition (AMI) and quadriceps dysfunction after primary and revision TKA. PubMed, Scopus, Embase, and the Cochrane Library were searched from inception through August 2026 using terms related to AMI, quadriceps activation, TKA, and rehabilitation. Relevant studies were selected for narrative synthesis, prioritizing direct TKA evidence and identifying evidence from other knee populations as indirect. AMI is impaired voluntary activation associated with altered joint afferent input and neural inhibition, whereas atrophy reflects loss of muscle tissue. Both can coexist and contribute to weakness. Surgical trauma, pain, effusion, inflammation, altered proprioception, and immobilization may impair early motor-unit recruitment. Preoperative weakness, inactivity, obesity, comorbidity, and revision surgery may further reduce physiological reserve. Muscle-preserving exposure may improve selected early outcomes, but must not compromise safe exposure or component positioning. Revision procedures add tissue injury, extensor-mechanism risk, systemic illness, and rehabilitation restrictions; direct voluntary-activation evidence after revision TKA remains limited. Assessment should combine strength and functional testing with measures of voluntary activation and muscle morphology when feasible. Management includes pain and swelling control, safe early activation, progressive strengthening, neuromuscular electrical stimulation, selected blood-flow-restriction training, motor-control rehabilitation, and targeted prehabilitation. Nutritional and laboratory assessment can identify modifiable risks but does not diagnose AMI. A practical perioperative framework is proposed to support identification and management of quadriceps dysfunction after primary and revision TKA.

Keywords: total knee arthroplasty; quadriceps; arthrogenic muscle inhibition; voluntary activation; rehabilitation; revision knee arthroplasty; prehabilitation; extensor mechanism

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1. Introduction

Total knee arthroplasty (TKA) is an effective treatment for end-stage knee osteoarthritis, providing predictable pain relief and durable implant survival for most patients. Functional recovery, however, is not determined by implant alignment or radiographic appearance alone. Many patients struggle with early transfers, stairs, gait asymmetry, balance, and confidence despite an uncomplicated operation. Quadriceps weakness is consistently associated with these limitations and may persist well after postoperative pain and wound

healing have resolved [1–3]. A muscle-centred approach is therefore clinically relevant to surgeons, anaesthetists, physiotherapists, and patients.

Quadriceps function declines rapidly after TKA. In one primary-TKA cohort assessed approximately four weeks after surgery, quadriceps strength, voluntary activation, and maximal quadriceps cross-sectional area had decreased by 62%, 17%, and 10%, respectively, from preoperative values. Both activation failure and atrophy contributed to strength loss, with activation failure contributing nearly twice as much as atrophy [4]. This phenomenon is usually described as arthrogenic muscle inhibition (AMI): a reflex and centrally mediated inability to fully activate the periarticular musculature after joint injury, inflammation, or surgery [5–7]. AMI is not simply pain avoidance or poor effort. It reflects altered afferent signalling from the joint and changes in spinal and supraspinal motor control, and it is often accompanied by swelling, inflammation, and reduced movement.

The distinction between AMI and atrophy matters. Structural loss of muscle mass develops with disuse, catabolism, and reduced loading, whereas AMI can prevent a patient from recruiting the remaining muscle effectively. The two processes reinforce each other: inhibition reduces loading, lower loading accelerates atrophy, and persistent weakness then sustains abnormal movement and reduced activity [6,7]. A patient may consequently have apparently satisfactory knee range of motion (RoM) but poor stair climbing, delayed straight-leg raise, or asymmetric gait. Routine rehabilitation that concentrates only on RoM may not adequately address this deficit.

The surgical setting is also relevant. Medial parapatellar, midvastus, and subvastus exposures differ in their relationship to the extensor mechanism and may influence early pain, active straight-leg raise, and quadriceps recovery. In revision TKA, additional dissection, multiple prior operations, infection-related inflammation, bone loss, extensor mechanism compromise, and more restrictive rehabilitation may magnify the same muscle problem. Tibial tubercle osteotomy (TTO) can provide safe extensile exposure in difficult revision surgery, but it alters the extensor mechanism rehabilitation pathway and should be considered when interpreting postoperative weakness [8–11].

Stevens-Lapsley et al. documented quadriceps and hamstring dysfunction after TKA; at one month, hamstring coactivation during maximal quadriceps effort was 144.5% higher in the operated than the nonoperative limb [12]. More recently, Le Guen et al. assessed AMI using the clinical SANTI classification in a mixed cohort that included knee arthroplasty and revision procedures. Among patients without preoperative AMI, 36% met the study's clinical criteria at 15 days and 12.4% at three months [13]. These pooled results were not reported separately for revision TKA and do not represent direct voluntary-activation measurements. Building on these studies and Rice and McNair's review of AMI mechanisms [5], the present review focuses on how AMI and quadriceps dysfunction are assessed and managed after primary and revision TKA. It integrates surgical and perioperative considerations, prehabilitation, and postoperative management; distinguishes direct TKA evidence from evidence extrapolated from other knee populations; and presents an integrated proposed perioperative pathway. The objective is not to imply that every patient requires specialized neurophysiological testing, but to provide a clinically useful framework for identifying meaningful muscle dysfunction and guiding interventions that support safe, progressive recovery.

2. Materials and Methods

PubMed, Scopus, Embase, and the Cochrane Library were searched from inception through August 2026. Search terms were combined in different configurations and included "arthrogenic muscle inhibition," "quadriceps activation," "voluntary activation," "quadriceps weakness," "muscle atrophy," "total knee arthroplasty," "revision total knee

arthroplasty," "rehabilitation," "prehabilitation," "neuromuscular electrical stimulation," and "blood-flow-restriction." MeSH terms and equivalent subject headings were used where available. Reference lists of relevant articles were also reviewed. The author screened the literature for relevance to AMI and quadriceps dysfunction after primary or revision TKA, considering evidence from these groups separately where available. Evidence from other knee populations was included when relevant to mechanisms or interventions and is identified as indirect. No predefined inclusion or exclusion criteria, independent duplicate screening, or formal risk-of-bias assessment were applied because this was a single-author narrative review. To reduce selection and interpretation bias, direct TKA evidence was prioritized where available, and relevant claims were checked against the cited publications. Only English-language publications were considered for inclusion in the narrative synthesis.

3. Defining Arthrogenic Muscle Inhibition and Postoperative Muscle Dysfunction

AMI is a reduction in the capacity to voluntarily activate a muscle surrounding an affected joint despite an intact peripheral motor pathway and without structural damage to the muscle itself. It is well recognized after knee injury, anterior cruciate ligament (ACL) injury, and osteoarthritis, and it is particularly relevant after knee surgery [5–7]. After TKA, AMI predominantly affects the quadriceps. Associated adaptations may coexist, including increased hamstring activation and co-contraction, altered hip strategies, and broader lower-limb deconditioning [2,12]. It is a functional neural deficit rather than a diagnosis based on one symptom, and its severity varies over time.

Voluntary activation is often quantified in research using superimposed electrical stimulation or the interpolated-twitch technique. During a maximal voluntary contraction, a stimulus that produces additional force indicates incomplete activation. The central activation ratio (CAR) is a related measure. CAR (%) is calculated as [voluntary force/(voluntary force + additional force elicited by stimulation)] × 100. With the interpolated-twitch technique, voluntary activation (%) is calculated as [1 − (superimposed twitch force/potentiated resting twitch force)] × 100. Because these methods can produce different estimates, the method used should be specified [14]. These techniques are valuable in mechanistic studies but require equipment and expertise. In clinical practice, a conspicuous inability to perform or sustain a straight-leg raise, marked asymmetry on dynamometry, extensor lag, poor sit-to-stand performance, and gait avoidance can serve as useful warning signs when interpreted alongside pain, RoM, and swelling [1,6,12].

AMI must be distinguished from other causes of weakness. True muscle atrophy results from reduced loading, postoperative catabolism, malnutrition, systemic disease, denervation, or resection. Femoral neuropathy, lumbar radiculopathy, tendon rupture, patellar fracture, extensor mechanism disruption, and component-related instability require specific evaluation and must not be labeled as AMI [12]. Pain-related guarding is common but does not fully account for the activation deficit observed after TKA. Similarly, labeling a patient as having low motivation is unhelpful and may delay treatment when motor output is genuinely inhibited [15].

The time course aids interpretation. Neural activation failure and swelling are prominent during the first days to weeks after surgery. Atrophy becomes increasingly important with prolonged limited loading. Persistent weakness beyond the expected recovery phase should prompt reassessment of pain, recurrent effusion, infection, extensor mechanism continuity, instability, arthrofibrosis, lumbar disease, medication effects, and adherence or barriers to rehabilitation [15,16]. This staged interpretation avoids both overdiagnosis of AMI and premature acceptance of chronic weakness as inevitable.

4. Mechanisms of Quadriceps Inhibition After TKA

The knee joint contains mechanoreceptors and nociceptors that contribute to proprioception and reflex control of the surrounding muscles. Joint effusion, capsular distension, inflammation, pain, and tissue injury alter afferent discharge from these structures. Experimental effusion reduces quadriceps activation, even at small volumes, and interventions that attenuate swelling or abnormal sensory input, such as cryotherapy, transcutaneous electrical nerve stimulation, and joint aspiration, can partially reverse inhibition [5,7,15]. After TKA, synovial resection, capsulotomy, bone cuts, soft-tissue balancing, and periarticular inflammation create a substantial sensory disturbance, even when the operation is technically uncomplicated.

At the spinal level, altered joint afferent activity influences interneuronal pathways and alpha-motoneuron excitability. Reduced voluntary drive can coexist with increased antagonist co-contraction, an understandable protective pattern that nevertheless makes extension inefficient. At the supraspinal level, pain, fear of movement, altered body representation, and reduced confidence may further reduce motor output. AMI should therefore be viewed as a multilevel neuromuscular response rather than a single local reflex [5,7,16]. This model also explains why interventions that simply ask the patient to "contract harder" may be ineffective in the acute phase.

Pain phenotyping is relevant because pain intensity alone may not capture altered pain processing. In a systematic review and meta-analysis of patients undergoing TKA, preoperative central sensitization was associated with greater postoperative pain; the included studies used measures such as the Central Sensitization Inventory, whole-body pain diagrams, and quantitative sensory testing [17]. These findings support assessment of pain distribution, neuropathic-like symptoms, fear, and pain-related avoidance when recovery is unexpectedly limited. They do not show that central sensitization is equivalent to AMI or that it directly causes quadriceps activation failure.

Muscle morphology changes in parallel. Postoperative inactivity and reduced mechanical loading lower protein synthesis and promote loss of quadriceps cross-sectional area. The operated limb can remain weaker than the contralateral side for months, even after standard rehabilitation [2,3]. Myosteatosis (fat infiltration within muscle) and poor preoperative muscle quality may reduce contractile efficiency, although their value as standalone prognostic biomarkers in routine TKA care remains uncertain. The important practical point is that ultrasound, CT, or MRI measurements of muscle size cannot substitute for strength or voluntary activation testing. Muscle quantity, muscle quality, and neural recruitment are related but distinct constructs.

Patient-related factors may influence perioperative recovery, but none establishes AMI. Modifiable risks, including obesity, diabetes, anemia, malnutrition, smoking, and opioid use, should be considered during preoperative optimization [18]. Older age and pre-existing sleep disturbance may affect perioperative sleep and pain [19]. Depression has been associated with persistent pain and poorer patient-reported outcomes after arthroplasty, although patients with depression may still achieve meaningful functional improvement [20]. These factors should inform individualized counselling, optimization, prehabilitation, and follow-up; they are not diagnostic markers of AMI, and their effect on surgical timing should be considered individually. Severe postoperative pain and persistent effusion may contribute to impaired quadriceps activation and should prompt assessment and management [5,13,15].

Sarcopenia and AMI describe related but distinct contributors to weakness. Sarcopenia involves reduced muscle strength and muscle quantity or quality, whereas AMI refers to impaired voluntary neural activation [21]. A patient with low preoperative muscle reserve may be less able to compensate for postoperative activation failure, pain, and restricted loading; however, a direct interaction between sarcopenia and AMI after TKA has not been

established. A recent meta-analysis estimated a pooled sarcopenia prevalence of 20.1% among TKA patients, with substantial heterogeneity between studies [22]. This supports attention to strength, mobility, nutritional risk, and frailty, but does not establish sarcopenia as a diagnostic marker of AMI.

5. Clinical Consequences and Assessment

Quadriceps function is essential for standing from a chair, controlling knee flexion during loading, descending stairs, and recovering from a balance perturbation. Patients with persistent weakness frequently adopt compensatory trunk lean, reduced knee flexion during gait, reliance on the contralateral limb, or prolonged use of walking aids [1–3]. These strategies may initially protect the painful knee but can prolong asymmetry and reduce community mobility. Quadriceps strength is more consistently associated with performance-based function after TKA than RoM alone [1,3,23].

Assessment should begin preoperatively. A simple baseline assessment, including active RoM, extensor lag, quadriceps strength, sit-to-stand performance, walking aid use, stair-climbing ability, and patient-defined functional goals, gives the postoperative team a meaningful reference [23]. Handheld dynamometry can be useful if the examiner uses a stable, standardized position; isokinetic dynamometry is more precise when available. Strength should be reported in absolute terms and, when appropriate, as limb symmetry, while recognizing that the contralateral knee may also have osteoarthritis [12,23].

Patient-reported measures add useful context but do not replace performance testing. A patient may report satisfactory pain relief while still avoiding stairs because of weakness or insecurity. Conversely, a low score may reflect pain sensitization, mood, or unmet expectations rather than a purely muscular deficit. Combining a knee-specific score with a timed functional test and a direct measure of extension strength provides a more interpretable clinical picture [23]. Whenever possible, the same measures should be used at each review; switching between tests makes it difficult to determine whether treatment has changed the recovery trajectory.

The early postoperative examination should document wound condition, pain trajectory, effusion, active straight-leg raise, extension lag, ability to transfer, gait pattern, and willingness to load the limb. Repeated measurements are more informative than a single poor performance. If weakness is disproportionate or worsening, structural and neurological causes must be actively excluded. New inability to extend the knee against gravity, a palpable defect, severe anterior pain after a fall, or a sudden change after revision surgery warrants urgent assessment for extensor mechanism injury [16].

Imaging and laboratory testing are adjuncts, not routine tests for AMI. Musculoskeletal ultrasound can quantify quadriceps thickness and estimate rectus femoris cross-sectional area at the bedside as a structural correlate of muscle size [24]. In patients after TKA, ultrasound-measured rectus femoris thickness has been associated with strength and physical function [25]. These measures assess muscle morphology, not neural activation. CT and MRI can assess muscle cross-sectional area and fatty infiltration when obtained for another clinical indication. Electromyography and nerve-conduction studies are reserved for suspected femoral neuropathy, radiculopathy, or another neurological disorder [12,15,16]. Serum albumin is a negative acute-phase reactant rather than a direct measure of muscle function. Low values should therefore prompt assessment of nutritional risk, systemic illness, or infection rather than a diagnosis of AMI. In high-risk primary or revision cases, a targeted panel can identify modifiable barriers to recovery. This may include a full blood count, renal function, glucose or glycated hemoglobin (HbA1c), albumin, and inflammatory markers when infection is suspected [18,26,27]. Table 1 summarizes practical distinctions between AMI, atrophy, and structural extensor mechanism pathology.

Table 1. Clinical differentiation of arthrogenic muscle inhibition, muscle atrophy, and structural extensor-mechanism or neurological pathology after TKA.

Feature	AMI	Muscle Atrophy or Poor Muscle Quality	Extensor-Mechanism or Neurological Pathology
Main mechanism	Altered neural activation from joint afferent and central pathways	Reduced contractile tissue or increased fatty infiltration	Tendon, nerve, or spinal pathology
Typical timing	Prominent immediately after surgery	Evolves with disuse and limited loading	Variable; often acute after trauma or related to revision/complication
Clinical clues	Poor voluntary contraction, extensor lag, effusion, pain	Reduced bulk and sustained weakness	Sudden loss of extension, palpable defect, sensory/reflex changes
Helpful assessment	Voluntary activation methods, dynamometry, serial function	Ultrasound, CT/MRI, strength testing	Radiographs/ultrasound/MRI and neurological testing as indicated
Management principle	Reduce inhibition and restore activation	Progressive loading and nutritional optimisation	Diagnose and treat the underlying structural or neurological cause

AMI: arthrogenic muscle inhibition; CT: computed tomography; MRI: magnetic resonance imaging; TKA: total knee arthroplasty.

6. Surgical Exposure and Extensor Mechanism Preservation

The medial parapatellar approach remains the most widely used exposure because it is familiar, extensile, and adaptable to a broad range of primary and revision situations. It involves arthrotomy through the medial quadriceps tendon and retinaculum and can temporarily affect the extensor mechanism. Midvastus and subvastus approaches aim to preserve more quadriceps continuity. A meta-analysis of 20 randomized trials (1893 primary TKAs) found that the subvastus approach shortened time to active straight-leg raise by 1.7 days (95% CI, 1.0–2.3), reduced day-1 pain by 0.8 points on a 10-point scale (95% CI, 0.2–1.4), and increased RoM at one week by 7° (95% CI, 3.2–10.7) compared with the medial parapatellar approach [8]. A network meta-analysis of 33 randomized trials also reported greater early RoM with the subvastus approach and lower early pain with the mini-subvastus approach; for example, on day 1, the mean difference was 6.99° for RoM (95% CI, 1.08–12.89) and −1.98 points for pain (95% CI, −2.93 to −1.03), respectively [9]. These differences diminished over time, and the first meta-analysis found no difference in Knee Society Scores at six weeks or one year. Neither review directly measured AMI [8,9].

These findings should be interpreted cautiously. Comparisons between approaches are influenced by surgeon experience, body habitus, deformity, stiffness, component design, analgesia, and rehabilitation protocol [8,9]. A muscle-sparing approach should not compromise exposure, component placement, ligament balancing, or safety. The relevant message for this review is not that one incision eliminates AMI, but that minimising avoidable extensor trauma and enabling early activation can support recovery in appropriately selected patients.

In revision TKA, safe exposure is frequently more important than preserving a theoretical early advantage. Dense scar tissue, patella baja, stiffness, prior osteotomy, large components, and bone loss can make a standard arthrotomy hazardous. Quadriceps snip, V-Y turndown, or TTO may be required. TTO provides controlled tibial tubercle osteotomy to improve exposure while protecting the extensor mechanism, but union, fixation, and the surgeon's postoperative loading protocol must guide rehabilitation [10,11]. The physiother-

apist should know that delayed active extension or modified loading may be intentional after an extensile exposure and should not be mistaken automatically for uncomplicated AMI.

Robotic assistance and computer navigation may affect the precision of bone preparation and component positioning, but their relevance to AMI remains uncertain. Robotic-assisted TKA may also allow more reproducible intraoperative assessment of ligament laxity, potentially informing soft-tissue balancing and the planning of bone resections and releases [28,29]. In a prospective randomized trial of 30 patients, robotic-arm-assisted TKA was associated with less observed periarticular soft-tissue injury and bone trauma than conventional jig-based TKA, together with a transient reduction in some inflammatory markers at postoperative day 7 [30]. These findings suggest a possible pathway through which robotic assistance could modify surgical trauma, but neither reproducible laxity assessment nor reduced trauma establishes that robotic assistance prevents AMI. Neither study directly measured voluntary quadriceps activation; this proposed link remains mechanistic and requires direct clinical study.

Periarticular analgesic infiltration and adductor-canal-based analgesia may influence early muscle recovery by supporting rehabilitation while limiting quadriceps motor block. In TKA studies, periarticular infiltration reduced opioid use relative to femoral nerve block during the first eight postoperative hours, while adductor canal block better preserved quadriceps strength than femoral nerve block [31,32]. Analgesia should not be judged only by a pain score. Motor-blocking techniques can transiently impede active knee control, while undertreated pain may reinforce protective inhibition [5]. The surgical and anesthetic plans should therefore be aligned with the intended rehabilitation pathway. A patient unable to actively control the knee on the first postoperative day requires a focused examination of motor block, pain, swelling, and extensor-mechanism continuity before activity is advanced.

7. Primary and Revision TKA

Primary TKA is usually performed on an osteoarthritic knee that has experienced years of pain-limited loading and quadriceps weakness. The operation removes a major pain generator, but it does not immediately restore motor control. Early recovery depends on achieving adequate analgesia, control of swelling, and repeatedly restoring activation before protective movement patterns become established. Patients with severe preoperative weakness, bilateral disease, limited mobility, or high fear of movement should be recognized before surgery rather than described later as slow rehabilitators [1–3].

Revision TKA creates a different and often more challenging environment for muscle recovery. Aseptic loosening may cause chronic pain, instability, recurrent effusions, and progressive activity restriction. Compared with aseptic revision, revision for periprosthetic joint infection (PJI) is associated with greater perioperative morbidity, including increased sepsis risk and longer hospitalization [33–36]. PJI also contributes substantially to the economic burden of arthroplasty [33], and infection-related revisions have a higher subsequent infection rate than revisions for aseptic failure [35]. Bone and joint infections are heterogeneous, and existing classification systems may capture only part of their clinical features [34]. PJI treatment may involve staged procedures with an interval antibiotic spacer and prolonged antimicrobial therapy; adverse events during prolonged antibiotic treatment have been reported [37,38]. Spacer type can affect interim knee motion and functional outcomes [39]. Stiffness and arthrofibrosis may further limit loading and motor retraining [13,40]. These treatment demands may complicate rehabilitation, but their specific effects on quadriceps activation have not been established. Our search identified no high-quality study directly measuring voluntary activation after revision TKA.

The extensor mechanism deserves particular attention in revision surgery. Previous lateral release, multiple arthrotomies, patellar fracture, patella baja, component malposition,

or prior tendon injury may reduce the margin for safe mobilization [9]. Reconstruction using allograft or mesh entails a different biological problem from AMI and commonly requires a deliberately protective rehabilitation protocol [41]. Once structural integrity is confirmed and restrictions allow, however, prolonged avoidance of quadriceps work can worsen inhibition and atrophy. Clear staging is therefore preferable: protect the repair first, restore activation as safely permitted, then progressively rebuild force and function.

Infection should not be equated with AMI. Painful failure after revision may indicate recurrent infection, instability, loosening, extensor-mechanism complication, or neuropathy, and it warrants surgical evaluation. Nevertheless, a muscle-focused pathway is valuable in this group: screen nutrition and anemia, treat infection appropriately, clarify permitted loading and active-extension restrictions, prevent prolonged avoidable immobilization, and establish realistic recovery milestones. Rehabilitation after two-stage PJI treatment should be individualized to the spacer type, bone and soft-tissue status, extensor-mechanism integrity, and surgeon-defined weight-bearing and range-of-motion restrictions; it should not automatically follow a routine primary-TKA protocol [38,39].

8. Prehabilitation and Perioperative Optimization

Prehabilitation is the planned improvement of functional capacity before surgery through exercise, education, optimization of symptoms, and management of modifiable risk. In TKA, the most relevant components are quadriceps and hip strengthening, functional training, aerobic conditioning within tolerance, gait-aid education, and clear expectations about early activation. Systematic reviews suggest that structured prehabilitation can improve preoperative strength and selected early postoperative functional and patient-reported outcomes, although programs vary substantially in intensity, duration, adherence, and co-interventions [42–45]. The evidence does not justify postponing needed surgery to reach an arbitrary strength target. It does support targeted intervention in patients with marked weakness, low mobility, fear of movement, or a likely complex recovery.

An effective program must be feasible. A four- to six-week supervised or hybrid program may include progressive sit-to-stand, knee extension within symptoms, hip abductor strengthening, balance work, walking or cycling, and a home plan [42–45]. High-load exercise may be inappropriate in severe pain, unstable gait, or significant deformity, so the prescription should be individualized. Education should prepare patients for the expected early loss of strength and emphasize the importance of regular muscle activation, rather than promising a painless or immediate recovery.

Prehabilitation should also establish postoperative self-efficacy. Practicing with a walker or crutches, rehearsing transfers and home exercises, and explaining the use of cryotherapy and compression and the warning symptoms to report all reduce uncertainty when postoperative pain and fatigue are greatest [42–45]. In revision cases, it is especially important to clarify in advance whether active extension, weight bearing, or flexion is limited by an osteotomy, extensor repair, flap, or spacer. These restrictions may be necessary for healing, which makes it even more important to have a plan ready to restore muscle activation as soon as they can safely be relaxed.

Optimization also includes medication review, smoking cessation, diabetes management, anemia evaluation, sleep, and nutrition [18]. Low serum albumin is associated with adverse arthroplasty outcomes in observational datasets, but it reflects illness and inflammation as well as nutritional status [26,27]. Routine hormonal screening without a specific clinical indication is not warranted. Dietetic referral is appropriate for patients with identified malnutrition, marked weight loss, poor intake, frailty, or complex revision or PJI treatment. Protein intake should be adequate to support recovery, but no single supplement can replace progressive mechanical loading and rehabilitation [46,47].

9. Postoperative Interventions to Restore Activation and Strength

Pain and swelling management are foundational. Multimodal analgesia, regional techniques where appropriate, cryotherapy, compression, elevation, and early safe movement can all improve participation in activation exercises. Cryotherapy may modestly reduce pain and effusion in the short term; it is an adjunct to exercise, not a substitute. Persistent effusion should prompt evaluation for infection, haemarthrosis, inflammatory flare, or mechanical causes rather than indefinite symptomatic treatment [15,48].

Digital and telehealth-supported rehabilitation can provide exercise instruction, monitoring, and contact with clinicians when in-person visits are difficult. A systematic review and meta-analysis of nine studies involving 1944 patients found broadly comparable longer-term pain and functional outcomes between home-based telerehabilitation and hospital-based rehabilitation, although the evidence was rated as low quality and some outcomes differed at earlier time points [49]. These studies evaluated rehabilitation delivery rather than AMI-specific treatment. Digital support may supplement an individualized program, but it should not replace clinical reassessment when weakness is worsening, nor override the closer supervision often required after complex revision surgery.

Neuromuscular electrical stimulation (NMES) can be considered when voluntary quadriceps recruitment is poor. In a randomized trial after primary TKA, NMES was started 48 h after surgery and prescribed as 15 contractions per session, twice daily, for six weeks, at the maximum tolerable intensity [50]. Avramidis et al. randomized 30 patients to standard physiotherapy with or without electrical muscle stimulation of the vastus medialis; stimulation was delivered at 40 Hz with a pulse duration of 300 µs for four hours daily, starting on postoperative day 2 and continuing for six weeks [51]. The stimulation group had greater walking speed at six and 12 weeks, although no significant between-group differences were found in the HSS knee score or Physiological Cost Index. This was the protocol used in that trial and should not be taken as an established standard dose. Broader clinical guidance on NMES at the knee recommends eliciting a contraction of at least 50% of same-day MVIC to support strengthening; this target has not been validated as a required dose after TKA [52]. NMES should be combined with attempted voluntary contraction and functional exercise when safe. To support adherence, clinicians can explain the purpose of treatment, provide a familiarisation period, supervise initial application, set stimulation intensity according to patient tolerance, monitor pain and discomfort, and use adherence tracking when available [53]. These adherence findings are relevant to tolerability but are not specific to the post-TKA population. Before NMES, clinicians should check device-specific precautions and contraindications and monitor skin integrity, sensation, discomfort, fatigue, and adherence.

Progressive resistance exercise is the core strategy for rebuilding muscle capacity. Begin with exercises that the patient can perform with controlled movement and within the surgeon's restrictions. Progress resistance, range, and task difficulty when technique is maintained and pain or swelling does not show a sustained worsening after the session. Reduce the load or reassess the patient if pain, effusion, wound status, or movement quality deteriorates. Progression should be individualized rather than tied to a fixed timetable, particularly after revision surgery, tibial tubercle osteotomy, or extensor-mechanism repair.

Blood-flow-restriction (BFR) training permits low-load exercise to provide a metabolic stimulus when heavier resistance is poorly tolerated. However, evidence specific to TKA remains limited. A case report described improved quadriceps measures and physical function after home-based BFR, whereas a retrospective study of patients with painful TKA found no significant strength benefit over standard physical therapy [54,55]. Protocols and patient selection therefore require further study. If BFR is considered, it should be prescribed by a trained clinician using individualized limb-occlusion pressure, with screening for

peripheral vascular disease, thromboembolic risk, uncontrolled hypertension, impaired sensation, and other relevant contraindications [56]. BFR is an optional adjunct for selected patients, not routine treatment. NMES, BFR, and exercise progression must remain within procedure-specific precautions; the surgeon's restrictions on weight bearing, RoM, and extensor-mechanism loading take priority.

Motor-control rehabilitation should address gait asymmetry, excessive co-contraction, confidence, and balance. Practical strategies include auditory or visual feedback during quadriceps exercise [57] and real-time movement-pattern biofeedback during functional tasks such as sit-to-stand, walking, and stair climbing [58]. In a randomized trial, feedback-based quadriceps exercise was associated with short-term improvements in pain and selected functional measures. A separate movement-pattern biofeedback trial did not improve its primary outcome of operated-knee loading at six months and reported mixed functional outcomes. These approaches may be considered for selected patients, but current evidence does not establish that one method is superior for all patients. A patient who fails to progress despite good adherence should be reassessed rather than simply given more repetitions. Table 2 outlines a muscle-focused intervention strategy and its supporting evidence. Evidence from ACL and other knee populations informs some proposed mechanisms and interventions but is indirect and should not be assumed to demonstrate efficacy after TKA.

Table 2. Muscle-focused interventions for quadriceps dysfunction after total knee arthroplasty.

Intervention	Primary Target	Practical Use	Evidence and Limitation
Pain and effusion control	Sensory drivers of AMI	Multimodal analgesia, compression, cryotherapy, elevation, early motion	Supports participation; persistent swelling needs diagnostic reassessment [15,48]
NMES	Poor voluntary quadriceps recruitment	Start early when appropriate and use a strong, visible, tolerable contraction. One primary-TKA trial used 15 contractions per session, twice daily, for six weeks at maximum tolerable intensity.	Use as an adjunct to exercise; protocols vary. Broader knee NMES guidance recommends a contraction of at least 50% MVIC, but this is not established as a required TKA dose [50,52,59]
Progressive resistance exercise	Strength and muscle capacity	Progress resistance and task difficulty at a tolerable effort, while maintaining movement quality and following healing and surgical restrictions.	Core rehabilitation approach; the optimal dose after complex revision surgery is not established [3,23]
BFR training	Low-load strengthening when heavier loads are poorly tolerated	If selected, use trained supervision and individualized limb-occlusion pressure, commonly 40–80%. Screen for contraindications and follow surgical restrictions.	TKA-specific evidence is limited to a case report and a retrospective study that found no significant strength advantage over standard physical therapy [54–56]
Prehabilitation	Low preoperative muscle reserve or functional capacity	Use targeted strengthening, conditioning, education, and a feasible home program.	Benefits may be concentrated in early recovery, and programs vary in content and duration [42–45]

AMI: arthrogenic muscle inhibition; BFR: blood flow restriction; MVIC: maximal voluntary isometric contraction; NMES: neuromuscular electrical stimulation; TKA: total knee arthroplasty.

10. A Practical Perioperative Pathway

Sections 8 and 9 summarize evidence-informed considerations for perioperative optimization and postoperative rehabilitation. Table 3 presents the author's proposed perioperative framework; it has not been prospectively validated and should support clinical reasoning rather than be treated as a fixed protocol. Because validated TKA-specific decision cutoffs have not been established, Table 3 uses clinical prompts for reassessment rather than numerical thresholds. Interpret changes relative to preoperative function and in light of age, contralateral-knee disease, revision indication, symptoms, and procedure-specific restrictions. Interpret limb-symmetry measures cautiously when the contralateral knee is affected by osteoarthritis. Before primary TKA, identify patients with poor straight-leg raise, marked quadriceps weakness, low mobility, bilateral disease, high pain-related avoidance, or anticipated complex exposure. Before revision surgery, consider additional risk factors such as prior extensor-mechanism procedures, infection, prolonged immobilization, bone loss, and planned TTO or other extensile exposure. Document baseline strength and function where feasible, and optimize pain, nutrition, anemia, glucose control, and social support according to individual risk.

Table 3. Proposed perioperative pathway for prevention and management of persistent quadriceps dysfunction after primary and revision TKA.

Phase	Key Assessment	Muscle-Focused Action	Reassessment Prompt
Preoperative	Strength, straight-leg raise, function, gait aid, risk factors	Targeted prehabilitation and education	Marked weakness, inability to perform a straight-leg raise, or important functional limitation; consider targeted prehabilitation and further assessment
Days 0–14	Pain, effusion, active extension, transfer and gait	Swelling control, activation, NMES when indicated, safe early mobilisation	Inability to extend against gravity; new or increasing extensor lag; escalating pain or swelling; neurological deficit; wound concern; or suspected structural complication. Surgical precautions take priority.
Weeks 2–12	Strength trend, gait, sit-to-stand, stairs	Progressive resistance, functional and motor-control training	Worsening or plateau in serial strength or function; persistent or increasing extensor lag; or failure to progress in gait, transfers, or stairs. Reassess possible barriers to recovery
Beyond 12 weeks	Functional goals, symmetry, confidence, participation	Higher-level strengthening and activity progression	Persistent disabling weakness or failure to improve on serial testing; reassess structural, neurological, infectious, pain-related, and systemic contributors. Use the same functional tests over time where feasible

NMES: neuromuscular electrical stimulation.

In the first two postoperative weeks, priorities are wound safety, active extension, swelling control, regular quadriceps activation, transfers, and gait. From weeks 2 to 12, rehabilitation should consolidate voluntary activation, restore range of motion, progress strengthening, and reduce dependence on walking aids. As recovery permits, the focus can shift toward gait symmetry, stair function, and return to meaningful activity. Beyond

12 weeks, continue progressive loading and activity progression with periodic objective reassessment. If expected improvement is absent, review pain, swelling, adherence, infection risk, instability, stiffness, extensor integrity, and neurological causes. This approach is more useful than attributing slow progress to the patient or applying the same program regardless of whether weakness is predominantly inhibitory, atrophic, or structurally restricted.

Communication between surgeon and rehabilitation team is essential. The operative note should specify the surgical exposure, the integrity and treatment of the extensor mechanism, weight-bearing and range-of-motion restrictions, and any complication that could alter progression. In primary TKA, this is usually straightforward. In revision TKA, failure to communicate a TTO, patellar-tendon repair, soft tissue flap, or compromised bone stock can lead either to excessive caution and avoidable deconditioning, or to overly aggressive loading that places the reconstruction at risk. The pathway must protect healing tissue while avoiding an unnecessarily passive recovery.

11. Knowledge Gaps and Future Directions

Evidence on quadriceps strength and function after primary TKA is considerably stronger than evidence on AMI measured directly using validated voluntary-activation techniques [1–3]. Direct evidence after revision TKA and for several adjunctive interventions remains sparse. Differences in study design, measurement methods, rehabilitation protocols, and outcome time points limit comparisons across studies. Future studies should distinguish muscle loss from activation failure, report analgesic and surgical-exposure details, and include revision-TKA populations rather than extrapolating from uncomplicated primary procedures.

The field also needs clinically usable thresholds. A research-grade activation ratio may identify a mechanism, but most services need feasible combinations of dynamometry, active extension, swelling assessment, and functional tests. Ultrasound and wearable activity measures may eventually support serial monitoring, but their incremental value must be demonstrated. Trials should test phenotype-guided programs: for example, early NMES for severe activation failure, BFR for patients unable to tolerate heavy loading, and intensified prehabilitation for those with poor baseline strength.

This narrative review has limitations. It was conducted by a single author, included only English-language publications, and did not use independent duplicate screening or a formal risk-of-bias assessment. Selection and interpretation bias may therefore have influenced the evidence summarized. The search aimed to identify the literature relevant to the review question rather than to provide a systematic or exhaustive account.

12. Conclusions

Quadriceps dysfunction after primary and revision TKA is multifactorial: AMI, atrophy, pain, effusion, altered motor control, and structural pathology may coexist. Assessment should include strength and functional testing and evaluation for structural or neurological causes; direct voluntary-activation evidence after revision TKA remains limited. Management should be individualized to muscle reserve, operative approach, extensor-mechanism status, and surgical restrictions. Prehabilitation, pain and swelling control, safe early activation, and progressive strengthening are central; NMES or BFR may be considered for selected patients, with progress reassessed functionally. The proposed pathway requires further validation in future prospective studies.

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