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Review

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From cortex to quadriceps: understanding and treating arthrogenic muscle inhibition (AMI) after ACL injury

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ABSTRACT

Arthrogenic muscle inhibition (AMI) limits voluntary quadriceps activation after anterior cruciate ligament (ACL) injury despite preserved muscle structure. This review aims to synthesize current evidence on the neurophysiopathology of AMI, measured via electroencephalography (EEG), functional magnetic resonance imaging (fMRI), and transcranial magnetic stimulation (TMS), and on rehabilitation strategies targeting AMI. A scoping review was undertaken. A comprehensive literature search was conducted in PubMed, Web of Science, and the Cochrane Library to identify studies examining the neurophysiological mechanisms underlying AMI, and evaluating interventions aimed at restoring quadriceps function. A total of 30 studies were included in the neurophysiological basis of AMI: 11 employing EEG, 8 using fMRI, and 11 utilizing TMS. These studies characterized the neurophysiological basis of AMI, demonstrating local joint inhibition, altered afferent signaling, cortical reorganization, and reduced corticospinal excitability, all contributing to impaired quadriceps activation. Twenty intervention studies showed that multiple strategies can enhance quadriceps activation and strength, particularly when applied early or in a phase-specific manner. Importantly, AMI is progressively reversible but leaves a subset of motoneurons difficult to recruit voluntarily, highlighting the need for interventions that specifically target the central nervous system, in addition to the muscle, to optimize neuromuscular recovery. AMI is driven by complex interactions between peripheral and central neural mechanisms, measurable via EEG, fMRI, and TMS. Early identification and targeted interventions can restore quadriceps function and mitigate long-term deficits. Understanding both neurophysiological alterations and effective therapeutic strategies is essential for designing individualized, evidence-based rehabilitation programs for ACL-injured patients.

Keywords: ACL, neuroscience, EEG, fMRI, TMS, therapeutic strategies

INTRODUCTION

Anterior cruciate ligament (ACL) rupture is among the most common and severe knee injuries, with an annual incidence of approximately 0.03% in the general population and between 0.15% and 3.67% among professional athletes.[1] Each year, an estimated 250,000 new ACL ruptures occur in the United States, primarily during pivoting or rapid change-of-direction activities.[2] The injury predominantly affects active young adults aged 15 to 30 years and is more frequent in female due to specific biomechanical and hormonal factors.[3] Although ACL rehabilitation is well standardized in clinical practice, return-to-sport (RTS) outcomes remain suboptimal. A recent meta-analysis reported that while 82% of patients resume some level of sport after ACL reconstruction (ACLR), only 44% return to their preinjury competitive level.[4]

Quadriceps activation failure is frequent and persistent impairment following ACL rupture or reconstruction.[5] This deficit not only affects the injured limb but also extends to the contralateral, uninjured side.[6] Such findings suggest a mechanism beyond local disuse or atrophy secondary to pain or immobilization. This phenomenon is attributed to arthrogenic muscle inhibition (AMI), a neurophysiological process characterized by altered excitability within the motor pathways, resulting in insufficient quadriceps activation.[7] The bilateral nature of AMI supports the involvement of centralized neurophysiological mechanisms. Altered sensory input from the injured joint modulates spinal and supraspinal circuits, leading to decreased motoneuron excitability and reflex inhibition of periarticular musculature-even in the contralateral limb.[8-10] This highlights the central, rather than

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purely peripheral, role of AMI in post-injury neuromuscular dysfunction.

AMI has been identified in over half of patients with acute ACL rupture.[5] Its consequences are clinically significant and include extension deficits, gait abnormalities, quadriceps atrophy, reduced function, persistent pain, dynamic instability, and early-onset osteoarthritis.[11] These effects often persist after returning to unrestricted physical activity and may contribute to recurrent injuries and long-term disability.[12] Preoperative identification of AMI is crucial in patients awaiting ACLR, as its presence increases the risk of postoperative stiffness and delayed recovery. Surgical intervention should be postponed until targeted rehabilitation protocols have addressed these deficits.[10] Warning signs suggestive of AMI include joint effusion, high pain scores, a short interval between injury and assessment, multi-ligament involvement, crutch dependence, and nocturnal knee support.[5] Sonnery-Cottet et al.[10] proposed a classification system for AMI based on the reversibility of vastus medialis oblique (VMO) inhibition, which can guide individualized treatment strategies.

AMI represents a major barrier to effective ACL rehabilitation.[11] It impairs voluntary muscle activation, leading to strength loss, proprioceptive deficits, and compromised motor control-factors that increase reinjury risk. Its bilateral impact further complicates recovery, prolongs rehabilitation, and contributes to persistent asymmetries. Moreover, AMI may affect patient motivation and engagement, adding a psychological dimension to its management. Recent advances in neurorehabilitation have challenged traditional paradigms of post-ACL care. Emerging interventions aim to restore quadriceps activation by targeting joint mechanoreceptors, peripheral neural structures, or directly modulating central nervous system excitability.[13,14] This shift reflects a growing recognition that successful rehabilitation requires addressing not only the "hardware" (the knee and its mechanical integrity) but also the "software" (the brain and its neural control).[15]

Despite increasing attention in AMI with a growing number of studies emphasizing its importance and clinical impact, its pathophysiology remains incompletely understood, particularly the contribution of cortical and subcortical mechanisms. A deeper understanding of the neurophysiological pathways involved in AMI is essential to develop more effective interventions and reduce recurrence following ACL rupture or reconstruction. Therefore, the purpose of this scoping review is to (1) examine the neural mechanisms underlying AMI and (2) discuss evidence-based therapeutic strategies for its management in patients after ACL rupture or reconstruction.

METHODS

This study is a scoping review of the literature designed to explore the neural mechanisms involved in the pathophysiology of AMI, as well as the therapeutic strategies used to manage it in patients following ACL rupture or reconstruction.

Search Strategy

The literature search and study selection process were conducted in a structured and transparent manner following the principles outlined in the PRISMA-ScR guidelines to ensure methodological rigor.[16] A comprehensive search was performed in PubMed, Web of Science, and the Cochrane Library databases between September and December 2025. The search was conducted in two stages to address the review objectives. Abstract screening and full-text eligibility assessment were conducted independently by two reviewers. Any disagreements regarding study inclusion were resolved through consensus discussion.

In the first stage, the search focused on the pathophysiology of AMI and the involvement of neural pathways following ACL rupture or reconstruction. Keywords and specific term combinations were formulated in English, using Boolean operators (AND, OR) to refine results. The primary search terms included: "AMI" (OR "Quadriceps weakness") AND "Neuroplasticity" (OR "Neural mechanisms", "TMS", "EEG", "fMRI") AND "ACL injury" (OR "ACL rupture", "ACL reconstruction").

In the second stage, the search targeted rehabilitation and therapeutic strategies addressing AMI after ACL rupture or reconstruction. The following search terms and combinations were used: "AMI" (OR "Quadriceps weakness") AND "Rehabilitation strategies" AND "ACL injury" (OR "ACL rupture", "ACL reconstruction").

Additionally, a manual search of the reference lists of included studies was performed to identify further relevant publications. This systematic approach ensured comprehensive coverage of the literature on both the neurophysiological mechanisms of AMI and innovative therapeutic interventions designed to address it.

Study Selection

Inclusion criteria comprised studies investigating the neural mechanisms underlying AMI or exploring treatment and rehabilitation interventions aimed at treating AMI in patients with ACL rupture or reconstruction, with no restriction on publication date. Exclusion criteria included studies on other forms of muscle atrophy unrelated to AMI, those that did not explicitly address neural pathway involvement, and studies that lacked a comparison between a control group and a patient group with AMI following ACL rupture or reconstruction. Only full-text articles published in English or French were included. Single case reports and author commentaries were excluded from the analysis.

Data Extraction

Data extraction focused on several key elements to address the objectives of this review. The following information was collected: author, study title, year of publication, journal, and study objective. Regarding participant characteristics, the sample size was recorded, distinguishing between the study group and the control group. Particular attention was given to the methods used to assess neural activity, transcranial magnetic stimulation (TMS), functional magnetic resonance imaging (fMRI), and electroencephalography (EEG). The outcome measures and main findings reported in each study were extracted to analyze the neural pathways involved in AMI.

For studies focusing on the management of AMI in patients with ACL rupture or reconstruction, the therapeutic

strategies, their application protocols, and their effects on AMI were also extracted and analyzed.

RESULTS

Neurophysiopathology of AMI

The initial literature search across PubMed, Web of Science, and the Cochrane Library identified 473 articles, supplemented by 13 additional studies from other sources. After removing duplicates, 312 articles remained and were screened based on titles and abstracts, leading to the exclusion of 226 articles deemed irrelevant. A total of 86 full-text articles were then assessed for eligibility. Of these, 66 were excluded due to case-series design, lack of direct association with ACL injury, or duplication of data previously published in other studies. Ultimately, 30 studies were included in the qualitative synthesis: 11 employing EEG[17-27] (**Table 1**), 8 fMRI[28-35] (**Table 2**) and 11 utilizing TMS (**Table 3**).[36-46] The study selection process is summarized in the PRISMA flow diagram (**Figure 1**).

Table 1. Summary of studies using EEG

Author/year	Population	Technique	Results
Piskin[17]	15 healthy vs. 10 ACLR	Spectral power analysis during a kicking task	Significant increase in frontal theta power and decrease in posterior alpha power in the ACLR group.
Bush[18]	20 healthy vs. 10 ACLR	Spectral power analysis during a knee proprioception test (JPS).	Increase in bilateral frontal theta power, although not statistically significant, and a slight increase in parietal alpha-2 power in the ACLR group.
An[19]	15 healthy vs. 15 ACLR	Spectral power analysis during a single-leg stance (first 3 seconds).	Increase in theta power in the contralateral primary motor cortex (frontal), and an increase in alpha-2 power in the ipsilateral premotor cortex in the ACL group, but not statistically significant.
Miao[20]	15 healhty vs 16 ACLD	Spectral power analysis during the following activities: walking, light jogging, and landing	Significant increase in spectral power across all frequency bands, and for all tasks, in the ACLD group.
Baumeister[21]	9 healthy vs 9 ACLR	Spectral power analysis during knee extension	Significant increase in frontal theta power (F3 and Fz) in ACLR patients.
Baumeister[22]	12 healthy vs 10 ACLR	Spectral power analysis during 40° knee flexion.	Significant increase in frontal theta band power (F3, F4, F8) and parietal alpha-2 power (P3, P4) in ACLR patients.
Iwasa[23]	8 healthy vs. 39 ACLR	ACL somatosensory evoked potentials (SEPs) during arthroscopy.	Reproducible SEPs were observed in all healthy patients and in 39 out of 45 ACLR patients.
Ochi[24]	19 healthy vs. 45 ACLD vs. 42 ACLR	ACL somatosensory evoked potentials (SEPs) during arthroscopy.	Reproducible SEPs were observed in 58% of ACL-deficient patients, 86% of ACLR patients, and 100% of individuals with a healthy ACL.
Ochi[25]	14 healthy vs 32 ACLD vs 23 ACLR	ACL somatosensory evoked potentials (SEPs) during arthroscopy.	Reproducible SEPs were observed in 47% of ACL-deficient patients, 96% of ACLR patients, and 100% of individuals with a healthy ACL.
Lavender[26]	4 healthy vs 7 ACLD	ACL somatosensory evoked potentials (SEPs) during arthroscopy.	All intact ACLs showed reproducible SEPs, whereas torn ACLs did not show reproducible SEPs.
Valeriani[27]	20 healthy vs 19 ACLD	Somatosensory evoked potentials (SEPs) of the common peroneal nerve	Seven participants in the ACL-deficient group showed SEP abnormalities following stimulation of the common peroneal nerve.

EEG: Electroencephalography, ACLR: Anterior cruciate ligament reconstruction, ACLD: Anterior cruciate ligament deficient, JPS: Joint position sense, ACL: Anterior cruciate ligament, SEPs: Somatosensory evoked potentials, F3, F4, F8, Fz, P3, P4: Standard EEG electrode positions (frontal and parietal regions)

Table 2. Summary of studies using fMRI

Author/year	Population	Results
Culiver[28]	15 healthy vs. 15 ACLR	Decreased activity: ipsilateral M1 and S1, contralateral S1, SMA, and precuneus in ACL-reconstructed individuals.
Guan[29]	15 healthy and 15 ACLD	Decreased connectivity between cerebellar regions and large cortical regions (occipital, parietal, and frontal cortices), as well as reduced fronto-parietal and parieto-occipital connections in ACL-deficient participants.
Baez[30]	12 healthy vs. 12 ACLR	Increased activity: inferior parietal lobule, mediodorsal thalamus, cerebellum, and inferior occipital regions in ACL-reconstructed participants.
Criss[31]	15 healthy vs. 15 ACLR	Increased activity: intracalcarine cortex, lingual gyrus, occipital fusiform gyrus, lateral occipital cortex, angular gyrus, and superior parietal lobule in ACL-reconstructed individuals.
Diefkuss[32]	12 healthy vs. 3 ACLD	Decreased connectivity between ipsilateral S2 and ipsilateral SMA, contralateral premotor cortex, contralateral SMA, ipsilateral S1, and ipsilateral M1 in ACL-deficient individuals.
Diefkus[33]	8 healthy vs. 2 ACLD	Decreased connectivity between left S1 and the right posterior cerebellar lobe in ACL-deficient individuals
Grooms[34]	Healthy vs. 15 ACLR	Increased activity: contralateral M1, lingual gyrus, and contralateral S2 in ACL-reconstructed participants. Decreased activity: ipsilateral M1 and cerebellum in ACL-reconstructed participants.
Kapreli[35]	18 healthy vs. 17 ACLD	Decreased activity: contralateral thalamus, posterior parietal cortex, S1, external pallidum, S2, cingulate motor area, premotor cortex, and ipsilateral cerebellum (vermis and anterior lobe), and ipsilateral SM1 in ACL-deficient individuals. Increased activity: SMA, posterior S2, and posterior inferior temporal gyrus in ACL-deficient individuals.

fMRI: Functional magnetic resonance imaging, ACLR: Anterior cruciate ligament reconstruction, ACLD: Anterior cruciate ligament deficient, M1: Primary motor cortex, S1: Primary somatosensory cortex, S2: Secondary somatosensory cortex, SMA: Supplementary motor area, ACL: Anterior cruciate ligament, SM1: Sensorimotor cortex

Table 3. Summary of studies using TMS

Author/year	Population	Measurement	Results
Scheure[33]6	16 healthy vs. 16 ACLR	-TMS/EMG: AMT, SICI, ICF - Dynamometer: MVIC -EMG/dynamometer: CAR	- AMT significantly higher in the ACLR group compared to controls. - MVIC significantly lower in the injured limb compared to the uninjured and control limbs. - No significant difference in CAR, SICI, or ICF.
Zarzycki[37]	18 healthy vs. 18 ACLR	-TMS/EMG: SICI, ICF - Dynamometer: MVIC	-ACLR group showed significantly reduced intracortical facilitation (ICF) in the operated limb compared to the contralateral limb, but no difference compared to controls. - No significant difference in SICI.
Lepley[38]	11 healthy vs. 11 ACLR	-TMS/EMG: AMT, MEP -EMG: H/M - Dynamometer: MVIC - fMRI -Structural MRI: Quadriceps muscle volume	- MVIC and muscle volume were lower in the injured limb compared to the contralateral and control limbs. - AMT higher bilaterally in the ACLR group vs. controls. - Smaller MEPs in the injured limb compared to contralateral and controls. - No difference in H/M ratio. - ACLR group showed greater activation in brain regions involved in pain perception: frontal lobe regions, inferior frontal gyrus, paracingulate gyrus, and anterior cingulate gyrus.
Norte[39]	30 healthy vs. 72 ACLR	-TMS/EMG: AMT - Dynamometer/EMG: MVIC, CAR -EMG: H/M	No significant differences.
Zarzycki[40]	18 healthy vs. 18 ACLR	TMS/EMG: RMT, MEP, SICI, ICF EMG: Hoffman reflex	- Higher RMT and greater MEP amplitudes in the ACLR group. - No significant difference for ICF, SICI, or H/M reflex.
Ward[41]	18 healthy vs. 18 ACLD	-TMS/EMG: MEP, AMT, CsP, SICI	-CsP was higher in the injured limb compared to the non-injured limb in ACLD -No significant difference was observed for corticospinal excitability (MEP, AMT) or SICI.
Petrosimone[42]	29 healthy vs. 28 ACLR	TMS/EMG: AMT EMG: H/M Dynamometer/EMG: CAR	-CAR was lower bilaterally compared to controls. -AMT was higher in the injured limb compared to the contralateral limb and to controls. -Higher H/M ratios in both limbs of ACLR participants.
Kuenze[43]	24 healthy vs. 22 ACLR	TMS/EMG: AMT Dynamometer / EMG: MVIC, CAR EMG: H/M	-AMT higher in the ACLR group. -MVIC and CAR lower in the ACLR group. -No difference in H/M ratio.
Lepley[44]	20 healthy vs. 20 ACLR	TMS/EMG: AMT, MEP Dynamometer/EMG: MVIC, CAR	-AMT significantly higher in the injured limb and contralateral limb compared to controls. -No significant difference in MEP. -MVIC and CAR lower in both limbs of ACLR participants compared to controls.
Lepley[45]	29 healthy vs. 29 ACLR	TMS / EMG: AMT EMG: H/M Dynamometer/EMG: MVIC, CAR	-AMT higher in the ACLR group. -H/M reflex higher in the ACLR group. -MVIC and CAR lower in both limbs compared to controls.
Heroux[46]	4 healthy vs. 10 ACLD	-TMS / EMG: RMT, MEP, SR curve -EMG: H/M	-RMT significantly reduced in ACLR compared to controls. -No significant difference in MEP, SR curve, or H/M reflex.

TMS: Transcranial magnetic stimulation, ACLR: Anterior cruciate ligament reconstruction, ACLD: Anterior cruciate ligament deficient, EMG: Electromyography, AMI: Arthrogenic muscle inhibition, SICI: Short-interval intracortical inhibition, ICF: Intracortical facilitation, MVIC: Maximal voluntary isometric contraction, CAR: Central activation ratio, AMT: Active motor threshold, CsP (CSP): Cortical silent period, EEG: Electroencephalography, fMRI: Functional magnetic resonance imaging, H/M ratio: Hoffmann reflex to M-wave ratio, MEP: Motor evoked potential, MRI: Magnetic resonance imaging, RMT: Resting motor threshold, SR curve/SR slope: Stimulus-response curve/slope

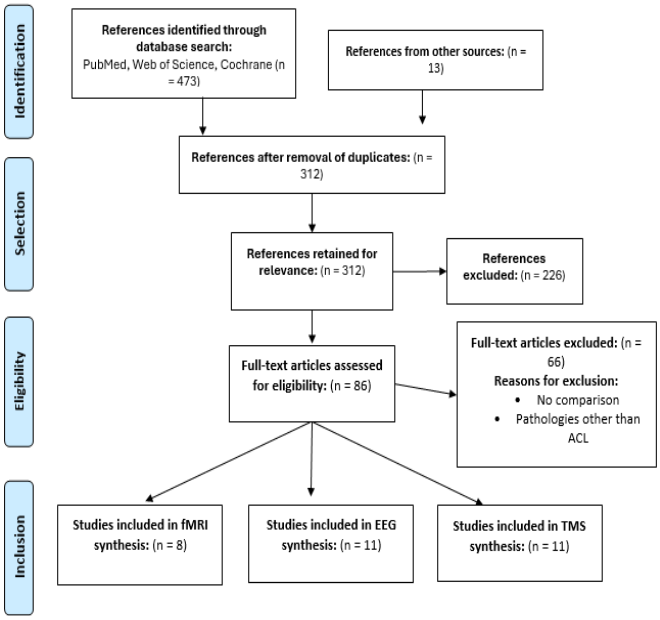

Figure 1. PRISMA diagram of studies included in the pathophysiology of AMI

PRISMA: Preferred reporting items for systematic reviews and meta-analyses, AMI: Arthrogenic muscle inhibition, ACL: Anterior cruciate ligament, fMRI: Functional magnetic resonance imaging, EEG: Electroencephalography, TMS: Transcranial magnetic stimulation

Therapeutic Strategies Targeting AMI

The search for studies addressing therapeutic strategies targeting AMI following ACL injury was conducted in PubMed, Web of Science, and the Cochrane Library, yielding 242 references, supplemented by 21 additional articles from other sources. After duplicate removal, 202 unique references were retained and screened for relevance based on titles and abstracts. At this stage, 145 articles were excluded for lack of direct relevance to AMI management. A total of 57 full-text articles were then reviewed for eligibility. Among these, 24 studies were excluded-primarily due to the absence of quadriceps strength assessments or inclusion of populations unrelated to ACL pathology (e.g., patients with prosthetic knees). Finally, 20 studies[47-66] (**Table 4**) met the inclusion criteria and were included in the synthesis. Given the limited number of comparative studies available, all identified articles were included, regardless of study design, and comparison group presence was not considered an exclusion criterion in this section of the analysis. This rigorous selection process enabled the construction of a focused and comprehensive narrative synthesis, as illustrated in the PRISMA flow diagram (**Figure 2**).

Table 4. Summary of strategies targeting AMI in ACL reconstruction patients

Author/year	Population	Treatment	Measurement	Results
Zarzycki[47]	20 ACLR	One session of transcranial direct-current stimulation (tDCS)	- Corticospinal excitability: AMT, SR slope - Dynamometer: Strength	No significant difference observed
Dos Anjos[48]	30 ACLR	Neuromotor program (motor imagery+low-frequency sound stimulation)	- Electromyographic activity - Knee extension deficit - Subjective knee value	- EMG activity significantly increased by 45% - Knee extension deficit significantly improved - SKV significantly increased
Khan[49]	60 ACLR	TENS; in 2 groups: - TENS therapy+exercises and ice packs (n=30) - Exercises+ice packs only (n=30)	- Dynamometer: Strength - Pain: VAS (Visual Analog Scale) - Function: Lysholm and IKDC scores - Thigh circumference	- Significantly lower VAS pain scores, greater muscle strength, and better Lysholm and IKDC scores in the TENS group - No significant difference in mean thigh circumference
Sherman[50]	20 ACLR vs. 20 healthy controls	External focus during single-leg stance under four conditions: - TENS - External focus based on an object - External focus based on a target - Internal focus	EEG: pectral analysis of theta and alpha activity	- ACLR individuals showed higher motor-planning activity, reduced sensory activity, and decreased motor activity - External focus based on a target reduced motor-planning activity and increased bilateral visual, sensory, and motor activity - No significant differences between other groups
Kim[51]	13 ACLR vs. 13 healthy	Effect of kinesiophopia → Observations of an action during a vertical drop jump	- fMRI - TSK 11: Psychological fear score	- Lower brain activity in the right ventrolateral prefrontal cortex, including the precentral gyrus, middle frontal gyrus, and corticospinal tract in the ACLR group - Higher TSK-11 scores in the ACLR group
Bodkin[52]	10 ACLR	Visuomotor therapy (visual biofeedback)	- TMS: MEP	Significant increase in MEP
Rush[53]	10 ACLR	tDCS: One session of transcranial direct-current stimulation	- EMG: MVIC - Pain and symptoms	No significant difference after a single session
Mendias[54]	19 ACLR	Hormone therapy: Growth hormone, two groups: - Treatment group: 9 - Control group: 10	- Dynamometer: Muscle strength - MRI: Muscle volume -PROMs	- Significant increase in muscle strength in the treatment group - No significant difference in muscle volume or PROMs
An[55]	20 ACLR vs. 20 healthy	Psychological effect → Response to three types of emotionally evocative negative images (fear or knee-injury-related images)	- EEG: Frontal and parietal electrodes - ROM	- Increased frontal and parietal spectral frequency in the ACLR group - Increased rigidity in the ACLR group
Lepley[56]	5 ACLR	Cross-eccentric exercises (nondominant limb)	- Brain activity: fMRI - EMG: H-reflex - TMS: AMT, MEP - PROMs: Pain, symptoms, function	- fMRI: Reduced neuronal activity in the frontal cortex - Significant increase in H-reflex and MEP - Significant improvements in PROMs
Lowe[57]	9 ACLR vs. 9 healthy	Hamstring fatigue → Induced by performing squats	-CAR	Significant increase in CAR in the ACLR group
Oliveira[58]	47 ACLR	Kinésio-taping, 3 groupes: - Control group - Placebo group - Taping group	- EMG/dynamometer: CAR - MVIC - Baropodometry: Postural balance	No significant difference
Lepley[59]	36 ACLR vs. 10 healthy	NMES/eccentric exercises, 4 groups: 1-NMES+eccentric exercises 2-Eccentric exercises only 3-NMES only 4-Standard rehabilitation	- MVIC - CAR	- CAR: Higher in the eccentric-exercise-only group - MVIC: Higher in the NMES and eccentric-only groups
Hart[60]	30 ACLR	Cryotherapy 3 groupes: 1-20 minutes of cryotherapy 2-One hour of rehabilitation exercise. 3-Cryotherapy followed by exercises	- MVIC - Reflex H/M	- Significant increase in MVIC in the cryotherapy+exercises group - No significant difference in H/M reflex
Baltaci[61]	30 ACLR	Biofeedback+motor imagery (Nintendo Wii Fit) Two groups: - Treatment group: 15 - Control group: 15	- Dynamometer: Knee strength - Coordination tests - Functional test: Squat	No significant difference
Maddison[62]	21 ACLR	Motor imagery (visual+kinesthetic), two groups: - Treatment group: 13 - Control group: 8	- Dynamometer: Knee strength - KT1000 arthrometer: Laxity - Psychological effect (Questionnaire-AIESQ)	- No significant difference in knee strength or in the AIESQ questionnaire - Significantly lower laxity in the treatment group
Lebon[63]	12 ACLR	Kinesthetic motor imagery, two groups: - Treatment group: 7 - Control group: 5	- EMG: Electromyographic activity - LEFS to assess ability to perform daily activities - Anthropometric data: joint ROM, effusion resorption - Pain: VAS	- Greater electromyographic activity in the treatment group - No difference in anthropometric measures, LEFS scores, or VAS pain
Ohta[64]	44 ACLR	Blood flow restriction Two groups: - Treatment group: 22 - Control group: 22	- Dynamometer: Muscle strength - Pre/post ratio of quadriceps cross-sectional area	- Significant increases in muscle strength. - Significant improvement in the preoperative/postoperative ratio of quadriceps cross-sectional area at 16 weeks in the treatment group.
Cupal[65]	30 ACLR	Intervention: Motor imagery (visual+kinesthetic) three groups - Treatment group: 10 - Standard physiotherapy group: 10 - Placebo group: 10	- Dynamometer: Muscle strength - Reinjury anxiety (questionnaire) - Pain	- Significantly higher muscle strength in the treatment group. - Significantly lower pain and reinjury anxiety in the treatment group.
Draper[66]	30 ACLR	EMG Biofeedback/TENS, two groups: -TENS group: 15 -EMG biofeedback group: 15	-Dynamometer: Muscle strength	The biofeedback group recovered significantly greater muscle strength compared to the electrical stimulation (TENS) group.

AMI: Arthrogenic muscle inhibition, ACL: Anterior cruciate ligament, ACLR: Anterior cruciate ligament reconstruction, tDCS: Transcranial direct current stimulation, TENS: Transcutaneous electrical nerve stimulation, NMES: Neuromuscular electrical stimulation, AMT: Active motor threshold, SR slope: Stimulus-response slope, EEG: Electroencephalography, fMRI: Functional magnetic resonance imaging, AIESQ: Athletic Injury Self-Efficacy Questionnaire, BFR: Blood flow restriction, CAR: Central activation ratio, EMG: Electromyography, H/M ratio: Hoffmann reflex to M-Wave ratio, IKDC: International Knee Documentation Committee score; KT: Kinesio taping, MEP: Motor evoked potential, MRI: Magnetic resonance imaging, VMO: Vastus medialis oblique, MVIC: Maximal voluntary isometric contraction, PROMs: Patient-reported outcome measures, ROM: Range of motion, SKV: Subjective knee value, TMS: Transcranial magnetic stimulation, TSK-11: Tampa Scale of Kinesiophobia-11 items, LEFS: Lower Extremity Functional Scale, VAS: Visual Analog Scale

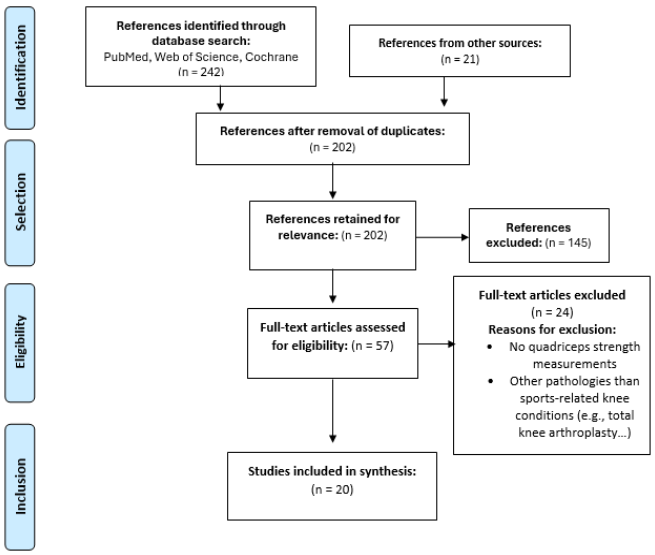

Figure 2. PRISMA diagram of studies included in the review of therapeutic strategies targeting AMI

PRISMA: Preferred reporting items for systematic reviews and meta-analyses, AMI: Arthrogenic muscle inhibition

DISCUSSION

AMI is a long-recognized neurophysiological phenomenon first described in 1965, when researchers demonstrated that experimentally induced knee effusion produced reflexive quadriceps inhibition through altered capsular feedback.[67] Subsequent work in the 1980s confirmed that the magnitude of inhibition was proportional to the volume of effusion, highlighting the strong link between disrupted joint afference and reduced muscle activation.[68,69] Although the association between reflex inhibition and muscle weakness was known for decades, AMI was formally defined only in 2000 by Hopkins and Ingersoll as a persistent reflex inhibition of the alpha-motoneuron resulting from altered afferent input from an injured joint.[70] Since this definition, research on AMI-particularly in the context of ACL injury-has advanced substantially.

Neurophysiopathology of AMI

Current evidence indicates that AMI is a multifactorial process involving interacting local, spinal, and supraspinal mechanisms.[71] Local factors such as damaged proprioceptors, inflammatory "biochemical soup," joint effusion, and mechanical instability disrupt sensory transmission to the central nervous system.[72-75] These altered afferents contribute to higher-level changes, including disrupted sensory integration (EEG), altered cortical connectivity (fMRI), and impaired descending motor drive (TMS), ultimately resulting in deficits in voluntary quadriceps activation.[71]

Sensory afferent pathway (EEG): Eleven studies investigated sensory afferent pathways in individuals with ACL injury using EEG, including power spectral analysis and somatosensory evoked potentials (SEP).[17-27] EEG provides an initial window into supraspinal mechanisms contributing to AMI by capturing cortical responses to altered proprioceptive input.

Across studies, spectral analysis consistently demonstrated increased frontal theta activity during sensorimotor tasks, interpreted as enhanced engagement of cortical networks supporting anticipatory motor control in the context of reduced proprioceptive reliability.[17,18,20-22] Several investigations also reported increased parietal alpha activity-typically associated with cortical deactivation-suggesting challenges in sensory processing or attentional allocation.[19] Conversely, reduced posterior alpha observed in ACL-reconstructed individuals during complex or dynamic tasks indicates a greater reliance on visual processing to compensate for sensory-motor deficits.[17]

SEP findings further characterized the integrity of afferent pathways by examining cortical responses following peripheral nerve or ACL stimulation. Although the overall transmission of sensory information to the cortex appeared preserved, studies consistently noted reduced reproducibility and stability of SEP waveforms in individuals with ACL injury.[23-27] These alterations suggest that despite intact structural pathways, the quality and consistency of proprioceptive input reaching the somatosensory cortex are compromised. Such disruptions in afferent signal processing likely contribute to the central component of AMI and may influence subsequent motor control adaptations

Cortical connectivity (fMRI): Neuroplastic reorganization following ACL injury reflects the brain's adaptive response to altered sensory-motor input. Functional MRI provides a window into these adaptations by quantifying blood-oxygen-level-dependent (BOLD) activity and mapping functional connectivity within networks responsible for motor planning and execution. Across eight comparative studies, consistent patterns of neurofunctional change have been identified in individuals with ACL injury, using both resting-state and task-based paradigms.[28-35]

A primary finding is the reduced activation of key motor and sensory regions, particularly the primary motor cortex (M1), primary somatosensory cortex (S1), and supplementary motor area (SMA).[28,29,34] This hypoactivation is commonly interpreted as impaired descending motor drive or a persistent centrally mediated inhibitory state, likely arising from partial deafferentation following ligament injury. At the same time, increased activation within associative and visual regions-including the thalamus, superior parietal cortex, angular gyrus, and inferior parietal lobule-suggests a compensatory shift toward heightened multisensory integration and cognitive control.[30,31] These changes indicate that individuals may rely more heavily on visual and higher-order cortical processing to compensate for degraded proprioceptive input during lower-limb tasks.[34]

Alterations in functional connectivity complement these activation patterns. Several studies report decreased connectivity between sensory and motor regions, such as between S1 and SMA or between premotor areas and M1, reflecting a disruption of motor network organization.[29,32,33] Conversely, increased connectivity within cognitive and premotor circuits has been observed, which may represent adaptive recruitment of alternative pathways to support movement planning and execution.[34,35] Together, these findings support the concept of a reorganized but less efficient motor control strategy, potentially contributing to persistent AMI and long-term deficits following ACL injury.

Motor efferent pathway (TMS): Eleven studies have used TMS to investigate alterations in efferent motor pathways following ACL injury.[36-46] TMS, typically combined with

EMG and dynamometry, allows simultaneous evaluation of corticospinal excitability, motor output, and voluntary activation.

Across studies, individuals with ACL injury consistently demonstrate reduced corticospinal excitability, evidenced by elevated active motor thresholds (AMT) and diminished motor evoked potentials (MEP) amplitudes in the injured limb compared with the contralateral side or healthy controls.[42,44,45] These findings suggest weakened descending drive and impaired transmission along the corticospinal tract. Evidence regarding intracortical mechanisms is more variable: while several studies report no significant changes in short-interval intracortical inhibition (SICI) or intracortical facilitation (ICF), others identify prolonged cortical silent period (CSP) duration, consistent with enhanced intracortical inhibition and a persistent limitation in motor output.[37,38]

Three recent meta-analyses provide additional clarity and reinforce the strength of the evidence. Across 13-14 studies and samples ranging from 381 to 611 participants, they consistently report bilaterally elevated AMT, reflecting a generalized decrease in cortical excitability after ACL injury. MEP amplitudes demonstrate asymmetrical reorganization, typically reduced in the injured limb while relatively preserved in the uninjured limb, suggesting compensatory redistribution of descending motor drive.[76-78]

Synthesis: AMI arises from the interaction between peripheral joint pathology and central nervous system adaptations following ACL injury. Locally, damage to ligamentous mechanoreceptors combined with pain, effusion, and inflammation disrupts proprioceptive afferent input and triggers protective inhibitory reflexes. EEG studies show altered cortical processing of this degraded sensory information, characterized by increased frontal activity and changes in sensorimotor rhythms, indicating greater cognitive demand and reduced efficiency of motor control. fMRI findings further reveal maladaptive cortical reorganization, with decreased activation of primary sensorimotor areas and compensatory recruitment of visual and associative regions. These supraspinal alterations translate into impaired descending motor drive, as confirmed by TMS studies demonstrating reduced corticospinal excitability and altered intracortical inhibition. Together, these local and central mechanisms explain the persistent inability to fully activate the quadriceps despite preserved muscle structure.

Therapeutic Strategies Targeting AMI

Most studies addressing AMI focus on individuals with ACL injury or reconstruction (43.9%) and almost exclusively target the knee joint (97.4%), highlighting the central role of AMI in knee pathology. Therapeutic interventions predominantly address the quadriceps, which is particularly susceptible to AMI following ACL reconstruction, whereas the hamstrings are less frequently targeted, mainly due to their contribution to dynamic knee stability and co-activation with the quadriceps.[14]

To improve clinical recognition and management, Sonnery-Cottet and colleagues[10] proposed in 2022 a graded clinical classification of knee AMI based on vastus medialis oblique (VMO) activation and knee extension deficit. This system distinguishes reversible forms that respond to simple in-clinic exercises from more severe forms requiring prolonged, targeted rehabilitation or, in chronic cases, surgical intervention. Early identification and treatment of reversible AMI are critical to restoring quadriceps function and preventing persistent deficits. Clinical management focuses on reducing reflexive inhibition through hamstring fatigue, addressing hemarthrosis when present, and re-establishing active quadriceps and VMO activation to restore full knee extension.[10] A prospective 2024 study of 300 patients with recent ACL injuries (<6 weeks) found that over 55% exhibited AMI, with nearly 80% being easily reversible through simple exercises; more severe forms (grades 1B and 2B) accounted for approximately 12% and required longer, specific rehabilitation.[5]

Overall, AMI is progressively reversible but leaves a subset of motoneurons difficult to recruit voluntarily, reinforcing the need for phase-specific neuromuscular interventions. In this review, therapeutic strategies are structured according to the recovery phases after ACL injury, following the framework of Norte et al.,[14] which considers both time and the evolving neurophysiological state of the patient, according to their mechanisms of action on neuromuscular inhibition. This phase-based organization is intended to facilitate clinical interpretation; however, rehabilitation progression in practice is often criterion-based rather than strictly time-dependent, and several interventions may overlap across recovery phases depending on individual patient presentation and rehabilitation goals.

Acute phase: 0-6 weeks: During the acute phase, AMI is maximal, primarily due to pain, edema, and inflammation. Voluntary quadriceps activation, as measured by the central activation ratio (CAR), can drop below 20%.[7,70] Despite a structurally intact muscle, ACL injury or reconstruction disrupts proprioceptive signaling through mechanoreceptor damage and nociceptive afferents. This triggers protective inhibitory reflexes at both the spinal and cortical levels, limiting motoneuron availability.[14] Early rehabilitation aims to reduce pain, effusion, and inflammation; promote muscle activation; and limit atrophy.

Cryotherapy: Targeted cryotherapy around the joint appears to attenuate AMI by modulating nociceptive and thermosensory afferents, enhancing motoneuron excitability and voluntary muscle activation.[79] It reduces nociceptor activity while stimulating thermoreceptors, decreasing inhibitory signals transmitted via small-diameter afferents and facilitating motor disinhibition.[80] Typically, ice or a cooling device is applied circumferentially for 20-30 minutes immediately prior to therapeutic exercises. Hart et al.[60] demonstrated that combining cryotherapy with exercise significantly improved quadriceps strength (MVIC) without affecting the H/M reflex.

Transcutaneous electrical nerve stimulation (TENS): TENS is frequently used in the acute phase to limit AMI. By modulating sensory afferent pathways, particularly small-diameter fibers, TENS can mask nociceptive signals responsible for reflexive motoneuron inhibition. This transient disinhibition helps restore motor pool excitability and facilitates voluntary quadriceps activation.[81] Sessions of 20-30 minutes at high frequency (120-150 Hz) before

or during exercise appear optimal. Khan et al.[49] reported reductions in pain (VAS) and improvements in muscle strength and functional scores (IKDC, Lysholm) when TENS was combined with exercise.

Neuromuscular electrical stimulation (NMES): NMES is widely used early in rehabilitation to bypass AMI. Its primary goals are to enhance intramuscular blood flow, maximal force, endurance, and prevent early atrophy.[82] NMES stimulates intramuscular nerve branches to elicit muscle contractions. Repeated depolarization of motor neurons produces involuntary maximal contractions to limit atrophy in inhibited muscles.[83] NMES is most effective in the early recovery phase, with diminishing returns as voluntary strength and activation improve.[82] Combined NMES and exercise, particularly eccentric training, has been shown to significantly increase quadriceps MVIC.[59]

Cross-education eccentric exercises: Cross-education exercises performed on the contralateral limb can stimulate muscle activation on the injured side via supraspinal mechanisms. This approach relies on interhemispheric communication activated during voluntary contractions of the healthy limb.[84] In the acute phase, when loading the injured joint is limited, cross-education offers a safe method to maintain neuromuscular excitability.[82] Lepley et al.[56] reported that a 4-week intervention increased H/M reflex and MEP responses and improved functional scores despite no direct work on the operated limb.

Blood flow restriction (BFR) training: BFR is a valuable option during the acute phase when high loads are contraindicated. By inducing localized muscular hypoxia, BFR activates metabolic pathways that promote anabolic growth factors, fast-twitch fiber recruitment, and myostatin inhibition, thereby limiting muscle atrophy.[86] Ohta et al.[64] demonstrated that a 16-week BFR program significantly increased quadriceps strength and cross-sectional area without heavy resistance. While BFR does not directly address AMI mechanisms, it supports early muscle preservation.

EMG biofeedback: EMG biofeedback provides patients with real-time feedback on muscle activation, enhancing conscious control over descending motor pathways.[87] This voluntary cortical engagement helps bypass spinal inhibitory reflexes and restore quadriceps activation. A 2023 meta-analysis of eight studies conducted 1-16 weeks post-ACL reconstruction found EMG biofeedback improves quadriceps strength and knee extension, though effects on pain and Lysholm scores were limited.[88] Draper et al.[66] reported significantly greater strength recovery with EMG biofeedback compared to TENS, and Pietrosimone et al.[42] observed increases in MEP and muscle strength in healthy subjects after isometric training with EMG-BF, suggesting a corticospinal activation effect.

Subacute phase: 6 weeks-6 months: During the subacute phase, AMI gradually decreases but remains present. Some motoneurons regain availability; however, the motor system often struggles to recruit them effectively, a phenomenon known as central activation failure.[42,59] Despite apparent physical recovery, voluntary quadriceps activation (CAR) often remains below 50%,[42,45] largely due to persistent cortical alterations detected by TMS, even when the muscle is structurally intact. Pain, effusion, and inflammation typically diminish during this phase. The main goals are to progressively increase joint load, continue muscle strengthening, and enhance voluntary activation.

Antagonist muscle fatigue: After ACL injury, antagonist muscles may become hyperactive via supraspinal mechanisms to stabilize the joint and protect intra-articular tissues. This overactivation inhibits agonist activity, such as the quadriceps, through reciprocal inhibition.[89] Voluntary fatigue of the hamstrings reduces this antagonistic inhibition, facilitating quadriceps activation.[90] While primarily applied in the subacute phase, antagonist fatigue can also be useful in the acute phase to counter early spinal reflex inhibition. Lowe et al.[57] demonstrated a significant increase in CAR following targeted antagonist fatigue exercises in ACL patients.

Eccentric exercises: Eccentric training targeting strength and voluntary activation effectively improves CAR, a key indicator of quadriceps activation after ACL injury.[14] Lepley et al.[59] reported that participants performing only eccentric exercises had significantly higher CAR compared with other groups, indicating enhanced voluntary activation via supraspinal and spinal excitatory pathways. Integrating eccentric exercises into ACL rehabilitation can therefore restore neuromuscular activation, essential for regaining quadriceps strength and function.

Kinesio taping (KT): KT involves applying elastic tape to the skin (e.g., quadriceps in an inverted Y configuration) to stimulate cutaneous receptors and facilitate muscle activation. Oliveira et al.[58] found that, at 4 months post-ACL reconstruction, KT alone did not significantly improve MVIC or CAR in patients with AMI. This suggests limited efficacy in advanced rehabilitation but supports its use in the acute phase to enable earlier quadriceps activation when pain, edema, and reflex inhibition predominate.[91]

Return-to-sport phase: >6 months: Beyond six months, AMI can persist despite structural recovery. Lepley et al.[59] reported that 30-40% of patients still exhibit significant AMI one-year post-surgery. Although quadriceps volume is often restored, neuromuscular activation quality remains compromised. Compensatory cortical strategies, including overactivation of premotor or visual cortical regions and recruitment of alternative muscle groups, can partially mask deficits.[37,50]

Motor imagery: Motor imagery-visual or kinesthetic simulation of movement without physical execution-engages mirror neurons, activating motor regions similarly to actual movement. This strengthens corticospinal circuits and facilitates voluntary motoneuron recruitment even without physical movement.[92] Cupal et al.[65] reported significant gains in muscle strength and reductions in pain and fear of reinjury after 6 weeks of motor imagery. Lebon et al.[63] observed increased EMG activity, while Maddison et al.[62] demonstrated reduced joint laxity. More recently, Dos Anjos and colleagues,[48] using the Allyane method, reported a 45% increase in EMG activity, a significant improvement in knee-extension deficit, and a significant increase in subjective knee value (SKV), further supporting the neuromuscular benefits of motor imagery-based interventions. Although results across studies remain heterogeneous, motor imagery is safe, noninvasive strategy that can complement conventional rehabilitation. While findings are encouraging, the current level of evidence for motor imagery remains moderate and somewhat heterogeneous, with most studies involving small sample sizes and variable protocols.

Visuomotor therapy: Bodkin et al.[52] showed that a single session of visuomotor therapy elicited a greater increase in quadriceps corticospinal motor response than sham therapy, suggesting that visuomotor training may serve as an effective adjunct when upregulation of corticospinal excitability is desired. Baltaci et al.[61] investigated Nintendo Wii Fit-based rehabilitation as an interactive, visually guided approach to balance and functional training that emphasizes visual feedback, external cues, and sensorimotor integration. Wii Fit-based rehabilitation resulted in functional outcomes comparable to conventional rehabilitation after ACL reconstruction, with greater improvements in balance-related measures. However, this intervention did not provide direct evidence of neuromodulatory effects on quadriceps strength or AMI. Evidence supporting visuomotor therapy remains preliminary, and additional controlled studies are needed to determine its specific effects on AMI and quadriceps neuromuscular recovery.

Supraphysiological hormone therapy: Experimental interventions using growth hormone aim to limit AMI by targeting post-traumatic muscle catabolism when voluntary activation remains limited. Mendias et al.[54] reported increased muscle strength post-ACL reconstruction with growth hormone treatment, though effects on muscle volume and functional scores were minimal. Benefits may relate to reduced myostatin expression and modulation of anabolic pathways. Due to low evidence, regulatory limitations, and potential systemic side effects, this approach remains experimental and insufficient to support routine clinical use in AMI management.

Attentional focus: Emerging evidence suggests that external attentional focus (directed toward an object or target) may enhance voluntary quadriceps activation and facilitate motor recruitment.[93] Focusing externally improves motor performance and reduces cortical inhibition more effectively than internal focus. Sherman et al.[50] compared different unipedal stance conditions in ACL patients and found that external target-focused attention decreased motor planning activity while increasing bilateral sensory-motor activation, promoting more automatic and efficient movement. Targeting patient attention to external goals during exercise is a simple yet effective neuromuscular strategy in late-phase ACL rehabilitation.

Transcranial direct current stimulation (tDCS): tDCS is a noninvasive method to modulate cortical excitability, especially in the primary motor cortex (M1), with the goal of enhancing voluntary motoneuron recruitment via descending pathways. Typical application involves 1-2 mA anodal stimulation for 20 minutes over the motor region contralateral to the injured limb.[47,53] Rush et al.[53] reported no significant improvement in quadriceps activation or pain after a single tDCS session during treadmill walking 39 months post-ACL reconstruction. Zarzycki et al.[47] similarly observed no significant effects on strength, H/M reflex, or MEPs during cycling at 6 months post-surgery, though tDCS was well tolerated. While feasible and safe, isolated tDCS efficacy remains limited and inconclusive, despite its feasibility and favorable safety profile.

Psychological factors: Psychological factors strongly influence neuromuscular recovery after ACL reconstruction. Kinesiophobia or fear of movement can disrupt motor circuits and perpetuate muscle inhibition. Kim et al.[51] found heightened activation in fear-related brain regions (amygdala, cerebellum) during observation of risky movements, correlating with high kinesiophobia scores. An et al.[55] demonstrated increased joint stiffness and elevated frontal and parietal spectral frequency in response to traumatic knee imagery. Assessing these aspects using tools like the ACL-RSI (Anterior Cruciate Ligament-Return to Sport after Injury) scale helps address confidence, emotions, and motivation.[94] Accounting for psychological barriers is essential for optimal neuromuscular recovery and sustainable return to sport.

Limitations

This scoping review has several limitations. The included studies were highly heterogeneous in terms of experimental paradigms, neurophysiological assessment methods (EEG, fMRI, and TMS), and clinical outcome measures, which limited direct comparison across studies and precluded quantitative synthesis. Most studies used cross-sectional designs, restricting interpretation of causal relationships between neurophysiological alterations and quadriceps dysfunction.

Additionally, definitions and classifications of AMI varied across studies, and AMI was often inferred indirectly rather than measured using standardized criteria. Substantial heterogeneity in patient characteristics across studies-including time since injury or surgery, graft type, concomitant injuries, gender, athletic level, pain status, and rehabilitation exposure-likely contributed to variability in neurophysiological and clinical findings and may partially explain inconsistencies between studies. Intervention studies rarely integrated neurophysiological outcomes with functional or strength-based measures, limiting conclusions regarding mechanistic links between cortical changes and clinical recovery.

Furthermore, the therapeutic evidence should be interpreted with caution, as many intervention studies were based on small sample sizes, heterogeneous rehabilitation protocols, or exploratory designs. Consequently, some interventions discussed in this review should be considered promising but preliminary, and their clinical effectiveness requires confirmation through larger, high-quality comparative and longitudinal studies.

Finally, as a scoping review, this work aimed to map and summarize the existing literature rather than formally assess methodological quality or risk of bias, and relevant studies may have been missed despite a comprehensive search strategy.

CONCLUSION

AMI is a multilevel reflex-mediated neural disorder that limits muscle activation despite preserved structural integrity. After ACL injury, alterations detected with EEG, fMRI, and TMS indicate impaired motor control and disrupted brain-muscle communication, contributing to persistent weakness beyond simple atrophy or deconditioning. These findings support the concept that effective rehabilitation should not rely solely on strength training but should also incorporate targeted neuromuscular interventions addressing both

peripheral and central mechanisms of inhibition. Clinically, early identification and classification of AMI using functional grading systems may help identify reversible forms amenable to immediate treatment. More persistent presentations require an individualized, mechanism- and recovery stage-based rehabilitation approach that considers motoneuron availability, voluntary recruitment capacity, pain, proprioceptive deficits, and psychological factors to optimize recovery and reduce long-term dysfunction. Future longitudinal and comparative studies are needed to clarify the causal relationships between neurophysiological alterations, AMI persistence, and functional recovery after ACL injury or reconstruction.

Findings

- Neurophysiological studies demonstrate that Arthrogenic Muscle Inhibition after ACL injury involves altered afferent input, cortical reorganization, and reduced corticospinal excitability, not solely peripheral quadriceps dysfunction.
- Intervention studies indicate that quadriceps activation and strength can be improved through phase-specific strategies, particularly when interventions target both neural and muscular mechanisms.

Implications

- These findings expand current understanding of AMI as a multi-level neurophysiological condition and support rehabilitation approaches that extend beyond traditional strengthening to include cortical and sensorimotor retraining.
- Clinicians should consider early identification of AMI and integrate neuromuscular and neurocognitive strategies to optimize quadriceps recovery after ACL injury.

Caution

- Most included studies were small, heterogeneous in design and outcome measures, which may limit generalizability to broader clinical populations and real-world rehabilitation contexts.

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