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REVIEW

Manipulation under anesthesia following total knee arthroplasty: a comprehensive review of literature

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Abstract

Introduction The etiology of the stiff knee after total knee arthroplasty (TKA) is largely unknown, although excessive scar tissue due to arthrofibrosis is an important reason for a limited range of motion (ROM) after this procedure. Persistent limited ROM after TKA results in poor patient-reported outcomes and is increasingly becoming a more prominent reason for TKA revision surgery.

Methods A narrative review of current literature on manipulation under anesthesia (MUA) after TKA analyzing etiology and risk factors for stiffness after TKA, effectiveness of MUA and what is known about rehabilitation after MUA.

Results Literature describes numerous risk factors for insufficient knee ROM after TKA, but a comprehensive valid risk model is lacking. MUA is an effective treatment option with evidence suggesting better outcomes if performed within the first 3 months after TKA. The wide variety in both the indication and timing for MUA, and the lack of scientific evidence on how to rehabilitate patients after MUA, complicates the interpretation of available literature. This is even more so the case on the reporting of one versus two or more MUAs after TKA.

Conclusion Future comparative trials, preferably with a randomized study design, should be conducted to elude more clear indications for MUA, to give clinical guidance on correct timing for MUA and on how to rehabilitate patients afterward.

Keywords Manipulation under anesthesia · Total knee arthroplasty · Etiology · Risk factors · Effectiveness · Rehabilitation

Introduction

With improvements in prosthesis materials in total knee arthroplasty (TKA), there is a shift from implant loosening toward other reasons for revision surgery after TKA, including knee stiffness [1]. Stiffness can be defined as limited ROM that inhibits optimal TKA function and affects a patient's ability to perform activities of daily living, thereby reducing quality of life [2].

Although the exact etiology of the stiff knee after TKA is unknown, a large number of risk factors for developing this complication are described in the literature (Table 1). And despite modern implant designs, improved surgical technique and optimized pain treatment permitting physiotherapy to start within hours after the procedure, manipulation under anesthesia (MUA) is needed in 1.3–13.5% of all TKA cases to restore knee range of motion (ROM) [3–10].

Both in clinical practice and scientific literature, there is limited attention for MUA. If recovery of ROM after TKA is delayed, patients have unsatisfactory knee function for weeks up to several months after initial surgery or even permanent. Of all readmissions after TKA, 6.6–36.1% is for MUA [11, 12], thereby increasing health-care costs considerably. MUA after TKA also requires a secondary anesthetic event and may result in complications related to both anesthesia and the manipulation procedure itself [8]. Clear criteria on the indication and timing of MUA are unknown [2], and although MUA is effective in improving ROM [13], in some cases not all gained ROM is sufficient or maintained in the long term, resulting in unsatisfactory outcome after TKA.

In scientific literature, evidence on the optimal rehabilitation after MUA is even more absent. Several authors

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Table 1 Risk factors limited ROM after TKA

Patient-related	
Age	Increased risk with younger patients [3, 6, 22, 34–37]
	Increased risk with patients aged > 70 years [56]
	No relationship [4, 7, 61]
Gender	Increased risk for female patients [9, 34, 36, 56, 61]
	Increased risk for male patients [6]
	Increased risk for flexion deficit for females and extension deficit for males [51]
	No relationship [4, 7, 35, 37, 50]
BMI	Increased risk for overweight or obese patients [36, 51, 61]
	No relationship [6, 22, 35, 37]
Diagnosis	Increased risk with RA compared to OA [56]
	Increased risk with knee osteonecrosis compared to OA [35]
	No relationship [4, 7, 34, 62]
Pre operative ROM	Increased risk with limited preoperative ROM [3, 7, 33–35]
	No relationship [22]
Pain	Increased risk with higher preoperative pain [3]
Previous knee surgery	Increased risk with previous knee surgery [19, 37, 63]
	No relationship [4]
Previous stiffness	Increased risk with stiffness after contralateral TKA [64]
Smoking	Increased risk for smoking patients [6, 35–37]
Kinesiophobia	Increased risk with kinesiophobia [32]
	No relationship [48]
Diabetes mellitus	Increased risk with diabetes [35, 65]
	No relationship [36, 37]
Surgical-related	
Surgical approach	No influence of surgical approach [4]
Component sizing	Increased risk with oversized component [2, 19, 31]
	Increased risk if the AP knee diameter increases ≥ 12% [22]
Component positioning	Increased risk with component malpositioning [2, 28, 31, 38, 39]
Tibial slope	Increased risk if tibial slope is changed from preoperative value [21]
	Increased risk with positive slope (posterior higher than anterior) [2, 31, 38]
Elevated joint line	Increased risk with elevated joint line [21, 38, 39]
Patellofemoral reconstruction	Increased risk with malpositioning [39], with limited translation or tilting [28] or with oversizing of the patellar component [2, 31, 66]
Osteophytes	Increased risk with inadequate osteophyte removal [2, 28, 31, 34, 38]
Soft tissue balancing	Increased risk with improper balance between flexion and extension gap [18, 28, 38]
PCL tension	Increased risk with high PCL tension [2, 28, 67]
Intra-operative ROM	Increased risk with decreased intra-operative ROM [31, 34, 62]
Component fixation	Increased risk with uncemented knee prosthesis [19]
Implant design	No relationship [7, 35, 40–42]
	Increased risk with specific designs [43–46]
Postoperative	
Postoperative pain	Possible increased risk with high postoperative pain [4]
Compliance	Increased risk with poor physiotherapy compliance [2, 28, 31]
Arthrofibrosis	Increased risk with arthrofibrosis [22–26]
Heterotopic ossification (HO)	Increased risk with advanced HO (> 5 cm) [68, 69]
	No relationship [22]
DVT/PE prophylaxis	Increased risk with aggressive DVT/PE prophylaxis [4, 9, 49]
Length of stay (LOS)	Increased risk with shorter LOS [8]
	LOS does not influence postoperative ROM [6, 37]

BMI body mass index, *RA* rheumatoid arthritis, *OA* osteoarthritis, *ROM* range of motion, *TKA* total knee arthroplasty, *PCL* posterior cruciate ligament, *DVT* deep venous thrombosis, *PE* pulmonary embolism

describe protocols for rehabilitation after MUA, which usually includes adequate pain control, ROM exercises, continuous passive motion machines (CPM) and cooling [3–5, 13–21], but a sound scientific basis for rehabilitation after MUA is lacking.

In this narrative review, we analyzed literature on the etiology of the stiff knee after TKA, on risk factors for knee stiffness leading to MUA, on the effectiveness of MUA and what is known about rehabilitation after MUA.

Pathogenesis of the stiff knee after TKA

Why a knee after TKA remains stiff is largely unknown, and considering the numerous risk factors described in literature (Table 1), it is most likely multifactorial and/or very diverse in cause. In the early postoperative period, persistent stiffness is commonly thought to be a result of excessive scarring due to arthrofibrosis, resulting in soft tissue contractures [22–26].

Excessive scar tissue formation with bands of dense, motion-limiting fibrous tissue and adhesions can form between the quadriceps muscle and the distal femur, occupying the suprapatellar pouch and medial and lateral gutters [21, 27, 28]. These bands of scar tissue prevent excursion of the quadriceps muscle when flexing the knee and cause a decrease in patellofemoral mobility [21]. Adhesive tissue matures with time and organizes during the first 6 months, but the degree of loss of motion is not related to the maturity of the tissue [29]. Possibly MUA is more effective in the first few months because there is less adhesive tissue in the knee [30]. Mariani et al. [29] postulated that tissue with less maturity has a lower resistance and is easier to overcome with manipulation.

Little is known on why certain patients have excess scar tissue formation after TKA surgery and others do not. Whether excessive scar tissue formation results in a stiff knee is ultimately also depending on patients' compliance with physiotherapy [2, 28, 31], mental status (fear of movement) [32] and a large variety of other risk factors for postoperative knee stiffness.

Risk factors for knee stiffness after TKA

Literature describes numerous patient, surgery and rehabilitation-related risk factors for postoperative stiffness of the knee (Table 1). Many preoperative risk factors are debated, of which only preoperative knee ROM is considered to be a major risk factor [3, 7, 33–35]. Several authors found that younger patients were more at risk for stiffness after TKA [3, 6, 22, 34–37]. Why younger patients are more at risk is unknown, but in case of younger patients, work and daily living requirements might lead to a lower threshold for MUA [35].

On peroperative risk factors, there is more consensus, especially on increased risk with oversizing and/or malpositioning of TKA components [2, 19, 22, 28, 31, 38, 39], insufficient osteophyte removal or improper soft tissue balancing [2, 18, 28, 31, 38]. Whether implant design is a risk factor remains unclear [7, 40–46].

Postoperative risk factors mainly focus on poor physiotherapy compliance and increased risk secondary to complications such as persistent pain or wound infections [2, 4, 18, 28, 31]. Where the relationship between wound infection and poor outcome seems obvious, studies comparing range of motion between infected and non-infected TKAs are scarce [47]. Poor kinesiophobia scores are usually associated with less postoperative ROM [32], but Doury-Panchout et al. [48] found no increased risk for poor knee ROM if kinesiophobia was present. Interestingly, several authors report an increased risk with more aggressive deep venous thrombosis (DVT) and pulmonary embolism (PE) prophylaxis or treatment [4, 9, 49], although evidence on this is still quite limited.

MUA: indication, timing and effectiveness

Knee ROM ranging from 0° to 110° is considered adequate and generally a measure of success after TKA [18]. MUA is the preferred treatment if during rehabilitation knee ROM fails to improve, since patients who declined MUA did not improve despite continued physiotherapy [16]. There is, however, little detail on the normal recovery of knee ROM in the first postoperative weeks [50, 51], making it no surprise that the decision when to use MUA varies widely both in clinical practice and in scientific literature.

In two systematic reviews (2010 and 2012), the time range for performing MUA varied from 1–2 weeks to 3 months postoperative, knee flexion used as a cutoff to perform MUA ranged from 65° up to 95°, and mean ROM gain was 30° to 47° (mean increase for flexion was 29.4° and 5.7° for extension) [30, 38].

More recent studies used a similar time range (4–12 weeks) and $\leq 90°$ knee flexion or unsatisfactory knee flexion as a threshold to perform MUA [6, 20]. Issa et al. [13] used a ROM of $< 110°$ at any time point beyond 6 weeks with no recent gains after physical therapy as an indication for MUA, while Djaza et al. [10] and Newman et al. [37] mentioned no standardized indications for manipulation after TKA.

Analgesic and/or anti-inflammatory medications are sometimes administered during or shortly after MUA to suppress the inflammatory response. High-quality studies comparing MUA with and without these medications

are lacking, leaving it unclear whether the additional use of these medications results in a better post-MUA ROM. Sharma et al. [5] retrospectively found that MUA with injection of a local anesthetic and steroid resulted in no loss of ROM after MUA, while in the MUA group without additional medication there was a loss of 12 degrees.

Final ROM after MUA is influenced by timing of the procedure (Table 2). Most studies support early manipulation [4, 13, 17, 22, 27, 37, 52–54], but there is a large variety in what is considered early or late. In contrast, some studies suggest that a similar final ROM is achieved irrespective of the interval between surgery and MUA [3, 14, 19, 55].

Manipulation immediately increases ROM, but this is somewhat lost in the weeks after MUA [3, 14, 52, 53, 55, 56], making early ROM gain after MUA a poor indicator of the final outcome. Long-term outcomes after MUA report a 33° gain in ROM [57].

Whether clinical outcomes after MUA are comparable to outcomes in TKA patients not requiring MUA is debated. Similar outcomes in 1-year knee ROM [21] or 3 months, 1- and 2-year ROM and patient-reported outcome measurements [10] are reported, while other authors concluded that results were inferior after MUA [8, 22, 27].

Limited knee extension is a less common indication for MUA. Moderate improvement of knee extension after MUA is reported [17, 53], which might only be achieved with early MUA (≤3 months) [27]. Dzaja et al. [10] reported an immediate mean improvement of 5.6° without data on long-term outcome, while Witvrouw et al. [15] observed no significant difference in both active and passive extension ROM after MUA.

MUA technique

Manipulation of the knee is described quite uniformly by the majority of studies [30, 38], usually performed under general anesthesia with some authors describing additional muscle relaxation achieved by administering succinylcholine [19, 22, 56]. The ipsilateral hip is flexed to 90°, the tibia is grasped quite proximally (to avoid excess leverage on the joint), and the knee is then flexed with application of gentle constant or gradually increasing pressure until the audible and palpable separation of adhesions no longer occurs, and an improvement in ROM is reached [3, 16–18, 56, 58]. Sometimes the achieved knee flexion position is maintained for 1 min [58]. Knee manipulation in extension is less commonly described, using cautious manipulation in case of flexion contracture of the joint [58]. Manrique et al. [18] also indicate to force the knee gently into full extension in case of a flexion contracture and additionally to manipulate the patella in the medial and inferior direction with the knee in extension, breaking adhesions in the suprapatellar pouch.

Forceful manipulation can provoke an inflammatory response [15]. To minimize this response, Werner et al. [20] described perioperative administration of intravenous and oral glucocorticoid in conjunction with 2 weeks of CPM. Scranton et al. [19] did MUA using intra-articular analgesics (bupivacaine) and anti-inflammatory drugs (methylprednisolone acetate), worked through the joint by repeated flexion and extension. Sharma et al. [5] performed MUA with an additional injection containing morphine, methylprednisolone acetate and bupivacaine after manipulation. Ferrel et al. [53] described MUA as an outpatient procedure using an intra-articular injection of local analgesia (Marcaïne) combined with a corticosteroid (Kenalog).

Single or repeated MUA

There is no consensus on the usefulness of repeated MUA. Choi et al. [58] reported an overall gain in ROM of 17.3° after a second MUA ($n=15$), indicated when patients did not show satisfactory ROM gain 4–6 weeks after the first manipulation. However, a successful final ROM (≥90°) was only achieved in approximately half of patients (54%). Desai et al. ([52], $n=21$) concluded there was no additional advantage from re-manipulations, not even from a third or even fourth MUA. In the study by Pariente et al. ([59], $n=65$), ROM after second MUA improved approximately 30°. Ferrel et al. [53] recommended a repeat MUA ($n=16$) if the patient had persistent loss of greater than 5° extension or less than 90°–100° flexion 2 weeks after the first MUA, gaining 24.6° in ROM. However, three of 16 (18.8%) patients required revision surgery. Issa et al. [60] showed that 59% of the repeat MUA cohort ($n=29$) achieved a mean flexion ROM increase of 29°. Final mean ROM in the repeat MUA group was less compared to the single MUA group (105° vs. 114°), but mean Knee Society Scores (KSS) were equal. Twelve patients (41%) in the repeat MUA group required more invasive procedures.

MUA additional to arthroscopy

Arthroscopic debridement for the stiff knee after TKA is generally indicated for patients with no ROM progression beyond 3 months due to arthrofibrosis, or if the posterior cruciate ligament (PCL) is tight making a release necessary [18]. There are no studies directly comparing effectiveness of arthroscopic debridement versus MUA, and the decision to choose arthroscopic release over MUA is not clear. Scranton et al. [19] choose to apply arthroscopic debridement if a patient presented with a stiff knee > 10 weeks after the index procedure, or in case of a failed manipulation when the knee still was stiff and felt "springy." In case the patient's

Table 2 Effectiveness of MUA after TKA related to procedure timing

References	MUA indication (n = total TKA)	Timing of the procedure		Improvement in ROM or knee flexion (degrees) after MUA	
		Early	Late	Early	Late
Yercan et al. [4]	< 75° flexion 10 days postoperative and/or < 95° flexion within 3 months postoperatively (n = 46)	< 3 weeks	3 weeks to 3 months	52° ROM	44° ROM
Daluga et al. [22]	< 70° flexion at discharge or insufficient progress (early); 65°–75° at discharge and insufficient progress or worsening (intermediate); < 80°–85° at 3 months (late) (n = 94)	0–21 days (early); 22–90 days (intermediate)	> 90 days	ROM not specified, but authors concluded: "No significant difference between early and intermediate group but significant better knee flexion after early vs late MUA (104° vs. 97°, respectively)"	
Ipach et al. [55]	< 90° flexion beyond 2 weeks (n = 52)	≤ 30 days	> 30 days	ROM not specified, but authors concluded: "No statistical significance in absolute flexion after MUA was detected between early and late manipulation (p = 0.3)"	
Newman et al. [37]	Unclear (n = 51)	≤ 6 weeks	> 6 weeks	47.3° flexion	27.8° flexion
Ferrel et al. [53]	> 5° extension and/or < 90°–100° flexion 6 weeks after surgery (n = 177)	≤ 50 days	> 50 days	ROM not specified, but authors concluded: "MUA performed less than 50 days after TKA led to a statistically significant increase in flexion compared to those patients undergoing MUA greater than 50 days after TKA (p = 0.029)"	
Bawa et al. [17]	< 90° flexion < 6 weeks or sooner if patients were not on pace to achieve this benchmark (n = 119)	≤ 75 days: Group 1: 0–45 days Group 2: 46–60 days Group 3: 61–75 days	> 75 days: Group 4: 76–90 days Group 5: > 90 days	Group 1: 48.9° ROM Group 2: 42.2° ROM Group 3: 41.0° ROM	Group 4: 19.3° ROM Group 5: 18.6° ROM
Yeoh et al. [14]	< 80° flexion despite intensive physiotherapy after 3 weeks (n = 48)	< 12 weeks	> 12 weeks	34° flexion	31° flexion
Keating et al. [3]	< 90° flexion 2 months postoperatively (n = 113)	≤ 12 weeks	> 12 weeks	ROM not specified, but authors concluded: "No significant difference in results on the basis of the postoperative timing of manipulation, less than or greater than 12 weeks; $p \leq 0.3597$"	
Issa et al. [13]	Plateau of < 110° ROM > 6 weeks after surgery (n = 144)	≤ 12 weeks (group 1: ≤ 6 weeks; group 2: week 7–12)	> 12 weeks (group 3: week 13–26; group 4: > 26 weeks)	36.5° flexion (group 1: 38°; group 2: 36°)	17° flexion (group 3: 21°; group 4: 12°)
Vanlommel et al. [54]	< 90° flexion and/or > 15° extension < 3 months after surgery (n = 158)	≤ 12 weeks (group 1: 0–6 weeks; group 2: 7–12 weeks)	> 12 weeks	Group 1: 32.8° flexion; group 2: 29.3° flexion	19.3° flexion
Namba et al. [27]	Unclear (n = 195)	≤ 90 days	> 90 days	33.0° flexion, 4.7° extension	17.0° flexion, 1.7° extension
Desai et al. [52]	Unclear (n = 86)	< 20 weeks	≥ 20 weeks	35° ROM	2° ROM

MUA manipulation under anesthesia, *TKA* total knee arthroplasty, *ROM* range of motion

knee had significant limitation of motion or the knee felt "woody," or the arthroscope could not be introduced secondary to severe fibrous tissue, then a modified open release with manipulation was used.

The usual technique with arthroscopic debridement is to remove large fibrous bands of scar tissue in the suprapatellar pouch, medial and lateral gutters and intercondylar notch and complete release of the PCL [19]. Usually, in addition to arthroscopic debridement for stiff knees after TKA, MUA is performed at the end of the procedure. According to the systematic reviews by Fitzsimmons et al. [30] and Ghani et al. [38], MUA and arthroscopy (with or without MUA) result in similar ROM gains.

Rehabilitation after MUA

Scientific literature is without consensus on type, frequency and detail of physiotherapy after MUA [3, 4, 14–18, 53]. To our knowledge, no studies compare different physiotherapy treatment modalities to optimize ROM after MUA. Yercan et al. [4] described that after MUA the knee of the patient was placed 3–4 days over a bolster holding both knee and the hip in 90 degrees of flexion. Keating et al. [3] started with active-assisted flexion and extension by the therapist on the recovery room within 30 min after the manipulation procedure.

Most authors prescribe intensive physical therapy after MUA for several weeks including both passive and active ROM exercises [3, 4, 13–15, 17, 18]. In addition quadriceps muscle-strengthening, gait exercises [3, 13] and CPM are applied, anywhere from the first 24 h [16] up to 2 weeks after manipulation [13, 19, 20]. Witvrouw et al. [15] described the use of CMP while the patient remained in hospital under spinal anesthesia for 48 h and in the study of Djaza et al. [10] MUA was followed by 48–72 h of CPM with epidural analgesia. Continuous epidural analgesia was also used by Pariente et al. [59] who applied CPM for 2–3 days, alternating between CPM for 2 h and placing the leg in a gravity-extension position for 1 h.

Manrique et al. [18] stressed the importance of optimizing pain control, including the use of a spinal catheter, to allow aggressive ROM rehabilitation. For this purpose, cooling is also applied [3, 13, 17].

Future directions

It is important to have concise preoperative risk factors for knee stiffness after TKA, to be better able to manage patient expectations. This is especially true in patients with poor preoperative knee ROM, and in younger patients who are more at risk for developing a stiff knee after TKA. Younger patients also demand to achieve higher activity levels postoperatively. More research is needed on the optimal postoperative management of TKA patients regarding prevention of a stiff knee. There is a need for comparative and preferably randomized trials to elude more clear indications for MUA and to establish correct timing for MUA. More detailed information on the natural course of knee ROM immediately after TKA can be important for early identification of patients with poor ROM recovery in the first postoperative weeks. This enables a more effective treatment strategy, since early MUA is proven to be more successful than MUA in a later stage.

Although MUA technique is generally quite uniformly described, future research might also investigate potential benefits of additional patellofemoral joint manipulation or the administration of anti-inflammatory drugs during MUA procedures. Special attention for the effectiveness of repeated MUA procedures is warranted, and effective post-MUA rehabilitation protocols should be designed based on results from prospective clinical trails.

Conclusion

Persistent limited ROM after TKA results in poor patient-reported outcome and is increasingly becoming a more prominent reason for TKA revision surgery. Its' etiology is largely unknown but can be due to excessive scar tissue, leading to arthrofibrosis. Numerous patient-related, surgical-related and postoperative risk factors for insufficient ROM after TKA are described in literature, but a comprehensive, valid risk model is lacking. MUA is an effective treatment option for knee stiffness after TKA, especially when performed within the first 3 months after TKA, although successful later or repeated MUA procedures are reported. There is a wide variety in indication and timing for MUA and virtually no scientific evidence on how to rehabilitate patients after MUA.

Compliance with ethical standards

Conflict of interest A. Kornuijt is a paid consultant for Cotera Inc. D. Das and W. van der Weegen receive research funding from Cotera Inc. W. van der Weegen is a paid consultant for Zimmer Biomet Inc. T. Sijbesma and L. de Vries have no conflict of interest to disclose.

Ethical approval This article does not contain any studies with human participants performed by any of the authors.

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