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SYSTEMATIC REVIEW

RETURN OF NORMAL GAIT AS AN OUTCOME MEASUREMENT IN ACL RECONSTRUCTED PATIENTS. A SYSTEMATIC REVIEW

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ABSTRACT

Background: Current clinical outcome measurements may overestimate the long term success of anterior cruciate ligament reconstruction (ACLR). There is a need to understand biomechanics of the knee joint during daily activities. This systematic review provides a comprehensive overview of the literature related to gait in patients following ACLR. The purpose of this systematic review was to investigate the available literature and provide a comprehensive overview of kinematic and kinetic variables that present during gait in patients after ACLR.

Methods: A literature search was performed in AMED, CINAHL, EMBASE, Medline and Scopus between January 2000 and October 2012. Inclusion criteria included articles written in English, German or Dutch, and those reporting on gait analysis in patients after ACLR. Kinematic and/or kinetic data of the uninjured and ACLR knee and healthy controls (CTRL) were outcome measurements of interest. Each study's methodological quality was assessed using the Critical Appraisal Skills Programme critical appraisal tool.

Results: Twenty two studies fulfilled the inclusion criteria. A total of 479 patients with a mean age of 27.3 were examined. Time between the injury and surgery and ranged from 3 weeks to 5.7 years. Gait analysis was done at a mean of 29.3 months after surgery. Gait was found to be altered in the sagittal, frontal and transverse planes after ACLR and may take months or years to normalize, if normalization occurs at all.

Conclusion: Patients after ACLR have altered gait patterns that can persist for up to five years after surgery. It is imperative that rehabilitation techniques are examined in order to minimize changes in knee biomechanics during gait, as they have the potential to impact on the development of osteoarthritis.

Level of evidence: 3a

Keywords: Anterior cruciate ligament, biomechanics, gait

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INTRODUCTION

Although an anterior cruciate ligament (ACL) injury is relatively uncommon in relation to exposure, the result is a debilitating injury to those afflicted. Those, who would like to stay active in cutting type sports are often counseled to undergo an ACL reconstruction (ACLR).[1] Despite restoration of nearly normal anterior translation of the knee, patients often face residual impairments including pain, strength, swelling or stiffness for years after surgery.[2] Logically, the focus on outcome measures has been directed towards impairments. Recent research indicates that greater emphasis should be placed on sports participation outcomes rather than on impairment-based outcomes.[3] For example, return to pre-injury level of sports is often not achieved in the first post-operative year, although 90% of the patients have a nearly normal knee according to impairment based outcomes. It appears that the current impairment measures may overestimate the success of ACLR. Hence, additional experiments are needed to provide clinically relevant, functional information in order to bridge the gap between basic science and patient relevant outcomes.[4] Gait is such an activity that has been studied in patients after ACLR showing that gait alterations are frequently encountered in this patient population. Of particular interest is altered loading of the knee in terms of higher adduction moments during gait, which has been suggested as a causative factor that may be related to the early onset of osteoarthritis (OA).[5] Although up to 80% of patients after ACLR may develop OA within 10-15 years after surgery if there was concomitant meniscal, medial collateral ligament and chondral injury,[6] there is no evidence to suggest that high adduction moments of the knee are a sole cause of OA after ACLR. Activities that have a repetitive nature have been suggested to be a causative factor for the development of OA.[7] Recently, a review on gait in patients with ACL deficient (ACLD) and ACLR knees was published but the authors reported only on sagittal plane moments.[8] To best of the authors' knowledge, a comprehensive overview of gait patterns after ACLR is still unavailable. The purpose of this systematic review was to investigate the available literature and provide a comprehensive overview of kinematic and kinetic variables that present during gait in patients after ACLR.

MATERIAL AND METHODS

PRISMA guidelines that use systematic and explicit methods to identify, select, and critically appraise relevant research were followed. A literature search was performed using AMED, CINAHL, EMBASE, Medline, and Scopus. The search terms in Medline were entered in two groups: Group one; 'Anterior Cruciate Ligament' [Mesh], Anterior Cruciate Ligament/surgery*, Knee-Joint' [Mesh]; Group two: 'Gait' [Mesh], 'Walking' [Mesh]. The search terms in each group were combined with the OR operator, the AND operator was used to combine the results from both groups to obtain the final yield. Comparable search terms were used for the other databases. Only studies that examined human adults and were conducted between January 2000 and October 2012 were assessed for inclusion. The titles and abstracts of the studies were screened for potential relevance. The following inclusion criteria were applied 1) full text published in English, German or Dutch; 2) studies reporting on gait analysis in patients after ACLR; 3) subjects between 18-45 years of age at time of recruitment; 4) studies published between 2000 and 2012 and 5) kinematic and/or kinetic data of the uninjured and ACLR knee and healthy controls (CTRL) were reported in numbers. Exclusion criteria were 1) animal studies; 2) case studies; 3) if no side to side comparison was conducted or lacked comparison with a CTRL group; 4) systematic reviews/meta-analysis and 5) only an abstract was available. In addition, reference lists of relevant articles were also screened to identify potential publications not identified in the formal search strategy. The following variables were of interest during the stance phase: initial contact, midstance, peak and minimum joint angles and moments for the sagittal, frontal and transverse planes. The discrete variables were chosen on the basis of their frequent use in the literature and their relation to possible development of OA.[9] Each study's methodological quality was assessed using the Critical Appraisal Skills Programme (CASP, available at http://www.casp-uk.net) critical appraisal tools that have been widely employed in systematic reviews to assess the methodological quality of clinical studies. The appraisal was independently conducted by two authors (AG and AB). Disagreements in appraisal were resolved by discussion.

RESULTS

The search terms for the various databases are shown in Table 1 and the total yield after assessment is depicted in Figure 1. A formal meta-analysis was not feasible due the heterogeneous data reported in the included studies. The results of the critical appraisal are presented in Table 2, demonstrating that all the studies clearly defined their research question, utilized an appropriate study design, and clearly defined their population and methods used. However, a major recurrent limitation was that only two studies based their sample size on a power calculation. Of the 52 potential relevant studies, 30 were excluded. Eleven studies did not provide kinematic

Table 1. *Search terms for databases.*

	MEDLINE	AMED	SCOPUS	CINAHL	EMBASE
Search terms	(Anterior Cruciate Ligament[Mesh] OR Anterior Cruciate Ligament/surgery* OR Knee-Joint[Mesh]) AND (Gait[Mesh] OR Walking[Mesh])	anterior cruciate ligament AND gait (keywords)	anterior cruciate ligament AND gait (keywords)	((MH "Anterior Cruciate Ligament") OR (MH "Anterior Cruciate Ligament/SU") OR (MH "Knee Joint") AND (MH "Gait") AND (MH "Gait Analysis") AND (MH "Walking"))	anterior cruciate ligament'/exp OR 'anterior cruciate ligament reconstruction'/exp OR 'knee'/exp AND 'gait'/exp

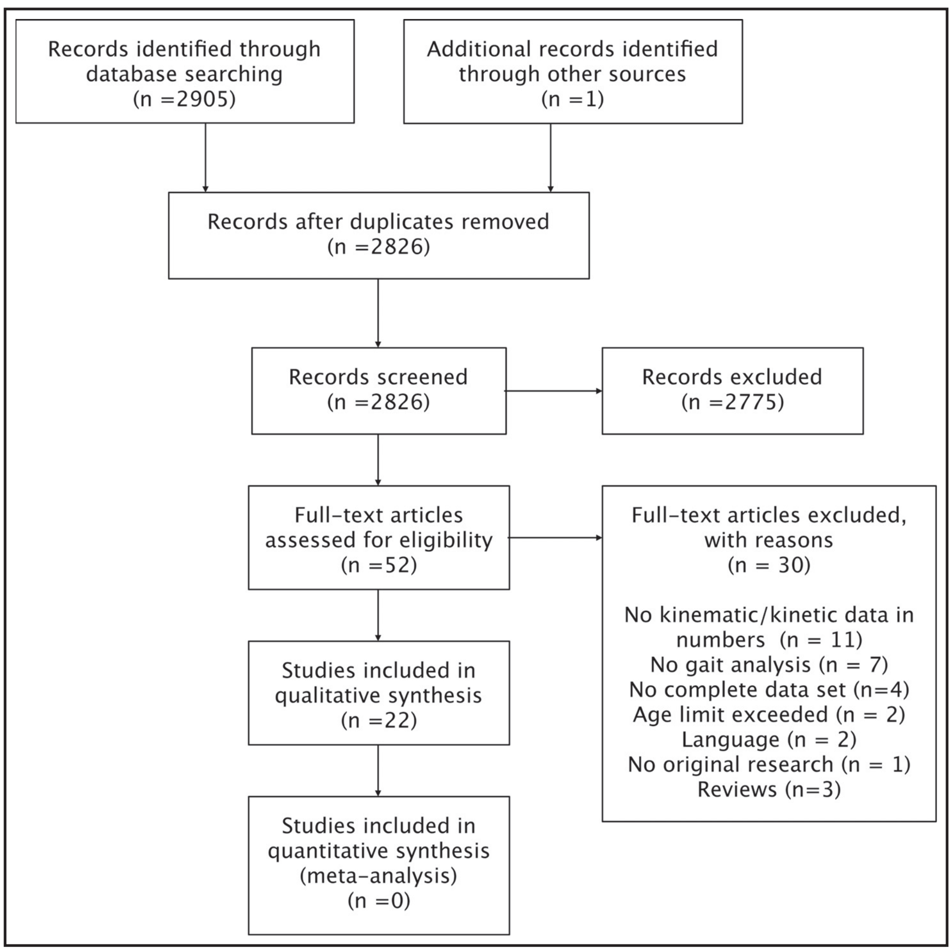

Figure 1. *PRISMA flow diagram of search strategy.*

Table 2. *Critical Appraisal Skills Programme (CASP) methodological appraisal checklist of included studies.*

Description	The population studied?	The outcomes considered?	Are the aims of the investigation clearly stated?	Was the sample representative of its target population?	Sample based on power calculation	Were the assessors blind to the different groups?	Could selective drop out explain the effect?	Are you confident with the authors' choice and use of statistical methods, if employed (results reported with mean or median, SD or CI and p value)	Were all important outcomes/ results considered?	Accept for further use as Type IV evidence?
Bacchini [41]	yes	yes	yes	yes	no	no	n/s	no (only mean and SD, not for individual results)	yes	yes
Beard [42]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD, 95%CI & p-value)	yes	yes
Butler [5]	yes	yes	yes	yes	yes	no	n/s	yes (mean, SD & p-value)	yes	yes
Decker [43]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Ferber [44, 45, 46]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Gao [22]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Georgoulis [47]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Gokeler [48]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Hooper [2]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Hooper [49]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Karanikas [50,51]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Knoll [52,53]	yes	yes	yes	yes	no	no	n/s	no (mean, SD & p-value)	yes	yes
Lewek [54]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Roewer [55]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Shin [56]	yes	yes	yes	yes	yes	no	n/s	yes (mean, SD & p-value)	yes	yes
Webster [9]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Webster [57]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes
Webster [58]	yes	yes	yes	yes	no	no	n/s	yes (mean, SD & p-value)	yes	yes

n/s: not stated

and/or kinetic data in numbers or of interest for this review.[10-20] Five studies reported incomplete data that precluded side to side comparison.[21-25] The corresponding authors were contacted with the request to provide data. One provided data,[22] three did not reply, and one could not be traced. These four studies were therefore excluded.[21,23-25] The results of seven studies did not include gait analysis[26-32] and the age limit was exceeded in two studies.[33,34] Two studies were excluded based on language restrictions.[35,36] One study was excluded based on including subjects with a confounding factor (extension deficit of 7°).[37] Three narrative reviews were excluded.[38-40] Hence, 22 studies[2,5,9,22,25,41-58] fulfilled the inclusion criteria of which several studies reported on the same patient cohort but with different outcomes.[44-46,50-53] The detailed demographic data of the ACLR and CTRL groups are depicted in Table 3. A total of 479 patients with a mean age of 27.3 years were examined across all studies. There were a total of 203 control subjects with a mean age of 26.7 years.

BIOMECHANICS

The results for the biomechanical outcome measurements are shown in Table 4. A large variance between the time of injury and surgery, and test dates after surgery exists. The time between the injury and surgery ranged from 3 weeks to 5.7 years. Gait analysis was performed at a mean of 29.3 months (range 6 weeks – 5.3 years) after surgery across all studies. It should be noted that gait analysis parameters reported such as flexion at initial contact, peak flexion, walking speed varied considerably. In addition several studies reported only on gait for the ACLR leg without comparison to the uninjured leg whilst others lacked a CTRL group.[2,5,9,22,50,51,52,53,54,56,57]

KNEE JOINT KINEMATICS

Flexion and extension

Knee flexion at initial contact for the ACLR leg ranged from -0.1° to 8.1°,[2,42,47,49,54,58] and 3° to 4° for the uninjured leg[2,49] in the ACLR group and 2.3° to 7.8° for the CTRL group[47,54,57]

Peak knee flexion during stance ranged from 15.7° to 55.4° for the ACLR leg and 12.1° to 28.1° for the uninjured leg.[22,50,52,53,55,57,58] Two studies reported on peak knee extension, one study noted a change for peak extension from 17.9° at six weeks to 5.4° at one year,[53] whilst another study found differences related to graft type with 1.5° for patellar tendon and 2.5° hamstring grafts, respectively.[58] Midstance ROM in ACLR ranged between 5.3° and 11.0°.[2,49,58] As a general trend, ROM did increase over time post ACLR.

Adduction and abduction

Gao et al found an increased adduction of the knee joint of 2-3° in the ACLR leg compared to the healthy

Table 3. *Characteristics of the patient groups and control groups.*

Author	Number of included subjects in Patient group (male, female)	Age in years (mean±sd/range)	Time between injury and surgery (mean±sd/range)	Time after surgery (mean±sd/range)	Type of graft	Number included subjects in control group (male, female)	Age in years (mean±sd)
Bacchini [41]	8 (8)	28.3 (21-33)	1-2 (N.R.) months	6.5 months (N.R.)	PT	N.A.	N.A.
Beard [42]	11 (9,2)	29.7±3.6	47±30 months	6.3 months (5-7)	5 PT, 6 HS	N.A.	N.A.
Butler [5]	17 (3,14)	23.6±5.8	N.R.	5.3±4.4 years	N.R.	17 (14,3)	23.4±5.7
Decker [43]	16 (N.R)	27.8±7.4	FDHO 3.9±3.4 months PSF 4.1± 3.5 months	6 and 12 weeks (N.R.)	PT	8 (N.R)	28.3±4.3
Ferber [44,45,46]	10 (5,5)	27.7±9.1	5.7±5.1 years	3 months (N.R.)	PT	10 (5,5)	24.4±3.1
Gao [22]	14 (12,2)	25.1±5.9	N.R.	3-12 months (N.R.)	PT, HS, AT	15(12,3)	22.8 (2.6)
Georgoulis [47]	21 (19,2)	25.0±4.0	N.R.	6.9± 3.9 months	PT	10 (8,2)	24.7±3.7
Gokeler [48]	14 (7,7)	24.0 (21.0-40.0)	N.R.	5.9 (N.R.)	PT	N.R.	N.R.
Hooper [49]	CKC 18 (13,5) OKC 19 (16,3)	N.R.	OKC 4.2± 5.1 years OKC 2.8± 2.5 years	6 weeks	PT (Kennedy augmentation)	N.A.	N.A.
Hooper [2]	ACL 6 months: 8 (5,3) ACL 1 year 9 (4,5)	ACL 6 months : 33.4±11.0 ACL 1 year: 34.2±10.4	N.R.	ACL 6.4±2.0 months ACL 11.9±2.4 months	PT	N.A.	N.A.
Karanikas [50,51]	3-6 months: 11 (N.R) 6-12 months: 11 (N.R) 1-2 years: 13 (N.R)	3-6 months: 27.0±8.0 6-12 months: 33.0±8.0 1-2 years: 29.0±8.0	N.R.	3-6 months, 6-12 months, 1-2 years (N.R.)	HS, PT	N.P.	N.P.
Knoll [52,53]	AC: 9 (9,0) CH: 16 (9,7)	AC: 29.9±6.5 CH male: 39.7±2.1 CH female: 30.3±9.5	N.R.	6, 16, 32, 52 weeks	PT	51 (31,20)	31.7±4.1
Lewek [54]	S.A.: 8 (5,3) W.A.: 10 (4,6)	S.A.: 21.4±6.0 W.A.: 25.0±7.8	N.R.	S.A.: 1.1±0.2 years W.A.: 1.6±0.7 years	HS, PT	10 (8,2)	32.2±6.6
Roewer [55]	26 (18,8)	29.6±10.7	N.R.	6, 24 months	HS, AL	N.A.	N.A.
Shin [56]	24 (9,15)	33.0±9.2	3.6±3.1 years	2.8±2.8 years	N.R.	N.A.	N.A.
Webster [9]	HS: 18 (16,2) PT: 18 (17,1)	HS: 26.6±6.0 PT: 23.8±6.0	HS: 11.0±8.2 weeks PT: 12.7±11.6 weeks	HS: 9.0±2.4 months PT: 10.9±2.0 months	HS, PT	18 (16,2)	24.7±5.0
Webster [57]	Female: 18 (0,18) Male: 18 (18,0)	Female: 27.7±6.8 Male: 27.4±6.6	Female: 13.1± 9.0 weeks Male: 14.8±10.0 weeks	Female: 19.8±11.6 months Male: 20.3±11.4 months	HS	N.A.	N.P.
Webster [58]	HS: 17 (16,1) PT: 17 (16,1)	HS: 26.8±8.0 PT: 23.8±5.0	HS: 10.6±8.3 weeks PT: 12.8±11.7 weeks	HS: 9.3±2.2 months PT : 11.0±1.9 months	HS, PT	17 (16,1)	24.7±5.0

PT: Patellar tendon
HS: Hamstrings graft
ACH: Achilles graft
AL: Allograft
AC: Patients with an acute injury of the ACL at the operation date
CH: Patients with a chronic injury of the ACL at the operation date
S.A.: Strong ACL-R: the injured leg 90% or more quadriceps strength compared to the non-involved quadriceps
W.A.: Weak ACL-R: the injured leg 80% or less quadriceps strength compared to the non-involved quadriceps
N.R.: Not reported
PSF: Preferred strike frequency
FDHO: Force driven harmonic oscillator

leg from the CTRL group.[22] Butler et al found the same trend with a mean peak adduction angle of 1.9° for patients after ACLR versus 0.9° for CTRL.[5] A difference was found comparing hamstring and patellar tendon grafts.[9] The hamstring group had a reduced adduction for the injured leg compared with both the patellar tendon and CTRL, but not when compared with the non-injured knee.

Internal and external rotation

Results regarding internal and external tibial rotation during gait showed mixed outcomes between studies. Gao and Zhang found increased internal rotation in the ACLR leg of patients compared to CTRL subjects.[22] Interestingly, Webster and Feller found that 86% of patients after ACLR walk with more external rotation compared to the CTRL group and 42% of patients to have greater than 5° of external rotation of the ACLR leg compared to their non-injured knee.[9]

KNEE JOINT KINETICS

Knee moments are expressed as external moments for the purpose of this review.

Flexion and extension

Knee extension moments ranged from 0.3 Nm/kg to 0.4 Nm/kg in the ACLR leg[2,54] and 0.7 Nm/kg in for CTRL[54]. Knee flexion moments ranged from 0.1 Nm/kg to 0.3 Nm/kg for both the ACLR and non-injured legs.[2,58] Shin et al. reported knee flexion moments by different unit of measure with respective values of 2.9 Nm/%BW/height for the ACLR leg and 3.7 Nm/%BW/height for the non-injured legs.[56] Webster et al found that graft type had an influence on the knee biomechanics during gait.[58] The external knee flexion moment at midstance was significantly smaller than that in the CTRL legs in 65% of patients in the patellar tendon group and 29% of patients in the hamstring tendon group. In contrast, the external knee extension moment at terminal stance was

Table 4. *Biomechanical outcome measurements.*

	Knee angles			Knee external moments		
	Injured leg ACLR	**Non-injured leg ACLR**	**Control**	**Injured leg ACLR**	**Non-injured leg ACLR**	**Control**
Author	° ± SD	° ± SD	° ± SD	Nm/kg	Nm/kg	Nm/kg
Bacchini [41]			N.R.	Terminal stance extension 32.2* Terminal stance adduction 24.6*	Terminal stance extension 21.0* Terminal stance adduction 18.4*	N.R.
Beard [42]	Flexion at IC Prior surgery 1.8±6.0 Post surgery -0.1±5.8					
Butler [5]	Peak adduction 1.9±2.9		Peak adduction 0.9±2.9	Peak adduction 0.4±0.1		Peak adduction 0.3±0.1
Decker [43]	PSF Midstance ROM 6 w: 5.3±1.9[1,2] 12 w: 6.8±3.1[1,2] FDHO Midstance ROM 6 w: 5.4±3.07[1,2] 12 w: 10.6±2.7[1,2]		Midstance ROM 15.1±3.3[1]			
Ferber [44, 45, 46]	17.6±4.4[1]	18.7±3.9[1]	Left leg: 13.0±0.9[1] Right leg: 15.2±1.9	9.9±3.3[1] (extensor angular impulse)	15.8±4.8[1] (extensor angular impulse)	Left leg: 4.2±1.2[1] Right leg: 3.4±0.5[1] (extensor angular impulse)
Gao [22]	Peak flexion 15.7±3.9 Peak adduction 0.2±0.5 Peak internal rotation 5.8±3.6		Peak flexion 18.6±5.0 Peak adduction 2.7±2.9 Peak internal rotation 2.8±4.7			
Georgoulis [47]	Flexion at IC 1.2±6.3 Peak loading response 13.4±10.0 Peak adduction 1.7±4.10 Peak external rotation 0.3±9.9		Flexion at IC 2.3±3.3 Peak loading response 14.5±5.8 Peak adduction 1.0±3.1 Peak external rotation 3.6±6.2	N.A.		N.A.
Gokeler [48]	Difference injured/non-injured flexion-extension 4.9±3.96		Difference left/right flexion-extension 1.3	Difference injured/non-injured 0.3± 0.2		Difference left/right leg 0.1±0.6
Hooper [2]	Flexion at IC 6 months 3.0±3.0 1 year 3.0±4.0 Midstance ROM 6 months: 9.0±3.0[3] 1 year: 11.0±6.0[3]	Flexion at IC 6 months 7.0±4.0 1 year 6.0±3.0 Midstance ROM 6 months: 12.0±4.0[3] 1 year: 15.0±4.0[3]	N.A	Peak extension 6 months: 0.3±0.1 1 year: 0.3±0.2 Peak flexion 6 months: 0.1±0.1 1 year: 0.2±0.1 Peak adduction 6 months: 0.2±0.0 1 year: 0.2±0.0	Peak extension 6 months : 0.4±0.1 1 year:0.4±0.1 Peak flexion 6 months: 0.1±0.2 1 year: 0.2±0.1 Peak adduction 6 months: 0.2±0.1 1 year: 0.2±0.1	N.A.
Hooper [49]	Flexion at IC CKC 1.0±4.0 OKC 2.0±2.0 Midstance ROM CKC 8.0±4.0 OKC 6.0±4.0	Flexion at IC CKC 4.0±6.0 OKC 4.0±4.0 Midstance ROM CKC 14.0±2.0 OKC 13.0±6.0	N.A.			
Karanikas [50,51]	Mean flexion during stance phase 12-26w: 5.0±4.1[3] 26-52w: 6.2±4.5 52-104w: 6.0±4.3 Mean extension during stance phase 12-26w: 3.7±5.4 26-52w: 7.2±5.7 52-104w: 5.0±3.2	Mean flexion during stance phase 12-26w: 9.3±4.1[3] 26-52w: 9.2±4.6 52-104w: 7.4±5.1 Mean extension during stance phase 12-26w: 8.6±5.1 26-52w: 12.1±7.2 52-104w: 6.5±3.3	N.P			N.P.
Knoll [52,53]	Male peak flexion: 6w: 47.2±1.5[1] 16w: 55.3±1.6 32w: 52.3±1.3 52w: 52.1±1.0 Male peak extension: 6w: 17.8±1.4 16w: 7.3±1.1 32w: 5.5±1.0 52w: 5.4±0.9 Female peak flexion: 6w: 45.1±2.2[1] 16w: 50.1±1.4 32w: 53.7±1.9 52w: 55.4±1.6 Female peak extension: 6w: 17.9±2.6 16w: 9.9±1.3 32w: 7.7±2.0 52w: 7.5±1.0		Male peak flexion: 52.3±1.3[1] Male peak extension: 5.5±1.0 Female peak flexion: 57.3±1.9 Female peak extension: 7.3±1.3	N.R.		N.R.
Lewek [54]	Weak ACLR: Flexion at IC: 5.9±3.1 Peak flexion: 21.0±6.2 Strong ACLR: Flexion at IC: 6.0±4.0 Peak flexion: 24.1±3.9		Flexion at IC: 7.8±5.3 Peak flexion: 26.5±4.9	Weak ACLR: Peak extension: 0.3±0.3 Strong ACLR: Peak extension: 0.4±0.3		Peak extension: 0.7±0.3
Roewer [55]	Peak flexion: 22.3±5.5 6mo: 23.2±6.6 12 mo: 24.4±7.0	Peak flexion: 25.2±5.4 6mo: 28.1±6.7 12 mo: 25.4±6.9				
Shin [56]				(%BW x Ht) Peak flexion: 2.9±0.8 Peak adduction: 2.3±0.8 Peak abduction: 0.6±0.3 Peak internal rotation: 1.0±0.3 Peak external rotation: 0.1±0.1	(%BW x Ht) Peak flexion: 3.7±0.8 Peak adduction: 2.5±0.7 Peak abduction: 0.7±0.5 Peak internal rotation: 1.0±0.3 Peak external rotation: 0.2±0.2	
Webster [9]	IR at midstance: HS: 8.7 PT: 8.9	IR at midstance: HS: 11.5 PT: 14.1				
Webster [57]	Male Peak flexion: 19.1±4.5 Peak adduction: 5.4±9.2 Female Peak flexion: 16.0±5.8 Peak adduction: 4.8±3.1	Male Peak flexion: 19.6±5.4 Peak adduction: 5.0±3.5 Female Peak flexion: 16.2±6.5 Peak adduction: 5.4±4.9		Male Peak flexion: 0.3±0.1 Peak adduction: 0.3±0.1 Female Peak flexion: 0.3±0.1 Peak adduction: 0.4±0.1	Male Peak flexion: 0.3±0.1 Peak adduction: 0.3±0.1 Female Peak flexion: 0.3±0.1 Peak adduction: 0.4±0.1	

Table 4. *Biomechanical outcome measurements. (continued)*

Webster [58]	Flexion at IC: HS: 8.1±5.4 PT: 6.2±3.3 Peak flexion: HS: 21.0±4.9 PT: 17.2±3.6 Peak extension: HS: 2.5±3.4 PT: 1.5±5.0		Flexion at IC: 7.8±4.1 Peak flexion: 21.5±5.6 Peak extension: 0.2±4.5			

PT: Patellar tendon graft
HS: Hamstrings graft
S.A.: Strong ACL-R: the injured leg 90% or more quadriceps strength compared to the non-involved quadriceps
W.A.: Weak ACL-R: the injured leg 80% or less quadriceps strength compared to the non-involved quadriceps
N.R.: Not reported
PSF: Preferred strike frequency
FDHO: Force driven harmonic oscillator
[1]: ensemble average obtained from 5 intervals during stance

significantly smaller than in the CTRL legs in 53% of subjects in the hamstring tendon group and 23% of subjects in the patellar tendon group.

Abduction-Adduction

In general, reported values for knee adduction moments ranged from 0.2 Nm/kg to 0.4 Nm/kg for both the ACLR and non-injured knees.[2,5,57] Different units of measure were reported with adduction moments of 2.3 Nm/%BW/height and 2.5 Nm/%BW/height for the ACLR and non-injured knees.[56] For abduction, they reported 0.6 Nm/%BW/height and 0.7 Nm/%BW/height for the ACLR and non-injured knees respectively.[56] Butler and co-workers noted that patients after ACLR had a 21% larger peak knee-abduction moments than the CTRL group.[5] In a gender comparison study by Webster et al., females were found to have 23% increased peak knee adduction moments in the ACLR knee compared to the ACLR knee in males.[57]

Rotation

One study examined rotational moments revealing 1.0 Nm/%BW/height for the peak internal rotation moment for both the ACLR and non-injured knees.[56] Peak external rotation moment for the ACLR and non-injured knees were respectively 0.1 Nm/%BW/height and 0.2 Nm/%BW/height.

DISCUSSION

The results of this review clearly show that biomechanical deficits evidenced during gait in ACLR patients are very common. Differences in knee angles and total ROM differences were noted, with the ACLR group showing less knee ROM one year after ACLR. Conflicting results were reported for knee rotation. For the knee moments, clear differences between the ACLR and CTRL groups are mostly demonstrated in the extension moments. These altered kinematic and kinetic patterns remain even up to five years after ACLR. It is therefore apparent that the restoration of a normal gait pattern after ACLR takes a long time and may never be complete. It is unclear whether gait patterns ever return to normal as this review indicated that there is a paucity of both longitudinal and five year follow-up data in this patient population.

The most frequently reported upon gait parameter in this review was the kinematics of knee flexion-extension. Results across studies were consistent for this variable and knee flexion or the total amount of knee range of motion is reduced in patients that have undergone ACLR compared to the non-injured knee or a CTRL group. Chaudhari et al postulated that altered biomechanical loading after ACLD may be the precipitating cause of knee OA as loads shift to areas of cartilage not typically loaded.[7] They documented that cartilage appears to be conditioned to load history and that conditioned cartilage likely has different abilities to respond to loads. Hence, altered knee kinematic changes observed in the sagittal plane may also lead to shift of joint load to an infrequently loaded area. Reversing the typical low knee flexion excursion strategy while walking may be advantageous, as joint loads may be distributed over a larger contact area more in line with the conditioned cartilage regions presumably similar to the pre-injury loading pattern.

The more recent studies in the review reported on tibial rotation. The results from these studies were variable, which may in part be attributed to differences in surgical techniques.

The debate on whether single-bundle versus double-bundle ACLR provides better restoration of rotational laxity continues. However, it should be noted that double-bundle reconstruction is not synonymous with "anatomic" ACL reconstruction.[59] Anatomic ACLR is defined by placing the ACL graft in the native ACL insertion site area.[60] This can be done using a single-bundle, or a double-bundle technique. Using in vivo dynamic stereo X-ray, Tashman et al have been able to show that during running, traditional non-anatomic ACLR resulted in altered knee kinematics especially when it comes to rotational stability of the knee.[61]

The knee adduction moment is the gait variable most commonly associated with knee osteoarthritis, as a higher moment is indicative of greater medial compartment loading. This review highlights the limited amount of information regarding this variable in patients after ACLR. Although there is currently no evidence that a higher adduction moment is detrimental in a knee with healthy articular cartilage, the higher adduction moment reported by Butler et al[5] in this review is one mechanism which has been suggested as a potential contributor to the development of OA in the ACLR knee. Future research is required to investigate such claims.

Pre-operative baseline data were lacking in most studies. Knoll et al examined 25 patients with ACLD prior to and six weeks, four months, eight months and one year after ACLR.[53] They found that reestablishment of pre-surgery gait patterns takes at least eight months to occur.[53] Others have found persistent gait deficits in patients up to five years after surgery.[25] Why do these deficits persist for such an extended period of time? The immediate situation after the surgery may require the patient to move the leg carefully in terms of preventing pain and/or fear of tearing the new ACL. These strategies may be regarded as useful in the acute stage after surgery. Nonetheless, it appears that gait alterations persist for years after surgery, a period during which one could assume that fear of pain or re-injury should have subsided. Decker et al recently presented a new model in terms of nonlinear variability.[62] It is based on the premise that a healthy biological system has the ability to adapt to changing environment. Specifically, healthy gait is characterized by optimal movement variability, which allows for flexibility, adaptability, and the ability to respond to unpredictable situations. Hemmerich et al suggested that patients after ACLR may exhibit an "immature stabilization strategy" by utilizing co-contraction in order to increase the joint stiffness and thereby limiting degrees of freedom about the knee joint.[29] It appears that altered sensorimotor control may cause changes in movement patterns. Kaprelli et al recently showed that patients with ACLD had diminished activation in several sensorimotor cortical areas.[63] This implies that changes in motor control may have occurred after injury to the ACL, which likely continues after ACLR, and may be a factor that explains in part, the altered gait patterns after ACLR.

Rehabilitation procedures after ACLR can influence the recovery of function and long-term clinical results. For example, Decker et al noted that altered gait can be improved by specialized gait retraining programs.[43] In a recent randomized controlled trial, patients with ACLD were allocated prior to surgery to a perturbation group performing neuromuscular training and quadriceps strength training, whereas the alternate group performed quadriceps strength training only.[34] Despite symmetrical strength achieved by both groups six months after ACLR, the strength group demonstrated decreased knee excursion between legs during midstance. On the other hand, patients who had received perturbation training had no differences between legs. This suggests that neuromuscular training prior to surgery is more effective in improving gait after surgery.

Lewek et al reported that patients who have 90% or more quadriceps strength in the injured leg compared to the non-injured leg have more normal joint kinematics, kinetics and muscle activation compared to patients with less than 80% quadriceps strength of the injured leg.[54] Webster et al found no relationship between the strength of the hamstrings and quadriceps and joint moments during gait.[58] Also Gokeler et al determined that the strength of the quadriceps had no relation with knee joint angles and moments during gait.[48] Significantly reduced quadriceps strength compared to the non-injured leg in the first two years after surgery has been reported.[51] The results of that study may indicate that improvement of gait and the muscle strength follows different time patterns.

LIMITATIONS OF THE INCLUDED STUDIES

A large variance existed between the time of injury and surgery, as well as time when gait tests were conducted after surgery. This limitation exists because many studies lacked baseline data. Variations in the utilization of 2-D or 3-D methodology were identified with regards to gait analyis parameters. The limitations of marker based motion analysis systems for assessing knee kinematics need to be acknowledged. Of particular relevance is the potential for the movement of markers on the skin and their inability to predict the underlying bone movement. Nonetheless, other techniques which allow for the bone to be directly imaged such as fluoroscopy and stereoradiographic systems cannot be utilized during gait due to limitations of restricted fields of view, quasi-static motion, or one knee being visually blocked by the other. Therefore, skin marker motion analysis is at present the most feasible way to measure knee kinematics during gait.

CONCLUSION

In spite of the different study methods, patients after ACLR have altered gait patterns that can persist for up to five years after surgery. An ACL injury should be regarded as a neurophysiological dysfunction, not simply a musculoskeletal injury. Altered sensorimotor control may be a factor that explains, in part, altered gait after ACL injury and even after subsequent ACLR. Time after injury and reconstruction surgery plays a role in these alterations. These alterations in neuromuscular control might be permanent after ACLR. It is imperative that rehabilitation techniques are examined in order to minimize changes in knee biomechanics during gait, as they have the potential to impact on the development of OA.

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