EMG activity of vastus medialis and vastus lateralis with patellar instability: a systematic review

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The evidence base for changes in electromyographic (EMG) onset and intensity of vastus medialis (VM) and vastus lateralis (VL) in patients with patellar instability is systematically reviewed. The databases AMED, CINAHL, the Cochrane Library, EMBASE, Ovid Medline, Physiotherapy Evidence Database (PEDro), Pubmed and Zetoc were searched from their inception to March 2008, in addition to a manual search of relevant journals. All English-language papers assessing the relative EMG onset and intensity of vastus medialis oblique (VMO) and VL, in patients with patellar dislocation, subluxation and general instability were included. Five papers, consisting of 73 knees with patellar instability, were reviewed. No studies were identified assessing VMO–VL onset in patients with patellar instability. Four studies reported no difference in relative EMG intensity of VMO and VL in patients with patellar instability compared with asymptomatic control subjects. One study reported some evidence of a difference in VM to VL EMG intensity in one cohort of patients with patellar subluxation. The CASP appraisal of the evidence base highlighted a number of methodological weaknesses. There was no robust evidence for any difference in the relative intensity of EMG activity between the VMO and VL in patients with patellar instability. There is no good-quality research evidence to suggest that abnormal vastii EMG intensity or onset are aetiological factors associated with patellar instability.

Keywords: Electromyography, patellar instability, patellofemoral joint, quadriceps, systematic review

Nomenclature

EMG	electromyography
Hz	Hertz
MRI	magnetic resonance imaging
PFPS	patellofemoral pain syndrome
PI	patellar instability
SLR	straight leg raise
SR	sampling rate
VL	vastus lateralis
VM	vastus medialis
VML	vastus medialis longus
VMO	vastus medialis oblique

Introduction

Patellar instability is a generic term which encompasses patients with patellar dislocation, subluxations, or general patellar instability symptoms.[1] It is a complex musculoskeletal disorder with a multifactorial aetiology.[2–6] One such factor may be an imbalance between the vastus medialis oblique (VMO) and vastus lateralis (VL), causing the patella to be transposed laterally.[4,7,8] Patellofemoral pain syndrome has a higher incidence than patellar

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instability.[9] Patients with both patellofemoral pain syndrome and patellar instability may report retropatellar pain. These conditions are differentiated in patellar instability by a patient's description of excessive patellar translation from the trochlear groove during functional activities.[10] Patients with patellar instability may also be differentiated from those with patellofemoral pain syndrome through clinical evidence of patellar apprehension and maltracking, in addition to radiological abnormalities such as abnormal sulcus angle, trochlear dysplasia, patella alta or abnormal patella tilt angle.[1,4,5,6,9,10]

In the area of patellofemoral pain syndrome, electromyography (EMG) has been used to measure both the relative onset[11,12] and the relative intensity[8] of VMO and VL activation. The relative VMO–VL onset is evaluated by determining the onset (also known as activation time) of both muscles during an activity,[11,12] and then subtracting one onset from the other to calculate the onset timing difference (in milliseconds).[11,12] Similarly, the relative intensity (or activation level) of VMO and VL is obtained by extracting and comparing the EMG intensity of both muscles, often presenting these data as a VMO/VL ratio value,[8] where lower ratios indicate relatively lower levels of VMO activity. Previous literature has suggested that delayed onset or reduced intensity of the VMO relative to the VL muscle may be an aetiological factor in the development of patellofemoral pain syndrome.[11–15] It has been speculated that a similar mechanism may cause the development of patellar instability.[4,7] For this reason, it has been suggested that physiotherapists should address this hypothesised abnormality in onset and intensity of VMO contraction relative to VL when designing their treatment programmes.[7]

The purpose of this study is to systemically review the evidence base which has investigated the relative EMG onset or intensity of the VMO and VL in patients with patellar instability.

Methodology

Inclusion–exclusion criteria

We included all full text, English-language papers assessing: (1) the relative onset of EMG activity in VM and VL and/or (2) the relative intensity of EMG activity, in VM and VL, in patients diagnosed with a previous patellar dislocation, or presenting with patellar subluxation or generalised patellar instability. These publications were non-specific for subject age, gender, or year of publication. Papers assessing distal VM were included to prevent the exclusion of results from EMG analysis of this distal component of VM when this was not specifically termed VMO.

We excluded animal and cadaver studies, or those assessing patellofemoral pain syndrome, anterior knee pain or chondromalacia patellae, without specifically acknowledging patellar instability or related instability symptoms. Non-English language papers, or unpublished material including university theses and dissertations were excluded, as were single-subject case reports, comments, letters, editorials, protocols, guidelines, abstracts, conference proceedings or review papers. The reference lists of review papers were scrutinised for any clinical papers deemed relevant to the research question.

Search strategy

The primary search strategy was an electronic search of the databases AMED, CINAHL, the Cochrane Library, EMBASE, Ovid Medline, Physiotherapy Evidence Database (PEDro), Pubmed and Zetoc from their inception to March 2008. These were searched separately using the MeSH and free-word, and Boolean operators: patellar dislocation; patellar subluxation; patellar instability AND electromyography. These broad search terms were adopted to avoid missing any relevant publications. A secondary search of specialist journals was also undertaken manually. These included: *Knee Surgery Sports Traumatology Arthroscopy* (1993–March 2008), *The Knee* (1994–March 2008), *Journal of Electromyography and Kinesiology* (1991–March 2008), *American Journal of Sports Science* (1988–March 2008), and *Journal of Orthopaedic Sports and Physical Therapy* (1991–March 2008).

Following online and manual searching, all highlighted titles and abstracts were screened independently by two investigators (TS, LD). The full manuscripts of each citation satisfying the eligibility criteria were ordered. In cases of uncertainty, full manuscripts of these papers were ordered to scrutinise in greater detail. Reference lists from each manuscript were assessed to identify any potential publications not identified through the previous search strategies. All full manuscripts were screened by the same two reviewers and only included if they fully satisfied the selection criteria. Any disagreement in respect to article selection was resolved though discussion. No paper was excluded on poor methodological quality. The two investigators were not blinded to the source or authors of the identified citations.

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Data extraction

All data from the included papers were entered into a spreadsheet by an individual investigator (TS) and this was then verified by a second investigator (LD). This spreadsheet tabulated: author names, publication date, study design, sample size, population characteristics including diagnostic criteria, subject age and gender, testing details and methods of EMG assessment, statistical analysis, results, and any relevant methodological limitations. Corresponding authors were not contacted to gain additional results for the papers included in this review.

Critical appraisal

All included papers were evaluated against the Critical Appraisal Skills Programme (CASP) appraisal tool for observational studies.[16] This tool assesses: study validity, methodological quality, presentation of results, and external validity. Each paper was independently assessed by two reviewers (TS, LD). In cases of disagreement both reviewers met and discussed the issue before reaching a consensus, in order to maintain reliability.

Results

Search results

Figure 1 shows a QUORUM flow chart, which illustrates the result of our search strategy. The search yielded five studies which met the eligibility criteria. The study details and results are summarised in Table 1 and discussed below.

A total of 73 knees with patellar dislocation, subluxation or general instability in 63 patients (13 males and 50 females) were assessed. For comparison, 36 asymptomatic knees using the contralateral

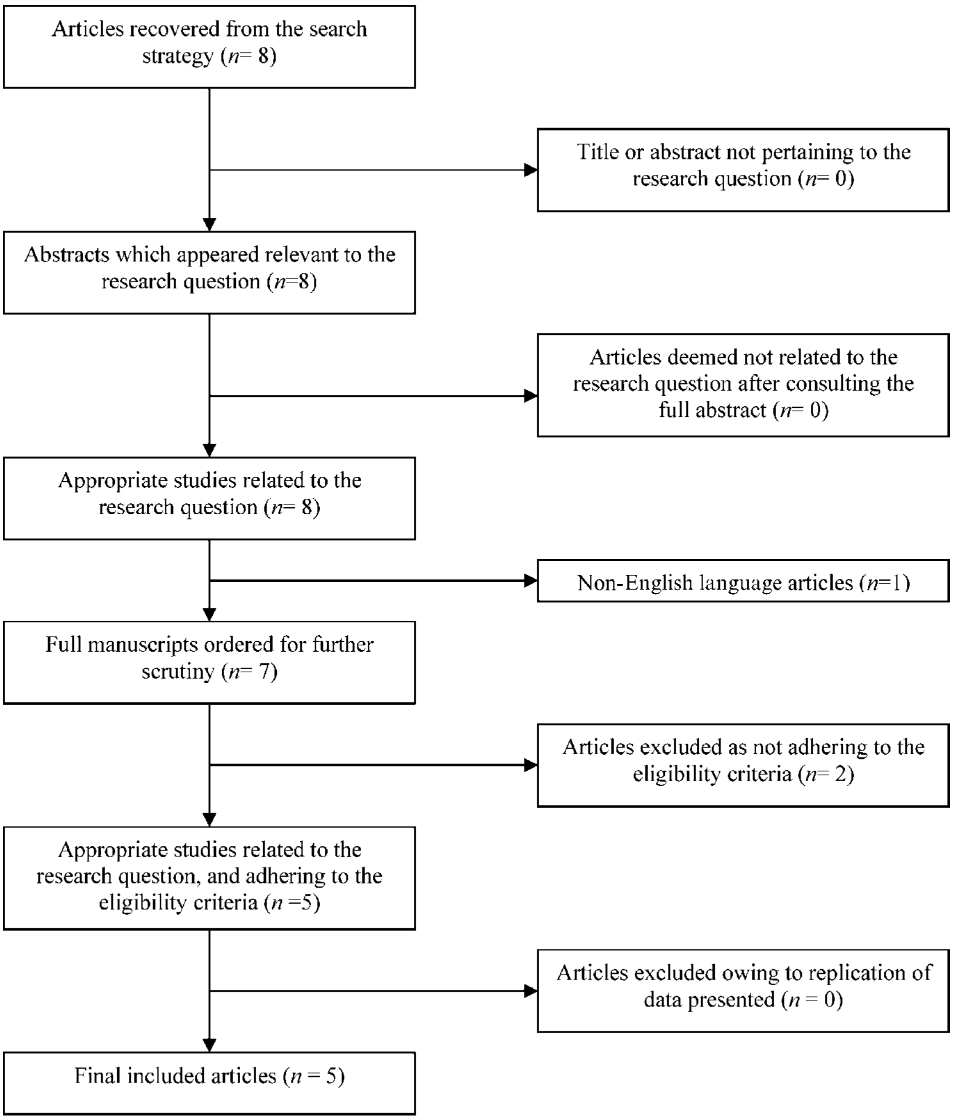

Figure 1 QUORUM Chart

Table 1 Summary of the papers reviewed

Study	Mariani and Caruso[17]
Design	Observational, pre and post surgery
Sample	8 patients with patellar subluxation. No control group, although the asymptomatic contralateral knees were tested
Criteria for patellar instability	Not documented
Population	Patients – age range 20–30; gender – 7 female/1 male; height – not documented
Test activity	Each subject performed three isokinetic knee extensions from 90° to 0° flexion, separately analysed at 90° to 60°, 60° to 30° and 30° to 0° knee flexion
Electrode placement; type; sampling rate; signal processing	Unclear
Method of analysis of EMG	A visual inspection of the raw EMG Activity for each knee flexion phase
Results – activity	Descriptively a decreased level of VM activity was observed compared with VL, which recovered post surgery
Study	Møller *et al.*[18]
Design	Observational
Sample	11 patients with patellar instability (11 knees). No control group, although the 28 asymptomatic contralateral knees were tested
Criteria for patellar instability	Not documented
Population	For entire sample – mean age not documented, range 18–35; gender – 21 female/7 male; height – not documented
Test activity	Quadriceps maximum voluntary isometric knee extension at 90°, 60°, 45°, 30°, 15°, 0° knee flexion (all 90° hip flexion). Each subject performed two repetitions of 5 s contractions. Comparison of affected and unaffected limb using this same methodology
Electrode placement; type; sampling rate; signal processing	Surface EMG electrode placed over VL and the most prominent part of VMO; SR not documented
Method of analysis of EMG	Raw signal used and EMG counts (the number of times the signal crosses zero per second) evaluated. Mean of repetitions used
PI results – activity	No evidence for any VMO–VL imbalance was found
Study	Mohr *et al.*[19]
Design	Observational
Sample	13 PFPS with non-traumatic lateral patellar subluxation. 11 asymptomatic control subjects, with no history of knee pain
Criteria for patellar instability	Abnormal patellar tilt and lateral patellar subluxation on Merchant view radiograph, confirmed by dynamic MRI
Population	Patients – mean age 40 years, range 17–60; gender – 11 female/2 male; height – not documented. Asymptomatic subjects – mean age 28 years, range 21–32; gender – 3 female 8 male; height – not documented
Test activity	Each subject performed three isokinetic functional activities (1) free walking in a straight line at 1·5 m s^{-1} (2) stair ascent on a three-step platform (3) stair descent on a three-step platform with 8 inch steps.
Electrode placement; type; sampling rate; signal processing	In-dwelling electrode placed as described by Basmajian and DeLuca[35]; SR 2500Hz; band pass 100–1000 Hz; data integrated and normalised to peak 0·5 s average during maximum voluntary isometric contraction
Method of analysis of EMG	Median normalised EMG activity was extracted for each of the following seven phases in activity (1) loading response; midstance; terminal stance; pre-swing; initial swing; mid swing; terminal swing. In Activity (2) and (3) this was done for five phases: loading response; midstance; terminal stance; pre-swing; swing
Results – activity	No evidence for any VMO–VL imbalance was found
Study	Wild *et al.*[20]
Design	Observational
Sample	18 patients with 'patellar disorders' (26 knees). 9 knees had recurrent subluxation and 17 knees previously dislocated. No control group
Criteria for patellar instability	Not documented
Population	Mean age not documented, range 11–42; gender – 14 female/4 male; height – not documented
Test activity	Each subject performed isometric knee extension contractions at 0° knee flexion and SLR with 0° knee flexion at 8 to 12 inch, with and without 5 lb ankle weights in hip neutral; internal; or external rotation
Electrode placement; type; sampling rate; signal processing	Surface electrode placed over the central mass of the VL and VMO; SR not stated; no signal processing; integration carried out manually by planimetry
Method of analysis of EMG	Not documented
Results – activity	No evidence for any VMO–VL imbalance was found
Study	Møller *et al.*[21]
Design	Observational
Sample	13 patients with patellar subluxation (15 knees). No EMG control group
Criteria for patellar instability	Not documented
Population	PI group – mean age not documented; gender – 9 female/4 male; height – not documented PFPS group – mean age not documented; gender – 9 female/9 male; height – not documented.
Test activity	Quadriceps maximum voluntary isometric knee extension at 90°, 60°, 45°, 30°, 15°, 0° knee flexion (all 90° hip flexion). Each subject performed two repetitions of a 5 s contraction. Comparison of affected and unaffected limb using this same methodology.

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Table 1 Continued.

Electrode placement; type; sampling rate; signal processing	Surface EMG electrode placed over VL and the most prominent part of VMO; SR not documented
Method of analysis of EMG	Raw signal used and EMG counts (the number of times the signal crosses zero per second) evaluated. Mean of repetitions used
PI results – activity	No evidence for any VMO–VL imbalance was found

knee,[17,18] and 11 separate asymptomatic subjects[19] were reviewed. The age of the cohorts with patellar instability, as described by Mohr *et al.*,[19] Wild *et al.*[20] and Mariani and Caruso,[17] was detailed as a mean of 40 years (17–60), or ranging from 11 to 42 years, or 20 to 30 years, respectively. One study reported the EMG intensity of the distal VM,[17] the remaining stated that the EMG results from the VMO were analysed. As Table 1 suggests, the five studies reviewed investigated EMG using a number of different tasks. Mariani and Caruso[17] analysed isokinetic knee extension in various ranges of knee motion in sitting. Møller *et al.*[18] and Møller *et al.*[21] also evaluated knee extension in sitting, but analysed a maximum voluntary isometric contraction. Mohr *et al.*[19] assessed isokinetic knee function during free walking, and step ascent and descent. Wild *et al.*[20] evaluated a maximum isometric straight leg raise exercise.

EMG onset of the VMO and VL

No identified papers assessed the relative EMG onsets of VM and VL in patients with patellar instability.

EMG intensity of the VMO and VL

The five papers identified by the search strategy assessed the distal VM or VMO and VL EMG intensity in patients with patellar instability. Only Mohr *et al.*[19] compared the patellar instability sample to a control group. Mariani and Caruso[17] and Møller *et al.*[18] compared data from symptomatic knees with the same subjects' asymptomatic knees. The remaining papers did not compare the variables of the symptomatic knees to those of a control group or to asymptomatic knees.[20,21]

Mohr *et al.*[19] reported that there was no difference in the relative intensity of VMO and VL during free-walk gait analysis between patients with patellar instability and asymptomatic subjects, i.e. no evidence for any preferential activation of VMO or VL. Wild *et al.*[20] and Møller *et al.*[18,21] also found no evidence for any VMO–VL imbalance. The exception to these findings was observed by Mariani and Caruso[17] in seven patients with patellar subluxation. These subjects exhibited a marked fall in VM EMG intensity compared with VL throughout knee range of motion, but particularly in the terminal 30° of extension. In the remaining case, there was no difference in EMG intensity between VMO and VL.[17]

There was some evidence to suggest that the intensity of both VMO and VL differ between patellar instability and asymptomatic knees, i.e. a difference between the groups in overall quadriceps activity rather than a VMO–VL imbalance. Mohr *et al.*[19] reported that during walking, in mid-stance, a significantly increased VMO and VL normalised intensity was recorded in the patellar instability group compared with the control group. In terminal swing, there was significantly decreased VMO and VL intensity in the patellar instability group compared with healthy subjects. Similarly, Mohr *et al.*[19] reported that during step ascent, patients with patellar instability displayed a significantly decreased VL and VMO intensity at loading response, i.e. open kinetic chain (OKC), but a significantly increased VL and VMO intensity during terminal stance, pre-swing and swing, i.e. closed kinetic chain (CKC), when compared with the asymptomatic group. On step descent, Mohr *et al.*[19] observed that the patellar instability group displayed significantly increased VMO and VL intensity throughout the task, in comparison to the asymptomatic group (i.e. OKC and CKC). In addition, Møller *et al.*[18] reported a trend for lower intensity levels in both VMO and VL between subjects' symptomatic patellar instability and asymptomatic control knees during an OKC activity.

Critical appraisal results

The results of the CASP appraisal are presented in Table 2. This suggests that the current evidence base presents with a number of methodological limitations. These include the limited description of subject characteristics, such as how subjects were recruited, symptom duration, the defined diagnostic parameters for patellar instability, physical capabilities or sporting participation, and whether there was any variation within the cohort for patient participation in physiotherapy on entering the trial. The studies reviewed used an appropriate observational design using EMG, a method which is highly appropriate to assess muscle activity in this cohort.[22] However, the sample sizes of patellar instability cohorts recruited

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Table 2 A summary of the CASP results

CASP factors	Mariani and Caruso[17]	Mohr *et al.*[19]	Møller *et al.*[18]	Møller *et al.*[21]	Wild *et al.*[20]
Clearly focused question	No	Yes	Yes	No	No
Appropriate design	Yes	Yes	Yes	Yes	Yes
Appropriate recruitment	No	No	No	No	No
Test procedure clearly defined	No	Yes	No	No	No
Appropriate outcomes used	Yes	Yes	Yes	Yes	No
Confounding factors identified	No	No	No	No	No
Confounding factors accounted	No	No	No	No	No
Greater than 85% of the sample on final follow-up	Yes	Yes	Yes	Yes	Yes
Suitable duration of follow-up	Yes	Yes	Yes	Yes	Yes
Precise statistical results presented	No	No	No	No	No
Appropriate interpretation	Yes	Yes	Yes	Yes	Yes
Possible bias acknowledged	No	No	No	No	No
Ability to generalise results	No	No	No	No	No
Interpretation related to the existing evidence	Yes	Yes	Yes	Yes	Yes

were small, ranging from 8 to 26.[17,20] No study justified their sample size using a power calculation. Accordingly, studies may have been underpowered, committing a Type II error.[23] The studies poorly described how they undertook their EMG analysis, electrode placement, sampling rate and signal processing, and data manipulation such as normalisation. The papers reviewed accurately reflected their findings and related these appropriately to the existing evidence base. All studies poorly analysed their findings using statistical tests. Neither Wild *et al.*[20] nor Mariani and Caruso[17] analysed their results using inferential statistics. No paper evaluated the precision of their findings using confidence intervals.

Discussion

This review found no robust evidence for a significant difference in the relative EMG onset or intensity of VMO and VL between patients with patellar instability and asymptomatic subjects. In one study, decreased VM intensity relative to VL was reported in patients with patellar subluxation.[17] This was, however, simply a qualitative observation of raw EMG traces. No studies were identified which assessed the relative onsets of VMO and VL in patients with patellar instability. There is, therefore, little support for a medial–lateral imbalance in this pathology. There is some evidence for a difference in overall intensity of both VMO and VL between patellar instability cohorts and asymptomatic knees.

The findings of this review are based on a limited evidence base which presented with a plethora of methodological weaknesses. These included poorly detailing population characteristics or methods used to recruit cohorts. This factor means that the replication of these studies remains difficult, limiting the reader's ability to generalise these findings to a defined sample in clinical practice. The papers reviewed recruited small, potentially underpowered cohorts, and did not present power calculations. This has considerable importance as an underpowered sample may be insufficient to detect a difference, where one existed.[24] All studies failed to present confidence intervals, which ensures that an appropriate appreciation of their individual and cumulative findings is limited when assessing the statistical significance of the data.[24,25] The papers did not acknowledge whether subjects were undergoing physiotherapy treatment. This is a potential confounding variable as physiotherapy may alter VMO or VL onset and intensity in patients with patellofemoral disorders.[8,12,26] These factors raise questions about the relevance of these findings.[22,25]

The limited description of EMG testing methodology employed was a recurrent and considerable weakness. Papers failed to document precise electrode placement, sampling rate and signal processing methods, all factors which may compromise the reliability, validity and generalisability of EMG results.[27–34] The two studies by Møller *et al.*[18,21] used the method of EMG counts or zero crossings (the number of times the raw bipolar signal crosses the zero value) rather than assessing EMG intensity. The relationship of EMG counts with muscle intensity has been questioned and this variable may provide little relevant information.[35]

It remains unclear whether the onset of VMO is delayed relative to VL in patients with patellar instability, as no identified studies investigated this issue. Therefore, there is currently no research evidence supporting attempts to retrain the timing of VMO relative to VL in patients with patellar instability using modalities such as biofeedback and taping, as suggested by McConnell.[7] Further high-quality investigations are therefore recommended.

Only Mariani and Caruso[17] identified evidence for a difference in relative EMG intensity between VM and VL EMG in a patellar instability cohort. This

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sole finding of reduced VM intensity could be due to the EMG methodology used. The EMG evaluation was a simple visual inspection and classification of the raw waveform, rather than an objective quantification, and it is therefore difficult to assess the validity of the results. The exact EMG methodology is also unclear in this study as electrode placements were not defined. Since we do not know which portion of VM was assessed, it remains unclear whether these findings are attributed to the vastus medialis longus (VML) or VMO. In addition, it is not possible to assess whether this finding differs from the other studies owing to variations in sampling, as the population was poorly described in Mariani and Caruso's[17] paper. These factors may account for the difference in results from the other four papers.

Although this review suggested that there is limited evidence for an imbalance between VMO–VL activity in patellar instability patients, authors have previously hypothesised that a difference in VMO–VL activity is evident between normal knees and those with patellar malalignment.[36,37] This is based on the principle that lateralisation of the patella from either reduced dynamic medial stability, or a greater pull from lateral retinaculum or a lateralised tibial tubercle, could influence length–tension relationships of the quadriceps mechanism and, therefore, cause abnormal EMG activity when compared with healthy asymptomatic knees.[38–41] Grelsamer[4] suggested that in patients with patellar malalignment problems, the distal portion of the VMO may be proximal to the proximal pole of the patella. Therefore, in these patients, the VMO may not directly attach to the patella.[4] Consequently, the muscle force vector would exhibit a more vertical line of pull than normal making the VMO less effective as a dynamic stabiliser.[42,43] Finally, Donell[44] suggested that patellar instability subjects exhibit VMO hypoplasia. Accordingly, if there is reduced muscle mass in this cohort, there should be substantially reduced EMG intensity. This review suggests that this notion has not been previously tested. In response, future studies should first confirm the presence of the VMO through ultrasound or MRI (magnetic resonance imaging), and then assess how this relates to EMG intensity.

Although 73 knees with patellar instability were reviewed in the five studies, the diagnostic procedures for the disorder were not defined. Since we are unable to comment on how these subjects have been defined as patients with patellar instability, the generalisation of these results into clinical practice remains unclear. This is a considerable weakness, particularly given the variety of difference signs and symptoms this patient group exhibits.[45] Similarly, it remains unknown whether the severity of instability in respect to patients' symptom reporting, or the degree of lateralisation on radiograph, correlates to reductions in EMG intensity. Further study, using MRI, ultrasound or computed tomography (CT) imagery, is required to quantify the influence of patellar position and degree of lateralisation on EMG. Further study is also recommended to specifically analyse a sufficiently powerful sample to determine whether the severity of patellar instability, from frank dislocation to mild subluxation, influences EMG intensity. This may then indicate whether altered quadriceps EMG findings should be used as a prognostic indicator for patellar instability.

Patellar instability may originate from a number of different anatomical abnormalities.[5,44,46] Patients with patellar instability may exhibit a variety of different pathophysiological mechanisms. These include a loss of osseous stability from trochlear dysplasia, reduced medial stability from a medial patellofemoral ligament rupture, or patella alta or infera. Such differences may affect the EMG activity of VMO and VL, possibly by altering the length–tension relationship of these muscles. An evaluation of the effect these different anatomical factors may have on EMG activity is therefore indicated.

Clinically, physiotherapists have been encouraged to prescribe exercises to preferentially strengthen the VMO relative to the VL with the aim of improving the dynamic stability of the patella in patients with patellar instability.[7,47] With the exception of seven subjects in Mariani and Caruso's[17] study, it appears that there is no difference between VM and VL EMG intensity in patellar instability cohorts. This raises the question that, if physiotherapists are able to preferentially recruit and strengthen the VMO, should they direct their treatment to this, or should they address other aetiological factors such as the lateral retinacular tightness or, if suggested, onset of the VMO and VL. An improvement in the size and quality of the evidence base in this field is required before such hypotheses can be considered in clinical practice.

Whilst there is limited evidence for a medial–lateral imbalance in this patient group, it appears that EMG intensity in both VMO and VL was reduced in patients with patellar instability compared with asymptomatic knees during OKC activities, and was increased during CKC activities, with the exception of a step descent task in the study by Mohr *et al.*[19] This trend for increased VMO and VL intensity during CKC compared with OKC has been exhibited in previous studies evaluating patellofemoral pain syndrome and asymptomatic cohorts.[48–51] Further study to evaluate whether specific OKC or CKC exercises can inhibit or enhance VMO or VL intensity specifically is recommended to determine whether these findings can be transferred to treatment regimens used to address potential vastii muscle imbalance which may contribute to patellar instability.[7]

Mariani and Caruso[17] assessed subjects pre- and post-medialisation of the tibial tubercle for surgical correction of patellar lateral subluxation. Once malalignment had been corrected, VM intensity increased and, in all but one case, equal VM and VL EMG intensity was restored.[17] Mariani and Caruso[17] did not, however, document the duration between operations to the restoration of this imbalance. This finding does question whether the correction of this biomechanical abnormality facilitated the restoration of vastii muscle balance. This may be through the correction of a mechanical advantage of the VM by transposing the patellar tendon attachment to a more medial position.[52,53] Similarly, EMG intensity may have increased through the decrease in pain by off-loading the lateral femoral condyle from increased retro-patellar pressures.[54–56] Conversely, a combination of each factor may have accounted for this improvement. This suggests that taping techniques may be indicated to temporarily alter VM's line of force to enhance EMG intensity if a difference between VMO and VL exists.[57,58] Further study is recommended to assess whether treatment strategies can affect EMG intensity in patellar instability cohorts.

Given that such a small number of papers was identified for inclusion in this review, it is particularly important to reflect whether our methodology subjected the review to any weakness that could influence our findings. Firstly, all unpublished materials were excluded including university theses, protocols, guidelines, or grey literature. This was to enable the reviewers to base the findings of the review on peer-reviewed material in an attempt to maintain quality, at the expense of the potential for publication bias.[59] The principle search strategy used was a computer search. This may be regarded as a limitation because Colville-Stewart[60] acknowledged that computer searches may omit some articles. Accordingly, we scrutinised the reference lists and conducted a manual search of pertinent journals. Finally, non-English language articles were excluded, owing to the costs of translation. However, this factor was minimal as Figure 1 details that this criterion only excluded one paper.[61]

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Conclusions

This review found no robust evidence for a difference in the relative EMG onset or intensity of VMO and VL in patients with patellar instability compared with asymptomatic control subjects. There was limited evidence of a difference in VM to VL intensity in one cohort of patients with patellar subluxation. No identified studies assessed relative VMO–VL onset in patients with patellar instability. These conclusions are based on a limited evidence base, both in size and methodological quality. Given the paucity of literature, it is not possible to state whether patellar instability is the result of abnormal vastii activity. It therefore remains unclear whether re-education of the quadriceps muscle on the premise of altered VMO–VL recruitment for patients with this disorder is indicated from these EMG studies. As well as improving the methodological quality of the present evidence base, further study should evaluate whether alterations in EMG onset or intensity of VMO relative to VL may be a precursor for patellar instability.

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