

**VASTUS MEDIALIS OBLIQUE – VASTUS LATERALIS MUSCLE
IMBALANCE IN PATELLOFEMORAL PAIN SYNDROME (PFPS)
PATIENTS**

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Sciences**

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Abstract

BACKGROUND and AIMS. Patellofemoral pain syndrome (PFPS) is complex and challenging musculoskeletal disorder. Maltracking of the patella is considered to be one of the primary causative factors. Vastus Medialis Oblique (VMO) and Vastus Lateralis (VL) muscle imbalance in terms of EMG magnitude and timed onset is implicated in either initiating or perpetuating the patellofemoral pain (Cowan et al, 2002, Witvrouw et al, 1996). Many physiotherapeutic treatments are aimed at addressing this muscle imbalance despite a lack of evidence confirming or refuting it exists and it's association with pain and function. The ultimate aim of the study was therefore to establish if it is appropriate to continue treating muscle imbalance in patients with clinically defined PFPS.

OBJECTIVES. The overall objectives of the study were to establish:

1. If VMO – VL muscle imbalance exists in PFPS patients and if so is it specific to this condition or does a similar VMO – VL muscle imbalance exists in a healthy population?
2. If muscle imbalance does exist is it related to clinical symptoms used as indications of pain syndrome in clinical practice?
3. Is muscle imbalance associated with lower limb muscle physiology i.e. lower limb and quadriceps muscle strength in both fresh and fatigued states.

METHODS. The study employed a cross-sectional design. 63 patients with patellofemoral pain syndrome (PFPS) and 63 age/sex matched healthy subjects were recruited and VMO & VL normalised EMG RMS amplitude and time onset differences were assessed during functional and experimental tasks. Additionally, correlations with pain level, functional status, muscular flexibility and biomechanical characteristics of the lower limb were explored.

RESULTS. The results revealed that the VMO-VL activation patterns are task specific and most significantly related to functional stepping down task at a fast speed of execution ($p=0.000$). This interesting link between the type of muscle contraction, the speed of execution and the recruitment pattern of the VMO-VL was also confirmed by the non-functional isokinetic eccentric contraction ($p=0.000$). Additionally, it is the timing of the VMO-VL activation rather the intensity that is important. Also, a correlation appears to exist between activation pattern and duration of symptoms and knee functional performance ($p=0.03$) but not with the level of pain.

CONCLUSION. The findings of the study suggest that the VMO-VL muscle imbalance does exist in a clinically defined PFPS population. Unlike previous studies however, this thesis suggests that specificity of the functional activities and speed of execution have a significant role to play in the muscular performance and it could be argued that this translates to a role in PFPS. It would therefore seem appropriate to continue addressing and treating this complex and challenging issue with physiotherapeutic interventions but this may need to be targeted to interventions that are tailored to addressing issues in relation to stepping down and at fast speed.

Declaration

No portion of the work referred to in this thesis has been submitted in support of an application for another degree or qualification of this or any other university or other institute of learning

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List of Abbreviations

AKP = Anterior Knee Pain

AKPS = Anterior Knee Pain Scale

BF = Biceps Femoris

CA = Congruence Angle

CKC = Close Kinetic Chain

CMP = Chondromalacia Patellae

CSA = Cross Section Area

CT = Computer Tomography

EMG = Electromyography

GM = Gluteus Medius

IEMG = Integrated EMG

ITB = Iliotibial Band

LEEMG = Linear Envelope EMG

MF = Median Frequency

MPF = Median Power Frequency

MRI = Magnetic Resonance Imaging

MVC = Maximal Voluntary Contraction

MVCon = Maximal Voluntary Concentric

MVEcc = Maximal Voluntary Eccentric

MVIC = Maximal Voluntary Isometric Contraction

NWB = Non-weight Bearing

OKC = Open kinetic Chain

PF = Patellofemoral

PFJ = Patellofemoral Joint

PFJRF = Patellofemoral Joint Reaction Force

PPF = Patellofemoral Pain

PFPS = Patellofemoral Pain Syndrome

PL = Peroneus Longus

PTA = Patellar Tilt Angle

RCT = Randomised Control Trial

RF = Rectus Femoris

RMS = Root Mean Square

ROM = Range Of Motion

ST = Semitendinosus

VAS = Visual Analog Scale

VI = Vastus Intermedius

VL = Vastus Lateralis

VLL = Vastus Lateralis Longus

VLO = Vastus Lateralis Oblique

VM = Vastus Medialis

VML = Vastus Medialis Longus

VMO = Vastus Medialis Obliquus

WB = Weight Bearing

ZCR = Zero Crossing Rate

DEDICATION

This thesis is dedicated with love and thanks to
my late Father,
my late Mother, who passed away recently,
my family, my beloved wife Maria and our children Harris and Nikolas

Chapter 1

Introduction and Overall Aims and Objectives

1. INTRODUCTION

The World Health Organisation (WHO) has declared the decade 2000-2010 as the “bone and joint decade” in order to emphasise to the great incidence of musculoskeletal disorders and the same time to reduce economic and social cost (Sanchis-Alfonso, 2006, p. ix). Patellofemoral pain syndrome (PFPS) remains one of the most common, challenging and yet controversial pathologies encountered by musculoskeletal clinicians and investigators (Dye et al, 1999; Wilk, 1998). The complexity of this pain syndrome is reflected by the different nomenclature employed for PFPS patients' diagnosis, the variety of suggested etio-pathogenetical factors and the numerous different therapeutic interventions (Thomeé et al, 2002; Thomeé et al, 1995). Additionally, the historical lack of understanding surrounding the patellofemoral pain problem is reflected in the plethora of different surgical techniques devised for this pathology. Patellofemoral pain syndrome has also been described as “the black hole” and “enigma” of orthopaedics, a phrase implying that no single theory has yet fully clarified the patellofemoral problem or established therapy leading to predictable resolution of the symptoms (Dye, 1999, Sanchis-Alfonso, 2010, p.1). Furthermore, several field experts have stated that, perhaps with the exception of surgical therapy for low back pain, the operative treatment of patellofemoral disorders has the second higher iatrogenic failure rate (Biedert, 2004, p. xxi; Dye, 2004, p. 4; Eriksson, 2006, p. vii; Zaffagnini, Dejour & Arendt, 2010, p. vii).

The true cause of patellofemoral pain syndrome (PFPS) is unknown, it is in reality a diagnosis of exclusion and probably the high incidence rates reported in the literature may derive from inherent difficulties in excluding other diagnoses (Näslund

et al, 2006). Unlike patients with a structural injury or failure of tissues that can be detected objectively, patients with PFPS have no identifiable structural abnormality (Dye et al, 1999). An additional fundamental factor differentiating this clinical entity from other musculoskeletal conditions of the knee joint is its diagnosis mainly through subjective reports and a possible final common pathway that is related to central nervous system events (Dye et al, 1999).

Despite the high reported incidence of the PFPS in the literature, its etiopathogenesis has remained unclear and enigmatic. There is no single factor causing patellofemoral pain symptoms and various authors have cited both extrinsic and intrinsic parameters as source of the etiopathogenesis. Extrinsic factors are considered, excessive loading, exercise or training errors, poor or inadequate equipment and ignorance of the condition. Intrinsic factors comprise lower extremity mal-alignment, leg length discrepancy, muscular imbalance and joint laxity (Thomeé et al, 1999; Fulkerson & Arendt, 2000; Tumia & Maffulli, 2002). Indeed, the interaction of both extrinsic and intrinsic factors may be associated with the development and/or perpetuation of PFPS (Witvrouw, Van Tiggelen & Willems, 2006, pp. 135-145).

The discouraging term 'failed patella' is used to describe the high rate of iatrogenic failure of the operative treatment of the patellofemoral pain, but also highlights the complexity of the PFPS. It is common ground that in many patients with PFPS the mechanical, biological and emotional aspect of pain can coexist and in order to truly understand what is happening it is imperative to sort out the relative contribution of each factor (Grelsamer and McConnell, 1998, pp. 257-263).

Despite a mixed aetiology, maltracking of the patella seems to receive the most attention and is thought to be of primary importance in PFPS. Furthermore, the contribution of the quadriceps dysfunction to the maltracking phenomenon is an important consideration.

1.1. Patellar tracking and patellofemoral pain

The way in which the patella articulates with the femoral trochlear groove is known as patellar tracking and is considered an important mechanical component of the extensor mechanism of the knee. The normal tracking of the patella depends on bony architecture and the function of the periarticular soft tissues of the knee. Patellar tracking affects the magnitude of forces acting on the patellofemoral joint, and both these factors, patellar tracking and magnitude of forces influence the patellofemoral pressure (Grabiner et al, 1994; Grelsamer & Weinstein, 2001; Lin et al, 2010). Normal patellar tracking is a complex function based on the balanced interaction of passive, active and neural factors. When the intricate balance of all the periarticular soft tissues of the knee fail, the patellofemoral pressure distribution is altered and this may lead to patellofemoral pain (Grelsamer & Weinstein, 2001; Lin et al, 2010; McConnell & Bennell, 2006, pp. 167-184).

In addition to the possible association of excessive stress distribution and abnormal joint mechanics, another aspect of the PFPS pathogenesis is related to the functional capacity of the patellofemoral joint to accept and transfer a range of loads, while maintaining tissue homeostasis. Supra-physiological loading, can result from a single

event (overloading) or by repetitive loading (overuse) and can lead to loss of homeostasis of the joint tissue. This supra-physiological loading can derange the normal physiological cellular and molecular process and can lead to structural damage described as structural failure zone. This concept outlines a crucial association between level of loading and etiopathogenesis of the PFPS (Dye, 2004, pp. 3-18).

1.2. Quadriceps femoris muscular dysfunction and patellar tracking

Abnormal lateral tracking of the patella has been proposed as the main maltracking phenomenon associated with PFPS. Lateral maltracking may increase patellofemoral contact pressure and precipitate pathology in articular cartilage (Fulkerson & Shea, 1990). One proposed mechanism for abnormal lateral tracking is an imbalance in the activity of the Vastus Medialis Obliquus (VMO) relative to the Vastus Lateralis (VL) (Insall, 1982). The VMO and VL muscles are considered as primary dynamic stabilisers of the patellofemoral joint. The imbalance could be caused either by reduction in the strength of the VMO (Ahmed et al, 1987) or altered temporal control of VMO & VL activity with delayed VMO onset time in PFPS patients (Voight & Weider, 1991). This imbalance in the active medial and lateral forces exerted by the VMO and VL is considered as one of the reasons for initiating or perpetuating patellofemoral pain syndrome (Davis & Powers, 2010; Witvrouw et al, 2010). In the former case, excessive lateral force on the patella could result due to insufficient strength in the VMO despite normal neural drive. In the later case, even a sufficiently strong VMO could produce less than the adequate forces if neural drive to the VMO is either of

inadequate magnitude or inappropriately timed, such that the VL is recruited enough in advance in relation to the VMO to cause a temporary medial-lateral force imbalance during the initial phase of the knee extensor activity (Karst & Willet, 1995).

1.3. Physiotherapy treatment for PFPS

There is general agreement that the first choice of treatment of PFPS should be directed towards quadriceps rehabilitation to address the problem outlined above (Biedert, 2004, p. xxi-xxii; Eriksson, 2006, p. vii). Consequently, most physiotherapists consider a quadriceps muscle strength assessment is essential and standardised physiotherapeutic intervention for this condition consists usually, of general quadriceps strengthening and/or vastus medialis obliquus (VMO) selective training in order to address the generalised quadriceps muscle weakness and/or the VMO – VL muscle imbalance (Fulkerson, 2002; McConnell & Bennell, 2006; pp. 167-184, Witvrouw et al, 2005; Witvrouw et al, 2006, pp. 135-145).

Furthermore, the concept of VMO & VL muscle imbalance appears to be gaining popularity amongst the physiotherapy community, clinicians and researchers. The treatment of PFPS patients by using electromyographic (EMG) biofeedback and other techniques in order to alter the relative amplitude or onset time of the VMO and VL is being advocated, although the scientific evidence seems to be inconclusive (Chester et al, 2008; Karst & Willet, 1995).

Trends in favour of VMO-VL muscle imbalance have been documented in the literature but it is evident that there is a significant degree of heterogeneity between

patients and across the studies. Indeed it might be the case that quadriceps imbalance is not a feature in all patients and it might be more appropriate to target intervention to those where it is apparent. Additionally, the existence of normative data for VMO-VL onset time and amplitude ratios is still insufficient and the clinical and therapeutic significance of the imbalance is difficult to assess (Chester et al, 2008; Werner, 2006, pp. 150-151; Wong, 2009). These deficiencies in the literature need to be addressed if treatment strategies for PFPS are to advance.

Overall aims and objectives

The ultimate aim of the study was to establish therefore if it is appropriate to continue addressing a VMO – VL muscle imbalance, and treating with physiotherapeutic interventions, patients with clinically defined patellofemoral pain syndrome (PFPS).

In order to address this aim the objectives were to establish if a VMO – VL muscle imbalance actually exists in a clinically defined PFPS population and to compare this to a pain free population to determine if this imbalance is related to clinical symptoms associated with the condition and/or lower limb muscle physiology.

Before proceeding with the specific aims of the study outlined in Chapter 6 a detailed search of the literature was undertaken. The purpose of this review was to establish the position in relation to current knowledge and in light of this knowledge to define the specific objectives.

Chapter 2

The patellofemoral joint

2. THE PATELLOFEMORAL JOINT

2.1. Anatomy of the patellofemoral joint

The patellofemoral articulation is a sellar joint between the femoral trochlea and the patella. The osseous components and the multiple periarticular soft tissues of the joint form a functional unit with complex synergistic functional interplay. The asymmetrical design and the functional morphology of the patellofemoral joint sustain high biomechanical loads (Biedert & Friederich, 2004, pp. 21-23; Goldblatt & Richmond, 2003).

2.1.1. The patella and the femoral trochlea

The patella is the biggest sesamoid bone of the body, embedded in the tendon of the quadriceps femoris muscle. The articular cartilage of the patella is the thickest cartilage of the human body and is avascular and aneural. The extraordinary thickness of the cartilage suggests that the patella is subjected to high joint forces, comprising an excellent example of the belief that "form follows function". The articular cartilage of the patella does not follow the topography of the underlying bone, a distinctive characteristic that should be borne in mind when interpreting x-rays (Grelsamer & McConnell, 1998, pp. 11-23; Oatis, 2004, pp. 761-773).

The articular surface of the patella has a central vertical ridge that creates a medial facet articulating with poor congruence to the medial trochlea, and a larger lateral facet articulating with reasonable congruence to the lateral trochlea. On the medial border of the medial facet a second smaller vertical

ridge forms a third smaller facet known as the 'odd' or border facet (Figure 2.1). The patellar facets vary considerably in shape and size. The central patellar ridge corresponds to the 'V' shaped trochlear groove consisting of the medial and lateral femoral condyles at the distal end of the anterior femur. The lateral condyle is always higher than the medial femoral condyle in normal knees. This is particularly characteristic of bipedal animals, and also a genomically specified mechanism of patellar retention. The variability of the shape and morphology of the osseous structure of the patella and distal femur is reflected in knee joint stability (Biedert & Friederich, 2004, pp. 21-23; Grabiner et al, 1994; Grelsamer & McConnell, 1998, pp. 11-23; Oatis, 2004, pp. 761-773).

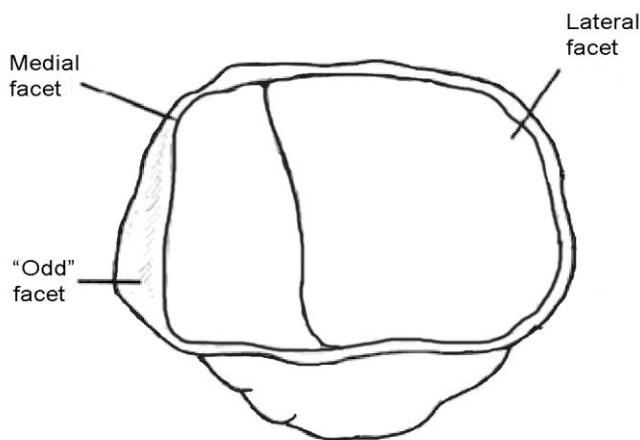


Figure 2.1: Articular surface of the patella.

2.2. Anatomy of the quadriceps femoris muscle

Although almost every portion of the human anatomy from the pelvis down (including hips and feet) has an effect on the extensor mechanism, the extensor mechanism properly begins above the hip joint and terminates at the tibial tuberosity. The extensor mechanism includes the four muscles of the quadriceps, the patella, the patellar tendon, all the other patellar tissues that attach to the patella, and the tibial tuberosity. In addition, the blood supply and innervation are key parts of the anatomy of the extensor mechanism. The quadriceps femoris muscle is probably the most active component of the complex extensor mechanism of the knee joint (Grelsamer & McConnell, 1998, pp.11-23; Oatis, 2004, 740-745).

The quadriceps femoris forms the main bulk of the anterior thigh muscle and collectively constitutes the largest and the most powerful muscles of the human body. The quadriceps consists of the rectus femoris (RF), the vastus intermedius (VI), vastus lateralis (VL) and vastus medialis (VM). The vastus medialis can be subdivided into vastus medialis longus (VML) and vastus medialis oblique (VMO). The quadriceps femoris has an extensive attachment to the femur, winding obliquely around it to attach to the patella, and via tendinous expansions (retinacula) to the tubercle of the tibia. All parts of the quadriceps are innervated by the femoral nerve. Taken as a group, the four parts act as extensors of the knee (Williams and Warwick, 1995, pp.637-640; Moore and Dalley, 1999, p.230).

The rectus femoris is the only one of the four muscles to cross the hip joint and therefore have the additional function of flexion of the hip (Williams and Warwick, 1995, pp.637-640; Moore and Dalley, 1999, p.232). The line of action (direction of the pull) of the rectus femoris is not parallel to the femoral shaft, but rather subtends an angle of about 5 degrees with the femoral shaft (this is said to be the insertion angle of the muscle)(Grelsamer & McConnell, 1998, pp.11-23).

The vastus intermedius has a line of action similar to that of the rectus femoris but differs in two ways: its origin is on the proximal part of the femur (therefore does not cross the hip joint), and its line of action is directly in line with the femur (it therefore forms an angle of about 5 degrees with the rectus femoris)(Grelsamer & McConnell, 1998, pp.11-23).

The quadriceps femoris plays a very important role in both maintaining the integrity of the knee joint and in the knee's function. The quadriceps femoris is a large and powerful muscle capable of generating in excess of 1000 lb (4450 N or 2200 kg) of internal force. Such force is needed in close kinetic chain (CKC) motion to elevate and lower the body, as in rising from a chair, climbing, and jumping, and to prevent the knee from collapsing in walking, running or landing from a jump. Here the quadriceps mechanism provides an active restraint to the femoral condyles on the tibial plateau to supplement passive restraints such as the posterior cruciate ligament and joint contours (Oatis, 2004, pp. 740-745; Smith et al, 1996, p.318).

2.2.1. Anatomy of Vastus Medialis and Vastus Lateralis

A. Vastus Medialis

The vastus medialis muscle has two distinct parts with different fibre orientation and specific functions: the vastus medialis longus (VML) and the vastus medialis oblique/obliquus (VMO). The vastus medialis longus (VML) originates from the medial aspect of the upper femur and inserts anteriorly into the quadriceps tendon. The angular fibre orientation is 15-18° off the long axis of the femur (figure 2.2, angle a) (Lieb & Perry, 1968; Bose et al, 1980; Reider et al, 1981; Williams & Warwick, 1995, pp.637-640; Nozic et al, 1997). The pennation angle for the VML has been estimated to be about 5 degrees (Wickiewicz et al, 1983).¹

The vastus medialis oblique (VMO) originates mainly from the tendon of the adductor magnus although some fibres arise from the adductor longus and the medial intermuscular septum (Bose et al, 1980; Thiranagama, 1990; Javadpour et al, 1991). The VMO has angular fibre orientation of 50-55° from the long axis of the femur in the frontal plane (figure 2, angle b) (Lefebvre et al, 2006; Lieb and Perry, 1968; Bose et al, 1980; Reider et al, 1981; Nozic and al, 1997). The physiological cross-section of the VMO has been estimated to be approximately 30% of the entire vastus medialis complex. The above features contribute to making the VMO a critical dynamic medial stabilising force (Raimondo et al, 1998).

¹ The pennation angle is the angle formed by the individual muscle fibres with the line of action of the muscle, and it is expressed as an average for the entire muscle. When all the fibres are essentially parallel to the line of pull, as with the rectus femoris or vastus intermedius, the pennation angle is 0 degrees. At the other extreme are the pectoralis major and deltoid, which are fan shaped (Wickiewicz, et al, 1983).

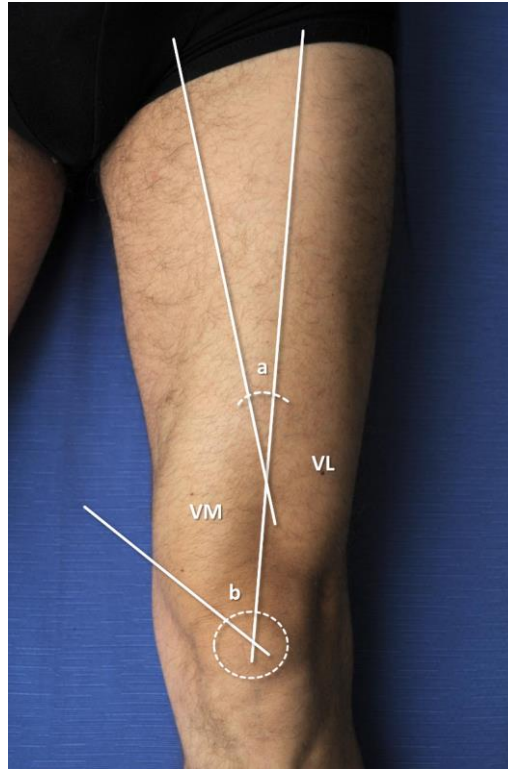


Figure 2.2: Measurement of the muscle fibre orientation in the VMO and VML: VM= vastus medialis muscle, VL= vastus lateralis muscle, a - VML= proximal angle - 15° - 18°, b - VMO= distal angle - 50° - 55°.

It is usually not easy to see where the VML ends and where the VMO begins. Grelsamer & McConnell (1998, pp. 11-23) states that in some patients, a thin layer of fat can be seen separating the two parts of the muscle. There have been reports in the literature of an “areolar fascial plane” (Lieb & Perry, 1968) or a fascial “investment,” in some cases, (Reider et al, 1981) separating the proximal and distal parts. Nozic et al (1997), in his study with 50 cadaver specimens found, only a single case of fascial plane division between the muscle parts. This non-significant result (one in 50 specimens) can be regarded, according to the author, as the exception rather than the rule. According to the results of a recent systematic review there is a substantial alteration in fibers alignment between the proximal

and distal muscle portion. Additionally, a fibrofascial plane was seen dividing VMO from VML in small proportion of both normal and pathological knees, but they concluded that the existing evidence is inconclusive in regard to the division of the VM in two separate components (Smith et al, 2009).

Investigation of the nerve supply to the two parts of vastus medialis has revealed some interesting findings. Lieb and Perry, (1968) reported that each part of the muscle has a separate nerve trunk which they refer to as a separate branch of the femoral nerve. Weinstabl et al (1989) further describe the branch of the femoral nerve, as lying in the plane of separation of the muscle parts. According to the study of Thiranagama (1990), the VMO receives a richer nerve supply from the femoral nerve compared to the VML. This may account for the higher action potentials recorded for the VMO in the study of Lieb and Perry (1971).

Gunal et al (1992) discovered that a branch of the saphenous nerve consistently went to the VMO. Stimulation of this branch could cause a VMO contraction, which shows the saphenous nerve as not just purely a sensory one. It was therefore suggested that injury to this supposed "sensory" nerve could lead to VMO malfunctioning and therefore might be important as a cause of patella malalignment. On the other hand Nozic et al (1997) argue that only branches of the femoral nerve supply the two parts of the muscle, with no particular pattern to the branching observed, and no other nerve seen to give off branches to supply this muscle in the 50 specimens studied. As a conclusion, the authors state that the

muscle should be classified as a single muscle with proximal and distal components.

Despite the previously described inconsistent findings, it is generally accepted that according to anatomic, physiologic and mechanical characteristics the vastus medialis is functionally classified into two distinct portions: the proximal part is the VML and the distal part is the VMO (Kasman, 1998, pp.142-147; Werner, 2006, pp.150-151) and are treated as such for the purpose of this thesis.

B. Vastus Lateralis

The vastus lateralis (VL) originates from the vastus ridge at the base of the greater trochanter and from a tough fibrous band at the posterior aspect of the femur called the linea aspera. It inserts anteriorly into the quadriceps tendon and laterally into the lateral retinaculum (Williams and Warwick, 1995, pp.637-640). It has a line of action of about 20 to 40 degrees to the long axis of the femur in the frontal plane (20 degrees for the upper fibres, up to 40 degrees for the lower fibres)(Figure 2.3) (Grelsamer & McConnell, 1998, p.11-23).

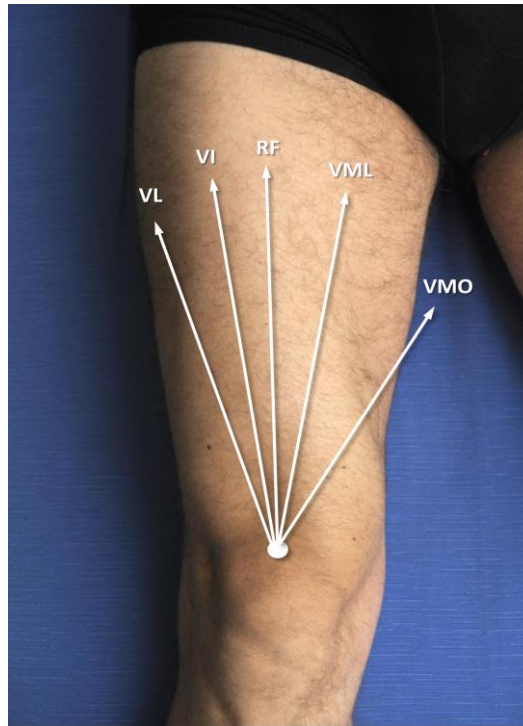


Figure 2.3: Action lines of the components of the quadriceps. VL=vastus lateralis, VI=vastus intermedius, RF=rectus femoris, VML=vastus medialis longus, and VMO=vastus medialis obliquus.

The vastus lateralis has also been described by some investigators as having two parts: vastus lateralis longus (VLL) and vastus lateralis oblique (VLO) (Hallisey et al, 1987; Weinstabl et al, 1989). The lower fibres (VLO) deviate laterally at an average angle of 32.4 degrees, and the upper fibres at 12.5 degrees from the longitudinal axis of the femur. Javandpour et al (1991) reported that an areolar fascial plane separated the lower fibres of the vastus lateralis from the main upper body of the muscle, but did not find separate nerve branches as in the vastus medialis. Unlike the vastus medialis, both parts of the vastus lateralis are generally considered to function as one integrated unit (Callaghan and Oldham, 1996) and are treated as such for the purpose of this thesis.

2.3 Phylogenetical evolution of the Vastus Medialis and Vastus Lateralis

The quadriceps femoris group, are muscles which are late developing in humans. This can be confirmed by observing the relatively small size and bulk of this muscle group in quadrupeds, and even in biped animals (Dye, 1987). The vastus medialis muscle is the last muscle to develop in the phylogenetically late developing quadriceps group. The vastus medialis is also the weakest muscle phylogenetically, and therefore the first to undergo atrophy of disuse. It is also the last to be rehabilitated after immobilisation, injury or surgery on the knee joint (Fox, 1975; Grana & Kriegshauser, 1985). Subsequently, it is postulated that possibly due to this reason vastus medialis hypotrophy is a common finding in patients with patellofemoral pain (Werner, 2006, p.150).

2.4 Biomechanics

The basic function of the patella is to increase the efficiency and mechanical advantage of the quadriceps muscle by increasing the knee extension moment by as much as 50%. The patella also, guides the forces produced by components of the patellar ligament, offers protection on the anterior part of the knee and distributes the compressive forces on the anterior part of the femur by increasing the contact area (Amis & Farahmand, 1996; Cox, 1990; Grabiner et al, 1994).

The articular surfaces of the joint are rather incongruent and there not configured to ensure great amount of stability. The patella, a small bone with considerably large range of motion, is responsible for accepting high forces from a range of

directions deriving from the surrounding powerful muscles. The normal function of the patellofemoral joint relies on multiple interactions between various mechanisms which can be mainly classified as follows: static stability factors – the geometry of the articular surfaces, active stability factors – the muscle tension, and finally passive stability factors – the retinaculae (Amis et al, 2004, pp. 37-53; Amis & Farahmand, 1996).

2.4. 1 Patellofemoral joint reaction forces

The existence of the extraordinary thick articular cartilage seen in the patella indicates that the joint is subjected to high forces. Patellofemoral joint reaction force (PFJRF) is a compressive force acting on the joint (Figure 2.4). The quadriceps pulls proximally on the patella (Amis et al, 2004, pp. 37-53, Oatis, 2004, pp 768-771). The patella function is considered as a non frictionless pulley and a simplified estimation of the patella forces considers that the magnitude of the quadriceps proximal pull is equal to the magnitude of the patellar ligament distal pull (Mason et al, 2008). When the knee is close to extension the PFJRF is smaller than the patellar ligament force and quadriceps tendon force. In a flexed knee position, for the same magnitude of patellar ligament tension the PFJRF exceeds the patellar ligament tension. This is the underlying mechanism that explains the “movie sign”, the pain experiences a PFPS patient after prolonged sitting with flexed knees (Amis et al, 2004, pp. 37-53).

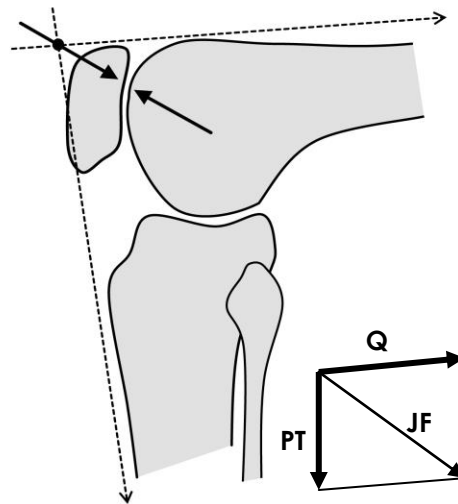


Figure 2.4: Near knee extension, the patellar tendon (PT) tension and quadriceps (Q) tension oppose each other and result in a small joint force, JF. In the flexed knee, PT and Q combine to cause a much larger joint force, JF, for the same value of PT. The quadriceps tension Q is also much larger, for the same PT, in the flexed knee. The joint force JF is distal on the patella in the extended knee, and proximal in the flexed knee, because it must be directed at the intersection of the lines of PT and Q.

The increase in patellofemoral contact force during knee flexion does not inevitably increase contact pressure on the articular cartilage because the contact area increases significantly during knee flexion, therefore the ratio of the force per unit contact area is not increased in the same way as the joint force. This explains the concave shape of the patella in the sagittal plane and its congruency with the femur in the proximal part of the articular surface (Amis et al, 2004, pp. 37-53). The PFJRF range from over 800 N (180 lb) in level walking to over 5000 N (1125 lb) in more demanding activities such as running and dancers' jump landing (Simpson et al, 1996).

2.4. 2 Patellofemoral joint contact areas

PFJRF should always be considered in relation to the contact areas of the patellofemoral joint and the produced joint stress ($\text{stress} = \text{force} / \text{area}$). When the knee starts to flex, the initial contact area on the patella is on the distal end of the central ridge, as the convex surface starts to meet the sulcus. When the patella engages the femoral trochlea at approximately 20° of the knee flexion the contact areas spread rapidly across the width of the distal patella. As knee flexion progresses the contact area migrates proximally to the concave area allowing the patella to fit congruently to femur and thus enabling the joint force to spread over a great area. This particular pattern of increasing contact area in order to decrease the stress fits perfectly with the rise of the PFJRF as the knee advances into flexion (Amis et al, 2004, pp. 37-53; Grabiner et al, 1994).

Additionally, knee kinematics controls the location of the contact area. When the knee is flexed over 90° there are two distinct contact areas, the lateral facet of the patella rests on the distal aspect of the lateral femoral condyle while the 'odd' medial facet wedges lie against the lateral-facing slope of the medial femoral condyle, at the edge of the intercondylar notch (Amis et al, 2004, pp. 37-53; Besier et al, 2005; Brechter et al, 2002).

High quadriceps tension, poor contact areas, indirect factors such as increased activity and shortening of hamstrings can cause translation and external rotation of the tibia which consequently can increase the PFJRF (Li et al, 2004). Furthermore, lateralisation of the quadriceps force vector can alter the PFJ

contact areas patterns (Whyte et al, 2010). Increased PFJ stress can disturb the tissue homeostasis and has been implicated in the pathogenesis of PF pathology (Dye, 2001; Holmes & Clancy, 1998).

2.4. 3 Quadriceps angle – Q angle

The quadriceps angle or Q angle was first defined by Brattström in 1964 as the angle formed by the line of pull of the quadriceps mechanism and that of the patellar tendon as they intersect at the center of patella. The Q angle is considered as measure of the patellar tendency to move laterally when the quadriceps muscles are contracted. The greater the Q angle, the greater this tendency (Fredericson & Yoon, 2006; Smith et al, 2008). The Q angle is formed by the intersection of the line of application of the quadriceps force - line from the anterior superior iliac spine to the center of the patella – with the center line of the patellar tendon – line from the center of the patella to the tibial tuberosity (Figure 2.5).



Figure 2.5: The quadriceps (Q) angle

The Q angle values varies from 11° - 14° for men and $15,8^{\circ}$ - 17° for women (Aglietti et al, 1983; Horton & Hall, 1989). The relationship between greater than normal Q angle and patellofemoral pain syndrome is controversial, some studies demonstrated a clear relationships between greater Q angle and PFPS patients (Aglietti et al, 1983), but other studies have demonstrated no difference between Q angle values in patients with PFPS and asymptomatic subjects (Caylor et al, 1993; Thomeé et al, 1995).

Sound scientific evidence has formed a clear consensus that the etiopathogenesis of the PFPS is multifactorial and one of the proposed factors is the relationship between the PFPS and the alignment and mechanics of the patella (Davis & Powers, 2010). Patellar maltracking is evident in PFPS patients (Draper et al, 2009).

The role of the VMO as an important medial stabilizer of the patellofemoral joint is confirmed by modeling and cadaveric studies (Elias et al, 2009; Farahmand et al, 1998; Lin et al, 2004; Neptune et al 2000). The VMO muscular dysfunction in terms of EMG magnitude and onset time has been implicated in patients with PFPS (Cowan et al, 2002; Voight & Wieder; 1991; Witvrouw et al, 2002), but this finding is not consistently evident in all studies (Brindle et al, 2003; Powers et al, 1996). Altered neuromuscular coordination between VMO and VL has been identified as a risk factor for the development of the PFPS in prospective studies (Thijs et al, 2007; Witvrouw et al, 2000). The disparity of the evidence is probably attributed, among others, to the fact that individuals with muscular dysfunction of VMO-VL are a subgroup of the total population of individuals with PFPS. Subsequently, future research should focus on the identification of subgroup of individuals with VMO-VL muscular dysfunction because this specific subset of PFPS patients may require specific rehabilitation of the vasti muscles and not just generalized quadriceps strengthening (Crossley, 2010).

Chapter 3

Patellofemoral Pain Syndrome

3. PATELLOFEMORAL PAIN SYNDROME

3.1 Nomenclature

Plethora of names has been used and there is little consensus about the terminology employed to describe the pain in the anterior part of the knee (Näslund et al, 2006; Thomeé et al, 2002). Chondromalacia patellae (CMP) was the first term used in the late 1920s, by Aleman, because the pain was attributed to pathological alterations (macroscopic softening, fissuring, fragmentation) in the patellar cartilage (Bentley et al, 1984; Grelsamer & McConnell, 1998, p. 6). During 1970s and 1980s through the development and advances in arthroscopic surgery it was discovered that there was no direct link between patellae cartilage pathology and patellofemoral pain. Arthroscopy of patellofemoral joint demonstrated that, in some cases of patients diagnosed with condromallacia patellae (CMP) was no evident cartilage pathology, and also in other cases, patients with evident cartilage pathology had no patellofemoral pain (Casscells, 1979; Lindberg et al, 1986).

Although condromallacia patellae (CMP) occasionally exists in some cases (Holmes & Clancy, 1998), this term is rarely used any more, and the last three decades has been gradually replaced mainly by names such as, patellofemoral pain syndrome (PFPS) (Crossley et al, 2002; Kannus & Niittymaki, 1994; Lindberg et al, 1986; Merchant, 1988) and anterior knee pain (AKP) (Cutbill et al, 1997; Garrick, 1989; Radin, 1985; Van Tiggelen et al, 2004). Although several suggestions has been proposed in terms of aetiology of the patellofemoral pain (Insall, 1979; Insall,

1982; Merchant, 1988; Witvrouw et al, 2004), nevertheless, the literature little consensus provides regarding the terminology for this pain syndrome (Witvrouw et al, 2004) and terms such as, idiopathic anterior knee pain (Holmes & Clancy, 1998), patellalgia (Percy & Strother, 1985), patellofemoral malalignment (Goldenberg, 1997), extensor mechanism disorder (Grana & Kriegshauser, 1985), patella compression syndrome (Doucette & Child, 1996) has also been used synonymously with PFPS (Thomeé et al, 2002). The term anterior knee pain is considered to encompass all pain related conditions of the anterior aspect of the knee and when during the clinical evaluation conditions as, intra-articular pathology, synovitis or bursitis are excluded, the remaining clinical signs & symptoms can be termed PFPS (Thomeé et al, 2002). For the purpose of this thesis we have chosen to use the term patellofemoral pain syndrome (PFPS).

3.2 Prevalence & characteristics

Twenty-five percent of knee problems are PFPS related (Devereaux & Lachman, 1984). In a USA university sports medicine department, Dehaven & Lintner (1986) was recorded for a seven year interval a 7,4% and 19,6% prevalence of PFPS and CMP of all injuries for male and female respectively. These percentages represented the 18,1% of male and 33,2% of female total knee injuries. In an outpatient sports clinic setting in Finland was found a 8% CMP prevalence of the total knee disorders (Kannus et al, 1987) and in Israel a 15% patellofemoral pain incidence of male army recruits was reported (Milgrom et al, 1991). In more recent studies, Bolling et al (2009) reported a 13,5% prevalence of PFPS in 1525 USA

navy recruits for a period of 2,5 years. The female PFPS prevalence was higher than the male with 15,3% and 12,3% respectively. Myer and associates (2010) studied 240 USA middle and high school female basketball athletes and reported a preseason patellofemoral pain (PFP) prevalence of 16,3%. The total preseason and season prevalence rate was found to be 22%. Finally, in a study with high school students in Denmark was reported a knee pain prevalence of 25% and the PFPS prevalence was 6% (Mølgaard et al, 2011). Conclusively, it is clearly evident that despite the cited PFPS prevalence, the data is derived mainly from studies based in school & university athletes, army recruits and school students. Therefore, robust epidemiological studies are necessary in order to expand the investigation of the prevalence of the PFPS in the general population (Callaghan and Selfe, 2007).

The clinical picture of PFPS is characterized by insidious and gradual onset of peripatellar pain usually not related to trauma or diagnosed pathology (Arrol et al, 1997; Garrick, 1989; Powers, 1998), although in some cases can be caused by trauma and the initial onset of pain can be acute (Dixit et al, 2007; Varatojo, 2010, p. 35-36). Other complaints include, crepitus, catching, giving way, occasional sensation of stiffness and sensation of swelling (Galanty et al, 1994; Thomeé et al, 1995; Thomeé et al, 2002; Varatojo, 2010, pp. 35-36). Usually the symptoms are aggravated during and/or after activity (Crossley et al, 2001; Crossley et al, 2002; Powers, 1998) and they are provoked by conditions that increase the patellofemoral joint stress and compression, thus resulting in inflammatory pain (Insall et al, 1982; Merchant, 1988). Activities that induce the symptoms are, stair

ascending/descending (Crossley et al; 2002, Powers, 1998; Wilson et al, 2003), prolonged sitting with flexed knees (Cowan et al, 2001; Crossley et al, 2002; Galanty et al, 1994), squatting and kneeling (Callaghan et al, 2001; Cowan et al, 2001; Cowan et al, 2002; McConnell, 2002) running, hopping/jumping (McClinton et al, 2007; Mohr et al, 2003; Stensdotter et al, 2006). Compounding these activities lead to chronicity of PFPS (Crossley, 2010; McConnell, 2002) and in some cases can cause restrictions or even cessation of the physical activity of the patients (Crosley, 2010; Devereaux & Lachman, 1984; Sandow & Goodfellow, 1985). Conclusively, PFPS can have a substantial multi-level impact on quality of life affecting performance of work, leisure and sport activities of the patients (Crosley, 2010).

Several theories have been proposed that attempt to describe the aetiology behind the provocation of the PFPS symptoms and the most widely accepted is the theory of excessive patellofemoral joint stress caused by abnormal tracking of the patellofemoral joint and thus provoking inflammatory pain (Insall et al, 1982; Merchant, 1988). Increased intraosseous pressure has been also been proposed that can result in ischemic pain (Hejgaard & Diemer, 1987). Another theory is related to neurogenic mechanisms, either due to neuromas and nerve tissue injuries of the lateral retinaculum (Mori et al, 1991; Sanchis-Alfonso et al, 1998) or due to patellar reflex sympathetic dystrophy (Merchant, 1988). Dynamic metabolic adaptations with bone turnover augmentation can also lead to subchondral bone pain (McCarthy, 1997; Dye & Boll, 1986).

3.3 Contributing factors of PFPS Aetiopathogenesis

Thorough understanding of the factors related to manifestation, development and perpetuation of the PFPS is essential to guide for the clinicians in the evaluation and in the treatment of the syndrome. Several attempts have been made in order to identify and clinically classify the contributing factors. A clinical classification system of the PFPS proposed by field experts of the European Rehabilitation Panel is dividing the contributing factors, according to their nature, in two main categories: a) factors related to malalignment and b) factors related to muscular dysfunction (Witvrouw et al, 2005). In the table 1 bellow is presented analytically the proposed by the European Rehabilitation Panel clinical classification system.

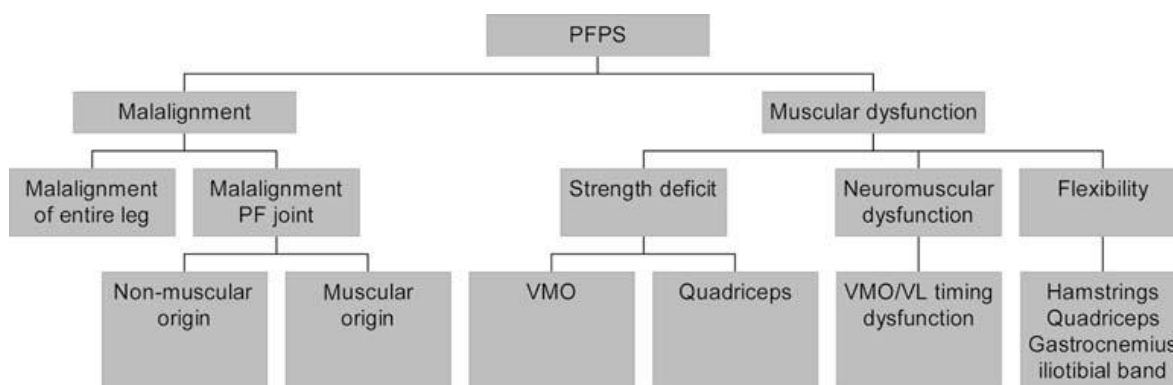


Table 1: Clinical classification system of PFPS according to European Rehabilitation Panel (adapted from: Witvrouw et al, 2005).

Another classification system was proposed recently by the PFPS International Research Retreat is using topographic anatomic characteristics and is dividing the aetiological factors to: a) local factors, related to patellofemoral joint and surrounding tissues, b) distal factors, related to foot and ankle mechanics and c) proximal factors, related to hip and pelvis (Davis & Powers, 2010).

3.3.1 Muscular Dysfunction

A. Quadriceps Muscle Strength Deficit

Generalised quadriceps weakness is evident in patients with PFPS and various patterns of muscle strength deficit have been reported (Witvrouw et al, 2005). Muscular weakness of the quadriceps has been observed during isokinetic evaluation of contractions in PFPS patients in comparison to healthy subjects (Stiene et al, 1996). Dvir and associates (1992) reported significantly reduced quadriceps strength of PFPS group compared to matched (age & activity) control group. The strength deficit was homogenous throughout the velocity spectrum (30°/s, 60°/s, 120°/s) and the average reduction was 34% in concentric and 39% in eccentric contraction. In a similar study by the same research group is also been demonstrated a 30%-40% quadriceps strength reduction of the PFPS patients compared to healthy (Dvir et al, 1990). Werner (1995) also found that patients with PFPS compared with a healthy control had considerably lower quadriceps isokinetic torque. Reduced quadriceps isokinetic torque has been identified in the athletic population. Isokinetic quadriceps muscle weakness has been found in runners suffering from PFPS (Duffey et al, 2000), and in high level football players (Olmo et al, 2007).

In addition, prospective studies have shown that isokinetic quadriceps muscle strength deficit is evident prior to the military training in sub-groups of healthy male and female army recruits which after the basic military training developed PFPS. Specifically, significantly reduced knee extension concentric contraction was found in male army recruits at 60°/s (Van Tiggelen et al, 2004a), and lower

concentric peak torque of quadriceps at 60°/s and 240°/s in female army recruits (Duvigneaud et al, 2008). The authors emphasised that although the nature of the PFPS is multifactorial, individuals with quadriceps muscle strength deficit are more prone to PFPS. Van Tiggelen et al (2004a) argues that confirmation of the association between quadriceps muscular strength deficit and PFPS comes from the fact that, interventional studies involving patients with PFPS have found that improvement in symptoms are related to increase of quadriceps muscle function either in terms of strength (Kannus & Niittymaki, 1994; Powers, 1998; Werner & Eriksson, 1993) or neuromuscular improvement (Gilleard et al, 1998). The results from a recent systematic review are supporting the clinical effectiveness of the supervised knee extension exercise on improvement of the knee function of PFPS patients (van Linschoten et al, 2012).

B. VMO-VL Muscle Imbalance

The VMO-VL muscle imbalance as assessed by electromyographic magnitude-ratio and onset time, remains up to today a controversial issue. Several studies have reported the implication of VMO-VL muscle imbalance in PFPS either in terms of magnitude-ratio (Boucher et al, 1992; Cesarelli, et al, 1999; Owings et al, 2002; Tang et al, 2001; Taskiran et al, 1998) or in terms of onset time (Cesarelli et al, 1999; Cowan et al, 2001; Cowan et al, 2002,) and reflex response (Voight & Wieder, 1991; Witvrouw et al, 1996). In contrast, other studies have found no implication in all aspects of the VMO-VL muscle imbalance in patients with PFPS (Brindle et al, 2003; McClinton et al, 2007; Powers et al, 1998; Sheehy et al, 1998; Stensdotter et

al, 2006). According to a systematic review-meta analysis of Chester et al (2008), overall tendency of delayed onset time of the VMO relative to VL is found in PFPS patients, and Wong (2009) also believes that “the evidence, although exists is not convincing yet”.

The controversial evidence possibly is attributed to several reasons:

- A) The most important is the complex nature of the PFPS aetiopathogenesis, therefore the muscular dysfunction of the vasti muscles is a feature of a subgroup of patients (Cowan et al, 2001; Chester et al, 2008; Crossley, 2010).
- B) Possible sources of heterogeneity such as *methodology, population characteristics and procedure characteristics* (Chester et al, 2008).

It is believed that the described above tendency of the VMO-VL muscle imbalance eventually can be reinforced by the following evidence found in the literature:

- A) Atrophy of the VMO – Magnetic resonance imaging (MRI), which is considered as the gold standard for muscle size assessment (Engsrtom et al, 1991), has revealed a 2cm² smaller cross section area (CSA) of the VMO in patients with PFPS in comparison to healthy controls. Authors reported to be the first MRI study of the CSA VMO (Pattyn et al, 2011). Another MRI study revealed total quadriceps volume decrease in PFPS patients. Significantly reduced VMO volume in PFPS patients in comparison to controls has also been documented by sonography (Jan et al, 2009). Generalised atrophy of

the entire quadriceps muscle in PFPS patient, (3,38% difference, but not significant) is also been reported by the sonographic study of Callaghan & Oldham (2004). According to the VMO phylogenetic characteristics, the muscle is prone to hypotrophy (Fox, 1975; Grana & Kriegshauser, 1985) and selective VMO hypotrophy is considered to be a common clinical finding in patients with PFPS (Werner, 2006, p. 150; Witvrouw et, 2005).

- B) Neuromuscular dysfunction of the VMO-VL is identified as an intrinsic factor for the PFPS in series of prospective studies (Witwrouv et al, 2010; Van Tiggelen et al, 2012).
- C) Chester and associates (2008) and Van Tiggelen and associates (2004b) suggest that the identifiable tendency of VMO-VL muscle imbalance in PFPS patients will probably be increased if evidence that therapeutic interventions can influence it in a favorable manner. Indeed, there is evidence confirming that the VMO delayed onset can be improved via specific treatment such as pain reduction and patella taping (Bolling et al, 2006; Cowan et al, 2002a; Cowan et al 2003; Gilleard et al, 1998).

C. The Role of Hip Abductors & External Rotators Muscle Deficit

Hip and pelvis muscles contribute to the normal lower limb alignment during weight bearing conditions and therefore have been part of the clinical examination in patients with PFPS (Crossley, 2010; Grelsamer & McConnel, 1998, pp.110-113; Werner, 2006, p.169-173). Impaired function mainly of hip abductors & external rotators muscles can alter femur kinematics and influence patellofemoral joint mechanics (Souza & Powers, 2009). Reduced strength or neuromotor control

of the external rotator muscles can lead to internal hip rotation which is associated with increased patellofemoral stress (Lee et al, 2003). Hip abduction muscle impairment can contribute to dynamic valgus of the lower limb and consequently to increase the lateral forces acting on the patellofemoral joint (Powers, 2003). A recent systematic review reveals muscle weakness of hip external rotation, abduction and extension (Prins & van der Wurf, 2009), and additionally delayed onset of the hip abductors is evident in PFPS patients in comparison to healthy controls (Brindle et al, 2003; Cowan et al, 2009). Probably PFPS patient with hip muscle impairment constitute a subgroup of PFPS population and further research is necessary in order to expand the existing knowledge of the underlying mechanism (Crossley 2010; Davis & Powers, 2010).

D. Muscular Flexibility Deficit

Muscle tightness of the lower limb is a common finding in the clinical evaluation of PFPS patients. Although the effect of some of these muscular impairments based on, either theoretical rational or research evidence, they have been suggested to contribute to the development of PFPS (Piva et al, 2009). It is accepted that they can alter the biomechanics by causing patellofemoral malalignment (Grelsamer & McConnell, 1998, p. 113-116; Werner, p.151).

1. Quadriceps muscle tightness

Quadriceps muscle tightness is a common finding and can be distinguished in two cases: a) tightness of the entire quadriceps muscle which limits the knee flexion range of motion (ROM), and b) isolated tightness of the biarticular rectus femoris

which limits ROM in the combined movement of hip extension and knee flexion (Oatis, 2004, pp. 740-745). Quadriceps muscle tightness can pull the patella superiorly (Hertling & Kessler, 1996, pp. 315-378) and thus to increase the patellofemoral joint stress and to predispose individuals either to develop symptoms or to increase the symptoms (Hertling & Kessler, 1996, pp. 315-378; Post, 2005; Witvrouw et al, 2000). Presence of tightness is evident in PFPS patients (Duffey et al, 2000; Piva et al, 2005; Smith et al, 1991; Witvrouw et al, 2000). In one study although is reported tightness to be present in 61% of the patients, they did not refer to the significance of the findings (Kibler, 1987), and in another study there was no difference between flexibility of PFPS patients and healthy controls (Papadopoulos et al, 2012).

2. Hamstrings muscle tightness

Tightness of the hamstrings muscles theoretically can cause slight flexion of the knee during activities and subsequently to force quadriceps contract harder in order to overcome the passive resistance of the hamstrings and thus to increase PF joint reaction forces (Piva et al, 2005). Another possible effect of hamstring tightness that has been described is the increase of the dynamic Q angle resulting in lateral tracking of the patella (Grelsamer & McConnell, 1998, p. 113-116). This theoretical rationale indirectly is supported by evidence because has been demonstrated in healthy individuals that, hamstrings tightness can reduce medial PF joint contact area and increase lateral PF joint stresses during squatting at 60° (Whyte et al, 2010). Additionally, in PFPS, patients have been reported higher PF contact forces due to increased co-contraction forces of the quadriceps &

hamstrings (Besier et al, 2009). Loading of the hamstrings, *in vitro* has been reported to translate posterior and rotate exterior the tibia (Elias et al, 2011; Kwak et al, 2000). Has been demonstrated also *in vivo* that the posterior translation of the tibia increases the anterior-posterior tilt of the patella (patellar flexion) (Seisler & Sheehan, 2007) and tibial external rotation leads to patellar lateral glide (lateral patellar shift) (Shehhan et al, 2009). Four studies have found hamstrings tightness in PFPS patients (Papadopoulos et al, 2012, Piva et al, 2005, Smith et al, 1991, White et al, 2008) and one study is reported 23% of tightness in patients but without p value reported (Kibler, 1987).

3. Iliotibial Band – Tensor Fascia Lata complex tightness

Iliotibial band (ITB) is anatomically connected to the lateral retinaculum and patella and to the lateral tibial tubercle. In cases of tightness of ITB, during knee flexion is caused lateral tracking and tilting of the patella and the lateral PF joint stresses are increased (Fredericson & Yoon, 2006; Grelsamer & McConnell, 1998; p.113, Winslow & Yoder, 1995). An additional result of the ITB tightness seen in some PFPS patients is overstretching of the medial retinaculum (Grelsamer & McConnell, 1998, p.113). *In vitro* is demonstrated that increased tension in ITB is responsible for a) patellar lateral translation (glide) and tilt which is causing increased lateral cartilage pressure, and b) increased tibial external rotation resulting in greater Q angle (Merican & Amis, 2009). Tightness of the ITB has been found in some studies with PFPS (Hudson & Darthuy, 2008; Puniello, 1993; Winslow & Yoder, 1995). Kibler (1987) is also reported 67% ITB tightness in his study without to report p values. Two

studies reported no difference in ITB flexibility between PFPS patients and healthy controls (Papadopoulos et al, 2012; Piva et al, 2005).

4. Plantar Flexor muscle tightness

Gastrocnemius and soleus tightness has been reported to have a complex kinetic chain effect. This reduces the range of motion of the ankle dorsi-flexion thus resulting in excessive subtalar joint pronation and internal rotation of tibia followed by internal rotation of the femur and thus is increasing the Q angle and consequently the PF joint stresses (Piva et al, 2005). Significant tightness of gastrocnemius muscle has been found in two studies comparing PFPS patients with healthy controls (Piva et al, 2005; Witvrouw et al, 2000) but others found no difference in flexibility of the calf muscles between patients and healthy (Duffey et al, 2000; Papadopoulos et al, 2012).

3.3.2 Structural and Postural Alterations of the Lower Extremity

Biomechanical characteristics related to structural or postural alterations such as quadriceps angle (Q angle) and abnormal foot pronation have been associated with the development of PFPS (Powers et al, 1995). Both measure Q angle and foot pronation and are an essential part of a thorough clinical examination and successful management of these structural – postural alterations has been considered a prerequisite for a successful long term conservative treatment (Witvrouw et al, 2005).

A. Excessive Foot Pronation

Excessive foot pronation has been associated with the development of PFPS. Excessive or prolonged foot pronation has a complex kinetic effect on the gait pattern. Foot over pronation is causing increased internal tibial rotation during the stance phase of the gait, due this subsequently the normal external tibial rotation required during knee extension in the mid-stance of gait is delayed or reduced, thus forcing the femur to compensate by increasing the internal rotation. This compensatory internal rotation of the lower extremity is increasing the dynamic Q angle (Tiberio, 1987). This alteration in the tibio-femoral kinematics is suggested to lead in reduced contact area and increased lateral PF joint compression and therefore to predispose to PFPS (Powers, 2003; Wilson, 2007). Research evidence has found that internal rotation of the femur increases the lateral contact pressure of the patella (Lee et al, 1994). PFPS patients with overpronation treated with foot orthotics reported decreased pain (Eng & Pierrynowski, 1993), but overpronation was a poor predictor of PFPS patients as has been reported that healthy runners had lower foot pronation than the runners with PFPS (Duffey et al, 2000).

B. Altered Quadriceps (Q) Angle

Data from an in vitro study have demonstrated that alterations of the Q angle, either increase or decrease the normal values, cause higher PF joint pressures and therefore may predispose to pathology (Huberti & Hayes, 1984). Increased Q angle values can lead to higher lateral PF contact pressures and higher risk for patellar dislocation. Reduced Q angle values probably not cause a medial shift of the patella but instead create a knee varus deformity and consequently increase

the medial tibio-femoral contact pressure (Mizuno et al, 2001). Some studies have demonstrated significantly higher Q angles values of PFPS patients in comparison to healthy controls (Aglietti et al, 1983; Haim et al, 2006; Messier et al, 1991) but others found no difference in Q angle between groups (Caylor et al, 1993; Duffey et al, 2000; Thomeé et al, 1995; Witvrouw et al, 2000). Although Thomeé et al, (1995) consider that there is no direct correlation between high Q angle and patellofemoral pain they stated that an abnormal Q angle may be a contributing factor in maintaining PFPS once the syndrome has been acquired. Finally, despite the traditional belief that women have greater Q angles than men, probably due to a wider pelvis, the small difference of 2.3° is attributed to height and not to pelvis dimensions between genders. Shorter people have greater Q angle values and apparently the height difference among men and women is determining these small Q angles differences (Grelsamer et al, 2005).

Chapter 4

Contribution of the VMO – VL
activity to PFPS

4. Background Literature Review

The purpose of this review is to undertake a critical analysis of the research to date relating to VMO vs VL activity and their contribution to PFPS. A search was conducted on the Medline & Cochrane databases for the period between 1979 and August 2011. The following key words were utilised in singular and in all possible combinations: *electromyography, quadriceps, VM, VMO, VL, patellofemoral pain syndrome, anterior knee pain, motor control, onset time, activation, reflex response, magnitude, ratio, fatigue, muscle performance, measurement*. The review included studies that assessed patellofemoral pain syndrome or anterior knee pain patients.

4.1.1 Review Search Strategy

The search strategy yielded 241 references. 198 studies that used only healthy subjects or measured the efficacy of specific exercise or rehabilitation intervention (RCTs, CCTs, systematic reviews), or descriptive and narrative reviews of patellofemoral pain syndrome or anterior knee pain (AKP) were excluded as they did not contribute to addressing the issue of the relative contribution VMO vs VL activity in terms of patellar maltracking. Of the remaining 43 publications 5 were excluded because two evaluated the overall quadriceps performance (Doxey & Eisenman, 1987; Werner, 1995), one focused on vastus medialis and rectus femoris only (Thomeé et al, 1995) without comparison between VMO vs VL, one used EMG Biofeedback to assess the muscle activity (O'Sullivan & Popelas, 2005) and one

study included patients suffering from dislocations or had undergone lateral release surgery (Wild, 1982). The final number of the reviewed papers was 38.

4.1.2 DETERMINING THE QUALITY OF THE PAPERS REVIEWED

For the purposes of the review a modified version of Bizzini's Quality Scale (Bizzini et al, 2003) was employed (see appendix I), and a total score from a possible 100 maximum was used as an indicator of the quality of the reviewed studies. The Bizzini's quality scale development was based on the Cochrane Collaboration Handbook and included factors that have been demonstrated to elicit bias and other factors that might affect the clinicians ability to incorporate the results into their clinical practice. The Bizzini's scale was specifically developed to judge quality of RCT's that assessed non-operative treatments for patellofemoral pain syndrome and to evaluate the quality of studies assessing VMO-VL muscle imbalance. The frame of the scale remained the same but was modified in terms of interventions and included two new items, a) the description of assessment protocol and b) technical & methodological issues. All studies and factors contributing to the quality rating are summarised in table 1. The reviewed studies were divided in three groups:

1. Studies that evaluated strength i.e. amplitude or ratio of VMO & VL electromyographic activity.
2. Studies that evaluated temporal control i.e. onset time, reflex response or synchronisation of VMO and VL.
3. Studies that assessed fatigue characteristics of the VMO & VL.

From the total 38 reviewed studies six evaluated both amplitude or ratio and onset time of the VMO and VL, thus in table 4.1 these publications are repeated in different sections and the total number presented is 46. In order to establish an initial estimation of the interrater reliability of the scores of the reviewed papers a second experienced itopic reviewer was employed. The second reviewer assessed nine out of 25 reviewed studies and the scores are also presented in the table 1. The nine papers were randomly selected and the second reviewer was blind to the score of the main reviewer.

Table 4.1: Overall findings, general information and score of the reviewed amplitude & ratio studies

1. AMPLITUDE & RATIO STUDIES						
No	Study	Outcome Measure	Patients	Results	Score in points (out of a 100max)	
					Reviewer	
					A	B
1.	Powers, 2000	Ratio	13	Not in favour	78	
2.	Ott et al, 2011	Amplitude	20	Not in favour	75,5	
3.	Santos et al, 2007	Ratio	12	Not in favour	70,5	
4.	Sheehy et al, 1998	Ratio	13	Not in favour	70	
5.	Liebensteiner et al,	Amplitude	19	Not in favour	69,5	
6.	McClinton et al, 2007	Ratio	20	Not in favour	68,5	76
7.	Stensdotter et al, 2006	Ratio	17	Not in favour	67	
8.	Powers et al, 1996	Intensity %	26	Not in favour	66	
9.	Owings et al, 2002	Amplitude	20	In favour	65	56
10.	Tang et al, 2001	Ratio	10	In favour	65	62
11.	Santos et al, 2008	Amplitude	10	In favour	64,5	
12.	Cesarelli et al, 1999	Amplitude	12	In favour	64,5	
13.	Boucher et al, 1992	Ratio	9	In favour	62,5	
14.	Mohr et al, 2003	% of max.	13	Not in favour	60	62
15.	MacIntyre, 1992	Amplitude	8	Not in favour	60	
17.	Souza & Gross, 1991	Ratio	9	In Fav.(N-N) Not in f(N)	56,5 NN 66,5 N	
18.	Taskiran et al, 1998	Ratio	18	In favour	54	
18.	On et al, 2004	Amplitude	13	In favour	48	
19.	Grabiner et al, 1992	Amplitude	8	Not in favour	46	
20.	Moller et al, 1986	Amplitude	28	Not in favour	44,5	
21.	Mariani & Carouso, 1978	MUAPS	8	In favour	38,5	

Legend: N = normalised N-N = non-normalised, **IN FAVOUR** = indicates results in favour of differences, **NOT IN FAVOUR** = indicates results not found any difference.

Table 4.2: Overall findings, general information and score of the reviewed amplitude & ratio studies

1. TIME ONSET & REFLEX RESPONSE STUDIES						
No	Study	Outcome Measure	Patients	Results	Score in points (out of a 100max)	
					Reviewer	
					A	B
1.	Cowan et al, 2001	Comp.algor. > 3SD	33	In favour	78	
2.	Pal et al, 2011	Comp.algor. > 3SD	40	Not in favour	77,5	
3.	Van Tiggelen et al, 2009	Comp.algor. > 3SD	26	In favour	76	
4.	Patil et al, 2011	Comp.algor. > 3SD	20	Not in favour	75,5	
5.	Cowan et al, 2002	Comp.algor. > 3SD	37	In favour	75	75
6.	Ng et al, 2011	Comp.algor. > 3SD	23	In fav. in VT Not in f. in PT	72	
7.	* Sheehy et al, 1998	Differenc. in onset	13	Not in favour	70	
8.	* McClinton et al, 2007	Comp.algor. > 2SD	20	Not in favour	68,5	76
9.	Witvrouw et al, 1996	Compar. Rest val.	19	In favour	67,5	
10.	Mellor & Hodges, 2005	MUAPS synchronis.	10	In favour	67,5	64
11.	* Stensdotter et al, 2006	Autoregres. algor.	17	Not in favour	67	62,5
12.	* Powers et al, 1996	Exceed 5% mmit	26	Not in favour	66	
13.	Brindle et al, 2003	> 5 SD from Rest val.	16	Not in favour	65,5	
14.	Karst & Willet, 1995	Comp.algor. > 1SD	15	Not in favour	65,5	
15.	* Owings et al, 2002	Compar. Rest val.	20	Not in favour	65	
16.	* Cesarelli et al, 1999	Temporal identif.	12	In favour	64,5	
17.	*Santos et al, 2008	Comp.algor. > 3SD	10	In favour	64,5	
18.	Bevilaqua et al, 2008	Compar. Rest val.	12	Not in favour	64	
19.	Voight & Weider, 1991	Compar. Rest val.	16	In favour	59,5	
20.	Cavazzuti et al, 2010	Double threshold statistical detector	15	Not in favour	59,5	
21.	*On et al,	Compar. Rest val.	13	In favour	58	
22.	Cavazzuti et al, 2009	Double threshold statistical detector	8	Not in favour	54	
23.	Morrish & Woledge, 1997	Lag factor	49	Not in favour	47	
2. FATIGUE STUDIES						
1.	Callaghan et al, 2001	Fatigue ratios	10	In favour (no statist. signif.)	74,5	
2.	Väätäinen et al, 1991	MPF & ZCR	21	In favour	28	26,5

Legend: **Comp.algor.**= computer algorithm, **Compar. Rest val.** = comparison with rest value, **MPF**=median power frequency, **ZCR**=zero crossing rate, **mmit** =maximum muscle isometric test, **identif.**= identifier, **IN FAVOUR** = indicates results in favour of differences, **NOT IN FAVOUR** = indicates results not found any difference, * = studies that have also measured and EMG amplitude or ratio.

4.1.3 IMPLICATIONS OF THE MODIFICATION OF THE QUALITY SCALE

In regard to the use of the modification and use of the quality scale, while it is useful and important to think about the various aspects of the study design that could result in bias and affect the applicability of the study findings, the use of scores and overall scores is questionable (Elliot, 2007, University of Manchester – Cochrane Bone, personal communication).

Scales vary considerably in dimensions covered and complexity. In many cases scales include items for which there is little evidence that they are related to the internal validity of the trial – study. It's not an unusual phenomenon that different scales lead to discordant results (Jüni, Witschi, Bloch & Egger, 1999; Jüni, Altman & Egger, 2001). The quality scale employed for this review was not significantly complex, not very expanded and included items related to the internal validity of the assessed studies.

Although composite quality scales may provide a useful overall assessment when comparing populations of trial studies, such scales should generally not be used to identify trials of apparent low quality or high quality in a given systematic review. Rather, the relevant methodological aspects should be identified a priori and assessed individually (Jüni et al, 2001). Therefore it can be argued that the modified scale employed in the current review has only an indicative role rather a decisive one.

4. 1. 4 INTRARRATER RELIABILITY OF THE QUALITY RATING

In order to establish the intrarrater reliability of the rating of the reviewed studies a second experienced reviewer, was employed and marked nine out of 25 reviewed studies. The studies were selected randomly and the second reviewer was blind to the scores of the main reviewer. The scores of the two reviewers are presented in the table 4.3.

Table 4.3: Scores of reviewed studies by reviewers A & B

1. AMPLITUDE & RATIO STUDIES					Score	
	Study	Outc.measure	Patients	Results	A-Reviewer	B
1.	McClinton et al, 2007	Ratio	20	Not in favour	68,5	76
2.	Owings et al, 2002	Amplitude	20	In favour	65	56
3.	Tang et al, 2001	Ratio	10	In favour	65	62
4.	Mohr et al, 2003	% of max.	13	Not in favour	60	62
2. TIME ONSET & REFLEX RESPONSE STUDIES						
1.	Cowan et al, 2002	Comp.algor. > 3SD	37	In favour	75	75
2.	McClinton et al, 2007	Comp.algor. > 2SD	20	Not in favour	68,5	76
3.	Mellor & Hodges, 2005	MUAPS synchronis.	10	In favour	67,5	64
4.	Stensdotter et al, 2006	Autoregres. algor.	17	Not in favour	67	62,5
3. FATIGUE STUDIES						
1.	Väätäinen et al, 1991	MPF & ZCR	21	Not in favour	28	26,5

Outc. = outcome, **Comp.algor.**= computer algorithm, **MPF**=median power frequency, **ZCR**=zero crossing rate, **identif.**= identifier, **IN FAVOUR** = indicates results in favour of differences, **NOT IN FAVOUR** = indicates results not found any difference,

From the scores of the two reviewers presented in the table 2 it is relatively clear that there is a general agreement between the scores in the most of the cases. Only in the studies of Owings et al, (2002) the difference in rating is 9 points. In one study the score is identical and in the rest of the studies vary from 1,5 – 7,5 points.

4.2 Studies in relation to VMO-VL Amplitude & Ratio in PFPS patients

4. 2.1 STUDIES IN FAVOUR OF VMO-VL AMPLITUDE & RATIO MUSCLE IMBALANCE IN PFPS PATIENTS

A total number of eight studies were generally in favour of VMO – VL amplitude & ratio muscle imbalance in PFPS patients.

In a robust study by Owings and Grabiner (2002) the EMG activation **amplitude** of VMO and VL was monitored in 20 patients with patellofemoral pain and 14 control healthy subjects. They recorded maximum voluntary concentric (MVCon) and maximum voluntary eccentric (MVEcc) contractions initiated from two angles, 80° and 20° knee flexion angle to full extension (0°) respectively. In order to avoid any potential discomfort especially during eccentric contractions PFPS patients performed two instead of three repetitions and at a slower isokinetic velocity, 15°/sec. instead 60°/sec. than for healthy volunteers. They identified that patients with PFPS had different functional activation amplitude profiles from those of the control group. The normalised activation amplitude of the VMO and VL of the patellofemoral pain subjects was altered to the greatest extent during eccentric contractions with VMO demonstrating less activity than the VL. The authors concluded that in PFPS subjects there was consistent evidence of lateral tracking of the patella during eccentric effort.

Tang, et al, (2001) in another robust study, but with relative small sample size, evaluated the EMG activity of VMO and VL in Closed Kinetic Chain (CKC) and Open Kinetic Chain (OKC) in 10 PFPS patients and 10 control healthy subjects. For the OKC exercise subjects performed isokinetic eccentric & concentric contractions from 90° - 0° (flexion – extension) at an angular velocity of 120°/sec. In the CKC, quadriceps eccentric contractions were recorded during squatting, whereas concentric contractions were recorded during squatting to standing tasks. In both exercises, OKC & CKC, EMG data was obtained at 15° intervals (0, 15, 30, 45, 60, 75, 90 degrees). The normalised VMO – VL **ratio** of the PFPS group was significantly lower than those of healthy subjects, in isokinetic OKC exercise. However, they did not find any statistical difference in VMO – VL ratio between healthy subjects and patients with PFPS during CKC exercises. Maximum VMO:VL ratio was obtained at 60° of flexion in CKC, therefore they argued that selective VMO activation was optimised at this knee angle.

Cesarelli et al. (1999) in their investigation, aimed to study the quadriceps femoris muscle control strategy in 11 anterior knee pain (AKP) patients and 30 control subjects during isokinetic concentric exercises. They monitored concentric contraction at a velocity of 90°/sec. within a range of motion between 90° - 0° of knee flexion. The results, obtained from normalised grand ensemble average of the Linear Envelope EMG (LEEMG), pointed out significantly lower **activity** of the VM compared to the VL in AKP group. They concluded that the differences identified in the AKP patients were not only due to weakness of VM, as reported in

other studies, but also to a modification of the neural recruitment strategy of the quadriceps muscle.

In a moderate quality study with relatively small sample size Santos et al, (2008) investigated the recruitment patterns of the vasti muscles with use of the normalised VMO:vastus lateralis longus (VLL) and VMO:vastus lateralis oblique (VLO) **ratios**. Ten PFPS patients and ten healthy controls were assessed during ten open kinetic chain (OKC) and functional exercises. Specifically they tested the following activities: isokinetic extension from 60° to 0° at 30°/sec, squat from 0° to 45°, step-up & down with knee flexion 45° and 75°, sit to stand, hopping, plantar and dorsi-flexion from standing. The overall between group comparison revealed a significance reduce in the VMO:VLO ratio in patients in comparison with the healthy but no significant differences in the VMO:VLL ratios. Also, in concentric isokinetic extension from 60° to 0° at 30°/sec and in step down with knee flexion 75° (eccentric contraction) found significant decrease of the VMO in relation to VLO among the PFPS patients. In the rest of the tested tasks no difference was apparent. The authors concluded that specifically the VLO has an antagonistic function relatively to VMO and eventually this muscle is responsible for the quadriceps muscle imbalance.

In a less robust study (only nine patients and nine healthy subjects) of Boucher et al, (1992) isometric maximum knee extension in 90°, 30° and 15° of knee flexion was tested in 9 healthy and 9 patients with PFPS. Patients had an above normal average Q angle (mean 21°) whilst the control subjects had normal values (mean

8. 25°). In further support of this trend, five individual patients exhibited Q angle values higher than 22°. The normalised VMO:VL **ratios** indicated no significant differences between groups for each of the measured angles. Although not significant, an interesting tendency was observed towards a decreased VMO:VL ratio across all angles of knee flexion in PFPS subjects.

Souza & Gross (1991) in a similarly small scale study (9 patients, 7 healthy subjects) measured the VMO:VL integrated EMG (IEMG) **ratios** under isotonic and isometric quadriceps femoris muscle contractions. Comparison of normalised data indicated no differences in IEMG ratio between the groups. However, when comparing non-normalised data they found that both lower limbs of patients (affected & non affected) had significantly lower EMG ratios than that of the controls. Despite these findings, non-normalised EMG data should be interpreted with extreme caution. Kasman (1998) provides a compelling argument, stating, that if the relative activities of the VMO & VL are aberrant in the same way for a normalising reference contraction as they are for a test activity, true differences between patients with PFPS and healthy subjects could be obscured. This may help to explain the interesting observation of Stensdotter et al. (2006) who stated that PFP subjects activated VMO less relative to VL during maximal voluntary contractions (MVC), while control subjects activated VMO more than VL. This resulted in opposite results for ratios based on values normalised to the MVC compared to corresponding non-normalised MVC values respectively.

Taşkiran et al, (1998) in a relatively poor study (non normalised data, non randomised exercise conditions, poor description of inclusion/exclusion criteria for PFP group) monitored the non-normalised VMO-VL IEMG **ratios** in three different groups. A total sample of 27 subjects was studied, one control group (A) with nine healthy subjects, one group (B) with ten patients with PFPS but without patellar instability and one group (C) with eight patients with patellar instability. They assessed isometric quadriceps contraction at 0°, 15°, 30° and 45° of flexion in CKC and also monitored the congruence angle (CA), patellar tilt angle (PTA) and sulcus angle (SA) by using computer tomography (CT). Group B had a VMO-VL ratio slightly less than 1 at 0°, 15° and 30° flexion. Group C showed a ratio of less than 1 at the same degrees of flexion. The only statistical significant difference occurred at 15° between group A (healthy) and group C (patellar instability). Another interesting aspect of the results was that they found an increased congruence angle (CA) together with the lowest ratio in group C (patellar instability) and increased patellar tilt angle (PTA) in both patients groups (B= PFPS patients without patellar instability and C= PFPS patients with patellar instability) at 0° and 15° associated with VMO-VL ratios below 1, although these correlations did not reach statistical significance.

In order to examine the effects of chronic knee pain on neural control of the quadriceps, motor evoked potentials (MEP) in response to trans-cranial magnetic stimulation (TMS) of the motor cortex, maximal M responses, patellar tendon responses and EMG activity were measured in 13 PFPS patients and 13 healthy control subjects in a study by On et al, (2004). The VMO-VL EMG **activity** was

investigated using maximal isometric knee extension contraction in supine position. In order to simulate non-weight bearing closed kinetic chain (CKC) conditions they used a special designed jig which was placed under the subject's feet. They employed this specific testing position because in previous study by the same research team (study of Taskiran et al, 1998, described above), VMO & VL demonstrated the greatest EMG activity in comparison to the 30° and 45° of knee flexion. Both VMO & VL demonstrated significantly lower EMG activity in the patients group compared to the healthy subjects ($p < 0.0$) and the decreased EMG activity have been more remarkable in the VMO relative to VL, but they did not state whether this difference was also significant. In regard to the EMG activity recordings of the VMO-VL, the authors did not state the sampling frequency and they did not specify whether they normalised the EMG data and therefore the results of this study are considered as low quality. In the general conclusions of the study the authors reported that, the electrophysiological differences observed between PFPS patient and control group support the pronounced inhibition of the VMO relative to VL and underpin the differential corticomotor control of the quadriceps muscle of a chronically painful knee.

One of the least robust studies (only eight patients, only visual analysis of the EMG activity) but interesting methodologically, undertaken by Mariani and Carouso (1979) examined five controls and eight patients with patellar subluxation. They monitored VM & VL EMG activity prior to and six to twelve months after a surgical procedure. The qualitative, visual analysis of the raw EMG recordings identified in patients a sharp fall in the non normalised **peak EMG** of the VM in comparison with

the VL throughout extension range from 90° - 60° - 30° - 0° that was especially evident in 30° and 0°. A similar picture was identified in the patients' unaffected limb. After the operation to correct the malalignment of the extensor mechanism, the VM activity was almost fully recovered. The authors hypothesised that functional insufficiency of VM was an aggravating factor contributing to static alterations such as an increased Q angle high patella (alta) and thus could be important feature in the pathogenesis of PFPS.

4.2.2 STUDIES NOT IN FAVOUR OF VMO-VL AMPLITUDE & RATIO MUSCLE IMBALANCE IN PFPS PATIENTS

A total number of eight studies were generally not in favour of VMO – VL amplitude & ratio muscle imbalance in PFPS patients.

Powers (2000) in his robust study assessed the influence of the normalised VL-VMO and VL-vastus medialis longus (VML) **ratios** on the patellar kinematics using fine wire electrodes and measuring the resisted knee extension in 0, 9, 18, 27, 36 and 45 degrees of flexion in supine position. The patellar tracking measures were acquired from MRI imaging in supine position and the same prone position was used for the EMG measurements. The EMG data showed no difference in the VL-VMO and VL-VML ratio between the PFPS and the healthy control subjects. The reported mean VL-VMO ratio across all knee flexion angles was 1.85 for the PFPS

patients and 1.17 for the healthy subjects, the VL-VML ratio was 0.78 and 0.94 respectively. The author concluded that there was no group effect or interaction. Although the overall quality of the study is good, a controversial issue is raised. It is interesting to note that a visual inspection of the data in a figure presenting VL-VMO mean ratios across all knee flexion angles shows a clear tendency: at 0 and 10 degrees of knee flexion the VL-VMO mean ratio had a small difference between the two groups, but in the rest of the knee angles (18, 27, 36 and 45 degrees) it is obvious that the difference between groups gradually increases as the knee flexion angle advances. A visual approximate calculation of the figure with the data of the VL-VMO ratio in 45° of knee flexion, reveals that VL-VMO ratio of the PFPS patients is 2.30 and of the healthy controls is 1.10 (difference between ratios= 1.20). The author reported a non-significant mean ratio between groups, PFPS patients VL:VMO ratio was 1.85 and the healthy VL:VMO ratio was 1.17 (difference between ratios= 0.68). This allows the reader to assume that the approximately 1.20 difference between the ratios of patients and healthy in 45° knee flexion would probably be statistically significant if was tested. This means that the VL activity was obviously higher than the VMO activity in patients in comparison with healthy controls in 45° of knee flexion and an interesting interaction exists between VL-VMO EMG ratio and knee flexion angle (patellar tilt and displacement) and the muscular action of the VM and VL. The study includes correlations only between the VL:VML EMG ratios and patellar tracking, and does not investigate the correlation between the VL:VMO EMG ratios. It is well known that the fiber angular arrangement of the VMO (angular fiber orientation of the VMO is 50°-55° and of the VML is 15-18° off the long axis of the femur) provides an

ideal stabilising component and therefore the VMO is considered as primary active medial stabiliser of the patella (Lieb & Perry, 1968; Lieb & Perry, 1971; Bose et al, 1980; Reider et al, 1981; Williams & Warwick, 1995, pp.637-640; Nozic et al, 1997; Raimondo et al, 1998). Nevertheless, the author stated that the increased activity of the entire VM seems to be associated with the abnormal patellar tracking.

In a recent robust study, Ott et al, (2011) compared the normalised activation **amplitude** of VMO, VL and gluteus medius (GM) in 20 PFPS patients and 20 healthy controls following an aerobic exercise programme. The testing procedure consisted of a single leg anterior reaching task, a dynamic exercise often used in musculoskeletal rehabilitation. Following the baseline measurement the aerobic exercise programme included 20 minutes treadmill walking at a self-selected speed with a minimum pace no less than 3.0 mph. The level of the fatigue was measured throughout the test every minute using the ratings of perceived exertion (RPE) and the level of pain was monitored on a VAS. During the first 15 minutes the treadmill incline was increased by 1%/min and in the last 5 minutes the participants adjusted the treadmill incline 1% either up or down in order to maintain a RPE of 15-17. The single leg anterior reaching task was repeated after the aerobic exercise programme. The overall analysis showed that the VMO activation was higher than VL in both groups but between the groups there was no difference before or after the aerobic exercises. However, the analysis of the PFPS group according to pain (high pain PFPS group and low pain PFPS group) revealed that the patients who experienced higher pain had a significant

reduction of the VMO activity by 25% and in the VL by 12%. The authors suggested that this was probably a compensatory muscle strategy in order to reduce the PF joint load and pain experienced.

The EMG activity **ratio** of the VMO-vastus lateralis longus(VLL) was recorded during level and 5% inclination treadmill walking in 12 PFPS patient and 15 healthy individuals (Santos et al, 2007). Although the authors reported that it was the first study comparing VMO-VLL ratio, they did not explain the reasons and criteria for the choice of the VLL instead of VL. Additionally, when they described the electrode positioning of the VLL they referred to the anatomical study of Weinstabl in 1999 in order to argue that the electrode's position of the VLL would be similar to the electrode positioning for the VL muscle used by several other studies. The normalised data revealed no significant difference in the VMO-VLL ratio between the two groups in both examined conditions. Despite the absence of significant difference, a tendency was observed with healthy controls having higher VMO-VLL ratio relative to PFPS patients in both level and inclined gait, and this was explained by the authors as a possible reduction of the medial stabilising force of the patella

In a very robust study by Sheehy et al, (1998) measured EMG activity VMO-VL **ratios** during ascending and descending stairs. They compared 15 asymptomatic subjects and 13 subjects with PFPS. Normalisation of the EMG was done relative to a maximum voluntary isometric contraction with the knee fully extended. The reliability correlation coefficients for the VMO & VL EMG data were at an

acceptable level, ranging from .83 to .89 for the isometric, concentric and eccentric contractions. The results indicated no differences between groups in terms of VMO-VL ratio. When data from both groups were combined, it was found that the VMO-VL ratio was smaller in the eccentric phase of descent compared with the concentric phase of the ascent. These findings suggested no differences between healthy individuals and patients, but that differences may exist in VMO-VL ratios between concentric and eccentric contractions. The authors recommended that further research is needed to determine if VMO and VL muscle imbalance contributes to patellofemoral dysfunction.

Liebensteiner et al, (2008) used a force-measuring leg press system (non weight-bearing closed kinetic chain position) in order to evaluate the influence of the maximum eccentric leg press exercise on the EMG amplitude of the VMO-VL, biceps femoris (BF), semitendinosus (ST), peroneus longus (PL) and gastrocnemius medialis (GM), and tibiofemoral alignment in the frontal plane in 19 PFPS patients and 19 healthy subjects. The examined task included maximum eccentric knee contraction from 50° -95° of flexion under two conditions a) stable footplate and, b) unstable footplate. The data were normalised to the MVIC on leg press in 70° of knee flexion. The authors recorded reduced knee maximum eccentric force in PFPS patients in comparison to healthy subjects in both stable and unstable conditions. A slight tibiofemoral varus alignment was found in both groups but with no significant difference and a greater but not significant perturbation effect was evident in patients in comparison to the healthy controls. In the VMO-VL normalised EMG amplitude was found no statistical difference between vasti

muscle and between groups. In contrast, they found significant lower activity in the hamstrings of the patients in comparison with the healthy controls but is unclear whether this was a compensatory neuromuscular pattern or a contributing risk factor for the PFPS.

Similarly in another robust study published recently McClinton et al, (2007) found that activation **magnitude** was similar between vasti muscles in individuals with and without PFPS. They measured 20 subjects with PFPS and 20 control subjects in a step-up/step-down task at five different step heights (8, 14, 20, 26, 32 cm). The stepping rate was paced by a metronome, and three dimensional kinematic data of the tested knee was recorded using an 8-camera motion capture system. The EMG signal was normalised to its instantaneous peak activity during stepping tasks. Although subjects with PFPS displayed greater knee flexion angles at a foot step contact than healthy subjects, the results did not reveal a between group difference in activation magnitude. Additionally step height during stair ascent did not appear to alter VMO and VL activation magnitude in individuals with patellofemoral pain, despite an increase in reported knee pain at the higher step heights. In conclusion the authors reported that inhibited or delayed VMO activation relative to VL among patients with PFPS was not supported by the results of the study.

Stensdotter et al, (2006) in a robust study investigated VMO & VL **ratios** under postural responses to unpredictable perturbations in 17 women with PFPS and 17 matched healthy controls. They monitored three dimensional kinematics of whole

body and normalised EMG of VMO & VL in postural responses to unpredictable support surface translations (anterior & posterior). The results showed that VMO and vastus medialis longus (VML) were generally more active than VL, in patients with PFPS, but the activation strategy of the VMO in relation to VL in PFPS patients was unclear. Furthermore, correlation with kinematic data suggested that the presence and nature of altered quadriceps activity in patients with patellofemoral pain may be task specific and part of an adaptive strategy in an attempt to compensate for muscle weakness and/or increase the safety margins for balance. This strategy might be part of an adapted response in an attempt to decrease patellofemoral joint loading and this learned response appears to be maintained even when the pain is no longer present.

In a robust study, Powers et al, (1996) assessed vastus muscle activity with fine wire electrodes during various functional activities including, free speed and fast level walking, ascending and descending stairs and ramps. They recruited 26 patients with patellofemoral pain and 19 healthy control subjects. From normalised data it was evident that all the vasti muscles demonstrated decreased electromyographic **activity** for free speed and fast level walking and ramp ascending and descending in PFPS. The authors suggested that the decreased activity was suggestive of a quadriceps femoris muscle avoidance pattern, which is similar to the response seen in subjects with anterior cruciate ligament tears. They argued that subjects with weak quadriceps femoris muscle or a painful knee avoid loading during knee flexion, as it is the point in the gait cycle where the muscular demands and knee joint reaction forces are the greatest. Therefore

these results do not support the hypothesis that differences between the VM and VL activity are associated with PFPS.

Mohr and colleagues (2003) in an attempt to address the muscle imbalance issue recruited 13 patients with patellofemoral pain associated with lateral subluxation and 11 healthy subjects. They compared muscle **activity** and total time of the different phases of gait, monitored with fine wire electrodes, during functional activities including walking and ascending and descending stairs. Normalisation of the EMG signal was achieved with the use of the peak EMG signal during a 0,5 sec of a 5 sec. maximal manual muscle test. They reported that VMO & VL had similar activity patterns during all conditions. Although the overall time needed to complete a gait cycle was the same between healthy volunteers and patients, the PFPS subjects spent more time than the healthy subjects during the most challenging phases of the gait cycle. Therefore these data suggest a generalised quadriceps weakness in PFPS patients, rather than the prevailing theory of quadriceps muscle imbalance as an etiology of patellofemoral pain.

MacIntyre and Robertson (1992) in a moderately robust study measured Linear Envelope EMG (LEEMG) **activity** during treadmill running (two different speeds) in eight runners with PFPS and 12 healthy runners. The grand ensemble EMG patterns of each subject were normalised by dividing by the maximum EMG per cycle. The authors concluded that any changes in the running pattern of the subjects with patellofemoral pain could not be detected by changes in the EMG patterns. Despite this it may be important to note here, that all the patients had

malalignment features such as rear and fore foot alignment $\geq 5^\circ$ and overpronation.

Grabiner and colleagues (1992) in a less robust investigation attempted to determine if patellofemoral pain was associated with abnormal excitation of VMO and VL during non constant isometric knee extension force conditions. Normalised EMG **activity** data for a control group of 15 healthy subjects was compared to eight patients with PFPS. The exercise conditions consisted of maximal isometric voluntary effort of knee extension force elicited at 20° of flexion in OKC for three tasks including: a) slow rate of extension force development to maximum knee extension force, b) maintaining a constant submaximal knee extension force and c) fast rate of knee extension force. Exercise conditions were not randomised. According to the results, PFPS patients presented significantly lower VMO and VL excitation levels than the control group during the fast rate force condition. The patients demonstrated similar excitation patterns of VMO & VL during the two exercise conditions (slow & constant task). The authors suggested that the results probably reflected disuse atrophy of high threshold motor units of both VMO & VL and/or decreased ability to recruit these motor units in PFPS patients. They argued additionally, that muscular power and underlying muscle excitation deficits might be more indicative of functional state in comparison with the traditionally measured strength values.

In 1986 Møller et al, in a relatively poor study (no normalisation of the EMG data, no randomising and with poor patient population selection criteria) evaluated three groups of subjects. Group A consisted of 14 healthy subjects, group B 11 patients with patellar instability and group C 17 patients classified as idiopathic chondromalacia patellae. They measured EMG **amplitude** of maximal isometric contraction of the VMO & VL at 90°, 60°, 45°, 30°, 15° and 0° of flexion without randomising the exercise conditions and normalising the EMG data. The results revealed a similar decreased muscular activity pattern in both patient groups (II & III) as compared with the asymptomatic knees (group I), and none of the groups revealed differences in the activity of VL and VMO suggesting muscular imbalance.

4.2. Studies in relation to VMO – VL Onset Time & Reflex Response in PFPS

Patients

4.2.1 STUDIES IN FAVOUR OF VMO-VL ONSET TIME & REFLEX RESPONSE MUSCLE

IMBALANCE IN PFPS PATIENTS

A total number of six studies were generally in favour of VMO – VL onset time muscle imbalance in PFPS patients.

In the most robust study, by Cowan et al, (2001) the issue of **onset time** was examined under functional conditions. They assessed the time of onset of VMO and VL in 33 patients with PFPS and 33 healthy subjects during step up (concentric contraction) and step down (eccentric contraction) activities. The EMG onset time was determined by using a computer algorithm to identify the point at which the EMG signal deviated by more than 3 standard deviations (SDs) for a minimum of 25 ms, above the baseline. This was also verified visually. In a PFPS population, the EMG onset time of the VL occurred before that of the VMO in both the step up and step down phases of the stair stepping task. In contrast, no such differences occurred in the onset of EMG activity of the VMO & VL in either phase of the task for the healthy subjects. Additionally, they found that the reliability of the determination of EMG onset timing of the concentric and eccentric phases of stair stepping was excellent (ICC= .91 & .96 respectively). Finally the authors concluded that the findings supported the hypothesised relationship changes in the timing of activity of the vasti muscles between healthy subjects and patients with PFPS.

Van Tiggelen et al, (2009) in a high quality prospective study investigated the role of the VMO delayed **onset time** as a potential intrinsic factor for the development of the PFPS. The onset time of the VMO-VL was recorded during the functional task of rocking back on the heels (dorsi-flexion of the foot) in 79 healthy army recruits before and after 6-weeks basic military training (BMT). The onset time was detected with the use of computer algorithm 3SDs method. 26 participants (32%) out of the 79 developed PFPS, and the VMO of healthy subjects was activated 4.86 ms earlier than VL before the BMT, and 1.69 ms earlier after the BMT. The

participants who developed PFPS had a 1.67 ms VMO delayed onset before the BMT and this delayed was significantly increased after the BMT (17.73 ms VMO delayed onset). The baseline differences (before the BMT) of the VMO-VL activation between the healthy and the participants who developed PFPS were significant. Additionally, after the BMT the VMO-VL onset time differences between the healthy and the PFPS were significant. The analysis of the relative risk ratio of developing PFPS for subjects with 15ms delayed VMO onset, revealed a relative low value (R^2 1.46) and the ROC (receiver operating characteristics) value was fair (0.68). The authors concluded that, although the nature of the PFPS is multifactorial and others variables besides the delayed onset should be included in order to construct a more robust predictive model, the VMO delayed onset as a single risk factor has predictive value for the development of the PFPS.

Another very robust study by Cowan et al, (2002b) investigated the recruitment of the VMO & VL during voluntary tasks (toes rise and heel rock) that challenged the stability of the knee. Their objective was to evaluate whether there was a change in the coordination of the postural response by the central nervous system in subjects with PFPS. EMG onset time was determined using the same technique described in their earlier study (Cowan et al, 2001). The results obtained from 37 PFPS patients and 37 asymptomatic sex-matched controls demonstrated a statistical significant delayed **onset time** of the VMO in comparison with the VL for the patients with patellofemoral pain. Additionally they observed a wide variation in the EMG onset time for both VMO & VL in both PFPS and control groups. In conclusion the authors supported the difference in motor control and the

hypothesised relation between changes in the timing of activity of the vastii muscles and patients with PFPS.

In the study of Ng et al, (2011) the VMO-VL temporal recruitment was compared during a) voluntary tasks such as, semi-squat, rise on the toes (foot plantar flexion), rock on the heels (foot dorsi-flexion) and b) involuntary postural control actions caused by perturbation tests under three conditions – normal standing, standing on the forefoot and standing on the heels. The perturbations were performed by using a pendulum swing with a 3 kg ball released from an angle 60° to the vertical, and hitting with 30J the back of subject's symptomatic knee in order to elicit an involuntary contraction of the quadriceps. The **onset time** was detected by using a computer algorithm 3SDs method, and one group of 23 PFPS patients was monitored. No control group was employed. Significant differences were found in the tip-toeing voluntary task with the VMO activation 58.9ms delayed in comparison to VL, and in the heel standing voluntary task with the VMO activation delayed 57.7ms relatively to VL. In contrast, during the perturbation tasks of toe-standing and heel standing the VMO was activated earlier (5.3ms and 7.8ms respectively) in comparison to VL. The findings of this study indicate an interesting twofold temporal activation pattern of the VMO-VL, during voluntary tasks when the VMO onset time is delayed significantly in comparison to VL. These findings concur with the study of Cowan et al, (2002) described previously. A reverse activation pattern is observed in the perturbation tasks. The authors in an attempt to interpret these results assumed that the delayed onset time of the VMO during the voluntary tasks was possibly provoked by suppression of the excitatory projection of the quadriceps motor neurons in these patients. During the

perturbations tasks the reverse pattern of the VMO activation was probably a result of the eventual stimulation of the knee mechanoreceptors due to the ball impact and vibration. The study of Ng et al, did not involve a comparison between patients and healthy subjects and consequently these results should be interpreted with caution.

In a similar robust study, Witvrouw et al, (1996) compared **reflex response time** after a patellar tap in a control group of 80 healthy adults and a group of 19 patients with PFPS. This group also found that the reflex response of the VMO was significantly shorter than that of the VL in healthy subjects. Furthermore, in the patient group a significant earlier firing was observed from the VL in comparison with VMO. Like Voight and Wieder (1991) they suggested that the results indicated an alteration in neuromuscular control of the two vasti muscles during a patellar tendon tap in patients.

Mellor and Hodges (2005) in another robust investigation evaluated the **synchronisation** of the motor unit action potentials (**MUAPs**) of the VMO & VL with fine wire electrodes at 30° of flexion in OKC. The authors argued that changes in motor unit firing may provide more definitive evidence in comparison with the conflicting data derived from onset time studies. They assessed ten healthy subjects and ten patients with anterior knee pain (AKP) and compared data to previous normative data. They found 80% of patients had values less than control subjects and 20% were within normal limits. The authors concluded that their results provide new evidence that motor unit synchronisation is modified in the presence

of pain and provided evidence for motor control dysfunction in AKP. Additionally they stated that, motor control dysfunction is a contributing factor in this condition and has implications for selection of the appropriate rehabilitation strategies. A plausible question about the motor unit action potentials approach recorded with fine wire electrodes is whether the individual motor unit activity can secure a total representation of the muscle function tested as a whole.

Cesarelli et al. (1999) in their study, mentioned previously in relation to VMO – VL activity (p. 52), they also monitored the quadriceps femoris muscle onset time in 11 anterior knee pain (AKP) patients and 30 control subjects during isokinetic concentric exercises. The results revealed a significant delay in the **onset time** of the VM compared to the VL in AKP group, and therefore stated that the delayed onset time of the VM was due to a modification of the quadriceps neural recruitment strategy and not only caused by VM weakness.

Reflex response time of the VMO and VL was evaluated in 16 PFPS subjects and 41 healthy subjects by Voight & Wieder (1991) by using the patellar tendon reflex. The results indicated that in normal subjects VMO fired significantly faster than VL, but in a PFPS patient group a reversal of the normal muscular firing order between the VMO and VL was evident. Like Cesarelli et al, (1999) the authors concluded that patients with PFPS may be demonstrating a neurophysiologic motor control imbalance that may account for or contribute to their anterior knee pain.

In a moderate quality study by On et al, (2004), the VMO-VL **reflex responses** were recorded in 13 PFPS patients and 13 healthy control subjects. The VMO-VL tendon responses were significantly decreased in PFPS patients in comparison to healthy subjects, and these changes have been more remarkable in the VMO relative to VL, but they did not stated whether this difference was also significant. The authors concluded the reflex responses findings and also the overall electrophysiological results indicate that chronic knee pain modifies central motor control of an adjacent muscle.

3.3.2 STUDIES NOT IN FAVOUR OF VMO-VL TIME ONSET & REFLEX RESPONSE MUSCLE IMBALANCE IN PFPS PATIENTS

A total number of eight studies were generally not in favour of VMO – VL onset time muscle imbalance in PFPS patients.

Pal et al (2011) in a high quality study tested the potential correlation between the patellar tracking and VM activation delay in different subgroups of PFPS patients. The open configuration PFPS patients MRI measures of the patellar tracking acquired in the weight bearing position were classified (according to patellar tilt and bisect offset) into normal and abnormal tracking groups. The VMO-VL EMG activity was measured during walking and jogging in 40 PFPS patients and 15 healthy controls by using two **onset time** detection methods, the 3 SD computer

algorithm or the 2% of the peak activation of the vasti. VM activation delays relatively to VL were observed in both groups with no significant difference during walking or jogging. Additionally, there was no correlation between the VM activation delay and measures of patellar tracking in healthy or patient group as whole. However, significant correlations between the VM delayed onset and PFPS patients subgroup with both abnormal tilt and bisect offset were observed. According to the authors, the results of the study underline the importance of appropriate classification of the PFPS patients prior of a clinical intervention.

The **onset time** of the VMO-VL in quadriceps maximal isometric contraction with the knee fully extended was investigated by Patil et al, (2011). They measured 20 patients with PFPS and 17 healthy control subjects and they used the 3 SDs computer algorithm method for the detection of the onset time of muscle activation. In the PFPS group the VMO onset time was delayed 37.3ms (SD 62.0ms) in comparison to VL, and the picture was similar in the control group with the VMO onset time delayed 59ms (SD 60.3ms). The mean difference between the groups was 21.7ms (SD -16.9 to 60.4ms) and was not statistically significant. The selection of the maximum contraction, the non-functional static conditions of execution of the contraction (open kinetic chain) and the marked variability of the onset time differences was mentioned by the authors as limitations of the study.

McClinton et al, (2007) and Sheehy et al, (1998) in previously mentioned studies both looked at VMO & VL **onset time**. **Neither studies found a** difference between the onset times associated with PFPS during functional tasks. Similarly, Powers et al,

(1996), reported no difference in VMO – VL **onset time** measured during free speed and fast level walking and ramp ascending – descending in patients with patellofemoral pain and asymptomatic individuals. In contrast, Stensdotter et al, (2006), reported a VMO earlier **onset time** relative to VL in 17 patients with PFPS, controlled under postural responses to unpredictable perturbations.

Brindle and associates (2003) examined EMG firing patterns of VMO, VL and gluteus medius (GM) between 16 patients with AKP and 12 healthy subjects while ascending & descending stairs. The **onset time** was calculated at the point where EMG activity exceeded five SD above the resting mean of the resting EMG. Consistent with the previous studies, subjects in the AKP group demonstrated no differences in the VMO onset relative to VL onset compared to the control group. Nevertheless, one probably interesting aspect of the results was that gluteus medius demonstrated delayed onset and shorter durations for stair ascent and shorter duration during descent. The action of gluteus medius on the lower extremity to control frontal plane moments at the hip can affect forces at the knee. Whilst the role of gluteus medius is outside the scope of this study it is interesting to note that the authors supported a hypothesis that the gluteus medius plays a role in AKP because its action could produce forces indirectly across the knee and stated that was likely the gluteus medius was part of a compensation strategy in AKP patients.

Karst & Willet (1995) investigated the **onset time** of VMO & VL during reflex knee extension elicited by a patellar tendon tap and voluntary muscle activity during

active knee extension in non-weight bearing (NWB) and weight-bearing (WB) conditions. They measured 15 PFPS patients and 12 asymptomatic subjects. Three patients reported onset of PFPS following knee surgery (meniscal or ligamentous injury) and two related to direct patellar trauma. A computer algorithm > 1 SD of the resting baseline amplitude was used to estimate the timed onset. They reported no significant differences (less than 0,25 ms for reflex onset and less than 4 ms for active knee extension under both WB & NWB conditions) between patients and healthy subjects. They concluded that the results of the study did not support the hypothesis that altered timing of VMO & VL activity plays role in initiating or perpetuating patellofemoral pain.

In another study by Owings and Grabiner (2002), mentioned earlier in relation to VMO & VL amplitude (p. 51), although they reported that VMO demonstrated decreased amplitude activity in comparison to VL, they identified no delayed **onset time** of the VMO relatively to VL between patients and healthy subjects.

The onset time of the VMO-VL was investigated by Cavazzuti et al, (2010) in 15 patients with PFPS and 20 healthy controls during the following tasks: sit to stand, stand to sit, squat, step-up & step-down. For the detection of the onset time a double-threshold statistical detector was employed which accuracy has been reported to be higher than other methods used in the literature. The results revealed a balanced activation onset of the VMO & VL between PFPS patients and healthy controls in all tested conditions. Although the onset time detection method employed by the study was of high quality, the standardisation of the

tested exercise protocol was not at the same high level. No randomisation of the tasks was employed and the pace of execution was self-selected by patients without any type of monitoring or effort for standardisation.

The **reflex response** time (RRT) was analysed by measuring from time zero to the peak electrical response of the VMO and vastus lateralis oblique (VLO) & vastus lateralis longus (VLL) in 12 PFPS patients and 12 healthy controls in the study of Bevilaqua-Grossi et al (2008). The analysis revealed that the VMO reflex response time was lower in comparison to the VLL & VLO for both groups but no significant difference was evident. Direct comparison of these results with other reflex response studies is limited because in this study the VL muscle was analysed as two distinct parts (longus & oblique). Additionally, the recruited PFPS patients had different pain status in comparison to other studies as they had to be completely pain-free at the time of the study and during the last two months prior to the study.

The **onset time** of the VMO-VL was measured in eight patients with PFPS and in 17 healthy controls in another study by Cavazzuti et al, (2009) with relatively low quality. The onset time detection method and the exercise protocol were similar as the one used by the same research team in the study of 2010 described above. The VMO was activated 0.001ms earlier than the VL in all tested conditions for both groups and no statistical significance was found. The inclusion-exclusion criteria and the examined test protocol of the study were poorly described.

Finally, in a not very robust study Morrish and Woledge (1997) monitored the **synchronisation** of VMO and the postero-lateral fibres of VL (VLO) during force development of a maximal isometric contraction at 20° of flexion. They recruited 49 patients with PFPS and 20 healthy subjects. They calculated a lag factor by plotting the EMG signal against the force record for the first 80% of the tension rise monitored by isokinetic dynamometer. They reported that the force rise was slower in the patients than the healthy subjects but the VMO and VLO activity remained approximately synchronous, and suggested therefore that they have a reciprocal action in controlling patellar position.

4.3. Studies in relation to VMO-VL Fatigue Characteristics and Muscle Imbalance in PFPS patients

4.4.1 STUDIES IN FAVOUR OF VMO-VL FATIGUE CHARACTERISTICS MUSCLE IMBALANCE IN PFPS PATIENTS

A total number of two studies were generally in favour of VMO – VL fatigue characteristics in relation to muscle imbalance in PFPS patients.

Callaghan and colleagues (2001) has been the only group to compare **fatigue** ratios between VM and VL in healthy and patient populations. They measured 10 patients with PFPS and 10 healthy volunteers during isokinetic closed kinetic chain

isometric contraction at 45° of knee flexion. A twitch interpolation technique was employed for the 100% MVIC measurement, and the fatigue assessment was monitored during a 60 sec contraction at a submaximal level of 60% of the MVIC. The raw EMG signal was subjected to Fast Fourier Transformation in order for the power density spectrum to be determined and the median frequency (MF) to be extracted. The median frequency (MF) was then normalised against initial MF and a linear regression was constructed from which was derived a slope indicating rate of change during fatiguing contraction. The normalised median frequency (MF) slope was used to express the fatigue rate. The results revealed no significant differences between the two groups nor between the muscles, but the linear regression slope for the VMO and VL were different between patients and control subjects. Additionally there was much larger variability in median frequency (MF) values for the patient group. In patients with PFPS the fatigue resistance of the VMO (-0.140) was lower than that of the VL (-0.079). Although, the results did not reach statistical significance and the sample size was relative small, they may indicate unusual features in the fatigue indices of the VMO and VL in PFPS. The authors have since noted however the difficulties in obtaining reliable results in the estimation of quadriceps muscle fatigue. Until such problems are overcome it may not be possible to explain the phenomenon of relative fatigue further. Therefore further research was recommended in order to determine the existence of any possible relationships.

In an earlier, similar but considerably poorer study (no normalising, sampling frequency not reported) Väättäinen et al, (1991) assessed 21 patients with

chondromalacia patella and 18 healthy individuals. They monitored the changes in **median power frequency** (MPF) and the zero crossing rate (ZCR) during dynamic isokinetic exercise without to normalise the EMG data and without stating the sampling frequency. Additionally the provided information about the characteristics of the patient population was insufficiently described. Nevertheless, the changes in EMG parameters (median power frequency and zero crossing rate) on VM muscle between affected side and control group were highly significant. In contrary the changes in EMG parameters were not significant on the VL muscle between patients with chondromalacia patella and healthy individuals. Subsequently they stated that the fatigue phenomenon appeared in the VM muscle faster than on healthy side or in the control group in patients.

3.4.2 STUDIES NOT IN FAVOUR OF VMO-VL FATIGUE CHARACTERISTICS MUSCLE IMBALANCE IN PFPS PATIENTS

Despite having performed an extensive literature search this has not revealed any available published studies not in favour of VMO-VL fatigue characteristics muscle imbalance in PFPS patients.

4.4 Does the VMO – VL Muscle Imbalance Exist?

Controversy exists in the reviewed literature as to the normal relationship between the amplitude-ratio & onset time of EMG activity of the VMO & VL and whether

this is different in a population with PFPS. From the reviewed studies 21 were in favour and 25 were not in favour of muscle imbalance when different parameters were taken into account. In regard the amplitude & ratio studies, from the total 21 reviewed, 9 studies were in favour and 12 studies were not in favour. In terms of onset time & reflex responses 10 studies found delayed onset of the VMO in comparison to VL and 13 studies reported synchronous activation of the vasti. Finally in regard to fatigue, the two available studies were both in favour of VMO muscle imbalance. In terms of quantity it could be argued that there is subtle tendency that is not in favour of VMO-VL muscle imbalance. An overall analysis though reveals that, there is almost a balanced difference between studies in favour and studies not in favour of VMO vs VL muscle imbalance. An initial interpretation of this balanced difference could possibly be that, in a certain percentage of patellofemoral patients the muscle imbalance is indeed apparent and in another part of patients with this pathology muscle imbalance does not exist. Considering the multi-factorial etiology of patellofemoral pain syndrome, might be possible that muscle imbalance is evident in some cases of patients with PFPS and in other cases of PFPS is not. Another possible explanation, although the reasons for the discrepancies are not very clear, may be related to a certain extent to the following factors:

4.4.1 METHODOLOGY

Methodology used to determine electromyographic onset time might be an important confounding factor (Cowan et al, 2001). Studies used different

methodologies to determine the onset time of EMG activity (Powers et al, 1995; Cesarelli et al, 1999; Witvrouw et al, 1996; Cowan et al, 2001; Mellor & Hodges 2005; Cavazzuti et al, 2010). Also studies did not express such onset time directly, instead as the time at which the amplitude exceeded 5% of a maximal voluntary quadriceps contraction without visual inspection for artefacts (Powers et al. 1995) or in indirect manner, as a relation to force changes, subsequently they failed to define the technique used (Morrish & Woledge, 1997). Such approaches are limited because the use of a maximal isometric contraction is problematic in PFPS subjects due to the potential presence of pain or fear of experiencing pain, therefore in both cases may affect the patients' ability to perform a maximal effort (Cowan et al, 2001).

A generally accepted method that has been reported to increase reliability of onset time evaluation and to decrease the need of experienced user is the use of a computer algorithm. Furthermore, in order to maintain the validity of the onset time determination by a computer algorithm through visual inspection for the elimination of artefacts & other interferences is recommended (Hodges & Bui, 1996). The reviewed studies using computer algorithms and visual inspection included Van Tiggelen et al, (2009), McClinton, et al, (2007), Cowan et al, (2002), Cowan et al, (2001) and Karst and Willet (1995). These were generally of good quality but even so three were in favour of a VMO:VL ratio muscle imbalance and two not in favour in PFPS. Morrish and Woledge (1997) did not define the method used and Powers et al, (1996) determined the onset time as the time at which the amplitude exceed 5% of maximal voluntary contractions without visual inspection despite the observation that the use of maximal contraction is considered

problematic due to pain inhibition in patients with PFPS (Cowan et al, 2001; Stensdotter et al, 2006).

Mellor & Hodges (2005) stated that, although the reasons for the discrepancies in the literature concerning the onset time are not clear, one method to resolve this debate is to study control at the level of motor unit activity, rather than studying whole muscle function. Recent data suggests that coordination of the vasti muscle is simplified by unexpectedly high synchronisation between VMO and VL motor units (Mellor & Hodges, 2006). Motor unit synchronisation is considered to be due to common synaptic input from branched presynaptic neurons or synchronised input via interneurons, resulting in a simultaneous discharge of action potentials in two or more motoneurons (Mellor and Hodges, 2005). A plausible question about this type of approach is whether the individual motor unit activity can guarantee a total representation of the muscle tested as a whole. . A recent computer algorithm employed in the onset time detection is based on double-threshold statistical detector, whose accuracy has been proposed to be higher than the rest of computer algorithms (Merlo et al, 2003). This method was employed by Cavazzuti et al, (2010) but the overall quality of the study was moderate. The selection of the appropriate onset time detection method has substantial role to play in avoiding type I errors (Chester et al, 2008) and when same or similar methods are used to enable direct comparisons between different studies.

Selection of the studied muscles and electrode positioning was another important issue. Although the majority of the studies recorded VMO and VL, some studies chose to measure VM instead of VMO (Pal et al, 2011) or VLL and VLO instead of VL (Bevilaqua-Grossi et al, 2008; Santos et al, 2007; Santos et al, 2008). Additionally not all studies reported in detail the methodology of electrode positioning. Some (Pal et al, 2011; Powers, 2000) just referred to previous publications (such as Perotto et al, 2005, or Basmajian & DeLuca, 1985) or previous studies (Liebensteiner et al, 2008) and others did not state the exact location of the positioning. Consequently, direct comparisons with other studies or replications are at least doubtful.

Another reason for the different results may be related to different experimental protocols, namely, the different joint angles, different type of contractions, different experimental conditions used in the studies [eg, open kinetic chain (OKC), closed kinetic chain (CKC), weight-bearing (WB), non-weight bearing (NWB)], functional tasks or less functional. It is generally accepted that the functional tasks have inherently larger variation than the non-functional tasks & reflex responses (Cowan et al, 2001). Reduced VMO activity in comparison to VL is reported under functional and CKC conditions (Souza & Gross, 1991, Taskiran et al, 1998, Santos et al, 2008) and also under non-functional & OKC conditions (Boucher et al, 1992; Cesarelli et al, 1999; Tang et al, 2001). In contrast other studies have failed to find any differences (Sheehy et al, 1998; Powers, 2000; Santos et al, 2007; Ott et al, 2011). These controversies may be attributed to motor unit recruitment (Chester et al, 2008) or task specific adaptations (Stensdotter, 2005). Kinematic characteristics are altered during fast speed walking and PFPS

patients tend to decrease knee flexion and reduce speed (Powers et al, 1997 & 1999). Similarly, during stair ascent-descent PFPS patients prefer slower speed and less knee flexion. Reduced gait velocity and knee flexion range consequently lead to, decreased quadriceps force demands, lower ground reaction forces and less PF joint loading (Brechtel & Powers, 2002). Additionally, disuse atrophy and decreased recruitment ability of the high threshold motor units of VMO & VL have been found in PFPS patients (Grabiner et al, 1992). Investigation of controlled tasks that limit the variability associated with functional movements is likely to provide more consistent data and also possibly to establish an accepted method to evaluate whether the recruitment of the VMO and VL is altered in people with PFPS but such tasks were rarely used (Cowan et al, 2002).

Only a few studies attempted to estimate the reliability of the determination of the EMG timing onset (Cowan et al, 2001; Karst & Willet, 1995; McClinton et al, 2007) and the EMG amplitude-ratio (Patill et al, 2011; Powers, 2000). Therefore, interpretation of results deriving from non-adequately described experimental conditions with questionable reliability may be interpreted with caution.

4.4.2 MEASURES AND CLINICAL SIGNIFICANCE

A number of studies investigated reflex responses (Voight & Weider, 1991; Karst & Willet, 1995; Witvrouw et al, 1996; On et al, 2004; Bevilacqua-Grossi et al, 2008) the

clinical significance of which may be questionable. Firstly, the functional relevance of the findings rests on the assumption that changes in reflex latencies are associated with similar changes in relative timing of onset of activity of the VMO and VL during voluntary activation of the knee extensors in functional tasks or less functional tasks. Secondly, the magnitude of any PFPS associated changes in timing must be sufficient to result in consequential muscle force imbalance. Additionally, the effect of subject height is strongly correlated with the reflex latency and must be taken into account when comparing absolute reflex latencies (Karst & Willet, 1995). In the reviewed studies of onset time three out of 15 studies have used the reflex response technique. According to Cowan et al, (2001) and Mellor and Hodges, (2005) measurement of the reflex responses has questionable clinical significance because evaluation of the response to a tendon tap provides information about the monosynaptic reflex and not muscle coordination associated with functional tasks.

Another issue about the timed onset is how large must be the delayed onset in milliseconds in order to have a meaningful clinical implication? Computer simulations findings suggested that a biomechanical alteration in PF joint can be caused from even 5ms delayed onset of the VMO (Neptune et al, 2000). Nevertheless, functionally it is not known whether a difference of 15-20 ms has a significant biomechanic effect on the patellofemoral joint yet this information would be required to evaluate the specific clinical effects of a deficit of this magnitude and to determine whether changes of this magnitude would be clinically identifiable (Cowan et al, 2001). Delayed onset time of the magnitude of

15-20 ms between VMO and VL might be significant statistically but not clinically significant (Brindle et al, 2003). This is complex issue that needs further investigation because is related also with anatomical and kinesiological factors, such as the condition of various static and dynamic stabilisers of the patella (e.g plica, patellar ligaments, muscles e.t.c) but is outside the scope of this thesis. Recently Pal et al, (2011) reported that significant relationship exist between vastus medialis (VM) delayed onset (21ms in jogging) and patellar maltracking (abnormal lateral tilt & glide) but is hard to make a clear assumption about the influence of the isolated VMO delayed onset. This finding stresses the necessity for detailed classification of the PFPS patients.

4.4.3 SAMPLE SIZE & HOMOGENEITY

The majority of the studies had a small sample size resulting in limited power of the results (minimum no of patients 8 and maximum 49, mean 17.8 patients per study). Sheehy et al, (1998) stated that in order for their study to detect a difference in VMO:VL ratio and onset time between groups with and 80% power a sample size of 45 subjects per group would be needed. Similarly, power calculation based on the data of Callaghan et al, (2001) revealed that a sample size of 186 in each group would be required to achieve 80% power in any new fatigue study. Therefore this indicates that the results have to be interpreted with caution as power is difficult to be achieved with the sample size used in the most, if not all, of

the reviewed studies. A possible appropriate method for ensuring the adequate sample size in future studies would be a power calculation.

Another crucial factor could have been a lack of homogeneity within the PFPS group (Brindle et al, 2003). The inclusion and exclusion criteria used in the majority of the studies were very vague and insufficient. Similarly the duration of PFPS symptoms had great variability ranging from few months to several years.

Furthermore in some cases it was obvious that poor patient population selection criteria had probably predetermined the direction of the results. For example in the study of MacIntyre (1992) all eight patients recruited had lower extremity malalignment features such as rear-fore foot alignment angles $\geq 5^\circ$ and over pronation of the foot. In a sample like this it is believed that is more likely the PFPS etiopathogenesis of the patients to be related with the above mentioned biomechanical characteristics and not with the muscular imbalance of the quadriceps. Additionally, in the study of Karst and Willet (1995) three out 15 patients reported an onset of PFPS following surgery for meniscal or ligamentous injury and in two out 15 patients onset resulted after direct patellar trauma. No attempt was made in any of the studies to carry out individual screening of the patients by using specific functional assessment scales in order to match, sub-classify or reduced the heterogeneity of the patient population.

The patellofemoral joints of the symptomatic subjects used in most of the subjects were not monitored for the pain level experienced during the time of data

collection. The only studies that monitored the pain level were, Sheehy et al, (1998), Cesarelli et al, (1999), Brindle et al, (2003), Cowan et al, (2001), Tang et al, (2001), Owings & Grabiner, (2002), McClinton et al, (2007), but they did not sufficiently describe the results. Nor were any attempts made to measure patients muscular performance under conditions that simulate the activities of daily living, for example measuring the muscular performance after the patients have had executed certain repetitive or sustained load task. Such activities are included in patients' everyday living routine and therefore are possible to correlate with the manifestation of their symptoms. Only one study attempted to monitor the VMO-VL activity during a dynamic tasks following aerobic exercise. PFPS patients experienced higher level pain after aerobic exercising had greater reduce in VMO activity compared to VL (Ott et al, 2011).

4.4.4 ANATOMICAL AND BIOMECHANICAL CHARACTERISTICS

It is possible that some anatomical and biomechanical characteristics of the patients such as, Q angle, genu valgum (tibiofemoral angle in the frontal plane < 170°), genu recurvatum (knee hyperextension > 10°) over-pronation of the foot could influence the selective firing of the VMO and VL. These structural or postural alterations have been linked to etiopathology of PFPS via altered joint mechanics and restoration of the correct – static & dynamic - postural alignment of the patellofemoral joint or the entire lower limb is considered as a prerequisite for a successful long-term conservative treatment (Fredericson & Yoon, 2006; Piva et al,

2006; Witvrouw et al, 2005). One of the reviewed studies made an attempt to rule out the Q angle parameter as potential factor associated with onset time imbalance (Boucher et al, 1992), and two studies tested the potential correlation between patellar tracking and quadriceps activity (Pal et al, 2011; Powers, 2000).

4.4.5 Summary

Much of the existing evidence in relation to VMO-VL muscle imbalance remains controversial and should be interpreted with extreme caution and as such it is difficult to conclusively determine from this review if VMO-VL imbalance exists in PFPS. The reviewed papers revealed large variations between subjects, groups and studies. Also are evident a marked heterogeneity and methodological limitations. Nevertheless, in agreement with the quote of Wong (2009) "that the evidence of the VMO-VL muscle imbalance although exists is not yet convincing" we would add that the evidence are there waiting to be revealed.

Chapter 5

Overview of the Quadriceps
Femoris muscular performance of
the healthy population

5. Overview of the Quadriceps Femoris muscular performance of the healthy population

5.1 Introduction

According to theoretical rationale, a necessary prerequisite for optimal patellar tracking in the healthy population is a balanced activity between VMO-VL (Cowan et al, 2001). Despite this theoretical perspective it has been proposed that the onset time of a VMO contraction should precede VL (Grabiner et al, 1994) because VL has larger cross sectional area (CSA) (Wickiewicz et al, 1983) and a higher velocity as a consequence of a greater number of fast twitch fibres (Johnson et al, 1973). Others have argued (Cowan et al, 2001) that VMO has mechanical advantage due to muscle fibre orientation (50° - 55° from the longitudinal axis) in comparison with the VL fibre orientation (12° - 15°) and therefore VMO is recruited first to counterbalance the superior force & velocity characteristics of the VL. It would appear therefore that there are differing opinions / rationale for what happens in terms of VMO-VL recruitment / activation strategies in a healthy population and before making comparisons between healthy and PFPS patients it is essential to understand what is really happening in people without lower limb pathology (Wong, 2009).

To enhance this understanding a search was conducted for the period between 1980 and 2010 and revealed 47 studies that assessed VMO – VL activity in healthy populations. Review of the studies revealed that in general, closed kinetic chain

(CKC) activities promoted earlier onset times and greater amplitude of the VMO in comparison with the VL, in 21 out of 47 from the reviewed studies² (Table 5.1, page 94). Similarly, in nine studies employing open kinetic chain (OKC) activities, the results showed that there was no evidence of VMO – VL muscle imbalance³ (Table 5.1, page 94). Furthermore, five studies employing both CKC & OKC activities in order to evaluate the VMO-VL amplitude & onset time in healthy subjects, also found that the VMO was recruited earlier & had greater activity than the VL⁴ (Table 5.1).

Several studies (11 out of 47 reviewed studies) revealed contradictory results even within the study, i.e. some of the tested conditions were in favour and other tested conditions were not in favour of VMO – VL muscle imbalance (Table 5.2).

During *closed kinetic chain (CKC) activities*, Kim et al (2009) compared VMO-VL ratio between genders during vertical drop landing and found a ratio of 1.07 in males and 0.78 in females respectively. Shields et al (2005) examined the VMO-VL amplitude during single limb squat (SLS) at 0°-40°-0° knee flexion-extension range using low (0%), medium (4%) and high (8%) resistance set as percentage of body weight. They identified no results indicating muscle imbalance in eccentric

² (Boling et al, 2006, Ciccotti et al, 1994, Cowan et al, 2000, Cowan et al, 2001, Cowan & Crossley, 2007, Earl et al, 2001, Earl et al, 2004, Edwards et al, 2008, Hertel et al, 2004, Hinman et al, 2005, Hopkins et al, 1999, Janwantanakul et al, 2005, Lange et al, 1996, Mohan, 2008, Reynolds et al, 1983, Rozzi et al, 1999, Serpanou & Trigkas, 2004, Torry et al, 2005, Tseng et al, 2007, Trigkas, 2001, Zografidi & Trigkas, 2004).

³ (Chan et al, 2001, Ebenbichler et al, 1998, Grabiner et al, 1991, Hanten & Schulthies, 1990, Mellor & Hodges 2006, Ono et al, 2002, Szczepanski et al, 1991, Worrell et al, 1995, Zakaria et al, 1997).

⁴ (Gryzlo et al, 1994, Herrington et al, 2006, Smith et al, 1995, Stensdotter et al, 2003, Wilk et al, 1996).

contraction with low resistance and in concentric contractions with medium & high resistance. In contrast, concentric contraction with low resistance & the eccentric contractions with medium and high resistance revealed results in favour of muscle imbalance. Hodges et al (2009) monitored the amplitude & onset time during stair stepping task and revealed that the VMO EMG activity occurred 13.2

Table 5.1: Overall findings and information of the not in favour of VMO-VL muscle imbalance reviewed studies with healthy population.

No	Study	Outcome Measure	No of healthy	Tasks	Result & Notes
1.	Hinman et al, 2005	Onset Time	66	CKC	Not in favour
2.	Hopkins et al, 1999	Amplitude	38	CKC	Not in favour
3.	Rozzi et al, 1999	Onset time & Amplitude	34	CKC	Not in favour
4.	Janwantakul et al, 2005	Amplitude	30	CKC	Not in favour
5.	Cowan & Crossley, 2007	Onset Time	29	CKC	Not in favour
6.	Edwards et al, 2008	Amplitude	25	CKC	Not in favour
7.	Ciccotti et al, 1994	Amplitude	22	CKC	Not in favour
8.	Earl et al, 2001	Amplitude	20	CKC	Not in favour
9.	Mohan et al, 2008	Amplitude	20	CKC	Not in favour
10.	Reynolds et al, 1983	Amplitude	20	CKC	Not in favour
11.	Trigkas, 2001	Amplitude	20	CKC	Not in favour
12.	Earl et al, 2004	Amplitude	19	CKC	Not in favour
13.	Boling et al, 2006	Amplitude	15	CKC	Not in favour
14.	Torry et al, 2005	Amplitude	13	CKC	Not in favour
15.	Serpanou & Trigkas, 2004	Amplitude	12	CKC	Not in favour
16.	Zografidou & Trigkas, 2004	Amplitude	12	CKC	Not in favour
17.	Tseng et al, 2007	Amplitude	11	CKC	Not in favour
18.	Cowan et al, 2000	Onset Time	10	CKC	Not in favour
19.	Cowan et al, 2001	Onset Time	9	CKC	Not in favour
20.	Hertel et al, 2004	Amplitude	8	CKC	Not in favour
21.	Lange et al, 1996	Amplitude	6	CKC	Not in favour
22.	Worrell et al, 1995	Amplitude	32	OKC	Not in favour
23.	Sczepanski et al, 1991	Amplitude	30	OKC	Not in favour
24.	Hanten & Schulthies, 1990	Amplitude	25	OKC	Not in favour
25.	Zakaria et al, 1997	Amplitude	20	OKC	Not in favour
26.	Ebenbichler et al, 1998	Amplitude	18	OKC	Not in favour
27.	Chan et al, 2001	EMD	17	OKC	Not in favour
28.	Grabiner et al, 1991	Fatigue	9	OKC	Not in favour
29.	Ono et al, 2002	Amplitude	7	OKC	Not in favour

30.	Mellor & Hodges 2006	Synchronisation	5	OKC	Not in favour
31.	Herrington et al, 2006 (a)	Amplitude	43	OKC & CKC	Not in favour
32.	Smith et al, 1995	Onset Time	24	OKC & CKC	Not in favour
33.	Gryzlo et al, 1994	Amplitude	12	OKC & CKC	Not in favour
34.	Stensdotter et al, 2003	Onset time & Amplitude	10	OKC & CKC	Not in favour
35.	Wilk et al, 1996	Amplitude	10	OKC & CKC	Not in favour

Legend: OKC= open kinetic chain, **CKC=** closed kinetic chain

ms before the VL in step-up but was delayed by 4.0 ms during step-down. The VL amplitude was greater than the VMO in both stepping conditions.

When both *open kinetic chain (OKC)* and *closed kinetic chain (CKC)* activities were tested during the same study, again the results were contradictory. Bowyer et al (2008) revealed muscle imbalance in OKC activity (straight leg raise) but in CKC (step-down) the VMO-VL ratio was not in favour of muscle imbalance. Similarly, Irish et al (2010) found muscle imbalance during OKC (90°-0° knee extension – VMO-VL= 0.72 ratio) but in weight-bearing CKC activities (double leg squat + isometric hip adduction, lounge exercise) the VMO-VL ratio was 1.18 & 1.14 respectively. Mirzabeigi et al (1999) also found no difference between VMO-VL amplitude in four OKC (full & short isokinetic arc of knee extension, from full extensions to full flexion with valgus & varus stress) and in two CKC (from full squat to upright position & jump squats) tasks but a VMO-VL muscle imbalance was revealed in three OKC tasks (MVIC at 15° with neutral, external & internal hip rotation).

Similar contradictory results were identified in studies employing only *open kinetic chain (OKC)* activities. Matheson et al (2001) examined VMO-VL amplitude during 13 OKC activities (including various types of resistance: isokinetics, elastic tubes,

free weights & various speeds) and revealed results in favour of VMO-VL muscle imbalance in only 2/13 tested conditions. Similar results were identified in the OKC study of Karst & Jewett (1993) with the VMO-VL activity being balanced during quadriceps set exercise but not balanced in three variations of the straight leg raise exercises. During isokinetic (OKC) evaluation of the quadriceps isometric torque, the VMO amplitude was higher in comparison to VL in 40°-60°-70°-80° & 90°, and respectively lower in 10°-20°-30° & 50° (Brownstein et al, 1985). In 20° of isometric knee extension Grabiner et al (1992) revealed results in favour of VMO-VL muscle imbalance at 25% of knee extension MVIC & also in 25% and 50% of knee extension MVIC with concurrent addition of 50% of hip adduction MVIC but the opposite was revealed at 50% & 75% of knee extension MVIC and in 75% of knee extension MVIC with the addition of 50% of hip adduction MVIC. Similarly, Herington & Pearson (2006b) at 25%-50% & 75% of MVIC during 30° of knee extension found no difference in onset time activity. The VMO-VL ratio was also not in favour of muscle imbalance at 100% MVIC (VMO-VL=2.35) but was in favour of muscle imbalance during the rest levels of MVIC (75% MVIC ratio=0.92, 50% MVIC=0.85 & 25% MVIC= 0.73)

Table 5.2: Overall findings and information of reviewed studies with contradictory results, in favour and not in favour.

No	Study	Outcome Measure	No of healthy	Tasks	Result & Notes
1.	Kim et al, 2009	Amplitude	30	CKC	(-) in Male (+) in Female
2.	Bowyer et al, 2008	Amplitude	30	OKC & CKC	(-) in CKC (+) in OKC
3.	Matheson et al, 2001	Amplitude	28	OKC	(-) in 11/13 tasks (+) in 2/13 tasks
4.	Irish et al, 2010	Amplitude	22	OKC & CKC	(-) in CKC (+) in OKC
5.	Shields et al, 2005	Amplitude	15	CKC	(-) in 3 CKC tasks (+) in 3 CKC tasks
6.	Karst & Jeweett, 1993	Amplitude	12	OKC	(-) in 2/5 tasks (+) in 3/5 tasks
7.	Brownstein et al, 1985	Amplitude	11	OKC	(-) in 40,60 ,70,80 & 90° (+) 10, 20, 30 & 50°
8.	Grabiner et al, 1992	Amplitude	10	OKC	(-) in 50 & 75% of MVIC (+) in 25% of MVIC
9.	Herrington & Pearson, 2006	Onset Time & Amplitude	10	OKC	(-) onset-time in 7/10 part. (+) onset-time in 3/10 part. (-) RMS in 75-50-25% MVIC (+) RMS in MVIC
10.	Hodges et al, 2009	Onset Time & Amplitude	10	CKC	Onset Time: (-) in step-up, (+) step down RMS: (+) in both stepping tasks
11.	Mirzabeigi et al, 1999	Amplitude	8	OKC & CKC	(-) in 4 OKC & 2 CKC tasks (+) in 3 OKC tasks

Legend: **OKC**= open kinetic chain, **CKC**= closed kinetic chain, **MVIC**= maximum voluntary isometric contraction, **part.**= participants

In only one out of 47 reviewed studies was it evident that the results were in favour of VMO-VL muscle imbalance (Table 5.3). In the study of Van Deun et al (2007) the onset time of the vasti muscle was monitored during the transition from double-leg stance position to a single-leg stance position. With the eyes open the VMO onset time was delayed 9 ms in comparison to VL and with the eyes closed the VMO onset time was 7 ms delayed.

Table 5.3: Overall findings and information of reviewed studies with healthy population revealed in favour of VMO-VL muscle imbalance results.

No	Study	Outcome Measure	No of healthy	Tasks	Result & Notes
1	Van Deun et al, 2007	Onset time	30	CKC	In favour

Legend: OKC= open kinetic chain, CKC= closed kinetic chain

5.2 RESULTS OF THE CONTROL HEALTHY SUBJECTS OF THE REVIEWED PFPS STUDIES

Without a clear understanding of whether VMO-VL muscle imbalance exists or not in a healthy population, it is difficult to make objective comparison between normal patients and those with lower limb pathology and to set the limits of a causative relationship between normal and abnormal data and eventually understand the contribution of VMO:VL activity to knee extensor mechanism disorders. In an attempt to have a more holistic view of the question of VMO-VL muscle balance / imbalance an overview of the data from the healthy subjects

recruited in the reviewed PFPS studies. The reason for this way of thinking is to increase the volume of available data of VMO-VL from healthy subjects. Additionally, we wanted to compare whether the trend of results presented in the healthy subjects studies is similar or not with the results of the healthy control subjects participated in the reviewed PFPS studies.

Table 5.4 presents an overview of the evaluation of EMG amplitude, onset time and fatigue of the control healthy subjects included in the reviewed studies. As it is shown the amplitude – ratio and fatigue study results of healthy subjects are not in favour VMO – VL muscle imbalance. Furthermore, 16 out 20 studies present onset time and reflex responses data that are not in favour of the muscle imbalance picture. In two studies where the results were partially in favour of muscle imbalance, Cowan et al. (2001) have found 27 % and 24% of the control subjects during the concentric and eccentric tasks respectively activated VL > 10ms earlier from VMO. Additionally Cowan et al. (2002) reported that in several subjects in the control group, the onset time of the VL occurred earlier of that of the VMO. In both cases the authors argued that these healthy subjects may be predisposed to future development of patellofemoral pain. Moreover, Sheehy et al. (1998) stated that there was a trend noted in the timing of peak muscle activity whereas peak VMO activity tended to occur after peak VL activity for the asymptomatic group. Santos et al, (2008) also reported that in 10 healthy control subjects the VMO-VLO (vastus lateralis oblique) ratio was 0,79 and the VMO was 4ms delayed in comparison to VLO.

Table 5.4: Results of the VMO-VL muscle imbalance in control healthy subjects recruited in the reviewed studies.

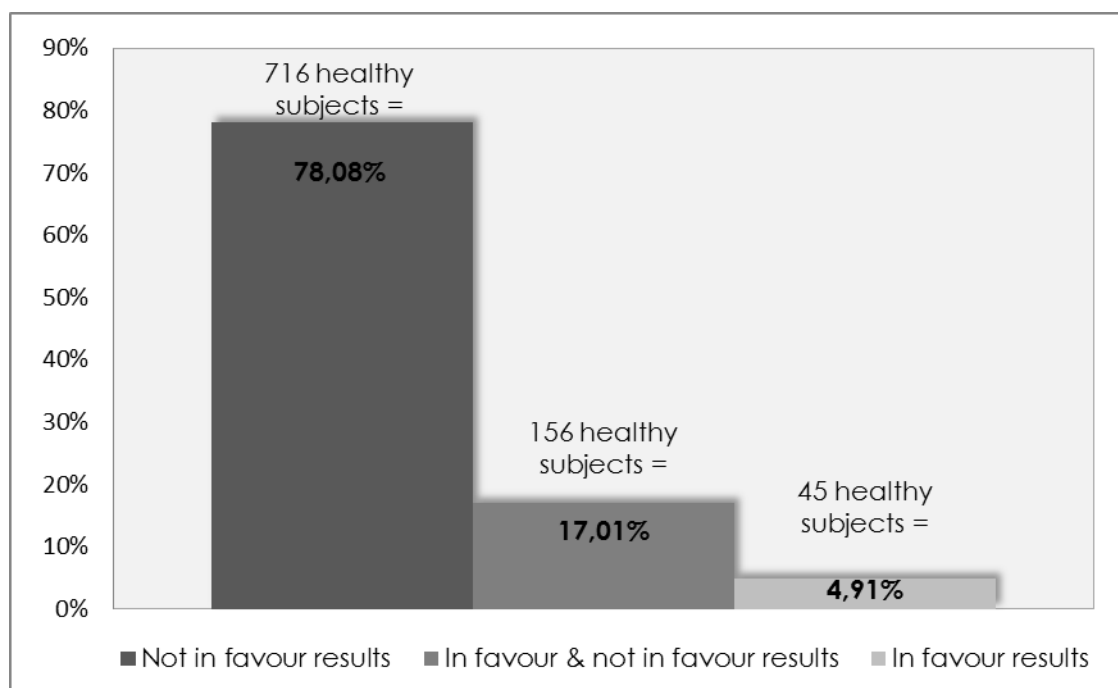
1. AMPLITUDE & RATIO STUDIES					
No	Study	Outcome Measure	Healthy subjects	Results	Score in points
1.	Powers, 2000	Ratio	12	Not in favour	78
2.	Ott et al, 2011	Amplitude	20	Not in favour	75,5
3.	Santos et al, 2007	Ratio	15	Not in favour	70,5
4.	Sheehy et al, 1998	Ratio	15	Not in favour	70
5.	Liebensteiner et al, 2008	Amplitude	19	Not in favour	69,5
6.	McClinton et al, 2007	Ratio	20	Not in favour	68,5
7.	Stensdotter et al, 2006	Ratio	17	Not in favour	67
8.	Powers et al, 1996	Intensity %	19	Not in favour	66
9.	Owings et al, 2002	Amplitude	14	Not in favour	65
10.	Tang et al, 2001	Ratio	10	Not in favour	65
11.	Santos et al, 2008	Amplitude	10	Not in favour	64,5
12.	Cesarelli et al, 1999	Amplitude	30	Not in favour	64,5
13.	Boucher et al, 1992	Ratio	9	Not in favour	62,5
14.	Mohr et al, 2003	% of max.	11	Not in favour	60
15.	MacIntyre, 1992	Amplitude	12	Not in favour	60
16.	Souza & Gross, 1991	Ratio	7	Not in favour	56,5(N-N)- 66,5(N)
17.	Taskiran et al, 1998	Ratio	9	Not in favour	54
18.	On et al, 2004	Amplitude	13	Not in favour	48
19.	Grabiner et al, 1992	Amplitude	15	Not in favour	46
20.	Moller et al, 1986	Amplitude	14	Not in favour	44,5
21.	Mariani & Carouso, 1978	MUAPS	5	Not in favour	38,5
2. TIME ONSET & REFLEX RESPONSE STUDIES					
1.	Cowan et al, 2001	Comp.algor. > 3SD	33	Partially Positive	78
2.	Pal et al, 2011	Comp.algor. > 3SD	15	In favour	77,5
3.	Van Tiggelen et al, 2009	Comp.algor. > 3SD	53	Not in favour	76
4.	Patil et al, 2011	Comp.algor. > 3SD	17	Not in favour	75,5
5.	Cowan et al, 2002	Comp.algor. > 3SD	37	Partially Positive	75
6.	* Sheehy et al, 1998	Differenc. in onset	15	Positive trend	70
7.	* McClinton et al, 2007	Comp.algor. > 2SD	20	Not in favour	68,5
8.	Witvrouw et al, 1996	Compar. Rest val.	80	Not in favour	67,5
9.	Mellor & Hodges, 2005	MUAPS synchronis.	10	Not in favour	67,5
10.	* Stensdotter et al, 2006	Autoregres. algor.	17	Not in favour	67
11.	* Powers et al, 1996	Exceed 5% mmit	19	Not in favour	66
12.	Brindle et al, 2003	> 5 SD from Rest val.	12	Not in favour	65,5
13.	Karst & Willet, 1995	Comp.algor. > 1SD	12	Not in favour	65,5
14.	* Owings et al, 2002	Compar. Rest val.	14	Not in favour	65
15.	* Cesarelli et al, 1999	Temporal identif.	30	Not in favour	64,5
16.	* Santos et al, 2008	Comp.algor. > 3SD	10	In favour	64,5
17.	Bevilaqua et al, 2008	Comp. Rest Value	12	Not in favour	64
18.	Voight & Weider, 1991	Comp. Rest Val.	41	Not in favour	59,5
19.	Cavazzuti et al, 2010	Double threshold	20	Not in favour	59,5
20.	* On et al, 2004	Comp. Rest Val.	13	Not in favour	58
21.	Cavazzuti et al, 2009	Double threshold	17	Not in favour	54
22.	Morrish & Woledge, 1997	Lag factor	20	Not in favour	47
3. FATIGUE STUDIES					
1.	Callaghan et al, 2001	Fatigue ratios	10	Not in favour	74,5
2.	Vöötäinen et al, 1991	MPF & ZCR	18	Not in favour	28

Outc. = outcome, **N** = normalised **N-N** = non-normalised, **Comp.algor.**= computer algorithm, **Compar. Rest val.** = comparison with rest value, **MPF**=median power frequency, **ZCR**=zero crossing rate, **mmit** =maximum muscle isometric test, **identif.**= identifier, **IN FAVOUR** = indicates results in favour of differences, **NOT IN FAVOUR** = indicates results not found any difference, ***** = studies that have also measured and EMG amplitude or ratio.

5.3 DOES THE VMO-VL MUSCLE IMBALANCE ALSO EXIST IN THE HEALTHY POPULATION?

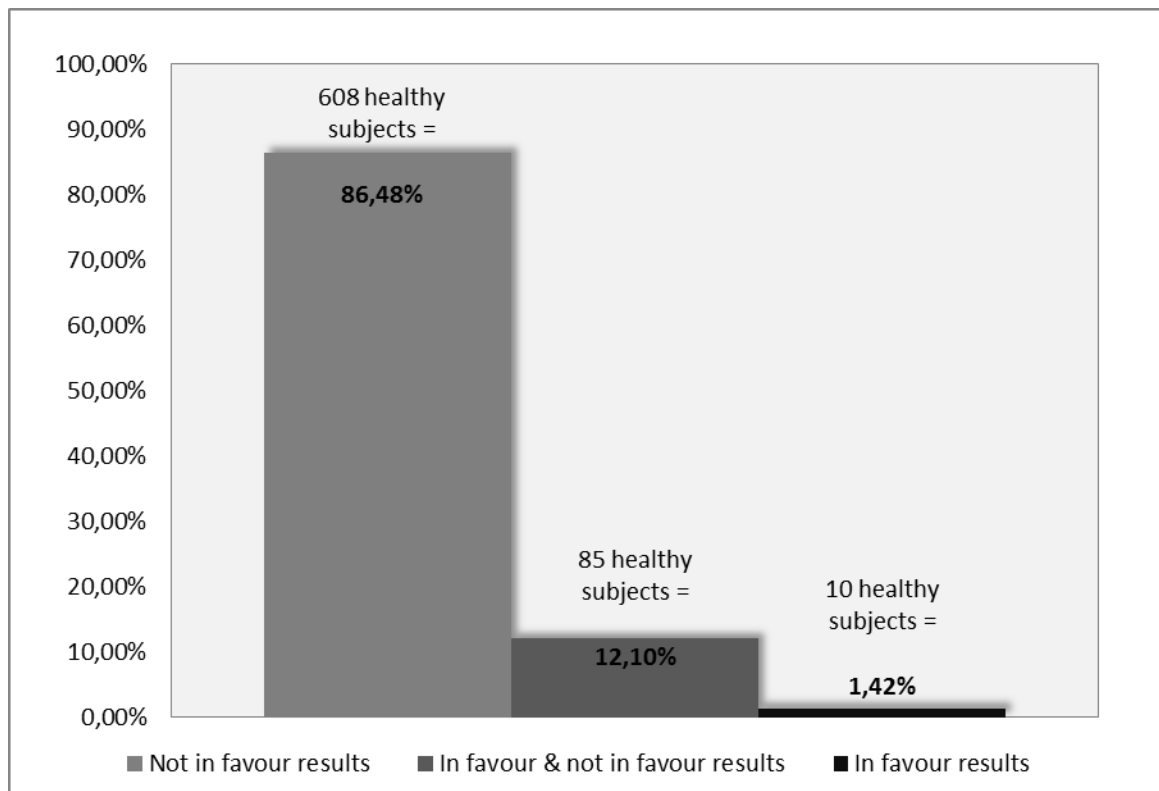
In the 47 studies reviewed that just looked at healthy populations the overview revealed that in total 917 healthy subjects participated in various OKC, CKC & in some cases in both. From the total number of 917 healthy participants, in 716 (78,1 %) the results of the VMO-VL EMG activity were not in favour of muscle imbalance. In 156 healthy subjects (17 %) mixed results were presented i.e. in some of the tested conditions a VMO-VL muscle imbalance was evident but in other experimental conditions was not. Only in 45 healthy subjects (4,9 %) was a clear VMO-VL muscle imbalance reported (Figure 5.1)

Figure 5.1: Presenting the percentage of the healthy subjects muscle imbalance results



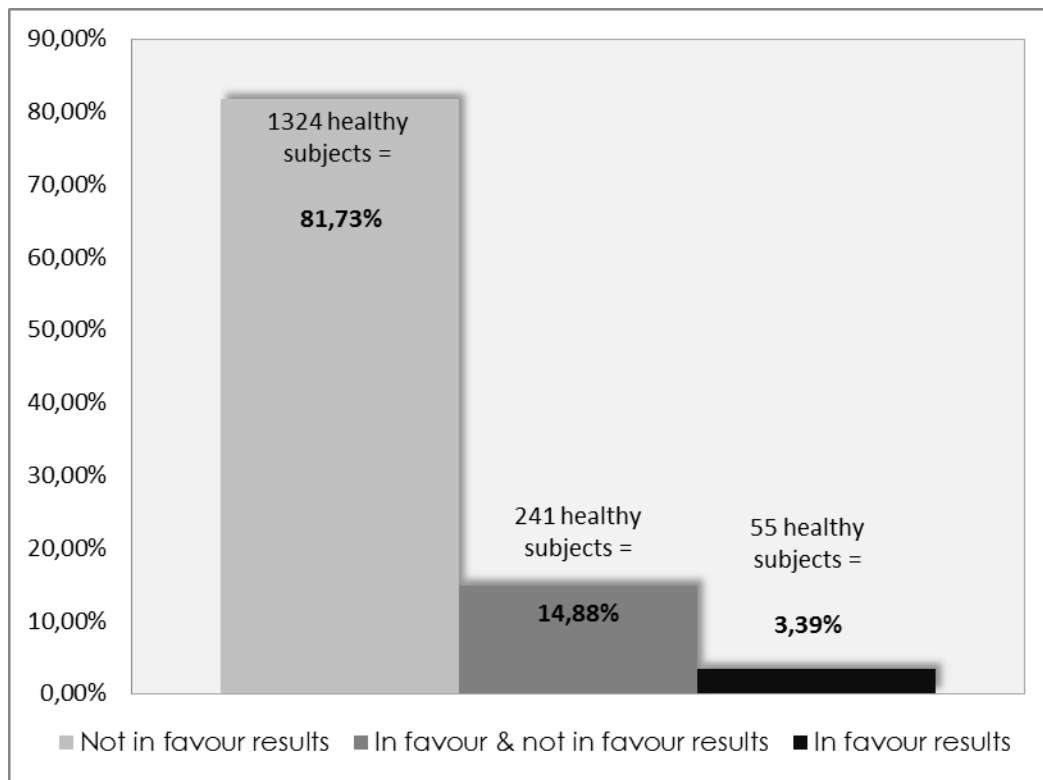
In the reviewed 38 PFPS studies the total healthy control subjects were 703, from which 608 healthy control subjects (86,4 %) had no VMO-VL muscle imbalance, 85 healthy subjects (12,1 %) presented mixed results i.e. in some cases the results were in favour of muscle imbalance and in other cases were not in favour. Finally in only 10 healthy control subjects (1,4 %) the results were clearly in favour of VMO-VL muscle imbalance (Figure 5.2).

Figure 5.2: Presenting the percentage of muscle imbalance results of the healthy control subjects participated in the reviewed PFPS studies.



As is obvious there is similar trend in terms of the existence of VMO-VL muscle imbalance in the healthy population participated in the reviewed PFPS studies and in the studies with only healthy subjects. Summing the results of the healthy subjects reveals that in total number of 1620 healthy participants, 1324 healthy subjects (81,7 %) had no VMO-VL muscle imbalance, 241 healthy subjects (14,8 %) had mixed results (i.e in favour and not in favour) and only 55 healthy subjects (3,3 %) had a clear manifestation of VMO-VL muscle imbalance (Figure 5.3)

Figure 5.3: Presenting the overall results of muscle imbalance of the healthy subjects participated in reviewed PFPS studies & in studies with only healthy subjects.



On the basis of these results it is likely that the VMO-VL muscle imbalance clearly exists in a small percent (3,3 %) of healthy subjects. Additionally, in another part of the population (14,8 %) there is evidence that there is trend in manifestation of muscle imbalance under certain conditions. Therefore, it is more likely that in a subgroup of the healthy population the VMO-VL muscle imbalance is apparent.

Cowan et al (2002a) argues that eventually healthy subjects with VMO-VL muscle imbalance are more susceptible to development of PFPS. Impaired neuromuscular control of the VMO-VL has been previously identified as contributing factor for the development of PFPS (Van Tiggelen et al, 2009, Witvrouw et al, 1996). In a prospective study with healthy subjects Van Tiggelen et al, (2009) reported that 32% of the population had 1,67ms VMO delayed onset in comparison to VL. After a six weeks strenuous basic military training the 32% of the recruits with the pre-existing muscle imbalance developed PFPS and the reassessment of the VMO-VL onset time revealed that the delayed onset of the VMO was increased to 17,73 ms. The authors stated that the delayed VMO onset should be regarded as causative factor due to pre-existence but also as a consequence because after the intense physical activity the muscle imbalance was exacerbated. Consequently, the VMO-VL muscle imbalance could be considered as the substrate on which eventual supra-physiological overloading can trigger either the initiation or exacerbation of the PFPS (Dye, 2004, pp. 3-18). It is well understood and documented that the PFPS is a multifactorial condition cannot be predicted by a single risk factor. Nevertheless the VMO-VL muscle

imbalance has a predictive value as a risk factor and should not be underestimated (Van Tiggelen et al, 2009).

The findings of the overview of the VMO-VL muscle activity in a healthy population it is obvious that it is generally balanced in the majority (81,73%) of, but is also clear that in a minority of the healthy subjects the muscle imbalance is either clearly evident (3,39%) or apparent (14,88%) under certain conditions. The findings from these studies demonstrate a considerable variability and are subject to heterogeneity either methodologically (convenience sample, relatively small sample size etc.) or in terms of the employed procedures (electrode placement, sampling frequency, onset time determination, smoothing & filtering etc.). Therefore it is necessary that future studies reduce methodological out limitations and flaws, and also to attempt clarify the patterns of physiological variability of VMO-VL muscular performance and finally, correlate muscular activity with patellar kinematics. This will help to establish a normative data base and thus to improve the determination & interpretation of the altered muscular activity of the VMO-VL in patients with PFPS.

Chapter 6

Aim, Hypothesis, Objectives,
Experimental Design

6. AIM – HYPOTHESIS – OBJECTIVES – EXPERIMENTAL DESIGN

6.1 AIM OF THE STUDY

As briefly outlined in Chapter 1 the ultimate aim of the study was to establish if it is appropriate to continue addressing a VMO – VL muscle imbalance, and treating with physiotherapeutic interventions, patients with clinically defined patellofemoral pain syndrome (PFPS).

In order to address this long term aim the current study was designed to establish if a VMO – VL muscle imbalance actually exists in a clinically defined PFPS population and if this imbalance is related to clinical symptoms associated with this condition and/or lower limb muscle physiology.

6.2 OBJECTIVES:

The overall objectives of the study were to establish:

4. If VMO – VL muscle imbalance exists in patellofemoral pain syndrome (PFPS) patients and if so is it specific to this condition or does a similar VMO – VL muscle imbalance exists in a healthy population?
5. If muscle imbalance does exist is it related to clinical symptoms used as indications of pain syndrome in clinical practice?
6. Is muscle imbalance associated with lower limb muscle physiology i.e. lower limb and quadriceps muscle strength in both fresh and fatigued states.

6.3 NULL HYPOTHESES TO TEST OBJECTIVE 1:

NH1 – The relative EMG amplitude of VMO vs VL during functional and experimental load tasks in fresh muscles will be no different between patients with PFPS and age/sex matched asymptomatic subjects.

NH2 – The relative activation timing of the VMO in relation to VL during functional and experimental load tasks in fresh muscles will be no different between patients with PFPS and age/sex matched asymptomatic subjects.

6.3.1 PLAN OF INVESTIGATIONS & PROCEDURES

6. 3.1.1 SUBJECTS:

63 patients with patellofemoral pain syndrome (PFPS) and 63 age/sex matched healthy subjects were recruited to this study. The sample size of the study was extracted using power calculation analysis based on the results of previous similar study (Sheehy et al, 1998) and is presented in Table 6.1. The descriptive statistics for the two groups (PFPS patients and healthy controls) are presented in Tables 6.2 page 110.

Ethics approval was granted by Committee on the Ethics of the Research on Human Beings of the University of Manchester (ref 08179) and informed consent obtained from volunteers at the screening visit.

Table 6.1: Power calculation analysis for sample size estimation based on study of Sheehy et al (1998) using the step-down phase (eccentric contraction).

α	power	Δ	σ	m	Sample size
.01	.80	1.493 -1.158=0.335	.527 + .559:2=.543 mean SD	1	63

Table 6.2: Descriptive statistics for the subjects

	N	Age (years)		Height (cm)		Weight (Kg)	
		Mean	± SD	Mean	± SD	Mean	± SD
PFPS patients	63	25.9	7.2	173.9	9.5	71.9	14.6
Healthy subjects	63	26	7.3	171.5	8.7	70.6	15.8

6.3.1.2 GROUP 1 - PFPS PATIENTS

Patients were referred from 5 regional hospitals (Orthopaedic clinics & Physiotherapy departments) and from local rehabilitation centres, medical and physiotherapy private clinics.

Inclusion criteria

The following inclusion – exclusion criteria are based on those used in other PFPS studies (Callaghan et al, 2001, Cowan et al, 2001, Cowan et al, 2002, McClinton et al, 2007, Mohr et al, 2003, Stensdotter et al, 2006). Subjects had a physician's diagnosis of PFPS for a minimum of 3 months, age 18 - 45 years in order to avoid difficulties in differentiating between PFPS, late symptoms of apophysitis and early

symptoms of osteoarthritis thereby reducing possible extraneous effects (Voight & Wieder, 1991). In order to ensure the diagnosis subjects were also required to report *anterior or retropatellar pain* on at least 2 of the following activities:

Squatting, kneeling, ascending/descending stairs, prolonged sitting with flexed knees, walking or running in level surface, walking or running uphill or downhill, hopping/jumping. These criteria were confirmed by the applied principal investigator during an initial screening visit in order to confirm the appropriateness of the referred patients. Pain on patella medial & lateral facet palpation, average pain level of 3 cm on a 10 cm visual analogue scale (VAS) and an insidious onset of symptoms unrelated to a traumatic incident provided further confirmatory evidence of diagnosis.

Exclusion criteria

Subjects of this group were excluded if they reported having:

Any previous knee surgery, osteoarthritis, history of traumatic patellar instability, dislocation or subluxation, meniscal involvement or ligament injuries of the knee, functional (symptomatic) instability in the lower extremity, chondral damage, knee soft tissue injury (i.e. fat pad impingement), knee soft tissue overuse (i.e. tendinopathy), apophysitis, synovial plica, spinal or hip referred pain, neuroma. These specific exclusion criteria were applied in order to avoid conditions or pathologies with symptoms that mimic PFPS but they are caused by different pathologies (Piva et al, 2006, Souza & Gross, 1991).

6.3.1.3 GROUP 2 - HEALTHY SUBJECTS

Healthy subjects were recruited from Institutional student and community populations and from the local community.

Inclusion criteria

The healthy subjects were also between 18 - 45 years age were matched (in five year band widths) and sex matched with the pathological group. They were included in the study if they had:

No previous knee pathology or history of injury or surgery to the lower limb or back, no pain, discomfort, restriction/limitation in motion of their lower limbs, no knee effusion, no involvement in elite competitive sport, any systemic or metabolic disorder.

6.3.2 EXPERIMENTAL DESIGN TO TEST OBJECTIVE 1

The study used a cross-sectional design. Subjects were asked to visit the laboratory on one occasion to measure VMO & VL amplitude and activation timing during different functional and experimental tasks.

6.3.2.1 MEASURES OF MUSCLE FUNCTION

Warm-up: Prior to the muscle function testing session all subjects completed a warming-up stretching regime to reduce any discomfort experienced especially

during the experimental maximal contractions and to reduce the intensity of any post exercise muscle soreness (De Vries, 1961). The warm-up aimed to improve the transition from rest to exercise state, via either physiological or psychological mechanisms. Stretching exercises are considered as an important component of a warming-up regime (Smith, 1994). Subjects were instructed by the main investigator on the stretching exercise for the quadriceps, hamstrings and plantar flexors muscle groups. They performed 3 repetitions of 20 seconds static stretch for each muscle group at a moderate intensity.

Measures of VMO – VL EMG amplitude and activation time were measured using the following techniques:

EMG recordings: The dominant lower extremity in all healthy subjects was used for EMG recordings and it was determined as the leg used to kick a ball. In subjects with PFPS, the affected knee was monitored. In cases with bilateral pathology the knee with the more severe symptoms was measured.

In order to avoid poor contact and to lower electrical impedance, electrode and skin preparation instructions were followed as described by Kasman (1998, pp. 165-169). The skin of each subject was prepared for EMG electrode placement by shaving the excessive hair (if necessary), abrading with fine sandpaper and then cleansing the recording site with surgical spirit.

The muscles of interest were large and superficial and therefore surface electrodes (silver/silver chloride pregelled self-adhesive electrodes – EF Medica SRL) were used as recommended by Basmajian and DeLuca (1985, pp. 36-37).

Two electrodes were placed over the relevant muscle belly (see below), parallel to the alignment of the muscle fibres with an inter-electrode distance of 2 cm. Optimal electrode placement sites were confirmed by observation and palpation of subject's quadriceps during isometric contraction with the knee extended (Kasman, 1998, pp. 165-169). Prior to the EMG recordings, in order to minimise the noise from the tissue-electrode junction, skin impedance was always monitored, using an alternating current impedance analogue multimeter. Every effort was made to maintain the skin impedance between each recording electrode at less than 5 K Ω (Mannion & Dolan, 1996). If it was necessary, the recording sites were prepared again, as described previously.

Electrode position was determined using a protractor and tape measure and was marked on the skin with indelible ink:

VMO: over the muscle belly approximately 4cm superior to and 3 cm medial to the superior-medial border of the patella and orientated at 55° from the long axis of the femur

VL: over the muscle belly 10 cm superior and 6 – 8 cm lateral to the superior border of the patella orientated at 15° from the long axis of the femur. The reference electrodes was placed on the tibial tubercle & lateral maleol (Figure 6.1) (Basmajian & Blumstein, 1980, Cowan et al, 2002).

Figure 6.1: Electrode placement for VMO & VL.

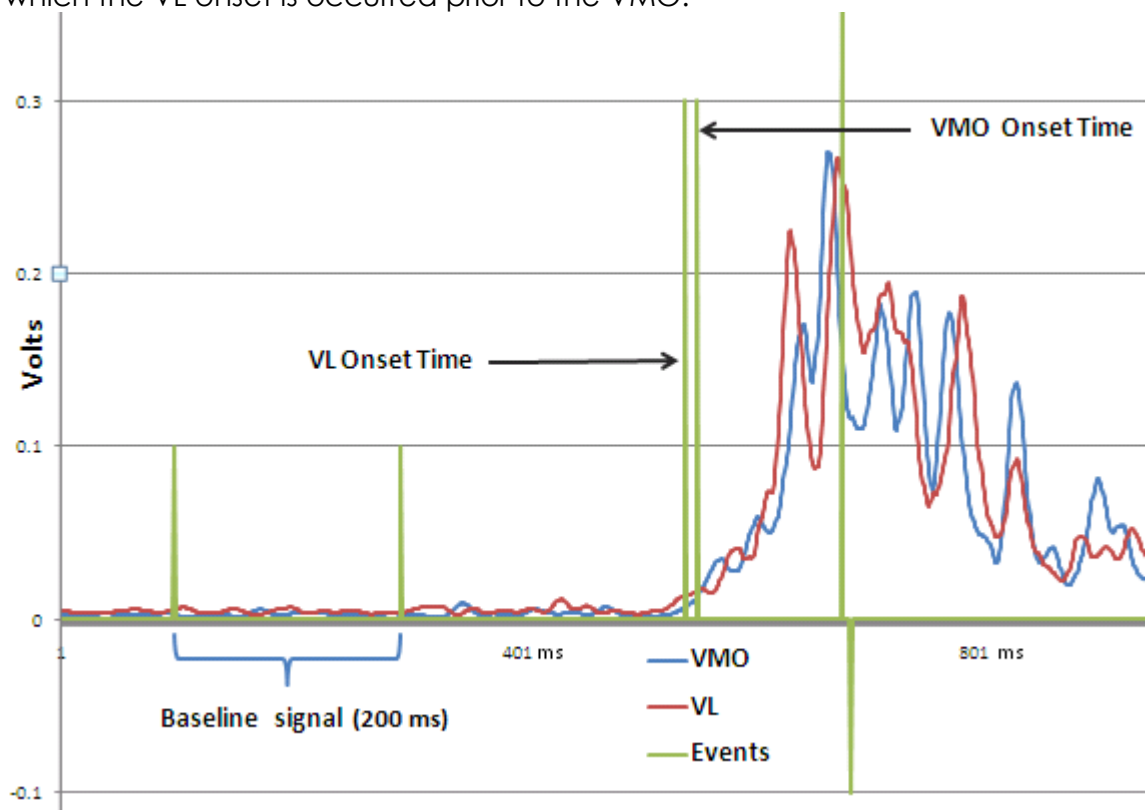


VMO – VL EMG amplitude: The EMG activity was sampled during concentric/eccentric & isometric contractions during functional, experimental & repetitive load tasks described below starting with a verbal command and a simultaneous activation of the EMG recording apparatus.

VMO – VL activation time: A previously validated computer algorithm was used to identify the onset time of EMG activity of the two vasti muscles. The algorithm identified the point at which the EMG signal deviated more than 3 standard deviations (SDs) for a minimum of 25 ms above the baseline level (Cowan et al, 2001). The rectified unfiltered EMG data was visually inspected to verify the onset detected by the computer. The sampling rate of the EMG recording apparatus

allowed a resolution of 1ms. Figure 6.2 shows an example of EMG data obtained from a PFPS patient during a step-up functional task. The EMG onset time was taken as the average of 3 repetitions. The relative difference in the time of onset of EMG activity of the VMO & VL was calculated by subtracting the EMG onset of the VMO from the VL onset, therefore negative values representing a delayed onset of the VMO in comparison with the VL and positive values the opposite.

Figure 6.2: Representative EMG data of a PFPS patient from a step-up task in which the VL onset is occurred prior to the VMO.



EMG instrumentation: A 16 channel EMG system (AD Instruments) was used for the EMG recordings during the open kinetic chain concentric/eccentric/isometric contractions of the experimental, functional and repetitive load tasks. Online real

time analysis of the electromyographic signal from VMO & VL was performed every second by the Power Lab system (AD Instrument Power Lab -16SP). The Common Mode Rejection Ratio (CMRR) of 110 dB minimum and a signal-to-noise ratio of 65 dB minimum was used. The signal was analogue-to-digital converted at a sampling rate of 1024 Hz.

Isokinetic dynamometer: A Biodex system 3 pro, isokinetic dynamometer (Biodex Medical Systems, Shirley, NY) was used for monitoring the knee extension torque during the open kinetic chain concentric/eccentric/isometric contractions of the experimental and repetitive load tasks and was synchronised with the EMG system.

The measures of muscle function described above were tested under the following functional and experimental load tasks and also under the repetitive load tasks to test objective 3.

6.3.2.2 FUNCTIONAL TASKS

The following functional task was included:

Ascending and descending stairs – whereby the step up component represents a concentric contraction and the step down an eccentric contraction. The step –

up & step down task was performed at a normal step height of 20 cm under two different conditions determined by a metronome:

- at a normal speed of 96 steps/minute,
- at a faster speed of 116 steps/minute.

The step height of 18 cm is generally considered as a normal step height found in most buildings and also has been employed in previous studies (Brindle et al, 2003, McClinton et al, 2007, Sheehy et al 1998).

The normal speed of 96 steps/minute is considered that approximates usual stair stepping pace (Cowan et al, 2001) and has been employed during studies of Cowan et al, (2001), Gilleard et al, (1998) and McClinton et al, (2007).

The faster speed of 116 steps/minute was employed in order to monitor whether the differing speed is affecting the EMG amplitude and activation time. Furthermore, the everyday life activities are performed at variable speed and it was important therefore to compare two different stepping speeds. Additionally, has been identified that a decreased explosive strength capacity is an intrinsic risk factor for patellofemorall pain (Witvrouw et al, 2000).

The order of stepping task (step up, step down, at normal and at fast speed) was randomized. Subjects were asked to perform three consecutive cycles of the step up and step down activity leading with the painful limb for the patients and dominant limb for the controls for both the concentric and eccentric phases of the cycle. In order to determine the initiation and the cessation of the concentric

phase (ascending) and the eccentric phase (descending) of the stepping task a foot switch (FSR/force sensitive resistors with 1000 gain) was placed in each subject's shoe and was synchronised with the EMG recordings. Prior to the EMG recordings subjects performed a minimum 5 of practice trials under the principal investigator supervision in order to familiarise themselves with the normal and fast rate of the stepping task determined by the metronome. A rest period of 30 sec between repetitions was employed in order to re-establish the base line and a rest period of 2 minutes was employed between ascending/descending activities.

The stair ascending/descending task was chosen because represents a closed kinetic chain weight bearing activity very often performed in everyday life activities and furthermore is one of the most common activities described as being associated with PFPS. The task has also been included as testing condition in many of the previous studies that evaluated VMO & VL EMG activity in PFPS patients and control subjects (Brindle et al, 2003, Cowan et al, 2001, McClinton et al, 2007, Sheehy et al, 1998, Powers et al, 1996).

6.3.2.3 EXPERIMENTAL TASKS

ISOKINETIC MEASUREMENTS

1. Open kinetic chain (OKC) Isokinetic measurements

Positioning & preparation: The subjects were seated in dynamometer with the trunk and the thigh supported and fastened by seatbelts placed above the pelvis,

the trunk and the proximal part of the thigh. The hip joint angle was set at 90° and knee at 60° of flexion for the normalising procedure (maximal voluntary isometric contraction-MVIC). The hip joint angle was set at 90° of flexion and the knee joint at 90° and 0° of flexion for concentric & eccentric contraction respectively, and 90° of hip flexion and 75° of knee flexion for the isometric contraction. The centre of motion of the lever arm was manually aligned with the flexion-extension transverse axis of the knee joint. The resistance pad was placed on the distal part of the tibia above the ankle. A few practice contractions were performed prior to data collection in order for the subjects to familiarise themselves with the experimental tasks. After the positioning, preparation and familiarisation subjects performed the following experimental tasks:

Normalisation procedure: Subjects performed three 5 second repetitions of maximal voluntary isometric contraction (MVIC) at 60° of knee flexion. A rest period of two minutes was allowed between the repetitions. The MVIC at the knee flexion at 60° was used in order to normalize the EMG data and thereby make comparisons between subjects during the functional, experimental and repetitive load tasks.

After the normalisation procedure a two minutes rest followed and then the subjects performed the three experimental tasks:

A. Three repetitions of maximal concentric contraction: from 90° knee flexion - to full knee extension, at an angular velocity of $90^\circ/\text{sec}$ (Figure 6.3).

B. Three repetitions of maximal eccentric contraction: from knee full extension – to 90° knee flexion, at an angular velocity of $90^\circ/\text{sec}$.

C. Three repetitions of maximal isometric contraction: performed at 75° of knee flexion with duration of five seconds each.



Figure 6.3: Patient position using Biodex dynamometer for the concentric task.

The order of 3 experimental tasks was randomized and a rest period of two minutes between the repetitions and the sets was allowed.

Both concentric & eccentric contractions were included due to their resemblance with every day life activity contraction demands. Open kinetic chain isokinetic contractions (concentric & eccentric) at range of motion 0° - 90° - 0° knee flexion with the angular velocity of 90°/sec have been found to be reliable and well

tolerated by PFPS patients (previous unpublished observation). Furthermore previous studies have used the same range of motion and the same angular velocity in order to avoid overloading and high articular stress of the patelofemoral joint (Cessarelli et al, 1999, Cessarelli et al, 2000). The isometric contraction at an angle of 75° of knee flexion has also proven to be reliable in patients with PFPS and additionally is an angle at which the quadriceps has the optimum length – tension properties. The joint position of 60° was chosen for the normalising procedure because in the mid range of knee flexion ($\approx 50^\circ - 80^\circ$) the length-tension relationship and moment arm of the quadriceps is almost ideal, therefore the quadriceps can reach the peak force (Knapik et al, 1983, Oatis, 2004, pp 756-757) .

6.4 NULL HYPOTHESES TO TEST OBJECTIVE 2

NH3 – The relative EMG amplitude of the VMO vs VL during functional, experimental load tasks will not correlate to the results from the measures of clinical symptoms of the PFPS patients.

NH4 – The relative activation time of the VMO in relation to VL during functional, experimental load tasks will not correlate to the results from the measures of clinical symptoms of the PFPS patients.

6.4.1 EXPERIMENTAL DESIGN TO TEST OBJECTIVE 2

The study used a cross-sectional design.

6.4.1.1 MEASURES OF CLINICAL SYMPTOMS

The assessment measures of clinical symptoms took place prior to the muscle function measures in order to avoid any possible alterations to patients' clinical picture due to functional, experimental and repetitive load exercising. Measures of clinical symptoms were subdivided into three areas of assessment namely I) pain & function, II) muscular flexibility and III) lower limb biomechanics.

6.4.1.2 Assessment of Pain & Function

1. OVERALL ASSESSMENT OF PAIN

Patients' overall evaluation of pain was performed using a 10 cm visual analog scale (VAS) for their worse (VAS-W) and usual (VAS-U) pain during the past week. They were required to mark their pain on a 10cm line anchored by the terms no pain (0 score) through to worst pain ever (10 score). The VAS has been found to be reliable, valid and responsive and subsequently is recommended for in clinical trials and clinical settings in patients with PFPS (Crossley et al, 2004).

Pain is the dominant feature of PFPS therefore the amount of knee pain is paramount in the evaluation of treatment outcome and is common to use a 10 cm VAS to assess pain (Crosley et al, 2004). Both, VAS-W and VAS-U have been used in intervention studies for PFPS (Harrison et al, 1999, Thomeé, 1997).

2. SELF ADMINISTERED ANTERIOR KNEE PAIN SCALE (AKPS)

The anterior knee pain scale (AKPS) (Kujala et al, 1993) is a questionnaire consisting of 13 questions divided in distinct categories related to various levels of current knee function. Categories included in each question are weighted and the responses are summed to provide an overall index in which 100 points represents no disability and 0 points represents the worse possible disability. The anterior knee pain scale (Kujala et al. 1993) has been proven to be reliable, valid and responsive for patients with PFPS (Watson et al, 2005, Crossley et al, 2004). Subjects were instructed how to complete the scale and they completed it in their own time and on their own. The anterior knee pain scale (AKPS) score is represented as an absolute value (i.e. 65) out of a total 100. The description of anterior knee pain scale (AKPS) is presented in the Appendix II.

Patellofemoral pain syndrome frequently leads to disability which usually means difficulty or avoidance performing activities that overload the patellofemoral joint and increase the articular stress. The majority of these activities are part of every day life activities such as, stairs descending/ascending, squatting, prolonged sitting with knees flexed and some are included in common sports activities such as running, jumping and hopping (Crosley et al 2004, Thomeé et al, 1995).

6.4.1.3 Assessment of muscular flexibility

Assessment of muscle flexibility comprised four measures including the quadriceps, hamstrings, iliotibial band-tensor fascia lata and plantar flexors.

Muscle tightness such as shortening of quadriceps, hamstrings, iliotibial band and plantar flexors have all been associated with extensor mechanism disorders and namely with PFPS. Additionally, evaluation of the flexibility of these muscles is an essential part of PFPS patients' clinical assessment. It has been suggested that tightness of quadriceps and hamstrings muscles may increase patellofemoral joint compression forces during daily life or sports activities and in this way predispose healthy individuals to PFPS (Piva et al, 2006, Smith et al, 1991, Witvrouw et al, 2000). Additionally, there is some evidence to support an association between tightness of foot plantar flexors and development of PFPS (Witvrouw et al, 2000). Tightness of the iliotibial band-tensor fascia lata may result in a lateral displacement of the patella and thus increase the stress in the patellofemoral joint and medial retinacular tissue (Brody & Thein, 1998, Wilk et al, 1998).

1. *Quadriceps length:*

Subjects were lying in the prone position and muscle length was assessed by measuring the knee angle during passive knee flexion using a clinical (gravity) goniometer (MIE Medical Research Ltd. Leeds, UK). Prior to the measurement the clinical goniometer was zeroed on the horizontal surface of the examining table. The examiner stabilised the patient's pelvis in order to control pelvis anterior tilting and/or lumbar spine extension. The knee joint was passively flexed and the knee

angle was recorded with the goniometer placed over the distal of the tibia (Figure 6.4) (Piva et al, 2006). The average measurement of 3 measures with 10 seconds rest time between each was recorded. The quadriceps length assessment has been found to have a substantial reliability in PFPS patients (Piva et al, 2006).



Figure 6.4: Quadriceps length assessment.

2. Hamstrings length:

The subject was lying supine with the tested knee in extension and the contralateral knee also in extension in order to avoid excessive posterior tilt of the pelvis. The examiner palpated the anterior superior iliac crest of the pelvis in order to monitor any posterior pelvis tilt. Prior to the measurement the gravity goniometer was zeroed on the distal part of the tibia and then the examiner passively lifted the leg in hip flexion until the knee started to flex or the pelvis demonstrated posterior tilt (Figure 6.5). The average measurement of 3 assessments with 10 seconds rest time between assessment was recorded. The assessment of hamstrings length has been found to have substantial reliability in PFPS patients (Piva, et al 2006).



Figure 6.5: Hamstrings length assessment.

3. *Iliotibial band (ITB) – tensor fascia lata (TFL) length (Ober's test):*

The subject was in a side-lying position with the examined leg upper most and the lower leg in 45° of hip and knee flexion to maintain pelvis and trunk stability. Prior to the test the gravity goniometer was zeroed on a horizontal level and then was positioned over the distal part of the Iliotibial – tensor fascia lata complex. The examiner was positioned behind the patient and grasped with his distal hand the examined leg to perform 90° of knee flexion. Initially, the examiner moved the patient's thigh in flexion and then through abduction, combined with extension, until the hip was placed in mid range abduction with neutral flexion-extension

position. From this position the thigh was allowed to drop until the point where the limb stopped moving toward to the table (Figure 6.6).



Figure 6.6: Iliotibial Band length assessment.

This was the point at which the measurement was taken and the result was recorded as a continuous variable, where negative values represent more tightness and positive values (bellow horizontal level) represent less tightness (Piva et al, 2006). The average of 3 measurements with 10 seconds rest time between trials was recorded. The iliotibial band-tensor fascia lata complex length assessment has been found to have substantial reliability in patients with PFPS (Piva, 2006).

4. *Plantar flexors length:*

The plantar flexors (gastrocnemius and soleus muscles) length was determined by measuring the range of ankle dorsi-flexion with the knee joint extended and

then flexed at 90 degrees. With the knee in an extended position the range of ankle dorsi-flexion is indicative of gastrocnemius muscle tightness and with the knee flexed the range of dorsi-flexion is indicative of soleus muscle and joint capsule tightness. The patient was lying in a supine position with the knee extended and the foot hanging outside the table. The subtalar joint was maintained in a neutral position. The examiner performed passive dorsi-flexion of the foot and the range of dorsi-flexion was measured with a standard goniometer as the angle formed by the lateral midline of the leg from the head of the fibula to the tip of the lateral malleolus and the lateral midline of the foot in line with the border of the rear foot – calcaneus (Figure 6.7). The exact same procedure was then repeated with the knee flexed at 90° in order to assess the soleus muscle and joint capsule length with the patient in prone position (Figure 6.8). The average of 3 measurements with 10 seconds rest time between each was recorded. The assessment of plantar flexors length has been proven to have substantial reliability in patients with PFPS (Piva et al, 2006).



Figure 6.7: Gastrocnemius length assessment.



Figure 6.8: Soleus length assessment.

6.4.1.4 Biomechanical characteristics

Assessment of biomechanical characteristics included quadriceps angle (Q angle) and foot pronation.

Biomechanical characteristics related to structural or postural alterations such as quadriceps angle (Q angle) and abnormal foot pronation have been associated with the development of PFPS (Powers et al, 1995). Also, abnormal quadriceps angle values have been described as a discriminatory factor between runners with patellofemoral pain and asymptomatic runners (Messier et al, 1991). Although Thomeé et al, (1995) suggested that there is no direct correlation between high Q angle and patellofemoral pain they stated that an abnormal Q

angle may be a contributing factor to maintaining PFPS once the syndrome has been acquired. Both, measures, Q angle and foot pronation, are an essential part of a thorough clinical examination and successful management of these structural – postural alterations has been considered as a prerequisite for successful long term conservative treatment (Witvrouw et al, 2005).

1. Quadriceps angle - Q angle measurement:

The quadriceps angle is measure of the acute angle between a line from the anterior superior iliac spine through mid patella and a line through mid patella and tibial tuberosity (Figure 6.9). The Q angle was measured with subjects lying in the supine position with the knee in full extension and the quadriceps muscle completely relaxed.



Figure 6.9: Q angle measurement.

2. *Foot pronation:*

Foot pronation was measured by using the navicular drop test. The test screens the height difference of the navicular at the subtalar joint during neutral position (corrected position) and during relaxed stance position. The subject was standing bare foot with his/her feet shoulder width apart and the examiner was positioned behind the patient with the eyes leveled at patient's feet. The examiner marked the navicular tuberosity of the patient and then helped the subject to put the subtalar joint in neutral position. The examiner measured the distance from the navicular to the floor using a tape measure placed perpendicular to the step and then the patient was instructed to relax and put his/her feet in a relaxed stance position and then the measurement was repeated (Figure 6.10). The distance between the two dots in the tape measure, which represents the difference in the position of the navicular tuberosity with respect to floor between the subtalar neutral & relaxed stance positions, was measured in millimeters. *Greater distances* between the two dots indicated greater *foot pronation*. The assessment of foot pronation has been documented to have substantial reliability values (Piva et al, 2006).



Figure 6.10: Foot pronation-Navicular drop test.

6.5 NULL HYPOTHESES TO TEST OBJECTIVE 3

NH5 – The relative EMG amplitude of the VMO vs VL after experimental and repetitive load tasks will be no different between patients with PFPS and age/sex matched asymptomatic subjects.

NH6 – The relative activation time of the VMO in relation to VL after experimental and repetitive load tasks will be no different between patient with PFPS and age/sex matched asymptomatic subjects.

6.5.1 EXPERIMENTAL DESIGN TO TEST OBJECTIVE 3

The study used a cross-sectional design.

6.5.1.1 REPETITIVE LOAD TASKS

According to Dye (2001) the function of any joint, including the patellofemoral joint, is characterized by a load – frequency distribution which is called the envelope function. The envelope function defines a range of painless loading that is compatible with homeostasis of the articular tissues. Excessive loading of the patellofemoral joint can cause loss of homeostasis and subsequently lead to pain and dysfunction. The so called, supraphysiologic loading can be caused either from single event or a repetitive articular loading. Therefore it is suggested that during a treatment programme the patient must be assisted to find his/her envelope of function (Dye 2001).

Based on this way of thinking about the etio-pathology of PFPS, the assessment of the amplitude and activation time of the VMO & VL after a repetitive loading task and comparison of these results with the previously obtained results under same experimental task but during different muscle physiology state (fresh state) could reveal interesting findings.

The repetitive load effect study was examined by isokinetic closed kinetic chain consecutive concentric & eccentric contractions at a range of 45° - 0° - 45° of knee flexion – extension (Figure 6.11). In order to obtain the maximum voluntary contraction, the subjects performed three maximum contractions at range of 45° - 0° - 45° knee flexion-extension with two minutes rest interval between each contraction. The average of the 3 measures was taken as the maximum voluntary

contraction (MVC). A rest period of 6 min followed and then the repetitive load task was performed during a 60 sec contraction at a submaximal level of 50% of the MVC of each subject. A second rest period of 15 minute followed and then the subjects performed:

- Three maximal isometric contractions in the isokinetic dynamometer at 75° of knee flexion with a duration of five seconds and simultaneous recording of the electromyographic signal from VMO & VL. Subjects performed the three MVC with a rest period of 2 minutes between the repetitions.

The close kinetic chain eccentric – concentric contraction at a range of 0° - 45° of knee flexion was chosen in preference to the standard isokinetic lever arm (open kinetic chain knee extension) because close kinetic chain tasks have been advocated for PFPS patients to minimise the patellofemoral joint reaction force and stress in comparison to the open kinetic chain tasks. Therefore it is more appropriate and acceptable for both assessment and training for PFPS patients. The range of 0° - 45° of knee flexion is also considered as safe range in relation to patellofemoral joint reaction force and stress (Callaghan et al, 2001).

The isometric contraction at an angle of 75° of knee flexion has also proven to be reliable in patients with PFPS and additionally is an angle at which the quadriceps has the optimum length – tension properties.



Figure 6.11: Patient position using the closed kinetic chain device.

Chapter 7

Study I: Measures of muscle function

7. STUDY I: MEASURES OF MUSCLE FUNCTION

7.1 Descriptive Statistics of the Subjects

Table 7.1 summarises the demographic information for the experimental PFPS patient group and for the healthy subject control group. A total of 126 people were recruited into the study – 63 PFPS patients and 63 age /sex matched healthy subjects (29 male, 34 female per group). Subjects were not matched on the basis of height and weight but possessed similar anthropometric characteristics ($p < 0.05$).

Table 7.1: Descriptive statistics for the subjects

	N	Age (years)		Height (cm)		Weight (Kg)	
		Mean	± SD	Mean	± SD	Mean	± SD
PFPS patients	63	25.9	7.2	173.9	9.5	71.9	14.6
Healthy subjects	63	26	7.3	171.5	8.7	70.6	15.8

7.2 Overall muscular performance analysis

Table 7.2 summarises the results of the overall VMO-VL time onset difference of the PFPS patients and healthy controls when all functional and experimental tasks results are combined. This summary picture is presented as many previous studies adopted this combined approach. Please note throughout this chapter, **positive values** represent an earlier onset of the VMO in comparison with VL and **negative values** represent a delayed onset of the VMO in comparison with VL,

Table 7.2: Summary of the overall VMO-VL time onset difference in patients & healthy subjects across all functional & experimental tasks.

	Group	Mean difference between muscles (ms)	SD	<i>t</i>	df	p
VMO-VL Time Onset difference	PFPS	1	33	-5,713	880	0.000
	HEALTHY	10	18			

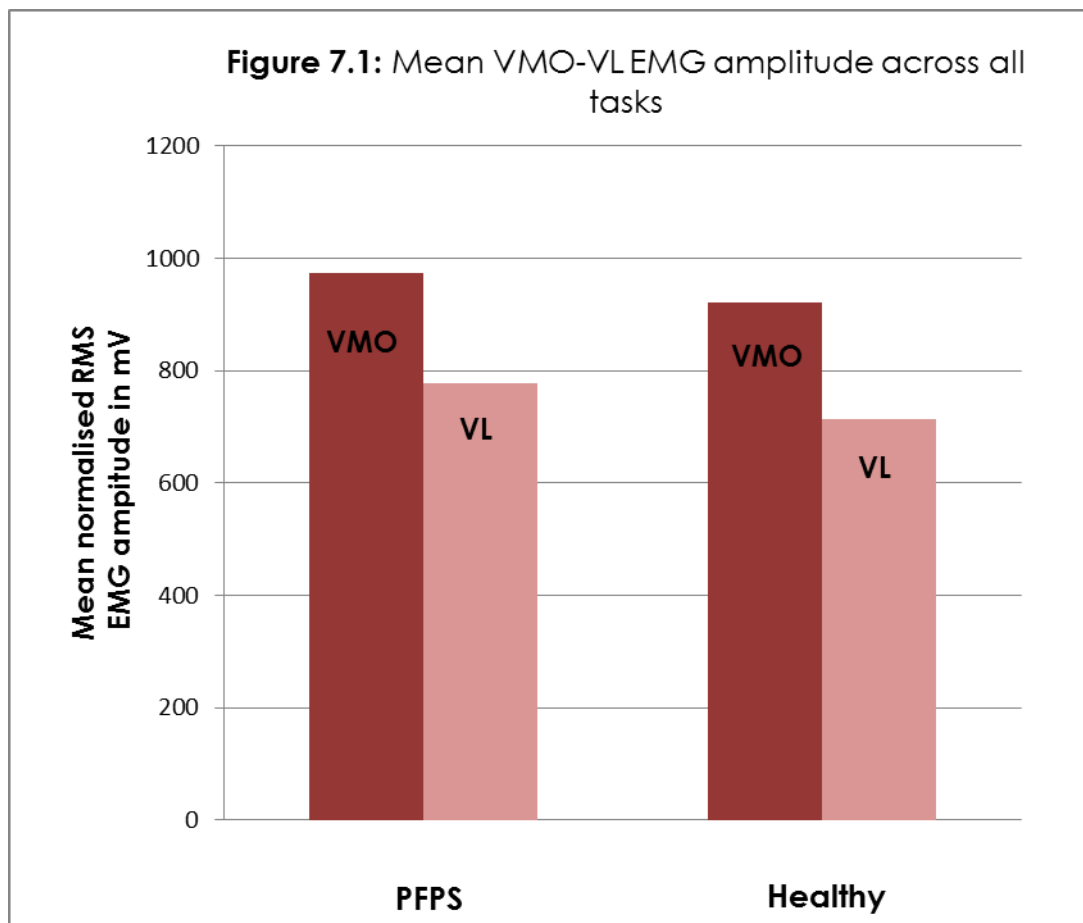
In PFPS patients the VMO was overall activated 1msec earlier than the VL and in matched healthy subjects the VMO was overall activated 10 msec earlier than the VL. The between group comparison of the VMO-VL time onset difference revealed that the 9ms difference between PFPS and healthy controls is highly significant ($p=0.000$).

Table 7.3 and Figure 7.1 summarise overall differences in the VMO vs VL EMG amplitude (normalised RMS) between PFPS patients and healthy controls across all functional and experimental tasks. Within group analysis revealed that the VMO EMG amplitude was significantly higher than VL in both groups ($p=0.000$). The differences in amplitude between VMO and VL was similar for both groups. In PFPS group the overall VMO amplitude was 196 millivolts (mV) higher than the VL and similarly in healthy subjects the overall VMO amplitude activation was 208 mV higher than the VL.

Table 7.3: Summary of the overall EMG amplitude difference (normalized RMS) for VMO-VL in PFPS patients & in healthy subjects across all functional & experimental tasks.

	Mean RMS mV	Mean RMS difference between muscles mV	SD	t	df	p
1.PFPS VMO VL	973 777	196	355	12,383	503	0.000
2.Healthy VMO VL	922 713	209	491	9,548	503	0.000

Figure 7.1: Mean VMO-VL normalised RMS EMG amplitude across all functional & experimental tasks combined.



Souza & Gross (1991) also have found higher VMO amplitudes (integrated EMG IEMG) in comparison to VL in both PFPS patients and healthy subjects (VMO:VL IEMG ratio was 1.30 and 1.10 respectively). Though, in this case the difference between groups was not significant. This lack of statistical significance may have been due to the small sample size (9 PFPS patients & 7 healthy subjects) and assessment of only two functional tasks (step-up & down) and one isokinetic task (submaximal isometric contraction at 10° of knee flexion), therefore direct comparison with the current study is difficult.

In contrast, the results of the current study are different from the findings of Santos et al (2008). They reported lower overall VMO activity than VLO (vastus lateralis oblique) in both PFPS patients and healthy subjects when combining all functional and experimental tested tasks. They also found that VMO had a significantly delayed ($p=0.01$) recruitment onset in comparison with VLO in both groups (10ms in PFPS group and 4ms in healthy controls). However, direct comparison with the current study is not possible due to some fundamental differences in design. Firstly Santos et al (2008) compared VMO to vastus lateralis oblique (VLO) rather than VL, their sample size was considerably smaller (10 PFPS & 10 healthy subjects) and the experimental and functional tasks were different.

In light of the limitations of the comparable studies looking at overall muscle performance (Souza & Gross 1991; Santos et al, 2008) it is difficult to confirm or refute the findings of the current study. Nevertheless the current study was well designed and well powered so it may be concluded that VMO is recruited before

and with a higher amplitude than VL in both PFPS patients and healthy controls though the relative delay in recruitment of VL is reduced in PFPS patients.

7.3 Overall muscular performance analysis by task

The previous section presents an overall view of muscle performance over a range of tasks but this approach may have masked true group differences. In the clinical setting it is common practice, during the assessment of a PFPS patient, to attempt to identify which movements trigger the pain and/or dysfunction in order to clarify the exact source or mechanism of the underlying pathology or injury. Among others, special attention is paid to specific functional tasks such as step climbing at different speeds. This thesis considers these parameters in more detail in order to ascertain if differences in muscular performance are task specific.

7.3.1 Functional tasks

7.3.1.1 Time Onset differences of PFPS patients vs. healthy subjects in step-up at normal speed (96 steps/minute)

In the concentric contraction phase of the step-up task at normal speed (96 steps/min) VMO was activated 9 ms earlier than the VL in PFPS patients and 14ms earlier for the healthy subjects (Table 7.4). The 5ms between group difference was not statistically significant ($p=0.08$).

Table 7.4: VMO-VL time onset difference in PFPS patients & healthy subjects during step-up at normal speed (96 steps/min).

	Group	N	Mean difference between muscles (ms)	SD	t	df	p
VMO-VL Time Onset Difference	PFPS	63	9	17	-1,415	124	0.08
	HEALTHY	63	14	15			

These results are similar to Brindle et al (2003) who using a similar stepping task procedure reported a 17.5 ms earlier activation of the VMO in PFPS patients and 13.7 msec in control subjects. Furthermore, as for the current study, the difference between the groups was not significant ($p>0.05$). However, the speed of the stepping task in the study of Brindle et al is not stated thus making the direct comparison problematic.

Taking the same speed and step height into account (20 cm step height, 96 steps/min) the same results as in the current study were reported by McClinton et al (2007) for PFPS with a mean 9.5 ms earlier VMO activation time than VL. In contrast, controls had a smaller (average 4.5ms) difference than PFPS compared to 14ms in the current study. Nevertheless, between group differences for both studies were not significant ($p>0.05$) thus confirming that at 96 steps/min there is no difference in activation time between VMO and VL between PFPS patients and normal controls.

In contrast to the picture emerging from the present study and confirmed by Brindle et al (2003) and McClinton et al (2007), Cowan et al (2001) reported that

the difference in onset time between PFPS and healthy volunteers was significant ($p < 0.05$). In a study, using the same step height and speed as in the current study, they reported the opposite findings for PFPS patients in that the VL activation time now preceded that of VMO by 15.8msec. In contrast VMO and VL had simultaneous activation times in the healthy controls. Similar findings were reported by Crossley et al (2004) who also found significant differences between groups with VL preceding VMO by 16.7 msec in PFPS compared to 2ms earlier activation of the VMO compared to VL in healthy controls. Just one year later the same research group in another similar study with a stepping task (Cowan et al, 2002) confirmed a VMO delayed activation relatively to VL in PFPS patients (16.6 ms) but this time demonstrated the complete opposite in healthy volunteers with VMO preceding VL activation by 15.9ms in healthy controls. Similarly, Bolling also found significant VMO delay (22.4ms) in the PFPS patient compared to 61.8ms in advance activation of the VMO relatively to VL in asymptomatic subjects (Bolling et al, 2006).

All of these finding (summarized in Table 7.5) seem to create a confusing picture regarding differences or similarities in VMO/VL activation times during stepping up at normal speed. Interestingly, the current study, designed to address limitations in previous studies supports the findings of McCinton et al (2007). This latter study was rated as one of the higher quality publications thereby possibly giving more credence to the results. It may be therefore that the trend is in favour of there being no statistically significant difference in activation times between VMO and VL between PFPS patients and healthy volunteers and that generally VMO is recruited before VL in a stepping up task at normal speed.

Table 7.5: Time onset differences (ms) in VMO & VL in PFPS patients and healthy controls in step-up task at a normal speed of 96 steps/min. Negative VMO-VL values indicate a VMO delayed onset in comparison to VL.

STEP-UP TASK STUDIES	PFPS			CONTROL			MEAN difference (ms)	STATISTICAL SIGNIF. Between groups
	Time differenc e (ms)	SD	N	Time differenc e (ms)	SD	N		
Trigkas current study	9	17	63	14	15	63	4	NO
Bolling 2006	-22.4	29	14	61.8	68. 7	14	84.2	YES
Brindle et al 2003	17.5	22.9	16	13.7	29. 9	16	3.8	NO
Cowan et al 2001	-15.8	29	33	0	18	33	15.8	YES
Cowan et al 2002a	-16.6	19	10	15.9	17. 3	12	32.5	YES
Crossley et al 2004	-16.7	17.6	47	2	1.5	18	18.7	YES
McClinton et al 2007	9.5	17.4	20	4.5	20. 1	20	5	NO

In Table 7.5 is presented a summary of the results of step-up tasks of our study and previous studies.

7.3.1.2 Time onset differences of PFPS patients vs. healthy subjects in step-up at fast speed (116 steps/minute)

In the concentric phase of the step-up at fast speed (116 steps/minute) VMO was activated 5 msec earlier than VL in PFPS patients. A similar picture was revealed in the healthy subjects when VMO time onset was 10 msec earlier than the VL (Table 7.6). The 5ms between group difference was statistically significant ($p = 0.021$).

Table 7.6: VMO-VL time onset difference in PFPS patients & healthy subjects during step-up at fast speed (116 steps/min).

	Group	N	Mean difference between muscles (ms)	SD	t	df	p
VMO-VL Time Onset Difference with 3SD	PFPS	63	5	16	-2,051	124	0.021
	HEALTHY	63	10	14			

It is apparent that the recruitment pattern during fast stepping up is similar to the one observed during the normal speed step-up. VMO still is activated earlier than the VL in both groups. The only notable alteration is that this earlier VMO activation is slightly reduced in both groups in comparison to the normal speed task. Direct comparison of the current differences revealed during the fast speed stepping task with previous data is impossible. All previously available studies either

employed a normal speed of step-up (96steps/min)(Bolling et al, 2006; Cowan et al. 2001; Cowan et al 2002a; Crossley et al, 2004; McClinton et al, 2007) or failed to control the stepping-up speed (Brindle et al, 2003; Sheehy et al, 1998; Powers et al, 1996). In light of knowledge to date, it would appear that the current study is the first to explore a controlled fast speed (116steps/min) of stepping up task. From the current results it would appear therefore, that the speed of stepping up does not influence recruitment time and order in both PFPS patients and healthy volunteers.

7.3.1.3 Time onset difference of PFPS patients vs. healthy subjects in step down at normal speed (96 steps/minute)

During the eccentric contraction phase of the step-down task at a normal speed (96 steps/minute) VMO was activated almost simultaneously with VL in the PFPS patients (Table 7.7: VMO-VL mean time onset difference <1 ms). In contrast, in the healthy subjects VMO was activated 16 ms earlier than VL (Table 7.7). The 15ms mean difference between the groups was statistically significant ($p=0.05$).

Table 7.7: VMO-VL time onset difference in PFPS patients & healthy subjects during step-down at normal speed (96 steps/min).

	Group	N	Mean difference between muscles (ms)	SD	t	df	p
VMO-VL Time Onset Difference	PFPS	63	<1	75	-1,617	124	0,05
	HEALTHY	63	16	23			

The results of the normal speed step-down task for the current study are different from previous studies. Previous studies testing a stepping down task at normal speed have reported a significant VMO delayed activation ($p=0.05$), ranging from 19.1 to 50.6 ms in PFPS patients and (with the exception of Brindle et al 2003) an earlier VMO activation time in healthy subjects ranging from 0.4ms to 57ms (Table 7.8) (Bolling et al, 2006; Cowan et al, 2001; Cowan et al, 2002a; Crossley et al, 2004,). The Brindle et al (2003) did not appear to agree with any other previous studies - VMO was delayed by 60.2ms in PFPS patients and 27.9ms in healthy controls but between group differences were not significant ($p>0.05$). However, the speed of the stepping task was not controlled and therefore direct comparisons cannot be made.

Table 7.8 summarises the results of step-down tasks and a normal speed from the current and previous studies. Whilst the results of the current study do not corroborate with previous findings a clear trend is emerging. In normal subjects VMO is recruited before VL and this trend is reversed in PFPS patients or as is the case in the current study there is no difference in recruitment time between VMO and VL.

Table 7.8: Time onset differences (ms) in VMO & VL in PFPS patients and healthy controls in step-down task. Negative VMO-VL values indicate a VMO delayed onset in comparison to VL.

STEP-DOWN TASK STUDIES	PFPS			CONTROL			MEAN difference (ms)	STATISTICAL SIGNIF. Between groups
	Time difference (ms)	SD	N	Time difference (ms)	SD	N		
Trigkas – current study	<1	75	63	16	23	63	15	YES
Bolling 2006	-50.6	82	14	57	54.7	14	107.5	YES
Brindle 2003	-60.2	35.3	16	-27.9	32.9	12	32.3	NO
Cowan 2001	-19.4	24.5	33	4	31	33	23.4	YES
Cowan 2002a	-19.7	15.8	10	12.9	27.7	12	32.6	YES
Crossley 2004	-19.1	17.1	47	0.37	5.7	18	19.4	YES

7.3.1.4 Time onset difference of PFPS patients vs. healthy subjects in step-down at fast speed (116 steps/minute)

During the eccentric phase of the step-down at fast speed, the order of recruitment of VM and VL now becomes reversed and VMO time onset was

delayed by 16 ms in comparison to VL in the PFPS patients. In contrast, the VMO was still activated earlier than VL by 10ms in healthy subjects (Table 7.9). The 26ms time onset difference between the groups during the step-down at a fast speed was highly significant ($p= 0.000$).

Table 7.9: VMO-VL time onset difference in PFPS patients & healthy subjects during step-down at fast speed (116 steps/min).

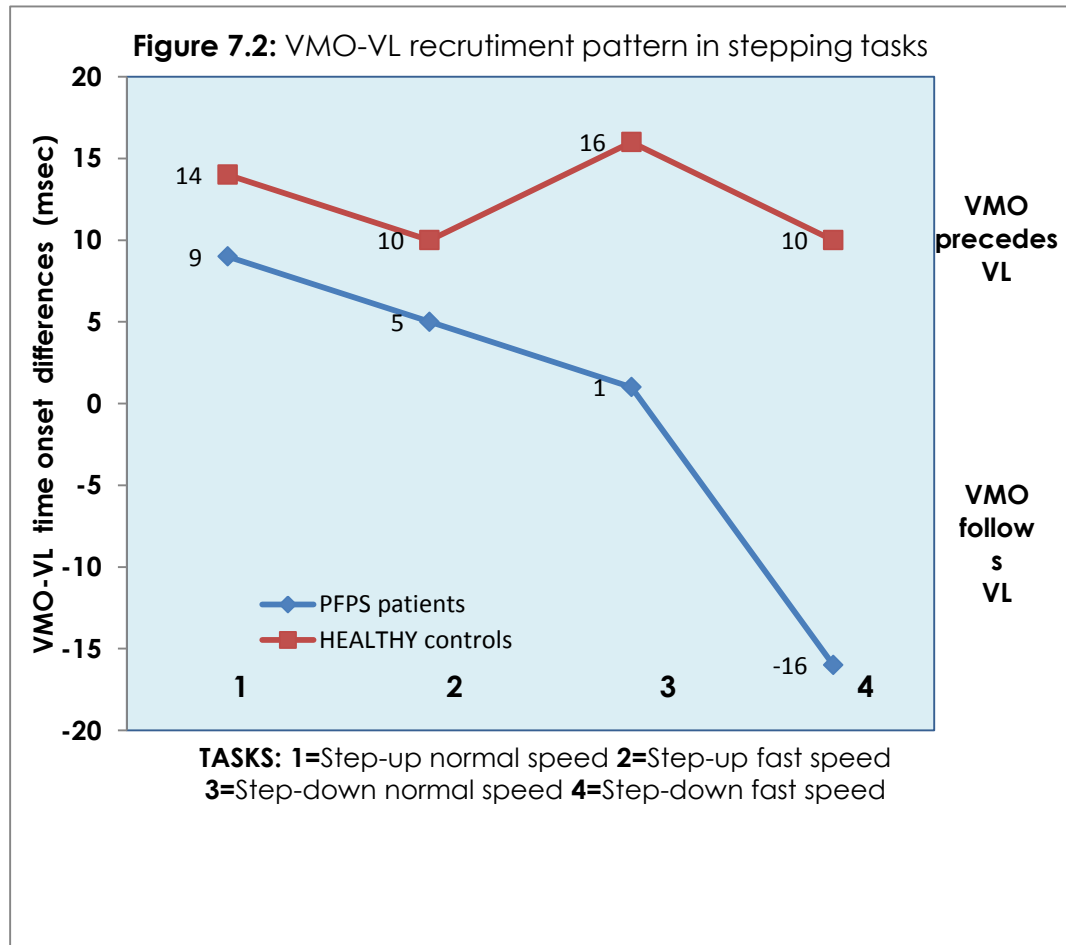
	Group	N	Mean difference between muscles (ms)	SD	t	df	p
VMO-VL Time Onset Difference	PFPS	63	-16	40	-4.310	124	0.000
	HEALTHY	63	10	26			

These results indicate that at a fast stepping down speed the normal order of recruitment ie VMO before VL is reversed in PFPS patients but not in healthy volunteers. Direct comparison of these results with previous data is not possible as existing studies either employed normal speed of step-down (96steps/min)(Bolling et al, 2006; Cowan et al, 2001; Cowan et al, 2002a; Crossley et al, 2004) or failed to control the stepping-down speed (Brindle et al, 2003; Sheehy et al, 1998; Powers et al, 1996). This study is the first to explore and report the employment of a fast speed stepping-down task in PFPS and a healthy population.

An overview of the results of all four stepping tasks employed in the current study reveals an interesting trend. A link appears to exist between the type of muscle contraction, the speed of execution of the task and the recruitment pattern of VMO-VL. It is clear that the recruitment pattern in healthy subjects is consistent across all four stepping tasks namely VMO activation precedes VL by an average 10ms-16ms depending on the specific task).

In contrast a different picture emerges for PFPS with a reversal of recruitment order becoming apparent in the fast step-down task. There is also a trend towards delayed VMO recruitment across all other functional tasks compared to healthy controls.

Figure 7.2 summarises the overall results and clearly depicts the changing trends in activation time between VMO and VL for different tasks and between different groups. The emerging patterns underlines the possible influence of the contraction type and the speed of execution of the task in the manifestation of the time onset muscle imbalance between VMO-VL in PFPS.



Whilst other studies have not considered a fast step down speed the existence of similar trends between step up and step down data confirms the current observations namely an increased delay in VMO recruitment time for step down compared to step up tasks (Bolling et al, 2006; Cowan et al, 2001; Cowan et al, 2002; Crossley et al 2004).

7.3.1.5 VMO-VL EMG amplitude differences (normalized RMS) of PFPS patients vs. healthy subjects in step-up at normal speed (96 steps/minute)

The within group muscle analysis revealed that the VMO EMG amplitude was significantly higher in comparison to the VL in both groups during the step-up at

normal speed (Table 7.10). In PFPS group the VMO amplitude was 427 mV significantly higher than the VL ($p=0.000$) and similarly in the healthy subjects the VMO amplitude activation was 406 mV significantly higher than the VL ($p=0.000$).

Table 7.10: VMO-VL EMG amplitude differences (normalized RMS) in PFPS patients & in healthy subjects during step-up at normal speed (96 steps/min).

	Mean RMS (mV)	N	Mean RMS difference between muscles (mV)	SD	t	df	p
1.PFPS	1.266						
VMO	839	63	427	423	8.004	62	0.000
VL							
2.Healthy	1.182						
VMO	777	63	406	689	4.673	62	0.000
VL							

The VMO-VL EMG amplitude results of this study during the step-up with the normal speed are in agreement with the study of Sheehy et al (1998) who have also found a VMO:VL ratio of 1.62 and 1.53 in healthy subjects and PFPS patients respectively but these between group differences were not significant ($p=0.31$). Similarly, Souza & Gross (1991) found a 1.26 & 1.18 VMO:VL ratio in patients with PFPS and healthy controls respectively, but this between group difference did not

reach statistical significance. In this study the step-up speed was slightly slower (92 steps/min) in comparison to the speed used in the current study (96 steps/min). One more study reported approximately similar results, but not significant during stair ascending, Powers et al (1996) also found a 30.7% VMO activity & 29.2% VL activity in PFPS patients, expressed as percentage of maximal muscle test. In the control healthy group found 27.5% for the VMO & 29.4% for the VL. In the study of Powers et al (1996) the speed of the stepping task was not controlled and the step height was 15 cm, therefore these results are not directly comparable to the current study.

In contrary, our results are different from the findings of the study of McClinton et al (2007) who reported VMO:VL ratio of 0.83. and 0.95 for the PFPS patients and healthy controls respectively during the step-up tasks with a normal speed of 96 steps/min but these differences were not significant between the groups ($p>0.05$).

7.3.1.6 VMO-VL EMG amplitude differences (normalized RMS) of PFPS patients vs. healthy subjects in step-up at fast speed (116 steps/minute)

In the PFPS group during concentric contraction of the step-up at fast speed the mean VMO EMG amplitude was greater than the VL and this difference was highly significant (Table 7.11, $p=0.000$). The VMO activity was also significantly higher in comparison to VL in the control group (Table 7.11, $p=0.000$).

Table 7.11: VMO-VL EMG amplitude differences (normalized RMS) in PFPS patients & in healthy during step-up at fast speed (116 steps/min).

	Mean RMS (mV)	N	Mean RMS difference between muscles (mV)	SD	t	df	p
1.PFPS							
VMO	1.453	63	504	450	8,886	62	0,000
VL	949						
2.Healthy							
VMO	1.424	63	467	856	4,324	62	0,000
VL	957						

The results from the fast speed step-up revealed a similar picture of VMO & VL amplitude as in the normal stepping task, the VMO had significantly higher amplitude than the VL in both groups. Additionally was clear that in both groups the VMO-VL amplitude was increased in comparison to the normal speed stepping task. Direct comparison of the current differences revealed during the fast speed stepping-up task with previous data is impossible. To our knowledge up to date, is the first time that a fast speed stepping-up task is employed in PFPS and healthy population measurement.

7.3.1.7 VMO-VL EMG amplitude differences (normalized RMS) of PFPS patients vs. healthy subjects in step-down at normal speed (96 steps/minute)

The VMO amplitude was significantly higher in comparison to VL in PFPS group during the step-down at normal speed (Table 7.12, $p=0.000$). A similar picture was also evident in the healthy controls with the VMO activated significantly higher than the VL (Table 7.12, $p=0.000$).

Table 7.12: VMO-VL EMG amplitude differences (normalised RMS) in PFPS patients & in healthy during step-down at normal speed (96 steps/min).

	Mean RMS (mV)	N	Mean RMS difference between muscles (mV)	SD	t	df	p
1.PFPS							
VMO	606	63	190	225	6.711	62	0.000
VL	416						
2.Healthy							
VMO	698	63	269	369	5.797	62	0.000
VL	429						

The results of stepping-down task of the current study are similar with the findings reported by Sheehy et al (1998), although they cannot be compared directly because of they did not control the speed of contraction as we did in the current study. Nevertheless, they also found higher VMO activation in comparison to VL in both groups. They reported a VMO:VL ratio of 1.359 for the healthy subjects and 1.147 for the PFPS patients, but these between group differences were not

significant ($p>0.05$). Similarly, Souza & Gross (1991) found a 1.13 & 1.15 VMO:VL ratio in patients with PFPS and healthy subjects respectively, but also this between group difference was not statistical significance. In this study, as was mentioned previously, the step-down speed was slightly slower (92 steps/min) in comparison to the speed used in the current study (96 steps/min). Furthermore, one more study reported approximately similar results, but not significant during step-down. Powers et al (1996) also found a 18.8% VMO activity & 12.4% VL activity in PFPS patients, expressed as percentage of maximal muscle test. In the control healthy group found 20.1% for the VMO & 19.6% for the VL. In the study of Powers et al (1996) the speed of the stepping task was not controlled and the step height was 15 cm, therefore these results are not directly comparable to the current study.

7.3.1.8 VMO-VL EMG amplitude differences (normalized RMS) of PFPS patients vs. healthy subjects in step-down at fast speed (96 steps/minute)

The EMG amplitude of the VMO was significantly higher than the VL in PFPS patients during the step-down at a fast speed (Table 7.13, $p= 0.000$). A similar picture was revealed in the control group, the VMO activation was also significantly greater in comparison to the VL (Table 7.13, $p= 0.000$).

Table 7.13: VMO-VL amplitude differences (normalized RMS) in PFPS patients & in healthy during step-down at fast speed (116 steps/min).

	Mean RMS (mV)	N	Mean RMS difference between muscles (mV)	SD	t	df	p
1.PFPS							
VMO	703	63	222	280	6,265	62	0,000
VL	482						
2.Healthy							
VMO	835	63	319	438	5,780	62	0,000
VL	516						

Direct comparison of the current differences revealed during the fast speed stepping-up task with previous data is impossible. To our knowledge up to date, is the first time that a fast speed stepping-down task is employed in PFPS and healthy population measurement.

An overview of the results of all four stepping tasks of the current study reveals that the VMO EMG amplitude is consistently higher than the VL in all four stepping tasks and this is evident in both groups. These differences between muscles are significant in patients and in healthy controls ($p=0.000$). Furthermore it is obvious that the type of the contraction and the speed of execution of the stepping task have relatively small influence in the intensity of the VMO & VL muscular

performance. Therefore, it is clear that the VMO-VL muscle imbalance in terms of amplitude electromyographic activity cannot be established from the results of the functional tasks of this study.

7.3.2 Experimental tasks

7.3.2.1 Time onset differences in PFPS patients vs. healthy subjects in knee extension isokinetic concentric contraction (from 90° - 0° at 90°/sec angular velocity)

In the knee extension isokinetic concentric contraction, the VMO was activated 1ms earlier in comparison to the VL in the PFPS patients, and was 7ms earlier activated than VL in healthy subjects respectively. The time onset difference of 6ms between the groups was significant (Table 7.14, $p=0,01$).

Table 7.14: VMO-VL time onset difference in PFPS patients & healthy subjects during isokinetic concentric contraction of knee extension from 90°-0° at angular velocity 90°/sec.

	Group	N	Mean difference between muscles (ms)	SD	<i>t</i>	df	p
VMO-VL Time Onset Difference	PFPS	63	1	16	-2.310	124	0.01
	HEALTHY	63	7	12			

These results are approximately similar with the activation pattern recorded in previous study of Owing & Grabiner (2002). Although direct comparison is not possible because they used a considerably slower angular velocity (15 degrees/sec) than the one we used in the current study (90 degrees/sec), they did not identified a VMO delayed onset in PFPS & healthy subjects. Our result differs from the study of Cesarelli et al (1999) who reported significant VMO delayed onset ($p=0,05$) during a same isokinetic procedure, range of movement from 90° of knee flexion to full extension with the same angular velocity of 90°/sec but employed a different onset detection method.

7.3.2.2 Time onset differences of PFPS patients vs. healthy subjects in knee extension isokinetic eccentric contraction from (from 0° - 90° at 90°/sec angular velocity)

The analysis of the time onset differences revealed a muscle imbalance of the VMO-VL activation pattern in the PFPS patients during the knee extension isokinetic eccentric contraction. The VMO time onset was marginally delayed (time onset difference < 1ms)* in comparison to the VL in the PFPS patients. In contrary, the VMO was 12ms earlier activated than VL in the healthy subjects (Table 7.15). The time onset difference of 11ms between the groups was statistical significant ($p= 0.000$).

Table 7.15: VMO-VL time onset difference in PFPS patients & healthy subjects during isokinetic eccentric contraction of knee extension from 90°-0° at angular velocity 90°/sec.

	Group	N	Mean difference between muscles (ms)	SD	t	df	p
VMO-VL Time Onset Difference	PFPS	63	< -1	21	-3.630	124	0.000
	HEALTHY	63	12	17			

It is interesting to underline that during the isokinetic measurements it is observed a similar influence of the muscular contraction type on the recruitment patterns of the VMO-VL which has been discussed in the previous section of this chapter. The isokinetic concentric contraction did not cause any alteration to VMO-VL time onset but the eccentric contraction seems to alter pathologically the recruitment pattern of the quadriceps. To our knowledge up to date, do not exist any similar result obtained by isokinetic eccentric measurements. Our finding is contradictory with the results of Owing & Grabiner (2002) who found an earlier activation of the VMO relatively to VL in PFPS patients.

7.3.2.3 Time onset differences of PFPS patients vs. healthy subjects in isokinetic knee extension 1st isometric contraction (fresh state) (at 75° of knee flexion)

In the knee extension isokinetic 1st isometric contraction, the VMO was activated 4ms earlier in comparison to the VL in the PFPS patients. In the healthy controls the

VMO was 7ms earlier activated in comparison to VL (Table 7.16). The VMO-VL time onset difference between the groups was not significant ($p = 0.10$).

Table 7.16: VMO-VL time onset difference in PFPS patients & healthy subjects during isokinetic knee extension 1st isometric contraction at 75° of knee flexion.

	Group	N	Mean difference between muscles (ms)	SD	<i>t</i>	df	p
VMO-VL Time Onset Difference	PFPS	63	4	17	-1.268	124	0.10
	HEALTHY	63	7	15			

To our knowledge up to date there is not available any previous similar to our isokinetic isometric measurement, therefore comparison to previous data is not possible.

7.3.2.4 VMO-VL EMG amplitude differences (normalized RMS) of PFPS patients vs. healthy subjects in isokinetic concentric contraction of the knee extension from 90°-0° at angular velocity 90°/sec.

The normalised RMS EMG amplitude of the VMO was significantly greater than VL in the PFPS subjects during the isokinetic concentric contraction (Table 7.17, $p=0.013$). Similar was the picture in healthy controls, the VMO EMG amplitude was also significantly higher than the VL (Table 7.17, $p=0.015$).

Table 7.17: VMO-VL RMS EMG amplitude in patients & in healthy during isokinetic concentric contraction of the knee extension from 90°-0° at angular velocity 90°/sec.

	Mean RMS (mV)	N	Mean RMS difference between muscles (mV)	SD	t	df	p
1.PFPS							
VMO	1.351	63	104	322	2.561	62	0.013
VL	1.247						
2.Healthy							
VMO	1.232	63	97	305	2.508	62	0.015
VL	1.135						

During the isokinetic concentric contraction no muscle imbalance was detected, the normalized VMO RMS EMG amplitude was significantly higher than the VL in both patients and healthy controls. In contrast to our results, Tang et al (2001) found a 0,831 VMO-VL ratio in PFPS patients and 0,959 VMO-VL ratio in controls. Cesarelli et al (1999) also reported significant lower VMO EMG activity relatively to VL during a same to ours isokinetic procedure. Additionally, Santos et (2008), using a different experimental protocol, found a lower but non-significant EMG amplitude in the VMO during isokinetic concentric contraction from 60° - 0° at 30

degrees/sec angular velocity in comparison to the Vastus Lateralis Longus (VLL) and Vastus Lateralis Oblique (VLO).

7.3.2.5 VMO-VL EMG amplitude differences (normalized RMS) of PFPS patients vs. healthy subjects in isokinetic eccentric contraction of the knee extension from 0°-90° at angular velocity 90°/sec.

There was no significant difference between normalized VMO-VL EMG amplitude in the PFPS patients (Table 7.18, $p \leq 0.083$). Similarly, the normalised VMO EMG amplitude was not also significantly higher in comparison to VL in the healthy controls. (Table 7.18, $p = 0.341$).

Table 7.18: VMO-VL EMG amplitude differences (normalized RMS) in patients & in healthy during isokinetic eccentric contraction of the knee extension from 90°-0° at angular velocity 90°/sec.

	Mean RMS (mV)	N	Mean RMS difference between muscles (mV)	SD	t	df	p
1.PFPS							
VMO	1.234	63	70	315	1.760	62	0,083
VL	1.164						
2.Healthy							
VMO	1.153	63	32	268	0,959	62	0,341
VL	1.120						

The VMO-VL EMG activation levels during the knee extension isokinetic eccentric contraction from 0°-90° at angular velocity of 90°/sec revealed slightly different picture relatively to isokinetic concentric contraction. Although did not perform analysis, it was clear that the VMO-VL amplitude was reduced during the eccentric contraction in relation to the concentric in both groups. The results of the current study are similar with the study of Tang et al (2001). They reported a 1.105 VMO-VL ratio in PFPS patients and 1.259 ratio in the controls, but the difference was not significant.

In contrast, our results differ from the study of Owings & Grabiner (2002) who reported significantly lower activity of the VMO in comparison to VL in PFPS patients. However, direct comparison is not possible because they used a considerably slower angular velocity (15 degrees/sec) than the one we used in the current study (90 degrees/sec)

7.3.2.6 VMO-VL EMG amplitude differences (normalized RMS) of PFPS patients vs. healthy subjects in isokinetic knee extension 1st isometric contraction (fresh state) (at 75° of knee flexion)

The normalized VMO EMG amplitude of the PFPS patients was not significantly greater than normalised VL amplitude (Table 7.19, $p=0.354$). In contrast, the normalised VMO EMG amplitude of the healthy controls was higher in comparison to the normalized VL amplitude (Table 7.19, $p=0.009$).

Table 7.19: VMO-VL RMS EMG amplitude in patients & in healthy during isokinetic knee extension 1st isometric contraction at 75° of knee flexion. Mean RMS & mean RMS difference between muscles is in mV.

	Mean RMS (mV)	N	Mean RMS difference between muscles (mV)	SD	t	df	p
1.PFPS							
VMO	580	63	17	144	934	62	0.354
VL	563						
2.Healthy							
VMO	418	63	35	105	2.694	62	0.009
VL	382						

The results of the current study are similar to the one that Møller et al (1986) reported. They identified slightly higher but no significant VMO activity levels in comparison to VL in PFPS patients during isometric contraction at 60° of knee flexion.

Chapter 8

Study II: Measures of clinical
symptoms

8. STUDY II: MEASURES OF CLINICAL SYMPTOMS

The current chapter presents and discusses the measures of clinical symptoms subdivided into three areas of assessment namely I) pain & function, II) muscular flexibility and III) lower limb biomechanics. These clinical characteristics have been theoretically & experimentally proposed as substantial factors associated to the aetio-pathogenesis of the PFPS (Piva et al, 2005, Thomeé et al, 1995).

This chapter also considers correlations between the measures of clinical symptoms and muscle function. Associations between some aspects of the clinical symptoms, such as pain, function, muscular impairments and biomechanical characteristics, although not extensively, have been previously reported in the literature (Piva et al, 2009). However, in relation to current knowledge, association between measures of clinical symptoms and measures of muscle function in patients with PFPS has not been explored previously.

8.1 Assessment of pain and function

8.1.1 Overall assessment of pain, duration of symptoms and function

Table 8.1 summarises the results of the visual analog scale (VAS) data for worse (VAS-W) and usual (VAS-U) pain during the past week of the measurements, the duration of symptoms since the onset of PFPS and the Anterior Knee Pain Scale score for the PFPS patients recruited into this study. The average level of pain for the PFPS patients was 7.2 (± 1.5) and 4.1 (± 4.1) for VAS worse pain and the VAS

usual pain respectively. The average time of the duration of symptoms was 47.9 (± 43.9) months & the Anterior Knee Pain Scale score was 74 (± 10).

Table 8.1: Total PFPS group results of the VAS-worse - VAS-usual, duration of symptoms in months & Anterior Knee Pain Scale (Kujala score).

	N	Range	Min	Max	Mean	SD
VAS worse total PFPS patients group	63	7	3	10	7,2	1,5
VAS usual total PFPS patients group	63	5	3	8	4,1	1,2
Duration of Symptoms - Time since onset of symptoms in months	63	260	4	264	47,8	43,9
Anterior Knee Pain Scale Kujala score	63	47	42	89	74	10

The average level of pain experienced by the PFPS patients in the current study was 7,2 ($\pm 1,5$) and 4,1 ($\pm 1,2$) for VAS worse pain and the VAS usual pain respectively. Similar levels of pain were recorded by Cowan et al (2001), who reported 7,1 ($\pm 1,6$) for VAS worse pain and 4,3 ($\pm 1,2$) for VAS usual pain. In another study by the same research group (Cowan et al, 2002) the reported level of the usual pain (7,2 $\pm 1,5$) was higher than in this current and their earlier study and was more usually associated with worse pain. Patil et al (2011) have also reported a VAS pain score of 5,6 ($\pm 2,1$) but did not clarify whether the pain level referred to was the usual or worse pain experienced by the PFPS patients. Similar results for usual level of pain were also reported by Piva et al (2005, 2006) who found average pain score of 3,9 ($\pm 2,2$) and 3,9 ($\pm 1,9$) respectively. In a recent study of Piva et al (2009) the level of the reported worse pain was 5,6 ($\pm 2,4$) and the level

of the usual pain was 3,6 ($\pm 2,1$). Lower levels of pain (mean 3.9) was also found by Ott et al (2011).

Few studies report duration of symptoms but this study revealed a average duration of 47,8 ($\pm 43,9$) months. Of the few studies that did report this data, Cowan et al (2001) reported similar results 42,2 ($\pm 49,9$) months mean time since the onset of symptoms. Similarly, On et al (2004), now considering years and not months, reported average duration of 3,46 ($\pm 1,9$) years. Considerably lower duration of knee pain was reported by Patil and associates, with mean duration of 7,3 ($\pm 1,1$) months, and also by Cowan et al (2002) with 10,9 ($\pm 22,3$) months. Overall, however it appears that the results from the current study for duration of symptoms are consistent with that presented in the literature.

The AKPS score is represented as an absolutely value from 0-100, and higher values indicating better functional status (Kujala et al, 1993). The average value reported in this study was 74 with quite a tight standard deviation of ± 10 . This would suggest that despite their AKP patients had a relatively high level of function. High levels of function were also reported by Liebsteiner et al (2008) who showed a median score of 85 in AKPS-Kujala scale and by Ott et al (2011) who reported a mean 81,7 ($\pm 10,9$) for the PFPS patients with high pain and a 84,1 ($\pm 9,2$) score for the patients with low pain.

8.1.2 Correlations of the pain level with measures of muscle function

The results of the correlations between the level of pain (VAS-usual & VAS worse) and the VMO-VL time onset difference in PFPS patients, revealed that there was no statistically significant association between the pain and muscle function (Table 8.2) Nevertheless it is evident that a tendency towards a weak negative correlation exists. In seven out of eight correlations there is a weak negative correlation ($r = -0.05$ - 0.17) between the level of pain and the measures of muscle function.

Table 8.2: Correlations between the level of pain (VAS-Usual & VAS-Worse) of PFPS patients and the VMO-VL time onset difference of the PFPS patients during functional tasks.

Correlations	Mean Time Onset Difference in msec	Pain Level	N	r	p
Step-Up with Normal speed – VAS-usual	9	4,1	63	-0,05	0,68
Step-Up with Normal speed – VAS-worse	9	7,2	63	-0.14	0,27
Step-Down with Normal speed – VAS-usual	<1	4,1	63	-0,17	0,27
Step-Down with Normal speed – VAS-worse	<1	7,2	63	-0,06	0,66
Step-Up with Fast speed – VAS-usual	5	4,1	63	-0,09	0,48
Step-Up with Fast speed – VAS-worse	5	7,2	63	0,08	0,55
Step-Down with Fast speed – VAS-usual	-16	4,1	63	-0,02	0,90
Step-Down with Fast speed – VAS-worse	-16	7,2	63	-0,11	0,40

This information is depicted in the form of scatter plots (Figures 8.1 – 8.8).

Figure 8.1: Scatterplot of the correlation of the VAS-usual pain with the VMO-VL time onset difference of the PFPS patients during the step-up at normal speed.

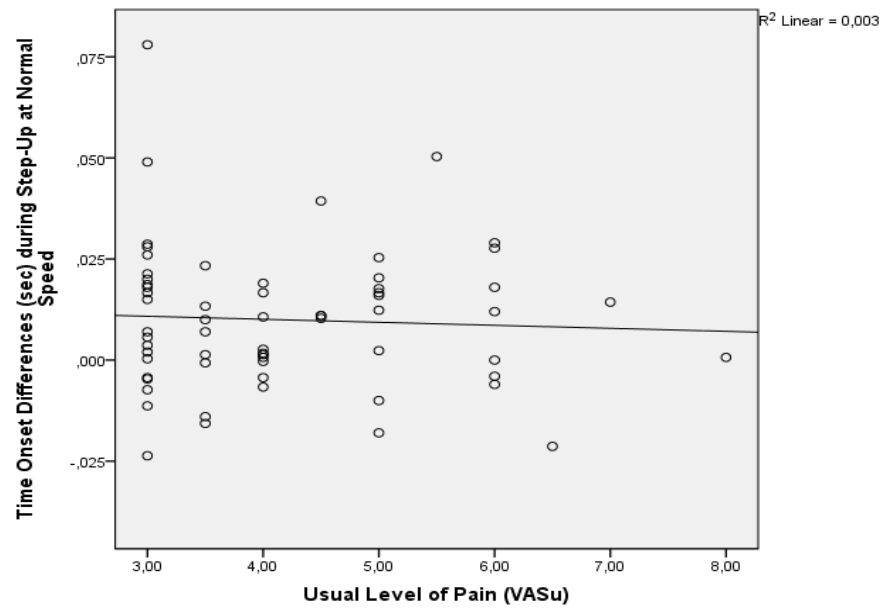


Figure 8.2: Scatterplot of the correlation of the VAS-worse pain with the VMO-VL time onset difference of the PFPS patients during the step-up at normal speed.

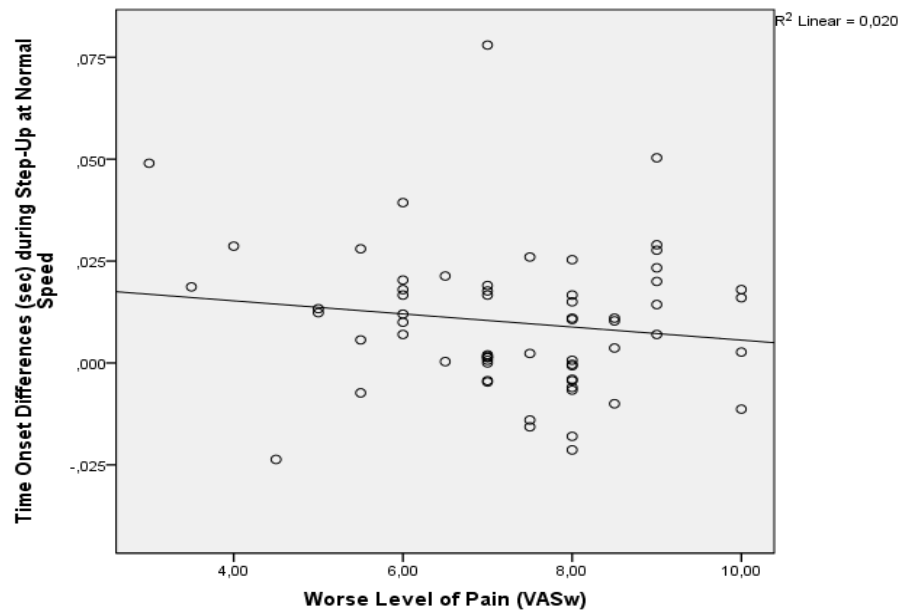


Figure 8.3: Scatterplot of the correlation of the VAS-usual pain with the VMO-VL time onset difference of the PFPS patients during the step-down at normal speed.

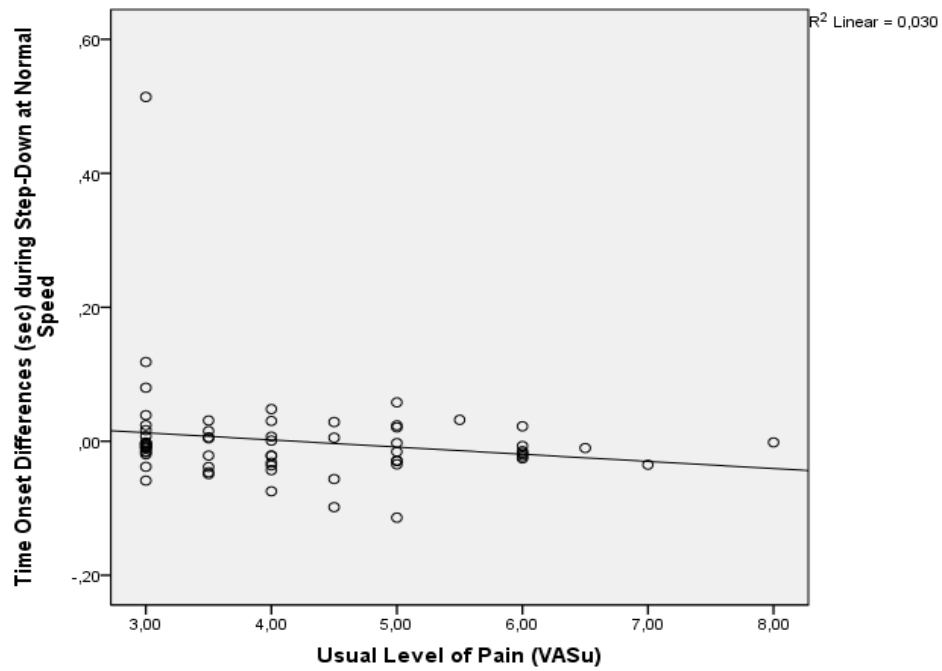


Figure 8.4: Scatterplot of the correlation of the VAS-worse pain with the VMO-VL time onset difference of the PFPS patients during the step-down at normal speed

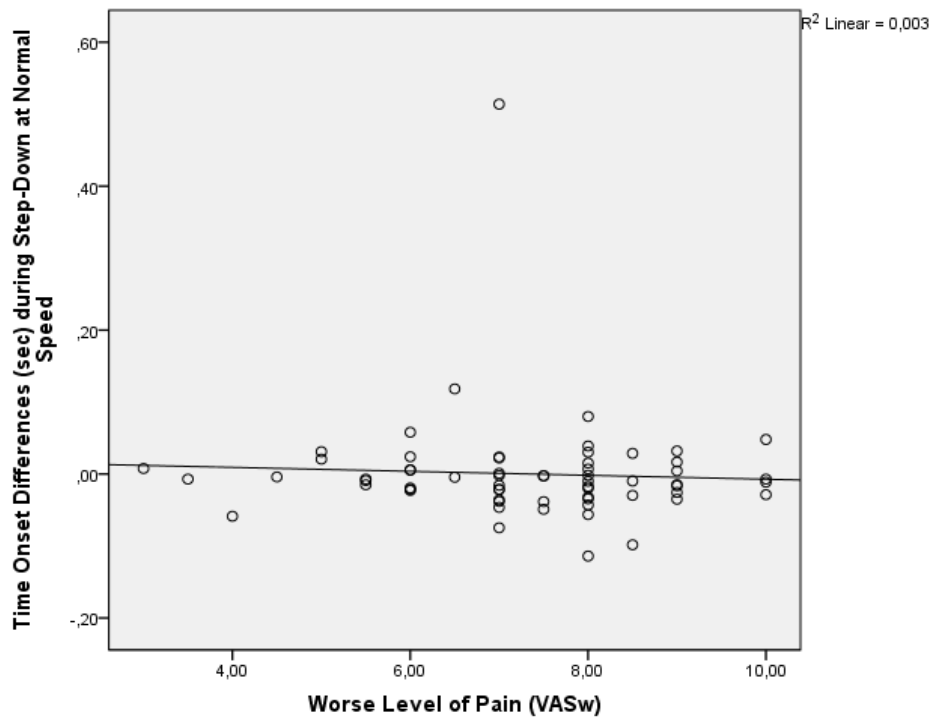


Figure 8.5: Scatterplot of the correlation of the VAS-usual pain with the VMO-VL time onset difference of the PFPS patients during the step-up at fast speed.

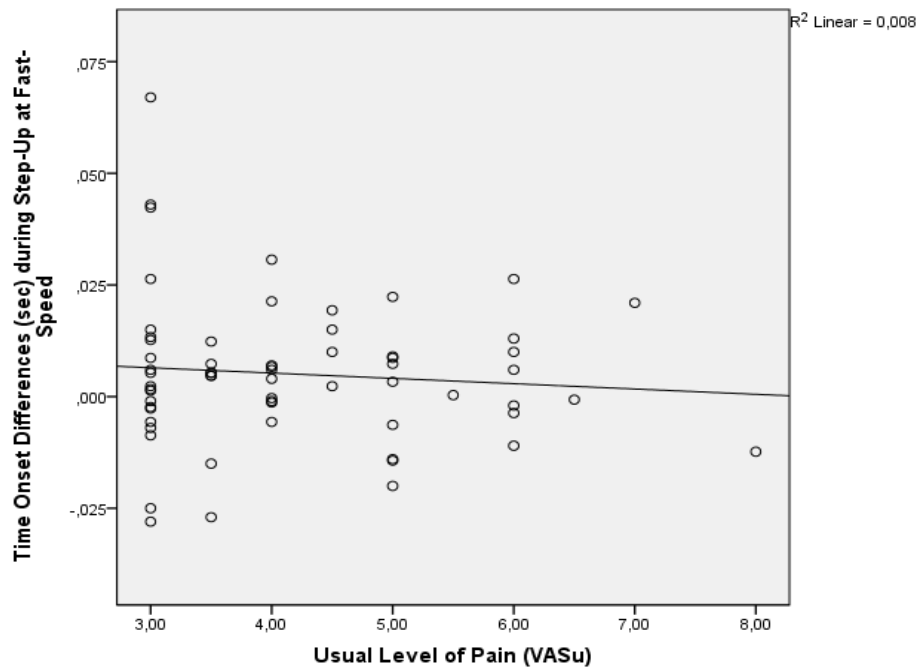


Figure 8.6: Scatterplot of the correlation of the VAS-worse pain with the VMO-VL time onset difference of the PFPS patients during the step-up at fast speed.

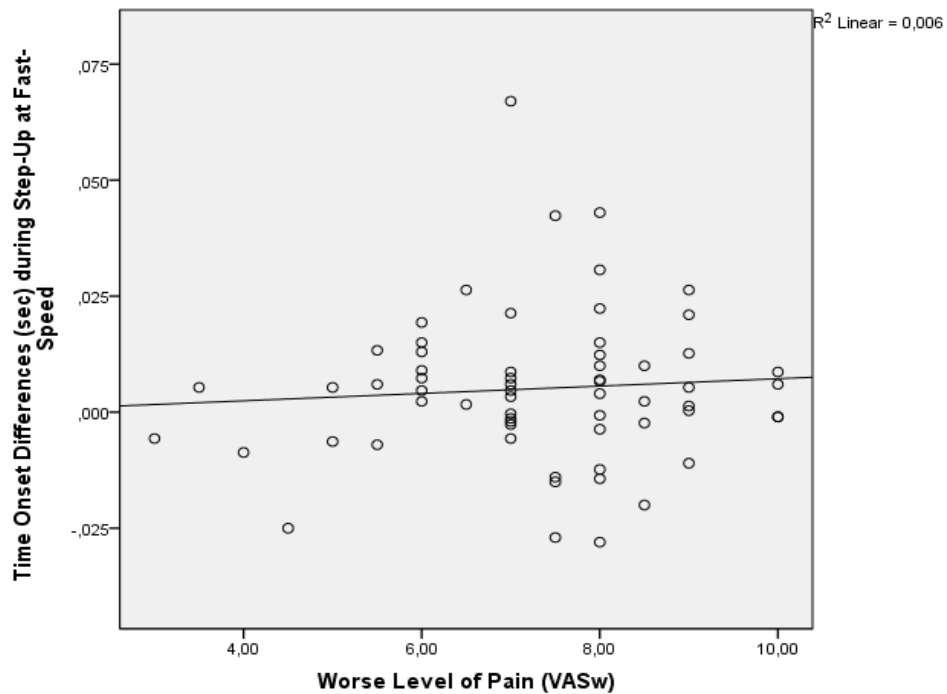


Figure 8.7: Scatterplot of the correlation of the VAS-usual with the VMO-VL time onset difference of the PFPS patients during the step-down at fast speed.

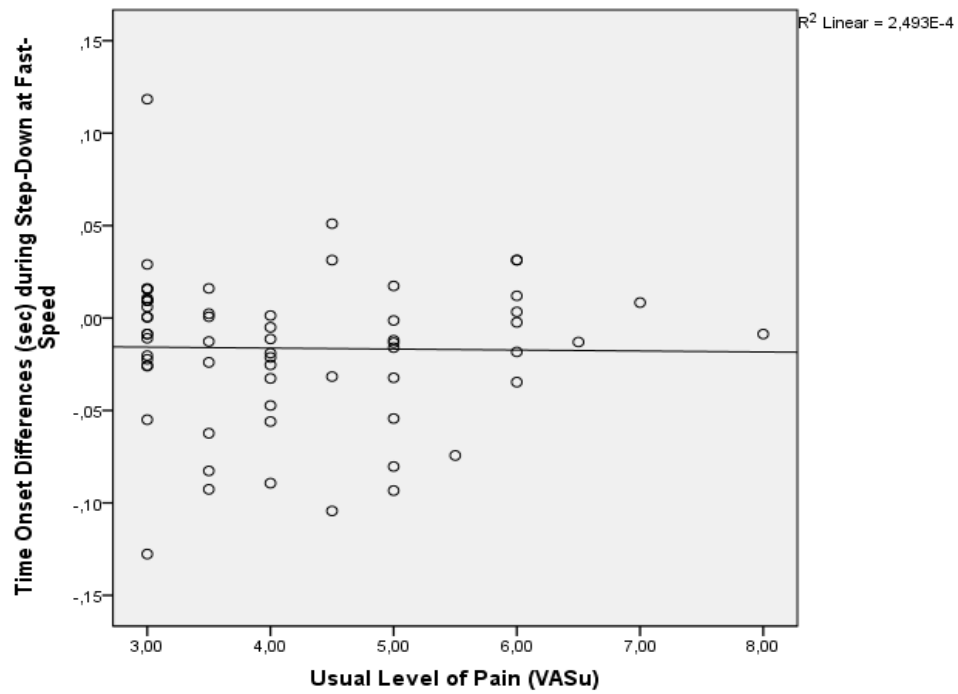
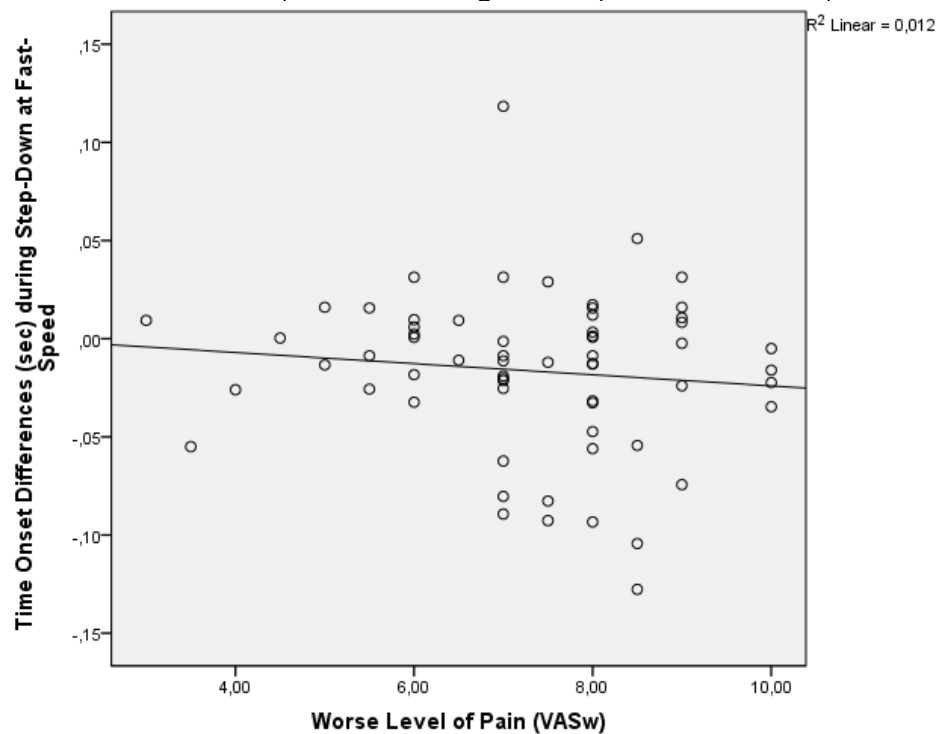


Figure 8.8: Scatterplot of the correlation of the VAS-worse with the VMO-VL time onset difference of the PFPS patients during the step-down at fast speed.



This study is the first attempt to correlate the level of pain (VAS usual and worse), the duration of symptoms and the knee function of the PFPS patients with measures of muscle function.

The correlations revealed that there is no direct association between pain level and VMO-VL time onset differences. Nevertheless, it is evident that a tendency exists towards a negative association between the VMO-VL time onset difference and the level of pain. This means that there is a tendency towards higher levels of pain level (VAS usual & worse being associated with a delayed onset in recruitment of VMO). What is not clear is whether the presence of high levels of pain in PFPS patients triggers a VMO-VL muscle imbalance in terms of activation time or vice versa. Furthermore the weak non significant correlation suggests any inferences regarding a relationship between pain and activation time should be interpreted with caution.

8.1.3 Correlations of the duration of symptoms with measures of muscle function

Table 8.3 summarises the results of the correlations between the duration of the symptoms and the VMO-VL time onset difference in PFPS patients during the functional stepping tasks. The correlation of the duration of symptoms with the normal speed of the stepping task revealed a weak and not significant positive association with Pearson correlation coefficient of $r=0.08$ ($p=0.49$) and $r=0.04$ ($p=0.75$) for the step-up and step-down respectively. A weak negative but also non-significant correlation was evident between the fast speed stepping task and duration of symptoms. For the step-up at fast speed the Pearson correlation coefficient was $r=-0.08$ ($p=0.53$) and for the step-down at a fast speed the Pearson correlation coefficient was $r=-0.17$ ($p=0.18$).

Table 8.3: Correlations between the duration of symptoms of PFPS patients and the VMO-VL time onset difference of the PFPS patients during functional tasks.

Correlations	Mean Time Onset Difference in msec	Mean Duration of symptoms in months	N	<i>r</i>	<i>p</i>
Duration of symptoms - Step-Up with Normal speed	9	47,8	63	0,08	0,49
Duration of symptoms - Step-Down with Normal speed	<1	47,8	63	0,04	0,75
Duration of symptoms - Step-Up with Fast speed	5	47,8	63	-0,08	0,53
Duration of symptoms - Step-Down with Fast speed	-16	47,8	63	-0,17	0,18

This information is depicted in the form of scatter plots (Figures: 8.9-8.12).

Figure 8.9: Scatterplot of the correlation between the duration of symptoms (in months) and the VMO-VL time onset difference (in seconds) of the PFPS patients during the step-up at normal speed.

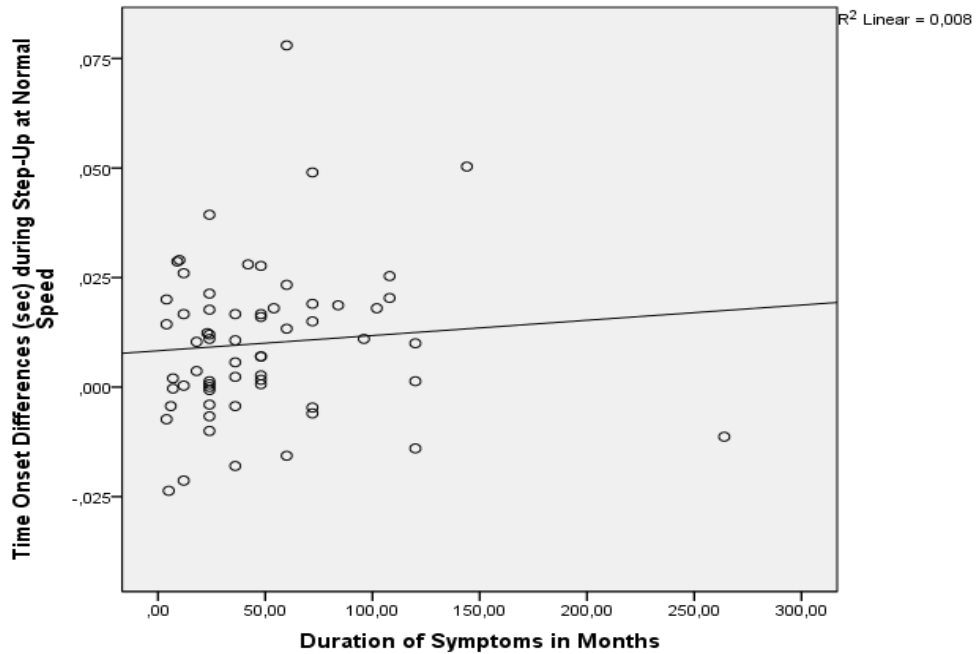


Figure 8.10: Scatterplot of the correlation between the duration of symptoms (in months) and the VMO-VL time onset difference (in seconds) of the PFPS patients during the step-down at normal speed.

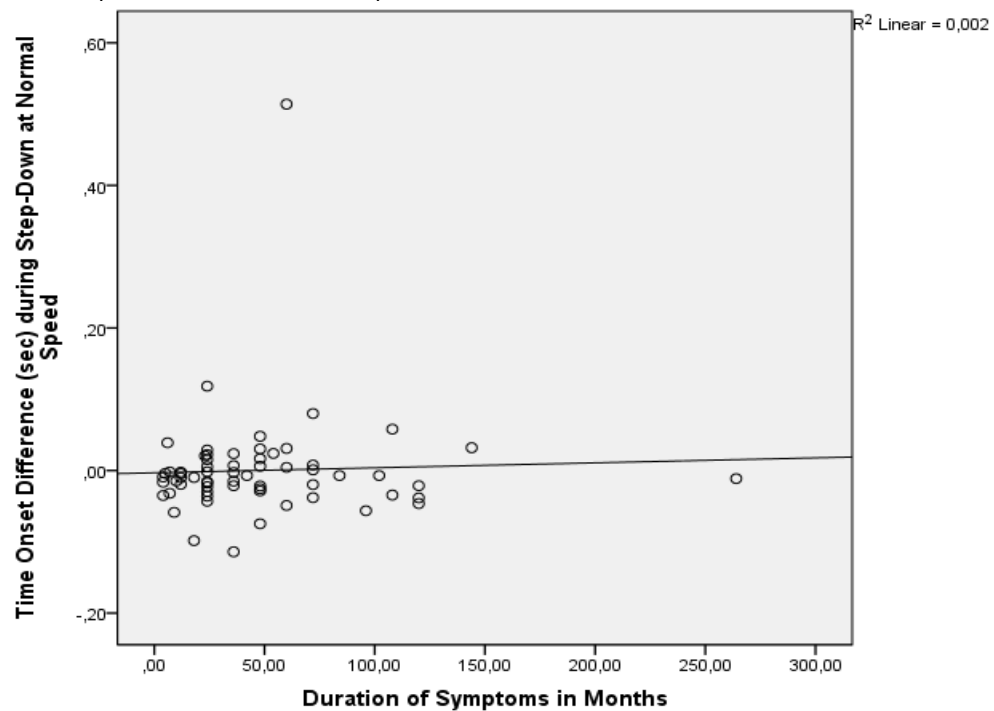


Figure 8.11: Scatterplot of the correlation between the duration of symptoms (in months) and the VMO-VL time onset difference (in seconds) of the PFPS patients during the step-up at fast speed.

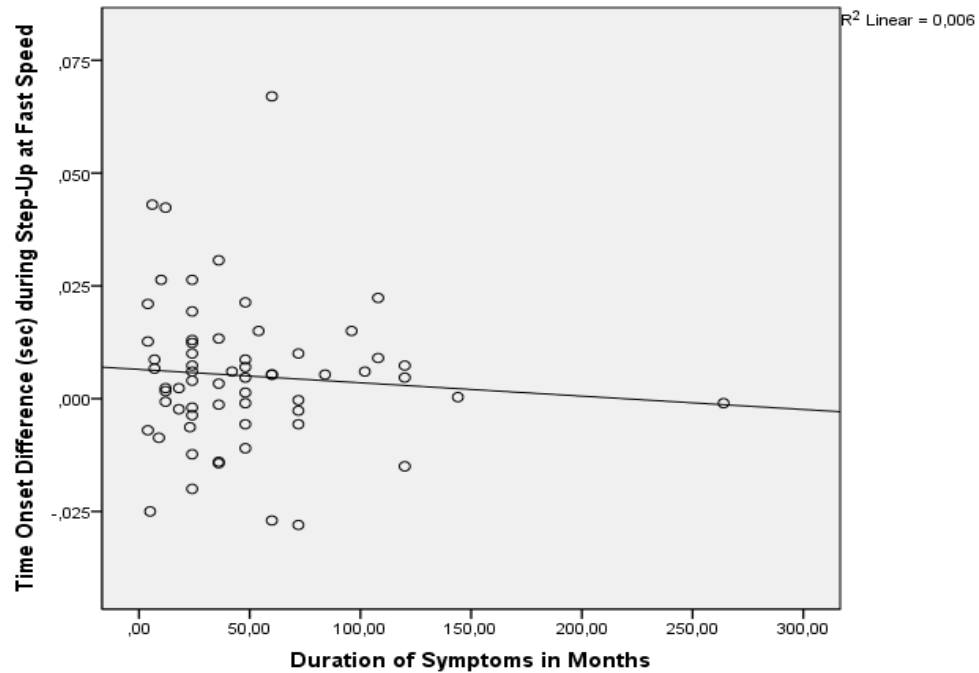
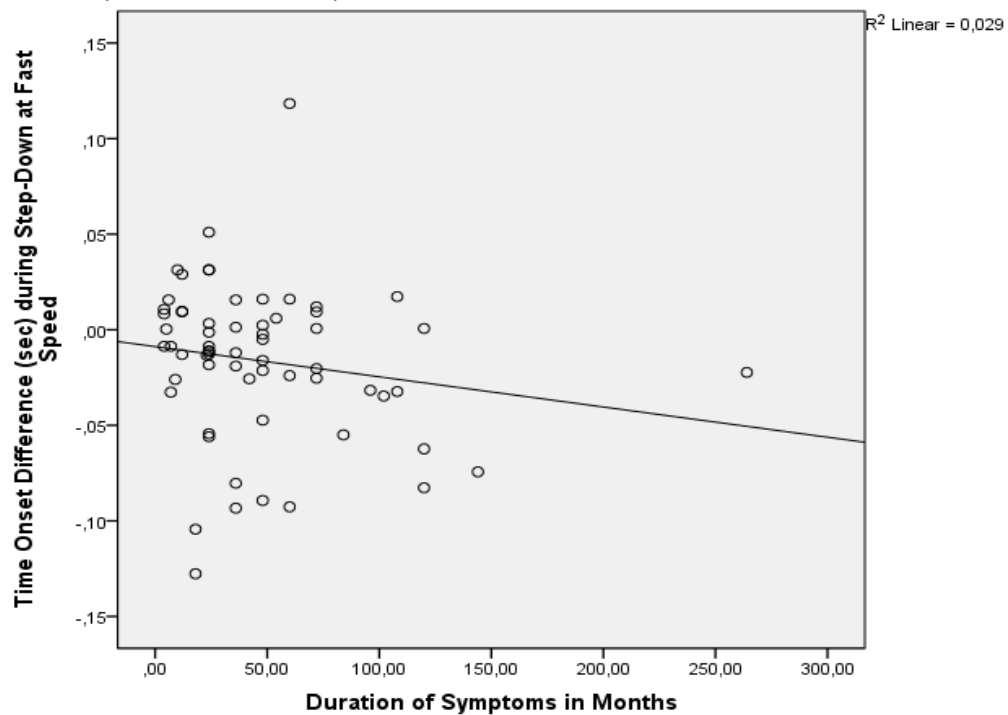


Figure 8.12: Scatterplot of the correlation between the duration of symptoms (in months) and the VMO-VL time onset difference (in seconds) of the PFPS patients during the step-down at fast speed.



The correlation between the duration of symptoms and the measures of muscle function revealed a mixed picture. A weak and non-significant positive correlation was apparent between the duration of symptoms and the time onset difference during the stepping task at normal speed. This finding means that chronicity of the PFPS has a minimal effect on the VMO-VL muscle imbalance during the functional tasks when are executed with normal speed. This relationship is reversed when the functional tasks are performed at fast speed. The chronicity of the PFPS symptoms seems to cause a tendency for VMO-VL muscle imbalance.

8.1.4 Correlations of the Anterior Knee Pain Scale (AKPS) score with measures of muscle function

The results of the correlational analysis revealed that the score of the current knee function (AKPS-Kujala score) was significantly positively associated with the VMO-VL time onset difference during the step-down at fast speed (Table 8.4, $r=0.27$, $p=0.03$). A positive association was also observed in the remaining stepping tasks but did not reach statistical significance. In the step-up & down at normal speed the Pearson correlation coefficient was respectively $r=0.11$ ($p=0.39$) and $r=0.19$ ($p=0.12$). Similarly, the time onset difference of the step-up at fast speed was positively correlated to the AKPS score, but without statistical significance (Table 8.4, $r=0.23$, $p=0.06$).

Table 8.4: Correlations between the Anterior Knee Pain Scale score (Kujala) of PFPS patients and the VMO-VL time onset difference of the PFPS patients during functional tasks.

Correlations	Mean Time Onset Difference in msec	Mean AKPS score (Kujala)	N	R	p
AKPS score - Step-Up at Normal speed	9	74	63	0.11	0.39
AKPS score - Step-Down at Normal speed	<1	74	63	0.19	0.12
AKPS score - Step-Up at Fast speed	5	74	63	0.23	0.06
AKPS score - Step-Down at Fast speed	-16	74	63	0.27	0.03

In the following pages are presented the scatterplots with the regression lines of the correlations between the Anterior Knee Pain scale and the measures of muscle function during the stepping tasks (figures 8.13-8.16).

Figure 8.13: Scatterplot of the correlation between the Anterior Knee Pain score (as an absolute value from 0-100) and the VMO-VL time onset difference (in seconds) of the PFPS patients during the step-up at normal speed.

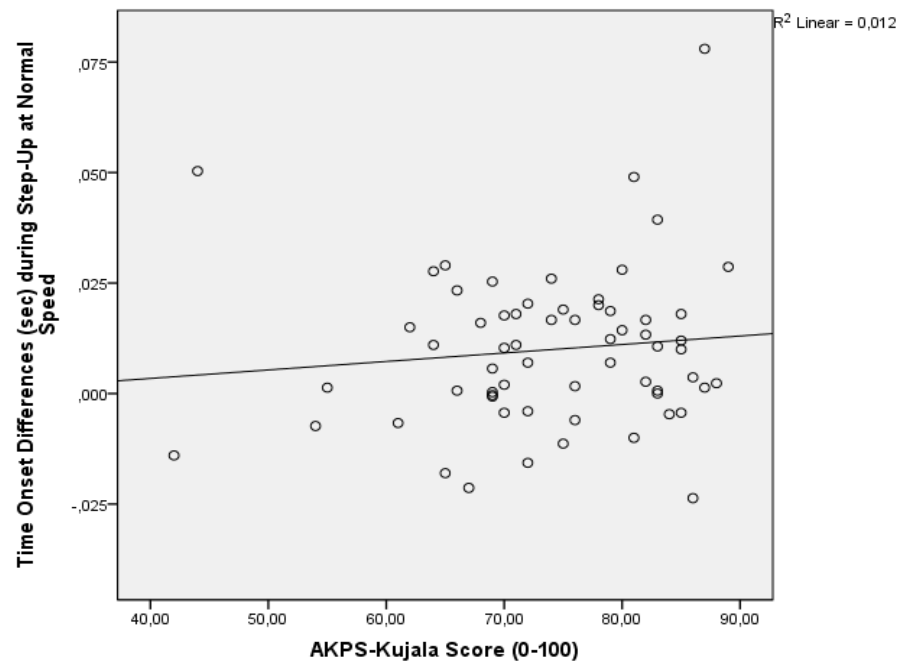


Figure 8.14: Scatterplot of the correlation between the Anterior Knee Pain score (as an absolute value from 0-100) and the VMO-VL time onset difference (in seconds) of the PFPS patients during the step-down at normal speed.

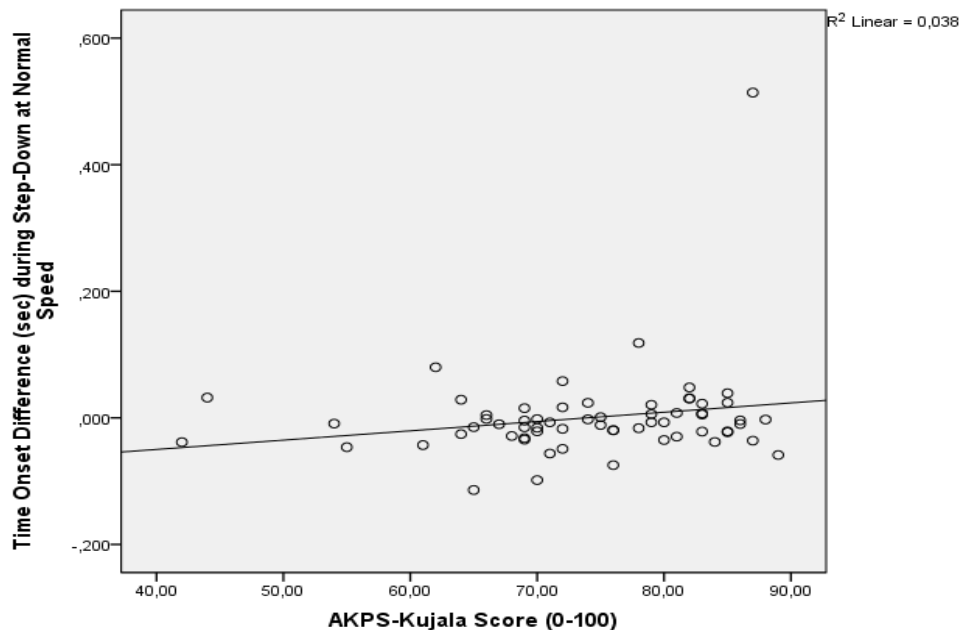


Figure 8.15: Scatterplot of the correlation between the Anterior Knee Pain score (as an absolute value from 0-100) and the VMO-VL time onset difference (in seconds) of the PFPS patients during the step-up at fast speed.

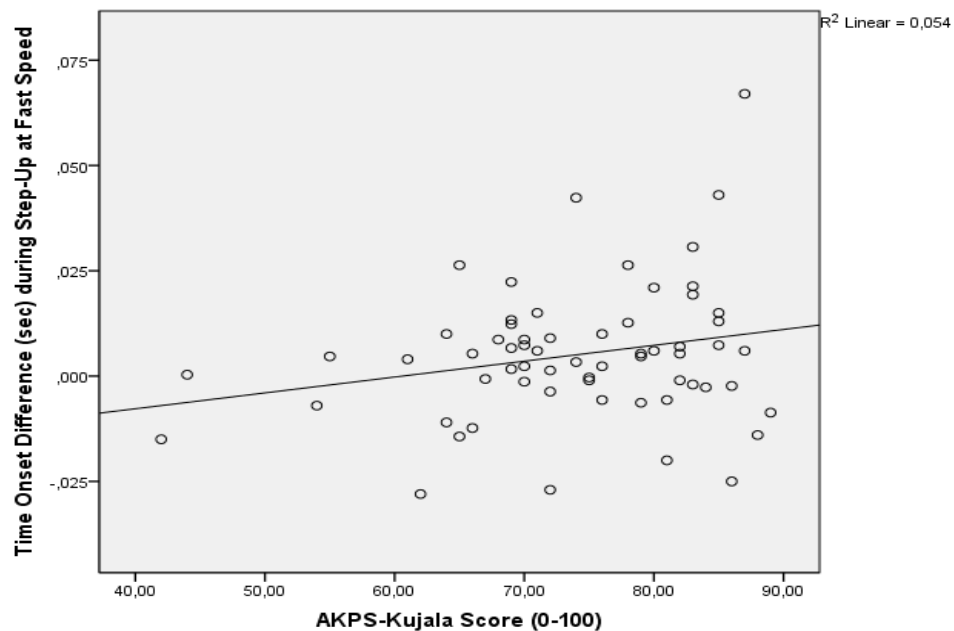
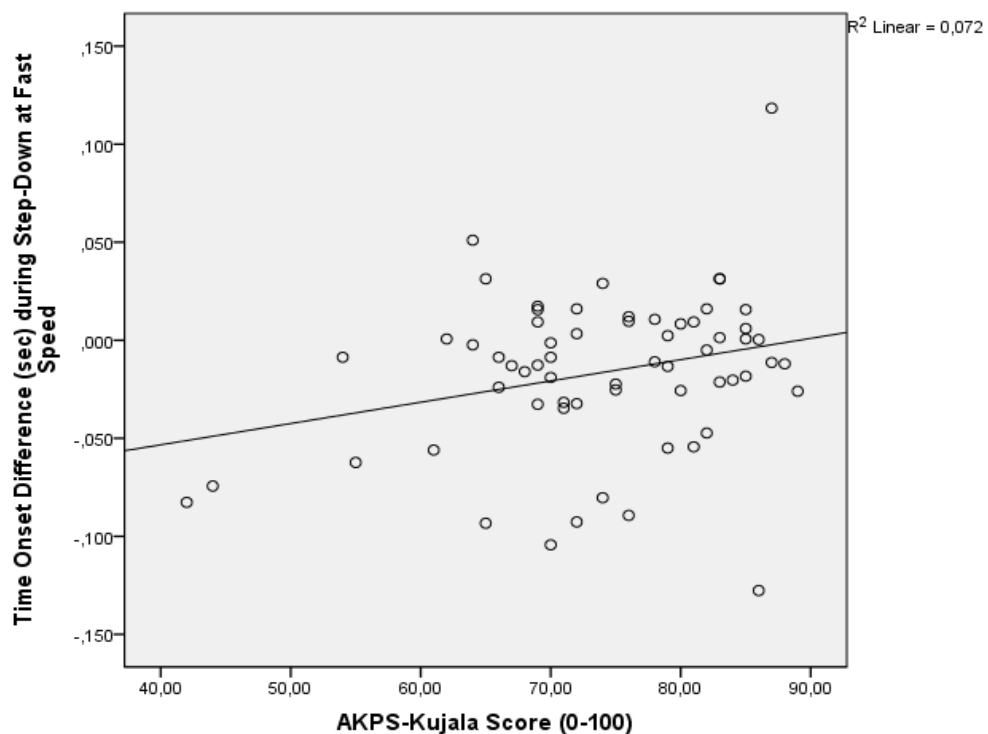


Figure 8.16: Scatterplot of the correlation between the Anterior Knee Pain score (as an absolute value from 0-100) and the VMO-VL time onset difference (in seconds) of the PFPS patients during the step-down at fast speed.



A positive association exists between the functional status of the knee in the PFPS patients and the results of the measures of muscle function. The higher the functional disability of the knee, the later VMO tends to be activated. Again it is not possible to determine which comes first the change in function leading to a change in muscle activation times or vice versa. There is however an apparent relationship between functional status of the knee, the type of muscle contraction and the speed of execution of the functional task and VMO-VL activation time. This might suggest that the change in activation time is the common factor and delayed VMO activation is associated with the ability to undertake activities at speed and ultimately overall function.

8.2 Assessment of muscular flexibility

Assessment of muscle flexibility comprised four measures including quadriceps, hamstrings, iliotibial band-tensor fascia lata and plantar flexors muscles. The results of the independent t test comparing PFPS patients and healthy matched controls are presented in Table 8.5.

Table 8.5: Results of the muscular flexibility Independent t test between PFPS and healthy subjects.

Muscle	Group	N	Mean (in degrees)	SD	Mean difference between muscles (in degrees)	t	df	p
Quadriceps	PFPS	63	139.25	8.22	-3.16	-2.247	124	0.026
	HEALTHY	63	142.42	7.59				
Hamstrings	PFPS	63	70.53	11.36	-5.54	-2.724	124	0.007
	HEALTHY	63	76.07	11.47				
Iliotibial band	PFPS	63	7.03	5.46	-0.35	-0.376	124	0.708
	HEALTHY	63	7.39	5.05				
Gastrocnemius	PFPS	63	98.30	5.15	-0.98	-1.157	124	0.249
	HEALTHY	63	99.28	4.36				
Soleus	PFPS	63	105.38	5.47	-2.83	-2.978	124	0.003
	HEALTHY	63	108.22	5.20				

Flexibility of the quadriceps ($t=-2.247$, $p=0.026$), hamstrings ($t=-2.724$, $p=0.007$) and soleus ($t=-2.978$, $p=0.003$) was significantly reduced in PFPS patients compared to healthy subjects. In contrast, there was no difference in the iliotibial band ($t=-0.376$, $p=0.708$) and the gastrocnemius ($t=-1.157$, $p=0.249$) muscle flexibility. It

should be noted however that the actual difference in terms of degrees was relatively small (only 3degrees) for the quadriceps and soleus and may not be clinically significant. On the other hand the hamstrings demonstrated a six degree difference in flexibility between patients and healthy controls. This is beginning to approximate a 10% difference which could be considered clinically meaningful.

The PFPS patients quadriceps and soleus flexibility was significantly lower in comparison to healthy subjects though it is questionable whether the difference was of clinical significance. Other studies have however reported the presence of quadriceps muscle tightness in PFPS patients in comparison to asymptomatic controls (Duffey et al, 2000, Kibler, 1987, Piva et al, 2005, Smith et al, 1991, Witvrouw et al 2000) and soleus tightness - though the measures used by Piva et al 2005 were not comparable to the current study. In contrast Messier et al (1991) found no difference between patients and healthy subjects in soleus flexibility. This mixed picture and the small degrees of difference in flexibility reported for the quadriceps and soleus suggests that flexibility in these muscles is not an issue in PFPS.

On the other hand, significantly higher muscular tightness of the hamstrings was evident in the PFPS patients in comparison to healthy subjects of the current study. This observation is consistent with two previous studies (Piva et al, 2005, Smith et al, 1991) but disagrees with results of Kibler (1987) and Witvrouw et al (2000).

The iliotibial band and gastrocnemius muscle were found to have no significant differences between patients with PFPS and the healthy controls. The iliotibial

band findings are similar to previous studies of Piva et al (2005) and Smith et al (1991) who also found no significant differences between healthy and PFPS patients. The results in terms of the gastrocnemius flexibility, contradict with the significant difference reported by Piva et al (2005) and Witvrouw et al (2000), but the studies are not directly comparable due to the use of different measurement techniques. It seems therefore that tightness of the iliotibial band is not important in PFPS but the role of the gastrocnemius remains questionable.

8.3 Assessment of biomechanical characteristics

Assessment of biomechanical characteristics included quadriceps angle (Q angle) and foot pronation measured by using the navicular drop test. The results of these tests are presented in table 8.6.

Table 8.6: Independent *t* test results of the Q angle measurement and the navicular drop test between PFPS and healthy subjects.

Measurement	Group	N	Mean	SD	Mean difference between groups	<i>t</i>	df	p
Q angle In degrees	PFPS	63	18.94	5.40	1.73	1.784	124	0.077
	HEALTHY	63	17.20	5.48				
Foot pronation- Navicular drop test in mm	PFPS	63	12.23	4.34	-1.36	-1.923	124	0.057
	HEALTHY	63	13.60	3.62				

The Q angle recorded for PFPS patients was higher (18.94°) than healthy controls (17.20°) but this small difference was not statistical significant (Table 8.6, *t*=1.784, *p*=0.007) and is unlikely to be of clinical significance. The PFPS patients also presented lower navicular drop values (12.23 mm) than the healthy subjects (13.60 mm) but this small difference was also non-significant.

The findings for Q angle are consistent with the results of previous studies of Caylor et al (1993), Duffey et al (2000), Thomeé et al (1995), Patil et al (2011) and Witvrouw et al (2000) who also failed to found differences between patients and healthy controls. Only three studies (Aglietti et al, 1983; Haim et al, 2006; Messier et

al, 1991) have found significant differences in Q angle values between PFPS patients and healthy subjects.

The results of the foot pronation using the navicular drop test revealed no difference between the PFPS patients and healthy subjects. To our knowledge this is the first study that used the navicular drop test to determine the foot pronation between the patients with PFPS and the healthy subjects. Piva et al (2006) in their reliability study they reported foot pronation values by using the navicular drop test as a measurement tool. They found lower values compared to the study (5.9, ± 2.7 mm) but reported problems with reliability of the test. This suggests these results should be interpreted with caution and do not add substantially to an understanding of the aetiology of PFPS.

Chapter 9

Study III: Repetitive load tasks

9. STUDY III: REPETITIVE LOAD TASKS

The current chapter presents & discusses the measures of muscle function, EMG amplitude & activation time of the VMO in relation to VL, after a repetitive loading task. The repetitive load task was carried out at the end of the measuring session as was described previously in Chapter 6. Theoretically has been proposed that repetitive articular loading can lead to dysfunction and/or pain (Dye, 2001). However, to date that has only been explored in relation to influence on VMO-VL EMG amplitude and not on activation time (Ott et al, 2011) in PFPS patients.

The objective of this task therefore, was to compare the assessment of the amplitude and activation time of the VMO and VL with the muscles in a 'fresh state' to that of a 'fatigued state' achieved after a repetitive loading task.

9.1 Overall muscular analysis performance of the repetitive loads task

9.1.1 Time onset differences

In order to examine the differences of the VMO-VL activation times under isometric testing conditions between the (fresh state) and (non-fresh state) of both, PFPS patients and a healthy control group, an independent measures multivariate analysis of variance (MANOVA) was performed. The analysis revealed (Table 9.1) that the difference in activation time between healthy

controls and PFPS patients is not influenced by whether the muscles are in a fresh or fatigued state ($F_{2,123}=0.971$, $p=0.38$).

Table 9.1: Independent measures Multivariate analysis of variance (MANOVA) of VMO-VL time onset difference between PFPS patients & healthy subjects in the fresh and fatigued states.

Multivariate Test (MANOVA)							
Time Onset Difference in msec		PFPS-Healthy Wilks' Lambda Effect value		F	Hypothesis df	Error df	p
Fresh state		0.984	0.971	2	123	0.38	
PFPS	4						
Healthy	7						
Fatigued state							
PFPS	4						
Healthy	7						

In relation to current knowledge, assessment of measures of activation time after repetitive loads in PFPS patients & healthy has not been explored previously. In the current study the repetitive loading task at 50% of the maximum level of contraction under isokinetic closed kinetic chain conditions lasted for 60sec. This set of parameters was chosen as it is considered an appropriate & acceptable way of assessment and training of PFPS patients in relation to patellofemoral joint reaction forces & stress (Callaghan, 2001). The average value of the Kujala AKPS functional status score reported in this study was 74, this would suggest that despite their AKP patients had a relatively high level of function. Therefore, it is likely that this repetitive loading task constitutes a mild level of repetitive activity relative to the level of functional ability and may not provide a true representation of a fatigued state and may account for the lack of difference between this and the fresh state. Another note of caution is that although the repetitive loading task

was a closed kinetic chain activity, this does not constitute a natural functional task due to the fact that was executed under non weight-bearing conditions. As such it may not be representative of the true-life fatigued situation. The results from previous chapters may suggest that VMO-VL activation times during a more dynamic activity such as, an functional stepping tasks at a fast speed is where the major influence of PFPS lies and creating a fatigue state by repetition of this task may provide more insight regarding the role fatigue may play.

9.1.2. VMO & VL normalised EMG RMS amplitude

In order to examine the differences in EMG amplitude between the 'fresh state' and 'fatigued' state in both PFPS patients and healthy control group a repeated measures analysis of variance (ANOVA) was performed. The analysis revealed (summarized in Table 9.2) that there is no difference in normalized VMO-VL amplitude between the fresh and fatigued state for both healthy controls ($F_{1,62}=0.449$, $p=0.50$) and PFPS patients ($F_{1,62}=0.486$, $p=0.48$).

Table 9.2: Repeated measures ANOVA of the normalized VMO-VL RMS EMG amplitude between PFPS patients & healthy subjects during isokinetic knee extension 1st and 2nd isometric contraction at 75° of knee flexion

Normalized VMO-VL RMS EMG	F	df	p
PFPS patients – fresh vs fatigued	0.486	1	0.48
Healthy subjects – fresh vs fatigued	0.449	1	0.50

These results are similar to those reported by Ott et al (2011). They found that the VMO activation was higher than the VL in both groups before and after aerobic exercise protocol. However, direct comparison between the current results and the findings of Ott et al (2011) is not possible due to differences in the experimental protocol employed by the two studies. Ott et al evaluated VMO & VL activity by using a closed kinetic chain weight-bearing task (single leg anterior reach task) and their repetitive loading task was more functional and weight bearing (20 minutes treadmill walking at self-selected speed). Additionally, it is interesting to note that an increase in perceived pain accompanied the 'fatigued' state described by Ott et al (2011), while in the current study although the level of pain during or after the repetitive loading was not recorded it was observed that the loading task was well tolerated by the PFPS patients and no complaints were observed. Alterations of the VMO & VL activity in PFPS patients reporting increased pain after exercises, have been previously reported (Anderson & Herrington, 2003; Ott et al, 2011), and interpreted as a possible compensatory mechanism to reduce the forces and stress applied on the patellofemoral joint. Further studies may therefore need to take into account functional activities as a means of creating a fatigued state and one in which greater pain is elicited before being able to make definitive statements regarding the role fatigue has to play on EMG amplitude in PFPS patients.

Chapter 10

Overall Discussion & Conclusion

10. DISCUSSION

10.1 Novelty characteristics

This study builds on the literature to establish if VMO-VL muscle imbalance exists in a PFPS population and is the first of its kind to attempt to establish if an imbalance exists, under different experimental and functional task conditions. Previous studies outlined in Chapter 4 were limited in terms of methodology, comparisons with a healthy population, relation of symptoms to experimental and functional tasks and correlations with pain & functional status.

Limitations in terms of methodology presented in previous studies were addressed in the current study by:

- Ensuring there was an adequate sample size based on the results of a previous similar study of Sheehy et al (1998).
- Age / sex matching healthy control subjects with the PFPS patients.
- Careful selection of PFPS patients on the basis of validated inclusion-exclusion criteria.

Additionally, novel elements were adopted in the current study by:

- Optimising testing conditions using a combination of previously validated functional and experimental tasks.
- The addition of some new and important functional parameters such as measures of fresh and fatigue states, different speeds of functional tasks and correlation between the measures of muscle function and clinical symptoms.

10.2 Synopsis of the findings

The overall analysis of the muscular performance across functional and experimental tasks revealed that VMO is recruited before and with higher amplitude than the VL in both PFPS patients and healthy controls though the relative delay in the recruitment of VL is reduced in PFPS patients. Without further analysis this overall picture might suggest that the VMO-VL activation parameters do not play a role in PFPS. However, the more detailed exploration undertaken in this study might suggest otherwise, as further analysis suggests that VMO & VL activation time is task and speed specific and responses vary between PFPS and matched healthy controls. Compared with walking, VMO appears to be activated still earlier than VL during a stepping up task. This difference is even more apparent in healthy subjects than PFPS patients though the differences do not seem to be speed related nor do they reach statistical significance. It could be argued therefore stepping up tasks do not seem to have a role to play in PFPS.

The greatest differences between PFPS patients and healthy controls were observed with stepping down tasks. For healthy volunteers, irrespective of speed, VMO continues to follow the same trend as for other tasks and continues to be activated earlier than VL. However, in PFPS patients there is a move towards simultaneous recruitment times for the VMO and VL at a normal speed and a highly complete reversal of recruitment order with the VL being recruited 16ms before VL during stepping down at fast speed. This interesting link between the type of muscle contraction, the speed of execution and the recruitment pattern of the VMO-VL was additionally confirmed by the findings observed during non-

functional isokinetic tasks with the delay in the VMO activation appearing to be related to the isokinetic eccentric contraction (0° - 90°) at a relatively fast speed.

In contrast, the results of the VMO-VL muscular performance, in terms of RMS EMG amplitude, do not appear to be influenced in the same manner by the type of the muscle contraction, the speed of execution nor the presence or absence of PFPS. VMO consistently presented with a higher EMG amplitude than VL in both PFPS and healthy controls for each functional and experimental task. Therefore, based on these results, the null hypothesis one (NH1) is accepted. The relative EMG amplitude of the VMO vs VL during functional and experimental load tasks in fresh muscles was no different between patients with PFPS and age/sex matched asymptomatic subjects. In contrast, the null hypothesis two (NH2) is rejected, as the relative activation timing of the VMO in relation to the VL during functional and experimental load tasks in fresh muscles is different between patients with PFPS and age/sex matched asymptomatic subjects.

In the light of the pain PFPS describe when walking down stairs it might anticipated that the differences in observed EMG parameters may have been related to clinical symptoms. The correlations between VMO-VL activation patterns and the measures of clinical symptoms however revealed mixed results. Indeed, no direct association was found between the level of pain and the activation pattern and only a weak non-significant tendency appeared to exist between higher levels of pain being associated with a delayed onset in the recruitment of VMO. Furthermore, there appeared to be no associated with a fresh or fatigued state.

Therefore, any inferences in regard relationship between pain, muscle fatigue and activation pattern should be interpreted with caution.

Correlations between the duration of symptoms and activation patterns revealed a weak negative association with a stepping down task executed at fast speed. Therefore, the chronicity of the symptoms may be associated with a tendency towards VMO delayed onset. Perhaps more interestingly, functional status is significantly ($r=0.27$, $p=0.03$) associated with VMO-VL activation pattern and the greater the delay in VMO activation time the higher the knee functional disability. It appears therefore that the delay in the VMO activation time, and indeed reversal of the VMO VL activation time in patients with the PFPS, is worse the longer the duration of symptoms and is associated with the degree of disability. It is not possible to tell however which changes come first ie disability or recruitment pattern but it is clear that the changes are not necessarily related to the amount of pain experienced.

In terms of muscular flexibility three out of five muscles (quadriceps, hamstring and soleus) presented significantly reduced flexibility in PFPS patients compared to healthy controls. However, although statistical significant, the actual differences in terms of degrees was relatively small (only 3 degrees) for the quadriceps & soleus and only the hamstring difference ($\approx 10\%$) could be considered as clinically meaningful. Furthermore, no differences were observed in Q angle and foot pronation. Based on these cumulative observations from the functional tasks and clinical symptom results the null hypotheses three & four (NH3, NH4) can only be

partially rejected as only the relative activation time of the VMO in relation to VL during certain functional tasks was correlated to the clinical symptoms. Null hypotheses five and six (NH5,NH6) are however accepted as the relative EMG amplitude and the relative activation time of the VMO in relation to the VL under isometric conditions between fresh and fatigued state was no different between PFPS patients and age/sex matched asymptomatic subjects.

10.3 Clinical implications of the findings

Historically the aetiology of PFPS syndrome has been considered multifactorial and impaired VMO neuromuscular function in terms of EMG magnitude and timing has believed to have been one of the major factors contributing to patellofemoral maltracking and therefore responsible for triggering or perpetuating the pain. Nevertheless, the evidence for the VMO impaired neuromuscular function has not been consistent across all studies (Brindle et al, 2003; Cowan et al, 2002; Powers et al, 1996; Voight & Weider, 1991; Witwrouw et al, 1996). Indeed, the extensive literature review carried out for the current study (Chapter 4) confirms that muscle imbalance remains controversial and should be interpreted with caution due to the marked heterogeneity and methodological limitations of the reviewed papers. The review did note however, that, although the evidence is not yet convincing, a relationship may exist between the dysfunction of the vasti muscles and patellofemoral pain.

The major finding of the current study is that impairment in VMO-VL activation appears to be task specific. This study was the first to optimise testing conditions using a combination of previously validated functional and experimental tasks and additionally employ two different speeds (normal & fast) of execution. It is clear that stepping down and at faster speeds has a role to play in the manifestation of the VMO-VL time onset muscle imbalance. It is also obvious that overall muscular performance analysis can mask true group differences. It is already recognized that motor unit recruitment strategies can be influenced by various factors such as the nature of the executed task (open or closed kinetic chain, weight-bearing or non-weight-bearing) which is likely in some cases to cause task specific adaptations (Stensdotter et al, 2003; Stensdotter, 2005). The type and speed of muscle contraction also has been reported to play crucial role in motor unit recruitment strategies (Chester et al, 2008, Grabiner et al, 1992). Additionally, has been reported that kinematic characteristics are altered during fast speeds of walking and PFPS patients tend to decrease knee flexion and reduce speed (Powers et al, 1997 & 1999). Similarly, during stair ascent-descent PFPS patients prefer slower speeds and less knee flexion. These previous findings seem to corroborate with those from the current study. Reducing gait velocity and knee flexion range may therefore lead to, decreased quadriceps force demands, lower ground reaction forces and less PF joint loading (Brechtel & Powers, 2002) and may provide pointers for further treatment strategies.

There does however remain a further important consideration ie how large must be the delayed onset of the VMO relative to the usual VMO-VL activation ratio in

order to have a meaningful clinical implication? In the current study the VMO delay was 16ms with previously reported results ranged from 16-50ms (Boling et al, 2006; Cowan et al, 2001; Cowan et al, 2002a; Crossley et al, 2004). It has been suggested that delayed onset time in the order magnitude of 16-50ms may not be clinically significant (Brindle et al, 2003). Nevertheless, computer simulation findings suggest that a biomechanical alteration in PF joint can be caused by even 5ms delayed onset of the VMO (Neptune et al, 2000) and more recently Pal et al, (2011) reported significant relationships between VM delayed onset and patellar maltracking (abnormal lateral tilt & glide). This would suggest that the results from this study are clinically meaningful. Nevertheless this is a complex issue that needs further investigation into the relationships between recruitment strategies and anatomical and kinesiological factors, such as the condition of the various static and dynamic stabilisers of the patella (e.g plica, patellar ligaments, muscles e.t.c)

Whilst it is clear from the current study that functional activities executed with fast speed had a significant impact in the muscular performance this was not exacerbated by repetitive functional loading. It could be argued that the moderate intensity, duration and non-weight bearing task used during the repetitive loading was not sufficient to elicit any potential differences or the severity of the PFPS was insufficient to be influenced by fatigue. It is not possible therefore to make any recommendations in terms of the clinical implications of the fatigue in the treatment of PFPS.

In terms of the correlations between the measures of muscle function and measures of clinical symptoms was evident that the pain level and the chronicity

of symptoms (duration) had no significant associations. The knee functional status (Kujala scale) was however significantly correlated with delayed VMO activation during the fast stepping down. This study was the first to attempt these types of correlations so comparisons cannot be made with findings of previous studies and is hard to draw any definitive conclusions. Furthermore, it is not possible to establish cause-effect relationship between pain, durations of symptoms and functional status. Nevertheless, there appears to be a vicious circle relationship between all these variables. It is known that pain can cause quadriceps arthrogenic muscle inhibition (Rice & McNair, 2010), and that VMO is reported to be the most vulnerable muscle (Grelasamer & McConnell 2010). As a subsequence of pain and muscle inhibition reduced functional ability can occur in order to protect the joint by decreasing the applied stresses. Even mild quadriceps arthrogenic inhibition can contribute to muscle atrophy and hinder rehabilitation (Hurley & Newham, 1993). Arthrogenic muscle inhibition is observed across various knee disorders including anterior knee pain (Suter et al, 1998). When the level of patellofemoral pain is managed efficiently this has a positive influence on the functional status of the joint (Zappalla et al, 1992). This functional improvement has been associated with increased quadriceps activity which in turns stabilizes the patellofemoral joint and results in a decrease in the pain level (Steinkamp et al, 1993). Although non-conclusively, there are evidence suggesting that when the pain level is reduced via taping the delayed VMO activation time is restored to normal (Balachandar et al, 2012). Although from the weak non-significant (pain & duration) and the significant but relatively weak (functional status) correlations no inferences can be made, the overall picture of the findings of this and other

studies implies that all these variables indirectly influence the activation pattern during the fast stepping down task and therefore deserve a detailed observation.

In terms of the muscular flexibility findings only the hamstrings demonstrated statistical and clinically significant changes associated with PFPS. Bearing in mind the multifactorial nature of the PFPS etiology it is not possible either refute or accept the association between this observation and PFPS, further studies are required to clarify any association between reduced flexibility and PFPS. Nevertheless, muscle tightness has been associated with PFPS and evaluation of the flexibility of these muscles considered an essential part of PFPS patients' clinical assessment. Tightness of quadriceps and hamstrings muscles may increase patellofemoral joint compression forces and in this way predispose healthy individuals to PFPS (Piva et al, 2006, Smith et al, 1991, Witvrouw et al, 2000). Additionally, there is some evidence to support an association between tightness of foot plantar flexors and development of PFPS (Witvrouw et al, 2000). Reduced flexibility of the iliotibial band may cause a lateral displacement of the patella and thus increase the stress in the patellofemoral joint and medial retinacular tissue (Brody & Thein, 1998, Wilk et al, 1998).

The assessment of the biomechanical characteristics such as the Q angle and foot pronation (with the navicular) drop test revealed no differences between PFPS patients and healthy controls. Previous studies have reported associations between these biomechanical features and PFPS patients (Powers et al, 1995, Messier et al, 1991, Witvrouw et al, 2005). Although Thomeé et al, (1995) suggested

that there is no direct correlation between high Q angle and patellofemoral pain they stated that an abnormal Q angle may be a contributing factor to maintaining PFPS once the syndrome has been acquired. Both measures Q angle and foot pronation, are considered an essential part of a thorough clinical examination and successful management of these structural – postural alterations has been considered as a prerequisite for successful long term conservative treatment (Witvrouw et al, 2005). It should be mentioned that Q angle and navicular drop are both measurements executed under static conditions, therefore their results do not necessarily reflect potential alterations eventually occurred under dynamic conditions. The value of these tests are therefore questionable and whilst they may still be included in any assessment the results should be interpreted with caution.

10.4 Limitations of the study & recommendations for future research

It has been proposed that PFPS patients with VMO-VL muscle imbalance may constitute a subgroup of the entire population of the patellofemoral pain group (Cowan et al, 2001, Cowan et al, 2002, Crossley, 2010). If such a group exists they may require modification of the usual assessment protocol and subsequently of the treatment approach. Sub group analysis was beyond the scope of this thesis nevertheless, further studies adequately powered for sub-group analysis may require careful consideration.

Another limitation of the current study was the lack of blinding of the assessor to sub group ie PFPS or normal. This may have led to a potential bias in reporting and

interpreting the results. It should be noted that none of the previous studies used blinding and due complexity and time constraints was not considered feasible in the present study. The current study did however employ an automated computer algorithm for the time onset detection. This method is considered highly successful in avoiding type I errors (Chester et al, 2008, Hodges & Bui, 1996) and therefore reduced some of the potential bias.

All other aspects of the study were controlled as rigorously as possible, the study was well designed, adequately powered using a calculation based on a previous similar study.

Finally, it might also be beneficial to include in the assessment of muscular performance a combination of optimised functional and experimental tasks giving emphasis to the use of multiple speeds of execution and employing task that more closely mimic fresh and fatiguing daily life activities.

10.5 Conclusions

This thesis is the first of its kind to attempt to establish if an imbalance exists under different experimental and functional task conditions in a clinically defined PFPS population. Additionally was the first attempted to determine if this muscular imbalance is related to clinical symptoms associated with the PFPS and/or lower limb physiology.

The ultimate aim of the study was to establish if it is appropriate to continue addressing a VMO – VL muscle imbalance and treating with physiotherapeutic interventions in patients with clinically defined PFPS.

The findings revealed that the VMO-VL activation patterns are task specific and most significantly related to stepping down tasks at a fast speed of execution. Furthermore, it is the timing of VMO/VL activation rather than the amount of activation that is important. Additionally, a link appears to exist between activation pattern and duration of symptoms and functional performance but not with pain.

The results of the study suggest that a VMO-VL muscle imbalance actually exists in a clinically defined PFPS population and subsequently it is appropriate to continue addressing and treating this complex and challenging issue.

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Appendices

Appendix II

Anterior knee pain scale (AKPS) (Kujala et al, 1993).

ANTERIOR KNEE PAIN SCALE (AKPS)			
For each question, circle the latest choice (letter) which corresponds to your knee symptoms			
1.	Limp a. none (5) b. slight (3) c. constant (0)	8.	Prolonged sitting with the knee flexed a. no difficulty (10) b. pain after exercise (8) c. pain forces to extend knees temporarily (4) d. unable (0)
2.	Support a. full support without pain (5) b. painful (3) c. weight bearing impossible (0)	9.	Pain a. none (10) b. slight & occasional (8) c. interferes with sleep (6) d. occasionally severe (4) e. constant and severe (0)
3.	Walking a. unlimited (5) b. more than 2 km * (3) c. 1-2 km * (2) d. unable (0)	10.	Swelling a. none (10) b. after severe exertion (8) c. after daily activities (6) d. every evening (4) e. constant (0)
4.	Stairs a. no difficulty (10) b. slight pain when descending (8) c. pain both when descending & ascending (5) d. unable (0)	11.	Abnormal kneecap (patellar) movements (subluxations) a. none (10) b. occasionally in sport activities (6) c. occasionally in daily activities (4) d. at least one documented dislocation (2) e. more than two dislocations (0)
5.	Squatting a. no difficulty (5) b. repeated squatting painful (4) c. painful each time (3) d. possible with partial weight bearing (2) e. unable (0)	12.	Atrophy of thigh a. none (5) b. slight (3) c. severe (0)
6.	Running a. no difficulty (10) b. pain after more than 2 km * (8) c. slight pain from the start (6) d. severe pain (3) e. unable (0)	13.	Flexion deficiency a. none (5) b. slight (3) c. severe (0)
7.	Jumping a. no difficulty (10) b. slight difficulty (7) c. constant pain (2) d. unable (0)		* 1 km = 5/8 mile † to score, sum the circled responses

Appendix III: Volunteer Information Sheet



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VOLUNTEER INFORMATION SHEET

Project Title: "Vastus Medialis Oblique-Vastus Lateralis muscle imbalance in Patellofemoral Pain Syndrome (PFPS) patients"

Researcher: Panagiotis Trigkas

Research Supervisor: Prof. J. Oldham

We are inviting you to take part in a study which I am undertaking as part of a doctoral degree at the University of Manchester, and in conjunction with the Technological Educational Institute (T.E.I.) of Lamia, in Greece. Before you decide whether you want to participate in the study, please read the following information in order to understand why we are doing this study. You do not have to immediately decide whether you want to participate in the study; you can (if you want) discuss it first with others and then make up your mind upon participation. If anything is not clear, please do ask and we are happy to provide you with further information.

What is the aim of the study?

The study is exploring if the muscle imbalance between different parts of the quadriceps muscle (big muscle in the front side of the thigh) exists in patients with

patellofemoral (knee cap) pain syndrome in comparison with healthy individuals. The difficulty in efficiently treating patellofemoral pain is well known. In order, to be able to deal with this problem, we need to know the specific clinical characteristics of the muscular performance of the patellofemoral (knee cap) pain patients. Patients with patellofemoral (knee cap) pain do not form a uniform clinical group but are comprised of smaller groups with similar characteristics (sub-groups). Therefore, the aim of this study is: a) to determine whether the muscle imbalance actually exists in patellofemoral (knee cap) pain patients and if it does whether it is specific to this condition or a similar quadriceps muscle imbalance exists in a healthy population b) if muscle imbalance exists whether it is related to clinical symptoms used as indications of pain syndrome in clinical practice.

Who is doing the research?

The research will be conducted by Panagiotis Trigkas, chartered Physiotherapist, Lecturer of the Physiotherapy Department of the Technological Educational Institute of Lamia, Greece, supervised primarily by Professor J. Oldham, Director of the Centre for Rehabilitation Sciences of the University of Manchester, UK and secondly by Professor G. Gioftsos, Vice President of the Technological Educational Institute of Lamia, Greece.

Is there any reason I should not take part in this study?

You should not take part in this study if you suffer from:

Breathing (respiratory) problems like, *asthma, bronchitis, emphysema, pneumonia, fibrosis*, or any other breathing problem.

Heart problems like, chest pain, heart attack, hardening of arteries (atherosclerosis), heart muscle disease (ischemic cardiomyopathy), hypertensive heart disease, valvular heart disease or any other heart problems.

Knee problems like, any previous knee surgery, osteoarthritis, history of traumatic patellar (knee cap) instability-dislocation or subluxation, meniscal injury or ligament injuries of the knee, knee soft tissue injury (i.e. fat pad impingement), knee soft tissue overuse (i.e tendinopathy), apophysitis, synovial plica or knee chondral damage.

Lower limb problems like, functional (symptomatic) instability in any joint of the lower extremity.

Other conditions like, knee pain caused by referred pain from low back disorders or hip arthritis, neuroma.

What will I have to do? (methods of the study)

Volunteers taking part in the study will be clinically examined by an experienced chartered physiotherapist. The clinical examination will involve the following 3 sections:

- i) measurements of muscle function of quadriceps muscle using surface electromyography during functional tasks (a simple exercise of ascending & descending a normal height step) and knee extension exercises (straitening your knee) in an isokinetic dynamometer (strength chair).
- ii) measurements of muscle function of quadriceps muscle using surface electromyography during repetitive load of knee extension exercises (straitening your knee) in a strength chair.

iii) Completing two scales – (these are some standardized easy to respond scales specifically designed for: a) pain assessment & b) assessment of the current level of the knee function), and undergoing some clinical tests: a) assessing flexibility of four muscle of the lower limb & b) measuring the quadriceps-patellar angle and the foot pronation.

The tests and measurements used for this study are the most commonly used clinical procedures involved in a standardized patellofemoral physical examination. The whole procedure will approximately take 75 minutes.

Do I have to take part?

No. Your participation is entirely voluntary. Your participation in this study will not affect the normal treatment you are receiving if you're a patient, and you are free to withdraw at any time without giving any reason.

What is the benefit from taking part in this study?

If you are a patient with PFPS, although there are not any direct benefits because the study is not including any therapeutic intervention but only assessment & evaluation, following the evaluation and the measurement procedure from the chartered physiotherapist, you will receive useful information regarding the nature of your condition. The information derived from your measurements will help to clarify what is the specific cause of your condition (for example, muscular dysfunction-selective muscle weakness or muscle shortening, biomechanical derangement of the patella (knee cap) or the foot). Additionally, we hope that the results of this study will be useful for future patients with PFPS.

If you're a healthy individual, you will receive, according to your results, relevant information about the muscular performance (flexibility & strength) of the muscles of the lower limb.

Are there any potential dangers, discomfort or inconvenience?

Although we have design very carefully and precisely the assessment protocol according to clinical & research standards, there is always a remote possibility for experiencing minor discomfort or inconvenience during the assessment. Experience from previous similar studies indicates there is unlikely for exacerbation of your condition, nevertheless, in case you feel any pain, discomfort or inconvenience during any part of the measurement, please feel free to stop immediately and withdraw from the study at any time. Additionally, we would like to inform you that, the researcher is an experienced and adequately trained physiotherapist who can offer first aid service, our laboratory is equipped with a first aid kit and furthermore the Medical & Nursing Services Center of our Institute is located close to our laboratory and is ready to provide any additional help in case of an emergency.

Is it confidential?

We certify that all your details will be confidential. We will always refer to your case with a number and never with your name. Therefore, your anonymity will be assured and the project team will be the only one who has access to your notes.

Respect your decision in taking part in the study.

It is your decision whether you want to participate in this study. If you decide to participate, you will be given a *Consent form* that you need to sign. You always have the right to withdraw from the study without giving us any explanations and this decision will not affect your treatment course.

Our commitments.

We will inform the Doctor who referred you of your participation in the study. If any of you want us to inform a consultant or other doctor involved in your care we are happy to do so. Also, we are committed to withdraw you from the study, if we should feel that it is appropriate.

My full details as well as the details of my supervisors involved are provided below. Thank you in advance for assisting this study which will contribute greatly to the search for effective management of patellofemoral pain syndrome.

Sincerely,

Panagiotis Trigkas

Principal investigator:

Panagiotis Trigkas

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Prof. Oldham, J.

University of Manchester, UK

Advisors:

1. Dr. McBeth, J.

University of Manchester, UK

2. Prof. Giotfos G.

Technological Educational
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Appendix IV: Consent Form

CONSENT FORM FOR CLINICAL TRIAL WITH COMPETENT ADULT VOLUNTEERS

Title of Project

"Vastus Medialis Oblique – Vastus Lateralis muscle imbalance in Patellofemoral Pain Syndrome (PFPS) patients"

Hospital/Institution: Centre for Rehabilitation Science, University of Manchester & Technological Educational Institute (T.E.I.) of Lamia

Subject's surname.....Other names.....

Date of Birth.....

Sex (please tick)

☐

Male

☐

Female

Age

I confirm that I have fully explained the purpose and nature of the investigation and the risks involved

Name of investigator

Signature

CONSENT FORM OF ADULT VOLUNTEERS FOR PARTICIPATION IN THE CLINICAL STUDY

Title: " Vastus Medialis Oblique – Vastus Lateralis muscle imbalance in Patellofemoral Pain Syndrome (PFPS) patients"

based in the following institutions: 1) Centre for Rehabilitation Science, Epidemiology & Health Sciences Department, University of Manchester, & 2) Technological Educational Institute (T.E.I.) of Lamia, Department of Physiotherapy

Please read carefully

The investigator has explained to me the nature of the research and what I would be asked to do as a volunteer, and has given me my own copy of the *Patient Information Sheet*, which I have read and understood.

Having enough time to consider my decision since seeing the information about the trial, I consent to take part as a volunteer and I understand that I am free to withdraw at any time without giving any reasons, and without detriment to myself.

I am aware that the results of the study may be presented in scientific conferences or journals in the future. However, the information that I will provide for the study will be kept confidential.

I also know that my doctor may be informed about my participation in this study and I agree to that.

Therefore, I agree to participate as a volunteer in the present study.

Signature

Name: _____ **Date:** _____

Address: _____

Telephone: _____