The development of a new comprehensive management model for Apophysitis Syndromes in adolescents, based on the assessment and management of passive muscular tension

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A thesis submitted in partial fulfilment of the requirements of the University of Greenwich for the Degree of Doctor of Philosophy

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DECLARATION

"I certify that this work contained in this thesis, or any part of it, has not been accepted in substance for any previous degree awarded to me, and is not concurrently being submitted for any degree other than that of Doctor of Philosophy being studied at the University of Greenwich. I also declare that this work is the result of my own investigations, except where otherwise identified by references and that the contents are not the outcome of any form of research misconduct".

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ABSTRACT

Introduction: To date there appears to be no consensus to the accepted management of Apophysitis syndromes (AS) and no coherent research towards one. Despite these conditions being amongst the most common injuries to affect adolescents, and in particular young athletes, most recommended approaches are based on opinions and experience only. This thesis presents a new comprehensive approach to the management of AS in the lower limb, based on an assessment of local passive muscular tension as demonstrated by reduced flexibility.

Method: A new theoretical model of passive and active tension underlying AS is proposed and used to demonstrate the new approach. Four main studies are presented. The first study examined a novel treatment of the most common AS in adolescents – Osgood-Schlatter's disease (OSD). The same approach was applied to a different AS – Sever's disease, in the second study. The third examined the role of growth and flexibility during adolescence in a longitudinal study to ascertain the influence of passive tension in the development of AS, and finally an experimental stretching intervention study addressing reduced flexibility in young footballers, was performed as a preventative strategy for AS, and compared to current incidence rates from 3 football academies. Together they present a coherent strategy for the treatment, aetiology and ultimately prevention of AS.

Results: Findings from the OSD study found that reducing passive tension in the quadriceps muscle enabled the recovery of patients in a median of 17 days (6- 40). Similar results of 13 days (5-42) occurred in the second study on Sever's disease treatment. Monitoring growth and flexibility during adolescence in academy football players found that flexibility significantly reduced in academy footballers prior to developing AS, and there was a strong negative correlation between growth rate and muscle flexibility (-0.96 and -0.97) for the quadriceps and calf muscles respectively. Introducing a stretching programme as an intervention to those whose flexibility exceeded set thresholds, showed none of these players went on to develop an AS. Incidence of AS was shown to have declined in the intervention club by 69% compared with control clubs 7% and 15%.

Conclusion: Increased passive tension during adolescent growth appears to be associated with the development of AS. Addressing this during treatment improves the outcomes for patients and can be part of a preventative strategy for potential at-risk players. Everyone involved in encouraging youth sports should be aware of the problem of AS and recognize the need for a proactive flexibility approach during the peak adolescent growth spurt. This study presents the arguments and creates the opportunity to promote widespread changes for the good of adolescent well-being and health.

ADDENDUM

Peer reviewed publications

Strickland J.M, Colpus M, Goss-Sampson MA. (2018). A Therapy protocol for the treatment of Osgood-Schlatter's Disease: case series. Journal of Sport Rehabilitation – accepted for final review July 2018. Manuscript ID: JSR.2018-0050.R1

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Conference proceedings

Key Note

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Strickland J. (2015). Apophysitis syndromes: There is something you can do. Biennial Conference of the Association of Chartered Physiotherapists in Sports Medicine, Brighton

Strickland J. Childhood Apophysitis Injuries. (2010). Zen and the art of manual therapy seminar. University of Kent, Chatham, Kent UK

Oral Presentations

Strickland J and Goss-Sampson MA. (2010). Sever's disease: a novel approach to treatment – pilot study. European College of Sports Science Annual Conference, Anatalya, Turkey

Strickland J, Coleman N, Brunswic M and Kocken R. (2008). Osgood-Schlatter's disease: an active approach using massage and stretching – pilot study. European College of Sports Science Annual Conference, Lisbon, Portugal

ABBREVIATIONS USED

ADL..... Activities of Daily Living

AIIS…….. Anterior Inferior Iliac Spine apophysitis

AS ……..... Apophysitis Syndromes

CP……. Crystal Palace Football Club Academy

CPD..... Continuous Professional Development

CSP..... Chartered Society of Physiotherapists

EBP……. Evidence-Based Practice

ECSS……. European Congress of Sports Science Conference

EPPP……. Elite Player Performance Profile

GP..... General Practitioner / Doctor

HCPC..... Health Care Professions Council

KPI..... Key Performance Indicator

MRI..... Magnetic Resonance Imaging

MRM……. Myofascial Release Massage

MSK..... Musculo-skeletal

MTU..... Muscle-tendon unit

NICE…… National Institute for Clinical Excellence

NHS..... National Health Service (UK)

OSD……..... Osgood-Schlatter's disease

PE…… Physical Education

PHV……. Peak Height Velocity

RCT……. Randomised Clinical Trial

SLJ……..... Sinding-Larsen-Johannsen disease

US..... Ultra-sound imaging

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CHAPTER 1. Introduction

1.1 Background

1.1.1 Apophysitis syndromes

Apophysitis syndromes (AS) are a series of related overuse injuries that affect adolescents during their secondary growth stages. These injuries affect the growing apophyseal regions of bones and particularly the attachment zones of major tendons such as the patella and the Achilles. It has been suggested by various expert opinions (Micheli 1987; Dalton 1992; Harries et al 1994; Christopher and Congeni 2002; Brukner and Khan 2012) that these apophyseal insertions are subjected to repeated traction forces and micro-trauma resulting in inflammation (apophysitis) and sometimes detachment of bony material (avulsion). The resulting pain and dysfunction can be debilitating for those affected. The most common AS are Osgood-Schlatter's (OSD) and Sever's disease which affect the knee and the heel respectively, but they can occur in other areas of the body (Figure 1). Whilst the mechanism of these overuse injuries remains unclear the association between growth spurts and physical exercise is widely documented and thus the most affected group are young athletes engaged in sports, aged between 8 and 15 years old (Dalton 1990; Peck 1995; Micheli and Fehlandt 1996; Lau et al 2008; Suzue et al 2014).

The scale and impact of AS varies from the individual to the general population and within different sports settings. The individual patient in a general population will have symptoms ranging from mild to severe pain and dysfunction. Two studies within a general population found incidences of OSD in 10% of musculo-skeletal patients in a general practice (De Inocencio 1998) and 7% in a school environment (De Lucena et al 2011). This latter study also found almost double the incidence of OSD in those children who played sports (13%). If the other AS conditions were also included the incidence of AS would be much higher. In a more sports-related environment the incidence of AS has been found ranging from 5% (Stracciolini et al 2014) up to 37% (Micheli and Fehlandt 1992) of adolescent patients presenting at sports medicine clinics. However most published data comes from more elite single-sports group samples but again with a wide range from 5% (Price et al 2004) up to 23% (Reece 2012). The impact of these injuries on an elite sports population can be that players with an AS may not be able to perform optimally and in a team environment this may

have a wider effect on team performance, as well as future prospects for the individual. AS therefore present a number of challenges at the individual and larger group population levels.

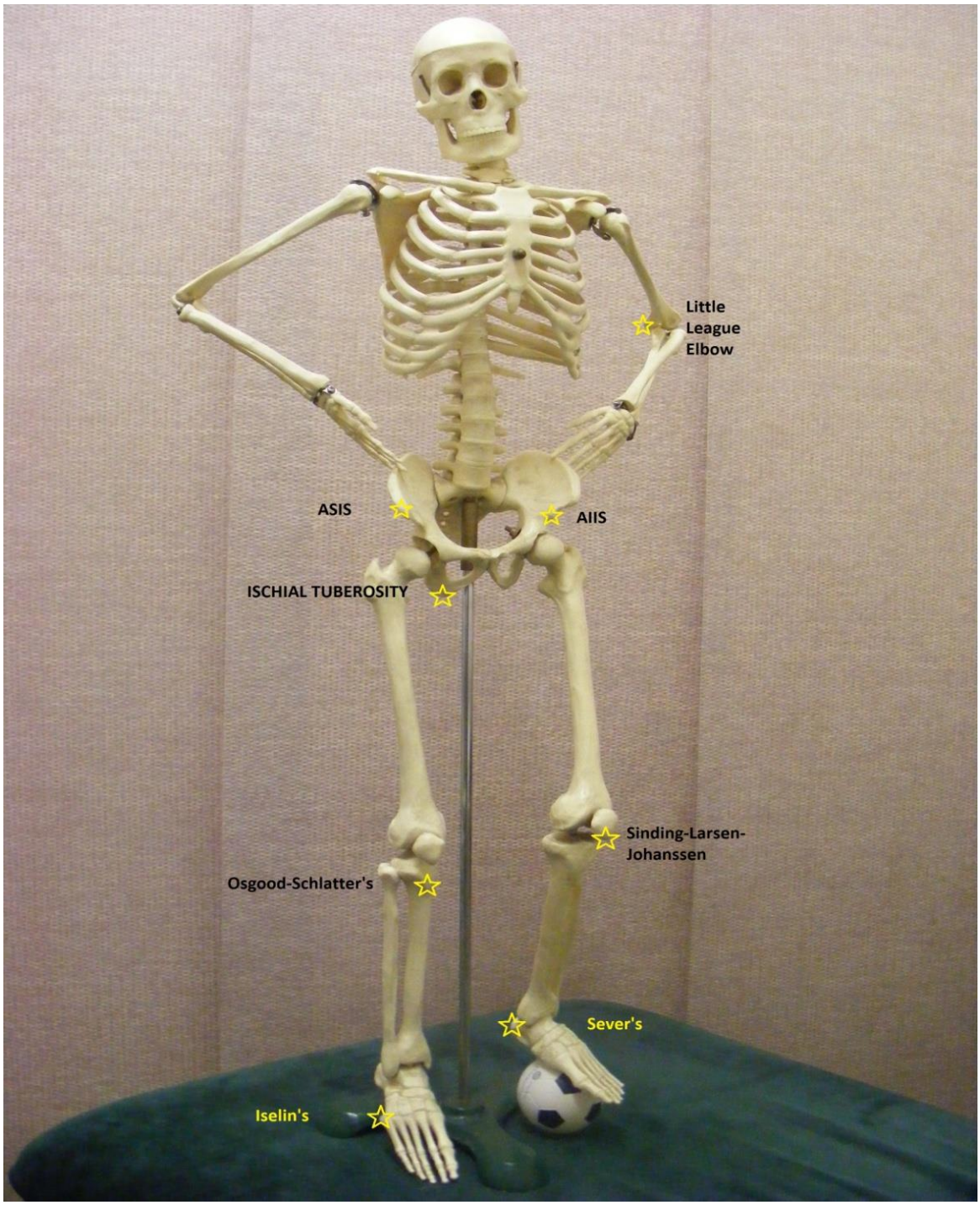


Figure 1. Common Apophysitis syndromes and their locations

The clinical approach for treatment efficacy needs to be addressed at the individual patient level. The standard treatment described in the literature has remained unproven and largely unchallenged for over 100 years and is simply based on symptomatic relief

and modification of activity until the child's skeleton reaches maturity (Osgood 1903; Sever 1912; Antich and Brewster 1985; Micheli and Ireland 1987; Peck 1995; Gholve 2007; Kivel 2011; Domingues 2013). This approach can take several months or even years leaving many patients unable to partake in their chosen sports or normal activities (Ehrenborg 1962; Harries et al 1995; Bloom et al 2004; Brukner and Khan 2012). In elite sports this can mean a premature end to their sporting careers as patients sit out entire seasons or are reluctantly released from their squads. Removing adolescents from healthy exercise can also have a detrimental effect upon them socially and emotionally (Booker 2008). The standard treatments have never been scientifically studied with no evidence as to their efficacy, nor consensus as to format (Bloom et al 2004), and therefore justification for their continued selection relies on historical opinion with poor results. Using a clear clinical evidence-based practice (EBP) approach as recommended by Sackett (1996), in a physiotherapy setting should enable a better-informed approach that challenges the status quo and seek to provide a more effective treatment for AS patients.

The second and third challenges are from a wider sports perspective. These are to attempt to understand the relationship between growth, flexibility and the development of AS, and whether prevention is possible. There have been limited studies into understanding the causes or risk factors of AS (Ikeda et al 1999; Mehdinasab and Fakoor 2005; Shiota et al 2016) and none, that, assess prevention strategies. Some patients never fully recover (Ross and Villard 2003) and 60% of OSD patients are affected into their adulthood (Krause et al 1990), yet the syndromes are dismissed as minor injuries that will resolve naturally (Harries et al 1994; Domingues 2013; Peterson and Renstrom 2017). Published literature relies almost entirely on professional medical opinions based on experience, not scientific scrutiny. AS are amongst the most common overuse injuries to affect this vulnerable age group (Dalton 1992) with a range from 5% (Peck 1995) to 37% (Micheli and Fehlandt 1992) in the injured sporting population, but 6% within a junior soccer club population (Suzue et al 2014), and the poor management of this problem is itself unchallenged. There is no consensus for treatment, aetiology or prevention strategies and little proactive research. It needs scientific investigation into a modern, and effective solution, and to understand why it happens in the first place, thereby enabling its prevention.

1.1.2 Context

Sport is not only a vehicle for the development of competitive physical endeavor but also for the promotion of healthy exercise and is a key goal from the UK Government's targets for a healthy population (HM Government 2015). Their policy paper the "Sporting Future: A new strategy for an active nation", states that sport can transform lives and improve physical and mental well-being and develops individuals and society in a positive manner. The government also made a commitment to its Olympic and Paralympic legacies to support grassroot sport and encourage the sharing of UK Sport's knowledge and expertise (HM Government 2015). Part of grass roots development is the encouragement of children and youths into sport participation at all levels and without barriers. There is a strong desire to see children and young people develop a love for sport and physical activity and enter a life time of participation and enjoyment which will create a healthier and happier population (Bergeron et al 2015; Timpson 2015).

Keeping sporting participants healthy is not just the responsibility of healthcare professionals, but also those engaged in promoting active lifestyles and supervision of sports e.g. sports coaches, schools and the local community (NICE 2009). Institutions such as schools and sports academies also have a 'duty of care' to vulnerable youngsters to provide a safe environment for sport and physical activity and should therefore be encouraged to become more involved in the early detection and prevention of sports injuries such as AS, which occur exclusively in children and adolescents, and reflect upon their current management and monitoring strategies. New approaches by medical and health practitioners should be patient-focused and based on evidence gained through sound scientific methodology (NHS 2018), and not on anecdotal evidence, no matter how esteemed its source. It is also important to share such research with the wider community for the benefit of all (HM Government 2015). Research should be aimed at prevention and this in turn needs to be based on accurate knowledge of incidence, severity and aetiology of injuries (Van Mechelen et al 1992). Comprehensive approaches should therefore involve all those professionals involved in supervising and guiding active children and adolescents, not just health care professionals who tend to be only involved after the injury occurs. A proactive approach by all would reap the most rewards for a positive and safe sporting future, in particular for the vulnerable adolescent age groups (Van Mechelen et al 1992; Bergeron et al 2015).

An elite sports environment such as a football academy presents a sample population which is at greater risk of AS compared to their school peers, and also has the facilities to undertake a longitudinal study to encompass the varying maturity rates of adolescents. It has also been recognized within the United Kingdom and the Football Association (FA) in particular, that a more comprehensive and scientific approach to young player care was needed. In 2012 the FA launched its Elite Player Performance Programme (EPPP) with a primary aim of increasing the quantity and quality of home-grown players into the professional game, however one of its secondary aims was to understand injury risk and try to reduce it within the academy setting. Specifically, it recognized that certain injuries were associated with the growth periods found in the academies and that it had a responsibility to be proactive. AS are the most common overuse injury found in elite sports populations and in the youth age range of 11-15 years in particular, therefore AS are a recognized problem within the context of the EPPP specifically. It has also been noted that young talented athletes have higher volumes and intensities of training and this may make them more vulnerable to injury than their school peers (Brenner 2007; Feeley et al 2015; Read et al 2016).

1.2 Rationale for the current research project

Despite over a century since they were first described, AS have had limited scientific research into treatment and aetiology, and no prevention studies have been performed. AS patients presenting for physiotherapy treatment were observed by the researcher to have a number of similar signs and symptoms. Not only did they have the well documented symptoms of pain and dysfunction in a growing adolescent, but the researcher also noted they had reduced flexibility in their muscles and were highly active individuals. The question raised was if these factors were fundamental to the possible development of AS and would addressing them help in their subsequent management.

This thesis develops new ideas and methods by combining good clinical and academic scientific practice. It seeks to improve the management for AS patients by not only improving treatment efficacy but also by addressing the cause and prevention. It will challenge traditional standard medical approaches and apply sound logic to clinical problem solving. It will also recommend a greater role for involvement of schools and sports academies in a more proactive provision of a safe and healthy sports environment for their children, safeguarding the health of our Olympic legacy.

1.3 Thesis outline

This thesis is presented in 6 Chapters. Chapter 1 outlined the thesis overview and introduced the background and context of the thesis by giving a review of the literature on the presentation of AS, their current management and the context of the environment where they are most prevalent. Chapters 2 and 3 presented a clinical approach on individual patients using evidence-based practice with two studies on a new method to treatment of the two most common AS – Osgood-Schlatter's disease and Sever's disease. Chapter 4 took an epidemiological approach and explored the relationship of anthropometric parameters, muscle flexibility and the development of AS during the peak adolescent growth period in group setting of high-risk academy male footballers, and Chapter 5 further explored the epidemiological line and examined the results of a stretching intervention programme on the incidence of AS in a group of 3 football academies. Finally, Chapter 6 summarised the findings and made recommendations for practical applications and future work.

1.4 Thesis Aims

Aim 1: To determine if a novel clinical approach, based on assessing and managing passive muscular tension, can be demonstrated to be effective in the treatment of AS patients.

Objectives:

1.1 Identify signs and symptoms associated with presentation of OSD and Sever's disease in adolescent patients

1.2 Create a model based on those factors as a basis for treatment intervention

1.3 Test the treatment model on OSD

1.4 If successful, extend test the model on a different AS

Aim 2: To determine if passive muscular tension may be a risk factor in the onset of AS and therefore can be used for its prevention.

Objectives:2.1 Identify risk factors in AS amongst academy footballers

2.2 Monitor and intervene in risk factors

2.3 Test the prevention strategy for efficacy

CHAPTER 2. Clinical Approach: Osgood-Schlatter's Disease

Study 1: A novel approach to the treatment of Osgood-Schlatter's disease: case series

2.1 Abstract

Osgood-Schlatter's Disease (OSD) is the most common overuse injury to affect growing children and teenagers, yet there is no consensus as to the best treatment, and recovery can take months to years. A quicker and more effective intervention is needed. The aim of this study was to assess the effectiveness of a novel therapy intervention on adolescent patients with OSD.

Method: A case series was undertaken in a private physiotherapy clinic, with 75 consecutive OSD patients (age range 8-17 years). A treatment protocol was implemented using massage or stretching, and rest from activity, depending on patient response to outcome measures. Time to discharge was the primary dependent variable based on pain free passive and active outcomes. Presence of pain at the patella tendon apophysis during a passive standing-quadriceps stretch and an active eccentric wall squat were the secondary dependent variables. Onset time from injury gave rise to patient sub-group classifications of acute, sub-acute and chronic pathology. Follow-up interviews regarding pain and activity levels were obtained from 2 to 8 years post-discharge.

Results: Patients were categorized into fully-compliant (FC) or partially-compliant (PC) based on adherence to the protocol. FC patients were discharged pain free on both passive and active tests in a median of 2.0 weeks (range <1 -6). Pain free passive stretch outcomes were achieved earlier in a median of 1.0 week (range <1-4). There were significant differences in response between the acute and chronic groups (p =0.01) with the acute achieving pain free active wall squat in median of 1.2 weeks (range <1 to 3) and chronic 2.4 weeks (range<1 to 7). Follow up was achieved in 58 patients with three recurrences (5%). There were significant differences in long-term responses between chronic and sub-chronic (acute + sub-acute) groups with the chronic group having higher incidences of pain when kneeling (82%:25%, p <0.01) and lower levels of sports return (29%:58%, p=0.28).

Conclusions: Results suggest this protocol is a simple, effective and non-invasive physical therapy intervention for OSD with recovery within 2 weeks, low recurrence

rates and good long-term results. Clear clinical markers are used for monitoring progress.

2.2 Introduction

2.2.1 Osgood-Schlatter's Disease

Over a century ago two orthopaedic specialists, Dr Robert Osgood and Dr Carl Schlatter, separately described a case presentation of an apophysitis condition at the tibial tubercle in young adolescents (Osgood 1903, Schlatter 1903). They reported patients with painful and swollen knees who appeared to have moderate to severe activity restrictions. At this first documentation it was noted that these patients were all at the adolescent growth stage and predominantly boys, but no further comments were made nor suggestions regarding its possible cause. Over the following century a variety of medical texts and articles have described the presentation and demographic data of OSD cases in different countries and clinical settings, but little scientific evidence is provided regarding either treatment recommendations or aetiology. Nearly all literature is based on professional experience and opinion rather than actual prospective or retrospective studies, but they all repeat similar advice - that the condition occurs during growth spurts, it is self-limiting requiring only symptomatic relief, and patients should grow out of it eventually (Ehrenborg 1962; Antich and Brewster 1985; Peck 1995; Harries et al 1995; Christopher and Congeni 2002, Bloom et al 2004; Domingues 2013; Tzalach et al 2016).

Incidence levels are difficult to ascertain due to the wide variation in reporting styles and patient populations and cohorts studied. De Inocencio (1998) reported 1000 consecutive paediatric visits to a primary care facility and stated that musculo-skeletal (MSK) pain represented 6 % of all visits – noting that over 70% were routine visits attributable to minor ailments such as ear, nose, throat and skin problems or routine health check-ups. Therefore, his finding that 6% from the 30% of non-routine care is due to MSK pain becomes more significant. Of the 61 patients who complained of MSK pain, 10% were diagnosed with OSD. Of course, this only takes into account a population sample of actual patients, not the general population of adolescents, but it does give an idea that the condition is relatively common. Foss et al (2014) documented all injuries amongst 268 female athletes in 5 schools in the United States

who played basketball, volleyball and soccer in one year. Disturbingly there was a 50% injury rate with 134 separate injuries recorded, of which OSD represented 10.4 % of injuries, or 5.2% of the sporting cohort. De Lucena et al (2010) investigated 956 school adolescents aged 12-15 in Brazil and found a 13% incidence of OSD in those who were sports active but only 7% in those who were more sedentary. This would suggest that levels of physical activity may be a risk factor for OSD, but it is not the only one. This is the only study that has looked at a matched age cohort to give a better overview of the true incidence in a normal population, not just an athletic one, however it omits the slightly younger cohort which may be the peak age for OSD in girls (11) and the rise to peak (12) in boys. Those authors who have looked at specialist cohorts such as training academies tend to show higher figures of between 3% (Price et al 2004) to 11% (Hirano et al 2002) and 14% (Dubravcic-Simuruijak 2003), but specialist groups are not indicative of the normal population and different sports may have greater impact on knee dynamics than others. Many other authors claim that OSD is one of the most frequent causes of knee pain in young athletes (Mital et al 1980; Micheli 1986; Wall 1998; Czyrny 2010) but do not give actual figures. There is also a great degree of different reporting and investigation styles which make meaningful analysis problematic. It would be much better if authors could report the overall population figures to improve analysis and relevance of their data. Overall it does appear that OSD may be a relatively common adolescent injury with actual incidence in the normal population possibly as much as 5-10%, and double that if the population are involved in sports activities to a greater extent than their school counterparts.

Patients have usually been reported as more commonly male and aged between 9-16 years (Ehrenborg 1962; Harries et al 1996; Gholve et al 2007), however gender imbalance may be related to levels of sporting participation rather than hormonal or anatomical factors. Smith and Tao (1991) found in their cohort of figure skaters that there was not a gender imbalance, possibly as both genders performed similar physical tasks. Foss et al (2014) only examined a female athletic cohort and found similar incidences amongst their players, so it is likely that the incidence levels are related to the type of sport played and levels of engagement, rather than gender differences. Increasing levels of female participation in sport throughout the world may show a more level distribution of OSD in the years to come.

There does appear to be a familial link with siblings of patients with OSD also having a high incidence of OSD (21%), and for those siblings who play sport the incidence is even higher at 32% (Kujala et al 1985). This may indicate a genetic component in the aetiology, possibly as growth levels and rates are influenced by hereditary elements (Rona 1981; Loesch et al 1995).

The typical presentation of OSD is one of gradual onset over a period of time, with increasing symptoms (Meisterling et al 1998), however Ehrenborg (1962) found that 48% of his patients recalled a sudden traumatic onset usually whilst playing sport. Typically, patients complain of pain localised to the top of their tibia, with a characteristic swollen and tender 'lump' over the tibial tubercle which may become permanent and can be disfiguring (Figure 2). The pain can range from mild to severe with patients' activity levels affected accordingly. Some patients struggle even with normal everyday activities e.g walking or climbing stairs or even pain at rest, but most patients find strenuous physical activity almost impossible without extreme pain. This therefore can have serious implications in those for whom physical activity is an important aspect in their lives, as the pain of the condition has a serious debilitating effect on their performance and enjoyment of that activity.

Patients may have OSD affecting one knee or both. Kridelbaugh (1948) found an incidence of 23% with bilateral lesions; to 48% in Jakobs study in 1981 and 80% in Wall's 1998 experience.

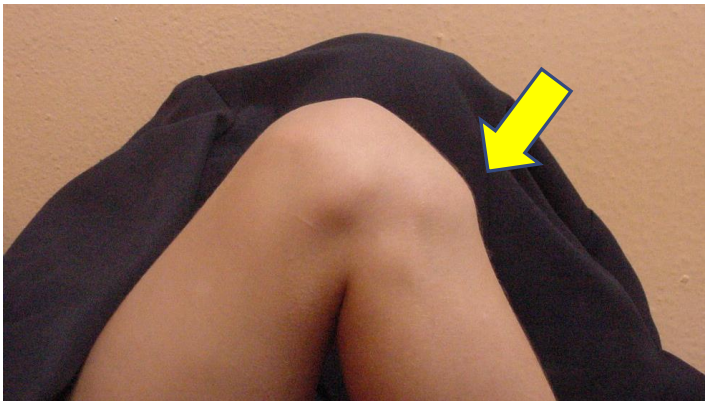


Figure 2. Inflamed tibial tubercle typically seen in Osgood-Schlatter's Disease (authors' photo)

Diagnosis is usually derived from simple clinical examination as the tender lump and age of the patient is highly indicative of OSD (Orava and Puranen 1978; Antich and Lombardo 1985; Duri et al 2002; Bloom et al 2004). Lau et al (2008) noted that 80% of their OSD patients had localised tenderness but only 61% complained of anterior knee

pain. Use of radiology can be used to exclude rarer differential diagnoses such as neoplasm (osteosarcoma), proximal tibial stress fracture, referred pain from a slipped capital epiphysis of the hip, or infection (osteomyelitis, cellulitis) (Pappas 1967; Antich and Brewster 1985), but exposure to X-ray near growth plates should be avoided whenever possible, and early stage cases of OSD do not always reveal bony fragmentation. MRI scans (magnetic resonance imaging) are very expensive but do however indicate the presence of soft tissue swelling over the tibial tubercle and can therefore be more accurate than X-rays (Hirano et al 2002; Chang et al 2013). The more recent widespread availability and cheaper cost of ultra-sound imaging has offered the opportunity to improve the documentation of staging of OSD and this seems to be an area with quite prolific literature (de Flavis et al 1989; Lanning and Heikkinen 1991; Bergami et al 1994; Yasher et al 1995; Aparicio et al 1997; Blankstein et al 2001; Mahlfeld et al 2001; Demirag et al 2004; Czyrny 2010; Kaya et al 2013; Lazovic et al 2013). However, most diagnoses will be made clinically by a child's family doctor or other primary care practitioner such as a Chartered Physiotherapist, without the need for further investigative procedures (Wall 1998; Micheli 1987; Halibasic et al 2012; Kabiri et al 2014).

MRI staging of the natural progression of OSD has been described by Hirano et al (2002) in their study of young male academy footballers (Table 1). They proposed 5 stages from the imaging findings with progressions either to the next more severe stage or healing stage. All patients had conservative intervention treatment, so the natural history could not be strictly verified.

Table 1. Pathological staging and levels of Osgood-Schlatter's disease (Hirano et al 2002)

Stage/ Level	**Secondary ossification centre**	**Patella tendon**	**Resolved**
Normal I	Centre appears		100%
Early II	Swelling at tibial tubercle		75%
Progressive III	Tear detected/ partial avulsion	Swelling in tendon	70%
Terminal IV	Complete detachment of fragment(s)	Thickened insertion and tendon	0%
Healing V	No separation	Thickened insertion and swelling in tendon	100%

Although 20% of these patients were lost to follow-up there appears to be a direct relationship between the stage of initial presentation and the possibility of reaching a state of healing, with those in a more advanced stage having less overall resolution, and those who had already progressed to a terminal stage with detached fragments did not heal at all during the 18-month study.

Recovery takes months to years (Kujala, Kvist, & Heinonen 1985; Soprano and Fuchs 2007), and is characterised by recurrent episodes. Length of suffering may also be affected by activity levels e.g. sporting vs sedentary adolescents. The natural history of OSD was recorded by Krause et al (1990) and far from it being a benign short–term condition as reported by most professionals, they found that 60% of the 50 patients questioned continued to have pain whenever kneeling, and 10% of patients require surgery to remove bone fragments or splinters that remain imbedded in the tendon (Mital et al 1980; Binazzi 1993; Orava et al 2000). A very few OS patients, suggested as a rare occurrence, may go on to completely avulse the tibial tubercle causing a fracture which requires internal fixation (surgery) to resolve it (Schiedts et al 1995). A pseudo-arthrosis between the detached ossicle and the tibial tubercle has also been described by Robertsen et al (1996) in a case report of an eighteen-year old male who continued to have symptoms for more than 3 years. The study by Ross and Villard (2003) in young adult men with a mean age of 20 (N = 50) found a significantly higher level of ongoing disability as measured by the Knee Outcome Survey Activities of Daily Living (ADL) and Sports Activity Scale in those who had suffered from OSD in the past.

The natural history shows many patients struggling along with their symptoms for some time before seeking treatment with Antich and Lombardo's study (1985) of 75 patients indicating that 72% had a duration of the condition for over 2 months with almost 30% present for over 1 year. They also noted that very few patients sought treatment within the first 2 weeks. The condition is also highly likely to recur, and patients find themselves in a cycle of remission and relapse, further prolonging the impact of the condition (Gholve et al 2007). Other authors have documented the condition lasting for many years (Bloom et al 2004; Antich and Brewster 1985; Flowers and Bhadreshwar 1995; Ehrenborg 1962; Kaya et al 2013; Loher et al 2012; Mital 1980) and as previously mentioned some patients will have permanent problems affecting them into adulthood (Ross and Villard 2003; Gholve et al 2007). Krause et al (1990) found when describing

the natural history of OSD that some 60% of previous patients could not kneel without pain, although they did not state whether these ex-patients were currently physically active or not. Some patients that have developed OSD to Stage IV find the detached ossicle of bone continues to give significant pain and disability requiring surgery for removal and resolution, but this is rarely performed before the end of their adolescence, again leaving these patients suffering for even more years (Bosworth 1934; Engel and Windhager 1987; Binazzi 1993; DeBerardino et al 2007; Lui 2013; Weiss et al 2007). Many patients will be left with enlarged and unsightly tibial tubercles for the rest of their lives (Figure 3).

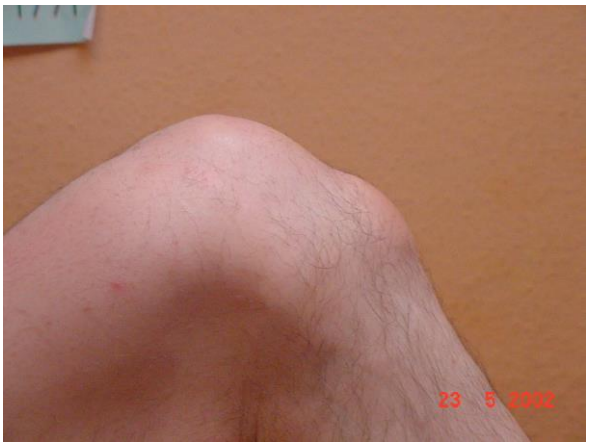


Figure 3. Residual enlarged tibial tubercle in an adult – 15 years post-OSD (author's photo)

Interestingly OSD may also occur across the species with reported similar occurrences in horses (Kold 1990; Oikawa and Nirami 1998) and dogs (Power 1976; Skelly et al 1997; von Pfeil et al 2009; Stigen and Mikalsen 2009) and most recently rabbits (Nehrbass et al 2014). These studies typically describe OSD-type lesions in athletic adolescent animals i.e. race horses and greyhound dogs, or those with explosive powerful actions like rabbits.

No studies have been performed into the aetiology of OSD. However only juveniles going through their secondary growth spurt are affected so growth is one factor agreed by authors (Ogden 1976; Smith 1991; Dalton 1992; Harries et al 1994; Outerbridge and Micheli 1995; Maddon and Mellion 1996; Suzuki 2001; Antosia and Lyn 2002; Brukner and Khan 2012; Petersen and Renstrom 2017). Theories based on observation have been expressed by various authors with some consensus regarding 'traction forces' overwhelming the softer apophysis causing the condition (Micheli 1987; Katoh 1988; Dalton 1992; Maddon and Mellion 1996; Brukner and Khan 2012; Petersen and

Renstrom 2017) however there have been no investigations into what has caused those traction forces, nor studies into other factors. A recent study by Nakase et al (2016) has found some risk factors associated with OSD including hypertrophied and tight quadriceps muscles, and this would strengthen the previous findings mentioned that athletic children are more likely to be affected. Limited flexibility has been observed by Mehdinasab & Fakoor (2005) in 45% of their patients but they also found reduced flexibility in other muscles indicating that it was not an isolated muscle issue. Ikeda et al (2001) and Tzalach et al (2016) found quadriceps tightness in all their OSD patients, yet De Lucena et al (2012) found 75% of their adolescent population had tight quadriceps but only 10% had OSD, so tightness in the quadriceps is unlikely to be the only factor. Other factors proposed include biomechanical abnormalities, footwear, playing conditions, over-weight or degree of skeletal maturity have not been investigated, only commented on as observations (Willner 1969; Jakob 1981; Sen 1988; Dalton 1992; Outerbridge & Micheli 1995; Aparacio et al 1997; Gigante et al 2003).

There is also no consensus as to the best treatment, but conservative management is usually recommended (Bloom et al., 2004), although no studies have been performed to test the efficacy of this approach. Standard advice is based on generic symptomatic relief using ice, medication for pain and inflammatory relief, and modifying activity levels to the patient's pain tolerance levels i.e. self-limiting, and waiting for skeletal maturity to develop (Ehrenborg, 1962; Stanitski 1993; Bruckner & Khan, 2012). However, no prospective studies on this standard advice have been performed (Bloom et al., 2004) so there is no proof of this approach being more effective than natural resolution with time. This current approach can result in chronic suffering for 1-2 years and recurrence is common, furthermore up to 10% of patients may need surgery to remove bone splinters (Antich & Brewster, 1985; Gholve et al., 2007). Only five intervention studies into OSD have been published to the authors' knowledge, of which only one was a randomized controlled intervention with a comparator group (Topol et al 2011), but all studies had a high risk of bias (Cairns et al 2018). Ehrenborg's study (1962) used plaster cast immobilisation and found a mean of 14 months for recovery in the plaster group and 28 months for the non-immobilised group, but both these groups had significantly poor responses in 33% and 50% respectively. Trail (1998) describes a retrospective non-randomised parallel study design using surgery or a mixture of

plaster casting, injections and physiotherapy, and found little difference in final outcomes based on feedback after 4-5 years from patients. The added complications of surgery led to their conclusion that surgery was not justified in the treatment of OSD (Trial 1998; Cairns et al 2018). Levine and Kashyap (1981) used intermittent infrapatellar bracing with good results in 6-8 weeks in their small study, and Topol et al. (2011) used dextrose injections with chronic cases and showed some promise with a return to sports within 3-6 months. Nakase et al. (2016) have repeated these injections over 3 months and found pain relief in both the control (lidocaine/saline) and lidocaine /dextrose groups but time to full recovery was not stated. Loher et al. (2012) used extra-corporeal shock wave treatment but only described the 5-year outcome, by which time many of their patients would assumed to have resolved naturally, so its efficacy has yet to be proven. Most of these studies have not used control groups nor used objective measures for determining recovery or progression, however Nakase et al. (2016) used the VISA index of severity (Visentini et al., 1998) designed for adults with patella tendinopathy, which gave a good evaluation of knee function. No studies have looked at the effect of massage, either at relieving symptoms or enhancing recovery time in OSD.

OSD therefore is a common overuse injury for adolescents, with limited efforts having been made to investigate its cause, effect or treatment options, leaving many young athletes struggling to cope for a prolonged period of time. Despite having been documented for over 100 years, there is little more than opinion to direct our understanding and much repetition of that opinion in the literature without validation or questioning. A structured approach does not appear to be been taken with any treatment studies on OSD, but only reactive ones based on symptomatic relief. No observations or theories have been proposed and tested, and there is little commonality and poor consensus. With the acknowledged chronic painful history and slow recovery, the impact of this debilitating injury on young adolescents should not be under-estimated. Overall there does not appear to be a consistent overall rationale for treatment selection for OSD nor consensus as to its form, and determination of efficacy relies on subjective pain self-reporting or professional opinion.

2.2.2 Clinical Evidence-based practice

None of the published studies appear to have followed clinical evidence-based practice (EBP) (Sackett et al 1996), which integrates the best research evidence available i.e. a systematic and critical review of literature, patient values and preferences, as well as clinical expertise, but have relied largely on clinical experience and a symptomatic approach to treatment. The integration of all three (best research evidence, patient values and clinical expertise) provides the best route to decision making for the improvement and development of patient care, and optimization of clinical outcomes and quality of life (Sackett 2002) and has been adopted by the World Confederation for Physical Therapy as a commitment to inform decision-making in patient care (World Confederation for Physical Therapy-European Region, 2015). The steps involved in clinical EBP are usually triggered by patient interaction and an enquiring mindset and clinicians are encouraged to question treatment efficacy and seek improvements throughout their professional lives as a basis for EBP (Sackett 1997; NICE guidelines; CSP). There are five steps to good EBP suggested by Sackett et al (2000) (see Table 2). It is these clear steps that will be demonstrated by the clinical responses in the following study on OSD.

Table 2. Evidence-based practice steps (summarised from Sackett et al 2000)

1. Create the question	Construct clinical question from the case
2. Acquire evidence	Select and search appropriate resources
3. Appraise the evidence	Appraise evidence for validity and applicability
4. Apply	Integrate evidence with clinical expertise and patient preferences, and apply to practice
5. Evaluation	Evaluate performance with patient response

In this specific case of developing a novel treatment approach for OSD using EBP, the patients would be assessed clinically for subjective symptoms such as pain and effect of the problem on their functional daily lives e.g ability to play sport, walking, or climbing stairs. Objective signs would be measured or noted including enlarged swollen lumps at the tibial tubercle, flexibility, current growth spurt and activity levels. This assessment leads to the formation of pertinent foreground questions such as the role of reduced flexibility in increasing traction forces and pain in OSD, and the efficacy of current treatment advice.

2.2.3 Development of theory

It is known that when treating an overuse injury that it is important to look at identifying and managing the causative elements as a direct and vital component of treating the condition (Renstrom 1994), but how does one do this if the cause is still unknown? Two common elements for AS appear to be agreed upon by all doctors –firstly the presence of a soft growing epiphysis (the secondary growth zone of bone that the apophysis attaches to); and secondly the stronger traction forces that cause the weaker apophysis to pull away resulting in chronic apophysitis.

It was not known if the flexibility restrictions and high activity levels observed by the researcher in their growing OSD patients were just the effect of the condition, i.e. a symptom, or whether they were part of the cause. A logical theory was developed that tight muscles could be a cause of passive tension forces pulling on the apophysis, causing adverse traction, and high activity levels could be responsible for active tension forces. Combined, the passive and active forces may overwhelm the softer growing apophysis junction causing the disruption of the tendon insertional fibres and the pathological reaction of apophysitis. In the pre-adolescent child, there is no weak epiphysis, so traction forces are not a problem, it is only during the secondary growth spurt that AS occur, so it is directly related to the existing conditions at that time. When the equilibrium is unbalanced between forces and capacity then the condition could occur.

2.2.3.1 Traction forces

OSD patients present with a number of typical features including shortened quadriceps muscles (de Lucena et al 2011; Nakase et al 2015) and clinical observation by the main author also noted pain at the tibial tubercle upon passive stretching. Patients also tended to have high physical activity levels and pain during or after exercise (Antich and Lombardo 1985; Krause and Williams 1990; Gholve et al 2007; Jayanthi et al 2015). These forces could contribute to the traction mechanism behind the injury (Krause and Williams 1990; Brukner and Khan 2012; Kabiri et al 2014). It is suggested that these could be further categorised into passive and active traction forces. Passive and active stress tests are commonly used as part of clinical assessments to indicate injury severity and healing response (Hayes and Peterson 2003), therefore they could also be used to ascertain the loading tolerance of the apophysis, and thus progression and healing response in OSD patients. Reducing or eliminating the traction forces on the tendon

may reduce symptoms such as pain and should also facilitate healing, and logically could become curative.

The active and passive components of a muscle consist of contractile and elastic components (See Figure 4). The active contractile elements are the actin and myosin cross-bridge structures, whereas the passive elastic components consist of parallel connective tissues such as titin molecules, epimysium, perimysium and endomysium within the muscle fibre, and the tendon connective tissue in series with the muscle fibres.

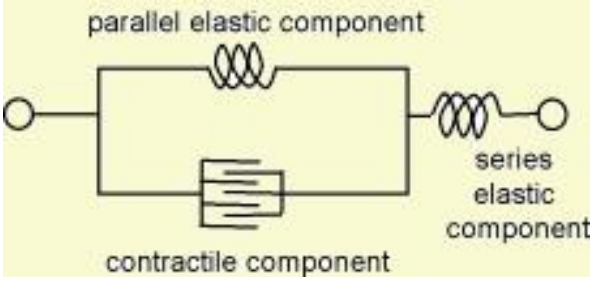


Figure 4. Mechanical model of a muscle fibre showing active (contractile) and passive (elastic) components (adapted from Hall (2012) page 149).

As muscle tissue is elongated the different components develop tension at different times with resting length determined by the onset of passive tension (Figure 5), hence resting length of a muscle can indicate the underlying point of passive tension in the elastic components (connective tissues) of the muscle (Hall 2012).

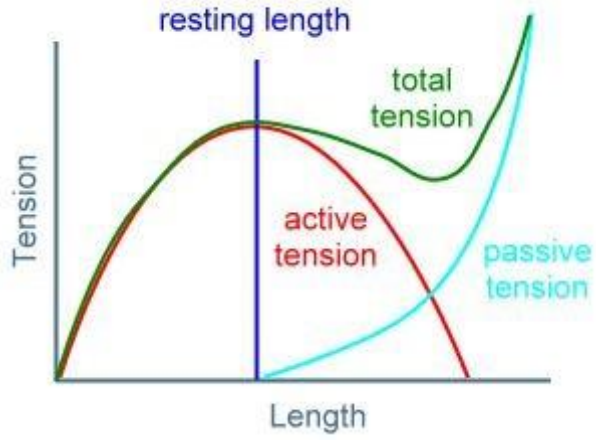


Figure 5. Length - tension curve of muscle tissue (adapted from Hall (2012) page 165

2.2.3.1.1 Passive forces

Stretching would normally be recommended to improve flexibility and reduce passive tension but in this situation, it is counter-productive, as it would further increase the traction of the apophysis, eliciting pain and possibly exacerbating the condition. An alternative method using massage which does not compromise the apophysis site, could be used, and massage has not been previously investigated as an intervention for OSD to the authors' knowledge. Myofascial release massage (St. George 1989) (MRM) has been used effectively by physiotherapists to lengthen tight myofascial units in many conditions, most notably with ilio-tibial band syndrome (Fredericson and Weir 2006). Once the tolerance had improved to enable a quadriceps stretch with no pain felt at the tibial tubercle, then normal stretching could safely commence instead of massage, to continue reducing passive tension on the apophysis and increasing flexibility further. Many authors suggested stretching as part of a treatment programme (Antich 1985; Micheli 1986; Smith 1995; Cassas 2006; Gholve 2007; Duri 2002; Chang 2013; Tzalach et al 2016) but these were recommendations based on expert opinion, not results from a prospective study.

2.2.3.1.2 Active forces

Moderating active loading is usually advised as part of the standard approach to OSD treatment as a way of self-managing provocative activities (Meisterling et al 1998; Gerulis et al 2004), but of itself appears not to be sufficient, as the described resolution still takes many months to years (Ehrenborg 1962; Meisterling et al 1998; Gholve et al 2007). Total immobilisation of the knee using plaster casts had limited success (Ehrenborg 1962) but physical activity levels in patients could be minimised. This would allow time for tissue healing and avoid the stronger traction of the tendon during intermittent physical activity. It would be counter-productive to address only one aspect of the tensile loading on the apophysis i.e. passive but not active. Active loading could be objectively tested using a closed chain squat which increases the force in the quadriceps as the knee flexion angle increases and the patient descends eccentrically (Escamilla 2001). Wall squats are a variation that have been recommended in the rehabilitation of OSD (Meisterling et al 1998).

2.2.4 Alternative treatment model

There is a need for a quicker and effective treatment that responds to the clinical signs of the OSD patient and provides a logical model for intervention selection and monitoring progress, based on the passive and active force tolerance of the apophysis. Progress can be objectively monitored to give patients and practitioner on-going feedback as to the treatment's efficacy. Considering the high prevalence of OSD and lack of studies, it is important to consider clinic-based interventions where most patients are likely to be treated.

A new model was theorised by the researcher as to the cause of increased tension observed and thus allow for a more directed and targeted treatment approach to be formulated (Table 3).

Table 3. New aetiological model for Osgood-Schlatter's Disease

AS factors	**Cause of Increased tension**	**Observed Effect of Decreased tolerance**
Passive tension	Adolescent growth spurt Flexibility decreased	Pain at apophysis on passive stretch
Active tension	High physical activity levels Sports played	Pain during/ after activity at apophysis

The growth of the child is something we can have little active influence over (Rona 1981). Children's growth is largely determined by genetics and health or nutrition factors (Tanner 1962; Malina et al 2004) and so is not a factor we can directly manipulate as part of an intervention strategy. However physical loading is also a factor of skeletal growth (Malina et al 2004) and we can influence the load placed on the apophysis i.e. the traction forces.

To reduce the forces from the quadriceps muscle would need a dual approach of addressing both passive and active aspects. A reduction in activity levels by resting is a common approach for many injury treatments and is already promoted as part of the standard advice for OSD. However as previously described it is only partially successful and the condition has a high recurrence rate and poor outcomes. Total rest by immobilising the leg in a plaster cast has also been advocated but again has poor outcomes measures and high recurrence (Ehrenborg 1962). Holding a muscle in a shortened position for a period of time has also been shown to increase its tightness and tension (Gossman et al 1982), and with the patient continuing to grow could further

increase the passive tensile element, even though the active element was controlled. This could be a reason behind the poor response and high recurrence rates once plaster casts were removed. It appears that moderating the active element is not enough of itself. To reduce the passive forces from the tight quadriceps muscle it would normally advocate a stretching programme but in this unique situation would be counter-intuitive as it would only serve to increase the traction forces further, thus exacerbating the condition. An alternative method would be needed to reduce the passive tension but not cause further traction and thus re-injury to the apophysis.

Massage has long been a tool used by physiotherapists to influence the tone or tension in soft tissues. This intervention could be used by applying the massage directly to the tight muscle belly itself, without compromising the apophysis injury site, thus reducing tension without increasing the traction. A common technique used for lengthening muscle directly is myofascial release massage (MRM) (MacDonald et al 2013) and this intervention had not been previously investigated in the treatment of OSD to the researcher's knowledge.

2.2.4.1 Massage

Although massage has been used by health professionals for millenia, there is much disagreement in the literature as to the proven benefits (Gasibat and Suwheli 2016). Traditional therapeutic manual massage is based on apllying mechanical pressure on muscle tissue with the aim of reducing adhesions within the tissue and improving muscle-tendon compliance and reducing stiffness (Magnusson 1998; Gasibat and Suwehli 2017). There are various forms of massage including Pettrisage (skin rolling or deep kneading), Effluerage (longitudinal light stroking), Tapotement (percussive striking), Frictions (deep tranverse or circular pressure) and Vibration (shaking) (Bell 1964; Goats 1994; Brummitt 2008). Each technique has a suggested clinical advantage from stimulating the nervous system and enhancing venous return (efflurage) to deeper muscle mobilisation and adhesion release (petrissage and frictions), and stimulation of tissues either directly or by reflex response (tapotement) (Gasibat & Suwehli 2017). Therefore, the choice of massage technique is dependent on the required or desired outcome. Brummitt (2008) highlights that many practitioners have a poor appreciation of the evidence behind each technique and therefore selection may be inappropriate. Many studies use a range of techniques making comparisons even more difficult e.g. Hopper et al (2005) used 'classic' massage as the control technique but this included

three different types of massage This could be a reason behind the poor results and inconclusive findings of some massage research.

The possible effects can be grouped under four systems that may be affected: biomechanical, physiological, neurological and psychological (Weerapong et al 2005; Brummitt 2008; Field 2016).

1. Biomechanical: The literature suggests that there may be an increase in muscle compliance which in turn could influence joint range of motion (ROM). This might be achieved by mobilising and elongating shortened or adhered connective tissue but there remains a lack of evidence (Weerapong et al 2005). Field (2016) describes studies where an increase in ROM was demonstrated and Hopper et al (2005) also found increases in flexibility although these were not retained after 24 hours. Swelling has been shown to decrease after massage (Goats 1994) but light effluerage was found to have no significant change in passive stiffness (Stanley et al 2001) however this may have been the incorrect technique chosen (Gasibat and Suwheli 2017). Weerapong et al did not find any studies on the effects of massage on active stiffness (2005).

2. Physiological: There is a paucity of good quality literature with papers offering opinion rather than evidence. Blood pressure changes have been observed dependent on the type of massage technique used (Cambron et al 2006), as well as hormonal changes which may decrease pain perception and enhance relaxation (Weerapong et al 2005; Field 2016; Gasibat and Suwheli 2017). Blood flow and skin and muscle temperature have been shown to increase (Goats 1994; Weerapong et al 2005; Gasibat and Suwheli 2017) after massage.

3. Neurological: Changes to neural excitability via the Hoffman reflex from deep massage has been suggested (Weerapong et al 2005; Gasibat and Suwheli 2016), as well as a reduction of pain and muscle spasm caused by local inhibition at the spinal cord via the pain gate theory (Melzack and Wall 1965). Field (2016) has also described studies where possible stimulus of the vagus nerve and cortisol levels were decreased and increases in parasympathetic activity were found with higher endorphin levels (Kaada and Torsteinbo 1989) which could lead to a reduction of pain.

4. Psychological: Feelings of well-being and lowered anxiety levels as found by improved profile of mood state questionnaires have been reported (Kaada and torsteinbo 1989; Leivadi et al 1999; Brummitt 2008), and these may be a direct result of hormonal changes already described.

Overall there are mainly theories and supposition as to the effects of massage, and studies done report inconclusive results. There is considerable variation on massage timing, duration and depth of specific technique and review authors have called for more clarity and uniformity to improve the quality of studies and improve our understanding of the specific effects of massage (Weerapong et al 2005; Brummitt 2008; Field 2016; Gasibat and Suwheli 2017). This would improve the selection and the appropriate technique for the specific outcome desired, and thus allow for meaningful comparisons and better application to patient problems, thus enhancing patient care (Field 2016).

2.2.4.2 Stretching

The evidence behind the use of stretching as a therapeutic intervention is also lacking in scientific rigor, although it is in common use within healthcare professions such as physiotherapy as a tool for regaining tissue extensibility post-injury and improving joint ROM (Frietas et al 2018). The actual effect of stretching on the structural properties of the muscle-tendon unit remain uncertain although theories have been proposed. Freitas (2018) has suggested that the mechanisms may be either mechanical or sensory, with Andrade et al (2016) noting that not only musculo-skeletal tissue can be affected but neurological tissue as well in the form of peripheral nerve tension. Changes in tissue stiffness, joint ROM, have been proposed as possible mechanical effects Magnusson 1998; Shrier and Gossal 2000; Frietas et al 2017), and changes to stretch tolerance and decreases in neural excitability via the Hoffman reflex have been suggested (Latash 1998; Avela et al 1999). Barnes and Kilding (2015) found that stretching might improve running economy, possibly by allowing greater swing phase range and thus stride length. Reducing muscle tension would also allow for less resistance to antagonistic muscle contraction resulting in less effort and energy required to produce movement (Hall 2012).

Commonly three types of stretching techniques are in use and these are static, ballistic and proprioceptive neuro-muscular facilitation (PNF) (Magnusson 1998; Vujnovich 1996; Shrier and Gossal 2000; Konrad 2017). Each type is suggested to have a slightly different effect, but this is also dependent on timing, duration and strength, variations of technique application in fact similar to massage (Shrier and Gossal 2000; Konrad 2017). There have been many studies published on the benefits and risks associated with stretching but again the literature is inconclusive with methodological inconsistencies which hamper direct and clear comparisons (Gleim and McHugh

1997). Increases in ROM have been found with all three types, but timings of stretches may have had more impact (Konrad et al 2017). Different timings may affect different neurological reflexes with short duration holds (< 5 second) possibly stimulating the stretch reflex and increasing tissue tension, and longer holds (>8 seconds) stimulating the golgi-tendon organs and thus producing tissue relaxation via the inverse stretch reflex. Konrad et al (2017) therefore postulated that longer stretch holds would result in increases in joint ROM and a decrease in muscle stiffness. Their results found that the type of stretch applied did not seem to matter as long as they were held for 30 seconds with 4 repetitions. In contrast to this study Mahieu et al (2007) found that the type of stretch made a significant difference to passive resistance (Static stretching) whereas ballistic stretching changed stiffness but not passive resistance. Nakamura et al (2011) also found static stretches increased ROM and decreased passive torque and MTU stiffness, but that these changes were only apparent for up to 10 minutes post-application and did not change the fascicle length which could indicate a change in the muscle's passive structure. Konrad et al (2017) suggest that static short duration stretches (1-2 minutes) may affect muscle tissue whereas longer duration of up to 10 minutes may affect tendon properties. Overall PNF stretching appears to be the most effective type for increasing ROM but the technique involves an eccentric contraction of the muscle and a change in stretch tolerance due to analgesia which may enhance performance but increase the risk of injury (Shrier and Gossal 2000). Therefore, stretch type should be carefully chosen so as to obtain the desired effect without compromising the athlete's safety, but more evidence of the specific effects of the different types is still needed to enable clinicians and others to prescribe accurately (Gleim and McHugh 1997; Magnusson 1998).

Duration of intervention varies greatly between studies with 15 second holds up to 5 minutes (Nakamura et al (2011), but the most commonly suggested is 30 seconds (Shrier and Gossal 2000) and Madding et al (1987) did not find any difference between durations of 15, 45 or 120 second holds on hip flexibility. Shrier and Gossal (2000) suggest that the effect may depend on the visco-elasticity of the particular muscle group, and this may explain the great variety of responses.

Frequency of stretching is also variable with some authors suggesting a single 30 second stretch per day (Bandy and Irion 1994) was as effective as three 30 second stretches (Bandy et al 1997) however there appears to be a plateau effect after seven

weeks (Borms et al 1987). Shrier and Gossal (2000) therefore suggest that 30 second stretches for six to seven weeks are likely to be the most effective, and Magnusson (1998) found programmes needed to be at least 3 weeks in duration to gain increases in ROM. Furthermore, the number of repetitions needed for effectiveness appears to be muscle and possibly individual-dependent (Madding et al 1987), with continuous stretching and repeated cyclical stretching being recommended (Starring et al 1988; Bandy et al 1998)

Not only is the type of stretch variable in studies but the strength applied to the stretch is also inconsistent with some authors suggesting the limitation of the stretch to be based on discomfort felt (Shrier and Gossal 2000; Konrad et al 2017), constant torque value (Kato et al 2010) or progressing changing angle of the joint (Kay et al 2015).

Temperature of the environment or tissue can also affect tissue elasticity (Gossman et al 1982) but again there is conflicting evidence with both cold and heat application showing increases in ROM but Shrier and Gossal (2000) suggest this may be due to the analgesic effect of heat or cold rather than decreased stiffness, which would back the previous finding of pain being the main limiter to the end of range (Konrad et al 2017).

The effects of massage and stretching may therefore be a combination of mechanical, neurological, physiological and psychological, but there is still much research needed to identify the mechanisms behind the different forms and varied application of each type and, in particular their effects on different tissue types. Their impact on pain should also be acknowledged as this study uses pain as an important indicator for treatment effectiveness. Whilst massage has been shown to decrease pain, the mechanism is not fully understood. (Gasibat and Suwheli 2017) suggest this effect may be due to the pain gate mechanism at the spinal cord where larger faster nerve fibres are stimulated from the mechano-tactile stimulus, and these over-ride the smaller and slower pain fibres. Stretching has also been shown in the previous section to have analgesic effects with changes to stretch tolerance and decreases in neural excitability via the Hoffman reflex (Latash 1998; Avela et al 1999). But pain too is a multi-faceted sensation and experience which is unique to the individual (Rio et al 2013). Although there is general acceptance of Melzack and Wall's pain gate theory (1965), the actual mechanisms behind pain perception and tolerance remain a significant challenge to science and medicine in particular. Pain is a subjective experience and can be affected by an

individual's psycho-social, cultural, emotional and health state, as well as coping strategies, habits and influences (Rio et al 2013). People can react differently to the same stimulus with some having stronger reactions than others. Sullivan et al (1995) has noted that a form of pain catastrophising can occur in individuals who dwell on their pain, exaggerate their pain or its severity, and also have a feeling of helplessness about how to deal with it. In the younger individual with a limited experience of pain it may be difficult for them to gauge relative levels of pain (Scharfbillig et al 2009) and there could be a propensity towards catastrophisation, especially as health professionals may focus on pain as the main symptom. Practitioners need to be aware of the unique perspective of young patients and help allay fears to reduce the likelihood of pain catastrophising (Sullivan et al 1995). The new treatment protocol model was developed to address both passive and active elements in the aetiology model together with monitoring flexibility in the quadriceps muscle and is summarised in Figure 6. This created a dual pathway of passive and active tests to see how the patient responded to the treatment. It also provided for clear markers for treatment selection.

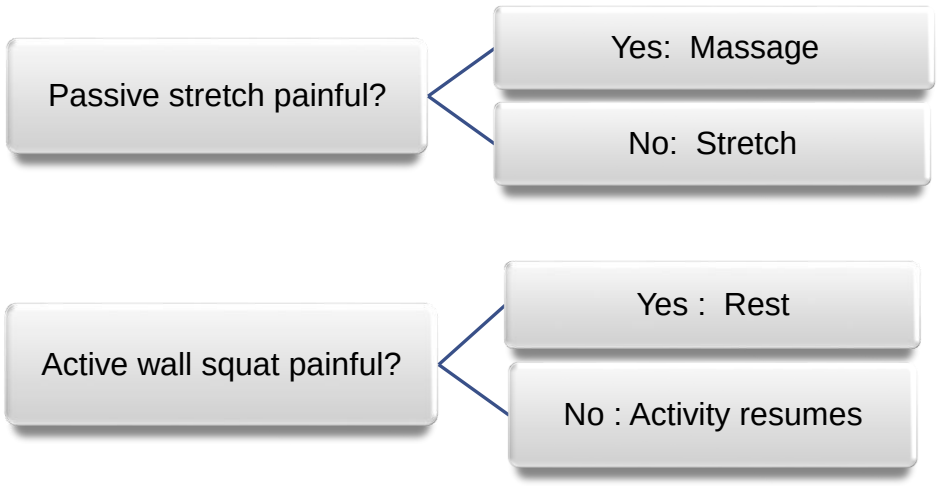


Figure 6. New Passive- active test and treatment model

By reducing both passive and active elements concurrently it was anticipated that there would be less tension in the muscle-tendon unit, which would reduce tissue deformation at the apophysis and allow for healing, thus leading to faster recovery and reducing pain.

2.2.5 Aims

The aims of this paper are to present a new evidence-based model founded on clinical observation and reason, based on reducing passive and active tension on the apophysis in AS. This model is then used as a basis for the development of an effective treatment protocol for OSD, and also provides for simple objective tests that can be used to quantify and monitor the progress and effectiveness of treatment in a clinic setting with minimal resources.

2.2.5.1 Hypotheses

H_0: The new treatment protocol will not impact on patient symptoms and recovery for OSD patients when compared with the natural history

H_1: The new treatment protocol will result in symptom relief and recovery for OSD patients when compared to the natural history

2.3 Method

2.3.1 Setting

A case series is presented of 75 OSD patients assessed, monitored and treated in a private sports clinic in Kent, United Kingdom by an experienced Chartered Physiotherapist. Consecutive OSD patients were either self-referring or referred for physiotherapy by medical practitioners to the clinic for physiotherapy treatment, which was paid for privately or by health insurance. Every successive OSD patient who attended for treatment was invited to try the new protocol as opposed to the general symptomatic advice, and parents/ guardians and the patients themselves gave informed consent (Appendix I, II and III). All patients volunteered to receive the new protocol. Inclusion criteria was based on initial diagnostic findings by the clinician of a tender and enlarged tibial tubercle, pain at the apophysis site at the tibial tubercle on or after physical activity, and typical age of pre/ adolescence from 9 – 16 years old (Malina et al 2004) . Furthermore, some patients also had radiographic evidence of an enlarged tibial tubercle confirming OSD. Patients could present with a unilateral or bilateral condition. Exclusion criteria was any concurrent non-related injury e.g. patella-femoral joint syndrome which could present with similar pain presentation, together with awareness of differential diagnoses of osteosarcoma, osteochondritis and infective arthritis. Patient demographics were recorded as well as physical activity levels prior to onset (hours per week), hours of physical activity were determined by adding up formal sports activities such as training, competitions and Physical Education (PE) lessons.

Time from the onset of their condition gave rise to sub-groupings of acute, sub-acute and chronic stage, based on the stages of healing (Norris 1998).

Ethical approval was gained by both the local area Health Authority (National Health Service) and University of Greenwich Ethics Committee, and these constituted part of the Research Ethical approval for the researcher's PhD study. Patients were classed as vulnerable due to their age so full ethical approval was required, as well as enhanced Disclosure and Barring criteria for the researcher. Parents and patients were also provided with information and given opportunity to ask questions prior to commencement of treatment, including their treatment participation as parents/ carers (Association of Paediatric Chartered Physiotherapists, 2018). They were both required to give verbal assent and signed consent.

2.3.2 Outcome measures

Time to recovery was the primary dependent variable (DV) used (DV1) and was determined by pain free completion of the passive and active tests (secondary outcomes DV2 and DV3), as assessed by the therapist, and pain free activities of daily living as reported by patient and parent e.g climbing stairs and squatting. Baseline self-reported numerical pain scores for activities of daily living were recorded at the beginning of each clinic visit for overall feedback, using a simple numerical VAS pain scale (Williamson and Hoggart 2005), however for the secondary outcome tests a simple threshold of onset of any pain (Yes/No) was used. Quantity of pain was not considered due to subjective differences between patients and ethical considerations, so a simple measure of presence or absence of pain on stressing the apophysis with a traction force was used. A dual approach was used to approximate passive and active tensile forces and two secondary tests were used to assess the effect of those forces on the apophysis. All tests were assessed and scored by the therapist, and the results of the tests determined the specific intervention selected for the treatment protocol (IV1) (Figure 7) and any decision to progress was taken by the therapist.

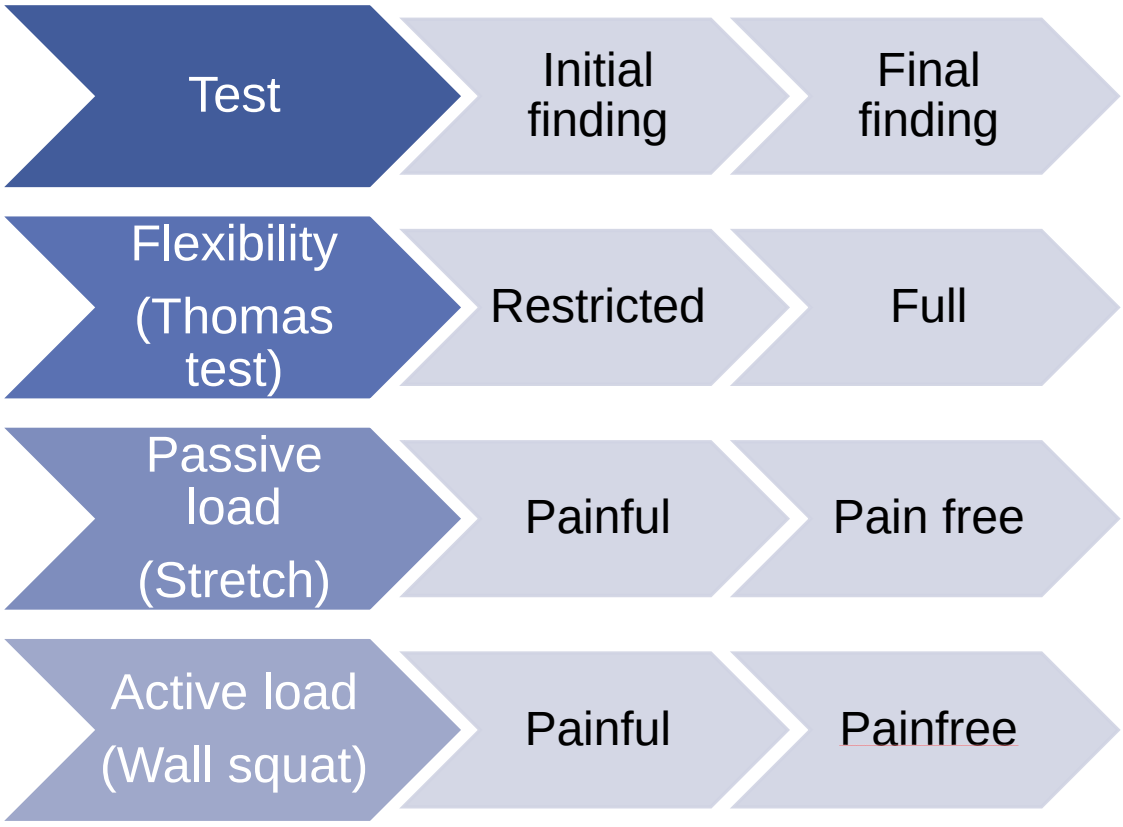


Figure 7. Treatment protocol flowchart

2.3.2.1 Monitoring Passive tension

Monitoring of passive muscle tension was also measured at the beginning of every clinic session by the experienced researcher-clinician using the Thomas test position (see Figure 8) for rectus femoris resting tension (Kendall et al 2005; de Lucena et al 2011). The resting length marks the muscle length at which passive tension begins to develop (Hall 2012) (see Figure 8). Knee flexion angle was measured using a standard manual goniometer (12" Baseline model 360˚ clear transparent, calibrated to international standards of measurement), and repeated 3 times with the mean recorded. The knee and hip were passively repositioned back to neutral (0˚) between tests so that each measure started from the same base position (Kendall et al 2005). Intra-tester error of measurement with the goniometer was previously assessed as ±3˚, which is similar to those found by Ellis and Stowe (1982). Other studies have found high reliability with intra-tester measurements of the knee (Rothstein et al 1983; Gajdosik and Bohannon 1987; Cejudo et al 2015). Angles of less than 60° were regarded as an indicator of tightness and angles of more than 80° within normal limits (Sahrmann 2002; Kendall et al 2005; De Lucena et al 2011). Subsequent measures were indicative of progression and enabled patient and parent feedback.

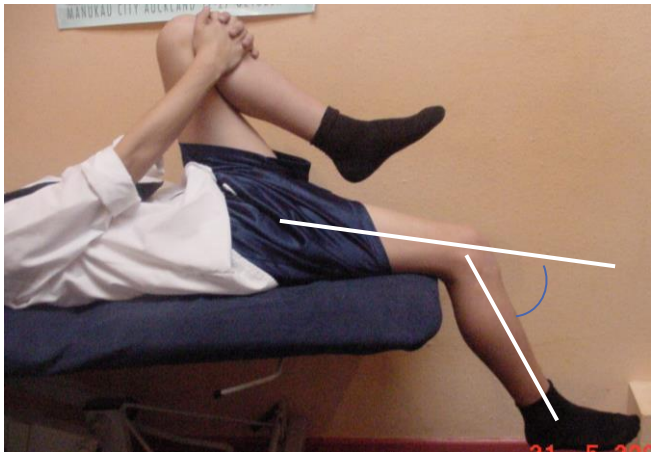


Figure 8. Thomas test position measuring the right quadriceps resting length by knee flexion angle (authors own photo)

2.3.2.2 Passive load tolerance test and intervention

Firstly, a standing-quadriceps stretch (St. George 1989) (Figure 9) was used to assess passive traction load tolerance on the apophysis (DV 2), with the test immediately stopped at onset of any pain, and not allowed to continue. Any pain felt at the tibial tubercle during the test resulted in a fail score, no pain with a full stretch range completed was given a pass score if they could repeat it twice. Technique was monitored to ensure the hip remained in neutral and the knee placed alongside the standing knee (St. George 1989).

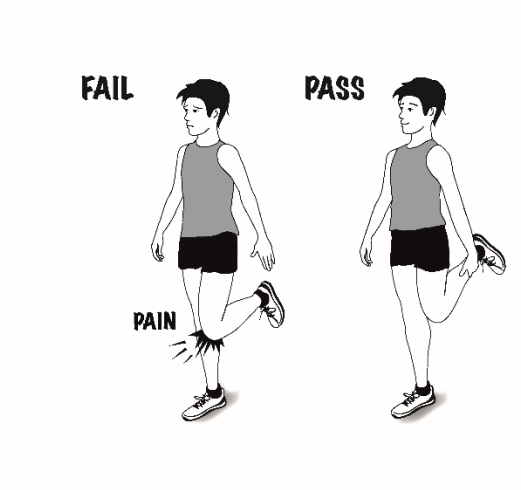


Figure 9. Quadriceps stretch test in standing (Passive load)

If the passive test was failed the primary intervention was massage (Independent variable IV2a), which is a key skill of physical therapy training. This specific intervention was myofascial release massage (MRM) performed once a day to the central muscle belly of the quadriceps for two minutes (St George 1989). MRM is a form of deep effleurage of continuous gliding strokes but at a depth similar to petrissage and frictions, with the aim of mobilizing and lengthening deep muscle and connective tissue (St. George 1989; Hopper et al 2004; Brummitt 2008; Field 2016; Gasibat and Suwheli 2017). This was performed with firm anterior-posterior pressure as tolerated by the patient, in a longitudinal proximal to distal direction with a small amount of massage oil to assist and improve comfort for the patient. Parents were instructed by the trained physical therapist researcher in the application of the massage technique, so they could carry on the treatment at home every day, thus enabling continuity between subsequent clinic visits, which were usually weekly. Parents demonstrated the technique on their child under the researcher's supervision and the patient gave feedback to the similarity

of massage technique between the researcher and the parent, to improve consistency of its application. The involvement of parents in their child's treatment is standard current practice within the physiotherapy profession and is aligned with parental duty of care (Association of Paediatric Chartered Physiotherapists, 2018)

Once the quadriceps stretch position was pain free (pass) then massage was stopped and replaced with stretching instead (IV 2b). A stretching routine using the same stretch test position (Figure 10) was then performed three times daily by the patient (in the morning, during the day, and in the evening), each stretch held for 10 seconds and repeated 5 times per set (St. George 1989). The days to pain free stretch were recorded.

2.3.2.3 Active load tolerance test and intervention

Secondly an eccentric wall squat (Meisterling et al 1998; Blandpied 1999) (Figure10) for active loading (DV3), was performed to the steady count of 5 seconds. Blandpied's study (1999) found the wall squat with feet forward activated the highest percentage of quadriceps muscle fibres and was significantly greater when compared with wall squats with feet aligned vertically with the hips, and squat machines. Meisterling et al (1998) used the same exercise for strengthening the quadriceps in OSD patients and found it was a safe exercise to perform. Any onset of pain felt at the tibial tubercle during the eccentric (downward) phase the test was immediately stopped and the patient returned to upright stance, and the test was deemed a fail. Failure of the eccentric wall squat meant patients were instructed to rest completely from physical activity other than necessary walking (IV3a). As eccentric actions (downward phase) require stronger muscular loading than concentric (upward phase) (Escamilla 2001) it was deemed sufficient to monitor only the eccentric section of the wall squat, until the patient could complete both the eccentric phase to the lowest possible position with maximum knee flexion, and return via concentric phase over 10 seconds, and repeat twice without any pain at the tibial tubercle, then the test was given a final pass score, A one minute rest was set between repeated tests. Eccentric exercise has been shown to result in possible micro-damage (Proske and Morgan 2001) therefore halting the test at the earliest onset of pain also safe-guarded the patient from excess loading that their apophysis may not be able to tolerate.

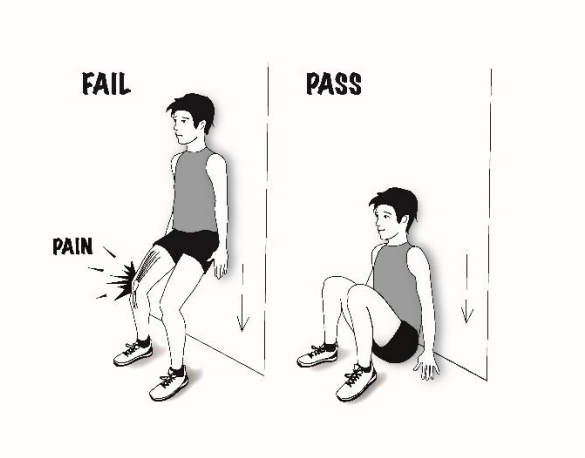


Figure 10. Wall squat test (Active load)

Once this wall squat was achieved (pass) then the return to normal physical activity could resume (IV3b). The days taken to this point were also recorded.

2.3.2.4 Discharge

The dual-approach (IV1) was completed when both tests were pain free as judged by the therapist, and patients were judged as recovered and discharged, and the treatment successful. Patient pain scores were also used for an overview of their home experience of activities of daily living such as climbing stairs and squatting, and parents reported their own observations for additional verification. In line with common physical therapy practice, on-going discharge advice on general stretching and monitoring was given to each patient to further support their full return to sport. This advice included quadriceps strengthening using the wall squat, and later more explosive exercises such as jumps, sprinting and kicking.

Treatment groups were defined as either fully-compliant (FC) or partially-compliant (PC) or unable to comply (UC). Compliance was assessed as either fully-compliant or partially-compliant dependent upon strict adherence to the protocol as judged by parental and patient feedback.

2.3.2.5 Follow-up

Follow-up interviews to gauge the longer-term success of the treatment protocol were made from 2 - 8 years (range based on time since discharge) by the researcher via phone call or subsequent clinic visit, and incidents of recurrence of their OSD (i.e. further pain at the site), level of return to sport or activity, and ability to kneel without pain were

recorded. Patients were asked to assess their level of sport as whether it was of the same standard, or higher or lower than their previous level prior to their OSD.

2.3.3 Statistical procedures and analysis

A case series study was used to evaluate the passive interventions of massage and stretching (IV2a and IV2b) on passive stretch test (DV2), and active intervention of rest (IV3) on the active wall squat test (DV3); and of the treatment protocol (IV1) on the overall recovery time of OSD patients (DV1). The distributions of the raw data sets were assessed using the Shapiro Wilks test to determine normal distribution. Descriptive statistical analyses including means and standard deviations (SD) were used for normal distributions, and medians and range for non-normal, and were used to describe the patient profiles and three sub-group comparisons, and their overall response to treatment (primary DV). Differences in passive (DV2) and active (DV3) outcome responses to treatment for the patient compliance groups, was analysed due to skewed distribution, using non-parametric testing (Mann-Whitney), and across and between three sub-groups (Kruskal-Wallis and Mann-Whitney) for significance. Pearson's Chi-Squared tests were used to assess relationships between onset sub-group and long-term recovery in the follow-up. SPSS 25 (SPSS Inc, Chicago, Il, USA) was used to analyse the data and all analyses were performed to a significance level of $p<0.05$ and 95% confidence interval.

2.4 Results

A total of 75 OSD patients were seen over a 13-year period by the main researcher and her Physical Therapy colleagues.

2.4.1 Treatment response

Patients were classified as fully-compliant (FC), partially-compliant (PC) or unable to comply (UC). 51 patients (68%) followed the protocol exactly as advised (FC) but 14 patients (19%) did not adhere to either the daily massage or the complete rest element of the protocol and were therefore only partially-compliant (PC). FC patients achieved a rapid recovery to pain free status in an overall median of 2.0 weeks (range <1-6) with the PC patients 7.0 weeks (range 4-9). Patients did respond to the treatment with symptom relief and recovery in a quicker time than normally expected therefore the null hypothesis is rejected, and the alternative is accepted. Differences between these cohorts were significant (Mann Whitney *U* test, $p = 0.014$) with a large effect size (rank

biserial correlation = 0.7). The FC group were further analysed by onset time at presentation. There were 5 patients who were excluded from the final treatment response analysis due to other factors having a possible impact on their recovery including other injuries affecting the area (patello-femoral joint syndrome (3), osteochondritis (1), meniscal tear (1)). Unfortunately, 5 patients also failed to attend their last appointment, so their final outcome was unknown. These 10 patients were deemed unable to comply (UC). The overall treatment response and time to discharge, in 65 patients, can be seen in Figure 11. Further group and sub-group analyses are described later in this section.

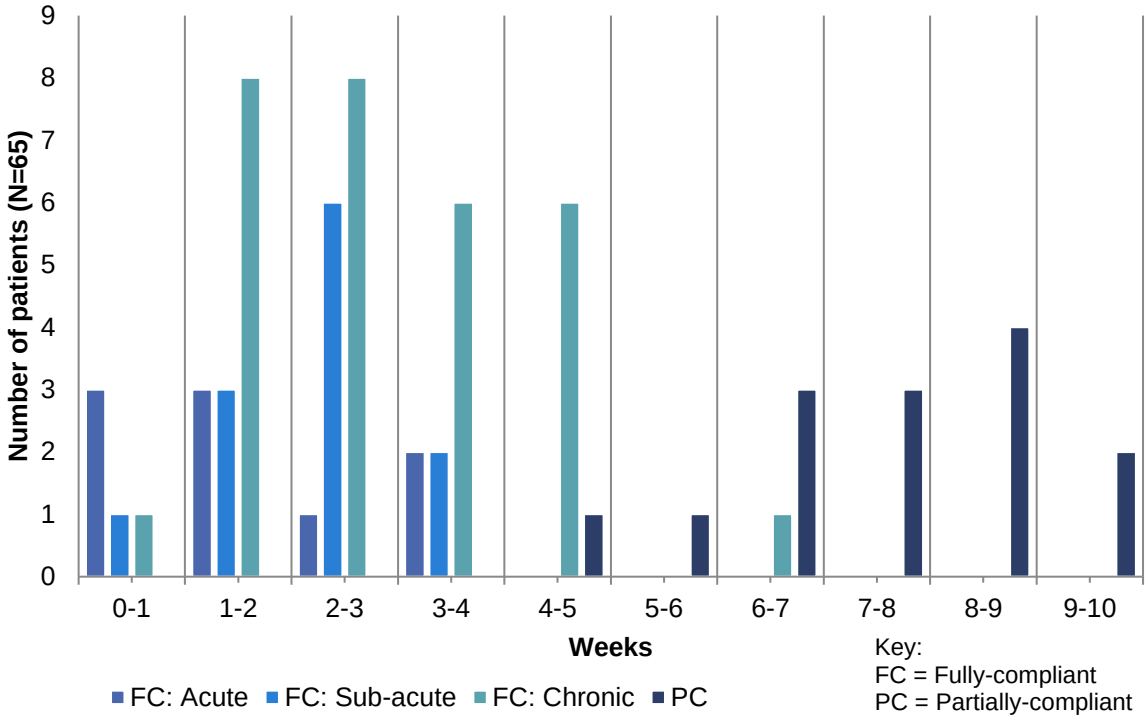


Figure 11. Treatment response (time to pain free discharge).

2.4.2 Presentation

A total of 77% of patients had already followed the standard symptomatic advice from other health professionals, involving rest, ice, and analgesic and/or anti-inflammatory medication (Kujala et al 1985; Krause and Williams 1990; Brukner and Khan 2012; Vaishya et al 2016), for a minimum of 6 weeks up to 84 weeks and had not improved, the remaining 23% had not yet started or followed any formal treatment advice All patients had entered a rapid growth spurt as confirmed by parental observation of rapidly changing shoe sizes and/or vertical growth. Boys outnumbered girls by 3:1 and their

ages and anthropometrics are summarized in Table 4. BMI was determined by dividing mass (kg^2) by height (m) to give a percentage (%). This data was normal in distribution.

Table 4. OSD patients' demographics (means and standard deviation)

Gender	Age at Onset (years)	Stature(m)	Mass (kg)	BMI (%)
Girls (n=18)	10.59 ± 1.30	1.51 ± 0.13	46.0 ± 11.4	19.8 ± 3.1
Boys (n=57)	12.20 ± 1.40	1.58 ± 0.10	51.2 ± 11.9	20.4 ± 3.7

The median time from onset to clinic presentation was 3 months (range 1 week to 2 years), but this median was calculated with four long tailed outliers of between 2.5 and 7 years removed as these did not affect the results. Onset time data was divided into 3 categories of acute (< 1 month), sub-acute (1 to 3 months) and chronic (more than 3 months), based on the stages of healing (Norris 1998). There were 12 acute, 17 sub-acute and 46 chronic patients in the 3 groups (Figure 12), giving a non-normal distribution skewed to the right, and the gender ratio of 3 boys :1 girl was the same throughout the categories.

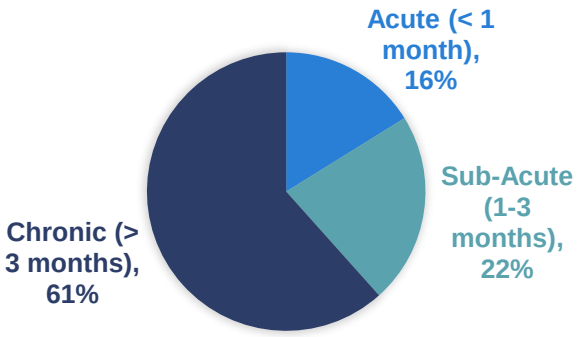


Figure 12. Osgood-Schlatter Disease patients on presentation

Bilateral presentation occurred in 52% of patients, with a small majority of patients (54%) most affected in their non-dominant leg. Hours actively engaged in organized sport in a normal week pre-injury was a mean of 14.2 hours ± 5.1 per week, and 44% of the patients played football as their primary sport, with 14% swimmers and 10% gymnasts

2.4.3 Monitoring passive tension

Quadriceps flexibility was generally quite restricted at initial measure with 81% of patients having only 60˚ resting length or less in the Thomas test position, with a mean of 54˚. The norm as described by Kendall et al. (2005) is between 80-90˚. There were

significant differences in flexibility ($p < 0.01$) immediately after the first massage intervention using t-test for 2 matched groups with a mean of 11˚ ± 8 improvement in range, and 36% gaining full resting length afterwards, and 90% of patients achieved a normal resting range of > 80˚ within 3 weeks. The final mean of quadriceps flexibility on discharge was 82˚ ± 6. Intra-tester relaibility for measuring the resting angle using the goniometer was previously determined as ±3°.

2.4.4 Passive load tolerance

All patients failed the quadriceps stretch (DV2) on initial testing therefore all patients started on the primary passive intervention of massage (IV2a). However, 3 patients reported no pain on stretch when it was repeated after the first massage application, and 57% of patients were pain free on the stretch test after 1 week, with 88% pain free by 2 weeks. The test was only regarded as a pass if patients could repeat it twice without pain. Patients then progressed onto the second passive intervention (IV2b) of stretching instead of massage (IV2a). The median time was 1 week (range 0-4) to achieve the pain free quadriceps stretch, in both groups, although full length was not necessarily restored by the same time.

2.4.5 Active load tolerance

All patients failed the eccentric wall squat on initial testing, but this component took longer to recover fully than the passive test. As stated previously FC patients were able to complete the wall slide fully and pain free in a median of 2.0 weeks (range <1-6), whereas the PC group took longer (7.0 weeks, range 4-10). (Table 5). Within the FC cohort there was a significant difference in final treatment response across the sub-groups (Kruskal-Wallis $P = .04$), but non-significant differences between the groups except for between the acute and chronic where the difference was significant Mann-Whitney U test, $p = 0.01$).

Table 5. Group and sub-group response to treatment.

Compliance Group	**Median Recovery time (weeks)**	**Sub-group**	**Median Recovery time (weeks)**
Full (all)	2.0 ± 1.0**	Acute	1 ± 0.8 ^^
		Sub-acute	2 ± 1.4
		Chronic	2 ± 0.7 ^^
Partial	7.0 ± 3.0 **		
Key: ** Significance *p*=0.014; and ^^ *p*= 0.01 (Mann-Whitney U)			

No patients were able to complete the wall squat before the passive stretch therefore all patients were treated with both massage and rest initially.

2.4.6 Discharge

All patients passed the passive component first before the active, therefore achieving the full wall squat signaled final recovery and pain free functional loading and thus they were discharged. The initial self-reported numerical VAS pain scale across the cohort was a median of 7 ± 1.8 (out of 10) but receded by the second clinic visit usually one week later, down to 1 ± 2.8, with a final outcome of 0 for all patients. Fifty percent of patients reported no pain during daily home activities by the second clinic visit. This numerical pain score was used only for an overview and feedback of the patients' daily experience rather than specific experimental variable but could have been used as an additional outcome measure.

2.4.7 Follow-up

Follow-up interviews were achieved in 58 patients (77%) with a mean of 4 years (range 2-8) since discharge, with 17 patients unable to be contacted. Three patients (5%) had a recurrence of their OSD and were all part of the partial compliance group. At the time of interview, return to sport was achieved to a higher level overall in 41% of patients or the same level in 29%, however further analysis showed 80% of acute returned to higher levels whereas 43% sub-acute and 29% chronic (see Figure 13). Patients themselves determined whether they had progressed to a higher or same level of sports competition, or whether they had regressed in standard compared to their peers, and this self-assessment could give a measure of their experience of long-term success of their

recovery. Four patients gave up sport entirely, one sub-acute and three chronic, two of the latter had suffered the condition for the longest times of 36 and 84 months. Statistical analysis showed a strong relationship between onset time and level of return to sport (Pearson Chi-Square χ^2 (1, N = 58) = 4.85, p = .028). Patients in the non-chronic groups (acute and sub-acute) had an odds ratio of 3.4 times higher level of future sports activity than the chronic patients Longer term follow-ups to see if these results were further sustained into adulthood had not been done at the time of writing this thesis.

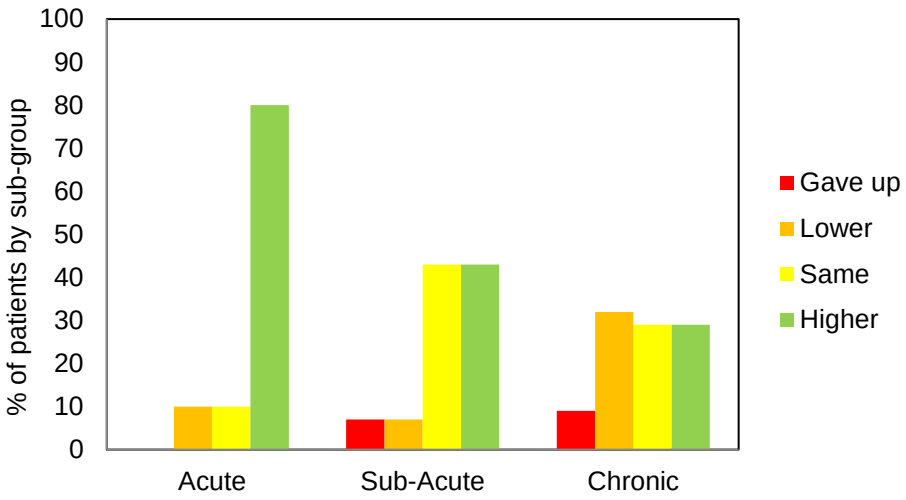


Figure 13. Sport level at follow-up

Ongoing painful kneeling was reported by 58%, most of whom (82%) were in the chronic group. Again, there was a difference between the groups with 90% of the acute and 64% of the sub-acute groups remaining pain free when kneeling, compared to only 18% of the chronic. These differences were significant between the acute and sub-acute compared with the chronic (Mann-Whitney U p <0.01 in both cases) but no difference between the acute and sub-acute (Mann-Whitney U p =0.16). There was a strong relationship between onset time and long-term pain if you were a chronic patient compared with acute or sub-acute (Pearson Chi-Square χ^2 (1) = 19.08, p <0.01). This represented an odds-ratio of 14 times more likely future pain in the chronic group compared with earlier intervention groups. This meant that although 82% of the chronic group still complained of pain when kneeling, nearly all (91%) continued to play sport at some level, though lower than the acute and sub-acute groups.

2.4.8 Summary of key results

- Patients responded very well to the treatment with the majority pain free in less than 3 weeks. FC patients responded quicker than PC, and the more recent the onset the quicker the response.
- All patients had reduced quadriceps flexibility on presentation which rapidly improved after massage
- All patients had pain on initial passive (stretch) and active (wall squat) tests
- Follow-ups found low rates of recurrence and better long-term reports in those who received treatment at the acute and sub-acute stages.

2.5 Discussion

2.5.1 Treatment response

The most important finding in this study is the rapid improvements in the FC group in recovering from this debilitating injury in 2 weeks. FC patients were pain free in both passive and active loading tolerance tests within a median of 2 ± 1 weeks from the treatment start. Resolution of pain, function and return to physical activities in 2 weeks compares very favorably with the literature which describes many months to years as the natural history (Krause and Williams 1990). It cannot be compared directly to a standard approach as there have been no published studies nor is there consensus on that standard advice (Bloom et al 2004; Vaishya et al 2016). The results do however compare favorably with the previous intervention studies mentioned where response time was described in months to years (Ehrenborg 1962; Levine and Kashyap 1981; Topol et al 2011; Lohrer et al 2012; Nakase et al 2016; Tzalach et al 2016) and the natural history as described by Krause and Williams (1990). Even those patients who were only partially-compliant still recovered in less than 2 months with a pain free passive test achieved by week 3 ± 1 (Table 3). The patients in this study were pain free on clinical testing and discharged within weeks with a significantly faster response in the fully-complaint cohort. Recovery was only confirmed at set clinic appointment times and some patients may have been able to complete the tests days before, so the resulting time scales may be under-reported. This finding supports the alternative hypothesis H_1 that the new treatment intervention results in a quicker symptom relief and recovery than would normally be expected. The long-term success also indicates that the treatment

intervention became curative as well as effective immediate recovery with low recurrence rates and high levels of ongoing sports participation.

Analysis of the onset groups showed the earlier the treatment started, the quicker the recovery. Although significant differences were not found between the acute and sub-acute responses, there was a significant difference between the acute and chronic groups indicating that the earlier treatment started, the quicker the recovery. However, the chronic group still responded well with passive stretch pain free by the second week and active wall squat by the third week. Although there were two outliers amongst the chronic group with one patient having suffered for over seven years and another for three years, they still responded well recovering in less than three weeks. It should be noted that exact time scales from onset could be affected by the accuracy of memory recall and isolating the precise onset of an overuse injury which, by its nature, may be gradual, therefore both parents and children were asked when symptoms first started and not when they became intolerable. It is likely that onset times and length of suffering could have been under-reported. The median of 3 months from onset meant that the majority of patients were chronic (61%). All the chronic group had already undertaken the standard advice and tried a combination of rest and symptomatic relief with poor results (pain and dysfunction), hence their referral for physiotherapy. This chronic group therefore could be viewed as their own control group with standard treatment for the first three months (poor response), then alternative intervention in this study (good response in 2 weeks).

2.5.2 Monitoring progress

The ability to monitor the patients' progress is an important feature of this protocol. It allows for immediate feedback to patients and parents, which may improve their engagement and adherence. It also highlights if there may have been problems with that adherence if response was slower than expected. All patients had restricted quadriceps flexibility and failed both passive and active tests at the beginning and passed both by the end. No other studies have monitored or assessed progression in the treatment of OSD, nor used a functional test to determine final treatment response. However, the most recent intervention study incorporating saline or dextrose injections into the patella tendon insertion and associated fat pad (Nakase et al 2016) used a VISA knee questionnaire (Visentini et al 1998) to assess recovery, and two used the same

questionnaire to assess long-term efficacy (Lohrer et al 2012; Nakase et al 2016). The injection study did show significant improvements in both groups over 3 months (Nakase et al 2016), but no difference between the groups, so whist efficacy appears to be shown, its rationale is unexplained. Our results show patients had no pain in less than 3 weeks which appears to show a quicker return than any of the above studies, but more information would have been gained from using a comprehensive knee score questionnaire such as the VISA (Visentini et al 1998) and this has been noted as one the limitations of this study. Pain thresholds in young adolescents may be affected by physical and hormonal stressors during puberty (Patton and Viner 2007), and accuracy of describing pain levels will depend partly on experience but also on vocabulary – both of which may be limited in the younger patient. Perception of pain may also be influenced by possible pain catastrophising giving rise to exaggerated feelings of helplessness and severity (Sullivan et al 1995). In these patients with a limited experience of pain there could be a propensity towards catastrophising it, or limited ability to describe the severity accurately (Sullivan et al 1995; Scharfbillig et al 2009; Ortqvist et al 2012). For this reason, initial onset of pain was used during testing rather than pain tolerance, and a simple numerical scale for feedback for overall pain (Williamson and Hoggart 2005).

2.5.2.1 Passive tension and Flexibility

All patients presented with reduced flexibility in their quadriceps with a mean of 55˚. This has also been observed by other authors (de Lucena et al 2011; Nakase et al 2015; Tzalach et al 2016) but it has not been recorded during a treatment intervention, either as a monitoring or outcome measure. Using the Thomas test (Kendall et al 2005; Cejudo et al 2015) allows for visible feedback for patients, parents and clinician to objectively assess in degrees the improvements in resting length and thus passive tension within the quadriceps (Hall 2012). Positive feedback from this gives further incentive to patients and parents to comply with the protocol. The improvements in flexibility to a mean of 82˚ indicates that flexibility was affected positively with the treatment, and changes measured after the first massage of 11˚ indicates the massage was responsible. Differences of > 7˚ have been shown to be reliable indicators of changes in muscle flexibility in the Thomas test (Cejudo et al 2015). Although the exact mechanism of massage is still unknown as stated earlier in this chapter, there is a clear improvement in range, passive tension and reduction in pain after the massage in this study, and

therefore further investigation into the effects of MRM, be they physiological, biomechanical, neurological or psychological or a mixture of all, is warranted.

2.5.2.2 Passive load tolerance

The passive test was also used to determine treatment selection i.e. when massage was needed or when stretching could safely recommence and was a key element in proving positive progress in treatment response. All patients started with having daily massage at home as all had a fail test on the quadriceps stretch. The differences in passive response between the onset groups was not-significant as they all showed an improvement in pain reduction by the second clinic session, with 65% being pain free in this time. Most patients had already been resting as advised by their doctors so the only change in intervention was the daily massage. The massage appeared to affect their passive test first as this was the first outcome to be resolved in all patients. It could be that the massage therefore directly reduced the passive tension in the quadriceps muscle thereby reducing the traction force on the apophysis. This would reduce the pain on the passive test and increase the load tolerance of the apophysis, but more research would be required to explore the effects of massage directly on muscle tissue and is outside the scope of this particular thesis. Once the stretch position was pain free patients were able to start a stretching regime to continue improving their quadriceps flexibility and keep the passive tension to a minimal level, as demonstrated by a full stretch position (St. George 1989; de Lucena et al 2011; Nakase et al 2016). Maintenance of good flexibility (St. George 1989) could be a key component in the control and ongoing maintenance of passive tension (Gossman et al 1982) and may have impacted on the low incidence of recurrence found in this group. No other studies have either assessed or monitored passive muscular tension as part of the treatment for OSD, yet some descriptive studies have observed its presentation as reduced flexibility (Gholve et al 2007; de Lucena et al 2011; Kabiri et a; 2014) or hypothesised its possible role as a risk factor (Nakase et al 2015; Watanabe et al 2018). This is the first study to directly address the observed painful stretch position and treat it with massage initially, then later with stretching. Progression was able to be monitored by the patients' pain response and feedback regarding the ease and range of the stretch.

2.5.2.3 Active load tolerance

Active loading of the apophysis as demonstrated by the wall squat, was achieved by patients a week later than the passive stretch test. The ability to perform a progressive eccentric loading exercise (Meisterling et al 1998; Escamilla 2001) gave valuable feedback to the patients themselves as they were able to see and feel the progression of their own recovery as they descended further before pain onset. This also gave confidence to patients that the treatment was working, and their condition was improving, and may have improved compliance and engagement. However, in this study only a pass/ fail judgement was made based on any pain during the wall squat plus full descent. This gave a clear indicator for the patients' ability for their apophysis to tolerate an active eccentric load on the apophysis and thus determine the decision to start the patients return to physical activity and sport, and discharge from treatment. Limitation of exercise to reduce active tension was also a key component of this protocol. There is little point of reducing one loading force whilst allowing another to continue, especially as physical activity has the potential to create greater forces on the apophysis than passive stretching (Hayes and Petersen 2003). This may have been reflected by every patient's passive tolerance being resolved before their active tolerance. Minimising rather than eliminating physical activity levels by immobilisation such as by plaster casting, reduces the active tension forces promoting healing yet still allows for some movement to avoid contractures or atrophy of muscles (Ehrenborg 1962; Gossman et al 1982) . Immobilising the limb may in fact cause further passive tension in the muscle due to the child's growth whilst in plaster, which may explain the high recurrence reported with this method (Ehrenborg 1962; Jakob et al 1981). Minimising activity is a practical imposition for active teenage patients to contemplate (Reeves et al 2006) especially when patients present bilaterally and can be rapidly adapted as the patient improves.

2.5.3 Massage and Stretching

The effects of massage as an intervention have not been described before and whilst this study does not examine the specific effects of massage on the muscle tissue, we can report on its effect on the symptoms of OSD. Previous research on the use of MRM indicates it can have an effect on the lengthening of muscle-tendon units (St. George 1989; Fredericsen and Wier 2006) and this may be the mechanism for reducing the

passive tension element observed in OSD patients. In this study the MRM intervention results in a rapid improvement in passive tension, quadriceps resting length, stretch tolerance and pain reduction in our patients. The possible reduction in this background tension appears to be an important first step in lessening the traction force on the apophysis, thereby allowing it to repair better. Further understanding of the underlying mechanisms of MRM, in particular with respect to its effect on myofascial length and pain reduction, should be investigated. The effect of MRM may be biomechanical by helping mobilise stiff muscle tissue as described by Weerapong et al (2005) as the resting length often improved immediately post-massage, which concurs with the findings of Field (2016) and Hopper et al (2005), although unlike the latter study, the effects lasted more than 24 hours. The mechanism may also be physiological in part as increases in muscle temperature found post-massage would result in improving tissue elasticity (Goats 1994; Weerapong et al 2005; Gasibat and Suwheli 2017). Pain perception can also be affected (Gasibat and Suwheli 2017) although this would not have directly affected the resting length of the muscle but could have reduced the pain felt at the apophysis site.

Replacing the massage with stretching when this was no longer painful, enabled the patient to take control and further improve their flexibility, thus reducing the passive tension on the apophysis. Although the mechanisms behind stretching still are being studied, its role in improving flexibility and ROM has been demonstrated (Konrad et al 2017) but methodology, types and timings of stretches used need greater consensus amongst researchers.

Follow-ups from 2- 8 years have determined sustained long-term results with 70% returning to sport at the same or higher levels However, there was a clear difference between onset groups with the acute group achieving a self-reported 80% higher level return to sport, sub-acute 43% but chronic only 29%. Most patients (58%) continued to have pain when kneeling which is similar to previous findings (Krause and Williams 1990; Ross and Villard 2003), however in this study further analysis showed large differences between the onset groups with only 10% of acute, 36% sub-acute but 82% of chronic patients having on-going persistent pain when kneeling at follow-up. This suggests that the earlier the intervention the fewer long-term problems are experienced. Chronic conditions may leave permanent bony changes and prominent tibial tubercles which could explain the on-going problems with kneeling (Ross and Villard 2003). There were three reported recurrences (5%) in the follow-up group, all who admitted they had

stopped their stretching regimes. The low recurrence rate compares favorably with one study's rate of 30% (Kujala et al 1985). The significant long-term observed responses showed significantly lower pain incidence and better sports return in the acute and sub-acute patients. This indicates that earlier treatment also has longer term benefits and reports the sustained positive effects of early and effective treatment. It is acknowledged however that there may be other factors involved in a patient's decision to continue sport and to what level, and therefore there may not be a direct relationship between the success of the treatment and returning to sport, however if a previous patient continued to suffer from pain it is less likely they will continue their sport.

2.5.4 Limitations

One limitation to this study was in its design as a case series. A control group was not included as there is currently no standard alternative to use for comparison (Bloom et al 2004; Reeves et al 2006; Vaishya et al 2016). Traditional scientific study design may also not be valid because of the multi-factorial presentation and the multiple interventions, which changed according to the individual's progression, not on pre-determined time scales or randomisation. Without a control group it is understood it cannot be claimed that this protocol is more or less effective than another, but it can present the results of recovery time which can be compared with the literature, and progression of the treatment response which no other paper has published. It should be noted that no other studies into the treatment of OS have used non-intervention control group, nor a structured rationale, rather individual symptomatic relief options as a single independent variable only.

Severity or staging of OSD was not determined nor recorded in this study and this could be an additional factor in recovery times. Hirano et al (2002) found that different stages of OSD had different resolutions with the more severe or chronic bony and tendon changes associated with poorer resolutions. Pathological staging of the cohort in future studies may provide more accurate results but in spite of this potential factor not having been assessed, the patients in this study responded well to the treatment. This was also a self-presenting group with a football bias, and they were fee-paying patients, so they may not truly reflect the general population of this age cohort. They may also have had a different motivation or expectation of results than a non-paying population may have had (Yoken and Berman 1984), however it could be argued that most parents and

patients wish to be rid of pain in the shortest possible time, irrespective of finances. The two chosen outcome measures of passive stretch and active wall slide may not fully test functional loading on the apophysis, however they are commonly used tests which are appropriate in a clinical setting with a lack of facilities and equipment. Other tests such as the explosive single-leg hop test (Kockum and Heijne 2014) or eccentric single-leg lunge (Cook and Khan 2000) may provide a stronger measure for the full recovery of the apophysis and therefore a more accurate determinant in the future for full return to sport. Alternately running a specific study to explore the different levels of the wall squat and its loading on the tendon and quadriceps would also help to further understand the study's findings and expand on the research by Blandpied (1999).

Using a specific knee function questionnaire during the study such as the KOOS questionnaire (Roos et al 1998) may have yielded more accurate feedback from patients in assessing the impact on their daily lives and changes during and after the treatment, and this would be a recommendation for future studies. Appropriateness for assessing recovery of the apophysis and in children and adolescents would need to be determined and therefore a child specific questionnaire could be a better option (Ortqvist et al 2012)

For monitoring of quadriceps' flexibility, the use of the Thomas test and goniometer is common in clinical practice (Kendall et al 1995; Sarhmann 2002; De Lucena et al 2011), however greater accuracy may be possible using the Ely position in prone, which standardizes the hip position better, and a calibrated inclinometer for improved accuracy (Tzalach et al 2016).

Patient and parent compliance are certainly a factor in the speed of recovery. There is difficulty in persuading active adolescents to rest but with the help of parents and explanation of how this was vital to the success of the treatment, most patients complied. It was also helpful with the results to show that partial compliance resulted in 2 or 3 times the length of treatment before success.

2.5.5 Summary

These results show that the researcher's treatment protocol is successful in treating OSD patients, in both the short and long-term. Specifically, massage appears to target the passive component of muscle tension quickly and effectively across all onset groups, giving discernible increase in muscle length, pain reduction and allowing for stretching to commence. The overall result is a short recovery time in all patients from a potentially chronic and debilitating injury, but with the quickest response in the earliest treatment group. The high percentage (82%) of chronic patients having problems with direct kneeling on their tibial tubercles compared to the acute sub-group (10%), demonstrates that early treatment also has better long-term results. The protocol may also provide a useful model for the treatment of other apophysitis syndromes and their treatment strategies. The use of clinical tests allowed for clear monitoring of progress and determination of progression and could be easily applied by the therapist. The changes in flexibility and improvements to passive tension as indicated by resting length of the quadriceps have also been presented which allow for direct monitoring of the effectiveness of the treatment. This study also highlights the importance of clinicians using their skill set and applied clinical reasoning to solve problems. It can be applied simply and consistently with the limited resources available to primary care health practitioners such as physical therapists or athletic trainers. With this protocol, parents and patients were also able to take ownership, empowering them to help control their own recovery and monitoring the progress of the injury.

2.6 CONCLUSIONS

There are very few studies into the treatment of OSD, and no consistent approaches exist to justify current treatments and their efficacy, nor is there consensus as to its format. This study presents the first massage-based treatment protocol using observed signs in the presentation of OSD, which enables a logical method that was successful, effective and quick within an everyday clinic setting. The protocol also provides for the first time, simple clinically-based tests for monitoring the progress of the patient and enables clarity in the selection of treatment intervention. This case series shows that OSD patients recovered from their signs and symptoms in 2 weeks, with short and long-term success, and very low recurrence rates indicating the treatment protocol is

both restorative and possibly curative. Both the treatment and monitoring were non-invasive and cost-effective requiring the minimum of resources.

2.6.1 Link to next Chapter

It appears that the passive - active model as a basis for treatment works successfully for OSD patients. Other AS are reported to have similar pathologies and possible aetiology (Dalton 1992; Micheli and Fehlandt 1992; Outerbridge and Micheli 1995), although again there are a lack of investigative studies and evidence behind treatment choices, with poor results and long recovery times. The question then arises as to whether the passive-active tension model and treatment protocol would have a similar good response in patients with other forms of adolescent AS such as Sever's disease – the second most common AS.

CHAPTER 3. Clinical Approach: Sever's Disease

Study 2. Sever's Disease- a novel approach to treatment. Case series

3.1 Abstract

Sever's Disease is one of the most common overuse injuries affecting children during their secondary growth spurts and is described as a self-limiting condition resolving naturally with skeletal maturity. It is suggested to be caused by progressive microtrauma to the bone-cartilage interface in the calcaneal apophysis partly due to large traction forces in the Achilles tendon. The current standard treatment consists mainly of rest and waiting for skeletal maturity. The aim of this case series was to investigate the effects of a novel treatment protocol on recovery rates in Sever's patients.

Method: Consecutive patients referred to a private physiotherapy clinic with Sever's disease were invited to have the new passive-active treatment protocol. A passive stretch to the Achilles tenson was used to determine tolerance to passive tension loading with onset of any pain as the marker. If any pain was felt, massage was performed daily for two minutes on the affected calf muscle until pain free passive dorsiflexion was achieved, and thereafter daily stretching was performed by the patient instead of the massage Functional active loading was tested using a standing wall squat and patients were required to rest from physical activity until this test achieved full decent and return and was pain free. Descriptive statistics were used to describe the responses.

Results: 22 patients volunteered to have the new treatment (4 female mean age 10.5 years (±1.3); 18 male mean age 10.9 years (±1.6). Onset of symptoms ranged from acute to chronic (2 to 77 weeks), with a mean of 27 (± 20) weeks. Patients achieved a pain free passive stretch to the tendon in a median of 1 week (range 5 days -3 weeks) with a full pain free active wall squat in 2 weeks (5 days-7 weeks) and were discharged and returned to their sporting activities. Follow-up interviews from 1-5 years reported no further problems except for one recurrence (5%).

Conclusion: These results were similar to the previous study and indicate that the passive -active model of treatment is an effective treatment for Sever's disease. The patients in this study returned to their sport in a significantly shorter time than is usually anticipated with the traditional treatment approach.

3.2 Introduction

3.2.1 Sever's disease

In 1912 Dr James Sever described similar apophysitis pathology at the calcaneus (heel) in a number of pre-teen children; however, he may not have been the first. Haglund is cited to have done so earlier in 1907 (James et al 2010) but it is Sever's name that has become synonymous with the condition – possibly because his article was in English, and Haglund's in German. Sever's paper was also the first to show associated X-ray changes which allowed clinicians to visualise the detachment of osseous fragments from the apophyseal junction (see figure 14 below). This detachment on the secondary growth zone, the epiphysis, was therefore suggested as the cause for the symptoms.

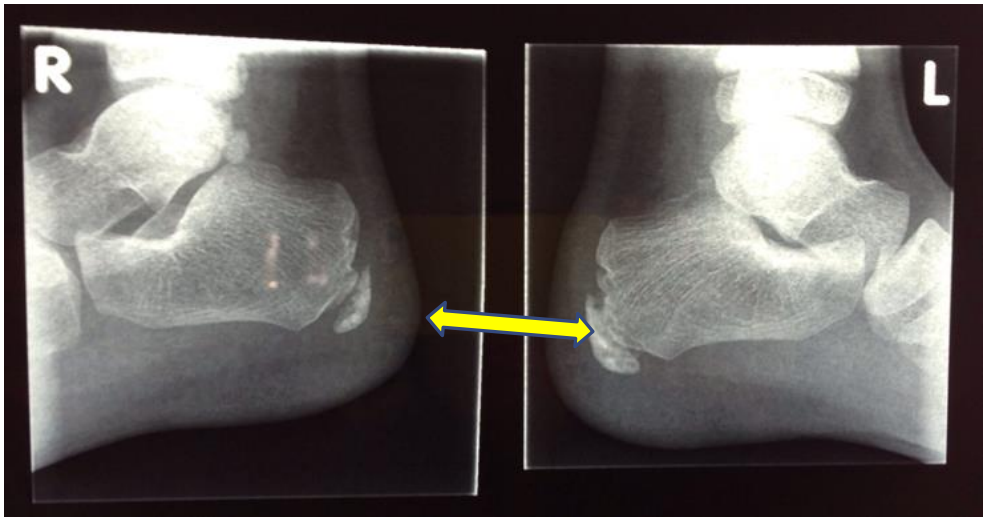


Figure 14. X-ray of bilateral Sever's disease showing detached bony fragments (arrows) in a 12-year-old boy (Author's photo)

Sever's disease is the most common overuse injury affecting the adolescent heel (Elengard et al 2010; Micheli and Ireland 1987; Cassas & Cassettari-Wahys 2006), but the incidence of Sever's disease amongst the population is as difficult to ascertain as that for OSD, for similar reasons of inconsistency of reporting parameters. Estimates of between 2 -15 % of injuries seems the most consistent (Orava and Puranen 1978; Kvist and Heinonen 1991; Kannus 1997; Price et al 2004; Jayanthi et al 2015), with much of the accurate data arising from specialist sports or orthopaedic clinics. Again, this would bias the selection cohort to either the more severe cases (reporting to a hospital) or specialised athletes (sports clinic), which means the true incidence rates

and thus risk awareness not reflecting the general population, however Wiegerinck et al (2014) recently studied the incidence of Sever's in the wider population and found a rate of 0.4%. and this may reflect the similar differences in rates between the more athletically active and sedentary adolescents found in OSD.

De Inocencio (1998) reported a 5% incidence of MSK patients with Sever's in their general practice data. Perhamre et al (2010a) state Sever's to be the dominant cause of heel pain of the 8-15-year age group, however there have only been a couple of prospective studies (Perhamre et al 2011; Weigerinck et al (2017), with all others being retrospective analyses of clinical records which could be affected by reliability and variations of clinical recording methods. Most authors record a lower incident rate for Sever's than for OSD but Larson (1973) reported otherwise, and this could be due to less recognition of the syndrome. Patients with Sever's disease are usually amongst the youngest of the AS patients with the range from 9-12 years (de Inocencio 1998; Kaeding and Whitehead 1998; |Hendrix 2005; Scharfbillig et al 2008; James et al 2013; Wiegerinck 2014). Sever's appears to occur close to the onset of the secondary growth spurt (Weigerinck et al 2014) which is the start of rapid elongation of the bones of the leg (Chamley et al 2005; Kanbur et al 2005). The calcaneus is also one of the most distal apophyseal sites in the skeleton and therefore may be under pressure first as foot growth and lengthening occurs distally before proximally (Dimeglio 2001; Balzer et al 2019), and this may be a reason why Sever's patients are at the youngest end of range for AS (Dalton 1990; Micheli and Fehlandt 1992; Scharfbillig et al 2008). The physiology of growing bones is covered in more detail in the next chapter on adolescent growth.

Diagnosis of Sever's is usually a clinical one based on patient presentation and history, but some radiological findings can show a specific detached epiphyseal fragment at the calcaneus. More recent radiological studies however have shown this detachment can be a variation of the norm, and can occur in completely non-symptomatic subjects (Elengard et al 2010; Kose 2010; Scharfbillig et al 2011; Chang et al 2013), therefore caution should be used when diagnosing the presence or staging of Sever's disease using X-ray alone. Other rarer causes of heel pain in this age group are osteomyelitis, stress fractures, neoplasms or infection, and therefore if doubt exists as to the diagnosis, then X-rays can be used to exclude these other pathologies (Chang et al 2013). Fragmentation seen on X-ray may be suggestive but not diagnostic (Liberson

et al 1995; Kose 2010). Diagnosis therefore is usually made by clinical examination with a thorough history taken and the 'squeeze' test (Szames et al 1990; Stanitski 1993; Madden and Mellion 1996; Cassas and Cassettari-Wayhs 2006; Soprano and Fuchs 2007; Weiner & Morscher 2007; Chang et al 2013) performed to localise the lesion to the insertion site (apophysis) rather than the tendon itself. Lau et al (2008) state that only 76% of their Sever's patients reported heel pain whilst 83% reported tenderness. Therefore, palpation could be a more reliable indicator of the condition and can be used to differentiate between other soft tissue conditions such as plantar fasciitis (under the heel), achilles tendonitis (tender on the tendon body itself); and achilles bursitis (tender over the achilles bursa). The age, activity level of the child and whether they are currently experiencing their adolescent growth spurt is also highly indicative (Micheli and Ireland 1987; Stanitiski 1993; Kannus 1997; Hendrix 2005; Smith and Varacallo 2017) and will help to exclude these latter injuries (Madden and Mellion 1996).

Patients present with typical pain, exquisite tenderness and swelling at the posterior aspect of the calcaneus in the rear foot and physical activities such as running and jumping are particularly affected (Madden and Mellion 1995; Smith and Varacallo 2017), but some children have pain on walking or even just weight bearing. This can be even more debilitating than OSD as just standing still can be painful. The unique location of the calcaneal epiphysis means that the lower border is at the most inferior aspect of the heel and therefore exposed to direct load on weight-bearing (see Figure 10). Therefore, these patients have two sources for possible distraction of the segment – the traction from the achilles tendon above; and the upward force from weight bearing on the heel.

The natural history is similar to OSD with pain and dysfunction fluctuating with activity levels and time (Madden & Mellion 1995; Volpon & de Carvalho 2002). The generally accepted time scale for resolution is around 7 months (Sever 1912; Agyekum & Ma 2015; Smith and Varacallo 2017) with the range been given from as few as 3 weeks (Cassas and Cassettari-Wayhs 2006) to final skeletal maturity (Madden & Mellion 1995; Kvist & Heinonen 1991; Sammarco 1995; Smith and Varacallo 2017). Most patients struggle to cope with physical education lessons at school and sport, and sports that focus on jumping such as basketball and gymnastics are particularly affected. This is likely to be because the Achilles apophysis is the attachment for the powerful foot plantar-flexors the gastrocnemius and soleus muscles, which are

predominant in these movements. It could also be relevant that gymnasts tend to have high training loads at young ages (Brukner and Khan 2012).

Elengard et al (2010) discuss the frustration of poor evidence in the treatment of Sever's stating that "most of the recommendations are based on the clinical experience of the authors, or are just a summary of what previous studies have recommended" (page 288) and that treatment recommendations are a mix of various strategies with no theory or rationale behind them and often integrated with each other meaning multiple interventions. It is therefore difficult to assess the efficacy of each modality or combination of interventions to determine whether there was a specific response. Nor are the recommendations tailored to the individual patient – a basketball player might need a different approach than a swimmer due to the different types of loading and muscle contractions used predominantly in those sports.

Severs treatment strategies again have relied on traditional advice of rest and symptomatic relief such as using ice or anti-inflammatory drugs, or addressing muscle deficits by stretching and strengthening (Micheli and Ireland 1987; Stanitiski 1989; Madden and Mellion 1996; Elengard 2010; Chang et al 2013), but as discussed in the previous chapter both stretching and strengthening exercises would create more traction on the soft apophysis causing further distraction and pain, which would be counter-productive for promoting injury repair. Two more recent intervention studies by Perhamre et al (2010 and 2011) using two different heel orthotics - a cup versus a wedge, were published. They used a cross-over design with initial randomized allocation of insole type, both of which reduced symptoms significantly in their 38-patient cohort. Patients were then allowed to choose which orthotic they wanted with 75% of patients choosing the heel cup in preference. However as 50% of those patients were still using their orthotics one year after the initial treatment intervention, the question is raised as to whether the improvement in symptomatic relief is only whilst actively wearing the orthotic, and whether this has to be a permanent intervention, and whether the patients had become psychologically 'dependent' upon them. As 19 of these remaining 22 patients were still rating the devices' effect on pain as good or excellent, it can only be assumed that they did indeed still have these symptoms of pain that required their continued use. Heel cups appear therefore to work for symptomatic relief but not for cure.

Gijon-Nogueron et al (2013) describe a case series of 18 young Sever's patients who were provided with moulded and cushioned heel cups and reported a significant reduction in pain within 15 days, and further reduction over 4 weeks. However, no control group was used for comparison, and no medium or long-term follow-up occurred so it is not known whether the orthotics had a symptomatic or curative effect. It was not reported whether the patients returned to normal activity, nor for how long they needed to wear the heel cups. Never-the-less it does appear that the heel cups were a useful tool in reducing pain in Sever's patients, and certainly less potentially problematic than medication alternatives of NSAIDs and analgesics. Micheli and Ireland (1987) also used a combination of heel cups and stretching and strengthening and had good results after two months, but again there was no control group nor indication as to how long the orthotics continued to be used or needed.

Wiegerinck et al (2016) have recently published the only randomized controlled trial (RCT) seen comparing three approaches for Sever's. They took 101 Sever's patients and assigned them to a) wait and see control group; b) heel raise orthotic group; or c) physiotherapy-supervised eccentric loading exercise regime. The final follow-up 3 months later found clinical and statistically significant improvements in pain for all three groups but there were no significant differences between the groups at that stage. This led the authors to conclude that all three approaches were equally useful and effective. However, the heel-raise group and eccentric exercise group showed more significant improvements at the 6-week stage compared with the control group, but this was from two different points of view – the children preferred the heel raises and the parents preferred the physiotherapy exercise approach. Placing a heel raise in your shoe requires minimal effort or disruption to your life which may have impacted on the children's appreciation for the intervention, whereas tailored and structured care from a health professional would have given the parents a sense of direct interaction and reassurance that their child was being 'cared for'. Both interventions demonstrated a faster resolution of pain for the patients and surely this is just as important a finding as the final outcome. The authors have not however commented on this. Surgery is rarely indicated (McKenzie 1981) although achilles tendon lengthening has been performed for a persistent case (Stress 1972).

There does not appear to be any long-term sequelae, and fusion of the apophysis to the main body of the calcaneus occurs by about 15 years of age, after which Sever's

will no longer occur (Madden and Mellion 1996; Elengard et al 2010). However, many Sever's patients develop OS later or concurrently – Kvist and Heinonen (1991) finding that 48% of their Sever's patients developed OSD, and Kujala et al (1985) found a double pathology incidence of 68%. This high concurrence of AS show that these conditions may be related, as it appears that the majority of Sever's patients develop OSD concurrently or at a later age. The researcher notes that this does not prove cause or effect, only that there appears to be a commonality.

Sever's disease therefore, tends to affect the youngest adolescents but appears to resolve faster than OS within a few months. It is still a painful and debilitating condition and literature depth and scope is sorely lacking (Scharfbillig et al 2008 Elengard et al (2010) were also critical of the available literature on Sever's disease highlighting the paucity of prospective studies with validated outcome measures and the over-reliance on clinical experience and a few retrospective studies, in fact, a similar picture to that of OSD disease.

There are a range of opinions on the aetiology of Sever's disease with the majority of authors either suggesting tight calf muscles (Szames et al 1990; Maddon & Mellion 1996; de Bengoa 2011; Wiegerinck et al 2014) or biomechanical mal-alignment (Micheli & Ireland 1987; Maddon & Mellion 1996; Hendrix 2005; Gijon-Nogueron et al 2013; Aygekum & Ma 2015). A recent paper by McSweeney et al (2019) found that Sever's patients had reduced stride lengths and higher cadence in their gait patterns which could be the result of tighter muscles or pain response. The original paper by Sever (1912) suggested being over-weight as a possible factor and James et al (2015) noted their patients were taller and heavier than their normal cohort, but neither papers stated if they felt this was cause or effect. Some footwear factors have been examined, mainly by podiatrists but this could give rise to possible bias as this is what podiatrists would be looking for. Walter & Ng (2002) felt that cleated shoes could be responsible for Sever's disease as they examined the negative position of the heel of 36 young footballers in cleated and non-cleated shoes. They found that the negative position of cleated shoes would place more pressure and traction on the calcaneal epiphysis and thus could be responsible for the greater incidence of Sever's disease amongst young footballers. It would have been further useful if they had performed a parallel study asking what type of boots any Sever's patients used. The footwear argument does not explain why swimmers also get Sever's disease.

Nearly all authors have noted the higher incidence of AS in the athletic population, with some specifically drawing association with higher levels of training and competition (Micheli & Ireland 1987; Stanitski 1989; Outerbridge & Micheli 1995; Hodson 1999; Antosia & Lyn 2002; Jakovljevic et al 2010; de Lucena et al 2011;). The fact that the incidence of AS increases with sports participation shows there is a strong association (de Lucena et al 2011), but not a definitive cause. Jayanthi et al's 2015 large study did find a strong correlation between hours spent in organised sport and overuse injury. In particular they looked at hours in relation to the age of the athlete and if the hours/ week exceeded the age of the child then this was a significant factor ($P<0.01$) in the development of an overuse injury. Single sports participation was also a significant factor in overuse injury onset. It would have been even more interesting if they had calculated the effect of single sports participants together with high weekly volumes, and whether this further increased their injury occurrence. This could have important ramifications as talented athletes come under greater pressure to specialise in a single sport and spend longer hours training e.g. football academies, tennis institutes, gymnastic and dance groups. Brenner (2007) also highlights that talented athletes in team sports may play for multiple teams so that coaches may not be aware or in control of the frequency, scope and volume of activity. Even the parent may not fully understand or appreciate the training load their child is undertaking and have an adverse influence (Stanitski 1989). Increasingly there is no such thing as an 'off season' for young athletes, which increases the risk from overuse injury or burnout (van Mechelen et al 1996; Lord and Whinell 2004). Further exploration of training loads will be highlighted in Chapter 6 on injury prevention, especially in the context of talented elite athletes.

Therefore, Sever's disease with a similar history, pathology and presentation to that of OSD, and with similarly limited treatment options, it is justified to see whether the researcher's new passive-active tension model and treatment protocol might also improve Sever's patients' recovery outcomes. There have been no studies either, on the use of massage for Sever's disease to the researcher's knowledge.

3.2.2 Aim

The aim of this case series study is to present the new passive-active treatment model as a basis for an effective treatment of Sever's disease in adolescents.

3.2.2.1 Hypotheses

H_0: The new treatment protocol will not improve symptoms or recovery for Sever's patients when compared to the literature.

H_1: The new treatment protocol will result in quicker symptom relief and recovery for Sever's patients when compared to the literature.

3.3 Method

A case series design and similar method as the previous chapter treatment study of OSD, was used for Sever's patients. The modifications in method reflect the difference in the target muscle location and functional stretch position, with the calf muscle replacing the quadriceps.

3.3.1 Setting

Twenty-two consecutive patients referred to the same private Physiotherapy practice as for the previous OSD study, over a 5-year period, with Sever's disease volunteered to have the new treatment protocol. Patient information sheets were provided and read by both patients and their parents. Informed consent was therefore agreed and signed (Appendix I, II and III), and all patients volunteered to have the new treatment protocol. The previous full ethics approval was gained as part of the previous study, as well as the enhanced Disclosure and Barring checks, as the patients were below the age of 16 years and therefore classed as vulnerable. Parents and patients were also provided with information and given opportunity to ask questions prior to commencement of treatment, including their treatment participation as parents/ carers (Association of Paediatric Chartered Physiotherapists, 2018). They were required to give verbal assent and signed consent.

A non-intervention group was not included in the study due to previously stated ethical concerns and lack of consensus on a standard control treatment option. Patient demographics were recorded as well as hours of organized physical activity per week, and time from onset of their condition, giving rise to acute or chronic classification.

3.3.2 Outcome measures

The main outcome measure was time to pain free recovery. Secondary measures were time to pain free stretch and pain free wall squat.

3.3.2.1 Passive load tolerance test and intervention

A standard calf stretch against the wall was used for passive pain onset and with the knee touching and distance of the big toe to the wall measured in cm for monitoring improvements in range (Hendrix 2005; St George 2005) (Figure 15). Instructions were given to keep weight bearing to a minimum on the front testing foot but to keep the heel grounded, and any onset of pain felt at the Achilles insertion at the calcaneus, received a fail score. No pain resulted in a pass score. Distance from the wall of the foot was used to estimate increases in calf flexibility range and was used for monitoring and feedback only.

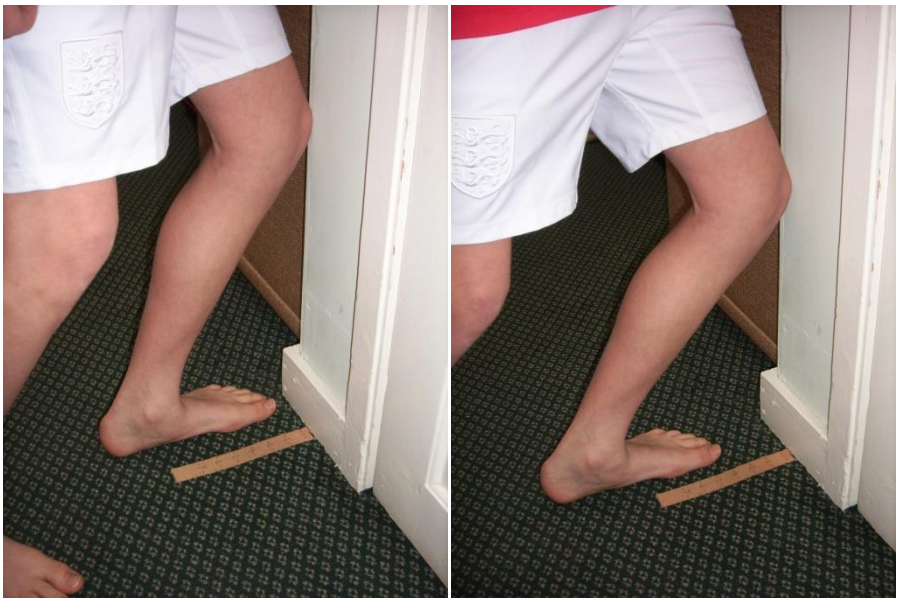


Figure 15. Calf stretch against the wall for Pain onset and monitoring

The intervention for the fail score was 2 minutes of MRM massage daily to the calf muscle, taking in both heads of the gastrocnemius muscle and the deeper soleus muscle (Figure 16). This was performed with firm anterior-posterior pressure as tolerated by the patient, in a longitudinal proximal to distal direction making sure that the popliteal fossa was avoided to protect the neuro-vascular structures there, and with a small amount of massage oil to assist and improve comfort for the patient. Parents were instructed by the trained physiotherapist researcher in the application of the

massage technique, so they could carry on the treatment at home every day, thus enabling continuity between subsequent clinic visits, which were scheduled weekly. Parents demonstrated the technique on their child under the researcher's supervision and the patient gave feedback to the similarity of massage technique between the researcher and the parent, to improve consistency of its application.

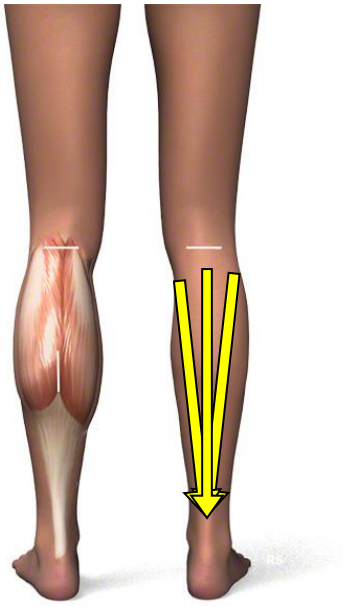


Figure 16. Site and direction of massage for Sever's Disease.

Once the calf stretch position was pain free (pass) for its entirety of stretch application from inner to outer maximum range, then massage was stopped and replaced with stretching instead. A stretching routine using the same stretch test position as well as a stretch for the more superficial gastrocnemius muscle (Figure 17), was then performed 3 times daily by the patient, each stretch held for 10 seconds and repeated 5 times (St. George 1989). Although other timings such as holding for 30 seconds or 5 minutes, have been suggested by other authors (Nakamura et al 2011; Konrad 2017) , as discussed in the previous chapter, there is a lack of consensus as to the optimal stretch timing and repetition, therefore the commonly clinically used 10 seconds x 5 repetitions was chosen for consistency within the clinical setting (St George 1989). The double stretch was used to include all muscles (soleus, gastrocnemius and plantaris) that insert into the calcaneus (Kendall et al 2005) and could be a source of passive tension.

The days to pain free stretch were recorded. Improvements in calf range as observed using the knee-to-wall test (Figure 15), were also noted for monitoring purposes.

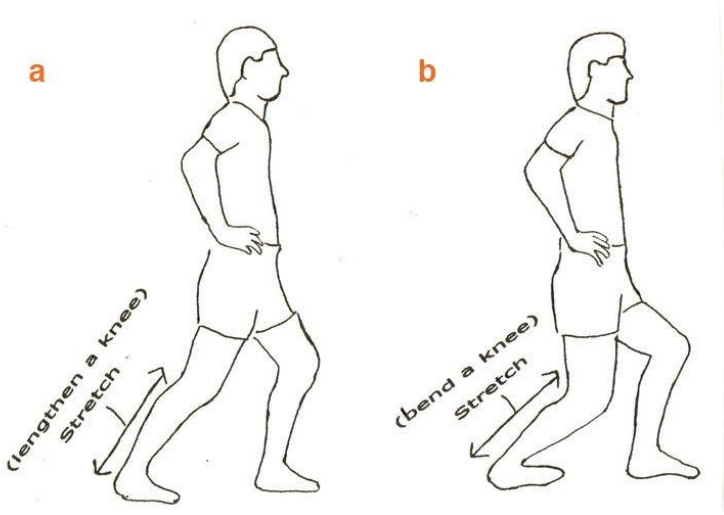


Figure 17. Gastrocnemius (a) and soleus (b) calf stretches (www.Kintec.net)

3.3.2.2 Active load tolerance test and intervention

The wall squat as used in the previous chapter was also used to assess active loading of the calf muscle and Achilles tendon (Blandpied 1999; Escamilla 2001). The wall squat was performed to the steady count of 5 seconds as for the previous study. At any onset of pain felt at the Achilles insertion during the eccentric (downward) phase, the test was immediately stopped, and the patient returned to upright stance, and the test was scored a fail. As eccentric actions (downward phase) require stronger muscular loading than concentric (upward phase) (Escamilla 2001; Hall 2011) it was deemed sufficient to only score the eccentric section of the wall squat for treatment progression. If the patient could complete both the eccentric phase to the lowest possible position, and return concentric phase over 10 seconds, and repeat twice without any pain at the calcaneus, the test was given a pass score.

Failure of the eccentric wall squat meant patients were instructed to rest completely from physical activity other than necessary walking. Once the wall squat was pain free and full decent to the floor and return assent was achieved (pass) then the return to normal physical activity could resume. The days taken to this point were also recorded. Improvements to the range of pain free descent were also noted for monitoring and patient and parent feedback.

3.3.2.4 Discharge

The dual-approach was completed when both tests were pain free as judged by the physiotherapist, and patients were judged as recovered and discharged, and the treatment successful (main outcome). In line with common physiotherapy practice, on-going discharge advice on general stretching and monitoring was given to each patient to further support their full return to sport. This advice included calf strengthening using the same wall squat, eccentric heel dips off the edge of a step for strengthening, and later more explosive exercises such as jumps, sprinting and hopping. Follow-up interviews over 1-5 years, by telephone or subsequent clinic visits, were made by the researcher to determine long-term efficacy of the treatment and feedback.

3.3.3 Statistical procedures and analysis

A case series study was used to evaluate the passive interventions of massage and stretching on a passive stretch test, and active intervention of rest on the active wall squat; and of the treatment protocol on the overall recovery time in Sever's patients. The distributions of raw data sets were assessed using the Shapiro Wilk test to determine normality of distribution. Descriptive statistics using Microsoft Excel's data Analysis ToolPak (means, standard deviations) were used to describe the normal distributions of patient anthropometrics, and non-parametric tests for the treatment response which had a skewed distribution (medians and ranges).

3.4 Results

3.4.1 Presentation

Over the period from 2012-17, 22 Sever's patients were treated, 4 girls with mean and standard deviation ages of 10.5 years (±1.3), and 18 boys mean age 10.9 years (±1.6). Onset of the condition ranged from 2 weeks to 80 weeks with a mean of 28 (±20) weeks, there were two acute patients (< 1 month), 4 sub-acute (1-3 months), and 16 chronic (> 3 months), thus most patients had a chronic condition. Classification for pathological stage were the same as for the previous study and based on the stages of healing (Norris 1998). Unilateral presentation was seen in 55% and there was equality in dominant or non-dominant involvement. Leg dominance was determined by asking which their preferred kicking leg was. Patients demographics are summarised in Table 6, and they were involved in organized sport prior to their Sever's onset for a mean of 14 hours per week (4), and 2 of the girls involved in gymnastics were training more than 20 hours per week.

Table 6. Sever's patients' anthropometrics (mean and standard deviation)

Gender	Age at Onset (years)	Stature (metres)	Mass (kgs)	BMI (%)
Girls (N = 4)	9.8 (± 0.8)	1.41 (± 0.13)	35.6 (± 8.2)	18 (± 2)
Boys (N = 18)	10.4 (± 1.6)	1.48 (± 0.09)	43.4 (± 10.1)	20 (± 3)

Measurements were only able to be performed at clinic appointments therefore the response time data may not be as accurate due to the vagaries of appointment attendances, however most patients were seen on a weekly basis.

3.4.2 Treatment response

Patients achieved a pain free stretch in a median of 1 week (range 5 days -3 weeks) with no patients having pain on stretch beyond 3 weeks. The data distribution was not normal but skewed to the left. (Figure 18). At this stage they ceased the massage intervention and replaced it with daily stretches. All patients achieved the passive test before passing the active wall squat, therefore there was a short period of time where patients were having massage and rest, and then later stretching and rest. All patients achieved a full and pain free wall squat in a median of 2 weeks (range 5 days -7 weeks) and were discharged from treatment. (Figure 18). Further analysis between

pathological stage groups was not performed due to the small numbers in each sub-group, therefore the whole patient cohort was described as a single group. The quick response to treatment means the null hypothesis is rejected and the alternative is accepted.

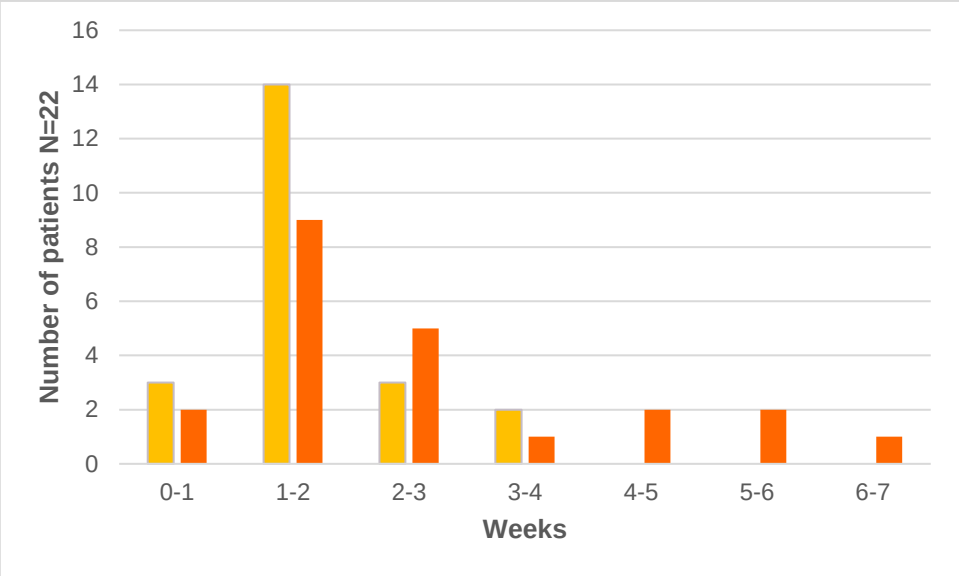


Figure 18. Sever's treatment response times (Pain free Stretch = light orange; Pain free wall squat = dark orange)

3.4.3 Follow-up

At various follow-up dates (1-5 years) all twenty-two patients were able to be contacted and only 1 patient reported a recurrence and all other patients had no further problems such as pain or dysfunction, associated with their Sever's. Changes in flexibility were unable to be analysed other than for feedback, due to incomplete data recorded but all patients had improved their flexibility by the treatment end.

3.4.4 Summary of key results:

- Patients were able to complete both passive and active tension tests on the apophysis without pain in a median of 2 weeks and discharged.
- Treatment responses appeared to have both short and long-term benefits.
- There was a low recurrence rate of 5%.

3.5 Discussion

3.5.1 Treatment response

The main outcome was the patient's quick response to final recovery time and concurrent reductions in pain and increasing function, and this was clearly shown in both passive and active tests. There was a quick response to the treatment protocol, with patients pain free on stretch and active loading in less than 2 weeks. This is in favorable contrast to published studies where 7 months is the described average recovery time (Sever 1912; Agyekum & Ma 2015; Weigerinck et al 2014), therefore the alternative hypothesis is accepted that the new approach resulted in a more effective treatment for Sever's disease than normally described in the literature. Although the recovery range has been described as from as few as 3 weeks (Cassas 2006) to final skeletal maturity (Kvist & Heinonen 1991; Madden & Mellion 1995; Sammarco 1995; Wiegerinck et al 2014). Although a wide range in recovery times has been described in the literature, the only RCT study by Wiegerinck et al (2015) showed improvements in all patients by 6 weeks but did not state if any groups improved faster than the others. It would be more useful to see which intervention led to the quickest response and perhaps more frequent measures and closer monitoring would have provided this data. The 3-week recovery as described by Cassas and Cassettari-Wayhs (2006) was based on their literature review and clinical observation, not on an experimental study of their own. Other intervention studies by Perharme et al (2010 and 2011) found reduction in symptoms after wearing heel cups but 50% of patients were still using them 1 year after, which suggests they may have continued to have on-going symptoms. Similarly, Gijon-Nogueron et al (2013) found significant pain relief using heel cups within 15 days, and Micheli and Ireland (1987) in 2 months, but it is unknown when patients were finally pain free or what the long -term response was. In all these clinical trials orthotics were used and recommend for long-term usage by the patients, but the researcher questions if the patients were indeed fully recovered, they should not require on-going intervention using orthotics.

The patients in this study did not require any orthotic intervention and the only on-going specific advice was to continue stretching their leg muscles until they finished their adolescent growth spurt. Therefore, this passive-active protocol study required minimal and simple intervention and nominal on-going care and monitoring. Massage appears

to reduce the passive tension in the MTU resulting in increased flexibility range in the calf muscle, and reduced pain. It is also suggested that by reducing this passive and active tension allows the apophysis to heal without further distraction forces, allowing for healing to be optimized.

Generic advice was given to patients at discharge on continuing with their stretching exercises and building up strength in their calves by performing the wall squat and eccentric heel dips off the edge of a step. Patients were encouraged to return to normal activities over a period of 7-10 days to encourage final healing before returning to previous levels of competition and training.Follow-up interviews found that 95% of patients had no further problems with their heels and had returned to their normal sports and physical activities. This indicates that the treatment protocol had both a restorative and a curative impact as patients had quick relief of symptoms and long-term benefits. Only one patient had a recurrence, 7 months after discharge, but they had forgotten to keep up with their stretching, and they were instructed to start the treatment protocol again until pain free in both passive and active tests. The strong response to treatment in a shorter time than is usually expected means the alternative hypothesis is accepted.

3.5.2 Limitations

With the relatively small cohort in this study, caution should be taken not necessarily to extrapolate the findings to the wider population. Again, these were a self-presenting group who were self-funded and so may not represent the wider adolescent population. There were a number of incomplete data sets regarding the flexibility measures which meant this aspect could not be fully analysed and was used for feedback purposes only. Clinic appointment times were by necessity patient-orientated and therefore response times for each test were based on these, not necessarily when the patient may have been initially pain free. Therefore, these results may have over-estimated the pain response times, and the results may have been even faster.

The outcome measure of passive stretch load on the tendon relied in part upon feedback from the patient as well as observation. It could be suggested that patients keen to be discharged and return to sport could mask the true extent their pain and reply in the positive, however care was taken to closely observe facial expressions and verify with parents that the patient was moving more normally without the previous limitations. Therefore, the validity and reliability of this as an outcome measure must

be viewed with this in mind. The subjective nature of pain is another reason that onset of any pain was used as the parameter and not amount of pain.

The wall slide also used the subjective parameter of pain but also an objective monitoring of wall slide descent, which the patient was blinded to as it was behind them. The subjective element has the same limitations as described above but it would be useful, as described in the previous chapter, to study the reliability and validation of the amount of wall slide to determine its accuracy in measuring the extent of the improvements in active loading on the various leg muscles (Blandpied 1999). Although the use of the wall slide is a simple common clinical test that allows for easy monitoring of progression of active loading of the leg muscles it may not be the most specific test for the calf muscles and Achilles tendon. It would be useful to find a more targeted validated outcome measure to quantitatively assess an eccentric load on the Achilles tendon using a heel dip, hop test, or landing from a jump but these may be too forceful to use during the initial assessment and treatment process when the patient is injured, and these may be more suitable as a test before final return to 100% sport active.

With a greater number of patients, it may have been possible to assess response to the treatment with respect to onset and pathological stage, as for the previous OSD study, and this may have given greater insight as to the treatment efficacy.

3.6 Conclusions

The findings of this study are similar to those of the OSD study in Chapter 2, with the passive stretch element in patients recovering faster than the active, and a quick overall treatment response compared to the literature and natural history. The result indicates that an approach dealing with dual aspects of passive and active tension returned patients to their normal activities with a much shorter recovery time seen than normally expected (2 weeks vs. 28 weeks). The final return to sports activities commenced in all patients with no further problems apart from one recurrence. The indication is that the use of massage, stretching and rest are likely to be an important intervention in the treatment and long-term cure of Sever’s disease, and that the passive-active treatment model works for a different AS at a different anatomical site.

3.6.1 Link to next chapter

The above studies have shown that addressing both passive and active tension in OSD and Sever's patients leads to a much speedier recovery than that described in the literature, but it is not known if these observed elements are a result of these conditions or may be factors involved in the development of them. The obvious question that then arises is if minimising passive and active tension results in the resolution of the conditions, are increased passive and active forces responsible for them occurring in the first place? Additionally, would monitoring passive and active loads on adolescents allow for identifying those at risk, and therefore pre-empt the conditions as a prevention strategy? It is not known whether the researcher's theory of the cause of passive and active tension overload is indeed correct but developing a research proposal into measuring and monitoring young athletes could provide useful information behind normal secondary growth and why these AS only occur during this specific phase.

CHAPTER 4. Epidemiological Approach: Aetiology

Study 3 - Assessment of growth and flexibility during adolescence in Academy male footballers, and its relevance to Apophysitis syndromes: A longitudinal observation study

4.1. Abstract

Apophysitis syndromes have been associated with children undergoing their secondary growth spurt in adolescence, and reduced muscle flexibility has also been observed amongst patients with an AS, but there have been no studies which explore if there are any associations. Such studies could give better understanding of possible aetiological factors that could give rise to identifying at risk players. A football academy was used as there is a higher incidence of AS amongst young athletes, and the academy setting provides an element of consistent physical activity amongst the players.

Method: The Crystal Palace Football Club Academy consisted of 9 teams of 20 male players at a given time, ranged from 9 -15 years of age, but the total number of players increased as new players were selected. This longitudinal study over 5 years monitored the changing dynamics of growth and flexibility by measuring standard anthropometrics (stature, mass, lower limb bone segments, thigh and calf girth) and muscle flexibility for the quadriceps and calf, for each player between 3 – 4 times per year. Players who developed an AS were re-measured at onset and removed from immediate training and treated according to the protocol described in the previous chapters. Peak Height Velocity (PHV) was estimated retrospectively from recorded height velocity measures reaching a peak. Comparative descriptive statistics were used to determine changes in anthropometrics, and analysis of variance to determine any differences between the AS group and their peers who did not develop an AS (Control Group = CG) and explore any relationships between the measures using univariate correlation analysis. Significance was set throughout at $p<0.05$, and statistical analysis was performed using the SPSS package (Version 25) from IBM.

Results: Academy players (N=220) were followed over 5 years. 31 players developed an AS during the period of the study with OSD accounting for 56%, Sever's disease 26%, Sinding-Larsen-Johanssen disease (SLJ) and Hip apophysitis (AIIS) with 9% each. PHV was estimated in 103 players (AS =30 and CG =73). There was an overall trend towards early maturation of players compared to the normal population. Players with AS were similar in height, BMI and age at PHV as their CG peers, however they had slightly faster growth rates at onset and at PHV, but less mass (Mann-Whitney U,

p<0.001 and *p*=0.027 respectively). There was an overall academy decrease in calf flexibility at age 11 years and for quadriceps flexibility at age 13 years, before gradually improving to previous levels by age 15. Flexibility reductions were also significantly different between the groups with quadriceps decreasing to 35˚ at onset of OSD, SLJ and AIIS; and calf reduced to 45˚ for the Sever's patients compared to the CG with 55˚ and 33˚ respectively (2-tailed T-test with equal variance, *p*<0.001). OSD, SLJ and AIIS onset was at or around the moment of PHV and Sever's occurred about 6 months prior to PHV. There were strong relationships found between growth height velocities and flexibility decreases indicating faster growth rates were associated with tighter quadriceps and calf muscles (Spearman's rho correlation -0.81 and -0.78 respectively), but even stronger relationships and higher values were found with the AS group (-0.96 and -0.97). Players treated with the new protocol were able to return to play in a mean of 20 days ± 9.

Conclusion: AS players grew at a slightly faster rate than their peers and had significantly reduced flexibility in their quadriceps and calf muscles prior to onset. There is a strong relationship between height velocity and reduced muscle flexibility in academy footballers, which is associated with PHV and occurs around the same time as the onset of the various AS seen in this study and may be a possible risk factor. Treatment of AS using the new protocol gives similar results to the previous chapters.

4.2. Introduction

Adolescence is a time of great physical, emotional and hormonal changes, many of which occur very rapidly (Tanner and Preece, 1989; Buckler 1990). It is also the prime period for the development and onset of AS with authors directly linking Apophysitis syndromes (AS) with adolescent growth (Micheli 1987; Dalton 1992; Outerbridge & Micheli 1995, Duri et al 1999; Gholve et al 2007; Brukner & Khan 2012; Chang et al 2013; Wiegerinck et al 2104). In trying to ascertain aetiological factors and understand growth-related injuries such as the AS, it would be useful to study a range of growth characteristics that might have an impact, specifically during the period of years of adolescent growth. Cross-sectional studies may give a snap shot of events and provide a quick assessment at a set point, but only longitudinal studies can provide answers to changes of characteristics over time (Buckler 1990). Due to the timing and magnitude of growth in individuals, a longitudinal study across a large population is needed to capture aspects of growth and identify critical moments (Tanner et al 1966, Buckler 1990), that may be pertinent to the aetiology of AS.

The previous studies (Chapter One and Two) provide a possible hypothesis for examining passive muscular tension (i.e. flexibility) in conjunction with anthropometric characteristics during this rapid growth period. Reported higher incidences of AS amongst athletes (Peck 1995; Price et al 2004; Jakovljevic et al 2010; de Lucena et al 2011) directs consideration to monitoring changes within a specific athletic environment, to increase the likelihood of having AS subjects. Football academies in the United Kingdom provide such an environment and a sample population which is likely to have higher incidences of AS than the general population (Kvist et al 1984; Kujala et al 1985; Hodson 1999) together with the prime target age range of players of 8-15 years of age (Dalton 1992; Outerbridge & Micheli 1995; Peck 1995). They also provide an environment where physical testing and profiling is performed on a regular basis, and players are familiar with testing procedures, together with a qualified medical team to assess and treat injuries such as AS and support those players optimally (Price et al 2004). Football academies also tend to have a mono-gender cohort which would reduce some variables.

4.2.1 Football academies

Football (also known as soccer) is the most popular sport in the UK (The FA 2018) and globally (FIFA 2018) and consists of two teams of 11 players competing to move a ball

around a field with the aim of placing the ball in the opponent's net and scoring a goal. The game is played primarily with movements of the lower limbs with explosive actions of kicking, sprinting, and jumping, high levels of aerobic and anaerobic energy output (Williams 2013). Lower limb muscles must provide the forces for these high explosive contractions as well as background stamina (Reilly and Williams 2003). At a professional level football can provide successful players with great wealth and high prestige and the competition to become a professional footballer and secure a contract with a club is intense. The competition is equally fierce between football clubs to identify and secure talented footballers and sign them as early as possible to optimise their training and development (Goncalves et al 2012). Therefore, many professional football clubs have feeder academies where young players are recruited and trained by professional coaches in the hope of developing an elite adult player ready for the professional game. In the UK all Premiership football clubs have their own academies, as well as many of the next two tiers of professional clubs (The FA, 2018).

The football club academy setting in England and Wales provides an intensive coaching environment with a long-term strategy for the development of an individual player. It introduces players to a multi-faceted approach encompassing physical, technical, tactical and psychological development across a wide age range from 9 - 21 years (EPPP 2011). It works across three age groups of 'Foundation' (ages 8 -11 years), 'Youth' (ages 12-16 years) and 'Professional Development' (Ages 17-23 years) (Read et al 2016). It aims to allow for a player-led approach enabling individuals to develop at their own pace and enhance their playing ability and support their transition into the professional game. Elite performance programmes were designed to improve selection and development of youth players. The strategy involved games programmes, education and elite performance with high-quality support from academy staff including medical, sports scientists, coaches and educators.

The games programmes led to an increase in competitive leagues, tournaments and matches and were designed to help bridge the gap between youth and senior competitions. Education was provided to deliver football-specific subjects including tactical, technical skills, physical and mental well-being.

In addition to player development was the importance of medical support and the development of a national injury surveillance project to better document and understand the types and incidence of injuries, and therefore develop prevention strategies to

reduce risk (EPPP 2011; Read et al 2018b). Data was collected quarterly from 27 Premier and Category One clubs and sent to the FA for analysis. The category was determined by an audit of facilities, staff, budgets and history of player development (Read et al 2016). The results of the data collected was sent back to the clubs to enable them to understand their injury profile and compare with benchmarks across the other clubs, and to give accurate data on the types of injuries occurring in academies across the country.

Growth and maturation screening programmes were also implemented along with standardized fitness tests, to give an individual player profile of their physical development. Regular body measures especially during the peak growth period enabled changes to be made to reduce load and injury risk and the FA set up an advisory panel of experts to help with research and guidance (Read et al 2018a). Pre-participation screening is accepted as normal practice in elite sporting environments (Bratton 1997; Carek 2002; Wen 2004) and the emphasis during youth development is on growth and maturation and training load. Continued monitoring was encouraged to identify significant changes to an individual's profile when compared to their peers (Gabbe et al 2005; Abernathy and Bleakley 2007). Pressure is on the academies to identify and develop talented players whilst providing a safe environment and duty of care to their players (Reilly & Williams 2003). Academies have a variety of professional and amateur staff including coaches, medical doctors and physiotherapists, and sports scientists, to provide that support, but the ultimate aim is to develop winning players and the drop-out and turnover rate of youth players is extremely high, with the chances of eventual success very low (Reilly & Williams 2003). In the 2016-17 premier league season less than 1% of footballers developed through their own club academy system (Daily Mail 2017) and some clubs had no academy graduates in their 1st team at all. The Football Association who run the game in the UK, introduced a quota system in 2010 to limit the number of overseas players to 17 out of a 25-man squad, to try to promote home grown players (i.e. those registered with a FA or Welsh FA club for at least 3 seasons prior to their 21st birthday). There is no restriction on Under-21-year-old players so this was designed to give academy youth more opportunity to succeed and eventually enhance the national game (The Independent 2010).

Players in academies are selected into chronological age groups for teams from Under-8's up to Under 16's. They are under huge pressure to succeed and may have elevated

status amongst their school peers, but there is a high turnover or failure rate as many academy players are ‘released’ (rejected) and replaced at the end of each season. The success rate of an academy entrant at age 8 to remain within their academy for the full 9 years until promotion to the U-18’s team (and thus leaving the academy) is about 0.5%, and overall only 0.012% of academy players in the UK will succeed to become professional footballers (Calvin 2017). However chronological age is not a good indicator to base athletic development between the ages of 10 -16 years because of the wide variation not only of physical development but also cognitive and emotional (Malina et al 2003; Le Gall et al 2007; Johnson et al 2009).

Long-term athlete development (LTAD) criteria are used to enhance and adapt training programmes for different stages of a child’s development (Maffuli et al 1994; Balyi and Way 2009) as it is increasingly recognised that children are not ‘little adults’ (Caine et al 2008; Viru et al 1999) but require careful handling and monitoring to make sure workloads on the immature skeleton are not excessive and cause break down or injury (Beunen and Malina 2005; Caine et al 2008; Balyi and Way 2009). The EPPP has embedded safe player development as a main aim, to enable increased player training without increased injury risk (Read et al 2016). The ultimate aim for any academy is the successful development and transition from a young academy player into a professional footballer – preferably within the same club. Injury prevention and management is a key component to this aim (Read et al 2016). However, the age grouping nature of the academy and competitions means that players are categorised by chronological age and not by biological age. This means that with the wide variation of maturity rates seen in adolescence players may be significantly mis-matched physically and could give rise to heightened injury risk (Balyi and Way 2005), and selection bias of physically more mature individuals (Vaeyens et al 2006). Indeed, Ostojic et al (2014) found that amongst male academy footballers at age 14, 44% were categorised as early maturers, 35% normal maturers and only 21% were late matures. This indicates a large bias towards selection of early maturers in their cohort, yet the same study found that those who succeeded in attaining professional contracts at the elite senior level were heavily weighted towards those of late maturation (60%) > normal (38%) > early (12%), so the academy selection maturation bias did not translate into adult success.

The year preceding this study the Crystal Palace Football Club Academy (CPFA) recorded up to 27% incident rate of AS amongst their academy teams. This meant that at any given point in time ¼ of a team's players were affected by pain and their performance was compromised. This was much higher than Price et al's UK study (2004) previously mentioned which found an AS incidence rate of 5% of total injuries. De Lucena et al (2011) found 9% amongst their school population but double that for athletes and Kujala et al (1986) found the prevalence of OSD amongst adolescent athletes to be 10% compared to 4.5% in those less active. Whether these differences in incidence rate is due to the different sports played in those countries or other factors is unknown, but the high rate of AS incidence amongst athletes is a significant problem in the health of young athletes, and it was becoming a problem at the CPFA. In line with the recommendations from the EPPP, understanding the risks to injury from early sports-specialisation is an important role of the academy staff, and especially during key stages of growth and maturation (Read et al 2016; Read et al 2018a). The cause(s) of AS and any relationship to growth has yet to be studied to this researcher's knowledge. Identifying those risks and acting on them should also lay the basis for prevention and thus improve player health and well-being within the academy setting and feedback into EPPP recommendations for the future (Read et al 2018c; Tears et al 2018).

4.2.2 Adolescent growth

Maturation has two main markers – timing and tempo, and measurements by anthropometry and biological age allow for comparisons across a population (Mirwald et al 2002). Balyi & Way (2009) describe six phases of human growth of which there are two periods of rapid skeletal growth in humans (Figure 19). The primary growth spurt occurs during infancy and early childhood with rapid initial growth which then decelerates (Phase 1), and stabilises during Phase 2 up to the age of around 8-10 years old in girls and 9-11 in boys (Tanner and Preece 1989; Buckler 1990; Beunen and Malina 2005; Bayli & Way 2009; Malina et al 2015). Buckler (1990) also notes that there is a short period of minimal growth velocity at the end of Phase 2 that briefly precedes the start of the accelerated phase, called the minimum height velocity (MHV). The secondary growth spurt starts with the onset of rapid growth and finishes with its peak at around 11-12 for girls and 13-14 for boys and usually lasts about 1-2 years

(Phase 3) (Dimeglio 2001; Bayli and Way 2009), followed by rapid deceleration for about 2 years (Phase 4) and slow decline and deceleration of growth (Phase 5) until cessation of growth and maturity (Phase 6).

It is the adolescent period represented by Phases 3 and 4 that is of most interest as, previously mentioned, these are the phases of greatest growth and these are the ages most commonly associated with AS. These rapid growth phases of 3 and 4 match the EPPP 'Youth' age group.

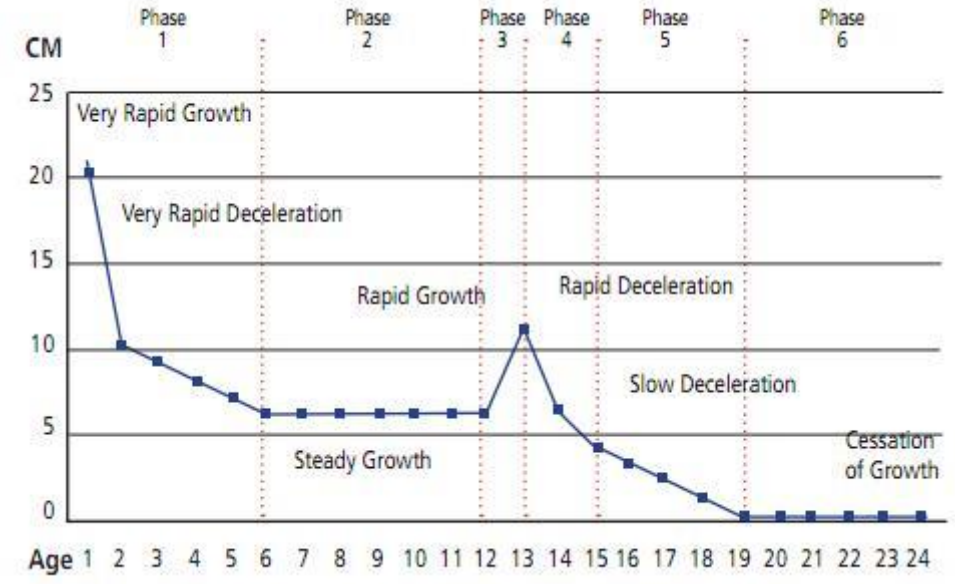


Figure 19. Six Phases of Childhood Growth (Balyi and Way 2009)

4.2.2.1 Skeletal growth in adolescence

Growth occurs in all bones as immature cartilage is replaced by the activity of osteocytes and the skeleton slowly ossifies. Skeletal height changes occur mainly in the diaphysis region of long bones where the bone grows from the centre and expands longitudinally and laterally. This region is known as the primary ossification centre (Drake et al 2005). The secondary growth spurt occurs at the start of puberty (adolescence) with a rapid spurt over 2-3 years and continues at a lesser rate for around 10 years. This adolescent growth occurs at the secondary ossification centre at the articular ends of long bones, which is known as the epiphysis (Chamley et al 2005; Drake et al 2005; Marieb & Hoehn 2013). At these epiphyses, endochondral ossification takes place where a layer of cartilage is replaced by bone at the articular ends and at the epiphyseal plate itself. The process of creating and laying down new cartilage then replacing it with bone causes the bone to lengthen in a process of both

modelling (new bone creation) and re-modelling (reshaping) (Marieb and Hoehn, 2013). The cartilage zone is weaker than the bone that replaces it, so during this process there is region of relative tissue weakness and vulnerability (Micheli 1987; Bruckner & Khan 2012). It is at these epiphyseal plates that the insertions of major tendons associated with AS are located e.g. patella tendon in OSD and Achilles tendon in Sever's disease (Dalton 1992; Duri et al 1999; Bruckner & Khan 2012).

Particularly rapid linear growth occurs in the long bones of the legs where it has been estimated that 60% of final total skeletal growth occurs proximal and distal to the knee joint (Dimeglio 2001), making this joint and its two epiphyses the greatest region of bone expansion during adolescence, and could therefore make this area more vulnerable to growth related injuries (Longo et al, 2016). Furthermore Pritchett (1992) found that 70% of femoral growth occurred at the distal epiphysis and up to 90% of this growth occurred at age 14 in boys (63% of the total femoral length). Similarly, according to Pritchett (1992) the proximal epiphysis accounted for 57% of the tibia's growth, of which 80% occurred at age 14 in boys (45% of the total tibial length). Therefore, the articular ends of the knee are under double growth influences from above and below and with very high proportions at the age of 14 years. This age is also strongly associated with Peak Height Velocity (PHV). Dimeglio (2001) describes boys growing on average 16.5 cm during the 2 years of accelerated growth (Phase 3) and 6 cm during the deceleration stage (Phase 4). Any anatomical structures that spanned this joint would also need to accommodate equally for that growth or there could be a mismatch between tissues (Lloyd et al 2014), and the quadriceps muscle and its patella tendon is such a structure (Kendall et al 2005). The stimulus of growth and bone lengthening transmits a longitudinal tensile force to the muscle-tendon unit (MTU) requiring an equal and opposite response in tissue lengthening (Hall 2012). The MTU responds to loading but it is a reactive process which requires time for tissue adaptation thus there may be a time lag between stimulus (bone growth) and tissue response (MTU lengthening) (Gossman et al 1982; Micheli 1983). Longitudinal bone growth therefore could cause increased passive tension to occur in the soft tissues spanning the bones and their joints, if they were unable to lengthen equally at the same time (Ogden and Southwick 1976; Micheli 1987; Outerbridge & Micheli 1995; Peck 1995; Kibler and Safran 2005; Smith and Bhimji 2018).

Within the secondary growth stage there are also differences in timing of different bones with not only the distal leg segments growing before the proximal (Chamley et al 2005; Kanbur et al 2005), but also the distal ends of bones preceding proximal. Thus, the feet grow first, then distal tibia before proximal, then the femur, and the legs lengthen before finally the trunk (Dimeglio 2001; Balyi and Way 2005; Balzar et al 2018). Therefore, the secondary growth zones, the epiphyses and secondary centres of short bones such as the calcaneus and patella, may be under pressure at different times as they initiate bone growth and finally fuse with the main body of bone. The patella does not fully ossify until just after puberty whereas the tibia secondary site appears 2 years prior to PHV and does not fully fuse with the shaft until around age 20 years (Chamley et al, 2005). Malina et al (2004) found that peak leg velocity occurred before overall PHV, which would concur with Pritchett's (1992) findings above, and peak trunk velocity after. Mirwald et al (2002) charted leg length to sitting height (i.e. trunk height) ratios during adolescent growth and found leg length increasing from 91% to 93 % in the two years preceding PHV reducing back to 90% two years after. Thus, there appears to be a rapid growth initially in the legs of adolescents especially at the proximal tibial and distal femoral epiphyses, up to the timing of PHV overall, followed by trunk height growth after PHV. The growth therefore is disproportionate and with different timings (Dimeglio 2001; Balyi and Way 2005; Balzar et al 2018). Overall nearly 25% of the eventual adult height is achieved during the adolescent growth spurt (Kanbur et al 2005).

It is also estimated that 26% of bone accrual occurs within a 2-year window of peak bone mineral content velocity but whose peak occurs about 7-9 months after PHV (Bailey et al, 1999; Jurimae et al 2018). At the age of PHV boys may have reached 90% of their eventual adult height but only 60% of their bone mineral content (Bailey et al, 1999). This lag between longitudinal linear bone growth and later increases to bone mineral density results in a period of relative weakness and coincides with a higher incidence in some fractures (Alexander, 1976; Burr 1997; Khosla et al, 2003), however this increased frequency could also be related to greater obesity amongst children and greater sports participation, as the authors note. However, studies have found greater bone mineral accrual amongst active adolescents than their sedentary peers and a positive relationship between high-impact weightbearing training and increased bone mineral accrual (Bailey et al 1999; Baxter-Jones et al 2008). Increased sports

participation helps to strengthen bones reducing the future likelihood of osteoporosis (Baxter-Jones et al, 2008; Jurimae et al, 2018) but may give rise to more opportunity for falls and accidents. Targeted strength and conditioning training programmes designed to optimize bone loading have also been shown to reduce injury risks in children (Faigenbaum et al, 2011) and greater muscle strength is a known factor for improving bone strength (Burr 1997), so correct preparation for physical activity appears the best prevention strategy for minimising injury risk during the vulnerable growth period of adolescence (Faigenbaum et al, 2011)

There is a peak to this secondary stature growth which is called PHV (Tanner, 1962; Buckler, 1990; Balyi and Way, 2005; Malina et al, 2015).

4.2.2.2 Anthropometrics - measuring growth

Measurement of children during rapid growth changes is problematic due to the wide variety of individuals patterns of growth and trying to capture those changes in a relatively short time span (Tanner 1962; Buckler 1990; Balyi et al 2013; Malina et al 2015). Tanner et al (1966) recommend measuring every 3 months during adolescence to obtain the most accurate growth velocity changes, and Buckler's longitudinal study of adolescent growth (1990) aimed to measure their children once per term (3), but recorded observations of between 3 to 6 months, whereas Dimeglio (2001) recommends twice per annum is enough to identify the first signs of pubertal growth of an increase of more than 0.5 cm per month or 6 cm per year. Some authors describe measuring only once a year (Philippaerts et al 2006; Malina et al 2015) but this could miss the point of onset and peak of Phase 3 growth (Figure 19) which may only be one year apart. However, Balyi and Way (2009) recommended measuring every quarter year (3 monthly) to increase the likelihood of recording the moment of change in growth rate and to allow for substantive growth over and above that due to error of measurement. More frequent measures could impact on the athletes becoming bored or anxious if their growth is perceived to be delayed (Bayli and Way 2009). Buckler (1990) and Balyi and Way (2005) tried to measure consistently on the same day of the week and time of day to reduce error and influence of fatigue and gravity effects by the end of the day (Tanner and Preece 1989). Measurements were also taken before training sessions to minimise any effects from physical exercise (Balyi & Way 2005), however both sets of authors acknowledged the practical difficulties of obtaining data in a longitudinal study with some subjects leaving the study or moving away, and

circumstances unavoidably varying, although maintenance of techniques were consistent (Buckler 1990; Bayli and Way 2005).

The fundamental anthropometrics for childhood growth includes standing stature, sitting stature (to apportion leg to trunk ratios), and mass (Tanner 1962; Buckler 1990; Eston and Reilly 2013; Dimeglio 2001; Bayli and Way 2005). Additional measures sometimes added include bone segmental length, limb girth, skin fold thicknesses, shoulder and hip width, humeral and femoral condyle diameters, Tanner pubertal staging for breast and genital development, and arm span depending on the aim and focus of the researchers (Tanner 1962; Buckler 1990; Bayli and Way 2005; Eston and Reilly 2013; Malina et al 2004).

4.2.2.2.1 Peak height velocity (PHV)

The acceleration of growth occurs during a period of around 2 years prior to its fastest or peak rate (PHV) and then decelerates over the corresponding 3 years, but the timing, magnitude, sequence and rate of this growth can have wide variation (Tanner 1962; Buckler 1990; Malina et al 2004; Balyi et al 2013). As such it is very difficult to apportion accurate ‘normal’ or even ‘average’ values, so Buckler termed the use of ‘reference values’ to enable comparison of individual profiles to gauge whether children are growing healthily within a range of adolescent measures. PHV occurs around 2 years later in boys than for girls but is more intense with faster growth rates and higher amounts (Balyi et al 2013). The age of maximum velocity of skeletal growth is called the age at PHV (Balyi and Way 2009). However, the range of ages at which PHV occurs can vary from 9.0 -15.0 years for girls and 11.5 – 17.3 years for boys (Philippaerts et al 2006; Malina et al 2015).

PHV is one of only two standard points in chronological age during puberty that can be determined with any degree of precision, the other being menarche (the age at which girls start their menstruation) (Buckler 1990). The timing of PHV is commonly used as a reference point for comparison of various adolescent growth parameters such as height, weight, limb and trunk heights, and visual pubertal characteristics (Buckler 1990; Balyi et al 2013). Therefore, PHV is the only consistent common point of reference for boys during adolescent growth and is a key measure for determining degree and timing of maturation (Buckler 1990; Mirwald et al 2002). Measuring PHV is the main tool used to track growth and is essential for coaches to be able to monitor the timing and tempo of young athletes in order to help coaches adapt training

schedules according to an athlete's maturational development (Balyi and Way 2005; Philippaerts et al 2006; Malina et al 2015). The range of age and height velocities at PHV also differs with Tanner et al (1966) finding that boys could be growing anywhere from 7.0 – 15.5 cm/year, more than double the lower value. This shows there is a great range and capacity for bone growth velocity and in their timing, which is highly individual and variable (Tanner et al 1966; Marshall and Tanner 1969; Malina et al 2015; Read et al 2016) (Table 7).

Table 7. Summary of PHV data from various authors

Author(s)	Age at PHV (years)	PHV (cm/year)	Cohort (N =)
Tanner et al 1966	14.1 ± 0.1	10.3 ± 0.2	Male school children (49)
		7.0 – 15.5 (range)	
Beunen & Malina 1988	14	9	Not stated
Buckler 1990	14.12 ± 0.9	9.8 ± 1.2	Male school children (216)
Bailey 1997	13.4 ± 0.7		Male school children (113)
Mirwald et al 2002	13.45		
Rauch et al, 2004	13.45 ± 1	10.4 ± 1.3	Male school children (70)
Philippaerts et al, 2006	13.8 ± 0.8	9.7 ± 1.5	Male elite footballers (33)
Malina et al 2015	11.5 – 17.3 (range)	8.2 – 10.3 (range)	Summary review (25 studies)

Timing of peak growth varies considerably but has been described as occurring early, middle or late dependent on the age of the child (Tanner & Davis 1985; Ross & Marfell-Jones 1991) (see Figure 20). Buckler (1990) described these as sub-groups with and noted marked differences in growth patterns. Buckler (1990) defined the earliest and last 20% chronological age relative to biological/ skeletal age, to determine the early and late maturation groups, with the 20-80% range for the middle/ average group. With these groups there appears to be differences in growth patterns with authors describing early developers having higher volumes, greater rates of growth but for shorter durations than middle developers and lesser levels occurring with the late developers (Tanner & Davies 1985; Beunen and Malina 1988; Buckler 1990; Iulaino-Burns et al 2001; Wright et al 2002; Malina et al 2004). However, the late developers had a longer but slower period of growth resulting in proportionally longer legs, but similar eventual adult stature as the middle and early developers (Buckler 1990).

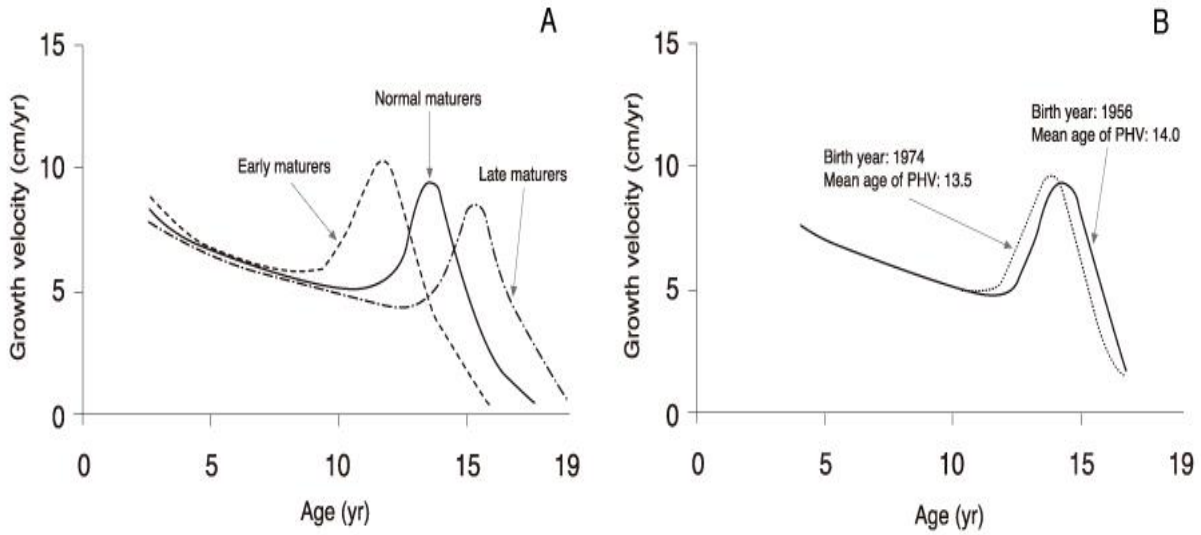


Figure 20. Peak velocity height curves for a) early, middle and late maturing boys and b) secular trend towards earlier maturation. (from Kim et al 2009 citing a) Tanner and Davies 1985; and b) Karlberg 2002)

There is also evidence that PHV is occurring earlier in the general population (Karlberg 2002) but the reason for this has not yet been identified, although over-eating resulting in obesity has been suggested as a trigger (Karlberg 2002). Onset of puberty is also determined by genetic factors as well as environmental (Rona 1981), and other authors also suggested there can be large differences found due to social status, physical activity and diet, ethnicity, and urbanisation. (Rogol et al 2002; Eston & Reilly 2013).

Advanced stature and earlier maturity have been found in elite young footballers (Malina 2003; Philippaerts et al, 2006; Hirose 2009). In the competitive environment of sports academies, taller and more mature players would have a physical advantage over smaller late developers who may share the same chronological age (Balyi & Way 2005), and athletes may be partially selected on the basis of their size and physicality (Malina et al 2000). Malina et al (2000) state that there is an early- middle selection bias together with late developer exclusion that is systematic within the sport of football. This places later developing athletes at a disadvantage and indeed possibility of non-selection, even though they may have the required talent (Vaeyens et al 2008). In the United Kingdom academy football teams are currently selected on the basis of chronological age not biological age (The FA 2012), but there is a growing awareness globally that this gives early maturing athletes a distinct advantage (Musch and Grondin 2001) and other countries and sports are increasingly basing youth sports on biological not chronological age, so called bio-banding (Cumming et al 2017; Read et al 2018).

This enables athletes to be able to compete physically with their biologically-matched peers (Malina and Beunen 1996; Myburgh et al 2016; Cumming et al 2017; Read et al 2018). Drop-out rates were also higher with physically less-mature players in the 11-14-year age range (Figueiredo et al 2009) and Malina et al (2000) also note that late maturing football boys may drop-out as age and sport-specialisation increase. However, although it has been observed that later maturing athletes spend more time on skill acquisition as they cannot initially compete physically (Cumming et al 2017), it may give them greater advantage as senior players once their maturation has caught up. Vandendriessche et al (2012) found any physical advantages found in earlier maturation were eliminated by the time the later developers caught up. Height advantage in early maturation is evened out through adolescence as late matures catch up with slower growth but over a longer period (Tanner 1966; Buckler 1990; Malina et al 2004), and final adult height is often higher in the later maturing cohort (Malina et al; 2004). Mujika et al (2009) noted that this bias towards selecting those with earlier maturation meant that a significant number of talented potential players may be lost to the sport of football. This could have a particular effect at the youth level where academy selection is constrained by age groupings and selection will determine the opportunity for professional training and career development.

4.2.2.2.2 Peak mass velocity (PMV)

PMV in adolescents normally occurs after PHV with Buckler (1990) recording a rapid increase in mass in boys peaking 0.15 years after their PHV. This increase in mass can be large with Buckler (1990) recording some instances of boys doubling their mass over the course of puberty. Their analysis found a peak rate of mass averaged 8.6 ± 1.7 kgs /year, which differed slightly from Tanner et al's study (1966) where boys at their PHV moment were on average 3 kgs lighter, but regained parity by late adolescence. Tanner et al (1966) found a maximal value at PMV of 9.8 ± 0.3 kgs/year and age at PMV of 14.3 ± 0.13 years, just slightly later than their PHV age (14.1 ± 0.13 years). Ranges of PMV values were very wide with Tanner et al (1966) recording PMV from 4.8 – 14.4 kgs/years, a potential threefold difference, and more recently Iuliano-Burns et al (2001) found a PMV of 10.3 kg/y ± 1.9 amongst Canadian boys, and it was noted that this occurred later than PHV in both boys and girls (Iuliano-Burns et al 2001; Malina et al 2004).

4.2.3 Function

4.2.3.1 Flexibility

Flexibility can describe different aspects of skeletal range of motion (Eston and Reilly 2013). It can describe the total range of movement of a joint or one part of the range, which may be achieved passively or dynamically (Hall 2012). It can describe overall range of many joints or body sections in different planes of movement, such as the sit-and-reach test (Strickland et al 2003), or it can describe specific tissue elasticity or passive resistance (St. George 1989; Kendall et al 2005; Alter 2004), or more qualitative definitions such as the ability to readily adapt to changes in position or alignment (Kendall et al 2005). It therefore falls to the topic and aspect being investigated for the specific definition of flexibility to be designated. In this study, the researcher was investigating muscle-tendon unit elasticity or passive tension (Kendall et al 2005) in specific muscles in the leg. Flexibility can be measured quantitatively using range of motion and a goniometer (Kendall et al 2005; Hall 2012; Eston and Reilly 2013) or qualitatively using descriptors such as normal, limited or excessive (Kendall et al 2005). In clinical practice a combination of both types of data are often used to describe a multi-dimensional characteristic and some judgements made based using the experience of the practitioner (St. George 1989; Sahrmann 2002; Kendall et al 2005). The resting position of a limb with respect to gravity is an indicator of specific muscle flexibility when positioned accurately, and this is the measure used to quantify the muscle's internal passive tension (Gossman et al 1982; Kendall et al 2005). Throughout this thesis the terms muscle flexibility and passive tension are used interchangeably.

There has been some recognition of changes to flexibility during adolescent growth (Kendall et al 2005) but limited research as to explain why, however there are very few studies to have investigated the reasons behind this observation. Kendall et al (2005) suggest the initial disproportionate lower limb growth means that classic tests such as the sit-and-reach will be harder for children to achieve until the upper limbs and trunk catch up in later growth. The study by Philippaerts et al (2006) in their cohort of young footballers, found a decrease in overall flexibility at PHV as demonstrated by the sit and reach test. Of note was the declining trend over the 12 months prior to PHV before improvement after. The reduction in flexibility in overall flexion reach may also be related to the timing of peak growth in the legs prior to PHV (Malina et al, 2004). De

Lucena et al (2011) found some rectus femoris muscle tightness amongst 72% of adolescent Brazilian schoolchildren without any pathology, but 93% of those with OSD demonstrated a highly significant difference in tightness.

Furthermore, adolescent football players with OSD had significantly reduced quads flexibility (43%) than their control peer group (Ikeda et al, 2001). Eiichi (2001) found significantly stiffer and weaker quadriceps in the dominant legs in OSD football youths. Furthermore, Ikeda's study found a 43% reduction in quadriceps flexibility in the OSD group. Smith et al (1991) found a strong relationship between poor thigh muscle flexibility and anterior knee pain in elite adolescent figure skaters but did not quantify either the degree of muscle tightness nor specific pathology.

Some authors have described observing reduced calf flexibility in Sever's patients (Szames et al 1990; Madden and Mellion 1996) but no specific measurements appear to have been taken in these studies, so there is no guide as to what determined their opinion as 'tight' or 'reduced'. However more recently Becerro-de-Bengoa-Vallejo et al (2014) found an active range of dorsiflexion in Sever's patients of 7˚ compared with 18˚ in their control group.

Conversely Feldman et al (1999) did not find an association between growth and overall flexibility during adolescent growth, but their study measured children in ages predominantly after their likely PHV (mean ages girls 13.9 years, and boys 14.2 years). Although this study found that flexibility decreased during the summer months during the period of greatest growth, they were unable to contribute their findings directly to the growth patterns observed. Aligning their research time frame to the periods of greatest adolescent growth i.e. Phase 3 and as seen in Fig 19 (Balyi and Way 2009) may have produced different findings.

The association between growth parameters, especially during adolescent Phases 3 and 4 (Balyi and Way 2009), and specific muscle flexibility, should be examined to determine whether there are aspects that could limit range and thus performance in the legs. General flexibility tests such as the sit-and-reach test which are commonly used (Feldman et al, 1999; Philippaerts et al, 2006) do not identify specific muscles which are affected in AS. Targeting these individual muscles would have greater relevance (Kendall et al 1995; Ayala et al 2013; Cejudo et al 2015) in measuring adolescents at risk of AS.

4.2.3.2 Strength

Muscle strength increases during puberty in response to hormonal changes and local stimuli. In athletes this is more marked due to greater local resistance from sports and physical activities, and sometimes targeted training (Buckler 1990; Faigenbaum et al 2011) but the natural peak gain in muscular development occurs after PHV (Malina et al 2004). However, Phillippaerts et al (2006) study of 33 youth footballers showed a variety of overall functional strength and speed tests also corresponded at the moment of PHV, not later. These tests included sit-ups (trunk strength), bent-arm hangs (upper body muscle endurance), jumps (explosive lower limb power) and sprints (speed and acceleration). However, it was a relatively small study population and there was not any specific muscle testing done so only overall performance in each area could be gauged. It would be interesting to observe differences in individual muscles during puberty to give more detailed information on the effect of rapid growth on muscles and therefore the load on their apophyseal attachments. Increases in muscular strength may be relevant to AS as it could be responsible for greater active tension on the apophysis, but it cannot be tested amongst patients due to decreased load tolerance of the condition, therefore measuring strength longitudinally may be useful during adolescent growth (Faigenbaum et al 2011; Read et al 2018).

AS only occur during the secondary growth spurt of adolescence and are more common in athletic individuals. Together with the observed reduction in flexibility seen in AS patients there is justification to study changes in anthropometric, growth and flexibility parameters in a cohort of adolescent athletes in a longitudinal study and ascertain if any differences are found between those that developed AS and those that did not. It would also be interesting to determine if any relationships between growth parameters and flexibility of muscles exists.

4.2.4 Aim

To identify any risk factors for the development of AS by measuring differences in the growth stage, flexibility and anthropometric characteristics in adolescents diagnosed with AS compared to a control (non-affected by AS) counterpart cohort over a 5-year period in an elite academy football setting, and their response to the treatment.

Objectives:

To determine the incidence rate and profile of AS within a football academy over a 5-year period, and their response to treatment using the protocol described in Chapter One and Two.

To analyse any differences in growth between AS patients and their matched control cohort

To determine any changes to flexibility in the quadriceps and calf muscles affected by AS.

To assess any relationship between growth rate and flexibility during adolescent growth

4.2.4.1 Null Hypotheses

H_{01}: There are no differences in growth characteristics between the academy control group and the AS group

H_{02}: There are no differences in muscle flexibility between the academy control group and the AS group

H_{03}: AS patients will recover118 in the usual time frame as described in the literature

4.3 Method

The Crystal Palace Football Club academy gave permission to study growth characteristics in a longitudinal study in their players. Teams ranged from Under 9's to Under 16's, with approximately 20 players per team.

Full ethics approval was gained from the University Ethics Committee and a Risk Assessment was performed and approved by the School of Science Health & Safety Officer. Players and their parents/ guardians were given participation sheets and consent forms at the study start, and were then invited to participate (Appendix IV, V and VI). All players and their parents gave consent for the testing and recording of data for use by the academy staff for the purposes of this study and on-going research based on player confidentiality and anonymisation of data. As participants were below the age of 16 years, all monitoring and testing staff had enhanced Disclosure and Barring checks completed.

4.3.1 Procedures

Planned measurements of standing and sitting stature, and mass were already being taken as part of the ongoing monitoring of youth athletic development following guidelines from The Football Association Youth Development policy and FIFA (Crystal Palace Academy Guidelines 2012). The additional measures for this study were for quadriceps and calf muscle resting position which is an indicator of passive flexibility (Kendall et al 2005; Hall 2012), leg segmental lengths, and thigh and calf girths. The International Society for the Advancement of Kinanthropometry (ISAK) testing procedures (Stewart and Marfell-Jones 2011) were followed for all the anthropometric measures, and Kendall's muscle testing for determining flexibility (Kendall et al, 2005).

4.3.2 Setting and frequency

Testing was performed mainly at the Crystal Palace training grounds in Beckenham, Kent prior to Sunday morning matches throughout the season, or occasionally at the Crystal Palace National Sports Centre. Consistency in the time of day was aimed for to reduce diurnal variation in measurements (Buckler 1990). Testing dates were scheduled, and measurements were taken on consenting participants approximately every two - three months over a period of four seasons (5 years), and before training or matches so that players and coaches were not inconvenienced. Summer break in the off season meant there was a period of around 3-4 months where players were

unavailable, and testing was unable to be done, therefore we aimed to achieve 3 to 4 tests regularly spaced out over each season.

4.3.3 Monitors

Testing was performed by Academy Physiotherapists, Sports Science students and the researcher. All monitors had prior training into measuring accurately for each test, and supervision of testing occurred on all occasions by either the researcher or Head Academy Physiotherapist. All monitors performed the same test measure for consistency and recorded the same players throughout. The researcher was responsible for all data collection. Any onset of apophysitis syndromes in players was reported by staff to the Head Academy Physiotherapist who confirmed the diagnosis and recorded an extra set of anthropometric data at the time.

4.3.4 Anthropometric monitoring

Four stations were set up to measure the different components. The researcher was responsible for all flexibility measures and all data collection. Players wore basic football shorts and shirts only. Shoes, boots, socks, hats, jumpers and tracksuits were removed prior to testing. Mass, stature and sitting height have been shown to have a reliability of measuring in excess of $R = 0.97$ (Marks et al 1989).

4.3.4.1 Stature, mass and leg dominance

Standing stature was measured using the stretch stature method (Stewart and Marfell-Jones, 2011) measuring from the floor to the vertex of the head. Players were instructed to stand with their heels, buttocks and upper part of their back in contact with the vertical wall. Players were asked to “look forwards” and “take in and hold a deep breath” whilst being measured, keeping their eyes level in the Frankfort plane and head still. The Frankfort plane is determined when the lowest margin of the eye socket is horizontal to the tragus of the ear. The monitor applied a firm upwards traction to the player’s head by cupping the jaw and lifting upwards to achieve stretch stature, whilst maintaining heel contact with the floor. The tape measure or stadiometer was brought firmly down onto the vertex of the head flattening the hair as much as possible (Figure 21). A fixed drop-down metal tape measure (Stanley) was used at the training ground and Leicester portable stadiometer (Marsden) was used at the National Sports Centre, with both pieces of equipment checked for accuracy prior to testing each session, and precision was determined as ± 1mm. Measures were taken in metres to the 3rd decimal (mm), e.g. 1.234 m. Once the measure was taken the player was asked to exhale and

step away before being tested again in the exact same method. If these measures were less than 4 mm different, the mean was recorded. If it was more than 4 mm different, a third test would be done and the median recorded (Mirwald et al 2002; Balyi & Way 2005).

Figures 21 and 22. Measuring stretch stature in standing and sitting (Marfell-Jones 1991, pg 19 and 20 respectively).

Sitting stature was determined by sitting on a bespoke wooden box set at 40/45/50 cm heights. This catered for the wide variation in tibia and femur lengths of individual players and meant each player could be accommodated to sit squarely on the box with feet resting flat on the floor and sacrum and upper back firmly pressed against the wall, with knees and hips at 90˚ angles (Figure 22). Box height was recorded that best suited the individual player on the day. Players were asked to “sit tall” on the box with hands resting on their thighs, and take a deep breath and hold, keeping the head and eyes level and still in the Frankfort plane. A gentle upward traction was applied by the monitor in the same manner as for standing height, and the tape or plane was lowered to the vertex of the head. The distance from the floor was recorded in metres to the 3rd decimal (mm) e.g. 0.987 m. Sitting stature was repeated as for standing with the mean of two tests recorded if differences were less than 4mm, and three tests median if differences greater than 4 mm (Mirwald et al 2002; Balyi & Way 2009). The box height was then subtracted to obtain the sitting (trunk) height. This then gave rise to leg length = Standing stature – Sitting stature

Body Mass was determined using a consistent set of scales (Seca 875 Electronic Class III), measured in kilograms to 1 decimal place. Batteries were checked prior to testing, and they were calibrated weekly. Players were asked to wear minimal football clothing (i.e shorts and shirts only) with shoes, socks, jumpers and tracksuits removed. Football

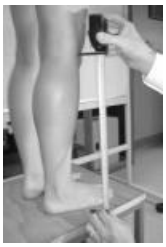
kit worn was thus consistent each time. Scales were checked to be reading zero then players were asked to step on the centre of scales with weight evenly distributed on both feet and remain still without support. Body mass was recorded to the nearest 0.1 kg. Players then stepped off the scales and repeated the test. If differences were more than 0.4 kg then three tests were performed, and the median taken. If differences were less than 0.4 kg, then the mean of two tests was taken and recorded (Bayli and Way 2009).

Player's dominant leg was nominated by each player – Right or Left. If a player was ambidextrous, they were asked which their preferred foot for kicking a penalty was.

4.3.4.2 Bone segmental length

Length of leg bones was measured in two segments: upper (femoral) and lower (tibial) following the ISAK method (Stewart and Marfell-Jones 2011). A metal tape measure (Lufkin speciality Executive Diameter tape W606PM, as recommended by ISAK) was used measuring the lengths in centimeters (cm) to one decimal point, and this was later converted into meters to the 3rd decimal place. Physiotherapists were used for this testing as their anatomical knowledge was deemed more proficient than the sports science students. The same monitor remained at the one station and measured the same players. Two measurements were done of each segment and if the difference was less than 0.4 cm, the mean was taken. If the difference was more than 0.4 cm, then another test was performed and the median recorded.

Measurement of the upper segment was taken from landmarks of the greater trochanter to the lateral tibia condyle with the player standing (Figure 23). Measurement of the lower segment landmarks were the medial tibial condyle and medial malleolus. The player remained standing for this test but asked to stand on a box for greater monitor access and accuracy (Figure 24). All data was collected by the researcher.

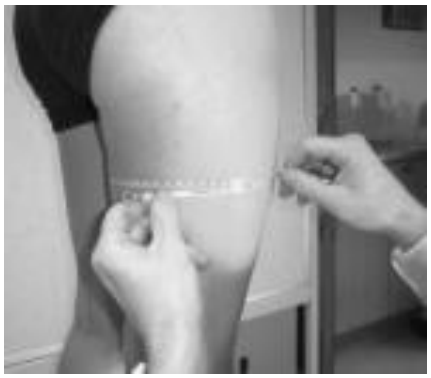


Figures 23 and 24. Femur and tibia lengths (Marfell-Jones 1991; pg. 12)

4.3.4.3 Girths

Girths were measured using the ISAK method (Stewart and Marfell-Jones 2011) and a girth measuring tape (Seca 201) in centimeters (cm) to the nearest 0.1 cm and the girth was measured perpendicular to the longitudinal axis of the segment. The player stood with feet slightly apart and weight evenly on both feet in a relaxed state. Measures were taken twice, and the mean recorded if the difference was less than 0.4 cm, or if greater than 0.4 cm a third measure was taken and the median recorded.

The thigh landmark was the midpoint between the greater trochanter and lateral tibia. The tape measure was used to determine the half-way point between these landmarks and a felt pen was used to mark the landmarks and midpoint. The tape was then held horizontally and wrapped around the thigh at the midpoint, and the measurement taken at the point where the tape crossed itself. (Figure 25). Calf girth was measured at the point of maximal girth of the calf and where the tape crossed back on itself (Figure 26).



Figures 25 and 26. Thigh and calf girth circumference measures (Marfell-Jones 1991, pg 15)

4.3.4.4 Peak Height Velocity (PHV)

The PHV was estimated mathematically by recording height changes (cms / weeks) between measures for every player. There were sometimes uneven periods of time between measures although attempts were made to record every 2 to 3 months for consistency. Recording height changes divided by the number of weeks allowed for greater accuracy in rates, and then converted into a monthly rate for more meaningful comparisons to be made between groups and with the literature. Therefore, the data is presented as monthly rates of growth as is the standard unit (Mirwald et al 2002).

4.3.5 Muscle flexibility

Measurements of resting length with respect to gravity for the gastrocnemius, soleus and quadriceps muscles were taken to determine the muscle's passive tension. The positions chosen depended on the specific muscle being tested (Kendall et al 2005). A standard manual 12-inch goniometer (NCD Prestige Medical) was used to determine joint angle in degrees˚ and the test was repeated, with the player repositioned each time. If readings were less than 4˚ different, the mean was recorded. If differences were more than 4˚ then a further test was performed, and the median taken. Tests were performed bilaterally by the researcher, or occasionally by the senior academy physiotherapist. Reliability for the manual goniometer measurements has been reported as high with intra-tester *R* =0.90 (Gajdosik and Bohannon 1987)

4.3.5.1 Quadriceps muscle

Quadriceps muscle was tested in the modified Thomas test position (Figure 3.5), with the player lying on the end of the plinth, one hip and knee fully flexed to the chest. The position was corrected by the monitor to achieve a neutral lumbar spine and pelvis. The testing leg was allowed to drop and rest in a gravity-assisted position described as the resting position. If the hip was flexed up from the plinth further correction of hip deep flexor tension from the psoas-iliacus muscle was performed by straightening the knee and allowing the hip to drop to neutral. The leg was then allowed to fall into its resting position against gravity but guided to eliminate abduction caused by tight lateral musculature such as the ilio-tibial tract. All these corrections are described by Kendall et al (2005) to obtain the best position to isolate the resting length for the quadriceps muscle and thus its passive tension (Hall 2012). Landmarks used for the axes were the greater trochanter, lateral knee joint line and lateral malleolus and they were marked before testing with washable pen (Figure 27). Knee angle was recorded in degrees of flexion (Kendall et al 2005).

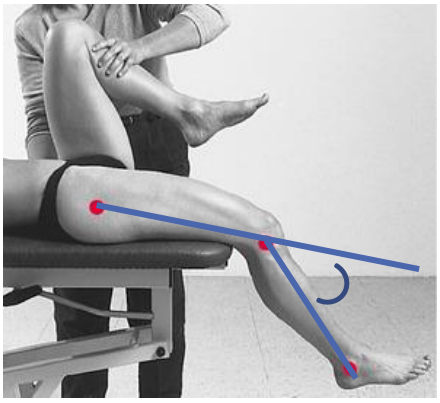


Figure 27. Resting position for the quadriceps muscle using the modified Thomas Test position. Knee flexion angle recorded. (Kendall et al 2005) image from (www.musculoskeletaltalkey.com)

4.3.5.2 Calf muscles

Gastrocnemius passive tension was tested with the player in prone position with their foot passively resting off the end of the plinth, and resting angle of the ankle joint recorded (Figure 28). Note in the actual test no external force is applied, other than gravity. Landmarks for axes were the fibula head, lateral malleolus and a parallel line with the sole of the foot and were marked with a washable pen prior to testing. Measurements were taken as degrees of plantar flexion from neutral (90˚) (Kendall et al 2005).

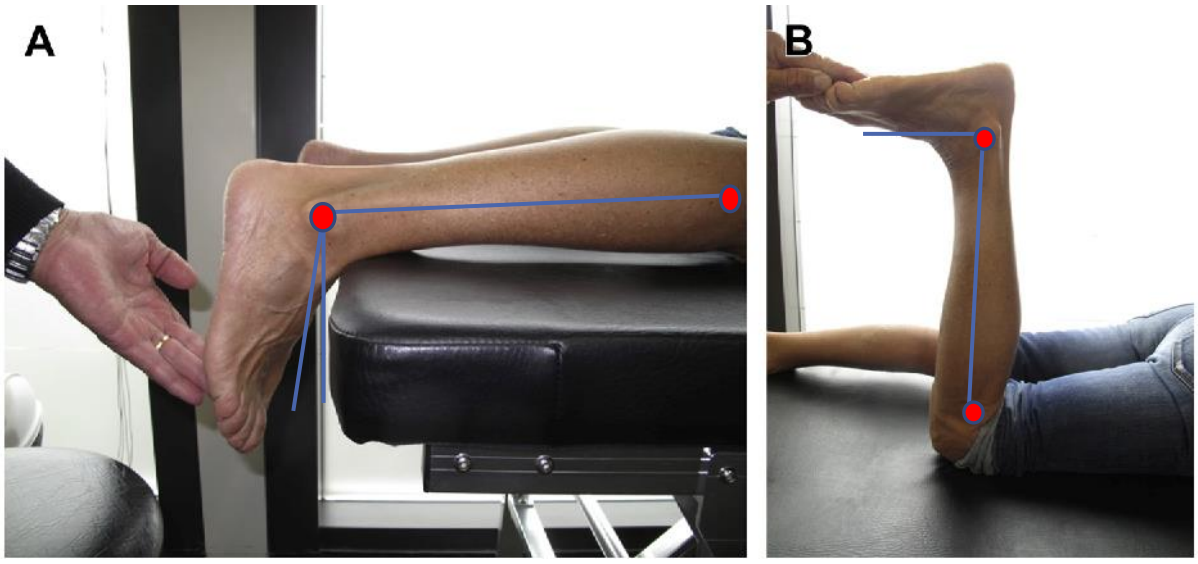


Figure 28. Testing position for A. Gastrocnemius and B. Soleus muscle length. Plantarflexion recorded in degrees (Barouk and Barouk, 2014 pg 660).

Soleus was tested with the player in prone as above but with the knee flexed to 90˚ to eliminate the influence of the gastrocnemius (Kendall et al 2005). Landmarks were the

same as for the gastrocnemius test, and the test was passive with only gravity providing an external force. The resting angle of the ankle was again recorded in degrees of plantar flexion (Kendall et al 2005).

After testing, all data was collected and recorded on spreadsheets by the researcher. Player anonymity was achieved by coding all the players with their team and number based on their alphabetical order e.g. G1-18, then later arrival for new recruits e.g. G19+. Data was kept safely on a desktop computer, with identifying names kept separately to the testing data.

Further information was available from the Academy records regarding individual player ages and birth dates.

4.3.6 Apophysitis patients

Any player who reported an injury was assessed by the Academy medical staff. If the injury was diagnosed as an apophysitis syndrome it was reported the Head Academy Physiotherapist who then performed a full set of anthropometric and flexibility tests as described above. The specific type of AS was recorded, as well as the date it was first noticed by the player. The use of the researcher's treatment protocol as described in Chapter One and Two, was enacted immediately according to the specific AS. The time taken for the player to be pain free on passive stretch and active wall slide was recorded, and the patient was regarded as fully recovered and return to training.

4.3.7 Statistical procedures and analysis

The statistical software package SPSS 25 (SPSS Inc, Chicago, Il, USA) was used for statistical analysis throughout. Descriptive statistical analyses (means, medians, standard deviations and variance) were used for the univariate data for the player anthropometric profiles of age, height, mass, BMI, PHV and flexibility. The raw data sets were assessed using the Shapiro Wilk test to determine normal distribution. Testing for significance of differences between the AS and CG groups and within AS sub-groups used both parametric and non-parametric testing depending on the distribution. Flexibility differences were analysed using parametric independent T-tests (2-tailed). Differences between groups in age, height velocity, PHV, mass, BMI were analysed using non-parametric tests of Mann-Whitney U, and Kruskal-Wallis for differences across the AS sub-groups. Spearman's correlation was used to explore any

association between flexibility and PHV. All analyses were performed to a significance level of $P < 0.05$ and with a 95% confidence interval.

4.4 Results

4.4.1 Academy profile

There were 220 Crystal Palace academy footballers monitored over 4 seasons from 2011 to 2015. Of these, 31 players developed an apophysitis syndrome (AS =14%), and therefore the non-affected cohort were designated as the control group (CG = 189 or 86%). Each team consisted of 20 players but numbers on a set testing day could differ due to absence or lateness. Player personnel changes also occurred during the 5-year study as some boys were 'released' i.e. dropped from the academy and replaced with new recruits. Teams could therefore have squad membership over the 5 years of more than 20 players. Of the 220 players assessed, 32 were released and replaced with 32 new players, therefore leaving a core cohort of 156 players for the full longitudinal study time frame.

A total of 92 sets of data were recorded over the 4 seasons with most teams having approximately 3 testing sessions per season or roughly one every 3-4 months (see Table 8).

Table 8. Data collection sessions per season per team.

TEAM	2011-12		2012-13		2013-14		2014-15		Totals
	Sessions	Age	Sessions	Age	Sessions	Age	Sessions	Age	
A							3	8	3
B					4	8	4	9	8
C			2	8	5	9	4	10	11
D			2	9	4	10	5	11	11
E			3	10	4	11	4	12	11
F	2	10	3	11	4	12	4	13	13
G	3	11	2	12	4	13	4	14	13
H	1	12	4	13	3	14	4	15	12
I	2	13	4	14	4	15			10
Totals	8		20		32		32		92

Team I was the oldest at the start of the study and Team A as the youngest, came on board only at the study end in 2014-15. Teams C – H were the primary focus for the data analysis as these covered the age ranges where AS are likely to occur i.e. from 9 – 15 years old, and we had access to at least 3 seasons of data from them. Team ages ranged from Under-9-year-olds to Under-16's inclusive.

There were some inconsistencies with timing of testing dates which resulted from last minute changes in match days, holidays, weather conditions and off-season periods, where testing was not possible, but scheduled dates covered even periods of time i.e. every 2-3 months, and missed dates were re-scheduled at the first opportunity thereafter. Usually 2 teams were measured each week, on a Sunday morning prior to matches played. Consistency was achieved by monitors through prior training and each monitor keeping to the same station measurements and measuring the same players. All data was collated and recorded by the researcher.

4.4.2 Apophysitis syndrome profile

AS was diagnosed in 31 players with the onset during this study, which gave an overall incidence of 14%, however when looking within each specific team the incidence was as high as 28.0% (Team G), with 2 other teams having over 20% (Teams H and E) (Table 9). The mean per team from the target age groups of C–H had an incident rate of 19%. Team I was older than the target age and Teams A and B were at the youngest age with only one year's data, we did not have access to the historical data for the older teams. Two players had a double pathology (two separate AS) and one player had a recurrence of the same AS, therefore there was an incidence of 34 AS recorded amongst 31 players.

Table 9. AS incidence per team 2012-2016 (Incidence of 20% or over highlighted in bold.)

Team	AS	Players with AS	Squad size	%
A	0	0	23	0
B	1	1	24	4.2
C	5	4	26	15.4
D	3	3	21	14.3
E	7	6	30	**20.0**
F	4	3	22	13.6
G	7	7	25	**28.0**
H	6	6	27	**22.2**
I	1	1	22	4.5
Total	34	31	220	

OSD represented over half of the conditions reported (56%), and Sever's disease 26%. There were smaller numbers of three patients with Sinding-Larsen-Johannsen (SLJ) (9%) and three with hip apophysitis (9%) at the anterior inferior iliac spine (AIIS). The data is summarised in Table 10 below together with mean age of onset.

Table 10. AS sub-groups and age of onset (mean and standard deviation)

AS	Number of patients	Percentage %	Onset Age (years)
OSD	19	56	12.3 ±1.6
Sever's	9	26	11.0 ±1.6
SLJ	3	9	13.0 ±0.5
Hip (AIIS)	3	9	13.2 ±0.5

The ages of the AS patients at onset ranged from 9.5 years old to 16.08 years with an overall mean of 12.1 years old ±1.6, with a normal distribution. The age of the Sever's cohort was also younger than the others but this was not significant statistically (One-

Way ANOVA P=0.064). The age groups of 12-year-olds (32%) and 13-year-olds (24%) had the greatest incidence of AS with the younger 10-year-olds having 20% incidence, possibly skewed by the higher incidence of Sever's disease in that age. All patients were in the foundation or youth age group classifications from the EPPP.

Table 11. Anthropometrics for AS patients at onset (medians and ranges)

AS	Stature (m)	Mass (kgs)	BMI (%)	Height Velocity (cm/ month)
OSD	1.63 (1.46 – 1.84)	48.73 (34.5 – 69.8)	18.17 (16.1 - 21.53)	1.00 (0.87 - 1.08)
Sever's	1.55 (1.45 – 1.69)	41.97 (36 -50)	17.35 (16.07 – 18.61)	0.87 (0.72 – 0.96)
SLJ	1.71 (1.65 – 1.76)	55.77 (50.3 – 59.5)	18.98 (18.69 – 19.88)	1.00 (1.00 – 1.08)
Hip	1.70 (1.64 – 1.75)	56.93 (53 – 60.8)	19.48 (18.83 – 19.92)	0.99 (0.99 – 1.04)

The distributions for the anthropometrics for the different AS sub-groups were skewed to the left with non-normal distributions and are summarised in Table 11, and there were significant differences across the group for mass (Kruskal-Wallis $P < 0.01$) and BMI (Kruskal-Wallis $P = 0.05$). Stature showed a borderline result, but it was not statistically significant (Kruskal-Wallis $P= 0.052$). However, post hoc statistical testing between cohort groups using the Mann-Whitney U test, found significant differences in mass and BMI between Sever's patients and those with SLJ and Hip AS ($P<0.02$). Significant differences in mass were found between OSD and Sever's, and between Sever's height and those of SLJ and Hip AS ($P <0.05$). (Table 12).

There was no significance found in height velocity at onset across the AS groups (Kruskall Wallis $P =0.86$), nor between groups (Mann Whitney U test $P = 0.58$).

Table 12. AS sub-group anthropometric differences (*= statistically significant, NS=no significance)

	OSD	Sever's	SLJ	Hip
OSD	x	Height = NS Mass P=0.048 * BMI = NS	NS	NS
Sever's	Height = NS Mass P=0.048 * BMI = NS	x	Height P =0.036 * Mass P =0.018 * BMI P =0.018 *	Height P=0.036* Mass P = 0.009* BMI P = 0.009*
SLJ	NS	Height P =0.036 * Mass P =0.018 * BMI P =0.018 *	x	NS
Hip	NS	Height P =0.036* Mass P = 0.009* BMI P = 0.009*	NS	x

4.4.3 Peak Height Velocity (PHV)

Direct comparisons of players across the academy were difficult to make with any accuracy due to the wide variation and timing of adolescent growth spurts, therefore PHV was used as the single common marker of adolescent growth (Buckler 1990, Malina et al, 2004) and comparisons made on all the anthropometrics at this snapshot marker in time, to create a cross-sectional view of the period prior to, during and after the peak.

Estimates for age at PHV were obtained in 73 of the control group and 30 of the AS group. The distributions for age at PHV were not normal so non-parametric testing was performed to determine any significant differences between the groups. The age at PHV was a median of 13.0 years ± 0.98 for the CG and 12.4 years ± 1.08 for the AS group (see Figure 29). The peaks seen in the graph appear to show earlier maturation in the AS group, although there was no statistical difference between the group medians (Mann-Whitney *U* P=0.14).

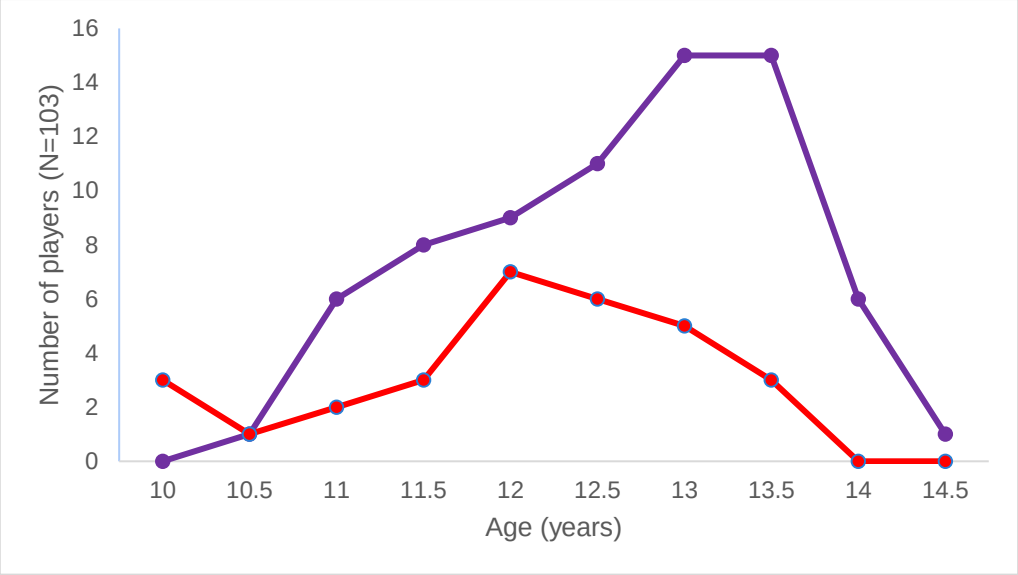


Figure 29. Age at PHV (AS = red; CG = purple)

Stature at PHV had a normal distribution and showed no significant difference between the groups with the AS group having a mean of 1.64 ± 0.09 m height and the control group 1.65 ± 0.07 m (Two-tailed T-test with equal variance P =0.62). Velocity of growth was determined by calculating growth in mms and dividing by the number of weeks between testing dates. This was then multiplied by 52 (weeks) and divided again by 12 (months) to give an estimate of monthly growth, which is the commonly used unit of measure for PHV (Malina et al 2004; Balyi et al 2013). However, comparing the velocity of growth at PHV showed significant differences between the groups with the CG median of 0.95 cm/month ± 0.08 growth compared with 1.05 cm/month ± 0.06 in the AS group (Mann Whitney U P <0.01). The distributions were not normal for PHV (see Figure 30 and Table 13). Although this was a statistically significant difference the reality of the small change seen over a short period means the differences are too close for practical use. Assessing the stature change over the preceding year again found a difference in total growth means and SD of 10.15 cm/year ± 1.2 in the AS group (n=23) and 9.54 cm/year ± for the CG(n=58). The distribution was tested for normality using Shapiro Wilks and an Independent Samples T-test (p =0.035) showed a statistically significant difference between the groups with the AS growing more than the CG. This difference however, is too close for practical use in the real world although the trend comes through and is worth reporting. Cohen's d test found a medium effect size of 0.53.

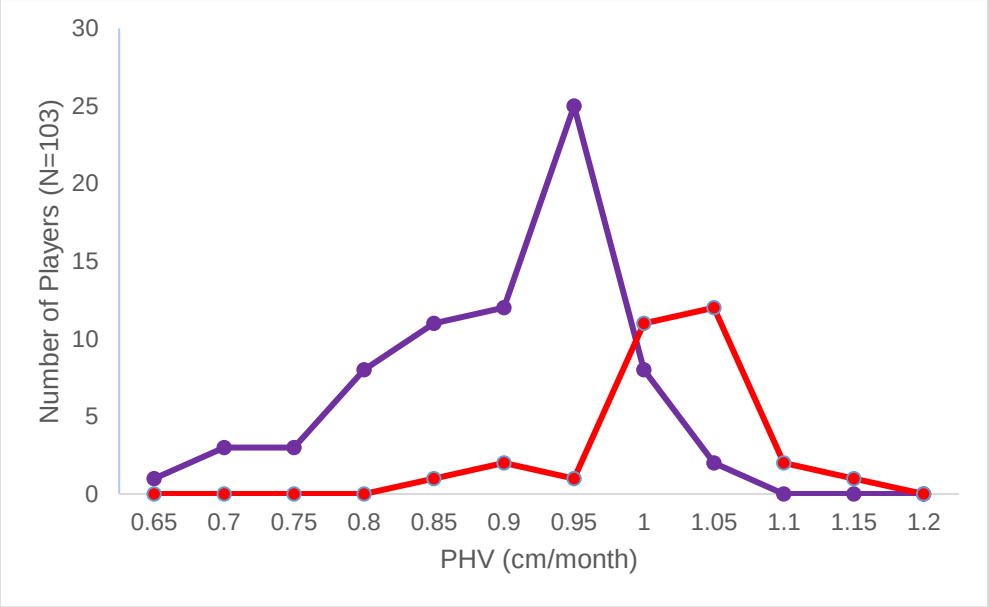


Figure 30. PHV comparison between control and AS groups (cm/month) (AS = red; CG = purple)

Combining these sets of data provides for a comparison of age and growth velocity between the CG and AS groups at PHV. (Figure 31)

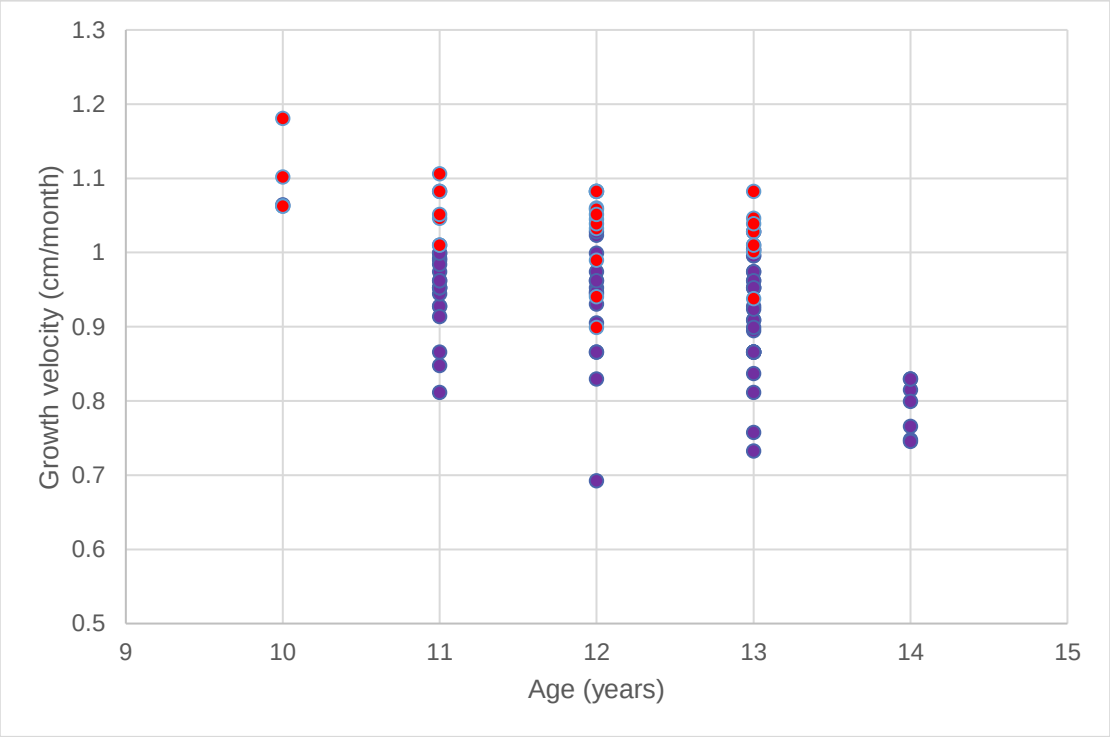


Figure 31. Age and growth velocity at PHV (AS = red; CG = purple)

Further analysis of each specific AS height velocity at onset and at PHV (summarised in Table 13) in distribution and comparison of medians was also found to be significantly different from the CG (Kruskal-Wallis $p<0.01$). However, the median height velocity at actual AS onset was 0.96 cm/month (range 0.72 -1.08), was already higher than the CG, and when compared statistically with the CG PHV both the medians of the AS onset height velocities and specific sub-group AS height velocities were significantly different. (Independent median test P<0.01) and distributions (Kruskal-Wallis P<0.01). (Table 13)

Table 13. Height velocities at peak and at AS onset compared to Control Group (medians and ranges; * differences statistically significant)

	PHV (cm/ month)	AS Onset (cm/month)
Control Group	0.95 (0.69 - 1.08)	-
AS group	1.05 (0.89 - 1.18) *	0.96 (0.72 - 1.08) *
OSD	1.04 (0.94 - 1.18) *	1.00 (0.87 - 1.08) *
Sever's	1.05 (0.89 – 1.08) *	0.87 (0.72 – 0.96) *
SLJ	1.00 (1.00 – 1.08) *	1.00 (1.00 – 1.08) *
AIIS	1.04 (0.99 – 1.08) *	0.99 (0.99 – 1.04) *

4.4.4 AS Onset – PHV relationship

The onset of the specific AS was slightly different with respect to PHV, but generally occurred close to their PHV, therefore measures for comparative analysis were taken from both groups at the individual's PHV timing. The distribution was normal and the mean onset for AS was 2 weeks prior to PHV, with a standard deviation of ±13 weeks, with 50% of players having their onset coinciding ± 1.9 months near the timing of their PHV; 33% developed their conditions prior to their PHV and 17% after. The range was from -31 to +29 weeks, so there appears to be a window of presentation around the PHV ± 8 months, with a clear approximation between the timing of PHV and AS onset (Figure 32). Data sets were organized into monthly band widths.

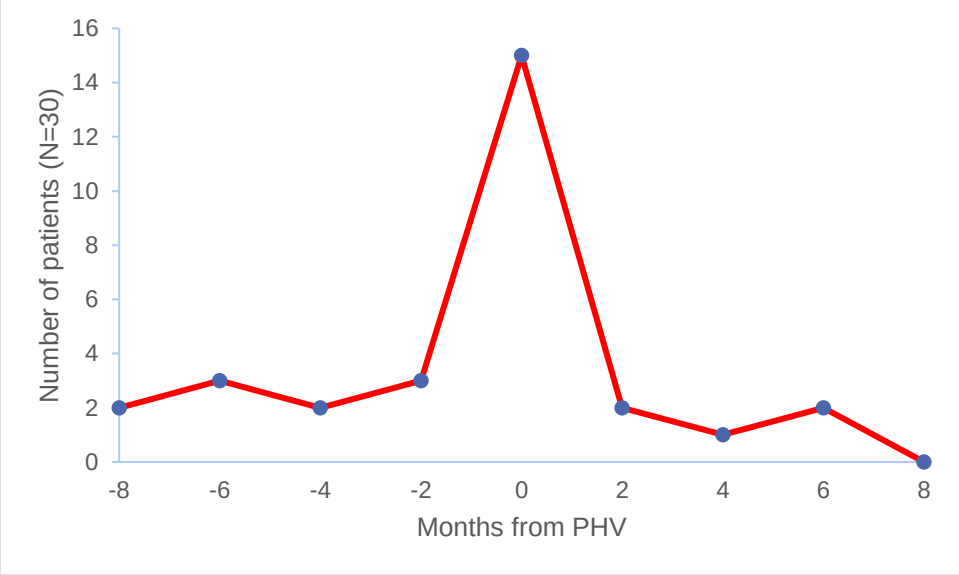


Figure 32. Relationship of AS onset timing and PHV

More detailed analysis for the two largest AS showed a significant difference in onset relationship between Sever's and OSD (Independent Samples Median test p =0.028), with Sever's occuring at a median of 24 weeks prior to PHV (range 32 weeks prior to 8 post-PHV), with the OSD median occuring at PHV (range 16 prior to 32 post-PHV) (See Figure 33). The difference between the two group distributions was statistically significant (Mann-Whitney U p<0.01), so Sever's disease tends to occur earlier in the growth spurt and OSD is closely associated with PHV. The six players with SLJ and AIIS all had their onset at their PHV timing (0 months).

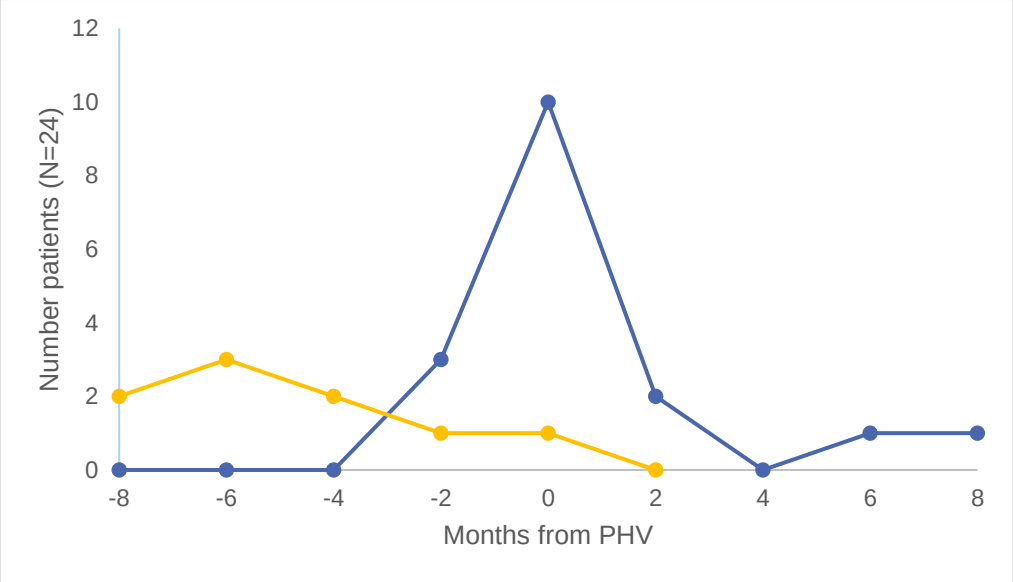


Figure 33. Timing of OSD and Sever's onset with respect to PHV at 0. (OSD = blue; Sever's = orange)

Mass of players recorded at PHV showed the AS group with a median of 45.0 kg (range 32 - 70) and the CG median of 50.2 kg (range 37 -71). The distribution was not normal and there was a significant statistical difference between the groups with the AS group lighter in mass (Mann-Whitney U test; *p* =0.027). The range was however quite large with the AS group from 31.6 to 69.8 kg and the Control group 37.2 to 71.3 kg, so caution should be used in interpretation (Figure 34).

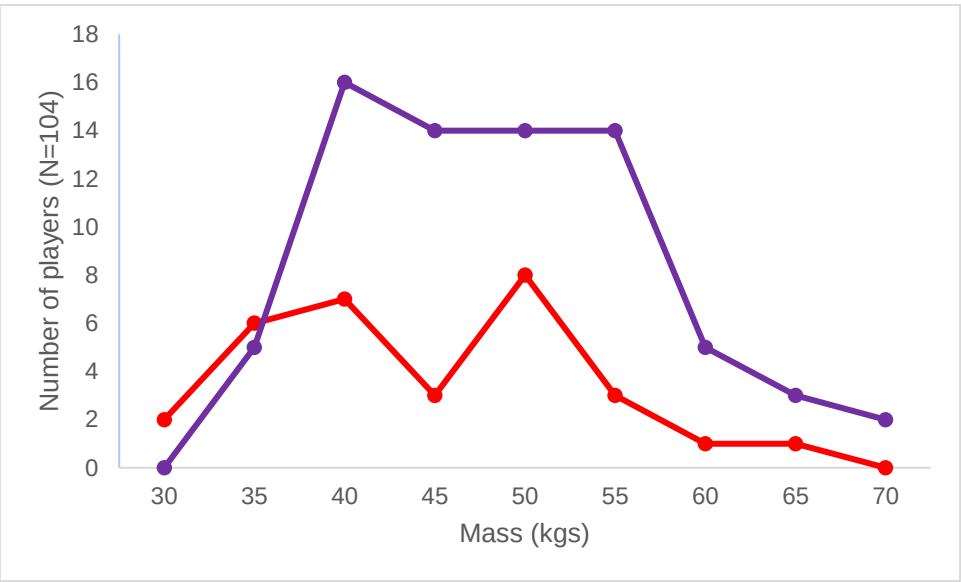


Figure 34. Mass of CG players and AS patients at PHV (AS = red; CG = purple)

Body Mass Index (BMI) at PHV remained within a range from 14 -24% with the AS group median of 17.94% ± 1.4 compared to the Control group median 18.64% ± 2.18 (Figure 35). There was no statistical difference between the groups (Mann Whitney U; *p*=0.211). None of the players were classed as overweight (i.e. above 25% BMI).

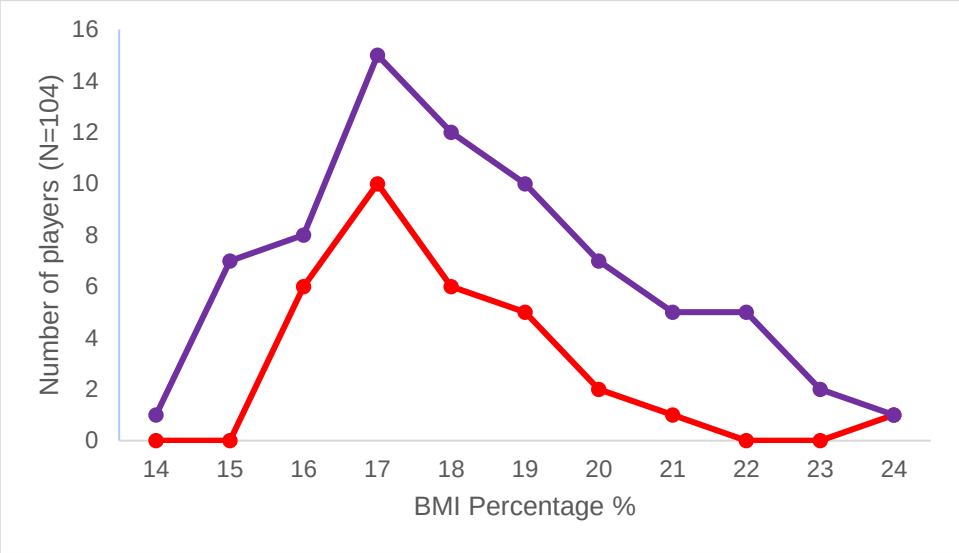


Figure 35. Body Mass Index for AS patients and CG players at PHV (AS = red; CG = purple)

Initial leg segmental lengths were found to be inaccurately measured and therefore this test was stopped after the first year. Leg length to trunk length ratios were determined by subtracting players' sitting heights from their standing heights, and then the legs reported as a percentage of trunk height. The mean leg length as a percentage of trunk height for the AS group at PHV was 94.9% ± 1.8 in the AS group compared with the CG at PHV of 93.5% ± 2.6. The differences were not significant (2-tailed T-test with equal variance *p*= 0.356).

4.4.5 Flexibility

Players were measured on both legs for quadriceps and calf muscle resting position. For this thesis study's analysis only, the dominant leg measure data have been used for comparisons. For those players who were ambidextrous we asked which their favored leg was for kicking and used this as their dominant. Dominant leg data only was used as the total volume of data using both legs was too great for the immediate scope of this thesis.

Flexibility intra-tester reliability was determined previously with an error of ±3˚.

Flexibility changed throughout the seasons and between the year groups. Excluding the AS group and those in the CG who had the interventions, the control group means (N=56 for calf and N=55 for quadriceps measures) showed a trend towards tightening of the quadriceps muscle during 12-14 years of age and the calf muscle at around 10 -12 years of age. (Figure 36)

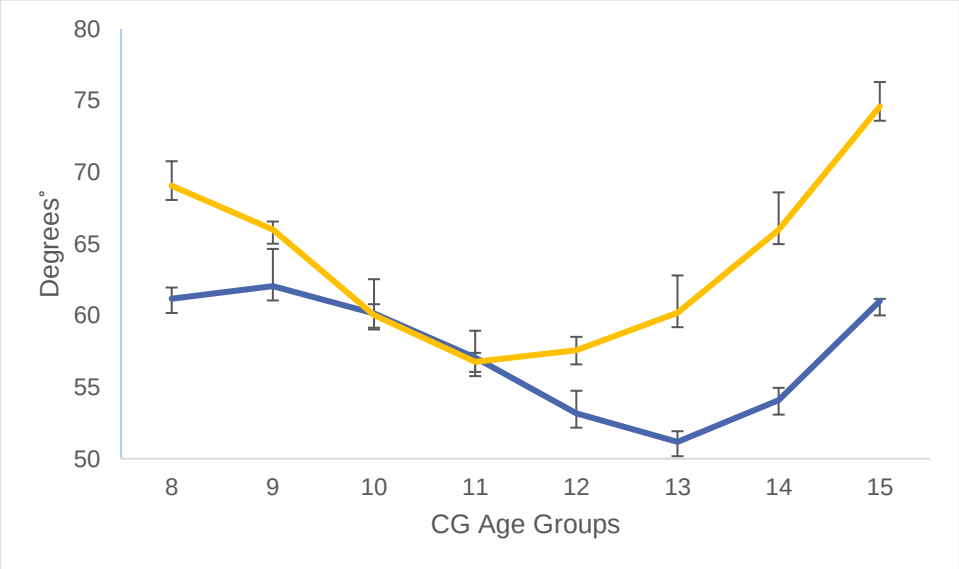


Figure 36. Muscle flexibility changes at each Control Group age group (means and standard deviations) (Quadriceps = blue; Calf = orange)

4.4.5.1 Quadriceps

There was a significant difference between the AS group (N=17) quadriceps flexibility at onset of the relevant AS (i.e. OSD, SLJ or AIIS) vs the control group (N=56) at PHV. The AS quadriceps flexibility was a mean of 36˚ ± 4 against CG quadriceps of 52˚± 4 (Two-tailed T-test with equal variance $p<0.01$) (Figure 37). However, the data set has been adapted as there was an active intervention if players recorded less than 45˚ during testing, and this intervention group's data was therefore excluded from the comparison. A total of 23 players had the quadriceps stretching intervention, and their flexibility subsequently improved. None of these players went on to develop an AS. It is notable that all the AS quadriceps at onset were less than 45˚ at resting length. In this measure, the lower the degree number the tighter the muscle.

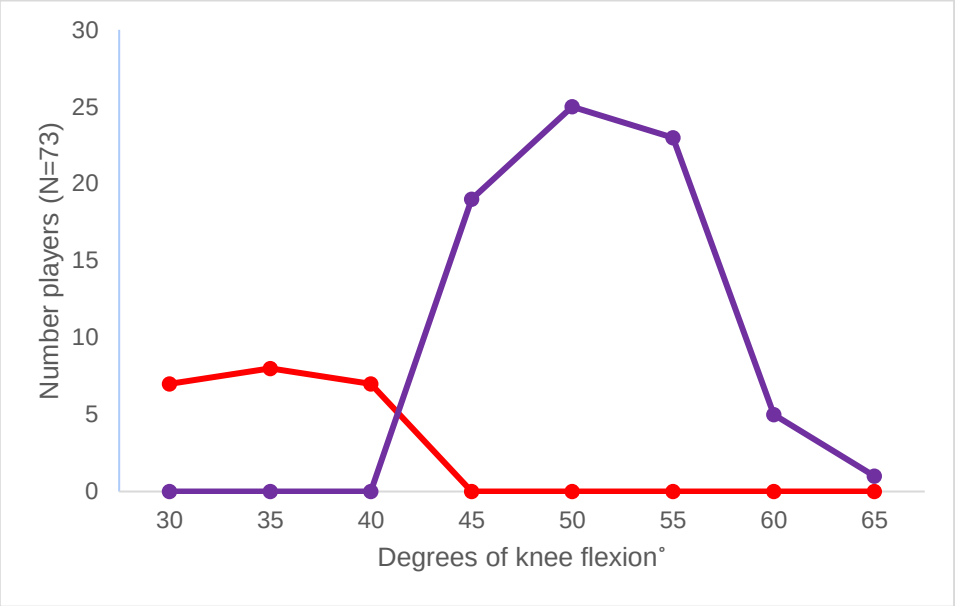


Figure 37. Quadriceps flexibility at OSD, SLJ and AIIS onset compared to Control group at PHV. (AS sub-groups = red; CG = purple)

Mapping flexibility for the year preceding PHV enabled changes to the quadriceps to be recorded. Removing the data sets of those in the CG whom had a stretching intervention, and those who we did not have enough recordings to map a preceding year, left a sample of 55 in the CG and 17 in the AS group. Of note the AS group had reduced their quadriceps by a mean of 21˚ in the preceding year to onset against CG 8˚ at their PHV, the difference was significant (Independent t-test (2-tailed) with equal variance; $p<0.01$). The standard deviations though for both groups were similar at 6 and 5 degrees/year respectively. The CG measures at PHV corresponded to the individual player's lowest flexibility point in 36% of cases, with 51% having their lowest flexibility prior to PHV, therefore 87% of players in the CG dropped to their lowest quadriceps before or at the time of their PHV (Figure 38).

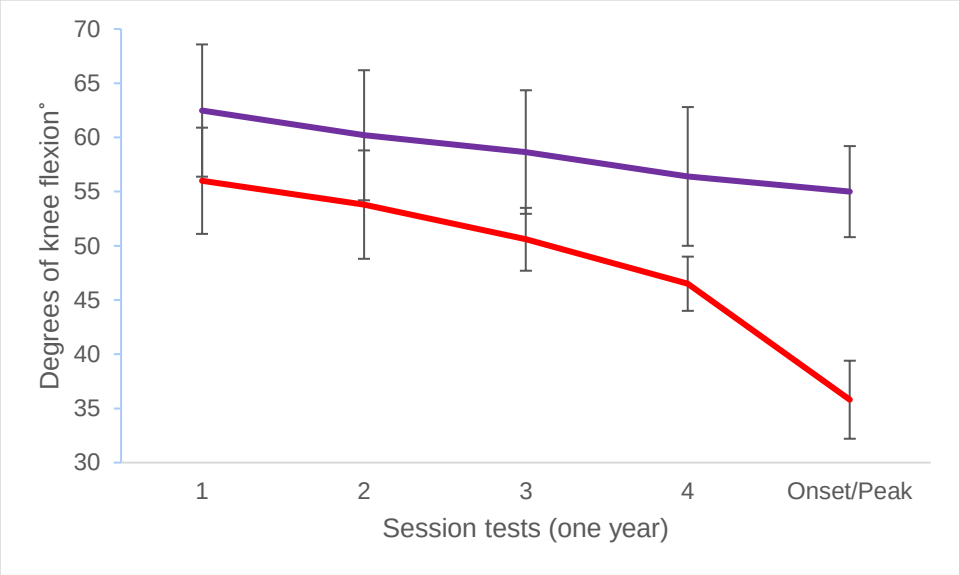


Figure 38. Change in Quadriceps flexibility over one year prior to PHV (means and standard deviations), (AS sub-groups = red; CG = purple)

Allowing for the standard deviations and measurement error means that at any given point in the preceding year the differences between the groups were not significant but the overall decrease over 1 year in the AS group was (21˚) compared to 8˚ (CG).

4.4.5.2 Calf muscle

Calf flexibility had a similar profile with two distinct groups with normal distributions. The AS group with Sever's disease (N=9) had a mean calf flexibility at onset of 44˚± 4 versus Controls at PHV with 34˚± 3 (N=56). Here the higher the degree the tighter the muscle. Again, an intervention was performed for any players showing signs of decreased flexibility of more than 38˚ which will have impacted on the composition of the control group, as these players were thus excluded due to the possible effect of the intervention. 15 players had a calf stretching programme prescribed for intervention, none of whom went on to get Sever's disease, and their data was excluded from the above analysis. A two-tailed T-test with equal variance showed there was a statistically significant difference between the groups ($P < 0.01$). (Figure 39).

The testing of soleus muscle with the knee flexed to 90˚ was stopped during the first year due to time constraints and some players could not achieve the 90˚ test position due to tight quadriceps. Therefore, only gastrocnemius flexibility was recorded for the remainder of the study.

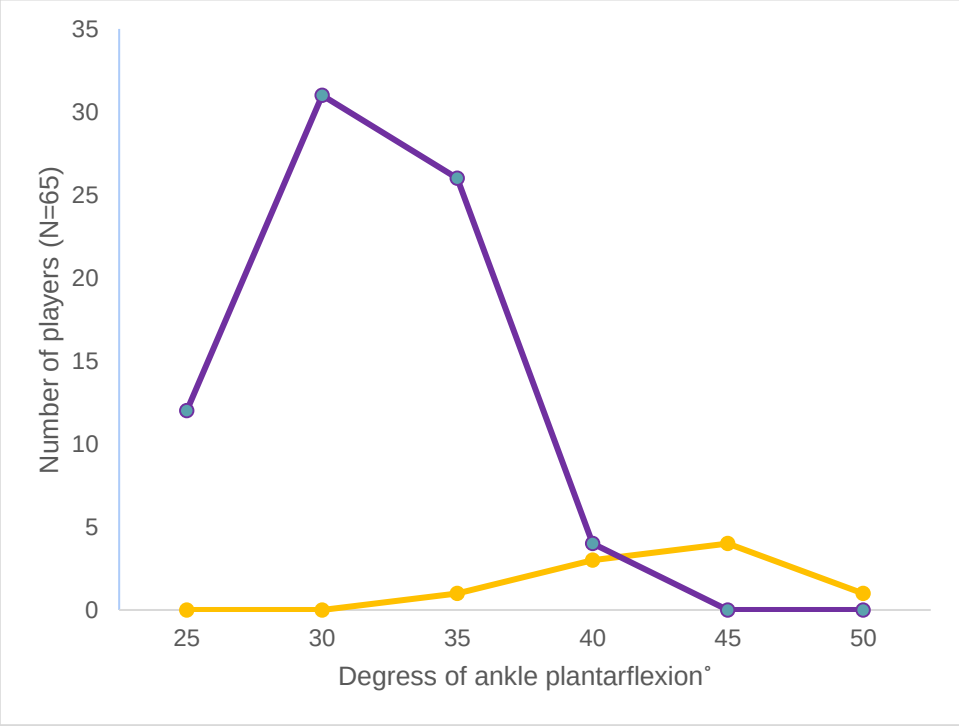


Figure 39. Calf flexibility at Sever's onset and PHV (CG). (Sever's = orange; CG = purple)

Tracking the available players in the year prior to onset of Sever's showed the AS group (N=8) having a reduction of calf flexibility mean and standard deviation of 21˚ ± 8 compared with the CG (N= 56) of 0.3˚ ± 3.8. However, all the Sever's group onset was prior to their PHV by an average of 24 weeks ± 12. When analysing their calf flexibility at the time of their PHV their flexibility was very similar to the CG at 35 ˚± 4 and 33˚± 3.3 respectively, and there was no significant difference at PHV between the groups (2-tailed T-test with equal variance p=0.32). Note the Sever's group had active treatment to improve their flexibility post-onset and all prior to their PHV. The change in calf flexibility over the year preceding PHV showed the CG having a mean decrease of 3˚ but the Sever's group having a mean decrease of 17˚, and this difference was statistically significant (Independent T-test (2-tailed) p<0.001). (Figure 40). However, it is noted that with the small sample number of Sever's patients in the data set, caution should be shown in interpretation of the data.

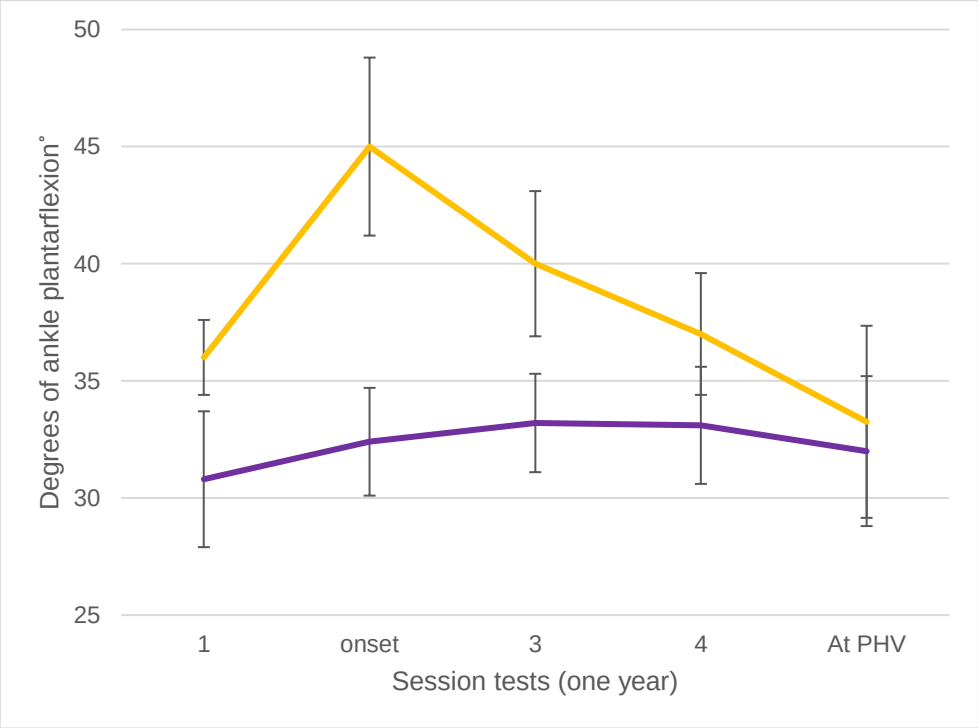


Figure 40. The change over one year in calf flexibility prior to player's PHV and at Sever's onset (means and standard deviations) (Sever's = orange; CG = purple)

4.4.6 Flexibility and PHV

4.4.6.1 Quadriceps and PHV

AS players whose quadriceps were directly involved in their condition (i.e. OSD, SLJ and AIIS) were included in the analysis of the quadriceps flexibility comparison. A strong correlation was found between flexibility and PHV in both the AS and control groups (Spearman's correlation AS = -0.96 compared to the CG = -0.81 (2-tailed with significance P <0.01 for both). Not only did the AS group have tighter quadriceps and higher PHV (36˚ and 1.05 cm/month vs 53˚ and 0.95 cm/month), but the relationship also had a stronger correlation with a steeper negative gradient (Figure 41).

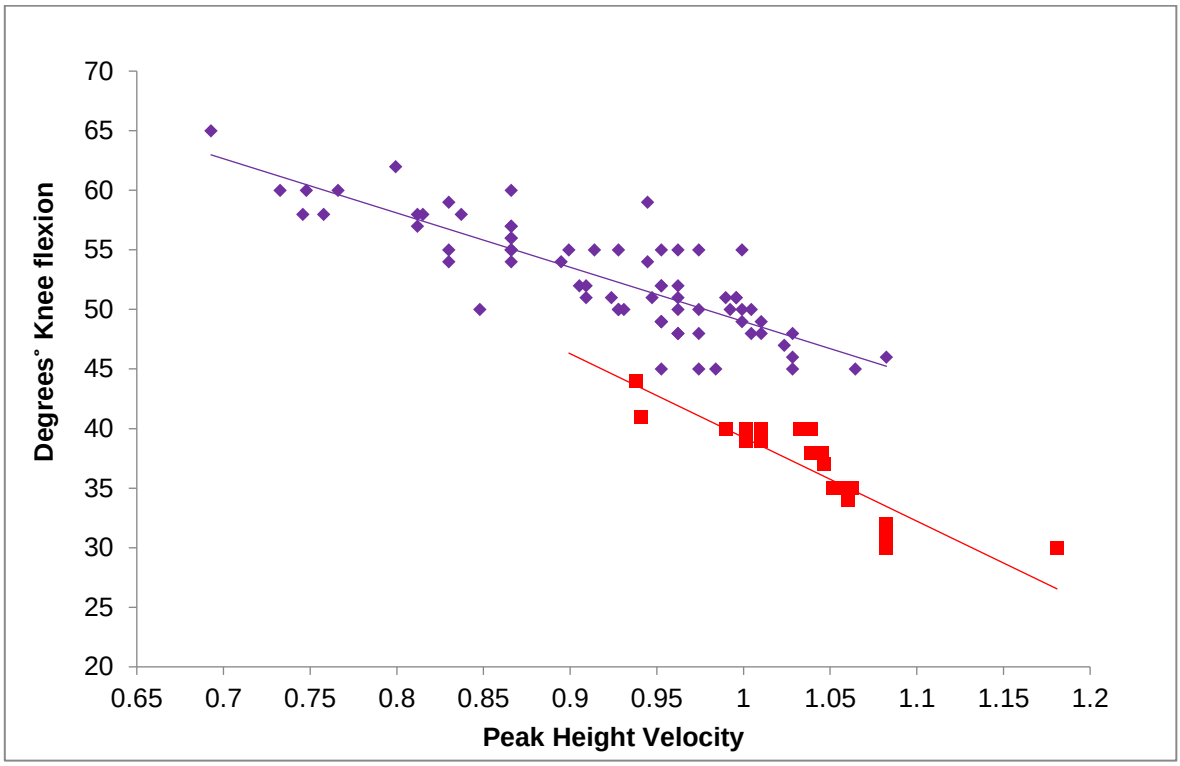


Figure 41. Relationship between Quadriceps flexibility and PHV (AS sub-groups = red; CG = purple)

4.4.6.2 Calf and PHV

A similar relationship was found with calf muscle flexibility and PHV with a strong correlation found for both groups but even stronger for the Sever's group (Spearman's correlation = 0.97 for the Sever's group and 0.78 for the CG; 2-tailed significance for both $p<0.01$). This chart should be viewed understanding that for the calf the higher the degree the tighter the muscle (Figure 42).

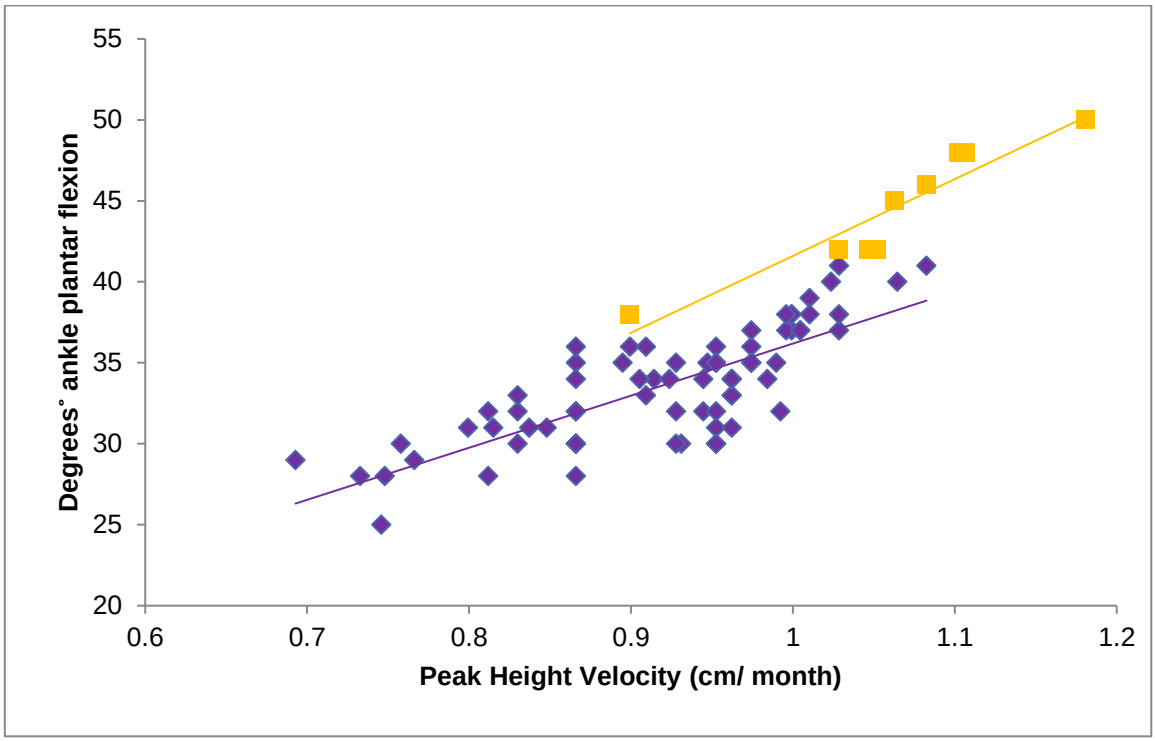


Figure 42. Relationship between calf flexibility and PHV (Sever's = orange; CG = purple)

Flexibility therefore in both calf and quadriceps muscles, was significantly different between the AS group and CG and thus the null hypothesis H_{02} is rejected.

4.4.7 Academy Anthropometrics

4.4.7.1 Stature

Players throughout the academy were at the taller end of the age group normal percentiles for height (CDC 2002) with the control group (CG) having a median of 91% and the AS group 95% (N=220). The difference between the AS and control groups was not significant (Mann-Whitney U; p=0.17). Median scores were taken over each year from each team to give an overall year median and range (Table 14). Heights varied considerably within a team with ranges of up to 40 cm, so distribution was non-normal, hence medians were used for comparisons.

Table 14. Academy age control group heights (medians and range)

	Age Group Heights (metres)							
Teams	8	9	10	11	12	13	14	15
A	1.351 (1.26 -1.45)							
B	1.350 (1.25 -1.49)	1.412 (1.30 -1.54)						
C	1.382 (1.26 -1.51)	1.416 (1.29 -1.52)	1.483 (1.34- 1.64)					
D		1.412 (1.34-1.46)	1.466 (1.38-1.56)	1.565 (1.43-1.67)				
E			1.477 (1.32-1.62)	1.561 (1.34-1.73)	1.655 (1.42-1.82)	1.700 (1.48-1.78)		
F			1.454 (1.30-1.55)	1.52 (1.33-1.66)	1.605 (1.43-1.83)	1.692 (1.43-1.83)		
G				1.538 (1.45-1.67)	1.62 (1.48-1.78)	1.704 (1.53-1.88)	1.791 (1.60-1.91)	
H					1.62 (1.56-1.75)	1.727 (1.58-1.86)	1.804 (1.66-190)	1.807 (1.65-1.94)
I						1.725 (1.55-1.80)	1.787 (1.60-1.96)	1.813 (1.68-1.98)

Comparisons of the same age groups showed a non-normal distribution and there was similarity across the age groups with a small range for differences between team medians at each age of less than 3 cm although there was a slightly higher range for the AS group of up to 8 cm at onset. The differences in height between the CG and AS group at each age were not significant (Mann-Whitney U; P = 0.481). (Table 15)

Table 15. Comparison of median stature and range at each age of CG and AS onset.

Age group	Control Group	AS onset group
8	1.35 (1.25-1.51)	
9	1.41 (1.29-1.54)	1.46 (1.45-1.48)
10	1.47 (1.30-1.64)	1.48 (1.45-1.53)
11	1.55 (1.33-1.73)	1.58 (1.53-1.64)
12	1.62 (1.42-1.83)	1.63 (1.48-1.74)
13	1.70 (1.43-1.88)	1.70 (1.62-1.81)
14	1.79 (1.60-1.96)	1.77 (1.74-1.79)
15	1.81 (1.65-1.98)	

4.4.7.2 Mass

Median mass (kgs) was recorded for each team per year (see Table 16) throughout the academy. There was a non-normal distribution for teams. Medians were around the 90% percentile mark of normal population data (CDC 2002) for both the CG and AS group for all ages but dipped to 75% percentile for the 12-year-olds.

Table 16. Academy age control group team mass (median and range)

	Age Group Mass (kgs)							
Teams	8	9	10	11	12	13	14	15
A	30.44 (23-40)							
B	31.4 (21-41)	36.4 (29-47)						
C	33.3 (24-45)	35.8 (28-42)	41.9 (30-51)					
D		36.7 (31-51)	41 (33-52)	48 (36-53)				
E			40.8 (29-52)	47.5 (26-53)	54 (34-59)	56 (40-72)		
F			42 (32-54)	48.5 (30-61)	47.3 (32-67)	58 (36-73)		
G				47.7 (33-65)	46.8 (37-68)	53.0 (38-71)	60.7 (42-75)	
H					50 (33-68)	54.4 (39-72)	63 (44-76)	70 (56-75)
I						61.5 (42-82)	66 (48-79)	73 (55-80)

There was no significant difference in mass between the CG and AS group at any age (Mann-Whitney U P=0.976), although there was one outlier in the AS age 12 group who was 30 kgs heavier than his peers, and as such markedly deviated from the rest of his peers, at the extreme tail of the distribution set and his data was excluded as it was a single construct outlier (Aguinis et al 2013). The age group distribution was normal and the overall mean and standard deviation for each CG age group was calculated and compared with those of the AS group. The greatest variations were found in the 12 to 13-year age CG and 11 to 13-year age AS group (Table 17)

Table 17. Comparison of mass at each age group of AS onset and CG (mean and standard deviation).

	Age Group mass (kgs)							
	8	9	10	11	12	13	14	15
CG mean + SD (kgs)	32.35 (1.46)	36.30 (0.46)	41.43 (0.61)	47.93 (0.43)	49.53 (3.30)	56.74 (3.85)	63.23 (2.66)	71.50 (2.12)
AS mean + SD (kgs)		37.60 (2.48)	41.70 (4.85)	44.00 (5.23)	48.00 (6.59)	59.10 (7.61)	64.30 (0.14)	

4.4.7.3 Body mass index (BMI)

BMI was calculated using the standard formula (Mass / Height ^{2}). Throughout the academy BMI for the CG was within a range of 14-24%, and AS were similar with a range from 16-24%, both with normal distributions There were no statistically significant differences between age groups in the CG nor between them and the AS group.

4.4.7.4 Segmental bone lengths

Length measurements were initially taken for 80 players' thigh, shank and foot heights on both legs of players. Unfortunately, despite additional training of personnel, these measures were found to be routinely inaccurately measured with standard deviation of ± 5.0 cm and standard error of the mean of greater than 0.56 cm (SEM = SD/√N; = 5/√80). For samples of more than 10 subjects the 95% confidence interval (C.I) extends two SEMs in either direction, therefore the SEM = ± 1.12 cm in either direction which was deemed by the researcher and supervisors as too large a margin of error for accurate data analysis. Indeed, some segmental bone lengths were deemed to have shortened during growth, and it was decided to stop further measures for the duration of this study. Measures were only therefore taken for the first year of study and no further analysis has been performed.

4.4.7.5 Girth measures

Circumferential measures were also initially taken for 80 players at mid-thigh and mid-calf to gauge muscular development and changes. Again, there were problems of inaccuracy of measures with standard deviation of ± 3.78 cm and standard error of the mean of 0.42 cm thereby extending two SEMs in either direction (C.I. 95%) giving rise to ± 0.84 cm error margins which were deemed by the researcher and supervisors as too large for accurate data analysis, and these measures were also stopped after the first season.

4.4.8 AS treatment response

Finally, the researcher's treatment protocol as previously described in Chapters One and Two, was implemented for all AS patients as soon as they were diagnosed by the academy medical staff. The response for all AS patients was a pain free return to training in a mean of 20.5 days ± 8.9, and therefore the null hypothesis H_{03} is rejected as patients recovered quicker than expected when compared to the literature.

4.4.9 Summary of key results:

- Over a five-year period from an academy population of 220 players, 31 players developed an AS
- AS occurred close to or at PHV, although the Sever's group were slightly earlier
- PHV was above normal population values on 0.86 cm/month in both groups with a difference in AS and CG rates of 1.05 and 0.95 cm/month respectively
- Flexibility decreased in the quadriceps from the ages of 9-13 and calf from 8-11 in the CG.
- Flexibility reduction was greater in the AS group in the year prior to PHV when compared with the CG
- Strong negative correlation between muscle flexibility and PHV in both groups but stronger in AS.
- Academy players are in the upper 10th percentile of their age group for stature
- Academy AS responded to the treatment protocol with similar results to those described in Chapter One and Two.

4.5 Discussion

4.5.1 Academy profile

A total of 220 players were monitored but the numbers per team were not consistent. The nature of an elite football academy is such that players are constantly under pressure to succeed and underperforming players are 'released' and replaced with more promising players. Therefore, whilst any team would have a maximum of 20 players at a given time, the individuals within that team could vary. Some teams had a turnover of up to 50% e.g. Team E had 30 players involved during the 4 seasons, and overall 40 players were released and replaced (22%).

4.5.2 AS profile

There were 31 players who developed an AS during the study over 4 seasons from 2011 to 2015. Whilst this shows an incident rate of 14% over the whole

academy aged from 8 -16 years of age, when assessed within teams the incidence was as high as 28%, and 3 teams had more than 20% of their squad having suffered from an AS during the period of this study. Specific age groups of 12-14 years accounted for 74% of cases, with another 22% occurring at age 10, therefore a closer analysis of incidence with respect to specific ages gives a more accurate presentation of prevalence within an academy. This could have a serious impact on player availability, and individual and team performances over these years as around 1/5th of a team was affected in the target age groups from ages 10-14 years, which agrees with Read et al (2018b) who found the highest incidence of injuries in the Under-13 age group. However, the overall incident rate is lower than that described in football academies of up to 23% (Price et al 2004; Reece 2012) or 21% (Kvist et al 1984) and 22% (Micheli and Fehlandt 1996) from sports paediatric clinics. The rate of patients decreased during the study from an initial 11 new patients in 2012-13 season down to 4 new patients in 2015-16, and the possible reasons for this will be discussed in the next Chapter 4, but this may have impacted on a lower incident rate than described by other authors above. This is contrary to the latest finding by Read et al (2018b) which shows a three-fold increase in injuries in academies since the inception of the higher training volumes recommended by the EPPP, although this study was not specifically focused on AS.

OSD was the most common condition found in 56% of the AS group, and 26% had Sever's disease, and these ratios are consistent with that found clinically by the researcher in Chapter 1 and Chapter 2, as well as the wider literature (Kvist et al 1984; Dalton 1992; de Inocencio 1998; Stracclioni et al 2007; Caine et al 2008). The small number of SLJ and AIIS (3 each) represented 9% each is slightly higher than that described in the literature where a range of 1% (Le Gall 2006) up to 7% (Orava & Virtanen 1982) has been reported. Sampling differences may account for the range of incident rates reported with some authors selecting from football academies (Le Gall 2006), paediatric clinics (Orava & Virtenen 1982; Micheli & Fehlandt 1992; Lau 2008), or schools (Barber Foss et al 2014), and some selecting from within pathological categories e.g. % AIIS patients from a larger AS subset (Orava & Virtanen 1982; Micheli & Fehlandt 1992) or from all overuse lower limb injuries (Lau

2008) or from all athletes (Barber Foss et al 2014). This makes direct comparisons difficult, and to determine the true incidence rates, but it does appear that both SLJ and AIIS are less common than OSD and Severs (Micheli 1987), as found in this study. There were two patients who had double pathologies suffering from both OSD and SLJ, and OSD and Sever's, but not necessarily at the same time. Double pathologies have been described in the literature (Kujala et al, 1985; Kvist & Heinonen 1991), and recurrence of these syndromes is common (Antich & Brewster, 1985; Flowers and Bhadreshwar, 1995; Bloom et al, 2004; Gholve et al, 2007; Kaya et al, 2013), although this study only had one recurrence reported.

The age of onset was a mean of 12.1 years old ±1.6 which was similar to other studies with sports-specific populations of 12.5 years old in figure skaters (Dubravcic-Simunjak et al 2003), but younger than more general populations found in an orthopaedic clinic with AS patients in the range of 13-16 years age (Osgood 1903) and a 14-15 years old range within a sports paediatric clinic (Micheli & Fehlandt, 1992), and more recently a school population with a range of 12-15 years old for AS onset (de Lucena 2011). Between the different AS there was no statistically significant difference in age for onset but there was a trend towards a lower age for Sever's (10.99 ± 1.57 years old) with no incidences of this condition later than age 13. It could be therefore that Sever's in a football academy may occur in a younger cohort as described in the literature as 12.3 years old (Micheli & Fehlandt 1992) or more recently reported at a mean of 10.8 years old (James 2013), but that the numbers in this study were too small for statistical significance. Further statistical analysis may be warranted. It was further noted that 74% of AS onset was between 12-14 age range and 20% at age 10 which may have been skewed from the higher incidence of Sever's disease. This indicates that there could be specific periods of heightened risk for AS occurrence that academies should be aware of during adolescent growth, and agrees with other authors (Micheli 1983; Dalton 1992; Hodson 1999; Read et al 2018)

4.5.3 Peak Height Velocity (PHV)

It is very difficult to compare growth parameters across a large group of individuals growing at different times, rates and over many years, therefore

taking a single common factor such as the peak of an individual's height growth allows for some comparisons to be made (Buckler 1990; Mirwald et al 2002). The use of PHV as the main non-invasive anthropometric marker is regarded as the industry standard when dealing with adolescent growth (Tanner 1966; Buckler 1990; Bayli and Way 2005; Mirwald et al 2002; Malina et al 2003). Other accurate measures of maturity such as Tanner staging using sexual organ development, are inherently more difficult to perform accurately and without major ethical considerations. The only other non-invasive consistent measure of maturity is menarche, which only occurs in girls. Therefore, the use of PHV can be a valuable marker for comparison of variables across a population (Mirwald et al 2002), and particularly in boys.

There are however some accuracy problems associated with catching a moment in time when the child reaches their absolute peak. One method used is to estimate years to PHV by using an algorithm based on a child's date of birth and using height, leg and trunk lengths (Mirwald et al 2002). Unfortunately, the researcher did not have access to the full records of birth dates from the academy and so the 'Mirwald method' was unable to be used. The other method which is possible in a longitudinal study is to measure standing heights over time to record actual changes with respect to time scales. Whilst this would not capture the peak moment for all players in the academy it was hoped that over 5 years enough players would peak to allow for comparisons to be made. Measuring only once a year could miss a peak which occurs over a 6 to 12-month period, and therefore could count the 6 months before and after a peak, missing the peak altogether. By measuring height more frequently, more data sets would allow for more accurate estimations of peak and monitoring of growth, but not having so many measures that would irritate the coaches and the players (Bayli & Way 2005). On average 4 sets of data were recorded for each team per annum which allowed for greater likelihood in recording growth as the individual neared their peak and therefore greater accuracy in estimating that peak.

The period of PHV was identified by the greatest rate of growth preceding a reduction in growth rate and was then recorded as the estimated PHV. 73 players from the CG (38%) and 30 of the AS group (97%) passed through their

PHV during this study's time frame and the amount was recorded retrospectively, as well as their ages. The median ages of the CG and AS group at PHV are not significantly different, with both groups reaching PHV at similar ages CG 13.0 years ± 0.98 and AS 12.4 ± 1.08 (P=0.139) but there was a small trend towards the AS group being slightly younger. The difference in distribution however is significant (Mann-Whitney U p <0.001) with the AS group more normally distributed and the CG more skewed towards the right and later development. However, both groups appear to be peaking at a younger age than is normally described (Tanner et al 1965). This may indicate earlier maturation as an academy group compared to their school peers, or it may be following the trend for earlier maturation generally in the current Western world (Karlberg 2002), but without contemporary comparisons with a local age-matched schoolboy population, this cannot be determined, and was beyond the remit of this study.

The heights at PHV showed little difference between the groups (CG 1.646m ± 0.073, AS 1.637m ± 0.088; P= 0.62), but the differences in actual velocities at peak are statistically significant between the groups with the CG PHV of 0.94 cm/month ± 0.08 compared to AS 1.05 cm/month ± 0.05 (p<0.001). This also compares with the current population norms of between 0.683 to 0.858 cm/month (Malina et al 2015). Growth for the preceding year also showed statistically significant differences of 10.15 cm for the AS group vs 9.54 cm for the CG. However, although these differences of I mm/ month and 6 mm/year are too close for practical use in the real-world setting, the trend should be reported and acknowledged as being of interest. Academy players are predominantly at the highest end of height growth rates, with the AS slightly higher still. Within the sub-groups of the AS there are also significant differences in PHV medians and distributions when compared with the CG (Kruskal-Wallis p< 0.001). Height velocity was also recorded in the AS group at the onset of their condition as well as their peaks. Differences between the AS onset height velocities (0.96 cm/month ± 0.09) were also significant from the CG at their peaks (p=0.006) which indicates that the AS group at onset had already exceeded the rate of the CG at their peaks. Again, this significance also occurred across the sub-groups (Kruskall-Wallis p<0.001). This could be an important finding as it suggests that the AS group at onset are already

growing at a rate which is significantly higher than the peak of their team mates, as well as achieving the highest absolute values at peak. This could give rise to identifying a marker of growth rate which may allow academy staff to monitor this rapid growth group more closely with the aim of possibly preventing occurrence of AS in the first place. However, there were 10 players in the CG whose PHV was estimated at more than 1.00 cm/month that didn't get AS, therefore growth rate of itself is unlikely to be the only factor in its development.

These findings place the AS group in the 97th percentile for PHV with the CG still around a high of the 91st percentile, and both with an earlier maturation age than normal. The 50th percentile expected PHV growth in a middle maturating boy is 0.79 cm/month at age 13.5 years (Tanner et al 1965). This study's PHV group median values have been superimposed on a graph below of PHV values found by Tanner, Whitehouse and Takaishi's study (1965) to show an overview of their positioning with respect to age means (Figure 43). Caution should be taken to compare these values too closely however as this original study and its findings were presented over 50 years ago and it has been observed that earlier maturation is now more common (Karlberg 2002). Whether the rate of growth has also increased with modern lifestyles and improved diets, has not been identified as yet to the researcher's knowledge.

The growth characteristics between the AS group and CG showed small but significant differences. However, it is acknowledged that these differences could be too small for meaningful application in the real-world setting, and therefore the null hypothesis H_{01} is accepted.

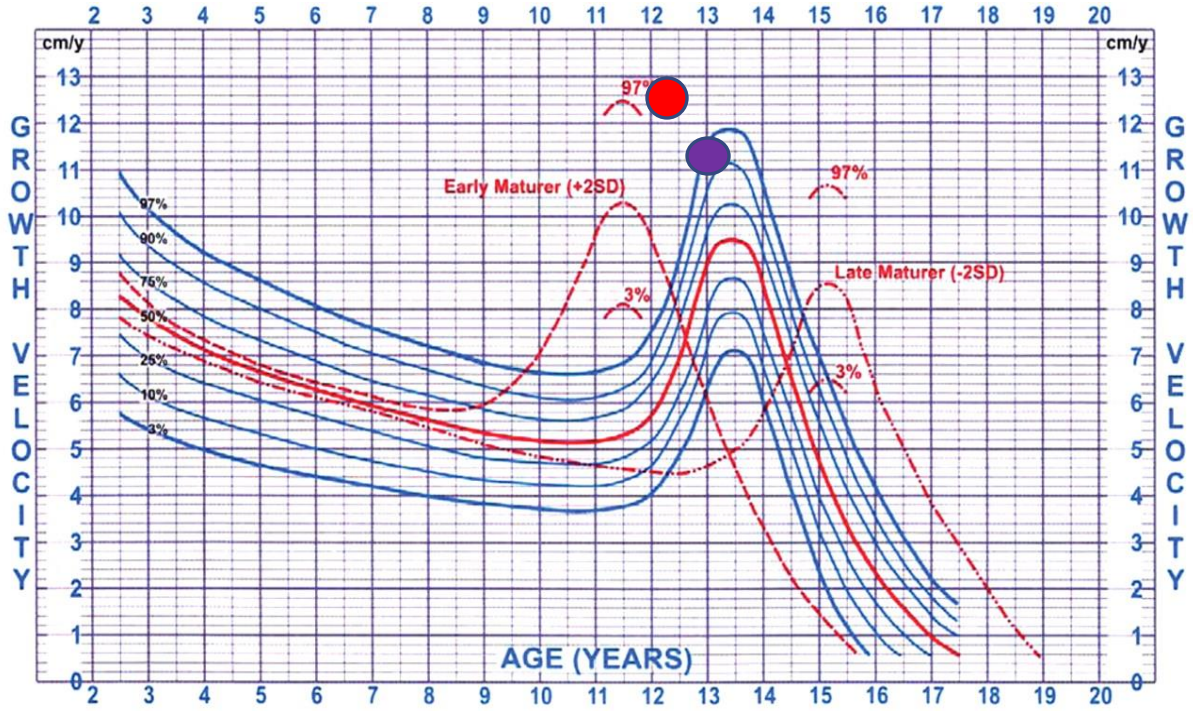


Figure 43. PHV maturation chart for boys (Tanner, Whitehouse and Takaishi 1965), with Crystal Palace Academy group medians superimposed. (AS = red; CG = purple)

4.5.4 AS onset and PHV

It is important to capture data at the point of onset of AS for players, so see if there are any parameters that may have an influence on the occurrence of that condition or not. Therefore, measures at onset were also recorded with respect to velocity growth rate at the point of onset. In 50% of cases the AS onset coincided with the players' PHV, but there were 33% of players who's AS occurred prior to their PHV and 17% after, therefore 83% of AS onset occurred before or coincided with their time of PHV. The AS onsets of this group occur close to their PHV with a range of ± 8 month, which could give rise to recognising a period of risk for AS onset that has not been previously identified or described. It is also noted that the velocities recorded at onset were also significantly faster rates than the CG (Independent median test P=0.006; and Kruskall-Wallis P=0.000). Therefore, it could be concluded that the AS group are growing at significantly faster rates than their counterparts (CG) although final heights and ages at PHV are not significantly different.

Further analysis within the two largest AS sub-groups shows that the Sever's patients do appear to have their onset significantly earlier into their growth spurt, about 6 months prior to their PHV compared to OSD (Independent median test P=0.028), and differences in distributions are also highly significant (Mann-Whitney U P=0.004) with the OSD more closely associated with the time of PHV. The latter is also true for the two smallest sub-groups of SLJ and AIIS with both sets having their onset at PHV.

4.5.5 Flexibility

Flexibility was recorded bilaterally in the gastrocnemius and quadriceps muscles throughout the study, and team means for each age were recorded. It was decided to concentrate on the dominant legs for players for this study's analysis due to the quantity of data. These dominant leg means were further collated to represent each age group comprising of between 3 to 4 teams' data and means calculated. There is a change demonstrated throughout the academy age groups during the study, with a difference of 17˚ in the gastrocnemius muscle from the lowest to highest mean for age, with a low (33˚) at age 11 and high (15˚) at age 15 (Figure 3.18). Soleus muscle recordings were also attempted at the outset of the study but with growing time constraints it was decided to record only the gastrocnemius muscle to represent the calf, as this was the muscle that would most reflect the ballistic nature of physical activity of jumping and sprinting in the football player, and therefore the most relevant muscle to monitor (Reilly and Williams 2003). It has also been found that gastrocnemius measures are a more reliable indicator of calf tightness than soleus (Becerro-de-Bengoa-Vallejo et al 2014).

Quadriceps muscles also show a reduction in flexibility across the age groups with a drop of 11˚ from the lowest at age 13 (51˚) from the highest at age 9 (62˚) closely followed by age 15 (61˚), but these could be affected by the 3˚ measurement error range and therefore absolute values may not be accurate, but the trend is more of note This contrasts with the findings of Feldman et al (1999) who did not find any growth-related changes in flexibility but this study's population mean age of boys was 14.2 years and were likely to be post-PHV and so recorded at a time of decelerating growth rates, and they were also only

measured annually. The findings of this study do concur with observations from other authors (Kendall et al 2005; de Lucena et al 2011) and specifically Philippaerts et al (2006) who found an overall drop in flexibility at PHV in footballers and with Malina et al (2004) who also described a drop in flexibility prior to PHV.

Moreover, the age for the tightest calf muscle (age 11) corresponds to that of the onset of Sever's disease (age 10.99 years old) and is very close (age 13) for OSD/SLJ/AIIS (12.34/ 13.00/13.19 years of age respectively). It appears from this study that there is a period around age 11 years where calf muscle flexibility decreases in the academy generally and then recovers and improves, and quadriceps muscle also decreases flexibility from age 9 to a low at age 13, and again recovers.

It is important to state at this stage of the discussion that an intervention did take place with respect to flexibility decreasing below a threshold in either the calf or quadriceps. The data above therefore reflects only those players who did not have any intervention and did not develop an AS (N = 55 for the quadriceps measures and N=56 for the calf measures). The intervention targeted players whose flexibility dropped below a threshold of 45˚ for the quadriceps and more than 38˚ for the calf. These players were prescribed a specific stretching programme to improve their flexibility and told the importance of performing them regularly every day (St. George 1989). The results of this intervention will be discussed in the following chapter.

4.5.5.1 Quadriceps

Comparing the quadriceps flexibility at onset for the AS sub-group (OSD, SLJ and AIIS) and the remaining CG at PHV shows a highly significant difference between the groups (T-test (2-tailed) P<0.000) with the AS group having a mean of 36.3˚ compared to the CG mean of 52.6˚. It could be argued that this could be because of contracting the AS rather than a possible cause, but tracing back to one year's data prior to AS onset and CG PHV shows an overall tighter AS group (by around 5˚) with a sudden drop in the measures before their onset from 47˚ down to 35˚, compared with the CG of 56˚ (a 9˚ difference). These players therefore were missed by the proposed intervention threshold of 45˚. Over the preceding year the AS group progressively dropped by a mean

of 20.5˚ and the CG only 7.7˚. The quadriceps flexibility for the CG is at its lowest value for 87% of players either prior to or at their PHV moment, so a direct comparison of these groups is justified. Therefore, this marked drop in quadriceps flexibility could identify an early risk factor for the sub-group AS involving OSD, SLJ and AIIS, and the threshold for intervention may need to be raised. It appears that the period prior to PHV shows a reduction in quadriceps flexibility in all players and this should be noted and monitored by academy staff. These findings concur with the studies by Eiichi (2001), Ikeda et al (2001), and de Lucena et al (2011) where all authors found increased quadriceps tightness in their OSD patients compared to their peer group.

4.5.5.2 Calf

Calf flexibility also shows a reduction in the period prior to onset for the Sever's sub-group with a highly significant difference when compared with the CG at PHV (T-test (2 tailed) P<0.000). It should be noted that there was a difference in timing with the Sever's onset a mean of 6 months prior to PHV, and patients with Sever's were then put on a treatment intervention of massage and stretching so that by the time of their own PHV values had normalised closer to their CG cohort and at this point there was no difference between the groups. At the point of onset, the Sever's group has a mean calf flexibility of 45˚± 4, but again it is not possible to determine if this is a possible cause or just effect from this single set of data. However, tracking back to the preceding year's data just prior to the Sever's onset shows a highly significant difference in calf tightness between the groups (Independent samples t-test (2-tailed) P<0.001) with the CG having a mean value of 31˚ and the Sever's group 36˚. Unfortunately, this was below the threshold set and these patients were missed for the intervention. The data shows that there is a period leading up to PHV of about 6-12 months where calf flexibility decreases a little for most players (mean of 3˚ of the year) but much more in the Sever's group (mean of 17˚) and this difference is highly significant (Independent T-test (2-tailed) P<0.001). The measurement error of 3˚ needs to be taken into account, especially for the CG so there may be a small change in calf flexibility leading up to PHV in the CG but a more marked change in the Sever's group, with their flexibility restored after treatment. These findings agree with other authors' observations that calf

tightness is observed in Sever's patients (Szames et al 1990; Madden and Mellion 1996; Becerro-de-Bengoa-Vallejo 2014). This study provides a starting point for academy staff to be aware of in the months prior to PHV where players caves get tighter and passing a threshold may increase the likelihood of developing Sever's disease. This flexibility threshold may need to be lowered further to catch all potential at risk players.

Flexibility was significantly different between the CG and AS groups with the AS group having tighter quadriceps and calf muscles prior to developing their AS, therefore the null hypothesis H_{02} is rejected. Understanding this difference may have opened an opportunity to further explore flexibility as a risk factor in the development of AS and could lead to its identification as a possible aetiological factor.

4.5.6 Flexibility and PHV

4.5.6.1 Quadriceps and PHV

Having found a significant difference between the groups in terms of PHV rate and muscle flexibility, it is useful to determine if there is any relationship between these factors. Exploring the relationship of PHV and quadriceps flexibility in the CG and AS sub-group (OSD, SLJ and AIIS), a very strong negative correlation is found in both the CG (r=0.834) and the AS group (r =0.873), with increasing quadriceps tightness found the faster the growth rate. The AS group have a slightly stronger association but both values of PHV and reduced flexibility are higher in this group as previously described. Therefore, it appears that the faster the growth rate, the tighter the quadriceps muscle becomes, with patients having even stronger relationships. This is the first time this association has been described and it may provide an insight into possible risk factors behind those who appear to get an AS and those who don't. The relationship between PHV and flexibility shows that the faster the growth rate the tighter the quadriceps, but if the flexibility stays below the threshold, these fast growth individuals do not develop an AS. This may be an important finding in this study and give rise to further exploration in the next chapter. The researcher is not aware of any studies that have been published to date on the relationship between growth rate and flexibility.

4.5.6.2 Calf and PHV

Similarly, there was a very strong correlation found between peak growth rate and calf flexibility with the CG having a correlation of r =0.785 and the Sever's sub-group having an even stronger correlation of r =0.8782, and with greater values particularly in calf tightness. There was one Sever's patient whose PHV was less than 1 cm/ month, but all the others were greater. Again, the data shows that the faster the growth rate at PHV, the tighter the calf muscles became, but that it seems the passive tension is the most important factor.

Together there is a clear and strong association with growth rate and a reduction in muscle flexibility in both the CG and AS group, but it cannot be proven that one causes the other at this stage – only that there is a strong correlation. It may be that passive tension in the muscle is attributable to rapid bone lengthening and further study into cause and effect is warranted. Academy staff should be made aware of this association as a possible risk factor for AS.

Considering that the academy players are a special population who appear to have been partly selected on the basis of physical maturity (Malina 2003; Philippaerts et al 2006; Hirose 2009; Ostojic et al 2014), with earlier maturation and therefore faster PHV, it could be that selection bias creates more potential for AS to occur. From this study, a faster growth rate as measured by PHV shows a strong relationship with tighter muscles, and earlier maturing athletes have the fastest PHV rates (Tanner & Davies 1985; Beunen and Malina 1988; Buckler 1990; Iulaino-Burns et al 2001; Wright et al 2002; Malina 2004), therefore these earlier maturing footballers may be more prone to AS, and therefore academies may be unwittingly selecting more at-risk players. There may be a case to present the development of bio-banding competitions where players of similar size and maturity compete, rather than the wide variations of size seen in chronological age groups (Buckler 1990; Vaeyens et al 2006). Bio-banding may also encourage academies to select players based on talent and skill, rather than physical maturity, which might improve the skill levels of the club academy as a whole. It also may translate into more successful academy graduations into senior levels (Ostojic et al 2014).

4.5.7 Anthropometrics

4.5.7.1 Height, Mass and BMI

The academy players are predominantly in the tallest 10 % in height and mass for their ages (Tanner & Davies, 1985; Buckler 1990; CDC 2002), and none are below the 50th percentile. It has been reported by other authors that advanced stature is a common feature of academy football players (Malina 2003; Philippaerts et al 2006; Hirose 2009) but it is surprising to find such a marked taller profile compared to the normal population. This may reflect a selection bias by the academy coaches and scouts, favouring taller players and/or earlier maturation (Malina et al 2003), but whether this approach is deliberate or accidental is unknown. This academy cohort's height profile therefore, is not reflective of the normal population and thus awareness of this as a special population should be noted. The slight drop in the mass medians from the 90th to the 75th percentile during the 12-year age group may be reflective on the latency of peak mass velocity occurring 6 -12 months later than PHV (Tanner 1965; Buckler 1990; Bayli & Way 2005). The widest range of mass is found amongst the 12-13-year olds, whereas the heights are more similar. BMI for the CG cohort is a healthy athletic range of between 14-24% (CDC 2000; Malina et al 2004).

The AS group have a taller percentile than their CG team players with the median of 95%, but there was not enough difference between the groups for statistical significance. However, this reflects the fact of these academy players being in the tallest percentiles of their peer group, and again this may highlight a bias that the selectors are possibly inadvertently targeting. Nor are there any significant differences in mass or BMI between the groups although slightly larger ranges were found in mass in the 10-13-year age groups in the AS group, and the BMI were similar. Therefore, the heights, mass and BMI throughout the ages are similar between the AS and CG, but differences occurred at the time of PHV which will be discussed later in detail.

Within the AS sub-groups (i.e. pathologies) there are no significant differences in height between OS and Sever's, but some significance is found between Sever's and SLJ and AIIS (P= 0.036), with the SLJ and AIIS players taller on average by around 15 cms. There are also significant differences in mass

(P=0.009) and BMI (P =0.049) across the groups and additionally between Sever's, and SLJ (P=0.018) and AIIS (P = 0.009), so there do appear to be some profile differences between the conditions. Whether these have a bearing on the type of condition that occurs, or its onset is not known from this study but may be worthy of later research.

Mass changes indicate a difference with AS players lighter than the CG at PHV by 5 kgs (Mann-Whitney U P=0.027), but with a large range from 31 to 71 kgs across the groups. Therefore the 5 kgs difference between the groups shows only 7 -15 % variation, and although this was significantly different from a statistical viewpoint, caution should be made in interpreting this finding as adolescent weight has many other influences other than growth, including diet and muscle mass which may be more pertinent (Buckler 1990, Faigenbaum et al 2011; Malina et al, 2004). More specific measures including fat: lean mass ratios would give greater understanding to the changes that occur during adolescence rather than just gross mass or BMI. The peak for mass velocity occurs about 6 months after PHV therefore the measures in this study taken at PHV may not be reflective of the period of greatest change in mass. Further investigation of this could be warranted due to their being a difference found and the effect of mass on load bearing, but as this tends to occur after the onset of almost all of the AS onsets, the researcher decided not to examine the final peak mass velocity of players.

Using the height and mass data affords a calculation of BMI which is routinely used as an indicator for under-weight/ healthy/ over-weight individuals (CDC 2000; Malina et al 2004). However, there is a wide variation during adolescence and therefore it is a less useful comparison tool more useful at the extremes of measure to indicate heaviness in the obese or under-nourished (Malina et al 2004). In this study the BMI for both groups is similar, even though there is a difference in mass. There is a greater influence of height than mass in the calculation of BMI (mass/ height^2), and this may explain why there was no significant difference found in BMI, with a similar range and median found in both groups (medians AS 17.94% vs CG 18.64%). No players are classed as over-weight (<25%) although some authors have felt that BMI is a poorer indicator of healthy weight in athletes due to muscle mass being heavier than

fat (Ode et al 2007), therefore muscular athletes may have a higher BMI than expected. In adolescents only the top 10th percentile is classified as over-weight or obese (CDC 2000) and it would not be expected that young and fit athletes would be obese, but increased muscle mass could shift the BMI's higher. This study did not find this, perhaps as the rapid increase in mass did not occur until after PHV as explained previously. Again, the use of lean: fat mass ratios is a better indicator for health, but this study did not have access to the additional equipment required to carry out this investigation. Further study into more accurate body mass composition could be useful future research into adolescent body composition changes, as athletes tend to have different lean: fat ratios than their sedentary peers and this could give greater insight into the higher incidence of AS amongst athletes (Micheli 1986; Czyrny 2010; De Lucena et al 2011).

Leg length to trunk length ratios also were higher than described for the norms of 93.2% by Iuliano-Burns et al (2001), with both groups having slightly longer legs proportionally at the time of PHV. However, the differences were not large enough to report any significance, therefore leg: trunk ratios may not be an area that needs further exploration at this stage. Malina et al (2004) states (pg 68) that leg length growth precedes the trunk up to PHV then the trunk continues to grow further and for longer once PHV has occurred, and in the end, contributes the most to final adult height. Therefore, leg growth occurs in a smaller time frame than trunk growth, which may impact on both the magnitude of leg bone growth and PHV rate. Mirwald et al (2002) stated these actual differences in mean age peaks at 13.12 years age for leg length growth, overall PHV at 13.45 years old and peak trunk growth at 13.68 years old, therefore there is a narrow window of opportunity to capture these changes and their differences.

4.5.7.2 Segmental bone lengths

Segmental bone lengths of the foot, tibia and femur were taken for the first year of the study but unfortunately proved to be highly inaccurate. Despite additional training and the use of physiotherapy practitioners whose anatomy knowledge and skills were deemed to be well-practiced, there was great inaccuracy and the data could not be relied upon. The same staff were used for the measures

and team players, but despite this consistency, the intra-tester reliability was poor.

Although random errors of measurement are a recognised problem within anthropometry (Buckler 1990; Malina et al 2004), when performed over a large-scale study they tend to cancel themselves out and are ordinarily not a major concern (Malina et al 2004). However, in this study the discrepancies of bone measures were regarded by the researcher and their supervisors as too large to ignore (SD= ± 5 cm; SEM ± 0.56 cm). There was also increasing pressure from coaches to finish the testing sooner to allow players to have a thorough warm-up before their games. Therefore, with the inaccuracy of measures and time constraints it was decided to cease further bone segmental measures for this study. It may be that with better training and more consistent staff this could be an interesting facet of adolescent growth to explore, especially in relationship to the changes prior to PHV and flexibility. It had been hoped to see if there were any comparisons to be made between developing the different AS conditions and specific bone segmental changes, but it was not possible to do so in this study, and this may be an interesting area for future research.

4.5.7.3 Girths

Measurements of thigh and calf circumference were initially taken to monitor changes in relative muscular development (Malina et al 2004). Mid-thigh measurements were used to approximate the development of the quadriceps and hamstring groups, and the calf at its largest circumference was used to approximate the development of the gastrocnemius muscle. Unfortunately, accuracy and consistency became a major problem as it did for segmental bone lengths, with some errors of even recording in the wrong units (tape measures were back to front, so inches were recorded instead of cm) and accurately and consistently identifying the correct positions for measuring. Errors of measures and time constraints meant that the girth measures were also halted after the first year.

One of the benefits of studying academy players is that they should have similar muscular and physical loads within each team, as players do the same training and matches within the academy, so there may not be much variation

in physical activity loads, and therefore possible muscular development. However external activity and sports were not recorded, and these may also influence loading. It may be therefore that girth measurements may not have been as important a factor in the development of AS, however this study is unable to present any evidence for this. Better accuracy of measures could give more insight into the impact of muscle girth and would be a valid area for future research.

Increases in muscular strength could be a factor in the development of muscle tightness (Gossman et al 1982) and therefore monitoring of girth could be a useful measure of both potential active load on the muscle-tendon unit and potential for tightness. With better training and consistency girth measures could be useful for monitoring the development of segmental musculature and thus load on specific apophysis regions. However, as muscle strength tends to peak after PHV (Malina et al 2004) this may not be a factor in the lead up to and development of the AS seen in this study, as these occurred predominantly at or before PHV. In contrast Philippaerts et al (2006) recorded peak muscle development in functional strength tests at PHV, but these were general tests and not muscle specific. There may be an opportunity for future research into specific muscle testing of the quadriceps and calf muscles in the year preceding PHV, to help determine any changes associated with muscular development. The relationship of leg and muscle dominance to injury, in sports such as football, could also be investigated and the additional data collected but not analysed in this study should be explored in the future.

The anthropometrics and growth characteristics were similar between the CG and AS groups with only statistically small differences in PHV, therefore the null hypothesis H01 is accepted.

4.5.8 Response to treatment for AS

31 players were diagnosed with an AS during the period of 5 years of this study. Medical staff were instructed on the new treatment protocol as described in Chapters One and Two of this thesis and all patients received the experimental treatment immediately upon diagnosis. Historical data from the club medical records showed a previous return to sport time scale of between 2 to 12 months

for patient recovery. After implementing the new protocol there was a mean return to sport in 20.52 days ± 8.9, which is very favorable for players and medical and coaching staff. Having patients recover in a shorter time results in less medical treatment and time required, less pain and dysfunction for patients, and better availability of players for the team coaches and selectors. The treatment response times were similar to that reported by the researcher in Chapters One and Two and previous research (Strickland et al 2008; Strickland et al 2010) and are much shorter than that described as the natural history of 21 months for OSD (Ehrenborg 1962) and 7 months for Sever's disease (Agyekum and Ma 2015).

The response to treatment using the researcher's protocol was quicker than is described in the literature and therefore the null hypothesis H_{O3} is rejected. Faster effective recovery from a painful condition should always be a goal for health professionals and therefore this finding reinforces the need to question existing standard advice based on opinion and not on clinical or scientific evidence. Questioning and changing approaches for the benefit of academy players also ties in with the stated aims of the EPPP.

4.5.9 Limitations

Whilst every effort is made to ensure the accuracy and reliability of data gathered, the changes in testing personnel means that this is difficult to achieve. In simplifying the testing to stature, mass and flexibility it was hoped that accuracy and consistency could be improved and maintained throughout the study. It is implicit that personnel are trained adequately to perform the tests with accuracy and reliability, but this study shows that this is not always achievable. Longitudinal studies may need to be revised as they progress in response to observations and clear errors, and refinements to a study can be made in response (Buckler 1990), therefore those tests which produced the most unreliable data were dropped. Further errors in linear measures due to inaccuracy of measurement, incorrect recording or diurnal variation were more apparent and able to be extrapolated from previous and subsequent recordings (Buckler 1990).

Changes in players who were either released or newly selected meant changing subject population, so testing of some individuals provided a shorter time frame and less data sets for study and analysis. All players needed to be measured as it was unknown which players may or may not develop an AS, or when players would achieve their PHV, therefore only 47% of the academy players were involved in this specific analysis.

Issues also occurred due to testing date changes from poor weather and cancellations, to longer periods of non-measures over the summer recess, so regularity between tests was not always maintained. Never-the-less more measures were taken than has been previously described by some authors (Buckler 1990; Dimeglio 2001) and closer to the 4 sets per year recommended by Tanner (1962) and Balyi and Way (2005), which meant more data points were collected and it is hoped that this would increase the accuracy of the estimates for growth in particular. Location and time of testing was largely consistent with 72 of the tests performed on a Sunday morning at the Crystal Palace training ground, and 20 tests performed at the Crystal Palace National Sports Centre at the pre-season evaluation one September evening. Weather was not an issue as all testing occurred indoors, but temperatures may have been different during the seasonal changes and this may have impacted on measures such as flexibility. Ideally all tests would have been carried out at the same time and location, however consistent equipment was used at both venues, as well as testing prior to activity. There are inherent difficulties in controlling variables in a longitudinal study, but these were kept as constant as possible.

One of the main difficulties was with the real-world pressures of performing tests prior to football training and matches. Coaches felt they should have priority over players' time and were impatient of the time taken to perform tests accurately. This pressure of trying to perform the tests as quickly as possible may have impacted on the quality and reliability of the data, as well as unsettling the players who also wanted to get out onto the pitch as quickly as possible. However, this is the reality of testing in the real world of sports academies and working with children, and compromises may need to be made to keep all parties compliant and voluntarily involved.

Whilst stature and mass measures can give an indication as to overall body size, individuals can vary in proportion, shape and dimension. At the time of greatest change during puberty, these anthropometric parameters are also likely to change the most. Therefore, isolating the point in time for the greatest growth (PHV) will differ for each individual, making it possible to only record this retrospectively. The use of PHV as a central marker for growth is consistent with the literature (Tanner 1962, Buckler 1990, Balyi and Way 2009; Malina et al 2015) but may not have been as relevant for those with developed Sever's disease and in the calf flexibility changes as these tended to occur in advance of PHV. Allowance for the difference in time scales meant that measures taken in the year prior to PHV were used for additional comparisons but the timings between these data sets was not consistent, therefore direct comparisons may not be as accurate as reported.

Overall, caution should be used in interpretation of the findings from this study due to less than ideal standards of accuracy and reliability, thus the trustworthiness of some of the data sets is acknowledged as a limitation and could reflect on the accuracy of the statistical findings and interpretations. However, this does reflect the common problems associated with collecting real life data on living (and moving) beings and has been reported in most other anthropometric studies (Tanner 1962, Buckler 1990, Ross and Marfell-Jones 1991; Malina et al 2004; Balyi et al 2013; Malina et al 2015). Wider application of the findings to the general population may not necessarily be valid due to the special selected nature of the academy cohort sample.

4.5.10 Summary

The CPFC academy presents a specialised sports cohort that has a higher incidence of AS than found in the general adolescent population. 31 players developed an AS during the study giving an academy incidence of 14%, but individual team incidence of up to 27% which would have a large impact on the selection and possible performance of the squad. OSD represented the largest sub-group of AS with 57%. 50% of the AS group had their onset at or close to their PHV, with the others within 6 months either side of this marker, with the Sever's group onset a mean of 24 weeks prior to PHV.

But amongst the most important findings was that flexibility in the quadriceps and calf muscles decreased generally prior to PHV across the academy but those who developed AS had significantly greater passive tension which breached the designated flexibility thresholds. These drops in flexibility in the AS group over the year prior to onset were over 20˚ in the quadriceps for the OSD, SLJ and AIIS sub-group; and 21˚ in the calf for the Sever's sub-group. These drops in flexibility may be an important early indicator for identifying at risk players, and thus addressing passive tension before it becomes a problem. This served the basis of the next chapter on prevention.

When looking at PHV the difference in rate of height velocity, although it was statistically significant, the AS group achieving median rates of 1.05 cm/month at peak compared with 0.95 cm/month with their CG and 0.79 cm/month for the norm 50th percentile middle maturing boy (Tanner et al 1965), the difference was between our cohort groups was only 1 mm, therefore this may not be reliable and useful for applied work . Height velocities at AS onset were also significantly higher than the CG at 0.96 cm/month and these were mainly prior to or at PHV, indicating the acceleration phase of growth was significantly greater for the AS group. It might be prudent for academy staff to closely monitor players growing at more than 1 cm/ month and reduce their volume of training as suggested by Read et al (2016) and reinforce the importance of good flexibility during the key stages of growth.

Assessing the relationship between these two factors (height velocity and flexibility) identifies a strong correlation, with the faster the height velocity the worse the flexibility. This relationship was even stronger and had higher values in the AS group. Although it cannot be proven that faster growth rate causes increased passive tension in the muscle, the exact relationship deserves greater investigation. This strong correlation might be able to help identify at risk players by monitoring those with the fastest growth rates and making sure these players maintain optimal flexibility. Both factors are easy to measure in the academy setting and provide academy staff with simple data sets to explore preventative strategies. Academy staff should also be aware of the selection of earlier maturing boys may inadvertently make their cohort more at risk to growth related injuries.

Academy players are on average within the tallest 10% for their age groups and those who developed AS were amongst the tallest 3% of these. The academy players also were earlier in maturation compared to normal age-matched data (CDC 2002), and the AS group also slightly younger than the CG, although not significantly so. The AS group responded well to the researcher's treatment protocol and recovered in a mean of 21 days to return to their football.

In identifying differences in flexibility factors that are apparent between the AS and CG, and the timing of AS onset aligned to PHV, it is hoped that academy staff may be able to proactively reduce the development of and incidence of AS in young athletes.

4.6. Conclusions

There is a small difference in peak height velocity but significant difference in quadriceps and calf muscle flexibility between the academy control cohort and those who developed an AS, with the AS group having slightly more rapid growth, but significantly reduced flexibility than their counterparts.

There is also a strong correlation between the rate of growth at PHV and flexibility reduction in the quadriceps and calf muscles in academy footballers, and stronger correlations are found in AS patients. Therefore, rapid growth rates and decreasing flexibility appear to be associated with the development of AS and academy staff and others involved with youth sports should be aware of this.

There appears to be a window of presentation close to the player's PHV where AS seem to occur, especially in the acceleration phase. This could give forewarning to academy staff that as a player approaches their PHV there should be closer monitoring of and maintaining muscle flexibility, especially in the target muscles involving AS and greater awareness of height velocity rates.

4.6.1 Link to next Chapter

The opportunity may present during this critical growth period in adolescence where flexibility deficits could be reversed by implementing a stretching programme for ‘at risk’ players whose flexibility drops below a given threshold. This would remove one identified risk factor from the suggested aetiology of AS and might provide the basis for a prevention strategy.

CHAPTER 5. Epidemiological Approach: Prevention

Study 4: An experimental study into the effects of a stretching intervention on Apophysitis Syndrome (AS) incidence rates in academy male footballers.

5.1 Abstract

AS are the most common overuse injuries found in adolescent athletes yet there have been no studies into developing a preventative strategy. This study aims to demonstrate one approach within the Crystal Palace (CP) academy football setting, identifying reduced flexibility in subjects and intervening with a stretching programme (IG) and compare the results with a matched group of subjects who developed an AS, and their control cohort (CG). Comparisons are made between different football clubs for analysis of subsequent incidence rates.

Method: Football academies from 3 clubs volunteered to take part, involving over 600 players from the age of 8-16 years. Incident rates and profiles of AS patients were recorded over 4 seasons as well as flexibility of the quadriceps and calf muscles in all players. Players were measured at least 3 times per year to enable changes in flexibility to be recorded. Descriptive statistics were used for club AS incidence and patient profiles, and flexibility overview from the CP groups. Comparisons between the CP group flexibility changes, and between clubs were performed using One-way ANOVAs, and 2-sample T-tests. For treatment response non-parametric analysis used Independent means samples and Kruskal-Wallis, as the data distribution was not normal. All data used 95% CI at $P<0.05$ for significance.

Results: Within the single academy (N=220), no players from the IG developed an AS during the study. Flexibility was significantly different between the CG and IG groups in both the quadriceps and calf (One-way ANOVA $P<.01$). Inter-club comparisons of quadriceps and calf flexibility were the same (2-sample T-test with equal variance $P =0.08$ and 0.51 respectively) and showed a reduction of flexibility at academies during the ages of 11 to 13 years. There were no differences between clubs in response time to AS treatment using the protocol (Independent- samples means test $p=0.19$), but distribution was significantly different (Kruskal-Wallis $p=0.03$).

Conclusions: Stretching interventions appear to have prevented AS from developing in the CP academy. Flexibility reduced during the secondary growth period at all the academies and together with objective thresholds, provides an opportunity for academy staff to intervene and proactively prevent AS.

5.2 Introduction

Sport transmits enormous benefits to young athletes from improvements in physical fitness, enhanced self-esteem, better social skills and academic performance (Magrini and Dahab, 2016) but injury rates amongst children from sport are on the increase (Brenner 2007). One of the primary roles of sports medicine practitioners and others involved in supervising or coaching sport, is to actively engage in injury prevention (Brukner and Khan 2012). To do this the extent of the problem should be identified and then the factors and mechanisms which play a part in its occurrence should be recognised and could be used to establish a prevention strategy (van Mechelen et al 1992). After prevention measures have been introduced, they need to be evaluated as to their effectiveness by establishing any changes in new incidence rates. As explored in the previous chapters, reduced muscle flexibility has a role in the presentation and treatment of AS patients (Chapters 2 and 3) and this muscle flexibility decreases significantly prior to the onset of AS (Chapter 4) compared to their control cohort. This chapter will discuss the scale of the AS problem with a review of literature on aetiology and risk factors, and the limited research into AS injury prevention. It will then present a study to assess whether proactively addressing reduced flexibility can have an influence on the incident rate of AS within a football academy.

5.2.1 Incidence

There are over 30 million children and adolescents in the USA playing organised sport and over 1/3rd of them will sustain an injury (Adirim and Cheng 2003) and it has been reported that overuse injuries represent 50% of injuries in highly trained athletes (Rejeb et al 2017). In this country Michaleff et al 2017 recorded data from 11 GP surgeries in the UK and found that knee problems were the 4th most common musculo-skeletal disorder reported by children and teenagers and were responsible for the 2nd most frequent visits. These also represented about 10% of a GP's overall childhood MSK workload, so knee problems in children and adolescents represent an important problem in health care in this country. The incidence increased to peak during the 12-15-year age group (42%) with boys slightly outnumbering girls (59:41%). This study also highlighted the different approaches that GPs use to record knee problems with the majority of doctors relying more on the symptomatic descriptions (ie

knee pain) rather than attempting a diagnosis, therefore the actual incidence of knee related AS in the wider population of the UK is unknown. This study agreed with others (Rathleff et al 2013; Witvrouw et al 2014) who also noted the increased reporting during this pubertal time in Denmark and Belgium respectively. Michaleff et al (2017) also noted that many GP's take a 'wait and see' approach to see if the problem resolves naturally or if a clearer diagnosis can be achieved after repeated visits, therefore reporting may also not occur for some time after the original visit. This approach could be down to lack of specialist sports medicine knowledge (Michaleff et al 2017) or a lack of quality treatment options (Bloom et al 2007). It has also been noted that injury incidence in UK football academies since the introduction of the EPPP has seen a three-fold increase amongst the players (Read et al 2018b).

As discussed in previous chapters, the true incidence of AS is difficult to ascertain with the varying recording methods, but Stracclioni et al (2007) suggest that AS represent around 5% of injuries to adolescents, with athletic children having greater incidences up to 23% (Reece 2012). It has also been suggested that the higher the level of sport being played, the higher the incidence (Maffuli et al 2005) and Read et al (2018b) found a 3-fold increase in injury risk in football academies in the UK, which peaked following the time of PHV. There are also issues with under-reporting. Rathleff et al (2013) found that 30% of contacted adolescents reported having had knee pain of which 2/3rds were insidious onset and 1/3 traumatic. However only 59% of adolescents sought medical care for their knee problem, 41 % did not. DiFiori (2010), Rathleff et al (2013) and Magrini and Dahab (2016) all commented that most adolescents with insidious onset of knee pain do not seek treatment as often as those with more traumatic onset injuries, which gives rise to concern as they both can have similar consequences regarding pain severity, duration and reduced quality of life.

5.2.2 Aetiology

Authors are in general agreement that the primary mechanism of injury for all AS is caused by repetitive overload mechanism causing overuse microtrauma (Outerbridge and Micheli 1995; Brukner and Khan, 2012; Peterson and

Renstrom, 2017), yet there are incidences that appear to be precipitated by a traumatic event such as a direct blow or fall (Kridelbaugh 1948; Madden and Mellion 1996). DiFiori (2010) states that the pathology behind overuse injuries is more akin to those of degenerative changes which suggests that poor healing and repair is a main factor, with a lack of acute inflammatory components in the tissue. Microtrauma would also lead to a weakening of the apophysis making it more vulnerable to further powerful muscle contractions or trauma, thus a degenerative cycle is set up leading to a greater vulnerability to further injury (DiFiori 2010). Therefore, some AS injuries may have components of both traumatic and overuse mechanisms (Meeuwisse 1994; Madden and Mellion 1996; Brukner and Khan 2012).

There is a lack of research into the aetiology of AS beyond expert opinion and supposition but identifying specific risk factors may help to draw understanding into which factors may be more relevant and help to explain why some adolescents develop AS and others do not.

5.2.2.1 Risk factors

Causal mechanisms for sports injuries are likely to be multi-factorial due to the number of factors that could play a role (Meeuwisse 1994). Dalton (1991) and Frank et al (2007) suggested dividing injury mechanisms into either intrinsic, occurring from internal forces within the body, or extrinsic from external forces or events from outside the body (Dalton 1991; Meeuwisse 1994; Bahr and Holme 2003; DiFiori 2010; Brukner and Khan 2012). Meeuwisse (1994) developed a multifactorial model that describes predisposing influences involving intrinsic risk factors, then affecting the athlete exposed to extrinsic risk factors, resulting in creating a susceptible athlete (Bahr and Holme 2003). Some of these factors will now be covered under the sub-headings of intrinsic and extrinsic risk factors.

5.2.2.1.1 Intrinsic risk factors

Age and maturation

Ivins in 1961 suggested OSD was caused by “growth disturbance” but Alpert (1962) refuted this idea stating that it was considered to be related to direct trauma. Antosia and Lyn (2002) suggested that OSD was related to the degree of skeletal maturity, but this does not explain why some adolescents get AS

and others do not, as all adolescents go through the same stages of growing albeit at different rates (Tanner 1965; Buckler 1990). It is apparent however that the skeleton grows first distally in the feet, then progressively up the leg, with the trunk later (Dimeglio 2001; Chamley et al 2005; Kanbur et al 2005), and it has been noted that ankle and foot problems tend to occur in younger adolescents and children with knee and hip problems later in adolescence (Fuglkjaer et al 2017). Faude et al's (2013) review noted that maturation status and proximity to PHV was a risk factor in adolescent footballers with 10-40% of all injuries classed as overuse and Read et al (2018c) found that advanced maturation was a risk factor for injury.

Kemper et al (2015) studied academy footballers and found that the injury risk was increased if they grew by more than 0.6 cm/ month. Van der Sluis et al (2014) also found a link between injury risk in elite young footballers during the year of their peak height velocity, although they found higher numbers of traumatic injuries than of overuse during this period, yet DiFiori (2010) noted an increase in overuse injuries associated with the growth spurt. In contrast to this Jayanthi et al (2015) performed a large case-controlled study with 1214 athletes and found that the risk of injury was not related to growth rate, however they were looking at the broad spectrum of injuries and not AS in particular. James et al (2015) noted that their 124 Sever's patients were significantly taller, heavier and had a greater body mass index (BMI) than their normative values but did not discuss its implications. Obesity could be an issue as greater weight would increase the active load on the skeletal system and Sever (1912) himself associated over-weight with his heel patients. However, bone growth is actually stimulated and enhanced by weight bearing (Hall 2011) so this should generally cause bones to become stronger not weaker. Excessive loading however which takes tissue beyond its normal tolerance levels will weaken it and could give rise to injury (Hall 2011).

Nordstrom et al (1995) found a higher than usual level of bone mineral density (BMD) in young athletes compared with non-active age matched subjects. They found that the BMD was site specific to the tibial tubercle and related to the increased muscle strength of the quadriceps. Interestingly 25% of their active group had previously had OSD and their BMD was significantly reduced.

It appears that strong quadriceps have a positive effect on the tibial tubercle BMD up to a point, but those with previous OSD have less density in spite of stronger quadriceps. OSD may therefore have a negative effect on BMD, or perhaps those with lower BMD were the ones who developed OSD. The direction of the relationship i.e. whether cause or effect, is unclear, and to this researcher's knowledge has not been studied prospectively.

Nakase et al (2015) used ultrasound imaging to stage development of tibial tubercle (TT) amongst 150 young male football players and tracked development over one year, finding 10 players who developed OSD (14.3% incidence) from the 70 that started at the individual stage TT classification, yet all were asymptomatic. They only found significant differences between the affected and non-affected groups with the OSD group having increased quadriceps and hamstring tightness and decreased quads strength, not growth stage of the TT.

Growth levels are closely linked to genetics and some authors have noted the preponderance of siblings and parents having suffered with AS (McKenzie 1981; Kvist et al 1985; Kujala et al 1986) but no analysis has been done to date to this researcher's knowledge. The onset of pubertal timing and skeletal growth has a strong genetic influence (Tanner 1965) so it could be a possibility that genetics has an influence, but it is unlikely to be the main factor, or far more siblings would be affected.

In overview Micheli and Fehlandt (1992) state that the incidence of AS is heightened during growth periods in the child and adolescent but have not identified which growth factor(s) may be responsible. It may be a single factor or a combination of factors of varying importance e.g. the specific rate or volume of growth or other factors, and how these may differ in those who develop AS and those who do not.

Flexibility

As previously discussed in Chapters 2-4 of this thesis, reduced flexibility has been suggested by many authors (Ogden 1976; Micheli 1987; Outerbridge and Micheli 1995; Madden and Mellion 1996; de Bengoa 2011; de Lucena 2010; Peterson and Renstrom 2017) as a possible factor in the development of AS, but few studies have actually investigated it. Ikeda et al (1999) first noted

increased tightness of the quadriceps muscle in the affected leg of OSD patients and went on to further investigate their findings in 2001 specifically amongst young footballers and found significant poorer flexibility and also and increased preponderance in the dominant leg. They suggested this could be due to the increase in eccentric contractions and rapid deceleration which occur when landing from jumping and landing in sport. This would place an increased tensile load on the patella tendon and its attachment at the tibial tubercle, compared to jogging, cycling or other cyclical activity (Escamilla 2001). Furthermore, an increased incidence of SLJ was found in cerebral palsy children who have characteristics of high tone/ tight muscles due to spasticity, but the direct investigation was not explored (Munk and Vellet 1993). In contrast to this a Danish study showed there was a high incidence of OSD and SLJ (61%) in children with hypermobility (Junge et al 2015), but hypermobility is an assessment of joint flexibility, so it is important that clarity of terminology is used to describe the different types of flexibility and tissue specificity, and their possible association with AS.

Mehdinasab and Fakoor (2005), Tzalach et al (2016) and Shiota et al (2016) also found significantly tighter quadriceps in their OSD footballers, but also found tighter hamstrings and hip rotators, which suggest a global thigh muscle tightness in OSD patients, not just the target muscle, and especially amongst the 2-joint (hip and knee) muscles i.e. those with the longest span. However, Allison et al (1998) found a reliability problem with measuring quadriceps stiffness in 10 OSD patients, as daily variability appeared to be high and could have just been normal standard errors of measurement. They concluded that there may have been limited clinical significance in their measures. They did not however compare their patients to a control group to see if there were any significant differences between the groups, rather than just the reliability of repeated measures.

Micheli (1983) and Smith (1991) suggested that muscle tightness could be a factor, and possibly due to the faster growth of long bones (such as the femur) compared with the slower accommodation of the muscle-tendon unit (Ogden 1976; Outerbridge and Micheli 1995). The only study (Feldman et al 1999) to directly examine changes in flexibility related to adolescent growth found no

association, however this study has already been criticised by the researcher as having chosen a time frame likely to be after the peak growth period and therefore could have missed the critical time frame. In contrast, De Lucena et al (2011) did find that flexibility of the quadriceps decreased during adolescent school children and was more marked for those that developed OSD.

Backman and Danielson (2011) found that a low range of dorsiflexion (of the ankle) predisposed their young basketballers to patella tendinopathy and Sarcevic (2008) found that 93% of their OSD patients also had reduced ankle dorsiflexion, which they interpreted as indicating possible tight calf muscles. Their suggestion was that tight calf muscles could in turn alter the biomechanics of the knee by requiring compensatory increases in knee flexion, thus increasing the stress on the quadriceps and therefore the tibial tubercle attachment. Clearly such a strong finding needs further investigation, but it may be another indicator that global lower limb muscle tightness is apparent in adolescents. Improving flexibility may have positive effects on young athletes as Smith et al (1991) found that 75% of their adolescent figure skaters eliminated their anterior knee pain by improving their quadriceps flexibility (N=46). Whilst they did not specify the pathology or diagnoses, increasing flexibility did decrease their pain, and it is entirely possible that some of their skaters had OSD or SLJ.

Reduced calf flexibility has been reported by a number of authors in Sever's patients with de-Bengoa et al (2014) finding a decreased range of dorsiflexion of 10˚, Szames (1990) finding tightness in 82% of their patients, and Scharfbillig et al (2011) also identifying calf tightness as a risk factor. Yet in contrast to these findings James et al (2015) reported that symptomatic patients in their study had a greater range of motion in their ankles. They describe the anomaly as possibly due to a taller and heavier group having longer growth phases, but it is unclear how this would increase joint range.

Overall it would appear however that the literature supports there being a period of some muscle tightness that is normally associated with adolescence, but degrees of tightness could become a risk factor for AS.

Muscle strength and conditioning

Bahr (2012) suggested that a combination of rapid muscle development together with large training loads may lead to the onset of AS, and Jakob et al (1981) also noted that hypertrophied quadriceps in OSD. Outerbridge and Micheli (1995) proposed that muscle imbalances could be a factor in OSD, and Katoh (1988) found increased eccentric strength ratios in their OSD patients compared to their controls. Katoh assessed concentric and eccentric quadriceps strength in the un-affected legs of OSD patients so whether this was an anomaly or characteristic of both legs is unknown. Yet Ikeda et al (2001) did not find any differences in quadriceps muscle strength between football players with OSD and those without, so the relationship between strength and muscle imbalances and AS has not been proven to date. It has not yet been commented on whether quadriceps dominant sports such as football have a higher incidence of OSD compared to other sports with more equal agonist: antagonist relationships or single leg dominance compared to more symmetrical sports such as running, swimming and tennis. Lord and Whinell (2004) recommended improved strength and conditioning as a strategy for preventing overuse injuries, as well as educating athletes on its importance.

Previous injury

Prior injury is a known risk factor for sports injuries generally (Wen 2007, DiFiori 2010) and the nature of overuse injuries is such that previous injury is inherent. Therefore, overuse injuries are more likely to recur and therefore constitute a risk factor in themselves.

Anatomical factors

Tissue studies on bovine animals have found adolescent osteochondral tissue 4.4 times weaker to shear forces than adult mature cartilage, but interestingly 1.5 times weaker even than immature cartilage (Flachsmann et al 2000). It therefore appears that there is a distinct weakening of the cartilage tissue during the adolescent period itself which may increase its vulnerability to injury. DiFiori (2010) states that the immature cartilage found at the epiphyseal growth plates make an athlete more vulnerable to injuries to these zones. Akiyoshi et al (2017) found that the immature stages of the development of the tibial tubercle (Stage 1-2 cartilaginous and apophyseal stage) and higher likelihood

of developing OSD. This suggests that immature status may make them more vulnerable, yet those with Stage 3-4 (epiphyseal and bony stages) still developed OSD, so it is not conclusive.

Other anatomical factors such as mal-alignment and joint laxity have been suggested as risk factors for AS (Willner 1969; Micheli and Ireland 1987; Aparacio et al 1997; DiFiori 2010) but evidence has been presented mainly in studies of Sever's disease (Szames 1990; Scharfbillig et al 2011; Gijon-Norueron et al 2013; Aygekum 2015). This evidence of biomechanical mal-alignment in Sever's may in part be due to the expertise of the reporting authors who were specialists in biomechanics of the foot – podiatrists. The main mal-alignment found in these cases was of equinus which means reduced dorsi-flexion of the ankle, which could be due to tight calf muscles. Micheli and Ireland (1987) found over-pronation in 18% of Sever's patients whereas Gijon-Nogueron et al (2013) found 50% in their sample. This does however mean that 82% and 50% respectively were not, so the case for over-pronation as a risk factor has not been proven to date. Evidence of mal-alignment for OSD is limited. Jakob et al (1981) found that 67% of their OSD patients (N=185) had mal-tracking of the patella and Gigante et al (2003) found greater external rotation of the tibia in their 41 OSD patients, but the degree of these mal-alignments was not given. Sen's two studies (1988 and 1989) found the patella in OSD was pulled laterally by 14˚ more than the control counterpart group (N=139) and also that the patella was pulled more proximally. This could add to the argument for increased quadriceps tightness pulling the patella (and its tendon) further away from its apophyseal attachment. However there have been no studies to explore the relationships between anatomical mal-alignment and AS, therefore it is not known whether the greater the degree of mal-alignment creates a greater risk factor, or whether this is just an observation.

5.2.2.1.2 External risk factors

Single sports specialisation (SSS)

SSS is defined as "intense, year-round training in a single sport with the exclusion of other sports" (Jayanthi et al 2013, pg 252). There is a belief

amongst coaches, parents and children themselves, that early single- sports specialisation (SSS) helps to identify and nurture talent amongst athletes and gives the best chance of elite success (Malina 2010; Feeley et al, 2015), however the literature does not support this. Authors have found that early specialisation did not actually translate into higher future performance (Malina 2010; Ford et al 2009; Jayanthi et al 2013; Read et al 2016), in fact they found the more general and unstructured the early physical activity, the better athletes developed. Therefore, there is a strong case against early specialisation given the questionable outcomes of performance and higher injury risk (Malina, 2012; DiFiori et al 2014; Magrini and Dahab,2016; Pasulka et al 2017). Magrini and Dahab (2016) recommend that young athletes should not specialise until late puberty,and take more breaks from organised sport during each week and for a couple of months per year, and that more time should be spent in un-structured play or multi-sports activities. Many authors (Brenner 2007; Feeley et al 2015; Myer et al 2015; and Pasulka et al 2017) also describe an increase in overuse injury risk from SSS in particular amongst the individual sports of tennis, swimming and gymnastics.and question its efficacy and implementation. The sports with the youngest specialisation ages were gymnastics (8 years), dance (10 years) and football (10 years). Read et al's (2016) comprehensive analysis of UK football academies recommended decreasing volume of training within SSS and allowing for more variety of physical activities during the key stages of growth. Hall et al (2015) specifically noted a four-fold increase in OSD and SLJ in athletes in SSS.

The training of young athletes (TOYA) study found that parents were the greatest influence on whether their child specialised in sport (Baxter-Jones et al, 2003), but school and coach encouragement are also important (Jayanthi et al 2013). Parental influence into SSS, may possibly be due to a parent's desire to give their child an edge over competitors (Malina 2010), but also the possibility of lucrative sports contracts and careers for the very few, becomes a strong incentive especially if the child shows 'talent'. Indeed, there is a whole sports goods and services industry and academic sciences built around identifying early talent and developing it to its potential (Malina 2010). With the greater pressure for athletes to begin high-intensity training at earlier ages (Bahr 2010; DiFiori et al 2014), concerns are being raised about the risks of

SSS to such an extent that position statements have been published both the American Medical Society for Sports Medicine (DiFiori et al 2014) and the American Orthopaedic Society for Sports Medicine (LaPrade et al 2016). Both statements agree that early sport specialisation is not a pre-requisite for future sporting success and may even be unhealthy physically and mentally, as the risk of injury increases and the levels of burn-out and withdrawal from participation of young athletes testifies. Indeed, Fabricant et al (2016) systematic review found a significantly increased level of overuse injury risk amongst SSS and withdrawal from tennis, however only 3 studies were of sufficient rigour and levels of evidence. There is a clear need for targeted research into early SSS, as the evidence is growing that the concerns raised by the medical community need urgent investigation (Bahr 2014; Feeley et al 2015).

Training volume

The volume and intensity of training has also been identified as a risk factor in overuse injuries (DiFiori 2010). Jayanthi et al (2015) stated more hours of sport per week than the child's age or a greater ratio of 2:1 of organised sport to play led to increased risk of serious overuse injury. There are some sports who promote a high training volume as essential to the development of elite sports skills and ability. Gymnastics, tennis and dance are individual sports that have been found to have greater training volumes, and subsequent higher overuse injury rates when compared with team sports and (Pasulka et al 2017). Pasulka et al (2017) also found that these sports and football had the youngest age of specialized athlete, together with the highest training volumes.

The standard advice guidelines from the UK Chief Medical Officer recommends at least 60 minutes of daily physical activity for children aged between 5 to 18 years old with a range of intensities ('Live well' guidelines www.nhs.uk 2018). The recent recommendations from the EPPP (2011) aim for a new greater training load than previous years for the football academies, in the quest for greater skills and player development. The guidelines suggest a marked increase (+120%) in accumulated contact hours over the academy life cycle from age 8-21, from 3760 hours to 8500 hours (EPPP, 2011), and specifically a significant increase during the Youth age groups from 12-16. This

age group coincides with the peak adolescent growth period but also reports the greatest injury incidence amongst youth footballers (Read et al 2016). Therefore increasing training load at this vulnerable time may be considered an unreasonable additional risk, in particular for growth-related injuries such as AS, Considering the wide variation in individuals' maturity levels, the volumes, intensity and frequency of training should be carefully monitored to allow for rest and recovery (and growth) (Maffuli et al 2004; Balyi and Wray 2005; Wen 2007; DiFiori 2010). Read et al (2018b) suggest a compromise of a reduction in volume of training during the vulnerable key stages of growth and maturation at foundation and youth levels, to allow for optimal skill acquisition but reduce injury risk. Athletes should also have targeted coaching and training to optimise their physical, mental and emotional development during the different phases of their individual developmental ages (Stanitski 1989; Lord and Whinell 2004; Bayli and Way 2005; Viera et al 2018). This may prove more difficult in a team situation, but care should be taken to try to respect individuals within the team as not being all equal (Bahr 2014). There are growing calls for team sports to be selected by biological, not chronological age, or "bio-banding" (Cumming et al 2017) as this allows for competition based on skill rather than physical maturity and allows for more targeted and appropriate training volumes (Balyi and Way 2005).

Equipment and footwear

Some authors have suggested that poor footwear may be partly responsible (Dalton 1992; Outerbridge and Micheli 1995; Madden and Mellion 1996; Walter and Ng 2002; Hendrix 2005). Given that children's feet are also growing rapidly during adolescence, the need for repeated new shoes that are well-fitting and supportive is important but costly (Hendrix 2005). Only Walter and Ng's study (2002) specifically looked at cleated boots in young footballers and drew an association between cleats and negative heel positioning (increased dorsiflexion of the ankle) having an influence on Sever's disease, but they did not look at the influence of the playing field conditions (hard or soft) which may equally have had an influence on the position of the boot itself. There have been no other studies on the effect of sports equipment on AS to this researcher's knowledge, other than the use of heel cups after the event i.e. the

development of Sever's; for treatment purposes (Perhamre et al 2010; Weigerinck et al 2016)

Psychological factors

Adolescents are also undergoing marked changes in self-awareness, confidence and other psychological developments associated with puberty (Tanner 1965, Buckler 1990, Bayli and Way 2005) and this may lead to an increase in vulnerability to injury (Brenner 2007; Di Fiori 2010; Bahr 2012). There have been some studies on the psychological impact of Sever's disease on patients (Scharfbillig et al 2009; James 2016), but no studies to this researcher's knowledge have looked at psychological issues as a risk factor in AS.

In summary there are some educated opinions about the aetiology of AS but a paucity of scientific research. As yet there have been no studies that have demonstrated a direct cause and effect of either intrinsic or extrinsic risk factors, with most authors repeating clinical observations or each other. It is unlikely too that there is a single over-riding factor Meuwisse (1994) so studies would need to consider multiple variables adding to the difficulty of producing good scientific evidence. Chapter 4 studied two of the most commonly cited suggested risk factors, of growth and flexibility, and this chapter describes a planned intervention based around these. Assessing the other risk factors mentioned is beyond the scope of this particular study and thesis but should be part of future comprehensive research into the aetiology of overuse injuries in children and adolescents.

5.2.3 Injury prevention

There have been no studies on AS prevention to this researcher's knowledge, but the American Medical Society for Sports Medicine position statement on overuse injuries in youth sports (DiFiori et al 2014) suggests a number of approaches based on available evidence. Limiting participation levels to include a variety of sports movements and including rest periods is highly recommended. Stanitski (1989) and Maffulli et al (2011) stated that intense training of young athletes is occurring at younger ages, and exposure of young athletes to programmes with excessive exercise exposes them to greater risk of injury. Reducing SSS and intensity of training loads especially during growth

spurts would help to reduce injury risk (DiFiori et al 2014). Appropriate pre-season strength and conditioning, and neuro-muscular training can also help to reduce lower extremity injuries (Stanitski 1989; Lord and Whinell 2004; Balyi and Way 2005; DiFiori et al 2014; Mackie and Taunton 2017) and making sure that protective sporting equipment worn and used is well-fitted and in good condition will help to reduce poor biomechanical factors (Stanitski 1989; DiFiori et al 2014; Mackie and Taunton 2017).

These tactics are quite generic across sports and overall overuse injuries but do provide a common-sense approach and to date represent the best available advice for injury prevention. Injury prevention at elite English football academies is a recent topic of investigation with Read et al (2018) attempting to draw similarity of approaches to injury prevention. Although they found the most common injuries were traumatic rather than overuse, the common strategies were similar to that promoted by DiFiori et al (2014) although they felt that training frequencies could be increased to enhance training response. Maffulli et al (2011, pg 187) state that "active prevention measures are the main weapon to decrease the (re)-injury rate and increase athletic performance" and represent a positive proactive approach to care of young athletes. Encouraging youth to embark on healthy physical exercise is beneficial but equally important is reducing the incidence and severity and possible long-term consequences of injury. There is growing concern amongst the medical and sports science fraternity about the increased risk of injuries amongst highly trained young athletes (Maffulli & Helms 1988; Lord and Whinell 2004; Caine et al 2006; Maffulli et al 2005), and it is vital that prevention strategies should be implemented wherever possible (van Mechelen et al 1992).

5.2.4 Progression of theory

In the previous chapters of this thesis, reduced muscle flexibility has been found to affect AS patients and is prevalent in athletes during their secondary growth spurts and is strongly associated with their rate of growth. Reducing the passive tension behind the tight musculature has also been shown to improve patient recovery response times and pain. This could give rise to using flexibility measures as a marker for prevention of AS during the peak growth period. Setting a threshold where players who drop below a certain level of acceptable

flexibility could give an opportunity to intervene and eliminate reduced flexibility as a risk factor and may impact on the incidence rate of AS positively.

5.2.5 Aims

To assess whether an intervention programme based on improving muscle flexibility will prevent the development of AS within a football academy and to create a basis for a prevention strategy to be used across academies and beyond.

To assess flexibility changes during growth and AS incidence rates across 3 football academies to see if there are any differences between matched populations.

Objectives

Analyse the effect of a stretching intervention programme on players whose flexibility dropped below guide thresholds, and monitor them over 4 seasons to see if they develop an AS.

Compare flexibility changes across 3 football academies over 4 seasons to see if the populations are similar in presentation and profiles.

Compare incidence rates of AS across 3 football academies as matched club academies, over 4 seasons to see comparisons in rates with the club with the intervention prevention strategy

Compare the recovery rates for AS patients between the academies

5.2.5.1 Hypotheses

H_{01}: There will be no difference in AS incidence rates within the CP academy

H_{A1}: A stretching intervention programme will reduce the incidence rates of AS within the CP academy

H_{02}: There will be no difference in AS rates between academies

H_{03}: Flexibility does not change during the adolescent growth period across between the CP academy groups

H_{04}: Flexibility does not change during the adolescent growth period across 3 football academies

H_{05}: There will be no difference in AS treatment response rates between the 3 football academies

5.3 Method

5.3.1 Procedure

Head Physiotherapists from three football academies volunteered to collect data for 5 years on AS incidence and monitor flexibility across the academy players for the purposes of this study. The Head Academy Physiotherapists of Crystal Palace (CP), Brentford (Club B), and Brighton and Hove Albion (Club C), gave informed consent, and full ethical approval was obtained from the University of Greenwich Ethics Committee as part of the research remit for the previous chapter. All the data was collected as part of their ongoing academy profiling for which all academies had parental informed consent and it was only access to their existing data that was required. All testing and medical staff had enhanced Disclosure and Barring checks.

Two studies constituted this overall prevention study. Firstly, an intervention study at one club targeted players (intervention group = IG) with reduced muscle flexibility and implemented a stretching programme and compared them with their control cohort (CG), and also those players who developed an AS. Secondly comparisons were made with other club academies as to the overall AS incident rate and flexibility changes to see if there were any differences between the clubs. Recording AS occurrences across the clubs over time would allow for comparisons of total incident rates and determine if any changes over time were found across the academies or only in the intervention club.

5.3.2 Settings and frequency

Data was collected on a tri-monthly basis for flexibility of the quadriceps and calf muscles, by the team physiotherapists, and collated by the Head Physiotherapist. Diagnoses of AS were made by team physiotherapists and confirmed by the Medical staff or Head Physiotherapist. At the end of every season the data was sent by encrypted email to the researcher for data and club protection. No individual identifying information was used and players were identified only by code number, which the researcher did not have access to.

5.3.3 Monitors

All testing was performed by Chartered Physiotherapists in each academy who were responsible for their own team, and data collected on site at each club by the Head Physiotherapist. The Head Physiotherapist supervised all flexibility testing to improve validity and reliability of measures. The researcher was responsible for collecting and collating the data from each club, as well as all analysis. All staff had pre-study training and explanations as to the nature of the research, and time for questions and clarification.

5.3.4 Muscle flexibility

5.3.4.1 Club comparisons

Resting length tests for the quadriceps and gastrocnemius muscles were performed in the same manner as the previous academy study in Chapter 4 and followed the procedures by Kendall et al (2005). Academies all used similar 12-inch clear medical grade goniometers (NCD Prestige Medical at CP, and True Angle by Gaiam Pro at Club B but the brand at Club C was unknown. The same procedures as Chapter 3 were followed for leg positioning with respect to gravity and passive resting tension of the quadriceps and calf muscles was recorded, with 2 measures taken and the mean recorded if the margin of difference was < 4˚ (Kendall et al 2005). If the difference between measures was > 4˚ then a third measure was taken and the median recorded. Tests were recorded bilaterally, and players nominated their dominant leg. Clubs reported the age group means at the end of each season and the researcher was responsible for all data collection.

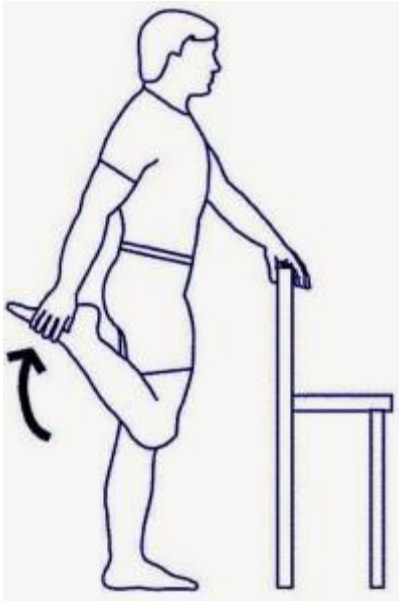
5.3.4.2 Stretching intervention

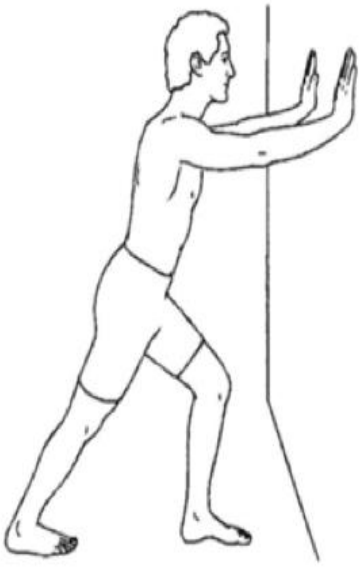
Only at the CP academy was an intervention strategy implemented if player's muscle flexibility measures at any testing date, dropped below thresholds of 45˚ flexion for the quadriceps or above 38˚ plantarflexion for the calf. These thresholds were set after discussion based on the expert clinical experience of the researcher and academy head Physiotherapists, as to what they regarded as a 'tight' threshold for each muscle and was in accordance with the literature (Kendall et al 1995; Sarhmann 2002). These guideline thresholds provided an initial baseline parameter for this study. These players were then prescribed daily extra stretching exercises for the appropriate muscle. Only the 'at risk'

players were chosen for the intervention strategy to allow for comparison with a control group and the AS group and determine any differences in flexibility between the groups and more importantly impact on AS incidence within the academy. The stretches were the same as for AS treatment protocol in Chapters 1 and 2 but are further described here in detail. Stretches were demonstrated to the players by staff, then players were asked to perform them under supervision to verify correct technique and the appropriate stretch sensation. Players were told that no pain should ever be felt with the stretches and to stop and clarify the technique with the team physiotherapists before commencing. Instructions given to the players were as follows:

5.3.4.2.1 Quadriceps stretch

Players were asked to stand on one leg holding onto a stable surface for balance, bring the opposite foot behind towards the buttocks, flexing at the knee and keeping the hip in neutral, and the knees close together with no rotation (St. George 1989). Pull the foot upwards, flexing the knee until a feeling of gentle tension was felt in the target quadriceps muscle belly, breathe out and relax as the stretch was taken up (Figure 44). Keep the stretch position for 10 seconds, lower the leg back to the floor, and repeat the stretch 5 times. Players were asked to perform this stretch regime 3 times daily, evenly spaced out during the day.





Figures 44 and 45. Standing stretch on the right quadriceps and gastrocnemius muscles

5.3.4.2.2 Gastrocnemius stretch

Standing facing a wall placing their hands against the wall for support and place the target leg behind with the heel and foot flat on the floor. Keep the body weight forwards weightbearing mainly through the front leg and arms, so that the back leg is minimally weight bearing, and the hip and knee were in a straight line. Lean forwards until a stretch was felt in the calf muscle. (Figure 45) (St. George 1989). Keep the stretch position for 10 seconds, return from the forward lean, and repeat the stretch 5 times. Players were asked to perform this stretch regime 3 times daily, evenly spaced out during the day.

Players were monitored every week by academy staff until flexibility thresholds were surpassed, and thereafter remained above the thresholds. These players constituted the intervention group, the other Crystal Palace academy non-AS players constituted the control cohort.

5.3.5 Incidence of Apophysitis Syndromes

Players in all 3 clubs diagnosed with AS were removed from training and competition and treated with the same protocol as described in Chapter 2 and Chapter 3. Training at the football clubs on the new treatment protocol for all club physiotherapists was held by the researcher at the start of the study, and ongoing email provision was given for any feedback and support required. All AS diagnoses were reported to the Head Physiotherapist who recorded the type of AS, onset date and the time taken for the player to recover fully and return to training and sport. The annual total occurrence of AS in a club was the main dependent variable as this would provide the basis for comparison and effectiveness of the intervention strategy. The other academies of Club B and Club C were used for comparisons of incidence rates in the overall study and were designated the control clubs where no interventions were used, with Crystal Palace the only intervention club. Further note was made of recovery time for all AS patients who were given the new treatment protocol, to assess its efficacy across the wider academy population.

5.3.6 Training load

An overview of each team’s training hours was obtained from academy staff with contact hours of training and perceived rates of exertion to determine

workload on players throughout the seasons. This was recorded as workload minutes per week and was based on the age group recommendations from the EPPP for ‘Foundation’ and ‘Youth’ age groups of 8-11 years of age and 12-16 years respectively.

5.3.7 Statistical procedures and analysis

The statistical software package SPSS 25 (SPSS Inc, Chicago, Il, USA) was used for statistical analysis throughout. The distribution of the raw data sets was assessed using the Shapiro Wilks test to determine normal distribution. Descriptive statistical analyses including means and standard deviations (SD) were used for normal distributions, and medians and ranges for non-normal were used to describe the academies’ AS incidence rates, and flexibility changes within the CP groups. Differences within the CP academy groups’ flexibility were analysed using One-way ANOVAs, and T-tests with two samples of equal variance, with Post-hoc Tukey’s HSD, to determine where any differences lay between the academies for muscle flexibility. Differences in AS treatment response between the clubs was analysed using Independent samples means test and Kruskal-Wallis. All analyses were performed to a significance level of $p < 0.05$ and with a 95% confidence interval.

5.4 Results

5.4.1 Stretching intervention at Crystal Palace

Within the Crystal Palace academy 23 players were found to have reduced quadriceps flexibility dropping below the given thresholds of 45˚, and 15 players exceeding above 38˚ for their calves, and together they formed the intervention group (IG N=38). Means are detailed in Table 18 together with comparisons from the control cohort and the relevant AS sub-groups (OSD, SLJ and AIIS for the quadriceps; Sever's for the calf). There were no significant differences found between the groups regarding age at the lowest flexibility for the calf (One-way ANOVA P=0.36) or for the quadriceps (One-way ANOVA P=0.43)

Table 18. Summary of ages and flexibility across the groups at their tightest measures (means and standard deviations)

Means and SD	N=	Quads flex˚	Age (years)	N=	Calf flex˚	Age (years)
Intervention Group (N=38)	23	45 (±1)	12.4 (±1.2)	15	40 (±3)	11.1 (±1.6)
AS group (N =27)	18	36 (±4)	12.52 (±1.45)	9	45 (±4)	10.9 (±1.57)
Control cohort (N = 105)	56	51 (±1)	13	49	33 (±1)	11

The IG profile of ages and muscle group are shown in Figure 46.

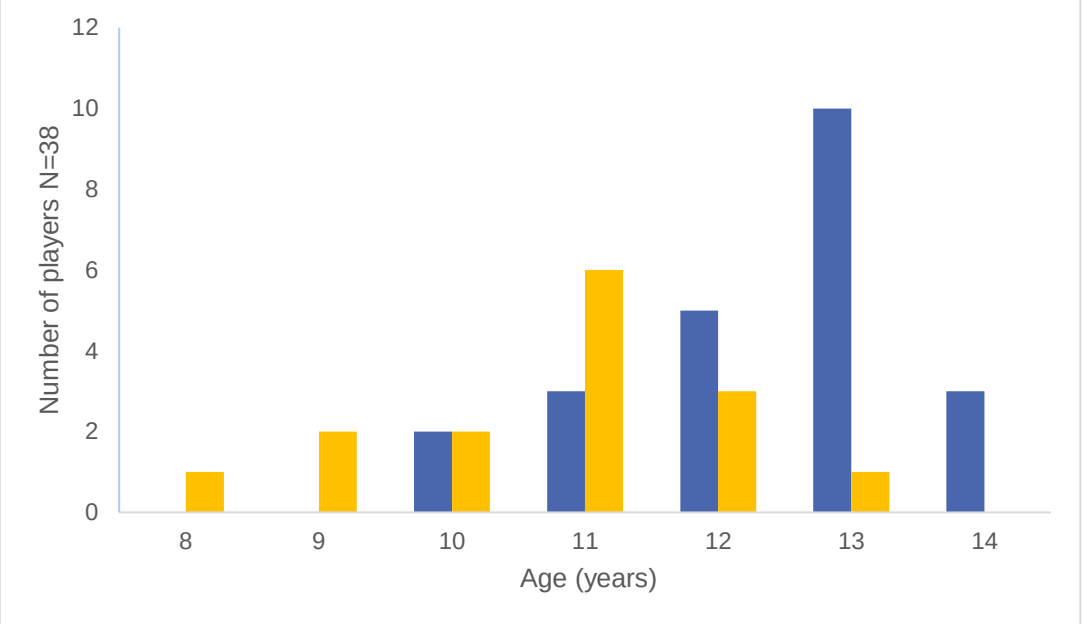


Figure 46. Profile of ages and targeted muscle in intervention group (Quadriceps = blue; Calf = orange).

5.4.1.1 Quadriceps flexibility

A comparison analysis of quadriceps flexibility across the three groups – intervention (IG), apophysitis sub-groups (AS = OSD, SLJ and AIIS) and control (CG) for the year leading up to and the year after their lowest readings, shows a reducing trend across all three groups with improvement thereafter, but the CG had a low point mean of 50˚ (±4.) compared to the AS low point mean of 35˚(±4). The IG were started on the intervention strategy when they dropped below 45˚ with a lowest point mean of 44˚ (±2). There was a significant difference in flexibility between the groups [One way ANOVA $F(2,95) = 131$, $p < 0.001$]. Post-hoc comparisons using the Tukey's HSD indicated that the inter-group mean scores were all significantly different from each other. The AS sub-group had a drop of 20˚ during the prior year compared to 11˚ for the IG and 7˚ for the CG. (Figure 47). The time frames for each measurement session was not consistent but was kept as close to between 2-3 months, therefore comparisons during the two years were difficult to make as they would be at different times for every team and player. Therefore, the lowest point of flexibility for each player was the one consistent point able to be used for

comparison and therefore its absolute value was chosen for the data set. The x-axis shows the 4 measurements prior to the lowest point and 4 sessions after as there were not consistent time periods, but the data was collected as closely as possible starting one year prior and finishing one year after. This graph should be viewed understanding that the lower the flexion angle, the tighter the quadriceps muscle.

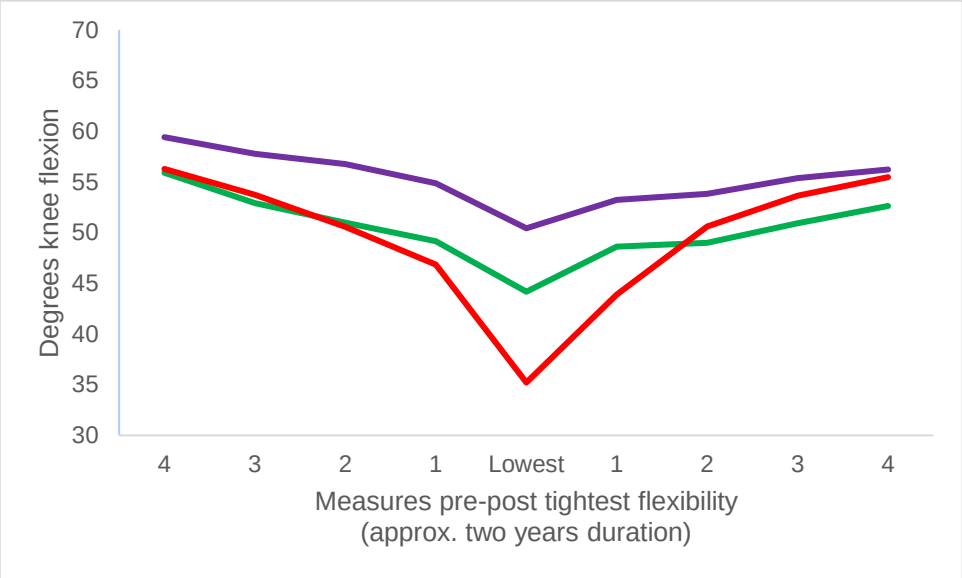


Figure 47. Mean changes to quadriceps flexibility pre/post-maximal tightness recorded over 2 years (AS= red; IG =green; CG= purple)

5.4.1.2 Calf flexibility

Similar analysis of calf flexibility showed the tightest mean flexibility for the Sever's group at 45˚ (±4), with the IG at 40˚ (±3) and the CG at 34.5˚(1.7). (Figure 48). Again, the IG had the intervention strategy introduced when their flexibility exceeded 38˚and this represented their lowest point. Differences between the groups were also significant [ANOVA $F(2,70) = 91$, $p < 0.01$). Post-hoc comparisons using the Tukey HSD test indicated that the inter-group mean scores were all significantly different from each other. The Sever's group had a decrease of 18˚ over the prior year, compared to IG of 9˚ and CG 5˚. This graph should be viewed on the understanding that the higher the ankle angle the tighter the calf muscle, and again the horizontal axis represents the 4 measurements in the preceding year and 4 measurements finishing a year after the lowest calf flexibility recording.

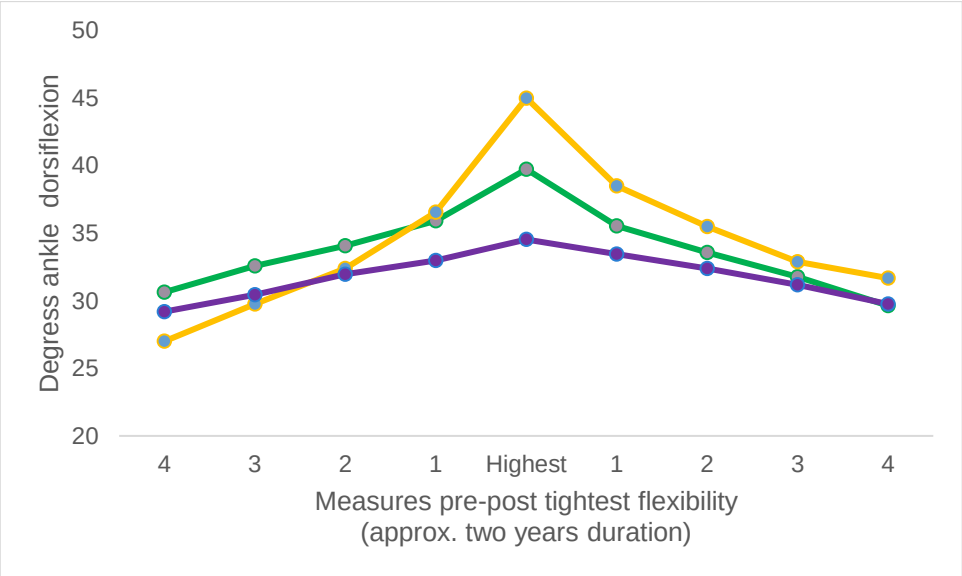


Figure 48. Changes to calf flexibility pre/post-maximal tightness recorded over 2 years (Severs =orange; IG =green; CG= purple)

Therefore, there was a significant difference between the groups' quadriceps and calf flexibility and thus the null hypothesis H_{03} is rejected.

5.4.2 Club flexibility

Year group means for quadriceps and calf flexibility were only obtained from CP and Club B, with Club C unable to provide any data due to changes in personnel and club priorities. Analysis of year group flexibility in the CP control cohort and club controls at Club B showed no significant difference between the clubs for quadriceps and calf flexibility (F-test one tail P=0.39; t-test: Two-sample with equal variance (two-tailed) with P=0.08 and P=0.51 respectively (Figure 49). The data had a normal histogram distribution. Note that for club comparisons the calf measurements have been calculated as number of degrees from 90˚ to allow for comparisons on the same graph i.e. 30˚ of plantarflexion would be visualised as 60˚ on this graph, therefore the lower the degree the tighter both quadriceps and calf muscles are. Both clubs showed a similar trend towards tighter calf muscles peaking at age 11, and quadriceps peak tightness at age 13, with both improving the following years, therefore inter-club results indicate changing flexibility over the adolescent years in both the quadriceps and calf muscles, and this also disproves the null hypothesis $H_{04,}$ and therefore this is rejected

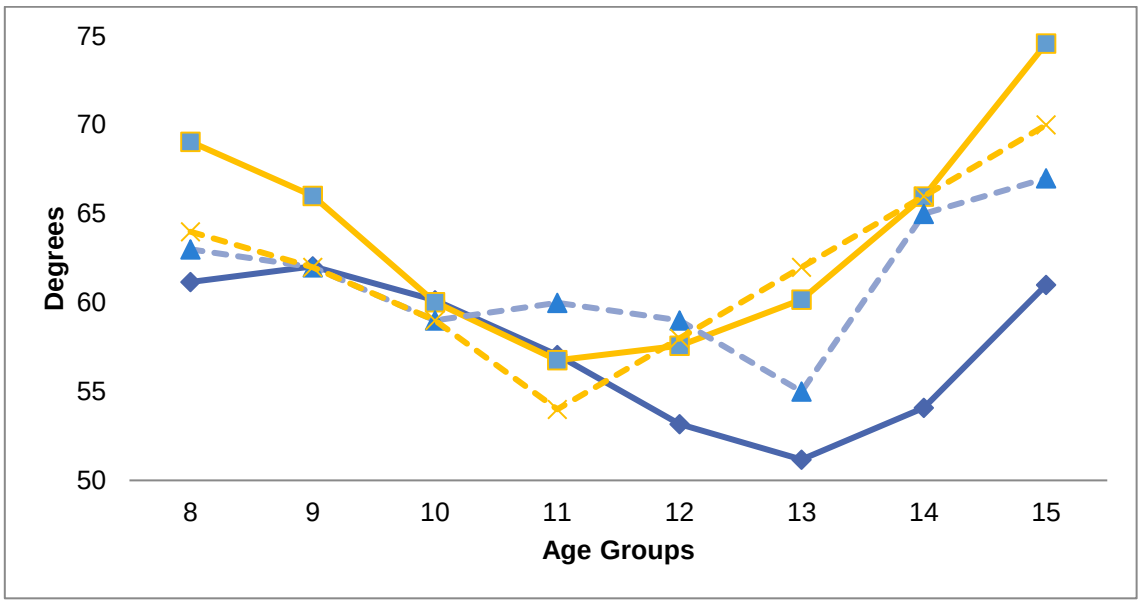


Figure 49. Age Group flexibility means for the quadriceps and calf muscles from 2012-15 (CP solid lines quadriceps = blue, calf = orange; Club B dashed lines quadriceps = blue, calf = orange)

5.4.3 Apophysitis syndrome

5.4.3.1 Incident rate

The incident rate for AS within the CP academy dropped from an initial 13 new AS patients annually to only 4 new patients by the final 4th season, a drop of 69% (Figure 50). This means the null hypothesis H_{01} is rejected and the alternate hypothesis H_1 is accepted as there was a decrease in incidence that may have occurred due to the stretching intervention. As described in the previous Chapter 3, a total of 34 AS conditions were reported with 25 patients with quadriceps-related apophysites (OSD, SLJ and AIIS) and 9 patients with calf-related Sever's disease. AS incidence data of new cases was received from Club B, and Club C Academies, but their rates were similar throughout with a slight decrease overall of 7% and 15% over the 4 seasons. Therefore, the null hypothesis regarding AS incidence rates between clubs, H_{02} is rejected as rates were different between academies by the end.

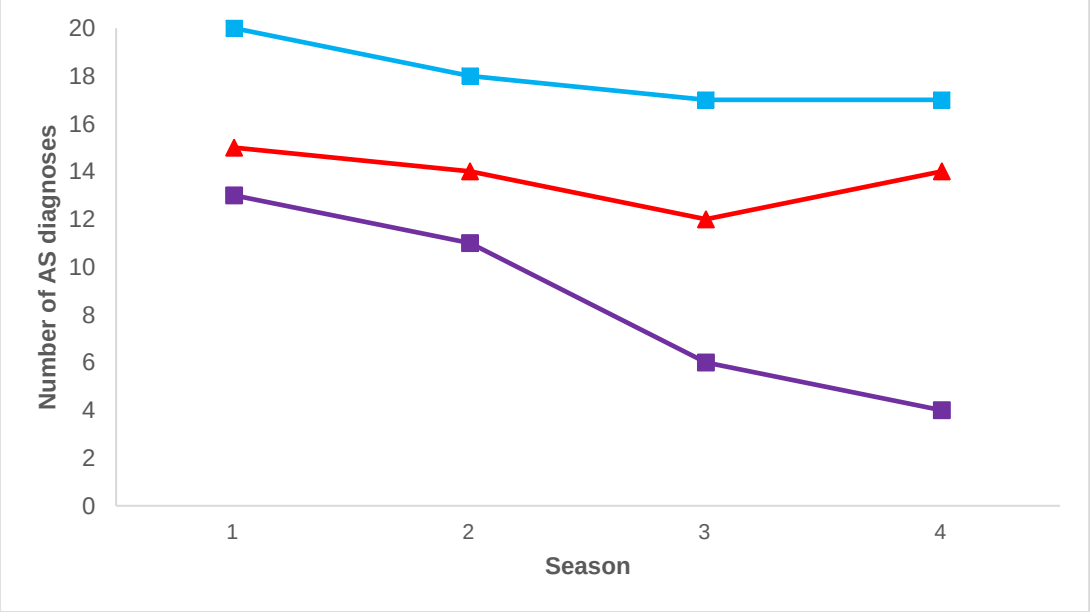


Figure 50. Comparison of new AS incident rates between clubs over 4 seasons (CP= purple; Club B = red; Club C = blue)

In total, Crystal Palace had 34 players diagnosed with an AS, Club B had 55 players and Club C had 72, giving a total of 161 AS patients. Therefore, CP accounted for 21% of the total reported injuries, Club B had 34% and Club C had 48%. The club comparison rates with each other at the start were CP 27%, Club B 31% and Club C 42% but at the end the rates were CP 11%, Club B 38% and Club C 50%. Further analysis by AS type is shown in Figure 51.

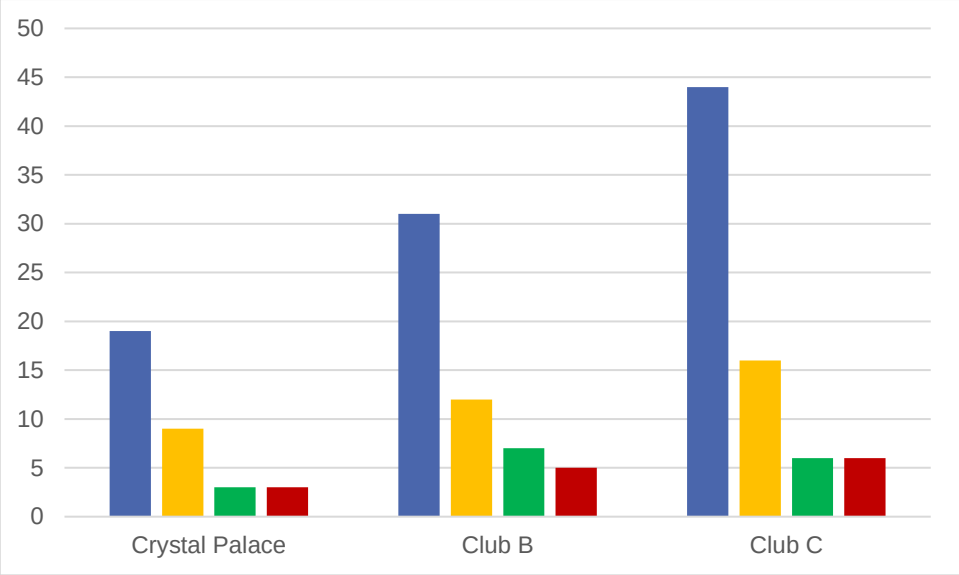


Figure 51. AS totals by club and type over 4 seasons (OSD= blue; Sever's = orange; SLJ= green; AIIS = yellow)

When compared with the total academy populations over the 4 seasons, CP had an overall club AS incident rate of 15%; Club B had 25% and Club C had 33%. But at a squad/ team level, CP had incidence levels as high as 28%, Club B had two squads with 50%, and Club C had one squad with 80%, and two others with over 50%. Profiles of AS were similar in ages across the clubs, and the information is summarised in Table 19.

Table 19. Apophysitis ages across clubs (means and standard deviations)

Age (means and SD)	Crystal Palace	Club B	Club C
OSD	12.4 (±1.6)	12.7 (±1.4)	13.4 (±1.18)
Sever's	11.0 (±1.6)	11.4 (±0.6)	12.0 (±0.9)
SLJ	13.0 (±0.5)	13.2 (±2.4)	13.9 (±1.1)
Hip	13.2 (±0.5)	14.0 (±1.0)	13.9 (±1.0)

5.4.3.2 Treatment response

AS patients at all academies used the same treatment protocol as outlined in Chapters 2 and 3, with all academies achieving a median return to play of under 3 weeks (Table 20), therefore the null hypothesis H_{05} is accepted. Data was not normal in distribution as it was skewed to the left. The differences of the club medians were not significant (Independent samples medians test

(P=0.19) but the distributions were (Kruskal-Wallis P=0.03). Previous historical data provided by the medical records showed the academies having a recovery time median and return to training of between 3 – 6 months for AS patients, and some players were released through poor recovery, though precise numbers were unable to be investigated in time for this study.

Table 20. Treatment response – days to return to play (medians and range)

Club AS diagnoses (N =)	Crystal Palace (N=34)	Club B (N =55)	Club C (N= 72)
Return to train (days)	20 (3 – 44)	16 (7 – 32)	14 (1 - 54)

5.4.4 Training load

Training load for each CP squad was documented with scheduled matches in 23 weeks of the year plus a small number of additional days for cup runs, which meant players were in competitive competitions for over 50% of the year. There were usually 14 free weekends during the season including Christmas and Easter, and the summer break consisted of a four month break from late May to early September. The individual squad contact training hours were within the EPPP guidelines for each age group i.e. ‘Foundation’ ages 8-11 years with 4 to 8 hour per week; ‘Youth’ ages 12-16 years between 12 to 16 hours per week. Variations of training intensity and type depended on the coaches’ assessment of squad and individual needs. The academy staff at CP used a combination of training hours per week multiplied by RPE to calculate player’s workloads, with a build-up of workload towards the weekend match, and this assessment also included total distances run, heart rates, and speeds. Further details of the training loads were not available for this study and were therefore unable to be analysed.

5.4.5 Summary of key results

- None of the players in the stretching intervention group (IG) developed an AS during the study
- There was a significant drop in new AS cases in the CP academy over the 4 seasons, from 11 down to 4 per year, whereas the incidence in the control clubs remained similar to previous levels (ranges Club B =12-15; Club C = 17-20)
- Individual squad incident rates could be very high at up to 80% over the 4 seasons
- Reductions in flexibility in the quadriceps and calf muscles occurred during the 9 to13-year ages in two academies
- Treatment responses to the protocol were similar across the academies resulting in quick recovery and return to training (3 weeks), compared to their historical outcomes (3 – 6 months).

5.5 Discussion

5.5.1 Stretching intervention

The first finding of this study is that no players who were given the stretching intervention went on to develop an AS. Whilst there have been no intervention studies with which to compare the prevention aspects of this study, the 38 players' flexibility in the IG, was decreasing over a period of time, and dropped below the set threshold. The stretching programme reversed that trend, restoring their flexibility closer to that of the CG. Some players who did not drop below these thresholds at a previous measurement, missed being identified as at risk and developed an AS, and it may be that lower thresholds could be set to identify them as well. The thresholds had originally been set on the basis of clinical experience with reference to the literature (Sahrmann 2002; Kendall et al 2005, but it is noted that the threshold amount was based on professional opinion. For example, the previous mean recording for the quadriceps prior to onset in the AS group was 47˚, and 37˚ for the calf muscle in the Sever's group. Setting new thresholds of 50˚ and 30˚ respectively may improve the identification of more 'at risk' players and provide an opportunity to intervene

in these players as well. Whilst it cannot be claimed that this stretching programme prevented AS, there is strong evidence that it may have helped as no cases of AS occurred within the intervention group during this study, and no other variables to their routines were altered. Once the intervention programme was started, the incident rate of AS at CP dropped by 69% over the 5 years. Further exploration of the role of flexibility in the development of AS (or not) is therefore justified.

The most important finding was that no players in the intervention group (N=38) went on to develop an AS within the time frame of this 4-season study, and the incident rate within the club substantially decreased, therefore the null hypothesis H_{01} is rejected and the alternate hypothesis H_1 is accepted.

This is the first cohort prevention study on AS, the results of which successfully show no incidents from the IG over the study period of 5 years. To date there have been no other systematic studies into the prevention of AS.

5.5.2 AS incidence and treatment response

The incidence level at Club B and Club C remained similar if showing a slight decrease over 4 seasons. This may have been due to greater awareness of AS due to their inclusion in the study and possible inadvertent stretching interventions, but to which extent this may have occurred is unknown. Although thresholds were not used at either Club B or Club C, medical staff may have recommended stretching if a player was noted to have marked reductions in flexibility and this may have caused an inadvertent intervention. This advice would be part of the staff's normal duty of care and is difficult to control as a possible variable. However, the overall rates at these clubs remained similar year-on-year. In contrast the CP rate dropped year-on-year from an overall rate of 7.2% of the whole club, to 2.2%; with Club C only dropping from 11% to 10%, and Club B staying the same at 8.3%. If the CP IG had gone on to develop an AS, then the rates would have been similar to both Club B and Club C (31 AS + 38 IG = 69 possible AS). These annual rates are higher than the 5% reported by Stracclioni et al (2007) but less than the 20% recorded by Reece (2012). It is not known at this stage why the incidence levels differed from the

literature but there is growing concern amongst medical staff at English academies that the increase in training hours introduced with the EPPP guidelines may be responsible for the recent increase in injuries, in particular for the 'Youth' age groups (Read et al 2018b). It is also important to note that individual squad incidence rates could be very high with one squad having 80% of their players suffering an AS during the time of the study, and many others were over 50%. This would have huge implications and ramifications for a coach being able to field their team based on player ability and skill and not just their injury status, let alone the team dynamics and season results. Having full availability of players allows for the optimal development of the individual and team and can enable the demonstration of the effectiveness of the coaching and academy ethos. This would also demonstrate one of the key components of the EPPP in understanding and promoting injury risk and ultimately prevention (EPPP 2011).

Consistency in reporting incident rates for AS as a group or as individual pathologies remains a challenge, and until an agreed formula is found true comparisons will be difficult to ascertain. It is this researcher's suggestion that AS should be grouped and recorded together, as they appear to have the same factors of reduced flexibility and growth related, and the same pathological characteristics (Pappas 1967; Micheli 1987; Reece 2012). This would allow better understanding of the true scale of these syndromes during adolescence. AS could then be further sub-categorised for more detailed incidence rates of each variation i.e. OSD/ Sever's.

What this study has shown is that the CP AS incidence levels dropped substantially compared to the control clubs and it is suggested that this was due in some part to the prevention strategy of the stretching intervention, therefore the null hypothesis HO2 is rejected. No other studies have been done that show a reduction in AS incidents rates – be it in a sporting or non-sporting arena, and the results of this study indicate that AS incidence can be proactively reduced.

Treatment response was similar to that found in the previous studies in Chapter 1 and 2, although there was a difference in distribution between the clubs. The reason for this is unclear but different club staff may have implemented their

own return to play criteria. It was very difficult to keep total control over final rehabilitation decisions from outside the club's immediate medical staff, and indeed some of those staff were not present at the start of this study. Overall the return to play median was under 3 weeks for all clubs and under 2 weeks for one (Club C) which is a very favorable result when compared with their historical anecdotal returns of between 3-6 months, and the clubs were very happy at the quick response. Their prior recovery rates using traditional treatment interventions of rest, anti-inflammatory and analgesic medication were in line with that proposed by other authors (Antich and Brewster 1985; Peck 1995; Madden and Mellion 1996; Nicholas 2007; Kivel 2011). Not only does the quicker return mean less pain and dysfunction for the patients, but also positively benefits the club allowing for full squad selections and talent development. Therefore, the null hypothesis H_{O5} is accepted, as clubs had similar responses to treatment using the new protocol

It also is a strong demonstration of the academy staff's duty of care and willingness to be at the forefront of using best practice for patient benefit (NHS - National Institute for Clinical Research, 2018). Whilst there is no direct written evidence, the academy staff noted that some players with AS had been released from the academies due to the chronic nature and slow recovery from the conditions. A faster and complete recovery may mean that players are not rejected on health grounds and can therefore fulfil their potential, and clubs retain the talented players they initially identified.

5.5.3 Flexibility changes

This is also the first experimental study to show different flexibility changes across a group of adolescents from the same football academy. The results show that muscle flexibility reduces during certain periods of growth, with significant reductions over the 12 months prior to the lowest recording, across all CP groups, but with the AS group showing the most significant drops. Therefore, the null hypothesis H_{03} is rejected as there were differences in flexibility between the CP groups.

Across the academies flexibility also appears to change over the period of adolescence thus the null hypothesis H_{O4} is rejected. The findings of this study

agree with other authors (Sarcevic, 2008; de Lucena et al, 2011; de-Bengoa et al, 2014) whilst in contrast to those of Feldman et al (1999). When compared to the other clubs, the overall year group means are similar which indicates that reducing flexibility is apparent across the 3 academies at around the same ages. This could give rise to identifying peak times of vulnerability for the quadriceps and calf muscles, and therefore the AS associated with them. As previously discussed in Chapter 3, reduced flexibility and specific growth velocity are closely associated with the onset of AS. These growth phases give rise to possible prime vulnerability periods of around age 11 for calf flexibility, and age 13 for quadriceps flexibility and AS, and coincide with the prime onset ages for the AS conditions seen in this and the previous study. This could provide an opportunity for academy staff to continually monitor flexibility changes longitudinally and take extra note during these periods of vulnerability, which would fit in the aims of the EPPP, specifically during the 'Youth' ages (Read et al 2016). It would be the researcher's recommendation to implement a targeted daily stretching programme aimed at the leg muscles as a matter of good practice with the aim of reducing AS, and to continue this during the peak growth period between the ages of 10 -14 years. Good quadriceps and calf muscle flexibility may reduce the incidence of the most common AS (OSD, Sever's and SLJ), but other leg muscles such as the hamstrings and sartorius have also been identified as additional origins of hip apophysites, and improving their flexibility could also help to reduce the development of other AS. Skeletal growth occurs in all planes therefore it is likely that a more encompassing flexibility programme, incorporating all the major leg muscle groups, will help to reduce AS incidence in the leg and pelvis.

5.5.4 Training load The training load at the CP academy was similar in hours to the EPPP guidelines for each age group but varied with each squad and at different times of the year, and over different years. It was not within the scope of this thesis to study the training loads in addition to the anthropometrics and flexibility of players and detailed information of the various training loads was not available at the time, therefore analysis was not possible for this study. Training load can be categorized as internal or external but Bourdan et al (2017) suggest a combination of both is best. External assessment is based

on multiple factors such as pitch counts, volume of hours spent training, and intensity based on a 5-point scale as related to match play. The ratings are Low, low-moderate, moderate, moderate-high, and high (Bourdan et al 2017). However, the accuracy and reliability of this can depend on the expertise level of the assessor and methods for monitoring (Barnes 2013). Internal assessment of training load can be based on resting heart rate, rate of perceived exertion (RPE) and training diaries, but there can be significant inaccuracies amongst young players in assessing their own physicality (Bourdan et al 2017). The CP staff used a combination of training hours and RPE to calculate the daily and weekly workload of playersIt has been suggested that acute: chronic workloads as determined by weekly: monthly ratios, may give better guidance for long-term planning and insight into injury risk (Hulin 2016; Bowen 2017) as higher or lower ratios tend towards a higher risk (Bourdan et al 2017). Equal loading had the lowest injury risk but does not take into account fatigue towards the end of season and some authors suggest taking into account rolling averages to better assess overall workload (Williams et al 2016; Menaspa et al 2017). Volume of training should be increased progressively over the years, but speed and intensity can be introduced as needed as part of a season-long plan to take into account peaks in competition (Malina 2003; Bowen et al 2017). Training load has been recently identified as a possible risk factor for injury in football academies (Read et al 2016) and the authors strongly recommend the close monitoring of training loads during the rapid growth period as these players were found to have the greatest injury rates and severity. It has also been suggested that increasing the amount of free play time and other physical activities away from SSS is beneficial for safer athlete development (Read et al 2016; Bourdan et al 2017). It may also need further research to ascertain whether the increase in training hours from the Foundation ages to the Youth age group as recommended by the EPPP, is too much too soon, as a player moving from age 11 to 12, could have an increase training hours from 8-12 hours per week – a 50% increase at their most vulnerable time (Read et al 2016).

5.5.5 Limitations

The main challenge to this study was from the multiple-club involvement. Changing personnel within clubs meant that we were unable to obtain flexibility

measures from Club C, and full anthropometric data from both Club B and Club C. New staff meant that their priorities may have changed and those of this study demoted. In the real world of high-pressure elite sports, the staff at the academies have to respond first to their leadership, but we were fortunate to continue to receive some input from these clubs.

Incidence rates may have been affected by player release and selection as clubs changed player personnel throughout the study. Some players may have been released before they developed an AS and their longitudinal data was not known, or players may have been recruited just prior to its onset, with no opportunity to intervene. Further analysis of the full historical club medical records and rationale behind individual player's selection or release and follow-up would need to be analysed to get a full picture. However, this reflects the real world of changing populations and as such the data was as accurate as possible.

This study aimed to assess the effect of a stretching programme targeting 'at risk' players and monitor whether they developed an AS. The limited scope of this thesis meant that other risk factors mentioned in the background section such as SSS, detailed training volume and load were unable to be investigated, and this means that the full understanding of AS risk has not been identified, just that of reduced muscle flexibility, and therefore there may be better prevention strategies still to be determined. It would be advantageous to examine all potential influences in the future to maximise our understanding of aetiology and prevention of AS and other injuries that affect young athletes (Read et al 2016).

5.5.6 Summary

This study primarily found that a stretching intervention programme prevented 38 young adolescent footballers' muscles from becoming too tight and restored their flexibility to that of their control peers. None of these players developed an AS. It is suggested that this intervention prevented them from developing an AS, as similar players with tightening muscles, who were not targeted, went on to develop an AS. This is the first prevention study on AS and the results suggest it has been successful.

This also study found that there were two specific time periods where adolescents had a natural decrease in flexibility. The calf muscles got tighter at a mean age of 11 years, and the quadriceps at a mean age of 13 years, with these time periods coinciding with the peak onset of AS. There may be other factors involved that this study has not assessed but it appears that addressing reduced flexibility has a positive effect on the incidence of AS in a football academy, reducing the rate in one academy from 7% down to 2%. Together with objective flexibility thresholds, it is suggested that AS could be prevented especially during these rapid growth periods, and in particular for those whose rate of growth is well above average. The findings provide a simple and objective prevention strategy for AS. This would empower academy staff to be proactive and reduce the incidence of one of the most problematic injuries seen in football academies.

5.6 Conclusion

There appear to be two time-periods in adolescence where muscle flexibility reduces in academy footballers, those around the ages of 11 years to 13 years. Targeting flexibility reductions beyond a given threshold and implementing a stretching programme appears to have prevented academy footballers from developing an AS. Academy staff should be aware of these windows of opportunity to carefully monitor flexibility and intervene if it drops below the thresholds set, and thus help to prevent AS developing in their players.

CHAPTER 6. General Discussion

6.1 Summary of thesis

There are limited studies into AS in general and most of the published literature is either descriptive or based of expert observation and opinion, yet these painful conditions continue to occur year-on-year to a new cohort of adolescents. It is estimated that AS affects up to million patients every year with documented poor outcomes from traditional advice and treatment, yet few intervention studies have been published and no prevention studies. Whilst a limited number of studies have explored aetiological and risk factors, there is a dearth of scientific endeavor into investigating cause, effect and prevention of AS, yet AS remain the most common overuse injury to affect this specific age group, with the highest incidences amongst young athletes. This thesis has explored a multi-stratified approach based on a logical model of passive and active traction forces, which appear to be the pathological cause of AS, and address them from a clinical angle incorporating a new treatment approach, and epidemiological angle observing growth and flexibility in an elite football environment., Together these encompass a management approach which aimed to answer some of the questions behind AS.

6.1.1 Clinical approach: New treatment protocol (Studies 1 and 2)

The use of massage in the treatment of AS has never been described before and its use appears to address the increased passive tension found in AS patients, as demonstrated by the patient's reduced muscle flexibility. Having a dual approach where both passive and active tension are addressed allows time for the affected apophysis to heal and patients responded to the treatment recovering in a much shorter time than is normally expected. Recovery also had long-term efficacy with a low recurrence rate of 5% reported therefore the protocol appears to be both restorative and curative. OSD patients who were treated in the acute and sub-acute stages had the quickest response to treatment in less than 2 weeks, and the best long-term outcomes as gauged by residual pain and level of sport. This thesis presents a sound argument for using the passive-active model in the treatment of both OSD and Sever's disease and return adolescents and children to their normal lives as quickly as

possible. It also provides cheap, simple clinic-based tests to allow for objective monitoring and feedback.

6.1.2 Epidemiological approach

6.1.2.1 New aetiological factors (Study 3)

The longitudinal football academy study allowed the tracking of a number of adolescent anthropometrics and growth parameters that may have had an influence of the development of AS. Identifying reduced flexibility and fast growth rates ties into the theory of growth causing increased passive tension, as observed in reducing flexibility, yet the researcher recognizes that cause and effect have not been proven, just that there is a strong association between the two parameters. Patients who developed an AS had the fastest growth rates and the tightest muscles in the academy, and the strongest correlation between flexibility and PHV. There may be additional risk factors that are involved as discussed in the literature such as training load, SSS and strength development, and this thesis acknowledges its limited scope in identifying only two.

6.1.2.2 Prevention (Study 4)

Prevention of injuries is the primary concern for all those involved in supporting sport either as a health professional, coach or sports administrator. Understanding some of the risk factors involved in the development of AS gives an opportunity to be proactive and intervene when those risk factors appear. This is the first intervention study into the prevention of AS and the results demonstrated that a cohort of targeted players (N=38) with reduced flexibility beyond a set threshold, did not develop an AS, unlike their matched AS cohort (N=31). The stretching intervention restored their flexibility back to the levels of the control group, and appears to have halted one risk factor associated with AS. This study also demonstrated that the incidence level of AS dropped only at the academy where the intervention was applied, with two other academies having similar annual incident rates throughout the study period. The multi-academy profiles also found that flexibility reduces between the ages of 11 and 13 years and could provide academy staff with a window of opportunity to closely monitor flexibility in players and intervene as necessary, to prevent AS.

6.2 Application of findings

Chartered Physiotherapists have a duty of care to their patients to deliver effective care, constantly evaluate treatments and to ensure that those treatments offered are the best evidenced available (CSP Quality Assurance Standards, 2017). They are in a position to be able to observe the effects of injuries on patients and the response to different treatment approaches, yet the scientific evidence to support many treatment techniques and approaches often lags behind their clinical use. Physiotherapists are in a prime position to use their skills and knowledge to observe, ask questions, and develop theories but not necessarily to test those theories in a clinical trial. The responsibility of treating the patient must always be their priority but there could be more collaboration between academics, sports scientists, strength and conditioning coaches and clinical practitioners to learn from each other and work together towards better understanding of treatment, aetiology and prevention of injuries. In a world of increasing demands on health budgets, targeting research on preventative strategies would benefit everyone and is likely to be cost-effective. Relying on traditional anecdotal treatment approaches such as those described for AS is not only ineffective, but also poor science, and health practitioners have a responsibility to challenge unproven approaches.

The current model of healthcare is firmly rooted in treatment, often aimed simply at relief of symptoms. This may or may not be influenced by vested interests or just the vocal demands of patients, but the effort, time and money spent on research into developing preventative strategies or investigating aetiology is a fraction of the budgets spent on symptomatic relief, benefiting a healthier population and less burdened health professionals. If a more structured and pragmatic approach were made then it might be possible to target understanding the aetiology on a multitude of health problems, especially those related to gradual onset or overuse. Once the aetiology is understood then prevention strategies could be put in place thus reducing the number of injuries and conditions requiring treatment and reducing the demand on stretched health resources. Not least of all it would reduce the number of

patients suffering, which is the ultimate patient benefit and should be the goal of every health professional and educator.

Current healthcare funding is limited yet demand is growing with an increasing and ageing population and higher expectations that every problem can be solved. The NHS has to find a more efficient way of managing healthcare but one that also provides the most effective treatment interventions for minimum cost. This is a challenge for all of us, and all health practitioners are in a prime position to help face it. They have the professional skills and the knowledge but may lack the confidence in scientific research to use it. This is a major under-utilised aspect of NHS staff skills that could be a solution to its problems.

Growing adolescents in this study showed reductions in muscle flexibility that were associated with their peak height velocities. Coaches, sporting bodies and health professionals who specialise in youth sports should be aware of the period around an athlete's PHV, in particular the acceleration phase, where leg muscle flexibility can be adversely affected. They should also be aware of the velocity of growth and its strong association with flexibility and AS. Intervening and implementing a stretching programme when this occurs will help to prevent AS and possibly other conditions such as muscle strains from occurring. Football academies can demonstrate their duty of care by understanding this vulnerable time and tailoring training programmes accordingly, taking care not to overload with volume of specialized training, but intersperse it with other physical activities for more rounded development. Academies might also review their selection policies as targeting early maturation players may increase the risk for AS and thus inadvertently increase the incidence within their squads.

There are difficulties associated with research on people, and adolescents in particular. People are unique with their own characteristics, genetics, environment, attitude, talent and opportunities. Attempting to perform rigorous scientific study means that these many variables have to be accepted, and the absolute quality and analysis of data compromised, but this is the real world of the coach and physiotherapist. Studying adolescents when there is such a wide variation in timing and profiles of growth is an even more difficult undertaking but should not dissuade research into this important population with their own

unique problems. Accepting the limitations of real-world research provides an opportunity to explore ideas and solve problems, the scientific evidence is difficult but not impossible to attain.

6.3 Future work

This thesis has identified two risk factors associated with AS but there are likely to be more. Future research should also examine the active loads placed on young athletes with immature skeletons. As discussed, there are a number of other factors such as training load, SSS, strength and skeletal segmental development, footwear and training environment that should be studied to gain a full picture of the physical influences on athletes. This thesis has not investigated the role of psychological influences, but they are part of an athlete's performance and attitude and should be explored to see if they pose an additional risk factor for overuse injuries such as AS. If other risk factors are identified they would give rise to further improve prevention strategies for the benefit of future athletes.

This study found the investigation of leg length segments to be unreliable, but better training and testing could improve understanding as why different AS affect different ages. It could be linked to bone development and the different rates of growth of different segments during adolescence.

It would be useful to see if the passive-active treatment protocol had similar results for the many other AS, and this researcher encourages other health professionals to proactively engage using it with their own patients. This study was not able to assess whether severity of OSD or Sever's had an influence on the treatment response, and future studies could use US imaging to identify the stage of AS and provide more clarity as to best treatment outcomes. US imaging is an exciting tool that may allow for greater understanding of the effect of massage on the muscle, and myofascial tissue in particular, to help understand why this technique appears to produce such a quick response from patients.

Further research on the passive stretch tests and wall squat should be performed to provide evidence of their reliability and validity. Even though

these tests may be in common use clinically there is a lack of scientific evidence to show they are addressing the factors we think they are. Professional opinion and use is not enough for good scientific studies, so the evidence must be found for these or better tests.

6.4 Conclusions

Treatment of the two most common types of AS has been proven successful, using a theoretical aetiological and treatment model based on the combination of reducing passive and active tension in the target muscle. Patients responded to the new treatment protocol in less than 3 weeks, recovering from symptoms and returning to their sport in a much shorter time than is usually expected. It also demonstrated long-term benefits which may provide the basis for a curative treatment. The protocol warrants potential application to other AS found in adolescents. This is the first study to use a logical model to develop a treatment protocol and to objectively monitor a patient's recovery.

Taking the passive-active model and monitoring adolescent growth found a strong correlation between growth rate and reduced flexibility and could be a risk factor for the development of AS as patients had slightly faster levels of growth, and significantly less flexibility together with a stronger association than their control counterparts. This is the first study that has identified and explored the relationship of these two potential risk factors and their role in the development of AS. Coaches and health professionals should be aware of the importance of these two factors and their relationship to the possible development of AS.

Flexibility decreased across the football academies during the players' growth spurt, but thresholds exceeded resulted in the development of AS. Players who were given a stretching intervention at these thresholds did not develop an AS, indicating that the intervention worked to prevent its onset. This is the first study to have implemented a cohort study into a prevention strategy for AS and the first to show success in reducing the incidence levels at one football academy.

This thesis is an investigation into the role of passive muscular tension as a factor in the development, treatment and prevention of AS. As such it

represents a holistic and comprehensive approach to the management of these injuries that could serve as a model for good and effective health care practice, and guideline for elite youth sports environments.

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APPENDICES

Appendix I. Example Patient Information Sheet – Apophysitis syndrome treatment

Participant Information Form

Title of Study: "A new physiotherapy treatment for Apophysitis syndromes"

Dear Participant,

We invite you to take part in this research which has been designed to study whether a new physiotherapy treatment will help you to improve the speed and quality of your injury recovery

What is the purpose of the study?

Apophysitis syndromes tend to take a long time to recover but there are few available options for patients. We want to find out whether a new approach to treating your injury will help you to recover faster and better. This study also forms part of the Doctorate study for Jenny Strickland, PhD student and Chartered Physiotherapist

Do I have to take part?

No. It's totally up-to-you and your parents. You do not have to give any reasons for not taking part. During the study you can stop and withdraw at any time without it affecting anything.

What will be expected of you?

If you decide to take part your parents/ guardian as well as you will have to fill in consent forms. If you withdraw from the study any data and information collected will be deleted and not used. The treatment uses standard physiotherapy techniques such as massage and stretching, and you may have to rest from sport and activities for a while too. You may have to have the massage performed daily at home in the early stages by your parents.

How will the recorded data be used?

We will listen carefully to what you and your parents have to say about your recovery and how you are doing. The data recorded will be analysed to help us see if the treatment has improved your recovery from injury and got you back to playing sport again in a quicker time than normal. The data collected from you may be published in journals and may provide us with advice for your fitness and medical team to help prevent you from getting certain injuries. However, ALL data will be kept confidential and your name will never be used. No-one will be able to recognise or identify you other than Jenny.

Are there any risks?

No, you are not at any risk. All procedures are standard physiotherapy techniques, but the massage can be uncomfortable, so you must tell us, and we will reduce the pressure accordingly. We will teach your parents how to massage you safely too so that the treatment can continue at home for a faster recovery.

Confidentiality

All the data we collect will be kept strictly confidential. Names will not be disclosed. You will be assigned a number or code word instead to identify your specific data, and this identifying key will be kept in a separate file to your data.

If you require any further information, please to not hesitate to contact either

Jenny Strickland J.M.Strickland@gre.ac.uk 0208 331 8614 (PhD student) or

Dr Mark Goss-Sampson M.A.Goss-Sampson@gre.ac.uk Senior Lecturer (Supervisor)

Appendix II. Example Parent/ Guardian Information Sheet – Apophysitis syndrome treatment

Parent/ Guardian Information Sheet

Title of Study: "A new physiotherapy treatment for Apophysitis syndromes"

Dear Parent/ Guardian,

We invite you to take part in this research which has been designed to study whether a new physiotherapy treatment will help the speed and quality of your child's recovery from their injury.

What is the purpose of the study?

Apophysitis syndromes tend to take a long time to recover but there are few available options for patients. We want to find out whether a new approach to treating your child's injury will help them to recover faster and better. This study also forms part of the Doctorate study for Jenny Strickland, PhD student and Chartered Physiotherapist

Do I have to take part?

No. It's totally up-to-you and your child. You do not have to give any reasons for not taking part. During the study you can stop and withdraw at any time without it affecting anything.

What will be expected of you?

If you decide to take part your child as well as you will have to fill in consent forms. If you withdraw from the study any data and information collected will be deleted and not used. The treatment uses standard physiotherapy techniques such as massage and stretching, and your child may have to rest from sport and activities for a while too. We will instruct you on how to apply the massage technique to their muscle at home for better treatment continuity,and will ask you to perform the massage on a daily basis in the early stages of the treatment.

How will the recorded data be used?

We will listen carefully to what you and your child have to say about their recovery and how you are doing. The data recorded will be analysed to help us see if the treatment has improved their recovery from injury and got them back to playing sport again in a quicker time than normal. The data collected may be published in journals and may provide us with better understanding on the best treatment for apophysitis syndromes. However, ALL data will be kept confidential and your name will never be used. No-one will be able to recognise or identify you or your child other than Jenny.

Are there any risks?

No, you are not at any risk. All procedures are standard physiotherapy techniques, but the massage can be uncomfortable, so you and your child must tell us, and we will make sure to reduce the pressure accordingly. We will teach you how to massage your child's muscle safely too so that the treatment can continue at home for a faster recovery.

Confidentiality

All the data we collect will be kept strictly confidential. Names will not be disclosed. You will be assigned a number or code word instead to identify your specific data, and this identifying key will be kept in a separate file to your data.

If you require any further information, please to not hesitate to contact either

Jenny Strickland J.M.Strickland@gre.ac.uk 0208 331 8614 (PhD student) or

Dr Mark Goss-Sampson M.A.Goss-Sampson@gre.ac.uk Senior Lecturer (Supervisor)

Appendix III. Example Consent form – Apophysitis syndrome treatment

PARTICIPANT/ PARENT CONSENT FORM

To be completed by the participant. If the participant is under 18, to be completed by the parent / guardian / person acting *in loco parentis*.

- I have read the information sheet about this study
- I have had an opportunity to ask questions and discuss this study
- I have received satisfactory answers to all my questions
- I have received enough information about this study
- I understand that I am / the participant is free to withdraw from this study:
  - At any time (until such date as this will no longer be possible, which I have been told)
  - Without giving a reason for withdrawing
  - (If I am / the participant is, or intends to become, a student at the University of Greenwich) without affecting my / the participant's future with the University
  - Without affecting any medical or nursing care I / the participant may be receiving.
- I understand that my research data may be used for a further project in anonymous form, but I am able to opt out of this if I so wish, by ticking here.
- I agree to take part in this study ☐

Signed (participant)	Date
Name in block letters	
Signed (parent / guardian / other) (if under 18)	Date
Name in block letters	
Signature of researcher J. Strickland	Date
This project is supervised by: Dr Mark Goss-Sampson	
Researcher's contact details (including telephone number and e-mail address): Jenny Strickland J.M.Strickland@gre.ac.uk 0208 331 8614 Mark Goss-Sampson M.A.Goss-Sampson@gre.ac.uk 0208 331 7986	

Appendix IV. Example Participant Information Sheet – Football Academy Growth Study

Participant Information Form

Title of Study: "Does growth rate affect the flexibility of the quadriceps and calf muscles in young male academy footballers, and does this predispose them to overuse injuries?"

Dear Participant,

We invite you to take part in this research which has been designed to study how quickly you are growing and whether it affects your flexibility.

What is the purpose of the study?

We want to understand whether the speed at which you are growing may affect your flexibility during your growth spurts. There are some sports injuries which may be partly caused by tight muscles and we want to see if they might be related so we can prevent them. This study also forms part of the Doctorate study for Jenny Strickland, PhD student

Do I have to take part?

No. It's totally up-to-you and your parents. You do not have to give any reasons for not taking part. During the study you can stop and withdraw at any time without it affecting anything.

What will be expected of you?

If you decide to take part your parents/ guardian as well as you will have to fill in consent forms. If you withdraw from the study any data and information collected will be deleted and not used.

The testing will include measuring your leg length and size, as well as your overall height, weight and muscle flexibility. These tests are like ones that you already do – we are just using the data specifically for this study.

How will the recorded data be used?

The data recorded will be mathematically analysed to help us see if there are any meaningful relationships that we can then use to see if some athletes will be more likely to get growth-related injuries. The data collected from you may be published in journals and may provide us with advice for your fitness and medical team to help prevent you from getting certain injuries. However, ALL data will be kept confidential and your name will never be used. No-one will be able to recognise or identify you other than Jenny.

Are there any risks?

No, you are not at any risk. All tests are standard body tests that you are already familiar with. And your investigators are all trained in working with children and supervised by physiotherapists.

Confidentiality

All the data we collect will be kept strictly confidential. Names will not be disclosed. You will be assigned a number or code word instead to identify your specific data, and this identifying key will be kept in a separate file to your data.

If you require any further information, please to not hesitate to contact either

Jenny Strickland J.M.Strickland@gre.ac.uk 0208 331 8614 (PhD student) or

Dr Mark Goss-Sampson M.A.Goss-Sampson@gre.ac.uk Senior Lecturer (Supervisor)

Parent/ Guardian Information Sheet

Title of Study: "Does growth rate affect the flexibility of the quadriceps and calf muscles in young male academy footballers, and does this predispose them to overuse injuries?"

Dear Parent,

We invite you and your child to take part in this research which has been designed to study growth rates and flexibility among young people who play sport.

What is the purpose of the study?

To improve our understanding of any relationship between growth rate and flexibility during adolescent growth spurts in order help prevent related growth overuse injuries. This study also forms part of Doctorate study of Jennifer Strickland, PhD student

Do I have to take part?

No. Taking part is not mandatory. You do not have to give any reasons for not taking part. During the study you are free to withdraw at any time without it affecting anything.

What will be expected of you?

If you decide to take part your child as well as you will have to fill in consent forms. If you withdraw from the study any data collected will not be used.

The testing will comprise of your child's height, weight, leg girth size, and leg length being measured 3 times annually over 4 seasons, together with your child's leg muscle flexibility. These tests are similar to ones that they already do – we are just using the data specifically for this study.

How will the recorded data be used?

The data recorded will be mathematically analysed to help us see if there are any meaningful relationships that we can then use to see if some athletes will be more likely to get growth-related injuries. The data collected may be published in journals and may provide us with advice for your child's fitness and medical team to help prevent certain sports injuries. However, ALL data will be kept confidential and names will never be used.

Are there any risks?

No, you are not at any risk. All tests are standard anthropometric tests that your child is familiar with. And your investigators are all trained in working with children and supervised by health professionals.

Confidentiality

All data collected will be kept strictly confidential. Names will not be disclosed. Subjects will be assigned a number or code word instead to identify their data, and this identifying key will be kept in a separate file to their data.

If you require any further information, please to not hesitate to contact either

Jenny Strickland J.M.Strickland@gre.ac.uk 0208 331 8614 (PhD student)

Dr Mark Goss-Sampson M.A.Goss-Sampson@gre.ac.uk Senior Lecturer (Supervisor)

Appendix VI. Example Consent form – Football Academy Growth Study

PARTICIPANT CONSENT FORM

To be completed by the participant. If the participant is under 18, to be completed by the parent / guardian / person acting *in loco parentis*.

- I have read the information sheet about this study
- I have had an opportunity to ask questions and discuss this study
- I have received satisfactory answers to all my questions
- I have received enough information about this study
- I understand that I am / the participant is free to withdraw from this study:
  - At any time (until such date as this will no longer be possible, which I have been told)
  - Without giving a reason for withdrawing
  - (If I am / the participant is, or intends to become, a student at the University of Greenwich) without affecting my / the participant's future with the University
  - Without affecting any medical or nursing care I / the participant may be receiving.
- I understand that my research data may be used for a further project in anonymous form, but I am able to opt out of this if I so wish, by ticking here. ☐
- I agree to take part in this study

Signed (participant)	Date
Name in block letters	
Signed (parent / guardian / other) (if under 18)	Date
Name in block letters	
Signature of researcher J. Strickland	Date
This project is supervised by: Dr Mark Goss-Sampson	
Researcher's contact details (including telephone number and e-mail address): Jenny Strickland J.M.Strickland@gre.ac.uk 0208 331 8614 Mark Goss-Sampson M.A.Goss-Sampson@gre.ac.uk 0208 331 7986	

Appendix VII. Osgood-Schlatter's disease Case Series Paper

Original Article – submitted the Journal of Sports Rehabilitation, revised copy (July 2018)
Manuscript JSR: 2018-0050.R1

"A therapy treatment protocol for Osgood-Schlatter's Disease: case series"

Authors: J. Strickland, M. Colpus and M. Goss-Sampson

Abstract

Context: Osgood-Schlatter's Disease (OSD) is the most common overuse injury to affect growing children, yet there is no consensus as to the best treatment, and recovery can take months to years. A quicker and more effective intervention is needed.

Objective: The aim of this study was to assess the effectiveness of a therapy intervention on adolescent patients with OSD.

Design: Case series

Setting: Private physical therapy clinic in Kent, United Kingdom

Patients: 75 OSD patients (age range 8-17 years)

Intervention: Protocol using massage, stretching and rest from activity, depending on patient response to outcome measures.

Main outcome measures: Time to discharge was the primary dependent variable. Presence of pain at the patella tendon apophysis during a standing-quadriceps stretch and an eccentric wall squat was the secondary dependent variable. Onset time gave rise to patient sub-groups of acute, sub-acute and chronic. Follow-up interviews regarding pain and activity levels were obtained from 2 to 8 years post-discharge.

Results: Patients were discharged pain free on both tests in a median of 2.0 ± 1.0 weeks. Pain free stretch outcomes were achieved earlier in a median of 1.0 ± 0.7 weeks. There were significant differences in response between the acute and chronic groups (P =.01) with the acute achieving pain free active loading in 1.2 ± 0.8 weeks and chronic 2.4 ± 0.6 weeks. Follow up was achieved in 58 patients with three recurrences (5%). There were significant differences in long-term responses between chronic and sub-chronic (acute + sub-acute) groups with higher incidences of pain (82%:25%, P <.00) and lower levels of sports return (29%:58%, P =.28).

Conclusions: Results suggest this protocol is a simple, effective and non-invasive physical therapy intervention for OSD with recovery within 2 weeks, low recurrence rates and good long-term results. Clear clinical markers are used for monitoring progress.

Keywords: Pediatric, overuse injury, massage, apophysitis, knee, physiotherapy

INTRODUCTION

Osgood-Schlatter's Disease (OSD) is a chronic debilitating injury that affects young adolescents during their secondary growth period(Brukner & Khan, 2012). It is the most common overuse injury to affect this age group (Micheli, 1987) and incidence rates have been reported from 7% of the adolescent population to double that for young athletes (De Lucena, Dos Santos Gomes, & Oliveira Guerra, 2011), but within specialist sporting academies the rate has been reported as high as 25% (Price, Hawkins, Hulse, & Hodson, 2004). It has been described as a chronic overuse injury caused by traction of the apophysis of the patella tendon as it inserts into the tibial tubercle(Brukner & Khan, 2012) but the mechanisms behind the traction have yet to be identified. It has been suggested by some authors (Krause & Williams, 1990; Micheli, 1987) that the traction force causes repetitive microtrauma to the softer growing apophysis and results in chronic inflammation (apophysitis) . This manifests itself as a swollen and tender tibial tubercle and patients complain of pain and reduced function of the knee, especially during or after physical activity (P. Gholve, Scher, Khakharia, Widmann, & DW, 2007). Recovery from OSD is variable but resolution normally occurs with skeletal maturity in 1- 4 years (Gerulis, Kalesinskas, Pranckevicius, & Birgeris, 2004) although it has been reported that 60% of ex-patients continue to have ongoing symptoms and dysfunction into their adult lives (Krause & Williams, 1990),(Ross & Villard, 2003) and recurrence rates of 30% have been described (Kujala et al., 1985). It is therefore a common, debilitating and often chronic injury (Antich & Lombardo, 1985). In spite of over a century since it was first described, published literature is largely limited to descriptive studies of incidence and presentation or professional opinions on management (Bloom, Mackler, & Barbee, 2004). There is however no consensus as to the best treatment but conservative management is usually recommended (Bloom et al., 2004). Common medical advice is based on generic symptomatic relief using ice, medication for pain and inflammatory relief, (Brukner & Khan, 2012; Krause & Williams, 1990; Kujala et al., 1985; Vaishya, Azizi, Agarwal, & Vijay, 2016). Modifying activity levels to the patient's pain tolerance levels i.e. self-limiting, and waiting for skeletal maturity to develop is also commonly advised (Bloom et al., 2004; Brukner & Khan, 2012; Ehrenborg, 1962; Meisterling, Wall, & Meisterling, 1998; Vaishya et al., 2016). However as no prospective studies on this conservative approach have been performed (Bloom et al., 2004) there is no evidence of this approach being more effective than allowing the condition to heal slowly as described in its natural history (P. Gholve et al., 2007; Krause & Williams, 1990). Unfortunately resolution takes on average 21 months(Ehrenborg, 1962; Meisterling et al., 1998) and can have a considerable effect on the patient and even their future sports career (Reeves KD, Fullerton B, Topol G, 2006). Later surgery may also be required to remove persistent ossicles from within the patella tendon in 9 -12% of patients (Orava et al., 2000).

Only five intervention studies in OSD have been published to the authors' knowledge(Ehrenborg, 1962; Levine & Kashyap, 1981; Lohrer, Nauck, Schöll, Zwerver, & Malliaropoulos, 2012; J. Nakase, Numata, Oshima, Takata, & Tsuchiya, 2016; Topol et al., 2011). The studies investigated chronic patients with no intervention for acute patients described. Treatment selection was based on providing either pain reduction using plaster

casts(Ehrenborg, 1962), a patella strap (Levine & Kashyap, 1981) or dextrose/ lidocaine injections(J. Nakase et al., 2016; Topol et al., 2011); or by enhancing healing with shock-wave therapy(Lohrer et al., 2012). There does not appear to have been a rationale used for the basis of these treatments other than symptomatic relief. Objective tests were not used to assess the condition nor monitor its progression or final efficacy of the treatment. Stated recovery relied on the patients' self-reported pain levels as determined with a Pain Scale (Topol et al., 2011) and/or a VISA questionnaire (Lohrer et al., 2012; J. Nakase et al., 2016), but the two earliest studies(Ehrenborg, 1962; Levine & Kashyap, 1981) did not qualify how they determined success. Varied responses were described in these studies with success rates using pain scales reported from 66%(Ehrenborg, 1962) – 84%(Topol et al., 2011) and recovery time from 2 to 14 months(Ehrenborg, 1962; Levine & Kashyap, 1981) but only 3 of the studies(Ehrenborg, 1962; J. Nakase et al., 2016; Topol et al., 2011) used a control group for comparison, the others being case series design(Levine & Kashyap, 1981; Lohrer et al., 2012). Follow-ups ranged from 1 to 6 years with up to 28% of patients in one study(Lohrer et al., 2012) having changed their sport due to on-going problems, and limited information on functional return in the others. Overall there does not appear to be a consistent overall rationale for treatment selection for OSD nor consensus as to its form, and determination of efficacy relies on subjective pain self-reporting or professional opinion.

Traction forces:

OSD patients present with a number of typical features including shortened quadriceps muscles (De Lucena et al., 2011; Junsuke Nakase et al., 2015) and clinical observation by the main author also noted pain at the tibial tubercle upon passive stretching. Patients also tended to have high physical activity levels and pain during or after exercise (Antich & Lombardo, 1985; P. Gholve et al., 2007; Jayanthi, LaBella, Fischer, Pasulka, & Dugas, 2015; Krause & Williams, 1990) . These forces could contribute to the traction mechanism behind the injury[1,2,5]. The authors suggest that these could be further categorised into passive and active traction forces. Passive and active stress tests are commonly used as part of clinical assessments to indicate injury severity and healing response (Hayes & Petersen, 2003) therefore they could also be used to ascertain the loading tolerance of the apophysis, and thus progression and healing response in OSD patients. Reducing or eliminating the traction forces on the tendon should also facilitate healing.

Passive forces

Stretching would normally be recommended to improve flexibility and reduce passive tension but in this situation, it is counter-productive, as it would further increase the traction of the apophysis, eliciting pain and possibly exacerbating the condition. An alternative method using massage which does not compromise the apophysis site, could be used, and massage has not been previously investigated as an intervention for OSD to the authors' knowledge. Myofascial release massage (St. George, 1989) (MRM) has been used effectively within physical therapy to lengthen tight myofascial units in many conditions, most notably with ilio-tibial band syndrome(Fredericson & Weir, 2006). Once the tolerance had improved to enable a quadriceps stretch with no pain felt at the tibial tubercle, then normal stretching could safely commence

instead of massage, to continue reducing passive tension on the apophysis and increasing flexibility further.

Active forces

Moderating active loading is usually advised as part of the standard approach to OSD treatment as a way of self-managing provocative activities(Gerulis et al., 2004; Meisterling et al., 1998) , but of itself appears not to be sufficient, as the described resolution still takes many months to years(Ehrenborg, 1962; P. Gholve et al., 2007; Meisterling et al., 1998). Total immobilisation of the knee using plaster casts had limited success (Ehrenborg, 1962) but physical activity levels in patients could be minimised. This would allow time for tissue healing and avoid the stronger traction of the tendon during intermittent physical activity. It would be counter-productive to address only one aspect of the tensile loading on the apophysis i.e. passive but not active. Active loading could be objectively tested using a closed chain squat which increases the force in the quadriceps as the knee flexion angle increases and the patient descends eccentrically (Escamilla, 2001). Wall squats are a variation that have been recommended in the rehabilitation of OSD(Meisterling et al., 1998).

Alternative treatment model:

There is a need for a quicker and effective treatment that responds to the clinical signs of the OSD patient and provides a logical model for intervention selection and monitoring progress, based on the passive and active force tolerance of the apophysis. Progress can be objectively monitored to give patients and practitioner on-going feedback as to the treatment's efficacy. Considering the high prevalence of OSD and lack of studies, it is important to consider clinic-based interventions where most patients are likely to be treated. Therefore, the aim of this paper is to present a therapy treatment protocol for OSD based on reducing passive and active tension on the apophysis.

METHODS

Design: Case series

Setting: Assessment, treatment and evaluation were completed in a private sports clinic in Kent, United Kingdom by experienced Chartered Physiotherapists (Physical Therapists).

Patients: 75 OSD patients were either self-presenting or referred by medical practitioners to the clinic for physiotherapy treatment, which was paid for privately or by health insurance. Every successive OSD patient who attended for treatment was invited to try the protocol as opposed to the general symptomatic advice, and parents/ guardians and the patients themselves gave informed consent. All patients volunteered to receive the new protocol. Inclusion criteria was based on initial diagnostic findings of a tender and enlarged tibial tubercle, pain at the apophysis on or after physical activity, and typical age of early adolescence. Patients could present with a unilateral or bilateral condition. Exclusion criteria was any concurrent non-related injury. Patient demographics were recorded as well as physical activity levels prior to onset, and time from the onset of their condition, which gave rise to sub-groupings of acute, sub-acute and chronic.

Ethical approval was gained by both the local area Health Authority (National Health Service) and University of Greenwich Ethics Committee.

Outcome measures: Time to recovery was the primary dependent variable (DV) used (DV1) and was determined by pain free completion of the passive and active tests (secondary outcomes DV2 and DV3), as assessed by the therapist, and pain free activities of daily living as reported by patient and parent. Baseline self-reported numerical pain scores for activities of daily living were recorded at the beginning of each clinic visit for overall feedback, using a simple numerical VAS pain scale (Williamson & Hoggart, 2005), however for the secondary outcome tests a simple threshold of onset of any pain (Yes/No) was used. Quantity of pain was not considered due to subjective differences between patients and ethical considerations, so presence or absence of pain was used. A dual approach was used to approximate passive and active tensile forces and two secondary tests were used to assess the effect of those forces on the apophysis. All tests were assessed and scored by the therapist, and the results of the tests determined the specific intervention selected for the treatment protocol (IV1) (Table 1) and any decision to progress was taken by the therapist.

Test	**Result**	**Intervention**
Quadriceps stretch (DV2)	Painful (FAIL)	Massage daily (IV2a)
	Pain free (PASS)	Stretch 3 x day (IV2b)
Eccentric Wall Squat (DV3)	Painful (FAIL)	Rest (IV3a)
	Pain free (PASS)	Return to physical activity (IV3b)

Table 1. Summary of treatment protocol.

Passive test: Firstly a standing quadriceps stretch(St. George, 1989) (Figure 1) was used to assess passive load on the apophysis (DV 2), with the test immediately stopped at onset of any pain, and not allowed to continue. Any pain felt at the tibial tubercle during the test resulted in a fail score, no pain with a full stretch range completed was given a pass score if they could repeat it twice. Technique was monitored to ensure the hip remained in neutral and the knee placed alongside the standing knee [25].

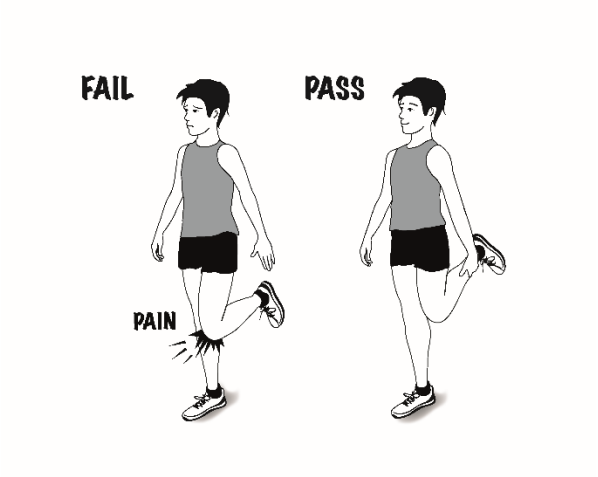

Figure 1 Quadriceps stretch test in standing

Passive intervention:

If the passive test was failed the primary intervention was massage (Independent variable IV2a), which is a key skill of physical therapy training. This specific intervention was myofascial release massage performed once a day to the rectus femoris muscle belly of the quadriceps for 2 minutes. This was performed with firm anterior-posterior pressure as tolerated by the patient, in a longitudinal proximal to distal direction with a small amount of massage oil to assist and improve comfort for the patient. Parents were instructed by the trained physical therapist researcher in the application of the massage technique, so they could carry on the treatment at home every day, thus enabling continuity between subsequent clinic visits, which were usually weekly. Parents demonstrated the technique on their child under the researcher's supervision and the patient gave feedback to the similarity of massage technique between the researcher and the parent, to improve consistency of its application

Once the quadriceps stretch position was pain free (pass) then massage was stopped and replaced with stretching instead (IV 2b). A stretching routine using the same stretch test position (Figure 1) was then performed 3 times daily by the patient, each stretch held for 10 seconds and repeated 5 times (St. George, 1989).

The days to pain free stretch were recorded.

Active test:

Secondly an eccentric wall squat(Meisterling et al., 1998) (Figure 2) for active loading (DV3), was performed to the steady count of 5 seconds. Any onset of pain felt at the tibial tubercle during the eccentric (downward) phase the test was immediately stopped and the patient returned to upright stance, and the test was deemed a fail. As eccentric actions (downward phase) require stronger muscular loading than concentric[27] (upward phase) it was deemed sufficient to only score the eccentric section of the wall squat. If the patient could complete both the eccentric phase to the lowest possible position, and return concentric phase over 10 seconds, and repeat twice without any pain at the tibial tubercle, the test was given a pass score.

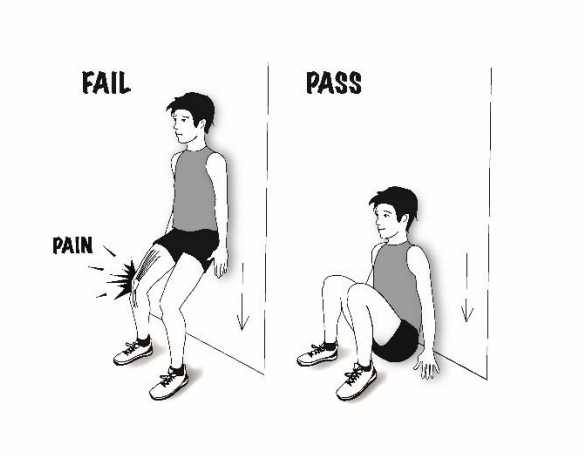

Figure 2 Active Wall squat test

Active intervention:

Failure of the eccentric wall squat meant patients were instructed to rest completely from physical activity other than necessary walking (IV3a). Once this wall squat was pain free and full decent to the floor and return assent was achieved (pass) then the return to normal physical activity could resume (IV3b). The days taken to this point were also recorded.

Discharge:

The dual-approach (IV1) was completed when both tests were pain free as judged by the therapist, and patients were judged as recovered and discharged, and the treatment successful. Patient pain scores were also used for an overview of their home experience of activities of daily living, and parents reported their own observations for verification. In line with common physical therapy practise, on-going discharge advice on general stretching and monitoring was given to each patient to further support their full return to sport. This advice included quadriceps strengthening using the wall squat, and later more explosive exercises such as jumps, sprinting and kicking.

Treatment groups were defined as either fully-compliant (FC) or partially-compliant (PC) or unable to comply (UC). Compliance was assessed as either fully-complaint or partially-compliant dependent upon strict adherence to the protocol as judged by parental and patient feedback.

Follow-up interviews were recorded from 2 - 8 years via phone call or subsequent clinic visit, and incidents of recurrence of their OSD (i.e. further pain at the site), level of return to sport or activity, and ability to kneel without pain were recorded. Patients were asked to assess their level of sport as whether it was of the same standard, or higher or lower than their previous level prior to their OSD.

Statistical analysis:

A case series study was used to evaluate the passive interventions of massage and stretching (IV2a and IV2b) on passive stretch test (DV2), and active intervention of rest (IV3) on the active wall squat test (DV3); and of the treatment protocol (IV1) on the overall recovery time of OSD patients (DV1). Descriptive statistical analyses were used to describe the patient profiles and sub-group comparisons, and their overall response to treatment (primary DV). Differences in passive (DV2) and active (DV3) outcome responses to treatment for the patient compliance groups, was analysed using non-parametric testing (Mann-Whitney), and across and between sub-groups (Kruskal-Wallis and Mann-Whitney) for significance. Pearson's Chi-Squared tests were used to assess relationships between onset sub-group and long-term recovery in the follow-up. SPSS 25 (SPSS Inc, Chicago, Il, USA) was used to analyse the data and all analyses were performed to a significance level of $P<.05$ and 95% confidence interval.

RESULTS

A total of 75 OSD patients were seen over a 13-year period by the main researcher and her Physical Therapy colleagues. A total of 77% of patients had already followed the standard symptomatic advice from other health professionals, involving rest, ice, and analgesic/anti-inflammatory medication[1,5,9,12], and had not improved. All patients had entered their adolescent

growth spurt as confirmed by parental observation. Boys outnumbered girls by 3:1 and their ages and anthropometrics are summarized in Table 2. This data was normal in distribution.

Table 2 Demographics (means and standard deviation)

Gender	Age at Onset (years)	Height (m)	Mass (kg)	BMI (%)
Girls (n=18)	10.59 ± 1.30	1.51 ± 0.13	46.0 ± 11.4	19.8 ± 3.1
Boys (n=57)	12.20 ± 1.40	1.58 ± 0.10	51.2 ± 11.9	20.4 ± 3.7

The median time from onset to clinic presentation was 3 months (range 1 week to 2 years), but this median was calculated with four outliers of between 2.5 and 7 years removed. Onset time data was divided into 3 categories of acute (< 1 month), sub-acute (1 to 3 months) and chronic (more than 3 months). There were 12 acute, 17 sub-acute and 46 chronic patients in the 3 groups (Figure 3), and the gender ratio of 3 boys :1 girl was the same throughout the categories.

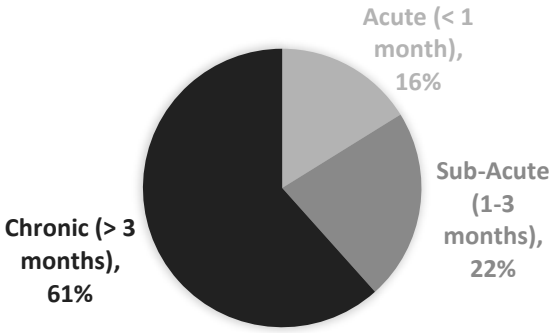

Figure 3 Osgood-Schlatter Disease patients on presentation

Bilateral presentation occurred in 52% of patients, with a small majority of patients (54%) most affected in their non-dominant leg. Hours actively engaged in sport in a normal week pre-injury was a mean of 14.2 ± 5.1 hours/ week, and 44% of the patients played soccer as their primary sport, with 14% swimmers and 10% gymnasts.

Treatment response: Patients were classified as fully-compliant (FC), partially-compliant (PC) or unable to comply (UC). 51 patients (68%) followed the protocol exactly as advised (FC) but 14 patients (19%) did not adhere to either the daily massage or the complete rest element of the protocol (PC). The FC group were further analysed by onset time at presentation. There were 5 patients who were excluded from the final treatment response analysis due to other factors having a possible impact on their recovery including other injuries affecting the area. Unfortunately, 5 patients also failed to attend their last appointment, so their final outcome was

unknown. These 10 patients were deemed unable to comply (UC). Therefore, the overall treatment response and time to discharge, in 65 patients, can be seen in Figure 4.

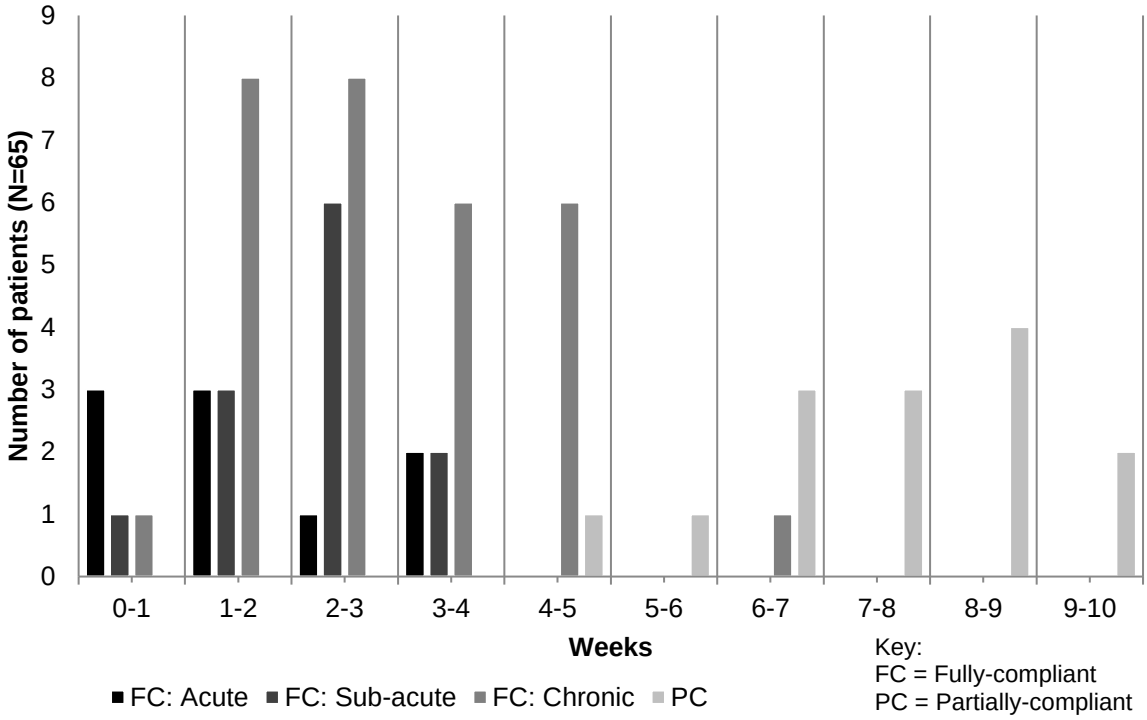

Figure 4 Treatment response (time to pain free discharge).

Passive:

All patients failed the quadriceps stretch (DV2) on initial testing therefore all patients started on the primary passive intervention of massage (IV2a). However, 3 patients reported no pain on stretch when it was repeated after the first massage application, and 57% of patients were pain free on the stretch test after 1 week, with 88% pain free by 2 weeks. The test was only regarded as a pass if patients could repeat it twice without pain. Patients then progressed onto the second passive intervention (IV2b) of stretching instead of massage (IV2a). The median was 1 week (range 0-4) to achieve the pain free quadriceps stretch, irrespective of whether patients were in the FC or PC group.

Active:

All patients failed the eccentric wall squat on initial testing, but this component took longer to recover fully than the passive test.

All FC patients achieved a full and pain free active wall squat in an overall median of 2.0 ± 1.0 weeks with the PC patients 7.0 ± 3.0 weeks (Figure 4). Differences between these cohorts were significant (Mann Whitney *U* test, *P* = .014) with a Post-hoc large size effect of .703. Within the FC cohort there was a significant difference in final treatment response across the sub-groups (Kruskal-Wallis *P* = .04), but non-significant differences between the groups except for between

the acute and chronic where the difference was significant (Mann-Whitney *U* test, *P* =.01) (Table 3). Post-hoc analysis showed this had a medium size effect.

Table 3. Group and sub-group response to treatment.

Compliance Group	Median Recovery time (weeks)	Sub-group	Median Recovery time (weeks)
FC (all)	2.0 ± 1.0**	Acute	1 ± 0.8 ^^
		Sub-acute	2 ± 1.4
		Chronic	2 ± 0.7 ^^
PC	7.0 ± 3.0 **		

Key: ** Significance P=.014; and ^^ P = .01 (Mann-Whitney U)

No patients were able to complete the wall squat before the passive stretch therefore all patients were treated with both massage and rest initially.

Discharge:

All patients passed the passive component before the active, therefore achieving the full wall squat signalled final recovery and discharge. The initial self-reported numerical VAS pain scale across the cohort was a median of 7 ± 1.8 (out of 10) but receded by the second clinic visit down to 1 ± 2.8, with a final outcome of 0 for all patients. 50% of patients reported no pain during daily home activities by the second clinic visit. This numerical pain score was used for an overview and feedback of the patients' daily experience rather than specific experimental variable.

Follow-up interviews were achieved in 58 patients (77%) with a mean of 4 years (range 2-8) since discharge, with 17 patients unable to be contacted. Three patients (5%) had a recurrence of their OSD and were all part of the partial compliance group. Return to sport was achieved at a higher level overall in 41% of patients or the same level in 29%, however further analysis showed 80% of acute returned to higher levels whereas 43% sub-acute and 29% chronic (see Figure 5.) Four patients gave up sport entirely, one sub-acute and three chronic, two of the latter had suffered the condition for the longest times of 36 and 84 months. Statistical analysis showed a strong relationship between onset time and level of return to sport (Pearson Chi-Square χ^2 (1) = 4.85, *P* = .028). Patients in the non-chronic groups (acute and sub-acute) had a calculated odds-ratio of 3.4 times observed higher level of future sports than the chronic patients.

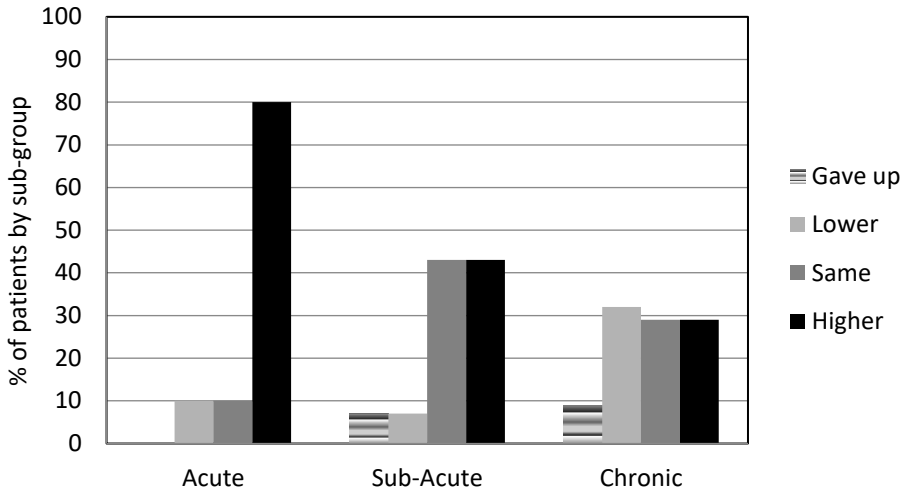

Figure 5 Sport level at follow-up

Ongoing painful kneeling was reported by 58%, most of whom were in the chronic group. Again, there was a difference between the groups with 90% of the acute and 64% of the sub-acute groups remaining pain free when kneeling or playing sport, compared to only 18% of the chronic. These differences were significant between the acute and sub-acute compared with the chronic (Mann-Whitney U P <.00 in both cases) but no difference between the acute and sub-acute (Mann-Whitney U P=.16). There was a strong relationship between onset time and long-term pain if you were a chronic patient compared with acute or sub-acute (Pearson Chi-Square χ^2 (1) = 19.08, P <.00). This represented a calculated odds-ratio of 14 times observed future pain in the chronic group compared with earlier intervention groups. This meant that although 82% of the chronic group still complained of pain nearly all (91%) continued to play sport.

DISCUSSION

Treatment response:

The most important finding in this study is the rapid improvements in both passive and active loading tolerance, and time to recovery and discharge. FC patients were pain free in both tests within a median of 2 ± 1 weeks from the treatment start. Resolution of pain, function and return to physical activities in 2 weeks compares favorably with the literature which describes many months to years as the natural history (Krause & Williams, 1990). It cannot be compared directly to a standard approach as there have been no published studies nor is there consensus on that standard advice (Bloom et al., 2004; Vaishya et al., 2016). The results do however compare favorably with the previous intervention studies mentioned where response time was described in months (Ehrenborg, 1962; Levine & Kashyap, 1981; Lohrer et al., 2012; J. Nakase et al., 2016; Topol et al., 2011) and the natural history (Krause & Williams, 1990). Those patients who were only partially-compliant still recovered in less than 2 months with a pain free passive test achieved by week 3 ± 1 (Table 3). The patients in this study were pain free on clinical testing and discharged within weeks with a significantly faster response in the fully-complaint cohort.

Recovery was only confirmed at set clinic appointment times and some patients may have been able to complete the tests days before, so the resulting time scales may be under-reported.

functional

Analysis of the onset groups showed the earlier the treatment started, the quicker the recovery. Although there were not significant differences between the acute and sub-acute responses, there was a significant difference between the acute and chronic indicating that the earlier treatment started, the quicker the recovery. However, the chronic group still responded well with passive stretch pain free by the second week and active wall squat by the third week. There were two large outliers amongst the chronic group with one patient having suffered for over seven years and another for three years, but they still responded well recovering in less than three weeks. It should be noted that exact time scales from onset could be affected by the accuracy of memory recall and isolating the precise onset of an overuse injury which, by its nature, is gradual, therefore both parents and children were asked when symptoms first started and not when they became intolerable. It is likely that onset times and length of suffering could have been under-reported. The median of 3 months from onset meant that the majority of patients were chronic (61%). All the chronic group had already undertaken the standard advice and tried a combination of rest and symptomatic relief with poor results (pain and dysfunction), hence their referral for physical therapy. This chronic group therefore could be viewed as their own control group with standard treatment for the first three months (poor response), then alternative intervention in this study (good response in 2 weeks).

Monitoring progress:

The ability to monitor the patients' progress is an important feature of this protocol. It allows for immediate feedback to patients and parents, which may improve their engagement and adherence. It also highlights if there may have been problems with that adherence if response was slower than expected. All patients failed both passive and active tests at the beginning and passed both by the end. No other studies have monitored or assessed progression in the treatment of OSD, nor used a functional test to determine final treatment response. However the most recent intervention study incorporating saline or dextrose injections into the patella tendon insertion and associated fat pad (J. Nakase et al., 2016) used a VISA knee questionnaire (Visentini et al., 1998) to assess recovery, and two used the same questionnaire to assess long-term efficacy (Lohrer et al., 2012; J. Nakase et al., 2016). The injection study did show significant improvements in both groups over 3 months[19], but no difference between the groups, so whist efficacy appears to be shown, its rationale is unexplained. Our results show patients had no pain in less than 3 weeks which appears to show a quicker return than any of the above studies, but more information would have been gained from using a comprehensive knee score questionnaire and this has been noted as one the limitations of this study. Pain thresholds in young adolescents may be affected by physical and hormonal stressors during puberty(Patton & Viner, 2007), and accuracy of describing pain levels will depend partly on experience but also on vocabulary – both of which may be limited in the younger patient. For this reason onset of

pain was used during testing rather than pain tolerance, and a simple numerical scale for feedback for overall pain (Williamson & Hoggart, 2005).

Passive:

The passive test was also used to determine treatment selection i.e. when massage was needed or when stretching could safely recommence and was a key element in proving positive progress in treatment response. All patients started with having daily massage at home as all had a fail test on the quadriceps stretch. The differences in passive response between the onset groups was not-significant as they all showed an improvement in pain reduction by the second clinic session, with 65% being pain free in this time. Most patients had already been resting as advised by their doctors so the only change in intervention was the daily massage. The massage appeared to affect their passive test first as this was the first outcome to be resolved in all patients. It could be that the massage therefore directly reduced the passive tension in the quadriceps muscle thereby reducing the traction force on the apophysis. This would reduce the pain on the passive test and increase the load tolerance of the apophysis, but more research would be required to explore the effects of massage directly on muscle tissue. Once the stretch position was pain free patients were able to start a stretching regime to continue improving their quadriceps flexibility and keep the passive tension to a minimal level, as demonstrated by a full stretch position (De Lucena et al., 2011; Junsuke Nakase et al., 2015; St. George, 1989). Maintenance of good flexibility[25] could be a key component in the control and ongoing maintenance of passive tension[32] and may have impacted on the low incidence of recurrence found in this group. No other studies have either assessed or monitored passive muscular tension as part of the treatment for OSD, yet some descriptive studies have observed its presentation as reduced flexibility(De Lucena et al., 2011; P. A. Gholve, Scher, Khakharia, Widmann, & Green, 2007; Micheli, 1987) or hypothesised its possible role as a risk factor(Junsuke Nakase et al., 2015). This is the first study to directly address the observed painful stretch position and treat it with massage initially, then later with stretching. Progression was able to be monitored by the patients' pain response and feedback regarding the ease and range of the stretch.

Active:

Active loading of the apophysis as demonstrated by the wall squat, was achieved by patients a week later than the passive stretch test. The ability to perform a progressive eccentric loading exercise(Escamilla, 2001; Meisterling et al., 1998) gave valuable feedback to the patients themselves as they were able to see and feel the progression of their own recovery as they descended further before pain onset. This also gave confidence to patients that the treatment was working, and their condition was improving, and may have improved compliance and engagement. However, in this study only a pass/ fail judgement was made based on any pain during the wall squat plus full descent. This gave a clear indicator for the patients' ability for their apophysis to tolerate active loading and thus determine the decision to start the patients return to physical activity and sport, and discharge from treatment. Limitation of exercise to reduce active tension was also a key component of this protocol. There is little point of reducing

one loading force whilst allowing another to continue, especially as physical activity has the potential to create greater forces on the apophysis than passive stretching (Hayes & Petersen, 2003). This may have been reflected by every patient's passive tolerance being resolved before their active tolerance. Minimising rather than eliminating physical activity levels by immobilisation such as by plaster casting, reduces the active tension forces promoting healing yet still allows for some movement to avoid contractures or atrophy of muscles(Ehrenborg, 1962; Gossman et al., 1982). Immobilising the limb may in fact cause further passive tension in the muscle due to the child's growth whilst in plaster, which may explain the high recurrence reported with this method(Ehrenborg, 1962; Jakob, von Gumppenberg, & Engelhardt, 1981). Minimising activity is a practical imposition for active teenage patients to contemplate(Reeves KD, Fullerton B, Topol G, 2006) especially when patients present bilaterally, and can be rapidly adapted as the patient improves.

Massage:

The effects of massage as an intervention have not been described before and whilst this study does not examine the specific effects of massage on the muscle tissue, we can report on its effect on the symptoms of OSD. Previous research on the use of MRM indicates it can have an effect on the lengthening of muscle-tendon units (Fredericson & Weir, 2006; St. George, 1989) and this may be the mechanism for reducing the passive tension element observed in OSD patients. In this study the MRM intervention results in a rapid improvement in passive stretch tolerance and pain reduction in our patients. The possible reduction in this background tension appears to be an important first step in lessening the traction force on the apophysis, thereby allowing it to repair better. Further understanding of the underlying mechanisms of MRM, in particular with respect to its effect on myofascial length and pain reduction, should be investigated.

Follow-ups from 2- 8 years have determined sustained long-term results with 70% returning to sport at the same or higher levels. However, there was a clear difference between onset groups with the acute group achieving 80% higher return, sub-acute 43% but chronic only 29%. Most patients (58%) continued to have pain when kneeling or playing sport which is similar to previous findings[5,8], however in this study further analysis showed large differences between the onset groups with only 10% of acute, 36% sub-acute but 82% of chronic patients having on-going persistent pain at follow-up. This suggests that the earlier the intervention the fewer long-term problems are experienced. Chronic conditions may have permanent bony changes and prominent tibial tubercles which could explain the on-going problems with kneeling. There were three reported recurrences (5%) in the follow-up group, all who admitted they had stopped their stretching regimes. The low recurrence rate compares favorably with one study's rate of 30% [9]. The significant long-term observed responses showed significantly lower pain incidence and better sports return in the acute and sub-acute patients. This indicates that earlier treatment also has longer term benefits and reports the sustained positive effects of early and effective treatment.

Limitations

One limitation to this study was in its design as a case series. A control group was not included as there is currently no standard alternative to use for comparison (Bloom et al., 2004; Reeves KD, Fullerton B, Topol G, 2006; Vaishya et al., 2016). Traditional scientific study design may also not be valid because of the multi-factorial presentation and the multiple interventions, which changed according to the individual's progression, not on pre-determined time scales or randomisation. Without a control group we understand we cannot claim that this protocol is more or less effective than another, but we can present the results of recovery time which can be compared with the literature, and progression of the treatment response which no other paper has published. Severity or staging of OSD was not recorded in this study and this could be an additional factor in recovery times. This was also a self-presenting group with a soccer bias, and they were fee-paying patients, so they may not truly reflect the general population of this age cohort. They may also have had a different motivation or expectation of results than a non-paying population may have had[34], however we would argue that most parents and patients wish to be rid of pain in the shortest possible time, irrespective of finances. Using a specific knee function questionnaire during the study may have yielded more accurate understanding and would a recommendation for future studies.

These results show that the authors' treatment protocol is successful in treating OSD patients. In particular massage appears to target the passive component of muscle tension quickly and effectively across all onset groups, giving discernible pain reduction and allowing for stretching to commence. The overall result is a short recovery time in all patients from a potentially chronic and debilitating injury, but with the quickest response in the earliest treatment group. The protocol may also provide a useful model for the treatment of other apophysitis syndromes and their treatment strategies. The use of clinical tests allowed for clear monitoring of progress and determination of progression and could be easily applied by the therapist. This study also highlights the importance of clinicians using their skill set and applied clinical reasoning to solve problems. It can be applied simply and consistently with the limited resources available to primary care health practitioners such as physical therapists or athletic trainers. With this protocol, parents and patients were also able to take ownership, empowering them to help control their own recovery and monitoring the progress of the injury.

CONCLUSIONS

There are very few studies into the treatment of OSD, and no consistent approaches exist to justify current treatments and their efficacy, nor is there consensus as to its format. This paper presents the first massage-based treatment protocol using observed signs in the presentation of OSD, which enables a logical method that was successful, effective and quick within an everyday clinic setting. The protocol also provides for the first time, simple clinically-based tests for monitoring the progress of the patient and enables clarity in the selection of treatment intervention. This case series shows that most OSD patients recovered from their signs and symptoms in 2 weeks, with short and long-term success, and very low recurrence rates. Both the treatment and monitoring were non-invasive and cost-effective requiring the minimum of resources.