Sever's Disease: What Does the Literature Really Tell Us?

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Background: Sever's disease is typical of many musculoskeletal conditions where observational annotations have slowly been accepted as fact with the passing of years. Acceptance of these nontested observations means that health professionals seeking information on this condition access very low-level evidence, mainly being respectable opinion or poorly conducted retrospective case series.

Methods: A comprehensive review of the literature was undertaken gathering available articles and book references relating to Sever's disease. This information was then reviewed to present what is actually known about this condition.

Results: Respectable opinion and poorly conducted retrospective case series make up the majority of evidence on this condition.

Conclusion: The level of evidence for most of what we purport to know about Sever's disease is at such a level that prospective, well-designed studies are a necessity to allow any confidence in describing this condition and its treatment. (J Am Podiatr Med Assoc 98(3): 212-223, 2008)

Sever's disease, also known as calcaneal apophysitis or calcaneoapophysitis, was first described by Haglund[1] in 1907, although Sever[2] has received more credit for describing the condition. Sever's disease has been described in various ways and attributed to a variety of causative factors. None of these causative factors has been tested prospectively and, where tested, none of the measurements has been carried out systematically, and reliability or validity of the measurements has not been considered. We present the views of authors who have written about Sever's disease and examine the strength of their observations. We conclude with the direction we believe research into this condition should progress.

Sever's Disease Defined

Sever's disease is an inflammation of the calcaneal apophysis. The calcaneal apophysis is a cartilaginous growth center onto which the Achilles tendon inserts.[3-5] Kvist and Heinonen[6] and Kim et al[7] refine this definition, adding that Sever's disease is a traction epiphysitis as opposed to other forms of inflammation at this site, such as bruising or infection.

Presentation of Sever's Disease

The generally accepted clinical picture of Sever's disease is that of an active male child 10 to 12 years old, often presenting at the beginning of a sport season, experiencing a growth spurt, and experiencing pain over the apophyseal area of one or both heels.[6, 8-11] The child will generally have limited ankle joint dorsiflexion and an underlying biomechanical deformity of variable nature, and increased activity will worsen the pain.[2, 8-16] The pain is often severe enough that at the end of physical activity the child will limp to take weight off the affected heel.[2, 8-11]

All of these traits, however, arise from anecdotal and observational surmises of authors[2, 11-13] or from data that have involved multiple practitioners' assessment and retrospective analysis of notes. These retrospective data may have been collected up to 10 years before publication.[6, 8-10, 15, 16] No authors have recorded the onset time of the discomfort during sports activity. There is no mention in any of these

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articles of the methods used to measure the biomechanical variables. Reliability or validity of methods used to obtain the ankle joint dorsiflexion or biomechanical malalignment data are not commented upon, thus reducing the quality of the data. Although pain and limping are mentioned as symptomatic traits,[2, 6, 17] there have been no attempts to quantify the pain or its effect on the individual. Table 1 summarizes the presentation demographics from the literature.

The location of the painful area is variably reported. Sever[2(p1025)] reported a clinical picture of "tenderness about the posterior aspect of the heel, low down." This corresponds to the apophyseal area located at the postero-plantar aspect of the calcaneus.[3, 8, 9, 11, 13, 14, 17-23, 25-33] Meyerding and Stuck,[18] basing their clinical picture on 21 cases retrospectively reviewed and on many other authors, with no study patient population mentioned,[4, 12, 34-39] describe the pain as being located posteriorly on the heel, which suggests a high likelihood of Achilles tendinitis or achillobursitis as a diagnosis as opposed to apophysitic pain.

A high activity level has been reported in symptomatic individuals, although it has not been specifically measured. Haglund[1, 40] titled his paper "Concerning Some Rare but Important Surgical Injuries Brought on by Violent Exercise," but he provided no justification of how violent the exercise was. Many authors[2, 6, 8, 10, 18, 19, 28, 33, 41-43] have noted anecdotally that affected children were active and vigorous, and that symptoms often presented at the beginning of the sport season and while the child was undergoing a growth spurt. However, none of these studies measured or reported the children's actual activity levels or evidence for growth spurts. Many of the larger studies sourced their subjects from sports medicine clinics or had authors who were physicians in sports-related disciplines. Data from sports medicine clinics introduce the potential bias that subjects are likely to be active people involved in sport.

Several authors have noted more-common sports among those with Sever's disease, but the specific sports implicated vary from study to study. Orava and Puranen,[19] in their prospective assessment of a Finnish child and adolescent sports-playing population, found that mainly children involved in track and field activities presented with Sever's disease. McKenzie et al,[8] in a retrospective study of 20 Sever's disease cases, found that in all of these cases, the child was involved in running-related sports, mainly track and field and soccer. On average, participation was four times per week, in addition to any physical activity engaged in as a part of school. Micheli and Ireland[9] found 29% of

Table 1. Summary of Articles on the Presentation Demographics of Sever's Disease

Study	Subjects with Sever's Disease	Ratio of the Percentage of Male to Female Subjects with Sever's Disease	Incidence of Bilateral Occurrence (%)	Age of Subjects (y)
Meyerding and Stuck[18]	21	19:2	NR	Average, 10.2; range, 7.5–17
Krantz[14]	36	28:8	NR	Mainly 9–11
Orava and Puranen[19]	24	22:2	NR	NR
McKenzie et al[8]	20	14:6	In 80	Average, 11.2
Orava and Vitanen[20]	NR	6:1	NR	NR
Micheli and Ireland[9]	85	64:21	In 61	Majority 10–12
Szames et al[15]	53	40:13	In 49.1	Average, 10.7 (male), 11.7 (female)
Kvist and Heinonen[6]	67	36:31	In 15	Average, 12.5 (male), 11.8 (female)
Fehlandt et al[21]	47	31:16	NR	Mean ± SD of 12.4 ± 2.4 (female), 12.3 ± 2.2 (male)
Micheli and Fehlandt[10]	92	64:28	NR	12.3 in boys, 11.8 in girls
Rozenblat and Bauchot[16]	68	25:43	In 69	Average 10 y, 10 mo; 11 y, 5 mo in boys; 10 y, 6 mo in girls
Liberson et al[22]	35	31:4	In 46	NR
Volpon and de Carvalho Filho[23]	69	All boys	Unilateral	6–15
Price et al[24]	57	Not stated, but assumed boys as soccer academy sample	NR	11–19

Abbreviation: NR, not reported.

the 85 patients with Sever's disease in their study were involved in soccer, with basketball, gymnastics, and running being the next most common sports. Micheli and Fehlandt[10] in their 10-year retrospective study of children with Sever's disease in the same sports medicine clinic as Micheli and Ireland[9] and over a similar period, found running and soccer to be the most common sports for male patients and gymnastics and soccer to be the most common sports for female patients. Kvist and Heinonen[6] found that 73% of 67 Sever's disease patients participated in track and field and running (with 90% of the runners specializing in middle and long distance). Twenty-four percent of subjects from this study were engaged in fewer than 5 hours per week of sports activity; 44% were engaged in 5 to 10 hours of weekly sports activity; 21% engaged in 10 to 15 hours; and the remaining 11% engaged in more than 15 hours of sports activity per week.

Rozenblat and Bauchot's[16] Sever's disease population consisted of more female than male patients. The most common sports were therefore different from those in many of the other studies. Two-thirds of the subjects practiced artistic gymnastics with an average of 11 hours of training per week. These findings suggest a strong link between Sever's disease incidence and the amount of activity undertaken. However, as there was no comparison to an asymptomatic population, these findings are less conclusive than they could be.

Kvist et al[44] did compare activity levels of children with calcaneal pain to those without but failed to identify if the pain was indeed Sever's disease. The authors found that active children were more likely to suffer from calcaneal pain than those children who had lower levels of activity. They used a nonvalidated retrospective questionnaire completed by the subjects' parents. As such, there was no testing to ascertain if parents accurately reported the pain of their children. Because of the lack of identification of Sever's disease and the reliability issues involved with parents accurately answering questionnaires for past conditions, their results should be viewed with some caution.

Kvist et al[44] offer no explanation for certain sports possibly being linked to the development of Sever's disease more than others. The studied populations' common sports—along with their training techniques, training surfaces, and weather—differed, so a definitive extrapolation may not be possible.

Katz[45] and Gregg and Das[46] both reported the symptom of pain on initial weightbearing after rest. Lewin[12] and Santopietro[42(p574)] both described "swelling and induration" of the heel in severe cases, but these are not mentioned elsewhere in the literature. None of these studies mentioned a patient population upon which these characteristics were based.

Existence of Sever's Disease Challenged

Some authors question the existence of Sever's disease. Ferguson and Gingrich[47(p90)] stated that in 25,874 patients, "no case has been seen of swelling centering about the apophysis or involving symptoms related to this area." Although their sample size is impressive, their methodology raises a number of concerns. The central concern is that the authors used only radiographs to reach this conclusion. They did not provide details on whether the large sample size involved symptomatic subjects or if any subjects had been diagnosed with Sever's disease. Ferguson and Gingrich[47] did not address the large sample size and the questions it raises about the number of investigators required to examine so many heels and the subsequent interrater reliability issues.

Brower[48] also questions the existence of calcaneal apophysitis. She attributes pain in the heel in children to plantar fasciitis, Achilles tendinitis, or retrocalcaneal exostosis without giving any justification for this point of view except clinical experience and the fact that x-ray diagnosis is inconclusive.

Ogden et al[5] dispute the involvement of the apophysis in symptomatic traits, on the basis of a study involving 14 subjects who did not respond to conservative treatment. All subjects were described only as having heel pain rather than giving a specific diagnosis of Sever's disease. The subjects' feet were immobilized in plaster casts or removable ankle-foot orthoses for 3 to 4 weeks as part of their treatment. Magnetic resonance imaging studies before and after immobilization showed so-called bony bruising ("oedema and haemorrage")[5(p488)] in the metaphysis of the calcaneus and no change to the apophysis. The authors give no mention of reliability and validity for their assessment. All of the references they cited for bony bruising directly related to one of the authors (Ogden), introducing the possibility of bias toward this diagnosis.

Incidence of Sever's Disease

Sever's disease affects children while bony growth is occurring at the calcaneal epiphysis, generally between the ages of 7 and 15 years in boys and 5 and 13 years in girls.[49, 50] Sever's disease is reported to have an incidence of 2% to 16% of musculoskeletal injuries in children.[6, 8, 10, 19, 24, 51, 52] All of these estimates arise from non-Australian populations. Reports on the incidence of Sever's disease have mainly been from retrospective reviews of sports medicine clinics, although

a few studies have been prospective on rates, and some targeted Sever's disease directly. None of the studies[6, 8, 10, 19, 24, 51, 52] mentioned testing of the ability of the assessors to distinguish Sever's disease from other conditions, and most studies had multiple assessors within the clinic, thus introducing individual variations within the studies.

No studies have examined the true incidence of Sever's disease within an entire population. Many studies were retrospective, and the authors relied on the quality of the clinical notes for their data. Table 2 presents a summary of the articles relating to reports on the incidence of Sever's disease.

Orava and Puranen's[19] 3-year Finnish study found 16.3% of exertion injuries in a child and adolescent population to be attributable to Sever's disease. Although a prospective study, it collected data from a sports-playing population that accessed a sports medicine clinic for treatment. This design reduces the ability to make inferences from the results to the wider population. Orava and Vitanen's[20] retrospective Finnish study of an outpatient sports medicine clinic is the most common study design that researchers use for this condition. The lack of standardization before data collection increases the risk of poorer-quality data with multiple assessors measuring with untested tools. Orava and Vitanen reviewed the data for 6 years of visits to their clinic with no mention of the number of treatment staff or evaluation criteria and found that 22.7% of the osteochondroses seen were Sever's disease. Because of the nature of the clinic and the type of injuries studied, their data cannot be extrapolated to provide a wider picture of incidence in the population.

Kannus et al[54] prospectively examined cases presenting to a Finnish sports medicine clinic over a 30-month period. They found that six cases (8%) of total injuries in boys and three cases (5%) of total injuries in girls were attributable to Sever's disease, but that 27% of ankle, heel, or foot injuries in boys and 12% of these injuries in girls were identified as Sever's disease. The sports-playing population sample again reduces extrapolation to the wider population factor; but this is remedied in a questionnaire-based study of a normal Finnish school population (n = 344).[44] The authors did not report on sampling criteria, validity, or reliability of tools. Kvist et al[44] cited an incidence of 11.3% of calcaneal pain as assessed by the child or parent; but because of the data collection method, the actual number of Sever's disease cases is open to question.

Apple,[53] in a retrospective study of adolescents' running injuries seen in a US sports medicine clinic, reported that 20% of 95 male runners and 38% of 29 female runners had calcaneal apophyseal injuries. However, he does not clarify how many of these apophyseal injuries were Sever's disease. Micheli and Ireland[9] stated that Sever's disease was the most common cause of heel pain in the growing athlete on the basis of results from their retrospective study of a sports medicine clinic, but they provided no figures to qualify this statement. In Micheli and Fehlandt's[10] retrospective review of the same clinic over a 10-year period, Sever's disease was the highest recorded apophysis or tendon injury in boys aged 8 to 19 years (12.7% of total injuries and 14.5% of lower-extremity). Because Micheli and Ireland's[9] and Micheli and Fehlandt's[10] data were drawn from the same clinic, the notes used for review may have overlapped. If they did, the independence of these studies would be reduced, and both studies would be subject to the aforementioned problems of retrospective design and targeted populations.

De Inocencio,[52] in a prospective study of 1,000 visits to a pediatric clinic in Spain, found that 5% of musculoskeletal complaints were attributable to Sever's disease. There was no information about how this di-

Table 2. Summary of Articles on the Incidence of Sever's Disease

Study	Incidence of Sever's Disease
Orava and Puranen[19]	24 of 147 injuries (16.3%) seen in a sports medicine clinic
Orava and Vitanen[20]	22.7% of osteochondroses seen in a sports medicine clinic
Apple[53]	20% of 95 male runners and 38% of 29 female runners had apophyseal injuries; no information on Sever's disease
Kannus et al[54]	6 cases (8%) and 3 cases (5%) of total injuries in boys and girls, respectively; of total ankle, heel, and foot injuries, 27% and 12% of total ankle, heel, and foot injuries in boys and girls, respectively, seen in a sports medicine clinic
Kvist and Heinonen[6]	11.3% of 344 cases of sports injuries in child population
Micheli and Fehlandt[10]	12.7% (92 of 724 injuries over 10 y); 14.5% (92 of 636) of lower-extremity injuries
De Inocencio[52]	5% of musculoskeletal complaints of 1,000 visits to a pediatric clinic
Price et al[24]	2% of total injuries in soccer academy injury audit

agnosis was made or how many investigators were used. However, because the clinic was a general rather than a sports medicine clinic, these data are perhaps the best available on incidence from a child population presenting for treatment. Price et al[24] relied on trainers at the English football academies taking part in their prospective audit to provide numbers of Sever's disease cases. This audit involved a possible increase in misdiagnosis because of a lack of uniform diagnostic criteria. That along with the specialized nature of the population make extrapolation to a wider population impossible.

Although the literature contains a fairly large number of studies that have reported the incidence of Sever's disease, the lack of consistency in reporting, use of multiple raters, and retrospective analysis of notes reduce the confidence with which the findings can be accepted.

Pathophysiology of Sever's Disease

The calcaneus is situated at the most plantar posterior aspect of the foot. It articulates superiorly with the talus at the subtalar joint and distally with the cuboid, making up part of the midtarsal joint.[55] The Achilles tendon inserts into the lower, posterior aspect of the calcaneus. The plantar fascia originates from the medial tubercle on the plantar aspect of the calcaneus. Near the attachment of the Achilles tendon to the calcaneus, a secondary bony growth center, or epiphysis (Fig. 1), opens in girls aged approximately 5 years and in boys aged 7 to 8 years. It fuses in girls aged approximately 13 years and in boys aged 15 years.[11, 49, 50, 56] Proximal to the epiphysis is the apophysis, where the Achilles tendon actually inserts. The apophysis has its own, slower-growing growth plate, separate from the physeal plate.[3, 5] The calcaneal growth plate and apophysis are situated in an area subject to high stress from the plantar fascia and Achilles tendon[22] and may be affected by increased tension on the calcaneus.

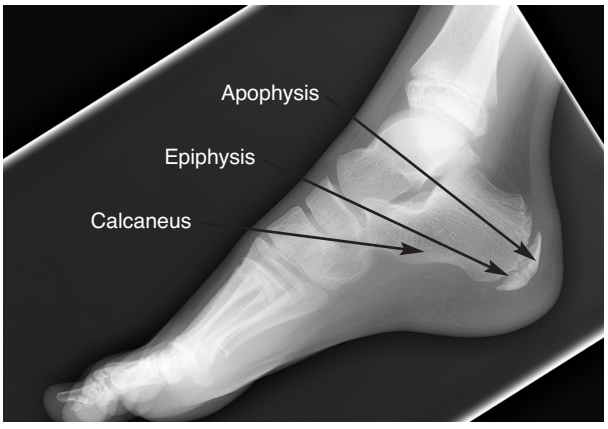

Figure 1. Apophysis of calcaneus.

The main theory in the literature on the pathophysiology of Sever's disease is that it is an overuse syndrome from repetitive microtrauma from an increased traction on the apophysis. This increased traction is believed to be initiated by running and jumping, which causes avulsion fractures on a tiny scale, followed by inflammation.[4, 9, 14, 45, 57-59] Because of the rapid proliferation of cells in growth plates, the apophysis is thought to be more susceptible to injury.[26] Liberson et al[22] examined calcaneal apophyses histologically and with computed tomography and found fibrous bands in the cartilage, perpendicular to os calcis. This finding indicated powerful stresses in the remodeling process, leading Liberson et al to suggest a traction-stress argument, where pain is thought to occur when remodeling exceeds certain rates. However, this argument remains a theoretical mechanism for Sever's disease, as the histological samples were from accident victims with no available medical history. Other proponents of this theory have failed to undertake experimentation to prove or disprove it.

The existence of inflammation in Sever's disease is based upon the radiographic finding that the symptomatic fragmented apophysis is more dense than the asymptomatic apophysis.[1, 2, 18, 40] However, blind testing of Sever's disease identification from x-rays has been unreliable.[56, 60] Hauser[13] stated that the density increased but that the asymptomatic foot often looked worse, which suggests that this particular inflammation assessment method was not valid. Volpon and de Carvalho Filho[23] examined lateral x-rays from 392 asymptomatic and 69 symptomatic male adolescents. The symptomatic males had unilateral heel pain for at least 6 months. The pain was initiated when walking or on palpation of the apophyseal area, and as there was no single criterion for inclusion, there is uncertainty about diagnosis correctness. Volpon and de Carvalho Filho did not show changes in density consistent with a repair process apparent in the heels of those subjects with Sever's disease, indicating that Sever's disease is unlikely to be vascular in origin. The study also showed increased fragmentation of the calcaneal apophysis in the Sever's disease group, which suggests greater mechanical demands during a vulnerable period but not inflammation per se.

There is no substantial evidence in the literature for the presence of an inflammatory process through either histologic or radiographic means. Long et al[61] contended that magnetic resonance imaging would

show inflammation in the apophysis but presented only a single example comparing an asymptomatic foot with the same subject's symptomatic foot. Ogden et al[5] refuted this contention and found changes in the metaphyses rather than the apophyses of 14 patients with recalcitrant heel pain that had been unresponsive to conventional treatment. However, the process leading to the diagnosis of these subjects with Sever's disease was not made clear, suggesting the possibility of a specific subset of subjects differing from the normal Sever's disease population due to their atypical characteristics. There was also no indication of acceptance, reliability, or validity of the assessment of the so-called bony bruising they suggested, except by referring to their own publications.

Although there is no strong evidence for the existence of inflammation per se in Sever's disease, the presence of pain on palpation and traction and changes evidenced histologically in the apophyseal area suggest an inflammation in this area if overload of the apophyseal area occurs.

Pathomechanics of Sever's Disease

Many theories have been put forward to explain the formation of Sever's disease, but like other areas of this condition, they have generally been hypothetical or formed on the basis of small populations. Theories may be split into the subheadings of growth and Achilles tendon tightness, biomechanical malalignment, trauma, infection, and overweight.

Growth and Achilles Tendon Tightness

Some have postulated that a period of rapid growth will lead to the presentation of symptoms of Sever's disease. That is, athletes experiencing a growth spurt may experience significant muscle-tendon imbalance where tight and inflexible muscle groups arise because muscle development is thought to lag behind bone development.[3, 10, 59] However, there is no evidence for this theory in the literature. The theory that tight triceps surae cause excessive tension through the Achilles tendon increasing the traction on the apophysis has been a commonly cited mechanical factor in much of the literature.[9-11, 13-15, 26, 29, 30, 33, 34, 42, 43, 46, 62-65] The studies that looked at a population of Sever's disease patients[9, 10, 14, 15] generally involved assessment by multiple raters of foot dorsiflexion on the leg, thereby reducing uniformity of measurement and reliability of results. There was generally no mention of how foot dorsiflexion was measured, whether it was assessed by multiple practitioners, or the reliability or validity of the measurements.

Szames et al[15] did report use of a visual assessment for tightness by multiple raters but gave no indication of any reliability assessment for this measurement technique. This omission calls into question the existence of excessive tightness in the triceps surae at all. No studies have compared whether symptomatic subjects are tighter in the triceps surae than their asymptomatic counterparts.

Biomechanical Malalignment

References to biomechanical influences in Sever's disease are scattered throughout the literature. However, no prospective systematic measurement of the feet of those identified with Sever's disease has been carried out, and certainly no comparison to an asymptomatic population. Sever[2] and Lewin[12] stated that there may be a slight amount of pronation present and that it should be addressed, but they gave no indication of the measurements taken to establish pronation. Hauser[13] continued this trend, theorizing that pes valgoplanus increases tension on the fascia and Achilles tendon and should be considered the most likely cause of Sever's disease. Hauser did not base this theory on any systematic studies of tension measurements or the finding that correction of the valgus relieved symptomatology in a cohort of symptomatic subjects.

Krantz[14] presented information contrary to the pronated foot being a causative factor in his retrospective analysis of radiographs of his patients. He classified the feet of his subjects with Sever's disease via calcaneal inclination angle measurement with an untested (in reliability and validity) radiographic technique, noting medium to high arch anatomy in his subjects. He postulated traumatic inflammation to the apophysis caused by this foot type and the observed limited ankle dorsiflexion. The first mention of more common podiatric measures of foot malalignment is by McKenzie et al.[8] They noted, retrospectively, that 95% of their Sever's disease subjects had a biomechanical imbalance of forefoot or rearfoot varus, though there is no mention of the method, reliability, or validity of measurement. They described this position as producing a whipping action in the Achilles tendon, increasing the stress on the apophysis. However, this theory has no evidence to support it.

The trend of cursory acknowledgment of possible biomechanical malalignment in the feet of subjects with Sever's disease is continued by McCrea,[26(p251)] who mentions that a "pathologic condition exists in the foot that may be contributing to calcaneal stress," referring to a foot malalignment, but gives no further details. This brief mention of possible malalignment was echoed by Tax,[66] Thomson,[17] Valmassey,[67] Dyment,[68]

Topham and White,[32] and Omey and Micheli.[33] However, in none of these instances is there mention of testing to establish malalignment, nor are any references supplied.

Micheli and Ireland,[9] in their group of 85 Sever's disease cases, noted foot malalignments in 23 cases. However, they did not provide an explanation of the classification system used, nor were reliability and validity issues mentioned. This was a retrospective study that involved a sports clinic at a hospital. One may assume that a variety of clinicians treated these subjects, with very little uniformity in their classification, thereby reducing the usefulness of this information. For example, 16 patients had forefoot pronation, 4 had pes planus, 2 had cavus feet, and 1 had pronation and hallux valgus. The first of these classifications is not a common term, and the remaining three are purely subjective classifications. The remaining 62 patients had "no associated foot conditions,"[9(p35)] with again no details as to how this was determined. However, 75% of the patients were treated with soft orthotics as part of their treatment regimen, which suggests that a foot condition was being managed. No reason is given for the apparent discrepancy.

Santopietro[42] commented that compensated transverse plane deformities or forefoot varus were invariably present in the Sever's disease subject. The deformities purportedly led to a harder heel strike rather than greater traction on the apophysis, but because no evidence was presented, this is purely anecdotal. Szames et al,[15] and later Madden and Mellion,[11] recommended biomechanical control via orthotic therapy after the symptoms have resolved to control a pronatory or cavus foot. They have assumed that these foot types are a risk factor for Sever's disease. Both articles fail to state directly that these foot types may be causative factors and provide no evidence for their assumption. Bartold[43] did not mention biomechanical influences in etiology, although he did suggest orthotic management in treatment. He theorized that shock attenuation is decreased and load increased on the Achilles tendon because of biomechanical abnormalities, causing increased tension at the apophysis. He offered only anecdotal advice and no supporting evidence.

Trauma

Some authors have mentioned acute and chronic trauma on a large scale as a cause of Sever's disease. Haglund[40] suggested that a calf muscle strain caused a fracture or separation of the apophysis. Sever[2] and Hauser[13] suggested a friction rub on the posterior aspect of the calcaneus as a possible cause. All of these examples relate to small samples assessed nonsystematically. Kurtz[69] reported two cases of trauma subsequent to a heavy landing and another after an ankle twist. He failed to look at other possible predisposing factors and, having enacted surgery upon at least one of his cases, most likely caused such a prolonged rest period that symptoms resolved.

Tax,[66] McCrea,[26] and Alexander[37] suggested that a single or repeated traumatic episode is a likely cause of Sever's disease but presented no evidence to justify this statement. Caspi et al[70] published a case report involving a 15-year-old female athlete who experienced a hard landing, leading to an apophyseal plantar migration. They do not elaborate on the assessment, and the radiographs do not appear to show an appreciable difference between the asymptomatic and the symptomatic foot's apophysis location. Wirtz et al[71] presented three case studies where apophysitis was purportedly caused by tae kwon do hook kicks. The diagnoses were made by radiograph, which has questionable accuracy, as discussed previously. The theory presented was that direct contact and shearing from plantar fascia tightening and a dorsiflexed foot position with an extended knee were the cause of these symptoms. However, this explanation does not fit the mechanics of this kick and fails to take into account the possibility of simple trauma.

Walling et al[72] associate Sever's disease with a more serious calcaneal fracture in their 13-year review of 11 patients of a hospital's orthopedic department. Calcaneal fracture is much different from the typical Sever's disease case; however, this trend continued with Ogden et al,[5] who postulated that Sever's disease is actually a metaphysis fracture. The sample involved 14 subjects who had failed to respond to conventional treatment and whose diagnosis and characteristics were poorly described. The fracture status was assessed with magnetic resonance imaging, but the authors do not mention the validity of this assessment, and all references for the condition were to the authors' own articles. Their findings are thus interesting but not definitive.

Madden and Mellion,[11] Peck,[3] and Topham and White[32] suggest anecdotally that a major traumatic event, minor trauma, or combination of the two are a likely cause of Sever's disease. Early authors[2, 12, 18] recommended padding as a treatment, which suggests that a lack of cushioning may be a causative factor, but they include no testing of this theory.

Infection

Ozgocmen et al[73] cite a case report of a 12-year-old boy who developed an apophyseal inflammation sec-

ondary to brucellosis infection, and other authors only anecdotally mention infection as a causative factor.[3, 15] However most authors refer to infective inflammation as a differential diagnosis rather than a causative factor.[9, 11, 16]

Overweight

Sever,[2] Meyerding and Stuck,[18] Trott,[74] and McCrea[26] have all noted that subjects with Sever's disease were overweight. Although it is not mentioned in the publications, one assumes that being overweight will increase pull on the apophysis by the harder-working Achilles tendon to lift the heavier subject. Another possible theoretical pathological pathway is that the increased weight will increase impact forces, leading to bruising of the apophysis. Simko,[25] however, reported three Sever's disease cases, of which two were underweight and one overweight by his (unexplained) assessment. Stess,[62] in his case study, presented a subject with normal weight and height. None of these articles details how overweight or underweight were assessed, so this factor has to be treated with some caution.

It is apparent that all of the pathomechanical theories authors have presented are based on poor, if any, data. The case for the collection of systematic information to strengthen any of these arguments is a compelling one.

Diagnosis of Sever's Disease

Many authors have stressed that Sever's disease is a diagnosis made clinically[9, 31, 36, 53] but did not present any studies to validate this statement or suggest a diagnostic technique. The main diagnostic tool is pain on medial-lateral compression of the calcaneus in the area of the growth plate (Fig. 2) while the growth plate is still open and in the absence of more serious causative factors such as extreme trauma.[72] It may be postulated that if the growth plate area is inflamed, applying pressure to it would cause pain, but the literature does not give the actual cause of the pain produced by this test. No other independent diagnostic tool exists in the literature, though radiographic techniques have been investigated since the initial description by Haglund[1] in 1907 and seems to be an area that refuses to be dispelled.

History of Imaging Modalities as a Diagnostic Tool

Authors—on the basis of no more than their subjective opinions—have examined radiographs of children with Sever's disease and found greater density of the apophysis in the affected heels.[1, 2, 18, 40, 69] This trait has been found to be inconclusive as a diagnostic tool in repeated studies by radiologists, doctors, and podiatric physicians.

Figure 2. The squeeze test. Medial-lateral compression of the calcaneal growth plate to elicit pain in Sever's disease.

The normal apophysis of a child is indistinguishable in appearance from the painful apophyseal area when assessed blindly against asymptomatic subjects of similar ages,[14, 56, 60, 75] asymptomatic heels in the same patient,[13] or when reporting on the characteristics of symptomatic and asymptomatic heels.[22] Volpon and de Carvalho Filho,[23] in a large study of symptomatic versus asymptomatic individuals, found a statistically significant decrease in density of primary and secondary nuclei of the apophysis in the apophysitis group when assessed by computer. They concluded that the density of the apophyseal area cannot be used for diagnosis. They did, however, measure greater fragmentation of the apophysis in the Sever's disease group, with computer imaging and assessment, though with no mention of reliability or validity of this technique. Many authors as recently as 2003[3, 35, 39] still postulate that there is a noticeable difference present in the radiographs of children with Sever's disease compared with asymptomatic counterparts. As such, the term *osteochondrosis*, with its suggested change in bony density,[45] is probably best not used for this condition, as there have been no conclusive results showing a definite difference between asymptomatic and symptomatic heels. Other authors acknowledge that

radiographs are inadequate for diagnosis but suggest that they are a useful differential diagnostic tool to aid in ruling out fractures and tumors.[9, 11, 43]

The literature contains one mention of scintigraphy (bone scanning with radioactive isotopes) for diagnosis,[20] although there is no mention in the references on sensitivity or, more important, specificity. Lokiec and Wientroub[76] used computed tomographic scanning of the heels for diagnosing what they called calcaneal osteochondritis but did not mention what they based this diagnosis upon. Liberson et al[22] investigated the heels of 35 Sever's disease subjects. They found that the density of the apophyseal area increased compared with the tuber section of the calcaneus, but they did not state or test that it differed in asymptomatic subjects. Long[61] showed magnetic resonance imaging changes consistent with apophyseal edema in a single apophysitis case. However, as there was no diagnostic information or confirmation via other tests that inflammation was present, this information is conjecture.

Ogden et al[5] refute this magnetic resonance imaging presentation in their sample of 14 heel pain sufferers, unfortunately with no diagnostic criteria mentioned and who had also failed to respond to conventional treatment. These subjects were reported to have changes in the metaphysis of the calcaneus rather than the apophyseal area consistent with bony bruising and microfracture of this area. However, this finding must be treated with caution because of the inadequate description of Sever's disease diagnosis and the lack of rigor of the diagnosis of bony bruising in the metaphysis. Additionally, Ogden et al are possibly biased toward this theory; they refer to only their own papers on this topic.

Diagnosis is best made with the aforementioned symptomatic picture, together with radiologic and other tools if the diagnostic or resolution pattern differs from the norm.

Differential Diagnosis of Sever's Disease

Although there are many conditions that may produce heel pain in adolescents, their symptom patterns are quite different from Sever's disease. These symptom patterns should be ruled out before a diagnosis of Sever's disease is made.

Musculoskeletal causes of adolescent heel pain include achillobursitis,[2, 6, 9, 11, 12, 16, 66] tenosynovitis,[2, 6, 9, 11, 12, 16] ankle sprains or peritendinitis,[6] retrocalcaneal exostosis or bursitis,[6, 9, 11, 16, 66] and plantar fasciitis.[2, 6, 9, 11, 12, 16] In all of these conditions, pain will be elicited on the posterior or plantar heel or ankle area and should be negative to a squeeze test of the apophyseal area. A child's ability to be this clinically specific may make this a challenging prospect.

Infective or internal causes of heel pain in adolescents include tuberculosis[2, 12]; rheumatoid arthritis, rheumatoid fever, and other arthridities[6, 9, 11, 16, 26, 77]; cysts and tumors[6, 9, 11, 16, 77]; and osteomyelitis.[6, 9, 11, 12, 16, 26] These cases will involve other parts of the body, such as generalized aches or symmetrical aching, raise in temperature, or malaise. There may also be different symptom patterns—for example, pain at night with a tumor—which would assist in differentiation.

Traumatic influences in adolescent heel pain include foreign bodies,[42] entrapment of the inferior calcaneal nerve,[30] ruptures of tendon or ligaments,[6, 9, 11, 16, 26, 77] fractures and stress fractures,[6, 9, 11, 16, 26, 77] tarsal tunnel syndrome,[6, 9, 11, 16, 26, 77] and contusions.[6, 9, 11, 16, 26, 77] In these situations, there will generally be a traumatic event cited in the subject's history and altered distribution and type of pain from Sever's disease noted in examination, again aiding in differentiation.

Other differential diagnoses mentioned have included tarsal coalition,[9, 11, 16, 77] in which the decreased range of motion at the subtalar joint will alert the therapist to this condition,[66] and a negative squeeze test. Rozenblat and Bauchot[16] include cutaneous conditions such as verrucae and dermatitis; their surface appearance makes them distinguishable from an unaffected heel.

Although there are many possible confounding diagnoses for heel pain in the child and adolescent, there are none that specifically mimic the positive squeeze test of Sever's disease without a wider spectrum of symptoms. Therefore the diagnosis of Sever's disease is one that may be made with relative clarity.

Treatment of Sever's Disease

Recommended treatment for Sever's disease is quite varied. Most authors have simply relayed what earlier authors have advocated. Other authors have included what they used for their patients and respective times of healing, though these have generally been retrospective, without clear guidelines on how treatment success was measured.[8, 9] Little information is reported on the mechanisms by which these treatment regimens work. A descriptive summary of the various treatment modalities is included in Table 3. Because there are no data about Sever's disease causation that have been scrutinized for reliability and validity to date, and no clinical trials comparing treatments carried out, none of these treatment regimens can be said to be better than others, thus making "best practice" a guessing game.

Table 3. Treatment Modalities Advocated by the Literature

Modified rest or cessation of sports
Haglund[1]
Sever[2]
Peck[3]
McKenzie[8]
Micheli and Ireland[9]
Micheli and Fehlandt[10]
Meyerding and Stuck[18]
McCrea[26]
Crawford and Gabriel[28]
Dalgleish[30]
Crosby and McMullen[31]
Subotnik[34]
Sammarco and Carrasquillo[35]
Adirim and Cheng[38]
Haglund[40]
Hetherington[41]
Bartold[43]
Apple[53]
Webster[58]
Garbett[63]
Dyment[68]
Trott[74]
McNamee[78]
Contompasis[79]
Wong and Gregg[80]
Griffin[81]

Heel lifts
Sever[2]
McKenzie[8]
Micheli and Ireland[9]
Micheli and Fehlandt[10]
Madden and Mellion[11]
Lewin[12]
Hauser[13]
Krantz[14]
Meyerding and Stuck[18]
McCrea[26]
Crawford and Gabriel[28]
Clain and Hershman[29]
Crosby and McMullen[31]
Subotnik[34]
Manusov et al[36]
Adirim and Cheng[38]
Hetherington[41]
Santopietro[42]
Bartold[43]
Katz[45]
Brantigan[57]
Stess[62]
Saperstein and Nicholas[64]
Dyment[68]
Wirtz et al[71]
Trott[74]
Griffin[81]
Spencer and Person[82]
Bours[83]

Orthoses (all types) or mobilizations
Sever[2]
McKenzie[8]
Micheli and Ireland[9]
Micheli and Fehlandt[10]
Hauser[13]
Simko[25]
Dalgleish[30]
Subotnik[34]
Sammarco and Carrasquillo[35]
Santopietro[42]
Bartold[43]
Katz[45]
Stess[62]
Garbett[63]
Contompasis[79]
Bours[83]
Wooten et al[84]
Tollafield and Merriman[85]

Stretching triceps surae, night splints, or strengthen extensors
Sever[2]
Peck[3]
McKenzie[8]
Micheli and Ireland[9]
Micheli and Fehlandt[10]
Madden and Mellion[11]
Lewin[12]
Czarniewski and Kachmar[27]
Clain and Hershman[29]
Dalgleish[30]
Crosby and McMullen[31]
Topham and White[32]
Omey and Micheli[33]
Sammarco and Carrasquillo[35]
Manusov et al[36]
Adirim and Cheng[38]
Hogan and Gross[39]
Hetherington[41]
Bartold[43]
Gregg and Das[46]
Apple[53]
Webster[58]
Garbett[63]
Saperstein and Nicholas[64]
Tax[66]
Dyment[68]
Contompasis[79]
Wong and Gregg[80]
Bours[83]
Wooten et al[84]
Tollafield and Merriman[85]
Evans[86]

Plantar fascial stretching
Dalgleish[30]
Crosby and McMullen[31]
Topham and White[32]
Saperstein and Nicholas[64]

Padding for shock absorption or strapping of heel
Sever[2]
Peck[3]
Madden and Mellion[11]
Lewin[12]
Krantz[14]
Meyerding and Stuck[18]
Simko[25]
Czarniewski and Kachmar[27]
Crawford and Gabriel[28]
Dalgleish[30]
Omey and Micheli[33]
Katz[45]
Brantigan[57]
Webster[58]
Stess[62]
Garbett[63]
Saperstein and Nicholas[64]
Tax[66]
Kurtz[69]
Contompasis[79]
Bours[83]
Tollafield and Merriman[85]
Evans[86]

Ultrasound, nonsteroidal anti-inflammatory drugs, ice, or steroid injection
Peck[3]
Micheli and Fehlandt[10]
Madden and Mellion[11]
Czarniewski and Kachmar[27]
Clain and Hershman[29]
Dalgleish[30]
Crosby and McMullen[31]
Topham and White[32]
Subotnik[34]
Manusov et al[36]
Hetherington[41]
Bartold[43]
Katz[45]
Stess[62]
Dyment[68]
Wirtz et al[71]
McNamee[78]
Wooten et al[84]

Casting or crutches
Ogden et al[5]
Madden and Mellion[11]
Lewin[12]
Hauser[13]
Meyerding and Stuck[18]
McCrea[26]
Crawford and Gabriel[28]
Manusov et al[36]
Santopietro[42]
Stess[62]
Kurtz[69]
McNamee[78]
Spencer and Person[82]
Seder[87]

Removal of apophysis
Caspi et al[70]

Conclusion

We have examined the definition, diagnosis, etiology, pathomechanics, and treatment of Sever's disease. Although several areas have been identified as possible causative factors, we have shown that none of these has been investigated in a scientific manner. The impact Sever's disease has on the individual has not been mentioned in the literature. An investigation into the true effect of the causative factors alluded to in the literature and the impact Sever's disease has on children would be a necessary and worthy addition to the body of knowledge and a necessary precursor to any higher-level study of most effective treatments.

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