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CURRENT OPINION

Inconsistencies and Imprecision in the Nomenclature Used to Describe Primary Periphyseal Stress Injuries: Towards a Better Understanding

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Abstract
Stress injuries involving the epiphyseal–physeal–metaphyseal complex affecting the extremities of child and adolescent athletes were first described in the early 1950s. Initially observed in Little League baseball players, these injuries are now known to affect skeletally immature athletes in a variety of sports that involve high-impact repetitive overuse activities. Collectively known as primary periphyseal stress injuries, they may affect the long bones around the shoulder, elbow, wrist, hand, hip, knee, ankle, and foot of young athletes. These injuries respond well to timely treatment and relative rest, while non-compliance with non-operative treatment can produce skeletal growth disruption and resultant limb deformity. A major concern raised from the existing literature on primary periphyseal stress injuries is the long history of inconsistent and imprecise terminology used to describe these injuries. A variety of terms have been used to describe primary periphyseal stress injuries, including those which potentially misinform regarding who may be affected by these injuries and the true nature and pathophysiologic mechanisms involved. These imprecisions and inconsistencies arise, at least in part, from a misunderstanding or incomplete understanding of the nature and mechanism of primary periphyseal stress injuries. In this article, we examine the inconsistent and imprecise nomenclature historically used to describe primary periphyseal stress injuries. We also offer a novel framework for understanding the pathophysiologic mechanisms behind these injuries, and provide suggestions for more standard use of terminology and further research moving forward.

1 Introduction

School-aged children and adolescents are increasingly engaged in high-level sport, with prolonged duration and intensity of physical loading, earlier specialization, year-round training, and increased difficulty of skills practiced [1]. The mechanical tolerance of the epiphyseal–physeal–metaphyseal (EPM) complex may be exceeded by the intense and continuous training of youth sports [2]. Overuse physeal stress injuries occur when repetitive loading of the extremity is imposed without a sufficient interval of rest to allow for structural adaptation [3]. These injuries may involve one or more constituents of the EPM complex; therefore, it makes sense to collectively refer to them as primary periphyseal stress injuries (PPSIs) [2]. The term "primary" is applied to specify that the injury typically involves the primary growth plate, which is responsible for the longitudinal growth of its respective bone; this is in contrast to the secondary growth plate, which is responsible for the growth of the secondary ossification center (SOC) in epiphyses and apophyses. Protecting these growth plates is essential, as impairment and dysfunction can result in lifelong morbidity and the risk for premature osteoarthritis [2, 4].

Skeletally immature athletes in sports with high-impact repetitive activities, including baseball, badminton, basketball, climbing, cricket, dance, gymnastics, rugby, running, soccer, swimming, table tennis, tennis, and volleyball, may sustain PPSIs to the long bones around the shoulder [5–35], elbow [36–43], wrist [44–72], hand [73–84], knee [85–97], ankle and foot [98–105]. Several studies point to the potential for PPSIs in association with slipped capital femoral epiphysis and Legg–Calve–Perthes disease [106–109], although the precise pathophysiologic mechanisms remain unclear, likely multifactorial, and will not be discussed further.

Although incidence data from prospective cohort studies are lacking, data arising from cross-sectional investigations suggest that PPSIs may be common in select groups of youth athletes, including the shoulder in baseball players [110–112], the wrist in platform divers [113] and gymnasts [114–122], and the hand in climbers [123–127]. Most

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Key Points

The nomenclature used in reports of stress injuries involving the epiphyseal–physeal–metaphyseal complex has been characterized by inconsistency and imprecision for more than half a century. In the absence of a classification system specific to these injuries, we recommend adopting "primary periphyseal stress injury" or "PPSI" as an umbrella term referring to stress-related injury involving one or more components of the epiphyseal–physeal–metaphyseal complex.

The mechanism of stress-related epiphyseal plate widening observed on imaging in pediatric patients begins in the metaphysis with disruption of the normal metaphyseal blood supply. The resultant physeal widening is not a true fracture, although it may mimic a Salter–Harris type I fracture. There is no fracture cleavage plane, discontinuity through the physeal cartilage, or displacement; also, the widening can be reversible. Thus, the use of the term 'Salter–Harris I fracture' to convey stress-related widening of the primary physis is not accurate and should be avoided. Rather, these represent primary periphyseal stress injuries that produced physeal stress reflected in physeal widening (e.g., proximal humerus primary periphyseal stress injuries with physeal widening).

Similarly, the use of Salter–Harris type II–IV fractures to refer to stress fractures is also inappropriate and should be avoided if there is no history of a single inciting traumatic event. Rather, in stress-related bone failure, these injuries represent a continuum that summates multiple small repetitive microtrauma that ultimately leads to this common endpoint (i.e., fatigue or insufficiency fracture).

PPSIs appear to respond well to timely treatment, but in the absence of treatment, delayed presentation or diagnosis, or non-compliance with conservative management of activity limitation and/or modification, these can produce ongoing injury, resulting in long-term skeletal growth disruption and resultant limb deformity [2, 4].

A recent systematic review of PPSIs [2] highlighted the need for rigorous prospective longitudinal epidemiological and imaging studies designed to provide incidence rates of PPSIs and to determine the effect of these injuries on the long-term skeletal health of youth athletes. Nevertheless, for this research to proceed effectively, there is a need to address the issue of inconsistent and imprecise terminology, which has been used to describe PPSIs since the 1950s. In the early phases of a new area of research, terminology may be used slightly differently. However, as this novel research stream evolves, words and definitions used to describe the nuances need to become more standardized [128].

The incorrect use of terms and nomenclature pose an obvious threat to research given the potential interference with comparisons of findings across studies. Well-defined, evidence-based terminology reduces ambiguity and decreases the risk for accidental misinterpretation [128]. A universal language, based on well-defined terminology and based on scientific evidence, allows accurate and effective communication among healthcare providers and researchers, allowing optimization of patient care, facilitating ongoing research and translation of these new discoveries into clinical practice.

Here, we examine the inconsistent and imprecise nomenclature historically used to describe PPSIs, and offer an explanation on the nature and cause of these injuries. We also make evidence-based suggestions for the appropriate use of terminology, and propose a novel framework for better understanding.

2 EPM Complex

The primary growth plates, located at the ends of long bone, are responsible for the longitudinal growth of their respective bone, and are critical components of the immature skeleton. Dysfunction or injury to this EPM complex can lead to future growth disturbance, including limb length discrepancy, angular deformity, and altered joint biomechanics, increasing the risk for premature osteoarthritis [129]. Resultant deformity and clinical symptoms depend not only on the patient's anatomic site of injury and remaining growth potential, but also on the severity of injury and which component or components of the EPM complex are predominantly injured [4].

The EPM complex consists of the epiphysis, physis, and metaphysis [4] (Fig. 1). Growth disturbance results from endochondral dysfunction, which can arise from injury to the growth plate proper (direct injury) or to the vasculature of the adjacent epiphysis or metaphysis (indirect injuries). The growth plate contains chondrocytes that mature as they move from the epiphyseal side toward the metaphysis. On histology, three distinct zones of chondrocytes are recognized (namely reserve, proliferative, and hypertrophic zones) based on their microscopic cellular morphology and function. The reserve or germinal zone contains randomly distributed chondrocytes with an abundant surrounding extracellular matrix responsible for storing nutrients. The proliferative zone contains dividing chondrocytes, organized into columns, that are responsible for bone lengthening. The hypertrophic zone contains chondrocytes that undergo internal vacuolation and apoptosis (programed cell death) with a

progressive accumulation of calcium salts within the extracellular matrix. The latter hinders the diffusion of nutrients and oxygen and coalesces to form the zone of provisional calcification [130, 131]. The perichondrium surrounds the physis and contains the groove of Ranvier that is responsible for the latitudinal growth of the growth plate, and the ring of LaCroix that is responsible for providing mechanical support [131, 132].

The integrity of both epiphyseal and metaphyseal vessels is critical to ensure proper physeal function and endochondral ossification. The epiphyseal vessels nourish the juxta-epiphyseal side of the growth plate and produce a gradient of decreasing oxygen tension that extends from the epiphyseal toward the metaphyseal side of the growth plate. On the metaphyseal side, the terminal vascular loops and capillary tufts within the metaphyseal spongiosa terminate just proximal to the zone of provisional calcification, ensuring anaerobic metabolism within the hypertrophic zone of the juxta-metaphyseal physis to facilitate chondrocyte apoptosis [131]. Disruption of the epiphyseal vascularity can lead to a spectrum of abnormalities from blunted longitudinal physeal growth to premature physeal closure [133]. In addition, disruption of the metaphyseal vascularity can lead to prolonged chondrocyte survival with resultant physeal widening and apparent chondrocyte extension into the metaphysis [92]. The latter is observed in youth athletes with physeal overuse injury where chronic microtrauma disrupts the integrity of the metaphyseal vessels, which can be reversible, depending on the severity, duration, and persistence of ongoing stress [2, 134–136].

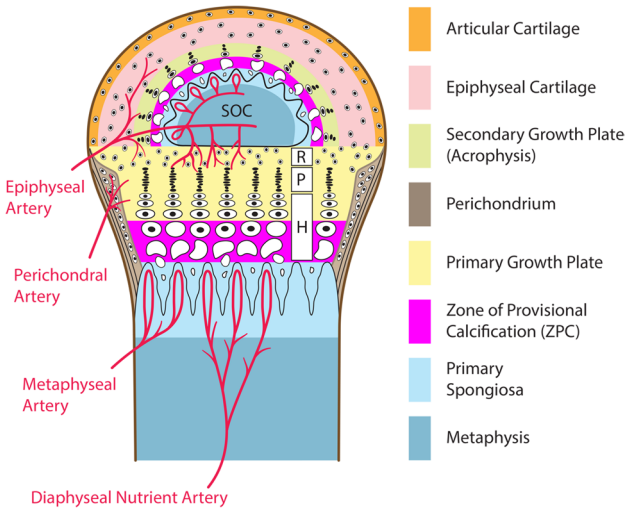

Fig. 1 Drawing illustrates the anatomy at the end of a long bone (not drawn to scale). Reproduced from Nguyen et al. [4], with permission. The primary growth plate is responsible for longitudinal growth of the bone and the secondary growth plate for the enlargement of the secondary ossification center (SOC). Both growth plates contain chondrocytes arranged in zones: randomly distributed reserve cells (R), linearly arranged proliferative cells (P), and enlarged hypertrophic cells (H), which undergo apoptosis at and near the zone of provisional calcification (ZPC) and are subsequently removed and replaced by laminar bone within the primary metaphyseal spongiosa. The epiphyseal vessels nourish the reserve and upper proliferative zones whereas the metaphyseal vascular loops terminate just proximal to the ZPC, leaving the hypertrophic zone relatively avascular. The perichondrium is responsible for the peripheral growth of the primary growth plate and contains its own blood supply

3 Inconsistent Terminology

In 1953, Dotter [35] described the case of a 12-year-old Little League pitcher with gradual onset of pain in the throwing shoulder. He referred to the injury as "Little leaguer's shoulder" (LLS), with radiographs showing a "fracture through the epiphyseal cartilage of the proximal humerus." Since then, LLS has been described using various terms, such as osteochondrosis of the proximal humeral epiphysis [7], proximal humeral epiphysiolysis [5], stress fracture of the proximal humeral epiphyseal plate [8], chronic overuse osteochondritis [23], and Salter–Harris type I fracture of the proximal humerus [22, 32]. Recent data suggest that the mechanism of repetitive torsional and distractive stresses at the EPM complex from throwing and other overhead activities results in reversible metaphyseal blood supply disruption, leading to endochondral dysfunction with persistence of chondrocytes and subsequent physeal widening [19, 20].

A wide range of terms have been used to describe PPSIs in other anatomical locations in the upper and lower extremities. These include chronic growth plate injury [69], chronic physeal stress injury [52], epiphysitis [70], epiphysiolysis [45], epiphyseal stress fracture [79], epiphyseal stress injury [42], growth plate overuse syndrome [103], growth plate stress reaction [44], osteochondrosis [7, 9], repetitive stress injury [63], epiphyseal growth plate stress injury [84], and stress-related growth plate widening [53, 57, 71, 92]. Additionally, many reports of PPSIs refer to these injuries as stress-related Salter–Harris injuries [41, 82], Salter–Harris stress-fractures [50, 51, 84], and chronic Salter–Harris type injuries [30, 40, 41, 43, 50, 51, 87, 93, 101].

Some terms used, such as "physeal stress injury," "growth plate overuse syndrome," "physeal widening," and "epiphyseal stress fracture," are mentioned in isolation and suggest a stress injury centered at the physis of the EPM complex. Finally, many of the terms used, including "stress fracture," "Salter–Harris type fracture," and "Salter–Harris type stress fracture," indicate that the fracture is the primary outcome rather than a superimposed or secondary effect because of an imbalance between mechanical load and bone quality.

The multiplicity of terms used to describe PPSIs likely arises from a lack of clarity about the true nature and cause

of these injuries as well as the overlapping imaging appearances of injuries that arise from different pathophysiologic mechanisms. The growing use of magnetic resonance imaging (MRI) has provided valuable insight and an improved description of PPSIs when compared with early reports that typically relied on radiographs [4, 5].

4 Imprecise Terminology

In addition to inconsistent nomenclature, the existing literature on PPSIs also uses imprecise terminology to describe these injuries. This includes (1) use of misleading terminology, (2) reference to the Salter–Harris physeal fracture classification, and (3) reference to one sport played, for example LLS (when the same pattern of injury occurs in young athletes participating in other sports).

4.1 Use of Misleading Terminology

Many of the terms describing this stress injury to the EPM complex, including "physeal stress injury," "chronic physeal injury," "overuse syndrome," "repetitive stress injury," "stress-related widening," and "growth plate stress reaction," can be misleading. They suggest that the primary physis is the only component of the EPM complex that has been directly injured, as evidenced by the physeal widening observed on imaging in many of these cases [2]. In reality, the overuse injury affects the primary physis indirectly or secondarily [2, 4]. The stress-related physeal widening observed on imaging pediatric patients (Fig. 2) actually begins in the metaphysis with disruption of the normal metaphyseal blood supply [92, 137]. The underlying mechanisms for PPSIs are described in Sect. 5.

4.2 Reference to the Salter–Harris Classification

Although numerous subsequent classifications and reclassifications of acute fractures that involved the physis and periphyseal structures have been advanced, the system originally proposed by Salter and Harris [138] remains the most widely used in clinical practice [139]. Initially intended for classifying direct or acute fractures involving the growth plate, this classification has often been applied to attempt to categorize metaphyseal insults in young athletes who are involved in a range of sports including baseball, basketball, climbing, dance, football, gymnastics, running, and volleyball (Table 1) [30, 32, 41, 50, 51, 65, 82, 84, 88, 93]. However, while the radiographic appearance of these injuries may appear similar to Salter–Harris type I fractures, the nature and mechanism of the injury are actually quite different. Unlike traumatic injury, overuse injury involving the EPM complex develops over time in response or

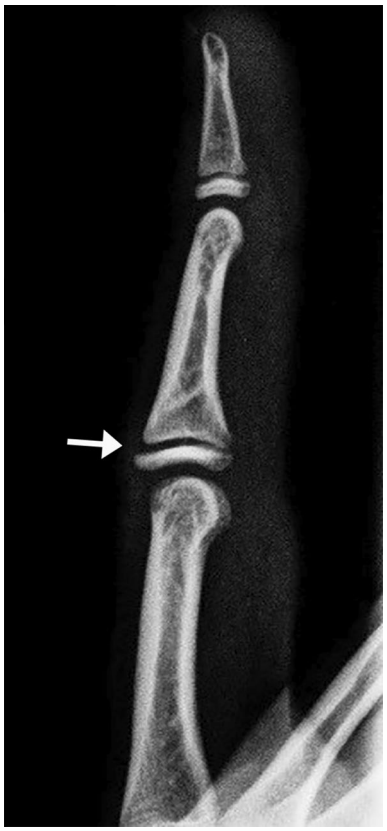

Fig. 2 14-Year-old male climber with gradual onset of localized pain after training. Lateral radiograph of the long finger shows subtle relative asymmetric widening involving the dorsal physis of the middle phalanx

secondarily to repetitive biomechanical overloading of the metaphysis and/or epiphysis [2, 4, 137, 140]. The resultant physeal widening, although it may mimic the radiographic appearance of a Salter–Harris type I fracture, is not a true fracture. Magnetic resonance imaging readily allows one to appreciate that there is no fracture cleavage plane, displacement, or discontinuity through the physeal cartilage; also, the widening can be reversible [137, 140, 141].

Although less common, there are also numerous reports of stress-related Salter–Harris types II–V fractures (Table 1). These injury types have been reported in physically immature athletes involved in ballet, baseball, basketball, gymnastics, climbing, running, and tennis [8, 22, 38–40, 42, 43, 48, 74, 75, 77–79, 81, 82, 84, 85, 94, 96, 98, 101]. However, rather than representing the result of a clear acute mechanism of injury (i.e., Salter–Harris fractures), they reflect fractures from either fatigue or insufficiency (Fig. 3).

A question thus arises as to the appropriateness of using the Salter–Harris terminology to adequately describe PPSIs. For PPSIs without fractures, the use of the term "Salter–Harris I fracture" to convey stress-related widening of the primary physis can lead to overtreatment. For PPSIs with

stress fractures extending into the metaphysis or epiphysis, optimal management entails not only treatment of the stress fractures, but also correction of the underlying biomechanical imbalance. This may involve eliminating excessive and reducing repetitive loading, improving nutritional intake, and strength training. Thus, reference to the Salter–Harris classification is not accurate, and should most likely be avoided [141].

4.3 Reference to Sport Played

Reports of stress injuries to the EPM complex occasionally refer to these injuries with reference to the sport played. For example, physeal widening of the epiphyseal cartilage of the proximal humerus has often been referred to as LLS since the 1950s [5]. However, the use of this term may be inappropriate for several reasons. First, LLS is a misnomer: it is actually more common among adolescents older than 12 years of age, and therefore beyond Little League years [141]. Second, this pattern of injury is not limited to baseball players, and has also been described in badminton and cricket players [27, 29], gymnasts [30–32], swimmers, tennis, and volleyball players [33, 34].

Similarly, stress injuries involving the EPM complex of the distal radius in young gymnasts are well documented and occasionally include the distal ulna [2, 53, 142, 143]. These injuries are commonly referred to as "gymnast's wrist," but a variety of other terms have also been used to describe this ailment in the distal radius such as "stress reaction" [44], "distal radial epiphysitis" [70], "stress-related Salter type I fracture" [65], "stress-induced widening of the distal radial growth plate" [53, 56, 57, 62, 71], "chronic impaction injury of the distal radius" [69], and "repetitive stress injury of the distal radial physis" [63]. However, as in LLS, this spectrum of findings in the "gymnast's wrist" has also been diagnosed in patients practicing other youth sports including badminton, break dancing, competitive diving, and climbing [45–47, 113]. As with LLS, the sports-related term "gymnast's wrist" is not representative and should most likely also be avoided.

We recognize that the use of terms such as LLS and gymnast's wrist to designate sport-specific PPSIs will not be easily converted or abandoned given the many decades of their usage. However, as described above, the precision of these terms has weakened over time, necessitating the need for better descriptors. The term "adolescent athlete's shoulder" has been suggested as a replacement for LLS given that the same insult of repetitive microtrauma, abnormal biomechanics from improper technique, and rotational torque occur in a variety of sports [33]. Similarly, "youth kicker's knee" has been used to describe PPSIs involving the distal femur and/or proximal tibial EPM complex of skeletally immature soccer and American football players [95]. However, these terms are also vague and assign a mechanism that may become restrictive. Instead, we suggest using the umbrella term PPSI to denote these repetitive mechanism injuries involving one or more components of the EPM complex [2].

5 Pathophysiologic Mechanisms for PPSIs

Given the imprecisions and inconsistences in the nomenclature used to describe PPSIs, the need for greater clarity in the description and understanding of these injuries is evident. These imprecisions and inconsistencies arise, at least in part, from a misunderstanding or incomplete understanding of the nature and mechanism of PPSIs. In this section, we introduce a novel framework for understanding the pathophysiologic mechanisms and outcomes of PPSIs.

Briefly, as described by Caine et al. [2], PPSIs develop following repetitive submaximal stress causing microtrauma to one or more constituents of the EPM complex. The precise nature of chronic microtrauma in these injuries depends on their anatomical location, sport played, offending action, and the forces involved that can include compressive, rotational, tension, and shearing strains. These injuries are usually reversible if treated with rest and in a timely manner [2, 4, 92, 137]. However, if PPSIs with physeal widening are undiagnosed or sub-optimally treated, progressive insults can lead to damage to the epiphysis and epiphyseal-sided blood supply, which can be gradual and progressive, or sudden in onset, increasing the risk for irreversible growth deformity [4, 81, 82]. Figure 4 depicts the pathophysiologic mechanisms for PPSIs, which are discussed below.

5.1 Repetitive Mechanical Stress

The most commonly reported imaging finding in PPSIs is physeal widening, reflecting damage to metaphyseal perfusion [2, 92] (Fig. 2). This is not surprising because the newly formed metaphyseal bone immediately subjacent to the physis is relatively fragile and has poor resistance to compressive forces, such as those that can occur from the chronic stress encountered with competitive sports activity [51]. Additionally, the relatively lower stiffness of the hypertrophic chrondrocytes, in combination with their large size, renders them particularly vulnerable to excessive mechanical loading [144].

Initially, this injury was believed to be a stress reaction or stress fracture through the primary physis [35, 44]. In reality, the stress-induced growth plate widening reflects alterations in metaphyseal perfusion that occurs with repetitive loading and interferes with the apoptosis of the hypertrophied chondrocytes [4, 92, 133, 137]. The hypertrophic zone continues to widen because of continued growth in the germinal and proliferative zones [137, 141]. The resulting physeal

Table 1 Case reports and case series reporting stress fractures in skeletally immature athletes that used the Salter–Harris classification

References	No. of subjects (age)	Anatomic location	Sports played	Imaging findings	Diagnosis
Salter–Harris type I					
Carter and Aldridge [50]	17M and 4F (age range: 9–17 years)	Distal radius	Gymnastics	Radiographs showed widening of the distal radial growth plate particularly on its volar aspect and narrowing of the adjacent epiphysis; there was also haziness of the growth plate due to irregularity and poor definition at the border between the growth plate cartilage and metaphyseal zone of ossification, and an increase in the transverse diameter of the metaphysis	Salter–Harris type I stress fracture
Carter et al. [51]	8M (age range: 14–16 years)	Distal radius	Gymnastics	Radiographs in all cases revealed bilateral widening of the distal radial growth plates. Growth plate margins were irregular, particularly on the metaphyseal side, which in the more florid cases showed an ill-defined cystic appearance and sclerosis. There was flaring of the distal radial metaphysis with spurring of its palmar aspect on the lateral radiographs. Discrete fragmentation was identified within the growth plate in six cases. All cases showed some flattening of the medial portion of the distal radial epiphysis, predominantly on the palmar aspect	Salter–Harris type I stress fracture
Frizzell and Brunet [65]	1M (age: 14 years)	Distal radius	Gymnastics	Radiographs showed widening of the distal radial physis, along with irregular margins, particularly on the metaphyseal side	Salter–Harris type I stress fracture
Daldorf and Byron [30]	1F (age: 15 years)	Proximal humerus	Gymnastics	Radiograph showed subperiosteal new bone distal and lateral to the physis, with medial and anterior displacement of the shaft relative to the epiphysis and physis	Salter–Harris type I displacement of the epiphysis

Table 1 (continued)

References	No. of subjects (age)	Anatomic location	Sports played	Imaging findings	Diagnosis
Nanni et al. [93]	1M (age 15 years)	Proximal tibia	Rugby	Radiographs revealed widening of the proximal tibial physis, asymmetrically affecting the medial than lateral side. The entire metaphyseal margin was irregular and sclerotic. MRI confirmed diffuse irregularity and widening of the tibial growth plate	Chronic Salter–Harris type I injury
Blatnik and Briskin [88]	1F (age: 12 years)	Distal femur	Gymnastics	Knee radiographs revealed mild widening of distal femoral physis bilaterally. Bilateral knee MRI confirmed slight widening of the physes	Salter–Harris type I stress fracture
Schoffl and Schoffl [82]	2M/F (age range: 12.7–15.8 years)	Middle phalanx of long finger	Climbing sports	Radiographs revealed stress injury involving the dorsal aspect of the middle phalanx	Salter–Harris type I injury
Oda et al. [41]	1M (age: 11 years)	Distal humerus	Gymnastics	Radiography and MRI showed a flexion deformity of both distal humeral epiphyses and callus formation	Stress-induced Salter–Harris type I injury
Kosnick [32]	1M (age: 13 years)	Proximal humerus	Gymnastics	MRI showed slight widening of the lateral physis, with edema within the adjacent metaphysis and epiphysis	Salter–Harris type I fracture
Schöffl et al. [84]	1M and 1F (age range: 15–16 years)	Middle phalanges of long and ring fingers	Climbing sports	MRI showed widening of the dorsal aspect of the physis	Salter–Harris type I fracture
Salter–Harris type II					
Cahill [8]	5M (age range: 11–12 years)	Proximal humerus	Baseball	Radiographs showed widening of the physis with metaphyseal bone separation representing a Thurston Hollindor type lesion. There was also metaphyseal and diaphyseal new bone formation secondary to periosteal stripping	Salter–Harris type II fracture
Cahill [94]	1M (age: 15 years)	Proximal tibia	Running	Radiographs revealed healing and physeal closure	Rotational or sheer-induced Salter–Harris type II stress fracture
Weber [96]	1M (age: 15 years)	Distal femur	Tennis	Radiographs revealed a Salter–Harris type II fracture extending obliquely from the lateral femoral metaphysis to the distal femoral physis, stabilized by external callus	Salter–Harris type II stress fracture

Table 1 (continued)

References	No. of subjects (age)	Anatomic location	Sports played	Imaging findings	Diagnosis
Hochholzer and Schöffl [81]	3M and 1F (age range: 13–16 years)	Middle phalanges of long finger (3 climbers) and ring finger (1 climber)	Climbing sports	Radiographs showed an epiphyseal abnormality involving the dorsal base of the long and ring finger middle phalanges. MRI T1-weighted sequences showed a complete signal loss of the whole epiphysis, and T2-weighted sequences demonstrated an effusion in the proximal interphalangeal joint	Salter–Harris II epiphysiolysis
Reeder and Smith [22]	1M (age: 13 years)	Proximal humerus	Baseball	Radiographs revealed a Salter–Harris type II fracture of the proximal humerus, with widening of the lateral physis. MRI confirmed a subacute Salter–Harris type II fracture of the proximal humerus, with metaphyseal marrow edema and uplifting of periosteum with fluid underneath	Salter–Harris II fracture
Salter–Harris type III					
Read [48]	2F (age range: 12–16 years)	Distal radius	Gymnastics	Radiographs revealed stress fracture of the radius that involved the metaphysis (case 1) and additionally extended into the epiphysis (case 2)	Salter–Harris types II and III stress fractures
Chell et al. [74]	1M, age 15	Middle phalanx of long finger	Climbing sports	Radiographs revealed bilateral Salter–Harris type III fractures, non-displaced on the left and slight displaced on the right. MRI confirmed fractures through the epiphyses	Salter–Harris type III stress fracture
Kadel et al. [98]	1M (age: 14 years)	Metatarsal of great toe	Basketball	Radiographs demonstrated a lucent line through the epiphysis and widening of the dorsal physis. CT confirmed a parasagittal lucency extending from the growth plate through the epiphysis into the first metatarsocuneiform joint and widening of dorsal aspect of the growth plate	Epiphyseal stress fracture (Salter–Harris III) of the first metatarsal
Hochholzer and Schöffl [81]	20M (age range: 13–16 years)	Middle phalanges of long and ring fingers	Climbing sports	All radiographs showed an epiphyseal abnormality of the dorsal base of the middle phalanx	Salter–Harris type III fractures

Table 1 (continued)

References	No. of subjects (age)	Anatomic location	Sports played	Imaging findings	Diagnosis
Huckaby et al. [40]	1F (age: 11 years)	Proximal radius	Gymnastics	MRI evaluation of both elbows indicated bilateral bone marrow edema in the epiphysis, metaphysis and proximal diaphysis of the radius. The left elbow MRI demonstrated thinned cartilage in the radial head and fracture lines through the epiphysis medially and through both the epiphysis and metaphysis laterally causing irregularity and a small notch in the metaphysis. Widening of the physis under the epiphyseal fragment was also noted	Salter–Harris fracture caused by repetitive microtrauma
Bayer et al. [75]	5 M and 2F (age range: 13–16 years)	Middle phalanx of long finger	Climbing sports	MRI showed anepiphyseal fracture line in all patients, including 1 patient with 2 injured fingers	Salter–Harris type III stress fracture
Schoeffl and Schoeffl [82]	15M and F (age range: 12.7–15.8 years)	Middle phalanx of long finger	Climbing sports	Radiographs revealed Salter–Harris type III injury (19 fingers) that extended into the proximal interphalangeal joint	Salter–Harris type III fracture
Desaldeleer and Le Nen [77]	1M (age: 17 years)	Middle phalanx of long finger	Climbing sports	Radiographs showed bilateral Salter–Harris type III displaced dorsal base fractures. Ultrasound confirmed fracture displacement	Salter–Harris type III displaced fracture
Csonka et al. [101]	1F (age: 10 years)	Proximal phalanx of great toe	Ballet	Radiographs of the feet showed segmentation of the epiphysis of the proximal phalanges of the first toe on both sides. These findings are consistent with both epiphyseal clefts and Salter–Harris type III fractures. MRI showed developing necrosis of the epiphyses on both sides	Non-healing Salter–Harris type III stress-induced fractures
Miller et al. [42]	4M/F (mean age: 11.7 years)	Proximal radius	Gymnastics	In addition to the Salter–Harris type III fracture bilaterally, imaging revealed proximal radial metaphyseal edema and contralateral metaphyseal irregularity	Salter–Harris type III epiphyseal stress injury

Table 1 (continued)

References	No. of subjects (age)	Anatomic location	Sports played	Imaging findings	Diagnosis
El-Sheikh et al. [78]	2M (age range: 13–14 years)	Middle phalanx of long fingers	Climbing sports	Case 1 (age 13 years): radiographs demonstrated Salter–Harris type III fractures of dorsal base of the middle phalanges of both long fingers. The fracture appeared dorsally displaced on the left hand and nondisplaced on the right hand. CT demonstrated cortication of the fracture margins on the left, suggesting a chronic non-union Case 2 (age 14 years): radiographs demonstrated an epiphyseal fracture. The patient was then allowed to restart climbing after 10 weeks and was completely free of symptoms. A routine follow-up radiograph 8 months later, which was performed before restarting high-load finger training, showed a non-union. MRI revealed open physes and confirmed the nonunion of the fracture site	Salter–Harris type III repetitive stress epiphyseal fractures
Halsey et al. [79]	1M (age: 16 years)	Middle phalanx of the long finger	Climbing sports	Radiographs revealed a Salter–Harris type III fracture at the base of the middle phalanx, which was confirmed on MRI	Salter–Harris type III epiphyseal plate fracture
Kadel et al. [98]	1M (age: 14 years)	Metatarsal of great toe	Basketball	Radiographs of the left foot demonstrated a lucent line through the epiphysis (anteroposterior view) and widening of the dorsal aspect of the physis at the base of the first metatarsal (lateral view) adjacent to a lead marker placed at the tender site. CT scan of the midfoot showed a parasagittal lucency extending from the growth plate through the epiphysis into the first metatarsocuneiform joint and widening of dorsal aspect of the growth plate (axial images), consistent with a Salter–Harris type III fracture	Salter–Harris type III stress fracture

Table 1 (continued)

References	No. of subjects (age)	Anatomic location	Sports played	Imaging findings	Diagnosis
Schöffl et al. [84]	14M and 6F (age range: 14–16 years)	Middle phalanges of long fingers and ring finger	Climbing sports	Ulrasound and MRI showed epiphyseal fractures of the dorsal base of the middle phalanx in the long (27 fingers) and ring finger (1 finger)	Salter–Harris type III fractures
Salter–Harris type IV					
Chan et al. [38]	3F (mean age: 13.5 years)	Proximal radius	Gymnastics	Radiographs revealed flattening and anterior depression of the radial head epiphysis, which developed into a chronic Salter–Harris type IV stress fracture in one patient who continued training	Salter–Harris type IV stress fracture
Miller et al. [42]	2M/F (mean age: 11.7 years)	Proximal radius	Gymnastics	In addition to the Salter–Harris type IV fractures, imaging revealed proximal radial metaphyseal edema and contralateral metaphyseal irregularity	Salter–Harris type IV epiphyseal stress injury
Santelli et al. [43]	3F (age range: 11–12 years)	Proximal radius	Gymnastics	Case 1: radiographs illustrated a Salter–Harris type IV fracture of the left proximal radial head with a chronic sclerotic appearance Case 2: radiographs demonstrated a Salter–Harris type IV fracture of the right radial head and an ill-defined lesion of the left radial head Case 3: radiographs demonstrated a Salter–Harris type IV injury of the radial head, which was later confirmed on MRI	Stress-related Salter–Harris type IV fractures
Salter–Harris typeV					
Maffulli et al. [39]	6M and 6F (age range: 11–18 years)	Proximal radius	Gymnastics	11 elbows (4 bilaterally) showed abnormalities of the radial head. A flattened radial head was evident in 4 elbows. In one gymnast, the radial head was inclined anteriorly	Salter–Harris type V partial growth arrest

Table 1 (continued)

References	No. of subjects (age)	Anatomic location	Sports played	Imaging findings	Diagnosis
Sato et al. [85]	1F (age 13 years)	Proximal tibia	Basketball	Radiographs showed that the lateral side of the right proximal tibial growth plate had closed, although other regional growth plates were still open. There was no deformity or length discrepancy of the tibia. MRI confirmed asymmetric and premature physeal closure, but there were no abnormal findings within the adjacent bone marrow and surrounding soft tissues	Chronic Salter–Harris type V injury
Schoffl and Schoffl [82]	1M/F (age range: 12.7–15.8 years)	Middle phalanx of long finger	Climbing sports	Radiographs revealed a crush injury of the physis bilaterally secondary to repetitive compressive forces	Salter–Harris type V stress injury

CT computed tomography, *F* female, *M* male, *MRI* magnetic resonance imaging

Fig. 3 Magnetic resonance imaging of a 13-year-old male climber with Salter–Harris type III fracture. Sagittal T1-weighted magnetic resonance image shows a vertically oriented, intra-articular fracture line in the epiphysis of the middle phalanx of the long finger. Note the primary periphyseal sports injury changes with asymmetric widening of the dorsal physis and decreased signal intensity within the juxtaphyseal metaphysis that reflects a combination of reactive marrow edema and sclerosis

widening is not a true fracture, although it may mimic a non-displaced Salter–Harris type I fracture on radiographs [92, 141]. On MRI, there is no tissue discontinuity, displacement, or fluid-like signal intensity fracture cleavage plane

through the physeal cartilage to suggest a fracture [92, 112, 141] (Fig. 5).

These findings are supported by vascular research on the growing bones of young animals [92, 145, 146]. Trueta and Amato [145] investigated the changes to the physis caused by interruptions to the metaphyseal and epiphyseal blood flow. When blood flow within the metaphyseal vessels was discontinued in young rabbits, calcification was reduced and the hypertrophied chondrocytes persisted, producing lengthening of the chondrocyte columns, which translates to growth plate widening. Further damage that also includes damage to the epiphyseal vessels can result in irreparable damage to the chondrocytes within the reserve and proliferative zones.

In a follow-up study, Trueta and Trias [146] investigated the impact that pressure had on the blood supply adjacent to the physis and found that persistent compression interfered with blood flow on one or both sides of the physis. However, despite exerting the same pressure on both sides of the growth plate, only the metaphyseal side was readily affected during the early stages of compression. Trueta and Trius [146] postulated that the solid "roof" constituted by the SOC or the epiphysis protects the underlying vessels responsible for nourishing the germinal and proliferative zones of the physeal cartilage. As a result, the epiphyseal blood supply is relatively more protected against excessive pressure than that on the metaphyseal side [146].

In a modified experimental design, Jaramillo et al. [133] used MRI with a histologic correlation to study abnormalities of the physis in rabbits after epiphyseal-sided and metaphyseal-sided injuries. They found that a metaphyseal-sided injury interfered with the normal process of endochondral new bone formation, producing focal thickening of the growth plate with long columns of hypertrophic

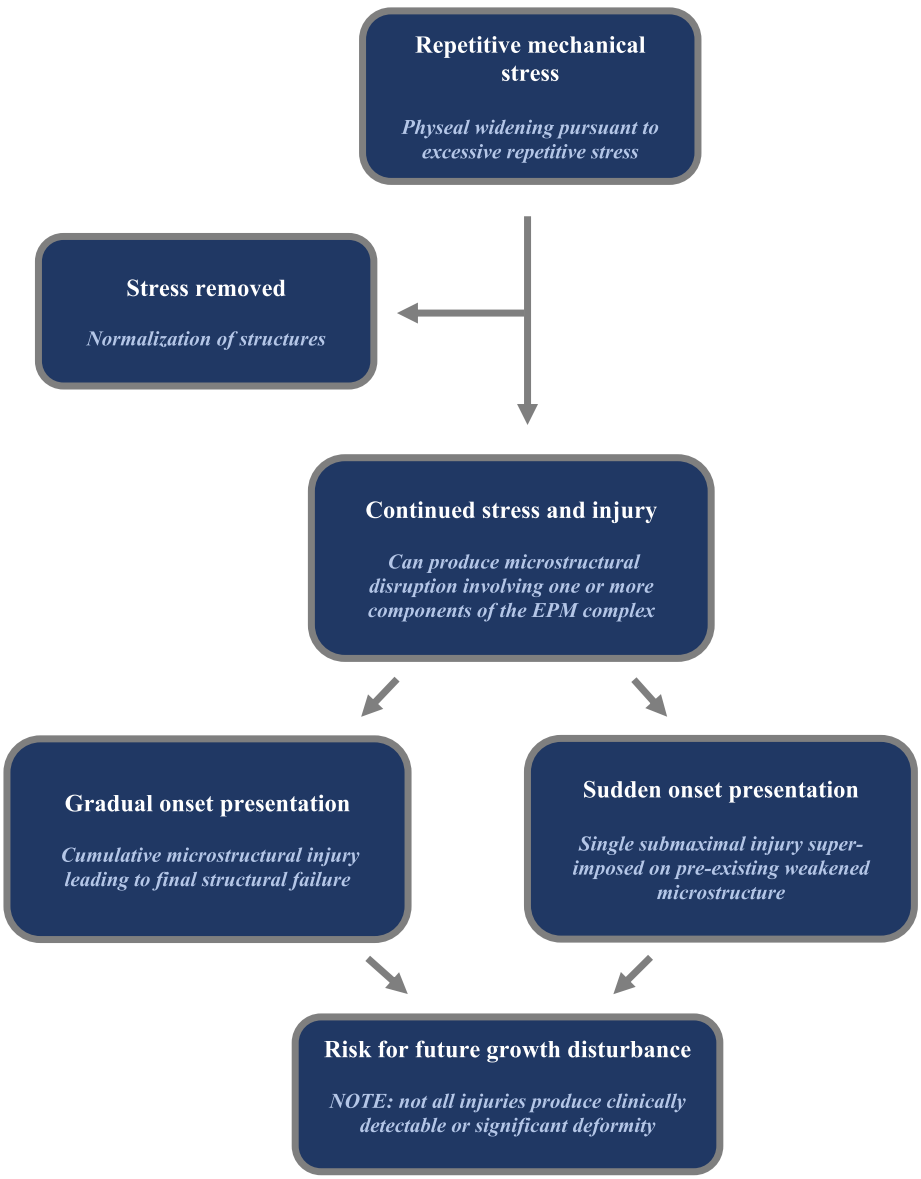

Fig. 4 Novel framework for understanding the pathophysiologic mechanisms and outcomes of primary periphyseal sports injuries

chondrocytes extending into the metaphysis [133]. Similar MRI findings have also been demonstrated in skeletally immature children who participate in high-level sports and sustain repetitive trauma [63, 114].

5.2 Stress Removed

With activity restriction and rest, healing occurs and normal osteogenesis resumes [92]. This outcome has been observed in stress-injured athletes who were treated with rest from the exacerbating activity [2, 8, 11, 12, 16, 17, 21, 33, 35, 45, 47, 49, 53, 56, 62, 88, 100]. The widening of the growth plate is usually reversible once normal perfusion is restored, as the resting and dividing cellular layers of the growth plate and the attendant epiphyseal blood supply are essentially undisturbed [137]. This leads to normalization of the growth plate and reconstitution of the zone of provisional calcification [7, 92, 137, 147] (Fig. 6).

This observation is supported by research in young animals that shows when no serious damage was caused by an indirect injury to the epiphyseal side, removal of compression resulted in very rapid total regeneration which was preceded by realignment of the vessels within the juxtaphyseal metaphysis [133, 137, 145, 146]. The apparent resilience of the zone of provisional calcification may be in part explained by the protective effect of the SOC or epiphysis. Xie et al. [144] reported the results from functional en vivo experiments, and mathematical modeling, which showed that the SOC has evolved to shield the growth plate chondrocytes, particularly the hypertrophic cells, from the deleterious effect of excessive mechanical stress. Thus, despite the relative susceptibility of these hypertrophic chondrocytes to mechanical stress, they may be particularly protected by the SOC, thus helping to preserve the mechanism of endochondral bone formation [144].

5.3 Continued Stress and Injury

Physeal widening from temporary deprivation of the metaphyseal blood supply to the hypertrophic chondrocytes may render it temporarily more mechanically susceptible to injury [137]. Progressive mechanical insults may lead to further injury of the EPM complex, especially if training is continued. Specifically, continued stress may produce microstructural disruption or stress fractures involving one or more components of the EPM complex, which can present with gradual or sudden onset, respectively. The hallmark in both scenarios is the gradual onset of subclinical symptoms and underlying stress-related physeal changes. Both scenarios can lead to long-term growth disturbance.

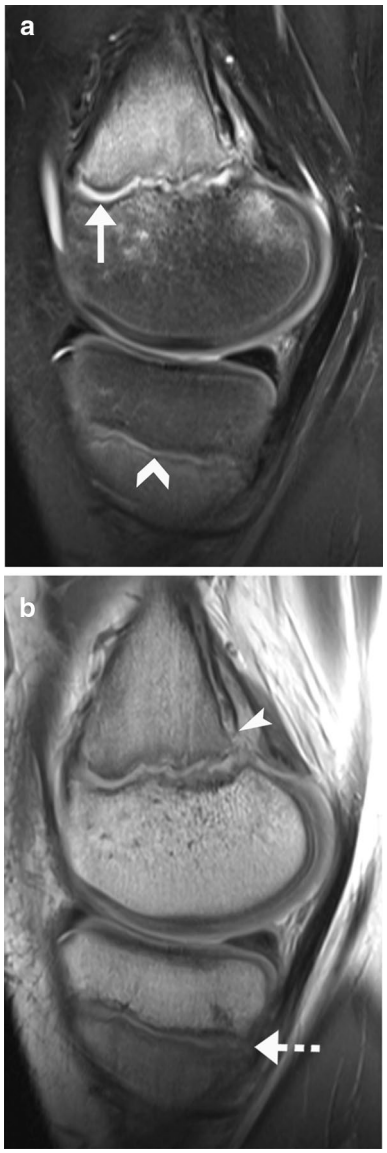

Fig. 5 11-Year-old male presents with a Salter–Harris type 1 fracture of the distal femur after a fall from the monkey bars. Sagittal T2-weighted magnetic resonance image (**a**) shows fluid-like signal intensity within the distal femur physis (arrow) when compared with the non-fractured proximal tibial physis (chevron). Corresponding intermediate-weighted magnetic resonance image (**b**) shows disruption of the posterior perichondrium and the torn end of the periosteum (arrowhead). Note the slight asymmetric thickness of the proximal tibial physis (dashed arrow) with subtle adjacent metaphyseal sclerosis that can be seen with primary periphyseal stress injuries

5.3.1 Gradual-Onset Presentation

In addition to the metaphyseal abnormality that is often present in PPSIs, there may be stress-related injury to the epiphysis [4, 51, 92, 133] and to the epiphyseal-sided blood supply responsible for nourishing the chondrocytes within

the resting and proliferative zones of the physis [92, 114, 133, 140]. Chondrocyte necrosis from complete ischemia leads to transphyseal bridge formation, which can be eccentric and cause asymmetric growth, or it may involve the entire physis and cause reduced or complete cessation of further longitudinal growth [4, 92].

This phenomenon has been demonstrated in animal models where damage caused by compression on the epiphyseal side of the growth plate correlated directly and proportionately with growth interference [146]. In these models, the duration and severity of this compression impact the microvascular blood supply and can lead to variable degrees of permanent growth arrest and long-term deformity [148–157]. In youth athletes, there are similar reports of PPSIs that have led to growth disturbance, including cases resulting in angular deformity [13, 24, 31, 36, 38, 39, 43, 49, 54, 55, 58, 60, 61, 64, 66–68, 81, 82, 84, 85, 92, 99, 102] (Fig. 7).

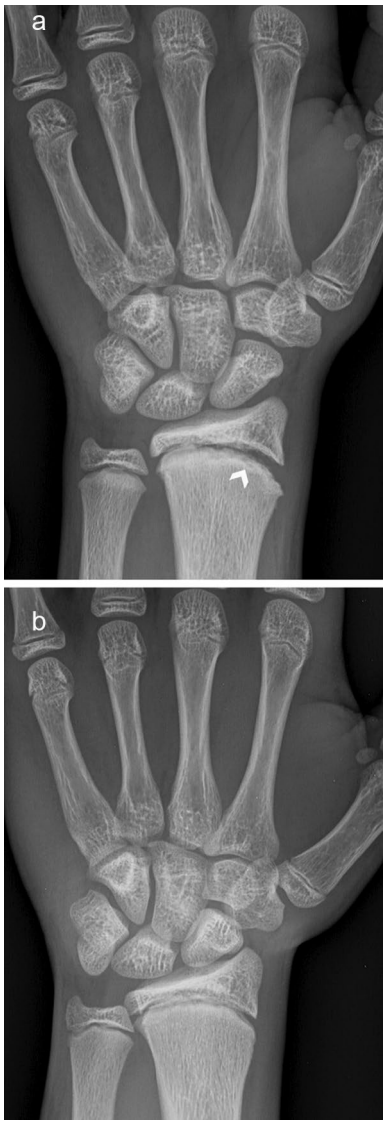

Fig. 6 11-year-old girl gymnast with left wrist pain that worsened in the previous 2 months. Posteroanterior wrist radiograph (**a**) shows an irregularly widened appearance of the distal radial physis (chevron) that likely also involves the distal ulnar physis, but to a lesser extent, with adjacent reactive metaphyseal sclerosis. Repeat radiograph (**b**) obtained 6 months later, after complete cessation of weight-bearing activity on the upper extremities, shows near complete normalization of these physes and the patient was symptom free

5.3.2 Sudden-Onset Presentation

If metaphyseal stress injuries are undiagnosed or sub-optimally treated, progressive mechanical insults may also lead to stress fractures involving one or more components of the EPM complex [4, 81, 82]. Ogden [158] analyzed the transphyseal linear ossific striations of the distal ulna and radius in child cadavers. He showed that these striations consist of trabecular bone extending from the metaphysis across all zones of the physis into a small focus of fibrous and necrotic tissue within the epiphyseal cartilage. He postulated that microtrauma is the mechanism responsible for their occurrence, which produced focal ischemia in the epiphyseal cartilage and germinal zone [158].

Interestingly, DiFiori and Mandelbaum [63] observed linear areas of high signal intensity on MRI consistent with vertical fractures within the radial metaphysis of a young artistic gymnast who complained of bilateral dorsal-sided wrist pain. Similarly, Shih et al. [114] reported MRI findings of bone bruising (17/92 = 18.5%), horizontal fractures (23/92 = 25%), and vertical fractures (8/92 = 8.7%) in the distal radial metaphyses of Chinese youth who undertook 2 h daily of gymnastic floor exercise training, 6 days per week, for 10 months per year. These findings underscore the vulnerability of these rapidly growing regions to injury [114].

This phenomenon has also been observed in animal research. In a study using a variable strain-rate machine, Bright et al. [159] loaded the proximal tibial epiphyses of skeletally immature Sprague–Dawley rats. On histological examination of tibiae loaded to 50% of failure energy, internal cracks within the plates were present that were oriented in planes that received the highest shear stress. These cracks were usually included in the eventual path of the ultimate failure crack. Bright et al. [159] concluded that these internal cracks reflect the first event in the final failure of the growth plate.

Microdamage as a material fatigue phenomenon has been used to describe the pathomechanistic failure of bone, fibrous tissue, and cartilage [160, 161]. Specifically, repetitive loading of a rigid or semi-rigid structure may cause microscopic cracks that can enlarge progressively with additional loading cycles [160]. Eventually, only a small portion of the structure remains intact and a final load (which is much smaller than the structure's original failure load)

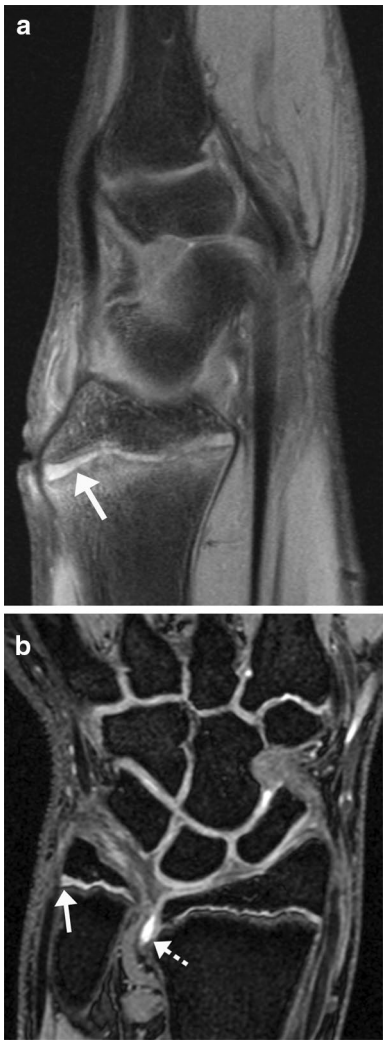

Fig. 7 12-year-old female gymnast with wrist pain. Sagittal intermediate-weighted fluid-sensitive magnetic resonance image (**a**) shows a thickened distal radial physis, more severe dorsally (arrow). Coronal double-echo steady state-weighted magnetic resonance image (**b**) also shows an asymmetrically thickened distal ulna physis, ulna-positive variance, and widened distal radioulnar joint (bracket), with trace effusion (dashed arrow)

may lead to a complete fracture [161]. This may explain the apparent sudden onset of Salter–Harris stress fractures, particularly involving the proximal radius in gymnasts [162], and middle phalanx in climbers [82]. Thus, stress fractures involving the EPM complex may represent a stress-related continuum that summates multiple smaller repetitive insults that ultimately lead to complete failure [63, 158]. This process may involve an on-going interplay between repetitive and acute mechanisms, leading to a frank fracture.

As summarized in Table 1, stress fractures in skeletally immature athletes typically involve two or more components of the EPM complex. These athletes may participate in a variety of high-impact repetitive youth sports, including baseball, basketball, gymnastics, rugby, running, and sport climbing, and sustain stress fractures involving the shoulder, wrist, hand, knee, and foot [8, 22, 30, 32, 38–43, 48, 50, 51, 65, 74, 75, 77–79, 81, 82, 84, 85, 88, 93, 94, 96, 98, 101] (Fig. 8). Although most of these stress fractures appeared to heal without complications in a short-term follow-up, several instances of growth disturbance have been reported [38, 39, 42, 43, 82, 85, 101].

5.4 Pathways to Future Growth Disturbance

Clinically, it is important to know how bone growth is influenced and can be altered by physiologic and excessive mechanical loading, respectively [163]. The association between loading and longitudinal bone growth is well established [163, 164]. Within the physiologic loading range, bone growth is either stimulated in tension or slowed in compression [163, 164]. When the range is exceeded in compression, growth is halted [164]. With earlier and increased participation of youth in sport, a more precise delineation between physiologic and excessive loading is critically important in optimizing patient treatment and reducing the risk for injury.

Growth disturbance, either longitudinal or angular, is the most feared complication of PPSI [136]. Not all PPSIs carry the same risk for growth disturbance [136]. Factors affecting the likelihood of subsequent growth disturbance pursuant to PPSI include severity and duration of injury, anatomic site, and remaining growth potential [4, 165]. A recent systematic review found that most PPSIs (391/448; 87.3%) were not associated with a permanent growth disturbance or a clinically actionable deformity at a short-term follow-up [2]. However, subclinical or temporary growth cessation and long-term deformities may be underdiagnosed and underreported. In particular, as physeal widening reflects indirect injury to metaphyseal perfusion, it is unclear to what extent this alteration resolves once normal metaphyseal osteogenesis resumes.

While metaphyseal insults predominate, associated injury to the growth plate and epiphysis can occur, and explains the cases of permanent growth arrest and long-term deformity previously reported [13, 24, 31, 36, 38, 39, 49, 51, 54, 55, 58, 60, 61, 64, 66–68, 81, 82, 85, 92, 95, 99, 102]. Two principal pathophysiologic pathways have been proposed, depending on whether the injury onset is indirect or direct [4].

In the indirect pathway, epiphyseal vascular compromise following epiphyseal injury leads to inadequate nourishment and cell death of chondrocytes within the juxta-epiphyseal growth plate (reserve and upper proliferative zones) [133, 166, 167]. The proliferative zone is most sensitive to alterations in perfusion because oxygen tension is highest in this

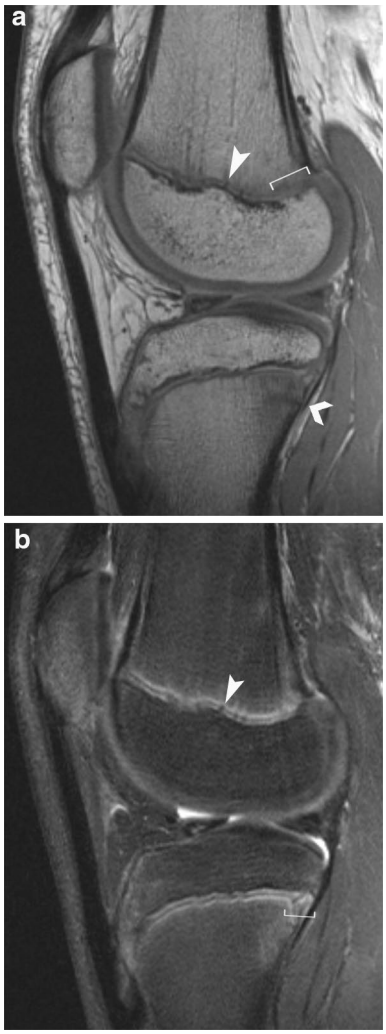

Fig. 8 12-year-old male baseball player with knee pain, stiffness, swelling, and a reduced range of motion. Sagittal intermediate-weighted (**a**) and T2-weighted fluid-sensitive (**b**) magnetic resonance images show subtle non-uniform thickening of the regional physes that contain transphyseal foci of low signal intensity (arrowheads), focal loss of the zone of provisional calcification (brackets), and a superimposed bone stress injury (chevron)

zone to facilitate robust aerobic metabolism and mitochondrial uptake of calcium [4]. This injury may result in a spectrum of epiphyseal deformities and physeal injuries. Imaging findings include fragmented or deformed epiphysis, blunted or cessation of longitudinal growth with or without the formation of focal transphyseal bar, or diffuse physeal closure.

In the direct pathway, an acute injury is superimposed on a repetitive mechanism producing Salter–Harris type fracture patterns (Table 1). In these injuries, damage to the epiphyseal-sided vessels may progress to the formation of micro-stress-fractures, which, in turn, may lead to complete micro-stress fractures, including those that pass through the physis with potential to harm chondrocytes within the reserve and proliferative zones. Death of chondrocytes within these zones may produce focal transphyseal bridge formation or diffuse premature physeal closure, depending on the extent of injury. In contrast to indirect pathway injuries, the epiphysis is not always involved in the direct pathway.

As summarized by Ogden [137], growth insult arising from either pathway may be localized and cause asymmetric growth, or it may involve the entire or near-entire physis and result in an overall slowdown or even complete cessation of growth. In either instance, premature closure of some or all of the physis may occur. Depending on the remaining growth potential, the altered growth can lead to significant long-term complications.

6 Summary and Conclusions

Our purpose for this article was two-fold: first, to highlight the inconsistent and imprecise nomenclature historically used to describe PPSIs; and second, to improve the clarity and understanding regarding the pathophysiologic mechanisms for PPSIs. Here, we provide an examination of these inconsistent nomenclatures used historically to describe PPSIs and to propose an explanation on the nature and cause of these injuries, and to suggest the use of a more uniform terminology moving forward.

We introduced a novel framework (Fig. 4) to help clarify the nature and pathophysiologic mechanisms involved in PPSIs. This framework includes repetitive mechanical stress, stress removed, continued stress and injury (gradual-onset presentation and sudden-onset presentation), and the risk for future growth disturbance. It is important to recognize that PPSIs typically involve more than one component of the EPM complex. Physeal widening, the most common PPSI manifestation, initially affects the metaphysis and, secondarily, the physis. Repetitive loading alters metaphyseal perfusion and, in doing so, indirectly interferes with endochondral ossification.

The widening of the growth plate mostly involves chondrocytes within the hypertrophic zone and is typically reversible by removal of the offending stressors, allowing restoration of normal metaphyseal perfusion. Most case reports and case series of PPSIs indicate that metaphyseal insult (i.e., physeal widening) typically resolves with rest during a short-term follow-up and that most stress-injured athletes are eventually able to return to their former levels of participation, although in some cases, at a different position or decreased competitive level, at least initially.

Although physeal widening predominates, associated injury of the growth plate and epiphysis can also occur, which explains the cases of permanent growth arrest and

long-term deformity. Continued mechanical stress applied to undiagnosed and sub-optimally treated PPSIs may lead to microstructural disruption or stress fractures involving one or more components of the EPM complex. The hallmark in each scenario is the gradual onset with insult to the epiphyseal vascular supply and subsequent ischemic change to the physis. However, with stress fractures, a sudden-onset presentation may be superimposed on a repetitive mechanism, resulting in fracture at submaximal load.

We described two pathways by which stress injury to the epiphysis and epiphyseal-sided vessels may cause growth disturbance. In repetitive mechanical stress with gradual-onset presentation, or indirect injury, epiphyseal vascular compromise leads to inadequate nourishment of the juxta-epiphyseal growth plate (the reserve and proliferating zones). In repetitive mechanical stress with sudden-onset presentation injury, there may be cartilage failure resulting from submaximal loading. This stress-related injury continuum ranges from small, isolated, micro-trabecular or focal fractures to cortical disruption with cleavage plane that passes through one or more components of the EPM complex. Both pathways may result in the death of chondrocytes within the reserve and proliferative zones, producing focal transphyseal bridge formation or diffuse premature physeal closure, depending on the extent of injury.

Against this background, we provide recommendations below for a revised nomenclature. It is our hope that these suggestions will help to facilitate more accurate and effective communication among healthcare specialists and researchers, allowing optimization of patient care, facilitating ongoing research and translation of these new discoveries into clinical practice.

1. Adopt "primary periphyseal stress injury" or "PPSI" as an umbrella term to describe stress-related injuries involving one or more components of the EPM complex.
2. Replace sport-specific descriptors for PPSIs (e.g., LLS, gymnast's wrist) with more inclusive mechanism-based terminology to describe these injuries. For example, use "proximal humerus PPSI" to describe what has formerly been referred to as LLS.
3. "PPSI with physeal widening" rather than "Salter–Harris I fractures" should be used to refer to stress-related physeal widening when a superimposed fracture is not confirmed.

We recognize that the use of the Salter–Harris classification to designate stress fractures of the EPM complex will not be easily converted or abandoned by clinicians given the many decades of usage. We also appreciate that stress fractures involving the EPM complex may eventually produce Salter–Harris-type fracture patterns. However, the Salter–Harris classification was designed for traumatic growth plate injuries in children. We suggest, instead, that clinicians consider using the term "PPSI with extension of stress fracture line into adjacent bone" (e.g., metaphysis, epiphysis) to describe these injuries.

It was not our purpose to propose a new classification system for PPSIs, but rather to propose more appropriate terminology given the current state of knowledge. However, given the short-comings of the Salter–Harris classification for describing these injuries, it follows that there is a need for the future development and testing of a more precise imaging-based classification to grade PPSIs that can be used to guide appropriate treatment.

We hope that this article has helped to expose a pattern of inconsistent and imprecise terminology used for describing PPSIs. We also hope that our description of the pathophysiologic mechanisms underlying PPSIs will help to provide clarity to this area that will facilitate more uniform clinical practice and stimulate future research.

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Declarations

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Conflict of interest Dennis Caine, Nicola Maffulli,Rachel Meyers, Volker Schöffl, and Jie Nguyen all declare that they have no conflicts of interests relevant to the content of this review.

Author contributions DC, JN and RM conceptualized this article. RM assisted DC with the systematic search and retrieval of the related literature. The first draft of the manuscript was written by DC and JN. JN provided a critical review of the work and assisted DC with the conceptualization of the novel framework for understanding the pathophysiologic mechanisms of PPSIs. JN, NM, and VS provided imaging and clinical interpretation of PPSIs. JN and VS provided the images used in the manuscript. All authors commented on previous versions of the manuscript, revising for intellectual content, and reviewed and approved the final manuscript.

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Consent for publication Not applicable.

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