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The progress of research on crankshaft phenomenon

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Abstract

The Crankshaft Phenomenon (CSP) is a significant complication that can occur after posterior spinal fusion, particularly in growing patients with scoliosis. It results from continued anterior spinal growth while the posterior column remains fused, leading to progressive spinal deformities such as loss of correction, increased vertebral rotation, and rib prominence. This phenomenon has been predominantly observed in pediatric patients with idiopathic, congenital, and neuromuscular scoliosis. Although clinical symptoms may be subtle, radiographic signs are crucial for diagnosis but can be challenging to evaluate due to postoperative changes and instrumentation. Current treatment options are limited, often requiring revision surgeries in cases of progressive deformities. This review aims to provide a comprehensive overview of the current understanding of CSP, including its pathophysiology, diagnostic challenges, risk factors, prevention strategies, and potential treatments.

Keywords Crankshaft phenomenon, Spinal fusion, Scoliosis, Vertebral rotation, Pediatric orthopedics, Spinal deformities

Introduction

The Crankshaft Phenomenon (CSP) was first described by Ponseti in 1950 as the progression of deformities following posterior spinal fusion. In 1957, Hallock et al. identified that posterior fusion in growing spines acts as a tether, aggravating deformities in the fused segments [1]. Roaf, in 1960, highlighted that significant growth imbalance between spinal regions renders posterior fusion insufficient to halt deformity progression. Dubousset formalized the term "Crankshaft Phenomenon" in 1973, describing how anterior vertebral growth around the tether of posterior fusion causes rotational distortion, initially observed in paralytic scoliosis cases and later in congenital and idiopathic scoliosis [2].

CSP is now defined as the recurrence or worsening of spinal deformities in growing children with scoliosis following isolated posterior fusion (Fig. 1). The phenomenon stems from continued anterior column growth in fused segments, primarily manifesting as exacerbated rotational deformities [3].

CSP is a significant complication of scoliosis surgery, often resulting in poor surgical outcomes, progressive deformities, and, in severe cases, the need for reoperation [4]. This review summarizes the current progress in understanding and managing the crankshaft phenomenon.

Physiopathology

Spinal growth originates from the apophyseal joints, the upper and lower vertebral endplates, and the neurocentral synchondrosis (NSC) (Fig. 2). NCS, a critical growth region connecting the vertebral body and pedicles, typically exhibits symmetrical growth on both sides [5, 6]. However, when NCS growth becomes

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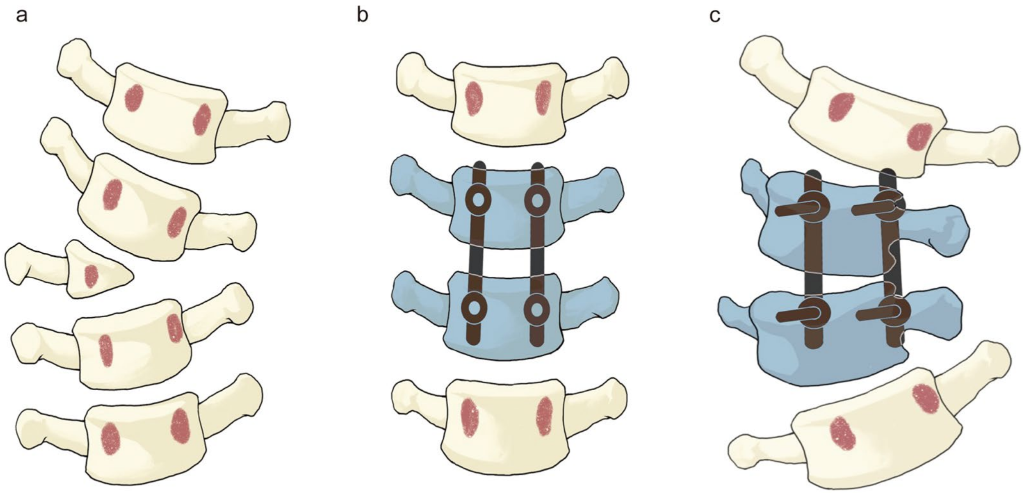

Fig. 1 Diagrams of the spine illustrating the progressive rotational deformities characteristic of the crankshaft phenomenon, **A**: Preoperative, **B**: Immediate Postoperative, **C**: Last Follow-up

asymmetric, it may lead to unequal development of the pedicles and vertebral body, resulting in vertebral rotation and scoliosis [7–9]. Studies have demonstrated that unilateral epiphyseal fixation of the NCS with screws disrupts growth on the affected side, leading to asymmetry between the pedicle and vertebral length, ultimately causing spinal curvature [10]. The closure timing of NCS varies across different spinal segments: it closes around the age of 5 in the cervical spine, nearly completes closure in the lumbar spine by age 11, and remains open until approximately age 17 in the thoracic spine [11]. Theoretically, after posterior spinal fusion, the endplates and NCS retain some growth potential, whereas anterior spinal fusion may inhibit NCS growth by forming a bony bridge at the pedicle base. As a result, continuous growth following posterior fusion may contribute to the development of the crankshaft phenomenon, whereas anterior fusion might provide some preventive effect.

Due to the spine's ability to rotate in three dimensions, any imbalance in growth can lead to spinal deformities [2]. This principle helps explain congenital scoliosis: the faster growth of hemivertebrae on the convex side elongates the vertebral segment, while restricted growth on the concave side, due to the presence of a bony bridge, results in vertebral shortening. This mechanism also accounts for the effective correction of curvature in young patients with idiopathic scoliosis after convex fusion. Post-fusion, growth on the convex side slows or ceases, while the concave side continues to grow, leading to the straightening of the curvature. Winter et al. (1981) observed that posterior spinal fusion performed after spinal growth concludes halts the progression of deformities, indicating that crankshaft deformity arises from continued growth within the fused segment [12].

Dubousset et al. (1989) further highlighted that the crankshaft phenomenon predominantly occurs in cases of rigid posterior fusion with reliable internal fixation. In such scenarios, sagittal deformities may be influenced by both anterior and posterior fusion. The solid posterior fusion restricts growth posteriorly, while anterior vertebral growth persists, resulting in rotational deformities characterized by vertebral displacement toward the convex side and exacerbated rib hump [2]. Using CT-based 3D reconstruction, Dubousset et al. demonstrated that after posterior fusion, apical vertebral rotation intensifies, with continued anterior growth contributing to increased kyphosis, effectively signifying an accentuation of convex-side vertebral rotation [2].

There is ongoing debate regarding the growth potential of fusion masses following posterior fusion. Risser et al. suggested that fusion masses in young patients possess a degree of biological plasticity, allowing for continued growth and curvature. Conversely, scholars like Hefti and McMaster have argued that in adolescents with idiopathic scoliosis, fusion masses cease growing after posterior fusion [13]. The prevailing consensus is that posterior fusion halts growth in the fused region, while anterior growth continues, contributing to spinal deformities. The severity of these deformities correlates with the number and growth potential of anterior ossification centers.

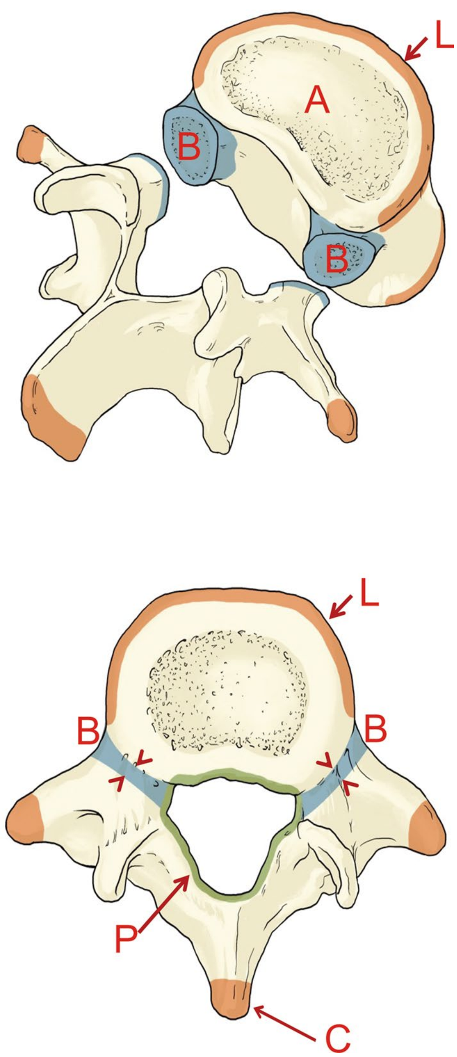

Fig. 2 A diagram highlighting the growth regions of vertebral bodies shows that development at the listel (L), or ring apophysis, initiates around ages 7–9 and completes fusion between ages 14 and 24, growth areas include the end plates (**A**), the neurocentral bipolar region (**B**, fusing by ages 7–8), the posterior elements (**C**), and periosteum remodeling zones (P)

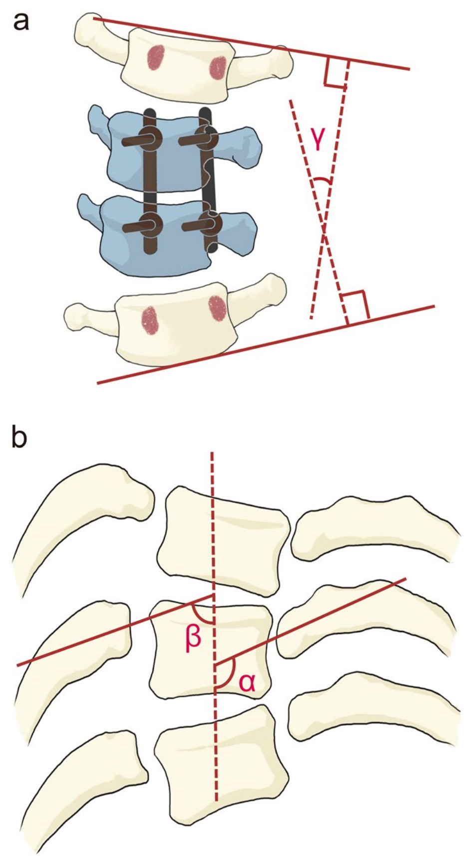

Fig. 3 A diagram illustrating the Cobb angle and the rib-vertebral angle difference (RVAD), **A**: γ represents the Cobb angle, **B**: The rib vertebral angle difference (RVAD) is calculated as the difference between the rib-vertebral angles on both sides, expressed as: RVAD = α − β

Diagnosis

Diagnosing crankshaft phenomenon (CSP) presents challenges due to difficulties in measurement despite its conceptual simplicity. Postoperative X-rays during follow-up are commonly used for assessment, focusing on coronal Cobb angle, vertebral rotation, rib vertebral angle difference (RVAD), vertebral translation, trunk shift, rib deformities, and internal fixation length. Among these, Cobb angle and RVAD are more accurate and reproducible, whereas vertebral translation is often limited by posterior fusion blocks, reducing its practicality. Typically, X-rays taken around one year postoperatively serve as the diagnostic basis, as the spine may undergo stress relaxation and anterior column growth absorption during this period [14].

Coronal Cobb angle

Initially introduced by John Robert Cobb to classify scoliosis, the Cobb angle has become a standard for assessing deformities in both coronal and sagittal planes [15]. Measuring the Cobb angle from upright anteroposterior radiographs is widely used to evaluate CSP (Fig. 3a). Delorme et al. conducted a study on the crankshaft phenomenon following surgery for idiopathic scoliosis, defining the diagnostic criteria for this phenomenon as an increase in the postoperative Cobb angle of ≥10° [16]. However, as CSP often manifests through rotational changes, Cobb angle alone may not capture its full extent.

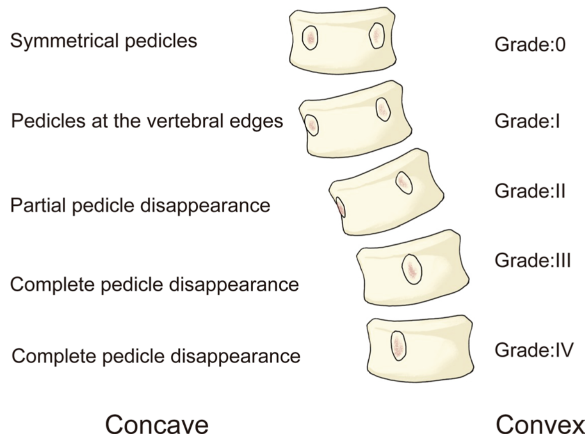

Fig. 4 The Nash–Moe method is utilized for assessing vertebral rotation

Vertebral rotation measurement

In 1948, Cobb first introduced a method for assessing vertebral rotation, which classified the degree of rotation based on the position of the spinous process. Nash and Moe identified difficulties in distinguishing vertebral rotation using Cobb's method and proposed an alternative approach in 1969, utilizing pedicle projection as a marker, now known as the Nash–Moe method [17] (Fig. 4). Due to the limitations in accuracy of the Nash–Moe method, Perdriolle (1985) developed a new measurement technique. This method employs a specialized template to measure pedicle displacement and calculates the degree of spinal rotation through a series of numerical computations [18]. However, postoperative fusion blocks and internal fixation devices often obscure anatomical landmarks, complicating measurement procedures. Lee et al. observed that the Cobb method and Perdriolle method had mean errors of 1.7° and 3.7°, respectively [14]. They recommended obtaining X-rays 10–12 months after surgery, following instrumentation removal, as the optimal reference for diagnosing crankshaft phenomenon (CSP), which is defined as vertebral rotation exceeding 5° or progression in the Cobb angle.

Rib vertebral angle difference (RVAD)

Mehta described RVAD in 1972 as the angular difference between the vertebra and rib at the curve apex, with the convex-side rib prominence reflecting vertebral rotation [19] (Fig. 3b). RVAD is considered a reliable indicator of CSP [20]. Hamill and Sanders found RVAD changes more sensitive than Cobb angle or Perdriolle rotation angle in detecting CSP [21, 22]. Lapinsky defined CSP as a Cobb angle or RVAD change greater than 10°, while Burton suggested scoliosis progression by 5° outside fused segments as indicative of CSP [23, 24].

Risk factors

Crankshaft phenomenon is primarily influenced by spinal growth potential, deformity type, and surgical factors, with growth potential being the most critical.

Growth potential

Physiological age: Indicators include puberty markers such as menarche, Tanner staging, and secondary sexual characteristics. Roberto et al. identified Tanner stages I-II as high risk, with a 52% incidence, while stages III–IV showed no occurrences [25].

Bone age: Common measures include Risser sign and triradiate cartilage [2, 21, 22, 26, 27]. Sanders et al. found crankshaft occurred only in Risser ≤ 2 cases, although Risser sign accuracy has been questioned [28]. Closure of triradiate cartilage is a stronger predictor [25]. In a follow-up, patients with closed cartilage had significantly lower crankshaft rates than those with open cartilage ($P = 0.004$) [22].

Peak height velocity (PHV): PHV, marking peak skeletal growth, is linked to crankshaft risk. Sanders et al. reported that surgeries before PHV, characterized by height increases ≥ 8 cm/year in girls and ≥ 9.5 cm/year in boys, resulted in higher crankshaft rates [29].

Predictive accuracy decreases as follows: age < 10 years, open triradiate cartilage, Risser 0, and premenarche [30, 31].

Deformities types

The incidence of crankshaft phenomenon varies significantly across different types of scoliosis. For idiopathic scoliosis, studies show that the incidence of crankshaft phenomenon in patients with Risser stage 0 and open triradiate cartilage after undergoing posterior spinal fusion is between 37 and 54% [22]. Age under 10 years and surgery during peak height velocity (PHV) are high-risk factors. Research by Lee et al. found that a bone age under 10 years, age under 11 years, and a top vertebra RVAD greater than 20° are significantly associated with the occurrence of the crankshaft phenomenon [14, 21, 25, 29, 32, 33]. In patients with neuromuscular scoliosis, the incidence of crankshaft phenomenon is relatively low when treated with posterior U-shaped rod fixation, and routine anterior epiphyseal arrest is not necessary [34]. For congenital scoliosis, the incidence of crankshaft phenomenon ranges from 15 to 30%, with age under 4 years and preoperative Cobb angle greater than 50° being the main risk factors [35–38]. Overall, the incidence of crankshaft phenomenon is higher in idiopathic scoliosis than in congenital scoliosis.

Surgical factors

The crankshaft phenomenon is influenced by factors such as the length of the posterior fusion segment, the severity of the preoperative deformity, and the residual deformity after surgery. Studies on the relationship between posterior internal fixation types and crankshaft phenomenon are limited.

Sanders et al. found that younger patients treated with the Harrington system had the most significant postoperative changes, while segmental fixation systems (TSRH, CD systems) in slightly older patients still caused 1–2 grades of crankshaft phenomenon, suggesting these systems do not fully prevent it [22]. Kioschos et al. observed that posterior spinal fusion with pedicle screw fixation could prevent kyphosis and control anterior spinal growth, leading to mechanical epiphysiodesis [39]. Burton et al. found that strong fixation at both ends of the curve, particularly with pedicle screw systems, could prevent the crankshaft phenomenon, even in patients with unclosed triangular cartilage [24].

Dubousset et al. noted that the presence of more normal vertebral segments within the fused segment increased the likelihood of crankshaft phenomenon [2]. Terek et al. found that complete segmental hemivertebrae increased the likelihood of crankshaft [35]. Shufflebarge et al. and Tredwell et al. identified preoperative Cobb angles > 60° and apex vertebral rotation > 20° as risk factors [27, 40], while Lee et al. found a trend toward higher Cobb angles in children with the phenomenon [14].

Recent advances

A multicenter retrospective cohort study published in May 2024 evaluated CSP in early-onset scoliosis (EOS) patients using an inverse trigonometric function. The study analyzed pedicle screw rotation angles to identify CSP prevalence and associated factors in 50 EOS patients aged ≤ 11 years, with follow-up extending up to five years. Results revealed a CSP incidence of 30%, strongly associated with shorter preoperative T1–T12 lengths. This novel method provides a quantitative approach to CSP evaluation, offering valuable insights for surgical planning and postoperative management in EOS patients [41].

In summary, key risk factors for the crankshaft phenomenon include being under 10 years old, open triradiate cartilage, Risser sign 0–1, premenarche, idiopathic scoliosis, residual deformity > 60° post-surgery, and apex vertebral rotation > 20°. No single indicator can predict its occurrence.

Prevention

There are various perspectives on preventing the crankshaft phenomenon. Some suggest combining anterior and posterior surgeries for patients with incomplete skeletal development, especially before or during growth. However, no consensus exists on the optimal preventive approach.

Dubousset et al. recommended using a brace for Risser grade 0 patients until grade 1 after fusion surgery [2]. Shufflebarger and Clark performed anterior growth arrest and fusion before posterior fusion in 8 high-risk patients, with no crankshaft phenomenon observed until skeletal maturity [27]. Dohin and Dubousset treated 12 cases with combined anterior and posterior fusion, achieving satisfactory outcomes in 11, while one case experienced internal fixation failure and deformity progression [42]. Lapinsky and Richards found that combined surgery was superior to posterior fusion alone in preventing crankshaft phenomenon [23]. Some researchers argue that crankshaft phenomenon is not severe and does not require routine anterior fusion [43]. Lee and Nachemson's analysis of 63 idiopathic scoliosis cases showed an average increase of 3° in scoliosis angle and axial rotation, with a 9° increase in children under 10 years old, suggesting that routine anterior fusion may not be necessary [14]. Mullaji et al. followed 30 idiopathic scoliosis cases treated with posterior fusion and found minimal changes in scoliosis and rib-vertebra angles, questioning the need for combined surgery [44]. Kioschos et al. found that posterior fusion with pedicle screw fixation inhibits anterior spine growth, preventing the crankshaft phenomenon without requiring anterior fusion [39]. Betz et al. attempted to compare the effects of anterior and posterior fusion but could not draw definitive conclusions due to limited data [45].

Several authors, including Leonard and Lapinsky, support combined anterior and posterior surgery to improve correction and prevent crankshaft [24, 27]. Dubousset et al. (1989) suggested anterior fusion for patients with Risser sign 0–1, significant deformity, and expected spine growth over 2 years [2], while Sanders et al. (1995) recommended anterior fusion for patients under 10 years old or premenarcheal, with unclosed "Y" cartilage and prior to peak height velocity (PHV) [22]. Mullaji et al. argued that in idiopathic scoliosis with immature skeletons, wearing a brace post-surgery can prevent crankshaft, and routine anterior fusion is not necessary [44].

In conclusion, combined anterior and posterior approaches are effective for preventing crankshaft, but robust posterior fixation alone may also prevent the phenomenon. Studies have shown that strong internal fixation systems can inhibit anterior growth, making anterior fusion unnecessary in some cases.

Treatment

The treatment of the crankshaft phenomenon has been minimally documented, but several studies have examined the efficacy of various strategies. Barrios et al. demonstrated that anterior epiphyseal block fusion performed via thoracoscopy effectively prevented further deformity progression [43]. Wenger et al. suggested that in patients with postoperative crankshaft phenomenon accompanied by significant spinal deformity, anterior epiphyseal block fusion with grafting can initially be utilized to halt progression. For more severe deformities, a combination of anterior surgery and posterior osteotomy with fusion may be necessary to achieve better correction [46].

Dubousset et al. reported a case of a 9-year-old boy with congenital paralytic scoliosis who experienced unacceptable deformity progression six years after surgery. The patient underwent anterior three-level osteotomy with Dwyer fusion, followed by posterior correction using Harrington instrumentation and fusion, achieving favorable outcomes [2]. Similarly, Lee et al. described a patient whose Cobb angle and apical vertebral rotation advanced by 15° and 18°, respectively. The patient achieved satisfactory results through extended segmental instrumentation and fusion [14]. Moreover, a study involving 638 pediatric patients with spinal deformities identified 50 cases requiring revision surgery due to complications such as decompensation, pseudoarthrosis, and crankshaft phenomenon, with eight specifically addressing the latter. These cases were managed primarily using posterior column osteotomies and combined anterior–posterior fusion techniques [47]. Bourghli et al. provided the first comprehensive case report detailing surgical management of the crankshaft phenomenon. Using a posterior-only approach, they performed posterior column osteotomies at the apex of the deformity to release prior fusion. This method safely and effectively restored proper coronal and sagittal alignment, yielding excellent clinical and radiological long-term results [48].

Conclusion

The Crankshaft Phenomenon remains a major challenge in the management of pediatric scoliosis, particularly in patients with residual growth potential. Despite advancements in surgical techniques, diagnosis remains difficult due to the subtlety of clinical signs and the complexities of radiographic evaluation. Key risk factors such as open triradiate cartilage, preoperative deformity severity, and patient age must be carefully considered when planning treatment. While combined anterior–posterior surgery shows promise in preventing CSP, robust posterior fixation alone may also suffice in certain cases. Future research should focus on refining diagnostic criteria and exploring innovative preventive and corrective strategies to minimize the incidence of this debilitating complication.

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Author contributions
Z.P: Conceptualized and wrote the initial draft of the manuscript. B.H was responsible for creating the figures. S.W and J.Z gave the final approval for the version to be submitted.

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Availability of data and materials
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Declarations

Ethics statement
Not applicable.

Competing interests
The authors declare no competing interests.

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