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REVIEW ARTICLE

The growing spine: how spinal deformities influence normal spine and thoracic cage growth

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Abstract
Purpose This article aims to provide an overview of how spinal deformities can alter normal spine and thoracic cage growth.
Methods Some of the data presented in this article are gathered from studies performed in 1980 and 1990, and their applicability to populations of different ethnicity, geography or developmental stage has not yet been elucidated. In the present article, older concepts have been integrated with newer scientific data available to give the reader the basis for a better understanding of both normal and abnormal spine and thoracic cage growth.
Results A thorough analysis of different parameters, such as weight, standing and sitting height, body mass index, thoracic perimeter, arm span, T1–S1 spinal segment length, and respiratory function, help the surgeon to choose the best treatment modality. Respiratory problems can develop after a precocious vertebral arthrodesis or as a consequence of pre-existing severe vertebral deformities and can vary in patterns and timing, according to the existing degree of deformity. The varying extent of an experimental arthrodesis also affects differently both growth and thoracopulmonary function.
Conclusions Growth is a succession of acceleration and deceleration phases and a perfect knowledge of normal growth parameters is mandatory to understand the pathologic modifications induced on a growing spine by an early onset spinal deformity. The challenges associated with the growing spine for the surgeon include preservation of the thoracic spine, thoracic cage, and lung growth without reducing spinal motion.

Keywords Early onset spinal deformities · Thoracic cage · Growth · Spine

Introduction

This article aims to provide a comprehensive review of how spinal deformities can affect normal spine and thoracic cage growth. Some of the data presented are from studies performed in the 1980s and 1990s and their applicability to different ethnic populations or developmental stages remains to be elucidated. The "universal" applicability of the values provided is therefore open to debate. However, older concepts have been integrated with newer scientific data to give the reader the basis for a better understanding of both normal and abnormal spine and thoracic cage growth.

From normal to abnormal growth

Only perfect knowledge of normal growth parameters allows a better understanding of both normal and abnormal spine growth and of the pathologic changes induced in a growing spine by an early onset spinal deformity. The growing spine is a mosaic of growth plates characterized by changes in rhythm. During growth, complex phenomena follow each other in very rapid succession. These events are well synchronized to maintain harmonious limb and spine relationships as growth does not occur simultaneously in the same magnitude or rate in the various body

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Table 1 Growth is a change in proportion

Developmental stage	Body segment		
	Head	Trunk	Lower extremities
Fœtus (early pregnancy)	50	34	18
Fœtus (mid to late pregnancy)	35	40	25
Newborn	25	40	35
Infant	23	37	40
Child	20	35	45
Pre-adolescent	18	34	48
Adult (skeletal maturity)	13	40	47

The ratio between lower limb and sitting height varies with age. Values are expressed in percentages (%) [1, 3–6]

Table 2 Growth velocity in boys and girls

Age (years)	Boys		Girls	
	Sitting height	Lower limb	Sitting height	Lower limb
1	12.4	9.9	12.3	8.9
2	5.3	5.3	5.3	5.1
3	3.3	4	3.4	4
4	3.2	4.2	3.3	4.1
5	2.8	3.9	3	3.8
6	2.3	3.2	2.4	3.3
7	2.3	3.2	2.4	3.3
8	2.3	3.2	2.4	3.3
9	2.3	3.2	2.4	3.3
10	2.3	3.2	2.4	3.3
11○	2.3	3.2	2.4	3.3
12○	2.3	3.2	3.4	4.4
13○,●	2.3	3.2	4.3	3
14●	3.7	4.7	2.5	1.2
15●	4.8	3.3	1.1	0.3
16	2.8	1.2	0.2	0.1
17	1	0.6		
18	0.3	0.1		

Values are expressed in centimetres (cm) and are average values [1, 3–6]. Pubertal spurt is between age 13 and age 15 in boys (●) and between age 11 and age 13 in girls (○)

segments (Table 1). The slightest error or modification can lead to a malformation or deformity with negative effects on standing and sitting height, thoracic cage shape, volume and circumference, and lung development [1–6].

All types of growth are interrelated. Thus, as the spinal deformity progresses by a "domino effect", not only spinal growth is affected, but also the size and shape of the thoracic cage are modified. This distortion of the thorax will interfere with lung development. Over time, the scoliotic disorder changes in nature and from a mainly orthopaedic issue, it becomes a severe paediatric, systemic disorder with thoracic insufficiency syndrome [7, 8], *cor pulmonale* [9], and reduced body mass index (BMI). In most severe cases, these alterations can be lethal [1–6].

How to evaluate growth

Growth is a ratio between remaining and elapsed growth and any surgical strategy should be adjusted according to remaining growth. A thorough analysis of standing and sitting height, arm span, weight, thoracic perimeter, T1–S1 spinal segment length, and respiratory function help the surgeon to plan the best treatment at the right time [1, 2].

Standing height

The gain in standing height is approximately 25 cm during the first year of life and around 12.5 cm during the second year. Between ages 2 and 3 and 3 and 4 years, the gain in standing height is approximately 9 and 7 cm/year, respectively. At 5 years of age, standing height increases by 5 to 5.5 cm each year in both boys and girls. At the onset of puberty, remaining growth is about 18 cm for girls (11%) and 20 cm for boys (13%). Standing height is a global marker composed of two components—sitting height and subischial height. As these two regions often grow at different rates and at different times, standing height does not always exactly correlate with trunk height loss in children with severe spinal deformities [1–6].

Sitting height

Sitting height correlates strictly with trunk height and is about 34 cm at birth on average, and 88 cm and 92 cm at the end of growth for girls and boys, respectively [1–6] (Table 2). In children with severe spinal deformities, the loss of sitting height is related to the severity of the deformity. For this reason, it is important to monitor changes in sitting height rather than in standing height in children with scoliosis. During the first 3 years of life, or if a child has a neurologic disorder or a collapsing spine, it is recommended to measure sitting height in a supine position. Growth is a succession of acceleration and deceleration phases comprising three periods. The first period is from birth to age five and is characterized by a gain in sitting height of 27 cm with 12 cm occurring during the first year of life. The second period is from age 5 to 10 years and is a quiescent phase as sitting height increases by 2.5 cm/year. The third period is characterized by a gain in sitting height of 12–13 cm and corresponds to puberty [1, 3–6, 10].

Arm span

The measurement of arm span is an indirect measurement to evaluate standing height and can be used to assess predicted height in non-ambulatory children with cerebral palsy or myelomeningocele. Arm span and standing height have an almost perfect linear correlation. Standing height corresponds approximately to 97% of arm span with a small gender difference in that boys have an arm span that is a greater proportion of total standing height than girls. This relationship persists throughout puberty and into adulthood. In 77% of healthy children, arm span will be 0–5 cm greater than standing height; in 22% it will be 5–10 cm greater; and in 1% it will be greater by 10 cm or more. As a rule of thumb, arm span divided by two is very close to sitting height and, divided by four, to the T1–S1 spinal segment [1, 3–6, 10].

Weight

Growth energy requirements during the first 3 years of life are enormous and much greater than those of adults: 110 calories versus 40 calories/kg/day; 2 g versus 1 g of proteins/kg/day; and 150 ml versus 5 ml of water/kg/day, respectively. Moreover, skeletal mineralization alone requires the storage of 1 kg of calcium between birth and adulthood [1–6].

Weight is a useful parameter for evaluating growth and increases by 20-fold from birth to skeletal maturity. At 5 years of age, weight is approximately 20 kg, 30 kg by 10 years, and reaches 60 kg or more by 16 years. It should be kept in mind also that during pubertal spurt, weight usually doubles [1–6]. As most children with severe spinal deformities or neurological impairment have a low BMI and thus a higher risk of surgical morbidity, weight is a precious indicator [11, 12]. In selected cases, a hypercaloric nutritive protocol should be initiated prior to surgery. Children with pulmonary insufficiency characteristically have poor nutrition as the energy expenditure from the extra work of breathing approaches the nutritional gain of eating. Skaggs et al. showed that although approximately eight of ten patients with severe spinal deformities and thoracic insufficiency syndrome were less than 5 percentile in weight, a significant improvement in the nutritional status of these children was obtained after expansion thoracoplasty surgery [13].

Average birth weight is approximately 3 kg, which means that blood volume is about 0.3 l. Weight plays an essential role in surgical planning and the margin for manoeuvre is very narrow for the surgeon. Any postoperative weight loss, however slight, after spinal surgery before 5 years of age can have serious consequences. A 1-kg post-operative weight loss in a 18-kg patient represents about 6% of the total body weight and, therefore, there is a major difference between operating on a 40-kg and a 20-kg child [1–6, 10–12]. For this reason, children with spinal deformities weighing less than 20 kg should be differentiated from those over 20 kg [1, 2].

Of note, BMI can be misleading. A nine-year-old child with severe scoliosis, a standing height of 110 cm, and a weight of 12 kg can have a BMI somewhat reassuring. However, this information does not reflect the reality as the standing height is similar to a child of 1–5 years and a weight comparable with a two-year-old child. In children with low weight, pubertal spurt changes are moderate as weight must be at least 40 kg for the spurt to be normal. Of note, weight gain in children after spinal surgery is a good indicator that the clinical situation is under control.

T1–S1 spinal segment

Assessment of the T1–S1 spinal segment is important as many spinal deformities originate in this segment. At birth, the T1–S1 segment measures about 20 cm and reaches 45 cm at skeletal maturity (Table 3). It should be recalled that the height of the spine accounts for 60% of total sitting height, with the head and the pelvis accounting for the remaining 40% [1, 3–6, 10]. The T1–S1 segment accounts for approximately 50% of the sitting height, two-thirds for the thoracic spine, and one-third for the lumbar spine. It grows around 10 cm during the first 5 years of life (2 cm/year), about 5 cm between ages five and 10 (1 cm/year), and about 10 cm between age 10 and skeletal maturity (1.8 cm/year).

T1–T12 spinal segment

T1–T12 is the posterior pillar of the thoracic cage and a strategic segment. It measures about 12 cm at birth, 18 cm at 5 years of age, and about 27 cm on average at skeletal maturity. The thoracic spine makes up 30% of the sitting height, and a single thoracic vertebra and its disc represent about 2.5% of sitting height. In normal children, the longitudinal growth of the thoracic spine is approximately 1.3 cm/year between birth and 5 years, 0.7 cm/year

Table 3 Evaluation of T1–T12 and L1–S1 segments at birth and during childhood, pre-adolescence and adulthood (skeletal maturity)

Developmental stage	Boys		Girls	
	T1–T12	L1–S1	T1–T12	L1–S1
Newborn	11	7.5	11	7.5
Child	18	10.5	18	10.5
Pre-adolescent	22	12.5	22	12.5
Adult (skeletal maturity)	28	16	26.5	15.5

Values are expressed in centimetres (cm) and are average values [1, 3–6]

between the ages of 5 and 10, and 1.1 cm/year during puberty. A precocious arthrodesis of this segment has effects on thoracic growth and lung development [14, 15].

In young children with progressive deformity, there is a decrease of longitudinal growth and a loss of the normal proportionality of trunk growth [1–6, 10]. Untreated, progressive early-onset spinal deformity has been associated with short trunk, short stature and, often, respiratory insufficiency. In untreated patients, the loss of vital capacity in those with early onset scoliosis has been shown to be 15% greater than in those with adolescent idiopathic scoliosis. Moreover, Karol et al. have shown also that a thoracic spine height of 18–22 cm or more is necessary to avoid severe respiratory insufficiency. Fusion is a cause of respiratory insufficiency and adds the loss of pulmonary function to the spinal deformity [15, 16]. Emans et al. showed that pelvic inlet width, measured by computerized tomograms or plain radiographs, is an age-independent predictor of the expected thoracic dimensions in unaffected children and adolescents. This study also establishes normal range standards of chest and spine dimensions to help the assessment of treatment outcomes [17, 18].

L1–L5 spinal segment

L1–L5 length is approximately 7.5 cm at birth and 16 cm on average at skeletal maturity (Table 3). The lumbar spine makes up about 18% of the sitting height, and a single lumbar vertebra and its disc represent 3.5% of sitting height. At age 10 years, the lumbar spine has reached about 90% of its final height, but only 60% of its definitive volume. A perivertebral arthrodesis of the lumbar spine performed after the age of 10 years results in minimal loss of sitting height [1, 3–6].

Thoracic cage dimensions: volume, circumference and shape

The thoracic cage is the fourth dimension of the spine [1, 4–6]. At birth, thoracic cage volume represents about 6% of its definitive size, 30% by age five, and about 50% by age 10. Moreover, between age 10 and skeletal maturity, the thoracic cage volume doubles and its volumetric growth stops. All types of growth do not progress at the same speed. At 5 years of age, the trunk has reached about 66% of its final height, whereas thoracic volume is only 30% of its definitive size [1–6, 10].

Thoracic circumference corresponds to 95% of sitting height and increases more both during the first 5 years of life and puberty. On average, the newborn thoracic perimeter is 32.3 cm in boys and 31.5 cm in girls and it will attain a mean value of 89.2 and 85.4 cm, respectively [1–6, 10].

Thoracic cage shape varies with age. At birth, the difference between thoracic depth and width is minimal and the ratio thoracic depth/thoracic width is very close to 1. Conversely, at skeletal maturity, the thoracic depth/thoracic width ratio is lower than 1 as width has grown more than depth. For this reason, the overall thoracic cage shape evolves from ovoid at birth to elliptical at skeletal maturity. At the end of growth, the thorax has an average thoracic depth of 21 cm in boys and 17.7 cm in girls with an average thoracic width of 28 and 24.7 cm, respectively. At skeletal maturity, thoracic depth and width represent about 20 and 30% of sitting height, respectively [1–6, 10]. The thoracic cage is part of the rib-vertebral-sternal complex [19].

Lung and thoracic cage growth

The "golden" period for both thoracic spine and thoracic cage growth occurs between birth and 8 years of age and coincides with lung development. The source of potential respiratory failure is therefore double extrinsic disturbances of the chest wall functions as thoracic cage deformities prevent hyperplasia of lung tissue and intrinsic alveolar hypoplasia. It is important to preserve both thoracic growth and lung volume during this critical period of life. Post-mortem studies showed that patients with early-onset deformities have fewer alveoli than expected with the presence of emphysematous changes in existing alveoli. These studies suggest that mechanical compression is not a factor in reducing the number of alveoli and this is probably due to a premature cessation of alveolar proliferation [20, 21]. Indeed, from the late foetal stage to 4 years, the number of alveoli grows by a factor of 10, and the development of the bronchial tree ends around 8–9 years of age. In a review of 1,050 normal CT scans of the chest with three-dimensional volumetric reconstruction of the pulmonary system, Gollogly et al. showed that lung parenchyma volume is a function of age. At birth, lung parenchyma volume is 400 cc, approximately 900 and 1,500 cc at ages 5 and 10 years, respectively, and around 4,500 cc for boys and 3,500 cc for girls at skeletal maturity [22]. An early-onset scoliosis therefore adversely affects thoracic growth in the critical period of maximum respiratory growth, which induces irreversible changes in the thoracopulmonary structure [1–6, 10, 20–27].

Campbell et al. have described the thoracic insufficiency syndrome, i.e., the inability of the thorax to ensure normal breathing due to severe alterations in the gas exchange between atmospheric air and blood, which involves reduction of O_2 and retention of CO_2 in blood. In the most serious cases, the clinical picture can be lethal [7–9, 23, 24].

Dubousset et al. have shown that that severe spinal deformities lead to penetration of the apical portion of the deformity inside the thoracic cage (endothoracic hump)

and have described the "spinal penetration index" [24]. It is now known that spine deformations adversely affect thorax development by changing its shape and reducing its normal motility. The rib-vertebral-sternal complex, which fits the thoracic cavity three-dimensionally, tends to constitute an elastic structural model similar to a cube in shape. However, in the presence of scoliosis, it becomes flat and rigid and turns elliptical, thus preventing the lungs from expanding [19]. These deformations, which can be lethal in the most severe cases, result from mutual interactions and influences among the various skeletal and organic components of the thoracic cage and cavity that are still not well understood [14, 18, 19, 25–27].

The development of the thoracic cage and lungs is a complex process that requires perfect synergy among the various components of the rib-vertebral-sternal complex. Alterations in any of these elements affect and change the development and growth of the others [1, 5, 14, 19, 25–27]. To preserve thoracic motility and permit a normal development of the respiratory tree, the treatment should not focus on the spine only, but should consider the rib-vertebral-sternal complex as a whole [19].

Microscopic and macroscopic spinal growth

Symmetric and harmonious growth characterizes normal spines although spinal growth itself is the product of more than 130 growth plates working at different paces [1, 3–6]. In severe scoliosis, growth becomes asymmetrical as a result of growth plate disorganization. Complex spinal deformities alter growth spine cartilage and vertebral bodies become progressively distorted and can perpetuate the disorder. Therefore, many scoliotic deformities can become growth plate disorders over time [1–6, 10].

The neurocentral synchondrosis is a physis in the spine located at the junction of the pedicle and the vertebral body and is important in the growth of the vertebral body and the posterior arch. It has been shown in a growing pig model that unilateral transpedicular screw fixation that traverses the neurocentral synchondrosis can produce asymmetric growth of the synchondrosis and create scoliosis with the convexity on the side of the screw fixation [28]. However, in humans, neurocentral synchondrosis fuses around age nine, and by 5 years of age the spinal canal has already grown to about 95% of its definitive size. Therefore, a perivertebral arthrodesis performed after age five should have no influence on the size of the spinal canal [1–6].

Can surgical treatment modulate spinal growth? The growth modulation concept

For the past several decades, the standard treatment of early onset deformities unresponsive to non-surgical treatment has been spinal fusion with the goal of arresting progression. Unfortunately, arthrodesis carried out in the thoracic spine at an early age does not address the impact of the deformity on lung parenchyma development or preservation of pulmonary function. Even its effect on completely preventing deformity progression has been questioned. In children with spinal deformities with strong progression potential, expansible materials can be used to support the expansion of the thoracic cage and lung growth [1, 7, 8, 19, 28–34]. However, modern techniques and instrumentations only control one plane of the deformity as distraction forces are applied to the spine or thoracic cage. At present, there is no instrumentation able to fully control the tri-dimensional nature of early onset spinal deformities.

Several studies have focused on anatomical influences of experimental arthrodesis on growth of the spine, chest development, and thoracopulmonary function. These reports have demonstrated that early arthrodesis, as well as severe spinal deformities, can adversely affect the development of the spine and the thorax by changing their shape and reducing normal mobility [1, 14, 22–24]. Canavese et al. evaluated the consequences of disturbed growth of vertebral bodies on the development of the ribs, sternum, and lungs, which form part of the rib-vertebral-sternal complex [14, 19, 25]. These influences are much more evident when the arthrodesis is carried out in the critical portion of the thoracic spine, i.e., the T1–T6 segment [1, 2, 14, 15, 19, 25, 26]. An experimental study by Mehta et al. demonstrated that a unilateral deformity of the spine or thorax induces scoliosis and thoracic cage deformity with asymmetrical lung volume, thus showing that there is an interaction between growth of the spine and thorax [14].

Respiratory problems can develop after a precocious vertebral arthrodesis or as a consequence of pre-existing severe vertebral deformities and can vary in patterns and timing, according to the existing degree of deformity. The varying extent of an experimental arthrodesis also affects differently both growth and thoracopulmonary function [1, 2, 14]. It must be borne in mind that early spinal fusion, especially if performed in the thoracic region, is a cause of respiratory insufficiency and adds the loss of pulmonary function to the spinal deformity. Karol et al. reported also that a thoracic spine height of 18 cm or more is necessary to avoid severe respiratory insufficiency. In addition, they showed that children undergoing a precocious spinal fusion have a reduction of thoracic depth and a shorter T1–T12 segment compared with normal subjects. The forced vital capacity may decrease 50% of predicted volume if more than 60% of the thoracic spine is fused, i.e., eight thoracic vertebrae, before the age of 8 years [29]. Karol et al. confirmed with their clinical work some of the experimental findings previously published [15, 29].

Campbell et al. showed that opening wedge thoracostomy can increase the thoracic volume ("parasol effect") [7, 8]. Of note, it is important to perform such a procedure before the end of the development of the bronchial tree, which occurs usually around 8 years of age [20, 21]. However, these procedures present a number of inconveniences by increasing the stiffness of the thoracic cage and the amount of energy needed to breathe. The effect of the surgical expansion of the thorax on pulmonary development is still controversial. In particular, Redding has pointed out that surgical expansion of the thorax can increase thoracic volume, but it does not automatically increase pulmonary capacity [3]. In particular, in their experimental work, Mehta et al. [26] have suggested that chest wall movement restriction, as well as reduced hemithorax size following unilateral rib tethering, may have a global effect on alveolar and capillary development.

The crankshaft phenomenon is the progression of the spinal deformity when the anterior portion of the spine continues to grow, whereas the posterior portion is blocked by arthrodesis [27, 35]. Goldberg et al. showed that early surgery in patients that developed scoliosis before 4 years of age does not modify the deformation produced by scoliosis or preserve respiratory function, even when the anterior growth of the spine is arrested [29]. Therefore, it is very important for the surgeon to consider the state of skeletal maturity and the amount of growth remaining in the segment of the spine that is to be fused.

During the past few years, several studies have demonstrated that growth close to normal can be attained with vertical expandable prosthetic titatnium rib (VEPTR) [7, 8, 34], growing rods [37, 38], or a Shilla-type procedure [36]. All these techniques aim to restore normal spinal growth by controlling the progression of the deformity [2, 3].

In the Shilla procedure, the most curved portion in the centre of the spine is held straight and fused. The ends of the rods are not fused, but held in place with growing screws, thus allowing the spine to lengthen without additional surgical interventions. The screws capture the rods, but slide as the patient grows. This procedure seems to reduce the number of surgical interventions compared with traditional techniques [36]. However, the Shilla procedure is very new and experimental and it still remains to be determined whether it has a long-term future in the treatment of severe scoliosis. Surgery should be limited as much as possible and extensive arthrodesis of the spine should be avoided.

In summary, five points must be remembered when surgery is planned: (1) distraction is effective, but it is not enough to control the tri-dimensional nature of a spinal deformity and—although not a rule—may create junctional kyphosis [27, 29, 38] (2) early arthrodesis, as well as severe spinal deformities, can adversely affect the development of the spine and the thorax by changing their shape and reducing their normal mobility [1–3, 14, 22–24] (3) opening wedge thoracostomy can increase thoracic volume ("parasol effect") and fight against chest restriction, but it does not automatically increase pulmonary capacity. The optimal time for opening wedge thoracostomy is more likely to be in early, rather than late, childhood [2, 7, 8]; (4) the crankshaft phenomenon is a constant risk. Apical vertebrae, for example, should be controlled as with the Shilla procedure [27, 29, 35]; (5) repetitive surgery should be avoided as it can contribute to creating spontaneous spine fusion and reducing thoracic cage motility [1, 2, 14–19, 23–26].

Conclusions

Only a critical analysis of all growth parameters over time allows to unmask and understand the magnitude of the deficits induced by an early onset spinal deformity. Spinal and thoracic growth obey strict rules and can be controlled only by following their requirements. Four different scenarios can be identified. (1) The clinical picture worsens. Abnormal growth leads to a deficit that sustains the deformity as a rolling snowball that gets bigger ("snowball effect"). Reduced BMI due to weight loss weakens, among others, the respiratory muscle and thus makes breathing more difficult. (2) The clinical picture is stable. (3) The clinical picture gets slightly better with improvement of different clinical parameters, such as weight, vital capacity, and sitting height. (4) The clinical picture returns to normal. In this ideal scenario, all clinical parameters catch up the deficit induced by the deformity. Unfortunately, this is unlikely to happen as most children with severe spinal deformities will end up at skeletal maturity with a short trunk, a significant loss of vital capacity, and disproportionate body habitus [2, 3].

Surgical strategies must consider the complete life span of the patient and should provide answers to two basic questions: (1) what is the functional benefit? and (2) what is the morbidity risk? It must be retained that the thoracic cage is part of the deformity (rib-vertebral-sternal complex) [19]. There is a normal interaction between the organic components of the spine, the thoracic cage, and the lungs. Both early onset spinal deformities and precocious spinal arthrodesis alter spinal growth and affect thorax development by changing its shape and reducing its normal mobility. Treatment of the growing spine is a unique challenge and involves preservation of the thoracic spine, thoracic cage, and lung growth without reducing spinal motion. The principle that a short straight spine, produced by an early fusion, is better that a long curved spine is no longer generally accepted [2].

Conflict of interest None of the authors has any potential conflict of interest.

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