## KEY POINTS

- The primary and most important goal of treatment should be to promote rib cage expansion and lung development.
- Repeated anesthesia in young children may cause permanent deleterious effect, especially in children younger than 3 years.
- Traditional growing rods are still the most predictable surgical technique for the management of EOS.
- Promising results may be expected for ongoing studies testing the efficacy and safety of bracing for the treatment of infantile scoliosis.

before 6 years. When left untreated or inappropriately treated, EOS can lead to TIS [3▪].

The source of the respiratory failure is a consequence of two different pathologic pathways, intrinsic alveolar hypoplasia and extrinsic disturbance of chest wall function.

### Intrinsic alveolar hypoplasia

Alveolar hyperplasia is the primary contributor to lung growth until the age of 8 years, at which point hypertrophy becomes dominant, augmenting volume until maturity. The expansion of lung volume until age 5 years is propelled by a rapid increase in peripheral airway conductance concurrent with airway enlargement. Subsequently, hypertrophy coincides with the enlargement of thoracic circumference from age 10 years until maturity, culminating in a twofold rise in thoracic volume.

When a child has not grown to an average height by the age of 5 years, there is a high likelihood of developing TIS. That is why the 5 first years of life have been considered 'The Golden Period'. The thoracic spine length increases by 50% (from 12 to 18 cm) from birth to 5 years of age, up to 60% of adult spine length during the initial 5 years. In conjunction with direct spinal elongation, the thoracic circumference doubles in magnitude after age 10 years. Therefore, it is essential to facilitate alveolar hypertrophy and hyperplasia with a normal thoracic circumferential and length growth during these critical periods of thoracic expansion.

### Extrinsic disturbance of chest wall function

External disruption of respiratory function arises from deformities in the ribs and/or chest wall, leading to reduced compliance or impaired function. Rib fusions lead to reduced compliance, where natural chest wall movements are constrained. In cases of rib absence that result in a localized flail or paradoxical chest wall segment, function is even more impaired. In patients suffering from neuromuscular conditions such as static encephalopathy, muscular dystrophy, muscle atrophy, spina bifida, or spinal cord injury, muscular impairment may exacerbate chest wall dysfunction [3▪].

Vital capacity is diminished in patients with congenital scoliosis as compared to those with idiopathic scoliosis having equivalent curve magnitudes. This reduction is likely attributable to coexisting rib anomalies that exacerbate chest wall dysfunction. In non congenital deformities, scoliosis-induced rib deformities contribute to inefficient respiration. The intercostal spaces on the concave hemithorax become constricted, impede expansion, and cause a restrictive state. Simultaneously, the convex hemithorax displays widened intercostal spaces incapable of generating normal expiratory force. Interestingly, a recent study in pig model was able to replicate the common clinical features of EOS such as rib fusion, asymmetric thoracic cage, increased cobb angle,

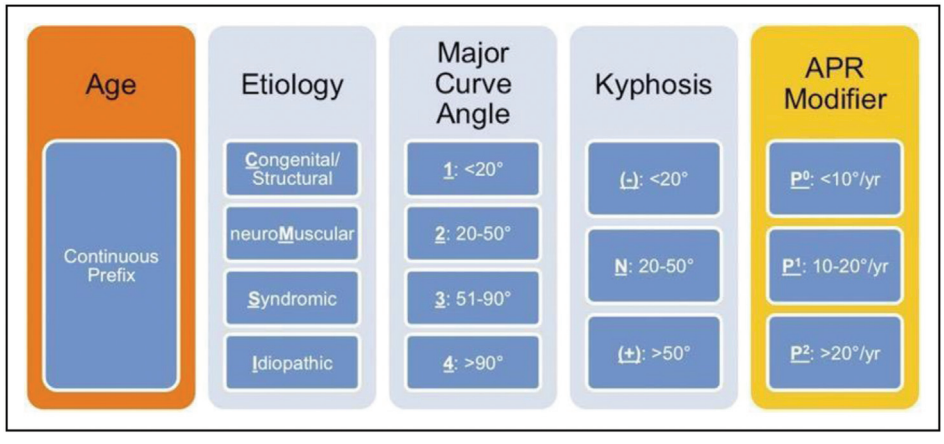


**FIGURE 1.** The classification of early-onset scoliosis (C-EOS). APR, annual progression ratio.