Central Sleep Apnea in Infants

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INTRODUCTION
Sleep-disordered breathing encompasses a wide variety of breathing disorders including obstructive sleep apnea, central apnea (CA), and nonobstructive sleep related hypoventilation. Central sleep apnea results from absent respiratory drive from breathing centers in the brainstem during sleep. The criteria that meets the definition of CA differ between children and adults. The American Academy of Sleep Medicine (AASM) defines CA in children as cessation of breathing during sleep without any breathing effort for a duration of 20 seconds or longer, or lasting at least 2 breaths’ duration with 3% oxygen desaturation or arousal.1 In infants, the CA is at least 2 breaths in duration and is associated with a decrease in heart rate to less than 50 beats per minute for at least 5 seconds, or less than 60 beats per minute for 15 seconds. Periodic breathing is a form of CA that has been described as greater than 3 episodes of CA lasting 3 seconds separated by no more than 20 seconds of normal breathing.2 Apnea following a sigh is not considered pathologic unless it is associated with arousal or desaturation. Isolated central sleep apnea (Fig. 1A), CA following sigh breathing (see Fig. 1B), CA following body movements, and periodic breathing patterns (see Fig. 1C) can be seen in healthy infants and children.2 It is common to see CA in healthy infants, but on rare occasions it can be a harbinger of ominous pathologic consequences, such as congenital central hypoventilation syndrome or Arnold-Chiari malformation.3 The severity of CA can be characterized using the apnea-hypopnea index (AHI), the total number of events overnight divided by hours of sleep. There is no clear description in the literature of pathologic central AHI, but studies have considered a central AHI from greater than 0.9 to AHI greater than 5 as abnormal.4–6 The adverse consequences of moderate and severe CA are well known, but those of CA of milder degree is still debated.7 The mild CA seen in otherwise healthy infants tends to improve with age, and older children can have rare CAs.5,8 The improvement in apnea frequency can be considered as maturation of respiratory control and chest-wall mechanics.
APNEA IN HEALTHY NORMAL INFANTS

Brief CA in full-term infants is very common, especially in the early months of life. The duration and frequency of CA improves with age. Several studies have focused on defining the prevalence of CA in healthy infants. Each study has used different criteria to define CA, different monitoring techniques, and different testing environments such as home versus in-laboratory polysomnography, which makes it difficult to make comparisons between the studies. As already noted, standardization of the definition of CA has been achieved, which will make interstudy comparisons in the future much easier and more fruitful. Home monitoring provides an opportunity to collect data in infants during sleep for a long

Fig. 1. (A–C) Sixty-second-long epoch of the polysomnography of a 21-month-old child born at full term, referred for evaluation of sleep apnea because of a family history of sudden infant death syndrome. (A) Central sleep apnea without arousal. (B) Central sleep apnea after arousal during stage 3 non–rapid eye movement sleep. (C) Periodic breathing during rapid eye movement sleep. abdm, abdominal plethysmography; C3, C4, central electroencephalogram leads; CHIN, chin electromyogram; E1, left eye electromyogram; E2, right eye electromyogram; EKG, electrocardiogram; ETCO, end-tidal carbon dioxide tracing; ETGR, End tidal graphical representation; F3, F4, frontal electroencephalogram leads; FLOW, tracing of oral thermistor; PLTH, Plethysmography; PTAF, for measurement of nasal air flow; SNOR, snore micrograph; SpO2, continuous pulse oximetry; thor, thoracic plethysmography.