

SLEEP MEDICINE PEARLS

A 14-Month-Old Child With Gastroesophageal Reflux and Central Apnea

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CASE

The patient was a 14-month-old male with a history of 22q11.2 deletion with a large ventricular septal defect and right aortic arch status post repair at age 4 months. He had dysphagia and gastroesophageal reflux for which he took 2 mL ranitidine (15 mg/mL) twice a day. The patient was referred to the sleep clinic by his gastroenterologist for concerns of snoring and noisy breathing. The patient was born at full term and is dependent on a nasogastric feeding tube but has had no difficulty with growth or weight gain. At the time of the study he was bolus-fed formula via nasogastric tube and only took purees by mouth due to dysphagia with high risk for aspiration (determined by a swallow study in November 2017). He did not have any overnight feedings. The patient's parents positioned him to sleep in a bassinet at a 30° incline per recommendations from their gastroenterologist to help prevent reflux. The patient typically slept from 10:00 PM to 10:30 AM and woke up at least

twice during the night. He was outgrowing the bassinet and the parents wanted to know whether the patient could sleep on a bed with a flat surface. The study was therefore ordered as split with a few hours with the head of the bed elevated at 30° and the remainder of the study with the child sleeping supine on a flat bed.

POLYSOMNOGRAPHY RESULTS

Standard dose of ranitidine was taken prior to the study. Complete split data are shown in **Table 1**.

QUESTION: What should be suggested to the patient's parents?

Table 1—Sleep study results.

POS Level	TST (minutes)	REM (minutes)	Avg O ₂ (%)	Obstructive Apneas	Central Apneas	Hypopneas	AHI (events/h)	Avg TcCO ₂	Min O ₂ Sat (%)
30°	139.0	21.5	97.3	–	3	2	2.2	47.8	85.5
Flat	282.0	66.0	97.3	–	36	1	7.9	43.8	91.0

AHI = apnea-hypopnea index, POS = position, REM = rapid eye movement, Sat = saturation, TST = total sleep time.

ANSWER: The patient should sleep on a wedge or at an incline.

DISCUSSION

There is mixed evidence regarding the clinical significance and causal relationship between gastroesophageal reflux (GER) and apnea.¹ A causal relationship between GER and central apnea in infants is postulated to be associated with an exaggerated laryngeal chemoreflex. The laryngeal chemoreflex was initially described in animals such as newborn lambs who experienced bradycardia, hypertension, and apnea in response to infusions of water into the larynx.² The chemoreflex is thought to be generated via stimulation of the sensory afferent nerve fibers of the superior laryngeal nerve whose nerve endings are present in the mucosal epithelium of the epiglottis, interarytenoid space, and aryepiglottic folds.³ Stimulation of the reflex induces adduction of the vocal cords via the efferent recurrent laryngeal nerve.⁴ Apneas have been reproduced in infant monkeys via electrical stimulation of the superior laryngeal nerve.⁵ Similar responses of apnea, obstructed respiratory effort, and cough have also been noted in human infants when warm saline was delivered to the oropharynx via nasopharyngeal catheter.⁶ It is hypothesized that this laryngeal chemoreflex is designed to prevent aspiration through glottic closure. However, in some children the reflex may be overexaggerated and lead to prolonged apnea in response to a stimulus such as gastric regurgitation. The reflex appears to be more common in newborns, particularly premature infants, and diminishes with age.³

The case of our 14-month-old child suggests that GER can indeed contribute to apnea. It is common practice to have infants and children who have GER sleep on a wedge or incline to decrease the incidence of reflux episodes and aspiration (as was suggested to the parents of our patient). Given the significant increase in apneic episodes after laying the child flat, one can hypothesize that it was an increase in the amount of reflux he experienced that lead to the apneas. It is plausible that the refluxes were triggering the laryngeal chemoreflex, which lead to the apneas. Other possible causes for the apneas after the child was laid down include aspiration and anatomical obstruction such as macroglossia or tracheomalacia. However, these etiologies are less likely given the lack of coughing, transient nature of the episodes that resolved without head movement and repositioning, normal development and growth, and lack of signs such as stridor or wheezing that might suggest another type of airway obstruction.

The child continues sleeping on a 30° angle elevation using a wedge and doing well. This case illustrates the benefit of head-of-the-bed elevation in children with GER and also could be

indicative of assessment of reflux in infants and children who incidentally show elevated central apnea in the sleep study.

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1. Children with reflux can present with elevated central apneas on sleep study.
2. A 30° angle elevation of the head of the bed may decrease the central apnea index in children with GER.
3. Sleep specialists could consider performing split sleep studies in their patients with comorbid GER to determine safety of discontinuing sleeping with the head of the bed elevated.

CITATION

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