A 10-year-old girl with history of severe to profound bilateral sensorineural hearing loss and anxiety presented with daily tiredness, difficulty waking in the mornings, and requiring a parent in bed with her in order to fall asleep. History was obtained from her and her mother, who also has sensorineural hearing loss, via American Sign Language (ASL) interpreter.

The patient’s bedtime was 9 PM with sleep onset within 30 minutes. She endorsed anxiety about falling asleep and mother stayed in bed with her overnight. Occasional night wakings lasted 10-30 minutes. On school days, mother started waking the patient 45 minutes before she had to be out of bed. She often seemed grumpy in the morning but was not chronically tardy.

Mother was not aware of whether the patient snored during sleep. The patient sometimes mouth breathed. She had not had labored breathing, bruxism, night sweats, nocturnal enuresis, or morning headaches, and did not sleep with neck in hyperextension. She had had at least one episode thought to be sleepwalking.

Physical examination was notable for a thin child with prominent though non-erythematous tonsils and a receding chin.

QUESTION: What else should the clinician do in order to decide whether or not a polysomnogram is necessary?
DISCUSSION

 Relatives of this patient with typical hearing reported her snoring within the past year. Subsequent overnight polysomnogram showed pediatric obstructive sleep apnea with an obstructive apnea-hypopnea index of 7.4 events per hour and sleep fragmentation (Figure 1). Oxygen nadir was 92%. Obstructive sleep apnea syndrome was diagnosed and the patient referred to otolaryngology.

 Hearing loss or deafness affects more than 48.1 million individuals in the United States, with nearly 1 in 5 displaying unilateral or bilateral hearing loss as defined by World Health Organization criteria, and 14.9% of US children affected by hearing loss or deafness. The etiology is heterogeneous, with a variety of congenital and acquired conditions that can also lead to associated neurocognitive insult or increase potential risk for sleep problems. Genetic deafness (such as connexin 26 mutation, also referred to as gap junction beta-2 protein [GJB2]) represents one of the most common forms of congenital deafness and can lead to families with deaf parents and deaf children, as in the case above.

 It is unknown at present if patients with hearing loss have a baseline increased risk for sleep disorders. However, sleep disorders are highly prevalent in the general population, and there is no reason to believe that they are less common among people who are deaf. For sleep medicine specialists, the case above highlights the risk of missing sleep disordered breathing in pediatric patients who are deaf or hard of hearing and raised by parents who are also deaf or hard of hearing. Among the elderly, where rates of age related hearing loss increase with each decade of life, an elderly bed partner with hearing loss may not endorse snoring. Integrating questions into clinical screenings that rely on visual signs or symptoms may improve detection of sleep disordered breathing in these patients, but ultimately snoring is an important sign of sleep disordered breathing. Involving other informants with typical hearing will increase detection of snoring in this population.

 In families where the primary form of communication is ASL, there is often increased difficulty accessing health care, especially more specialized care, due to the shortage of providers trained and experienced in working with individuals who are deaf or hard of hearing. There is also a documented increased risk of difficulties communicating with health and mental health care providers due to the need for accommodations such as interpreters. Previous research has demonstrated deficits in health care knowledge in individuals who are deaf or hard of hearing compared with the general population. Providing health care information in written format does not always remedy this problem: the average reading level of students who are deaf or hard of hearing leaving high school is below a basic proficiency level in reading comprehension.4

Figure 1—120 second epoch tracing of polysomnography recorded during N1 sleep reveals multiple obstructive apneic and hypopnic events accompanied by arousals and mild desaturations

ANSWER: Question family members who can hear about snoring.

EEG, electroencephalogram; F3-M2, C3-M2, and O1-M2, electroencephalogram leads; EOG, electrooculogram; E1-M2 and E2-M2, electrooculogram leads; ChinL, chin electromyelogram, left; SpO2, pulse oximetry; Thermistor, oronasal thermal flow; Flow_CU, nasal pressure; Thorax and Abdomen, ribcage and abdominal movements.
**CLINICAL PEARLS**

1. Increased suspicion of sleep disordered breathing in pediatric patients who are deaf or hard of hearing is required when caregivers are also deaf or hard of hearing and may not hear snoring.
2. Seek out collateral informants with typical hearing in elderly patients who present with other symptoms of sleep disordered breathing but have a bed partner with hearing loss.
3. Involvement and interview of hearing family members of the deaf child may be necessary in order to confirm auditory symptoms of sleep disordered breathing. Alternately, visual cues for sleep disordered breathing may be used when working with parents who are deaf or hard of hearing.

**REFERENCES**


**CITATION**


**DISCLOSURE STATEMENT**

This was not an industry supported study. The authors have indicated no financial conflicts of interest.