

A Framework for Information Processing in the Diagnosis of Sleep Apnea

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INTRODUCTION

Obstructive sleep apnea (OSA) is one of the most common sleep disorders. It is characterized by repetitive obstruction of the upper airways during sleep. The frequency of such events can range up to hundreds of events per sleep-hour. Full closure of the airways is termed *apnea*, and a partial closure is known as *hypopnea*. The number of apnea/hypopnea events per hour is known as the AHI-index, and is used by clinical community as a measure of the severity of OSA.

OSA, when untreated, presents as a major public health concern throughout the world. OSA patients use *health facilities at twice the average rate* (Delaive, Roos, Manfreda, & Kryger, 1998), causing huge pressures on national healthcare systems. OSA is associated with serious complications such as cardiovascular disease, stroke, (Barber & Quan, 2002; Kryger, 2000,) and sexual impotence. It also causes cognitive deficiencies, low IQ in children, fatigue, and accidents. Australian Sleep Association reported (ASA, 1999) that in the state of New South Wales alone 11,000–43,000 traffic accidents per year were attributable to untreated-OSA.

OSA is a highly prevalent disease in the society. An estimated 9% of the women and 24% of the men in the U.S. population of 30 to 60 years was found to be having at least mild OSA (Young, Evans, Finn, & Palta, 1997). In Singapore, about 15% of the *total population* has been estimated to be at risk (Puvanendran & Goh, 1999). In a recent study in India (Udwadia, Doshi, Lonkar, & Singh, 2004), 19.5% of people coming for routine health checks were found to have at least mild OSA.

The full clinical significance of OSA has only recently been understood. Partly as a result of this, the public awareness of the disease is severely lacking. Healthcare systems around the world are largely unprepared to cater to the massive number of OSA patients. This problem is especially severe in the developing world, where OSA diagnostic facilities are rare to find.

BACKGROUND

Definition of Sleep Apnea and Hypopnea

Sleep apnea refers to a cessation of breathing at night, usually temporary in nature. The American Academy of Sleep Medicine Task Force formally defines *apnea* as:

- a. Cessation of airflow for a duration ≥ 10 s, or
- b. Cessation of airflow for a duration < 10 s (for at least one respiratory cycle) with an accompanying drop in blood oxygen saturation by at least 3%.

Hypopnea is defined as a clear decrease ($\geq 50\%$) in amplitude from base line of a valid measure of breathing (eg., airflow, air pressure) during sleep for a duration ≥ 10 s, *plus* either:

- a. An oxygen desaturation of $\geq 3\%$, or
- b. An EEG-arousal (EEGA) (Flemons & Buysse, 1999).

The average number of obstructive sleep apnea and hypopnea events per hour of sleep, as computed over the total sleep period, is defined as the *Apnea Hypopnea Index (AHI)*.

The Current Standards in Apnea/Hypopnea Diagnosis

The current standard of diagnosis of OSA is Polysomnography (PSG). Routine PSG requires that the patients sleep for a night in a hospital *Sleep Laboratory*, under video observation. In a typical PSG session, signals/parameters such as ECG, EEG, EMG, EOG, nasal/oral airflow, respiratory effort, body positions, body movements, and the blood oxygen saturation are carefully monitored. Altogether, a PSG test involves over 15 channels of measurements *requiring physical contact* with the patient.

Drawbacks of PSG and Possible Improvements

At present, the hospital-based PSG test is the definitive method of diagnosis of the disease. However, it has the following drawbacks, particularly when employment as a community-screening tool is considered:

1. *Poor data integrity is a common problem* in routine PSG tests. Even when the test is done in the hospital, it is common to see cases of data loss (or quality deterioration), due to various reasons (eg., improper sensor contact due to electrodes/sensors coming loose, SpO2 sensor falling off, and measurement problems, such as movement artifacts).
2. *PSG interpretation is a tedious task*, due to the size of the data gathered, complexity of the signals, and measurement problems such as data loss.
3. *PSG requires contact instrumentation*; channels such as EEG/ECG/EOG/EMG require Galvanic contact with the patient. It is especially unsuited for pediatric use.
4. *PSG is not suitable for mass screening* of the population. A trained medical technician is required to connect the patient to the PSG equipment, and the patient needs to be monitored overnight to avoid incurring data losses.
5. *PSG is expensive*; this is another factor working against mass screening uses.
6. *AHI index* (and a variant of it known as the respiratory disturbance index, RDI) is used as

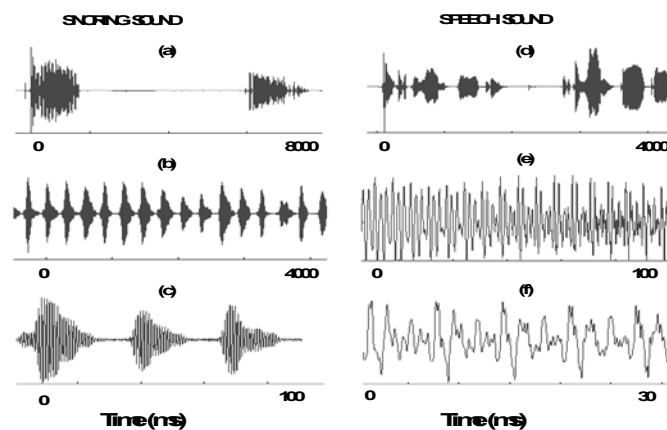
the golden clinical measure on the severity of apnea. However, AHI (or RDI) does not always correlate strongly with the symptoms of apnea as experienced by patients.

There is an enormous clinical need for a simplified diagnostic instrument capable of convenient and reliable diagnosis/screening of OSA at a home setting (Flemons, 2003). Similarly, hospital-based, full PSG testing requires better measures to characterize the disease. This article explores possible solutions to both of these problems.

There has been a flurry of recent activities at developing technology to address the issue of home screening of OSA. Four different classes of OSA monitors are under development (Flemons, 2003, and references therein). These devices varied from two-channel (eg., airflow and oximetry) systems (designated Type-IV), to miniaturized full-PSG (Type-I) units. Their major drawbacks are:

- Existing take-home devices have *at least* one sensor which requires physical contact. This makes them difficult to use by untrained persons, and cumbersome to use on pediatric populations. TYPE-IV systems, with the smallest number of sensors, suffers from the fact that oxymetry identifies oxygen saturation in blood only as a surrogate for OSA, and the absence of significant desaturation does not mean the absence of the disease (Flemons, 2003).

Figure 1. Similarities and differences between speech and snoring: (a),(b),(c) snoring at different time scales; (d),(e),(f) speech at different time scales



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