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CLINICAL REVIEW

Assessment of respiratory effort during sleep: Esophageal pressure versus noninvasive monitoring techniques

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SUMMARY

Monitoring of respiratory effort is paramount in the clinical diagnostic recording of sleep. Increased respiratory effort is a sign of obstructive sleep-disordered breathing and is associated with arousals from sleep. Respiration is the result of muscle activity that induces negative intrathoracic pressure and expansion of the thoracic and abdominal cavities. Therefore respiratory effort may be recorded from mechanical, electrical and electromechanical signals. Several techniques are available for the recording of respiratory effort. Monitoring of esophageal pressure is still the method of choice, as the pressure signal directly reflects the respiratory muscle force. However, esophageal pressure monitoring is cumbersome and may be replaced with noninvasive techniques. In order to be reliable, these techniques must be validated against the esophageal pressure standard. The present review presents a concise description of the technical principles and, if available, a comparison with esophageal pressure data, based on a systematic literature search. Most data are available on respiratory inductance plethysmography, and confirm that this technique is suitable for routine diagnostic investigation of respiratory effort during sleep. Pulse transit time, diaphragmatic electromyography, snoring loudness, suprasternal pressure monitoring, midsagittal jaw movement and forehead venous pressure monitoring are promising alternative techniques although only limited validation is available.

Introduction

Respiratory disorders of sleep are quite common. In contemporary medical practice, advanced diagnostic tools are employed to characterize the nature of sleep-disordered breathing (SDB). Whilst the monitoring of respiratory effort is paramount, different and sometimes disparate monitoring techniques are being used for this purpose. The present review aims to concisely describe the various methods for physiological measurement of respiratory effort in the pathophysiological framework of obstructive SDB. Arousal from sleep is a key feature of obstructive SDB, and therefore, the relation between respiratory effort and arousal is discussed first. A major part of this review deals with noninvasive monitoring techniques and their significance with respect to (invasive) esophageal manometry, which is recognized as the gold standard for the assessment of respiratory effort. Based on a systematic literature search, it was assessed whether existing noninvasive methods have been validated against measurement of esophageal pressure.

Note that the characterization of respiratory effort in central sleep apnea is outside the scope of this paper.

Obstructive respiratory events and arousals in sleep

Obstructive sleep apnea, hypopnea and respiratory effort related arousals

To denote a condition with recurrent events of upper airway (UA) narrowing and impaired airflow during sleep, the term obstructive sleep apnea (OSA) is commonly used [1]. In this context, 'apnea' itself means complete closure of the UA with cessation of airflow, whereas 'hypopnea' refers to a partial obstruction of the UA with reduced airflow. According to current scoring criteria, these events last at least 10 s in adults, whereas in children they should

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Abbreviations

AASM	American Academy of Sleep Medicine
EMG	electromyography
FVP	forehead venous pressure
OSA	obstructive sleep apnea
Pes	esophageal pressure
PPG	photoplethysmography
PTT	pulse transit time
RERA	respiratory effort related arousal
RIP	respiratory inductance plethysmography
SDB	sleep-disordered breathing
UA	upper airway
UARS	upper airway resistance syndrome

encompass two or more disrupted breaths [2]. Ongoing and usually increasing inspiratory effort is the hallmark of obstructive SDB [3]. Typically, termination of these events is associated with a transient arousal from sleep, during which UA dilating muscles are activated, UA patency is restored, and airflow resumes [4]. In 1999, Guilleminault and co-workers for the first time associated arousal from sleep with peak esophageal pressure in patients with excessive daytime sleepiness but no apparent sleep apnea, and coined this disorder the upper airway syndrome (UARS) [5]. In more recent years, further attention has been drawn on subtle obstructive breathing events that are associated with increased respiratory effort, but no obvious or only slight reduction in airflow. As these events are also associated with an arousal, they are called 'respiratory effort-related arousals' (RERAs) [6].

UARS is a form of SDB characterized by the predominant presence of RERAs. As a clinical condition, however, it is currently subsumed under the diagnosis of OSA [1].

The relation between respiratory effort and arousals

Increasing respiratory effort preceding arousal from sleep is a key factor in the pathophysiology of OSA. Vincken et al. observed that during obstructive apneas the force of the diaphragm increases progressively with each occluded inspiratory effort, to reach a point at which arousal from sleep occurs [7]. This level of inspiratory force was found to approximate the threshold of muscle fatigue. A close temporal relationship was observed between this critical point, onset of arousal and opening of the UA. Gleeson et al. examined the mechanisms underlying the arousal response to respiratory strain in sleep [8]. In eight healthy men, arousal-inducing effects of different respiratory challenges were studied, including added inspiratory resistive load, hypoxia, and hypercapnia. In agreement with the observations of Vincken et al., it was found that the appearance of arousal events was primarily linked to increasing respiratory effort. The peak-negative esophageal pressure at inspiration prior to arousal, whilst different between individuals, was consistently reproducible within each individual under the various stimulus conditions. These results corroborated the hypothesis that increasing ventilatory effort is the final common pathway to arousal from sleep, irrespective of the mechanisms that incite the rising drive to breathe. It was postulated that pulmonary and/or thoracic mechanoreceptors may be involved in the arousal mechanism [8]. Alternatively, it is conceivable that the central respiratory drive, rather then the peripheral mechanonoreceptor input may be the primary mechanism that triggers the arousal [9].

More recently, it was reported that the temporal association between the onset of arousal and the restitution of breathing is variable among OSA patients [10]. Younes investigated 82 OSA patients and provoked UA obstruction by dial-down of continuous positive airway pressure. In 17% of the induced obstructive respiratory events, there were no arousals at all, whereas in 22% resumption of breathing preceded the onset of arousal by 0.5–12.0 s. While these findings would challenge the concept that arousing from sleep is an indispensable mechanism in the termination of UA obstruction, they do not invalidate the fact that the vast majority of obstructive respiratory events are associated with arousals, regardless of their role in restoring the patency of the UA.

Other authors have emphasized the importance of UA mechanoreceptors in OSA [11,12]. Pressure-sensing mechanisms play a prominent role in modulating upper airway neuromuscular activity during wakefulness and sleep. A negative pressure reflex within the upper airway serves to stabilize the upper airway during inspiration. Impairment of this reflex may predispose to OSA [12]. Application of local anesthesia to the UA increases apnea duration, suggesting that suppression of UA mechanoceptor activity impairs the arousal response to obstructive breathing [13]. In an investigation on the relation between increased UA resistance and arousal, it was observed that flow limitation is invariably present in respiratory-related arousals, and often precedes rises in respiratory effort [14]. In this study, which involved normal subjects in whom alcohol was used to increase upper airway resistance, increased respiratory effort preceded arousals in only 23% of the respiratory events. This finding would imply that other mechanisms than increased respiratory effort are associated with sleep interruption in normal subjects under certain conditions. Indeed, susceptible non-apneic individuals might awaken easily from sleep by mere stimulation of UA receptors, whereas this mechanism may be blunted and forceful inspiratory effort may be required to cause arousal from sleep as the severity of SDB progresses over time.

Measurement of respiratory effort

As respiratory effort is paramount in the pathophysiology and operational definition of OSA, its measurement is an integral part of the standard polysomnography montage. The revised manual for the scoring of sleep and associated events by the American Academy of Sleep Medicine (AASM) recommends the use of esophageal manometry, or dual thoracoabdominal respiratory inductance plethysmography for this purpose [2,15]. Esophageal pressure (Pes) monitoring is the technique of choice, because the pressure signal directly reflects the respiratory muscle force. As it is an invasive method, it is faced with problems of poor acceptance and tolerability. Moreover, there are concerns that an indwelling catheter in the UA could modify pharyngeal dynamics [16]. It has been suggested that Pes monitoring may negatively affect sleep quality [17,18]. Most sleep centers do not routinely employ this technique, because it is quite cumbersome and expensive. For these reasons, it would be preferable to use noninvasive monitoring techniques as they are better accepted by patients. Several alternative methods for monitoring respiratory effort are available to date. They are based on physiological signals related to breathing activity, including thoraco-abdominal movement, pulse transit time (PTT), respiratory sound, electromyography (EMG) of the respiratory muscles, suprasternal pressure, jaw movement, and venous pressure. The fundamental question is, however, whether these techniques can measure respiratory effort as reliably as Pes monitoring?

Research methods

PubMed and Cochrane central register were searched up to September 2014, using a combination of MeSH terms and free text.