Detection of Increased Upper Airway Resistance During Overnight Polysomnography

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Study Objectives: To examine the utility of four methods used to detect increased upper airway resistance leading to arousal from sleep.

Design: Ten overnight sleep studies were conducted on normal subjects who reported increased snoring and/or witnessed apneas following alcohol ingestion. Alcohol was used to increase upper airway resistance in these normal subjects before overnight polysomnography. Four methods to detect the presence of increased upper airway resistance were used: esophageal pressure manometry; respiratory inductive plethysmography; a piezoelectrically treated stretch sensor adhered to the supraclavicular fossa; nasal flow measured with oxygen cannula and differential pressure transducer.

Setting: Private Sleep Laboratory

Participants: ten normal, healthy volunteers (5 male, 5 female).

Interventions: Alcohol ingestion as red wine (14% alcohol), 180-540mL one to two hours before overnight polysomnography. Esophageal catheterisation.

Measurements and Results: Two hundred twenty-seven electroencephalogram arousals were preceded by inspiratory flow limitation and/or increased respiratory effort. Flattening of the nasal flow profile preceded all 227 arousals. In contrast, only 40% of arousals were preceded by an increase in the size of the stretch sensor signal, 22% by more-negative deflection of the esophageal pressure signal and 21% by increase in the signal size of respiratory inductance plethysmography.

Conclusion: These findings indicate that the most reliable method of detecting increased upper airway resistance leading to arousal from sleep is the nasal cannula/pressure transducer method and suggest that many arousals induced by increased upper airway resistance may be caused by mechanoreceptor afferents.

Key words: increased upper airway resistance; esophageal pressure manometry; nasal flow profile; overnight polysomnography; respiratory inductive plethysmography.

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INTRODUCTION

AROUSAL FROM SLEEP DUE TO INCREASED UPPER AIRWAY RESISTANCE BUT WITHOUT APNEAS OR HYPOPNEAS MAY BE DUE TO CHANGES IN BLOOD GASES, CHANGES IN THORACIC DYNAMICS, AND POSSIBLY UPPER AIRWAY STIMULATION. We specifically investigated brief episodes of increased upper airway resistance that did not result in hypopnea but caused arousal, and we examined 4 different methods to detect the increased resistance leading to arousal.

In 1993 Guilleminault et al[1] described the upper airway resistance syndrome (UARS) as being characterized by symptoms typical of the obstructive sleep apnea-hypopnea syndrome (OSAHS) but with a polysomnographic recording that showed the presence of loaded breathing due to high upper airway resistance without the typical findings of repetitive obstructive apnea. The evidence that the high upper airway resistance in sleep was causal came from the improvement in symptoms in these subjects during a treatment trial of nasal continuous positive airway pressure. Guilleminault[1] used an esophageal catheter to measure inspiratory driving pressure (Pes) and demonstrated the presence of high upper airway resistance by showing very large levels of negative inspiratory pressures, indicating large efforts against a partially closed upper airway.

These authors provided evidence that the high upper airway resistance led to multiple brief arousals in a manner similar to that which occurs in OSAHS and argued that these arousals induced by high upper airway resistance led to daytime sleepiness.

Pes manometry requires the insertion of either a balloon-type or water-filled catheter through the nasal airway into the lower esophagus—an invasive and uncomfortable procedure that can cause sleep disruption in its own right[2] and can lead to refusal by patients to have the procedure performed. In subsequent published research on the UARS, Pes manometry has been used to identify high upper airway resistance.[3-12]

Research by Ayappa et al[13] compared Pes with nasal cannula/pressure transducer in the detection of elevated upper airway resistance, specifically those events that have been termed respiratory effort-related arousals (RERAs) by the American Academy of Sleep Medicine (AASM) taskforce.[14] A RERA is defined as at least 10 seconds of increased Pes terminating in arousal and a return to less negative Pes. Ayappa et al[13] found that the nasal cannula/pressure transducer method detected the same RERA events as Pes.

The gold standard measurement of airflow during overnight polysomnography (PSG) is the pneumotachograph, but it is rarely used in routine sleep studies due to the cumbersome mask that must be worn by the patient and the discomfort that the mask causes. Thermistors have been routinely used to measure flow during overnight PSG, but many studies have been performed that have found this method to be inadequate in the detection of hypopneas and flow limitation in comparison to nasal flow using

cannula and pressure transducer.[15-21]

The aim of this work was to examine the utility of noninvasive methods of identifying episodes of high upper airway resistance that cause arousal from sleep and to determine if these methods improve identification of arousals caused by upper airway resistance changes. Only those arousals that were preceded by increased upper airway resistance but did not meet the criteria for hypopnea as outlined in the AASM Task Force report[14] were examined. Many of the arousals that were analyzed were preceded by only 1 to 2 breaths that demonstrated increased upper airway resistance.

METHODS

Subjects

Ten volunteers underwent overnight PSG. The subjects were recruited from colleagues associated with the Peninsula Private Sleep Laboratory. All the subjects were healthy individuals who reported snoring following alcohol ingestion, and 5 of the 10 subjects had documented evidence of snoring and mild OSAHS on previous overnight PSG. A model of increased upper airway resistance during sleep was therefore obtained using alcohol ingestion in normal subjects before the sleep studies were performed. This semicontrolled method resulted in sufficient electroencephalogram (EEG) arousals, due to increased upper airway resistance, so that analysis of the methodologies for detection of these events could be performed.

The 5 men and 5 women had a mean age of 43.4 ± 8.5 years (range 29-59 years) and a mean body mass index of 24.9 ± 3.3 kg/m^2 (range 20.3-31.6 kg/m^2). All of the women were premenopausal. All subjects had 1 to 3 glasses (180-540 mL) of red wine (14% alcohol) 1 to 2 hours before the sleep study. The amount of alcohol ingested was according to each individual's reported amount that induced snoring.

Overnight PSG was performed using the Compumedics S Series (Compumedics, Melbourne, Australia) computerized system. All PSG recordings used standard electrode placement for EEG (C3/A2, O2/A1), left and right electrooculogram, and submental electromyogram to score sleep. Sleep was staged according to the recognized criteria of the American Sleep Disorders Association[22] based on 30-second epochs.

A 2-lead electrocardiogram was recorded. Oxyhemoglobin saturation (SaO_2) was measured via a pulse oximeter (Ohmeda™ Biox 3740, B.O.C Healthcare Inc., Louisville, CO, USA) with a finger probe.

Respiratory parameters that were measured were abdominal and thoracic uncalibrated respiratory inductive plethysmography (RIP); Pes using an air-filled balloon-type catheter connected to a Validyne differential pressure transducer; nasal flow using an oxygen cannula and the differential pressure transducer in the Compumedics preamplifier (NPF); and Optiflex™ UAR sensor (Sleepmate Technologies, VA, USA), which is a piezoelectrically treated 8-mm by 5-cm plastic strip, adhered into the supraclavicular fossa, with a cable incorporating a transducer that is connected to the Compumedics patient-interface box and preamplifier. The signal from the UAR sensor is a sinusoidal waveform, corresponding to respiratory efforts, which becomes larger with increasing respiratory effort. All respiratory parameters were recorded at a sampling rate of 50 per second.

The protocol of this study was approved by the Ethics and Research Committee of Manly Hospital and Community Health services. Each volunteer gave informed consent.

Analysis of the Respiratory Signals in Upper Airway Resistance Arousals

Each arousal that was attributed to upper airway resistance was analyzed in relation to the respiratory signal that preceded it. Changes in the respiratory signals, indicating increased upper airway resistance, were considered in the analysis when such changes occurred for at least 1 breath and no longer than 1 minute. Changes in each respiratory signal were compared to the previous 2 minutes of baseline stable breathing.

Analysis of the respiratory signals was made in order to compare the accuracy and effectiveness of each measurement device ie, Pes, NPF, UAR sensor, and RIP.

The order in which each signal occurred before each upper airway resistance arousal and the time in seconds were measured and compared.

Definitions of Arousal Types

Apneas and hypopneas were classified according to the report of the AASM Task Force,[14] that is, a clear amplitude reduction of a validated measure of breathing during sleep, lasting 10 seconds or longer, and associated with either an oxygen desaturation of more than 3% or an arousal. NPF was used to detect reductions in airflow.

Leg movement arousals were those EEG arousals that were preceded by an increase in the electromyogram activity of the anterior tibialis in the absence of any changes in any other signal.

Arousals that were deemed to be due to increased upper airway resistance were those that were preceded by any 1 of the following occurring for 1 or more breaths but for less than 1 minute: increased negative Pes, flattening of the nasal flow profile on the NPF signal, an increase in the signal size of the UAR sensor, an increase in signal size of the RIP signal, or respiratory paradox seen on the RIP signal. A return to non-flow–limited breathing followed each UAR arousal.

Arousals that were attributed to increased respiratory effort, seen as increases in negative Pes preceding the arousal, were called IPesAs. There were 50 such arousals, and these IPesAs were analyzed separately. We did not use the term *RERA* that has been used by the AASM Task Force[14] because that term is applied to those arousals that were preceded by at least 10 seconds of increased negative Pes and a return to a less-negative Pes after arousal. The arousals in our study that were termed IPesAs consisted of at least 1 breath of increased negative Pes, EEG arousal, and a return to a less-negative Pes. We used these criteria because we were interested in brief subtle events that lead to arousal.

To examine those arousals that were considered to be due to increasing respiratory effort detected by increasingly negative Pes, the IPesAs were also analyzed as a separate subgroup of UAR arousals.

Only those respiratory events (ie, flow limitation, increased respiratory effort, or respiratory paradox) that were associated with EEG arousal were considered in the analysis.

Paired 2-tailed *t* tests were used when comparing 2 means, and single-factor analysis of variance when more than 2 means were being compared. A *P* value of .05 or less was considered significant in both statistical tests; however, all *P* values are reported.
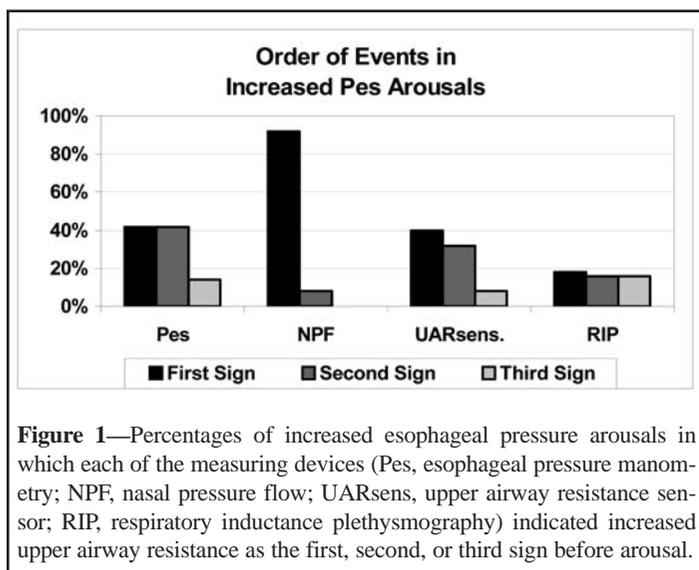
All values reported are mean ± SD.

RESULTS

Ten sleep studies, in which total sleep time (TST), percentage of each sleep stage, latency to sleep onset, and latency to REM sleep, were analyzed.

There was a wide variability in the TST for the group, ranging from 211 to 389 minutes (297 ± 67.2 minutes). The TST did not reflect the amount of time subjects reported sleeping on a normal night (439 ± 64 minutes, $P < .05$).

The amount of time spent in REM sleep was also highly variable, ranging from 4.3% to 29.7% of the TST, with an average percentage of 15.9% ± 9.3% (normal percentage of REM sleep is 20%-25%[23]). The architecture of the non-rapid eye movement (NREM) sleep was relatively normal, with stage 2 NREM sleep constituting 53.6% ± 8.6% of TST (normal percentage is about 50%[23]), while the combination of stages 3 and 4 NREM sleep (slow-wave sleep) represented 25.4% ± 7% of TST, which is within normal limits for this age group. The total number of arousals from sleep in the group was 8.54 ± 3.8 per hour, which is slightly higher than the normal range of 5 per hour for healthy subjects[23] (although a 1995 finding by Mathur & Douglas[24] demonstrates a much higher arousal index for normal subjects ie, 21 per hour). However, the apnea-hypopnea index, according to standard criteria,[14] was within normal limits (1.01 ± 1.4 per hour). The mean SaO_2 for the night was relatively normal ($> 90\%$) with mean SaO_2 for the group being 95.2% ± 2.8% and the minimum SaO_2 being 90.6% ± 3.4%. However, 1 subject had an abnormal minimum SaO_2 of 82% in the absence of any respiratory abnormality, and this was thought to be artifactual due to poor contact of the finger probe. When the data from this subject were excluded from the analysis, the minimum SaO_2 was 91.7% ± 2.2%. There was no significant difference between the minimum SaO_2 in NREM and REM sleep (91.7% ± 2.8% in NREM and 91.3% ± 3.7% in REM, $P = .80$).

The total arousal index was 8.54 ± 3.8 per hour; this included arousals due to apneas and hypopneas (0.77 ± 1.1), leg movements (0.02 ± 0.06), increased upper airway resistance (6.24 ± 3.2), and spontaneous arousals for which no cause was discernable (1.51 ± 1.56).

Figure 1—Percentages of increased esophageal pressure arousals in which each of the measuring devices (Pes, esophageal pressure manometry; NPF, nasal pressure flow; UARsens, upper airway resistance sensor; RIP, respiratory inductance plethysmography) indicated increased upper airway resistance as the first, second, or third sign before arousal.

Arousals Deemed to be Induced by Upper Airway Resistance Events

A total of 227 arousals from sleep were deemed to be due to increased upper airway resistance (according to the criteria outlined in Methods) and called UAR arousals. All 10 subjects had arousals from sleep due to increased upper airway resistance (UAR arousals, including IPesAs).

Analysis of Increased Esophageal Pressure Arousals

The total number of arousals due to increased respiratory effort demonstrated by increasingly negative Pes lasting for at least 1 breath but not longer than 1 minute (IPesAs) was 0.91 ± 0.4 per hour. This totalled 50 arousals in all. Of these, changes in nasal flow occurred in all 50 IPesAs.

All IPesAs were, by definition, preceded by changes in the Pes, indicating increased respiratory effort. Changes in Pes were 1 of the first signs in 42% of IPesAs, 1 of the second signs in 42%, 1 of the third signs in 14%, and occurred an average of 3.2 ± 3.4 seconds before the IPesA. In some IPesAs the first, second, third, or fourth sign is shared by more than 1 of the respiratory signals; therefore, the percentages sometimes exceed 100%.

Change in the nasal flow profile (derived from the NPF), indicating inspiratory flow limitation, was present in all 50 IPesAs and was 1 of the first signs in 92% and 1 of the second signs in 8%. These changes occurred an average of 4.9 ± 4.6 seconds before the IPesA.

The order in which each signal changed before the IPesA (Figure 1), and the time in seconds, were measured and compared. Changes in the UAR sensor signal (ie, increased size, indicating increased respiratory effort) was present in 43 (86%) of the IPesAs and was 1 of the first signs in 40%, 1 of the second signs in 16%, and 1 of the third signs in 8%. These changes occurred an average of 3.5 ± 4.2 seconds before the IPesA.

Changes in the RIP signal (ie, increased size or respiratory paradox, indicating increased respiratory effort) was present in only 28 IPesAs (56%) and was 1 of the first signs in 18%, 1 of the second signs in 16%, and 1 of the third signs in 16%. These changes occurred an average of 2.6 ± 3.5 seconds before the IPesA.

When comparing time in seconds before IPesAs, the Pes was significantly different from only NPF ($P = .05$). There were no significant differences between Pes and the UAR sensor ($P = .80$) or between Pes and RIP ($P = .40$).

None of the respiratory signals demonstrated increased upper airway resistance for longer than 21 seconds before EEG arousal occurred (mean time for all signals, 3.0 ± 1.2 seconds).

The 50 IPesAs were analyzed to determine the percentage in which changes in NPF, UAR sensor, and RIP were present before the arousal (Table 1). Changes in the NPF profile (flattening of the flow curve) were present in all 50 IPesAs. Increases in the size of

Table 1—Percentages of Arousals Related to Increased Esophageal Pressure*

	Measurement device		
	NPF	UAR sensor	RIP
Arousals detected, %	100	86	53

*Defined as electroencephalogram arousals preceded by increased negative esophageal pressure that were also detected by nasal pressure flow (NPF), the upper airway resistance (UAR) sensor, or respiratory inductance plethysmography (RIP)

the UAR sensor-signal size were present before 86% of IPesAs. Increases in the size of the RIP signal or paradox of the chest and abdominal RIP were present before only 56% of IPesAs.

The UAR arousals and IPesAs were distributed across the 5 sleep stages in a similar pattern, with 24.6% of UAR arousals occurring in REM sleep and 25.5% of IPesAs occurring in REM sleep.

The order in which each signal changed before the UAR arousal (Figure 2), and the time in seconds, were measured and compared. A highly significant difference was found ($P < .001$). The percentages of UAR arousals detected by each signal were analyzed (Table 2).

Means of the time in seconds before UAR arousal that each of the signals indicated increased upper airway resistance were compared.

A change in the nasal flow profile of the NPF signal, indicative of upper airway flow limitation, was present in 100% of the 227 UAR arousals and was 1 of the first signs in 93%, occurring an average of 4.7 ± 4.3 seconds before the UAR arousal. The UAR sensor indicated increased upper airway resistance or increased respiratory effort in 40% of UAR arousals and was 1 of the first signs in 18%, occurring an average of 2.4 ± 3.2 seconds before the arousal. The Pes indicated upper airway resistance or increased respiratory effort in 23% of UAR arousals and was 1 of the first signs in 10%, occurring an average of 3.2 ± 3.4 seconds before the UAR arousal. The RIP indicated upper airway resistance or increased respiratory effort in 21% and was 1 of the first signs in 7%, occurring an average of 2.0 ± 2.9 seconds before the UAR arousal.

The first sign was often shared by more than 1 signal, as were the second and subsequent signs—therefore, the totals for each sign may be greater than 100%. Only the first, second, and third signs and not the fourth sign are reported.

DISCUSSION

The principal aim of this study was to compare 4 different methods that are currently used to identify the cause of arousals that may have been due to increased resistance to upper airway flow. Three of the 4 methods, Pes manometry, RIP, UAR sensor provide a measure of increased inspiratory effort. The other method, nasal pressure using oxygen cannula and a pressure transducer (NPF), measures the nasal pressure differential providing a surrogate of flow limitation. The major finding of this
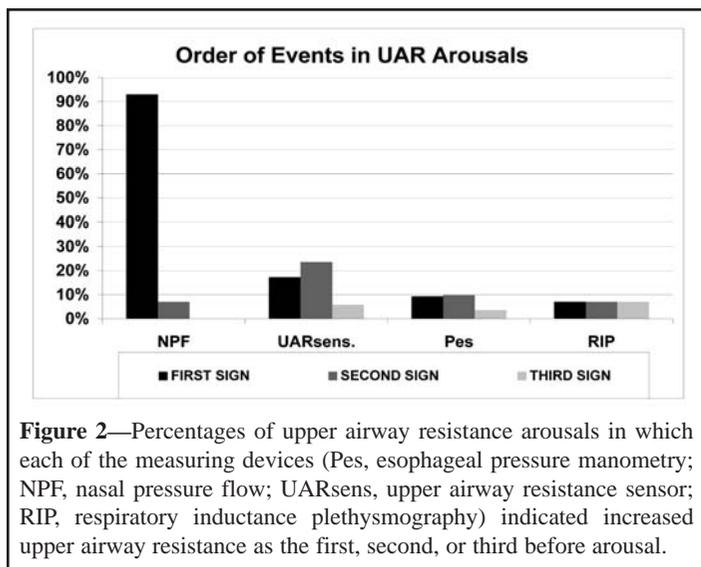
research is that monitoring nasal flow using a pressure transducer (NPF) during overnight PSG is more effective than Pes, UAR sensor, or RIP in identifying arousals induced by partial upper airway obstruction.

Our analysis of UAR arousals included any arousal that was preceded by 1 or more breaths that demonstrated flow limitation or increased respiratory effort. We did not use the term *RERA* because many of the arousals that we included in the analysis were preceded by less than 10 seconds of increased Pes; instead we used the term *IPesA*. When a single breath that was flow limited or required increased effort resulted in an EEG arousal, we considered it to be physiologically important because repetitive arousal during sleep, from whatever cause, has been shown to be associated with the development of excessive daytime sleepiness.[25-28] This study demonstrates that NPF detects very brief and subtle events of increased upper airway resistance that are often missed by other measurement devices. It is clinically important to detect the cause of arousal in order to recommend appropriate treatment.

During the sleep studies of these normal subjects, there were periods of inspiratory flow limitation, detected by NPF that did not result in EEG arousal; these events were not considered in the analysis. Flow limitation during sleep has been shown to be a normal phenomenon,[29] but we were interested in only those events with flow limitation or increased upper airway resistance that were associated with arousal.

When comparing measurement methods for detecting increased upper airway resistance, arousals were separated into 2 groups (ie, IPesAs, in which there was a detectable increase in negative Pes terminating in arousal and a return to less-negative Pes, and UAR arousals, in which any of the 4 measurement methods (NPF, Pes, UAR sensor, or RIP) indicated increased upper airway resistance before the arousal. The 227 UAR arousals included 50 IPesAs.

Hypopneas were not included in the analysis despite increased upper airway resistance being present in those events. There were 53 hypopneas and no apneas in the 10 sleep studies. The criteria recommended by the AASM Task Force[14] were used to score hypopneas in this study (ie, clear reduction in the amplitude of a valid measure of breathing). We believe that many hypopneas have previously been undetected because thermistors were used to measure breathing during PSG and this method has been shown to be ineffective in detection of hypopneas[14,15,17,19-21]. The NPF, on the other hand, detects subtle decreases in breathing (flattening of the nasal flow profile) that terminate with arousal and a return to less flow limitation. By using the AASM Task Force[14] recommendations for scoring hypopneas using NPF, many more events, previously undetected by thermistor, are included in the hypopnea definition. In our study, we scored events as hypopneas that consisted of at least 10 seconds of reduced amplitude of the NPF if they terminated with arousal or

Figure 2—Percentages of upper airway resistance arousals in which each of the measuring devices (Pes, esophageal pressure manometry; NPF, nasal pressure flow; UARsens, upper airway resistance sensor; RIP, respiratory inductance plethysmography) indicated increased upper airway resistance as the first, second, or third before arousal.

Table 2—Percentages of Upper Airway Resistance Arousals, Detected by Nasal Pressure Flow, that also had Increasing Negative Esophageal Pressure

	Measurement device		
	NPF	UAR sensor	RIP
Arousals detected, %	23	40	21

NPF refers to nasal pressure flow, UAR, upper airway resistance sensor; RIP, respiratory inductance plethysmography.

were associated with a 3% or greater oxygen desaturation. Most hypopneas in our study consisted of more than 10 seconds of reduced amplitude of breathing, measured on the NPF, by 10% or more but less than 50%. Thus, only relatively short episodes of flow limitation or flattening of the NPF profile were included in the analysis of UAR arousals and IPesAs.

In this study, all UAR arousals (including IPesAs) were preceded by flow limitation evident on the NPF, but this sign was also the first indication of increased upper airway resistance in 93% of the UAR arousals. This research adds to the body of evidence from other studies[15-21] that suggest that nasal pressure used as an indicator of nasal airflow should be routinely monitored during diagnostic PSG to accurately detect an increase in upper airway resistance. However, our work extends previous studies by showing that many arousals that are associated with upper airway flow limitation occur before any evidence of increased breathing effort.

The way in which NPF detects changes in flow limitation in the upper airway is by the use of nasal oxygen cannula or prongs connected to a differential pressure transducer that records pressure at the nares relative to barometric pressure. The NPF detects airway collapsibility, giving a surrogate of flow limitation. A study by Issa and Sullivan[30] examined arousal responses to airway occlusion in normal subjects and found that the time between airway occlusion and arousal from NREM sleep varied widely. Some arousals occurred after 1 occluded breath, while other arousals did not occur until a minute of occlusion had occurred. They proposed that there are at least 2 major afferent systems producing arousal from sleep in response to airway occlusion, ie, short latency responses mediated by fast mechanoreceptors in the upper airway and the slower responses being mediated by chemoreceptors, in particular, the carotid bodies. In another study by Issa, McNamara, and Sullivan[31] comparing snout mask occlusion with tracheostomy occlusion in sleeping dogs, the authors demonstrated the importance of upper airway mechanoreceptors, postulated as being present in the mucosa, as being the faster mechanism causing arousal from sleep when the airway is occluded. They proposed that this finding complements their previous findings of short-latency arousal responses to nose mask occlusion in humans.[30] Basner et al[32] and Berry et al[33] used topical anaesthesia of the nasopharyngeal airway and showed that time to arousal from sleep following mask occlusion was significantly increased. Both of these studies demonstrated the importance of upper airway receptors in the arousal response from upper airway occlusion. Our current results also suggest that stimuli associated with upper airway flow limitation (presumably mucosal mechanoreceptors) are an important arousal trigger and may be missed if the indicator of increased resistance is that of increased effort. However, the presence of such upper airway mucosal mechanoreceptors remains speculative.

Our study has clearly demonstrated that inspiratory flow limitation is the most common event observed before arousal from sleep deemed to be due to increased upper airway resistance. Flow limitation, if severe enough to cause a reduction in tidal volume, results in a fall in arterial oxygen saturation and a rise in arterial carbon-dioxide concentration. These changes in arterial oxygen and carbon-dioxide levels stimulate the chemoreceptors and the respiratory control feedback system to increase breathing effort. However, in the arousals ana-lyzed in our study, subtle changes in inspiratory flow, in most cases without leading to increased breathing effort, were sufficient to cause EEG arousal. These subtle changes were detectable only from NPF. The finding, that arousal frequently occurs in response to flow limitation before any blood-gas change is evident, has major significance for understanding and identifying the high UARS. In this study an indicator of increased respiratory effort (Pes, UAR sensor, or RIP), often lasting for only 1 or 2 breaths (probably not long enough to cause changes in arterial oxygen or carbon dioxide) was present before many of the arousals but not present in a substantial proportion, while all had evidence of changes in the contour of the NPF flow signal, suggesting increased collapsibility of the upper airway.

Increases in Pes preceded less than a quarter of the UAR arousals and were the first sign in only 9%, indicating that it is not necessary to have a detectable increase in pleural pressure to cause arousal from sleep due to increased upper airway resistance and that detectable flow limitation, with or without increased inspiratory effort, is sufficient to cause arousal.

It is clear from this study that Pes is not necessary in the detection of increased upper airway resistance leading to arousal in normal subjects with some minor degree of OSAHS or UARS. These data suggest that increased upper airway resistance in sleep, leading to the repetitive arousals that cause sleep fragmentation and excessive daytime sleepiness associated with the UARS, can be identified by flattening of the NPF signal.

The subjects in this study were normal volunteers rather than those diagnosed with the UARS, and alcohol was used to induce increased upper airway resistance in sleep. The amount of alcohol given to each subject was in accordance with the individual's reported amount that caused snoring, witnessed apneas, or other signs of increased upper airway resistance during sleep after alcohol ingestion. The subjects were recruited due to the reported effects that alcohol had upon their breathing during sleep. Each subject ingested between 200 and 540 mL (1 glass = 180 mL) of red wine (14% alcohol) 1 to 2 hours before sleep.

The effects of alcohol on snoring and increased upper airway resistance have been known for many years, and several studies have been performed to demonstrate the effects of alcohol ingestion on normal subjects.[34-35] Alcohol is known to depress arousal responses and may well have altered arousal thresholds in these subjects. The arousal index attributed to "spontaneous" causes was reduced in this group when compared to previous work performed to estimate the number of arousals normal individuals experience during overnight PSG.[24] However, our subjects had many rapid arousals associated with upper airway flow limitation. Therefore, an effect of alcohol cannot explain our results. Normal subjects may respond more briskly to inspiratory airway resistance, while those with the UARS may be more sensitive to mechanical changes such as increasing negative pleural pressure. Patients with the UARS, due to sleep deprivation and changes to arousal threshold, may tolerate higher levels of upper airway resistance before arousal is induced. The next logical step would be to repeat the study in subjects who have been diagnosed with the UARS.

It is clear from this study that monitoring nasal flow using NPF not only is sufficient to monitor upper airway resistance, but is actually more useful in detecting subtle changes in the upper airway that lead to EEG arousal.

ACKNOWLEDGEMENTS

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