Upper Airway Resistance Syndrome Is a Distinct Syndrome

Gastaut and coworkers described sleep-disordered breathing in polymorbid somnolent patients or "Pickwickians" (1). Subsequently, we characterized obstructive events during nonrapid eye movement (NREM) sleep in somnolent but nonobese subjects as obstructive sleep apnea syndrome (OSAS) (2). Further investigations in nonobese individuals revealed repetitive increased respiratory effort terminated by transient arousals, but without associated airway collapse, hypoventilation, or oxygen desaturation: the "upper airway resistance syndrome" (UARS) (3).

UARS is a distinct syndrome that occurs in a distinct population. There is a false assumption that it forms a continuum between primary snoring and OSAS. This is because investigations have not meticulously eliminated confounding factors such as android obesity and other comorbidities in the study populations. Any attempt to understand the differences between OSAS and UARS must be based on investigations in nonobese subjects, and must include sensitive measures of respiratory effort, i.e., esophageal manometry (4), as well as electroencephalogram (EEG) arousal.

In our series of 400 cases of UARS, 93 have "pure" UARS. These patients frequently complain of insomnia, sleep fragmentation, and fatigue (5, 6). Their mean age is 38 $\pm$ 14 yr; 56% are women, and 32% are of east Asian origin. Hence, their sex, age, and racial distribution are different from those with OSAS. The mean body mass index is $\leqslant$ 23.2 $\pm$ 2.8 kg/m^2, the mean respiratory disturbance index is 1.5, and oxygen saturation is $\geqslant$ 95%. Their craniofacial anatomy reveals a predominantly high and narrow hard palate, an abnormally small intermolar distance, an abnormal overjet $\geqslant$ 3 mm, and a thin soft palatal mucosa with a short uvula. In 88% of the subjects, there is a history of early extraction or absence of wisdom teeth (7). Their psychological profile shows a high anxiety score. Other clinical features are cold extremities, postural hypotension, history of fainting, and low blood pressure. In a subgroup of 15 subjects, between 20 and 30 yr of age, orthostasis is present by tilt testing, and is associated with a low mean systemic arterial blood pressure. Four breathing patterns are noted with repetitive transient arousals (8): (*1*) "Pes crescendo": progressively increasing esophageal pressure (Pes), terminated by reversal of the Pes to baseline; (*2*) increased Pes, without crescendo, terminated by a Pes reversal; (*3*) one or two breath increases in Pes preceding a Pes reversal; and (*4*) tachypnea with normal Pes, abruptly terminated by a normal breath. At the beginning of the sleep study, the average peak inspiratory effort during NREM sleep is low (mean Pes, $-$2.5 cm H_2O). Typically, the events are terminated at low negative peak inspiratory pressure ($-$6 cm H_2O) (9).

In contrast, in OSAS, collapse of the upper airway typically occurs when the intrathoracic pressure falls to $-$20 to $-$30 cm H_2O (10, 11). The arousal threshold is at inspiratory pressures of $-$40 to $-$80 cm H_2O, thus indicating that the arousal threshold for increased inspiratory effort is elevated in OSAS (11, 12).

In UARS, the arousal threshold is lower. The recognition of the internal respiratory load is exquisitely sensitive, therefore allowing the patient to wake up in response to small increases in inspiratory effort. The sleep EEG in UARS shows an increase in alpha rhythm (13, 14). There is a relative increase in delta sleep, which persists in the later cycles of sleep. These patients may present with hypotension. The mechanism by which hypotension can occur in UARS has been outlined by Seals and colleagues (15).

In contrast, sleep in OSAS shows a predominance of stage 1 and 2 NREM sleep with a decrease in delta sleep. The absolute power of distribution of EEG bands during sleep shows a preponderance of theta rhythm (13). In addition, there is overactivation of the autonomic nervous system with demonstrable increases in muscle sympathetic nerve activity and increased blood pressure both during sleep and waking hours (13, 16). Clearly, UARS and OSAS markedly differ from each other in terms of their clinical presentation, sleep EEG, and autonomic nervous system responses.

The argument that UARS eventually evolves into OSAS is too simplistic. This does not account for the occurrence in our group of overweight individuals of UARS that does not evolve into OSAS, over a period of years (5). Berry and Gleason (11) hypothesized that polyneuropathy of the upper airway nerve endings induced by snoring (Friberg and coworkers [17, 18]) may lead to impaired upper airway mechanoreceptor function and hence to OSAS. However, this cannot explain the presence of UARS in patients who do not snore. Others have postulated that UARS may progress to OSAS secondary to chronic sleep fragmentation. Why would other conditions associated with chronic sleep fragmentation (such as periodic limb movement disorder) not lead to the development of obstructive sleep-disordered breathing?

We believe that distinct functional arousal reflex pathways originating from peripheral mechanoreceptors exist in these two groups. The subjects with UARS have intact, sensitive receptor function while the subjects with OSAS have primary receptor dysfunction. In other words, subjects with blunted mechanoreceptor responses would develop OSAS, while those with intact or hypersensitive responses would develop UARS. This would explain our group of untreated patients whose UARS did not evolve into OSAS over time. Central nervous system responses to respiratory effort, mediated by these mechanoreceptors, have been investigated by studying respiratory related evoked potentials (19–21) during sleep. Preliminary data from patients with OSAS indicate that these are blunted compared with normal controls (I. M. Colrain, personal communication, 1999).

In summary, the data suggest that a fundamental difference exists between patients with UARS and patients with OSAS. This difference is determined by the different mechanoreceptor function in the two groups, which is, presumably, genetically predetermined and environmentally altered. This might explain

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why subjects with a hypersensitive response pattern will develop UARS, whereas subjects with a dysfunctional response pattern, modified by factors such as chronic respiratory allergies, postpubertal tongue enlargement, etc., will directly develop OSAS. In addition, it is interesting to note that the autonomic nervous system responses are also polar opposites in the two groups (9, 17). We believe that two different "brain" responses best explain the two different syndromes. If appropriate physiologic investigations had focused more on nonobese subjects, these differences would have been observed much earlier.

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Upper Airway Resistance Syndrome Is Not a Distinct Syndrome

The term "upper airway resistance syndrome" (UARS [1]) proved useful in highlighting deficiencies in the definition of the sleep apnea/hypopnea syndrome (SAHS). However, UARS is not distinct from SAHS, nor is there adequate evidence that UARS exists.

A syndrome is "a complex of signs and symptoms resulting from a common cause" (2). Thus, a distinct syndrome has distinct signs and symptoms. Patients with UARS usually have no signs—abnormalities on physical examination (2)—and the symptoms of UARS (1, 3) are identical to those for SAHS (4). Thus, UARS is not a distinct syndrome.

Having dismissed UARS on important semantic grounds, it is equally vulnerable medically. To be a distinct condition, it must have the following:

1. Distinct diagnostic criteria
2. Diagnostic criteria that are abnormal
3. Diagnostic criteria that are specific
4. Evidence of a causal link between the diagnostic abnormalities and clinical features (consequent morbidity)

UARS fails on all criteria.

1. DISTINCT DIAGNOSTIC CRITERIA

In the original description (1), diagnosis required sleepiness, a low apnea–hypopnea index (AHI), and frequent arousals. All reported sleepiness but no specific level of subjective or objective sleepiness was required. No subject "had obstructive sleep apnea syndrome as currently defined" but their AHI was not reported. They had to have at least 10 "short" arousals per hour slept. Esophageal pressure was not a diagnostic criterion for UARS (1). Criteria have been further complicated—not clarified—by use of differing AHI thresholds (3, 5), the introduction of esophageal pressure criteria (6, 7), and the possible use of flattening of the flow–time profile to diagnose UARS (8).

Major diagnostic confusion about UARS centers on the scoring of hypopneas. Many centers have scored hypopneas from thermal sensors. These are excellent for detecting apneas, but poor for identifying hypoventilation; exhalations of 50 and 500 ml have identical temperature. Thus, when it was

recognized that hypopneas and apneas had similar consequences, and it was recommended that hypopneas should be defined by semiquantative rather than thermal techniques (9) and these now include inductance plethysmography, nasal pressure, and pneumotachography (10). Those centers that use these methods to define hypopneas rarely, if ever, classify patients as having UARS, whereas centers that rely on thermal definitions report UARS frequently (5, 6, 11).

2. DIAGNOSTIC CRITERIA MUST BE ABNORMAL

Sleepiness

The threshold for significant sleepiness is based on self-reports and nearly 20% of women and about 7% of men in the normal population report sleepiness (12). An Epworth score of > 8 has been used (3), but the normal range is up to 12 or even 15 (13).

Low AHI

The criterion of low AHI is deliberately set to define normality.

Arousal

The arousal frequency used to define UARS, > 10 per hour, is normal on the first night of polysomnography whether without (median, 16 [95% confidence interval 6–33] per hour for age < 60 yr [14]) or with (mean, 24 [SD 12] per hour [7]) the sleep-disturbing effect of esophageal pressure monitoring. Thus, it is erroneous to use > 10 arousals per hour as a cutoff for abnormality in patients undergoing polysomnography (1, 5, 6). Indeed, all patients with UARS in some studies have normal arousal frequencies at < 30 per hour (3).

Negative Pleural Pressure

The addition of criteria requiring progressive falls in esophageal pressure seemed sensible, but such falls are not synonymous with increasing resistance, and may also result from increased ventilation. A decreasing pressure over 10 s (6) may mean as few as two consecutive breaths with increasing pressure generation and normal subjects have frequent such episodes, especially during rapid eye movement (REM) sleep (15).

Thus, all four criteria are common in the normal population.

3. DIAGNOSTIC CRITERIA MUST BE SPECIFIC

Sleepiness

Sleepiness is not specific to UARS.

Low AHI

AHI is low in all causes of sleepiness except for SAHS.

Arousal

Many sleep disorders including narcolepsy and periodic limb movement disorder have arousal frequencies in the range specified.

Thus, none of the three features of UARS is individually specific to the syndrome. Coexistence of all three is also not specific, as this is common in patients with other medical or psychological causes of their sleepiness. The addition of a pleural pressure condition seems unlikely to result in diagnostically useful specificity, as such episodes are also common in normal subjects (15).

4. CONSEQUENT MORBIDITY

Evidence for morbidity may come from epidemiological or intervention studies. There is no epidemiological evidence that UARS causes morbidity. The most robust interventional evidence comes from randomized controlled trials (16), but none have been performed in UARS. In a nonrandomized uncontrolled trial, patients with UARS had decreased symptoms, sleepiness, and arousals on continuous positive airway pressure (CPAP) (1). It is impossible to draw firm conclusions from decreased symptoms in an uncontrolled trial, particularly as placebo-controlled trials in SAHS have shown marked improvements in symptoms with placebo (17, 18). The decrease in arousal frequency from 31 (SD 13) per hour on diagnostic polysomnography to 8 (SD 2) per hour after CPAP looks superficially impressive. However, the CPAP results were obtained on each subject's sixth polysomnographic night and thus increased familiarity with sleeping while being monitored was a major confounder. There is certainly a need for a randomized placebo-controlled trial of patients fulfilling the criteria for UARS to determine whether this entity truly exists. Sleep medicine has suffered enough from evidence-based medicine "experts" (16) and the term "UARS" should not be used unless robust random controlled trial (RCT) evidence is gathered to show it exists.

Even if such evidence were gathered, it should not become a distinct syndrome, as it would be merely part of a disease spectrum that includes SAHS. I believe there should be a new name, possibly something like the *respiratory arousal syndrome*, to focus on the breathing problems causing sleep disruption. The importance of such all-inclusive terminology goes beyond semantics. The use of separate titles leads to confusion, particularly among nonspecialists, and to the misguided belief that the condition is entirely different from the remainder of the disease spectrum.

CONCLUSION: IS THE UPPER AIRWAY RESISTANCE SYNDROME DISTINCT?

Clearly, the answer is *no*. Patients with UARS have precisely the same symptoms as patients with SAHS. On overnight recordings, their "characteristic" respiratory and neurophysiological findings are normal. The continued use of the term to describe sleepy patients with an ill-defined constellation of sleep study findings allows it to be used as a dustbin term for the diagnostically destitute. The term "UARS" should be abandoned.

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REBUTTAL FROM DRS. GUILLEMINAULT AND CHOWDHURI

Undoubtedly, there are misquotations of our published work in the outline presented by our colleague. The complete absence of publications on *Medline* until 1993, relating to nonsnoring, chronically tired individuals, underscores the heuristic value of our description of the syndrome. To date, patients with these symptoms have been dismissed as having major depression, idiopathic hypersomnolence, chronic fatigue syndrome, etc., clearly with important treatment ramifications.

Unfortunately, many have failed to pursue our lead of identifying the temporal relationship between a carefully defined respiratory event and a cortical response based on central EEG leads and more sophisticated techniques (1, 2). We now know that there exists a complex hierarchy of central nervous system responses, from the trigger of brainstem autonomic reflexes to the passage of the thalamic gate, to the reinforcement of sleep patterns before a cortical activation and arousal. However, many have focused, unnecessarily, on the task of merely counting the number of visually defined arousal patterns.

More importantly, instead of dwelling on semantics and dissecting out the diagnostic criteria, one must ask the right questions. The key questions are as follows:

- How does the central nervous system continuously adjust to changes in upper airway patency during different sleep stages?
- Is this adjustment an inherent attempt to overcome the negative consequences?
- Are there specific preconditions that will lead to an abnormal response during sleep?
  If so, how can these be preconditions be identified and the abnormal pathways be delineated?
- Finally, do the individual responses vary according to the genetic make-up?

To respond to these questions and to decipher the genotypical associations of sleep-disordered breathing we must first accurately identify the clinical variants. For example, we have identified craniofacial dysmorphia, related to a small jaw, in the family members of infants classified as having experienced an ALTE (acute life-threatening event), i.e., obstructed breathing during sleep, and have noted its possible association to a specific gene (3); hence, the importance of differentiating UARS from OSAS. We envision that the progressive subdivision of the different phenotypes will ultimately lead to the identification of the specific genotypes of sleep-disordered breathing.

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REBUTTAL FROM DR. DOUGLAS

The proposers do not clarify the "distinct" nature of "UARS." Patients with mild sleep apnea/hypopnea syndrome are often thin women. Middle-aged women with insomnia and anxiety are common in sleep clinics, and east Asians common in the Bay Area. The other reported clinical features need to be examined in case-control trials to determine whether they are more common in UARS, but they are certainly not specific.

The pleural pressures reported at event termination are within the range found in normal subjects (median, −11 [interquartile range, −8 to −12] cm H_2O [1]). While most of their patients with UARS therefore had no sleep breathing abnormality, some may have had mild, but missed, sleep apnea/hypopnea syndrome. The definitions used for UARS in one of their articles cannot be checked as it is not yet published. In the other (2), hypopneas were not defined, but during the time of recruitment the authors were using thermal sensors plus desaturation for hypopnea identification (3). Thermal sensors are insensitive to hypopneas and thin, young, well-oxygenated people do not desaturate with brief apneas or hypopneas, so hypopneas could have been missed.

The "relative increase in delta sleep" is further proof that these patients do not have increased arousals, despite the proposers' claim of "sleep fragmentation." Both the proposers (4) and we (5) have shown that sleep disruption with arousals that are either visible (4) or not visible (5) on the EEG shows marked *decreases* in slow-wave sleep. This, along with the normal arousal frequencies and normal Pes at arousal, are convincing evidence that most patients labeled as having UARS have nothing wrong with their breathing during sleep.

Unless and until there is a robust evidence base that the syndrome exists, patients should be treated by firm reassurance, not labeled as having an unsubstantiated illness.

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