JCSM Journal of Clinical Sleep Medicine

COMMENTARY

It's possible: why don't we do it?

Commentary on Dupuy-McCauley KL, Mudrakola HV, Colaco B, Arunthari V, Slota KA, Morgenthaler TI. A comparison of 2 visual methods for classifying obstructive vs central hypopneas. *J Clin Sleep Med.* 2021;17(6):1157–1165. doi:10.5664/jcsm.9140 Winfried Randerath. MD

Krankenhaus Berthanien, Institute for Pneumology at the University of Cologne, Solingen, Germany

Sleep medicine currently faces substantial challenges: health care insurers and providers of treatment devices urge simple and cheap sleep studies to reduce costs and manage more patients in shorter periods, respectively. In addition, the SARS-CoV-2 pandemic has led to essential reductions in sleep medical services both in staff and in available beds.¹ All these aspects focus on 1 point: simplification of diagnosis and usage of polygraphy rather than polysomnography. Despite some indisputable benefits-reduction in waiting lists, faster services for severely affected patients, and implementation of telehealth into the portfolio-this approach implies the concentration of 1 single question: does the patient fit into the scheme of obstructive sleep apnea (OSA) and should they be treated with continuous positive airway pressure? It is obvious that this approach thwarts the idea of personalized medicine and individualized therapy, which currently refaces almost all medical fields. This paradigm shift has also brought major developments to sleep medicine in recent years. In particular, the concept of understanding and discriminating pathophysiological traits of OSA allows increased opportunities to select and combine optimally tailored solutions for individual patients. Similar considerations can be described in the broad spectrum of central breathing disturbances.²

Precision sleep medicine means registration of patients' symptoms, analysis of polysomnographic patterns such as rapid eye movement sleep OSA or positional OSA, and accurate description and interpretation of respiratory events. Preconditions of personalized sleep medicine include the differentiation and recognition of OSA phenotypes, the description of subtypes of central sleep apnea without or under positive airway pressure therapy, and the knowledge of benefits and harm of central sleep apnea treatment. This simple enumeration indicates that a precise analysis of obstructive and central breathing disturbances during sleep is of crucial importance.

One must therefore consider the largest portion of disturbances the hypopneas. However, current practice in many sleep laboratories and automated analyses of polysomnography systems often do not differentiate the various disturbances. This is not only of academic interest but may lead to substantial misdiagnosis and mistreatments. For example, if central hypopneas remain undiscovered in the baseline study, then the application of positive airway pressure may uncover the central component. Although the central disease is pre-existing in this case, it could erroneously be diagnosed as treatment-emergent central sleep apnea. The recognition of central hypopneas has a huge clinical impact as it may guide and underline the necessity of devices, which sufficiently eliminate these events.

Therefore, the paper of Dupuy-McCauley et al³ published in this issue of the Journal of Clinical Sleep Medicine is more than valuable as it points out the discrimination of obstructive and central hypopneas. The authors compared the American Academy of Sleep Medicine criteria with our step-by-step algorithm.^{4,5} The most important message of the paper is that both algorithms allow differentiating 60%-70% of hypopneas. The specific strength of both procedures is that they precisely detect nonobstructive events as nonobstructive and identifying central events as central events. The authors therefore confirmed that the detection of central hypopneas is feasible noninvasively (ie, without measurement of the esophageal pressure) based simply on the standard procedures of sleep laboratories. In other words, the differentiation of the vast majority of respiratory disturbances is possible with a routine armamentarium; it is helpful to avoid misdiagnoses and therefore essential for optimal treatment. We just have to do it.

Both algorithms show important similarities and some differences. Parameters focusing on the limitation of the airflow through the upper airways (flattening) and on breathing effort (paradoxical breathing) play an important role in both algorithms, thus causing very similar efficacy of the algorithms. However, snoring within the event complements the American Academy of Sleep Medicine algorithm, while our algorithm includes parameters focusing on respiratory drive (termination of the event, position of the arousal, sleep stages). The interrater reliability shows room for improvement in both algorithms. This underlines that training and experience are required to interpret the respiratory events optimally.

The question arises if the combination of the "best of 2 worlds" might allow for optimal sensitivity and specificity of a noninvasive algorithm. It is worth studying if the addition of the termination of the event and the electroencephalogram

parameters to the American Academy of Sleep Medicine algorithm might improve the results. This is in line with current approaches to detect the pathophysiological traits of OSA based on easily accessible polysomnography parameters, which nevertheless include the neurologic part. Eckert et al⁶ elucidated the 4 components: upper airway collapsibility, muscle responsiveness, arousal threshold, and respiratory drive. These insights might guide clinicians to differential treatment, such as continuous positive airway pressure or mandibular advancement devices for patients with overwhelming mechanical components, stimulation of upper airway muscles for those with impaired responsiveness, and pharmaceutical approaches to optimize arousability or respiratory drive.⁷ However, before these concepts can be implemented broadly into clinical practice, tools for noninvasive evaluation are required. Several investigators addressed this need and described patterns of breathing disturbances or simple tools, applicable during daytime or sleep, to analyze the pathophysiological components.^{8–18} This is especially advantageous regarding the arousability and the loop gain, 2 aspects that have not been considered in the interpretation of polysomnography so far.

We may cautiously state that all these works provide a base for integrating precision sleep medicine into daily routine. They advocate for individualized and specific rather than simplified and general approaches.

CITATION

Randerath W. It's possible: why don't we do it? *J Clin Sleep Med.* 2021;17(6):1149–1150.

REFERENCES

- Grote L, McNicholas WT, Hedner J; ESADA Collaborators. Sleep apnoea management in Europe during the COVID-19 pandemic: data from the European Sleep Apnoea Database (ESADA). *Eur Respir J*. 2020;55(6):2001323.
- 2. Orr JE, Ayappa I, Eckert DJ, et al. Research priorities for patients with heart failure and central sleep apnea. An Official American Thoracic Society Research Statement. *Am J Respir Crit Care Med.* 2021;203(6):e11–e24.
- Dupuy-McCauley KL, Mudrakola HV, Colaco B, Arunthari V, Slota KA, Morgenthaler TI. A comparison of 2 visual methods for classifying obstructive vs central hypopneas. J Clin Sleep Med. 2021;17(6):1157–1165.
- Berry RB, Budhiraja R, Gottlieb DJ, et al; Deliberations of the Sleep Apnea Definitions Task Force of the American Academy of Sleep Medicine. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. *J Clin Sleep Med*. 2012;8(5): 597–619.
- Randerath WJ, Treml M, Priegnitz C, Stieglitz S, Hagmeyer L, Morgenstern C. Evaluation of a noninvasive algorithm for differentiation of obstructive and central hypopneas. *Sleep.* 2013;36(3):363–368.

- Eckert DJ, White DP, Jordan AS, Malhotra A, Wellman A. Defining phenotypic causes of obstructive sleep apnea: identification of novel therapeutic targets. *Am J Respir Crit Care Med.* 2013;188(8):996–1004.
- Owens RL, Edwards BA, Eckert DJ, et al. An integrative model of physiological traits can be used to predict obstructive sleep apnea and response to non positive airway pressure therapy. *Sleep*. 2015;38(6):961–970.
- Osman AM, Tong BK, Landry SA, et al. An assessment of a simple clinical technique to estimate pharyngeal collapsibility in people with obstructive sleep apnea. *Sleep*. 2020;43(10):zsaa067.
- Osman AM, Carberry JC, Burke PGR, Toson B, Grunstein RR, Eckert DJ. Upper airway collapsibility measured using a simple wakefulness test closely relates to the pharyngeal critical closing pressure during sleep in obstructive sleep apnea. *Sleep.* 2019;42(7):zsz080.
- Hirata RP, Schorr F, Kayamori F, et al. Upper airway collapsibility assessed by negative expiratory pressure while awake is associated with upper airway anatomy. J Clin Sleep Med. 2016;12(10):1339–1346.
- Mann DL, Terrill PI, Azarbarzin A, et al. Quantifying the magnitude of pharyngeal obstruction during sleep using airflow shape. *Eur Respir J*. 2019;54(1):1802262.
- Vena D, Azarbarzin A, Marques M, et al. Predicting sleep apnea responses to oral appliance therapy using polysomnographic airflow. *Sleep*. 2020;43(7): zsaa004.
- Messineo L, Taranto-Montemurro L, Azarbarzin A, et al. Breath-holding as a means to estimate the loop gain contribution to obstructive sleep apnoea. *J Physiol.* 2018;596(17):4043–4056.
- Pavsic K, Herkenrath S, Treml M, et al. Mixed apnea metrics in obstructive sleep apnea predict treatment-emergent central sleep apnea. *Am J Respir Crit Care Med.* 2021;203(6):772–775.
- Herkenrath SD, Lacerda C, Treml M, et al. Loop gain in heart failure with reduced ejection fraction and periodic breathing is associated with sleep stage and arousals. *Ann Am Thorac Soc.* 2019;16(12):1591–1595.
- Sands SA, Edwards BA, Terrill PI, et al. Phenotyping pharyngeal pathophysiology using polysomnography in patients with obstructive sleep apnea. *Am J Respir Crit Care Med.* 2018;197(9):1187–1197.
- Bosi M, De Vito A, Kotecha B, et al. Phenotyping the pathophysiology of obstructive sleep apnea using polygraphy/polysomnography: a review of the literature. *Sleep Breath*. 2018;22(3):579–592.
- Dutta R, Delaney G, Toson B, et al. A novel model to estimate key OSA endotypes from standard polysomnography and clinical data and their contribution to OSA severity. *Ann Am Thorac Soc.* 2021;18(4):656–667.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication March 29, 2021 Submitted in final revised form March 30, 2021 Accepted for publication March 30, 2021

Address correspondence to: Winfried Randerath, MD, Krankenhaus Berthanien, Institute for Pneumology at the University of Cologne, Aufderhoeher Strasse 169, 42699 Solingen, Germany; Email: randerath@klinik-bethanien.de

DISCLOSURE STATEMENT

W.R. reports personal fees and travel grants from Weinmann, Heinen & Löwenstein, Resmed, Philips Respironics, Inspire, and Bioprojet. The author reports no conflicts of interest in relation to this commentary.