Obstructive sleep apnea (OSA) has serious cardiovascular and neurocognitive consequences, and the vibrations of snoring alone may contribute directly to atherosclerosis. Despite these detrimental effects, one of the more common motivations for treatment is some variation of “My snoring is bothering my spouse.” Snoring, which can be louder than a vacuum cleaner at a 1 meter distance (~70 decibels), affects the sleep quality of bed-partners, but at least may prompt evaluation of the snorer. Unfortunately, by itself, snoring is not specific for OSA, and further testing is usually required. However, in this issue of JCSM Nakano and colleagues report a breakthrough to simplify this problem: they have used snoring sounds alone to measure OSA severity, and have done so using no more specialized equipment than a “smartphone.”

In their study, respiratory sounds were monitored using a smartphone placed on the chest while patients were simultaneously assessed for snoring and sleep apnea severity in a controlled laboratory setting. Respiratory sounds have a characteristic frequency profile (e.g. in musical terms a particular bass, mid and treble) that provides for ready analysis. To quantify snoring, the authors used the smartphone to measure the peak (top 1%) sound pressure in a low frequency range (bass: 50-300 Hz), which was tightly associated with the peak (top 1%) sound pressure level measured at the trachea and at a microphone 1.2 m from the patient. Moreover, the hypopneic/apneic periods of OSA were identified, paradoxically, by relative peace despite often vigorous efforts to breathe and hypoxemia. It is the accompanying “dips” in total sound pressure level (in the bass and mid frequency range of 50-2000 Hz) that were assessed quantitatively: transient 3 decibel dips in sound pressure (halving of sound power level) were shown to identify respiratory events with impressive accuracy when compared to gold standard polysomnography (PSG). In theory, any modern smartphone could be used in this fashion.

We applaud their efforts. If future research confirms their results, the advance by Nakano and colleagues paves the way for widespread screening for OSA. There is a desperate need for methods to screen patients for OSA, for example, before surgery, and current methods and questionnaires lack specificity. Although home testing with portable monitoring has made impressive advances, they are still nowhere near as ubiquitous and portable as smartphones. Their work also allows for the ongoing real-time home monitoring of OSA in patients already diagnosed with OSA, something not currently possible given the limited access to sleep laboratories and portable monitors for repeated assessments. Accurate home monitoring would enable patients to take control of their disease, similar to blood glucose monitoring for diabetes (rather than hemoglobin A1c measurements every 3 months). Patients could use such technology to observe a progressive reduction in OSA severity with weight loss, providing essential motivation for adhering to a strict weight-loss regimen. Conversely, the effects of weight gain or alcohol intake on their OSA severity may help patients avoid behaviors that adversely affect health. Combined with accelerometry (available on most smartphones) the effects of position therapy could also easily and conveniently be assessed. Perhaps a smartphone with accelerometer could alert patients with supine-dependent OSA to roll onto their side, preempting loud snoring, and the unhappy bed-partner’s inevitable elbow?

Of course, additional research is needed before this technology is ready for home use. A noisier home environment, or multiple snorers (“He/she snores louder than me!”) may overwhelm the technology; a second smartphone nearer to the bed-partner may be needed to subtract this interference. Quieter forms of sleep apnea, such as central or mixed apnea, may go undetected by sound analysis. We encourage the authors to validate and refine their technology for use outside the controlled laboratory environment.

More broadly, these impressive preliminary results emphasize that techniques for OSA diagnosis are now far ahead of our understanding of OSA pathophysiology and treatments for OSA. Put another way—what will we do with all of the patients that we might diagnose with OSA using such technology? CPAP is likely to be refused by many, and thus new treatments are desperately needed. Identifying why an individual patient has OSA (their “phenotype”) is highly likely to help direct treatment. Just as the field is moving away from PSGs for OSA diagnosis, we are now discovering that they contain a wealth of quantitative physiological information that can be used to measure OSA phenotypic traits. For example, the overshoot-undershoot airflow patterns can reveal the ventilatory control (“loop gain”) contribution to OSA, a phenotype that may be amenable to treatment with acetazolamide or oxygen. Frequent but mild oxygen desaturation and a greater proportion of wake and stage N1 sleep may indicate a low arousal threshold or “sleep state instability” that is amenable to treatment with
sedatives. In the future, we envision the use of mobile technology for providing similar clues as to the underpinnings of OSA in an individual. Do snoring frequency profiles identify the site of airway narrowing? Does a faster, more regular “cycling” of obstructive events reveal a major ventilatory control contribution to OSA? Advances in both understanding OSA pathophysiology and monitoring technology could eventually allow us to use our smartphones not only to diagnose and monitor OSA, but to make smart treatment plans.

CITATION
Sands SA; Owens RL. Does my bed partner have OSA? There’s an app for that! J Clin Sleep Med 2014;10(1):79-80.

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DISCLOSURE STATEMENT
Dr. Sands is supported by a National Health and Medical Research Council of Australia Early Career Fellowship (1053201) and R.G. Menzies award. Dr. Owens is supported by the National Institutes of Health (K23 HL105542). He serves as a consultant for Philips Respironics.