Response from the authors to the letter "Pulse rate trends in obstructive sleep apnoea: a reliable tool to predict long term response to CPAP?"

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To the editor,

We appreciate the interest of Dr. Navarro-Esteva in our study (1). We are well aware of the contributions of previous studies within the field by Kufoy *et al.* (2) and Kawano *et al.* (3), which described the changes in physiological parameters but no symptomatic response to continuous positive airway pressure (CPAP) in patients with obstructive sleep apnoea (OSA).

We believe we have explicitly acknowledged the methodological limitations of pulse oximetries pointed out by Dr. Navarro-Esteva in our paper. However, nocturnal pulse oximetries are easily available, often used in clinical settings and comply with evidence-based guidance to diagnose OSA (4). In addition, in a randomised controlled trial they were found to be non-inferior when compared to inpatient polysomnography (5).

Obese patients with lower oxygen saturations are more likely to suffer not only from OSA but also from obesity hypoventilation syndrome (6). This can only be confirmed by nocturnal carbon dioxide measurements, a measurement that is not included in a standard polysomnography setup. Hence, we argue that the contention by Dr. Navarro-Esteva that "for research purposes, diagnosis should be confirmed by polygraphy or polysomnography" is hardly feasible in practice, as the best approach to diagnose OSA remains a controversial issue. The decision over which overnight investigation to employ is influenced by the available resources in the respective health care system. Moreover, in our study any decision to offer treatment was derived from evidence-based guidelines (4) and only 6 out of 58 (10%) patients with mild OSA were offered CPAP following physicians' review.

Dr. Navarro-Esteva is correct in stating that the symptom of hypersomnolence is of multi-factorial aetiological origin (7). Although OSA has got a high prevalence in the general population (8), hypersomnolence affects a larger group of people than those who suffer with sleep-disordered breathing.

The main inclusion criteria in our study was a 4% oxygen desaturation index (ODI) greater than 5/h. We added the pulse rise index (PRI) as additional inclusion criteria for the control group to exclude patients with additional conditions which could lead to autonomic arousals from sleep and affect pulse rate variability.

Zamarrón *et al.* studied the approximate entropy (ApEn) analysis of heart rate data derived from nocturnal pulse oximetries in OSA. They concluded that '(...) the results presented prove that this method is very well suited to recognize the sleep-apnoea-specific cyclic variability of heart rate'. They also postulated that studying the heart rate signal was an effective tool to understand how brain, sleep and autonomic nervous system interact (9).

As regards the definition of PR trend that left Dr. Navarro-Esteva uncertain, in the method section of our paper we have defined '*PR trend*' as the difference between the average pulse rate of the first and the last hour of sleep. We hypothesised that an increase in 'PR trend' reflected an increased sympathetic activation throughout the night and that 'PR trend' could be a predictor of treatment success in

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a group of OSA patients treated with CPAP.

In our study, there was no significant difference in CPAP compliance between groups. The observed trend towards a longer use in the group with a negative 'PR trend' is consistent with the clinical observation that patients who respond better to CPAP treatment are more likely to use it longer.

Heart rate variability, pulse rate and the change in nocturnal pulse rate are important markers of autonomic nervous activity in OSA. Combined with the Epworth Sleepiness Scale (ESS), a patient-based symptoms score, and the response to CPAP therapy they are crucial markers to identify patients who are at an increased risk of cardiovascular events.

In conclusion, our data support the usefulness of nocturnal pulse oximetry recordings in a clinical setting of a sleep centre. Although polysomnography remains the Gold-standard for the diagnosis of sleep disorders, nocturnal pulse oximetry has been shown to provide reliable results (10). Indeed, pulse oximetry data convey not only data on ventilation and oxygen saturation, but they also provide an insight into autonomic nervous activity at-aglance with the potential to identify elevated cardiovascular risks. This finding is not negated by the aforementioned methodological limitations.

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