# Changes in heart rate during obstructive sleep apnoea

## To the Editor:

A paper was recently published in the Journal [1] concerning heart rate (HR) in obstructive sleep apnoea. In this paper a new hypothesis was put forward to try to explain HR changes in obstructive apnoeas. According to that hypothesis, upper airway receptor stimulation in apnoeas could activate postinspiratory neurons; postinspiratory time could then increase and this, in turn, could contribute to HR changes. However, the authors did not perform any expiratory time measurement in apnoeas, that might support their hypothesis. In fact, they only reported their data on total respiratory cycle duration, and assumed that postinspiratory time might undergo similar changes.

Our paper concerning expiratory timing in obstructive sleep apnoeas was previously published in the Journal [2]. In that paper, we showed that postinspiratory time behaviour in obstructive apnoeas is independent of the other phases of the respiratory cycle. In fact, whilst inspiratory time increased, and total expiratory time decreased, progressively, in the apnoeic obstructed breaths, postinspiratory time remained remarkably stable throughout the whole apnoea. In addition, postinspiratory time was sig-

## REPLY

#### From the authors:

CIBELLA et al. [1] studied the expiratory timing in obstructed sleep apnoea in great detail. In addition to a polysomnogram they recorded diaphragmatic electromyogram and airflow with a pneumotachograph. The time of postinspiratory activity was measured from the peak to the end of any detectable diaphragmatic electromyographic activity (representing phrenic nerve after discharge). During preapnoeic breaths the postinspiratory time lasts for about 65% of the total expiratory time. This was found by other investigators for normal breathing [2]. Intracellular recordings of postinspiratory neurons in anaethetized cats has shown that stimulation of the superior laryngeal and vagus nerves activates postinspiratory neurons in the medullary respiratory centre [2, 3]. This causes prolongation of postinspiration, or even apnoea, depending on the strength of the stimulus, and a suppression of the afterdischarge of phrenic motoneurones [3]. Thus, a shortened diaphragmatic postinspiratory activity during obstructed apnoea, as found by CIBELLA et al. [1], is likely to be explained by activation of postinspiratory neurons. Therefore,

nificantly shorter in the apnoeic efforts, than in the interapnoeic breaths.

Therefore, we believe that any inference on postinspiratory time duration in apnoeas, based on the behaviour of total respiratory cycle duration, is unwarranted.

In our study, we examined only obstructive apnoeas, whilst a considerable number of mixed apnoeas was taken into consideration in the study by ANDREAS *et al.* [1]. We have no data concerning postinspiratory time duration in mixed apnoeas. However, we believe that, at least as concerns obstructive apnoeas, the hypothesis advanced by ANDREAS *et al.* [1] cannot be accepted.

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#### References

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we believe, that the important findings of CBELLA et al. [1] are not contradictory to our results, but can be explained by the hypothesis put forward in our paper. However, we concede that this hypothesis should be tested, by measuring postinspiratory activity during obstructed apnoea.

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