

Interrupted breathing during sleep affects brain neurons necessary to regulate heart rate

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Sufferers of a common sleep-breathing disorder have diminished activity among neurons responsible for keeping heart rate low, reveals a new study published today in *The Journal of Physiology*.

The research discovered that in <u>obstructive sleep apnoea</u> (OSA), <u>neurons</u> in the brainstem that control heart rate experience a blunting of their activity. The reduction of <u>neuronal activity</u> likely contributes to the increased heart rate, blood pressure and risk of adverse cardiovascular events that occur in patients with OSA.

OSA is a common cardiovascular disease, occurring in 24% of adult males and 9% of adult females, which causes repetitive interruptions of breathing during sleep. Lack of oxygen during these episodes brings the person to a lighter state of sleep or brief wakefulness to restore normal breathing. Cycles of interrupted breathing and arousal from sleep can occur as frequently as once per minute.

Dr David Mendelowitz, who led the study at The George Washington University USA, says:

"Lack of sleep leaves the mind and body tired, leading to poor mental and physical performance, and if untreated OSA increases a person's risk of developing hypertension and irregular heartbeats. Therefore it is very important that we have discovered some of the underlying mechanisms



that could injure the heart and other cardiovascular tissues.

"Our study shows that progression of blunted cardiovascular reflexes is accompanied, and likely maintained by, inhibition of neurons in the <u>brainstem</u> that protect the heart and normally maintain a low resting heart rate. This study would predict that patients who have OSA, and also take sleep medicines, might be at heightened risk for an exaggerated reduction of essential neuronal activity that protects the heart."

The team explored these mechanisms in rats, by mimicking OSA for four weeks and studying the changes in blood pressure, heart rate, and synaptic activity in parasympathetic neurons that control heart rate.

Future work will need to build from this foundation and focus on finding targets to restore the usual cardio-protective function of these neurons to help reduce the risk of arrhythmias, elevated <u>heart rate</u>, and <u>blood</u> <u>pressure</u> that occur with this disease.

Provided by Wiley

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