Sleep apnea may offer unusual protection for heart attack patients

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(Medical Xpress)—People who suffer from breathing disorders such as sleep apnea are usually at higher risk for cardiovascular disease. But an intriguing new study from Technion-Israel Institute of Technology scientists suggests that some heart attack patients with these conditions may actually benefit from mild to moderate sleep-disordered breathing.

Apnea and other types of sleep-disordered breathing can boost the numbers and functions of rare cells that help to repair and build new blood vessels, according to the Technion's Dr. Lena Lavie and her colleagues. They say the findings could help predict which patients are at a greater health risk after a heart attack, and may even suggest ways to rebuild damaged heart tissue.

Sleep-disordered breathing is characterized by cycles of apnea-induced hypoxia, where the sleeper experiences a temporary drop in oxygen levels. It occurs in about 5 to 10% of the general adult population, but is extremely common in patients with cardiovascular diseases—somewhere between 40-60%. Many studies have shown that sleep apnea is a risk factor for everything from high blood pressure to chronic heart failure, Lavie noted. Earlier studies by the Technion scientists suggest apnea increases oxygen-related stress and inflammation in the heart and blood vessels.

The scientists' study in the American Journal of Respiratory and Critical Care Medicine could help resolve a puzzling medical issue. If sleep disordered breathing is associated with cardiovascular disease, why is it

that people who suffer from breathing disorders in sleep seem to do as well as healthy sleepers after a heart attack?

Lavie, along with researchers Dr. Slava Berger, Prof. Doron Aronson and Prof. Peretz Lavie, looked for clues to this puzzle in 40 male patients—a mix of healthy sleepers and those with sleep disordered breathing—who had had a heart attack just a few days earlier.

Blood samples drawn from these patients revealed that the sleep disordered breathing patients had markedly higher levels of endothelial progenitor cells (EPCs), which give rise to new blood vessels and repair the injured heart, than the healthy sleepers. They also had higher levels of other growth-promoting proteins and immune cells that stimulate blood vessel production.

The Technion researchers were able to trigger a similar increase in vessel-building activity in vascular cells taken from a second set of twelve healthy men and women, by withholding oxygen from the cells for short periods. "Indeed, our results point at the possibility that inducing mild-moderate intermittent hypoxia may have beneficial effects," Lena Lavie said.

In an accompanying editorial in the journal, Dr. Leila Kheirandish-Gozal of the University of Chicago and Prof. Ramon Farré of the Universidad de Barcelona said the Technion study moves toward reconciling the ideas that apnea can stress the heart but also "pre-condition" it for repair.

Patients with sleep-disordered breathing, they noted "are essentially better prepared to harness the recruitment of EPCs when [a heart attack] comes knock at the door."

"Heart attack is a potent stimulus for EPC mobilization," said Aronson, who is also affiliated with RAMBAM Medical Center. He also explained

that the cells move from bone marrow to the heart to repair damaged tissue after a heart attack.

"The field of cell-based cardiac repair has struggled to find the best approach to enhance recruitment of EPCs to the heart following myocardial infarction," said Aronson. The Technion findings, he said, suggest that intermittent periods of oxygen deprivation in heart attack patients "provides a simple and powerful means to boost EPC mobilization."

"It should be further investigated if inducing intermittent hypoxia immediately after a heart attack, in patients without sleep disordered breathing, will also have such an effect," Lena Lavie said.

The researchers would like to test this possibility in animal studies, as well as expand their studies of the underlying mechanisms that activate EPCs and other vessel-building factors.

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