

## Apnea Treatment Gets Heart Back in Shape

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1 comment(s)

Treating obstructive sleep apnea may help prevent heart failure by reversing structural and functional impairment seen with the sleep condition, researchers found.

Moderate-to-severe obstructive sleep apnea was associated with significantly larger left ventricles and poorer filling and emptying parameters compared with controls, similar to what was seen in hypertensive adults in a study by Gregory Y.H. Lip, MD, of the University of Birmingham, England, and colleagues.

But after six months of treatment with continuous positive airway pressure (CPAP), most of those measures normalized or improved, the group reported online in *Circulation: Heart Failure*.

"This may imply that obstructive sleep apnea could be crucial in development of left ventricular diastolic dysfunction, which may lead to heart failure and increased mortality if left untreated," they concluded.

The study included 40 consecutive, otherwise-healthy patients who had confirmed moderate to severe obstructive sleep apnea seen at the sleep laboratory at a single hospital.

A battery of cardiac assessments, including 2D and 3D echocardiography, was done on them at baseline and after treatment with CPAP for 26 weeks.

Structure of the left ventricle was impaired at baseline in this group to the same degree as in a group of 40 otherwise healthy essential hypertension patients without sleep apnea and when compared with 40 healthy patients without either condition.

The posterior wall was thicker at 1.2 cm in both groups compared with 1.0 cm in the controls ( $P<0.0001$ ).

Left ventricular mass index, too, was higher at 124 g/m<sup>2</sup> in the apnea group and 121 in the hypertension group compared with 102 in the controls ( $P=0.009$ ).

But the apnea group saw improvements with treatment:

Septal thickness declined from 1.1 to 0.99 cm ( $P<0.0001$ ).

Posterior wall thickness decreased from 1.2 to 1.1 cm ( $P=0.02$ ).

That reduction in left ventricular thickness "may have long-term prognostic implications," the group noted.



### Action Points

Several studies have suggested that obstructive sleep apnea contributes to the development of left ventricular systolic and diastolic dysfunction and, possibly, heart failure.

Note that this study indicates that moderate-to-severe sleep apnea can cause structural and functional changes in left ventricular function comparable to that seen in hypertension and, further, these abnormalities significantly improve following CPAP therapy.

Nearly every measure of left ventricular diastolic function came out significantly worse for the apnea and hypertension groups than for healthy controls.

Again, though, CPAP therapy improved the outcomes compared with baseline for the apnea group. These changes included:

Ratio of early to late filling improved from 1.0 to 1.4 ( $P<0.0001$ ).

Ratio of mitral early inflow to septal velocity improved from 9 to 8 ( $P=0.02$ ).

Iso-volumetric relaxation time improved from 0.09 to 0.07 ms ( $P<0.0001$ ).

Left atrial volume index improved on both 2D and 3D echo (both  $P<0.0001$ ).

Those changes in left atrial volume may have an impact on outcomes too, because an enlarged left atrium independently raises risk of atrial fibrillation, heart failure, and other cardiovascular events, Lip's group noted.

With regard to left ventricular systolic function, tissue Doppler imaging showed that mitral annular 'S' wave velocity was slower in both apnea and hypertension groups compared with controls ( $P<0.0001$ ), but CPAP treatment drew the apnea group even with controls ( $P=0.01$  for improvement).

Other functional improvements with CPAP treatment for the apnea group were:

Left ventricular end-diastolic volume jumped from 114 to 136 ml on M mode imaging ( $P<0.0001$ ).

Left ventricular end-systolic volume tended to improve on M mode and 2D echo imaging as well ( $P=0.08$  and  $P=0.09$ , respectively).

Left ventricular ejection fraction increased from 64% to 74% on M mode imaging, with similar improvements seen on 2D and 2D echo ( $P=0.02$  to  $P<0.0001$ ).

How exactly CPAP improves systolic function isn't clear, but it may involve reducing blood pressure, hypoxia, rapid intrathoracic pressure changes, and secondary hemodynamic disturbances, the researchers suggested.

Blood pressure fell from a mean of 144/83 mm Hg before apnea treatment to 133/80 mm Hg after 26 weeks ( $P<0.0001$  for systolic and  $P=0.02$  for diastolic pressure).

The mechanism for diastolic improvements likewise need further study but may be correction of surges in blood pressure, hypoxia, and hypercapnia with over-activation of the sympathetic system, Lip's group added.

They cautioned about the unblinded nature of the study and that 24-hour ambulatory blood pressure monitoring wasn't required.

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The researchers reported having no conflicts of interest to disclose.

#### From the American Heart Association:

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