

Second-generation drug used for hypertension aids heart function independent of blood pressure

April 22 2012

Heart failure is the most common cause of death throughout the world, typically the result of chronic high blood pressure, also known as hypertension. As a result, research efforts have focused on an array of approaches aimed at preventing and treating high blood pressure. Recently, Japanese researchers examined the utility of an anti-hypertensive drug, moxonidine, which acts on the imidazoline receptors in the cardiovascular center of the brainstem. They found, using an animal model, that the drug can improve heart function and survival independent of its effect on blood pressure. They also found the drug had a favorable effect on oxidative stress, which is related to insulin resistance, the underlying abnormality in diabetes, which is common in people with heart failure.

An abstract presentation about the findings will be offered at the meeting Experimental Biology 2012, being held April 21-25 at the San Diego Convention Center. The study was conducted by Yoshitaka Hirooka, Nobuhiro Honda, Ryuichi Matsukawa, Koji Itou and Kenji Sunagawa, all of the Department of Cardiovascular Medicine, Kyushu University Graduate School of Medical Sciences in Fukuoka, Japan. It is entitled, "Central sympathoinhibition improves left ventricular function during the transition from hypertrophy to [heart failure](#) in Dahl salt-sensitive rats." The abstract is sponsored by the American Society for Investigative Pathology (ASIP), one of six scientific societies sponsoring the conference which last year attracted some 14,000 attendees.

Heart failure is a chronic disease that takes many forms and a variety of medications are used to treat it. Drugs such as ACE inhibitors and beta blockers target the causes of systolic heart failure. Clonidine, a first-generation central sympathoinhibitory drug, targets [brain receptors](#) that reduce cardiac output and [lower blood pressure](#). Moxonidine, a second-generation drug, targets diastolic heart failure and function by reducing the effect of the [central nervous system](#) (CNS) receptors to decrease sympathetic activation and thus reduce [blood pressure](#). In the study, salt-sensitive, hypertensive rats either received Moxonidine or were assigned to the control group. Researchers later found that the animals who received the drug had a marked inhibition of the sympathetic activity (an area of the brain) compared to those that did not. The findings suggest that inhibition of the central sympathetic outflow is important in the mechanism of hypertension. According to Dr. Hirooka, "The findings are important because they suggest that moxonidine may be useful in targeting the central receptors in the brain that are known to occur in patients with hypertension."

Next Steps

The study is the latest in a series conducted by the research team whose focus is on neural control of circulation in hypertension and heart failure. Looking ahead, they will work to identify the precise mechanisms involved in the beneficial effect of moxonidine, Dr. Hirooka said. They will also study other ways to see if the compound is a possible therapeutic tool for hypertensive heart disease to prevent heart failure. As the drug had beneficial effects on [insulin resistance](#), they would like to further investigate the issue, he added.

Moxonidine is available in select countries in Europe and Asia. It is not currently available in the United States.

Provided by Federation of American Societies for Experimental Biology

Citation: Second-generation drug used for hypertension aids heart function independent of blood pressure (2012, April 22) retrieved 25 April 2024 from

<https://medicalxpress.com/news/2012-04-second-generation-drug-hypertension-aids-heart.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.