Editorial Comment

Understanding and clinical application of acute dilation of the left ventricle on ventricular arrhythmias

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In this issue of the Journal of Geriatric Cardiology, Yin et al discussed the effects of calcium preconditioning (CPC) and streptomycin (S) on acute dilation of the left ventricle.

This bench experiment was set up with an isolated heart connected to a Langendorff apparatus. Then the left ventricle was dilated with a latex balloon placed within the ventricle. The study composed of four groups of experiments: a control group to be treated with placebo, a CPC group to be treated with CPC, an S group to be treated with S, and a CPC + S group to be treated with both calcium preconditioning and streptomycin.

The results showed that the group with the highest number of premature ventricular beats (PVCs) (90%) and tachycardia (70%) was the control group. The group with the lowest frequency of PVC (5%) and tachycardia (10%) was the group treated by both CPC and streptomycin. The streptomycin group showed less arrhythmia than the CPC alone group. This study tried to prove that mechanical overload and wall stretch lead to a mass influx of calcium into the cell. These positive ionic charges triggered the action potential and altered the intracellular calcium cycling through the stretch-activated ion channels (SACs). These changes were resulted on the surface electrocardiogram as frequent single PVCs or repetitive PVCs, which are called ventricular tachycardia. After infusion of either or both CPC and streptomycin, these arrhythmias became rare because CPC allowed the cell to gradually adapt to the new environment while streptomycin inhibited the stretch-activated ion channels. On a clinical basis, CPC and streptomycin prevented the development of ventricular arrhythmias from acute mechanical ventricular dilation.

From the point of view of a clinical cardiologist, which clinical scenario mimics the above bench experiment? In a patient with acute aortic regurgitation (AAR) due to infarction or perforation of a mitral valve leaflet from endocarditis, there is sudden increase in the left ventricular size secondary to the presence of a new large regurgitant jet and volume in diastole. The patient could develop sinus tachycardia, low blood pressure, or shock. The increased heart rate and the lower blood pressure are the differences when comparing with the fixed heart rate and stable blood pressure of the isolated hearts in this bench study of Yin et al. The reason is that the hearts of the rats are isolated, with all the sympathetic and parasympathetic nerves cut: these hearts are truly denerved. If these hearts are studied on an intact animal, in order to neutralize the sympathetic and parasympathetic effect, then betablockers should be given to prevent the increase in heart rate while the vagus nerve should be isolated and cut to prevent paradoxical bradycardia.

The next clinical situation which could best mimic acute dilation of the left ventricle is acute mitral regurgitation (AMR). It happens from acute rupture of a papillary muscle due to infarction or perforation of a mitral valve leaflet from endocarditis. In these clinical scenarios, the hemodynamic disturbances are so overwhelmed (severe shortness of breath, hypotension) and eclipse the more benign arrhythmia (sinus tachycardia) at that moment. The explanation is because the main hemodynamic change of AMR is sudden elevation of the left atrial pressure without increase in left atrial volume or left ventricular end diastolic pressure and volume. So AMR does not duplicate the main mechanism of the bench study, where acute dilation of the left ventricle was induced by a small balloon.

Which other clinical situations could mimic the enlarged ventricle of the bench study? In the case of enlarged (dilated) cardiomyopathy due to long standing alcohol intake, the myocardium is badly damaged with diffuse fibrotic changes, while the hearts of the rat in this experiment are quite healthy and young.

What is the difference between the enlarged (dilated) ventricle of diabetic or ischemic cardiomyopathy and the hearts of this experiment? The main difference is that the coro-
nary arteries are patent in the rat heart while they are diseased in patients with diabetic or ischemic cardiomyopathy. However, in the case of gradual increased size of the ventricle as seen in patients with alcoholic or viral cardiomyopathy or ischemic cardiomyopathy, the influence of calcium preconditioning and streptomycin would be beneficial because the high incidence of severe and repetitive ventricular arrhythmia (tachycardia) in these patients.

This bench experiment of S and CPC on SACs by Yin et al. opens up new possibilities in cardiovascular treatment and preventive measures. Active athletes with large and hypertrophic hearts or patients with chronic aortic regurgitation are prime candidates for further studies. The strenuous lifestyles of these people, when combined with a lot of sweating (so a big loss of potassium) only make them more vulnerable for arrhythmias. Further multicenter, blinded clinical trials are needed.

As clinical cardiologists, we appreciate the time, enthusiasm and sacrifice dedicated by basic scientists such as Yin et al. for the development of science. The results of their work help us to understand more the normal physiology or the disease state of the heart in life, in sickness or in death. Their noble efforts are always treasured and appreciated.