Abstract

Atrial fibrillation (AF) is the most common supraventricular rhythm disorder that adversely affects hemodynamic parameters through a combination of loss of atrial contribution, too fast or slow ventricular response, irregular heart rhythm and neurohumoral activation. The relative contribution of each pathological mechanism to the deterioration of hemodynamic parameters during AF has not yet been elucidated. One reason for this is the absence of an adequate study model.

Plasma natriuretic peptide (NP) levels are elevated in patients with FS compared with patients with sinus rhythm (SR), irrespective of the presence of concomitant heart failure. It is unclear whether this relationship is mediated by hemodynamic changes during AF or by heart rhythm irregularity itself. Decreases in cardiac output and beat-to-beat heart rate variations in AF are associated with impaired cerebral perfusion. Similar changes can be expected in other organs with high minute flow, such as the kidneys.

In this study, we evaluated the relative contribution of loss of atrial contraction, ventricular shortening, and heart rhythm irregularities to hemodynamic indices during simulated AF. In the second part of the study, we investigated the acute effects of heart rate, heart rate irregularities, and left atrial pressure on plasma natriuretic peptide levels during induced AF and during rapid regular atrial pacing. In the last part, we used intravascular echocardiography to assess renal artery blood flow and compared measurements during sinus rhythm, induced AF, and regular atrial pacing.

We demonstrated a negative effect of FS on various parameters of left ventricular (LV) systolic and diastolic function. Loss of effective atrial contraction and irregular heart rhythm contributed significantly to the adverse hemodynamics. Heart rate (HR) alone increased the impact of loss of atrial contribution and heart rhythm irregularity.

Another finding was that plasma natriuretic peptide concentrations increased during AF independently of pressure changes and HR and that irregularity of cardiac action was responsible for the elevation.

In a last work, we demonstrated the feasibility of measuring renal blood flow by intracardiac echocardiography during different supraventricular rhythms. We showed that induced AF does not cause a significant acute reduction in renal flow in healthy subjects, probably due to the maintenance of stable systemic arterial pressure and compensation by increased HR.

Key words

Atrial fibrillation, hemodynamic changes, cardiac output, heart failure, natriuretic peptides, renal arteries