Summary

The haemodynamic benefit of the atria to the net performance of the heart is called atrial contribution (AC). Definitions of AC are different. In cardiology, AC is linked either to ventricular filling or to the systolic function of the heart, i.e. to the systolic volume or cardiac output. Several non-invasive methods for the quantification of AC are described in the literature but none of them are commonly used in the routine practice of cardiac pacing centres. The reasons for this vary; for example the time-consuming nature of the methods, observer-dependency or the limited reproducibility of measured values.

The aim of the presented thesis was to develop an original method for the estimation of an index of atrial contribution to the systolic volume of the left ventricle (ACSp) in patients treated by dual chamber pacing. In principle, the method is based on the quantitative analysis of a „beat-to-beat" induced decrease in the pulse amplitude of the pulse oximetry signal (SpO_2). The change in the amplitudes is being induced by a sudden „cut-off" of the stimulus to the right atrium, when the rate of ventricular stimuli is strictly maintained at a constant level. The obtained ACSp is given as a percentage of the pulse amplitude of SpO_2. The method is independent of the observer, non-invasive and relatively fast. Good agreement between ACSp and another atrial contribution index estimated by a similar method using the tracing of the invasively registered aortal pressure was proven in the study.

In a group of 47 patients, the following values of ACSp were obtained when measured at an AV delay of 200 ms: mean 33.5 %, median 34.6 %, minimum 6.2 % and maximum 71.8 %. Good reproducibility of ACSp was proven from analyses of paired values obtained within the time range of either 10-20 minutes (average difference between two paired measurements -0.5 ± 6.1 %) or 24 hours (average difference between two paired measurements 2.1 ± 7.9 %).

In a group of 24 out-patients, a substantial increase in ACSp was shown 2 hours after the administration of intravenous furosemide. The median ACSp increased from 30.4 % to 43.3 % ($P < 0.001$). The increase in ACSp was probably partly mediated by activation of the sympathetic nervous system. The results were compatible with the hypothesis of the important physiological role of the atria for the maintenance of cardiac output when a rapid loss of extracellular fluid occurs.