The present study using Fourier analysis WI mode to 4D5j dv^dt in DDD mode 1 to 5D4j but not in VVI mode. Diastolic dv^dt was significantly greater during Ex than at rest in DDD mode (3.2 vs 2.6 EDV/sec, p<0.05). However, it showed no significant increase in VVI mode (3.3 vs 3.5, NS). In three patients an increase in ventricular rate from 70 to 110 bpm enhanced diastolic dv^dt in DDD mode (2.1 to 5.4), but not in VVI mode (3.1 to 4.5).

The present study using Fourier analysis of LV volume curve suggests that DDD pacing is more desirable to support LV function during Ex than VVI mode.


We evaluated the mechanism of the inspiratory reduction of the left ventricular stroke volume (LVSV) in patients (pts) with impaired LV function. Eighteen pts with a variety of LV function underwent the respiratory and ECG gated radionuclide ventriculography and LV volume changes were measured during spontaneous respiration. LVSV decreased during inspiration in all the pts. In nine pts with a LVEF > 0.35, LV end-diastolic volume (EDV) significantly (p<0.05) decreased from expiration (159+28 ml) to inspiration (147+34 ml) without a significant change in LV end-systolic volume (ESV) (exp:82+24 ml, insp:82+29 ml). In contrast, in nine pts with a LVEF < 0.35, LVEDV was unchanged (exp:211+93 ml, insp:218+89 ml) and LVEBV significantly (p<0.001) increased from exp (152+72 ml) to insp (168+89 ml).

These results suggest that the inspiratory reduction of the LVSV is the result of two mechanisms: a decrease in preload and an increase in afterload of the left ventricle. Which mechanism predominantly operates is related to the functional state of the left ventricle.

The effect of left ventricular (LV) contraction on left atrial (LA) filling was studied in 17 normal subjects and 29 patients with heart disease (without regurgitation or shunt). LV and LA time activity curves of chamber counts (C) and these first derivatives (dc/dt) were obtained by using ECG-gated radionuclide angiography. LV peak ejection rate (LV=PER) and LA peak filling rate (LA-PFR) were calculated as follows:

LV=PER (1/sec)= (LV systolic maximal dc/dt)/end-diastolic LV-C; LA-PFR (1/sec)= (maximal dc/dt during LA filling phase)/maximal LA-C. The timing of LV=PER was nearly coincided with that of LA-PFR. LV maximal volume change in LV systole(LV max dv/dt) and LA maximal volume change in LA filling phase (LA max dv/dt) were calculated as follows:

LV max dv/dt(ml/sec) = (LV=PER) x LVEDV; LA max dv/dt/ml/sec = (LA-PFR) x LAV.

There was significant (p<0.001) correlation between LV max dv/dt, as the index of LV contraction, and LA max dv/dt as the index of LA filling (r=0.603).

Thus, these result suggest that LA filling is closely affected by LV contraction.