Analysis on Dynamic Pressure-Volume (P-V) - Relations for Reliable Prediction of Chamber Remodelling in Patients with Acute Myocardial Infarction (AMI)

WC Hu[1], CP Liu[2], LY Shyu[1], T Kao[3], JJ Wang[4]

[1]Chung Yuan Christian University, Chung LI, Taiwan, ROC
[2]Kaohsiung Veteran General Hospital , Kaohsiung, Taiwan, ROC
[3]National Yang-Ming University, Taipei, Taiwan, ROC
[4]I-Shou University, Kaohsiung, Taiwan, ROC

Abstract

A self-developed program that analyzed the load-independent LV pressure-volume (P-V) data from acute myocardial infarcted (AMI) patients to examine the process of chamber remodeling was reported. The extracted pressure-volume relationship at first week was compared to the 12th week data after PTCA procedure. The analysis program extracted static and dynamic parameters from ventricular pressure and volume acquired from catheter. The static parameters are stroke volume (SV), ejection fraction (EF), arterial elastance (Ea), the index of LV relaxation constant (τ, Tau) and many others. The dynamic parameters are the index of LV elasticity, the end systolic pressure volume relationship (ESPVR), and the constant for myocardial compliance, the end diastolic pressure volume relationship (EDPVR). At the 12th week after PTCA, the value of τ was significantly increased in comparison to the value at the 1st week. The index of LV compliance was significantly decreased at the 12th week after PTCA in comparison to the value at the 0th week. The index of arterial elasticity (Ea) was significantly decreased This result suggests that with the good recovery of myocardial compliance in relaxation phase and the fast release of LV pressure at the end of systole, the arterial impedance was adjusted significantly that prevents the development of chamber remodeling.

1. Introduction

Post AMI, chamber remodeling is an important mechanism to maintain adequate cardiac performance in the infarcted heart. At the infarcted area, the disadvantage of dilatation is the extra workload that will be imposing on normal myocardium and will be increasing wall tension. The mechanism of chamber remodeling is the overloaded wall stress that forces infarcted ventricle to dilate. The progressive changes in LV shape by infarcted area creates mechanical disadvantage to the heart that will be worsening LV pump function [1]. Although remodeling process involves complex molecular and cellular mechanisms [2], the remodeling process appears to be associated with the enlargement of end diastolic volume with a major contributor of LV pressure overload.

On a functional point of view, LV enlargement must occur in order to maintain or to restore forward output (Starling's Law). In fact, despite unchanged fiber shortening at infarcted area, an enlarged ventricle can eject a larger stroke volume. If the increase of wall tension and end diastolic volume is not adequate to maintain stroke volume, then a vicious progressive dilatation cycle will start. The process of chamber remodeling may lead to many complications that will result in subsequent poor prognosis.

The dynamic pressure-volume (P-V) relation can provide index that predicts the direction of chamber remodeling. For example, the report of rightward shifts in LV diastolic P-V relations that has induced the decrease of LV pumping function [3]. Many reports have concluded that the pumping functions are mediated by chamber remodeling but not by modifications in myocardial material properties [4, 5 & 6].

The increased end diastolic chamber volume contributes to the increases in systolic wall stress that will be leading to afterload mismatch and will be sustaining LV overload. The result of hemodynamic abnormality activates compensatory neurohormonal mechanisms that, over time, further contribute to disease progression.

Theoretically, ventricular load can be assessed by effective arterial elastance (Ea). Thus, the ventricle with less Ea can provide more effective performance. The change of Ea in the dynamic P-V relations is able to predict mechanical and structural relation of ventricle. For analyzing these data, we have developed a graphical

interface program to extract all possible parameters from ventricular pressure and volume data for the analysis. In this report, a reliable predictor, the effective arterial elastance (Ea), was analyzed and correlated to myocardial compliance and left ventricular pressure relaxation constant that the chamber remodeling can be practical indexed for long-term prognosis and can be used in pragmatic therapy for patient with AMI.

2. Methods

Twenty-one subjects aged 45-74 years were studied in the catheterization laboratories of the Veterans General Hospital, Kaohsiung, Taiwan. All subjects had AMI in the anterior or inferior wall with one-vessel coronary occlusion. The study protocol was approved by the Human Investigation Committee of the Veterans General Hospital, Kaohsiung. Informed consent was obtained from each patient. Patients were medicated with diazepam (10 mg p.o.). Introducer sheaths were placed in the right femoral artery and vein (8F and 9F, respectively). Left ventricular pressure-volume relations were simultaneously determined by conductance (volume) catheter (Webster Labbs, Baldwin Park, Calif.) attached to a stimulator/processore (Sigma-5, CardioDynamics, Rijnsberg, The Netherlands). The conductance catheter had a pigtail tip and was positioned over a guide wire to lie at the left ventricular apex. A 3F micromanometer catheter (SPC-330A, Millar Instruments) was then placed within the lumen of the conductance catheter and extended to its distal end for LV pressure acquisition. In this way, the LV pressure and volume were sampled with 200Hz using self-developed acquisition program.

A self-developed computer program that will analyze left ventricular pressure and volume relationship to extract parameters from load-independent ventricular pressure and volume data. The extracted parameters can be used to analyze and to predict chamber remodeling. The parameters extracted are including the static parameters such as stroke volume (SV), arterial elastance and many others as well as extracting dynamic parameters such as the index of LV elasticity from end systolic pressure volume relationship (ESPVR) and the index of LV pressure relaxation constant (τ, Tau) and the constant for myocardial compliance from the end diastolic pressure volume relationship (EDPVR). The extracted parameters are listed in Table 1. In this way, the status of left ventricle will be practically indexed.

A typical example of P-V data obtained from a subject (patient) was shown in Figures at Left. The static analysis of PV Loop data was shown in Figure 1. The dynamic analysis of PV loop data was show in Figure 2.

There are two graphical display of PV data. The bottom part of the figure was showing three line traces of ECG (labeled as ECG at right), Pressure (LVP) and

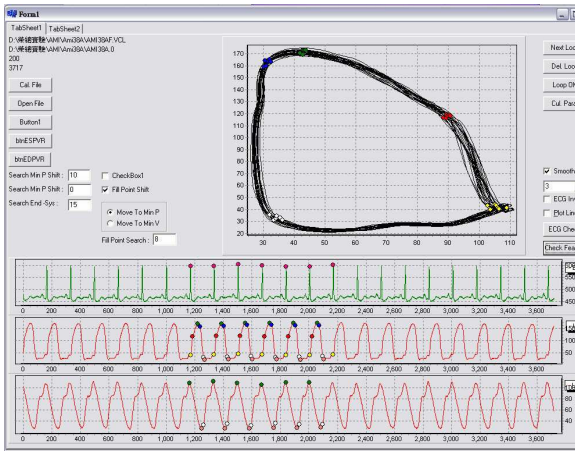

Figure 1. The self-developed program that be using the R-Peak of ECG as a marker to identify the timing of end diastole, ejection point, maximum LV pressure, end systole and beginning of LV filling as shown in the figure. The PV loop is showing at the upper right corner of the window. The three line graphs were showing the traces of ECG, LV pressure and LV chamber volume. The identified cardiac events were marked at the corresponding line traces as well as at the PV loop.

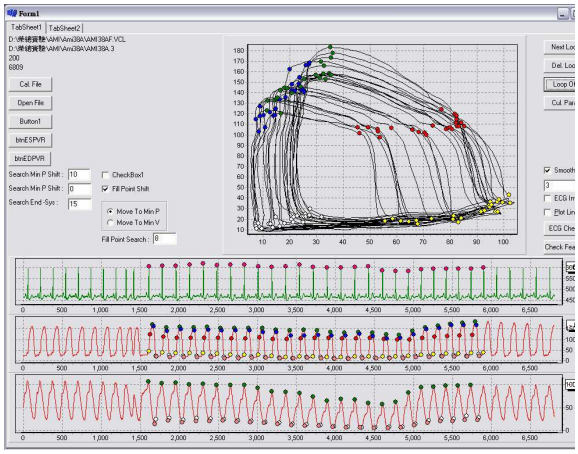

Figure 2. The beat by beat analysis of dynamic LV hemodynamics was showing in this figure. The inferior venus cava was blocked to reduce the venus return. As LV volume was reduced, the LV pressure was decreased. And, the PV loop was shifted left and downward. In this way, the dynamics of LV function such as ESPVR (the elasticity of LV, function of contraction) and EDPVR (the compliance of LV chamber) can be analyzed.

Volume (LVV). The axis represented sampling point (time). The Y-axis was representing the magnitude of electric potential, pressure in mmHg, volume in millimeter (ml).

The other graphical display in the given figure was showing the Pressure-Volume (P-V) loop. The X axis represents ventricular volume (in ml) and the Y-axis

Table 1. List of extracted parameters form left ventricular pressure and volume data.

Heart Rate (HR)	Minimum Volume	Minimum dV/dt	Filling Volume
Maximum Pressure	Maximum dP/dt	End-Diastolic Pressure	End-Systolic Pressure
Minimum Pressure	Minimum of dP/dt	End-Diatolic Volume	End-Systolic Volume
Maximum Volume	Maximum dV/dt	Filling Pressure	Ejection Pressure
Stroke Volume	Effective Ejection Fraction	Pressure at dp/dt Maximum	Volume at dp/dt Maximum
Stroke Work	Cardio Output (CO)	Pressure at dv/dt Maximum	Compliance, dV/dP
Theory Ejection Fraction	Compliance at the mid-filling phase, Comp.->mid.	Pressure relaxation constant Tau-ln (using logarithm calculation)	Effective Arterial Elastance, Ea
Ejection Volume	Compliance at the end-filling phase Comp.->ED	Correlation of Pressure relaxation constant (Tau-ln)	Correlation of Pressure relaxation constant (Tau-dp)
Time of end-diastolic	Time of end-systolic	Pressure relaxation constant Tau-dp (using dp for calculation)	

represents pressure (in mmHg). One P-V loop represents a cardiac beat. In each loop, upper left corner is end-systolic point and lower right corner is end-diastolic point. ESPVR is the regression line of the end-systolic points that is fitted by a linear regression. EDPVR is consisted of a set of end-diastolic data obtained from later third diastolic phase.

The functional assessments are including systolic property and diastolic property. The systolic properties are including the traditional ventricular indexes calculated from the steady-state ventricular P-V loop, such as (listed in Table 1) ejection fraction (EF), dp/dt_{max}, stroke work (SW), and cardiac output (CO). For the dynamic systolic parameters such as chamber elastance is the slope of end-systolic pressure-volume relation (E_{es});

$$Ees = P_{es} / V_{es} - V_0$$

where, Ees is chamber elastance obtained from the slope of ESPVR. P_{es} and V_{es} represent end-systolic pressure and volume respectively. V_0 is the intercepted volume data obtained from ESPVR when pressure is zero. The slope of stroke work to the end diastolic volume relation (M_{sw})

$$M_{sw} = SW / V_{ed}$$

where, SW represent the stroke work that is calculated from the area in each PV loop, V_{ed} is the end-diastolic volume in each cardiac beat.

As for the diastolic properties, at the early diastolic phase (Isovolumic relaxation), the relaxation time constant (Tau) is obtained by the method described by Glantz et al.[7]. At the mid-diastolic phase (the passive filling), the volume corrected ventricular peak filling rate (PFR/V_{ed}) was calculated. At the late-diastolic phase, the chamber compliance, (C_m) is calculated using linear regression from the slope of end-diastolic PV relation (EDPVR). The vascular load condition, the Effective arterial elastance (E_a), can be used to evaluate the status of system impedance to the work of ventricle. The E_a is defined as the slope of a line between end-systolic point and end-diastolic point.

$$E_a = (P_{es} - P_{ed})/SV$$

where, P_{es} is the end systolic pressure; P_{ed} is the end diastolic pressure and SV is the stroke volume. The slope is equal to $(P_{es} - P_{ed})$/stroke volume, where pressure is the product of the flow and the resistance. During one cardiac cycle, flow will be offset by stroke volume. Thus, this slope should be correlated to vascular resistance.

3. Results

In present study, the load-independent LV pressure-volume (P-V) data at the 1st week and at the 12th weeks from twenty-one AMI patients after PTCA procedure were analyzed to access the effects of early reperfusion on LV function. In result, as shown in Figure 3, the value of τ was significantly improved at the 12th week after PTCA in comparison to the value at the 1st week. The index of LV compliance was decreased at the 12th week after PTCA in comparison to the value at the 1st week, however, as shown in Figure 4, the improvement was not significant. The index of arterial elastance was decreased as LV end diastolic volume increased at the 1st week. The normalized percentage change of difference that was accessing LV chamber remodeling was shown in Figure 5. Thus, the improvement of good relaxation in LV pressure release and unload the myocardial stress will prevent chamber remodeling. Early reperfusion can prevent left ventricular (LV) to develop chamber remodeling, reduce infarction zone, salvage myocardial injury and improve clinical outcomes.

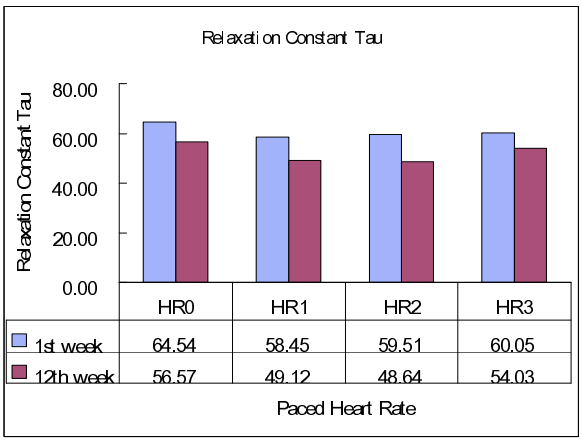

Figure 3. The comparison of relaxation constant over 4 different paced heart rate.

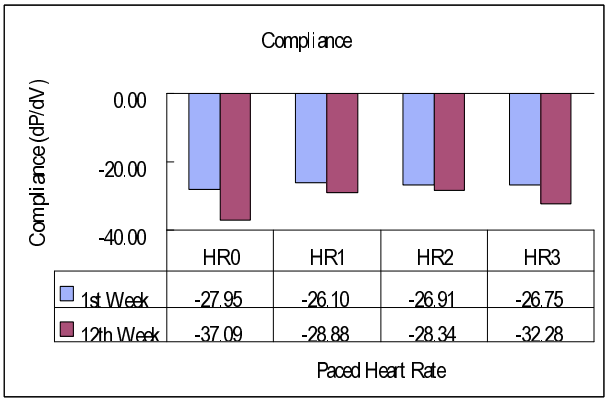

Figure 4. The comparison of compliance over 4 different paced heart rate.

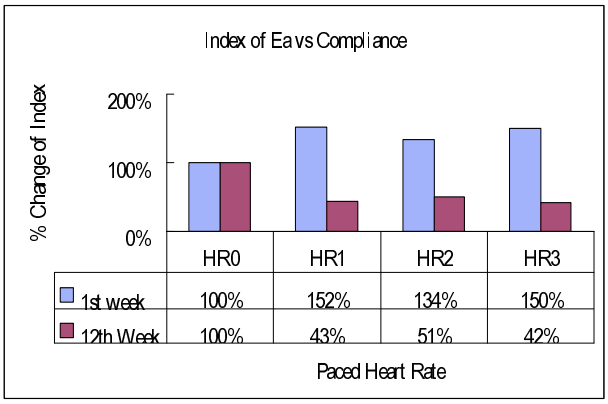

Figure 5 Index of normalized percent change of Ea with compliance over 4 different pacing heart rate. At 12th week, the index of Ea with the compliance did not change as increased heart rate. This indicates that the remodeling of LV chamber was corrected.

4. Discussion and conclusions

The proposed method provides several unique advantages: 1) in human studies, the functional assessments are less load-dependent; 2) it can provide the dynamic mechanical and structural relation of ventricle; and 3) the method will simultaneously assess ventricular systolic and diastolic function within one cardiac cycle. These advantages allow a simple and repeatable measurement to predict complicates mechanical/structural changes in patient with AMI. This prediction can offer a practical therapeutic guide and a long-term prognosis.

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Address for correspondence

Weichih Hu
Dept. of Biomedical Engineering,
Chung Yuan Christian University,
Chung Li, Taiwan, ROC
weichih@be.cycu.edu.tw