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Early detection of abnormal left ventricular relaxation in acute myocardial ischemia with a quadratic model

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ABSTRACT

Aims: The time constant of left ventricular (LV) relaxation derived from a monoexponential model is widely used as an index of LV relaxation rate, although this model does not reflect the non-uniformity of ventricular relaxation. This study investigates whether the relaxation curve can be better fitted with a "quadratic" model than with the "conventional" monoexponential model and if changes in the LV relaxation waveform due to acute myocardial ischemia could be better detected with the quadratic model.
Methods and results: Isovolumic relaxation was assessed with quadratic and conventional models during acute myocardial ischemia performed in 6 anesthetized pigs. Mathematical development indicates that one parameter (Tq) of the quadratic model reflects the rate of LV relaxation, while the second parameter (K) modifies the shape of the relaxation curve. Analysis of experimental data obtained in anesthetized pigs showed that the shape of LV relaxation consistently deviates from the conventional monoexponential decay. During the early phase of acute myocardial ischemia, the rate and non-uniformity of LV relaxation, assessed with the quadratic function, were significantly enhanced. Tq increased by 16% ($p < 0.001$) and K increased by 12% ($p < 0.001$) within 30 and 60 min, respectively, after left anterior descending (LAD) coronary artery occlusion. However, no significant changes were observed with the conventional monoexponential decay within 60 min of ischemia.
Conclusions: The quadratic model better fits LV isovolumic relaxation than the monoexponential model and can detect early changes in relaxation due to acute myocardial ischemia that are not detectable with conventional methods.

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1. Introduction

During acute myocardial ischemia (AMI), left ventricular (LV) relaxation remains incompletely understood, while it is a major determinant in diastolic dysfunction [1]. 'Relaxation' relates to the process where cardiac muscle returns, after contraction, to its initial length or tension [2]. The fall of LV pressure (P) is the in vivo manifestation of isometric relaxation and is the direct expression of cardiac muscle inactivation [3,4]. The time constant of LV relaxation derived from a monoexponential model is usually used as an index for evaluating LV relaxation rate in both experimental and clinical studies [3,5].

It is well established that LV relaxation is non-uniform and that there is a significant interaction between non-uniformity and loading conditions [6–8]. In acute LV ischemia, the loading conditions influence both the regional response to myocardial ischemia and the mechanical consequences of the interaction between the ischemic zone and non-ischemic areas [6,9,10]. Since LV diastolic dysfunction may precede systolic dysfunction during AMI, early detection of abnormal LV relaxation may be useful in clinical practice [11].

LV relaxation is usually assessed by a monoexponential model of LV P fall in time [5]. However, this model cannot capture the non-uniformity of ventricular relaxation and cannot discriminate early from late relaxation [5,8]. Moreover, deviation of relaxation behavior from this monoexponential decay model is well

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established in a number of clinically important disease states, such as regional ischemia associated with segmental coronary disease, hypertrophic cardiomyopathy and heart failure [8,12–14]. Hence, a better model than the monoexponential model is required.

We investigated whether LV relaxation can be better assessed with a quadratic function, based on a logistic equation, during AMI [8,15]. We used such a quadratic function to assess non-uniformity of LV relaxation in phase plane analysis. We examined the parameters of the quadratic function under control conditions and during experimental situations of AMI.

2. Materials and methods

All experimental procedures and protocols in this investigation were reviewed and approved by the Ethics Committee of the Medical Faculty of the University of Liege. All procedures conformed to the Guiding Principles in the Care and Use of Animals of the American Physiological Society and were performed according to the Guide for the Care and Use of Laboratory Animals (NIH publication no. 85-23, revised 1996).

Experiments were performed on 6 healthy pure pietran pigs of either sex (20–28 kg). The animals were premedicated with intramuscular administration of ketamine (20 mg/kg) and diazepam (1 mg/kg). Anesthesia was then induced and maintained by a continuous infusion of sufentanil (0.5 μg/kg/h) and sodium pentobarbital (3 mg/kg). Spontaneous movements were prevented by pancuronium bromide (0.1 mg/kg). After endotracheal intubation through a cervical tracheostomy, the pigs were connected to a volume cycled ventilator (Evita 2, Dräger, Lübeck, Germany) set to deliver a tidal volume of 10 mL/kg at a respiratory rate of 20/min. End-tidal PCO_2 measurements (Capnomac, Datex, Helsinki, Finland) were used to monitor the adequacy of ventilation. Respiratory settings were adjusted to maintain end tidal CO_2 in the range of 35–40 Torr (4.67–5.33 kPa). Arterial oxygen saturation was closely monitored and maintained above 95% by adjusting the FiO_2 as necessary. Central temperature was measured with a rectal probe and maintained at 37 °C by means of a heating blanket. A standard lead electrocardiogram was used for the monitoring of heart rate (HR).

The chest was entered through median sternotomy, the pericardium was incised and sutured to the chest wall to form a cradle for the heart, and the root of the aorta was dissected clear of adherent fat and connective tissue. A combined conductance-micromanometer catheter (CD Leycom, Zoetermeer, The Netherlands) was inserted through the right carotid artery and advanced into the left ventricle. Right atrial *P* was measured with a micromanometer-tipped catheter inserted into the cavity through the superior vena cava.

Thrombus formation along the catheters was prevented by administration of 100 U/kg of heparin sodium intravenously just before the insertion.

3. Experimental protocol

To provide similar states of vascular filling, the animals were continuously infused with Ringer lactate (5 mL/kg/h) and, when necessary, with hydroxyethylstarch 6% to increase central venous pressure up to 6–7 mmHg over 30 min.

LV volume and *P* baseline measurements were recorded. All measurements were taken immediately after the animal was briefly disconnected from the ventilator to sustain end-expiration. Thereafter, the left anterior descending (LAD) coronary artery was ligated after the origin of the first diagonal artery. In all animals, measurements were obtained at baseline (T0) and each 30 min during 120 min (T30, T60, T90, T120) after LAD occlusion.

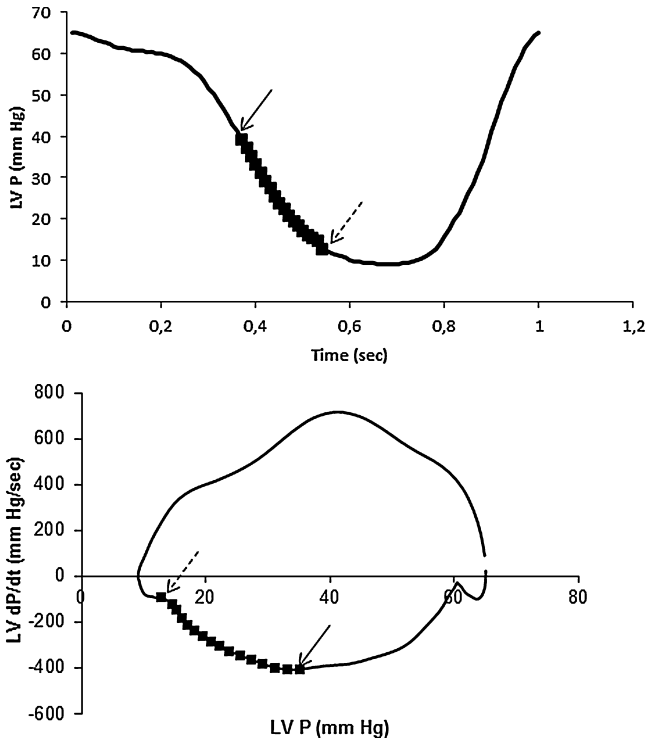

Fig. 1. Time course of LV pressure and corresponding phase plane plot (at baseline). The isovolumic relaxation period corresponds to the period between the time point of peak negative dP/dt (dP/dt_{min}) (solid arrow) and the time at which dP/dt reached 10% of the dP/dt_{min} value (dashed arrow).

4. Data analysis

All measurements were performed at end-expiration. The conductance catheter was connected to a Sigma-5 signal conditioner processor (CD Leycom, Zoetermeer, The Netherlands). All analog signals and the ventricular *P*-volume loops were displayed on screen for continuous monitoring. The analog signals were continuously converted to digital form with appropriate software (Codas, DataQ Instruments Inc, Akron, OH) at a sampling frequency of 200 Hz.

5. Mathematical analysis

First derivative of LV *P* (dP/dt) was plotted against LV *P* to generate phase plane loops [16]. Indeed, information about diastolic function that cannot be discerned from the usual *P* vs. time display format is easily visualized and quantitated using the phase plane plot. Phase plane analysis is carried out on graphs of precisely periodic or nearly periodic functions plotted such that the function is the abscissa and its time derivative is the ordinate [5,16]. The isovolumic relaxation period was defined as the period between the time point of peak negative dP/dt (dP/dt_{min}) and the time at which dP/dt reached 10% of the dP/dt_{min} value (Fig. 1).

The quadratic model for LV $P(t)$ during isovolumic relaxation was based on the logistic function and defined [15]:

$$\frac{P}{P^{\circ}} = \frac{e^{-t/Tq}}{1 + K \cdot P^{\circ} \cdot (1 - e^{-t/Tq})} \quad (1)$$

where P° is the initial *P* at the start of the relaxation, *K* is a constant and *Tq* is the time constant of the exponent. It was assumed here that there was no non-zero asymptote. This assumption seems to hold from previously published results on the logistic model [7,16].

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