Prognostic Value of Cardiodynamic Indexes with Respect to the Development of Heart Failure during Massive Pulmonary Embolism

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The cardiodynamics of the right ventricle was studied during the first 30 min of massive pulmonary embolism complicated and uncomplicated by heart failure. Both variants were accompanied by diastolic dysfunction of the right ventricle. Some changes in the cardiodynamics observed during complicated massive pulmonary embolism indicated a lower increase in the contractile activity, decreased myocardial contractility, and more pronounced dilation of the right ventricle. The absolute indexes of mechanical activity including indexes of contractility are not reliable criteria for early diagnostics of heart failure in the acute stage of massive pulmonary embolism, whereas changes in these indexes hold much prognostic value.

Key Words: *experimental massive pulmonary embolism; cardiodynamics; heart failure*

Our previous experiments showed that the first 30 min after a sharp increase in right ventricular (RV) afterload during massive pulmonary embolism (MPE) is a critical period for the development of heart failure [11]. Therefore, it was interesting to compare cardiodynamic indexes during MPE complicated and uncomplicated by heart failure.

Here we studied cardiodynamic indexes of RV during the initial stages of complicated and uncomplicated MPE and evaluated their prognostic significance with respect to the development of heart failure.

MATERIALS AND METHODS

Experiments were performed on 52 mongrel dogs weighing 15-20 kg without thoracotomy (natural respiration). Premedication was performed with promedol (10 mg/kg intramuscularly). Anesthesia was produced by fractional intravenous administration of 20 mg/kg sodium thiopental.

Group 1 included 20 dogs with MPE without heart failure (uncomplicated heart failure). Group 2 included 12 dogs with MPE complicated by heart failure, which developed during the first 30 min and led to lethal outcome. Group 3 served as the control (20 dogs without MPE). Groups 1 and 3 animals were euthanized by intravenous administration of sodium thiopental 6.5 h after heart catheterization.

MPE was modeled as described elsewhere [2]. Catheterization was conducted through peripheral vessels. Blood pressure in the aorta, right atrium, and cardiac ventricles was recorded by external Pressure Transducers 746 connected to Pressure Amplifiers 863. First derivative of intraventricular pressure was plotted using a Contractility 868 electron differentiator. ECG was recorded by standard limb leads using an ECG Amplifier 850. Multichannel recording was performed on a Mingograf-82 device (Siemens-Elema).

Cardiodynamic indexes were determined polycardiographically by recording simultaneously the above parameters. Curves of RV pressure and its first derivative were analyzed by the following parameters: end-diastolic pressure, P_0, PVP, PVP_t, ^+DP, ^-DP, ^+DP_t, ^-DP_t, $^-DP_t{-}0_t$, and $^+DP{-}DP_t$ (Fig. 1). From these parameters, the following indexes were calculated and used for the analysis of systolic and diastolic functions of the heart [1,3,4,9,15]: $^+DP/^-DP$, ^+DV ($^+DP/^+DP_t$),

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$^{-}$DV ($^{-}$DP/$^{-}$DP$_t$), $^{+}$DV/$^{-}$DV, ($^{+}$DP+$^{-}$DP)/$^{+}$DP^{-}DP$_t$, $^{+}$DP/P$_0$, $^{+}$DP/PVP$_t$, PVP/$^{+}$DP, PVP/$^{+}$DP$_t$, PVP/PVP$_t$, and PVP×PVP$_t$. The analysis of the cardiac cycle structure included estimation of the durations of isometric contraction, tension, and ejection periods, and mechanical and total systoles (Fig. 1). The duration of the cardiac cycle was determined by R-R intervals.

The data were analyzed by Student's *t* test.

RESULTS

Mean arterial pressure and heart rate (HR) did not differ form the initial levels during the first 30 min of uncomplicated (136±7 mm Hg and 186±8/min, respectively) and complicated (134±11 mm Hg and 181±10/min, respectively) MPE. Mean right atrial pressure and RV end-diastolic pressure did not differ during complicated and uncomplicated MPE and insignificantly surpassed the control levels.

Uncomplicated and complicated MPE were accompanied by a sharp increase in RV maximum systolic pressure (PVP) to 70±4 and 78±4 mm Hg, respectively (p>0.1). A comparable rise in blood pressure indicated similar embolic damages during complicated and uncomplicated MPE, which was confirmed by pathoanatomical studies. The data suggest that changes in studied indexes during MPE does not depend on the degree of damage.

Other amplitude indexes of RV pressure curve and its first derivative (P$_0$, $^{+}$DP, and $^{-}$DP) also markedly increased in dogs with complicated and uncomplicated MPE. The rise of RV maximum pressure was accompanied by a delay of its attainment (PVP$_t$). $^{-}$DP$_t$ in the diastolic portion of the *dP/dt* curve also increased. Interval indexes in the systolic portion of the *dP/dt* curve were unchanged during uncomplicated and complicated MPE.

In both uncomplicated and complicated MPE, $^{+}$DP^{-}DP decreased, $^{+}$DV, $^{-}$DV, ($^{+}$DP+$^{-}$DP)/$^{+}$DP^{-}DP$_t$, PVP/$^{+}$DP$_t$, PVP/PVP$_t$, and PVP*PVP$_t$ increased, and $^{+}$DV/$^{-}$DV, $^{+}$DP/P$_0$, and PVP/$^{+}$DP remained unchanged. The $^{+}$DP/PVP$_t$ ratio increased during uncomplicated MPE and did not differ from the control during complicated MPE.

The duration and structure of the cardiac cycle did not change over the first 30 min of MPE.

Changes in RV cardiodynamic indexes during complicated and uncomplicated MPE were similarly directed but differed in the magnitude (Fig. 2). The increase in $^{+}$DP, $^{+}$DV, and $^{+}$DP/PVP$_t$ was less pronounced during complicated MPE, while the decrease in $^{+}$DP/$^{-}$DP was more pronounced than during uncomplicated MPE. The increase in diastolic indexes of RV performance ($^{-}$DP and $^{-}$DV) during complicated MPE was much greater than during uncomplicated MPE. $^{+}$DP/P$_0$ tended to decrease to 81.8±8.5% during complicated MPE and was practically unchanged during uncomplicated MPE. These data are consistent with the results of clinical studies showing that RV $^{+}$DP/P$_0$ (Veraguth—Krienbule index) decreases only in patients with MPE accompanied by heart failure [10].

The *dP/dt* curve is directly related to the rate of the increase in the strength of contractions, and inversely depends on changes in RV size [8]. Therefore, a minor rise in $^{+}$DP and related indexes during complicated MPE is probably due to a less pronounced increase in the contractile activity or marked dilation of RV typical of acute pulmonary heart caused by pulmonary embolism [5,10,14]. Considerable changes in diastolic $^{-}$DP and $^{-}$DV indexes during complicated MPE can also be associated with RV dilation.

Thus, diastolic dysfunction of RV develops at the early stages of complicated and uncomplicated MPE, which is confirmed by lengthening of the $^{-}$DP$_t$ interval. Complicated MPE is accompanied by cardiodynamic changes, which indicate a less pronounced increase in the contractile activity, reduced myocardial contractility, and marked dilation of RV. According to current views, indexes of the central hemodynamics and pump functions of the heart, RV pressure curves and its first derivative, and other parameters calculated from the above indexes reflect cardiac mechanical ac-

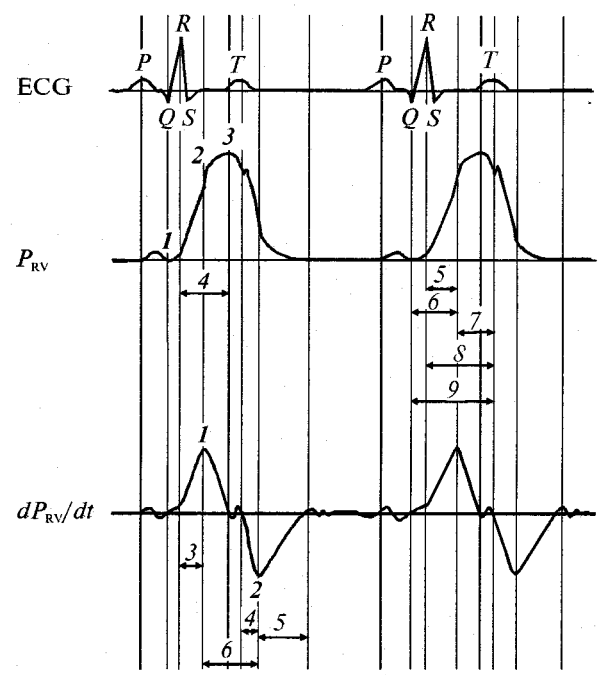

Fig. 1. Right ventricular pressure (P_{RV}) curve and its first derivative (dP_{RV}/dt), and phases and periods of cardiac cycle. P_{RV} curve: end-diastolic pressure (*1*), P$_0$ (*2*), PVP (*3*), PVP$_t$ (*4*), isometric contraction (*5*), tension (*6*), ejection (*7*), mechanical (*8*) and total systoles (*9*). dP_{RV}/dt curve: $^{+}$DP (*1*), $^{-}$DP (*2*), $^{+}$DP$_t$ (*3*), $^{-}$DP$_t$ (*4*), $^{-}$DP$_t$$^{-}0_t$ (*5*), and $^{+}$DP^{-}DP$_t$ (*6*).

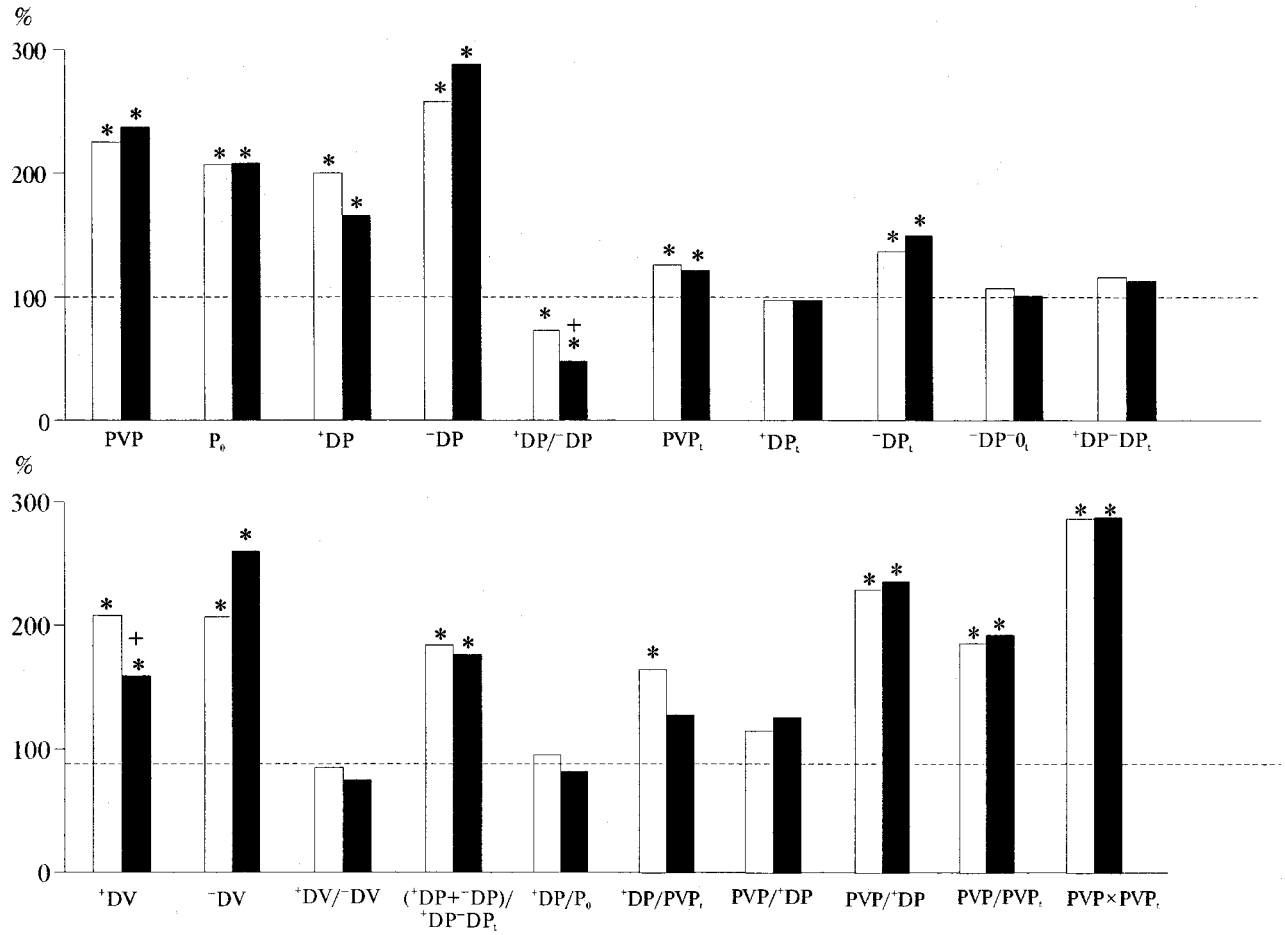

Fig. 2. Cardiodynamic indexes of the right ventricle (% of the initial values presented by dashed lines) in the initial stages of massive pulmonary embolism uncomplicated (light bars) or complicated (dark bars) by heart failure. $p<0.05$: *compared with the initial level and +compared with uncomplicated form.

tivity under certain hemodynamic and regulatory conditions rather than myocardial contractility [3,4, 6,8,9,15] and, therefore, characterize cardiac performance but not the state of cardiac muscle. Therefore, the analysis of the state of cardiac muscle, in particular its contractility, should consider complex hemodynamics, cardiodynamics, intramyocardial processes, and regulatory effects. Our previous studies of intramyocardial and neurohumoral processes during the initial stages of complicated and uncomplicated MPE showed that complicated MPE causes structural and metabolic changes impairing cardiac muscle contractility [7,12,13]. Sympathectomy, which increases the importance of the humoral control, promotes these effects. Therefore, dogs with MPE complicated by heart failure have decreased myocardial contractility.

Our experiments showed that complicated and uncomplicated MPE only little changed cardiodynamic indexes. The absolute values of studied parameters did not differ in the initial stages of complicated and uncomplicated MPE. Thus, cardiodynamic indexes recorded during the initial period of MPE, as well as various indexes of mechanical activity, are unable to predict the development of heart failure even 5-10 min before its appearance. This conclusion agrees with previously reported data [3,6,9] that indexes of mechanical activity, including the so-called indexes of contractility, are not reliable criteria for early diagnostics of cardiac insufficiency.

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