Myocardial Shortening Velocity as an Index for the Assessment of Myocardial Contractility

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SUMMARY

The myocardial shortening velocity of 20 patients was recorded by inserting a pair of prongs with strain gauges through a cardiac catheter into the right ventricular muscle. The patients were divided into 3 groups; Group 1: 5 normal controls, Group 2: 9 patients with heart diseases but without heart failure, Group 3: 6 patients with physical and hemodynamic findings of right heart failure. All the patients of Group 3 showed lower maximal myocardial shortening velocity, higher right ventricular end-diastolic pressure and higher tension-time index compared to those of Group 1 patients. The values of Group 2 scattered between those of Groups 1 and 2. Myocardial shortening velocity thus measured is not completely free from the effects of preload and afterload, but can easily be obtained also by other methods. The applicability of the shortening velocity for the assessment of myocardial contractility was discussed.

Additional Indexing Words:
Heart failure Preload and afterload Hypertrophy Tension-time index Max V\text{CE} Strain gauge

The precise assessment of myocardial contractility in clinical cases is of great importance to the cardiologist, and recently the contractile state of the myocardium for the intact heart has been shown to be described in terms of instantaneous relations between force and velocity.\textsuperscript{1)} The present study was undertaken to evaluate the myocardial contractility of clinical cases by recording myocardial shortening velocity with a direct method.

METHODS

Studies were performed on a total of 20 patients. The majority of the patients had organic heart diseases which affected the right ventricular function, and ranged in age from 22 to 63 years as shown in Table I. Some subjects and some were not receiving digitalis at the time of this study. The patients were not anesthetized but were premedicated with sedatives (pethidine hydrochloride 35–70 mg, intra-
muscularly or diazepam 5 mg orally). Right heart catheterization was performed on all subjects. Pressures were recorded by means of Statham P 23 Db strain gauge pressure transducer.

Furthermore, right ventricular myocardial contraction was recorded by inserting a pair of prongs equipped with strain gauges into the subendocardial muscles through a catheter (Fig. 1). The prongs were inserted into the subendocardial muscle fibers of the free wall near the apex of the right ventricle.²,³ The myocardial contraction detected by the strain gauges attached to the base of the prongs was amplified electrically and was recorded oscillographically at the paper speed of 100 mm/sec. Shortening velocity was obtained by means of a differentiator circuit (Fig. 2). Myocardial contraction was expressed as per cent shortening; i.e. the ratio of myocardial contraction to end-diastolic fiber length. Maximal myocardial shortening velocity was expressed as below:

\[
\text{maximal shortening rate per an amplitude of muscular contraction} = \frac{\text{maximal slope of myocardial contraction wave}}{\text{total amplitude of myocardial contraction wave}} \ (\text{sec}^{-1})
\]

As mentioned earlier, the myocardial contraction of the free wall of the right ventricle near the apex was recorded, where the muscle fibers are presumed to run in a circular fashion.⁴ However, still the myocardial contraction recorded may apparently vary according to the variation of the direction of the prongs inserted. Therefore, maximal shortening rate per an amplitude of the muscular contraction
was obtained to cancel out the effect.

Tension-time index (TTI) for the right ventricle was calculated as follows:\[ \text{TTI} = \text{mean systolic pressure of the right ventricle} \times \text{right ventricular ejection time (mmHg} \times \text{sec)} \].

All measurements were done during end-expirium so as to exclude respiratory fluctuations.

The patients were divided into 3 groups based on the clinical symptoms and hemodynamic findings.

- **Group 1.** includes 5 patients with atypical chest pain or palpitation but without any abnormal physical, laboratory or hemodynamic findings.
- **Group 2.** includes 9 patients who had heart diseases which affect the right ventricular functions but without clinical findings of right heart failure.
- **Group 3.** includes 6 patients who had heart diseases and who had the clinical findings of right heart failure (edema, hepatomegaly more than 2 finger-breadths and venous congestion), and elevated right ventricular end-diastolic pressure (>7 mmHg).

**RESULTS**

The data obtained are summarized in Table I. In Group 1, right atrial pressure ranged 2 to 5 (average 3.2) mmHg, right ventricular end-diastolic pressure 2.5 to 6 (4.5) mmHg, pulmonary arterial mean pressure 10 to 14.5 (12.9) mmHg, tension-time index 3.4 to 4.4 (3.9) mmHg sec, and maximal myocardial shortening velocity ranged 12.0 to 14.7 (13.5) sec⁻¹. In Group 2, right atrial pressure ranged –3 to 7 (average 2.9) mmHg, right ventricular end-
diastolic pressure -3 to 8 (3.9) mmHg, pulmonary arterial mean pressure 8 to 38 (21.6) mmHg, tension-time index 2.2 to 11.0 (6.5) mmHg·sec, and maximal shortening velocity ranged 6.1 to 13.9 (9.6) sec⁻¹. In Group 3, right atrial pressure ranged 5 to 18 (average 11.3) mmHg, right ventricular end-diastolic pressure 7.5 to 20 (13.0) mmHg, pulmonary arterial mean pressure 30 to 55 (39.9) mmHg, tension-time index 7.8 to 14.8 (11.1) mmHg·sec, and maximal myocardial shortening velocity ranged 5.9 to 9.4 (7.2) sec⁻¹.

The significant findings of the data are illustrated in Fig. 3. All the
patients of Group 3 had higher ventricular end-diastolic pressure, larger tension-time index, and lower maximal myocardial shortening velocity than those of Group 1 patients. In Group 2, these values scattered between those of 2 other groups.

The relationship between maximal myocardial shortening velocity and ventricular end-diastolic pressure is shown in Fig. 4. In 3 patients, who had marked elevation of end-diastolic pressure (>15 mmHg), maximal shortening velocity was fairly low. For the rest of the patients, no definite relation
Fig. 3. Right ventricular end-diastolic pressure, tension-time index and maximal myocardial shortening velocity of 3 groups of the patients.

Fig. 4. The relationship between end-diastolic pressure and maximal myocardial shortening velocity (symbols, same as in Fig. 3).
was revealed between these parameters.

Fig. 5 illustrates the relationship between maximal myocardial shortening velocity and pulmonary arterial mean pressure. A rough inverse relationship exists between these 2 values.

DISCUSSION

As shown in Fig. 3, in any patient of Group 3 tension-time index of the right ventricle was greater than that of Group 1 patients. In other word, patients of Group 3 were performing more work compared to Group 1 or 2 patients. However, the extra stress can be compensated by the increase of the cardiac muscle mass—ventricular hypertrophy. In fact, 6/6 of Group 3 patients and 5/9 of Group 2 patients showed roentgenographic and/or electrocardiographic findings of right ventricular hypertrophy. On the other hand, maximal myocardial shortening velocity of this study separated out the 2 extreme groups completely; all of the normal patients (Group 1) showed higher values compared to the patients with clinical signs and abnormal hemodynamic measurements to show ventricular failure. Because the shortening velocity is unaffected with the effects of ventricular hypertrophy and shortening velocity can well represent the contractile state of the heart,6 it is presumed from these data that the heart with increased stress on the ventricle has de-
creased myocardial contractile state even though the heart is performing greater work.

It is well known that ventricular pump action is determined by 3 major components: preload, afterload and myocardial contractility. Based on the experiments on isolated cardiac muscles and experimental animals, Sonnenblick and his associates presented that max $V_{CE}$ (maximal shortening velocity of the contractile element at zero load) could be the best index to represent myocardial contractility unaffected by the effects of preload and afterload.¹ Hugenholz et al⁷ calculated max $V_{CE}$ from the pressure and dp/dt tracings. However, this technique requires a strict isovolumetric portion of systole and is not applicable in the presence of valvular regurgitation, shunt or asynergic contraction.⁸ Moreover, 3 component model should be used rather than 2 component model to exclude the effect of preload⁹ and in this situation the extrapolation of the force-velocity curve to zero-load is fairly difficult to determine.¹⁰

Thus, in this study the shortening velocity of cardiac fibers was measured directly by inserting needles through a cardiac catheter. Although the myocardial contraction of a small portion of the ventricle can be recorded with this method, we have shown that total ventricular performance can be well detected with this method.¹¹ On the other hand, the myocardial shortening velocity measured with this method is not the shortening velocity of the contractile element ($V_{CE}$) but that of the circumferential fibers ($V_{CF}$, $V_{CF} = V_{CE} + V_{SE}$, $V_{SE}$: velocity of series elastic component).¹¹ Therefore, logically maximal myocardial shortening velocity obtained in this study is affected by the effects of preload and afterload.

However, as shown in Fig. 4, the patients who had marked elevation of end-diastolic pressure showed low shortening velocity and for the rest of the patients there were no definite relations between end-diastolic pressure and shortening velocity. In the depressed heart, the ventricular function curve becomes flat in its configuration.¹² For this reason, in the depressed heart the increase of end-diastolic volume or pressure might not affect the cardiac contractile force so much as demonstrated in the isolated cardiac muscle or experimental animals. Moreover, end-diastolic pressure does not necessarily have a direct relation to end-diastolic volume because of the different compliance in the heart with various diseases.¹³ On the other hand, there was a rough inverse relation between the pulmonary arterial mean pressure and maximal shortening velocity. Also in other study, we have shown that the change in afterload affects myocardial contraction greatly, using the same method as in this study.¹¹

In the present study, myocardial contraction was measured on the right
ventricle of the patients because of technical feasibility. However, usually the assessment of the contractility of the left ventricle is of major importance for the cardiologist. Fortunately, as far as the left ventricle is concerned, usually the afterload does not vary so much except several situations; aortic stenosis, hypertension and cardiogenic shock. In taking all these situations into consideration, circumferential shortening velocity which can be measured by thermodilution method, cineangiographic method, echocardiographic technique and so on, can be one of the indexes to represent myocardial contractile state. And combined understanding of several informations on the heart including shortening velocity, intraventricular pressures and volumes will offer clinically satisfactory evaluation of the myocardial contractility of the heart, even though the trial to obtain a single ideal index to represent myocardial contractility completely free from the effects of loads should be continued.

References