The Improvement of Systolic Function of Depressed Left Ventricle by External Vibration at Diastole

YOSHIRO KOIWA, TAKEHIKO TAKAGI, JUN-ICHI KIKUCHI, HIDEYUKI HONDA, NOBUO HOSHI and TAMOTSU TAKISHIMA

The First Department of Internal Medicine, Tohoku University School of Medicine, Sendai 980

KOIWA, Y., TAKAGI, T., KIKUCHI, J., HONDA, H., HOSHI, N. and TAKISHIMA, T. The Improvement of Systolic Function of Depressed Left Ventricle by External Vibration at Diastole. Tohoku J. Exp. Med., 1989, 159 (2), 169-170 —— The left ventricular (LV) incomplete relaxation (IR) has been reported to play an important role in the pathophysiology of the congestive heart failure such as causing higher diastolic pressure and/or impeding the coronary perfusion during diastole. Therefore, using open chest canine preparations (n = 4), we examined 1) whether the minute external vibration during diastole could release IR and 2) what occurred to LV systolic function in this perturbation. LV failure with IR was induced by propranolol administration and, if necessary, by rapid atrial pacing up to 180 beats/min. When we applied a 50 Hz, sinusoidal vibration of 2.1 mm magnitude during diastole to the epicardium of LV with complete relaxation, there was no significant change in the ventricular function. However, the systolic functional improvement (3.8±1.1 mmHg elevation in LV systolic pressure) was observed when the vibration was applied to LV with IR. We concluded that external vibration at diastole could release IR and would be useful to improve the systolic function of the depressed heart with IR. incomplete relaxation ; external minute vibration ; functional improvement of depressed LV

Despite that the precise mechanism of incomplete relaxation (IR) has not been elucidated completely, its important role in the congestive heart failure, ischemic heart disease and hypertrophic cardiomyopathy has been stressed (Brutsaert et al. 1980). That is, IR induces higher left ventricular (LV) diastolic pressure and impedes coronary perfusion during diastole. When external vibration is applied to LV, the perturbation has been reported to detach the force generating myocardial crossbridge (Koiwa et al. 1989a, b). Therefore, if the underlying mechanism of IR is incompleteness of crossbridge detachment, the external vibration might release IR in the depressed heart. We examined this idea that external vibration could release IR. Moreover, we also examined what occurred to LV systolic function by this external vibration. We used four adult mongrel dogs (13-17.5 kg), anesthetized with sodium pentobarbital (25 mg/kg) and maintained with artificial ventilation. The chest was opened and a pericardial cradle was constructed. A flat, disc-shaped tip (1 cm diameter) of a vibrator (Emic 511B ; Shinn Nippon Sokki, Tokyo) was attached.

Received September 4, 1989; revision accepted for publication September 30, 1989.
Mailing address : Y. Koiwa, M.D., the First Department of Internal Medicine, Tohoku University School of Medicine, 1-1 Seiryo-machi, Aoba-ku 980, Sendai, Japan.
perpendicularly to the LV lateral epicardial surface. We sutured silver electrodes for atrial pacing. LV pressure and aortic flow were measured with a catheter-tip micromanometer and an electromagnetic flowmeter. In the control condition, a 50 Hz, 2.1 mm-magnitude sinusoidal vibration of 40-200 msec duration was applied during diastole of succeeding 10 beats (diastolic vibration input). Then, we injected 0.05-0.25 mg/kg of propranolol HCl intravenously to induce IR of LV beats. If the IR has not been observed in the steady contraction at 15 min after bolus injection, pacing rate was increased until IR became manifested. Here, we regarded LV relaxation was complete when the pressure remained constant and flat at diastole. But when the succeeding beat starts on the declining portion of the ventricular pressure and diastolic pressure became higher compared to those with complete relaxation, we regarded IR occurred. When IR was observed by this procedure, the diastolic vibration input was repeated for 10 beats and compared the change of the LV function between before and during the vibration. External vibration resulted no significant change in LV function (LV pressure and SV) in control beat with complete relaxation. However, the slight decrease in diastolic pressure (approx. 1.1±0.7 mmHg) and improvement of LV systolic functions, e.g., peak LV pressure increased by 3.8±1.1 mmHg, were observed by diastolic vibration input to the beat with IR (Fig. 1).

We concluded that the external vibration at diastole could release IR and improve the systolic function of the depressed heart. This improvement might be induced by an increase in the LV diastolic compliance, increase in LV diastolic volume and finally through the Frank Starling’s mechanism.

References


Fig. 1. Improvement of left ventricular (LV) systolic function by diastolic vibration input. In these beats, functional depression and incomplete relaxation was induced by propranolol 0.05 mg/kg administration (pacing rate 115 bpm). LV systolic pressure became higher by application of diastolic vibration (the beat with closed circle). Moreover, this figure showed the decrease in LV diastolic pressure by applying external vibration. We regarded, observing this phenomenon, that the incomplete relaxation could be released by this vibration input.