The Response of Normal and Failing Heart to Externally Applied Vibration in the Canine Open Chest Preparation

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KOIWA, Y., HOSHI, N., OHYAMA, T., TAKAGI, T., KIKUCHI, J., HONDA, H. and TAKISHIMA, T. The Response of Normal and Failing Heart to Externally Applied Vibration in the Canine Open Chest Preparation. Tohoku J. Exp. Med., 1989, 157 (2), 183-184 — We examined the left ventricular functional response to externally applied vibration using four canine open chest preparations. A sinusoidal 30 Hz vibration (2.7 mm in amplitude) was applied to the ventricular epicardium at each level of propranolol-induced myocardial depression. External vibration in control conditions induced no significant change either in peak left ventricular pressure (LVP) or in stroke volume (SV). With propranolol, 0.1 and 0.3 mg kg, peak LVP and SV were depressed by the application of external vibration, even though there was no significant change of these values in the nonvibrating condition compared to control. We conclude that the ventricular response to vibration depends on the underlying myocardial viability.

The inhibitory effect of external vibration on muscle contraction has been reported using isolated skeletal muscle, vascular smooth muscle, papillary muscle, and the isolated working rabbit heart (Vukas et al. 1978). However, it has not been shown whether this depression also exists in the intact ejecting heart preparation and whether myocardial viability modifies the response to external vibration. To answer these questions, we examined four adult mongrel dogs (14-18 kg) anesthetized with sodium pentobarbital (25 mg/kg) and maintained with artificial ventilation. The chest was opened and a pericardial cradle was constructed. We attached a flat, disc-shaped tip of a vibrator (1 cm diameter, Emic 511B, Shin Nippon Sokki, Tokyo) to the left ventricular (LV) lateral epicardial surface to apply the sinusoidal vibration. A miniature vibration sensor (1 g mass, Emic 540M, Shin Nippon Sokki) was attached to the vibrator shaft to monitor the input vibration. LV pressure, aortic flow, and aortic pressure were measured with a catheter-tip micromanometer (PC481, Millar Inst. Inc., Houston, TX, USA), an electromagnetic flow meter (MF46, Nihon Kohden, Tokyo), and a fluid filled manometer, respectively.

In the control condition, we applied a 30 Hz vibration of 2.7 mm magnitude for 30 sec. Then we injected 0.1 mg/kg of propranolol HCl intravenously. After 15 min of injection,
the vibration was applied again as before. We continued to administer propranolol HCl to further depress myocardial contractility (cumulative dose ≈ 0.3 mg/kg) and the vibration was repeated.

In control, no change in LV pressure was induced by external vibration (top of the figure). However, at 0.1 mg/kg and 0.27 mg/kg of propranolol, peak LV pressure was depressed following the onset of vibration and recovered immediately to the quiescent, i.e., without vibration, level by stopping the vibration (middle and bottom of the figure). The vibration induced depression was larger at 0.27 mg/kg than at 0.1 mg/kg propranolol. The LV pressure in the quiescent condition with propranolol up to 0.3 mg/kg did not differ from that in control. However, with a higher concentration of propranolol 0.5 mg/kg, the peak LVP did decrease in the quiescent condition (not shown in the figure). On the other hand, the magnitude of the depression caused by vibration at this high level of propranolol did not differ from that of a propranolol dose of 0.27 mg/kg. The vibration induced change in stroke volume showed a similar trend as those in peak LV pressure.

It was clear that vibration induced depression (VID) did not occur in the open chest intact and ejecting heart preparation at the control condition. However, VID did occur following myocardial depression by propranolol. This VID was much more sensitive when the depression was compensated and latent. Considering following characteristics of VID that 1) it recovered within one beat in a stepwise fashion, 2) only systolic pressure was depressed, 3) no significant change in HR, LVEDP and systemic vascular resistance occurred, we speculate that externally applied vibration might affect the contractile machinery by causing detachments of force-generating cross bridges in the presence of propranolol. Furthermore, the VID reported in previous studies should be reconsidered taking into account the underlying physiological condition of the preparation, since the depression seen in the failing state might be the expression of a direct vibration effect on the contractile element.

Reference