

Myocardial Tactile Stiffness: A Variable of Regional Myocardial Function

KAGAMI MIYAJI, MD, SEIRYO SUGIURA, MD, SADA O OMATA, PhD,*
YUKIHIRO KANEKO, MD, TOSHIYA OHTSUKA, MD, SHINICHI TAKAMOTO, MD

Tokyo and Fukushima, Japan

Objectives. We developed a new sensor system for in situ measurement of myocardial tactile stiffness—stiffness in a direction perpendicular to the wall—and validated its use for providing a reasonable estimation of regional myocardial function.

Background. Numerous attempts have been made to directly assess regional myocardial function. The complexity and highly invasive nature of the measuring devices have hampered their in situ application.

Methods. In open chest mongrel dogs, myocardial tactile stiffness, ventricular pressure and ventricular volume were monitored. Under the preload reduction, these variables were measured to determine the relation between the end-systolic pressure–volume relation (ESPVR) and the end-systolic tactile stiffness–volume relation (ESSVR). The changes in myocardial tactile stiffness were monitored in the regional ischemic myocardial model and infarcted model to evaluate their usefulness as indexes of regional myocardial function.

Results. Myocardial tactile stiffness changed cyclically and followed a time course similar to left ventricular pressure. When preload was altered, the ESSVR was as linear as the ESPVR. The slope of the ESSVR and that of the ESPVR showed a strong correlation over a wide range of contractility. These results suggest that myocardial tactile stiffness can be a good index of regional wall stress or fiber stress. End-systolic myocardial tactile stiffness of ischemic and infarcted regions decreased significantly, with a concomitant increase in end-diastolic stiffness compared with that of intact myocardium.

Conclusions. Using our tactile sensor system, regional myocardial tactile stiffness of a beating heart was measured with reasonable temporal resolution. We consider myocardial tactile stiffness to be a useful index of regional myocardial function.

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Evaluation of regional myocardial contractile function is of clinical importance because various disease states affect the myocardium heterogeneously. Many investigators have attempted to quantify the regional wall strain–stress relation either by measuring it directly or by calculating it based on a model (1–6). However, clinical application of these methods is very limited, mainly because of the difficulties involved in direct measurement of wall stress. An alternative approach is to measure the stiffness of the myocardium (7–12). In 1987, Halperin et al. (10,11) reported that transverse stiffness, which is the ratio of indentation stress to strain as the ventricular wall is indented in the direction perpendicular to the wall, is linearly related to in-plane wall stress. Although their finding strongly supported the idea that myocardial transverse stiffness can be used to quantify accurately the regional contractile state, their experimental apparatus was too complex for in situ application. For accurate measurement of myocardial transverse

stiffness in situ, we have developed a new tactile sensor system based on a device we had introduced previously to thoracoscopic surgery for detecting small and invisible pulmonary nodules (12). In this study, the stiffness measured by our sensor system is defined as “tactile stiffness.” The results obtained from experiments with dogs show that this system can assess regional myocardial function accurately and has potential for clinical use.

Methods

Tactile sensor system. The principle of our tactile sensor has been described previously (13,14). Briefly, each material has its own resonance frequency and when the material vibrating in this frequency touches an object, a shift of the resonance frequency is observed. The difference between the frequencies under nontouching and touching conditions depends on the stiffness or hardness of the object. On the basis of this principle, when a tactile sensor vibrating in its own frequency touches an object, a shift in resonance frequency can be observed. Because this shift in frequency depends on the stiffness of the object, we can estimate the stiffness by monitoring the shift in frequency.

Our tactile sensor system is composed of a sensor probe, an amplifier and a filter. We modified the sensor in the following

From the Department of Cardiothoracic Surgery and Second Department of Internal Medicine, Faculty of Medicine, University of Tokyo, Tokyo; and *College of Engineering, Nihon University, Kohriyama, Fukushima, Japan.

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Address for correspondence: Dr. Kagami Miyaji, Department of Cardiothoracic Surgery, Faculty of Medicine, University of Tokyo, Hongo 7-3-1 Bunkyo-ku, 113 Tokyo, Japan.

Abbreviations and Acronyms

EDTS	= end-diastolic tactile stiffness
E _{max}	= slope of end-systolic pressure-volume relation
ESPVR	= end-systolic pressure-volume relation
ESSVR	= end-systolic tactile stiffness-volume relation
ESTS	= end-systolic tactile stiffness
P-V loop	= pressure-volume loop
PVA	= pressure-volume area
S _{max}	= the slope of end-systolic tactile stiffness-volume relation
S-V loop	= stiffness-volume loop
σ _f	= muscle fiber stress

manner for measuring myocardial tactile stiffness. The sensor probe is 5.5 cm long, 7 mm in diameter, weighs 2.08 g and is equipped with a small tip, 3 mm in diameter, that is connected acoustically to a piezoelectric transducer made of lead zirconate-barium titanate ceramic with a resonance frequency

Figure 1. Tactile sensor structure. The sensor is 5.5 cm long, 7 mm in diameter, weighs 2.08 g and is equipped with a small tip 3 mm in diameter connected to a piezoelectric transducer made of lead zirconate-barium titanate ceramic. The contact surface of the sensor is made of hard epoxy resin and forms a smooth curved surface to ensure that the contact area between the sensor and the myocardium is constant. Tactile sensor system: A/D converter = analog to digital converter; D1 = first diagonal branch; D2 = second diagonal branch; LAA = left atrial appendage; LAD = left anterior descending branch; LV = left ventricle; PA = pulmonary artery; RV = right ventricle.

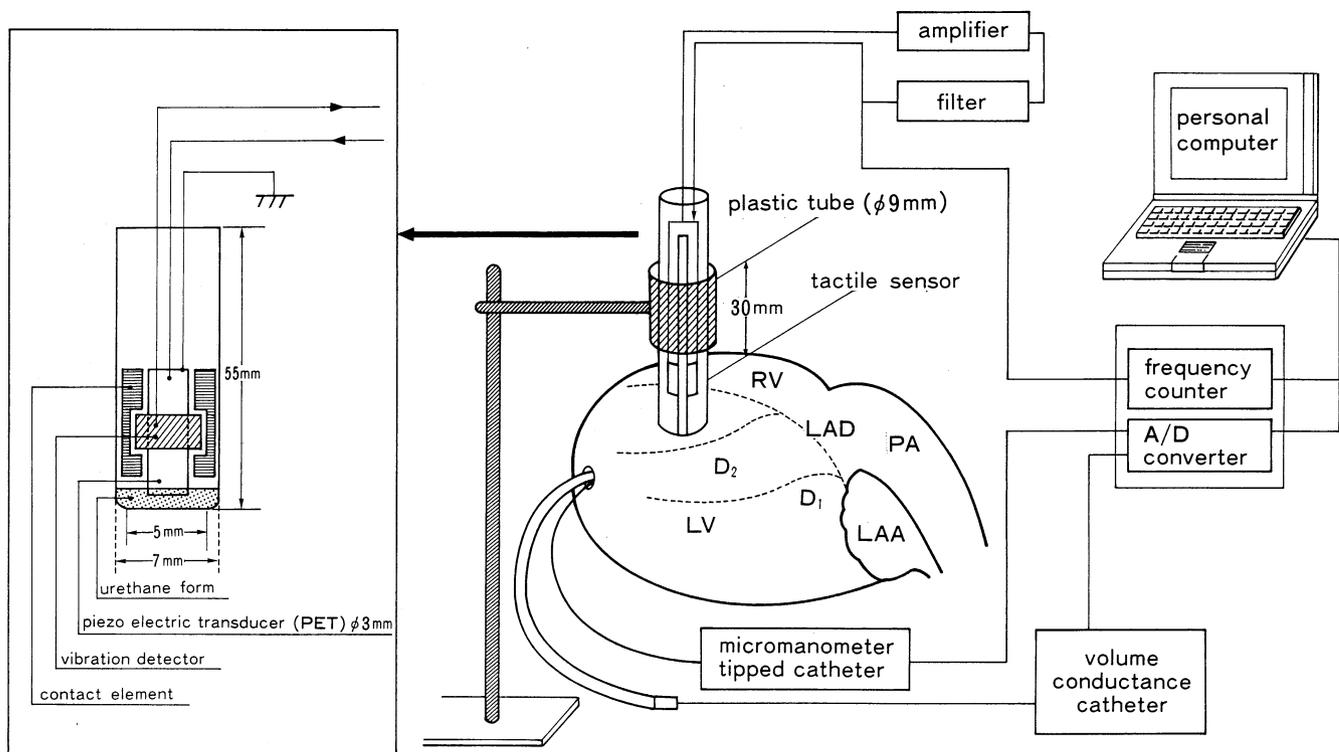
of 68 kHz. The contact surface of the sensor is made of hard epoxy resin and forms a smooth curved surface (Fig. 1).

When the sensor probe touches an object and the resonance frequency shifts, the vibration detector picks up the change in frequency and sends a signal to the amplifier, which keeps the piezoelectric transducer vibrating at the new frequency.

Measurement was made 150 times/s with a frequency counter device (AX-CNT1001, Axiom Co. Ltd, Koriyama, Japan) and the delta f value was processed sequentially by a personal computer (PC9821-Ne, NEC Inc, Tokyo, Japan) (Fig. 1).

Measurement and calibration of stiffness. The sensor probe was inserted into a plastic tube, 9 mm in diameter and 30 mm long, which was fixed to a stand. The position of the tube was adjusted so that the probe was able to slide up and down smoothly within the tube, to follow the beating myocardium under a constant load (2.08 g). The relation between stiffness and delta f for our tactile sensor was derived by the counterbalance method (13,14) employing bovine gelatin, the stiffness of which was predetermined by a viscoelastance measurement device (AX-SFD001, Axiom Co. Ltd). Based on these measurements, we obtained the following calibration formula, which was used in this study (Fig. 2): stiffness (g/mm²) = Exp [(delta f (Hz) + 517.876)/302.069].

Study protocol. All the animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institute of Health (NIH publication 85-23, revised 1985). Sixteen mongrel dogs



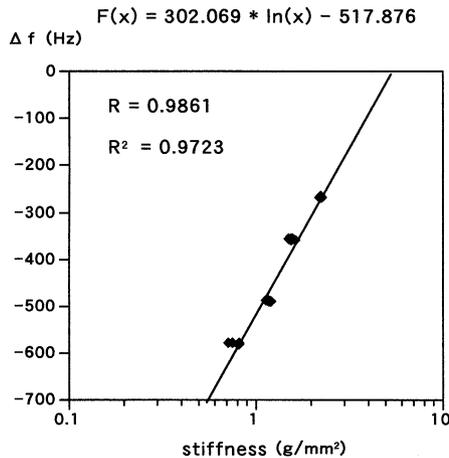


Figure 2. Strong correlation between stiffness and shift in frequency (Δf) was obtained using our tactile sensor ($R = 0.9861$, $R^2 = 0.9724$). The plots represent the stiffness and shift in frequency derived from gelatin made from bovine skin.

weighing 12 to 18 kg were anesthetized with ketamine (15 mg/kg body weight intramuscularly) and sodium pentobarbital (35 mg/kg intravenously). The animals were then mechanically ventilated through an endotracheal tube. After left thoracotomy, pericardiotomy was performed. A 7F volume conductance catheter (single field, eight electrodes, Leycom, Oegstgeest, Netherlands) and a 4F micromanometer-tipped catheter (MPC-500, Millar Instruments) were inserted into the left ventricle from its apex in order to record the left ventricular volume and pressure, respectively. In all animals the tactile sensor for the measurement was placed in the region between the left anterior descending artery and the second diagonal branch. All data were collected simultaneously by an analogue to digital converter (AX-ADC1001, Axiom Co., Ltd) and a personal computer.

Myocardial tactile stiffness during the cardiac cycle. In 16 mongrel dogs, myocardial tactile stiffness and left ventricular volume and pressure were measured to study phasic changes in these variables during the cardiac cycle under baseline conditions.

Preload alteration run. In 10 mongrel dogs, a 14F Fogarty occlusion catheter (Model 6208014F, maximum balloon inflation cavity 10 ml, Baxter Healthcare Corp.) was advanced to the inferior vena cava via the femoral vein. During preload reduction by inferior vena cava occlusion, left ventricular pressure and volume were measured to determine the end-systolic pressure-volume relation (ESPVR) and its slope (E_{max}). Simultaneously, the relation between tactile stiffness and volume was studied.

Similar measurements were made while varying the contractile state by drug administration. 1) In five dogs contractility was depressed by propranolol administration up to a total dose of 1.5 mg/kg in 0.25 mg/kg steps (0.25, 0.5, 0.75, 1.0 and 1.5 mg/kg) and preload reduction was performed at each dose. 2) In another five dogs dobutamine was administered intravenously at doses of 1.0, 3.0 and 5.0 $\mu\text{g}/\text{kg}/\text{min}$ at 10-min

intervals. One hour after the end of dobutamine infusion, propranolol was given at doses of 0.25, 0.5, 0.75 and 1.0 mg/kg at 20-min intervals. At each dose of the two drugs, all the parameters were measured during preload reduction.

Myocardial tactile stiffness during acute ischemia. In six dogs, the left anterior descending artery was dissected. After measuring the baseline myocardial stiffness, the artery was clamped proximal to the second diagonal branch to induce regional ischemia. Care was taken not to induce global left ventricular dysfunction with this procedure. The change in myocardial stiffness under acute ischemia was monitored for 30 s, and the result was compared with baseline tactile stiffness at both end-systole and end-diastole.

Tactile stiffness of infarcted myocardium. In these six dogs, after measurement of myocardial tactile stiffness during acute ischemia, the dissected left anterior descending artery was ligated just proximal to its second diagonal branch. The distal end of the left anterior descending artery and the second diagonal branch were also ligated to prevent coronary perfusion due to collateral flow from the circumflex artery. Both the pericardiotomy and the left thoracotomy were then closed. Two weeks after ligation of the left anterior descending artery, all the animals were anesthetized with ketamine (15 mg/kg intramuscularly) and sodium pentobarbital (35 mg/kg intravenously) and then mechanically ventilated through an endotracheal tube. After left thoracotomy, pericardiotomy was performed. The tactile sensor was placed in the ischemic region between the left anterior descending artery and the second diagonal branch and the intact region between the left anterior descending artery and its first diagonal branch. The tactile stiffness of the ischemic region was monitored and compared with that of the intact region at both end-systole and end-diastole.

Histopathologic examination. After measurement of tactile stiffness, the left ventricle was excised and subjected to histopathologic examination. All specimens were fixed in 10% formaldehyde solution, embedded in paraffin, sectioned and then stained with hematoxylin-eosin, and Mallory's azan to examine the myocardial infarction in the ischemic region.

Statistical analysis. All results are reported as the mean value \pm SD. The two-tailed paired Student *t* test was used for the comparison under acute ischemia. Differences were regarded as statistically significant at $p < 0.05$. The two-tailed Student *t* test was used for comparison between intact and infarcted myocardium. Differences were regarded as statistically significant at $p < 0.05$.

Results

Myocardial tactile stiffness during the cardiac cycle. Phasic changes in myocardial tactile stiffness and left ventricular pressure are shown in Figure 3A. The tactile stiffness followed a similar time course to left ventricular pressure, indicating a close relation with wall stress. In Figure 3B, myocardial tactile stiffness is plotted against left ventricular volume. The stiffness-volume (S - V) loop was very similar to the pressure-volume

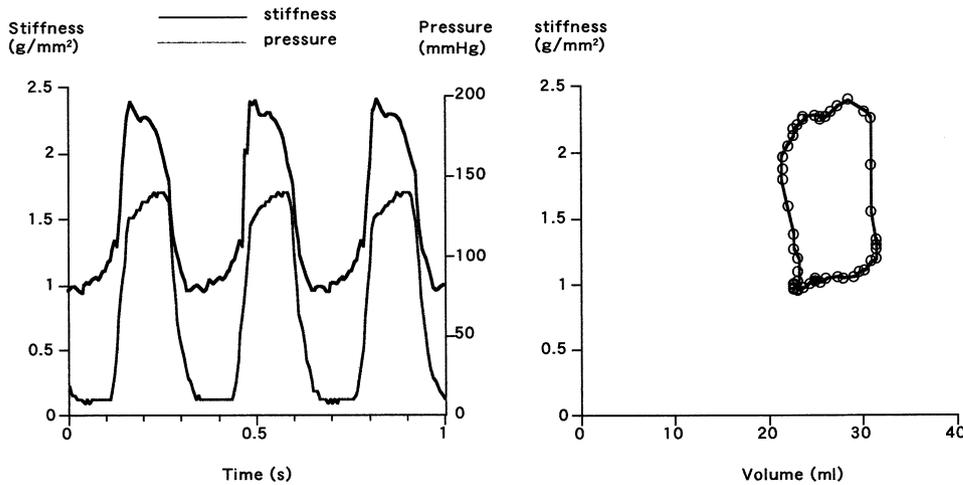


Figure 3. Left panel, Change in myocardial tactile stiffness and left ventricular pressure wave. Right panel, Relation between myocardial tactile stiffness and left ventricular volume in a cardiac cycle (S-V loop).

(P-V) loop. Baseline end-systolic tactile stiffness (ESTS) (upper left corner of the loop) was $2.52 \pm 0.38 \text{ g/mm}^2$ ($n = 16$) and end-diastolic tactile stiffness (EDTS) (lower right corner of the loop) was $1.20 \pm 0.22 \text{ g/mm}^2$ ($n = 16$).

Preload alteration run. When the preload was reduced by inferior vena cava occlusion, tactile stiffness decreased (Fig. 4A). This suggests that the stiffness we measured was dependent on the preload. Furthermore, the S-V loop shifted to the left and downward during preload reduction, with a highly linear end-systolic tactile stiffness-volume relation (ESSVR) ($r = 0.975$) (Fig. 4B). From the analogy with ESPVR we determined the slope of ESSVR, and defined it as S_{max} ($0.069 \text{ g/mm}^2/\text{ml}$ in this case). The changes in ESSVR under different contractile states are shown in Figure 5. The ESSVR shifted to the right and downward as the dose of propranolol increased.

In the intact ventricle, wall stress is homogeneous and regional elastance or myocardial fiber stress is related to the total ventricular elastance. Accordingly, we compared the ESSVR (and its slope S_{max}) as a measure of regional elastance with the ESPVR (and its slope E_{max}) as a measure of

ventricular contractility. The relation between E_{max} and S_{max} in one dog (Fig. 6A) shows a highly linear correlation ($r = 0.968$). Similarly, a good correlation was observed in each of the dogs given dobutamine alone or with propranolol (Table 1). Figure 6B shows pooled data from all 10 dogs. The correlation coefficient was significant ($r = 0.890, p < 0.0001$).

Myocardial tactile stiffness during acute ischemia. During acute ischemia, the P-V loop shifted slightly to the right, but the left ventricular pressure did not change (Fig. 7B). The pressure-volume area (PVA), which represents the total mechanical energy generated by a left ventricular contraction, also did not change. These data indicate that the global ventricular function remained at a similar status. However, myocardial tactile stiffness (Fig. 7A) showed a remarkable change. A few seconds after clamping of the left anterior descending artery, the ESTS started to decrease and the EDTS started to increase. About 15 to 20 s after clamping, the myocardial tactile stiffness stabilized.

The ESTS before myocardial ischemia was $2.74 \pm 0.49 \text{ g/mm}^2$ ($n = 6$), and this decreased significantly to $2.18 \pm 0.46 \text{ g/mm}^2$ ($n = 6, p < 0.01$). The EDTS showed a signifi-

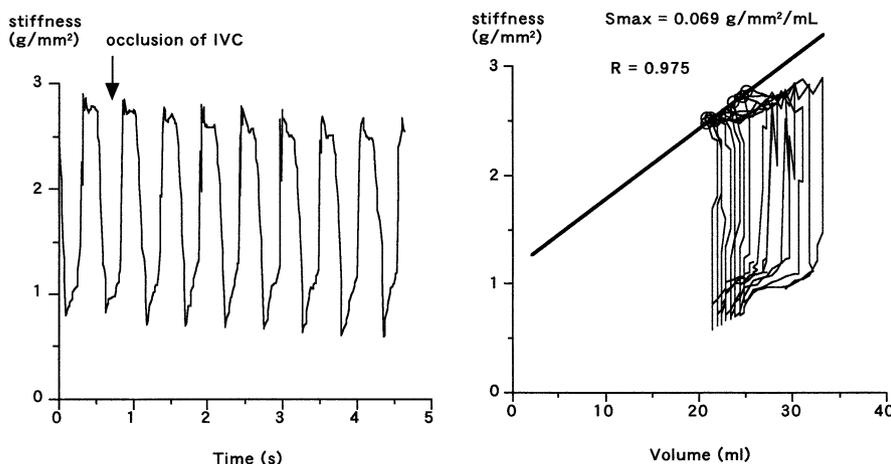


Figure 4. Left panel, Change in myocardial tactile stiffness after occlusion of the inferior vena cava (IVC). Right panel, Change in stiffness-volume relation (S-V loop) after occlusion of the inferior vena cava. The change in the ESSVR was linear ($r = 0.975$).

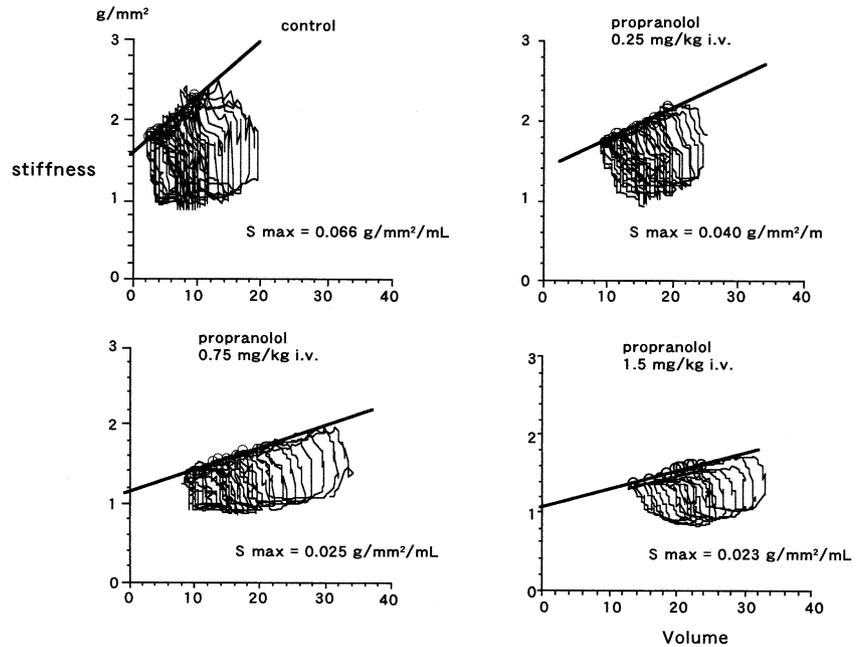


Figure 5. Changes in Smax during administration of propranolol. As the dose of propranolol increased, Smax decreased. i.v. = intravenously.

cant increase after ischemia ($1.03 \pm 0.23 \text{ g/mm}^2$ to $1.28 \pm 0.26 \text{ g/mm}^2$ [$n = 6, p < 0.01$]).

Tactile stiffness of infarcted myocardium. Phasic changes in the myocardial tactile stiffness of the ischemic and intact regions in Dog 1 are shown in Figure 8A and B, respectively. It is clear that the ESTS of the infarcted myocardium was less than that of the intact myocardium, whereas the EDTS of the former was greater than that of the latter. The ESTS of both the infarcted and intact myocardium in six animals is shown in Table 2. The ESTS of the infarcted region was $2.02 \pm 0.31 \text{ g/mm}^2$ ($n = 6$), whereas that of the intact region was $2.62 \pm 0.36 \text{ g/mm}^2$ ($n = 6$). The end-systolic myocardial stiffness of the infarcted region was decreased significantly in comparison with the intact region ($p < 0.05$). The EDTS of both the infarcted and intact myocardium in six animals is also shown in Table 2. The EDTS of the infarcted region was $1.46 \pm 0.21 \text{ g/mm}^2$ ($n = 6$), whereas that of the intact region was $1.18 \pm 0.18 \text{ g/mm}^2$ ($n = 6$). The end-diastolic myocardial

stiffness of the infarcted region was increased significantly in comparison with the intact region ($p < 0.05$).

Histopathologic examination. In all six dogs, transmural myocardial infarction was seen in the ischemic region between the left anterior descending coronary artery and its second diagonal branch. The region between the left anterior descending artery and its first diagonal branch also represented intact myocardium. Widespread coagulation necrosis accompanied by hemorrhage was seen in the ischemic region. Histopathologic study revealed that the ischemic region in all six dogs included myocardial infarction.

Discussion

Indexes of regional myocardial function. Because of its clinical and basic importance, many attempts have been made to estimate regional myocardial function accurately. Indexes derived so far include regional myocardial shortening (15-21),

Figure 6. Left panel, Relation between Smax and Emax in one dog (no. 7) given both propranolol and dobutamine. A strong correlation between Smax and Emax was obtained. Right panel, Overall relation between Smax and Emax in all 10 dogs. A strong correlation between Smax and Emax was obtained ($r = 0.890, p < 0.0001$).

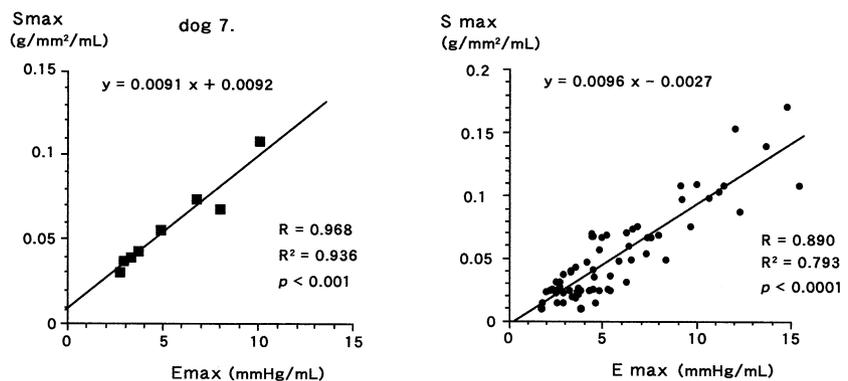


Table 1. Relation Between the Slopes of the End-Systolic Tactile Stiffness–Volume Relation and the End-Systolic Pressure–Volume Relation

Dog No./Drug Given	Correlation Coefficient (r)	Regression Equation
1/P	0.968	$y = 0.0081x + 0.0075$
2/P	0.985	$y = 0.0090x - 0.013$
3/P	0.992	$y = 0.0061x - 0.0015$
4/P	0.986	$y = 0.011x - 0.013$
5/P	0.973	$y = 0.0086x - 0.023$
6/D+P	0.955	$y = 0.0085x - 0.0014$
7/D+P	0.968	$y = 0.0091x + 0.0092$
8/D+P	0.983	$y = 0.012x - 0.0038$
9/D+P	0.936	$y = 0.0106x - 0.012$
10/D+P	0.930	$y = 0.0074x + 0.030$

D = dobutamine; P = propranolol.

regional myocardial work (15,17,22–25) and the length–thickness (17,18) or pressure–thickness relation (21,27,28) at end-systole. Also, direct measurement of ventricular wall stress has been attempted for accurate evaluation of regional myocardial function (1–6). However, the measurement systems and devices have been so complicated that their clinical application has been impractical.

Transverse stiffness as a measure of wall stress. Since Suga et al. (29) proposed the concept of the time-varying elastance model, which regards the ventricular wall as an elastic body that changes its elastance during the cardiac cycle, the use of end-systolic myocardial stiffness has been suggested as a good index of myocardial contractility (7–11). Sideris et al. (30) measured the hardness of the contracting myocardium using a calibrated spring and reported that the heart became harder during systole than during diastole, and that hardness was correlated with left ventricular pressure. However, they were unable to show clearly whether hardness was a property of the muscle per se or simply reflected a change in intracavity pressure. In 1987, Halperin et al. (10) defined transverse stiffness as the ratio of indentation stress to strain when the ventricular wall was indented in a direction perpendicular to the wall. They showed that the transverse stiffness was propor-

tional to the stresses in the plane of the wall using arterially perfused canine ventricular septa mounted in an apparatus that could exert biaxial load in the plane of the wall. Moreover, when similar wall stresses were applied, the transverse stiffness of the contracting ventricular septum was greater than that of the relaxed one. On the basis of these findings, they concluded that measurement of transverse stiffness might allow accurate quantification of the regional contractile state. Again, the experimental setting was far from in situ application.

Tactile sensor system. Since Omata and Terahuma (13) developed an original design for a tactile sensor in 1989, its applications have been studied in various fields, including medical science (14,31–34). In 1994 we applied this tactile sensor to thoracoscopic surgery for detection of small and invisible pulmonary nodules (12). Although we found the sensor to be very useful for this purpose, it was not readily applicable to the beating heart because of its slow dynamic response and its inability to provide an absolute value of stiffness. Therefore, we developed a new tactile sensor system that was fast enough to follow the heart beat and also provided an absolute value of stiffness. Using this sensor system, we have succeeded in measuring myocardial stiffness in situ. The stiffness followed a similar time course to left ventricular pressure.

Nature of tactile stiffness. As discussed earlier, tactile stiffness reflects well the contractile state of the myocardium. However, its precise nature should be discussed. In our study, the S–V loop shifted to the left and downward during preload reduction by inferior vena cava occlusion. This suggests that the stiffness we measured was dependent on the preload. Because the ESSVR was found to be linear, we defined the slope of the ESSVR as S_{max} ($g/mm^2/ml$), as was ESPVR defined as E_{max} . S_{max} varies according to the ventricular contractile state. It increased under administration of positive inotropic agents and decreased under administration of a beta-adrenergic blocking agent. Therefore, the correlation between S_{max} and E_{max} was studied in order to clarify the relation between the regional myocardial stiffness measured by the tactile sensor (tactile stiffness) and regional elastance in the time-varying elastance model based on the assumption that the intact ventricle is a homogeneous elastic body. The correlation

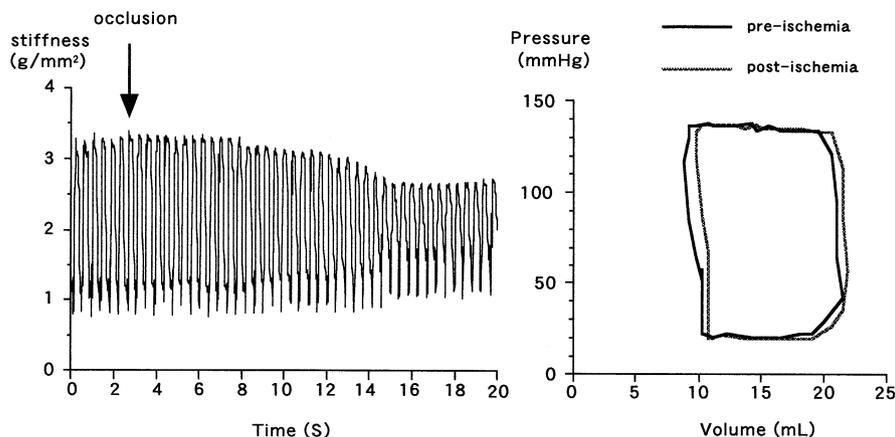
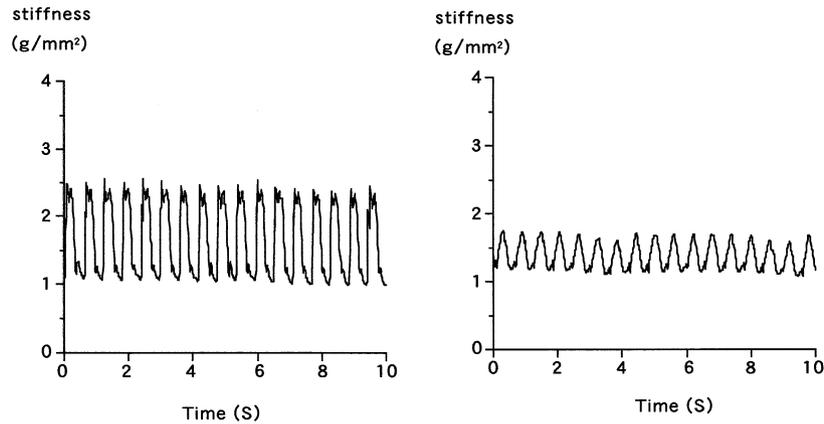


Figure 7. Left panel, Change in myocardial tactile stiffness in an acute ischemic heart model by occlusion of the distal left anterior descending coronary artery. Right panel, The P–V loop shifted to the right after myocardial ischemia. However, left ventricular pressure and the PVA did not change.

Figure 8. Left panel, Phasic changes in myocardial tactile stiffness of the intact regions in dog 1. **Right panel,** Stiffness of the infarcted myocardium in dog 1, revealing that the ESTS of the infarcted myocardium was less than that of the intact myocardium and that the EDTS of the former was greater than that of the latter.



between E_{max} and S_{max} in various contractile states was significantly strong, suggesting that regional myocardial tactile stiffness can be a good index of regional wall stress or muscle fiber stress.

To further elucidate this point, we calculated the fiber stress based on a model and compared it with the tactile stiffness. According to Arts et al. (35) the relation between muscle fiber stress (σ_f) and left ventricular cavity pressure (Plv) in a thick-walled rotationally symmetric geometry is shown as follows: $Plv/\sigma_f = (1/3) * \ln(1 + \text{left ventricular wall volume}/\text{left ventricular cavity volume})$. Using this formula, we calculated the fiber stress (σ_f) in five dogs administered both dobutamine and propranolol. Representative tracing of phasic changes in myocardial tactile stiffness and fiber stress under baseline condition are shown in Figure 9A. The tactile stiffness followed a similar time course to that of the fiber stress. The relation between ESTS and end-systolic fiber stress in these five dogs revealed a strong correlation ($R = 0.893$) over a wide range of contractility (Fig. 9B). These results support the idea that tactile stiffness reflects left ventricular muscle fiber stress. Furthermore, when the ventricular volume does not change greatly, its absolute value can be a sensitive indicator of the contractile state.

Myocardial ischemic and infarcted model. In the model of acute myocardial ischemia used in this study, it was shown that

ESTS decreased significantly, whereas global function did not change appreciably, indicating that the tactile sensor provides a very sensitive measure of changes in regional myocardial function.

In the myocardial infarction model, it was shown that ESTS of the infarcted myocardium was decreased significantly in comparison with that of the intact region, indicating that the tactile sensor is very useful for evaluating regional myocardial contractile function in ischemic heart disease.

End-diastolic tactile stiffness. We also studied the end-diastolic tactile stiffness (EDTS). It is well known that the outer mechanical pressure of the left ventricle, a change in the cellular composition of the left ventricular wall and relaxation of myocardial cells affect left ventricular diastolic function (36). The first two are thought to be passive factors, whereas the third is thought to be an active factor. As indicators of regional myocardial diastolic function, the indexes of the early diastolic phase (38-40) for estimating the rate of relaxation, and those of the late diastolic phase (37,40-45) for estimating regional distensibility or stiffness have been advocated. The decreased regional myocardial rate of relaxation, which is an index of the early diastolic phase, is known to provide a sensitive evaluation of the relaxation reduction caused by the active factor, whereas the late diastolic indexes are considered to reflect the regional diastolic dysfunction caused by not only active but also passive factors. The pressure-length relation (40-45), pressure-thickness relation (37,43) and regional myocardial stiffness (37,43,45) have been reported to be late diastolic indexes. The EDTS measured using a tactile sensor in this study provides a late diastolic index and is related to the concept of regional myocardial stiffness. Because left ventricular compliance decreases in diastolic dysfunction owing to passive factors such as a change in the cellular composition of the ventricular wall and the thickness of the ventricular wall, the EDTS, which is the reciprocal of compliance, should increase. In fact, EDTS was increased significantly in the acute ischemia model. Moreover, in the infarcted myocardium, EDTS was also increased significantly in comparison with the intact myocardium. Therefore, in the ischemic and infarcted heart, end-diastolic myocardial stiffness measured by our tactile sensor was shown to be a

Table 2. Comparison Between Stiffness of Infarcted and Intact Myocardium

Dog No.	Stiffness of Infarcted Myocardium (g/mm ²)		Stiffness of Intact Myocardium (g/mm ²)	
	End-Systolic	End-Diastolic	End-Systolic	End-Diastolic
1	1.73	1.25	2.42	1.08
2	2.07	1.49	2.84	1.27
3	2.58	1.79	3.12	1.30
4	2.03	1.29	2.64	0.85
5	1.97	1.61	2.67	1.35
6	1.75	1.34	2.05	1.22
Mean	2.02	1.46	2.62	1.18
±SD	±0.31*	±0.21†	±0.36*	±0.18†

*p < 0.05. †p < 0.05.

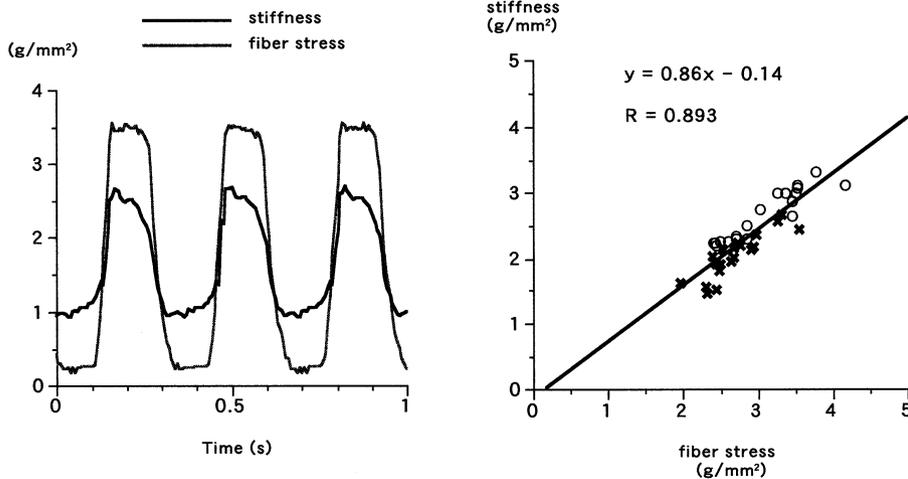


Figure 9. Left panel, Change in myocardial tactile stiffness and left ventricular fiber stress wave. Right panel, Overall relation between ESTS and end-systolic fiber stress (σ_f) in five dogs over a wide range of contractility. A strong correlation between ESTS and end-systolic σ_f was obtained ($r = 0.893$, $p < 0.0001$). Open circles = data during administration of a positive inotropic agent (dobutamine); Crossmarks = data during administration of beta-blocker (propranolol).

useful variable for indicating precisely changes in regional myocardial diastolic function.

Conclusions. We showed that myocardial stiffness measured by our tactile sensor (tactile stiffness) can be a very useful index for accurately quantifying regional myocardial function. With further improvements of the tactile sensor probe and additional experimental studies, we should be able to expand its clinical application from cardiac surgery to cardiac catheterization to evaluate the regional changes in myocardial contractility that are often encountered in ischemic heart disease.

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