Journal of Cardiology 18: 451–456, 1988

Effect of heart rate on the end-systolic wall stress-mean velocity of circumferential fiber shortening relation

Koji WATANABE
Kenji KISHIDA
Noriyuki HANEDA
Nobuko HORINO
Toshikazu NISHIO
Chuzo MORI

Summary

The correlation of left ventricular end-systolic wall stress (ESS) with the mean velocity of fiber shortening (mVcf) is an index of the myocardial contractile state, independent of the ventricular loading conditions (afterload). However, there have been few reported estimates of the effect of changes in the heart rate on the ESS-mVcf relationship. In the present study, 25 subjects with histories of Kawasaki disease (mucocutaneous lymph node syndrome: MCLS) were evaluated for the effects of changes in heart rate, afterload and preload, on the ESS-mVcf relationship. The latter was independent of afterload.

After atrial pacing, ESS decreased and mVcf increased, both significantly, compared to those of subjects at rest. The change of the ESS-mVcf relationship induced by atrial pacing approximately paralleled the mean regression line obtained in the resting state. However, the shift induced by preload was not parallel to the mean regression line for the resting population.

These data suggest that the ESS-mVcf relationship is independent of any change in heart rate, and that it may depend on preload.

Key words

Heart rate End-systolic-mean Vcf relation Contractility index

Introduction

In patients with congenital heart diesase, congestive heart failure and myocardial disease, accurate noninvasive evaluations of the ventricular systolic function are very important for assessing the severity of disease and effect of therapy. Ejection fraction, percent fractional shortening and mean velocity of circumferential fiber shortening (mVcf) are generally employed as left ventricular contractility indices[1,2]. Such indices, however, are affected not only by contractility but by changes in preload and afterload[3,4]; therefore, it is impossible to correctly analyze the contractility of the ventricle using them. Previous investigators[5,6] have reported

Department of Pediatrics, Shimane Medical University, Enya-cho 89–1, Izumo, Shimane 693

Received for publication July 27, 1987; accepted September 17, 1987 (Ref. No. 34–PS1)

that the relationship between left ventricular end-systolic wall stress (force) and the extent of shortening is useful for assessing the contractile state in changes of afterload. Later, Colan et al.[7] suggested that the left ventricular end-systolic wall stress-velocity of fiber shortening relation (ESS-mVcf relation) is a sensitive measure of the contractile state, being independent of preload and afterload. This relation may be a very useful index for clinically assessing contractility, because the position of a single point reliably indicates a position on the entire line[8]. It is very important to analyze the effect of heart rate on the ESS-mVcf relationship, since mVcf is influenced by increased heart rate[9]. However, there have been no reports of investigations of the relationships of heart rate to the ESS-mVcf. The purpose of the present study was to analyze such influence of heart rate on the ESS-mVcf relationship and to evaluate the usefulness of this relationship.

Subjects and methods

The study subjects consisted of 20 children, 6 months to 12 years of age with histories of Kawasaki disease (MCLS)[10], who had neither coronary artery lesion nor other cardiovascular disease. An ATL system V with a 3 MHz transducer was use dfor echocardiographic and phonocardiographic recordings. LV echograms were recorded at high chordal levels after long-axis and transverse scanning. The paper speed was 100 mm/sec. LV pressures were recorded with Millar micromanometer catheters inserted through a sheath by the percutaneous femoral approach. All subjects were premedicated with pethilorfan (2 mg/kg). After baseline recordings, methoxamine (total 1 mg) was intravenously infused for three min in 11 patients. During methoxamine infusion, right atrial pacing was performed to maintain the heart rate at 125/min. Right atrial pacing was performed in 13 subjects. Heart rate was gradually increased to the point pacing failure appeared. Dobutamine intravenous infusion (0.03 μg/kg/min) and saline infusion (10 m*l*/kg) into the right atrium were performed for two subjects. During saline infusion, the mean pulmonary wedge pressure increased by six mmHg in each subject. Recordings under baseline conditions were performed for a total of 15 patients.

To determine meridional wall stress, records were calibrated and digitized on an Oskon system angiogram analyzer III (Oskon Co). End-systole was identified as the time of initial appearance of the smallest left ventricular internal dimension (LVID). End-systolic pressure (ESP), LVID and posterior wall thickness (PWT) were measured at end-systole. End-diastole was identified as the time of the largest LVID. The data were measured during three to five cardiac cycles, then averaged. mVcf was calculated by dividing fractional shortening (FS) by ejection time (ET). The ESS was calculated using the following formula:

$$ESS\ (g/cm^2) = P \times D \times (1 - D^2/2L^2)/2WT$$

where P was left ventricular pressure in mmHg, D was left ventricular cavity radius in cm, L was

Table 1. Hemodynamic responses to augmented heart rate and afterload (means±SD)

	Baseline	Pacing	Afterload
Heart rate (beat/min)	112±12	139±10*	125± 2*
Peak pressure (mmHg)	95± 8	92±12	136±10*
End-diastolic volume (m*l*)	40±15	27± 8*	42±15*
End-systolic volume (m*l*)	9± 4	6± 2	11± 4*
ESS (g/cm^2)	54±12	38± 7*	62±11*
mVcf (circ/sec)	1.37±0.21	1.51±0.13*	1.24±0.19*

Significant difference from baseline condition: $p<0.05$.
ESS=end-systolic stress; mVcf=mean velocity of circumferential fiber shortening.

the long axis of the LV, and WT was PWT of the LV. In this study, L was assumed to be twice the minor diameter in all instances.

Simple linear regression by the least squares method was used to calculate the ESS-mVcf equation. The Student's t test for paired data was used for the initial statistical analysis.

Results

Hemodynamic characteristics during rest, after methoxamine, and after right atrial pacing are summarized in **Table 1**. After methoxamine infusion, the left end-systolic volume (LVESV) and ESS increased compared with those at baseline. ESS and end-diastolic volume decreased by 30% and 33% with atrial pacing, respectively. End-systolic volume during atrial pacing also decreased significantly compared to that at rest. Atrial pacing and augmented afterload induced by methoxamine were associated with an increase and a decrease in mVcf, respectively. **Fig. 1** shows ESS-mVcf relationship at rest. The relation was inversely linear. After RA pacing, the position at rest shifted to the upper left direction, and the shifting was approximately parallel to the mean regression line for the rest-

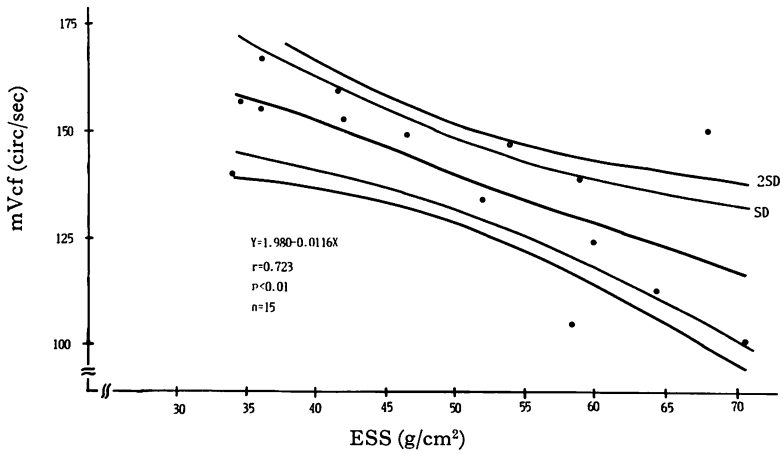

Fig. 1. Relationship between mVcf and ESS at rest.

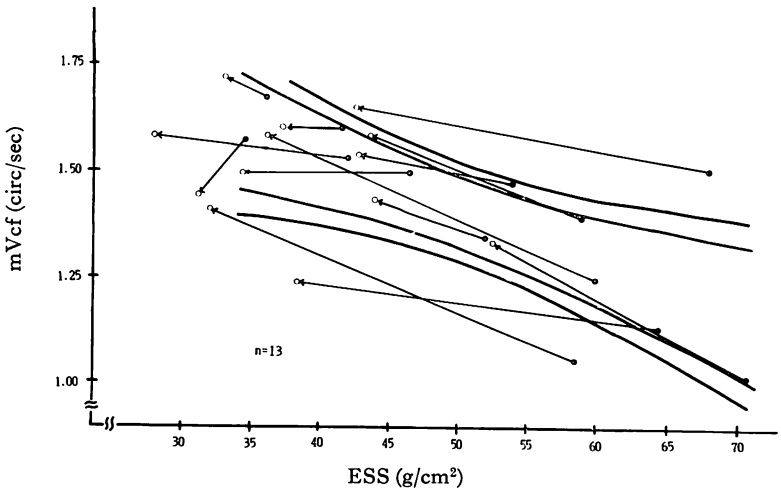

Fig. 2. Changes in the ESS-mVcf relationship by right atrial pacing.

ing population (**Fig. 2**). Most of the shifting was within the normal range (±2 SD). An increased afterload shifted the position of the ESS-mVcf relationship to the lower right direction (**Fig. 3**). The shifting induced by methoxamine was also parallel with the mean regression line at rest. During dobutamine infusion, mVcf increased markedly in spite of no remarkable change in ESS. After the saline infusion into the RA, both mVcf and ESS increased slightly. Thus, preloading was associated with a 10% mean increase in ESS and was accompanied by a one and three % increase in mVcf (**Fig. 4**).

Discussion

The ESS-mVcf relationship is reportedly very useful for noninvasively estimating the contractility of the ventricle[7,8]. However, several

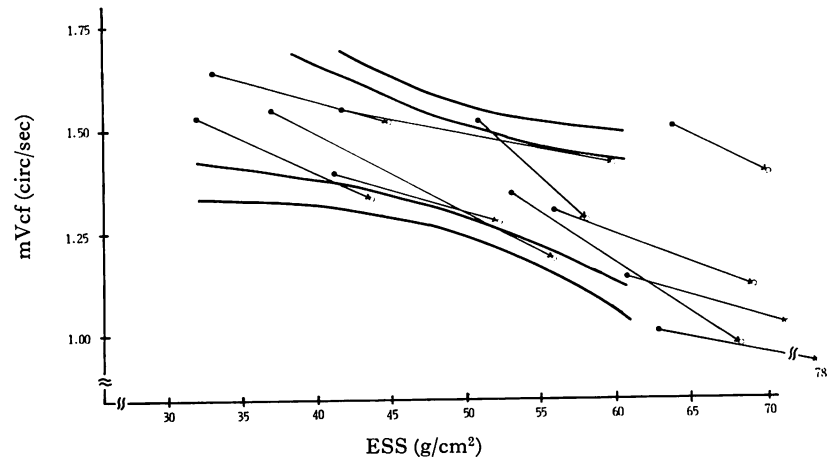

Fig. 3. Changes in the ESS-mVcf relationship by methoxamine.
Heart rate is kept constant by right atrial pacing (125/min).

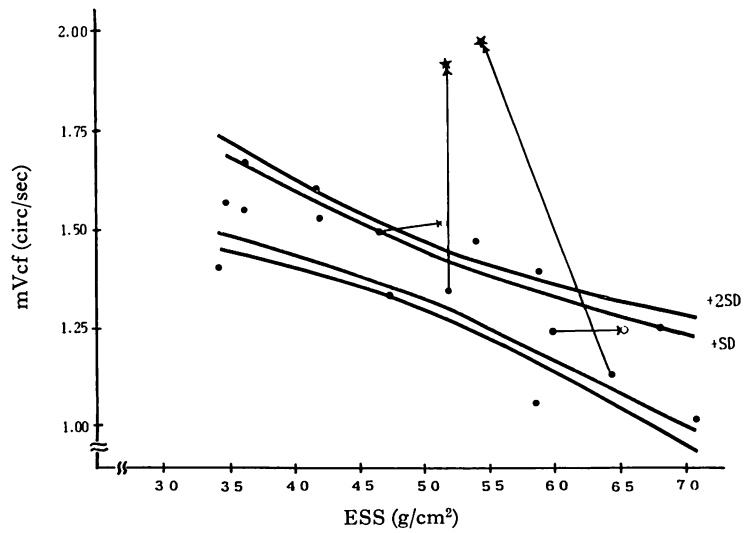

Fig. 4. Changes in the ESS-mVcf relationship by dobutamine (asterisks) **and saline** (open circles) **infusions.**

investigators[9,11,12] have reported that both mVcf and ESS are influenced by changes in heart rate. Quinones et al.[11] reported that mVcf, max dp/dt and Vmax increased by 46%, 25% and 50% repsectively, with atrial pacing. The present study also indicated a significant decrease in ESS and an increase in mVcf after atrial pacing. In our study blood pressure was recorded using tip catheters to accurately determine the change of blood pressure due to variations in heart rate. The decrease in ESS by atrial pacing may be caused by decreased end-systolic volume. The decrease was considered due to the shortening of the diastolic duration and disturbance of diastolic function caused by increased heart rate[13]. Increased mVcf seems to be caused by shortened ejection time. Suga et al[14] suggested that increases in heart rate produced by pacing had no effect on Emax, a contractility index, and was independent of cardiac loading[15,16]. The position of the ESS-mVcf relation was shifted to a left upper direction by atrial pacing. The shifting was approximately parallel to the regression line at rest. These data suggest that the ESS-mVcf relation as a contractility index is not influenced by changes in heart rate like Emax, and that the relation can be used without correction by heart rate.

The shifting of the position in the ESS-mVcf relationship induced by methoxamine was essentially parallel to the mean ESS-mVcf regression line at rest. This result is similar to those previously reported[7,8]. The data suggest that the ventricular contractility could be estimated by using this relation without acute afterloading by methoxamine, because the position of a single point reliably indicates the position of the entire line.

Reichek et al.[17] reported that subjects with aortic regurgitation, whose left ventricles were chronically volume-loaded, had mildly elevated ESS, although their intrinsic contractile states were normal. However, Colan et al.[7] suggested that no significant changes in the relation were induced by acute preload augmentation. In the present study, preloading was accompanied by a 10% mean increase in ESS. The differences between these data may have been caused by variations in volume loading. In the present study, mVcf was not influenced by preload augmentation. Previous reports suggested that increasing preload was associated with small changes in mVcf (1.3% decrease and 1.1% increase)[7,11], but the changes were not significant. Therefore, mVcf may not be changed by preload augmentation within the physiological range. These data suggest that the ESS-mVcf relationship as a contractility index is dependent on preload, because the shifting of the position in the ESS-mVcf relation by increasing preload was not parallel to the mean ESS-mVcf regression line at rest. Further investigations are required to establish the usefulness of this relation as a contractility index, independent of preload.

要　　約

収縮末期壁応力・円周短縮速度関係に及ぼす心拍数の影響

島根医科大学　小児科

渡辺弘司，岸田憲二，羽根田紀幸，堀野信子

西尾利一，森　忠三

左室収縮末期壁応力－左室平均円周短縮速度(ESS-mVcf)関係は，後負荷に関係なく，心収縮力を示すとされているが，前負荷による影響，心拍数による補正の必要性等については，詳細な報告がない．そこで我々は，冠動脈障害を伴わない川崎病既往児(20例)を対象に，ESS-mVcf 関係における心拍数，後負荷，前負荷，心収縮圧の変化による影響を検討した．心エコー図，左室圧測定を心臓カテーテル検査中に行い，11例にメトキサミン負荷（総量 1 mg，右房 pacing 125 beat/min)，13例に右房 pacing (基礎心拍数 +20% 以上)，2例に生理食塩水注入 (10 ml/kg)，2例にドブタミン負荷 (0.03 μg/kg/min) を施行した．

右房 pacing により，ESS は減少，mVcf は増大し，ESS-mVcf 関係では，安静時の傾き ($Y=-0.0116X+1.980$) とほぼ平行に左上方へ偏位した．メトキサミン負荷に対しては，ESS は増大，

mVcf は減少し，ESS-mVcf 関係では，右房 pacing と逆に右下方へ偏位した．生理食塩水負荷に対しては，ESS は増大したが mVcf は有意な変化を示さず，ESS-mVcf 関係は右方へ水平に偏位した．ドブタミン負荷では，ESS は有意の変化を示さなかったが，mVcf は著明に増大し，ESS-mVcf 関係は，著しく上方へ偏位した．

以上から，以下の4項目が明らかとなった．1) ESS-mVcf 関係は心収縮力を示すよい指標となり得る．2) ESS-mVcf 関係は，生理的範囲内（+20%）の心拍数の増加には影響を受けない．3) ESS-mVcf 関係は，後負荷に依存しない．4) ESS-mVcf 関係は，急速な前負荷の変化に影響される可能性がある．

文　献

1) Paraskos JA, Grossman W, Saitz S, Dalen JE, Dexter L: A noninvasive technique for the determination of velocity of circumferential fiber shortening in man. Circ Res **29**: 610–615, 1971
2) Fortuin NJ, Hood WP, Craige E: Evaluation of left ventricular function by echocardiography. Circulation **46**: 26–35, 1972
3) Mahler F, Ross J Jr, O'Rourke RA, Covell JW: Effects of changes in preload, afterload and inotropic state on ejection and isovolumic phase measures of contractility in the conscious dog. Am J Cardiol **35**: 626–634, 1975
4) Weber KT, Janicki JS, Hunter WC, Shroff S, Peariman ES, Rishman AP: The contractile behavior of the heart and its functional coupling to the circulation. Prog Cardiovasc Dis **24**: 375–400, 1982
5) Borow KM, Green LH, Grossman W, Braunwald E: Left ventricular end-systolic stress-shortening and stress-length relations in humans: Normal values and sensitivity to inotropic state. Am J Cardiol **50**: 1301–1308, 1982
6) Borow KM, Arensman FW, Webb C, Radley-Smith R, Yacoub M: Assessment of left ventricular contractile state after anatomic correction of transposition of the great arteries. Circulation **69**: 106–112, 1984
7) Colan SD, Borow KM, Neuman A: Left ventricular end-systolic wall stress-velocity of fiber shortening relation: A load-independent index of myocardial contractility. J Am Coll Cardiol **4**: 715–724, 1984
8) Borow KM, Henderson IC, Neuman A, Colan S, Grady S, Papish S, Goorin A: Assessment of left ventricular contractility in patients receiving doxorubicin. Ann Intern Med **99**: 750–756, 1983
9) Hirshleifer J, Crawford M, O'Rourke RA, Karliner JS: Influence of acute alterations in heart rate and systemic arterial pressure on echocardiographic measures of left ventricular performance in normal human subjects. Circulation **52**: 835–841, 1975
10) Sekiguchi M, Takao A, Endo M, Asai T, Kawasaki T: On the mucocutaneous lymph node syndrome or KAWASAKI disease: Progress in Cardiology (ed by Yu PN, Goodwin JF). Lea & Febiger, Philadelphia, 1985, p 97
11) Quinones MA, Gaasch WH, Alexander JK: Influence of acute changes in preload, afterload, contractile state and heart rate on ejection and isovolumic indices of myocardial contractility in man. Circulation **53**: 293–302, 1976
12) Quinones MA, Gaasch WH, Cole JS, Alexander JK: Echocardiographic determination of left ventricular stress-velocity relations in man with reference to the effects of loading and contractility. Circulation **51**: 689–700, 1975
13) McIntosh HD, Morris JJ Jr: The hemodynamic consequences of arrhythmias. Prog Cardiovasc Dis **8**: 330–363, 1966
14) Suga H, Sagawa K, Shoukas AA: Load independence of the instantaneous pressure-volume ratio of the canine left ventricle and effects of epinephrine and heart rate on the ratio. Circ Res **32**: 314–322, 1973
15) Suga H, Sagawa K: Instantaneous pressure-volume relationships and their ratio in the excised supported canine left ventricle. Circ Res **35**: 117–126, 1974
16) Suga H, Kitabatake A, Sagawa K: End-systolic pressure determines stroke volume from fixed end-diastolic volume in the isolated left ventricle under a constant contractile state. Circ Res **44**: 238–249, 1979
17) Reichek N, Wilson J, Satton M St J, Plapport TA, Goldberg S, Hirshfeld JW: Noninvasive determination of left ventricular end-systolic stress: Validation of the method and initial application. Circulation **65**: 99–108, 1982