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Cardiac hypertrophy in hypertrophic cardiomyopathy and hypertension evaluated by echocardiography and body surface isopotential mapping

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Summary

To elucidate the useful indices for differentiating cardiac hypertrophy due to essential hypertension (EH) from that due to hypertrophic cardiomyopathy (HCM), we examined standard 12-lead ECGs, chest radiographs and echocardiograms (Echo) in 66 EH and 46 HCM cases. Body surface isopotential mappings (MAPs) were recorded in 16 cases of EH and 18 of HCM.

The thickness of the interventricular septum (IVST) and the IVST/PWT ratio (PWT=the thickness of the posterior wall) were greater and left ventricular diastolic diameter (LVDd) was smaller in the HCM group than in the EH group.

The septal activation time (SAT), the time interval during which the maximum positive potential moves from the mid-sternal line or the left sternal border to the left mid-clavicular line in the QRS complex, correlated directly with the IVST ($r=0.55$, $p<0.005$) and the IVST/PWT ratio ($r=0.61$, $p<0.005$). When the SAT was longer than 30 msec, the IVST was over 25 mm and the IVST/PWT ratio was over 2.0, all cases belonged to the HCM category.

When subjects were limited to patients with IVST less than 25 mm, the SAT of the HCM group was significantly greater than that of the EH group.

These data suggest that the SAT may reflect the etiological differences between the septal hypertrophy of the EH group and that of the HCM group, and that these parameters of MAPs may be helpful to distinguish cardiac hypertrophy due to EH from that due to HCM.

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Key words
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Introduction

To determine the useful indices for differentiating hypertrophy secondary to essential hypertension (EH) from that due to hypertrophic cardiomyopathy (HCM), the standard 12-lead electrocardiograms (ECGs), chest radiographs and echocardiograms (Echo) in 66 EH and 46 HCM cases were investigated. The results were compared with data obtained by body surface isopotential mappings (MAPs) recorded in 16 cases of EH and 18 cases of hypertrophic cardiomyopathy (HCM).

Methods

The data of 155 subjects, including 66 hypertensives, 46 HCM, and 43 normal controls were examined. These were classified into four groups based on the following Echo findings: ① the ASH group, interventricular septal thickness (IVST) greater than 15 mm, and a ratio of interventricular septal thickness to left ventricular posterior wall thickness (IVST/PWT) more than 1.3; ② the SH group, the sum of IVST and PWT, more than 26 mm and IVST/PWT less than 1.3; ③ the IVSTh group, IVST greater than 20 mm; and ④ the LVDdh group, left ventricular end-diastolic diameter (LVDd) greater than 55 mm. Furthermore, each group was composed of two subgroups based on a history of hypertension (**Table 1**). A history of hypertension was found in 56 men and 23 women. The remaining 59 subjects (38 men and 21 women) had no history. In ASH and SH, hypertensives were significantly older than those without hypertension.

Echo was recorded with a Toshiba SSH-11A echocardiograph at a paper speed of 50 mm/sec. MAPs with a HPM-5100 system were performed for 16 cases having EH and for 18 HCM. The recording and construction of MAPs were made using the methods of Yamada et al.[1]

Table 1. Subjects

Echocardiographic findings	HT*	Cases (sex)	Age
Normal	−	43 (M 26, F 17)	51± 8
ASH	+	42 (M 25, F 17)	56±11
	−	21 (M 13, F 8)	48±15
SH	+	21 (M 16, F 5)	57± 9
	−	16 (M 13, F 3)	47±14
IVSTh	+	20 (M 11, F 9)	58±12
	−	13 (M 7, F 6)	51±16
LVDdh	+	6 (M 4, F 2)	47±12
	−	9 (M 6, F 4)	46±13

* History of hypertension.
ASH=IVST>15 mm and IVST/PWT>1.3; SH=IVST+PWT>26 mm and IVST/PWT>1.3; IVSTh=IVST>20 mm; LVDdh=LVDd>50 mm.

Results

The results of Echo measurements are shown in **Fig. 1**. IVST and PWT did not differ irrespective of a history of hypertension. In the ASH group, however, the ratio of IVST/PWT of the non-hypertensives was apparently greater than that of the hypertensives ($p<0.005$), and in the IVSTh group, greater left ventricular end-diastolic diameter (LVDd) was observed in the non-hypertensives. The LAD of the SH group was larger than that of the ASH group in the hypertensives ($p<0.025$), and ejection fraction (EF) of the LVDdh group tended to be less than that of the other groups. The hypertensives in the SH group had the greater cardiothoracic ratio (CTR) compared with that of the non-hypertensives, and SV1±RV6 in ECG did not differ significantly by group in this study (**Fig. 2**). Systolic blood pressure of the patient groups categorized as having abnormal Echo findings was approximately 20 mmHg higher than that of the normal controls. **Fig. 3** represents mean values and standard deviations of each parameter in each hypertensive group. LVDd did not differ

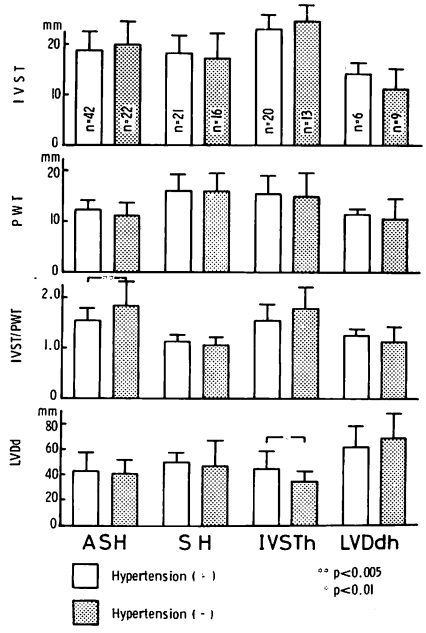

Fig. 1. Comparison of echocardiographic indices between hypertensives and non-hypertensives.

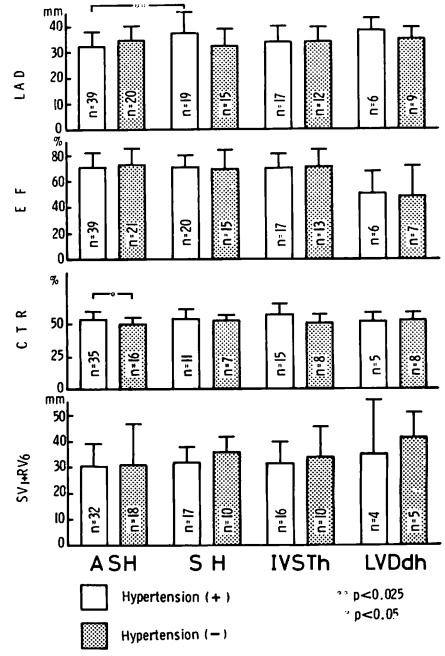

Fig. 2. Comparison of left atrial dimension (LAD), ejection fraction (EF), cardiothoracic ratio (CTR) and $SV_1=RV_1$ in standard 12-lead ECG between two groups.

by group, except for the LVDdh group. The latter group had larger LVDd and significantly lower EF than those of the other groups. The mean age of the LVDdh group was slightly less than those of the other groups, and the LVDdh group had a history of hypertension longer than the other groups. Therefore, it seemed that the LVDdh group experienced earlier onsets of hypertension.

To determine the useful indices for differentiating hypertrophy due to EH from that due to HCM, MAPs were studied in 16 EH and 18 HCM cases. **Fig. 4** shows the actual ECG and MAPs of a hypertensive case, and **Fig. 5** shows those of a HCM patient. In this study " the septal activation time (SAT) " derived from MAP indicates the time interval during which the maximal positive potential moves from the mid-sternal line or left sternal border to the left mid-clavicular line. The SAT reflected the duration of electrical excitation of the interventricular septum. Furthermore, Rmax-V, Smax-

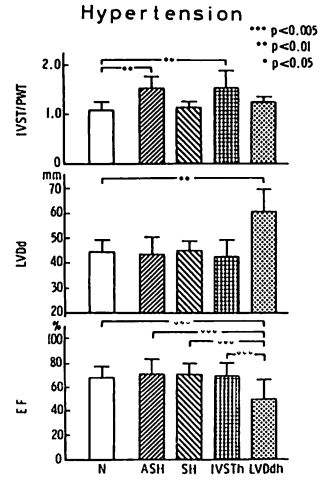

Fig. 3. IVST/PWT, LVDd and EF of hypertensive cases and normal controls (N).

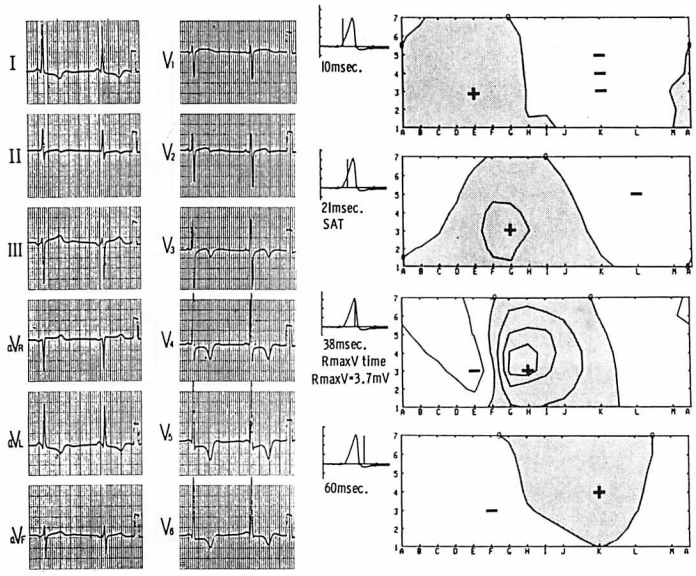

Fig. 4. An example of MAPs and standard ECG in a patient with hypertension (64-year-old woman).

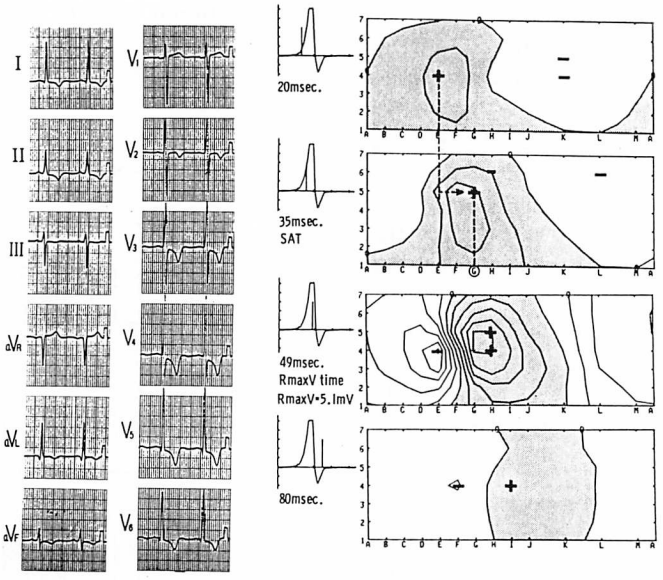

Fig. 5. An example of MAPs and standard ECG in a patient with hypertrophic cardiomyopathy (51-year-old man).

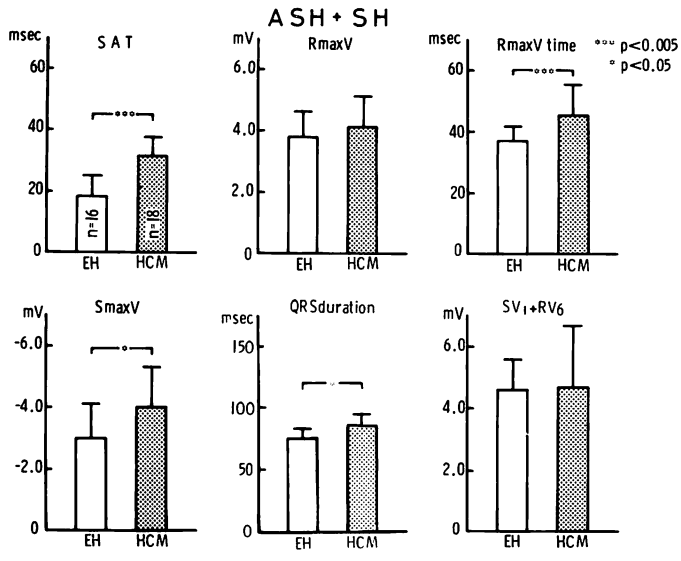

Fig. 6. Comparison between EH and HCM concerning indexes from standard 12-lead ECG (SV_1+RV_6) and indices from MAPs (SAT, Rmax-V, Rmax-V time, Smax-V, QRS duration) **in ASH+SH groups.**

QRS duration is calculated every 1 msec with root mean square of lead voltage in 87 body surface points.

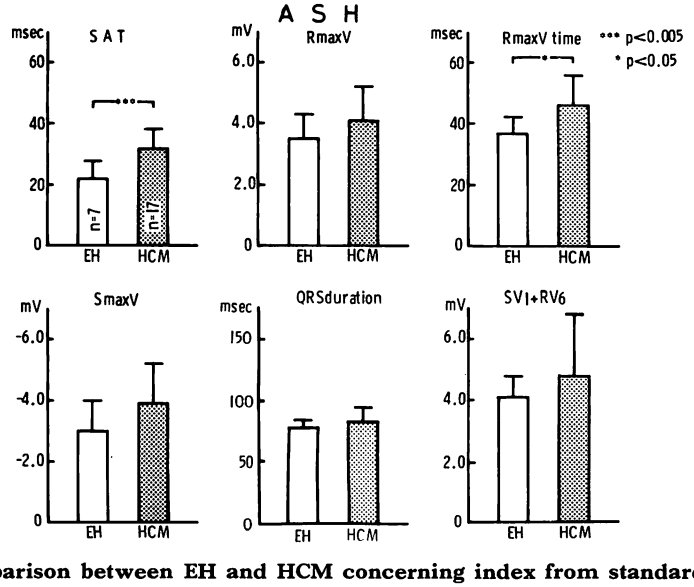

Fig. 7. Comparison between EH and HCM concerning index from standard 12-lead ECG (SV_1+RV_6) and indices from MAPs (SAT, Rmax-V, Rmax-V time, Smax-V, QRS duration) **in ASH group.**

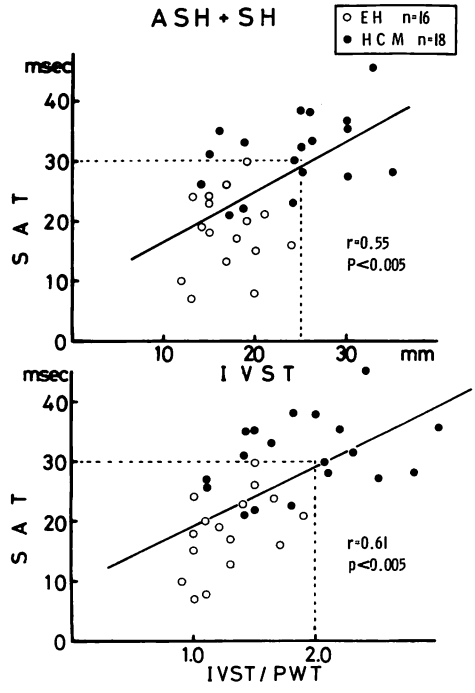

Fig. 8. Correlation between SAT and echocardiographic values (IVST and IVST/PWT).

V, Breakthroughtime (BT-time) and the QRS-intervals were examined in this study, where Rmax-V means the maximal positive potential, and Smax-V means the minimal negative potential of the QRS complex. Comparison of hypertensives having ASH or SH on Echo with HCM having ASH or SH but no hypertension revealed more SAT, Rmax-V and Smax-V in HCM than in the hypertensives. However, voltage criteria derived from the standard ECG (SV_1+RV_6) showed no significant differences between the hypertensives and those with HCM (**Fig. 6**). In 34 subjects whose MAPS were recorded, SAT could distinguish HCM from the hypertensives more effectively than could IVST obtained using Echo (**Fig. 7**). SAT, which was expected to reflect the time interval of electrical excitation of the IVS, correlated with the IVST measured using Echo ($r=0.55$, $p<0.005$). SAT and the IVST/PWT ratio also correlated significantly ($r=0.61$, $p<0.005$). These results suggested

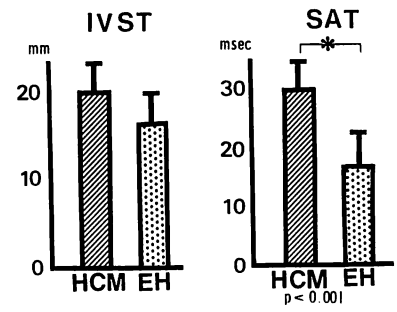

Fig. 9. Comparison of IVST and SAT of HCM and EH with mild to moderate septal hypertrophy.

SAT is useful for evaluating the etiology of ventricular hypertrophy (**Fig. 8**). Especially, cases with SAT more than 30 msec, IVST over 25 mm, and the IVST/PWT ratios over 2.0 could belong to the HCM group. **Fig. 9** shows the difficulty to differentiate HCM from EH by IVST, but SAT was able to discriminate two types of hypertrophy ($p<0.001$).

Discusion

Though it is a difficult problem, it is important to differentiate noninvasively ventricular hypertrophy due to EH from that of HCM. The causes of cardiac hypertrophy have not been clearly distinguishable by any noninvasive methods including Echo[2,3] 12-lead ECG[4], or chest X-P[5].

However, in mild to moderate septal hypertrophy (IVST less than 25 mm), the electrical index derived from MAPs differed significantly for hypertensives and non-hypertensives, despite the fact that IVST and the IVST/PWT ratio were not significantly different between these two groups even in the group with ASH. Van Dam et al.[6] reported that in epicardial ECGs, the total septal activation time was prolonged in hearts with hypertrophy. MAPs have many leads on the body surface, so that they reflect topical hypertrophic changes in the myocardium more precisely than do standard ECGs. Ishikawa et al.[8] studied MAPs of 23 non-hypertensive patients with interventricular

septal hypertrophy, and compared their data with those of healthy controls. Their results indicated that SAT of interventricular septal hypertrophy was significantly longer than that of controls. In the present study, SAT responded to interventricular septal hypertrophy; SAT correlated with IVST and IVST/PWT. Thus, SAT seems to be a useful parameter with which IVST can be evaluated quantitatively.

Conclusions

1. To ascertain the indices for differentiating cardiac hypertrophy due to EH from that due to HCM, standard 12-lead ECGs, chest radiographs and echocardiograms were recorded for 66 EH and 46 HCM cases. MAPs were obtained for 16 EH and 18 HCM cases.

2. The IVST/PWT ratio was apparently greater in the non-hypertensives with ASH than in the hypertensives.

3. SV1+RV6 in ECG could not distinguish EH from HCM.

4. Whenever the patients had SAT longer than 30 msec, IVST over 25 mm and the IVST/PWT ratio over 2.0, all cases belonged to HCM, but not EH. This indicates that SAT could be helpful in the differential diagnosis of EH and ICM.

高血圧と肥大型心筋症に関する心エコー図および体表面電位図による検討

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肥大型心筋症による肥大心と高血圧による肥大心の差異を検討する目的で，心エコー図上異常を示す高血圧症 66 例と，特発性心筋症と診断した 46 例を対象として，心エコー図，胸部レ線および心電図所見を比較検討した．さらに，高血圧症 16 例および肥大型心筋症 18 例において，体表面心臓電位図 (MAPs) を記録した．

肥大型心筋症群では，高血圧群に比し，心室中隔厚 (IVST) および心室中隔と左室後壁厚との比 (IVST/PWT) がより大きく，左心室拡張末期径 (LVDd) はより小であった．

MAPs を用い，心室中隔興奮に要する時間を推定する目的で，QRS 開始時に，胸骨正中線上あるいは左傍胸骨線上に存在していた極大が，左鎖骨中線上に移動するまでの時間を求めて，中隔興奮推定時間 (SAT) とした．SAT は IVST 厚と正相関を示し (r=0.55)，IVST/PWT 比とも正相関を示した (r=0.61)．

特に，IVST が 25 mm 以上，SAT が 30 msec 以上で，かつ，IVST/PWT が 2.0 以上のものは，全例，肥大型心筋症と考えられた症例であった．

中等度以下の中隔肥大 (IVST：25 mm 以内) を示す症例に限って肥大型心筋症と高血圧症とを比較すると，IVST に有意差は無いが，肥大型心筋症群の SAT は，高血圧群よりも有意に延長を示した．

これらの結果より，SAT は肥大型心筋症と高血圧症における病因的な差異を反映しており，これらの指標を用いて，両群の肥大心を識別し得る可能性が示唆された．

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