Bull Yamaguchi Med Sch 38(3-4) : 71-81, 1991

Spectral Analysis of Endocardial Electrogram on the High-Lateral Right Atrium during Sinus Rhythm and Pacing stimuli: The Relationship between the Frequency Spectrum of Atrial Wave and the Occurrence of Atrial Fibrillation

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(Received April 8, Revised Jun 11, 1991)

Summary The provocation of atrial fibrillation (Af) was studied in 23 patients by 10 single extrastimulations, and by rapid pacing over three minutes at the mid-lateral right atrium. The frequency spectrum of atrial electrogram on the high-lateral right atrium (HRA) was simultaneously computed with the fast-Fourier transform analysis.

Results were shown as follows: 1) The single extrastimulation induced no Af in any patients, but rapid pacing induced Af in 20 patients with various heart diseases and the shorter interval of atrial effective refractory period (AERP). 2) All of the 5 patients with a history of transient Af had significantly shorter AERP of the right atrium than that in patients without a history of Af. 3) During both single extrastimulation and rapid pacing, both the higher- and lower-frequency components of atrial electrogram in patients with induction of Af were greater in amplitude than those in patients without induction of Af.

The patients with induction of Af by pacing may have the vulnerable electrophysiologic condition around the region of HRA, probably related to the higher sensitivity of right atrium to the pacing stimuli, regardless of the basal heart disease.

Key words : Atrial fibrillation, Atrial vulnerability, Frequency analysis, Rapid pacing, Fragmentation

Introduction

The bipolar endocardial electrogram can be changed by the location and direction of electrodes, and the intrinsic delection amplitude due to the active myocardium at the contact of electrode, and so on[1]. These factors have various influences on the electrogram waveform to conform the various types of endocardial electrogram[2]. During the electrical pacing, these factors have more various influences than those before pacing. Especially in the atrium, the fragmented type of electrogram often appear preceding or

during the atrial fibrillation (Af) after pacing stimulation[3,4]. Therefore, we consider that there may be a need for the quantitative analysis of waveform of endocardial electrogram during pacing, and that there may be a possibility for the detection of the electrophysiologic condition in atrium, especially before the Af.

The quantitative analysis of frequency spectrum for the body-surface electrocardiogram (ECG) and the intracardiac electrogram has been reported in the last two decades[5-9]. However, the measurement of the pacing-induced change in waveforms of endocardial electrogram has not been reported. In this study, we attemped to calculate the frequency spectrum of the endocardial electrogram in the right atrium during pacing stimuli, with the fast-Fourier Transform (FFT) analysis using an optimal window function. And then, we attempted to investigate 1) the cycle length required to initiate the Af by single extrastimulation and rapid pacing, 2) the changes in frequency spectra of endocardial electrogram in the atrium by the variable cycle length of single extrastimulation and rapid pacing, and 3) the clinical relationship between the changes in frequency spectra of atrial electrogram and the atrial vulnerability to Af.

Materials and methods

Patients : The study group consisted of 23 patients (14 males and 9 females, mean age of 55 years old, age ranged from 16 to 74 years) referred to the Hospital of Yamaguchi University for electrophysiologic evaluation from 1984 to 1986. The clinical diagnosis are listed in Table 1.

Electrophysiologic study : Schema of the right atrial mapping and measurement system we used is shown in Figure 1. Two bipolar catheters (No.6F #, USCI) were inserted percutaneously into the femoral vein of each patient and were advanced to the high-lateral right atrium (HRA), and to the low-lateral right atrium (LRA) or the right ventricular (RV) apex under fluoroscopic guidance. In addition, two quadripolar catheters were inserted to the mid-lateral right atrium (MRA), and to the tricuspid area for His bundle potential (HBE). Bipolar catheter for the HRA and quaripolar catheter for the MRA had an interelectrode distance of 10mm, while quadripolar catheter for the HBE and bipolar catheter for the LRA or RV apex had an interelectrode distance of 5mm. The width of the electrode ring was all the same size of 2mm.

Electrograms of the HRA, MRA, LRA and HBE were recorded with the use of catheter an interelectrode distance of 10mm. The electrogram on the HRA and LRA were accomplished with the distal electrode paired to the proximal electrode and the HBE electrogram was recorded with the distal electrode paired to the third electrode ring, while the MRA electrogram was performed with the third electrode paired to the fourth electrode ring. These electrograms were filtered at 5 to 550 Hz. The intracardiac electrograms were recorded at variable gain to achieve the best electrographic definition and were accomplished by a calibration of 1 mV obtained from an amplifier. If the signal adequacy of atrial electrogram was less than 0.5 mV or if there was significant beat-to-beat variation, the electrode was repositioned to conform a complete electrode contact and to achieve the best stability. These electrograms were displayed simultaneously on a multichannel oscilloscope (Siemens-Elema AB) and recorded on a 8 channels Mingograf (Siemens-Elema 82) at a paper speed of 100mm/sec, and stocked in the magnetic tape recorder (TEAC, R-61D) with the frequency characteristics from DC to 625 Hz at 50 dB.

Stimulation protocol : The site of the MRA was stimulated by the quadripolar catheter with the distal electrode paired to the second electrode ring. Twice threshold, 1.8 msec rectangular stimuli were delivered from a programmable battery operated stimulator (Medtronic, model 5325). The stimulation protocol was performed as follows:

1) Incremental atrial pacing of single extrastimulation scanning the atrial activation at sinus rhythm is accomplished starting with the cycle length of 750 msec, which was shorter than that of sinus rhythm, and continuing with decremental coupling interval down to 300 msec by a stepwise decrement of 150 or 100 msec. Subsequently, coupling intervals were decreased stepwisely 20 by 20 msec until stimuli reached the refractoriness.

2) Rapid pacing was then started with the rate of 80 beats/min, and the rate of pacing was increased up to 368 beats/min in a stepwise increment of 20 beats/min. The duration of each pacing was for 3 minutes. The endpoint of rapid pacing was the induction of the sustained Af, or the minimum cycle length of 170msec.

Table 1 Clinical diagnosis and Electrophysiologic finding

No.	Age	Sex	Clinical and Electrophysiologic diagnosis	Previous history of Af	Electrophysiologic finding During sinus rhythm PP	PA	AH	HV	SACT	max SRT	AERP
Patients with induction of Af											
1	48	F	SSS		900	20	110	40	100	1340	230
2	54	M	SSS, AP		950	40	100	30	100	1070	340
3	64	F	SSS, II°AVB(IHB), HT		1310	30	150	55	−	1860	260
4	74	F	SSS, PSVT		900	20	110	40	−	5370	300
5	48	M	SSS	(+)	1680	30	110	50	−	15500	350
6	60	F	SSS, AT		1300	-10	110	40	−	4520	320
7	65	M	SSS, DCM		1400	30	110	40	−	3920	300
8	62	M	SSS, VPCs	(+)	1750	40	90	40	−	6620	250
9	62	M	SSS	(+)	1100	50	70	50	70	1550	240
10	48	M	SSS, I°AVB(AHB), VPCs, Af	(+)	1000	30	120	40	170	2650	230
11	72	M	SSS, I°AVB(AHB)		1230	40	240	40	195	4420	−
12	58	M	III°AVB(HVB)		650	40	110	45	−	−	200
13	49	M	II°AVB(AHB), Wonder pace		1200	30	100	40	−	1550	280
14	61	F	Drug-induced AVB		860	35	100	40	−	1480	−
15	67	M	III°AHB(AHB, IHB)		1980	40	100	40	230	1550	−
16	27	F	PSVT(AVN reentry)		1050	50	90	30	150	1370	290
17	47	M	VPCs, PSVT, Af	(+)	970	30	100	40	−	1310	200
18	16	M	I°AVB(AHB), VT salvo		1090	70	100	35	65	1060	260
19	71	M	CRBBB, II°AVB(HVB), IHD, HT		870	40	70	30	50	1410	300
20	36	F	WPW, PSVT		1080	40	100	30	130	1100	−
Patients without induction of Af											
21	68	F	SSS, CRBBB, II°AVB(HVB), HT		1400	40	90	55	175	8450	400
22	61	F	SSS		2250	50	80	40	−	10150	360
23	45	M	VT salvo, Afl		1100	50	100	50	280	1680	330

Values : Age and Electrophysiologic data were represented by years old and msec, respectively. Abbreviations: M=Male; F=Female; SAB=Sino-atrial block; AP=Angina pectoris; IHD=Ischemic heart disease; HT=Hypertension; I°, II° or III° AVB=The first, second or third degree of atrio-ventricular block; AHB=Atrio-His bundle block; IHB=intraHis bundle block; HVB=His bundle-ventricular block; CRBBB=Complete right bundle branch block; DCM=Dilated cardiomyopathy; VPCs=Ventricular premature contractions; PSVT=Paroxymal supraventricular tachycardia; SSS=Sick sinus syndrome; AT=Atrial tachycardia; Wonder pace=Wondering pacemaker; AVN reentry=Atrioventricular nodal reentry; VT salvo=Short run of ventricular tachycardia; Af=Atrial fibrillation; PSVT=Paroxysmal supraventricular tachycardia; WPW=Wolff-Parkinson-White syndrome; Afl=Atrial flutter; Others see text for details.

The coupling interval of vulnerable zone to Af by single extrastimulation or rapid pacing was used to initiate the sustained Af with the duration of 30 seconds or longer[3,4].

Definitions : PP interval (PP), PA interval (PA), AH interval (AH), HV interval (HV) and Atrial effective refractory period (AERP) were measured according to the method of Josephson et al.[10]. Maximum sinus node recovery time (max SRT) and calculated sinoatrial conduction time (SACT) were measured according to the methods of Narula et al.[11] and Strauss et al.[12], respectively.

Sampling of A wave : As shown in Figure 1, the micro-computer system and peripheral equipments (NEC/San-ei, Signal processor 7T17) have a combined system with the preamplifers (maximum input range of ±10 V), A/D converters (12 bits accuracy, sample holds 2μsec, the lowest resolution of 4.9μV), microprocessor (42bits),

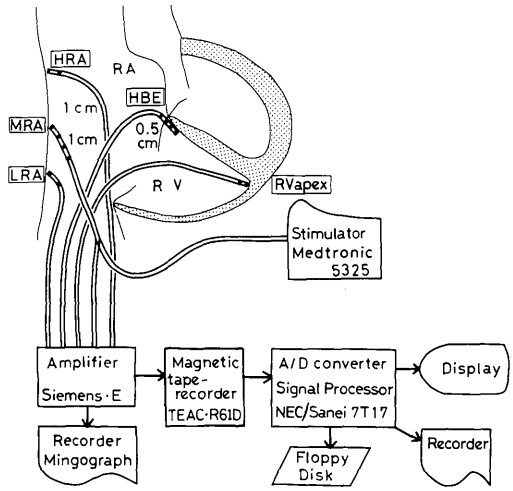


Figure 1 Schema of the atrial mapping electrode and a block diagram of the computer-based data analysis system. Abbreviations: HRA=The high-lateral right atrium; MRA=The middle-lateral right atrium; LRA=The low-lateral right atrium; HBE=The His-bundle electrogram; RA=The right atrium; RV=The right ventricle.

display, floppy disk and the printer. The digitized signal flowing from magnetic tape recoder was optimized the amplitude to show the maximum input range on the display.

In this study, we chose the A wave on the HRA electrogram for the sample window of FFT analysis. The HRA electrogram is relatively stable free from wall motion. In the HRA position, the far-field ventricular wave (R wave) is smallest in peak-to-peak amplitude, and the ratio of amplitude of A wave to the amplitude of R wave is greatest than those in other position[7-9]. Generally, the far-field repolarization wave (T wave) on the HRA electrogram is almost diminutive in amplitude or no deflection[7-9]. If an amplitude of T wave was significantly recognized, we excepted its case from the sampling to the FFT analysis.

In sinus rhythm, the sample time of A wave was set at the duration of 150 or 160 msec (Figure 2(A)). Using the one cursor-line on the display by controlling the joy-stick, the beginning of sample time was set at 30 msec or more before the initial upstroke of A wave. Whenever possible, to avoid the small R wave and to encompass the A wave, the ending of sample time was set using the other cursor-line at 150 or 160 msec from the beginning of sample time.

During both single extrastimulation and rapid pacing, the sample time was set also at the duration of 150 msec using the two cursor-line (Figure 2(B,C)). In order to avoid the afterpotential followed by pacing pulse, the beginning of sample time was set at 20 or 30 msec or more after the initial upstroke of pacing pulse. Subsequently, the ending of sample time was also set at 150 msec after the beginning of sample time. If there was the R wave due to Wenckebach phenomenon on the sample time of A wave, its A wave was excepted from the sampling[13].

FFT analysis : In order to avoid edge discontinuity or to eliminate the source of harmonic error, we used the Hamming window function to the FFT analysis[14]. The amplitude of each frequency by its function was ranged more than -50 dB (by the accuracy of 10μV at the calibration of 1 mV)[14].

Firstly, after the sampling of A wave was performed, the digitized signal of sample time was multiplicated by Hamming window function. Secondly, the multiplied data were accomplished using the 1024-points to the FFT analysis. The fundamental or least-resolutional frequency of the FFT analysis is the reciprocal of sample window. In detail, the fundamental frequency from our program (NEC/San-ei, Modified of Standard package No.2) was the frequency of 2.5 Hz at the maximum frequency of 1 KHz as shown by the equation: f min =1 KHz/400, where f min is fundamental frequency. These processes of both the multiplication and the FFT analysis were summed 100 times in sinus rhythm and 10 times during both single extrastimulation and rapid pacing. Subsequently, each power spectrum was averaged for each frequency to obtain a spectrum free from beat-to-beat variation and to eliminate the random noise as much as possible. The averaged spectrum was transformed to the natural logarithm by the equation $-20 \times \log W(f)$ dB, where W(f) represents the power of each frequency on the basis of calibration signal of 1 mV.

The endpoint for the sampling during single extrastimulation was the AERP, or the minimum coupling interval of 200 msec. During rapid pacing, the sampling endpoint was the cycle length before the Af initiated, or the minimum cycle length of 200 msec.

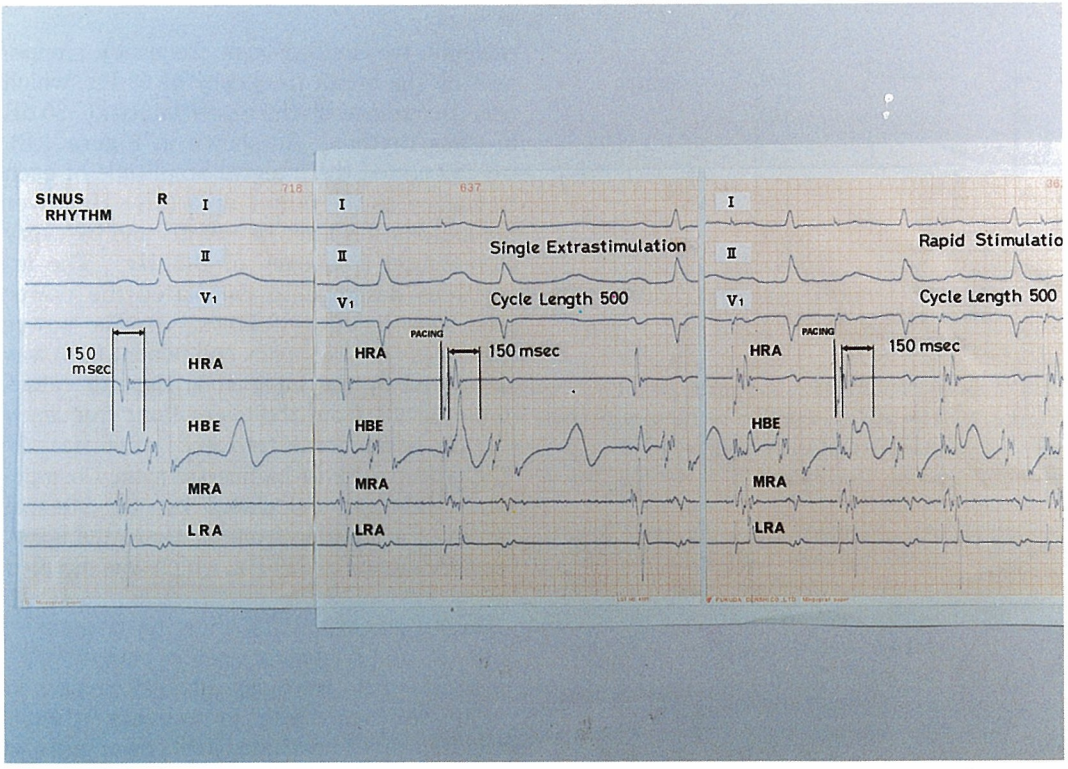


Figure 2 The sampling time of atrial wave.
(A): In sinus rhythm.
(B): During single extrastimulation.
(C): During rapid pacing.
Abbreviations: I, II, V1=The electrocardiogram (ECG) on the body surface; HRA, MRA, LRA, HBE=The electrogram from high-lateral right atrium, mid-lateral right atrium, low-lateral right atrium, and His-bundle, respectively.

Statistics : Statistical analysis was performed using Student's paired t-test or Cochran-Cox non-paired test. The p value of less than 0.05 was taken significant. All data were presented as a mean ±standard error (SEM).

Results

Provocation of Af : Table 1 summarizes the clinical and electrophysiologic diagnosis in 23 patients.

The single extrastimulation by every coupling interval could not induce the Af in any patients. In contrast, the rapid pacing could induce the Af in 20 patients with various heart diseases. The history of Af and the cycle length of rapid pacing at the initiation of Af are listed in Table 1. The cycle length of 5 patients with induction of Af who had had a previous history of Af was more significantly prolonged than that of other 15 patients with induction of Af who had had no history of Af (304±30 vs 217±11 msec, $p<0.05$)(Figure 3(A)).

The duration of the AERP was significantly shorter in patients with induction of Af than that in patients without induction of Af (272±11 vs 363±20 msec, $p<0.01$)(Figure 3 (B)). The duration of the AERP was shorter in 5 patients who had had a previous history of Af than that in patients who had had no history of Af, but the difference was not statistically significant (298±14 vs 254±25

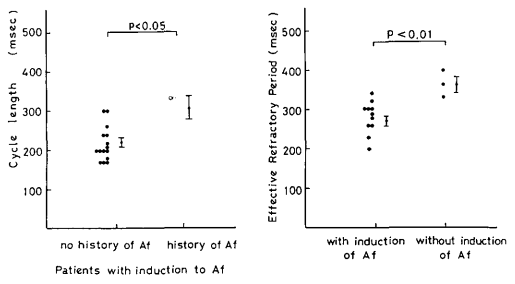


Figure 3 The atrial effective refractory period (AERP) and cycle length to induce the atrial fibrillation (Af) during rapid pacing.
(A): Comparison of the cycle length in the patients with induction of Af (n=20) between who had a previous history of Af ((+); n=5; open circle) and who had no history of Af ((−); n=15; closed circle).
(B): Comparison of AERP between the patients with induction of Af (Af(+); n=20) and the patients without induction of Af (Af(−); n=3). Open and closed circles represent as same as those in (A).
Values represent mean ±SEM msec.

msec, NS).

The FFT analysis : In sinus rhythm, the peak to peak amplitude of A wave from the HRA electrogram was 1.84±0.56 mV and the duration of deflection of A wave was 106±4 msec.

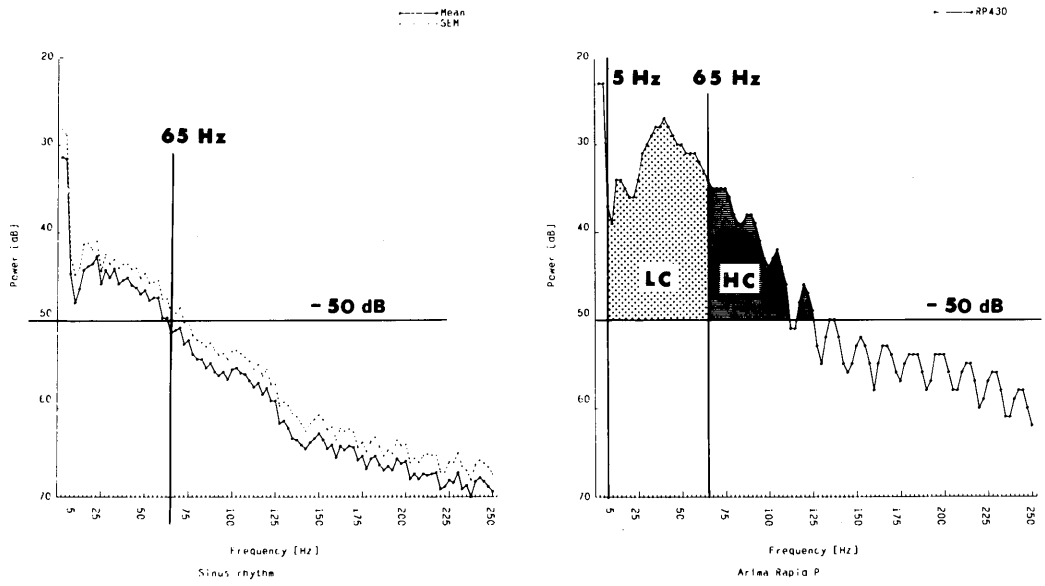
In sinus rhythm, power spectrum pattern of the sampled A wave from the HRA electrogram is shown by mean ±SE in Figure 4(A). The power spectrum of the A wave ranged within 65 Hz at the level of -50 dB, and its peak frequency of mean value was 22.5 Hz at -43 dB.

During both single extrastimulation and rapid pacing, most parts of the power spectrum of A wave were significantly greater in amplitude than those of A wave in sinus rhythm, and were especially increased by rapid pacing.

Frequency component : In order to compare the changes in frequecy spectrum among the pacing cycle lengths and among the patients, we divided each frequency component by the break frequency of 65 Hz, which was determined by the mean values at -50 dB in sinus rhythm. As shown in Figure 4(B), we calculated the integral amplitude of each frequency as the power area (dB×Hz), over the elimination limit of -50 dB and by every incremental frequency of 2.5 Hz. The argorithms designed to calculated the power area were listed as follows: 1) The power area of lower-frequency component (LC) was calculated by the integral arithmetic values of amplitude from the lower limit frequency of 5 Hz to the break frequency of 65 Hz. 2) The power area of higher-frequency component (HC) was calculated by the integral values of amplitude from 65 Hz to the upper limit frequency of 550 Hz, which was dictated by our system.

During single extrastimulation (Figure 5), both HC and LC power area in patients with induction of Af were significantly greater in amplitude than those in patients without induction of Af, excluded HC power area at the cycle length of 750 msec. And then, both HC and LC power area were not significantly increased by the decremental coupling interval in both patients with and without induction of Af (Figure 5, upper and lower).

During rapid pacing (Figure 6), each of LC power area in patients with induction of Af by every cycle length was greater in amplitude than that in patients without induction of Af (Figure 6, lower). In addition, each amplitude of LC power area in patients both with and without induction of Af was slightly increased in the decremental cycle length shorter than 333 msec. In contrast, HC power area in both patients with and without induction of Af could not be increased in the cycle length longer than 375 msec, but could be significantly and stepwisely increased followed by the decrease in the cycle length shorter than 333 msec. In the cycle length longer than 333 msec, each of HC power area in patients with induction of Af was greater in amplitude than that in patients without induction of Af. Moreover, in the cycle length shorter than 260 msec, there was no significant difference of the amplitude in HC power area between the patients with and without induction of Af (Figure 6, upper).



a b

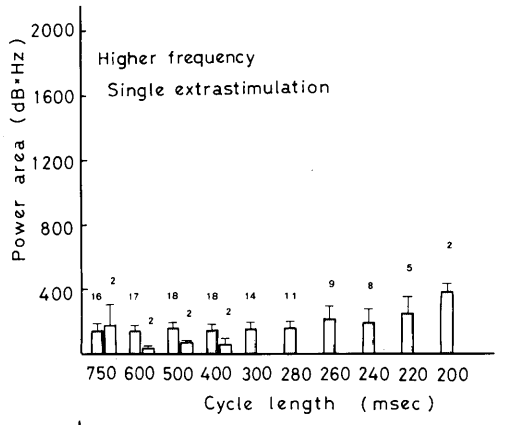
Figure 4 The power spectrum of A wave and the data analysis method.
(A): The spectrum pattern of the HRA-A wave in sinus rhythm. Solid line=mean values. Dotted line=one standard error.
(B): The calculation method of the power area (dB×Hz). LC=Lower-frequency component over -50 dB from 5 to 65 Hz. HC=Higher-frequency component over -50 dB from 65 to 550 Hz.

Discussion

Atrial vulnerability : In the present study, the patients with shorter AERP had more susceptible condition to Af than the other patients. Moreover, the patients with a history of Af had more shorter AERP and more longer duration in the cycle length of rapid pacing than that the patients with no history of Af had. Furthermore, the more cumulative effect by rapid pacing might promote the developing of atrial vulnerability than that by single extrastimulation. On the other hand, there was no difference of basal heart disease between the patients with and without induction of Af. Regarding to the predisposing condition of shorter AERP and the effect of rapid pacing, the previous studies have been demonstrated the same results as our study[4,15,16]. In other words, the results in our study suggested that the atrial myocardium in patients with induction of Af or with a history of Af might have the vulnerable condition of shorter AERP, and that the prolonged duration of rapid pacing on the sensitive atrial myocardium might promote the Af, regardless of basal heart disease.

Spectral analysis : Some other investigators have reported the amplitude, duration and frequency spectrum of atrial electrogram, using a pacemaker lead on atrial appendage[7]. Using the same standard bipolar electrode as that we used, Timmis et al. and Aubert et al. defined as the bandwidth of A wave; 33.0±18.6 Hz (25% thereof), and 40.6±25.6 Hz (-3 dB thereof) (mean ±SD), respectively[8,9]. If we calculate our data according to these author's method, the bandwidth ranged from 5 to 65 Hz (25% thereof) and from 15 to 42.5 Hz (-3 dB thereof), from the mean spectral plotts. Our data of A wave were approximately compatible to their values.

Considerations : In the literature, the bipolar electrogram was influenced by many



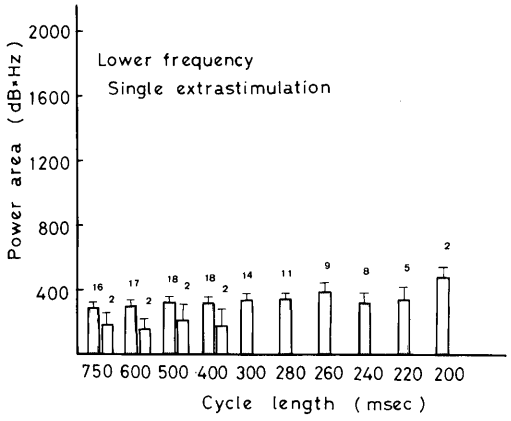
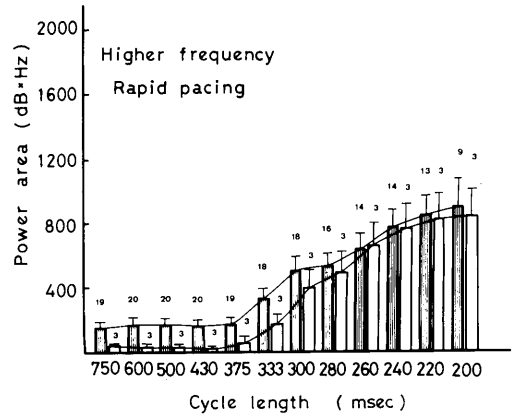


Figure 5 The power area (dB×Hz) during single extrastimulation.
Upper: In the higher-frequency component (HC). Lower: In the lower-frequency component (LC). Hatched barrs: The patients with induction of atrial fibrillation (Af). Open barrs: The patients without induction of Af. Shoulder numbers are the number of patients.
Values represent mean ±SEM.



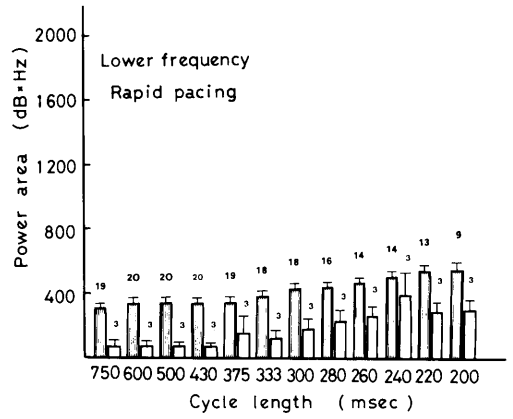


Figure 6 The power area (dB×Hz) during rapid pacing.
Upper: In the higher-frequency component (HC). Lower: In the lower-frequency component (LC). Others see in Figure 5.

electrode factor such as location and direction of the electrode, intrinsic deflection amplitude due to the active myocardium, input impedence, interelectrode distance, surface area of electrodes, electrode contact to the active tissue and depolarization wavefront, etc[1,6]. Especially, the interelectrode distance and the velocity or direction of wavefront could determine the timing of the cathode (tip) and anode (ring) intrinsic deflection of bipolar electrodes[6]. The relation between the deflection of bipolar electrode and the wavefront under various conditions is schemmatically shown in Figure 7.

In sinus rhythm, when the wavefront is parpendicular to the line between the cathode and the anode, the bipolar signal is low amplitude, as shown in Figure 7-A. When the wavefront is parallel, the bipolar signal is high amplitude (Figure 7-B). If the parallel wavefront is fast in conduction, the bipolar signal is more sharp and larger in amplitude[6] (Figure 7-C). On the other hands, the bipolar

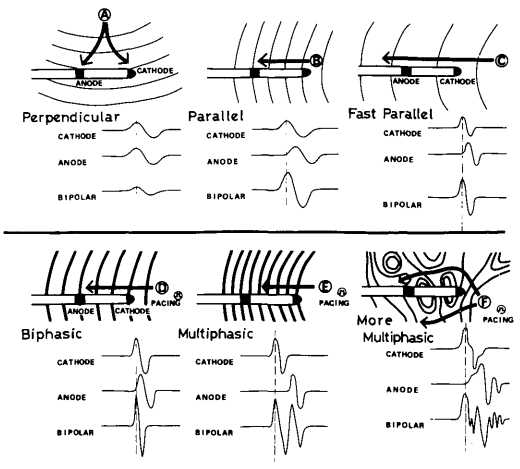


Figure 7 The schematic illustrations of the relation between the deflection waveform of bipolar electrode and the excitation wavefront. The deflection waveform of cathode and that of anode sums into the deflection waveform of bipolar electrode. The slow velocity of wavefront makes a round and small deflection waveform, but the fast velocity of wavefront makes a peaked and large deflection waveform. Moreover, the excitation wavefront during pacing stimulation produces a larger deflection waveform contributing to the high activation of myocardium than that in sinus rhythm.
Upper: In sinus rhythm.
Perpendicular (A), Parallel (B), Fast parallel (C) represent the direction or velocity of wavefront, and the waveform deflection to the bipolar electrode.
Lower: During pacing stimulation.
Biphasic (D), Multiphasic (E), More Multiphasic (F) represent the pattern of electrogram waveform on the bipolar electorde.

signal during pacing stimuli should be considered to be a different phenomenon from that during sinus rhythm. We hypothesized the following phenomenon during pacing (Figure 7-D to F):

The action potential of wavefront during pacing stimuli may become larger in amplitude than that of wavefront from the sinus node, since the pacing stimuli elevates the maximum rate of rise of action potential (dV/dt max)[17]. Consequently, the bipolar signal during pacing may be larger and steeper biphasic deflection than that before pacing (Figure 7-D). In our results, during both single extrastimulation and rapid pacing, both the HC and LC power area in patients with induction of Af were greater in amplitude than those in patients without induction of Af. These results might be mainly caused by the action potential of wavefront. These results suggested that the atrial myocardium in patients with induction of Af might have more sensitive to the pacing stimuli or larger volume in myocardial mass with rapid rise of action potential than that in patients without induction of Af, around the region of the high-lateral right atrium.

Furthermore, when the cycle length of rapid pacing is shortened, the velocity of wavefront may become slower and slower in conduction[17,18]. The more slower wavefront may cause the bipolar signal to become the larger and multiphasic waveform (Figure 7-E). If the wavefront become slower in conduction partially and inhomogeneously, the bipolar signal may be larger in amplitude and more multiphasic waveform (Figure 7-F). This hypothesis may be explained from our result that the HC power area during rapid pacing was markedly and stepwisely increased followed by the decrease in the cycle length shorter than 333 msec (Figure 5, upper). Probably, when the cycle length of rapid pacing was shortened step by step, the wavefront from the pacing site might become slower in conduction partially and inhomogeneously, and might become distorted. The distorted wavefront from the mid-lateral right atrium of pacing site passed through the high-lateral right atrium. Consequently, the enlarged and multiphasic peaked waveform on the bipolar electrode might cause the greater amplitude of the HC power area, whenever Af originated. Generally, the power spectrum of intra-atrial electrogram during regular tachycardia was greater in amplitude of both higher- and

lower-component of electrogram than that during the sinus rhythm, and especially the higher-frequency component significantly increased[19].

Clinical implication : In this study, the patient with higher incidence of pacing-induced Af had the shorter AERP and the greater amplitude of both the higher- and lower-frequency component of atrial electrogram around the high-lateral right atrium. In the clinical field, the pathologic or old-aged hearts have a tendency to associate the Af. These hearts have a lower myocardial activity or lesser volume of atrial myocardial mass due to the myocardial fibrosis. Therefore, there was the difference of background condition in the atrium between these hearts and the patients in our study. Probably, we thought that the different promotive factors of pacing-induced Af from the clinical Af might cause the non-physiologic phenomenon on background condition in the atrium.

Limitations : Some of the following problems should be considered to analyze the change in waveforms during the pacing; prolonged latency, prolonged duration of the deflection, changing afterpotential, hyperkinetic or dysynchronous wall motion, augmentation of far-field R wave, and so on[13]. In addition, rate-dependent multiple wavefront from multicentric pacemaker origin should be considered in mind, when the analysis was performed around the sinus node[20]. Although we wish to avoid all of these problems as much as possible, the system used have some limitations of hardware and software purelification.

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