Original Article

Conduction Velocity around the Tricuspid Valve Annulus during Typical Atrial Flutter by Electro-anatomic Mapping System

Akira Sawa MD, Akihiko Shimizu MD, Takeshi Ueyama MD, Yasuhiro Yoshiga MD, Shinsuke Suzuki MD, Naoki Sugi MD, Masunori Matsuaki MD

Department of Cardiovascular Medicine, Yamaguchi University Graduate School of Medicine

Objective: Conduction velocity around the tricuspid valve annulus (TA) during typical atrial flutter (AFL) has been shown to be slowest in the inferior vena cava-tricuspid valve (IVC-TV) isthmus when compared to the septal or free wall segments of the TA. We investigated the conduction velocity in IVC-TV isthmus, dividing into three areas. Methods: We evaluated conduction velocity around the TA during typical AFL in 10 patients, using an electro-anatomic mapping system (CARTO™). Conduction velocity was calculated at six areas around the TA including the septal wall, upper wall, lateral wall, and isthmus wall, which was further divided into three areas, lateral isthmus, mid isthmus, and septal isthmus. Results: Conduction velocity around the TA during typical AFL was slowest in the IVC-TV isthmus. Further, conduction velocities (m/sec) in the mid isthmus (0.44 ± 0.17) and septal isthmus (0.45 ± 0.22) were significantly slower ($p < 0.05$) than that in the upper wall (0.67 ± 0.26). Conclusions: The relatively slower conduction in IVC-TV isthmus resulted from the relatively slower conduction in the area from mid to septal isthmus.
(J Arrhythmia 2006; 22: 31–36)

Key words: Conduction velocity, Atrial flutter, Electro-anatomic mapping system

Introduction

Atrial flutter (AFL) has been defined traditionally as an atrial-tachycardia with a rate above 240 bpm and a continuously waving baseline. However, electrophysiologic studies (EPS) have put in evidence the inadequacy of current classifications of atrial tachycardias and AFL, based only the ECG pattern.[1] The orifices of the superior vena cava (SVC) and inferior vena cava (IVC), linked by the Crista terminalis thus constitute the posterior obstacle and the Tricuspid Annulus (TA), the anterior obstacle that make the virtual ring of myocardium supporting reentry in typical AFL.[2] Typical AFL activation turns in a ring of muscle bounded anterior by the TA and posterior by the openings of both vena cavae and the line of functional block in the posterior wall of the right atrium (RA). The IVC-TV isthmus (CTI) is an essential part of the typical AFL circuit, and the isthmus has been identified as an area of the slow conduction.[3–6]

Some authors have already found that conduction

Received 10, October, 2005: accepted in final form 23, January, 2006.
Address for correspondence: Akira Sawa MD, Department of Cardiovascular Medicine, Yamaguchi University Graduate School of Medicine 1-1-1 Minami-kogushi, Ube, Yamaguchi, Japan 755-8505. TEL: 0836-22-2248 FAX: 0836-22-2246
E-mail: firstrebirth@yahoo.co.jp

through the isthmus was slower than that though the anterior RA wall.[7] But activation of the low RA usually covers less than 30% of the cycle, far from the 70–80% in ventricular tachycardia (VT), and conduction is modestly slower and fragmented electrograms are not present before radiofrequency application. Up to now, the multi-polar catheter, for example the HALO 20-pole catheter, had been used to identify AFL activation around the TA. A better understanding of the electrophysiologic substrate of typical AFL is of scientific interest, particularly the precise location of the area of slow conduction in the reentrant circuit.

In recent years electro-anatomic mapping systems have come to be used frequently for EPS, and it is useful for the identification of a complex tachyarrhythmia circuit. One such system, the CARTO electro-anatomic mapping system, produces a three dimensional, high-resolution anatomic activation map of the entire AFL circuit in the right atrium.[8–10] Electro-anatomic mapping systems allow the distance and the time difference can be measured, and thus the conduction velocity can be calculated.

In the present study, we investigated the conduction velocity around the TA in typical AFL patients, especially focusing in greater detail on the isthmus, using the electro-anatomic mapping system.

Methods

1) Patients characteristics

The study population consisted of 10 patients (7 men and 3 women, age 57 ± 13 years) with typical atrial flutter ECG (**Table 1**). By electro-cardiographic criteria, atrial flutter was classical in appearance (saw-toothed pattern in inferior leads) in all patients. They were referred for radiofrequency catheter ablation (RFCA). Five patients had a history of paradoxical atrial fibrillation. Six patients had cardiovascular diseases, including four with hypertension, one with valvular disease and one with coronary disease.

2) Electrophysiologic study

Informed written consent was obtained from all patients. As described previously, all anti-arrhythmia drugs were discontinued for at least five half-lives before the study.[11] In all patients, a 7-French 20-pole, deflectable Halo catheter with 10-mm paired spacing was positioned around the TA to record RA activation in the high septal wall, roof, lateral wall, and CTI simultaneously. A 5-French, deflectable, 10-pole catheter with 2-mm interelectrode distance and 5-mm space between each electrode pair also was inserted into the coronary sinus via the subclavian vein. The position of the proximal electrode pair was at the ostium of the coronary sinus. 5-french multi-polar catheters were placed at high right atrium, His bundle, and right ventricular apex. A 8-French sheath (Preface Biosense-Webster, Johnson & Johnson, USA) placed in the right femoral vein was used to introduce the electro-anatomic mapping catheter. A 7-French, 4-mm tip electrode mapping/ablation catheter (NAVI-STAR Biosense-Webster, Johnson & Johnson, USA) was advanced into the right atrium. Three-dimensional atrial mapping was started in sinus rhythm in most of the patients. If spontaneous AFL was found at the onset of the study, Halo catheter mapping, and electro-anatomic mapping were performed to investigate the reentrant circuit. When the study found the presence of typical AFL mapping was performed during the AFL. If sinus rhythm was present at baseline, low RA or coronary sinus pacing until 2:1 atrial capture was preformed to induce AFL. When AFL was induced a new map was started, initially using the anatomical information obtained during sinus rhythm. Then, the right atrial geometry was rewritten at a different point from that during sinus rhythm. All stimuli were delivered through an external stimulator at a 2-msec pulse width at twice the diastolic threshold.

3) Electroanatomic mapping system

The CARTO™ nonfluoroscopic electro-anatomic mapping and navigation system (Biosense-Webster, Johnson & Johnson, USA) has been recently described elsewhere. A 4-mm tip catheter (NAVI-STAR,

Table 1 Clinical characteristics of patients.

Pt	Age (year)	Sex (M/F)	Diagnosis (Excluding AFL)	AFLCL (msec)
1	54	M	Coronary disease, HT	245
2	74	M	PAF, HT	290
3	76	M	None	285
4	70	M	None	270
5	55	M	PAF, Valvular disease	255
6	52	M	HT	285
7	54	M	PAF, HT	235
8	48	F	LV dysfunction	340
9	59	F	PAF	250
10	31	F	PAF	240
Mean ± SD	57 ± 13			269 ± 32

AFL = atrial flutter, PAF = paroxysmal atrial fibrillation, HT = hypertension, LV = Left ventricular, AFLCL = atrial flutter cycle length.

Biosense-Webster, Johnson & Johnson, USA) was used for the mapping and ablation. Because of its positional and morphological stability, the electrogram recorded by the coronary sinus catheter was chosen to be the reference for local activation time. The three-dimensional geometry of the chamber is reconstructed in real time with electrophysiologic information, which is color coded and superimposed on the anatomic map. The right atrium was plotted by dragging the mapping catheter over the endocardium. Mapping was complete when all lesions of the right atrium had been systematically sampled and when a sufficient density of points had been acquired to determine the circuit during AFL.

4) Catheter ablation

RFCA of the CTI was performed by using with a 4-mm tip catheter (NAVI-STAR, Biosense-Webster, Johnson & Johnson, USA) during AFL or pacing from the proximal coronary sinus during sinus rhythm.

The preset temperature was 55–60 degrees and the preset duration of each RF was 60 seconds. RFCA application was performed to create linear lesions in the CTI. Successful ablation was defined as achievement of bidirectional isthmus and gap conduction block without induction of typical AFL.

5) Definitions

Typical AFL was defined as a macro-reentrant atrial tachycardia circulating around the TA and using the CTI. Counter-clockwise (CCW) typical AFL was defined as a typical atrial flutter with a descending activation sequence in the free wall and an ascending sequence in the septal wall. Clockwise (CW) typical AFL was defined as a typical atrial flutter with a descending activation sequence in the septal wall and an ascending sequence in the free wall. Scar area was defined from an electrophysiological standpoint as an electrically silent area with an EGM amplitude of <0.04 mV that displays no distinguishable or repetitive EGM patterns.

6) Management after ablation and follow up

After ablation patients were monitored for 48 hours by telemetry. Transthoracic echocardiography was preformed by the end of the same day after ablation.

7) Measurement

The RA, based on the anatomical map, was observed from the left anterior oblique projection, and the screen was set so that the TA might come to the front only in the side of rotation. A perpendicular line initially was dropped from the picture, then two lines that intersected this line by 45 degrees were set (**Figure 1a**).

The area surrounding the TA was divided into four with borders at these two orthogonal lines. These divided areas were defined as the following. The one

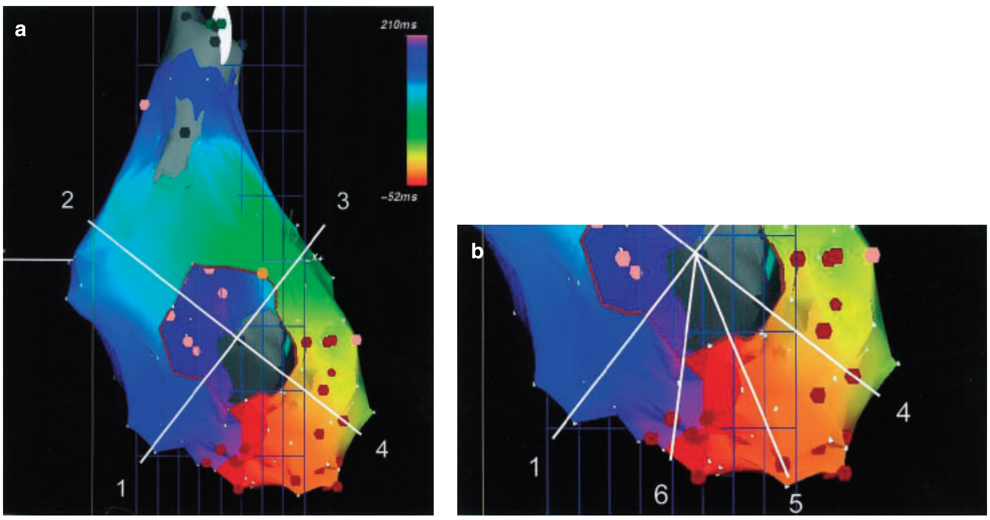

Figure 1

a: Right atrium was observed from the left anterior oblique projection, the screen was set so that the TA might come to the front only in the side of rotation. The 4 areas surrounding on the TA was divided into four bordering these two orthogonal lines, and these were defined as lateral wall (1-2), upper wall (2-3), septal wall (3-4), and isthmus (4-1).

b: The isthmus area was divided into three computation areas by equiangular degrees, and these were defined as septal isthmus (4-5), mid isthmus (5-6), and lateral isthmus (6-1).

including the CTI (anatomical isthmus) was defined as the isthmus area, and from there in a clockwise direction as lateral wall, upper wall, and septal wall. The percentage of conduction time in the area of isthmus to the cycle length of AFL was calculated. The isthmus area was divided into three computation areas, and these were defined as septal isthmus, mid isthmus, and lateral isthmus (**Figure 1b**). These six areas around the TA were set as stated above. The pattern of the excitement conduction was confirmed in the activation map. Two adjoining points (separated by a distance of 5 mm to 20 mm) were set to the vicinity of the center in these areas within 15 mm from the TA ring and the two points were set at the direction of the line between two points as parallel as possible to the TA ring.

8) Distance and conduction velocity measurement

Conduction velocity was calculated as the ratio of the distance between the two points and the difference in activation time between these two points. This distance of two points was assumed to be 20 mm from 5 mm this time (<5 mm, to minimize the error caused by endocardial curvature).

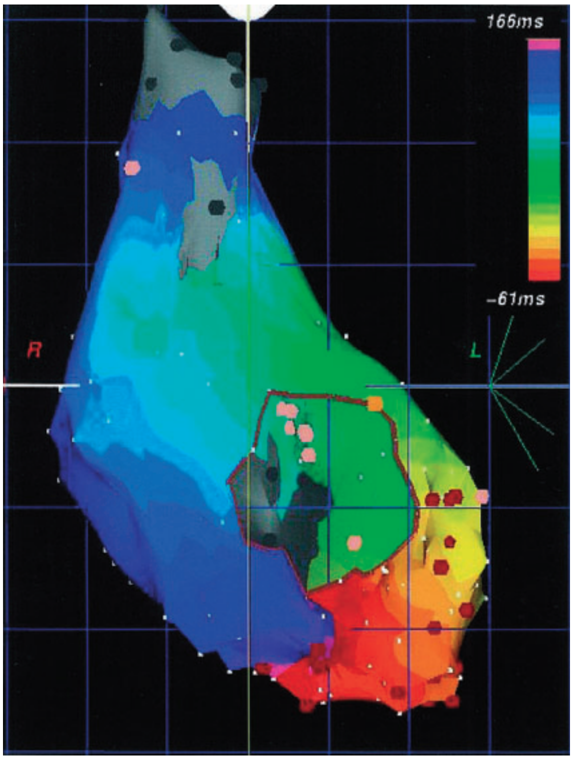

Figure 2 Right atrial endocardial electroanatomic map (Isochronal map: isochronal step = 10 msec) in a patient (#4) with typical AFL.
Note the counterclockwise flutter activation sequence around the tricuspid annulus, indicating the common or typical form of type1 AFL. The colors represent early (red) to late (purple) activation, referenced to the proximal coronary sinus electrode. Red dots indicate ablation points.

9) Statistical analysis

Continuous values are expressed as mean ± standard deviation. One-way analysis of variance (ANOVA) with Bonferoni–Dunn method for pair multiple comparisons was used to determine the statistical significance of the difference in mean values for conduction velocity between the various areas around the TA. A P-value of 0.05 or less was considered significant.

Results

1) Mapping results

In the all 10 patients, complete electro-anatomic maps were obtained for typical AFL (mean cycle length = 269 ± 32 msec, range 220–340 msec). All patients had CCW activation of RA limited anteriorly by the TA, an example of which is shown in **Figure 2**. Pacing from the CTI during atrial flutter showed concealed entrainment with the post-pacing-interval equal to flutter cycle length in all patients. During CCW atrial flutter, the leading edge of the activation wavefront emerged from the low postero-septal region, traveled up the septal and posterior wall, reached the roof, spread down the antero-lateral wall, and entered the CTI. The direction of the activation wavefront demonstrated by electro-anatomic mapping system was consistent with the activation sequence recorded by the HALO catheter in all patients. The percentage of conduction time in the area of isthmus to the cycle length of AFL (269 ± 32 msec) was 34 ± 5%.

2) Analysis of regional conduction velocities

In all patients, the conduction velocities in the six areas were measured. Regarding the distance of the two points in these six areas, the longest distance was observed in the septal wall, followed by the upper wall, lateral wall, lateral isthmus, and septal isthmus, while the nearest distance was found in the mid isthmus (12.5 ± 6.3 mm, 11.9 ± 4.1 mm, 10.3 ± 3.6 mm, 9.6 ± 4.3 mm, 8.8 ± 3.7 mm and 8.0 ± 4.5 mm; N.S), no significant difference was seen in these six areas. Of the six areas around TA initially analyzed (**Table 2**), mean conduction velocity (m/sec) was significantly slower ($p < 0.05$) in the mid isthmus (0.44 ± 0.17) and septal isthmus (0.45 ± 0.22), than in the upper wall (0.67 ± 0.26). Mean conduction velocity tended to be slow ($p = 0.06$) in the mid isthmus and septal isthmus compared with

Table 2 Conduction velocity (m/sec) in 6 areas around the TA during AFL.

Pt	Septal wall	Upper wall	Lateral wall	CTI		
				Lateral	Mid	Septal
1	0.37	0.35	0.58	0.35	0.30	0.26
2	0.88	0.53	0.80	0.38	0.51	0.29
3	1.00	0.58	0.72	0.62	0.58	0.69
4	0.40	1.11	0.58	0.60	0.35	0.72
5	0.86	0.48	0.54	0.99	0.27	0.50
6	0.64	0.50	0.66	0.51	0.47	0.49
7	0.32	0.59	0.49	0.30	0.51	0.73
8	0.44	0.61	0.46	0.92	0.66	0.13
9	0.64	1.09	0.51	0.43	0.64	0.42
10	0.57	0.86	0.93	0.62	0.14	0.23
Mean ± SD	0.61 ± 0.24	0.67 ± 0.26	0.63 ± 0.15	0.57 ± 0.23	0.44 ± 0.17*#	0.45 ± 0.22*#

Septal wall = right atrial septal wall, upper wall = right atrial upper wall, lateral wall = right atrial lateral wall, CTI = IVC-TV isthmus, lateral = lateral isthmus, mid = mid isthmus, septal = septal isthmus.
* = $P < 0.05$ comparing mid isthmus and septal isthmus to upper wall.
# = $P = 0.06$ comparing conduction velocity in all other segments.

all other segments. In these patients, lowest conduction velocities in each case were in isthmus area (5 patients in mid isthmus, 4 patients in septal isthmus, 1 patient in lateral isthmus).

Discussion

This study has shown that the medial mid-isthmus and septal isthmus are the slowest-conducting areas in the reentrant circuit in typical AFL in humans. Although the patient numbers studied were small, and therefore this cannot be substantiated by this study, it is consistent with previous studies. Previous studies, the earliest of which used pacing entrainment and multi-electrode catheter mapping technique, have consistently demonstrated that the TV-IVC isthmus is an area of slow conduction in the AFL reentrant circuit.[12,13)]

Tai et al. measured isthmus conduction velocity during pacing (but not AFL) by dividing the bipolar electrogram intervals by interelectrode spacing and found that isthmus conduction velocities were lower than in our study (0.336 ± 0.045 m/sec) and were lower in the mid isthmus compared with the lateral isthmus.[14)] This may be explained because distance traveled across a complex curved surface is likely to underestimated the distance between electrodes on a straight catheter bridging this distance. Shilling et al. measured isthmus conduction velocity during AFL by noncontact mapping system (Ensite™) and found that isthmus conduction velocities were higher than in our study (0.74 ± 0.36 m/sec).[15)]

Hassankhani et al. measured conduction velocity during AFL around the TA by electro-anatomic mapping system (CARTO™) and found that inferior septum and medial isthmus were the areas of slowest conduction in typical AFL,[16)] which findings were similar to our results.

In isolated pig and dog hearts, Hocini et al. reported that fibers were parallel to the TA in the posterior part of Triangle-of-Koch. In the midjunctional area, the direction of the fibers changed to an orientation perpendicular to the TA. During stimulation from posterior and anterior sites, activation proceeded parallel to the TA at a high conduction velocity (0.5 to 0.6 m/sec). During stimulation from sites near the coronary sinus, a narrow zone of slow conduction occurred in the posterior part of the Triangle-of-Koch where activation proceeded perpendicular to the fiber orientation. Above and below this zone, conduction was fast and parallel to the annulus.[17)] A correlation was observed between parallel versus perpendicular fiber orientation with respect to the TA and fast versus slow conduction in the triangle-of-Koch. These observations of myocardial fiber anisotropy in the triangle-of-Koch[17–19)] are consistent with the observations from this study and may account for the fact that the slowest conduction in the AFL circuit is in areas of the septal isthmus.

In this study, typical AFL with relatively slower conduction in IVC-TV isthmus resulted from the relatively slower conduction in the area from mid to septal isthmus. This relatively slower conduction area is supposed be related to the onset and maintenance of AFL. However, further examination will be necessary to prove this issue.

Study Limitation

In measuring conduction velocity distance, measurements were calculated using a series of short straight-line distance that an approximation of the true curved surface of the RA and are therefore subject to error. The closer the distance between two points, the less error is introduced in calculating the distance between two points on a sphere, given that a line is the asymptotic measure of two points with closest proximity on a curved surface. It is enumerated that neither the direction where the wave side travels nor the set direction of these two points are completely corresponding.

Conclusion

Electroanatomic mapping system of the right atrium in patients with typical AFL reveals that the relatively slower conduction in IVC-TV isthmus resulted from relatively slower conduction in the area from the mid to septal isthmus, and the mid isthmus is the region of slowest conduction around the TA.

References

1) Saoudi N, Cosio F, Waldo A, Ches S-A, Iesaka Y, Lesh M, Saksena S, Salermo J, Schoels W: Classification of atrial flutter and regular atrial tachycardia according to electrophysiologic mechanism and anatomic basis: A statement from a joint expert group from the working group of arrhythmias of European Society of Cardiology and the North American Society of Pacing and Electrophysiology. J Cardiovasc Electrophysiol 2001; 12: 852–866
2) Cosio FG, Arribas F, Lopez Gil M, Palacios J: Atrial flutter mapping and ablation. I. Studying atrial flutter flutter mechanisms by mapping and entrainment. PACE 1996; 19: 841–853
3) Waldo AL: Pathogenesis of atrial flutter. J Cardiovasc Electrophysiol 1998; 9: S18–25
4) Daoud EG, Morady F: Pathophysiology of atrial flutter. Annu Rev Med 1998; 49: 77–83
5) Disertori M, Inama G, Vergara G, Guarnerio M, Del Favero A, Fulanello F: Evidence of a reentry circuit in the common type of atrial flutter in man. Circulation 1983; 67: 434–440
6) Chauchemez B, Haissaguerre M, Fisher B, Thomas O, Clementy J, Coumel P: Electrophysiological effects of catheter ablation of inferior vena cava-tricuspid annulus isthmus in common atrial flutter. Circulation 1996; 93: 284–294
7) Tai C-T, Chen S-A, Chiang C-E, Lee S-H, Ueng K-C, Wen Z-C, Huang J-L, Chen Y-J, Yu W-C, Feng A-N, Chiou C-W, Chang M-S: Characterization of low right atrial isthmus as the slow conduction zone and pharmacological target in typical atrial flutter. Circulation 1997; 96: 2601–2611
8) Nakagawa H, Jackman WM: Use of a three-demensional, nonfluoroscopic mapping system for catheter ablation of typical atrial flutter. Pacing Clin Electrophysiol 1998; 21: 1279–1286
9) Feld GK: Evolution of diagnostic and interventional cardiac electrophysiology: A brief historical review. Am J Cardiol 1999; 84: 115R–124R
10) Gepstein L, Hayam G, Benttaim SA: A novel method for non fluoroscopic catheter-based electroanatomical mapping of the heart: In vitro and vivo accuracy results. Circulation 1997; 95: 1611–1622
11) Kakugawa H, Shimizu A, Yamagata T, Esato M, Ueyama T, Yoshiga Y, Kanemoto M, Matsuzaki M: Decrease in the spatial dispersion at the termination of atrial fibrillation by intravenous cibenzoline. Circ J 2003; 67: 810–815
12) Feld GK, Mollerus M, Brigersdotter-Green U, Fujimura O, Bahnson T, Boyce K, Rahme M: Conduction velocity in the tricuspid valvu-inferior vena cava isthmus is slower in patients with type1 atrial flutter compared to those without a history of atrial flutter. J Cardiovasc Electrophysiol 1997; 8: 1338–1348
13) Shah DC, Jais P, Haissaguerre M, Chouairi S, Takahashi A, Hocini M, Garrigue S, Clementy J: Three-dimensional mapping of the common atrial flutter circuit in the right atrium. Circulation 1997; 96: 3904–3912
14) Tai C, Chen S, Chiang C, Lee S, Ueng K, Wen Z, Huang J, Chen Y, Yu W, Feng A, Chiou C, Chang M: Characterization of low right atrial isthmus as the slow conduction zone and pharmacological target in typical atrial flutter. Circulation 1997; 96: 2601–2611
15) Shilling RJ, Peters NS, Goldberger J, Kadish AH, Davies DW: Characterization of the anatomy and conduction velocities of the human right atrial flutter circuit determined by noncontact mapping. J Am Coll Cardiol 2001; 38: 385–393
16) Hassankhani A, Yao B, Feld GK: Conduction velocity around the tricuspid valve annulus during type 1 atrial flutter: Defining the location of areas of slow conduction by three-dimensional electroanatomical mapping. J Interv Card Electrophysiol 2003; 8: 121–127
17) Hocini M, Loh P, Ho SY, Sanches-Quintana D, Thibault B, de Bakker JM, Janse MJ: Anisotropic conduction in the triangle of Koch of mammalian hearts: Electrophysiologic and anatomic correlations. J Am Coll Cardiol 1998; 31: 629–636
18) Wang K, Ho SY, Gibson DG, Anderson RH: Architecture of atrial musculature in humans. Br Heart J 1995; 73: 559–565
19) Nakagawa H, Lazzara R, Khastgir T, Beckman KJ, McClelland JH, Imai S, Pitha JV, Becker AE, Arruda M, Gonzales MD, Widman LE, Neuhauser J, Wang X, Calame JD, Goudeau MD, Jackman WM: Role of the tricuspid annulus and the Eustachian valve/ridge on atrial flutter: Relevance to catheter ablation of the septal isthmus and a new technique for rapid identification of ablation success. Circulation 1996; 94: 407–424