Effects of Right Coronary Artery PTCA on Variables of P-Wave Signal Averaged Electrocardiogram

Marco Budeus, M.D., Marcus Hennersdorf, M.D., Stefan Dierkes, M.D., Michael Preik, M.D., Matthias P. Heintzen, M.D., Malte Kelm, M.D., and Christian Perings, M.D.

From the Department of Cardiology, Pneumology and Angiology, Medical Clinic and Polyclinic B Heinrich-Heine-University, Duesseldorf, Germany

Background: P-wave signal averaged ECG has been used to detect atrial late potentials that were found in paroxysmal atrial fibrillation. Ischemia is supposed to trigger ventricular late potentials, which indicate an elevated risk for ventricular tachycardia. Preexistent ventricular late potentials measured by ventricular signal averaged ECG is supposed to be eliminated by successful PTCA.

Methods: We examined the incidence of atrial late potentials in patients with a proximal stenosis of the right coronary artery and new onset of atrial fibrillation. Furthermore, we investigated the anti-ischemic effect of a successful percutaneous transluminal coronary angioplasty (PTCA) of the right coronary artery. P-wave signal averaged ECG from 23 patients who had a PTCA of the right coronary artery (group A) were compared to age, sex, and disease-matched control subjects (group B) one day before, one day after, and one month after PTCA.

Results: A new appearance of paroxysmal atrial fibrillation was presented in eight patients before PTCA (group A1) of group A. Patients with a stenosis of the right coronary artery had a significantly higher incidence of supraventricular extrasystoles in a 24-hour-Holter ECG (131.1 ± 45.4 vs 17.1 ± 18.9 , $P < 0.0002$). The duration of the filtered P wave was longer (124.8 ± 11.9 vs 118.5 ± 10.1 ms, $P < 0.04$) and the root mean square of the last 20 ms (RMS 20) was significantly lower in group A than in group B (2.87 ± 1.09 vs 3.97 ± 1.12 μ V, $P < 0.01$). A successful PTCA caused an increase in RMS 20 (2.87 ± 1.11 vs 4.19 ± 1.19 μ V, $P < 0.02$) and a decrease in filtered P-wave duration (124.8 ± 11.9 vs 118.4 ± 10.4 ms, $P < 0.04$). Preexistent atrial late potentials were found among 15 patients before PTCA. After successful PTCA only 3 out of 15 patients were affected ($P < 0.0004$) after one day, as well as after one month. All patients with a history of atrial fibrillation did not suffer from an arrhythmic recurrence within the following six months after successful PTCA.

Conclusion: A stenosis of the right coronary artery is associated with atrial late potentials. A successful PTCA of the right coronary artery eliminates preexistent atrial late potentials and may reduce the risk of atrial fibrillation.

A.N.E. 2003;8(2):150-156

P-wave signal averaged ECG; paroxysmal atrial fibrillation; PTCA; atrial late potentials

Atrial fibrillation is the most common supraventricular cardiac arrhythmia in human beings affecting 0.4% of the population.¹ The Framingham study showed an increased incidence of atrial fibrillation with advancing age.² There are many risk factors for atrial fibrillation such as coronary heart disease, hypertension, rheumatic valve disease, and heart failure.³

Atrial late potentials have been described using P-wave signal averaged ECG in patients with paroxysmal atrial fibrillation, after coronary artery bypass surgery or recurrence of atrial fibrillation after

successful cardioversion.⁴⁻⁸ The high incidence of atrial late potentials might be the reason for the occurrence of atrial fibrillation in paroxysmal atrial fibrillation, after coronary artery bypass surgery or recurrence of atrial fibrillation after successful cardioversion.⁴⁻⁸

Coronary heart disease causes cardiac arrhythmias as ventricular tachycardia or atrial fibrillation.⁹⁻¹⁸ Percutaneous transluminal coronary angioplasty (PTCA) eliminates preexistent ventricular late potentials, which were indicators of ventricular tachycardia.⁹⁻¹² There is a correlation between

stenosis of the right coronary artery and the occurrence of atrial fibrillation,^{13–18} which might be caused by a prolongation of filtered P-wave duration due to a stenosis of the right coronary artery (RCA)¹⁹ because of the main supply of the atria from the RCA.²⁰ We hypothesize that a successful PTCA of an RCA stenosis reduces the incidence of atrial late potentials that represents an arrhythmic effect of ischemia. The measurements were performed among patients who had a proximal stenosis of a dominant RCA before and after a PTCA.

METHODS

The study population consisted of 46 patients: 23 patients (group A) with a proximal stenosis of 70–99% in the RCA and 23 age, sex, and disease-matched control patients (group B) without a stenosis of the RCA. Patients of group A had angina pectoris (18 patients) or an ischemic proof of the stenosis (five patients) which was delivered by thallium myocardial scintigraphy. A stenosis was defined as narrowing of greater than or equal to 50% of lumen diameter. In group A eight patients (group A1) had a new appearance of paroxysmal atrial fibrillation that was documented by Holter ECG one to three times before. All patients underwent coronary angiography. The baseline clinical characteristics are given in Table 1.

Patients did not take part in this study if they had a myocardial infarction, left atrium diameter > 40 mm, unstable angina, myocarditis, or hyperthyroidism. Other exclusive criteria were pregnancy, known pulmonary hypertension, previous electrical cardioversion, mitral valve prolaps, valvular heart disease, aortocoronary bypass operation class Ic-therapy within the last three months. Patients neither have a vessel obliteration nor have visible collateral vessels.

In this study, the patients were examined before, one day and one month after PTCA with the P-wave signal averaged ECG in our hospital. After an interval of six months a second coronary angiography was performed. Only patients without restenosis were included in this study.

Atrial late potentials were defined as root mean square of the last 20 ms (RMS 20) $\leq 3.5 \mu\text{V}$ and duration of the filtered P wave over 120 ms using P-wave signal averaged ECG.⁴

Acquisition and Analysis of the P-Wave Signal Averaged ECG

The P-wave signal averaged ECG was recorded from an X, Y, and Z lead system (Corazonix Corporation, Predictor) using the time-domain analysis. An orthogonal lead arrangement identical to that used for conventional QRS signal averaging was

Table 1. Patients Characteristics

Characteristics	Group A	Group A1	Group A2	Group B
Number	23	8	15	23
Males/Females	16/7	6/2	10/5	16/7
Age (years)	64.2 $\pm$ 9.2	65.1 $\pm$ 7.1	63.5 $\pm$ 9.7	64.2 $\pm$ 9.2
Heart rate (beats/min)	68.3 $\pm$ 11.4	66.4 $\pm$ 8.1	69.1 $\pm$ 6.5	66.5 $\pm$ 9.5
Supraventricular extrasystoles/24 hours	131.1 $\pm$ 45.4 P < 0.0002 ^a	162.7 $\pm$ 40.4 P < 0.0001 ^a P < 0.003 ^b	109.1 $\pm$ 54.9 P < 0.0004 ^a	17.1 $\pm$ 18.9
Ejection fraction (%)	58.8 $\pm$ 9.6	59.1 $\pm$ 6.9	58.2 $\pm$ 9.9	62.9 $\pm$ 7.7
Coronary heart disease (n)	23	8	15	23
Right coronary artery (n)	23	8	15	0
Left anterior descending artery (n)	14 (61%)	5 (63%)	8 (53%)	15 (65%)
Left circumflex artery (n)	8 (35%)	2 (25%)	6 (40%)	11 (48%)
LA (mm)	38.2 $\pm$ 1.5	38.8 $\pm$ 1.1	38.1 $\pm$ 1.4	38.0 $\pm$ 1.5
Diabetes mellitus (n)	3 (13%)	1 (13%)	2 (13%)	3 (13%)
Hypertension (n)	16 (70%)	6 (75%)	10 (67%)	16 (70%)
Sotalol (n)	2 (9%)	2 (25%)	0	0
Verapamil (n)	3 (13%)	2 (25%)	1 (7%)	0
β -blockers (n)	17 (74%)	4 (50%)	13 (87%)	17 (74%)

Abbreviations: ECG: electrocardiogram, Group A1: patients with paroxysmal atrial fibrillation, Group A2: patients with sinus rhythm, LA: left atrium diameter, RMS 20: root mean square of the last 20 ms of the P wave,^a: in relation to group B,^b: in relation to group A2.

used. The P wave was retained as a trigger of the averaging process with the fiducial point shifted to the extreme right side of the 600 ms window to expose the P wave and the PQ interval. The signals were digitized at a frequency of 1000 samples/s with 16-bit accuracy.

After eliminating supraventricular and ventricular extrasystole signals from each lead were amplified and filtered between 40 and 250 Hz by using a bidirectional filter. A P-wave template was selected by detection of the first peak of the filtered P wave, and P-wave complexes not matching the template with a 99% correlation coefficient were automatically rejected to minimize P-wave jitters. P waves were recorded until a noise end point of $0.5 \mu\text{V}$ was achieved in the PQ interval. For every measurement 500 beats were used to complete the signal averaging with a mean average of 512.4 ± 9.4 beats.

All data were stored in a floppy disk and analyzed with a computer and accompanying software. The P-wave complexes of filtered X, Y, and Z leads were combined to a vector magnitude square root ($X^2 + Y^2 + Z^2$).

The onset of the P-wave vector magnitude was defined as the first signal point that was ≥ 3 times the noise level and that was followed by three consecutive signal points that were > 3 times the noise level. The offset of the P-wave vector magnitude was defined as the last signal point that was ≥ 2 times the noise level and that was followed by three consecutive signal points that were not more > 2 times the noise level. The interval between the onset and offset points defined the P-wave duration of the vector magnitude.

Statistics

Patients were divided into a study group (group A) suffering from coronary artery disease with stenosis of the RCA and a control group (group B). The study group was subdivided into patients who had atrial fibrillation within the last six months (group A1) and those who never had atrial fibrillation before (group A2). All variables were compared between groups A and B and between groups A1 and A2. Data are presented as mean values $\pm$ SD. Statistical analysis was performed with the Mann-Whitney U test and chi-square test for baseline characteristics. Kruskal-Wallis analysis of variance (ANOVA) with post hoc Student *t*-test was used for values of P-wave signal

averaged ECG. A P value of ≤ 0.05 is considered significant.

RESULTS

There were no significant differences in heart rate, left atrial diameter, and left ventricular ejection fraction between the groups. Patients with a stenosis of the right coronary artery had a significantly higher incidence of supraventricular extrasystoles in a 24-hour-Holter ECG (131.1 ± 45.4 vs 17.1 ± 18.9 , $P < 0.0002$). The patients of group A1 also had a significantly higher incidence of supraventricular extrasystoles than those in group A2 (162.7 ± 4.4 vs 109.6 ± 54.9 , $P < 0.003$). The duration of the filtered P wave was longer (124.8 ± 11.9 vs 118.5 ± 10.1 , $P < 0.04$) and the root mean square of the last 20 ms (RMS 20) was lower in group A than in group B (2.87 ± 1.09 vs $3.97 \pm 1.12 \mu\text{V}$, $P < 0.01$). A successful PTCA caused an increase in RMS 20 (2.87 ± 1.09 vs $4.19 \pm 1.19 \mu\text{V}$, $P < 0.02$) after one day and after one month (2.87 ± 1.09 vs $4.12 \pm 1.01 \mu\text{V}$, $P < 0.02$). There was a decrease in filtered P-wave duration after one day (124.8 ± 11.9 vs 118.4 ± 10.4 ms, $P < 0.04$) as well as after one month (124.8 ± 11.9 vs 117.2 ± 9.5 ms, $P < 0.04$). A high correlation between the measurements after one day and one month could be observed for filtered P-wave duration ($r = 0.84$, $P < 0.0001$) and RMS 20 ($r = 0.73$, $P < 0.0001$). We found a correlation between the number of supraventricular extrasystoles and the duration of the filtered P wave ($r = 0.27$, $P < 0.05$). The left atrial size did not change before and one month after PTCA (38.2 ± 1.5 vs 38.4 ± 1.4 mm, $P = \text{ns}$) with a high correlation between both measurements ($r = 0.934$, $P < 0.0001$). Figure 1 depicted two original tracings of the signal averaged ECG in representative patients before and one month after PTCA. Table 2 summarizes the P-wave signal averaged ECG results.

There were no significant differences between the subjects of group A1 and A2 in filtered P-wave duration or RMS before and after PTCA. After successful PTCA, there were no significant differences in the variables of P-wave signal averaged ECG between group A and B.

The left ventricular end diastolic pressure did not change before and after PTCA (8.7 ± 5.2 vs 8.1 ± 5.9 mmHg, $P < 0.84$) or between groups A and B (8.7 ± 5.2 vs 8.3 ± 6.4 mmHg, $P < 0.62$). The antiarrhythmic therapy also did not change in the

A

B

Figure 1. Original tracings of P-wave SA-ECG of a patient before (A) and one month after (B) PTCA.

follow up of six months. All patients with a history of atrial fibrillation did not suffer from an arrhythmic recurrence within the following six months after successful PTCA.

Fukunami et al.⁴ defined atrial late potentials as $\text{RMS } 20 \leq 3.5 \mu\text{V}$ and $\text{P-wave duration } \geq 120 \text{ ms}$ at the same time. Using this definition 15 patients (65%) of group A had atrial late potentials before

Table 2. Variables of P-Wave Signal Averaged ECG

	Group A1	Group A2	Group A	Group B
P-wave duration (ms)	128.1 ± 11.3	123.3 ± 9.1	124.8 ± 11.9	118.5 ± 10.1
before PTCA				
1 day after PTCA	119.1 ± 10.9 ^a	117.5 ± 12.1 ^a	118.4 ± 10.4 ^a	
Δ P (ms)	5.7 ± 5.2	5.1 ± 5.1	5.3 ± 5.9	
1 month after PTCA	118.4 ± 9.8 ^a	117.8 ± 13.5 ^a	117.2 ± 9.5 ^a	
Δ P (ms)	6.1 ± 4.8	5.7 ± 5.3	5.9 ± 6.8	
RMS 20 (μV)				
before PTCA	2.71 ± 1.17 ^b	2.99 ± 1.22 ^b	2.87 ± 1.09*	3.97 ± 1.12
1 day after PTCA	3.95 ± 1.56 ^c	4.24 ± 1.38 ^c	4.19 ± 1.19 ^c	
Δ RMS (μV)	0.91 ± 1.64	0.89 ± 1.22	0.90 ± 1.72	
1 month after PTCA	4.01 ± 1.73 ^c	4.19 ± 1.53 ^c	4.12 ± 1.01 ^c	
Δ RMS (μV)	0.98 ± 1.95	0.99 ± 1.61	0.99 ± 1.72	

Abbreviations: Group A1: patients with paroxysmal atrial fibrillation, Group A2: patients with sinus rhythm, RMS 20: root mean square of the last 20 ms of the P wave, ^a: P < 0.04 in relation to before PTCA, ^b: P < 0.01 in relation to group B, ^c: P < 0.02 in relation to before PTCA, Δ P: difference of P-wave duration before and after PTCA, Δ RMS: difference of RMS 20 before and after PTCA.

PTCA. A successful PTCA caused a reduction of preexistent atrial late potentials to only three patients (P < 0.0004) in both measurements after PTCA. The result is shown in Figure 2. In six out of eight patients (75%) with an anamnesis of atrial fibrillation atrial late potentials could be detected before, and in only one patient (12%) after successful PTCA. In group A2 only 9 out of 15 patients (60%) had preexistent atrial late potentials. After a successful PTCA these late potentials were detected in only two patients (22%). We found more atrial late potentials (6/8 vs 9/15 patients) in subjects with a history of paroxysmal atrial fibrillation. Only in four patients (17%) of the control-group atrial late potentials were found. The results are shown in

Figure 3. The subjects in group A had a higher incidence of atrial late potentials than in group B (15/23 vs 4/23 patients, P < 0.0007).

DISCUSSION

Coronary artery disease is a main risk factor for atrial fibrillation.² This study had shown the feasibility to record atrial late potentials using a program for ventricular late potentials. The program was modified properly for the trigger point on the P wave and the prevalence of atrial late potentials due to a stenosis of the RCA. We excluded the influencing effects of enlarged left atrium,^{21,22} propafenone or flecainide,²³ or which extended the

Figure 2. Atrial late potentials of patients of group A before and after PTCA. (black = atrial late potentials)

Figure 3. Atrial late potentials of groups A1, A2, A, and B in percentages before and after PTCA. (black = atrial late potentials)

filtered P-wave duration. An effect of the antiarrhythmic therapy with sotalol,²⁴ β -blocker, or verapamil²⁵ on the filtered P-wave duration could be excluded. We observed a longer filtered P wave in the group with an important stenosis in the RCA. The stenosis was of some relevance because of symptoms of angina pectoris or an ischemic proof by thallium myocardial scintigraphy. Christiansen et al.¹⁹ also found a correlation between a longer filtered P-wave duration (3.4 ms, $P < 0.05$) and a stenosis in the RCA. This observation correlated with our results and demonstrated the influence of the RCA on the variables of the P-wave signal averaged ECG.

In our study the duration of the filtered P wave tends to be longer in patients with paroxysmal atrial fibrillation than in those with sinus rhythm (128.1 vs 123.3 ms, $P < 0.07$). A longer duration of the filtered P wave of 137.0 ms⁴ or 138.5 ms⁵ was detected in other studies with paroxysmal atrial fibrillation. The difference of a shorter duration of filtered P wave could have been caused by using a fewer number of patients, by using different noise levels, or by the method of defining the onset–offset of the P wave.

A stenosis of the RCA was associated with a prolongation of the filtered P wave and high incidence of preexistent atrial late potentials. There also is a high incidence of ischemic triggered preexistent ventricular late potentials, which were indicators of ventricular tachycardia.^{9–12} The measurement of ventricular or atrial late potentials depends on an averaging process of ventricular or atrial signals. The evidence of slow-fragmented conduction

in the myocardium is detected with signal averaged ECG.^{9–12,26–28} Slow-fragmented conduction in the atrial myocardium is a risk factor for atrial fibrillation.^{26–28} P-wave signal averaged ECG can detect delayed conduction within the atria, which is a reason for atrial fibrillation.²⁹ Therefore, a longer filtered P-wave duration and atrial potential is observed in high sensitivity and specificity in paroxysmal atrial fibrillation.^{4,5}

The main difference between patients with or without a history of atrial fibrillation was a higher incidence of supraventricular extrasystoles in a 24-hour-Holter-ECG (162.7 vs 109.1, $P < 0.003$) in our study. Patients without a stenosis of the RCA had a lower incidence of supraventricular extrasystoles (131.1 vs 17.1, $P < 0.0002$). Our results show that a stenosis of the RCA is associated with a higher incidence of supraventricular extrasystoles, which might be a risk factor for the occurrence of atrial fibrillation.³⁰ Atrial fibrillation is initiated by supraventricular extrasystoles arising in different regions of the atrium triggered either by coronary artery disease or hypertension.³⁰

The prolongation of the filtered P wave was reversible due to a successful PTCA of the RCA in our study. After PTCA there was a reduction of the incidence of preexistent atrial late potentials. PTCA can also eliminate ischemic triggered preexistent ventricular late potentials that were indicators of ventricular tachycardia.^{9–12} Thus, ischemia is the trigger of atrial and ventricular late potentials.

We conclude from these results that a stenosis of the RCA represents an increased risk of atrial fibrillation through the development of regional

conduction delay seen as atrial late potentials and higher incidence of supraventricular extrasystoles. A successful PTCA can protect these patients from developing atrial fibrillation. Thus, a larger prospective trial of the predictive power of atrial late potentials for the development of atrial arrhythmias is conducted.

Acknowledgments: *The authors thank Andrea Tüffers and Sabine Jacob for secretarial support and technical assistance; Cornelia Mohrs and Hannelore Esser for technical assistance.*

REFERENCES

- Ostrander LD Jr, Brandt RL, Kjelsberg, et al. Electrocardiographic findings among the adult population of a total nature community. *Circulation* 1965;31:888-98.
- Kannel WB, Abbott RD, Savage DD, et al. Epidemiologic features of chronic atrial fibrillation. The Framingham Study. *N Engl J Med* 1982;306:1018-22.
- Benjamin EJ, Levy D, Vaziri SM, et al. Independent risk factors for atrial fibrillation in a population-based cohort. The Framingham Heart Study. *JAMA* 1994;271:840-4.
- Fukunami M, Yamada T, Ohmori M, et al. Detection of patients at risk for paroxysmal atrial fibrillation during sinus rhythm by P-wave triggered signal averaged electrocardiogram. *Circulation* 1991;83:162-9.
- Kontoyannis DA, Sideris SA, Mouloupoulos SD. Atrial late potentials: Paroxysmal supraventricular tachycardia versus paroxysmal atrial fibrillation. *Int J Cardiol* 1993;41:147-52.
- Zaman AG, Archbold RA, Helft G, et al. Atrial fibrillation after coronary artery bypass surgery: A model of preoperative risk stratification. *Circulation* 2000;101:1403-8.
- Opolski G, Scislo P, Stanislawski J, et al. Detection of patients at risk for recurrence of atrial fibrillation after successful electrical cardioversion by signal averaged P-wave ECG. *Int J Cardiol* 1997;60:181-5.
- Yamagata T, Shimizu A, Ueyama T, et al. Assessment of right atrial mapping and P-wave triggered signal average in patients with paroxysmal atrial fibrillation. *J Electrocardiol* 1999;32:243-51.
- Steinbigler P, Haberl R, Steinbeck G. Ischemia-induced changes of the signal averaged electrocardiogram: Experimental investigation during percutaneous transluminal coronary angioplasty balloon-occluded coronary artery. *J Cardiovasc Electrophysiol* 1999;10:1316-22.
- Hermosillo AG, Horma ME, Zamora J, et al. Effects of delayed recanalization of occluded acute myocardial infarction-related artery using coronary angioplasty on late potentials. *Coron Artery Dis* 1995;6:169-77.
- Masui A, Tamura K, Iwasaka T, et al. The effect of successful angioplasty on variables of signal averaged electrocardiogram and ventricular wall motion in patients with first myocardial infarction. *Clin Cardiol* 1994;17:479-83.
- Berkalp B, Oral D, Pamir G, et al. Effects of percutaneous transluminal coronary angioplasty on late potentials and high frequency mid-QRS potentials. *Cardiology* 1994;85:216-21.
- Kyriaklidis M, Barbetseas J, Antonopoulos A, et al. Early atrial arrhythmias in acute myocardial infarction. *Chest* 1992;101:944-7.
- Mendes LA, Connelly GP, McKenney PA, et al. Right coronary artery stenosis: An independent predictor of atrial fibrillation after coronary artery bypass surgery. *J Am Coll Cardiol* 1995;25:198-202.
- Sakata K, Kurihara H, Iwamori K, et al. Clinical and prognostic significance of atrial fibrillation in acute myocardial infarction. *Am J Cardiol* 1997;15:1522-7.
- Horimoto M, Igarashi K, Takenaka T, et al. Coronary vasospasm as a potential cause of myocardial infarction and paroxysmal atrial fibrillation in a relatively young woman. *Clin Cardiol* 1991;14:699-702.
- Gokshyn S, Mewis C, Kuhlkamp V. Atrial fibrillation in coronary artery disease. *Int J Cardiol* 2000;72:133-6.
- Sugiura T, Iwasaka T, Takahashi N, et al. Factors associated with atrial fibrillation in Q-wave anterior myocardial infarction. *Am Heart J* 1991;121:1409-12.
- Christiansen EH, Frost L, Pilegaard H, et al. Within and between patient variation of signal averaged P wave in coronary artery disease. *Pacing Clin Electrophysiol* 1996;19:72-81.
- James TN, Burch GE. The atrial coronary arteries in man. *Circulation* 1958;17:90-8.
- Ogawa H, Inoue T, Yoshida A, et al. The signal averaged electrocardiogram of P wave in patients with documented atrial fibrillation or flutter and in patients with left or right overload without atrial fibrillation. *Jpn Heart J* 1993;34:29-39.
- Faggiano AA, Zanelli E, Gualeni A, et al. Contribution of left atrial pressure and dimension to signal averaged P-wave duration in patients with chronic congestive heart failure. *Am J Cardiol* 1997;79:219-22.
- Aspromonte N, Auruti A, Ceci V. Efficacy of class Ic antiarrhythmic drugs predicted by P-wave signal averaged ECG in patients with paroxysmal atrial fibrillation. *J Amb Monitoring* 1994;7:115.
- Stafford PJ, Cooper J, Bono DP, et al. Effect of low dose sotalol on the signal averaged P wave in patients with paroxysmal atrial fibrillation. *Br Heart J* 1995;74:636-40.
- Budeus M, Hennersdorf M, Perings C. The effect of antiarrhythmic therapy on variables of P-wave signal averaged ECG among paroxysmal atrial fibrillation. *Z Kardiol (in revision)*
- Simson RJ, Foster JR, Gettes LS. Atrial excitability and conduction in patients with inter-atrial conduction defects. *Am J Cardiol* 1982;50:1331-7.
- Simson RJ, Amara I, Foster JR, et al. Thresholds, refractory periods and conduction times of the normal and diseased human atrium. *Am Heart J* 1988;116:1080-90.
- Buxton A, Waxman H, Marchlinski FE, et al. Atrial conduction: Effects of extrastimuli with and without dysrhythmias. *Am J Cardiol* 1984;54:755-61.
- Guidera S, Steinberg JS. The signal averaged P-wave duration: A rapid and noninvasive marker of risk of atrial fibrillation. *J Am Coll Cardiol* 1993;21:1645-51.
- Saksena S, Prakash A, Krol RB, et al. Regional endocardial mapping of spontaneous induced atrial fibrillation in patients with heart disease and refractory atrial fibrillation. *Am J Cardiol* 1999;84:880-9.