Permanent cardiac pacing in adult patients with congenital heart disease after cardiac surgery

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Abstract

Introduction: Impulse formation and conduction disturbances demanding permanent pacing are present in some adult patients with congenital heart disease (CHD). Almost half of these patients had a cardiac surgery in the past. The aim of this study was to analyze causes and modes of permanent cardiac pacing in this population.

Material and methods: Data of 32 adult patients (20 women) mean age 28.4 years after pacemaker implantation were selected from a database of Adult Congenital Herat Disease Outpatient Clinic of the 1st Cardiology Department in Poznan. The mean age at the cardiac surgery in this group was 13.3 years.

Results: The first implantation in this group was done at 20.5 years of age. In 25 patients a complete or advanced heart block was a cause of pacemaker implantation, in 12 directly after the cardiac surgery. Sinus node dysfunction was a reason of pacing in other 7 patients. In half of the cases the epicardial system was applied initially. A multiple implantation was done in 17 (double); 7 (triple) and 2 (quadruple). **Conclusions:** The necessity of permanent pacing in adult patients with CHD may appear directly after the operation as well as in long term follow up. Many of these patients need multiple pacemaker implantations during their life span. The implantation technique in CHD patients often differs from standard procedures in general "pacemaker" population. This requires from the implantation team a broad knowledge of defect anatomy and cardiac surgery mode.

Key words: permanent cardiac pacing, congenital heart disease in adults.

Introduction

The dynamic development of pediatric cardiology and cardiosurgery seen over 50 years, allows most of the patients with congenital heart diseases (CHD) to live to adulthood [1]. There are however some defect residua due to anatomical abnormalities and postoperative complications. These may lead to life threatening impulse formations and conduction disturbances [2-4]. The need for a pacemaker implantation may appear at any age, but only 1% of this procedure is done during the first two decades of life [5-10]. Pacing experience in adults with acquired heart disease is not necessarily representative of this group, and may not be appropriate to extrapolate from experience in the pediatric paced population. Implanting a pacemaker in these patients is sometimes a very difficult procedure, thus a superb knowledge of electrophysiology, defect anatomy and postoperative changes is crucial [5, 9, 11]. Choosing the implantation and pacing modes an operator must take into consideration multiple reimplantation in the entire patient lifespan. Complication and reintervention rates specific to this population are unknown.

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The aim of this study was to analyze causes and modes of permanent pacing in this population.

Material and methods

The data of 32 adult patients (20 women) aged 18-57 (mean age 28.4±12.2) after a pacemaker implantation were selected from a database of Adult Congenital Herat Disease Outpatient Clinic of I Cardiology Department in Poznan (1997-2005). A cardiac surgery was done in this group at 0.5-56 years of age (mean 13.3±15.6). Following CHD were the reasons for surgery: ventricular septal defect (VSD) in 9 patients, Fallot's tetralogy (TOF) in 7 patients, combined aortic valve disease (VAC) in 3 patients, atrial septal defect (ASD) in 6 patients: in 3 ASD II, in 2 ASD I, in 1 ASD sin ven. Three patients presented with single ventricle (one after Fontan's operation, the second after palliative Blalock-Taussig anastomosis, the third unoperated). There were single patients with: complete atrio-ventricular canal (CAVC), transposition of the great arteries post Mustard operation (dTGA), congenitally corrected l-transposition of the great arteries coexisting with ventricular septal defect (ITGA+VSD) and double outlet right ventricle (DORV).

Results

The main cause for pacemaker implantation in the studied population was a complete heart block (AV III block) diagnosed in 20 patients (3 patients presented with AV III block before the surgery). Other reasons: advanced heart block type II (AV II block) in 5 patients, sick sinus syndrome (SSS) in 7 patients and finally a complete heart block after unsuccessful atrial flutter RF ablation in 1 patient. In 12 cases implantation was done directly after the surgery -Table I. The mean age at the first implantation was 20.5±15.9 on average 6.7±7.9 years after the surgery. The second implantation was done in 17 (mean age 17.4±6.8) patients 5.6±2.9 years after the initial one. The third procedure was done in 7 patients (aged 21.7±9.2) after 6.4±2.8 years form the former and the fourth so far in 2 patients (aged 22.6±6.4) after following 5.5±3.5 years of follow up – Table II. The types of implanted pacemakers are shown in Table III. Initial epicardial pacing systems were applied in 17 cases, mainly VVI devices (DDD - 7 patients, AAI and VVIR only in single individuals). Urgent damaged epicardial lead replacement was performed in just one case. If defect anatomy made it possible transvenous systems were introduced afterwards. Finally epicardial pacing was continued in one patient with TOF with recovery of atrio-ventricular conduction, in 2 patients with physiologically single ventricle and in one after Mustard's operation. In one patient with single ventricle and dTGA after a palliative surgery (artero-pulmonary anastomosis) epicardial implantation procedure during tricuspid

valvuloplasty was performed. This procedure was unsuccessful. Two days after the surgery the patient died due to multiorgan dysfunction.

Discussion

As a result of anatomical malformations of the heart, it happens that patients with CHD present also conduction anomalies [2, 3, 11, 12]. Moreover, cardiac surgery often leads to conduction system injury causing a complete heart block in 1% of cases [3, 6, 13, 14]. The analysis of our patients showed that most often pacemaker implantation was done after VSD correction. In this defect AV III block is usually caused by surgical injury of Hiss bundle which is located near to the edge of the VSD [15]. It is seen in 4-28% of cases in an early postoperative period, but remains in about 1-2% [11, 16, 17]. Sometimes AV III block may appear many years after a spontaneous closure of the VSD [16].

In patients with Fallot's tetralogy causes of pacing were diverse: AV III block in 3 patients, advanced AV II block in 3 patients, SSS in 1 patient. The long-term follow up indicates that the need for pacing in TOF patients is about 1-2% of adults, operated in childhood [4, 17]. The late onset of AV III block is not necessarily preceded by bifascicular block as believed previously [4]. It appears however, and is more often in those patients, who presented with an early, transitory complete heart block in the perioperative period [18]. In one patient discussed above, a return of the sinus rhythm was observed in the long-term follow up. Reports of the sinus rhythm restoration are very rare. It was published in patients after the surgery due to CAVC [14] and in a patient with corrected transposition of the great arteries [19]. Sinus node dysfunction as a reason for pacing is rare in TOF patients and appears in the long term follow up [4, 10, 18].

Topography of the conduction system offers a great risk of its injury during the surgery of the aortic stenosis, but in our observation permanent pacing in these patients is rarely needed – 3 cases in our group. Such good results in patients with aortic stenosis are probably the effect of continuous improvement in the surgical technique [6, 10, 11, 20].

As reported by other authors, pacemaker implantation in our patients with ASD I, II and ASD sinus venosus type, were performed, due to sick sinus syndrome [6, 10, 20]. Usually silent SSS is observed in 41-83% in ASD patients, especially in the familial form of this disease and in elderly patients [4]. In one female patient after a cardiac surgery due to CAVC, the pacemaker was implanted because of AV III block. Complete heart block is seen only in 2% of patients with CAVC even though defect anatomy predestinates to advanced conduction disturbances [4, 17]. Furthermore, cardiac surgery in CAVC may lead to sinus node injury, and atrial scars facilitate the onset of supraventricular arrhythmias, which may need

n	Initials	Gender	Age (years)	Age at surgery (years)	Congenital heart disease	Cause of pacemaker implantation
1	RM	f	22	3	VSD	Congenital AV III block
2	TM	f	24	4	VSD, ASD II	AV III block
3	SI	f	21	5	VSD	Congenital AV III block
4	WH	f	21	2	VSD	AV III block
5	MA	f	32	4	VSD	AV III block
6	EJ	f	33	7	VSD	AV III block
7	GG	m	18	1	VSD	AV III block
8	BP	f	32	22	VSD	AV III block
9	FK	f	18	11, 10, 17	VSD IA prosthetic aortic valve	AV III block
10	WM	m	20	5	TOF	AV III block
11	DM	m	19	7	TOF	Advanced AV II block, VEB
12	PK	f	22	3	TOF	Advanced AV II block
13	SP	m	21	5	TOF	Advanced AV II block
14	PM	f	43	17	TOF	AV III block
15	PA	f	37	9	TOF	SSS
16	SM	m	36	2	TOF	AV III block (regression)
17	ZD	m	24	9	VAC	AV III block
18	JA	m	22	20	VAC prosthetic valve	AV III block
19	ND	m	19	9, 16	VAC prosthetic valve	AV III block
20	DL	f	56	56	ASD II	SSS
21	UA	f	57	50	ASD II	SSS
22	BJ	m	55	43	ASD II	SSS
23	AK	f	21	10	ASD sin ven	SSS
24	HH	m	56	55	ASD I	SSS
25	KA	f	21	7	ASDI	SSS
26	SzJ	f	22	8	Single ventricle, DTGA, post Fontan surgery	AV III block
27	MT	m	18	18	Single ventricle, DTGA, BT-shunt	Advanced AV II block, VEB
28	JP	m	31	14	Tricuspid atresia	Advanced AV II block
29	BM	f	20	8	CAVC, VCS sin	AV III block
30	DI	f	30	1	DTGA post Mustard surgery	AV III block (post RF ablation of Fl A)
31	KP	f	19	3	DORV	AV III block
32	TE	f	19	17	LTGA, VSD	Congenital AV III block

Table I. Demographic characteristics, congenital defect type and reason for pacemaker implantation in the studied group

VSD – ventricular septal defect, TOF – Fallot's tetralogy, VAC – combined aortic valve disease, ASD – atrial septal defect, CAVC – complete atrio-ventricular canal, DTGA – transposition of great arteries, LTGA – congenitally corrected l-transposition of great arteries, DORV – double outlet right ventricle, SSS – sick sinus syndrome, AV II/III block – atrio-ventricular II/III block

a pacemaker back-up during antiarrhythmic treatment [6, 10, 16, 19].

AV III block post unsuccessful atrial flutter ablation was a reason of pacemaker implantation in a female patient with dTGA post Mustard operation. In these patients complex supraventricular arrhythmias are often observed as a result of raised atrial pressure and incisional re-entry phenomenon [12, 21]. Approximately half of the dTGA patients present additionally signs of SSS resulting from histological changes and sinus node vascular supply anomalies. Loss of the sinus rhythm reaches about 2.4% annually. This makes a necessity of implanting the pacing system in 6% of this group of patients [22].

Congenitally corrected TGA, with VSD closure, observed in one of our patients, is a defect with

Table II. The age at cardiac surgery and pacemaker implantation

n Init	itials Aget at implantation (years)	Time surgery – implantation (years)	Age at 1 reimplantation (years)	Age at 2 reimplantation (years)	Age at 3 reimplantation (years)	Time implantation-1 reimplantation (years)	Time 1-2 reimplantation (years)	Time 2-3 reimplantation (years)
1 RM	M 3	0	17	0	0	14		
2 TM	М 5	1	10	18	0	5	8	
3 SI	6	1	13	17	0	7	4	
4 WF	'H 12	10	0	0	0			
5 MA	A 4	0	8	19	27	4	11	8
6 EJ	33	26	0	0	0			
7 GG	G 1	0	9	15	18	8	6	3
8 BP	20	4	28	0	0	8		
9 FK	K 17	0	0	0	0			
10 WA	'M 5	0	16	0	0	11		
11 DM	M 18	11	0	0	0			
12 PK	< 22	19	0	0	0			
13 SP	21	16	0	0	0			
14 PM	M 30	13	36	43	0	6	7	
15 PA	4 37	28	0	0	0			
16 SM	8 N	6	12	0	0	4		
17 ZD) 9	0	16	23	0	7	7	
18 JA	20	0	0	0	0			
19 ND	D 16	0	0	0	0			
20 DL	L 56	0	0	0	0			
21 UA	A 57	7	0	0	0			
22 BJ	54	9	0	0	0			
23 AK	Κ 20		10	0	0	0		
24 HH	H 55	0	0	0	0			
25 KA	A 12	5	20	0	0	8		
26 SzJ	zJ 16	8	21	0	0	5		
27 MT	T 18	9	0	0	0			
28 JP	23	9	31	0	0	8		
29 BM	8 N	0	15	17	0	7	2	
30 DI	20	19	28	0	0	8		
31 KP	12	2	15	0	0	3		
32 TE	17	0	19	0	0	2		
	20.5±15.9	6.7±7.9	17.4±6.8	21.7±9.2	22.5±6.4	5.6±2.9	6,4±2,8	5,5±3,5

n	Initials	Pacing mode 1	Pacing mode 2	Pacing mode 3	Pacing mode 4
1	RM	VVI epicardial	DDD	0	0
2	TM	VVI epicardial	VVI epicardial	DDD	0
3	SI	VVI epicardial	VVI epicardial	DDD	0
4	WH	VVI epicardial	0	0	0
5	MA	VVI epicardial	VVI epicardial	VVI epicardial	VDD
6	EJ	DDD	0	0	0
7	GG	VVI epicardial	VVI epicardial	VVI epicardial	DDD
8	BP	VVI	VDD	0	0
9	FK	VVI	0	0	0
10	WM	VVI epicardial	VDD	0	0
11	DM	VVI epicardial	0	0	0
12	PK	DDD	0	0	0
13	SP	DDD	0	0	0
14	PM	DDD	DDD	DDD	0
15	PA	DDD	0	0	0
16	SM	VVI epicardial	VVI epicardial	0	0
17	ZD	VVI epicardial	VVI epicardial	DDD	0
18	JA	DDD	0	0	0
19	ND	VVI	0	0	0
20	DL	VVI	0	0	0
21	UA	DDD	0	0	0
22	BJ	AAI	0	0	0
23	AK	DDD	0	0	0
24	НН	VVI	0	0	0
25	KA	VVI epicardial	DDD	0	0
26	SzJ	VVI epicardial	VVI epicardial	0	0
27	MT	Attempt to implant VVI epicardial	0	0	0
28	JP	VVI epicardial	VVI epicardial	0	0
29	BM	VVI epicardial	VVI epicardial	DDD	0
30	DI	VVI R	VVI R	0	0
31	KP	VVI epicardial	DDD	0	0
32	TE	DDD epicardial	DDD	0	0

Table III. Pacing modes

a high probability of advanced conduction disturbances. It is a result of abnormal topography of conduction fibers and frequently coexisting double sinus node [26].

Surgical closure of VSD was also a most probable cause of AV III block in a female patient with DORV [10, 15-17].

The most difficult choice of the pacing mode takes place in patients with the heart with physiology of a single ventricle. For that reason there are no possibilities to implant an electrode in the right ventricle, reports of pacing in these patients are rare [9, 10, 24, 25]. In our group, two patients had an implanted epicardial, ventricular electrode (VVI mode) many years after the surgery. In the third patient there was an attempt to implant an epicardial system during surgical tricuspid valvuloplasty, this patient died two days after the operation. Fishberger et all. [8] reported that 9.2% patients from among 500 who underwent Fontan surgery, had a pacemaker implanted due to SSS and advanced heart block with coexisting supraventricular arrhythmias demanding

treatment. Aproximatelly 10% of patients after this operation need a permanent pacing in long term follow up [24-26]. In the beginning, the implanting procedure consisted of sewing on the epicardial leads on the ventricle [8]. Werfield et al. [3] had implanted epicardial leads as prophylaxis during Fontan surgery. Dual chamber pacing, which improves venous return by atrio-ventricular synchronization is especially indicated after Fontan surgery. A higher survival rate was proven in these patients [9, 14, 27]. Atrial leads implantation was then introduced - some operators used the transvenous approach which demanded anticoagulation [3, 9, 8]. Mixing of the venous and arterial blood in a single ventricle is, in fact, an intracardiac shunt, facilitating thrombosis and embolism, thus it is an absolute indication for anticoagulation [3, 8, 15, 28]. Some preferred epicardial right or left atrial leads or as mentioned above, others implanted them as prophylaxis during Fontan procedure [8, 20, 24]. Recently, DDD transvenous pacing was started, using the coronary sinus approach to place a ventricular lead [7]. Many authors emphasize that such a mode of pacing should be used only in highly experienced CHD centers [25, 27].

The first pacing system in our studied population was implanted 30 years ago. The majority of the operated patients then were protected by epicardial pacing. As in other centers [6, 10], the multiple reimplantations were done successively. Sachwech et al. [6], published a retrospective analysis of the pacing modalities in children treated in the Aachen University Hospital in 1979-98: epicardial systems were used in 69% of cases, endocardial ones in 31%, usually forms of the single chamber ventricular pacing among them. The use of the endocardial pacing in children is also limited by the growth process [3, 8, 25]. Till the eighties, transvenous systems were used in children over 4 years of age and with at least 15 kg of weight and with no intracardiac shunt. Moreover, in children transvenous lead implantation may injure the vessel wall in 35-40% [6, 9, 29]. Epicardial stimulation due to higher pacing threshold diminishes battery longevity [5, 24, 27]. Steroids eluting epicardial leads have improved this situation substantially [5, 6, 20, 30]. The choice of epicardial leads in children is supported by possibility to implant them during the cardiac surgery [3, 9, 20]. But leads replacement in this system is a severe cardiac surgery with a high risk of intensified hemorrhage during surgery and poststernothomy syndrome afterwards [5, 6, 20, 28]. Epicardial leads are more prone to damages and infections (although in our observations these complications were very rare) [6, 14, 17,31]. Based on these experiences, some authors promote an early use of endocardial systems, especially dual chamber devices, emphasizing a role of the atrias in cardiac output (which increases with age) [7, 25, 28]. Despite that dual chamber system is physiologically more favorable, even in best centers most of the epicardial

systems implanted between 1990-2000 were single-chambered, probably due to simplification of a very complex surgical intervention [27].

Conclusions

The presented above analysis of adult patients with CHD, treated previously in different centers, and now gathered in one university centre shows that:

- The necessity of permanent pacing in adult patients with CHD may appear directly after the operation as well as in the long term follow up. Many of these patients need multiple pacemaker implantations during their life span. A new group of patients has arisen, who are dependent on pacing through most of their lives.
- The pacemaker implantation technique in adult CHD patients differs from the standard procedures in the general "pacemaker" population. This requires from the implantation team a broad knowledge of the defect anatomy and cardiac surgery mode.

References

- 1. Somerville J. Grown-up congenital heart disease medical demands look back, look forward 2000. Thorac Cardiovasc Surg 2001; 49: 21-6.
- 2. Morgan DR, Hanratty CG, Dixon LJ, Trimble M, O'Keeffe DB. Anomalies of cardiac venous drainage associated with abnormalities of cardiac conduction system. Europace 2002; 4: 281-7.
- 3. Warfield DA, Hayes DL, Hyberger LK, Warnes CA, Danielson GK. Permanent pacing in patients with univentricular heart. Pacing Clin Electrophysiol 1999; 22: 1193-201.
- 4. Perloff JK, Child JS. Congenital heart disease in adult. Saunders Company. Philadelphia, 1998: 316-41.
- 5. Kerstjens-Frederikse MW, Bink-Boelkens MT, de Jongste MJ, Homan van der Heide JN. Permanent cardiac pacing in children: morbidity and efficacy of follow-up. Int J Cardiol 1991; 33: 207-14.
- Sachweh JS, Vazquez-Jimenez JF, Schondube FA, Daebritz SH, Dorge H, et al. Twenty years experience with pediatric pacing: epicardial and transvenous stimulation. Eur J Cardiothorac Surg 2000; 17: 455-61.
- 7. Heinemann MK, Gass M, Breuer J, Ziemer G. DDD pacemaker implantation after Fontan-type operations. Pacing Clin Electrophysiol 2003; 26: 492-5.
- Fishberger SB, Wernovsky G, Gentles TL, Gamble WJ, Gauvreau K, et al. Long-term outcome in patients with pacemakers following the Fontan operation. Am J Cardiol 1996; 77: 887-9.
- 9. Ramesh V, Gaynor JW, Shah MJ, Wieand TS, Spray TL, et al. Comparison of left and right atrial epicardial pacing in patients with congenital heart disease. Ann Thorac Surg 1999; 68: 2314-9.
- Walker F, Siu SC, Woods S, Cameron DA, Webb GD, et al. Long-term outcomes of cardiac pacing in adults with congenital heart disease. J Am Coll Cardiol 2004; 43: 1894-901.
- 11. Bonatti V, Agnetti A, Squarcia U. Early and late postoperative complete heart block in pediatric patients submitted to open-heart surgery for congenital heart disease. Pediatr Med Chir 1998; 20: 181-6.
- 12. Silvetti MS, Drago F, Pastore E, Turchetta A, Calzolari A, et al. Does chronic pacing affect exercise capacity after

Mustard operation for transposition of the great arteries? Pediatr Cardiol 2002; 23: 3-8.

- 13. McGrath LB, Gonzalez-Lavin L, Morse DP, Levett JM. Pacemaker system failure and other events in children with surgically induced heart block. Pacing Clin Electrophysiol 1988; 11: 1182-7.
- 14. Kratz JM, Gillette PC, Crawford FA, Sade RM, Zeigler VL. Atrioventricular pacing in congenital heart disease. Ann Thorac Surg 1992; 54: 485-9.
- 15. Nygren A, Sunnegardh J, Berggren H. Preoperative evaluation and surgery in isolated ventricular septal defects: a 21 year perspective. Heart 2000; 83: 198-204.
- Moller JH, Patton C, Varco RL, Lillehei CW. Late results (30 to 35 years) after operative closure of isolated ventricular septal defect from 1954 to 1960. Am J Cardiol 1991; 68: 1491-7.
- 17. Klug D, Vaksmann G, Jarwe M, Wallet F, Francart C, Kacet S, et al. Pacemaker lead infection in young patients. Pacing Clin Electrophysiol 2003; 26: 1489-93.
- Deanfield J, Thaulow E, Warnes C, Webb G, Kolbel F, et al. Management of grown up congenital heart disease. Eur Heart J 2003; 24: 1035-84.
- 19. Ngarmukos T, Werres R. Normal sinus rhythm in a patient with corrected transposition of great arteries after 30 years of complete heart block. Pacing Clin Electrophysiol 1999; 22: 1116-7.
- 20. Cohen MI, Bush DM, Vetter VL, Tanel RE, Wieand TS, et al. Permanent epicardial pacing in pediatric patients: seventeen years of experience and 1200 outpatient visits. Circulation 2001; 103: 2585-90.
- 21. Gillette PC, Wampler DG, Shannon C, Ott D. Use of cardiac pacing after the Mustard operation for transposition of the great arteries. J Am Coll Cardiol 1986; 7: 138-41.
- 22. Gewillig M, Cullen S, Mertens B, Lesaffre E, Deanfield J. Risk factors for arrhythmia and death after Mustard operation for simple transposition of the great arteries. Circulation 1991; 84 (suppl. 5): III187-92.
- 23. Fischbach PS, Law JH, Server GS. Congenitally corrected transposition of the great arteries: abnormalities of atrioventricular conduction. Prog Pediatr Cardiol 1999; 10: 37-43.
- 24. Cohen MI, Vetter VL, Wernovsky G, Bush DM, Gaynor JW, et al. Epicardial pacemaker implantation and follow-up in patients with a single ventricle after the Fontan operation. J Thorac Cardiovasc Surg 2001; 121: 804-11.
- Fan K, Yung TC. Permanent ventricular pacing from coronary sinus after Fontan operation using newly designed left ventricular lead. J Interv Card Electrophysiol 2002; 7: 89-93.
- Cohen MI, Wernovsky G, Vetter VL, Wieand TS, Gaynor JW, et al. Sinus node function after a systematically staged Fontan procedure. Circulation 1998; 98 (suppl. 19): II352-8.
- Ten Cate FU, Breur J, Boramanand N, Crosson J, Friedman A, et al. Endocardial and epicardial steroid lead pacing in the neonatal and paediatric age group. Heart 2002; 88: 392-6.
- 28. Bostan OM, Celiker A, Karagoz T, Ozer S, Ozme S. Dual chamber cardiac pacing in children: Single chamber pacing dual chamber sensing cardiac pacemaker or dual chamber pacing and sensing cardiac pacemaker? Pediatr Int 2002; 44: 635-40.
- 29. Valsangiacomo E, Molinari L, Rahn-Schonbeck M, Bauersfeld U. DDD pacing mode survival in children with a dual-chamber pacemaker. Ann Thorac Surg 2000; 70: 1931-4.
- 30. Horenstein MS, Walters H 3rd, Karpawich PP. Chronic performance of steroid-eluting epicardial leads in a growing pediatric population: a 10-year comparison. Pacing Clin Electrophysiol 2003; 26: 1467-71.
- Figa FH, McCrindle BW, Bigras JL, Hamilton RM, Gow RM. Risk factors for venous obstruction in children with transvenous pacing leads. Pacing Clin Electrophysiol 1997; 20: 1902-9.