CARDIAC PACING

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Permanent pacemaker implanatation is now being used widely for the treatment of a variety of cardiac rhythm distrubances since the 1st implantation by Elmquist and Senning in 1959. We have implanted 76 permanent pacemakers in the National Institute of Cardiovascular Diseases in the last 3 years. The purpose of this paper is to report our findings as regards indications, technique and complications of permanent pacemaker implantation in these patients.

Materials & Methods :-

Seventy two patients had permanent pacemaker implantation Table I shows the underlying rhythm, age and sex of these patients.

46 patients had temporary pacemaker inserted prior to the implantation of a permanent pacemaker. The rest of the patients were stable at the time of permanent pacemaker and were receiving isupril drip intra venously.

Most of our patients presented to the E.R. in Adam's Stokes (table II) attacks and thus had a temporary pacemaker inserted prior to having a permanent pacemaker. Rest of the patients had complete heart block and past history of unconciousness and were admitted from the O.P.D. for a permanent pacemaker implantation. Four of these patients developed asystole after the injection of local anaesthetic 2% xylocain and had cardiac message and later on Isupril infusion was started. Since then all patient not having temporary pacemakers are started on Isupril drip on arrival to the Cardiac Catheterization Laboratory. This may alter the stimulation threshold or increase the VPC'S during implantation but we have not seen any asystole since then during implantation. Four patients had extremely small cephalic vein and the external jugular vein has to be utilized. All units are tested prior to implantation by Medtronic Pacemaker Analyzer. Pulse duration, pulse amptitude and cycle length are recorded. Similarly lead resistance and endocardial or myocardial threaholds are also checked and recorded. All patients are monitored for 3 days and antibiotics are given on the day and for 7 days after the implanatation. No patients had died waiting either in the group with temp. pacemaker or in the group without temp: pacemaker waiting for a permanent pacemaker.

Table No. 3 shows the complications associated with permanent pacemaker implantations. None of our patients developed lead displacement either in the right ventricular outflow tract, pulmonary artery, or back into the Right atrium. However three patients developed diaphramgatic contractions after the implantation. In two

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cases the lead was readjusted with normal function initially, however after a week one patient redeveloped diaphragmatic contractions and myocardial leads were used in this case with success. The 3rd case had spontaneous recovery without any problem with sensing or pacing. Two other patients developed sudden unconciousness following excessive forceful Jerky movement of the right arm 6-8 months following implantation of the permanent pacemaker. One is a doctor who developed syncope after Jerking the Thermometre to take the temp. of the patient. The other patient wanted to frighten his grand son with a stick and after sudden exessive rapid elevation of the right arm developed weakness and syncope. Both events resulted in displacement of the lead which needed repositioning.

The commonest complications we have observed is the extrusion of the power pack from the pocket due to presure necrosis of the overlying skin and secondry infection. This was noted in 5 cases. Three of these patients had diabetes mellitus and all of them received steroids for a long period of time for chronic heart block.

Four of these cases utimately had myocardial leads with power packs in the rectus sheath. One was repositioned successfully in the same pocket. Table No. 4 shows the type of device used. The single units used were generally donations from the companies concerned.

DISCUSSION

Syncope occuring in patients with a slow heart rate due to complete heart block, commonly known as Adams-Stokes Syndrome is recognised since the time of Morgagny (1) More recently this has been described in the setting of Sick Sinus Syndrome (2) and prolonged QT interval associated with or without deafness (3).

The mechanism of syncope is based upon the cessation of blood supply because of prolonged asystole (Ventricular standstill with or without sinus activity) in 80%, due to failure of primary pacemaker and non availability of the second escape focus due to exit block or suppression by the same cause factor responsible for the failure of primary pacemaker. In 20% of cases, transient ventricular fibrillation is also present (4). The rapidity of unconciousness depends upon the status of cerebral circulation, cardiac muscle status and posture of the patients at the time of onset of asystole. It is usually between 4-8 seconds in standing and 10-15 seconds in recumbent subjects. (5)

Many of these patients are being diagnosed epilepsy and in this series 5 patients were receiveing anti-epileptic drugs. In this setting other cardiac and extracardiac causes for syncope and seizures should be considered. However, a patient with syncope or seizures who either has 2° or 3° A-V Block, or bifascicular and trifascicular block, Stoke Adam's syndrome must be considered until proven otherwise (6). On rare occasions epilepsy and S.A. Syndrome may be associated.

Majority of patients with chronic acquired A.V. Block has a degenerative pathology (7, 8). The bundle branches and the main bundle of His are replaced by fibrous tissue. Calcification may be seen. Harris (8) found that 40% cases had degenerative changes in the bundle branches without any other cardiac pathology Coronary artery disease was found only in 15% cases, 23% cases had associated myopathy or myocarditis.

Chronic heart block results in wide pulse pressure (Average 107 mm Hg) and increased

stroke volume. Cardiac out put is low and the resistance in pulmonary and systemic circuits is increased. In patients having complete heart block in this series 6 patients presented with signs and symptoms of C.H.F. Eighteen others also had associated C.H.F.

Some patients with C.H.B. have subtle signs of insufficicent cerebral circulation called "Little Strokes".

Nine patients were diagnosed to have sick sinus syndrome. Two girls had A.S.D. repair and after an average of one year were found to have periods of sinus arrest. They were started on sublingual Isoprenaline but with temporary relief only. Eventually both patients developed Stoke Adams attacks and pacemakers were installed for Sick Sinus Syndrome. The rest of our seven patients had either severe bradycardia or periods of sinus arrest of more than 2.5 seconds.

As shown in table 2, the average age of female patients was 10 years younger than the complete heart block group. This is because of these two post operative cases, otherwise the mean age of these patients is about the same as those of C.H.B. cases.

Sick Sinus Syndrome was first categorised by Ferrer in 1968, and its six major components were:

1. Persistent, severe and unexpected sinus bradycardia.
2. Sinus arrest with escape rhythm, atrial or junctional.
3. Sinus arrest without atrial or junctional escape rhythm, but ventricular arrhythmias may follow.
4. Transitory A.F. with or without slow Ventricular rate.
5. Sino-atrial block not due to drugs.
6. Slow recovery of sinus function after cardioversion of S.V.T.

It was further found that 60% of these also have defects in the rest of the conducting system.

Since its original description, Sinus node disturbances have come to occupy greater and greater number of patients receiving permanent pacer therapy. In one study 36% of all permanent pacemaker patients had S.S.S. The relatively few patients in our study may be due the lack of recognition of this entity and subsequent referal.

The primary manifestation i.e. Sinus bradycardia of S.S.S. has always been thought to be a benign rhythm, and only continuous monitoring to document periods of sinus arrest or tachy-brady sequence is needed to unmask the serious nature of the arrhythmia. Prolonged monitoring with electromagnetic tape recorders are of great value.

Various provocative tests are available for evaluation of symptomatic sinus bradycardia and other varieties of S.S.S. Overdrive suppression is used with increasing frequency and a figure beyond 120-130% is taken as abnormal. In six of our patients overdrive suppression was done and was positive.

One of our patients was 60 years old male who was admitted to another hospital with history of fainting spells and palpitations. He was found to have episodes of recurrent V.T. He was started on I/V Xylocaine, Inderal and Quinidine, but with very little effect. He was shifted to N.I.C.V.D. and here his quinidine dose was increased, but was still in effective I/V Pronestyl was added subsequently, but the arrhythmia was still not controlled. A temporary pace maker was inserted, and the tachyarrhythmia was terminated as soon as the pacer entered the R.V. He was paced at 100 S.P.M. Which controlled the V.T. completely. All antiarrhythmics were withdrawn and he was started on Norpace. After one week of therapy his pacer was turned off after having gradually decreased the rate. However, he had recurrence the same night and controlled again with pacing. A programmed pacer was installed and he is symptom free over the last one year.

The use of pacing for the treatment of tachyarrhythmia is manifold. The commonest and easy to understand is guaranteed minimal Ventricular rate while pharmacologic agents are poured into the patient. Secondly in patients having degitalis overdose where cardioversion is extemely risky. The third one involves the termination of tachyarrhythmia uncontrollable by pharmacologic therapy. The tachyarrhythmia may themselves be terminated by random pacer stimulus (random extrasystole), and then may be prevented by pacing at a slightly higher than his normal sinus rate coupled with pharmacological suppression. In other instances the tachycardia, can only be terminated with pacing rates higher than the tachyarrhythmia (10-30 S.P.M. above the tachycardia), and then gradually slowing the pacer rate till there is take over by the sinus without ecotopy or tachycardia.

Short rapid bursts of pacing at very short coupling intervals (BRVP) may successfully terminate the techycarida Similarly, 1 to 3 stimuli, critically timed may cause termination.

While the atria may be paced safely upto 1000 S.P.M. because of the long refractory period of the A-V node, the rate for pacing the ventricles is much slower because of the marked reduction in cardiac out put at higher rates.

In short pacer termination of tachyarrythmias is superior to repeated Cardioversion and in cases where the Cardioversion is dangerous. Repeated testing with temporary pacer is required before a decision for a permanent pacer for this purpose is taken.

If the tachy cardia can be easily and repeatedly terminated and controlled by the above methods, and if the timed stimulus is not very precise, permanent pacing must be considered for prevention and termination of tachyarrhythmia.

The efficacy of different drug regimens may be tested in the hospital using the technique of reproducing the arrythmia, by repeated testing optimal combinations of antiarrythmics may be chosen which results in delayed induction or non induction of the tachycaria with the temp. pacer. If any drug causes easy induction it may be withdrawn.

In the series of Furman et al., long follow up of patients treated with both pacing and pharmacologic suppression, the results were

encouraging as regards the reduction in the number and duration of episodes, as well as the number of hospitalizations. Thus the patients having recurrent tachyarrhythmia must be tried with vaious manoeuvres of temporary pacing termination and prevention and then a programmable pacer installed for termination of the tachyarrhythmia.

In case of recurrence of tachycardia at home termination may be done by the patient himself or in the majority of cases by his physician using the above measures.

Till 1965 all permanent Pacemakers were installed via the transthoracic (thoracatomy under G.A.) route, mortality and morbidity although low, was significant (10) and old patients thought to be high risk for G.A. and throractomy could not avail this life saving device. After the introduction of the transvenous route utilizing cephalic-vien under local anaesthesia the procedure became extremely low risk and safely available even to otherwise high risk patients. The main disadvantage of this technique is the diplacement of the endocardial lead from the apex of the Rt. Ventricle to either the Rt. Ventricls outflonw, pulmonary arery or back into the right atrium with resultant loss of capture and at times with dive consequences. Perfortation of the myocardium is also seen (11) with resultant diaphragmatic contractions. However only repositioning is required and tamponade is extremely rare. Lead displacement is seen in approximately 5-10% cases of endocordial perpermanent pacing necessitating repositioning and this is thought to be due to inexperience and lack of attention to the proper position of the electrode in the Rt. Ventricular apex. This complication is quite low in centres where experienced teams are doing the pacemaker work. However two other alternatives are available for diminislring this complication.

(1) Lead Design:

Actively gripping endocardial leads are introduced with nylon or metal barbs, wires or tynes. Screws and balloons are also available! more recently another lead having porous tip of fine platinum fibres is available. It is said to stabilize in 24 hours and fibrotic reaction is complete in a week time as compared to the 2-4 weeks for the ordinary electrode.

(2) **Myocardial lead implantation:** by sutureless myocardial leads either through a limited thoracotomy or sub-xipphyoid approach with musclespliting incision and local anaesthesia. In the present series the myocardial electrodes were only utilized in cases of repeated perforation and repeated cases of prssure necrosis and infection resulting in power pack extrusion. It is said that this complication is due to a low grade infection of staph albus. However we have not cultured this organism from the wound. Which may be due to the fact that most of these patients were receving antibiotics as soon as redness appeared on the wound site. In our experience most of our patients were receiving steroids: they were older mostly diabetic and the large size "Asian Model" was used in most of these cases.

Table No. 4 shows the type of devices we have used. Most of our pacemakers were ventricular inhibited unipolar or Bipolar pacemakers low initial cost, longevity, reliability and reliable energy depletion indicator are most important factors in our Country where close follow up of most of our pacemaker patient is not possible

because of the socio economic conditions. Initially we have used the Medtronic Asian Model Pacemaker because of its low initial cost. This pacemaker which is no longer available in the market was powered with Zinc Mercury oxide cells, because of the electrolyte solution and chemical reaction gas was produced and therefore complete hermetic sealing was not possible and thus power drain and tissue reaction could not be minimised. Also because of its size and heavy weight pressure necrosis and power pack extrusion were commonly seen.

The other unit which we used was the xytron model. Although the conventional Znc Hg cells were used it has a longer life because of reduction in power drain due to a shorter pulse duration coupled with constant energy feature, that is when battery out put voltage declines the pulse duration automatically increase so that a constant energy level is available to the patient for pacing and loss of capture in the later months is minimized. More recently we have started using the Lithium powered units. This source of energy which was 1st used in France in November 1972, has distinct superiority over the other sources e.g. Znc Hg., etc. Because of the absence of electrolytes complete heremetic sealing is possible and thus the problem of tissue fluid entering the cell is minimized. In addition low output pacers are used thus the life of the pacemaker is further increased. Now an expected life time warranty packemaker is available in these units. The 3rd major benefit of these units is their predictability of power exhaustion and thus minizing the chances of sudden power failure in the last months of pacer life. The most common indicator of battery depletion in these units is an increase in the interspike interval that is decrease in the pacer rate thus elective pacemaker replacement is easy.

We have not installed any nuclear power pacemaker because of forbidding cost and the possible nuclear hazards to our patients, and that it has to be taken out of the body after death which is not practical in our country. Since the introduction of the lithium powered units the nuclear and the reachargeable nickle cadmium batteries, has extremely limited indication if any (12).

More recently pacemakers are introduced where the rate, out-put (pulseduration, voltage and current) and other parameters (refractory period, sensitivity pacing mode and A-V delay) can be programmed through an externally supplied device to fit the need of the patient.

The rate programming is of great benefit to patients with arrythmias, angina pectoris and C.H.F.

Great advances are in sight in this field for detection and termination of arrythmias as well as pacemaker malfunction detection and self correction, and A-V sequential pacing giving a more or less physiological role to pacemakers.

Our experience show that this is a low risk procedure with a very low incidence of serious complications. The main limiting factor in our country to the use of this life saving device is the cost especially of the newer lithium powered units. "Japanese Edition" type pacemakers would increase the number of pacemaker used in this country tremendously bringing it to most of our patients population in need of this life saving device.

Table-1

Diagnosis	Males		Females	
	No.	Age	No.	Age
C.H.B.	38	61 years	24	54 years
S.S.S.	4	64 years	5	44 years
V.T.	1	60 years	—	—
Total	43		29	

S.S.S.—Sick Sinus Syndrome.

Table-2

Adam Stockes ..	..	—	65
C.H.F. ..	..	—	6
Rec. V.T. ..	..	—	1
Total		—	72

Table 3:-Post-Operative Complications

1.	Lead Displacement	Nil
2.	Lead Fracture	Nil
3.	Perforations	3
4.	Infections:	
	(*a*) Local	3
	(*b*) Systemic	Nil
5.	Non-Specific Reactions	2
6.	Extrusion of Pacemaker	5

Table-4 Types of Devices Implanted

Make	Power Source	Name if any	No.
Medtronic			
	Znc Hg	Asian	16
	—do—	Xytron	28
	Lithium	Xyrel	12
Telectronics	Lithium	—	15
C.P.I.	Lithium	Microlith	2
Cordis	Zn Hg		1
Biotronics	Znc Hg		1
G.E.	Zn Hg		1
Total			76

Table-5 Technique Used

Transvenous		
Endocardial	Cephalic Vein	72
	Ext. Jugular	1
	Int. Jugular	1
Transthoracic		
Epicardial		8
Total Procedures		82

Table-6 Code for Identification of Pacemakers (13)

1st Letter Chamber Paced	*2nd Letter Chamber Sensed*	*3rd Letter Mode of Pacing*	*Generic Description*	*Previously Used Designation*
V	O	O	Ventricular pacing; no sensing function	Asynchronous; fixed rate; set rate
A	O	O	Atrial pacing; no sensing function	Atrial fixed rate; atrial asynchronous
D	O	O	Atrioventricular pacing; no sensing function	A-V sequential fixed rate (asynchronous)
V	V	I	Ventricular pacing and sensing, inhibited mode	Ventricular inhibited; R-inhibi- R-blocking; R-suppressed; non-competitive inhibited; demand; standby
V	V	T	Ventricular pacing and sensing, triggered mode.	
A	A	I	Atrial pacing and sensing, inhibited mode	Atrial inhibited, etc. (See VVI, but substitute atrial P wave for ventricular R wave)
A	A	T	Atrial pacing and sensing, triggered mode	Atrial triggered, tc. (See VVT, but substitute as in AAI)
V	A	T	Ventricular pacing; atrial sensing, tirggered mode	Atrial synchronous; atrial synchronized; A-V synchronous
D	V	I	A-V pacing; ventricular sensing, inhibited mode	Bifocal sequential demand; A-V sequential

A—atrium; A-V—atrioventricular; D—double chamber; I—inhibitied; O—not applicable; T—triggered V—ventricle.

Table-7 Pacemaker Power Sources Available

Source	*Longevity*	*Problems*
Zinc Mercury Oxide	2-4 yrs	Short life span unpredictable end of life.
Lithium Iodide Silver Chromate Lead iodine Thionyl Chloride Copper Sulfide Bromide	10-15 yrs	Low output capcity.
Nickle Cadmium		Frequent Recharging.
Nuclear 238 Pu	20 yrs	Radiation and disposal.
147 PM	5-10 yrs	

Table-8 Methods of Increasing The Life span of Power Packs.

1. Energy Source.
2. Low output units.
3. Decrease in pulse duration.
4. Decrease in electrode surface area.
5. Hermetic sealing of power source as well as the circuitary.
6. Digital circuitary.
7. Hybrid circuit technology.
8. Integrated circuits.
9. Minimal Thresholds (Both Voltage and current) (14) endocadial.

Table-9 Modes of Pacing Used

1. Asynchronous (fixed rate)
   The pacemaker fires at a present rate without regard for the under lying patients own heart rythm.

2. Demand
   Inhibited:—A pacemaker whose output is blocked by a sensed signal.

   Triggered:—A unit whose output is fired by a sensed signal.

3. A—V sequential utilizing separate Atrial and Ventricular leads both atria and ventricles may be sensed and paced with a given A—V time.

4. Programmable
   rate, output,
   refractory period sensitivity etc.

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