CARDIAC PACING IN THE MANAGEMENT OF THE SICK SINUS SYNDROME

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Much has been written about Malignant Ventricular Arrhythmias, but the field of malignant atrial arrhythmias remains relatively unexplored. Of particular significance is the group of malignant atrial arrhythmias called the Sick Sinus Syndrome, briefly referred to as S.S.S., a term first coined by Lown (1967) to describe post-cardioversion arrhythmias characterised by "chaotic atrial activity with continual changes in P wave contour with bradycardia interspersed with multiple and recurrent ectopic beats with runs of atrial or nodal tachycardia". The underlying mechanism seems to be an unstable or totally depressed sino-atrial node.

Over a two year period, nineteen patients with clinical and E.C.G. manifestations of S.S.S. were collected at the Department of Clinical Medicine, Medical Unit II, General Hospital, Singapore. Patients with myocardial infarction were excluded as S.S.S., when present in this group, were observed to be mostly a transient phenomenon. The clinical features of this series of S.S.S. have been reported elsewhere (Wan, Lee and Toh, 1972).

Our diagnostic criteria are mainly based on E.C.G. and they include the following:—

TABLE I

CRITERIA FOR DIAGNOSIS OF S.S.S.

1. Evidence of disease of S.A. Node—
   (i) Depressed Sino-Atrial Impulse formation or a total Sinus arrest, with or without
   (ii) poor response to rapid atrial pacing, with often a high threshold.
2. Evidence of disorderly subsidiary pacemaker escapes—either they come on too late or too many of them emerge to vie for dominant pacemaker.
3. The sinus mechanism, when dominant, may be unstable or irritable.
4. To exclude—
   (i) persistent sinus bradycardia in Athletes.
   (ii) transient sino-atrial disorders induced by Vagotonic Drugs.

THE CLINICO-ELECTROCARDIOGRAPHIC CLASSIFICATION OF S.S.S.

When tabulated by reference to the type of arrhythmias and mechanism of ectopism, as shown in Table II, the S.S.S. will fall into definitive groups, each of which possesses distinctive features.

Rhythmwise, it may be bradycardiac, tachycardiac or mixed. The Bradycardiac S.S.S. is often associated with Sino-atrial block (S.A.B.) or Sino-atrial arrest (S.A.A.) with chronic nodal rhythm. It is noteworthy that in 70% of these bradycardiac S.S.S., A-V conduction is impaired. Such A-V dysfunction may be manifest, as in the presence of P.A.T. and paroxysmal atrial fibrillation (P.A.F.) with high grade A-V block, (Cases 19, 4, 13) or occult, concealed by an inherent slow supraventricular pacemaker, requiring stress by rapid atrial pacing to expose the A-V conduction defect, as in cases 18,5.

In the Brady Tachy arrhythmias, the Tachycardia is usually supraventricular in origin, while the bradycardia may be intrinsic by virtue of a depressed S. A. Node, or it may result from Tachycardia-induced post-drive suppression (Lange 1965).

THE CLINICAL PROFILE OF S.S.S.

Based on our analysis, the clinical profile of S.S.S. is characterised by the following features (See Table III).

THE MANAGEMENT OF S.S.S.

The management of patients with S.S.S. has presented a great challenge. As shown in Table IV, response of brady-cardiac S.S.S. to Atropine sympathomimetic agents such as Isoprenaline (including Saventrin) and Orciprenaline are discouraging. Intermittant doses of I.V. Atropine may prevent S.A. block but effects are transient and they may precipitate paroxysmal tachy-arrhythmias. Saventrin and Orciprenaline may have only marginal effects.

On the other hand, digitalisation for supraventricular tachyarrhythmias in S.S.S. does not seem practical in patients with brady-tachyarrhythmias, as it may suppress the already depressed S. A. Node. The same may be said of beta-blockers and other cardiac suppressant drugs currently in use. (Table IV)

With drug therapy in abeyance, cardiac pacing seems to be the only alternative. It accelerates the drug-resistant bradyarrhythmias, thereby preventing the precipitation of escaped tachycardia. Occasionally, it over-drives the recurrent tachyarrhythmias, or failing that, it provides for the liberal exhibition of anti-arrhythmic agents in the presence of co-existing bradycardias.

Ideally, patients with S.S.S. should be paced from the right atrium. Atrial pacing is more physiological than ventricular pacing. It preserves the atrial transport mechanism and avoids the mechanical irritation of the ventricle. Besides, it has the distinct advantage of easy insertion by the bed-side in emergency under the monitor of intra-atrial E.C.G. using the semi-floating electrode. The following case illustrates this point in view (Fig. 1).

CASE REPORT

Mdm. L.L. (Pat. No. 8), a 63 year old Chinese woman had symptoms of thyroitoxicosis (untreated) for nine months when she was admitted for syncopal spells from prolonged S.A. arrest, recurring frequently over one week period. In fact, monitoring E.C.G. revealed cyclical rhythms of P.A.T. or P.A.F. terminating abruptly in asystole from prolonged S.A. arrest, the Brady-Tachycardia Syndrome. (Fig. 1) I.V. atropine did not help the S.A. arrest from post-drive suppression, it only served to precipitate further episodes of P.A.F. Over 45 minutes after being monitored, she developed 3 episodes of Stokes-Adams attack from cardiac standstill. As an emergency bedside procedure, an Elecath, unipolar semi-floating electrode was inserted into the mid-right atrium and stable atrial pacing was instituted at 70/min. on fixed pacing for 72 hours, after which, she remained in stable sinus rhythm, as her thyrotoxicosis gradually came under control with carbimazole therapy.

However, atrial pacing is not without disadvantages and it has three setbacks in the management of S.S.S. *In the first instance*, the diseased S.A. Node or right atrium may acquire a high threshold, or they may not respond to atrial pacing at all. To evaluate the sino-atrial responsiveness, MANDEL *et al*, (1971) advocated rapid atrial pacing up to 150/ min. for 3 min. to induce "over-drive suppression" of the S.A. Node; the duration of this post-pacing arrest is regarded as a measure of S.A. inertia. However, we feel that this may be too risk and could precipitate Stoke-Adams' attacks.

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TABLE II

E.C.G. ANALYSIS OF S.S.S.

Case No.	S.A.B.	S.A.A.	Active Ectopic Foci: Atrial	Junct.	Vent.	S.V.T.	P.A.F.	Passive Ectopic Foci (Escapes): Atrial	Junct.	Vent.	S.V.T.	P.A.F.	A-V Block	Group Characteristics
1.	+													Bradycardiac S.S.S. with/without S.A.B. and S.A.A.
2.	+													
16.	+	+			+				+	+				
17.	+	+	+		+					+				
18.		+							+				+	Association with A-V conduction disturbances which may be (i) Occult or (ii) Manifest.
5.	+	+	+		+			+	+				+	
19.		+				+			+				+	
4.							+		+				+	
13.		+				+		+	+				+	
9.	+						+	+	+					Tachycardiac S.S.S.:— Intermittent S.A.B. accompanied by Paroxysmal Supraventricular Tachyarrhythmias
15.	+					+	+							
10.	+								+			+		
11.	+			+		+	+		+					
14.	+					+		+	+		+	+		
12.	+					+			+	+				
7.	+					+	+							
6.	+		+	+		+								
8.	+	+				+	+	+	+		+	+		Brady-Tachyarrhythmias:— S.A.B. + S.A.A. Intermingled with P.A.T. + P.A.F.
3.	+	+				+	+	+						

TABLE III

THE CLINICAL PROFILE OF S.S.S.

1. An unstable or depressed sino-atrial node with disorderly subsidiary pacemaker escapes.
2. Associated disease of atrial myocardium with tendency to P.A.T. and P.A.F.
3. Associated A-V Node dysfunction—"Bi-nodal Disease".
4. Infra-nodal conduction defects seldom encountered, and ventricular myocardium stable.
5. The dominant rhythm may be homogenous (e.g. persistent Sinus Bradycardia) or heterogenous (e.g. Brady-Tachycardia Syndrome).

TABLE IV

RESPONSES OF BRADYCARDIAC S.S.S. TO DRUG THERAPY

Patient No.	Principal Rhythm	I.V. Atropine	Probantheline	Isoprel (Saventrin)	Orciprenaline
1.	S. Bradyc. with S.A.B.	±	0	—	0
2.	Bradyc. S.S.S.	+	0	0	0
3.	Bradyc. with S.A.B.	A.V. Dis-soc.	0	0	±
6.	Brady.—Tachy. Synd.	—	0	0	0
13.	Junct. Bradyc.	+	+	+	+
14.	Junct. Bradyc.	—	0	—	+
15.	Junct. Bradyc.	—	0	0	0
16.	Brady.—Tachy.	+	0	0	0
17.	Brady.—Tachy.	+	+	+	+

In the second place, even when the atria respond adequately to pacing, the pacing impulses may not be conducted effectively across the A-V junction. As mentioned earlier, A-V Node dysfunction can be manifest or occult. To exclude occult A-V dysfunction, His Bundle recording could determine accurately the degree of A-V conduction delay. Failing that, one could carry out rapid atrial pacing up to 130/min. as a stress test to establish the functional integrity or otherwise of the A-V region. As shown earlier in our charts, A-V nodal dysfunction in the bradycardiac S.S.S. supervened in 70% of cases. Our limited experience concurs with those of Onkar *et al*, (1971) who reported prolonged A-V conduction time using His Bundle Electrogram analysis, while Riely *et al* (1970) found 5 out of 11 patients with Sluggish Sinus Node Syndrome to have prolonged PH time using the same technique.

It is thus mandatory to carry out a "Stress Test" or "A Trial of Rapid Atrial Pacing" before embarking upon therapeutic pacing in the management of S.S.S., in order to evaluate the S.A. and A-V nodal functions.

The following case illustrates the importance of "Stress Tests" to evaluate atrial and A-V junction function and its influence on the choice of pacing sites in management of S.S.S. (Fig. 2).

P.S.L. (Pat. No. 5), a 21 year old Chinese girl with thyrotoxicosis suffered from sino-atrial arrests with an unstable atrial pacemaker. She lapsed into persistent sinus bradycardia and developed syncopal spells occasionally, I.V. Atropine accelerated both atrial and nodal beats, but in atrio-ventricular dissociation. Trial of atrial pacing using an Elecath Semi-floating catheter electrode induced the

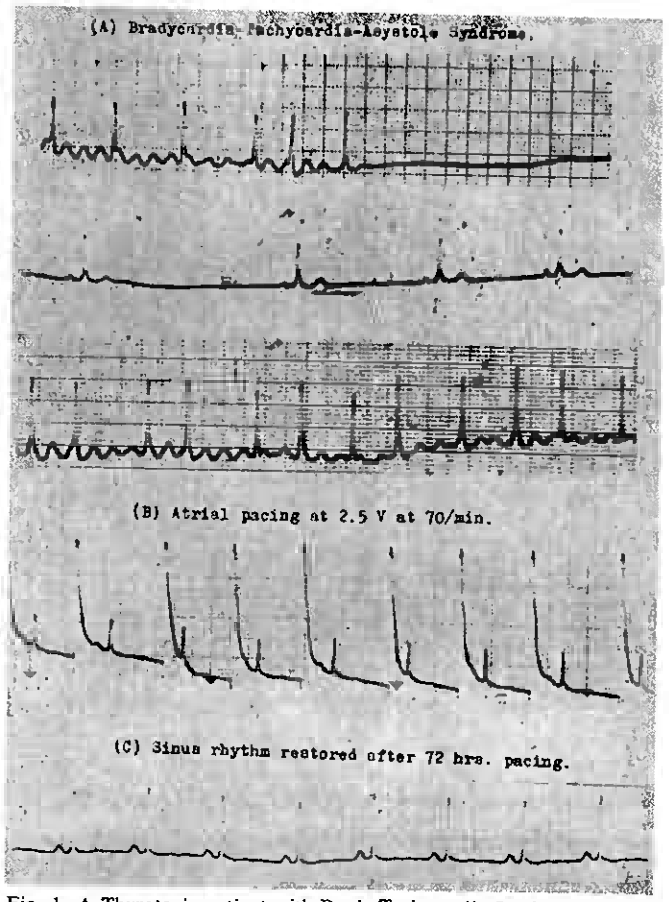

Fig. 1. A Thyrotoxic patient with Brady-Tachycardia Syndrome treated by emergency strial pacing.

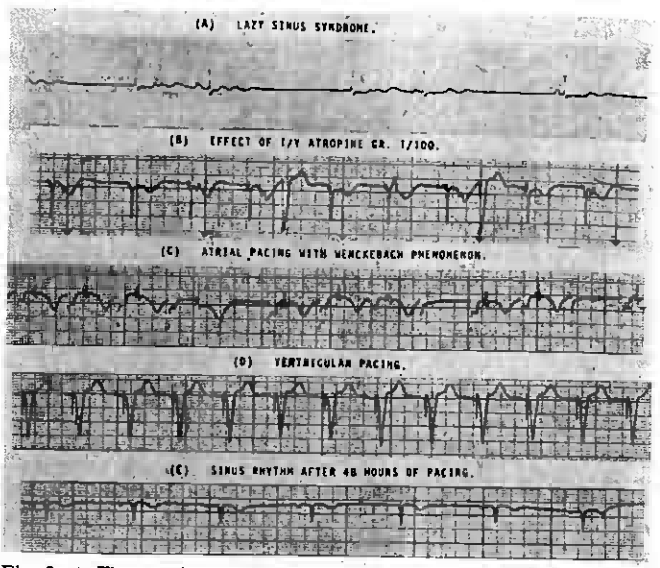

Fig. 2. A Thyrotoxic patient with bradycardiac S.S.S. Atrial pacing at 80/min. induced Wenckebach phenomenon, thereby exposing a "latent" A-V block, and Ventricular Pacing became mandatory.

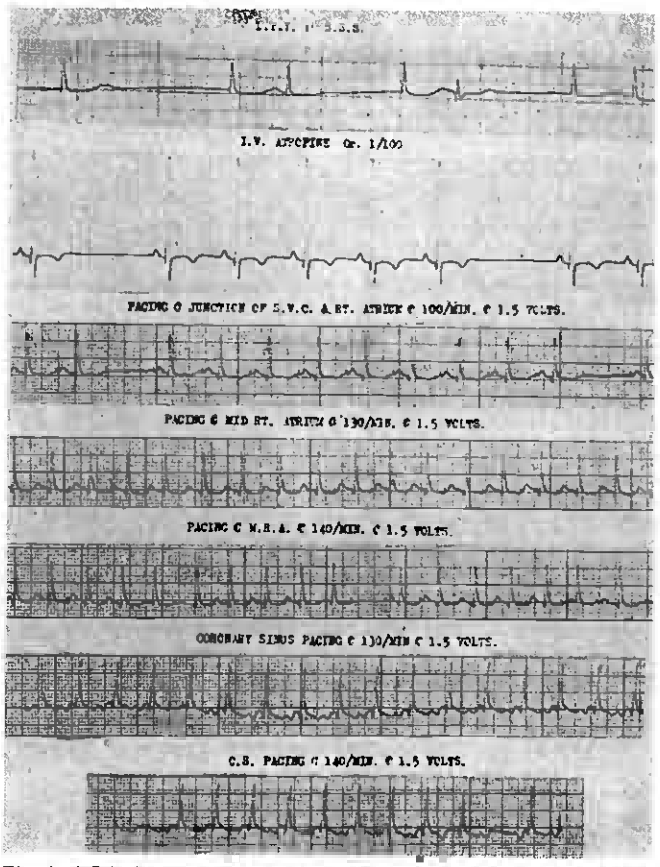

Fig. 3. S.S.S. Stress Test:— Sino-atrial nodal "Innertia" to exogenous drive a 100/min., while mid-right atrial myocardium and coronary sinus responded adequately to paeing up to 130/min.

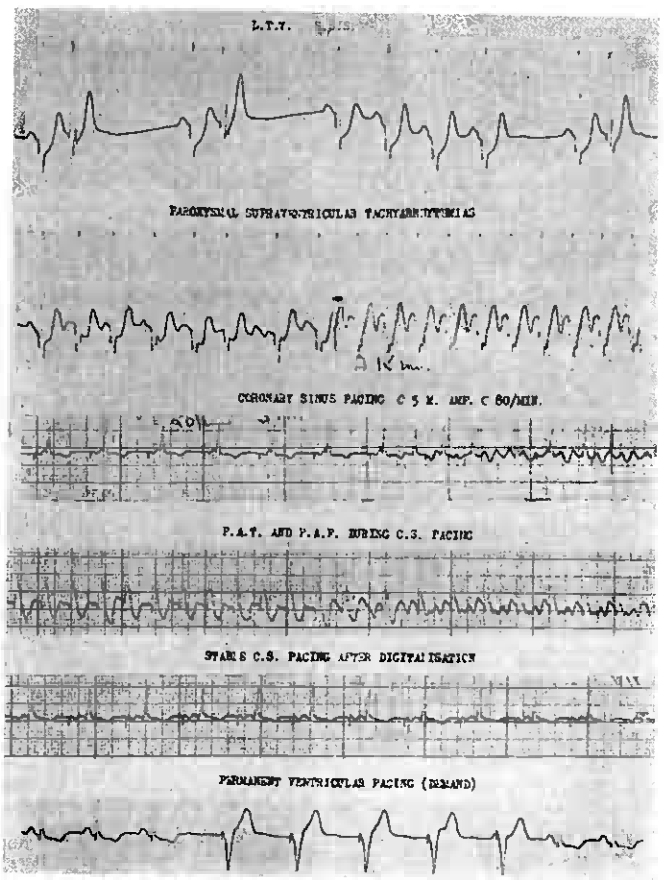

Fig. 4. S.S.S.: Initial combined therapy of coronary sinus pacing with digitalisation. Finally treated by permanent ventricular pacing (demand) due to high coronary sinus threshold.

Wenkebach's phenonmenon at 80/min., thus exposing an occult A.V. block.

For this reason, the Elecath Catheter was "floated" further into right ventricle for pacing, which remained stable and after 48 hours, she returned to sinus rhythm, though somewhat sluggish.

The third disadvantage of atrial pacing relates to a technical problem, that of electrode instability, especially on long term basis. Whereas in ventricular pacing, the papillary musculature of the ventricular wall provides natural anchorage for the wedging electrode, with atrial pacing such natural anchorages are lacking in the relatively smooth-walled right atrium, the atrial electrode practically floats free.

To ensure electrode stability, Arthur Moss (1969) first advocated atrial pacing from the coronary sinus, in which the electrode could be securely wedged. The following case illustrates some of the Pro's Con's and of coronary sinus pacing in the treatment of S.S.S.

L.T.Y. (Pat. No. 3), a 60 year old Chinese barber with Ischaemic Heart Disease was said to have noticed an irregular pulse from childhood. In July 1972, while being monitored in the C.C.U. for chest pain, he was noticed to develop episodes of S.A. arrest, which responded to intermittent doses of Atropine but was later complicated by P.A.T. and P.A.F.

A "Stress Test" by rapid atrial pacing was carried out under screening with an U.S.C.I. bipolar electrode (Fig. 3). The S.A. Node failed to respond to the exogenous drive at 100/min. while the atrium and coronary sinus responded to pacing up to 130/min. with 1:1 A-V conduction, suggesting that there is no A-V conduction defect. The catheter electrode was then wedged in the mid-coronary sinus, the pacing threshold being 2·5 m. amp.

Coronary sinus pacing remained stable till the 3rd day (Fig. 4) when some 7 episodes of recurrent supraventricular tachyarrhythmias emerged and this could not be overdriven with rapid atrial pacing. Meanwhile the pacing threshold was fluctuating between 2·5 to 7 m. amp. It was decided to digitalise this patient and the paroxysmal supraventricular tachyarrhythmias came under control. After one week of coronary sinus pacing, a permanent Demand pacer unit was implanted with a bipolar electrode inserted into right Ventricle. Ventricular rather than the coronary sinus pacing was employed in view of the unstable and alarming high threshold encountered in the latter. Thereafter, the patient remained in good state.

This case illustrates the combination of anti-arrhythmic drugs and pacemaker therapy in the management of S.S.S. with brady-tachyarrhythmias. While pacing prevents cardiac standstill from prolonged S.A. arrest, it also provides for liberal exhibition of suppresant drugs for paroxysmal S.V. tachyarrhythmias. This seems to be the final court of appeal in the therapeutic dilema of S.S.S.

It would seem that coronary sinus pacing ensures electrode stability in atrial pacing at the cost of a high and fluctuating threshold, the risk of thrombosis of the coronary sinus and its perforation (Kitamura *et al* 1971).

Summing up: As shown in Table V: of the 19 patients with S.S.S., 6 required some form of cardiac pacing while 5 were successfully paced atrially including 2 from coronary sinus (cases 16 and 17), by way of temporary measures. Two of these subsequently required ventricular pacing due to impaired A-V conduction (cases 3 and 17), while a third (case 18) needed ventricular pacing outright as an emergency procedure due to chronic atrial fibrillation with slow idionodal rhythm. Of two patients subjected to coronary sinus pacing, one case responded satisfactorily for one week, though a permanent demand ventricular pacer was implanted ultimately for fear of a rise of coronary sinus pacing threshold.

It may be concluded that, sound and logical as it may seem in theory, patients with S.S.S. should have atrial rather than ventricular pacing, ventricular pacing is more often recoursed to in practice for the following reasons:—

1. The presence of associated A-V conduction defects, whether manifest or occult, precludes atrial pacing;
2. Ventricular endocardial pacing provides for reliable electrode stability;

TABLE V

CARDIAC PACING IN S.S.S.

Patient No.	Sites of Pacing			Stress Test			
	Atrial	Coronary Sinus	Rt. Vent.	@S.A.N.	@M.R.A.	@C.S.	A-V Dysfunction
3	+	0	+	0	70/min.	0	+
6	+	0	0	0	130/min.	0	—
13	+	Transient	0	?	110/min.	0	+
16	+	+	+	90/min.	130/min.	140/min.	—
17	+	+	+	—	—	120/min.	+
18	0	0	+	0	0	0	+

S.A.N. = Sino-atrial Node;
M.R.A. = Mid-right atrium;
C.S. = Coronary Sinus.

3. Atrial pacing from the coronary sinus is often associated with a high and fluctuating threshold, while the threshold of ventricular pacing is relatively constant;
4. The ventricles are relatively stable in S.S.S. (as evidenced by the lack of associated P.V.C.'s), and any consideration for ventricular pacing should not be deterred by the fear of mechanically induced ventricular tachyarrhythmias from ventricular irritability;
5. Finally, physicians are, at the moment, more acquainted with the techniques of permanent ventricular rather than atrial pacing.

Perhaps, in a moment, we will learn of the advantages of Bi-focal pacing in this situation by the next learned speaker.

SUMMARY

1. 19 patients of S.S.S. are reviewed. A clinico-electrocardiographic classification of S.S.S. into three groups is proposed, namely, Bradycardiac S.S.S., Tachycardiac S.S.S., and the mixed, and so-called Brady-Tachyarrhythmias.
2. The clinical profile of the S.S.S. is delineated. It is characterised by evidence of disease of S.A. Node, with disorderly emergence of subsidiary pacemakers. The prevailing pacemaker is often unstable, while associated A-V nodal disease (Bi-nodal Disease) is present in 70% of patients with bradycardiac S.S.S.
3. Drug therapy for S.S.S. often yields discouraging results. In severely symptomatic patients, especially the Brady-Tachy syndrome, cardiac pacing is mandatory. However, only some 30% of the whole series required cardiac pacing and the rest, especially the bradycardiac group, could adapt themselves to survive, somewhat incapacitated though.
4. Theoretically, atrial pacing seems the logical and physiological answer to S.S.S. However, this is often deterred by the associated A-V node dysfunction. To exclude this, it is mandatory to perform a "stress test" by rapid atrial pacing prior to therapeutic atrial pacing.
5. Coronary sinus pacing, while it ensures of electrode stability, suffers from the disadvantage of high and fluctuating threshold. Thus permanent (demand) ventricular is usually recoursed to in the management of S.S.S.

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