Upsala J Med Sci 80: 113–117, 1975

Mode of Spontaneous Onset and Termination of Supraventricular Tachyarrhythmias

BENGT FURBERG and LARS NORDGREN

From the Department of Clinical Physiology, University Hospital, Uppsala, Sweden

ABSTRACT

ECG tracings from 16 patients with spontaneous onset and/or termination of supraventricular tachyarrhythmias (SVTA) were studied. Of these recordings, 13 were made with a special technique which gives a high resolution of the atrial waves. At the onset of SVTA, the first atrial wave invariably had an aberrant configuration. The coupling index (coupling interval (P–^{1}P) preceding cycle length) was 0.50 or less in 9 of 15 cases but more than 0.60 in 4 cases. In the 5 cases of onset of atrial fibrillation, the intervals between the first few atrial waves corresponded to a frequency of 300–350 per minute. Acceleration of the atrial activity occurred within the first 30 seconds. At the termination of SVTA, no successive modification of the atrial activity was found. The termination often did not occur at or shortly after a QRS complex. It is concluded that a premature atrial beat—even a single one—with a short coupling interval may well initiate a circus-movement SVTA, while an ectopic atrial beat with a long coupling interval apparently must be followed by repeated rapid discharges from the ectopic focus in order for SVTA to ensue. The functional conditions of the atria may then determine which kind of SVTA eventually results.

INTRODUCTION

The mechanisms responsible for the initiation, maintenance and termination of supraventricular tachyarrhythmias (SVTA), have been the subject of continuing controversy and investigation. Experiments with electrical or chemical stimulation of the atrium, while yielding important information concerning the electro-physiological characteristics of SVTA, have not solved the problems of onset and termination of spontaneous SVTA.

Since the first electrocardiogram showing the onset of atrial fibrillation was presented in 1918 (10), numerous tracings of SVTA onset and termination have been published. Most of these tracings were made with a conventional recording technique, which is not sufficiently sensitive to clearly exhibit changes in the atrial wave configuration. In the present study, a recording technique which gives a high resolution of the atrial waves has been used, thereby offering better possibilities for precise assessment of P wave aberration and P–P intervals.

MATERIAL AND METHODS

The study was performed on ECG tracings from 16 patients (9 men and 7 women, with ages between 19 and 72) with spontaneous onset and/or termination of SVTA. In tracings with more than one bout of SVTA a representative one was selected for the study. The change from sinus rhythm to atrial fibrillation was studied in 5 tracings, to atrial flutter in 8 and to atrial tachycardia in 2. The reversion to sinus rhythm from atrial fibrillation was studied in 2 tracings, from atrial flutter in 6 and from atrial tachycardia in 2. In 3 patients the ECG was recorded with conventional amplification (1 mV=10 mm), lead V_1 and at least 3 other leads being recorded. In 13 patients a special recording technique was used (7), using three bipolar leads. The common reference electrode was placed at the midline of the angle of sternum and the three different electrodes were placed at the highest attainable point of the armpit in the left mid-axillary line (S 1), at the caudal end of the sternal body (S 2) and at a point over the vertebral column at the transthoracic level of the sternal angle (S 3). The three bipolar leads were recorded with conventional amplification, as well as with a tenfold greater amplification (0.1 mV=10 mm), using a 3-channel differential preamplifier and a Mingograph 81 (Siemens-Elema Ltd., Sweden).

For measurements, the system illustrated in Fig. 1 was used. The SVTA type was defined according to the main atrial wave frequency. An atrial wave frequency of 150–210/min was judged as supraventricular tachycardia (SVT), of 220–340 as atrial flutter (AFu) and of 350 or more as atrial fibrillation (AFi).

RESULTS

The tracings of SVTA with the special recording technique permitted analysis of the atrial wave con-

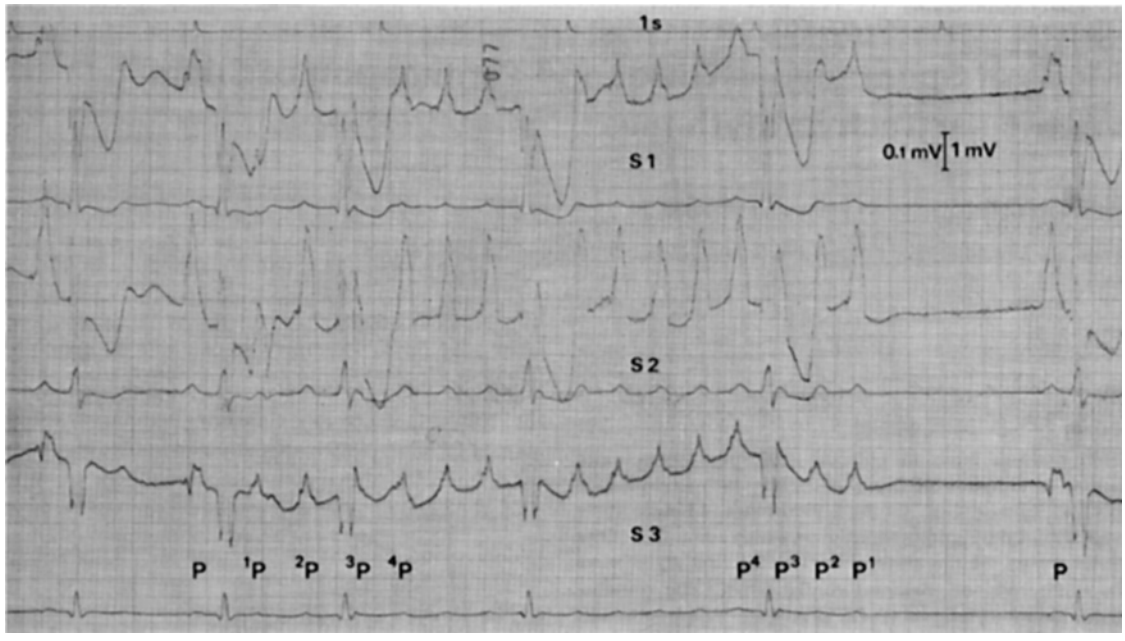

Fig. 1. Onset and termination of atrial flutter (patient no. 9). The nomenclature of the atrial waves is shown. The three special leads S 1–S 3 are recorded with conventional and tenfold amplification.

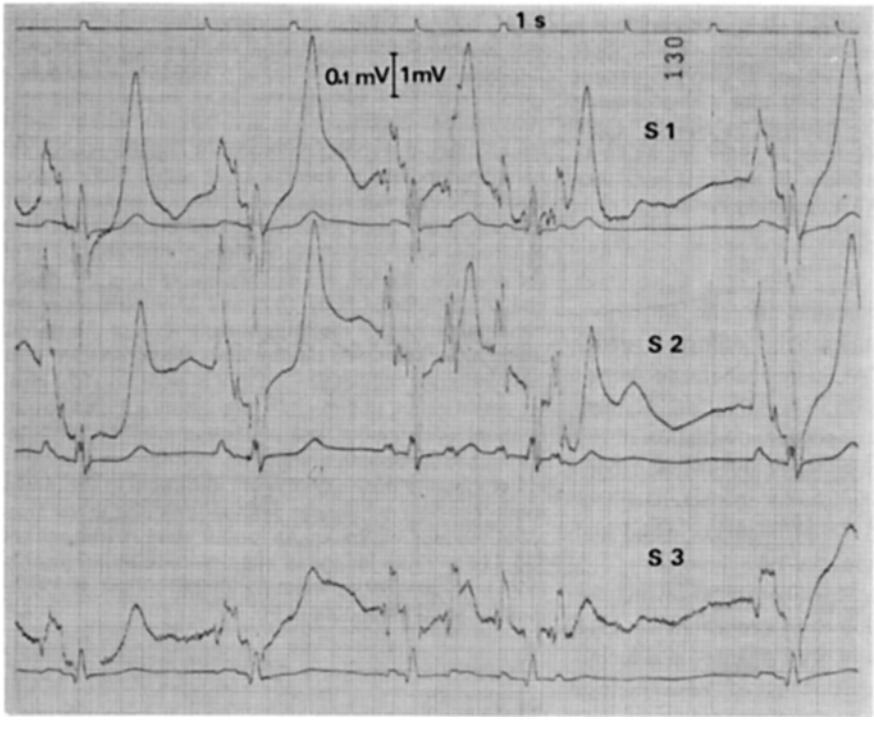

Fig. 2. Short episode of atrial flutter (patient no. 12). Note the long coupling interval.

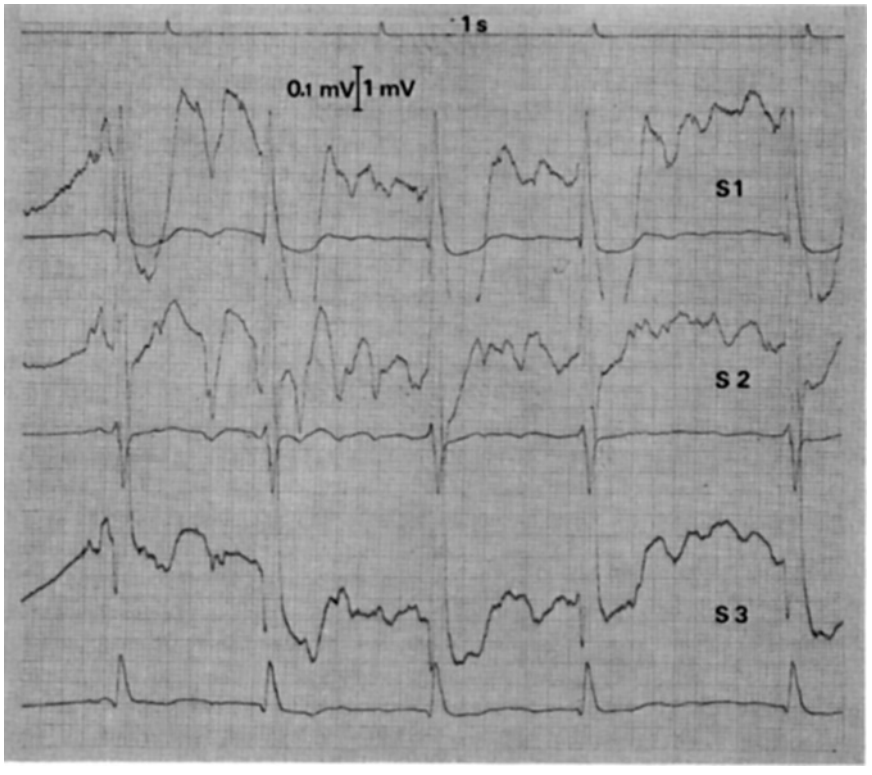

Fig. 3. Onset of atrial fibrillation (patient no. 4). The premature atrial beat is followed by a rapid atrial tachycardia and within 2 s by atrial fibrillation.

figuration and intervals; this had been difficult to obtain with the conventional recording technique. Examples of these new recordings are shown in Figs. 1–3.

At the onset of SVTA, the first atrial wave (^{1}P) invariably had an aberrant configuration in comparison to the ordinary P wave. The following atrial waves (^{2}P–^{n}P) of the SVTA had the same configuration as ^{1}P in the 2 SVT cases and in 6 of the 8 AFu cases. In the 5 AFi cases the configuration changed. In 9 of the 15 cases, the 'coupling index' (the ratio of coupling interval (P–^{1}P) to preceding cycle length (P–P)) was 0.50 or less, while it was more than 0.60 in 1 AFi case, in 2 AFu cases and in 1 SVT case. Acceleration of the atrial activity after ^{4}P was seen in the 5 AFi cases, in 3 of the AFu cases and in none of the SVT cases. The final atrial wave frequency was always obtained within 30 s.

The termination of SVTA occurred in 5 of the 10 cases studied within 30 cs from the beginning of a QRS complex. The point of termination was defined as the start of the P^1 wave plus the P^2–P^1 interval. There was no deceleration of the last few atrial impulses before the termination and the form of the atrial waves did not change. The first atrial wave after termination of SVTA exhibited an ordinary configuration in all but one case.

DISCUSSION

When dealing with the mechanism of SVTA onset and maintenance, some authors have concluded that at least SVT and AFu, and possibly AFi, are evoked by an ectopic atrial focus with a high impulse-formation rate. Other authors have concluded that SVTA depends on the establishment of an atrial circus movement with or without engagement of the atrio-ventricular node. From experimental studies there is evidence that SVTA can result from either of these mechanisms. The arguments in favour of both hypotheses have been reviewed by Hecht et al. 1953 (4), Scherf, Schaffer & Blumenfeld 1953 (11), Katz & Pick 1960 (5), Rytand 1966 (8) and Guiney & Lown 1972 (3). Studies of the

Table I. *Onset of supraventricular tachyarrhythmia*

Patient no.	Ordinary P–P, cs	P–^{1}P (cs)	^{1}P–^{2}P (cs)	^{2}P–^{3}P (cs)	^{3}P–^{4}P (cs)	Final atrial frequency (waves/min)	$\frac{P-{}^{1}P}{P-P}$	Constant configuration of ^{1}P–^{n}P
Sinus rhythm to atrial fibrillation, n=5								
1	186	38	20	17	16	400	0.20	–
2	83	40	17	18	17	390	0.48	–
3	65	43	19	17	16	410	0.66	–
4	110	26	26	26	19	430	0.24	–
5	80	28	19	18	19	360	0.35	–
Sinus rhythm to atrial flutter, n=8								
6	126	38	22	20	20	340	0.30	+
7	84	46	30	20	20	330	0.55	+
8	108	54	27	25	25	230	0.50	+
9	80	35	26	26	26	280	0.44	+
10	80	40	28	19	20	280	0.50	–
11	81	52	28	21	19	330	0.64	–
12	84	79	29	26	27	230	0.94	+
13	216	112	28	22	21	270	0.52	+
Sinus rhythm to atrial tachycardia, n=2								
14	70	49	38	36	37	160	0.70	+
15	98	40	39	40	41	150	0.41	+

spontaneous onset and termination of SVTA may have some bearing on this problem. Killip & Gault (6) studied changes from sinus rhythm to AFi in 14 patients. An atrial premature discharge preceded the onset of AFi in all 14 episodes. In 10 of these the premature P wave was immediately followed by AFi. In the other 4 cases the premature P wave appeared to initiate a brief run of SVT (less than 6 beats) which accelerated to AFi. The relative prematurity of ectopic atrial beats was expressed by calculating the coupling index. The coupling index was less than 0.50 in 9 of the 14 patients. Killip & Gault thus concluded that a spontaneously occurring atrial premature impulse may initiate AFi. When the coupling index is less than 0.50, the chance of ensuing AFi is high; when it is greater than 0.60, the chance of ensuing AFi is small.

Bennett & Pentecost (1) studied the onset of AFi in 8 patients with acute myocardial infarction. One intra-atrial and one surface lead were registered. A

Table II. *Termination of supraventricular tachyarrhythmia*

Patient no.	Ordinary P–P, cs	P^{4}–P^{3} (cs)	P^{3}–P^{2} (cs)	P^{2}–P^{1} (cs)	P^{1}–P (cs)	$\frac{P^{1}-P}{P-P}$	Termination within 30 cs from beginning of QRS
Atrial fibrillation to sinus rhythm, n=2							
5	80	19	20	22	83	1.04	+
16	69	17	16	16	52	0.75	+
Atrial flutter to sinus rhythm, n=6							
6	126	18	18	16	27	0.21	–
8	108	27	25	25	86	0.80	+
9	80	21	22	20	104	1.30	–
11	81	20	20	22	96	1.19	–
12	84	29	26	27	96	1.14	+
13	216	22	22	22	132	0.61	+
Attrial tachycardia to sinus rhythm, n=2							
14	70	36	37	36	76	1.09	–
15	98	39	40	41	102	1.04	–

total of 32 episodes were studied, and on each occasion the arrhythmia was preceded by a premature atrial beat. The premature atrial beat was always followed by a rapid regular atrial tachycardia of variable duration with a rate of approximately 340 beats/min; on some occasions the tachycardia lasted as little as 1 or 2 s, but at other times the duration was up to 30 s.

Bigger & Goldreyer (2) studied the repeated onset of paroxysmal SVT in 5 patients. The SVT always began with an atrial premature depolarization (APD). Spontaneous episodes of SVT were initiated only by APDs occurring in the relative A–V refractory period, while electrical stimuli during the atrial vulnerable period did not elicit SVT. From these and other findings Bigger & Goldryer concluded that paroxysmal SVT is most often due to reentry utilizing the A–V conduction system.

The results of the present study in general correspond well with the results of the earlier reports. The first premature atrial wave was shown to be aberrant in all cases studied and the coupling interval was in most cases short. The coupling index, however, was more than 0.60 in 4 of our cases (Fig. 2 shows one of these cases). In these cases the first premature atrial wave could scarcely have occurred during the vulnerable period of the atria or the relative refractory period of the A–V conduction system. In these cases, therefore, the onset of SVTA probably depended on a rapidly firing ectopic focus rather than on circus movement.

Our study of the termination phase indicates that there is not a successive modification of atrial activity during the last seconds of a SVTA: the SVTA always ended abruptly in our cases. Rytand (9) reported similarly for 3 cases of AFu, while Bennett & Pentecost (1), in their study of AFi in patients with acute myocardial infarction, found that a change of atrial wave form in the intra-atrial lead preceded the cessation of AFi on all 28 occasions observed.

Several explanations for the interruption of SVTA have been offered, for example, exit block from a rapidly firing ectopic focus or interruption of a circus movement with or without engagement of the atrio-ventricular node. One possible cause of such interruption could be retrograde depolarization of the atrio-ventricular node. In the latter case, the SVTA should be expected to terminate at or shortly after a QRS complex. This was observed in only 5 of our cases of AFi or AFu, so that this explanation is probably not the only one.

Our results thus indicate that the theory of circus movement does not explain the mode of spontaneous onset or termination of SVTA in all cases. Onset and maintenance of SVTA in some cases may perhaps be governed by different mechanisms, the onset being initiated from an atrial ectopic focus and a circus movement being responsible for the maintenance. The importance of the vulnerable period of the atria could be that one single ectopic and premature atrial impulse occurring during the vulnerable period of the atria may initiate a circus movement SVTA, while an ectopic atrial beat with a long coupling interval must be followed by repeated rapid discharges from the ectopic focus in order for SVTA to ensue. The functional conditions of the atria may then determine which kind of SVTA eventually results.

REFERENCES

1. Bennett, M. & Pentecost, B.: The pattern of onset and spontaneous cessation of atrial fibrillation in man. Circulation *41:* 981, 1970.
2. Bigger, T. & Goldreyer, B.: The mechanism of supraventricular tachycardia. Circulation *42:* 673, 1970.
3. Guiney, T. & Lown, B.: Electrical conversion of atrial flutter to atrial fibrillation. Brit Heart J *34:* 1215, 1972.
4. Hecht, H., Katz, L., Pick, A., Prinzmetal, M. & Rosenblueth, A.: The nature of auricular fibrillation and flutter. A symposium. Circulation *7:* 591, 1953.
5. Katz, L. & Pick, A.: Current status of theories of mechanisms of atrial tachycardias, flutter and fibrillation. Progr Cardiov Dis *2:* 650, 1960.
6. Killip, T. & Gault, J.: Mode of onset of atrial fibrillation in man. Am Heart J *70:* 172, 1965.
7. Nordgren, L.: A new method to study atrial activity—intended for clinical use. Acta Soc Med Upsal *74:* 186, 1969.
8. Rytand, D.: The circus movement (entrapped circuit wave) hypothesis and atrial flutter. Ann Int Med *65:* 125, 1966.
9. Rytand, D.: Electrocardiographic patterns at the termination of atrial flutter. Am Heart J *74:* 741, 1967.
10. Semerau, M.: Über Rückbildung der Arrhythmia Perpetua. Arch Klin Med *126:* 161, 1918.
11. Scherf, D., Schaffer, A. I. & Blumenfeld, S.: Mechanism of flutter and fibrillation. Arch Intern Med *91:* 333, 1953.

Received November 28, 1974

Address for reprints:

Bengt Furberg, M.D.
Department of Clinical Physiology
Central Hospital
S-800 07 Gävle
Sweden