Coexistence of Sick Sinus Rhythm and Atrial Flutter-Fibrillation

JOSEPH ANTHONY C. GOMES, M.D., PRITPAL S. KANG, M.D., MARIANNE MATHESON, R.N., WILLIAM BRADLEY GOUGH, JR., PH.D., AND NABIL EL-SHERIF, M.D.

SUMMARY A 58-year-old man with hypertensive cardiovascular disease and atrial flutter underwent electrophysiologic studies, including multiple intra-atrial recordings and atrial stimulation. Although the surface ECG suggested the presence of atrial flutter, intra-atrial recordings demonstrated the presence of (1) sinus-like rhythm localized to an area of approximately 5 mm in and around the region of the sinus node, which was protected by entrance block; (2) flutter and/or fibrillation of the remaining parts of the right atrium; (3) fibrillation of the left atrium; and (4) transient degeneration of flutter into fibrillation at right atrial sites, with predominant flutter activity. Although a major part of the right atrium was in flutter and/or fibrillation, we could assess sinus node function by overdrive stimulation of the area of sinus node activity. Sinus node function studies revealed an underlying sick sinus syndrome.

THE DIAGNOSIS of atrial arrhythmias is essentially based on the analysis of the configuration, timing, and rate of P waves on the surface ECG. However, recent studies in selected patients have demonstrated dissimilar atrial rhythms with direct intra-atrial recordings otherwise not discernible on the surface ECG.1-9 In this paper we report the electrophysiologic findings in a patient in whom the ECG revealed atrial flutter and intra-atrial recordings demonstrated the presence of sinus-like rhythm in and around the region of the sinus node, flutter and/or fibrillation of the remaining parts of the right atrium and fibrillation of the left atrium. We also assessed sinus node function, although a major part of the right and left atria were in flutter and/or fibrillation. Assessment of sinus node function revealed an underlying sick sinus syndrome.

Case Report

A 58-year-old man with hypertensive cardiovascular disease was admitted to the Medical Service of the Brooklyn Veteran’s Administration Medical Center for uncontrolled hypertension, congestive heart failure and atrial flutter of recent onset. A 12-lead ECG suggested the presence of atrial flutter at a rate of 240 beats/min, with a ventricular response of 120 beats/min and poor R-wave progression from V1 to V4 (fig. 1). Chest x-ray film showed cardiomegaly and pulmonary congestive changes. Echocardiogram revealed left atrial and left ventricular enlargement. The patient's hypertension and congestive heart failure responded promptly to medical therapy. He was then referred to the cardiology service for elective electrical conversion of the atrial flutter after medical conversion with digitalis and quinidine had failed.

Electrophysiologic Studies

The patient underwent electrophysiologic studies to convert the atrial flutter by overdrive atrial stimulation. The procedure was explained to the patient, who gave signed consent. The electrophysiologic studies were performed after withholding digitalis and quinidine for 2 days. Serum digoxin level was 0.5 ng/ml on the day of the study. Two quadrupolar #6F USCI catheters with 10-mm interelectrode distance were introduced percutaneously into the femoral vein and positioned in the right atrium. Three standard ECG leads and intra-atrial electrograms, filtered at frequency settings of 40–500 Hz and time lines generated at 40, 200 and 1000 msec, were displayed on a multichannel oscilloscope (Electronics for Medicine VR12) and recorded on a tape recorder (HP#3698A) and on thermal paper at paper speeds of 50, 100 and 150 mm/sec. The right and left atria were mapped by sequentially positioning the catheters in the high, middle, low and septal right atrium and the coronary sinus.

When one of the two catheters was positioned in the high right atrium in close proximity to the location of the sinus node, a combination of the distal two poles of the catheter recorded a distinct atrial electrogram at a cycle length of 1170–1360 msec (rate 44–51 beats/min), whereas the two proximal poles of the same catheter recorded atrial activity suggestive of atrial flutter-fibrillation (fig. 2A). The electrogram recorded with the distal poles had no relationship with the flutter activity or the QRS complex. The proximal and distal connections of the second catheter, when positioned in the low and septal right atrium and the coronary sinus, recorded, respectively, atrial activity suggestive of atrial flutter (fig. 2A), atrial activity suggestive of atrial flutter-fibrillation (fig. 2B) and atrial fibrillation (fig. 2C). Variations in rates at sites of more regular atrial activity and transient degeneration to atrial fibrillation were often observed (figs. 2A, B and 3A–C). We believed that the distinct atrial electrogram observed in the region of the high atrium using the distal two poles of the catheter (fig. 2A) was sinus node activity. The area of the latter recording was then mapped by advancing the electrode catheter.

From the Cardiology Department, Brooklyn Veterans Administration Medical Center, and Downstate Medical School, State University of New York, Brooklyn, New York.

Address for correspondence: Joseph A.C. Gomes, M.D., Electrophysiology Laboratory, Department of Cardiology, Brooklyn Veterans Administration Medical Center, Brooklyn, New York 11209.

Received December 5, 1979; revision accepted May 7, 1980.

Circulation 63, No. 1, 1981.
located in the high atrial region both cephalad and caudally. When the catheter was advanced cephalad by approximately 5 mm, sinus node activity was noted in the proximal poles (fig. 3A); pulling back the catheter to its original position revealed sinus node activity in the distal poles (fig. 3B), and pulling the catheter more caudally by approximately 5 mm revealed irregular decremented atrial activity suggestive of atrial fibrillation (fig. 3C). To assess the effects of autonomic modulation on the atrial activity in the region of the sinus node, more maneuvers were performed. Carotid sinus massage revealed slowing of the atrial rate (fig. 4A). Atropine, 1 mg, demonstrated speeding of the atrial rate (fig. 4B) from an average rate of 47 beats/min to 54 beats/min. We also attempted to suppress the atrial activity and assess its recovery time by positioning the second catheter in close proximity to the distal poles of the first catheter (fig. 5). Recovery times were assessed by pacing for 1 minute at cycle lengths of 1100, 900, 700 and 600 msec during control and after atropine. The corrected recovery time was prolonged during control and decreased after atropine. The longest corrected recovery time during control was 850 msec after pacing at a cycle length of 900 msec (fig. 6A). The longest corrected recovery time after atropine was 500 msec after pacing at a cycle length of 700 msec (fig. 6B). In addition, secondary pauses greater than 4 seconds were often observed (fig. 6A).

Atrial pacing of the low atrium at rates greater than those of atrial flutter resulted in transient capture of the respective atrial site, but did not result in capture of the area of the sinus node. The flutter could be transiently converted to fibrillation by rapid atrial pacing but could not be converted to sinus rhythm despite repeated attempts at pacing different right atrial sites.

To assess the reproducibility of our findings, the catheters were left in situ and the next day, the recordings were reattempted. The recordings were consistent with those of the previous study. DC cardioversion was attempted with the catheters in place. It resulted in transient conversion to an ectopic atrial rhythm, but the patient immediately reverted back to atrial flutter-fibrillation.

**Discussion**

Since Schrumpf's original publication of the first ECG that he interpreted to represent atrial dissociation in a patient with digitalis toxicity, several clinical studies have shown the existence of atrial dissociation and dissimilar atrial rhythms in man. Despite electrocardiographic observations, the existence of dissimilar atrial rhythms was questionable because direct intra-atrial recordings were rarely performed. However, recent observations with the use of intra-atrial electrode catheter recordings in selected patients have revealed (1) atrial fibrillation in one atrium and atrial flutter in the other atrium; (2) no recordable atrial activity in one atrium and a tachyarrhythmia recorded in the other atrium; (3) atrial flutter degenerating into atrial fibrillation in a segment of the same atrium; (4) paroxysmal left atrial tachycardia with separation of left and right atrial components of the P waves by an isoelectric period due to intra-atrial
conduction delay; and (5) right atrial standstill and left atrial activity without contraction.

The surface ECG in our patient suggested atrial flutter; however, intra-atrial recordings from multiple atrial sites revealed distinct atrial activity at a rate of 44–51 beats/min in and around the region of the sinus node, atrial flutter and/or fibrillation in remaining segments of the right atrium, atrial flutter transiently degenerating into atrial fibrillation in the same segments of the right atrium, and atrial fibrillation of the left atrium. While both right and left atria were in

flutter and/or fibrillation, a discrete island of atrial tissue approximately 5 mm around the region of the sinus node was in sinus rhythm. Although it is difficult to establish unequivocally sinus node activity without direct recordings of a sinus node electrogram, the atrial activity around the region of the sinus node was indeed sinus-like, as suggested by (1) slowing of the atrial rate by carotid sinus massage; (2) speeding up of the atrial rate after the administration of atropine; (3) presence of secondary pauses after overdrive suppression, and (4) localization of the atrial activity in and

FIGURE 2. Intra-atrial electrograms from different right atrial sites and the coronary sinus. (A) Simultaneous recordings from the high right atrium (HRA) and low right atrium (LRA). The combination of the distal poles of the catheter located in the high right atrium (HRA_L) reveals a distinct atrial electrogram; a combination of the proximal poles (HRA_p) demonstrates atrial activity suggestive of flutter-fibrillation. The atrial electrogram on the HRA_p has no relationship to the flutter activity or to the QRS complex. Recordings from the LRA reveal flutter activity. Flutter activity transiently degenerates into atrial fibrillation at the same recording sites (solid arrows). (B) Simultaneous bipolar recordings from the HRA and septal right atrium (SRA). Note the presence of flutter-fibrillation in the SRA recordings. (C) Simultaneous bipolar recordings from the HRA and coronary sinus (CS). Whereas the HRA_p demonstrates a distinct atrial electrogram and the HRA_p demonstrates atrial flutter, recordings from the CS show atrial fibrillation. L_1, L_2, and V_1 = standard ECG leads; TL = time lines; d = bipolar recordings from the combination of the distal poles of the catheter; p = bipolar recordings from the combination of the proximal poles of the catheter.
FIGURE 3. Mapping of the area of sinus node activity. (A) When the catheter in the high right atrium (HRA) is advanced cephalad by approximately 5 mm, the distinct atrial electrogram originally seen in the distal combination (fig. 1) is seen on the proximal combination (HRAp); the distal connection shows no atrial activity (HRA). Recordings from the second catheter in the low right atrium (LRA) reveal flutter activity with transient degeneration to fibrillation. (B) Pulling back the catheter by approximately 5 mm to its original position reveals the distinct atrial electrogram on the HRA and flutter activity on the HRAp recordings. (C) Pulling the catheter caudally by approximately another 5 mm reveals irregular decremented atrial activity suggestive of atrial fibrillation. Abbreviations: See figure 2.
Figure 4. The effect of carotid sinus massage (CSM) and atropine. (A) CSM induced slowing of the atrial rate; the longest pause was 2820 msec. (B) Administration of 1 mg of atropine intravenously resulted in a decrease in cycle length from a control value of 1260 msec to 1104 msec. The atrioventricular nodal response to both CSM and atropine, as reflected in changes of the average ventricular rate, was normal. Abbreviations: See figure 2.

Figure 5. Simultaneous intra-atrial electrograms from the region of the high right atrium (HRA) obtained by positioning the two catheters in close proximity to each other. That the distal poles of catheter 2 (HRA\textsubscript{d}\textsuperscript{2}) are in close proximity to those of catheter 1 (HRA\textsubscript{d}\textsuperscript{1}) is evidenced by the fact that the atrial electrogram on HRA\textsubscript{d}\textsuperscript{1} and HRA\textsubscript{d}\textsuperscript{2} are coincident with each other. The distal poles of the second catheters (HRA\textsubscript{d}\textsuperscript{2}) were used to stimulate the region of the HRA showing sinus node activity for assessment of sinus node function. Abbreviations: See figure 2.
The observation that block around the left atria may be caused by fibrosis in the region of the sinus node. Coexistence of sinus-like activity with flutter-fibrillation of the right and left atria suggests that a mass of atrial tissue surrounding the sinus node was protected by entrance block. The observation that capture of the low atrium did not result in capture and suppression of the sinus node area supports this hypothesis. Although we do not know whether the causative lesion for entrance block in and around the sinus node is an anatomic or physiologic barrier, it is probably related to considerable fibrosis in the approaches to the sinus node and atrial myocardium.

We could assess sinus node function in our patient, although a major part of the right and left atria were in flutter and/or fibrillation. The presence of an underlying sick sinus syndrome is supported by (1) the presence of sinus bradycardia; (2) dampened response to atropine; (3) prolonged corrected sinus node recovery time, and (4) secondary sinus node pauses after overdrive suppression.

Segmental right atrial flutter-fibrillation is probably related to the presence of isolated islands of dissociated atrial activity. Impulses from the fibrillating left atrium or parts of the right atrium could excite other areas of the right atrium to produce regular activity, the latter depending on the frequency of stimulation and refractory period of the right atrium. 

Variations in rates at sites of more regular atrial activity and transient degeneration to atrial fibrillation support the latter possibility. However, as indicated by Wells et al., the atrial activity suggestive of atrial flutter and/or flutter-fibrillation may represent a type I atrial fibrillation or type IV atrial fibrillation.

Atrial stimulation is one method for converting atrial flutter to sinus rhythm. However, previous studies have indicated a variable success rate in converting flutter by atrial stimulation. Our inability to convert atrial flutter to sinus rhythm may be related to the presence of dissimilar atrial rhythms, as suggested by Zipes and DeJoseph, or to the possibility that dissimilar atrial rhythm may be one of the expressions of atrial fibrillation.

Clinical Implications

Our findings and those previously reported suggest that (1) the ECG may not accurately reflect atrial activity; (2) sinus rhythm may coexist with atrial flutter and or fibrillation; and (3) it may be possible to assess sinus node function and diagnose the presence of sick sinus syndrome even in the presence of atrial flutter-fibrillation if the area of the sinus node is protected by entrance block. An underlying sick sinus syndrome warrants a temporary pacemaker before attempted conversion by DC shock.

Acknowledgment

The authors acknowledge Theresa Luppowitz for preparation of the manuscript and Mike C.S. Yu for the art work.

References

Coexistence of sick sinus rhythm and atrial flutter-fibrillation.
J A Gomes, P S Kang, M Matheson, W B Gough, Jr and N El-Sherif

Circulation. 1981;63:80-86
doi: 10.1161/01.CIR.63.1.80
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/63/1/80

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/