Entrainment and Interruption of Atrial Flutter with Atrial Pacing

Studies in Man Following Open Heart Surgery

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SUMMARY To examine the question of why the pacing rate and duration of atrial pacing are crucial factors in the successful interruption of atrial flutter, studies were performed on 30 patients in the period following open heart surgery. In each patient the diagnosis of atrial flutter was made using a pair of wire electrodes placed on the right atrial epicardium at the time of operation and brought out through the anterior chest wall. The same electrodes were used for atrial pacing. Pacing faster than the spontaneous rate of the atrial flutter which failed to interrupt the atrial flutter was associated with transient entrainment of the atrial flutter up to the atrial pacing rate. Atrial flutter was interrupted successfully when the atria were paced at a rate which was too fast for the atrial flutter to follow. This was heralded by the conversion of previously negative flutter waves to positive atrial complexes in ECG lead II. When pacing the atria at a constant rate, 2-22 seconds with a mean of 10 seconds were required to interrupt the atrial flutter.

WE DEMONSTRATED RECENTLY that classical atrial flutter occurring in patients following open heart surgery always can be interrupted with rapid atrial pacing.1,2 In these studies, it became clear that a critical pacing rate above the spontaneous rate of the atrial flutter had to be achieved before atrial flutter could be interrupted. Furthermore, it was equally clear that a critical duration of pacing at the appropriate rate also was required. The present report fully describes some later results of those preliminary observations.

Methods

Thirty patients who developed atrial flutter in the immediate period following open heart surgery were studied. Informed consent was obtained for each study. The age range of patients was 21 to 67 years with a mean age of 52 years; four patients were operated on for aortic valve disease, five patients for mitral valve disease, one patient for aortic and mitral valve disease, 15 patients for coronary artery disease, one patient for coronary artery disease and aortic valve disease, two patients for asymmetric hypertrophy of the heart, one patient for tetralogy of Fallot, and one patient for a secundum atrial septal defect. At the time of the study, 13 patients were receiving digoxin, two patients procaine amide, five patients digoxin and procaine amide, three patients digoxin and quinidine, one procaine amide and quinidine, and six patients were receiving no cardiovascular drugs.

At our institution a pair of Teflon-coated, stainless steel wire electrodes is routinely implanted on the right atrial epicardium around the sinus node and brought out through the anterior chest wall for diagnostic and therapeutic use in the postoperative period.3,4 These electrodes were utilized to establish the diagnosis of atrial flutter and to pace the atria. Patients were divided into two groups. Group I (17 patients): After the diagnosis of atrial flutter was established, bipolar atrial pacing was initiated at a rate about 10 beats faster than the spontaneous atrial rate. After demonstrating that during the pacing the atrial rate increased to the pacing rate, atrial pacing was continued for at least 30 seconds. Then, the atrial pacing was either 1) terminated abruptly, or 2) slowed gradually, or 3) the pacing rate was changed in a one-step fashion to 120 beats/min. If the atrial flutter was not interrupted successfully, atrial pacing was repeated, the pacing rate being increased in increments of 10 beats/min. Group II (13 patients): After the diagnosis of atrial flutter was established, atrial pacing was initiated at a rate about 10 beats/min faster than the spontaneous rate. Once the atrial rate had been demonstrated to increase to the pacing rate, the pacing rate was increased gradually to a target rate an additional 10 beats/min faster, i.e., 20 beats/min faster than the atrial flutter rate. When that increment had been reached, the rate was gradually slowed. If the atrial flutter was not interrupted successfully, atrial pacing was repeated, the target rate being increased in increments of 10 beats/min. For patients in both groups I and II, the pacing study was terminated with the successful interruption of the atrial flutter. Pacing was performed using a Medtronic Model 1349A or a Model 5320 or a Model 1379 battery-powered pacemaker.

ECGs and electrograms were recorded on a single-channel Hewlett-Packard ECG Machine Model 1151B or on a Medical Systems Corporation three-channel ECG machine Model #DU-35 or on an Electronics-for-Medicine DR12 switched beam oscilloscopic recorder. When using the Hewlett-Packard and Medical Systems Corporation ECG machines, the paper recording speeds were either 25 or 50 mm/sec. When using the Electronics-for-Medicine recorder, paper recording speeds were either 50 or 100 mm/sec. When using the Medical System Corporation and Electronics-for-Medicine recorders, either the bipolar atrial electrogram or the stimulus artifact was recorded simultaneously with at least two ECG leads. When using the DR12 Electronics-for-Medicine recorder, the data were recorded simultaneously.

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on magnetic tape (Honeywell Model 5600) for later playback and analysis. The bipolar atrial wires always were isolated from ground and from the recording devices. When using the DR12 Electronics-for-Medicine recorder, ECGs were recorded between a band pass of 0.1–500 Hz, and electrograms were recorded between a band pass of 0.1–500 Hz and/or 12–500 Hz. When using the other recorders, all ECGs and electrograms were recorded between a band pass of 0.1–100 Hz.

For each study, the following beat-to-beat measurements were made: atrial cycle length (A-A interval) from the interval between recorded bipolar atrial electrograms; stimulus cycle length (S-S interval) from the interval between recorded stimulus artifacts; and ventricular cycle length (R-R interval) from the interval between QRS complexes of the recorded ECG. For all data recorded on magnetic tape, the intervals were measured by two Hewlett-Packard 5300A interval counters connected in series. The output of each counter then was connected via a Hewlett-Packard K105055A duplexer to a character generator which printed out the intervals in milliseconds on the Electronics-for-Medicine recorder. These interval measurements were crosschecked randomly using a vernier measuring device with an accuracy of 1 msec at a paper recording speed of 100 mm/sec. For all other data, intervals were measured directly from the records using the same vernier device.

Results

Critical Pacing Rate

Figures 1–6 illustrate a representative study from a group I patient. In figure 1, ECG lead II is recorded simultaneously with a bipolar atrial electrogram. Atrial flutter is demonstrated clearly at a relatively slow atrial rate (mean rate of 230 beats/min, the corresponding mean beat-to-beat atrial cycle length being 264 msec) with 2:1 A-V conduction. Atrial pacing was initiated at a cycle length of 254 msec (rate of 235 beats/min). The flutter wave interval or f-f interval promptly shortened to the pacing interval, i.e., the atrial rate increased, and the R-R interval shortened accordingly as 2:1 A-V conduction was maintained. After 30 seconds of atrial pacing at the cycle length of 254 msec, pacing was abruptly terminated (fig. 2) and the atrial flutter at the previous spontaneous cycle length promptly resumed. During pacing, the flutter waves in the ECG were little different from those during the spontaneous atrial flutter.

Pacing then was initiated at a cycle length of 242 msec, i.e., a faster heart rate. Once again, the f-f interval decreased to the pacing interval, i.e., the atrial rate increased, and the R-R interval shortened accordingly as 2:1 A-V conduction was maintained. Following 30 seconds of atrial pacing at this cycle length, abrupt termination of atrial pacing resulted in prompt resumption of atrial flutter at the same spontaneous cycle length present in the control records (fig. 3). It is even clearer in this record than in figure 2 that the R-R interval was different during pacing from that during spontaneous atrial flutter. Also, during atrial pacing, the morphology of the flutter waves was virtually the same as during the spontaneous atrial flutter. Thus, despite the fact that the atria were being paced from a site near the sinus node which should produce positive P waves in lead II, the morphology of the spontaneous flutter waves in the ECG was maintained. We have interpreted the ability of the atrial flutter to increase its rate to the atrial pacing rate as entrainment of atrial flutter.

The atria then were paced at a cycle length of 232 msec. Again, the f-f interval shortened to the pacing interval, i.e., the atrial rate increased, and the R-R interval shortened accordingly as 2:1 A-V conduction was maintained. The atrial flutter rate was entrained up to the pacing rate again. Following 30 seconds of atrial pacing at this cycle length, the pacing was abruptly terminated (fig. 4), whereupon the atrial

![Figure 1](http://circ.ahajournals.org/lookup/suppl/doi:10.1161/01.CIR.56.5.738/-/DC1)

**Figure 1.** ECG lead II recorded simultaneously with a bipolar atrial electrogram (AEG) from a patient who developed atrial flutter following open heart surgery. Using a pair of interval counters and a character generator, the beat-to-beat intervals of the atrial electrograms were measured (bottom trace) and printed out in milliseconds on the recording paper (3rd and 4th traces). Using this technique, the millisecond print-out represents the previous beat-to-beat atrial cycle length. Thus, the first atrial cycle length is 265 msec, the second atrial cycle length is 264 msec, the third atrial cycle length is 264 msec, etc. This method of beat-to-beat interval print-out will be the format for figures 2–12 as well. A-A = interval between atrial electrograms, meas = measured. Paper recording speed 50 mm/sec, time lines at 1 second intervals in all figures unless otherwise noted.
flutter promptly resumed. Morphology of the spontaneous flutter waves in the ECG was very little different from that during atrial pacing, except for some minimal change in the flutter wave morphology during pacing. By comparing the position of the stimulus artifact to the flutter waves in this figure with that in previous figures during pacing at slower rates (i.e., at longer cycle lengths), the stimulus artifact is seen to move leftward in its position relative to the negative portion of the flutter waves.

The atria next were paced at a cycle length of 224 msec. Flutter again was entrained so that the f-f interval shortened to the pacing interval, but morphology of the flutter waves was still very little different from that during spontaneous atrial flutter. Figure 5 begins 10 seconds after the onset of

**Figure 2.** ECG lead II recorded simultaneously with the stimulus artifact (Stim) from the same patient as in figure 1 at the termination of 30 seconds of rapid atrial pacing at a cycle length of 254 msec. S-S = stimulus interval, S = stimulus artifact, A = atrial electrogram.

**Figure 3.** ECG lead II recorded simultaneously with the stimulus artifact (Stim) in the same patient as in the previous figures recorded at the end of 30 seconds of pacing at the cycle length of 242 msec.

**Figure 4.** ECG lead II recorded simultaneously with the stimulus artifact (Stim) in the same patient as in the preceding figures at the termination of 30 seconds of atrial pacing at a cycle length of 232 msec.
atrial pacing at this cycle length. The stimulus artifact now appears even earlier relative to the negative portion of flutter waves. With the seventh stimulus artifact in this figure (22 seconds after the onset of atrial pacing), the flutter waves suddenly became positive in lead II. This is what one would have expected when pacing the atria from a site near the sinus node. Panel A of figure 6 is continuous with figure 5, and panel B is recorded two seconds later. With abrupt cessation of rapid atrial pacing, spontaneous sinus rhythm promptly resumed (panel C).

The results from this representative study were consistent in all patients, regardless of the intrinsic rate of the atrial flutter, the type of atrial pacing performed, the method of termination of atrial pacing or the presence or absence of cardioactive drugs. For patients in both groups I and II, atrial flutter never was interrupted successfully until the atrial pacing was sufficiently rapid so that atrial flutter could no longer be entrained to the paced rate. This always was manifest by a change in the morphology of the atrial complexes from negative to positive in leads II, III, and aVF.

Critical Morphology of Flutter Waves

As the atrial pacing rate approached the critical rate for overcoming entrainment, atrial activation in the electrocardiogram often was expressed by a morphology intermediate between that characteristic of the spontaneous atrial flutter and that characteristic of atrial pacing from a high right atrial site. A representative example is demonstrated in figures 7–12 recorded from a group II patient who developed atrial flutter at a rate of about 294 beats/min, the corresponding atrial cycle length being 204 msec (fig. 7). Atrial pacing was initiated at a cycle length of 194 msec and then the pacing cycle length was decreased gradually. As the pacing cycle length was decreased from 175 to 172 msec (fig. 8), the morphology of the flutter waves in lead II changed from negative to biphasic and in lead III from negative to flat. When the pacing cycle length was increased from 169 msec to a cycle length which was longer, i.e., a rate which was slower, than that of the spontaneous atrial flutter, the atrial flutter recurred (fig. 9). Thus, although the morphology of the flutter waves changed when the pacing cycle

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**Figure 5.** ECG lead II recorded simultaneously with the stimulus artifact (Stim) in the same patient as in the preceding figures during atrial pacing at a cycle length of 224 msec. Note that with the 7th atrial beat in this tracing, and after 22 seconds of atrial pacing, the flutter waves suddenly became positive.

**Figure 6.** Panel A is continuous with the record from Figure 5. Panel B is ECG lead II recorded 2 seconds after the tracing in panel A. The first beat in panel C is identical to the last beat in panel B.
length was decreased, the entrainment of atrial flutter was not overcome, i.e., atrial flutter was entrained but not interrupted.

The pacing was repeated, this time decreasing the cycle length, i.e., increasing the pacing rate, more than previously (fig. 10). As the pacing cycle length was decreased from 171–157 msec, the atrial complexes became completely positive in both leads II and III. Now, when the pacing cycle length was increased, i.e., when the pacing rate was decreased, atrial capture was maintained (figs. 11 and 12),
During a pacing cycle length of 221 msec, which is longer than that of spontaneous atrial flutter, the P waves remained positive (fig. 11). As the pacing cycle length was increased further, the atrial flutter did not recur (fig. 12). In fact, atrial capture was maintained as the pacing cycle length was increased to 545 msec, a pacing rate of 110 beats/min. Thus, atrial flutter initially was entrained and finally interrupted by the atrial pacing.

In four group I patients and two group II patients, atrial fibrillation was precipitated by rapid atrial pacing. In each
of these six patients, atrial pacing at rates slower than those which precipitated atrial fibrillation entrained the atrial flutter but failed to interrupt it. This was manifest in the ECG by failure of the flutter waves to become positive in the appropriate leads. Figure 13 illustrates a representative example in a group I patient. Atrial pacing at 400 beats/min simply entrained the atrial flutter but did not interrupt it. When the atria were paced at 410 beats/min, atrial fibrillation was precipitated. For these six patients atrial fibrillation was transient in five, the rhythm becoming sinus in four and returning to atrial flutter in one. In the other patient, atrial fibrillation, which was the patient’s preoperative rhythm, remained stable.

Critical Duration of Atrial Pacing

The duration of rapid atrial pacing also was important in the successful interruption of atrial flutter. In the first example illustrated above, it took 22 seconds of pacing at the cycle length of 224 msec before the atrial complexes in ECG lead II became positive. It was possible to quantitate this duration for 12 of the 17 group I patients studied. In four patients, atrial fibrillation developed, and in one additional patient, the actual ECG recording was stopped when the ECG recording machine ran out of paper. For the latter patient, the initial 12 seconds of pacing were recorded and did not demonstrate any change in the morphology of the atrial complexes in lead II. By the time the ECG recording was resumed, the complexes had become positive. If we include the latter patient with the 12 other patients, the range of the interval from the onset of pacing to the onset of positive atrial complexes in lead II was 2–22 seconds, with a mean of 10 seconds. It was not possible to quantitate this duration for group II patients because of the nature of the ramp pacing technique.

Discussion

Critical Pacing Rate

Atrial flutter clearly can be entrained up to faster rates by rapid atrial pacing. When atrial flutter is so entrained, termination of pacing is not followed by interruption of the atrial flutter because a critical rate of pacing has not been reached. This would seem to explain some reported failures of rapid atrial pacing to interrupt atrial flutter. Examination of these previous studies indicates that in some cases, although the atria were paced at rates faster than the spontaneous rate of atrial flutter, the pacing rate merely entrained the atrial flutter but was not fast enough to interrupt it. In order to interrupt atrial flutter successfully with rapid atrial pacing, the atria must be paced at a rate which is too fast for the flutter rhythm to follow.

Importance of Morphology of the Flutter Waves during Rapid Atrial Pacing

From these studies it appears that atrial pacing from the high right atrium at a proper rate to terminate flutter is associated with a predictable change in the configuration of atrial complexes in lead II. Normally, pacing from sites high in the human right atrium produces positive P waves in leads II, III, and aV_{F}.11, 12 As this study demonstrates, during entrainment of atrial flutter, the atrial complexes retain their spontaneous morphology completely or in considerable part. However, the appearance of positive atrial complexes in leads II, III, and aV_{F} during rapid atrial pacing from high right atrial sites indicates that the atrial flutter has been interrupted. This only occurs after the atrial pacing rate is sufficiently faster than the spontaneous flutter rate.

All previously reported cases we could find which describe conversion of atrial flutter to sinus rhythm by the technique of overdrive rapid atrial pacing were reviewed. The published ECGs were not identified by lead in most instances. Only three published records were found in which lead II in two cases and leads II and III in another case were identified, and in which cessation of rapid atrial pacing was associated with conversion of the atrial flutter to sinus rhythm without going through a period, however brief, of atrial fibrillation.16-18 In each example, the negative flutter waves present in the ECG during spontaneous atrial flutter became positive during the rapid atrial pacing. These cases are important not only because they correspond to our own observations but also because they demonstrate that similar results prevail in nonsurgical patients.

Duration of Atrial Pacing

The reason for a necessary minimal duration of sustained atrial pacing is unclear. This may be related to the location of the atrial pacing wires in our patients.7 For instance, if atrial flutter is generated at any focus (automatic, re-entrant, or something as yet unappreciated) which is located low in the left or right atrium, the distance of the pacing site from the flutter focus may influence the necessary duration.
of pacing at a critical rate which is required to interrupt the atrial flutter. The fact that atrial conduction seems to be slow during atrial pacing may be important in this regard. Similarly, the necessary duration of atrial pacing may relate in part to a beat-to-beat increase in the extent of atrial tissue activated by a wavefront coming from the pacing site, taking a relatively long time until it can overcome atrial activation which results from a wavefront coming from the flutter focus.

The Nature of Transient Entrainment of Atrial Flutter

If atrial flutter is an automatic rhythm, one would expect that rapid atrial pacing would overdrive and suppress the automatic focus, unless an entrance block into the automatic focus prevented this. From such reasoning, transient entrainment of atrial flutter which occurs during pacing is not consistent with the automaticity hypothesis for the pathogenesis of atrial flutter. However, it has been demonstrated in the canine heart that automatic foci tend to accelerate and compete with imposed drives which exceed the spontaneous rates by a relatively small percentage (10-15%). Additionally, if atrial flutter is the spontaneous counterpart of a triggered automatic rhythm recently demonstrated in vitro, and since these latter rhythms may be precipitated and even interrupted by pacing, then the transient entrainment of atrial flutter which we demonstrated could represent a pacing-induced acceleration of triggered automaticity.

On the other hand, if atrial flutter is a re-entrant rhythm, and if the site of re-entry is in the atria, then the rate of atrial flutter likely is dependent upon the atrial refractory period. Since the atrial refractory period is rate-related, pacing at rates more rapid than the spontaneous atrial flutter rate may shorten the atrial refractory period and thereby permit the rate of the atrial flutter to increase to the paced rate, i.e., to become transiently entrained. Still more rapid pacing would decrease the refractory period and prevent the re-entrant loop from being sustained. Clearly, more studies are required before anything but a speculative explanation of transient entrainment can be offered.

Mechanism of Atrial Flutter

As summarized recently atrial flutter is thought either 1) to result from some form of circus movement, i.e., re-entry, probably around the great veins, or 2) to be initiated by an automatic mechanism at a single site or focus. In favor of the single focus hypothesis, atrial flutter has been produced by placing aconitine or delphamine on the atrial epicardium, and flutter waves have been mimicked by pacing the atria from the coronary sinus, and mapping studies in the canine and human heart have suggested that flutter starts from a focus. Also, there have been suggestions based on vector analysis that atrial flutter is initiated at a focus, most likely inferior in the left atrium. Our findings are compatible with either the re-entrant or automatic hypothesis. However, some observations regarding mechanism can be made. The fact that in the ECG there appears to be some atrial fusion prior to the interruption of the atrial flutter, i.e., prior to overcoming the ability of the atrial flutter to become entrained to the pacing rate, demonstrates that the atrial flutter can be sustained in one part of the atrium even when other parts are being depolarized from a site high in the right atrium. This would make less tenable the old hypothesis that atrial flutter is a rhythm which is generated by re-entry around the great veins. It additionally suggests that atrial flutter may be generated at a focus which is low in one of the atria. The latter is supported further by our own studies and those of others on the morphology and polarity of the P waves in leads II, III, and aVF. Therefore, we suggest that whatever the mechanism of the atrial flutter, the arrhythmia is generated inferiorly in either the right or left atrium. This does not imply an automatic mechanism for if one accepts the suggestion that atrial flutter may have a focal origin, it is still possible that the responsible focus consists of a small circus movement around one of the vessels which enters the atria inferiorly, such as the inferior vena cava, or one or more of the pulmonary veins, or the coronary sinus.

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 Transthoracic Ventricular Defibrillation in the 100 kg Calf with Unidirectional Rectangular Pulses

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SUMMARY The effectiveness in reversing ventricular fibrillation of 30 seconds duration of unidirectional rectangular-wave shocks having pulse widths of 0.5 through 64 msec, pulse amplitudes of 35, 50, 70, 100, and 140 amp, and pulse energies of 109 through 1,660 J was studied in 3,303 transthoracic fibrillation-defibrillation episodes in 100 kg calves. A total of 38 animals were used in the study. Postdefibrillation electrocardiograms were recorded. Families of curves of percent successful defibrillation vs pulse duration, percent successful defibrillation vs pulse energy, duration of postdefibrillation complete block or standstill vs energy, and time required for a return to normal sinus rhythm vs energy were derived. The most effective waveform studied (70 amp — 8 msec — 8622 J) yielded defibrillation on the initial attempt in 93% of 120 episodes. In general, the duration of complete block or standstill and the time required for a return to normal sinus rhythm increased with increasing pulse current and pulse energy.

ALTHOUGH THE ENERGY SUPPLIED by most presently available commercial defibrillators is generally adequate for satisfactory transthoracic ventricular defibrillation of small and medium sized patients, there is considerable controversy concerning the energy requirements for a defibrillator intended for large and very large patients. Based upon the results of their laboratory studies involving nonhuman subjects weighing 2.3–340 kg, a review of defibrillation experience at the teaching hospitals of the Baylor College of Medicine and of the Mayo Clinic, and a survey of the literature, Geddes, Tacker, and colleagues have generally argued for defibrillators with higher energy output capabilities.1,2 They have recommended, for example, dosage levels of 6.6 or more J/kg body weight for patients weighing over 46 kg.3 Pantridge and colleagues4 and Crampton and Hunter,5 on the other hand, have stressed the operational advantages of small defibrillators. Pantridge et al. have presented clinical evidence to suggest that even when operated considerably below its maximum energy level (400 J stored, approximately 330 J delivered), and particularly when multiple shocks are used, the Belfast defibrillator can be effective in larger patients. Crampton and Hunter have reported successful low-energy transthoracic defibrillation in a limited number of patients. Each of the three groups was concerned about the possibility of myocardial damage from defibrillatory shocks of excessive energy.

The present paper, based on a total of 3,303 fibrillation-defibrillation episodes in 38 animals, is a report of a systematic experimental study of the effectiveness of unidirectional rectangular-wave pulses in achieving transthoracic defibrillation of 100 kg calves. The study, which serves to complement our earlier rectangular-wave studies in dogs,5,6 may have important implications for the defibrillation of large human patients.

Methods

Equipment

Described in detail elsewhere,8 the ultrahigh-energy hydrogen thyratron/silicon controlled rectifier research defibrillator which was used in the study contains three voltage sources. The first is a 60 Hz supply of adjustable

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