The Pattern of Onset and Spontaneous Cessation of Atrial Fibrillation in Man

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SUMMARY

The atrial electrocardiogram was studied in eight patients who developed intermittent atrial fibrillation following myocardial infarction. Thirty-two episodes of onset of atrial fibrillation and 28 episodes of spontaneous cessation were observed. An atrial premature beat followed by atrial tachycardia of variable duration preceded the onset of atrial fibrillation. Spontaneous cessation of atrial fibrillation was preceded by a change of atrial wave form which suggested slowing of an ectopic focus. The time lapse between the cessation of atrial fibrillation and the onset of sinus rhythm exceeded the subsequent sinus cycle length which suggested overdrive and suppression of the sinoatrial node during the period of atrial fibrillation.

Additional Indexing Words:
Myocardial infarction Atrial electrocardiogram

When atrial fibrillation complicates myocardial infarction, it is frequently intermittent and presents a suitable opportunity for the study of both its modes of onset and cessation, about which little is known in man. That the onset of atrial fibrillation was preceded by atrial premature beats has been suspected clinically for many years. This clinical observation was not confirmed until Killip and Gault, in a predominantly surface electrocardiographic study of patients in whom atrial fibrillation was converted to sinus rhythm by DC precordial shock, showed that relapse into atrial fibrillation was always preceded by an atrial premature beat. Atrial fibrillation has been noted to end abruptly with a following pause which exceeded the sinus cycle length; the precessation wave form, however, has received little attention.

The surface electrocardiogram presents difficulties in the definition of atrial complexes which can be overcome by recording from an intra-atrial electrode. It was considered that the study of intra-atrial electrocardiograms in patients after myocardial infarction would be likely to improve our knowledge concerning impulse formation in atrial fibrillation.

Methods

Eight patients with recent myocardial infarction were studied. All had intermittent atrial fibrillation or frequent atrial ectopic activity. There were six male patients and two females; their ages ranged from 52 to 74 years (mean, 62 years). In four the infarction was predominantly anterior as determined by the 12-lead electrocardiogram, and the remainder were diaphragmatic. Six patients were digitalized intravenously during the study; the other two were already receiving digitalis as part of antifailure therapy.

A unipolar pacing catheter (U.S.C.I. 6F) was introduced into a superficial vein at the antecubital fossa. It was advanced under fluoroscopic control to the right atrium, and the tip was positioned approximately 3 to 5 cm below the junction of that chamber with the superior vena cava. The catheter was often left in situ for 48 hours, and the position of the tip was, therefore, found to vary. The intra-atrial electrocardiogram was recorded via this electrode catheter on magnetic tape together with a simultaneous surface recording from an electrode situated in the V position. The magnetic tape was subsequently analyzed, and episodes of onset and cessation of fibrillation were recorded permanent-
The onset of atrial fibrillation was always preceded by an atrial premature complex and a short episode of atrial tachycardia. Simultaneous recordings from the precordial (V₁) and right intra-atrial electrodes (R.A.L.). The arrow indicates the atrial premature complex.

Results

A total of 32 episodes of onset of atrial fibrillation was studied, and on each occasion the arrhythmia was preceded by a premature atrial beat (figs. 1 to 5). The premature 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 for as little as 1 or 2 sec (fig. 1), but at other times the duration was up to 30 sec. The duration of the prefibrillation tachycardia varied considerably when multiple episodes of atrial fibrillation occurred in the same patient. Finally, the clearly defined atrial wave formation of atrial tachycardia deteriorated into the pattern typical of atrial fibrillation. Throughout the period of atrial fibrillation there was, however, some variation of atrial wave form as recorded through the intra-atrial lead with occasional reappearance of the clearly defined wave form of atrial tachycardia (fig. 6).

Not all atrial premature beats were followed by the onset of atrial fibrillation. Many were conducted normally through the ventricles; others, aberrantly with a conduction pattern of right bundle-branch block (fig. 5). A comparison was made between the coupling times of those atrial premature complexes which produced atrial fibrillation and those that did not (fig. 7). Although there was some overlap between the two groups, atrial premature beats that preceded atrial fibrillation had a significantly shorter coupling time than those that did not. Rapid atrial tachycardia did not always proceed to the development of atrial fibrillation; isolated short periods of tachycardia were seen which reverted spontaneously to sinus rhythm (fig. 8).

A change of atrial wave form preceded the cessation of atrial fibrillation on all 28 occasions observed. The rapid and apparently random wave formation of atrial fibrillation became better defined and resembled that of the atrial tachycardia observed earlier in its natural history. The subsequent appearances

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Figure 2

An atrial premature complex (arrow) preceding atrial tachycardia whose wave form changed seconds later to that of atrial fibrillation.

Figure 3

An atrial premature complex precedes atrial tachycardia whose wave form rapidly fragments to that of atrial fibrillation.
well-defined wave form of atrial tachycardia immediately preceded by an atrial premature beat. The disorganized wave pattern of atrial fibrillation develops 4.6 sec later.

suggested a slowing of ectopic focal discharge, with an eventual cessation of ectopic activity (figs. 9 and 10). A pause followed, after which sinus rhythm was restored (figs. 9 and 10). It was regularly observed that the duration of the interval between the last ectopic atrial beat and the first P wave was longer than the preceding cardiac cycle length (fig. 6). This suggested suppression of the sinoatrial node by the preceding tachycardia.

Discussion

An electrical stimulus applied to the heart during the vulnerable period has been a classic experimental technic for the induction of fibrillation in either atrium or ventricle. The fact that the coupling times of atrial premature beats which provoked atrial fibrillation in this study were significantly shorter than those which did not is evidence both that such a vulnerable period exists in the human atria and that endogenous ectopic discharge occurring during this phase may provoke atrial fibrillation. Atrial premature beats which fell later than the vulnerable period were conducted to the ventricles normally or aberrantly depending on their degree of prematurity. The vulnerable period has been demonstrated to coincide with the early phase of the excitability recovery curve of the dog's atrium, and in the same animal Lown and associates have shown it to coincide with the descending limb of the R wave or the S wave of the electrocardiogram. A similar period was found to exist for the ventricle. Clinically, it is well recognized that ventricular tachycardia or fibrillation may follow a ventricular premature beat which coincides with the T wave of the preceding QRS complex.

(A) Atrial premature beats not precipitating atrial fibrillation but aberrantly conducted through the ventricle showing a right bundle-branch block pattern.

(B) An atrial premature complex preceding atrial tachycardia whose wave form changed subsequently to that of atrial fibrillation.
ONSET AND CESSATION OF FIBRILLATION

Atrial pacing by catheter electrode has occasionally provoked atrial fibrillation, and an estimate of the duration of the vulnerable period of the atrium in man has been made by single and paired pacing. Haft and associates provoked 26 brief periods of atrial fibrillation among three healthy subjects in this way and found that the P-stimulus period or interstimulus period required was between 180 and 280 msec. This value is slightly less than the mean of 300 msec observed here, but errors of measurement in the present study are probably greater than those of Haft's group. In addition the vulnerable period might be expected to vary considerably with local conditions such as tachycardia, atrial distention, and hypoxemia, all of which are to be expected following myocardial infarction.

A period of rapid atrial tachycardia was regularly observed to precede the final development of atrial fibrillation although there was considerable variation in its duration. The method by which the tachycardia was sustained following the premature atrial beat is unknown, but the mechanism of after-potential augmentation and the creation of areas of delayed or irregular repolarization by the premature stimulus are possibilities. The atrial tachycardia was observed by Killip and Gault in only a minority of their patients in whom reversion to atrial fibrillation occurred but this discrepancy between the two studies may be more apparent than real. Figure 4 shows that although the atrial lead demonstrated well-defined tachycardia of approximately 340/min, the surface electrocardiogram suggested atrial fibrillation with an irregular ventricular response. Surface electrocardiography may, therefore, be misleading and not allow a clear distinction to be made between fast tachycardia and the early stages of fibrillation.

The question naturally emerges as to why the tachycardia changed to fibrillation. The

Figure 6
Variation in wave form during the arrhythmia was frequently seen. Well-defined atrial complexes of atrial tachycardia interchange with areas of atrial fibrillation. (A) Atrial fibrillation merging with atrial tachycardia and the reappearance of fibrillation before (B) when atrial tachycardia is again established. (B) Precession of the emergence of well-defined P waves is seen followed by a pause exceeding the sinus cycle length.

Figure 7
Atrial premature complexes preceding atrial fibrillation had a significantly shorter mean coupling time than those that did not.
Not all atrial premature complexes lead to atrial fibrillation. This single continuous record shows atrial premature beats (arrowed) followed immediately by sinus rhythm, and a short episode of atrial tachycardia reverting spontaneously to sinus rhythm.

Figure 8

P waves are clearly seen immediately before cessation of atrial fibrillation. A pause follows and sinus rhythm is established.

Figure 9
of rapid atrial tachycardia. These findings could be explained either by a slowing of the ectopic focus or by periods of more uniform conduction of the depolarization wave throughout the conducting tissue of the atria.

The pattern of spontaneous cessation of atrial fibrillation observed in this study suggested the gradual slowing of stimulus formation by an ectopic focus. Eventually no further premature discharges were seen, and after a pause, normal sinus rhythm was restored. The pause was always in excess of the normal cycle length. The suppression of both normal and abnormal pacemaker sites has been observed before and serves as the therapeutic basis for pacing in the prevention of arrhythmias.

References
5. Lange G: Action of driving stimuli from intrinsic and extrinsic sources on in-situ cardiac pacemaker tissue. Circulation Research 17: 449, 1965
16. James TN: Genesis of Atrial Arrhythmias in the Presence of Myocardial Infarction in Mecha-
nisms and Therapy of Cardiac Arrhythmias. New York, Grune & Stratton, Inc., 1966


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