A Study of Atrioventricular Conduction in Atrial Fibrillation and Flutter in Man Using His Bundle Recordings

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SUMMARY
Seven patients with atrial fibrillation and six patients with atrial flutter were studied using the technic of His bundle recordings. All 13 patients received digitalis. In atrial fibrillation each QRS complex (except for premature ventricular beats) was preceded by a single His bundle deflection. Complete block distal to the His bundle was not observed. Thus, the zone of concealment could be localized to the region proximal to the His bundle. The clinical findings were confirmed in three animal experiments. In atrial flutter the nonconducted P waves were also blocked proximal to the His bundle. In atrial fibrillation aberrant ventricular beats were distinguished from premature ventricular beats by the presence of a preceding His deflection in the former and its absence in the latter.

Additional Indexing Words:
A-V nodal potentials       Concealed conduction       Aberrant ventricular conduction
Premature ventricular beats Zone of concealment     Decremental conduction
Right bundle-branch potentials     Carotid sinus stimulation

Atrial fibrillation is a commonly encountered clinical arrhythmia which is characterized by rapid, irregular atrial impulses (> 350/min) and irregular ventricular responses. In atrial fibrillation the inherent capacity of the A-V conducting system to accept and transmit impulses to the ventricles is exceeded by the rapid, irregular atrial rate. Thus, many atrial impulses are blocked within the conduction system. It has been suggested that these blocked impulses produce concealed conduction, which is considered to be the major cause of the irregular ventricular response in atrial fibrillation.1-8

It has been previously demonstrated in animal studies that conduction delay and complete block of impulse transmission may occur in either the A-V nodal region or His-Purkinje system.7-9 More recently, these findings have been confirmed in man using an electrode catheter technic for recording electrical potentials of the specialized conducting fibers.10, 11

The purpose of this study was to determine more precisely the zone of concealment of conduction in clinical cases of atrial fibrillation by using the technic of His bundle recordings.12 The level of A-V block was similarly determined in clinical cases of atrial flutter.

Methods

A right heart catheterization was performed on 13 subjects in the post-absorptive, nonsedated state. Seven subjects had atrial fibrillation, and six had atrial flutter. All of the patients were taking digitalis. Using local anesthesia, a tripolar electrode catheter (no. 6-7F) was percutaneously introduced into the right femoral vein and fluoroscopically positioned across the tricuspid valve.
The proximal terminals of the electrode catheter were plugged into a distribution switch box* from which simultaneous bipolar leads could be selected (electrodes 1 and 2, 2 and 3, and 1 and 3). Each channel of the distribution switch box was led into an AC input of an ECG preamplifier. The filter frequencies of the ECG preamplifier were set at 40 to 500 cps. A standard electrocardiographic lead was simultaneously recorded. The electrode catheter was slowly withdrawn across the tricuspid valve until a rapid biphasic or triphasic spike appeared between the atrial and ventricular electrograms and within the P-R segment of the standard ECG lead. Recordings were made on a multichannel oscilloscopic photographic recorder* at paper speeds of 100 to 200 mm/sec. Measurements in milliseconds were made of the intervals between the P wave and the His deflection (P-H interval) and from the H deflection to the Q wave of the QRS complex (H-Q interval).

The validity of our findings in man was tested in three animal experiments. Mongrel dogs were anesthetized with pentobarbital-Na and artificially respirated. Right thoracotomy was performed, and the right atrium and basal portions of the right ventricle were exposed through a wide pericardial opening. Electrograms of the bundle of His were obtained by inserting two fine, Teflon-coated stainless steel wires into the region of the bundle of His. By using an AEL† stimulator, atrial fibrillation was electrically induced by applying stimuli to the right atrium at a frequency of 20/sec. His bundle recordings were repeatedly obtained in the control state, during the induced atrial fibrillation, and after spontaneous recovery to normal sinus rhythm.

Results

Atrial Fibrillation

His bundle recordings were obtained in six of the seven cases of atrial fibrillation. In all six cases a single His bundle deflection preceded each QRS complex (except for ventricular premature beats). The H-Q intervals were constant from beat to beat during atrial fibrillation. Representative findings are presented in figures 1 to 3. Figure 1 illustrates the recordings of His bundle activity in a patient during atrial fibrillation and following DC cardioversion to normal sinus rhythm. Panel A of figure 1 is a recording during atrial fibrillation, and panel B depicts recordings obtained after conversion to normal sinus rhythm. In both rhythm strips a single His deflection precedes each QRS complex. The H-Q intervals measure 63 msec during atrial fibrillation and also during normal sinus rhythm. Block below the His bundle was not observed in any of the cases. The position of the sensing electrodes was such that they clearly record His bundle activity but not the coarse atrial fibrillatory waves.

Figure 2 illustrates a representative tracing from the animal experiments. His bundle recordings were obtained at normal sinus rhythm (panel A) and during electrically induced atrial fibrillation (panel B). Note the

*Electronics for Medicine, White Plains, N. Y.
†American Electronics Laboratory, Colmar, Pennsylvania.

Figure 1

Panel A depicts recordings of His bundle activity during atrial fibrillation. Panel B depicts recordings taken in the same patient immediately after conversion to normal sinus rhythm by DC shock. The H-Q intervals in both rhythm strips are 63 msec.
A-V CONDUCTION IN ATRIAL FIBRILLATION

Figure 2
Tracings from an animal in which recordings of His activity were obtained from wires inserted directly into the His region. Panel A was recorded during normal sinus rhythm. The H-Q interval measures 30 msec. The H-Q interval remains 30 msec during electrically induced atrial fibrillation. The fourth and sixth complexes are aberrantly conducted beats which are preceded by a His deflection (panel B).

Figure 3
In this clinical case of atrial fibrillation the first, fourth, and sixth complexes represent the normally conducted beats, each of which is preceded by a His deflection. The second complex, which is also preceded by a His deflection, represents an aberrant beat. The third complex is a premature ventricular contraction. No His potential precedes this beat.

absence of P waves during atrial fibrillation and the constancy of the H-Q intervals in both tracings. In panel B the fourth and sixth beats represent aberrant beats which are preceded by a His deflection.

Several examples of aberrant ventricular conduction were also recorded in our clinical cases of atrial fibrillation. Aberrantly conducted beats were easily distinguished from premature ventricular beats by the His bundle deflection which preceded the former. In figure 3, the first, fourth, and sixth beats represent the normal complex during atrial fibrillation. Each normal complex is preceded by a His deflection. The third beat (wide QRS complex) represents a premature ventricular contraction which is not preceded by a His deflection. The second complex (also of wide

Circulation, Volume XL, July 1969
duration) represents an aberrantly conducted beat of supraventricular origin, confirmed by the recording of a preceding His deflection.

Atrial Flutter

The findings in all six cases of atrial flutter were similar. Each QRS complex was preceded by a single His deflection, and the non-conducted P waves were blocked proximal to the His bundle. Figure 4 illustrates these findings. The atrial flutter rate is 330/min, and the ventricular response is 165/min. The electrogram recording (HBE) reveals that each QRS complex is preceded by a single His deflection. In panel B of figure 4, right bundle-branch potentials were also recorded. The effects of carotid sinus stimulation in this case are shown in figure 5. Carotid sinus stimulation increased the degree of A-V block, and the atrial flutter waves can be more easily visualized. The absence of His deflections in the nonconducted beats indicates that the level of block was proximal to the common bundle.

In figure 6 His bundle recordings were obtained in atrial flutter (panel A) and after cardioversion to normal sinus rhythm (panel B). The H-Q intervals (60 msec) are the same in both rhythm strips.

Figure 4

A case of atrial flutter. In panel A the atrial rate is 330/min, and the ventricular response is 165/min. The nonconducted P waves are blocked proximal to the His bundle. The H-Q interval measures 48 msec. In panel B a right bundle-branch potential was also recorded. The RB-Q interval measures 19 msec.
A-V CONDUCTION IN ATRIAL FIBRILLATION

CAROTID SINUS STIMULATION

![ECG and HBE tracings showing atrial flutter and sinus rhythm]

Figure 5
Demonstration of the effects of carotid sinus stimulation during atrial flutter. All of the non-conducted P waves are blocked proximal to the His bundle.

A
P-P 230msec
R-R 460
H-Q 60

ATRIAL FLUTTER

ECG

HBE

Figure 6
Recordings obtained in the same patient as seen in figure 5 during atrial flutter (panel A) and immediately following conversion to normal sinus rhythm (panel B). The H-Q intervals are constant at 60 msec in both rhythm strips.

Discussion
Lewis and Master first demonstrated that premature beats which are delayed or blocked within the A-V conduction system may affect the conduction of succeeding beats. The term “concealed conduction” was introduced into clinical electrocardiography by Langendorf to define concisely the aftereffects of an impulse that incompletely penetrates the A-V conducting system. Langendorf and associates and Carleton and Graettinger have experimentally induced concealed conduction in the human heart during right atrial pacing.

More recently, these observations in man have been extended using His bundle recordings. Figure 7 illustrates an example which satisfies the definition of concealed A-V conduction. The top panel demonstrates a 1:1
Concealed A-V conduction. In the top panel the right atrium is being paced at a rate of 100/min. The P-H interval measures 104 msec. In the bottom panel, 2:1 block occurs at a paced atrial rate of 200/min. The nonconducted P waves are blocked proximal to the His region and the conducted P waves have a P-H interval which measures 197 msec. The nodal potential is labeled N. This tracing also demonstrates that for the conducted atrial beats the greater delay in A-V transmission occurs within the N-H interval.

atrioventricular response at a paced atrial rate of 100/min. The P-H and H-Q intervals measure 104 and 47 msec, respectively. In the bottom panel 2:1 block occurs at a paced atrial rate of 200/min. The ventricular response (100/min) is the same as before. The nonconducted atrial beats are blocked proximal to the His bundle, and the conducted beats are associated with a prolonged P-H interval (197 msec). The recording of the A-V nodal potential (N) permits further localization of the A-V delay for the conducted atrial beats to the N-H interval. Likewise, the area wherein complete block occurs for the non-conducted atrial beats can also be localized to the N-H interval. Delay within the N-H interval has previously been demonstrated.10,11

Concealed conduction has also been invoked as the major factor determining the irregular R-R intervals in atrial fibrillation.2,3 It is known that during atrial fibrillation the various ventricular cycle lengths are not uniformly distributed. Studies of the frequency distribution curves of ventricular cycle lengths in spontaneously occurring and experimentally induced atrial fibrillation have demonstrated multiple peaks, indicating the more frequent occurrence of certain cycle lengths.2 Moe and
Abildskov\(^4\) characterized some of the features of the frequency distribution of ventricular cycle lengths in atrial fibrillation and postulated that concealed conduction may well explain this distribution.

Based on a study of clinical electrocardiograms Langendorf and associates,\(^4\) proposed that evidence of concealed A-V and V-A conduction during atrial fibrillation is suggested by: “(a) Occurrence of a ‘compensatory pause’ following a ventricular premature systole. (b) Failure of an AV nodal escape to appear at the expected time due to concealed discharge of a subsidiary AV nodal pacemaker by a penetrating atrial impulse. (c) Acceleration of the ventricular rate when atrial fibrillation changes to atrial flutter (elimination of concealed conduction with slowing of the atria). (d) A tendency for two or more long cycles to occur in succession (each containing one or more concealed responses).”

In studies on experimental animals the atrio-nodal junction, A-V node, nodal-His region, and His-Purkinje system have all been demonstrated to be regions where delay or block of impulse transmission may occur.\(^7\>-\(^9\)

During premature atrial stimulation in man, A-V conduction delay and complete block have been demonstrated to occur in the A-V node, nodal-His region, and the His-Purkinje system.\(^11\) An example of complete block within the His-Purkinje system during premature atrial stimulation is illustrated in figure 8. The results of the present study indicate that the zone of concealment in atrial fibrillation is located proximal to the His bundle. This conclusion is based on the fact that we have never recorded or monitored a His bundle potential that was not followed by a QRS complex, as illustrated in figure 8. Our recordings involve well over 3,000 QRS complexes obtained in seven patients with atrial fibrillation.

Our findings in man are consistent with those of Moore\(^5\) who studied the mechanism of ventricular dysrhythmia associated with atrial fibrillation in the isolated rabbit heart using microelectrode technics. He demonstrated that the variation in ventricular cycle lengths resulted from concealed conduction within the A-V node. At times, as many as seven to nine consecutively concealed responses were recorded from the A-V node.

That all of our patients were taking digitalis at the time of study may explain in part why complete block distal to the His bundle was not observed. Digitalis, which causes A-V conduction delay and block, is known to act at the nodal-His region. It would be expected, therefore, that in atrial fibrillation digitalis would cause fewer impulses to be presented to the His-Purkinje system. Furthermore, digitalis would cause sufficient delay of the transmitted impulses to allow for more complete repolarization of the His-Purkinje fibers.

In all six cases of atrial flutter, the level of block for the nonconducted P waves was also found to be proximal to the His bundle. No examples of complete block or decrement within the His-Purkinje system were recorded. Two patients exhibited atrial flutter and atrial fibrillation while they were being studied. In both cases the ventricular rate during atrial flutter was higher than during atrial fibrillation.
The clinical importance of recognizing aberrant ventricular conduction in atrial fibrillation has been stressed previously.\textsuperscript{16–20} Aberrant conduction may be mistaken for premature ventricular beats or ventricular tachycardia when occurring in successive beats. Attempts have been made to formulate workable rules for the clinical recognition of aberrant conduction by electrocardiographic patterning.\textsuperscript{19, 20} In the absence of recognizable P waves, as in atrial fibrillation, the diagnosis of aberrant ventricular conduction remains difficult. The technic of recording His bundle activity appears to be the most valid method for diagnosing aberrant conduction in atrial fibrillation. The procedure is relatively simple to perform and, in many cases, may help to resolve the therapeutic dilemma facing the clinician.

Acknowledgment

The authors wish to thank Anne Mazzella, Theresa Halloran, Audrey Pedersen, Joan Cumming, and Michael Moretti for their assistance.

References

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_Circulation_. 1969;40:71-78
doi: 10.1161/01.CIR.40.1.71

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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