Induction of Iatrogenic Electrocardiographic Patterns During Electrophysiologic Studies

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SUMMARY Indwelling cardiac catheters by producing local mechanical stimulation or trauma can induce electrocardiographic (ECG) patterns which simulate known electrophysiologic phenomena. Catheter-induced ECG patterns were analyzed in 447 consecutive patients undergoing electrophysiologic studies. Iatrogenic nature of these patterns was suggested by 1) absence prior to placement of catheter; 2) sudden appearance with catheter placement and disappearance with catheter repositioning; 3) reoccurrence with re manipulation of catheters; and 4) simulation (in some cases) by programmed electrical stimulation from the catheter. Common catheter-induced patterns were 1) right bundle branch block (RBBB) lasting less than 24 hours occurred in 19 patients; 2) transient third degree atrioventricular block in His-Parkinson system developed in 3/13 patients with pre-existing left BBB; 3) catheter-induced ventricular pre-excitation which simulated ECG patterns of type B Wolff-Parkinson-White syndrome; 4) fortuitous synchronization of right ventricular excitation from the catheter, and left ventricular excitation from sinus beat resulted in normalization of the QRS complexes in 5/68 patients with pre-existing RBBB; 5) premature beats from the atri, right ventricle, and His bundle, which were common, resulted in complex ECG patterns. These iatrogenic ECG patterns must be identified in order to avoid errors in interpretation.

THE TECHNIQUE OF INTRACARDIAC ELECTROCARDIOGRAPHY is increasingly used for the study of human cardiac electrophysiology.1-4 It has become customary to use several electrode catheters in order to study the cardiac conduction system more elaborately. It has also become apparent that intracardiac catheters can induce local mechanical stimulation producing electrocardiographic (ECG) patterns which closely simulate known electrophysiologic phenomena.6-14

The purpose of this report is to create general awareness concerning the catheter-induced ECG patterns associated with introduction, manipulation, and/or placement of cardiac catheters. The common ECG patterns produced by electrode catheters in 447 consecutive patients undergoing electrophysiologic studies will be discussed.

Materials and Methods

Patients were studied in a post-absorptive nonsedated state for a variety of reasons. The nature of the procedure was explained and a signed consent obtained. Using percutaneous technique electrode catheters were positioned under fluoroscopic guidance in the region of the tricuspid valve and right atrium in all patients.1 When feasible, additional electrode catheters were positioned in the right ventricle and coronary sinus. A 12-lead electrocardiogram was recorded immediately prior to the introduction of catheters. Subsequently, standard ECG leads I, II, III, and V6 were displayed on a multichannel oscilloscope and monitored during the introduction of the catheters. After catheter placement, the ECG leads, intracardiac electrograms (filter frequency setting 40-500 Hz) and time lines were displayed simultaneously and recorded on magnetic tape and the records were later replayed. The various methods of electrophysiologic studies as performed in this laboratory as well as the definitions of antegrade and retrograde conduction time and refractory periods have been previously published.1, 5

When unexpected electrophysiologic events occurred during the studies their nature and origin were further explored. The iatrogenic nature of these events was suggested by 1) absence on previous ECG recordings; 2) absence during 15-45 min monitoring before placement of catheters; 3) appearance soon after positioning of the offending catheter; 4) prompt disappearance on either complete withdrawal or repositioning (sometimes with minimal manipulation) of the suspected catheter; 5) reproducibility by mechanical and/or electrical stimulation from the offending catheter in some cases.

Results

The data presented here were obtained from 447 patients studied in this laboratory over a three year period (January 1973 to December 1975). For the purposes of this communication only the more common iatrogenic ECG patterns will be presented. These are summarized in table 1.

Right Bundle Branch Block (RBBB)

This form of traumatic RBBB was generally produced with the electrode catheter in the atrioventricular (A-V) junction, used for recording the electrical activity of the bundle of His. All degrees of RBBB varying from minimal terminal conduction delay to so-called complete RBBB pattern were observed. The duration over which RBBB persisted was also variable, lasting from a few beats to the entire length of the study (up to 4 hours). When the RBBB was transient the degree of conduction delay was generally constant at all cycle lengths available. However, during the course of study when RBBB conduction improved, more complete forms of RBBB occurred with changes in heart rate (fig. 1). Although more complete forms of RBBB could be induced at critically long and/or short cycle lengths,18 the latter could not be exclusively ascribed to catheter-induced trauma since BBB during atrial premature stimulation is commonly observed during electrophysiologic studies.
Following normalization of RBB conduction the block could be reproduced at will (in 10/13 patients in whom it was attempted) by manipulation of the offending catheter. When available for comparison the His-Purkinje conduction times (H-V intervals) during RBBB and normal intraventricular conduction measured the same (fig. 1) in any given case.

Ativoventricular (A-V) Block

Temporary complete interruption in A-V conduction (third degree A-V block) occurred in 3/13 patients with pre-existing fixed complete LBBB pattern. In all instances, the offending catheter was in the A-V junctional region. In 2/3 patients the His bundle electrogram was being recorded at the time A-V block occurred, and it could be documented that the block was distal to the His bundle recording site. In the remaining patient the block occurred before the recordings could be obtained. The A-V block was transient in all cases and none of these patients demonstrated further episodes of A-V block following withdrawal of the catheters during two days monitoring after the study. To date (a

**Table 1. Catheter-Induced Electrocardiographic Patterns**

<table>
<thead>
<tr>
<th>No.</th>
<th>Type of Pattern</th>
<th>Frequency</th>
<th>Duration</th>
<th>Treatment</th>
<th>Helpful measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RBBB</td>
<td>19/379 pts (&lt; 5%) without pre-existing RBBB</td>
<td>Variable but &lt; 24 hours</td>
<td>Not necessary</td>
<td>Gentle &amp; careful manipulation of catheters in A-V junction</td>
</tr>
<tr>
<td>2</td>
<td>High degree A-V block</td>
<td>3/13 pts (23%) with pre-existing LBBB</td>
<td>Transient episodes lasting &lt; 30 sec</td>
<td>Temporary demand ventricular pacing</td>
<td>Gentle &amp; careful manipulation of catheters in A-V junction</td>
</tr>
<tr>
<td>3</td>
<td>Ventricular pre-excitation (type B)</td>
<td>14/447 pts (3%)</td>
<td>Transient and intermittent</td>
<td>Not necessary</td>
<td>Catheter repositioning</td>
</tr>
<tr>
<td>4</td>
<td>Normalization of QRS complex</td>
<td>5/68 pts (7.5%) with pre-existing RBBB</td>
<td>Transient and intermittent</td>
<td>Not necessary</td>
<td>Catheter repositioning</td>
</tr>
<tr>
<td>5</td>
<td>Premature beats</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. ventricular</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. atrial</td>
<td>Common</td>
<td>Transient and intermittent</td>
<td>Catheter withdrawal although usually not necessary</td>
<td>Catheter repositioning</td>
<td></td>
</tr>
<tr>
<td>c. junctional</td>
<td>18/447 pts (4%)</td>
<td>Transient and intermittent</td>
<td>Catheter withdrawal although usually not necessary</td>
<td>Catheter repositioning</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: LBBB = left bundle branch block; RBBB = right bundle branch block; pts = patients.

**Figure 1. Catheter-induced right bundle branch block (RBBB).** Tracings from top to bottom in each panel are ECG leads I, II, V₁, high right atrial (HRA), His bundle electrogram (HBE) and time lines. Panel A shows A-V nodal Wenckebach phenomenon during sinus rhythm with normal intraventricular conduction. The A, H, and V represent bipolar atrial, His bundle, and ventricular deflections respectively. Progressive prolongation in A-H intervals (A-V nodal conduction time) is followed by a block of impulse proximal to the H. The H-V interval (His-Purkinje conduction time) during all conducted beats measures 45 msec (not labelled). Tracing shown in panel B was taken after the RBBB was produced by the catheter. Note that the conduction abnormality is constant at all cycle lengths (R-R or H-H) depicted and the H-V intervals measured the same as in panel A. During the course of study RBB conduction improves (panel C) and more complete degree of RBBB is observed following longer R-R intervals (3rd and 5th QRS complexes).
follow-up of one to three years) all three patients have demonstrated intact A-V conduction and have not needed pacemaker therapy.

Ventricular Pre-excitation

Local mechanical excitation by catheters in the right ventricle (in any position) in close temporal relation to ventricular activation from sinus impulse produced the typical ECG pattern of Wolff-Parkinson-White syndrome, i.e., a short P-R interval and a delta wave. Generally, catheter-induced ventricular pre-excitation occurred as single isolated beats; but in some patients the phenomenon occurred over several consecutive beats. Depending upon the position (i.e., apex, outflow, or inflow) and the point of excitation along the course of the catheter, the initial forces of ventricular depolarization (i.e., the iatrogenic delta) were directed superiorly or inferiorly.

In most instances right ventricular catheters produced posteriorly directed delta waves in V1, thereby simulating the ECG pattern of type B Wolff-Parkinson-White syndrome. Repositioning and/or complete withdrawal of the offending catheter always resulted in complete abolition of ventricular pre-excitation. Although both right and left ventricular (from a catheter in coronary sinus) pre-excitation could be induced by programmed electrical stimulation, left ventricular pre-excitation due to mechanical stimulation from the catheter in the coronary sinus was not observed during the present study.

Normalization of the QRS Complex

Intermittent normalization of the QRS in patients with pre-existing RBBB was another commonly observed phenomenon. In 5/68 patients with pre-existing RBBB catheter-induced right ventricular excitation coincided temporally with the arrival of sinus impulse at the left ventricle in such a manner that a perfect normalization of the QRS complex resulted (fig. 2). In 2/5 patients normalization of the QRS complex was associated with shortening of the P-R and H-V intervals which further documented the fusion nature of these QRS complexes, where right ventricular activation preceded activation of the left ventricle. In both cases a local bipolar ventricular electrogram from the offending catheter was being recorded which documented altered sequence of ventricular activation during "normalized" beats compared to those displaying a RBBB configuration (fig. 3).

Premature Beats

Ventricular Premature Beats

Single or multiple ventricular beats from local mechanical stimulation of the right ventricle were almost universal and not unexpected. At times isolated catheter induced ventricular beats still occurred after apparent stabilization of catheter position and were recognized by their morphology and bizarre and unexpected physiologic behavior. On occasion, catheter induced premature ventricular beats closely
mimicked other electrophysiological phenomena (fig. 4). A careful search of the entire record, however, usually revealed the true nature of the electrophysiologic pattern (fig. 5). Catheter-induced premature right ventricular beats showed the expected LBBB pattern. However, the axis orientation of mechanically-induced ventricular premature beats was often different from that of paced beats from the same catheter suggesting that the tip of the catheter (i.e., near the pacing electrodes) was not necessarily the point of mechanical stimulation (fig. 5, panels A and C). The incidence of catheter-induced ventricular beats increased when two catheters were positioned in the right ventricle. In some patients, withdrawal of a suspected catheter to an atrial position did not abolish the ventricular beats if the catheter continued to touch any point along the course of a second ventricular catheter. Further withdrawal of the first catheter out of contact with the second was necessary to abolish the premature ventricular beats. These observations suggest that tangentiality of catheters will increase the incidence of premature ventricular beats beyond that expected from the number of catheters alone.

**Atrial Premature Beats**

The incidence of mechanically-induced premature atrial beats during placement and manipulation of cardiac

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**Figure 4.** Catheter-induced ventricular premature beats simulating aberrancy. In panels A and B atrial premature beats (Ap) are coupled to preceding sinus beats (As). The Ap in both panels either block below the H or appear to conduct to the ventricles with varying H-V intervals. The QRS complexes following Ap in panels A and B show a typical LBBB pattern. The ventricular activation in panel B following longer H-V intervals (compared to panel A) is followed by retrograde atrial activation (Ar). The retrograde atrial activation in panel B is compatible with echo phenomenon from intraventricular conduction delay (H-V intervals) since the A-H intervals of all As in both panels measure 85 msec (not labelled). Figure 5 however, reveals the true origin of these QRS complexes with LBBB pattern. S denotes stimulus artifact.

**Figure 5.** Catheter-induced premature ventricular beats. Panel A shows random occurrence of a ventricular premature beat with QRS morphology identical to those seen in figure 4. These beats were, in fact, produced by the right ventricular catheter and occurred fortuitously during atrial premature stimulation. The Ar represents retrograde atrial activation from ventricular premature beats. Panel B demonstrates true aberration of the LBBB type observed in this patient during an episode of A-V nodal re-entrant supraventricular tachycardia. Panel C illustrates right ventricular apical stimulation from the catheter thought to be responsible for production of premature ventricular beats shown in panel A and figure 4. Compare the QRS morphologies and axis orientation of beats shown in the three panels.
catheters was high. The premature beats arose from different parts of the atria and all configurations of P waves and atrial activation sequences were noted. In patients with pre-existing atrial premature beats and/or those suspected of intermittent arrhythmias associated with atrial premature beats, i.e., re-entrant supraventricular tachycardias, atrial fibrillation, etc., the existence of catheter-induced beats created a problem. In these patients it was impossible at times to make a clear cut distinction between the spontaneous atrial premature beats and those produced by the catheters. In most instances, however, the guidelines used above (Materials and Methods) helped determine the true origin.

Premature Beats Arising from the Bundle of His

Less common than atrial or ventricular beats were the extra beats arising from the bundle of His (seen in < 4% of all patients). These were produced exclusively by catheters positioned across the tricuspid valve in close proximity to the bundle of His. Their site of origin was suggested by H-V intervals and QRS complex morphologies which were similar to those of sinus beats. Generally, these beats were randomly distributed as single isolated beats with no definite relation to any cardiac events electrical or mechanical. At times confusion was created by fortuitous occurrence of these beats in association with other electrophysiologic events and their true nature became apparent only after locating their random occurrence at other times during the study.

Discussion

The catheter-induced ECG patterns during cardiac catheterization (for whatever purpose) are frequent enough to warrant calling attention to their occurrence. Since these catheter-induced patterns can closely simulate spontaneous ECG abnormalities, the recognition of their iatrogenic nature is of utmost importance if misinterpretations are to be avoided.

The sudden appearance of persistent RBBB during introduction or manipulation of any cardiac catheter in the vicinity of the right side of the interventricular septum is not difficult to recognize, and has been previously noted. Transient forms of traumatic RBBB may be missed if the monitored ECG tracings are not constantly watched. The most obvious serious problem associated with the traumatic RBBB is the ever present possibility of inducing a high degree A-V block in patients with pre-existing LBBB. The sudden occurrence of A-V block in patients with pre-existing LBBB during cardiac catheterization has been reported by several authors and the present data confirms the previous observations. It is recommended therefore, that in patients with pre-existing LBBB, facilities should be available for ventricular pacing in the event a high degree A-V block is induced. This can be accomplished by prior careful positioning of a catheter in the right ventricle introduced via an arm vein since traumatic RBBB is less commonly produced by these catheters unless excessively manipulated. Also, during positioning, the catheter should be connected to a pacing device in a demand mode.

The superficial anatomic location of the RBB makes it prone to catheter-induced trauma and therefore the occurrence of traumatic RBBB during cardiac catheterization is not surprising. The reason for higher incidence of traumatic RBBB in patients with pre-existing LBBB compared to the remainder of the group (i.e., 23% vs 5%) is not clear and the occurrence may be fortuitous. However, longer H-V intervals in patients with LBBB (60-80 msec vs normal range of 30-55 msec) in this series may be indicative of pre-existing abnormal conduction in the RBB making it more susceptible to complete interruption of conduction with lesser degrees of trauma.

Whether the complete forms of RBBB patterns seen represented slow conduction or complete block could not be determined. The induction of third degree A-V block in patients with pre-existing LBBB strongly suggests that both the traumatic RBBB and the pre-existing LBBB in these patients represented complete interruption of conduction in the respective fascicles.

The finding of rate related forms of BBB following trauma was not unexpected and similar observations have been made previously during animal studies. It goes without saying that once a traumatic RBBB is diagnosed caution needs to be exercised regarding interpretation of data pertaining to conduction and refractoriness of the His-Purkinje system.

Catheter-induced A-V block within the bundle of His or A-V node was not encountered during electrophysiologic studies. The A-V node is protected from catheter-induced trauma, probably by virtue of its posterior location in the A-V junction. Despite the proximity of the catheter to the bundle of His, catheter-induced trauma affecting the entire thickness of this structure may be difficult to produce, a fact that can explain the rarity of intra-His block during cardiac catheterization.

Based upon present observations it is tempting to postulate that on rare occasions traumatic LBBB and A-V block (in patients with pre-existing RBBB) may occur with catheters excessively manipulated in the vicinity of LBBB for any reason. On theoretical grounds it would appear that patients with preexisting RBBB and left anterior or posterior hemiblock will be more vulnerable to the above complication.

The realization that iatrogenically-induced ventricular pre-excitation can mimic the ECG pattern of Wolff-Parkinson-White syndrome is extremely important. The distinction between catheter-induced and true ventricular pre-excitation is further complicated by the fact that true forms of Wolff-Parkinson-White syndrome in some cases may be intermittent and previously unsuspected. The appearance of ventricular pre-excitation during placement and manipulation of catheters and complete disappearance of the phenomenon with either repositioning and/or withdrawal of the offending catheter strongly favors the iatrogenic nature of the event and such observations have been made previously. It is difficult to explain the rhythmic occurrence of this phenomenon over several consecutive beats with catheters in apparently stable positions. The most likely explanation is that phasic changes in venous return and/or atrial contraction produce catheter movement sufficient to excite the ventricle. It is suggested therefore that whenever an unsuspected ventricular pre-excitation is noted with ventricular catheters, its true nature should be fully investigated and the iatrogenic forms excluded. Even though not seen
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during the present study, iatrogenically-induced left ventricular pre-excitation from catheters in the coronary sinus or left ventricle remains a possibility.

The mechanism of normalization of the QRS complexes in patients with pre-existing RBBB in essence is quite similar to that of ventricular pre-excitation. If the right ventricular excitation from a catheter perfectly coincides with left ventricular activation from the supraventricular impulse, a more normal QRS complex will result. The paradox of “normal” QRS complexes in patients with BBB produced by ectopic ventricular activity is well known and the present findings have the same basis.22, 23

The occurrence of single or multiple premature beats during intracardiac manipulation of catheters is understandable. On the other hand it is hard to pinpoint how premature beats occur when the catheter appears to be stable. Various factors, i.e., cardiac movement associated with respiration, changes in venous return, atrial contraction and minor movement of the patient singly or in combination, may be responsible. When the premature beats are occasional their fortuitous occurrence in relation to other electrophysiological phenomena can be a source of confusion.18 A careful review of the entire record however will generally reveal their true origin.

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