Function of Potential Bypass Tracts for Atrioventricular Conduction

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SUMMARY

Function of potential bypass tracts for the atrioventricular conduction was examined in isolated rabbit hearts using microelectrodes, a suction electrode, and bipolar electrocardiographic leads. After the normal A-V conduction was blocked by acetylcholine, conduction to the ventricle occurred with a much shorter A-V conduction time and without significant QRS change, when a point at the root of the inferior vena cava was stimulated. By analyzing the site of delay, this route was presumed to go from the right atrium to the lower portion of the His bundle bypassing the A-V node. When a point inferior to this point was stimulated, marked shortening of conduction time and marked QRS change occurred. This second tract seemed to exist between the right atrium and ventricle bypassing the specialized system. The third tract was found when an anterior point on the right atrium was stimulated. The conduction was markedly prolonged without changing the QRS. This route seemed to enter some point of the A-V node, inducing a detour within the node.

Additional Indexing Words:
Wolff-Parkinson-White syndrome
Suction electrode
Microelectrode
Pre-excitation
Mahaim’s connections
Bundle of Kent
A-V node
Bundle of His
Acetylcholine

FROM THE RESULTS of anatomic studies several investigators have inferred the presence of some bypass tracts of the atrioventricular (A-V) conduction or anatomic connections between the atrium and the ventricle other than the His-Tawara A-V conduction system.\(^1\)\textsuperscript{-15} In the clinical electrocardiogram there is also some evidence suggestive of the presence of bypass tracts such as Wolff-Parkinson-White (WPW) syndrome.\(^16\)\textsuperscript{-19} Although most of the reciprocal beats or rhythm can probably be explained by the histologic netlike structure of the A-V node, the possibility cannot be ruled out that at least in some cases they are caused by some bypass tracts. As for the functional studies, Kent first carried out such an experiment himself on the bundle of Kent,\(^5\)\textsuperscript{,}7 but there are few functional studies on the other bypass tracts.

Since our study\(^20\) on the A-V node in 1964 we believed that all evidence suggesting more than one pathway between the atrium and the ventricle could be explained by the netlike structure of the A-V node. Recently, however, we observed several phenomena which suggested the presence of extranodal bypass tracts in addition. This study was carried out, not to demonstrate functional potency of bypass tracts suggested recently by some anatomists but rather to enlarge and supplement our previous functional studies in this context.

Methods

Rabbits were heparinized and killed instantly by a blow to the back of the head and the heart was quickly removed. The heart was perfused with aerated Tyrode solution via the coronary system through an L-shaped glass cannula inserted into the ascending aorta and mounted in the center of a plexiglass chamber containing about 70 ml of Tyrode solution.
equilibrated with 95% O₂ and 5% CO₂. Three electrodes were mounted in the left and right upper corner and the right lower corner of the chamber corresponding to the right and left arm and left leg in the human, respectively, and either lead II or III was recorded. A suction electrode was placed in the middle of the anterior free wall of the right ventricle.

To determine the mode of conduction, a transverse incision was made below the A-V ring on the free wall of the right ventricle, and the atrial and ventricular septa were exposed. One microelectrode was inserted in the A-V node and another in the bundle of His. One pair of electrodes of an adjacent bipolar lead was placed near the fossa ovalis inside the right atrium and another in the center of the free wall of the right ventricle.

Our method of producing and employing microelectrodes was reported elsewhere.²¹

Results

In the control state on perfusion with Tyrode solution, stimulation of various epicardial points of the right atrium, as shown in figure 1, induced conduction to the ventricle, judging from the action potential obtained by the suction electrode on the center of the free wall of the right ventricle and from the ventricular complex of the lead II electrocardiogram (fig. 21a).

In three of 12 experiments the following evidence for one type of bypass tract of the A-V conduction was found: When acetylcholine was added to the Tyrode solution in the concentration of 1 to 10 μg/ml, complete or incomplete A-V block eventually occurred when the usual points of the outer wall of the right atrium were stimulated (fig. 21bA, B, and C). Stimulation of all the epicardial points of the right atrium was then tried to find points still inducing conduction to the ventricle. Such a point was found anterior to the root of the inferior vena cava (point 5, fig. 1). The conduction to the ventricle induced by its stimulation is shown in figure 21bD. It was presumed that a bypass tract was still available for the A-V conduction after the specialized A-V conduction system was blocked. In this conduction the interval between the stimulus artifact and the ventricular action potential was 58.4 msec, much shorter than that in the control state (fig. 21bE). No significant alteration of the QRS configuration was observed. A slight change of its rising limb could not be ruled out because of the stimulus artifact. Later acetylcholine was washed out. In continuing the stimulation of point 5 with new Tyrode solution, the interval between the stimulus artifact and the ventricular action potential suddenly prolonged to the control value within several minutes (fig. 21IC). When electrical stimulation was stopped, the normal conduction was found to occur by the sinus node impulses (fig. 21ID).

In two experiments, while searching for the mode of conduction, an abnormal sequence of conduction was noticed in spontaneous sinus rhythm. As is shown in figure 3a, the ventricle

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Figure 1

The sites of stimulation of the right atrium of the isolated rabbit heart. RA = right auricular appendage; RV = right ventricle; SVC = superior vena cava; IVC = inferior vena cava.
Normal responses to the Tyrode solution (panel Ia) and A-V conduction through the first bypass tract (panel Ib). The upper tracing of each figure shows lead II electrocardiogram and the lower tracing action potentials obtained by the suction electrode.

By giving acetylcholine to the concentration of $10^{-6}$ g/ml, a complete or incomplete A-V block appeared on stimulation of most atrial points, as is seen in figure 1 in responses on stimulation of point 1 (A), point 2 (B), and 3 (C). Only when point 5 was stimulated, were there still enough responses but A-V conduction time was shorter (D and E). In E, the response is shown with a faster sweep when point 5 was stimulated. The QRS complex does not show conspicuous changes. In panel II, the recovery after washing out acetylcholine is shown. Tracing IIA is the same as IbD but with a slower sweep, that is responses to stimulation of point 5 under the influence of acetylcholine. IIB shows responses immediately after washing out acetylcholine and in the new Tyrode solution. After several minutes the interval between the stimulus artifact and the action potential prolonged suddenly, as in IIC. At this time electrical stimulation was stopped, and the A-V conduction of the sinus node impulses was found to be restored, as in IID.
The reappeared. The point immediately showed impulse normally after the response. The stimulation was restored, of sequence distal point showed. His, of sequence below this point, block occurred, as is shown by this response. The bypass tract was supposed to pass through point 5, the incision, and this point. The other study showed a similar response.

In two of the 12 experiments data suggesting a second bypass tract were obtained. Stimulation of a point marked as 6 close to, and postero-inferior to, point 5 at the root of the inferior vena cava showed conduction to the ventricle even after the normal A-V conduction (fig. 4A) was blocked by giving acetylcholine (fig. 4B). In this stimulation the interval between the stimulus artifact and the ventricular action potential was 53.7 msec, much shorter than that of 120.7 msec in the control state. The QRS complex in the lead II electrocardiogram was inverted and its duration was prolonged to 57.7 msec compared with upright configuration and duration of 50.2 msec in the control state, as is shown in figure 4. When a transverse incision was made below this point, complete block of conduction occurred, as shown by no response in this figure (fig. 4C). At this time stimulation below this incision caused a ventricular response similar to that made on stimulation of point 6 (fig. 4D). Thus another bypass tract was presumed to run through this incision.

In an experiment searching for the mode of conduction, the conduction times at each site and their relations were similar to those in the

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**Figure 3**

Mode of conduction by the first bypass tract of the A-V conduction. A and V are taken by adjacent bipolar leads applied near the fossa ovalis inside the right atrium (RA) and in the center of the free wall of the right ventricle (RV), respectively. N and H are action potentials obtained by the microelectrode inserted in the A-V node and the bundle of His, respectively. The difference in conduction arrival time between two regions is shown in milliseconds.

In this experiment an anomalous response of earlier activation of the ventricle is seen from the beginning in spontaneous sinus rhythm (a). A transverse incision of the presumed first bypass tract brings about normal order of excitation (b).
spontaneous sinus rhythm, when most of the atrial epicardial points were stimulated. One example, stimulation of point 2, is seen in figure 5c. When point 6 was stimulated, however, A-V conduction was much shorter (fig. 5b). The main reason seemed to lie in the earlier response of the ventricle, considering the above experiment. The responses in the atrium, the A-V node, and the bundle of His occurred more or less simultaneously and may have no relation with the ventricular response.

A third bypass tract was suggested by the following observations in two experiments: In one, sinus rhythm was maintained when the atrial septum was cut through transversely as in figure 6A and B. No prolongation of the P-R interval was observed in lead II (fig. 6B). When the incision reached some point of the posterior free wall, marked prolongation of the P-R interval was induced (fig. 6C). By this incision all the preferential pathways to the A-V node, which were reported elsewhere, were presumed to be cut. Therefore, the delayed but still persistent conduction to the ventricle was supposed to be via another A-V bypass tract. Another possibility, namely that the pacemaker had shifted to some remote point, was excluded since similar conduction to the ventricle occurred by electrical stimulation near the sinus node (fig. 6D). Then the incision was continued transversely in the free wall of the right atrium. When the incision reached some point far anteriorly, complete A-V block occurred (fig. 6E). This showed that one bypass tract passed through this far anterior point.

The mode of conduction was examined in an experiment shown in figure 7. Stimulation of most epicardial points induced responses in the A-V node, the bundle of His, and the ventricle similar to those by sinus rhythm (fig. 7a), as is shown in the result of stimulation of point 1 (fig. 7b). Only when point 3 was stimulated, was an abnormal response noticed (fig. 7c). The delay was most conspicuous between the A-V node and the bundle of His, but the interval between the bundle of His and the ventricle was also prolonged. It was supposed that the stimulus entered the A-V node at an unusual place, which would cause an especially tortuous path in the network of the A-V node and the bundle of His.

Discussion
In contrast to the general belief that the only normal communication between atria

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Figure 4
A-V conduction through the second bypass tract. In the normal Tyrode solution stimulation of most atrial points induced normal responses as is seen on stimulation of point 1 (A). By giving acetylcholine to the concentration of 10\(^{-6}\) g/ml the A-V block occurred and no responses were observed by stimulation of most points (not shown). Only stimulation of point 6 induced conduction to the ventricle, but the A-V conduction time (the interval between the stimulus artifact and the QRS complex of lead II in the upper tracing or the action potential obtained by the suction electrode in the lower tracing) shortened with a marked change of the QRS configuration (B). After a transverse incision below this point, no response was observed except for stimulus artifact (C). Stimulation of a point distal to the incision induced a response similar to B (D).
and ventricles in mammals is via the A-V node and A-V bundle. Kent\(^1\)–\(^7\) reported a muscular bridge connecting the right atrium and the right ventricle and other pathways bridging the atria and ventricles in human and lower mammalian hearts. Later some investigators regarded these pathways as a basis of a clinical syndrome referred to now as the WPW syndrome.\(^17\)–\(^18\) Considerable disagreement has persisted on this correlation and as to whether this bundle of Kent exists in the human heart. In several cases it has been demonstrated,\(^18\)–\(^19\) but in other cases no A-V bridge has been found.\(^20\)–\(^24\) Lev\(^15\) pointed out that in more and more cases of the WPW syndrome this bundle was demonstrated when complete serial sections of all possible pathways were made.\(^25\)–\(^29\) but at least in one such study it was not found.\(^29\) Truex and his associates\(^30\) found the bundle only up to the age of 6 months. James\(^31\) had occasionally seen it in adults without WPW. Especially there still is lack of knowledge on whether the bundle of Kent is found normally without the WPW syndrome.

Recently increasing evidence has been proposed for this correlation in the open chest. Durrer and Roos\(^31\) found the earliest point of excitation near the right lateral part of the anterior A-V sulcus in a patient with the WPW syndrome. Burchell and his associates\(^32\) abolished the WPW phenomenon by injecting procaine at the area of pre-excitation during operation. Cobb and co-workers\(^33\) made an incision to interrupt surgically the bundle of Kent after localizing the earliest area of anomalous ventricular activation by epicardial mapping in a patient with the WPW syndrome. The incision abolished its electrocardiographic features and the recurrent tachycardia. All of these were concerned with the so-called type B of the WPW pattern. Dreifus

**Figure 5**

*Mode of conduction by the second bypass tract of the A-V conduction. Abbreviations and figure arrangements are similar to figure 3: a is a response to sinus rhythm, and b and c are responses to stimulation of points 6 and 2 on figure 1, respectively.*

Note that stimulation of point 2, like stimulation of most points (not shown), induced a response similar to that in sinus rhythm. Only stimulation of point 6 induced a different response.
ATRIOVENTRICULAR CONDUCTION

![Figure 6](image)

**Figure 6**

A-V conduction through the third bypass tract. The upper tracings are lead II electrocardiograms and the lower tracings are action potentials obtained by the suction electrode. See text for explanation.

et al., ligated surgically the A-V bundle of a patient with WPW, type A, and found no A-V block, which suggested activation of the ventricles by the accessory A-V bundles. Complete elimination of recurrent tachycardias after A-V ligation was emphasized.

Anatomic studies, on the other hand, afforded findings for other A-V connections. Mahaim and his associates frequently found small connections between the A-V bundle and the interventricular septum. They also found small communications between the left bundle branch near the bifurcation and the interventricular septum, and occasionally between the antero-inferior part of the A-V node and the septum. This was confirmed anatomically in man by some investigators. James reported specialized atrial fibers bypassing the upper A-V node or atrionodal junction. Lev accepted all of these pathways and the right- and left-sided bundles of Kent.

Although our study shows the presence of some functioning bypass tracts of the A-V conduction, we do not know the relation of our findings with anatomic findings as yet. The first bypass tract showed a shortening of the P-R interval and no significant change in the QRS complex (fig. 2bD and 1bE). The anomalous conduction made by this tract was shown only in the earlier excitation of the ventricle with no significant changes in arrival time in the A-V node and the bundle of His (fig. 3). It is supposed, therefore, that this tract bypasses the A-V node and an upper part of the bundle of His and enters its lower part (fig. 8). The second bypass tract showed a shortening of the P-R interval and induced a widening and inversion of the QRS complex (fig. 4). The negativity of its chief deflection in lead II suggested that this type corresponded to type B of WPW pattern. The localization of this tract suggested also that this is probably the bundle of Kent, bypassing the whole A-V conduction system at the lateral wall of right atrium and ventricle (fig. 8). The earlier activation of the ventricle by this tract compared to the control conduction confirms this supposition, but the earlier activation of the A-V node and the bundle of His, while rather unexpected, is not entirely contradictory (fig. 5). This might depend upon the way of stimulation and needs further studies. The third bypass tract showed a prolongation of the P-R interval and little change in the QRS complex (fig. 6). The delay occurs in the A-V node (fig. 7). The third tract is supposed to enter the A-V node in an unusual way bypassing some part of this node (fig. 8). The anomalous entry may cause a delay due to detour in this netlike tissue. Since counterparts for these three types can be found easily in clinical electrocardiography, it is possible that this finding in the rabbit heart can be applied to the human heart.
These bypass tracts operated usually by stimulation of special atrial points in the presence of A-V block and only occasionally in the presence of sinus rhythm. The normal spread of the sinus node impulse probably enters the bypass with difficulty. There may be some anatomic reason at some region that prevents easy conduction. At least under ordinary conditions normal conduction through the His-Tawara A-V conduction system occurs more easily. In the propagation from the normal direction, one of the bypass tracts can be entered when the difficult conduction is overcome by some condition. But it can be entered also by the propagation from a different direction, namely, when the pacemaker shifts from the ordinary site in the sinus node to a slightly different site or it appears at another point in the atrium. There is a possibility for the third bypass tract that it is latent in the normal A-V conduction and becomes manifest in the A-V block.

The histologic examination of these bypass tracts is being carried out in our laboratory.

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Figure 7

Mode of conduction by the third bypass tract of the A-V conduction. Abbreviations and figure arrangements are similar to figure 3; a is a response to sinus rhythm, and b and c are responses to stimulation of points 1 and 3, respectively.

Stimulation of point 1, and also of most points (not shown), induced a response similar to that in sinus rhythm. Only stimulation of point 3 induced a different response.

In this experiment atrial responses were too small to be determined accurately. Therefore stimulus artifact (S) was adopted for measurement instead of atrial activation (A).
Figure 8

Schematic diagram of presumed bypass tracts. (1) The first bypass tract with a short P-R interval and a normal QRS complex. (2) The second bypass tract with a short P-R interval and an abnormal QRS complex. (3) The third bypass tract with a prolonged P-R interval and a normal QRS complex. Abbreviations are the same as in figure 1.

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