EDITORIALS

Conduction and Block
in the Right Bundle Branch
Real and Imagined

ELECTROCARDIOGRAPHY is a subject now sufficiently old that it should contain more science than art, particularly in view of the sophisticated methods currently available to study the electrical activity of the heart. That many quaint terms have persisted in electrocardiographic nomenclature may be more attributable to their charming lilt than their scientific accuracy. We have long suspected that “incomplete right bundle-branch block” was one of these, and in the October issue of CIRCULATION Moore and his colleagues presented firm evidence to support that suspicion.1 If the electrocardiographic pattern of QRS prolongation up to 0.11 sec (in adults), with a terminal r’ in V1 and S wave in leads V5 and V6 or standard lead I, were often the sole consequence of delayed conduction within the right bundle branch, then the term incomplete right bundle-branch block to describe this pattern might be appropriate. Conversely, if delay in conduction in the right bundle branch is only inconsistently present in this electrocardiographic constellation, then the diagnosis of incomplete right bundle-block would be at best imprecise and often incorrect.

What Moore and his colleagues have now demonstrated electrophysiologically is the absence of a conduction delay in the right bundle branch, and a histologically normal right bundle branch in dogs with a heritable electrocardiographic pattern of incomplete right bundle-branch block. Their conclusion that the electrocardiographic findings in these dogs were caused by a heritable focal hypertrophy of the right ventricle, and not a delay in conduction in the right bundle branch, is elegantly documented. The study proves the inaccuracy of the electrocardiographic diagnosis of incomplete right bundle-branch block in these dogs, and invites an analysis of the possible alterations in the electrical activation of the heart that may produce the identical electrocardiographic pattern in man. All explanations for incomplete right bundle-branch block are likely to share the requirement that some portion of the right ventricle continue to depolarize after the completion of left ventricular depolarization.

Delayed Termination of Right Ventricular Depolarization

Sherf and James2 have suggested that the pathways traveled from the sinus node to each

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portion of the myocardium must be the same from one normal beat to the next and that any change in the route of spread of the impulse may alter the activation of localized areas of the heart. If this concept is correct, then a lesion in either the atrial internodal pathways or the atrioventricular node or the His bundle may block those fibers conducting the impulse destined for the right bundle branch and thus delay depolarization of parts or all of the right ventricle. The essential point is that the ancient and useful electrocardiographic term bundle-branch block may be an anatomic misconception in many instances.

In his detailed electrocardiographic-pathologic correlative studies of bundle-branch block, Lenegre\(^3\) noted that the right bundle branch was histologically normal in 76\% of 33 patients with incomplete right bundle-branch block, but that 94\% had right ventricular hypertrophy. There are at least three ways in which right ventricular hypertrophy per se may contribute to delay in right ventricular depolarization: (1) by prolonging conduction time in the normal right bundle branch; (2) by prolonging conduction time in the normal right ventricular subendocardial Purkinje network; and (3) by increasing the duration of right ventricular endocardial to epicardial activation time due to increased wall thickness. Prolongation of conduction time within the right bundle branch and the subendocardial Purkinje network may be the result of right ventricular dilatation, increased right ventricular intracavitary pressure, or both. Therefore, several factors may contribute to the incomplete right bundle-branch block pattern with underlying right ventricular hypertrophy. To call attention only to the increased conduction time within the right bundle branch is not only scientifically misleading, but from a clinical standpoint it may distract the electrocardiographer from considering the diagnosis of right ventricular hypertrophy.

Typical and atypical right bundle-branch block are frequently noted following open intracardiac operations, and are most often attributed to trauma to the right bundle branch. Several studies\(^4,\,5\) have now shown that these conduction defects occur precisely at the moment of right ventriculotomy and are therefore more logically the result of interruption of the right ventricular subendocardial Purkinje network than of damage of the right bundle branch.

**Early Termination of Left Ventricular Depolarization**

Thus far, the mechanisms postulated as responsible for the incomplete right bundle-branch block pattern have been those causing delay in completion of right ventricular depolarization. A short duration or abnormally early onset of left ventricular depolarization may also cause terminal right ventricular activation to be unopposed. The former frequently occurs in transition from infancy to childhood consequent to the changing ratio of right-to-left ventricular wall thickness. The latter may theoretically result from preexcitation of the left bundle branch.

The evidence presented by Moore and his colleagues that the incomplete right bundle-branch block pattern may be due to a heritable focal hypertrophy of the right ventricle, and not because of delayed conduction within the right bundle branch, should stimulate us to reappraise the validity of our electrocardiographic vocabulary. Continued anatomic and electrophysiologic research is vital to verify or disprove our current electrocardiographic concepts, so that the words accurately reflect the best known facts about electrical activity of the heart.

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**References**


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