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-branch 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 atroventricular 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-patho-
logic 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 net-
work; and (3) by increasing the duration of
right ventricular endocardial to epicardial
activation time due to increased wall thick-
ness. Prolongation of conduction time within
the right bundle branch and the subendo-
cardial 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 mis-
leading, but from a clinical standpoint it may
distract the electrocardiographer from consid-
ering the diagnosis of right ventricular hyper-
trophy.

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 interrup-
tion 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 preexcita-
tion 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 conduc-
tion 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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Circulation, Volume XLV, January 1972

Conduction and Block in the Right Bundle Branch: Real and Imagined
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Circulation. 1972;45:1-3
doi: 10.1161/01.CIR.45.1.1

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

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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