THE USE OF artificial pacing has greatly intensified interest in chronic A-V block. This interest, in turn, has brought into sharp focus the rare type of A-V block described independently in 1906 by Wenckebach and by Hay and classified by Mobitz in 1924 as type II. This type of block is characterized by failure of a ventricular response, without antecedent progressive lengthening of A-V conduction time. Furthermore, in its more advanced form several consecutive atrial impulses are blocked, giving rise to high degree partial block and to intermittent periods of prolonged ventricular asystole. Although Mobitz had appreciated the distinction between the functional nature of type I and the organic nature of type II block and had stated clearly that type II block was apt to be a forerunner of complete A-V block and Stokes-Adams attacks, it is surprising how the distinction between the genesis and prognosis of these two types of block has become blurred in clinical electrocardiography. Although a definitive anatomic-electrocardiographic study that would relate type II block to a particular anatomic lesion is still missing, the observations of Mahaim, Yater and associates, Lev and Unger, and Lenègre and Moreau strongly suggest that bilateral bundle-branch block may be the usual anatomic substrate of type II A-V block. Recent clinical observations have shown that second degree A-V block of type II (Mobitz) is frequently associated with bundle-branch block, especially right bundle-branch block with left axis deviation.

It is surprising that physiological studies to explain the nature of type II block are almost nonexistent. The recent electrophysiological observations of Watanabe and Dreifus appear to be in keeping with the assumption, based on clinical observations, that an infra-A-V nodal lesion is the cause of type II A-V block.

The need for distinction between the two types of second degree A-V block with respect to their underlying pathology and their clinical significance is critical when A-V block develops acutely in the course of recent myocardial infarction: In our experience, recent posterior (inferior) wall infarction associated with reversible ischemia of the A-V node or common bundle, or both, as a rule leads to type I A-V block (Wenckebach periods), whereas anteroseptal infarction associated with an irreversible lesion (necrosis) of the bifurcation, or the bundle branches or both, usually leads to type II second degree A-V block (stable P-R interval, block of two or more successive atrial impulses) or complete (antegrade) A-V block (slow, unstable idioventricular pacemaker).
If 2:1 block predominates, the distinction between type I and type II may be difficult; however, in the majority of cases the proper classification is possible by close observation which usually will reveal a transition from, or to, a lesser degree (3:2) of A-V block, or to A-V dissociation with occasional ventricular captures. Comparison of the P-R intervals of the conducted beats or of the captures in the same record will reveal the expected variations of the P-R interval in the case of a type I block and a constant P-R interval in the case of a type II block. A study\textsuperscript{11} published in this issue of Circulation lists every 2:1 A-V block as type II. If the authors avoided classifying 2:1 block as type II in those cases in which the development from, or to, a type I block (“in a stepwise fashion”) was observed, then the data tend to confirm not only the above-mentioned relationship between location of the infarct and site of the lesion in the conduction system but also between the site of the lesion and the type of second degree A-V block. At the same time the different prognostic significance of the two types of A-V block becomes evident.

Complete A-V dissociation developing in posterior (inferior) wall infarction is frequently due to the concomitant acceleration of an A-V junctional pacemaker and should not be classified as complete A-V block.\textsuperscript{12} The erroneous equation of “third degree” or complete A-V block and complete A-V dissociation has caused confusion regarding both prognosis and management of A-V block in recent myocardial infarction. A-V block associated with posterior (inferior) wall infarction as a rule does not produce prolonged asystole and tends to subside spontaneously. In our experience, artificial pacing has been unnecessary except (1) in cases of type I A-V block with a ventricular rate under 50/min, after an attempt to accelerate the rate with isoproterenol has failed and (2) in the rare cases of type II A-V block. It would appear that type I block does not significantly influence immediate prognosis unless it is associated with shock or congestive failure; however, unexpected “late” death, a week or more after restoration of A-V conduction, is not uncommon. In contrast, A-V block associated with anterior wall infarction worsens the immediate prognosis, causes Stokes-Adams attacks, and requires artificial pacing immediately.

This editorial is, therefore, a plea for clinical distinction between type I and type II A-V block, especially in recent myocardial infarction. It emphasizes the need to investigate the physiological basis and the anatomic-electrocardiographic correlation of the two types of A-V block. It calls for reappraisal of the prognostic significance of A-V block in recent myocardial infarction and of the indications for artificial pacing based upon the following criteria: (1) localization of the infarct (anterior vs. posterior or inferior), (2) type of A-V block (Wenckebach vs. Mobitz type II), (3) site of the subsidiary pacemaker (supraventricular vs. idioventricular), and (4) cause of A-V dissociation (interference vs. block), as indicated by the ventricular rate.

Richard Langendorf
Alfred Pick
Chicago, Illinois

References

7. Lenègre, J.: Contribution à l’étude des blocs de


Atrioventricular Block, Type II (Mobitz)—Its Nature and Clinical Significance
RICHARD LANGENDORF and ALFRED PICK

Circulation. 1968;38:819-821
doi: 10.1161/01.CIR.38.5.819

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1968 American Heart Association, Inc. All rights reserved.
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:
http://circ.ahajournals.org/content/38/5/819.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/