A-V CONDUCTION DISTURBANCE: ITS PATHOPHYSIOLOGY AND PHARMACOLOGY

By Yoshio Watanabe

Recent experimental and clinical studies have elucidated many complex aspects of A-V conduction disturbance. This paper illustrates dissimilarity of response produced in different portions of the A-V transmission system by various pathophysiologic and pharmacologic factors. Transmembrane potentials were recorded from the A-V junctional region of isolated, perfused rabbit hearts together with atrial and ventricular electrograms, permitting subdivision of the total A-V interval into intraatrial (A), intra-A-V nodal (N) and His-Purkinje (HP) conduction times.

Generalized ischemia or anoxia always caused a marked prolongation of the N conduction time and A-V nodal block, little affecting A and HP intervals. Action potential duration of the A-V junctional fibers was shortened, particularly with anoxia. Ligation of the left coronary arteries more markedly prolonged HP conduction time and caused subnodal block. Cardiac glycoside, low potassium or high magnesium concentration predominantly depressed N conduction, often causing block within the A-V node, but without significantly affecting A and HP intervals. In contrast, quinidine, propranolol, diphenylhydantoin, tetrodotoxin and high potassium prolonged A and HP intervals, without affecting or sometimes even shortening the N conduction time. These agents tended to increase the amplitude and duration of the action potential from the A-V nodal fibers. A third group of factors (low sodium concentration and SU-13197) depressed conduction in all three portions of the A-V transmission system.

These results well explain different patterns of A-V block associated with anterior and posterior wall infarctions, and also suggest specific indications of various pharmacologic agents in the presence of A-V conduction disturbances.

Hahnemann Medical College, Philadelphia, Pennsylvania, U.S.A.

* * * * *

LOCALIZATION OF A-V BLOCK BY HIS BUNDLE RECORDINGS AND ITS CLINICAL SIGNIFICANCE

By Roger P. Javier and Onkar S. Narula

His Bundle (BH) recordings were obtained in 75 patients with normal ECG's (Group A) and 193 patients with various degrees of A-V block (Group B). The conduction times through the atrium (P-A), A-V node (A-H) and His Purkinje system (H-V) were analyzed during sinus rhythm and atrial pacing (AP). In Group A the control P–A=25–45 msec (37 $\pm$ 7), control A–H= 50–125 msec (77 $\pm$ 16) and the control H–V=35–45 msec (40 $\pm$ 3); 1:1 conduction was seen on AP from 130-180/ min and second degree block, when seen, was always localized in the A–V node.

Group B: (1) In 80 patients with 1° block the delay was localized in the P-A (4%), A-H (90%) and H–V (60%); (2) In eleven patients with 2° type I block, the block was localized in the A-H (82%), BH (9%) and H-V (9%); (3) In 16 patients with 2° type II block the defect was localized in the BH (31%) and H-V (69%); (4) In 16 patients with high grade 2° A-V block (2:1, 3:1) the block was localized in the A-H (38%), BH (12%) and H-V (50%); (5) In 70 patients with complete heart block, the block was situated in the A-H (14%), BH (14%) and distal to the BH (72%).

This study demonstrates that all three degrees of A-V block can result from conduction abnormalities present in any region of the conduction system and may be of great therapeutic and prognostic value.

Division of Cardiology Mount Sinai Medical Center, Miami Beach, Florida 33140.