CASE REPORTS

Complete His-Purkinje Block Produced by Carotid Sinus Massage

Report of a Case

By Ernesto A. Jonas, M.D., Bernard D. Kosowsky, M.D., and Krishnaswamy Ramaswamy, M.D.

SUMMARY

A case of complete heart block (CHB), localized in the His-Purkinje system, induced by carotid sinus massage (CSM) is presented. A 63-year-old male with right bundle branch block and left anterior hemiblock was evaluated for recurrent syncope. Right or left CSM produced brief periods of CHB with presyncopal symptoms. His bundle (HB) studies during normal sinus rhythm revealed normal conduction times (A-H interval = 80 msec; H-V interval = 48 msec). Carotid sinus massage produced progressive slowing of the sinus rate, and complete heart block below the HB occurred whenever the sinus rate fell below 42 beats/min. During atrial pacing at 70 beats/min, CSM produced 2:1 block above the HB with an effective rate to the HB of 35 and complete block below the HB. Atrial pacing at rates above 93 beats/min resulted in 2:1 block below the HB. Administration of intravenous atropine produced an apparent junctional tachycardia with 2:1 block below the HB. Thus, complete heart block related to both bradycardia (phase 4) and tachycardia (phase 3) was demonstrated. The complete heart block induced by CSM was thought to be secondary to bradycardia-induced left posterior fascicular or intra-His block. However, the possibility of a direct vagal effect on ventricular conduction could not be ruled out.

Additional Indexing Words:
Bradycardia-dependent block
Phase 3 heart block
His bundle recording
Vagal effect
Phase 4 heart block
Trifascicular block

SYNCOPE due to transient complete heart block has been well described.1 His bundle studies have confirmed that failure of impulse propagation occurs usually in the ventricular Purkinje system,2,6 an area considered to be devoid of significant cholinergic innervation.4 Patients prone to Stokes-Adams attacks often have evidence of bundle branch disease during periods of intact atrioventricular conduction.7 It is presumed that syncope results from the transient interruption of conduction in the functioning fascicle(s).

There are several mechanisms that may explain the intermittent nature of this heart block but in most cases one cannot be isolated. Bundle branch block induced by tachycardia has been well documented8 and may explain the occurrence of complete heart block brought on by exercise.9 Less commonly, bradycardia can cause a conduction defect. Massumi10 reviewed the subject of bradycardia-dependent bundle branch block and proposed criteria for its diagnosis. This phenomenon has also been demonstrated in dogs following acute ligation of the anterior septal coronary artery.11

The following case history illustrates tachycardia- and bradycardia-related block in the posterior fascicle of the left bundle in a patient with right bundle branch block (RBBB) and left anterior hemiblock (LAH), which develops into complete heart block. In this patient, trifascicular (complete) block could be consistently reproduced by carotid sinus massage (CSM). We believe this to be the first documented report of this occurrence.
Case History

A 63-year-old male was seen on March 27, 1973, for evaluation of three episodes of syncope. The electrocardiogram revealed normal sinus rhythm at a rate of 60 beats/min with a P-R interval of 0.19 sec and a Q-T interval of 0.45 sec. There was RBBB with an axis of $-60^\circ$. Comparison with previous tracings from other physicians showed a normal ECG in 1967 and RBBB with left axis deviation in 1969.

A review of a 24-hour monitor tape revealed normal sinus rhythm at rates of 60 to 88 beats/min with frequent runs of 2:1 A-V block without prolongation of the P-R interval preceding the nonconducted P waves. The patient was hospitalized for pacemaker implantation and carotid sinus massage was performed in order to help assess A-V nodal conduction. This procedure produced progressive slowing of the sinus rate to 53 beats/min, followed by three blocked atrial beats associated with ventricular asystole lasting 5.2 sec and then resumption of normal conduction.

In order to establish the site of A-V conduction disturbance, a His bundle study was performed prior to the implantation of a permanent pacemaker. A 6 USCI bipolar pacing catheter was passed percutaneously from the right femoral vein and positioned across the tricuspid valve to record the His bundle electrocardiogram (HBE). A 5 USCI bipolar pacing catheter was passed from the same vein to the right atrium and utilized for atrial pacing. His bundle electrograms were recorded at 25 and 100 mm/sec on a Cambridge multichannel fiberoptic photographic recorder, using a frequency range of 40 to 500 Hz. Studies were performed during normal sinus rhythm and during atrial pacing with and without left or right carotid sinus massage. The effect of infusion of 1 mg atropine intravenously was also studied.

Results

His bundle electrograms recorded at rest during normal sinus rhythm at 63 beats/min (P-P = 950 msec) revealed an A-H interval of 80 msec and an H-V interval of 48 msec (fig. 1). On several occasions, left or right carotid sinus massage induced progressive sinus slowing from rates of 60 to 75 beats/min (P-P = 800 - 1000 msec) to approximately 42 beats/min (P-P = 1430 msec). Transient complete heart block followed this slowed rate. During these periods, there were three to seven successive P waves without a ventricular depolarization. Following this, conduction resumed and the sinus rate returned toward control levels. His bundle electrograms revealed gradual prolongation of the A-H interval with unchanged H-V times after the initiation of CSM. In each instance, His bundle deflections followed the blocked sinus beats (fig. 2).

Atrial pacing with 1:1 capture could be achieved at rates of up to 93 beats/min. Above this, 2:1 block occurred. His bundle electrograms during atrial pacing at 93 beats/min with 1:1 conduction revealed an A-H interval of 95 msec and an H-V interval of 48 msec. At faster pacing rates 2:1 A-V block occurred during which His bundle depolarizations followed every P wave (fig. 3).

In an attempt to maintain a constant atrial rate during CSM, atrial pacing at a rate of 71 beats/min (P-P = 850 msec) was performed. Prior to CSM, the P-R interval was 0.19 sec. Figure 4 depicts the effect of CSM during atrial pacing. There is an initial prolongation of P-R interval to 0.21 sec with no apparent change in H-V time. Ventricular asystole follows due to four nonconducted atrial beats. The
first and third of these beats are blocked within the A-V node; no His spikes are discernible on the HBE. The second and fourth beats are associated with His deflections with normal A-H intervals but are blocked beyond the His bundle. Thus, CSM produced transient 2:1 A-V nodal block with a His bundle rate of 35 beats/min, and complete block below this area. This was interrupted when the next beat was conducted through the A-V node with a prolonged A-H time, upsetting the ventricular bradycardia. This block alternating between the A-V node and His-Purkinje system was reproducible with CSM during constant rate atrial pacing.

The slow intravenous administration of 1 mg atropine produced an accelerating junctional rhythm. When the junctional rate exceeded 86 beats/min (R–R = 700 msec), apparent 2:1 antegrade block ensued, with 1:1 retrograde activation of the atria. His bundle electrograms demonstrated normal H-V intervals (45 msec) during 1:1 antegrade conduction (fig. 5). The amplitude of the His spikes which were conducted to the ventricles was smaller than that of the blocked beats, and the H-V time of these beats was slightly shorter than during 1:1 antegrade conduction. Although the His to His spike interval is constant throughout the strip depicted in figure 5, it is conceivable that the last four ventricular beats represent escape beats originating in the distal His-Purkinje system causing retrograde depolarization of the His bundle followed by antegrade conduction to the ventricles. This interpretation results in an H spike preceding each QRS complex with an H-V time shorter than that of normal antegrade conduction. Carotid sinus massage immediately following atropine administration produced no conduction abnormalities.

**Discussion**

The ventricular conduction system is composed of three main anatomic and physiologically discrete fascicles: the right bundle branch and the anterior and posterior divisions of the left bundle branch. In his review of intraventricular trifascicular blocks, Rosenbaum considers eight different combinations of intraventricular and atrioventricular conduction disturbances based on whether conduction is permanently or only intermittently interrupted in these fascicles. Chronic asymptomatic fascicular block, whether idiopathic or the result of ischemia, is frequently bifascicular in nature, involving most often the right bundle and the anterior division of the left bundle. Under these circumstances, His bundle studies have shown variable conduction patterns in the remaining fascicle. Narula and Samet reported prolonged H-V conduction times in nearly 75% of asymptomatic patients with RBBB and LAH. On the other hand, His bundle studies in patients with bifascicular block and a history of syncope have shown impaired conduction in the remaining fascicle in almost 100% of the cases, even in the presence of a normal P-R interval on the surface ECG, although some exceptions have been reported.

The present case illustrates right bundle branch block, left anterior hemiblock, and syncopal attacks, with a normal P-R interval and apparently normal conduction through the posterior division of the left bundle, as evidenced by the normal H-V time. Alterations in conduction through the posterior fascicle could be elicited by carotid sinus massage or by increasing the heart rate via atrial pacing.

Transient complete heart block occurs frequently during CSM. This usually results from suppression of conduction in the A-V node and is often accompanied by evidence of first degree or Mobitz I block immediately preceding or following the period

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*Figure 4*

The effects of carotid sinus massage recorded on leads II, V1, and HBE during atrial pacing at a rate of 71 beats/min. Note the presence of H spikes following the second and fourth blocked atrial beats.

*Figure 5*

Leads II, V1, and HBE following administration of 1 mg atropine sulfate intravenously.
of A-V dissociation. His bundle recordings have confirmed that A-V block induced by CSM is located proximal to the His bundle.\textsuperscript{19, 20} In the present case, A-V block during CSM was localized below the His bundle. In view of the pre-existing bifascicular disease, complete block may have resulted from transient blockade in the posterior division of the left bundle.

The ability of carotid sinus massage to affect the ventricular conduction pattern was first reported by Wilson in 1915.\textsuperscript{21} He noted prolongation of the QRS complex in one patient during carotid massage and with deep respiration. Several subsequent reports have demonstrated the induction of bundle branch block or left axis deviation by CSM. In each instance, there was slowing of the sinus rate associated with the conduction defect. Comeau et al.\textsuperscript{22} suggested a direct effect of the vagus on ventricular conduction. Dresler\textsuperscript{23} implicated a coronary vasoconstrictor effect of vagal stimulation as the cause for impaired conduction. Wallace and Laszlo\textsuperscript{24} suggested either vagal inhibition of conduction or hypotension secondary to bradycardia as the mechanism of bundle branch block induced by CSM. Puech et al.\textsuperscript{25} demonstrated prolongation of H-V conduction time during carotid sinus stimulation in a patient with left bundle branch block. The authors attributed this to a vagal effect from carotid sinus stimulation. However, there was also a slowing of the sinus rate associated with this change.

Although vagal fibers are found primarily in the sinoatrial node, atrial muscle fibers, and the A-V node, recent studies have demonstrated parasympathetic neural elements extending to the ventricular myocardium.\textsuperscript{26} Both positive and negative inotropic effects have been demonstrated following vagal stimulation in intact animals and isolated hearts.\textsuperscript{27} Scherf et al.\textsuperscript{28} demonstrated enhanced ventricular ectoropic activity with carotid sinus massage and suggested a direct vagal mechanism as a possible etiology. Others have reported the induction of ventricular fibrillation following CSM.\textsuperscript{29, 30} Kent et al.\textsuperscript{31, 32} have shown that stimulation of the vagus nerve decreases vulnerability of the canine ventricle to fibrillation. This effect is thought to be mediated by a rich network of cholinergic nerves intimately related to Purkinje fibers in the ventricular septum. Bailey and associates demonstrated that acetyl-choline can affect conduction in spontaneously firing Purkinje fibers.\textsuperscript{33} However, a direct parasympathetic effect on intraventricular conduction has never been demonstrated \textit{in vivo}.\textsuperscript{24} In all instances in which ventricular conduction has been altered by vagal influences, there have been attendant rate changes which could adequately account for the phenomena.

Several recent reports have implicated electrophysiological rather than neuro-humoral mechanisms as the cause of CSM-induced conduction block.\textsuperscript{35-37} The rate of initial depolarization of the action potential is a major determinant of the speed of conduction in Purkinje fibers.\textsuperscript{38} The rate of this phase 0 depolarization depends primarily on the level of the membrane potential at the time of stimulation. The transmembrane potential is reduced before completion of repolarization (phase 3), and stimulation during this period may result in aberrant conduction. This mechanism would account for bundle branch or complete block induced by tachycardia, as manifested in this patient.

The membrane potential of Purkinje fibers may also be low as a result of spontaneous phase 4 depolarization and/or hypopolarization. In the presence of a small degree of hypopolarization, spontaneous phase 4 depolarization may cause impairment of conduction, especially when the rate is sufficiently slow.\textsuperscript{35} Bradycardia-dependent bundle branch block\textsuperscript{10, 36, 37} and left anterior hemiblock have been described.\textsuperscript{35} Counel et al.\textsuperscript{39} reported two cases of A-V block elicited by premature beats. In one case with RBBB, premature atrial beats induced complete heart block and His bundle recordings localized the conduction defect to the left bundle branch. The block was dependent on the interval length from the preceding conducted beat and was thought to represent bradycardia-induced A-V block. El-Sherif et al.\textsuperscript{11} have observed tachycardia- and bradycardia-dependent left bundle branch block in three patients with acute myocardial infarction. They were able to produce similar types of block in dogs subjected to ligation of the anterior septal coronary artery.

Both tachycardia- and bradycardia-dependent block were demonstrated in the present case. Rapid heart rates induced by atrial pacing caused 2:1 block, presumably due to incomplete recovery in the posterior division of the left bundle. Carotid sinus massage induced atrial slowing and transient complete heart block. When attempts were made to stabilize the atrial rate by pacing the atrium at a constant rate of 71 beats/min, carotid sinus massage induced 2:1 block in the A-V node. This allowed only half of the paced atrial impulses to be transmitted through the His bundle to the ventricular Purkinje system, leading to bradycardia-induced block.

Phase 4 depolarization at slow heart rates has been implicated in the mechanism of conduction block induced by CSM. Rosenbaum and associates\textsuperscript{40} concluded that the most likely explanation for bradycardia-dependent block was hypopolarization plus spontaneous diastolic depolarization and a shift of the threshold potential toward zero. The resultant lowered membrane potential would adversely affect
the rate of depolarization of the subsequent beat and could produce conduction block. In their reported cases, cessation of paroxysmal A-V block was always associated with a ventricular escape beat which presumably activated the blocked region, thus depolarizing it, and allowing antegrade conduction to resume. In the present report, however, conduction resumed without escape beats. Rosenbaum alluded to this latter phenomenon and attributed block in these circumstances to repetitive concealed conduction; that is, repeated partial penetration of the conduction system resulting in block of several consecutive beats. Normal conduction could resume under these circumstances without the need for an escape beat. The mechanism for the resumption of conduction in the present case is not entirely clear.

Some authors have questioned the role of phase 4 depolarization in bradycardia-dependent block. Further experimental and clinical data will be necessary in order to make a more definitive determination of the mechanisms involved.

It is possible that block may have occurred within the His bundle itself, or that slowed conduction in this area contributed to the left posterior fascicular block. Narula has shown that the His bundle can be considered as a separate portion of the conduction system which is subject to block within it. In the present case, there appeared to be a decrease in the amplitude of His spikes which failed to conduct to the ventricle (fig. 3). Although this variation was readily reproducible, it, in fact, may have been a reflection of catheter tip motion. However, it is conceivable that altered conduction within the His bundle caused smaller HB complexes and antegrade block.

During the apparent junctional rhythm with 2:1 antegrade block induced by atropine, the blocked beats displayed larger His spikes than did the conducted beats. This may reflect alternating conduction within the bundle. If this rhythm is interpreted to be an escape mechanism originating in the distal His-Purkinje system, then the site of A-V block could be within the distal portion of the His bundle rather than in the posterior fascicle.

The role of tachycardia- and bradycardia-dependent block in causing the clinical manifestation, Stokes-Adams syncope, is not fully known. Rosenbaum suggests that combined phase 3 and phase 4 block may be a frequent cause of complete heart block. In the present patient and in Coumel’s case, the “zone of opportunity” during which A-V conduction could occur was relatively small. Beats falling earlier or later than this period in diastole were blocked. In El-Sherif’s experiments, this zone narrowed progressively until complete block ensued several hours following infarction. Re-establishment of this critical zone occurred before complete recovery of conduction took place. Exercise-induced syncope in patients with Stokes-Adams syndrome may reflect tachycardia-dependent block. It is conceivable that syncopal attacks occurring at rest, during sleep, or following minor changes in vagal tone, may represent bradycardia-dependent block.

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ERNESTO A. JONAS, BERNARD D. KOSOWSKY and KRISHNASWAMY RAMASWAMY

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