Reversible Wenckebach Type Atrioventricular Block Associated with Severe Coronary Artery Disease

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HIGH-grade atrioventricular heart block in patients with coronary artery disease is generally thought to be irreversible and in most cases atropine produces little or no effect. Nevertheless, the possibility of significant vagal action should always be considered in any patient with heart block. When such a factor is present, the administration of atropine will produce a striking improvement in atrioventricular conduction.

We are presenting detailed observations on an unusual patient with heart block: chronic atrioventricular block of the Wenckebach type was associated with severe coronary artery disease and congestive heart failure; atropine abolished the block, prevented Stokes-Adams attacks, and controlled an increase in the block that was produced by digitalis. These observations indicate that there may be functional as well as organic components in this type of heart block, and emphasize the importance of testing the effects of atropine in such patients.

Case History

L. R., an 85-year-old man, was admitted to the Beth Israel Hospital in October 1955 because of increasing congestive heart failure. He had had 2 acute myocardial infarctions (in 1943 and 1951) followed by angina pectoris and congestive heart failure. In 1951 he began to have episodes of syncope associated with first-degree heart block (P-R interval 0.30 second) and sinus bradycardia (rate 55 to 60 per minute). Frequent episodes of light-headedness and occasional attacks of syncope occurred during the ensuing 4 years. Partial heart block with the Wenckebach phenomenon was present for at least 3 years prior to 1955.

Repeated attempts at digitalization, both at home and in the hospital under close observation, had been unsuccessful because drug administration was associated with increased heart block. Frequent periods of 2:1 atrioventricular block were produced with ventricular rates as low as 30 beats per minute.

On examination severe congestive heart failure was found. An electrocardiogram (fig. 1A), as in the past, showed sinoatrial bradycardia and atrioventricular block with Wenckebach periods.

During this admission it was decided to analyze in detail the patient's conduction defect in an attempt to clarify both the response to digitalis and the mechanism of the syncopal episodes. The effects on conduction of atropine, carotid sinus pressure, digoxin, and exercise were studied. Atropine abolished the spontaneous block and produced normal atrioventricular conduction. Carotid sinus pressure, digoxin, and exercise increased the conduction defect; in each case atropine prevented the increase. These observations form the basis of this report and are detailed in the next section.

Observations

The cardiac effects of carotid sinus pressure, atropine, digoxin, and exercise were studied over a 27-day period; 124 electrocardiograms were taken on 17 days during this period. Each electrocardiogram was recorded from lead I for at least 1 minute; most tracings were longer, some as long as 4 minutes when dropped beats were infrequent or absent. All electrocardiograms unless otherwise specified were taken with the patient at rest in bed in a semirecumbent position for at least 10 minutes.

The following measurements were obtained from each electrocardiogram: (1) the maximum and minimum P-P intervals during a period of at least 1 minute; (2) the sinoatrial rate (beats per minute) determined by counting the number of P waves in 15 to 60 seconds; (3) the maximum and minimum P-R intervals during a period of at least 1 minute; (4) the average number of dropped beats (nonconducted P waves) per minute determined from the full length of each tracing; (5) the ventricular rate (beats per minute) determined...
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by subtracting the number of dropped beats from the sinoatrial rate.

**Effects of Carotid Sinus Stimulation**

Light pressure applied to either the right or the left carotid sinus (day 7) produced complete heart block with a slow atrioventricular rhythm (fig. 1B); there was no change in the sinoatrial rate.

**Effects of Atropine**

One milligram of atropine sulfate intravenously (day 11) completely abolished the spontaneous atrioventricular block; the P-R interval became normal (0.16 second) and dropped beats disappeared, despite a marked increase in the sinoatrial rate (fig. 1C). At the time of this atropine effect firm pressure applied to either carotid sinus had no effect on atrioventricular conduction or on the sinoatrial rate.

Atropine sulfate administered subsequently by mouth also improved atrioventricular conduction: 2 hours after 0.6 mg. (day 12) dropped beats were absent and the P-R interval was stable at 0.28 second; 3½ hours after 1.2 mg. (day 13) dropped beats were absent and the P-R interval was stable at 0.22 second. Atropine in doses of 1.2 mg. by mouth every 6 hours produced moderate dryness of the mouth.

**Combined Effects of Atropine and Digoxin**

Atropine sulfate and digoxin were administered simultaneously for 6 days (days 13 to 18): 1.2 mg. of atropine sulfate by mouth every 6 hours and a total of 2.0 mg. of digoxin (0.75 mg. intravenously on day 13 and then 0.25 mg. by mouth daily for 4 days). During the first 5 days the P-R interval (determined 4 hours after a dose of atropine) gradually increased: on the fourth day of combined drug therapy (day 16) it was 0.26 second; on the fifth day (day 17) 0.32 second. On the sixth day (day 18) there were periods of 3:2 block, 2:1 block, and marked respiratory sinus arrhythmia (fig. 2). With this high degree of block

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Fig. 1 Top. Lead I. A. Control: sinoatrial bradycardia (P-P 1.24 seconds) and partial atrioventricular block. There is progressive increase in the P-R intervals before the non-conducted P wave (Wenckebach-type block). B. During right carotid sinus pressure: complete atrioventricular block (P-P 1.40 seconds, R-R 2.0 to 2.2 seconds). C. Three minutes after intravenous atropine: sinoatrial rhythm (P-P 0.72 second) with normal atrioventricular conduction (P-R 0.16 second).

Fig. 2 Bottom. Lead I. Digoxin effects: A, 3:2 atrioventricular block; B, 2:1 atrioventricular block (R-R 2.28 seconds); C, sinoatrial arrhythmia.
the ventricular rate was sometimes as low as 4 beats in 10 seconds and the patient complained of lightheadedness and dizziness. Accordingly, digoxin was omitted and atropine administration (1.2 mg. by mouth every 6 hours) was continued.

The action of atropine sulfate was studied in detail (days 19 to 23) as the effects of digoxin on atrioventricular conduction disappeared. Figure 3 shows the changes observed between 2 oral 1.2 mg. doses of atropine sulfate given 6 hours apart on the second day after the digoxin was stopped (day 20). At the time of the first dose, there were 16 dropped beats per minute, the P-R interval varied between 0.28 and 0.38 second, and the P-P interval ranged from 1.05 to 1.13 seconds. At the height of the atropine effect, 1½ hours after the dose, all sinoatrial beats were conducted to the ventricle, the P-R interval was stable although still longer than normal, and there was no sinus arrhythmia. At 3½ hours after the dose, the sinus arrhythmia reappeared, at 5 hours the P-R interval again began to vary, and at 5½ hours dropped beats were again observed.

On the following day (day 21) also, dropped beats reappeared 5½ hours after a dose of atropine sulfate. However, on the fourth and fifth days after the omission of digoxin (days 22 and 23) no dropped beats were noted at any time, just as had been the case before digoxin was started (days 12 and 13). The P-R interval varied from 0.24 second (1 to 3 hours after a dose of atropine sulfate) to 0.28 second (5 to 6 hours after a dose of atropine sulfate).

**Effects of Exercise**

All these observations were carried out with the patient at rest, since it was frequently noted that exercise increased the number of dropped beats. This effect of exercise was studied by taking electrocardiograms after the patient had performed 3 consecutive sit-ups in bed from a semirecumbent position.

On days 20 and 21, when a definite digoxin effect was present, 5 observations on the effect of exercise were made 5 to 6 hours after a dose of atropine at a time when dropped beats were occurring at rest: in each case exercise increased the number of dropped beats per minute (by 3 to 10). On day 22, when there was no longer a digoxin effect and atropine completely prevented dropped beats at rest, exercise had no effect when tested 5 times between 3½ and 6 hours after a dose of atropine.

In order to study the effects of exercise in the absence of digoxin or atropine effects, a single dose of atropine was omitted on day 23 and the effects of exercise were observed for 11 hours after the last dose (fig. 4). Dropped beats appeared with exercise 8½ hours after the dose; they did not appear at rest until 2½ hours later. When dropped beats were present at rest, exercise increased their frequency, so that there were occasional periods of 2:1 block. With the mild exercise that was sufficient to increase the conduction defect no change was observed in the resting sinoatrial rate, which varied between 56 and 62 beats per minute.

A new, long-acting preparation of atropine (Prydon) was tested on day 28: 0.8 mg. by mouth prevented dropped beats for at least 12 hours both at rest and with exercise. At the time of discharge on the thirty-second hospital day the patient was receiving 0.8 mg. of this preparation by mouth twice a day and he was free of Stokes-Adams attacks.

**Discussion**

This patient with heart block of the Wenkebach type had a remarkable lability of atrioventricular conduction, which served as an extremely sensitive indicator of extracardiac influences on the conduction mechanism.
Atropine abolished the spontaneous block, indicating that the conduction defect was caused by intrinsic vagal activity. This observation is striking, since it is most unusual for atropine to abolish such a high degree of heart block in a patient with severe coronary artery disease.  

Carotid sinus pressure, digoxin, and exercise increased the block. These effects were of vagal origin as shown by the fact that atropine abolished them.

Marked sensitivity of the heart to reflex vagal stimulation is often observed in patients with coronary artery disease as shown by the frequent finding of a hyperactive carotid sinus reflex. Usually the sinoatrial node is more sensitive to carotid sinus stimulation than the atrioventricular node, and considerable slowing of the sinoatrial rate occurs before there is any appreciable effect on conduction. In this patient, however, the atrioventricular node was far more sensitive than the sinoatrial node, and light carotid sinus pressure produced complete heart block although there was no slowing of the sinoatrial rate.

That digoxin increased the block in this patient is clear on the following grounds: the same phenomenon had been observed several times before when a cardiac glycoside had been given; such high degrees of block (3:2 and 2:1) were never observed in the resting state at other times; and conduction returned to its previous state 4 days after digoxin was stopped, as would be expected from the known duration of action of the drug. Although cardiac glycosides do not ordinarily affect preexisting heart block, it is well recognized that they may interfere with atrioventricular conduction through a vagal mechanism as was observed in this patient. This vagomimetic action should not be confused with the direct action of the glycosides on conduction, which may be observed with larger doses and which is not changed by atropine. The sinus arrhythmia associated with digoxin was also a vagal effect; this respiratory arrhythmia increases as vagal action on the sinoatrial node increases.

The vagomimetic effect of exercise observed in this patient is contrary to what we might have expected, since exercise is generally associated with sympathetic stimulation and vagal inhibition. Indeed, others have observed in individuals without heart disease that Wenckebach block reversible by atropine is also abolished by exercise. The effect of exercise in this patient with severe coronary artery disease, however, may have been associated with slight alterations in coronary blood flow or may be explained by accepted neurophysiologic phenomena. Cessation of reflex stimulation of the sympathetic pathways may be followed by rebound overactivity of the previously inhibited vagal pathways. Thus, immediately following exercise in this patient, when the vagomimetic effect was observed, there may well have been a rebound of the previously inhibited vagal supply to the atrioventricular node. It should be emphasized that the effect of exercise was observed only at the atrioventricular node; there was no associated increase in the sinoatrial rate, which might otherwise have explained the increased block.

The observations in this patient are all consistent with localized changes in the atrioventricular node, causing an increase in its sensitivity to neurogenic and pharmacologic stimuli. Similar localized sensitivity has been described in another patient with coronary artery disease and a hyperactive carotid sinus cardiac reflex, and may be attributed to degenerative changes in the intrinsic cardiac ganglia or in the nodal tissue.

These observations were valuable in the clinical management of this patient by clarifying his response to digoxin. Digitalization was impractical because uncomfortable dryness of the mouth occurred, even with doses of atropine that only partially controlled the increased block. However, in an emergency, it was thought that digitalization could be safely accomplished by simultaneously giving larger doses of atropine.
This patient with partial atrioventricular block had Stokes-Adams disease manifested by attacks of syncope or other symptoms of cerebral ischemia due to a slow ventricular rate. Complete heart block was observed with carotid sinus pressure (fig. 1B) and periods of 2:1 block with either exercise or digoxin administration; in either case ventricular rates were slow enough to produce cerebral ischemia. Atropine in doses that did not produce dryness of the mouth prevented these attacks. This therapeutic effect of atropine in our patient is unusual, since in most cases of Stokes-Adams disease the heart block is not affected by the drug.  

**Summary**

A patient with severe angina pectoris and congestive heart failure had chronic Wenckebach-type atrioventricular block and episodes of syncope. Atropine abolished the block and prevented the Stokes-Adams attacks. Carotid sinus pressure, digoxin, and exercise increased the block; atropine prevented these effects.

**Summario in Interlingua**

Un paciente con severa angina de pectore e congestiva disfallimento cardiac habeva chronico bloco atrio-ventricular del typo Wenckebach e episodios de syncope. Atropina aboliva le bloco e preveniva le attaceos de Stokes-Adams. Pression al sinus carotidie, digoxina, e exercicio augmentava le bloco. Atropina preveniva iste effectos.

**REFERENCES**


Aortic stenosis was clinically diagnosed in 326 patients from the Johns Hopkins Hospital and the Cardiac Clinic of the Harriet Lane Home. Sixty-three of the 326 patients were found to have neurologic manifestations such as syncope, headaches, convulsions, dizziness, hemiparesis, and deafness. Hemiparesis occurred in 7 patients with acquired aortic stenosis, but did not occur in any of the patients with congenital aortic stenosis; deafness occurred in 3 patients with the congenital lesion, but in none of the patients with acquired aortic stenosis. Syncope and seizures were the most common neurologic manifestations occurring in 36 of the 63 patients.
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