Atrial Ectopic Activity Associated with Sinus Bradycardia

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SUMMARY
Five cases of atrial ectopic activity associated with sinus bradycardia are reported. The ectopic activity was abolished in all cases when the sinus rate was increased spontaneously or by atropine. The report demonstrated that the incidence of ectopic activity increases in the atrium at the slower basic rates as it does in the ventricle. It was emphasized that atrial ectopic activity associated with sinus bradycardia should be treated by atropine or by rapid pacing of the atrium because atrial premature beats have the potential of inducing more serious atrial tachyarrhythmias. The possible role of the vagus nerves in facilitating the induction of atrial tachyarrhythmias was discussed, and it was suggested that atropine may have an added benefit of reducing the likelihood of development of atrial tachyarrhythmias.

Additional Indexing Words:
Atrial tachyarrhythmias Increased vagal activity Atropine Pacing of atrium

It has been well established that ectopic beats occur in the ventricle more frequently when the basic rate is relatively slow and that these ectopic beats disappear when the basic rate is increased by artificial pacing or by administration of atropine.\textsuperscript{1-6} The efficacy of increased heart rate in supressing ventricular ectopic beats has been particularly apparent in patients with acute myocardial infarction.\textsuperscript{4-6} Han and associates\textsuperscript{2} suggested that the increased incidence of ectopic activity at slow ventricular rates might be due to re-entrant activity resulting from an increase in the asynchrony of repolarization among different myocardial fibers. They demonstrated a greater range of refractory periods at various points on the ventricular surface when the basic rate was slow. In the same study, the range of refractory periods was also increased in the atrial tissue at slow basic frequencies. It follows, then, that ectopic activity should be more frequent in the atrium when the basic rate is relatively slow. The present paper describes clinical cases of increased atrial ectopic activity associated with sinus bradycardia.

Report of Cases

Case 1
H. C., a 75-year-old female, developed weakness, dizziness, nausea, and vomiting 30 min after receiving meperidine (Demerol), 50 mg, and promethazine (Phenergan), 25 mg, intramuscularly as preoperative medications for cataract surgery. She had heart disease of unknown etiology for 15 years, and she was started on digitalis about a year ago for mild heart failure. Physical examination revealed an irregular pulse of 56/min and blood pressure of 70/10 mm Hg. The patient was lethargic but oriented. There was cardiomegaly with a left parasternal heave and a palpable systolic thrill in the fourth intercostal space. A grade III/VI pansystolic murmur was audible in the same area and at the apex. There were also bilateral basal rales. The
chest x-rays revealed an increase in the cardiac silhouette with a slight increase in pulmonary vascular markings. The ECG was consistent with sinus bradycardia of 50/min with nonphasic sinus arrhythmia, nonconducted premature atrial beats occurring at approximately 10/min, and complete right bundle-branch block (fig. 1A). Her central venous pressure at this time was 17 cm H₂O. Because of bradycardia, hypotension, and congestive failure, the patient was given atropine 1 mg intravenously in two divided doses, which brought a dramatic increase in the sinus rate to 105/min (fig. 1B and C). With the increased sinus rate, premature atrial beats were completely abolished, BP rose to 120/70 mm Hg, central venous pressure fell to 11 cm H₂O, and the lungs cleared in a short time.

**Case 2**

O. B., a 73-year-old male, was first found to have sinus bradycardia with occasional atrial premature beats about 20 years ago during a preoperative ECG for herniorrhaphy. He admitted to having had some symptoms of congestive failure for some years, for which he has taken digitalis and a diuretic. He was admitted this time for increasing dyspnea and frequent anginal attacks. Physical examination showed an irregular pulse at 40/min and BP of 150/90 mm Hg. There was cardiomegaly up to the anterior axillary line with a grade II/VI systolic ejection murmur, and the third heart sound gallop was audible at the apex. The ECG (fig. 2) revealed a sinus rate of about 40/min, a sinus arrhythmia, frequent conducted and nonconducted atrial premature beats occurring in bigeminy, and left bundle-branch block (fig. 2A). After administration of 0.5 mg of atropine intravenously, the sinus rate increased to 80/min, and the ectopic activity disappeared completely (fig. 2C). Since chronic atropine administration was not feasible in this patient, a demand ventricular pacemaker was implanted. As shown in figure 2D, the ventricle was depolarized at a rate of 75/min by spontaneous sinus beats and the pacemaker stimuli alternately. Significant improvement of his general condition resulted.

**Case 3**

E. B., a 83-year-old female, was brought to the emergency room because of sudden onset of weakness, dyspnea, dizziness, nausea, and recurrent vomiting. She denied chest pain or past history of any heart disease, diabetes, and hypertension. On physical examination, BP was 90/50 mm Hg, pulse rate was 47/min, and respiration, 26/min. The patient was oriented and showed no sign of congestive failure. The ECG showed sinus bradycardia at a rate of 50/min with frequent premature atrial beats and changes of acute inferior myocardial infarction. Runs of atrial tachycardia were occasionally initiated by atrial
premature beats (fig. 3A). The patient was given atropine, 1 mg intravenously, the sinus rate increased to 105/min, and premature beats disappeared completely (fig. 3B).

**Cases 4 and 5**

J. S., a 79-year-old female, was known to have diabetes and hypertension, and had been taking digitalis and chlorothiazide for several years. She was admitted this time for fibrosarcoma of left shoulder. The significant physical findings included a grade II/VI systolic ejection murmur over the precordium and BP of 170/100 mm Hg. The ECG showed sinus bradycardia of 40/min with frequent premature atrial beats (fig. 4A) and left ventricular enlargement. When the sinus rate was subsequently increased to 70/min, premature atrial activity was not present (fig. 4B).

H. F., a 63-year-old male, was admitted for observation because of anginal pain and dyspnea. The ECG showed no significant changes except for mild sinus arrhythmia. As shown in the bottom tracing of figure 4, premature atrial beats appeared during the phase of slower rhythm at about 65/min and disappeared when the sinus rate increased to 70/min.

**Figure 2**

Case 2. (A) Very frequent conducted and nonconducted premature atrial beats occurring in bigeminy during sinus bradycardia of 40/min. (B) Decreased atrial ectopic activity with an increase in the sinus rate to 65/min after atropine. (C) Complete disappearance of premature atrial beats at a sinus rate of 80/min. (D) Functioning QRS-triggered demand pacemaker set at a rate of 75/min.

**Figure 3**

Case 3. Lead V1. (A) Sinus bradycardia at 50/min with a run of atrial tachycardia. (B) No atrial ectopic activity during sinus tachycardia of 105/min after administration of atropine.
Lead I

A

B

Lead V1

Figure 4

Case 4. (A) Atrial ectopic activity during sinus bradycardia at 40/min. (B) Disappearance of premature atrial beats with a spontaneous increase in the sinus rate to 70/min. Case 5 (the bottom tracing). Sinus arrhythmia with atrial ectopic activity during the slow phase.

Discussion

The cases described clearly demonstrate that ectopic activity is increased in the atrium when the basic heart rate is slowed just as it is in the ventricle. One of the possible mechanisms of ectopic impulse formation is focal re-excitation of already repolarized fibers by the flow of current between these and neighboring fibers which are still depolarized. When neighboring fibers repolarize within a few milliseconds of each other, re-excitation cannot occur, for the difference in potential between them will not exceed the threshold of already repolarized fibers, but increased temporal dispersion of repolarization would increase the potential difference and initiate re-excitation. Han and associates demonstrated that the range of refractory periods at various points in the ventricular or atrial tissue is increased when the basic heart is relatively slow. Accordingly, closely coupled ectopic beats would be more likely to develop at slow basic frequencies. Sinus bradycardia in some of our cases may be due to moderate vagal stimulation. Vagal stimulation shortens refractory periods nonuniformly at various points in the atrium and hence increases the range of refractory periods. This may further increase the asynchrony of recovery of excitability in the atrium. It is, therefore, suggested that the administration of atropine would be more beneficial than the use of artificial pacing when they are used to increase the sinus rate. In all of our patients who received atropine, the sinus rate increased significantly, resulting in the disappearance of atrial ectopic activity.

Atrial ectopic beats are of great significance in patients with acute myocardial infarction because of their potential of initiating atrial tachyarrhythmias and seriously compromising cardiac output. Supraventricular tachycardia, either atrial or A-V junctional in origin, has been known to be initiated by atrial premature beats. The tachycardia may result from sustained reciprocation of impulses between the atrium and the nodal tissue (the
sinus or A-V node). A premature impulse may enter the node slowly and emerge to the surrounding atrial tissue as a re-entrant beat (an echo), and such re-entrant activity may be sustained to underlie an episode of tachycardia. The possible increased vagal activity in some patients with sinus bradycardia may facilitate the induction of such re-entry by slowing conduction in the nodal tissue and by shortening refractory period in the atrium. Atrial flutter may be due to a circus movement around an obstacle (most probably one of the caval openings) in the atrium, which can be initiated by a premature atrial response. If the premature beat is early enough, it may propagate in only one direction but be blocked in the opposite direction (unidirectional block) around an obstacle to initiate the circus movement. Such unidirectional block would be more likely to occur when the asynchrony of recovery of excitation is increased as a result of nonuniform vagal effects on the atrial refractory periods. Degeneration of atrial flutter into fibrillation would be facilitated in patients whose sinus bradycardia was related to an increase in vagal activity, since it is believed to set the stage for formation of irregular independent wavelets of excitation by increasing the nonuniformity of excitability in the atrial tissue.

The treatment of sinus bradycardia in patients with acute myocardial infarction should be of great value, for this may not only reduce the likelihood of development of atrial premature beats and the resulting tachyarhythmias but may also improve cardiac output and symptoms of congestive failure. This can be achieved by rapid atrial pacing or administration of atropine or catecholamines, but atropine may have an added beneficial effect in reducing the chance of initiation of tachyarrhythmias when patients are suspected to have increased vagal activity as a cause of sinus bradycardia, as in cases of inferior myocardial infarction. One of our patients (case 3) had episodes of potentially serious atrial tachycardia during sinus bradycardia which completely disappeared when the sinus rate was increased by atropine. In case 5, the sinus rate was critical; an increase in the sinus rate of only 5/min abolished atrial ectopic activity.

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