Case Report

Different treatments for different mechanisms in vasovagal syncope
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Abstract The treatment of vasovagal syncope has been by far unsatisfactory. Beta-blockers may prevent vasovagal syncope, but they exacerbates heart asystole. Cardiac pacing prevents syncope but not presyncope. The frequent, serious vasovagal syncope attacks of a 63-year-old woman patient were completely prevented by administration of 100 mg metoprolol (b.i.d) for 3 months until the patient experienced a complete heart block. A DDD pacemaker implantation abolished syncope but not the presyncope, which was eventually prevented in a follow-up period of 24 months by adding 75 mg atenolol twice a day. This case suggests a different mechanism involved in vasovagal syncope. (J Geriatr Cardiol; 2006; 3(1):61-64)

Key words vasovagal syncope; beta-blocker; cardiac pacing; tilt table test; heart block

Beta-blockers have been used effectively to prevent vasovagal syncope,1,2 especially in those having tachycardia immediately before syncope attacks.3 However they may also exacerbate syncope in some cases.4 Although it was reported that cardiac pacing did not prevent or even delay the onset of vasovagal syncope,1,2,5,6 many studies recently reported that pacemaker implantation for patients with vasovagal syncope could significantly reduce the recurrent syncope but not the presyncope.7,8 These results suggest that there might be multiple factors involved in the mechanism of this syndrome and the treatment should be individualized.

Here we report a case aiming at emphasizing the importance to understand the mechanism of syncope for an appropriate treatment.

Case report

A 63-year-old female patient with recurrent syncope for 3 months was admitted to Peking Union Medical Hospital. The recurrence of syncope became increasingly frequent from once a week to once or twice a day. Most of the attacks occurred while she was standing and were preceded with palpitation and a hot feeling in the chest. The patient was injured several times, including left arm fracture and scalp laceration, during the syncope episodes.

On physical examination the cardiovascular system was unremarkable. She was moderately overweight and had well-controlled diabetic mellitus, hypercholesterolemia, and mild hypertension. Coronary angiography showed a localized stenosis (less than 50%) in the proximal left anterior descending coronary artery. No structural abnormalities were found by echocardiography. The ECGs varied from normal (Fig. 1) to various abnormalities including complete right bundle branch block recorded with no cardiac symptoms (Fig. 2). Sinoatrial conduction block was found by tilt table test during syncope.

![Fig. 1. Normal ECG recorded without symptoms](http://example.com/fig1.png)
(Fig. 3), and left bundle branch block was recorded after syncope attack (Fig. 4). An intracardiac electrophysiology study showed normal sinus node function, AH 80ms, HV 52ms, and AV Wenckebach at 450ms. No ventricular tachyarrhythmias were induced with right ventricular apex pacing at both 600/S1/S2/S3 and 400/S1/S2/S3. A vascular ultrasound discovered mild to moderate atherosclerotic lesions in both carotid arteries. No syncope and ECG abnormalities were induced during carotid sinus massage. No abnormalities were found by electroencephalography.

On the first tilt table test the patient’s heart rate was 80 bpm and BP 120/70 mmHg in supine posture. The heart rate progressively increased to 140 bpm at 20 minutes, 75 degree tilting and suddenly dropped to 46 bpm accompanied with loss of consciousness. ECG monitoring showed a 3 to 1 sinoatrial conduction (not recorded). BP was not taken promptly enough until the patient was back to supine posture and was then recorded at 100/60 mmHg. The patient was given a bolus of 2 mg atropine intravenously and gradually recovered to consciousness and normal sinoatrial conduction after 2 minutes. Syncope and presyncope attacks were reduced with metoprolol titration. The patient was discharged after being symptom free for 3 weeks by treatment with metoprolol, 100 mg twice daily. She was free of syncope for a total of 3 months until it recurred one morning during very cold weather. She was immediately sent to a nearby hospital and was found to have complete atrial ventricular block with ventricular escape rhythm at 25-40 bpm. A temporary ventricular pacing was applied and metoprolol stopped. The patient was sent back to our hospital the next day and a DDD pacemaker was then implanted with pacing rate set at 65 bpm. Two weeks later the patient had a repeat tilt table test and it induced presyncope with 2 to 1 atrial-ventricular block (Fig. 2) and BP 86/67 mmHg. Metoprolol was again initiated and maintained at a low dose of 25 mg twice daily. Unfortunately the patient had two more presyncope attacks and incontinence in the following two weeks in the hospital and her BP was below 90/50 mmHg with a heart rate of 65 bpm. Metoprolol was changed to atenolol and then titrated up to 75 mg twice daily. The patient has been free of symptoms for 24 months.

Several therapeutic regimens have been tried for treatment of vasovagal syncope. These include beta-blockers, disopyramide, theophylline, transdermal scopolamine, fludrocortisone, fluoxetine hydrochloride, and dual-chamber

![Fig. 2. Sinoatrial conduction block while syncope during tilt table test](image1)

![Fig. 3. Complete right bundle branch block without symptoms](image2)
Among these regimens, the use of a beta-blocker was the most effective and widely used agent. Akhtar et al reported a successful treatment with beta-blockers in more than 70 percent of patients. A study by Sra et al showed that after a follow-up for 16 months, 18 of the 19 patients (94%) who were treated with drugs alone (metoprolol in 10, theophylline in 3, and disopyramide in 6) remained free of recurrent syncope or presyncope. In a study by O'Marcaigh et al symptoms were improved in 17 of the 19 (89%) patients taking metoprolol therapy. A study by Mahanonda et al randomized 42 patients with vasovagal syncope into oral atenolol medication and a placebo control group. The response rates by negative tilt test after one month of treatment were 62% vs 5% (P=0.0004) in the atenolol and placebo control group, respectively. Moreover, it was reported that patients who received atenolol felt better compared with those who received a placebo (71% vs 29%, P=0.02) (cite literature? to be confirmed by author). Beta blockade decreases contractility and may play roles in preventing the activation of “C” fibers and the subsequent reflex withdrawal of sympathetic support. However, if beta blockade does not prevent sympathetic withdrawal, it may aggravate bradycardia. It has been reported that beta-blocker therapy may cause prolonged cardiac asystole during tilt table test. Moreover, a patient in whom syncope was prevented by oral beta-blocker in a repeated tilt test may experience worsening of syncopal episodes and prolonged asystole. In the present case, a prolonged asystole occurred three months after initiation of a beta-blocker and eventually lead to the implantation of a permanent DDD pacemaker.

Ten years ago, Kus et al reported a case of vasovagal syncope in an otherwise healthy 74-year-old women. Attempts to prevent symptoms with ventricular and atrioventricular sequential temporary pacing proved inadequate. However, the addition of a beta-blocker to permanent DDD pacing was clinically successful in markedly diminishing the symptoms. A report by Sra et al. showed that pharmacological therapy was superior to pacing therapy for the prevention of syncope. A study by el-Bedawi et al found that cardiac pacing did not prevent or even delay the onset of postural syncope. They thought that bradycardia was an unimportant component of vasovagal attacks. However, there are some reports on the usefulness of pacing therapy in malignant vasovagal syncopal patients. In a very recent clinical trial 54 patients were randomized to pacemaker or no pacemaker therapy. There was a statistically significant reduction (85%) in the risk of recurrent syncope in the pacemaker implanted patients but no difference in the risk of presyncope between the two treatment groups. This effect of a pacemaker implantation was confirmed in the present case, in which the presyncope not abolished by atrioventricular sequential pacing was prevented by a beta-blockade.

An additional lesson we get from the present case is that although many abnormalities that might potentially cause syncope are present, the real direct cause of the vasovagal syncope could be something different and can only be explored by tilting.

References


