A 50-year-old male patient continued to experience syncope after implantation of a pacemaker. During cardiovascular examination, the patient showed a typical vasovagal response, with normal pacemaker function. Leg crossing, which prohibits the pooling of blood in the legs and abdomen, at the onset of symptoms helped to prevent this response. The authors recommend a course of leg crossing as a measure to treat vasovagal syncope.

Key words: vasovagal syncope, leg crossing, pacemaker.

A 50-year-old male patient with known paroxysmal atrial fibrillation since 1991 began to experience frequent syncopal episodes in 1997. In 1998, he was admitted to the hospital several times with recurrent episodes of atrial fibrillation. During chemical conversion using intravenous flecainide, sinus arrests of up to 5 seconds occurred, with the patient reporting near syncope. These findings led to the implantation of an AAI pacemaker in February 1998. Several months after implantation, the patient experienced nausea during a stressful conversation and collapsed again. Pacemaker function and routine laboratory examination results were normal at that time.

On his first visit to our outpatient clinic in autumn 1998, the patient told us that, during his syncopal events, he was described as being pale. He also described feeling sweaty and nauseated. The syncopal attacks were provoked by emotional events but also could occur without any direct cause. He regained consciousness within a few minutes. As a child, he had experienced frequent fainting with venipuncture. When visiting the dentist and hospital, he also felt light-headed and sweaty. To condition himself to avoid these spells, he became a blood donor. Remarkably, his syncopal problems resolved for several years, until their recurrence in 1997.

During orthostatic cardiovascular examination in our laboratory with a Finapres device (TNO-Biomedical Instrumentation, Amsterdam, The Netherlands) [1], a pacemaker rhythm with a rate of 69 beats per minute and a finger arterial pressure of 125/85 mm Hg were measured, with the patient in the supine position. The initial orthostatic response in the first 30 seconds after standing was normal [2]. After several minutes of quiet standing, symptoms of sweating, “not feeling well,” and yawning appeared. The patient looked pale. He recognized the symptoms as those he experienced before syncope in daily life. Blood pressure measurements decreased from values of approximately 125/80 mm Hg after 3 minutes of standing to 85/60 mm Hg after 4 minutes. The symptoms resolved quickly when the patient lay down. After standing up for a second time, symptoms appeared within 1 minute, accompanied by hypotension (Fig. 1). The third time, we asked the patient to stand with his legs crossed. No symptoms appeared and blood pressure remained normal (Fig. 1). Pacemaker function was normal during the entire examination.

This history and physiologic response are typical for vasovagal syncope [3]. The patient received an explanation of the underlying mechanism and was advised to increase his salt intake and to apply a routine of maneuvers that increase venous return, such as leg crossing, with the onset of symptoms. The patient’s syncopal problems disappeared for a follow-up period of 18 months.

Discussion

Vasovagal reactions are characterized by decreases in blood pressure and heart rate. Hypotension is caused by vasodila-
Figure 1. (Upper panel) Blood pressure (measured noninvasively using Finapres [FINAP]) and heart rate (HR) of the patient standing up for the second time. The dotted line indicates the moment of standing up. The arrow indicates the onset of symptoms, coinciding with a decrease in blood pressure. (Lower panel) Blood pressure and heart rate of the patient standing with legs crossed, bpm = beats per minute.

tation in the skeletal muscle because of inhibition of sympathetic vasoconstrictive activity [4,5]. In laboratory experiments, a moderate decrease in heart rate has little effect on cardiac output when venous filling pressure is low; therefore, only severe bradycardias contribute to decreases in arterial pressure in vasovagal fainting [3]. This view is supported by the observations that atropine [6,7] and pacemakers [3,8] often have no effect on hypotension in patients with vasovagal syncope or on the speed with which it occurs. Other investigators have reported some beneficial effects of pacing with vasovagal syncope under laboratory conditions [9]. A clinical trial using pacing in patients with vasovagal syncope was also positive [10]. Currently, these conflicting results are difficult to explain. Clearly, more data from the laboratory and from randomized, controlled, double-blind trials are needed. However, the consensus is that pacing in vasovagal syncope is indicated only if pharmacologic and nonpharmacologic management has failed [11].

Leg crossing is a simple method to combat orthostatic intolerance in patients with autonomic failure and in patients with vasovagal episodes [12,13]. Patients are advised to cross their legs closely together and to stand firmly on both legs. Blood pressure increases because of mechanical compression of the leg veins. In healthy subjects and in patients with autonomic failure, this leads to a translocation of blood to the chest, which, in turn, leads to an increase in cardiac output and an increase in blood pressure [14].

This case is of particular interest for two reasons. First, orthostatic stress testing indicated a vasovagal cause for symptoms in this patient with many years of undiagnosed syncope. Second, in this patient with vasovagal syncope, a pacemaker did not improve symptoms, but a program of leg crossing did. We recommend orthostatic stress testing to assess susceptibility to vasovagal syncope, and, if it is diagnosed, we recommend a course of leg crossing as one of the nonpharmacologic measures to treat it.

References