Neurocardiogenic syncope: a case of prolonged asystole

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Head-up tilt test; Pacing; Syncope.

We report the case of a 48-year-old woman with frequent episodes of loss of consciousness. The patient was submitted to head-up tilt testing that evoked a prolonged asystole associated with sphincteric incontinence and loss of urine.

The patient was treated with dual-chamber pacemaker implantation; at a follow-up of 18 months no other episodes of syncope had occurred.

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Introduction

Syncope is defined as a sudden temporary loss of consciousness associated with a loss of postural tone with spontaneous recovery[1]. Loss of consciousness is due to the reduction of blood flow to the medulla, particularly to the nucleus tractus solitarius. Syncope may be due to orthostatic hypotension, a decreased cardiac output, arrhythmias, neurological diseases, and reflex-mediated mechanisms. Vasovagal syncope is the most common and is provoked by activation of the Bezold-Jarisch reflex that is characterized by inappropriate vasodilation and/or bradycardia. The vasodepressor and cardioinhibitory response that causes a sudden fall in blood pressure is extremely variable among patients.

We describe a case of a woman with frequent episodes of syncope for whom head-up tilt testing resulted positive for the cardioinhibitory response with prolonged asystole.

Case report

A Caucasian 48-year-old woman with frequent episodes of syncope was referred to our Department of Cardiology for evaluation. Her history was not suggestive of cardiovascular diseases. Six months before she had fainted while eating in a crowded restaurant. Two weeks previously she suffered two episodes of syncope: the first one was very brief and occurred during abdominal pain. The second time the patient fainted after the perception of pain evoked by a cervical traction maneuver during a physiotherapy visit. In the latter case the husband referred that the loss of consciousness lasted almost 3 min and was associated with loss of urine; he had not noticed any clonic movements or biting of the tongue.

Clinical examination, ECG and echocardiography ruled out the presence of cardiac disease. A massage of the carotid sinus was performed: it did not induce hypotension nor a significant asystolic period. ECG Holter did not show bradyarrhythmias. Head-up tilt testing was performed in accordance with the following Italian protocol: patient slope of 60°, 20 min + 15 min after sublingual nitroglycerin[2]. Sixteen minutes after the beginning of the test the patient fainted without any prodromic symptoms. The hemodynamic profile was characterized by a blood pressure drop followed, after a few seconds, by a prolonged asystole which lasted for 32 s. After 26 s three chest thumps were performed and spontaneous cardiac activity was established in a few seconds (Fig. 1).

As the patient's consciousness was restored a brief period of psychomotor excitation and urinary incontinence followed. In spite of the few episodes of syncope we decided to treat the patient with dual-chamber pacemaker implantation because of the severe cardioinhibitory response. One day after implantation she was symptomatic for pre-syncope immediately after defecation.

One week after implantation the patient underwent head-up tilt testing which induced syncope with a different clinical and

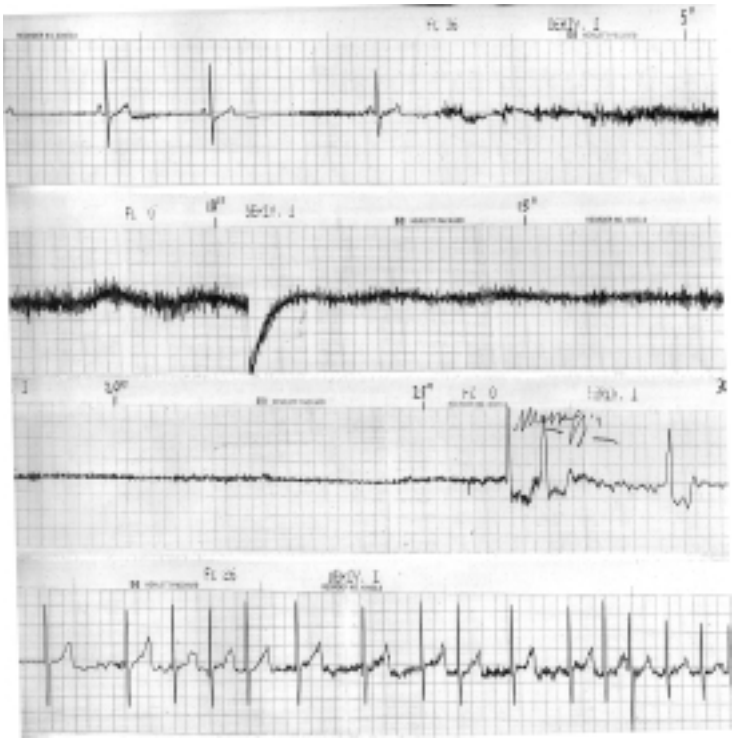

Figure 1. *Surface ECG recording of asystole during head-up tilt testing. The numbers on top of the ECG show how many seconds the asystole lasted. The QRS complexes observed in the third stripe indicate chest thumps. The recovery of spontaneous cardiac activity coincides with the first complex in the fourth stripe.*

hemodynamic profile: there was a prolonged prodromic phase before fainting associated with a severe blood pressure drop and the recovery of consciousness was observed immediately after having placed the patient in a clinostatic position. The ECG showed a correct intervention of the pacemaker immediately after the appearance of the first symptoms. The patient did not present with any other symptoms at a follow-up observation of 18 months.

Discussion

Syncope represents a common event in cardiological clinical practice and affects people of all ages; these patients have a poor quality of life which is inversely proportional to the frequency of syncopal spells[3]. Syncope is often the cause of useless and expensive diagnostic investigations. Often, an accurate anamnesis and clinical examination allow a differential diagnosis between cardiogenic syncope and events caused by other pathologies such as epilepsy.

There are many situations that can direct the diagnosis to a neurally mediated syncope including: prolonged orthostatism, blood vision, pain, and emotional stress. At the same time, there are prodromic symptoms that characterize the same syndrome: nausea, perspiration, dizziness, amaurosis.

In some cases, the absence of a prodromic phase and symptoms such as involuntary movements, sphincteric incontinence and traumas may render the distinction between cardiogenic and epileptic syncope very arduous. These symptoms have always been considered as specific for epilepsy even though they should be considered as poorly specific since they may manifest even in cardiogenic syncope. This is because cerebral anoxia caused by prolonged hypoperfusion may cause clinical symptoms similar to those present in an epileptic attack.

In the clinical case previously described, we hypothesized a neurally mediated syncope due to the fact that both syncopal events were induced by pain. On the other hand, the sphincteric incontinence and clonic movements that occurred during the second episode could also have been due to an epileptic attack. The neurocardiogenic genesis of the second syncopal episode was confirmed by the clonic movements associated with loss of urine observed during head-up tilt testing. Since the asystolic pause lasted for 32 s, we concluded that they were caused by prolonged cerebral hypoperfusion and excluded a neurological disease such as epilepsy.

The benefits of cardiac pacing in patients with cardioinhibitory syncope are presently subject of debate: in three controlled observational studies a total of 77 patients received pacemakers with rate hysteresis and rate-drop responsiveness. The frequency of syncope before and after pacing was compared: pacemaker implantation seemed to provide an improvement in the prevention of vasovagal syncope[4-7]. Similarly three randomized trials demonstrated a decrease in fainting

episodes after permanent pacemaker implantation[8-10]. On the other hand, patients with a cardioinhibitory syncope treated by pacemaker could exhibit subjective benefits due to the placebo effect; the Second Vasovagal Pacemaker Study (VPS II)[11] is a randomized trial assessing whether dual-chamber pacing is superior to placebo. The preliminary results seemed to indicate a favorable but non-significant trend to benefit from pacing. The results of a second placebo-controlled study, Synpace, are awaited[12].

With regard to the present case, we have to admit that the patient was not very symptomatic, having experienced only three syncopal episodes. For this reason, we cannot exclude a possible therapeutic effect linked just to the head-up tilt testing. Nevertheless, the pre-syncopal episode the day after pacemaker implantation, the prolonged prodromic phase before syncope associated with the rapid recovery of consciousness and the correct intervention of the device observed during the second head-up tilt testing as well as the absence of symptoms during a follow-up of 18 months would seem to rule out a placebo effect and could indicate a possible favorable effect of cardiac pacing for the treatment of cardioinhibitory syncope.

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