



Universiteit
Leiden
The Netherlands

Understanding syncope in the framework of transient loss of consciousness

Saal, D.P.

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CHAPTER V

Five cases of complete atrioventricular block induced by bending forward: unusual but not unique

Saal DP | Thijs RD | Bootsma M | Brignole M | van Dijk JG

Abstract

Aims

We describe five patients with syncope caused by a complete AV-block (AVB) while they were bending forward, not rising after bending, and aim to describe the occurrence and the association between bending forward and AVB.

Methods and results

In two patients, bending forward was the exclusive trigger for syncope, while in the remaining three other postural changes (sitting down, standing up, exertion) could also provoke syncope. Complete AVB as the cause of syncope was documented using ECG monitoring in two cases and an implantable loop recorder in the other three. Ectopic beats without preceding sinus slowing occurred before syncope in four cases. Two cases had a left bundle branch block. All patients responded favourably to cardiac pacing.

Conclusion

This is the first case series on complete AVB provoked by bending forward. Syncope during bending forward should suggest a search for an AVB. Arguments in favour of a vagal mechanism were syncope triggered by bending forward and that other triggers could also evoke syncope. However, the absence of sinus slowing before syncope in some cases and the fact that bending forward did not seem to provoke reflex syncope without AVB, cast doubts on a reflex mechanism. There were also arguments favouring conduction disorder: i.e. ectopic beats before syncope and pre-existing conduction disturbances in two cases. The cases are reminiscent of paroxysmal AVB. Discrimination between paroxysmal AVB and vagal AVB is important because a pacemaker is warranted in arrhythmic complete AVB, while the benefit is limited or absent in reflex AVB.

Keywords: paroxysmal AVB, reflex syncope, bending forward, intrinsic AVB

Introduction

The diagnosis of syncope rests on history taking, and, when that does not yield a highly probable diagnosis, on documenting attacks with ECG or blood pressure measurements.¹

The encounter with one patient with a complete atrioventricular block (AVB) during bending forward led us to search for other cases, which revealed four. In all five cases, history taking revealed that syncope could be triggered by bending forward, although it was not the only syncope trigger in some cases. The ECG showed a complete AVB during syncope in all five cases.

We describe the five cases and discuss them in the context of AVB in reflex syncope and as an expression of diseases of the conduction system.

Case 1

A 73-year-old former physician had a history of vasovagal syncope since childhood. He was referred because of episodes of transient loss of consciousness (TLOC) that felt different from his earlier vasovagal syncope. He had had attacks once a month for two years. They started with a tingling sensation in the head that he had learned to recognise as a certain warning of impending TLOC. To avoid injury, he tried to take off his glasses and lie down, but often did not have enough time to do so. He had fallen repeatedly and had sustained injuries. He had broken his glasses on several occasions. Even when he lay down in time to prevent a fall, he still always lost consciousness a few seconds after lying down. His wife, a former nurse, observed that the signs of his spells depended on their duration: during short attacks, his eyes remained closed, he did not become cyanotic and did not snore. During longer attacks, lasting about a minute, his eyes opened, he became cyanotic, made snoring sounds and could become incontinent. Afterwards he was usually disorientated for a few seconds and always felt tired afterwards. The attacks were always triggered: they could occur after sitting down heavily on a chair, straining on the toilet or exerting force on a bicycle pedal, but they were particularly often provoked during bending forward. He therefore tried to prevent this and no longer tied his own shoelaces.

Visits to a neurologist resulted in a normal brain MRI and EEG; two cardiologists had reporting a normal echocardiogram and exercise ECG; no-one had tried to provoke an attack.

A tilt table test with video-EEG monitoring with nitroglycerin provocation and carotid sinus massage was performed, which showed no abnormalities. Afterwards he was asked to stand and bend forward, during which he felt an attack come on. He lay down on a mat and immediately lost consciousness. The ECG showed a third degree AVB with asystole of

28 seconds, preceded by several ventricular escape beats. He snored during the attack, and the EEG flattened during syncope (Figure 1). His wife, present at the recording, recognised this as a typical long attack. He was referred to the cardiology department where he received a pacemaker (Boston Scientific, type: Ingenio MR DR J176, mode DDD with AV search 50-130 beats/min), after which a 12-month follow-up showed an absence of attacks. Follow-up revealed less than 1% pacing.

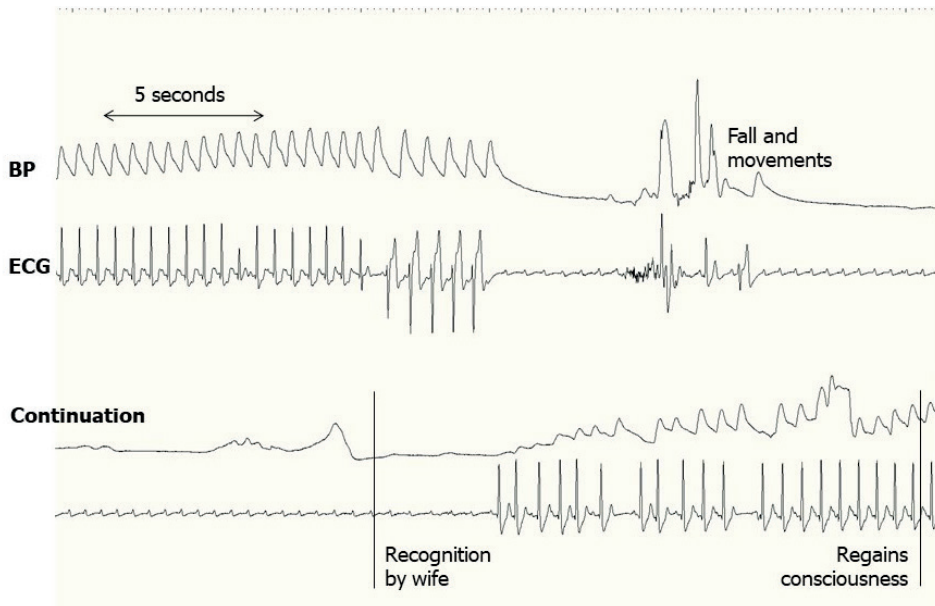


Figure 1. Blood pressure and ECG recording of case 1.

Blood pressure was taken from a finger plethysmograph. Note that the attack starts with several ventricular escape beats, followed by a complete AVB. One or two heart beats appear when he lost consciousness, followed by a longer period of complete AVB. The ECG was recorded using EEG equipment with filter setting different to those of ECG machines, meaning ECG waveforms may be distorted. BP; blood pressure, ECG; electrocardiography, EEG; electroencephalography.

Case 2

A 55-year-old woman with a history of vasovagal syncope in childhood was referred because of about eight TLOC incidents in 18 months, with an increasing frequency. The attacks started with ringing of the ears directly followed by blurred vision. She had no time to sit or lie down as she always lost consciousness within seconds after first noticing the ringing. Bystanders reported pallor of the face and kicking of the legs during attacks. When she regained consciousness, she at first felt like she was waking up and then felt tired, which she recognised as similar to her earlier vasovagal episodes. The attacks were

triggered by bending forwards; examples were picking groceries from a low supermarket shelf, trying on new shoes and bending over to pick up the turds of her dog. After recognising bending forward as a trigger, she tried to avoid doing so, which appeared to help.

Earlier neurological (MRI and EEG) and cardiological examinations had shown no abnormalities, including a normal tilt table test. A Valsalva manoeuvre resulted in a blood pressure decrease to 80/40 mmHg with nausea, but she did not recognize this as similar to the recent spells. A 24-hour ECG and echocardiogram were normal, but no episode had occurred during the recording period. A repeat tilt table test including carotid sinus massage showed no abnormalities. Bending forward did not result in an attack or ECG changes.

After implantation of an Implantable Loop Recorder (ILR) (Reveal Linq, Medtronic) she suffered a typical spell, accompanied by asystole of eight seconds accompanied by an AVB without preceding sinus slowing (Figure 2). A pacemaker (Medtronic, type; Advisa DR MRI A3DR01, Mode: DDD with a lower rate of 50 beats per minute (BPM) and upper 130 BPM) was implanted, and during 9-month follow-up she had no more attacks. The pacemaker reported right atrial (RA) pacing of 0,5% and right ventricle (RV) pacing 0%.

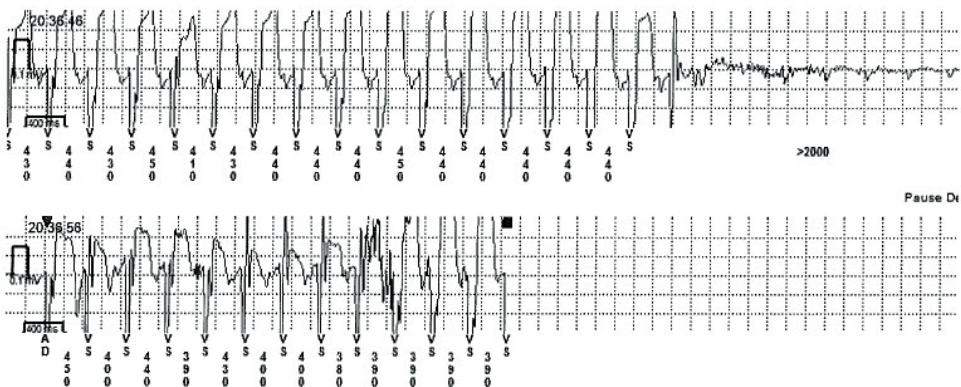


Figure 2. ILR data in case 2.

The ECG shows an asystole for eight seconds without preceding sinus slowing. ILR; implantable loop recorder.

Case 3

A 70-year-old woman with a history of diabetes mellitus type II, had had six TLOC episodes over 10 years. She had been admitted twice because of resulting concussions. The attacks had happened while bending forward. She felt nauseous, followed almost instantaneously

by loss of consciousness and a fall. Eyewitnesses reported that she appeared stiff during unconsciousness. The attacks had taught her not to pick up objects from the floor.

ECG, echocardiogram, a 24-hour ECG recording and a coronary angiogram had showed no abnormalities. A tilt table test including carotid sinus massage, bending forward and a Valsalva manoeuvre showed no abnormalities. After implantation of an ILR (Reveal Linq, Medtronic), several periods of asystole and complete AVB were observed. She subsequently received a pacemaker (Medtronic, type: Advisa DR, MRI/A3DR01, Mode: DDD with AV delay 180ms), resulting in a cessation of attacks (pacing RA 1.6% and RV 1.1% at follow-up).

Case 4

This case has been published previously.² In short, a 64-year-old woman with a history of type 2 diabetes mellitus presented with more than 40 TLOC incidents over seven months. The attacks were triggered by changes in posture, including standing up, bending forwards and sitting down. Her husband described that she became pale, lost consciousness for 30-60 seconds and kicked her legs during the attacks. The ECG had showed a left bundle branch block. A tilt table test showed no abnormalities. She was asked to mimic the last spontaneous attack that had occurred while she picked up fallen oranges from the floor. While doing so, she grunted and slumped forwards to the floor. The ECG showed slowing of a sinus rhythm followed by a complete AVB with asystole of about 10 s. (Figure 3). After implantation of a pacemaker, she suffered no further attacks.

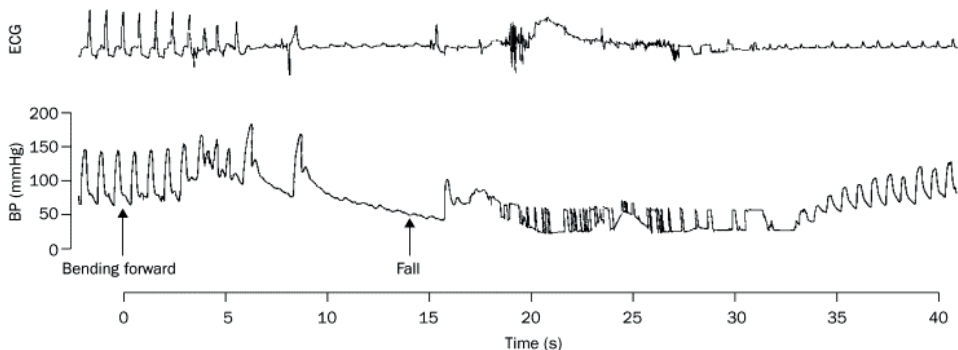


Figure 3. ECG and BP in case 4.

The ECG first showed slowing of a sinus rhythm, followed by a complete AVB with asystole of about 10 s. As in case 1, the ECG was recorded using EEG equipment.

Case 5

A 74-year-old woman presented with TLOC of recent onset. About once every two weeks she experienced episodes of light-headedness, blurred vision and TLOC during standing up, bending forwards or sitting down. Her spouse reported that her eyes widened and turned upwards, and that she then fell over, stiff as a log. There was no time for her to sit or lie down to prevent TLOC. The events caused her great distress and she no longer dared to walk alone or to drive.

Previous cardiological work-up revealed a left bundle branch block. A 24-hour ECG, echocardiogram and exercise testing revealed no new findings. A tilt table test with nitroglycerin provocation and carotid sinus massage did not provoke an attack. Arrhythmic syncope was considered because of the presence of an LBBB¹ and her inability to prevent TLOC by sitting down, so an external loop recorder was ordered. This approach failed to capture the next attack that happened when she bent forward to pick up a piece of soap while showering, because she had detached the electrodes before showering. An ILR was implanted, which did capture a typical event while lying in bed. This was accompanied by asystole and a complete AVB (fig 4) with an asystole of eleven seconds without preceding sinus slowing. A pacemaker (Medtronic, type: Advisa DR MRI A3DR01, Mode: DDD with lower rate 60 and upper rate 130 BPM) was implanted and the patient had no further attacks. Follow-up showed RA 2.7% and RV 9.2% pacing.

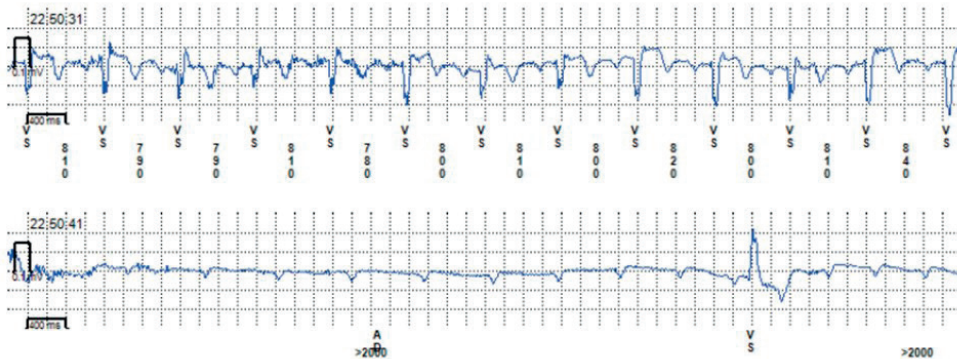


Figure 4. ILR data in case 5.

The ECG shows a complete AVB with asystole for eleven seconds, without preceding sinus slowing.

Discussion

This appears to be the first description of syncope due to complete AVB triggered by bending forward. The diagnosis rested on provoking an attack in two cases and on an ILR in three. Recognising the association between bending forward and an AVB may prompt attempts to provoke syncope and may suggest pacemaker implantation.

Reflex AVB or intrinsic conduction disorder?

A complete AVB may occur as an acquired high-degree or complete heart block, a vagally mediated AV block, a congenital heart block and as 'paroxysmal AVB'.³ The presence of a trigger for syncope usually suggests reflex syncope.¹ However, syncope can also be triggered by external circumstances in some cardiac arrhythmias.

AVB as an expression of reflex syncope

AVB as part of a reflex mechanism was recently described.⁴ Such a 'reflex AVB' is in the majority of cases preceded by slowing of the sinus rhythm.^{4,5,6} This bradycardia suggests that the block is due to parasympathetic influences on the SA- as well as the AV-node.

A preceding bradycardia is however absent in 20% of reflex AVB, as shown by the ISSUE 2 & 3 studies.⁴ An alternate explanation is syncope associated with low adenosine levels.⁴

Reflex AVB has most often been described in syncope with a clear external trigger, such as swallowing⁷, coughing⁸ or emotional distress. However, a trigger can also occur in arrhythmias, such as in 'paroxysmal AVB' (see below), the long QT syndrome (cold water, loud noise)⁹ or the Brugada syndrome (large meal). In the Brugada syndrome, the ST segment change to a type 1 and the occurrence of ventricular fibrillation are related to high vagal tone. This suggests that autonomic influences may trigger arrhythmia.¹⁰

Reflex AVB occurred in approximately 5% of tilt-evoked vasovagal syncope.^{11,12} Patients with reflex AVB often had a long history of syncope, including syncope in childhood.¹³

Complete AVB as an expression of a conduction disorder

The term 'paroxysmal AVB' has been used to indicate any sudden AVB; reflex AVB has in fact been classified as paroxysmal AVB.⁴ However, Lee et al. (2009)³ defined paroxysmal AVB as a sudden, pause-dependent phase 4 AVB, occurring in a diseased conduction system. Paroxysmal AVB in this sense is typically precipitated by slowing of a heart rate, which may be confused with a vagal mechanism. The following characteristics were stated to help differentiate it from reflex AVB. First, paroxysmal AVB was initiated by atrial, His-bundle or ventricular premature extrasystole. Second, tachycardia could suppress AV conduction

and initiate paroxysmal AVB. Third, paroxysmal AVB in the setting of baseline complete AV nodal block has been reported. Fourth, sinus acceleration has been observed during ventricular asystole without affecting the block.³

Finding a balance

Distinguishing between AVB due to a conduction disorder or to a reflex is important, as a pacemaker is warranted in arrhythmic AVB, while studies showed no benefit from a pacemaker in reflex AVB.^{14,15}

The sinus slowing in cases 1 and 4 can be interpreted as a reflex mechanism. Arguments favouring a paroxysmal AVB were ectopic beats before AVB in three cases (cases 1, 4, 5), pre-existing conduction disturbances (i.e. LBBB) in two cases (case 4 and 5), and excellent pacing results in all cases, in line with a study on paroxysmal AVB.¹⁶

A slight modification of heart rate in a bundle branch block may trigger a tachy-dependent or brady-dependent intrinsic AV block.³ Ventricular beats occurred at the beginning of syncope in cases 4 and 5, with a LBBB. Ectopic atrial or ventricular beats can cause paroxysmal blocks.³ AVB was triggered in 19 of 30 cases by atrial or premature ventricular beats or His extrasystole; the trigger in the remaining 11 cases included a Valsalva manoeuvre, reminiscent of bending forward.³ Bending forward may also cause an increase in ventricular preload, with a resulting sudden dilatation of the right heart. The opposite haemodynamic change seems to trigger vasovagal syncope, namely falling venous return. The right atrial baroreceptors are 'stretch' type receptors and could be affected similarly by decrease or increase in right heart volume.

Some of our cases could represent the low adenosine type, given the lack of prodromes; this type also responds well to pacing.⁴

Diagnostic hints

Our study underlines the importance of history taking and documenting circulatory events in syncope. Attempts to provoke syncope with unusual triggers can easily be added to a tilt table test, as recently advocated.¹⁷ If this is unsuccessful, ECG monitoring may be indicated.

Syncope due to bending forward should be distinguished from syncope due to straightening up after bending, as occurs in initial orthostatic hypotension^{18,19} and in classical orthostatic hypotension.

A consistent case feature was a very short warning period before syncope. In a sudden cardiac standstill, the time between the last beat and the onset of loss of consciousness

is only 7-10 seconds.²⁰ Patients may only become aware of something amiss after 4-5 seconds of asystole, leaving them only a few seconds to act. Case one stressed that lying down did not prevent syncope; a failure of lying down to prevent syncope suggests that syncope is due to circulatory standstill rather than to low blood pressure with normal heart rhythm.

Conclusion

AVB should be considered in those presenting with syncope provoked by bending forward.

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