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EDITORIAL

Observations of a syncope doctor during self-induced (near)syncope episodes

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This contribution to the special issue to commemorate the 30th anniversary of *Clinical Autonomic Research* summarizes my observations as a clinician and clinical physiologist on the hemodynamic responses and accompanying symptoms of self-induced (near)syncope elicited by three different provocations: (1) vasovagal near-syncope; (2) fainting lark; and, (3) sinus arrest.

In the early 1990s, Roger Hainsworth introduced tilt-table testing combined with the application of lower body negative pressure (LBNP) using a vacuum cleaner. The provocation caused a large shift of blood from the thorax to the lower body providing a progressive test of orthostatic tolerance [2]. In 1995 the Amsterdam Medical Center syncope unit obtained a tilt-table-LBNP device for physiological studies. Because of my interest in the temporal course of the hemodynamic changes during a vasovagal presyncope, I induced an episode onto myself under the supervision of two experienced syncope doctors (Fig. 1a) [3]. At 38–40 min, increasing LBNP to a negative pressure of − 40 mmHg induced a classical vasovagal reaction with decreases in heart rate (HR), cardiac output, and BP (Fig. 1b). My lowest BP was 30/20 mmHg. I experienced a feeling of warmth followed by lightheadedness but did not faint. After a rapid tilt-down and the discontinuation of the LBNP, there was a recovery of my BP to baseline levels followed by an overshoot with almost immediate disappearance of my lightheadedness. I did not experience visual disturbances.

The classical fainting lark involves squatting in a full knee bend position and hyperventilating by taking about 20 deep breaths. The subject then stands up suddenly and performs a forced expiration against a closed glottis (i.e., a Valsalva maneuver). The fainting lark combines the effects of systemic arterial hypotension induced by acute vasodilatation of the lower limbs (the combination of standing up and the post-ischemic effect of squatting) and decreased cardiac output (effects of arising and raised intrathoracic pressure) and cerebral vasoconstriction induced by hypocapnia (due to hyperventilation) [9]. Using the fainting lark, I induced syncope in myself because of my interest in the time course of the BP changes and accompanying symptoms during this provocation [8]. No such tracings were available in the literature. The fainting lark was performed with minimal straining in order to prevent deep prolonged hypotension with myoclonic jerks [4]. The experiment was supervised by two experienced syncope doctors. I had a precipitous and deep fall in BP when I performed the maneuver. About 4 s after the onset of the fall in BP I experienced a short-lasting lightheadedness and blacked out. The duration of the loss of consciousness was 2–3 s. My lowest BP was 40/20 mmHg, which, upon lying down, rapidly increased with an overshoot (Fig. 1c).

In September 2015 I experienced several episodes of severe lightheadedness when walking in a supermarket. I experienced no other symptoms. I recognized the sensation as identical to those induced by the self-induced syncope episodes described above immediately and presented myself to the cardiac emergency department in the Amsterdam Medical Center. My physical exam was normal. My ECG showed periods of sinus arrest up to 5–8 s and ST elevations compatible with pericarditis (Fig. 1d). In retrospect, I did have mild chest pain that became worse on lying down in the days before the presyncope. Using the ECG monitor in the coronary care unit I made the following observations during admission. In the supine position, periods of asystole up to 7 s did not cause any symptoms. In the upright position, standing against the bed, episodes of 5–7-s asystole elicited severe lightheadedness, but visual disturbances or near-syncope did not occur. The timing of the onset of lightheadedness was very reproducible during about five episodes. I was diagnosed with a sick sinus syndrome and implanted with a dual-chamber pacemaker. A causal relation with the

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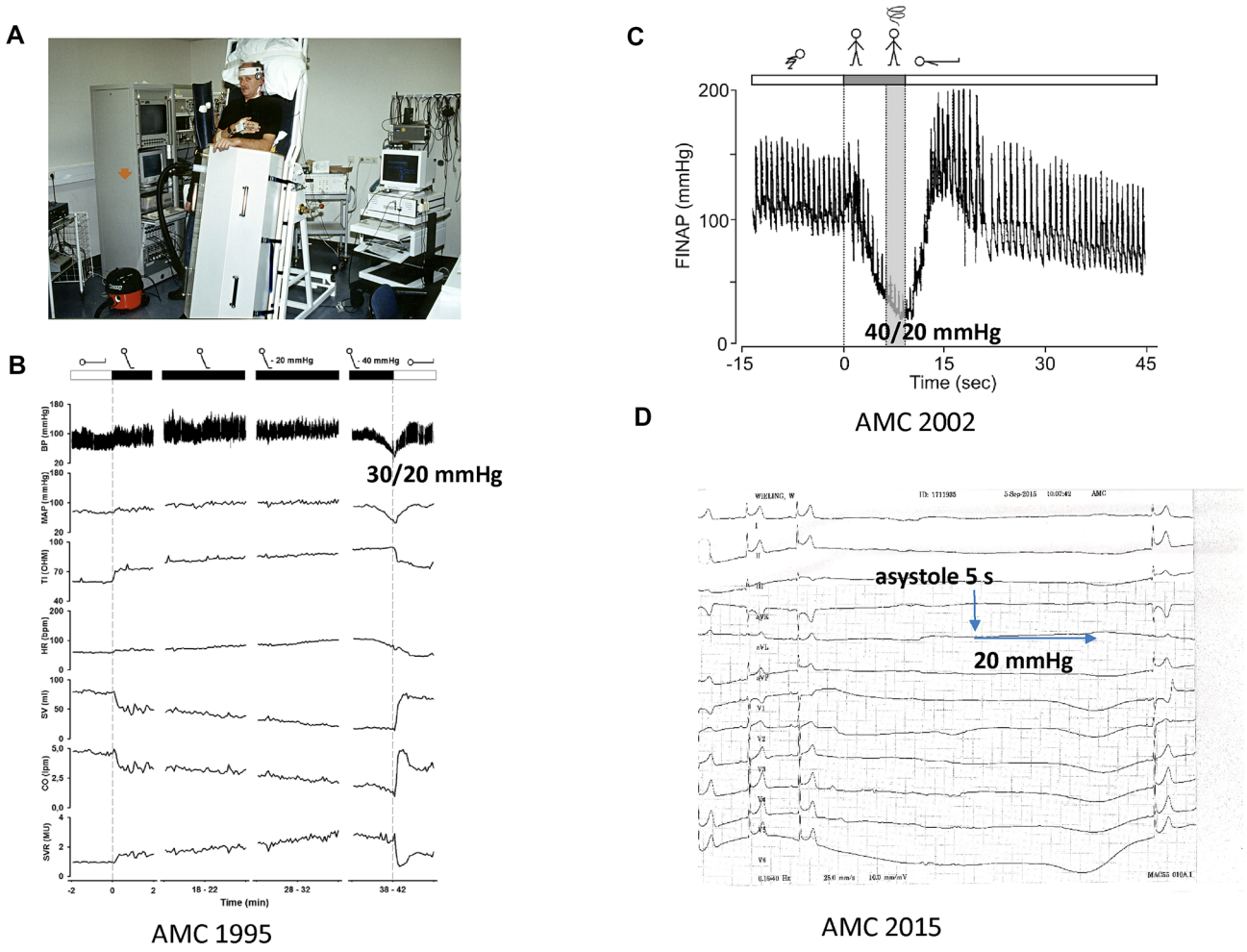

Fig. 1 A syncope doctor's self-induced syncopal episodes. **a** The author (Wouter Wieling) on a tilt-table inside the lower body negative pressure (LBNP) equipment. The arrow indicates a vacuum cleaner. **b** Hemodynamic monitoring during LBNP. At 38–40 min, increasing LBNP to a negative pressure of − 40 mmHg induced a classical vasovagal reaction with decreases in heart rate (HR), cardiac output, and BP. My lowest BP was 30/20 mmHg. I experienced a feeling of warmth followed by lightheadedness but did not faint. After a rapid tilt-down and the discontinuation of the LBNP, there was a recovery of my BP to baseline levels followed by an overshoot with almost immediate disappearance of my lightheadedness. **c** Beat-to-beat blood pressure during my fainting lark. The duration of the loss of consciousness was 2–3 s. My lowest BP was 40/20 mmHg, which rapidly increased with an overshoot upon lying down. **d** My 12-lead ECG showing a period of sinus arrest up to 5–8 s with ST elevations compatible with pericarditis. My systolic blood pressure during asystole was as low as 20 mmHg

acute pericarditis was, however, unlikely since in this condition sinus tachycardia is a consistent finding. During my 5-year follow-up visit, the activation of my pacemaker only occurred during my sleep.

The sensation of lightheadedness that I experienced during (near)syncope was identical during the three provocations. But only with the fainting lark, I experienced seeing black and true syncope. Seeing black due to retinal hypoperfusion is a warning sign of impending loss of consciousness [8]. BP values were not available during the periods with sinus arrest, but it is well known that, when asystole occurs, the initial fall in BP is very steep. BP falls within 5 s to a mean circulatory filling pressure of ~ 10 mmHg [1, 7]. In the standing position, I experienced lightheadedness starting after about 5 s of asystole, but a sinus arrest of 7 s did not induce syncope. These self-observations confirm data from the literature that, after a cardiac arrest, the time interval preceding complete loss of consciousness in the upright posture is 5–8 s [7, 8]. This is known as the cerebral anoxia reserve time [8, 10]. When I was lying down, I hardly experienced symptoms with asystole up to 7 s. This is in agreement with a much longer reported cerebral anoxia reserve time in the supine position (12–15 s) [8]. The explanation is that in the upright position the brain at eye level is located 25–30 cm above the heart. The hydrostatic effect, secondary to gravity, lowers the mean pressure at eye level by about 20 mmHg

[8]. The time course of the hemodynamic events involved in the fainting lark was more abrupt than during spontaneous vasovagal syncope. Syncope occurred only with the fainting lark despite similar low BPs suggesting that the rate of fall in BP influences the occurrence of syncope. The hypocapnia-induced cerebral vasoconstriction by hyperventilation in the fainting lark is likely to contribute importantly since I did not lose consciousness during 5–7 s of asystole (Fig. 1d) known to be accompanied by a steep fall in BP. The potential role of dynamic cerebral autoregulation as a compensatory mechanism for a sudden fall in systemic BP should be considered. There are two reasons why cerebral autoregulation plays only a minor if any role in the pathophysiology of syncope induced by the three provocations. First, cerebral autoregulation is fairly slow. The latency from the start of a sudden drop in BP pressure to the onset of cerebrovascular counter-regulation (i.e. vasodilatation) is about 3–5 s and it takes 5–10 s before the full effect of counter-regulation is reached. Secondly, the BP falls quickly resulted in values below the lower limit of the autoregulatory range of cerebral BP (mean arterial pressure below 70 mmHg), and cerebral autoregulation, even at maximal capacity, cannot compensate [7]. The only remaining role for cerebral autoregulation in provocations like the fainting lark is a faster recovery of cerebral blood flow than of arterial pressure.

My self-induced (presyncope) episodes were minor. Heroic "self-induced" syncope has been reported during World War II by researchers who were interested in G-force-induced blackout in military pilots. Rossen, Kabat, and Anderson self-experimented extensively to design experiments that studied the effects of cerebral arrest by completely stopping the flow of blood to their brain with an inflatable head-cuff applied to the lower third of the neck [6]. The experiments were performed during the early 1940s to elucidate the mechanisms underlying G-force-induced loss of consciousness in military pilots. The neck cuff experiments provided unique data on the sequence of events induced by the arrest of the cerebral blood flow, yet unethical under current standards. Earl Howard Wood (1912–2009), a physician-scientist at the Mayo Clinic Aero Medical Unit, which developed the first civilian human centrifuge in the USA during World War II, is credited with inventing the G-suit and a voluntary exhaling technique (the "M1" breath-holding maneuver) used by military pilots to prevent blackouts. The Mayo Clinic team tested these interventions on themselves in the Mayo Clinic human centrifuge. Earl Wood estimated that, during his several hundred rides in the human centrifuge, he had remained unconscious for a total of at least 15 min, without any obvious sequelae [5].

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest statement.

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