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Short communication

Onset of sweating depends on the type of reflex syncope

Walter Struhal *, Antonija Mišmaš [1], Matthias Kirchmayr, Sigrid Bartl, Andrija Javor, Milan R. Vosko, Gerhard Ransmayr

Autonomic Unit, Department for Neurology and Psychiatry, General Hospital of the City of Linz, Krankenhausstr. 9, A-4020 Linz, Austria

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ABSTRACT

Reflex syncope is classified based on the efferent autonomic system as vasodepressant type, cardioinhibitory type and mixed type. We employed quantitative sweat testing to assess differences in sudomotor sympathetic activity in relation to the type of reflex syncope. In cardioinhibitory type sweating started in 7/9 patients after and in vasodepressor type in 11/12 patients before syncope. In mixed type sweating in 20 patients started before and in 10 after syncope. The onset of sweating correlated significantly with the onset of syncope symptoms. These results possibly reflect different onsets of emotional sweating.

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1. Introduction

The pathophysiology of reflex syncope remains poorly understood. The diagnostic test for reflex syncope is the head up tilt test (HUT) (Moya et al., 2009). Patients with a positive response are classified on whether the syncope is caused by a sudden drop in blood pressure (vasodepressor), a sudden drop in heart rate (cardioinhibitory), or both (mixed) (Brignole et al., 2000). In a protocol combining HUT with active standing, vasodepressor syncopes were dominated by vagal withdrawal, while cardioinhibitory syncopes were dominated by pronounced sympathetic modulation (Holmegard et al., 2012). These findings suggest different disease mechanisms.

Profound sweating is regularly reported by patients suffering reflex syncope (Wieling et al., 2009). Sweat glands are innervated by sudomotor neurons. Sudomotor neurons fire at high and are silent at low ambient temperatures (Janig and Habler, 2003). In addition to thermoregulation, these neurons are activated by emotional (non-thermal) stimuli. Psychological distress is common in patients suffering reflex syncope and might actively influence the chronic relapsing condition of reflex syncope (Gracie et al., 2006).

This is the first study to evaluate quantitative sweat production in reflex syncope patients.

2. Material and methods

All consecutive patients during a period from July 2008 to March 2012 who met the following criteria were retrospectively included: (I) previous history of syncope without evidence of cardiovascular disease (excluded by a cardiologist specialist), (II) no history of sweat disturbance, (III) no clinical signs of polyneuropathy, (IV) no medication with autonomic side effects, (V) positive head up tilt test (syncope or presyncope while performing a tilt table test) with simultaneous quantitative sudomotor recordings, and (VI) at or above the age of 18 years.

Patients were divided into three groups (mixed [type 1], cardioinhibitory [type 2], or vasodepressant [type 3]) (Brignole et al., 2000): *type 1*: heart rate decrease for more than 10% from its peak but not less than 40 beats per minute (bpm) for more than 10 s without asystole ≥3 s, blood pressure decrease precedes heart rate decrease; *type 2*: heart rate decrease below 40 bpm for more than 10 s with or without asystole, or asystole ≥3 s; or *type 3*: blood pressure decrease without heart rate decrease for more than 10% from peak heart rate.

Patients who did not show any increase in sweat production where then excluded from further evaluation. Sweat production was routinely recorded quantitatively (QSWEAT®) from the forearm, a location with both, a well investigated region of decreased peripheral resistance before reflex syncope (Goldstein et al., 2003) and high sudomotor activity. The sweat capsule was positioned on the medial left forearm three fourths of the distance from the ulnar epicondyle to the pisiform bone. Onset of sweating was defined as an increase of sweat production by a minimum

* Corresponding author at: Autonomic Unit, Department for Neurology and Psychiatry, Krankenhausstr. 9, A-4020 Linz, Austria. Tel.: +43 732 7806 73347; fax: +43 732 7806 74 6866.
E-mail addresses: Walter.Struhal@akh.linz.at (W. Struhal), antonija.mismas@gmail.com (A. Mišmaš), Matthias.Kirchmayr@akh.linz.at (M. Kirchmayr), Sigrid.Bartl@akh.linz.at (S. Bartl), Andrija.Javor@akh.linz.at (A. Javor), Milan.Vosko@akh.linz.at (M.R. Vosko), gerhard.ransmayr@akh.linz.at (G. Ransmayr).
[1] Present address: University Hospital Centre Zagreb, Department of Neurology, Neurological Intensive Care Unit, Kispaticeva 12, 10000 Zagreb, Croatia.

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of 20 nl/min compared to baseline. Heart rate, blood pressure and breathing pattern where recorded noninvasively with a self-developed recording system (Fig. 1). The heart rate was recorded by 3-channel ECG, amplified by a biosignal amplifier (g.Bsamp®, g.tec). Blood pressure was recorded using Portapres® (Finapres Medical Systems) and respiration using a piezosensor band around the chest (g.RESPsensor®, g.tec). All biosignals were sampled with 1000 Hz per channel and a resolution of 16 bits using an A/D-converter (NI DAQCard®, National Instruments Inc.) and a recording and analysis software developed employing Labview® (National Instruments Inc.).

Recordings were performed in an acclimatized room kept on a stable controlled temperature of 23 °C.

Autonomic function tests were conducted at the autonomic unit by a well-trained medical research scientist. Protocol comprised 70° HUT for 45 min and a provocative left forearm venous puncture 20 min after HUT onset. During autonomic testing patients were asked to report any uncomfortable or uncommon feelings. These observations were marked within the signal record. In addition the medical research scientist proactively interviewed the patients for any symptoms, if the patient looked pale or blood pressure decreased during tilt table test. We evaluated the onset of symptoms by the first discomfort occurring to the patients. In addition we evaluated the first onset of cardiovascular signs mounting in presyncope/syncope by a) the first systolic blood pressure decrease exceeding 20 mm Hg or b) first heart rate decrease exceeding 10% from heart rate peak. The table was tilted back to prevent syncope wherever possible and HUT regarded positive for presyncope if the patient's systolic blood pressure fell below 70 mm Hg (Brignole et al., 2000).

To exclude any influence from signal artifacts, time series were manually screened before further processing. By these means, ECG artifacts, extrasystoles and Portapres® calibration intervals were excluded from statistical evaluation.

The study was approved by the local Ethics Committee of Upper Austria.

Statistical differences ($p < 0.05$) were calculated employing Mann–Whitney-U-Test, Kruskal–Wallis-Test, and Chi-Squared-Test, significant correlations using Pearson correlation coefficient (PASW® v18.0.0-SPSS Inc.).

3. Results

62 patients were included (age 39 ± 15 years (mean ± SD), 26 male/36 female). Patients were divided into three groups according to their reflex syncope pattern type (Brignole et al., 2000). Due to not sweating during presyncope/syncope 11 patients (18%) had to be excluded (5 mixed 6 vasodepressor reflex syncope). Those 11 patients were significantly older (age 50 ± 12 years, $p = 0.012$). Excluded patients did not significantly differ in any other aspect (Table 1). In these 11 patients an autonomic neuropathy was not known. However, this does not rule out, that those patients might suffer a structural cause of sweat disturbance.

The remaining 51 patients, all with a history of reflex syncope, were included (age 37 ± 15 years, 21 male/30 female): 30 patients with mixed, 9 with cardioinhibitory, and 12 with vasodepressor reflex syncope (Table 1).

3.1. Prodromal phase

There was no significant difference regarding blood pressure and heart rate values in supine position (systolic 126 ± 22 mm Hg, diastolic 65 ± 11 mm Hg, 66 ± 9 bpm) between the three syncope types. The duration between mentioning first symptoms to occurrence of syncope did not vary significantly (types 1: 611 ± 581 s; 2: 792 ± 745 s; 3: 667 ± 574 s). However, the duration from the onset of cardiovascular signs (i.e. blood pressure decrease or heart rate decrease) to presyncope/syncope did vary significantly between types (types 1: 133 ± 131 s; 2: 46 ± 31 s; 3: 139 ± 240 s; $p = 0.007$) as did the duration from the sweat onset to presyncope/syncope (types 1: 406 ± 552 s; 2: 7 ± 140 s; 3: 72 ± 103 s; $p = 0.009$).

The onset of sweating significantly differed (Table 1): in only 2 of 9 patients (22%) of cardioinhibitory type sweat production started before presyncope/syncope (Fig. 1a) compared to 11 of 12 patients (92%) in vasodepressor type (Fig. 1b). In mixed type sweating started in 20 of 30 patients (67%) before presyncope/syncope. The duration between the onset of sweating and presyncope/syncope correlated with total sweat production (Pearson 0.286, $p = 0.042$), the duration between the first symptom to presyncope (Pearson 0.283, $p = 0.044$), but not the duration between first cardiovascular sign to presyncope (Pearson 0.029, $p = 0.839$).

3.2. Presyncope/syncope

There was no significant difference between blood pressure values between the three types during presyncope/syncope, but as a consequence of the diagnostic criteria a highly significant difference in heart rate ($p < 0.001$). There was no significant difference between the total

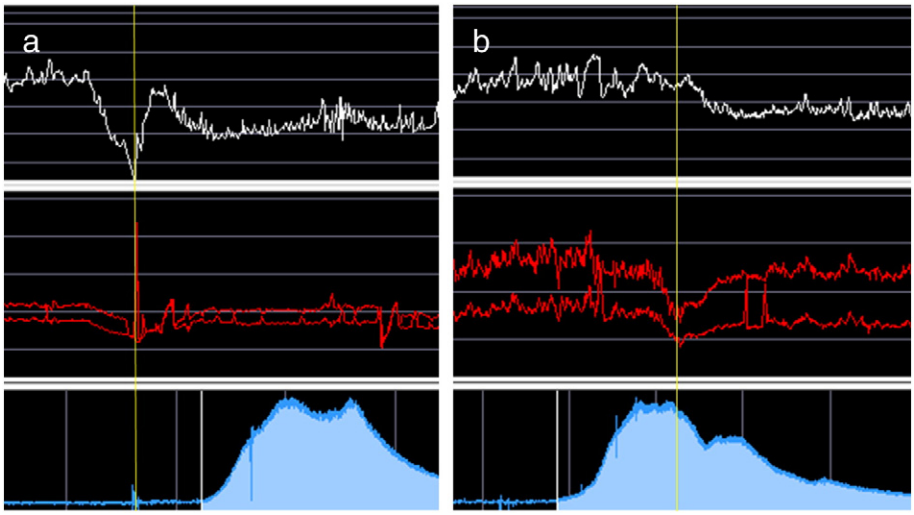

Fig. 1. a) Patient suffering cardioinhibitory type syncope. b) Patient suffering vasodepressor type presyncope. Upper trace: heart rate, middle trace: systolic and diastolic blood pressure, lower trace: quantitative sweat production; yellow line: presyncope/syncope; white line lower trace: sweat onset.

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