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Orthostatic Hypotension: When Volume Depletion Coexists With Polyneuropathy

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Abstract

The heart rate increase (ΔHR) reported to the systolic blood pressure decrease (ΔSBP) during orthostatic hypotension (OH) allows to distinguish neurogenic OH (ΔHR/ΔSBP <0.5) from non-neurogenic OH (ΔHR/ΔSBP >0.49). We report the case of unusual inconsistency of the ΔHR/ΔSBP under changing clinical scenarios. A 72-year-old man was recovering from perforation of the bowel, shock, and acute renal failure. The small bowel was resected with 1.4 meters left. In the followings, severe OH occurred and was attributed to fluid losses through high output ileostomy and polyuria. We assessed whether the ΔHR/ΔSBP can provide indication to the patient's volume status. To this aim 30 bedside orthostatic tests were performed during six-months of hospitalization: OH occurred on 23 tests. In 7 instances the ΔHR/ΔSBP was >0.49 (consistent with volume depletion), and in 16 instances the ΔHR/ΔSBP was <0.5 (consistent with efferent baroreflex failure). Thereupon, critical illness polyneuropathy was diagnosed. Volume depletion and polyneuropathy were both involved in this patient's OH. Either is known to determine a predictable and different pattern of HR-response under hypotension. However, in this patient the HR response to hypotension was unpredictable. We conclude that in complex clinical settings the ΔHR/ΔSBP might not keep with the rule, and that the ΔHR/ΔSBP cannot help in diagnosing a person's volume status.

Keywords: Seizure-tolerant and seizure-sensitive rats • Passive avoidance response • Fluoxetine • Serotonin • Dopamine• Noradrenaline

Introduction

Orthostatic hypotension (OH) has been formally defined by expert consensus as a fall in systolic blood pressure (SBP) of at least 20 mmHg and or diastolic blood pressure of at least 10 mmHg within 3 minutes of standing [1]. When a healthy individual stands, 10% to 15% of the blood is pooled in the lower extremities and splanchnic veins. This causes a reduction in venous return, a decrease in stroke volume, cardiac output and finally in blood pressure. The fall in blood pressure activates baroreceptors with subsequent reflex increase in sympathetic outflow and parasympathetic inhibition, leading to vasoconstriction, increased heart rate and contractility. These compensatory responses stabilize the blood pressure within seconds [2,3]. In the healthy, volume replete person, transition from supine to standing position may cause but a slight fall in SBP of less than 10 mmHg, a slight increase in DBP of approximately 2.5 mmHg, and a modest increase in heart rate (HR) by 5-12 beats per minute. If the compensatory responses fail OH may occur upon assuming the upright posture [4].

Numerous factors may affect the blood pressure homeostasis during a change in posture, including functioning of the autonomic nervous system, the intravascular volume, the heart and blood vessels' anatomic and functional integrity, duration of the upright posture, the postprandial state, and the ambient temperature. Common causes of OH in older persons are hypovolemia (dehydration, postobstructive polyuria, bleeding), autonomic neuropathies whether primary (primary autonomic failure, multiple system atrophy, Parkinson's disease) or secondary (in diabetes mellitus, chronic renal failure, chronic liver disease, alcohol-induced, vitamin B12 deficiency, Guillain-Barré syndrome, paraneoplastic, critical illness), medications (diuretics, nitrates, antihypertensives, tricyclic antidepressants), and blood pooling in large varicose veins. Old age is associated with an increased risk of OH [5,6]. Postprandial OH (postprandial hypotension) is common in elderly subjects [7]. Postprandial OH occurs frequently in survivors of critical illness [8]. Although the pathophysiology of postprandial OH is poorly understood, impairments in sympathetic and baroreflex function, release of vasodilatory peptides, the rate of small intestinal nutrient delivery, gastric distension, and splanchnic blood pooling, are involved [9].

Recording the HR is integral to testing for OH and is indispensable for the differentiation between certain OH subtypes. In a healthy subject, the HR will increase by 5 to 12 beats per minute (bpm) when rising upright from recumbence [10]. Failure of the HR to rise when standing up in tandem with the decrease in blood pressure indicates sympathetic autonomic insufficiency that is neurogenic OH [11]. There are exceptions to this rule: healthy older individuals may display a blunted HR response on standing up due to the downregulation of baroreceptors [12]. The HR may be slowed by medication. On the other hand, there may be a paradoxical increase in HR in patients with autonomic failure, presumably due to parasympathetic withdrawal [13]. An exaggerated increase in HR in the upright position may be indicative of a contracted intravascular volume [14].

A simple, bedside test allows distinguishing neurogenic from non-neurogenic causes of OH, based on the ratio of the increase in heart rate to the decrease in SBP. In the followings, the label △HR/△SBP stands for the ratio of the increase in heart rate (beats per minute) to the decrease in SBP (millimeters of mercury) under postural challenge at the bedside. The heart rate response to hypotension is pronounced in patients with non-neurogenic orthostatic hypotension but is blunted in those with efferent baroreflex failure. A recent study found that △HR/△SBP <0.5 during active standing indicates efferent baroreflex failure and provides a sensitive (91.3%) and specific (88.4%) cutoff value [15,16].

The aim of the present case study was to observe the clinical correlates of △HR/△SBP in a patient under changing hydration status. We tested whether the △HR/△SBP can assist in the assessment of the patient's volume status and serve to guide fluid replacement.

The bedside supine-to-standing orthostatic test was used in this study. It is standardized and validated, though its reproducibility is far from optimal. However, no better test exists [17]. After 15 minutes of silent rest in the supine position the patient's blood pressure and HR are recorded five times at one-minute intervals. Next, the patient stands up and measurements are done with

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the patent's cuffed arm supported at heart level, at one-minute intervals for another five minutes. The patient is asked to report dizziness, faintness, or light-headedness. The procedure is aborted for safety reasons if the blood pressure drops precipitously or symptoms of presyncope evolve. When the patient is unable stand a supine-to-sitting orthostatic test is performed built on a similar protocol, with the patient moving from the supine to a sitting position at the edge of his bed. Orthostatic hypotension (OH) is diagnosed as a fall in SBP of ≥20 mmHg and/or diastolic blood pressure (DBP) of ≥10 mmHg within 3 minutes of standing [1]. Postprandial OH (postprandial hypotension) is diagnosed as a decrease in SBP of at least 20 mmHg within two hours after a meal [18]. The △HR/△SBP ratio [15] is computed solely under OH. Two time points are selected for analysis: A. is the time when the SBP decreases by at least 20 mmHg, and B. is calculated when SBP is lowest (SBP nadir). The △HR refers to the change from baseline to time points A. and B. Volume depletion is diagnosed based on patient history, physical examination, fluid balance, a battery of laboratory tests (serum sodium, osmolality, BUN:serum creatinine ratio, hematocrit), along with an expert clinician's impression of dehydration, since no single measure is accurate to express the volume depleted state [19,20].

Case History

A 72-year-old man was admitted for post-acute care, having recently recovered from complications of an elective surgery. He was a longtime hypertensive with the blood pressure fairly controlled on valsartan treatment. Eight years ago, he was diagnosed with carcinoma of the rectum stage 3B, underwent anterior resection with colostomy and regional radiation therapy. The fecal output through the stoma was less than 1000 mL/day. There was no recurrence of the neoplasia. The patient was physically fit until recent abdominal surgery for the closure of ileostomy and repair of peristomal hernia. Under surgery perforation of the small bowel occurred, complicated with peritonitis. The small bowel was resected with 1.4 meter left. The right colon was resected. An ileostomy was formed. Shock, acute renal failure, and liver failure followed. After the patient's condition stabilized he was transferred to our institution for rehabilitation. The medications at that time were propafenone, apixaban, bisoprolol (1.25 mg), esomeprazole, dibasic sodium phosphate, and peptamine. The ileostomy output was about 3000 mL/day, the diuresis 2000-2500 mL. The patient's vital signs were normal, inclusive the supine blood pressure. However, when trying to sit he fainted, the BP dropping within a minute as low as SBP 57 mmHg. The hematocrit was 34%, serum sodium 130 mEqu/L, potassium 5.2 mEqu/L, BUN 53 mg/dL, creatinine 2.5 mg/dL, eGFR 24 mL/min/1.73 m^2, albumin 3.8 g/dL, urinary sodium 142 mMol/L (i.e., 22 g salt under diuresis of 3 liter). The morning total cortisol was 371 nmol/l and increased to 817 nmol/l after administration of corticotropin, excluding adrenal insufficiency. Large volumes of intravenous fluids were administered and electrolytes to correct deficiencies. The diet was tailored to match osmotic diarrhea and malabsorption. Loperamide 16 mg/day was administered to reduce the intestinal transit time and improve absorption. With improved volume status and renal function, sitting and standing became possible (Figure 1A). When the patient's condition further improved, the eGFR returned to 90 mL/min/1.73 m^2 and the ileostomy output decreased to 1500 mL, we tried to wean the patient from parenteral fluids. This failed, as OH returned (Figure 1B) associated with deterioration of renal functions. The context suggested hypovolemic OH since the change in fluid management preceded to the recurrence of severe OH and aggravation of renal function. Subsequently, strict control of the fluid balance by appropriate fluid intake orally completed with intravenous isotonic saline brought about clinical improvement (Table 1).

Yet, OH persisted. Bisoprolol was discontinued. Thereafter, orthostatic tests were performed once or twice weekly. In total, 30 orthostatic tests were performed during the six months of the patients stay in our ward. The procedures were adapted to the patient's ability to sit and stand. There were 16 supine-to-standing tests. The remainder were supine-to-sitting or supine-to-sitting-to-standing tests. Twenty-three tests provoked OH (77%). In comparing the △HR/△SBP on first drop of SBP by ≥20 mmHg with △HR/△SBP during SBP nadir the difference was not statistically significant (p = 0.39). So, we choose

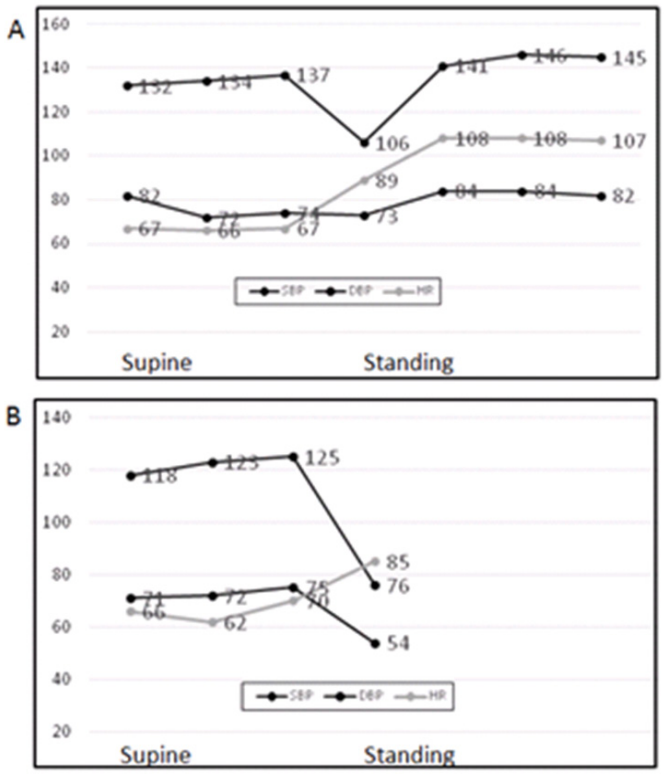

Figure 1. (A). Bedside postural test, January the 12th, with the patient wearing foot-to-groin elastic bandages. Measurement at one-minute intervals. The transient OH during the first minute of standing was asymptomatic. Remarkable was the substantial increase in the HR (△HR/△SBP = 0.71). (B). Recurrence of OH March 1s (△HR/△SBP = 0.30).

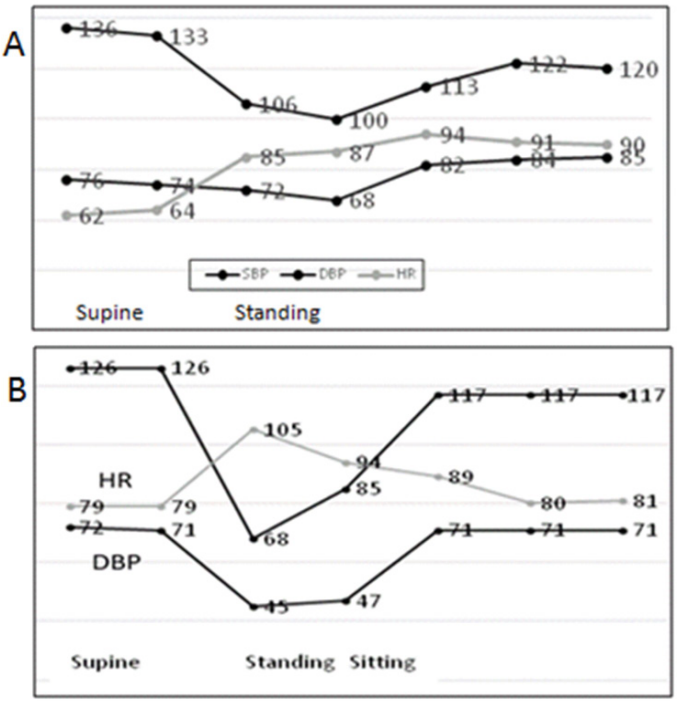

Figure 2. Supine-to-standing postural tests. (A). May 26th, thirty minutes after breakfast the patient developed asymptomatic OH. The △HR/△SBP was 0.78 on first upright measurement and 0.70 during nadir SBP. (B). June 4th, forty minutes after breakfast. Severe symptomatic OH occurred. The △HR/△SBP was 0.44 on first upright measurement and 0.36 on subsequent measurement with the patient seated.

the first drop of SBP ≥20 mmHg to compute the △HR/△SBP. In 7 instances the △HR/△SBP was >0.49 (consistent with volume depletion or other, non-

Table 1. Fluid balance, body weight, walking tolerance, and selected laboratory data through March - July. PPH signifies postprandial hypotension, BW: body weight, Walk toler: walking tolerance, sOsm: serum osmolality (normal range 275-295 mOsm/L).

Date	Oral mL	Saline mL	Feces mL	Urine mL	Htc%	s No mMol/L s	sk m Mol/L	sOsm mMol/L	sMg mg/dl	u No mMol/L	uOsm mMol/L	eGFR mL	BWkg	Walk toler
1.3	1700	0	900	1700	39	142	5.6	315				71		-
12.4	2700	500	1200	2200	46.8	137	4.9		1.9			65		+
20.4	2950	500	950	2400	45.4	141	4.9	297	1.8	59	332	67	63.5	+
28.4	3000	500	900	1700	44.9	143	4.8	290	1.7			72	63.7	++
29.4	2750	500	1250	1500										++ PPH*
26.5	2800	1000 0.9%	1200	1800	46.7	140	4.2	295	1.8			85	65.3	+++
2 .6	2700	1000 0.45%	1300	1800										+++
4.6	2800	1000 0.45%	1350	2200									64.5	+++
8.6	2900	1000 0.45%	1200	1800	47.7	137	4.7	297	1.8	85.7		74	66.3	+++
2 .7	3000	0	1100	1850	46.2	139	4.8	293	2	56	217	68		+++
8.7	3300	0	1200	2000										+++
14.7	3350	0	1200	1800	47.3	141	5	297	2.1			65	67.7	+++

Table 2. The △HR/△SBP ratios on supine-to-standing postural test, the patient being clinically euvolemic. Four of eight examinations in the fasting state produced △HR/△SBP >0.5, suggesting hypovolemia.

Date	Fasting state ΔHR/ΔSBP	Postprandial state ΔHR/ΔSBP
April 30	0.71	–
April 30	–	0.85
May 25	–	0.37
May 26	0.57	–
June 1	0.35	–
June 2	0.37	–
June 4	–	0.44
June 7	0.55	–
June 11	0.55	–
June 11	–	0.73
June 24	0.41	–
June 26	–	1.26
July 5	0.42	–
July 5		1

neurogenic OH), and in 16 instances the △HR/△SBP was < 0.5 (consistent with efferent baroreflex failure, i.e., neurogenic OH).

During the final 10 weeks of hospitalization the fluid input and output were balanced, and the patient appeared to be euvolemic. Results of △HR/△SBP are of particular interest during this period (Table 2). The patient's condition had markedly improved. He had gained weight without developing edema, was sitting on a chair most time of the day and was walking without assistance 50-100 meters three times daily. The fecal output through ileostomy had decreased to 1100-1300 mL (versus 3000 on admission). The diuresis remained close to 2000 mL. The hematocrit, serum Na and the eGFR were within the normal range. Currently the patient's medications were loperamide Tab 16 mg/day, magnesium citrate Tab 200 mg, calcium carbonate Tab 1800 mg, vitamin D3 Tb 2000 U, and famotidine Tab 40 mg. However, OH remained being usually asymptomatic. In the postprandial state, alike under fasting, the △HR/△SBP varied: four out of six tests produced △HR/△SBP >0.5, consistent with volume depletion, other results were consistent with efferent baroreflex failure (Table 2 and Figure 2).

The possibility that the patient had acquired autonomic failure was considered. Clinical hints to dysautonomia were lacking: there was neither diabetes mellitus, amyloidosis, Guillain-Barre syndrome, porphyria, Parkinson disease, multiple sclerosis, multiple system atrophy, nor familiar dysautonomia. The pupillary light reactions, skin temperature and color, core temperature, and sweating were normal. There was no muscle weakness and there was no sensory loss. Vibration sense, proprioception, and the patellar reflexes were normal. Achilles reflexes were abolished. Voiding of the bladder was normal. Impotence had occurred eight years earlier after pelvic surgery and radiation therapy. Under emotional challenge by arithmetic tests or intimate questioning the responses differed. Twice there was no increase in the HR. On a third attempt, later in the course of hospitalization, the HR increased from 62 to 73 bpm and SBP from 163 to 180 mmHg. Critical illness polyneuropathy [21] was regarded a likely condition, triggered by shock and sepsis six month before. Now, electrophysiological testing revealed distal axonal polyneuropathy with reduced compound muscle action potential, fibrillation potentials, and decreased motor unit action potentials. Large and small fibers were affected.

Hence, neurogenic and non-neurogenic causes were involved causing OH in this patient. At the time of writing, three months after being discharged, he was free of symptoms under transfers, sitting and walking. The fluid balance remained strictly controlled. Medications were the same. The CBC and blood chemistry remained normal. Additional orthostatic tests were not performed. We considered the patient's condition to be satisfactory according to the principle that a fair result in treating OH is avoidance of orthostatic symptoms not necessarily forestalling OH [22]. The treatment goal in OH should be to improve symptoms and functional status, and not to target arbitrary blood pressure values [23].

Discussion

During lengthy observation and on frequent orthostatic tests the patient's △HR/△SBP varied, often disobeying the rule [15,16]. So, under

volume depletion the expected △HR/△SBP >0.5 was inconsistently present. After correction of volume depletion, OH might be caused by critical illness polyneuropathy. Yet, the expected △HR/△SBP <0.5 of neurogenic OH was also inconsistently present.

Critical illness polyneuropathy affects 25%–45% of subjects with critical illness and up to 100% of those with multiorgan failure [24]. Established risk factors include duration of sepsis, number of organ systems involved and immobility. According to the prevailing hypothesis, microcirculatory damage impairs peripheral nerve and muscle perfusion, and with reduced oxygen supply these neurons are unable to generate action potentials. The diagnosis of critical illness polyneuropathy is based on a history of critical illness, evidence of limb or respiratory weakness, and results of electrodiagnostic testing consistent with axonal motor and sensory polyneuropathy that is not explained by another cause [25]. Recovery from critical illness polyneuropathy is late with over one-third of patients having substantial functional limitations at one year [26]. Critical illness polyneuorpathy is a recognised cause of OH [27,28]; during OH the △HR/△SBP is expected to be <0.5 [15,16]. In this patient, however, △HR/△SBP <0.5 was inconsistently present.

In the postprandial state, postural challenge elicited in the patient variable responses: e.g. △HR/△SBP 0.78 which is consistent with hypovolemia, and △HR/△SBP 0.44, 0.36 which is consistent with efferent baroreflex failure (Figure 2). Since the patient was currently in a stable clinical state it was sensible to expect a constant response pattern. This was not the case. Moreover, it appeared to be contra intuitive that an increase of the HR by 21 bpm should be attributed to hypovolemia because the △HR/△SBP was 0.78 (Figure 2A), while an increase of the HR by 26 bpm should be attributed to autonomic failure because the △HR/△SBP was 0.44 – 0.36 (Figure 2B). Therefore, we questioned whether △HR/△SBP <0.5 is an unconditional indication of the neurogenic mechanism in OH, or there might exist exceptions to the rule? Indeed, older individuals may display a blunted HR response due to downregulation of baroreceptors related to aging [12]. On the other hand, there may be a paradoxical increase in HR in patients with autonomic failure, presumably due to parasympathetic withdrawal [13]. Does an increase of the HR by >20 bpm during OH always stands for non-neurogenic OH, independently on the presence of autonomic failure? Or should a patient's clinical context shape our interpretation of the △HR/△SBP ratio? [14-26]

Conclusion

Two disorders in this patient, volume depletion and polyneuropathy, are known to influence the HR response to hypotension according to a predicable rule and in opposite directions. But the HR response to hypotension occurred erratically, without obeying to the rule. A large body of evidence indicates that the △HR/△SBP ratio <0.5 is diagnostic of baroreflex failure, providing a sensitive and specific cutoff value. The proposito might represent an exception to the rule. Also, △HR/△SBP >0.5 was not a reliable indicator of volume depletion. No single clinical sign or laboratory measure is accurate to express the volume depleted status and, in this patient, the △HR/△SBP had no added value to this aim. We learned that interpretation of the △HR/△SBP ratio in complicated clinical settings needs prudence.

Conflict of Interest

There is none concerning this work.

Funding Statement

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