

# Orthostatic Hypotension in a Complex Clinical Setting Use of the Heart Rate Increase/Systolic Blood Pressure Decrease Ratio as a Diagnostic Aid

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## Abstract

A simple bedside test to distinguish neurogenic from non-neurogenic causes of orthostatic hypotension (OH) is the ratio of heart rate increase during OH to the simultaneous decrease in systolic blood pressure (SBP), i.e. the  $\Delta HR/\Delta SBP$  ratio. In a patient suffering from persistent OH we monitored the  $\Delta HR/\Delta SBP$  ratio to aid with a targeted therapy. During a six-week period four pairs of postural tests were performed, one in the fasting and the other in the postprandial state. Inconsistency of the  $\Delta HR/\Delta SBP$  ratio under the patient's apparently stable clinical condition was confusing. So, the  $\Delta HR/\Delta SBP$  ratio did help in taking therapeutic decisions.

**Keywords:** orthostatic hypotension; systolic blood pressure; Diagnostic Aid

## Introduction

A simple bedside test to distinguish neurogenic from non-neurogenic causes of orthostatic hypotension (OH) is the ratio of heart rate increase to the simultaneous decrease in systolic blood pressure during OH ( $\Delta HR/\Delta SBP$  ratio) [1]. Since the heart rate response to hypotension is pronounced in patients with non-neurogenic OH but is blunted in those with efferent baroreflex failure, a  $\Delta HR/\Delta SBP$  ratio  $< 0.5$  indicates baroreflex failure and is considered to provide a sensitive and specific cutoff value during active standing [2]. In a patient who suffered from persistent OH we sought help for therapeutic decisions by repeatedly observing the  $\Delta HR/\Delta SBP$  ratio.

## Case history

A 72-year-old man was admitted for post-acute care, having recovered from complications of elective surgery. He was a longtime hypertensive with the blood pressure (BP) fairly controlled on valsartan treatment. Eight years ago, he was diagnosed with carcinoma of the rectum and underwent anterior resection with colostomy, followed by radiation therapy. There was no recurrence of carcinoma. He was physically and mentally fit and independent in daily activities, until he recently underwent surgery for closure of the colostomy and repair of peristomal hernia. Surgery was complicated by perforation of the small bowel, peritonitis, shock, acute renal failure, and liver failure. A large segment of the small bowel needed to be resected with 1.4 meters left. An ileostomy was formed. After the patient's condition stabilized, he was

transferred to our institution for rehabilitation. The ileostomy output was roughly 3000 ml, the diuresis 2000–2500 ml. The patient was alert and conscious. The vital signs were normal, inclusive the body temperature, supine BP, heart rate (HR), and oxygen saturation. However, when trying to sit the patient fainted repeatedly, the BP dropping within a minute as low as SBP 57 mmHg. Large volumes of intravenous fluids were administered, and electrolytes as needed 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. When the patient's condition further improved, he could be weaned from parenteral fluids. He had a good appetite, was sitting on a chair most of the day, was walking without assistance 50 meters three times a day. He had gained weight without developing edema. The fecal output through ileostomy 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. However, asymptomatic OH remained. 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. The fluid balance was supervised daily. Postural tests were done weakly, along with monitoring laboratory markers of dehydration (hematocrit, serum Na, serum osmolality, eGFR). Since hypovolemia and deconditioning had resolved but OH persisted we considered that critical illness neuropathy might be implicated in the causation of OH [3, 4]. However, there was no muscle weakness, the deep tendon reflexes were normal, and there was no sensory loss. The patient postponed electrophysiologic testing in the face of the corona epidemic.

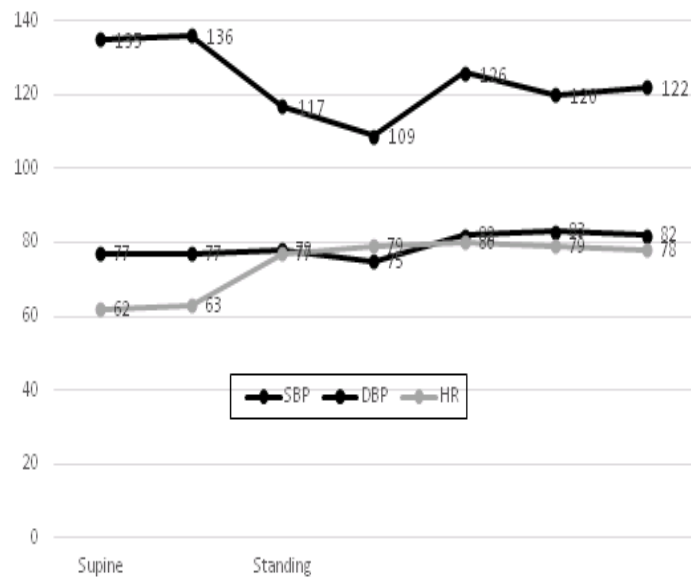
**Methods**

The supine-to-standing postural test is standardized and validated, though, reproducibility is far from optimal; no better test exists [5]. According to the protocol the BP and HR are recorded after 15 minutes of rest in the supine position, 5 times, at one-minute intervals. Next, the patient stands up and measurements are taken with the cuffed arm supported at heart level at one-minute intervals for another 5 minutes. The procedure is aborted for safety reasons if the BP drops precipitously or symptoms of presyncope evolve. We measured the patient's BP on his right arm at heart level, using a Spot Vital SignsR validated automatic oscillometric device (Welch Allyn Inc. Corporate, NY 13153-0220 USA). The HR was recorded by pulse oximetry on the other arm. At awakening the patient was instructed to drink 500 mL water and apply he fitted foot-to-groin compression garments. First at 8 a.m. in the fasting state a supine-to-standing postural test was performed. About 60 minutes later, after the patient had eaten breakfast (equivalent to approximately 900 kcal), the supine to standing postural test was repeated. OH was diagnosed as a fall in systolic BP of  $\geq 20$  mmHg and/or diastolic BP of  $\geq 10$  mmHg within 3 minutes of standing [6].

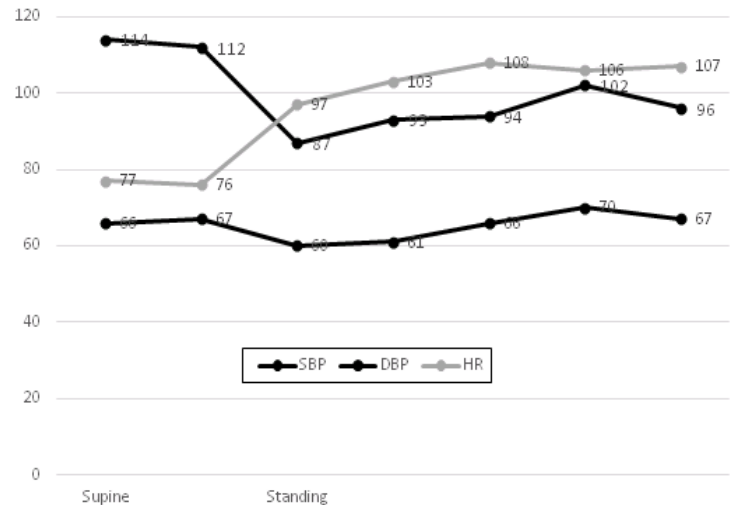
Postprandial hypotension (PPH) was diagnosed as a decrease in SBP of at least 20 mmHg within two hours after a meal (7).  $\Delta HR/\Delta SBP$  ratio during OH was computed related to SBP nadir. The  $\Delta HR/\Delta SBP$  is the ratio of the increase in heart rate (beats per minute) to the decrease in SBP (millimeters of mercury).  $\Delta HR/\Delta SBP$  ratio  $< 0.5$  indicates neurogenic OH, distinguishing between neurogenic from non-neurogenic OH [1, 2].

**Results**

We analyzed paired postural tests completed through June – July when the patient was clinically stable, the fluid input and output were balanced, the body weight was stable, and laboratory markers showed no evidence of dehydration. In all instances, OH occurred within the first two minutes of standing, was asymptomatic, and improved subsequently, as illustrated in Figures 1 and 2.



**Figure 1.** Postural test at 8.15 a.m. in the fasting state. OH was mild and transient. The  $\Delta HR/\Delta SBP$  was 0.59.



**Figure 2.** Postural test at 9.30 a.m., after breakfast, next to the test illustrated in Figure 1. The supine BP was lower and the HR higher than in the fasting state. The BP decline when standing was deeper. The patient remained free of symptoms. The  $\Delta HR/\Delta SBP$  was 0.84.

Four pairs of postural tests were available for analysis; the  $\Delta HR/\Delta SBP$  ratios are shown in Table 1. With reference to the  $\Delta HR/\Delta SBP < 0.5$  threshold, indicating neurogenic OH, there was inconsistency of the results both in the fasting and in the postprandial state.

$\Delta HR/\Delta SBP$ Fasting	$\Delta HR/\Delta SBP$ Postprandial
0.57	0.37
0.71	0.85
0.42	1
0.59	0.84

**Table 1.**  $\Delta HR/\Delta SBP$  ratios under OH in the fasting and subsequent postprandial state

**Discussion**

Postural BP homeostasis depends on the normal functioning of the autonomic nervous system, euvoemia, anatomic and functional integrity of the heart and blood vessels, the postprandial state, and the ambient temperature. When healthy individuals stand 10% to 15% of the blood is pooled in the legs and lower abdomen, thereby diminishing the venous return, with ensuing decline of cardiac output and BP. An adequate homeostatic response to the decrease in BP occurs by activation of baroreceptors, reflex sympathetic stimulation that prompts vasoconstriction, tachycardia, and an enhanced cardiac contractility. When the homeostatic mechanisms are normally functioning, there may be but a slight fall in the SBP as a person stands up, associated with a slight rise in diastolic BP and a mild increase in HR. Failure of one or the other compensatory mechanism may cause OH, i.e. a disproportionate fall in BP occurring early after standing up [8]. Common causes of OH are medications (diuretics, nitrates, antihypertensives, tricyclic antidepressants), hypovolemia (dehydration, post obstructive polyuria, bleeding either overt or occult, blood pooling in large varicose veins), and autonomic neuropathies either 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-Barre syndrome, paraneoplastic, etc) [9,10]. In many instances, the cause of OH is obvious. In other instances, several mechanisms may be involved, and identifying the major mechanism responsible for OH may be challenging. An aid in such situation is provided by the HR response under postural challenge: it is blunted in the presence of baroreflex failure contrasting with the pronounced acceleration of the HR in non-neurogenic OH. The  $\Delta\text{HR}/\Delta\text{SBP}$  has been reported to distinguish accurately neurogenic from non-neurogenic OH [2]. In the proposito, six out of eight tests produced  $\Delta\text{HR}/\Delta\text{SBP} > 0.5$ , that is consistent with hypovolemia. Yet, the analysis was limited to a period when all clinical and laboratory parameters were inconsistent with dehydration [11]. Also, limited to the time of the study, OH could not be attributed to additional conditions displaying a  $\Delta\text{HR}/\Delta\text{SBP} > 0.5$ . Indeed, the patient was not taking medications known to cause OH; adrenal insufficiency has been excluded by normal values of basal and stimulated cortisol; the patient had no varicose veins; there was no evidence of a neoplasia on recent comprehensive investigation. In the proposito, two tests showed a blunted HR response ( $\Delta\text{HR}/\Delta\text{SBP} < 0.5$ ), contrasting with the pronounced HR acceleration in six instances. Inconsistency of the HR response to hypotension was confusing and did not lend support to a targeted therapy.

Other questions arose while caring for the patient, only to be listed here. First, was critical illness neuropathy part of the problem, contributing to OH? [12] Second, was postprandial splanchnic blood pooling affected by the extensive resection of small and large bowel? [13]. these are important issues but are marginal to the aim of the present study that was to substantiate the role of  $\Delta\text{HR}/\Delta\text{SBP}$  for evaluation of OH in a complex clinical situation. Inconsistency of the HR response to hypotension did not support a targeted therapy based on the  $\Delta\text{HR}/\Delta\text{SBP}$  ratio in this patient's complex clinical situation. For answering the query there is need for more studies and the covering a wide spectrum of clinical settings.

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