

## Orthostatic Hypotension

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### About the Authors

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### Case Report

An 83-year-old man was admitted with a brief episode of syncope, witnessed by a friend, as he was attempting to stand from a seated position. The gentleman lived alone and was highly independent and functional. Ten weeks prior to admission, he had fallen on a patch of ice, injuring his head. A moderately large right subdural hematoma had been identified on a computed tomography scan, but the patient had declined surgical treatment. One week prior to the current admission, the patient had suffered an outbreak of shingles in the right upper face (ophthalmic branch of trigeminal nerve), and he had been treated with oral famciclovir.

His past medical history was significant for hypertension, bilateral cataracts, and osteoarthritis. Four years previously, he had been treated for prostate cancer (T1c, Gleason score 8, prostate-specific antigen 5.9) with external beam radiation therapy and adjuvant hormonal therapy. His medications on admission included lorazepam, pantoprazole, famciclovir, amlodipine, and valsartan-hydrochlorothiazide. The valsartan-hydrochlorothiazide was held on admission due to hypotension and hyponatremia.

The patient was in his usual state of health when he attempted to stand up from a chair the day of admission. Additional history from the patient's friend indicated that the room was quite hot. As he attempted to stand, the patient briefly lost consciousness, falling back in his chair. The patient had no history of excess alcohol use. Physical examination, including a full neurological examination, was unremarkable, except for the resolving zoster rash. A syncope workup was initiated. Blood tests showed an elevated level of creatinine (172 mmol/L) and hyponatremia (123 mmol/L), and these gradually normalized with rehydration. Blood glucose and complete blood count were normal. His electrocardiogram (EKG) and troponin values were normal. An electroencephalogram was

After consultation with neurosurgery, it was decided that there was no indication for surgical intervention.

The patient was noted to have a persistently elevated white blood cell count ( $17 \times 10^9$  cells/L) with a predominance of lymphocytes. This high white count had been present during the patient's hospital visit 2 months previously. He was not febrile, and splenomegaly was not identified on an ultrasound scan. On review of a blood smear, it was felt that he had chronic lymphocytic leukemia (stage 0) that required no specific therapy at the time.

While in hospital, he did have a significant orthostatic drop in blood pressure (Table 1). He became dizzy within 15 minutes of standing, and, despite hypotension, he failed to mount a compensatory tachycardia. Amlodipine was discontinued. Thyroid function tests and a 24-hour urinary cortisol excretion were both normal. Neurological illnesses such as Parkinson's disease, cerebellar dysfunction, and multiple system atrophy were excluded. The patient appeared to have isolated sympathetic failure as he denied constipation, urinary retention, loss of ability to sweat, or impotence (parasympathetic symptoms common to pure autonomic failure).

The patient was referred for tilt-table testing. This brought out significant orthostatic hypotension without compensatory tachycardia, both at the 80-degree head-up tilt and subsequently at 30 and 45 degrees. The QSART – quantitative sudomotor axon reflex test – was absent from all three sites in the lower extremities but present at the wrist. These results confirmed that the patient had diffuse sympathetic autonomic failure.

The patient was prescribed leg compression stockings, but these proved ineffective in relieving his symptoms. On day 15 of his admission, he was started on fludrocortisone therapy, in dosages of up to 0.3 mg/d. On days 19 and 20, the patient still had a postural drop of 20 mm Hg systolic, without compensatory tachycardia. However, he was no longer symptomatic and could stand and walk for long periods without difficulty. He was discharged from hospital.

**Table 1. Orthostatic Blood Pressure Change, Day 12**

Time (min)	Position	Blood Pressure (mm Hg)
0	Supine	176/70
3	Sitting	160/78
8	Standing	150/66
15	Standing	120/50
17	Standing	108/48

unremarkable. Telemetry monitoring did not uncover a pathological arrhythmia. An echocardiogram was normal. Magnetic resonance imaging of the brain showed a decrease in the size of the right subdural hematoma.

### Discussion

Two case reports in the literature have documented orthostatic hypotension following intracerebral hemorrhage.<sup>1,2</sup> In both cases, the patients suffered an acute hemorrhagic stroke, resulting in parenchymal brain hemorrhages. In neither case could the etiology of the orthostatic hypotension be found; however, the authors theorized that the hemorrhage could have been a precipitating factor, by affecting the regulation of the autonomic nervous system. We initially believed that this may have occurred in our patient. In the two previous case reports, both

patients were managed with prolonged physiotherapy and subsequently recovered.

Our case illustrates the typical approach to postural hypotension in the adult. First, we rehydrated the patient and held his antihypertensive medications. His initial hyponatremia was thought to be due to a syndrome of inappropriate antidiuretic hormone (SIADH), secondary to his subdural hematoma. We then proceeded to rule out cardiac abnormalities by obtaining an EKG, serial troponins, telemetry, and an echocardiogram. Finally, we ruled out thyroid and adrenal dysfunction. Neurological causes were then considered, given that the patient lacked the normal compensatory tachycardic response to postural hypotension. Features of Parkinson's disease and cerebellar dysfunction that would suggest Shy-Drager syndrome were absent. Lastly, pure autonomic failure was considered, but urinary retention, constipation, failure to sweat, and impotence were not present.

At this point, the patient was referred for specific autonomic function studies. Tilt-table testing and QSART confirmed he had significant sympathetic dysfunction. Did the subdural hematoma have a role to play in this illness? Given that the autonomic dysfunction was no doubt longstanding, why would the patient maintain cerebral autoregulation until this point? It is well known that increases in intracranial pressure (e.g., caused by acute SDH) can lead to overactivity of the sympathetic nervous system and underactivity of the parasympathetic system, resulting in transient hypertension. As the sympathetic nervous system is the critical effector mechanism for maintaining cerebral perfusion in orthostatic changes, we initially thought it possible that the increased demands on this system caused by the SDH could have led to further dysfunction – and worsened autonomic failure.

After consultation with a specialist in autonomic dysfunction (R. S.), we realized that our hypothesis was untenable for several reasons. Firstly, patients with autonomic failure typically remain asymptomatic for long periods of time, either completely denying symptoms or exhibiting vague complaints such as neck pain upon standing, leg weakness, or fatigue. Furthermore, these patients only come to medical attention once a

serious event, such as a fall, makes their dysfunction evident. Such patients may even deny dizziness, loss of consciousness, and syncope when reporting a fall, and attribute their fall to mechanical causes. It is thus entirely possible that in our patient's case, the autonomic dysfunction caused the initial fall. In any case, it is evident that the temporal association of events (the SDH clearly preceding symptom onset) was not as relevant as we first thought. Secondly, during autonomic testing, the QSART, a measure of post-ganglionic sudomotor function, was impaired, suggesting that our patient had an impairment in the peripheral ganglia of his sympathetic nervous system. It is implausible to suggest that a central mechanism, such as an SDH, could contribute to impairment in the peripheral autonomic ganglia in the absence of any other neurological impairment.

Finally, this case illustrates a common pitfall in the management of patients with orthostasis. They may cope quite well at home until an event (a fall, fracture, etc.) brings them to medical attention. Once their autonomic dysfunction is diagnosed, they are often encouraged to remain supine and refrain from ambulating, for fear they might fall again. This prolonged immobility can lead to a worsening of their orthostatic symptoms, as in our patient's case. Therefore, in addition to the treatment modalities mentioned above, these patients may require rehabilitation with physiotherapy for help with progressive ambulation, and counselling about lifestyle modifications. The presentation and management of orthostatic hypotension are often quite complex, but a worthy challenge to the clinician internist.

## References

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2. Ohta E, Naito H, Nagoa S, et al. [Hypertensive recurrent intracerebral hemorrhage accompanied with orthostatic hypotension and labile hypertension]. *No To Shinkei* 1990;42:277–81.

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