http://www.kidney-international.org

'Labile hypertension' can be due to autonomic nervous system failure

Kidney International (2009) **75,** 860; doi:10.1038/ki.2008.665

To the Editor: The recent review of baroreflex failure was timely and excellent.[1] However, there were some atypical clinical features in the described patient that might profitably be considered.

The baroreflexes maintain blood pressure (BP) homeostasis. Baroreflexes can fail in three circumstances, namely: (1) failure of afferent signaling because of damage to the baroreceptor, such as in the carotid sinus (BRF); (2) problems with central processing; or (3) damage to sympathetic and parasympathetic nerves (autonomic failure (AF)), which are the effector arms of the reflex.[2]

Patients with BRF have more changes in BP, typically in response to central stimulation (pain or emotions). In contrast, patients with AF typically have significant BP changes due to body position—often with severe supine hypertension and marked orthostatic hypotension,[3] as in this case. We cannot know whether the patient's 'labile BP' during ambulatory monitoring results from the changes in the patient body position or purely because of central stimulation.

The cold pressor test induced hypotension in this patient, the opposite of what would be expected in BRF. One possible explanation is that the patient is hyperventilated in response to cold-induced pain. Patients with AF often become acutely hypotensive in response to hyperventilation (Figure 1), probably through a hypocarbia-induced peripheral vasodilation,[4] although this cannot explain the decreased heart rate.

The importance of this distinction between afferent BRF and efferent AF is that treatments may be different. Although central sympatholytics and anxiolytics would be therapeutic in BRF, AF often responds better to short-acting pressors (for hypotension) and vasodilators.

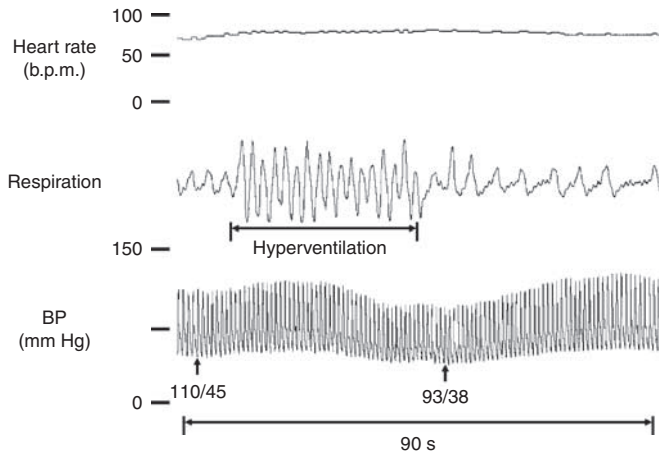

Figure 1 | Hyperventilation in a patient with autonomic failure. Heart rate, respirations, and blood pressure (BP) are shown before, during, and after a 30-s episode of hyperventilation. In this example, the BP decreases (17 mm Hg in systolic BP) with sustained hyperventilation.

ACKNOWLEDGMENTS

Supported in part by NIH grants K23 RR020783, R01 HL0171784, P01 HL56693, and UL1 RR024975.

1. Zar T, Peixoto AJ. Paroxysmal hypertension due to baroreflex failure. *Kidney Int* 2008; **74**: 126–131.
2. Raj SR, Garland EM, Robertson D. Autonomic dysfunction syndromes: orthostatic hypotension and orthostatic tachycardia. In: Mohler ER, Townsend RR (eds). *Advanced Therapy in Hypertension and Vascular Disease*. BC Decker: Hamilton, 2006.
3. Freeman R. Clinical practice. Neurogenic orthostatic hypotension. *N Engl J Med* 2008; **358**: 615–624.
4. Onrot J, Bernard GR, Biaggioni I *et al.* Direct vasodilator effect of hyperventilation-induced hypocarbia in autonomic failure patients. *Am J Med Sci* 1991; **301**: 305–309.

Satish R. Raj[1,2], James M. Luther[1], Kyoko Sato[1] and André Diedrich[1]

[1]*Department of Medicine, Vanderbilt University, Nashville, TN, USA and* [2]*Department of Pharmacology, Vanderbilt University, Nashville, TN, USA*

Correspondence: *Satish R. Raj, Departments of Medicine and Pharmacology, Vanderbilt University, AA3228 MCN, 1161 21st Ave. South, Nashville, Tennessee 37232-2195, USA. E-mail: satish.raj@vanderbilt.edu*

Response to ''Labile hypertension'' can be due to autonomic nervous system failure'

Kidney International (2009) **75,** 860–861; doi:10.1038/ki.2008.670

We thank Raj and coworkers for their comments[1] and how they may apply to the patient whom we described in our 'Renal Consult'.[2]

As to the issue of autonomic failure (AF), we have little evidence of its presence in our patient. His orthostatic blood pressure (BP) change in 19 outpatient visits to our clinic, during the 43 months of follow-up, averaged 9/4 ± 11/3 mm Hg. In only four instances did his BP drop by >20/10 mm Hg upon standing, all related to poorly tolerated treatments or intercurrent illnesses. Furthermore, his sleep BP was lower than awake BP levels on all three ambulatory BP recordings performed. This is in marked contrast to patients with AF who often have supine hypertension and a reversal of the normal day/night BP rhythm.[3] Therefore, despite the limitation that our BP recordings were not coupled to position sensors (or a detailed diary), we have little reason to believe that his BP liability was due to positional changes in the setting of AF.

We found intriguing the suggested explanation for his paradoxical BP fall during the cold pressor test as due to hyperventilation. We had not entertained it, as we never thought our patient had AF. Despite this belief, we invited our patient to return for retesting of his pressor responses on 26 August 2008. His BP and heart rate responses to