Clinical Use of Pulse Wave Analysis: Proceedings From a Symposium Sponsored by North American Artery

Clinical Case Example VI:
Hypertensive Patient with Chronic Kidney Disease

Clinical Question:
Altering Therapy in the Presence of Kidney Failure and Low Diastolic BP

- 70-year-old woman with hypertension and chronic kidney disease stage 4 (estimated glomerular filtration rate 28 mL/min/1.73 m^2)
- Brachial BP: 177/56 mm Hg
- Heart rate: 46 beats per minute
- Chlorthalidone 25 mg daily, carvedilol CR 20 mg daily, and lisinopril 20 mg daily

Initial PWA is shown in Figure 15.

Townsend RR et al. *Journal of Clinical Hypertension.* 2015; 17:7, 503–513.
DOI: 10.1111/jch.12574
http://bit.ly/2gc5mdD

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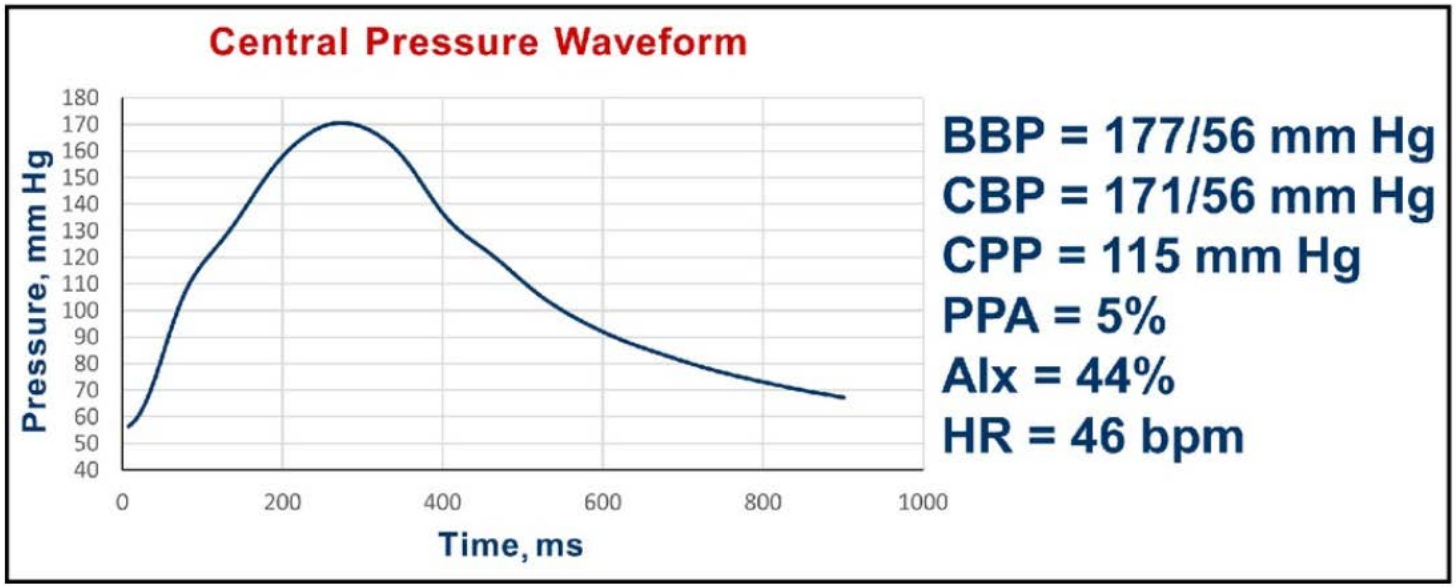
Clinical Case Example VI

Figure 15



Interpretation:

The central pressure profile indicated a pulse pressure amplification of 5%. The central systolic pressure of 171 mm Hg is higher than the desired value of <124 mm Hg. The AIx is about 44%. In this case, the onset of the second peak is found in the aortic pressure contour at approximately 120 mm Hg. With a central pulse pressure of 115 mm Hg, this indicates that about 51 mm Hg of the 115 mm Hg, ie, an AIx of approximately 44%, central pulse pressure is augmented pressure.

BBP indicates brachial blood pressure systolic/diastolic; CBP, central blood pressure systolic/diastolic; CPP, central pulse pressure; AIx, augmentation index; HR, heart rate; bpm, beats per minute.

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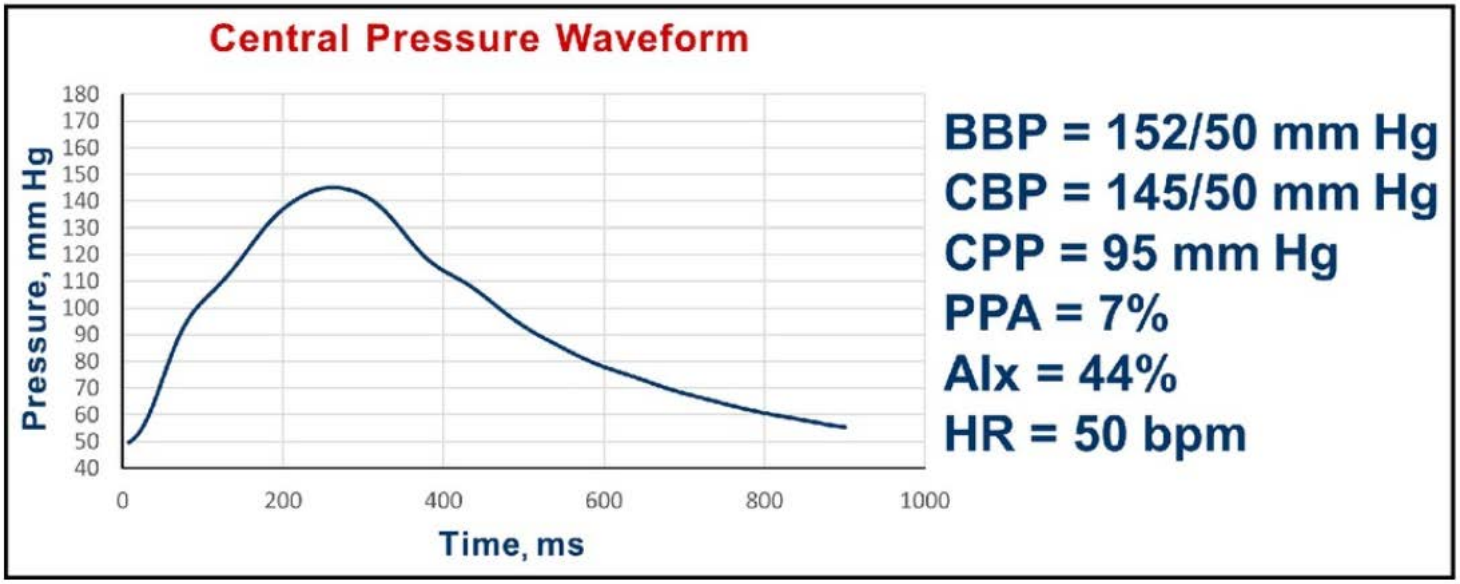
Clinical Case Example VI

Intervention:
Acknowledging that the patient was already taking several medications and had low diastolic BP, the physician remained concerned about poor central pulse pressure amplification and the high brachial systolic pressure; therefore, amlodipine 5 mg daily was added, with the intention to lower brachial and central pressures but not heart rate. Follow-up study showed the waveform (Figure 16).

Figure 16



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Clinical Case Example VI

Interpretation:

The central pressure profile indicated virtually no change in pulse pressure amplification, which was 5% and is now 7%. The central systolic pressure of 145 mm Hg remained higher than the desired value of 124 mm Hg. The AIx remained at about 44%. At this time, the physician decided that 50 mm Hg of diastolic pressure was the limit she felt comfortable accepting and no further changes were made.

Summary:

In this case, the addition of amlodipine produced an expected disproportionate decline in brachial systolic (25 mm Hg) compared with brachial diastolic (6 mm Hg) BP. Changes in the central pressure profile from the PWV provided assurance that the central pressure declined in parallel with brachial pressure. However, the central systolic pressure remained above desirable levels. The addition of vasoactive agents that selectively reduce central pressures without profoundly reducing mean arterial pressure or diastolic pressure (such as organic nitrates) may have desirable hemodynamic consequences on central hemodynamics in this context. However, no clinical guidance exists regarding the use of such agents in this setting. This should be the focus of future research.

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