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THE WATER HAMMER PHENOMENON: PHYSIOLOGICAL MECHANISMS UNDERLYING CHANGES IN SYSTOLIC, DIASTOLIC, AND MEAN ARTERIAL PRESSURE

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The water hammer phenomenon, clinically recognized as Corrigan's or bounding pulse, is a hemodynamic pattern characterized by a rapid and forceful arterial upstroke followed by sudden collapse (Pabba et al., 2023). Although it is classically associated with chronic aortic regurgitation, it also provides a useful physiological model for examining how changes in cardiac function and vascular characteristics differentially influence systolic, diastolic, and mean arterial blood pressure. This perspective highlights that blood pressure is not a single uniform variable, but a composite measure shaped by several interacting mechanisms (Magder, 2018).

The water hammer phenomenon's differential effect on systolic pressure is possibly its most prominent characteristic. The convergence of two main mechanisms results in an evident rise in systolic pressure. First, a greater amount of blood is delivered into the arterial system during each cardiac cycle due to the increased stroke volume caused by increased preload or decreased afterload. Second, and more critically, the decreased arterial impedance impairs the elastic arteries' ability to dampen the pressure wave caused by left ventricular ejection by compromising their Windkessel function. Usually during systole, the elastic aorta expands, absorbing some of the stroke volume and reducing the pressure increase (Reymond et al., 2011). This buffering capacity is reduced in the water hammer state due to decreased compliance and fast flow, which enables the full force of ventricular ejection to be transferred to the periphery. The result is an enlarged systolic peak that rises more quickly and has a greater magnitude, giving arteries like the radial or carotid their distinctive sharp upstroke (Armstrong, 2025).

The effect on diastolic pressure is very different from the effect on systolic pressure, indicating that different forces are in engage in the same hemodynamic state. The water hammer phenomenon usually results in a significant reduction of diastolic pressure. The main cause of this decrease is the rapid diastolic flow, where blood leaves the arterial system during diastole. This drainage happens via two different paths in aortic regurgitation which is backward flow through the incompetent aortic valve into the left ventricle and forward flow via arterioles into the peripheral circulation. Rapid arteriolar drainage without valvular involvement remains possible by the decreased systemic vascular resistance in non-valvular causes of the water hammer phenomenon, such as high-output conditions like anemia, fever, or thyrotoxicosis (Themes, 2016). As a result, arterial pressure drops rapidly during diastole, frequently to levels that would be harmful in other clinical situations.

The most complex and physiologically instructive aspect of the water hammer phenomenon is the effect it has on the mean arterial blood pressure. Unlike the systolic and diastolic blood pressure, which are affected by the changes in the pulse pressure curve, the mean arterial blood pressure maintains relative stability. This is because the mean arterial blood pressure is mainly affected by the cardiac output and the systemic vascular resistance. The product of the cardiac output and the systemic vascular resistance maintain an acceptable value despite the changes in the pulse pressure. In the case of aortic regurgitation, the stroke volume compensates for the reduced systemic vascular resistance (DeMers & Wachs, 2025).

The water hammer phenomenon demonstrates the importance of interpreting systolic, diastolic, and mean blood pressure components together, as a widened pulse pressure with high systolic and low diastolic values can signify a hemodynamic profile of high stroke volume and low impedance rather than hypertension or shock. This shows that systolic, diastolic, and mean arterial pressure should be interpreted in context, since they are influenced by different physiological factors.