Medical Image of the Week: Pulsus Alternans

Figure 1. Telemetry display including arterial pressure waveform, which demonstrates alternating beats of large (large arrows) and small (small arrows) pulse pressure. Concurrent pulse oximetry could not be performed at the time of the image due to poor peripheral perfusion.

A 52 year old man with a known past medical history of morbid obesity (BMI, 54.6 kg/m²), heart failure with preserved ejection fraction, hypertension, untreated obstructive sleep apnea, and obesity hypoventilation syndrome presented with increasing dyspnea over several months accompanied by orthopnea and weight gain that the patient had treated at home with a borrowed oxygen concentrator. On arrival to the Emergency Department, the patient was in moderate respiratory distress and hypoxic to SpO₂ 70% on room air. Physical examination was pertinent for pitting edema to the level of the chest. Assessment of jugular venous pressure and heart and lung auscultation were limited by body habitus, but chest radiography suggested pulmonary edema. The patient refused aggressive medical care beyond supplemental oxygen and diuretic therapy. Initial transthoracic echocardiography was limited due to poor acoustic windows but suggested a newly depressed left ventricular ejection fraction (LVEF) of <25%. The cause, though uncertain, may have been reported recent amphetamine use. The patient deteriorated, developing shock and respiratory failure; after agreeing to maximal measures, ventilatory and inotropic/vasopressor support was initiated.

Shortly after placement of the arterial catheter, the ICU team was called to the bedside for a change in the arterial pressure waveform (Figure 1), which then demonstrated alternating strong (arrow) and weak beats (arrow head) independent of the respiratory cycle. The waveform was recognized as pulsus alternans. Repeat bedside echocardiography suggested severe biventricular systolic impairment and LVEF of

approximately 5-10%, later confirmed by formal transesophageal echocardiography performed prior to a cardioversion for atrial flutter.

Pulsus alternans was first formally described in 1872 and associated with severe left ventricular systolic dysfunction (1). The pattern of pulsus alternans is detectable by palpation, arterial pressure waveform analysis, and Doppler echocardiography. Competing theories in the early 20th century attempted to explain this finding. Wenkebach and Straub, using the Starling relationship, suggested that the alternating force of the pulse is due to alternating filling volumes: greater diastolic volumes accommodated by increased fiber length caused forceful contraction/greater stroke volume with subsequently reduced end systolic and therefore end diastolic volumes for the next (weaker) beat; the consequently reduced force left again greater end systolic and end diastolic volumes for the next (more powerful) beat thereafter. Gaskell, Hering, and Wiggers alternatively proposed the phenomenon was rooted in myocardial contractility fluctuations independent of volumes. Laboratory and animal data supported both theories, but seminal clinical work in the 1960s using concurrent ventriculography and ventricular pressure measurements demonstrated that both mechanisms, in fact, occur in different human subjects (2). The second, Starling-independent mechanism is now thought to be due at least in part to delayed intracellular calcium cycling leading to rhythmic fluctuations in excitation-contraction coupling (3).

Regardless of the underlying physiology, the significance of pulsus alternans as a harbinger of severe ventricular dysfunction and poor prognosis has been recognized and unquestioned since its description. This was unfortunately true in the case of our patient, who developed multiorgan failure despite resuscitative efforts and died three days after admission.

Luke M. Gabe, MD
University of Arizona College of Medicine
Department of Internal Medicine
Division of Pulmonary, Allergy, Critical Care and Sleep Medicine
1501 N. Campbell Ave.
Tucson, AZ USA

References

1. Traube L. Ein fall von pulsus bigeminus nebst bemerkungen tiber die leberschwellungen bei Klappenfehlern und über acute leberatrophie. Ber Klin Wschr. 1872;9:185.
2. Cohn KE, Sandler H, Hancock EW. Mechanisms of pulsus alternans. Circulation. 1967 Sep;36(3):372-80. [\[CrossRef\]](#) [\[PubMed\]](#)
3. Euler DE. Cardiac alternans: mechanisms and pathophysiological significance. Cardiovasc Res. 1999 Jun;42(3):583-90. [\[CrossRef\]](#) [\[PubMed\]](#)