Understanding Arterial Pressure Waveforms

Dr IM Moxham
Medunsa

Introduction
Direct arterial pressure monitoring of patients under anesthesia began more than fifty years ago.1 Even though analysis of the morphology and detail of the arterial pressure (ART) waveform can provide useful diagnostic information, modern physicians seemingly pay little attention to it.2 This change in practice is attributed to the reliance on cuff sphygmomanometry providing “numbers which can be linked in a simplistic way to cardiac strength (systolic pressure) and arteriolar tone (diastolic pressure).”3

Discussion
Indications
Direct measurement of arterial pressure is indicated when,4
• sudden, large changes in blood volume are anticipated
• rapid and extreme changes in blood pressure is expected e.g. surgery for pheochromocytoma
• the shape of the arterial waveform can provide useful information e.g. aortic valve disease
• myocardial function is disturbed e.g. certain dysrythmias

Normal waveform components and their relation to the cardiac cycle
To appreciate the diagnostic clues provided by the ART waveform, one should have full understanding of,
• normal waveform components
• their relation to the cardiac cycle
• the difference in waveforms recorded from different body sites

The arterial pulse is the result of a wave of vascular distention, initiated by the impact of the stroke volume ejected into a closed system with every heartbeat. The forward-propagating pressure wave has both a fast-moving (10m/sec) and slower (0.5m/sec) component.5 The wave is reflected back mostly by the arteriole, which provides the majority of peripheral vascular resistance.6 Peak aortic blood flow acceleration produces the initial rise of the pressure pulse, whereas the ejection of the ventricular volume fills out and sustains the pulse waveform. The systolic components follow the ECG R wave. The interval between the two reflects the time required for the spread of a depolarisation wave, isovolumic left ventricular contraction, aortic valve opening, left ventricular ejection, transmission of the aortic pressure wave to the radial artery and of the pressure signal from catheter to transducer.

The incisura is the dicrotic notch recorded directly from the central aorta and relates to aortic valve closure.7 The peripheral arterial waveform has a later, smoother dicrotic notch that rather reflects arterial wall properties. Simultaneous recording of pressure waveforms from different arterial sites have different morphologies because of the impedance and harmonic resonance of the vascular tree. Predominantly pressure wave reflection influences the shape of the waveform as it travels peripherally,6 because the high resistance to flow in the arterioles diminishes pressure pulsations in small downstream ves-

Figure 1: Normal waveform components and their relation to the cardiac cycle

1. systolic upstroke
2. systolic peak pressure
3. systolic decline
4. dicrotic notch
5. diastolic runoff
6. end-diastolic pressure

(From Mark JB: Atlas of Cardiovascular Monitoring. New York, Churchill Livingstone, 1998: figure 8-1)

Figure 2: The femoral arterial waveform, compared to that in the aortic arch, demonstrates

1. a delayed upstroke
2. a delayed, slurred dicrotic notch
3. a more prominent diastolic wave

In the periphery, systolic pressure is higher, diastolic and mean pressures are lower, and the waveform displays greater amplitude

(From Mark JB: Atlas of Cardiovascular Monitoring. New York, Churchill Livingstone, 1998:fig 8-4)
sels but augment upstream arterial pressure pulses owing to wave reflection.

Even more importantly, the relation between central and peripheral arterial pressures varies with age and is altered by
• physiological changes
• pathological conditions
• pharmacological interventions

Diagnostic clues to pathological conditions

A. Normal arterial and pulmonary artery pressure (PAP) waveform morphologies
• similar timing relative to the ECG R wave

B. Aortic stenosis
• arterial pressure waveform rises slowly (pulsus tardus) and appears overdamped
• delayed systolic peak
• anacrotic notch on pressure upstroke
• dicrotic notch may not be discernible
• arterial pressure small in amplitude (pulsus parvus)

C. Aortic regurgitation
• arterial pressure wave rises rapidly
• pulse pressure increases
• diastolic pressure is low due to the runoff of blood into the left ventricle and periphery during diastole
• two systolic peaks (pulsus bisferiens) from left ventricular ejection and a reflected wave from the periphery respectively

D. Hypertrophic cardiomyopathy
• the bisferiens pulse (spike-and-dome configuration) results from rapid left ventricular ejection in early systole, a rapid fall in arterial pressure as dynamic left ventricular outflow obstruction develops during midsystole, and a late systolic reflected wave

Beat-to-beat variability in ART waveform morphologies
A. Pulsus alternans
- alternating beats of larger and smaller pulse pressures
- usually in severe left ventricular systolic dysfunction, mostly in advanced aortic stenosis
- may occur during general anaesthesia, because of reduced sympathetic nervous system activity in patients with underlying impairment of ventricular contractility
- pulsus alternans is distinguished from a bigeminal pulse in that both create an alternating pulse pressure, but the rhythm is regular in the former

B. Pulsus paradoxus
- an exaggeration of a normal inspiratory fall in systolic arterial pressure, exceeding 10 mm Hg during quiet breathing;
- occurs often in pericardial constriction; almost universal in cardiac tamponade – pulse pressure and left ventricular stroke volume fall during inspiration
- patients with airway obstruction or any condition in which there are large swings in intrathoracic pressure; pulse pressure is relatively unchanged
- occurs during spontaneous breathing, but not in positive-pressure mechanical ventilation during which cyclical changes in SBP of ±10 mm Hg are often seen

C. Hypovolaemia
- a change in peak systolic pressure greater than 15 mm Hg with positive pressure ventilation is predictive of low PCWP (preload); even when arterial blood pressure is maintained near normal by compensatory arterial vasoconstriction

Unusual ART waveforms

A. Intra-aortic balloon pump
- presystolic dip pressure (4) lower than the unassisted end-diastolic pressure (0) and assisted systolic pressure peak (5) lower than the unassisted systolic pressure peak (1) demonstrates effective afterload reduction

B. Cardiopulmonary bypass
- phasic pressure variations (arrows) result from the mechanical action of the bypass roller pump; flow rate is calculated by measuring pulsation frequency

Conclusion
Nowadays, clinicians benefit from the widespread availability of high resolution, multicoloured displays. Renewed interest in ART waveform analysis could expand existing clinical monitoring capabilities. To appreciate the diagnostic clues provided by the ART waveform, the foundation must be laid by

- understanding normal waveform components
- their relation to the cardiac cycle
- differences in waveforms recorded from various body sites, under different physiological and pathological conditions

ART waveforms provide a qualitative approximation of alterations in the circulation. The area under the curve is affected by myocardial performance, peripheral vascular resistance and stroke volume.

References
1. Eather KF, Peterson LH, Dripps RD: Studies of the circulation of anesthetized patients by a new method for recording arterial pressure and pressure pulse contours. Anesthesiology 10:125,1949
2. Mark JB, Slaughter TF, Reves JG: Cardiovascular Monitoring. In Miller RD (ed): Anesthesia, 5th ed vol 1. New York, Churchill Livingstone, 2000, p 1131-1142
3. O’Rourke MF, Gallagher DE: Pulse wave analysis, J Hypertens 14:S147,1996
4. Sykes MK, Vickers MD, Hall CJ: Principles of Measurement & Monitoring in Anaesthesia and Intensive Care, 3rd ed, 1991, p 165-174
5. Bruner JMR et al: Comparison of direct and indirect methods of measuring arterial blood pressures. Med Instrum 15:11-21,1981
6. O’Rourke MF, Yaginuma T: Waveform reflections and the arterial pulse. Arch Intern Med, 144:368-371,1984
7. Braunwald E et al: Time relationship of dynamic events in the cardiac chambers, pulmonary artery and aorta in man. Circ Res 4:100,1956
8. Franklin SS, Weber MA: Measuring hypertensive cardiovascular risk: The vascular overload concept. Am Heart J 128:793,1994
9. Sivarajan M: Cardiovascular Monitoring. SA National Part 1 Anaesthesia Refresher Course, 2002, p 2-3
10. Freeman AB, Steinbrook RA: Recurrence of pulsus alternans after fentanyl injection in a patient with aortic stenosis and congestive heart failure. Can Anaesth Soc J 32:654,1985
11. Braunwald E: The physical examination. In Braunwald E (ed): Heart Disease, A textbook of cardiovascular medicine. Philadelphia, WB Saunders Company, 1992, p 13
12. Wynne J, Braunwald E: The cardiomyopathies and myocarditides: Toxic, chemical and physical damage to the heart. In Braunwald E (ed): Heart disease, A textbook of cardiovascular medicine. Philadelphia, WB Saunders Company, 1992, p 1398
13. McGregor M: Pulsus Paradoxus, N Engl J Med 480,1979
14. Mark JB: Pericardial constriction and cardiac tamponade. In Mark JB (ed): Atlas of cardiovascular monitoring. New York, Churchill Livingstone, 1998, p 313
15. Fowler NO: Clinical problem solving, A broken heart. N Engl J Med 334:1474,1996
16. Mark JB: Hemodynamic observations during cardiopulmonary bypass. In Mark JB (ed): Atlas of cardiovascular monitoring. New York, Churchill Livingstone, 1998, p 327
17. Lake CL: Monitoring of Arterial Pressure. In Lake CL (ed): Clinical Monitoring for Anaesthesia and Critical Care, 2nd ed, WB Saunders, 1994, p 115-126