Cardiac output measurement

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Abstract

Cardiac output measurement is used to guide fluid and inotropic drug therapy. Techniques employ modelling of the circulation to derive estimates of cardiac output from readily measured variables, including thermodilution, analysis of arterial pressure waveforms, Doppler measurements of blood flow velocity, and electrical bioimpedance.

Keywords Bioimpedance; cardiac output; oesophageal Doppler; pulse contour analysis; thermodilution

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Cardiac output is the product of ventricular stroke volume and heart rate, and estimates of it are used to guide fluid and inotropic therapy in intraoperative and critical care settings, in an attempt to improve clinical outcomes.

The pressure–flow relationship

When the stroke volume is ejected from either the left or right ventricle, pressure is generated in the aorta and pulmonary arteries by a combination volume change and the propagation and reflection of waves generated by the energy of ejection. Arterial pressure is the sum of these effects (Figure 1).

Volume change: the great vessels are compliant, so during systole, more blood is ejected into them than actually leaves. In diastole, they passively empty into smaller arteries, and return to their original calibre, at a rate determined by arterial compliance and vascular resistance.[1] The volume of blood entering a vessel or cardiac chamber must equal the volume of blood leaving, **during each cardiac cycle**, or distension would occur. Stroke volume therefore consists of systolic and diastolic components, preventing systolic pressure overshoot during rapid ejection, and allowing for it to be delivered to the arteries throughout the cardiac cycle.[2] Aortic pressure peaks during systole and declines exponentially during diastole, determined by a time constant (τ).

Wave generation: the energy of stroke volume ejection generates compression and decompression waves in the arterial system, which rapidly propagate and are reflected at points of branching and calibre change. Waves propagating through the arterial system are the result of successive ventricular ejections, rather than harmonic oscillation. This is demonstrated by the exponential pressure decline to an asymptote, during the prolonged diastole after a premature ventricular complex.[3]

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Learning objectives

After reading this article, you should be able to:

- understand the relationship between cardiac output, stroke volume, heart rate and arterial pressure
- describe how estimates of cardiac output are derived from other measured variables
- outline additional information derived from each measurement technique

Measurement techniques

The circulation is a complex system of branching vessels with variable flow velocity, calibre and compliance, so pressure and flow vary across measurement sites. Blood flow measurement creates challenges, because of difficulty siting instruments in or near blood vessels of interest. The relationships between arterial pressure, blood flow, vascular resistance and vessel dimensions are determined by Ohm's Law and the Hagen-Poiseuille Law of laminar flow. Haemodynamic monitors measure a spectrum of variables, including arterial pressure, blood velocity, indicator dilution, or electrical impedance to estimate cardiac output, and other values reflecting haemodynamic status.

Measurement of indicator dilution

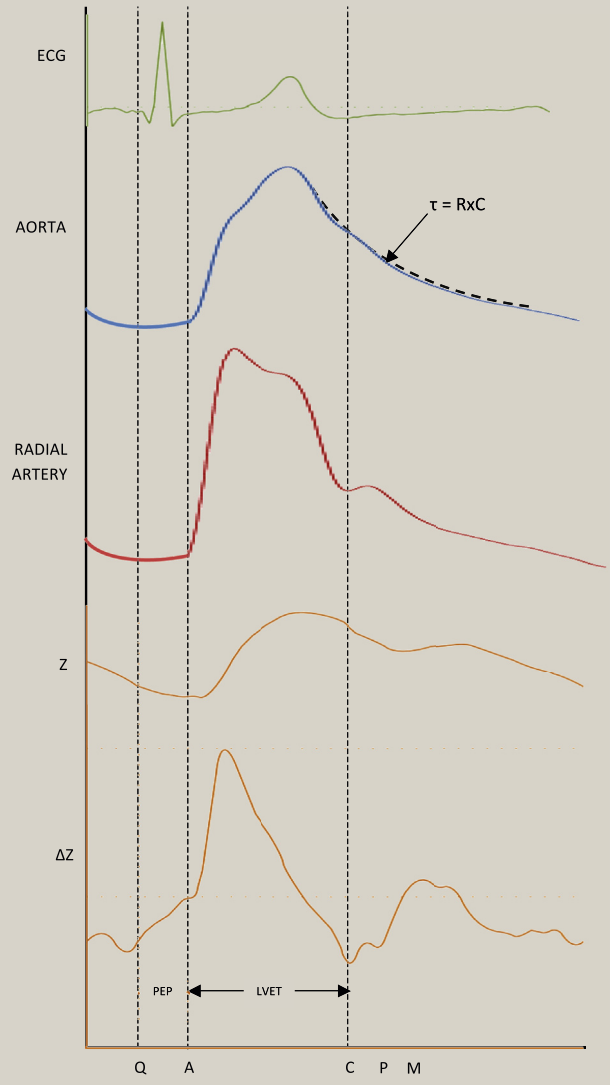
Indicator dilution employs the principle that a known **volume** (V_0) and **concentration** (C_0) of an indicator is injected into the circulation, diluting the indicator within a **volume of distribution** (V_1). The **indicator concentration** (C_1) is detected at a remote site, where it initially increases and then decreases in a non-stepwise manner. If $V_1 = C_0 \times V_0/C_1$ and cardiac output (Q) $= V_1/t$, then cardiac output is inversely related to the area under the concentration–time curve (Figure 2a).

Thermodilution uses the concept of a **thermal indicator**, where cold crystalloid solution injected into the circulation produces a blood temperature change at the measurement site.[4,5] Cardiac output is inversely proportional to the area under the blood temperature change–time curve.

The pulmonary artery catheter was developed as the route by which boluses of cold crystalloid solution could be injected into the right atrium, with proximal and distal thermistors to detect injectate temperature and blood temperature in the pulmonary artery. The catheter is inserted via a central vein and is floated into the pulmonary artery using a small inflatable balloon on the catheter tip. As the temperature change is measured across the right ventricle, this technique estimates right ventricular stroke volume.

Continuous cardiac output (CCO) monitors use an element in the proximal part of the catheter to intermittently heat the surrounding blood, as it passes through the right atrium. The temperature change is much smaller than with cold crystalloid injection, making the measurement more prone to error from 'thermal noise'.

ECG, aortic and radial artery pressure and bioimpedance waveforms



Note systolic pressure augmentation and secondary, non-dicrotic peaks caused by wave effects in the radial artery waveform. Ventricular activation, Q, aortic valve opening, A, aortic valve closing, C, pulmonary valve opening, P, and mitral valve opening, M. LVET, left ventricular ejection time; PEP, pre-ejection period. Bioimpedance, Z, bioimpedance change, ΔZ.

Figure 1

Transpulmonary thermodilution (TPTD) employs temperature measurement in a peripheral systemic artery (e.g. femoral artery) following cold crystalloid injection into a central vein. This has the advantage of not requiring catheterization of the pulmonary artery. The indicator is diluted to a greater extent, producing a smaller thermodilution curve. This technique estimates left ventricular stroke volume.

Lithium dilution cardiac output (LiDCO) monitors employ the injection of lithium into a central vein, and measurement of lithium concentration with an ion-sensitive electrode attached to a peripheral arterial line. Lithium recirculation limits the ability to repeat measurements at short time intervals.

Additional clinical data derived from indicator dilution measurement

Global end-diastolic volume (GEDV) is a reflection of the adequacy of preload. It represents end-diastolic volume of all cardiac chambers, and is calculated as the difference between ITTV and PTV (Figure 2b). The 'normal' range is 680–800 ml m^{-2}, which is greater than the actual end-diastolic volume of the cardiac chambers.

Extravascular lung water (EVLW) is a reflection of the severity of pulmonary oedema. It is theoretically represented by the volume of indicator sequestered in the lung during transit, and is the difference between pulmonary thermal volume (PTV) and pulmonary blood volume (PBV). Experimentally derived formulae are used to calculate EVLW, which estimate the difference between ITTV and the sum of GEDV and PBV [Reuter, Bendjelid]. One example is: $EVLW = (CO \times MTT_T) - (1.25 \times GEDV)$.

There is **controversy** about EVLW and GEDV calculations. Firstly, CO, MTT_T and EDT_T are all derived from the same thermodilution curve, so any error in the detection of the indicator is multiplied. Secondly, there is conflict between the assumptions that the calculation of cardiac output should occur with no loss of indicator while at the same time the calculation of EVLW assumes that there is. Thirdly, GEDV is calculated from cardiac output, assuming that they are invariably related, which they are not.[6]

Pulse contour analysis

Early investigators discovered an empirical correlation between central pulse pressure and stroke volume. Analysis of the peripheral arterial pressure waveform to derive estimates of stroke volume and cardiac output necessitates accurate reproduction of the central aortic pressure waveform and an estimation of arterial resistance.

The time constant (τ) of an exponential decay curve fitted to the diastolic part of the central aortic pressure waveform provides a combined estimate of large vessel compliance (C) and total arterial resistance (R) (Figure 1). To derive a value for arterial resistance, compliance must be estimated. Current technology employs two main models:

Windkessel model: so-called because the term was used by Starling to describe the compliance 'chamber' represented by the distensible aorta (Figure 3a). This model attempts to fit the area under the curve (AUC) of arterial waveforms measured over several cardiac cycles to physiological models which predict the central aortic pressure waveform. These approaches require calibration against another method of CO measurement, such as thermodilution or indicator dilution, to calculate a constant which represents large vessel compliance. Derived values for compliance and vascular resistance are then entered in a calculation of cardiac output, and subsequent changes in measured values are used to estimate cardiac output from the point of calibration.[7]