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Non-invasive method for rapid bedside estimation of inotropy: theory and preliminary clinical validation

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Editor's key points

- Measurements of myocardial contractility could guide diagnosis and therapy when cardiac function is impaired.
- The authors have developed formulae for bedside assessment of inotropy and arterial impedance.
- These formulae require as inputs variables from Doppler ultrasonography, and arterial and central venous pressure.
- Retrospective analysis showed good separation of these measures in control and left ventricular failure patients.

Background. There are numerous techniques which attempt to quantify inotropy (or myocardial contractility). None has yet found general acceptance in anaesthesia and critical care as a practical method. We report a novel approach to the determination of inotropy as a bedside procedure which could identify low inotropy states in patients with clinical heart failure.

Methods. We estimated the potential and kinetic energy delivered by the left ventricle using continuous-wave Doppler ultrasound (ultrasonic cardiac output monitor, Uscom, Sydney, Australia) and data available at the point of care. A formula to calculate effective inotropy [Smith-Madigan inotropy index (SMII)] was tested against historical haemodynamic data for 250 control subjects (ASA I patients from preoperative clinic) and 83 patients with acute left ventricular failure (LVF) of New York Heart Association Grade 4 (LVF group). The ratio of potential to kinetic energy (PKR) was investigated as a measure of arterial impedance.

Results. Significant differences were found between the control and LVF groups for cardiac index, mean (range)=3.37 (2.84–5.32) *vs* 1.84 (1.43–2.26) litre min $^{-1}$ m^{-2}; stroke volume index (SVI), 49.2 (39–55) *vs* 34.3 (23–37) ml m^{-2}; systemic vascular resistance, 893 (644–1242) *vs* 1960 (1744–4048) dyn s cm^{-5}; SMII, 1.78 (1.35–2.24) *vs* 0.73 (0.43–0.97) W m^{-2}; and PKR, 29:1 (24–35:1) *vs* 124:1 (96–174:1), $P<0.001$ in each case. Normal ranges were calculated for SMII and PKR as mean (+/−1.96) standard deviations, yielding 1.6–2.2 W m^{-2} for SMII, and 25–34:1 for PKR.

Conclusion. The method clearly identified the two clinical groups with no overlap of data points. The discriminant power of SMII and PKR may offer valuable diagnostic methods and monitoring tools in anaesthesia and critical care. This is the first report of normal ranges for SMII and PKR.

Keywords: Doppler ultrasonography; inotropism cardiac; left ventricular function; myocardial contractility; systemic vascular resistance

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While inotropy (or myocardial contractility) as a concept is well known to all clinicians, it is seldom thought of as a measurable quantity. We sought to develop a simple bedside test based on haemodynamic theory that could evaluate inotropy in a wide range of clinical conditions, and which could be performed by non-experts in a clinically timely manner. We selected left ventricular patients presenting as emergencies to the hospital as the obvious group to study.

In critical care, the diagnosis of left ventricular failure (LVF) is based on the history and clinical signs, sometimes aided by echocardiography and chest radiology. The absolute degree of inotropic failure in the overall clinical presentation is seldom if ever quantified. Commencement, dosing, and withdrawal of vasopressors, vasodilators, and inotropes is still largely based on clinical assessment, sometimes assisted by measurement of surrogates of inotropy such as ejection fraction (EF) or aortic ejection velocity, despite the well-known shortcomings of these indices in critical care. This is particularly so in complex surgical patients where vascular tone and fluid loading status are highly variable and changing.[1–5] More sophisticated echocardiographic techniques have been developed in an attempt to overcome the problems of sensitivity to preload and particularly afterload, but have achieved little penetration in critical care.[2 4 6]

Comparatively little research has been performed to evaluate methods of impedance matching of the left ventricle to the vascular tree.[7–12] Despite its fundamental physiological importance, this is seldom considered in anaesthesia or

critical care, largely because of the difficulties in assessing ventriculo-aortic coupling. A normally powered ventricle will struggle to eject a normal stroke volume (SV) against a high afterload, while a low afterload may be of considerable benefit to the failing heart. Vasodilator use in heart failure is a good example of this concept of matching or coupling the arterial impedence to the capability of the ventricle, while excessive vasodilation of the healthy circulation leads to a mismatch of ventriculo-arterial impedence with resulting hypotension.[8 10 12]

If a simple and rapid method of determining inotropy could be developed then questions regarding the usage, dosing and combination of inotropes would be greatly simplified. Ideally, for any method to gain clinical acceptance it should be simple, quick, accurate, reproducible, easily learned and taught, and applicable to the entire range of emergent patients. A simple bedside method to evaluate arterial impedance and ventriculo-aortic impedance matching (coupling) would be a desirable additional feature.

Methods

The publication of the historical data used in this study was approved by the ethical committee of the Greater Western Area Health Service.

Inotropy calculation

We developed a formula based on haemodynamic theory, to calculate the potential and kinetic energy developed by the ventricle, which results from ventricular inotropy, which is then transferred to the aorta, using data easily obtained in the operating theatre or critical care situation. We then tested the formula using a bespoke computer program against stored data for 250 healthy subjects, the control group, and 83 patients known to have acute LVF, the LVF group (see below).

Basic theory

If we consider a simple hand-operated water pump, as shown in Fig. 1, then each time the pump handle moves through a full sweep it will generate one SV of the pump. This SV will be produced at a hydrostatic pressure (HP) and flow velocity (*V*) which is determined by the force on the pump handle. If we now move the handle more forcibly, the pump will still produce the same SV, but in a shorter flow time (FT) and with a greater HP and flow velocity. The differences in FT, mean velocity (*V*mean) and HP are solely attributable to the increased power that was applied to the pump handle. If these variables can be measured for the heart then instantaneous output power can be calculated, which is a direct function of inotropy. Figure 2 shows a schematic Doppler ejection waveform for blood flow through the aortic valve into the aorta. The measured maximum or peak flow velocity (*V*pk), and the calculated mean velocity of ejection (*V*mean) (see below), and the total duration of flow in one ejection (heart beat) flow time (FT) are shown.

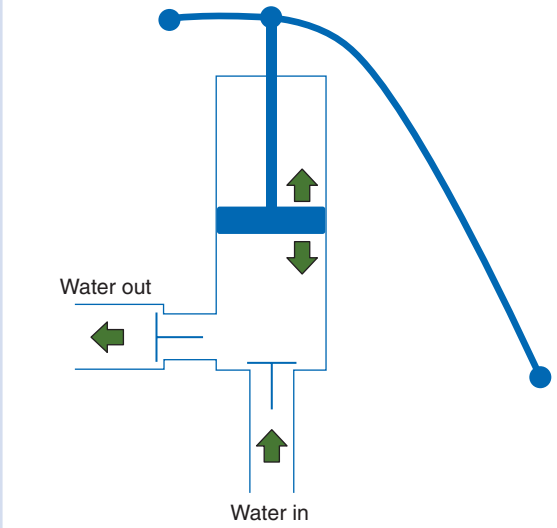

Fig 1 A simple piston water pump. With each sweep of the handle the pump will deliver one stroke volume SVol, at a flow velocity *V*, hydrostatic pressure HP, and in a given flow time (FT) determined by the force exerted on the handle. The parameters of SVol, *V*, HP, and FT can therefore be used to calculate the power transferred to the system.

When the heart contracts it follows the 'all or nothing rule', it will contract with all the power that it has available at that moment in time, which depends on its inotropy.[13] SV, FT, and *V*mean [derived by integrating the area under the curve in Fig. 2 and known as the velocity–time integral (vti)] can be measured using Doppler ultrasound, and the mean arterial pressure (MAP), which equates to HP, by automated oscillometry or from arterial lines. From these, along with blood density which can be derived from haemoglobin

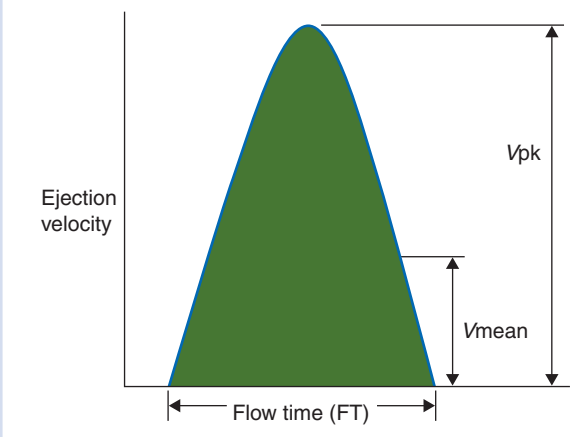

Fig 2 Diagrammatic representation of aortic transvalvular flow as measured by continuous wave Doppler ultrasound. *V*mean is calculated by integration of the area under the velocity–time curve to give the velocity time integral, vti.