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REVIEW PAPER

The use of the oesophageal Doppler in perioperative medicine: new opportunities in research and clinical practice

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Abstract The oesophageal Doppler (OD) is a minimally invasive haemodynamic monitor used in the surgical theatre and the ICU. Using the OD, goal-directed therapy (GDT) has been shown to reduce perioperative complications in high-risk surgical patients. However, most GDT protocols currently in use are limited to stroke volume optimisation. In the present manuscript, we examine the conceptual models behind new OD-based measurements. These would provide the clinician with a comprehensive view of haemodynamic pathophysiology; including preload, contractility, and afterload. Specifically, volume status could be estimated using mean systemic filling pressure (MSFP), the pressure to which all intravascular pressures equilibrate during asystole. Using the OD, MSFP could be readily estimated by simultaneous measurements of aortic blood flow and arterial pressure with sequential manoeuvres of increasing airway pressure. This would result in subsequent reductions in cardiac output and arterial pressure and would allow for a linear extrapolation of a static MSFP value to a "zero flow" state. In addition, we also demonstrate that EF is proportional to mean blood flow velocity measured in the descending thoracic aorta with the OD. Furthermore, OD-derived indexes of blood flow velocity and acceleration, as well as force and kinetic energy, can be derived and used for continuous assessment of cardiac contractility at the bedside. Using OD-derived parameters, the different components of afterload: inertia, resistance and elastance, could also be individually determined. The integration of these additional haemodynamic parameters could assist the clinician in optimising and individualising haemodynamic performance in unstable patients.

Keywords Haemodynamic monitoring · Doppler technique · Intensive care · Perioperative

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1 Introduction

Goal-directed therapy (GDT) which is the use of therapeutic interventions to achieve haemodynamic targets and optimize tissue oxygen delivery, has been shown to improve perioperative outcomes in surgical patients at high-risk for post-operative mortality [1–3]. This group, usually named high-risk surgical patients (HRSPs), is characterized by older patients with important comorbidities submitted to high-risk, often emergent, surgery [4].

In recent years, a growing number of minimally invasive technologies have been made available to the clinician, allowing cardiac output monitoring at the bedside [5]. This

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paper deals specifically with the oesophageal Doppler (OD) which consists of an orally or nasally inserted ultrasound probe that measures beat-to-beat blood flow velocity in the descending thoracic aorta. By integrating the velocity–time waveform (Fig. 1a) it calculates stroke distance (SD) which is the distance travelled by a column of blood that passes in front of the probe at each left ventricular systole; stroke volume (SV) is then calculated as the product of SD and aortic cross sectional area [6]. The latter can be directly measured by M-mode echography or estimated based on normograms that account for the patient's age, height and weight; both solutions have benefits and pitfalls and we refer the reader to the many reviews on the use of the OD for cardiovascular monitoring where the general limitations of this technology are discussed in detail (e.g. [6, 7]). More recent ODs allow for the simultaneous acquisition of an invasive arterial pressure signal in addition to the flow signal (Fig. 1b).

Randomized trials using OD-guided therapy in cardiac [8], abdominal [9–12] and orthopaedic [13, 14] surgery have shown a reduction in post-operative complications and/or hospital length of stay. However, most algorithms used in trials of GDT still rely on limited information to guide therapy: an estimate of cardiac output complemented, at best, by a surrogate for volume status, thereby potentially over-simplifying the patient's haemodynamic assessment. This may lead the clinician to use unnecessary or even harmful interventions.

Instead, conceptualisations of the heart as a haemodynamic pump with the three critical components for optimal haemodynamic performance: preload, contractility, and afterload would allow for a more pragmatic approach. Furthermore, integration of additional information originating from simple, reliable, and dynamic indexes of these components is likely to contribute to a better understanding of patient physiology. This would consequently allow for individualised therapy to improve oxygen delivery and organ function in HRSPs. Innovative analysis of the OD signal has the ability to provide such comprehensive information.

In this manuscript, the authors attempt to examine the conceptual models behind new continuous OD-based haemodynamic measurements that could be obtained at the bedside and how these new haemodynamic markers could be integrated to guide haemodynamic therapy for HRSPs.

2 Volume status assessment

Accurate bedside assessment of the intravascular volume status of a haemodynamically unstable patient is important and remains challenging. Indeed, overzealous or under-administration of intravenous fluid loading can lead to

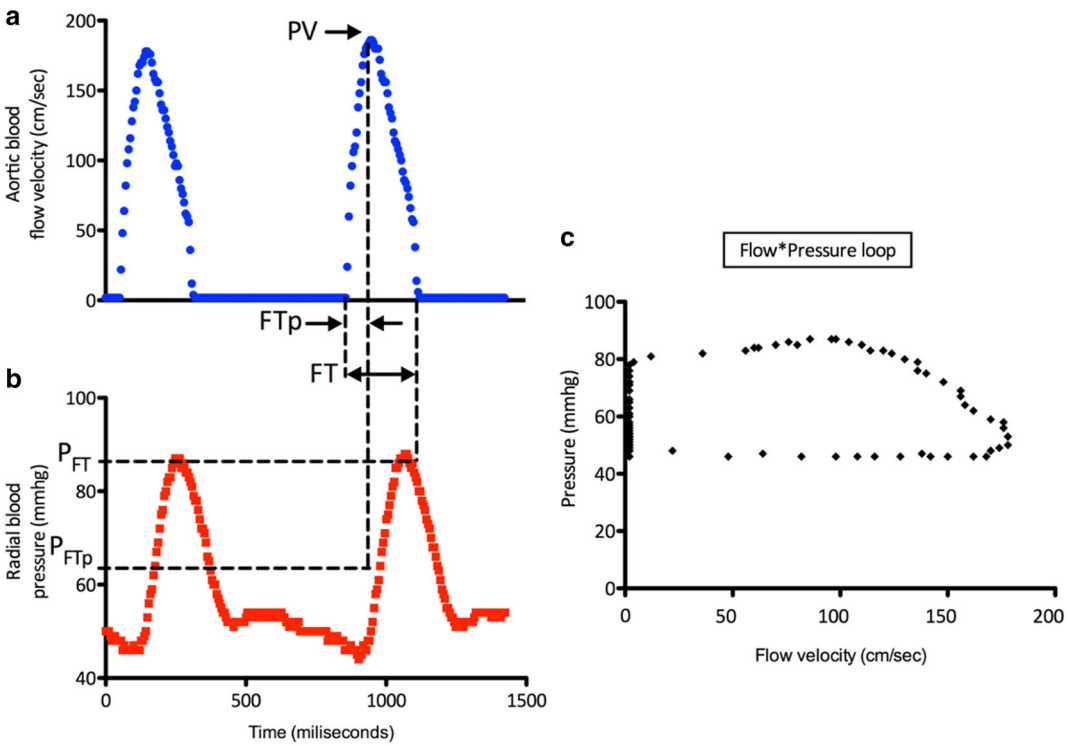

Fig. 1 Flow velocity and Pressure curves in the Oesophageal Doppler Monitor (ODM). **a** Blood flow velocity in the distal thoracic aorta as a function of time. **b** Recent versions (ODM+) also allow for the simultaneous display of a continuous invasive arterial pressure signal. **c** Plotting simultaneously-acquired flow velocity and pressure measurements could therefore yield a flow-pressure loop of 1 cardiac cycle. *FT* flow time, *FTp* flow time to peak, P_{FT} pressure at flow time (end of systole), P_{FTp} pressure at peak flow, *PV* peak velocity

associated morbidity and mortality [15]. Thus, a patient-specific "U-shaped curve" for complications versus fluid administration would theoretically exist [16]. Clinicians therefore aim to hit the "sweet spot" when managing a patient's haemodynamic status.

Clinical symptoms, such as skin turgor, are notoriously unreliable in assessing volume status [17]. In addition, static filling pressures such as central venous pressure (CVP), capillary wedge pressure and mean arterial pressure (MAP) have not much aided the clinician to discriminate hypotended patients who would be fluid responsive [18]. Consequently, dynamic parameters that explore heart–lung interactions have become a focus of interest to assess fluid responsiveness in deeply sedated patients. Indeed, respiratory-induced variations in stroke volume (SVV) and pulse pressure (PPV) are thought to be better markers to discriminate hypotented patients who would be fluid responsive. These dynamic parameters do not directly measure volaemia but provide an estimate of the patient's position on the Frank-Starling curve; pathophysiologically, it refers to the relation between preload and stroke volume at a given time and for a specific contractility state. In addition, conditions that frequently occur in the operating theatre and the ICU such as atrial fibrillation, open-chest conditions, assisted ventilation, tidal volume ventilation below 8 ml/kg, and high abdominal pressure render these measurements less reliable to guide fluid therapy [19]. Passive leg raising, PEEP, or mini-fluid challenges have been used as alternatives, but limitations still exist; principally in the operative room. Furthermore, the reliability of these tests is still unsatisfactory [20, 21].

We are therefore still unable to accurately and universally define hypo-, hyper- or normovolaemia in the OR. i.e: is the stressed blood volume adequate in this patient undergoing hypotension? From a physiological point of view, Arthur Guyton provided us with a number of valuable insights in haemodynamic and venous return physiology. Guyton experimented with mean systemic filling pressure (MSFP) to ultimately assess vascular compliance and stressed volume (Vs) in dogs [22]. Note that Vs is that volume that *stresses* the vascular walls and results in a distending pressure. This distending pressure is subsequently referred to as the *mean systemic filling pressure* (MSFP). Furthermore, MSFP is the pressure to which all intravascular pressures, arterial and venous alike, equilibrate during conditions of zero flow (as in cardiac arrest), and it is determined by systemic venous vascular compliance. MSFP can be regarded as a surrogate of capillary pressure, the "pivot" between arterial resistance (Ra) and venous resistance (Rv).

Guyton also found that MSFP is the driving pressure for venous return (VR) [23]. Furthermore, VR is equal to cardiac output (CO):

$$CO = VR = (MSFP - CVP)/Rv. \quad (1)$$

where, Rv is the resistance to VR.

On its own, MSFP has shown to be a reasonably good indicator of stressed blood volume [24–26]. In the present review, the authors surmise that MSFP could be of additional value when utilized with other parameters in GDT protocols, such as SV or SVV. Conditions observed in the operating theatre are often accompanied by considerable fluid shifts and positional changes, which can make these "atypical" physiologic conditions even more extreme, while also rendering traditional haemodynamic parameters difficult to interpret. Even if MSFP is not able to predict fluid responsiveness, the authors suggest that MSFP is a way to assess the stressed blood volume and to validate that the volume infused has tested the cardiac preload. The present concept is of great importance when patients suffer from inflammatory disease and capillaries leak and when the quantity of volume to infuse to increase cardiac output (fluid responsiveness) is difficult to estimate. Assessment of the determinants of VR could help the physician weigh management options.

Previously, MSFP values could only be measured in patients who were in cardiac arrest [27]. Nonetheless, three methods have been developed that can be used at the bedside [24, 26, 28]; One method requires a rapid cuff inflator and uses the arm as a model for the rest of the body; as flow is stopped arterial and venous pressure equilibrate in the arm [24]. This arm pressure (Parm) is a surrogate for MSFP. Another method developed by Parkin uses the concept of capillary pressure or MSFP as the pivot between arterial and venous pressure [26]. The fluctuations in venous and arterial pressure from diastole through systole are used to determine the pivot value (almost like a seesaw mechanism). These method and a discussion about accuracy is beyond the scope of this article.

One specific dynamic method has been developed by Jansen and co-workers which uses heart–lung interactions to estimate MSFP [29] and employs a series of 12-s respiratory "holds" at incremental airway pressures at 5, 15 and 25 cm H_2O (Fig. 2a). Subsequently, mean systemic arterial pressure and cardiac output decrease and central venous pressure increases until a new steady state is reached for all three variables, after approximately 7 s (Fig. 2a).

Consequently, the MAP, CVP, and CO measured at the end of each hold, could all be linearly extrapolated to 'zero flow' conditions, assuming a constant arterial tone. The point where no flow occurs, which is the MSFP value for the patient, would coincide with the intersection CVP and MAP curves. In fact, this principle can be used with any combination of these three variables (MAP, CVP and CO), using the OD or other monitor, as illustrated in Fig. 2b with

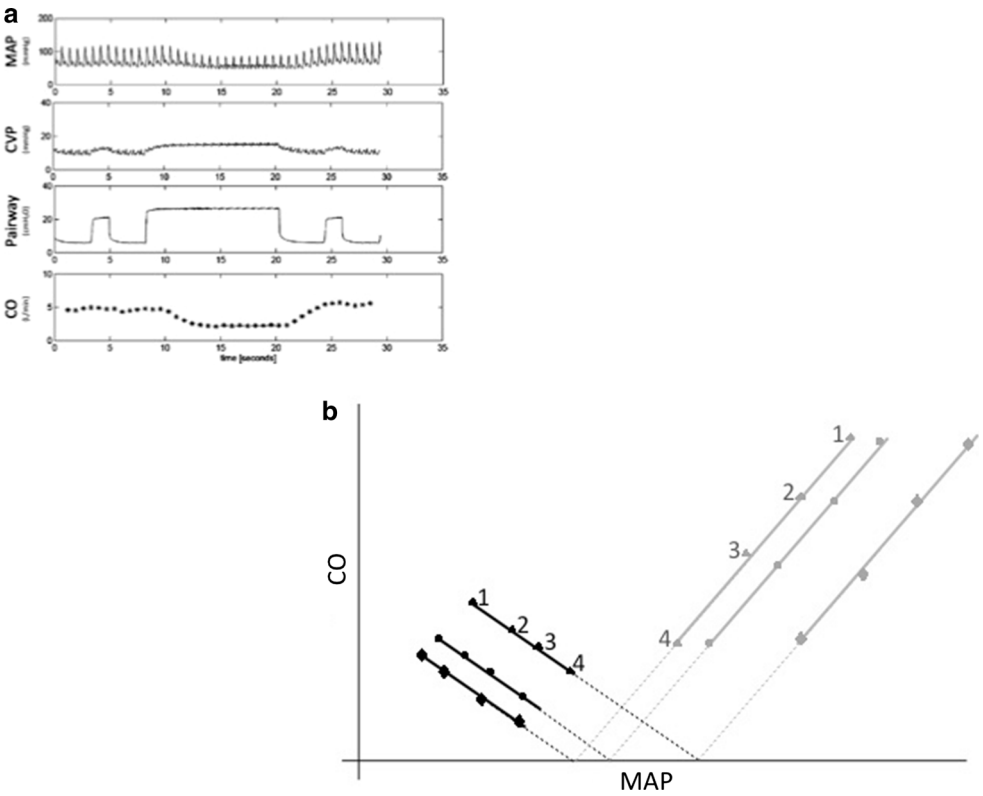

Fig. 2 Calculation of mean systemic filling pressure (MSFP) with the Oesophageal Doppler Monitor. **a** An example of an inspiratory hold of 12 s at 25 cm H_2O. New steady state values of arterial and venous pressures and cardiac output, reached after approximately 7 s, can be used to compute MSFP. **b** Repeated respiratory "holds" (1–4) with incremental inspiratory pressures decrease cardiac output (CO) and mean arterial pressure (MAP). MSFP is the point where the extrapolated lines of decreasing CO (*black*) and MAP (*grey*) intersect at conditions of zero flow in euvolemic (*circles*), hypovolemic (*lozenges*), and hypervolemic (*triangles*) states. *CO* cardiac output, *CVP* central venous pressure, *MAP* mean arterial pressure, P_{airway} airway pressure

the use CO and MAP after 4 respiratory holds with increasing airway pressures.

Although this setup could be laborious, it is likely to be the most accurate method to determine the true MSFP at the bedside because both CVP and MAP (or MAP and CO) are used to estimate the same MSFP value (i.e., a double verification). However, respiratory holds may recruit blood from the pulmonary circulation to the general circulation, thus contributing to an overestimation of MSFP. In spite of this, the pulmonary vasculature will retain no more than 300 ml of blood, and the subsequent overestimation will therefore be within a 5 % range of total blood volume.

With the ODM+, this technique could be simplified because both (aortic) blood flow and (peripheral) arterial pressure are measured simultaneously in one single device. This subsequently makes the extrapolation of conditions of "zero flow" with increasing airway pressures, and hence MSFP, easier without the need of central venous cannulation. Furthermore, MSFP values could be measured accurately using only two respiratory holds [30]. This process would simplify the procedure even more and make bedside assessment in the surgical theatre or the ICU feasible. Thus, using continuous and simultaneous pressure and flow measurements, MSFP could be measured within 1 min by extrapolation of two variables (CO and MAP) to conditions of "zero flow" with two respiratory holds. One might also envision that the decline in both pressure and flow during the initial phase of the respiratory hold process may be extrapolated to calculate MSFP from a pressure-flow loop (Fig. 1c). This would consequently make MSFP measurement very straightforward.

3 Assessment of ventricular performance

Hypotension and tissue hypoperfusion may persist despite adequate volume assessment and preload optimisation. Therefore, in this setting, the evaluation of ventricular performance by assessment of contractile capacity could help to define causes of circulatory failure, support the judicious use of inotropic drugs, and monitor response to therapy. Indeed, clinical signs of acute heart failure such as hypotension, mottled skin or pulmonary crackles, may be insidious, unreliable and non-specific in assisting the clinician to estimate intrinsic heart function in an acutely unstable patient.

Ejection fraction (EF) has traditionally been utilized as the "gold standard" for the clinical measurement of contractility:

$$EF = \frac{SV}{EDV} \tag{2}$$

where SV represents stroke volume, and EDV is end-diastolic volume.

However, the primary limitation of this ventricular function marker is that it requires a skilled physician to

perform continuous cardiac imaging during the course of surgery. Furthermore, although EF is relatively specific to systolic function in the case of stable vascular load, it may not be sufficiently sensitive during clinical use, particularly in highly "dynamic" environments, such as the operating theatre or the ICU, where left ventricle preload and afterload can change dramatically over a short period of time [31]. Thus, EF may be thought of as an index of how left ventricle contractility is able to respond to changes in afterload and not an index of contractility per se. Therefore, a continuous minimally-invasive means of measuring contractility would be ideal. In this regard, the OD could be utilized.

The distal thoracic aorta can be modelled as a cylinder and it can be shown that [32]:

$$EF = \frac{k \cdot \pi \cdot r^2 \cdot \bar{v} \cdot FT}{EDV} \tag{3}$$

where $\bar{v}$ represents average or mean blood flow velocity, FT is the flow time or ejection period and $\pi \cdot r^2$ is the cross sectional area of the aorta. Note that $\bar{v} \cdot FT$ is mathematically identical to the integral of velocity over time (VTI) [33]. The constant k is used to "correct" for the portion of the proximal aortic blood flow that does not "reach" the distal aorta. Note that k has a typical value of approximately 1.4 and is dimensionless [34].

Inspection of Eq. (3) demonstrates that EF is proportional to the velocity of distal aortic blood flow. This has been demonstrated in critically ill patients where the OD-derived peak velocity (PV) and mean acceleration have been found to correlate with echocardiography-derived EF [35]. Importantly, PV was also shown to be a sensitive marker of low EF. Changes in PV also correlated with changes in EF following dobutamine infusion [35].

The ODM readily quantitates the mean acceleration (MA) of blood flow from the beginning of systole to PV, know as the flow time to peak, or FTp (Fig. 1a):

$$MA = \frac{PV}{FTp} \tag{4}$$

Although the ODM does not measure EDV, both PV and MA may be useful as "clinical markers" for contractility. Importantly, these are available on a continuous beat-to-beat basis with the ODM. Nonetheless, both PV and MA are both influenced by changes in volume status and afterload. However, changes in volume status and afterload can be assessed with the ODM on a continuous basis and in real time [36].

The force (F) and kinetic energy (KE) associated with the ejection of the entire stroke volume (SV) from the left ventricle through the aorta, during the period ranging from the beginning of systole until peak flow, can also be readily determined with an ODM. These parameters may also be used in quantitating contractility [32]:

$$F = \rho \cdot (SV) \cdot (MA) = M_{SV} \cdot MA \tag{5}$$

The product of blood density, ρ, and SV is the "stroke mass" (M_{sv}). Thus, kinetic energy (KE) can also be assessed with an ODM:

$$KE = \frac{1}{2}\rho \cdot SV \cdot (PV)^2 = \frac{1}{2} M_{SV} \cdot (PV)^2 \tag{6}$$

Kinetic energy is the work necessary to accelerate the M_{sv} from a velocity of zero to PV. Therefore, a *change* in velocity or acceleration is necessary for KE to exist [37].

Clinical contractility indices may be divided into those that are velocity-based versus those that are acceleration-based [32]. Using mathematical models, it can be demonstrated that acceleration-based indices are more sensitive to changes in contractility than those that are velocity-based. However, it is conceivable that acceleration-based indices are less specific and are influenced more by changes in preload and/or afterload.

In a preliminary clinical examination, KE and F had greater discriminative power than either MA or PV, whereas MA had more discriminative power than PV [32]. Further clinical research is necessary to determine which ODM-based parameter(s) would be the most useful for contractility assessment in HRSPs. Age and gender-appropriate normal value ranges will need to be established. In addition, the effect of afterload on these contractility parameters (ventricle–arterial coupling) needs to be evaluated. This could also be accomplished with an ODM.

4 Assessment of left ventricle afterload

Clinicians have traditionally used an Ohm's law analogy to calculate left ventricle afterload [36]:

$$SVR = \frac{(MAP - CVP)}{CO} \cdot 80 \tag{7}$$

where SVR is systemic vascular resistance, CO is cardiac output, and CVP is central venous pressure. The constant 80 is used for unit correction. MAP represents mean arterial pressure, which is typically defined as:

$$MAP = \frac{2}{3} DBP + \frac{1}{3} SBP \tag{8}$$

This definition is typically utilized with non-invasive blood pressure measurements. MAP could also be calculated with other formulas that include corrections for heart rate, which might be an important factor in critically ill patients [38].

CVP is an invasive measurement and requires cannulation of the internal jugular or subclavian veins. Bleeding, infection, pneumothorax, and dysrhythmias can subsequently occur. Furthermore, CVP values can be erroneous, due to external factors such as the concomitant use of positive end-expiration pressure (PEEP) fluid administration through the catheter during pressure measurements and variations in transducer height (zeroing).

Ignoring the effect of CVP on afterload yields an expression which is referred to as total systemic vascular resistance or TSVR [36]:

$$TSVR = \frac{(MAP)}{CO} \cdot 80 \tag{9}$$

A preliminary clinical investigation has shown that SVR and TSVR strongly correlate [36]. Furthermore, the hour-to-hour changes in TSVR produces values that are in near-agreement with the hour-to-hour changes in SVR.

A significant "flaw" in the Ohm's law analogy is that the haemodynamic effects of **inertia** (L), **resistance** (Rs), and **elastance** (Ea) are not individually determined. Utilizing these parameters, pressure P(t) and flow Q(t) can be represented as [33]:

$$P(t) = L \cdot \dot{Q}(t) + Rs \cdot Q(t) + Ea \cdot SV(t) \quad 0 \le t \le FT \tag{10}$$

Equation (10) expresses the relationship between blood flow and blood pressure *throughout* systole (FT or flow time). It should be noted that:

$$\dot{Q}(t) = \frac{dQ}{dt} \quad \text{and} \quad SV(t) = \int Q(t)dt$$

Note that both $Q(t)$ *and* $SV(t)$ are functions of time. Specifically, $SV(t)$ is the indefinite integral of $Q(t)$.

The combination of L, Rs, and Ea play a "key role" in determining blood pressure during systole. Importantly, specific disease states and drugs may uniquely alter the individual values of L, Rs, or Ea. Specifically, conditions such as hypovolemia or a Valsalva maneuver may decrease L while simultaneously increasing both peripheral Rs and Ea [39].

As an example of how the components of afterload may individually vary, a preliminary retrospective review of vascular research on the brachial artery has demonstrated that vessel diameter and Young's modulus (vessel wall stiffness) may change *independently* of each other under various clinical and pathologic conditions [40]. It would seem reasonable that this could also apply to the arterial system as a whole.

Use of concomitant pressure and flow measurements at diastole ($t = 0$), peak flow ($t = $ FTp), and end-systole ($t = $ FT) yields the following matrix relationship (Fig. 1a, b) [33, 36]:

$$\begin{bmatrix} \dot{Q}(0) & Q(0) & SV(0) \\ \dot{Q}(FTp) & Q(FTp) & SV(FTp) \\ \dot{Q}(FT) & Q(FT) & SV(FT) \end{bmatrix} \cdot \begin{bmatrix} L \\ Rs \\ Ea \end{bmatrix} = \begin{bmatrix} P(0) \\ P(FTp) \\ P(FT) \end{bmatrix} \tag{11}$$

The solution for L, Rs, and Ea is therefore:

$$\begin{bmatrix} L \\ Rs \\ Ea \end{bmatrix} = \begin{bmatrix} \dot{Q}(0) & Q(0) & SV(0) \\ \dot{Q}(FTp) & Q(FTp) & SV(FTp) \\ \dot{Q}(FT) & Q(FT) & SV(FT) \end{bmatrix}^{-1} \cdot \begin{bmatrix} P(0) \\ P(FTp) \\ P(FT) \end{bmatrix} \tag{12}$$

Utilizing this relationship: $Q(t) = A \cdot v(t)$, where A represents the cross-sectional area of the distal thoracic aorta, Eq. (12) yields:

$$\begin{bmatrix} L \\ Rs \\ Ea \end{bmatrix} = \frac{1}{A} \left\{ \begin{bmatrix} \dot{v}(0) & v(0) & SD(0) \\ \dot{v}(FTp) & v(FTp) & SD(FTp) \\ \dot{v}(FT) & v(FT) & SD(FT) \end{bmatrix}^{-1} \cdot \begin{bmatrix} P(0) \\ P(FTp) \\ P(FT) \end{bmatrix} \right\} \tag{13}$$

Similarly, $SV(t) = A \cdot SD(t)$. Note that $v(t)$ represents the velocity of blood flow, $\dot{v}(t)$ represents acceleration and $SD(t)$ is stroke distance. To take into account the quantity of the blood flow which does not reach the distal thoracic aorta the constant k can be utilized. As previously stated, this has a value of approximately 1.4 and is dimensionless:

$$\begin{bmatrix} L \\ Rs \\ Ea \end{bmatrix} = \frac{1}{(k \cdot A)} \left\{ \begin{bmatrix} \dot{v}(0) & v(0) & SD(0) \\ \dot{v}(FTp) & v(FTp) & SD(FTp) \\ \dot{v}(FT) & v(FT) & SD(FT) \end{bmatrix}^{-1} \cdot \begin{bmatrix} P(0) \\ P(FTp) \\ P(FT) \end{bmatrix} \right\} \tag{14}$$

Therefore, by taking advantage of currently available simultaneous arterial pressure and Doppler measurements (Fig. 1c), L, Rs, and Ea could be readily and individually determined on a beat-to-beat basis. Numerical examples, which utilize the above matrix inversion methodology have been examined. Note that this technique is also *independent* of heart rate.

In conclusion, afterload and its underlying parameters could be determined using minimally invasive technologies in real-time. Various medications and pathologic states may also yield individual condition-specific changes in L, Rs, and Ea. Further clinical research is needed to determine the applicability and limitations of this matrix-based technique as well as the use of TSVR as a substitute for SVR.

5 Clinical application and limitations

Low cardiac output is a common problem in HRSPs. Using a simplified yet mechanistic analogy of the heart as an hemodynamic pump, cardiac function depends on the volume arriving to the right atria (venous return and

Table 1 Illustrative examples of two scenarios where integration of information from the oesophageal Doppler could help to establish a pathophysiological-based hypothesis for hypotension

Clinical information	Preload	Contractility	Afterload	Interpretation
63-year-old patient known for ischemic cardiomyopathy and 3rd degree heart block Develops hypotension five hours after on-pump coronary artery bypass grafting	Mean systemic filling pressure → ↑	Kinetic energy↓	Inertia↓ Resistance↑ Elastance↑	Decreased contractility due to myocardial stunning. Consider an inotrope. Assure adequate analgesia, comfort and haemoglobin
85-year-old patient known for dementia and diabetes Develops hypotension during surgery for hip fracture	Mean systemic filling pressure↓	Kinetic energy↓	Inertia↓ Resistance↑ Elastance↑	Bleeding and hypovolaemia. Check coagulation parameters and haemoglobin. Consider crystalloids or blood accordingly

preload), the ventricular capacity for fibber shortening and generation of stroke volume (contractility) and the force needed to overcome and eject this volume into the arterial tree (afterload). The present concept aims to provide, in one device, surrogates of these three parameters that help the clinician to quickly build a hierarchy of pathophysiology-based hypotheses for his observation and guide treatment accordingly. Two illustrative examples are given in Table 1.

The concepts presented here arise from the authors' own clinical experience and scientific work complemented with a non-systematic literature search in our personal libraries and Medline and synthesis of the best available evidence according to our own expertise. Further evidence is needed to support our approach that the integrated use of these markers can translate into improved patient outcomes.

6 Conclusions

The oesophageal Doppler is a minimally invasive monitor utilised in goal-directed protocols of haemodynamic therapy in HRSPs in the surgical theatre and the ICU. However, nowadays, its current use is mainly limited to cardiac output monitoring. The authors believe that integration of information from new measurements of volume status, contractility and afterload could provide a better understanding of the patient's haemodynamic status and assist the clinician in optimising and individualising blood circulation on the basis of robust pathophysiological concepts.

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Compliance with ethical standards

Conflicts of interest BG has performed consultancy work for Edwards Lifescience LCC on behalf of his hospital employer. There are no other conflicts of interest to be declared.

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