Special review

The Use of the Oesophageal Doppler Monitor in the Intensive Care Unit

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ABSTRACT

Objective: *To review the theory and clinical use of the oesophageal Doppler monitor (ODM) in the intensive care setting.*

Data sources: *A literature search using the key-words in both Medline and Pubmed databases. Information concerning insertion techniques and various waveforms was also obtained from the manufacturers.*

Summary of review: *Both clinical and non clinical means of assessing the cardiac output have inherent inaccuracies. Some methods (e.g. the pulmonary artery catheter) are associated with potentially life-threatening complications. The oesophageal Doppler monitor offers a less-invasive, real-time indicator of cardiac output. This review describes the theory of the ODM, the technique for its insertion, the waveforms seen in various pathological states and appraises the available literature on its use.*

Conclusions: *The ODM offers a minimally invasive means of continuous haemodynamic monitoring with an extremely low incidence of complications. It is easy to insert and has been validated against established methods of cardiac output monitoring. However, whilst it has been shown to be of particular benefit in guiding fluid management and peri-operative care, there is less evidence of its usefulness in guiding inotrope requirements. Additionally, any reduction in morbidity and resource consumption has not yet been reported to be associated with an improvement in ICU survival.* (**Critical Care and Resuscitation 2004; 6: 113-122**)

Key words: Oesophageal Doppler, cardiac output, stroke volume, pulmonary artery catheter

The haemodynamic status of the critically ill patient is often monitored using clinical signs (e.g. capillary refill and urine output) and measurements of blood pressure and central venous pressure. Clinical signs however, are often unreliable as demonstrated by Rodriguez and Berumen,[1] who showed that physicians in an emergency department were unable to predict accurately the haemodynamic status of the majority of critically ill patients by clinical signs alone. This is particularly true in the early stages of shock when compensatory mechanisms cloud the clinical picture.[2,3] In 37 young critically ill trauma patients Abou-Khalil *et al*,[4] demonstrated that despite normal heart rate, blood pressure and urine output, invasive haemodynamic monitoring revealed significant myocardial depression requiring inotropic therapy in 32 patients.

If an accurate picture of the patient's haemodynamic status is required, then the pulmonary artery catheter (PAC) is traditionally used. However, over the last 15 years the oesophageal Doppler monitor (ODM) has provided a minimally invasive continuous 'real time' means of monitoring cardiac output that correlates well with conventional thermodilution methods.

The ODM was first described in 1971 by Side and Gosling,[5] and was subsequently modified by Singer *et al* in 1989.[6] It has been available in Australasia since 2000.

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Its advantages include ease of use (taking moments to site) and absence of complications that can be associated with other more invasive methods of determining cardiac output. As the oesophageal Doppler provides a continuous monitor, it allows the effects of fluid and inotropic therapy to be observed immediately, potentially facilitating optimal titration of therapy.

In this article we discuss the principles underlying the oesophageal Doppler monitor, it's advantages compared with other means of obtaining similar information and it's limitations in the critical care setting.

Principles of measurement

The Doppler effect was originally reported by the Austrian mathematician and physicist, Christian Doppler (1803-1853). He described an apparent shift in the frequency of waves (e.g. sound or light) generated by a source that moved relative to an observer. The relationship between the apparent frequency and the actual frequency (i.e. the Doppler shift), is given by the following equation:

$$f_2 = f_1 \times \frac{v}{(v-v_s)}$$

Where f_2 = apparent frequency perceived by the observer,
f_1 = actual frequency emitted by the source,
v = speed of the wave in the medium and
v_s = speed of the source moving through that medium.

A negative sign indicates the source is moving towards the observer whereas a positive sign indicates the source is moving away.

Using a Doppler probe this principle can determine the rate of blood flow in the aorta and thus cardiac output. An ultrasound beam is directed along the path of the flow of blood in the aorta and is reflected off the red blood cells. As these cells are flowing towards or away from the probe, they cause a shift in the frequency of the reflected ultrasound waves that is proportional to the velocity of blood flow. The actual velocity of blood flow can then be determined by using the Doppler equation which incorporates this shift in frequency.

$$V = c \times \frac{f_d}{2f_t \cos \vartheta}$$

Where
V = velocity of blood,
c = speed of sound in body tissue (1540 m/s),
f_d = Doppler frequency shift,
$\cos \vartheta$ = cosine of the angle between the sound beam and the direction of flow, and
f_t = transmitted ultrasound frequency.

The Doppler waveform

Spectral analysis of the Doppler shift gives velocity-time waveforms (figure 1). Since distance travelled is velocity multiplied by time, the area under the curve gives the stroke distance (i.e. the distance a column of blood travels along the aorta during each ventricular systole). The shape of the waveform allows assessment of the left ventricular preload, contractility and after-load. The base of the wave gives the flow time (i.e. the left ventricular ejection time) which is corrected for heart rate to give the corrected flow time (FTc) Figure 2.

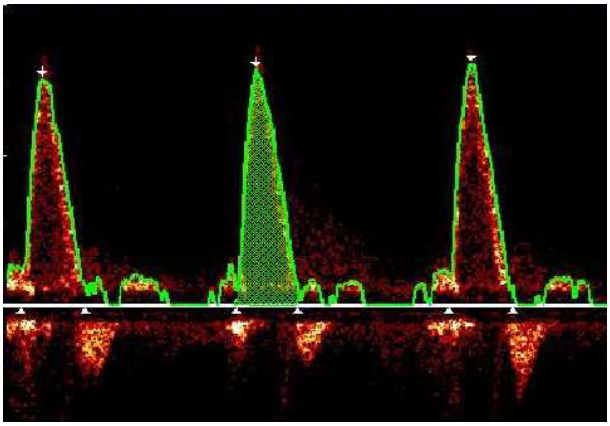

Figure 1. The velocity-time Doppler waveform

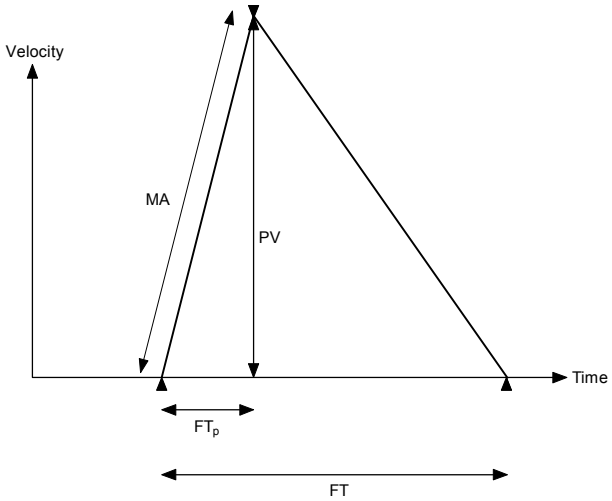

Figure 2. A diagram of the velocity-time Doppler waveform segments (MA = Mean acceleration, PV = Peak velocity, FT = Flow time, FTp = Flow time to peak velocity).

The height of the waveform gives the peak velocity (usually in centimetres per second) and the upstroke of

the wave can provide the mean acceleration (usually in centimetres per second per second). These are the parameters from which the remaining cardiovascular indices, such as stroke volume, cardiac output and systemic vascular resistance, are derived (Table 1).

Table 1. Values calculated from Doppler measurements

Parameter	*Calculation*
Stroke distance	Flow time × velocity
Minute distance	Flow time × velocity × heart rate
Stroke volume	Stroke distance × cross-sectional area of aorta
Cardiac output	Stroke volume × heart rate
Cardiac index	Cardiac output/body surface area
Systemic vascular resistance	(MAP – CVP)/cardiac output

Assumptions

Two assumptions are made when using the ODM to assess cardiac output. Analysis of the Doppler shifts gives information regarding blood flow velocity but in order to determine cardiac output, further information is required as cardiac output = stroke volume (SV) × heart rate. Stroke volume can be calculated by multiplying the stroke distance (SD) by the cross sectional area (π x aortic radius^2) of the aorta (i.e. SV = SD × πr^2). Stroke distance is the distance travelled by a column of blood during ventricular systole and is derived by multiplying the flow velocity (derived from the Doppler equation) by the left ventricular ejection time.

However, the first assumption made when using the Doppler probe is that the cross sectional area of the aorta can be derived from a nomogram based on the age, weight and height of the individual. The second assumption is that a constant percentage of the cardiac output enters the descending aorta, as measurements with the Doppler probe made in the descending aorta exclude coronary and cerebral circulations. Consequently, a correction factor is required to give the overall cardiac output.

However, the proportion of the cardiac output which passes into the descending aorta varies with different disease states. For example, in hypovolaemic shock there will be preservation of blood flow to coronary and the cerebral circulations so a greater proportion of the blood will leave the aorta proximal to the descending aorta. Conversely, with a lower limb regional block, vasodilatation due to loss of sympathetic tone will cause a greater proportion of the blood to be delivered beyond the descending aorta.

Available models

There are currently two versions of the ODM marketed in Australasia: the CardioQ™ (Deltex Medical, Chichester, UK) and the Hemosonic™ (Arrow International, USA). Both have a probe which attaches to a portable monitor, allowing a display of menus, waveforms and derived parameters.

Whilst both models have similar monitors, there are differences between the probes. Additionally, as with different brands of ventilator, there are slightly different names for similar measurements. For example, the CardioQ™ will display the corrected flow time (FTc) whilst the Hemosonic™ equivalent is the LVETc (corrected left ventricular ejection time).

The CardioQ™ (Figure 3)

The disposable CardioQ probe is 6 mm in diameter (i.e. approximately the same diameter as a nasogastric tube). Notches on the probe are placed at 35 and 40 cm from the tip to aid the correct insertion depth. The probe has an internal spring coil to provide an optimum balance between the flexibility and rigidity needed to position the probe. At the tip of the probe the Doppler transducer is angled at 45° and provides a continuous ultrasound frequency of 4 MHz.

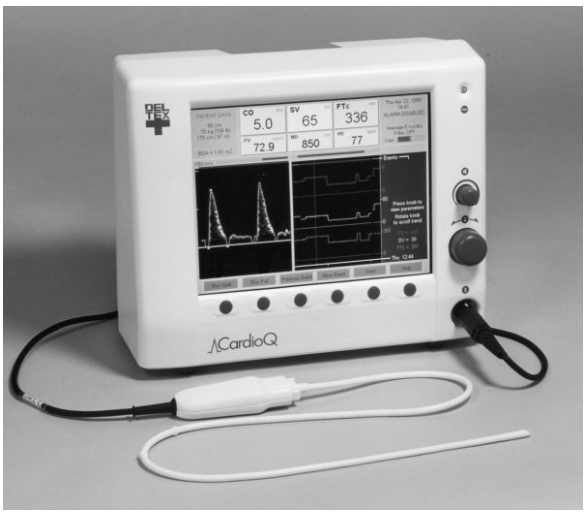

Figure 3. The Deltex CardioQ™ (With permission Deltex Medical Ltd, UK)

The Hemosonic™ (Figure 4)

The probe for the Hemosonic has a handle to control the rotation of the flexible insertion tip. Ring markers are placed along the shaft of the probe to indicate the insertion depth. To provide an M-mode echo probe in addition to the Doppler tranducer, it has a slightly larger diameter (nearer 7 mm) than the CardioQ probe. The M-mode echo serves two purposes: firstly the echo signal

gives an indication of the optimum Doppler probe placement; and secondly it allows for a true estimate of the aortic diameter, rather than relying on a normogram. Once the probe is correctly inserted, the transducers in the tip are rotated via the control handle, until the optimum M-mode signal is obtained.

As expected, from its added complexity, the probe is not a single-use one and is inserted covered by a disposable sterile sheath. This sheath can be left in situ and the probe can be inserted into another patient, allowing several patients to be monitored.

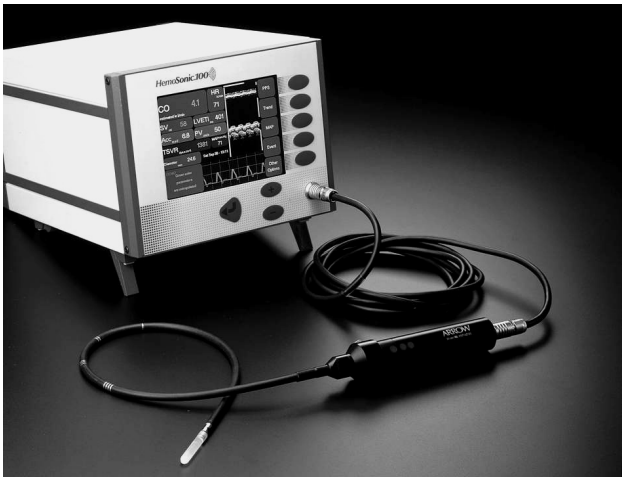

Figure 4. The Arrow Hemosonic™ monitor and probe (With permission Kimal plc, UK)

Insertion of probe

With the Deltex CardioQ™ monitor the probe is easily inserted into the oesophagus via the nasal[7] or oral route, although due to the internal spring coil, it adopts a shape in the pharynx which is not well tolerated by the awake patient. Once in the oesophagus, the volume on the monitor is increased to give an audible cue as to its correct placement. It is then inserted and advanced into the distal oesophagus to reach the T5-6 level (i.e. 35 - 40 cm from the teeth). The probe is then rotated to direct the ultrasound beam posteriorly towards the descending aorta to obtain the optimum waveform (figure 5).

The appearance of a white area on the leading edge of the waveform indicates that the waveform has been optimised. With the Deltex CardioQ™ monitor, a green line will outline the velocity-time envelope of the Doppler waveform. White arrows appear at the beginning, peak and end of the envelope to show the points used to measure the time and velocity values. By viewing the waveforms present on the monitor it is possible to see whether the probe is inserted too far (e.g. a trace typical of the coeliac artery is seen, figure 6), or not far enough (e.g. a pulmonary or azygous trace will be seen, figure 7). Nevertheless, even when the probe is inserted to the correct depth, if it is not rotated in the right direction, the signal recorded could be intra-cardiac (figure 8) or venous (figure 9).

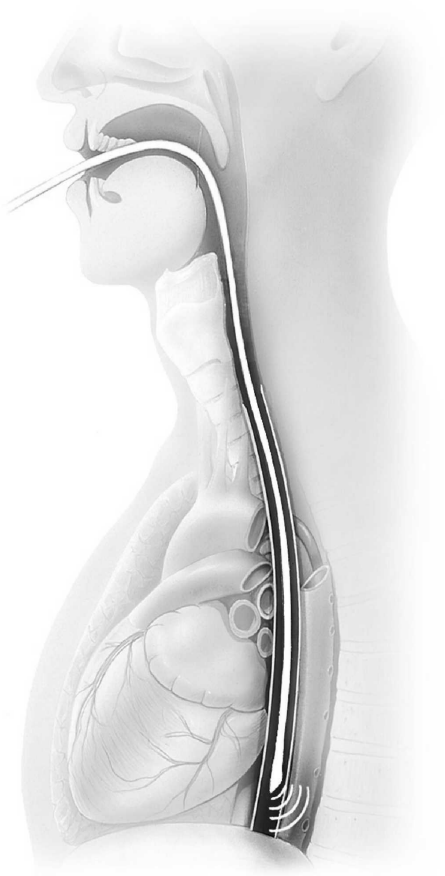

Figure 5. Correct positioning of the oesophageal Doppler probe.

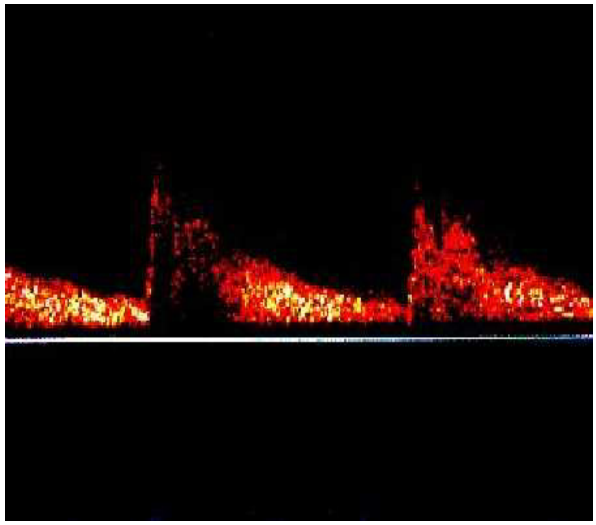

Figure 6. Coeliac artery trace.

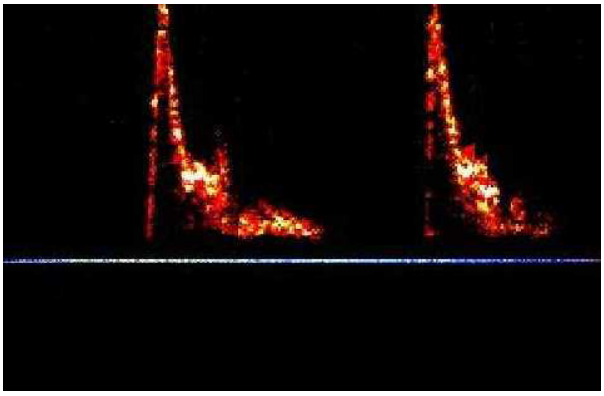

Figure 7. Pulmonary artery trace.

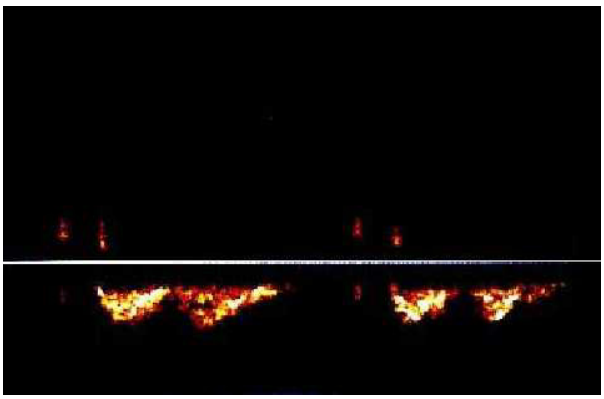

Figure 8. Intra-cardiac trace.

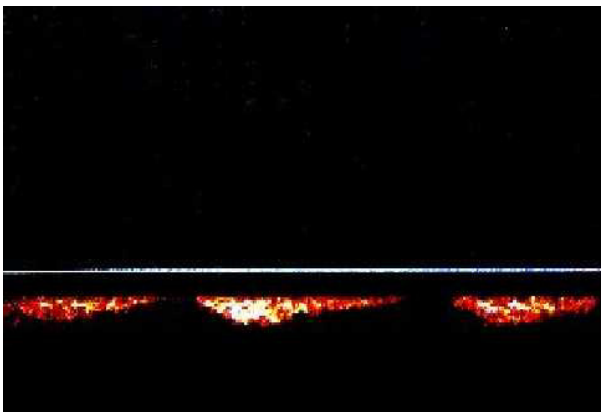

Figure 9. Venous trace.

The oesophageal Doppler is used predominantly in intubated patients with the main problem in its use relating to probe positioning and probe dislodgement. Some operator dependence has also been noted. The presence of a nasogastric tube may prevent the conduction of the Doppler sound-wave into the aorta.

Unlike the PAC, the ODM cannot measure pulmonary artery occlusion pressures or mixed venous oxygen saturations. The presence of an intra-aortic balloon pump or severe aortic coarctation will produce turbulent flow in the descending aorta and excludes it effective use. Other contraindications for ODM use are summarised in table 2.

Table 2. Contraindications to insertion of an oesophageal Doppler monitor probe

Local disease
Oesophageal stent
Carcinoma of the oesophagus or pharynx
Previous oesophageal surgery
Oesophageal stricture
Oesophageal varices
Pharyngeal pouch
Aortic Abnormalities
Intra-aortic balloon pump
Coarctation of the aorta
Systemic
Severe coagulopathy

Information obtained from the Doppler waveform

Normal physiology

The FTc value gives an indication of the left ventricular preload. For a given myocardial contractility and afterload, the stroke volume will increase as the preload increases. Blood is ejected from the left ventricle at a force which can be considered as the force of myocardial contraction (i.e. its contractility) less the resistance in the vascular tree (i.e. the afterload). At a given left ventricular contractility, the lower the resistance, the greater the aortic blood acceleration. This will be reflected in the blood reaching a higher maximum velocity (i.e. a higher peak to the Doppler waveform), and also a higher mean acceleration (i.e. a steeper slope of the waveform).

Peak aortic blood flow acceleration occurs in the first few milliseconds after the aortic valve opens. The systemic resistance to blood flow increases throughout systole. Therefore, the peak acceleration gives a good representation of left ventricular contractility independent of afterload.

As it is extremely difficult to process the Doppler signal at the beginning of systole, instead of measuring the peak acceleration, the mean acceleration is usually taken as an estimate of peak acceleration.

Pathological states

Hypovolaemia. Hypovolaemia is characterised by a waveform with a narrow base (i.e. shortened FTc), but a normal peak velocity (Figure 10).

With constant myocardial contractility, a fall in intravascular volume will result in a smaller stroke volume ejected in a shorter time (Figure 11).

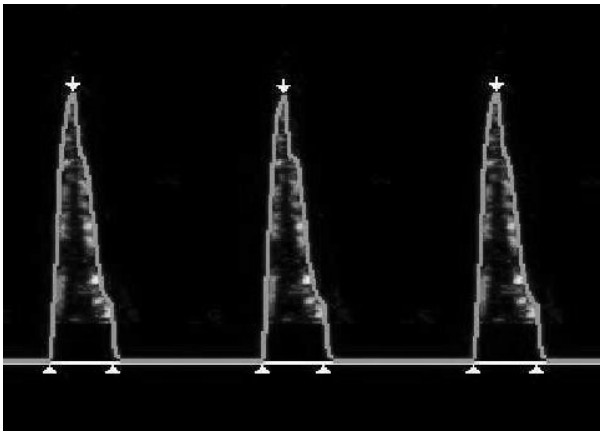

Figure 10. The Doppler waveform in hypovolaemia.

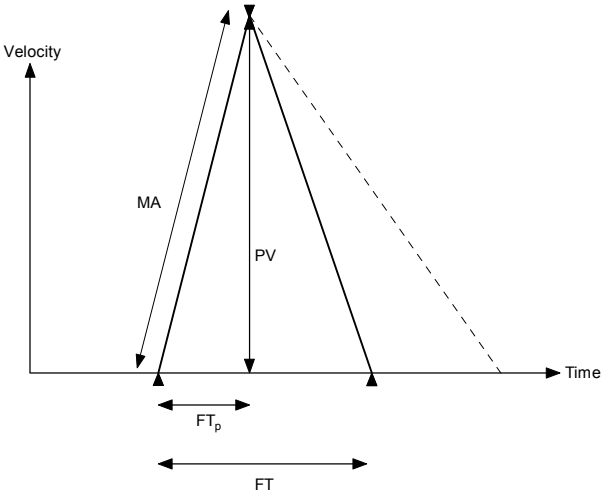

Figure 11. A diagram of the Doppler waveform segments in hypovolaemia (MA = Mean acceleration, PV = Peak velocity, FT = Flow time, FTp = Flow time to peak velocity). Also compare with figure 2.

Left ventricular failure. Left ventricular failure decreases the wave height and reduces the upstroke of the waveform (i.e. a reduction in mean acceleration, Figure 12). If fluid overload exists, there may also be a widened waveform base (> 360msec). The waveform will also be domed-shape rather than triangular (Figure 13).

Vasoconstriction. Vasoconstriction will cause the Doppler waveform to be shorter and narrower (i.e. reduced peak velocity and reduced flow time, Figure 14). At a constant myocardial contractility, an increased systemic vascular resistance results in a lower maximum velocity a faster rise in pressure for a given stroke volume and shortened flow time (Figure 15).

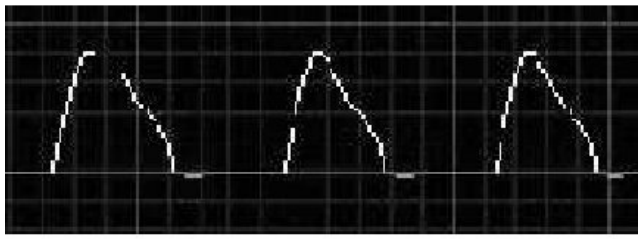

Figure 12. The Doppler waveform in left ventricular failure

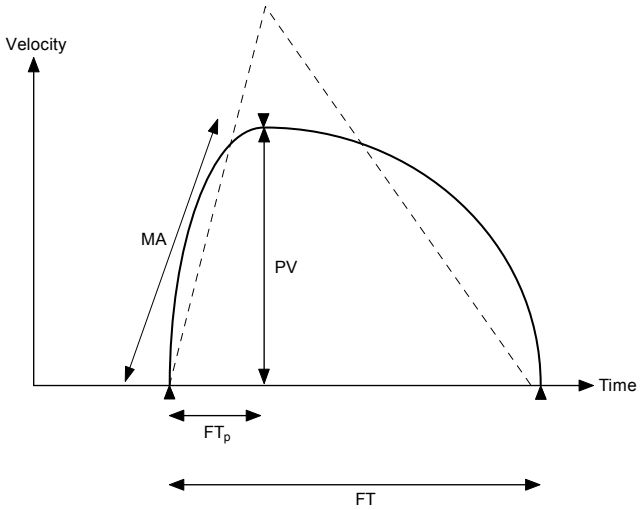

Figure 13. A diagram of the Doppler waveform segments in left ventricular failure (MA = Mean acceleration, PV = Peak velocity, FT = Flow time, FTp = Flow time to peak velocity) compare with figure 2.

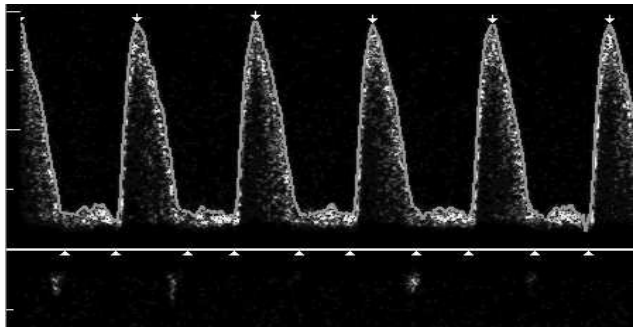

Figure 14. The Doppler waveform in vasoconstriction

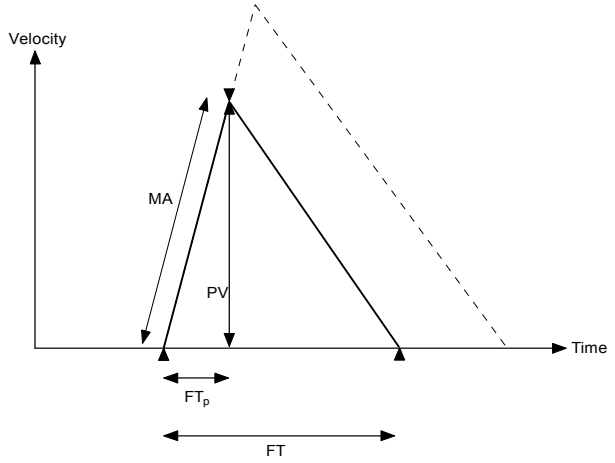

Figure 15. A diagram of the Doppler waveform segments in vasoconstriction (MA = Mean acceleration, PV = Peak velocity, FT = Flow time, FTp = Flow time to peak velocity) compare with figure 2.

The detection and monitoring of trends in hypovolaemia are the specific advantages of the ODM as one can observe the widening of the Doppler waveform after a fluid challenge. Myocardial contractile insufficiency may also be detected indicating a requirement for inotropes rather than vasoconstrictive agents. Nonetheless, in clinical practice the waveform will give little help concerning the decision to administer a vasoconstrictor agent (e.g. noradrenaline) when vasodilatory shock (e.g. septic shock) exists. This decision is often made by a process of elimination by excluding hypovolaemia or left ventricular failure as the cause of shock.

Clinical application of the oesophageal Doppler monitor

For many years the PAC has been used for the purpose of assessing and managing patients with haemodynamic insufficiency. However, its use has been criticised concerning the lack of evidence supporting an outcome benefit,[8,9,10] its complication rate,[11,12,13] measurement errors[14] and misinterpretation of the information obtained.[15,16] Connors *et al*,[8] in 1996 published an observational study of 5735 patients, reporting a risk-adjusted comparison of the outcome of critically ill patients who received or did not receive a PAC within the first 24 hours of admission. Their results strongly suggested that PAC was associated with increased mortality, length of stay and financial costs. Subsequently a Consensus Statement was published identifying the lack of prospective randomised control trials concerning the PAC and acknowledging the well described complications, although it continued to support the use of PAC.[10] Currently a multicentre, open and randomised control trial is being conducted in the UK (the PACMAN trial), to evaluate the clinical and cost effectiveness of PAC in patient management.[17]

In contrast to the PAC, the ODM offers a minimally invasive, continuous 'real time' means of assessing cardiac function. The ODM probe is easy to insert, requires little training, is not associated with any significant complications, may be left in situ for greater than two weeks,[18,19] and has been used in a number of critical care settings including theatre, emergency departments as well as the intensive care unit. A study by Lefrant *et al*, demonstrated that a period of training involving no more than 12 patients is sufficient to ensure reliable ODM measurements of cardiac output.[20] Interobserver variability has been shown to be less than 10%,[21] while intraobserver variability is less at 8%. This compares favourably with thermodilution methods of cardiac output measurement, where a study by Valtier *et al*, [22] recorded a 12% variability.

Validation of the ODM

There are a number of methods of monitoring cardiac output including arterial waveform analysis, suprasternal Doppler, thoracic impedance and modified Fick techniques.[23] However, thermodilution remains a widely accepted standard for the comparison of other techniques.[6] Several studies have found good correlation between the ODM and PAC derived cardiac output measurements[6,20,24,25] even during times of severe haemodynamic instability.[26] In a multicentre study comparing ODM with cardiac output measurements by thermodilution,[22] a good correlation was found between thermodilution and ODM cardiac output (r = 0.95). However, another study comparing PAC and ODM measurements in severely pre-eclamptic women demonstrated that the ODM values for cardiac output were approximately 40% lower than the PAC values.[7] Nonetheless, almost all studies agree that the ODM accurately reflects the direction and magnitude of any change of cardiac output over time.

Assessment of Preload

The pulmonary capillary wedge pressure (PCWP) is often used as a measurement of left ventricular end diastolic pressure (LVEDP) and hence left ventricular end diastolic volume (LVEDV). However, PCWP is often a poor and misleading measurement of left ventricular preload.[27] Many disorders can alter the relationship between PCWP and LVEDP, and there is often no direct relationship between LVEDP and LVEDV due to factors influencing ventricular interdependence and factors altering left ventricular compliance, particularly in the critically ill patient.

In comparison, the corrected flow time (FTc) has been shown to correlate well with preload.[3,6,24,28,29]

Singer and Bennett studied 43 ICU patients or patients undergoing cardiothoracic surgery who had a PAC in situ.[28] They demonstrated good correlation between FTc and preload. When preload was increased from hypovolaemic states, the FTc increased, when preload was decreased due to haemorrhage or the use of nitrates in normovolaemia, the FTc decreased. However, in hypervolaemic patients, administration of intravenous nitrates produced an initial rise in FTc (indicating optimal cardiac output) before a fall in FTc whereas the PCWP only decreased, indicating that FTc is a useful measurement when optimising ventricular preload. Kincaid *et al*, also concluded that FTc was a better indicator of preload than PCWP when resuscitating hypovolaemic trauma patients in shock.[29]

Assessment of contractility

In contrast to the many reports on preload assessment and intravenous fluid management, there appear to be little clinical data on the use of the ODM to guide the administration of inotropes or in the assessment of left ventricular contractility. Nonetheless, in contrast to the PAC the ODM does provide an estimation of contractility by the measurement of peak velocity (PV).

However, in an animal study by Wong *et al*,[30] that compared ODM cardiac output measurements with a reference flow measurement method of transit time ultrasound (TTU), where contractility was increased using dobutamine and decreased with a combination of halothane and sodium thiopental, the ODM changes in cardiac output were sometimes in the opposite direction to the TTU measurements and generally underestimated changes in contractility. Nevertheless, studies of ODM cardiac output by Singer *et al*, have shown consistency in the alteration of waveform shape in both patients and normal subjects with the use of inotropes.[6,31] For example, an infusion of dobutamine in normal subjects increased the PV in a dose dependent manner while esmolol had the opposite effect.

Assessment of afterload

Afterload can be assessed by the shape of the Doppler waveform and pattern recognition, although there are few data reported to substantiate the ODM in accurately assessing afterload. Further to Wong's reports of the validity of the ODM in assessing contractility, afterload was also assessed.[30] An increased afterload was achieved using neosynephrine while a decrease was obtained using sodium nitroprusside. However, results were disappointing with an exaggeration in cardiac output being recorded following a decrease in afterload. Nevertheless, in human studies, Singer *et al*,[31] demonstrated considerable accuracy in ODM parameters when altering afterload using nitroprusside or phenotolamine for high systemic vascular resistance (SVR) or metaraminol as a vasoconstricting agent. Lowering the SVR resulted in an increased stroke distance both by an increase in PV and FTc while increasing the SVR had the opposite effect. In a study on 18 patients undergoing cardiac surgery, ODM was performed to assess the effects on SVR and cardiac output following a bolus of phenylephrine.[32] This study demonstrated a maximal increase in mean arterial pressure (MAP) and SVR and a simultaneous decrease in cardiac output occurring at an average of 42 seconds following the administration of the drug.

Effect on patient outcome

Following the criticism of the PAC, it has become important to establish whether the use of the ODM, or indeed any new technology, reduces morbidity or mortality in acutely ill patients.

Several authors have shown an improved outcome when using ODM perioperatively.[2,33-35] To test the hypothesis that perioperative plasma volume expansion would preserve gut mucosal pH during elective cardiac surgery, Mythen and Webb[33] studied sixty patients who were randomised to either a control group, receiving treatment according to standard practice, or to a protocol group. The protocol group were given repeated boluses of 6% hydroxethyl starch to maximise ODM measured stroke volume (SV). Compared with the control group, the protocol group had a lower incidence of gut hypoperfusion (5% vs 56%, $P < 0.001$), fewer major complications (0 vs 6 patients, $P = 0.01$), shorter mean lengths of ICU stay (1 vs 1.7 days) and shorter overall hospital stay (6.4 vs 10.1 days, $P = 0.011$). In another study by Sinclair *et al*, a similar protocol was used to maintain maximal stroke volume and FTc in a group of patients undergoing proximal femoral fracture repair.[34] All patients had an ODM but were randomised to either a control or protocol group to which the anaesthetist was blinded. The protocol group received significantly more fluid and had a higher mean FTc and cardiac output than the control patients, despite similar heart rates and blood pressures. Postoperative recovery was found to be significantly faster in the protocol group with the median time (i.e. declared fit for discharge) being 10 vs 15 days. Similar results, including decreased admission rates to critical care facilities and reduced hospital stay, have been seen in several other studies.[2,35] In a pilot study of 20 postoperative cardiac surgery patients, Poeze *et al*,[3] used the ODM to predict post operative complications. Initially they found no difference in MAP, central venous pressure, PaO_2:F_IO_2 ratio or core to toe temperature difference in those patients who subsequently developed complications compared with those who did not. However, they found

a significantly lower stroke volume in those patients who went on to develop complications.

Limitations of the ODM

The assumptions made in the measurement of cardiac output are one of the main problems of the ODM. Several authors have found that the ODM measured cardiac output is inaccurate during acute blood loss or haemorrhage.[21,36] It has been postulated that this is due to the varying diameter of the aorta under such conditions which is not accounted for by the nomogram. List *et al*,[37] examined the relationship between MAP and aortic diameter in sheep and altered the MAP from 80 mmHg to 40 mmHg using enflurane, and from 80 mmHg to 175 mmHg using neosynephrine. The cross sectional area of the aorta correlated closely with the MAP (aortic area = 139.1 + (0.765 + MAP)) indicating that concurrent measurement of aortic area may improve the accuracy during hypo or hypertension.

The second assumption made by the ODM (i.e. that a constant proportion of the cardiac output enters the descending aorta) has also contributed to its inaccuracy in certain clinical situations. During aortic cross clamping in aortic reconstructive surgery, poor correlation has been reported between thermodilution and ODM cardiac output.[38] This may be due to redistribution of blood flow between the ascending and descending aorta. Such a mechanism is also thought to be responsible for the overestimation of the cardiac output in the presence of a lumbar epidural.[39]

Many studies of the ODM that have reported a benefit have investigated its use in intra-operative fluid management. In this situation, hypovolaemia is the most common reason for haemodynamic insufficiency. In contrast, during the postoperative period in the intensive care unit, a systemic inflammatory response will result in relatively more complex mechanisms for hypoperfusion.

Conclusions

The ODM offers a minimally invasive means of continuous haemodynamic monitoring with an extremely low incidence of complications. It has been validated against established methods of cardiac output monitoring such as thermodilution and the modified Fick principle. The ODM probe is easy to insert, requiring only a short period of training for both correct insertion and interpretation. However, whilst it has been shown to be of particular benefit in guiding fluid management in the perioperative period, there is less evidence of its usefulness in guiding inotrope requirements in the critically ill patient. Additionally, any reported reductions in morbidity and resource consumption have not been shown to be associated with an improved ICU survival.

Moreover, due to the assumptions inherent in its operation, care should be taken in certain clinical situations. In particular, it is not clear if it is accurate enough to justify the reduced complications from its use compared with the PAC. Hopefully, further clinical trials will clarify the role of the ODM in the care of the critically ill patient, especially those who require inotropic therapy.

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