Oesophageal Doppler monitoring overestimates cardiac output during lumbar epidural anaesthesia

H. A. Leather and P. F. Wouters*

Department of Anaesthesiology, University Hospitals, Katholieke Universiteit Leuven, U. Z. Gasthuisberg, Herestraat 49, B-3000 Leuven, Belgium

*Corresponding author

Oesophageal Doppler monitoring (ODM) has been advocated as a non-invasive means of measuring cardiac output (CO). However, its reliance upon blood flow measurement in the descending aorta to estimate CO is susceptible to error if blood flow is redistributed between the upper and lower body. We hypothesize that lumbar epidural anesthesia (LEA), which causes blood flow redistribution, causes errors in CO estimates. We compared ODM with thermodilution (TD) measurements in fourteen patients under general anaesthesia for radical prostatectomy, who had received an epidural catheter at the intervertebral level L2–L3. Coupled measurements of CO by means of the TD and ODM techniques were performed at baseline (general anaesthetic only) and after epidural administration of 10 ml of 0.25% bupivacaine. The two methods were compared using Bland-Altman analysis: before LEA there was a bias of −0.89 litre min⁻¹ with limits of agreement ranging between −2.67 and +0.88 litre min⁻¹. Following lumbar sympathetic block, bias became positive (+0.55 litre min⁻¹) and limits of agreement increased to −3.21 and +4.30 litre min⁻¹. ODM measured a greater increase in CO after LEA (Δ=+1.71 (1.19) litre min⁻¹ (mean (SD)) compared with TD (Δ=+0.51 (0.70) litre min⁻¹). We conclude that following LEA, measurements with the Oesophageal Doppler Monitor II overestimate CO and show unacceptably high variability. Blood flow redistribution may limit the value of ODM.

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The accuracy of oesophageal Doppler monitoring (ODM) of cardiac output (CO) is controversial. Some authors have claimed it to be at least as accurate as thermodilution (TD). Others have shown that ODM is less accurate than the TD technique, which is the standard clinical method. The developers of commercially available ODM equipment agree that it gives less accurate absolute measurements than TD, and that the clinician using it should concentrate on trends in CO rather than absolute values. Several of the validation publications of the ODM machines show bias plots of relative change but not absolute values. Theoretically, ODM should be a relatively non-invasive, cheap and easy means of measuring CO. The technique is easier to master, less expensive and less invasive than the use of a TD catheter, but, training is required to best use the ODM technique. Variations in cross-sectional area of the descending aorta and the angle between the probe and the descending aorta may not be taken into account. The ODM II machine (Abbot Laboratories, North Chicago, IL, USA) uses a nomogram to estimate CO from Doppler flow measurements in the descending aorta. To calculate CO, the oesophageal Doppler machine has to take into account the blood flow that it does not measure (i.e. flow to the upper body), and assumes that 30% of CO goes to the upper body. Variations in blood flow distribution may, therefore, lead to errors in CO estimates. Situations involving blood flow redistribution where ODM is inaccurate include haemorrhage and aortic cross-clamping. Lumbar epidural anaesthesia (LEA) increases blood flow to the lower body by sympathetic nerve block, and reduces flow to the upper body by compensatory vasoconstriction caused by increased sympathetic nerve activity. The ODM II machine could overestimate CO in these circumstances.

We measured the accuracy of CO estimation using ODM during LEA as an example of a clinical setting involving
redistribution of blood flow between the upper and lower body.

Methods

The study was approved by the ethical committee of the University Hospitals of the Katholieke Universiteit Leuven. Informed consent was obtained from participating patients.

Fourteen male patients (mean age 65 yr (range 58–73), mean weight 80 kg (range 61–110), mean height 176 cm (range 172–184), ASA class I or II), all undergoing radical prostatectomy under general anaesthesia, were included in the study. A history of severe cardiac disease, respiratory or oesophageal pathology were exclusion criteria. The oesophageal Doppler monitor used for this study (ODM II) emits a continuous 4 MHz ultrasound signal, which is received through a 300 Hz high-pass filter. The blood flow velocity curve is displayed continuously and an on-line display of CO, heart rate and stroke volume is provided.

For the purpose of intra- and post-operative analgesia, a lumbar epidural catheter was inserted at the level of intervertebral space L2–L3 before induction of anaesthesia. Anaesthesia was induced with propofol 1.5–2 mg kg\(^{-1}\), vecuronium 0.1 mg kg\(^{-1}\) and sufentanil 0.2–0.3 \(\mu\)g kg\(^{-1}\), and maintained by means of isoflurane 1.2–1.5 MAC and 65% nitrous oxide with supplemental sufentanil. Following orotracheal intubation, the lungs were mechanically ventilated with a mixture of 35% oxygen and nitrous oxide. A 20-gauge catheter was inserted in the radial artery and a TD catheter was introduced via the right internal jugular vein.

The oesophageal probe was inserted in the standard manner by experienced personnel; optimal positioning was obtained from the appearance and sound of the Doppler signal. Patient age and weight were entered into the ODM console nomogram.

Coupled measurements of CO by TD (triplicate measurements with ice-cold saline) and ODM techniques were performed three times at fixed intervals during the general anaesthetic and then 20, 25 and 30 min after injection of 10 ml bupivacaine 0.25%. Arterial pressure, pulmonary artery pressure, central venous pressure and pulmonary capillary wedge pressure were recorded immediately after each CO determination. An i.v. infusion of 500 ml of a colloid solution (Geloplasma\(^\text{®}\)) was then administered over 30 min in order to prevent severe arterial hypotension.

CO measurements with ODM and TD were compared using Bland-Altman analysis. Paired \(t\)-tests were used to test the significance of changes in haemodynamic parameters after epidural anaesthesia. A probability of less than 0.05 was considered statistically significant to reject the null hypothesis.

Results

Upon awakening, all patients were free of pain and had loss of temperature sensation in the corresponding dermatomes.

The epidural anaesthesia reduced arterial pressure (mean arterial pressure from (mean (SD) 74 (12) (range 61–97) to 63 (10) (range 50–79) mm Hg) and central venous filling pressure (from 11 (3) (range 6–17) to 10 (4) (range 4–17) mm Hg) as measured at the moment of peak CO (TD method). Heart rate, pulmonary artery pressure and pulmonary capillary wedge pressure did not change (Table 1).

Measurements after LEA remained stable over the 10-min observation period (no significant difference between variables obtained 20 and 30 min after the onset of LEA respectively; paired \(t\)-test).

Before LEA, TD and ODM techniques showed acceptable agreement (mean difference –0.89 litre min\(^{-1}\), limits of agreement –2.67 and +0.88 litre min\(^{-1}\); SE of the mean difference=0.95, SE of the limits of agreement=0.23 (Figs 1 and 2A)). Both ODM and TD CO increased after LEA. However, the increase measured by ODM (from 4.84 (1.26)

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<tr>
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<th>Baseline</th>
<th>Post-epidural</th>
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<tr>
<td>ABPs</td>
<td>104 (17) [88–144]</td>
<td>90* (15) [74–115]</td>
</tr>
<tr>
<td>ABPd</td>
<td>58 (10) [43–74]</td>
<td>49* (8) [39–61]</td>
</tr>
<tr>
<td>ABPm</td>
<td>74 (12) [61–97]</td>
<td>63* (10) [50–79]</td>
</tr>
<tr>
<td>CVP</td>
<td>11 (3) [6–17]</td>
<td>10* (4) [4–17]</td>
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<tr>
<td>MPAP</td>
<td>21 (4) [14–28]</td>
<td>21 (5) [14–27]</td>
</tr>
<tr>
<td>PCWP</td>
<td>14 (3) [7–17]</td>
<td>13 (3) [8–19]</td>
</tr>
<tr>
<td>HR</td>
<td>62 (10) [51–80]</td>
<td>66 (12) [52–88]</td>
</tr>
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Table 1 Haemodynamic data before and after LEA. ABPs, d, m=systolic, diastolic and mean arterial pressure, CVP=central venous pressure, MPAP=mean pulmonary artery pressure, PCWP=pulmonary capillary wedge pressure, HR=heart rate. Data are mean (SD) [range]. Pressures are in mm Hg, heart rate is in beats min\(^{-1}\), *=P<0.05 vs control. Data displayed post-epidural are obtained from the measurement coinciding with peak CO as measured with TD.
to 6.54 (1.99) litre min⁻¹; $\Delta = +1.71$ (1.19) litre min⁻¹) was greater than that measured using the TD technique (from 5.60 (1.16) to 6.11 (1.10) litre min⁻¹; $\Delta = +0.51$ (0.70) litre min⁻¹; $P < 0.05$). During LEA, in contrast to baseline circumstances, ODM overestimated CO (mean difference $+0.55$ litre min⁻¹, SE of the mean difference=$0.29$) and displayed unacceptably wide limits of agreement (limits of agreement $-3.21$ and $+4.30$ litre min⁻¹, SE of the limits of agreement=$0.50$) (Figs 1 and 2B).

**Discussion**

We observed an acceptable agreement between ODM and TD in baseline conditions, with a negative bias. This slight underestimation of CO by ODM has been reported previously but not consistently. Compared with several other studies, we found similar limits of agreement.

LEA increased CO as measured by both TD and ODM, explained by a reduction in systemic vascular resistance and maintenance of an adequate preload by volume infusion.

The main result of this study is that ODM overestimates CO during LEA. There is no theoretical reason to believe that lack of agreement between ODM and TD measurements during epidural anaesthesia should be caused by an underestimation of CO by TD. LEA causes blood pooling in the capacitance vessels of the lower limbs, but this should not affect the accuracy of the TD method which measures blood flow in the pulmonary circulation.

Movement artefacts are unlikely to cause error considering the type of surgery involved. Variation in cross-sectional area of the aorta has been suggested as an important potential cause of inaccuracy of ODM. Considering our patient characteristics, errors caused by aortic compliance are unlikely. A decrease in area of the descending aorta could theoretically overestimate CO. The decrease in arterial pressure which we observed could have caused a small decrease in aortic area, but it is very unlikely that it should have caused a 4-fold decrease needed if this were to be the sole cause of error.

Overestimation by ODM is probably caused by the redistribution of blood to the lower body, at the expense of the upper body segment. The fixed distribution assumed by the ODM II suggests that the machine cannot compensate for such variations in blood flow distribution. ODM has been found inaccurate in haemorrhage and aortic cross-clamping where redistribution is likely. Klotz and co-workers observed an increase in bias between TD and ODM from $-0.96$ to $-1.51$ litre min⁻¹ during aortic cross-clamping in six patients. Underestimation of CO by ODM during aortic cross-clamping can be explained by blood flow redistribution from the lower to the upper body.

The new generation of ODM devices (such as the Hemosonic 100, Arrow) measure aortic diameter. Although this may assess any possible inaccuracy of ODM caused by variations in aortic CSA, it will not solve the problem of blood flow redistribution between the upper and lower body.

We propose that the blood flow redistribution in circumstances like LEA, aortic cross-clamping, haemorrhagic and distributive shock can partially explain the inaccuracy of ODM CO measurements, and caution is necessary when using ODM to estimate CO.

**References**

4. Schmid ER, Spahn DR, Tornic M. Reliability of a new generation
Oesophageal Doppler and epidural anaesthesia

transoesophageal Doppler device for cardiac output monitoring. Anesth Analg 1993; 77: 971–9
9 Singer M, Bennett D. Optimisation of positive end expiratory pressure for maximal delivery of oxygen to tissues using oesophageal Doppler ultrasonography. BMJ 1989; 298: 1350–3
21 Wright PMC, Fee JPH. Cardiovascular support during combined extradural and general anaesthesia. Br J Anaesth 1992; 68: 585–9