Continuous measurement of cardiac output during aortic cross-clamping by the oesophageal Doppler monitor ODM 1

K.-F. KLOTZ, S. KLINGSIEK, M. SINGER, H. WENK, S. ELEFTHERIADIS, H. KUPPE AND P. SCHMUCKER

Summary
We have compared the Doppler against the thermodilution technique for measurement of cardiac output in six patients during aortic surgery. The correlation coefficient between the two methods was between 0.76 and 0.84 during the different periods of the operation. Using the integral nomogram instead of direct calibration, the Doppler system underestimated cardiac output in atherosclerotic patients. However, the Doppler method did register accurately significant changes in cardiac output.

Key words
Measurement techniques, cardiac output. Measurement techniques, Doppler echocardiography.

Patients requiring surgery on the aorta are frequently affected by degenerative vascular disease and are elderly [1]. As they are recognized to be at high risk of circulatory decompensation [2, 3], a high level of haemodynamic monitoring is recommended [2], including frequent evaluation of cardiac output [4, 5]. In addition to thermodilution (TD) there are other methods which measure cardiac output, such as transtracheal [6] or suprasternal [7] Doppler of aortic blood flow, or thoracic bioimpedance [8, 9]. Oesophageal Doppler monitoring (DOP) of cardiac output is a safe, non-invasive method [10] which allows continuous measurement and provides additional information on circulatory state [11]. Easy handling and successful use are described, even in elderly patients [12]. It has also been shown to provide a good indication of alteration in cardiac output caused by changes in the level of PEEP [13] or intravascular volume shifts during transurethral prostatectomy [14]. However, it is not clear if the particular oesophageal Doppler device used in the above studies [10-14] is appropriate in situations considered difficult by authors using earlier devices of different design [15, 16]. One such situation is during the clamping phase of aortic surgery.

Patients and methods
After approval by the Ethics Committee of the Medical University of Lubeck, we studied six patients (table 1) who gave written, informed consent. Patients were excluded if they had known cardiac valve defects or any history of oesophageal disorder. Despite the small number of patients, a wide range of age, height, weight and body surface area was covered. All patients (two female, four male), ASA II and III, were undergoing reconstructive surgery because of atherosclerotic disease of the abdominal aorta and common iliac arteries. Past medical history included coronary artery disease, previous myocardial infarction and peripheral vascular disease. No patient with diabetes mellitus was included in the study. All were scheduled for replacement of the diseased vessel segments by a Y-shaped prosthesis via an open transabdominal approach using a median laparotomy. The aorta was exposed up to the left renal vein and down to the iliac vessels as far as necessary. In all cases it was possible to clamp distal to the renal artery. Heparin 5000 u. was given i.v. for anticoagulation. A proximal end-to-end anastomosis was performed using either a Dacron or PTFE-graft, usually of diameter 16 mm. In case of arterial occlusive disease a proximal end to side anastomosis was preferred.

The method of anaesthesia and perioperative monitoring is shown in table 2. Anaesthesia was given by an independent anaesthetist who was not involved in the study. Routine premedication with flunitrazepam 1.0 mg was given 1 h before surgery. On arrival in the operating theatre, peripheral i.v. and radial arterial cannulae were inserted. Fluid

| Table 1 | Patient characteristics. BSA = Body surface area |
|---|---|---|---|---|
| Age (years) | Height (cm) | Weight (kg) | BSA (m²) |
| Mean | 66.2 | 171.3 | 71.3 | 1.8 |
| SD | — | 8.6 | 15.1 | 0.2 |
| Median | 65.5 | 173 | 75.5 | 1.915 |
| Max. | 75 | 180 | 88 | 2.03 |
| Min. | 55 | 156 | 50 | 1.35 |

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management was guided by cardiac filling pressures and consisted of infusions of electrolyte solutions and plasma expanders (hydroxyethyl starch and serum albumin solutions).

For monitoring purposes a central venous and a 7-French gauge flow-directed pulmonary artery catheters (93A-131-7F, Baxter Edwards Laboratories, Irvine, CA, USA) were inserted via the right internal jugular vein. Ventilator settings were adjusted according to data obtained from repeated arterial blood-gas samples.

METHODS OF HAEMODYNAMIC MONITORING
Continuous monitoring of the various blood pressures was performed with the mid-heart level taken as the reference point. The rate–pressure product was calculated by multiplication of systolic arterial pressure by heart rate. All data were obtained using the Sirecust 1281 monitor (Siemens, Erlangen, Germany).

THERMODILUTION MEASUREMENTS
Cardiac output was measured intermittently by injection of 10 ml of ice-cold saline (0–5 °C) using the Sirecust 1281 haemodynamic monitoring computer. All measurements were distributed randomly over the whole respiratory cycle. Cardiac output was taken as the mean of three measurements showing close agreement. However, for comparative purposes, all single injectate values were taken into account.

OESOPHAGEAL DOPPLER MONITOR FOR CONTINUOUS CARDIAC OUTPUT MEASUREMENT
The DOP measurements were performed by an anaesthetist not involved in the anaesthetic management of the patient. After tracheal intubation a Doppler probe of 7 mm in diameter was inserted via the mouth into the oesophagus. The probe was connected to the Doptek ODM 1 Computer (Deltex Ltd, Chichester, West Sussex, UK). This device uses 4-MHz continuous wave Doppler ultrasound and presents both an acoustic signal and a visual spectral analysis of the Doppler frequency shifts on a real-time colour display. This dual output allows, after a short learning period, easy manipulation of the Doppler probe into an appropriate position in the oesophagus to obtain clear Doppler signals of blood flow in the descending thoracic aorta. The monitor performs online estimations of total cardiac output, either by primary calibration of the monitor using another evaluation method, for example, TD, or by using a fixed stored nomogram from manually imported biometric data, including age, sex, body weight and height.

Measurement of absolute left ventricular output is correct only when using an appropriate nomogram, and assuming a constant aortic cross-sectional area during systole, and a constant distribution of cardiac output between the vessels originating from the aortic arch and the remainder which constitutes flow in the descending aorta. These assumptions may be incorrect in patients with aortic degenerative disease as the diameter of the descending aorta may be aneurysmatically increased or degeneratively decreased. The relative distribution of blood flow between the supra-aortic vessels and the descending aorta may be changed, especially during aortic cross-clamping. These potential problems thus formed the basis of this study. The technique and handling of the probe is described in detail elsewhere [12]. We used the nomogram method for measurement of total cardiac output to allow determination of the validity of this particular device. Cardiac output was measured on a beat-to-beat basis.

EXPERIMENTAL DESIGN
All data from both Sirecust and ODM 1 monitors were recorded onto videotape for offline evaluation. For each determination of cardiac output by TD, a corresponding value for DOP was made by averaging the individual beats over the approximate 10 s needed for TD measurements. The use of single injectate TD measurements for comparison was necessary because of the rapid changes in cardiac output during aortic surgery. The period of surgical clamping and unclamping was analysed closely. From 1 min before to 5 min after these procedures all haemodynamic data were noted on a second-by-second basis.

STATISTICAL ANALYSIS
The paired values obtained by TD and DOP were divided into three groups, namely the period before clamping of the aorta (pre-clamp), the period with the aorta completely occluded by the clamp (clamp), and then following removal of the aortic clamp (post-clamp).

Haemodynamic data from each patient were averaged to obtain a single mean value for each period. Data from all patients were used to compute the mean (sd). Cardiac output data were compared by regression analysis and the correlation coefficient (r) computed. Bias and precision of DOP compared with TD were computed by the Bland–Altman
technique. Bias indicates the mean deviation of DOP from TD whereas precision shows the distribution of the DOP values around the mean DOP value. All consecutive TD measurements, where an increase or decrease of more than 2 litre min⁻¹ occurred, were included in a further evaluation which included correlation and regression analysis. This was performed to verify consistency in the direction of change in cardiac output measured by the two techniques.

Results

Haemodynamic data are shown in table 3. There were no statistically significant differences in any value between the three periods, although rate-pressure product, DOP, stroke volume, mean arterial pressure and mean pulmonary artery pressure decreased during cross-clamping while systemic vascular resistance increased slightly. All variables returned to pre-clamp values after unclamping of the aorta.

It was possible to measure cardiac output in all patients at all attempts and it proved very easy to obtain adequate Doppler signals. In figure 1 the data of 55 measurements of cardiac output obtained during the pre-clamping period by TD are plotted against the simultaneous DOP values. The correlation coefficient was 0.84 and the regression line followed the equation $DOP = 0.91 \times (TD) - 0.38$. The complete results of the statistical evaluation of all three periods are shown in table 4.

For comparison of 15 large (more than 2 litre min⁻¹) differences between consecutive measurements of TD with corresponding DOP values, measurements from all surgical periods were included in the evaluation. The values correlated with a correlation coefficient of $r = 0.89$. The regression equation was delta DOP = 0.69 Δ TD + 0.53.

Discussion

Castor and colleagues [17] simultaneously compared three methods of evaluating cardiac output, namely TD, thoracic electrical bioimpedance and aortic Doppler ultrasound by the suprasternal approach. Whereas bioimpedance and TD showed a good correlation, the Doppler device differed significantly. This was caused mostly by problems with the handling of the suprasternal Doppler probe. Wong and colleagues [18] measured cardiac output with an oesophageal Doppler flow probe in pigs. Their results were unsatisfactory although this may be related to both the device used and the increased aortic wall elasticity in the young animals monitored. Siegel, Fitzgerald and Engstrom [6] compared transtracheal Doppler with TD and found a correlation coefficient of 0.63. This was inferior to our results with the Doptek oesophageal Doppler system which we feel solves many of the methodological problems associated with the oesophageal approach. The good auditory and visible display of the ultrasound signal helps considerably in determining correct probe placement, a feature lacking in all other machines.

It is noteworthy that we did not calibrate the DOP measurements at any time during the study and that we compared the DOP values with single injectate measurements of cardiac output by the TD technique. The inherent bias and variation of the TD method, which may be as high as 50% [19], is included in all the correlation data presented. This variation may adversely affect the comparison with DOP.

During the pre-clamp period, cardiac outputs measured by DOP correlated well with that obtained by TD. Our correlation coefficient of 0.84 is comparable with the values indicated by other authors [15] for more complicated methods. The pre-clamp bias of 0.96 litre min⁻¹ may be related to the integral stored nomogram for estimation of aortic diameter which might not be applicable in patients with severe atherosclerotic disease of the aorta. The DOP measurements were, on average, approximately 20% lower; this may result from underestimation of the greater aortic diameter in this particular group of patients because of pathological widening. However, as we studied only six such patients, more data are required before recommending any change in the integral nomogram.

During aortic cross-clamping, the correlation coefficient of 0.79 was similar to the pre-clamp period and sufficiently accurate, although the bias did increase to 1.51 litre min⁻¹. Theoretically, this may be caused by aortic dilatation induced by the clamp and also redistribution of blood flow. The diameter of the aorta may possibly be influenced by mean arterial pressure although this is more likely to affect the elastic aorta of the young compared with the elderly patient whose aorta is far more rigid [20, 21]. We observed a slight decrease in mean arterial pressure from 85.4 to 79.0 mm Hg, thus pressure-related changes in aortic diameter would be small and tend to allow dilatation rather than constriction if they occurred at all.

The ODM 1 measurements are based on a fixed blood flow distribution of 30% to the supra-aortic vascular structures and 70% to the descending aorta. This distribution is assumed by the device to be constant. However, during cross-clamping there may be redistribution to the supra-aortic regions. This leads to underestimation of the complete cardiac output by the DOP method, as seen in our study.

After declamping of the aorta, the correlation between DOP and TD in our study decreased to 0.74 whereas Perrino, Fleming and LaMantia [15] found a correlation coefficient of 0.85. However, the precision of their measurements was decreased compared with ours. Gelman and colleagues [22] investigated blood flow distribution and metabolic changes produced by declamping of the aorta. There seemed to be a significant release of vasoactive mediators from the intermittently malperfused tissues to the systemic circulation with a complex vascular and therapeutic response. This may possibly lead to redistribution of blood flow to the upper and lower body, although this hypothesis required confirmation.

The advocates of the DOP method claim that this technique does not indicate "true" cardiac output,
Table 3  Haemodynamic data during the three surgical periods (mean (SD)). HR = Heart rate, MAP = mean arterial pressure, MPAP = mean pulmonary arterial pressure, SV = stroke volume, DOP = Doppler (ODM 1) cardiac output, SVR = systemic vascular resistance, RPP = rate-pressure product

<table>
<thead>
<tr>
<th></th>
<th>Pre-clamp</th>
<th>Clamp</th>
<th>Post-clamp</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beat min⁻¹)</td>
<td>85.2 (23.6)</td>
<td>82.7 (22.6)</td>
<td>82.9 (18.5)</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>85.4 (15.4)</td>
<td>79.0 (11.5)</td>
<td>88.7 (13.8)</td>
</tr>
<tr>
<td>MPAP (mm Hg)</td>
<td>21.6 (4.9)</td>
<td>17.9 (5.3)</td>
<td>24.0 (5.8)</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>55.5 (20.1)</td>
<td>44.9 (19.4)</td>
<td>60.6 (15.2)</td>
</tr>
<tr>
<td>DOP (litre min⁻¹)</td>
<td>4.73 (2.22)</td>
<td>3.64 (1.82)</td>
<td>5.01 (1.86)</td>
</tr>
<tr>
<td>SVR (dyn cm⁻⁵ s⁻¹)</td>
<td>1394 (412)</td>
<td>1739 (571)</td>
<td>1290 (269)</td>
</tr>
<tr>
<td>RPP</td>
<td>10693 (3811)</td>
<td>9384 (2583)</td>
<td>10582 (2623)</td>
</tr>
</tbody>
</table>

but rather a reasonable estimate. However, it does provide a good qualitative guide to changes [18]. We found that combining the results of the correlation tests, a very high r value of 0.89 was obtained for cardiac output changes. All marked changes in measurements of cardiac output by TD were accompanied by appropriate changes in DOP measurements. This was not seen in the study performed by Wong and colleagues [18] who found that 5–7% of changes occurred in the opposite direction. However, in our study we selected only paired measurements where there were large changes in cardiac output measured by TD. This excludes “false small differences” caused by the variability of the TD technique; a 14% change in cardiac output measured by TD is needed to be confident that this is a true change [23].

MECHANISMS OF COMPENSATION OF THE UNCLAMPING MANOEUVRE

The advantage of the DOP method for monitoring patients during reconstructive surgery on the aorta is that we may be able to rapidly identify deterioration and initiate therapy. Bush and colleagues [24] described a method of preventing hypotension after declamping of the aorta by optimal volume loading. This loading may not be guided directly by the filling pressure before the declamping procedure because there is often no change in left ventricular filling pressure, even with marked changes in left ventricular end-diastolic volume loading [22, 25]. Gregoretti and colleagues [26] showed that healthy animals responded to the release of aortic blood flow with a marked change in cardiac output. This is in contrast with our findings in patients and may be caused by the differences in elasticity and compliance of the arterial system. To illustrate the difference in the cardiovascular behaviour and the possibilities of rapid diagnosis, the original haemodynamic recordings are shown from two of our six patients. Figure 2 shows the change in mean arterial pressure during declamping and this was similar in both patients. However, as shown by the DOP measurements given in figure 3, there was marked difference in the cardiac output response; the cardiac output of patient A increased immediately after release of the cross-clamp, whereas in patient B cardiac output, after a small and short-lived elevation, stayed at the same level. The reason for this difference could be because of the different

Table 4  Results of the statistical evaluation of the comparative measurements during the three different surgical periods

<table>
<thead>
<tr>
<th>Period</th>
<th>n</th>
<th>Correlation coefficient</th>
<th>Regression equation</th>
<th>Bias (litre min⁻¹) (mean DOP – mean TD)</th>
<th>Precision (litre min⁻¹) (95% confidential intervals)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-clamp</td>
<td>55</td>
<td>0.84</td>
<td>DOP = 0.91 x (TD) - 0.38</td>
<td>-0.96</td>
<td>1.33 to -3.24</td>
</tr>
<tr>
<td>Clamp</td>
<td>75</td>
<td>0.79</td>
<td>DOP = 0.74 x (TD) - 0.14</td>
<td>-1.51</td>
<td>-0.02 to -2.99</td>
</tr>
<tr>
<td>Post-clamp</td>
<td>65</td>
<td>0.76</td>
<td>DOP = 0.43 x (TD) + 1.75</td>
<td>-1.47</td>
<td>0.86 to -3.79</td>
</tr>
</tbody>
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References


