CME Perioperative Hemodynamic Monitoring with Transesophageal Doppler Technology

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Invasive cardiac output (CO) monitoring, traditionally performed with transpulmonary thermodilution techniques, is usually reserved for high-risk patients because of the inherent risks of these methods. In contrast, transesophageal Doppler (TED) technology offers a safe, quick, and less invasive method for routine measurements of CO. After esophageal insertion and focusing of the probe, the Doppler beam interrogates the descending aortic blood flow. On the basis of the measured frequency shift between the emitted and received ultrasound frequency, blood flow velocity is determined. From this velocity, combined with the simultaneously measured systolic ejection time, CO and other advanced hemodynamic variables can be calculated, including estimations of preload, afterload, and contractility. Numerous studies have validated TED-derived CO against reference methods. Although the agreement of CO values between TED and the reference methods is limited (95% limits of agreement: median 4.2 L/min, interquartile range 3.3–5.0 L/min), TED has been shown to accurately follow changes of CO over time, making it a useful device for trend monitoring. TED can be used to guide perioperative intravascular volume substitution and therapy, with vasoactive or inotropic drugs. Various studies have demonstrated a reduced postoperative morbidity and shorter length of hospital stay in patients managed with TED compared with conventional clinical management, suggesting that it may be a valuable supplement to standard perioperative monitoring. We review not only the technical basis of this method and its clinical application but also its limitations, risks, and contraindications.

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Hemodynamic optimization is one of the crucial goals of anesthesia management in ensuring adequate perioperative organ perfusion. Adequate perfusion, however, not only relies on sufficient perfusion pressure but also on systemic blood flow, i.e., cardiac output (CO), to deliver oxygen and substrates to the organs and to eliminate metabolic by-products. Although arterial blood pressure is measured perioperatively in most patients, CO is not routinely monitored. Thermodilution techniques, requiring insertion of a pulmonary artery catheter, are considered to be the clinical standard of CO measurement.[1,2] However, major risks, high costs, and considerable additional time needed for pulmonary artery catheter insertion limit the routine assessment of CO. Intraoperative CO monitoring could, however, be useful in many patients to guide fluid administration and therapy with vasoactive and inotropic substances. Therefore, transesophageal Doppler (TED) ultrasonography of the descending aorta could be a useful monitoring device. TED allows a continuous estimation of CO and facilitates the assessment of preload, afterload, and myocardial contractility by calculating advanced hemodynamic variables. Various studies have demonstrated improved patient outcome and reduced length of hospital stay when hemodynamic management is guided by TED,[3–12] suggesting that this technique may be a valuable supplement to the current standard hemodynamic monitoring. Early TED devices were not user friendly and were difficult to operate, which prevented widespread clinical use. In recent years, new devices have been developed, which combine the benefits of safe and continuous CO monitoring with the advantages of simple operation and straightforward display of the measured data. We review the technical basis and clinical applications including limitations, risks, and contraindications.

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TECHNICAL PRINCIPLES

Doppler Sonography

The Doppler effect describes an apparent change in the frequency of a wave noticed by an observer moving relative to the source of the wave. The frequency shift, i.e., the discrepancy between actual and

noted frequency, is directly proportional to the relative velocity between the emitter and receiver. By measuring this Doppler frequency shift (Δf), which is produced when moving red blood cells are interrogated by an ultrasound beam, blood flow velocity (v) can be determined by the standard Doppler equation[13]:

$$V = \frac{\Delta f \times c}{2 f_T \times \cos \theta}$$

where c is the velocity of the ultrasound waves in body tissue and f_T, the transmitted frequency. The cosine of the angle between the Doppler beam and blood flow ($\cos \theta$) serves as a correction factor to adjust for the angle of insonation. Note that deviations of the actual from the assumed angle of insonation will result in erroneous velocity calculations. Because of the nonlinear character of the cosine function, this error rapidly increases at increasing angles. For example, a deviation of 1° in the actual from the assumed angle results in approximately 1%, 3%, and 10% error at an insonation angle of 30°, 60°, and 80°, respectively, which increases to approximately 100% at 90°. Ultrasound and Doppler physics have been extensively reviewed in text books and review articles.[14–18]

To determine aortic blood flow, the ultrasound beam can be directed from an intercostal space or the suprasternal notch toward the aortic arch or ascending aorta.[19,20] However, with this approach, continuous monitoring is complicated because it is hardly possible to keep an external transducer in place, thus avoiding changes in the insonation angle or loss of the signal. The esophagus, as a natural guide rail in the thorax allows the probe to stay in place and in close proximity to the descending aorta. The Doppler transducer is mounted within the probe at a fixed angle, and because the esophagus and aorta run almost parallel at the midthoracic level, the insonation angle is approximately the same as that between the probe and the transducer (Fig. 1).[21–23] These considerations prioritize the esophageal route for the continuous assessment of the aortic blood flow.

TED Sonography

TED sonography was first described by Side and Gosling[24] in 1971 and further refined by other investigators.[21,23,25–30] Several devices have been developed; however, most of them are no longer commercially available. Some were no longer marketed after they had been further developed to more modern devices, and others were not user friendly, were technically outdated, or purchased and abandoned by competitors. Modern TED devices use 4-MHz continuous wave or 5-MHz pulsed-wave Doppler, with angles of insonation at 45° or 60°. Currently, three devices are marketed (CardioQ, Deltex Medical, Chichester, UK; HemoSonic 100, Arrow International, Reading, PA; Waki TO, Atys Medical, Soucieu en Jarrest, France) and their technical characteristics are summarized in Table 1.

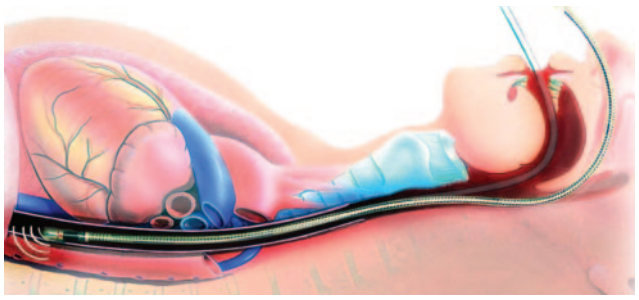

Figure 1. Esophageal Doppler probe *in situ*. The probe is inserted via the oral or nasal route to the midthoracic level (between the 5th and 6th thoracic vertebra). At this level, the aorta and esophagus run approximately parallel, allowing interrogation of the descending aortic blood flow with a known angle of insonation (Adapted with permission from Deltex Medical, Chichester, UK).

The method of CO determination with TED is analogous to one of the established principles used in transesophageal echocardiography, which combines measurements of transvalvular blood flow velocity with determination of the valve area. In contrast to transesophageal echocardiography, TED does not allow direct visual estimation of ventricular filling, contractility, or valvular function, which might be desirable in patients with complex cardiac pathophysiology or extended surgery.

With TED, the descending aortic blood flow velocity is calculated based on the Doppler equation as erythrocytes pass the ultrasound beam in the descending aorta (Fig. 1). Modern monitors display a wave form of the velocity plotted against time, which closely resembles flow-time diagrams obtained from transthoracic Doppler measurements of aortic blood flow.[31–33] Its systolic portion is typically triangular, and the base of the triangle represents the systolic ejection time, which is also referred to as flow time (Fig. 2). Because flow time depends on the heart rate, it is usually corrected by a modification of Bazett's equation (flow time divided by the square root of the cycle time), which is used to correct the QT interval of an electrocardiogram.[34] The resulting flow time corrected (FTc) represents the systolic ejection time adjusted to one cardiac cycle per second. The upslope of the graph shows the acceleration of blood in the descending aorta, from which mean and peak acceleration can be determined. The peak of the wave form corresponds to the peak blood velocity, followed by a down-slope which depicts the deceleration of flow during later systole (Fig. 2).

The area under the systolic portion of the curve represents the stroke distance, i.e., the distance that the blood column has moved forward in the aorta during systole. Descending aortic stroke volume (cm^3) can then be determined by multiplying the stroke distance (cm) with the aortic cross-sectional area (cm^2;

Table 1. Technical Details of Three Transesophageal Doppler (TED) Devices. Information as Provided by the Manufacturer

	CardioQ	HemoSonic 100	Waki TO
Manufacturer	Deltex Medical, Chichester, UK	Arrow International, Reading, PA	Atys Medical, Soucieu en Jarrest, France
Doppler-mode	Continuous wave Doppler	Pulsed wave Doppler	?
Frequency	4 MHz	5 MHz	4 MHz
Angle of insonation	45°	60°	?
M-mode	None	10 MHz	Yes
Probe diameter	14–17 French (4.7–5.7 mm), single use	20 French (6.7 mm), reusable	?, reusable
Unit dimensions	320 × 250 × 170 mm, weight 6 kg	300 × 250 × 200 mm, weight 4 kg	290 × 280 × 160 mm, weight 8 kg
Translation of flow measurement into cardiac output	Nomogram (based on patient's age, weight, and height)	Determination of aortic diameter through M-mode aortography	Nomogram

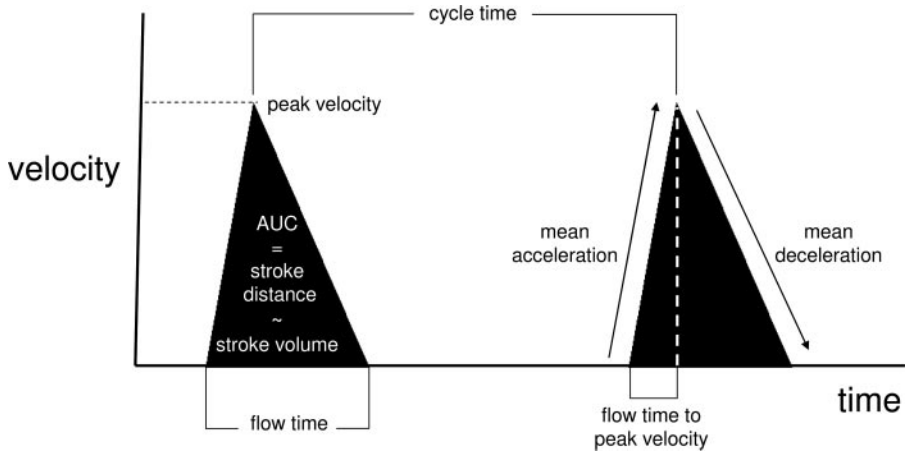

Figure 2. Velocity-time plot. The systolic portion is typically triangular and its base represents the flow time (=systolic ejection time). The peak of the waveform depicts the peak velocity in the descending aorta. Flow acceleration and deceleration are derived from the upslope and downslope of the velocity curve. The area under the systolic portion of the curve (AUC) represents the stroke distance, i.e., the distance that the blood column has moved forward in the aorta during systole. The stroke distance is proportional to the stroke volume under the assumption that aortic diameter and distribution of blood flow between the supra-aortic vessels and the descending aorta remains constant.

Fig. 3). Because blood flow in the descending aorta is only a fraction of total CO, a constant proportion of the blood flow between the descending aorta (approximately 70%) and the coronary and brachiocephalic arteries (approximately 30%) needs to be assumed to estimate systemic stroke volume and CO. The exact mode of how stroke distance is translated to CO varies according to the manufacturer. The HemoSonic 100 device (Arrow International, Reading, PA) measures the aortic cross-sectional area via an integrated 10-MHz M-mode ultrasound probe, whereas the CardioQ device (Deltex Medical, Chichester, UK) uses a nomogram based on the patient's age, height, and weight.

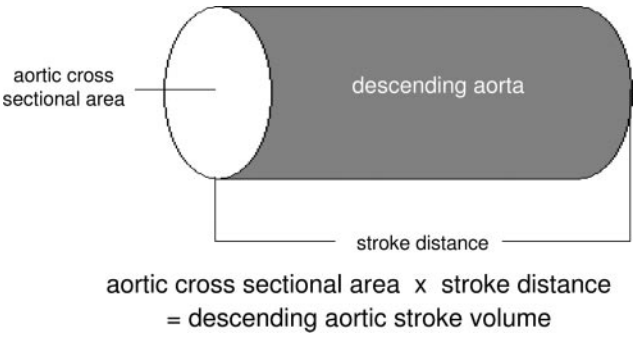

Figure 3. The descending aortic stroke volume can be determined by multiplying the stroke distance with the aortic cross-sectional area.

CLINICAL APPLICATION

Probe Insertion and Placement

Insertion of the esophageal probe can easily be performed within a few minutes[28,35,36] via the oral or nasal route similar to the placement of a gastric tube (Fig. 1). Literature suggests that training in not more than 12 patients is needed to achieve adequate probe positioning and reliable CO measurements.[22,37,38] The CardioQ probes (Deltex Medical, Chichester, UK) are disposable, whereas the multiuse HemoSonic 100 probes (Arrow International, Reading, PA) must first be placed in a single-use sheath. Probes can be inserted in conscious and anesthetized patients.[28,39,40] Optimal insertion depth is the midthoracic level measured between 5th and 6th thoracic vertebra. This

depth can be estimated by superficially mapping the distance from the incisors or nose to the third sternocostal junction, which usually corresponds to a depth between 35 and 45 cm for adults depending on the route of insertion. For children, insertion depth from the lips can be approximated using the formula, 0.2 × patient height in cm + 7 cm.[36] After insertion, the probe is rotated until the ultrasound beam is directed toward the descending aorta as confirmed by visualization of the typical aortic wave form and a characteristic pulsatile sound pattern. Optimal focus is accomplished by slight manipulations of the probe until the largest and sharpest possible wave form with minimal spectral dispersions and maximal pitch is found. With the HemoSonic 100 device (Arrow International, Reading, PA), the integrated M-Mode also allows direct visualization of the aortic wall. Optimal signal quality is crucial to obtain valid results. Accidental manipulations of the probe (especially probe rotation) may result in impairment of the signal quality. Therefore, the operator should check the signal quality regularly by observing the displayed wave forms while assessing the patient's hemodynamic variables. If the signal quality decreases, the probe needs to be refocused.[38]

Risks and Contraindications

The incidence of complications associated with TED use has not been systematically investigated. Reported complications include minor trauma to the buccal cavity,[28] transient vagal response during probe insertion,[41] unintentional removal of a gastric tube during probe removal,[42] epistaxis,[43] and two cases of tracheal or bronchial probe misplacement.[44,45] One major adverse event has been reported related to the endobronchial probe placement. This probe had likely compromised the seal of the tracheal tube cuff, resulting in aspiration of gastric fluid.[44] Although the probe was located endobronchially, the aortic Doppler trace appeared normal. Thus, a normal signal does not exclude probe misplacement. Two unconfirmed cases of esophageal perforation due to probe insertion were reported in the Manufacturer and User Facility Device Experience Database of the United States Food and Drug Administration. One of the patients was receiving long-term corticosteroid medication, which might have been a predisposing factor. Although it is likely that minor complications are underreported, the risk of TED insertion seems to be low so that it can be considered as a safe technology.

Contraindications include any pathology which predisposes the patient to an increased risk of injury or bleeding, including esophageal or oropharyngeal malformations, strictures, tumors, varices, esophagitis, recent esophageal or upper airway surgery, and long-term corticosteroid therapy or severe bleeding disorders (Table 2). In patients with craniofacial trauma, it may be safer to avoid the nasal route in analogy to the placement of nasogastric tubes.[46–55]

Table 2. Contraindications for the Use of Transesophageal Doppler (TED) Devices and Conditions in which the Device may Potentially Give Inaccurate Readings

Contraindications
1. Local esophageal or oropharyngeal pathology
Malformations
Esophageal varices
Tumors
Strictures
Esophagitis
Recent esophageal or upper airway surgery
2. Systemic pathology increasing the risk of local tissue damage or bleeding
Long term corticosteroid treatment
Severe bleeding disorders
3. Specific pathology depending on the route of probe insertion
Craniofacial trauma or basilar scull fracture for nasal route
Conditions in which the device may potentially give inaccurate readings
1. Conditions potentially resulting in turbulent aortic blood flow
Aortic coarctation
Severe aortic stenosis
Intraaortic balloon counter pulsation
2. Conditions potentially resulting in major deviations of the insonation angle
Severe scoliosis
Operative manipulations of the anatomic relationship between esophagus and aorta
3. Conditions potentially resulting in altered distribution of blood flow
Aortic cross-clamping
Neuraxial anesthesia
Severe aortic insufficiency
4. Conditions restricting free access to patient's head
Head and neck surgery

Clinical Validation

Numerous investigators have compared measurements of CO derived from TED with reference methods in various patient populations and under various conditions (Table 3).[22,28,30,36–38,42,43,45,56–90] Most often, the transpulmonary thermodilution technique has been used as the reference method. Validation studies observed a positive correlation between TED measurements and reference values with a median correlation coefficient of 0.80 (interquartile range 0.73–0.89), suggesting a linear association between the two measurement techniques (Table 3). In Bland-Altman analyses, a median bias of 0.37 L/min (interquartile range, 0.15–0.69 L/min) was observed between TED and reference methods. The 95% limits of agreement values are wide (median 4.2 L/min, interquartile range, 3.3–5.0 L/min). These observations suggest that individual CO measurements obtained with TED may differ considerably from CO values derived by the thermodilution technique, so that the two techniques are not interchangeable. In this context, it has to be considered that TED measurements are based on the assumption of a constant diversion of blood flow. This assumption may be violated, e.g., by aortic cross-clamping or other conditions (Table 3). Furthermore,

Table 3. Studies Comparing Doppler Derived Hemodynamic Parameters to Reference Methods in Humans (Medline-Indexed, English Literature Only)

Reference	Year	Device	Population	*n*/paired data	Parameters compared: Doppler	Parameters compared: Reference	*R*	Bias[a]	95% LOA[a]
Knirsch et al.[61]	2008	CardioQP	Pediatric cardiologic	40/120	CO	PAC bolus TD	0.81	+0.66	−1.13 to +2.45
Lafanechère et al.[62]	2006	HemoSonic 100	Infrarenal aortic surgery	22/NR	CO	PAC bolus TD			
			After probe insertion				0.87	+0.10	−1.36 to +2.19
			Preclamping				0.73	+0.13	−2.23 to +2.49
			10 min after clamping				0.75	+0.43	−1.65 to +2.51
			Before declamping				0.77	+0.54	−1.56 to +2.64
			10 min after declamping				0.80	+0.18	−1.82 to +2.18
			End of surgery				0.82	+0.15	−1.85 to +2.15
					Change in CO		0.84	NR	NR
Sharma et al.[63]	2005	TECO	Post off-pump cardiac surgery	35/140	CO	PAC bolus TD	0.59	+1.18	−1.54 to +3.90
Collins et al.[64]	2005	HemoSonic 100	Off-pump cardiac surgery	50/302	CO	PAC bolus TD			
			After probe insertion				NR	−0.1	−2.1 to +1.9
			Before heart displacement				NR	+0.6	−1.4 to +2.6
			During heart displacement				NR	+0.5	−1.1 to +2.1
			Before sternal closure				NR	+0.7	−0.7 to +2.1
Bein et al.[65]	2004	HemoSonic 100	Cardiac surgery	10/107					
					CO	PAC continuous TD	NR	−0.15	−2.33 to +2.03
					CO	Pulse contour analysis	NR	−0.58	−2.70 to +1.54
Decoene et al.[66]	2004	HemoSonic 100	Off-pump cardiac surgery	15/NR	CO	PAC continuous TD			
			Before heart displacement				NR	+0.19	−0.89 to +1.27
			During heart displacement 1				NR	+1.4	−1.2 to +4.0
			During heart displacement 2				NR	+1.5	−0.8 to +3.8
			During chest closure				NR	+0.4	−0.8 to +1.6
Kim et al.[67]	2004	CardioQ	Escharectomy after burns	20/92	CO	PAC bolus TD	0.80	+0.77	−1.97 to +3.51
Hullett et al.[68]	2003	CardioQ	Off-pump cardiac surgery	20/331	CO	PAC bolus TD	0.62	−0.56	−1.84 to +0.72
Iregui et al.[42]	2003	EDM I	Medical and surgical ICU	24/24	CO	PAC bolus TD	0.78	NR	NR
Jaeggi et al.[69]	2003	HemoSonic 100	ICU after cardiac surgery	20/85	CI	PAC bolus TD	0.3	+0.23	−1.4 to +1.8
Seoudi et al.[70]	2003	EDM	Surgical ICU	15/150	CO	PAC bolus TD	0.97	NR	NR
Roeck et al.[71]	2003	CardioQ	ICU	19/	CO	PAC bolus TD			
			Investigator 1 before volume bolus	/20			NR	−0.39	NR
			Investigator 2 before volume bolus	/20			NR	+0.17	NR
			Investigator 1 after volume bolus	/20			NR	+0.28	NR
			Investigator 2 after volume bolus	/20			NR	+0.24	NR
Moxon et al.[45]	2003	HemoSonic 100	Cardiosurgical ICU	13/47	CO	PAC bolus TD	0.81	−0.23	−2.35 to +1.89
Su et al.[56]	2002	HemoSonic 100	Cardiac surgery	12/185	CO	PAC bolus TD	0.64	0.11	−2.13 to +2.35
				12/192	CO	PAC continuous TD	0.92	0.05	−0.93 to +1.03
Leather et al.[72]	2001	EDM II	Radical prostatectomy	14/84	CO	PAC bolus TD			
			Before epidural anesthetic administered				NR	−0.89	−2.67 to +0.88
			After epidural anesthesic administered				NR	+0.55	−3.21 to +4.30
Odenstedt et al.[73]	2001	Dynemo 3000	Liver transplantation	14/124	ABF	PAC bolus TD	0.78	NA	NA
					Change in ABF		0.80	NA	NA
DiCorte et al.[74]	2000	EDM	Cardiac surgery	34/160	CO	Aortic flow probe	0.77	NR	NR
Penny et al.[43]	2000	EDM	Preeclampsia	17/NR	CO	PAC bolus TD	NR	−2.0	−4.0 to +1.0
Tibby et al.[36]	2000	EDM	Pediatric intensive care	100/198	CO	Femoral artery TD	0.90	NR	NR
					Change in CO		NR	0.87%	−16.0% to +17.7%
Baillard et al.[75]	1999	EDM II	ICU	10/145	CO	PAC continuous TD		−0.01	−0.97 to +0.96
Madan et al.[76]	1999	EDM	Surgical ICU	14/118	CO	PAC bolus TD	0.77	NR	NR
Bernardin et al.[77]	1998	Dynemo 3000	Medical ICU	22/60	ABF	PAC bolus TD	0.92	NA	NA
				16/16	Change in ABF		0.81	NA	NA
Cariou et al.[59]	1998	Dynemo 3000	ICU		ABF	PAC bolus TD	0.80	NA	NA
Colbert et al.[78]	1998	EDM	Liver transplantation	18/234	CO	PAC bolus TD	0.71	+0.07	−4.10 to +4.23
Lefrant et al.[22]	1998	EDM	ICU		CO	PAC bolus TD			
			During training period	11/107			0.53	+1.2	−2.0 to +4.4
			After training in 12 patients	49/320			0.89	+0.1	−2.1 to +2.3
Valtier et al.[79]	1998	EDM	ICU						
				46/136	CO	PAC bolus TD	0.95	+0.24	−1.56 to +2.04
				17/53	CO	Suprasternal Doppler	0.94	+0.16	−1.58 to +1.90
				13/46	CO	Indirect calometry	0.89	−0.2	−2.74 to +2.16
				NR/88	Change in CO	PAC bolus TD	0.90	0	−1.7 to +1.7
Krishnamurthy et al.[38]	1997	EDM II	Cardiac surgery		CO	PAC continuous TD			
			First 11 patients, no readjustments of probe position	11/513			NR	+0.8	−2.2 to +3.8
			Subsequent patients, probe position checked and refocused before measurements	5/285			NR	+0.14	−0.58 to +0.85

(*Continued*)

Table 3. Continued

Reference	Year	Device	Population	*n*/paired data	Parameters compared: Doppler	Parameters compared: Reference	*R*	Bias[a]	95% LOA[a]
Keyl et al.[80]	1996	EDM II	Cardiac surgery	24/NR	CO	PAC Bolus TD			
			After induction of anesthesia (A)				NR	+0.38	−1.7 to +2.5
			After start of surgery (B)				NR	+0.48	−2.3 to +3.3
			After sternotomy (C)				NR	+0.69	−2.2 to +3.6
			Change from A to B		Change in CO		NR	+0.10	−1.7 to +1.9
			Change from B to C				NR	+0.21	−1.8 to +2.2
Klotz et al.[81]	1995	EDM I	Infrarenal aortic surgery	6/	CO	PAC Bolus TD			
			Preclamping	/55			0.84	−0.96	−3.24 to +1.33
			During clamping	/75			0.79	−1.51	−2.99 to −0.02
			After declamping	/65			0.76	−1.47	−3.79 to +0.86
			All periods	/15	Changes in CO >2 L/min		0.89	NR	NR
Murdoch et al.[82]	1995	EDM prototype	Cardiosurgical pediatric ICU	11/	Change in MD	PAC bolus TD	NR	−0.5%	*−10.7% to +9.7%*
Schmid et al.[83]	1993	Accucom 2	Post cardiac surgery	16/140	CO	PAC bolus TD	0.56	−0.37	−3.77 to +3.03
Perrino et al.[84]	1991	Accucom 2	Abdominal aortic surgery	39/		PAC bolus TD			
			Preclamping	/193	CO		0.94	+0.4[b]	Bias ± 1.4
			During clamping	/134	CO		0.72	−0.7[b]	Bias ± 2.6
			After declamping	/147	CO		0.88	+0.1[b]	Bias ± 1.5
			Preclamping	/159	Change in CO		0.84	NR	NR
			During clamping	/108	Change in CO		0.61	NR	NR
			After declamping	/127	Change in CO		0.82	NR	NR
Singer et al.[58]	1991	Prototype	ICU and cardiothoracic surgery	43/49	Change in CO	PAC bolus TD	NR	+0.6%	*−16.4% to +17.6%*
Stein et al.[85]	1991	Lawrence 3000	Cardiac surgery	11/106	CO	PAC bolus TD	NR	−1.0	−4.6 to +2.6
Perrino et al.[57]	1990	Accucom 1	Noncardiac surgery	20/107	CO	PAC bolus TD	0.72	+0.45[b]	−2.0 to +2.9[b]
		Accucom 2	Noncardiac surgery	23/184	CO	PAC bolus TD	0.91	+0.2[b]	−1.2 to +1.6[b]
Spahn et al.[86]	1990	Accucom 1	Post cardiac surgery		CO	PAC bolus TD			
			Calibration method 1	NR/79			0.65	NR	NR
			Calibration method 2	NR/49			0.76	NR	NR
				NR/96	Change in CO		0.55		
Kumar et al.[87]	1989	Ultracom	Surgical patients (various specialties)	14/246	CO	PAC bolus TD	0.75	?	NR
Singer et al.[28]	1989	Prototype	ICU and cardiac surgery			PAC Bolus TD			
			All patients	38/200	Change in CO		NR	+0.6%	*−13.5% to +14.7%*
			Age range 18–39 yr	2/NR	CO		0.98		
			Age range 40–59 yr	12/NR	CO		0.74		
			Age range 59–78 yr	24/NR	CO		0.81		
Singer et al.[60]	1989	Prototype	Acute respiratory failure	6/19	Change in CO	PAC bolus TD	NR	−0.3%	*−14.4% to +13.9%*
Ueda et al.[88]	1989	Accucom	Elective surgery	16/71	CO	PAC bolus TD	0.94	+0.06	NR
Siegel et al.[89]	1988	Lawrence 3000	Cardiac or vascular surgery	9/25	CO	PAC bolus TD	0.68	−0.1	NR
					Change in CO		0.81	NR	NR
Freund et al.[37]	1987	Ultracom and Accucom	Noncardiac surgery	23/420	CO	PAC bolus TD			
			All patients				0.67	+0.16	NR
			Only last 13 patients				0.85	NR	NR
Mark et al.[90]	1986	Ultracom	Heart surgery pre bypass	16/82	CO	PAC bolus TD	0.92	NR	NR
Lavandier et al.[30]	1985	Prototype	ICU	21/300	ABF	PAC bolus TD	0.98	NR	NR

Because of different mathematical approaches, the algebraic sign reported with the bias does not indicate whether Doppler over- or underestimated the respective reference value in a particular study.

ABF = aortic blood flow; CI = cardiac index; CO = cardiac output; ICU = intensive care unit; LOA = limits of agreement; MD = minute distance; NA = not applicable; NR = not reported; PAC = pulmonary artery catheter; TD = thermodilution.

[a] Bias and LOA are generally reported in L/min unless otherwise indicated; changes in CO have sometimes been reported in percent % (italic numbers).

[b] Value estimated from figure. The EDM Devices, actually forerunners of the CardioQ (Deltex Medical, Chichester, UK), were called ODM (Oesophageal Doppler Monitor) on the European market, therefore this term is found in some of the referenced studies. The Dynemo 3000 (Sometec, Paris, France) was a forerunner of the HemoSonic 100 (Arrow International, Reading, PA). All other devices are no longer marketed.

interpretation requires adequate training of the operator in the understanding of the hemodynamic relationships between preload, afterload, contractility, pressure, and flow.

The positive correlation between TED and the reference methods suggests that the direction of changes in CO can be tracked by TED and that a high value obtained with one technique will likely be reflected by a high reading with the other technique. Indeed, numerous studies report that TED measurements accurately follow changes in CO over time.[28,36,43,59,62,73,77,79,81,82,84] Thus, TED may be used as a trend monitor rather than a device for the exact measurement of CO when the anesthesiologist or intensivist would like to know whether certain events or interventions cause a relevant change in CO.

Hemodynamic Variables: Estimation of Preload, Contractility, and Afterload

Although monitoring of CO can provide information about the hemodynamic situation, this variable by itself is often insufficient to make therapeutic decisions. A low CO, for instance, could be due to hypovolemia, heart failure, or an abnormally increased total peripheral resistance—conditions which require different therapeutic approaches. Compensatory mechanisms can camouflage the hemodynamic situation if

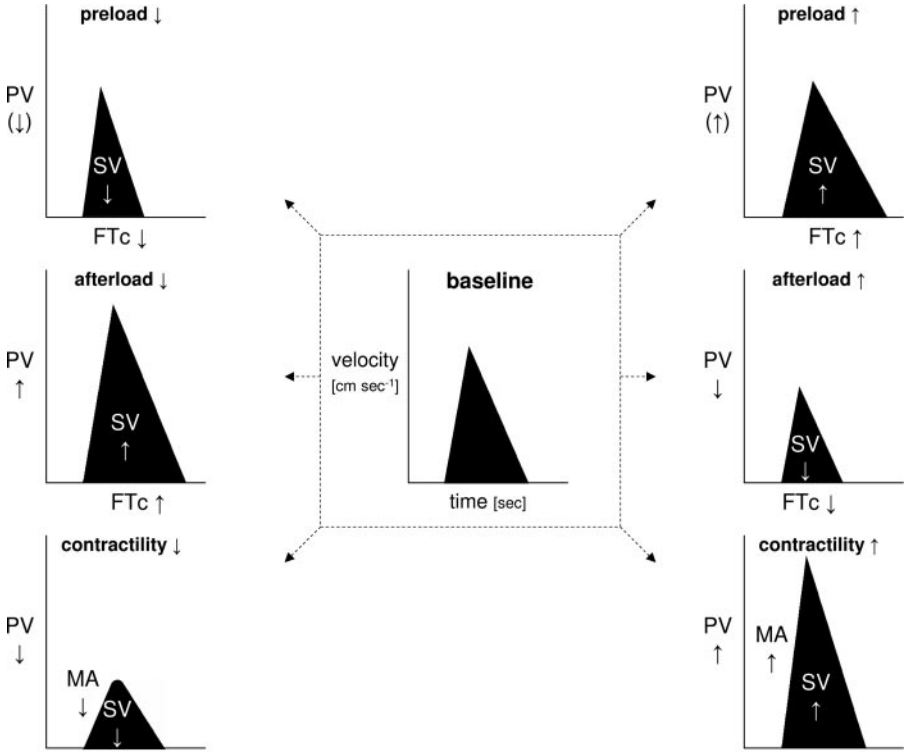

Figure 4. Velocity-time waveforms of aortic blood flow under various hemodynamic conditions. The constellations depicted for preload do not apply to hypovolemic shock or hypervolemic cardiac decompensation, both of which are associated with a marked reduction in SV, PV, and eventually FTc. SV = stroke volume; FTc = flow time corrected; PV = peak velocity; MA = mean acceleration; ↑ and ↓ direction of change.

only standard monitoring is performed,[91,92] and clinical assessment is frequently insufficient or misleading.[42,93,94] New TED devices facilitate estimations of preload, afterload, and ventricular contractility by allowing visual evaluation of the velocity wave form (Figs. 4 and 5) and by reporting advanced hemodynamic variables, such as FTc, peak velocity, or mean acceleration. In general, the FTc primarily responds to changes in preload and afterload, whereas contractility is predominantly reflected by peak velocity and mean acceleration. Reference values of these variables have not been well established. Because the systole takes approximately one-third of the entire cardiac cycle, an FTc in the range of 330–360 ms is generally considered normal.[95] However, the ascending aortic blood flow measurements in volunteers suggest that the FTc actually depends on age.[96] The FTc was 292 ± 22 ms (mean ± SD) in 21–30-yr-old volunteers (n = 18), 324 ± 38 ms in 41–50-yr-old volunteers (n = 18) and remained fairly constant with increasing age. In contrast, the peak velocity decreases with increasing age from approximately 110–130 cm/s at childhood and adolescence to about 60–70 cm/s at 70 yr.[96–98] Gender does not have an influence on either variable.

No single measured variable is specific for preload, afterload, or contractility. Changes in one variable will be accompanied by compensatory changes of the other variables. It is often the combination of different variables and the way they respond to dynamic cardiovascular events or challenges, which clarifies the hemodynamic situation. The characteristic patterns of the variables, which are described later and in Figures 4 and 5, are derived from transcutaneous Doppler measurements of the ascending aorta in healthy volunteers[33] and critically ill patients,[31,32] and from two studies of the transesophageal descending aortic blood flow measurements in critically ill and surgical patients.[28,58]

Preload and Afterload

The FTc is frequently claimed to indicate preload. Indeed, the FTc allows the assessment of fluid responsiveness in hypovolemic patients.[99] One study suggests that the FTc is superior to pulmonary artery wedge pressure in predicting preload.[76] However, because the FTc is inversely related to systemic vascular resistance, a shortened FTc may also indicate a preload-independent vasoconstriction, for example, due to hypothermia, vasopressors, or heart failure. To differentiate between hypovolemia and increased afterload, peak velocity also needs to be assessed. Compensated hypovolemia is characterized mainly by a shortened FTc with a normal or only moderately reduced peak velocity, reflected by a narrow wave form with a nearly normal amplitude (Figs. 4 and 5). In contrast, a shortened FTc accompanied by a considerably reduced peak velocity, resulting in a wave form with a narrow base and a reduced amplitude, suggests an increase in afterload. In equivocal cases, changes in stroke volume secondary to a volume bolus can clarify the situation.

The normal, not volume over-loaded heart, operates on the ascending limb of the Starling curve (stroke volume plotted against the preload) and a fluid challenge will result in an adequate increase in stroke

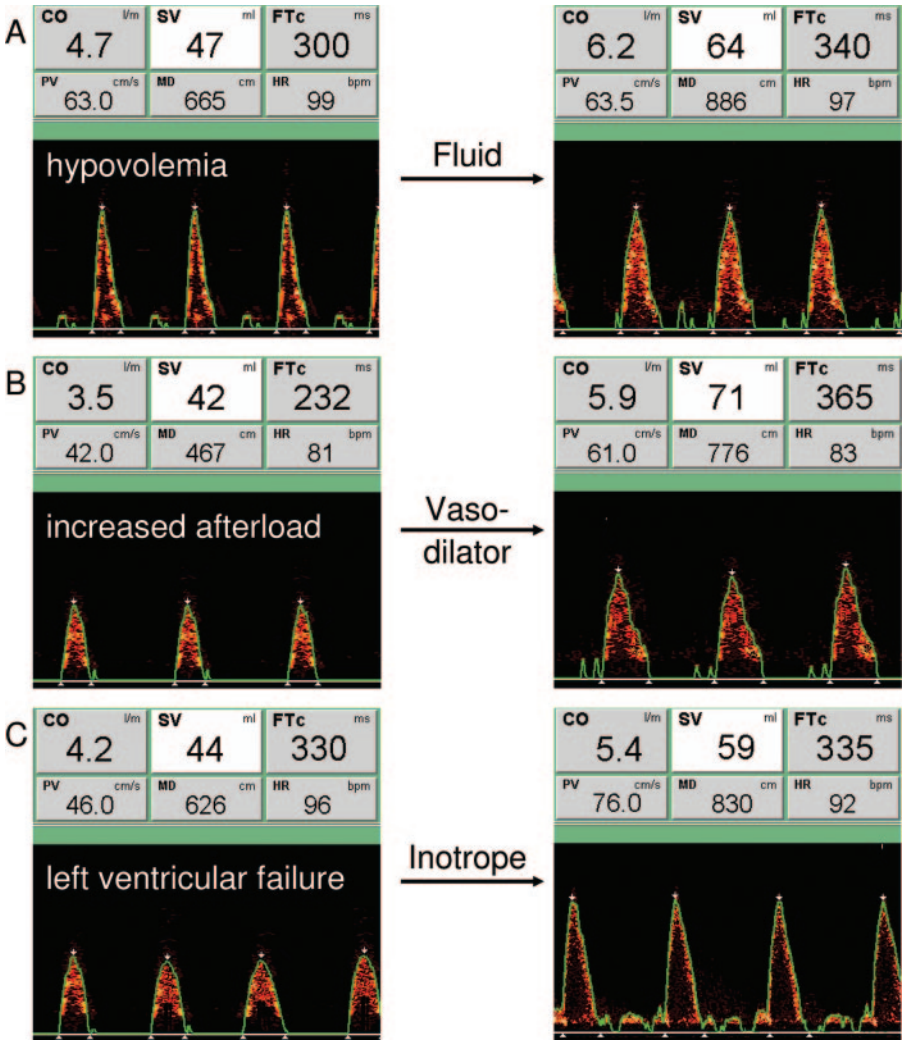

Figure 5. Original recordings of waveforms and variables from patients with hypovolemia (A), increased afterload (B), and left ventricular failure (C) and the response to adequate treatment. CO = cardiac output; SV = stroke volume; FTc = flow time corrected; MD = minute distance (=stroke distance multiplied with heart rate); HR = heart rate (CardioQ device, Adapted with permission from Deltex Medical, Chichester, UK).

volume. In contrast, the absence of an adequate increase indicates that this particular heart operates on the flat portion of the Starling curve and may likely become hypervolemic with further filling. Therefore, situations in which intravascular fluid administration is not accompanied by adequate increases (or even decreases) in stroke volume despite a low FTc should not prompt the clinician to assume (persisting) hypovolemia but rather alert the anesthesiologist to suspect a pathologically increased afterload. Note that the absence of an adequate response to a volume bolus can in some instances be caused by pathological states preventing left ventricular filling, such as ongoing hemorrhage, severe mitral stenosis, lung embolism, heart tamponade, or extravascular infusion of the administered fluids.

Contractility

Peak velocity and mean acceleration are markers of left-ventricular contractility. Left ventricular failure is associated with a decrease of both variables, resulting in a reduced height, slower upslope, and rounded apex of the wave form. In contrast, inotropes will increase peak velocity and mean acceleration (Figs. 4 and 5).

Guidance of Perioperative Intravascular Volume Therapy with TED

Optimal perioperative fluid management is controversial, and restrictive as well as liberal fluid managements have been advocated.[100–107] Recipe-like concepts do not consider that preoperative fasting, volumes of distribution, blood and urine loss, and evaporation vary considerably among patients. Although hypovolemia jeopardizes oxygen transport and is a critical contributor to tissue hypoperfusion and hypoxia,[108–111] hypervolemia may promote venous congestion, lung edema, or heart failure[112,113] and has been shown to worsen the outcome after various types of surgery.[102,107,114–116] Therefore, it seems advantageous to guide fluid replacement and, hence, stroke volume individually to avoid hypovolemia and hypervolemia. Arterial blood pressure and heart rate alone are insufficient to detect compensated states of hypovolemia because they can remain fairly normal, whereas microcirculation and tissue oxygenation are already markedly reduced.[91,92] TED has been suggested to be a useful supplement to current standard monitoring,[58,117] because it allows early recognition of hypovolemia and guides intravascular volume

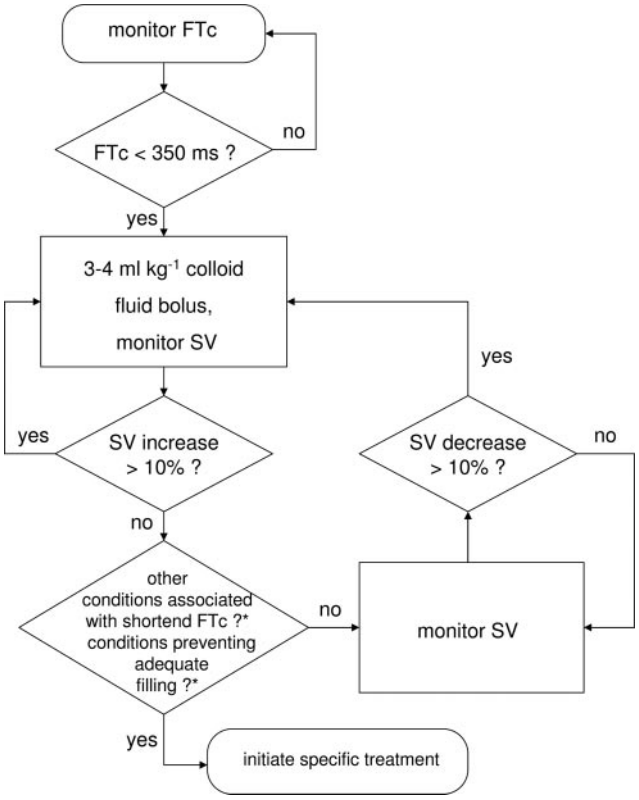

Figure 6. Algorithm to guide intraoperative fluid therapy with Doppler derived variables. FTc = flow time corrected; SV = stroke volume. *These conditions include but are not limited to increased afterload, heart failure, ongoing hemorrhage, paravenous fluid infusion, severe mitral stenosis, lung embolism, or heart tamponade.

replacement while avoiding hypervolemia. Practically, repetitive fluid boli are applied until no further adequate fluid responsiveness is observed, indicating that the optimal fluid load has been reached. In general, an increase in stroke volume of more than 10% after a colloid bolus of approximately 3 mL/kg is considered as an adequate response.[3–5,7–9,11] There is no single protocol which can currently be recommended as best practice to guide fluid therapy. Figure 6 shows an example of an algorithm for practical clinical use based on the synthesis of experimental protocols and literature.[3–12,118]

Numerous studies have compared perioperative Doppler-guided intravascular volume replacement strategies with conventional clinical volume replacement in various groups of surgical patients, including abdominal, cardiac, orthopedic, urologic, and gynecologic surgery and multiple-trauma patients (Table 4).[3–12] The investigators used different experimental protocols, with the common basis that in the TED groups fluid boli were administered according to an algorithm, until a defined hemodynamic target was reached. All of these studies, with nearly 1000 patients, conclusively report beneficial effects in the Doppler-guided groups (Table 4). Patients managed with esophageal Doppler required fewer days in an intensive care unit and were medically fit for discharge from hospital earlier.[3–12] This is not only advantageous for the individual patient but may also lead to a saving in overall costs.[5] Moreover, the data suggest that Doppler-guided fluid replacement reduces the risk of postoperative complications and morbidity.[3–5,7,12] Reductions in the incidence of postoperative nausea and vomiting, a shorter recovery time of gut function, and resumption of enteral nutrition have also been reported in the volume-optimized groups.[5,6,9] As a rough guide, such beneficial effects have been observed with operations exceeding 1 h, operations involving entry into a body cavity, or in surgery with an anticipated blood loss of more than 1 L.[3–12] None of the studies was actually powered to detect reductions in mortality; however, Chytra et al.[3] observed a trend toward increased survival in multiple-trauma patients managed with Doppler-guided fluid therapy. Nevertheless, a conclusive determination of the role of TED in reducing morbidity and mortality is not yet possible and requires additional and adequately powered studies.

Other Potential Clinical Indications

In addition to hemodynamic guidance, TED has been suggested to be useful for optimization of positive end-expiratory pressure. Because delivery of oxygen to body tissues is a function of arterial oxygen content and CO, decreases in CO by increasing positive end-expiratory pressure may decrease oxygen delivery despite increases in arterial oxygen content.[60] Other reported clinical applications of TED are early detection of hypervolemia associated with transurethral resection syndrome[119] and monitoring of successful ligation of patent ductus arteriosus, by observing disappearance of the typical ductal flow pattern.[120]

Limitations of TED

First, Doppler velocity measurements assume that all erythrocytes travel in the same direction and at the same speed. In healthy patients, descending aortic blood flow is usually laminar and is assumed to show a relatively uniform velocity distribution over most of the aortic cross-section.[121] However, this may not necessarily be the case in patients with aortic pathology, such as coarctation, severe stenosis of the aortic valve, dissection, or aneurysm of the aorta or intraaortic balloon counter pulsation. Moreover, determination of flow velocity relies on accurate knowledge of the angle of insonation. The assumption that the angle of insonation equals the angle between the probe and the transducer might be flawed in patients with altered thoracic anatomy, e.g., in patients with severe scoliosis or during surgical manipulations affecting the anatomic relationship between the esophagus and aorta.

Second, the aortic cross-sectional area is either measured or estimated. Both approaches are subjected to a certain inaccuracy. The HemoSonic 100 directly

Table 4. Clinical Studies Comparing Conventional Fluid Therapy to Doppler-Guided Goal Directed Therapies (Medline-Indexed, English Literature Only)

Reference	Year	Conflict of interest A[a]	Conflict of interest B[a]	Device	Population	*n*	Main outcome (Doppler-guided vs conventional fluid replacement)
Chytra et al.[3]	2007	No	No	HemoSonic 100	Multiple-trauma patients in intensive care unit	162	Reduced infectious complications, shorter length of ICU and hospital stay, lower lactate concentrations, trend toward increased survival
Noblett et al.[4]	2006	No	No	CardioQ	Colorectal resection	103	Reduced morbidity and shorter length of hospital stay
Wakeling et al.[5]	2005	No	Yes	CardioQ	Major bowel surgery	128	Reduced gastrointestinal and overall morbidity, faster recovery of gut function and shorter length of hospital stay, reduced costs
McFall et al.[6]	2004	No	Yes	CardioQ	Colorectal resection	70	Reduced length of hospital stay, earlier resumption of unrestricted diet
McKendry et al.[7]	2004	Yes	Yes	CardioQ	Intensive care unit after cardiac surgery	174	Reduced length of hospital stay, trend toward reduced length of ICU days and toward less postoperative complications
Conway et al.[8]	2002	No	No	TECO 2	Major bowel surgery	57	Reduced need for ICU admission
Gan et al.[9]	2002	Yes	?	EDM	Major general, gynaecological, or urologic surgery	98	Reduced length of hospital stay, less postoperative nausea and vomiting, earlier solid oral regimen
Venn et al.[10]	2002	No	No	EDM	Proximal femoral fracture surgery	90	Earlier medically fit to discharge
Sinclair et al.[11]	1997	No	No	EDM 2	Proximal femoral fracture surgery	40	Reduced length in acute hospital bed stay and reduced length of total hospital stay
Mythen et al.[12]	1995	No	No	EDM 1	Cardiac surgery	60	Reduced incidence of gut hypoperfusion, less major complications, reduced length of ICU and hospital stay

The EDM Devices, actually forerunners of the CardioQ (Deltex Medical, Chichester, UK), were called ODM (Oesophageal Doppler Monitor) on the European market; therefore, this term is found in some of the referenced studies.

[a] Conflict of interest A: direct support by manufacturer. B: study conducted by investigators who received fees (speakers bureau, etc.) from manufacturer.

measures the aortic diameter, from which the cross-sectional area is calculated assuming a circular shape of the aorta. However, the aortic cross-section is not a perfect circle. Because the ejected volume distends the aortic wall, the aortic cross-section undergoes changes during the cardiac cycle,[122,123] making accurate determinations of the cross-sectional area difficult. A study performed with the Dynemo3000 (Sometec, Paris, France), a precursor of the HemoSonic 100, reveals an average 5% error in the determination of the aortic diameter.[59] These errors in aortic diameter determination are magnified, because calculation of the cross-sectional area of a circle includes the square of its radius. Moreover, aortic plaque burden in the elderly and decreased aortic compliance may affect the diameter and aortic flow dynamics. To avoid this imprecision, the CardioQ does not measure the aortic diameter but uses a nomogram, which incorporates patient's age, height, and weight. This nomogram is not merely a means of estimating aortic cross-sectional area but provides a calibration factor to translate the descending aortic blood flow velocity to total left ventricular CO over a wide range of patient conditions.[124] The nomogram is derived from average values in a population and does not necessarily predict the true value for an individual patient. The nomogram could possibly be further individualized by incorporating the patient's actual arterial blood pressure, because changes in blood pressure have been particularly shown to affect the aortic diameter.[123,125,126]

Third, CO calculations assume a constant diversion of blood flow between supra-aortic vessels and the descending aorta. Actually, this proportion is not constant. Various states of acute illness, including hemorrhage as well as septic, cardiogenic, and anaphylactic shock, lead to redistribution of CO.[127–136] Moreover, general anesthetics exert variable effects on vascular tone and may alter the distribution of CO to various organs significantly.[137] Similarly, aortic cross-clamping and sympathetic blockade during spinal or

epidural anesthesia have also been shown to affect the diversion of CO.[72,138] In patients with severe aortic insufficiency, a relevant portion of the systolic aortic blood flow regurgitates into the left ventricle during diastole and does not contribute to organ perfusion.

Regardless of the three limitations discussed earlier, trend monitoring of CO should theoretically be possible as long as the basic conditions remain unaltered. As soon as changes of the basic conditions occur, for example, because of clamping or unclamping of the aorta or epidural injection of local anesthetics and subsequent sympathicolysis, measurements of CO do not necessarily reflect the true changes in CO associated with such interventions.

In addition, in patients with atrial fibrillation, all measured values may vary considerably from beat to beat. This may counteract reliable calculations of the FTc and can make the interpretation of results difficult. Averaging the readings over several beats can be helpful in such cases.

Finally, free access to the head is needed because occasional refocusing of the probe can be necessary. Therefore, use of TED monitoring during head and neck surgery can be difficult. Conditions in which the device may potentially give inaccurate readings are summarized in Table 2.

CONCLUSIONS

TED allows the continuous trend monitoring of CO and other advanced hemodynamic variables. It may facilitate early recognition of hemodynamic changes, so that the anesthesiologist can anticipate rather than react to hemodynamic deterioration. It may help to guide individual fluid administration and therapy with vasoactive and inotropic drugs. Perioperative hemodynamic guidance using TED has been shown to reduce postoperative morbidity and the length of hospital stay.

The limitations of TED technology are derived from the assumptions that are needed to translate descending aortic blood flow velocity to CO. Because these assumptions may not always hold true, the shortcomings of the method need to be considered to avoid misinterpretations of the measured data. In particular, it should be noted that TED-derived CO values are not interchangable with the thermodilution technique.

In summary, TED is a safe and easy technique with the potential to improve patient outcome, making it a valuable supplement to current standard hemodynamic monitoring when inherent limitations of the method are considered.

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