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Cardiac output measured by electrical velocimetry in the CT suite correlates with coronary artery enhancement: a feasibility study

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Background: Cardiac output (CO) is inversely related to vascular contrast medium (CM) enhancement during computed tomography (CT). Impedance cardiography with a new technique, electrical velocimetry (EV), may create opportunities to measure CO pre-examination for adaptation of CM injection parameters.

Purpose: To relate CO_{EV} measured by radiology staff to aortic attenuation as a measure of coronary artery attenuation during CT coronary angiography (CTCA), and to formulate a tentative statistical model to adapt CM injection parameters to CO.

Material and Methods: CO_{EV} was measured immediately before 100 kVp CTCA (64-multirow detector) in 27 patients with presumed coronary artery disease. For CTCA, 260 mg I/kg (maximum dosage weight: 80/90 kg for women/men) was injected intravenously during 12 s. Simple linear regression analysis was performed to explore the correlation between aortic attenuation (Hounsfield units, HU) and body weight, the influence of CO_{EV} on aortic attenuation adjusted to injected CM dose rate (HU per mg I/kg/s), and to establish a tentative formula on how to adapt CM injection parameters to CO_{EV} and desired aortic attenuation.

Results: The correlation between aortic attenuation and body weight was weak and non-significant ($r=-0.14$ after outlier exclusion). A significant negative correlation ($r=-0.63$) was found between aortic attenuation adjusted to injected CM dose rate (HU per mg I/kg/s) and CO_{EV}. The resulting formula, CM dose rate$=CO_{EV}\times$(aortic attenuation–240)/55, made it possible to calculate CM volumes and injection rates at various COs and, for example, the present mean aortic attenuation (438 HU), injection time (12 s), CM concentration (320 mg I/ml), and a certain body weight.

Conclusion: EV makes it possible to measure CO in the CT suite before vascular examinations. Hence, CM doses may be decreased in low CO states to reduce the risk of CM-induced nephropathy without jeopardizing diagnostic quality and may be increased in high CO states to avoid poor enhancement.

Keywords: Contrast enhancement; coronary angiography; contrast medium-induced nephropathy

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During the past decade, computed tomographic coronary angiography (CTCA) has become a clinical reality as a consequence of major advances in CT technology. Optimized administration of iodine (I) contrast medium (CM) is crucial to obtain the balance between diagnostic vascular enhancement and avoidance of complications such as CM-induced nephropathy (CIN) (1, 2). The risk of CIN is related to CM dose, degree of renal impairment, age, and other risk factors such as diabetes mellitus and cardiac function. Since coronary artery disease (CAD) and CIN risk factors occur in the same elderly population and the two are closely interrelated (3), many patients subjected to CTCA may be at risk of CIN.

Vascular enhancement is dependent on a number of factors such as CM dose, injection rate, blood volume, and cardiac output (CO) (4–6). Blood volume is related to body weight. By dosing CM in relation to body weight and using a fixed injection duration, a fixed injected dose rate (mg I/kg/s) is obtained and vascular enhancement becomes essentially unrelated to body size (4, 6, 7). However, arterial enhancement

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increases with decreasing CO (4) and at the same time poor cardiac function is an independent risk factor for CIN (2). Knowing in advance that cardiac function is poor means that the CM dose could be reduced, thereby decreasing the risk of CIN while preserving diagnostic vascular enhancement. On the other hand, a patient with no CIN risk factors and hyperkinetic circulation may need and tolerate a higher CM dose than normal to achieve diagnostic quality without jeopardizing renal function.

Test injections of CM have been used for three purposes: determination of circulation time for correct scan start, CO measurement, and prediction of CM enhancement (8, 9). This test dose, e.g. 20 ml 370 mg I/ml (7.4 g I), may constitute about 15–25% of the diagnostic dose (8, 9) and contribute to nephrotoxicity in patients with multiple CIN risk factors. Correct timing may be achieved without test injections using CT software packages automatically triggering scan start by arrival of the CM to the area of interest (10). Thus, what remains to add is a simple noninvasive and clinically acceptable method that can be handled by the radiology staff at site to evaluate CO immediately before CTCA. Both the classic Fick method and thermodilution are invasive procedures and Doppler ultrasound techniques require a trained operator. However, classical impedance cardiography (ICG) modified with a novel technique to measure CO, electrical velocimetry (CO_{EV}) (11–13), has the potential to fulfill our demands in the CT suite.

The primary purpose of this feasibility study was to evaluate how coronary artery attenuation, adjusted to injected CM dose rate, correlates with CO_{EV} measured by radiology staff immediately before CTCA.

Material and Methods

Patients

From October 2007 to January 2008, 27 consecutive patients with presumed CAD (13 females), being part of a prospective study regarding both planned and emergency CTCA, were included in this study. Demographic and anthropometric patient data are given in Table 1. Exclusion criteria were cardiac arrhythmia, renal impairment (serum creatinine above reference level), known CM hypersensitivity, hyperthyroidism, women of child-bearing potential, and inability to participate in measuring body height and weight. The study was approved by the local ethical committee and performed in compliance with the ethical principles for medical research involving human subjects established in the Helsinki Declaration of 1975 and as revised in 2004 (http://www.wma.net/en/30publications/10policies/b3/index.html). Informed consent was obtained from all patients.

Table 1. Demographic and anthropometric patient data

Parameter	Median (1st and 3rd quartile)	Range
Age (years)	63 (56, 73)	28–80
Body weight (kg)	82 (68, 93)	56–131
Body height (cm)	167 (164, 177)	152–190
Body mass index (kg/m^2)	27 (26, 30)	21–41

CT coronary arteriography

CTCA was performed using 64-multirow detector equipment (LightSpeed VCT, GE Healthcare, Milwaukee, Wisc., USA). CM were injected into an antecubital vein via a 1.2 mm (18 gauge outer diameter) catheter (BD Venflon™ Pro, Becton Dickinson Infusion Therapy AB, Helsingborg, Sweden) and using a power injector (Medrad Stellant®, Medrad Sweden AB, Västra Frölunda, Sweden).

Automatic bolus tracking for correct timing between CM bolus arrival to the coronary arteries and CTCA was not used in the present study, since a test bolus injection was the established routine by this time. Twenty ml of iodixanol 320 mg I/ml (Visipaque 320, GE Healthcare, Solna, Sweden) was injected as a test bolus into an antecubital vein with the same injection rate as later planned in the diagnostic examination (see below), followed by a 40 ml saline chaser. The time from the injection start to the maximum enhancement in the aorta (time-to-peak) was used to calculate the delay time at CTCA.

A two-phase CM injection regime was used. For the CTCA, iodixanol 320 mg I/ml was injected at a dose of 260 mg I/kg with a maximum dose set at weight 80 kg for women and 90 kg for men. A fixed injection time of 12 s was used, resulting in an injected dose rate of 21.7 mg I/kg/s. The CM volume and injection rate were calculated using a dedicated computer program (OmniJect, distributed in Nordic countries by GE Healthcare, Sweden). Immediately after this CM injection a 40/60% CM/saline mixture was injected at the same rate as the former injection and followed by a 40 ml saline chaser. The intention of the second CM injection was to reduce enhancement in the superior vena cava and in the right half of the heart to avoid beam hardening artifacts during the CTCA but at the same time to keep some enhancement in the right half of the heart to be able to study the cardiac chambers and the septum.

The CTCA acquisitions were obtained with ECG-triggering in cranio-caudal direction during end-inspiratory breath-hold. Acquisition started after a delay equal to time-to-peak in the test bolus series plus 4 s. To achieve a stable heart rate the patients held their breath for an extra 5 s before the acquisition started. The acquisition time was approximately 6 s.

The acquisition parameters used were as follows: X-ray tube potential 100 kVp, ECG modulation (50–80%), tube current varied from 600 mA during the 50–80% R–R interval to 190 mA during the rest of the interval, gantry rotation time 350 ms, pitch 0.16–0.24 varying with heart rate, and collimation 64×0.625 mm.

Images were retrospectively reconstructed in 10% steps through 0–90% of the R–R interval using a field of view of 250 mm, a 512^2 matrix, and a standard type software kernel and thickness of 0.625 mm without overlap. Images were then transferred to a workstation (GE Advantage Workstation AW 4.3, GE Healthcare) for further processing.

Aortic attenuation measurements

Post-contrast signal intensity (Hounsfield units, HU) of the ascending aorta was used to represent the attenuation of the coronary arteries. It was measured in a circular region of interest (ROI) in an image without visible artefacts in the ascending aorta, approximately 3–5 cm above the origin of the coronary arteries. The diameter of the ROI was about half the diameter of the ascending aorta to minimize possible partial volume effects.

CO measurements

ICG based on EV (Electrical Velocimetry™, Aesculon® Cardiac Output Monitor, size 29×31×19 cm, weight 6 kg, Osypka Medical GmbH, Berlin, Germany) was used to measure CO_{EV}. ICG measures cardiac stroke volume based on variations in thoracic electrical bioimpedance (TEB; impedance = resistance in an alternating current circuit) due to changes in thoracic conductivity during the cardiac cycle. Multiplying stroke volume with heart rate then gives the CO value.

In classic ICG, the origin of the rapid changes of thoracic conductivity lies in the volumetric changes of the ascending aorta (Windkessel effect) (14). According to the new technique, EV, the characteristic changes in TEB are caused by alternating orientation of the red blood cells (RBCs) in the aorta during systole and diastole (11, 15). This induces a pulsatile increase and decrease in electrical conductivity of the blood (the most highly conductive substance in the thorax), which is reflected in a decrease in TEB during systole and increase during diastole registered by ECG electrodes.

Two ECG electrodes are attached on the left side of the body, one at the base of the neck and one at the inferior aspect of the thorax (http://www.osypkamed.com). A high frequency (50 kHz) low amperage (2 mA) alternating electrical current is then applied across the left side of the thorax in the direction of the body axis via these two so-called stimulating electrodes. This current (I) application causes a voltage (E), that is registered by two sensing electrodes, placed 5 cm below and above the upper and lower stimulating electrodes, respectively. Changes in thoracic impedance (Z) during the cardiac cycle cause voltage variations between the sensing electrodes. The ratio of the varying voltage (E) to the applied current (R) thus equals changes in TEB (Z = E/I according to Ohm's law). The maximum rate of change of TEB is interpreted as the ohmic equivalent of the mean blood flow velocity in the ascending aorta during left ventricular ejection and stroke volume is calculated via mathematical equations (14–16). ECG and heart rate are simultaneously recorded on the cardiac output monitor.

Stroke volume and heart rate were measured simultaneously three times for 30 s immediately before the CTCA. Aesculon® calculates CO_{EV} automatically by multiplying stroke volume with heart rate. The mean value of the three measurements was registered for each patient. Body weight, body height, and age were used for stroke volume correction by the Aesculon® software.

Aortic attenuation adjusted to the injected dose rate (HU per mg I/kg/s) was then correlated to CO_{EV} in each patient. The correlation was made in relation to the first phase CM dose of 260 mg I/kg, assuming that the diluted second phase CM dose had not reached the coronary arteries during CTCA.

Statistical analysis

All data were expressed as median, first and third quartile, and range. Simple linear regression analysis (with only one independent variable at a time) was performed to explore (i) the correlation between aortic attenuation (HU) and body weight, (ii) the influence of CO_{EV} on aortic attenuation adjusted for injected CM dose rate (HU per mg I/kg/s), and (iii) to establish a tentative formula on how to adapt CM injection parameters to CO and desired aortic attenuation. Data were analyzed in SPSS 15.0 for Windows (SPSS Inc., Chicago, Ill., USA). A P value <0.05 was considered statistically significant.

Results

All examinations were successfully accomplished. The CM injection parameters, aortic attenuation, CO_{EV}, and heart rate are summarized in Table 2.

A weak and nonsignificant negative linear correlation was found between aortic attenuation and body weight (Pearson's coefficient of correlation $r=-0.32$, $P=0.10$). The coefficient of determination (r^2) was 0.10, indicating that body weight only explained 10% of the variation in aortic attenuation. If the patient

Table 2. Results for contrast medium injection parameters, aortic attenuation, cardiac output, and heart rate

Parameter	Median (1st and 3rd quartile)	Range
Contrast media		
mg I/kg	260 (250, 262)	176–303
gram iodine	21 (18, 23)	17–24
volume (ml)	65 (56, 73)	53–75
injection rate (ml/s)	5.4 (4.7, 6.0)	4.4–6.1
dose rate (mg I/kg/s)	22 (21, 22)	15–25
Ascending aorta		
attenuation (HU)	447 (372, 518)	280–585
attenuation related to dose rate (HU per mg I/kg/s)	21 (17, 24)	14–27
Heart rate (beats per minute)	68 (60, 77)	44–127
Cardiac output (L/min)	6.5 (5.1, 6.7)	2.4–11.5

weighing 131 kg was excluded, r^2 decreased to 0.02 (r=–0.14; P=0.49, Fig. 1).

A significant negative linear correlation (r=–0.63, r^2=0.40, P<0.001) was found between aortic attenuation adjusted for injected CM dose rate and CO_{EV} (no exclusion of extreme values necessary, Fig. 2) resulting in the following relationship (values within parentheses = 95% confidence intervals, CI):

$$\text{adjusted aortic attenuation (HU per mg I/kg/s)} = 28^{(24\text{ to }32)} - 1.2^{(-1.8\text{ to }-0.57)} \times CO_{EV}\text{ (L/min)}$$

A significant positive linear correlation (P<0.001) was found between aortic attenuation (HU) and CM dose rate/CO_{EV} (mg I/kg/s per L/min). After exclusion of one outlier (9 mg I/kg/s per L/min), r^2 rose from 0.42 (r=0.65) to 0.49 (r=0.70) and resulted in the following linear regression equation (values within parentheses = 95% CI) (Fig. 3):

$$\text{aortic attenuation (HU)} = 240^{(150\text{ to }330)} + 55^{(31\text{ to }78)} \times \text{CM dose rate}/CO_{EV}$$

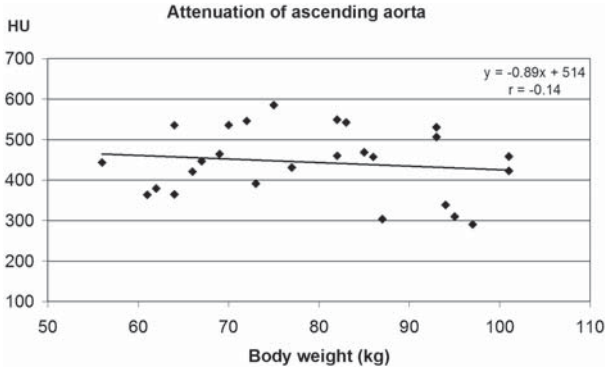

Fig. 1. Attenuation of the ascending aorta in Hounsfield units (HU) as a linear function of body weight during CT of coronary arteries in 26 patients after exclusion of 1 outlier weighing 131 kg.

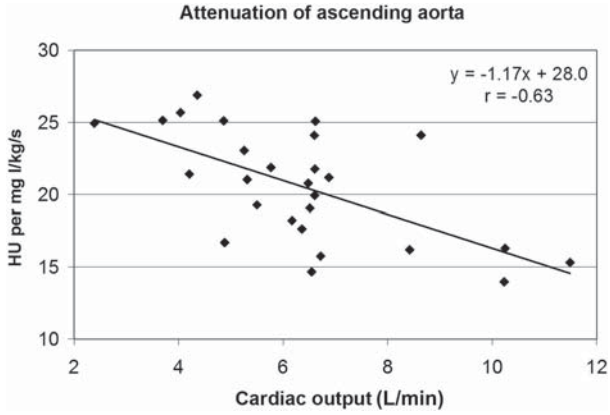

Fig. 2. Attenuation of the ascending aorta in Hounsfield units (HU) adjusted to injected contrast medium dose rate (mg I/kg/s) as a linear function of cardiac output measured with electrical velocimetry during CT of coronary arteries in 27 patients.

Thus, if the goal is to reach an aortic attenuation of, for example, 438 HU (present mean value), it leads to the following expression:

$$438 = 240 + 55 \times \text{CM dose rate}/CO_{EV}$$

Solving the equation for CM dose rate yields the following formula:

$$\text{CM dose rate} = CO_{EV} \times (438–240)/55$$

Based on this model, Table 3 demonstrates how the CM injection parameters may be calculated dependent on CO.

Discussion

CO is a key physiologic factor that affects arterial enhancement, particularly in the first pass dynamics (4, 6, 17). In conditions with reduced CO, blood flow slows down resulting in less dispersion of the CM bolus as well as slower inflow of nonCM-enhanced

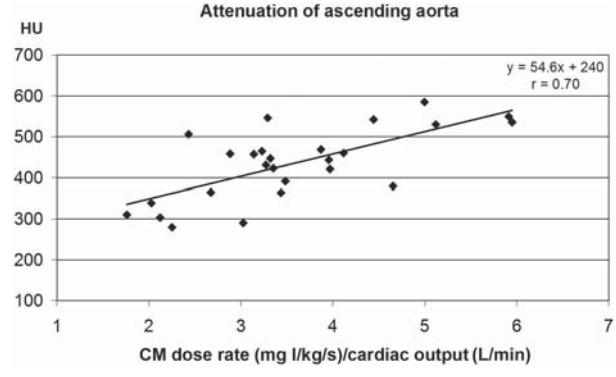

Fig. 3. Attenuation of the ascending aorta in Hounsfield units (HU) as a linear function of contrast medium (CM) dose rate divided by cardiac output measured with electrical velocimetry during CT of coronary arteries in 26 patients after exclusion of 1 outlier (see Results).

Table 3. Calculation of contrast medium (CM) injection parameters*

Cardiac output (L/min)	Aortic attenuation† (HU)	Intercept (HU)	Slope	CM parameters						
				Dose rate (mg I/kg/s)	Injection time (s)	Dose per kg (mg I/kg)	Total dose (gram I)	Concentration (mg I/ml)	Volume (ml)	Injection rate (ml/s)
2	438	240	55	7.2	12	86	7	320	22	1.8
5	438	240	55	18	12	216	17	320	55	4.6
8	438	240	55	29	12	348	28	320	87	7.3

*Calculations were made after exclusion of one outlier (n=26) at various cardiac outputs (2, 5, and 8 ml/min) based on the present linear regression equation: aortic attenuation (438) = 240 (intercept) + 55 (slope) × CM dose rate/cardiac output. Accordingly, CM dose rate in mg I/kg per second equals cardiac output × (438–240)/55. Using a fixed injection time of 12 s, a body weight of 81 kg (mean value) and a CM concentration of 320 mg I/ml as in the present study, total CM dose (CM dose rate × body weight × injection time) and CM volume (total CM dose/concentration), and CM injection rate (CM volume/injection time) can be calculated.
†Mean value in the present study.

blood from the ipsilateral jugular vein, contralateral brachiocephalic vein, and inferior vena cava causing less dilution of the bolus. Hence, vascular enhancement is inversely related to CO.

In the present study we found a good correlation (r=–0.63) between increasing aortic attenuation and decreasing CO_{EV}. HUSMANN et al. (10) found a similar negative correlation between the attenuation in the left main (r=–0.56) and right (r=–0.42) coronary artery on one side and CO based on CT measurements of the end-diastolic and -systolic volumes on the other side. These results combined with our tentative statistical model indicate a possibility to predict CM injection parameters by nonCM-based noninvasive CO measurements. The mathematics can be easily solved with a spreadsheet.

HUSMANN et al. (10) concluded that "adaptation of CM regimen to the actual patient's hemodynamic condition, especially in cases of high CO, may be of potential benefit". Such a patient may require increased CM doses and/or injection rates to obtain adequate vascular enhancement. The other extreme includes patients with a low CO and an increased risk of CIN. Prophylactic hydration to avoid CIN in patients with poor cardiac function might be problematic due to the risk of overhydration. However, knowing that the patient has a poor CO means that the CM dose may be markedly reduced. This may offer the best protection against CIN while preserving diagnostic quality. According to the present statistical model a patient with a reduced CO of 2 L/min may need a total dose of only 7 g of iodine, compared with 18 and 28 g of iodine at a CO of 5 and 8 L/min, respectively, for the same aortic enhancement. Injecting 22 ml (7 g I), a dose similar to that of a test bolus (8, 9), at a rate of 1.8 s may seem insufficient for adequate enhancement. However, similar injection parameters have been used during 80 kVp CT pulmonary angiography with satisfactory enhancement to diagnose pulmonary embolism in elderly patients with moderate to severe renal impairment and presumably decreased CO (18).

A relatively wide variation in aortic attenuation occurred despite the use of a constant CM dose rate related to body weight. Apart from imprecision in CO methodology, this may be explained by the fact that body weight is only a rough estimate of patients' blood volume. Body weight may be the same despite wide variation in body composition, i.e. metabolically active muscle mass contra metabolically inactive fatty tissue, a relationship affecting blood volume and which may be better predicted by body surface area, weight-height equations or lean body mass (19–21). Variation in hydration state and other physiological and pathological parameters may also affect enhancement.

The interest in impedance technology to measure CO was introduced during the 1960s (11). Although ICG is technically straightforward, its use in clinical practice during several decades resulted in much controversy about its validity because of varying results, especially in comparison with thermodilution to estimate CO (CO_{TD}) (11). However, a true gold standard does not exist for CO measurements, only technologies that measure a quantity indirectly related to CO. Therefore evaluation of a new technology has to be made in comparison with an existing accepted technology, such as thermodilution, with its own inherent sources of error. Such comparisons have included correlation and regression analysis, Bland-Altman analysis for bias and precision statistics (22), and "percentage error" recommended to be <30% (23).

Meta-analysis of studies in cardiac patients using classical ICG methods shows an overall correlation coefficient of 0.77 (95% CI 0.71–0.82) when compared with thermodilution (24). In another meta-analysis comparing classical ICG with other techniques of CO measurements the overall bias was +0.6 L/min and "percentage error" was >30% (mean 37%) in all but

one study (23). However, it was pointed out that, except for patients with "wet lung", the bioimpedance method was more accurate than the current Doppler technique (mean error of 60%).

Advances in ICG hardware and software, particularly improved digital signal processing, refined bioimpedance equations, and improved artifact rejection, have enabled better and more reliable CO determinations in studies from the early 2000s as well as superior intra-patient reproducibility compared with CO_{TD} (16, 25). Stout et al. demonstrated no interference of "wet lungs" and body mass index ≥30 with impedance CO measurements (26).

In this study we used the latest TEB technology based on the Bernstein-Osypka equation, EV (15). EV focuses on the changes in the compartment with the greatest conductivity, the aortic blood, while high resistance, low conductivity compartments such as the lung, gas, and surrounding tissues are neglected (11). Thus, in contrast to classic ICG, this novel technique is expected to provide more accurate information on CO independent of the volume of the surrounding tissues (11) or of increased thorax fluid content (12). In fact, recent studies also demonstrate a close correlation, minimal bias, and clinically acceptable limits of agreement (<30%) between CO_{EV} and CO_{TD} (13) or CO measured with transesophageal Doppler echocardiography (11) including both hemodynamically stable and unstable patients (12) (Table 4). However, it should be noted that TEB as a measure of CO may be unreliable in cases of, for example, aortic valve regurgitation, conditions significantly affecting the shape and alignment of RBCs (high heart rates), pulmonary hypertension decreasing the relative share of the aorta in contributing to the impedance change, conditions creating poor electrode skin contact, motion artifacts close to the electrode locations (e.g. turning of the head), high frequency (>10 kHz) current applications to the body such as measurement of the respiration rate, other impedance-based instruments, and electrical interference from external sources (Aesculon® Instructions for Use, e-mail communication Dr Markus Osypka).

Although cardiac function may be evaluated with conventional echocardiography before CTCA in a patient at high risk of CIN, the technique generally demands an experienced operator outside the radiology department, usually not available round the clock. The EV system (Aesculon®) does not require an experienced operator, is simple to use, involves only the application of standard ECG electrodes and is quickly performed. A smaller and cheaper handheld device using the same technique for CO_{EV} measurement is now available (Icon®, Osypka Medical GmbH, Berlin, Germany).

Table 4. Published results in peer-reviewed journals of electrical velocimetry to measure cardiac output (CO_{EV}) or cardiac index (CO normalized to body surface area) with regard to Bland-Altman analysis (bias, limits of agreement, and percentage error) (22) and Pearson's coefficient of correlation in comparison with thermodilution (TD) and transesophageal Doppler echocardiography (TOE)

First author, year	Patient category	Not included	*n*	Comparative method	Bias*	Limits of agreement†	Percentage error‡	Correlation
Zoremba, 2007 (13)	Critically ill postsurgical	Arrhythmias Left heart valve dysfunction	25	Pulmonary artery TD	–0.05 L/min	±1.42 L/min	27%	NR
Zoremba, 2007 (13)	Critically ill postsurgical	Arrhythmias Left heart valve dysfunction	25	Femoral artery TD	0.22 L/min	±1.56 L/min	26%	NR
Suttner, 2006 (12)	Post cardiac surgery, hemodynamically stable	Atrial fibrillation Hemodialysis Left heart valve dysfunction	40	Pulmonary artery TD	0.02 L/min/m²	±0.47 L/min/m²	19%	0.86
Suttner, 2006 (12)	Post cardiac surgery, hemodynamically unstable	Atrial fibrillation Hemodialysis Left heart valve dysfunction	34	Pulmonary artery TD	0.06 L/min/m²	±0.68 L/min/m²	28%	0.79
Schmidt, 2005 (11)	Preoperative coronary bypass surgery	Atrial fibrillation Left heart valve dysfunction	37	TOE	0.18 L/min	±1.18 L/min	29%	0.93

NR, not reported .

∗The difference between CO_{EV} and CO_{TD} or CO_{TOE} plotted against the arithmetic mean of CO values of both measurements.

†Two standard deviations of the mean bias value, i.e. the range including 95% of the bias measurements.

‡Two standard deviations of the mean bias value divided by the arithmetic mean value of CO_{EV} and CO_{TD} or CO_{TOE}; an error ≤30% is regarded as clinically acceptable (23).

Thus, measurements can be performed immediately before CTCA at any time in the CT suite by the radiology staff. This has the additional advantage that the measured CO will reflect cardiac function at the time of the CM injection. A CO measured by echocardiography ≥1 h before CTCA may result in inadequate CM injection parameters, since CO is highly dependent on pulse rate, which may rapidly change for a number of reasons.

Finally, it should be noted that the principle of adjusting CM dose to CO should be applicable to CT of any of the aortic branches. Regarding parenchymal enhancement such as the liver, CO does not affect the peak hepatic enhancement but only the time-to-peak, which may be controlled by applying available CT software, i.e. automatic scan start triggered by the CM bolus (17).

The present study has several limitations. Obviously, the number of patients is small as the study was designed primarily to examine if it was at all feasible to correlate CO_{EV} measured in the CT suite with aortic attenuation. There is also the possibility of CO measurement error, as already discussed.

The present formula for adjustment of CM injection parameters to CO is tentative and dependent on the present CM injection principles. Before using such a model for general purposes it should be based on a substantially larger population and also has to be validated in a prospective study.

Arterial attenuation was not measured in the coronary arteries but in the ascending aorta. This point of measurement was chosen since the far smaller size of the coronary arteries makes them more susceptible to partial volume effects. Since the contrast-enhanced blood in the ascending aorta represents the blood that drains into the coronary arteries during diastole, the attenuation in the ascending aorta and coronary arteries should be equal.

In conclusion, it seems feasible to use EV as a handy technique for radiology staff to measure CO in the CT suite immediately before vascular examinations. Hence, CM doses may be decreased in low output states to reduce the risk of CIN without jeopardizing diagnostic quality and may be increased in high CO states to avoid poor arterial enhancement.

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