Thoracocardiography*

Part 1: Noninvasive Measurement of Changes in Stroke Volume Comparisons to Thermo- dilution

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The thoracocardiograph (TCG) is a new noninvasive monitoring device that measures cardiac oscillations transmitted to the external surface of the thorax. It consists of 2.5 cm in height, elastic inductive plethysmographic transducers placed transversely in the proximity of the xiphoid process to provide changes in cross-sectional area on a transverse plane across the minor ventricular axis. Cardiac oscillations synchronous with each heart beat are extracted from the respiratory signal during breathing with an ensemble-averaging technique using the electrocardiograph as a trigger pulse. The average cardiac waveform at locations near the xiphoid process in normal humans has the appearance of a ventricular volume curve. The latter is also found in the majority of patients with heart disease although in some, outward (dyskinetic) rather than inward motion during systole occurs at one or more locations of the TCG transducers. As in echocardiography, such findings are consistent with ischemic or scarred myocardium invalidating computation of changes in stroke volume from such sites. In anesthetized dogs and critically ill patients with normal ventricular wall motion, changes in TCG derived ventricular volume waveform amplitudes agreed well with changes of thermodilution estimates of stroke volume during atrial pacing and fluid loading in the dogs on the one hand and with application of extrinsic positive end-expiratory pressure (PEEP) in patients on the other hand. Thoracocardiography has the potential for noninvasive, continuous monitoring of stroke volume and cardiac output as well as for detection of ischemic or scarred myocardium.

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TCG = thoracocardiography; TD = thermodilution; CO = cardiac output; ANOVA = analysis of variance; VVC = ventricular volume curve; ID = indicator dilution

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The ventricular volume waveform is a parameter potentially available for continuous, noninvasive monitoring of mechanical cardiac function. Its amplitude represents stroke volume (SV), which together with heart rate, allows computation of cardiac output. Analog recordings of the left ventricular volume curve can be obtained with digitized echocardiography and radionuclide ventriculography, but postural constraints, labor-intensiveness, dependence on highly trained personnel, costliness of equipment, and radiation hazards (for nuclear scans) limit their application to the diagnostic laboratory.

Cardiogenic oscillations, amounting to about 1 to 5 percent of total signal content, are contained within the analog breath waveform from such external respiratory monitoring devices as the bellows pneumograph, mercury in Silastic strain gauge, and respiratory inductive plethysmograph. The significance of such oscillations has not heretofore been understood. By placing transducers of these devices at or near the xiphoid process and eliminating the respiratory signal with breath holding or an ensemble-averaging procedure incorporating the electrocardiograph as a trigger pulse, the configuration of cardiogenic oscillations appears quite similar to ventricular volume waveform recordings obtained with low frequency, precordial movement and fluorodensitometric devices, as well as external length gauges placed directly on the heart.

We investigated one of these devices, ie, the respiratory inductive plethysmograph, because it measures changes in cross-sectional area of the chest wall. Since height of a single transducer band multiplied by cross-sectional area is proportional to change of segmental thoracic volume, we reasoned that such a signal might provide quantitative estimates of stroke volume by subtending a segmental ventricular volume that could reflect global ventricular performance in a way analogous to echocardiography. We compared thoracocardiographic (TCG) derived changes of stroke volumes and cardiac outputs with values from the established thermodilution (TD) method in anesthetized dogs and critically ill patients.

METHODS

Preparation

Dogs: Mongrel dogs were anesthetized with intravenous administration of pentobarbital sodium (25 mg/kg), intubated with No. 8 F cuffed oral endotracheal tube, and connected to a mechanical ventilator. They were paralyzed with intravenous injection of 1 ml of succinylcholine solution and maintained anesthetized and paralyzed throughout the experiment. A No. 7 F pulmonary artery
directed TD catheter inserted into the internal jugular vein was connected to a TD cardiac output (CO) computer system (model 952 with CO-SET injection system, American Edwards Laboratory, Santa Ana, CA). Five milliliters of iced saline solution was used as the injectate. A 3.5 F bipolar electrode catheter was passed along side of the flow-directed TD catheter for pacing the right atrium.

Two TCG transducers, viz, elastic cloth bands, 2.5 cm in height, which had a sinusoidal length of insulated wire sewn onto them (Non-Invasive Monitoring Systems, Inc [NIMS], Miami Beach, FL) were placed so as to totally encircle the lower rib cage at the transverse level of the xiphoid process and just below it. The wires from these transducers were connected to oscillator modules that in turn were led to a two-channel demodulator/amplifier unit (Respirtrak, NIMS). Frequency response was linear from DC to 100 Hz but a low-pass filter for noise reduction on its output side limited response to 15 Hz. Its signals along with the electrocardiograph were led to a polygraph recorder and then to a 286 based personal computer.

Critically Ill Patients: In supine patients who were intubated and supported by mechanical ventilation and had a flow-directed TD catheter located within the pulmonary artery for clinical management, a TCG transducer was placed just 3 cm caudal to the xiphoid process (designated TCG-Reference) and another one 6 cm cephalad to it (TCG +6 cm) (Fig 1). It had been established in normal humans that changes of SV could be best determined from TCG transducers encircling the thorax 3 cm caudal to the xiphoid process (TCG-Reference), at the xiphoid process (TCG + 3 cm), and 6 cm cephalad to TCG-Reference (TCG + 6 cm).12 The study was carried out in an ongoing quality control of ventilator management of critically ill patients and received approval by the institutional research committee.

Auto-positive end-expiratory pressure (PEEP) levels were determined with respiratory inductive plethysmography.13 Changes of SV and CO estimated with TCG were compared with TD measurements during 2.5 to 5 cm H2O increments in extrinsic PEEP up to a maximum of 25 cm H2O. Following initial TCG estimation of CO, three TD determinations of CO employing injections of 10 ml of 5 percent glucose solution at room temperature were carried out at end-expiration through the proximal port of a flow-directed catheter. The three TD CO values were averaged and if any deviated greater than 10 percent from mean, then the following was carried out. Two additional runs were made and the high and low values of the five were discarded and the remaining three averaged. Following completion of the TD measurements, the CO was measured with TCG and the duplicate values for this method were averaged. If the variation was greater than 10 percent from the mean, then three additional TCG estimates were carried out, the low and high runs were discarded, and the remaining three were averaged. Values for TD and TCG estimations of CO were available to the investigators shortly after the measurements were obtained. The aforementioned procedure of additional determinations was necessary in only four runs from 2 of the 23 patients studied.

Data Processing

The electrocardiogram was displayed on the videoscreeen of the computer that set a horizontal line to serve as a threshold to discriminate the R from the T wave with the portion of the R wave above the threshold being the trigger pulse. This horizontal threshold could also be adjusted through the keyboard. The program accepted analog signals at 500 pts/s and displayed them in real time on the videoscreeen during data collection.

Extraction of the cardiogenic oscillations from the respiratory waveforms was achieved with an ensemble-averaging procedure.14 The periodic portion considered was the heart beat and the random portion of the signal the respiratory waveform. The onset of the sampling cycle was triggered by the R wave of the electrocardiogram. The operator selected the number of heart beats to be averaged to best minimize or eliminate the respiratory waveform. This ranged from 32 to 150 heart beats with most data being collected with a 32- to 50-beat average. Selection of greater number of heart beats for averaging was done only if a significant portion of the respiratory waveform contaminated the cardiac signal as indicated by upward or downward drift of the display of the averaged ventricular volume waveform.

After completion of the data collection, up to four channels of averaged TCG waveforms along with an averaged electrocardiographic waveform were displayed on the videoscreeen of the computer. The program selected the maximal and minimal amplitudes of the TCG waveforms and denoted these points with a hatched vertical line. Data were displayed either to the full amplitude of the channel height through an autoscaling technique or this option could be suppressed and other scaling factors could be assigned to the vertical axis.

The operator could calibrate the TCG ventricular volume waveform from arbitrary computer units to an absolute value by inputting concurrent values of SV from the TD method. Once having set the baseline value, this calibration value was stored in memory and applied to subsequent curves to provide changes of SV in absolute values over time and with pharmacologic or mechanical interventions. Changes of SV (TCG) with repetitive measurements could also be obtained by arbitrarily designating a value such as 100 to the baseline measurement and subsequent values computed as a percentage of baseline control. Either method gave quantitative changes in SV. The TCG waveform could not be calibrated to absolute volumes apart from using a concurrent method that provided such a value, but the latter was not needed for solely assessing changes of SV(TCG) and CO(TCG) over time or with interventions.

Statistical Analysis: Two-way analysis of variance (ANOVA) with repeated measures and frequency histograms were used. Level of statistical significance was \( p < 0.05 \).

Protocols

Anesthetized Dogs: These studies were carried out in six anesthetized, paralyzed, supine dogs weighing between 20 and 30 kg. Atrial pacing rates were instituted over baseline at approximately 20-beat increments from 155 to 246 beats for three to four runs. Complete capture of the atrium with the pacing stimulus was accomplished in four of the six dogs and constitutes the data to be reported.

After completion of the atrial pacing experiments, another baseline calibration of TCG with TD was obtained and 100- to 200-ml aliquots of hyperosmolar dextan solution were infused intrave-
FIGURE 2. Representative thoracocardiographic (TCG) waveforms obtained with the ensemble-averaging computer program during normal sinus rhythm and rapid atrial pacing. The ventricular volume curve (VVC) configurations in the anesthetized dog for the two cardiac rhythms are similar. The greater drift during the atrial pacing runs relates to the incomplete elimination of the respiratory waveform by the ensemble-averaging process.

nously within a minute or two. After an interval of 10 to 15 minutes, duplicate measurements of TCG and TD were obtained. Each dog received at least 400 ml of hyperosmolar dextran solution with a maximum up to 1,200 ml in one animal.

Critically Ill Patients: This was designed to compare TCG with TD estimates of SV and CO after application of 2.5 to 5 cm H2O increments of PEEP in intubated, mechanically ventilated, critically ill patients who had flow-directed TD catheters inserted for clinical management. Eleven men (mean age, 76 years; SD, 12 years) and 12 women (mean age, 70 years; SD, 12 years), all in sinus rhythm, comprised the study population. Two patients were studied on two occasions separated by a few weeks interval when the major diagnosis changed. Eight of the patients had ischemic heart disease and six had mitral regurgitation. The SV(TCG) was determined

FIGURE 3. Schematic of changes in intracardiac pressure along with fluctuations in ventricular volume as measured with a plethysmographic chamber placed around the ventricles* (by permission of author).
RESULTS

Anesthetized Dogs

Figure 2 depicts autoscaled ensemble-averaged ventricular volume curve (VVC) from the TCG transducer placed at the transverse level of xiphoid process during baseline and atrial pacing. These waveforms are comparable to those waveforms obtained by direct plethysmographic recording of canine VVCs (Fig 3). The general shape of VVC was maintained during atrial pacing. The waveform from the transducer placed just below the xiphoid often showed an outward movement toward the end of systole suggestive of contamination by the abdominal aortic pressure pulse and was not analyzed. Both TCG and TD estimates of SV fell with paced rapid atrial rates and there was no significant difference in SV between the methods. Maximal fall of SV(TCG) stroke volume from baseline ranged from 21 to 58 percent and for SV(TD) 6 percent to 63 percent in individual dogs. Cardiac outputs during atrial pacing at rates greater than 200 beats per minute were lower than baseline values in all instances (Fig 4).

Figure 5 depicts representative raw analog waveforms during hyperosmolar dextran infusion, viz., electrocardiogram and respiratory waveform (VT), including TCG. It shows that the amplitude of cardio- genic oscillations at end-expiration increased with dextran infusion. Figure 6 shows the ensemble-averaged TCG waveforms with increasing aliquots of dextran; these revealed an earlier rise to the end-

from the TCG-Reference location in all but one patient in whom TCG + 6 was used since TCG-Reference gave a dyskinetic waveform. The TCG was employed in an additional five patients with ischemic heart disease, but changes of CO could not be assessed because dyskinetic systolic deflections of the ventricular volume curves were present at both initial TCG locations as well as at all locations over the precordium.

![Image](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21625/)

**Figure 4.** Cardiac output during rapid atrial pacing in four anesthetized dogs as estimated by thermodilution (TD) and thoracocardiographic (TCG) methods. The trends for both methods are similar, viz., cardiac output remains constant or decreases as atrial pacing rate increases thereby signifying a fall of stroke volume (SV).

![Image](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21625/)

**Figure 5.** Electrocardiograph (EKG) and combined respiratory and thoracocardiographic (TCG) waveforms (VT + TCG) in anesthetized dog during mechanical ventilation. Arrows designate TCG waveforms that correlate to EKG period. These oscillations superimposed on VT were most prominent at end-expiration. Their amplitude increased with 10 percent dextran 40 infusion at both TCG transducer sites but to a greater extent at the xiphoid process.
diastolic volume level, thereby rendering the isovolumetric contraction period at beginning systole less prominent.

For combined pacing and fluid expansion experiments, excluding baseline calibration values, 90 percent of SV(TCG) values fell within ±20 percent of the SV(TD) (Fig 7). Values of heart rate, SV, and CO in all six dogs after 200 ml and 400 ml of hyperosmolar dextran infusion are listed in Table 1. There were no significant differences among heart rates at baseline and after hyperosmolar dextran infusion. The SV(TCG) and CO(TCG) showed a graded increase proportional to the hyperosmolar dextran infusion volume but corresponding values of SV(TD) and CO(TD) did not differ at the 200- and 400-ml levels.

**Critically Ill Patients**

Figure 8 depicts normal-appearing ventricular volume waveforms in a critically ill patient at TCG-Reference during application of extrinsic PEEP; abnormal or dyskinetic waveforms are present at TCG +6 cm location. Figure 9 depicts identity plots of SV(TCG) vs SV(TD) and CO(TCG) vs CO(TD) after application of extrinsic PEEP with mechanical ventilator. Initial baseline values are omitted from analysis since SV(TCG) was calibrated to equal the value of SV(TD). Cumulative frequency histographic values are listed in Table 2. Seventy-nine percent of SV(TCG) and 82 percent of CO(TCG) values fell within ±20 percent of corresponding TD values.

Complete data in 15 patients were available for assessing extrinsic PEEP on CO at levels of 5 to 9 and
Table 1—Effect of Hyperosmolar Dextran Infusion on Hemodynamics in Dogs*  

<table>
<thead>
<tr>
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<th>Heart Rate, Beats/min</th>
<th>Stroke Volume, ml</th>
<th>Cardiac Output, L/min</th>
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<tr>
<td></td>
<td>TCG</td>
<td>TD</td>
<td>TCG</td>
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<tr>
<td>Baseline</td>
<td>133 (15)</td>
<td>34 (8)</td>
<td>34 (8)</td>
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<tr>
<td>200 ml dextran</td>
<td>124 (23)</td>
<td>45 (7)†</td>
<td>46 (6)†</td>
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<td>400 ml dextran</td>
<td>127 (11)</td>
<td>56 (14)‡</td>
<td>50 (14)‡</td>
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*Six anesthetized dogs, means (SD). TCG = thoracocardiography; TD = thermodilution.
†p<0.05; means significantly greater than baseline.
‡p<0.05; means significantly different between 200 ml and 400 ml values.

10 to 14 cm H₂O above the auto-PEEP level. The mean baseline auto-PEEP value was 5 cm H₂O, SD 3 cm H₂O, and mean pulmonary capillary wedge pressure was 17 mm Hg, SD 7 mm Hg. Neither TD nor TCG CO were affected by extrinsic PEEP at levels of 5 to 9 cm H₂O (p>0.05). There was a 10 percent fall of CO from the baseline value at or below the auto-PEEP determined with TD after application of extrinsic PEEP 10 to 14 cm H₂O (p<0.02) but a nonsignificant fall in CO of 3 percent measured with TCG (Fig 9).

**DISCUSSION**

*Theory*

The TCG transducer, which transversely overlies and encircles the heart, reflects changes of cross-sectional area across an oblique plane of the minor axis of a segment of the left, right, or both ventricles. Transformation of such a measurement into SV requires that ventricular motion during systole can be approximated with one degree of freedom. Since even the normal left ventricle does not contract or relax with completely uniform segmental motion, such an approximation is subject to an error that can only be evaluated by comparing changes of SV obtained with the TCG to a standard method.

Changes of external cross-sectional area of the ventricle have been used in dogs to measure SV. Hawthorn7 employed an extensible transducer sewn directly onto the left ventricle; the transducer sensed changes in mutual inductance proportional to changes of cross-sectional area which in turn produced a

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**Figure 8.** This depicts averaged ventricular volume waveforms from thoracocardiographic (TCG)-Reference and TCG + 6 cm in a patient with pneumonia and coronary artery disease. TCG-Reference shows normal systolic deflections up to positive end-expiratory pressure (PEEP) of 10 cm H₂O. TCG + 6 cm depicts dyskinetic or abnormal systolic deflections at these PEEP levels.
waveform closely resembling the ventricular volume curve. With the preceding transducer, Hawthorn and Appleyard and Glantz, using sonomicrometry, found that changes of external cross-sectional area in the plane of the left ventricular minor axis tracked changes in SV. Biplane ventricular angiograms of left ventricular geometry agree in principle with such observations. In normal or abnormal hearts, 80 to 86 percent of left ventricular SV can be accounted for by changes of the minor axis during systole; shortening of the major axis is responsible for the remainder.

Utilization of noninvasive transducers to measure changes of cross-sectional area of the ventricles for derivation of SV requires that ventricular movements be projected onto the chest wall in a similar or proportional distribution as present on the cardiac surface. Kinetocardiographic and cardiokymographic methods indicate that focal movements of variable amplitude and configuration that are periodic with the heart beat occur over the anterior and posterior surfaces of the entire chest wall. On a transverse plane of the thorax overlying the ventricles, such focal movements reveal waveforms resembling VVCs, arterial pressure waveforms, venous pressure waveforms, and combinations. Despite local differences in appearance, amplitude, and timing relationships of such waveforms, the TCG, which produces a weighted average of these movements, has given a ventricular volume waveform in all of more than 100 normal subjects studied in our laboratory. Therefore, there appears to be proportional transmission of ventricular wall movements to the thorax but not with well-defined, anatomic projections from the cardiac surface to the overlying thorax.

In patients with ischemic heart disease in the current investigation who had regional ventricular wall dyskinetic motion, the TCG performed like the kinetocardiograph. The latter depicts dyskinetic systolic movements in all patients having such an abnormality by echocardiography but with lack of correspondence between the methods regarding site and severity of such abnormalities. In this respect, the TCG has the same limitation of the echocardiograph in assessing overall cardiac function, ie, global assessment of ejection fraction or SV is inaccurate if measurements are obtained solely from abnormally contracting ventricular wall segments. On the other hand, the onset dyskinetic motion (change in polarity of systolic deflections) in the course of TCG monitoring would theoretically serve as an early detector of myocardial ischemia.

Comparison Of TCG Established Methods

Verification of the accuracy of SV estimations with the TCG depends on comparisons to established methods during hemodynamic alterations because SV cannot be obtained in absolute values without using an independent method that provides such a value. In the current study, TD measurements of SV and CO were taken as this method. Identity plots and cumu-
Relative frequency histograms between TCG and TD were employed to assess accuracy of the TCG method since data in the literature allow direct comparisons. Gribbe et al found that 67 percent of single plane ventricular cineangiographic estimation of SV in 36 trials in dogs and cats fell within ±20 percent of Fick CO. Ganz et al, in the course of right heart catheterization of 20 cardiac patients, observed that 100 percent of 63 comparisons of SV(TD) fell within ±20 percent of SV indicator dilution (ID) estimates. In 125 similar comparisons in ten patients immediately postoperative following open heart surgery, 82 percent of CO(TD) fell within ±20 percent of CO(ID). Hillis et al compared CO(ID) and CO(TD) with CO(Fick) in 808 patients undergoing elective cardiac catheterization. They analyzed their data using groupings of low CO (<2 L/min/sq m), high CO (>4 L/min/sq m), normal CO (2 to 4 L/min/sq m), and presence or absence of aortic or mitral regurgitation. Seventy-seven percent of CO(ID) and CO(TD) values in patients with low CO fell within ±20 percent of CO(Fick), 94 percent of values in patients with normal CO fell within ±20 percent of CO(Fick), 94 percent of values in patients with high CO fell within ±20 percent of CO(Fick), and 77 percent of values in patients with aortic or mitral regurgitation fell within ±20 percent of CO(Fick). The larger discrepancies between dye and TD methods vs Fick in low CO states and left-sided regurgitation lesions were attributed to heat loss of indicator due to low blood flow in the case of CO(TD) and distortion of the dye dilution curves with left-sided sampling in patients with mitral or aortic regurgitation.

All injections of indicator for CO(TD) in the critically ill patients on mechanical ventilators in the current study were made at end-expiration to ensure reproducibility of measurements. Stevens et al reported that CO(TD) did not vary in patients on mechanical ventilators when injections were carried out at end-expiration or end-inspiration. However, when injections were carried out at random throughout the respiratory cycle, values of CO were 10 percent less than those obtained with the fixed injection points.

The CO (TCG) measurements were obtained throughout several respiratory cycles over much greater time periods (five to ten times more heart beats were averaged) than CO(TD). This difference in mode and timing of collection of data for CO computations might contribute to the small discrepancy between TCG and TD in the current study. Further, six of the 23 critically ill patients in the study had mitral regurgitation that would have affected accuracy of TD and also rendered its comparison to TCG inappropriate since the former measures forward flow while the latter reflects both forward and backward flow.

In normal subjects, TCG transducer location in the vicinity of the xiphoid process is not crucially sensitive to detecting changes of SV and CO provided the transducer position remains fixed. In the current study in critically ill patients, TCG transducers were placed at transverse levels of the thorax using visual approximation of the transducer widths once the xiphoid process was palpated. Application of the TCG transducers took less than a minute in comparison to much lengthier times for accurate placement of echocardiographic probes prior to data collection.

The major limitations to TCG estimations of changes in SV and CO include cardiac arrhythmias and dyskinetic ventricular motion. Ensemble-averaging for extracting an average cardiogenic oscillation from the respiratory waveform during breathing holds only with a periodic or near-periodic cardiac rhythm. Breath holding and examining individual heart beats obviate this requirement but requesting critically ill patients and even some normal subjects to breath hold is impractical. We attempted to extract cardiac waveforms on a beat-by-beat basis from the respiratory waveform using bandpass digital filtering techniques but failed to achieve consistency owing to the overlapping frequency spectra of the cardiac and respiratory waveforms. We have had recent success in obtaining beat-to-beat cardiac waveforms in an off-line analysis using a combination of linear bandpass and adaptive digital filtering techniques and plan to implement this method in a real-time environment. This should then allow beat-by-beat SV determinations during tidal breathing despite cardiac arrhythmias.

The occurrence of dyskinetic ventricular volume waveforms on TCG recordings poses a different problem. If all precordial ventricular volume waveforms depict such a finding, then changes of SV cannot be estimated with TCG, although useful diagnostic information regarding abnormal wall motion is still available. With a combination of dyskinetic motion from one TCG transducer and normal motion at another TCG location, changes of SV can be estimated from the normally appearing waveform as accomplished in the critically ill-patient population of the current study. However, the validity of this approach over wide changes of SV requires confirmation since this is contrary to the assumption underlying the TCG method that the ventricle contracts with approximately one degree of freedom of motion.

Another problem with SV (TCG) includes distortion of the cardiac waveform by incomplete extraction from the respiratory waveform during the ensemble-averaging process. This occurs when the number of heart beats during inspiration differs widely from those during expiration as in widely differing inspiratory/expiratory times such that the respiratory signal is incompletely cancelled thereby imparting upward or
downward drift to the averaged signal.

The ensemble-averaging process gives a waveform that does not provide absolute end-systolic and end-diastolic values thereby preventing calculation of ejection fraction as can be measured from curves obtained with nuclear scans. Finally, there is uncertainty as to whether the VVC arises purely from the left ventricle using the TCG-Reference location in which transverse computed tomographic images indicate that the left ventricle is the major cardiac structure transected.

**Atrial Pacing**

In humans, CO is maintained when modest elevations of heart rate are produced by atrial pacing. This leads to progressive decreases of SV. Pacing above the rate of 140 beats/min is associated with falls in CO. In the pacing experiment involving anesthetized dogs in the current study, CO and SV estimations with both TCG and TD methods followed qualitatively similar courses to those reported during human cardiac pacing.

**Fluid Loading**

In normal humans and patients with rheumatic heart disease, intravenous infusion of hyperosmolar dextran solution increased CO and SV as determined with the Fick method. This also took place in the current experiments in anesthetized dogs as measured with both CO(TCG) and CO(TD).

**Effect Of Peep in Critically Ill Patients**

Thermodilution estimations revealed that CO fell 10 percent at levels 10 to 14 cm H_2O above the auto-PEEP level but that TCG-derived CO measurements did not detect this change despite a trend toward a decrease. The study most comparable to the current one was carried out by Tittley et al. in 50 patients six hours after elective coronary bypass surgery. They compared CO values at 5 to 15 cm H_2O PEEP (without measuring auto-PEEP) using thermodilution and found a 10 percent fall with the higher PEEP level. Volume loading with 250 ml of plasma to raise left atrial pressure 2 to 4 mm Hg completely prevented this decrease. Since our patients appeared to be volume overloaded as evidenced by the mean pulmonary capillary wedge pressure of 17 mm Hg, their observations indicate that large falls in CO with extrinsic PEEP would not be expected.

**Conclusion**

The TCG method has major labor-saving properties over other noninvasive methods for obtaining VVCs such as echocardiography and nuclear cardiac scanning as well as the potential for continuous monitoring of mechanical cardiac function. Given the variability of TD method for determination of SV and CO, TCG estimations of changes of these values to this standard compare favorably. Thus, 90 percent of SV(TCG) values fell within ±20 percent of SV(TD) in dogs during pacing and fluid overloading, and 79 percent SV(TCG) values fell within ±20 percent SV(TD) in critically ill patients during application of extrinsic PEEP. Although SV(TCG) and CO(TCG) cannot be independently calibrated to absolute volumes, the current study indicates that the method reflects quantitative changes.

**Financial Disclosure:** Marvin A. Sackner, M.D. is Chief Executive Officer and Chairman of the Board of Non-Invasive Monitoring Systems, Inc. (NIMS) which is the manufacturer of the equipment described in the investigation. He and his wife and Pension Trust hold approximately 7,659,000 shares of NIMS, a publicly traded company listed on NASDAQ Supplemental Exchange. This amounts to 23% of the total shares. Jonathan David Sackner holds approximately 146,000 shares of common stock which amounts to 0.4% of the total shares. Dr. Bruce P. Krieger serves as a member of the Scientific Advisory Board to NIMS and he and his wife own approximately 126,000 shares amounting to 0.4% of the total shares.

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