The diagnosis of cardiac dysfunction in critically III trauma patients with blunt chest trauma and presumed myocardial contusion: the critical nature of end diastolic volume

Künt göğüs yaralanması ve olası miyokart kontüzyonu olan kritik travma hastalarında kardiyak işlev bozukluğu tanısı: Diyastol sonu volümün kritik doğası

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BACKGROUND

To evaluate prospectively the effectiveness of monitoring end-diastolic volume (EDV) vs pulmonary artery occlusion pressure (PAO) as an estimate of cardiac preload in hemodynamically unstable critically ill 23 myocardial contusion patients with blunt chest trauma admitted to a university trauma center.

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METHODS

Patients were resuscitated (ATLS guidelines) with intubation and volume replacement for altered consciousness, hypoxemia or hemodynamic instability. Volume resuscitation and pulmonary artery catheterization were started to measure PAO, EDV, and oxygen utilization parameters. Myocardial performance was determined in both static (0., 24., 48., 72. hours) and dynamic condition after 500cc fluid bolus.

RESULTS

A moderate injury severity (GCS 9, TS 11, ISS 34) with MVA mechanism (91%), an average ICU stay of 14 days and a 39% mortality were revealed. Correlation of PAO, EDV with CO, CI demonstrated that EDV is more reliable than PAO However, factoring heart rate into CO determination was more reliable than single preload estimates of EDV-PAO-HR, HR-EDV, and HR-PAO. EDV-PAO-HR were correlated more strongly with cardiac output from 24 to 96 hours.

A higher AV02 decrease was associated with improved survival, and a nearly significant decrease in EDV. Dynamic comparison found no change in cardiac performance with a small volume challenge.

CONCLUSION

Simultaneous consideration of PAO, EDV, HR allowed the most accurate determination of cardiac output.

Key Words: cardiac preload parameters, unstable myocardial contusion, cardiac output

AMAÇ

Kardiyak önyükün ölçütü olarak diyastolsonu volüm (EDV) ve pulmoner arter tıkanma basıncını (PAO) izlem etkinliğinin üniversite travma merkezine başvuran 23 stabil olmayan myokart kontüzyonu ve künt göğüs travmalı kritik hastalarda karşılaştırmalı değerlendirilmesi

GEREÇ VE YÖNTEM

Hastalara bilinç bozulması, hipoksemi veya hemodinamik instabilite nedeniyle ATSL yönergelerine göre entübasyonla resüsitasyon , yoğun volüm replasmanı ve PAO, EDV ve oksijen tüketim parametrelerini ölçmek için pulmoner arter kateterizasyonu uygulanmıştır.Hem statik (0., 24., 48., 72. saatlerde) hem de 500 cc sıvı bolusu verdikten sonra dinamik koşullarda miyokart performansı belirlenmiştir.

BULGULAR

Baskın MVA mekanizması (% 91), YBÜ'nde ortalama 14 gün yatış süresi ve % 39 mortalite oranıyla orta derecede travma şiddet derecesi (GCS 9, TS 11, ISS 34) saptanmıştır. EDV'nin CO ve CI ile korelasyonu PAO'dan, kalp hızının CO belirlenmesine katkısı ise tek bir önyük EDV-PAO-HR, HR-EDV ve HR-PAO'dan daha güvenilirdi. EDV-PAO-HR ise 24-96 saatlerdeki kalp debisiyle daha güçlü bir korelasyon içindeydi AVO2'de daha yüksek bir düşüş iyileşmiş bir sağkalım ve EDV'de anlamlı bir azalmayla ilişkiliydi. Küçük bir volüm testiyle dinamik karşılaştırmada kardiyak performansta herhangi bir değişiklik saptanmamıştır.

SONUÇ

Eş zamanlı PAO, EDV ve HR ölçümleri kalp debisinin en doğru değerlendirmesine olanak tanımıştır..

Anahtar Sözcükler: kardiyak önyük parametreleri, stabil olmayan myokart kontüzyonu, kalp debisi

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INTRODUCTION

Conventional Pulmonary Artery Catheterization: Current Opinion. After mechanical ventilation, pulmonary artery catheterization is the procedure most often associated with an ICU stay. The amount of convincing evidence of benefit is not extensive, while there are reports questioning harm. DeBoyd evaluated 528 PA catheter insertions with minor complications found in 26% (136 of 528) while serious complications occurred in 4.4% (23 of 528).[1] They suggested that since 80% of practioners subjectively perceived benefit, clinical use was indicated in specific cases in spite of the risk.

Furthermore, when Iberti administered a multiple choice examination to 496 physicians to assess their knowledge and understanding of the device, they found a mean test score of 67% (19-100%) correct .[2] These scores varied with the level of training, frequency of data use in management, frequency of insertion, and whether hospital was a primary medical school affiliate.

The clinical use of the PA Catheter (PAC) was explored examining diagnostic, therapeutic and prognostic strategies. Mitchell performed a randomized prospective trial in 101 critically ill patients where the PA Catheter was used to measure wedge pressure and extra vascular lung water to minimize positive fluid balance decreasing both ventilator and ICU days. [3] Steingrub evaluated 154 medical/surgical ICU patients to determine that prediction of hemodynamics by clinical evaluation alone is inaccurate, in 45% new information resulted in a major change in therapy. [4] However, the overall classification rate of residents and attendings were 47% for pulmonary artery wedge pressure (PAWP), 51% for cardiac output (CO) and only 36% for systemic vascular resistance (SVR).

The use of the PAC has long been the "standard of care" for cardiac patients. Zion reported a 6.4% (n = 371) increase of use in 5841 acute myocardial infarction (AMI) patients and found the catheter tended to be used in sicker patients with the in hospital monitoring excess due to more severe disease.[5] Lastly, individual variables such as the pulmonary artery mean pressure were compared and was found to be 32% higher (25 vs. 8 mm/hg) in decedents and is independent predictor of mortality providing crucial prognostic information.[6]

History: Branthwaite described a technique for the measurement of cardiac output by thermal dilution technique, especially after acute circulatory disturbance.[7] They described a technique where a themistor mounted at the tip of miniature cardiac catheter floated to the pulmonary artery to sense a change in temperature after injection of room temperature saline through catheter in internal jugular vein. The cardiac output is calculated by the equation where heat injected = heat detected on V (volume of injection in ml) • Di (density of injectate) • S (specific heat of injectate) • (Tb - Ti) temperature in pulmonary artery - temperature of injectate = Q (CO L/min) • dt (average temperature change) • t (duration of temperature change in seconds) • Db (density of blood) • Sb (specific heat of blood) • 1000/60. These values were highly significantly correlated by regression analysis to CO values obtained by the direct Fick method measuring the arteriovenous oxygen difference = arterial oxygen saturation & - mixed venous oxygen saturation - hemoglobin (g/dl) - 1.36 ml of oxygen/gram of Hb normally 3.0 - 5.0 mg/dl where X (Fick CO) = 0.9617 y (thermodilution CO) - 0.2007, $p < 0.006$.

The first clinical use of the flow directed pulmonary artery catheter was described by Swan in 1970, but with little clinical supporting data.[8] Ganz went on to compare the thermodilution cardiac output method (COTD) to dye dilution methodology (CO Dye) in 63 patients.[9]

$$\text{The calculated } CO_{TD} = \frac{V_I - (T_B - T_I) \bullet S_I \bullet CI \bullet 60}{S_B \bullet C_B \, S^{\infty}\Delta \, T_B \, (+)\, dt}$$

where V_I = volume of injectate ml; T_B, T_I, S_B, S_I, C_B, C_I = temperature specific gravity and specific heat of blood and indicator respectively; and $S_I \bullet C_I / S_B \bullet C_B = 1.08$ when 5% dextrose is used as indicator.

The dye dilution technique utilized 2.72mg/1.1ml solvent indocyanine green mixture flushed into the SVC or RA while a withdrawal pump sampled femoral artery blood for densitometer analysis, in serial fashion after baseline level was obtained. Likewise, the correlation of the thermodilution CO with dye determination was excellent with CO to = 0.96 CO dye + 0.7 (L/min), with cor-

Table 1: Demographic Profile of Myocardial Contusion Patients

Injury Profile	Patients (23)
Glasgow Coma Score	8±
Trauma Score	11±
Injury Severity Score	36±
Intervention	
Motor Vehicle Accident	91%
Pulmonary Injury	50%
Celostomy	36%
Outcome	
ICU Stay	15 Day
Hospital Stay	22 Day
Mortality	36%
Diagnosis	**Abnormal**
Electrocardiogram	92%
Chest Radiography	87%
Creatine Phosphokinase	
Total	100%
MB	9%
Echocardiogram	37%

relation coefficient R = 0.96, p < 0.001. Some variability was noted with thermodilution over estimation in AI, AS, MI, and MS, while decreased with VSD.

Lastly, a newer modification of the thermodilution technique utilizes a heated, thermal filament in the catheter wall located 14-25cm from the distal tip. [10, 11] Cardiac output is estimated by the indicator dilution principle where the heat transferred at the RA is measured at the downstream thermistor. This continuous cardiac output (CCO) catheter (Baxter, Edwards Critical Care) uses the thermal fragment to function as the indicator; where a stochastic system identification allows measurement of fluid flow senses in an on-off binary fashion forming a downstream thermodilution "washout" curve every 3 minutes with 30-60 second display uptake.[10-12] Advantages include less interobserver variation and risk of contamination.

MATERIALS AND METHODS

Protocol: This study was approved by the University of Pittsburgh Investigation Review Board. The human subjects included 23 critically ill blunt chest trauma patients admitted to the trauma intensive care unit in a convenience sample all to receive protocolize standard ICU care attempting to maintain oxygenation, perfusion, pH and tonicity. Patients were monitored for up to a 96 hour period, as warranted by patient condition.

Emergency department resuscitation: Patients were resuscitated and stablized according to standard Advanced Trauma Life Support guidelines. Adequate oxygenation was ensured and intubation and mechanical ventilation was instituted for altered level of consciousness, hypoxemia managed as PaO2 < 60 mmHg or oxygen saturation < 90% or hemodynamic instability. Circulatory support was instituted with large bore access - femoral 8.5 FR introducer, hemorrhage eternal, including operative intervention if necessary. Fluid administration consisted of crystalloid - lactated ringers to a total of 2000cc followed by blood administration as necessary to achieve a SBP > 90 mmHg, followed by transport to the OR or ICU.

Intervention: Patient was admitted to the trauma intensive care unit reevaluating cardiopulmonary stability - ensuring adequate oxygenation (pO2 > 60 mmHg) and ventilation (pCO_2, 40 mmHg). Patients were administered high flow oxygen at 12-15 LPM to ensure saturations > 90%. If unable to maintain the mechanical ventilation was begun with FiO_2 0.5-1.0 and positive end expiratory pressure of 5 cmH_2O. Volume control ventilation in an assist control mode was provided at 10 ml/kg to maintain peak pressure < 40 cm H_2O.

Adequate systemic perfusion was maintained by administration of additional crystalloid in 10 ul/kg boluses. If systolic blood pressure was inadequate then a 8.5 FR introducer and REF Explorer pulmonary arterial catheter (Baxter Edwards Critical Care Division, Anaheim, California) using an internal jugular or subclavian vein approach. The oximetric device was calibrated in an invivo or invitro fashion depending on patient condition.

Table 2: Regression Analysis of Cardiopulmonary Variables

Hours	0	24	48	72	96	Mean
CO	.24633	.32780	.35442	.39550	.48705	.3419
HR	.12106	.38196	.49880	.56848	.31074	.3494
EDV	.20616	.44278	.62680	.20095	.90019	.4722
HR-PAO	.27681	.60802	.41124	.61766	.50366	.4735
HR-EDV	.21558	.63046	.65460	.80405	.94699	.6712
EDV-PAO	.21884	.54951	.68376	.35856	.92336	.5417
EDV-PAO-HR	.22485	.82358	.75581	.81280	.95149	.7079
	.22485	.82358	.75581	.81280	.95149	.7079
MEAN	0.2157	0.5377	0.5695	0.5368	0.7176	
	N=23					

Multiple Regression
ANOVA with Pearson Correlation
RMANOVA

Intensive Care: Additional crystalloid was adminstered to ensure adequate performance. Dopamine was used as a vasoconstriction agent at a dose of 2.5-20 ug/kg/min if refracting hyoptension. Dobutamine at a 5-20 ug/kg/min dose was inotropic support. Norepinephrine or epinephrine at a 0.1-1.0 ug/kg/min dose was used less commonly for refractory cardiovascular failure. Mechanical support including extracorporeal membrane oxygenate (ECMO) and intraaortic balloon pump (IABP) was provided for refractory hypotension, <90mmHg SBP or MAP <60mmHg. Fentanyl at 1-2 ug/kg/hr was used for sedation and ventricular 0.01 mg/kg/hr for muscle relaxation to inhibit in patient sedation and control for ventilation.

Temperature: Temperature control was achieved by maintaining patients in a 36-38° C range. Acid base status was also maintained with base defect correction to ≤ 5 by the administration of sodium bicarbonate 0.1mEq/kg. Calcium chloride (0.01mg/kg) also used to correct hemodynamically significant hypocalcemia (Ca ionized <0.90m Eq/L).

Assessment: Demographic variables were assessed including age, sex, past medical history, and history of present illness, diagnosis and surgical intervention. The diagnosis of myocardial contusion included electrocardiogram (EKG), chest radiography (CXR), creatine phosphokinase (CPK) with myocardial fraction - muscle - brain (MB), and transthoracic (TTE) or transesophageal (TEE), echocardiogram.

Cardiopulmonary variables were obtained in static fashion at standardized intervals (0, 24, 48, 72, 96) and in a dynamic fashion after a 500cc fluid challenge. The information obtained included hemodynamic variables or work indices such as cardiac output (CO), ejection fraction (EF), or stroke volume (SV), as well as length indices such as central venous pressure (CPV), pulmonary artery occlusion pressure (PAOP), end diastolic volume(EDV) and end systolic volume (ESV). Additional respiratory variables measured included oxygen delivery (DO_2), oxygen consumption

Table 3: Effect of preload variables on cardiac output.

24 Hours	**48 Hours**	
A	A`	PAO - HR: CO
B	B`	EDV - HR: CO
C	C`	EDV - PAO - HR: CO

Table 4: Cardiopulmonary parameters at presentation.

Cardiac Time (Hrs.)	Survivors	Nonsurvivors	Significance
CO	5.9 ± 2.6 12	5.2 ± 2.7 6	NS
CI	2.9 ± 1.2 12	2.9 ± 1.0 5	NS
CVP	13.5 ± 3.9 6	16.5 ± 0.7 2	NS
PAO	16.6 ± 6.3 12	15.7 ± 5.3 6	NS
EDV	183.3 ± 33.2 7	242.5 ± 61.6 4	p0.06
HR	109.3 ± 26.5 12	96.8 ± 15.8 6	NS
EF	38.0 + 4.6 3	22.0 1	p0.09
SV	77.0 ± 20.2 3	39.0 1	NS
ESV	124.0 ± 20.1 3	139.0 1	NS
MAP	77.3 ± 31.4 6	77.0 ± 11.3 2	NS
MPP	31.2 ± 7.4 5	43.0 1	NS
LVSW I	32.8 ± 16.3 6	0 0	--
RVSW I	7.4 ± 2.5 5	0 0	--
SVR I	2357.5 ± 665.6 6	1444.0 1	NS
Pulmonary			
AV02	5.1 ± 1.2 6	2.1 ± 1.7 2	p0.03
D02	854.2 ± 225.8 6	1024.0 1	NS
V02	263.0 ± 50.1 6	237.0 ± 24.0 2	NS
02EI	0.29 ± 0.1 5	0.24 1	NS

Student's t test

(VO_2), as well as arterial and venous oxygen saturation and content. Data was compared for survivors and nonsurvivors.

Data Analysis: Demographic data was reported as frequently, and proportions of survivors compared to nonsurvivors were analyzed using Fisher's exact test were appropriate for differences. The descriptive variables were reported as mean standard deviation and range, as well as, objective numeral data where condition variables were analyzed using Student's t test, cardiopulmonary data was analyzed with regression and analysis of variance (ANOVA) comparisons with significance indicated by standard criteria ($p < 0.05$).

RESULTS

Demographic analysis revealed a moderate in-

Table 5: Cardiopulmonary parameters at 24 hours after presentation.

Cardiac Time (24 Hrs)	Survivors	Nonsurvivors	Significance
CO	7.9 ± 3.4 9	8.4 ± 2.8 8	NS
CI	4.1 ± 1.4 9	3.8 ± 1.2 7	NS
CVP	11.7 ± 2.4 8	13.9 ± 5.1 8	NS
PAO	15.0 ± 8.3 9	17.2 ± 8.3 8	NS
EDV	224.3 ± 59.3	223.3 ± 68.2	NS
HR	102.8 ± 32.8 10	94.1 ± 38.6 8	NS
EF	36.8 ± 10.2 7	36.3 ± 9.6 7	NS
SV	79.0 ± 18.4 7	83.7 ± 43.4 7	NS
ESV	146.0 ± 61.1 7	139.0 ± 33.7 7	NS
MAP	80.5 ± 11.3 9	74.0 ± 15.2 8	NS
MPP	27.2 ± 8.2 9	31.1 ± 10.8 8	NS
LVSW I	8.6 ± 12.9 9	30.0 ± 22.4 8	NS
RVSW I	8.6 ± 4.7 9	8.1 ± 3.9 8	NS
SVR I	1540.7 ± 747.2 9	1326.2 ± 564.8 7	NS
Pulmonary			
AV02	4.0 ± 1.3 8	4.4 ± 1.3 7	NS
D02	1071.2 ± 445.2 8	1105.0 ± 412.5 7	NS
V02	275.4 ± 117.5 8	353.5 ± 139.3 6	NS
02EI	.23 ± 0.07 8	.31 ± .10 6	p = 0.09

Student's t test with Levine test for quality of variances.

jury severity population with a Glasgow Coma Score (GCS) of 8.6 ± 5.8, Trauma Score (TS) of 11.3 ± 3.9, and Injury Severity Score (ISS) of 36.6 ± 12.7. Patients were predominantly admissions for motor vehicle accidents in 91%, with pulmonary pathology in 39%, and undergoing celiotomy in 43.5%. They went on to require a 13.4 ± 10.7 day ICU, 26.5 ± 38.1 day hospital stay and overall mortality was 36% (Table 1).

Diagnostic intervention revealed an abnormal EKG in 92%, chest x-ray in 87%, creatine phosphokinase total (CPK) in 100% and myocardial specific fraction (CPK-MB) in 9%.

Analysis of the temporal relationship of correlation between preload variables and cardiac output finds that prediction is most reliable after the initial presentation (R = 0.2157), and treatment phase 24, 48, 72 hours (R = 0.5377, 0.5695, 0.5368) instead focused on the convalescent phase (R = 0.7176) (Table 2).

Examination of select preload variables finds that in individual analysis overall correlation is poor, but greater for EDV (R = .4722) than PAO (R = .3419). However, when preload variables were

Table 6: Overall comparison of cardiopulmonary parameters.

Survivors	Nonsurvivors	Significance	
CO	8.3 ± 3.3 23	6.7 ± 2.9 22	P =0.086
HR	114.7 + 23.7 23	112.9 ± 21.1 22	NS
PAO	16.4 ± 6.8 23	19.2 ± 9.5 22	NS
CI	4.1 ± 1.7 23	3.3 ± 1.2 22	NS
EDV	212.9 ± 65.9 20	193.7 ± 51.4 21	NS

Student's t test

combined to factor in the heart rate when CO correlation is determined, then EDV-HR (R = .6712) is more significantly correlated than PAO-Hr (R = .4735)(Table 3, A-B). The most significant correlation is found when all variables, EDV-PAO-Hr (R = .7079) were considered (Table 3, C). In fact, when the initial presentation correlation (R = .2248) is excluded the combined preload determination (EDV-PAO-Hr) is strongly correlated over the remainder of intervals 24 hours (R = 0.8236), 48 hours (R = 0.7558), 72 hours (R = 0.8128) and 96 hours (R = 09.515) (Table 4).

Isolated cardiopulmonary parameters have been correlated to survivorship to demonstrate a significant correlation between widened AVO2 difference (5.1 vs. 2.1, PO.03) as well as, a trend toward significance with decreased EDV (183.3 vs. 242.5ml, PO.06) and trends toward improved ejection fraction (38 to 22%, PO.09) (Table 5). However, at 24 hours only the oxygen extraction index approaches significance with a decreased extraction ratio (23 vs. 31%, PO.09) associated with improved survivorship.(Table 5)

In addition, overall comparison of cardiopulmonary parameters found only cardiac output approaching significance with increased CO (8.3 vs. 6.7 L/min, PO .08) associated with increased survival (Table 6).

Lastly, the dynamic comparison of cardiopulmonary parameters found no appreciable difference in preload measures (CVP, EDV) or work indices (CO, CI, AVO_2).(Table 7)

The need for a method to continuously assess cardiac performance is substantial in the critically ill patient. However, the need to assess preload accurately in the setting of primary or secondary

Table 7: Cardiopulmonary analysis with preload variations.

Cardiac	Pre-Intervention	Post-Intervention	Significance
CO (L/min)	7.7 ± 3.5	7.6 ± 3.1	NS
CI ($L/min/M^2$)	3.8 ± 1.8	3.7 ± 1.6	NS
CVP (CMH20)	14.5 ± 4.3	12.5 ± 3.8	NS
Pulmonary $AV0_2$	4.7 ± 1.2	4.2 ± 1.0	NS
EDV	1183.5 ± 492.8	1128.4 ± 523.9	NS

lung disease requiring mechanical ventilation and extrinsic PEEP require knowledge of RV volume as well as filling pressure.[13] As PEEP increases, there is an accompanying linear decrease in CI, while the PAP increases in direct proportion to the PEEP. However, this change may be bimodal-dependent on the initial RVEDVI, if > 120 ml than RVEF decreases and RVEDVI increases due to effect of increasing afterload with PEEP, but if < 120 ml RVEF is unchanged and RVEDVI is decreased due to effect on venous return at 15cm H2O of extrinsic PEEP in acute respiratory failure.[14] The intricate nature of these individual changes requires the capacity to continually and reliably monitor the intracardiac and intrathoracic condition.

Kay described the reproducibility, accuracy and clinical applicability of ventricular ejection fraction derived by thermal dilution technique in both dogs and humans compared to radionuclide angiography using a modified Baxter, American Edwards PA (Anaheim, CA) catheter and computer to amplify thermisistor output signal.[15] The RVEF measured in 9 dogs (1,014 determinations) and 8 patients (744 determinations) was reproducible ± 5%, as was the correlation of LVEF in 10 patients. They found this to be a reliable technique with a thermal - radionuclide correlation of R = 0.86 - 0.93, p < 0.02 and easily performed in the critically ill.

Their methodology included varying injectate volume (5-10ml) site (RV, LA), temperature (4 - 24∞C), myocardial condition to simulate low RVEF (17 - 30%) by increasing RV afterload with respiratory acidosis or phenylephrine, normal RVEF (35 - 40%) and high RVEF (55-70%) by volume loading and isoproterenol proving reliability across a wide range of critical conditions.

The calculation of EF is based on the conservation of thermal energy, and ventricular volume based on the CO and HR. The conservation of energy requires T (ESV) (C) + Tb (EDV - EBV) (C) = T2 (EDV) (C), where T1 = temperature, T2 = temperature of the blood in the ventricle during second systole and C = constant related to the specific gravity and heat capacity of blood.[15, 16] Thus, the heat energy of the ventricular blood at end-systole and the heat energy of the ventricular blood during diastole must equal the sum of the energies in the ventricle when two blood volume are mixed and ejected during the second systole.[14]

Similarly, the ejection fraction is the proportion of blood as a percentage left in the ventricular chamber at end-diastole that is ejected at end systole, where

$$EF = \frac{EDV - ESV}{EDV} = 1 - \frac{ESV}{EDV}$$

Reasoning the former eqation to

$$\frac{EDV}{EDV} = \frac{T2-TB}{T1-TB} = \frac{TB-T2}{TB-T1}$$

and substituting the $EF = 1 - \frac{TB - T2}{TB - T1}$

where ejection fraction can be calculated by measuring the incoming baseline blood temperature as well as two "ejected" samples.[14]

Cardiac output can be derived from the same thermal washout curve CO = K/area under curve, where K = constant of injectate temperature and volume. Since stroke volume is SV = CO/HR and EF = SV/EDV then EDV = SV/EF and ESV = EDV - ESV, therefore, EF, CO, ESV, and EDV can be calculated by measuring the temperature of ejected blood during consecutive systoles, baseline blood temperature, heart rate and area under the thermal washout curve.[14]

Again, the correlation is reasonable with regression equation y = 0.92 (x) + 18, where once the high intercept of 18 is explained due to thermal technique overestimate or gated technique underestimation the slope correlates well (0.92) in spite of the 18% correction.[14] This thermodilution overestimation seemed most significant in low output conditions (< 25 %) in the right heart.

This thermodilution washout curve can be illustrated graphically (Figure 1). The technique involves injection of iced or room temperature saline into the RA, mixing with RV blood, and temperature change inversely proportional to flow as detected by the pulmonary artery thermisistor. Profeta compared iced and room temperature injectate to define a reasonable correlation for ESV (R = 0.85) and R = 0.95 for CO, acceptable in light of the ease of preparation.[14] This downslope of the thermal washout curve follows exponential delay interrupted by a series of diastolic plateaus.[17] Each successive increase in blood temperature in the pulmonary artery represents the RV ejection of progressively warm blood as the cold RV indicator is warmed by additional inflow. Calculation of the ratio between the temperature decrease of two suc-

cessive diastolic plateaus yields an estimate of the residual fraction, or blood remaining in the ventricle predicting EF = 1 - RF.[14]

Technologic improvements have allowed more accurate prediction of EF, but some limitations remain. The fast response thermistor which provided 100% of step change in 125 m sec became limited by catheter body with decrease to 82 - 92% at 0.5 sec and 88-96% at 1.0 sec, demonstrating a important slow component with second time constant induced by catheter body so equilibration was not complete for 6 seconds, slightly decreasing the measured EF.[18]

However, the REF-1 right ventricular ejection fraction catheter and computer (Baxter, Edwards Critical Care, Anaheim, California) were validated with a pulsatile bench cardiac output over a CO range of 1-6.0 BPM and EF of 15-60% demonstrating a correlation coefficient of R2 0.94 for EF and R^2 0.99 for CO.[19]

The RVEF technology also compared well with biplane angiography. Urban evaluated 13 patients using a modified Simpson's rule for computing angiographic volumes demonstrating an acceptable correlation, R = 0.83 for RVEF and R 0.71 for RVEDV.[20] They found that thermodilution underestimated RVEF and overestimated RVEDV compared to angiography with a 12 ± 5% variability with patients. Voekler performed a similar comparison examining 22 precardiac catheterization patients demonstrating a mean RVEF of 52% (32 - 71%).[20] There was good correlation with angiography (RO.80) especially in those with small RV (</60ml), lower Hr (<65/min) and CO < 5.5 L/min.

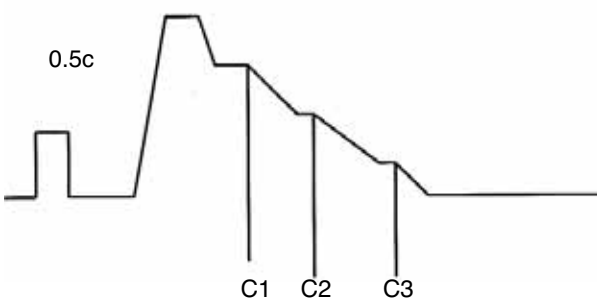
Lastly, the RVEF thermodilution technique has reasonable correlation with echocardiography. Jardin compared RVEF and RV volume with simultaneous 2-dimensional transthoracic echocardiography (TTE), demonstrating significant correlation between thermodilution RVEF and TTE with fractional area contraction (R = 0.74), RVEDV and end-diastolic area (R = 0.70) and RVESV and end-systolic area (0.78). [21] However, the RVEDP as an indicator of RV preload did not correlate, suggesting volume and not pressure measurements were more accurate.

Likewise, Koorn compared the rapid-response thermisistor PA catheter to transesophageal echocardiography (TEE) in 25 perioperative patients.[22] Linear regression showed good correlation between RVEF and TEE (R = 0.76), where RVEF was calculated as RVEDA-RVESA/RVEDA. The bias analysis yielded an error of 11.5 ± 9% for the 2d TEE RVEF values with 95% confidence limits of -8.3 - 31.3%. Thus, TEE is less reliable than thermodilution with overestimation of EF as well as variability with wide 95% confidence interval.

Another significant advance in RVEF thermodilution technology was the development of computer algorithms that determine the best fit to the exponential washout curve using least squares regression.[17, 23, 24] Here, the computer simultaneously acquires an electrocardiographic signal along with the thermodilution curve, facilitating the identification of diastolic plateau. The residual fraction is then calculated by combining the measured R-R interval and the best-fit thermodilution curve. Cardiac output is the calculated from the same PA temperature curve by using the Stewart-Hamilton equation. Since the CO, HR, SV and EF are defined the volumes are now calculated where RVEDV = SV/RVEF and RVESV = RVEDV - SV.[17]

Vincent performed a clinically relevant study evaluating 14 critically ill patients with stable hemodynamic status comparing RVEF with radionuclear gated first pass technique.[23] This catheter system was modified to a fast response thermistor, intracardiac ECG monitor, a more proximal lumen injection port ending 21 cm above tip of the catheter to facilitate mixing of cold indicator above the tricuspid value, and the use of a new algorithm based on the exponential thermodilution curve analysis limiting variability to 7.6% (Figure 1). They found acceptable correlation between thermodilution and radionuclide techniques (y = 0.49x + 12.7, R = 0.67), although the values obtained by thermodilution were lower, especially at high RVEF values.

Dhainaut performed a similar evaluation of 34 patients using a new algorithm compared to the conventional plateau method, as well as first pass and gated nuclear techniques. In the conventional plateau method the computer stores five thermodilution data points each time it detects an R wave, with the average of 5 points equivalent to the plateau.[14] The first plateau (T1) starts when the first R wave after 80% of the peak deviation is reached on the thermodilution curve. The thermodilution



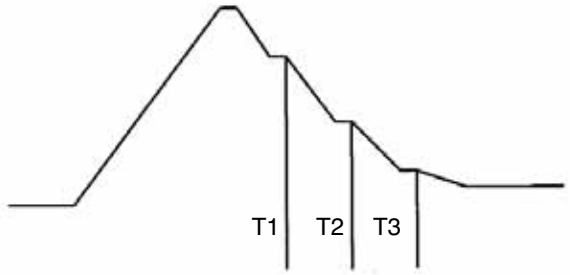
EF = 1 - RF (mean residual fraction)

$$FR = \frac{C2}{C1}$$

$$RF2 = \frac{C3}{C2}$$

$$RF = \frac{RF1 + RF2}{2}$$

New Algorithm



T1 . ESV . C + T3 (EDV - ESV) . C = T2 . EDV . C

T1 . ESV . T3 EDV - T3 . ESV = T2 EDV

$$RF = \frac{ESV}{EDV} = \frac{T2 - T3}{T1 - T3} = \frac{T3 - T2}{T3 - T1}$$

RVEF = 1 - RF

Figure 1: Thermodilution Washout Curve Conventional

signal and ECG monitor are amplified and computer processed to calculate two successive RVEF where

$$EF1 = 1 - \frac{T2 - TB}{T1 - TB} \text{ and } EF2 = 1 - \frac{T3 - TB}{T2 - TB}$$

where T1, 2, 3 are successive plateaus and TB is the basal temperature.[24] (Figure 1)

The new algorithm uses the system response of a single pulsatile chamber, the RV to a pulsed ingest, call water bolus.[24] This results in a single first order exponential washout curve which indicates the residual fraction of the indicator in the chamber. The residual fraction occurring with each R-R interval is computed after first order exponential curve fit is established on the downslope of the thermodilution curve. The average ejection fraction is the sum of the average resident volume subtracted from 1.

They concluded that this new method improved reproducibility ability where thermodilation RVEF compared favorably to first pass (R = 0.92) and gated (R = 0.81) technique.

The patient population that was evaluated in this sample accounted for only a small proportion of total trauma admissions with blunt chest trauma refractory to conventional volume resuscitation. This select population had a high likelihood of cardiac dysfunction and was associated with significant morbidity manifested as a prolonged hospitalization and significant mortality.

The temporal trends in correlation between preload variables (EDV, PAO) and cardiac indices (CO) demonstrated significant variability on hospital presentation that became more stable over time. Therefore, if the cardiac performance is evaluated with a pulmonary artery catheter it is often prudent to realize that these parameters may only be reliable in the 24 to 96 hour post injury period, but not on initial presentation.

Interestingly, there has always been a search for a single preload variable CVP, PAO or EDV to predict cardiac output. This strategy is limited in the acutely injured patient with myocardial dysfunction, although EDV performs better than PAO after presentation through most of the acute injury phase at 24, 48, and 96 hours. Therefore, in the injured heart with decreased compliance the EDV calculated by the right heart ejection fraction explored REF explorer catheter (Baxtor-Edwards Critical Care, Anaheim, California) appears to be a more reliable "indirect" estimate of myocardial fiber length than the "direct" pressure measurement. The irony is that the patients most in need of this diagnostic modality predisposed by myocardial ischemia, contusion on intrathoracic pressure alterations are also prone to inaccuracy of preload estimation based on intracardiac chamber pressure measurement.

Clearly, controlling for heart rate offers a more accurate CO prediction, whether length or pressure measurements are used for preload estimation,

according to the conventional cardiac output relationship (CO = SV . HR). Caution is then warranted in comparing CO measurements with temporal variations in heart rate precluding standardization for comparison between groups.

Ideally, then the most significant correlation between preload and output is found when both EDV and EDP correlate and the heart rate effect is standardized. Again, the difficulty is that more often than not the EDP and EDV are discrepant and in those cases the EDV appears to be more reliable in predicting volume status.

Isolation of cardiopulmonary variables as they relate to survivorship correlate a number of trends in the acute phase of injury from 0-24 hours. The most consistent relationship was found in the AVO2 difference where a normalized value (5.1) was associated with survival while a narrow value (2.1) indicative of hyperemia was not. A relative hyperemia can be found in those patients with significant myocardial dysfunction resulting in a lack of systemic perfusion, effectively a shunt where the venous effluent has a falsely elevated oxygen content. Survivors had a more rapid return of oxygen coupling with a normalized oxygen extraction ratio (23%) compared to the ischemic profile (31%) in nonsurvivors.

The "stunned" myocardial profile appears reliable with a normalized EDV (183.3 + 33.2ml), CO (8.3 L/min) and EF (38%) compared to a distended EDV (242.5 + 61.6ml), noncomplaint EF (22%) heart with decreased ouput CO (6.7 L/min) in nonsurvivors. Therefore, caution is warranted ensuring adequate volume resuscitation, but avoiding ventricular overdistention. As a reference point in the acute phase a 500ml volume challenge is not sufficient for the average trauma patient since the heart may not be capable of responding adequately in this injury period. However, in either scenario aggressive volume resuscitation can be combined with careful EDV monitoring to avoid ventricular overdistention further worsening myocardial performance, as well as, deciding what patients may benefit from inotropic on vasopressor support.

CONCLUSION

The need for continuous monitoring of the critically ill trauma patient is defined by the clinical circumstances such as a patient refractory to empiric therapeutic manipulation. This monitoring should have the capability of measuring preload as EDV, contractility as EF and afterload as ESV, as well as routine intracardiac pressure measurements and oxygen delivery - consumption measurements to confirm the treatment plan.

The right heart is extremely sensitive to afterload alteration, loading to overdistention compromising RV coronary flow, as well as adversely affecting LV diastolic mechanics due to septal shift in the setting of interventricular dependence.

Therapy should be directed at restoring adequate preload guided by EDV especially in the setting of altered vasomotor tone in sepsis, or increased intrathoracic pressure with mechanical ventilation or intrinsic PEEP. Then, in light of the exquisite sensitivity of the RV to afterload increase, a short acting vasodilating agent can be instituted, such as nitroprusside in the medical patient.

Introtropic support instead is chosen in the unstable trauma patients, using an agent such as dobutamine or nitroglycerine with a side effect of afterload reduction. Caution is warranted to prevent against use in this hypovolemic patients as adverse effects in the systemic vascular resistance will largely occur causing hypotension.

Lastly, mechanical support is a bridge to additional medical or surgical therapy, and can be attempted but is often precluded by infection or hemorrhagic risk.

Patients with poor myocardial performance due to a decrease in compliance may benefit from monitoring EDV in addition to PAO determinations. Adequacy of volume resuscitation can be monitored by preload determination (EDV, PAO), myocardial performance (CO, EF), and oxygen delivery - utilization parameters performance (AVO2, O2EI) maximizing performance by avoiding excess extravascular lung water and adding inotropic support to those with ongoing refractory oxygen debt.

REFERENCES

1. DeBoyd K, Thomas S, Gold J, Boyd A: A prospective study of complications of pulmonary artery catheterization in 500 consecutive patients. Chest 1983; 84:245-249
2. Iberti T, Fischer E, Leibowitz A, Panacek E, Silverstein J, Albertson T, Pulmonary Artery Catheter Study Group: A multicenter study of physicians'

knowledge of the pulmonary artery catheter. JAMA 1990; 264:2928-2932
3. Mitchell JP, Schuller D, Calandrino FS, Schuster DP: Improved outcome based on fluid management in critically ill patients requiring pulmonary artery catheterization. Am Rev Respir Dis 1992; 145:990-98
4. Steingrub JS, Celoria G, Vickers-Lahti M, Teres D, Bria W: Therapeutic impact of pulmonary artery catheterization in a medical/surgical ICU. Chest 1991; 99:1451-55
5. Zion MM, Balkin J, Rosenmann D, Goldbourt U, Reicher-Reiss, H, Kaplinsky E, Behar S: Use of pulmonary artery catheters in patients with acute myocardial infarction. Chest 1990; 98:1331-35
6. Cooper R, Ghali J, Simmons BE, Castaner A: Elevated pulmonary artery pressure. Chest 1991; 99:112-20
7. Branthwaite M, Bradley R: Measurement of cardiac output by thermal dilution in man. Journal of Applied Physiology 1968; 24:434-438
8. Swan H, Ganz W, Forreston J, Marcus H, Raymond G, Charnette D: Catheterization of the heart in man with the use of a flow direction balloon. NEJM 1970; 283:447-451
9. Ganz W, Donoso R, Marcus H, Forrester J, Swan H: A new technique for measurement of cardiac output by thermodilution in man. American Journal of Cardiology 1971; 27:392-396
10. Iberti T, Silverstein J: Continuous cardiac output measurements in critically ill patients. Journal of Cardiothorasic and Vascular Anesthesia 1992; 6:267
11. Yelderman ML, Ramsay MA, Quinn MD, Paulson AW, McKowan RC, Gillman PH: Continuous thermodilution cardiac output measurements in intensive care units patients. Journal of Cardiothorasic and Vascular Anesthesia 1992; 6:270-274
12. Guilbeau J, Applegate A: Thermodilution: an advanced technique for measuring continuous cardiac output. Dimensions of Critical Care Nursing 1996; 15:25-29
13. Assmann R, Falke KJ: Pressure and volume assessment of right ventricular function during mechanical ventilation. Intensive Care Medicine 1988; 14:467-470
14. Kay HR, Afshari M, Barash P, Webler W, Iskandrian A, Bemis C, Hakki H, Mundth E: Measurement of ejection fraction by thermal dilution techniques. Journal of Surgical Research 1983; 34:337-346
15. Baxter Edwards Critical Care: REF Explorer. Article Insert 1994.
16. Profeta JP, Roth JV, Wiener LB: Comparison of room temperature vs. iced injectate using the REF-1 pulmonary artery catheter. Anesthesiology 1991; 75:3A
17. Maruschak GF, Schauble JF: Limitations of thermodilution ejection fraction : Degradation of frequency response by catheter mounting of fast response thermistors. Critical Care Medicine 1985; 13:679-682
18. Easton S: Preliminary pulsatile flow bench validation of a thermodilution right ventricular ejection fraction system. Intensive Care Medicine 1988; 14:458-460
19. Urban P, Scheidegger D, Gabathuler J, Rutishauser W: Thermodilution determination of right ventricular volume and ejection fraction: a comparison with biplane angiography. Critical Care Medicine 1987; 15:652-655
20. Voekler W, Gruber HP, Ickrath O, Unterbert R, Karsch KR: Determination of right ventricular ejection fraction by thermodilution technique—a comparison to biplane cineventriculography. Intensive Care Medicine 1988; 14:461-466
21. Jardin F, Gueret P, Dubourg O, Farcot JC, Margairaz A, Bourdarias JP: Right ventricular volumes by thermodilution in the adult respiratory distress syndrome. Chest 1985; 88:34-39
22. Koorn R, Reich DL, Tamman R, Narang J, Hillel Z: Right ventricular ejection fraction measurement; thermodilution vs. 2-D transesophageal echocardiography. Anesthesiology 1991; 75:3A
23. Vincent JL, Thirion M, Brimioulle S, Lejeune P, Kahn RJ: Thermodilution measurement of right ventricular ejection fraction with a modified pulmonary artery catheter. Intensive Care Medicine 1986; 12:33-38
24. Dhainaut JF, Brunet F, Monsallier JF, Villemant D, Devaux JY, Konno M, DeGournay JM, Armaganidis A, Iotti G, Huyghebaert MF, Lanore JJ: Bedside evaluation of right ventricular performance using a rapid computerized thermodilution method. Critical Care Medicine 1987; 15:148-152