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Title: DIFFERENTIAL VENTRICULAR CARDIOPLEGIA PRESERVATION THRESHOLDS QUANTIFIED BY THERMODILUTION AND ECHOCARDIOGRAPHIC SYSTOLIC PERFORMANCE INDICES

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Introduction. There is a lack of controlled clinical studies detailing the relationship between cardiopulmonary bypass (CPB) variables and changes in simultaneously determined right and left ventricular (R/LV) global systolic performance indices. The following study addressed this issue.

Methods. 16 patients undergoing coronary artery bypass grafting and fulfilling HIC criteria were studied. Hemodynamic estimates were measured with a 50 msec response thermodilution RV ejection fraction ($RVEF\text{-}3D_{TD}$) catheter appropriately positioned. Two-dimensional echocardiographic (2D-echo) measurements were obtained with a 5.0 MHz transesophageal phased-array transducer. Measurements included heart rate (HR), $RVEF\text{-}3D_{TD}$, RV end-diastolic volume index ($EDVI_{TD}$), pulmonary vascular resistance (PVR), RV long axis (LA) planed area shortening fraction ($RVEF\text{-}2D_{LA}$), RV LA maximal minor axis diastolic dimension (MMD_D),[1] LV short axis (SA) planed area shortening fraction ($LVEF\text{-}2D_{SA}$) and Freeland-Simpson's rule LVEF ($LVEF\text{-}3D_S$). Procedures standardized as to anesthesia management (Fentanyl/Pancuronium; same attending), surgical technique (two stage venous cannula/pulmonary venous venting/core temperature 28°C/"topical cold" solution and crystalloid cardioplegia (CP) myocardial preservation; same surgeon), ultrasonography (standardized views and gain; same attending) and thermodilution measurement technique (injectate temperature, catheter positioning). CP was administered by roller pump infusion via the aortic root. Data were obtained following induction/intubation, sternotomy and procedure conclusion. Analyses were by linear regression and paired or Student's t test, as appropriate.

Results. CPB-associated changes in $RVEF\text{-}2D_{LA}$ (0.47 ± 0.10 vs 0.39 ± 0.11) and $RVEF\text{-}3D_{TD}$ (0.40 ± 0.13 vs 0.36 ± 0.13) were significantly related to total CP dose (1.6 ±0.39 L) ($P < 0.01$; $r = 0.74$ and 0.69, respectively). CP dose was unrelated to corresponding changes in $LVEF\text{-}2D_{SA}$ (0.42 ± vs 0.48 ± 0.19) and $LVEF\text{-}3D_S$ (0.25 ± 0.08 vs 0.25 ± 0.09). Significant CP dose-associated changes were unrelated to RCA disease presence and severity, number of bypass grafts, cross-clamp time, CPB duration, pre-CPB RV function and changes in HR, $RVEDVI_{TD}$, MMD_D, and PVR.

Comment: 1) Right and left ventricular CPB-associated changes in systolic performance indices differed with respect to relationships with CP dose. This inverse correlation between RV thermodilution/2D-echo findings and CP values suggests inadequate right-sided myocardial preservation at lower dose ranges. In contrast, LV performance was maintained across the dose spectrum, suggesting a lower preservation threshold. 2) RV changes were independent of pre-existing RV function, loading conditions and bypass variables other than CP. 3) The differential CP preservation threshold may underscore the syngergism between CP and "topical cold" myocardial protection, with the anteriorly located RV being more susceptible to ambient rewarming.

References:

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TITLE: PROPOFOL-INDUCED ARTERIAL COMPLIANCE INCREASE ACCOUNTS FOR SYSTOLIC ARTERIAL PRESSURE DECREASE.

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Propofol (P) induces a sustained blood pressure fall with transient reduction of cardiac output (CO) and systemic vascular resistance (R), at constant heart rate (HR) and preload.[1] The mechanism of systemic hypotension may be due to systemic vasodilator and/or myocardial depression. The more pronounced decrease of systolic than diastolic blood pressure allows an analysis of the determinants of systolic blood pressure. Among these, systemic arterial compliance (C) seems most important because stroke volume and HR do not change.

After Human Research Committee approval, we studied 11 ventilated patients, 12 hours after coronary bypass surgery, without any vaso-active or inotropic drugs, prior to chest tube withdrawal. We measured: HR (b/min); systolic (SAP), mean (MAP) and diastolic (DAP) arterial pressures (mmHg, low compliance radial catheter); right atrial pressure (RAP, mmHg) and CO (l/min, thermodilution); R=(MAP-RAP)/CO (mmHg/ml/s) and C (ml/mmHg/m^2): before (C) and 5, 10, 15, 20, 30 min after a 2 mg/kg IV bolus injection of P. C was estimated using a linear two-element Windkessel model of the arterial system.[2] Monoexponential diastolic decay was verified for each beat ($R > 0.985$). The last two thirds of the diastolic segment was sampled at 25 ms intervals and individual values of pressure plotted against time on semi-log paper; reciprocal value of the slope and time constant τ was calculated as the mean of 6 consecutive beats and $C = \tau/R$. Zero-flow pressure asymptote was assumed to be at zero level.[3] Statistical analysis: two-way ANOVA and Neuman-Keuls test for multiple comparison procedure.

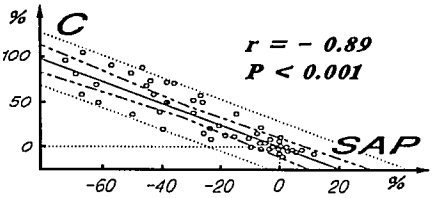
Results:

	HR	CO	SAP	DAP	PWP	R	C
C	86±8	5.5±1.5	113±13	59±6	13±3	12±3	1.02±.28
5	83±8	4.8±1.1 *	77±15 #	43±9 #	11±3	10±2 #	1.62±.32 #
10	82±8	5±1.2	84±11 #	47±7 #	11±3	11±2	1.50±.31 #
15	81±7	5.3±1.1	93±11 #	52±6	12±3	11±2	1.41±.25 #
20	82±7	5.3±1.9	99±11	53±7	12±3	11±2	1.30±.25 *
30	84±7	5.7±0.7	105±15	55±9	13±3	12±2	1.16±.18

* P < 0.01 # P < 0.001 VERSUS C

Systemic hypotension was sustained and primarily due to a reduced systolic component. HR remained unchanged; the low frequency range allowed evaluation of the passive transient response of the vascular tree in diastole. A proportional relationship between the diastolic decay time constant τ and R was found with an intercept close to zero ($R = 0.46$, $P < 0.05$) demonstrating that R approximated total peripheral resistances. C largely increased after P and a strong linear negative relationship exists between C and SAP variations ($R = -0.89$, $P < 0.001$) (figure). In conclusion, the increase of estimated large artery compliance accounts for the sustained hypotension induced by P injection.

Figure:



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