TITLE: THE SYSTOLIC PRESSURE VARIATION MAY UNMASK ACUTE LEFT VENTRICULAR ISCHEMIC DYSFUNCTION UPON WEANING FROM CARDIOPULMONARY BYPASS.

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In the setup of weaning from cardiopulmonary bypass (CPB) aggressive management of blood volume and liberal use of nitrates and inotropic medications may obscure underlying acute ischemic dysfunction. Diagnosis is customarily made according to the left ventricular (LV) function curves as well as clinical signs. We report a simple and sensitive method of diagnosing this problem by analysis of the respiratory induced variations in the systolic pressure (SPV).

Normally a mechanical breath causes an early increase of the systolic blood pressure (SBP) relative to the SBP during apnea (Δup), reflecting a transient augmentation of LV output due to mainly an increase in preload as pulmonary blood is squeezed into the LV. The up is normally followed by a decrease in SBP relative to SBP during apnea (Δdown), reflecting the decrease in venous return due to the mechanical breath (fig 1).

In 28 patients undergoing coronary artery bypass grafting we analyzed the arterial pressure waveform during separation from CPB(A), when sternal retractors were removed (B) and after chest closure (C). Our results are shown in the following table (mean+S.D., *P<0.01:compared to A):

	Δup (mmHg)	Δdown (mmHg)	LAP (mmHg)	SBP (mmHg)
A	4±2	0.5±1	8±4	75±23
B	5±2*	4±2*	7±3	94±16*
C	4±2	8±3*	8±4	103±16*

Fig.1 

Two patients developed acute ischemic LV dysfunction because of inadequate mammary flow and had to be returned to CPB. Both patients showed complete absence of systolic variation and Δup (fig. 2). Both patients improved following an additional graft to the left anterior descending coronary artery. SPV returned immediately, Δup reappeared, and second weaning from CPB was smooth with no catecholamine support (fig. 3).

We conclude that the Δdown reflects the degree by which venous return is hampered by the mechanical breath and as expected, is maximal when the chest is closed. The Δup however, remains prominent even with a fully opened chest as blood is still squeezed into the LV with each breath. The absence of the Δup reflects the loss of LV volume responsiveness and is a unique sign of ischemic LV dysfunction during weaning from CPB.

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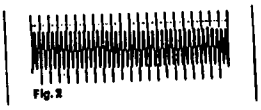
Fig.2


Fig.3

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Title: TRANSESOPHAGEAL ATRIAL PACING FOR INTRAOPERATIVE BRADYCARDIA: PACING THRESHOLDS WITH MODIFIED ESOPHAGEAL STETHOSCOPE IN ADULTS

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Bradycardia due to sinus node dysfunction (SND) or AV junctional rhythm (AVJR) can be the cause for circulatory insufficiency, particularly in patients with marginal cardiac performance due to ischemic, dilated or hypertrophic cardiomyopathy. Temporary pacing could have advantages over drugs for management of bradycardia in these patients (prompt restoration of hemodynamically effective rhythm, no adverse side effects/drug interactions), but established pacing routes (transvenous, epicardial) are too invasive for routine use and transcutaneous ventricular pacing[1] fails to preserve atrial transport function or may be ineffective in some patients. Transesophageal atrial pacing (TAP) can be used to manage bradycardia due to SND or AVJR.[2] TAP would appear ideal for use by anesthesiologists in unconscious, intubated patients, except that available TAP systems (Arzco, Chicago, IL) are designed for use with an electrode which is swallowed by the patient. Consequently, we have modified thermistor-equipped esophageal stethoscopes (ES) for TAP by incorporating a bipolar pacing probe (Tapcath™, Arzco) into the ES. We report here TAP thresholds using this system in adult surgical patients.

With our Human Subjects Committee approval, 48 consenting adults (ASA status 1 to 3) had TAP thresholds determined following induction of general anesthesia and endotracheal intubation. TAP electrodes (3.0 mm wide x 1.0 cm apart) were exposed at the level of the ES diaphragm in a manner which did not interfere with acoustics or thermistor function. TAP electrode distances (cm from incisors) and thresholds (mA) were recorded at: 1) point of maximum amplitude, bipolar esophageal ECG P-wave (P_{max}); 2) point of minimum obtained TAP threshold (T_{min}). TAP pulse duration was 10 msec at a rate 10-15% above spontaneous. Data (mean±SEM) for 28 male (age 47±4 yrs) and 20 female patients (age 48±4 yrs) are tabulated.

	Pmax		Tmin	
	(cm)	(mA)	(cm)	(mA)
Male	33±0	9.0±1.0	34±0	5.6±0.6
Female	32±1	9.6±1.9	33±1	5.4±0.9

T_{min} were constant over 1.6±0.1 cm in male and 1.4±0.2 cm in female patients. Sixteen male and 4 female patients were paced for sinus bradycardia (≤60 beats/min), all with hemodynamic improvement. No patients were paced for AVJR, and there were no complications of TAP.

Observed pacing thresholds are slightly lower than previously reported,[3] possibly due to differences between the pulse generators and electrodes used. We conclude that TAP provides safe and practical management for bradycardia due to SND in anesthetized adults.

References: 1) *Anesth Analg* 69:229, 1989; 2) *Circulation* 75 (Suppl III):86, 1987; 3) *Anesthesiology* 65:428, 1986