Title: ARE REGIONAL WALL MOTION ABNORMALITIES DETECTED BY TRANSESOPHAGEAL ECHOCARDIOGRAPHY TRIGGERED BY ACUTE CHANGES IN SUPPLY AND DEMAND?

Authors: J. Leung M.D., B. O'Kelly M.B., W. Browner M.D., M.P.H., J. Tubau M.D., D.T. Mangano Ph.D., M.D.

Affiliation: Departments of Anesthesia, Medicine, and Epidemiology, University of California, San Francisco and the Veteran's Administration Medical Center, San Francisco, CA 94121

Introduction

Regional wall motion abnormalities (RWMA) as detected by intraoperative transesophageal echocardiography (TEE) are more sensitive and earlier markers of myocardial ischemia than electrocardiographic changes.[1] More importantly, the persistence of RWMA may predict postoperative myocardial infarction. Despite the growing use of TEE for detection of regional ischemia, the etiology of these RWMA is unknown. In order to assess the relative contributions of acute changes in myocardial oxygen supply and demand to the cause of RWMA, we continuously measured hemodynamic variables (heart rate, systemic and pulmonary arterial blood pressures) and related these changes to RWMA detected by TEE in high risk patients undergoing coronary artery bypass grafting (CABG).

Methods

Prior approval from the Human Research Committee was obtained to study 40 patients (42-78 years of age) undergoing elective CABG. Anesthetic agents consisted of either high dose sufentanil, fentanyl, or isoflurane. Heart rate (HR), systolic and diastolic blood pressure (SBP,DBP), as well as systolic and diastolic pulmonary arterial pressures (PAS,PAD) were recorded continuously from induction to the early ICU period onto hard copy at 1mm/sec. Pulmonary capillary wedge pressure (PCWP) was measured as clinically indicated intraoperatively, as well as hourly in the ICU upon arrival for 4 hours. A significant change in hemodynamics was defined as an acute change ±20% of control at 5 and/or 10 min before the onset of RWMA. Following endotracheal intubation and placement of the TEE probe, the left ventricle was continuously imaged at the level of the mid-papillary muscles throughout the peri-operative periods (prebypass, postbypass-intraoperative), and also for 5min/hr for 4hrs in the ICU. This short axis view was divided into 4 equal segments using the papillary muscles as landmarks. The echo data was edited and each segment graded by 2 investigators using 0=normal, 1=mild hypokinesis, 2=severe hypokinesis, 3=akinesis, 4=dyskinesis. Significant RWMA were defined as those segments with a change of grade ≥2. All clinicians were blinded to the echo data collected during the perioperative periods.

Results

A total of 49 episodes of RWMA were detected in 20 patients: 17 prebypass, 22 postbypass, and 10 in the ICU. RWMA were preceded by acute ↑ in HR in 8% of cases, ↑ in SBP (14%), ↓ in DBP (12%), or ↑ in PAD/PCWP (10%). Overall, 39% of all echo changes were preceded by acute increases in either HR, SBP or PAD/PCWP, or acute decreases in DBP (Fig.1). The incidence of RWMA with hemodynamic-related changes (↑HR, ↑SBP, ↓DBP, or ↑PAD/PCWP) was 4/17(24%) prebypass, 12/22(55%) postbypass, and 3/10(30%) in the ICU (Fig.2). Although the majority of RWMA was not associated with acute changes in hemodynamics, RWMA in the early postbypass period tended to be associated with lower SBP and DBP. RWMA in the ICU however, were associated mostly with higher HR (table 1).

Discussion

This is the first study to examine the relationship of acute changes in supply and demand to RWMA. We demonstrated that: 1) approximately one-third of all RWMA were preceded by a >20% increase in HR, SBP, PAD/PCWP or decrease in DBP, 2) most of these hemodynamic-related echo abnormalities occurred in the early postbypass period, and 3) only 10% of all RWMA were preceded by an acute rise in PAD/PCWP, suggesting that such an elevation is not an early marker of regional myocardial ischemia.

Thus, tighter control of hemodynamics may be important in the immediate postbypass period in reducing myocardial ischemia when there are rapid fluctuations in supply and demand. Since the majority of the RWMA was not triggered by acute changes in hemodynamics, other mechanisms that involved a primary decrease in myocardial oxygen supply must be considered and deserve further investigation.

References

1. Smith JS et al: Circulation 72(5):1015-1021,1985

Fig. 1 RWMA with hemodynamic-related changes

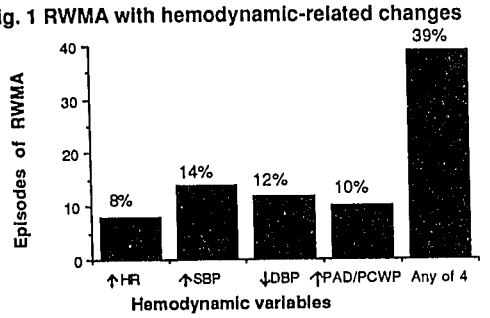

Fig.2 Distribution of hemodynamic-related RWMA

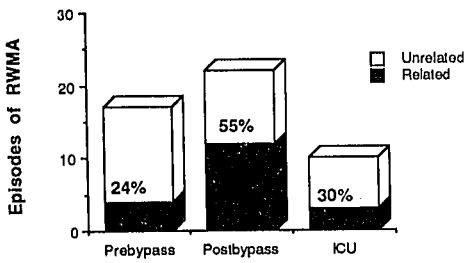

Table 1 Hemodynamics 10",5" before, and at onset of RWMA (means±SEM)

Pre=prebypass, post=postbypass

		10" before	5" before	Onset
HR	Pre	62±3	63±2	62±2
	Post	79±3	79±4	78±3
	ICU	84±8	90±5	92±5
SBP	Pre	121±4	122±5	117±5
	Post	89±7	91±7	92±7
	ICU	119±5	130±7	125±11
DBP	Pre	64±2	64±3	64±3
	Post	49±2	52±3	52±3
	ICU	63±2	72±2	70±3
PAD	Pre	15±1	14±1	15±1
	Post	11±1	12±2	12±1
	ICU	13±1	12±1	12±1