

SCA 102

BIVENTRICULAR PACING AS TREATMENT FOR HEMODYNAMIC INSTABILITY DURING OPCAB SURGERY IN PATIENTS WITH ISCHEMIC CARDIOMYOPATHY AND LOW EJECTION FRACTION.

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Introduction: Biventricular pacing (BVP) is a recognized treatment option to improve functional status, ejection fraction (EF) and quality of life in patients with severe chronic heart failure-NYHA class III [1,2]. BVP or resynchronization therapy improves in dp/dt, mitral regurgitation (MR), and end systolic and diastolic volumes by improving synchrony between LV and RV contraction [3].

OPCAB surgery has been reported to be associated with less morbidity compared to CPB-CABG and is increasingly being considered in high risk patients with compromised LV function. We here report four cases where BVP was used to acutely augment cardiac output and maintain hemodynamic stability in patients with low EF ischemic cardiomyopathy during OPCAB surgery.

Method: All patients received a general anesthetic with the induction and maintenance agents selected by the attending anesthesiologist caring for the patients. Intraoperative monitoring included a pulmonary artery catheter, arterial line, continuous cardiac output monitor with pulse wave contour analysis and intraoperative transesophageal echo (TEE). Prior to incision, a comprehensive intraoperative TEE exam was performed. Baseline tissue Doppler imaging and analysis along with CO, EF, HR and MAP were recorded. When pacing was deemed clinically necessary, three epicardial pacing leads were placed on the heart: one on the free wall of the RV, one on the inferior wall of the LV, and one on the right atrium. The patient was paced (Medtronic®Milaca, MN) using an external pacer generator set at 20 mAmp voltage and DDD pacing mode at a rate of 90 bpm. During BVP, tissue Doppler imaging and analysis along with CO, EF, heart rate and MAP were again recorded. Tissue Doppler analysis included measurement of peak systolic tissue velocity (TV), strain (e) and strain rate (SR) as index of myocardial performance[4]. Time to peak TV and SR were also measured and used as markers of synchronicity between the RV and LV. All patients had normal QRS duration and were paced at rates similar to baseline rates.

Case #1: Patient PP is a 78-years-old female patient with history of hypertension (HTN), coronary artery disease (CAD), admitted to the emergency room in fulminant pulmonary edema. She went to OR for 3-vessel CABG. BVP was used to improve cardiac index (CI) along with hemodynamics.

Case #2: Patient CI is a 48-year-old female with history of insulin dependent diabetes mellitus, asthma, HTN, systemic lupus erythematus with renal insufficiency, proteinuria and SOB. Patient went to OR for four vessels CABG. BVP was used to decrease MR during manipulation of the heart.

Case #3: Patient DR is a 63-year-old male with history of hyperlipidemia, congestive heart failure, three vessels CAD, symptomatic with shortness of breath and chest pain. MR worsened during positioning for anastomosis and BVP was initiated to help improve cardiac output and reduce MR.

Case #4: Patient BS is a 81-year-old female with history of HTN and hyperlipidemia, with dyspnea and an aching substernal chest pain. Catherization revealed left main diseases. Patient went to OR for 3-vessel CABG. BVP initiated to improve CO.

	Case #1		Case #2		Case #3		Case #4	
	Base	BVP	Base	BVP	Base	BVP	Base	BVP
CI (l/min m ²)	2.2	3.4	2.83	6.4	2.67	7.1	3.94	6.55
EF %	30	40	40	55	35	55	25	45
Septal TV (cm/s)	-1.2	-1.7	-2.25	-2.75	-3.14	-3.93	-1.19	-1.45
Time to peak TV (ms)	232	88	91	79	114	90	214	227
Peak %	-6.9	-9.3	-5.5	-10.6	-14.9	-17.5	-13.3	-16.9
Peak SR (1/s)	-7	-9	-1.1	-1.6	-1.8	-4.1	-1.2	-1.9
Time to peak SR (ms)	154	113	133	64	103	97	372	136

Summary: We observed improvement in all markers of systolic performance i.e. (CI, EF, peak TV, strain and SR) following BVP. Time to reach peak systolic function also improved with BVP. Acute ventricular resynchronization therapy is feasible, improves cardiac performance and warrants further studies in the setting of cardiac surgery for treatment of acute low output syndrome.

References: 1. NEJM 2001. 344(12); 873. 2. NEJM 2002. 364(24): 1845. 3. Circulation 2002. 105(4): 438. 4. JACC 2001. 38(7); 1966