

E370 JACC March 27, 2012 Volume 59, Issue 13

Acute Coronary Syndromes

ROLE OF PASSIVE LEG RAISING TO EVALUATE PRELOAD RESPONSIVENESS IN PATIENTS WITH CARDIOGENIC SHOCK AFTER ST ELEVATION MYOCARDIAL INFARCTION (STEMI) TREATED WITH INOTROPIC THERAPY

ACC Moderated Poster Contributions McCormick Place South, Hall A Sunday, March 25, 2012, 9:30 a.m.-10:30 a.m.

Session Title: Acute Coronary Syndromes: Therapy II Abstract Category: 4. Acute Coronary Syndromes: Therapy Presentation Number: 1159-592

Authors: <u>Marco Poli</u>, Paolo Trambaiolo, Valentina Basso, Marina Mustilli, Massimo De Luca, Vjerica Likic, Maurizio Simonetti, Federica Ferraiuolo, Giuseppe Ferraiuolo, Intensive Cardiac Care Unit - Sandro Pertini Hospital, Rome, Italy

Background: Optimal cardiac filling is essential for maintaining an adequate cardiac output and organ perfusion in patients with cardiogenic shock (CS) after STEMI). Passive leg raising (PLR) represents a "self-volume challenge" that could predict fluid response; the transient hemodynamic effect of PLR on left ventricular stroke volume (SV) detect preload responsiveness in patients with CS after STEMI. Aim of this retrospective study was to determine the agreement between PLR and early hemodynamic status of patients with CS after STEMI.

Methods: During observation period (16-months), 79 patients were admitted in our ICU for CS after STEMI; patients who required an intra-aortic balloon pump (n = 15) and/or mechanical ventilation (n=21) and which were not monitored with PiCCO[®] (n=31) were excluded from the analysis. The final pool included in the study was 12 patients. The hemodynamic management of studied patients was based on guideline treatment, and to maintain individual cardiac index between 1.5 and 2.7 L/min/m2, all patients were treated with an inotropic agent. Heart rate (HR), arterial blood pressure (ABP), CVP, PAOP, lactate plasma levels (LAC), SV, were recorded in a supine position (baseline position).and after that patients were in a supine position with the lower limbs elevated 30° to 45°. Each hemodynamic measurement was recorded within the first 5 mins. Patients were considered as fluid preload responsiveness if PLR induced SV increased by $\geq 10\%$. We measured SV using PiCCO[®] monitor, we calculated changes in SV, induced by PLR. Among 12 patients included in this study, 5 had a SV increase of >10% after PLR.

Results: In responders group (n = 5) SV was significantly increased by PLR from 47 ± 14 mL to 50 ± 14 mL (p < .001). In this patiens infusion within 15 mins by 100 mL of 6% hydroxyethyl starch (Voluven[®]) increased SV from 47 ± 14 mL to 53 ± 15 mL (p <.001). In non responders group (n = 7) SV has not significantly increased by PLR, from 42 ± 12 mL to 45 ± 11 mL. In this patiens volume expansion increased SV of 45 ± 14 mL.

Conclusions: Changes in SV and radial pulse pressure induced by PLR are accurate and interchangeable indices for predicting fluid responsiveness in patients with CS after STEMI