dynamic parameters (heart rate, blood pressure, grade 3 or 4 Killip, left ventricular ejection fraction (LVEF)). In group 1, predictive factors of mortality were: age, three-vessel or left main coronary artery disease, blood pressure, grade 3 or 4 Killip and LVEF.

Conclusion: Despite recent improvement in the management of STEMI, incidence of patients requiring CAD for CS is still high, and mortality elevated.

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Effect of acute hypoxia on left ventricular twist assessed by speckle tracking imaging

Chantal Dedobbeleer (1), Gael Deboeck (2), Michel Lamotte (2), Robert Naeije (3), Philippe Unger (1)
(1) Hôpital Erasme, Cardiologie, Bruxelles, Belgique - (2) Hôpital Erasme, Bruxelles, Belgique - (3) Hôpital Erasme, Physiopathologie, Bruxelles, Belgique

Background: The impact of hypoxia on left ventricular (LV) myocardial contractility remains debated.
Objectives: We hypothesized that acute hypoxia in healthy people increases LV twist as a consequence of the cardiovascular adaptation to stress (increased sympathetic nerve activity). Because hypoxia increases heart rate, we aimed to control for this change by studying the same subjects under atropine during normoxia.

Methods: We studied 21 subjects (mean age: 27 ± 7 years) without medical history. Echocardiography was performed in normoxia and after 30 minutes of hypoxia (12% FiO_2). Short axis basal and apical views were analyzed using speckle tracking software. LV twist was defined as the net difference between the apical and basal rotation. The effect of atropine on LV twist was tested in the same subjects under normoxia.

Results: As expected, hypoxic breathing decreased arterial saturation in oxygen below 80% (table). Peak LV twist increased, as did heart rate, LV ejection fraction (LVEF) and systolic mitral annular velocity. Despite a decrease in mitral E/A ratio, early diastolic LV untwisting (at 5%, 10% and 15% of diastole) was not significantly modified under hypoxia.
Atropine did not alter peak systolic twist (8.9 ± 3.1° versus 10.1 ± 2.6°,p=0.09, without and with atropine, respectively), despite an increase in heart rate (from 64 ± 11 to 77 ± 11 beats/min, p=0.0001).
Conclusions: Acute hypoxia increases LV twist as well as other parameters of LV contractility. This change in LV twist does not result from the chronotropic effect of hypoxia.

Values are means (SD)

	Normoxia	Hypoxia	p value
SaO_2 (%)	97 (1.3)	76 (7.8)	0.0001
Heart rate (beats/min)	64 (11)	70 (9)	0.008
LVEF (%)	65 (5)	68 (7)	0.011
Systolic mitral annular velocity (cm/s)	8 .0 (1.1)	8.7 (1.4)	0.03
E/A	2.1 (0.6)	1.8 (0.4)	0.015
Peak LV twist (°)	8.9 (3.1)	11.1(2.8)	0.003

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Predictors and prognostic value of contrast-induced nephropathy in patients undergoing primary angioplasty

Antonio Gaspar, Joao Costa, Vitor Ramos, Silvia Ribeiro, Catarina Vieira, Sérgio Nabais, Sergia Rocha, Pedro Azevedo, Miguel Pereira, Alberto Salgado, Adelino Correia
Hospital de Braga, Cardiologie, Braga, Portugal

Purpose: Contrast-induced nephropathy (CIN) after coronariography has been associated to increased morbidity and mortality. Patients submitted to primary angioplasty seem to be at higher risk for CIN development, owing in part to hemodynamic status. We sought to determine the prevalence, predictors and prognostic value of CIN occurrence after primary angioplasty.

Methods: A total of 141 patients consecutively submitted to primary angioplasty and admitted to our coronary unit were reviewed. CIN was defined as impairment of renal function occurring within 48 hours after administration of contrast media and manifested by an absolute increase in the serum creatinine level of at least 0.5 mg/dl or by a relative increase of at least 25% over the baseline value (in the absence of another cause). The primary end points were in-hospital and six-month mortality.

Results: CIN developed in 18.4% of the patients (n=26). Patients with CIN were older (68 ± 13 vs 61 ± 13 years; p <0.05) and more often had diabetes mellitus (38.5% vs 15.7%; p <0.05). Although statistical significance was not reached, there was a trend for higher prevalence of hypertension (61.5% vs 42.6%; p=0.09), female gender (30.8% vs 18.3%; p=0.18) and Killip class higher than one at admission (26.9% vs 16.3%; p=0.1) among patients with CIN. Patients with CIN had an higher mean time from symptoms to reperfusion (304 ± 192 vs 397 ± 206 minutes; p <0.04). By multivariate analysis, independent correlates of CIN were older age (OR=1.04; 95%CI=1.01 – 1.08) and diabetes mellitus (OR=2.99; 95%CI=1.08 – 8.3). Patients with CIN had higher in-hospital (19.2% vs 0.9%; p <0.05) and 6-month mortality (28.6% vs 4.9%; p <0.05).

Conclusions: CIN was a frequent complication of primary angioplasty (18.4% of the patients). Independent predictors of CIN after primary angioplasty were older age and diabetes mellitus. Patients with CIN had a worse prognosis, both during in-hospital stay and at 6 months.

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Use of recombinant factor VIIa (NovoSeven) in patients treated with fondaparinux for ongoing life-threatening bleeding

Francois Schiele (1), Paul Luporsi (1), Nicolas Meneveau (1), Evelyne Racadot (2), Vincent Descotes-Genon (1), Romain Chopard (1), Marie-France Seronde (1), Sebastien Janin (1)
(1) CHU de Besançon, Cardiologie, Besançon, France - (2) EFS Bourgogne-Franche Comte, Besançon, France

Background: Recombinant factor VIIa (rFVIIa) may be used to reverse the anticoagulant effect of fondaparinux. We report a single centre experience in 8 patients with severe bleeding.

Methods: Patients pretreated with fondaparinux, with life-threatening bleeding were treated with 90µg/kg rFVIIa. Life-threatening bleeding was defined as TIMI 3 bleeding or a drop in hemoglobin > 5g or hemodynamic shock and elevated antiXa activity. Endpoints were (1) death (2) persistent bleeding (clinical or continued drop of hemoglobin) (3) uncontrolled hemodynamic shock (4) clinical arterial or venous thrombosis and (5) peak of thrombin generation.

Results: Between June 2008 and November 2009, among 1224 patients treated with fondaparinux, 8 presented with life-threatening bleeding (3 with venous thrombo-embolic disease, 5 acute coronary syndrome (ACS)). Patients with ACS had double (n=2) or triple (n=3) antiplatelet therapy. Bleedings were related to vascular access in 5, gastro-duodenal in 2 and lung in 1. Five patients had hemorrhagic shock, mean drop in hemoglobin was 6.1 g/dL. Anti-Xa activity ranged from 0.67 to 1.62, rFVIIa dose ranged from 3.6 to 7.65mg. One patient died from uncontrolled shock, no patient had signs of persistent bleeding or thrombotic complication. In patients with the highest basal anti-Xa activity (1.14 to 1.62), the time to peak of thrombin generation remained low.

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