Does atrial differences in endothelial damage, leukocyte and platelet activation contribute to chamber specific thrombogenic status in patients with atrial fibrillation?

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Background: In atrial fibrillation (AF), the reasons why most of the thrombus form in the left atrium are mainly unknown. In the vasculature, endothelial damage together with platelet activation and inflammation contribute to initiation of blood coagulation and thrombus growth.

Objective: The purpose of this study was to investigate whether atrial-specific differences in endothelial damage, leukocyte activation, platelet stimulation occur in patients with AF.

Methods: Twenty patients (15 men, 5 women; age 55 ± 8 years, 15 paroxystic AF, 5 persistent AF) with AF undergoing ablation were investigated. Blood samples from the left and right atrium were obtained at the start of the procedure. Procoagulant microparticles (MP), reliable markers of vascular damage were measured by capture assays. Their procoagulant abilities were quantified by functional prothrombinase assay and their cellular origin were determined (endothelium, platelet, leukocyte). In addition, platelet reactivity was evaluated by whole blood flow cytometry for expression of platelet P-selectin (CD62P), active glycoprotein IIbIIIa receptor (PAC-1). Platelet aggregation was evaluated using Arachidonic acid (AA), ADP, TRAP and collagen-induced whole blood aggregometry.

Results: No atrial-specific differences in the levels of total procoagulant MP, leukocyte-derived-MP and platelet-derived MP could be evidenced. Conversely, endothelial-derived MP (CD105+) were slightly elevated in the right atrium (RA 0.96 ± 0.53 vs. LA 0.80 ± 0.45 nm PthDser Eq.; p = 0.041). Likewise, collagen-induced platelet aggregation was evidenced in the right atrium (Collagen 1mg/l: RA: 48 ± 33 % vs LA 37 ± 29%; p = 0.035; collagen 2.5mg/l: RA: 76 ± 25 % vs LA: 60 ± 29%; p = 0.001).

Conclusions: In patients with AF, endothelial damage and collagen-induced platelet aggregation appear slightly more pronounced in the right atrium. Our data did not substantiate the view that chamber specific enhanced thrombogenic status could be a reliable explanation for the increased propensity for thrombus formation observed in the left atrium in AF patients.

T wave variability (TWV); circadian variations in patients after acute coronary syndrome (ACS)

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Introduction: Risk stratification is a major challenge in ischemic cardiomyopathy. The prognostic significance of TWV measured by amplitude variance of T wave amplitude has been proved. But, short-term and midterm circadian variations of TWV are unknown.

Objectives: We measured TWV during 24 hours in pts with normal or moderately altered LVEF at day 7 (D7) and after cardiac rehabilitation at day 40 (D40), after an ischemic event.

Methods: Study population consisted in 48 pts after ACS (45 males, aged 59±11, LVEF 53±10%, treated by angioplasty (88%), CABG (8%) or medically (4%). All pts have had 24H Holter recording (Sorin group, sampling at 1000Hz) divided in 30 min-periods of measurement of TWV ; we compared TWV at day 8am-9pm and nighttime TWV (11pm-6am). Holters have been performed at D7 and D40 (90±54). We also compared TWV at rest during the first 30min-period at D7 and D40. There were no changes in LVEF and coronary status between both Holters.

Results: At D7 and D40, circadian analysis showed maximum values from noon to 4 pm followed by a down sloping with a nadir between 6 and 7 am and an increase between 7 and 9 am. On the short term curve (D7), the daytime TWV amplitude was lower. At D7, TWV did not differ significantly between day and night (20.3±4.2 µV vs 18.4 ± 7.7 µV, p=0.072, decrease of 9.35%). At D40, TWV was significantly higher during day period (21.1±5.4µV vs 18.4±6.9µV, p=0.009, decrease of 12.79%). Between D7 and D40, TWV at rest during a 30 min-period were not significantly different (18.3±5.4 µV vs 18.2 ± 6.8 µV, p=0.885).

Conclusion: TWV has circadian variations, as shown for blood pressure and heart rate. The TWV was significantly higher during daytime than in sleeping period. These findings lead to recommend measuring TWV after cardiac rehabilitation but further studies are needed to precise the best study period during daytime.

Extreme inter-atrial conduction slowing without Bachman’s bundle block.

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Introduction: A complete inter-atrial conduction block (CIA) is defined as a surface ECG p wave duration 120ms with a biphatic pattern in inferior leads. The main mechanism is thought to be in complete Bachman’s bundle block responsible for a retrograde left atrium depolarization. Some patients may meet the p wave duration criterion for CIA without a biphatic pattern in inferior leads. We hypothesized that some patients may present extreme inter-atrial conduction slowing without Bachman’s bundle block.

Methods: Ten patients with p wave prolongation (p > 120ms) were divided into two groups: five patients with a biphatic pattern in inferior leads (BP group), and five patients without a biphatic pattern in inferior leads (No-BP group).

Conduction velocity from right to left atria was measured invasively using a 24-pole mapping catheter placed around the tricuspid annulus with distal dipoles positioned at the lateral wall of the left atrium (at 3 hours around the mitral annulus in LAO 45° view), and proximal dipoles at the lateral wall of the right atrium (at 9 hours around the tricuspid annulus in LAO 45° view).

Results: BP group had shorter p wave duration compared to No-BP group (140 ± 10ms vs 170 ± 15ms vs, p=0.05). As expected, BP patients had a retrograde left atrium depolarization confirming a complete Bachman’s bundle block conduction block. In contrast, wave front collisions were recorded at the lateral wall of the left atrium (distant dipoles 1-2) in all five No-BP patients.
Conclusions: As opposed to patients with CIACB, patients with an extreme p wave prolongation (>150ms) and no biphasic pattern may have a delayed conduction through the Bachmann’s bundle without a complete block.

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Study of the circadian variation of the QT dynamics in myocardial infarction
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Introduction: The relation between the QT interval and heart rate is linear. The QT interval/RR interval relationship is generally described as QT dynamics. The linear QT/RR slope is influenced by the autonomic nervous system. In healthy individuals, the slope exhibits circadian variations: it is steeper during the day than during the night.

Aim: The aim of this study is to evaluate the circadian variation of the QT interval and the QT/RR slope in patients experiencing myocardial infarction.

Methods: This prospective study included 90 patients having myocardial infarction. They underwent 21 days after the acute phase, 24 hours ambulatory ECG (Holter) recording. The following parameters were studied: the QT end interval (QTe), the QT apex interval (QTA) and the slopes of QTe/RR and QTa/RR during diurnal and nocturnal periods.

Results: There was no significant difference regarding the QTe and QTa intervals during the day and night. The mean diurnal slope and nocturnal slopes of QTe/RR were similar (0.147±0.073 vs 0.131±0.062, p = NS). The mean diurnal slope and nocturnal slopes of QTa/RR were also comparable (0.123±0.067 vs 0.119±0.065, p:NS).

Conclusion: The lack of circadian variation of the QT interval and the linear QT/RR slope in myocardial infarction patients reflects an imbalance in the autonomic nervous system. These results suggest an increase in the nocturnal sympathetic tone and may be associated with a higher risk of arrhythmia in this population. Further studies are needed to evaluate the role of the QT/RR slope in risk stratification after myocardial infarction.

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Infection on cardiac devices. A monocentric prospective study
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Purpose: The infections after cardiac device implantation (CDI) are not well known in the real life because of the multiplicity of the circumstances. This is a monocentric prospective study.

Method: 304 consecutive pts had a CDI in 6 months (feb to aug 2009): male 69% age 70±15 yo. The data of the pts were consecutively collected:

- type of device (VVI PM 10%, DDD PM 42%, CRT P 7%, VVI ICD 13%, DDD ICD 10%, CRT D 17%) (Primo Implant 73%, Device Replacement 21%, Lead Replacement 8%, Burying 2.3%);
- components of the NNIS score (N1, Nosocomial Infection Surveillance): -1 duration of the procedure (83±40 min) - 2 ASA score (Am.Soc.Anesth)xascal=4%, asa2=22%, asa3=43%, asa4=30%, asa5=10%); -3 surgical site Infection (SSI) Class: Clean wound 73%, fever the day before 6%.
- the prevention with antibiotic therapy was: - conventional: Cefuroxime 1.5 g 30 mn before and 0.75g each 2hr (93% ; 69% timing conformity); - Vancomycine 1gr 1hr before (3%);
- the follow-up was of 3 months.

Results: 7 SSI occurred at 43±36 days (2.3%) for DDD PM=5, VVI ICD=1, CRT D=1. They were 2 endocarditis (2 infections on the other side after an extraction, with the same germ) sepsis = 1 (diabetes), loge infection = 4 (2 after buring). The 7 pts had an extraction of the device and the leads. The risk factors of SSI were antibioticociation (7/7 ; p =0.05); controlateral implantation after previous sepsis (2/7 ; p < 0.05), buring on the same site (2/7 ; tendency), admission in Intensive Care Unit (3/7 ; tendency), antibiotic therapy too early (3/7 ; tendency).

Conclusion: SSI and sepsis after CDI depends of the clinical surroundings (Anticoagulation, Previous Sepsis, Buring, Intensive care unit. The complexity of the device (PM, ICD, CRT) has a low role.

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Is high sensitive C reactive protein related to clinical and echocardiographic risk of thrombo-embolism in patients with atrial fibrillation?
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Introduction: Atrial fibrillation is associated with a prothrombotic state with an increased risk of stroke. Recent studies suggested that there is an apparent link between thrombogeneses and inflammation.

Aim: We sought to study the relation between high sensitive C reactive protein (HS CRP) and clinical (CHAD score) and echocardiographic prothrombotic indexes in patients with atrial fibrillation.

Methods: We prospectively measured HS CRP in 100 patients with atrial fibrilation. The mean age was 56±12 years. All patients underwent transesophageal echocardiography (TEE). The TEE risk factors for thromboembolism considered were: a peak left appendage velocity 0.2 m/s, the presence of a thrombus and a dense spontaneous echo contrast.

Results: HS CRP was correlated with the clinical CHAD score (r =0.54, p =0.0001). CRP value was significantly higher among patients with a CHAD score 2 (6.9± 4.4 mg / dl vs 4.35 ± 3.8, p =0.001). Values of HS CRP were comparable between patients having 1 TEE risk factor and those with no TEE risk factor (5.13 ± 3.7 vs 6.5 ± 0.5 p = NS)

Discussion and conclusion: The significant correlation between the CHAD score and HS CRP could be explained by the existence in this score of factors associated with elevated CRP. Although no apparent relation was found between the HS CRP and echocardiographic risk factors of thromboembolism, these results do not exclude the inflammatory hypotheses in the pathogeneses of thromboembolism. A study of the correlation between CRP and thromboembolic complications during follow up of patients is mandatory.

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Renal failure after CRT implantation: more than a contrast nephropathy
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Background and purpose: We studied short term effect of CRT implantation on renal function and focused especially on the role of contrast agent dose.

Method: Acute renal failure (ARF) was defined as a decrease of more than 25% of the clearance of creatinine (CI Cr) calculated with MDRD method within four days following implantation.

Results: We considered 141 patients referred for CRT implantation: male 79%, mean age: 68 +/-12; diabetes: 71%; HTA: 38%; CI Cr: 48 mL +/-19, median contrast dose: 48 mL (IR: 30/60), median BNP variation: -30% (IR: -58/-8), Haemoglobin (Hb) variation: -6.3% +/-11.2.

ARF occurred in 19 patients (13.5%), 3 of them died and one was lost. Renal function of 14 out of the 15 remaining completely recovered. In-hospital stay longer than 10 days was more frequent in ARF group (OR=5.18, p<0.03).

Decrease of Hb was the only independent factor of ARF with a negative correlation (OR=0.94, p < 0.01).

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