HEART RHYTHM SOCIETY - 36. SCIENTIFIC SESSION 2015

13.-16. MAI I BOSTON

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Her et kort summarisk referat fra en delvis sengeliggende stedlig redaktør.

Årets Heart Rhythm Society-kongress fant sted i Boston, et sted hvor kongressen har vært mange ganger før. Antallet deltakere var angivelig nesten 5000. Etter flere år med meget få norske deltakere klarte jeg å telle 15 norske arytmokardiologer. Det kan virke som om de lokale helseforetak har innsett viktigheten av at vi må holde oss oppdaterte.

Det var 6 norske bidrag, derav 2 muntlige. Hans Henrik Odland fra Oslo universitetssykehus, Rikshospitalet, ga en fin oversikt med tittelen: «Dyssynchrony-Induced Cardiomyopathies: When Do I Ablate Pre-Excitation or PVCs in Cardiomyopathy?», og Svein Færestrand presenterte «More Options for Targeted Placement of the Left Ventricular Lead By Using a New Transvenous Active Fixation Lead» på veiene av Haukeland universitetssykehus.

Det er dessverre ikke så lett å få skriftlige bidrag fra travle kongressdeltagere, men det inntrykket jeg sitter igjen med er at det ikke var store revolusjonerende nyheter på årets kongress. Noe jeg sitter igjen med:

Når det gjelder antikoagulasjon er NOAK-ene veletablert, og i praksis brukes disse før og etter radiofrekvensablasjon for atrieflimmer i større og større grad. Antidot er rett rundt hjørnet, og problemet med at man ikke kan monitorere behandlingen (etterlevelse) før radiofrekvensablasjon løser de fleste med transøsofageal ekkokardiografi før prosedyren.

Etter STAR-AF2-publikasjonen, som viser at lungeveneisolering kan være et godt nok første trinn ved ablasjon ved persisterende atrieflimmer, er det utbredt usikkerhet rundt substratmodifikasjonens plass. Likevel det er flere som ikke har forandret ablasjonsstrategi på grunn av denne ene studien. Det blir spennende å se om funnene kan bekreftes ...

Livsstilsintervensjonen, som i seg selv har effekt ved paroksysmal atrieflimmer, og som trolig øker sjansen for vellykket ablasjon, har fått større oppmerksomhet i form av en egen sesjon, og jeg ser frem til å se hvordan dette området utvikler seg.

Det var overraskende lite om venstre aurikkel-lukning. Er det de invasive kardiologene som driver mest med dette i det store utlandet ?

Norske abstrakter på kongressen

AB17-05 - More Options for Targeted Placement of the Left Ventricular Lead By Using a New Transvenous Active Fixation Lead. Svein Faerestrand, MD, PhD, Haavard Keilegavlen, MD and Thomas Hovstad, MD. Dept. of Clinical Science, University of Bergen, Bergen, Norway, Dept. of Heart Disease, Haukeland University Hospital, Bergen, Norway

Introduction: Dislodgements of left ventricular (LV) leads are a challenging problem in cardiac resynchronization therapy. The novel active fixation 4Fr Attain® Stability™ (Medtronic,Inc) LV lead was implanted to study the handling, performance, safety and long-term stability.

Methods: The 82 enrolled patients (pts; age 69year±9year) included pts with dislodged LV leads and with very difficult coronary vein anatomies. The lead was targeted to a vein concordant to the LV segment with latest mechanical contraction decided by radial strain echocardi-

ography. The lead body was rotated clockwise to engage the side helix in the vein wall and the stability was tested by pushing and pulling the lead during fluoroscopy.

Results: The LV lead was successfully implanted to the target position and the helix precisely fixated in 80 patients of 82 patients (97 %). The LV lead was successfully fixated to the target position in the first attempt in 57 pts (68 %), and in the second fixation attempt in 18 pts (21 %). In 1 pt lead fixation was not achievable. The LV lead implant time was 18 min±16 min and was proportional to increasing complexity of the vein anatomy. Mean pacing threshold was 1.1V± 0.6V. There was no LV lead related complications and no LV lead dislodgments during removal of guiding catheters. No LV lead dislodgement has been observed during an average follow-up of 6.9 months (0.5 months to 11.5 months).

Conclusions: Active fixation of this thin LV lead offers greater flexibility to place the lead precisely and stable in targeted vein segments over a wide range of different vein anatomies. The lead remained stable and no long-term dislodgements have been observed.

PO05-06 - Strong association between CAV1/CAV2 loci and P wave duration in patients with early onset lone atrial fibrillation

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Introduction: Recent studies have shown that not only PR-interval prolongation but also shortening is associated with atrial fibrillation (AF), while higher P wave duration (PWD) contribution to the PR-interval (P/PR ratio) is associated with mortality. Previous studies suggest that some single nucleotide polymorphisms (SNP) exert effect on atrial electrophysiology obtainable from ECG. We aimed to asses if 8 SNPs previously associated with PR-interval duration or AF would also be associated with altered P/PR ratios and PWD in patients with lone AF.

Methods: Surface ECG during sinus rhythm was recorded in 172 unrelated individuals with early-onset (age < 50 y) paroxysmal lone AF (median age 33 [19 - 63], 164 men). The SNPs rs2200733 (PITX2), rs2106261 (ZFHX2), rs13376331 (KCNN3), rs11708996 (SCN5A), rs6800541 (SCN10A), rs3807898 (CAV1/CAV2), rs251253 (NKX2-5), rs11047543 (SOX5) were genotyped.

Results: Carriers of risk allele (in either homo- or heterozygous state) of the SNP near the gene CAV1/CAV2 had significantly shorter PR-interval and PWD, compared to those not carrying any risk allele (see the Table). SNPs located near SCN10A, CAV1/CAV2 and SOX5, were associated with altered P/PR ratio.

No association between risk allele carrying and either PWD or P/PR was observed in regard to the 5 other SNPs.

Conclusion: We report for the first time an association between the SNP near the cardiac developmental gene CAV1/CAV2 and PWD in patients with lone AF.

Lone AF risk allele carriers of the SNP near the sodium channel gene SCN10A had a lower PWD contribution to the PR interval, whereas SOX5 and CAV1/CAV2 risk allele carriers had a higher P/PR ratio.

SNP; nearby gene	Allele	PR-interval (ms)	P-value	P-wave duration (ms)	P-value	P/PR ratio	P-value
rs6800541; *SCN10A*	risk-allele	162 ± 27	ns	124 ± 17	ns	0.77	0.023
	non-risk allele	158 ± 25		126 ± 14		0.81	
rs3807989; *CAV1/CAV2*	risk-allele	151 ± 25	<0.001	121 ± 16	0.003	0.81	0.019
	non-risk allele	167 ± 25		128 ± 15		0.78	
rs11047543; *SOX5*	risk-allele	152 ± 27	ns	125 ± 21	ns	0.82	0.04
	non-risk allele	161 ± 26		125 ± 15		0.79	

PO05-101 - Increase of power could enhance effectiveness of radiofrequency ablation at the same force-time integral level

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Introduction: The critical role of force-time integral (FTI) for effective lesion creation in radiofrequency (RF) ablation has been proven by several studies. However, the interaction between power and FTI has not been clearly elucidated.

Methods: 381 ablation points were acquired from 20 patients with paroxysmal atrial fibrillation who underwent pulmonary vein isolation for the first time. The points (ablation 60s) were collected at the beginning of the procedure at separate sites to avoid the mutual affect. A Thermocool SmartTouch catheter was used for RF ablation and all data were recorded on a Carto 3 system. Points were grouped by power (25W, n=124; 30W, n=125 and 35W, n=132). Impedance drop (ID) was used as a surrogate for measure of ablation effect. ID ≥10Ω was regarded as an adequate lesion formation. The real-time ID under various FTI (200-1000g·s) were recorded.

Results: ID rose either with FTI increase under a constant power setting or with power increase under a certain FTI level until FTI crossed 600g·s (Table). FTI required to reach the ID of 10Ω under 25W, 30W and 35W were 140 (76-276), 105 (72-188) and 68g·s (46-118), respectively. The corresponding ablation times were 12 (8-28), 10 (7-15) and 9s (6-12). Under power of 35W, less FTI (P<0.01) and shorter ablation time (P<0.01) were needed to reach the ID of 10Ω compared with both 25W and 30W. There was no statistical difference between groups of 25W and 30W.

Conclusions: Increase of power could enhance the ablation effect at the same FTI level. This effect might not be strengthened after FTI passed 600 g·s. Reinforcing power to 35W could decrease the minimal requirement of FTI to obtain an adequate ablation lesion.

Impedance drop (Ω) under different force-time integral levels and power settings

FTI (g·s)	200	300	400	600	800	1000
25W	8.9±4.4	10.2±4.8	11.1±5.1	13.6±6.9	16.6±7.0	17.5±6.0
30W	11.3±5.6*	13.1±6.8*	15.1±7.1*	16.9±8.3**	18.9±8.7	17.7±6.6
35W	14.5±7.8*†	15.7±7.7*†	17.1±8.4*	18.2±7.6*	20.5±9.0	23.1±11.4

** P<0.01, ** P<0.05 compared to 25W; † P<0.01 compared to 30W. Impedance drop rose with FTI increase under a settled power until FTI crossed 600 g·s(P<0.01). FTI: Force-Time Integral.*

PO03-174 - Heart Rate During Maximal Exercise Testing in Patients with Permanent Atrial Fibrillation

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Introduction: Many patients with permanent atrial fibrillation (AF) experience impaired exercise capacity. Several studies have shown that patients with AF seem to have an early and often excessive heart rate (HR) response to minor exercise. The aim of this study was to investigate the relation between HR response to exercise and exercise capacity (peak VO_2) in patients with permanent AF, with and without rate-reducing drug treatment.

Methods: Sixty patients (mean age 71±9 years, 18 women) with permanent AF and normal left ventricular function were included in the study. After a two-week wash-out period of any rate-reducing drugs, the patients underwent a maximal cardiopulmonary exercise test on a bicycle ergometer. Thereafter, all patients received diltiazem 360 mg, verapamil 240 mg, metoprolol 100 mg and carvedilol 25 mg once daily for three weeks, in a randomized sequence. Exercise tests were repeated on the last day of each treatment period. HR was manually counted as the mean of 20 sec print-outs every 2 minutes during exercise and recovery.

Results: Treatment with all four rate-reducing drugs lowered HR both at rest and during all stages of exercise and recovery, compared to baseline (p<0.001 for all) In multivariate regression analysis, adjusting for age, gender, BMI, ejection fraction and FEV_1, peak VO_2 was positively correlated to the heart rate reserve (HRR, r=0.40, p<0.001) and inversely correlated to the relative increase in HR during the four minutes warm-up phase (r=-0.22, p<0.001).

Conclusion: Preserved HRR correlated to better exercise capacity while excessive HR response to minor exercise was predictive of reduced exercise capacity in patients with permanent AF.

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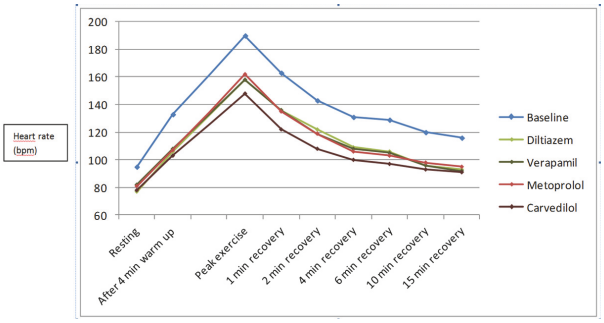

PO05-150 - Physical and psychological consequences of left cardiac sympathetic denervation in inherited heart diseases

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Introduction: Left cardiac sympathetic denervation (LCSD) is a permanent intervention reducing risk of life threatening cardiac events in long QT syndrome (LQTS) and catecholaminergic polymorphic ventricular tachycardia (CPVT). Sympathectomy is known to produce side effects, but these have not been systematically analysed in the genetic heart disease population.

Methods: After counselling by the senior author and surgeon, patients with LQTS (40) and CPVT (7) underwent video-assisted thoracoscopic LCSD. Median follow-up was 29 months (1 month-5.5 years). Clinical records were reviewed, and 44 completed a telephone survey assessing physical and psychological side effects.

Results: 25/47 patients (53 %) were symptomatic pre-operatively (syncope 15, near drowning 7, resuscitated sudden death 3). There was no peri-operative mortality. QTc did not change (461±60ms versus 476±54ms post-operatively (P=0.49). Principle indications for LCSD were beta-blocker intolerance (15, 32 %) or non-adherence (10, 21 %), disease related factors (18, 38 %; CPVT (6), aggressive presentation (near drowning (2), syncope during race (1), symptoms on therapy (2), LQT3 (1) or QTc>520ms (6)). The remainder (4, 9 %) chose to proceed due to high level sports participation (2), family history of sudden death (1) or other (1). Morbidity was reported by 42 of 44 (95 %). 29 (66 %) complained of dry skin on the left side of the body, 26 (59 %) a Harlequin type facial flush, 24 (55 %) contralateral hyperhidrosis, 17 (39 %) differential hand temperatures, 5 (11 %) permanent ptosis (4 (9 %) also had transient ptosis). 5 (11 %) have thermoregulation difficulties, 4 (9 %) have a sensation of left arm paraesthesia and 3 (7 %) reported a loss of sympathetic flight/fright response. This contrasts with post-operative satisfaction: 35 (80 %) felt positive about the procedure, 33 (75 %) safer, 38 (86 %) were happy it happened and 40 (91 %) would recommend the procedure to a similarly affected person. 40 (91 %) patients were happy with their surgical scar.

Conclusions: Despite significant morbidity resulting from LCSD, patients with LQTS and CVPT have high levels of post-operative satisfaction.