ReGenHeart Kick-off Meeting
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NOGA MAPPING

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NOGA MAPPING

The left ventricle was mapped with
- approximately 100 points to create maps of
- myocardial viability (voltage map) and
- contractility (local linear shortening map [LLS map])

On a unipolar voltage map, local activity
- >15mV is considered normal and viable myocardium,
- 5–15mV as an infarct border zone, and
- <5mV as a non-viable infarcted scar

LLS measures the local contractility of myocardium.
- LLS >6% is considered a normal contraction,
- LLS <6% a reduced contraction, and
- LLS <2% a paradoxical contraction

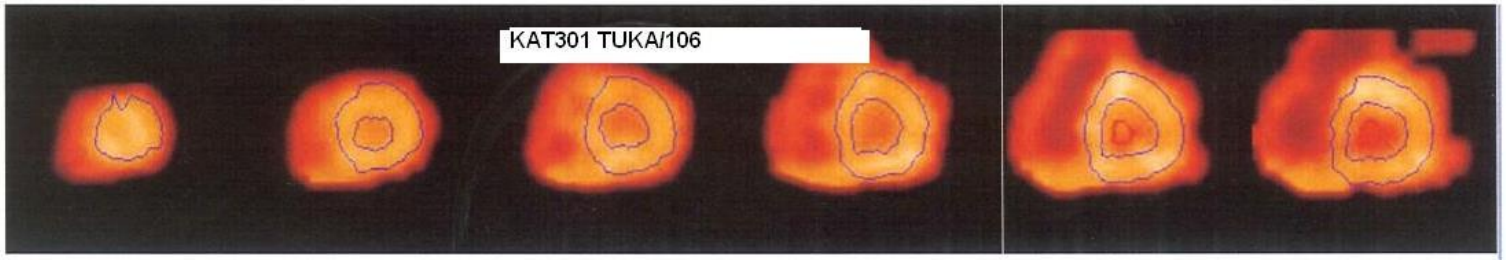
NOGA maps displayed as
- three-dimensional colormaps of the left ventricle
- two- dimensional bull's-eye maps with 17 segments identical to PET

Fill Threshold set as 8-15 ?

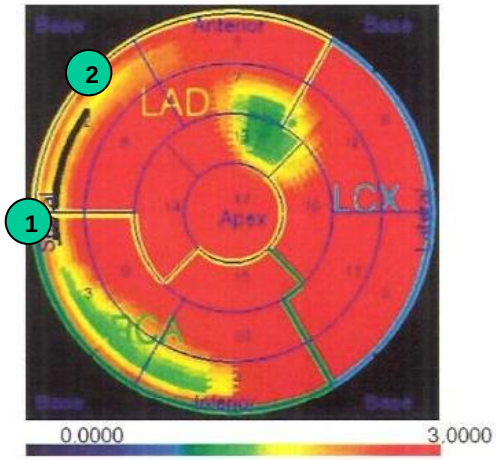
KAT301-TUKA 106

Screening PET

STRESS PET

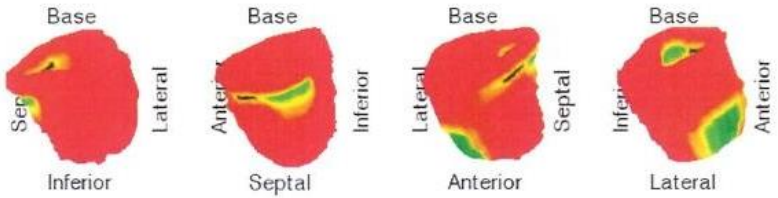


Patient name:
Patient ID:
Patient gender:
Study description: d4660 d4661
Institute: TURKU PET CENTRE
Isotope: 15O
Radiopharmaceutical: H2O -- water
Data file: Ø61973.PTDC.1
Input function: Image_based
Modeling method water
Frame limits

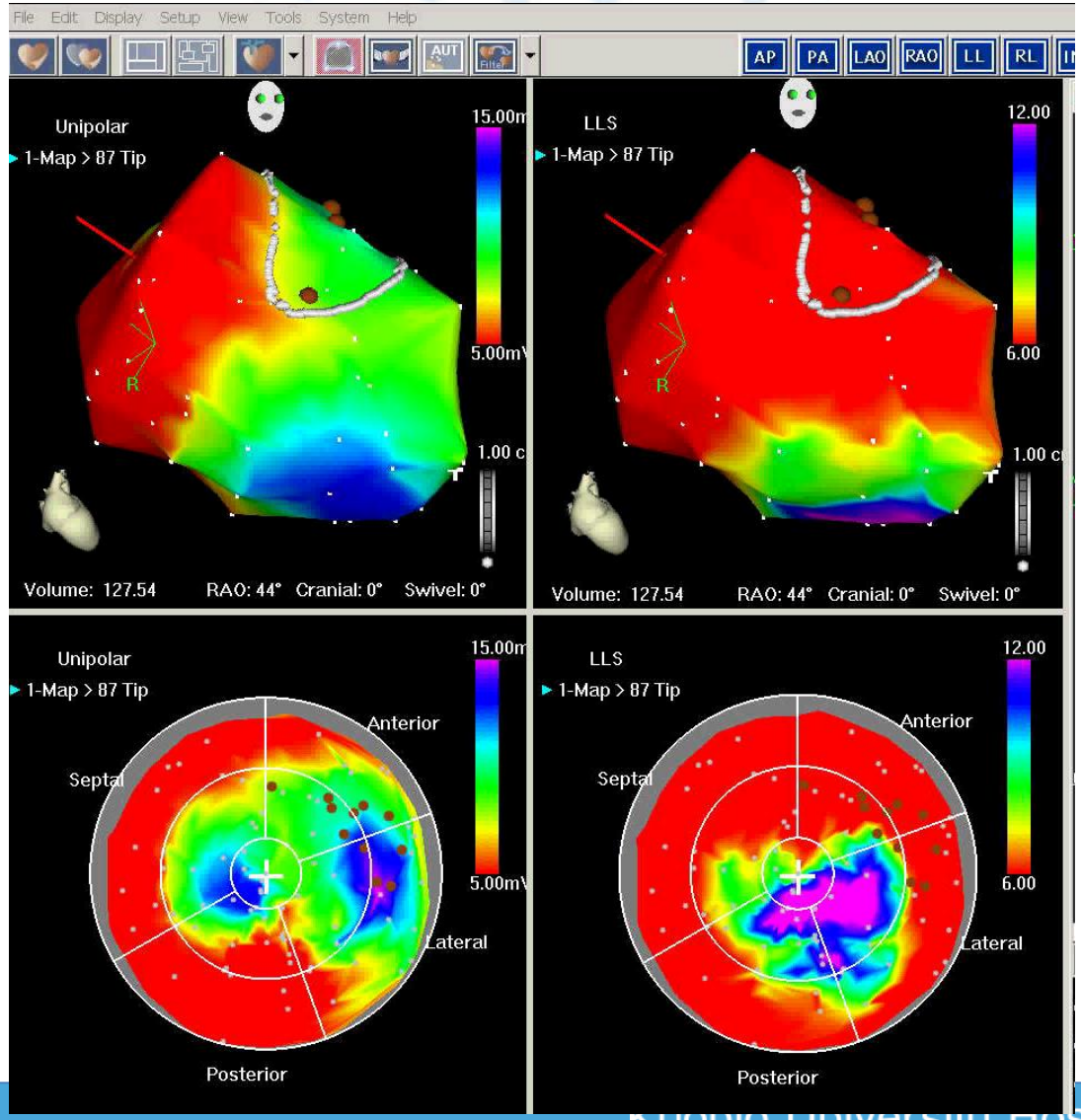


	Flow ml/g/min	Va	PTF
GLOBAL	3.8221	0.5332	0.3647
LAD	3.9201	0.5391	0.3933
RCA	3.3551	0.5084	0.3698
LCX	3.8612	0.5408	0.3048
LADwa	3.1094	0.4475	0.4949
APEX	4.7678	0.6584	0.2672
Seg 1	3.2285	0.6177	0.3153
Seg 2	99.9600	0.6152	0.2408
Seg 3	1.5292	0.3796	0.5021
Seg 4	2.1774	0.4456	0.3374
Seg 5	4.1278	0.5420	0.3516
Seg 6	4.1393	0.5474	0.2766
Seg 7	2.1034	0.5066	0.3852
Seg 8	4.3171	0.4977	0.5306
Seg 9	2.9344	0.4934	0.4796
Seg 10	3.4251	0.6067	0.2346
Seg 11	5.1273	0.6632	0.2413
Seg 12	3.1541	0.5231	0.3128
Seg 13	2.3876	0.4011	0.4344
Seg 14	4.3334	0.4811	0.5543
Seg 15	5.5924	0.6872	0.2491
Seg 16	3.2111	0.4872	0.3163
Seg 17	4.7678	0.6584	0.2672

Generated by CarimasTurku
Fri Feb 4 17:26:06 2011



Electromechanical assessment of Viability (mV) and Wallmotion (LLS)

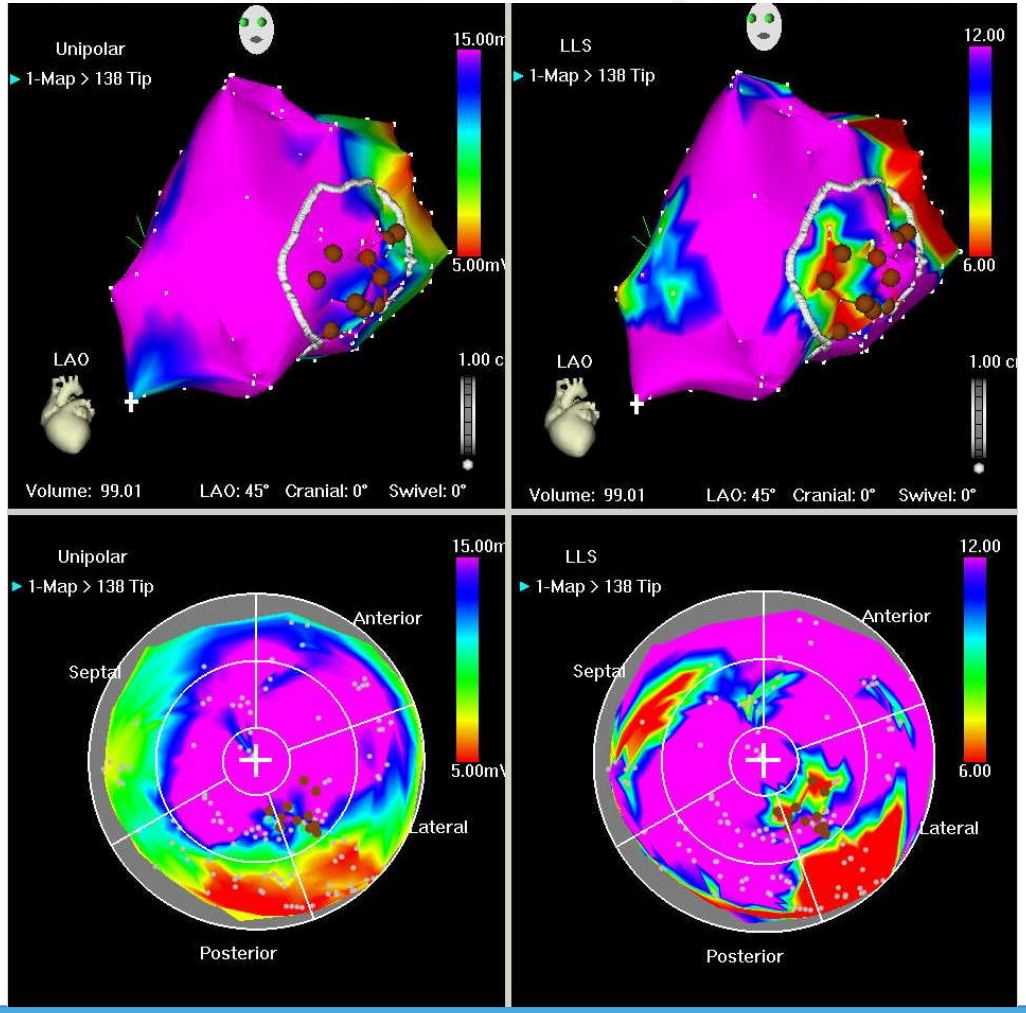


Red areas in the unipolar voltage map show SCAR areas

Red areas in the LLS map show “low wall movement”

Injections aim for the VIABLE, but HYPOKINETIK areas.

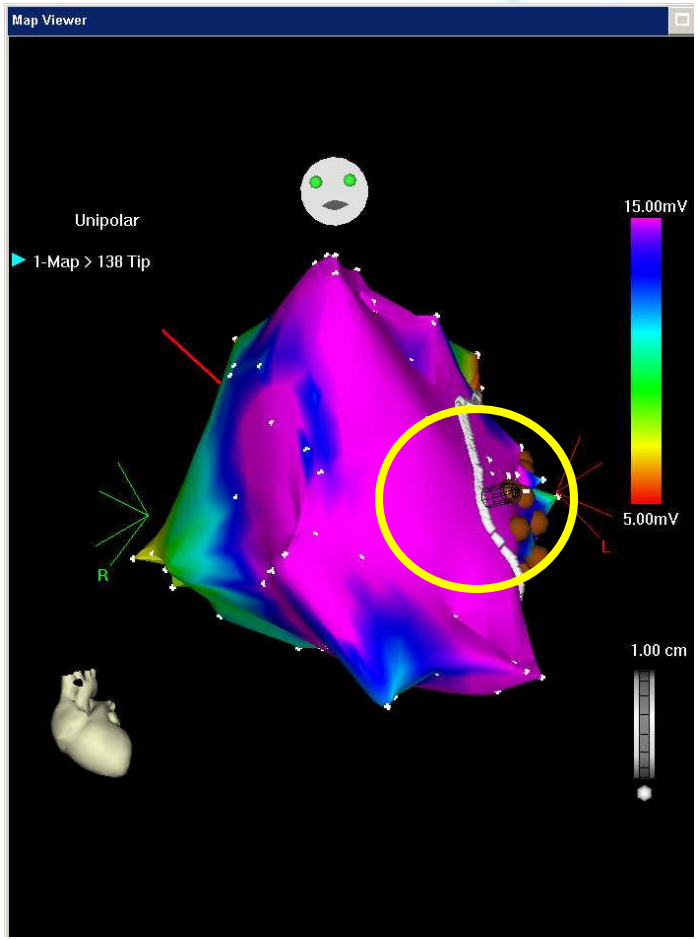
Refractory Angina

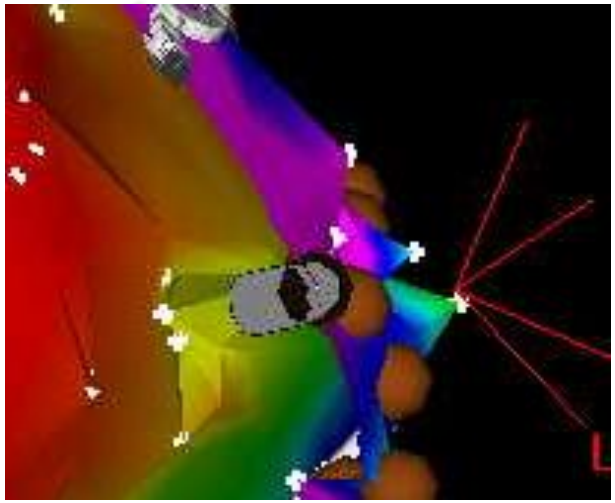


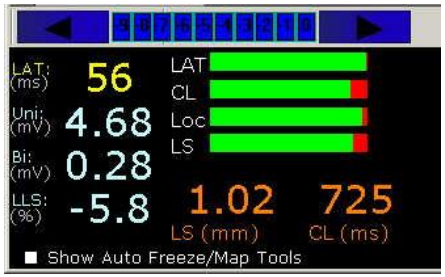
Please note the high viability (mV) in the unipolar map, and the low wall motion (red) in the LLS map

= hibernating tissue

Controlled injection conditions







Perpendicular catheter orientation, and stability criteria (catheter versus wall) allow a reproducible and controlled intramyocardial injection

Intramyocardial Gene Therapy Directed to Hibernating Heart Muscle Using a Combination of Electromechanical Mapping and Positron Emission Tomography

Iiro Hassinen,[1] Antti Kivelä,[1] Antti Hedman,[1] Antti Saraste,[2] Juhani Knuuti,[2] Juha Hartikainen,[1,3] and Seppo Ylä-Herttuala[1,4,5,*]

[1]Heart Center and [5]Science Service Center and Gene Therapy Unit, Kuopio University Hospital, Kuopio, Finland; [2]Turku PET Centre, Turku University Hospital, Turku, Finland; and [3]Institute of Clinical Medicine and [4]A.I. Virtanen Institute for Molecular Sciences, University of Eastern Finland, Kuopio, Finland.

Cardiac gene transfer for the treatment of ischemic diseases has suffered from low gene transfer efficiency and inability to target treatment genes to the ischemic myocardium. A combined method has been developed based on electromechanical mapping and radiowater PET imaging to target gene therapy to viable but ischemic and hibernating areas of the myocardium. Electromechanical NOGA mapping produces three-dimensional images of myocardium with both an electric activity map and a myocardial contractility map. These have been converted to 17-segment 2D bull's-eye maps, which were superimposed onto PET radiowater perfusion imaging maps of the myocardium. This technique was applied in a Phase I/IIa clinical trial to target gene therapy for refractory angina patients. It was found that by combining electromechanical map with PET imaging, targeting of gene therapy to hibernating ischemic myocardium can be significantly improved. Here, the methods for the identification of viable, ischemic, and hibernating myocardium for gene transfer are described, and examples of treated refractory angina patients who have benefited from the improved gene transfer method to the ischemic myocardium are presented.

Hassinen et al. Human Gene Therapy 2017