1268 | BENCH
A batteryless cardiac pacemaker powered by cardiac motion

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Introduction: The battery is one of the most limiting factors of modern pacemaker designs. Battery replacements require repeated surgical interventions with associated morbidity and costs. Alternative power sources not relying on stored energy become essential to improve a patient's quality of life.
Harvesting cardiac energy might provide a continuous energy source to power batteryless pacemakers. A possible mechanism to convert cardiac motion into electrical energy can be found in the automatic wristwatch: the movement of a person's wrist accelerates an eccentric mass in the clock housing. An integrated generator converts the rotation into electrical energy.
Methods: A harvesting device was derived from the clockwork of an automatic wristwatch. The harvester was then anchored on the heart thanks to a custom-made housing (total mass 16.7 g, figure). For an in-vivo study with a 60 kg domestic pic, a sternotomy was performed to suture the prototype onto the anteroapical part of the left ventricle. The harvesting device was connected to a custom-built single-chamber pacemaker. Finally, an epicardial bipolar pacing wire was used to deliver the pacemaker stimulus.
Results: The energy harvesting device supplied the pacemaker with enough energy to perform continuous VVI pacing (pacing threshold 1.0 V/0.5 ms, sensing 9.8 mV, impedance 1279 Ω) at 130 bpm (pacing output 1.6 V/0.8 ms). Simultaneously, the harvesting device generated a mean output power of 52 μW over an additional load resistor of 1 kΩ.

Conclusion: We demonstrated the feasibility of pacing the heart using its own mechanical activity. The harvested energy exceeded the power requirement of a modern pacemaker (~10 μW). Furthermore, we expect to increase the performance by optimising this first-generation prototype.

Poster Session 2

LIPOPROTEINS IN CARDIOVASCULAR PREVENTION

P1270 | BEDSIDE
Triglycerides are independent risk predictors in stable coronary artery disease

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Objective: Risk prediction with fasting compared to postprandial serum triglycerides (TG) in patients with cardiovascular disease remains unclear, especially in patients who are treated with a statin. The aim of this prospective study was to analyze the role of fasting and postprandial serum triglycerides (TG) as risk modifiers in patients with coronary artery disease (CAD).
Methods: A sequential oral triglyceride (OTT, 75g cream fat) and glucose tolerance test (OGT, 75g glucose) was applied to obtain standardized measurements of postprandial TG glucose kinetics in patients with stable CAD confirmed by angiography. Lipid and glucose parameters were measured at fasting, 3, 4, and 5 hours after the OTT/OGT. The primary outcome was the composite endpoint of death and hospitalization for acute coronary syndrome or hospitalization for unplanned, symptom-induced coronary angiography and revascularization within 48 months follow-up.
Results: N=514 patients with angiographically confirmed, clinically stable CAD were enrolled. Median age was 68 years, 83% were male, 95% were treated with statins and median LDL-cholesterol concentration was 105mg/dl.
Fasting TG were strongly associated with the area under the curve (AUC) of the postprandial TG increase (R=0.93, p<0.0001). Patients were stratified by TG tertiles for statistical comparisons of baseline variables and time-to-event analyses. Compared to the lowest tertile of fasting TG (<106mg/dl), patients in the highest tertile (>150mg/dl) were younger, more obese, more were smoking, they had a higher blood pressure and pulse, lower HDL- and higher LDL-cholesterol and more had glucose intolerance or a metabolic syndrome. A similar association was observed with the AUC tertiles.
Follow-up at was 100% complete. Both fasting and postprandial TG predicted the primary outcome (fasting TG >150mg/dl vs. <106mgdl: HR 1.79, 95%-CI 1.31-2.45, p=0.0001; AUC >1120mg/dl vs. <750mg/dl: HR 1.78, 95%-CI 1.29-2.45, p=0.0003). Postprandial TG parameters did not improve risk prediction compared to fasting TG. The number of cardiovascular deaths and myocardial infarctions was higher in the upper tertile of fasting TG >150mg/dl (HR 1.79, 95%-CI 1.04-3.09, p=0.03). Risk prediction by TG was independent of traditional risk factors, medication, glucose metabolism, LDL- and HDL-cholesterol.
Conclusions: Fasting serum triglycerides >150 mg/dl independently predict cardiovascular events in patients with coronary artery disease on guideline-recommended medication. Assessment of postprandial TG does not improve risk prediction compared to fasting TG in these patients.

P1271 | BEDSIDE
Impact of statin therapy on low-density lipoprotein cholesterol and triglyceride levels in patients with hypertriglyceridaemia: a VOYAGER meta-analysis

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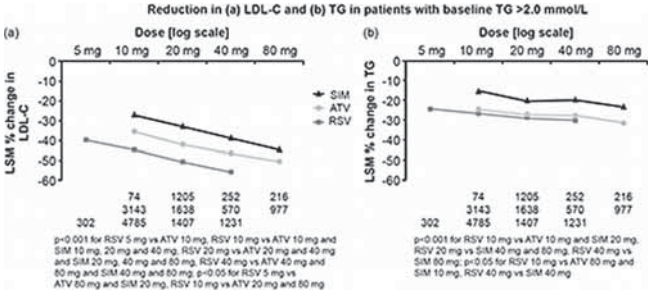
Background: Several studies have indicated a causal role for triglycerides (TG) in the development of coronary artery disease. Recently published guidelines from the European Atherosclerosis Society (EAS) Consensus Panel define mild-to-moderate hypertriglyceridaemia as a TG level of 2.0–10.0 mmol/L and recommend a TG level <1.7 mmol/L as desirable; however, the primary treatment goal in these patients remains the LDL-C goal. Individual patient data (n=32,258) from the VOYAGER meta-analysis of 37 studies were used to analyse LDL-C and TG reduction in patients with baseline TG >2.0 mmol/L.
Methods: The least-squares mean (LSM) % change from baseline in LDL-C and TG was compared during 15,800 patient exposures to rosuvastatin (RSV) 5, 10, 20 and 40 mg, atorvastatin (ATV) 10, 20, 40 and 80 mg, and simvastatin (SIM) 10, 20, 40 and 80 mg in patients with baseline TG >2 mmol/L. Comparisons were made using a single mixed-effects model using only data from studies directly comparing treatments by randomised design.
Results: LSM % reductions in LDL-C and TG are shown in the figure. Reductions in LDL-C with RSV 10–40 mg doses were significantly greater than for equal or double doses of ATV and SIM (all p<0.05). RSV 10 mg produced a significantly greater (p<0.05) reduction in TG than ATV 10 mg, but reductions with RSV 20 and 40 mg were similar to equal doses of ATV. Doses of RSV 10–40 mg resulted in significantly greater (p<0.05) reductions in TG than equal or double doses of SIM.



Conclusions: In patients with hypertriglyceridemia, LDL-C reduction, the primary treatment goal, was substantial and dependent on the choice and dose of statin. The reduction in TG was numerically less than for LDL-C, and additional therapy may be considered to potentially further reduce residual cardiovascular risk.

P1272 | BEDSIDE
Association of high serum apolipoprotein B-48 Level with increased proteinuria and reduced estimated glomerular filtration rate (eGFR)

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Postprandial hypertriglyceridemia (PHTG) is one of residual risk factors for coronary heart disease (CHD) and caused by the impaired clearance of chylomicron remnants (CM-R), which directly influence the progression of atherosclerotic plaque. We developed ELISA and CLEIA system for measuring serum apolipoprotein (apo) B-48 for evaluating the accumulation of CM-R. In our former study, we found that high fasting apoB-48 level significantly correlated with the prevalence of CHD. Proteinuria and reduced estimated glomerular filtration rate (eGFR) are independent risk factors for cardiovascular events and renal dysfunction. In the present study, we have investigated whether serum apoB-48 levels are associated with renal dysfunction.