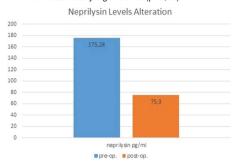
was 25,70±3,89 kg/m²) were included the study. 42.6% (20) of the patients had ischemic etiology. Neprilysin levels significantly decreased from 175,28pg/ml to 75,30 pg/ml 3 months after LVAD implantation (p:0,007) Seven patients who had died after LVAD implantation has elevated neprilysin levels but weren't the statistically significant cause of limited mortality event (1798,51 pg/ml, 371,59 pg/ml p=0,134). Neprilysin levels were unrelated with age, gender, body mass index, renal function and etiology of heart failure. A cut-off value of 431 pg/ml was found to be predictive of adverse events within 3 months after the implantation with 71% sensitivity and 77% specificity (EAA: 0,682; %95 CI: 0.387–0.977; p=0.128). Patients with LVAD complication in first three months had liminal higher neprilysin levels and statistically significance (p=0.05).



Neprilysin Levels Alteration

Conclusion: Although neurohormonal system activation decreases after LVAD implantation, it never returns to normal. Those patients who had complications within 3 months after the operation still had high levels of neurohormonal activation. For this reason, it is recommended to continue optimal medical therapy in patients who still have high levels of neprilysin after LVAD implantation.

P5119 Differences in VWF activity in von willebrand disease type 2A patients versus LVAD patients with the acquired von willebrand syndrome

S. Deconinck¹, C. Terseeg¹, E. Bailleul², L. Delrue¹, N. Vandeputte¹, H. Deckmyn¹, S. De Meyer¹, K. Vanhoorelbeke¹, M. Vanderheyden². ¹KU Leuven, KULAK, Laboratory for Thrombosis Research, IRF Life Sciences, Kortrijk, Belgium; ²OLV Hospital Aalst, Cardiovascular Center, Aalst, Belgium

Background: Patients suffering from von Willebrand disease (VWD) type 2A are diagnosed by an increased bleeding diathesis due to an impaired von Willebrand factor (VWF) function with severe loss in high molecular weight (HMW) VWF multimers. Patients with implanted left ventricular assist devices (LVAD) show a moderate decrease in HMW VWF multimers and are therefore nowadays diagnosed with the acquired von Willebrand syndrome (aVWS). However, only a small portion of LVAD patients suffers from bleeding complications.

Aim: Side by side comparison of VWF function in patients with VWD type 2A and LVAD-induced aVWS to demonstrate the difference in VWF activity, and hence their different effect on bleeding, in both groups of patients.

Methods: Plasma samples from 9 known VWD type 2A and from 9 LVAD patients were analyzed for VWF:Ag, VWF:CB and VWF:RCo using ELISA and for VWF multimers using sodium dodecyl sulphate (SDS) agarose gel electrophoresis and compared to plasma of healthy donors.

Results: As expected, VWF function was impaired in all VWD type 2A patients with a severe reduction in HMW VWF multimers compared to healthy individuals (0.0% (0.0−12.3) versus 34.2% (31.7−38.9) respectively, p<0.0001) ensuing in decreased (<0.7) VWF:CB/VWF:Ag and VWF:RCo/VWF:Ag ratios. In contrast, VWF function was less affected in LVAD patients where only a moderate reduction in HMW VWF multimers was noted (20.3% (15.8−21.7)) with six out of 9 LVAD patients having a VWF:CB/VWF:Ag or VWF:RCo/VWF:Ag ratio within normal range (≥0.7).

Conclusions: Whereas the decrease in HMW VWF multimers together with the depressed VWF function are responsible for the severe bleeding disorder observed in VWD type 2A pts, HMW VWF multimers and hence VWF function are only moderately impaired in LVAD-induced aVWS patients,. Accordingly, these small defects in VWF function can not alone be responsible for the bleeding diathesis in LVAD pts which might explain why only a small portion of them suffer from bleeding complications.

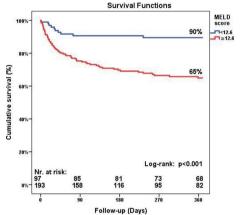
P5120

Pre-operative liver dysfunction is associated with higher mortality rates at 1-year after left ventricular assist device implantation

R. Muslem¹, Y. Yalcin¹, J. Brugts¹, R.J. Tedford², G. Whitman³, A.J.J. Bogers¹, S. Hsu², K. Caliskan¹. ¹Erasmus Medical Center, Cardiology, Rotterdam, Netherlands; ²Johns Hopkins University of Baltimore, Cardiology, Baltimore, United States of America; ³Johns Hopkins University of Baltimore, Cardiothoracic Surgery, Baltimore, United States of America

Purpose: In the current study, we determined the incidence and impact of preoperative liver dysfunction on mortality. Methods: A retrospective multi-centre cohort study was conducted, including all patients undergoing LVAD implantation (HeartMate II n=225 (78%), HeartMate 3 n=43 (15%) and HeartWare n=22 (7%). The definition of model for end-stage liver disease (MELD) score proposed the United Network for Organ Sharing was used to define liver dysfunction. Optimal cut-off value for MELD score was determined by receiver operating characteristics curve analysis, subsequently liver dysfunction was defined as a MELD score of ≥12.6.

Results: Overall, 290 patients (mean age 55 [44–62], 76% male) were included, of whom 193 (67%) patients had pre-operative liver dysfunction. One-year survival rate in patients with and without liver dysfunction was 65%, and 90%, respectively (log-rank p≤0.001, Figure). After multivariable adjustment, age (HR 1.03 95% CI 1.01–1.05, p=0.002), body mass index (HR 1.02 95% CI 1.00–1.04, p=0.021), INTERMACS class 1 (HR 6.64 95% CI 2.25–17.50 p≤0.001), INTERMACS class 2 (HR 3.40 95% CI 1.30–8.88 P=0.013) and MELD score of ≥12.6 (HR 3.71 95% CI 1.76–7.85, p=0.001) were independent predictors of mortality at 1 year after implantation. Baseline total bilirubin improved significantly compared with 6 months and 12 months after LVAD implantation (p<0.001, p=0.005), respectively.



Survival by MELD score

Conclusion: In this large multi-centre cohort study, we found that pre-operative liver dysfunction is associated with higher mortality rates at 1-year after LVAD implantation. Though, during the first year after LVAD implantation a significant improvement in the liver function was frequently observed.

P5121

Invasive exercise haemodynamics predict functional capacity in patients with advanced heart failure implanted with a left ventricular assist device

A. Koshy¹, T. Green², O.G. Fernandez², S. Schueler², D. Jakovljevic¹, G. MacGowan². ¹Newcastle University, Newcastle upon Tyne, United Kingdom; ²Freeman Hospital, Newcastle upon Tyne, United Kingdom

Background: Left ventricular assist devices (LVADs) have been used as an effective therapeutic option to improve survival, quality of life and functional capacity in patients with advanced heart failure. Yet understanding physiological mechanisms and haemodynamic responses to a LVAD therapy warrant further investigations. Purpose: The present study i) evaluated haemodynamic response to physiological stress in LVAD and advanced heart failure patients; and ii) assessed the relationship between heamodynamic response and functional capacity.

Methods: A prospective, observational, single centre study included patients implanted with a continuous flow centrifugal LVAD (N=8, age 39.5±12.8 years, 1 female) and those with advanced chronic heart failure (CHF) receiving optimal medical therapy (N=11, age 54.1±9.8 years, 2 females). All patients underwent right heart catheterisation with thermodilution cardiac output assessment at rest and in response to active leg raise to exhaustion (stress). The Borg Scale (1–10) was used to assess rate of perceived exertion. Three hours later, patients completed maximal graded cardiopulmonary exercise stress test using cycle ergometer.

Results: All patients completed the study protocol. LVAD and CHF patients achieved exhaustion with active leg raise test within 193±62 vs 156±53 seconds, p=0.19, and the Borg Scale score of 5.9±2.2 vs 6.0±3.2, p=0.94. Haemodynamic response from rest to peak (Δ) active leg raise test included i) in LVAD patients increase mean pulmonary artery pressure by 47.7% (28.1±14.2 to 41.5±10.3, p<0.01), pulmonary artery wedge pressure by 58.8% (19.4±9.9 to 30.8±8.4, p<0.01), cardiac index by 8% (2.5±0.5 to 2.7±0.5 L/min/m², p=0.19), heart rate by 34.1% (69.0±11.4 to 92.5±15.3 beats/minute in p<0.01), and a decrease in stroke volume by 18.4% (71.0±17.4 to 57.9±12.9 mL, p<0.04), ii) in CHF patients increase in mean pulmonary artery pressure by 41.0% (24.9±13.0 to 35.1±12.7, p<0.01), and pulmonary artery wedge pressure by 64.2% (16.2±9.2 to 26.6±9.5 p<0.01), cardiac index by 10% (2.0±0.5 to 2.2±0.6 L/min/m², p<0.04), heart rate by 32.9% (72.6±9.0 to 96.5±15.0 beats/minute, p<0.01), and a decrease in stroke volume by 18.1% (55.0±17.1 to 44.6±21.1 mL, p<0.04) There were no significant