Conclusion These results indicate that in principle \( V_{2\max} \) in ml/kg/min as an indirect indicator of cardiac function or for cardiac transplantation selection is unreliable when applied to overweight heart failure patients. Extending this concept to the entire spectrum of body weights, the practice of correcting \( V_{2\max} \) by body weight in cardiological practice would also require urgent reconsideration.

**Background** Non-invasive blood pressure monitoring by continuous finger photoplethysmography (Finometer) may have value in pacemaker optimisation. However, the immediate increment in blood pressure seems to diminish somewhat in the initial minute: it is unclear whether this is due to an (undesirable) fall in stroke volume or a (desirable) compensatory reduction in peripheral resistance.

**Methods and Results** We studied this question by measuring beat-by-beat stroke volume (flow) using Doppler echocardiography, and blood pressure using continuous finger photoplethysmography, during and after atrioventricular delay adjustment from 40 to 120 ms in 19 subjects with cardiac pacemakers. Quintuplicate experimental runs were performed. Blood pressure and stroke volume (flow) both increased immediately (\( p<0.0001 \) within one heartbeat). The immediate pressure increment correlated strongly with the immediate flow increment (\( r=0.74, p=0.0001 \)). Pressure showed a partial decline a few seconds later (average rate 0.65 mm Hg/beat, \( r=-0.98, p<0.0001 \)), in contrast, flow did not decline (\( p=NS \), Abstract 100 figure 1). Signal-to-noise ratio was significantly better for pressure than flow (6.5±3.6 vs 2.1±1.4, \( p<0.0001 \)), Abstract 100 figure 2.

**Conclusions** Improving atrioventricular delay immediately increases blood pressure; however this effect decays slightly over the subsequent minute. This is due to compensatory vasodilatation rather than a reduction in cardiac function. Pressure changes are simpler to measure and easier to distinguish from random variation than Doppler measurements of flow, but are best measured immediately, before the vascular compensation.

**WHAT DEGREE OF PULMONARY HYPERTENSION PREDICTS POOR OUTCOME IN PATIENTS WITH LEFT VENTRICULAR SYSTOLIC DYSFUNCTION? A 10-YEAR FOLLOW-UP STUDY**

doi:10.1136/heartjnl-2011-300198.101

B R Szwejkowski, 1D H J Elder, 1A M J Choy, 2S D Pringle, 1A D Struthers, 1C C Lang, 1University of Dundee, Dundee, UK; 2Department of Cardiology, NHS Tayside, Dundee

**Introduction** The presence of pulmonary hypertension in left ventricular systolic dysfunction (LVSD) is an ominous sign. It remains unclear the level at which pulmonary hypertension conveys a mortality risk in patients with LVSD.

**Methods** We performed a record-linkage study in Tayside, UK (population approximately 400 000) utilising the Tayside echocardiogram database (>100 000 echo’s) maintained by the Health Informatics Centre (HIC). Datasets from HIC include mortality data and other health care activities linked anonymously by the community health index (CHI) number. Patients were included in the analysis if they had LVSD and had a right ventricular systolic pressure (RVSP) measurement. Cox proportional hazards regression analysis was used to examine the effects of different ranges of RVSP measures on all cause mortality.

**Results** 2910 patients (mean age, 74.5±11.4 years; 45 % male) met entry criteria. Mean RVSP was 45.3 ± 12.7 mm Hg and median follow was 362 days (IQR 129—850 days). There was a significant correlation between RVSP and survival (\( p<0.0001 \)). In quartiles of RVSP, the HR after adjustment for confounding factors including LVSD and the presence of chronic obstructive pulmonary disease (COPD) were: RVSP 35—41 mm Hg, HR 1.12 (95% CI 0.95 to 1.32, \( p=0.175 \), RVSP 42—50 mm Hg, 1.27 (1.07 to 1.49, \( p<0.001 \)) and RVSP 51—106 mm Hg 1.62 (1.38 to 1.1, \( p<0.001 \)). For each 5 mm Hg stepwise increase in RVSP the HR for all cause mortality was 1.07 (1.04 to 1.09, \( p<0.001 \)). Abstract 101 figure 1 shows the Kaplan-Meier survival curves for all cause mortality for all patients expressed as different RVSP quartiles.

**Conclusion** These results indicate that in principle \( V_{2\max} \) in ml/kg/min as an indirect indicator of cardiac function or for cardiac transplantation selection is unreliable when applied to overweight heart failure patients. Extending this concept to the entire spectrum of body weights, the practice of correcting \( V_{2\max} \) by body weight in cardiological practice would also require urgent reconsideration.

**Background** Non-invasive blood pressure monitoring by continuous finger photoplethysmography (Finometer) may have value in pacemaker optimisation. However, the immediate increment in blood pressure seems to diminish somewhat in the initial minute: it is unclear whether this is due to an (undesirable) fall in stroke volume or a (desirable) compensatory reduction in peripheral resistance.

**Methods and Results** We studied this question by measuring beat-by-beat stroke volume (flow) using Doppler echocardiography, and blood pressure using continuous finger photoplethysmography, during and after atrioventricular delay adjustment from 40 to 120 ms in 19 subjects with cardiac pacemakers. Quintuplicate experimental runs were performed. Blood pressure and stroke volume (flow) both increased immediately (\( p<0.0001 \) within one heartbeat). The immediate pressure increment correlated strongly with the immediate flow increment (\( r=0.74, p=0.0001 \)). Pressure showed a partial decline a few seconds later (average rate 0.65 mm Hg/beat, \( r=-0.98, p<0.0001 \)), in contrast, flow did not decline (\( p=NS \), Abstract 100 figure 1). Signal-to-noise ratio was significantly better for pressure than flow (6.5±3.6 vs 2.1±1.4, \( p<0.0001 \)), Abstract 100 figure 2.

**Conclusions** Improving atrioventricular delay immediately increases blood pressure; however this effect decays slightly over the subsequent minute. This is due to compensatory vasodilatation rather than a reduction in cardiac function. Pressure changes are simpler to measure and easier to distinguish from random variation than Doppler measurements of flow, but are best measured immediately, before the vascular compensation.

**WHAT DEGREE OF PULMONARY HYPERTENSION PREDICTS POOR OUTCOME IN PATIENTS WITH LEFT VENTRICULAR SYSTOLIC DYSFUNCTION? A 10-YEAR FOLLOW-UP STUDY**

doi:10.1136/heartjnl-2011-300198.101

B R Szwejkowski, 1D H J Elder, 1A M J Choy, 2S D Pringle, 1A D Struthers, 1C C Lang, 1University of Dundee, Dundee, UK; 2Department of Cardiology, NHS Tayside, Dundee

**Introduction** The presence of pulmonary hypertension in left ventricular systolic dysfunction (LVSD) is an ominous sign. It remains unclear the level at which pulmonary hypertension conveys a mortality risk in patients with LVSD.

**Methods** We performed a record-linkage study in Tayside, UK (population approximately 400 000) utilising the Tayside echocardiogram database (>100 000 echo’s) maintained by the Health Informatics Centre (HIC). Datasets from HIC include mortality data and other health care activities linked anonymously by the community health index (CHI) number. Patients were included in the analysis if they had LVSD and had a right ventricular systolic pressure (RVSP) measurement. Cox proportional hazards regression analysis was used to examine the effects of different ranges of RVSP measures on all cause mortality.

**Results** 2910 patients (mean age, 74.5±11.4 years; 45 % male) met entry criteria. Mean RVSP was 45.3 ± 12.7 mm Hg and median follow was 362 days (IQR 129—850 days). There was a significant correlation between RVSP and survival (\( p<0.0001 \)). In quartiles of RVSP, the HR after adjustment for confounding factors including LVSD and the presence of chronic obstructive pulmonary disease (COPD) were: RVSP 35—41 mm Hg, HR 1.12 (95% CI 0.95 to 1.32, \( p=0.175 \), RVSP 42—50 mm Hg, 1.27 (1.07 to 1.49, \( p<0.001 \)) and RVSP 51—106 mm Hg 1.62 (1.38 to 1.1, \( p<0.001 \)). For each 5 mm Hg stepwise increase in RVSP the HR for all cause mortality was 1.07 (1.04 to 1.09, \( p<0.001 \)). Abstract 101 figure 1 shows the Kaplan-Meier survival curves for all cause mortality for all patients expressed as different RVSP quartiles.