acute coronary syndrome, are associated with early atherosclerotic changes in patients with SLE.
Methods: The number of circulating CD4+CD28null cells was assessed using a validated method, as reported by our group previously, in 16 SLE patients (age 40±11 years; disease duration 11±5.8 years) without classical coronary risk factors and 15 age and gender matched normal volunteers (controls). In SLE patients, global damage index (SLICC/ACR score), disease activity and treatment-related parameters were assessed. In both patients and controls, carotid artery intima-media thickness (IMT) and systemic endothelial function (brachial artery flow mediated dilation (FMD)) were assessed.
Results: Total cholesterol levels were not significantly different in patients and controls (5.1±1.6 vs. 4.9±1.0 mmol/L, p=0.74). IMT was similar controls and patients (0.57±0.10 vs 0.54±0.11 mm, p=0.34). FMD, however, was significantly lower in SLE patients compared to controls (2.55±1.93 vs. 6.31±1.98, $p<0.0001$). Circulating CD4+CD28null cells were absent in the control group but present in 11 out of 16 patients. Of these, seven had persistent expansion of CD4+CD28null T cells constituting over 15% of the total CD4+ compartment. Patients with persistent expansion of CD4+ CD28null cells had a more pronounced decrease in brachial artery FMD compared to SLE patients, in whom the CD28 null population constituted <15% of the CD4+ T cells (1.38±1.9 vs 3.46±1.5, p=0.028). A significant positive correlation was found between number of CD4+CD28null cells and IMT values (r=0.86, $p<0.0001$) whereas there was a significant negative correlation between number of CD4+CD28null cells and FMD responses (r=-0.62, p=0.006) in SLE patients.
Conclusions: Circulating CD4+CD28null lymphocytes are increased in SLE patients, and these cells correlate with endothelial dysfunction and early atherosclerotic changes. Thus these T cells may contribute to CV disease in patients with SLE.

P741 Chronic renal failure is not associated with the prevalence and severity of coronary artery disease: analysis in 5641 consecutive patients

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Background: Chronic renal failure (CRF) is associated with an increased incidence of cardiovascular events. However, few data exist on the relation between CRF and the prevalence and severity of coronary artery disease (CAD).
Methods: 5641 consecutive patients undergoing coronary angiography for the evaluation of chest pain were analysed. Cardiovascular risk factors were assessed by standardised questionnaire and routine blood chemistry. Severity of CAD was graded by visual estimation of lumen diameter stenosis. Significant stenoses were defined as lumen diameter reduction ≥70% in at least one major coronary artery. Coronary angiograms were graded as non-significant CAD, as 1-, 2- or 3-vessel disease (VD) or as non-CAD. Renal function was assessed by estimation of the glomerular filtration rate (GFR) using the abbreviated Modification of Diet in Renal Disease Equation (MDRD2). The GFR was then corrected for body surface area (Dubois formula).
Results: Overall, the GFR was lower in CAD (n=4124) compared to non-CAD patients (n=1517) (68.7±19.7 vs 72.8±20.0 ml/min; $p<0.001$), but was not different between 1-VD, 2-VD, 3-VD and non-significant CAD. CAD patients had lower HDL levels (51.9±15.3 vs 60.3±18.5mg/dl), were older (65.2±10.5 vs 59.9±11.4y), more often smokers (18.7 vs 16.5%), diabetics (19.9 vs 10.8%) and hypertensives (85.6 vs 69.6%) (all $p<0.005$), had similar LDL levels (124.5±38.3 vs 126.0±36.3mg/dl; p=NS) and were more frequently on chronic statin therapy (43.4 vs 27.9%; $p<0.001$). However, in multinomial logistic regression analysis (table) GFR was not independently associated with the presence and severity of CAD.

Table 1. Multinomial logistic regression analysis: CAD vs non-CAD

	Odds ratio	95%CI	Wald	p-value
Age	1.061	(1.053-1.068)	259.116	p<0.001
Gender	2.728	(2.352-3.165)	175.636	p<0.001
HDL	0.977	(0.973-0-982)	103.296	p<0.001
Hypertension	1.657	(0.795-1.434)	30.187	p<0.001
Diabetes	1.665	(1.355-2.047)	23.439	p<0.001
Smoking habit	1.795	(1.459-2.210)	30.526	p<0.001
GFR	1.001	(1.005-1.099)	0.512	p=NS

Conclusion: In this large consecutive patient cohort, CRF is not independently correlated with the angiographically documented prevalence and severity of CAD.

ECHO IN VALVULAR HEART DISEASE

P742 Echocardiographic assessment of prevalence, severity, progression and risk factors for paravalvular regurgitation (PVR) after heart valve replacement from the AVERT study

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Objective: To determine prevalence, severity, progression and risk factors for PVR by echocardiography after heart valve replacement in the Artificial Valve Endocarditis Trial (AVERT).
Methods: Patients with aortic (AVR, n=221) &/or mitral (MVR, n=159) valve replacement had serial echocardiograms over a 7-year period. Large, asymmetric, or eccentric jets that originated outside of the sewing ring were considered indicative of PVR. A qualitative assessment defined the presence or absence of PVR, and a semiquantitative assessment defined the severity of PVR.
Results: A total of 1409 echocardiograms were performed: 800 AVR and 609 MVR. The mean number of studies/valve was 3.7 (range:1-10); mean echo follow-up was 5.7±1.5 years (2166 valve-years). The overall prevalence of PVR was 51 (13%): AVR 32 (14%) and MVR 19 (12%). For those with serial studies, AVR-PVR was mild in 66%, moderate in 34% and severe in 0%; MVR-PVR was mild in 74%, moderate in 5%; and severe in 21%. Severity of PVR increased in 21% of AVR-PVR and 13% of MVR-PVR. Clinical outcomes (valve replacement/repair, death) were similar for those with AVR-PVR compared to those without AVR-PVR; however, compared to those who did not have PVR, a significantly higher percentage of subjects in the MVR-PVR group underwent valve replacement &/or repair (11% vs. .7%, $p<.05$). Multivariate analysis identified carotid artery disease and everted suture as predictor of PVR, but not type or position of valve replacement.
Conclusions: After mechanical valve replacement the prevalence of echocardiographically-detected PVR in asymptomatic patients is rather low, PVR severity progression of low, and clinical outcomes are for the most part similar except in those with MVR-PVR, who have higher rates of valve replacement and/or repair.

P743 Real-time three-dimensional echocardiography: a new tool for assessing mitral valve regurgitation in a pediatric population

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Purpose: Real-time three-dimensional echocardiography (RT3DE), commercially available nowadays, allows accurate left ventricular (LV) volumetric measurements without any geometric assumptions in adult patients. In pediatric patients three-dimensional LV volume measurement was validated only with an offline reconstruction technique. Our purpose is to validate this new method by measuring the stroke volume in a normal pediatric population. Then we use RT3DE to calculate regurgitant volume in pediatric patients with mitral regurgitation (MR).
Methods: Thirty-nine pediatric patients (aged one week to 16 years, median 6 years), with normal left ventricular outflow tracts and no ventricular septal defects (29 without MR and 10 with MR) had bi-dimensional echocardiography coupled with a RT3DE volumetric acquisition of the left ventricle (matrix probes, Philips). Stroke volume was calculated by the Doppler method at the aortic annulus (SVD). End-systolic and end-diastolic volumes of the LV were measured with the semi-automated method of QLab (Philips). Three-dimensional stroke volume (SV3D) was calculated as their difference. Mean time for measuring SV3D was 1 minute in patients with good endocardial detection and 3 minutes when manual corrections were needed. In the MR group regurgitant volume was calculated by the PISA method (VRPISA) and as the difference between SV3D and SVD (VR3D). Regurgitant fraction was also evaluated by the two methods (RFPISA and RF3D respectively).
Results: Measurements feasibility was 89% (impossible 3D acquisition due to agitation or poor quality acquisition). In the normal pediatric patients group, SV3D (27.9±18.1 ml) was highly correlated with SVD (30.7± 19.6 ml): r = 0.98, $p < 0.0001$, y = 0.90x + 0.08). Mean difference was 2.8±3.8 ml. The correlation was highly significant in both subgroups of patients with good endocardial detection (12 patients) and in those needing manual correction (13 patients), but was slightly better when no endocardial contour correction was needed (r = 0.97 and 0.94 respectively). In the MR group, VRPISA (20.7±16.9 ml) and VR3D (12.9±11.1 ml) are well correlated (r=0.92, $p<0.001$). Regurgitant fraction values are as also well correlated by the two methods: RFPISA=39.9±16.8, RF3D=32.8±17.0, r=0.79, p=0.006.
Conclusions: RT3DE is a simple, rapid and reliable method for evaluating stroke volume in children. Hence, its use may be of particular interest in evaluating regurgitant volume and fraction in MR. A larger population with volumetric overload (MR or ventricular shunt) is needed to reliably assess feasibility in this group.