



EUROPEAN  
SOCIETY OF  
CARDIOLOGY

Volume 25 Abstract Supplement August/September 2004 ISSN 0195-668X



# European Heart Journal

Journal of the European Society of Cardiology

**ESC Congress 2004**  
**28 August – 1 September**  
**Munich, Germany**

Editor-in-Chief: **Frans Van de Werf**

Deputy Editors: **Stefan Janssens**  
**Frank Rademakers**



# Contents

<i>Programme number (P = poster)</i>	<i>Page</i>
<b>Abstract Selection</b> .....	iii
<b>Abstract Review Committee</b> .....	vii
<b>Abstracts</b> .....	1
 <b>Day 2 — Sunday 29 August 2004</b>	
117–122 Trends and risk factors in cardiovascular disease .....	3
123–128 Risk factors management in prevention .....	4
129–134 Predictive factors in dilated cardiomyopathy .....	6
135–140 New applications of strain and strain rate imaging .....	7
149–154 From mice to men: lessons in myocardial protection and hypertrophy .....	9
155–160 Impact of non surgical treatment and physiological stress on the GUCH-heart .....	10
161–166 Vascular growth and collateral vessels: opportunities and drawbacks .....	12
176–181 Drug eluting stent in complex lesion subsets .....	13
182–187 Gene variants in hypertension, coronary artery disease and dilated cardiomyopathy .....	15
188–193 Markers of severity in hypertrophic cardiomyopathy .....	16
199–204 How to perfect coronary artery bypass grafting? .....	18
209–214 Intravascular ultrasound and plaque characterisation .....	19
215–220 Epidemiology and treatment of pulmonary arterial hypertension .....	21
221–226 Revisiting the electrocardiogram and electrophysiologic markers of arrhythmic events .....	22
229–234 Nursing aspects of acute cardiac care .....	24
244–249 Follow-up in the pacemaker and implantable cardioverter-defibrillator Era: old and new issues .....	25
250–255 Threatening complications with sophisticated devices .....	27
256–261 New risk markers of sudden death after acute myocardial infarction .....	28
275–280 Inotropic intervention: new mechanisms .....	30
281–286 Muscle wasting and cachexia in heart failure: ready for intervention .....	31
304–309 Percutaneous coronary intervention and beyond for ST-elevation acute myocardial infarction .....	33
310–315 IIb or not to be in acute myocardial infarction? .....	34
P356–P361 Moderated e-Posters I: Cardiac dysfunction: new insights .....	36
P362–P377 Atherosclerosis I – Basic Science .....	37
P378–P384 Nuclear cardiology/Magnetic resonance imaging and cardiac radiology .....	41
P385–P386 Computers in cardiology .....	43
P387–P401 Echocardiography/Doppler .....	44
P402–P417 Atherosclerosis I – Basic Science .....	48
P418–P424 Nuclear cardiology/Magnetic resonance imaging and cardiac radiology .....	52
P425 Computers in cardiology .....	53
P426–P441 Echocardiography/Doppler .....	54
P442–P449 Moderated Poster session I: Arterial hypertension .....	58
P450–P466 Atrial fibrillation .....	60
P467–P498 Mechanisms of arrhythmias .....	64
P499–P540 Heart failure: clinical aspects .....	71
P541–P558 Pulmonary circulation .....	81
P559–P606 Thrombosis .....	86
P607–P623 Coronary plaque imaging .....	98
P624–P652 Endothelial function .....	102
P653–P682 Myocardial disease .....	109
683–686 Computed tomography: Imaging of coronary arteries .....	116
688–691 Young Investigators' Award Session – Basic Science .....	117
693–696 Young Investigators' Award Session – Thrombosis .....	118
698–701 Young Investigators' Award Session – Population Sciences .....	119
703–706 Young Investigators' Award Session – Clinical Science .....	120
729–734 New prevention strategies .....	121
735–740 Women, men and cardiovascular disease .....	123
741–746 Multiple uses of myocardial deformation assessment by echocardiography .....	124

747–752	Communication in vascular smooth muscle cells	126
767–772	Oxidative stress and the endothelium	127
794–799	Improving risk stratification by nuclear cardiology	129
817–822	Pathophysiological aspects of resynchronisation therapy	130
823–828	Determinants and prevention of post myocardial infarction left ventricular remodelling	132
831–836	Ablation of atrial fibrillation: from source to success	133
846–851	Heart failure with preserved systolic function: whats new?	135
852–857	Diagnosis and evaluation of heart failure – still a challenge in the everyday clinical practice	136
876–880	Novel therapeutic strategies in chronic stable angina	138
913–918	Dilated cardiomyopathy or viral myocarditis?	139
P955–P960	Moderated e-Posters II: Brugada syndrome	141
P961–P976	Atherosclerosis II – Basic Science	142
P977–P983	Nuclear cardiology/Magnetic resonance imaging and cardiac radiology	146
P984–P985	Computers in cardiology	147
P986–P1000	Echocardiography/Doppler	148
P1001–P1016	Atherosclerosis II – Basic Science	152
P1017–P1023	Nuclear cardiology/magnetic resonance imaging and cardiac radiology	156
P1024–P1025	Computers in cardiology	157
P1026–P1039	Echocardiography/Doppler	158
P1040–P1047	Moderated Poster session II: Exercise testing	161
P1048–P1076	Implantable cardioverter-defibrillator therapy	163
P1077–P1096	Ventricular tachycardia	170
P1097–P1148	Heart failure: experimental aspects and risk of inflammation	175
P1149–P1196	Ischaemia basic aspects	188
P1197–P1225	Non-coronary interventions	199
P1226–P1254	Hypertension	206
P1255–P1276	Valve surgery	213
1277–1280	Aortic interventions	218
 <b>Day 3 — Monday 30 August 2004</b>		
1300–1305	Endothelial function, oxidative stress and nitric oxide	223
1306–1310	Gene expression, contractile function and hypertrophy	224
1312–1317	Physical fitness: correlations and determinants	225
1318–1323	Percutaneous interventions in congenital heart disease	227
1337–1342	Myocardial perfusion during acute ischaemia: the added value of myocardial contrast echocardiography (MCE)	228
1343–1348	New aspects of endothelial function related to atherosclerosis	230
1353–1358	Markers and identification of the vulnerable plaque	231
1369–1374	Protection against ischaemia and ischaemia-reperfusion injury	233
1375–1380	Frequent unwanted companions: hypertension and hyperlipidaemia	234
1381–1386	New horizons in risk stratification of unstable coronary syndromes	236
1399–1403	Resistance to antiplatelet agents	237
1405–1410	Intensive treatment for diabetes: key to improved prognosis	238
1411–1416	Oral treatment in prevention of in-stent restenosis	240
1425–1430	Atrial fibrillation – clinical presentation risk factors and prognosis	242
1458–1463	Risk stratification tools in the prediction of coronary events	243
1469–1474	Statins in heart failure: friend or foe?	244
1475–1480	Betablockade: new aspects in chronic heart failure	246
1496–1501	Primary percutaneous coronary intervention – special issues	248
1502–1507	Evolving concepts in the management of aortic stenosis	249
1512–1517	Registries and guidelines in acute coronary syndromes	251
P1544–P1559	Angiogenic and stem cell therapy – Basic Science	252
P1560–P1566	Nuclear cardiology/Magnetic resonance imaging and cardiac radiology	256
P1567–P1568	Computers in cardiology	258
P1569–P1583	Echocardiography/Doppler	258
P1584–P1598	Angiogenic and stem cell therapy – Basic Science	262
P1599–P1606	Nuclear cardiology/Magnetic resonance imaging and cardiac radiology	266
P1607–P1608	Computers in cardiology	268
P1609–P1623	Echocardiography/Doppler	268
P1624–P1631	Moderated Poster session III: Catheter ablation	272
P1632–P1665	Catheter ablation	274



P1666–P1709	Peptides and heart failure .....	282
P1710–P1766	Acute coronary syndromes .....	292
P1767–P1811	Percutaneous coronary intervention .....	306
P1812–P1826	Nursing .....	317
P1827–P1844	Exercise testing and rehabilitation .....	321
P1845–P1871	Hypertension therapeutic aspects .....	325
1872–1875	Advanced cardiac imaging .....	332
1929–1934	Bio-markers of cardiovascular disease .....	333
1935–1940	Novel magnetic resonance techniques to assess coronary artery disease .....	334
1941–1945	Mechanisms underlying plaque instability .....	336
1947–1952	Hormones and cardiac function .....	337
1961–1966	Clinical and genetic aspects of arrhythmias .....	339
1967–1972	Recent advances in three-dimensional echocardiography .....	340
1973–1978	Ischaemia-reperfusion, slow flow and no reflow .....	342
1989–1994	Computer-based advances in cardiology .....	343
1995–2000	Vascular and myocardial damage and remodelling .....	345
2001–2006	Subclinical peripheral artery disease: a marker for severity of coronary artery disease .....	346
2007–2012	Stem cells in heart failure .....	348
2026–2031	Percutaneous interventions in unstable coronary syndromes .....	349
2032–2037	Neurally mediated syncope: a recurrent syndrome revisited .....	351
2042–2047	Novel pharmacological approaches for the treatment of atrial fibrillation .....	352
2052–2057	New developments in thrombolysis: fibrinolysis .....	354
2087–2091	A new era in the prognostic assessment of heart failure patients .....	355
2097–2102	New evidence for the beneficial effects of exercise training .....	357
2107–2112	Primary percutaneous coronary intervention in acute myocardial infarction .....	358
2113–2117	Many things to remember in CABG .....	360
2119–2123	Exercise testing in mitral valve disease .....	361
2129–2134	Drug eluting stents in diabetic patients .....	362
2140–2145	Bad and good habits .....	364
P2166–P2178	Stress-induced myocardial damage and protective mechanisms – Basic Science .....	365
P2179–P2185	Nuclear cardiology/MRI and cardiac radiology .....	368
P2186	Computers in cardiology .....	370
P2187–P2201	Echocardiography/Doppler .....	370
P2202–P2213	Stress-induced myocardial damage and protective mechanisms .....	374
P2214–P2220	Nuclear cardiology/Magnetic resonance imaging and cardiac radiology .....	377
P2221–P2222	Computers in cardiology .....	379
P2223–P2237	Echocardiography/Doppler .....	379
P2238–P2245	Moderated Poster session IV: Inflammation and coronary artery disease .....	383
P2246–P2265	Pacing .....	385
P2266–P2286	Mechanisms of arrhythmias .....	390
P2287–P2341	Resynchronisation therapy .....	395
P2342–P2409	Acute myocardial infarction and reperfusion therapy .....	408
P2410–P2438	Coronary circulation: functional evaluation .....	425
P2439–P2484	Epidemiology .....	432
2485–2488	Databases and guidelines .....	443
 <b>Day 4 — Tuesday 31 August 2004</b>		
2605–2609	Congenital heart disease: secondary challenges after early “repair” .....	447
2611–2616	Experimental cell transplantation: preclinical and clinical developments .....	448
2617–2622	More fuel to inflammation .....	449
2636–2641	Chronic heart failure: new treatment options .....	451
2642–2647	Oral anticoagulation for atrial fibrillation – still unresolved .....	452
2648–2653	The diabetic vessel .....	454
2667–2672	Heart failure news from off the beaten track .....	455
2673–2677	Catheter ablation of ventricular tachycardia .....	457
2709–2714	Diabetes and the heart .....	458
2725–2730	Treatment and outcome of acute pulmonary embolism .....	459
2768–2773	Coronary artery disease and renal failure: prognostic aspects and prevention .....	461
2774–2779	Clopidogrel loading – 600 mg may be better .....	462
2799–2804	Mitral regurgitation: from prognosis to treatment .....	464
P2830–P2835	Moderated e-Posters III: Drug therapy in cardiovascular disease .....	465





P2836–P2851	Endothelial function, vascular care and remodelling	467
P2852–P2858	Nuclear cardiology/Magnetic resonance imaging and cardiac radiology	471
P2859	Computers in cardiology	472
P2860–P2874	Echocardiography/Doppler	473
P2875–P2887	Ion channels and arrhythmias	476
P2888–P2894	Nuclear cardiology/Magnetic resonance imaging and cardiac radiology	479
P2895–P2896	Computers in cardiology	481
P2897–P2911	Echocardiography/Doppler	482
P2912–P2919	Moderated Poster session V: Trials in heart failure	485
P2920–P2950	Atrial fibrillation	488
P2951–P2995	Heart failure: clinical aspects	495
P2996–P3046	Coronary artery disease in high-risk patients	506
P3047–P3079	Restenosis and drug-eluting stents	519
P3080–P3123	Risk factor	527
P3124–P3144	Myocardial disease	537
3145–3148	Simulation and modelling	543
P3265–P3270	Moderated e-Posters IV: Nurse-led interventions in patients with heart failure	544
P3271–P3284	Cardiac hypertrophy and remodelling contractile function – Basic Science	545
P3285–P3291	Nuclear cardiology/Magnetic resonance imaging and cardiac radiology	548
P3292–P3293	Computers in cardiology	550
P3294–P3308	Echocardiography/Doppler	551
P3309–P3323	Cardiac hypertrophy and remodelling contractile function – Basic Science	554
P3324–P3330	Nuclear cardiology/Magnetic resonance imaging and cardiac radiology	558
P3331–P3332	Computers in cardiology	560
P3333–P3347	Echocardiography/Doppler	560
P3348–P3354	Moderated Poster session VI: In stent restenosis	564
P3355–P3374	Electrocardiography	566
P3375–P3394	Syncope	571
P3395–P3418	Heart transplantation	575
P3419–P3444	Heart failure – medical therapy	581
P3445–P3485	Valvular disease	588
P3486–P3512	Coronary artery disease: risk stratification and medical therapy	597
P3513–P3531	Stroke	604
P3532–P3544	Exercise testing and rehabilitation	608
P3545–P3577	Grown-up and congenital heart disease	611
P3578–P3588	Coronary artery bypass grafting	620
3589–3592	Databases	622
<b>Day 5 — Wednesday 1 September 2004</b>		
3617–3622	Pericarditis	627
3627–3632	Structure and function of cardiac ion channels	628
3637–3642	Lipid modulation in atherogenesis – beyond cholesterol	630
3643–3648	Pulse pressure, distensibility and cardiovascular risk	631
3649–3654	Fat, lean or active: all relevant for cardiovascular risk?	633
3655–3660	Conduction of the electrical impulse	634
3661–3666	Novel players in receptor-mediated signalling in cardiac myocytes	636
3671–3676	Nuclear cardiology – beyond the future	637
3681–3686	Challenges in aortic interventions	638
3687–3692	Stenting of supra-aortic extracranial arteries	640
3693–3698	New navigation and mapping techniques for targeting the arrhythmogenic substrate	641
3703–3708	Reducing thrombotic risk – old concepts, new therapies	643
3713–3718	Acute myocardial intervention and diabetes	644
3723–3728	Pacing mode and lead position: does it matter?	646
3729–3734	How important is abnormal respiration in heart failure?	647
3735–3740	New insights into native or repaired mitral valve dynamics	649
3745–3750	Renal dysfunction and anaemia – overlooked parts of the heart failure syndrome	650
3751–3756	Ablation of atrial fibrillation: new techniques	652
3757–3762	Management of acute heart failure	653
3763–3768	Multiple applications of cardiac computed tomography	654
3778–3783	Challenging issues after valve replacement	656
3784–3789	Clinical assessment of resynchronisation therapy	658

3790–3795	Hypertension and heart failure .....	660
3801–3806	Blood pressure reduction and cardiovascular risk .....	661
3811–3816	Heart economics .....	663
3821–3826	Physical training and secondary prevention .....	664
3827–3831	Percutaneous coronary interventions in diabetic patients .....	666
3833–3838	Prognostic value of stress echocardiography .....	667
3839–3843	Restenosis following bare stents .....	669
3845–3850	Drug eluting stent to treat in-stent restenosis .....	670
<b>Author Index</b> .....		673
<b>Index of Topics</b> .....		717

Legend

Every abstract is identified as either "BENCH" or "BEDSIDE".

BENCH (Animal studies or in vitro experiments at the molecular, cellular or tissue level (including human tissue). The core data are generated in the lab.)	
BEDSIDE (Patients studies. The core data are generated during clinical studies or interventions.)	

This abstract book has been produced electronically by Elsevier B.V.

Every effort has been made to faithfully reproduce the abstracts as submitted. However, no responsibility is assumed by the organizers for any injury and/or damage to persons or property as a matter of product liability, negligence or otherwise, or from any use or operation of any methods, products, instructions or ideas contained in the material herein. Because of the rapid advances in the medical sciences, we recommend that independent verification of diagnoses and drug dosages should be made.

**218 Inhaled treprostinil is a potent pulmonary vasodilator in severe pulmonary hypertension**



R. Voswinckel, M.G. Kohstall, B. Enke, T. Gessler, F. Reichenberger, H.A. Ghofrani, W. Seeger, H. Olschewski. *Medical Clinic 2, Department of Internal Medicine, Giessen, Germany*

**Background:** Treprostinil has been approved for therapy of PAH (US and Canada) as continuous subcutaneous infusion. However, local pain at the infusion site is a major drawback. Inhaled therapy with another stable prostacyclin analogue (iloprost) has been approved for PPH (EMA). In this study we investigated the acute hemodynamic response to inhaled treprostinil.

**Methods:** Open-label, single blind placebo-controlled clinical study. After placement of a Swan-Ganz catheter and a femoral artery line, patients inhaled solvent solution (placebo) (n=8) or treprostinil for 6 min (OptiNeb ultrasound nebulizer, Nebu-tec, Germany) in concentrations of 16, 32, 48, and 64 µg/ml (n=6, 6, 6, and 3 patients). Measurement was performed before and after 0, 15, 30, 60, 90, 120, 150 and 180 min. The mean area between the placebo and the treprostinil curves (ABC186) was calculated (baseline=100%).

**Results:** We investigated idiopathic PAH (n=10), collagen vascular disease (n=5), chronic thromboembolic disease (n=9), and pulmonary fibrosis (n=5), f/m = 19/10, age 56 ± 3 years, PAP, PAWP, and CVP 51.3 ± 2.2, 9.2 ± 0.8, and 6.6 ± 0.6 mmHg, CO 4.4 ± 0.3 l/min, SvO<sub>2</sub> 62.3 ± 1.2%, PVR 885 ± 72 dyn s cm<sup>-5</sup>. At 16µg/ml there were no significant adverse events. Headache, cough or bronchoconstriction were observed in 2, 1, and 2 patients at 32, 48, and 64 µg/ml. These were mild and transient in all patients but one (64 µg/ml) who complained of major headache for 1 hour. Placebo inhalation was followed by slowly increasing PVR. Compared to this, the maximum treprostinil effect was reached after about 50 min and half-maximal effects at about 110 min. The ABC186 for PVR was -24.7 ± 4.4, -28.7 ± 4.9, and -29.0 ± 4.7%; PAP -14.4 ± 3.3, -13.5 ± 5.2, -13.1 ± 2.6%; SAP -5.1 ± 3.0, -6.0 ± 3.1, -3.8 ± 2.1% at 16, 32 and 48 µg/ml.

**Conclusion:** Treprostinil inhalation results in a significant long-lasting pulmonary vasodilatation. With the applied technology, at a concentration of 16µg/ml, near maximal pulmonary vasodilatation is achieved without adverse effects. At higher doses, local and systemic side effects may occur, whereas pulmonary selectivity is preserved.

This study was supported by Lung Rx.

**219 The endothelin-receptor antagonist bosentan for the treatment of pulmonary arterial hypertension associated with congenital heart defects**



O. Sitbon<sup>1</sup>, M. Beghetti<sup>2</sup>, J. Petit<sup>3</sup>, L. Iserin<sup>4</sup>, M. Humbert<sup>1</sup>, V. Gressin<sup>5</sup>, G. Simonneau<sup>1</sup>. <sup>1</sup>Hôpital Antoine Bécclère, Clamart, France; <sup>2</sup>HUG, Pediatric Cardiology Unit, Geneva, Switzerland; <sup>3</sup>CMC Marie-Lannelongue, Le Plessis-Robinson, France; <sup>4</sup>Hôpital Necker, Paris, France; <sup>5</sup>Actelion Pharmaceuticals France, Paris, France

**Background:** Treatment with the oral dual endothelin-receptor antagonist bosentan has been shown to be an effective alternative option to intravenous epoprostenol in functional class (FC) III idiopathic pulmonary arterial hypertension (PAH) patients. In patients with PAH associated with congenital heart defects (CHD), an improvement of exercise capacity and hemodynamics has been demonstrated with epoprostenol in one uncontrolled study (Rosenzweig et al. *Circulation* 1999; 99: 1858-65).

The aim of this retrospective study was to evaluate the efficacy and safety of bosentan in FC III-IV CHD-PAH patients.

Study population consisted in 24 patients (22 females, mean age 35 ± 15 years [8-68]) with CHD-PAH: atrial septal defect (ASD: 13), ventricular septal defect (VSD: 4), partial abnormal pulmonary venous return (3, associated with ASD in 2 and repaired common atrium in 1), patent ductus arteriosus (PDA: 2), VSD associated with PDA (1), aortopulmonary window (1). Four patients had undergone previous cardiac surgery.

Patients had deteriorated despite conventional therapy (including oral anticoagulants, oxygen, diuretics) and were treated with chronic oral bosentan.

**Results:** Before starting bosentan, 22 patients were in FC III and 2 in FC IV, with a resting O<sub>2</sub> saturation (SaO<sub>2</sub>) of 89 ± 9%. Mean 6-min walk distance (6MWD) was 288 ± 94 m and mean Borg index 3.0 ± 1.9. At last evaluation performed after 10 ± 9 months of bosentan treatment, 1 patient was in FC I, 8 were in FC II, 13 remained in FC III and 2 in FC IV. The mean 6MWD improved by 49 m (349 ± 85 m, p = 0.008) with no change in Borg index (3.0 ± 1.8) and resting SaO<sub>2</sub> (89 ± 6%). There were no differences between pre and post-tricuspid shunt subgroups in terms of baseline characteristics and response to bosentan therapy. After 13 ± 9 months of follow-up, all patients are alive on bosentan, but 3 (1 ASD, 1 VSD, 1 aortopulmonary window) required combination therapy with intravenous epoprostenol after 5, 7 and 9 months on bosentan.

**Conclusion:** Chronic oral bosentan treatment improves exercise capacity in patients with PAH associated with CHD who deteriorated despite conventional therapy. Bosentan had no adverse effect on arterial oxygen saturation. As previously demonstrated in patients with idiopathic PAH, long term bosentan may be an important therapeutic option for patients with PAH associated with CHD.

**220 Sildenafil in the treatment of primary pulmonary hypertension**



A. Dharmadhikari<sup>1</sup>, M. Kulkarni<sup>2</sup>, V. Tzifos<sup>3</sup>, F. Airoidi<sup>4</sup>, I. Sheiban<sup>5</sup>, <sup>1</sup>Nashik, India; <sup>2</sup>Rajebahadur Heart Foundation, Cardiology, Nashik, India; <sup>3</sup>Hospital Hunry Dununt, Interventional Cardiology, Athens, Greece; <sup>4</sup>Hospital San Raffaele, Interventional Cardiology, Milano, Italy; <sup>5</sup>University Hospital, Torino, Interventional Cardiology, Torino, Italy

**Background:** The role of sildenafil as a pulmonary vasodilator is being extensively evaluated in the treatment of pulmonary hypertension.

**Aim:** This was a prospective study to assess the benefit of adding sildenafil in patients with high pulmonary artery pressures secondary to atrial septal defect (ASD), already receiving the conventional therapy.

**Methods:** Thirty consecutive patients with moderate to severe primary pulmonary hypertension were included in this study. All the patients were diagnosed previously and were receiving the conventional therapy with digoxin, diuretic and a calcium channel blocker. Sildenafil was added in the dose of 50 mg twice a day without changing the previous regimens. Changes in the New York Heart Association (NYHA) symptom class, distance covered during the six minute walk test and modified Borg dyspnea score were evaluated monthly. Acceptance of the new drug was assessed every week in the first month and then at the monthly follow up. Echocardiography and Doppler study was undertaken at baseline and every month for a period of six months. The parameters studied were the pulmonary artery systolic pressure (PASP) by tricuspid regurgitation (TR) jet and pulmonary artery diastolic pressure (PADP) by pulmonary regurgitation (PR) jet.

**Results:** Mean age of the subjects was 42.6±9.3 years. Twenty seven (90%) were females and 3 (10%) were males. Sildenafil was well tolerated and there was no dropout because of undesirable effects of the drug. Changes in the heart rate and systemic blood pressure were not significant enough to warrant withdrawal of the drug. Two patients died during the follow-up period. At the beginning of the therapy, 22 (73.3%) patients were in NYHA Class III or IV while at the end of six months, only 8 patients remained in either of these classes (p<0.05). By the 6 min walk test, functional capacity improved from 181.5±122.4 meters to 302.7±150.3 meters (p<0.05). Modified Borg dyspnea score improved from 5.6±1.2 to 3.3±1.1 (p<0.05). PASP by TR jet (mmHg) was down from 81.3±14.7 to 51.4±11.7 (p<0.05). PADP by PR jet (mmHg) reduced from 56.2±11.7 to 33.4±9.1 (p<0.05).

**Conclusion:** Sildenafil is well tolerated, improves symptoms, and reduces the systolic and diastolic pulmonary artery pressures in patients with moderate to severe primary pulmonary arterial hypertension.

**REVISITING THE ELECTROCARDIOGRAM AND ELECTROPHYSIOLOGIC MARKERS OF ARRHYTHMIC EVENTS**

**221 Prevalence of brugada-type ecg in an apparently healthy european population**



G. Forleo<sup>1</sup>, F. Di Liberato<sup>2</sup>, L. De Luca<sup>2</sup>, G. Magliano<sup>2</sup>, V. Morgià<sup>2</sup>, F. Clementi<sup>2</sup>, M.M. Gallagher<sup>2</sup>, F. Romeo<sup>2</sup>. <sup>1</sup>University of Rome, Department of Cardiology, Roma, Italy; <sup>2</sup>University of Tor Vergata, Department of Cardiology, Rome, Italy

**Background:** The Brugada Syndrome ECG is characterized by ST-segment elevation in right precordial leads and elevated risk of lethal arrhythmias in absence of identifiable structural heart disease. Few data are available on the Brugada type ECG, especially in Europeans. No epidemiological study has applied the diagnostic criteria recently proposed by the Study Group of the Molecular Basis of Arrhythmias of the ESC.

**Methods:** We analysed the ECG and clinical data of apparently healthy European adults undergoing routine medical examinations for occupational reasons. At each examination subjects underwent a medical interview, physical examination, blood pressure measurement and 12-lead ECG. Enrolment was confined to persons without a history of heart disease at the time of first attendance in whom at least one 12-lead ECG of good quality was recorded. The ECG records of all 7483 subjects (89.6% male, age 29.5±10.8 years at first attendance) were reviewed by three cardiologists. We reviewed 1,97±2.1 ECGs for each subject. We considered a patient having a Brugada ECG pattern if 2 or more of the cardiologists judged that at least one of that persons ECGs fulfilled the criteria of the ESC Study Group.

**Results:** The Brugada pattern was present in 26 patients (0.35%), all male (table). In 17 cases (65.4%), information was available about the progress of the subject subsequent to the ECG on which the Brugada pattern was first recorded. No sudden death or cardiac arrhythmia was recorded among these patients in a follow-up of 5.2±4.6 years (total follow-up 87.8 patient-years).

	Total	Pattern 1	Pattern 2	Pattern 3	Tot. Brugada
Pts (n)	7383	2	21	3	26
Male	6618	2	21	3	26
Female	765	0	0	0	0
Male prevalence (*10000)	-	3.02	31.73	4.53	39.29
Total Prevalence (*10000)	-	2.71	28.44	4.06	35.22