patient with thrombus (score=4) had hypertrophic cardiomyopathy, mild LV dysfunction and sub-therapeutic INR (1.5) at time of TOE whilst the other patient (score=2) was female with hypertension and mild LV dysfunction. No patients with a score of 0 or 1 had LAA thrombus.

**Conclusions** Patients classed as low riskby the$CHA_2DS_2VASc$ score (score of 0–1) do not require a pre-ablation TOE to screen for LAA thrombus provided they have been anticoagulated with a therapeutic INR. This would lead to a significant reduction in healthcare expenditures by reducing unnecessary TOE tests and improve patient experience by avoiding TOE.

# Cardiac Rhythm Management

## 66 SUDDEN DEATH IN WOLF-PARKINSON-WHITE. DESCRIPTION OF POST-MORTEM PATHOLOGICAL FINDINGS AND CLINICAL CORRELATES IN 19 CASES

[1]Gherardo Finocchiaro*, [2]Michael Papadakis, [2]Harshil Dhutia, [1]Alexandros Steriotis, [3]Jan Lukas Robertus, [1]Elijah Behr, [1]Mary Sheppard, [1]Sanjay Sharma. *[1]St George's University of London Hospital; [2]St George's University Hospital; [3]St George's University of London Hospital and Royal Brompton Hospital; *Presenting Author*

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**Aims** The prevalence of pre-excitation (Wolff-Parkinson-White (WPW) pattern) is estimated at 0.3%. The risk of malignant arrhythmias in asymptomatic individuals is low and ablation of the accessory pathway is considered to diminish the risk of sudden cardiac death (SCD). The aim of the study was to describe clinical characteristics and pathological features of SCD victims with a pre-morbid diagnosis of WPW.

**Methods** Between 1994 and 2014, 3684 cases of SCD were referred to our cardiac pathology centre; 19 (0.5%) with known pre-excitation on their ECG. Clinical information were obtained from referring coroners. All subjects underwent detailed post-mortem evaluation including histological analysis by an expert cardiac pathologist.

**Results** The majority of patients were males (n = 16, 84%) of Caucasian descent (n = 17; 89%). The mean age was 31 ± 15 years (2 patients over the age of 50). Five cases (26%) were asymptomatic. Of the 14 symptomatic patients, 13 (68%) reported palpitations, 1 (5%) syncope. Five individuals (26%) had a previous ablation, 4 of which were judged to be successful with resolution of pre-excitation on the ECG. In the majority of cases (n = 15; 79%) SCD occurred at rest. The mean heart weight was 408 ± 105 g. In 10 patients (53%) the post-mortem exam revealed a normal heart, in 5 cases there was a definitive cardiac pathology (n = 4 with hypertrophic cardiomyopathy, n = 1 with cardiac sarcoid), and 4 cases demonstrated autopsy findings of uncertain significance (n = 2 with idiopathic left ventricular hypertrophy, n = 1 with idiopathic fibrosis, and n = 1 with enlarged left ventricle). Out of the 5 asymptomatic patients, the post-mortem revealed HCM in 3 and a normal heart in 2 cases. The patients that underwent previous ablation were characterized by a normal heart in 3 cases and by idiopathic left ventricular hypertrophy at the post-mortem.

**Conclusions** One out of four SCD victims was asymptomatic, raising concerns relating to the value of symptoms in risk

**Abstract 66 Table 1**

| No. | Age | Sex | Ethnicity | Symptoms | Circumstances of death | Heart weight | MWT | LV fibrosis | RV fibrosis | Valvular abnormalities | Other diagnosis |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 7 | Male | White | Known WPW, asymptomatic | Unknown | 200 | | No | No | No | Normal heart |
| 2 | 48 | Male | White | Known WPW, asymptomatic | Died at rest | 526 | 20 | Yes | No | No | HCM |
| 3 | 28 | Male | White | Known WPW, palpitations, previous attempted ablation | Died at sleep | 316 | 19 | No | No | No | Mild LVH |
| 4 | 20 | Male | Indian | Known WPW, palpitations, planned ablation | Died at rest | 237 | 9 | No | No | No | Normal heart |
| 5 | 26 | Male | White | Recent diagnosis of WPW, palpitations | Died at rest | 361 | 16 | No | No | No | Normal heart |
| 6 | 20 | Male | White | Known WPW, palpitations, symptom free for 11 years, recent palpitations | Died at rest | 444 | 14 | No | No | No | Normal heart |
| 7 | 20 | Female | White | Known WPW, palpitations | Died at rest | 311 | | No | No | No | Normal heart |
| 8 | 27 | Male | White | Known WPW, previous ablation | Died during exertion | 426 | 21 | No | No | No | Normal heart |
| 9 | 24 | Male | White | Known WPW, previous ablation | Died at rest | 578 | | No | No | No | ILVH |
| 10 | 46 | Male | Black | Known WPW, currently under evaluation | Died at rest | 474 | 16 | No | No | No | Cardiac sarcoid |
| 11 | 33 | Male | White | Known WPW, palpitations | Died at rest | 532 | 21 | No | No | No | Enlarged left ventricle |
| 12 | 50 | Female | White | Known WPW, asymptomatic | Died during sleep | 384 | 18 | No | No | No | HCM |
| 13 | 16 | Male | White | Known WPW, palpitations | Died at rest | 366 | 25 | No | No | No | HCM |
| 14 | 26 | Male | White | Known WPW, athlete, asymptomatic | Died during exertion | 510 | | Yes | No | No | HCM |
| 15 | 36 | Male | White | Known WPW, syncopal episode | Died at rest | 498 | | Yes | No | No | Idiopathic fibrosis |
| 16 | 18 | Male | White | Known WPW, asymptomatic | Died at rest | 390 | 19 | No | No | No | Normal heart |
| 17 | 55 | Female | White | Known WPW, palpitations, on betablockers | Died at rest | 385 | 13 | No | No | No | Normal heart |
| 18 | 65 | Male | White | Known WPW, palpitations, previous ablation | Died at rest | 516 | | No | No | No | Normal heart |
| 19 | 28 | male | White | Known WPW, palpitations, previous ablation | Died during sleep | 302 | 12 | No | No | No | Normal heart |

stratification of individuals with pre-excitation. In addition, accessory pathway ablation did not eliminate the risk of SCD possibly due to multiple pathways. Finally, pre-excitation was associated with additional structural abnormalities in almost 50% of cases, underscoring the necessity of performing baseline echocardiography in all individuals and suggesting that the combination of pre-excitation with additional cardiac pathology may render individuals at higher risk of SCD.

## 67 OBESITY AND SUDDEN DEATH. PATHOLOGICAL INSIGHTS FROM A LARGE PATHOLOGY REGISTRY

[1]Gherardo Finocchiaro*, [2]Michael Papadakis, [2]Harshil Dhutia, [1]Ahmed Merghani, [1]Stathis Papatheodorou, [2]Elijah Behr, [2]Sanjay Sharma, [1]Mary Sheppard. *[1]St George's University of London Hospital; [2]St George's University Hospital; *Presenting Author*

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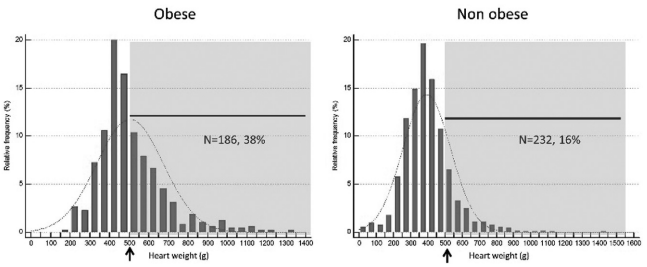
**Aims** Obesity is a rising public health problem and widely known risk factor for cardiovascular diseases. The relationship between obesity and sudden cardiac death (SCD) is unclear and based on small cohort studies. The aim of the study was to determine the main features and aetiologies in a large cohort of SCD occurred in obese subjects

**Methods** Between 1994 and 2014, 3684 consecutive cases of SCD were referred to our cardiac pathology centre. In 1954 body mass index (BMI) data were available; obesity was defined by a BMI $\geq$ 30. All subjects underwent macroscopic and microscopic post-mortem evaluation by an expert cardiac pathologist. Clinical information were obtained from the referring coroners.

**Results** Four hundred ninety-one patients (25%) were obese. The average heart weight (HW) in obese patients was 505 ± 170 g and 186 (38%) had a HW of more than 500 g. Obese patients were older at death (39 ± 14 vs 35 ± 16 years in non-obese, $p < 0.001$). In obese SCD victims the most common post-mortem findings were: normal heart (sudden arrhythmic death syndrome, SADS) (n = 192, 39%), followed by idiopathic left ventricular hypertrophy (ILVH) (n = 88, 18%) and critical coronary artery disease (CAD) (n = 57, 12%). Less frequently observed were hypertrophic cardiomyopathy (HCM) (n = 24, 4%) and arrhythmogenic right ventricular cardiomyopathy (ARVC) (n = 22, 4%). When compared with non-obese SCD victims, SADS was less common (39 vs 51%, $p < 0.001$) while ILVH and critical CAD were more frequent (18 vs 3%, $p < 0.001$ and 12 vs 6%, $p < 0.001$ respectively). In young patients (<35 years old) the prevalence of critical and non-critical CAD was significantly higher in obese subjects (23 vs 10% in non-obese, $p < 0.001$).

**Conclusions** Various conditionsunderlie SCD inobese patients, with a prevalence of SADS, ILVH and CAD. The degree of hypertrophy measured by heart weight appears in excess even after correction for body size, postulating its possible pathogenetic role in the development of fatal arrhythmias. Almost one in four young obese sudden death victims shows some degree of CAD, underscoring the need for primary prevention in this particular subgroup.




**Abstract 67 Figure 1**

## 68 RIPPLE MAPPING THE VENTRICULAR SCAR: A NOVEL APPROACH TO SUBSTRATE ABLATION OF POST-INFARCT VENTRICULAR TACHYCARDIA TO PREVENT IMPLANTABLE DEFIBRILLATOR THERAPY

[1]Vishal Luther*, [2]Nicholas W Linton, [2]Shahnaz Jamil-Copley, [2]Michael Koa-Wing, [2]Norman Qureshi, [2]FuSiong Ng, [2]Phang Boon Lim, [2]Zachary Whinnett, [2]David Wyn Davies, [2]Nicholas S Peters, [2]Prapa Kanagaratnam. *[1]North West Thames Deanery; [2]Imperial College Healthcare NHS Trust; *Presenting Author*

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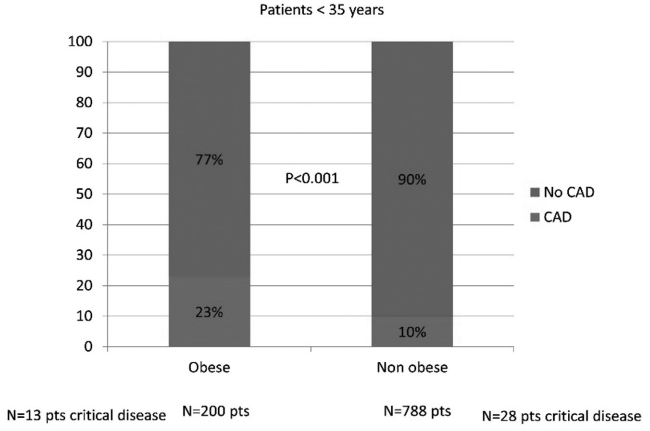
**Introduction** Ventricular Tachycardia (VT) is a common cause of mortality post myocardial infarction. Any mortality benefit of an implantable cardiac defibrillator (ICD) may be offset by the significant morbidity caused by VT shocks from the device. The VT circuit is dependent upon channels of surviving myocardium within the infarct substrate. As VT is often poorly tolerated, ablation of characteristic electrograms (fractionated and late potentials) associated with these channels in sinus rhythm is preferred to reduce ICD therapies, though recurrence rates remain high. Ripple Mapping (RM) displays all electrogram components from each anatomical point as a dynamic bar that protrudes from its 3D location. We have described how RM might visualize "conducting channels" (RM-CCs) of these characteristic electrograms within the infarcted substrate in a retrospective series. In this study, we used RM prospectively (CARTO3v4™) to characterize the VT substrate to guide ablation.

**Methods** Consecutive pts referred for VT ablation following episodes of sustained VT or ICD therapies post distant MI were included. High point density bipolar LV endocardial electrograms were collected in sinus rhythm or ventricular pacing within the infarct "scar" (<1.5mV) and reviewed for RMCC identification. Ablation targeted all RMCCs and isolated clusters of late activation considered an endocardial portion of an




**Abstract 67 Figure 2**