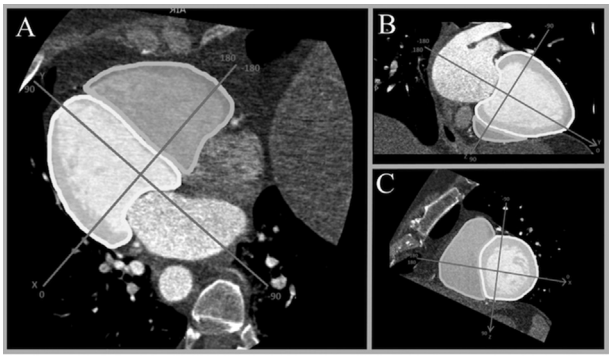
dimensional electrocardiographic method, can differentiate between pacing modes in patients with cardiac resynchronisation therapy (CRT).

Methods At a tertiary cardiology centre, CGM recordings were performed using four pacing modes: no pacing; right ventricular (RV) pacing; left ventricular (LV) pacing and biventricular (BIV) pacing. Three orthogonal CGM planes orientated to the long axis (XY), the frontal plane (YZ) and the short axis (XZ) of the heart were constructed (see figure), and the direction of the QRS axis was calculated for each pacing mode in each plane. During BIV pacing, the direction of CGM QRS axis was compared between patients with optimal and non-optimal 12-lead ECG pacing variables. Optimally paced 12-lead ECG variables were defined as an R/S ratio greater than or equal to 1 in V_1 and/or R/S ratio less than or equal to 1 in lead I.

Results Eleven participants (aged 77.4±11.5; 63.6% male, LVEF 31±6%) were consecutively recruited. Only QRS axis measured in the XY plane could distinguish between LV and BIV pacing vs. no pacing ($p=0.005$ and $p=0.001$ respectively). Mean QRS axis and 95% confidence intervals (CI) for each pacing mode is shown in the table. Mean QRS axis in the XY plane with pacing off and during RV pacing was leftwards and basal; LV pacing was apical; and BIV pacing was rightwards and basal. There was a marked difference in the direction of QRS axis between patients with optimal vs. non-optimal paced QRS morphology in the XY plane (rightwards and basal vs inconsistent).

Abstract 8 Table 1

	XY plane	*YZ plane*	*XZ plane*
No pacing	−23 (95% CI: −35 to 11)	147 (95% CI: 117 to 177)	−8 (95% CI: −45 to 29)
RV pacing	−52 (95% CI: −66 to −40)	144 (95% CI: 136 to 152)	−10 (95% CI: −23 to 4)
LV pacing	148 (95% CI: 88 to −152)	−24 (95% CI: −166 to 113)	74 (95% CI: 7 to 157)
BIV pacing	−130 (95% CI: 161 to −62)	150 (95% CI: 29 to −88)	−91 (95% CI: 102 to 71)



Abstract 8 Figure 1 Cardiac CT sections taken in the same approximate sections as the CGM planes – XY plane (panel A), YZ plane (panel B), XZ plane (panel C). Green shading represents the right ventricle; yellow shading represents the left ventricle.

Conclusions CGM recorded in the XY plane can accurately detect differences between ventricular pacing sites. Further studies are needed to determine whether CGM has clinical utility in optimising patients undergoing CRT.

9 IDENTIFICATION OF A POTENTIAL CAUSE OF NON-RESPONSE TO BIVENTRICULAR PACING USING INTRAVENOUS ADENOSINE

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Aim We aim to evaluate the efficacy and safety of determining the presence of true biventricular pacing by administration of intravenous adenosine. This aims to determine whether non-response to biventricular pacing therapy is partially explained by over-counting of the percentage of pacing therapy by the presence of fusion beats. We compared the proportion of patients with fusion beats in non-responders compared to responders

Methods This will be a single centre, prospective, cohort study. 71 consecutive patients with implanted cardiac resynchronisation devices between July 2006 and November 2015 were identified. Patients underwent the adenosine trial at least 12 months post CRT device implantation to detect the presence of potentially ineffective biventricular pacing. This was compared with the recorded percentage of biventricular pacing reported during device interrogation. Correlation was made between response to CRT therapy and presence of effective biventricular pacing.

Results Preliminary results from 24 patients demonstrate that symptomatic adenosine administration was successful in identifying true effective biventricular paced beats. This has allowed us to identify patients with fusion beats. 8 patients were non-responders to CRT, whereas 16 patients were responders. 50% (n=4) of non-responders and 12.5% (n=2) of responders had definite electrocardiography (ECG) changes through the symptomatic adenosine administration, showing that a percentage of the paced beats counted by the CRT device may in fact be fusion beats. Figure 1 shows the clear change in the morphology of the QRS complex in one of the non-responders.

Figure 1 Electrocardiogram of a non-responder during symptomatic adenosine administration

Conclusion Preliminary results suggest that symptomatic adenosine administration is successful in identifying true effective biventricular paced beats which may be overestimated by CRT device interrogation. Correlation with response to CRT is yet to be established, but in 50% of the non-responders we identified a definite change in the morphology of the paced rhythm during the symptomatic response to adenosine. This raises a question of whether the poor response to CRT for these patients is actually due to the insufficient percentage of effective biventricular pacing (<98%).