irregular pulse and indications for contacting a physician. In addition, an information movie was on continuous looping display. Two AF specialist nurses guided passers-by through the campaign information and provided them with answers to their questions. After looking at the displayed material, patients received a take-home information brochure. To assess AF knowledge, this study interrogated the public by a pre- and posttest, and compared the results with a post-only comparison group. Participants were randomly selected to the pre-test group and received a 6-item AF knowledge questionnaire at the entrance of the hospital prior to and out of sight of the info stand. The post-test questionnaire was identical for both groups. The answers of the pre-posttest (n = 111) and post-only comparison group (n = 256) were compared.

Results. In general, a low AF knowledge level was recorded in the pre-test group (48% to 75% correct answers). In contrast, after the info campaign, the percentage of participants being unable to define AF decreased from 32% to 5%, the percentage of those not knowing that AF can be asymptomatic decreased from 51% to 16% ($P < 0.05$), and the proportion being unaware that AF prevalence increases with age was reduced from 52% to 5%. Knowledge about stroke-risk, symptoms and pulse self-examination as a means to diagnose AF increased respectively from 71%, 75% ($P < 0.05$) and 58% to 98% of all. Although both post-test groups showed high AF knowledge (84% to 99% correct answers), those that had also done the pre-test scored better than those that only did the post-test, (e.g. knowledge about age-related prevalence = 94% vs 86%; $P < 0.05$), indicating that a pre-test can improve knowledge uptake from a directed info campaign.

Conclusion. A simple information campaign significantly increases awareness and knowledge of AF. In addition, a pre-test measurement has added value, as it directs participants to search actively for specific information.

Terminal QRS axis in right bundle-branch block and Brugada syndrome: a new look at right ventricular conduction delay. — P. Koopman[1], D. Keulards[2], M. Falter[3], H. Gruwez[3], R.M.A. ter Bekke[2], P. Dendale[1], P.G.A. Volders[2], A.P.M. Gorgels[2] *([1]Jessa Hospital, Hasselt, B, [2]Maastricht University Medical Centre, Maastricht, NL, [3]University Hasselt, Hasselt, B)*

Objectives. Brugada syndrome (BrS), complete right bundle branch block (CRBBB) and incomplete right bundle branch block (IRBBB) are associated with delayed electrical activation of the right ventricle. The aim of this study was to assess direction of the terminal axis (TA) of the QRS complex on 12-lead electrocardiogram (ECG) as a new method to add to the pathophysiological understanding of these conditions.

Methods. We retrospectively determined TA in 23 patients with BrS, 15 with CRBBB and 16 with IRBBB, as compared to 58 normal ECGs. TA was also analyzed in 53 patients undergoing ajmaline testing. TA was defined as the frontal plane axis of the terminal QRS activation at 20 ms before the end of the QRS. TA was categorized in four quadrants (Q) on a circular diagram: Q1 (-60 to + 30), Q2 (+30 to + 120), Q3 (+120 to –150) and Q4 (-150 to –60 degrees).

Results. Influence of diagnosis on TA distribution was significant ($P = 0.001$). In normal ECGs, TA was equally distributed in all quadrants. In the presence of CRBBB, TA was directed towards Q3 (73%, $P = 0.0134$). TA was directed towards Q4 for IRBBB (69%, $P = 0.0457$) and BrS (78%, $P = 0.0147$, specificity 59.6%, sensitivity 78%). TA was directed towards Q4 for baseline and peak ajmaline ECG (positive test: baseline 78%, peak 78%, P = ns; negative test: baseline, 68%, $P = 0.0018$; peak 66%, $P = 0.0009$), with no significant change in TA direction from baseline to peak administration ($P = 0.288$).

Conclusion. TA of BrS patients is significantly directed towards the electro-anatomical location of the right ventricular outflow tract (RVOT, Q4), suggesting involvement of RVOT conduction delay in BrS pathophysiology. Based on TA, discrimination can be made between BrS and CRBBB.

Repeat genetic testing in a large LQTS family reveals a new pathogenic mutation c.2038delG in KCNH2 initially missed with DHPLC. — T. Robyns, C. Kuiperi, T. De Ravel, A. Corveleyn, C. Garweg, J. Ector, R. Willems, H. Heidbüchel, D. Nuyens (*UZ Leuven, Leuven, B)*

A 54 year old woman with a medical history noticeable for epilepsy is hospitalized for an epileptic seizure in a local hospital in 2007. In hospital she suffers a new 'epileptic insult', however electrocardiographic monitoring shows evidence for torsades de pointes, spontaneously recovering to sinus rhythm. Her ECG is noticeable for a prolonged QTc interval of 600 ms.

Beta blocker therapy is started and she gets implanted with an ICD. Antiepileptic drugs can be withdrawn and she remains free of seizures subsequently. A detailed family history reveals that one son died suddenly at the age of 14 years old in rest and a further 2 siblings of the index patient died suddenly at young age. Family members were than referred for further phenotypical evaluation at our institution. Mutation screening of the LQTS susceptibility genes with denaturing high pressure liquid chromatography (DHPLC) was undertaken in the index patient in 2007 including KCNH2, KCNQ1, SCN5A, KCNE1 and KCNE2. The result came back negative. In the meantime next generation sequencing has arrived and a panel including 15 LQTS susceptibility genes was designed and validated for targeted capture and massive parallel sequencing at our