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Is a “Narrow and Tall” QRS Complex an ECG Marker for Sudden Death?

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Introduction

The identification of the various causes for idiopathic ventricular fibrillation and sudden death has often started off from the analysis of peculiar ECG patterns in patients with aborted sudden death or relatives of sudden death victims. The present report follows this line. Recent years have witnessed the genetic identification and electrophysiologic (EP) characterization of inherited arrhythmogenic syndromes associated with a risk of syncope and/or sudden cardiac death (SCD), including Long QT- (LQT), Short QT- (SQT) and Brugada syndrome. Some of these inherited channelopathies proved to be mirror images of each other, e.g. SQTS 1 and LQTS 2 caused by gain and loss of function of I_{Kr} , SQTS 2 and LQTS 1, caused by gain and loss of function of I_{Ks} , SQTS 3 and Andersen-Tawil syndrome (LQTS 7), caused by gain and loss of function of I_{K1} , and Timothy Syndrome (LQTS 8) and the new clinical entity comprised of SQTS and Brugada Syndrome, caused by a gain and loss of function of L-type calcium. ^{1, 2}

The available data suggest that major changes or shifts in EP parameters in either direction can predispose to development of the EP substrate for potentially life-threatening ventricular tachyarrhythmias. The present study examines whether this hypothesis holds true in the case of increased conduction velocity within the ventricles of the heart.

Clinical Presentation

Two cases of unexplained repetitive syncope and one case with exercise-induced pre-syncope in unrelated Caucasian patients are described. Special attention is given to the striking ECG abnormalities that share common morphological features, particularly in the inferior and left precordial leads:

1. Shorter than “normal” duration of the QRS complex

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2. Abnormally high amplitude of the R wave (“tall QRS”) with a steep upstroke of the initial portion, especially evident in the inferior and left precordial leads with which it is associated
3. Non-specific fluctuating ST-T abnormalities in the same leads; both, the magnitude and the displacement of the ST segment are modified by either spontaneous change in heart rate (HR) or by exercise.

Case 1

A 41-year old male with no history of hypertension is presented with a history of paroxysmal atrial fibrillation and recurrent syncope at the age of 15, 17 and 41 years, all occurring at rest. The patient’s father died suddenly and unexpectedly at the age of 42 years without any apparent prodromi (no ECG recording is available).

The resting ECG at a HR of 91 beats per minute (bpm) displayed an inferior axis with normal PR-interval of 152 ms, an averaged QRS duration over the 12 leads of 66 ± 11 ms and the QT interval was 325 ms. (Figure 1) Biphasic (+/-) and inverted T waves were evident in the lateral (V5 and V6) and inferior limb leads, respectively. He displayed high R wave amplitudes in the inferior limb leads. The R/S ratio in aVL was <1 . Further, the Cornell index, a highly specific ECG marker for the absence of LVH, was negative.³ Also, inverted P wave (amplitude >1 mm with a duration of 40 ms) can be noted in lead V1. All relevant ECG parameters are displayed in table 1 including the slew rate of the initial R wave upstroke (mV/ms).

The patient had a normal body mass index (BMI) of 24.16, body surface area (BSA) of 1.89 m² and no thorax deformities such as pectinate chest, scoliosis or kyphosis visible on inspection. Overall, ECG findings are highly suggestive for the left ventricular hypertrophy (LVH). However, both echocardiography and cardiac magnetic resonance imaging (MRI) ruled out LVH. The wall thickness was 4 mm for the right ventricle, 11 mm for the septal and 9 mm for the posterior left ventricular wall. Left heart catheterisation revealed a normal left ventricular function and ruled out coronary artery disease.

The baseline arterial blood pressure was 125/80 mm Hg. During exercise both systolic and diastolic blood pressures were within normal range and HR increased from 91 to 169 bpm. At higher heart rates the J point elevations disappeared and ST segment elevations turned into ST segment depression in the corresponding leads. During the recovery phase, basal J point elevations and pre-terminally negative and biphasic T waves in the inferior and left precordial leads returned. Repetitive 24-hour Holter recordings were unremarkable for brady- or tachyarrhythmic events.

EP study was performed using a transfemoral approach with three quadripolar leads placed in the high right atrium, at the His-bundle and in the right ventricular (RV) apex (RV) and right ventricular outflow tract (RVOT). Sinus node recovery time, AV-nodal conduction and AH- and HV-interval were within normal range. Programmed atrial stimulation revealed an AERP of 240 ms with VERP of 210 ms at 500 ms basic driving cycle length. During programmed stimulation (at three different basic driving cycle lengths of 500, 430 and 370 ms with up to three extrastimuli until refractoriness) ventricular tachyarrhythmias were not inducible.

Molecular genetic screening for SCN5A, KCNH2, KCNQ1, KCNJ2, KCNE1, KCNE2, KCNJ4, KCNJ11, KCNJ12, SCN1B, SCN3B, SUR2A, CACNA1C, CACNB2B and CaCNA2D1 and CX43 did not reveal any mutation. A complete neurological work-up including EEG, cranial MRI and tilt-testing revealed no pathological findings.

An implantation of an internal loop recorder was recommended, but refused by the patient. During a 17-months follow-up, no symptoms have been reported.

The two children of the patient, 13 and 10 year old males, showed normal baseline ECGs with intervals within normal ranges adjusted to their ages. They were both asymptomatic. The QRS intervals were 86 and 71 ms, the QT intervals 356 and 332 ms at rates of 75 and 98 bpm, respectively.

Case 2

The second subject is a 19-year-old male with a history of recurrent syncope both during effort and absence of effort, while sitting and walking at normal speed. The family history was negative for syncope and sudden cardiac death. His resting ECG displayed an inferior QRS axis of $+82^\circ$ (Figure 2). The average QRS duration at a rate of 62 bpm was 80 ± 8 ms (the shortest QRS 74 ms in lead V6). As patient #1, this patient exhibited high R wave amplitudes and a sharp upstroke of the initial depolarisation in leads II, III and aVF as well as in lead V4 and V5. The ECG further displayed J point elevations in leads II, III, aVF and V3-V6. The R/S ratio was <1 in aVL. The Cornell index was negative. The QT interval at 62 bpm was 381 ms. The relevant ECG parameters are displayed in table 1.

The patient had a normal BMI (22.7), BSA (1.75m^2) and no thorax deformities such as pectinate chest, scoliosis or kyphosis visible on inspection. Structural heart disease and LVH were ruled out by the echocardiography and the cardiac MRI. The wall thickness of the right ventricle was 4 mm, the septal wall 11 mm and the posterior left ventricular wall measured 10 mm. The baseline arterial blood pressure was 120/70 mm Hg. During exercise, both systolic and diastolic blood pressure was within normal range. During exercise testing, the patient exhibited a basal HR of 88 bpm which increased to a maximum of 188 bpm. At higher heart rates, the J point elevations disappeared and ST segment elevations turned into ST segment depression in the corresponding leads. During the recovery phase, the J point elevations returned together with preterminally negative and biphasic T waves in the inferior and left precordial leads as observed in patient #1 and #3. During exercise the ECG displays a U wave/ p wave-fusion. This complicates the detection of the end of the T wave to rule out exercise induced long QT syndrome, although the QT interval at rest is normal. However, syncope in this patient occurred also at rest, where QT interval was normal in all recordings obtained. During 24-hour Holter recordings, no brady- or tachyarrhythmias were documented. Intravenous ajmaline challenge was performed to rule out Brugada syndrome. At maximum dosage of ajmaline, no J point elevations in leads V1-V3 could be provoked. The QRS duration slightly increased and the J point elevations remained in leads II, III and aVF but disappeared in leads V4-V6. The T wave turned into a normal morphology with symmetrical, positive T waves.

EP study demonstrated normal sinus node recovery time of 1172 ms, normal AV-nodal conduction, AH-interval (80 ms), and HV-interval (44 ms). Programmed atrial stimulation revealed an AERP of 240 ms, while VERP was 200 ms at 500 ms basic driving cycle length. During programmed stimulation at three different basic driving cycle lengths of 500, 430 and 370 ms using up to three extrastimuli until refractoriness, no ventricular tachyarrhythmias were inducible.

Genetic testing was negative for mutations in SCN5A, KCNH2, KCNQ1, KCNJ2, KCNE1 and KCNE2.

The neurological work-up including EEG, cranial MRI and tilt-testing revealed no pathological findings.

Since there was no electrocardiographic documentation of ventricular tachyarrhythmias, no structural heart disease and the patient had no inducible ventricular tachyarrhythmias at programmed stimulation close follow-up and implantation of an internal loop recorder was recommended. The patient, however, refused a loop recorder. There were two more syncopes

during follow-up. One syncope occurred, stepping out of a tram and one at rest with prodromi of nausea and dyspnea. This syncope was witnessed by his wife who reported a sudden collapse with cold extremities and no palpable pulse. After approximately 30 seconds, he immediately regained full consciousness without post-syncope fatigue.

Case 3

The third case describes an 18-year-old male, who suffered from a collapse with presyncope during a soccer match. The family history was negative for SCD or syncope.

The resting ECG revealed similar ECG abnormalities in the inferior and left precordial leads as previously described in the two other patients. (Figure 3) The ECG displayed an inferior axis. The R/S ratio in lead aVL was <1 . The average QRS duration at a rate of 69 bpm was 78 ± 10 ms, being shortest (63 ms) in lead V4. The QT interval at 69 bpm was 359 ms (QTc 387 ms). All relevant ECG parameters are displayed in table 1.

Both the complete cardiological work-up, including cardiac MRI with special attention to LVH, and the neurological work-up were negative. No cardiac abnormalities or signs of LVH were diagnosed by echocardiography and cardiac MRI. The wall thicknesses for the right ventricle, the septal wall and the posterior wall measured 4 mm, 10 mm and 9 mm, respectively. The BMI was 23.7 and the BSA 1.96m^2 .

The baseline arterial blood pressure was 125/75 mm Hg. During exercise, both systolic and diastolic blood pressures were within normal range.

At baseline, the patients exhibited a basal HR of 70 bpm. During exercise HR increased to a maximum of 169 bpm. At higher heart rates the J point elevations disappeared and ST segment elevations turned into ST segment depression in the corresponding leads. During the recovery phase, the J point elevations returned together with preterminally negative and biphasic T waves in the inferior and left precordial leads as observed in patient #1 and #2.

As in patient #1 and #2, 24-hour Holter ECG was unremarkable for brachy- or tachyarrhythmias.

Sinus node recovery time (914 ms), AV-nodal conduction (167ms) and AH-interval (77 ms) and HV-interval (50 ms) were within normal range. Programmed atrial stimulation revealed an AERP of 200ms. VERP was 200 ms at 500 ms basic driving cycle length. During programmed stimulation at three different basic driving cycle length of 600, 500 and 400 ms using up to three extrastimuli until refractoriness, no ventricular tachyarrhythmias were inducible. Genetic screening is ongoing.

All ECGs from his mother, his father and his two sisters were normal. During a follow-up of 4 months, no symptoms were reported.

Control group

A total of 20 healthy, normal male subjects (mean age 28 ± 8 years) with normal cardiac dimensions and no signs of LVH served as a control for R wave amplitude, slew rate ($\Delta V/\Delta t$) of the initial R wave and QRS duration. It was ensured, that all of the controls had a normal BMI (mean 23.77 ± 3.2), BSA (mean $1.92\text{m}^2 \pm 0.16$) and no thorax deformities such as pectinate chest, scoliosis or kyphosis visible on inspection. The mean values for the different ECG parameters are displayed in table 1.

Discussion

Defects in inter- and/or intraventricular conduction are associated with a widening of the duration of the QRS complex and are a well known contributor to a variety of arrhythmogenic mechanisms. The question is whether a shorter than normal QRS duration (potentially secondary to accelerated inter- and intraventricular conduction) is likely to lead to the development of tachyarrhythmias?

Here we present three cases with the clinical picture of a “narrow (and tall) QRS Syndrome”: a brief QRS complex, tall R waves in the inferior leads limb leads and in the left precordials with a sharp upslope of the initial portion of the QRS complex that might correspond to a fast initial depolarisation (dV/dt), fluctuating repolarization changes in terms of ST segment elevation and depression, together with recurrent syncope, presyncope and one familial SCD.

These observations may represent a new ECG marker of arrhythmia risk. The question arises as to whether the abbreviation of the QRS complex with a steep upstroke of the initial QRS and tall R waves reflects a channelopathy or a disease of cell coupling. There are no relevant systematic data on the limits of “normal” R wave amplitude, other than the Sokolow-Lyon-Index. Moreover, there are no systematic data on the lower limits for QRS duration or for $\Delta V/\Delta t$ of the initial R wave upstroke. One report on normal limits of the electrocardiogram in Chinese individuals by Wu et al. analysed baseline electrocardiograms in 5360 healthy individuals.⁴ In their study the QRS duration, averaged over all leads, was 94 and 96 ms in male individuals of 18–29 and 40–49 years, respectively. The 2nd percentile was 73 ms for 18–29 years and 74 ms for 40–49 years, respectively. The median R wave amplitude in leads V4–V6 were 1.95 mV, 1.71 mV, 1.32 mV for the age of 18–29 years, 1.80 mV, 1.59 mV, 1.21 mV for the age of 30–39 years and 1.75 mV, 1.55 mV and 1.15 mV for 40–49 years, respectively. The 98th percentile for age 18–29 was 3.16 mV, 2.88 and 2.14 mV and 2.86 mV, 2.55 and 1.96 mV for age 40–49, respectively. The median R_{peak} time in lead V5 and V6 was 38 and 37 ms (the 2nd percentile in V5 and V6: 28 and 29 ms). In another series of 79,743 ECG recordings were analysed by Mason et al.⁵ In their study the median QRS duration for males within the age range of 20–99 years was 95 ms with 73 ms being the 2nd percentile and 109 ms the 98th percentile. In the present report the data of the three patients were further compared to 20 healthy Caucasian male controls. The mean QRS-durations were 93 ± 12 ms, 92 ± 12 ms and 91 ± 14 ms in V4, V5 and V6 in the control group. The mean R wave amplitudes were 1.8 mV, 1.9 and 1.6 mV for leads V4–V6. The slew rates at these amplitudes, however, were lower compared to the three patients described here. (for ECG parameters see table 1)

Possible mechanisms

The ECGs presented here could be the early manifestation of left ventricular hypertrophy and although cardiac imaging could not detect increased wall thickness and these striking repolarization changes are most unlikely to be seen in early hypertrophy, it cannot be completely ruled out.

Also, the ST segment elevation at rest can be interpreted as an early repolarization comparable to the findings of Haissaguerre, Bouineau and a Japanese group recently published.^{6–9} It is also known, that in some cases of early repolarization the QRS complex can be abbreviated and the R wave amplitude is increased.

The difference, yet, between the present patients and the ones described from the other authors is, that in our series we observe preterminally negative T waves and that during exercise there is not only disappearance of the J point elevation at higher rates, but occurrence of ST depression, which has not been described by the other authors.

Intuitively, a phenotype of superconduction secondary to either enhanced intercellular communication or boosted depolarisation forces might be expected to be antiarrhythmic because of its association with a shorter wavelength for reentry.

It is, however, possible that it represents the fingerprint of an enhanced sodium channel gain-of-function that could contribute to the development of triggered activity. At least in one patient, where an ajmaline challenge was performed, at maximum dosage of ajmaline the T waves turned into a normal morphology and the J point elevations in leads V4-V6 disappeared. Although this may point to an alteration of sodium currents, the normalization of the T waves can also be caused by an I_{to} -blockade, so that the result of the test is not conclusive for the sodium current hypothesis.

Alternatively, it may be the expression of an abnormal Purkinje fiber network that may promote both, re-entrant and triggered arrhythmias. The ECG manifestation of a shortened QRS duration or accelerated depolarisation could be due to augmented net depolarization forces in the form of intensified inward currents or an improvement in cellular communication secondary to enhanced gap junctional conductance or gap junction density. The changes in repolarization may be a direct reflection of altered conduction velocity or direction or secondary to remodeling caused by altered stress/strain relationships.¹⁰ In the literature, there are no data on shortening of the QRS complexes in the setting of increased density of connexins.¹¹⁻¹⁶ Genetic screening of the two patients failed to uncover any significant alteration in the major ion channel currents that contribute to the ventricular action potential or to mutations in connexin 43 in one patient, the main contributor to the formation of gap junctions in the ventricular myocardium. These initial probes do not rule out the possibility of variations in the promoter of these ion channel genes, nor do they rule out the possibility of multiple copies of genes or chromosomal cross-recombinations.

Another possible explanation for the apparently accelerated conduction may be an increase in the number of Purkinje fibers or the degree of penetration of Purkinje fibers across the ventricular wall. In the normal human heart, Purkinje fibers penetrate only the endocardial one-third of the ventricular wall, whereas in ungulates, they span the full transmural length of the wall. In a canine model, Boineau et al. could elegantly demonstrate that in case of an increased trabecularisation, the Purkinje fibers extend deeper in the left ventricular midmyocardium and lead to a decreased transversal conduction time with shortening of the QRS complex.⁸

The main goal of this article is to bring clinicians' attention to another unusual and striking ECG abnormality that may be associated with increased risk of sudden cardiac death and be used as novel ECG marker for potentially life-threatening ventricular arrhythmia (if proven), rather than claim for the discovery of a new clinical syndrome.

We present these cases with the hope that similar cases could be uncovered and that these will stimulate further research into elucidation of the cause and etiology of this ECG and the associated symptoms.

Conclusion

A short QRS interval with ST segment alterations in the inferior limb and the left precordial leads associated with syncope and a risk of SCD may represent a novel clinical entity. The cause remains presently unknown and may involve altered ion channel activity, enhanced intercellular communication and/or altered distribution of the His-Purkinje network. Future studies will be needed to further elucidate the cause and etiology of this clinical observation.

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Figure 1.

This figure depicts the 12-lead-ECG (upper panel) and the ECG during exercise (lower panel) of patient #1 (paper speed 25 mm/s). There is a slight J-point elevation in lead V5 together with preterminally negative T waves at rest in leads II, III, aVF and V5, V6. During exercise there is ST segment depression in lead II, III and aVF as well as in V5 and V6. Paper speed is 25 mm/s.

Figure 2.
The baseline ECG of patient #2 is shown. The high R waves amplitudes and J-point elevation in the infero-lateral leads are striking. (Paper speed 25 mm/s)

Figure 3.

This figure depicts the resting 12-lead ECG and ECG during exercise of patient #2. In this case there are slight ST segment elevations in leads II, III and aVF together with a J point elevation. The same alterations are clearly visible in leads V3 to V6. During exercise, the patient develops ST segment depression in the corresponding leads with disappearance of the J point elevations. (Paper speed 50 mm/s)

Figure 4.
The baseline ECG of patient #3 is shown with steep and tall R waves infero-laterally with pre-terminal negative T wave in III and biphasic T wave in V5. (Paper speed 25 mm/s)

Figure 5.

This figure depicts the 12-lead-ECG at rest, during exercise and during the recovery phase of patient #3. It displays a similar ST segment morphology and steep R waves at rest with a ST segment depression during exercise in the inferior and left precordial leads as in patient #1. Note that in the recovery phase there are preterminally negative T waves similar to baseline in the left precordial and the inferior leads. The paper speed is 25 mm/sec.

Figure 6.

This figure depicts the example of the steep upstroke of the initial QRS complex. The R peak-interval in this patient for this lead is 29 ms.

Table 1

Electrocardiographic findings

	Patient 1	Patient 2	Patient 3	Control (Means)
PQ interval (ms)	152	166	163	154 ± 25
Mean QRS (ms)	66 ± 11	80 ± 8	78 ± 10	91 ± 14
QTc interval (ms)	408	387	387	375 ± 35
QRS II (ms)	55	77	88	94 ± 14
QRS III (ms)	58	82	90	84 ± 12
QRS aVF (ms)	57	81	86	84 ± 13
QRS V4 (ms)	77	75	63	93 ± 12
QRS V5 (ms)	59	76	77	92 ± 12
QRS V6 (ms)	57	74	75	91 ± 14
R wave II (ms)	2.34	2.44	2.1	1.2 ± 0.4
R wave III (ms)	2.06	2.20	1.31	0.6 ± 0.4
R wave aVF (ms)	2.21	2.34	1.57	0.8 ± 0.4
R wave V4 (ms)	0.6	3.61	2.54	1.8 ± 0.6
R wave V5 (ms)	1.9	2.57	3.07	1.9 ± 0.5
R wave V6 (ms)	2.09	1.99	1.96	1.6 ± 0.5
Slew rate II (mV/ms)	0.08	0.13	0.06	0.03 ± 0.01
Slew rate III (mV/ms)	0.07	0.06	0.03	0.02 ± 0.01
Slew rate aVF (mV/ms)	0.07	0.08	0.04	0.01 ± 0.01
Slew rate V4 (mV/ms)	0.03	0.12	0.06	0.04 ± 0.02
Slew rate V5 (mV/ms)	0.1	0.08	0.07	0.05 ± 0.01
Slew rate V6 (mV/ms)	0.07	0.05	0.05	0.04 ± 0.01