EKG Workshop – Beyond the Basics

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Introduction

The ability to correctly interpret ECGs is a key skill of the modern life insurance medical director. The use of the electrocardiogram (ECG) has been a very valuable risk selection tool in both clinical and insurance medicine for many years. It is easily available, noninvasive, inexpensive, and reproducible, providing a wealth of information related to the diagnosis and prognosis of heart disease.

Traditionally, the process of ECG interpretation has consisted of two steps. The first step is a descriptive analysis which involves a review of the different wave forms, intervals, etc. in order to identify the ECG`s unique findings. The second step is to interpret those specific findings in light of the clinical or insurance medicine context. i.e., we try and determine whether the ECG is normal or abnormal. A normal ECG does not exclude disease. If abnormal, the objective becomes to determine if the abnormality is benign or not. This is best done by considering the context of the ECG recording and by determining what disease process is causing the ECG abnormality as mortality is greatly influenced by whether structural heart disease is present of not.

The objectives of this workshop are to go beyond basic electrocardiography and discuss the interpretation of ECGs representing common underwriting challenges as well as newer less common ECG patterns which have significant mortality implications. The focus will be more on ECG morphology than the clinical features of the underlying structural heart disease.

Since this is a workshop, it will be interactive, conducted in part like an ECG Rounds in the clinical world. However, because of time constraints and the desire to present as many abnormalities that time will allow, it cannot be entirely interactive.

Asymptomatic 42 year old female with an almost flat line in lead II

This is a relatively common technical error. The keys are: 1) recorded lead II shows a (almost) straight line. 2) Recorded lead AVR is identical with recorded lead AVF which is never possible with correct lead connections. In addition, recorded lead I is actually lead III inverted and recorded lead III is the true lead III. Recorded leads AVR and AVF are each actually a half-sized lead III.

The cause of this pattern is a technical error as a result of a lead interchange involving the right leg electrode. The neutral right leg electrode is by convention placed on the right ankle but could be placed anywhere on the body without affecting the ECG waveform. On the other hand, interchange of its cable with the left or right arm cable affects the shape of all precordial leads (because the central terminal is changed) and of all the unipolar limb leads whereas only one of the bipolar leads is unaffected.

This ECG is the most common of the three "flat line" or ground electrode technical errors.

The other two have the following features that were illustrated in the supplementary slides for this workshop:

LA/RL lead reversal:

- Lead I becomes identical to lead II.
- Lead II is unchanged.
- Lead III records a flat line (zero potential).
- Lead AVR approximates to an inverted lead II.
- Leads AVL and AVF become identical.

RA/RL plus LA/LL lead reversal:

- Lead I records a flat line (zero potential).
- Lead II approximates an inverted lead III.
- Lead III is inverted.
- AVR and aVL become identical.
- AVF looks like negative lead III.

Reference: MacKenzie R. "Flat line" in a limb lead. J Insur Med. 2012;43(2):116-9.

A 48 year old man with a pacemaker for congenital heart block

There are P-waves followed by a paced QRS. It is slightly unusual that there is an RBBB pattern. Most pacing catheters are in the apex of the RV and thus will have a negative QRS in V1-V6. Here the RBBB pattern suggests that the pacing wire is in the left ventricle. When there is RBBB pattern in pacing, there are several possibilities: 1) the lead has perforated the interventricular septum into the LV, 2) the lead is the coronary sinus or 3) the lead is epicardial rather than transvenous.

The supplementary slide for this case shows reliable ventricular pacing with a LBBB pattern as a result of the pacing catheter being in its proper location in the RV apex.

55 year old male – past history of possible MI - pre-ex. test ECG

This ECG does not show any evidence of a previous MI. There are minor T wave changes. However, the red flag here is that this is a *pre-exercise test* ECG.

For exercise ECG stress testing, in order to minimize the effect of motion artifact from electrodes on the extremities, peripheral electrodes are placed on the torso. The Mason-Likar ECG lead modification is the most commonly used adaption used for exercise ECG stress testing. In the Mason-Likar ECG lead modification, the wrist electrodes are moved to the medial border of the deltoid, 2 cm below the lower border of the clavicle in the right and left infra-clavicular fossae. The left and right leg electrodes are moved to the anterior axillary line midway between the iliac crest and the costal margin.

Placement of the limb electrodes onto the torso distorts the electrocardiogram causing a rightward shift of the mean QRS axis, a significant reduction in R-wave amplitude in leads I and AVL, and a significant increase in R-wave amplitude in leads II, III and AVF, which may produce a loss of inferior Q waves and the development of new Q waves in AVL. The R-wave amplitude of the chest leads is also altered.

The supplementary ECG with this case, done with the leads in their standard position, shows the missing inferior myocardial infarction. Most high quality exercise labs perform a standard ECG before hooking the individual up with the exercise ECG configuration.

47 year old man with history of previous MI

There are deep, wide Q waves in all three inferior leads, indicating old inferior MI. There are also deep Q waves in V5 and V6 indicating lateral wall involvement.. Most striking are tall right precordial R waves giving a "reverse" type R wave progression indicating true posterior MI.

The applicant could have had several previous individual MIs with occlusion of the artery to the inferior wall, then occlusion of another vessel to the lateral wall. However, it is also possible that the coronary artery supplying this individual's inferior wall was probably large, wrapping around the apex and supplying the lateral wall as well resulting in a large inferior infarction. Inferior MIs are usually smaller and less consequential than anterior MIs; this case may be an exception.

Note the fairly prominent initial r wave in lead AVR. When inferior MI occurs, there is a primary loss of inferiorly directed depolarization forces in the frontal plane and a reciprocal gain in superiorly directed forces. Thus, in inferior infarction, the initial 0.04 second vector tends (sometimes but not invariably) to be displaced superiorly, producing an initial r wave in AVR as is seen in this case. Benign Q waves in the inferior leads, in contrast to the pathologic Q waves of infarction, reflect the orientation of initial forces horizontally and to the left rather than superiorly and to the right. Consequently, when positional Q waves are present in the inferior leads, lead AVR will usually not show an rS type of complex, but show a QS, QR or Qr complex.

Finally, this tracing demonstrates the combination of inferior MI and left anterior fascicular block, both of which may produce left axis deviation. If you drop a line down from the terminal r wave in AVR you will see it comes after the R wave in AVL in keeping with the counter clockwise vector loop of the fascicular block.

Asymptomatic 54 year old male applicant

The rhythm is sinus at 65 beats per minute with a PR interval of 170 msec and a QRS axis of 20 degrees. There are Q waves in leads II, III and AVF. The Q waves in the inferior leads are borderline, but the Q in III is more suggestive of infarction

(more than one third of the R-wave amplitude, with a borderline duration of 0.30 seconds).

Inferior Q waves do not necessarily mean an infarction. Normal Q waves are seen more frequently in young people and when the frontal plane QRS axis is vertical (+70 to +90 degrees) or rightward. Some cardiologists consider Q waves in the inferior leads to be within normal limits if they are not greater than 0.04 seconds in duration. Normal persons may have Q waves with an amplitude up to 4 mm, or up to one third of the subsequent R wave. The Q wave in lead III may also occasionally exceed these limits in normal individuals, but not in leads II and AVF. Width > 0.03 seconds in III and AVF is the most commonly relied upon criteria for the diagnosis of inferior infarction.

The diagnosis of true posterior infarction is based on an abnormal R wave in the anterior precordial leads, however in this ECG there is an inverse progression of R-wave amplitude from V1 to V3 with a normal pattern from V4 to V6 suggesting a lead switch (V1 registered as V3 and vice versa) rather than a posterior MI. The P wave morphology can help to differentiate this rather common pattern of lead reversal. The typical biphasic P-wave morphology of lead V1 can be identified in the tracing labeled as V3.

In summary, this case illustrates the dilemma of inferior Qs in an asymptomatic applicant. The Qs here are borderline, there is no true posterior MI or ST-T changes to help us. There is a tiny initial r in AVR that might favor old MI but this also is borderline. Thus this is a tough call for the medical director.

Reference: MacKenzie R. The challenge of inferior Q waves. J Insur Med. 2007;39(2):126-34.

56 year old male entrepreneur who had chest discomfort one year ago while travelling in Southeast Asia

Borderline sinus rhythm is present at rate of about 57/min. The QRS axis is borderline rightward at about +95 degrees, associated with pathologic QS waves in lead I and aVL (or minuscule r waves in latter). Slight T wave inversions are also present in leads I and AVL (as well as in V2).

Significant Q waves primarily localized to leads I and AVL are usually felt to be indicative of a high lateral MI**.** The rSr' in V1 with normal QRS duration is non-diagnostic. Note that the rightward QRS axis here is due to loss of lateral forces with MI, not due to right ventricular overload or left posterior fascicular block.

The lateral wall of the LV is supplied by branches of the left anterior descending (LAD) and left circumflex (LCX) arteries. Infarction of the lateral wall usually occurs as part of a larger territory infarction, e.g. anterolateral STEMI. There are three broad categories of lateral infarction: anterolateral MI due to LAD occlusion, inferior-posterior-lateral MI due to LCX occlusion and isolated lateral infarction due to occlusion of smaller branch arteries such as the first diagonal branch (D1) of the LAD, the obtuse marginal branch (OM) of the LCX, or the ramus intermedius. Isolated lateral STEMIs are less common. Lateral extension of an anterior, inferior or posterior MI indicates a larger territory of myocardium at risk with consequent worse prognosis.

41 year old man - history of erythema nodosum age 14.

The applicant also has hilar adenopathy. The question here is whether there is evidence of cardiac sarcoid.

The ECG shows borderline resting sinus tachycardia with evidence of right ventricular overload, including a tall terminal R wave in lead V1 as part of a narrow QRS complex with an rsR' morphology and with borderline right axis deviation (about +96 degrees). There are T wave inversions in the right to mid-precordial leads.

In leads III, and AVF, V2-V4, there is notching of the R wave, in leads V6, there is notching of the S wave.. This is called *fragmentation* of the QRS.

The term fragmented QRS complexes (fQRS) usually includes the presence of an additional R wave (R□) or notching in the nadir of the S wave or the presence of > one R□ in two contiguous leads (as illustrated in the supplementary slide).

The presence of fQRS on a routine 12-lead ECG is a marker of a depolarization abnormality secondary to myocardial scar. fQRS is associated with increased

mortality and ventricular arrhythmic events in patients with coronary artery disease and other forms of structural heart disease. In coronary disease, fragmented QRS complexes may indicate the presence of a remote non-Q-wave myocardial infarction (i.e., the equivalent of a Q wave). The presence of fQRS in patients with pulmonary sarcoidosis is associated with cardiac involvement and increased mortality risk.

References:

1. Das MK et al. Fragmented QRS on a 12-lead ECG: a predictor of mortality and cardiac events in patients with coronary disease. *Heart Rhythm* 2007; 4: 1385-1392.

2. Homsi M et al. Fragmented QRS complexes on 12-lead ECG: a marker of cardiac sarcoidosis as detected by gadolinium cardiac magnetic resonance imaging. *Ann Noninvasive Electrocardiol*. 2009 Oct; 14(4):319-26.

38 year old stockbroker – ECG for "age and amount"

This ECG was obtained for age and amount and arrived for interpretation before the attending physician statement. The right precordial leads show poor R wave progression with R wave height unchanged from V1 to V2, then decreased in V3, then replaced by a dramatic QS in V4. There is no evidence of any bundle branch block or accessory pathway.

Accompanying the APS was information and an ECG documenting the occurrence of a large anterior MI 5 years earlier at age 33.

To illustrate normal R wave progression, a slide showing a horizontal (transverse) vector loop is provided. Normally, the loop is transcribed in a counter clockwise direction. Initial forces of septal activation travel anteriorly and from left to right. As the vector loop continues in its counter clockwise direction, the major portion of the LV is activated with forces directed posteriorly and to the left.

As a result the chest leads show an rS in V1 with a steady increase in the size of the R wave towards the left chest and a decrease in S amplitude. V5 and V6 generally show a qR with the R wave in V5 often taller than in V6 due to the

attenuating effect of the lungs. At some point, generally around V3 or V4, the QRS complex changes from a predominantly negative pattern to a predominantly positive pattern with an R/S >1. This is called the transition zone.

With anteroseptal MI, there is a loss of septal forces. Consequently, initial forces will no longer be oriented rightward and anteriorly but are directed immediately posteriorly. Electrocardiographically, QS complexes will be seen since initial forces move away from V1 to V4.

A common cause of PRWP is left ventricular hypertrophy where there is a large increase in LV mass and the main vector becomes larger and more posteriorly oriented. With this comes a corresponding decrease in right and anterior forces. Thus R waves increase in their amplitude in the later chest leads, while in the anterior leads the r wave decreases in height and the S wave increases.

A common technical error is placing the parasternal electrodes V1 and V2 at chest positions superior (cranial) to their normal locations in the 4th intercostal space. Such a departure from proper procedure can potentially yield recorded waveforms that mimic the ECG diagnosis of anteroseptal MI, conventionally defined by QS or Qr complexes (i.e., absence of an initial positive deflection) in the precordial lead V2 (an abnormal finding) and possibly V1 (not necessarily abnormal).

The key tip-off for this technical error involves orientation of the P wave in lead V2 assuming sinus rhythm and the absence of the P wave distorting effects of left atrial enlargement. Because atrial electrical activity begins in the sinus node in the high right atrium from whence it proceeds anteriorly, inferiorly and leftward – the mean P wave vector points anteriorly, inferiorly and leftward. Given that V2 correctly situated in the left 4th intercostal space lies directly in the path of this vector, an upright monophasic P wave is typically registered. On the other hand, when V2 is displaced to successfully higher locations on the chest, the P wave amplitude becomes diminished or isoelectric (flattened). Therefore the clue is the absence of an upright P wave with frank inversion an even more suggestive sign.

References:

1. MacKenzie R. Poor R-wave progression. J Insur Med. 2005; 37(1):58-62.

2. Clark MB. Poor R wave progression revisited. J Insur Med. 2005; 37(4):318-9.

3. Ilg KJ, Lehmann MH. Importance of recognizing pseudo-septal infarction due to electrocardiographic lead misplacement. Am J Med. 2012; 125: 23-27.

49 year old woman who had a severe episode of gastroenteritis after climbing Mount Kilimanjaro in Tanzania

Sinus rhythm, left atrial enlargement, left bundle branch block, a QR pattern in AVR, and relatively large QRS voltage in the precordial leads with small QRS voltage in the limb leads. The overall pattern is that of a dilated cardiomyopathy. LBBB occurs in many conditions involving of the left ventricle and often signifies enlargement of that chamber. When LBBB is accompanied by right axis deviation or a large R wave in AVR, enlargement of both the right and left ventricles is highly probable. Likewise, with or without LBBB, large QRS voltage in the precordial leads with relatively small QRS voltage in the limb leads often indicates dilatation of both ventricles.

The supplementary slide with this case is another example of DCM voltage discrepancy without a LBBB.

40 year old asymptomatic male applicant

Note the abnormally deep dagger-like Q waves in leads II, III, AVF, V5–V6, and prominent R waves caused by septal hypertrophy in V1, V2. In hypertrophic cardiomyopathy, the right or left ventricular free walls or both become thickened. The interventricular septum can also become hypertrophied and can lead to LV outflow tract obstruction. When the septum hypertrophies, normal septal forces that travel left to right through the septum are exaggerated on the ECG because of the enlarged septal mass. Septal hypertrophy can produce larger-than-normal Q

waves in lateral leads I, AVL, V5, and V6 that can mimic lateral wall MI and can result in larger-than-normal R waves in V1 and V2 that mimic posterior wall MI. If the LV free wall is hypertrophied, a QS complex can be recorded in V1, V2, and sometimes V3, which can mimic anteroseptal MI. If the ST segment is not elevated or shows an upward concave elevation and the T wave is upright in the presence of a QS complex in V1or V2, this favors LV hypertrophy. If the ST segment shows convex elevation with an inverted wave, anteroseptal MI is more likely.

Reference: MacKenzie R. Q waves--does depth matter? J Insur Med. 2011; 42(2-4):92-6.

36 year old asymptomatic male college basketball coach

This is a variant of early repolarization (ERP) with 1–3 mm ST segment elevation ending in an inverted T wave in the midprecordial leads along with preserved R waves. Exercise tends to normalize the ST – T changes. This pattern is most often seen in young African American men, a few of whom at other times manifest the typical early repolarization pattern. It is distinctively different from ERP in that the T waves are inverted in these leads while they are upright and tall in ERP. The subsequent supplementary slides review the traditional pattern of ERP followed by an example and then a slide contrasting normal precordial pattern seen in young men with traditional ERP and the less common variety.

In the traditional pattern of ERP, the ECG example shows prominent precordial voltage without other signs of left ventricular hypertrophy. There are J point elevation (V2-V5) and a distinctive J point notching without PR segment deviations or reciprocal changes. The ST segments are elevated with a concave upwards pattern in leads I, AVL, V1-V6. Upright T waves are noted in all the leads except lead III and AVR. In leads V2–V5, the T waves are tall with asymmetric sides, a rounded peak and a wide base (vs the T waves of hyperkalemia, which are tall with a sharp peak, symmetric sides and a narrow base).

The pattern is a relatively common ECG finding. It is present in 1%–5% of the general population with a greater prevalence in men, young adults, athletes and dark-skinned persons, yet it is not rare in women or in older or inactive individuals. It is related to a dominant parasympathetic neural effect on the heart. It is transiently normalized by exercise and tends to disappear with age. Since it was

first reported over 60 years ago, early repolarization has been considered a benign ECG phenomenon. However, clinical interest in the early repolarization pattern has been rekindled recently mainly because of its clinically established association with fatal cardiac arrhythmias, particularly in otherwise healthy individuals with no structural diseases of the heart.

In the slide contrasting the three forms of ST elevation seen in young men:

- Tracing 1 shows normal ST-segment elevation. Approximately 90 percent of healthy young men have ST-segment elevation of 1 to 3 mm in one or more precordial leads. The ST segment is concave.

- Tracing 2 shows the early-repolarization pattern, with a notch at the J point in V4. The ST segment is concave, and the T waves are relatively tall.

- Tracing 3 shows the less common variant that is characterized by terminal T-wave inversion. The QT interval tends to be short, and the ST segment is coved.

References:

1. MacKenzie R. Early Repolarization ECG Pattern – Still a Benign Finding? J. Insur Med 2010; 42:34–40.

2. Tikkanen JT, Anttonen O, Junttila MJ, et al. Long-term outcome associated with early repolarization on electrocardiography. N Eng J Med. 2009; 361:2529–2537.

3. Wang K, Asinger RW and Marriott HJL. ST-segment elevation in conditions other than acute myocardial infarction. N Engl J Med 2003;349:2128-35.

24 year old woman with history of palpitations

Sinus rhythm with tall (Himalayan) P waves (<2.5 mm in lead II and >1.5 mm in leads V1 and V2) and broad P waves (0.12 seconds in the precordial leads) indicating atrial enlargement, predominantly right. The QRS complexes are wide (0.13 seconds) with broad S waves in leads I and V6, broad R waves in lead AVR

and a multiphasic (rSr□s□) configuration of relatively low voltage in the anterior precordial leads. Thus the pattern is that of atypical right bundle branch block.

This ECG is typical of Ebstein's anomaly. Ebstein's anomaly is a rare congenital heart disorder occurring in ≈1 per 200 000 live births and accounting for <1% of all cases of congenital heart disease. The principal feature of Ebstein's anomaly is apical displacement of the septal leaflet of the tricuspid valve from the insertion of the anterior leaflet of the mitral valve by at least 8 mm/m2 body surface area. The ECG is abnormal in most patients with Ebstein's anomaly. It may show tall and broad P waves as a result of right atrial enlargement, as well as complete or incomplete right bundle-branch block. The R waves in leads V1 and V2 are small. Bizarre morphologies of the terminal QRS pattern result from infra-Hisian conduction disturbance and abnormal activation of the atrialized right ventricle.

32 year old male with systolic murmur at the apex

Sinus rhythm 65/min. PR 0.16. QRS 0.10. QT normal. Axis -70 degrees. The ECG is abnormal due to incomplete RBBB, left anterior fascicular block and poor R wave progression. The presence of a heart murmur, IRBBB and LAFB in a relatively young individual raises the possibility of a primum ASD. The primum defect is an abnormality of the endocardial cushion, which is also the origin of the mitral and/or tricuspid valves and the upper part of the interventricular septum. Primum ASDs usually affect the anterior fascicule: LAFB thus points to a Primum ASD (and a normal axis to a Secundum ASD).

The supplementary ECG tracing is suspicious of a secundum atrial septal defect. Atrial septal defects may present in young or middle-age adults (and sometimes even later in life). The ECG with the secundum (common) type ASDs typically shows a right ventricular conduction delay (as seen here) with a vertical to rightward QRS axis (not present). Notching of the R wave peak in one or more of the inferior leads ("crochetage sign") is also often present (up to 73% of secundum ASDs, especially those with large defects and/or shunts). Approximately 35% disappear following repair. ST-T abnormalities in the right to mid precordial leads may be related to the RV conduction delay as well as to right ventricular overload. Tall P waves due to right atrial abnormality may be seen (not present

here). In older individuals, especially those with systemic hypertension or coronary disease, signs of left or bi-atrial abnormality may be present.

The QRs changes in secundum ASDs are due to right heart volume overload rather than a congenital conduction defect. PR prolongation may be seen in both secundum and primum ASDs but is more common in the latter.

LAD in the case of primum ASD is probably a result of anomalous development of the left bundle in the region of the endocardial cushion abnormality. Posterior displacement of the left bundle and hypoplasia of the anterior fascicle have been observed in anatomical studies

40 year old male – Tetralogy of Fallot repair at age 5

The ECG shows sinus rhythm, a P-R interval at the upper limit of normal (0.20 seconds) and a right bundle branch block with an extremely long QRS duration of 0.21 seconds.

Right ventricular outflow is the defining feature of Fallot's tetralogy and must be relieved at the time of operative repair. Repair often involves either causing pulmonic regurgitation as the valvular stenosis is relieved or rendering the valve totally ineffectual as a patch is placed across the valve ring to enlarge the entire undersized right ventricular outflow tract.

As long as there is little or no residual obstruction to right ventricular outflow, patients may tolerate even wide-open pulmonic regurgitation for decades, but not forever. The right ventricle progressively dilates and fails. As that occurs, the tricuspid annulus enlarges, and tricuspid regurgitation places a second volume load on the already struggling right ventricle.

Virtually all patients who have undergone repair of Fallot's tetralogy have right bundle branch block. The larger the right ventricle, the more prolonged the QRS complex is likely to be. Just as a prolonged QRS complex is a poor prognostic sign in patients with left ventricular systolic failure and left bundle branch block, so too is right bundle branch block with a wide QRS complex in patients after operation for tetralogy of Fallot. In both groups death is often sudden.

25 year old asymptomatic male

The case ECG is an example of Type 2 Brugada pattern. This is followed by a slide showing examples of the three types.

Brugada syndrome (BS) is a familial, genetically determined syndrome characterized by an autosomal dominant inheritance in about 50% of cases with variable penetrance. The diagnosis is suggested by a clinical history in a patient with a specific ECG pattern (Brugada pattern). Sometimes, it is necessary to use other ECG findings and methods to confirm the diagnosis.

According to the Second Consensus Conference on Brugada Syndrome, 3 ECG repolarization patterns have been recognized, *Type 1*, which is the only one diagnostic of BS, is characterized by a coved ST-segment elevation ≥2 mm (0.2 mV) followed by a negative or flat T wave. The *type 2* ECG repolarization pattern is characterized by ST segment elevation, which has a "saddleback" appearance with a high takeoff ST-segment elevation of ≥2 mm and either positive or biphasic T wave. *Type 3* has either a saddleback or coved appearance with an ST-segment elevation of <1 mm.

Type 2 and 3 are not diagnostic for BS, but the diagnosis of BS is also considered positive when a type 2 or 3 pattern is observed in >1 right precordial lead under baseline conditions and conversion to the diagnostic type 1 pattern occurs after sodium channel blocker administration. ECG recordings from V1 and V2 leads at higher (3rd and 2nd) intercostal spaces increase the sensitivity and specificity of the ECG diagnosis for the Brugada phenotype.

Consequently, according to the Second Consensus Conference criteria, BS is diagnosed when a type 1 ST-segment elevation is observed in >1 right precordial leads (V1-V3) in the presence or absence of a sodium channel-blocking agent and in conjunction with one of the following events: documented ventricular fibrillation, (self-terminating) polymorphic VT, a family history of SCD under the age of 45 years, presence of coved-type ECG in family members, inducibility of VT with programmed electrical stimulation or syncope. Patients displaying the

characteristic coved-type ECG pattern without further clinical criteria should be considered as having a Brugada ECG pattern and not BS.

In the third slide there is an example of type 1 Brugada pattern. The prevailing rhythm is sinus in origin and there is a left anterior fascicular block QRS pattern. The ECG is notable for ST elevations in leads V1-V3 with a "coved" appearance in V1-V2, associated with slight T wave inversion.

Of note, there is no true complete right bundle branch block (RBBB) in this case, consistent with the Brugada variant which produces a "pseudo" RBBB appearance in the right precordial leads. In contrast to typical RBBB, prominent S waves are not present with the Brugada pattern in V5-V6. The Brugada pattern can also be simulated by a normal variant (early repolarization pattern) in the right chest leads (usually with a "saddle-back" ST appearance). The distinction between normal variants and an actual Brugada abnormality can be difficult.

All the 3 patterns described above may be observed spontaneously in serial ECG tracings from the same patient, as well as "pseudo-normalization" of the ECG. In clinical practice, these ECG fluctuations can make it quite difficult to identify individuals affected by BS and at risk for SCD. In addition to sodium channel blockers, many agents and conditions are reported to unmask a type 1 BS ECG phenotype, including body temperature, changes in autonomic tone and drugs affecting ion channel function, such as calcium-channel blockers, beta-blockers, antiarrhythmic drugs, psychotropic drugs and alcohol or cocaine toxicity.

The most recent consensus review of Brugada ECG patterns has reduced the patterns to two, combining patterns 2 and 3.

References:

1. MacKenzie R. The Brugada Syndrome—An Electrocardiogram With Important Mortality Implications. *J Insur Med* 2001;33:106–109

2. Antzelevitch C, et al. Brugada syndrome: report of the Second Consensus Conference. *Heart Rhythm*. 2005; 2(4):429-40.

3. Leung V, Chow CM. Brugada Syndrome – A Case and a Review of the Literature. *J Insur Med* 2009;41:216–220 .

4. De Luna AB et al. Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report. J Electrocardiol 2012; 45: 433-442.

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46 year old asymptomatic college professor

T waves are inverted in leads V1 to V3 and biphasic in V4. The shape of the T waves is normal, i.e., they are asymmetrical with the first half having a more gradual slope than the second half. The depth of the T waves decreases from V1 to V3.

T wave inversion in V1-V3 or beyond is a normal variant in children under 12 years of age and is known as the juvenile pattern. Such T wave inversion in two or more of the right precordial leads in an adult is known as a persistent juvenile pattern. This variant is present in 1%-3% of the healthy population aged 19 to 45 years and 87% of patients with arrhythmogenic right ventricular cardiomyopathy (ARVC).

Some rules about the direction of the normal T wave can be formulated. The normal T wave generally follows the direction of the main deflection of the QRS complex in any lead. The normal T wave is always negative in lead AVR but positive in lead II. Left-sided chest leads such as V4 to V6 normally always show a positive T wave.

The T wave in the other leads may be variable. In the right chest leads (V1 and V2), the T wave may be normally negative, isoelectric, or positive. In most adults the T wave becomes positive by lead V2 and remains positive in the left chest leads, i.e., it is almost always positive from lead V3 to V6. Furthermore, if the T wave is positive in any chest lead, it must remain positive in all chest leads to the left of that lead. Otherwise, it is abnormal. For example, if the T wave is negative in leads V1 and V2 and becomes positive in lead V3, it should normally remain positive in leads V4 to V6.

The polarity of the T wave in the limb leads also depends on the electrical axis of the heart. With a horizontal axis, the main QRS deflection is positive in leads I and AVL and the T wave is also positive in these leads. With an electrically vertical axis, the QRS is positive in leads II, III, and AVF and the T wave is also positive in these leads. On some normal ECGs with a vertical axis, however, the T wave may be negative in lead III.

The presence of symmetrical, inverted T waves is highly suggestive of myocardial ischemia, though asymmetrical inverted T waves are frequently a non-specific finding. No widely accepted criteria exist regarding T wave amplitude. As a general rule, T wave amplitude corresponds with the amplitude of the preceding R wave, though the tallest T waves are seen in leads V3 and V4.. Also, the T wave progressively becomes less deeply inverted as the patient ages.

Reference: Marcus F. Prevalence of T-wave inversion beyond V1 in young normal individuals and usefulness for the diagnosis of arrhythmogenic right ventricular cardiomyopathy. Am J Cardiol 2005; 95:1070-1071.

21 year old asymptomatic male

There are subtle but important differences in this ECG when compared with the last one. The ECG shows sinus rhythm with normal intervals and QRS axis. There is borderline low limb lead voltage. The QRS shows a narrow rSr morphology in V1 and V2, with T wave inversions in V1-V3/V4 and nonspecific inferior ST-T changes as well.

The individual has ARVC, formerly called arrhythmogenic right ventricular dysplasia (ARVD), and required an implanted cardioverter-defibrillator for recurrent sustained ventricular tachycardia. A variety of ECG findings have been reported with ARVC including: T wave inversions V1-V3/V4 (present here) in the absence of a RBBB, right ventricular conduction delays (narrow rSr only here), wide right precordial S waves (>55ms) in V1-V3, and epsilon waves in V1/V2 (very small amplitude high frequency waves in the ST segment of right precordial leads; not seen here). In addition, ventricular ectopy, especially with a left bundle branch block morphology, may be seen.

Reference: Jain R, et al. Electrocardiographic features of arrhythmogenic right ventricular dysplasia. Circulation 2009; 120: 477-487.

25 year old asymptomatic male

The ECG shows sinus rhythm with a right ventricular conduction delay and right axis deviation, consistent with a right ventricular overload syndrome. There are T wave inversions across the precordium. In addition, there is subtle, but consistent low amplitude notching located on the ST segment immediately just after the end of the QRS in the right precordial leads (V1-2). The latter finding is called an epsilon wave, and it is the electrocardiographic hallmark of arrhythmogenic right ventricular cardiomyopathy (ARVC). It is a highly specific but not sensitive finding with ARVC, reported to occur in about 30%of patients.

ECG changes are usually the first clinical abnormalities recognized in patients with suspected ARVC. Typically these include depolarization abnormalities, such as right precordial QRS prolongation and epsilon waves, and repolarization abnormalities consisting of negative T waves in the precordial leads beyond V2. Depolarization abnormalities reflect delayed and fragmented conduction in the right ventricle, due to the presence of fibro-fatty tissue.