Slide Notes
Advanced EKGs - Concurrent Session
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Introduction

The ability to correctly interpret EKGs is a key skill of the modern life insurance medical director. The use of the electrocardiogram (EKG) 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 EKG 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 EKG`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 EKG is normal or abnormal. A normal EKG 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 EKG recording and by determining what disease process is causing the EKG abnormality as mortality is greatly influenced by whether structural heart disease is present of not.

The objective of this session was to go beyond basic electrocardiography and discuss the interpretation of EKGs representing common underwriting challenges as well as newer less common EKG patterns which have significant mortality implications. The focus was more on EKG morphology than the clinical features of the underlying structural heart disease.

Slide 2

To introduce the presentation, a brief anecdote was shared about my experience with the Reggie Lewis sudden unexpected death claim.

Slides 3 and 4

In 1985, Dr. Irene Ferrer, a cardiologist and medical director at Metropolitan Life Insurance Company in a *Journal of Insurance Medicine* article [Ferrer I, *J Insur Med*, 1985; 16 (2):6-13], reported on a survey 19,734 electrocardiograms obtained in insurance applicants over a five-year period. Roughly 70% were normal and 30% were abnormal. These two slides update this information by dividing the abnormal EKGs into two groups, those that are common with relatively low mortality risk and those that are uncommon but may have higher mortality implications. Examples from each group were shown.

Slides 6 to 13

These slides discuss some of the challenges posed by **inferior Q waves**.

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.

In **slide 7,** there are deep, wide Q waves and negative T waves in all three inferior leads, indicating a recent inferior MI. Note the 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 (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 (**slides 8 and 9**), 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. **Reference:** MacKenzie R. The challenge of inferior Q waves. J Insur Med. 2007;39(2):126-34.

Slides 10 and 11

Asymptomatic 40-year-old male

There is a regular sinus rhythm, with P waves preceding each complex. The PR interval is approximately 0.12 seconds and the QRS duration is also approximately 0.10 seconds. In some leads (I, AVL, V2-V6) the P wave is followed immediately by the onset of the QRS complex with no isoelectric PR segment. The onset of the QRS complex is more gradual than the later portion of the QRS. This is a delta wave. These are the essential features of ventricular preexcitation via an extranodal accessory pathway (Wolff-Parkinson-White syndrome). The delta waves are negative in III and AVF, and positive in V1-V3 which is consistent with a left posterior bypass tract. A prominent QS wave is present in III and AVF, measuring 0.04 seconds in duration. Although these measurements indicate the Q wave is abnormal, it would be incorrect to make a diagnosis of previous inferior myocardial infarction. In preexcitation the initial forces are abnormal because excitation begins from an abnormal location. It is unreliable to infer myocardial infarction from the presence of abnormal Q waves when the Wolff-Parkinson-White pattern is present.

Slides 12 and 13

51 y.o. asymptomatic male hardware store manager

In **slide 12** there are the relatively narrow, but deep and sharply inscribed ("lancet- or dagger-like") Q waves in the inferior and antero-lateral (V4-V6) leads (and a qR in V3) with relatively tall R waves in V1 and V2. The T waves are also inverted in V1 and V2, as well as in I and AVL, and upright in the leads with prominent Q waves. Slight ST segment elevation is present in the inferior leads. Voltage criteria for LVH are met.

These Q waves are typical of those seen in hypertrophic cardiomyopathy when 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.

Inferior MI Q's are usually wider and less deep whereas HCM Q's tend to be narrow and deep. The transthoracic echocardiogram showed classic asymmetric septal hypertrophy (HSM), with a hyperdynamic left ventricle. HCM is an important cause of "pseudo-infarct" Q waves, attributable, at least in part, to increased septal forces. However, the ECG can vary widely from non-diagnostic or normal to showing classical signals of LVH. Absence of ECG abnormalities does not exclude the HCM syndrome, as family studies with affected ECG members have repeatedly demonstrated.
Reference: MacKenzie R. Q waves--does depth matter? *J Insur Med*. 2011; 42(2- 4):92-6.

Slides 14-19

44 year old asymptomatic obese female - old anteroseptal MI?

These slides discuss some of the challenges posed by **poor R wave progression** (PRWP) as illustrated in **slide 14**.

The definition of *poor R wave progression* (PRWP) varies in the literature:

- a common one is when the R wave is less than 2–4 mm in leads V_3 or V_4
- and/or there is presence of a reversed R wave progression, which is defined as:
  R in V_4 < R in V_3 or R in V_3 < R in V_2 or R in V_2 < R in V_1, or any combination of these.

To illustrate normal R wave progression, **slide 15** shows a normal horizontal (transverse) vector loop. 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.

Poor R wave progression is commonly attributed to anterior myocardial infarction, but it may also be caused by left bundle branch block, Wolff-Parkinson-White syndrome, and left ventricular hypertrophy as well as by faulty ECG recording technique. With anteroseptal MI (**slide 15**), 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 (**slide 16**). 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.

Slides 17-21

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 EKG 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.

Slides 22-29

These slides illustrate some of the issues related to **early repolarization** (ER).

29-year-old son of your company's CEO - ok for Everest Expedition?

Slide 22 EKG shows sinus rhythm 60 bpm with normal AV conduction (PR interval .18 sec). The precordial voltage is prominent without other signs of left ventricular hypertrophy. There is J point elevation (V2-V5) and a distinctive J point notching without PR segment deviations or reciprocal changes consistent with what is commonly referred to as the *benign* early repolarization (ER) pattern.

The pattern is a relatively common EKG finding. It is present in 1%–13% 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 transiently normalized by exercise and tends to disappear with age. Several large studies have confirmed that this type of ER carries no negative prognostic implications in the general population. Indeed, because this type of ER is so common in young athletes, some have considered it an EKG sign of good health.

Slide 23 summarizes the classical features. In the traditional pattern of ER, there is often prominent precordial voltage without other signs of left ventricular hypertrophy. There are J point elevation and a distinctive J point notching without PR segment deviations or reciprocal changes. The ST segments are elevated with a concave upwards pattern. Upright T waves are noted in all the leads except lead III and AVR. 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).

Slide 24

36 -year-old asymptomatic. male college basketball coach - silent ischemia?

This slide shows a variant of ERP in a 36-year-old male with 1–3 mm ST segment elevation in leads V2 through V5 ending in an inverted T wave in the midprecordial leads along with preserved R waves. It is distinctively different from classical ER pattern in that the T waves are inverted in these leads while they are upright and tall in the classical ER pattern. 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. The age and clinical presentation distinguish this pattern from acute coronary disease.

Slide 25 contrasts normal precordial pattern seen in young men with classical benign ER and the less common normal variant.

- **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 classical benign 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 normal variant that is characterized by terminal T-wave inversion. The QT interval tends to be short, and the ST segment is coved.

Slide 26

29 -year-old male – screening ECG for Everest attempt. It's your CEO's son – still good to go?

This slide shows a different early repolarization EKG pattern that has been associated with an increased risk of fatal cardiac arrhythmias. Note the differing and widespread J-wave, and ST segment abnormalities. J-waves are seen in the anterior, inferior, and lateral leads. Elevated ST segments are elevated in the same leads, but their contour is either horizontal or down-sloping.

Slide 27

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 2008, a seminal study by Haïssaguerre et al challenged this view by demonstrating an association between ER and an increased risk of idiopathic ventricular fibrillation (VF). Subsequent case reports, case-control studies, and population studies confirmed this association.

Slide 28

To reconcile these apparently contradictory views, it is necessary to first understand the semantic confusion that has arisen around the term ER. The original definition of ER is based on ≥0.1-mV concave ST-segment elevation with or without accompanying J waves (defined as a deflection after the QRS that seems as a late delta wave or a small secondary R wave), in ≥2 anterolateral leads (Figure 1A and 1B). The emerging definition of ER, which was introduced by Haïssaguerre is defined as inferolateral J-wave notching or slurring at the end of the QRS complex (Figure 1C and 1D). Thus, the term ER is used today to describe 2 different EKG morphologies. The benign form of ER is characterized by anterior ST elevation, whereas the potentially malignant form mainly involves inferolateral J-waves followed by a horizontal or downsloping ST segment.

Slide 29

This slide illustrates the estimated cardiac mortality risk associated with the corresponding electrocardiography pattern (highest risk on the top of the pyramid and lowest on the bottom) and also the estimated prevalence of the pattern in general population (width of the pyramid). Importantly, the risk of malignant ventricular arrhythmia seems to vary across different ER patterns. J-waves involving multiple leads, particularly inferior leads, and higher J-wave amplitudes (≥0.2 mV) have been associated with higher risk. Furthermore, a horizontal or downsloping ST segment (i.e, the absence of STE) after the J wave is also associated with higher arrhythmia risk.

Slides 30-32

31-year-old asymptomatic woman with history of an innocent heart murmur

The EKG shows sinus rhythm at about 70 bpm with an incomplete right bundle branch block type pattern (rSR's' morphology in lead V1 and QRS duration =0.11 second). The QRS axis is normal. There is also notching of the R wave in leads III and AVF. ST-T abnormalities are present in leads V1-V4.

The tracing is suspicious of a secundum atrial septal defect. Atrial septal defects may present in young or middle-aged adults (and sometimes even later in life). The EKG 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. A crochetage pattern of the R wave in inferior limb leads is frequent in patients with atrial septal defect, correlates with shunt severity and is independent of the right bundle branch block pattern. Sensitivity and specificity of this sign are remarkably high when it is associated with an incomplete right bundle branch block or present in all inferior limb leads. ST-T abnormalities in the right to mid precordial leads may be related to the RV conduction delay as well as to right ventricular volume 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.

Ostium primum ASDs are associated with endocardial cushion defects with cleft mitral valves and sometimes ventricular septal defects. The EKG classically shows right ventricular conduction delays with left axis deviation (LAD), and sometimes AV conduction abnormalities. 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.

Slides 33-34

32-year-old asymptomatic female computer scientist

The ECG reveals sinus rhythm with the following abnormalities left atrial abnormality (LAA) with a relatively tall R wave in lead V1 and an inverted T wave in that lead, along with right axis deviation (RAD).

The important finding on this ECG is the relatively tall R wave in V1. The differential diagnosis of this finding includes normal, childhood EKG, posterior myocardial infarction (usually with Q's inferiorly), RVH (usually with right axis and T wave inversions in V1-V2), rightward displacement of the heart (e.g., left pneumothorax), Wolf-Parkinson-White with posterior or lateral wall pre-excitation, hypertrophic cardiomyopathy with large septal forces (usually see large lateral Q waves as well), RBBB. In this case, the presence of RAD and anterior T wave suggests RVH as the cause for the tall R in V1, and the concomitant presence of LAA strongly suggests the diagnosis of mitral stenosis. Evidence for LAA here is better seen at higher gain. The P waves are broad (>120 ms in duration) in multiple leads and there is a biphasic P wave in lead V1 with a broad (>40ms) terminal portion. The late P wave forces also go toward lead AVL.

Slide 35

40-year-old male – Tetralogy of Fallot repair at age 5 – now a standard risk?

The EKG 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.