EKG Changes

2.0 Contact Hours

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EKG CHANGES

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Objectives:

After completing this course, the student will be able to:

1. Have a basic understanding of how to properly and effectively assess an EKG.
2. Recognize basic EKG changes and some of their possible clinical correlations.
3. Determine when and how to properly manage tachycardia.
4. Distinguish the EKG changes found with acute MI versus previous cardiac injury.
5. Recognize EKG findings consistent with bundle branch blocks and chamber enlargement
6. Recognize EKG changes consistent with electrolyte abnormalities.

The Basics

Assessing an EKG

When you begin assessing a 12-kead EKG utilize the following steps:

- Ignore aVR
- Determine heart rate.
- Determine the rhythm using the P wave. (ex. Does each QRS have a P wave? Are there P waves? Do the P-waves and the QRS complexes appear related?)
- Measure the intervals (PR interval: prolonged when > .20 sec; QRS complex: wide when > .10 sec; OT interval: prolonged when > ½ of the R-R interval)
- Scan all of the other 11 leads for Q waves, noting which leads do not have a Q wave.
- Check the EKG for R wave progression (ex. Is the R-wave in V1 taller than V2?)
- Check for changes in the ST segment

-Check for T wave changes

Basic Lead Groups

-Inferior leads: II, III, aVF

-Septal leads: V1, V2

-Anterior leads: V2 and V4

-Lateral (left-sided) leads: lateral pre-cordial leads V4 to V6/high lateral leads: I, aVL

EKG Characteristics:

Intervals and Abnormalities

QT-interval

The QT interval is the period on the EKG illustrating the beginning of ventricular depolarization to the end of ventricular re-polarization. This period is considered prolonged when it measures more than half of the R-R interval. However, it is important to note that in patients with tachycardia this "rule" does not apply because the measure of the QT interval has little meaning. When measuring the QT interval, start measuring at the beginning of the Q wave (or the R wave if there is no clear Q wave) and end at the end of the T wave. It is easier to measure if you examine the entire EKG and measure using the lead where the T wave and QRS complexes are clearest. It would also be appropriate to use the lead in which the QT interval appears the longest. The QT interval is measured only in regard to whether or not it is prolonged and not necessarily for the precise time measurement. Common non-cardiac causes of a prolonged QT interval are: medications (type 1a antiarrhythmic agents, tricyclic antidepressants, phenothiazines), hypokalemia, hypocalcemia, hypomagnesemia, stoke, coma, seizure, and intracranial bleeding.

R-wave Progression

In the healthy adult with a "normal" EKG the R wave will progress as it moves from lead V4 to V6. In this case, progression means that the wave increases in size and grows taller. On the other hand, "poor progression" is when the R wave does not increase in size at all or increases very slowly. Poor R wave progression can be related to: LVH, RVH, pulmonary disease, anterior infarct, anteroseptal infarction, conduction defect, cardiomyopathy, and chest wall deformity. However, it may also be a normal finding in some patients, as no two people have the same heart. This is why it is always important to compare the patient's current EKG with a former EKG whenever possible. There is also a chance for poor R wave progression to be present when the EKG leads are misplaced, be sure to check lead placement before proceeding to the treatment phase.

Q-wave/T-wave Inversion

Inversion of the Q waves and/or T waves may be either an abnormal or normal finding depending on the patient. It can be a normal finding when moderate to large Q waves and/or inverted T waves are evident in leads III, aVF, aVL, aVR, and V1. T wave inversion is also most likely a benign finding when it is isolated to lead III, aVF, or aVL and is unlikely to be reflective of ischemic changes when the QRS is negative in the same leads.

ST segment depression

The ST segment is considered "depressed" when it dips downward instead of demonstrating a positive/incline wave. ST segment depression is relatable to multiple clinical pathologies. Ischemia demonstrates symmetric T wave inversion in multiple leads; on the other

hand, asymmetric depression of the T wave is an indicator of cardiac strain. In the case of cardiac strain, the ST segment appears "scooping."

Pericarditis is another disease pathology that can cause ST depression; however, diagnosis should involve three distinct parts: patient history positive for preceding viral illness, physical exam positive for pericardial friction rub, and EKG changes. In this case ST depression in four stages:

-Stage 1: ST segment elevated in almost all leads

-Stage 2: transition phase during which the EKG begins to look "normal"

-Stage 3: ST segment depressed in almost all leads

-Stage 4: normalization phase

Clinical Pathologies

Tachycardia

There are two pathways to managing tachycardia, which pathway is used depends on one important factor, the patient. Whether the patient is hemodynamically stable and/or symptomatic determines how aggressively the tachycardia should be treated. If the patient is NOT hemodynamically stable or is symptomatic for tachycardia then emergent cardioversion is warranted and necessary. If the patient is hemodynamically stable and non-symptomatic then a 12-lead EKG can be used to determine the underlying rhythm. Supraventricular tachycardia (SVT) is likely if the patient has a narrow QRS complex in all 12 leads. If the QRS complex is wide the patient is likely going to be diagnosed with "wide complex tachycardia." SVT is likely related to: sinus tachycardia, atrial flutter (with a ventricular rate of > 150), and atrial fibrillation (rhythm is not regular as with atrial flutter). The most common example of wide complex

tachycardia is ventricular tachycardia, in which case the patient will be symptomatic and warrant immediate cardioversion.

IMMEDIATE REVIEW:
--is the patient hemodynamically stable?
No—cardioversion
Yes--what is the underlying rhythm?

Narrow qrs in all 12 leads = svt (sinus tach, atrial flutter, psvt---use rate to determine difference between: sinus tacy not > 150; a.flutter has ventricular rate of about 150; not a.fib> if the rhythmn is regular)
Wide qrs = wide complex tachycardia (v.tach)

Sinus tachycardia (sinus tach) is defined as a heart rate greater than 100 bpm with an EKG rhythm similar to that of normal sinus rhythm with an R-R interval of less than 0.6 seconds, visible P waves, and a QRS complex that coordinates with each P wave. Sinus tachycardia is often associated with a decrease in stroke volume from a decrease in ventricular filling time and decreased pulse pressure from the low stroke volume. The tachycardia may also be a symptom of a separate underlying clinical pathology such as: increased sympathetic stimulation of the heart from the SA node, fever, and/or cardiac toxicity.

Supraventricular tachycardia (SVT) is defined as a rapid heart rate that is generated when the origin of the electrical signal that "jumpstarts" the heart is the atria or the AV node. Symptoms of SVT include: palpitations, dizziness, shortness of breath, anxiety, chest pain, weakness, and numbness. Treatment for SVT varies widely from physical maneuvers (vagal maneuvers: cause blockage of the AV node of the parasympathetic nervous system, valsalva maneuvers: increase intrathoracic pressure affecting the barorecpetors at the arch of the aorta) to medical management with Adenosine, Diltaizem, or Verapamil and finally cardioversion which

is used when the patient is extremely unstable and/or attempts to use physical maneuvers or medical management have failed.

Infarction and Ischemia

One third of all cardiac infarctions are "silent MIs" meaning that the patient presented without complaint of chest pain. In these cases the patients often complained of dyspnea, changes in mental status, or no cardiac symptoms. A separate 1/3 of patients diagnosed with acute MI had no EKG changes. Athrosclerosis of the larger coronary arteries is the most common anatomic condition associated with decreased cardiac blood flow and causing myocardial infarction (MI). EKGs in patients experiencing acute MI often demonstrate ST elevation, inverted T waves, the presence of Q waves, and reciprocal ST depression. However, it is critical that EKGs always be compared with the patient's previous EKGs to determine whether or not there are any EKG changes specific to the patient.

When a patient experiences an MI the "typical" EKG demonstrates an "A through F progression." During stages A and B the patient has a normal QRS with possible widening of the Q wave. Stage C is the hyperacute phase where the earliest changes of acute MI are present but the broad/peaked T-waves can be very subtle and are easy to miss, especially if the EKG is not checked with one of the patient's previous EKGs. Stage D is the conventional stage in which the P wave changes become more prominent. During stages E and F the Q waves further expand, maximum ST elevation is reached, and the T waves invert. Other possible EKG changes include: ST depression alone, T wave inversion alone, and absent or resolving Q waves. Non-Q wave infarcts are considered incomplete because they are not transmural and with early

interventions such as catheterization and/or stenting they can be easily resolved. But be ware, these MIs also carry a high rate of reoccurrence. Enzymatic elevation is expected in both types of infarcts and when ST and/or T-wave changes are noted without enzyme elevation they are attributed to previous injury or ischemia not an acute cardiac event.

The location of the blockage within the coronary vessels also affects the patient's EKG, see below:

-occluded right coronary artery = acute inferior MI and/or posterior or right ventricular MI

-occluded left main coronary artery = sudden death r/t massive infarct

-occluded left anterior descending = anterior infarct (bundle branch block, second degree AV block)

-occluded circumflex artery = lateral infarct

NOTE: Development of collateral circulation can change area of infarct

Treatment of acute MI is aimed at restoring blood flow to the occluded area. This is often achieved through acute angioplasty (with or without stents) or thrombolytic therapy.

Bundle Branch Blocks

When the patient experiences a bundle branch block (BBB) they often experience changes in three "key leads", leads I, V1, and V6. There are two different types of BBB, a right BBB and a left BBB. There is also an IVCD, or Intraventricular Conduction Delay, which

shows similar changes to a BBB on EKG but has a different pathology. A *RBBB* is demonstrated on EKG by a wide QRS complex of at least .11 seconds and a wide, terminal S wave in leads V1 and V6. A *LBBB* on the other hand has wide QRS of >.12 seconds, an upright, monophasic QRS complex that may have a notch but no prominent T wave, a predominantly negative QRS in V1 and no Q-wave in any of the left sided leads (V1 thru V6). IVCD is often the result of a multitude of clinical pathologies that may resemble a BBB but clearly demonstrate some sort of defect. IVCD can be caused by anything from cardiomyopathy to MI. The QRS complex in IVCD is wide in this case as well but none of the other characteristics of an RBBB or LBBB in any of the three key leads. The case is often determined IVCD if RBBB or LBBB is not evident in ALL THREE of the key leads.

Wolff-Parkinson White (WPW)

WPW is a disease affecting 2 in every 1,000 people. It occurs when there an in extra conduction pathway around the heart for electric activity causing premature ventricular excitation. This facilitates rapid atrial fibrillation and commonly leads to re-entry arrhythmias. EKG findings in patients with WPW may not be evident in all leads but include: widening of the QRS complex, presence of a delta wave, and a shortened PR interval. It is important to carefully examine these EKGs because the delta wave may appear as a Q-wave and can still stimulate an infarction should it fall at the incorrect time. EKG changes in patients with WPW may also mimic changes present with other disease processes. The patient's medical history is crucial in diagnosing WPW, as the EKG may demonstrate non-specific changes.

Left Ventricular Hypertrophy (LVH)

EKG is a poor diagnostic tool for chamber enlargement. However when evaluating the EKG of a patient with suspected chamber enlargement, based on findings from the patients history and physical, look specifically for the following criteria (the patient must be > 35 years old for the criteria to be utilized):

-The deepest S wave can be observed in V1 and V2

-The tallest R wave is in lead V5 or V6 measures > 35 volts

- (or) R wave in VL is > 12 volts

-R wave > 20 volts in any inferior lead

-Deep S wave in lead V1 or V2

Echocardiography is a superior diagnostic tool for chamber enlargement. However, when using an EKG make sure to check for signs of cardiac strain which puts patients at increased risk for LVH. Cardiac strain is evidenced by an asymmetric pattern of ST segment depression and T wave inversion, commonly seen in one or more leads reflective of the left ventricle.

Electrolytes

The majority of electrolyte imbalances will cause evident EKG changes; most notably abnormal potassium levels cause EKG changes and potentially fatal arrhythmias. Below are the common EKG changes seen in patients with hyperkalemia and hypokalemia. The changes are described using the A thru F progression of EKG changes based on clinical pathology (as previously described in the section for Infarct/Ischemia)

-Hyperkalemia:

A: normal EKG

B/C: peaked T waves (T wave will become taller and more peaked as the K increases)

D: P wave decreases in amplitude, PR interval gets longer, QRS widens

E: P wave disappears, QRS becomes sinusoid

F: Ventricular fibrillation occurs

-Hypokalemia: (EKG changes not as reliable as in hyperkalemia)

A: normal EKG

B: flat T wave

C/D: U wave develops, possible slight ST depression

E/F: noticeable ST depression, U wave gets bigger eventually overtaking the T wave leading to Q-U prolongation

Conclusion

Analyzing an EKG is often a tedious task that takes a lot of practice to perfect. However, it is a valuable skill to posses especially in clinical areas that care for high-risk, high-acuity patients. And while EKGs can be a critical diagnostic tool, they can also be misleading. It is still important to obtain an accurate and detailed patient history and physical. The information collected during the patient's exam can be used to "back up" the findings on the EKG and/or prompt the provider to search for other reasons for the EKG changes. It is also important to remember that no two hearts are the same and whenever possible the EKG should be compared

to an old EKG from the same patient. This will ensure that the provider has a good picture of the patient's baseline and can accurately gauge the EKG changes for severity.

REFERENCES

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** As adapted from: Grauer, K. (2001). 12-Lead EKGs a Pocket Brain for Interpretation 2nd Edition