Original Article: How to Determine the Electrical Axis of the Heart

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ABSTRACT

Shows the axis of the heart to propagate depolarization that extends into the heart to constrict the myocardium. In fact, the heart axis shows the direction in which most electrical stimuli move. When you read electrocardiograms, the heart axis indicates the direction of propagation of the stimulus. In addition, the direction of the heart axis indicates many disorders and changes in the heart. Usually at least two derivations are required to determine the axis. Algebraic sum is used to calculate the positive and negative of the QRS complex. For example, if the complex has a long R and a short S, count the number of cells occupied by R on the electrocardiogram paper vertically and count it as a positive digit, and count the number of negative cells as a negative digit. We put and then calculate its algebraic sum. If the biphasic complex is one and its size S and Rare exactly equal, the algebraic sum will be zero, which is why in this derivation the electric force is equal on both sides, so it is at the central point. But if the algebraic sum is a positive or negative numerical derivation, that is, the corresponding derivation line is deviated in the positive or negative direction. To determine the axis, it is usually best to see which of the 6 organ derivatives is biphasic, and then find the derivative perpendicular to it, the best lead being the AVF lead perpendicular to the D1 lead or the AVL lead perpendicular to the D2 lead and the AVR lead It is perpendicular to the D3 lead, the most common being the first. Examining an EKG will show that as the location of the positive electrode in the breast derivatives changes sequentially. The waves also show progressive changes from V1 to V6, so that in derivation V1 most of the QRS complex is negative, but gradually until derivation V6 most of it is positive.

Introduction

EKG papers usually have a small vertical line at the top of the paper every 3 seconds and the number of complexes can be counted in 6 seconds and multiplied by 10 [1-3]. Select one of the R waves, which is located exactly on one of the sides of the large squares, as the R start, and name the bold black lines (small square sides) with the numbers 50, 60, 75, 100, 150, 300, respectively. And keep in mind. In this case [4-6], R was placed on each of

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these lines after the start, that person's heart rate will be the same number [7-9].

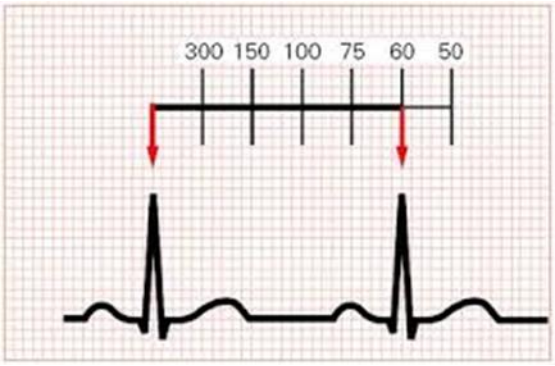

Figure 1: Determining heart rate

Normal sinus rhythm

Each pulsating impulse from the SA node propagates as a wave of progressive depolarization in both atria. The SA node naturally sends its pulses 60 to 100 times per minute, creating a regular sinus rhythm [10-12]. We know that the SA node is called a sine node. Therefore, the rhythm resulting from this node is called sinus rhythm [13-15].

Characteristics of normal sinus rhythm (NSR) include the following:

- ✓ There are P waves.
- ✓ There is a QRS complex after each P wave.
- ✓ Heart rate is 60-100 beats per minute.

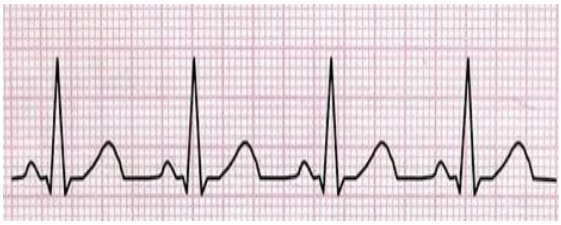

Figure 2: Normal sinus rhythm

Heart rhythm disorders

Dysrhythmias, also called arrhythmias, are disorders of the heart rate and rhythm that are caused by disorders of the conduction system and may lead to obvious hemodynamic changes [16-18]. Causes of cardiac arrhythmias are usually one or a combination of the following disorders of the heart conduction system:

A) Disorder of rhythm production by the SA node [19-21].

B) The location of the pacemaker from the sinus node to another location of the heart [22-24].

C) The appearance of blocks at different points in the path of impulse transmission in the heart.

D) Spontaneous production of abnormal impulses originating from any part of the heart.

Risk factors in dysrhythmias:

Dysrhythmias can be caused by primary heart discomfort or in response to systemic disorders, electrolyte disturbances, or drug intoxication.

Clinical manifestations in dysrhythmias:

Heart palpitations, aching pain, fainting, shallow breathing, swelling of the extremities. Physical findings may include bradycardia or tachycardia, severely irregular heartbeat or rhythm [25], changes in heart rate, sudden onset of symptoms, CHF shock, and angina pectoris [26-28].

Atrial sinus arrhythmias

If the activity of the sinus node changes periodically, the P-P intervals also change, which is called a sinus arrhythmia. This arrhythmia is seen in normal people and does not need treatment [29-31]. This arrhythmia may be related to the time of inhalation and exhalation, i.e. the heart rate increases in the tail and decreases in the exhalation (the reason for this arrhythmia during inhalation and exhalation is that during the inhaled and exhaled periods of the respiratory center In the medulla oblongata, it sends signals to the heart activity control center in the medulla oblongata, and these signals change the frequency of activity of the vagus nerve or the sympathetic nerve of the heart (so the number of beats changes periodically) [32-34]. Other causes of this dysrhythmia include increased vagus nerve

tone, coronary artery disease, myocardial infarction, digitalis intoxication, increased intracranial pressure, or hypertension [35-37].

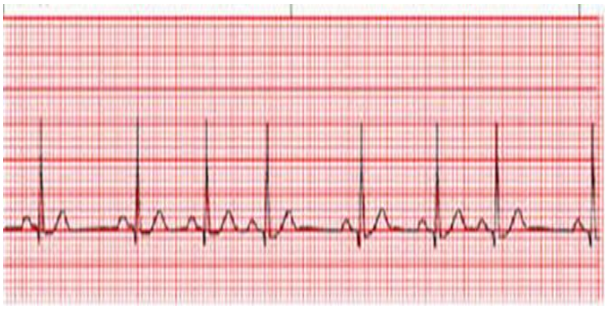

Figure 3: Sinus arrhythmia

In this arrhythmia, there is a variety of P-P intervals of more than 10%.

1- Types of sinus arrhythmias:

Respiratory: There are alternating periods of gradual increase and decrease in P-P intervals.
Ventriculo-phasic: which is seen in complete heart block (sometimes in grade II block) [38-40]. The P-P distances between a QRS complex are shorter than the distances without a QRS complex [41-43].

Sinus bradycardia

Reducing the sinus heart rate to less than 60 beats per minute causes this type of arrhythmia. Sinus bradycardia is a common finding in people [44-46]. In the absence of conduction disturbances and normal intervals on the ECG, resting sinus bradycardia is normal. This phenomenon indicates the adequacy of the cardiovascular system and is common in athletes. Sinus bradycardia may be caused by drugs derived from digitalis [47-49], beta-blockers, or calcium channel blockers. Various diseases such as hypothyroidism, sleep apnea and other conditions that cause hypoxemia can slow the sinus rhythm [50-52].

Characteristics of sinus bradycardia rhythm

- ✓ Beating and rhythm: Beats less than 60 bpm and regular rhythm [53].
- ✓ QRS complex: usually normal.
- ✓ The P wave precedes the QRS and has a fixed shape [54].
- ✓ The P-R distance is usually normal.
- ✓ Conduction: Across the AV atrium, nodules and bundles of hiss and ventricles are normal [55].
- ✓ Treatment: In asymptomatic patients, there is no need for treatment, but in cases where hemodynamics are disturbed, atropine injection of 0.5-0 mg IV every 5 minutes up to a maximum of 2 mg and, if necessary, action for A temporary pacemaker is helpful [56].

Sinus tachycardia

It is normal to increase the number of sinus beats between 100 and 160 with a P wave and a QRS complex. This is a benign rhythm that depends on the individual's clinical condition. This rhythm is a reaction of healthy people to exercise and activity [57-59], but sinus tachycardia in a patient recovering from a myocardial infarction may be a sign of a large infarction and heart failure. Although this rhythm does not require special treatment, it should be considered as it can indicate insufficient function or left ventricular failure [60-62]. In other diseases, sinus tachycardia may be a sign of decompensation. Non-cardiac diseases such as thyrotoxicosis, anemia, and fever can cause sinus tachycardia. Medications such as thyroid hormones, catecholamines, caffeine, and amphetamines can also cause this condition [63-65].

Characteristics of sinus tachycardia rhythm:

- ✓ Rate: 100 to 160 times per minute.
- ✓ Rhythm: regular.
- ✓ P waves precede QRS and have a fixed shape, may merge into a T wave.

- ✓ PR distance is usually normal.
- ✓ The QRS complex is normal [66].
- ✓ Conduction: AV nodes and hiss bundles are normal throughout the atrium.

Treatment

The primary cause must be eliminated. When observing sinus tachycardia, one should look for the underlying factor [67-69]. For example, painkillers, diuretics for heart failure, and aspirin for fever and pericarditis are recommended. However, the use of beta-blockers in patients whose tachycardia is due to hypovolemia or heart pump failure is contraindicated [70-72].

Nursing care

Recording dysrhythmia and patient monitoring and control of hemodynamic parameters.

Sick Sinus Syndrome

Patient sinus syndrome refers to a set of symptoms (loss of consciousness, fatigue, syncope, and congestive heart failure) caused by sinus node dysfunction (SA) and rapidly appearing as sinus bradycardia, sinus stop, or atrial sinus block. And is associated with atrial tachycardia such as SVT, float and atrial fibrillation. The cause of rheumatic heart disease is pericarditis, atrial infarction and in the elderly with unknown cause or hypertension and cardiomyopathy [73-75].

Clinical symptoms

There are intermittent episodes of syncope, confusion, bradycardia, palpitations, and flushing following tachycardia. On the electrocardiogram, the P-wave varies according to the type of rhythm and usually appears with each of the QRS complexes. The PR interval can vary depending on the type of rhythm, but is within the normal range [76-78]. The QRS complex and the T-wave are normal, but the QT interval may vary depending on the type of rhythm [79-81].

Treatment

The treatment of this dysrhythmia is permanent pacemaker implantation and brady and tachyarrhythmias are controlled with medication, which includes the following disorders:

- ✓ Sinus spontaneous bradycardia.
- ✓ Sinus stop or sinus outlet block, atrium.
- ✓ Regular or irregular atrial seizures.
- ✓ Inadequate heart rate response during exercise [82-84].

Premature Atrial Contracture (PAC)

In this disorder, an ectopic focus in the atria produces a current sooner than the next impulse leaves the sinus node. This current is distributed abnormally in the atria and then naturally in the ventricles.

- ✓ Waves: P is an early P wave that is different in shape from other Ps.
- ✓ PR intervals: PR for early P is different from other PR seasons [85-87].
- ✓ QRS width: usually 0.04 to 0.12 seconds.

Treatment

This disorder, like many other disorders, does not need treatment if hemodynamic disorders do not occur, and only identifies and eliminates the causative agents. If the number is high or the hemodynamic status is disturbed, drugs such as calcium channel blockers, beta-blockers, and anti-anxiety drugs are used to treat the disorder.

Acute heart failure (acute pulmonary edema)

The patient develops pulmonary edema rapidly, sometimes referred to as acute pulmonary edema due to an increase in the volume of blood in the lungs [88-90]. The pathophysiology of acute pulmonary edema is similar to that of heart failure. Increased left atrial pressure increases pulmonary vein pressure and

ultimately increases the hydrostatic pressure of the pulmonary capillaries, which results in fluid flowing from the pulmonary capillaries into the interstitial space and alveoli. The liquid mixed with air is expelled through the mouth and nose. Note: The classic sign of pulmonary edema is foamy pink sputum. Due to the presence of fluid in the alveoli, the gas balance is disturbed and air is not able to enter, resulting in severe hypoxemia [91].

Clinical manifestations

Decreased blood flow to the patient's brain becomes increasingly restless and anxious. Shortness of breath, sudden suffocation, cold, wet, gray skin, cyanotic nails, with the development of acute pulmonary edema, anxiety, increased restlessness and the patient suffers from dizziness, rapid and noisy wet breathing, decreased arterial oxygen saturation, bloody fluid It comes out of the patient's mouth.

Diagnosis

By assessing the clinical manifestations of pulmonary congestion, chest X-ray to confirm involvement of the pulmonary veins, sudden onset of left HF signs and symptoms (pulmonary auscultation, absence of JVP, absence of edema in dependent areas). Dry, short cough, complaints of fatigue, weight gain, decreased activity may be early signs of pulmonary edema. Pulmonary edema in the early stages may resolve with relatively simple measures. These include placing the patient in an upright sitting position with the legs dangling, avoiding strenuous activity and emotional stress to reduce left ventricular work pressure.

Conclusion

In order to reduce fluid load, improve left ventricular function, increase respiratory exchange, which includes oxygen therapy, medication, respiratory and psychological support.

A) Oxygen therapy: It is prescribed to relieve hypoxemia and shortness of breath. First with a face mask in case of severe respiratory failure of the endotracheal tube or mechanical ventilation and use of peep. In order to reduce venous return, reduce the movement of fluid out of the capillaries and improve oxygen delivery, oxygen delivery is controlled with the help of pulse oximetry and ABG.

B) Medication: including diuretics and morphine in the amount of 4-5 mg IV by reducing the resistance of peripheral arteries and venous return reduces the pressure of pulmonary capillaries and reduces the volume of fluid in the lung tissue and also reduces the patient's anxiety. The patient should be monitored for severe respiratory distress, hypotension, and vomiting. Naloxone hydrochloride should be prescribed if morphine-induced side effects occur.

For example, furosemide intravenously causes vasodilation and hyperemia in the arteries and reduced blood flow. Before applying the diuretic effect, doctors may prescribe bumetanide (Bumax) and metazone (Microx) instead of forezamide. Sitting, preferably with the legs hanging from the bed, has an immediate effect on reducing venous return, reducing cardiac output, and reducing pulmonary congestion. If the patient is unable to maintain this position, he should be placed in a supine position in bed. Psychological support the patient feels fear and anxiety due to respiratory problems and also has a sense of imminent death. Reassuring him and skillful care and nursing are the main components of treatment. A patient receiving an intravenous infusion of vasoactive drugs will need ECG monitoring and repeated measurements and vital signs.

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