Original Article: Accelerated Junctional Rhythm



James Beki

Sustainable Infrastructure, Department of Medicine, Swinburne University of Technology, Melbourne, Australia



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ABSTRACT

Junctional rhythm occurs when the junctional areas of the AV node become the heart pacemaker instead of the SA node. For example, when SA node activity is slow or AV junction automatics increase. Elusive junctional rhythm occurs if the AV junction discharge rate is between 40-60 times per minute and its causes include patient sinus syndrome, digitalis intoxication, lower MI, rheumatic heart disease and vagus nerve stimulation. Accelerated junction rhythm occurs when the junction rate is 60-100 beats per minute. Digital poisoning, hypokalemia, lower extremity MI heart problems and rheumatic heart disease. The presence of three premature beats of AV junction or more at a speed of 200-60 beats per minute is called junctional tachycardia and its possible causes include digitalis intoxication, ischemia or infarction of the lower or posterior myocardium, congenital heart disease in children and inflammation of the junction AV is after heart surgery.

Introduction

he junction rhythm may be asymptomatic or produce signs and symptoms of decreased cardiac output, and if the heart rate is low, the use of atropine or pacemaker is effective. Verapamil and cardioversion are used [1-3].

Electrocardiographic characteristics of junction rhythms: Rate: In nodal rhythm 40-60 times per minute, in accelerated junction junction 60 to 100 times per minute and in

tachycardia junction 100-200 times per minute [4-6].

- ✓ **Rhythm:** Regular.
- ✓ Wave P: Not seen before or after QRS.
- ✓ P-R distance 0.12 seconds.
- ✓ The QRS complex is usually normal.
- ✓ **Conduction:** Retrograde in the atria and normal in the ventricles.

Nursing measures: Controlling the patient's hemodynamic symptoms, paying attention to the patient's tolerance to arrhythmia,

^{*}Corresponding Author: James Beki (beki.j.u@gmail.com)

controlling the level of electrolytes and digital in the blood [7-9].

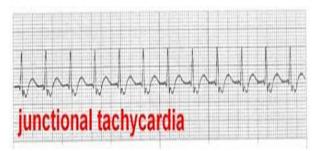


Figure 1: Junctional tachycardia

Ventricular dysrhythmias

Ventricular dysrhythmias originate below the level of AV junction. These dysrhythmias are characterized by ectopic impulses that result from increased myocardial excitability or the phenomenon of reentry [10-12]. Ventricular dysrhythmias are generally more dangerous and threatening than atrial or iunctional dysrhythmias [13-15]. They are often associated with intracardiac disease and usually cause hemodynamic dysfunction. Naturally, impulses travel through the shortest and most effective ventricular pathways, causing a narrow QRS complex [16-18]. If an impulse originating from the ventricles follows an abnormal path through the ventricular muscle tissue, it narrows into a QRS complex [19-21].

Premature Ventricular Complex

Most people have premature ventricular contraction (PVC) and this is a common finding in most ECGs. Because these contractions originate in one ventricle, the activation of the two ventricles is not simultaneous and the QRS complex expands [22-25]. PVCs and other ventricular arrhythmias usually originate from an automated center that separates itself from the surrounding muscle tissue and automatically pulses at a constant rate. The enclosure around them is depolarized and irritated, creating a PVC discharge [26-28]. On the other hand, if these ectopic foci are emptied when the ventricles are

depolarized before the ventricular or depolarization period (during the QRS complex time or T wave), we will not have PVC due to the ventricular excitability [29-31]. Older, fixed-rate pacemakers work according to this pattern. They act at a constant rate and only stimulate the ventricles when they are ready. In this disorder, abnormal. traumatic shock prematurely [32-34]. At the beginning of the complex, there is no P wave and separation of the atrium and ventricle is evident. There is a complete compensatory pause after each premature stroke, meaning that the R-R interval between heartbeats and after PVC is exactly twice the regular R-R interval. In most cases, Pwaves may be inside the T-wave after PVC, which can be due to reverse conduction (early and negative P-wave) or dissociated sinus events. In isolated sinusoidal events, the p-p shape and distance are normal [35-37]. When premature ventricular seizures take many forms, they are called multifocal [38-40].

Common causes of premature ventricular shock

- ✓ Occurs in healthy individuals.
- ✓ Digoxin poisoning.
- ✓ Ischemic heart disease.
- ✓ Left ventricular dysfunction.
- ✓ Electrolyte disorders such as hypokalemia and hypocalcemia, hypoxia, acidosis.

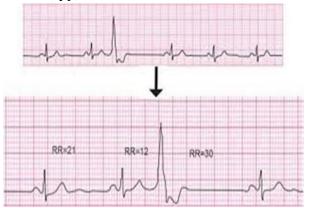


Figure 2: PVC

Types of premature ventricular shock:

- ✓ PVC: Uniform PVCs are of the same shape and have a focus.
- ✓ Multiform PVC: PVCs are multiform and often have multiple foci.
- ✓ Bigeminy PVC: PVC means a normal beat, then a PVC.
- ✓ Trigeminy PVC: means two normal beats, then one PVC.
- ✓ Quadrigeminy PVC: PVC means three normal beats, then one PVC
- ✓ Couplet PVC: means two PVCs in a row

Treatment: Not all abnormal ventricular contractions need treatment. In people without heart disease, premature ventricular shock does not cause sudden death. In case of clinical symptoms, the initial treatment is to eliminate the cause of the arrhythmia [41-43]. Short-term lidocaine treatment and longer-term beta-adrenergic blocking drugs can be effective. PVCs need treatment if they have the following properties and affect hemodynamics [44-46].

- ✓ PVC following acute myocardial infarction.
- ✓ Repeatable PVCs (bi-level, tri-level, quadratic).
- ✓ PVC more than 6 times per minute.
- ✓ PVC polymorphic or multi-focal.
- ✓ RONT PVC.
- ✓ Couplet PVCs [47-49].

A) Lidocaine: It is an antiarrhythmic drug and prevents the activation of sodium channels in heart cells and this drug is metabolized in the liver. The initial dose of this drug is 1-2 mg / kg, which is taken intravenously [50-52].

Nursing care: Record dysrhythmia, control hemodynamic status, reduce patient stress, control electrolyte and digital serum levels [53].

Ventricular Bigeminy: In this arrhythmia, there is an early ventricular shock after each

sinus shock [54-56]. The distance between normal and abnormal impact is usually constant. In most cases, P-waves may be within the T-wave of an abnormal ventricular shock, which may be due to reverse conduction or isolated sinusoidal events [57-59].

Common causes of ventricular dysfunction

May occur in healthy individuals, ischemic heart disease, and digoxin poisoning, left ventricular dysfunction.

Treatment: Lidocaine, procaine amide, quinidine [60-62].

Nursing care: Keep in mind that grounded PVCs are much more dangerous than single PVCs because they can cause a lot of hemodynamic disturbances and are much larger in number and can even be converted to VTac. So monitor the patient very carefully and record any dysrhythmias. Monitor the patient's hemodynamic status [63-65]. Reduce patient stress. Pay attention to electrolyte disorders.

Ventricular tachycardia

This arrhythmia is a condition in which 3 or more ectopic ventricular beats occur with a frequency of more than 100 beats per minute. Pregnant ventricular tachycardia (PVC), or VT for short, is caused by the rapid and sudden activity of a highly excitable ventricular foci (in the speed range of 150 to 250 times per minute) and is characterized by large, sequential complexes. Similar to PVC. In the VT, the SA node continues to pulsate [66-68], but large, giant ventricular complexes hide P-waves, so that these single P-waves are only occasionally visible. Thus the atria and ventricles pulsate independently (a kind of atrioventricular separation) [69-71]. Any tachycardia that has a broad QRS is a ventricular tachycardia (VT), unless proven otherwise. No diagnostic criteria

can differentiate all types of tachycardias with a wide complex [72-74].

Causes of ventricular tachycardia

Ischemic heart disease, especially acute myocardial infarction. left ventricular [75-77], dysfunction long QT interval, medications, electrolyte disturbances. catecholamine sensitivity, may occur apparently healthy hearts, CHF, Dig poisoning, and ventricular aneurysm [78-80].

Symptoms of V.Tach electrocardiogram

- ✓ Atrial rhythm: Depends on the underlying rhythm and is less than ventricular rate.
- ✓ Ventricular rate: 100 to 250 [81-83].
- ✓ Rhythm: Usually regular but rarely irregular.
- ✓ The P wave is not visible and if it appears, it forms as teeth in different places without any connection with the QRS complex.
- ✓ PR distance: not measurable.
- ✓ The QRS complex (equal to or greater than 0.12 seconds) is broad and has an abnormal shape [84-86].

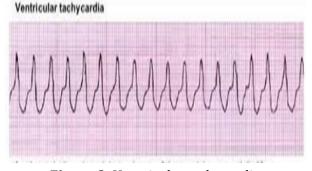


Figure 3: Ventricular tachycardia

Treatment: If the patient is hemodynamically stable, antiarrhythmic drugs can be used. First, lidocaine is used, which is slowly injected intravenously at a dose of 1 mg / kg, and if it is not effective, this dose can be repeated for another 10 minutes. If lidocaine is not effective, injectable procaine amide is given as an

intravenous infusion of 20 mg / min, and the disadvantage is that the injection takes a long time. The complication of procaine amide is hypotension and ORS complexes also flatten, which should be taken care that the QRS width does not exceed 50% of the original width. If procaine amide is not effective, the next choice is injectable amiodarone, which results in bradycardic sinusitis, AV block, and decreased BP. Bertlium has little use in this case, as it often causes a drop in blood pressure, and the effect of amiodarone is better than that. If the drug measures are not effective, cardioversion is performed with DC synchronized shock. In the treatment of ventricular tachycardia with DC shock, it is necessary to pay special attention to the fact that the shock must be synchronized, in which case the leads of the shock monitoring device are attached to the patient's chest so that the device can sense the QRS complexes in ventricular tachycardia. Apply the necessary shock to the patient's chest at the same time as the R-wave and the QRS complex. Treatment of ventricular tachycardia with synchronized shock is called cardioversion [87-89].

General conditions for successful separation of the patient from the ventilator

- A) 12 to 24 hours before separation, the patient should have a stable and acceptable blood pressure without the use of vasopressor drugs.
- B) Low doses of dopamine may be used to maintain renal perfusion.
- C) Low doses of nitroglycerin (intravenously) may be used to counteract the sudden increase in venous blood return due to positive ventilation pressure and to reduce cardiac pressure.
- D) CNS depressant drugs should generally be avoided. Also avoid bronchoconstrictors such as propranolol, neuromuscular blockers, and some antibiotics such as neomycin, gentamicin, and streptomycin, which relax the respiratory muscles [90].

- E) Electrolyte balance is maintained.
- F) For better diaphragm activity, the patient is sitting or half-sitting while exchanging objects.
- G) If there are defecation problems that can cause bloating and electrolyte disturbances, it should be corrected.

Therefore, if the patient is separated from mechanical ventilation, the separation from mechanical ventilation needs careful examination. Upper airway function should be normal in patients whose tracheal tube is removed, but this is difficult to diagnose in tubular patients.

Periods of activity are gradually increased so that the patient remains stable at 4 breaths per minute or less without the need for rest. The CPAP method can then be used before disconnecting the pipe. PSV Usually PSVmax starts at a level that is sufficient for full ventilation support. PSV is adjusted slightly below the pressure the patient needs during the volume cycle. The level of compressive support is then gradually increased to a point where the number of breaths decreases to 5 CmH2O and increases to more than 25 breaths per minute. At this point, alternating periods of high-pressure support are replaced by periods of low-pressure support, to ensure that the muscles change position without causing fatigue in the diaphragm. The gradual decrease in PSV continues until the level of support is sufficient only to overcome the resistance of the endotracheal tube (approximately CmH2O5). At this time, the support can be disconnected and the pipe removed. Balance and imbalance of ventilation and blood flow Ventilation is the flow of gas in and out of the lungs, and blood flow is the filling of the pulmonary capillaries with blood. Adequate gas exchange depends on the adequacy of the ventilation-to-blood ratio. This ratio may vary in different parts of the lung. Factors such as changes in pulmonary artery pressure, alveolar pressure, and gravity may affect blood flow. Airway obstruction, local

changes in compliance, and gravity may interfere with ventilation.

Note: The risk of Fio2 is high for long-term oxygen poisoning.

We set the current volume equal to 15/15~kg / kg. The normal person breathes 6-8 times a day to prevent microatelectasis. This condition can also be applied to a patient under a ventilator. That is, we give 2-5 times equal to the volume 6-8 times per hour of breathing, but to prevent barotrauma, we use this method when the current volume is below 10~kg / kg and the number of breaths is RR / min 7-15. Inspiratory flowrate of 60Lit / min is usually sufficient.

Note: We adjust the oxygen saturation with PIP, PEEP, FIO2 PH, Paco with the number of breaths and the current volume [91].

Ventilation is the starting point of ACV, although SIMV can also be used. We use the current volume of ventilator varicose veins in patients without cardiopulmonary problems according to Rule 12-12. That is, the current jam is 12cc / kg with a number of min / 12. In COD patients, we use the 10-10 rule. That is, the current volume is 10cc / kg with a respiration rate of 10 min.

Note: In ARDS, to prevent barotrauma, the current volume is adjusted in the range of 8cc / kg, and the pressure of the revinum pathway to prevent barotrauma, the necessary pressure applied to the lungs for pulmonary ventilation should be adjusted in amounts above 45 cmH2o. Another index that can be used to prevent barotrauma is Platue pressure.

Peep is the main indication for Peep in cases where Fio2 is affected by pain and cannot provide Pa02 equivalent to 60mmHg or the shunt is estimated to be more than 25%.

Conclusion

Therefore, if the patient is breathing through a tube into the trachea and experiences multiple stridor or aspiration as the tube exits, the upper airway dysfunction or abnormal swallowing mechanism should be considered and a plan for a stable airway established. Coughing when emptying respiratory secretions through suction is a good indicator of the patient's ability to expel secretions. Respiratory thrust and thoracic wall function are measured by the number of breaths, current volume, tail pressure, and vital capacity. Separation index is defined as the ratio of the number of breaths to the current volume (breath per minute per liter). In the prediction of the possibility of temporary removal of the tube is shown to be sensitive and specific.

If this ratio is above 105 when the patient is in the trachea without mechanical support with a tube inside the trachea, the removal of the tube is highly likely to be successful. Inhalation pressure greater than 2030 cmH and critical capacity greater than 10 ml / kg are acceptable performance criteria for chest wall and diaphragm. Alveolar ventilation is generally appropriate when appropriate. Suction should indicate a plan to establish that CO2 output from the blood to maintain arterial pH in the range of 7.25 to 7.40. The establishment of SaO2 above 90% is also achieved when FiO2 is less than 50% and PEEP is maximum 5cmH20. Although many patients do not meet all the criteria for separation, the patient's likelihood of tolerating segregation without difficulty increases as the number of criteria increases.

Many approaches have been proposed in separating the patient from ventilatory support. Isolation with T-section and CPAP is better tolerated in patients who have had short-term mechanical ventilation and do not have respiratory muscle changes. SIMV and PSV, on the other hand, are suitable for patients who have been intubated for a long time and have a gradual change in the shape of the respiratory muscles. Isolation of a piece is done by trying to breathe briefly with a complementary 02.

These efforts usually start at 5 minutes per hour and are followed at one hour intervals. These efforts are increased for 5 to 10 minutes at a time until the patient is able to breathe independently of the device for several hours and then the tube can be removed.

Separation by CPAP method is similar to separation by T-piece, except that the breathing cycles are automatically guided by the device and by CPAP method. Isolation using SIMV is done by gradually reducing the number of mandatory supports in the amount of 2 to 4 breaths per minute along with continuous measurement of blood gas indices and respiratory rate. More than 25 breaths per minute during the reduction of forced ventilation breaths indicates fatigue of the breathing muscles and the need to accompany periods of activity and rest.

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