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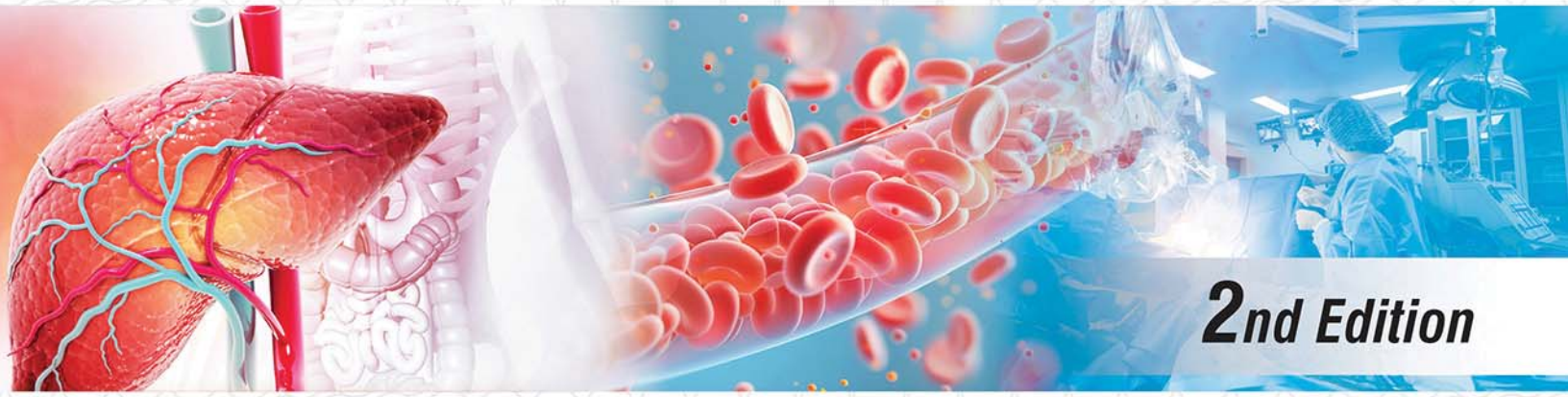

2nd Edition

Adult Health Nursing-I

(Medical Surgical Nursing)

With Integrated Pathophysiology
(Including BLS/BCLS Module & Diabetes Care)

As per the Revised INC Syllabus for BSc Nursing

Sukhpal Kaur

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Contents

Unit I: Introduction to Medical Surgical Nursing

1. **Basics of Medical Surgical Nursing** 3
   Sukhpal Kaur
2. **Roles and Responsibilities of a Nurse in Medical Surgical Settings** 21
   Sukhpal Kaur
3. **Quality Improvement/Quality Quest in Nursing** 26
   Roopa Rawat Singhvi
4. **Inflammation, Infection, Immunity, and Wound Healing** 35
   Sukhpal Kaur, Manjeet Singh, Arti Saini

Unit II: Perioperative Nursing (Intraoperative Nursing)

5. **Preoperative Nursing Care** 55
   Raman Kalia, Sukhpal Kaur
6. **Intraoperative Nursing** 66
   Pooja Shokeen, Vaishakh Tharavath
7. **Postoperative Nursing** 84
   Raman Kalia, Sukhpal Kaur

Unit III: Nursing Care of Patients with Common Signs and Symptoms and Management

8. **Fluid and Electrolyte Imbalance** 95
   Kajal Gupta, Sukhpal Kaur, Manjeet Singh
9. **Acid-Base Imbalance** 109
   Kajal Gupta, Sukhpal Kaur, Manjeet Singh
10. **Common Signs and Symptoms and Management** 118
    Fatima D'silva, Drago Melba Bazel, Cleeta Aniline Pinto, Latha S Upadhyaya
11. **Pain** 154
    Pooja Shokeen, Priyanka Gera
12. **Shock** 168
    Nadiya Krishnan

Unit IV: Nursing Management of Patients with Respiratory Problems

13. **Review of Anatomy and Physiology: Respiratory System** 181
    Tulika Gupta, Chiman Kumari, Daisy Sahni
14. **Assessment and Diagnostic Evaluation of Respiratory System** 187
    Ruchi Saini
15. **Common Upper Respiratory Tract Infections** 202
    Tanveer Rehman, Soundappan Kathirvel
16. **Common Respiratory Disorders** 209
    Ruchi Saini
17. **Disorders of the Pleura: Pleuritis, Pleural Effusion, Pneumothorax, Hemothorax** 250
    Latha T
18. **Thoracic Injuries** 259
    Rita Lakhani
19. **Inhalation Injuries** 267
    Rita Lakhani
20. **Lung Cancer** 273
    Ajee KL, Rita Lakhani
21. **Interstitial Lung Disorders** 290
    Davinder Kaur
22. **Cystic Fibrosis** 297
    Davinder Kaur
23. **Pulmonary Vascular Disorders** 301
    DK Seth, Puneet Kaur
24. **Respiratory Failure** 313
    Prabhjot Kaur

Unit V: Nursing Management of Patients with Disorders of Digestive System

25. **Review of Anatomy and Physiology: Gastrointestinal Tract** 327
    Anjali Aggarwal, Daisy Sahni

26. Assessment and Diagnostic Evaluation of Gastrointestinal System 335
Meenakshi Agnihotri, Arti Saini

27. Disorders of Oral Cavity 351
Ruchika Rani

28. Nutritional Disorders 362
Ishwarpreet Kaur

29. Disorders of Esophagus 371
Prabhjot Saini, Amanjot Kaur

30. Disorders of Stomach and Duodenum 389
Vinay Kumari

31. Irritable Bowel Syndrome 402
Dimple Sandhu

32. Acute Inflammatory and Infectious Bowel Disorders 409
Shine Stephen, Vijay VR

33. Chronic Inflammatory Bowel Disease 418
Vijay VR, Shine Stephen, Ajaya Ghosh RU, Manas Kumar Panigrahi

34. Disorders of Absorption 429
Navreet Kaur

35. Neoplastic Disorders of Intestine 436
Navreet Kaur

36. Structural and Obstructive Disorders 445
Jyoti Kathwal

37. Anorectal Disorders 466
Jyoti Kathwal

38. Gallbladder Disorders 484
Sumanpreet Kaur, Latika Rohilla

39. Liver and Biliary Tract Diseases 491
Tarika Sharma

40. Disorders of Pancreas 515
Navreet Kaur

Unit VI: Nursing Management of Patients with Cardiovascular Problems

41. Review of Anatomy and Physiology: Cardiovascular System 535
Daisy Sahni, Richa Gupta

42. Assessment and Diagnostics of Cardiovascular System 541
Bandna Kumari, Ankita

43. Coronary Artery Disease 563
Harmeet Kaur Kang, Fatima D'silva

44. Disorders of Cardiac Rhythm 580
Ankita

45. Inflammatory Diseases and Infections of Heart 600
Nisha Thomas

46. Disorders of Cardiac Structure 619
Shreedevi Balachandran

47. Other Common Cardiovascular Disorders 634
Bandna Kumari

48. Disorders of Blood Pressure Regulation 671
DK Seth, Puneet Kaur

49. Disorders of Aorta and its Branches 686
DK Seth, Puneet Kaur

50. Disorders of Peripheral Vascular Disease 700
Sarita Ahwal

51. Disorders of Venous Circulation 713
Madhavi Verma

Unit VII: Nursing Management of Patients with Disorders of Blood

52. Physiology of Blood 733
Harshdeep Singh Trehan

53. Red Blood Cell Disorders 740
Sunita Srivastava

54. White Blood Cell and Lymphoid Tissue Disorders 757
Sunita Srivastava

55. Disorders of Bleeding and Coagulation 777
Sunita Srivastava

Unit VIII: Nursing Management of Patients with Disorders of Endocrine System

56. Review of Anatomy and Physiology: Endocrine System 791
Anjali Aggarwal, Daisy Sahni

57. Assessment and Diagnostic Evaluation of Endocrine System 798
Kanika Rai

58. Disorders of Thyroid Gland 806
Monika Sharma, Narinder Kaur Walia

59. Parathyroid Gland Disorders 818
Narinder Kaur Walia, Monika Sharma

60. Disorders of Adrenal Gland 826
Kanika Rai

61. Disorders of Pituitary Gland 836
Neena Vir Singh, Sumanpreet Kaur, Arti Saini

62. Diabetes Mellitus 852
Latika Rohilla, Arti Saini

Unit IX: Nursing Management of Patients with Disorders of Integumentary System

63. Review of Anatomy and Physiology: Integumentary System 879
Chiman Kumari, Daisy Sahni

64. Assessment and Diagnostic Evaluation of Integumentary System 884
Jyoti Kathwal

65. Common Skin Problems 893
Jyoti Kathwal, Manjeet Pal Singh

66. Infections and Infestations of the Skin 903
Jyoti Kathwal

67. Inflammatory Disorders of Skin 912
Soufila KT, Kavita, Tarun Narang

68. Malignant Skin Disorders 929
Shirin Bakshi, Kavita, Tarun Narang

69. Hair and Nail Disorders 937
Vignesh Narayan R, Kavita, Tarun Narang

Unit X: Nursing Management of Patients with Musculoskeletal Problems

70. Review of Anatomy and Physiology: Musculoskeletal System 951
Richa Gupta, Daisy Sahni

71. Assessment and Diagnostic Evaluation in Musculoskeletal Disorders 959
Mandeep Kaur

72. Traumatic Injuries 968
Geeta Thapar, Rashi Sharma

73. Metabolic Bone Diseases 996
Mandeep Kaur

74. Degenerative Disorders: Knee Osteoarthritis 1006
Vishal Kumar, Meenakshi Sharma, Sukhpal Kaur

75. Infectious Disorders (Osteomyelitis and Septic Arthritis) 1014
Prasoon Kumar, Siddhartha Sharma

76. Bone Tumor 1019
Rashi Sharma

77. Disorders of Spine 1031
Vishal Kumar, Aditya Vardhan Guduru, Sanjay Singh Rawat, Aditya Aggarwal

78. Fibromyalgia 1049
Sandeep Patel, Shahnawaz Khan

79. Common Disorders of Foot and Ankle 1052
Sandeep Patel, Shahnawaz Khan

80. Congenital Musculoskeletal Deformity 1058
Geeta Thapar, Sukhpal Kaur

81. Rehabilitation Nursing 1067
Somya Saxena, Anchin Saharan

Unit XI: Nursing Management of Patients with Communicable Disease

82. Respiratory Infections 1085
Monika Pebma, Kapil Goel

83. Surface and Intestinal Diseases 1092
Monika Pebma, Kapil Goel

84. Zoonoses and Arthropod-borne Infection 1101
Kapil Goel, Monika Pebma

85. Sexually Transmitted Diseases 1112
Kapil Goel, Monika Pebma

INC Modules 1119
Objective Structured Clinical Examination (OSCE) 1121
Annexures 1131
Appendix 1163
Multiple Choice Questions 1169
Index *1177*

CHAPTER 44

Disorders of Cardiac Rhythm

Ankita

"His whisper was the softest sound I ever knew, which seemed to bring the loudest heartbeat."
—Dominic Riccitello

Learning Objectives

After going through the chapter, the learner will be able to:

- Enlist the indications of doing an electrocardiograph.
- Enumerate the interpretation of ECG.
- Distinguish between normal and abnormal cardiac rhythm.
- Elaborate on the basic plan of action.

Key Terms

- **Arrhythmia:** The irregular or abnormal rhythm. Arrhythmia can be fatal or non-fatal.
- **Bradycardia:** The heart rate <60 beats/min.
- **Complex:** Combination of multiple waves joined together, e.g., QRS Complex.
- **Depolarization:** Change in the cell membrane from negative to positive. This usually corresponds to the contraction of the heart.
- **Electrocardiograph:** The graphical representation of electrical activity of the heart.
- **Heart rate:** The number of times a heart beats in a minute.
- **Interval:** The time between two specific events, e.g., PR interval, QT interval, and RR interval.
- **Isoelectric line:** The baseline in ECG in which the amplitude remains constant throughout.
- **Repolarization:** Restoration of difference in the charge between the inside and outside of cell membrane. This also corresponds to the relaxation of the cell membrane or resting of the membrane potential.
- **Rhythm:** Any regular repeated or recurring symmetrical pattern.
- **Segment:** The length between two specific points on ECG. The specific points should be at a baseline amplitude, e.g., PR-segment and ST-segment.
- **Sinus:** Any electrical stimuli originating from SA node of the heart.
- **Tachycardia:** The heart rate >100 beats/min.

INTRODUCTION

Electrocardiography (ECG) is the graphical representation of the electrical activity of the heart. Though many advance technologies have emerged for diagnostic evaluation of cardiovascular disease still electrocardiograph remains the primary test to rule out rhythm abnormalities and detect ischemia and abnormalities related to conduction system of the heart. The ECG also adds up greater value to evaluate the impact of antiarrhythmic therapy, cardiomyopathy, and pericarditis.

The gold standard approach to any cardiac disorder is electrocardiogram. The current chapter first elaborates on the normal sinus rhythm and then a detailed description of arrhythmias.

CARDIAC ARRHYTHMIAS

Arrhythmia is any rhythm that is not normal sinus rhythm with normal atrioventricular rhythm. Normal sinus rhythm is a rhythm that originates from the sinus node (i.e., from

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the primary pacemaker of the heart). As the conduction in the heart proceeds from the sinoatrial (SA) node to the term points Purkinje fibers, the events are seen as waveforms in the electrocardiograph. The progression of the conduction indicates that atrial contraction followed by a ventricular contraction can be seen as P, Q, R, S, T, and sometimes U waves. The amplitude and duration of these waveforms indicate that major events such as atrial and ventricular contraction and relaxation are occurring accurately.

Precise decision is required to rule out arrhythmias and this may take time even for the experts in the field.

The arrhythmias can originate from:

- **Atrial origin**
- **Atrioventricular (AV) junction**
- **Ventricular**

ATRIAL ORIGIN

- Supraventricular tachycardia (SVT)
- Atrial fibrillation
- Atrial flutter
- Atrial tachycardia
  - Ectopic focus (enhanced automaticity)
  - Interatrial or sinoatrial re-entry
- Atrial extrasystoles (atrial premature beats)

AV JUNCTION

- Atrioventricular nodal re-entrant tachycardia
- Atrioventricular re-entrant tachycardia
- AV junctional rhythm (enhanced automaticity)
- AV junctional extrasystoles

VENTRICULAR

- Ventricular tachycardia
  - Sustained
  - Non-sustained
- Ventricular fibrillation

NORMAL SINUS RHYTHM

Normal sinus rhythm is the rhythm originating from SA node of atrium and contains PQRST components. This is the default rhythm of the heart. In a normal sinus rhythm, the impulses originate from the SA node and follow the path of conduction system of the heart. The normal sinus rhythm consists of a heart rate of 60–100 bpm with a regular rhythm; normal R-R interval (i.e., 3–5 large boxes). The P:QRS ratio should be 1:1 which means every P wave followed by QRS complex. The PR interval should be of 0.12–0.20 second duration and constant. There should be constant QRS complex with 0.08–0.12 second duration. The above-discussed components make a rhythm normal sinus rhythm **(Fig. 44.1)**.

SINUS BRADYCARDIA

When a heart rate is less than 60 beats/min, it is called bradycardia **(Fig. 44.2)**. It may occur for many reasons and may or may not affect the cardiac output. In the severe condition, it may severely affect and reduce cardiac output and may lead to asystole.

Bradycardia may originate from SA node. Sinus bradycardia is sometimes a normal condition when the body is at rest and does not require much oxygen or in cases of athletes with an efficient body to meet body demands even with low cardiac output. Other factors may include some medical conditions (sinus node disease, hypothermia, hypoxia) or drug-induced (e.g., digitalis, beta-blockers, or calcium channel blockers).

As the main impulse production is initiated from SA node, hence, at times when failure of SA nodes occurs the beats are produced from AV node. The AV node has intrinsic rate of about 40–60 beats/min, hence low rate is produced at the time of failure of SA node.

Causes

Various causes of sinus bradycardia are enlisted in **Table 44.1**.

Clinical Features of Bradycardia

The clinical presentation of sinus bradycardia has a heart rate of <60 beats/min. The person with sinus bradycardia may present with a weak pulse and low blood pressure. As sinus bradycardia progresses there is a decrease in cardiac output which may cause syncope or lightheadedness.

Diagnosis

The diagnostic evaluation involves history-taking about signs and symptoms. The physical examination may involve listening to the heart rate. Counting the heart rate for a complete 1 minute gives an accurate measurement. The gold standard for diagnosis is to conduct an electrocardiograph after identification of a slow pulse on physical examination

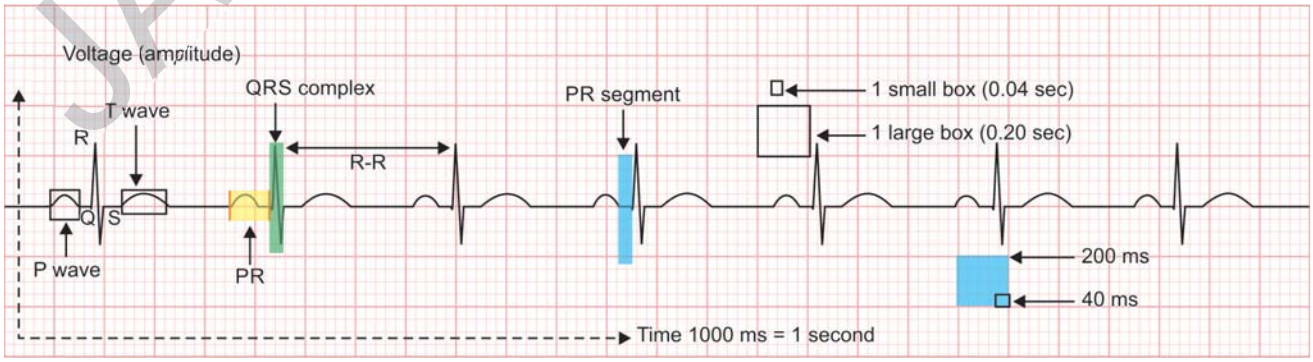

Fig. 44.1: Description of ECG waveform. The large boxes are of 0.04 second in duration; however, the small ones are 0.04 second in duration. Each P wave is followed by QRS complex in 1:1 ratio.

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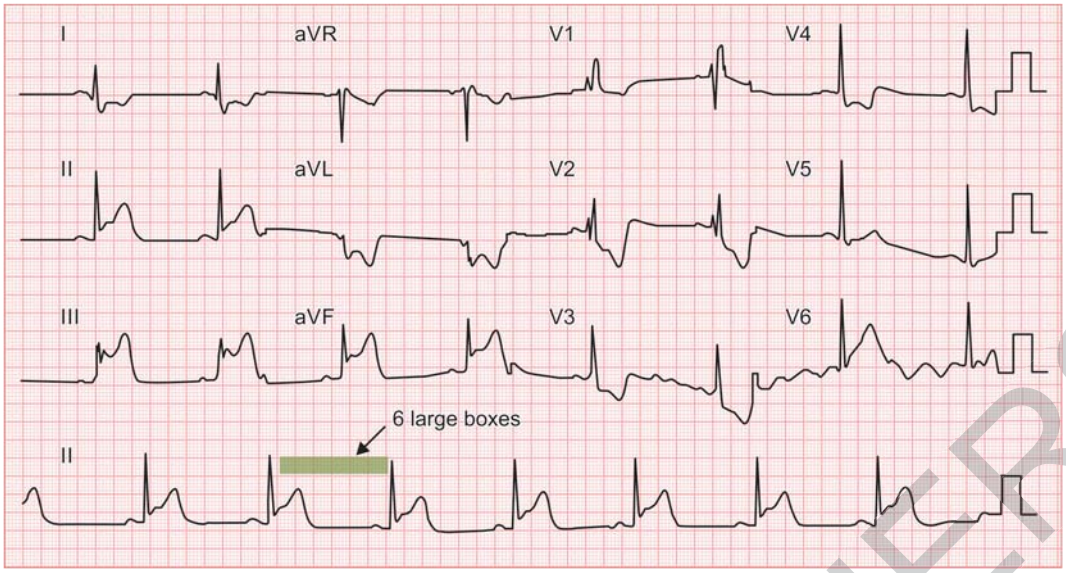

Fig. 44.2: Sinus bradycardia.

Rate: Count the large boxes between R-R intervals in the figure. There are six large boxes, thus 300/6 = 50 bpm.
P wave is followed by QRS complex.
When the rhythm has all the components of normal sinus rhythm but rate is <60 bpm, then it is called sinus bradycardia.

TABLE 44.1: Causes of sinus bradycardia.

Pharmacological	*Nonpharmacological*
• Beta-blockers • Digoxin • Calcium channel blockers • Amiodarone • Organophosphate poisoning • Lithium • Verapamil • Procainamide • Quinidine	• Normal while sleeping • Vagal stimulation/increased vagal tone • Inferior wall MI • Hypothermia • Hypothyroidism • Electrolyte imbalance; hyperkalemia, hypermagnesemia • Myocarditis/pericarditis • Ischemic heart disease/chronic ischemia • Carotid sinus hypersensitivity • Cardiomyopathy • Trauma • Idiopathic degenerative disorder • Anorexia nervosa

(MI: myocardial infarction)

or heart rate less than 60 bpm. ECG leads along with cardiac monitor should be applied to assess for further deterioration.

Treatment

The treatment involves a step-wise approach. Attach the cardiac monitor to assess the rhythm. Asymptomatic bradycardia does not require any treatment. Treating and correcting the underlying cause can improve the condition. In the patients who are hemodynamically unstable, administering injection atropine (1.0 mg IV push, repeat in 3–5 minutes with a maximum dose of 3 mg) is considered a first action. If IV atropine therapy fails, IV dopamine or epinephrine are preferred. Sidewise it is necessary to rule out the overdose of calcium channel blockers or beta-blockers, if present then to administer IV glucagon (3–10 mg IV bolus) followed by titration with continuous infusion of 3–5 mg per hour. At failure of medical treatment, temporary cardiac pacing is done. The ACLS guidelines can be followed to manage the condition.



NURSING CONSIDERATIONS

- Thorough physical assessment of the patient, assessment of the vital signs.
- Calculate heart rate carefully before administering the drugs which may affect heart rate, e.g., Beta-blockers, calcium channel blockers
- Notify physician if the patient has the following: heart rate <60 beats per minute, chest pain, or shortness of breath.
- Watch for signs of deterioration.
- Educate the patient about the signs of deterioration and when to seek for help.

Management

The algorithm for the management of bradycardia is shown in **Figure 44.3**.



NURSING MANAGEMENT

- Acute chest pain related to bradycardia/decreased heart rate:
  - Assess the level of pain by using pain scale.
  - Provide comfortable position, e.g., Fowler's or semi-Fowler's position.
  - Treat the underlying cause causing bradycardia
- Ineffective tissue perfusion/decreased heart rate:
  - Assess for oxygen saturation using pulse oximeter or cardiac monitor.
  - Provide comfortable position.
  - Observe for discoloration of skin or nail beds or of mucous membrane.
  - Provide supplemental oxygen if saturation is less than 94%.
- Decreased cardiac output/sinus bradycardia:
  - Assess the heart rate.

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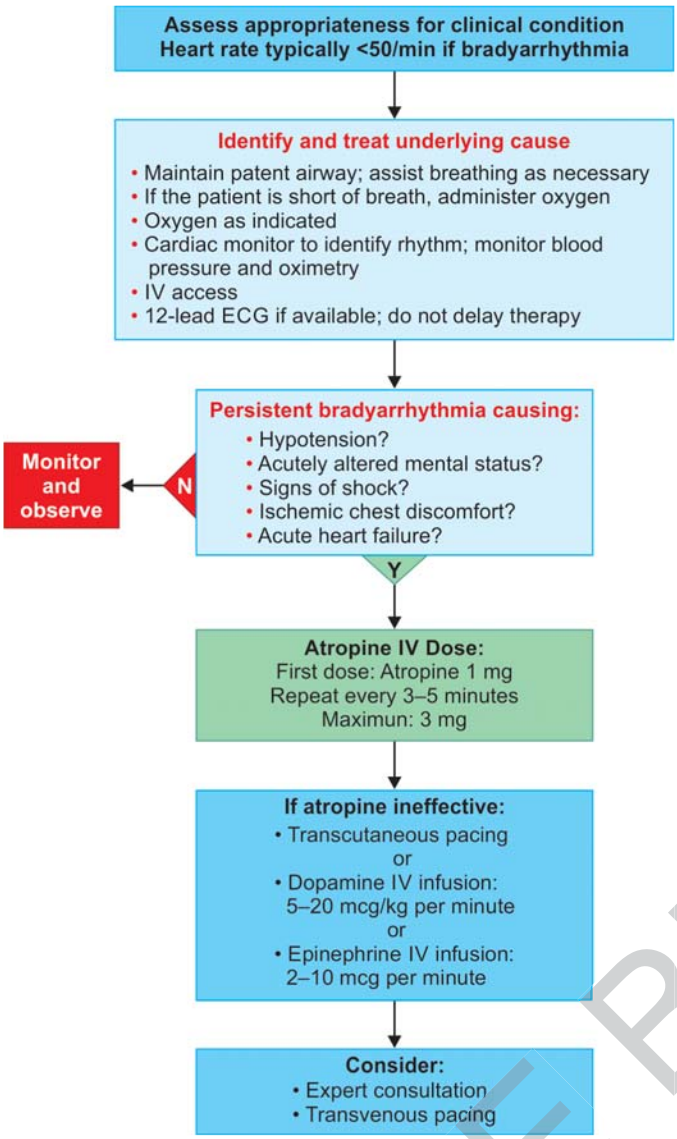

Fig. 44.3: Management of bradycardia.

- Limit physical activities to restore energy and reduce cardiac overload.
- Maintain intake and output to reduce cardiac overload.

❖ Activity intolerance/fatigue/reduced cardiac output:
- Assess the capacity of activity of daily living.
- Plan rest with balanced physical activity for the patient to prevent fatigue.
- Assist in physical activities of daily living to reduce fatigue.

❖ Impaired gas exchange/reduced cardiac output:
- Assess the respiratory rate or any abnormal breathing pattern, depth and effort, watch for use of accessory muscle, nasal flaring.
- Assess for oxygen saturation using pulse oximeter or cardiac monitor.
- Auscultate for diminished breath sounds or presence of any additional breath sound.
- Assess for patients' mental status for restlessness, agitation, and confusion.
- Observe for discoloration of skin or nail beds or of mucous membrane.

SINUS TACHYCARDIA

A condition with increase in heart rate >100 beats/min is known as **tachycardia (Fig. 44.4)**. As the heart rate increases, there is increase in the myocardial oxygen demand and consumption and ultimately it affects the cardiac output of the person. When the tachycardia arises at the sinus level, it is termed sinus tachycardia. The sinus tachycardia can be due to increased sympathetic nervous system stimulation, e.g., increased activity/vigorous activity, exercise, stress, fear or anxiety, fever, hypoxia, or shock.

The narrow complex (narrow QRS complex) tachycardia arises above the ventricles and is supraventricular tachycardia. However, wide complex tachycardia arises below the bundle of His.

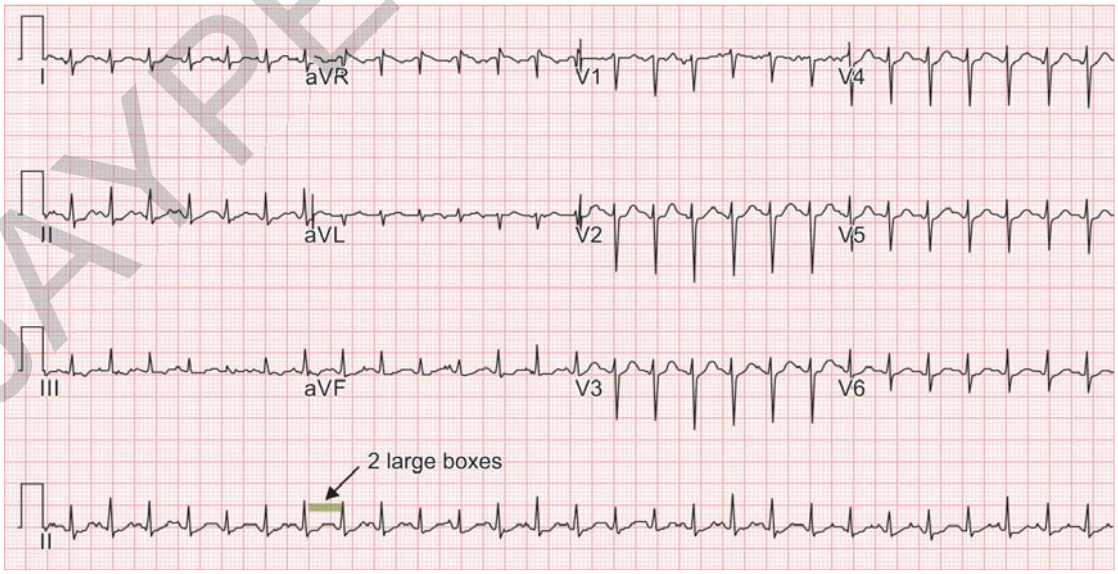

Fig. 44.4: Sinus tachycardia.

Rate: Count the large boxes between R-R interval, in the figure. There are six large boxes, thus 300/2 = 150 bpm.
P wave is followed by QRS complex.
When the rhythm has all the components of normal sinus rhythm but rate is >100 bpm then it is called sinus tachycardia.

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Causes

- Fever
- Fluid deficient
- Shock/hypotension
- Anemia
- Hypoxia
- Acute coronary ischemia or myocardial infarction
- Pain
- Anxiety
- Hyperthyroidism
- Chronic pulmonary disease
- Pheochromocytoma
- Pulmonary embolism

Clinical Features

There are no direct symptoms of sinus tachycardia. The patients may report symptoms of palpitations (i.e., hearing/feeling own heartbeat). Tachycardia can be a symptom attributed to associated conditions (e.g., fever or pain). Persistent tachycardia can lead to incomplete ventricular filling and results in poor cardiac output. Decreased cardiac output causes increased oxygen consumption in the myocardium and decreased coronary blood flow. Persistent and prolonged tachycardia may result in symptoms such as dyspnea, angina, dizziness, syncope/presyncope, or chest discomfort.

Diagnosis

It is important to differentiate between atrial and ventricular tachycardias. The initial step of evaluation involves complete history-taking for symptoms such as palpitations and chest pain.

Treatment

Treating the underlying etiology can help to attain normal sinus rhythm. Understanding and evaluating the underlying cause is important to start the treatment. The treatment usually starts with rate control therapy. A controlled rate can help to differentiate between atrial and ventricular arrhythmias. The rate control therapy involves the use of beta-blockers. Beta-blockers mostly require high dose for significant heart rate control, e.g., Metoprolol 50 mg, with upward titration for rate and symptom control.

For symptomatic inappropriate sinus tachycardia, ivabradine (5–7.5 mg twice) is recommended with or without beta-blockers. In patients with symptomatic inappropriate sinus tachycardia with failed therapeutic treatment, radiofrequency catheter ablation is tried.



NURSING MANAGEMENT

- Ineffective tissue perfusion related to inadequate ventricular filling:
  - Assess for blood saturation using pulse oximeter or cardiac monitor.
  - Observe for discoloration of skin or nail beds or of mucous membrane.
  - Provide supplemental oxygen if saturation is <94%.
  - Provide comfortable position to promote effortless breathing.
- Risk for decreased cardiac output/disease condition:
  - Assess the heart rate.
  - Limit physical activities to restore energy and reduce cardiac overload.
  - Maintain intake and output to reduce cardiac overload.
  - Administer prescribed medication for the patient.
- Activity intolerance/fatigue/decreased cardiac output:
  - Assess the capacity of performing activities of daily living.
  - Plan rest, balanced physical activity for the patient to prevent fatigue.
  - Assist physical activities of daily living to reduce fatigue.
  - Assess strength to perform range of motion exercises.
- Impaired gas exchange/pulmonary embolus/thrombus:
  - Assess the respiratory rate or any abnormal breathing pattern, depth and effort, watch for use of accessory muscles, nasal flaring.
  - Assess for oxygen saturation using pulse oximeter or cardiac monitor.
  - Auscultate for diminished breath sounds or presence of any additional breath sound.
  - Assess for patients' mental status for restlessness, agitation, and confusion.
  - Observe for discoloration of skin or nail beds or of mucous membrane.
- Risk of ineffective cerebral tissue perfusion related to reduced cardiac output:
  - Assess the saturation level of the patient.
  - Check for signs of discoloration of skin and nail beds.
  - Assess for quality of pulse.
  - Assess for any changes in the mental status or for altered sensorium.
  - Monitor for higher functions such as speech if the patient is alert.



NURSING CONSIDERATIONS

- Assessment of hemodynamic monitoring and continuous rhythm assessment.
- Recognize for signs of complication and symptoms like chest pain, anxiety and palpitations.
- Educating patient for signs of deterioration and when to see the doctor.

Management

Management of tachycardia as per ACLS guidelines is shown in **Figure 44.5**.

Sick sinus syndrome is a dysfunction of the SA node leading to persistent sinus bradycardia, sinus pauses or arrest, and sometimes alternating bradycardia–tachycardia episodes. It commonly occurs due to age-related fibrosis, ischemic heart disease, or drug effects. Patients present with dizziness, fatigue, palpitations, or syncope. Management involves correcting reversible causes and treating with a permanent pacemaker in symptomatic cases.

ATRIAL FIBRILLATION

It is one of the most common types of arrhythmias. The features are associated with an irregularly irregular rhythm

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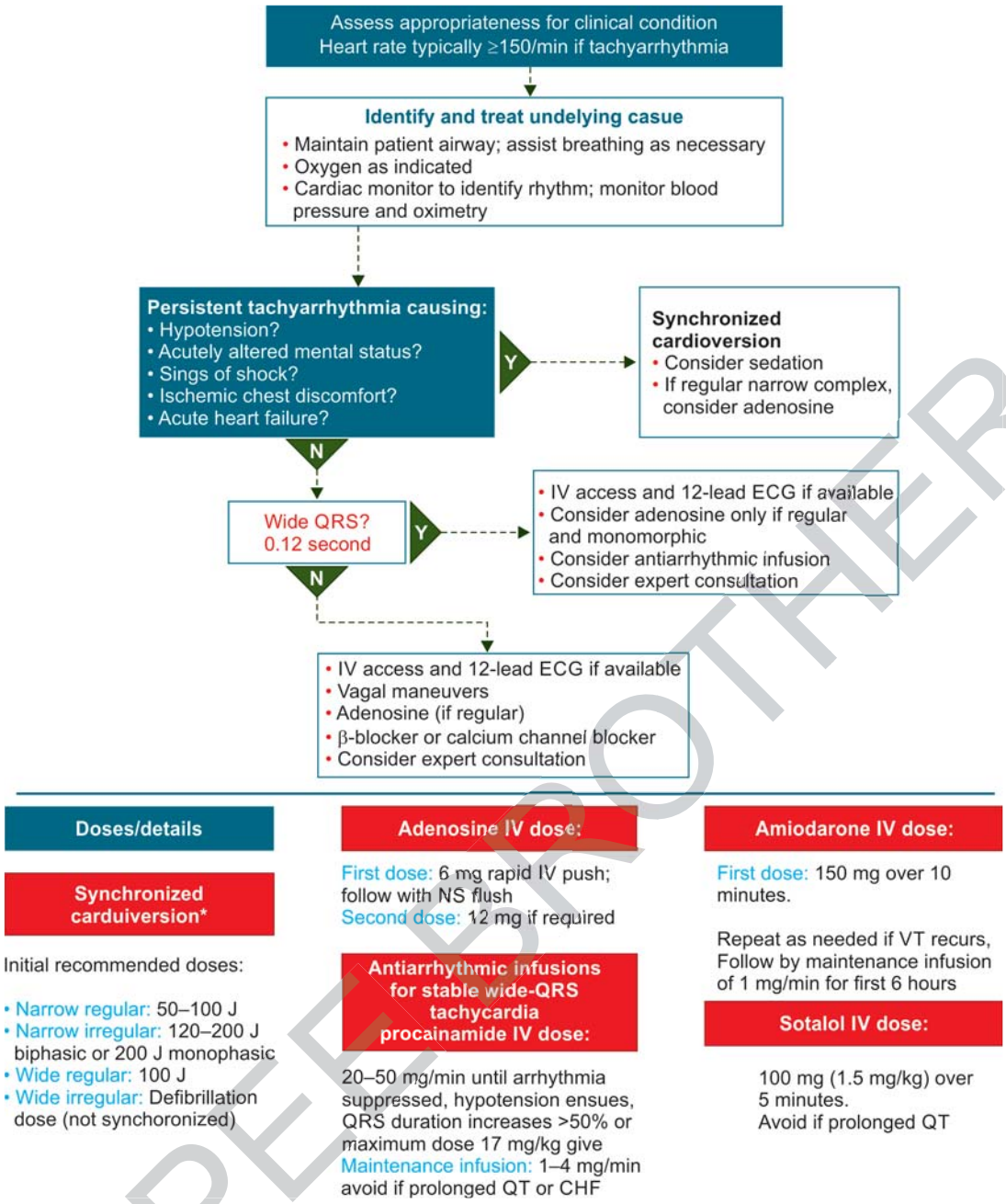

Fig. 44.5: Management of tachycardia as per ACLS guidelines.

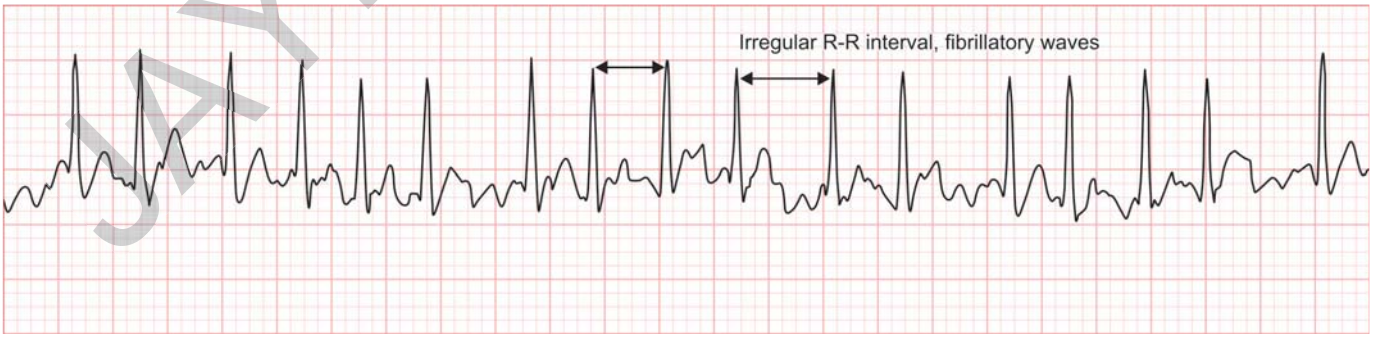

Fig. 44.6: Atrial fibrillation rhythm. As shown in above picture there are no definite P waves, irregular R-R interval. Fibrillatory waves, absence of isoelectric line.

and the absence of definite P waves **(Fig. 44.6)**. Underlying medical conditions and advanced age increases the chances of atrial fibrillation (AF).

Features of Atrial Fibrillation

❖ No repetitive pattern in RR interval. Thus, it is also called "irregularly irregular" rhythm.

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- No definite P waves. Fibrillatory waves are present or they can either be fine or coarse in occurrence.
- Absence of an isoelectric line
- QRS complex is usually <120 ms
- Fibrillatory wave may resemble P waves and may lead to misdiagnosis
- AF usually has ventricular rate between 110 and 160.
- AF with rate >100 bpm is fast AF.
- AF with rate <100 bpm is termed as slow AF.
- Slow AF may occur due to some medication toxicity (e.g., digoxin) and sinus node dysfunction.

Disease Conditions that may be Responsible for Atrial Fibrillation

- Hypertension
- Coronary heart disease (CHD)
- Rheumatic heart disease (RHD)
- Congestive heart failure
- Hypertrophic cardiomyopathy
- Congenital heart disease
- Thyroid dysfunction
- Ischemia

Mechanism of Atrial Fibrillation

Atrial remodeling: Atrial fibrillation is initiated by rapid firing from the pulmonary veins. In the early stages the atrium is healthy and the triggers terminate spontaneously and the rhythm returns to normal sinus rhythm, but later over the course of time, there is extensive damage and atrial remodeling.

Structural and electrical changes occur simultaneously in the atrium. The changes create re-entrant circuits which trigger and lead to atrial fibrillation. Later in time, it becomes difficult to maintain the normal sinus rhythm and the atrial fibrillation sustains.

Electrical remodeling: Atrial fibrillation induces changes in atrial electrophysiology due to electrical remodeling of the atrium leading to continuous decrease in atrial response. High electrical activation due to electrical remodeling stimulates atrial fibrillation.

Importance of Atrioventricular Node

The number of impulses that reach the ventricle from the atrium is regulated by atrioventricular node. The ventricular rate can be slow, moderate, or rapid on the basis of conduction of AV node.

The AV conduction rate depends upon the electrical properties of AV node and is influenced by the autonomic nervous system. Certain drugs also affect the AV node function, e.g., calcium channel blockers or digoxin.

Post cardiac surgery: Atrial fibrillation occurs in most of the patients after cardiac surgery. The mechanism of atrial fibrillation is multifactorial.

Classification

According to American Heart Association/American College of Cardiology/Heart Rhythm Society guidelines, the atrial fibrillation can be classified as follows:

- **Paroxysmal AF:** It is defined as AF that terminates within 7 days of onset or spontaneously. It is self-terminating in nature and also is called intermittent AF. The episodes may be variable in nature.
- **Persistent AF:** It is defined as the AF which fails to self-terminate within 7 days of occurrence. The termination may require some pharmacological management of electrical cardioversion to restore to normal sinus rhythm. Even after treatment, persistent AF patients may have paroxysmal AF episodes.
- **Long-standing persistent AF:** It is described as the AF which is lasted for more than 12 months of duration.
- **Permanent AF:** It is a term used to identify the individuals having persistent AF when clinician decides no longer to use rhythm control therapy for such cases.

Signs and Symptoms

The signs and symptoms of atrial fibrillation include low cardiac output due to the fibrillatory mechanism of the heart. The low cardiac output usually results in symptoms such as palpitations, fatigue, weakness, dizziness, lightheadedness, and syncope.

Evaluation

The diagnostic evaluation involves complete history taking and assessment. Focused history should be taken of symptoms and episodes of atrial fibrillation. The assessment should be done to rule out underlying disease conditions such as rheumatic heart disease or cardiac abnormalities or history of any surgical procedure involving heart valves. After obtaining a 12-lead ECG to make an accurate diagnosis, Holter monitoring can be done to assess the rhythm changes for a longer duration. Electrocardiography or transesophageal echocardiography can be done to see the changes in the left atrium (i.e., size and volume).

Treatment

The primary goal is to treat the underlying cause. If AF is reversible and is occurring due to some reversible underlying cause of trigger then controlling the cause and the trigger may help in reducing the episode of AF. Identification of duration and type of AF are important to deciding the treatment. The choice of treatment may vary according to the type—acute or chronic AF as well as depending on the heart rate of the patients. In patients with AF along with hemodynamic instability with fast ventricular rate, cardioversion is required. The treatment algorithm for management includes:

Rate Control Therapy

The main aim of atrial fibrillation primarily is to control the rate. After rate control is achieved rhythm control is focused, either using anti-arrhythmic drugs, percutaneous catheter ablation, or by surgical intervention, or by elective cardioversion.

The rate control medicine slows the conduction across atrioventricular (AV node, e.g., Beta-blockers or non-dihydropyridine calcium channel blockers).

Rhythm Control Therapy

The rhythm control therapy includes drugs such as anti-arrhythmic drugs.

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Cardiac Rhythm Therapies

When atrial fibrillation does not respond to medication or cardioversion, it may require cardiac rhythm therapies such as radiofrequency ablation, maze or mini-maze, or convergent procedure.

Ablation Therapy

In ablation therapy, a catheter is advanced near the origin of arrhythmia. High-frequency and low-energy sound waves are passed through the catheter which causes thermal injury and damage to localized tissue and leads to scar formation. Cryoablation can also be done by applying extreme cold temperature through a specific catheter to damage the tissues causing arrhythmia.

The main aim of every ablation procedure is to eliminate dysrhythmia.

Convergent Procedure

It is a hybrid procedure requiring both electrophysiological as well as cardiothoracic skills. A small incision is made through the diaphragm for visualization and toward the posterior wall of the heart. The ablation of epicardia wall and endocardial area of the pulmonary vein is carried out. The procedure usually requires 4–5 days of hospital stay.



NURSING CARE POST ABLATION THERAPY

A post-ablation patient requires similar care to the patients with post-cardiac catheterization.

- Monitoring the patient for reoccurrence of dysrhythmia
- Monitoring the patient for hematoma
- Evaluating for the signs and symptoms of stroke and pericardial tamponade
- Evaluating for risk of vascular access
- Focusing on pain management and providing comfort

Complications of Catheter Ablation

- AV block
- Pericardial tamponade
- Phrenic nerve injury
- Hematoma
- Stroke

Assessment of the Need for Anticoagulant

Anticoagulation therapy should be started on patients who will undergo cardioversion. The risk of thromboembolism and bleeding should be done by CHA2DS2-VASc **(Table 44.2)** or HAS-BLED **(Table 44.3)**. Patients with atrial fibrillation may receive oral anticoagulant therapy for the long term. Oral or long-term anticoagulants reduce the risk of stroke or thromboembolism in atrial fibrillation patients but may also increase the risk of bleeding. Thus, it is important to assess the risk versus benefits of starting anticoagulant therapy.

HAS-BLED score ≥3 indicates to be cautious while prescribing oral anticoagulation and recommends a regular evaluation.

Abnormal renal function is described as the presence of either chronic dialysis, renal transplantation, or elevated serum creatinine level ≥200 mmol/L. Abnormal liver function is described as chronic hepatic disease (e.g., cirrhosis) or biochemical evidence of significant hepatic derangement, i.e., bilirubin 2–3 times more than the upper limits of normal, elevation of aspartate aminotransferase/alanine aminotransferase/alkaline phosphate 2–3 times to normal limits, history of bleeding or anemia, liable INR, antiplatelet or nonsteroidal anti-inflammatory drugs or excessive alcohol use.

TABLE 44.2: CHA_2DS_2-VASc score.

CHA_2DS_2-VASc	*Score*
Congestive heart failure	1
Hypertension	1
Age >75 years	2
Diabetes mellitus	1
Stroke/TIA/TE	2
Vascular disease (prior MI, PAD or aortic plaque)	1
Age 65–74 years	1
Sex (Female)	1
Total	**9**

(TIA: transient ischemic attack; TE: thromboembolism; MI: myocardial infarction; PAD: peripheral artery disease)
The maximum CHA_2DS_2-VASc score is 9. The interpretation is as follows:
0 = no antithrombotic therapy
1 = antithrombotic therapy either antiplatelet or anticoagulants preferably anticoagulants
≥2 = oral anticoagulant therapy

TABLE 44.3: HAS-BLED score.

HAS-BLED	*Score*
Hypertension (SBP >160 mm Hg)	1
Abnormal renal/liver function	1 point each (1 or 2)
Stroke	1
Bleeding tendency/predisposition	1
Labile INR	1
Elderly (age >65 years)	1
Drug or alcohol	1 point each (1 or 2)
Total	**9**

(SBP: systolic blood pressure; INR: international normalized ratio)
The maximum HAS-BLED score is 9

Maze procedure: The maze procedure is a surgical intervention toward atrial fibrillation. Small incisions are made transmurally in the atrium. The scar tissues formed prevents re-entry conduction of electrical impulses. Usually, the procedure requires open-heart surgery and thus is planned with patients who are undergoing another cardiac surgery. Sometimes the maze procedure may cause injury to the SA node or in the atria and may require a permanent pacemaker.

However, a smaller version of the maze called mini-maze can also be performed by making small incisions in the atrium with the help of a video-guided instrument. The mini-maze does not require cardioplegia and hence reduces the risk of infection and shorten the time of recovery.

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NURSING MANAGEMENT

- Ineffective tissue perfusion related to decreased cardiac output:
  - Assess for blood saturation using pulse oximeter or cardiac monitor.
  - Observe for discoloration of skin or nail beds or of mucous membrane.
  - Provide supplemental oxygen if saturation is less than 94%.
  - Provide comfortable position to promote effortless breathing.
- Risk for decreased cardiac output/disease condition:
  - Assess the heart rate.
  - Assist patient in activities of daily living to prevent fatigue.
  - Adhere to strict intake output regimen to prevent cardiac overload.
- Activity intolerance/fatigue/decreased cardiac output:
  - Assess the capacity to perform activity of daily living.
  - Plan rest and balanced physical activity for the patient to prevent fatigue.
  - Assist in performing the physical activities of daily living to reduce fatigue.
  - Assess for environmental safety or raise the side rails to prevent fall.
- Impaired gas exchange/pulmonary embolus/thrombus:
  - Assess the respiratory rate or any abnormal breathing pattern, depth and effort, watch for use of accessory muscle, nasal flaring.
  - Assess for oxygen saturation using pulse oximeter or cardiac monitor.
  - Auscultate for diminished breath sounds or presence of any additional breath sound.
  - Observe for discoloration of skin or nail beds or of mucous membrane.
  - Administer medication as prescribed by the physician.
- Risk of ineffective cerebral tissue perfusion related to reduced cardiac output:
  - Assess for neurological status and signs of deterioration.
  - Assess for risk of hypoperfusion.
  - Limit the physical activity to reduce cardiac workload.
- Risk of stroke related to disease condition:
  - Assess for risk of bleeding or stroke using HAS-BLED or CHA_2DS_2-VASc score.
  - Educate the patients for adherence to anticoagulant therapy.
  - Assist patient in mobility or if the patient is bedridden apply DVT stockings.
  - Assist patient in range of motion exercises to promote circulation.
- Risk of bleeding related to prolonged anticoagulation therapy:
  - Assess for risk of bleeding and adherence to anticoagulant therapy.
  - Educate the patient about risk associated with anticoagulant therapy.
  - Educate the patient to get INR monitoring if patient is on coumarin derivative anticoagulants.
  - Assess the environment and remove all the sharp articles to prevent injury.

ATRIAL FLUTTER

It is a type of supraventricular tachycardia caused by additional re-entrant circuit formed within the right atrium **(Fig. 44.7)**. The length of the re-entrant loop corresponds to the size of the right atrium. The heart rate usually is around 300 bpm.

Etiology and Risk Factors

- It can occur after starting anti-arrhythmic drugs such as flecainide, propafenone, or amiodarone.
- Thyrotoxicosis
- Obesity
- OSA (obstructive sleep apnea)
- Sinus node dysfunction
- Pulmonary disease
- Pericarditis
- Pulmonary embolism

Classification

The atrial flutter is classified as *typical atrial flutter and atypical atrial flutter*. This classification system is based on the anatomical location and direction of re-entry circuits formed.

Typical Atrial Flutter

It is a common type of flutter. This can be further classified on the basis of the direction of the re-entrant circuit. The circuit usually follows two patterns clockwise and anticlockwise.

Anticlockwise circuit: This is the most common path followed. In this, retrograde or anticlockwise conduction is

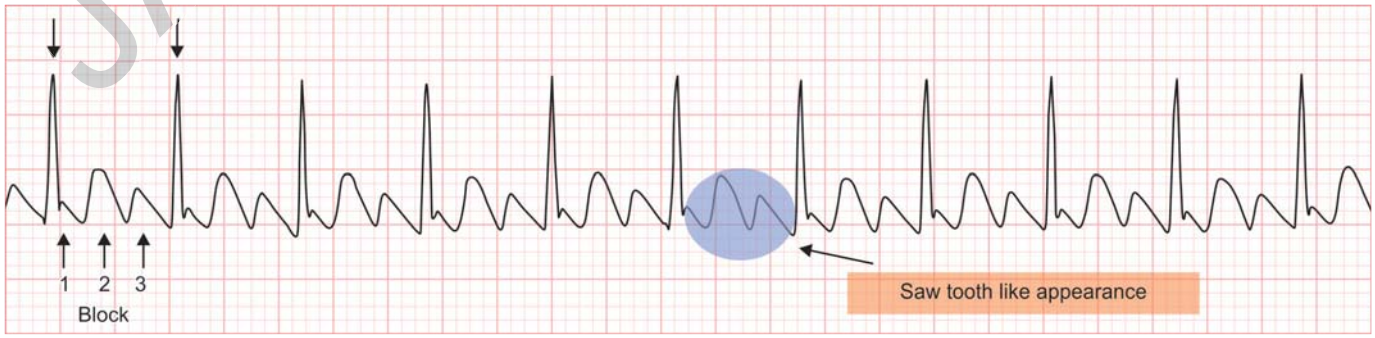

Fig. 44.7: Atrial flutter with 3:1 pattern as three fibrillatory waves can be seen within one R-R interval.

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produced. In cases of anticlockwise atrial flutter, inverted flutter waves are seen in leads II and III and in aVF. There are positive flutter waves in V1, these flutter waves may resemble P wave in appearance.

Clockwise circuit: This uncommon variant produces the opposite pattern. In this type, positive flutter waves are seen in II, III, and a VF. There can be broad, inverted flutter waves seen in V1.

Atypical Atrial Flutter

This is uncommon and type II flutter. It does not fulfill the criteria for typical atrial flutter. It is associated with high atrial rates and rhythm instability.

Features of Atrial Flutter

- Regularly irregular rhythm—due to repetition of the same pattern after a particular interval of time
- Usually rate is >250 beats/min.
- It is a narrow complex tachycardia.
- Saw-tooth appearance in lead II, III, and a VF can be easily identified by turning the ECG paper upside down.
- No isoelectric line and flutter waves of V1 may resemble P wave.
- There may be a pattern of alternating 2:1, 3:1, and 4:1 conduction ratios, i.e., there may be 2 fibrillatory or 3 fibrillatory or 4 fibrillatory waves within an R-R interval.

Clinical Manifestations

The atrial flutter patients usually present with signs and symptoms of hypotension leading to low cardiac output. The atrial flutter has a high heart rate. The increased heart rate decreases the ventricular filling and causes syncope. The person may experience symptoms such as shortness of breath, palpitations, lightheadedness, and angina.

Evaluation

The gold standard to assess the rhythm is the electrocardiograph. A 12-lead ECG should be obtained to assess the rhythm. Holter monitoring can be done followed by an electrocardiograph to make a diagnosis and to proceed with the treatment. Electrocardiograph is to be done to rule out underlying heart conditions. A blood count and hemodynamic monitoring should be done to assess the electrolyte imbalance.

Treatment

The major component of management of atrial flutter is the same as that of atrial fibrillation:

- Rate control therapy
- Reversal to normal sinus rhythm
- Maintenance of NSR
- Prevention of embolism

The preferred choice of treatment over medical therapy is radiofrequency ablation due to its minimal complications. However, synchronized cardioversions can be done for the flutters with short duration happening for less than 48 hours and the energy can be increased with subsequent shocks. Anticoagulant drugs should be added to the therapy to lower the risk of thromboembolism.

Rate control therapy: The rate control therapy in atrial flutter is the same as in the case of atrial fibrillation including the use of beta-blockers and non-dihydropyridine calcium channel blockers.

Reversal to normal sinus rhythm: Definite treatment to control the episodes of recurrence is radiofrequency catheter ablation.

Maintenance of NSR: The rate of recurrence is reduced with the correction of cause. In patients with typical atrial flutter, radiofrequency ablation is preferred over long pharmacological therapy and also it has a high success rate in reducing episodes of the atrial flutter.

Prevention of embolism: The risk of embolism is low in atrial flutter as that of atrial fibrillation but opting for anticoagulants reduces the risk of systemic embolism.



NURSING MANAGEMENT

- Ineffective tissue perfusion/decreased cardiac output/rapid heart rate:
  - Assess for blood saturation using pulse oximeter or cardiac monitor.
  - Observe for discoloration of skin or nail beds or of mucous membrane.
  - Provide supplemental oxygen if saturation is less than 94%.
  - Provide comfortable position to promote effortless breathing.
- Risk for decreased cardiac output/incomplete ventricular filling:
  - Assess the heart rate.
  - Limit physical activities to restore energy and reduce cardiac overload.
  - Maintain intake and output to reduce cardiac overload.
- Activity intolerance/fatigue/reduced cardiac output:
  - Assess the capacity of the patients for activity of daily living.
  - Plan rest and balanced physical activity for the patient to prevent fatigue.
  - Assist in physical activities of daily living to reduce fatigue.
- Impaired gas exchange/reduced cardiac output:
  - Assess the respiratory rate or any abnormal breathing pattern and depth and effort; watch for the use of accessory muscle and nasal flaring.
  - Assess for oxygen saturation using pulse oximeter or cardiac monitor.
  - Auscultate for diminished breath sounds or presence of any additional breath sound.
  - Assess the patient for altered sensorium.
  - Observe for discoloration of skin for any changes.
- Risk of fall/injury related to lightheadedness/syncope/hypotension:
  - Assess for risk of fall.
  - Limit physical activity of the patient.
  - Assist the patient in physical activity.
  - Check for environmental safety and raise the side rails to prevent fall.

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SUPRAVENTRICULAR TACHYCARDIA (SVT)

It is described as abnormally rapid heart rhythm. The supraventricular tachycardia originates above the ventricle often with a narrow QRS complex. SVT refers only to the atrioventricular (AV) reciprocating mechanism (AVRT and AVNRT) excluding primary origin atrial tachycardia, atrial fibrillation, and flutter **(Fig. 44.8)**. Primary origin tachycardias are already discussed in this chapter.

Mechanism

The re-entrant rhythms involve distinct pathways for conduction with unidirectional block in one of the two pathways. The direction of the circuit can be antegrade or retrograde with rapid ventricular contractions producing a high rate.

AV re-entrant tachycardia: In this type, an extra accessory pathway is present between the atrium and ventricle which maintains conduction exchange between both.

The conduction pathways can be anywhere between the right and left AV groove. The common site is the left lateral atrioventricular groove and posteroseptal region. The pre-excitation pathways between atrium and ventricle can be from atrium to ventricle, low AV node (atrio-nodal bypass tract), the bundle of His (atrio-fascicular bypass tract), and from AV node to bundle of branch (nodofascicular bypass tract) or AV node to ventricular tissue (nodoventricular bypass tract) or from bundle branch to ventricular tissue (fasciculoventricular bypass tract).

Antegrade versus Retrograde

The terms antegrade and retrograde are based on the direction of electrical conduction through pathways between the atrium and ventricle. When the conduction is between the atrium and ventricle, then it is considered in antegrade direction and when it happens between ventricle to atrium it is in retrograde direction. Some accessory pathways can be capable of conduction in both antegrade as well as in retrograde directions.

Orthodromic versus Antidromic

This refers to the direction of electrical conduction through AV node.

Mostly the supraventricular tachycardia is orthodromic AVRT, where the conduction in the circuit occurs antegrade down to AV node by His-Purkinje system to the ventricles and then in the retrograde up to the accessory pathway resulting in the formation of narrow complex tachycardia.

However, in cases of antidromic AVRT, a wide complex tachycardia is present due to conduction of impulses antegrade down in accessory pathway without involving ventricular conducting system and then retrograde up to AV node. Hence conduction taking place through ventricular myocardium is slower than conduction system.

Clinical Features

Infants with supraventricular tachycardia may exhibit symptoms such as poor feeding, irritability, fussiness, pallor, or cyanosis. Commonly the infants arrive with signs of heart failure (e.g., tachycardia, fatigue, or poor weight gain). However, the children and adolescents may exhibit symptoms of chest discomfort, fatigue, lightheadedness, or palpitations. The duration of episodes varies from individual to individual. On average one episode of SVT lasts for 10–15 minutes whereas some may last for 1–2 minutes or may even last for hours.

The heart rate in infants maybe 220–280 bpm, however, in children and adolescents, it may be 150–200 bpm.

Treatment of Supraventricular Tachycardia

A baseline ECG should be done to determine the rhythm. Assess the patient for tachycardia and try to find out the cause of tachycardia whether it is primary or secondary to any underlying disease condition. Assessment for signs and symptoms of ischemia and low cardiac output such as altered mental status, hypotension, heart failure, and symptoms of shock should be done. Breathing discomfort and need for supplemental oxygen should be assessed. The cardioversion should be done if the patient is unstable.

To start with treatment, IV line should be established. As an initial therapeutic choice vagal maneuver can be done to stable supraventricular tachycardia. Carotid sinus massage can be done alternative to vagal maneuver. The carotid sinus is located behind the angle of the jaw. The person is made to sit or lie down. Unilateral firm pressure should be applied

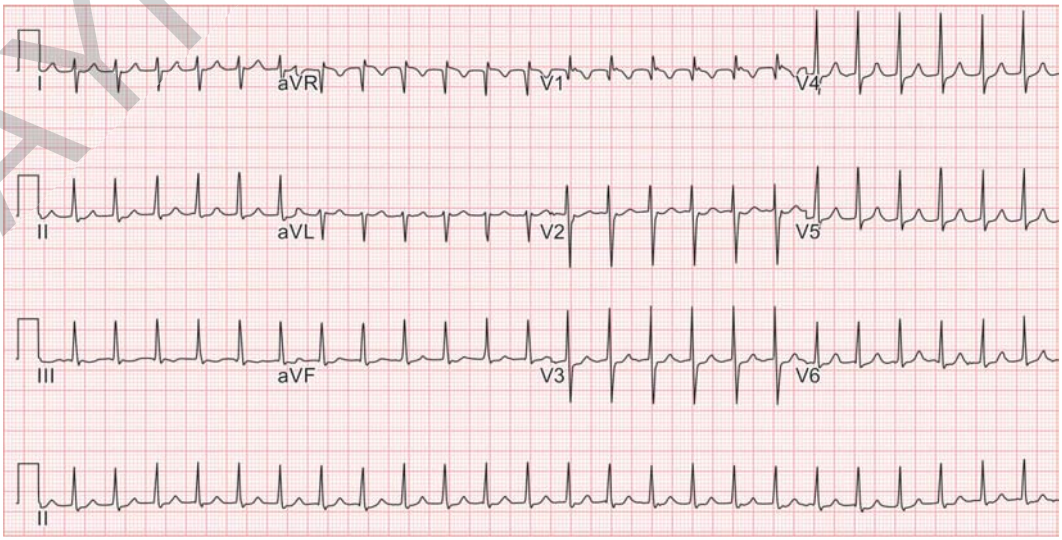

Fig. 44.8: Narrow QRS complex can be seen in this picture with rate approximately 150 bpm. No visible P waves are seen.

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for 5 seconds. The carotid sinus massage should be done in patients with carotid bruits and carotid artery disease.

If the patient is not hypotensive then adenosine is the drug of choice. It should be given as 6 mg rapid IV push (followed by 10 mL saline flush). The adenosine inhabits and thus slows the AV nodal conduction. The half-life of adenosine is less than 10 seconds and should be given with 10 mL rapid flush. The 6 mg stat adenosine should be given followed by 12 mg after 1–2 minutes (maximum 18 mg should be given). A second, higher dose can be administered if the rhythm does not convert with 12 mg within 1–2 minutes. If the rhythm does not settle with vagal maneuvers and adenosine and recurs after this treatment, then long-acting AV nodal block agents should be given as a treatment choice, such as non-dihydropyridine calcium channel blocker (e.g., diltiazem or verapamil) or beta-blocker. Frequent attacks require radiofrequency ablation to destroy the abnormally-conducting cells. Calcium channel blockers and beta-blockers remain the option. If adenosine fails, calcium channel blockers or beta-blockers can be given to acutely slow down AV node conduction. Calcium channel blockers should be avoided in patients with left ventricular dysfunction.



NURSING MANAGEMENT

- Acute chest pain related to hypoxemia and rapid heart rate:
  - Assess the level of pain by using pain scale.
  - Provide comfortable position, e.g., Fowler's or semi-Fowler's position.
  - Treat the underlying cause for tachycardia.
- Ineffective tissue perfusion related to decreased cardiac output/rapid heart rate:
  - Assess for blood saturation using pulse oximeter or cardiac monitor.
  - Observe for discoloration of skin or nail beds or of mucous membrane.
  - Provide supplemental oxygen if saturation is less than 94%.
  - Provide comfortable position to promote effortless breathing.
- Decreased cardiac output related to inadequate ventricular filling:
  - Check the heart rate of the patient.
  - Plan rest and balanced physical activity for the patient to prevent fatigue.
  - Maintain strict intake output for the patient.
  - Administer medication to control the heart rate to prevent further deterioration.
- Impaired gas exchange related to low cardiac output:
  - Assess blood oxygen level of the patient.
  - Provide high Fowler's or semi-Fowler's position to the patient.
  - Provide supplemental oxygen if required for the patient.
- Activity intolerance related to fatigue, poor physical activity:
  - Assess the capacity to perform physical activity.
  - Limit the physical activity of the patient.
  - Plan for rest and balanced physical activity and provide assistance in performing physical activities.
- Ineffective breathing pattern related to shortness of breath and pulmonary edema:
  - Assess the respiratory rate and breathing pattern of the patient.
  - Assess for abnormal breath sounds if present.
  - Provide comfortable position to promote ease breathing.
  - Provide steam inhalation or nebulization if there are secretions to loosen the secretions.
- Anxiety related to altered health state:
  - Assess the level of anxiety by conversion with the patient.
  - Provide psychological support to the patient.
  - Educate the patient about the disease condition to enhance understanding about the disease.

Premature atrial contractions (PACs) are early heartbeats originating from an ectopic focus in the atria, occurring before the next expected sinus beat. They may be caused by stress, caffeine, alcohol, electrolyte imbalance, or underlying cardiac disease. Most patients are asymptomatic, though some may feel palpitations or a "skipped beat." PACs are usually benign and require no treatment unless symptomatic, in which case triggers are avoided and underlying causes corrected **(Fig. 44.9)**.

HEART BLOCKS (ATRIOVENTRICULAR NODE BLOCKS)

A 55-year-old female presents in emergency department with complaints of palpitations, no chest pain, or shortness of breath. She is hypertensive and diabetes controlled with medications. As per the assessment done, her heart rate was 68 beats per minute. The physician ordered an

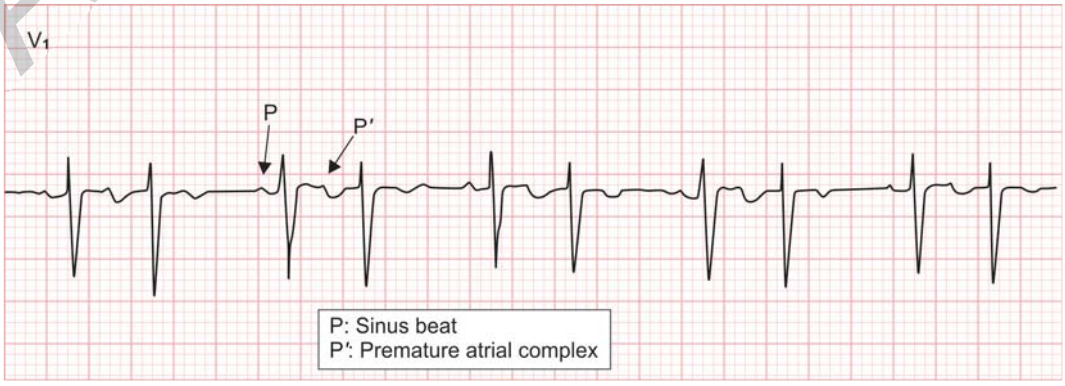

Fig. 44.9: Premature atrial contractions.

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electrocardiograph. While analyzing the electrocardiograph the physician found that the PR interval is increased in duration >0.20 second and is constantly increased throughout the rhythm. The physician labeled the patient as having a first-degree heart block. Let us look in detail what heart blocks are like.

Heart Block

The partial or complete delay or interruption in the conduction from the atrium to ventricle. The block can be anywhere at AV node, the bundle of His, or bundle branches. The common causes of atrioventricular blocks are ischemia, myocardial infarction, or degenerative disease of the conduction system. The cardiac output can be low with the atrioventricular blocks if ventricular rates are low.

The atrioventricular blocks can be of different types:

- First-degree atrioventricular block
- Second-degree atrioventricular block
- Third-degree atrioventricular block

First-degree Atrioventricular Block

In first-degree heart block, all impulses are conducted from atria to ventricle but there is delay in conduction at AV node. The delayed conduction from atria to ventricle results in prolonged PR interval (>200 ms). The prolonged PR interval can be seen with no beat-to-beat variation **(Fig. 44.10)**.

Causes: The atrioventricular blocks may have the following causes:

- **Cardiac causes**—cardiac conditions like myocardial infarction, ischemia at AV node or age-related changes, mitral valve surgery, myocarditis
- **Non-cardiac cause**—hypothyroidism, hypo- or hyperkalemia
- **Drugs**—beta-blockers, calcium channel blockers, or antiarrhythmic drugs

ECG appearance: The classical appearance is prolonged PR interval which is greater than 0.20 second in duration with constant beat to beat.

Treatment: There is no specific treatment as cardiac output is not affected. The main focus is done on the identification of cause and treatment.

Second-degree Atrioventricular Block

The second-degree atrioventricular block is of two types: (1) Mobitz type I, and (2) Mobitz type II.

It is a block at the AV node or bundle of His or bundle branches, leading to atrial impulses which are not conducted in ventricles. Type II AV blocks are less common than type I and may further lead to complete heart block.

Mobitz Type 1 (Wenckebach)

It has prolonged PR interval. PR interval is longest in duration before a dropped beat and shortest immediately after a dropped beat. The PR interval continues to progress throughout the cycle till it drops a beat. However, the PP interval remains constant **(Fig. 44.11)**.

Mechanism: This usually occurs due to conduction block at level of AV node. Malfunctioning AV nodal cells fail to conduct impulses due to fatigue.

Causes:

Cardiac

- Myocardial infarction
- Myocarditis
- Valvular repair
- Tetralogy of Fallot repair

Drugs

- Calcium channel blockers
- Beta-blockers
- Amiodarone

Treatment:

- Asymptomatic patients do not require any treatment.
- Symptomatic patients respond to atropine.
- Type I has less chances to convert further into complete heart block.

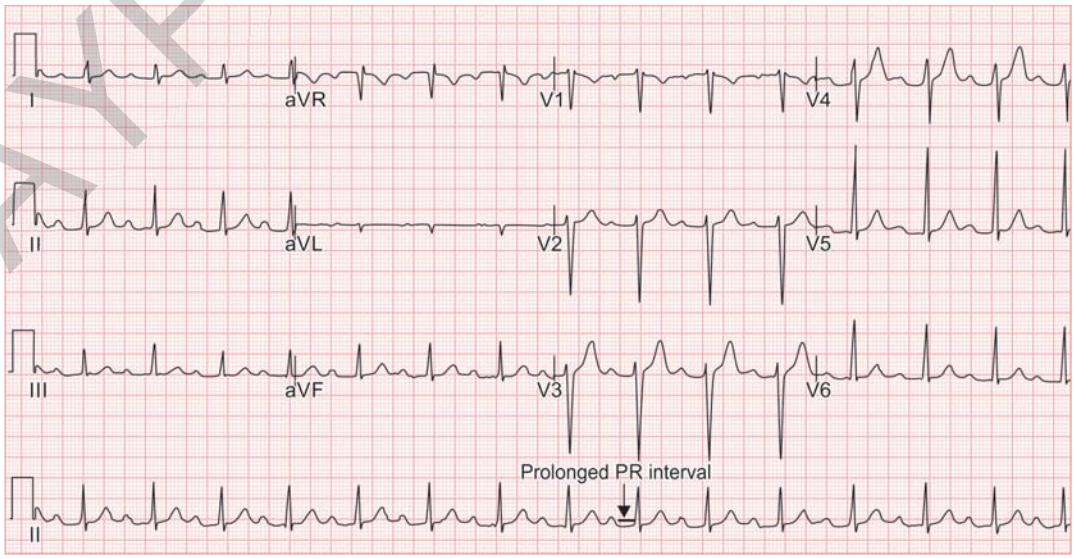

Fig. 44.10: First-degree atrioventricular block. The normal PR interval is 0.12–0.20 sec. In first-degree atrioventricular block, there is prolonged PR interval.

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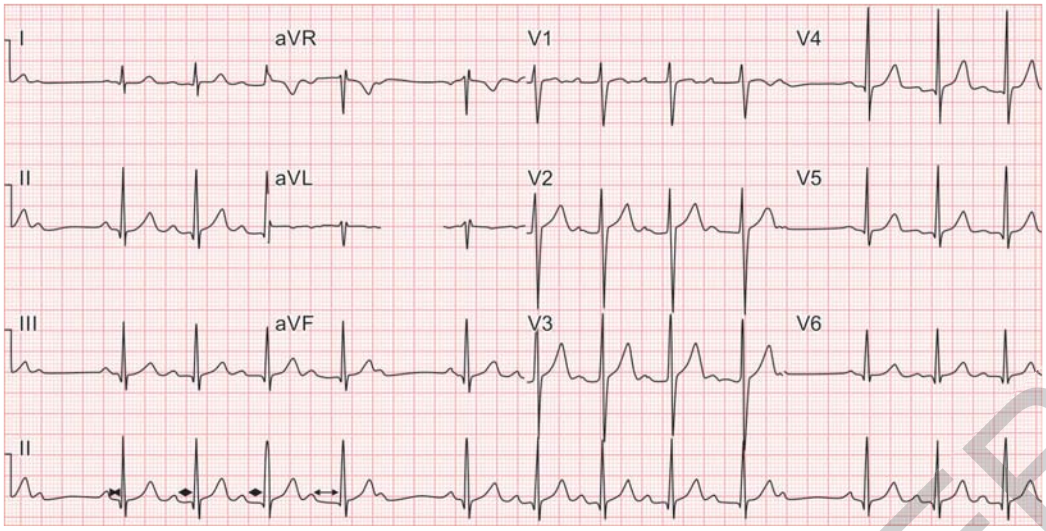

Fig. 44.11: Second-degree atrioventricular block Mobitz Type 1. The normal PR interval is 0.12–0.20 sec. In second-degree atrioventricular block, there is prolongation of PR interval till it has a non-conducting P wave.



Fig. 44.12: Second-degree atrioventricular block Mobitz Type II. The normal PR interval is 0.12–0.20 sec. In second-degree atrioventricular block, the PR interval is constant but it has non-conducting P waves.

Mobitz Type II (Hay Block)

The second-degree heart block is a type of atrioventricular heart block in which the PR interval is constant but has some non-conducted P wave.

Mechanism: This occurs due to conduction failure at His-Purkinje system (i.e., below the AV node). Mobitz I is usually due to functional suppression of AV conduction (e.g., drugs or reversible ischemia), while Mobitz II usually occurs due to structural damage to the conduction system (e.g., fibrosis, necrosis, or infarction) **(Fig. 44.12)**.

Causes

See **Table 44.4**.

Clinical presentation: The heart block causes hemodynamic instability; severe bradycardia usually in cases of Mobitz II progresses to third-degree heart block. The person may have symptoms of hypotension, chest pain, and syncope and if the symptoms persist, they may lead to sudden cardiac death.

TABLE 44.4: Causes of Mobitz type II.

Cardiac	*Non-cardiac*	*Drugs*
• Myocardial infarction • Idiopathic fibrosis of conduction system • Mitral valve repair	• Rheumatic fever • Lyme disease • Autoimmune condition—SLE, amyloidosis	• Calcium channel blockers • Amiodarone • Digoxin • Beta-blockers

(SLE: systemic lupus erythematosus)

Third-degree Atrioventricular Block

In the third-degree atrioventricular block there is an absence of atrioventricular conduction hence resulting in severe bradycardia; the ECG shows complete AV dissociation with independent atrial and ventricular rates **(Fig. 44.13)**. There is complete absence of atrioventricular conduction; no atrial impulse is conducted to ventricle. Complete heart block may result in sudden cardiac death post syncope.

Pathophysiology: Complete heart block occurs especially as an endpoint of Mobitz I or Mobitz II atrioventricular block.

Causes

Cardiac: Myocardial infarction, idiopathic degeneration of conduction system

Noncardiac: Calcium channel blockers, beta-channel blockers, or digoxin

Clinical Presentation

- ❖ Bradycardia
- ❖ Low cardiac output
- ❖ Chest pain
- ❖ Hypotension

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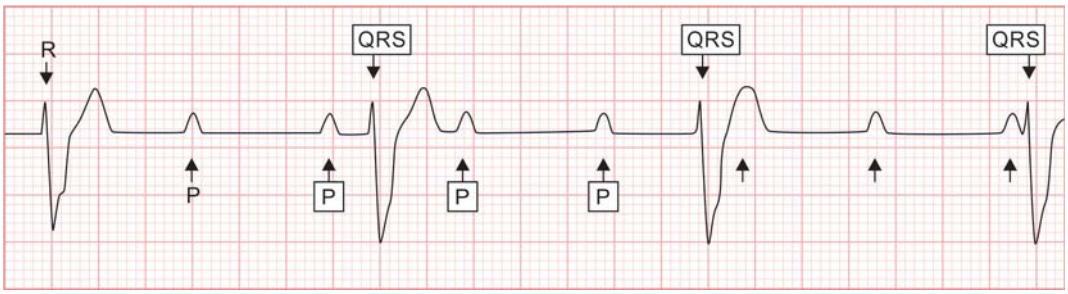

Fig. 44.13: Third-degree atrioventricular block, both atria and ventricle are independent of each other. There is a complete AV dissociation. As can be seen in figure where more P waves are present than the QRS complexes. In case of third-degree/complete heart block both atrium and ventricle contract and relax at their rate independent of each other.

TABLE 44.5: Summary of atrioventricular blocks.

Type of AV block	*Rhythm*	*P wave*	*QRS complex*	*PR interval*
First-degree AV Block	Regular	Present, normal followed by QRS complex	Normal	>200 ms
Second-degree type I AV Block	Irregular	Present, normal Not all P waves are followed by QRS complex	Normal or wide	Continues to progress till one QRS is missed
Second-degree type II AV Block	Can be both regular/ irregular	Present, normal Not all P waves are followed by QRS complex	Normal or wide	Constant for all conducted beats
Third-degree AV Block	Rhythms are regular but independent of each other	No synchronization between P wave and QRS complex	Wide in ventricular focus cases	Not measurable

- Syncope
- Hemodynamic instability
- High-risk of ventricular standstill and sudden cardiac death.

Treatment

- Symptomatic patients should be treated as per symptoms.
- IV line should be initiated.
- Oxygen therapy should be started.
- Injection atropine and epinephrine or dopamine can be given.
- Transcutaneous pacing should be started for patients who have bradycardia.

Summary of Atrioventricular Blocks

Summary of various AV blocks along with their ECG findings is shown in **Table 44.5**.



NURSING CONSIDERATIONS

- Assess for vital signs
- Cardiac monitoring
- Check for any rhythm or laboratory abnormality.
- Monitor for signs of fluid retention.
- Monitor for pacing if the patient is on temporary pacing.
- Observe for signs of complications.
- Provide a comfortable position.
- Administer prescribed medications and look for signs of complications.
- Assess for baseline activity capacity and schedule activities as per performance; assist in activities of daily living.
- Watch for signs of presyncope/syncope.



NURSING MANAGEMENT

- Acute chest pain related to hypoxemia, disease condition:
  - Assess the level of pain by using pain scale.
  - Provide comfortable position, e.g., Fowler's or semi-Fowler's position.
  - Treat the underlying cause for bradycardia.
- Ineffective tissue perfusion related to decreased cardiac output/decreased heart rate:
  - Assess for blood saturation using pulse oximeter or cardiac monitor.
  - Observe for discoloration of skin or nail beds or mucous membrane.
  - Provide supplemental oxygen if saturation is less than 94%.
  - Provide comfortable position to promote effortless breathing.
- Risk for decreased cardiac output related to incomplete ventricular filling:
  - Assess the heart rate.
  - Limit physical activities to restore energy and reduce cardiac overload.
  - Maintain intake and output to reduce cardiac overload.
- Activity intolerance related to fatigue/reduced cardiac output:
  - Assess the capacity to perform physical activity.
  - Limit the physical activity of the patient.
  - Plan for rest and balanced physical activity.
- Impaired gas exchange related to reduced cardiac output:
  - Assess blood oxygen level of the patient.

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- Provide high Fowler's or semi-Fowler's position to the patient.
- Provide supplemental oxygen if required for the patient.

❖ Risk for infection related to percutaneous pacemaker insertion:
- Assess for body temperature and pacemaker insertion site.
- Follow aseptic technique while touching the site or avoid touching the site.
- Do proper hand washing.

VENTRICULAR TACHYCARDIA

Ventricular tachycardia is a wide complex, fast dysrhythmia arising from ventricles. When 6–10 premature ventricular complexes (PVC) occur in a row, it is called sustained ventricular tachycardia (VT). The VT can happen with or without pulse and the patient can be stable or unstable **(Fig. 44.14)**. VT usually proceeds to ventricular fibrillation (VF) and then to sudden cardiac death.

Causes

The common cause of ventricular tachycardia is increased myocardial irritability which can be triggered by increased automaticity.

Effects

In a clinical scenario, VT is always significant. The VT can be perfusing or non-perfusing. Even if the VT has a pulse, it should be considered unstable as the patient has risk to develop life-threatening rhythm. Usually, VT indicates underlying cardiovascular disease. Rapid heart rate in VT leads to compromised cardiac output and decreased coronary and cerebral perfusion.

Presentation

The VT appears with rapid heart rate usually >100–250 beats/min with wide QRS complex with more than 0.12 seconds. There can be absence of T wave or it will be in opposite direction to R wave. Usually, rhythm is regular.

Management

The primary goal of the treatment is to maintain the hemodynamical stability of the patient and to rule out the primary cause. Vigilant treatment is required in patients who are unstable. The steps of initial management include:

❖ Attaching the patient to continuous cardiac monitoring
❖ Obtaining a 12-lead ECG
❖ Maintaining the IV access
❖ Supplement the individual with oxygen
❖ Blood samples

In the patients who are unstable, the primary goal is to make them hemodynamically stable. At ongoing assessment, it is important to rule out whether the rhythm is shockable or not. The guidelines of ACLS are to be followed as a part of management. Primarily cardioversion is done if the rhythm is shockable followed by antiarrhythmic drug therapy.

Symptomatic Patients

Initial therapy should be started with beta-blockers in symptomatic patients. Non-dihydropyridine calcium channel blockers (i.e., diltiazem and verapamil) can also be added along with beta-blockers or in cases where beta-blockers are contraindicated.

Polymorphic Ventricular Tachycardia

The ventricular tachycardia can be monomorphic or polymorphic depending on the appearance of QRS complex. The presentation of polymorphic ventricular tachycardia gives a spiral-like appearance. The rate is typically 200–250 beats per minute.

Causes

❖ The torsades de pointes tachycardia is associated with long QT syndrome, hence prolonged QT segment can be seen on ECG.
❖ Long QT syndrome can be inherited as a congenital mutation of action potential or of the drugs block cardiac ion currents.
❖ It can be a result of heart block, myocardial ischemia, Prinzmetal's angina.
❖ Drugs that may cause prolonged QT segment may include amiodarone, quinidine, procainamide, psychotropic drugs (phenothiazines, tricyclic antidepressants, lithium), chloroquine, clarithromycin, or haloperidol.
❖ Electrolyte imbalances such as hypocalcemia, hypokalemia, and hypomagnesemia, or can be due to excessive fluid loss from the body.

Treatment

The management is the same as of ventricular fibrillation **(as discussed in ventricular fibrillation)**. Initially, shock can be given. In the patients with normal QT segment amiodarone can be started. Magnesium sulfate can be given to patients to maintain the electrolyte imbalance.

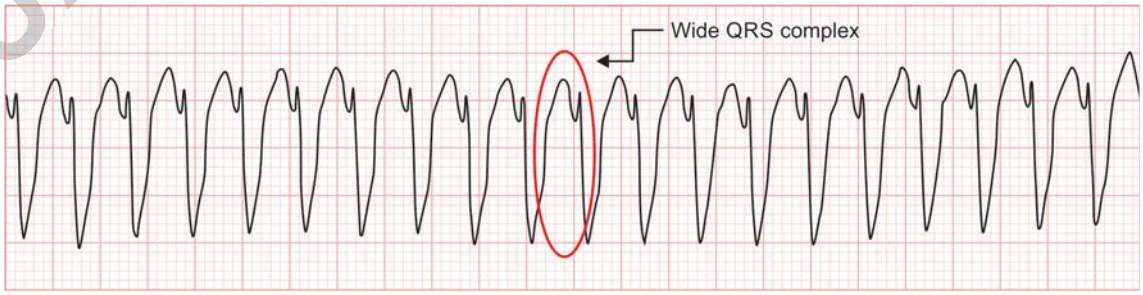

Fig. 44.14: Monomorphic ventricular tachycardia. No components of normal sinus rhythm are seen and wide QRS complex can be differentiated (i.e., >120 ms).

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VENTRICULAR FIBRILLATION

Ventricular fibrillation results from multiple conductions produced in ventricle. There is no uniform ventricular contraction in cases of ventricular fibrillation resulting in low cardiac output and low blood pressure. The ECG presentation in ventricular fibrillation shows rapid heart rate (300–400 beats/min) and irregular and shapeless QRST of varying amplitude and interval **(Fig. 44.15)**. Over the course of time, these waveforms decrease in amplitude and result in finer waveform. In the end, if left untreated it causes asystole.

Causes

- The main cause is significant disease of cardiovascular system such as myocardial ischemia, cardiomyopathy, coronary artery disease, and untreated ventricular tachycardia.
- **Drugs:** Epinephrine, quinidine, procainamide, overdose of cocaine or tricyclics
- **Non-cardiac cause:** Electrolyte imbalance such as hyper- or hypokalemia or hypercalcemia, acid-base imbalances
- Smoking
- Early repolarization

Treatment

High-quality CPR and defibrillation.
ACLS algorithm: Management of ventricular fibrillation as per ACLS guidelines is shown in **Figure 44.16**.



NURSING MANAGEMENT

- Acute chest pain related to hypoxemia, reduced cardiac output:
  - Assess the level of pain by using pain scale.
  - Provide comfortable position, e.g., Fowler's or semi-Fowler's position.
  - Treat the underlying cause for tachycardia.
- Risk for ineffective cardiac tissue perfusion related to inadequate ventricular filling/rapid heart rate:
  - Assess the environment for any irritants that may impair the gas exchange.
  - Provide comfortable position to the patient Fowler's or semi-Fowler's position to promote breathing.
  - Position the patient every 2 hourly and monitor the oxygen saturation.
- Risk for ineffective peripheral tissue perfusion related to edema, pulmonary edema:
  - Assess the peripheral blood saturation.
  - Assess the skin and nail beds for any discoloration or changes.
  - Involve and assist the patient in passive exercises to promote peripheral circulation.
- Altered sensorium related to dizziness/confusion:
  - Assess the mental status.
  - Do not leave the patient alone or unattended to prevent fall.
  - Assess for higher functions such as speech.
  - Check for changes in mental status from time to time.
- Risk for decreased cardiac output related to incomplete ventricular filling/fast heart rate:
  - Assess the heart rate.
  - Limit physical activities to restore energy and reduce cardiac overload.
  - Maintain intake and output to reduce cardiac overload.
- Impaired gas exchange related to hypoxemia/ventricular-perfusion imbalance/orthopnea/reduced cardiac output
  - Assess blood oxygen level of the patient.
  - Assess the respiratory rate, depth, and efforts for breathing.
  - Assess both lungs for decreased ventilation.
  - Provide high Fowler's or semi-Fowler's position to the patient.
  - Provide supplemental oxygen if required for the patient.
  - Encourage or assist in ambulation as per physicians' order.

Premature ventricular contractions (PVCs) are early, wide, and abnormal heartbeats originating from an ectopic focus in the ventricles, occurring before the next normal sinus beat. They may be triggered by stress, caffeine, alcohol, electrolyte disturbances, hypoxia, or underlying heart disease. Patients may experience palpitations or a sensation of a "missed" or "thumping" beat. Occasional PVCs are usually benign, but frequent or multifocal PVCs may require evaluation and treatment of the underlying cause, along with correction of electrolyte imbalances **(Fig. 44.17)**.

Torsades de pointes is a form of polymorphic ventricular tachycardia characterized by a twisting QRS pattern around the baseline and is typically associated with a prolonged QT interval. It may be triggered by electrolyte disturbances (especially low magnesium or potassium), certain medications, congenital long-QT syndromes, or bradyarrhythmias. Patients can present with palpitations, dizziness, or syncope, and it may progress to ventricular fibrillation. Management includes immediate IV magnesium

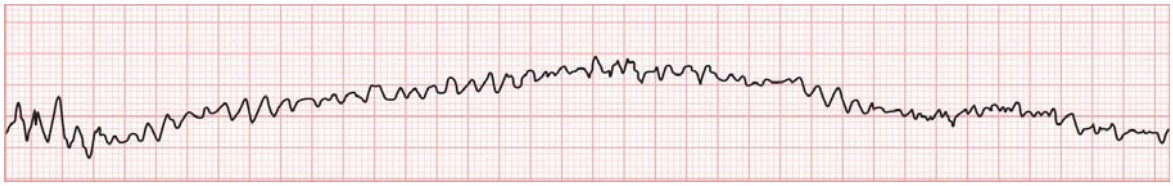

Fig. 44.15: Ventricular fibrillation. No components of normal sinus rhythm are seen. There is presence of fibrillatory waves non-corresponding to isoelectric line.

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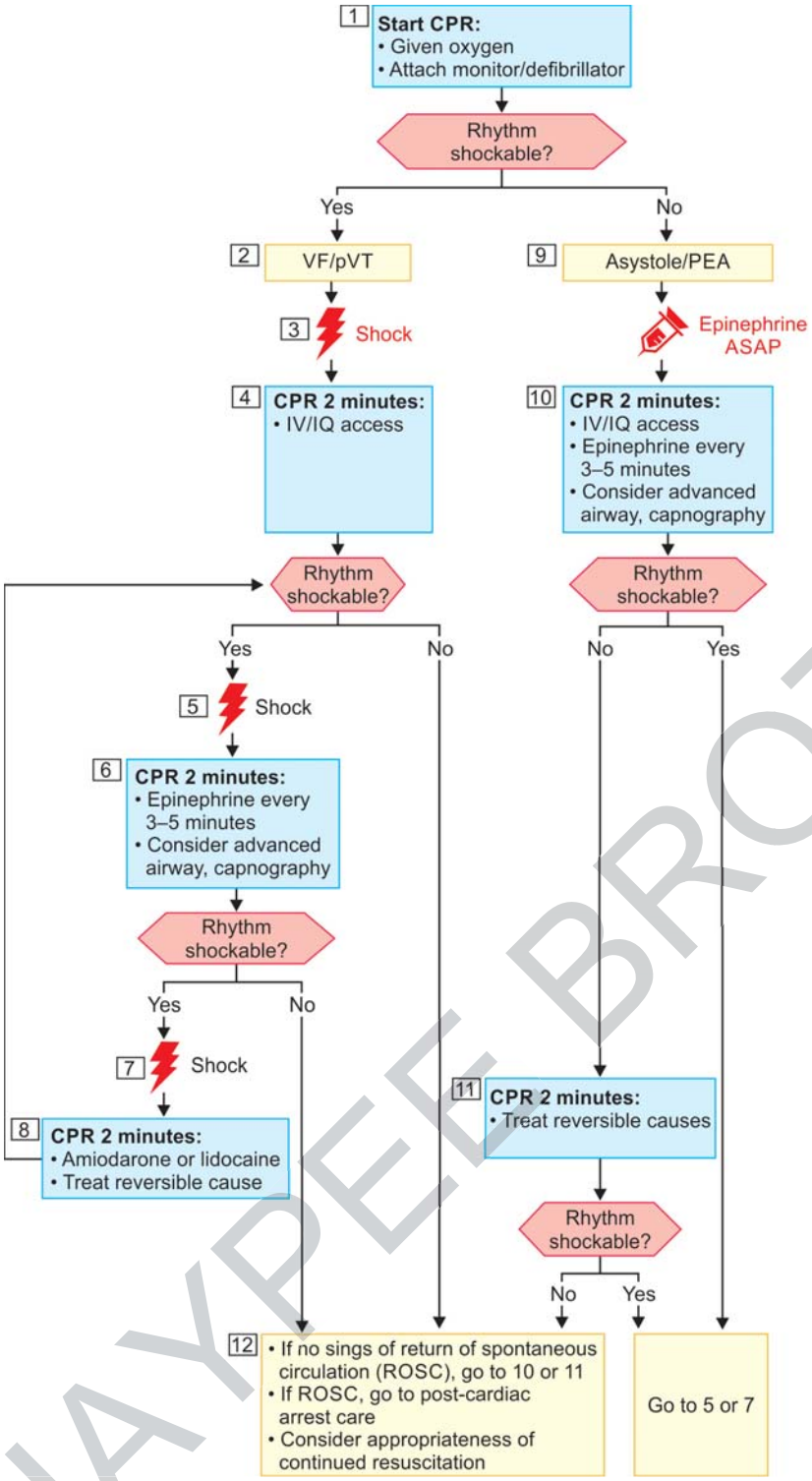

CPR quality

- Push hard [at least 2 inches (5 cm)] and fast (100 to 120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
- If $PETCO_2$, is low or decreasing, reassess CPR quality.

Shock energy for defibrillation

- **Biphasic:** Manufacturer recommendation (e.g., initial dose of 120 to 200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered
- **Monophasic:** 360 J.

Drug therapy

- **Epinephrine IV/IO dose:** 1 mg every 3–5 minutes:
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus Second dose: 150 mg or
- **Lidocaine IV/IO dose:** First dose: 1–1.5 mg/kg Second dose: 0.5–0.75 mg/kg

Advanced airway

- Endotracheal intubation or supraglottic advanced airway.
- Waveform capnography or capnometry to confirm and monitor ET tube placement.
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions.

Return of spontaneous circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in $PETCO_2$ (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Fig. 44.16: Management of ventricular fibrillation.



Fig. 44.17: Premature ventricular contractions.

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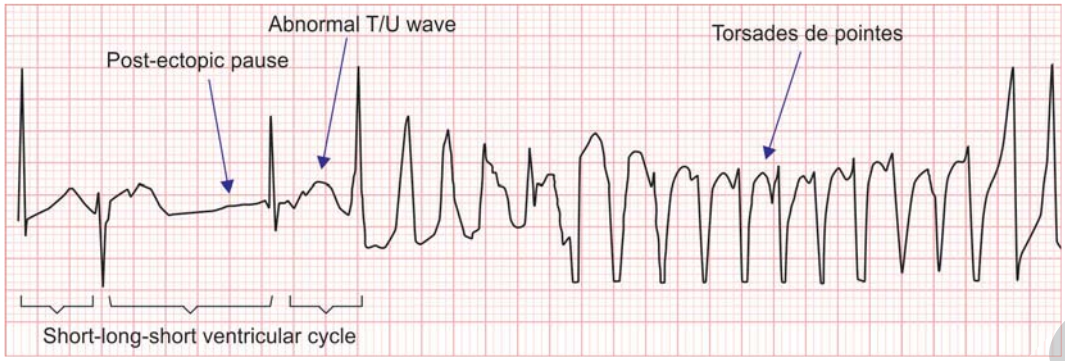

Fig. 44.18: Torsades de pointes.

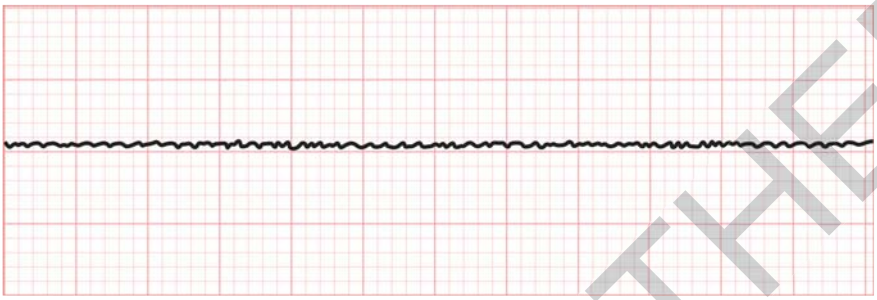

Fig. 44.19: Asystole. There is absence of cardiac activity in case of asystole, thus there is a straight line corresponding to isoelectric line.

sulfate and correction of underlying causes; defibrillation may be required if unstable **(Fig. 44.18)**.

ASYSTOLE

Asystole is described as a state of absence of any cardiac activity. It can be due to cardiac arrest or due to complete heart block. Asystole is a predictor of death as likelihood of resuscitation is less **(Fig. 44.19)**.

ELECTROLYTE IMBALANCE

Various changes in the ECG in the patients with electrolyte disturbances are shown in **Table 44.6**.

TABLE 44.6: ECG changes with electrolyte disturbances.

Potassium level	*ECG changes*
Hypokalemia <2.5–3.0	Prolonged QT interval
	Bifid T wave (Pathological U waves)
	Torse de pointes
Hyperkalemia 5.5–6.5	T wave peaking
>6.5	Wide QRS
	PR interval prolongation
>8.0	Loss of P waves (Sinoventricular rhythm)
	Wide QRS complex
	Low ventricular rate
	Asystole
Hypercalcemia	Short QT interval
Hypocalcemia	Prolonged QT interval
Hypomagnesemia	Prolonged QT interval
	Premature ventricular contractions
Hypermagnesemia	Atrioventricular and intraventricular disturbances

GERIATRIC CONSIDERATIONS

Most of the heart diseases cause impaired blood circulation. Mostly the patients may present with chest pain, dizziness, fatigue, activity intolerance, and breathing difficulty. To rule out the underline cardiac condition, ECG is done as a primary test.

Geriatric cardiology involves the needs of a growing population or old age, i.e., above 65 years or older. The approach in geriatric patients is selected by taking all the medical, physical, and mental profile of the patient into consideration to make it more individualized. Complete knowledge about geriatric assessment and age-related pharmacokinetic changes should be taken into consideration while making a diagnosis or treatment choice.

Case Scenario

A 35-year-old woman wakes up in the morning. As she stepped out of the bed, she begins to feel shortness of breath and palpitations and passes out. Her husband brought her to the emergency department. The husband stated that this kind of episode has occurred previously also where she had irregular heartbeat, sweating and palpitations. It happened 5–6 times in the past 1 month and lasted up to 1–3 minutes. She is healthy otherwise and is not on any medication. The family has no history of any cardiac disorder. On examination, she is found to have a heart rate of 156 beats/min. ECG interpretation shows narrow QRS complex and inverted P waves.

- How will you approach?
- What can be the potential diagnosis?

Decoding: The patient has symptoms of shortness of breath, and palpitation. The ECG picture shows narrow QRS complex tachycardia with a heart rate of 156 beats/min. On taking history, the patient had previous similar episodes of irregular heart rate with palpitation and sweating. The rhythm is suggestive of supraventricular tachycardia.

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CLINICAL VIGNETTE

Mr A, a 60-year-old man with a history of hypertension, was brought to the emergency department with complaints of palpitations, dizziness, and mild chest discomfort for the past few hours. On examination, his pulse was irregularly irregular at 130 bpm, and blood pressure was 100/60 mm Hg. ECG showed absence of P waves with irregular R-R intervals, confirming atrial fibrillation (AF). Nursing problems identified included decreased cardiac output related to ineffective atrial contractions, risk of thromboembolism, activity intolerance due to fatigue, and anxiety. Nursing interventions focused on continuous ECG monitoring, administering prescribed antiarrhythmic and anticoagulant medications, monitoring for signs of embolic events such as sudden limb pain or neurological changes, advising rest and avoidance of caffeine or stimulants, and educating the patient about medication compliance and pulse monitoring at home. Emotional reassurance was provided to alleviate fear of cardiac complications. Expected outcomes included stabilization of heart rhythm, maintenance of adequate cardiac output, prevention of complications like stroke, and improved patient understanding of long-term management.

SUMMARY

The electrocardiograph is the representation of the electrical activity of heart and is done as a primary investigation to rule out any cardiac disorder. The waveform includes P wave, PR interval, QRS Complex, ST segment, T wave and sometimes U wave. Advanced knowledge and critical evaluation of ECG is required to decode and strengthen the diagnosis. Majority of arrhythmias can be ruled out by looking at the strip thoroughly. A systematic approach involves analysis of rate, rhythm, intervals and QRS Complex. The further treatment depends on the final diagnosis.

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