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Jerash University

Faculty of Nursing

Major: Nursing

COURSE SYLLABUS

Critical Care Nursing Clinical

Course File Copy

This Course Syllabus is to be kept in the course file for this course. All changes, update and/or modifications should be reflected on the form, and should be kept in the Course Quality Assurance Form.

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Faculty	**Faculty of Nursing**
Department	**Nursing**

Course Code	**801468**
Course Title	**Critical Care Nursing Clinical**
Pre-co requisites	**0801319+0801317**
Credit Hours	**3 hours/ week**
Coordinator	**Dr. Rana Al Awamleh**
Instructor/s	**Mrs. Doaa Ayasrah**
Office Hours	**3 hours/week**
Email	**rana@jpu.edu.jo**

Course Description	**This course is designed to provide nursing students with the skills required to care competently and safely for critically ill patient. It focuses on having the students expand their knowledge base and master critical care nursing psychomotor skills associated with assessment and provision of comprehensive nursing care for patient with acute life threatening conditions and attitudes through reflection in and on action in clinical settings.** **It also focuses on the application of immediate rapid and accurate nursing assessment and provision of quality nursing care according to priorities. It enable the students to provide comprehensive quality nursing care for critically ill patient with different body system alterations, with different types of invasive devices and with different types of machines encountered in critical care settings and to understand the critical care environment in which practice occurs in order to provide care for the critically ill patients in the different critical care settings.** **Nursing process is used as an approach of providing holistic patient care .Critical thinking, clinical judgment, decision making and team work are emphasized in this course.**
Textbook(s) & Supplementary Materials	**Primary: 1. AACN Essentials of Critical Care Nursing, 4th Edition, 2019 by Suzanne M. Burns (Author), Sarah A. Delgado (Author).** **2. Introduction to Critical Care Nursing 8th Edition, 2021 by Sole PhD RN CCNS CNL FAAN FCCM, Mary Lou (Author), Klein MSN RN APRN-BC CCRN FAHA, Deborah Goldenberg (Author), Moseley PhD RN CCNS, Marthe J. (Author)**

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	Optional: **American association of critical care nurses. www.aacn.org** **End of life education center. www.aacn.nche.edu/elnec**
Objectives	1. Demonstrate the ability to assess critically ill patients with the different invasive devices & machines and their families 2. Provide comprehensive nursing care for the critically ill patients with the different acute and life threatening conditions and their families. 3. Demonstrate the most commonly used critical care nursing procedures 4. Demonstrate the routine care for the most commonly inserted invasive devices 5. Demonstrate the routine care for the most commonly used machines
Intended Student Learning Outcomes (ISLOs) Addressed by the Course	**At the completion of the course the student will be able to:** **Knowledge and Understanding** 1. Discuss the scope of practice in critical care units. 2. Recognize ethical and legal issues relevant to critically ill patients in ICU. 3. Identify critically ill patient' and their families' needs and problems 4. Recognize the principles of judgments and limitations of practice in critical care units. **Intellectual Skills** 1. Assess the needs of critically ill patient. 2. Identify the appropriate action for critical situations. 3. Prioritize actions according to the needs of the situation utilizing critical thinking process. 4. Integrate knowledge from nursing science, medicine and other sciences in nursing care of critically ill patient 5. Ensure that the available resources in critical care unit and complete personnel are used efficiently and effectively. 6. Assist consumers and health care team when taking decisions. **Professional and practical skills** 1. Monitor hemodynamic, oxygenation, blood chemistry and acid base balance of critically ill patient.

	2. Develop technical skills necessary to function competently within critical care as a professional nurse. 3. Demonstrate technical skills in dealing with advanced technology in critical care setting. 4. Implement nursing process to provide high quality nursing care to critical care patient. **General and transferable skills** 1. Communicate effectively with patients, their families and other colleagues 2. Develop confidence in clinical decision-making in the critical care setting. 3. Collaborate in a respectful manner with peers within critical care setting. 4. Develop leadership skills. 5. Practice within legal and boundaries.
	Intended Student Learning Outcomes(ISLOs) and Objective Matrix
Objectives	**ISLOs**
Objective 1	Knowledge: 2,3,4/ Intellectual: 1/ Professional: 1
Objective 2	All
Objective 3	Intellectual: 2, 3,4,5,6/ Professional: All/ General: 4
Objective 4	Knowledge: 1, 3/ Intellectual 3,4/ Professional: All/ General: 1,2,3,5
Objective 5	Intellectual 4,5/ Professional 2,3

Assessment & Evaluation Plan for the Course*	Nursing Care Plan	20 Marks
	Attendance and performance evaluation	10 Marks
	Mid-term Exam	20 Marks
	Case study	20 Marks
	Final Exam	30 Marks

Policies	**1. Class Attendance:** • Absence from lab sessions or hospital settings shall not exceed 15%. • One day absent will lead to two grades decrease in score of professionalism in addition to decrease in the total score of the student. • Students who exceed the 15% limit without a medical or emergency excuse acceptable to and approved by the Dean of the faculty shall not be allowed to take the final examination and shall receive a mark of zero for the course. **2. Exams:** 1. No bulky jackets (sweat shirts or small sweaters are allowed), no hats, caps, or sunglasses are to be worn while testing.

	2. No talking; no phones or iPods. 3. Cell phones are to be turned off before you enter the testing area. 4. Keep eyes on paper; once you leave the testing room-no reentering 5. Do not bring any bags, purses or backpacks in the testing area, 6. Place belongings in front of the room.
Method of Teaching	• Orientation Lab, discussion, demonstration and re-demonstration and role play. • Role modeling, clinical practices, reflection in action in clinical setting, reflection on clinical patient round, post clinical conference, assignment ,seminar and quiz, reflection on clinical documents, • Exercises on Glasgow Coma Scale, sedation scale, ABG interpretation, ECG strips analysis and dysrrhythmia interpretation, infusion rate & drug dosage calculation. • Practice under supervision in the clinical settings.

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CALENDAR & OUTLINE OF TOPICS

WEEK	Skills
1,2	Course Introduction and Overview
3	ECG and CPR
4	ABGs and Hemodialysis
5	
6	Mechanical ventilation, Chest tube, Central line
7	
8	Care of patient with Acute MI/Unstable Angina Care of patient pre-/post- cardiac surgery Care of patient with CHF
9	
10	
11	Hemodynamic Monitoring interpretation, Care of patients with TPN or NGT
12	Care of patients with DKA, Insulin pump
13	Spinal Cord Injury, Drug Overdose, Acute Head injury
14	

Appendices

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Critical Care Practicum

Case Study

Student Name: ---------- I.D. No: ----------

Date: ----------

The main purpose of this assignment is to connect the theoretical part that the student learned in the theory class with the real case that the student encounters during his clinical practice. Moreover, this assignment encourages the student to utilize the research findings in his clinical practice. Therefore, the student expected to identify one research study that is relevant to his case and presented his case and the research findings to his group. The research report is expected to be recent (within the last 5 years). Nursing studied are encouraged. However, medical studies are also accepted.

- ***Demographics:***

Client's Initials: ---------- Age: ----------

Gender: ---------- Admission Date: ----------

Ward: ---------- Medical Diagnosis: ----------

- ***Definition of the disease:***

- ***Background and Etiology of the disease:***

In your patient	In text book

- ***Clinical manifestations:***

In your patient	In text book

- ***History:(Past and family):***

- ***Diagnostic procedures and tests:***

Date	Type	Results and Interpretation	Nursing consideration

- ***Medications:***

Name of Drug	Action and Classification	Dose	Route	Frequency	Main S/E & Ng considerations

- ***Nursing Process:***

Diagnosis (prioritize)	Plan / Objectives	Interventions and Rational	Evaluation

- ***Medical/Surgical/ Nursing management and treatment:***

Management	Rational

- ***Research studies (related to the case subject):***

- ***Study:***

- ***Nursing implications for practice, education and research:***

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Case Study Evaluation Form

	Items	0	1
	***Written Assignment:**		
1	Original format of case study sheets & hand writing		
2	Clear writing manner		
3	Writing correct dictation.		
4	Content (concise, simple, clear, based on scientific knowledge)		
5	Organized in systematic sequence		
6	Correct nursing process and prioritized		
7	Comprehensive health assessment, nursing process and pathophysiology		
8	Present a list of references / sources of research study part.		
9	Focus on nursing role		
10	Time compliance in submitting the assignment		
	***Oral Presentation:**		
1	Introduction of self & item		
2	Manner of speech (clear, audible)		
3	Clarifies technical terminology		
4	Use clear & relevant examples		
5	Speech based on scientific knowledge		
6	Attract audience attention		
7	Control group participation		
8	Discuss the topic according to the written material		
9	Give chance for interaction, asking questions for participation		
10	Develop conclusion related to purpose of presentation		
	Total Grade: 20 points (…../20)		

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Critical Care Practicum

Nursing Care Plan

Student Name: _____ I.D. No: _____

Date: _____

Each student is responsible for completing one nursing process, (one nursing care plan during the semester) within the framework of the nursing process, the nursing care plan should reflect acute and current psychological and physiological responses, learning needs of critically ill patients, and expected health problems post-hospitalization. Interventions and strategies should reflect application of theoretical knowledge from nursing and other disciplines. It is the student's responsibilities to complete the one nursing care plane before the dead line date, it is also should reflect critical health disruption in core body system; cardiovascular, respiratory, nervous and multisystem organ failure.

- ***Health Assessment:***

  - **Demographics:**

    Patient name: ---------- Age: ---------- Sex: ----------

    Marital status: ---------- Ward: ---------- Room/bed: ----------

Date of Admission: ---------- Occupation: ----------

Source of Data: ----------

- **Health characteristics:**

Ht: ---------- Wt: ---------- LOC: ----------

Orientation: ---------- Medical Diagnosis: ----------

Smoking: ---------- Allergies: ----------

Surgical interventions: ----------

Hemodynamic baseline:

Temp: ---------- pulse: ---------- Resp: ---------- BP: ----------

CVP: ----------

- **Activity/ Exercise Pattern:**

Activity	1	2	3
Feeding			
Bathing			
Dressing/Grooming			
Toileting			
Mobility			

(Use Codes: 1= Independent, 2= Needs assistance, 3= Dependent).

- **Past History:**

- **Present complain:**

- **Family History:**

- **Physical exam for the most affected systems:**

- **Subjective Data:**

- **Objective Data:**

Review of problematic systems:

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- ***Diagnostic procedures and tests:***

Date	Type	Results and Interpretation	Nursing consideration

- ***Medications:***

Name of Drug	Action and Classification	Dose	Route	Frequency	Main S/E & Ng Consideration

- ***Intake and Output:***

Time	Intake type and amount	Time	Output

- ***Nursing Process:***

Diagnosis (prioritize)	Plan / Objectives	Interventions and Rational	Evaluation

- ***Discharge plan:***

Actual / Potential problems	Suggestion for solution

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Critical Care Practicum

Final Exam Evaluation Sheet

Student Name _____ I.D. NO_____

Clinical instructor _____

Clinical area _____

Scale:

91-100 Excellent

81-90 Very good

71-80 Good

51-70 Poor

Less than 50 Failed

NO	Items	Maximum Score	Student Score
1.	**Assignments:** • Submission on time. • Clear. • Realistic.	1 0.5 0.5	
2.	**General appearance:** • Proper uniform, name tag, clean and tidy. Proper shoes and head cover. • Pens (blue and red), Stethoscope and scissors.	1 1	
3.	**Communication skills :** • Introduce him/her self. • Use clear, concise statement. • Harmony between verbal and non verbal communication. • Call patient by name. • Ability to listen.	0.5 0.5 0.5 0.5 0.5	
4.	**Medical diagnosis and pathophysiology:** • Identify patient medical diagnosis. • Properly discuss pathophysiology.	1 3	
5.	**Nursing assessment:** • Able to perform physical assessment for the patient. • Able to collect health history. • Able to gather subjective data. • Able to collect objective data.	2 1 0.5 0.5	
6.	**Diagnostic tests:** • List the major diagnostic tests for patient. • Recognize the normal/abnormal result. • Know the preparation for each test. • Interpretation for each result.	1.5 1 0.5 1.5	

7.	**Nursing diagnosis:** • Identify at least 3 nursing diagnosis based on priorities. • Terminology. • Derived from subjective and objective data.	3 1 1	
8.	**Planning:** • Able to identify at least 3 goals according to the nursing diagnosis. • Able to classify the goals into short and long term goals.	1.5 1	
9.	**Nursing intervention:** • Implement the proper nursing interventions based on priorities. • Terminology for each intervention. • Rational.	2 0.5 0.5	
10.	**Evaluation:** • Indicate how and why objectives were achieved or not achieved.	0.5	
	Total	30	

Instructor Signature----------

Student Signature----------

Date: ----------

Comments: ----------

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CRITICAL CARE

Critical Care Nursing Manual

Dr. Rana Al Awamleh

Jerash University

Faculty of Nursing

2020/2021

Myocardial Infarction (MI)

- Myocardial infarction (MI) or acute myocardial infarction (AMI), commonly known as a heart attack, is the interruption of blood supply to part of the heart, causing some heart cells to die.
- Either one of the following criteria satisfies the diagnosis for an acute, evolving, or recent myocardial infarction:

1. Typical rise and gradual fall (troponin) or more rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis with at least one of the following:

a. Ischemic symptoms

b. Development of pathologic Q waves on the electrocardiogram

c. Electrocardiographic changes indicative of

ischemia (ST segment elevation or depression)

d. Coronary artery intervention (e.g., coronary angioplasty).

2. Pathological findings of an acute myocardial infarction

Pathophysiological Principles

- Most patients who sustain an MI have coronary atherosclerosis.
- The thrombus formation occurs most often at the site of an atherosclerotic lesion, thus obstructing blood flow to the myocardial tissues.
- Plaque rupture is believed to be the triggering mechanism for the development of the thrombus in most patients with an MI.
- When the plaques rupture, a thrombus is formed at the site that can occlude blood flow, thus resulting in an MI.
- Pathophysiological Principles
- Irreversible damage to the myocardium can begin as early as 20 to 40 minutes after interruption of blood flow.
- The dynamic process of infarction may not be completed, however, for several hours.
- Necrosis of tissue appears to occur in a sequential fashion.

- Reimer and associates demonstrated that cellular death occurs first in the subendocardial layer and spreads like a "wavefront" throughout the thickness of the wall of the heart.
- Using dogs, they showed that the shorter the time between coronary occlusion and coronary reperfusion, the greater the amount of myocardial tissue that could be salvaged.
- Pathophysiological Principles
- a substantial amount of myocardial tissue can be salvaged if flow is restored within 6 hours after the onset of coronary occlusion.
- Assessment HISTORY
- Patients with MI describe a heaviness, squeezing, choking, or smothering sensation.
- Patients often describe the sensation as "someone sitting on my chest."
- The substernal pain can radiate to the neck, left arm, back, or jaw.
- Unlike the pain of angina, the pain of an MI is often more prolonged and unrelieved by rest or sublingual nitroglycerin.
- Associated findings on history include nausea and vomiting, especially for the patient with an inferior wall MI.
- These gastrointestinal complaints are believed to be related to the severity of the pain and the resulting vagal stimulation.

PHYSICAL EXAMINATION

- Patients usually appear restless and in distress.
- The skin is warm and moist.
- Breathing may be labored and rapid. Fine crackles, coarse crackles, or rhonchi may be heard when auscultating the lungs.
- an increased blood pressure related to anxiety or a decreased blood pressure caused by heart failure.
- The heart rate may vary from bradycardia to tachycardia.

- When the patient is placed in the left lateral decubitus position, abnormalities of the precordial pulsations can be felt. These abnormalities include a lack of a point of maximal impulse or the presence of diffuse contraction.
- On auscultation, the first heart sound may be diminished as a result of decreased contractility.
- A fourth heart sound is heard in almost all patients with MI, whereas a third heart sound is detected in only about 10% to 20% of patients.
- Transient systolic murmurs may be heard
- After about 48 to 72 hours, many patients acquire a pericardial friction rub
- Patients with right ventricular infarcts may present with jugular vein distension, peripheral edema, and an elevated central venous pressure.

DIAGNOSTIC TESTS

The Electrocardiogram

Ischemia:

- On the ECG, myocardial ischemia results in T-wave inversion or ST segment depression in the leads facing the ischemic area.
- The inverted T wave representative of ischemia is symmetrical, relatively narrow, and somewhat pointed.
- ST segment depressions of 1 to 2 mm or more for a duration of 0.08 second may indicate myocardial ischemia.
- Ischemia also should be suspected when a flat or depressed ST segment makes a sharp angle when joining an upright T wave rather than merging smoothly and imperceptibly with the T wave

Injury:

- The injury process begins in the subendocardial layer and moves throughout the thickness of the wall of the heart like a wave.
- If the injury process is not interrupted, it eventually results in a transmural MI.

- On ECG, the hallmark of acute myocardial injury is the presence of ST segment elevations.
- In the normal ECG, the ST segment should not be elevated more than 1 mm in the standard leads or more than 2 mm in the precordial leads.
- With an acute injury, the ST segments in the leads facing the injured area are elevated.
- The elevated ST segments also have a downward concave or coved shape and merge unnoticed with the T wave

Infarction: When myocardial injury persists, MI is the result.

- During the earliest stage of MI, known as the hyperacute phase, the T waves become tall and narrow. This configuration is referred to as hyperacute or peaked T waves.
- Within a few hours, these hyperacute T waves invert.
- Next, the ST segments elevate, a pattern that usually lasts from several hours to several days.
- In addition to the ST segment elevations in the leads of the ECG facing the injured heart, the leads facing away from the injured area may show ST segment depression. This finding is known as reciprocal ST segment changes. Reciprocal changes are most likely to be seen at the onset of infarction, but their presence on the ECG does not last long. Reciprocal ST segment depressions may simply be a mirror image of the ST segment elevations. However, others have suggested that reciprocal changes may reflect ischemia due to narrowing of another coronary artery in other areas of the heart.
- The last stage in the ECG evolution of an MI is the development of Q waves, the initial downward deflection of the QRS complex.
- Q waves represent the flow of electrical forces toward the septum. Small, narrow Q waves may be seen in the normal ECG in leads I, II, III, aVR, aVL, V5, and V6.
- Q waves compatible with an MI are usually 0.04 second (one small box) or more in width or one-fourth to one-third the height of the R wave.
- Q waves indicative of infarction usually develop within several hours of the onset of the infarction, but in some patients may not appear until 24 to 48 hours after the infarction.
- Within a few days after the MI, the elevated ST segments return to baseline. Persistent elevation of the ST segment may indicate the presence of a ventricular aneurysm.

- The T waves may remain inverted for several weeks, indicating areas of ischemia near the infarct region. Eventually, the T waves should return to their upright configuration.
- The Q waves do not disappear and therefore always provide ECG evidence of a previous MI.
- Q waves indicate tissue necrosis and are permanent. A pathologic Q wave is one that is greater than 3 mm in depth or greater than one-third the height of the R wave.
- To detect posterior wall abnormalities, three of the precordial electrodes are placed posteriorly over the heart, a view known as V7, V8, and V9.
- V7 is positioned at the posterior axillary line; V8 at the posterior scapular line; and V9 at the left border of the spine

Laboratory Tests

Creatine Kinase

- CK-MB appears in the serum in 6 to 12 hours, peaks between 12 and 28 hours, and returns to normal levels in about 72 to 96 hours.
- Serial samplings are performed every 4 to 6 hours for the first 24 to 48 hours after the onset of symptoms

Myoglobin: Myoglobin is an oxygen-binding protein found in skeletal and cardiac muscle. Myoglobin's release from ischemic muscle occurs earlier than the release of CK.

- The myoglobin level can elevate within 1 to 2 hours of acute MI and peaks within 3 to 15 hours.
- Because myoglobin is also present in skeletal muscle, an elevated myoglobin level is not specific for the diagnosis of MI. onsequently, its diagnostic value in detecting an MI is limited

Troponin. (troponin T and troponin I):

- Troponin I levels rise in about 3 hours, peak at 14 to 18 hours, and remain elevated for 5 to 7 days.
- Troponin T levels rise in 3 to 5 hours and remain elevated for 10 to 14 days

Management

EARLY MANAGEMENT

- The patient's history and 12-lead ECG are the primary methods used to determine initially the diagnosis of MI.
    - 1. Administer aspirin, 160 to 325 mg chewed.
    - 2. After recording the initial 12-lead ECG, place the patient on a cardiac monitor and obtain serial ECGs.
    - 3. Give oxygen by nasal cannula.
    - 4. Administer sublingual nitroglycerin (unless the systolic blood pressure is less than 90 mm Hg or the heart rate is less than 50 or greater than 100 beats/minute).
    - 5. Provide adequate analgesia with morphine sulfate. Provide adequate analgesia with morphine sulfate.

Thrombolytic Therapy

- Thrombolytic drugs lyse coronary thrombi by converting plasminogen to plasmin.
- Thrombolytic therapy provides maximal benefit if given within the first 3 hours after the onset of symptoms.
- Significant benefit still occurs if therapy is given up to 12 hours after onset of symptoms.

Contraindications

■ Previous hemorrhagic stroke at any time; other stokes or cerebrovascular events within 1 year

■ Known intracranial neoplasm

■ Active internal bleeding (does not include menses)

■ Suspected aortic dissection

Thrombolytic Therapy Cautions/Relative Contraindications

■ Severe uncontrolled hypertension on presentation (blood pressure >180/110 mm Hg)

■ History of prior cerebrovascular accident or known intracerebral disease not covered in contraindications

■ Current use of anticoagulants in therapeutic doses (international normalized ratio [INR] ≥2:3); known bleeding diathesis

■ Recent trauma (within 2–4 weeks), including head trauma or traumatic or prolonged (>10 minutes) cardiopulmonary resuscitation (CPR) or major surgery (<3 weeks)

■ Noncompressible vascular punctures

■ Recent (within 2–4 weeks) internal bleeding

■ For streptokinase/anistreplase: prior exposure (especially within 5 days to 2 years) or prior allergic reaction

■ Pregnancy, Active peptic ulcer

Primary Percutaneous Transluminal (Coronary Angioplasty (PTCA))

- (PTCA) is an effective alternative to reestablish blood flow to ischemic myocardium.
- Primary PTCA is an invasive procedure in which the infarct-related coronary artery is dilated during the acute phase of an MI without prior administration of thrombolytic agents.
- Primary PTCA may be an excellent reperfusion alternative for patients ineligible for thrombolytic therapy.
- The nurse must carefully monitor the patient after a primary PTCA for evidence of complications.
- These complications can include retroperitoneal or vascular hemorrhage, other evidence of bleeding, early acute reocclusion, and late restenosis.

INTENSIVE AND INTERMEDIATE CARE MANAGEMENT

- Prophylactic antidysrhythmics during the first 24 hours of hospitalization are not recommended.
- IV nitroglycerin is continued for 24 to 48 hours.
- Daily aspirin is continued on an indefinite basis.
- Clopidogrel may be used for patients who are intolerant of aspirin.

- IV beta blocker therapy should be administered within the initial hours of the evolving infarction, followed by oral therapy. Beta blockers are one of the few pharmacological agents that have been shown to reduce morbidity and mortality in the patient with an MI. They reduce oxygen demand by decreasing the heart rate and contractility. They also increase coronary artery filling by prolonging diastole.
- Calcium channel blockers may be given to patients in whom beta blocker therapy is ineffective or contraindicated.
- Angiotensin-converting enzyme (ACE) inhibitors are administered to patients with anterior wall MI and to patients who have an MI with heart failure in the absence of significant hypotension. ACE inhibitors help prevent ventricular remodeling (dilation) and preserve ejection fraction.
- Heparin is given to patients undergoing percutaneous or surgical revascularization and for those receiving thrombolytic therapy with alteplase.
- Low–molecular-weight heparin should be used for patients with non–Q-wave MI

Hemodynamic Monitoring

- Use of a pulmonary artery catheter for hemodynamic monitoring is indicated in the patient with MI who has severe or progressive congestive heart failure or pulmonary edema, cardiogenic shock, progressive hypotension, or suspected mechanical complications.

MI Complications

- Hypotension/cardiogenic shock, Pericarditis , Congestive heart failure, Pericardial effusion, Deep venous thrombosis, Pulmonary embolism, Pulmonary embolism, VF, V-TAG, AV BLOCK

Nursing Diagnoses

- Acute Pain related to oxygen supply and demand imbalance
- Anxiety related to chest pain, fear of death, threatening environment
- Decreased Cardiac Output related to impaired contractility
- Activity Intolerance related to insufficient oxygenation to perform activities of daily living, deconditioning effects of bed rest

- Risk for Injury (bleeding) related to dissolution of protective clots

Nursing Interventions

Handle patient carefully while providing initial care, starting I.V. infusion, obtaining baseline vital signs, and attaching electrodes for continuous ECG monitoring.

- Maintain oxygen saturation greater than 92%.
- Administer oxygen by nasal cannula if prescribed
- Encourage patient to take deep breaths may decrease incidence of dysrhythmias by allowing the heart to be less ischemic and less irritable; may reduce infarct size, decrease anxiety, and resolve chest pain.
- Offer support and reassurance to patient that relief of pain is a priority.
- Administer sublingual nitroglycerin as directed; recheck BP, heart rate, and respiratory rate before administering nitrate therapy and 10 to 15 minutes after dose.
- Administer opioids as prescribed (morphine: decreases sympathetic activity and reduces heart rate, respirations, BP, muscle tension, and anxiety).
    - Use caution when administering opioids to elderly patients and those with chronic obstructive pulmonary disease, hypotension, or dehydration.
    - Remember that meperidine is rarely used because it can have a vagolytic effect and cause tachycardia, thus increasing myocardial oxygen demands.
- Obtain baseline vital signs before giving agents and 10 to 15 minutes after each dose. Place patient in a supine position during administration to minimize hypotension.
- Give I.V. nitroglycerin as prescribed. Monitor BP continuously with automatic BP machine (contraindicated with antithrombolytic therapy) or intra-arterially or every 5 minutes with auscultatory method while titrating for pain relief.

PULMONARY EMBOLISM PULMONARY

Originate primarily from deep venous of lower extremities Ilio -femoral thrombi femoral thrombi and pelvic veins pelvic veins appear to be the most clinically recognized source be the most clinically recognized source Air, amniotic fluid amniotic fluid and fat emboli fat emboli are rarer

The commonest scenario is a patient with a risk The commonest scenario is a patient with a risk factor who becomes factor who becomes breathless suddenly breathless suddenly,

Risk factors for deep venous Risk factors for deep venous thromboembolism : Triad of Triad of Virchow Virchow ’ s

- Endothelial injury
- Stasis
- Hypercoagulation status

The last 2 components predominate in venous thrombosis

Most PE are small, and infarcts are usually associated with small PE associated Small embolism may produce dyspnea, pleuritic chest pain, and occasionally , hemoptysis Small embolism will reach the periphery of the Small embolism will reach the periphery of the lung, sometimes producing lung, sometimes producing wedge -shaped shadow on CxR, and may cause , pulmonary infarction

A large embolism suddenly obstructing a major pulmonary vessel has marked effects on cardiac function, often associated with anterior anterior chest pain and collapse

Pulmonary infarct following a large embolism is less common

Chronic recurrent pulmonary embolism may develop hypertension and right ventricular failure

The most common risk factors identified

1. Immobilization
2. Surgery or trauma within the last 3 months
3. Increasing age
4. Malignancy

Idiopathic or primary venous thromboembolism should be further evaluated for the underlying abnormalities abnormalities For example: pancreatic cancer, prostate cancer, late in the course of breast, lung, uterine ,, or brain malignancies

Heart failure Heart failure and underlying cardiac disease underlying cardiac disease usually are associated with infarct formation usually are associated with infarct formation

SYMPTOMS AND SIGNS

The following frequency of S/S was noted: Dyspnea (84%) Pleuritic (74%) Anterior chest pain (68%) Cough (53%) Hemoptysis (30%) Asymptomatic (10%)

SIGNS Tachypnea (70%) Rales (51%) Tachycardia (30%) S4 (24%)

Symptoms of cardiac compromise are important and indicate a large PE, including collapse or and indicate a large PE, including collapse or dizziness on standing, severe dyspnea and severe anterior chest pain Signs of right heart strain should prompt early,

LAB ABNORMALITIES

Nonspecific:

leukocytosis, ESR elevation, LDH, , ESR elevation, LDH, SGOT elevation with normal SGOT elevation with normal bilirubin CK, CK -MB or Troponin

Troponin -I should be checked to rule out AMI

ABG usually revealed hypoxemia, , hypocapnia, with respiratory alkalosis collapse and hypotension due to massive pulmonary embolus may reveal combined respiratory and metabolic acidosis respiratory

ECG CHANGES

ECG most commonly revealed nonspecific ST segment and T wave changes, PE More severe right ventricular dysfunction severe right ventricular dysfunction with obstruction of obstruction of more than 50% more than 50% of pulmonary of pulmonary vasculature vasculature in a previously healthy patient may in a previously healthy patient may reveal T wave inversion in the T wave inversion in the precordial precordial leads V leads V 1 - V3 or P V3 or P pulmonale pulmonale, RAD, RBBB , RAD, RBBB S1Q3T3 (seen in only 25% of large PE: Stein et al) S1Q3T3 (seen in only 25% of large PE: Stein et al), or sinus tachycardia tachycardia can also be found. On occasion, PE can also be found. On occasion, PE can precipitate can precipitate atrial flutter flutter or AF

RADIOGRAPHY

Atelectasis pulmonary parenchymal abnormality is the most frequent is the most frequent radiographic abnormalities Westermark ’s sign Hampton’s hump

Evaluation of DVT It may support a diagnosis of It may support a diagnosis of thromboembolic disease in disease in patients in whom a patients in whom a Ventilation -Perfusion scan is Perfusion scan is nondiagnosed

Pulmonary Angiography

It should be performed whenever clinical data and whenever clinical data and noninvasive tests are equivocal or contradictory It is appropriate in patients with a It is appropriate in patients with a high probability high probability of PE by Ventilation by Ventilation -Perfusion scan, or if Perfusion scan, or if vena cava vena cava interruption or thrombolytic therapy is is being considered It may be the appropriate initial diagnostic tests in in patients with patients with unstable hemodynamics It remains

D –Dimer <500ng is powerful excluding tool for PE

PRIMARY TREATMENT

Supplemental oxygen for hypoxemia

Specific treatment is with intravenous heparin infusion intravenous heparin infusion following an initial bolus dose of 5000 units aPTT should be monitored should be monitored 4 -6 hours 6 hours after initiation, 6 after initiation, 6 -10 hours after any dosage change, then daily with a target hours after any dosage change, then daily with a target of 1.5 -2.5 times 2.5 times normal Heparin does not reduce acute mortality but Heparin does not reduce acute mortality but significantly significantly reduces further events reduces further events

LMWH could be used as an alternative choice of Heparin could be used as an alternative choice of Heparin

supportive treatments for If the PE is large, supportive treatments for hypotention hypotention or reduced CO or reduced CO should be given should be given IVF, Levophed, or Dopamine

Spiral CT should be performed if the patient is once Spiral CT should be performed if the patient is once stable

THROMBOLYTIC THERAPY

Thrombolytic therapy is used when there is therapy is used when there is significant cardiac compromise, hemodynamic changes changes not responding to IVF and vasopressor resuscitation

THROMBOLYTIC

Cerebral hemorrhage can occur in up to 1% of cases of cases It has been used successfully and safely in a It has been used successfully and safely in a pregnant woman and this is not a contraindication unless immediately postpartum immediately postpartum

DRUG REGIM EN

Streptokinas e 250 000 unit s in 20- 3- minutes followed by 100000 unit s/hour up t o 2 4 hours

T-pa 10 mg intravenously over 1-2 minutes followed by an infusion of 90 m g over 2 hours

SECONDARY PREVENTION

Oral anticoagulants (and LMWH Oral anticoagulants (and LMWH subcutaneously subcutaneously) Vena cava filters Vena cava filter

TREATMENT ANTICOAGULATION

LMWH keeps a PTT between 1.5 between with a therapeutic INR and a PTT is recommended Persistent oral warfarin should be prescribed for 3 months till the absence of risk factors.

How to read an Electrocardiogram (ECG).

Part One: Basic principles of the ECG. The normal ECG Introduction

The electrocardiogram (ECG) is one of the simplest and oldest cardiac investigations available, yet it can provide a wealth of useful information and remains an essential part of the assessment of cardiac patients.

With modern machines, surface ECGs are quick and easy to obtain at the bedside and are based on relatively simple electrophysiological concepts. However junior doctors often find them difficult to interpret.

This is the first in a short series of articles that aim to:

- Help readers understand and interpret ECG recordings.
- Reduce some of the anxiety juniors often experience when faced with an ECG.

Basic principles

What is an ECG?
An ECG is simply a representation of the electrical activity of the heart muscle as it changes with time, usually printed on paper for easier analysis. Like other muscles, cardiac muscle contracts in response to electrical*depolarisation* of the muscle cells. It is the sum of this electrical activity, when amplified and recorded for just a few seconds that we know as an ECG.

Basic Electrophysiology of the Heart (see Figure 1)
The normal cardiac cycle begins with spontaneous depolarisation of the sinus node, an area of specialised tissue situated in the high right atrium (RA). A wave of electrical depolarisation then spreads through the RA and across the inter-atrial septum into the left atrium (LA).

The atria are separated from the ventricles by an electrically inert fibrous ring, so that in the normal heart the only route of transmission of electrical depolarisation from atria to ventricles is through the atrioventricular (AV) node. The AV node delays the electrical signal for a short time, and then the wave of depolarisation spreads down the interventricular septum (IVS), via the bundle of His and the right and left bundle branches, into the right (RV) and left (LV) ventricles. Hence with normal conduction the two ventricles contract simultaneously, which is important in maximising cardiac efficiency.

After complete depolarisation of the heart, the myocardium must then *repolarise,* before it can be ready to depolarise again for the next cardiac cycle.

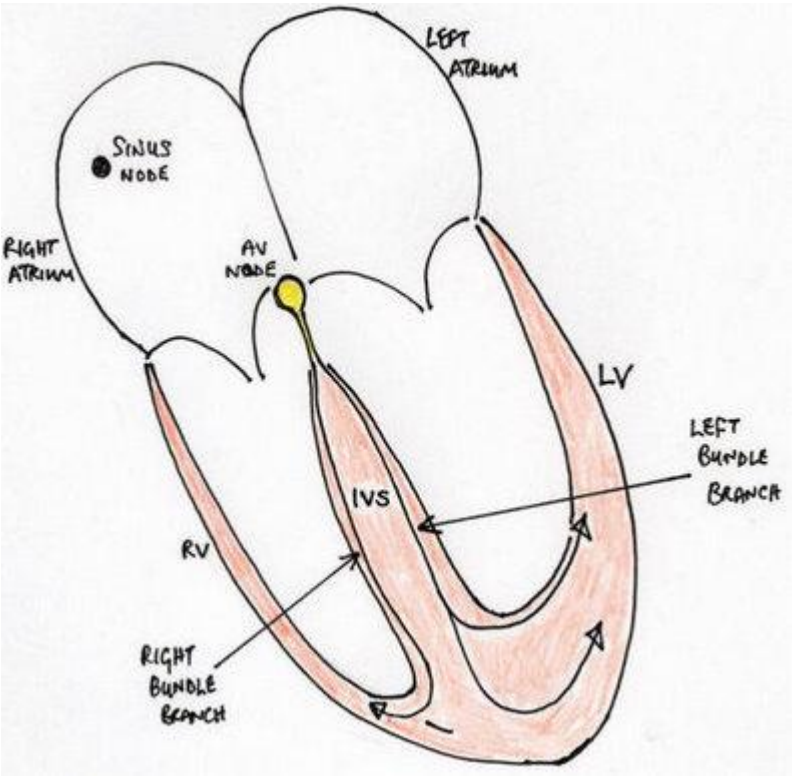


Figure 1. Basic electrophysiology of the heart

Electrical axis and recording lead vectors (see Figures 2 and 3)
The ECG is measured by placing a series of electrodes on the patient's skin – so it is known as the 'surface' ECG.

The wave of electrical depolarisation spreads from the atria down though the IVS to the ventricles. So the direction of this depolarisation is usually from the superior to the inferior aspect of the heart. The direction of the wave of depolarisation is normally towards the left due to the leftward orientation of the heart in the chest and the greater muscle mass of the left ventricle than the right. This overall direction of travel of the electrical depolarisation through the heart is known as the *electrical axis*.

A fundamental principle of ECG recording is that when the wave of depolarisation travels toward a recording lead this results in a positive or upward deflection. When it travels away from a recording lead this results in a negative or downward deflection.

The electrical axis is normally downward and to the left but we can estimate it more accurately in individual patients if we understand from which 'direction' each recording lead measures the ECG.

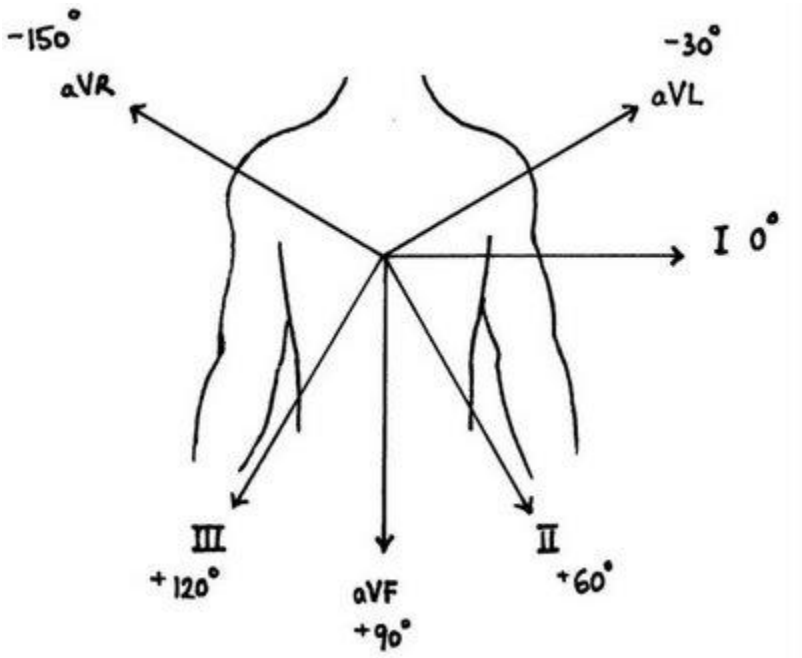


Figure 2. Orientation of the limb leads showing the direction from which each lead 'looks' at the heart

By convention, we record the standard surface ECG using 12 different recording lead 'directions,' though rather confusingly only 10 recording electrodes on the skin are required to achieve this. Six of these are recorded from the chest overlying the heart – *the chest or precordial leads*. Four are recorded from the limbs – *the limb leads*. It is essential that each of the 10 recording electrodes is placed in its correct position, otherwise the appearance of the ECG will be changed significantly, preventing correct interpretation.

The limb leads record the ECG in the coronal plane, and so can be used to determine the electrical axis (which is usually measured only in the coronal plane). The limb leads are called leads I, II, III, AVR, AVL and AVF. Figure 2 shows the relative directions from which they 'look' at the heart. A horizontal line through the heart and directed to the left (exactly in the direction of lead I) is conventionally labelled as the reference point of 0 degrees (0^{o}). The directions from which other leads 'look' at the heart are described in terms of the angle in degrees from this baseline.

The electrical axis of depolarisation is also expressed in degrees and is normally in the range from -30^{0} to $+ 90^{0}$. A detailed explanation of how to determine the axis is beyond the scope of this article but the principles mentioned here should help readers to understand the concepts involved.

The chest leads record the ECG in the transverse or horizontal plane, and are called V1, V2, V3, V4, V5 and V6 (see Figure 3).

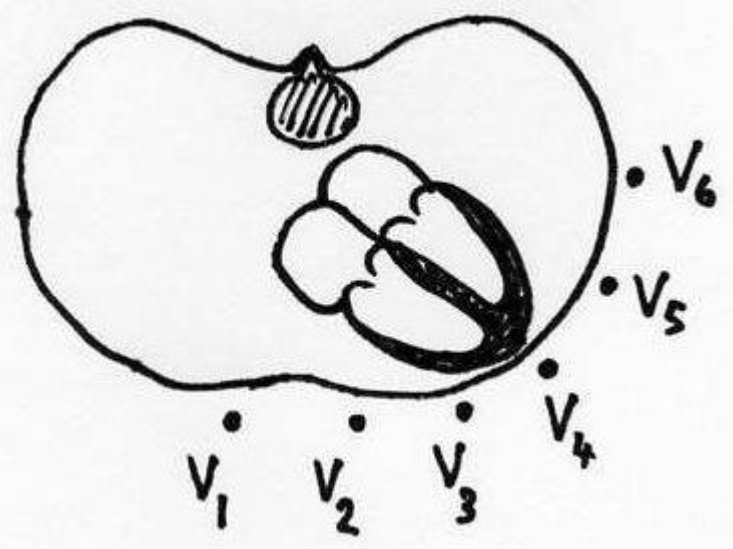


Figure 3. Transverse section of the chest showing the orientation of the six chest leads in relation to the heart

Voltage and timing intervals

It is conventional to record the ECG using standard measures for amplitude of the electrical signal and for the speed at which the paper moves during the recording. This allows:

- Easy appreciation of heart rates and cardiac intervals and
- Meaningful comparison to be made between ECGs recorded on different occasions or by different ECG machines.

The amplitude, or voltage, of the recorded electrical signal is expressed on an ECG in the vertical dimension and is measured in millivolts (mV). On standard ECG paper 1mV is represented by a deflection of 10 mm. An increase in the amount of muscle mass, such as with left ventricular hypertrophy (LVH), usually results in a larger electrical depolarisation signal, and so a larger amplitude of vertical deflection on the ECG.

An essential feature of the ECG is that the electrical activity of the heart is shown as it varies with time. In other words we can think of the ECG as a graph, plotting electrical activity on the vertical axis against time on the horizontal axis. Standard ECG paper moves at 25 mm per second during real-time recording. This means that when looking at the printed ECG a distance of 25 mm along the horizontal axis represents 1 second in time.

ECG paper is marked with a grid of small and large squares. Each small square represents 40 milliseconds (ms) in time along the horizontal axis and each larger square contains 5 small squares, thus representing 200 ms. Standard paper speeds and square markings allow easy measurement of cardiac timing intervals. This enables calculation of heart rates and identification of abnormal electrical conduction within the heart (see Figure 4).

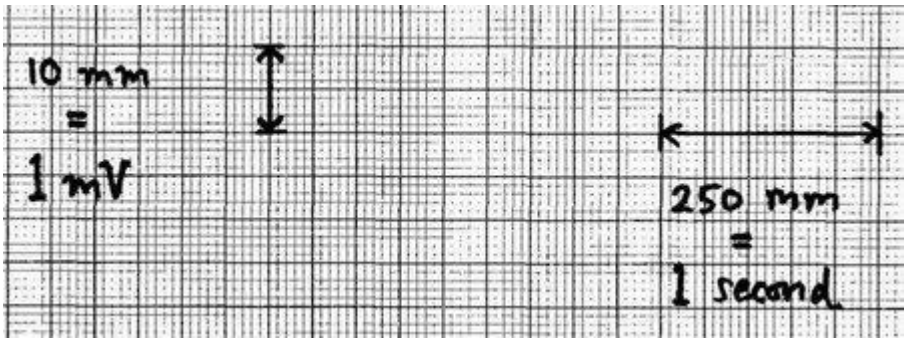


Figure 4. Sample of standard ECG paper showing the scale of voltage, measured on the vertical axis, against time on the horizontal axis

The normal ECG

It will be clear from above that the first structure to be depolarised during normal sinus rhythm is the right atrium, closely followed by the left atrium. So the first electrical signal on a normal ECG originates from the atria and is known as the **P wave**. Although there is usually only one P wave in most leads of an ECG, the P wave is in fact the sum of the electrical signals from the two atria, which are usually superimposed.

There is then a short, physiological delay as the atrioventricular (AV) node slows the electrical depolarisation before it proceeds to the ventricles. This delay is responsible for the PR interval, a short period where no electrical activity is seen on the ECG, represented by a straight horizontal or 'isoelectric' line.

Depolarisation of the ventricles results in usually the largest part of the ECG signal (because of the greater muscle mass in the ventricles) and this is known as the **QRS complex**.

- The Q wave is the first initial downward or 'negative' deflection
- The R wave is then the next upward deflection (provided it crosses the isoelectric line and becomes 'positive')
- The S wave is then the next deflection downwards, provided it crosses the isoelectric line to become briefly negative before returning to the isoelectric baseline.

In the case of the ventricles, there is also an electrical signal reflecting repolarisation of the myocardium. This is shown as the **ST segment** and the **T wave**. The ST segment is normally isoelectric, and the T wave in most leads is an upright deflection of variable amplitude and duration (see Figures 5 and 6).

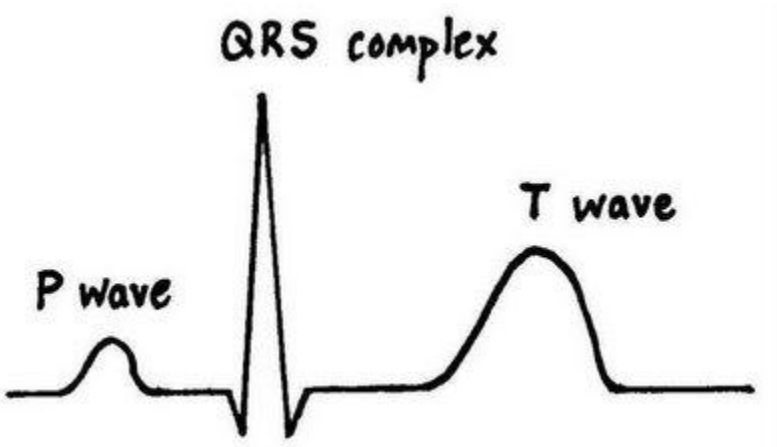


Figure 5. The major waves of a single normal ECG pattern

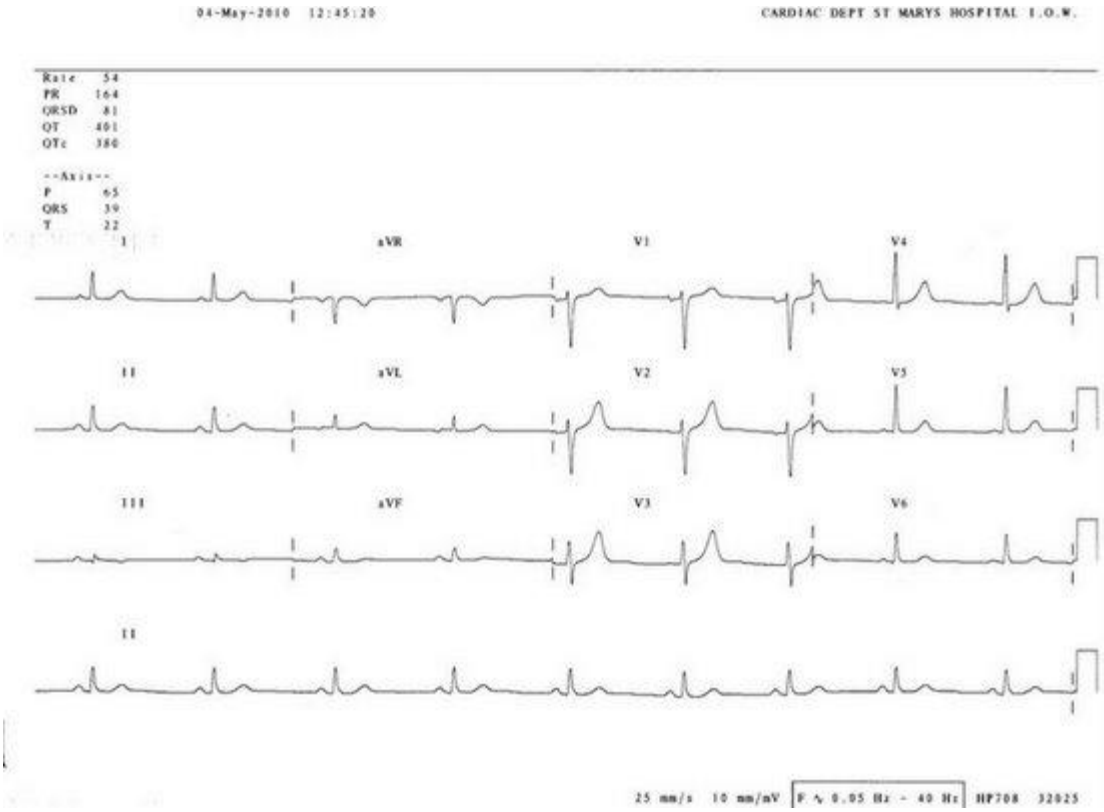


Figure 6. Example of a normal 12 lead ECG; notice the downward deflection of all signals recorded from lead aVR. This is normal, as the electrical axis is directly away from that lead

Normal intervals

The recording of an ECG on standard paper allows the time taken for the various phases of electrical depolarisation to be measured, usually in milliseconds. There is a recognised normal range for such 'intervals':

- **PR interval** (measured from the beginning of the P wave to the first deflection of the QRS complex). Normal range 120 – 200 ms (3 – 5 small squares on ECG paper).
- **QRS duration** (measured from first deflection of QRS complex to end of QRS complex at isoelectric line). Normal range up to 120 ms (3 small squares on ECG paper).
- **QT interval** (measured from first deflection of QRS complex to end of T wave at isoelectric line). Normal range up to 440 ms (though varies with heart rate and may be slightly longer in females)

Heart rate estimation from the ECG

Standard ECG paper allows an approximate estimation of the heart rate (HR) from an ECG recording. Each second of time is represented by 250 mm (5 large squares) along the horizontal axis. So if the number of large squares between each QRS complex is:

- 5 - the HR is 60 beats per minute.
- 3 - the HR is 100 per minute.
- 2 - the HR is 150 per minute.

Heart Rate and Pulse Rate

Heart rate is the number of QRS complexes present in a minute.

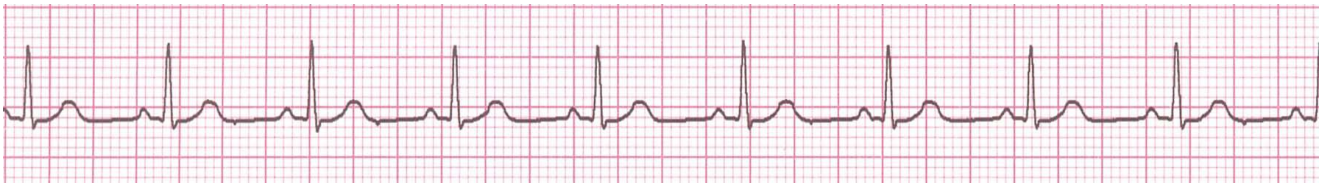
Pulse rate is the rate of perfusion of blood to the tissue

Heart rate is not always the same as pulse rate

Calculating Heart Rate

Six Second Count, multiplying the number of QRS complexes found over six seconds by a factor of 10 to get the QRS complexes found in a minute.

3 sec	3 sec



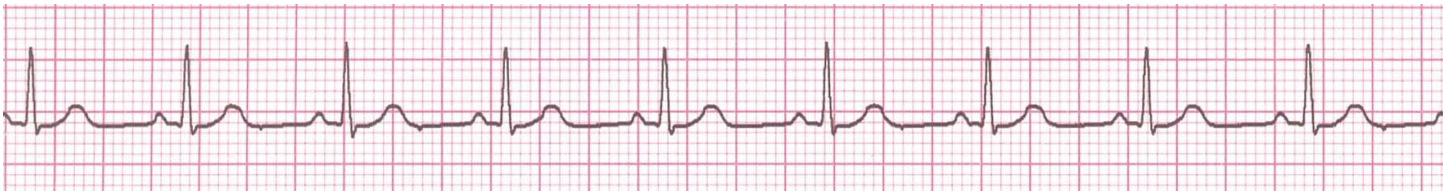
9x10=90

Triplicate the fastest method to figure a regular heart rate. Memorize the following numbers 300, 150, 100, 75, 60, 50

3 1 1

0 5 0 7 6 5 4 3 3 3 2

↓ 0 0 0 5 0 0 3 8 3 0 7

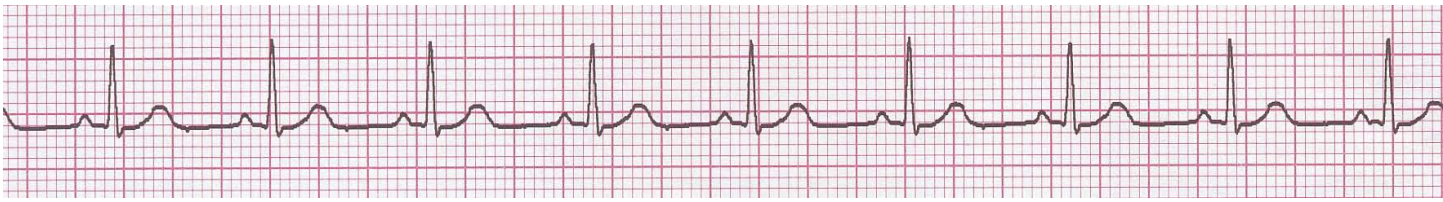


↑ ↑

Find an R wave that land on a bold line.

Count the # of large boxes to the next R wave. If the second R wave is 1 large box away the rate is 300, 2 boxes - 150, 3 boxes - 100, 4 boxes - 75, etc. *Approx. 1 box less than 100 = 95 bpm*

*Normal Sinus Rhythm



The electrical impulse is formed in the SA node and conducted normally.

This is the normal rhythm of the heart; other rhythms that do not conduct via the typical pathway are called arrhythmias.

- Rate 60 - 100 bpm (adult) age dependent in pediatrics
- Regularity regular
- P waves normal
- PR interval 0.12 - 0.20 s
- QRS duration 0.04 - 0.12 s

<u>Any deviation from above is sinus tachycardia, sinus bradycardia or an arrhythmia</u>

Arrhythmias

Arrhythmias can arise from problems in the:

Sinus node

- Firing to slow = Sinus bradycardia
- Firing to fast = Sinus tachycardia

Sinus tachycardia may be an appropriate response to stress

Atrial cells

- Fire occasionally from a focus = Premature Atrial Contraction (PAC's)
- Fire continuously due to a looping reentrant circuit = Atrial Flutter
- Fire continuously from multiple foci or
- fire continuously due to multiple micro re-entrants = Atrial Fibrillation

AV junction

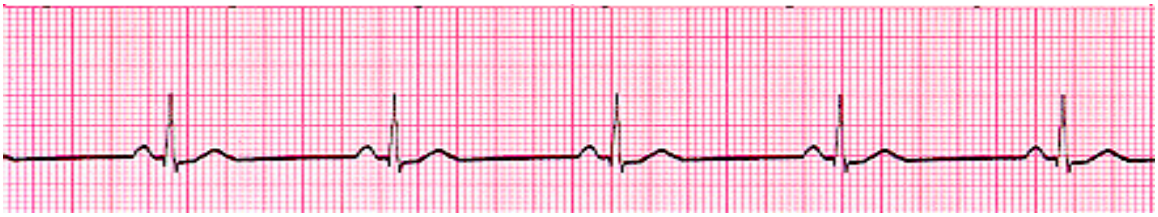
- Fire continuously due to a looping reentrant circuit = Supraventricular Tachycardia

- Block impulses coming from the SA node = AV Junctional Block

Ventricular cells

- Fire occasionally from 1 or more foci = Premature Ventricular Contractions (PVC's)
- Fire continuously from multiple foci = Ventricular Fibrillation
- Fire continuously due to a looping reentrant circuit = Ventricular Tachycardia

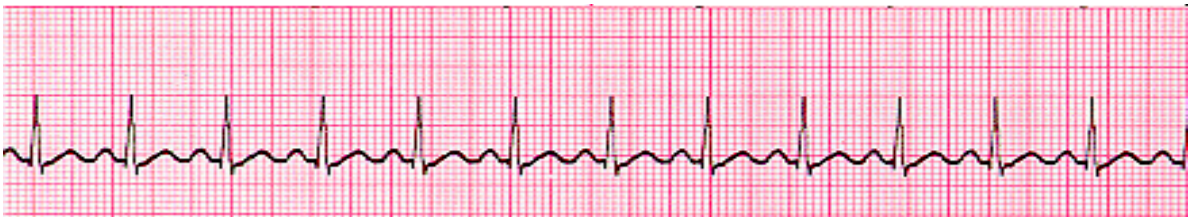
*Sinus Bradycardia: Looking at the ECG you'll see that:



A heart rate less than 60 beats per minute (BPM).

- Rhythm - Regular
- Rate - less than 60 beats per minute
- QRS Duration - Normal
- P Wave - Visible before each QRS complex
- P-R Interval - Normal
- Usually benign and often caused by patients on beta blockers

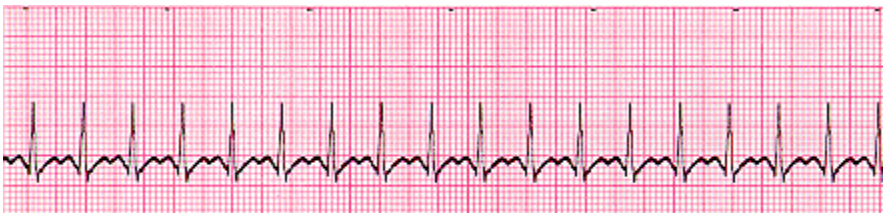
*Sinus Tachycardia: Looking at the ECG you'll see that:



- Rhythm - Regular
- Rate – Greater then norm for child's age
- QRS Duration - Normal
- P Wave - Visible before each QRS complex
- P-R Interval - Normal

- The impulse generating the heart beats are normal, but they are occurring at a faster pace than normal.

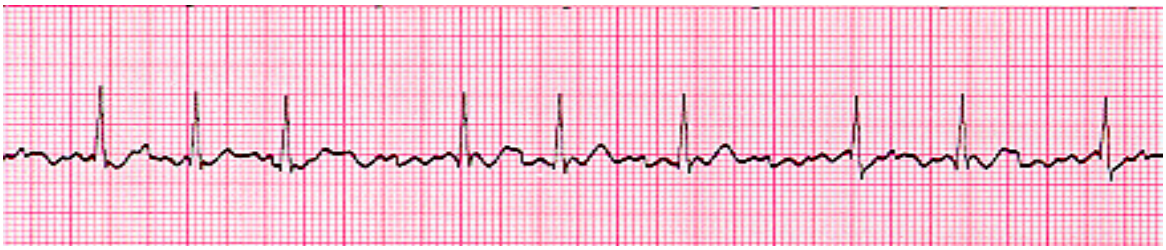
*Supraventricular Tachycardia (SVT): Looking at the ECG you'll see that:



A narrow complex tachycardia or atrial tachycardia which originates in the 'atria' but is not under direct control from the SA node.

- Rhythm - Regular
- Rate – Greater than 180
- QRS Duration - Usually normal
- P Wave - Often buried in preceding T wave
- P-R Interval - Depends on site of supraventricular pacemaker
- Impulses stimulating the heart are not being generated by the sinus node, but instead are coming from a collection of tissue around and involving the atrioventricular (AV) node

Atrial Fibrillation: Looking at the ECG you'll see that:

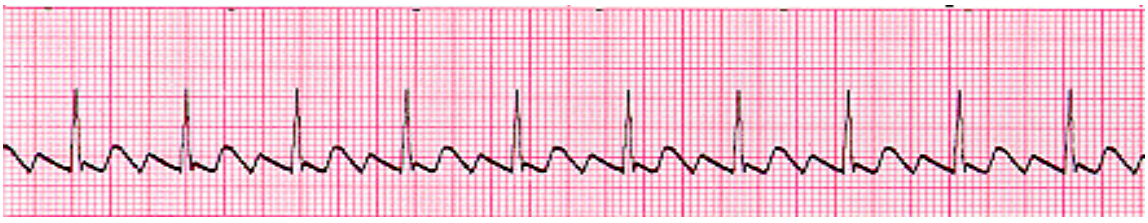


Many sites within the atria are generating their own electrical impulses, leading to irregular conduction of impulses to the ventricles that generate the heartbeat. This irregular rhythm can be felt when palpating a pulse.

- Rhythm - Irregularly irregular
- Rate – Atrial rate ranges from 350-600 per min. The ventricular response is irregularly irregular and may be fast or slow.
- QRS Duration - Usually normal
- P Wave - Not distinguishable as the atria are firing off all over

- P-R Interval - Not measurable
- The atria fire electrical impulses in an irregular fashion causing irregular heart rhythm

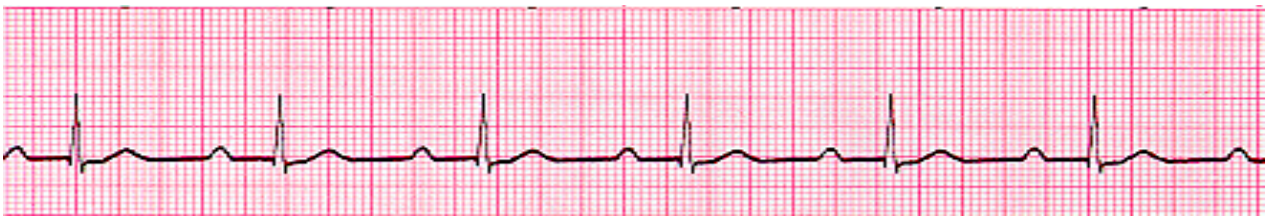
Atrial Flutter: Looking at the ECG you'll see that:



As with SVT the abnormal tissue generating the rapid heart rate is also in the atria, however, the atrioventricular node is not involved in this case.

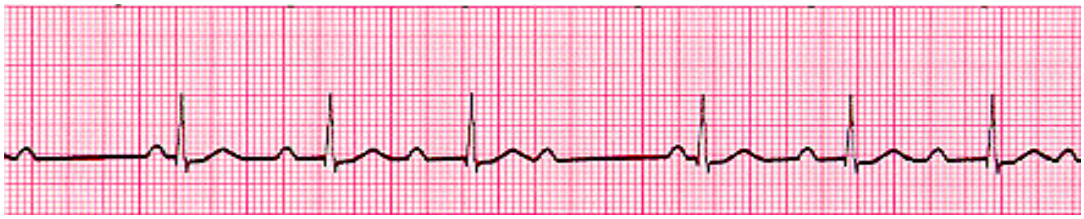
- Rhythm - Regular
- Rate – Atrial rate ranges 240 – 300 per min.
- QRS Duration - Usually normal
- P Wave - Replaced with multiple F (flutter) waves, usually at a ratio of 2:1 (2F - 1QRS) but sometimes 3:1
- P Wave rate - 300 beats per minute
- P-R Interval - Not measurable

1st Degree AV Block: Looking at the ECG you'll see that:



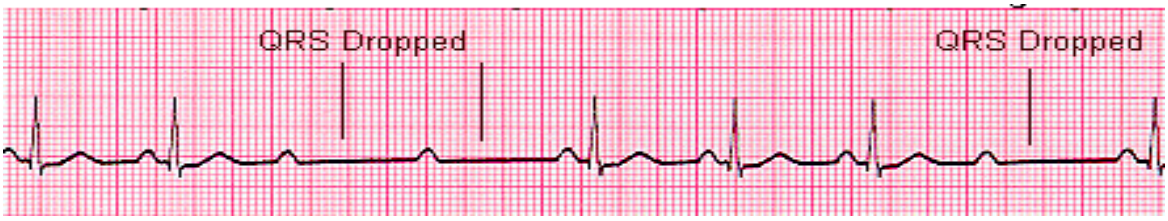
- Rhythm - Regular
- Rate - Normal
- QRS Duration - Normal
- P Wave - Ratio 1:1
- P Wave rate - Normal
- P-R Interval - Prolonged (>5 small squares)

2nd Degree Block Type 1 (Wenckebach): Looking at the ECG you'll see that:



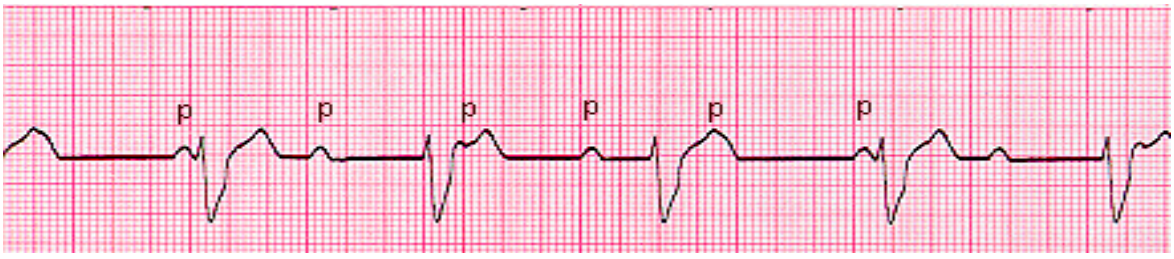
- Rhythm - Regularly irregular
- Rate - Normal or Slow
- QRS Duration - Normal
- P Wave - Ratio 1:1 for 2, 3 or 4 cycles then 1:0.
- P Wave rate - Normal but faster than QRS rate
- P-R Interval - Progressive lengthening of P-R interval until a QRS complex is dropped

2nd Degree Block Type 2: Looking at the ECG you'll see that:



- Rhythm - Regular
- Rate - Normal or Slow
- QRS Duration - Prolonged
- P Wave - Ratio 2:1, 3:1
- P Wave rate - Normal but faster than QRS rate
- P-R Interval - Normal or prolonged but constant

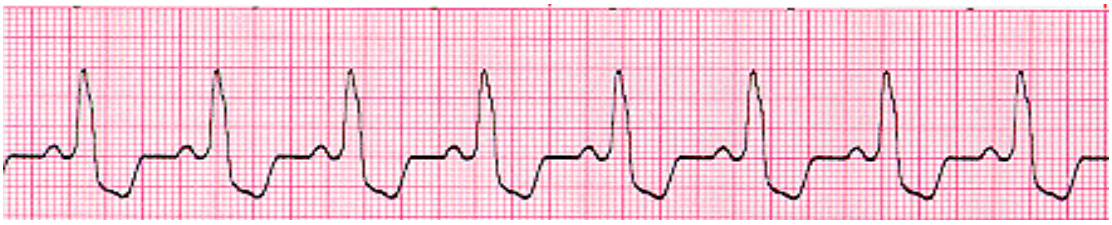
3rd Degree Block: Looking at the ECG you'll see that:



- Rhythm - Regular
- Rate - Slow

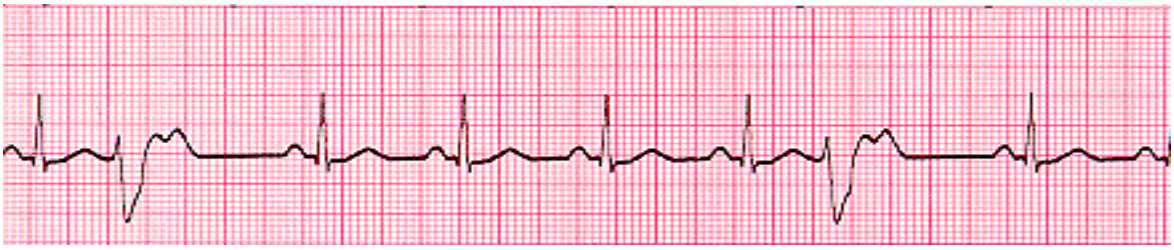
- QRS Duration - Prolonged
- P Wave - Unrelated
- P Wave rate - Normal but faster than QRS rate
- P-R Interval - Variation
- Complete AV block. No atrial impulses pass through the atrioventricular node and the ventricles generate their own rhythm

Bundle Branch Block: Looking at the ECG you'll see that:

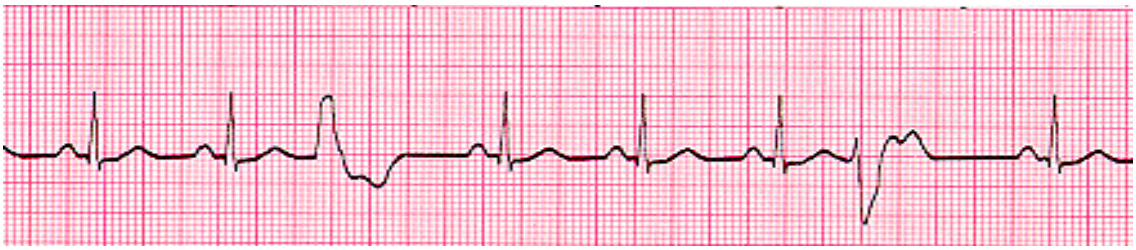


- Rhythm - Regular
- Rate - Normal
- QRS Duration - Prolonged
- P Wave - Ratio 1:1
- P Wave rate - Normal and same as QRS rate
- P-R Interval - Normal

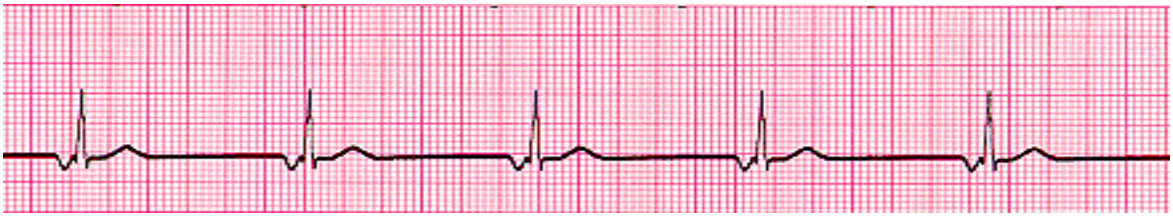
Premature Ventricular Complexes: Looking at the ECG you'll see that:



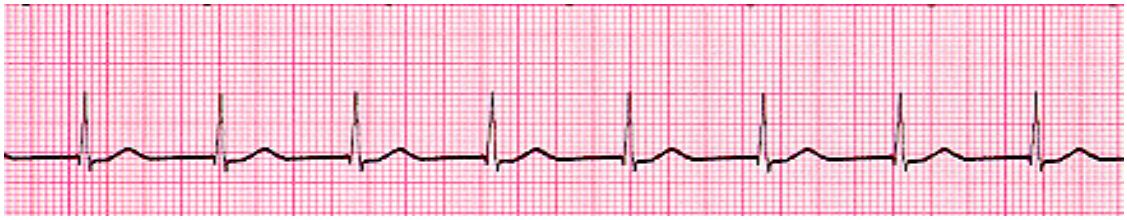
- Rhythm - Regular
- Rate - Normal
- QRS Duration - Normal
- P Wave - Ratio 1:1
- P Wave rate - Normal and same as QRS rate
- P-R Interval - Normal
- Also you'll see 2 odd waveforms, these are the ventricles depolarizing prematurely in response to a signal within the ventricles.(Above - unifocal PVC's as they look alike if they differed in appearance they would be called multifocal PVC's, as below)



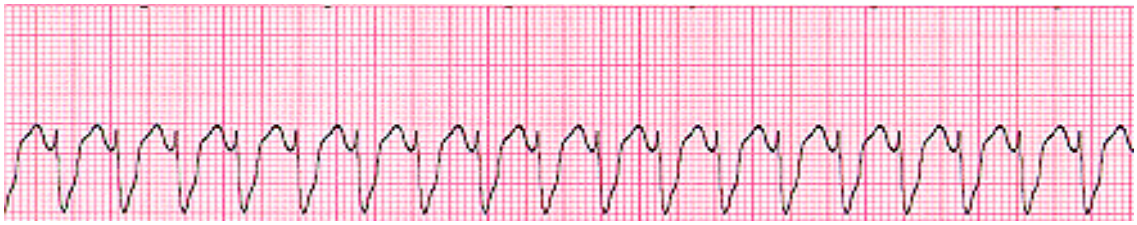
Junctional Rhythms Looking at the ECG you'll see that:



- Rhythm - Regular
- Rate - 40-60 Beats per minute
- QRS Duration - Normal
- P Wave - Ratio 1:1 if visible. Inverted in lead II
- P Wave rate - Same as QRS rate
- P-R Interval - Variable
- Below - Accelerated Junctional Rhythm



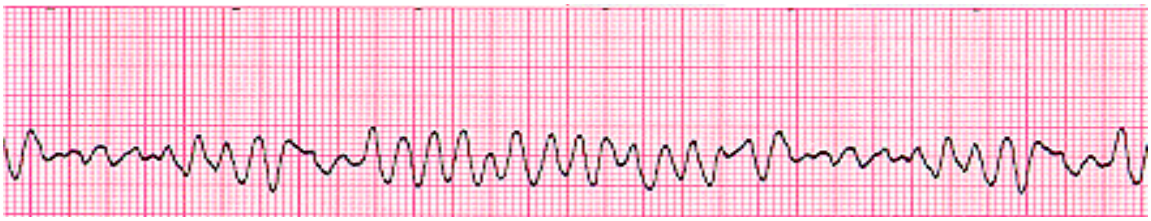
*Ventricular Tachycardia (VT) Looking at the ECG you'll see that:



- Rhythm - Regular

- Rate - 180-190 Beats per minute
- QRS Duration - Prolonged
- P Wave - Not seen
- Results from abnormal tissues in the ventricles generating a rapid and irregular heart rhythm. Poor cardiac output is usually associated with this rhythm thus causing the pt to go into cardiac arrest. Shock this rhythm if the patient is unconscious and without a pulse

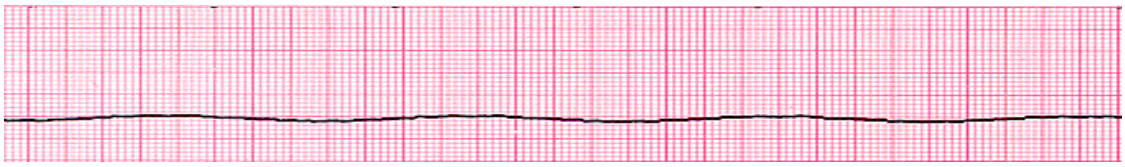
*Ventricular Fibrillation (VF)



Looking at the ECG you'll see that:

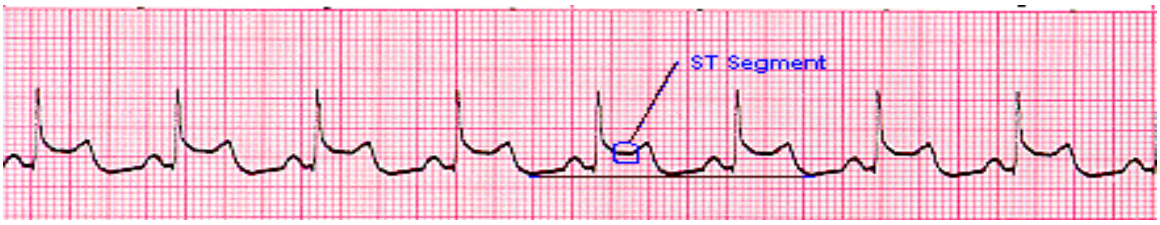
- Rhythm - Irregular
- Rate - 300+, disorganized
- QRS Duration - Not recognizable
- P Wave - Not seen
- This patient needs to be defibrillated!! QUICKLY

*Asystole



- Rhythm - Flat
- Rate - 0 Beats per minute
- QRS Duration - None
- P Wave - None
- Carry out CPR!!!

Myocardial Infarct (MI)



- Rhythm - Regular
- Rate - 80 Beats per minute
- QRS Duration - Normal
- P Wave - Normal
- S-T Element does not go isoelectric which indicates infarction

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Mechanical Ventilation

- Mechanical Ventilation is ventilation of the lungs by artificial means usually by a ventilator.
- A ventilator delivers gas to the lungs with either negative or positive pressure.

Purposes:

- To maintain or improve ventilation, & tissue oxygenation.
- To decrease the work of breathing & improve patient's comfort.

Indications:

1- Acute respiratory failure due to:

- Mechanical failure, includes neuromuscular diseases as Myasthenia Gravis, Guillain-Barré Syndrome, and Poliomyelitis (failure of the normal respiratory neuromuscular system)
- Musculoskeletal abnormalities, such as chest wall trauma (flail chest)
- Infectious diseases of the lung such as pneumonia, tuberculosis.

2- Abnormalities of pulmonary gas exchange as in:

- Obstructive lung disease in the form of asthma, chronic bronchitis or emphysema.
- Conditions such as pulmonary edema, atelectasis, pulmonary fibrosis.

- Patients who has received general anesthesia as well as post cardiac arrest patients often require ventilatory support until they have recovered from the effects of the anesthesia or the insult of an arrest.

Positive-pressure ventilators

- Positive-pressure ventilators deliver gas to the patient under positive-pressure, during the inspiratory phase.

Types of Positive-Pressure Ventilators

1- Volume Ventilators.

2- Pressure Ventilators

3- High-Frequency Ventilators

1- Volume Ventilators

- The volume ventilator is commonly used in critical care settings.
- The basic principle of this ventilator is that a designated volume of air is delivered with each breath.
- The amount of pressure required to deliver the set volume depends on :-

  - Patient's lung compliance

  - Patient–ventilator resistance factors.

- Therefore, peak inspiratory pressure (PIP) must be monitored in volume modes because it varies from breath to breath.
- With this mode of ventilation, a respiratory rate, inspiratory time, and tidal volume are selected for the mechanical breaths.

2- Pressure Ventilators

- The use of pressure ventilators is increasing in critical care units.
- A typical pressure mode delivers a selected gas pressure to the patient early in inspiration, and sustains the pressure throughout the inspiratory phase.

- By meeting the patient's inspiratory flow demand throughout inspiration, patient effort is reduced and comfort increased.
- Although pressure is consistent with these modes, volume is not.
- Volume will change with changes in resistance or compliance,
- Therefore, exhaled tidal volume is the variable to monitor closely.
- With pressure modes, the pressure level to be delivered is selected, and with some mode options (i.e., pressure controlled [PC], described later), rate and inspiratory time are preset as well.

3- High-Frequency Ventilators

- High-frequency ventilators use small tidal volumes (1 to 3 mL/kg) at frequencies greater than 100 breaths/minute.
- The high-frequency ventilator accomplishes oxygenation by the diffusion of oxygen and carbon dioxide from high to low gradients of concentration.

Types of ventilator settings (cycles)

1- Volume-cycled ventilator

- Inspiration is terminated after a preset tidal volume has been delivered by the ventilator.
- The ventilator delivers a preset tidal volume (VT), and inspiration stops when the preset tidal volume is achieved.

2- Pressure-cycled ventilator

- In which inspiration is terminated when a specific airway pressure has been reached.
- The ventilator delivers a preset pressure; once this pressure is achieved, end inspiration occurs.

3- Time-cycled ventilator

- In which inspiration is terminated when a preset inspiratory time, has elapsed.
- Time cycled machines are not used in adult critical care settings. They are used in pediatric intensive care areas.

Modes of Mechanical Ventilation

A- Volume Modes

B- Pressure Modes

A- Volume Modes

1- A- Volume Modes (Assist-control (A/C))

2- A- Volume Modes (Synchronized intermittent mandatory ventilation (SIMV))

1- Assist Control Mode A/C

- The ventilator provides the patient with a pre-set tidal volume at a pre-set rate.
- The patient may initiate a breath on his own, but the ventilator assists by delivering a specified tidal volume to the patient. Client can initiate breaths that are delivered at the preset tidal volume.
- Client can breathe at a higher rate than the preset number of breaths/minute
- The total respiratory rate is determined by the number of spontaneous inspiration initiated by the patient plus the number of breaths set on the ventilator.
- In A/C mode, a mandatory (or "control") rate is selected.
- If the patient wishes to breathe faster, he or she can trigger the ventilator and receive a full-volume breath.
- Often used as initial mode of ventilation
- When the patient is too weak to perform the work of breathing (e.g., when emerging from anesthesia).

Disadvantages: .....Hyperventilation,

2- Synchronized Intermittent Mandatory Ventilation (SIMV)

- The ventilator provides the patient with a pre-set number of breaths/minute at a specified tidal volume and FiO_2.
- In between the ventilator-delivered breaths, the patient is able to breathe spontaneously at his own tidal volume and rate with no assistance from the ventilator.
- However, unlike the A/C mode, any breaths taken above the set rate are spontaneous breaths taken through the ventilator circuit.
- The tidal volume of these breaths can vary drastically from the tidal volume set on the ventilator, because the tidal volume is determined by the patient's spontaneous effort.
- Adding pressure support during spontaneous breaths can minimize the risk of increased work of breathing.

- Ventilators breaths are synchronized with the patient spontaneous breathe. (No fighting)

- Used to wean the patient from the mechanical ventilator.
- Weaning is accomplished by gradually lowering the set rate and allowing the patient to assume more work

B- Pressure Modes

1- Pressure-controlled ventilation (PCV)
2- Pressure-support ventilation (PSV)
3- Continuous positive airway pressure (CPAP)
4- Positive end expiratory pressure (PEEP)
5- Noninvasive bilevel positive airway pressure ventilation (BiPAP)

1- Control Mode (CM), Continuous Mandatory Ventilation (CMV)

- Ventilation is completely provided by the mechanical ventilator with a preset tidal volume, respiratory rate and oxygen concentration
- Ventilator totally controls the patient's ventilation i.e. the ventilator initiates and controls both the volume delivered and the frequency of breath.
- Client does not breathe spontaneously.
- Client cannot initiate breathe

2- Pressure-Controlled Ventilation Mode (PCV)

- The PCV mode is used
  - If compliance is decreased and the risk of barotrauma is high.
  - It is used when the patient has persistent oxygenation problems despite a high FiO_2 and high levels of PEEP.
- The inspiratory pressure level, respiratory rate, and inspiratory–expiratory (I:E) ratio must be selected.
- In pressure controlled ventilation the breathing gas flows under constant pressure into the lungs during the selected inspiratory time.

- The flow is highest at the beginning of inspiration (i.e. when the volume is lowest in the lungs).
- As the pressure is constant the flow is initially high and then decreases with increasing filling of the lungs.
- Like volume controlled ventilation PCV is time controlled.

Advantages of pressure limitations are:

1- Reduction of peak pressure and therefore the risk of barotruma and tracheal injury.
2- Effective ventilation. Improve gas exchange

When the PCV mode is used, the mean airway and intrathoracic pressures rise, potentially resulting in a decrease in cardiac output and oxygen delivery. Therefore, the patient's hemodynamic status must be monitored closely.

Used to limit plateau pressures that can cause barotrauma & Severe ARDS

3- Pressure Support Ventilation (PSV)

- The patient breathes spontaneously while the ventilator applies a pre-determined amount of positive pressure to the airways upon inspiration.
- Pressure support ventilation augments patient's spontaneous breaths with positive pressure boost during inspiration i.e. assisting each spontaneous inspiration.
- Helps to overcome airway resistance and reducing the work of breathing.
- Indicated for patients with small spontaneous tidal volume and difficult to wean patients.
- Patient must initiate all pressure support breaths.
- Pressure support ventilation may be combined with other modes such as SIMV or used alone for a spontaneously breathing patient.
- The patient's effort determines the rate, inspiratory flow, and tidal volume.
- In PSV mode, the inspired tidal volume and respiratory rate must be monitored closely to detect changes in lung compliance.
- It is a mode used primarily for weaning from mechanical ventilation.

4- Continuous Positive Airway Pressure (CPAP)

- Constant positive airway pressure during spontaneous breathing

- CPAP allows the nurse to observe the ability of the patient to breathe spontaneously while still on the ventilator.
- CPAP can be used for intubated and nonintubated patients.
- It may be used as a weaning mode and for nocturnal ventilation (nasal or mask CPAP)

5- Positive end expiratory pressure (PEEP)

- Positive pressure applied at the end of expiration during mandatory \ ventilator breath
- Positive end-expiratory pressure with positive-pressure (machine) breaths.
- Uses of CPAP & PEEP
- Prevent atelactasis or collapse of alveoli
- Treat atelactasis or collapse of alveoli
- Improve gas exchange & oxygenation
- Treat hypoxemia refractory to oxygen therapy.(prevent oxygen toxicity
- Treat pulmonary edema (pressure help expulsion of fluids from alveoli

6- Noninvasive Bilateral Positive Airway Pressure Ventilation (BiPAP)

- BiPAP is a noninvasive form of mechanical ventilation provided by means of a nasal mask or nasal prongs, or a full-face mask.
- The system allows the clinician to select two levels of positive-pressure support:
    - An inspiratory pressure support level (referred to as IPAP)
    - An expiratory pressure called EPAP (PEEP/CPAP level).

Common Ventilator Settings parameters/ controls

- Fraction of inspired oxygen (FIO_2)
- Tidal Volume (VT)
- Peak Flow/ Flow Rate
- Respiratory Rate/ Breath Rate / Frequency (F)
- Minute Volume (VE)
- I:E Ratio (Inspiration to Expiration Ratio)
- Sigh

● Fraction of inspired oxygen (FIO_2)

- The percent of oxygen concentration that the patient is receiving from the ventilator. (Between 21% & 100%) (room air has 21% oxygen content).
- Initially a patient is placed on a high level of FIO_2 (60% or higher).
- Subsequent changes in FIO_2 are based on ABGs and the SaO_2.
- In adult patients the initial FiO_2 may be set at 100% until arterial blood gases can document adequate oxygenation.
- An FiO_2 of 100% for an extended period of time can be dangerous (oxygen toxicity) but it can protect against hypoxemia
- For infants, and especially in premature infants, high levels of FiO_2 (>60%) should be avoided.
- Usually the FIO_2 is adjusted to maintain a SaO_2 of greater than 90% (roughly equivalent to a PaO_2 >60 mm Hg).
- Oxygen toxicity is a concern when an FIO_2 of greater than 60% is required for more than 25 hours

Signs and symptoms of oxygen toxicity :-

1- Flushed face
2- Dry cough
3- Dyspnea
4- Chest pain
5- Tightness of chest
6- Sore throat

● Tidal Volume (VT)

- The volume of air delivered to a patient during a ventilator breath.
- The amount of air inspired and expired with each breath.
- Usual volume selected is between 5 to 15 ml/ kg body weight)
- In the volume ventilator, Tidal volumes of 10 to 15 mL/kg of body weight were traditionally used.
- the large tidal volumes may lead to (volutrauma) aggravate the damage inflicted on the lungs
- For this reason, lower tidal volume targets (6 to 8 mL/kg) are now recommended.

● Peak Flow/ Flow Rate

- The speed of delivering air per unit of time, and is expressed in liters per minute.

- The higher the flow rate, the faster peak airway pressure is reached and the shorter the inspiration;
- The lower the flow rate, the longer the inspiration.

● Respiratory Rate/ Breath Rate / Frequency (F)

- The number of breaths the ventilator will deliver/minute (10-16 b/m).
- Total respiratory rate equals patient rate plus ventilator rate.
- The nurse double-checks the functioning of the ventilator by observing the patient's respiratory rate.

For adult patients and older children:-

With COPD

- A reduced tidal volume
- A reduced respiratory rate

For infants and younger children:-

- **A small tidal volume**
- **Higher respiratory rate**

- **In special cases, hypoventilation or hyperventilation is desired**

In a patient with head injury,

- Respiratory alkalosis may be required to promote cerebral vasoconstriction, with a resultant decrease in ICP.
- In this case, the tidal volume and respiratory rate are increased (hyperventilation) to achieve the desired alkalotic pH by manipulating the $PaCO_2$.

In a patient with COPD

Baseline ABGs reflect an elevated $PaCO_2$ should not hyperventilated. Instead, the goal should be restoration of the baseline $PaCO_2$.

- These patients usually have a large carbonic acid load, and lowering their carbon dioxide levels rapidly may result in seizures.

● I:E Ratio (Inspiration to Expiration Ratio):-

- The ratio of inspiratory time to expiratory time during a breath (Usually = 1:2)

● Sigh

- A deep breath.

- A breath that has a greater volume than the tidal volume.
- It provides hyperinflation and prevents atelectasis.
- Sigh volume :----------Usual volume is 1.5 –2 times tidal volume.
- Sigh rate/ frequency :---------Usual rate is 4 to 8 times an hour.

● Peak Airway Pressure:-

- In adults if the peak airway pressure is persistently above 45 cmH_2O, the risk of barotrauma is increased and efforts should be made to try to reduce the peak airway pressure.
- In infants and children it is unclear what level of peak pressure may cause damage. In general, keeping peak pressures below 30 is desirable.

● Sensitivity(trigger Sensitivity)

- The sensitivity function controls the amount of patient effort needed to initiate an inspiration
- Increasing the sensitivity (requiring less negative force) decreases the amount of work the patient must do to initiate a ventilator breath.
- Decreasing the sensitivity increases the amount of negative pressure that the patient needs to initiate inspiration and increases the work of breathing.
- The most common setting for pressure sensitivity are -1 to -2 cm H2O
- The more negative the number the harder it to breath.

Ventilator alarms:-

Mechanical ventilators comprise audible and visual alarm systems, which act as immediate warning signals to altered ventilation.

- Alarm systems can be categorized according to volume and pressure (high and low).
- High-pressure alarms warn of rising pressures.
- Low-pressure alarms warn of disconnection of the patient from the ventilator or circuit leaks.

Pneumothorax and Hemothorax

Pneumothorax occurs when the parietal or visceral pleura is breached and the pleural space is exposed to positive atmospheric pressure. Normally the pressure in the pleural space is negative or subatmospheric; this negative pressure is required to maintain lung inflation.

When either pleura is breached, air enters the pleural space, and the lung or a portion of it collapses.

Hemothorax is the collection of blood in the chest cavity because of torn intercostal vessels or laceration of the lungs injured through trauma. Often both blood and air are found in the chest cavit (hemopneumothorax).

Types of Pneumothorax

Simple Pneumothorax

A simple, or spontaneous, pneumothorax occurs when air enters the pleural space through a breach of either the parietal

Clinical Manifestations

Signs and symptoms associated with pneumothorax depend on its size and cause:

- Pleuritic pain of sudden onset.
- Minimal respiratory distress with small pneumothorax; acute respiratory distress if large.
- Anxiety, dyspnea, air hunger, use of accessory muscles, and central cyanosis (with severe hypoxemia).
- In a simple pneumothorax, the trachea is midline, expansion of the chest is decreased, breath sounds may be diminished, and percussion of the chest may reveal normal sounds or hyperresonance depending on the size of the pneumothorax.
- In a tension pneumothorax, the trachea is shifted away from the affected side, chest expansion may be decreased or fixed in a hyperexpansion state, breath sounds are diminished or absent, and percussion to the affected side is hyperresonant. The clinical picture is one of air hunger, agitation, increasing hypoxemia, central cyanosis, hypotension, tachycardia and profuse diaphoresis.

Medical Management

The goal is evacuation of air or blood from the pleural space.

- A small chest tube is inserted near the second intercostals space for a pneumothorax.
- A large-diameter chest tube is inserted, usually in the fourth or fifth intercostal space, for hemothorax.
- Autotransfusion is begun if excessive bleeding from chest tube occurs.
- Traumatic open pneumothorax is plugged (petroleum gauze); patient is asked to inhale and strain against a closed glottis to eject air from the thorax until the chest tube is inserted, with water-seal drainage.
- Antibiotics are usually prescribed to combat infection from contamination.
- The chest wall is opened surgically (thoracotomy) if more than 1,500 mL of blood is aspirated initially by thoracentesis (or is the initial chest tube output) or if chest tube output
- An emergency thoracotomy may also be performed in the emergency department if a cardiovascular injury secondary to chest or penetrating trauma is suspected.

- The patient with a possible tension pneumothorax should immediately be given a high concentration of supplemental oxygen to treat the hypoxemia, and pulse oximetry should be used to monitor oxygen saturation.
- In an emergency situation, a tension pneumothorax can be decompressed or quickly converted to a simple pneumothorax by inserting a large-bore needle (14-gauge) at the second intercostal space, midclavicular line on the affected side. A chest tube is then inserted and connected to suction to remove the remaining air and fluid, reestablish the negative pressure, and reexpand the lung.

Nursing Management

- Promote early detection through assessment and identification of high-risk population; report symptoms.
- Assist in chest tube insertion; maintain chest drainage or water-seal.
- Monitor respiratory status and reexpansion of lung, with interventions (pulmonary support) performed in collaboration with other health care professionals (eg, physician, respiratory therapist, physical therapist).
- Provide information and emotional support to patient and family.

ABG

To make things simple, I will only refer to the three basic ABG values in this post

1. Acid Base Balance .....(pH) pH = 7.35 to 7.45
2. Carbon Dioxide..... (CO2) CO2 = 35 to 45
3. Bicarbonate..... (HcO3) HcO3 = 22 to 26

You also must note the following:

1. CO2 greater than 45 is acidotic
2. HcO3 less than 22 is acidotic
3. Co2 less than 35 is alkalotic
4. HcO3 greater than 26 is alkalotic
5. Alkalotic: If the pH is greater than 7.45 the patient is Alkalotic.
6. Acidotic: If the pH is below 7.35 the patient is acidotic.

To interpret these results, all you have to do is memorize these four basic questions, and then answer them in order.

A. Is the ABG normal?

1. If all the values fall within the normal parameters, then you have a normal ABG and you can stop here: The ABG is normal.
2. If any one of the values is out of the normal range, then you must move on to the next question.

B.

C. Is the cause respiratory or metabolic? To determine this you look at pH and compare it with HcO3 and CO2. If the pH is acidotic, you look for whichever value (HcO3 or CO2) is also acidotic. If the pH is alkalotic, you look for whichever value (HcO3 or CO2) is also alkalotic.

In this sense, you match the pH with HcO3 and CO2. If the pH matches with the CO2, you have respiratory. If the pH matches with the HcO3, you have metabolic.

Or, put more simply:

1. Metabolic Alkalosis: If the pH is alkatotic and the HcO3 alkalotic.
2. Respiratory Alkalosis: If the pH is alkalotic and the CO2 is alkalotic
3. Metabolic Acidosis: If the pH is acidotic and the HcO3 acidotic.
4. Respiratory Acidisis: If the pH is acidotic and the CO2 is acidotic.

A special case is when the pH, CO2 and HCO3 are all alkalosis or all acidotic. In this case you have a case of combined alkalosis or combined acidosis.

1. Combined Alkalosis: If the pH is alkalotic, CO2 is alkalotic, and HCO3 is alkalotic
2. Combined Acidosis: If the pH is acidotic, CO2 is acidotic, and HCO3 is acidotic

D. Is the cause compensated or uncompensated?

1. Compensated: pH is anywhere inside the normal ranges (Anything between 7.35 to 7.45)
2. Uncompensated: pH is anywhere outside the normal ranges (greater than 7.45 or less than 7.35). Also, the value (CO2 or HCO3) that does not match the pH will still be in the normal range.
3. Partially compensated: pH is anywhere outside the normal range, and the value that does not match the pH (CO2 or HCO3) will be outside its normal range. This indicates the body is attempting to get the pH back to normal. Example: A patient is in respiratory failure and his CO2 is 50 (acidotic) and pH is 7.24 (acidotic). An HCO3 of 27 (alkalotic) means the body is attempting to get the pH back to normal, and this is considered compensation.

A cerebrovascular accident (CVA), an ischemic stroke or "brain attack," is a sudden loss of brain function resulting froma disruption of the blood supply to a part of the brain. Stroke is the primary cerebrovascular disorder in the Unted States. Strokes are usually hemorrhagic (15%) or ischemic/nonhemorrhagic (85%). Ischiemic strokes are categorized according to their cause: CVLarge artery thrombotic strokes (20%), small penetrating artery thrombotic strokes (25%), cardiogenic embolic strokes (20%), cryptogenic strokes (30%), and other (5%). Cryptogenic strokes have no known cause, and other strokes result from causes such as illicit drug use, coagulopathies, migraine, and spontaneous dissection of the carotid or vertebral arteries. The result is an interruption in the blood supply to the brain, causing temporary or permanent loss of movement, thought, memory, speech, or sensation.

Risk Factors

Nonmodifiable

- Advanced age (older than 55 years)
- Gender (Male)
- Race (African American)

Modifiable

- Hypertension
- Atrial fibrillation
- Hyperlipidemia
- Obesity
- Smoking
- Diabetes
- Asymptomatic carotid stenosis and valvular heart disease (eg, endocarditis, prosthetic heart valves)
- Periodontal disease

Clinical Manifestations

General signs and symptoms include numbness or weakness of face, arm, or leg (especially on one side of body); confusion or change in mental status; trouble speaking or understanding speech; visual disturbances; loss of balance, dizziness, difficulty walking; or sudden severe headache.

Motor Loss

- Hemiplegia, hemiparesis
- Flaccid paralysis and loss of or decrease in the deep tendon reflexes (initial clinical feature) followed by (after 48 hours) muscle tone (spasticity)

Communication Loss

- Dysarthria (difficulty speaking)
- Dysphasia (impaired speech) or aphasia (loss of speech)
- Apraxia (inability to perform a previously learned action)

Perceptual Disturbances and Sensory Loss

- Visual-perceptual dysfunctions (homonymous hemianopia [loss of half of the visual field])
- Disturbances in visual-spatial relations (perceiving the relation of two or more objects in spatial areas), frequently seen in patients with right hemispheric damage
- Sensory losses: slight impairment of touch or more severe with loss of proprioception; difficulty in interrupting visual, tactile, and auditory stimuli.

Impaired Cognitive and Psychological Effects

- Frontal lobe damage: Learning capacity, memory, or other higher cortical intellectual functions may be impaired. Such dysfunction may be reflected in a limited attention span, difficulties in comprehension, forgetfulness, and lack of motivation.

- Depression, other psychological problems: emotional lability, hostility, frustration, resentment, and lack of cooperation.

Medical Management

- Recombinant tissue plasminogen activator (t-PA), unless contraindicated; monitor for bleeding
- Anticoagulation therapy
- Management of increased intracranial pressure (ICP): osmotic diuretics, maintain PaCO2 at 30 to 35 mm Hg, position to avoid hypoxia (elevate the head of bed to promote venous drainage and to lower increased ICP)
- Possible hemicraniectomy for increased ICP from brain edema in a very large stroke
- Intubation with an endotracheal tube to establish a patent airway, if necessary
- Continuous hemodynamic monitoring (the goals for blood pressure remain controversial for a patient who has not received thrombolytic therapy; antihypertensive treatment may be withheld unless the systolic blood pressure exceeds 220 mm Hg or the diastolic blood pressure exceeds 120 mm Hg)
- Neurologic assessment to determine if the stroke is evolving and if other acute complications are developing

Management of Complications

- Decreased cerebral blood flow: Pulmonary care, maintenance of a patent airway, and administration of supplemental oxygen as needed.
- Monitor for UTIs, cardiac dysrhythmias, and complications of immobility.

During Acute Phase (1 to 3 days)

- Weigh patient (used to determine medication dosages),

- and maintain a neurologic flow sheet to reflect the following nursing assessment parameters: Change in level of consciousness or responsiveness, ability to speak, and orientation
- Presence or absence of voluntary or involuntary movements of the extremities: muscle tone, body posture, and head position
- Stiffness or flaccidity of the neck
- Eye opening, comparative size of pupils and pupillary reactions to light, and ocular position
- Color of face and extremities; temperature and moisture of skin
- Quality and rates of pulse and respiration; ABGs, body temperature, and arterial pressure
- Volume of fluids ingested or administered and volume of urine excreted per 24 hours
- Signs of bleeding
- Blood pressure maintained within normal limits

Postacute Phase Assess the following functions:

- Mental status (memory, attention span, perception, orientation, affect, speech/language).
- Sensation and perception (usually the patient has decreased awareness of pain and temperature).
- Motor control (upper and lower extremity movement); swallowing ability, nutritional and hydration status, skin integrity, activity tolerance, and bowel and bladder function.
- Continue focusing nursing assessment on impairment of function in patient's daily activities.

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Sub.	Course Syllabus	
Year	2020/2021	