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1 Cardiac Science: Cardiac ArrhythmiasRed: very important. Green: Doctors notes. Pink: formulas. Yellow: numbers. Gray: notes and explanation. Physiology Team 436 Cardiovascular Block Lecture 6 Lecture: If work is intended for initial studying. Review: If work is intended for revision. 2 Objectives From the students guide: Identify normal rhythmStudy Smart: focus on mutual topics. From the students guide: Identify normal rhythm Identify the main pathophysiological causes of cardiac arrhythmias (Arrhythmogenesis) } Identify sinus arrhythmias Define different ectopic foci of excitation and the mechanism of re-entry phenomena } Identify conduction block. Identify electrolyte abnormalities (K+, Ca++) Enumerate the common arrhythmias and describe the basic ECG changes 3 The Normal Conduction SystemONLY IN FEMALES SLIDES Interpretation Develop a systematic approach to reading EKGs and use it every time. The system we will practice is: Heart Rate Rhythm (including intervals and blocks) Axis Hypertrophy Ischemia ONLY IN MALES SLIDES SA node: cardiac pacemaker Paces at rate of bpm (average of 70 bpm) AV node: bpm Purkinje: bpm 4 Interpretation: Heart RateONLY IN FEMALES SLIDES Interpretation: Heart Rate Rule of 300: Divide 300 by the number of boxes between each QRS = rate Number of big boxes Rate 1 300 2 150 3 100 4 75 5 60 6 50 HR of per minute is normal HR > 100 = tachycardia HR < 60 = bradycardia BOTH MALES & FEMALES SLIDES Heart rate in picture below:(300 / 6) = 50 bpm Heart rate may vary depending on: Age Activity Time of day 5 6 Normal Sinus Rhythm; NSRRegular Each P followed by QRS with resulting PR interval is constant and within normal range PP interval is constant. ONLY IN FEMALES SLIDES Doctor said we dont need to memorize all of this. 7 Classification of Cardiac Arrhythmias(According to Heart Rate) 2) Tachyarrhythmias 1) Bradyarrhythmias Cardiac Arrhythmias (According to Mechanism of Arrhythmia) 2) Arrhythmias due to alterations or disturbances of impulse formation 1) Arrhythmias due to disturbances of impulse conduction ONLY IN FEMALES SLIDES Blocks at different points in the spread of impulse through the heart ONLY IN MALES SLIDES Classifications of Cardiac Arrhythmias: Rate above or below normal (tachy vs. bradycardia) Regular or irregular rhythm Narrow or broad QRS complex Relation to P wave Supraventricular Vs. ventricular 8 ONLY IN FEMALES SLIDESAnatomical Classification of Cardiac Arrhythmias (origin of the arrhythmia) ONLY IN FEMALES SLIDES Cardiac Arrhythmias Supraventricular arrhythmias Atrioventricular (AV) junctional arrhythmias Atrial Arrhythmias (Narrow QRS) 1-Atrial extrasystoles (APC) 2-Atrial Tachycardia 3-Atrial Flutter 4-Atrial Fibrillation Ventricular arrhythmias (Wide QRS) 1-Ventricualr extrasystole 2-Ventricular Tachycardia 3-Ventricular Fibrillation * * Extrasystole = premature beats= premature complexes= premature contractions * Also considered as Arrhythmia due to triggered activity and reentry. 9 Arrhythmias Due to Alterations or Disturbances of Impulse FormationMechanisms: Enhanced automaticity. Triggered activity. Reentrant circuits (circus movement). Arrhythmias due to alterations or disturbances of impulse formation Arrhythmias due to alterations in SA node pacemaker activity Arrhythmias due to enhanced automaticity of the AV junction Arrhythmias due to triggered activity and reentry ONLY IN FEMALES SLIDES Other mechanism Abnormal rhythmicity of the pacemaker Blocks at different points in the spread of impulse through the heart 10 1. Enhanced automaticity 3. Re-entry (Lewis circus movement)Mechanisms of Arrhythmias Due to Alterations or Disturbances of Impulse Formation EXTRA 1. Enhanced automaticity 2. Triggered activity 3. Re-entry (Lewis circus movement) Enhanced automaticity is defined as an inappropriate increase in the rate of discharge of a tissue having physiological pacemaker properties (i.e., sinus node, atrioventricular node or Purkinje fibers). The term triggered activity is used to define the appearance of automaticity as a result of external stimulation, and may arise in tissues that do not demonstrate physiological automaticity. Re-entry (reentry): occurs when a cardiac impulse re-excites some region of the heart through which the impulse has already passed in that heart beat. Unidirectional or partial conduction block is required to initiate re-entry. 11 Causes of Cardiac ArrhythmiasONLY IN FEMALES SLIDES Causes of Cardiac Arrhythmias Abnormal rhythmicity of the pacemaker Shift of the pacemaker from the sinus node to another place in the heart Blocks at different points during the spread of the impulse through the heart Abnormal pathways of impulse transmission through the heart Spontaneous generation of spurious impulses in almost any part of the heart Rate above or below normal Regular or irregular rhythm Narrow or broad QRS complex Relation to P waves 12 Arrhythmias Due to Alterations in SA Node Pacemaker Activity1) Sinus Bradycardia Heart rate slower than 60 per minute 2) Sinus Tachycardia Heart rate faster than 100 per minute 1- Physiological Causes: --Expiration. 2- Pharmacological and pathological Causes: - Parasympathetic stimulation Causes: 1- Physiological: -During exercise, sympathetic stimulation, increased body temperature -Inspiration 2- Pharmacological and pathological: -Sympathetic stimulation - Digoxin drug. 13 Differential Diagnosis of TachycardiaONLY IN FEMALES SLIDES Tachycardia Narrow Complex Wide Complex Regular Sinus Tachy SVT Atrial flutter ST w/ aberrancy SVT w/ aberrancy VT Irregular A-fib A-flutter w/ variable conduction MAT A-fib w/ aberrancy A-fib w/ WPW 14 Arrhythmias Due to Alterations in SA Node Pacemaker Activity3) Respiratory sinus arrhythmia Sinus arrhythmia is an increase in heart rate during inspiration and a decrease during expiration It is a normal finding in children and young adults and tends to diminish or disappear with age. Causes: 1- Sinus arrhythmia is due to variations in the vagal tone that affects the SA node. ONLY IN MALES SLIDES Sinus Arrhythmia: Result from spillover of signals from the medullary respiratory center into the adjacent vasomotor center during inspiration and expiratory cycles of respiration. The spillover signals cause alternate increase and decrease in the number of impulses transmitted through the sympathetic and vagus nerves to the heart 15 Atrial and Ventricular ExtrasystolesAbnormal Cardiac Rhythms that Result from Impulse Conduction Block ONLY IN FEMALES SLIDES Extrasystoles = premature beats = premature contractions = premature complexes If the focus is in the ventricles, the premature beat is called ventricular premature beat. If the focus is in the atrial wall, the premature beat is called atrial premature beat. Ectopic foci can also cause a premature beat in AV junction. Causes: Ischemia Irritation of cardiac muscle by calcified foci Caffeine Sinoatrial Block The impulse from the S-A node is blocked before it enters the atrial muscle Cessation (no) of P waves A-V Block When impulse from the S-A node is blocked Causes: Ischemia of the A-V node Compression of the A-V node by scar formation Inflammation of the A-V node Strong vagal stimulation 16 Atrial Extrasystoles Short P-R interval depending on how far the ectopic foci from the AV node. Pulse deficit if there is no time for the ventricles to fill with blood. The time between the premature contraction and the succeeding beat is increased (Compensatory pause). 17 Atrial Flutter Atrial flutter occurs when the atria are stimulated to beat at a rate 250 beats per minute. Atrial flutter results in poor atrial pumping since some parts of the atria are relaxing while other parts are contracting. A single large wave travels around and around in the atria The refractory period of the AV node causes 2-3 beats of atria for one single ventricular beat 2:1 or 2:3 rhythm. ONLY IN MALES SLIDES 18 Atrial Fibrillation (AF)Same mechanism as ventricular fibrillation. It can occur only in atria without affecting the ventricles. It occurs more frequently in patients with enlarged heart. The atria do not pump if they are fibrillating. The efficiency of ventricular filling is decreased 20 to 30% No P wave, or high frequency of low voltage P wave A person can live for years with atrial fibrillation Treatment: DC shock 19 Ventricular extrasystolesProlong QRS complex because the impulses are carried out with myocardial fibers with slower conduction rate than Purkinje fibers Increase QRS complexes voltage because QRS wave from one ventricle can not neutralize the one from the other ventricle After PVCs, the T wave has an electrical potential of opposite polarity of that of the QRS because of the slow conduction in the myocardial fibers, the fibers that depolarizes first will repolarize first Causes: drugs, caffeine, smoking, lack of sleep, emotional irritations 20 Ventricular FibrillationThe most serious of all arrhythmias Cause: impulses stimulate one part of the ventricles, then another, then itself. Many part contracts at the same time while other parts relax. (Circus movement) Causes: sudden electrical shock, ischemia Tachycardia Irregular rhythm Broad QRS complex No P wave Treatment : DC shock 21 Abnormal Cardiac Rhythms that Result from Impulse Conduction BlockONLY IN FEMALES SLIDES Sinoatrial Block Blockade of the S-A node impulse before entering atrial muscle. Cessation of P wave (difficulty of seeing p wave) A-V Block When impulse from the S-A node is blocked First degree block Second degree block Third degree block Causes of SA and AV Block: Ischemia of the A-V node Compression of the A-V node by scar formation Inflammation of the A-V node Strong vagal stimulation 22 Other Arrhythmias Due to Disturbances of Impulse Conduction (impulse conduction block)Arrhythmias due to Alterations in impulse conduction Bundle branch block Heart block = Atrioventricular (AV) block 1st degree block (partial block): all impulses pass through the AV node but with greater than normal delay. 2nd degree block: some, but not all impulses pass through the AV node. 3rd degree block (complete block): no impulses pass through AV node atria and ventricles beat independently. 23 Heart Block 24 Heart Block = Atrioventricular (AV) Block1st degree block: all impulses pass through the AV node but with greater than normal delay. Every P wave is followed by R wave. P-R interval is prolonged = 0.2 second 2nd degree AV block: P-R interval > 0.25 second Only few impulses pass to the ventricles atria beat faster than ventricles dropped beat of the ventricles Mobitz I: Progressive increase in P-R interval until one QRS is dropped. Mobitz II: Constant PR prolongation + dropped beat 3rd degree AV block: (complete heart block). Complete dissociation of P wave and QRS waves Ventricle escape from the influence of S-A node Atrial rate is 100 beats/min Ventricular rate is 57 beats/min. (40 in females) Stroke-Adams Syndrome:: AV block comes and goes Mobitz I: Mobitz II 25 ONLY IN FEMALES SLIDESDiseases in ECG: 1. Ischemia: common use of ECG is acute chest pain. Cause: restriction of blood flow to the myocardium due to reversible: angina pectoris Irreversible: myocardial infarction. Ischemia injury infarction 2. Reversible Ischemia: Inverted T wave - ST segment depression. 3. Myocardial infarction: ST segment elevation Deep Q wave. Complete loss of blood supply to the myocardium resulting in necrosis or death of tissue. 4. Hypokalemia: Flat T wave 5. Hyperkalemia: Tall peak T wave. 26 Quiz to Editing File (Please be sure to check this file frequently for any edits or updates on all of our lectures.) References: Girls and boys slides. Guyton and Hall Textbook of Medical Physiology (Thirteenth Edition.) 27 Thank you! The Physiology 436 Team: Team Leaders: Contact us: . The Physiology 436 Team: Female Members: Amal AlShaibi Rana Barasain Male Members: Mohammed Almutllaq Team Leaders: Qaiss Almuhaideb Lulwah Alshiha Contact us: @Physiology436 0 ratings0% found this document useful (0 votes)6 viewsThe document outlines various cardiac emergencies, including myocardial infarction, cardiac arrest, and arrhythmias, emphasizing the importance of prompt treatment to reduce mortality. It deSaveSave cardiac emergencies ppt For Later0%0% found this document useful, undefined0 ratings0% found this document useful (0 votes)6 viewsThe document outlines various cardiac emergencies, including myocardial infarction, cardiac arrest, and arrhythmias, emphasizing the importance of prompt treatment to reduce mortality. It de1. CARDIAC EMERGENCY AND ARREST MANALI H SOLANKI F.Y.M.SC.NURSING J G COLLEGE OF NURSING 2. The amount of myocardial stretch just beforesystole caused by the pressure created by thevolume of blood within a ventricle The amount of resistance to ejection of bloodfrom a ventricle 3. The inability of the heart to pump sufficientblood to meet the needs of the tissues foroxygen and nutrients; signs and symptoms ofpulmonary and systemic congestion may ormay not be present Amount of blood pumped out of theventricle with each contraction 4. INTRODUCTION Cardiovascular disease is the number one killer of adults. Prompt recognition and initiation of appropriate treatment can save lives during three of the most deadly cardiac emergencies: angina pectoris, myocardial infarction and congestive cardiac failure. 5. ANATOMY AND PHYSIOLOGY OF HEART 7. DEFINITION: Angina (angina pectoris - Latin for squeezing of the chest) is chest discomfort that occurs when there is a decreased blood oxygen supply to an area of the heart muscle. 8. The major types of angina are stable, unstable, variant (Prinzmetal's), and microvascular. 9. Stable angina is the most common type of angina. It occurs when the heart is working harder than usual. Stable angina has a regular pattern. Stable angina isn't a heart attack, but it suggests that a heart attack is more likely to happen in the future. 10. Unstable angina doesn't follow a pattern. It may occur more often and be more severe than stable angina. Unstable angina is very dangerous and requires emergency treatment. This type of angina is a sign that a heart attack may happen soon. 11. Variant angina is rare. A spasm in a coronary artery causes this type of angina. Variant angina usually occurs while you're at rest, and the pain can be severe. It usually happens between midnight and early morning. Medicine can relieve this type of angina 12. Microvascular angina can be more severe and last longer than other types of angina. Medicine may not relieve this type of angina. 13. Coronary artery spasm Coronary-artery disease with partial blockage or spasm of arteries that supply blood to the heart. Anaemia. Overactive thyroid gland. Heart-valve disease. 14. Diabetes Family history of coronary heart disease before age 50 High blood pressure High LDL cholesterol and low HDL cholesterol Obesity Smoking Cold weather Emotional stress Large meals 16. Tightness, squeezing, pressure or ache in the chest. Sudden breathing difficulty (sometimes). Chest pain similar to indigestion. A choking feeling in the throat. Chest pain that radiates to the jaw, teeth or earlobes. Heaviness, numbness, tingling or ache in the chest, arm, shoulder, elbow or hand usually on the left side. 17. A feeling of indigestion or heartburn Dizziness or light-headedness Nausea, vomiting, and sweating Palpitations Shortness of breath Unexplained tiredness after activity 22. Nitrates Glyceryl Trinitrate Isosorbide dinitrate Beta blockers e.g. Propranolol, Atenolol, Metoprolol, Pindolol, Oxprenolol 23. Coronary Angioplasty 24. Coronary artery bypass grafting 27. Myocardial infarction (MI) or acute myocardial infarction (AMI), commonly known as a heart attack, is the interruption of blood supply to a part of the heart, causing heart cells to die 28. CAUSES Atherosclerosis High cholesterol levels High blood pressure Diabetes Obesity and Overweight Smoking Heredity 30. SIGN AND SYMPTOMS Chest discomfort, mild pain Coughing Crushing chest pain Dizziness Dyspnea (shortness of breath) Face seems gray 31. A feeling of terror that your life is coming to its end Feeling really awful (general feeling) Nausea Restlessness The person is clammy and sweaty Vomiting 33. Blood tests Chest X-ray 34. COMPLICATIONS Arrhythmia. Cardiogenic shock. Heart failure Pulmonary edema Pericarditis. 35. Other complications for MyocardialInfarction (MI) include: Rupture of the atrial or Ventricular septum, Ventricular wall, or valves; Ventricular aneurysms Cerebral or pulmonary emboli 36. Anti-platelet medicines Nitroglycerin ACE (angiotensin converting enzyme) inhibitors Beta-blocking agents Coronary Angioplasty Coronary Artery Bypass Grafting 39. DEFINITION Heart failure, also called "congestive heart failure," is a disorder where the heart loses its ability to pump blood efficiently. 40. CAUSES: coronary artery disease and heart attack (which may be "silent") cardiomyopathy high blood pressure (hypertension) heart valve disease congenital heart disease alcoholism and drug abuse 42. SIGN AND SYMPTOMS Fatigue Swelling (oedema) of the ankles and legs or abdomen An increase in body weight Shortness of breath Increased urination 43. Chest x-ray Electrocardiogram (ECG) Blood tests Echocardiogram MUGA scan Stress test 44. Angiotensin II receptor blockers (ARBs) Vasodilators/Nitrates Potassium or magnesium Aldosterone inhibitors (spironolactone) Calcium channel blockers (for diastolic dysfunction only) Inotropic therapy Angiotensin-converting enzyme (ACE) inhibitors Beta blockers 47. Sudden cardiac arrest (SCA) is a condition in which the heart suddenly and unexpectedly stops beating. If this happens, blood stops flowing to the brain and other vital organs. 48. Coronary artery disease Heart attack Enlarged heart (cardiomyopathy) Valvular heart disease Congenital heart disease Electrical problems in the heart 49. Risk Factors Who have had a heart attack (myocardial infarction) Who have experienced heart failure (poor pumping heart) Who have survived a previous sudden cardiac arrest With a family history of sudden cardiac arrest With a low ejection fraction 51. SIGN AND SYMPTOMS Breathlessness Fatigue Breathlessness on lying down Waking up gasping for breath at night Swollen abdomen Enlarged liver Visible venous pulse in neck Swollen legs and ankles Decreased exercise tolerance Enlarged heart 52. Echocardiogram Electrocardiogram (ECG) Chest x-ray Exercise test (stress test) Cardiac catheterization Laboratory studies may include CBC, ESR, electrolytes, BUN/creatinine, gluc ose, TSH, calcium, magnesium, and phosphorus Blood cultures 53. Angiotensin II receptor blockers (ARBs) Vasodilators/Nitrates Potassium or magnesium Aldosterone inhibitors (spironolactone) Calcium channel blockers (for diastolic dysfunction only) Inotropic therapy Diuretics Angiotensin-converting enzyme (ACE) inhibitors 54. Coronary artery bypass surgery 57. Nursing Diagnosis Decreased Cardiac Output related to impaired contractility and increased preload and afterload Impaired Gas Exchange related to alveolar edema due to elevated ventricular pressures 58. Excess Fluid Volume related to sodium and water retention Activity Intolerance related to oxygen supply and demand imbalance Cardiac Arrhythmiaarrhythmias Abnormal pacemaker, rhythm and AV coordination of heart beats. all arrhythmias result from (1) disturbances in impulse formation, (2) disturbances in impulse conduction, (3) both.A. Electrophysiological basis of arrhythmias 1. Normal cardiac electrophysiology Excitability: ability to produce action potentials maximal diastolic potentials (MDP) threshold levels Automaticity: pacemaker phase 4 slope Conductivity: ability to conductimpulse conduction pathways, phase 0 amplitudeFast response cell Action potential and ion transportAction potential and ion transport Fast response cellAPD ERP APD Action potential Duration (APD) and effective refractory period (ERP)A schematic representation of Na+ channels cycling through different conformational states during the cardiac action potential. Transitions between resting, activated, and inactivated states are dependent on membrane potential and time. The activation gate is shown as m and the inactivationA. Electrophysiological basis of arrhythmias 2. Slow and fast response cells slow response cells: pacemaker cells fast response cells: conduction and contraction cells Fast response Slow response phase 4 potential90 mV 70 mV depolarizationNa+, 120 mV, 1-2 ms Ca2+, 70 mV, 7 ms automaticitylow0.02 V/s high0.1 V/s conductionfast200-1000 V/s slow10 V/s effectsconductionpacemakerSlow response Fast response Action potentials of slow and fast response cellssinoatrial (SA) node atrioventricular (AV) node Slow response pacemakers in sinoatrial (SA) node and atrioventricular (AV) nodeImpulse generation and conduction in the heartA. Electrophysiological basis of arrhythmias 3. Abnormal generation of impulse (1) Augmented automaticity Augmented automaticity in the myocardial cells other than the sinoatrial node cells will produce arrhythmias Maximal diastolic potential (MDP) in phase 4:ischemia, digitalis, sympathetic excitation, imbalance of electrolytes Fast spontaneous depolarization in phase 4fast response cells slow response cells a.increased phase 4 slope Slow response cells a. decreased MDP b.decreased threshold levelsA.increased phase 4 slope B.decreased threshold levels C. decreased MDP levels in phase 4 fast response cellsA. Electrophysiological basis of arrhythmias (2) Afterdepolarization and triggered activity early afterdepolarization (EAD,) phases 2, 3; Ca2+ inward flow increases induced by drugs, plasma K+ delayedafterdepolarization (DAD ,) phase 4; Ca2+ inward flow leads to transient Na+ inward flow induced by digitalis intoxication, plasma Ca2+, K+A.early afterdepolarization (EAD) B.delayed afterdepolarization (DAD) Triggered beat Triggered beatsingle EAD multiple EAD triggered beat triggered beats multiple DAD triggered beatsA. Electrophysiological basis of arrhythmias 4. Abnormal conduction of impulse (1) Simple conduction block slow and small depolarization in phase 0, reduced MDP level in phase 4 MDP in ischemia, inflammation, metabolic disorders Usually occurred in atrioventricular regionsA. Electrophysiological basis of arrhythmias (2) Reentrant reexcitation (reentry,) Circuits (especially in enlarged ventricles) (Wolff-Parkinson-White syndrome) Unidirectional (one-way) block (myocardial injury) Slow conduction Heterogeneity in ERP ERPReentrant reexcitation (reentry)Reentry formationAbnormal conduction pathway of Wolff-Parkinson-White syndromeB. Electrophysiological effects and classification of antiarrhythmic drugs 1.Electrophysiological effects of antiarrhythmic drugs (1) Reducing abnormal automaticity decreasing phase 4 slope increasing threshold levels increasingMDP levels in phase 4 increasing action potential duration (APD)A.decreasing phase 4 slope B.increasing threshold levels C. increasing MDP levels in phase 4 D.increasing action potential durationAPD D fast response cellsb.decreasing phase 4 slope Slow response cells c.increasing threshold levelsd.increasing MDPB. Electrophysiological effects and classification of antiarrhythmic drugs class IV drugs decrease automaticity of slow response cells class I drugs decrease automaticity of fast response cells class II drugs decrease the augmented automaticity caused by sympathetic excitationB. Electrophysiological effects and classification of antiarrhythmic drugs (2) inhibiting afterdepolarization and triggered activity EADrepolarization (class IB), inward current (class I, IV) DADclass IV, I Sympathetic excitation or digitalisclass IIB. Electrophysiological effects and classification of antiarrhythmic drugs (3) Modulating conduction Accelerating conduction Abolishing reentry one-way block two-way block abolishing one-way blockone-way blockreentry normal two-way block abolishing block B. Electrophysiological effects and classification of antiarrhythmic drugs (4) Modulating effective refractory period (ERP) prolonged ERP prolonged ERP/APD homogeneity of ERPReducing membrane responsiveness increasing APD and ERP2. CARDIAC ARRHYTHMIASCARDIAC ARRHYTHMIAS An abnormality of the cardiacrhythm is called a cardiacarrhythmia. Arrhythmias may causesudden death, syncope, heartfailure, dizziness, palpitations or nosymptoms at all. There are twomain types of arrhythmia:1. bradycardia: the heart rate isslow (< 60 b.p.m.)2. tachycardia: the heart rate isfast (> 100 b.p.m.).CardiacCardiacarrhythmiasarrhythmias 3. Tachycardias are more symptomaticwhen the arrhythmia is fast andsustained. Tachycardias aresubdivided into supraventriculartachycardias, which arise from theatrium or the atrioventricularjunction, and ventriculartachycardias, which arise from theventricles.CardiacCardiacarrhythmiasarrhythmias 4. SINUS RYTHMSINUS RYTHM The normal cardiac pacemaker isthe sinus node its rate of discharge is controlled bythe autonomic nervous system;sympathetic system increases heartrate, while parasympathetic systemdecrease it. Normal sinus rhythm ischaracterized by P waves that areupright in leads I, and II of the ECG,but inverted in the cavity leads AVRand V1.CardiacCardiacarrhythmiasarrhythmias 5. Sinus arrhythmiaSinus arrhythmia Fluctuations of autonomic toneresult in phasic changes of the sinusdischarge rate. During inspiration,parasympathetic tone falls and theheart rate quickens, and onexpiration the heart rate falls. Thisvariation is normal, particularly inchildren and young adults. Typicallysinus arrhythmia results in aregularly irregular pulse.CardiacCardiacarrhythmiasarrhythmias 6. Arrhythmia FormationArrhythmia Formation Arrhythmias can arise fromproblems in the: Sinus node Atrial cells AV junction Ventricular cells Arrhythmias caused by 2 mainmechanism: Automaticity Re-entry.CardiacCardiacarrhythmiasarrhythmias 7. SA Node ProblemsSA Node ProblemsThe SA Node can: fire too slow fire too fastSinus BradycardiaSinus TachycardiaCardiacCardiacarrhythmiasarrhythmias 8. Atrial Cell ProblemsAtrial Cell ProblemsAtrial cells can: fire occasionallyfrom a focus Fire continuouslydue to a loopingre-entrant circuitPremature AtrialContractions(PACs)Atrial FlutterCardiacCardiacarrhythmiasarrhythmias 9. Atrial Cell ProblemsAtrial Cell ProblemsAtrial cells can also: fire continuouslyfrom multiple focior fire continuouslydue to multiplemicro re-entrantwaveletsAtrial FibrillationAtrial FibrillationCardiacCardiacarrhythmiasarrhythmias 10. AV Junctional ProblemsAV Junctional ProblemsThe AV junction can: fire continuously dueto a looping re-entrant circuit block impulsescoming from the SANodeParoxysmalSupraventricularTachycardiaAV JunctionalBlocksCardiacCardiacarrhythmiasarrhythmias 11. Ventricular Cell ProblemsVentricular Cell ProblemsVentricular cells can: fire occasionally from1 or more foci fire continuously frommultiple foci fire continuously dueto a looping re-entrant circuitPrematurePremature VentricularVentricularContractionsContractions(PVCs)(PVCs)VentricularVentricularFibrillationFibrillationVentricularVentricularTachycardiaTachycardiaCardiacCardiacarrhythmiasarrhythmias 12. Sinus BradycardiaSinus Bradycardia A sinus rate of less than 60 b.p.m.during the day or less than 50 .m. atnight is known as sinus bradycardia. CAUSESPhysiological In athletes During sleepCardiacCardiacarrhythmiasarrhythmias 14. Intrinsic causes1. Acute ischaemia and infarction ofthe sinus node (as a complicationof acute myocardial infarction).2. Chronic degenerative changessuch as fibrosis of the atrium andsinus node (sick sinussyndrome).CardiacCardiacarrhythmiasarrhythmias 15. Sick sinus syndromeSick sinus syndromeSick sinus syndrome or sinoatrialdisease is usually caused byidiopathic fibrosis of the sinus node.Other causes of fibrosis such asischemic heart disease,cardiomyopathy or myocarditis canalso cause the syndrome. Patientspresent episodes of sinusbradycardia, sinus arrest,paroxysmal superaventricular tachycardia or tachy-bradysyndrome.CardiacCardiacarrhythmiasarrhythmias 16. CLINICAL FEATURESCLINICAL FEATURES Pt may be asymptomatic Severe bradycardia may causeweakness, confusion & syncope Atrial & ventricular ectopics aremore apparent to occur with slowsinus rate.CardiacCardiacarrhythmiasarrhythmias 17. TREATMENTTREATMENT Identification & treatment of thecause Acute symptomatic bradycardiaresponds to atropine o.6 mg i.v. Temporary pacemaker insymptomatic bradycardia if there isreversible cause Permenant pacemaker insymptomatic irreversible causeCardiacCardiacarrhythmiasarrhythmias 18. Sinus tachycardiaSinus tachycardia Resting sinus rate of more than 100b.p.m. is known as sinus tachycardia.CausesCausesNon cardiacAcute1. Fever2. Hypovolemia3. Pain4. Infection5. Exercise6. Emotion/anxietyCardiacCardiacarrhythmiasarrhythmias 19. CardiacCardiacHF with compensatoy tachycardiaCLINICAL FEATURESCLINICAL FEATURES Onset & termination are gradual not exceed 160 b.p.m. Pt complain of palpitationTREATMENTTREATMENT Treatment of the cause Symptomatic sinus tachycardia can bereduced with beta-blocker and/orVerapamilCardiacCardiacarrhythmiasarrhythmias 20. HeART bloCkHeART bloCkHeart block or conduction blockmay occur at any level in theconducting system. Block in eitherthe AV node or the His bundleresults in Atrioventricular (AV) block,whereas block lower in theconduction system produces bundlebranch block.CardiacCardiacarrhythmiasarrhythmias 21. Atrioventricular blockAtrioventricular blockThere are three forms:First-degree AV block This is simple prolongation of thePR interval to more than 0.22 s.Every atrial depolarization isfollowed by conduction to theventricles but with delay. Delay of conduction may be withinatrium, AV node, bundle of his orbundle branches.CardiacCardiacarrhythmiasarrhythmias 22. Second-degree AV blockSecond-degree AV blockThis occurs when some P wavesconduct and others do not. There areseveral formsThere are three types:-I.I. Mobitz I block (Wenckebach blockMobitz I block (Wenckebach blockphenomenon)phenomenon) is progressive PRinterval prolongation until a P wavefails to conduct. The PR interval beforethe blocked P wave is much longerthan the PR interval after the blockedP wave.CardiacCardiacarrhythmiasarrhythmias 23. 2.2. Mobitz II blockMobitz II block occurs when adropped QRS complex is notpreceded by progressive PRinterval prolongation.CardiacCardiacarrhythmiasarrhythmias 24. 33 2 : 1 or 3 : 1 (advanced) block2 : 1 or 3 : 1 (advanced) block It may present as either type I ortype II AV block in which there are 2p waves or 3 to each QRScomplex. If PR interval is prolongedand QRS is narrow then it is type I.if PR normal and QRS is wide thenit is type II 2nddegree AV block.CardiacCardiacarrhythmiasarrhythmias 25. Causes of AV blockCauses of AV block Ischemia of the AV node or AVbundle fibers Compression of the AV bundle byscar tissue of calcified portions ofthe heart Inflammation of the AV node Extreme stimulation ofparasympathetic system(for example carotid sinus syndrome)CardiacCardiacarrhythmiasarrhythmias 26. Third-degree (complete) AV blockThird-degree (complete) AV block Complete heart block occurs whenall atrial activity fails to conduct tothe ventricles. In patients withcomplete heart block the etiologyneeds to be established. Cardiacaction is maintained by spontaneousescape rhythm.CardiacCardiacarrhythmiasarrhythmias 27. Etiology of complete heart block1. Congenital2. Idiopathic fibrosis3. Ischaemic heart disease4. Infiltrations (e.g. amyloidosis,sarcoidosis, neoplasia)5. Cardiac surgery6. Drug-inducede.g. digoxin,amiodarone7. Infections: Endocarditis, Lymedisease & Chagas' disease8. Connective tissue diseasese.g.SLE, rheumatoid arthritisCardiacCardiacarrhythmiasarrhythmias 28. SYMTOMS OF HEART BLOCKsymptom develop due tobradycardia & loss of AV nodesynchrony Exercise intolerance Easy fatigability Dyspnea on exertion Dizzy spills Near syncope & frank syncopeCardiacCardiacarrhythmiasarrhythmias 29. TreatmentHeart block complicate acute MIHeart block complicate acute MI Acute inferior wall MI is oftencomplicated by transient AV blockwhich may respond to atropine 0.6 mgi/v repeated as necessary, if it failtemporary pacemaker should beinserted. In majority AV block willresolve within 7-10 days. Pt maydevelop heart failure or hypotensiondue to slow heart rate in alreadydamaged heart requiring pacemaker.CardiacCardiacarrhythmiasarrhythmias 30. Mobitz II or complete heart blockcomplicating anterior wall MI is usuallya sign of extensive myocardial damageand carries a poor prognosis, asystoleoften occurs and temporary pacemakershould be inserted prophylactically assoon ass possible. If the pt presents with asystole,atropine 0.6 mg i/v repeated asnecessary and isopronaline1-5mg in500ml dextrose 5% infusion I/V atminimum rate until temporarypacemaker can be insertedCardiacCardiacarrhythmiasarrhythmias 31. Chronic heart block Asymptomatic 1stdegree or Mobitztype I 2nddegree AV block requiresno treatment. Permanent pacemaker in pt withsymptomatic bradycardia withcomplicating AV block. Permanent pacemaker in pt withasymptomatic Mobitz II 2ndorcomplete heart block.CardiacCardiacarrhythmiasarrhythmias 32. Bundle branch blockBundle branch block The His bundle gives rise to the rightand left bundle branches. The leftbundle subdivides into the anteriorand posterior divisions of the leftbundle. Various conductiondisturbances can occur.CardiacCardiacarrhythmiasarrhythmias 33. Normal Impulse Conduction Sinoatrial node AV node Bundle of His Bundle Branches Purkinje fibersCardiacCardiacarrhythmiasarrhythmias 34. So, depolarizationof the BundleBranches andPurkinje fibers areseen as the QRScomplex on the ECG.Therefore, aconduction block ofthe Bundle Brancheswould be reflected asa change in the QRScomplex.RightBBBCardiacCardiacarrhythmiasarrhythmias 35. With Bundle Branch Blocks you will seetwo changes on the ECG.1. QRS complex widens (> 0.12 sec).2. QRS morphology changes (varies dependingon ECG lead, and if it is a right vs. left bundlebranch block).CardiacCardiacarrhythmiasarrhythmias 36. Why does the QRS complex widen?When theconduction pathwayis blocked it willtake longer for theelectrical signal topass throughout theventricles.CardiacCardiacarrhythmiasarrhythmias 37. Right Bundle Branch BlocksRight Bundle Branch Blocks What QRS morphology is characteristic?For RBBB the wide QRS complexassumes a unique, virtually diagnosticshape in those leads overlying the rightventricle (V1 and V2).V1Rabit ear (M)CardiacCardiacarrhythmiasarrhythmias 38. Causes of right bundle branch blockCauses of right bundle branch blockIt is a normal finding in 1% of youngadults and 5% of elderly adults Congenital heart disease Pulmonary disease Acute myocardial infarction Cardiomyopathy Conduction system fibrosisCardiacCardiacarrhythmiasarrhythmias 39. Left Bundle Branch BlocksLeft Bundle Branch BlocksWhat QRS morphology is characteristic?For LBBB the wide QRS complex assumes acharacteristic change in shape in those leadsopposite the left ventricle (right ventricular leads -V1 and V2).Broad,deep SwavesNormal 40. . Causes of left bundle branch blockCauses of left bundle branch block Left ventricular outflowobstruction Aortic stenosis Hypertension Coronary artery disease Acute myocardial infarction Severe coronary disease (two- tothree-vessel disease)CardiacCardiacarrhythmiasarrhythmias 41. In each of them there are wide QRS Broad notched R wave in leads V1 &V2 in RBBB but in leads V5, V6, I &aVL in LBBB. Wide deep S waves in leads V5 &V6 in RBBB. But in leads V1 & V2 inLBBB. LBBB it indicate more severedisease diagnosis of LVH or MI is difficult inthe presence of LBBB.CardiacCardiacarrhythmiasarrhythmias 42. Right bundle branch block alonedoes not alter the electrical axis ofthe heart. Axis deviations signifyright ventricular hypertrophy (RVoverload) or coexistent fascicularblock. The combination of rightbundle branch block with left axisdeviation is associated with ostiumprimum atrial septal defects.CardiacCardiacarrhythmiasarrhythmias 43. PATHOLOGICAL TACHYCARDIAPATHOLOGICAL TACHYCARDIAECTOPIC RHYTHMECTOPIC RHYTHMImpulse arises somewhere other thanSA node.May arise from:- Supraventricular (including, AVnode or other AV junctionaltissue). Ventricular in the ventricleCardiacCardiacarrhythmiasarrhythmias 46. supraventricular ectopic rhythmsupraventricular ectopic rhythmEctopic beatsEctopic beats Mostly asymptomatic Can give sensation of an extra orextra beat ECG shows a premature beat with anormal QRS complex.CardiacCardiacarrhythmiasarrhythmias 47. supraventricular tachycardia (SVT)supraventricular tachycardia (SVT) This is a tachycardia occurring inepisodes with rate of 140-240, as aresult of re-entry or rapidly firingectopic focus in the atria or AV node. Itmay last from few seconds to manyhours (if untreated) Predisposing factors:Predisposing factors: Anxiety Excess tobacco or coffee Hyperthyroidism Exertion AlcoholCardiacCardiacarrhythmiasarrhythmias 48. Clinical featuresClinical features Pt feels that has suddenly start tobeat fast (palpitation). Fainting, breathlessness, dizziness,neck pulsation, central chest pain,and weakness polyuria is some time a feature.ECGECG shows tachycardia with normalQRS complexCardiacCardiacarrhythmiasarrhythmias 49. ManagementManagementAcute managementIn the absence of heart disease,serious side effects are rare, andmost attacks break spontaneously.Terminate attack if cardiac failure,syncope or anginal pain develops orif there underlying cardiac orcoronary disease.CardiacCardiacarrhythmiasarrhythmias 50. vagotonic maneuversvagotonic maneuvers Carotid sinus message Valsalva maneuver facial immersion in cold water Pressure on eyes Self inducing vomiting Breath holding Lowering the head between thekneesCardiacCardiacarrhythmiasarrhythmias 51. drugs If physical manoeuvres have notbeen successful, intravenous adenosine 6mg bolus, ifno response within 1-2 minutes 2nd&3rd12mg should be given. SEbronchospasm, flushing & chest pain. verapamil 5-10 mg i.v. over 5-10minutes Maintenance 2.510 mg/h. Beta blokers ismolol 500microg/mkgwithin one minute foolwed by 25-200microg/min.CardiacCardiacarrhythmiasarrhythmias 52. Digoxin is also effective but it takelonger time to act Beta blocker and digoxin are lesscommonly used Amiodarone is not required fortermination of attack, it is used forprevention of occurrence. if pt hymodynamically unstable or ifadenosine or veramapil arecontraindicated or in effective.Synchronized DC cardioversionshould be performed.CardiacCardiacarrhythmiasarrhythmias 53. Long-term managementLong-term managementVerapamil, diltiazem, and beta-blockers have proven effective in 60-80% of patients.If drug therapy is not effective thenperform

Emergency arrhythmias. Cardiac emergency protocols. Cardiac arrhythmia emergency management.