

OVERALL PLAN AND FUNCTIONS OF THE CARDIOVASCULAR SYSTEM (CVS). FUNCTIONAL ANATOMY OF THE HEART: RECAPITULATION OF CARDIAC MUSCLE PROPERTIES; MED & SURG 200, NURSING 200, MED LAB 200, OPT 300.

INTRODUCTION

- CARDIOVASCULAR SYSTEM IS THE SYSTEM OF HEART AND BLOOD VESSELS THAT CIRCULATE BLOOD THROUGHOUT THE BODY. IT CONSTITUTES ONE OF THE COORDINATING AND INTEGRATING SYSTEMS OF THE BODY.
- IT CONSISTS OF THE HEART, BLOOD AND BLOOD VESSELS (ARTERIES, VEINS, AND CAPILLARIES). THE ARTERIES CARRY BLOOD (OXYGENATED) FROM THE HEART TO THE REST OF THE BODY, AND THE VEINS CARRY DEOXYGENATED BLOOD BACK TO THE HEART WITH THE EXCEPTION OF THE PULMONARY VESSELS.
- THE HEART WORKS CONTINUOUSLY WITHOUT A BREAK THROUGHOUT THE LIFE SPAN OF AN INDIVIDUAL. CESSATION OF 3-5MINS IS CALLED CARDIAC ARREST AND LEADS TO SUDDEN DEATH WITHOUT RESUSCITATION. THE HEART BEATS AN AVERAGE OF 70BEATS/MINUTE.

THE HEART WORKS AS A PUMP THAT PUSHES BLOOD TO THE ORGANS, TISSUES, AND CELLS OF THE BODY. BLOOD PERFORMS THE FOLLOWING FUNCTIONS:

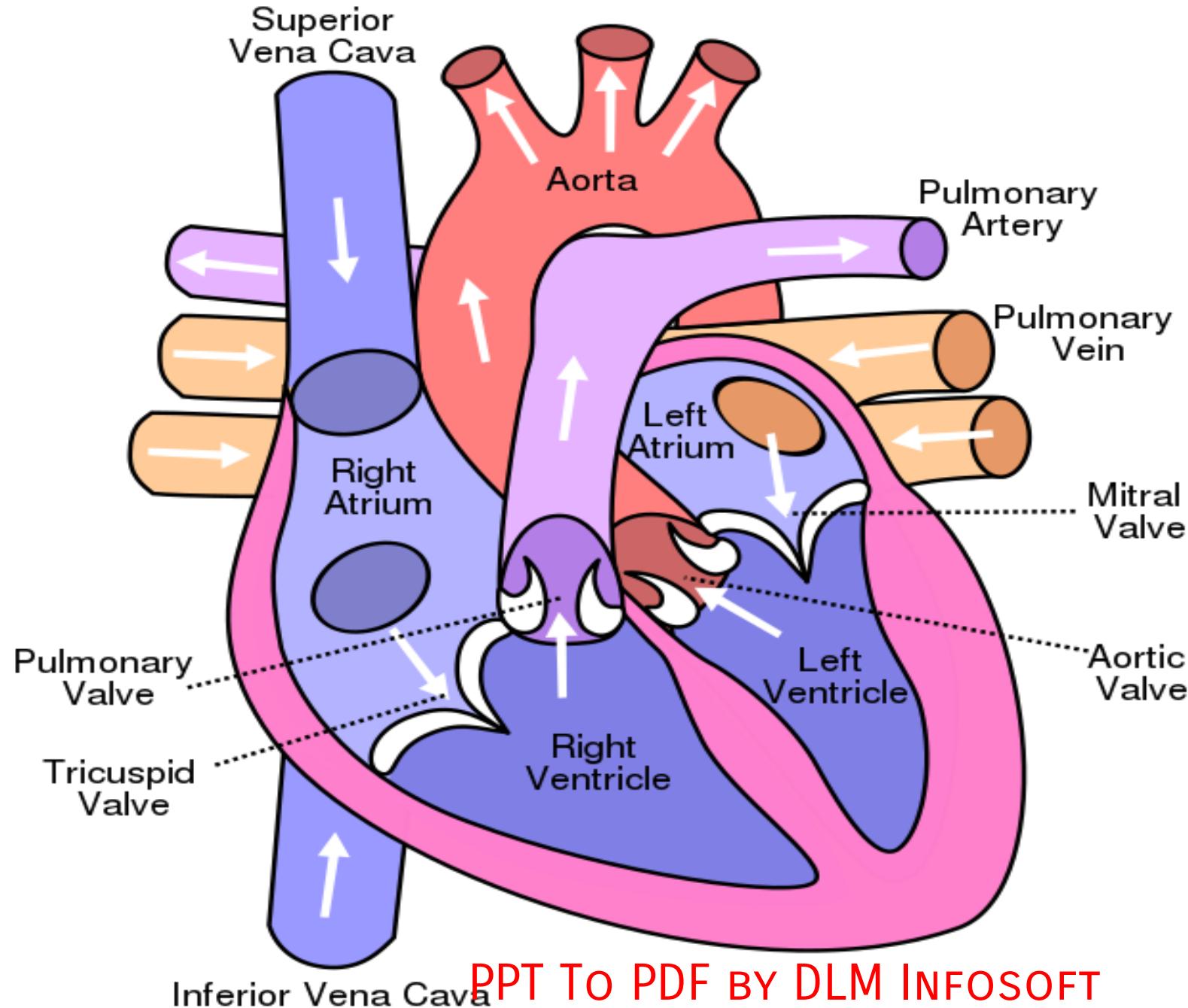
- v DELIVERY OF OXYGEN AND NUTRIENTS TO THE TISSUES.
- v TRANSPORT OF WASTE PRODUCTS OF METABOLISM TO THE EXCRETORY ORGANS.
- v CIRCULATION OF ELECTROLYTES AND HORMONES.
- v BODY TEMPERATURE REGULATION- THERMOREGULATION.
- v TRANSPORT OF IMMUNE SUBSTANCES FOR BODY DEFENSE MECHANISMS.

THE HEART

- THE HEART IS LOCATED BETWEEN THE LUNGS IN THE MIDDLE OF THE CHEST, BEHIND AND SLIGHTLY TO THE LEFT OF THE STERNUM (BREAST BONE).
- THE HEART WEIGHS BETWEEN 200 AND 425G, A LITTLE LARGER THAN THE FIST.
- THE HEART BEATS ABOUT 72 TIMES IN A MINUTE PUMPING ABOUT 5LITERS OF BLOOD AND 100,000 TIMES A DAY, PUMPING ABOUT 7,571 LITERS (2 GALLONS) OF BLOOD.
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FUNCTIONAL ANATOMY OF THE HEART

- THE HEART IS A MUSCULAR ORGAN THAT PUMPS BLOOD THROUGHOUT THE CIRCULATORY SYSTEM. IT IS SITUATED BETWEEN THE 2 LUNGS IN THE MEDIASTINUM.
- THE HEART ITSELF IS MADE UP OF 4 CHAMBERS, 2 ATRIA AND 2 VENTRICLES. THE MUSCULATURE IS THICKER IN THE VENTRICLES, THAN THE ATRIA. THE WALL OF THE L VENTRICLE IS 3 TIMES THICKER THAN THE R VENTRICLE.
- THE HEART IS MADE UP OF 3 LAYERS: OUTER PERICARDIUM, MIDDLE MYOCARDIUM AND INNER ENDOCARDIUM.
- THE PERICARDIUM IS MADE OF 2 LAYERS; OUTER PARIELTAL PERICARDIUM AND INNER VISCELAR PERICARDIUM.
- THE 2 LAYERS ARE SEPARATED BY A SPACE CALLED THE PERICARDIAL



SEPTA OF THE HEART

- ATRIA ARE SEPARATED FROM ONE ANOTHER BY A FIBROUS SEPTUM CALLED INTERATRIAL SEPTUM.
- THE VENTRICLES ARE SEPARATED BY THE INTERVENTRICULAR SEPTUM WHICH IS MUSCULAR.

VALVES OF THE HEART

THERE ARE 4 HEART VALVES

- THE TWO ATRIOVENTRICULAR (AV) VALVES, THE MITRAL VALVE (BICUSPID VALVE), AND THE TRICUSPID VALVE, WHICH ARE BETWEEN THE UPPER CHAMBERS (ATRIA) AND THE LOWER CHAMBERS (VENTRICLES). THEY CONTROL BLOOD FLOW FROM THE ATRIA TO THE VENTRICLES.
- THE TWO SEMILUNAR (SL) VALVES NAMELY THE AORTIC VALVE AND THE PULMONARY VALVE, WHICH ARE IN THE ARTERIES LEAVING THE HEART. THESE CONTROL BLOOD FLOW OUT OF THE VENTRICLES.

VESSELS OF THE CARDIOVASCULAR SYSTEM

- AORTA
- ARTERIES
- ARTERIOLES
- CAPILLARIES
- VENULES
- VEINS
- VENAE CAVAE

S/N	CHARACTERISTICS	ARTERIES	VEINS
1	BLOOD CIRCULATION	ARTERIES CARRY BLOOD AWAY FROM THE HEART TO THE TISSUES OF THE BODY.	VEINS CARRY BLOOD FROM THE TISSUES OF THE BODY BACK TO THE HEART.
2	BLOOD TYPE	ARTERIES CARRY OXYGENATED BLOOD EXCEPT PULMONARY ARTERY.	VEINS CARRY DEOXYGENATED BLOOD EXCEPT PULMONARY VEIN.
3	THICKNESS	ARTERIES HAVE THICK ELASTIC MUSCULAR WALLS.	VEINS HAVE THIN NON ELASTIC LESS MUSCULAR WALLS.
4	POSITION	ARTERIES ARE USUALLY POSITIONED DEEPER WITHIN THE BODY.	VEINS ARE USUALLY POSITIONED CLOSER BENEATH THE SURFACE OF THE SKIN.
5	VALVES	VALVES ARE ABSENT.	VALVES ARE PRESENT.
6	LUMEN	THESE POSSESS NARROW LUMEN.	THESE POSSESS WIDE LUMEN
7	PRESSURE	BLOOD FLOWS UNDER HIGH PRESSURE.	BLOOD FLOWS UNDER LOW PRESSURE.
8	COLOR	THESE ARE REDDISH IN COLOR	THESE ARE BLUSH IN COLOR
9	TYPES	PULMONARY AND SYSTEMIC ARTERIES.	SUPERFICIAL VEINS, DEEP VEINS, PULMONARY VEINS AND SYSTEMIC VEINS.
10	INTERNAL DIAMETER	NARROWER (4MM)	WIDER (5MM)
11	VOLUME	LOW (15%)	HIGH (65%)
12	MOVEMENT	THESE SHOW SPURTY MOVEMENT OF BLOOD GIVING PULSE.	THESE SHOW SLUGGISH MOVEMENT OF BLOOD.
13		PULSE IS DETECTABLE IN	PULSE NOT DETECTABLE

- THE CARDIOVASCULAR SYSTEM IS COMPOSED OF TWO CIRCULATORY PATHS: **PULMONARY CIRCULATION**, THE CIRCUIT THROUGH THE LUNGS WHERE BLOOD IS OXYGENATED; AND **SYSTEMIC CIRCULATION**, THE CIRCUIT THROUGH THE REST OF THE BODY TO PROVIDE OXYGENATED BLOOD. THE TWO CIRCUITS ARE LINKED TO EACH OTHER THROUGH THE HEART, CREATING A CONTINUOUS CYCLE OF BLOOD THROUGH THE BODY.

PULMONARY CIRCULATION

- PULMONARY CIRCULATION IS THE MOVEMENT OF BLOOD FROM THE HEART TO THE LUNGS FOR OXYGENATION, THEN BACK TO THE HEART AGAIN. OXYGEN-DEPLETED BLOOD FROM THE BODY LEAVES THE SYSTEMIC CIRCULATION WHEN IT ENTERS THE RIGHT ATRIUM THROUGH THE SUPERIOR AND INFERIOR VENAE CAVAE. THE BLOOD IS THEN PUMPED THROUGH THE TRICUSPID VALVE INTO THE RIGHT VENTRICLE. FROM THE RIGHT VENTRICLE, BLOOD IS PUMPED THROUGH THE PULMONARY VALVE AND INTO THE PULMONARY ARTERY. THE PULMONARY ARTERY SPLITS INTO THE RIGHT AND LEFT PULMONARY ARTERIES AND TRAVEL TO EACH LUNG.
- AT THE LUNGS, THE BLOOD TRAVELS THROUGH CAPILLARY BEDS ON THE ALVEOLI WHERE GAS EXCHANGE OCCURS, REMOVING CARBON DIOXIDE AND ADDING OXYGEN TO THE BLOOD. THE OXYGENATED BLOOD THEN LEAVES THE LUNGS THROUGH PULMONARY VEINS, WHICH RETURNS IT TO THE LEFT ATRIUM, COMPLETING THE PULMONARY CIRCUIT. AS THE PULMONARY CIRCUIT ENDS, THE SYSTEMIC CIRCUIT BEGINS.

SYSTEMIC CIRCULATION

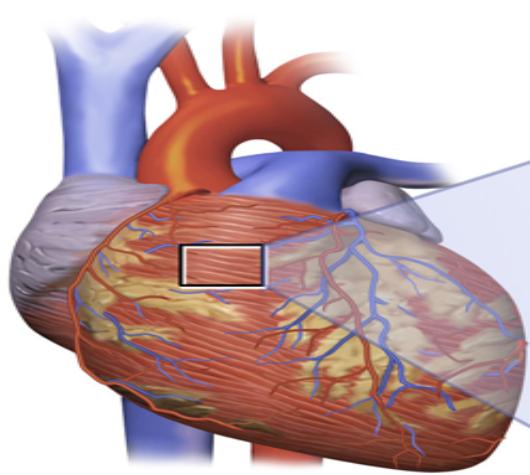
- SYSTEMIC CIRCULATION IS THE MOVEMENT OF BLOOD FROM THE HEART THROUGH THE BODY TO PROVIDE OXYGEN AND NUTRIENTS TO THE TISSUES OF THE BODY WHILE BRINGING DEOXYGENATED BLOOD BACK TO THE HEART. OXYGENATED BLOOD ENTERS THE LEFT ATRIUM FROM THE PULMONARY VEINS. THE BLOOD IS THEN PUMPED THROUGH THE MITRAL VALVE INTO THE LEFT VENTRICLE. FROM THE LEFT VENTRICLE, BLOOD IS PUMPED THROUGH THE AORTIC VALVE AND INTO THE AORTA, THE BODY'S PPT TO PDF BY DLM INFO SOFT LARGEST ARTERY TO OTHER PARTS

- THE ARTERIES BRANCH INTO SMALLER ARTERIES, ARTERIOLES, AND FINALLY CAPILLARIES. GAS AND NUTRIENT EXCHANGE WITH THE TISSUES OCCURS WITHIN THE CAPILLARIES THAT RUN THROUGH THE TISSUES. METABOLIC WASTE AND CARBON DIOXIDE DIFFUSE OUT OF THE CELL INTO THE BLOOD, WHILE OXYGEN AND GLUCOSE IN THE BLOOD DIFFUSES OUT OF THE BLOOD AND INTO THE CELL. SYSTEMIC CIRCULATION KEEPS THE METABOLISM OF EVERY ORGAN AND EVERY TISSUE IN THE BODY ALIVE, WITH THE EXCEPTION OF THE PARENCHYMA OF THE LUNGS, WHICH ARE SUPPLIED BY PULMONARY CIRCULATION.

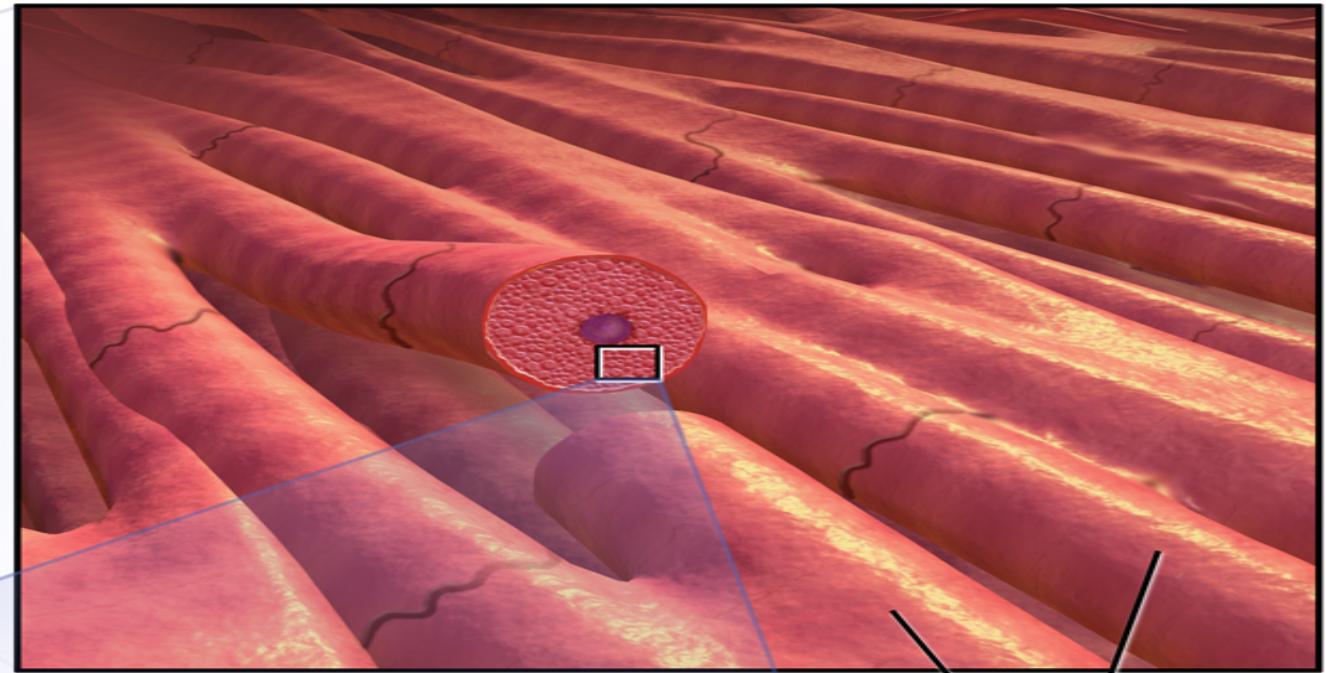
- THE DEOXYGENATED BLOOD CONTINUES THROUGH THE CAPILLARIES WHICH MERGE INTO VENULES, THEN VEINS, AND FINALLY THE VENAE CAVAE, WHICH DRAIN INTO THE RIGHT ATRIUM OF THE HEART. FROM THE RIGHT ATRIUM, THE BLOOD WILL TRAVEL THROUGH THE

CARDIAC MUSCLE

- COORDINATED CONTRACTIONS OF CARDIAC MUSCLE CELLS IN THE HEART PUMP BLOOD OUT OF THE ATRIA AND VENTRICLES TO THE BLOOD VESSELS.
- ALTHOUGH IT IS STRIATED, CARDIAC MUSCLE DIFFERS FROM SKELETAL MUSCLE IN THAT IT IS HIGHLY BRANCHED WITH CELLS CONNECTED BY OVERLAPPING PROJECTIONS OF THE SARCOLEMMA CALLED **INTERCALATED DISCS**. THESE DISCS CONTAIN DESMOSOMES AND GAP JUNCTIONS
- THESE CROSS STRIATIONS ARE FORMED BY ROTATING SEGMENTS OF THICK AND THIN PROTEIN FILAMENTS. LIKE SKELETAL MUSCLE, ~~PPT TO PDF BY DLM INFOSOFT~~ THE PRIMARY STRUCTURAL PROTEINS

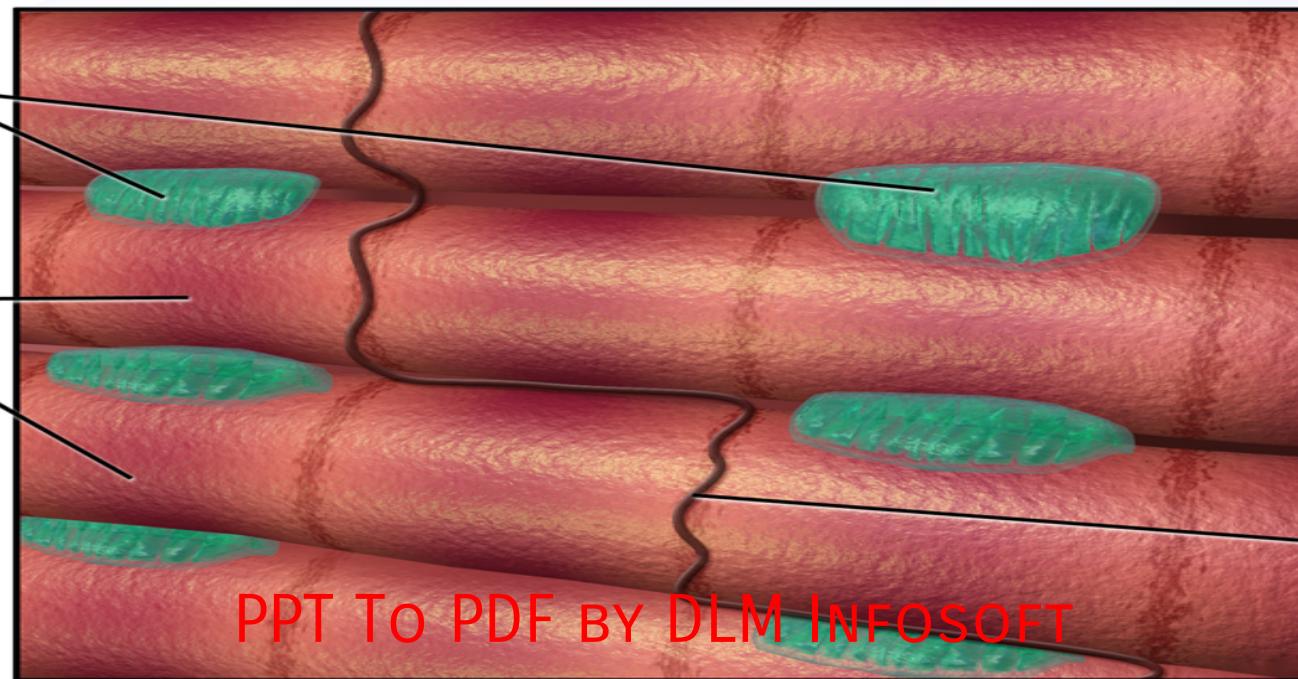


Cardiac Muscle



Mitochondria

Myofibrils



Cardiac muscle cells

Intercalated disc

CARDIAC MUSCLES ARE MADE UP OF

INTERCALATED DISCS

- INTERCALATED DISCS ARE SMALL CONNECTIONS THAT JOIN CARDIAC MUSCLE CELLS (CARDIOMYOCYTES) TO EACH OTHER.

GAP JUNCTIONS

- GAP JUNCTIONS ARE PART OF THE INTERCALATED DISCS. WHEN ONE CARDIAC MUSCLE CELL IS STIMULATED TO CONTRACT, A GAP JUNCTION TRANSFERS THE STIMULATION TO THE NEXT CARDIAC CELL. THIS ALLOWS THE MUSCLE TO CONTRACT IN A COORDINATED WAY.

DESMOSOMES

- LIKE GAP JUNCTIONS, DESMOSOMES ARE ALSO FOUND WITHIN INTERCALATED DISCS. THEY HELP HOLD THE CARDIAC MUSCLE FIBERS TOGETHER DURING A CONTRACTION.

NUCLEUS

- THE NUCLEUS IS THE “CONTROL CENTER” OF A CELL. IT CONTAINS ALL OF THE CELL’S GENETIC MATERIAL. WHILE SKELETAL MUSCLE CELLS CAN HAVE MULTIPLE NUCLEI, CARDIAC MUSCLE CELLS TYPICALLY ONLY HAVE ONE NUCLEUS.

ELECTROPHYSIOLOGIC PROPERTIES OF THE HEART/CARDIAC MUSCLE

1. AUTOMATICITY – THE ABILITY TO SPONTANEOUSLY GENERATE AND DISCHARGE AN ELECTRICAL IMPULSE.
2. RHYTHMICITY; THE ABILITY TO SEND ELECTRICAL IMPULSES IN A REGULARLY, EVENLY MANNER.
3. EXCITABILITY – THE ABILITY OF THE CELL TO RESPOND TO AN ELECTRICAL IMPULSE.
4. CONDUCTIVITY – THE ABILITY TO TRANSMIT AN ELECTRICAL IMPULSE FROM ONE CELL TO THE NEXT.
5. CONTRACTILITY – THE ABILITY OF THE CELL TO SHORTEN AND LENGTHEN ITS FIBERS.
6. REFRACTORINESS; CELLS' INABILITY TO RESPOND TO ANOTHER ELECTRICAL IMPULSE

AUTOMATICITY/RHYTHMICITY

- ABILITY OF A TISSUE TO PRODUCE ITS OWN IMPULSES REGULARLY- ALSO CALLED AUTORHYTHMICITY OR SELF EXCITATION.
- THIS PROPERTY IS PRESENT IN ALL TISSUES OF THE HEART. HOWEVER, THE HEART HAS SPECIALIZED EXCITATORY STRUCTURE FROM WHICH DISCHARGE OF IMPULSES IS RAPID. THIS SPECIALIZED STRUCTURES ARE CALLED PACEMAKERS.

PACEMAKER; DEFINED AS PART OF THE HEART FROM WHICH IMPULSES FOR HEARTBEAT ARE PRODUCED NORMALLY.

IN MAMMALIAN HEART , THE PACEMAKER IS THE SINOATRIAL (SA)NODE NAMED BY LEWIS SIR THOMAS IN 1918

LOCATION OF THE SA NODE

- SA NODE, IS A SMALL STRIP OF MODIFIED CARDIAC MUSCLE SITUATED IN THE SUPERIOR PART OF THE R ATRIUM JUST BELOW THE OPENING OF THE SVC.

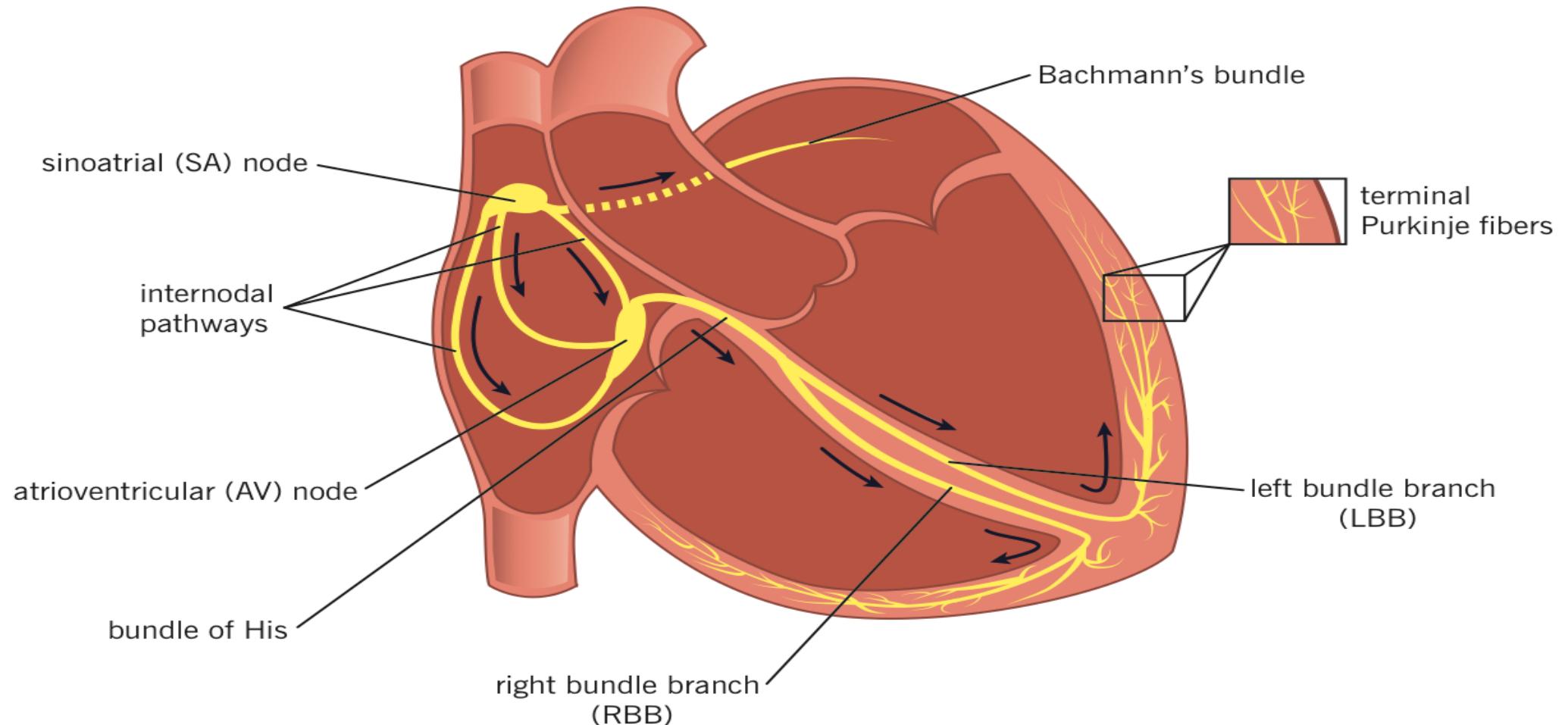
RHYTHMICITY OF OTHER PARTS

- SA NODE 70-80/MINUTE
- AV NODE 40-60/MINUTE
- ATRIAL MUSCLE 40-60/MIN
- VENTRICULAR MUSCLE 20-40/MIN
- PURKINJE FIBERS 35-40/MIN

CONDUCTIVITY

- IT IS THE ABILITY OF CARDIAC MUSCLE TO SPREAD EXCITATORY IMPULSES IN THE HEART TISSUE THROUGH A SPECIALIZED CONDUCTIVE SYSTEM IN THE CARDIAC MUSCLE NAMELY SA NODE, AV NODE, BUNDLE OF HIS, RIGHT & LEFT BUNDLE BRANCHES AND THE PURKINJE FIBRES.

CONDUCTIVE PATHWAY OF THE HEART



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VELOCITY OF IMPULSE AT DIFFERENT PARTS OF THE CONDUCTIVE SYSTEM

- ATRIAL MUSCLE 0.3METER/SECOND
- AV NODE 0.05M/S
- BUNDLE OF HIS 0.12M/S
- PURKINJE FIBERS 4M/S
- VENTRICULAR MUSCLE 0.5M/S

EXCITABILITY

- IN ALL TISSUES, THE INITIAL RESPONSE TO A STIMULUS IS THE DEVELOPMENT OF ACTION POTENTIAL. IT IS FOLLOWED BY THE PHYSIOLOGIC ACTION, IN THIS CASE CONTRACTION, SECRETION ETC.

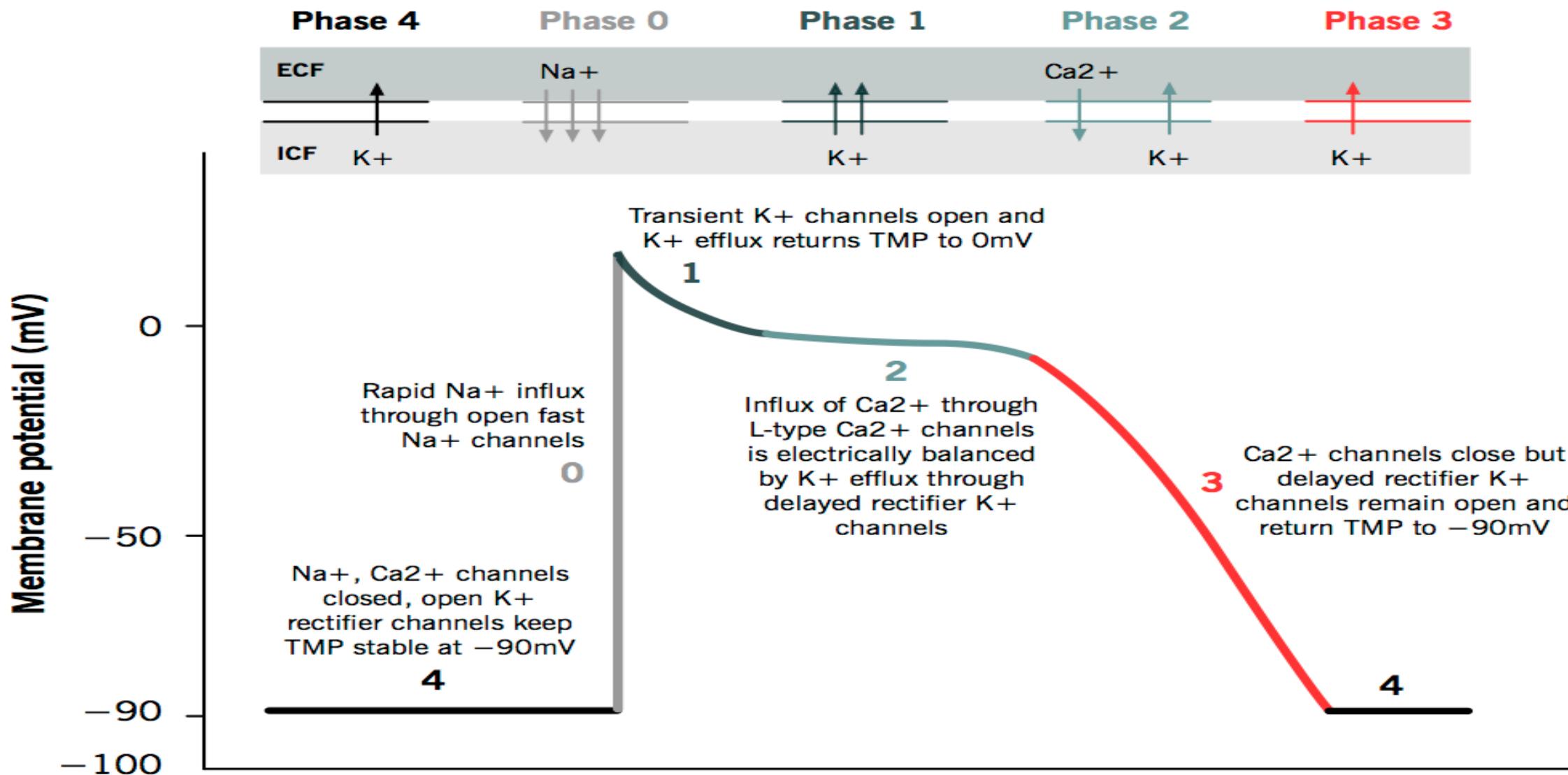
CARDIAC MEMBRANE POTENTIAL

- THE TRANSMEMBRANE POTENTIAL (TMP) IS THE ELECTRICAL POTENTIAL DIFFERENCE (VOLTAGE) BETWEEN THE INSIDE AND THE OUTSIDE OF A CELL. WHEN THERE IS A *NET* MOVEMENT OF +VE IONS *INTO* A CELL, THE TMP BECOMES MORE +VE, AND WHEN THERE IS A *NET* MOVEMENT OF +VE IONS *OUT* OF A CELL, TMP BECOMES MORE -VE.
- ION CHANNELS HELP MAINTAIN IONIC CONCENTRATION GRADIENTS AND CHARGE DIFFERENTIALS BETWEEN THE INSIDE AND OUTSIDE OF THE CARDIOMYOCYTES.

PROPERTIES OF CARDIAC ION CHANNELS

Action potential of cardiac muscles

Grigoriy Ikonnikov and Eric Wong



ACTION POTENTIAL: ELECTRICAL STIMULATION CREATED BY A SEQUENCE OF ION FLUXES THROUGH SPECIALIZED CHANNELS IN THE MEMBRANE (*SARCOLEMMA*) OF CARDIOMYOCYTES THAT LEADS TO CARDIAC CONTRACTION.

ACTION POTENTIAL IN CARDIOMYOCYTES

- THE ACTION POTENTIAL IN TYPICAL CARDIOMYOCYTES IS COMPOSED OF 5 PHASES (0-4), BEGINNING AND ENDING WITH PHASE 4.

PHASE 4: THE RESTING PHASE

- THE RESTING POTENTIAL IN A CARDIOMYOCYTE IS -90 MV DUE TO A CONSTANT OUTWARD LEAK OF K^+ THROUGH *INWARD RECTIFIER CHANNELS*.

PHASE 0: DEPOLARIZATION

- AN ACTION POTENTIAL TRIGGERED IN A NEIGHBOURING CARDIOMYOCYTE OR PACEMAKER CELL CAUSES THE TMP TO RISE ABOVE -90 mV .
- FAST Na^+ CHANNELS START TO OPEN ONE BY ONE AND Na^+ LEAKS INTO THE CELL, FURTHER RAISING THE TMP.
- TMP APPROACHES -70mV , THE **THRESHOLD POTENTIAL** IN CARDIOMYOCYTES, I.E. THE POINT AT WHICH ENOUGH FAST Na^+ CHANNELS HAVE OPENED TO GENERATE A SELF-SUSTAINING INWARD Na^+ CURRENT.
- THE LARGE Na^+ CURRENT RAPIDLY DEPOLARIZES THE TMP TO 0 mV AND SLIGHTLY *ABOVE* 0 mV FOR A TRANSIENT PERIOD OF TIME CALLED **THE OVERSHOOT**; FAST Na^+ CHANNELS CLOSE (RECALL THAT FAST Na^+ CHANNELS ARE *TIME-DEPENDENT*).

PHASE 1: EARLY REPOLARIZATION

- TMP IS NOW SLIGHTLY POSITIVE.
- SOME K⁺ CHANNELS OPEN BRIEFLY AND AN OUTWARD FLOW OF K⁺ RETURNS THE TMP TO APPROXIMATELY 0 mV.

PHASE 2: THE PLATEAU PHASE

- L-TYPE CA²⁺ CHANNELS ARE STILL OPEN AND THERE IS A SMALL, CONSTANT INWARD CURRENT OF CA²⁺. THIS BECOMES SIGNIFICANT IN THE *EXCITATION-CONTRACTION COUPLING* PROCESS DESCRIBED BELOW.
- K⁺ LEAKS OUT DOWN ITS CONCENTRATION GRADIENT THROUGH *DELAYED RECTIFIER* K⁺ CHANNELS.
- THESE TWO COUNTERCURRENTS ARE ELECTRICALLY BALANCED, AND THE TMP IS MAINTAINED AT A *PLATEAU* JUST BELOW 0 mV THROUGHOUT PHASE 2.

PHASE 3: REPOLARIZATION

- Ca^{2+} CHANNELS ARE GRADUALLY INACTIVATED.
- PERSISTENT OUTFLOW OF K^+ , NOW EXCEEDING Ca^{2+} INFLOW, BRINGS TMP BACK TOWARDS RESTING POTENTIAL OF -90 mV TO PREPARE THE CELL FOR A NEW CYCLE OF DEPOLARIZATION.
- NORMAL TRANSMEMBRANE IONIC CONCENTRATION GRADIENTS ARE RESTORED BY RETURNING Na^+ AND Ca^{2+} IONS TO THE EXTRACELLULAR ENVIRONMENT, AND K^+ IONS TO THE CELL INTERIOR. THE PUMPS INVOLVED INCLUDE THE SARCOLEMMAL $\text{Na}^+-\text{Ca}^{2+}$ EXCHANGER, Ca^{2+} -ATPASE AND Na^+-K^+ -ATPASE.

CONTRACTILITY

- THE ABILITY OF THE CELL TO SHORTEN AND LENGTHEN ITS FIBERS(CONTRACTION) AFTER RECEIVING A STIMULUS.
- FACTORS AFFECTING THE CONTRACTILE PROPERTIES OF THE CARDIAC MUSCLE ARE;
 - I. **ALL OR NONE LAW;** IF STRENGTH OF STIMULUS IS BELOW THRESHOLD LEVEL, NO RESPONSE.
 - II. **STAIRCASE PHENOMENON;** GRADUAL INCREASE IN THE FORCE OF CONTRACTION.
 - III. **SUMMATION OF SUBLIMINAL STIMULI;** STIMULUS WITH SUBLINGUAL STRENGTH NO RESPONSE BUT FEW STIMULI WITH SAME SUBLINGUAL STRENGTH IN SUCCESSION, RESPONSE THRU CONTRACTION.

ALL OR NONE LAW

- WHEN A STIMULUS IS APPLIED, WHATEVER MAY BE THE STRENGTH, THE WHOLE CARDIAC MUSCLE GIVES MAXIMUM RESPONSE OR IT DOES NOT GIVE ANY RESPONSE AT ALL.
- BELOW THE THRESHOLD LEVEL, (STRENGTH OF THE STIMULUS NOT ADEQUATE), THE MUSCLE DOES NOT GIVE RESPONSE.
- ALL OR NONE LAW IS APPLICABLE TO WHOLE CARDIAC MUSCLE. IT IS BECAUSE OF SYNCYTIAL ARRANGEMENT OF CARDIAC MUSCLE. IN THE CASE OF SKELETAL MUSCLE, ALL OR NONE LAW IS APPLICABLE ONLY TO A SINGLE MUSCLE FIBER.

STAIRCASE PHENOMENON

- GRADUAL INCREASE IN THE FORCE OF CONTRACTION.
- IT OCCURS BECAUSE OF THE BENEFICIAL EFFECT WHICH FACILITATES THE FORCE OF SUCCESSIVE CONTRACTION. SO THERE IS A GRADUAL INCREASE IN FORCE OF CONTRACTION.

SUMMATION OF SUBLIMINAL STIMULI

- WHEN A STIMULUS WITH A SUBLIMINAL STRENGTH IS APPLIED, THE QUIESCENT HEART DOES NOT SHOW ANY RESPONSE. WHEN FEW STIMULI WITH SAME SUBLIMINAL STRENGTH ARE APPLIED IN SUCCESSION, THE HEART SHOWS RESPONSE BY CONTRACTION. IT IS DUE TO THE SUMMATION OF THE STIMULI.

REFRACTORY PERIOD

- DEFINED AS THE TIME FROM PHASE 0 UNTIL THE NEXT POSSIBLE DEPOLARIZATION OF A MYOCYTE, I.E. ONCE ENOUGH FAST Na^+ CHANNELS HAVE RECOVERED (AS TMP DECREASES BELOW -50 mV).
- CARDIOMYOCYTES HAVE A LONGER REFRACTORY PERIOD THAN OTHER MUSCLE CELLS GIVEN THE LONG PLATEAU FROM SLOW Ca^{2+} CHANNELS (PHASE 2). THIS IS A PHYSIOLOGICAL MECHANISM ALLOWING SUFFICIENT TIME FOR THE VENTRICLES TO EMPTY AND REFILL PRIOR TO THE NEXT CONTRACTION. TOTAL DURATION IS 0.53s

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SIGNIFICANCE OF LONG REFRACTORY PERIOD IN CARDIAC MUSCLE

- 1. SUMMATION OF CONTRACTIONS DOES NOT OCCUR**
- 2. FATIGUE DOES NOT OCCUR**
- 3. TETANUS DOES NOT OCCUR**

Contractility

- ◆ The ability of the myofibrils to shorten in length and produce a contraction.
- ◆ Not measured directly
- ◆ Factors that affect contractility:

PRELOAD

AFTERLOAD

DRUGS

**CARDIAC OXYGENATION
FUNCTIONAL MYOCARDIUM**

CARDIAC CYCLE

- THE CARDIAC CYCLE IS THE SEQUENCE OF COORDINATED EVENTS THAT OCCURS WHEN THE HEART BEATS. AS THE HEART BEATS, IT CIRCULATES BLOOD THROUGH PULMONARY AND SYSTEMIC CIRCUITS OF THE BODY.
- ONE CARDIAC CYCLE IS COMPLETED WHEN THE HEART CHAMBERS FILL WITH BLOOD AND BLOOD IS THEN PUMPED OUT OF THE HEART.
- THE CARDIAC CYCLE COMPRISSES A COMPLETE RELAXATION AND CONTRACTION OF BOTH THE ATRIA AND VENTRICLES, AND LASTS APPROXIMATELY **0.8 SECONDS**.
- THUS, **CARDIAC CYCLE** IS THE PERIOD OF TIME BETWEEN THE ONSET OF ATRIAL CONTRACTION (ATRIAL SYSTOLE) AND VENTRICULAR RELAXATION (VENTRICULAR DIASTOLE).
- THE CARDIAC CYCLE INTEGRATES PRESSURE, VOLUME, AND

CARDIAC CYCLE PHASES

- AT THE BEGINNING OF THE CARDIAC CYCLE, BOTH THE ATRIA AND VENTRICLES ARE RELAXED (DIASTOLE). BLOOD IS FLOWING INTO THE RIGHT ATRIUM FROM THE SUPERIOR AND INFERIOR VENAE CAVAE AND THE CORONARY SINUS.
- BLOOD FLOWS INTO THE LEFT ATRIUM FROM THE FOUR PULMONARY VEINS. THE TWO ATRIOVENTRICULAR VALVES, THE TRICUSPID AND MITRAL VALVES, ARE BOTH OPEN, SO BLOOD FLOWS UNIMPEDED FROM THE ATRIA AND INTO THE VENTRICLES. APPROXIMATELY 70–80 PERCENT OF VENTRICULAR FILLING OCCURS BY THIS METHOD. THE TWO SEMILUNAR VALVES, THE PULMONARY AND AORTIC VALVES, ARE CLOSED, PREVENTING BACKFLOW OF BLOOD INTO THE RIGHT AND LEFT VENTRICLES FROM PPT TO PDF BY DLM INFOSOFT THE PULMONARY TRUNK ON THE RIGHT AND THE AORTA ON THE LEFT.

SEVEN (7) EVENTS OF THE CARDIAC CYCLE

1. ATRIAL CONTRACTION
2. ISOVOLUMETRIC VENTRICULAR CONTRACTIONS
3. RAPID VENTRICULAR EJECTION
4. REDUCED VENTRICULAR EJECTION
5. ISOVOLUMETRIC VENTRICULAR RELAXATION
6. RAPID VENTRICULAR FILLING(VENTRICULAR GALLOP AND S3)
7. REDUCED VENTRICULAR FILLING (ATRIAL GALLOP AND S4)

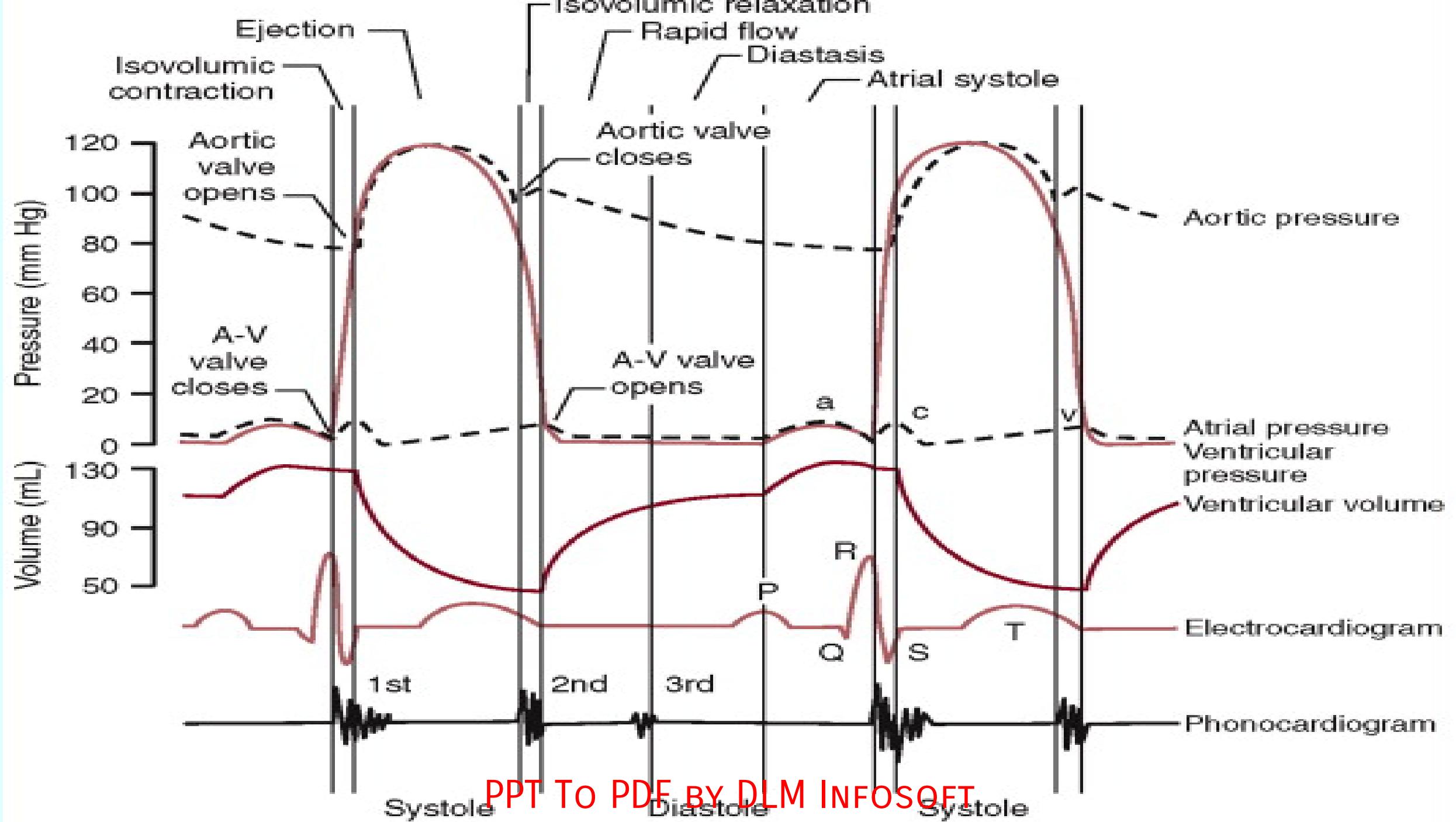
ATRIAL CONTRACTION (A-V VALVES OPEN; SEMILUNAR VALVES CLOSED) PHASE 1

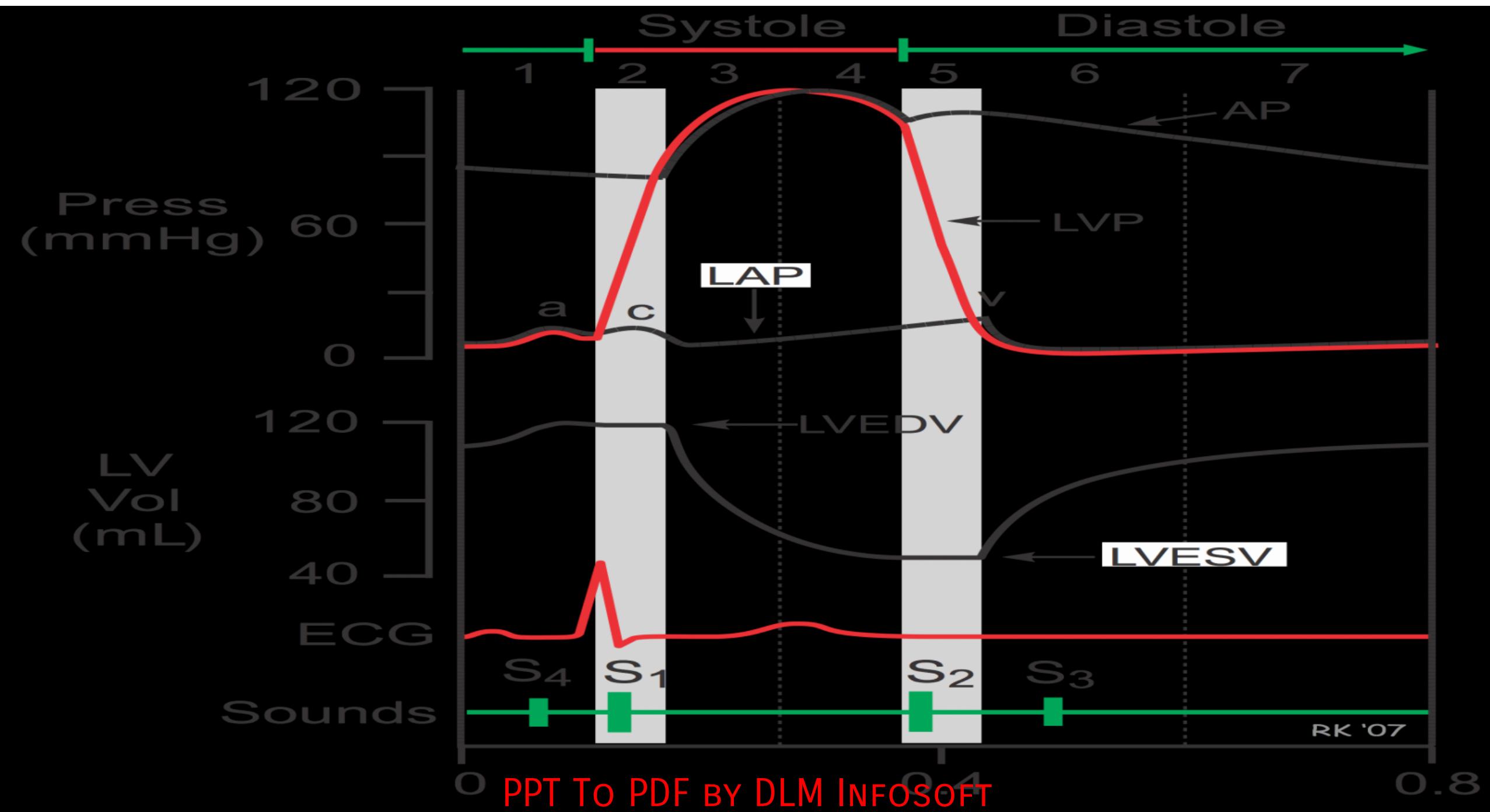
- ATRIAL DEPOLARIZATION INITIATES CONTRACTION OF THE ATRIAL MUSCULATURE. AS THE ATRIA CONTRACT, THE PRESSURE WITHIN THE ATRIAL CHAMBERS INCREASES, WHICH FORCES MORE BLOOD FLOW ACROSS THE OPEN ATRIOVENTRICULAR (AV) VALVES, LEADING TO A RAPID FLOW OF BLOOD INTO THE VENTRICLES.
- ATRIAL CONTRACTION (ATRIAL KICK) NORMALLY ACCOUNTS FOR ABOUT 10- 20% OF LEFT VENTRICULAR FILLING. IT LASTS FOR ABOUT 0.11s.
- ATRIAL CONTRACTION DOES PRODUCE A SMALL INCREASE IN VENOUS PRESSURE THAT CAN BE NOTED AS THE "A-WAVE" OF THE LEFT ATRIAL PRESSURE (LAP). JUST FOLLOWING THE PEAK OF THE A-WAVE IS THE

- AFTER ATRIAL CONTRACTION IS COMPLETE, THE ATRIAL PRESSURE BEGINS TO FALL CAUSING A PRESSURE GRADIENT REVERSAL ACROSS THE AV VALVES. THIS CAUSES THE VALVES TO FLOAT UPWARD (PRE-POSITION) BEFORE CLOSURE. AT THIS TIME, THE VENTRICULAR VOLUMES ARE MAXIMAL, WHICH IS TERMED THE END-DIASTOLIC VOLUME (EDV). THE LEFT VENTRICULAR EDV (LVEDV), WHICH IS TYPICALLY ABOUT 120 ML, REPRESENTS THE VENTRICULAR PRELOAD AND IS ASSOCIATED WITH END-DIASTOLIC PRESSURES OF 8-12 MMHG AND 3-6 MMHG IN THE LEFT AND RIGHT VENTRICLES, RESPECTIVELY.
- A HEART SOUND IS SOMETIMES NOTED DURING ATRIAL CONTRACTION (FOURTH HEART SOUND, S4). THIS SOUND IS CAUSED BY VIBRATION OF THE VENTRICULAR WALL DURING ATRIAL CONTRACTION. GENERALLY, IT IS NOTED WHEN THE VENTRICLE COMPLIANCE IS REDUCED ("STIFF" VENTRICLE). AS OCCURS IN VENTRICULAR HYPERTROPHY AND IN MANY OLDER INDIVIDUALS

PRESSURE CHANGES IN THE ATRIA (THE A, C, AND V WAVES)

- THE *A WAVE* IS CAUSED BY ATRIAL CONTRACTION. ORDINARILY, THE *RIGHT* ATRIAL PRESSURE INCREASES 4 TO 6 MM HG DURING ATRIAL CONTRACTION, AND THE *LEFT* ATRIAL PRESSURE INCREASES ABOUT 7 TO 8 MM HG.
- THE *C WAVE* OCCURS WHEN THE VENTRICLES BEGIN TO CONTRACT; IT IS CAUSED PARTLY BY SLIGHT BACKFLOW OF BLOOD INTO THE ATRIA AT THE ONSET OF VENTRICULAR CONTRACTION BUT MAINLY BY BULGING OF THE A-V VALVES BACKWARD TOWARD THE ATRIA BECAUSE OF INCREASING PRESSURE IN THE VENTRICLES.
- THE *V WAVE* OCCURS TOWARD THE END OF VENTRICULAR CONTRACTION; IT RESULTS FROM SLOW FLOW OF BLOOD INTO THE ATRIA FROM THE VEINS WHILE THE A-V VALVES ARE CLOSED DURING VENTRICULAR CONTRACTION. THEN, WHEN VENTRICULAR CONTRACTION IS OVER, THE A-V VALVES OPEN.





ISOVOLUMETRIC VENTRICULAR CONTRACTIONS (ALL VALVES CLOSED) (PHASE 2)

- THIS PHASE OF THE CARDIAC CYCLE BEGINS WITH THE APPEARANCE OF THE QRS COMPLEX OF THE ECG, WHICH REPRESENTS VENTRICULAR DEPOLARIZATION. THIS TRIGGERS EXCITATION-CONTRACTION COUPLING, MYOCYTE CONTRACTION AND A RAPID INCREASE IN INTRAVENTRICULAR PRESSURE. EARLY IN THIS PHASE, THE RATE OF PRESSURE DEVELOPMENT BECOMES MAXIMAL. THIS IS REFERRED TO AS MAXIMAL dP/dt .
- THE AV VALVES CLOSE WHEN INTRAVENTRICULAR PRESSURE EXCEEDS ATRIAL PRESSURE.
- CLOSURE OF THE AV VALVES RESULTS IN THE FIRST HEART PPT TO PDF BY DLM INFOSOFT (S1) T (S2) (S3)

- DURING THE TIME PERIOD BETWEEN THE CLOSURE OF THE AV VALVES AND THE OPENING OF THE AORTIC AND PULMONIC VALVES, VENTRICULAR PRESSURE RISES RAPIDLY WITHOUT A CHANGE IN VENTRICULAR VOLUME (I.E., NO EJECTION OCCURS).
- VENTRICULAR VOLUME DOES NOT CHANGE BECAUSE ALL VALVES ARE CLOSED DURING THIS PHASE. CONTRACTION, THEREFORE, IS SAID TO BE "ISOVOLUMIC" OR "ISOVOLUMETRIC."
- THE RATE OF PRESSURE INCREASE IN THE VENTRICLES IS DETERMINED BY THE RATE OF CONTRACTION OF THE MUSCLE FIBERS, WHICH IS DETERMINED BY MECHANISMS GOVERNING

RAPID VENTRICULAR EJECTION (AORTIC AND PULMONIC VALVES OPEN) PHASE 3

- RAPID EJECTION: THIS PHASE REPRESENTS INITIAL, RAPID EJECTION OF BLOOD INTO THE AORTA AND PULMONARY ARTERIES FROM THE LEFT AND RIGHT VENTRICLES, RESPECTIVELY. EJECTION BEGINS WHEN THE INTRAVENTRICULAR PRESSURES EXCEED THE PRESSURES WITHIN THE AORTA AND PULMONARY ARTERY, WHICH CAUSES THE AORTIC AND PULMONIC VALVES TO OPEN.
- NO HEART SOUNDS ARE ORDINARILY NOTED DURING EJECTION BECAUSE THE OPENING OF HEALTHY VALVES IS SILENT. THE PRESENCE OF SOUNDS DURING EJECTION (I.E., SYSTOLIC MURMURS) INDICATE VALVE DISEASE OR INTRACARDIAC SHUNTS.
- LEFT ATRIAL PRESSURE INITIALLY DECREASES AS THE ATRIAL BASE IS PULLED DOWNWARD, EXPANDING THE ATRIAL CHAMBERS. BLOOD CONTINUES TO FLOW INTO THE ATRIA FROM THEIR RESPECTIVE VENOUS INFLOW TRACTS AND THE

ISOVOLUMETRIC RELAXATION (PHASE 5) ALL VALVES CLOSED

- WHEN THE INTRAVENTRICULAR PRESSURES FALL SUFFICIENTLY AT THE END OF PHASE 4, THE AORTIC AND PULMONIC VALVES ABRUPTLY CLOSE (AORTIC PRECEDES PULMONIC) CAUSING THE SECOND HEART SOUND (S2) AND THE BEGINNING OF ISOVOLUMETRIC RELAXATION. VALVE CLOSURE IS ASSOCIATED WITH A SMALL BACKFLOW OF BLOOD INTO THE VENTRICLES AND A CHARACTERISTIC NOTCH (INCISURA OR DICROTIC NOTCH) IN THE AORTIC AND PULMONARY ARTERY PRESSURE TRACINGS.
- AFTER VALVE CLOSURE, THE AORTIC AND PULMONARY ARTERY PRESSURES RISE SLIGHTLY (DICROTIC WAVE) FOLLOWING BY A SLOW DECLINE IN PRESSURE.
- ALTHOUGH VENTRICULAR PRESSURES DECREASE DURING THIS PHASE, VOLUMES DO NOT CHANGE BECAUSE ALL VALVES ARE CLOSED. THE VOLUME OF BLOOD THAT REMAINS IN A VENTRICLE IS CALLED THE END-SYSTOLIC VOLUME AND IS ~50 ML IN THE LEFT VENTRICLE. THE DIFFERENCE BETWEEN THE END-DIASTOLIC VOLUME AND THE END-SYSTOLIC VOLUME IS ~70 ML AND REPRESENTS THE STROKE VOLUME.
- LEFT ATRIAL PRESSURE (~~PPT TO PDF BY DLM INFO SOFT~~) CONTINUES TO RISE BECAUSE OF VENOUS RETURN FROM THE LUNGS. THE PEAK LAP AT THE END OF THIS PHASE IS TERMED THE V-

RAPID FILLING (PHASE 6)- A-V VALVES OPEN

- AS THE VENTRICLES CONTINUE TO RELAX AT THE END OF PHASE 5, THE INTRAVENTRICULAR PRESSURES WILL AT SOME POINT FALL BELOW THEIR RESPECTIVE ATRIAL PRESSURES. WHEN THIS OCCURS, THE AV VALVES RAPIDLY OPEN AND PASSIVE VENTRICULAR FILLING BEGINS.
- THE OPENING OF THE MITRAL VALVE CAUSES A RAPID FALL IN LAP. THE PEAK OF THE LAP JUST BEFORE THE VALVE OPENS IS THE "V-WAVE." THIS IS FOLLOWED BY THE Y-DESCENT OF THE LAP. A SIMILAR WAVE AND DESCENT ARE FOUND IN THE RIGHT ATRIUM AND IN THE JUGULAR VEIN.
- VENTRICULAR FILLING IS NORMALLY SILENT. WHEN A THIRD HEART SOUND (S3) IS AUDIBLE DURING RAPID VENTRICULAR FILLING, IT MAY REPRESENT TENSING OF CHORDAE TENDINEAE AND AV RING DURING

REDUCED FILLING (PHASE 7)-A-V VALVES OPEN

- AS THE VENTRICLES CONTINUE TO FILL WITH BLOOD AND EXPAND, THEY BECOME LESS COMPLIANT AND THE INTRAVENTRICULAR PRESSURES RISE. THE INCREASE IN INTRAVENTRICULAR PRESSURE REDUCES THE PRESSURE GRADIENT ACROSS THE AV VALVES SO THAT THE RATE OF FILLING FALLS LATE IN DIASTOLE.
- IN NORMAL, RESTING HEARTS, THE VENTRICLE IS ABOUT 90% FILLED BY THE END OF THIS PHASE. IN OTHER WORDS, ABOUT 90% OF VENTRICULAR FILLING OCCURS BEFORE ATRIAL CONTRACTION (PHASE 1) AND THEREFORE IS PASSIVE.

VOLUME CHANGES IN THE CARDIAC CYCLE

ACTIONS OF THE HEART

FOUR TYPES

1. CHRONOTROPIC ACTION
2. INOTROPIC ACTION
3. DROMOTROPIC ACTION
4. BATHMOTROPIC ACTION

CHRONOTROPIC ACTION

- IS THE FREQUENCY OF HEART BEAT OR HEART RATE. THERE ARE 2 TYPES

Ø TACHYCARDIA- INCREASE IN HR

Ø BRADYCARDIA- DECREASE IN HR

INOTROPIC ACTION

- IS THE FORCE OF CONTRACTION OF THE HEART. THERE ARE 2 TYPES

Ø POSITIVE INOTROPIC- INCREASE IN THE FORCE OF CONTRACTION

Ø NEGATIVE INOTROPIC- DECREASE IN THE FORCE OF

DROMOTROPIC ACTION

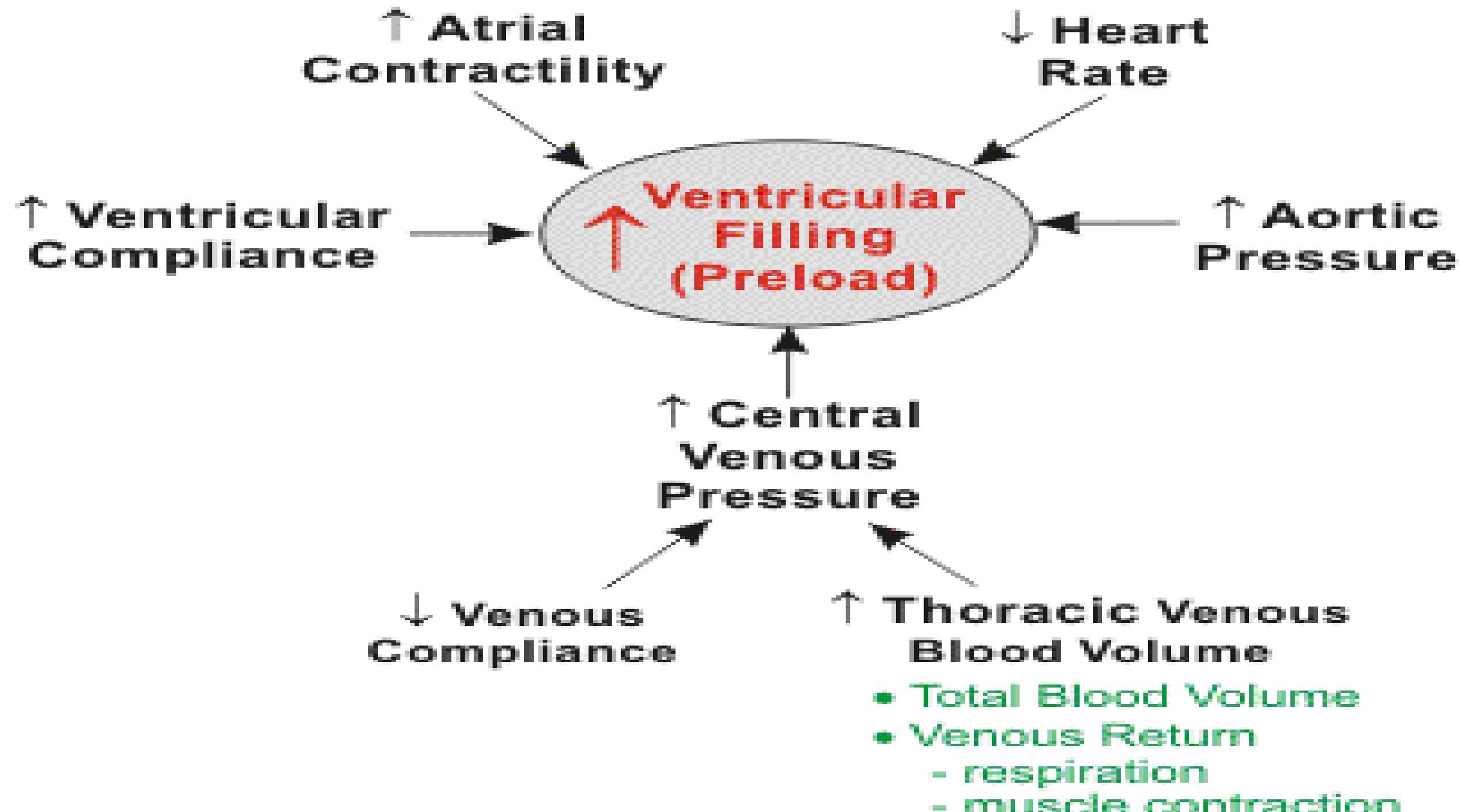
- IS THE CONDUCTION OF IMPULSE THROUGH THE HEART. THERE ARE 2 TYPES
 - Ø POSITIVE DROMOTROPIC ACTION : INCREASE IN VELOCITY OF CONDUCTION
 - Ø NEGATIVE DROMOTROPIC ACTION: DECREASE IN VELOCITY OF CONDUCTION

BATHMOTROPIC ACTION

- IS THE EXCITABILITY OF CARDIAC MUSCLE. THERE ARE 2 TYPES
 - Ø POSITIVE BATHMOTROPIC ACTION: INCREASE IN THE EXCITABILITY OF CARDIAC MUSCLE
 - Ø NEGATIVE BATHMOTROPIC ACTION: DECREASE IN THE EXCITABILITY OF

- PRELOAD CAN BE DEFINED AS THE INITIAL STRETCHING OF THE CARDIAC MYOCYTES PRIOR TO CONTRACTION. PRELOAD, THEREFORE, IS RELATED TO MUSCLE SARCOMERE LENGTH. BECAUSE SARCOMERE LENGTH CANNOT BE DETERMINED IN THE INTACT HEART, OTHER INDICES OF PRELOAD ARE USED SUCH AS VENTRICULAR END-DIASTOLIC VOLUME OR PRESSURE.
- AFTERLOAD IS RELATED TO THE PRESSURE THAT THE VENTRICLE MUST GENERATE IN ORDER TO EJECT BLOOD INTO THE AORTA. AN INCREASE IN AFTERLOAD (E.G., INCREASED AORTIC PRESSURE) PPT TO PDF BY DLM INFOSOFT DECREASES SV, AND CAUSES ESV TO

FACTORS THAT INCREASE PRELOAD



HEART SOUNDS

- HEART SOUNDS ARE THE SOUNDS PRODUCED BY THE MECHANICAL ACTIVITIES OF THE HEART DURING EACH CARDIAC CYCLE. HEART SOUNDS ARE PRODUCED BY MOVEMENTS OF
- BLOOD THROUGH THE CHAMBERS OF THE HEART
- CARDIAC MUSCLE
- VALVES OF THE HEART
- HEART SOUND ARE HEARD BY PLACING THE EAR OVER THE CHEST, STETHOSCOPE, MICROPHONE.

HEART SOUNDS

FOUR (4) HEART SOUNDS ARE PRODUCED DURING EACH CARDIAC CYCLE

- 1. FIRST HEART SOUND S₁**
- 2. SECOND HEART SOUND S₂**
- 3. THIRD HEART SOUND S₃**
- 4. FOURTH HEART SOUND S₄**

FEATURES	1 ST HEART SOUND	2 ND HEART SOUND	3 RD HEART SOUND	4 TH HEART SOUND
OCCURS DURING	ISOMETRIC CONTRACTION PERIOD AND PART OF EJECTION PERIOD	PROTODIASTOLE AND PART OF ISOMETRIC RELAXATION	RAPID FILLING PHASE	ATRIAL SYSTOLE (CONSIDERED PHYSIOLOGIC ATRIAL SOUND.)
CAUSE	CLOSURE OF A-V VALVES	CLOSURE OF SEMILUNAR VALVES	RUSHING OF BLOOD INTO VENTRICLE	CONTRACTION OF ATRIAL MUSCULATURE
CHARACTERISTICS	LONG, SOFT AND LOW PITCHED. RESEMBLES THE WORD “LUBB”	SHORT, SHARP AND HIGH PITCHED. RESEMBLES THE WORD ‘DUB’	LOW PITCHED (CAN BE HEARD WITH THE STETHOSCOPE IN CHILDREN AND ATHLETES)	INAUDIBLE SOUND
DURATION (SEC)	0.10-0.17	0.10-0.14	0.07-0.10	0.02-0.04
FREQUENCY (CYCLES PER UNIT)	25-45	50	1-6	1-4
RELATION WITH ECG	COINCIDES WITH PEAK OF ‘R’	PRECEDES OR APPEARS 0.09S AFTER PEAK OF ‘T’ WAVE	BETWEEN ‘T’ WAVE AND ‘P’ WAVE	BETWEEN ‘T’ WAVE AND ‘Q’ WAVE
No. OF VIBRATIONS IN PHONOCARDIOGRAM	9-13	PPT TO PDF BY DLM INFO SOFTWARE	1-4	1-2

ELECTROCARDIOGRAM (ECG)

- WHEN THE CARDIAC IMPULSE PASSES THROUGH THE HEART, ELECTRICAL CURRENT ALSO SPREADS FROM THE HEART INTO THE ADJACENT TISSUES SURROUNDING THE HEART. A SMALL PORTION OF THE CURRENT SPREADS ALL THE WAY TO THE SURFACE OF THE BODY. IF ELECTRODES ARE PLACED ON THE SKIN ON OPPOSITE SIDES OF THE HEART, ELECTRICAL POTENTIALS GENERATED BY THE CURRENT CAN BE RECORDED; THE RECORDING IS KNOWN AS AN ELECTROCARDIOGRAM

ELECTROCARDIOGRAM (ECG)

- ELECTROCARDIOGRAPHY IS THE TECHNIQUE BY WHICH THE ELECTRICAL ACTIVITIES OF THE HEART ARE STUDIED. THIS TECHNIQUE WAS DISCOVERED BY DUTCH PHYSIOLOGISTS, EINHOVEN WILLEM WHO IS CONSIDERED THE FATHER OF ECG.
- ELECTROCARDIOGRAPH IS THE INSTRUMENT (ECG MACHINE) BY WHICH THE ELECTRICAL ACTIVITIES OF THE HEART ARE RECORDED.
- ELECTROCARDIOGRAM IS THE RECORD OR GRAPHICAL REGISTRATION OF ELECTRICAL ACTIVITIES OF THE HEART, WHICH OCCUR PRIOR TO THE ONSET OF MECHANICAL ACTIVITIES. ECG IS RECORDED IN 12 LEADS.
- THE PAPER USED FOR RECORDING ECG IS CALLED ECG PAPER. ECG GRID REFERS TO THE MARKINGS (LINES) PPT TO PDF BY DLM INFOSOFT

ECG GRID

- THE ECG PAPER HAS HORIZONTAL AND VERTICAL LINES AT REGULAR INTERVALS OF 1MM. EVERY 5TH LINE (5MM) IS THICKENED.
- DURATION: THE DURATION OF DIFFERENT ECG WAVES IS DENOTED BY THE VERTICAL LINES.

Ø INTERVAL B/W 2 THICK LINES (5MM)=0.2s

Ø INTERVAL B/W 2 THIN LINES (1MM)=0.04s

- AMPLITUDE: THE AMPLITUDE OF ECG WAVES IS DENOTED BY HORIZONTAL LINES

Ø INTERVAL B/W 2 THICK LINES (5MM)=0.5mV

Ø INTERVAL B/W 2 THIN LINES (1MM)=0.1mV

THE 12 LEADS OF THE ECG ARE CLASSIFIED INTO

- 1. BIPOLEAR LEADS**
- 2. UNIPOLEAR LEADS**

BIPOLAR LEADS, ALSO CALLED STANDARD LIMB LEADS

TWO ACTIVE ELECTRODES (POSITIVE AND NEGATIVE) ARE CONNECTED TO GET THESE LEADS.

THEY ARE THREE

- 1. LIMB LEAD I**
- 2. LIMB LEAD II**
- 3. LIMB LEAD III**

LIMB LEADS

LIMB LEAD 1

RIGHT ARM CONNECTED TO NEGATIVE TERMINAL
LEFT ARM CONNECTED TO POSITIVE TERMINAL

LIMB LEAD 11

RIGHT ARM CONNECTED TO NEGATIVE TERMINAL
LEFT LEG CONNECTED TO THE POSITIVE TERMINAL

LIMB LEAD 111

LEFT ARM CONNECTED TO NEGATIVE TERMINAL
LEFT LEG CONNECTED TO THE POSITIVE TERMINAL

UNIPOLAR LEADS

- THEY ARE OF TWO TYPES
 - 1. UNIPOLAR LIMB LEADS
 - 2. UNIPOLAR CHEST LEADS

UNIPOLAR LIMB LEADS

- ALSO CALLED AUGMENTED LIMB LEADS. ONE ELECTRODE IS ACTIVE WHILE THE OTHER IS AN INDIFFERENT ELECTRODE.
- THERE ARE THREE UNIPOLAR LIMB LEADS
 1. AVR LEAD
 2. AVL LEAD
 3. AVF LEAD

AVR LEAD	ACTIVE ELECTRODE FROM R ARM, INDIFFERENT CONNECTED TO R ARM AND L LEG
AVL LEAD	ACTIVE ELECTRODE FROM L ARM, INDIFFERENT CONNECTED TO R ARM AND L LEG
AVF LEAD	ACTIVE ELECTRODE FROM L LEG, INDIFFERENT CONNECTED TO R ARM AND L ARM

UNIPOLAR CHEST LEADS

- CHEST LEADS ARE ALSO CALLED PRECARDIAL LEADS. THE INDIFFERENT ELECTRODE IS OBTAINED BY CONNECTING THE THREE LIMBS- L LEG, LARM AND R ARM THROUGH A RESISTANCE OF 5000 OHMS. THE ACTIVE ELECTRODE IS PLACED ON SIX POINTS OVER THE CHEST CALLED V1, V2, V3, V4, V5 AND V6. V INDICATES VECTOR

UNIPOLAR CHEST LEADS

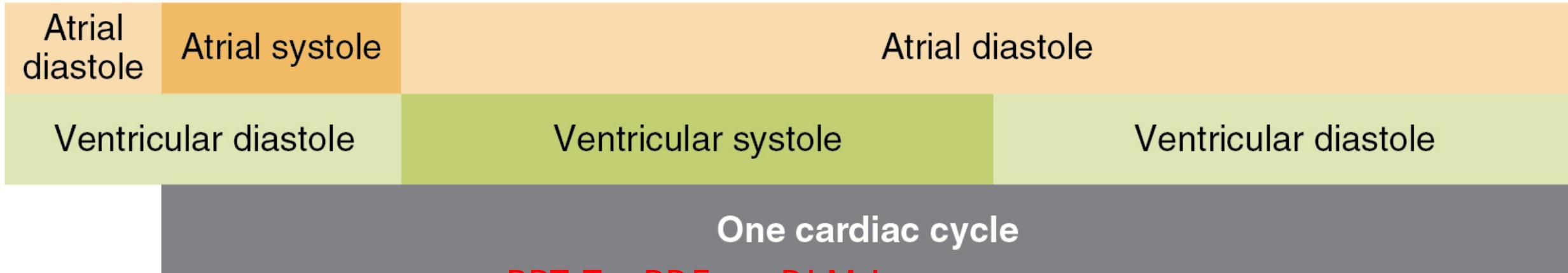
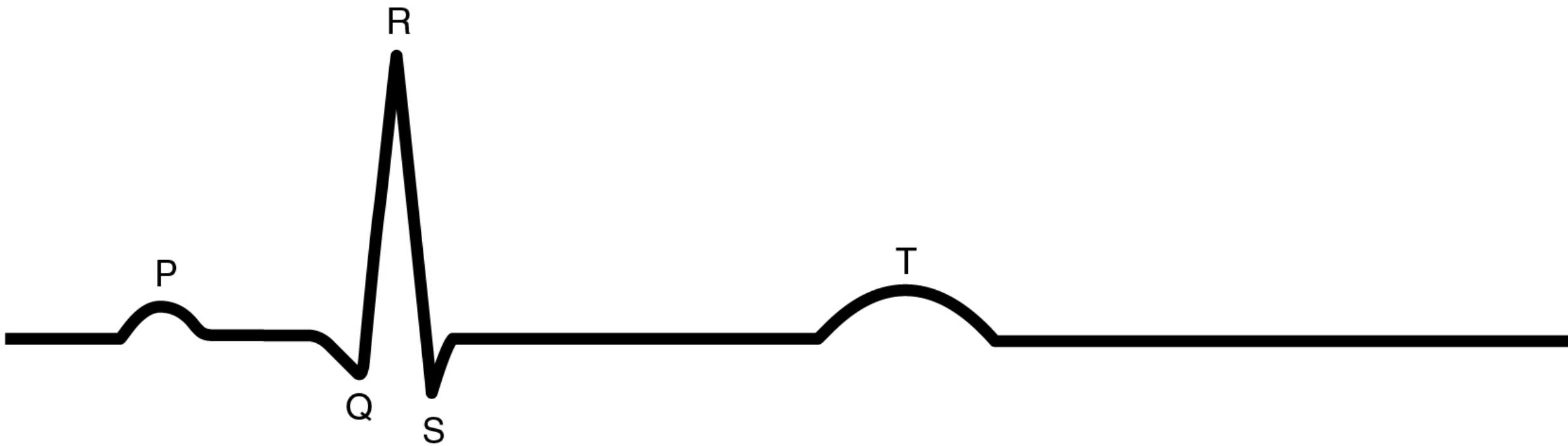
LEAD	POSITION
V1	OVER THE 4 TH INTERCOSTAL SPACE (4ICS) NEAR RIGHT STERNAL MARGIN
V2	4ICS NEAR LEFT STERNAL MARGIN
V3	BETWEEN V2 AND V4
V4	5 TH INTERCOSTAL SPACE MIDCLAVICULAR LINE (5ICSMCL)
V5	5 TH INTERCOSTAL SPACE ANTERIOR AXILLARY LINE (5ICSAAL)
V6	5 TH INTERCOSTAL SPACE MID AXILLARY LINE (5ICSMAL)

WAVES OF THE NORMAL ECG

- A NORMAL ECG CONSISTS OF WAVES, COMPLEXES, INTERVALS AND SEGMENTS
- WAVES OF ECG RECORDED BY LIMB LEAD 11 ARE CONSIDERED AS THE TYPICAL WAVES
- THE NORMAL ELECTROCARDIOGRAM IS COMPOSED OF A P WAVE, A QRS COMPLEX, AND A T WAVE.

WAVES OF NORMAL ECG

WAVE/SEGMENT	FROM-To	CAUSE	DURATION(S)	AMPLITUDE (mV)
P WAVE		ATRIAL DEPOLARIZATION	0.1	0.1-0.12
QRS	ONSET OF Q WAVE TO THE END OF S WAVE	VENTRICULAR DEPOLARIZATION	0.08-0.10	Q=0.1-0.2 R=1 S=0.4
T		VENTRICULAR REPOLARIZATION	0.2	0.3
P-R INTERVAL	ONSET OF P WAVE TO ONSET OF Q WAVE	ATRIAL DEPOLARIZATION AND CONDUCTION THROUGH AV NODE	0.18-(0.12-0.2)	
Q-T INTERVAL	ONSET OF Q WAVE AND END OF T WAVE	ELECTRICAL ACTIVITY IN VENTRICLES. INDICATES VENTRICULAR DEPOLARIZATION AND VENTRICULAR REPOLARIZATION	0.4-0.42	



P-WAVE

- P -WAVE IS POSITIVE (UPRIGHT) IN LEADS I,II,AVF, V4, V5, AND V6 AND NEGATIVE (INVERTED) IN AVR AND VARIABLE IN OTHER LEADS

CLINICAL SIGNIFICANCE

RIGHT ATRIAL HYPERTROPHY	TALL P WAVE (>2.5MM) IN LEAD II
LEFT ATRIAL HYPERTROPHY	- TALL AND BROAD P WAVE (M SHAPE)
HYPERKALAEMIA	ABSENT OR SMALL P WAVE
ATRIAL FIBRILLATION	ABSENT P WAVE
SINOATRIAL BLOCK	INVERTED OR ABSENT P WAVE

QRS COMPLEX

- ALSO CALLED INITIAL VENTRICULAR COMPLEX. IT IS USUALLY AS A RESULT OF DEPOLARIZATION OF VENTRICULAR MUSCULATURE
- Q WAVE DUE TO DEPOLARIZATION OF BASAL PORTION OF INTERVENTRICULAR SEPTUM
- R WAVE DUE TO DEPOLARIZATION OF APICAL PORTION OF INTERVENTRICULAR SEPTUM
- S WAVE DUE TO DEPOLARIZATION OF BASAL PORTION OF VENTRICULAR MUSCLE NEAR THE ATRIOVENTRICULAR RING

CLINICAL SIGNIFICANCE OF QRS WAVES

- BUNDLE BRANCH BLOCK: QRS IS PROLONGED OR DEFORMED
- HYPERKALAEMIA: QRS PROLONGED

T WAVE

- FINAL VENTRICULAR WAVE, USUALLY DUE TO VENTRICULAR REPOLARIZATION
- USUALLY POSITIVE IN LEADS I, II, AND V5 & V6. IT IS NORMALLY INVERTED IN AVR AND VARIABLE IN OTHER LEADS.

CLINICAL SIGNIFICANCE

- ACUTE MYOCARDIAL ISCHEMIA; TALL, BROAD BASED T WAVE (HYPERACUTE T WAVE)
- OLD AGE, HYPERVENTILATION, ANXIETY, MYOCARDIAL INFARCTION, L VENTRICULAR HYPERTROPHY AND PERICARDITIS- T WAVE IS SMALL, FLAT OR INVERTED.
- HYPOKALAEMIA: T WAVE IS SMALL, FLAT OR INVERTED.
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U- WAVE

- NOT ALWAYS SEEN
- INSIGNIFICANT BUT REPRESENTS REPOLARIZATION OF PAPILLARY MUSCLE

CLINICAL USES OF ECG

- TO OBTAIN INFORMATION ABOUT THE STRUCTURE AND FUNCTION OF THE HEART.
- TO OBTAIN INFORMATION ABOUT THE FUNCTION OF ITS ELECTRICAL CONDUCTION SYSTEM.
- TO MEASURE THE RATE AND RHYTHM OF HEARTBEATS, THE SIZE AND POSITION OF THE HEART CHAMBERS.
- TO ASSESS THE PRESENCE OF ANY DAMAGE TO THE HEART'S MUSCLE CELLS OR CONDUCTION SYSTEM.
- TO ASSESS THE EFFECTS OF DRUGS TO THE HEART.
- TO ASSESS THE FUNCTION OF IMPLANTED PACEMAKERS.

INDICATIONS FOR PERFORMING ECG

1. SUSPECTED MYOCARDIAL INFARCTION(HEART ATTACK) OR CHEST PAIN.
2. SUSPECTED PULMONARY EMBOLISM OR SHORTNESS OF BREATH.
3. PERCEIVED ARRHYTHMIA.
4. FAINTING OR COLLAPSE
5. SEIZURES
6. MONITORING THE EFFECTS OF MEDICATION ON THE HEART.
7. ASSESSING THE SEVERITY OF ELECTROLYTE ABNORMALITIES EG HYPERKALAEMIA.
8. HYPERTROPHIC CARDIOMYOPATHY (E.G DUE TO SPORTS)
9. PERIOPERATIVE ASSESSMENTS.
10. CARDIAC STRESS TESTING.

CARDIAC OUTPUT

- CARDIAC OUTPUT (CO) IS THE AMOUNT OF BLOOD PUMPED FROM EACH VENTRICLE. USUALLY, IT REFERS TO LEFT VENTRICULAR OUTPUT THROUGH AORTA. CARDIAC OUTPUT IS THE MOST IMPORTANT FACTOR IN CARDIOVASCULAR SYSTEM, BECAUSE RATE OF BLOOD FLOW THROUGH DIFFERENT PARTS OF THE BODY DEPENDS UPON CARDIAC OUTPUT.

USUALLY, CARDIAC OUTPUT IS EXPRESSED IN THREE WAYS:

1. STROKE VOLUME
2. MINUTE VOLUME
3. CARDIAC INDEX.

- HOWEVER, IN ROUTINE CLINICAL PRACTICE, CARDIAC OUTPUT REFERS TO MINUTE VOLUME.

DEFINITION OF TERMS

STROKE VOLUME

- STROKE VOLUME IS THE AMOUNT OF BLOOD PUMPED OUT BY EACH VENTRICLE DURING EACH BEAT. NORMAL VALUE: 70 mL (60 TO 80 mL) WHEN THE HEART RATE IS NORMAL (72/MINUTE).

MINUTE VOLUME

- MINUTE VOLUME IS THE AMOUNT OF BLOOD PUMPED OUT BY EACH VENTRICLE IN ONE MINUTE. IT IS THE PRODUCT OF STROKE VOLUME AND HEART RATE:

MINUTE VOLUME = STROKE VOLUME × HEART RATE

NORMAL VALUE: 5 L/VENTRICLE/MINUTE

CARDIAC INDEX

- CARDIAC INDEX IS THE MINUTE VOLUME EXPRESSED IN RELATION TO SQUARE METER OF BODY SURFACE AREA. IT IS DEFINED AS THE AMOUNT OF BLOOD PUMPED OUT PER VENTRICLE/MINUTE/ SQUARE METER OF THE BODY SURFACE AREA. NORMAL VALUE: 2.8 ± 0.3 L/SQUARE METER OF BODY SURFACE AREA/MINUTE (IN AN ADULT WITH AVERAGE BODY SURFACE AREA OF 1.734 SQUARE METER AND NORMAL MINUTE VOLUME OF 5 L/MINUTE).

EJECTION FRACTION

- EJECTION FRACTION IS THE FRACTION OF END DIASTOLIC VOLUME THAT IS EJECTED OUT BY EACH VENTRICLE. NORMAL EJECTION FRACTION IS 60% TO 65%.

CARDIAC RESERVE

- CARDIAC RESERVE IS THE MAXIMUM AMOUNT OF BLOOD THAT CAN BE PUMPED OUT BY HEART ABOVE THE NORMAL VALUE. CARDIAC RESERVE PLAYS AN IMPORTANT ROLE IN INCREASING THE CARDIAC OUTPUT DURING THE CONDITIONS LIKE EXERCISE. IT IS ESSENTIAL TO WITHSTAND THE STRESS OF EXERCISE. CARDIAC RESERVE IS USUALLY EXPRESSED IN PERCENTAGE. IN A NORMAL YOUNG HEALTHY ADULT, THE CARDIAC RESERVE IS 300% TO 400%. IN OLD AGE, IT IS ABOUT 200% TO 250%. IT INCREASES TO 500% TO 600% IN ATHLETES. IN CARDIAC DISEASES, THE CARDIAC RESERVE IS MINIMUM OR NIL.

VARIATIONS IN CARDIAC OUTPUT

PHYSIOLOGICAL VARIATIONS

1. AGE: IN CHILDREN, CARDIAC OUTPUT IS LESS BECAUSE OF LESS BLOOD VOLUME. CARDIAC INDEX IS MORE THAN THAT IN ADULTS BECAUSE OF LESS BODY SURFACE AREA.
2. SEX: IN FEMALES, CARDIAC OUTPUT IS LESS THAN IN MALES BECAUSE OF LESS BLOOD VOLUME. CARDIAC INDEX IS MORE THAN IN MALES, BECAUSE OF LESS BODY SURFACE AREA.
3. BODY BUILD: GREATER THE BODY BUILD, MORE IS THE CARDIAC OUTPUT.
4. DIURNAL VARIATION: CARDIAC OUTPUT IS LOW IN EARLY MORNING AND INCREASES IN DAY TIME. IT DEPENDS UPON THE BASAL CONDITIONS OF THE INDIVIDUALS.

5. ENVIRONMENTAL TEMPERATURE: MODERATE CHANGE IN TEMPERATURE DOES NOT AFFECT CARDIAC OUTPUT. INCREASE IN TEMPERATURE ABOVE 30°C RAISES CARDIAC OUTPUT.

6. EMOTIONAL CONDITIONS: ANXIETY, APPREHENSION AND EXCITEMENT INCREASES CARDIAC OUTPUT ABOUT 50% TO 100% THROUGH THE RELEASE OF CATECHOLAMINES, WHICH INCREASE THE HEART RATE AND FORCE OF CONTRACTION.

7. AFTER MEALS: DURING THE FIRST ONE HOUR AFTER TAKING MEALS, CARDIAC OUTPUT INCREASES.

8. EXERCISE: CARDIAC OUTPUT INCREASES DURING EXERCISE BECAUSE OF INCREASE IN HEART RATE AND FORCE OF CONTRACTION.

9. HIGH ALTITUDE: IN HIGH ALTITUDE, THE CARDIAC OUTPUT INCREASES BECAUSE OF INCREASE IN SECRETION OF ADRENALINE. ADRENALINE SECRETION IS STIMULATED BY HYPOXIA (LACK OF OXYGEN).

10. POSTURE: WHILE CHANGING FROM RECUMBENT TO UPRIGHT POSITION, THE CARDIAC OUTPUT DECREASES.

11. PREGNANCY: DURING PREGNANCY, CARDIAC OUTPUT INCREASES BY 40%.

PATHOLOGICAL VARIATIONS

INCREASE IN CARDIAC OUTPUT

1. FEVER: DUE TO INCREASED OXIDATIVE PROCESSES
2. ANEMIA: DUE TO HYPOXIA
3. HYPERTHYROIDISM: DUE TO INCREASED BASAL METABOLIC RATE.

DECREASE IN CARDIAC OUTPUT

1. HYPOTHYROIDISM: DUE TO DECREASED BASAL METABOLIC RATE
2. ATRIAL FIBRILLATION: BECAUSE OF INCOMPLETE FILLING OF VENTRICLES
3. INCOMPLETE HEART BLOCK WITH CORONARY SCLEROSIS OR MYOCARDIAL DEGENERATION: DUE TO DEFECTIVE PUMPING ACTION OF THE HEART
4. CONGESTIVE CARDIAC FAILURE: BECAUSE OF WEAK CONTRACTIONS OF HEART
5. SHOCK: DUE TO POOR PUMPING AND CIRCULATION

DISTRIBUTION OF CARDIAC OUTPUT

- THE WHOLE AMOUNT OF BLOOD PUMPED OUT BY THE RIGHT VENTRICLE GOES TO LUNGS.
- THE BLOOD PUMPED BY THE LEFT VENTRICLE IS DISTRIBUTED TO DIFFERENT PARTS OF THE BODY.
- FRACTION OF CARDIAC OUTPUT DISTRIBUTED TO A PARTICULAR REGION OR ORGAN DEPENDS UPON THE METABOLIC ACTIVITIES OF THAT REGION OR ORGAN.

DISTRIBUTION OF BLOOD PUMPED OUT OF LEFT VENTRICLE

ORGAN	AMOUNT OF BLOOD (ML/ MINUTE)	PERCENTAGE
LIVER	1,500	30
KIDNEY	1,300	26
SKELETAL MUSCLES	900	18
BRAIN	800	16
SKIN, BONE AND GI TRACT	300	6
HEART	200	4
TOTAL	5000	100

FACTORS MAINTAINING CARDIAC OUTPUT

CARDIAC OUTPUT IS MAINTAINED (DETERMINED) BY FOUR FACTORS:

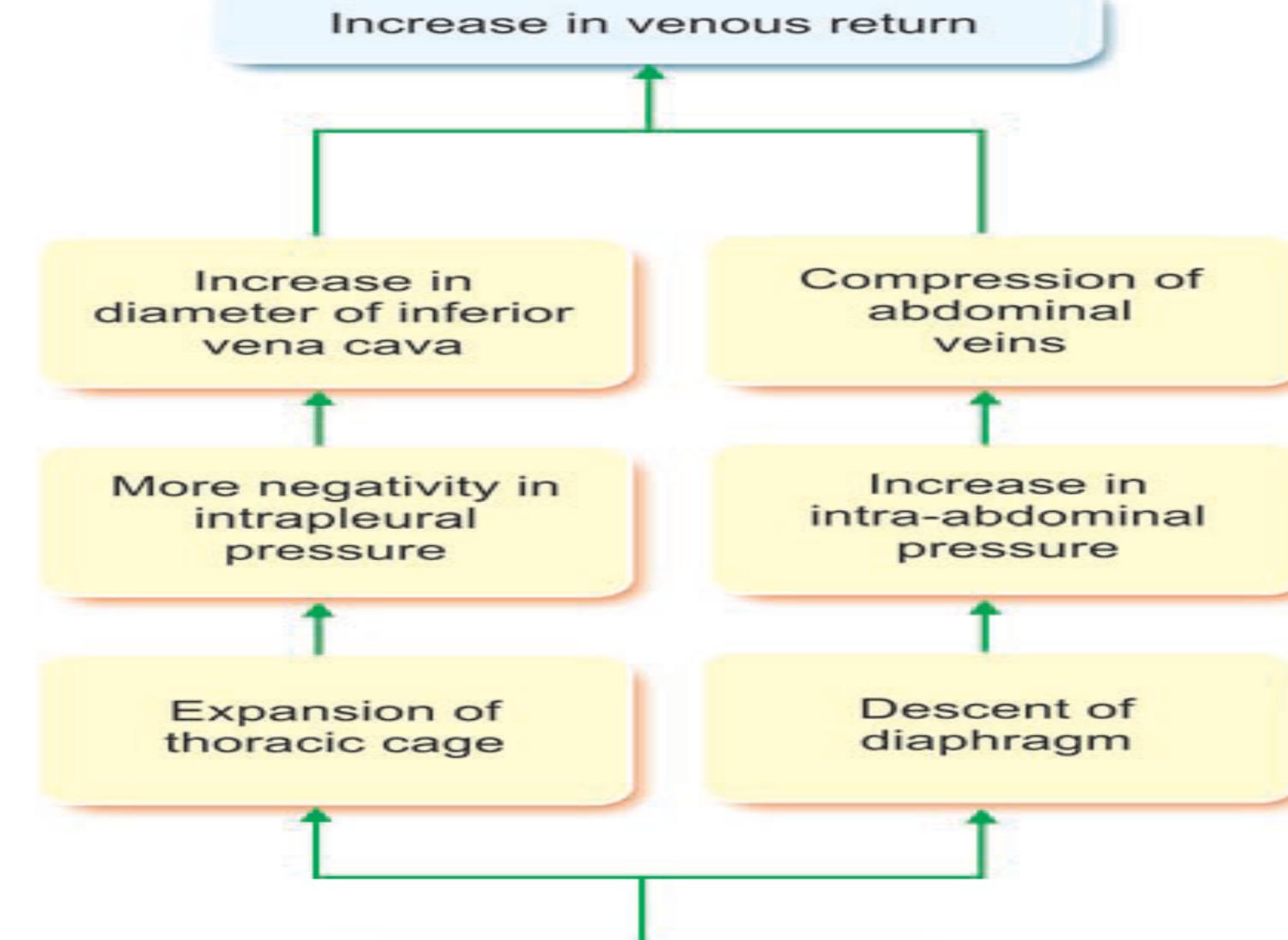
1. VENOUS RETURN
2. FORCE OF CONTRACTION
3. HEART RATE
4. PERIPHERAL RESISTANCE

1. VENOUS RETURN

- VENOUS RETURN IS THE AMOUNT OF BLOOD WHICH IS RETURNED TO HEART FROM DIFFERENT PARTS OF THE BODY. WHEN IT INCREASES, THE VENTRICULAR FILLING AND CARDIAC OUTPUT ARE INCREASED. **THUS, CARDIAC OUTPUT IS DIRECTLY PROPORTIONAL TO VENOUS RETURN, PROVIDED THE OTHER FACTORS (FORCE OF CONTRACTION, HEART RATE AND PERIPHERAL RESISTANCE) REMAIN CONSTANT.**
- VENOUS RETURN DEPENDS UPON FIVE FACTORS:
 - I. RESPIRATORY PUMP
 - II. MUSCLE PUMP
 - III. GRAVITY
 - IV. VENOUS PRESSURE
 - V. SYMPATHETIC TONE.

RESPIRATORY PUMP

- RESPIRATORY PUMP IS THE RESPIRATORY ACTIVITY THAT HELPS THE RETURN OF BLOOD, TO HEART DURING INSPIRATION. IT IS ALSO CALLED **ABDOMINOTHORACIC PUMP.**



MUSCLE PUMP

- MUSCLE PUMP IS THE MUSCULAR ACTIVITY THAT HELPS IN RETURN OF THE BLOOD TO HEART. DURING MUSCULAR ACTIVITIES, THE VEINS ARE COMPRESSED OR SQUEEZED. DUE TO THE PRESENCE OF VALVES IN VEINS, DURING COMPRESSION THE BLOOD IS MOVED TOWARDS THE HEART. WHEN MUSCULAR ACTIVITY INCREASES, THE VENOUS RETURN IS MORE. WHEN THE SKELETAL MUSCLES CONTRACT, THE VEIN LOCATED IN BETWEEN THE MUSCLES IS COMPRESSED.

GRAVITY

- GRAVITATIONAL FORCE REDUCES THE VENOUS RETURN. WHEN A PERSON STANDS FOR A LONG PERIOD, GRAVITY CAUSES POOLING OF BLOOD IN THE LEGS, WHICH IS CALLED VENOUS POOLING. BECAUSE OF VENOUS POOLING, THE AMOUNT OF BLOOD RETURNING TO HEART DECREASES.

VENOUS PRESSURE

- VENOUS PRESSURE ALSO AFFECTS THE VENOUS RETURN. PRESSURE IN THE VENULES IS 12 TO 18 MM HG. IN THE SMALLER AND LARGER VEINS, THE PRESSURE GRADUALLY DECREASES. IN THE GREAT VEINS, I.E. INFERIOR VENA CAVA AND SUPERIOR VENA CAVA, THE PRESSURE FALLS TO ABOUT 5.5 MM HG. AT THE JUNCTION OF VENAE CAVAE AND RIGHT ATRIUM, IT IS ABOUT 4.6MM HG. PRESSURE IN THE RIGHT ATRIUM IS STILL LOW AND IT ALTERS DURING CARDIAC ACTION. IT FALLS TO ZERO DURING ATRIAL DIASTOLE. THIS PRESSURE GRADIENT AT EVERY PART OF VENOUS TREE HELPS AS A DRIVING FORCE FOR VENOUS RETURN

SYMPATHETIC TONE

- VENOUS RETURN IS AIDED BY SYMPATHETIC OR VASOMOTOR TONE WHICH CAUSES CONSTRICTION OF VENULES. VENOUS CONSTRICKTION PUSHES THE BLOOD TOWARDS HEART

2. FORCE OF CONTRACTION

CARDIAC OUTPUT IS DIRECTLY PROPORTIONAL TO THE FORCE OF CONTRACTION, PROVIDED THE OTHER THREE FACTORS REMAIN CONSTANT. ACCORDING TO FRANK-STARLING LAW, FORCE OF CONTRACTION OF HEART IS DIRECTLY PROPORTIONAL TO THE INITIAL LENGTH OF MUSCLE FIBERS, BEFORE THE ONSET OF CONTRACTION. FORCE OF CONTRACTION DEPENDS UPON PRELOAD AND AFTERLOAD.

PRELOAD

- PRELOAD IS THE STRETCHING OF THE CARDIAC MUSCLE FIBERS AT THE END OF DIASTOLE, JUST BEFORE CONTRACTION. IT IS DUE TO INCREASE IN VENTRICULAR PRESSURE CAUSED BY FILLING OF BLOOD DURING DIASTOLE. STRETCHING OF MUSCLE FIBERS INCREASES THEIR LENGTH, WHICH INCREASES THE FORCE OF CONTRACTION AND CARDIAC OUTPUT. FORCE OF CONTRACTION OF HEART AND CARDIAC OUTPUT ARE DIRECTLY PROPORTIONAL TO PRELOAD

AFTERLOAD

- AFTERLOAD IS THE FORCE AGAINST WHICH VENTRICLES MUST CONTRACT AND EJECT THE BLOOD. FORCE IS DETERMINED BY THE ARTERIAL PRESSURE. AT THE END OF ISOMETRIC CONTRACTION PERIOD, SEMILUNAR VALVES ARE OPENED AND BLOOD IS EJECTED INTO THE AORTA AND PULMONARY ARTERY. SO, THE PRESSURE INCREASES IN THESE TWO VESSELS. NOW, THE VENTRICLES HAVE TO WORK AGAINST THIS PRESSURE FOR FURTHER EJECTION. THUS, THE AFTERLOAD FOR LEFT VENTRICLE IS DETERMINED BY AORTIC PRESSURE AND AFTERLOAD FOR RIGHT VENTRICULAR PRESSURE IS DETERMINED BY PRESSURE IN PULMONARY ARTERY.
- FORCE OF CONTRACTION OF HEART AND CARDIAC OUTPUT ARE INVERSELY PROPORTIONAL TO AFTERLOAD.

3. HEART RATE

- CARDIAC OUTPUT IS DIRECTLY PROPORTIONAL TO HEART RATE PROVIDED, THE OTHER THREE FACTORS REMAIN CONSTANT. MODERATE CHANGE IN HEART RATE DOES NOT ALTER THE CARDIAC OUTPUT. IF THERE IS A MARKED INCREASE IN HEART RATE, CARDIAC OUTPUT IS INCREASED. IF THERE IS MARKED DECREASE IN HEART RATE, CARDIAC OUTPUT IS DECREASED.

4. PERIPHERAL RESISTANCE

- PERIPHERAL RESISTANCE IS THE RESISTANCE OFFERED TO BLOOD FLOW AT THE PERIPHERAL BLOOD VESSELS. PERIPHERAL RESISTANCE IS THE RESISTANCE OR LOAD AGAINST WHICH THE HEART HAS TO PUMP THE BLOOD. So. THE CARDIAC OUTPUT IS INVERSELY

MEASUREMENT OF CARDIAC OUTPUT

CARDIAC OUTPUT CAN BE MEASURED THROUGH
DIRECT METHODS USED TO MEASURE CARDIAC OUTPUT IN
ANIMALS:

1. BY USING CARDIOMETER
2. BY USING FLOWMETER.

INDIRECT METHODS USED TO MEASURE CARDIAC OUTPUT
(USED FOR ANIMALS AND HUMANS):

1. BY USING FICK PRINCIPLE
2. INDICATOR (DYE) DILUTION TECHNIQUE
3. THERMODILUTION TECHNIQUE
4. ULTRASONIC DOPPLER TRANSDUCER TECHNIQUE
5. DOPPLER ECHOCARDIOGRAPHY
6. BALLISTOCARDIOGRAPHY.

- ADOLPH FICK DESCRIBED FICK PRINCIPLE IN 1870. ACCORDING TO THIS PRINCIPLE, THE AMOUNT OF A SUBSTANCE TAKEN UP BY AN ORGAN (OR BY THE WHOLE BODY) OR GIVEN OUT IN A UNIT OF TIME IS THE PRODUCT OF AMOUNT OF BLOOD FLOWING THROUGH THE ORGAN AND THE ARTERIOVENOUS DIFFERENCE OF THE SUBSTANCE ACROSS THE ORGAN.

AMOUNT OF SUBSTANCE TAKEN OR GIVEN = AMOUNT OF BLOOD FLOW /MINUTE × ARTERIOVENOUS DIFFERENCE

E.G. AMOUNT OF BLOOD FLOWING THROUGH LUNGS IS 5,000 mL/MINUTE

O₂ CONTENT IN ARTERIAL BLOOD = 20 mL/100 mL OF BLOOD

O₂ CONTENT IN VENOUS BLOOD = 15 mL/100 mL OF BLOOD

AMOUNT OF O₂ MOVED FROM LUNGS TO BLOOD = AMOUNT OF BLOOD FLOW /MINUTE
X ARTERIOVENOUS DIFFERENCE OF O₂

i.e. 5,000 × 20-15/ 100 = 250 mL/MINUTE

• AMOUNT OF OXYGEN MOVED FROM LUNGS TO BLOOD = 250 mL/MINUTE

MODIFICATION OF FICK PRINCIPLE TO MEASURE CARDIAC OUTPUT

- FICK PRINCIPLE IS MODIFIED TO MEASURE THE CARDIAC OUTPUT OR A PART OF CARDIAC OUTPUT (AMOUNT OF BLOOD TO AN ORGAN). THUS, CARDIAC OUTPUT OR THE AMOUNT OF BLOOD FLOWING THROUGH AN ORGAN IN A GIVEN UNIT OF TIME IS DETERMINED BY THE FORMULA:

CARDIAC OUTPUT= $\frac{\text{AMOUNT OF SUBSTANCE TAKEN OR GIVEN BY THE ORGAN/ MINUTE}}{\text{ARTERIOVENOUS DIFFERENCE OF THE SUBSTANCE ACROSS THE ORGAN}}$

BY MODIFYING FICK PRINCIPLE, CARDIAC OUTPUT IS MEASURED IN TWO WAYS:

- I. BY USING OXYGEN CONSUMPTION
- II. BY USING CARBON DIOXIDE GIVEN OUT.

MEASUREMENT OF CARDIAC OUTPUT BY USING OXYGEN CONSUMPTION

- FICK PRINCIPLE IS USED TO MEASURE THE CARDIAC OUTPUT BY DETERMINING THE AMOUNT OF OXYGEN CONSUMED IN THE BODY IN A GIVEN PERIOD OF TIME AND DIVIDING THIS VALUE BY THE ARTERIOVENOUS DIFFERENCE ACROSS THE LUNGS.

- CARDIAC OUTPUT = O₂ CONSUMED (IN mL/MINUTE)
ARTERIOVENOUS O₂ DIFFERENCE
- *OXYGEN CONSUMPTION*: AMOUNT OF OXYGEN CONSUMED IS MEASURED BY USING A RESPIROMETER OR BMR APPARATUS (BENEDICT ROTH APPARATUS).
- *OXYGEN CONTENT IN ARTERIAL BLOOD*: BLOOD IS COLLECTED FROM ANY ARTERY TO DETERMINE THE OXYGEN CONTENT IN ARTERIAL BLOOD. OXYGEN CONTENT IS DETERMINED BY BLOOD GAS ANALYSIS.

- *OXYGEN CONTENT IN VENOUS BLOOD:* ONLY MIXED VENOUS BLOOD IS USED TO DETERMINE THE OXYGEN CONTENT OF VENOUS BLOOD, SINCE OXYGEN CONTENT IS DIFFERENT IN DIFFERENT VEINS. MIXED VENOUS BLOOD IS COLLECTED FROM RIGHT ATRIUMOR PULMONARY ARTERY. IT IS DONE BY INTRODUCING A **CATHETER**THROUGH BASILAR VEIN OF FOREARM. OXYGEN IS DETERMINED FROM THIS BLOOD BY **BLOOD GAS ANALYSIS**

- *CALCULATION*
- FOR EXAMPLE, IN A SUBJECT, THE FOLLOWING DATA ARE
- OBTAINED:

O₂ CONSUMED (BY LUNGS) = 250 mL/MINUTE

O₂ CONTENT IN ARTERIAL BLOOD = 20 mL/100 mL OF BLOOD

O₂ CONTENT IN VENOUS BLOOD = 15 mL/100 mL OF BLOOD

CARDIAC OUTPUT = O₂ CONSUMED (IN mL/MINUTE)

ARTERIOVENOUS O₂ DIFFERENCE

$$\text{CO} = \frac{250}{5/100} = \frac{250 \times 100}{5}$$
$$= 5,000 \text{ML/MIN}$$

5 mL OF OXYGEN IS TAKEN BY 100 mL OF BLOOD WHILE PASSING THROUGH THE LUNGS. THUS, 250 mL OF OXYGEN IS TAKEN BY 5,000 mL OF BLOOD. SINCE CARDIAC OUTPUT IS EQUIVALENT TO THE AMOUNT OF BLOOD PASSING THROUGH PULMONARY CIRCULATION, THE CARDIAC OUTPUT = 5 L/MINUTE.

MEASUREMENT OF CARDIAC OUTPUT BY USING CARBON DIOXIDE

- CARDIAC OUTPUT IS ALSO MEASURED BY KNOWING THE ARTERIOVENOUS DIFFERENCE OF CARBON DIOXIDE AND AMOUNT OF CARBON DIOXIDE GIVEN OUT (REMOVED) BY LUNGS

$$\text{CARDIAC OUTPUT} = \frac{\text{CO}_2 \text{ EVOLVED (IN mL/MINUTE)}}{\text{ARTERIOVENOUS CO}_2 \text{ DIFFERENCE}}$$

CALCULATION

FOR EXAMPLE, IN A SUBJECT CO₂ REMOVED BY LUNGS = 200 mL/MINUTE

- CO₂ CONTENT IN ARTERIAL BLOOD = 56 mL/100 mL OF BLOOD
- CO₂ CONTENT IN VENOUS BLOOD = 60 mL/100 mL OF BLOOD

200

60-56 mL/100mL

200 x 100 = 5,000mL = 5L/MIN.

- SINCE CARDIAC OUTPUT IS EQUAL TO THE AMOUNT OF BLOOD PASSING THROUGH LUNGS (PULMONARY CIRCULATION), THE CARDIAC OUTPUT = 5 L/MINUTE
- NITROUS OXIDE IS ALSO USED TO MEASURE CARDIAC OUTPUT BY APPLYING FICK PRINCIPLE.

ADVANTAGE OF MEASUREMENT OF CARDIAC OUTPUT BY FICK PRINCIPLE

- THE RESULTS ARE ACCURATE.

DISADVANTAGE

- FICK PRINCIPLE IS AN INVASIVE METHOD AND INVOLVES THE INSERTION OF CATHETER THROUGH SUBJECT VEIN.