CARDIAC OUTPUT AND ITS REGULATION

DR. ARWA RAWASHDEH

OBJECTIVES

- **DEFINE CARDIAC OUTPUT**

HEART RATE

STROKE VOLUME

- **REGULATOR OF CARDIAC OUTPUT**

1.POSITIVE AND NEGATIVE FACTORS AFFECTING HEART RATE (CHRONOTROPIC ACTION)

2. POSITIVE AND NEGATIVE FACTORS AFFECTING STROKE VOLUME (INOTROPIC ACTION)

CARDIAC OUTPUT

CARDIAC OUTPUT= HEART RATE X STROKE VOLUME

- **HR= BEATS PER MINUTE BEAT /MIN**

- **SV= TOTAL VOLUME OF BLOOD THAT THE ACTUAL VENTRICLES ARE EJECTING FOR EVERY ONE BEAT**

  SV= END DIASTOLIC (ED)- END SYSTOLIC (ES)ML/BEAT

- **CO= ML/MIN**

  CO= 70BPM X 70ML/BEAT= 5000ML/MIN=5L/M

HEART RATE

- AVERAGE IS 60-80BPM
- SA NODE IS THE GENERATION OF SINUS RHTHYM

HOW WE CAN FLUCTUATE THE HEART RATE?

POSITIVE CHRONOTROPIC ACTION ; INCREASE THE RATE OF THE HEART

1.PRODUCTION OF SYMPATHETIC NERVOUS SYSTEM NEUROTRANSMITTERS; NOREPINEPHRINE (NE) ACTING ON BETA-1ADRENERGIC RECEPTORS

2. EPINEPHRINE ON BETA-1 ADRENERGIC RECEPTORS

3. THYROID HORMONES T3 AND T4 VERY POWERFUL REGULATOR ; INCREASE THE HEART RATE

4. INCREASE THE BODY TEMPERATURE; INCREASE THE METABOLIC RATE

5. CALCIUM RATE; HYPERCALCEMIA MORE CALCIUM IN THE CELL MORE DEPOLARIZATION; DESPITE CONFLICTING EVIDENCES OF THAT

6. LOW POTASSIUM CAN CAUSE ARRHYTHMIAS

7. TO SOME EXTENT STIMULATION OF PERIPHERAL CHEMORECEPTORS DUE TO HYPOXIA, INCREASE IN THE CO2, DECREASE IN THE PH.

CONTINUED HEART RATE

NEGATIVE CHRONOTROPIC ACTION; DECREASE THE HEART RATE

1. ACETYLCHOLINE PART OF PARASYMPATHETIC NERVOUS SYSTEM ACTING ON MUSCARINIC TYPE 2 RECEPTORS
2. LOW CALCIUM EFFECT
3. HYPERKALEMIA; HIGH POTASSIUM LEVELS IN THE BLOOD ; LESS POTASSIUM WOULD MOVE OUTSIDE THE CELL AND THIS VERY DANGEROUS BECAUSE IT IS GOING TO CAUSE CARDIAC ARREST ; AFFECT HEART FROM BEING ABLE TO SEND ACTION POTENTIALS AND CAUSE A PERSON TO GO INTO CARDIAC ARREST

AGE

- FETUS: 120-140 BPM
- ADULTS: 60- 80 BPM
- MALE: 64-72BPM
- FEMALE: 72-80BPM
- AS YOU GETTING OLDER THE HEART RATE INCREASES

CONTINUED HEART RATE

ATRIAL BAINBRIDGE REFLEX (ATRIAL REFLEX); REGULATOR OF HEART RATE

INCREASE IN THE VENOUS RETURN CAUSES INCREASE IN THE STRETCH AND STIMULATE CARDIAC ACCELERATOR CENTER IN THE BRAIN AND GO TO SA NODE VIA SYMPATHETIC AND PARASYMPATHETIC PATHWAY AND INCREASE THE HEART RATE

BRADYCARDIA

IS BASICALLY DEFINED AS WHENEVER THE HEART RATE IS LESS THAN 60 BPM

- PHYSIOLOGICALLY COULD BE DUE TO ENDURANCE RUNNERS WHO RUN EXCESSIVELY THEY PUT THEIR HEART THROUGH LOT OF WORK
- PARASYMPATHETIC ACTIVITY

TACHYCARDIAC

IS BASICALLY DEFINED AS THE HEART RATE IS MORE THAN 100BPM

- INCREASE SYMPATHETIC NERVOUS ACTIVITY
- T3 AND T4
- ANXIETY

STROKE VOLUME

- **STROKE VOLUME = END DIASTOLIC (ED)- END SYSTOLIC (ES)ML/BEAT**

- **END DIASTOLIC VOLUME** :THE AMOUNT OF BLOOD THAT REMAINS IN THE VENTRICLE JUST BEFORE VENTRICULAR SYSTOLE IS THE EDV; 120ML

- **END SYSTOLIC VOLUME**: THE AMOUNT OF BLOOD THAT REMAINS IN THE VENTRICLE AT THE END OF VENTRICULAR SYSTOLE IS THE ESV; 50ML

- **SV**= 70ML / BEAT

CONTINUED STROKE VOLUME

- DIVIDED INTO THREE CATEGORIES:

A. PRELOAD ; THE DEGREE OF STRETCH OF CARDIAC MUSCLE ; HOW MUCH THE MYOCARDIUM STRETCHING WHEN IT IS GETTING FILLED WITH BLOOD

- **INCREASE EDV**

1. INCREASE VENOUS RETURN

2. INCREASE MUSCLE MILKING ACTIVITY DURING EXERCISING

3. RESPIRATORY PUMP WHENEVER YOU ARE BREATHING YOU INCREASE THE ABDOMINAL CAVITY PRESSURE , DECREASE THE THORACIC CAVITY PRESSURE AND SUCK BLOOD UP LIKE A VACUUM

4. VENOMOTOR TONE ; VENOCONSTRICTION CAUSED BY NOREPINEPHRINE RELEASED FROM SYMPATHETIC CAUSING VENOUS SMOOTH MUSCLE CONTRACTION AND SQUEEZING BLOOD UP

- **FILLING TIME**; GIVING THE HEART ADEQUATE TIME TO FILL WITH BLOOD

1. INCREASE THE HEART RATE WOULD DECREASE FILLING TIME AND DECREASE PRELOAD

CONTINUED STROKE VOLUME

- **MYOCARDIUM INFARCTION**

DECREASE STRETCHING MYOCARDIUM CONTRACTION BECAUSE THE HEART MUSCLES IS REPLACED WITH FIBROUS TISSUES SO THE STARCHING ABILITY IS REDUCED THEN THE PRELOAD IS REDUCED

- **FRANK STARLINGS LOW**

WHENEVER YOU HAVE AN INCREASE STRETCH OF THE HEART, IT ALLOWS FOR THIS LINK TENSION RELATIONSHIP MORE CROSS- BRIDGE TO BE ACTIVE SO WHENEVER STRETCHING OF THE HEART AND THERE IS OPTIMAL CROSS BRIDGE CONNECTIONS THAT INCREASES THE PRELOAD

GREATER STRETCH GREATER THE ACTUAL FORCE OF CONTRACTION

CONTINUED STROKE VOLUME

B. CONTRACTILITY

SYMPATHETIC NERVOUS SYSTEM

- EPINEPHRINE AND NOREPINEPHRINE ON BETA-1 ADRENERGIC RECEPTORS ; THEY INCREASE THE CALCIUM LEVEL OF THE CELL INCREASE THE ACTUAL CONTRACTILITY

HORMONES

- T3 AND T4 BINDS TO INTRANUCLEAR RECEPTORS AND STIMULATES THESE GENES CAN DO IS THEY CAN PRODUCE A BUNCH OF DIFFERENT TYPES OF PROTEINS ONE OF THE PROTEINS IS IT INCREASE THE EXPRESSION OF THOSE BETA-1 ADRENERGIC RECEPTORS; SO MORE RECEPTORS TO EPINEPHRINE AND NOREPINEPHRINE AND AMPLIFIED THE ACTION
- GLUCAGON

DRUGS

- DIGITALIS; SODIUM POTASSIUM ATP ASE INHIBITER INCREASE CALCIUM LEVEL
- DOPAMINE NEUROTRANSMITTER PRECURSOR OF NOREPINEPHRINE
- DOBUTAMINE ; BETA-1 ADRENERGIC RECEPTORS
- ATROPINE ; BLOCKS THE ACETYLCHOLINE ON THE M2 RECEPTORS

INHIBIT CONTRACTILITY

- BETA BLOCKERS
- CALCIUM BLOCKERS

CONTINUED STROKE VOLUME

IONS

- HYPERCALCEMIA +
- HYPOCALCEMIA _
- HYPERKALEMIA -
- HYPERNATREMIA –
- ACIDOSIS; HIGH PROTON -

POSITIVE INOTROPIC ACTION; AGENTS INCREASE THE CONTRACTION OF THE HEART

NEGATIVE INOTROPIC AGENT; AGENTS DECREASE THE CONTRACTION OF THE HEART

CONTINUED STROKE VOLUME

C. AFTERLOAD

THE AMOUNT OF RESISTANCE THAT MUST BE OVERCOME IN ORDER FOR THE VENTRICLES TO EJECT BLOOD INTO THE ACTUAL AORTA OR INTO THE PULMONARY TRUNK

THE PRESSURE IN THE VENTRICLES (SYSTOLIC PRESSURE)ABOUT 120MMHG AND THE PRESSURE IN THE AORTIC (DIASTOLIC PRESSURE) ABOUT 80MMHG ; IF I INCREASE THE PRESSURE OF ATRIUM TO 100MMHG . BEFORE THE DIFFERENCE WAS FROM 120 TO 80 BUT NOW THE DIFFERENCE IS 120 TO 100

THAT MEANS I WANT TO MOVE FROM HIGH PRESSURE TO A LITTLE HIGH PRESSURE IN NORMAL ; IF I INCREASE 80 TO 100 I WOULD INCREASE THE RESISTANCE THAT I HAVE TO OVERCOME TO PUSH BLOOD FROM VENTRICLES INTO THAT VESSELS THERE.

WHAT THINGS COULD CHANGE THE PRESSURE

1. PLAQUES OR OCCLUSION BUILD UP
2. STENOTIC
3. HYPERTENSIN DUE TO INCREASE VASCULAR RESISTANCE

OVERALL CONCEPT OF FACTORS AFFECTING STROKE VOLUME

- INCREASE IN AFTERLOAD WOULD DECREASE THE STROKE VOLUME
- WHEREASES INCREASE IN PRELOAD WOULD INCREASE THE STROKE VOLUME
- INCREASE IN CONTRACTILITY INCREASE IN STROKE VOLUME