PHYSIOLOGY TEAM 432

LECTURES : 8 + 9

Cardiac Output and Venous Return

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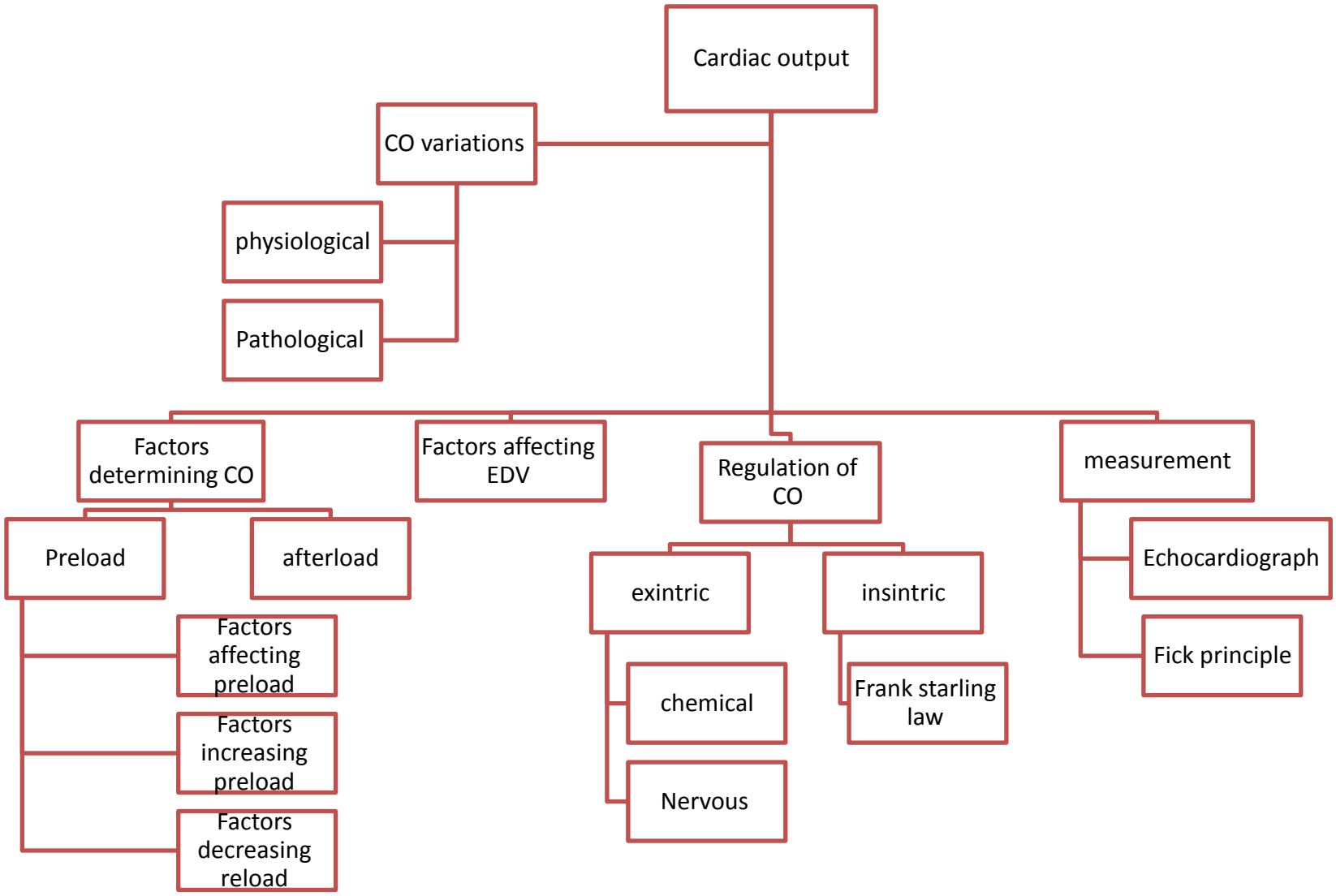
OBJECTIVES

Were not given

MIND MAP


Cardiac output
CO variations
physiological
Pathological
Factors determining CO
Factors affecting EDV
Regulation of CO
measurement
Preload
afterload
Factors affecting preload
Factors increasing preload
Factors decreasing reload
exintric
insintric
chemical
Nervous
Frank starling law
Echocardiograph
Fick principle

Definitions

- **Cardiac output (CO):**
  Amount of blood pumped by each ventricle per minute.
  Right CO = Left CO
- **Stroke volume (SV):** (Cardiac Output أكثر شيء يأثر بـ)
  Volume of blood ejected by each **ventricle** per **beat**
  SV x HR = CO (if SV increase CO increase)
  Stroke volume = ml/beat and
  heart rate = beats/minute
  cardiac output = ml/minute or expressed in liters/minute.
- **Cardiac Index (CI):** (more accurate)
  The index is usually calculated using the following formula:
  CI= CO/ BSA= (SV x HR)/BSA. (Body Surface Area)
- **End-diastolic volume (EDV):**
  Amount of blood remaining in the heart by the end of diastole =140 mL
  (increase in Bradycardia because of the time given to the heart to fill with blood)
- **End-systolic volume (ESV):**
  Amount of blood remaining in the heart by the end of systole =70 mL
- **SV = EDV-ESV**

Factor affecting CO

Physiological variations	
Increased by:	***Decreased by:***
Exercise (up to 700%)	Sitting or standing from lying position (20-30%)
Eating (30%) (All the GIT active and need blood splay)	
High environmental temperature	
Pregnancy (There is 2 circulations)	
Anxiety (50-100%)	
Sympathomimetic, epinephrine	

Pathological variations	
Fever	Hypothermia
Hyperthyroidism	Hypothyroidism
Anemia	Myocardial diseases e.g. infarction, failure
	Rapid arrhythmias

Factors determining CO

1- Preload (VR)

can be defined as the initial **stretching** of the cardiac myocytes prior to contraction. (if the EDV ↑ ➔ degree of stretching ↑ ➔ Force of contraction ↑ ➔ CO ↑)

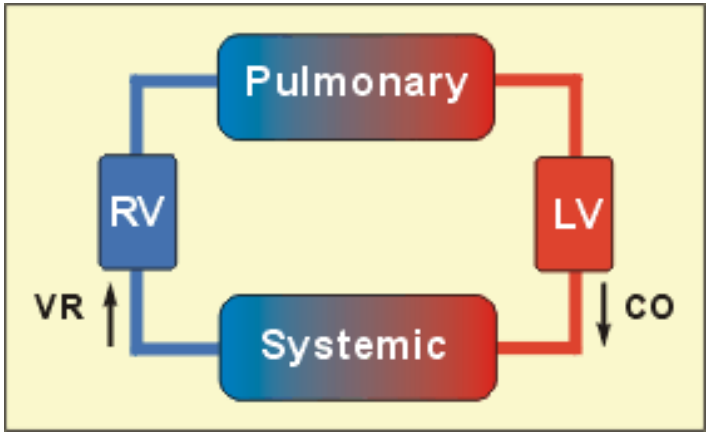
Venous Return (Preload)

- is the flow of blood back to the heart. Under steady-state conditions, venous return must **equal** cardiac output (CO) when averaged over time because the cardiovascular system is essentially a closed loop. Otherwise, blood would **accumulate** in either the systemic or pulmonary circulations. Although cardiac output and venous return are interdependent, each can be independently regulated .
- The concept of preload can be applied to either the ventricles or atria. Regardless of the chamber, the preload is related to the chamber **volume just prior to contraction**.

Factors determining CO

1- Preload (VR)

- Venous return (VR) to the heart from the venous vascular beds is determined by a **pressure gradient** (venous pressure), PV ,minus right atrial pressure, (PRA).



- Venous sympathetic vasoconstriction tone make the different in pressure between right atrium and systemic circulation
- Acute loss of sympathetic vasoconstriction tone ➔ Vasodilation ➔ no different in pressure ➔ ↓ VR ➔ ↓ CO ➔ Shock

Definition of Frank-Starling Law (Intrinsic regulation of CO)

Ability of the heart to change its force of contraction and therefore stroke volume in response to changes in venous return is called the **Frank-Starling mechanism**

Frank-Starling law states that the heart can pump all blood coming to it without allowing systemic venous stasis (within physiological limit).

force of cardiac contraction is directly proportional to the initial length of cardiac muscle (EDV)

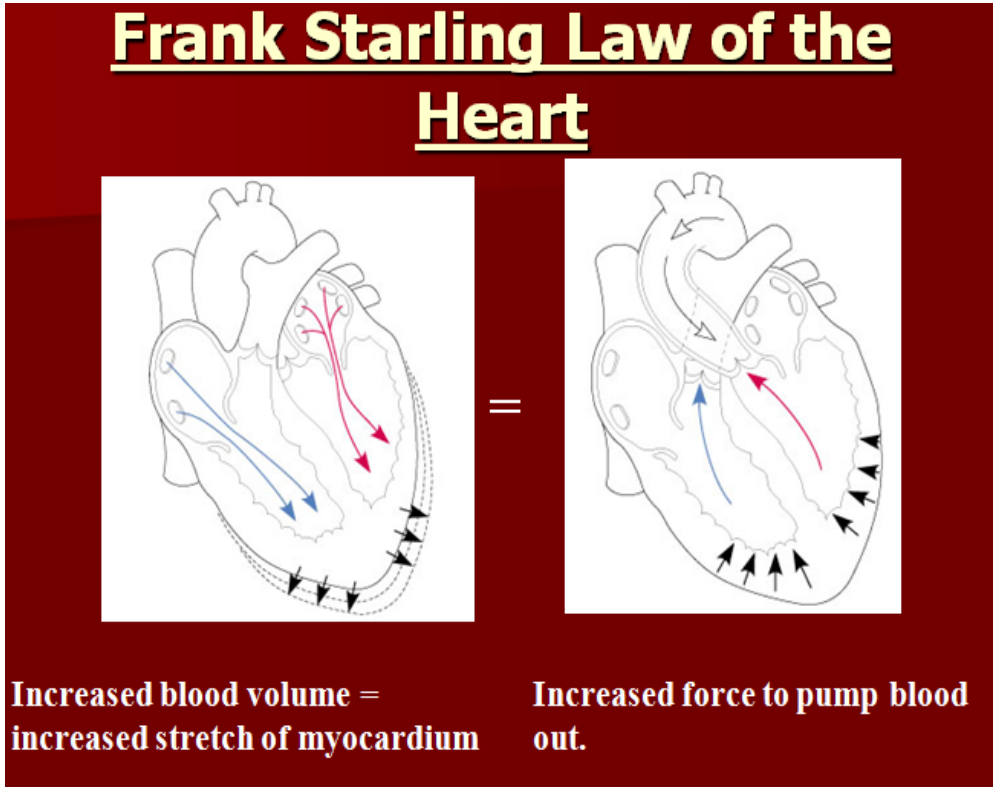
e.g hyper dynamic condition like hyperthyroidism →fast contraction →heart failure (if not treated)
The blood is pumping too much because of the fast contraction and the heart is doing its best pumping all the blood it receives but eventually have heart failure

Starling law

Increased venous return increases the ventricular filling (end-diastolic volume) and therefore preload which is the **initial stretching** of the cardiac myocytes prior to contraction.

This mechanism enables the heart to **eject** the additional venous return, thereby **increasing stroke volume.**

(↑VR ➔ ↑SV)



Factors affecting VR (Preload)		
1	Muscle pump	contraction of limb muscles as during normal locomotion (walking, running, swimming) **increases** venous return by the muscle pump mechanism.
2	Sympathetic vasoconstrictor tone	Sympathetic activation of veins increases central venous pressure and **increases** venous return and cardiac output through the Frank-Starling mechanism, which increases the total blood flow through the circulatory system.
3	Respiratory activity	During inspiration, the venous return **increases** because of a decrease in right atrial pressure
4	Vena cava compression	when the thoracic vena cava becomes compressed during a Valsalva maneuver or during late pregnancy, **decreases** venous return
5	Tissue metabolism	An increase in tissue metabolism as after meals, or in hyperthyroidism causes an **increase** in venous return
6	gravity	venous return **decreases** when changing from supine to erect posture

	Factors increase PRELOAD	Factors decrease PRELOAD
Venous pressure	**Increase** result from sympathetic vasoconstriction or increased thoracic blood volume	**Decreased** resulting from reduced blood volume, (hemorrhage) or gravity causing blood to pool in the lower limbs when standing upright
Atrial contraction	**Increase** resulting from sympathetic stimulation of the atria or from increased filling of the atria and therefore increased atrial contractile force through the Frank-Starling mechanism.	**Impaired** that can result from atrial arrhythmias such as atrial fibrillation.
Heart rate	**Reduced** which increases ventricular filling time	**Increased** which reduces ventricular filling time.
	Increased ventricular volume which results in a greater expansion of the chamber during filling at a given filling pressure	Decreased ventricular afterload, which enhances forward flow (i.e., ejection) thereby reducing end-systolic volume and end-diastolic volume secondarily.
	Increased aortic pressure, which increases the **afterload on the ventricle, reduces stroke volume by increasing end-systolic volume ,and leads to a secondary increase in preload.**	Inflow (mitral and tricuspid)valve stenosis which reduces ventricular filling.
	Pathological conditions such as ventricular systolic failure and valve defects such as aortic stenosis ,aortic regurgitation, pulmonary valve stenosis and regurgitation have similar effects on right ventricular preload	Ventricular diastolic failure decreased ventricular compliance) caused, for example, by ventricular hypertrophy or impaired relaxation.

Factors affecting End diastolic volume (EDV)

Increase:

- Stronger atrial contraction.
- Increased total blood volume
- Increased venous tone.
- Increased skeletal muscle pump.
- Increased negative intrathoracic pressure

Decrease:

- Standing
- Increased intrapericardial pressure.
- Decreased ventricular compliance

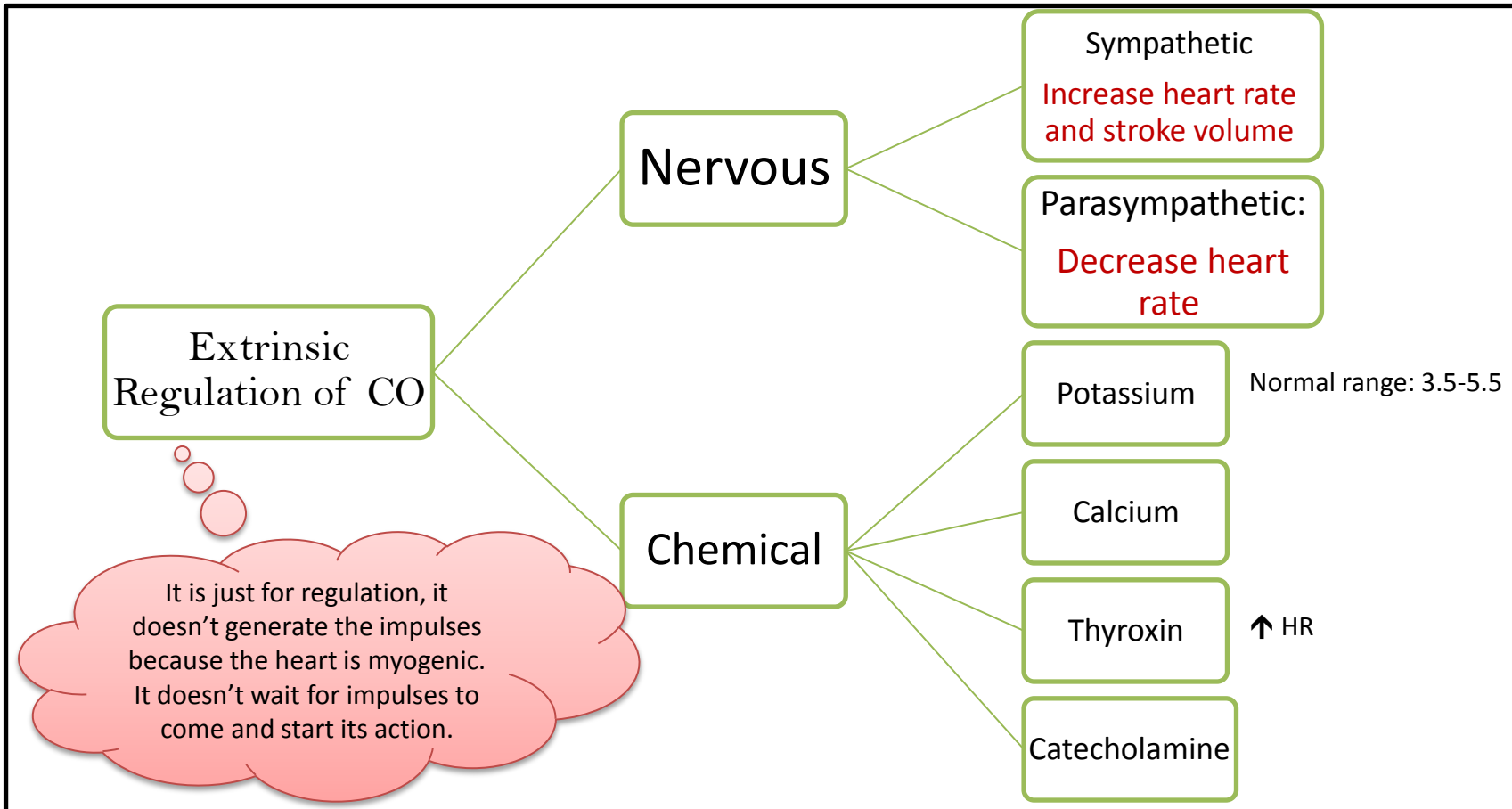
Factors determining CO

2- Afterload (Aortic Pressure)

Afterload can be thought of as the "load" that the heart must eject blood against. In simple terms, the afterload is closely related to the aortic pressure.

When arterial pressure is reduced, the ventricle can eject blood more rapidly, which increases the stroke volume and thereby decreases the end-systolic volume. Because less blood remains in the ventricle after systole, the ventricle will not fill to the same end-diastolic volume found before the afterload reduction. Therefore, in a sense, the end-diastolic volume (preload) is "pulled along" and reduced as end-systolic volume decreases. Stroke volume increases overall because the reduction in end-diastolic volume is less than the reduction in end-systolic volume .

In Hypertension patient, the heart try to compensate and exert more efforts which leads to hypertrophy to maintain CO in normal level.



Factors affecting cardiac output

- Venous return
- Heart rate
- Stroke volume
- Blood volume.
- ABP (Arterial Blood Pressure)
- Neural factors

Measurement of CO

Echocardiographic
techniques and radionuclide imaging techniques can be used to estimate real-time changes in ventricular dimensions, thus computing stroke volume, which when multiplied by heart rate, gives cardiac output.

Fick Principle :
An old technique based on the can be used to compute cardiac output (CO) indirectly from **whole body oxygen consumption (VO2) and the mixed venous (O2ven) and arterial oxygen contents (O2art);** however, this technique is seldom used. The CO is calculated as follows:

From any artery **From pulmonary vein !!**

$$CO = VO2/(O2art - O2ven)$$

To calculate CO, the **oxygen contents of arterial and venous blood** samples are measured, and at the same time, whole body oxygen consumption is measured by analyzing expired air. The blood contents of oxygen are expressed as ml O2/ml blood, and the VO2 is expressed in units of ml O2/min. **If O2art and O2ven contents are 0.2 ml and 0.15 ml O2/ml blood**, respectively, and **VO2 is 250 ml O2/minute**, then **CO = 5000 ml/min, or 5 L/min**. Ventricular **stroke volume** would simply be the **cardiac output divided by the heart rate.**

SUMMARY

- **SV= EDV-ESV** → what increases the SV, either EDV increse or ESV decrease .
- All **heart disease** decrease CO .
- Cardiac index is important to determine whether **CO is enough** for a person BSA(body surface area) and it differs from one person to another .
- **↓ HR → ↑ EDV → ↑ stretch of heart → ↑ force of contraction → ↑ CO**
- The blood enters the heart as **venous return** and leaves it as **cardiac output** meaning both amounts has to be **equal**.
- Systemic veins are supplied by sympathetic fibers which give the vein vasoconstrictor tone which is essential for maintenance of the **pressure gradient** between systemic circulation and right atrium if the **tone is lost** there would be **acute venous dilation** →no pressure gradient → ↓ VR → ↓ CO → **shock**
- The significance of the **frank starling law** that is **prevents venous stasis**
- Hyperthyroidism, Exercise, fever and velocity increase **tissue metabolism** ➔ **↑ VR**
- **Anemia** ➔ Tissue hypoxia ➔ Vasodilatation ➔ ↑ Blood flow ➔ ↑ heart rate ➔ **↑ CO**

SUMMARY

- In **Hyperthyroidism** : ↑ sensitization of catecholamine receptor ➔ augmented effect of B-receptor ➔ ↑ contraction of blood vessels ➔ ↑ HR ➔ **↑ CO**
- **Rapid arrhythmias** : in Tachycardia no enough time for ventricle to filled up with blood ➔ **↓ CO**
- Reduced HR (**bradycardia**) ➔ ↑ ventricular filing ➔ ↑ stretch ➔ ↑ force of Contraction → **↑ CO**
- But CO=HR x SV , doesn't that mean HR increases CO? **Physiological limit**

It depends on the HR and how much it increase (e.g. **exercising** will increase HR therefore **increase CO** up to 7 fold the normal range)

But in **tachy-arrhythmia** the HR will increase up to (300-400 Bpm) thus will decrease ventricular filling therefore the amount of blood is less and that will **decrease CO**

- How the VR is related to CO ?

When **VR increase**, **CO will increase** and vise versa

- How does VR regulate CO ?

VR regulate CO through **frank starling law mechanism** when VR increases it will lead to a degree of stretch of ventricle so this will increase the force of contraction therefore CO increase .

THE END

If there are any problems or suggestions
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THANK YOU

Actions speak louder than Words