### ≥ Drug interaction :- DON'T use the following drug with digitalis;

- ✓ Ca ++ → Synergism.
- ✓ Adrenaline → Arrhythmia.
- ✓ Atropine → Anti-vagal.
- ✓ Virapamile → Negative inotropic & ↑ digitalis absorption → ↑ toxicity.
- ✓ All diuretics except K-retaining → hypokalaemia → precipitate for toxicity.
- $\checkmark$  β-blockers → -ve inotropic effect .

### ≥ Preparations of digitalis :-

- ✓ Digoxine (Lanoxine):- tablet (0.25 mg), ampule (0.5 mg); metabolized in kidney.
- ✓ Digitoxine: tablet (0.1 mg), ampule (0.2 mg); metabolized in liver & GIT.
- ✓ Cedilanid: ampule (0.4 mg); metabolized in kidney.
- ✓ Oubain: ampule (0.5 mg) metabolized in kidney & GIT, rabid onset & short duration.

#### ≥ Dose of digitalis :-

- ✓ Therapeutic serum level of digitalis = 0.5 ng/ml (measured by radio-immuno assay), if it  $\uparrow$  to 2.5 ng/ml  $\rightarrow$  toxicity.
- ✓ There are 2 doses for digitalis:-
  - \* Digitalizing dose: 1.25 mg, till reach the therapeutic serum level.
  - Maintenance dose :- 0.5-1 tablet (about 0,25 mg)
- ✓ There are 2 methods to give digitalis:-
  - Non-Digitalizing method :- ( in chronic stable HF )
    - Begin with maintenance dose & digitalis will reach therapeutic level in 2 weeks.
  - \* Digitalizing method: (In acute HF & paroxysmal atrial tachycardia)
    - → It can be given either I.V. or oral.
    - → In I.V. method:-
      - ☑ Give 2-3 ampoules in one day (1mg / 1/2h) then continue by maintenance dose after end of digitalization.
      - It is important for patient not to take digitalis 2 weeks before digitalization to avoid toxicity
    - → In Oral method:- It is given on 1, 3 or 7 days, as follow;

digitalizing dose + (maintenance X No. of days)

### <u>Digitalization dose / day = </u>

No. of days (1, 3 or 7)

### Criteria of response to digitalis:-

- ✓ Improvement of symptoms & signs.
- ✓ Clearing of congested lung bases.
- ✓ Diuresis with loss of edema and ↓ BW.
- ✓ Slowing of heart rate.
- ✓ Early manifestations of digitalis toxicity.

### Digitalis toxicity:-

### ☆ Predisposing factors :- (20,4H&2D)

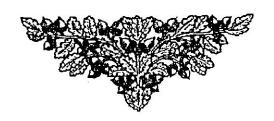
- <sup>▲</sup> Hypomagnisaemia.
- ▲ Hypothyroidism: thyroxine ↑ clearance of digitalis.
- <sup>⊥</sup> Hypokalaemia: It potentiate action of digitalis → K is pushed outside cells.
- ▲ Hypocalcaemia:- It has synergetic effect.
- \* Diseases:- liver, renal disease & failure.
- A Drugs: Verapamile, quinidine, amiodarone & β-blocker.

#### ☆ Clinical presentation :-

- \* GIT:- Anorexia (1st), nausea, vomiting & diarrhea.
- \* CVS:- Bradycardia (1st), Pulsus bigeminus or trigeminus (pathognomonic), heart block & any type of arrhythmia except sinus tachycardia.
- ▲ Occular: Yellow vision blurring of vision & optic neuritis.
- \* CNS:- Headache, confusion & convulsions.
- ▲ Others: Gynaecomastia & hyper-coagulability.

### ☆ Investigations to asses digitalis toxicity:-

- ▲ Pulse:- ↑ in under-digitalization & ↓ in over digitalization.
- ▲ ECG:- Electrical (pulsus) bigeminus.
- \* Radio-immuno assay:- detect serum level of digitalis.





### ☆ Treatment of digitalis toxicity :-

- ▲ Stop the drug.
- \* Stop any drug causing hypokalaemia (laxatives, diuretics & steroids).
- A Give KCl, I.V., 1 gm, 3 times / day.
- ▲ Give Ca chelating agents: as EDTA; 4 gm / 500 cm glucose infusion.
- ▲ Symptomatic treatment :- for;
  - ◆ Vomiting & nausea:- chloropromazine; 25 mg.
  - Bradycadia: atropine; 1 mg/SC.
  - · Heart block :- artificial pace-maker .
  - Cardiac arrest :- Intra-cardiac adrenalin , Cardiac massage & C-P resuscitation .
  - \* Ventricular tachy-arrhythmia:- anti-arrhythmic drugs (lidocaine & phenytoin).
  - VF: direct current cardioversion → depolarize all irritable areas except SAN.
- ▲ Digitalis antibodies: FAB fragments from sheep RBCs (recent & help digitalis excretion)

  N.B.: Digitalis is bound to tissues so, it is un-dialyzable.

### 5) Treatment of Cardiogenic Pulmonary oedema (Acute HF)

المنتن 1- Hospitalization .

- 2- Semi sitting position with leg hanging down.
- 3- Aspiration of secretions .
- 4- Oxygen inhalation :- 5-6 L / min by mask or nasal tube .
  - 5- Drugs :- (in order)

### → Morphine:-

- Action :- 1 Cardiac work, it is not given in non-cardiogenic pulmonary oedema.
- $\triangle$  Dose: 5-10 mg I.V. or S.C.
- ▲ Value:-↓ preload, ↓ dyspnea, ↓ agitation, ↓ VF & Analgesic.
- Lasix :- till 1 gm (40 ampoules), it ↓ cardiac work.
- ⇒ Vasodilators:- ↓ pre- & after load.
- Dopamine & Dobutamine :- they have +ve inotropic effect .
- Aminophylin :- I.V.; 250 gm slowly then 250 infusion ( if rapid → arrhythmia ).
- Digitalis.
- Rotating tourniquet: apply 3 tourniquets to 3 limbs & change their site to avoid ischemia.
- ○ Correction of precipitating factors: to ↓ VR.
- Treatment of complication :- e.g. digitalis toxicity.



#### N.B. :-

oxtimes Medical venesection:-dilators & diuretics (  $\downarrow$  blood volume).

🗵 Surgical venesection :- Small incision in vein to loose some blood (not used now).

☑ Treatment of acute pulmonary oedema:-(involve 2 items in answer)

◆ Treatment of Cardiogenic pulmonary oedema ( as before ).

\* Treatment of non-Cardiogenic pulmonary oedema :- ttt of the cause (refer to G. medicine)

### 6) Treatment of refractory Heart failure (Chronic HF - CHF)

★ Definition of refractory HF:- HF resisting the normal line of treatment.

### \* Aetiology:- there are 4 groups of causes;

- → Disease :- Un-corrected causes & Un-corrected pre-disposing factors . ( mention )
- → Patient :- Un-compliance ( stop digitalis , excess salt & in-adequate rest ) .
- → Doctor :- either;
  - \* Wrong diagnosis: pregnancy, Pericardial effusion, obesity, constrictive pericarditis.
  - Iatrogenic :- under dose of digitalis or diuretics , inadequate salt restriction , CCBs , COC ,  $\beta$ -blockers & NSAIDs .
- Others: Sever damage of heart (advanced cardiomyopathy & extensive MI).

#### ☆ Treatment :-

- الرواهد 1- Treatment of the cause ⇒ example . مثالرواهد
- 2- Treatment of precipitating factors ⇒ example. Uin le
- 3- Proper diagnosis of HF.
- 4- Proper dose of digitalis.
- 5- Improve compliance:-
  - ◆ Adequate salt restriction (0.5-1 gm/day).
  - Strict instructions to patient (drugs given by nurse).
- 6- If No response: add I.V., Dobutamine, diuretics & Vasodilators.
- 7- Surgical repair :-
  - Cardiomyoplasty: using latissmus dorsi muscle to help heart in its work till cardiac transplantation.
  - + Cardiac transplantation.
  - ◆ Intra-aortic ballon counter pulsations :- it acts as peripheral heart which relax in systole & contract in diastole ( it is filled with Helium ).



# a Important N.B. &

El Heart maybe enlarged & compensated without heart failure (as in athletes); so chamber enlargement not mean HF.

☑ Cardiomegally may be absent in HF (i.e. HF with normal sized heart) in;

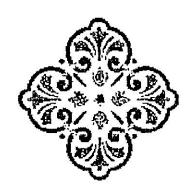
- ✓ Acute HF.
- VMS.
- ✓ Restrictive cardiomyopathy .
- ✓ Constrictive pericarditis .
- $\checkmark$  Cor-pulmonale due to COPD because heart is compressed bilaterally by hyper-inflated lungs .
- $\boxtimes$  1 severity of dyspnea in LSHF means  $\Rightarrow$  development of <u>RSHF on top</u>.

### ☑ Differential diagnosis of LSHF:-

- ✓ Other causes of dyspnea (chest dyspnea).
- ✓ Cardiac asthma.
- ✓ DD of aetiology of HF.

Parameter.	Chest dyspnea	Cardiac dyspnea
* Cough & expectoration :-	• Early .	◆ Late & positional .
≭ PN Dyspnea :-	◆ Not occur	◆ Characteristic
* Orthopnea :-	• late .	◆ Early , ↓ when RSHF occur
★ Ejection fraction :-	• $OfRV \rightarrow I$	• $OfLV \rightarrow 1$ .

Digitalis has no effect on normal heart.



# SYSTEMIC HYPERTENSION

Definition: - Persistent elevation of blood pressure above normal (140/90).

#### Classification :-

#### A) Systolic or diastolic?

- → Systolic HTN:- ↑ systolic blood pressure with normal diastolic blood pressure.
- → Diastolic HTN:-↑ diastolic blood pressure with normal or ↑ systolic BP.

#### B) Labile or established:-

- → Labile HTN: Transient ↑ in DBP during excitation, exercise & .... then return normal.
- → Established HTN:- persistent.

### C) Malignant or Benign :-

- → Malignant HTN:- show the following criteria;
  - ✓ Diastolic pressure > 130 mmHg.
  - ✓ Papilloedema.
  - ✓ Fibrinoid necrosis of blood vessels.
  - ✓ With or without ⇒ acute heart failure or acute renal failure.
- → Benign HTN:- No presence of the above mentioned points.

### D) Primary or secondary:-

- → Primary HTN:- Has NO aetiology but theories (hereditary, neural & hormonal)
- → Secondary HTN: due to many causes; renal, endocrinal, CNS, ..... etc.

### Aetiology of HTN :-

### A) Aetiology of Systolic hypertension :- (factors that † SV & | elasticity of aorta)

- → ↓ Elasticity of aorta: atherosclerosis.
- → ↑ SV:- due to AR, complete HB, PDA & Thyrotoxicosis.

### B) Aetiology of Diastolic HTN: (common); it is either;

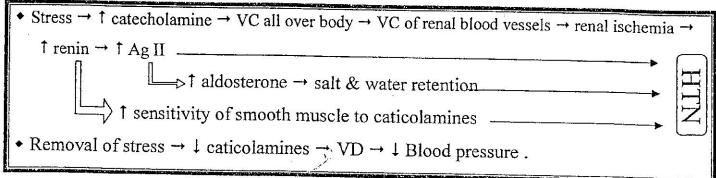
### I] Primary HTN (essential HTN):- (95 %, 35-50 y, males = females)

### ☆ Causes :-

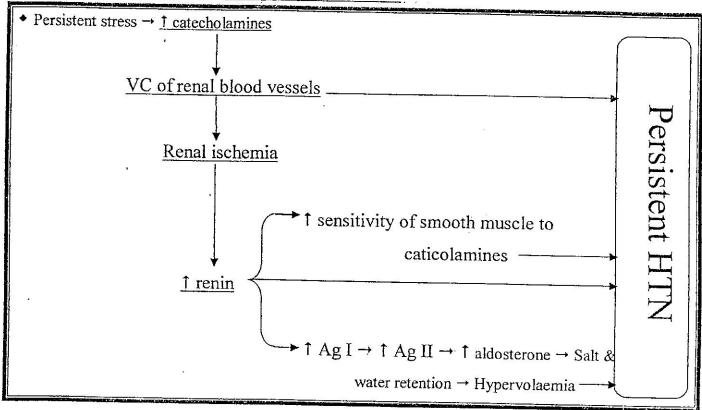
- 1- Hereditary theory.
- 2- Neural theory: excessive emotion → ↑ adrenalin → HTN.
- 3- Hormonal theory:-
  - √ ↑ release of vasopressors ( renin & corticosteroids )
  - √ ↓ release of vasodilators ( PGs ).
- 4- Multifactorial theory: (most accepted); different factors co-operate with each other.

### \* Mechanism of Primary HTN :-

#### A In Labile HTN:-



### A In established hypertension:-



### II) Secondary HTN:- It is due to 2ry causes else where;

### 1) Renal causes :-

<u>≥ Reno- paranchymatous causes</u>:- (glomerular, tubular & interstitial tissue)

- Example: TB, hydronephrosis, glomrulonephritis & ....etc.
- Mechanism: salt & water retention, 1 excretion of vasopressors & 1 release of VDs.

### 🖎 Reno-vascular causes :-

- Example: Renal artery stenosis → ↑ rennin secretion.
- Causes :-
  - ✓ Atherosclerosis :- bilateral , old age & male .
  - ✓ Congenital fibro-muscular dysplasia of wall: unilateral, young & female.
  - ✓ Others: trauma, thrombosis, compression by tumour & Vasculitis.

### 2) Endocrinal causes :-

- A Pheochromocytoma ( † catecholamine release ).
- ➤ Caushing's disease (↑ cortisone).
- Շonn's syndrome (↑ aldosterone).
- Adreno-genital syndrome.
- ➤ Thyrotoxicosis → Systolic HTN.
- ≥ Myxedema: due to associated atherosclerosis → Diastolic HTN.
- ➤ Acromegally (↑ GH).
- $\geq$  DM:- we diagnose HTN in diabetic patient if BP > 130 / 85.
- > Hyperparathyroidism :- due to hypercalcaemia .

#### 3) CNS causes :-

- 🖎 Bulbar poliomyelitis.
- ➤ Lead poising → Polyneuritis.
- ➤ Encephalitis.
- ransection of spinal cord.

### 4) Vascular causes:-

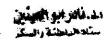
- Co-arcitation of aorta.
- Repoly-arthritis noose.
- Polycythaemia rubra vera .
- Acute intermittent porphyria.

### 5) latrogenic causes:-

- ≥ Post-commissurotomy.
- ➤ Contraceptive pills.
- ≥ Corticosteroids.
- ≥ Sympathomimetics .
- ≥ Liquorice → Pseudo-hyperaldosteronism.
- ≥ MAOIs with excess tyramine → ↑ adrenaline & NE.

### 6) Miscellaneous causes:-

- 🗷 Pre-eclampsia & eclampsia.
- Renin producing tumour.
- Barter's syndrome → ↑ renin .
- 🔁 Hypercalcaemia & hypervolaemia .



#### ⇒ Grading of HTN (Joint Nation Committee [ JNC ] classification ):-

Grade	Systolic BP	Diastolic BP
🖎 Normal :-	<b>•</b> 100-129	<b>•</b> 60-84
≥ High normal :-	<b>◆</b> 130-139	<b>*</b> 85-89
≥ Mild HTN :-	<b>+</b> 140-159	<b>+</b> 90-99
≥ Moderate HTN :-	◆ 160-179	<b>•</b> 100-109
≥ Sever HTN :-	<b>→</b> ≥ 180	<u>→ ≥ 110</u>

N.B. :- High normal = hypertensive diabetic patient.

#### ⇒ Clinical picture of HTN :-

#### A) Uncomplicated hypertension:-

I] Symptoms: - Asymptomatic, Occipital headache in morning, tinnitus & epistaxis.

#### II ] Signs :-

- 为 Blood pressure on measurement ( refer to grades of HTN )
- ≥ Pulse :- it involves ;
  - ☆ Pulse volume :- Big ( in systolic HTN ) & small or normal ( in diastolic HTN ).
  - ☆ Pulse character :- pulsus alternans ( LV Strain ).
- Auscultation:-(as pulmonary HTN but on Lt. side)
  - ☆ LV apex ( mention its criteria ) .
  - ☆ Accentuated S<sub>2</sub> ( paradoxical splitting if LSHF ).
  - ☆ Ejection click on aortic area.
  - ☆ Ejection systolic murmur: due to functional AS; due to dilatation distal to the valve.
  - ☆ Later on → Early diastolic murmur; due to functional AR; due to dilatation of valve ring.

### B) Complicated hypertension :- [Complications of HTN]

### ≥ Causes of these complications :-

- ☆ Accelerated atherosclerosis.
- ☆ Sudden rapid ↑ in blood pressure → rupture vessel & haemorrhage.
- ☆ Thrombosis & embolism on top of atherosclerosis.

≥ Types of Complications :-

### 1- Cerebral complications:-

- ☆ Transient ischemic attacks (TIAs) → refer to neurology.
- ☆ Haemorrhage (intra-cranial Haemorrhage):- Sudden rise of BP → rupture vessel.
- Arr Thrombosis  $\rightarrow$  lacunar infarction.

#### ☆ Hypertensive Encephalopathy:-

- <u>✓ Definition</u>:- Transient neurological manifestations due to sudden ↑ in BP above cerebral auto-regulation mechanism.
- ✓ Pathogenesis: Sudden † BP → Reflex VD of cerebral b.v. → Brain oedema → † capillary permeability → leakage of fluid from capillaries into brain → C/P.

#### ✓ Clinical picture :-

- Sever HTN.
- ◆ C/P of ↑ ICT & brain edema :-
  - Headache, vomiting, disturbed consciousness.
  - → Focal deficit → aphasia & hemiplegia, later ⇒ Coma without lateralization.
- ✓ Treatment: all manifestations reversible with treatment.

### N.B. :-

 $\star$  Normally;  $\mathit{IBP} \to \mathit{peripheral}\ \mathit{VC}$ , but in brain, it causes  $\mathit{VD}$  to maintain cerebral blood flow.

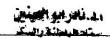
★ In HTN; there is cerebral VC, so; BP = 150/60 mmHg. If BP  $t > 150 \rightarrow VD$  of cerebral blood vessels  $\rightarrow$  Brain oedema  $\rightarrow$  Hypertensive encephalopathy with disturbed level of consciousness but without inflammation.

### 2- Cardiac complications :-

- ☆ Coronary heart disease.
- ☆ AF...
- ☆ Hypertensive HF.

### ☆ Brenhime effect :-

- ✓ Pressure over load on LV → concentric hypertrophy → bulging of septum into RV cavity → RVF → Systemic congestion.
- ✓ Reversed Brenhime effect: In pulmonary HTN → the same occur but on Lt side → LSHF → Pulmonary congestion.



### 3- Renal complications :- ( C / P of renal HTN )



- Accelerated atherosclerosis → renal artery stenosis → nephrosclerosis → ↑ BP,

  ↓ GFR, tubular dysfunction, proteinuria & microscopic haematuria.
- ☆ Chronic renal failure → due to glomerulosclerosis.
- ☆ Haematuria.
- ☆ Abnormal kidney function → urine examination, ↑ creatinine, ↑ blood urea.
- Acute renal failure in malignant HTN & Aortic dissection .

### 4- Retinal Complications :- ( hypertensive retinopathy )

- ☆ It has the following grades (SNOP);
  - ✓ Mild sclerosis of retinal vessels → Silver wire.
  - $\checkmark$  Marked sclerosis of retinal vessels with compression on veins (  $\underline{N}$  icking of veins ) by arteries at point of crossing.
  - ✓ Flame shaped haemorrhage & fluffy cotton exudates; once occur ⇒ accelerated HTN; ( Oedema, haemorrhage & Exudate).
  - $\checkmark$  Papilloedema; once occur  $\Rightarrow$  Malignant hypertension.

### 5- Vascular complications:-

- ☆ Small b.v.: rupture capillary → epistaxis, hypertensive retinopathy & fibrinoid necrosis of small blood vessels.
- \* Medium sized b.v.: accelerated coronary and cerebral atherosclerosis,

Hypertrophy of the media of arterioles (in malignant HTN).

☆ Large b.v. :- Cystic necrosis in media of aorta → Dissecting aneurysm .

### N.B. :-

- ightharpoonup Accelerated HTN means diastolic BP > 130 with NO papilloedema .
- → On measuring BP:-
  - ★ Measure 2 times :-
    - The  $I^{st} \rightarrow$  to get an idea about its level.
    - The  $2^{nd} \rightarrow to$  read accurately.
  - \* It must be under complete physical & mental rest.
  - \* It must be after some period of interview to avoid (stress white coat HTN) which is treated by assurance & ampulatory blood pressure monitoring.

#### ⇒ Investigations of HTN :-



### A) Basic investigation; for all patient: (3 U, sugar, salt, fat & 3 others)

- 1- Urine analysis: Pus cell cast, red cell cast (GN), fixed SG(RF) & sugar (DM).
- 2- Urea & creation in blood: to evaluate kidney function.
- 3- Uric acid in blood:- Hyperuricaemia & stop diuretic causing it.
- 4-Blood sugar: DM, Pheochromocytoma, Cushing's & Diuretics.

### 5-Na+, K+ & Ca++ in blood:-

- ↑ Ca<sup>++</sup> → first manifestation in HTN.
- Conn's syndrome → ↑ Na & ↓ K, before diuretic therapy.
- 6- Lipid in blood: cholesterol & TGs.

### 7- X-ray chest:-

- Prominent aortic knuckle with unfolding of aorta ( in non-complicated HTN ).
- Calcified Aortic knuckle.

### <u>8- ECG:</u>-LVH.

9- Fundus examination: to evaluate patient; fundus is the mirror of kidney.

### B) Selected investigations: (according to suspected Aetiology); for example;

- 1- Co-arcitation :- chest X-ray (Rosler sign), catheter & Angiography.
- 2- Polycythaemia:- blood picture.
- 3- Investigations for renal HTN:-
  - \*\*Reno-parenchymatous:- US kidney, kidney function test & IVP.
  - \*\*Reno-vascular causes:- Angiography, DVI, Renal vein renin level of both kidneys (normally = 1:1 & changed if unilateral renal artery stenosis)

### 4- Investigations of endocrinal causes :-

- \* Thyroid: T3, T4 & TSH.
- ☆ Acromegally:-GH.
- \* Cushing's syndrome:- cortisone & Glucose in blood.
- \*\* Conn's syndrome :- aldosterone in blood , Na & K in blood .
- \* Pheochromocytoma:- COMT in blood & VMA in urine.

# Treatment of Hypertension

### When to treat HTN?

- ☆ If diastolic BP > 90-100 mmHg.
- ☆ Marked symptoms.
- ☆ Target organ damage (kidney, heart & retina)
- ☆ Other risk factors: → DM.

### Aim of treatment of HTN:-

- ☆ Prevention of complications .
- ☆ Improvement of life style .

### 🗷 General ( non-pharmacological ) measures :-

- ☆ Assurance :- Avoid anxiety & stress.
- ☆ Diet :- Salt restriction, ↓ total calories & ↓ lipids.
- ☆ Habits:- stop smoking.
- ☆ Exercise: Walking (best), avoid vigorous exercise (complications).
- ☆ Complete physical & Mental rest

### Treatment of primary HTN :-

### A) Treatment of labile HTN:-

- General measures, if NO response, add ...
- $\rightarrow$   $\beta$ -blockers.

### B) Treatment of established HTN:-

- **Mild HTN**: (Stepped care approach)
  - Begin with diuretics, if NO improvement, add, ...
  - $\rightarrow$   $\beta$ -blockers, if NO improvement, add, ....
  - → Vasodilators.

### N.B.: Why we don't start with $\beta$ -blockers in treatment of HTN?

✓ High renin HTN.

✓ Hyperdynamic circulation .

✓ Arrhythmia.

✓ Ischemic heart diseases .

Orthostatic HTN

✓ Emotions .

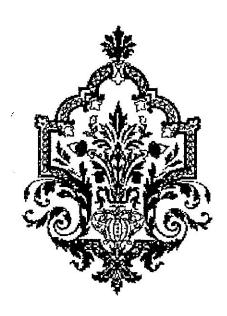
### Moderate & Sever HTN: there are 2 strategies;

# 1-Stepped care approach (Not used now):-

- $\rightarrow$  Begin with diuretic &  $\beta$ -blockers , If NO improvement , add , ....
- → Vasodilator drugs, if NO improvement ⇒ Resistant HTN, which is due to:-
  - \* Drug interaction: Sympsthomimetics in treatment of common cold.
  - ★ Secondary HTN.
  - \* Non compliance of patient.
  - ★ Wrong diagnosis.
  - \* Excess salt intake.
- Try o treat these causes, if NO improvement, the treatment will be as follows;
  - $\star \alpha$ -blockers (prazocin) +  $\beta$ -blockers (propranolol).
  - ★ Centrally acting anti-hypertensives ( stimulate VD center ) + Peripheral  $\alpha$ -blockers (block VC effect of  $\alpha$ -receptors).
  - \* Hydralazine.
  - \* Menoxidil.
  - ★ Ca<sup>++</sup> channel blockers (CCBs)
  - \* ACE inhibitors...

### 2-Direct use of strong anti-hypertensives :-

As; CCBs or ACEIs, with adjustment of the dose according to the degree of HTN.



### ★ Treatment of Secondary HTN:-

- ☆ Co-arcitation: resection anastomosis.
- ☆ Conn's syndrome: resection of the tumour.
- \* Reno-parenchymatous:- control renal condition, use drugs safe on kidney.
- ☆ Reno-vascular:- if the kidney is;
  - Damaged → Nephrectomy .
  - Healthy → Per-cutaneous trans-luminal renal angioplasty.
- ☆ Thyrotoxicosis :- Anti-thyroid drugs .
- ☆ Myxedema:- replacement therapy (Thyroxin).

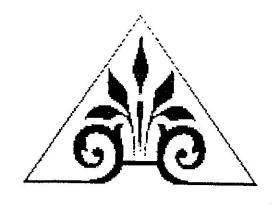
### <u>≥ Treatment of complications of HTN :-</u>

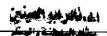
### A) Treatment of hypertensive encephalopathy:-

- → Hospitalization.
- → ↓ BP, by use of:-
  - In urgent states → Nifedipene, diazoxide & Na nitroprosside.
  - In less urgent states  $\rightarrow$  Hydralazine , reserpine &  $\alpha$ -methyl dopa .
- Anti-convulsant drugs: Phenobarbitone.
- → ↓ Brain oedema:- by using cerebral dehydrating measures, as; frusemide, Glucose 25 %, Mg-Sulphate, Mannitol 25 % or 10 % & Dexamethazone.

### B) Treatment of hypertensive nephropathy:-

- Use: α-methyl dopa, Hydralazine, Menoxidil, CCBs, Prazocin, ACEIs (↑ intervals [] doses) & β-blockers.
- → Don't use: K-retaining diuretics (due to † K) & Thiazides (as it ↓ RBF).
- → Dialysis & even Renal transplantation.





# Amti-Hypertensive Drugs

### 1) Centrally acting Anti-hypertensives:-

### ☆ α-Methyl dopa ( Aldomet ):-

- Dose:- tablet; 250 & 500 mg.
- $\bullet$  Mechanism: Stimulate central  $\alpha$ -receptors & act as a false transmitter.
- Side effects: Headache, hypotension, Hepatitis, Haemolytic anaemia & Impotance.

### ☆ Clonidine ( Catapress ) :-

- Dose: Pills; 0.1, 0.2 & 0.3 mg.
- Mechanism :- Central  $\alpha$ -agonist  $\rightarrow \downarrow$  Sympathetic out flow.
- Side effects :- Rebound HTN after sudden withdrawal .

### 2) Ganglion Blockers:-

- Example :- Trimetaphan .
- Side effects: Postural Hypotension & Atropine like side effects.

### <u>3 ) Sympatholytic drugs :-</u>

\( \alpha \) a-Methyl dopa: - It interferes with the synthesis of adrenaline & Nor-adrenaline.

### ☆ Reserpine (Bernardine):-

- ◆ Dose: Tablet; 0.1 mg.
- Mechanism :- Prevention of Epinephrine & NE re-uptake → depletion of them in nerve endings
- Side effects: Depression, Nasal congestion, † muscle tone (parkinsonism) & PU

### 4) β-Blockers:-

- Examples :-
  - ✓ Propranolol (Indral ) :- 10 & 40 mg ; Non-selective  $\beta$ -Blocker .
  - ✓ Atenolol ( Tenormin ) :- 50 & 100 mg ; Cardio-selective  $\beta_1$  blocker .
- Mechanism: ↓ Renin, ↓ COP & Re-adjustment of Baro-receptors.
- Side effects:- HB, precipitate HF, Bronchospasm, Aggrevate PVD & Mask hypoglycaemic symptoms.



### 5) & Blockers:-

- \* Examples: Prazocin (Minipress); 1, 2 & 5 mg.
- Mechanism: α-receptor blocker.
- Side effects:- First dose hypotension, so; BEGIN with 0.5 mg, at bed time.

### 6) Combined α& β-Blockers:

- Example:- Labetalol.
- \* Side effects :- Postural hypotension & Pheochromocytoma .

### 7) Angiotensin Converting Enzyme Inhibitors (ACEIs):-

- Examples :-
  - ✓ SH-group:- Captopril (Capotin); 20 & 25 mg.
  - ✓ Non-SH group :- Enalapril, Ramipril, Lisinopril, Kenapril & Benasipril.
- Mechanism :- Inhibit ACE which convert Ag-I into Ag-II.
- \* Side effects:- Metallic taste, Irritating cough, Proteinuria & Leucopenia.
- Advantages of Captopril: Contain SH group which protect Heart against active O2.

### 8) Ag-II Blockers:-

- Example:- Valsartan & Losartan...
- Side effects:- Less side effects than ACEIs.

## 9) Diuretics: -(↓ Pre-load → ↓ Plasma volume, ↓ Na in smooth muscles → ↓ sensitivity to NE)

### ☆ K-Loosing diuretics :-

- Examples: Thiazides (↑ Ca) & Loop diuretics → Frusemide (↓ Ca)
- Side effects: Hypokalaemia, Hyperuricaemia, Hyperglycaemia & Hyperlipidaemia.

### ★ K-Retaining diuretics :-

- Examples :-
  - ✓ Spironolactone ( Aldactone ) :- 25 & 100 mg , SE :- Hyperkaemia & Gynaecomastia .
  - √ Triamterene :- 100 mg , Side effect :- Stone formation .
  - ✓ Ameloride :- 10 mg.

### 10) Calcium Channel Blockers (CCBs):- It involves many members,

### ☆ Nifedipene :-

- Trade name :- Epilat , Adalat & Epilat retard .
- Dose: 10-20 mg, capsule (10 mg). It acts mainly on blood vessels.
- SE:- Headache, Flushing, Hypotension, Tachycardia & oedema LL dt peripheral VD.

### ☆ Verapamil (Isoptin)

- Dose: 80 mg. It's an anti-arrhythmic for SV tachycardia & act mainly on heart.
- Side effects: Bradycardia due to -ve inotropic effect & Constipation.
- ☆ Diltiazem :- 60 mg, act on the heart & cause coronary VD with less SE than verapamil.
- ☆ Isradipine (Lomir):-2.5 mg, high coast & low side effects.
- ☆ Amlodipine (Norvasc, Vasonorm):- 5 mg, low side effects.

### 11) Vasodilators:-

### ☆ Arteriolar vasodilators :- ( ↓ after load )

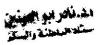
- Example :- Hydralazine , Saralazine & Minoxidil .
- ◆ Dose :- 10-20 mg; Tablet.
- Mechanisms: † Blood flow to kidney.
- Side effects :-
  - ✓ Hydralazine: Headache, palpitation, Tachycardia, SLE (6m)
  - ✓ Minoxidil: Pericarditis, Hirsutism (used for treatment of alopecia).

### ☆ Venodilators :- ( ↓ Pre-load )

- Example :- Nitrates .
- Side effects:- Headache, Tolerance & Met-haemoglobinuria.

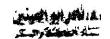
### ★ Balanced Vasodilators:-(cause VD of both arteries & veins)

- Examples: Diazoxide (500), Na-Nitroprosside (50, 100).
- Side effects :-
  - ✓ Diazoxide: Hypotension, Salt and water retention (use diuretic before it)
    & Hyperglycaemia.
  - ✓ Na-Nitroprosside: Hypotension, Salt and water retention & Cyanide poisoning (in liver).



# ≥ Special Conditions in treatment of HTN:-

Disease associated with HTN	Drugs used	Drugs NOT used
	ACEIs, CCBs & α-Blockers	
☆ Diabetes Mellitus	➤ Diuretics causes hyperglycaemia .	
	⇒ β-Blockers mask symptoms of hypoglycaemia.	
☆ Hyperlipidaemia	☞ As DM	← As DM.
	⊃ Diuretics & β-Blockers cause Hyperlipidaemia.	
A Peripheral Vascular Diseases	→ Diuretics, CCBs & α-Blockers	
(PVD)	⊃ β-Blockers aggrevates PVD &	cause $\downarrow$ renin $\rightarrow \downarrow$ RBF $\rightarrow$ RF.
	S AS PVD.	♂ As PVD.
☆ Renal Artery Stenosis	→ ACEIs can precipitate Re	nal failure.
# #	<b>⊃</b> β-Blockers causes ↓ renin → ↓ RBF → RF.	
	ு α-Methyl dopa,	Diuretics, CCBs, ACEIs,
☆ Pregnancy	Hydralazine & diazoxide	α & β-Blockers.
	⇒ Be sure that all drugs used are safe in pregnancy.	
☆ Ischemic Heart diseases	ACEIs, CCBs, Diuretics,	☞ NO drugs
(IHD)	α & β-Blockers.	
(1111)	⇒ Best is Nitrates & β-Blockers as it ↓ Cardiac work.	
☆ Renal Failure	☞ β & α-Blockers, CCBs.	Diuretics & ACEIs.
	☐ If diuretic is needed use Lasix only ( ACEIs is used with caution )	
☆ Heart failure	Diuretics, ACEIs & α-Blocker.	ு β-Blockers & CCBs .
	→ β-Blockers & CCBs aggrevate HF.	
	Diuretics, ACEIs,	
☆ Bronchial Asthma	α-Blockers & CCBs.	
	⊃ β-Blockers cause → Bronchospasm.	
<b>☆</b> Gout	CCBs, ACEIs,	☞ Diuretics .
	α& β-Blockers.	
u"	⊃ Diuretics cause → Hyperu	ricaemia .
		3



### ➤ Treatment of Hypertensive Emergencies:- (APCDEPhMM)

- 1- Acute renal failure :- Diuretics & Dialysis .
- 2- Pulmonary oedema :- Lasix & Vasodilators.
- 3- Cerebral Stroke ( Haemorrhage & Thrombosis ):- Na nitroprosside .
- 4- Dissection of the aorta: Na nitroprosside.
- 5- Encephalopathy: Na nitroprosside.
- 6- Pheochromocytoma Crisis: Combined α & β-Blockers.
- 7- Malignant HTN: Diuretics & Na nitroprosside.
- 8- Myocardial Infarction :- Nitrates .

### ≥ Types of Hypertensive Urgencies:-

- 1- Accelerated HTN.
- 2- HTN in renal transplant patient.
- 3- HTN in patient going to surgery.

#### N.B. :-

- ✓ Mean Blood pressure = Diastolic + 1/3 pulse volume.
- ✓ Suspect secondary HTN, if :-
  - ◆ Age < 35 or 50 years...
  - \* No +ve family history....
  - \* Detect Aetiology in clinical examination ...
  - ◆ Detect Aetiology in investigations ...

### ≥ Examples of Curable HTN:-

### A) Labile HTN.

### B) Most types of Secondary HTN:-

- ☆ Iatrogenic HTN: Stop the drug causing HTN.
- ☆ All Endocrinal HTN except DM .
- ☆ Pre-eclampsia & Eclampsia :- Treatment is Termination of pregnancy.
- ☆ Co-Arcitation of Aorta:- Surgery.
- ☆ Unilateral Kidney diseases:- Nephrectomy.
- AGN :- Corticosteroids .
- ☆ Renal artery stenosis:- Angioplasty.
- ☆ Polycythaemia:- Treatment of the cause, Venesection & Cytotoxic drugs.

# DYSRRHYTHMIA

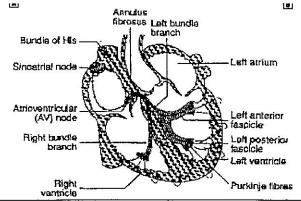
### ⇒ Structure of conducting system of the heart:-

★ SAN → inter-nodal tracts → AVN → bundle of hiss → the left bundle branch only → divides into anterior & posterior hemi-branch → the 3 terminal branches give origin to Berkinje fibers → Myocardium.

Annualus fibrosus Left bundle

### > Normal rate of impulses in the heart :-

- $\star$  SAN  $\rightarrow$  60-90 beat / m.
- $\star$  AVN  $\rightarrow$  40-60 beat / m.
- ★ Idio-ventricular rhythm → 25-40 beat / m.



#### N.B :-

- → Heart will obey the faster focus, so; if ectopic focus is faster than SAN, heart will obey it.
- → Types of ectopic cardiac rhythm :-
  - \* SAN fails  $\rightarrow$  another focus control heart  $\Rightarrow$  Passive rhythm.
  - \* Focus faster than SAN controls the heart  $\Rightarrow$  Active rhythm.
- All cardiac fibers have the conductivity criteria while conductive fibers of heart ONLY have the automaticity criteria.

### \* Definition of Dysrrhythmia:- It means any disturbance in rate or rhythm .

### \* Types of classifications of dysrrhythmia:-

Patho-Physiologial classification: there are 2 types;

### A) Disturbance of impulse formation:-

- ☆ Sinus dysrrhythmia:-[Normal SAN rhythm = 60-90 b / m·]
  - Sinus tachycardia  $\rightarrow >90 \text{ b/m}$ .
  - Sinus bradycardia  $\rightarrow$  <60 b / m.
  - Sinus arrhythmia  $\rightarrow$  60-90 b / m; but irregular rate .

### ☆ Ectopic dysrrhythmia :- [ ectopic focus ]

- Passive escape rhythm: There is failure to SAN; Atrial escape rhythm, Nodal rhythm & Idio-ventricular rhythm.
- Active ectopic rhythm: There is an ectopic focus faster than SAN;
  - ✓ Premature beats :- Atrial, Ventricular & Junctional.
  - ✓ Paroxysmal tachycardia:- Atrial, Ventricular & Junctional.
  - ✓ Flutter :- Atrial , Ventricular ( Group of fibers contract simultaneously )
  - ✓ Fibrillation: Atrial, Ventricular (each fiber give impulse).

### B) Disturbance of impulse conduction:-

- ☆ ↓ Conduction :- Heart block.
- ☆ ↑ Conduction:- Pre-excitation syndrome [e.g. WPW syndrome].

#### N.B. :- Wolf Parkinsonian white syndrome :-

- ✓ Definition:- It is a syndrome in which; there is accessory bundle [] atrium & ventricle which rapidly conduct impulses because; there is NO physiological delay as in AVN.
- ECG. Findings: Short P-R interval, wide QRS complex, Delta wave on ascending limb of R-wave.
- Complications: SVT, AF & Atrial flutter:
- ✓ Treatment: Surgery, Verapamil & Amiodarone.

### Clinical classification of Dysrrhythmia:-

#### A) Tachycardia:-

### ☆ Regular tachycardia :-

- Sinus tachycardia . (ST)
- Ventricular tachycardia (VT)
- Supra-ventricular tachycardia (SVT)
- Atrial flutter.

### 🖈 Irregular Tachycardia :-

- Sinus tachycardia + Premature beats.
- Supra-ventricular tachycardia (SVT) + Variable HB.
- Atrial flutter + Variable HB
- Atrial fibrillation (AF)

### <u>B ) Bradycardia :-</u>

### ☆ Regular Bradycardia :-

- Sinus bradycardia (SB).
- Partial fixed HB (conduction 2:1 to ventricles).
- \* Complete HB ⇒ Idio-Ventricular rhythm .
- A-V nodal rhythm (60 b/m).

### 🖈 Irregular Bradycardia :-

- Sinus bradycardia with Premature beats.
- Partial variable HB.
- AF under Digitalis
- Sinus arrhythmia.

### Other classification of Dysrrhythmia:-



### A) Formation abnormality:-

- ☆ Site: Supra-ventricular or Ventricular.
- ☆ Rate: Tachycardia, Bradycardia, Flutter & Fibrillation.
- B) Propagation abnormality: Sino-atrial, Intra-atrial, A-V & Intra-ventricular block (Lt. bundle, Rt. Bundle or Bifasicular block).
- C) Irregular heart with normal rate: Pre-mature Beat & Slow AF.

### **Important Notes**

#### Types of functional arrhythmia:-

- ✓ Massive tachycardia.
- ✓ Massive Bradycardia .
- ✓ Sino-atrial block.
- ✓ Adam's stock attacks.
- ✓ Sick sinus syndrome.

#### Vagal stimulation is done by:-

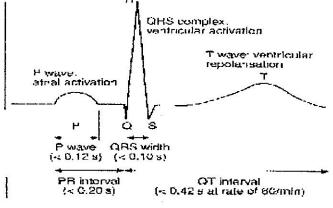
- ✓ Induction of vomiting.
- ✓ Occular compression .
- ✓ Carotid massage :- It MUST be ;
  - → Unilateral not bilateral.
  - → Contraindicated in Carotid bruit.
  - → Compression is done against transverse process of cervical vertebrae .
  - → Blow to epigastrium ...
  - → Neostigmine; 0.5 mg/I.V...

### A scheme for the points which will be discussed in any type of arrhythmia:-

- ✓ Definition: It must involve ⇒ rate, rhythm & focus.
- ✓ Symptoms: Asymptomatic, dyspnea, .....
- ✓ Signs: There are 3 important signs;
  - Radial pulse: Denotes ventricular activity.
  - → Neck veins: Atrial activity (A wave ⇒ Atrial contraction & CV wave ⇒ Systole)
  - → Auscultation.

#### ✓ ECG:-

- → P-wave: there are 4 possibilities;
  - → Originated from SAN ⇒ Normal P-wave .
  - → Originated from atria but extra-nodal ⇒ Deformed P-wave .
  - → Originated from AVN ⇒ Inverted P-wave .
  - → Originated from Ventricles ⇒ Absent P-wave (NO retro-grade conduction)
- ORS-complex :-
  - → If originated from supra-ventricular structures (SAN, Atria or AVN) ⇒ Normal.
  - → If originated from ventricles ⇒ Wide complex.
- The relation [] P-wave & QRS-complex is equal to that [] a-wave & cv-waves
- <u>Cannon a-wave occurs in :-</u> Nodal rhythm, Complete HB, Paroxysmal Nodal & V. tachycardia <u>Fa-wave is absent in AF</u>.



## A) Sinus Dysrrhythmia

Parameter	1- Sinus Tachycardia	2- Sinus Bradycardia
☆ Definition :-	◆ Regular impulses from SAN > 100 b / m	• Regular impulse from SAN < 60 b / m
☆ Aetiology :-	<ul> <li>Physiological:- children, exercise &amp; emotion.</li> <li>Drugs:- parasymatholytics, Sympathomimetics &amp; thyroxine</li> <li>Pathological:- Hypotension, hypoxia, hyperthermia, HF &amp; Hyperdynamic circulation.</li> </ul>	<ul> <li>Physiological: - athelets, Sleep, Carotid sinus syndrome.</li> <li>Drugs: - Parasympathomimetics, Sympatholytics, Digitalis &amp; CCBs.</li> <li>Pathological: - Obstructive jaundice, Myxedema, MI, Rapid † in ICT, Ascending cholangitis &amp; Meningitis.</li> </ul>
☆ Symptoms :-	<ul> <li>Asymptomatic .</li> <li>Palpitation .</li> <li>Dyspnea (due to 1 of cardiac reserve)</li> <li>Angina , MI , Syncope , nausea , vomiting</li> </ul>	<ul> <li>Asymptomatic .</li> <li>Palpitation .</li> <li>Low COP symptoms .</li> <li>g polyuria &amp; NO haemodynamic changes</li> </ul>
☆ Signs :-		
→ Radial pulse :-	• Regular, > 100 b/m.	• Regular < 60 b / m.
→ Neck veins :-	◆ 1 No of wave = pulse, normal shape	• the same but 1 No.
→ Auscultation :-	• Tachycardia, † S <sub>1</sub> .	• Bradycardia, Weak S1.
→ Special test:-	◆ Vagal stimulation → ↓ HR	◆ Sympathetic stimulation or vagal inhibition → ↑ HR gradually as they originate from atrium
☆ ECG:-		
→ P-wave :-	• Normal shape .	• Normal shape .
→ QRS-complex :-	Normal shape .	Normal shape .
→ A & V rate :-	◆ Atrial rate = Ventricular rate > 100	• Atrial rate + V. rate < 60 b / m.
☆ Treatment :-	<ul> <li>Treatment of the cause .</li> <li>Drugs :- <ul> <li>Para.mimetics :- Prostigmine .</li> <li>Sympatholytics :-</li> <li>a-blocker :- reserpine .</li> <li>β-blocker :- Propranolol</li> </ul> </li> </ul>	<ul> <li>Treatment of the cause .</li> <li>Drugs :-         <ul> <li>✓ Para.lytics :- Atropine (not in glaucoma &amp; senile prostate)</li> <li>✓ Sympathmimetics :- Adrenaline &amp; Isoprenaline</li> <li>✓ Artificial pacing in resistant cases</li> </ul> </li> </ul>

N.B. :-

<sup>✓</sup> All paroxysmal tachycardias are of rapid onset & offset, as they are produced by ectopic focus except sinus tachycardia & nodal tachycardia, they are of gradual onset & offset as they are produced by normal foci ⇒ [Warming up phenomenon].

### 3-Sinus Dysrrhythmia

- ★ Definition: Impulses arise from SAN at rate of 60-90 b / m but with alternating periods of slowing & acceleration.
- \* Aetiology:- It is common I young age with NO pathological significance.

### ☆ Types :-

- Respiratory sinus arrhythmia: 1 HR with inspiration & 1 with expiration.
- Non respiratory sinus arrhythmia: due to congenital rhythmic variation of the vagal tone
- 🖎 Sick sinus syndrome.

### ☆ Clinical presentation:-

- \* Asymptomatic .
- It can be missed clinically.
- It may mask underlying serious disorder.

### ☆ Investigations :-

- ECG: It shows the following;
  - ✓ Rhythmic variation in the rhythm of cardiac cycle.
  - ✓ Periods of tachycardia & others of Bradycardia with normal intervals (in each period separately)

### ☆ Treatment :-

- If Asymptomatic ⇒ NO need for treatment.
- If chronic ⇒ It may need artificial pace-maker.

### **B** ) Ectopic rhythm

### ➤ Types of Ectopic rhythm:-

- 1- Pre-mature beats (Extra-systole).
- 2- Paroxysmal tachycardia :- either supra-ventricular or Ventricular .
- 3- Atrial flutter.
- 4- Atrial Fibrillation.

# 1) Pre-Mature Beats (PMB - Extra-Systoles)

\*\*Definition:- It is a condition in which the ectopic impulses occur earlier than expected, the focus present either in atrium, AVN or ventricles, it may occur once, the rate is either irregular, normal or Bradycardia.

### Aetiology :-

- Physiological:- Exercise, Emotion, tea, Coffee & Fatigue.
- Drugs: Digitalis toxicity, Tobacco, Alcohol, thyroxine & adrenaline.
- \* Pathological:- Coronary HD, CHD, Valvular HD, HTN, Fever, Hypoxia, .....

\* Patho-Physiology:- The ectopic focus may present in atria; AVN or Ventricles

\* Types:- Atrial, Junctional or Ventricular.

### ☆ Clinical presentation :-

#### → Symptoms :-

- Asymptomatic , Palpitations , Anxiety & Sweating .
- \* It the heart is diseased :- Angina, Infarction, Syncope, HF or Low COP symptoms.

Atrial Pre-mature Beats

• If Multiple extra-systoles :- Angina, Heart failure.

#### → Signs :-

- Radial pulse: Usually Bradycardia (to allow its appearance), occasional irregularity, pulse deficit < 10 min & you can count 4 regular successive beats.
- Neck veins :- It differs according to it origin;
  - ✓ Atrial PMB: Normal shaped waves but earlier in timing.
  - ✓ Junctional PMB :- Regular Cannon a-waves .
  - ✓ Ventricular PMB :- Occasional Cannon a-waves .
- Auscultation: Occasional irregularities & NO Cannon heart sounds.
- Special tests: Exercise → disappearance of PMBs as ↑ HR → ↓ chance for ectopic bear to appear.

### ☆ Investigations (ECG):-

### \* Atrial PMB :-

- ✓ Deformed P-wave, or;
- ✓ Occasional irregularity occur earlier than expected followed by compensatory pause due to refractory period which mask next normal wave .

### + Junctional PMB :-

- ✓ Inverted P-wave, or;
- ✓ Masked by QRS complex with short PR interval.

### • Ventricular PMB :-

- ✓ No P-wave with wide QRS complex, or;
- ✓ Maybe associated with another normal atrial P-wave.

#### \$ Treatment :-

- 1- Treatment of the cause.
- 2- Sedation & Assurance.
- 3- According to the type of Premature beats :-

#### ○ Atrial or Junctional PMB :-

- Digitalis, if associated with HF.
- \* Quinidine .

#### ○ Ventricular PMB :-

- Lidocaine, if associated with myocardial infarction.
- Phenytoin, if results from digitalis toxicity.

### **⊃** Both atrial & Ventricular PMB :-

- Propranolol: 1 gm / 3 min, I.V...
- Procainamide: 100 mg/3 min, I.V., till signs of toxicity.

N.B. :- The serious PMB are ⇒ Ventricular PMB & Multiple PMB.

# 2) Paroxysmal Tachycardias

### I] Supra-Ventricular Tachycardia:

b/m, with NO Heart block at all (conduction is maintained at 1:1)

\* Aetiology:- as PMBs + GIT disturbance & chest infection.

### ☆ Clinical presentation:-

### → Symptoms :- ( Sudden onset )

- Palpitation, Anxiety, Exhaustion & Dizziness.
- ◆ In diseased heart :- Angina , ...... & Polyuria ( due to ↑ ANF ) .
- GIT disturbance.

### → Signs :-

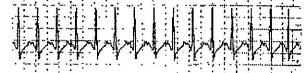
- Radial pulse :- Regular > 100 b / m.
- Neck veins: No. of waves = Radial pulse, Regular Cannon (if junctional).
- Auscultation: Tachycardia & Accentuated S<sub>1</sub>.
- Special test: Vagal stimulation → ↓ HR or disappearance of this tachycardia.

#### ☆ Investigations (ECG):-

- ◆ P-wave :- Deformed or inverted (if junctional), maybe before, after or masked by QRS complex.
- QRS complex: Normal shape, each P-wave is followed by QRS complex

### \$ Treatment :-

1- Sedation for anxiety & emotions.



- 2- Vagal stimulation & if the patient is hypotensive, DO;
  - \* Use Metaraminol (sympathomimetic) to ↑ BP then → Vagal stimulation.
  - ↑ BP to 180 mmHg by infusion → reflex Vagal stimulation → ↓ HR (Marrie's law)
- 3- Drugs therapy:-
  - ◆ Verapamil:-1 mg, I.V..
  - Propranolol:- 1 mg I.V.
  - Digitalis to protect ventricles .
- 4- Direct current (DC) Cardioversion (Last choice).

### II ] Ventricular tachycardia:-

\* Definition:- Regular impulses arise in ventricles with rate > 100 b / m.

### Aetiology:-

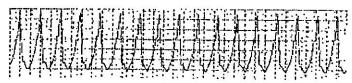
- Drugs :- Digitalis :.
- Pathological :- As before...

### \* Clinical presentation :-

- $\rightarrow$  Symptoms:- (sudden onset).
  - Palpitation, anxiety, dizziness.
  - Diseased heart :- Angina, ....., syncope & sudden death due to VF.

### → Signs :-

- \* Radial pulse:- Regular > 100 b / m.
- Neck veins: No. of waves ≠ pulse → Occasional a-wave.
- Auscultation: Tachycardia, Cannon St due to exaggerated closure of valve as a result of simultaneous atrial & Ventricular contraction.
- Special test: Vagal stimulation → No response (vagus don't supply ventricles).



### ☆ Investigations (ECG):-

- P-wave: Normal shape with rate = 80 b / m (Normal SAN rhythm).
- QRS complex: Wide with rate > 150 b / m.
- NO fixed relation [] P-wave & QRS complex ⇒ AV Dissociation.
- NO heart block but ⇒ The heart obey the faster focus.

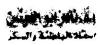
#### ☆ Treatment :-

- 1- Emergency treatment:-
  - ✓ DC Cardio-version (First choice).
  - √ Myocardial depressant :- as;
    - Lidocaine :- 50-75 mg . I.V. .
    - Procainamide :- I.V., if lidocaine fails.
    - Propranolol:- If both lidocaine & propranolol fail.
    - Phenytoin.
- 2- In Resistant cases :- Intra-Cardiac pacing ⇒ Implantation of Cardiovertor Defibrillator (ICD).
- 3- In [] the attacks: Treatment of the cause.

Parameter	3- Atrial Flutter	4- Atrial Fibrillation
☆ Definition :-	• Impulses arises from atrium, regular, at rate of 300 b / m but due to physiological delay only, 1/2, 1/3, 1/4 impulses are conducted to ventricles.	• Impulses arise from atrium, irregular, at rate of 400-600 b/m, due to physiological delay, Not all impulses are conducted to the ventricles (rate of V. = 150-200 b/m)
☆ Aetiology :-	<ul> <li>Drugs: Digitalis Toxicity.</li> <li>Diseases: CHD, Coronary</li> <li>HD, Valvular HD, HTN,</li> <li>Thyrotoxicosis,</li> <li>Cardiomyopathy &amp; Constrictive</li> <li>Pericarditis.</li> </ul>	<ul> <li>Drugs: Digitalis toxicity.</li> <li>Diseases: A.Flutter + Loan AF which is of unknown aetiology.</li> </ul>

myinte.





<u>Parameter</u>	3- Atrial Flutter	4- Atrial Fibrillation
☆ Patho-Physiology :-	◆ Circus movement theory :-	◆ Circus movement theory :-
10 10 21	→ one impulse circulate around	→ As A.Flutter, but at a rate 400-600
	IVC or SVC opening at rate of	, impulses rapidly fractionate &
	300 b/m & with each turn	stimulate different fibers ( Not
	daughter impulse arise to aria &	accepted).
	ventricles	
el N	◆ Ectopic focus theory :-	* Ectopic focus theory :-
20 E	→ Atrium contain ectopic focus produce	→ As A.Flutter but at rate of 400-600
	impulses at rate of 300 b/m.	
☆ Types :-		<u>◆ Paroxysmal AF:</u> Recent (new) <u>◆ Established AF:</u> > 2 weeks.
\$ Symptoms :-	• Sudden onset & offset .	• Paroxysmal AF:- as A.Flutter.
	Palpitation . In diseased heart:	• Established AF: minimal, better tolerated due to slow HR.
\$ Signs :-		
→ Radial pulse:-	• Regular, 75, 100, 150 b/m	• Irregular , 150-200 b/m , pulse
lid.		deficit > 10 min . You CAN'T count
5		4 regular successive beats.
→ Neck Veins :-	• No. of (a) waves = double,	• Absent (a) wave.
	treble or quadruple pulse rate.	• Systolic expansion if with TR.
		Marked irregularity in force , rate ,
		rhythm of contractions.
→ Auscultation:-	◆ S1 intensity depends on	Variable S <sub>1</sub> due to variable amount
1	ventricular rate.	of blood in LV $\rightarrow$ Variable rhythm.
	Atrial contractions maybe heard	◆ Absent S₂ in some cycles dt weak
	as distinct clicks [] heart sounds	impulse which can't open semi-lunar
•	F	valves.
→ Special signs :-	Vagal stimulations → ↑ AV	
	block from 2:1 to 3:1 or 4:1 in a	• Special test :-
Y.	mathematic fashion.	✓ Vagal stimulation → ↓ HR & ↑ block.
A-Y	A D manage 11	✓ Exercise → ↑ HR & ↑ Irregularity.
☆ Investigations :-	• P-wave:- replaced by regular flutter waves.	P-wave :- Replaced by irregular fibrillations waves .
(ECG)	• QRS complex :- normal, rate =	QRS-complex :- Normal shape &
	75, 100, 150. • Regular P-R intervals.	Irregular in timing . ◆ Irregular P-R interval .