Neo - New
plasia - Tissue growth → New tissue growth

Neoplasia Vs Hyperplasia & Repair

- Neoplasia is unregulated ✓
  irreversible ✓
  monoclonal. ✓

Hyperplasia:

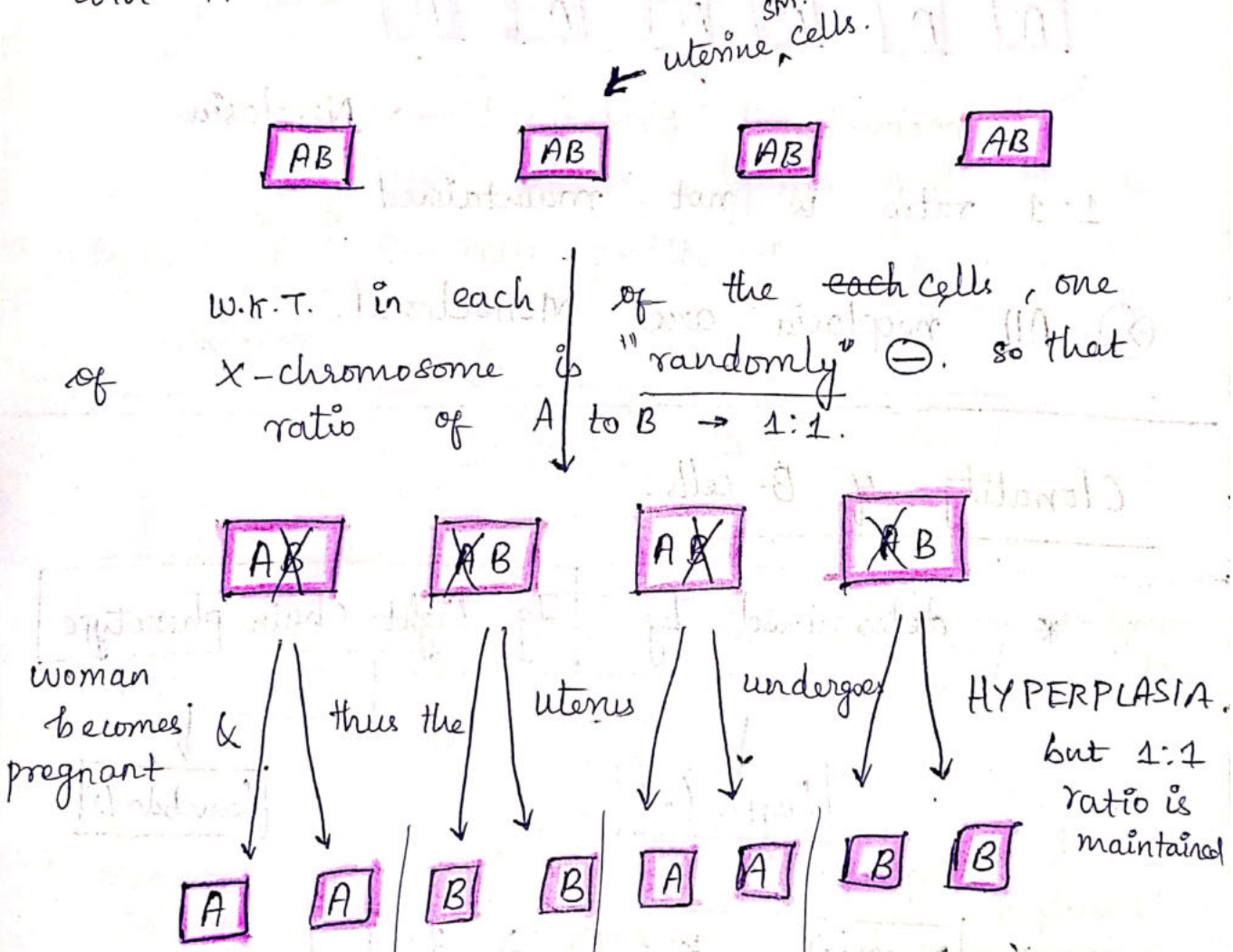
Women is pregnant (stress).
↓
growth of uterus
↓
But regulated by physiology of pregnancy
reversible after delivery
polyclonal - derived from many cells.

Monoclonal: Neoplastic cells are derived from a single mother cell.

Clonality is determined by.
- G6PD isoforms.
- Androgen Receptor isoforms

Isoforms of G-6-P-D : A B

Lets assume this female inherits ② isoforms A & B ... So each of cells will have both A & B.



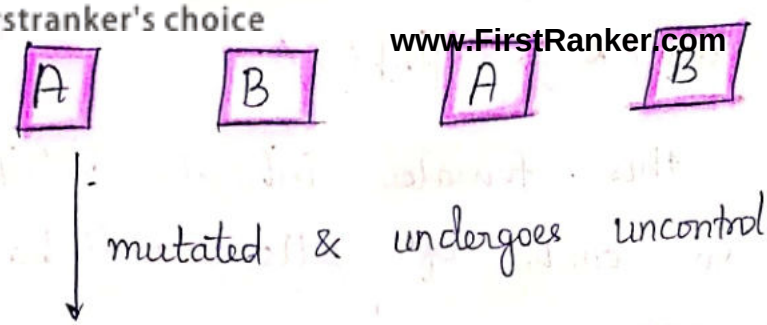
Hyper plasia ⇒ "Polyclonal" proliferation.

↓

cells are derived from many mother cells.

* Also in polyclonal prolifern, 1:1 ratio is maintained.

Uterine SM cells.



1:1 ratio is not maintained.

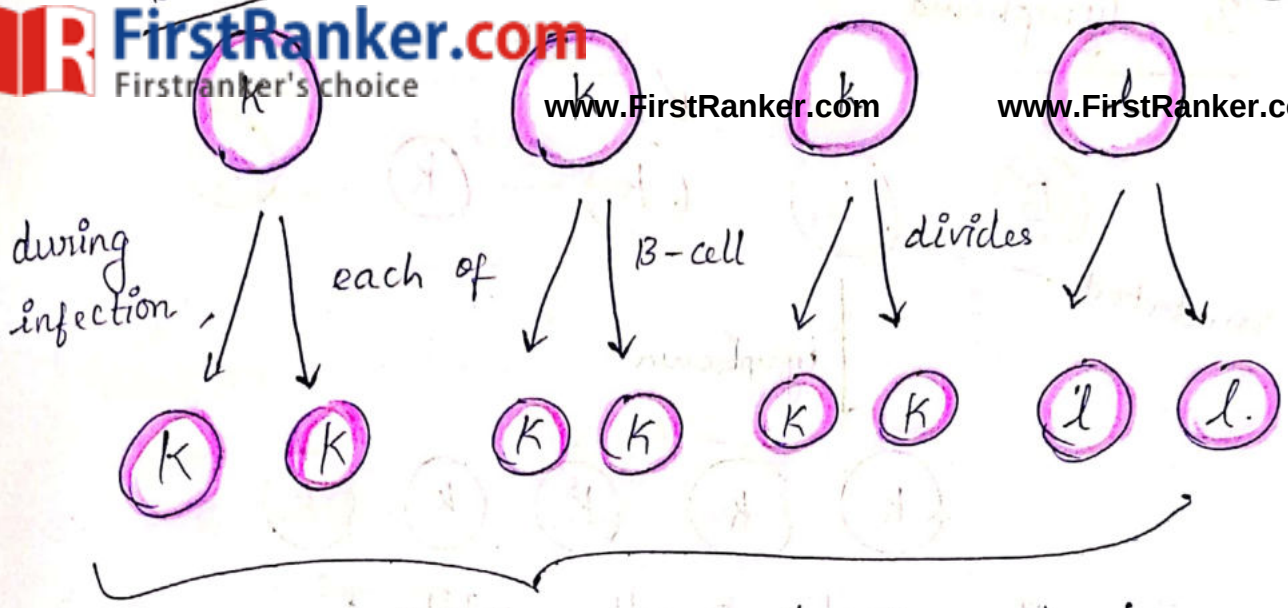
(X) All neoplasia are Monoclonal.

Clonality of B-cells:

* determined by **Ig light chain phenotype.**
  - **Kappa (k)**
  - **Lambda (l)**
* **Normal k : l ⇒ 3 : 1.**

k : l.

- **Normal**
  - ⇓
  - 3 : 1, maintained
  - in polyclonal proliferatn. (Hyperplasia) of B-cell.
  - ⇓
  - ~~during infections~~
- **Neoplasia**
  - Ratio ↑ > 6 : 1 (or) inverted. (1 : 3).
  - ⇓
  - monoclonal (lymphoma)



when infecⁿ occurs, lymph node becomes bigger due to B- cells proliferatⁿ ⇒ "polyclonal" prolifern.

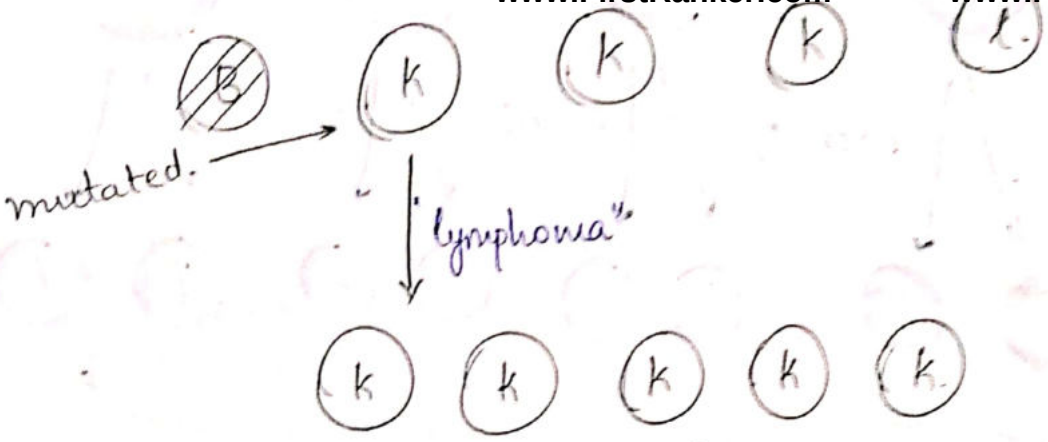
K : λ = 3 : 1 ratio is maintained.

Differential diagnosis in Enlarged L.N.

L.N- Lymph Node

i) Metastatic Cancer.	ii) Reactive Hyper-plasia (during infecⁿ).	iii) Lymphoma
* no-proliferⁿ of lymphocytes.	* proliferⁿ ✓ of lymphocytes * K : λ → 3 : 1	* -do- * K : λ > 6 : 1 (or) inverted.

B-cells.



* all the malig. B-cells will have same Ig light chain phenotype as that of mother cell.

⇒ "Monoclonal."

* Here k:l > 6:1 for eg: 20:1..

Neoplasia:

* Both Benign tumour & malignant tumour are Neoplasia & Both are monoclonal.

* Benign tumour → localized & don't metastasize

* Malig. tumour (aka Cancer) → invade locally & have the potential to metastasize

Note: malig. tumour (cancer) can metastasize in a subset of populat but that doesn't mean it has to metastasize in every single patient.

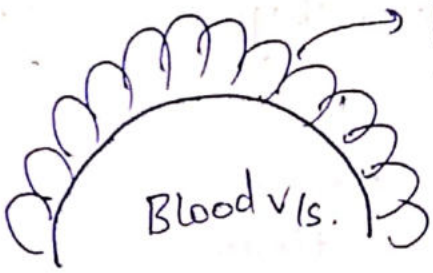
(4) major tissues of human body

- Epithelium → Cells that line surface of the body.
- Mesenchyme → Connective tissue (or) soft tissue. → [fat, blood V/s, bone, cartilage.]
- lymphocyte
- melano-cyte

lineage of Differentiatn.	Benign.	Malignant (aka cancer)
1) Epithelium.	Adenoma (glands.)	Adeno carcinoma
	Papilloma (Finger like structure around a Conn. tissue)	Papillary carcinoma
2) Mesenchyme	lipoma	Lipo-sarcoma
	Angioma	Angio-sarcoma
3) Lymphocyte	Doesn't exist	lymphoma leukemia.
4) Melanocyte	Nevus. (mole) → common name.	Melanoma

finger like/papillary growth of cancer around

A · Connective tissue such as blood v/s.



Epidemiology: (USA).

* Cancer → 2nd leading cause of deaths in BOTH adults & children.

ADULTS

* No. 1 cause of death → Cardiovascular ds.
  No. 2 " " → Cancer
  No. 3 " " → Cerebrovascular ds.

CHILDREN

No. 1 " " " → Accident
. 2 " " → Cancer
. 3 " " → Congenital defects.

---

* excluding squamous cell carcinoma & basal cell carcinoma (SKIN CANCERS).

* Although skin cancers are more common, they rarely metastasize

* they can be easily reveled as they on skin.

* they can be easily treated & don't cause any mortality.

m/c cancers in Adults.

By incidence.	By mortality
* i) Breast / Prostate (♀) (♂)	i) Lung.
ii) Lung	ii) Breast / Prostate (♀) (♂)
iii) Colorectal.	iii) Colorectal.

(X) Note: Easily Δ^{able}. & Easily Curable/preventable Cancer among the Above 3, is "Lung Ca" bcoz the cause is mostly Smoking.

So $R_x \longrightarrow$ "SIMPLY STOP SMOKING"

that it can't be screened early.

That's why, its the ~~no.1~~ most common cause of death among (3).

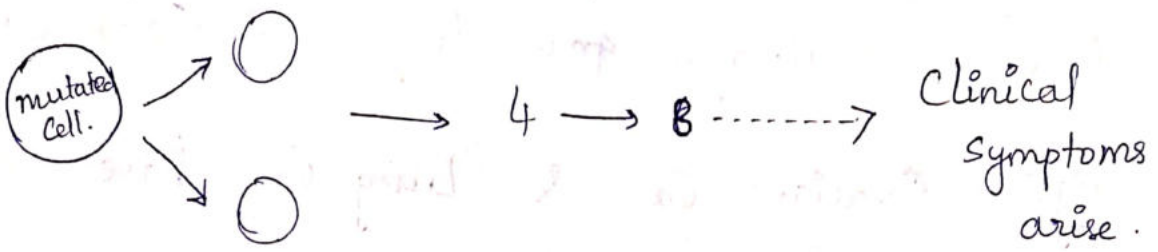
* 5 years survival of a lung Ca pt 30 yrs ago is 15%, even now, its 15%. This is bcoz there are No screening mech. available.

Screening tests:

(i) Breast → Mammogram & physical Examinatn.

(ii) Prostate → PSA (or) Digital Rectal examinatn (Dedated). (DRE).

(iii) Colorectal. → Heme-occult testing in feces (or) Colonoscopy.



~30 divisions occur before earliest symptoms arise.

Dysplasia → single mutated cell. → 2 → 4 → 8 ------> Clinical Symptoms.

mutations ↑ bcoz DNA are not being repaired : check systems are not working.

* Cancers that are not detected (or) doesn't produce symptoms until late in ds will have undergone > 30 divisions & many mutations *

↓ why not detected early?

Example : Ovarian cancer ⇒ Large empty "space" to grow without any symptoms.

Lung Cancer ⇒ Lung has Huge "reserve" so doesn't produce Any symptoms

have a poor prognosis bcoz they have (i) already grown large (ii) many mutations

eg: Ovarian Ca & Lung Ca have poor prognosis.

* Pancreatic Ca:

Pancreas also has a large space to grow & have a huge reserve before tissue damage can reveal itself.

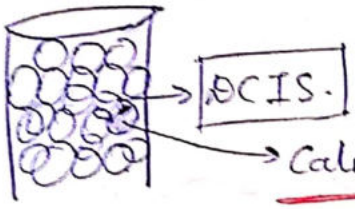
---

② Goals of screening

- Catch dysplasia before it becomes Carcinoma.
  - eg: PAP smear
  - ⇓
  - we look for CIN → dysplasia not cancer
  - CIN - Cervical Interepithelial Neoplasia.
  - CIN (dysplasia) —untreated→ Cervical Cancer)
  - ⇓ has mutations but reversible.
  - ⇓ irreversible.
- detect carcinoma before clinical symp. arise
  - eg: Mammography - breast Ca
  - PSA & DRE - prostate Ca
  - Haemoccult test & Colonoscopy - Colo-rectal Ca.

To find out DCIS (Ductus Carcinoma Insitu) before it invades nearby blood V/s / Lymph V/s

⇓



Duct

⇓

identified by mammography

To find out Ca. of size ~ 1 cm. which is not palpable (or) IO clinically (phy. exam)

⇓

Normally 2cm lump are alone identif by phy. examn.

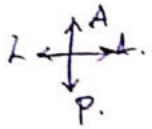
⇓

(~1cm screened earlier).

Prostate Ca:

prostate cancer.

C.S. of prostate



⇓

post. peripheral aspect

~~silently~~ grows without any C/F.

⇓

That why PSA. & DRE can IO this early

doesn't produce any Urinary Symp. like BPH. bcoz Urethra is in centre

But late prostate Ca Can compress urethra

Hemoccult test & Colonoscopy:

Colonic Adenoma → Colonic – Carcinoma

⇓

* Can be ID in Colonoscopy & can be easily they won't develop into Ca.
* Also Colonoscopy helps to find C/F appears.