

# DIABETIC RETINOPATHY

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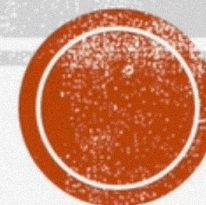
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# WHAT , WHEN AND HOW OF DIABETIC RETINOPATHY?

## ETIOPATHOGENESIS



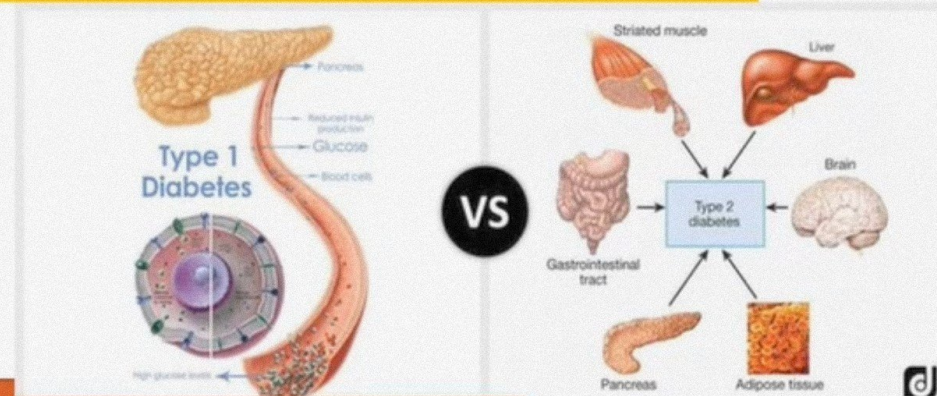
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# WHAT IS DIABETIC RETINOPATHY

- Diabetic retinopathy is a vascular complication of diabetes.
- Dysfunction of the retinal bloodvessels due to chronic hyperglycemia



After 20 years of diabetes

**99%** of patients with **TYPE 1 DM**

**60%** of patients with **TYPE 2 DM**

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# RISK FACTORS FOR DEVELOPING DR?

- Duration of diabetes
- Poor control of diabetes
- Pregnancy
- Hypertension,
- Nephropathy
- Miscellaneous like hyperlipidemia, smoking, cataract surgery, obesity, and anemia

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# GOOD CONTROL OF BLOOD SUGAR LEVELS



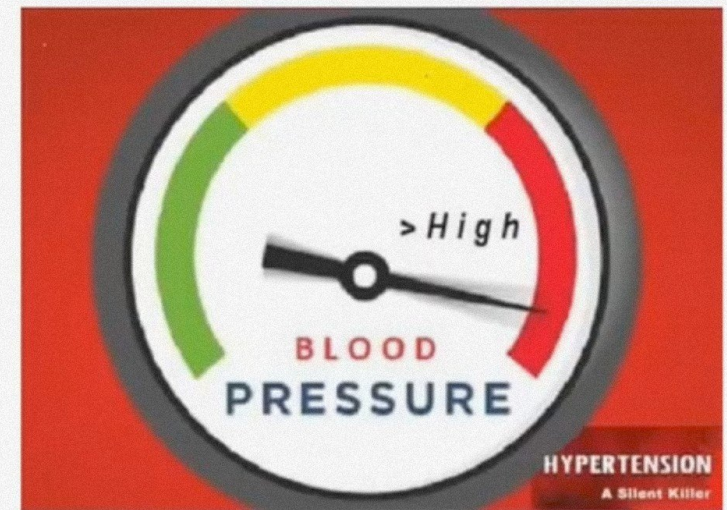
- **10% decrease in HbA1c --> 40% reduction in risk**





# CONTROL OF HYPERTENSION

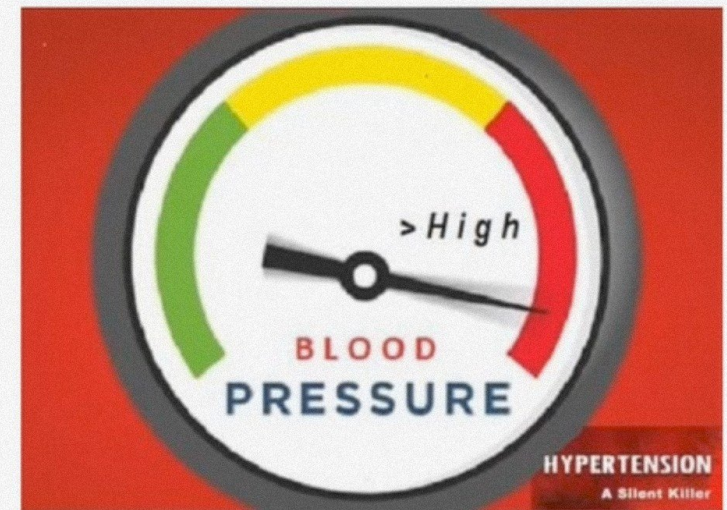
- Especially important in type 2 DM





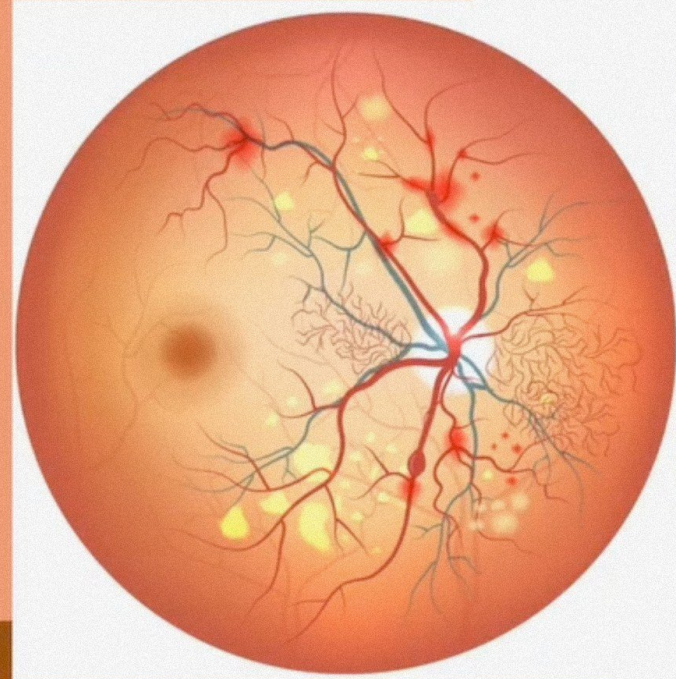
# CONTROL OF HYPERTENSION

- Especially important in type 2 DM
- HTN :- **37%** REDUCTION IN MICROVASCULAR COMPLICATIONS OF DM





# WHAT HAPPENS INSIDE VESSELS IN DIABETIC RETINOPATHY



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(A)

Glucose



Hexokinase/glucokinase

Glucose-6-phosphate  $\rightarrow$   $\text{H}_2\text{O} + \text{CO}_2$

ADP + Pi

ATP



Glycogen  $\leftarrow$

$\rightarrow$  Pentose phosphate pathway



Fatty acids

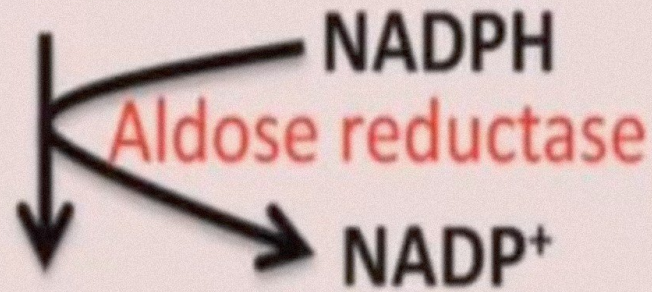
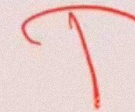
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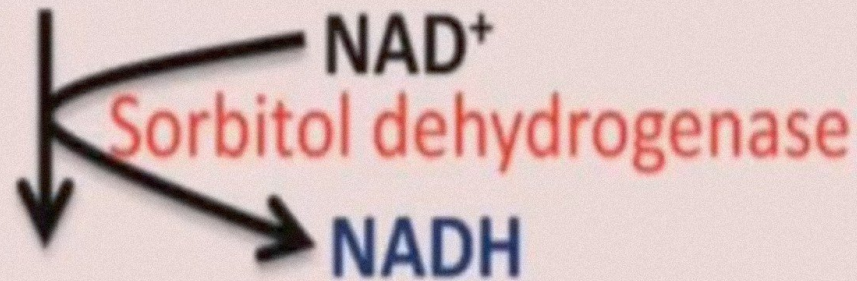


3)

Glucose



Sorbitol



Fructose

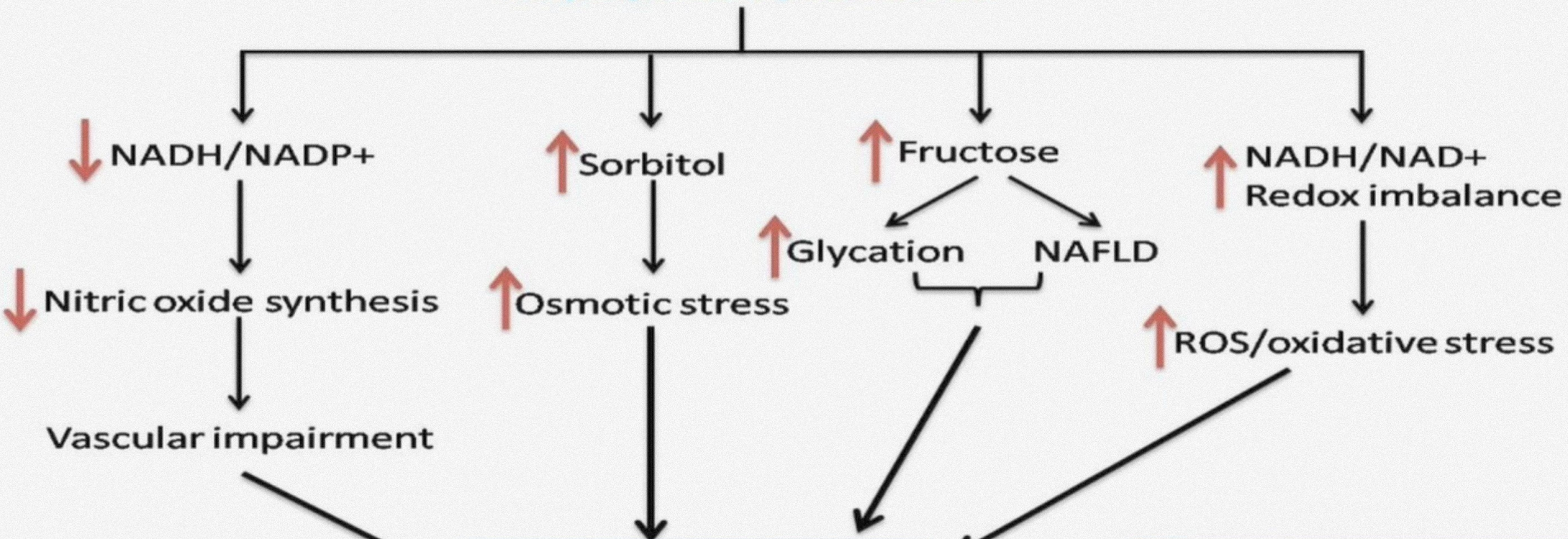
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# Persistent hyperglycemia

## Polyol pathway activation



**Diabetic complications:**  
Retinopathy  
Nephropathy  
Neuropathy

↓ Indicates a decrease  
↑ Indicates an increase

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**INFLAMMATION  
OXIDATIVE  
STRESS**

00:08:26





**Cytokines and chemokines**

**INFLAMMATION  
OXIDATIVE  
STRESS**

*WBC  
leuko cytes*



*Leukostasis*

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# DIABETES

Systemic Factor Control

Hypertension

HYPERGLYCEMIA

Dyslipidemia

Biochemical and Molecular Abnormalities  
(Polyol, AGE, PKC & Hexosamine Pathway Activation)

Vascular Dysfunction

HYPOXIA

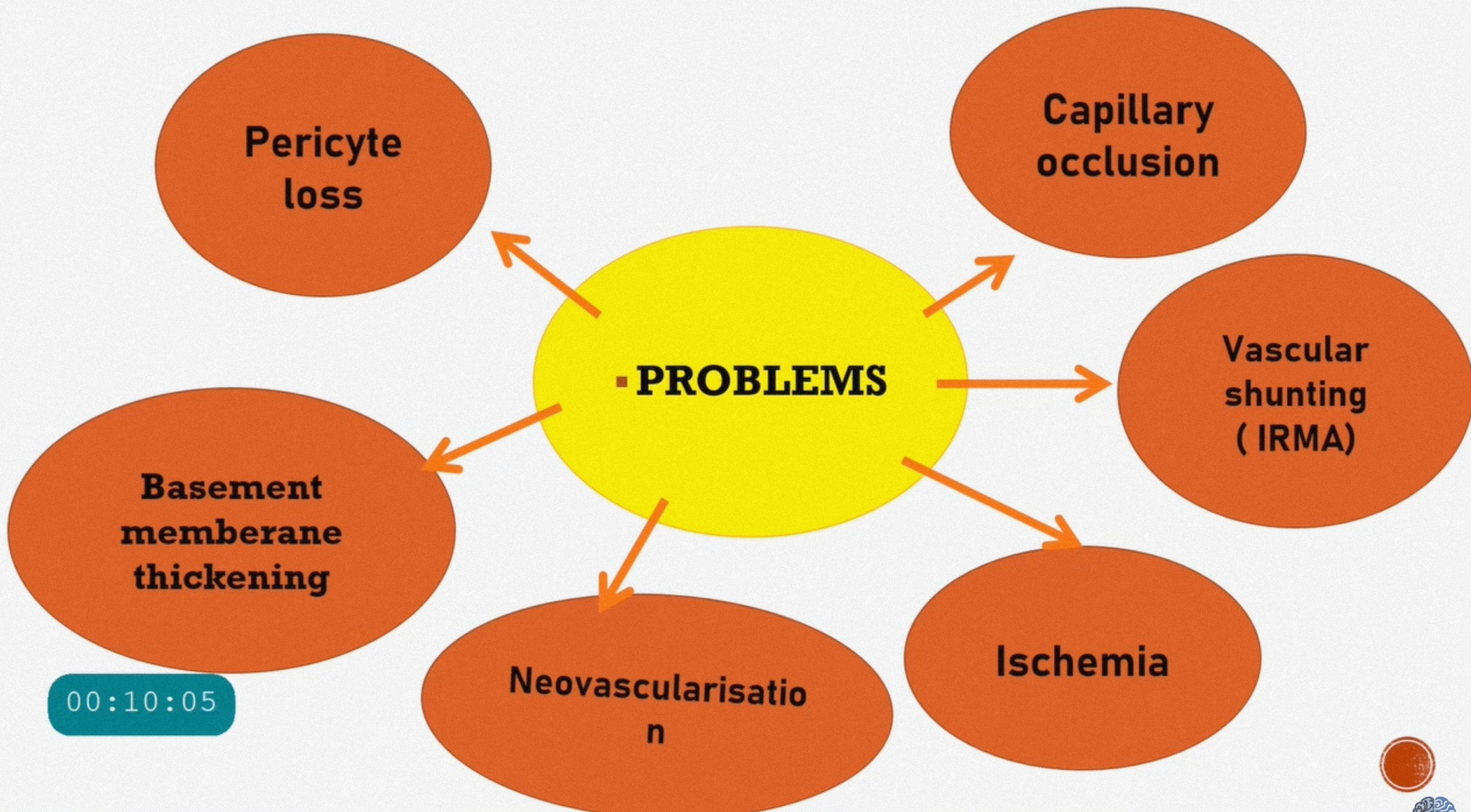
ROS

INFLAMMATION

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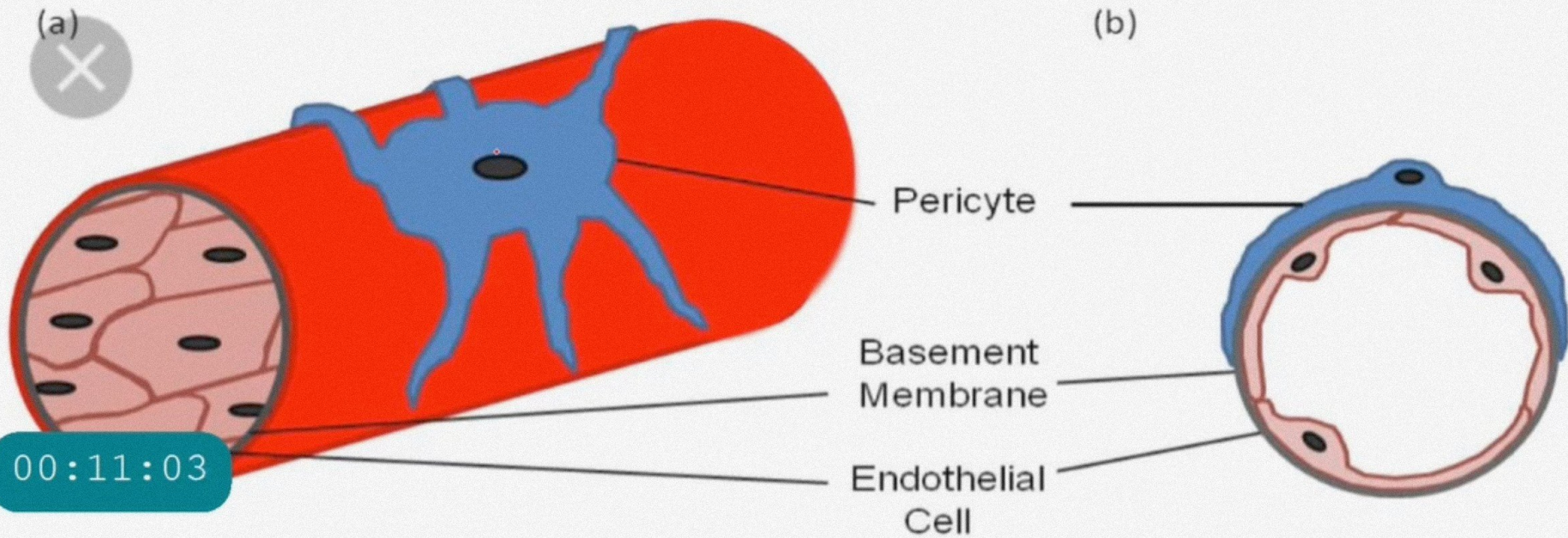


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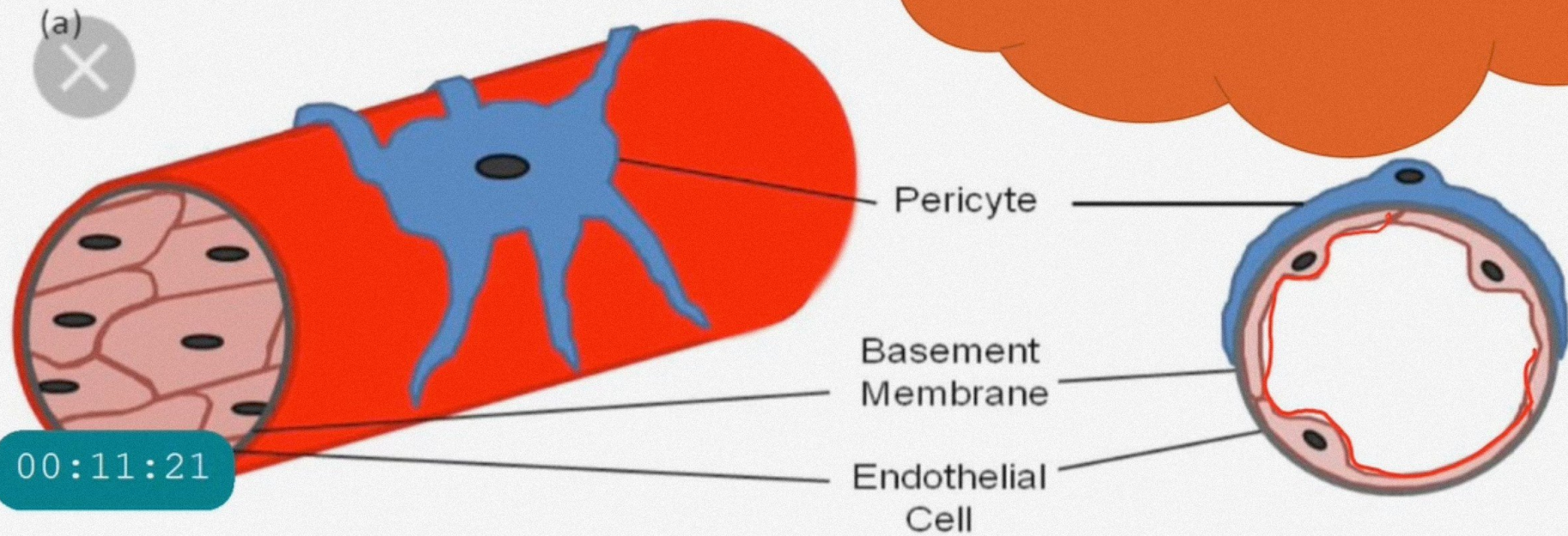
# LOSS OF PERICYTES





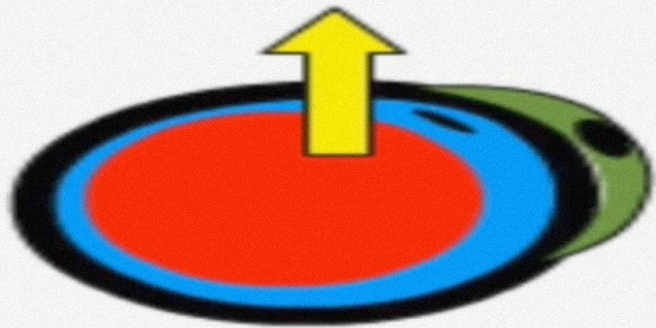
# LOSS OF PERICYTES

Abnormal pericytic deposition of material due to sorbitol or advanced glycation end products (AGEs) through the aldose reductase polyol pathway





# THICKENING OF BASEMENT MEMBRANE



reduced  
blood flow;  
thickened  
basement  
membrane

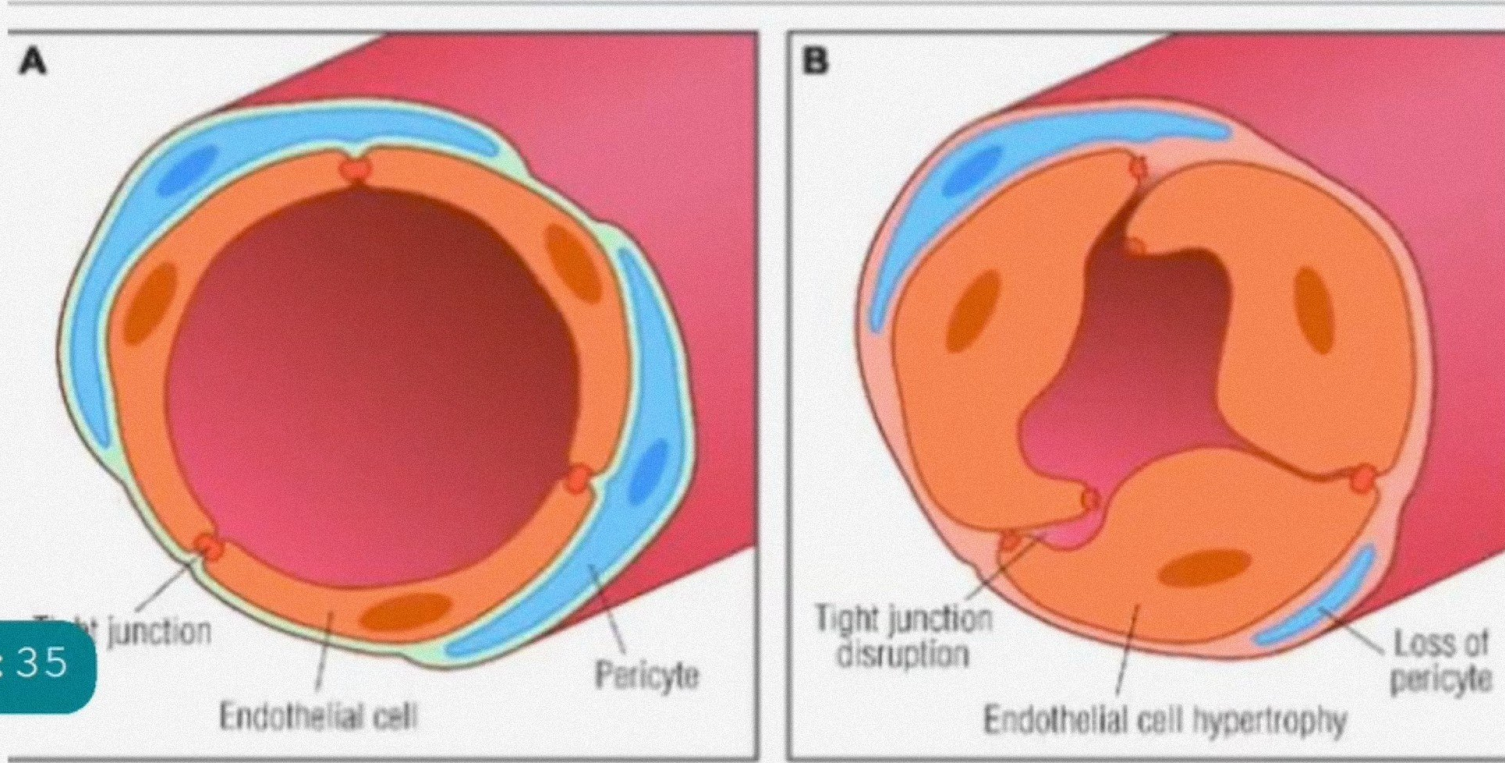
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- Normal thickening is 0.5 microns
- As much as 5 folds, which results in impairment of diffusion & transfer of nutrients & metabolites





# TIGHT JUNCTION AND ENDOTHELIUM DAMAGE



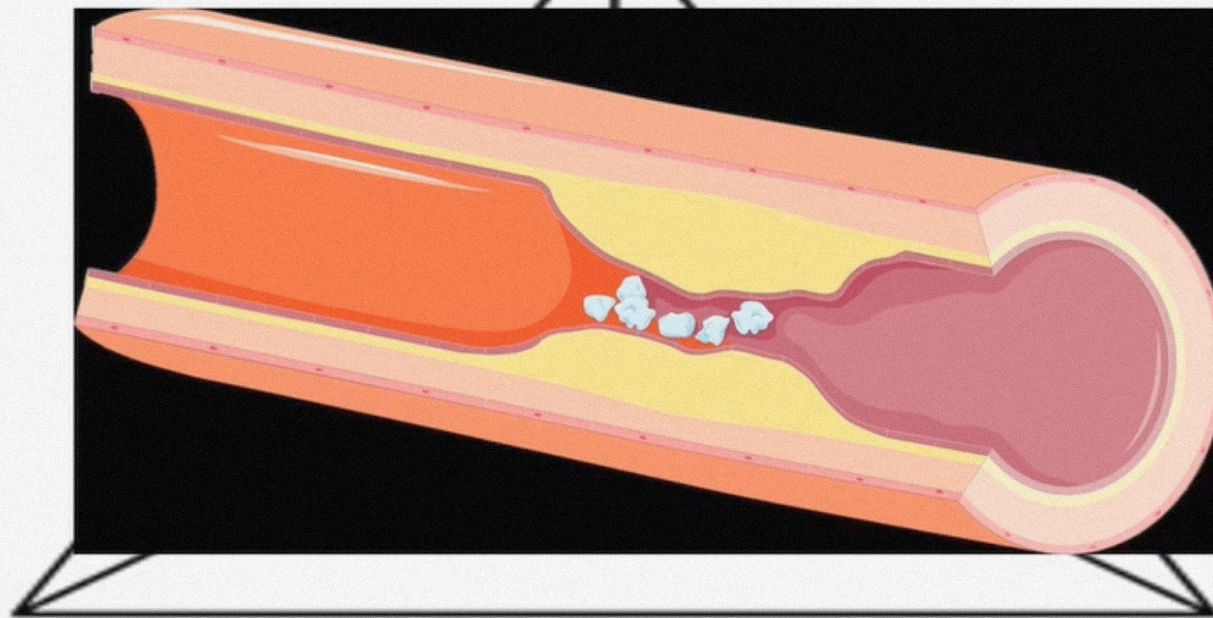
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# Virchow's Triad

Stasis of blood flow



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Endothelial injury

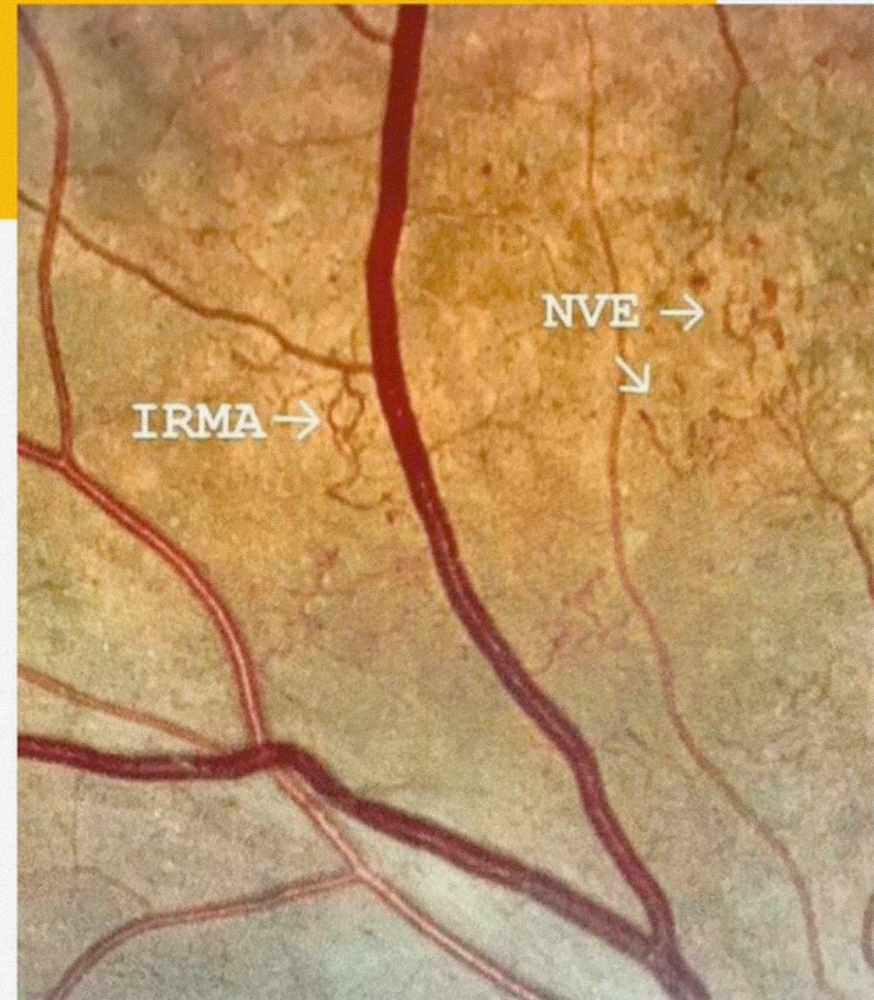
Hypercoagulability





# OCCLUSION AND ISCHEMIA

- When a focal area loses its perfusion, the overlying retina suffers acute ischaemic changes
- These occlusions ophthalmoscopically appears as cotton-wool spots (CWS).
- Microvascular dilatation is often found near areas of capillary obliteration & is called **intraretinal microvascular abnormalities. (IRMA)**



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# VESSEL ABNORMALITY

- Venous beading
- Microaneurysm

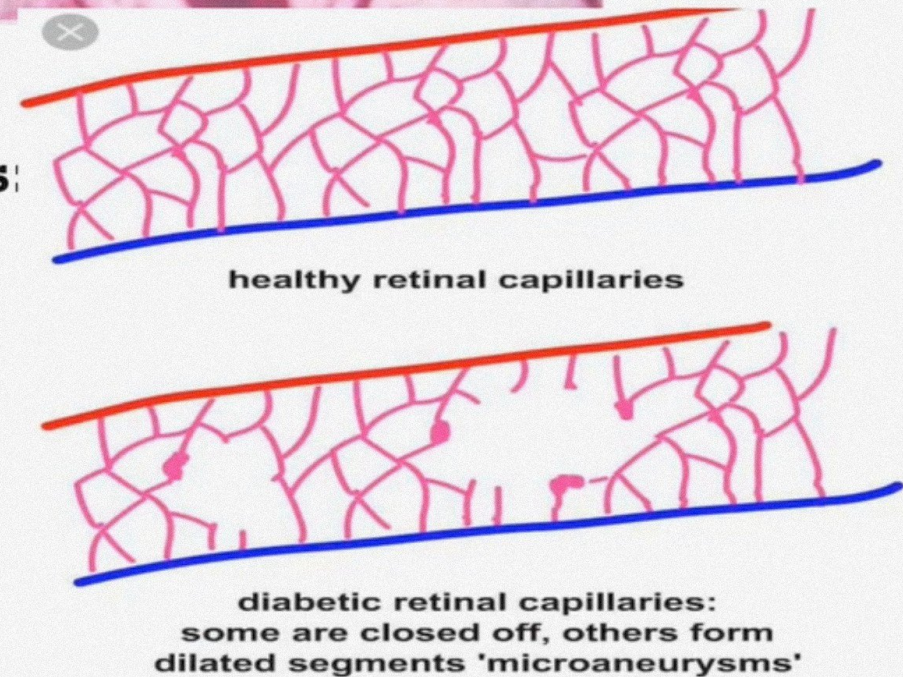


## Mechanism of formation of Microaneurysms:

Pericyte loss –local weakening

Alteration of the capillary BM

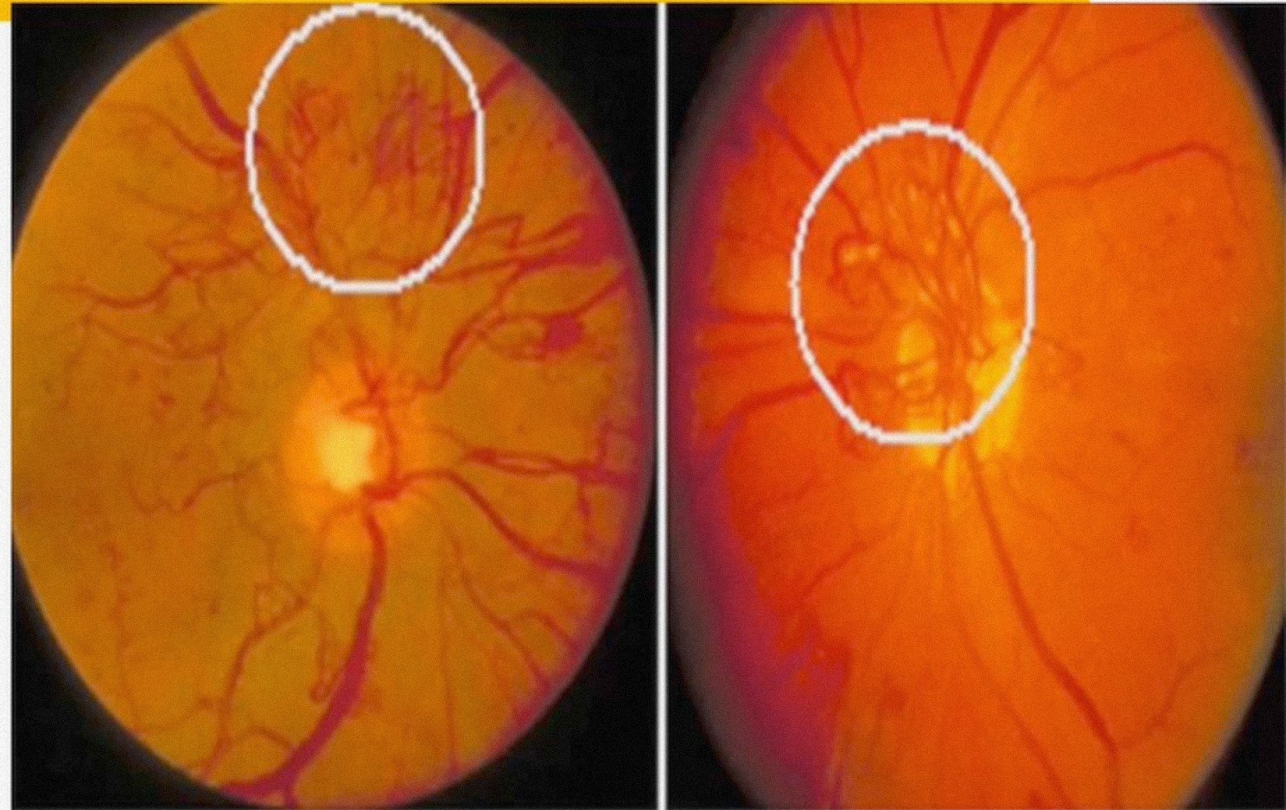
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# NEOVASCULARISATION

- Vascular endothelial growth factor (VEGF),
- Platelet-derived growth factor
- Hepatocyte growth factor.



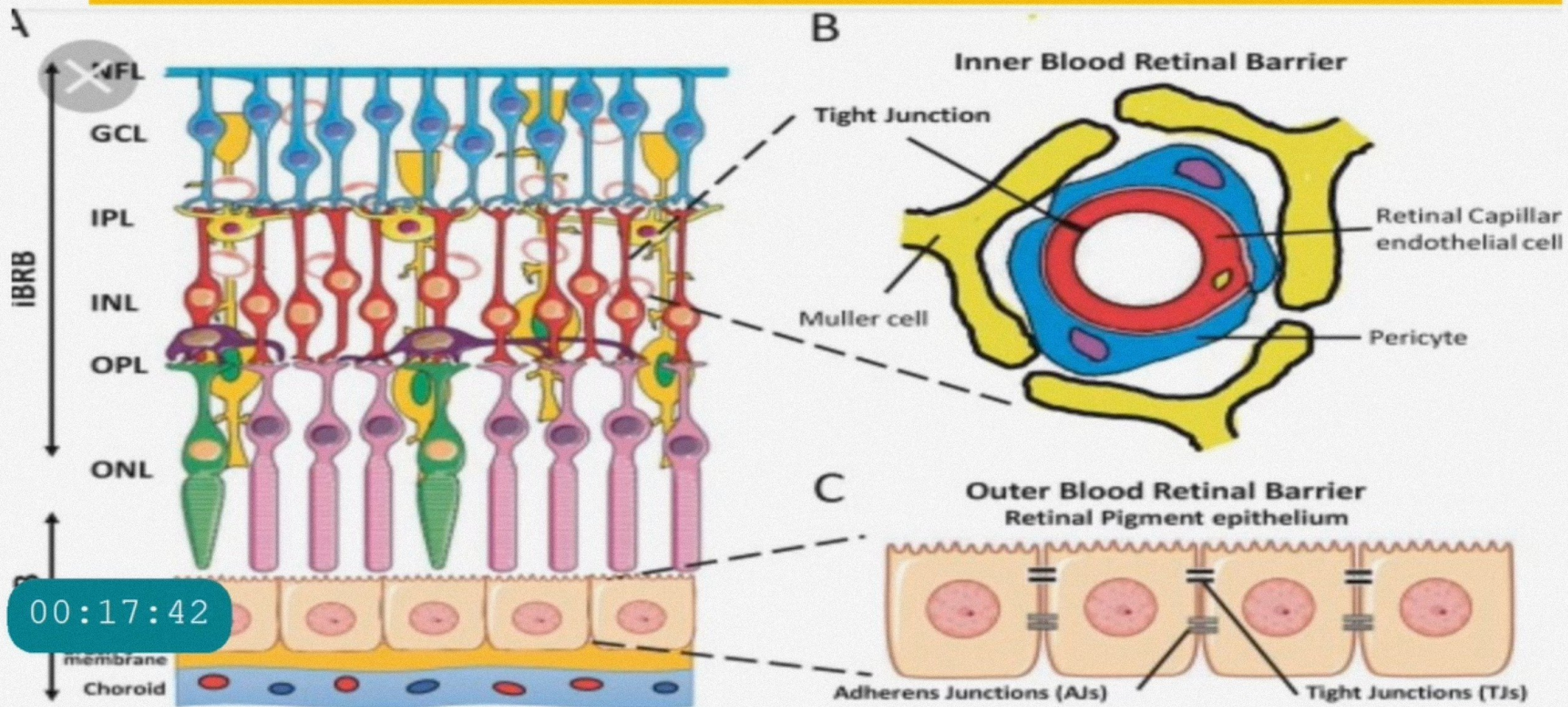
(a) New vessels elsewhere (NVE) (b) New vessels on disc (NVD)

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# BLOOD RETINAL BARRIER





**Endothelial Cell  
Junction Breakdown**

**Pericyte  
Loss**

**Thickening of  
BM**

**Leukostasis**

**Alteration of Blood-Retinal Barrier**

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# DIABETIC MACULAR EDEMA

↓  
Increased vascular permeability

↓  
**Diabetic  
Macular  
Edema**

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