

# Εκτίμηση της Νεφρικής Λειτουργίας

## ΟΞΕΙΑ ΝΕΦΡΙΚΗ ΑΝΕΠΑΡΚΕΙΑ



Δημήτριος Β. Βλαχάκος

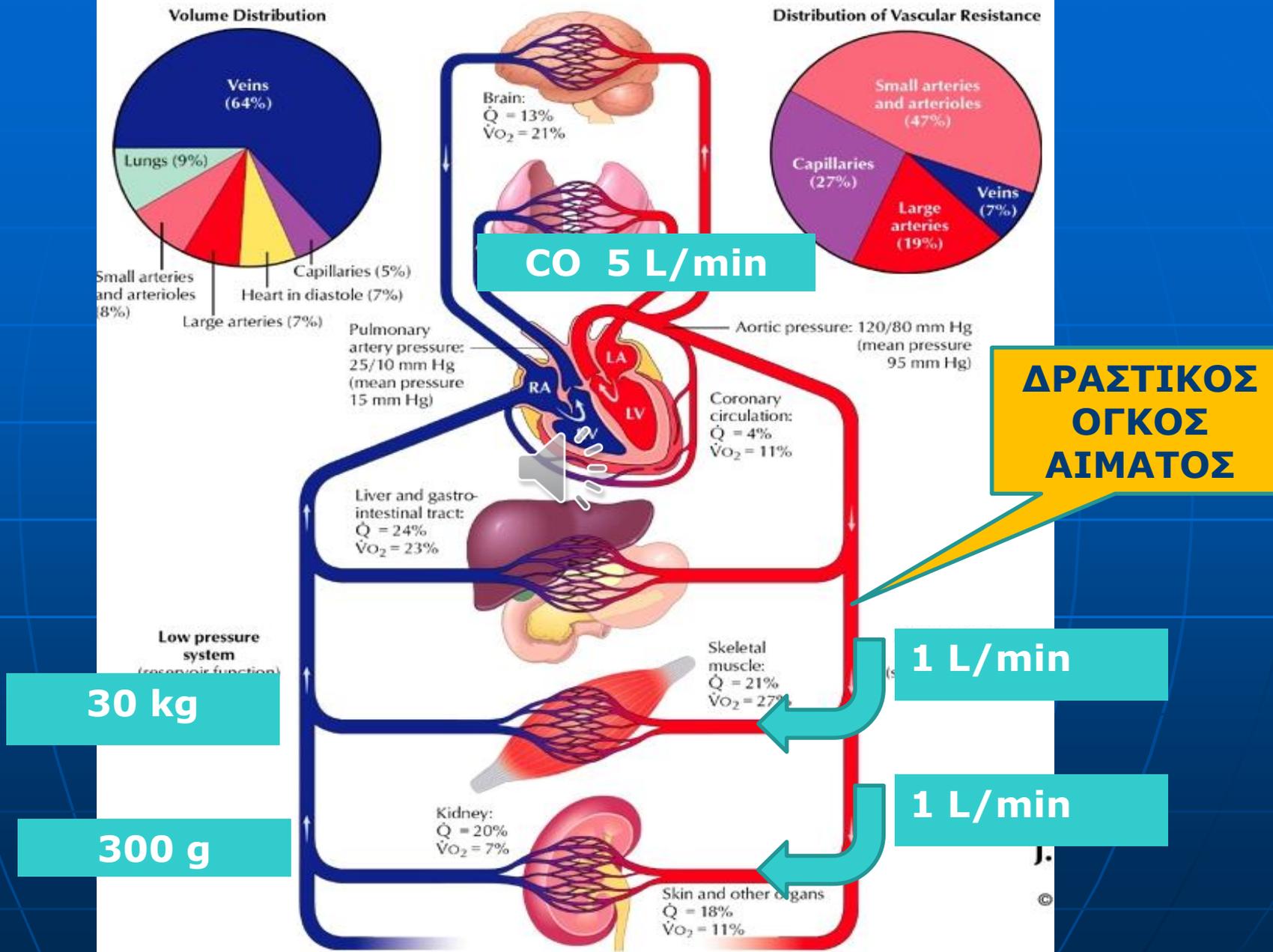
Καθηγητής Παθολογίας - Νεφρολογίας

Υπεύθυνος Νεφρολογικής Μονάδας

Β' Προπαιδευτική Παθολογική Κλινική

Πανεπιστημιακό Γενικό Νοσοκομείο «ΑΤΤΙΚΟΝ»

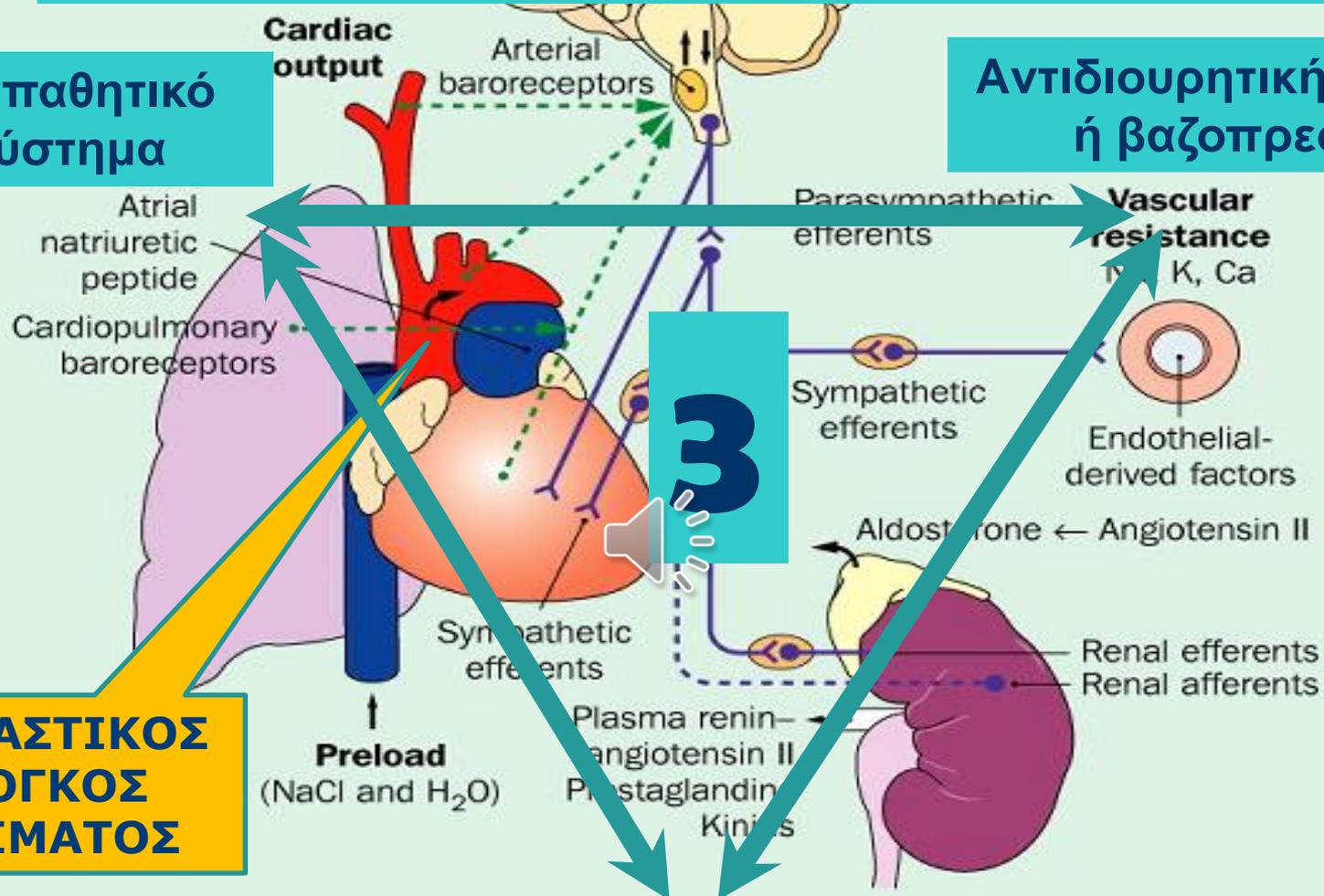
# Overview of the Cardiovascular System



# ΑΠ = ΚΛΟΑ x Αντιστάσεις

Συμπαθητικό  
Σύστημα

Αντιδιουρητική ορμόνη  
ή βαζοπρεσίνη

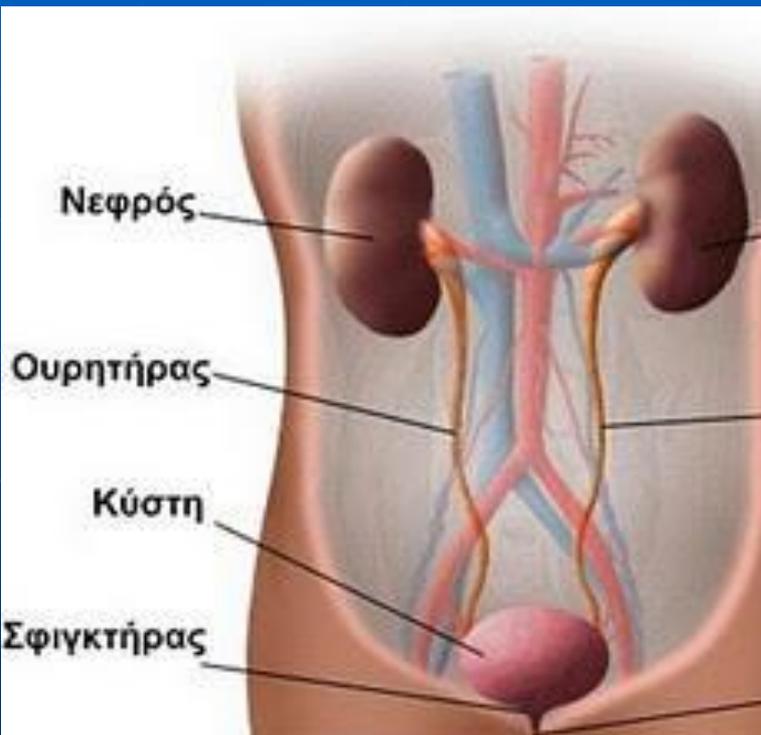


ΔΡΑΣΤΙΚΟΣ  
ΟΓΚΟΣ  
ΑΙΜΑΤΟΣ

Renin- Angiotensin-  
Aldosterone System

tion of  
H<sub>2</sub>O

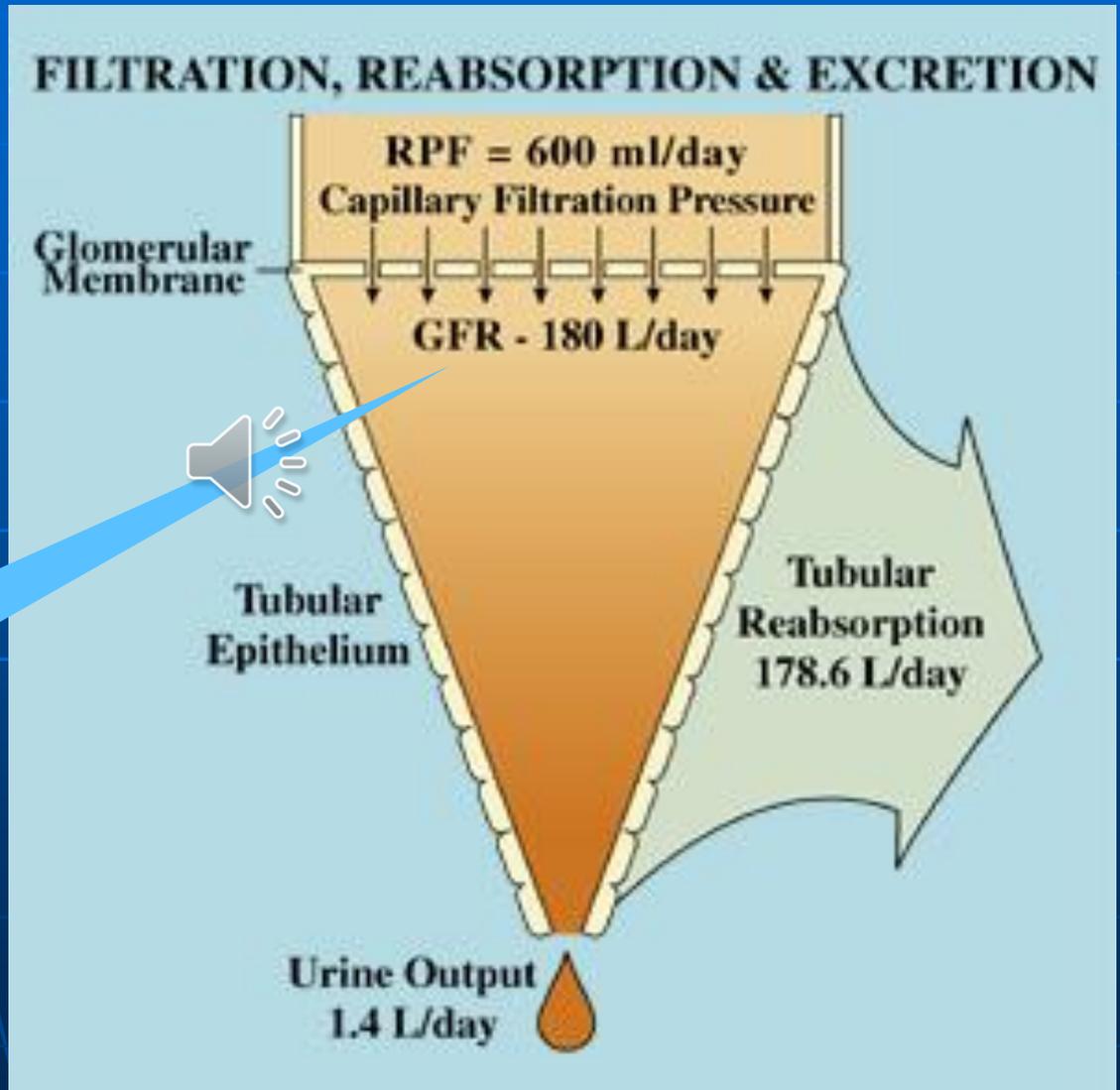
# Οι νεφροί, ως εξωκρινείς και ενδοκρινείς αδένες



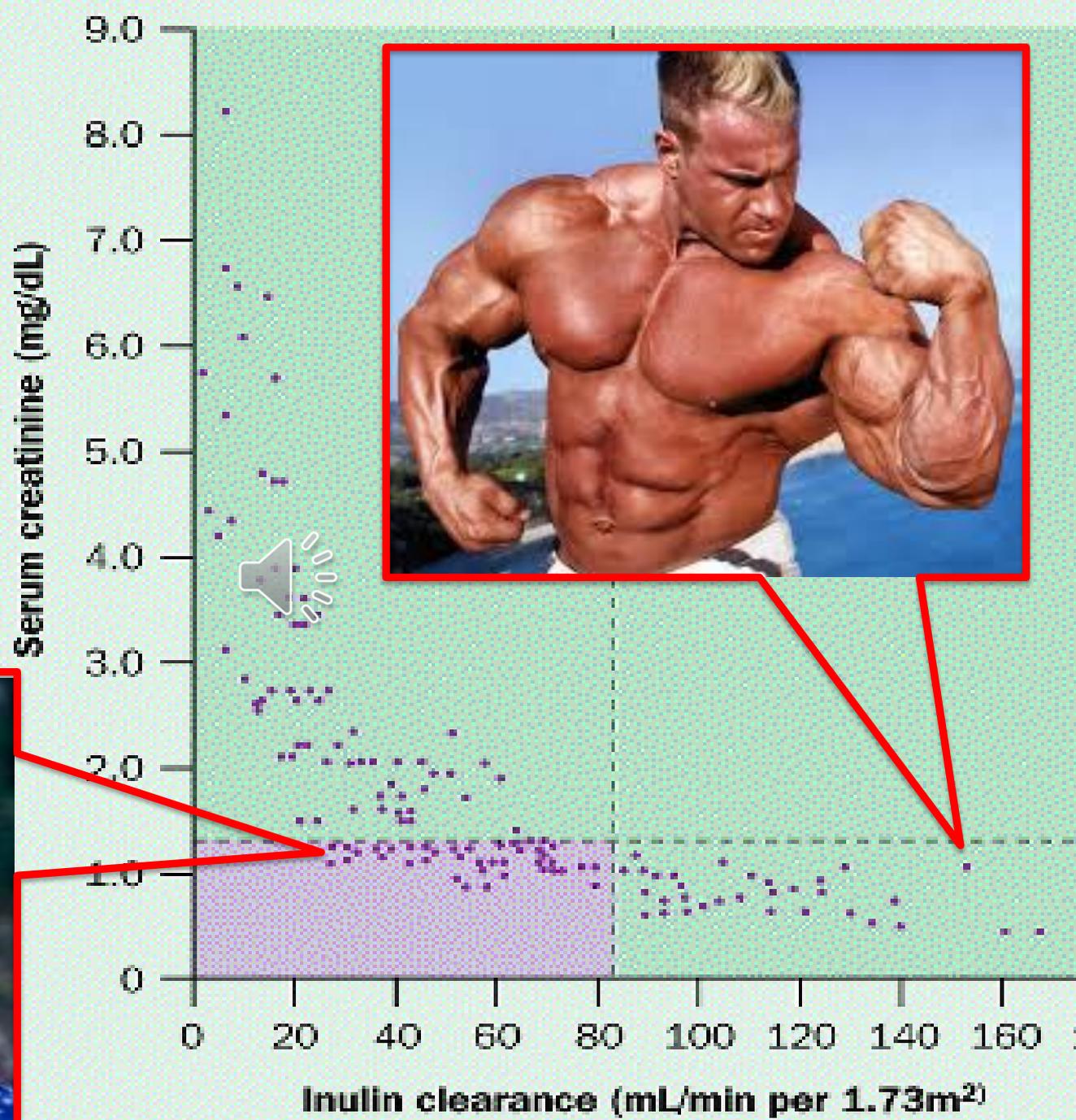
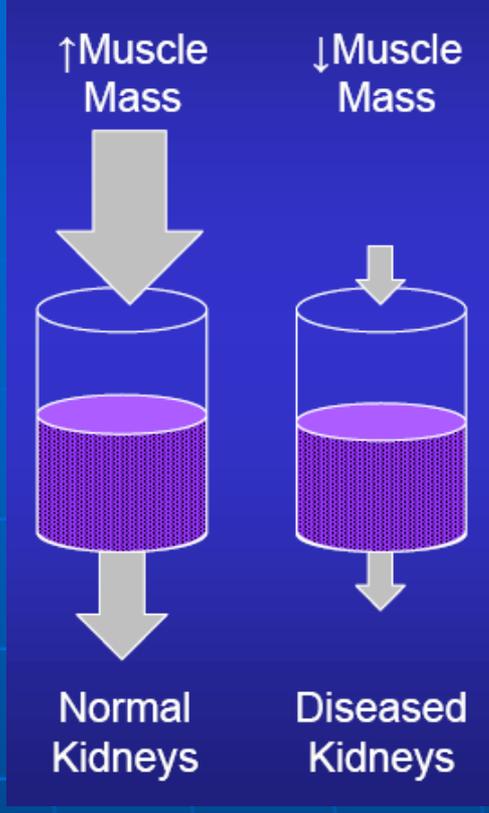
## Λειτουργίες Νεφρών

- Παραγωγή ούρων, αποβολή άχρηστων προϊόντων μεταβολισμού, διαφόρων τοξικών και φαρμάκων
- Ρύθμιση ισοζυγίου ύδατος και ηλεκτρολυτών
- Ρύθμιση οξεοβασικής ισορροπίας (αποβολή  $H^+$  και αποβολή ή παραγωγή διττανθρακικών)
- Μεταβολική και ορμονική δραστηριότητα (ενεργοποίηση βιταμίνης D, παραγωγή ερυθροποιητίνης, ρενίνης) κ.ά

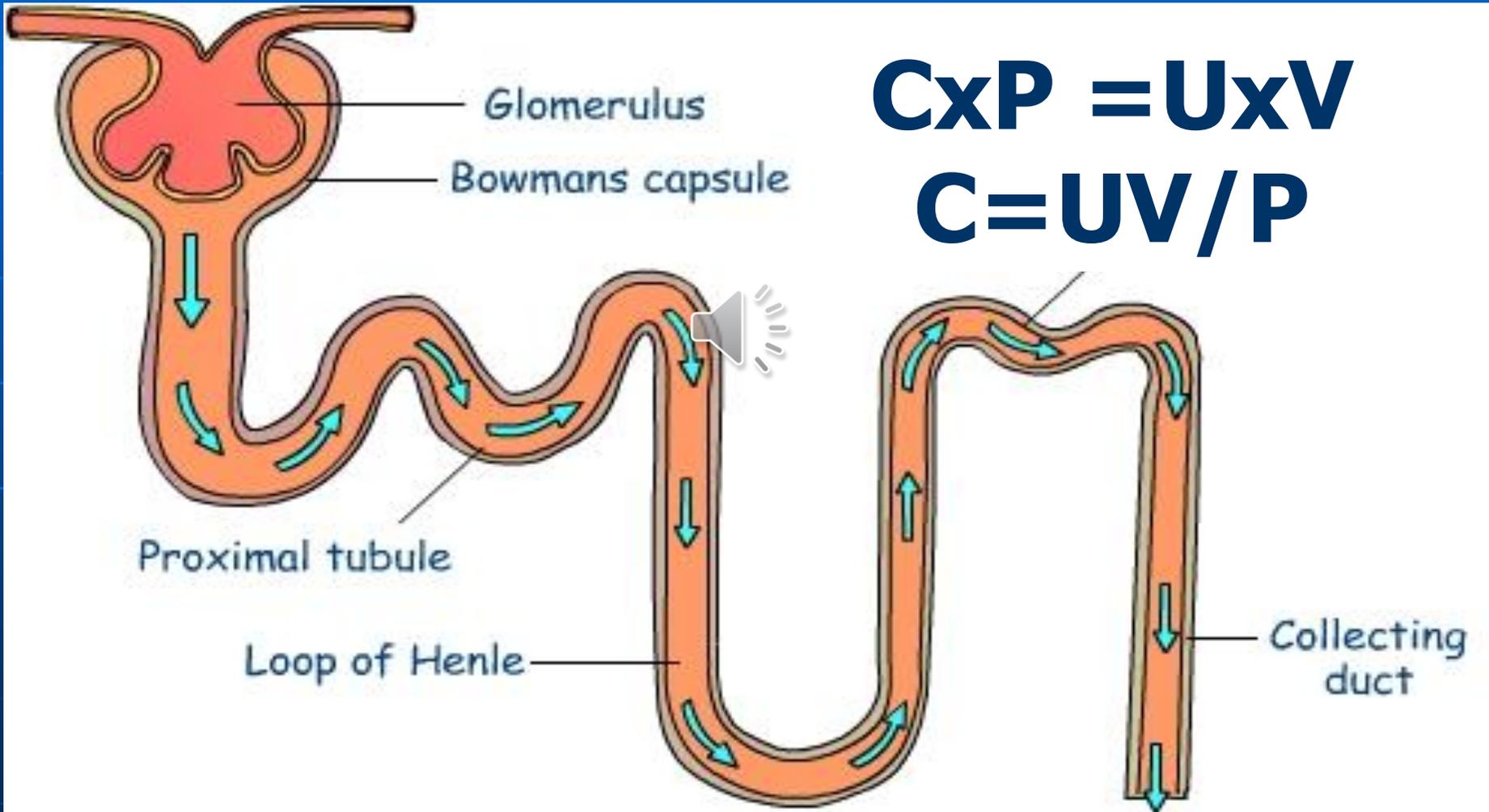
# Η έννοια της κάθαρσης



**GFR = παροχή  
πρόουρου  
125 ml/min**



# Η μέτρηση της κάθαρσης

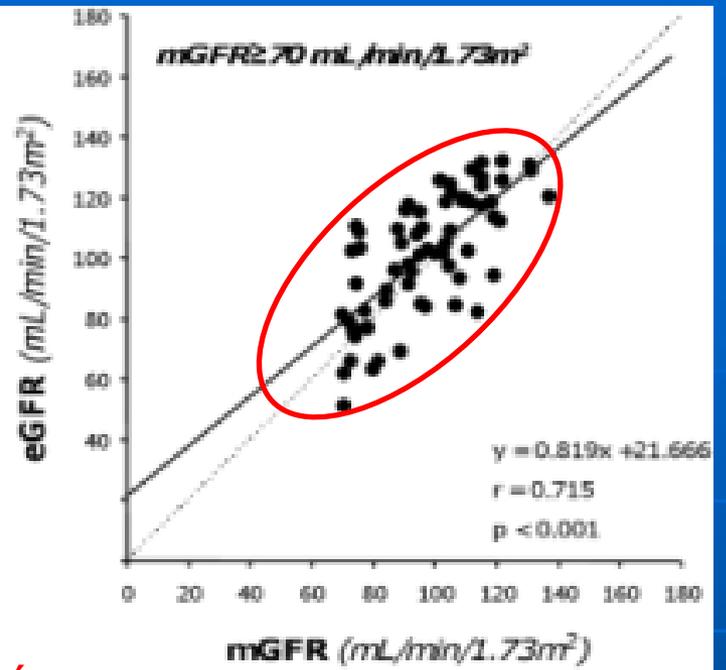
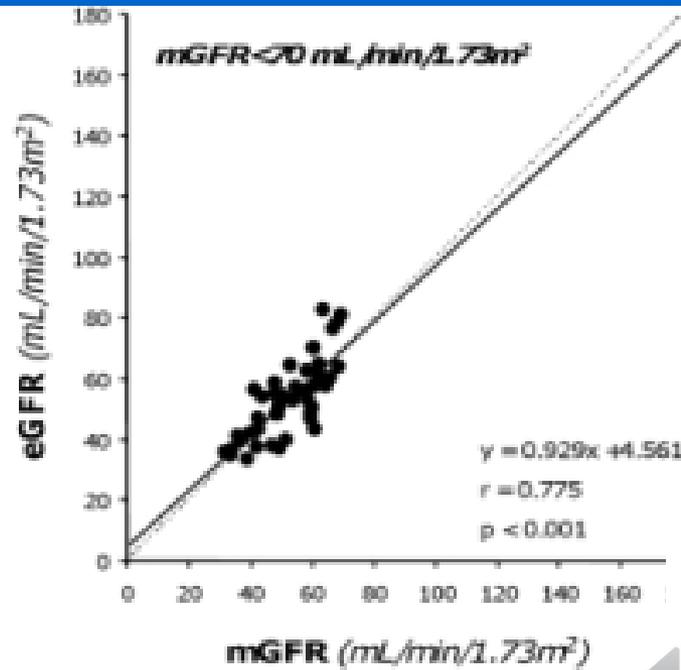


# Τύπος των Cocroft and Gauld

(140 - ηλικία) X Βάρος σε Kg

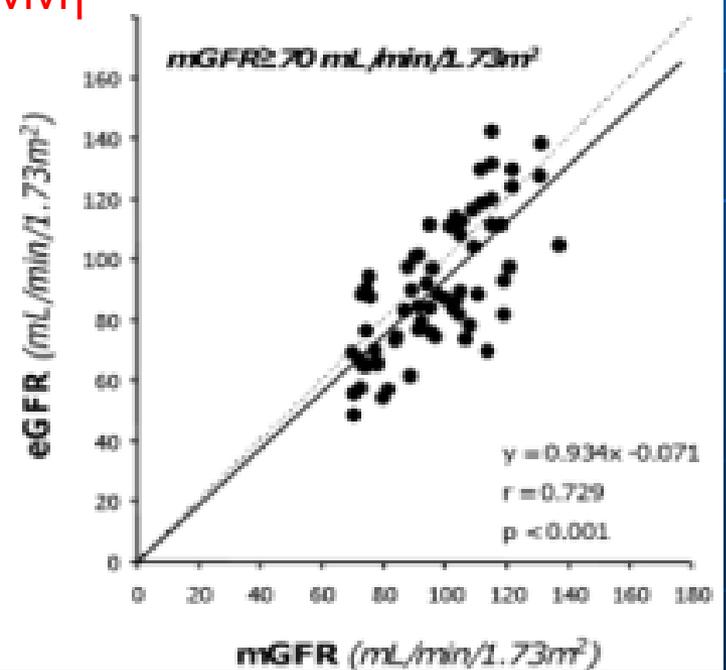
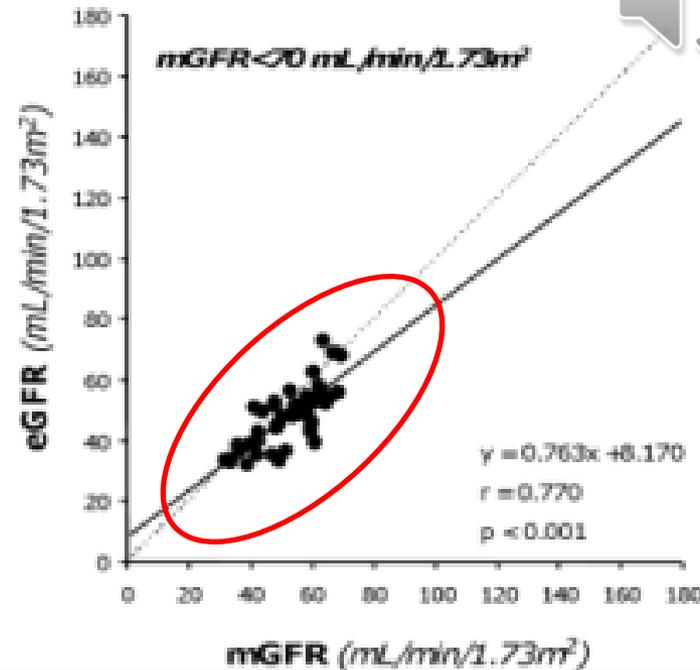
(72  ή 85 ) X Κρεατ ορού

# CKD-Epi



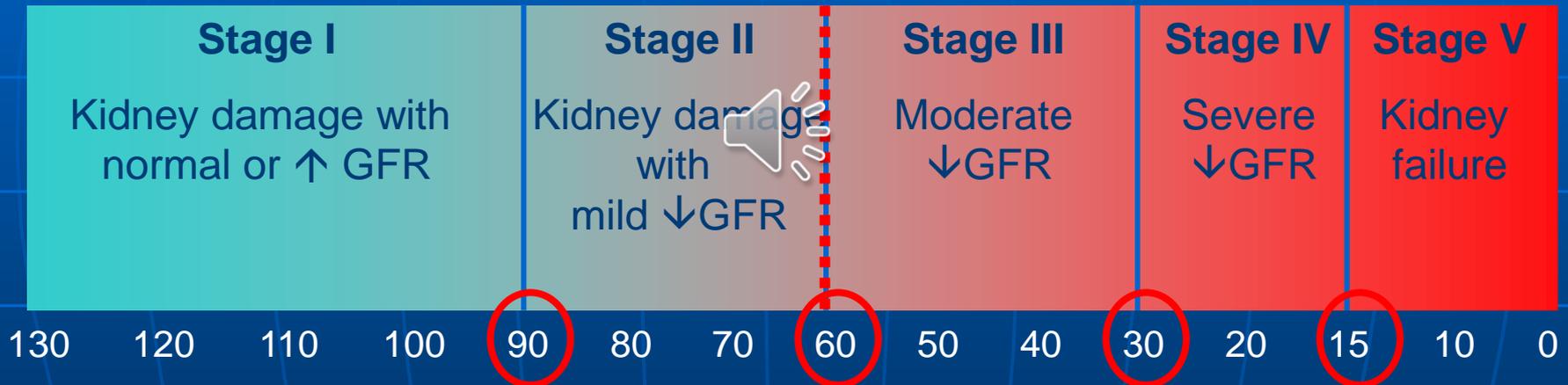
- Ηλικία
- Φύλο
- Φυλή
- Κρεατινίνη

# aMDRD



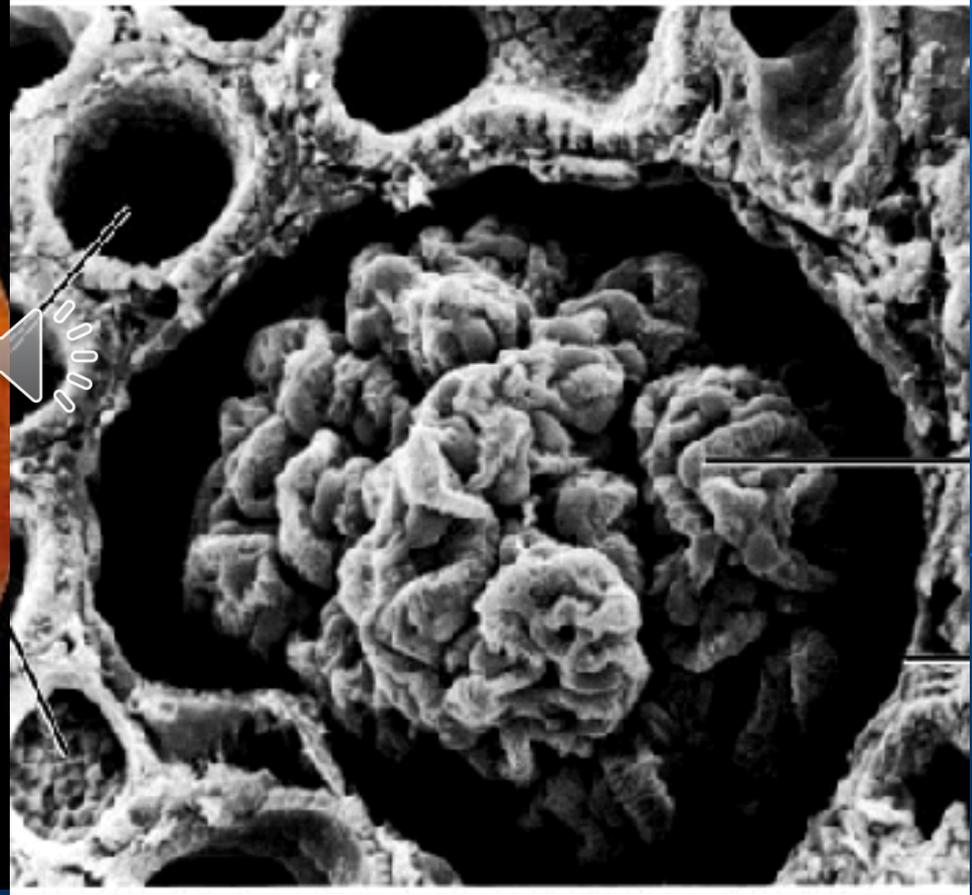
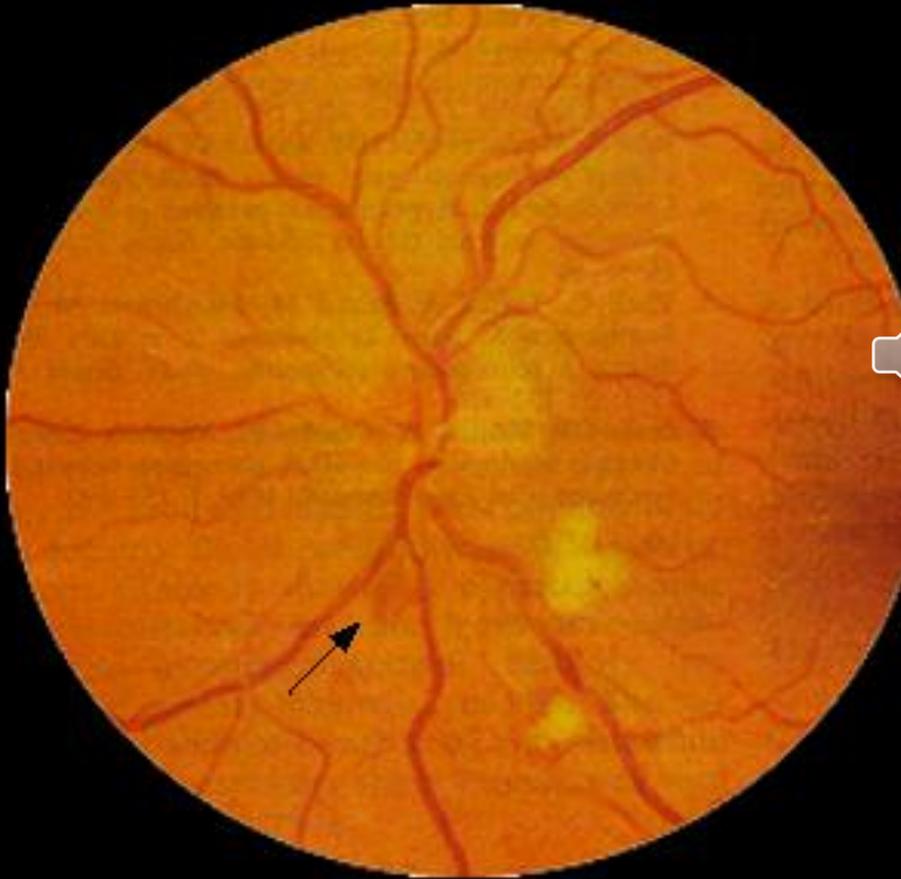
**<60 ml/min για > 3 μήνες**

**Βλάβη**      **Νόσος**



**Glomerular filtration rate (mL/min/1.73m<sup>2</sup>)**

# Μικροαγγειοπάθεια



Parameter	Normal	Micro-albuminuria	Macro-albuminuria
Urine AER (μg/min)	< 20	20 - 200	>200
Urine AER (mg/24h)	< 30	30 - 300	>300
Urine albumin/Cr# ratio (mg/gm)	< 30	30 - 300	>300



**Βλάβη**

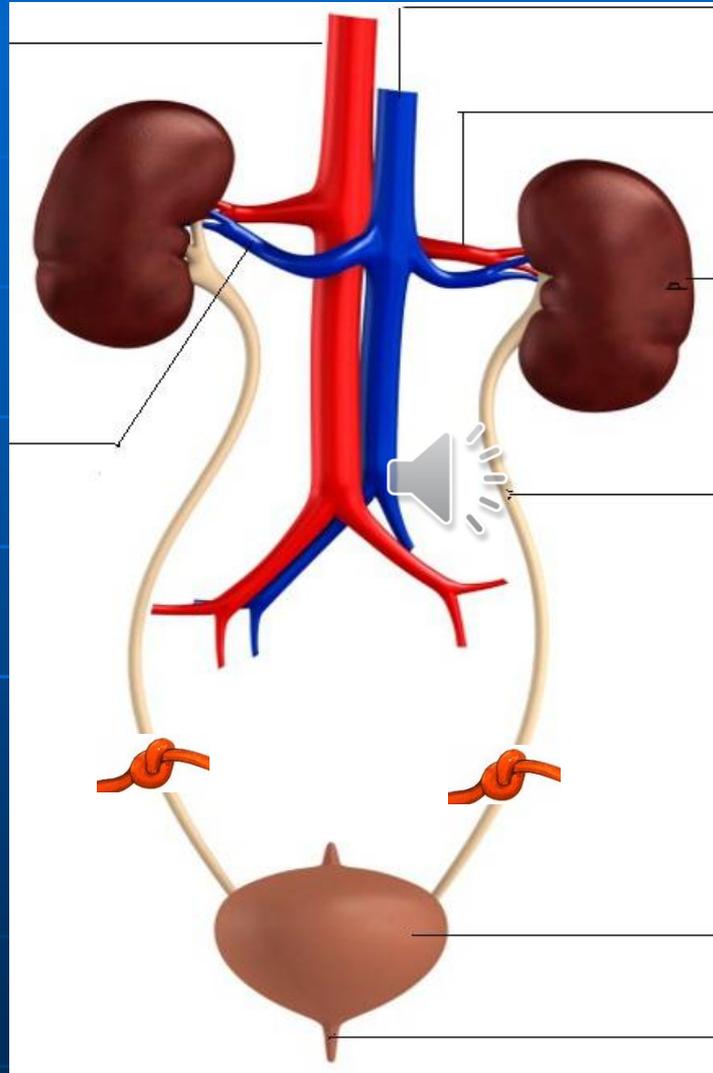
**Νόσος**

AER=Albumin excretion rate

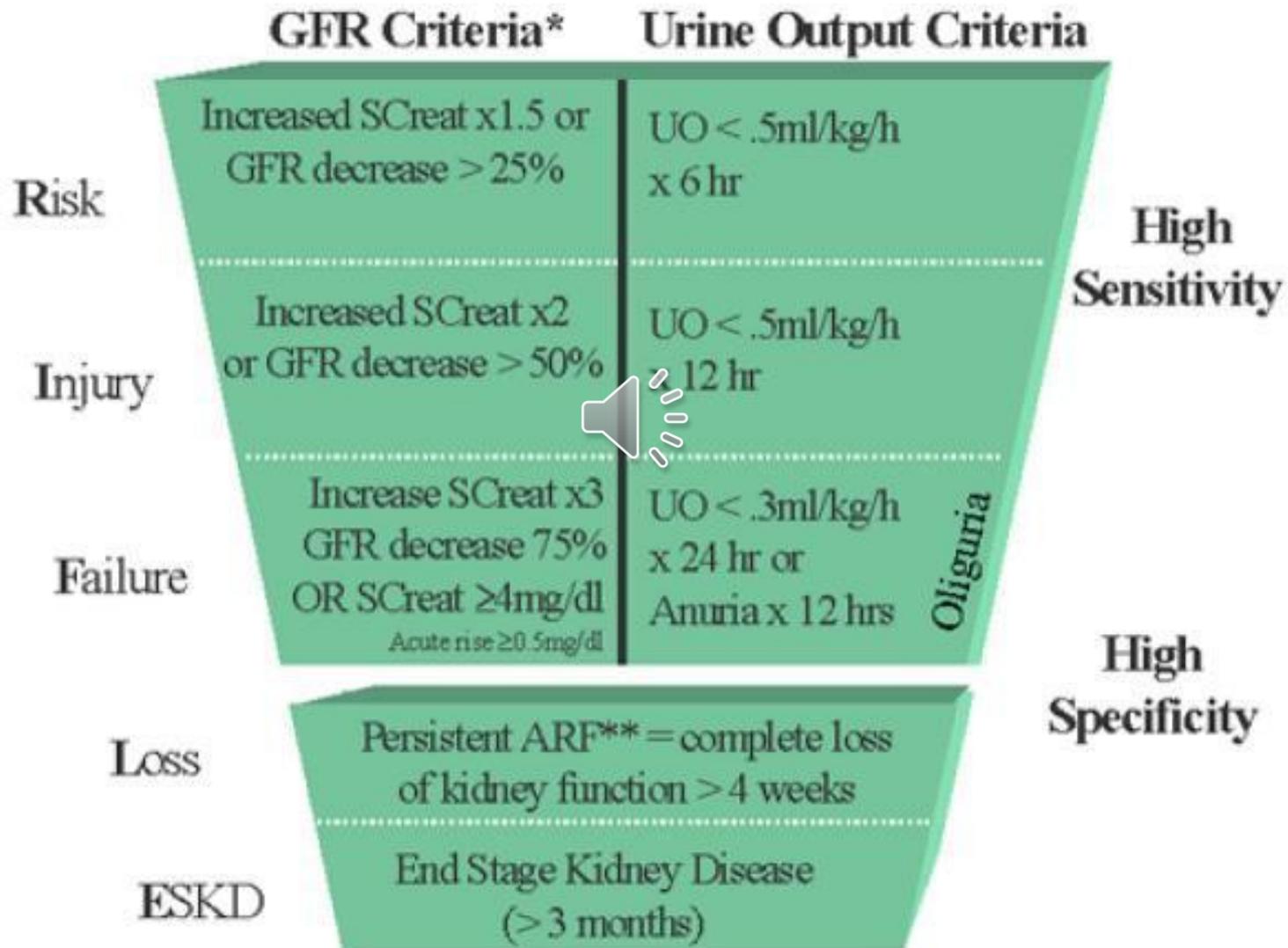
CR# =creatinine



«0» ούρα -> «0» κάθαρση



# AKI RIFLE Criteria: ADQI II



# Εκτίμηση της νεφρικής λειτουργίας στην οξεία νεφρική ανεπάρκεια

- Ρυθμός μεταβολής της κρεατινίνης από  
ημέρα-σε-ημέρα:



$< 1 \text{ mg/dl}$  = όχι πλήρης ανεπάρκεια

$1-3 \text{ mg/dl}$  = πλήρης ανεπάρκεια

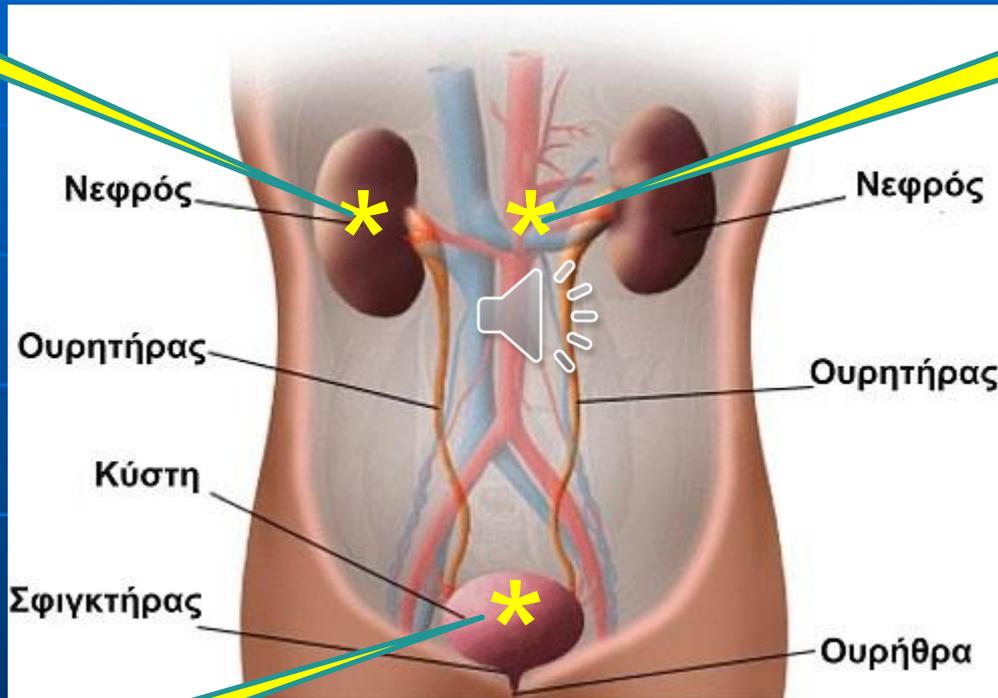
δηλαδή  $\text{GFR} < 10 \text{ ml/min}$

$> 3 \text{ mg/dl}$  = ραβδομυόλυση

# 1<sup>η</sup> ερώτηση:

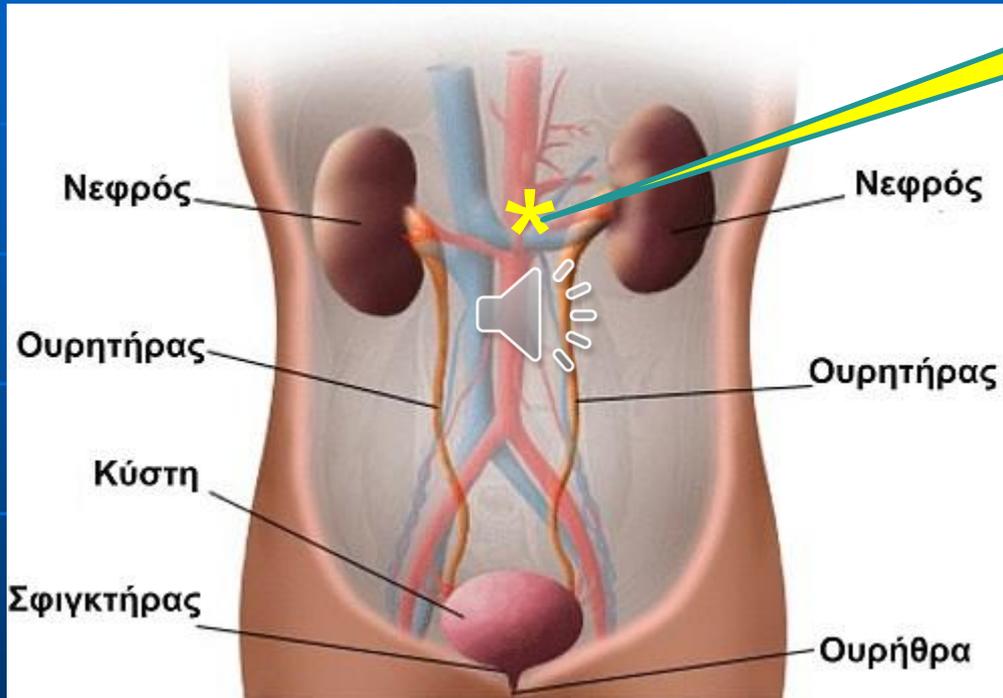
**Νεφρικά  
αίτια**

**Προνεφρικά  
αίτια**



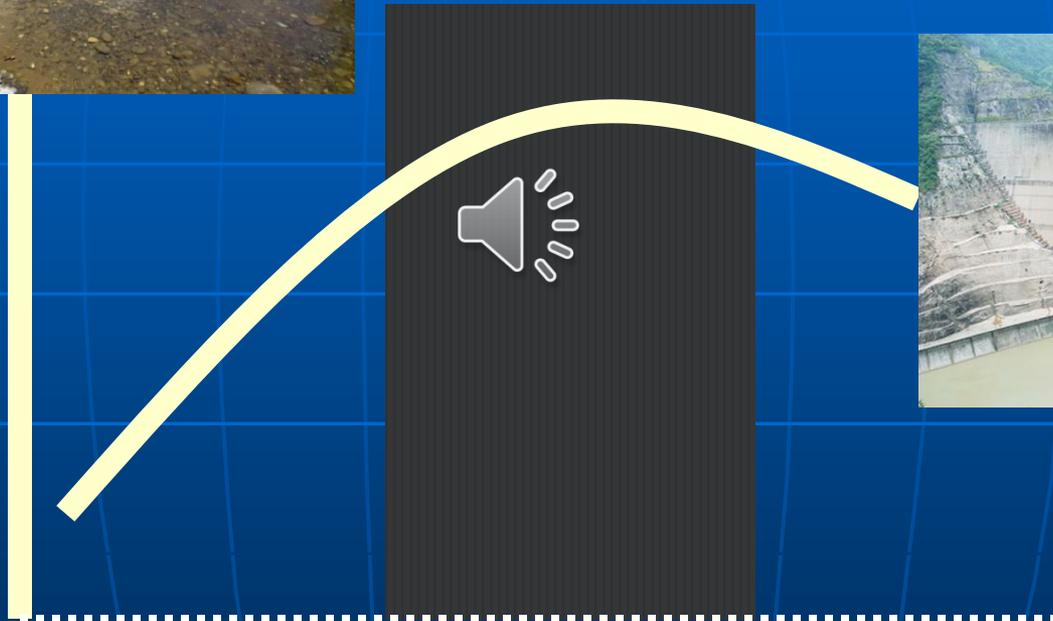
**Μετανεφρικά  
αίτια**

**Προνεφρικά  
αίτια**





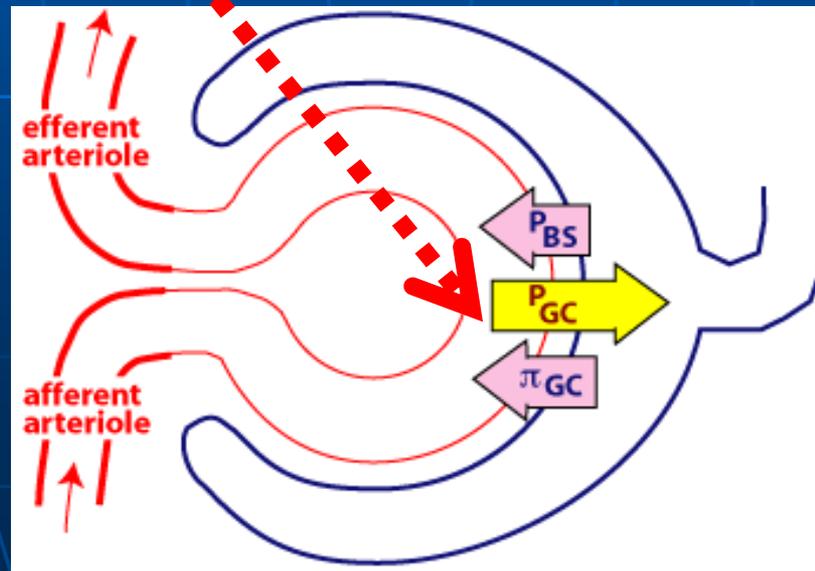
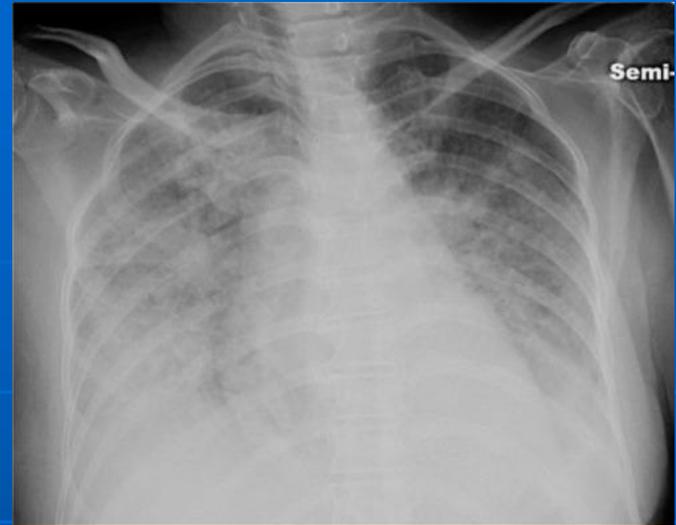
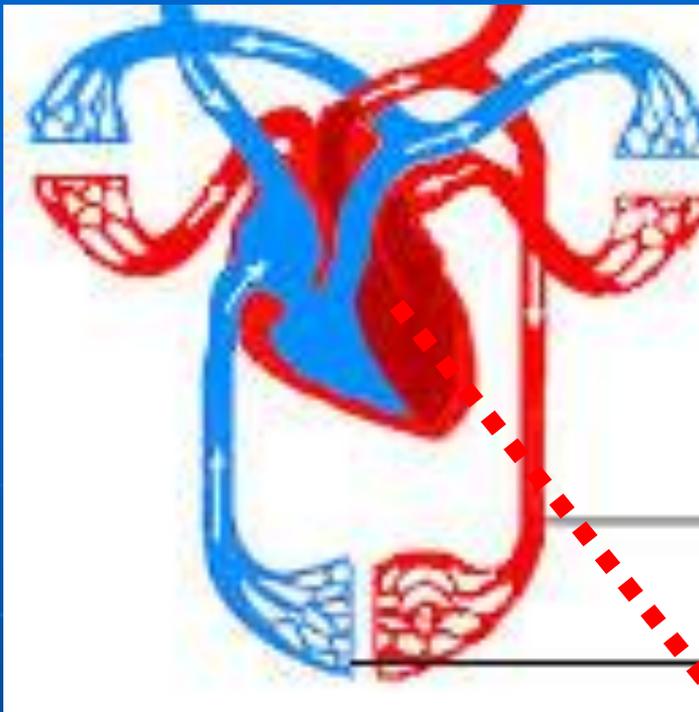
ΚΛΟΑ



Υποογκαιμικός

Υπερυδατωμένος

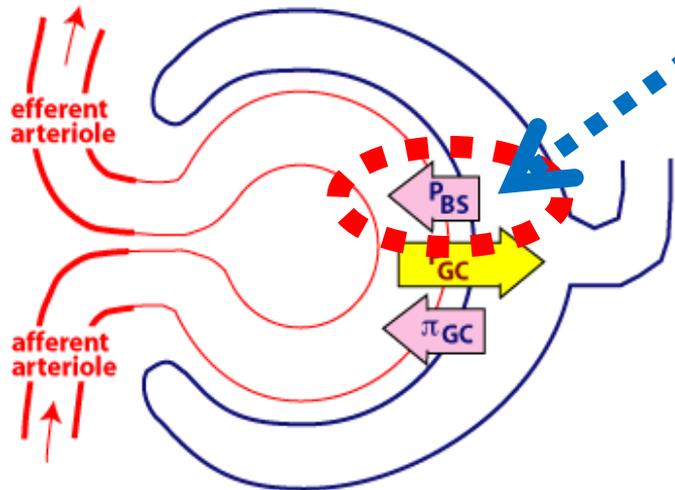
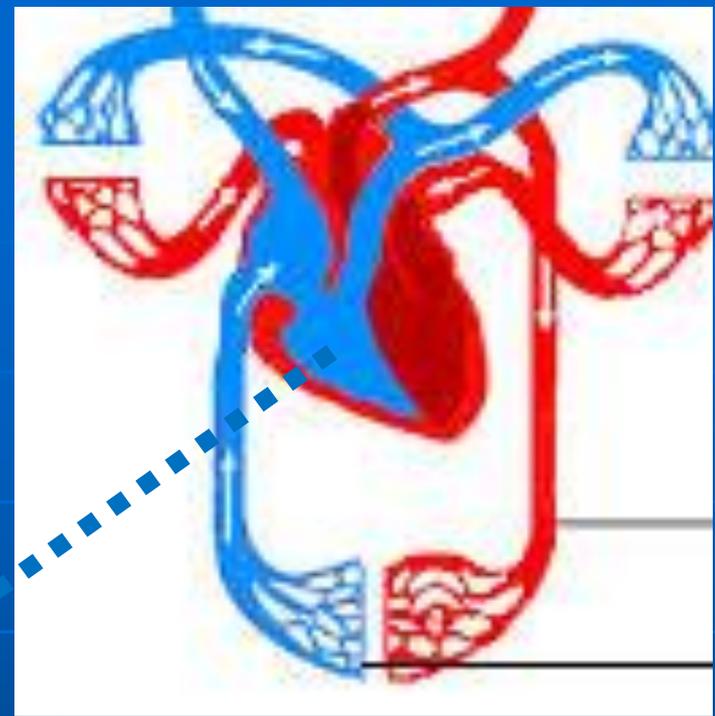
Πίεση Ενσφήνωσης



$$P_{GC} = 60 \text{ mmHg}$$

$$P_{BS} = -15 \text{ mmHg}$$

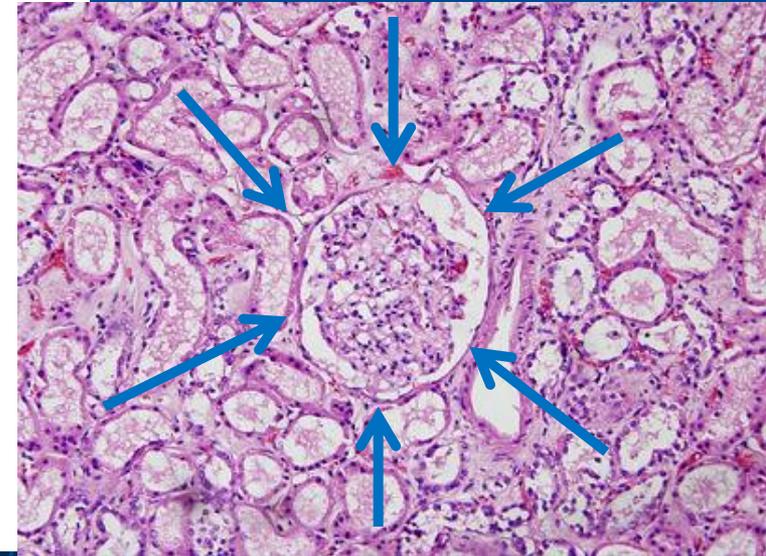
$$\pi_{GC} = -29 \text{ mmHg}$$



$P_{GC} = 60 \text{ mmHg}$

$P_{BS} = -15 \text{ mmHg}$

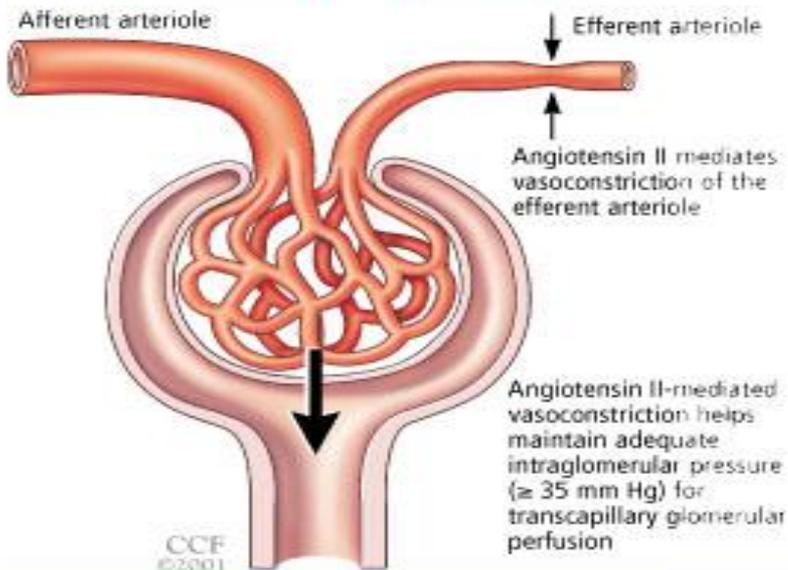
$\pi_{GC} = -29 \text{ mmHg}$



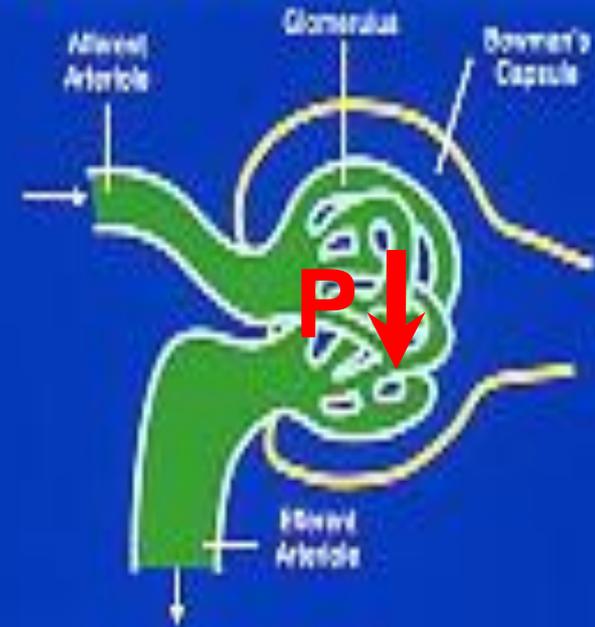
# Προσοχή στους αναστολείς του Συστήματος Ρενίνης-Αγγειοτασίνης

The role of angiotensin II in maintaining adequate intraglomerular pressure

**Αγγειοτασίνη II**

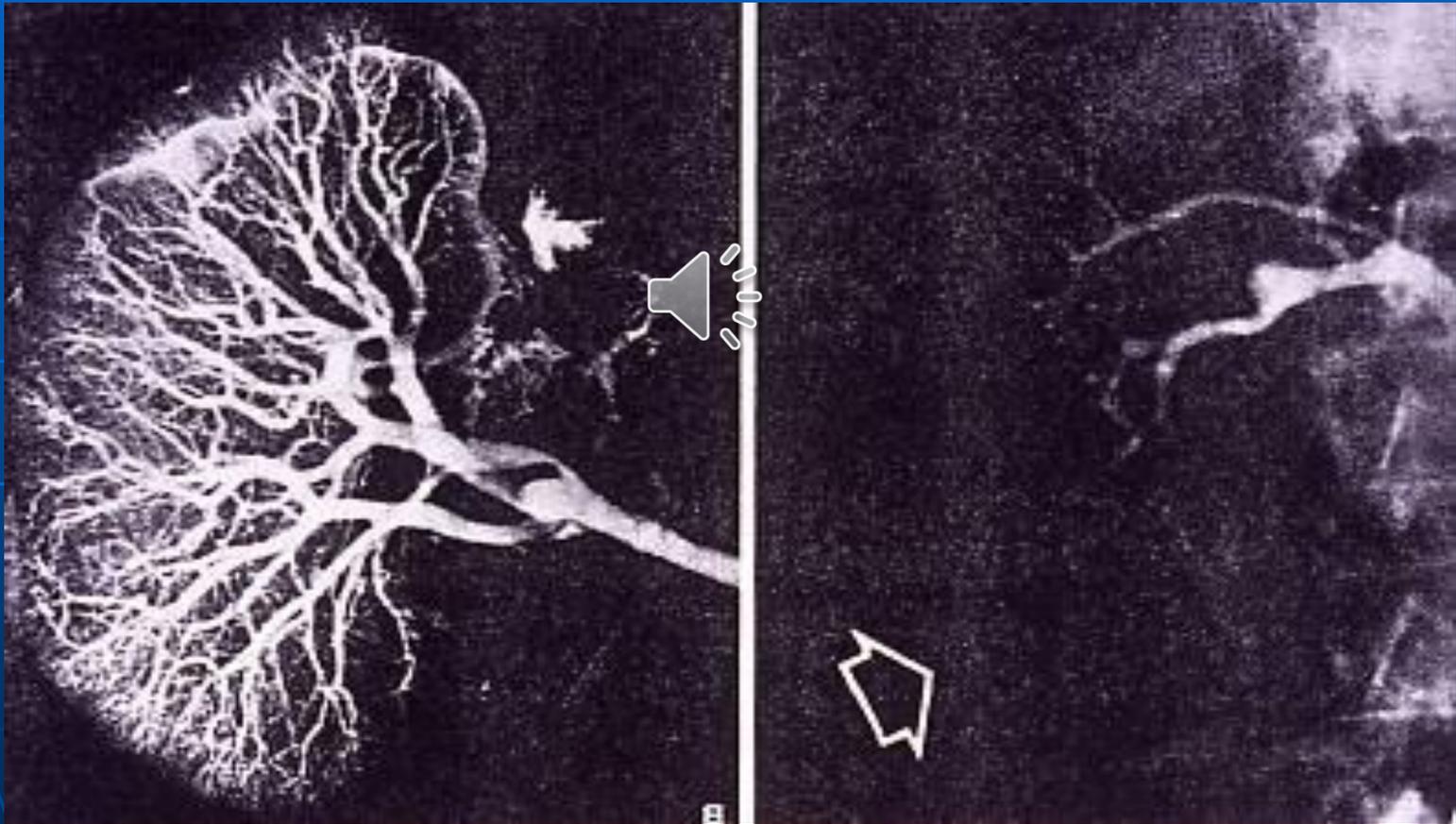


## Intrarenal Effects of ACE Inhibitors



**ACEi, ARB's**

# Προσοχή στα ΜΣΑΦ



# Θεραπευτική προσέγγιση ασθενών με προνεφρικά αίτια ΟΝΑ

## Μειωμένος

### εξωκυττάριος όγκος

- Αντιμετώπιση πρωταρχικής αιτίας
- Αναπλήρωση με αίμα, πλάσμα, κρυσταλλοειδή ή κολλοειδή διαλύματα

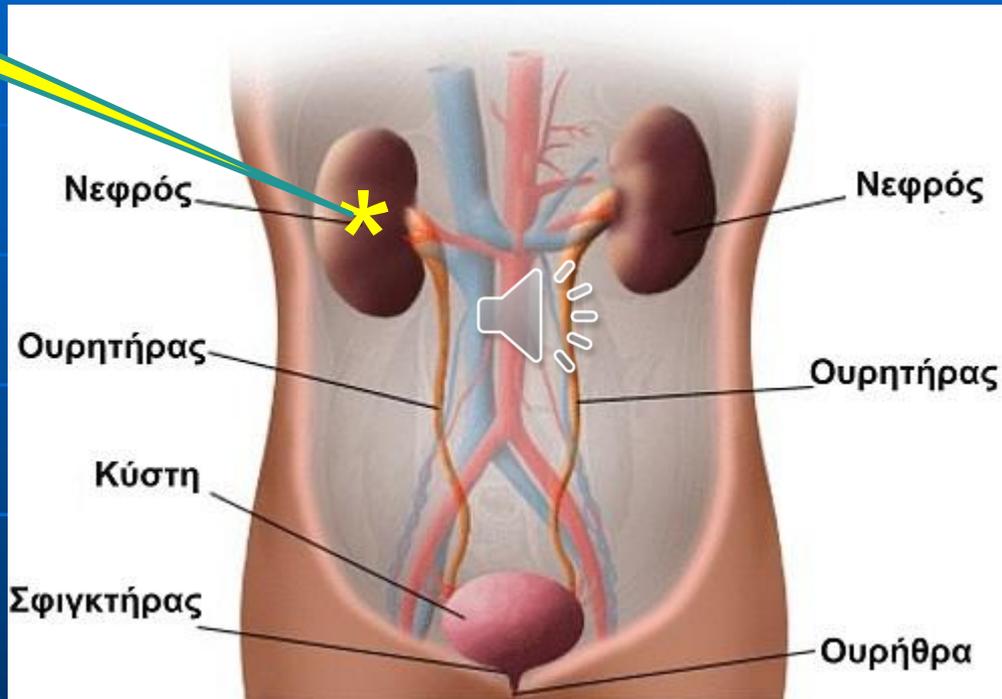


## Αυξημένος

### εξωκυττάριος όγκος

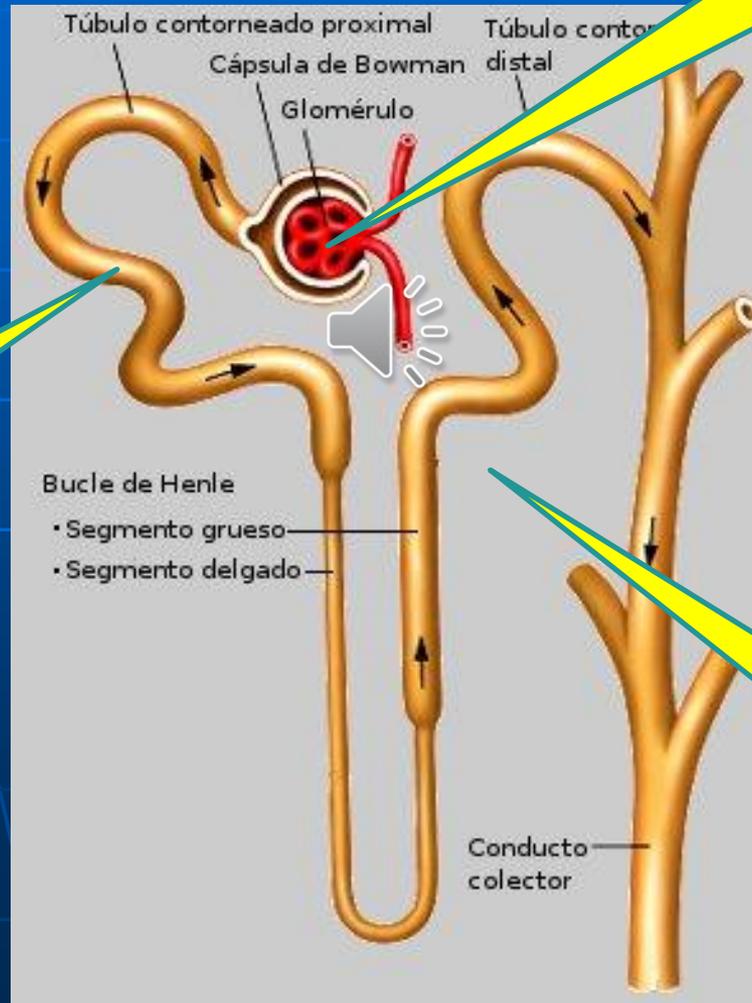
- Στέρηση άλατος και ύδατος
- Καρδιοτόνωση με κατεχολαμίνες, ενδοαορτικό μπαλόνι
- Διουρητικά

## Νεφρικά αίτια



# 2<sup>η</sup> ερώτηση

**Σπειραματονεφρίτις  
(5%)**



**Οξεία  
σωληναριακή  
νέκρωση  
(85%)**

**Διάμεσος  
Νεφρίτις  
(10%)**

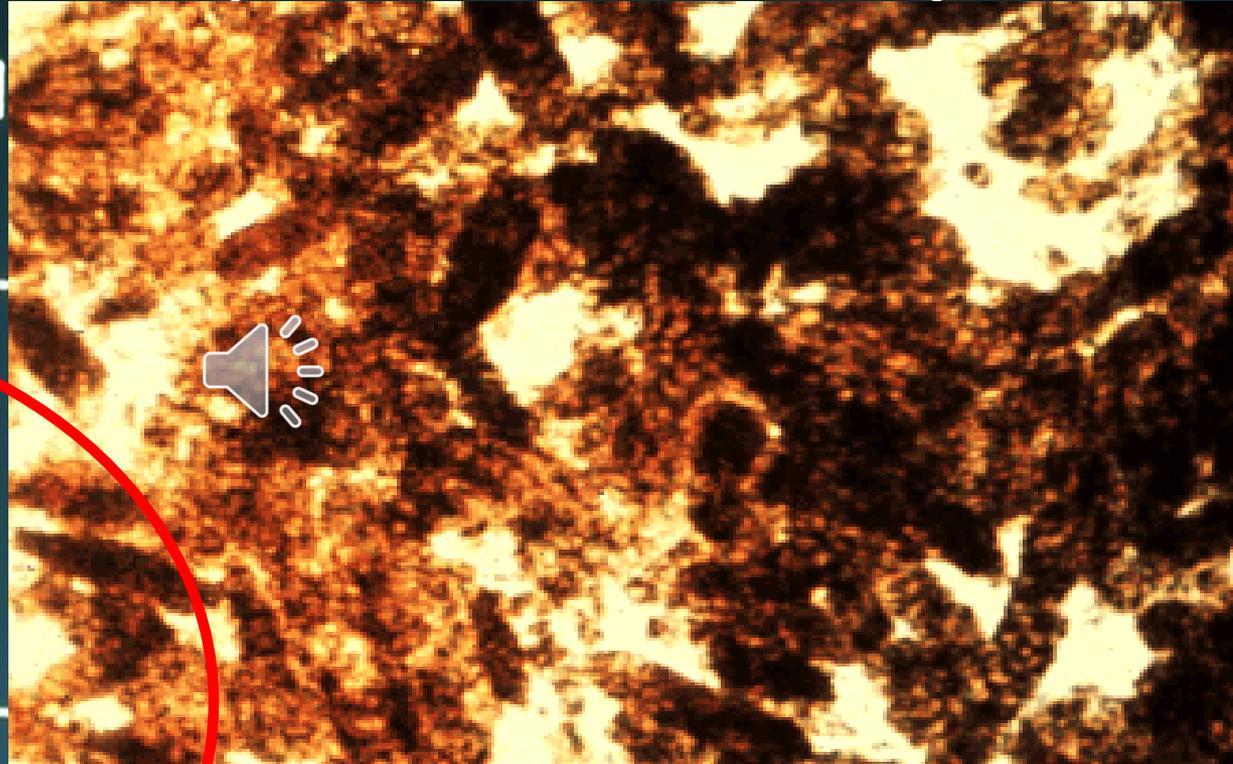
# Acute renal failure

Prerenal causes

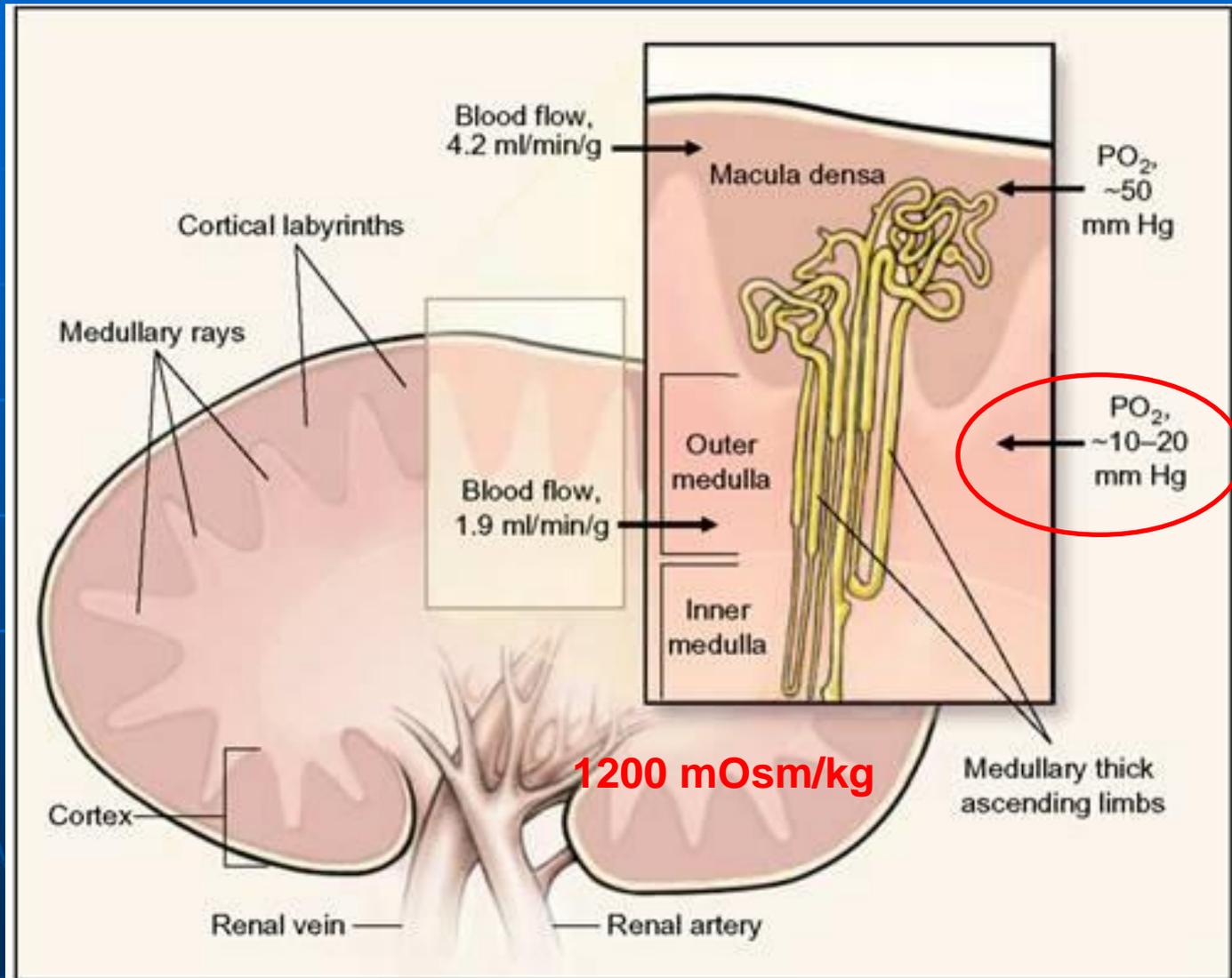
Tubular necrosis

Ischemia  
(50% of cases)

Toxins  
(35% of cases)



# Vulnerability to Ischaemia



# Causes of Toxic Acute Renal Failure Associated with Cardiological or Cardiovascular Interventions.

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## Antibiotics

Aminoglycosides

Sulfonamide Cotrimoxazole

Quinolones

Amphotericin B

Acyclovir

## Anesthetic Agents

Methoxyflurane

Enflurane



## Contrast Media

## Heme Pigments

Myoglobin

Hemoglobin

## Miscellaneous

Dextrans

IV gamma globulins

Mannitol

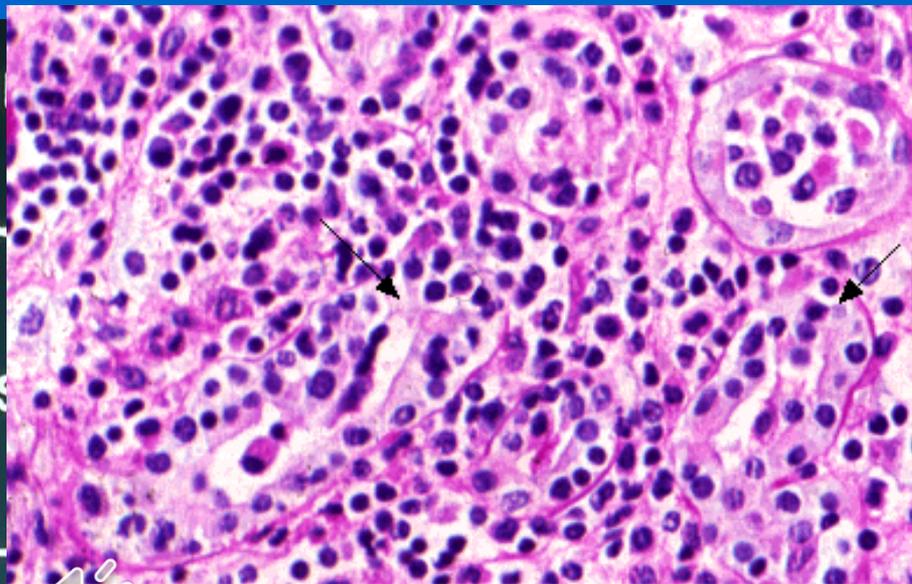
Cyclosporine

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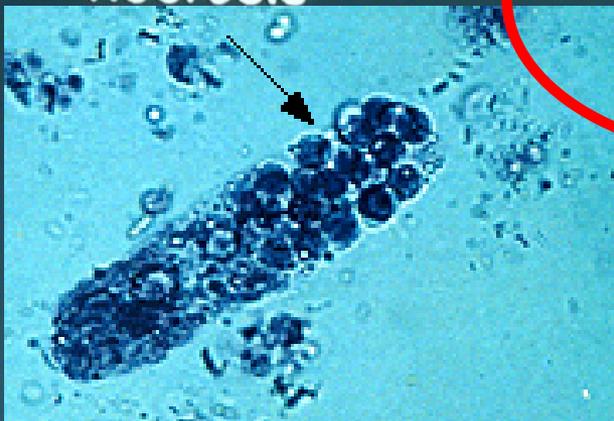
Acute

Prerenal  
causes

Intrinsic



Tubular  
necrosis



Interstitial  
nephritis  
(10% of cases)

Acute  
glomerulonephritis  
(5% of cases)

Glomerulonephritis  
(cases)

# Αίτια Διαμέσου Νεφρίτιδας

- 1) Φάρμακα
- 2) Λοιμώξεις
- 3) Σαρκοείδωση 
- 4) Sjogren's syndrome
- 5) Ενδημική Νεφροπάθεια των Βαλκανίων
- 6) Chinese Herb Nephritis

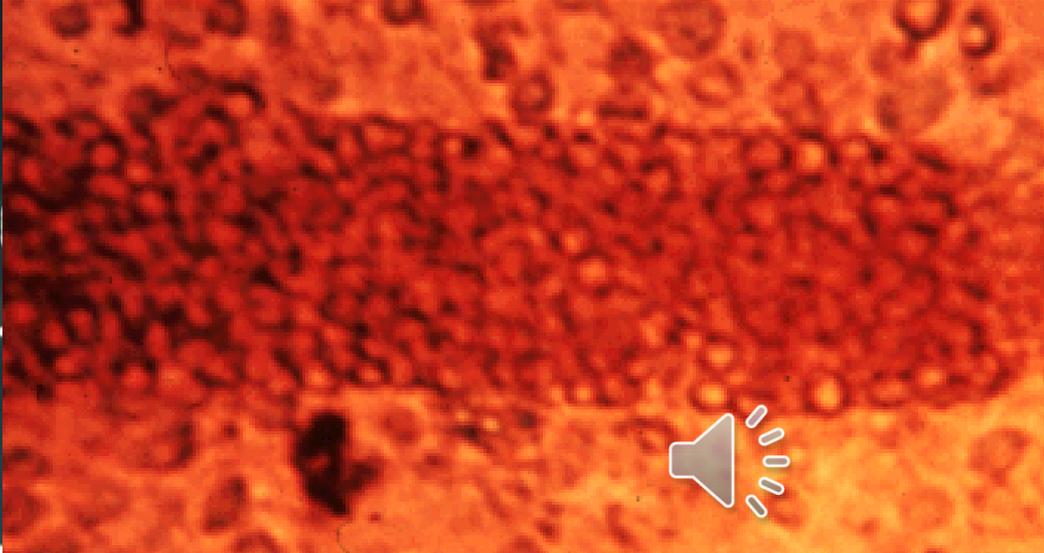
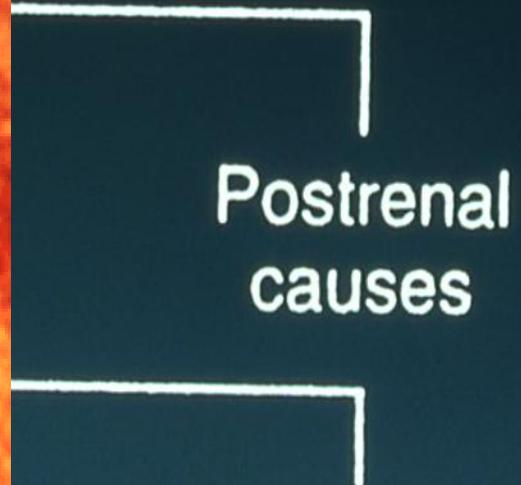
# Φάρμακα και Διάμεσος Νεφρίτις

- β-λακτάμες π.χ. μεθικιλλίνη, πενικιλλίνη, κεφαλοσπορίνες
- Ριφαμπικίνη
- Φάρμακα με σουλφομάδα π.χ. Φουροσεμίδη, Σουλφαμεθοξαζόλη, Σουλφασαλαζίνη
- Σιπροφλοξασίνη
- Μη στερινοειδή αντιφλεγμονώδη π.χ. φενοπροφένη

# Λοιμώξεις και Διάμεσος Νεφρίτις

- Legionella
- Leptospirosis 
- Streptococcal infections
- Viruses

# Acute renal failure



Pre  
cau

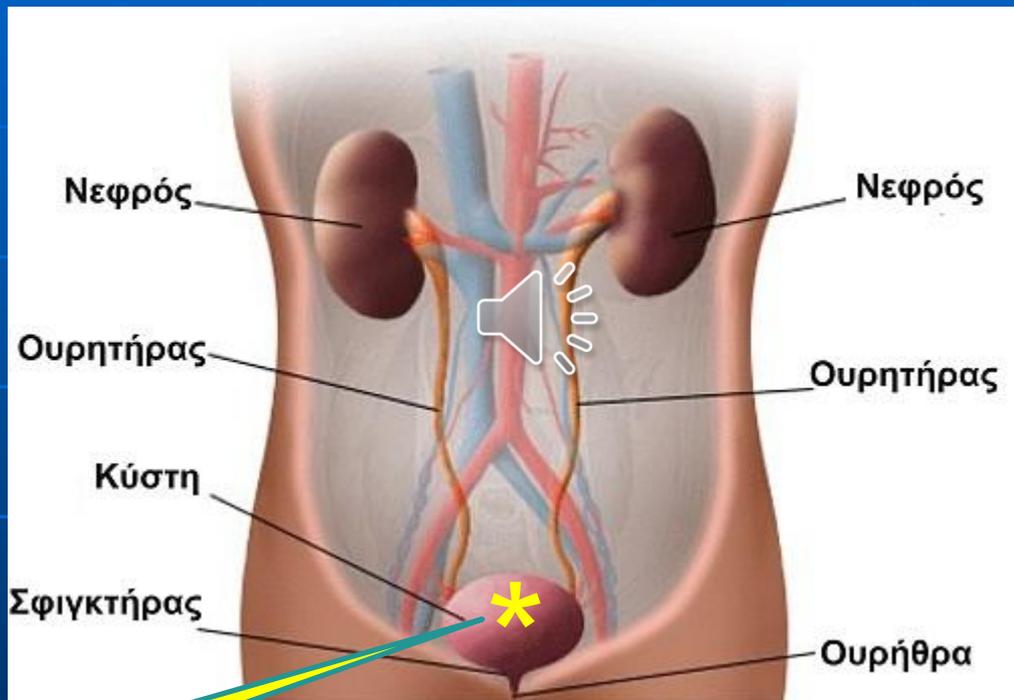
Tubular  
necrosis

Ischemia  
(50% of cases)

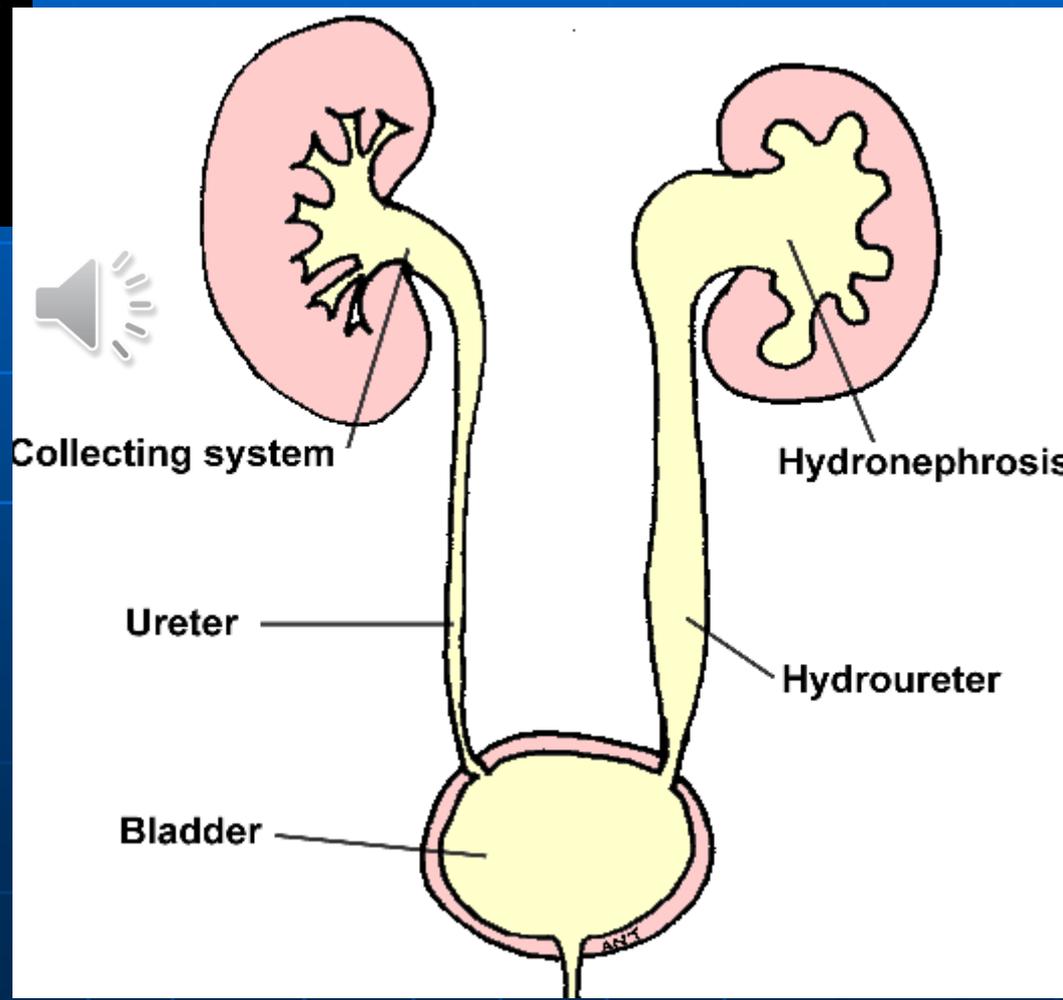
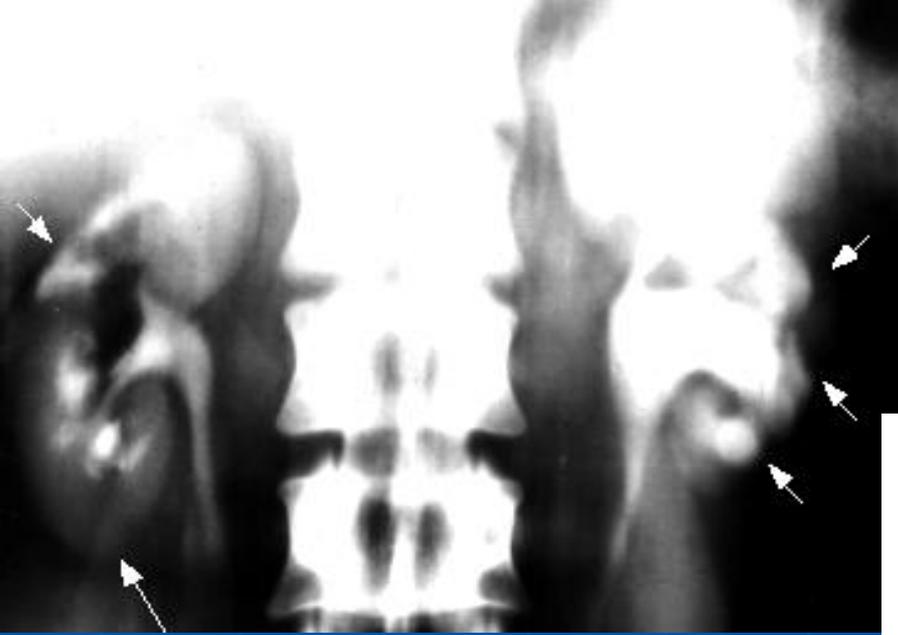


(55% of cases)

Acute  
glomerulonephritis  
(5% of cases)

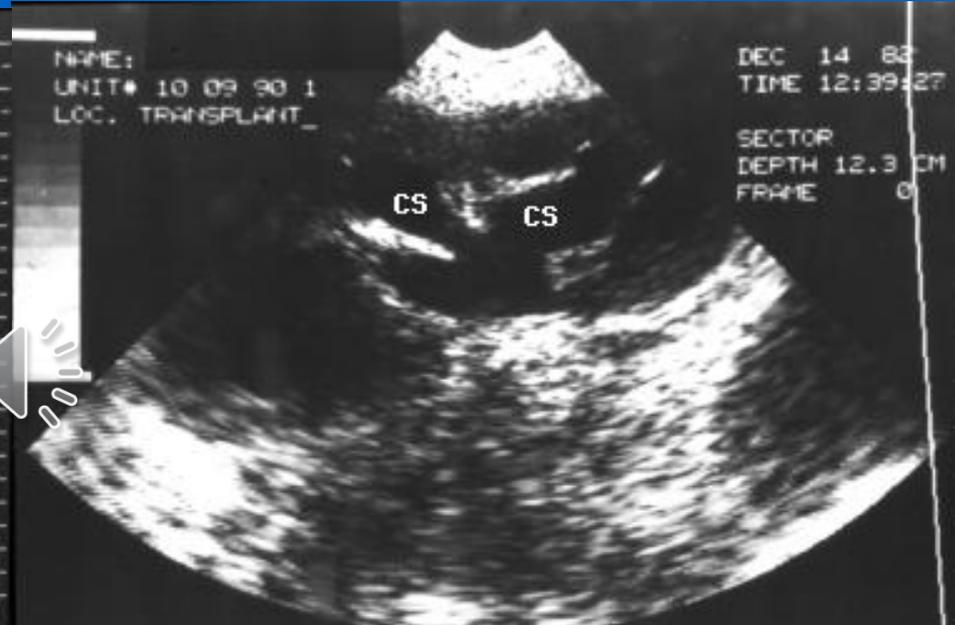


**Μετανεφρικά  
αίτια**





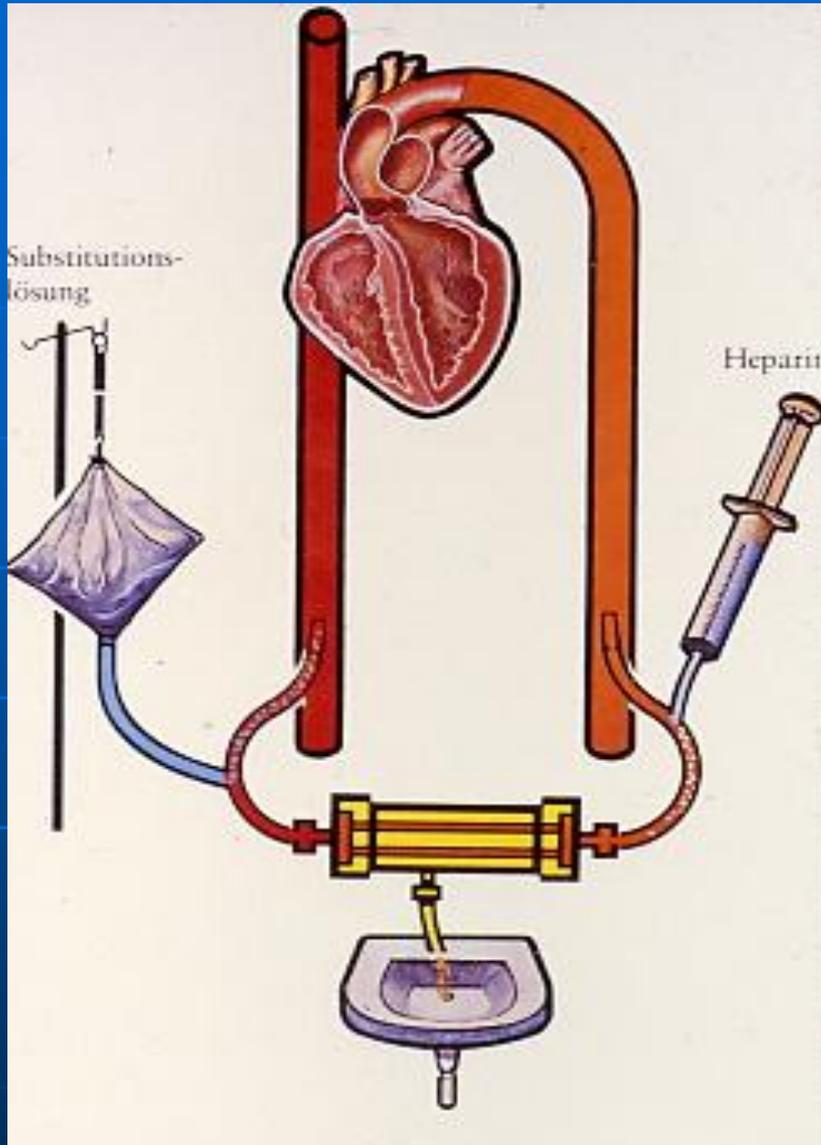
**Normal renal ultrasound** Normal renal ultrasonogram showing the renal outline and the normal width of the renal parenchyma (RP) which is represented by the black area between the renal capsule and, in white, the collecting system (CS). The collecting system structures are closely bunched together.



**Urinary tract obstruction** Renal ultrasonogram showing hydronephrosis due to urinary tract obstruction. The collecting structures (CS) are distended by fluid, rather than being closely bunched together as in the normal kidney. This study was performed in a renal transplant.

# Θεραπευτικά μέτρα επί ΟΝΑ

- Προσπάθεια μετατροπής της ολιγουρικής σε μη-ολιγουρική ΟΝΑ
- Ισοζύγιο υγρών και καθημερινό ζύγισμα
- Αναλος διαίτα, χαμηλή σε κάλιο
- Περιορισμός προσλαμβανόμενων λευκωμάτων (0.8 g/kg βάρους)
- Αποφυγή νεφροτοξικών ουσιών
- Προσαρμογή δοσολογικού σχήματος φαρμάκων



## ΕΝΔΕΙΞΕΙΣ ΕΞΩΣΩΜΑΤΙΚΗΣ ΚΑΘΑΡΣΗΣ

1) Κατακράτηση υγρών –  
Καρδιακή κάμψη

2) Ηλεκτρολυτικές διαταραχές

3) Σοβαρές διαταραχές της ΟΒ  
ισορροπίας

4) Ουραιμική συνδρομή

# ΧΡΟΝΙΑ ΝΕΦΡΙΚΗ ΑΝΕΠΑΡΚΕΙΑ



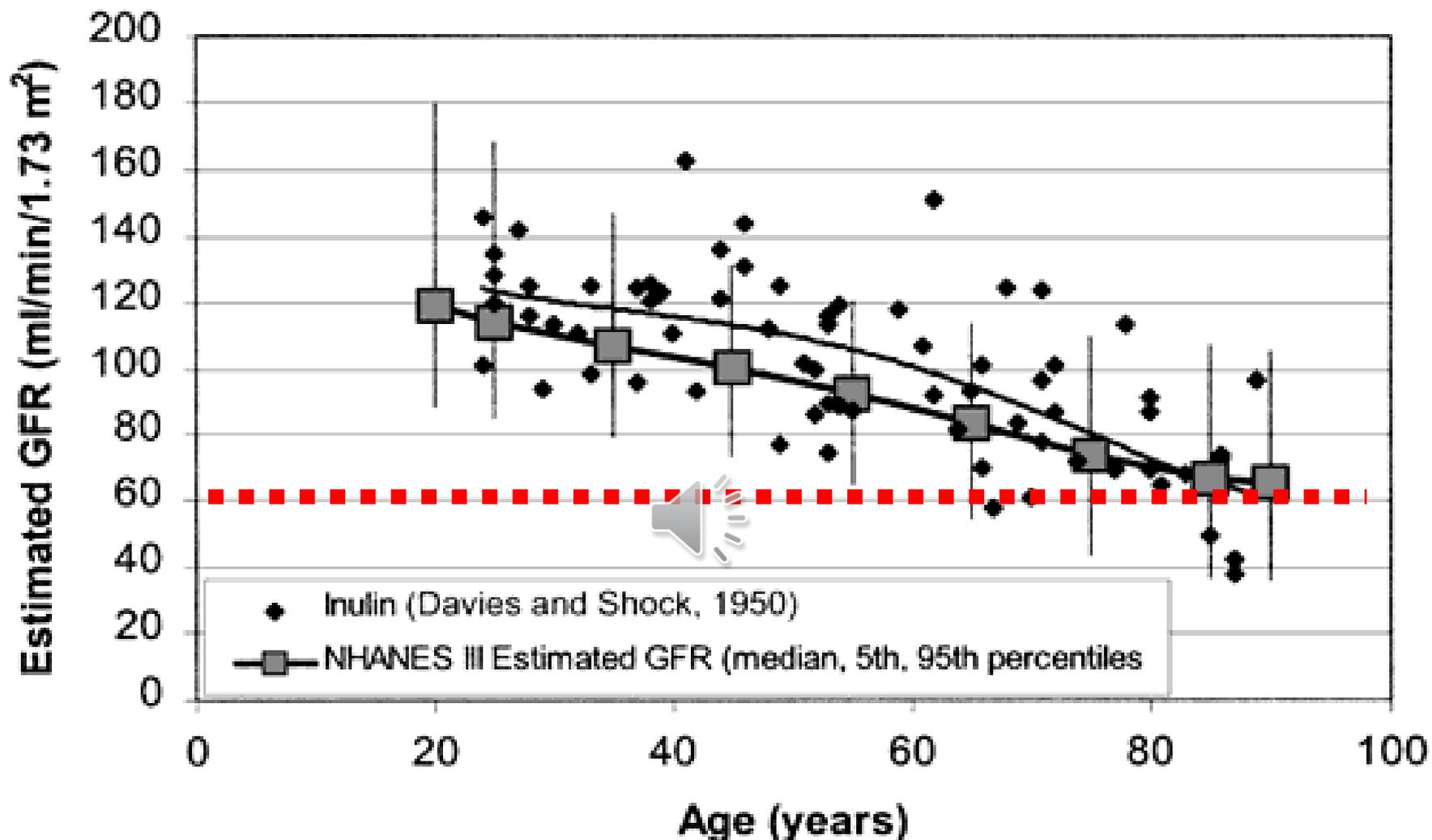
Δημήτριος Β. Βλαχάκος

Καθηγητής Παθολογίας - Νεφρολογίας

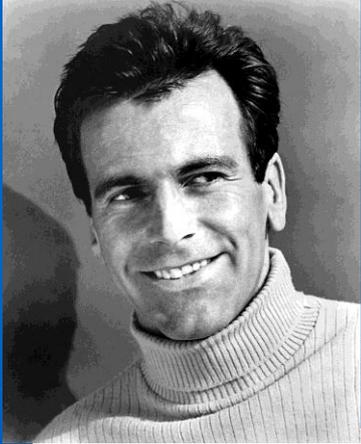
Μονάδα Νεφρολογίας και Υπερτάσεως

Β' Προπαιδευτική Παθολογική Κλινική

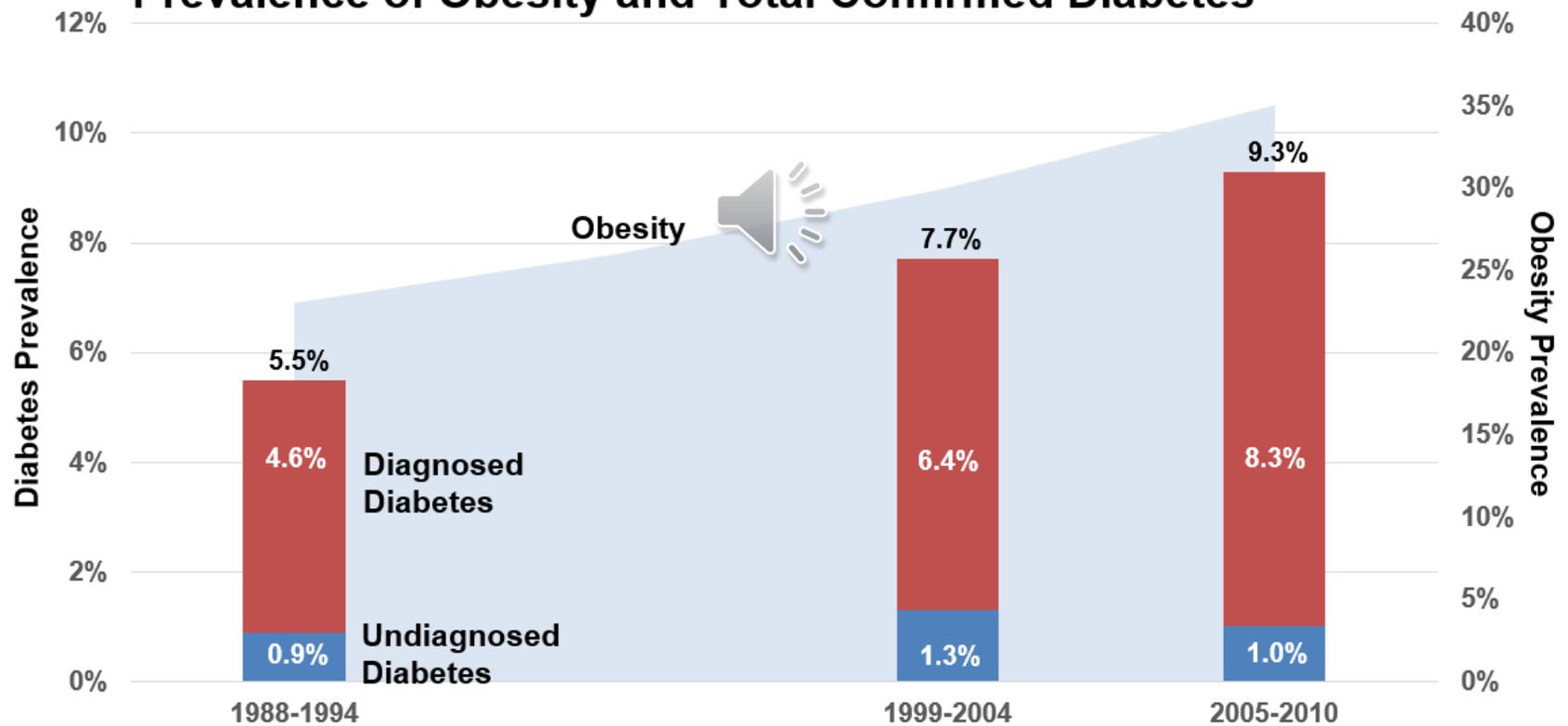
Πανεπιστημιακό Γενικό Νοσοκομείο «ΑΤΤΙΚΟΝ»



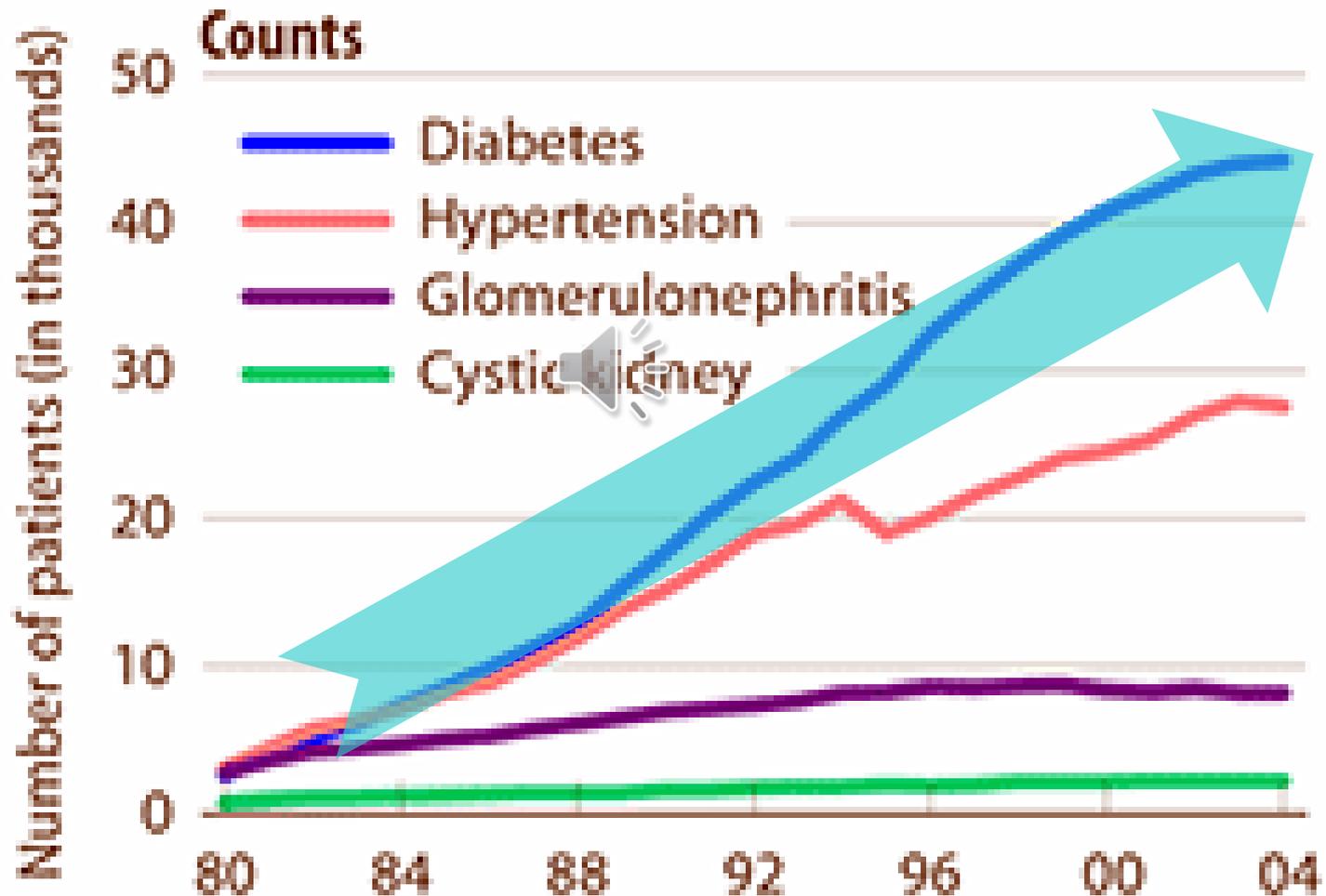
**Figure 9** GFR versus age. Estimated GFR percentiles for the US population using NHANES III serum creatinine, age, sex, and race data (see Part 10, Appendix 2) by age compared to a regression of inulin clearance measurement of GFR on age among 70 healthy male participants. (Data abstracted from Davies and Shock [72])



## Prevalence of Obesity and Total Confirmed Diabetes



# 1/3 of diabetics develop CKD





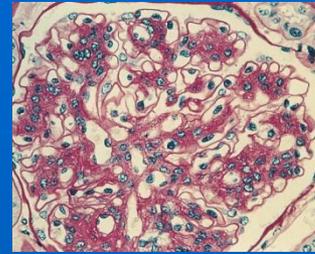
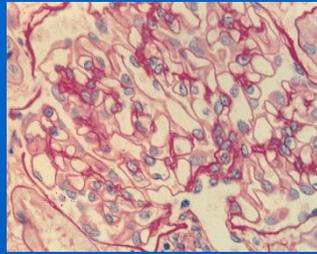
**ΔΙΑΒΗΤΙΚΗ  
ΝΕΦΡΟΠΑΘΕΙΑ**

# Διαβητική Νεφροπάθεια

- Λευκωματουρία (0,5 γρ 24/ωρο)
- Διαβητική  
αμφιβληστροειδοπάθεια 
- Απουσία κλινικών ή εργαστηριακών στοιχείων άλλης νεφρικής νόσου

# Στοιχεία υπέρ μη Διαβητικής Νεφροπάθειας

- Παθολογικό ίζημα ούρων (*αιματουρία, πυουρία, δύσμορφα ερυθρά, ερυθροκυτταρικοί κύλινδροι*)
- Απουσία διαβητικής *αμφιβληστροειδοπάθειας*
- Ταχεία επιδείνωση πρωτεϊνουρίας
- Ταχεία άνοδος της κρεατινίνης του ορού
- Άτυπες υπερηχογραφικές αλλοιώσεις των νεφρών (*ασυμμετρία του μεγέθους των νεφρών*)
- Διάρκεια του ΣΔ τύπου 1 λιγότερο από 5 χρόνια



## Microalbuminuria    Macroproteinuria

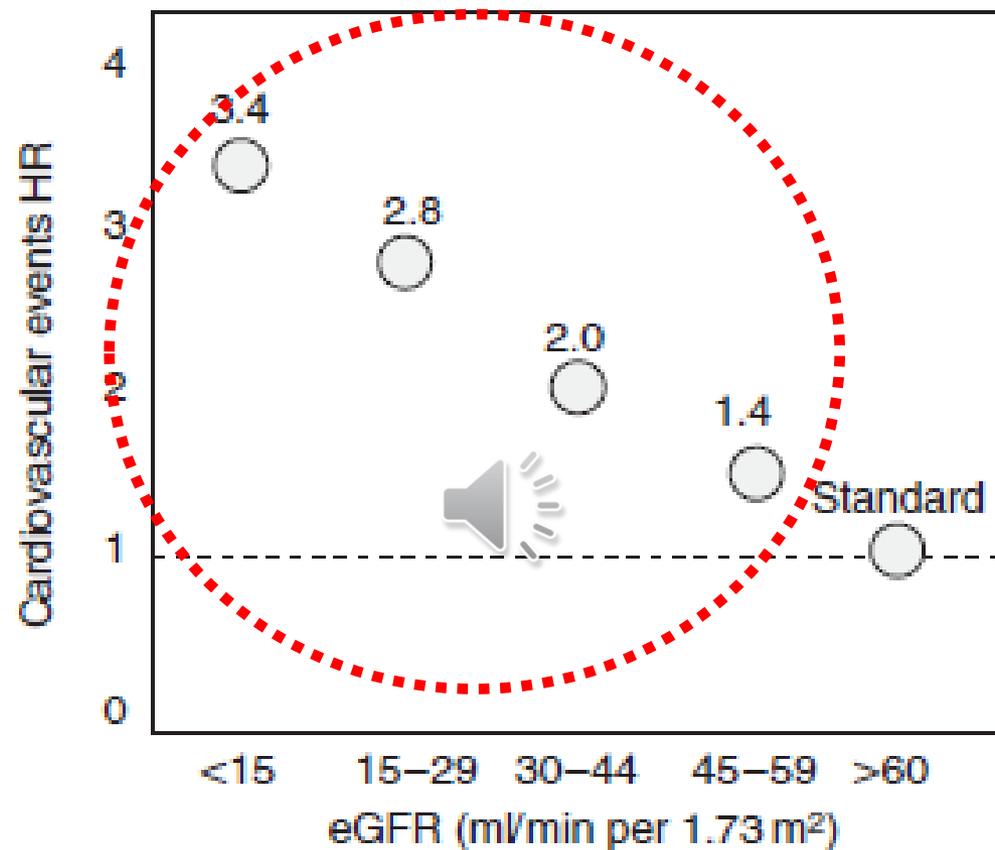




**Think Kidney!**



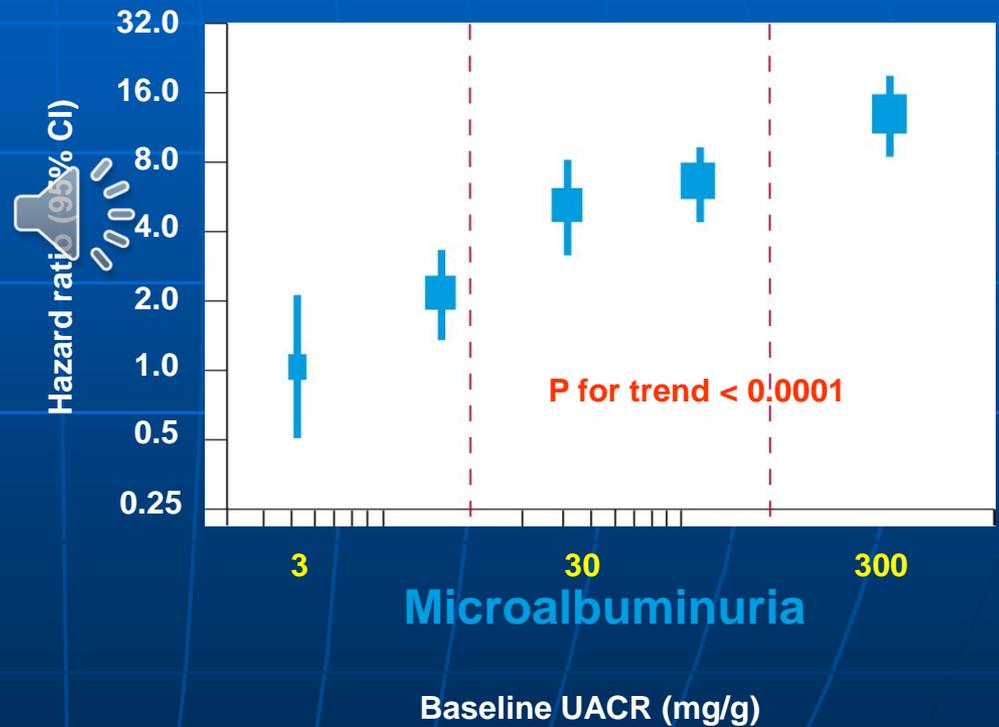
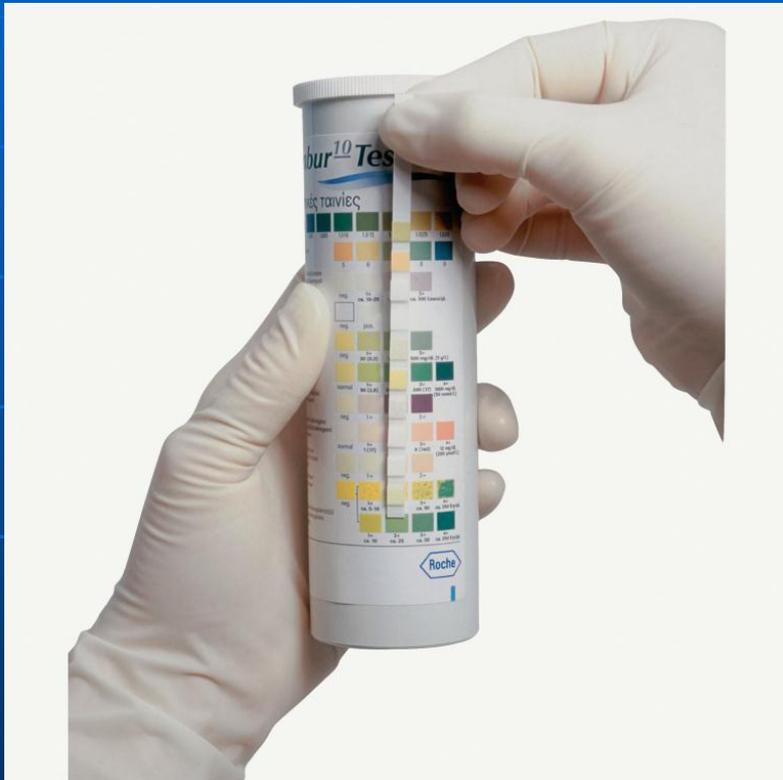
**PROGNOSIS**



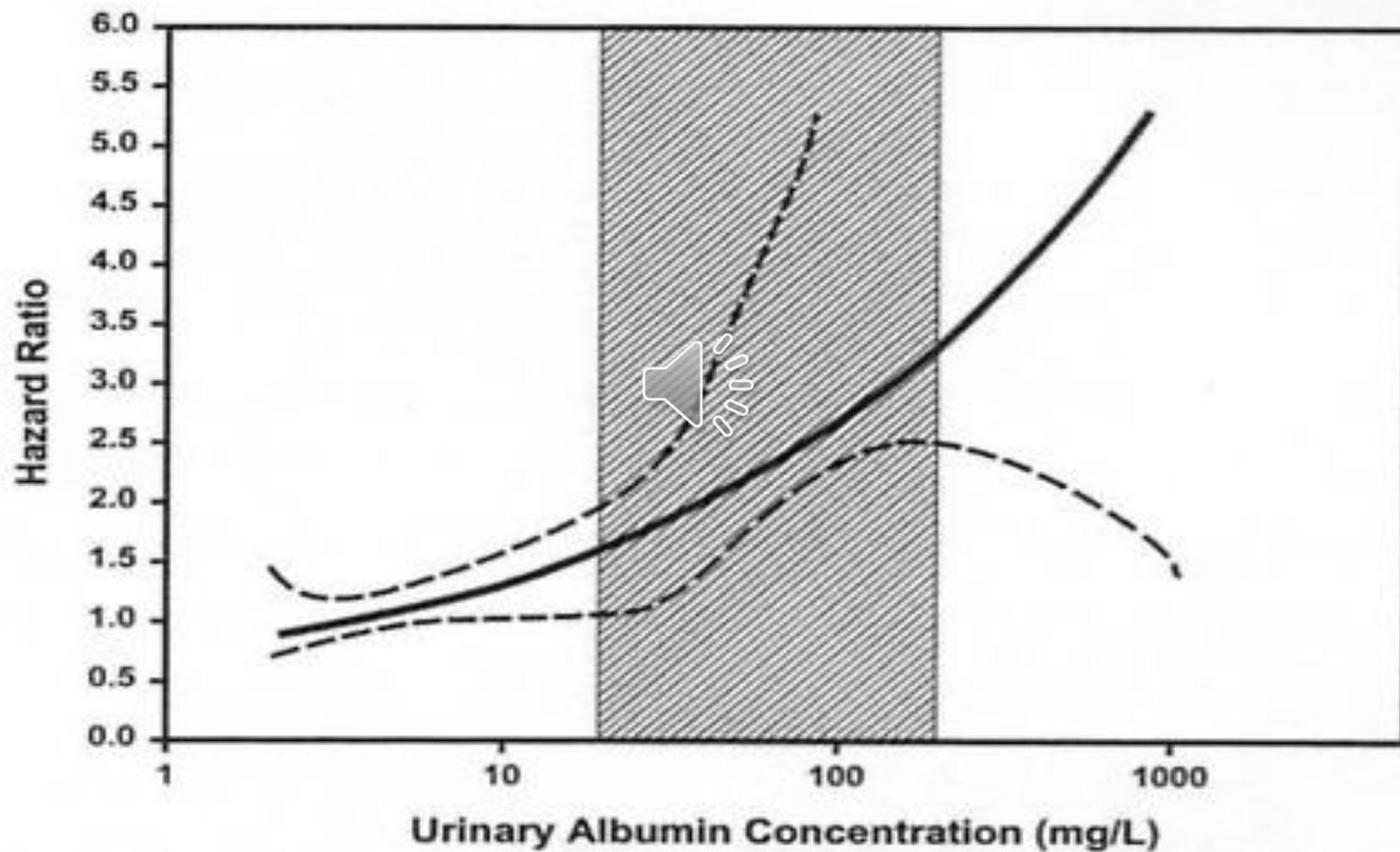
**Figure 1 | Relationship between glomerular filtration rate (GFR) and the risk for cardiovascular events.** Data are adjusted for age, sex, and other Framingham risk factors. HR, hazard rate; eGFR, estimated GFR.



# Albuminuria predicts renal events

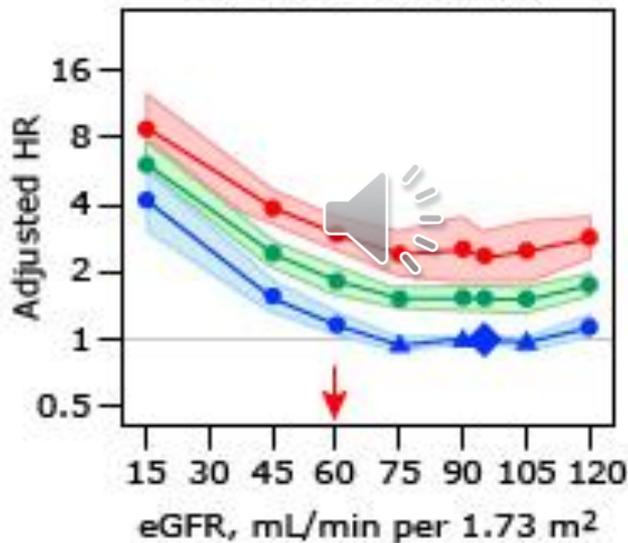


## Cardiovascular death

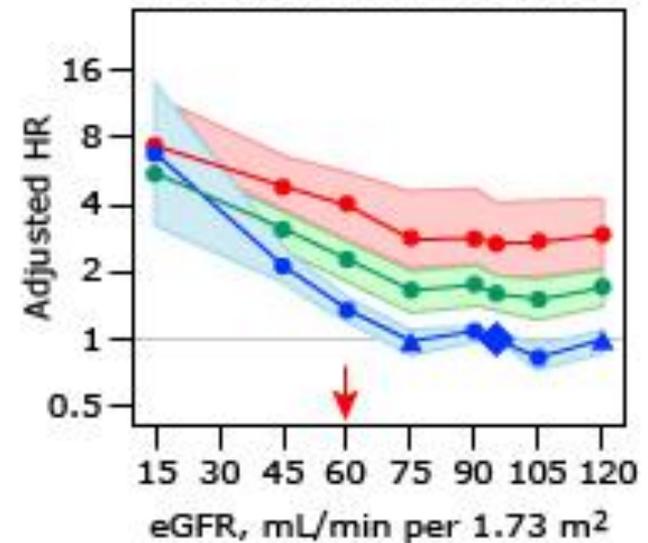


- Urine ACR >300 mg/g or dipstick >2+ positive
- Urine ACR 30-299 mg/g or dipstick 1+ positive
- Urine ACR of <30 mg/g or dipstick negative and trace

**All-cause mortality**



**Cardiovascular mortality**





**Think Kidney!**



**TREATMENT**

# Treatment Targets for Diabetic Renal Disease With Hypertension

GLUCOSE

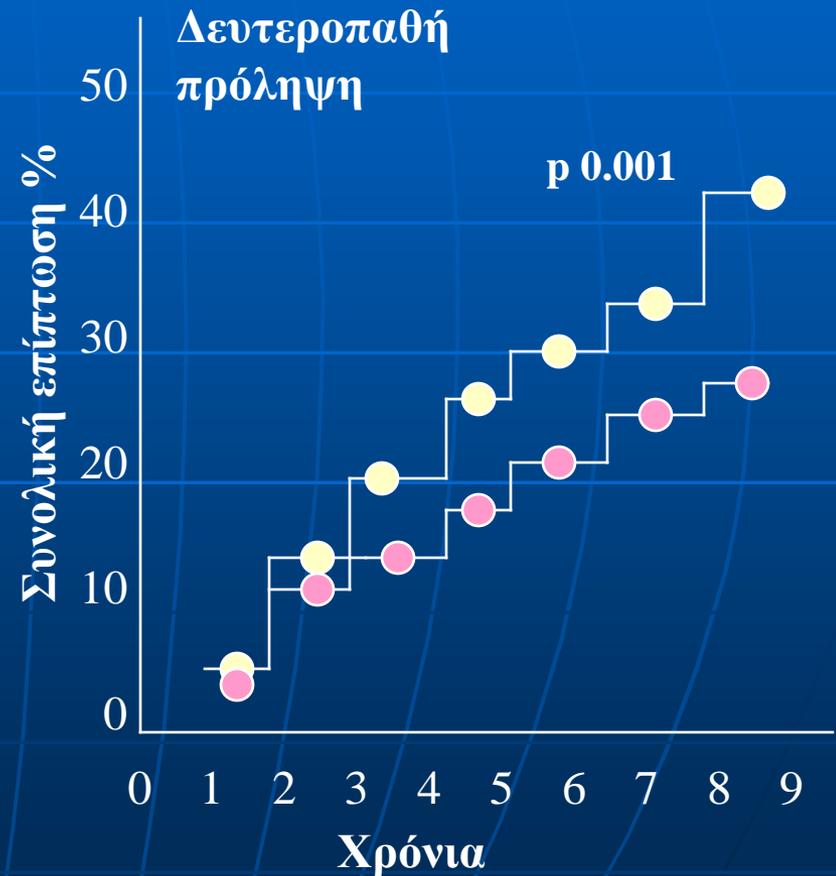
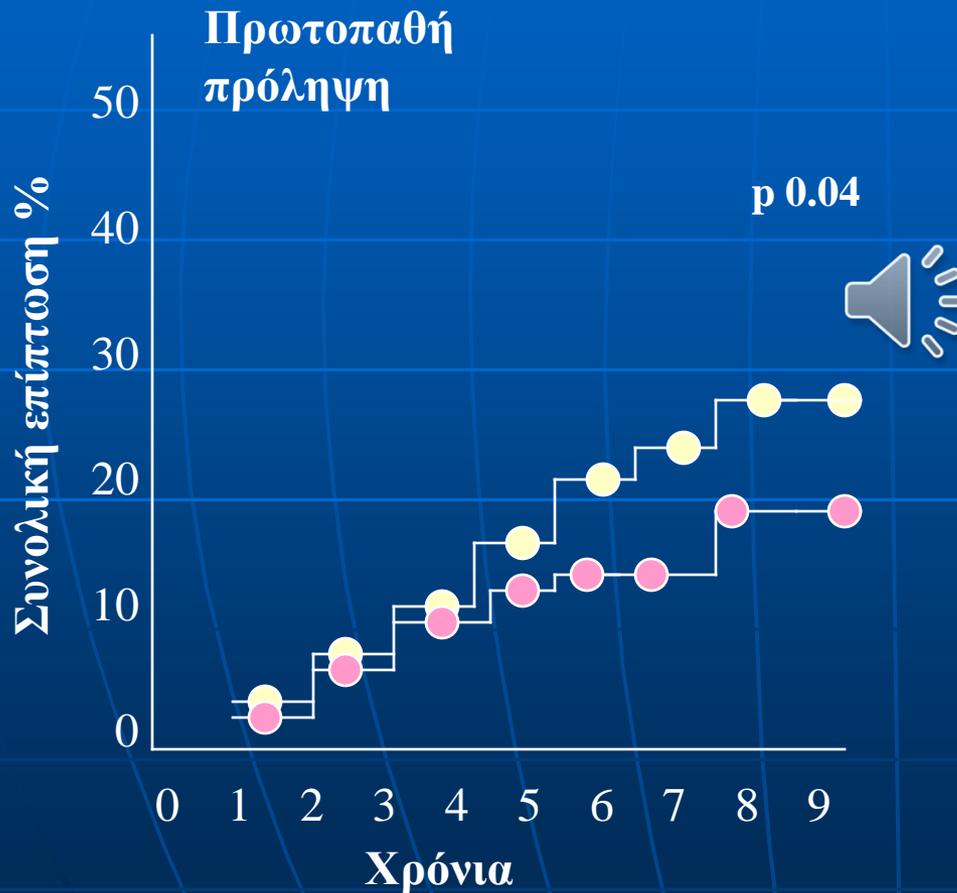


BP



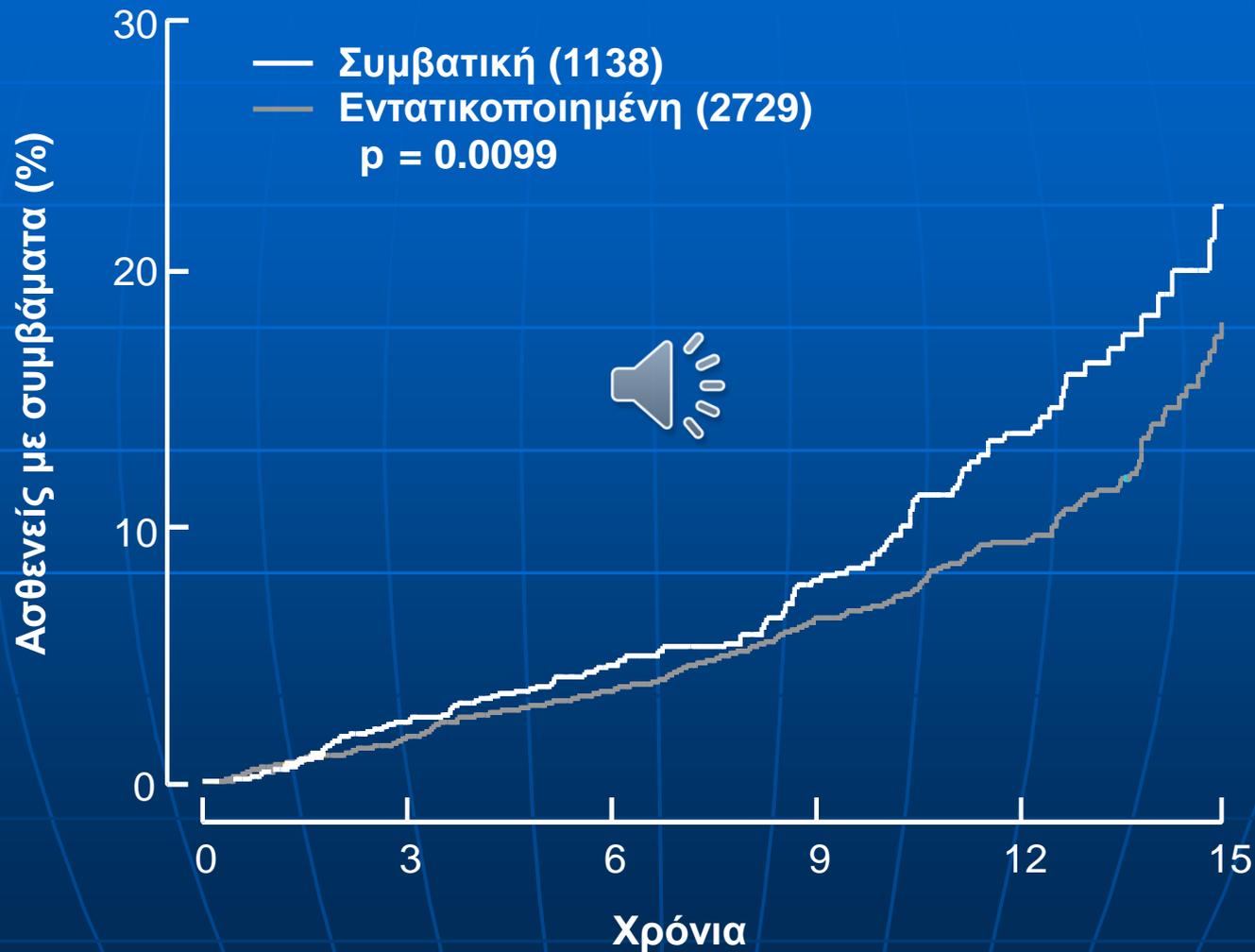
LIPIDS

# Διαβητική Νεφροπάθεια και ΣΔ Τύπου 1



- Εντατικοποιημένη
- Συμβατική

# Μικροαγγειακές Επιπλοκές και Ρύθμιση Γλυκόζης

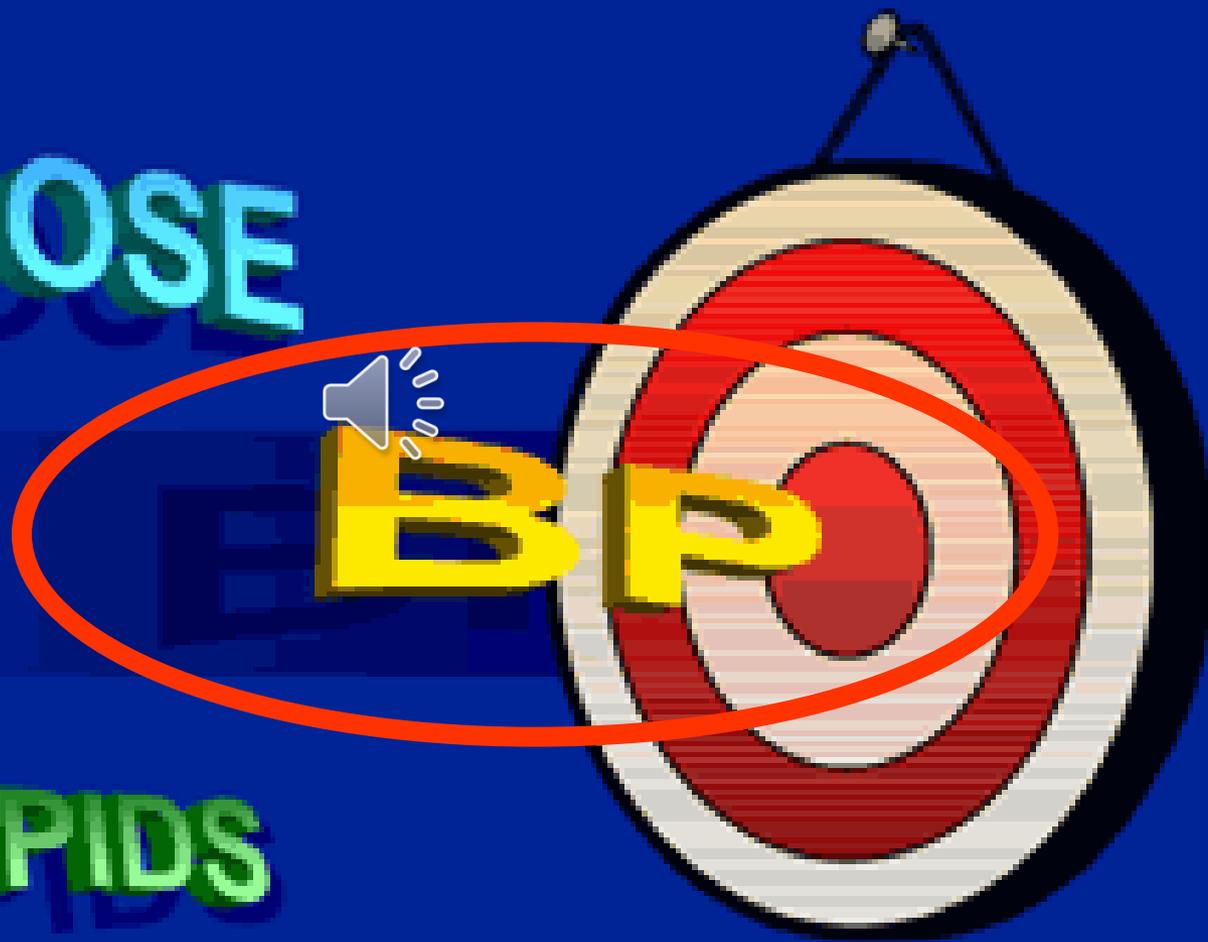


# Treatment Targets for Diabetic Renal Disease With Hypertension

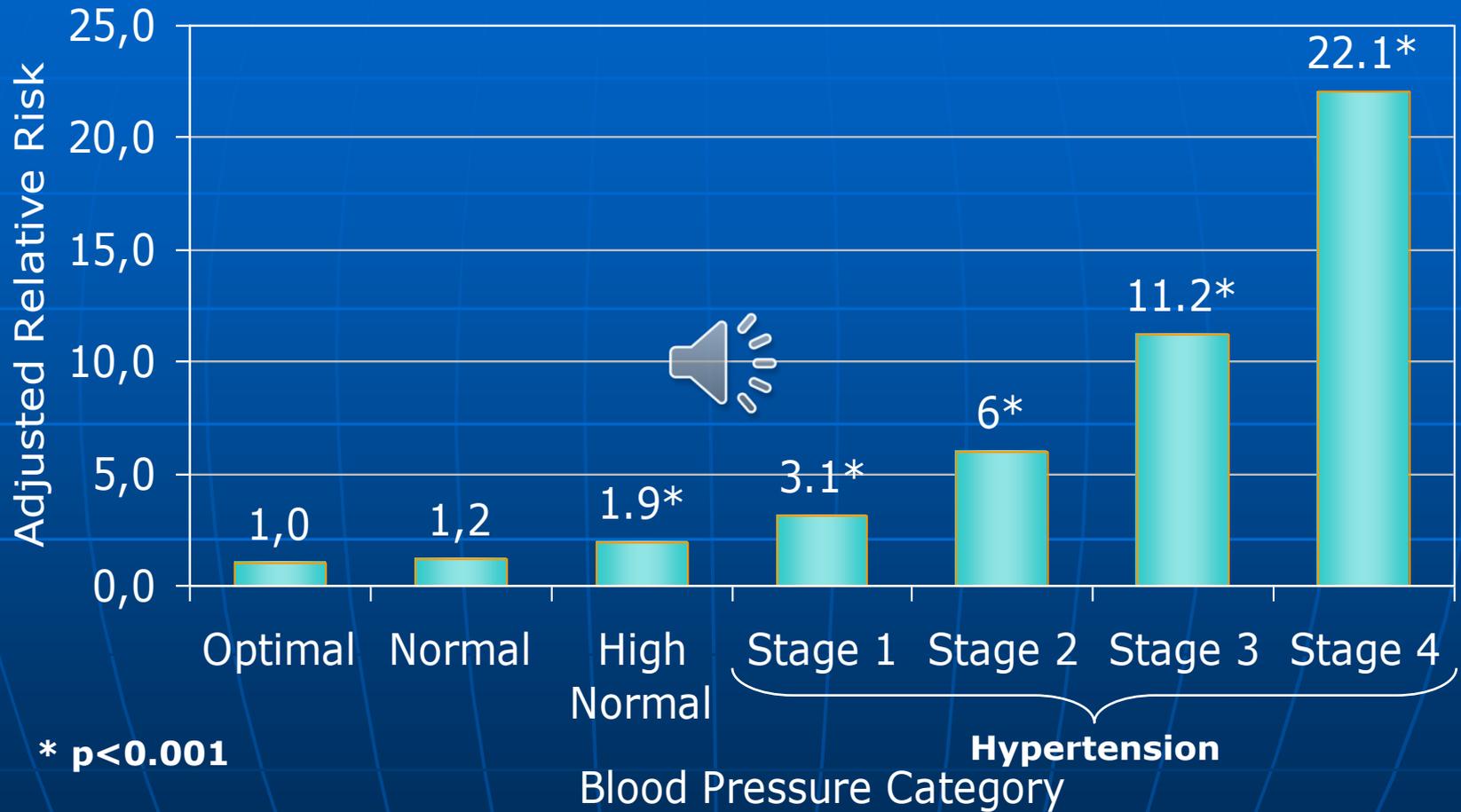
GLUCOSE

BP

LIPIDS

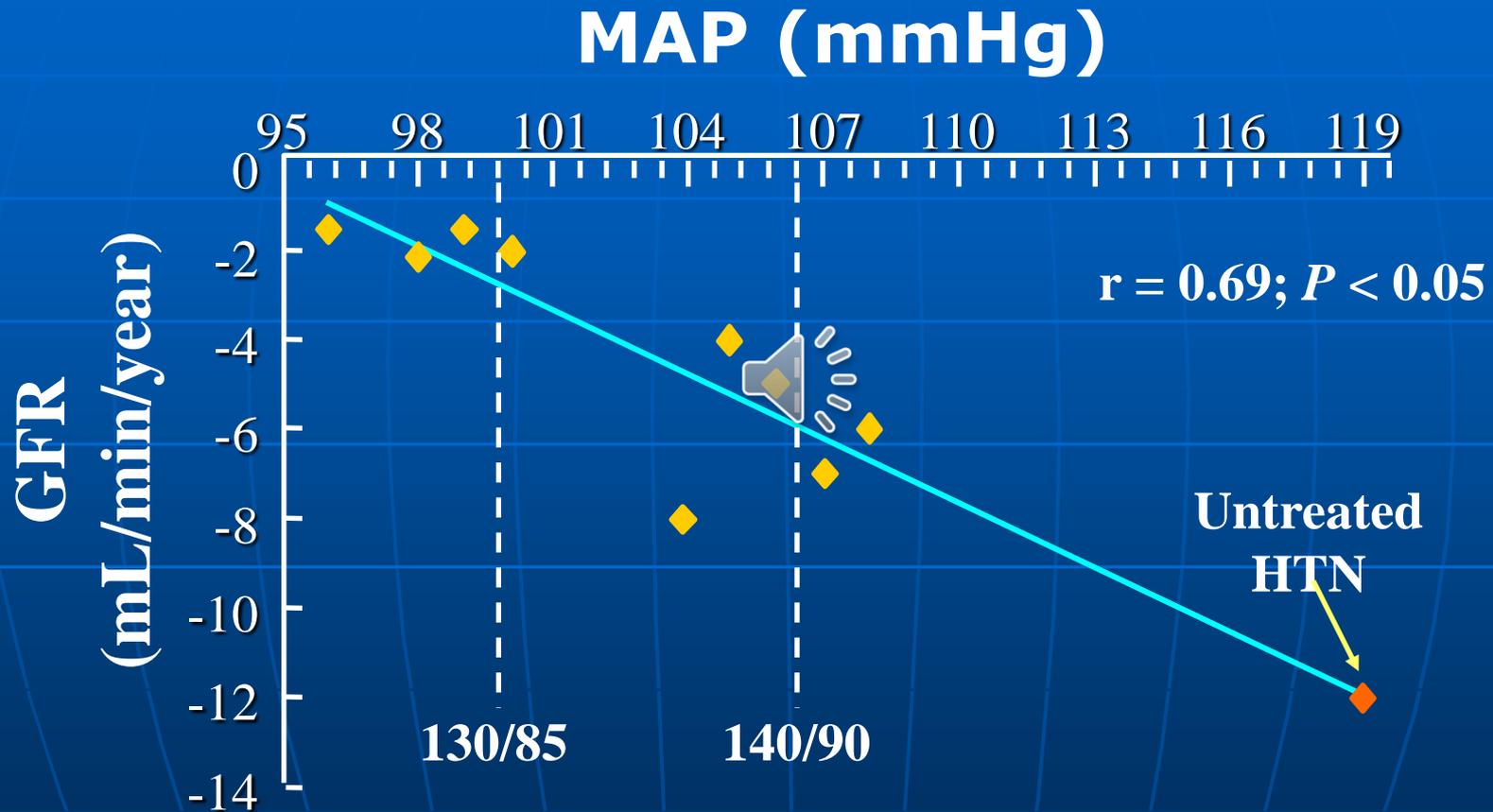


# ESRD Due to Any Cause In 332,544 Men Screened for MRFIT Adjusted Relative Risk<sup>§</sup>



§ Men with optimal blood pressure was the reference category.

# Meta Analysis: Lower Mean BP Results in Slower Rates of Decline in GFR in Diabetics and Non-Diabetics

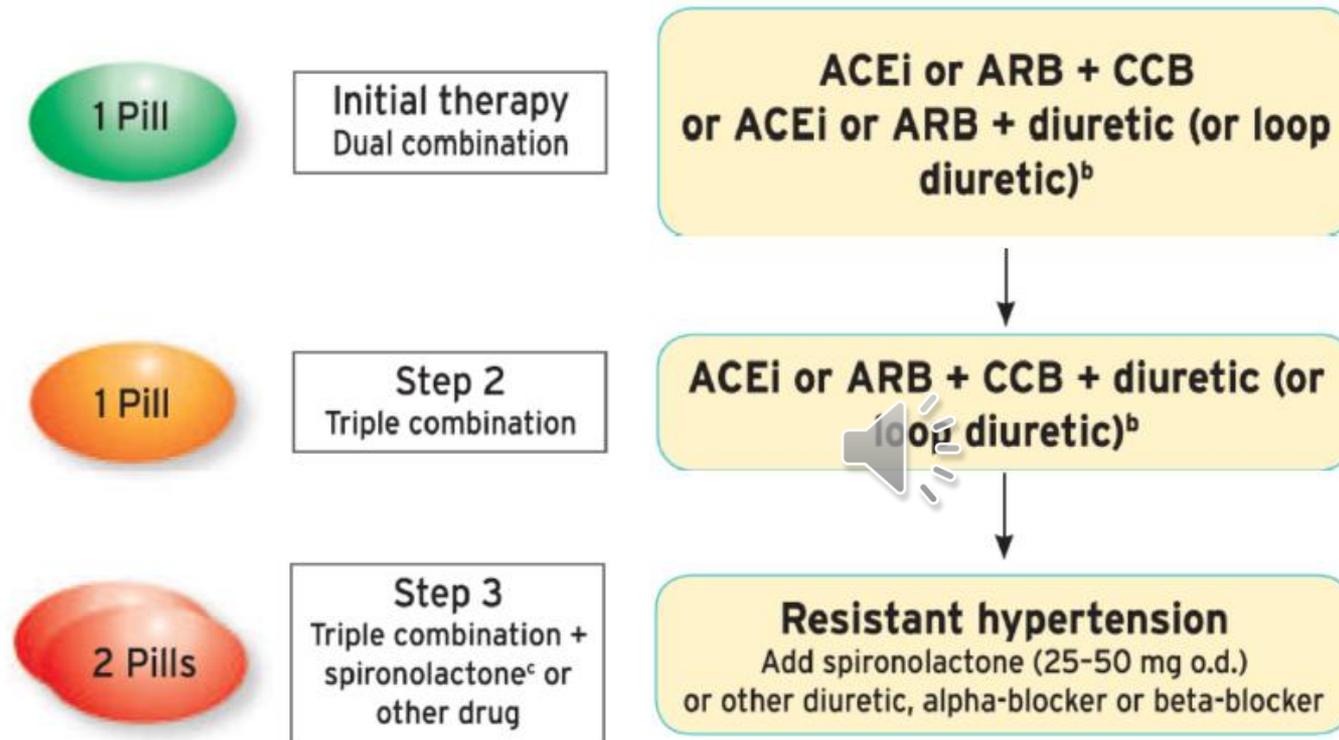


Bakris GL, et al. Am J Kidney Dis. 2000;36(3):646-661.

# Office BP treatment target range

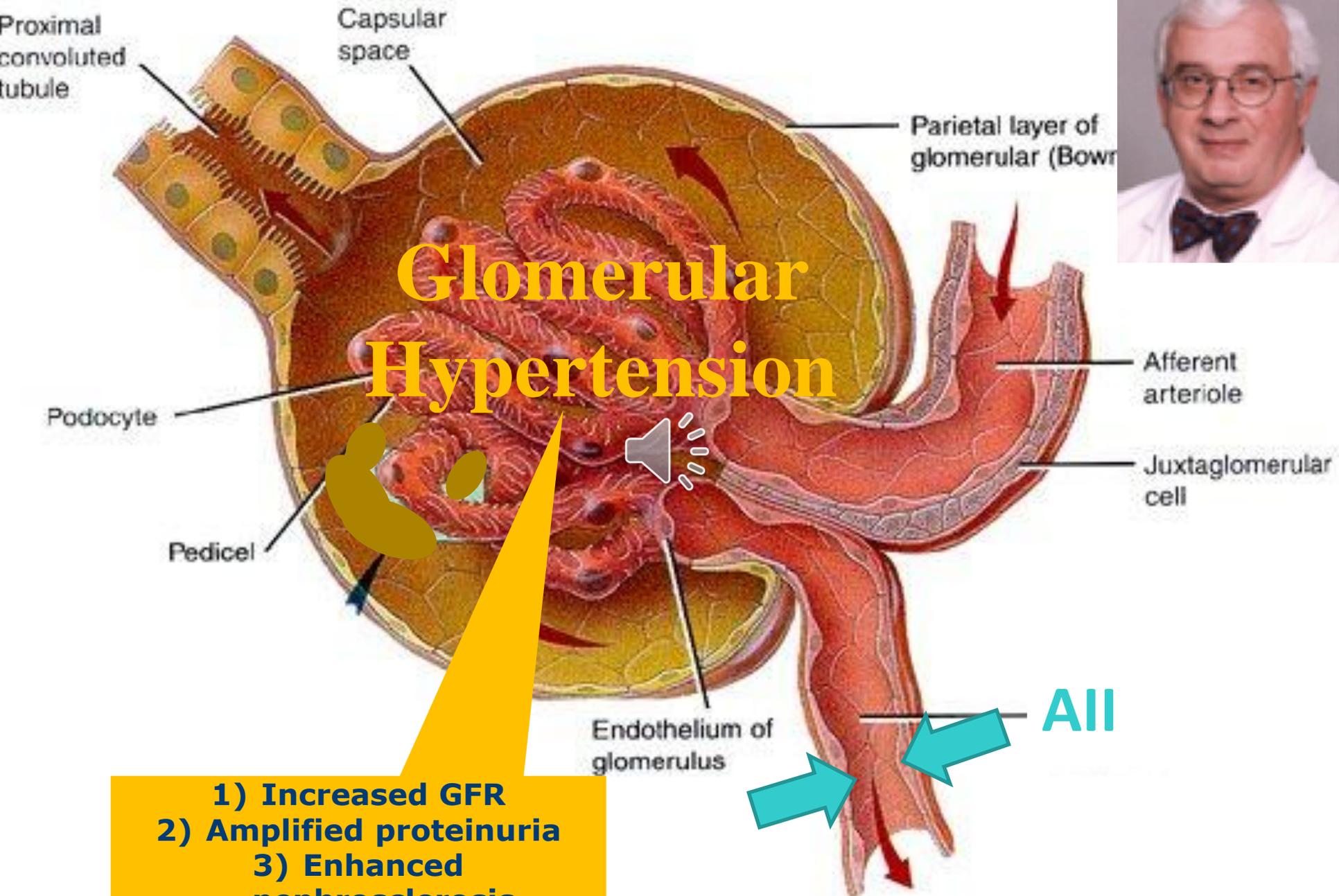
Age group	Office SBP treatment target ranges (mmHg)					Office DBP treatment target range (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke/TIA	
18–65 years	Target to 130 <i>or lower if tolerated</i> Not < 120	Target to 130 <i>or lower if tolerated</i> Not < 120	Target to < 140 to 130 <i>if tolerated</i>	Target to 130 <i>or lower if tolerated</i> Not < 120	Target to 130 <i>or lower if tolerated</i> Not < 120	70-79
65–79 years	Target to < 140 to 130 <i>if tolerated</i>	Target to < 140 to 130 <i>if tolerated</i>	Target to < 140 to 130 <i>if tolerated</i>	Target to < 140 to 130 <i>if tolerated</i>	Target to < 140 to 130 <i>if tolerated</i>	70-79
≥ 80 years	Target to < 140 to 130 <i>if tolerated</i>	Target to < 140 to 130 <i>if tolerated</i>	Target to < 140 to 130 <i>if tolerated</i>	Target to < 140 to 130 <i>if tolerated</i>	Target to < 140 to 130 <i>if tolerated</i>	70-79
Office DBP treatment target range(mmHg)	70-79	70-79	70-79	70-79	70-79	

# Drug-treatment strategy for hypertension and CKD



**Beta-blockers**  
Consider beta-blockers at any treatment step, when there is a specific indication for their use, e.g. heart failure, angina, post-MI, atrial fibrillation, or younger women with, or planning, pregnancy

A reduction in eGFR and rise in serum creatinine is expected in patients with CKD<sup>a</sup> who receive BP-lowering therapy, especially in those treated with an ACEi or ARB but a rise in serum creatinine of >30% should prompt evaluation of the patient for possible renovascular disease.



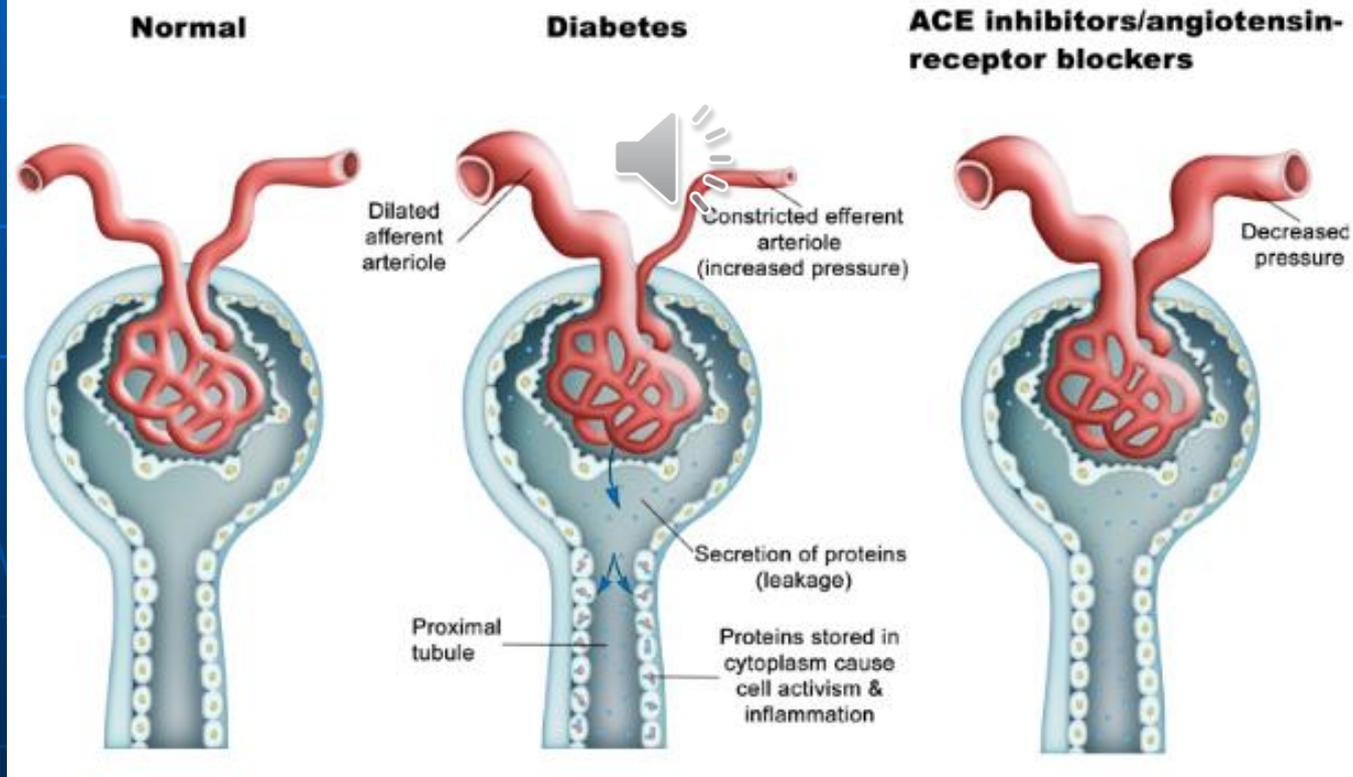
# Glomerular Hypertension

- 1) Increased GFR
- 2) Amplified proteinuria
- 3) Enhanced nephrosclerosis

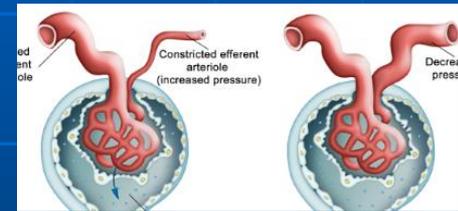
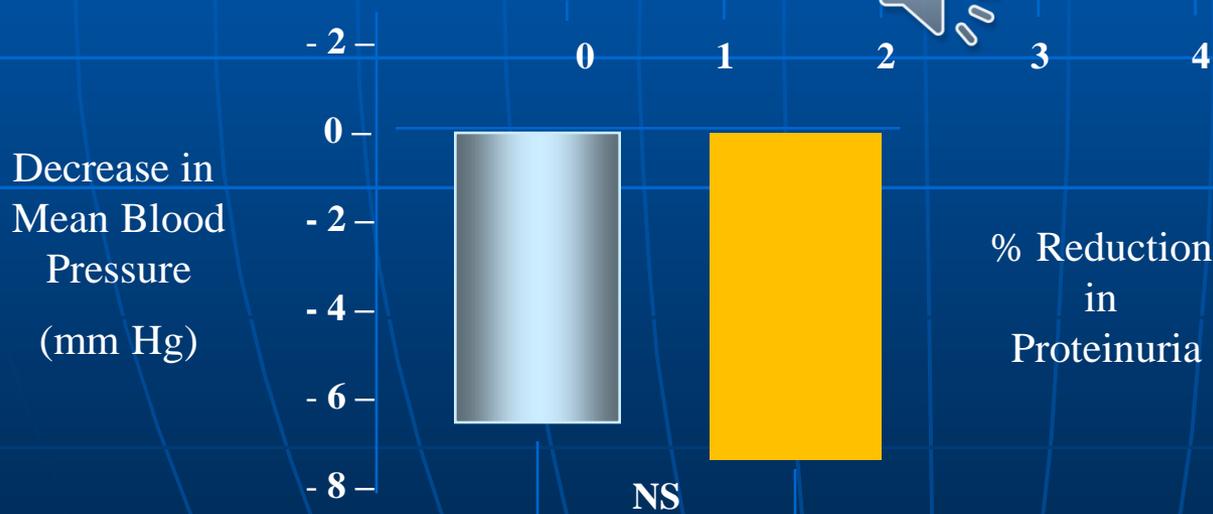
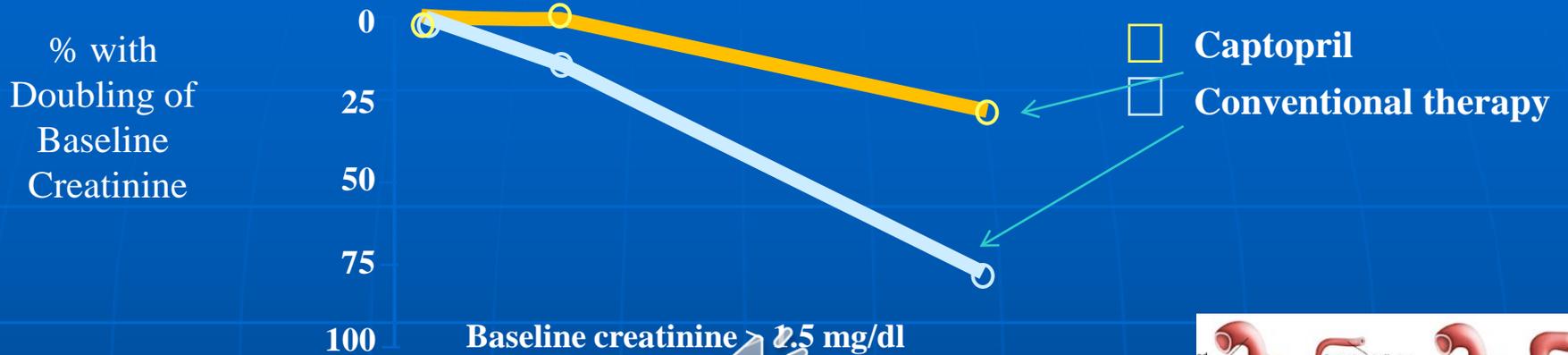
All

# Σπειραματική Υπέρταση από ενεργοποίηση του ΣΡΑΑ

Local effects of ARBs and ACEIs in the kidney in the patient with type-2 diabetes. Vasoconstriction in the efferent arteriole is reduced and less protein crosses the glomerular filter into the tubule of the nephron



# ACE-I is More Renoprotective than Conventional Therapy in Type 1 Diabetes (Total N = 409)

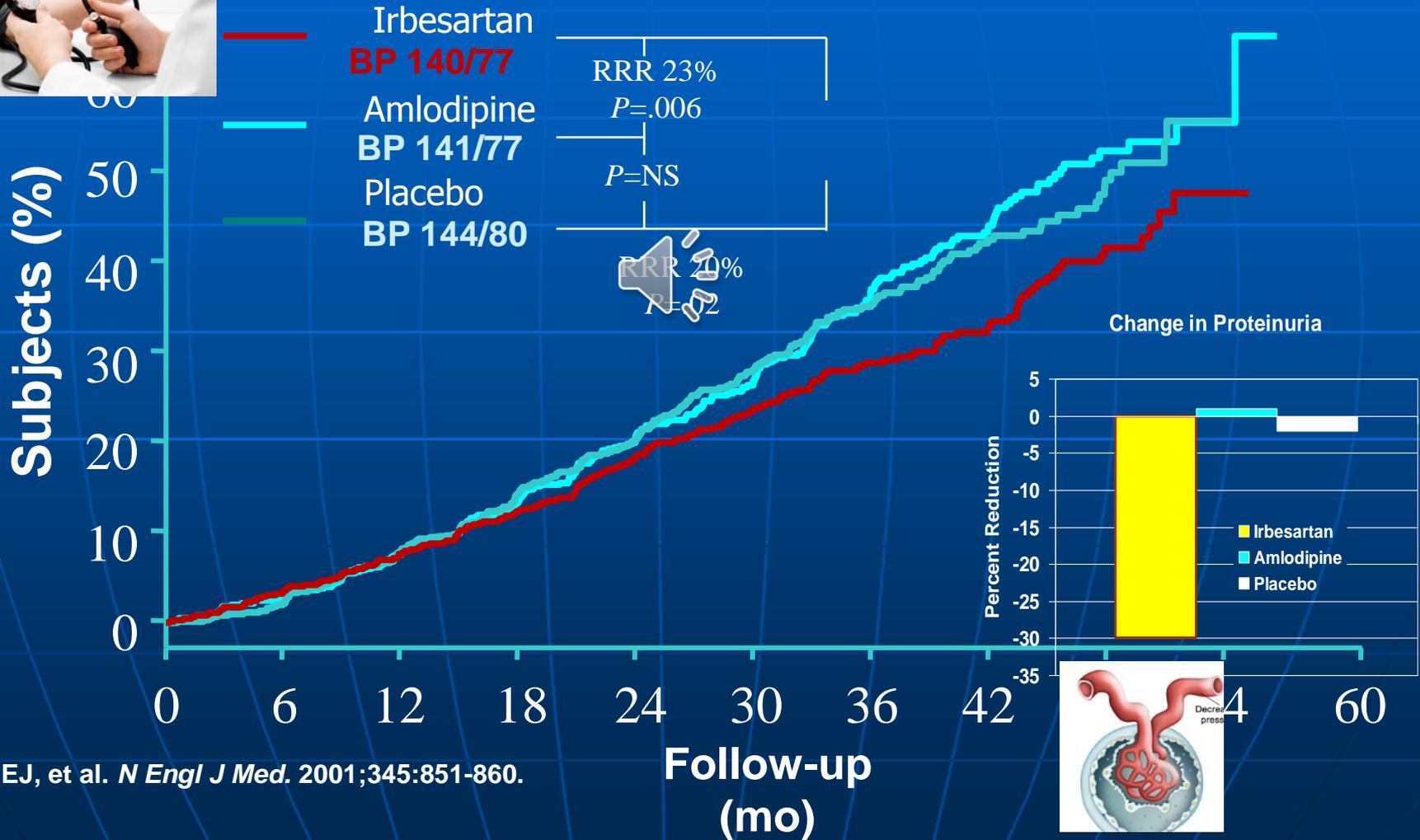


***P < .001***

Lewis et al. *N Engl J Med.* 1993;329:1456-1462.

# Irbesartan in Diabetic Nephropathy Trial: Time to Doubling of Serum Creatinine, ESRD, or Death

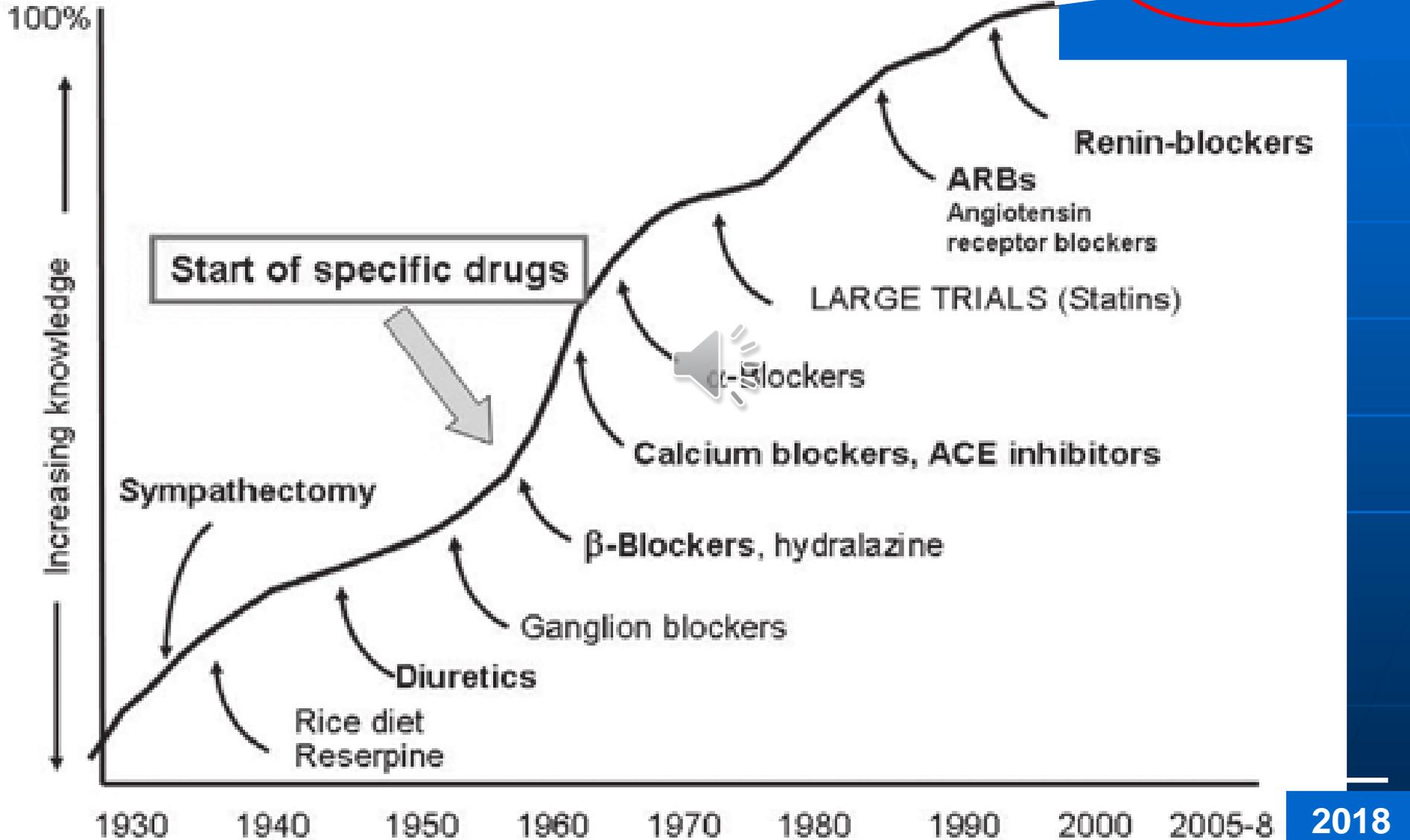
1,715 Type 2 Diabetics with Nephropathy



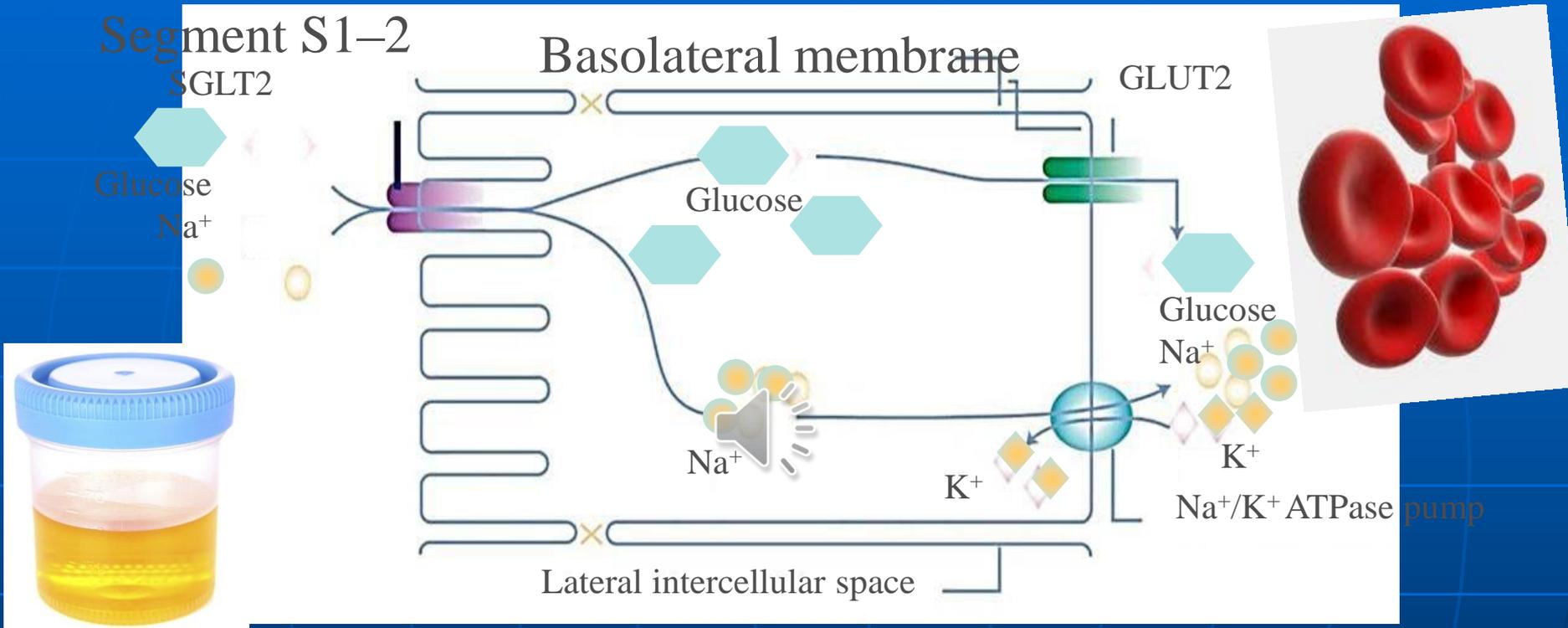
Lewis EJ, et al. *N Engl J Med.* 2001;345:851-860.

# Drugs for hypertension

SGLT – 2  
Inhibitors



# SGLT2 is a sodium glucose cotransporter<sup>1,2</sup>

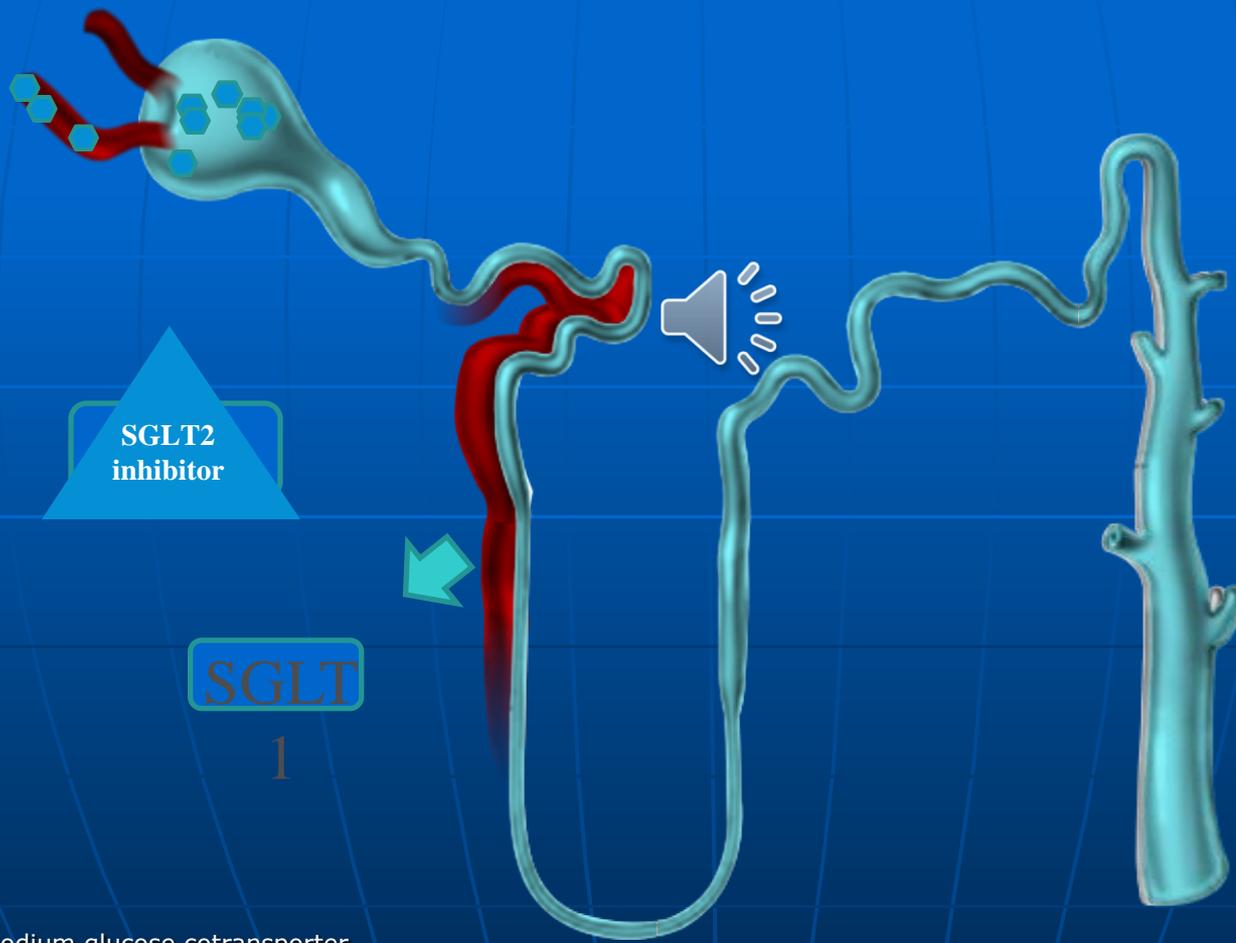


SGLTs transfer glucose and sodium (Na<sup>+</sup>:glucose coupling ratio for SGLT1 = 2:1 and for SGLT2 = 1:1) from the lumen into the cytoplasm of tubular cells through a secondary active transport mechanism

GLUT, glucose transporter; SGLT, sodium glucose cotransporter.  
1. Wright EM, et al. *Physiology*. 2004;19:370-376. 2. Bakris GI, et al. *Kidney Int*. 2009;75:1272-1277.  
3. Mather A, Pollock C. *Kidney Int Suppl*. 2011;120:S1-S6.

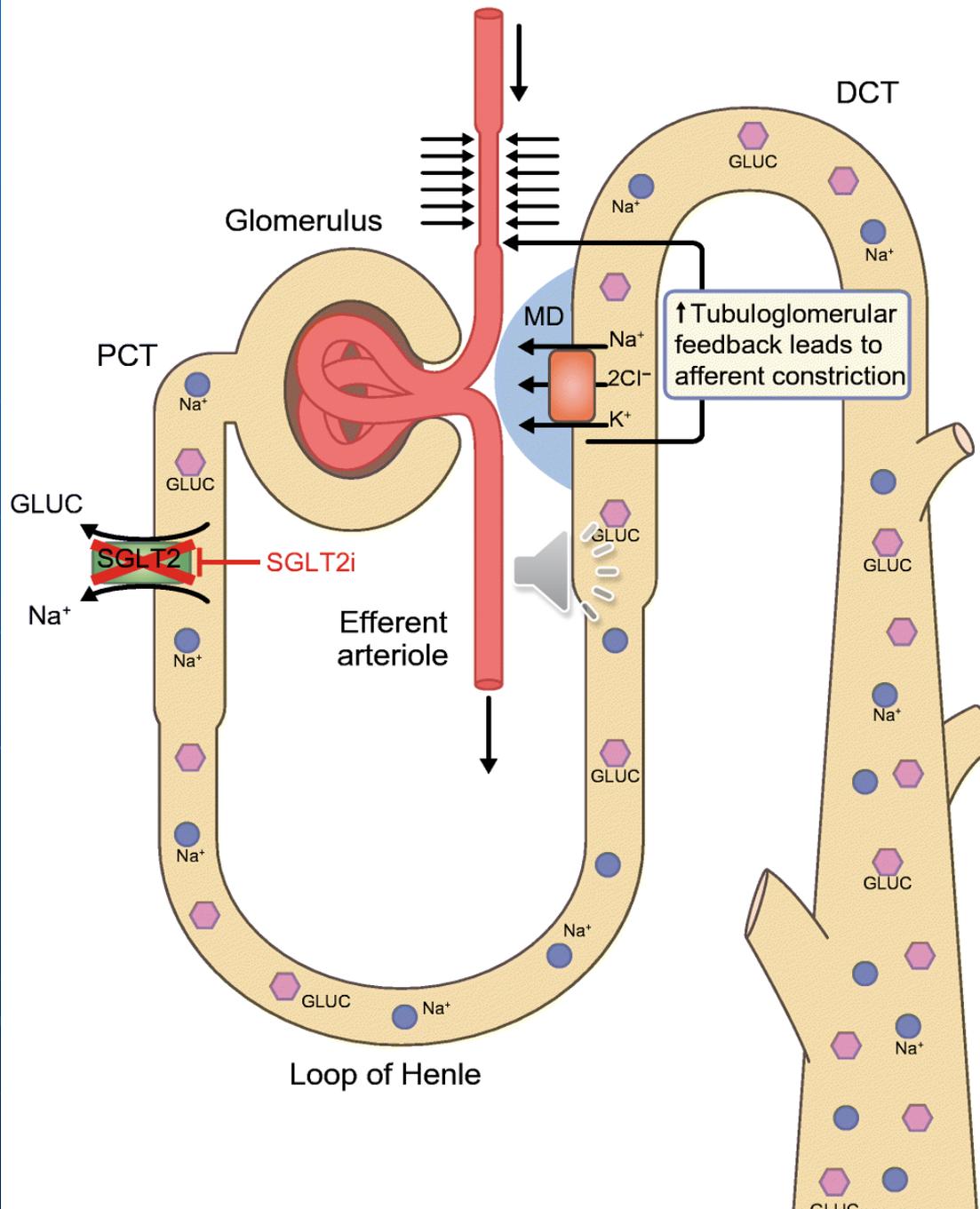
# Urinary glucose excretion via SGLT2 inhibition<sup>1</sup>

Filtered glucose load > 180 g/day

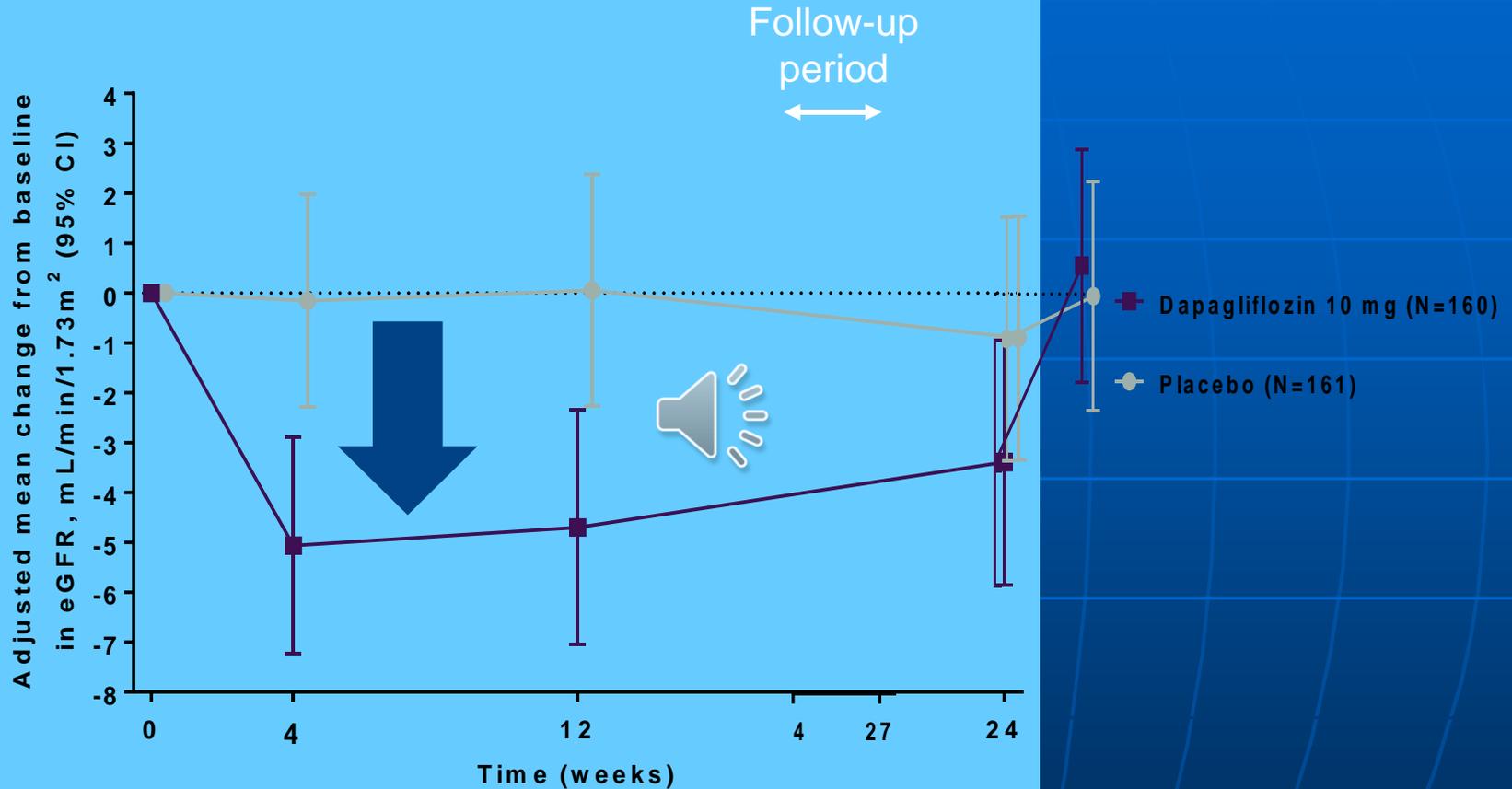


SGLT2 inhibitors reduce glucose re-absorption in the proximal tubule, leading to urinary glucose excretion\* and osmotic diuresis

SGLT, sodium glucose cotransporter.  
\*Loss of ~ 80 g of glucose per day = 320 cal/day.  
1. Bakris GL, et al. *Kidney Int.* 2009;75;1272-1277.

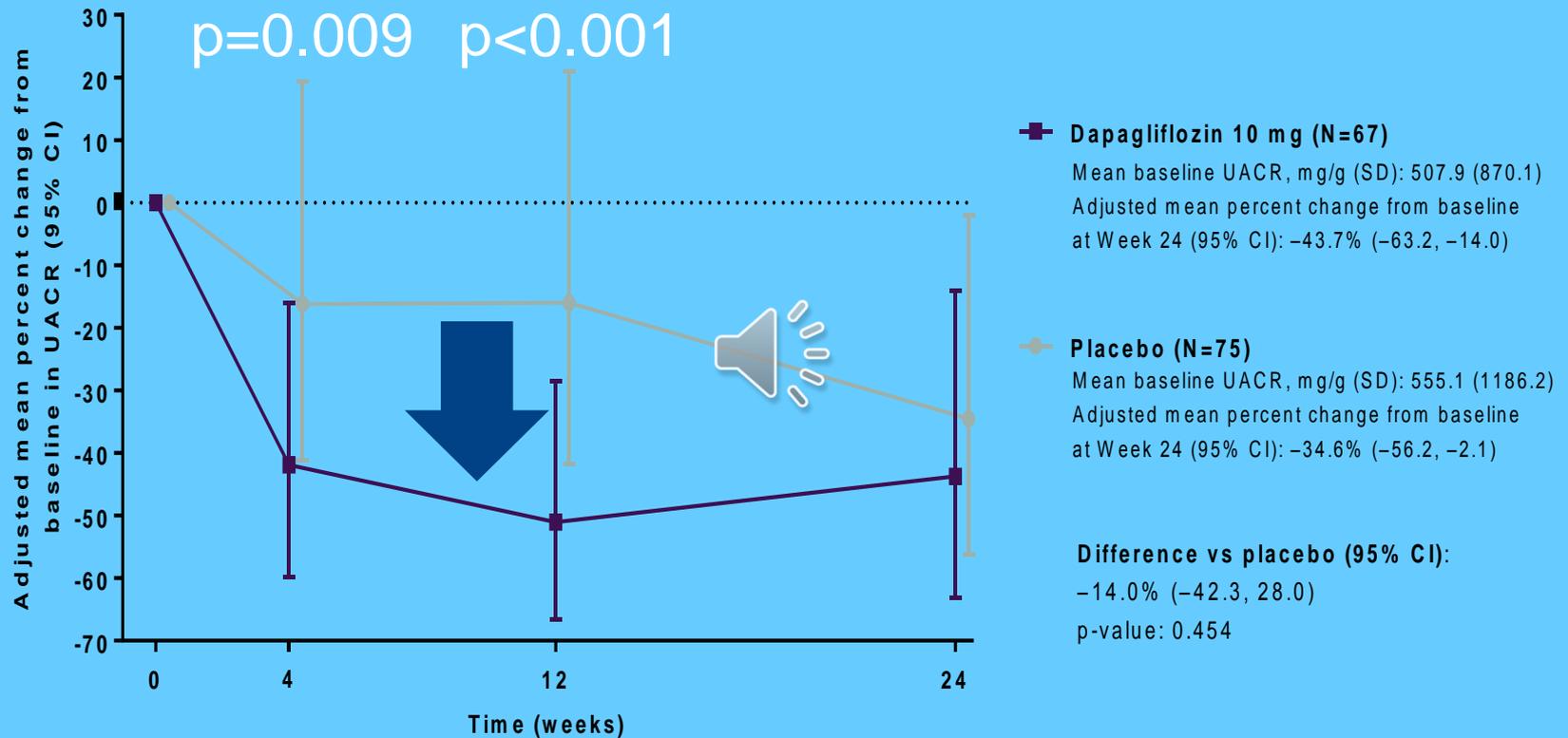


# eGFR – change from baseline over 24 week



Dapagliflozin	155	148	150	142
Placebo	157	152	151	140

# UACR – change from baseline over 24 weeks\*1



Dapagliflozin	67	67	66	60
Placebo	75	74	72	69

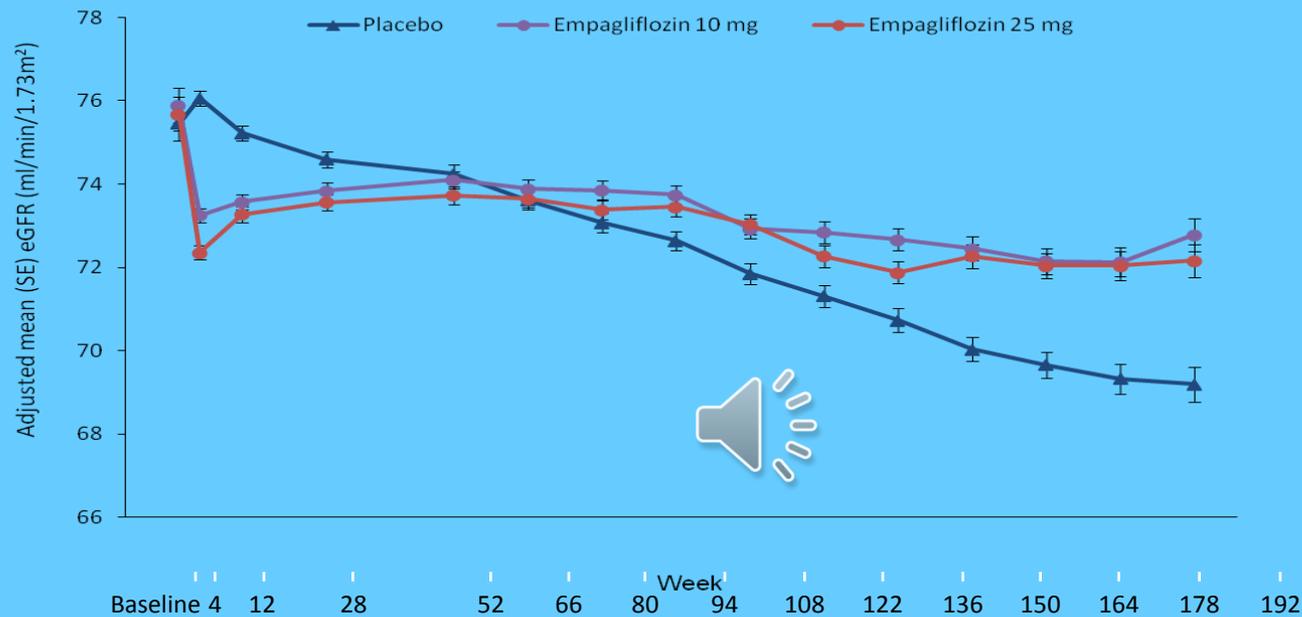
1. Fioretto P et al. Presented at: The Endocrine Society Annual Meeting and Expo; March 17-20, 2018; Chicago, Illinois

Data are based on the full analysis set.

\*Patients with baseline UACR  $\geq 30$  mg/g.

CI, confidence interval; SD, standard deviation; UACR, urine albumin:creatinine ratio

# eGFR (CKD-EPI) over 192 weeks



## No. analyzed

Placebo	2323	2295	2267	2205	2121	2064	1927	1981	1763	1479	1262	1123	977	731	448
Empagliflozin 10 mg	2322	2290	2264	2235	2162	2114	2012	2064	1839	1540	1314	1180	1024	785	513
Empagliflozin 25 mg	2322	2288	2269	2216	2156	2111	2006	2067	1871	1563	1340	1207	1063	838	524

## No. in follow-up for adverse/outcome events

Total	7020	7020	6996	6931	6864	6765	6696	6651	6068	5114	4443	3961	3488	2707	1703
-------	------	------	------	------	------	------	------	------	------	------	------	------	------	------	------

Pre-specified mixed model repeated measures analysis in all patients treated with  $\geq 1$  dose of study drug who had a baseline and post-baseline measurement.  
 eGFR, estimated glomerular filtration rate; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration.

# META-ANALYSIS OF CVOTs: RENAL END-POINTS BY PRESENCE

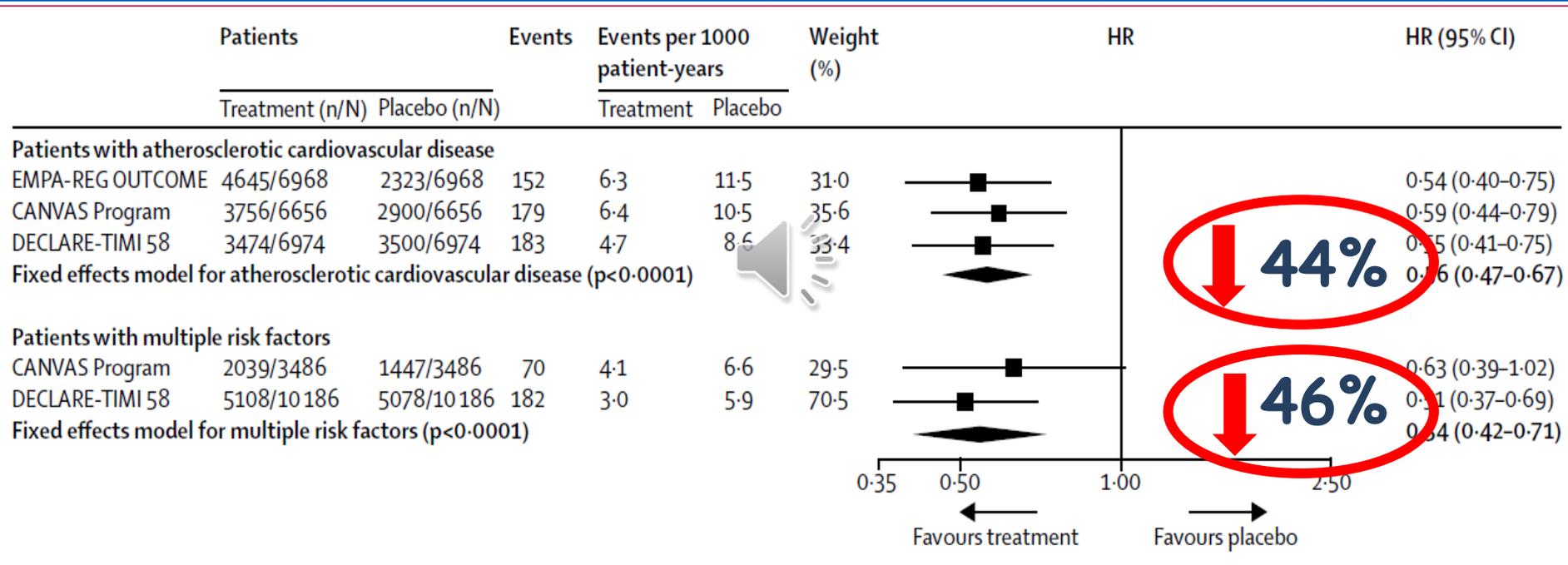


Figure 4: Meta-analysis of SGLT2i trials on the composite of renal worsening, end-stage renal disease, or renal death stratified by the presence of established atherosclerotic cardiovascular disease

**XNA  KAI  
ANAIMIA**

# Diagnosis of anemia

- Diagnose anemia in adults and children >15 years with CKD when the Hb concentration is **<13.0 g/dl in males and <12.0 g/dl in females** 
- Diagnose anemia in children with CKD if Hb concentration is <11.0 g/dl in children 0.5–5 years, <11.5 g/dl in children 5–12 years, and <12.0 g/dl in children 12–15 years.



# Mechanisms of Anaemia in CKD

Haemodilution

Plasma Volume ↑

Chronic inflammation

TNF $\alpha$  - production of Epo ↓  
- Epo activity in BM ↓

Forward failure

Bone Marrow (BM)

- dysfunction

“UREMIC TOXINS”

Drugs

 RAS INHIBITION:  
Epo synthesis ↓  
Epo activity in BM ↓

Iron deficiency

Fe<sup>++</sup> uptake ↓

malabsorption

chron. bleeding (Aspirin)

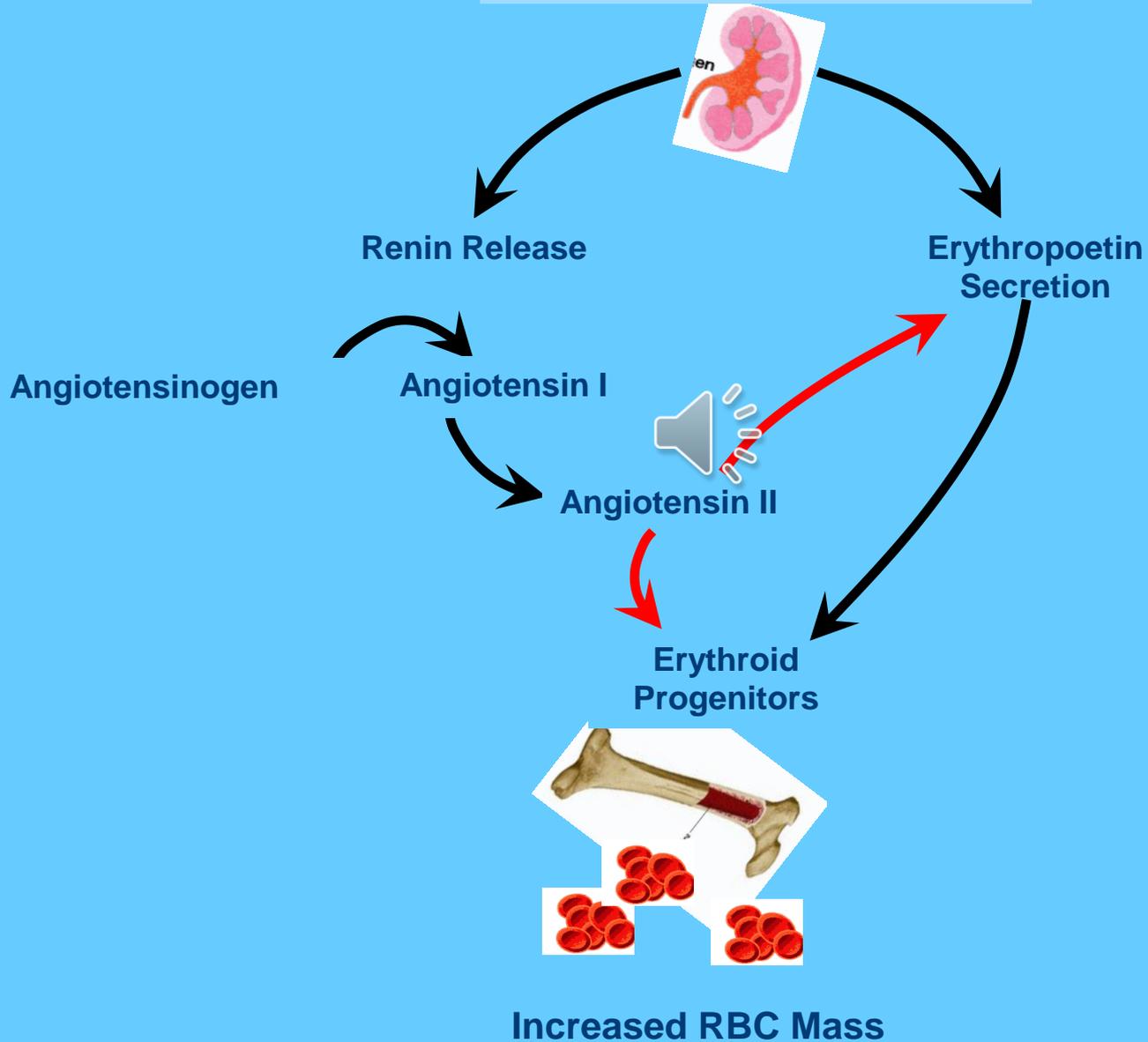
Chronic kidney failure

Production of Epo ↓

Loss in urine ↑

# Vlahakos et al. AJKD 2010

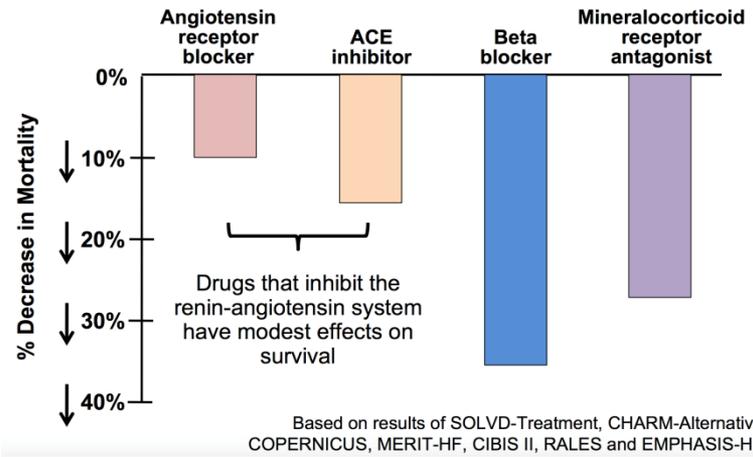
Renal Hypoperfusion (Hypoxia)



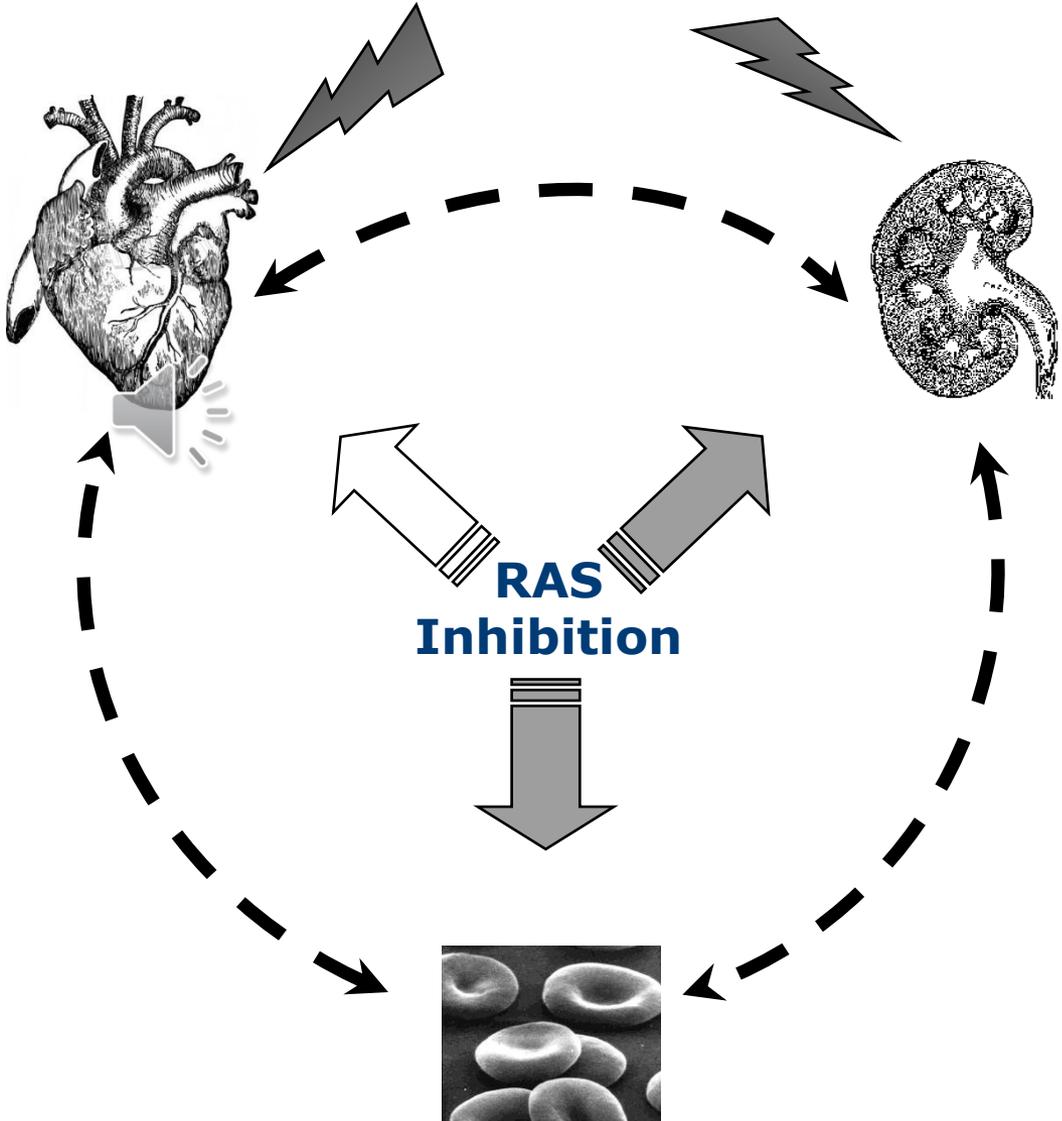
# Hematocrit lowering effect +/- anemia after RAS blockade

## Marathias et al. AJN, 2020

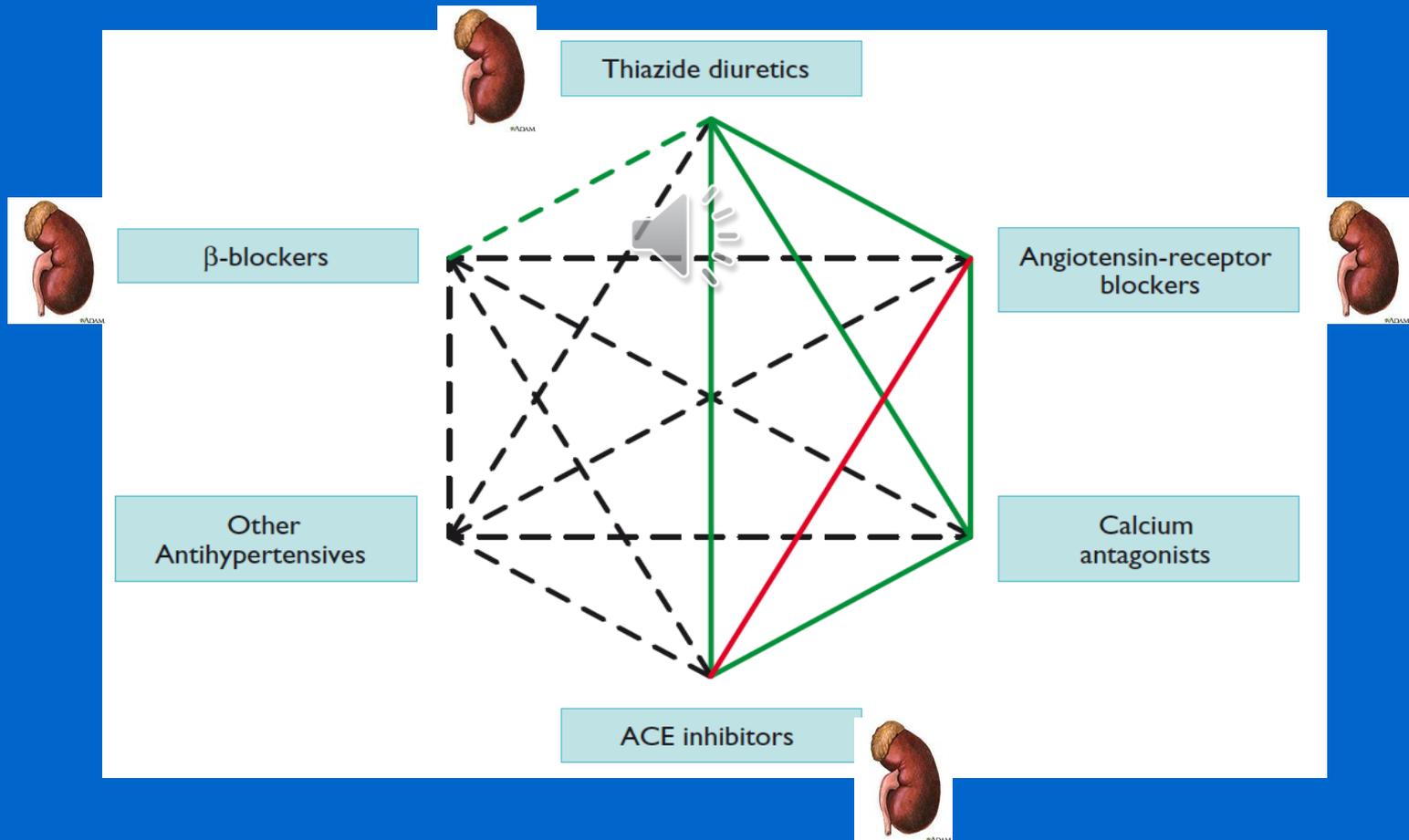
Reference /year	No of patients	Hct pre %	Hct post %	P value	Clinical Setting	Drug	Duration months
Kamper <sup>44</sup> 1990	27	36.7	32.4	0.01	Adults with <u>CKD</u>	Enalapril	3
Vlahakos <sup>60</sup> 19 91	10	42	33	0.001	<u>Renal transplant recipients</u>	Enalapril	3
Gaston <sup>61</sup> 1991	12	57	47	0.001	Posttransplant <u>erythrocytosis</u>	Enalapril	9
Herrlin <sup>62</sup> 1991	8	42.6	39	0.01	Stable Moderate <u>Heart Failure</u>	Enalapril	3
Barr <sup>63</sup> 1997	41	41.4	37.7	0.01	Early <u>Heart Failure</u>	Enalapril	12
Mazzali <sup>64</sup> 1998	27	56	46	0.05	Posttransplant <u>erythrocytosis</u>	Enalapril	3
Vlahakos <sup>41</sup> 20 01	9	56	46	0.001	COPD with <u>polycythemia</u>	Losartan	1
Plata <sup>39</sup> 2002	13	63.5	56.8	0.0001	Altitude <u>polycythemia</u>	Enalapril	12
Iodice <sup>65</sup> 2003	10	43.8	41.4	0.01	Nonnephrotic <u>proteinuria;</u> normal renal function	Ramipril	4
Wühl <sup>66</sup> 2004	352	36.6	34.8	0.005	Children with <u>CKD</u>	Ramipril	6



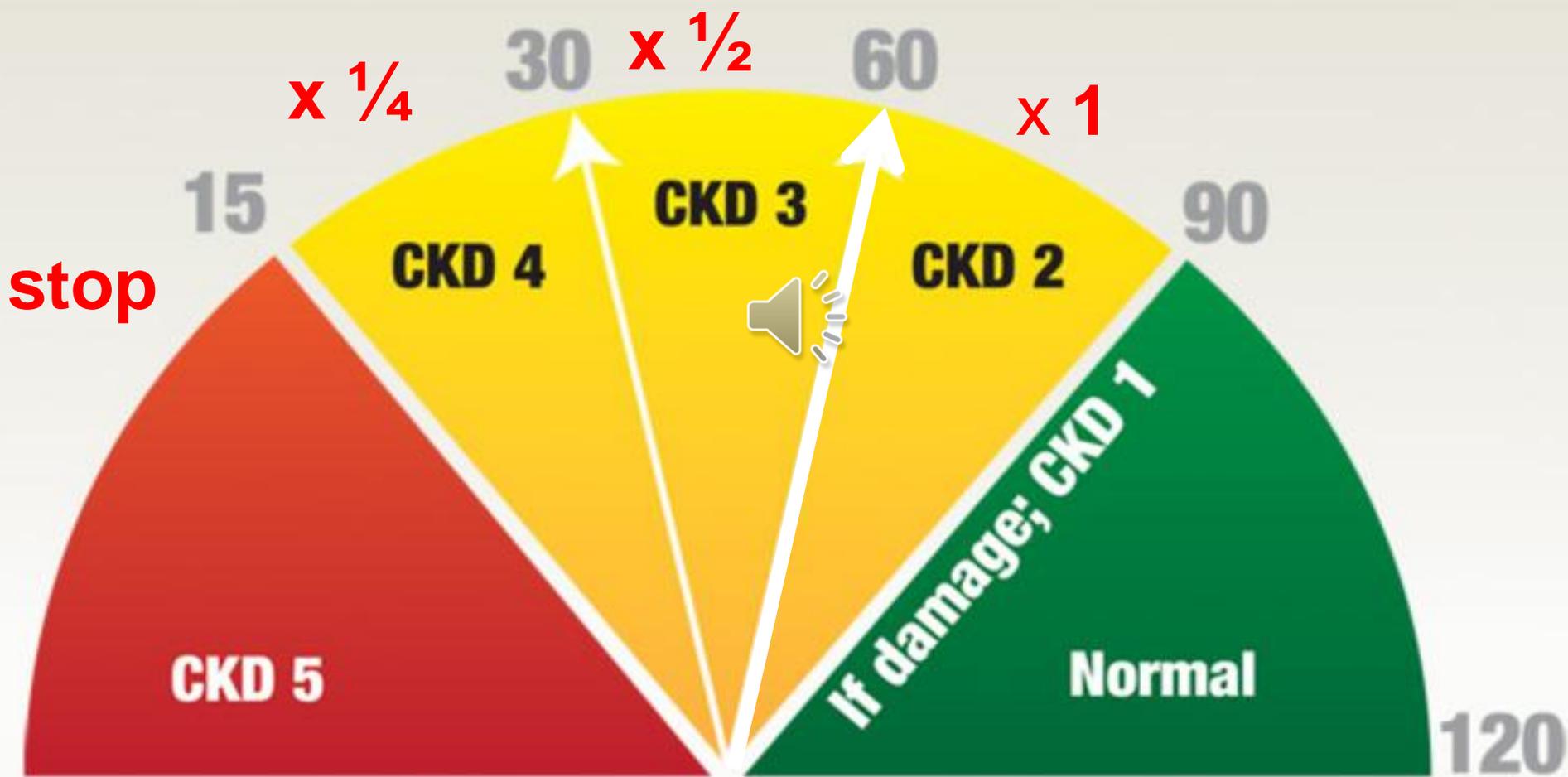
**Aging/Hypertension  
Obesity/Diabetes**



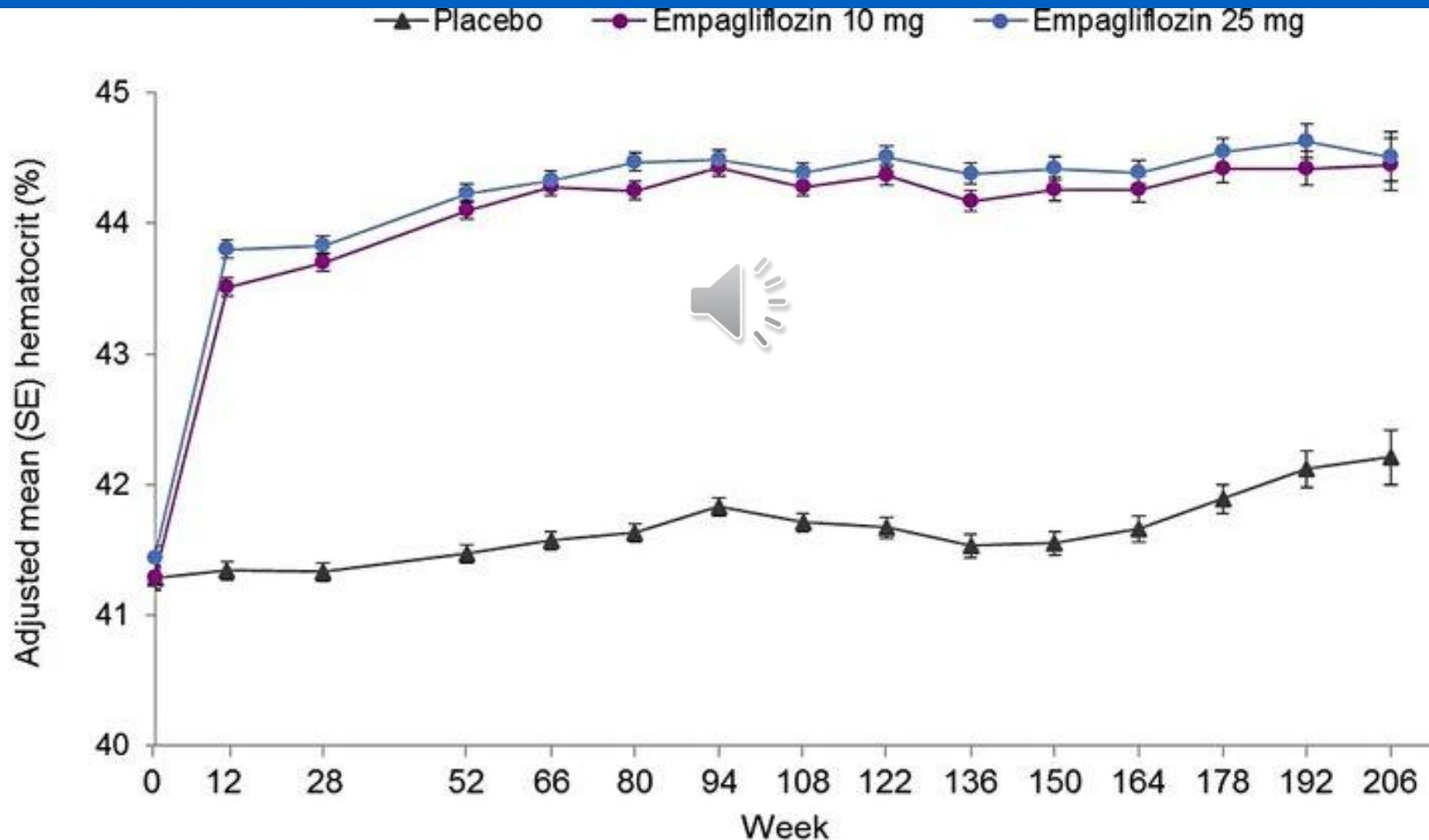
# Possible Combinations Of Antihypertensive Drug Classes



# Προσαρμογή δόσης αΜΕΑ ή ARB:



# Increased Hematocrit values with SGLT-2 inhibitors



# Hematocrit increases after administration of SGLT2 inhibitors

## Marathias et al. AJN, 2020

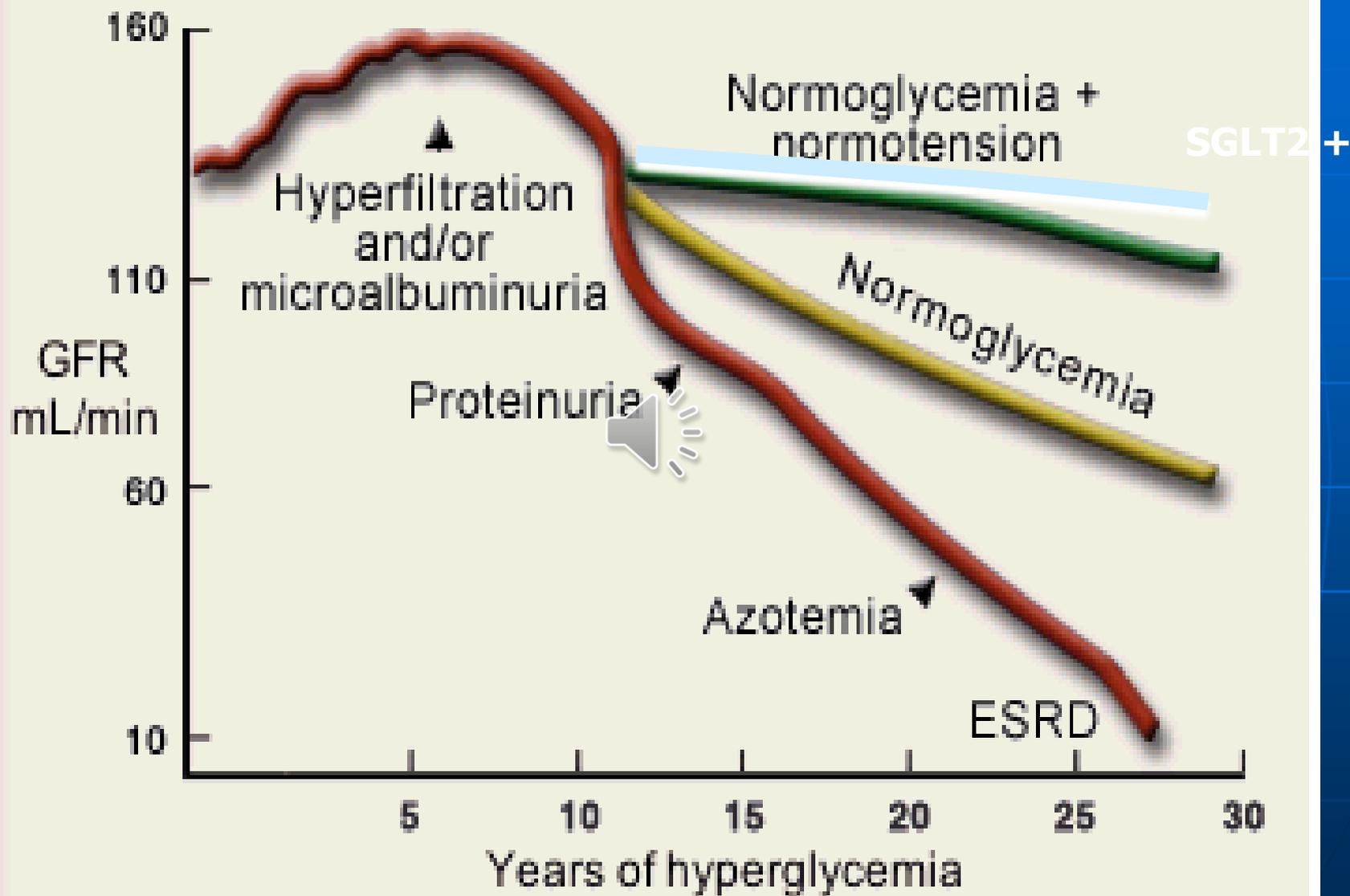
**Table 2.** A modest increase in hematocrit values by 2–4% points is consistently observed following administration of all 6 gliflozins (empagliflozin, canagliflozin, dapagliflozin, ipragliflozin, ertugliflozin and sotagliflozin)

Reference, year	Number of patients	Hct pre, %	Hct post, %	Clinical condition	Drug	Duration, months
Zinman et al. [69], 2015	7,020	41.20	46.00	T2DM at high CV risk	Empagliflozin	30
Januzzi et al. [70], 2017	450	41.00	43.00	Older subjects with T2DM	Canagliflozin	25
Kimura et al. [52] 2018	31	39.10	41.70	T2DM and hypertension	Ipragliflozin	3
Maruyama et al. [56], 2019	9	37.10	40.40	T2DM with anemia	Canagliflozin	3
van Raalte et al. [71], 2019	1,049	42.00	44.00	T1DM	Sotagliflozin	13
Gallo et al. [72], 2019	199	42.12	45.13	T2DM	Ertugliflozin	3
van Bommel et al. [73], 2020	24	40.70	42.50	T2DM	Dapagliflozin	3

T1DM, type 1 diabetes mellitus; T2DM, type 2 diabetes mellitus.

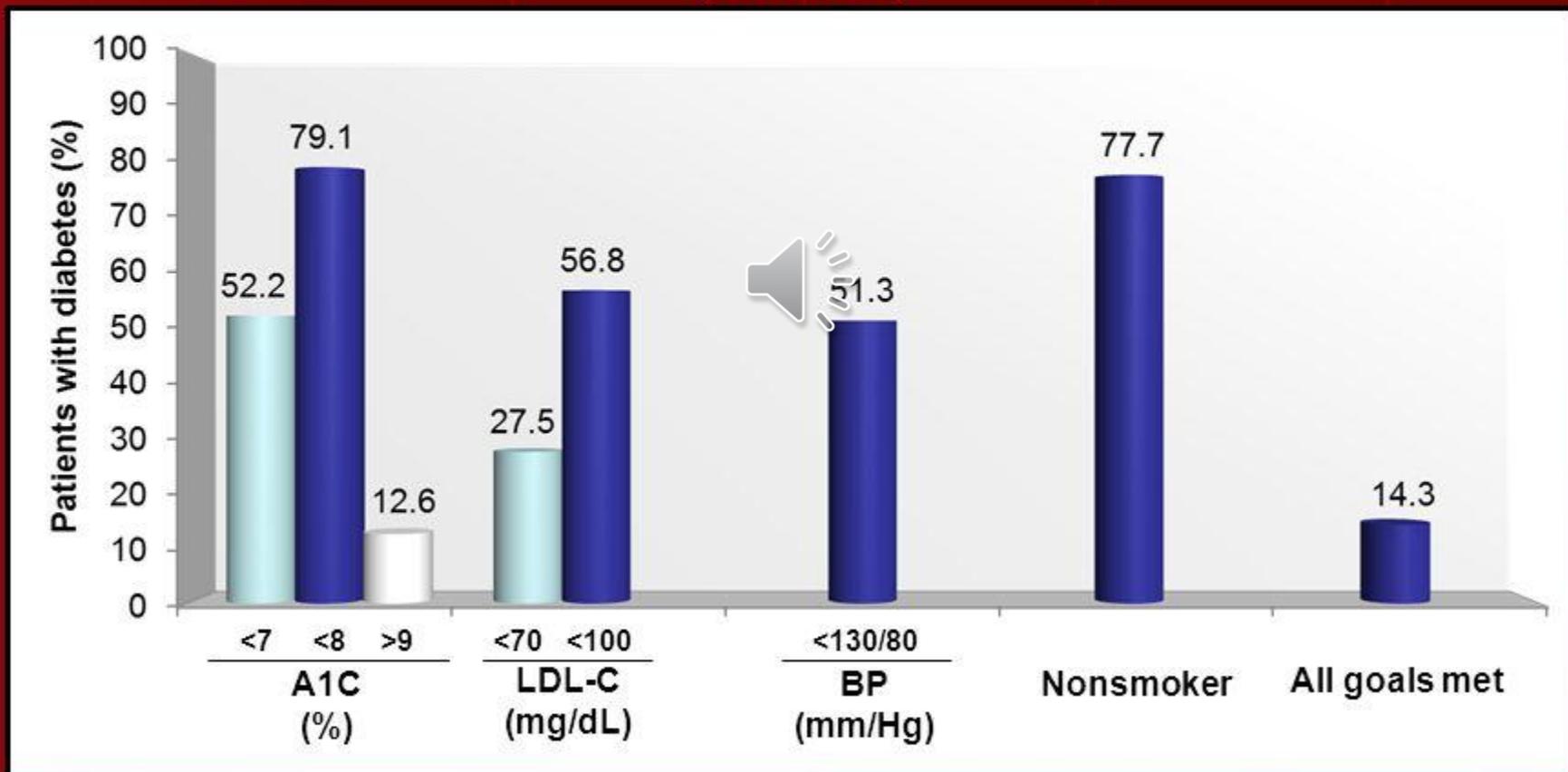
40% > 43%

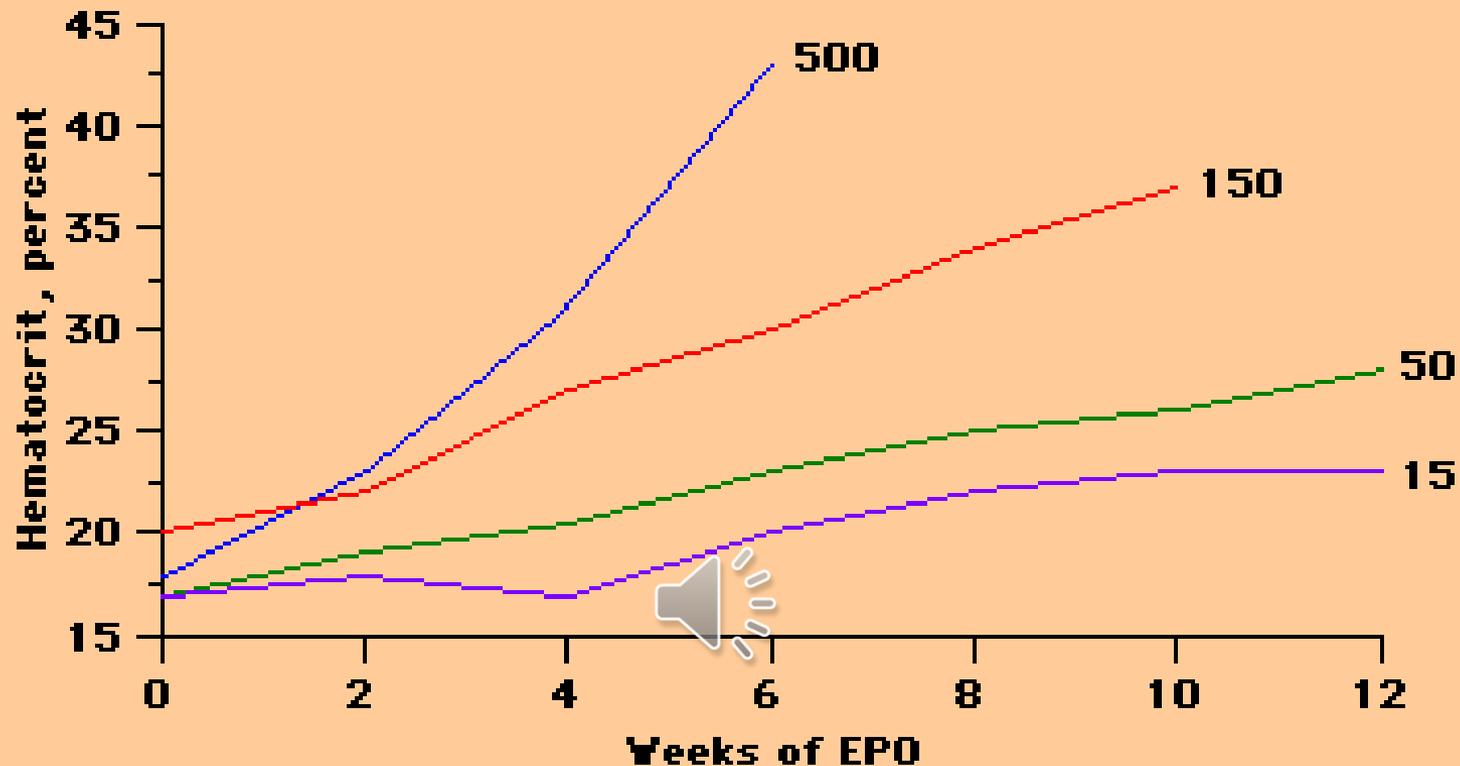
# Glomerular filtration rate (GFR) in diabetes



# Goal Achievement in Diabetes

NHANES 2007-2010  
(N=1444)

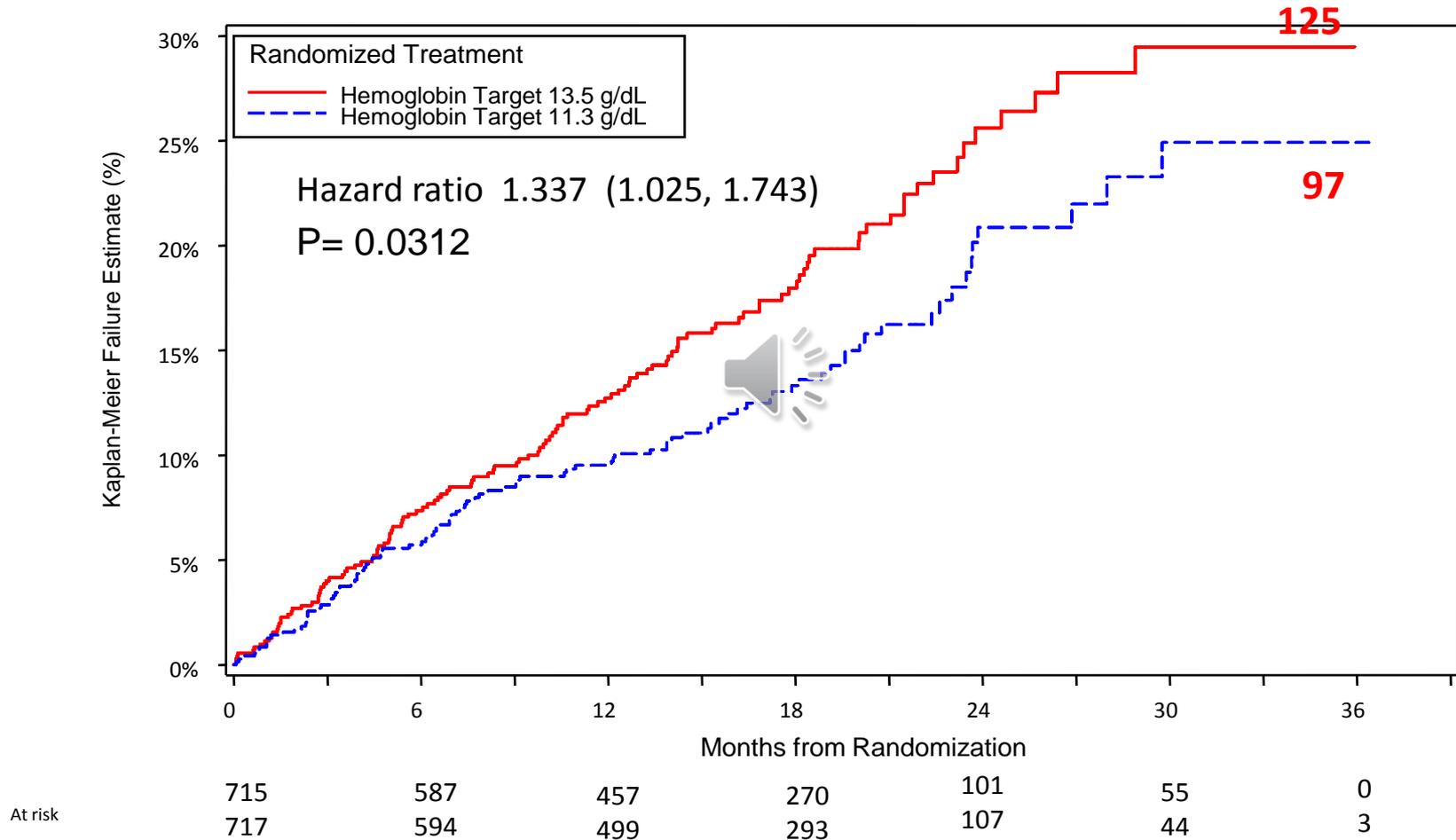




### **Dose response of EPO-induced correction of uremic anemia**

Slope of the rate of increase in the hematocrit in patients on maintenance dialysis given various doses (15 to 500 units/kg) of erythropoietin (EPO) three times per week. The response is fastest at the highest dose, but a gradual and adequate rise in hematocrit is achieved in most patients with 50 units/kg. (Data from Eschbach, JW, Egrie, JC, Downing, MR, et al, N Engl J Med 1987; 316:73.)

# Kaplan-Meier Plot of the Time to the Primary Composite Event between Randomization and Termination: ITT Population



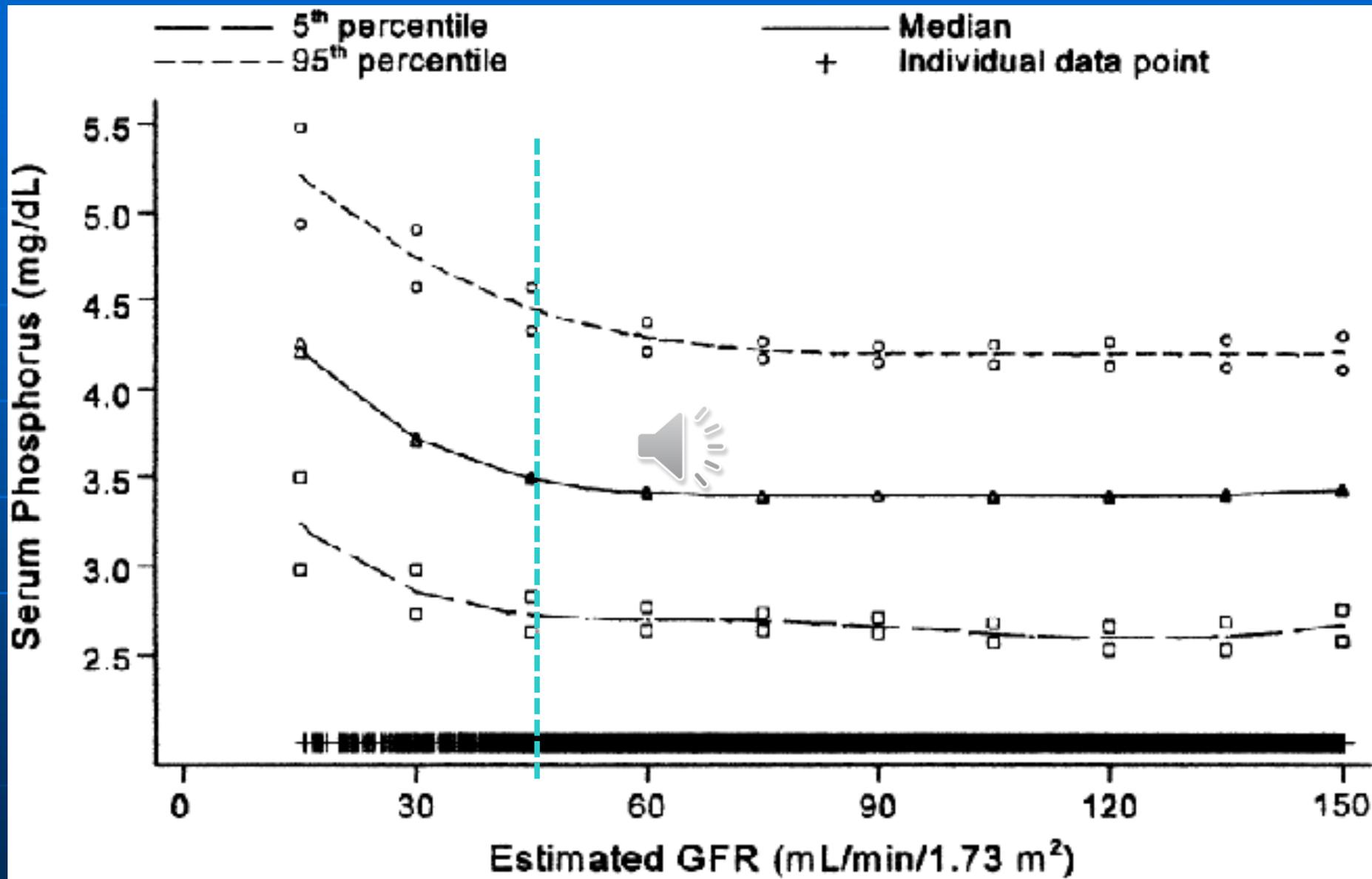
Primary Composite Endpoint:  
Death, MI, CHF hosp (no RRT) and/or stroke

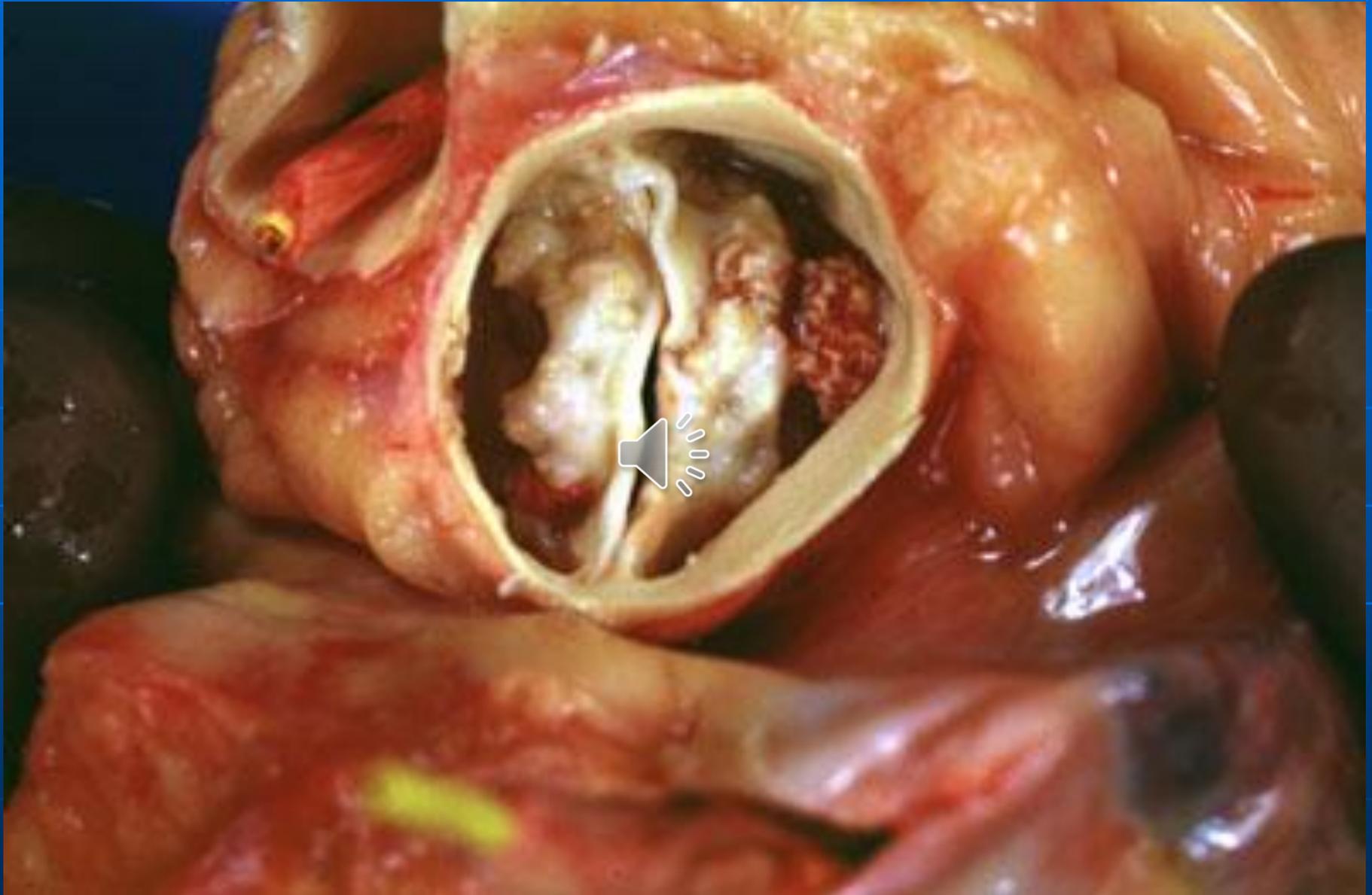
# NKF KDOQI Guidelines

- **2.1.2 In the opinion of the Work Group, in dialysis and nondialysis patients with CKD receiving ESA therapy, the selected Hb target should generally be in the range of **11.0 to 12.0 g/dL**. (Clinical Practice RECOMMENDATION)**

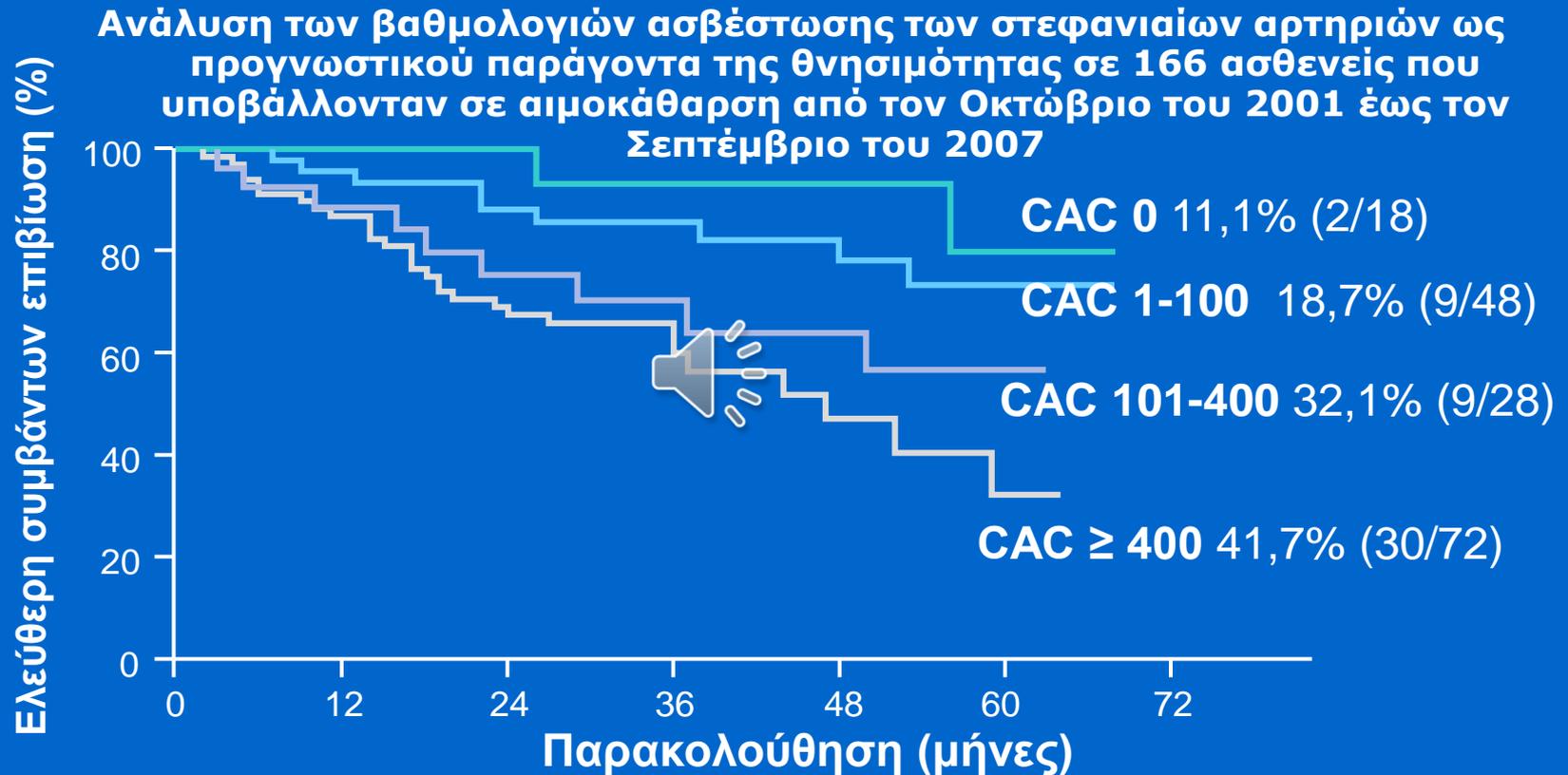
**2.1.3 In dialysis and nondialysis patients with CKD receiving ESA therapy, the Hb target should not be greater than 13.0 g/dL. (Clinical Practice GUIDELINE - MODERATELY STRONG EVIDENCE)**

ΧΝΑ  
ΚΑΙ  
ΟΣΤΙΚΟΣ  
ΜΕΤΑΒΟΛΙΣΜΟΣ





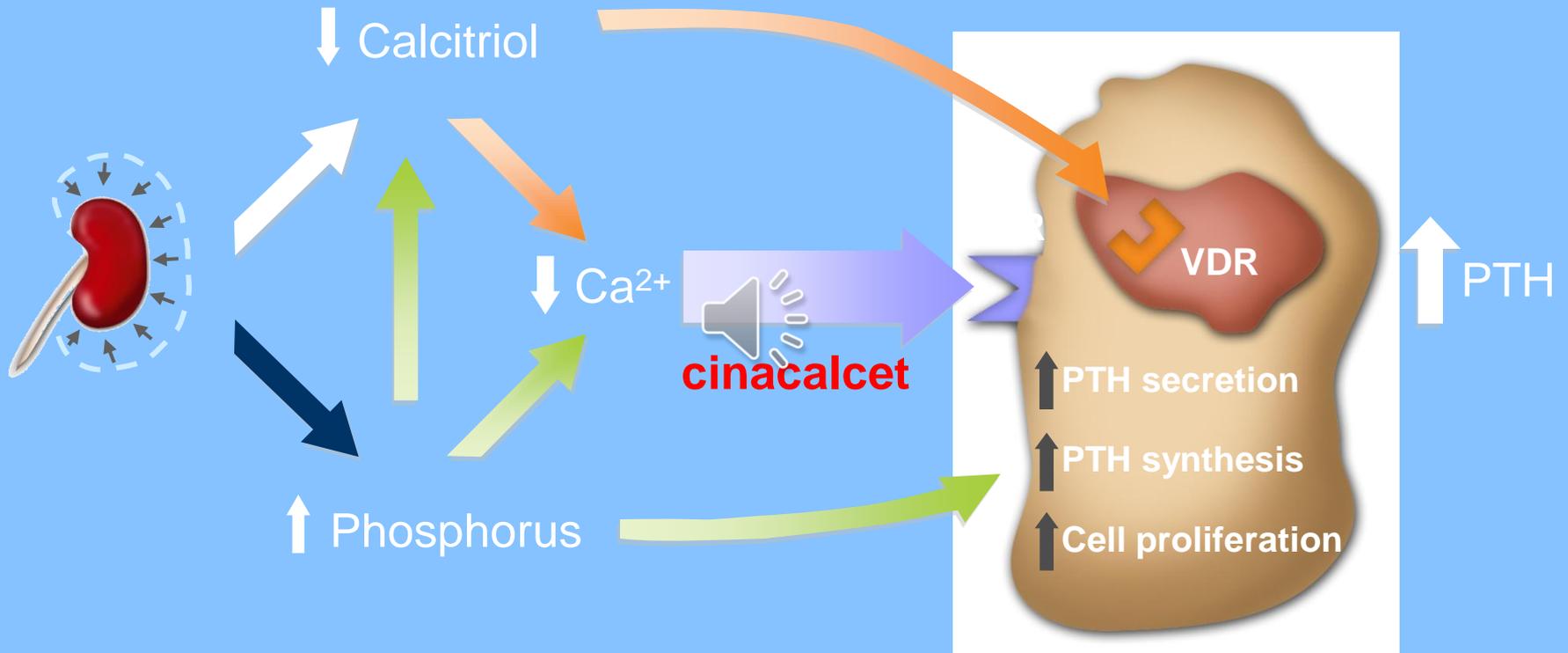
# Οι Βαθμολογίες Ασβέστωσης Στεφανιαίων Αρτηριών Αποτελούν Ανεξάρτητους Προγνωστικούς Παράγοντες της Θνησιμότητας σε Ασθενείς που Υποβάλλονται σε Αιμοκάθαρση



## Αριθμός ασθενών σε κίνδυνο

CAC 0	18	16	15	10	9	5	3
CAC 1-100	48	42	35	26	18	6	1
CAC 101-400	28	21	17	10	8	2	0
CAC 400+	72	59	44	16	9	2	0

- Ergocalciferol
- calcitriol or a synthetic analog



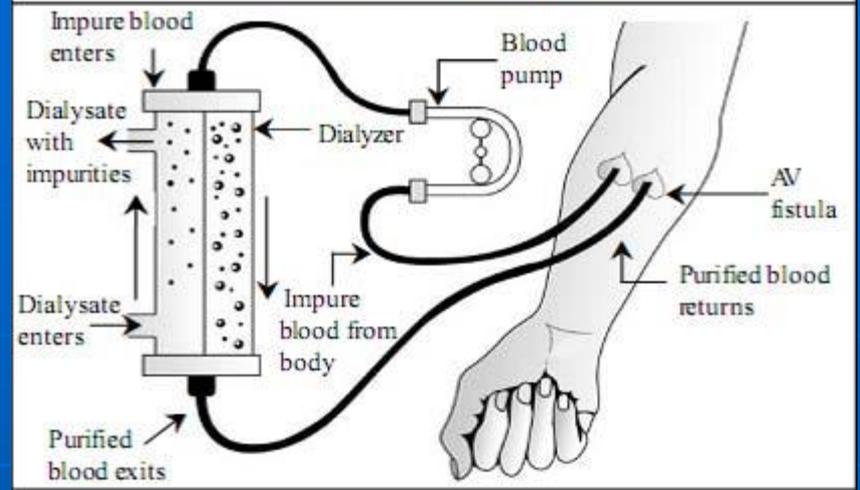
- limit phosphate intake to 900 mg/day
- calcium- and non-calcium-based phosphate binders

# ΕΞΩΝΕΦΡΙΚΗ ΚΑΘΑΡΣΗ



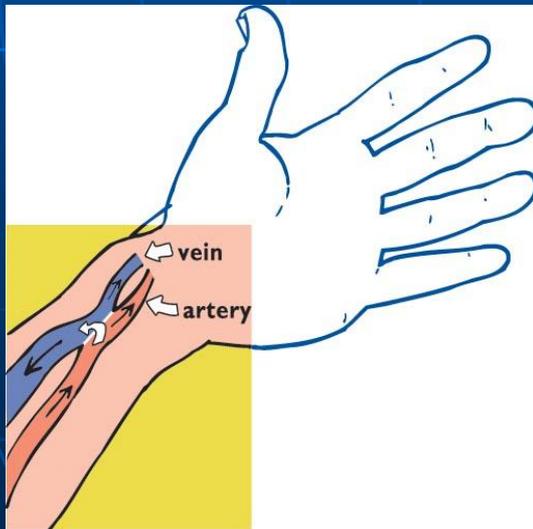
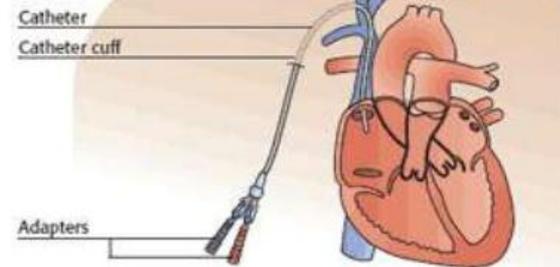


## Process of Hemodialysis

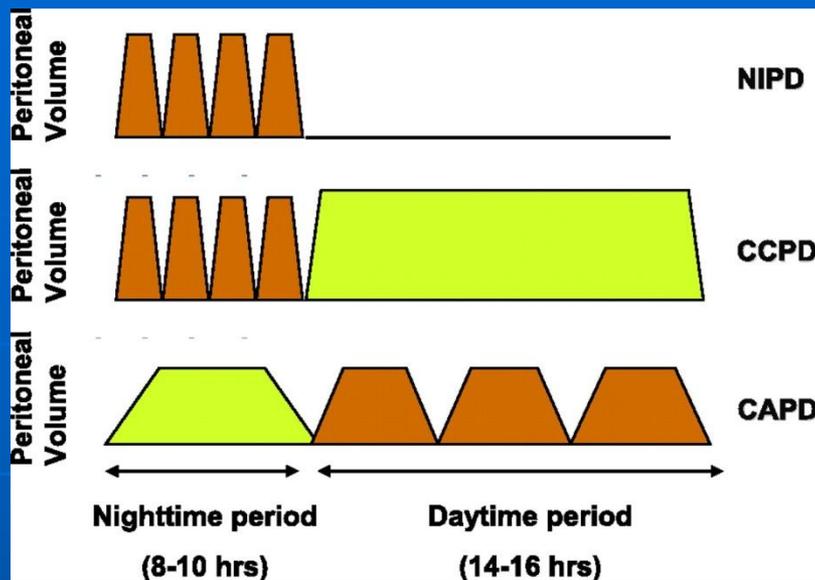


## Cuffed Dialysis Catheter

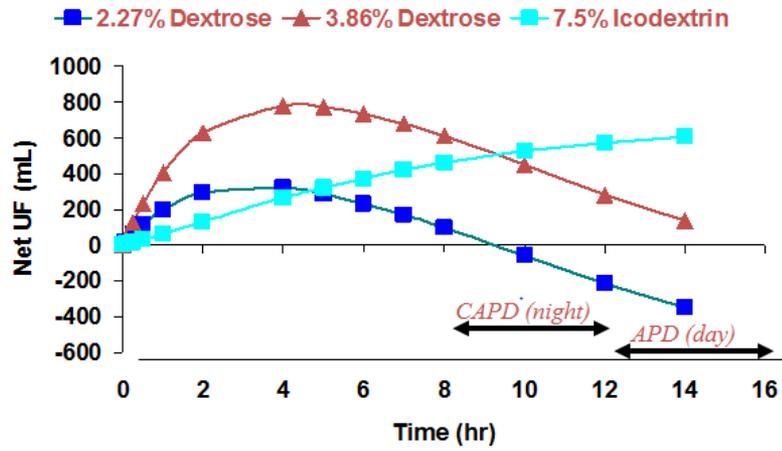
### Double-lumen, cuffed hemodialysis catheter



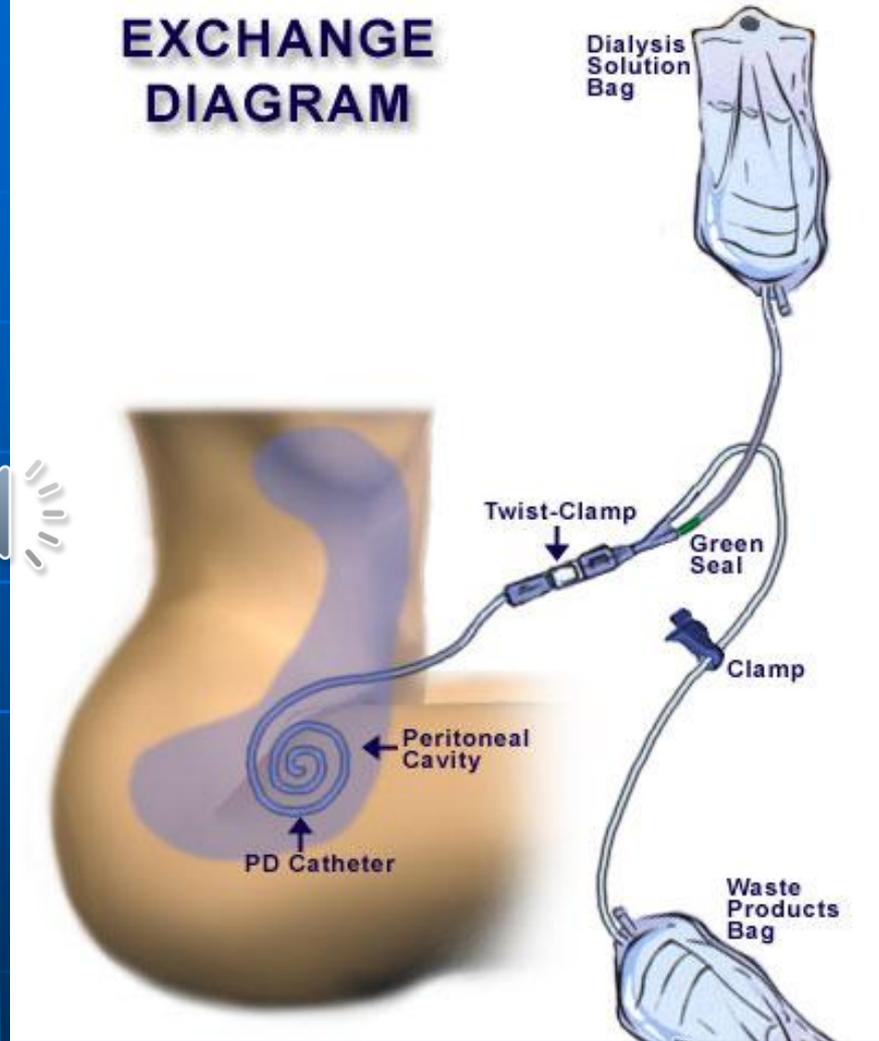




### Fluid Management in PD: Icodextrin Comparison with 2.27% and 3.86%



### EXCHANGE DIAGRAM



# ΕΙΔΗ ΠΕΡΙΤΟΝΑΪΚΗΣ ΚΑΘΑΡΣΗΣ ΑΥΤΟΜΑΤΟΠΟΙΗΜΕΝΗ

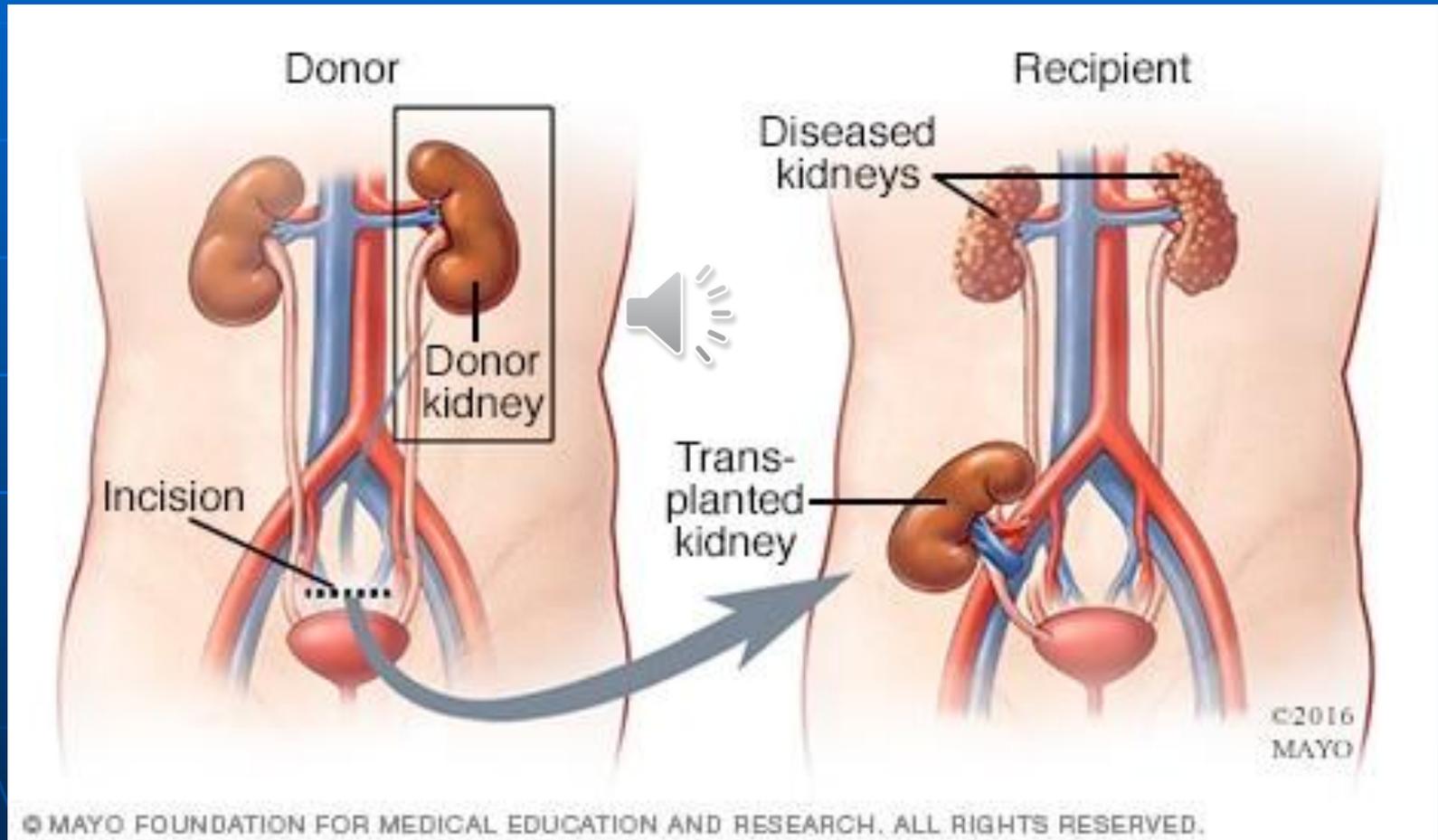


# ΜΕΤΑΜΟΣΧΕΥΣΗ ΝΕΦΡΟΥ



# Πρώτη Μεταμόσχευση Νεφρού: 1954

Dr. Murray, Brigham and Women's Hospital, Boston,  
MA, USA



# Πλεονεκτήματα νεφρικής μεταμόσχευσης

- Αποφυγή εξωσωματικής κάθαρσης
- Αρση διαιτητικών  περιρισμών
- Βελτιωμένη ποιότητας ζωής
- Βελτιωμένη επιβίωση
- Χαμηλότερο κόστος

**ΕΥΧΑΡΙΣΤΩ  
ΓΙΑ ΤΗΝ  
ΠΡΟΣΟΧΗ ΣΑΣ**

