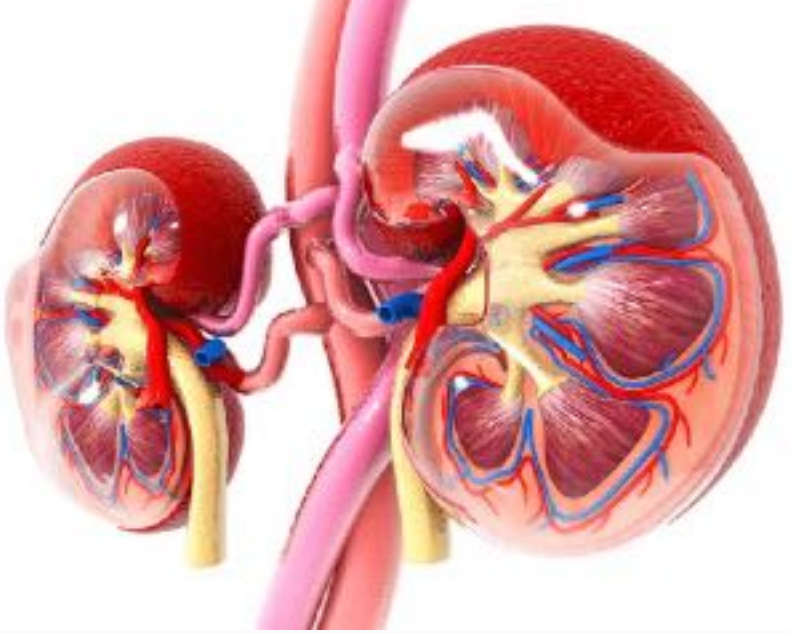

Renal Protection in the NICU

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Definition of AKI

- A **sudden and often reversible** reduction in kidney function, that results in reduced GFR, altered fluid and acid base balance, and disturbed electrolytes homeostasis.
- In neonates, AKI is defined by increase in serum creatinine by 0.3mg/dL from baseline level OR decrease in urine output.
- Both are challenging indicators in neonates, as baseline sCr is difficult to determine, and babies rarely become oliguric

Causes of AKI

- Pre-renal causes (85%): reduced blood flow to the kidney due to systemic hypoperfusion from hypovolemia or hypotension . Can also occur due to capillary leak (sepsis, or decreased oncotic pressure from hypoalbuminemia).
- Renal causes (11%) include acute tubular necrosis which can result from prolonged renal ischemia, sepsis, and nephrotoxic drugs .
- Post-renal causes (3%) mainly include obstructive causes

DRUGS: both intrinsic renal cell damage and prerenal ARF due to vasoconstriction

Neonatal AKI (KDIGO) Classification *(Jetten et al 2016)*

Stage	Serum Creatinine	Urine Output /24 hours
0	No change in serum creatinine Or rise <0.3 mg/dl	>1 mL/kg/h
1	SCr rise ≥0.3 mg/dL from baseline *or* sCr rise between ≥1.5 - 1.9 mg/dL times baseline* sCr within 7 d	Between >0.5 and ≤1 mL/kg/h
2	sCr rise between ≥2 and 2.9 times baseline sCr	Between >0.3 and ≤0.5 mL/kg/h
3	sCr rise ≥3 times baseline sCr *or* sCr ≥2.5 mg/dL[b] *or* receipt of dialysis	≤0.3 mL/kg/h

**Baseline Creat : lowest SCr prior measured*

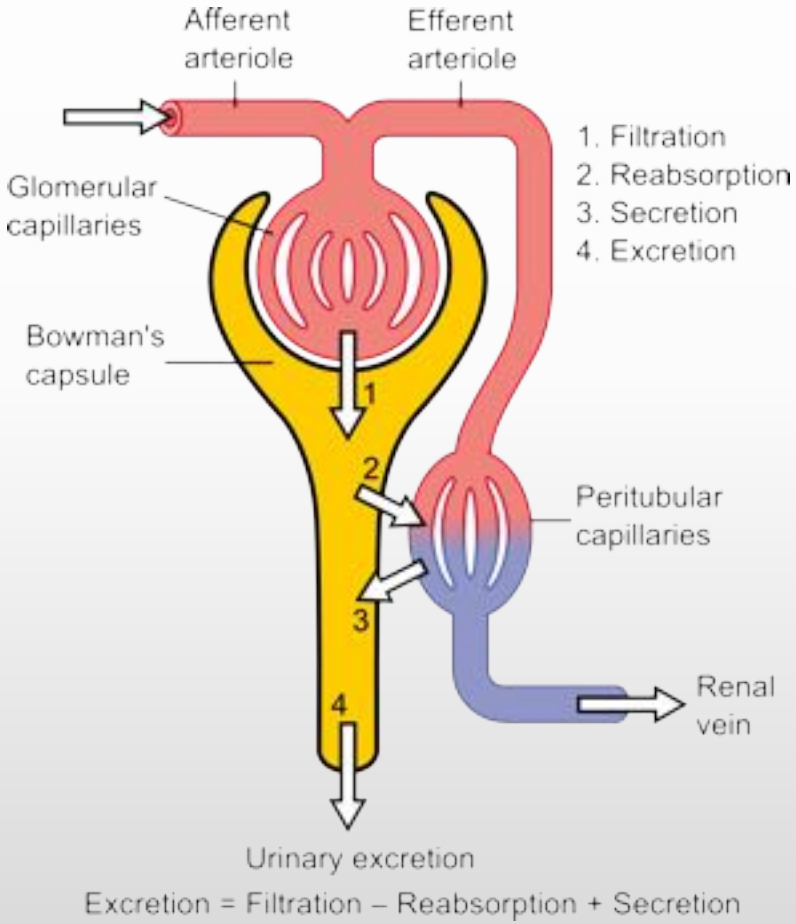
Incidence of AKI

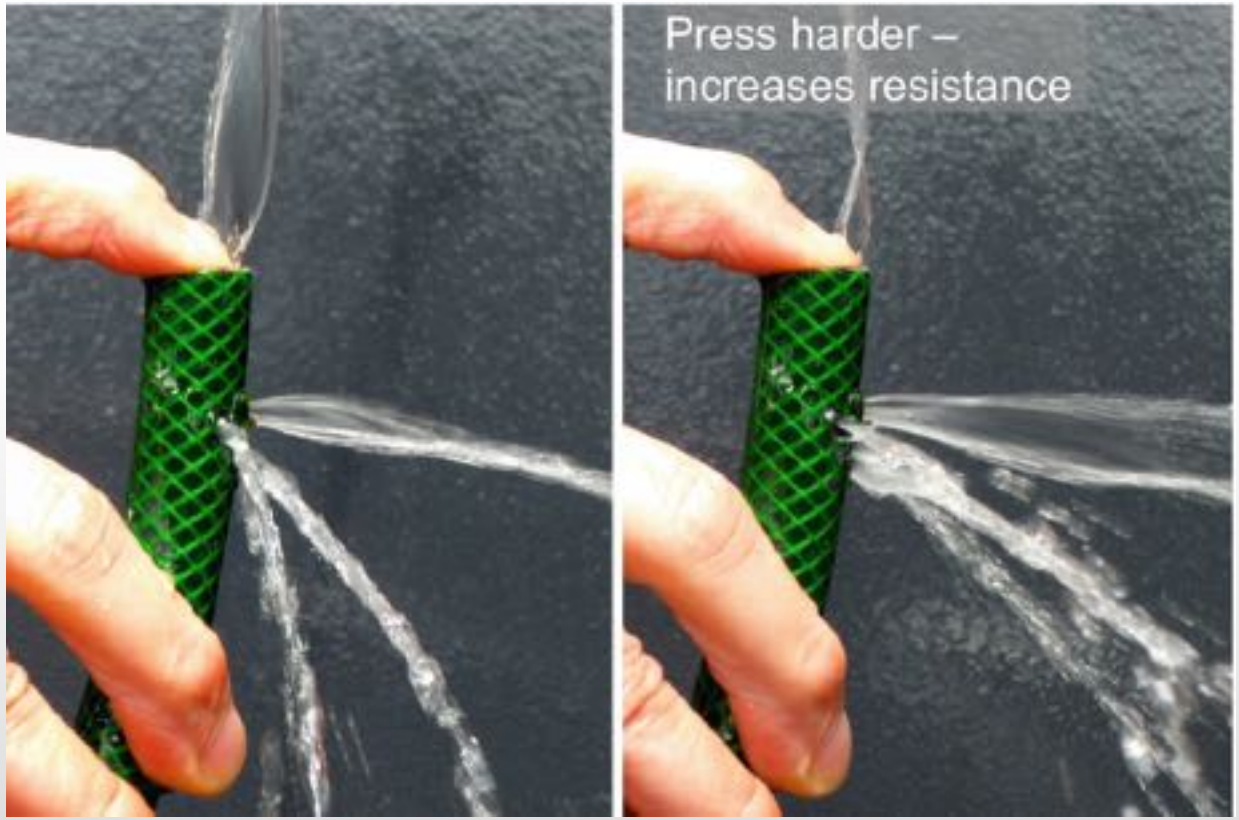
- The incidence of early AKI in the NICU is around 20-25%.
- Early AKI is associated with higher risk of death and longer duration of hospitalization (Jeniffer et al, 2019)
- The first week after birth is a vulnerable time for the development of neonatal AKI due to antenatal, intrapartum, and early postnatal transition factors.

Nephrogenesis

- Nephrogenesis commences around *week 5* of gestation and is complete by 35 wk.
- Post-natal nephrogenesis continues after preterm birth but abnormal glomeruli may result (*Gubhaju et al, 2009*)
- Babies are born with their mother's serum creatinine (sCr), which they clear over the first week or two of life.
- sCr should be approximately 0.2-0.3 mg/dl by D14.

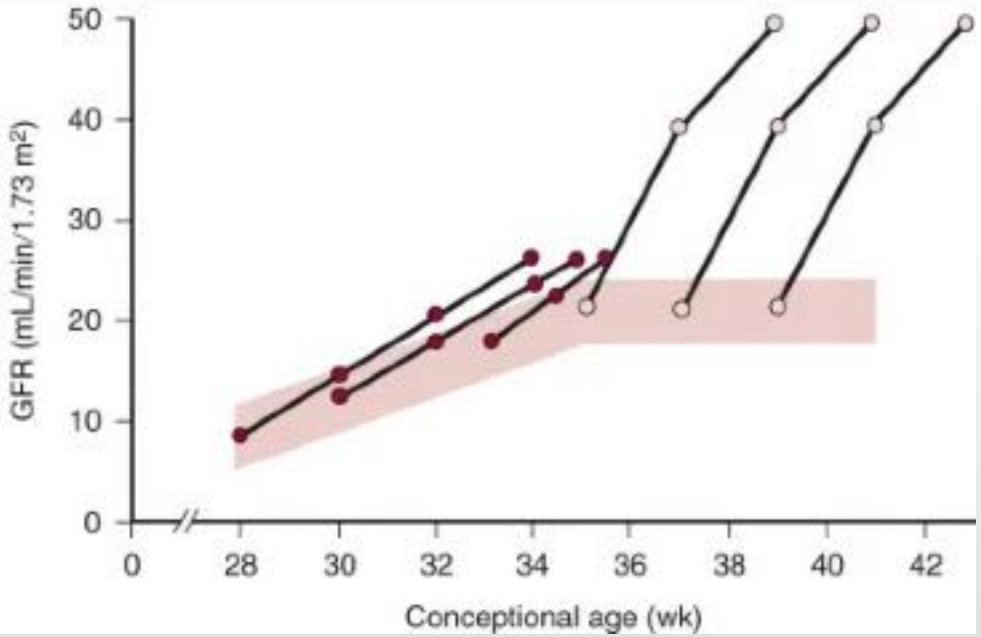
Renal physiology and the concept of GF



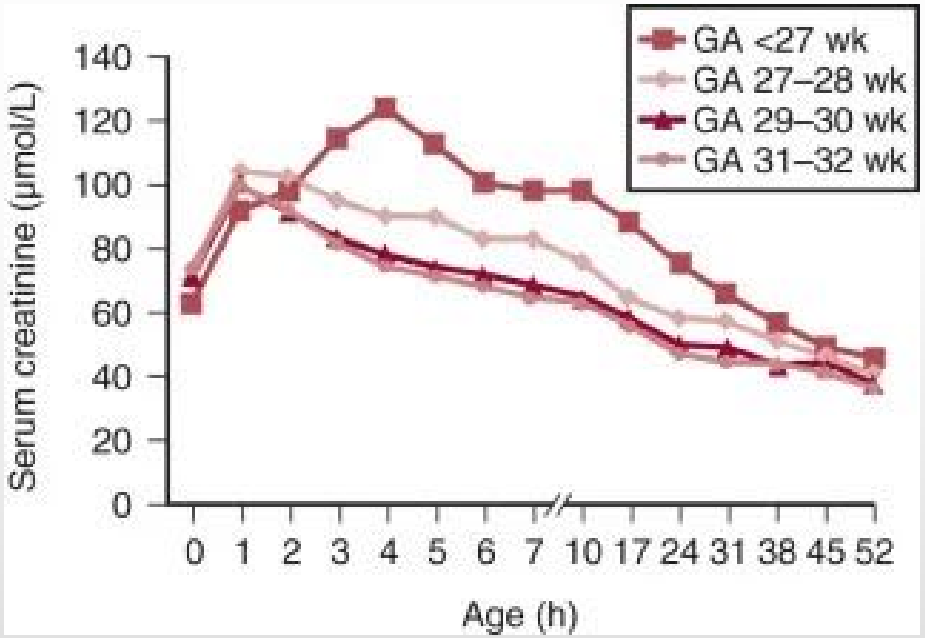


Changes in GFR and SCr levels

When water is reabsorbed along the nephron, the concentration of filtered creatinine rises so that creatinine back-diffuses into the blood according to its concentration gradient, thus raising its plasma concentration.



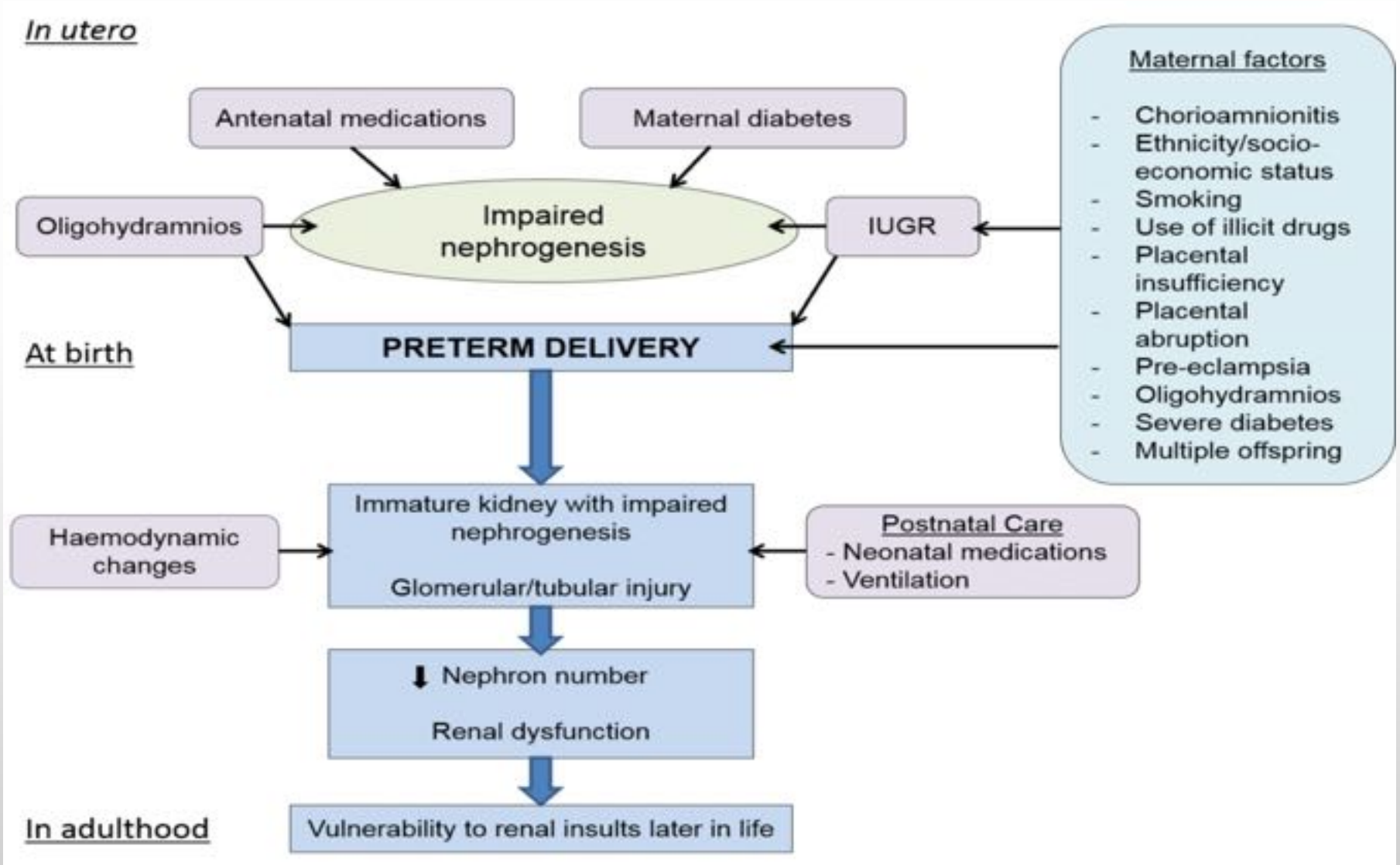
Guignard and John,1986



Gallini et al , 2000

In preterm infants the elevated P_{creat} further increases transiently to reach a peak value between the second and fourth days of postnatal life as a result of creatinine reabsorption across leaky tubules > tiniest preterm

The risks for the preterm kidney



Additional risk of AKI *(Charlotte et al, 2019)*

- Transfer from an external hospital (sicker infants, delay)
- Admission for surgical indication
- Admission with hyperbilirubinemia (hemolysis and tubular function, exchange , anemia, dehydration, phototherapy)
- Need for prolonged resuscitation and perinatal asphyxia

Renal protection

- Prevention of injury
- Close monitoring
- Timely Intervention

Avoid Injury

Avoid Injury

- Start from intrauterine life :
  - Proper antenatal care (avoid prematurity , Diabetes, Drugs)
- Proper neonatal resuscitation
- Fluid balance
- Optimization of blood pressure
- Avoid nephrotoxic drugs

Avoid certain drugs

Avoid Aminoglycosides *(Kent et al, 2014)*

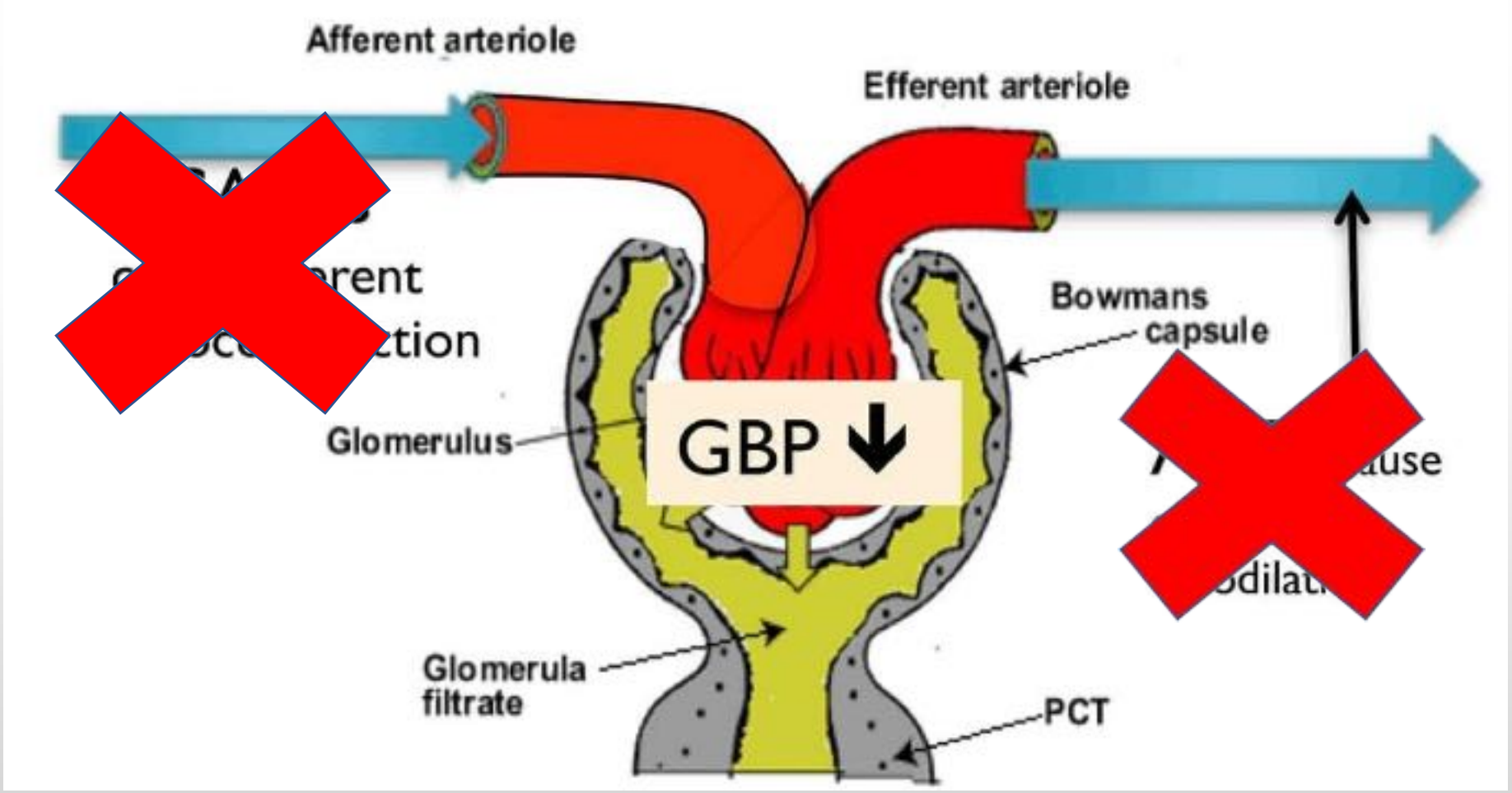
- Excreted in urine
- 5% to 10% of the drug accumulates in the renal cortex.
- High concentrations cause lysosomal dysfunction of the proximal tubules.
- Nephrotoxicity is related to the dose and duration of the antibiotic therapy, as well as the level of kidney function before the initiation of aminoglycoside therapy (a SINGLE dose is much safer) !
- Presents as nonoliguric AKI
- Reversible upon discontinuation of aminoglycosides

Avoid ACE inhibitors *(Gantenbein et. Al, 2008)*

- The renin-angiotensin-system (RAS) : essential for renal development. Its interruption: oligohydramnios, renal failure.
- Exposure in 2nd and 3rd trimesters causes > renal abnormalities than first.
- Avoid use in neonates < 34 GA when nephron formation is still incomplete.
- ACE inhibitors can result in AKI: if essential, *monitor intravascular volume and avoid simultaneous nephrotoxic medications*
- AKI due to ACE inhibitors: reversible upon their discontinuation

Avoid NSAID *(Hanna et al, 2016)*

- NSAIDs may precipitate AKI by affecting intrarenal hemodynamics.
- Neonates have high levels of circulating prostaglandins which are involved in maintaining arteriolar vasodilation, renal blood flow, and renal water clearance.
- Inhibition of prostaglandins: decrease in RBF and reduction in GFR.
- Indomethacin therapy for PDA closure in PT is associated with kidney dysfunction (56% reduction in urinary flow rate, a 27% in GFR, and a 66% in free water clearance along with a reduction in urinary sodium excretion and FENa.)


Afferent arteriole
Efferent arteriole
Bowmans capsule
Glomerulus
GBP ↓
Glomerula filtrate
PCT

Avoid Vancomycin (*Constance et al, 2016*)

- Vancomycin has been implicated in nephrotoxicity in the neonates (oxidative injury and stress at the PCT)
- Recent studies suggest that monotherapy with appropriate trough levels may not cause AKI.
- Increased risk of AKI with vancomycin:
  - if high trough levels
  - additional nephrotoxin use (such as NSAIDs)
  - Positive blood cultures, PDA, low birth weight, and severity of illness.

Avoid Amphotericin B *(Goldman et al, 2004)*

- Amphotericin B **binds to sterols in cell membranes, thereby creating pores that compromise membrane integrity and increase membrane permeability**; this is what accounts for its nephrotoxicity.
- Wasting of K and Mg also occurs due to the pores
- Lipid-based preparations of amphotericin B decrease but do not eliminate the nephrotoxicity

AKI secondary to nephrotoxins is avoidable!

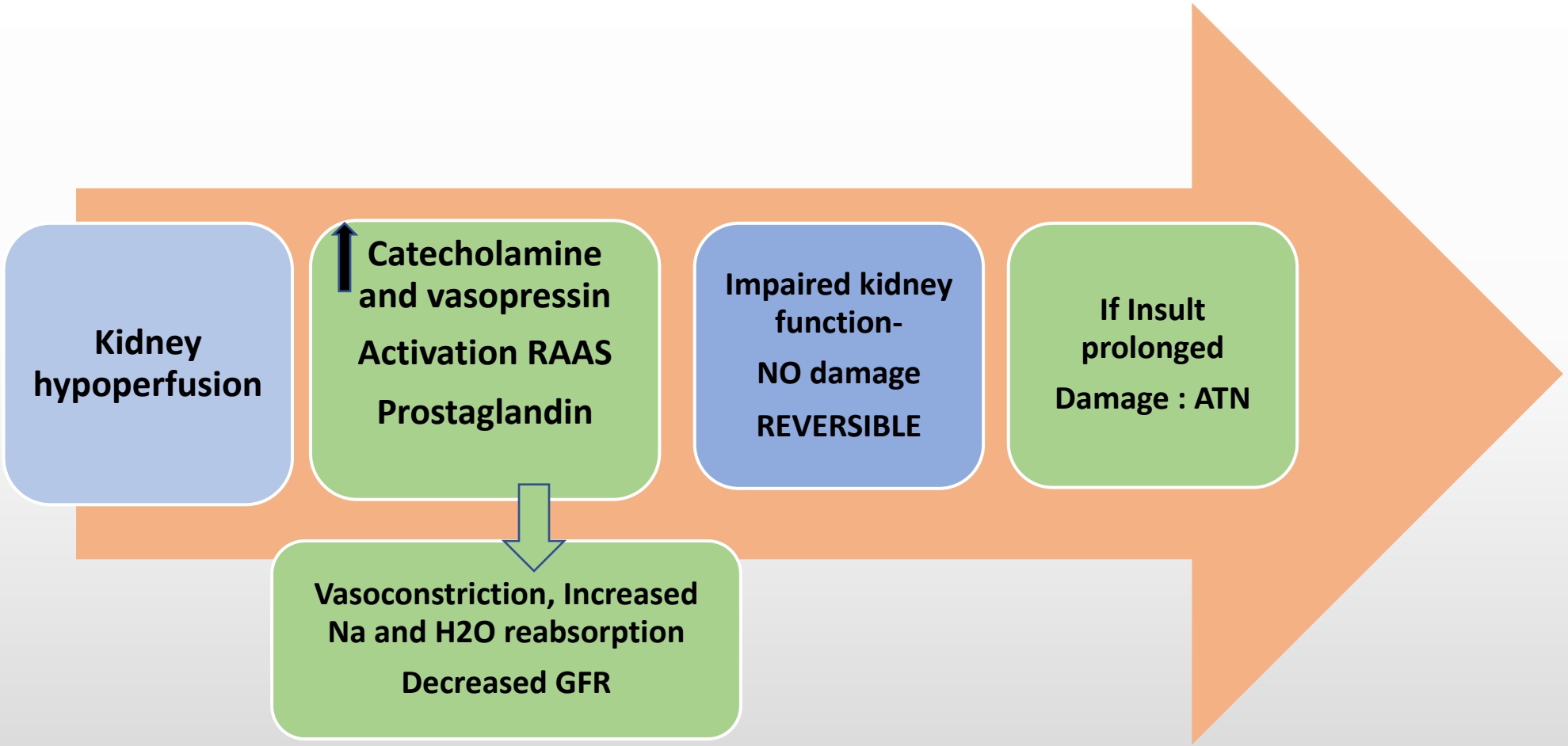
- While a single nephrotoxic medication increases the risk of AKI, the use of multiple nephrotoxic medications increases the risk exponentially more.
- Concomitant factors such as neonatal age, acuity of illness, and dosage and duration of medications all play a role in the degree of resulting AKI.

Avoid Prerenal Insult

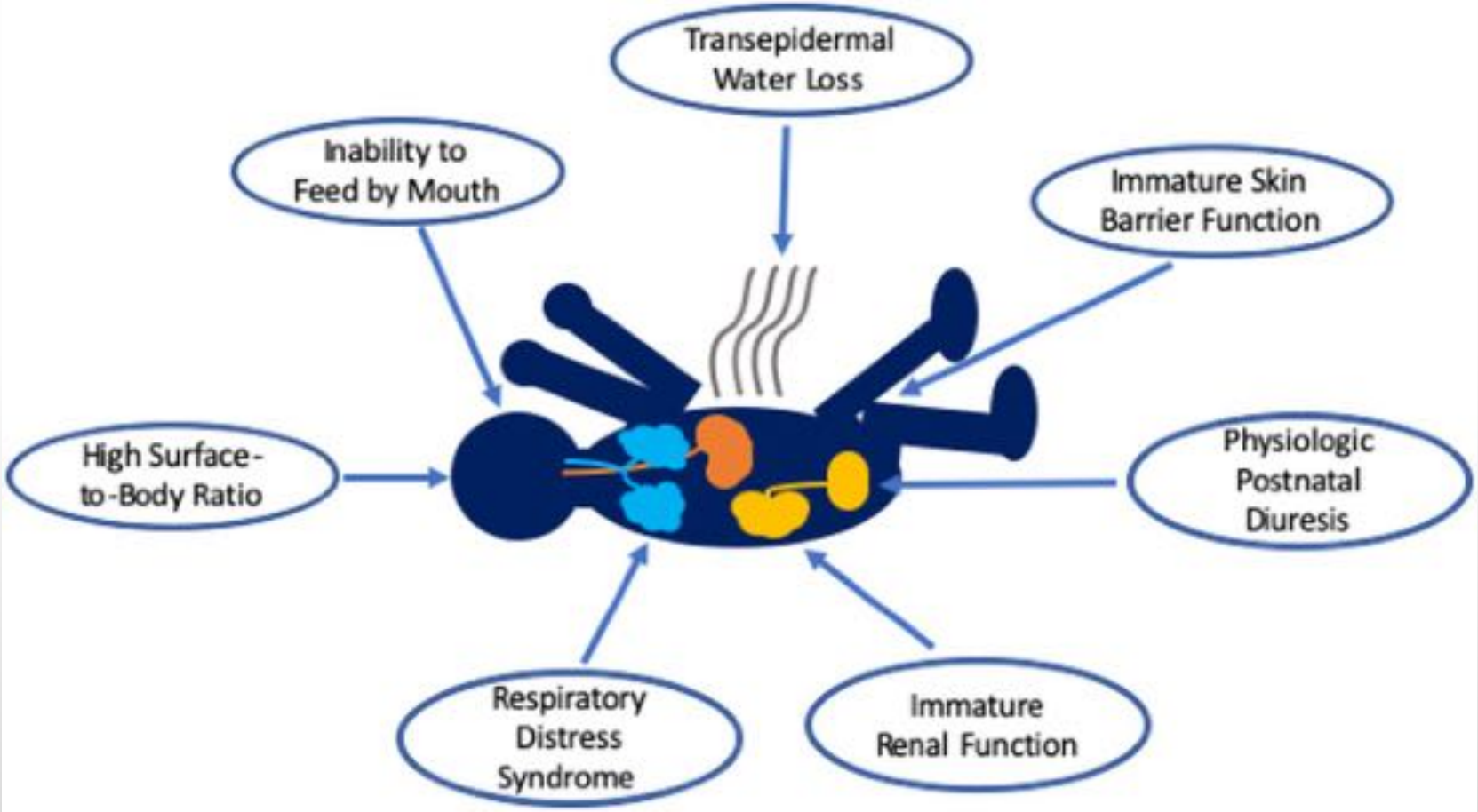
Prerenal AKI (Nada et al, 2017)

- Definition: A functional change (rise in SCr and drop in UOP) without actual kidney damage due to decreased effective renal blood flow.
- It is most frequent cause of AKI in neonates (85% of cases)
- Causes:
  - compromised placental blood flow (e.g. placental abruption)
  - excessive gastrointestinal losses
  - increased insensible losses, especially in premature neonates with skin immaturity.
  - Third spacing as seen in sepsis, or decreased oncotic pressure in hypoalbuminemia.
  - Hypotension and compromised cardiac output.
  - Non-steroidal anti-inflammatory drugs (NSAIDs) may cause renal vasoconstriction via blockage of cyclooxygenases and prostaglandin synthase.

In Prerenal AKI



Fluid balance very difficult in the PT



Assessment of fluid and electrolyte status

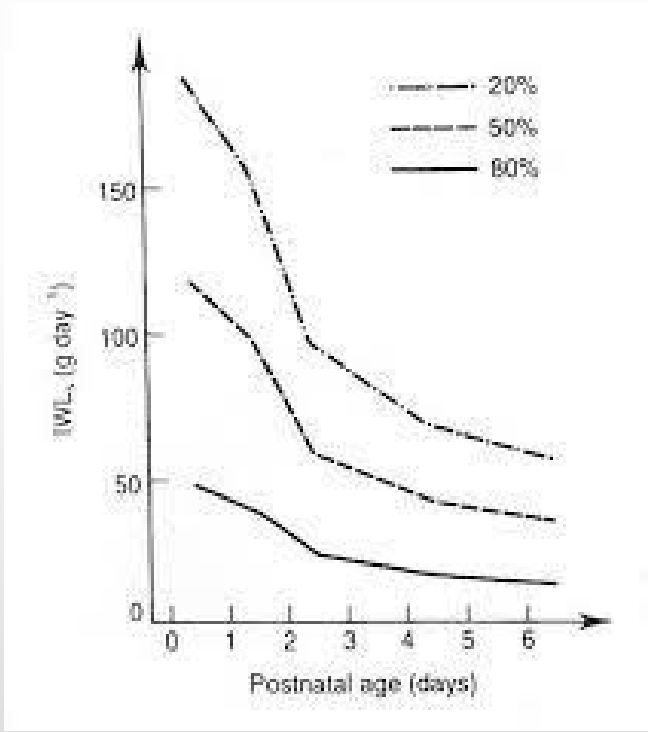
- History
  - Maternal : fluid during delivery/ oxytocin, Indomethacin, ACE inhibitors , furosemide , aminoglycosides
  - Asphyxia , oligohydramnios
- Clinical signs
  - *Insensitive indicators* : skin turgor, dry mucus membranes, fontanel .
  - *Late signs of dehydration* are seen (tachycardia, hypotension , poor perfusion, oliguria)
  - Oedema (*early sign*) in overload , or third spacing

Best assessment for fluid status

- Daily weight (physiological weight loss 10% by day 5)
- Vital signs: Blood pressure, HR
- Input and output (Normal urine output : once in the first 24 hours, gradual increase to 3-5 ml/kg/hour by day 5)
- Functional US
- Serum electrolytes

Proper fluid balance (*Branagan et al, 2022*)

- Aim: Euvolemia
- Insensible losses (substantial in ELBW): Increases with decreased GA and BW. Humidification reduces ISL.
- Fluid resuscitation: restore circulating volume and optimize renal perfusion in hypovolemia.
- Close monitoring of the response to fluid challenge (10–20 ml/kg over 1–2 h) to avoid fluid overload as is associated with poorer outcomes



Close Monitoring

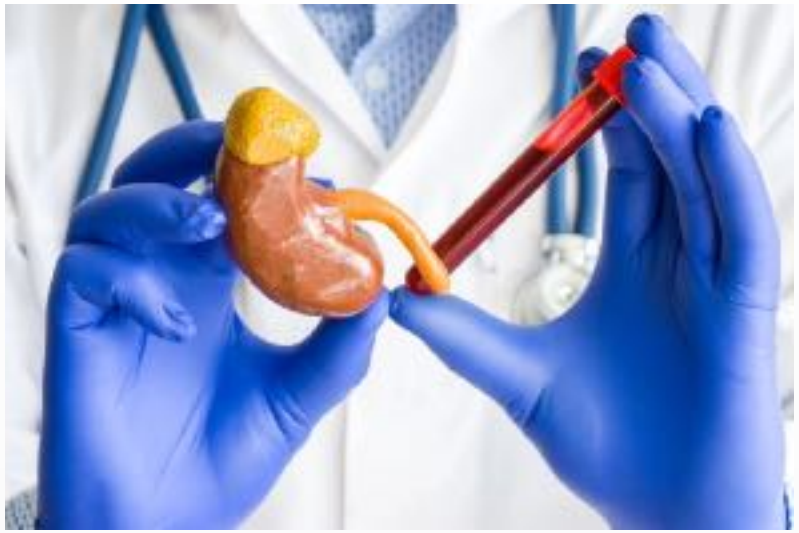
Urine output

- Urine output is a poor marker for kidney function in PT. They can continue to make urine even if they are volume depleted (Immature tubules (DCT, CT) unable to concentrate urine)
- The most common cause of anuria in the first 24 hours of life is false alarm (missing it in delivery room).
- **48 hours of life without urine output is the CONCERN.** Until that time: Low GFR

AKI in newborn maybe non oliguric

- Bigger amount of total body water compared to adults specially in PT
- Immature renal development (less absorption) more urine
- Injury of the tubular cells in asphyxia, natreurisis and consequently diuresis due to failure of sodium reabsorption

Markers of renal injury



- **Serum creatinine :** most commonly used
- **Cystatin C (glomerular)**: A protein filtered by the kidney released by all cells, not affected by GA or mother
- **Urinary NGAL** (neutrophil gelatinase-associated lipocalin): An iron transport protein accumulates in urine in kidney injury.
- **N-acetyl-D-glucosaminidase** is a lysosomal enzyme present in cells of the proximal **tubules**, and increased activity in the urine reflects proximal tubular injury after asphyxia

Is SCr a good marker of renal injury?

- Serum creatinine (sCr) : most commonly used KFT
- Imperfect marker: (*Ashkenazi et al, 2018*)
  - determined by muscle mass and clearance.
  - Insensitive: lag between injury and rise (rises when 25%-50% of the kidney function is lost)
  - At lower GFR , It overestimates renal function due to its tubular secretion
  - It is easily dialyzed so cannot be used to assess kidney function in a dialyzed patient

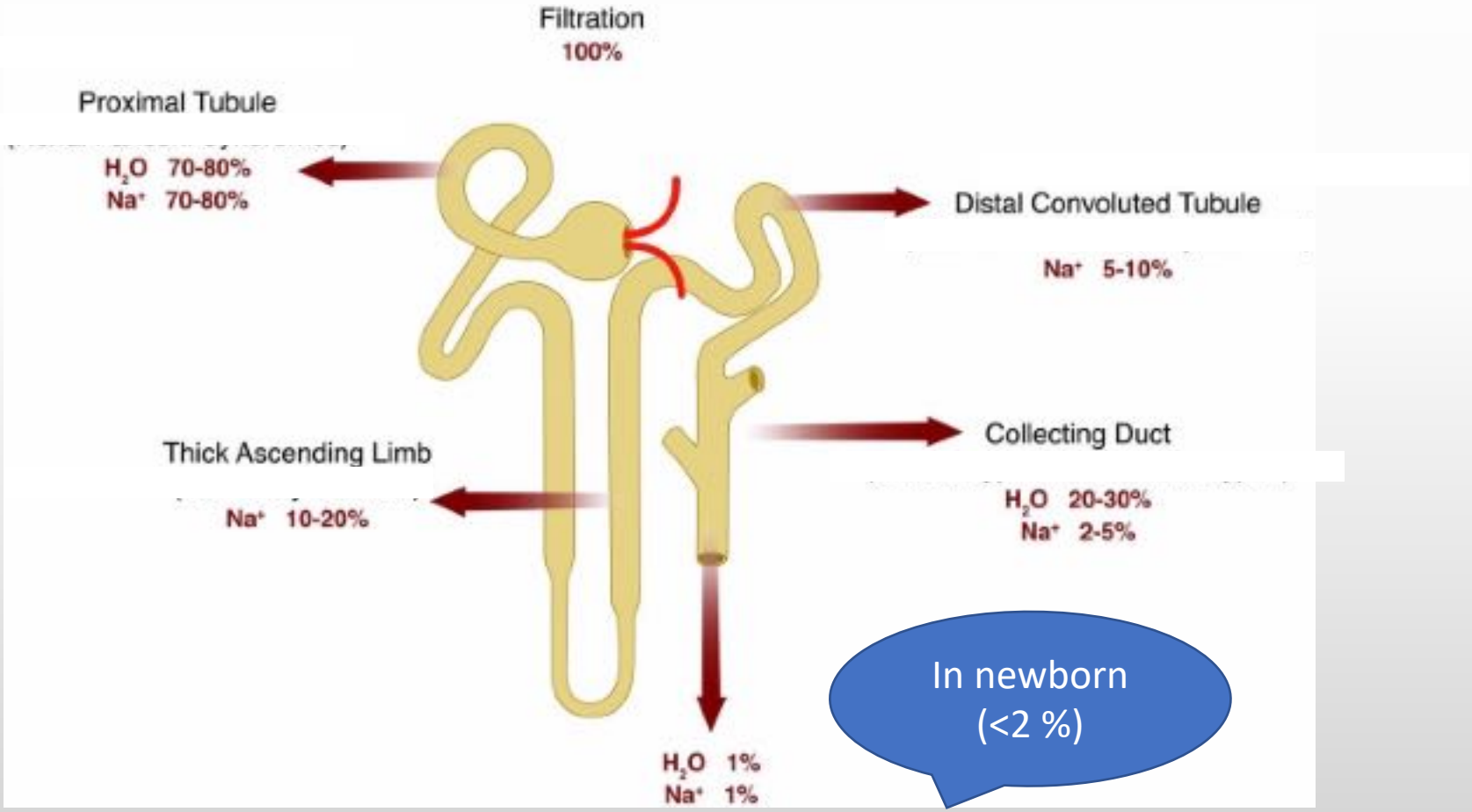
Monitoring (cont)

- Monitor blood pressure
- Measure electrolytes
- Functional US for assessment of IVC flow and diameter
- Renal and bladder US
- Calculate GFR
- NIRS

Difference between Prerenal Injury and ATN?

- During prerenal injury:
  - tubules are working appropriately
  - Respond to decreased renal perfusion
  - conserve sodium and water.
  - urine osmolality is greater than 400 to 500 mOsm/kg H_2O, the urine sodium is less than 10 to 20 mEq/L, and the FENa is less than 1% in children.
- In vasomotor nephropathy, the irreversible injury of the tubules prevents conservation of sodium
- The urine osmolality, urine sodium concentration and the fractional excretion of sodium (FENa) proposed for differentiation

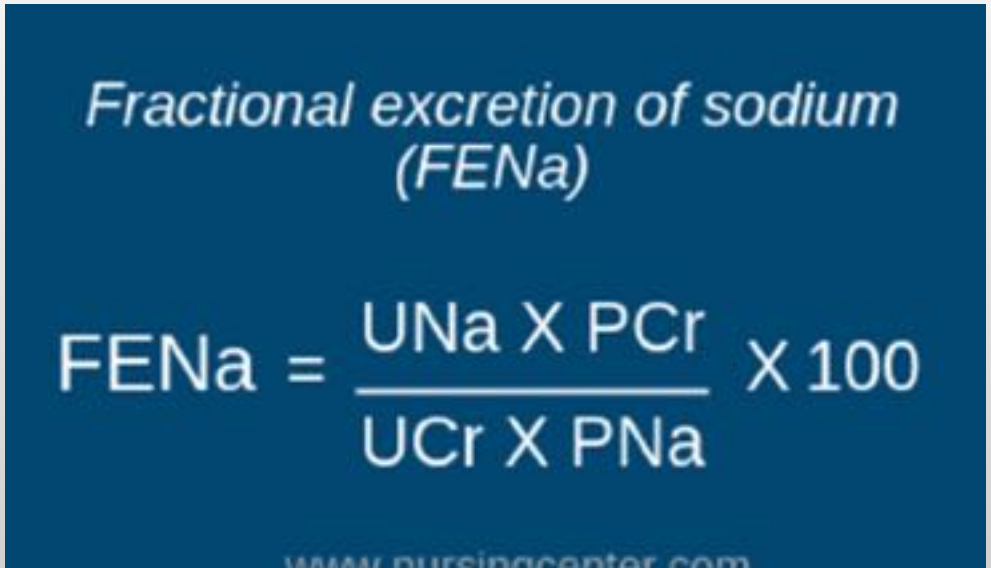
Na filtration , reabsorption



Kleta and Bockenhouer, 2018

Fractional excretion of sodium

- It is the ratio of Na clearance to the creatinine clearance expressed in percent.
- It is dependent on tubular function which depends on autoregulation of blood flow to the kidney
- it is a calculation based on the concentrations of sodium and creatinine in the blood and urine.

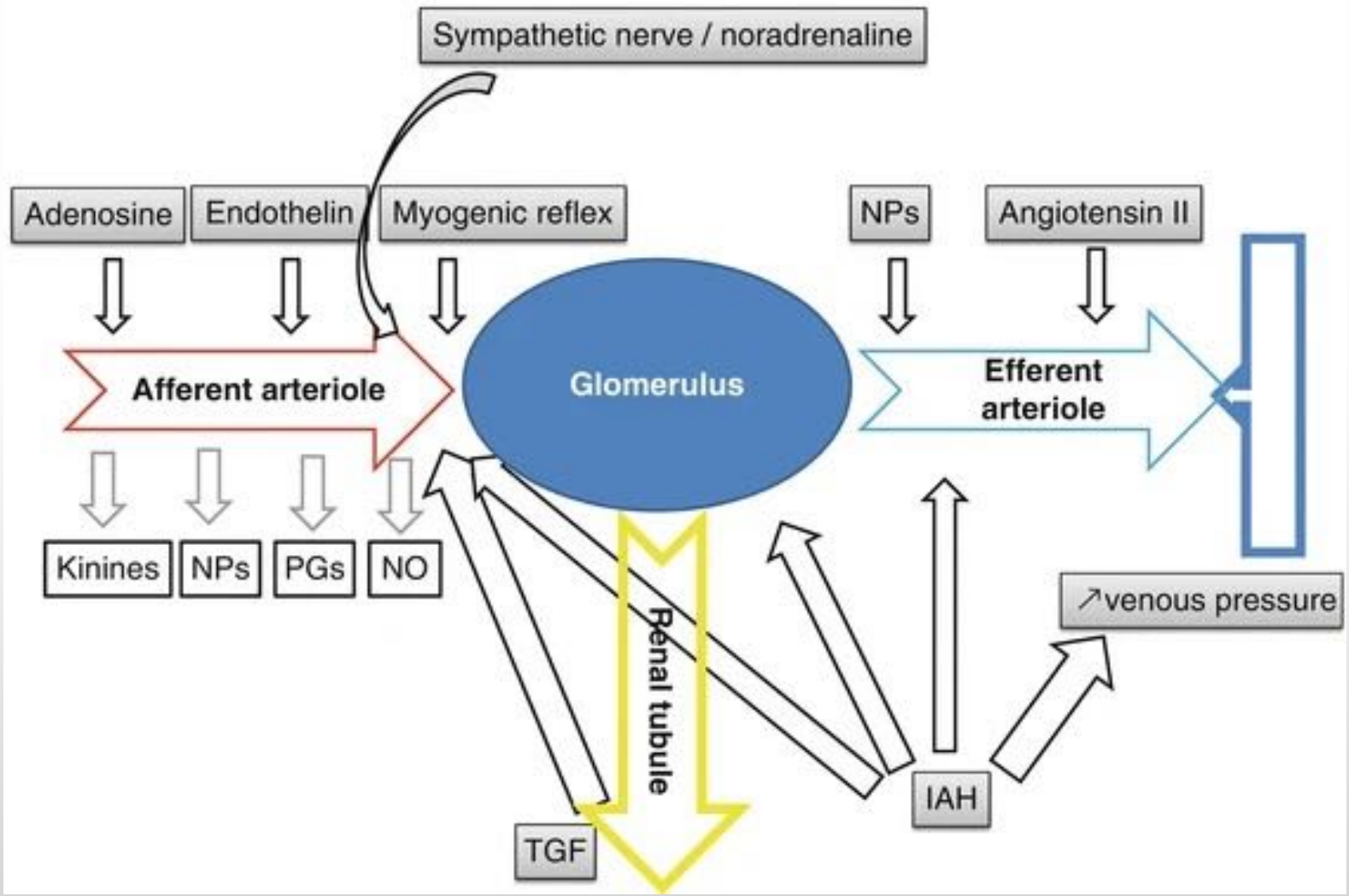


Timely Intervention

Xanthines

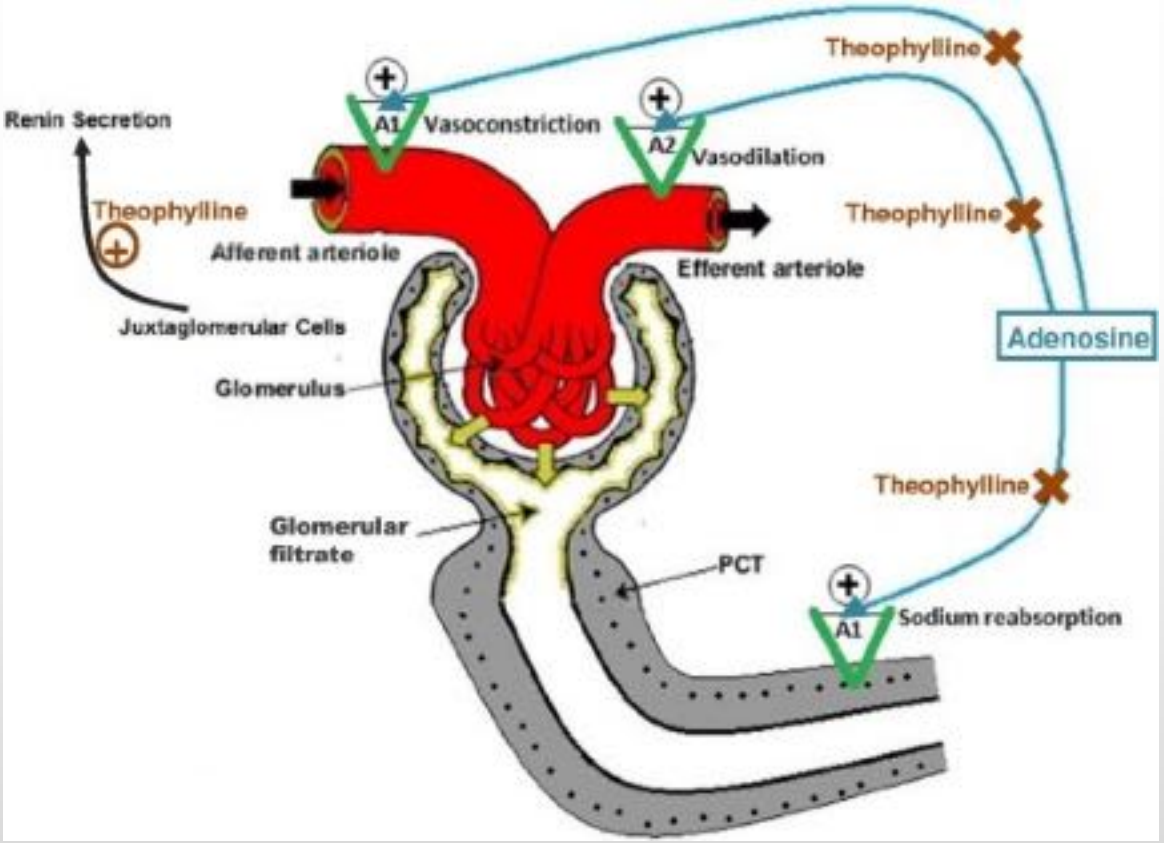
- Methyl Xanthines are adenosine antagonists that act via A_1 and A_{2A} receptors in the brain, heart, respiratory system, GIT and kidneys
- Adnosine is an autacoid, generated in the cytosol and extracellular sites of kidney
- Its formation is enhanced during oxygen deficiency.
- Adenosine lowers glomerular filtration rate by constricting afferent arterioles, especially in superficial nephrons, and thus lowers the salt load and transport work of the kidney to reduce renal metabolism
- Adult studies :Theophylline protects against contrast induced nephropathy and induces diuresis (Bing Dai et al, 2012)

Autoregulation of renal blood flow



Hoste and De Laet, 2012

Theophylline in protection from renal injury



Mcullough et al, 2012

Treating asphyxiated newborns with theophylline at birth protects the kidney (Raina et al, 2016)

- 159 severely asphyxiated term newborns randomized to receive a single dose of 5 mg/kg intravenous theophylline (n = 78) or a placebo (n = 81) during the first hour of life.
- Results: Intravenous (IV) theophylline in severely asphyxiated neonates given within the first hour after birth was associated with improved fluid balance, improved creatinine clearance, and reduced serum creatinine levels with no neurologic or respiratory complications
- PPHN was slightly higher among theophylline treated group

AWAKEN: Is early caffeine administration associated with reduced acute kidney injury (AKI) in preterm neonates?

Retrospective
Observational cohort

AWAKEN cohort
Multicenter and international

Neonates
Gestational age <33 weeks

Neonatal acute kidney injury
KDIGO Definition
< 7 days after birth

Before AKI

N = 675

Caffeine citrate exposure n = 447

No exposure n=228

Neonatal acute kidney injury

Total; **18.1%**
122 of 675

	Caffeine Citrate	AKI	No Exposure
	11.2% (50 of 447)	Adjusted OR 0.20 (0.11-0.37)	31.6% (72 of 228)
	1.3% (6 of 447)	AKI 3 Adjusted OR 0.20 (0.12-0.34)	10.5% (24 of 228)

Decreased odds of early AKI in neonates

Extremely preterm by **87%** OR 0.13

Very preterm by **73%** OR 0.27

Conclusion: Caffeine administration in preterm neonates is associated with reduced incidence and severity of AKI. Further studies should focus on the timing and dosage of caffeine to optimize the prevention of AKI.

Reference: Harer et al. *Association Between Early Caffeine Citrate Administration and Risk of Acute Kidney Injury in Preterm Neonates Results From the AWAKEN Study.* 2018. 10.1001/jamapediatrics.2018.0322

Visual abstract by Verner Venegas MD MHA

@Vernisartan

Caffeine citrate in the PT for prevention of AKI

- Potential mechanisms of action through which caffeine and possibly other methylxanthines could directly reduce AKI:
- 1) Increased renal blood flow, enhanced sodium excretion, and a higher glomerular filtration rate.
- 2) Caffeine citrate counteracts the hypoxemia-induced renal hemodynamic changes by maintaining renal vascular resistance.
- 3) Another potential mechanism of caffeine-mediated renal protection may involve attenuation of oxidative stress and injury on endoplasmic reticulum

Caffeine citrate in the PT for prevention of AKI

- Because of the benefits and favorable adverse effect profile of caffeine, it may be reasonable to use prophylactic caffeine in neonates of 28 to 32 weeks' gestational age to prevent or reduce AKI, even when apnea of prematurity is absent.
- For extremely preterm neonates, evaluation of the optimal dose, duration, and timing of initiation of caffeine therapy to prevent and reduce the severity of AKI should be explored with a standard protocol for evaluating renal function and injury.

Theophylline and aminophylline for prevention of acute kidney injury in neonates and children: a systematic review

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ABSTRACT

Objective To compare the efficacy and safety of theophylline or aminophylline for prevention of acute kidney injury (AKI) in neonates and children.

Design Systematic review and meta-analysis with application of Grading of Recommendations, Assessment, Development and Evaluation system.

Data sources PubMed/MEDLINE, Embase, Google Scholar and Cochrane renal group were searched from 1970 to May 2018.

Eligibility criteria Randomised clinical trials and quasi-randomised trials comparing the efficacy and safety of prophylactic theophylline or aminophylline for prevention of AKI in neonates and children were included. The primary outcomes were: incidence of AKI,

What is already known on this topic?

- Acute kidney injury (AKI) is associated with increased mortality, longer duration of hospital stay and an increased cost.
- Care for neonates and children with AKI remains supportive.

What this study adds?

- A single dose of adenosine antagonists reduces the incidence of AKI in term neonates with severe birth asphyxia by 60% without

Outcome measures

- **Primary outcome measures**
- 1. Incidence of AKI/severe renal dysfunction in initial 7-day period.
- 2. Serum creatinine (mg/dL) levels daily up to 5 days.
- 3. All-cause mortality.

Secondary outcome measures

1. Fluid balance daily up to 5 days and Estimated GFR
2. Urinary β2 globulin levels (mg/L) during initial 5 days.
3. Disease complications and treatment emergent adverse events.

Study or Subgroup	Theophylline Events	Total	Control Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Bakr 2005	5	20	12	20	10.7%	0.42 [0.18, 0.96]
Bhatt 2006	10	40	18	30	18.3%	0.42 [0.23, 0.77]
Eslami 2009	2	17	8	19	6.7%	0.28 [0.07, 1.14]
Jenik 2000	4	24	15	27	12.5%	0.30 [0.12, 0.78]
Khurshid 2017	13	40	20	40	17.8%	0.65 [0.38, 1.12]
Raina 2016	12	78	39	81	34.0%	0.32 [0.18, 0.56]
Total (95% CI)		**219**		**217**	**100.0%**	**0.40 [0.30, 0.54]**
Total events	46		112			

Heterogeneity: $Chi^2 = 4.28$, $df = 5$ ($P = 0.51$); $I^2 = 0\%$

Test for overall effect: $Z = 6.21$ ($P < 0.00001$)

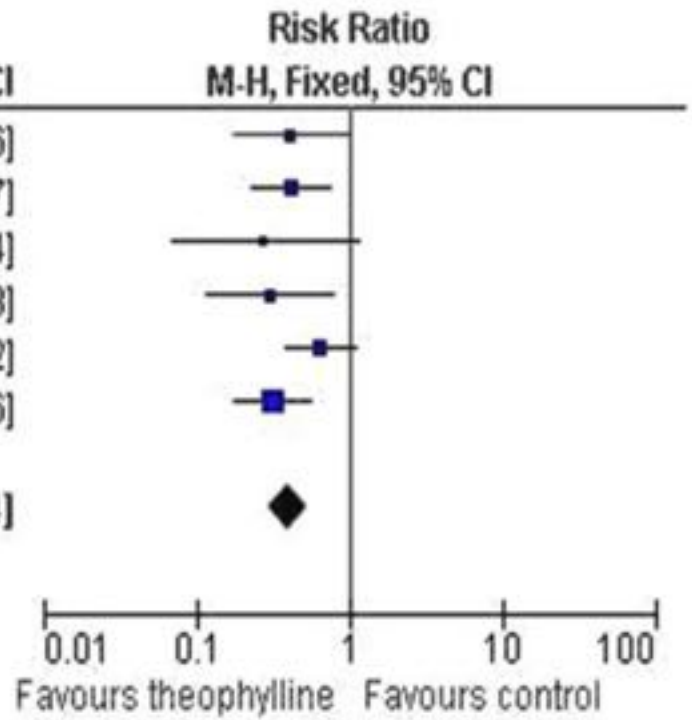


Figure 2 Forest plot showing incidence of AKI in neonates with severe birth asphyxia (theophylline vs placebo). AKI, acute kidney injury.

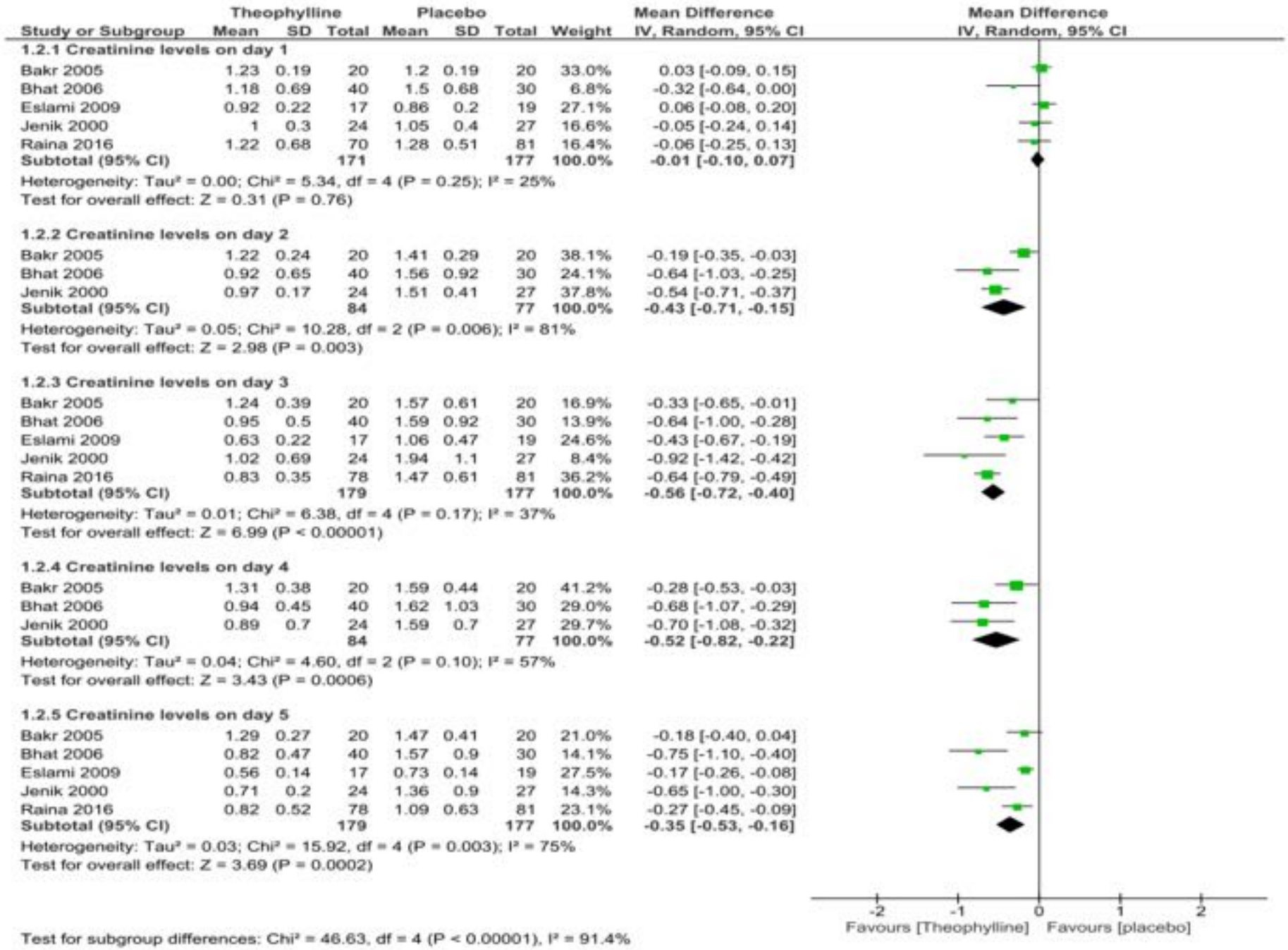


Figure 3 Forest plot showing serum creatinine in neonates with severe birth asphyxia (theophylline vs placebo).

Therapeutic hypothermia

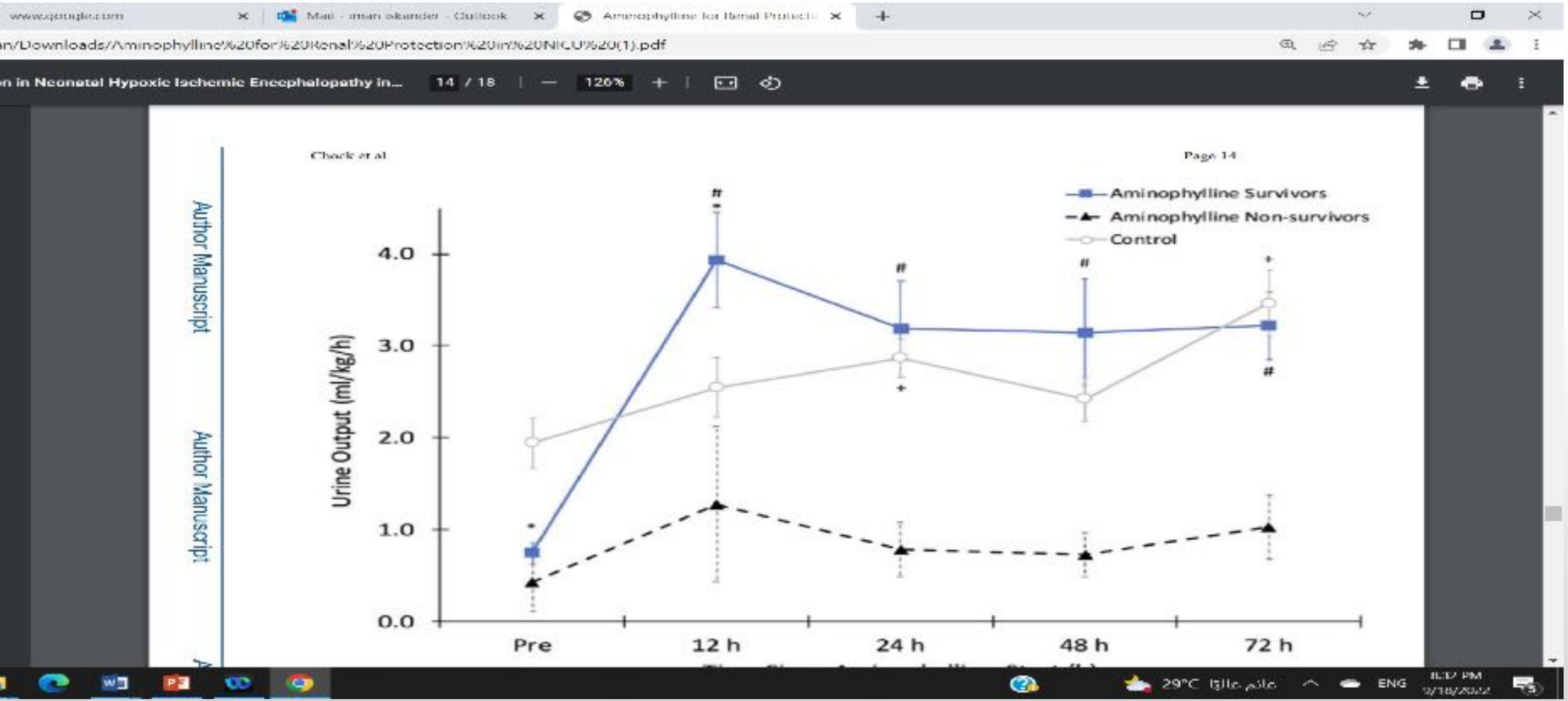
- Mechanism of protection of TH against AKI in neonatal HIE:prevention of ischemic reperfusion injury through:
  - decreased metabolic demand,
  - reduction of free radical production
  - limitation of apoptosis

Continuing kidney injury may persist in asphyxiated newborn despite improvement in serum creatinine and UOP as evident by persistent elevation of serum NGAL and cystatin C (Nour et al, 2019, Journal of Neonatal-Perinatal Medicine)

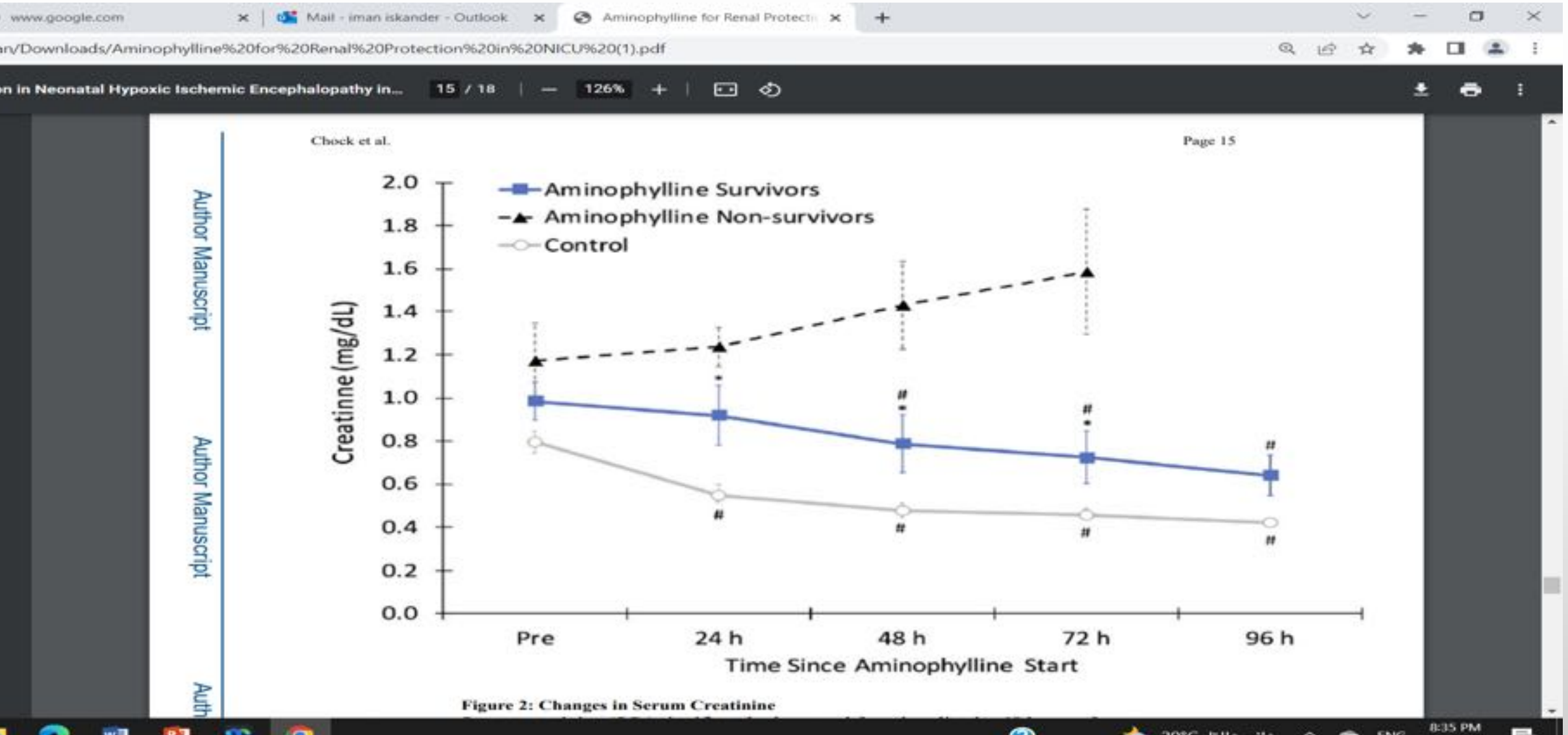
Aminophylline during TH (Chock et al,2021):

- During hypoxia/ischemia, intrarenal vasoconstriction occurs as a consequence of higher adenosine levels with a subsequent reduction in renal blood flow and fall in glomerular filtration rate (GFR) and filtration fraction which was reversed by theophylline in animal models (Gouyon & Guignard , 1998),
- Aminophylline was initiated for low UOP, rising SCr and/or other concern for AKI with an intravenous loading dose of 5 mg/kg followed by maintenance dose of 1.8 mg/kg every 6 hours. Theophylline levels were monitored as part of clinical care and adjusted to maintain a level of 5-7 mg/L.

Urine Output



Serum Creatinine

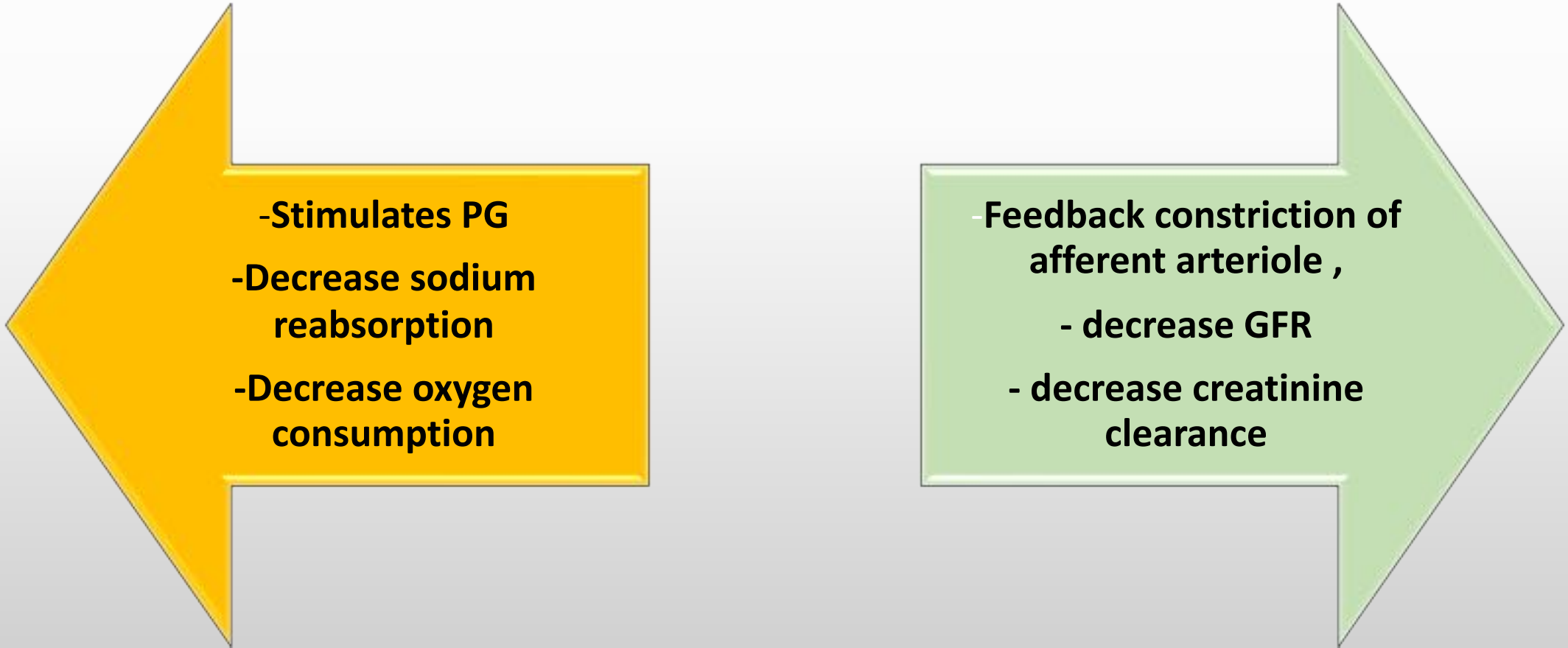


Conclusions

- Aminophylline exposure in neonates with HIE undergoing therapeutic hypothermia was associated with increased UOP and a decline in SCr in survivors.
- Control neonates not exposed to aminophylline exhibited a smaller increase in UOP over time and had a similar decline in SCr after starting from a lower baseline.
- No adverse effects were associated with aminophylline administration

Diuretics

Diuretic therapy to stimulate urine output eases management of AKI, but conversion of oliguric to nonoliguric AKI has not been shown to alter the course of AKI.



Diuretics (*Segar and Jetton,2021*)

- Use of diuretic is appropriate for fluid overload and for treatment of hyperkalemia.
- In the absence of such conditions, and with the understanding that diuretics themselves have little benefit to kidney function, they should be avoided
- Dose : Lasix (1–5 mg/kg per dose) increases intra-tubular urine flow
- May be less toxic with continuous infusion
- High doses of furosemide in kidney injury: ototoxicity and hypercalcuria

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Intensive Care Med (2001) 27: 206–210
DOI 10.1007/s001340000775

NEONATAL AND PEDIATRIC INTENSIVE CARE

Ingrid Prins
Frans B. Plötz
Cuno S. P.M Uiterwaal
Hans J. van Vught

Low-dose dopamine in neonatal and pediatric intensive care: a systematic review

NO EVIDENCE

Low dose dopamine

- Dopamine is a proximal tubular dilator that increases Na(+) delivery to tubular cells
- Increases effective renal blood flow and sodium excretion and urine volume, However, no studies have demonstrated that low-dose dopamine is effective in decreasing the need for dialysis
- Increases effective cardiac output which enhances renal blood flow,
- Experimental studies show renal protective effect (Seri et al, 1988) however, most recent studies did not show this benefit

Side effects of low dose Dopamine

- Gangrene if peripheral extravasation occurs
- Tachycardia and possible arrhythmia

The use of dopamine should be reconsidered till well controlled randomized trials are performed

Fenoldapam

- Fenoldopam is a potent short-acting selective dopamine-1 receptor agonist that decreases vascular resistance while increasing kidney blood flow.
- RCT in newborn infants on cardiopulmonary bypass to determine if the use of fenoldopam in neonates on conventional diuretics improved renal function--- No reduction in AKI or improvement in urine output was noted .
- Additional studies utilizing fenoldopam need to be performed in children and neonates with AKI.

Erythropoietin (Vesna et al, 2014)

- It has tissue-protective effects in clinical models and human studies across several organ systems.
- Erythropoietin receptors are present on glomerular, mesangial, and tubular epithelial kidney cells.
- Animal studies of ischemia–reperfusion injury and sepsis-induced AKI show that rhEpo preserves kidney function, protects renal proximal tubular cells by decreasing apoptosis, and decreases proinflammatory cytokine expression in the renal cortex.

Doxycycline (*Laboissiere et al , 2015*)

- Doxycycline is a matrix metalloproteinases (MMP) inhibitor
- MMP activation leads to AKI in ischemia reperfusion injury
- it is one of the few tetracyclines approved to be used in neonatal and infant populations
- Objective :assess the effect(s) of doxycycline on renal hemodynamics and injury using an in vivo newborn swine model
- Post resuscitation administration: improved renal perfusion, attenuated renal injury, and reduced tissue oxidative stress

frontiers in Pediatrics

ORIGINAL RESEARCH
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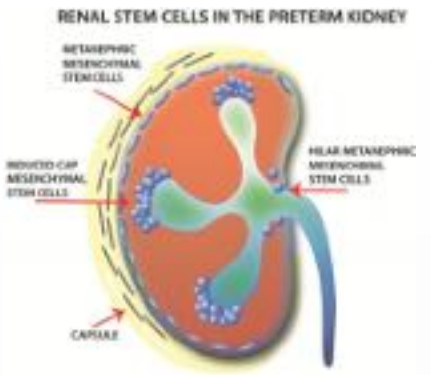
Attenuation of Acute Renal Injury After the Post-resuscitation Administration of Doxycycline in Surviving Newborn Piglets With Severe Hypoxia-Reoxygenation

Tze-Fun Lee[1,2], *Min Lu*[1,2], *Matteo P. Pasquin*[2,3], *Georg M. Schmölzer*[1,2] *and Po-Yin Cheung*[1,2,4*]

In newborn piglets surviving hypoxia-reoxygenation, a weak but significant and persistent attenuation of renal injury and improved recovery with the post-resuscitation administration of doxycycline was noted .

Potential therapies



- Antioxidant, antiadhesion molecule therapy, the administration of vascular mediators, or mesenchymal stem cells (MSCs) to prevent injury or promote recovery.
- Melanocyte-stimulating hormone has antiinflammatory activity and has been shown to protect renal tubules from injury. [149]
- Scavengers of free radicals and reactive oxygen and nitrogen molecules, as well as antiadhesion molecules, have been shown to decrease the degree of injury in animal models of AKI. [148]
- Multipotent MSCs may play a role in promoting recovery from AKI in animal models. [152]

Take Home Messages

Bundle for prevention of AKI in the neonate *(Harer et al, 2021)*

- Coordinated efforts to build awareness and educate neonatologists on issues related to AKI
- Identify baby risk factors (Pre, ante and post natal)
- Neonates have very long hospital stays with continues possibility of developing AKI. Incorporate a simple practical risk assessment system for procedures or new events is essential
- Routine KHA (kidney health assessment) done at 48 hours, then weekly for the first month then monthly
- ABCD of KHA: AKI history, Daily weight , urine output, **B**lood pressure, Creat determination, urine analysis, Drug list review
- KHR: medication adjustment, minimize exposure, monitoring

Table 2 High-risk neonates.

From: Improving the quality of neonatal acute kidney injury care: neonatal-specific response to the 22nd Acute Disease Quality Initiative (ADQI) conference

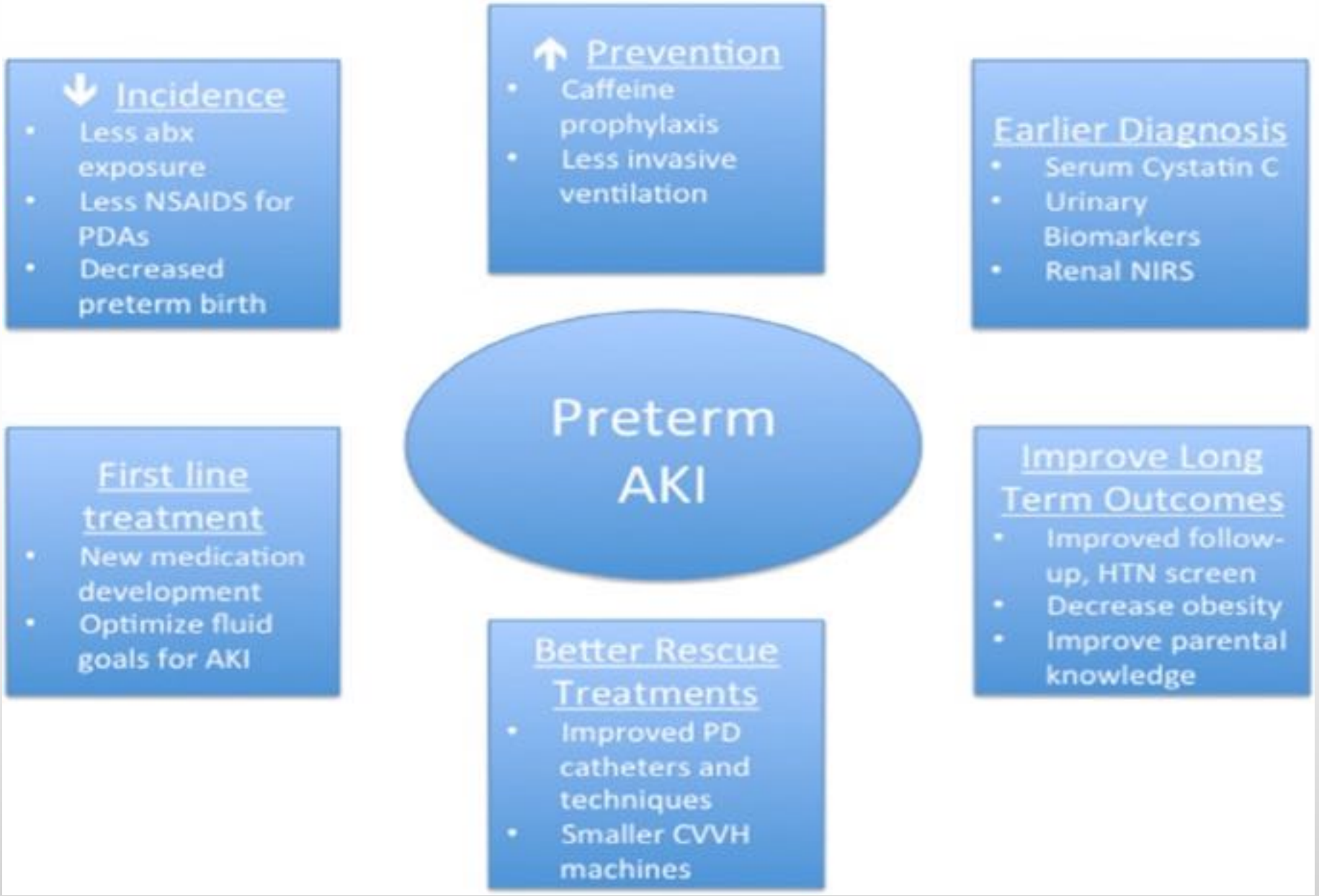
Population
Preterm birth <28 weeks
Small for gestational age
Birth weight <1500 grams
Congenital anomalies of the kidney or urinary tract (CAKUT)
Congenital heart disease
Undergoing cardiopulmonary bypass
Single ventricle physiology
History of heart transplant
Extracorporeal membrane oxygenation (ECMO)
Hypoxic-ischemic encephalopathy
High risk of dehydration
Unrepaired gastroschisis
Inherited cutaneous conditions

Table 4 Neonatal AKI bundle.

From: Improving the quality of neonatal acute kidney injury care: neonatal-specific response to the 22nd Acute Disease Quality Initiative (ADQI) conference

Evaluation of potentially modifiable risk and complications of AKI
Monitoring volume intake and output and daily weights
Calculate and track cumulative fluid overload
Evaluation of medications for nephrotoxic potential
Evaluation of underlying AKI cause/physiology
Treatment of hypoalbuminemia, hypotension, high abdominal pressure, hypoperfusion, bladder obstruction
Evaluation of nutrition/electrolyte composition of fluid intakes
Multidisciplinary approach: pharmacy, dietitian, bedside nurses

Goals to Improve Preterm AKI




Thank
you!