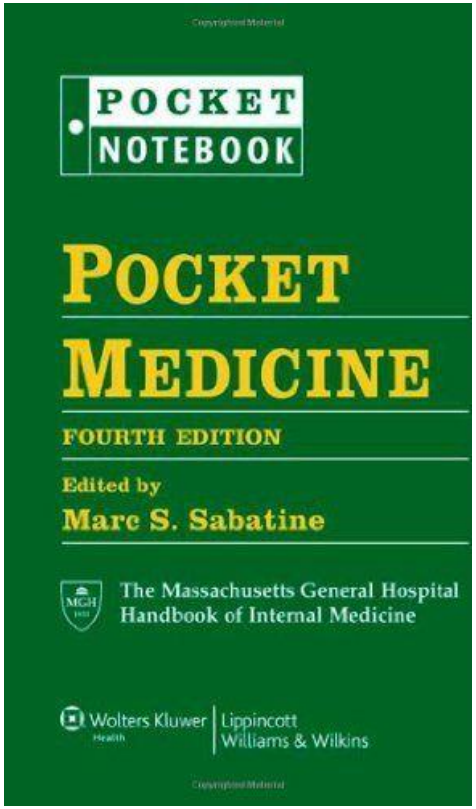
112 年度國考複習

腎臟科 CR 巴重翰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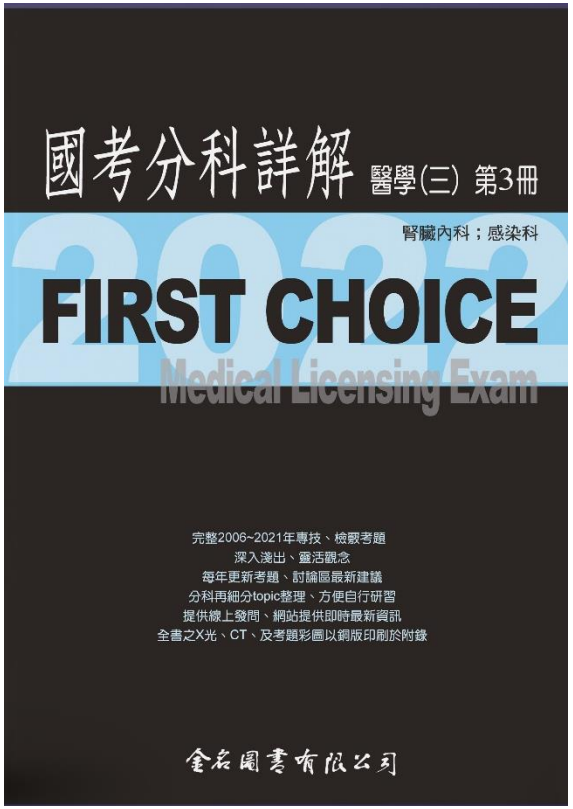
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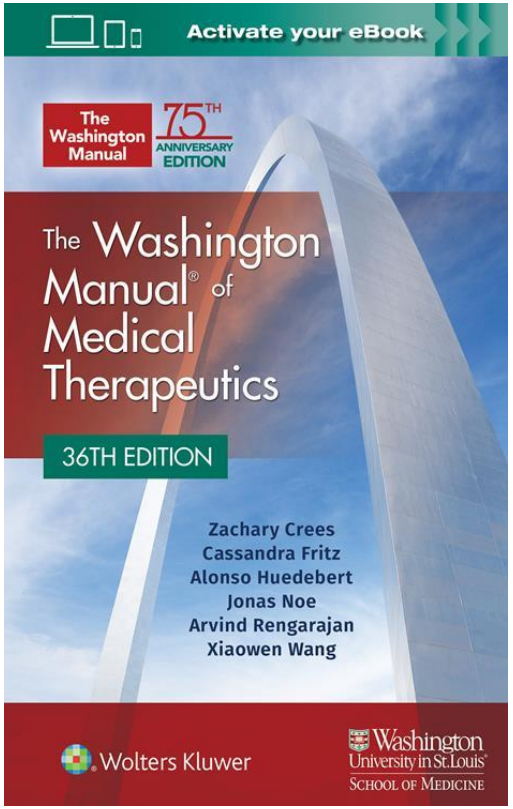
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醫師國考出題統計

- 約**5-6題? 連外科都出!** 都是考**臨床題**
- 是得分的重要部分
- 太難的就算了，反正大家都在猜
- 就算不會，猜大部分也猜的到！

By 蔡尚峰醫師

必拿考題!!!

- **酸鹼診斷判斷**
- 鉀離子**(低-診斷；高-診斷、處置)**
- **CKD-AKI**

聯想、關鍵字(疾病)!!!

	96	97	98	99	100	101	102	103	104	105	106	107	108	109	110	Total
第一篇　腎臟內科	9	20	16	16	18	15	18	14	12	16	12	16	19	8	2	211
甲、酸鹼平衡			1	2	2	1	3	1	1	2	1	5	3			22
乙、離子平衡	3	6	1	2	2	5	9	6	5	5	6	3	2	3	1	59
丙、急性腎損傷		5	1	4	6	1	1	2	1	2		1	6			30
丁、腎臟疾病各論	1	3	5	4	6	2	3	3	1	4	3	3	3			41
戊、慢性腎臟病	2	1		2	2	5	1		2	1		1	4	3		24
己、透析治療	3	1	4	2				1	2	1	1	1				16
庚、腎臟移植		1	1								1		1	2	1	7
辛、多囊性腎病		3	3			1	1	1		1		2				12

111-1-4

某位70歲女性因為喝有機磷（organophosphate）農藥自殺而被送來醫院急救。當農藥毒性發作時，最不可能導 致下列何種徵象？

A.肌肉無力

B.口水、淚液減少

C.癲癇發作（seizure）

D.腹瀉、大便失禁

有機磷中毒的症狀

111-1-5

承上題，針對上述病人之治療，下列何種方式最不適當？

A.服用活性碳
B.注射atropine
C.注射physostigmine
D.注射pralidoxime（2-PAM）

有機磷中毒的處置

111-1-23

53歲男性腎臟移植病人，移植腎功能惡化，1個月內serum creatinine 由1.0 mg/dL，上升至2.0 mg/dL，移植腎 切片顯示內皮細胞損傷（endothelial injury）、C4d 沉積，下列何種治療最不適當？

A.血漿置換（plasmapheresis）
B.免疫球蛋白注射（immunoglobulin infusion）
C.anti-IL2 接受器（anti-IL2 receptor）單株抗體注射
D.anti-CD20 單株抗體（rituximab）注射

腎移植急性排斥的處置

AMR or ACR?

111-1-25

一名50歲男性尿路結石病人的尿液檢查發現結晶物質如附圖所示，下列何項建議最為適當？

A.consume a low-calcium diet from now on
B.need not modulate urine pH
C.start taking tiopronin or penicillamine
D.supply high-dose vitamin C

腎結石種類與鏡檢

Urine routine 判讀

尿液酸鹼?

Radiolucent/opaque?

111-1-26

下列何項不是急性間質性腎炎（acute interstitial nephritis）的常見臨床表現？

A.尿液分析僅輕度蛋白尿（＜1.0 g/24 hours）和血尿
B.血清肌酸酐上升合併鈉排出率（fractional excretion of sodium）＞1.0
C.使用氫離子幫浦阻斷劑後發生腎病症候群程度的蛋白尿（nephrotic-range proteinuria）
D.腎衰竭發展耗時數日至數週，符合原發性免疫反應動力學（kinetics）

Nephritis的臨床表現

Nephrotic/nephritis的定義?
侵犯Interstital/glomerular的表現

111-1-27

有關多囊性腎臟病的敘述，下列何者錯誤？

A.自體顯性多囊性腎臟病（autosomal dominant polycystic kidney disease, ADPKD）是最常見的遺傳性腎臟疾病

B.自體顯性多囊性腎臟病（autosomal dominant polycystic kidney disease, ADPKD）的致病基因目前已知的有兩 型：第一型：PKD1 基因，位在第16對染色體短臂上（16p13.3）；第二型：PKD2 基因，位在第4對染色體 長臂上（4q21-q23）

C.多囊性腎臟病的患者，約3.2%~10%會發生腦部動脈瘤，男性有較高的盛行率，特別是年紀小於30歲者

D.多囊性腎臟病的患者與一般末期腎病不同的是，患者發生嚴重貧血的機率較小，當惡化至尿毒症時，患者一 樣可接受透析治療或腎臟移植

多囊腎

ADPKD/ATPKD

AD, 基因型, 年齡, 併發多囊肝

症狀, 感染之處置

111-1-28

下列原發性腎病變引起的腎病症候群（nephrotic syndrome），那一種最容易發生血栓併發症（如：renal vein thrombosis、pulmonary embolism和deep-vein thrombosis）？

A.type I membranoproliferative glomerulonephritis（MPGN）

B.focal segmental glomerulosclerosis（FSGS）

C.membranous nephropathy（MN）

D.minimal change disease（MCD）

Neprhotic syndrome

各GN類型; 凝血因子製造增加

成人,小孩,Lupus nephritis, 感染後(IgA)

111-1-29

在皮質集尿管（cortical collecting duct）有三種上皮細胞，下列敘述何者正確？

A.主細胞（principal cells）是負責鉀離子的再吸收細胞

B.作用在主細胞（principal cells）包括：醛固酮，K + -sparing利尿劑及醛固酮拮抗劑

C.A型間細胞（type A intercalated cells）控制酸的再吸收與重碳酸根分泌

D.B型間細胞（type B intercalated cells）控制酸的分泌與重碳酸根再吸收

腎小管各段功能(太專了...)

111-1-30

關於急性腎損傷處置的敘述，下列何者錯誤？

A.對於非出血性休克病人的靜脈輸液，建議使用等張晶體溶液（isotonic crystalloid solution）

B.建議重症病人以低蛋白飲食來延緩透析

C.在體液容積足夠的情況下，furosemide stress test能夠作為急性腎損傷惡化的預測工具

D.不建議使用低劑量多巴胺（dopamine）來預防急性腎損傷

AKI處置

AKI的分類與原因 (pre/post/intrinsic)

AKI/CKD異同

111-2-1

病人主訴頻尿，每次尿量都很多，並未使用利尿劑。收集 24 小時尿液檢查結果：總尿量 3500 mL/day，urine osmolality 450 mosmol/L。病人的檢查數值，最符合下列那一種多尿症的診斷？

A.溶質性利尿（solute diuresis）
B.原發性多飲症（primary polydipsia）
C.中樞性尿崩症（central diabetes insipidus）
D.腎源性尿崩症（nephrogenic diabetes insipidus）

Polyuria的鑑別診斷

Osmolality, hypernatremia等判斷

111-2-2

關於懷孕期間慢性高血壓的治療建議，下列何者最不適當？

A.首選藥物為血管收縮素轉化酶抑制劑（angiotensin-converting enzyme inhibitor）或血管收縮素受體阻斷劑（angiotensin receptor blocker）

B.可以使用 labetalol 或 nifedipine 控制血壓

C.尚無足夠證據證實輕微高血壓的治療能改善懷孕預後

D.收縮壓目標控制在 130～150 mmHg，而舒張壓目標控制在 80～100 mmHg

懷孕期間高血壓用藥選擇

切記不可用

ACEI/ARB inhibitor!!!

111-2-25

下列何項不是馬兜鈴酸腎病變（aristolochic acid nephropathy）的臨床特徵？

A.marked protein-energy malnutrition
B.rapid progression to renal failure
C.upper urinary tract urothelial cancer
D.disproportionate anemia

表面上說馬兜鈴腎病變
實際上考AKI症狀與處置!!!

111-2-26

一名 60 歲女性，3 小時前開始左腰疼痛並出現血尿，體溫 38.3℃，靜脈腎盂攝影檢查 10 分鐘前、後呈像如附 圖左、右。下列何項治療處置應最優先考慮?

A.給與α1腎上腺素受體阻斷劑
B.照會泌尿科醫師尋求介入治療
C.投與經驗性抗生素並追蹤培養結果
D.開立止痛藥並鼓勵大量喝水

AKI應先排除post renal

Post renal的鑑別診斷與處置

一根shelly還是一根Foley?

111-2-28

有關 focal segmental glomerulosclerosis 何者錯誤?

A.FSGS 以血尿、高血壓、各種程度的蛋白尿及腎功能不全表現 的風險會進入透析治療

B.病理報告應包括在 corticomedullary junction disease（MCD）

C.病理報告 collapsing type FSGS with segmental or global glomerular

D.治療續發性的 FSGS，不需使用類固醇

各類GN的病理表現與治療

111-2-29

下列那一段腎小管（renal tubule）可透過抗利尿荷爾蒙（antidiuretic hormone, ADH）的作用增加對水分的通透性？

A.近端腎小管（proximal tubule）
B.Henle 氏環下行枝（descending limb of Henle's loop）
C.遠端腎小管（distal convoluted tubule）
D.集尿管（collecting duct）

ADH作用在後段腎小管
各段腎小管之功能?

111-2-30

一位 50 歲男性因懷疑腎結石接受 intravenous urography（IVU）檢查，第 3 天發現 BUN 由 30 mg/dL 升至 70 mg/dL，血中 creatinine 由 1.5 mg/dL 升至 4.5 mg/dL。下列有關顯影劑與腎病變的敘述，何者正確？

A.顯影劑可經過 reactive oxygen species 之機轉，直接傷害腎絲球

B.血中 creatinine 通常在 10～14 天達高峰，於 2～3 週後開始恢復

C.顯影劑之種類、劑量與腎損傷之嚴重程度無關

D.病人若有 multiple myeloma 或既有的腎臟疾病，較容易發生此併發症

Contrast-medium nephropathy與AKI

MM會讓血液黏稠

Acid-Base Disturbances

Sodium and Water Homeostasis

Potassium Homeostasis

Renal Failure/ other electrolyte

Glomerular Disease

Urinalysis

Nephrolithiasis

Acid-Base Disturbances

Gas 判斷:

Acid-**B**ase → **C**ompensate

Diuretic- **BG syndrome**

Etiology- Rx

必記數字!

pH **7.4**, CO_2 **40**, HCO3- **24**,

AG=[Alb]*2.5, Δ AG/ Δ HCO_3^- = 1~2

UAG (-), osm G: 10

Acidemia vs. acidosis

pH絕對值: ex. pH< 7.35= acidemia

-osis: 預測值相比較，身體**處理過程**

primary vs. secondary disorder

Primary Disorders				
pH	HCO_3	P_aCO_2	Primary Disorder	Problem
↓	⇓	↓	Metabolic acidosis	Gain of H^+ or loss of HCO_3
↑	⇑	↑	Metabolic alkalosis	Gain of HCO_3 or loss of H^+
↓	↑	⇑	Respiratory acidosis	Hypoventilation
↑	↓	⇓	Respiratory alkalosis	Hyperventilation

primary

Metabolic Respiratory

secondary

身體代償會使HCO_3^-, CO_2 同方向

106-01

- 一位病人血液氣體分析顯示pH 7.51，PaCO2 49 mmHg， HCO_3^- 38 mmol/L，下列何者正確？
- A.代謝性酸中毒(酸血症)
- **B.代謝性鹼中毒**
- C.呼吸性酸中毒
- D.呼吸性鹼中毒

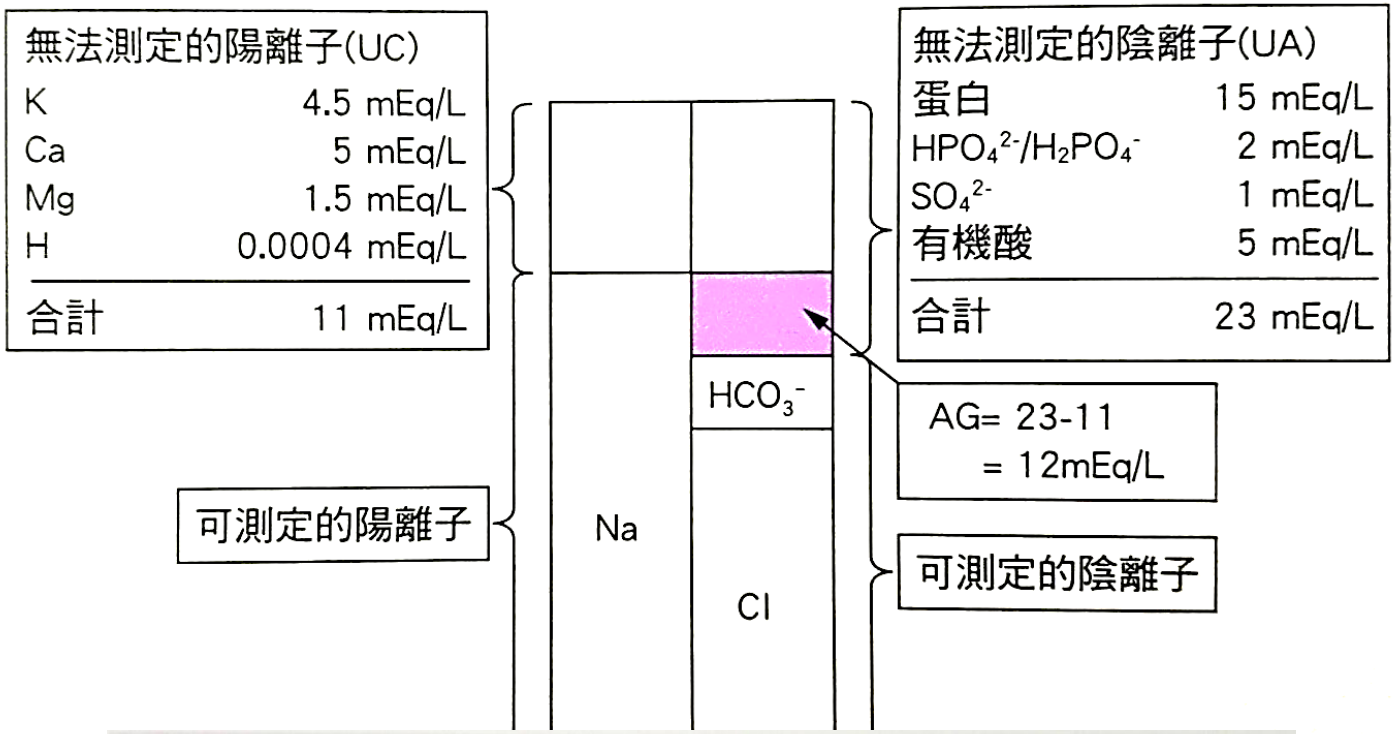
Compensation?

Compensation for Acid/Base Disorders (*NEJM* 2014;371:1434)		
Primary Disorder	**Expected Compensation**	
Metabolic acidosis	↓ $P_aCO_2 = 1.2 \times \Delta HCO_3$ or $P_aCO_2 = (1.5 \times HCO_3) + 8 \pm 2$ (Winters' formula) (also, $P_aCO_2 \approx$ last 2 digits of pH)	**1.25**
Metabolic alkalosis	↑ $P_aCO_2 = 0.7 \times \Delta HCO_3$ or $P_aCO_2 = 0.7\ (HCO_3\text{-}24) + 40 \pm 2$ or $HCO_3 + 15$	**0.75**
Acute respiratory acidosis	↑ $HCO_3 = 0.1 \times \Delta P_aCO_2$ (also, ↓ pH = $0.008 \times \Delta P_aCO_2$)	**1**
Chronic respiratory acidosis	↑ $HCO_3 = 0.35 \times \Delta P_aCO_2$ (also, ↓ pH = $0.003 \times \Delta P_aCO_2$)	**4**
Acute respiratory alkalosis	↓ $HCO_3 = 0.2 \times \Delta P_aCO_2$ (also, ↑ pH = $0.008 \times \Delta P_aCO_2$)	**2**
Chronic respiratory alkalosis	↓ $HCO_3 = 0.4 \times \Delta P_aCO_2$	**4**

Metabolic acidosis

AG →Δ/Δ → KILR or OG

NAG →GI, (NEP → UAG)



可測定的陽離子 [Na^+] ＋無法測定的陽離子 [UC]
＝可測定的陰離子 [Cl^- ＋ HCO_3^-] ＋無法測定的陰離子 [UA]
※UC: unmeasured cation, UA: unmeasured anion

- AG 異常時，可以推測以下疾病。

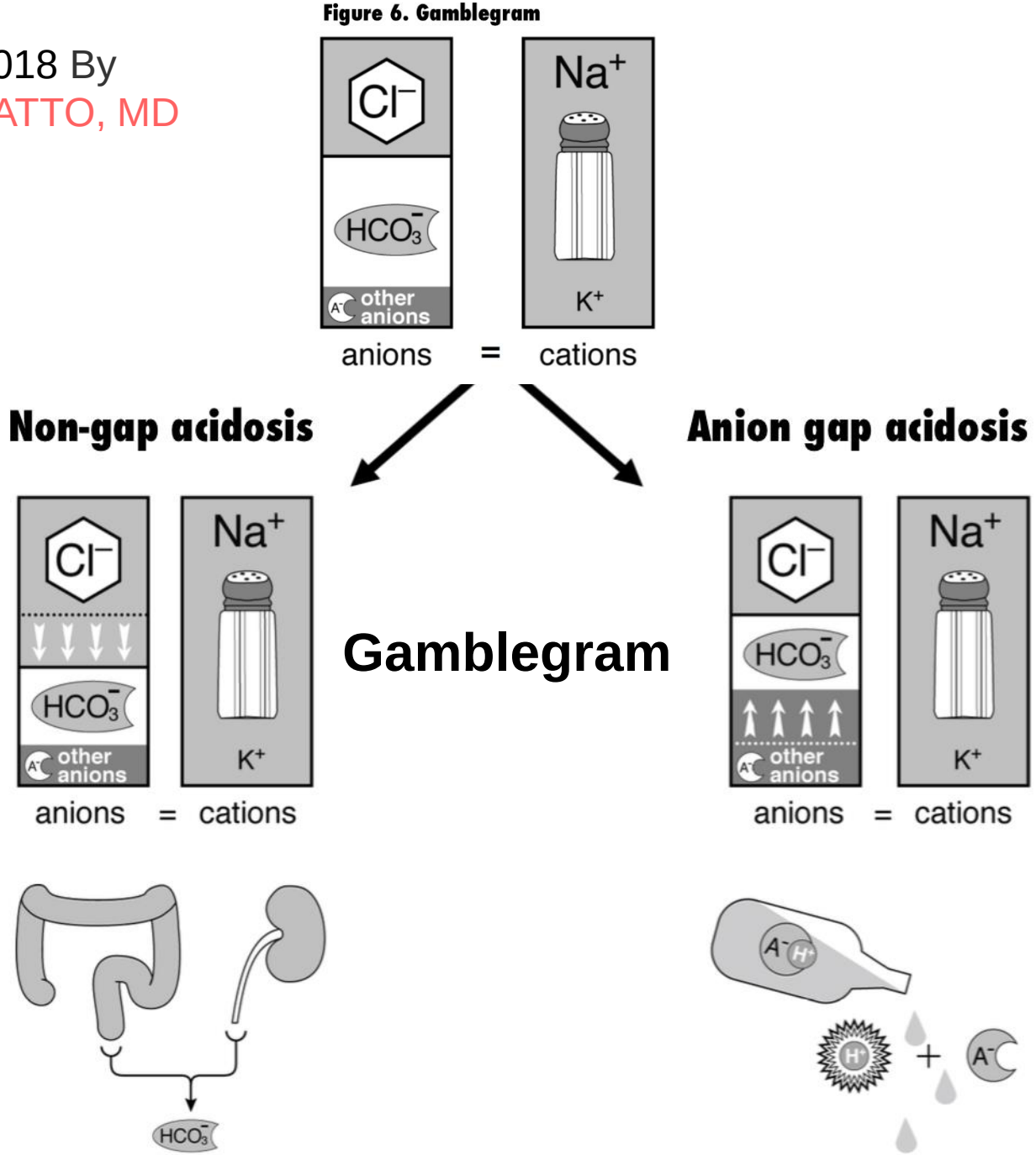
AG	UC（無法測定的陽離子）	UA（無法測定的陰離子）
⬆ ＞ 12 增加	⬇ hypogammaglobulinemia 低鉀、低鈣、低鎂	⬆ MUDPILES（見後述）
⬇ ＜ 12 減少	⬆ hypergammaglobulinemia 高鉀、高鈣、高鎂、鋰中毒	⬇低白蛋白血症

95-21下列何者**不會**造成血清低離子間隙(low anion gap)？

1. 鋰鹽中毒（Lithium intoxication）
2. 腎病症候群 (Nephrotic syndrome)
3. **高血磷症（ Hyperphosphatemia）**
4. 高血脂（ Hyperlipidemia)

low anion gap: UA↓ or UC↑ (Li,)
4. lab error due to hyperlipidemia (Spurious Hyperchloremia and Decreased Anion Gap in Hyperlipidemia)

MARCH 25, 2018 By
MATTHEW WATTO, MD



High anion gap ID: 1940104

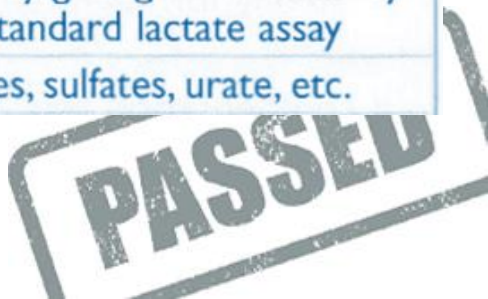
K

I

L

R

Etiologies of AG Metabolic Acidosis	
Ketoacidosis	**Diabetes mellitus,** alcoholism, starvation (*NEJM* 2014;372:546)
Ingestions	**Methanol** (windshield fluid, antifreeze, solvents, fuel): metab to formic acid **Ethylene glycol** (antifreeze): metab to glycolic and oxalic acids **Propylene glycol** (pharmaceutical solvent, eg, IV diazepam, lorazepam, and phenobarbital; antifreeze): lactic acidosis **Salicylates:** metabolic acidosis (from lactate, ketones) + respiratory alkalosis due to stimulation of CNS respiratory center **Glutathione depletion:** acetaminophen → ↑ endogenous organic acid 5-oxoproline in susceptible hosts (malnourished, female, renal failure)
Lactic acidosis (*NEJM* 2014; 371:2309)	**Type A:** impairment in tissue oxygenation eg, **circulatory or respiratory failure, sepsis,** ischemic bowel, carbon monoxide, cyanide **Type B:** no impairment in tissue oxygenation. ↓ clearance (eg, hepatic dysfxn) or ↑ generation [eg, malig, EtOH, thiamine def., meds (metformin, NRTIs, salicylates, propylene glycol, propofol, isoniazid, linezolid)] **D-lactic acidosis:** short bowel syndrome → precip by glc ingest → metab by colonic bacteria to D-lactate; not detected by standard lactate assay
Renal failure	Accumulation of organic anions such as phosphates, sulfates, urate, etc.



High anion gap- high osmolal gap

- **Osmolal gap** (OG) = measured osmoles – calculated osmoles
  calculated osmoles = (2 × Na) + (glucose/18) + (BUN/2.8)
  (can + [EtOH/4.6] if have EtOH level and want to test if other ingestions)
  OG >10 → suggests ingestion (see below) but lacks specificity (can be elevated in lactic acidosis, DKA, and alcoholic ketoacidosis)
  for methanol/ethylene glycol: early on, OG precedes AG; later OG may be nl with ⊕ AG

Ingestions (*NEJM* 2018;378:270) Call poison control for guidance (800-222-1222)

AG	OG	Ingestion	Other Manifestations
↑	nl	Acetaminophen	Hepatitis
		Salicylates	Fever, tachycardia, tinnitus; met. acid. + resp. alkalosis
↑	↑	Methanol	ΔMS, blurred vision, pupillary dilation, papilledema
		Ethylene glycol	ΔMS, cardiopulm. failure, hypoCa. **Ca oxalate crystals** → AKI. Urine fluoresces under UV light.
		Propylene glycol	AKI, liver injury
		Diethylene glycol	AKI, N/V, pancreatitis, neuropathy, lactic acidosis
nl/↑	↑	Isopropyl alcohol	ΔMS, fruity breath (acetone), pancreatitis, lactic acidosis
		Ethanol	Alcoholic fetor, ΔMS, hepatitis; keto + lactic acidosis ± met. alk. (vomiting)

104-19

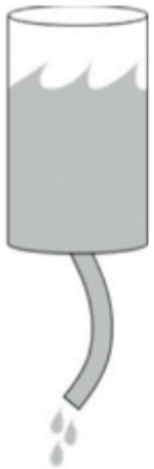
下列何者不是高陰離子間隙代謝性酸中毒(high-anion-gap metabolic acidosis）的原因?

- A. lactic acidosis
- B. ketoacidosis
- **C. diarrhea**
- D. salicylate intoxication

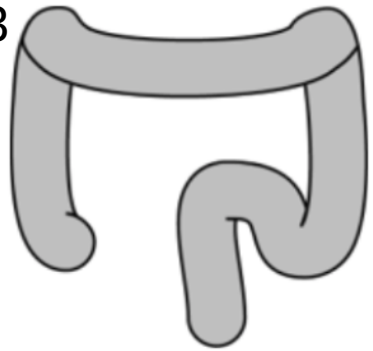
(叫不出酸的名字的！)

Three buckets of Non gap metabolic acidosis

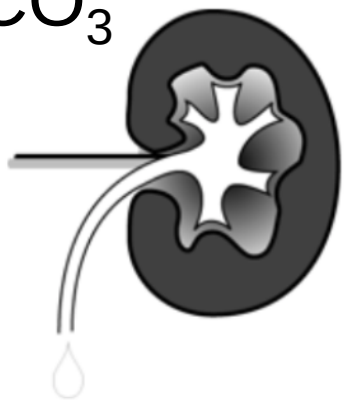
給 Cl



漏 HCO_3^-



漏 HCO_3^-



Chloride intoxication

Dilutional acidosis

HCl intoxication

Chloride gas intoxication

Early renal failure

GI loss of HCO_3

Diarrhea

Surgical drains

Fistulas

Ureterosigmoidostomy

Obstructed ureteroileostomy

Cholestyramine

Renal loss of HCO_3

Renal tubular acidosis

- Proximal
- Distal
- Hypoaldosteronism

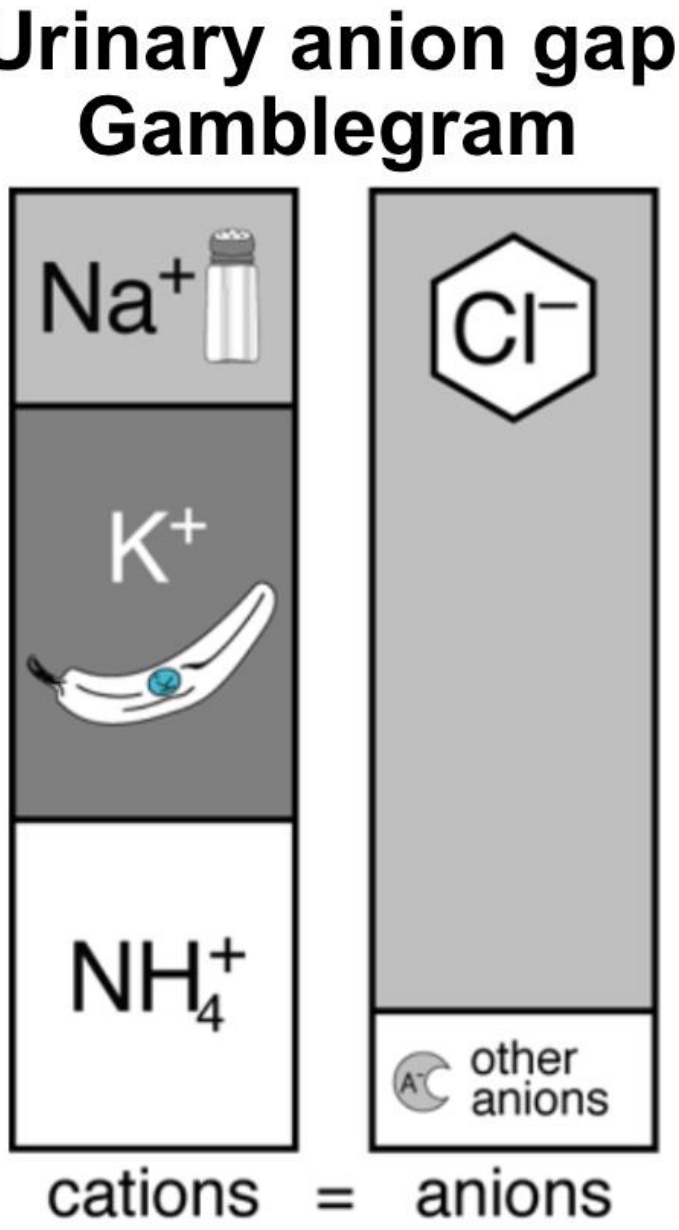
MARCH 25, 2018 By MATTHEW WATTO, MD

normal anion gap: GI, NEP

Etiologies of Non-AG Metabolic Acidosis	
GI losses of HCO_3	Diarrhea, intestinal or pancreatic fistulas or drainage
RTAs	*See section on renal tubular acidoses below*
Early renal failure	Impaired generation of ammonia
Ingestions	Acetazolamide, sevelamer, cholestyramine, toluene
Dilutional	Due to rapid infusion of bicarbonate-free IV fluids
Posthypocapnia	Respiratory alkalosis → renal wasting of HCO_3; rapid correction of resp. alk. → transient acidosis until HCO_3 regenerated
Ureteral diversion	Colonic Cl^-/HCO_3^- exchange, ammonium reabsorption

Urine anion gap= Urine: Na+K-Cl

- 腎排酸是以$NH_3+H^+=$**NH_4^+**來排出
- Urine: uNa+uK+**未**測cation=uCl+uHCO_3^-+**未**測得anion
- UAG=**未**測得anion-**未**測cation=uNa+uK-uCl
- 若腎功能好的，處於metabolic acidosis時，應該要排酸，故**urine中NH_4^+**應該是**上升**的
- 而NH_4^+屬於未測得cation，故**UAG<0**



metabolic acidosis

UAG= uNa+uK-uCl <0

MARCH 25, 2018 By MATTHEW WATTO, MD

PASSED

AG

KILR

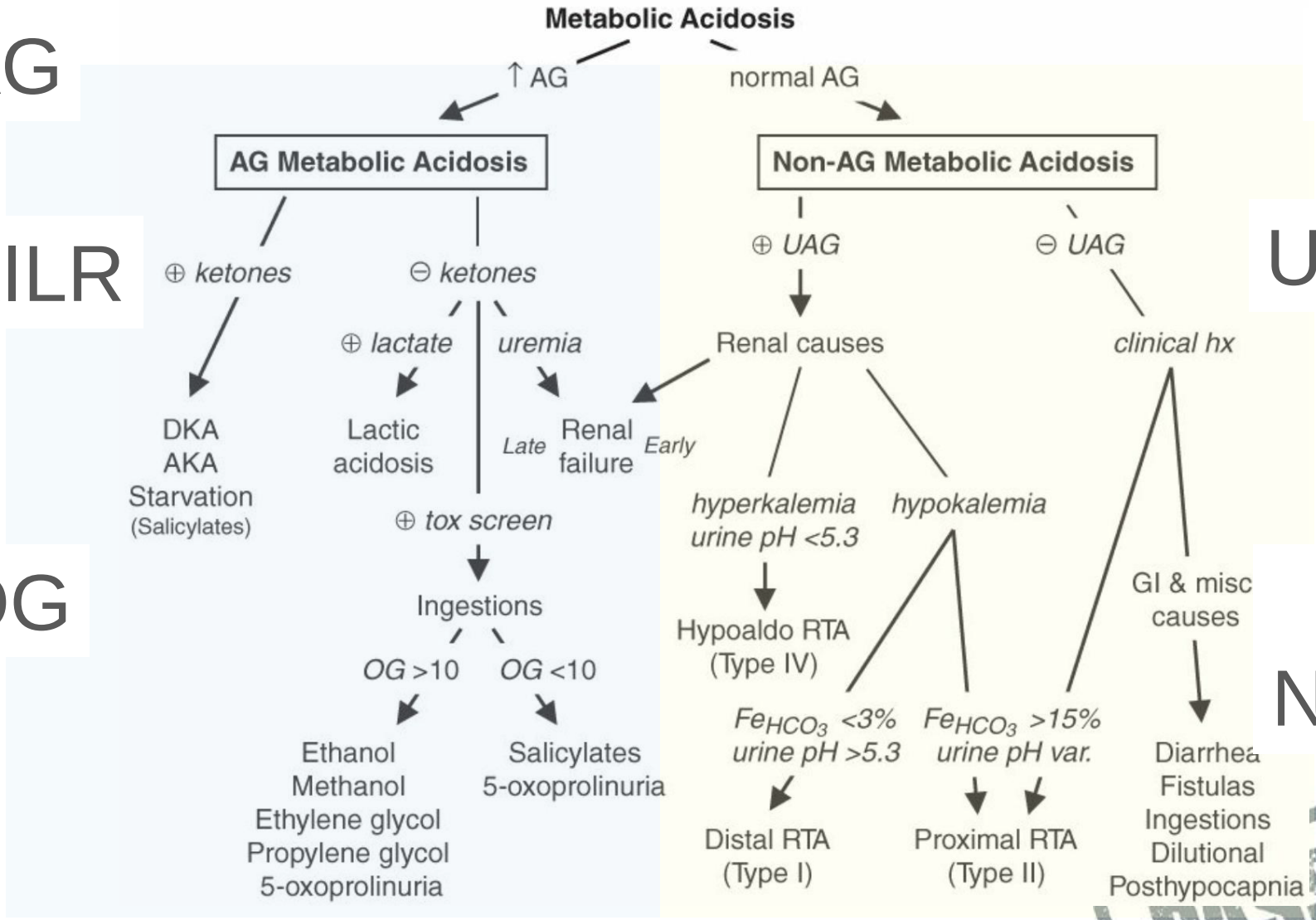
OG

AG

UAG

GI

NEP



RTA (UAG>0)

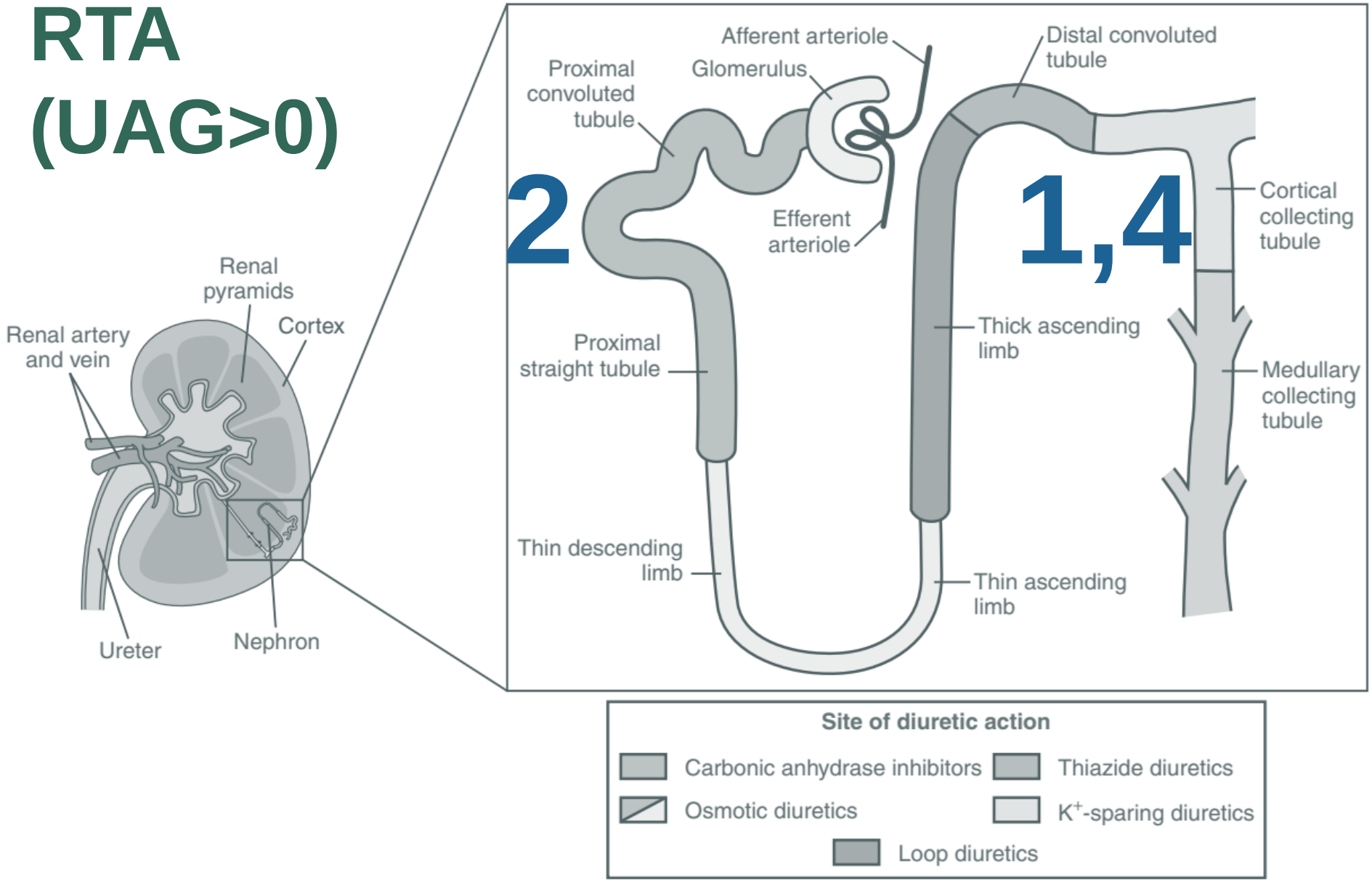


FIGURE 21–1 The nephron, depicting its location, segments, and the sites of action of different classes of diuretics.

Proximal (Type II): ↓ proximal reabsorption of HCO3

1° (Fanconi's syndrome) = ↓ proximal reabsorption of HCO3, PO4, glc, amino acids

Acquired: paraprotein (MM, amyloidosis), metals (Pb, Cd, Hg, Cu), ↓ vit D, PNH, renal Tx

Meds: acetazolamide, aminoglycosides, ifosfamide, cisplatin,topiramate, tenofovir

Distal (Type I): defective distal H+ secretion

1°, autoimmune (Sjögren's, RA, SLE), hypercalciuria, meds (ampho, Li, ifosfamide); normally a/w ↓ K; if **with ↑ K sickle cell, obstruction, renal transplant**

Hypoaldo (Type IV): hypoaldo → ↑ K → ↓ NH3 synthesis → ↓ urine acid-carrying capacity

↓ renin: diabetic nephropathy, NSAIDs, chronic interstitial nephritis, calcineurin inh, HIV

↓ aldo production: 1° AI, ACEI/ARBs, heparin, severe illness, inherited (↓ 21-hydroxylase)

↓ response to aldosterone

meds: **K-sparing diuretics, TMP-SMX**, pentamidine, calcineurin inhibitors
tubulointerstitial disease: sickle cell, SLE, amyloid, DM

酸鹼情境

High AG:

- DM(DKA, metformin)
- 誤食OOXX
- sepsis, shock
- Late renal failure

Non-AG: GI (吐拉), NEP

- DM: type 4 RTA
- Autoimmune: type 2 RTA (RA, SLE, SS)
- Early renal failure

Renal Tubular Acidosis								
Location	**Type**	**Acidosis**	**UAG**	**HCO_3^-**	**UpH**	**FE_{HCO_3} [b]**	**K**	**Complications**
Proximal	II	Moderate	±	12-20	<5.3[a]	>15%	↓	Osteomalacia
Distal	I	Severe	⊕	<10	>5.3	<3%	↓[c]	Kidney stones
Hypoaldo	IV	Mild	⊕	>17	<5.3	<3%	↑	Hyperkalemia

Treatment of severe metabolic acidoses (pH <7.2) (*Nat Rev Nephrol* 2012;8:589)

- DKA: insulin, IVF, K repletion (*NEJM* 2015;372:546); AKA: dextrose, IVF, replete K, Mg, PO_4
- Lactic acidosis: treat underlying condition, avoid vasoconstrictors, avoid "Type B" meds
- Renal failure: hemodialysis
- Methanol & ethylene glycol: fomepizole (20 mg/dL), vit. B_1 & B_6

  (ethylene glycol), folate (methanol), dialysis (if AKI, VS unstable, vision Δ or >50 mg/dL) (*NEJM* 2018;378:270)
- Alkali therapy: if pH <7.1 or <7.2 and co-existing AKI (*Lancet* 2018;392:21)
- $NaHCO_3$: amps by IV push or infusion of three 50-mmol amps in 1 L D_5W if less urgent

  can estimate mmol of HCO_3 needed as [desired-current $HCO_3]_{serum}$ × wt (kg) × 0.4

  side effects: ↑ volume, ↑ Na, ↓ ICa, ↑ P_aCO_2 (& ∴ intracellular acidosis; ∴ *must ensure adequate ventilation* to blow off CO_2)

} **underlying**

22. 56歲男性**糖尿病**人因胸部不適至急診，初步檢查血中creatinine 2.0 mg/dL、**K^+ 5.8** mEq/L、Na^+ 139 mEq/L、Cl^-116 mEq/L、$HCO3^-$ 18 mEq/L、osmolality 290 mOsm/kg·H_2O、尿中creatinine 12 mg/dL、K 9.6 mEq/dL、osmolality 580 mOsm/kg·H_2O，病人最可能的**診斷**是

A.第二型腎小管酸血症(type 2 renal tubular acidosis）

B.metformin引發酸血症(metformin related acidosis)

C.低腎素低醛固酮血症(hyporeninemic hypoaldosteronism)

D.糖尿病酮酸血症(diabetic ketoacidosis）

AG 139-116-18=5

95-10 一酗酒病患因意識不清，劇烈嘔吐被送至急診室。其血液生化檢查及動脈血分析如下: **pH: 7.40, PaCO2: 40 mmHg, HCO_3^-:24 mEq/L**, Glucose: 120 mg/dL, BUN: 10 mg/dL, Cr: 0.7 mg/dL, **Na: 134 mEq/L, K: 2.6 mEq/L, Cl: 80 mEq/L,** Acetone: 3+。則 (100-4數字完全一樣又考一次)

下列敘述何者正確？

1. 病患血液酸鹼值正常無代謝性酸鹼疾病
2. **病患患有代謝性酸中毒合併代謝性鹼中毒**
3. 病患患有代謝性酸中毒合併呼吸性鹼中毒
4. 病患患有代謝性鹼中毒合併呼吸性酸中

102-26

下列那一個病例較符合所列之動脈血氣體分析和血清電解質的檢查結果？ **pH 7.32， PaO2 110 mmHg， PaCO2 30 mmHg， HCO3- 18 mEq/L； Na+ 138， K+ 3.5， Cl- 97**(電解質的單位是 mmol/L)

A. 70 歲病人因便秘嚴重，服用 magnesium sulfate 導致**腹瀉**數天

B. 28歲病人診斷為**修格連氏症候群**(Sjögren's syndrome)，無意間發現腎鈣化(nephrocalcinosis)，尿液酸鹼值為 6.5；給予 NH4Cl(0.1 g/kg體重) 後，尿液酸鹼值為 6.0

C. 20 歲病人第一型糖尿病病史 5 年，血糖控制不佳，最近因為期末考胰島素注射次數減少

D. 60 歲病人因膽道阻塞放置**引流管引流膽汁**

AG=138-97-18=23>12

100-29
下列那一種是陰離子隙增加之代謝性酸中毒 (high anion gap metabolic acidosis) ?

1. 腹瀉(diarrhea)

2. 飢餓 (starvation)

3. 嘔吐(vomitting)

4. 輸尿管 - 乙形結腸造口術後

(ureterosigmoidostomy)

產生ketone (並無loss or gain)

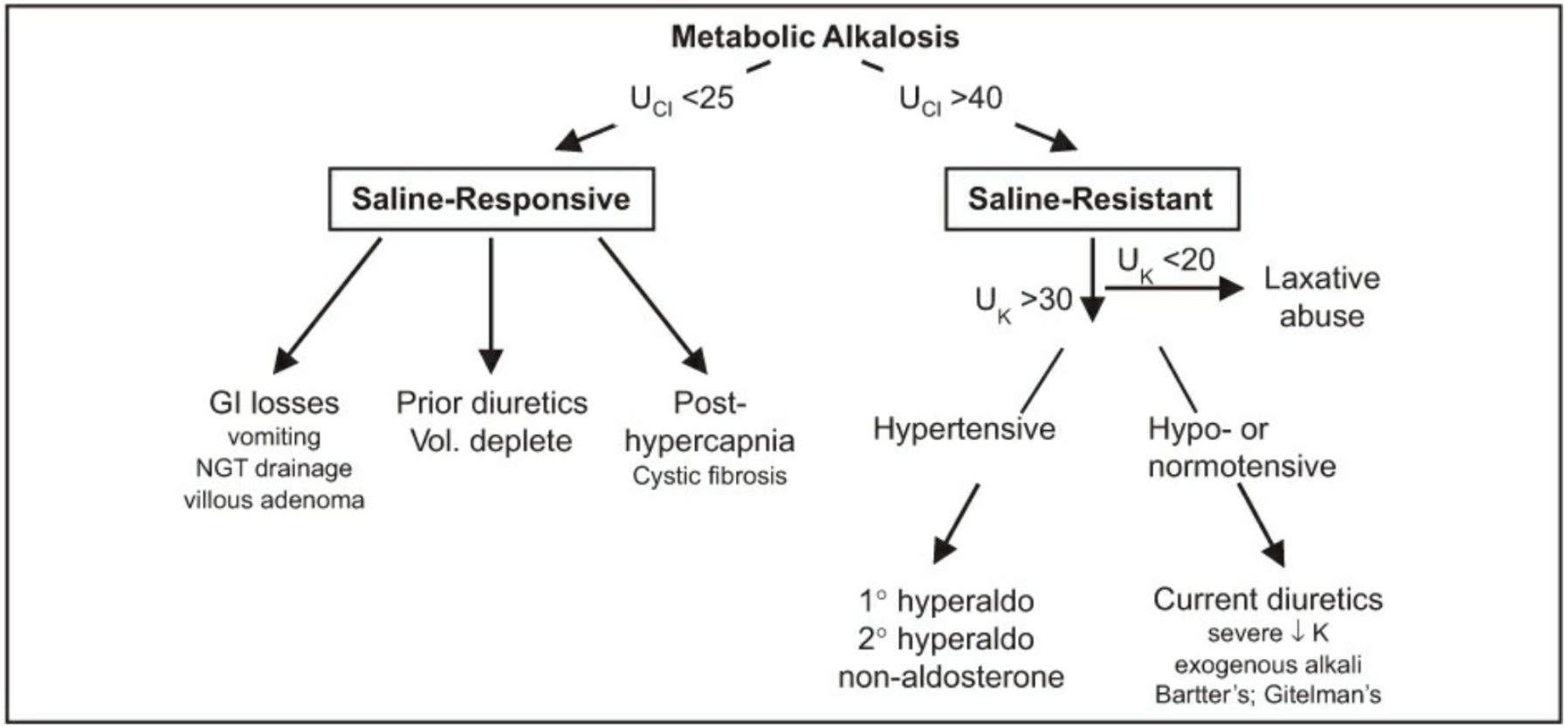
Metabolic alkalosis

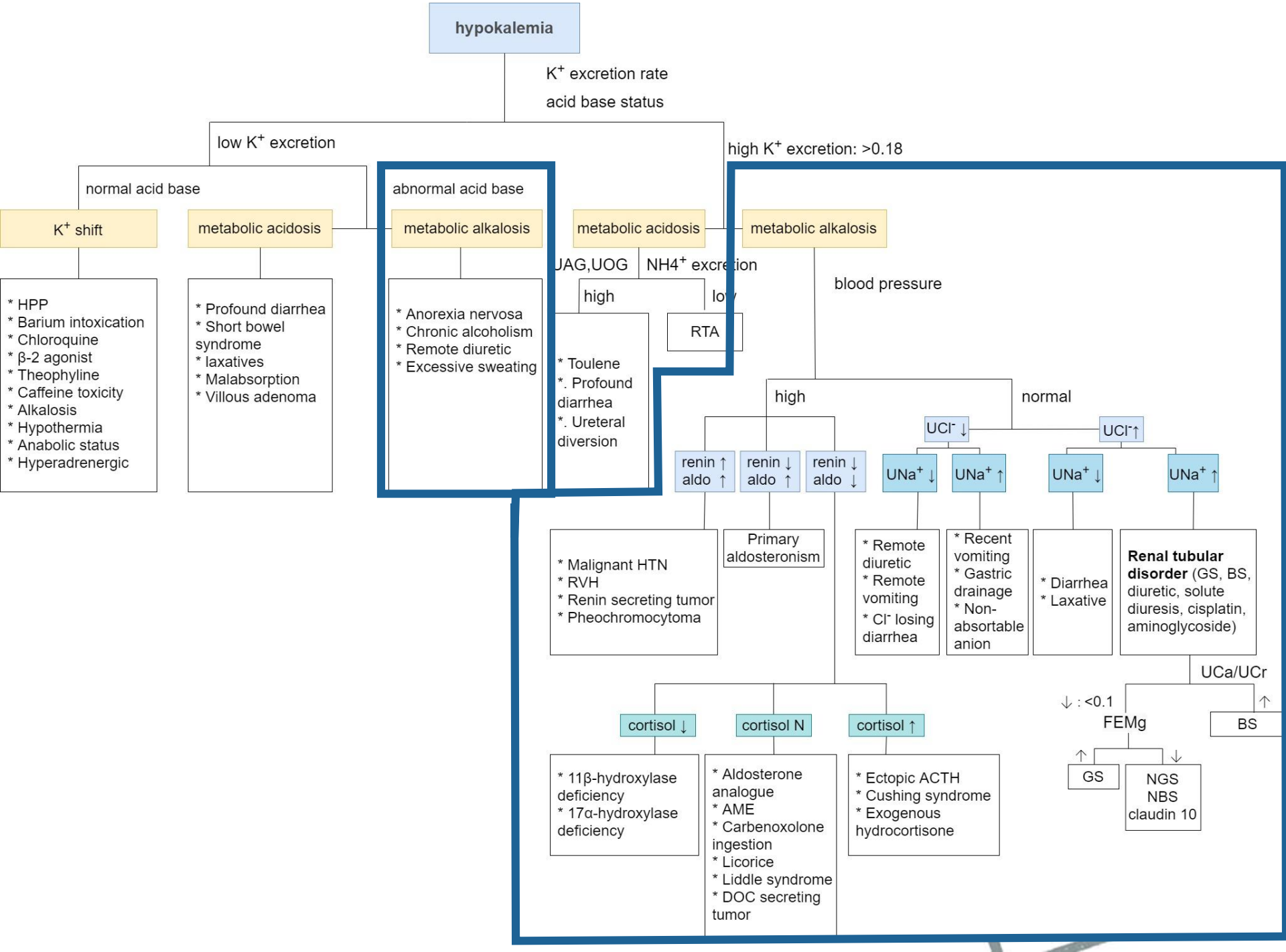
20 or 25

Etiologies of Metabolic Alkalosis	
Saline responsive UCl < 25 **Remote diuretic**	**GI loss of H^+**: emesis, NGT suction, villous adenoma, chloridorrhea **previous** **Renal loss**: loop/thiazide, ↓ Cl intake, milk-alkali, Pendred syndrome Posthypercapnia, sweat losses in cystic fibrosis
Saline resistant UCl >40 **endocrine**	*Hypertensive* **(mineralocorticoid excess)** 1° hyperaldosteronism (eg, Conn's) 2° hyperaldosteronism (eg, renovascular dis., renin-secreting tumor) Non-aldo (Cushing's, Liddle's, exogenous mineralocorticoids, licorice) *Normotensive* Severe hypokalemia (K<2); exogenous alkali load (w/ AKI or ↓ vol) Bartter's syndrome (loop-like); Gitelman's syndrome (thiazide-like)

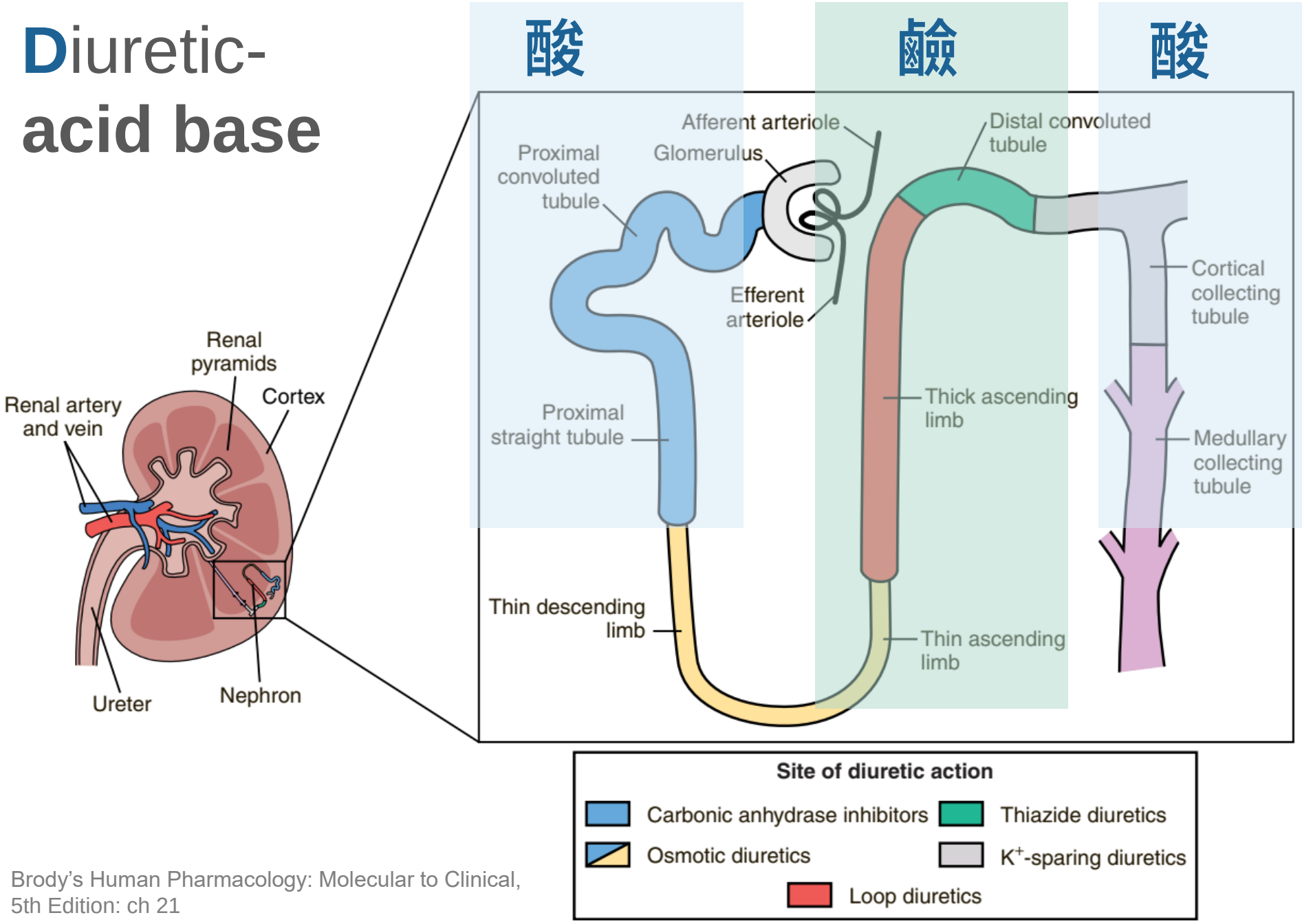
Metabolic alkalosis

用**UCl-**來看ECF volume的量！ ∵ urine HCO3-會把UNa拉出來故不準！




hypokalemia
K+ excretion rate
acid base status
low K+ excretion
high K+ excretion: >0.18
normal acid base
abnormal acid base
K+ shift
metabolic acidosis
metabolic alkalosis
metabolic acidosis
metabolic alkalosis
UAG,UOG
NH4+ excretion
high
low
blood pressure
* HPP
* Barium intoxication
* Chloroquine
* β-2 agonist
* Theophyline
* Caffeine toxicity
* Alkalosis
* Hypothermia
* Anabolic status
* Hyperadrenergic
* Profound diarrhea
* Short bowel syndrome
* laxatives
* Malabsorption
* Villous adenoma
* Anorexia nervosa
* Chronic alcoholism
* Remote diuretic
* Excessive sweating
* Toulene
*. Profound diarrhea
*. Ureteral diversion
RTA
high
normal
renin ↑ aldo ↑
renin ↓ aldo ↑
renin ↓ aldo ↓
UCl- ↓
UCl-↑
UNa+ ↓
UNa+ ↑
UNa+ ↓
UNa+ ↑
* Malignant HTN
* RVH
* Renin secreting tumor
* Pheochromocytoma
Primary aldosteronism
* Remote diuretic
* Remote vomiting
* Cl- losing diarrhea
* Recent vomiting
* Gastric drainage
* Non-absortable anion
* Diarrhea
* Laxative
Renal tubular disorder (GS, BS, diuretic, solute diuresis, cisplatin, aminoglycoside)
UCa/UCr
↓ : <0.1
FEMg
↑
BS
↑
↓
GS
NGS
NBS
claudin 10
cortisol ↓
cortisol N
cortisol ↑
* 11β-hydroxylase deficiency
* 17α-hydroxylase deficiency
* Aldosterone analogue
* AME
* Carbenoxolone ingestion
* Licorice
* Liddle syndrome
* DOC secreting tumor
* Ectopic ACTH
* Cushing syndrome
* Exogenous hydrocortisone

Diuretic-acid base

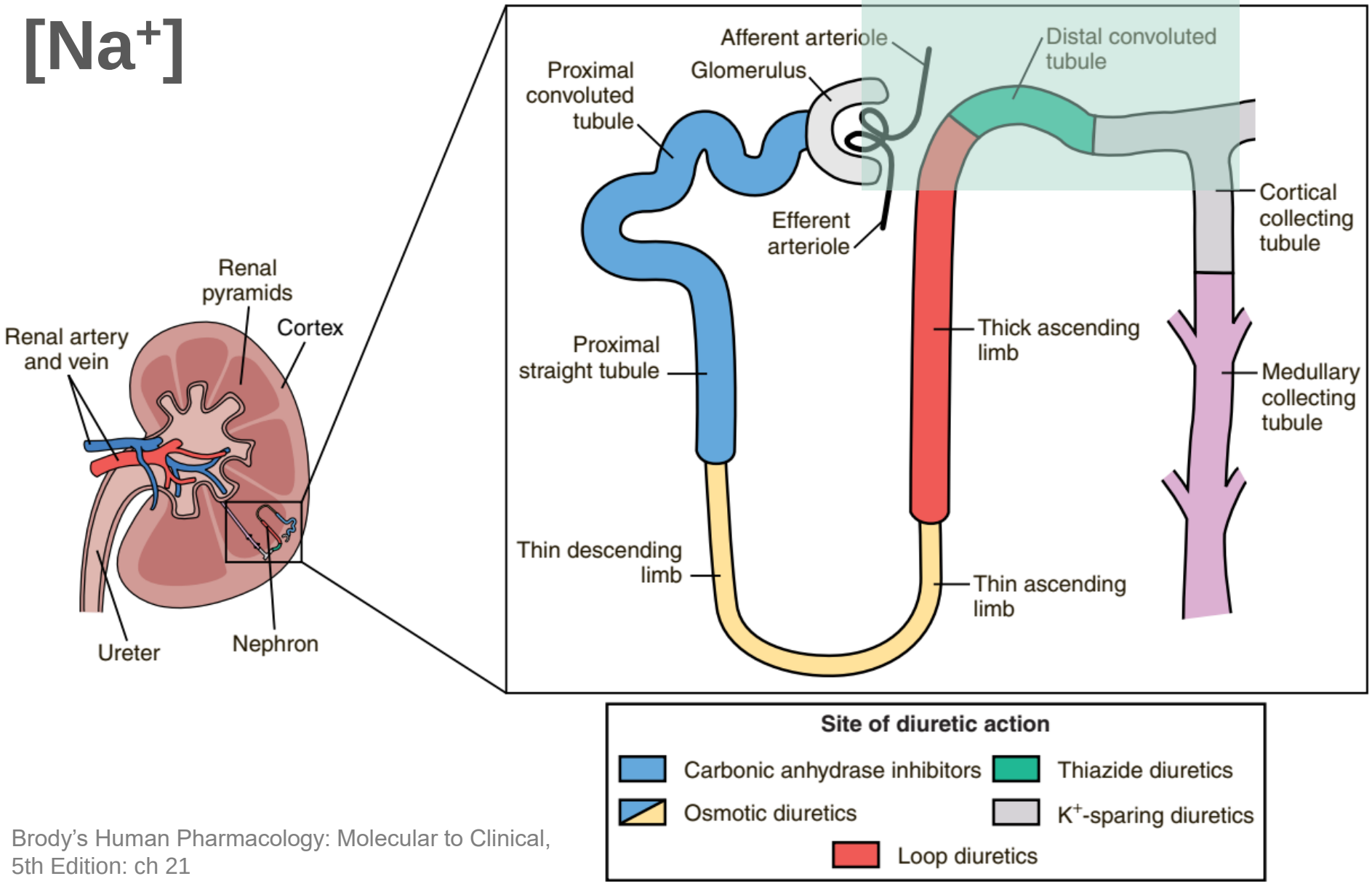


Brody's Human Pharmacology: Molecular to Clinical, 5th Edition: ch 21

FIGURE 21–1 The nephron, depicting its location, segments, and the sites of action of different classes of diuretics.

Diuretic-
[Na^+]

↓血鈉



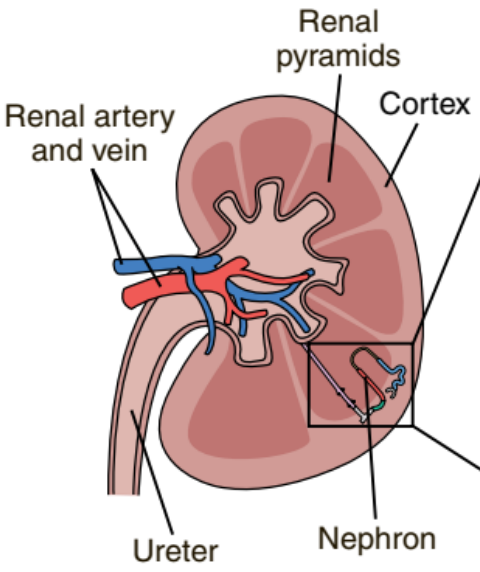
Brody's Human Pharmacology: Molecular to Clinical, 5th Edition: ch 21

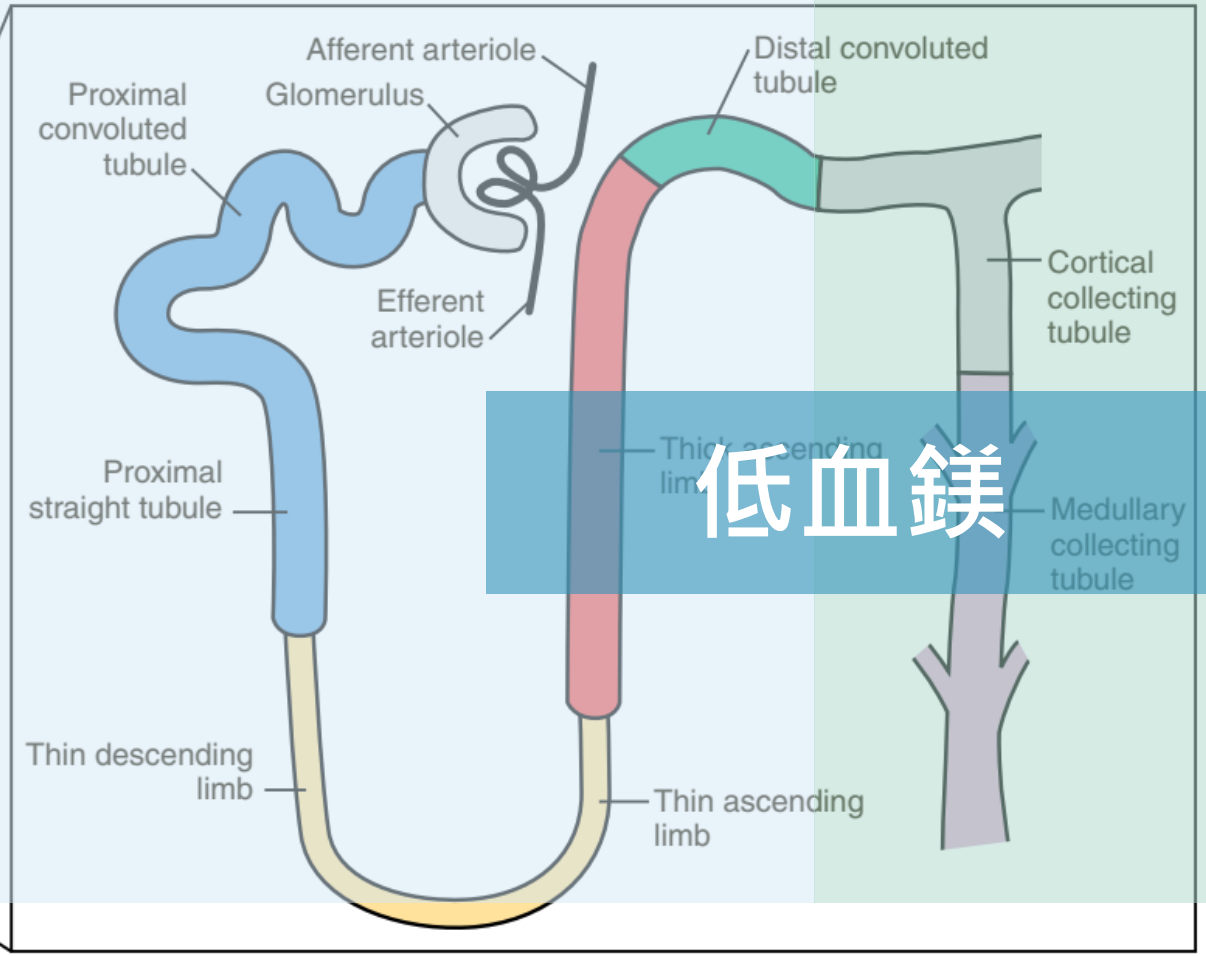
FIGURE 21–1 The nephron, depicting its location, segments, and the sites of action of different classes of diuretics.

Diuretic-
$[K^+]$ $[Mg^{2+}]$

低鉀

保鉀



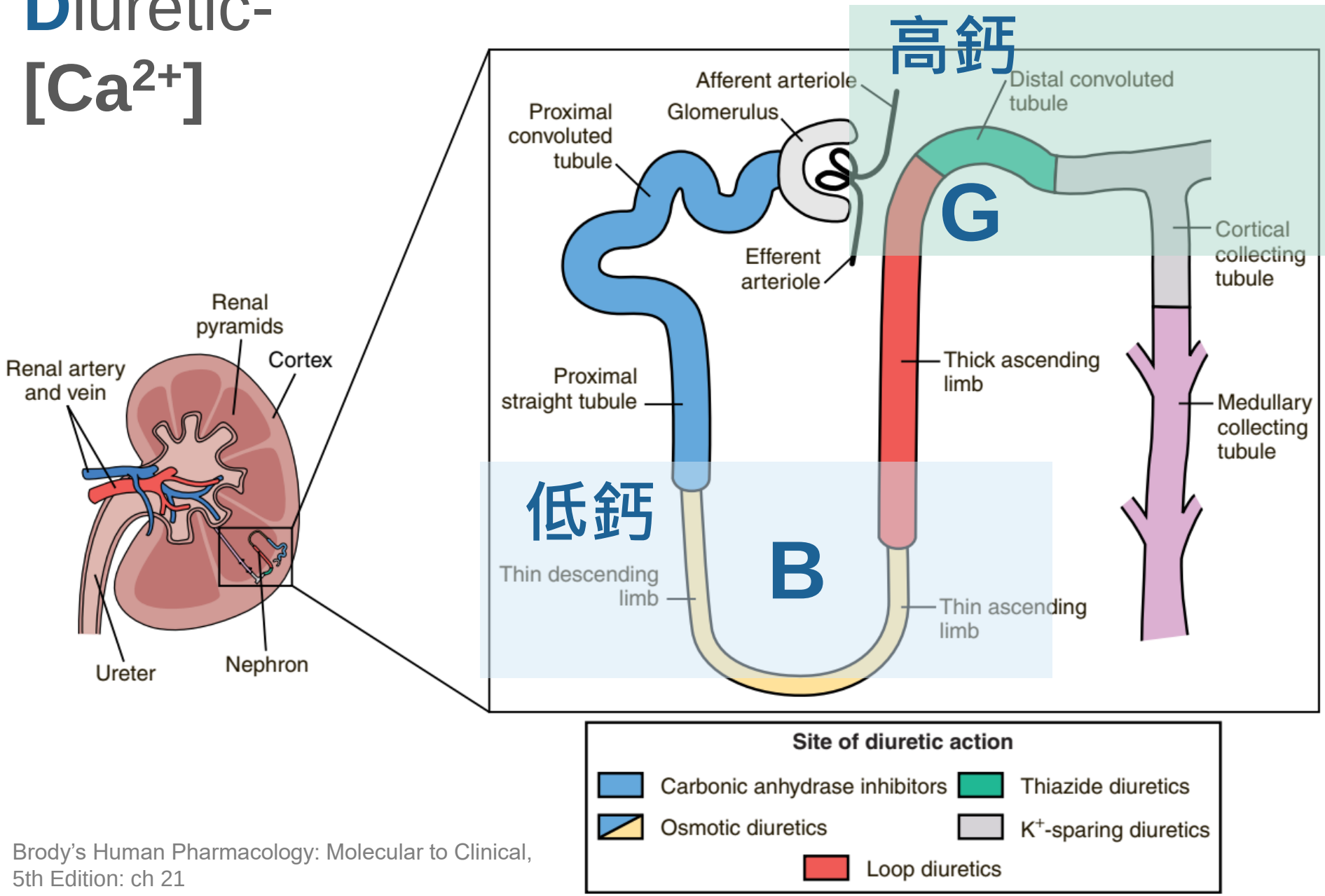


低血鎂

Brody's Human Pharmacology: Molecular to Clinical, 5th Edition: ch 21

FIGURE 21–1 The nephron, depicting its location, segments, and the sites of action of different classes of diuretics.

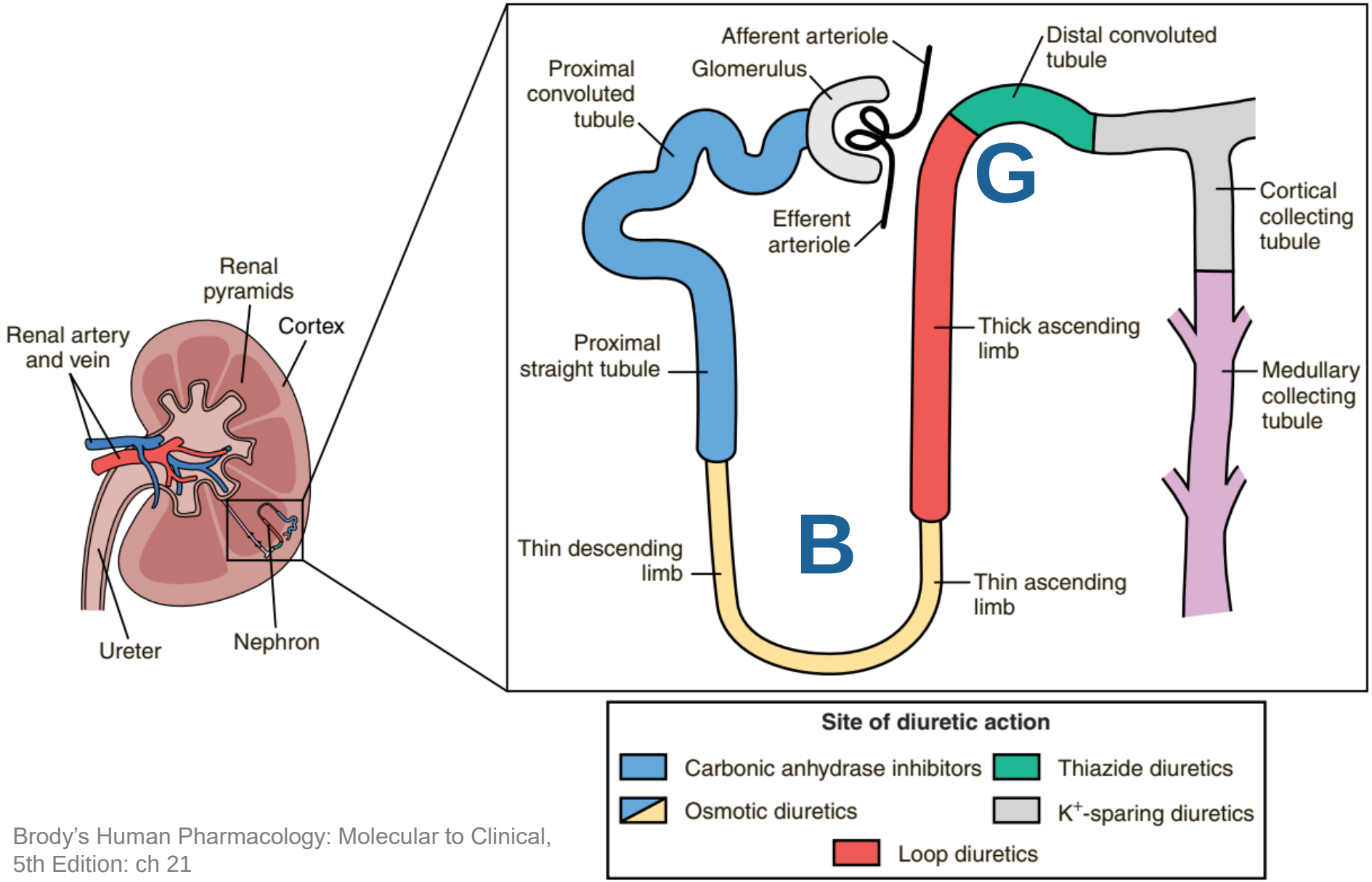
Diuretic-
[Ca^{2+}]



Brody's Human Pharmacology: Molecular to Clinical, 5th Edition: ch 21

FIGURE 21–1 The nephron, depicting its location, segments, and the sites of action of different classes of diuretics.

Diuretic- **BG syndrome**



Brody's Human Pharmacology: Molecular to Clinical, 5th Edition: ch 21

FIGURE 21–1 The nephron, depicting its location, segments, and the sites of action of different classes of diuretics.

	Barters	**G**itelman
位置	Thick ascending loop	Distal collecting duct
Transporter	Na-K-2Cl (NKCC2)	Na-Cl (NCC)
Medication analogue	Lasix (loop)	Thiazide
K	低血鉀	
Ca	Uca↑→血 Ca↓ ∴高血鈣要用lasix	Uca↓→血 Ca↑ ∴降結石用 thiazide
Mg	UMg↑	UMg↑

64.下列那一個病例**不符合**所列之動脈血氣體分析和血清電解質的檢查結果［pH 7.49，PaO_2 90 mmHg，$PaCO_2$ 43 mmHg，HCO_3^- 28；Na^+ 139，K^+ 3.0，Cl^- 89（電解質的單位是mmol/L）］？

(A)甲：40歲女病人，最近半年發現高血壓；病人有時會下肢無力，血漿皮質醛固酮濃度為38 ng/dL（正常為10～25）

(B)乙：37歲女病人，患有Sjögren's syndrome，腎臟超音波發現兩側腎臟的腎盂有鈣化跡象

(C)丙：30歲男病人因急性胰臟炎住院，給予鼻胃管引流，每天約引流出3000 mL

(D)丁：22歲女病人長期服用thiazide diuretics減肥

65.【題組】65.承上題，檢查該病人尿液，發現氯離子濃度為10 mmol/L。何者最為符合此結果？

(A)甲病人

(B)乙病人

(C)丙病人

(D)丁病人

102-31

31下列那一個病例不符合所列之動脈血氣體分析和血清電解質的檢查結果？ pH 7.49， PaO2 90 mmHg， PaCO2 48 mmHg， HCO3 32 mEq/L； Na+ 140， K+ 2.7， Cl-92（電解質的單位是 mmol/L）

A. 40歲甲病人，血壓 160/108 mmHg，血漿腎素活性 0.12 ng/mL/hr（正常值 1.0-3.5 ng/mL/hr）

B. 20歲乙女性，使用利尿劑 (hydrochlorothiazide)減重

C. 40歲丙病人，血壓 162/102 mmHg，長期食用甘草（ licorice）

D. 60歲丁病人使用 acetazolamide治療青光眼

100-26 下列選項何者不是 Gitelman’s syndrome 的特徵？

1. 低血鉀
2. 代謝性鹼中毒
3. 低血鎂
4. **高尿鈣**

Respiratory acidosis

Etiologies (also see “Hypercapnia”; PaCO2 = VCO2/VE(1-VD/VT); VE=RR x VT)

產CO_2酸、CNS到呼吸道、周邊肌肉

CO2 production (↑ VCO2): fever, thyrotoxicosis, sepsis, steroids, overfeeding

CNS depression: sedatives (opiates, benzos,etc.), CNS trauma, central sleep apnea, obesity, hypoventilation, hypothyroidism

Neuromuscular: Guillain-Barré, poliomyelitis, ALS, MS, paralytics, myasthenia gravis, muscular dystrophy, severe ↓ P & K, spinal cord injury

CHE review

Respiratory alkalosis

Hypoxia → hyperventilation: pneumonia, CHF, PE, restrictive lung disease, anemia

Primary hyperventilation
CNS stimulation, pain, anxiety, trauma, stroke, CNS infection, pontine tumors
drugs: salicylates toxicity (early), β-agonists, progesterone, methylxanthines, nicotine, pregnancy, sepsis, hepatic failure, hyperthyroidism, fever

Pseudorespiratory alkalosis: ↓ perfusion w/ preserved ventilation (eg, CPR, severe HoTN) → ↓ delivery of CO_2 to lungs for excretion; low $PaCO_2$ but ↑ tissue CO_2

2. 下列那一種藥物或狀況最不可能造成呼吸性鹼中毒？

A.水楊酸鹽(salicylates）

B.高山

C.懷孕

D.嗎啡(morphine）

108年第二次專 醫學(三)

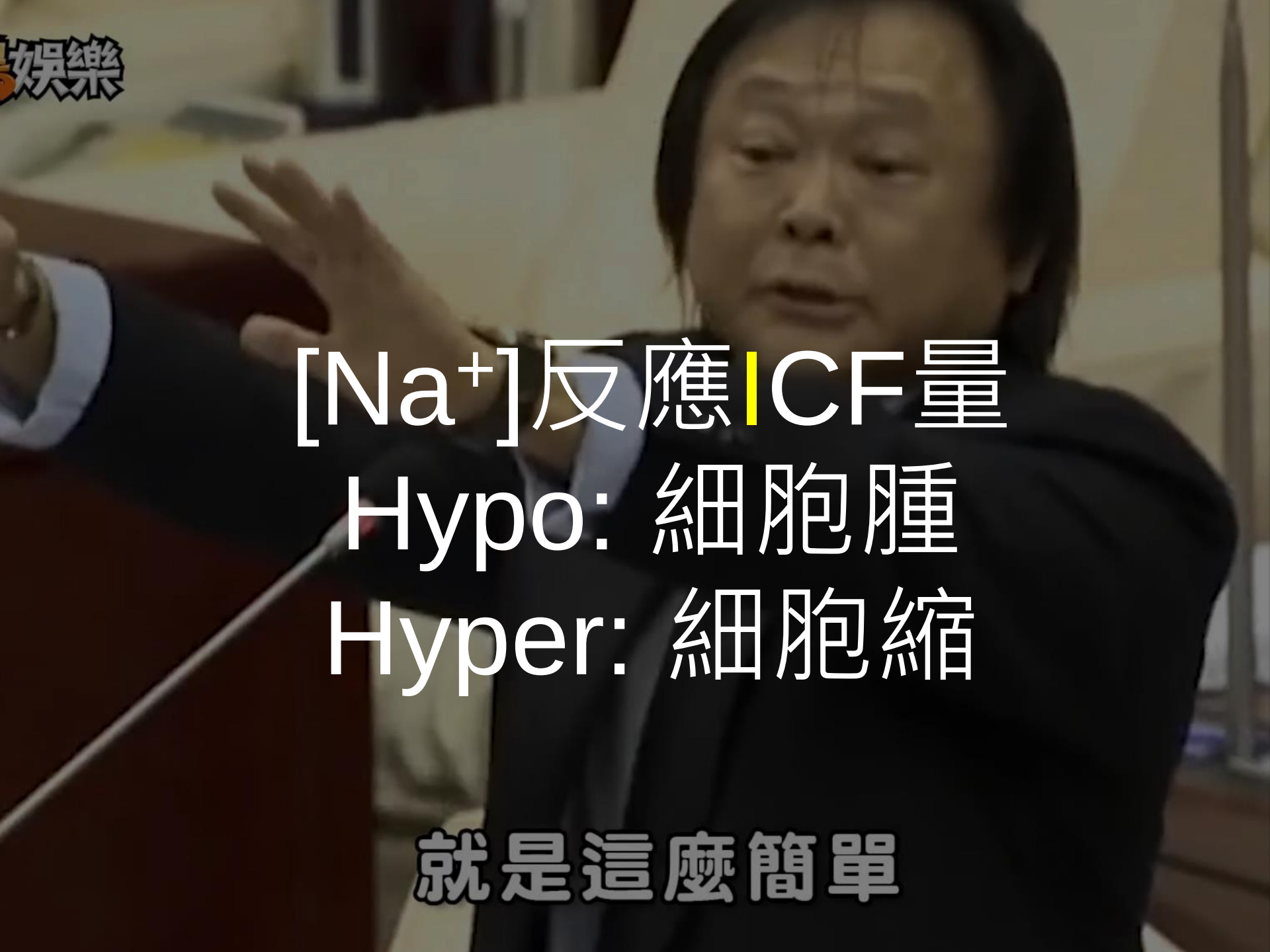
Sodium and Water Homeostasis

低鈉鑑別診斷、對應治療

高鈉鑑別診斷(尿崩)、對應治療

Overcorrection- complication


娛樂
Hyponatremia是指濃度低
Excess H2O relative to Na, usually due to ↑ ADH
就是這麼簡單


娛樂
[Na+]反應ICF量
Hypo: 細胞腫
Hyper: 細胞縮
就是這麼簡單

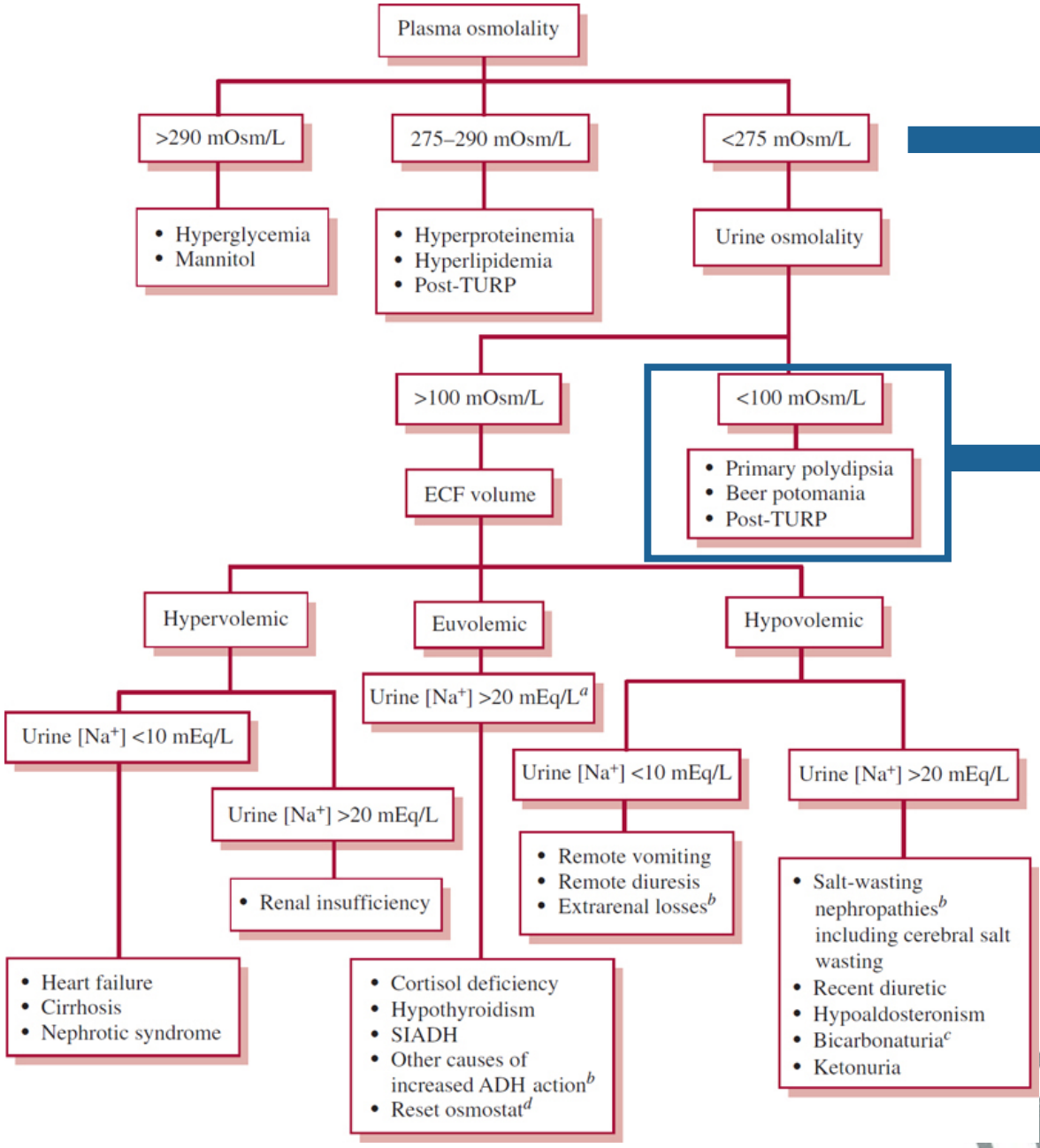
Hyponatremia

- 第一件事先**check tonicity**

1. Hypertonic hyponatremia：tonicity↑ eg. Mannitol, glucose↑

2. Isotonic hypontremia:假性, eg. Lipid↑, protien↑ (MM)

3. **Hypotonic hyponatremia**: 真的低血鈉，再往下approach

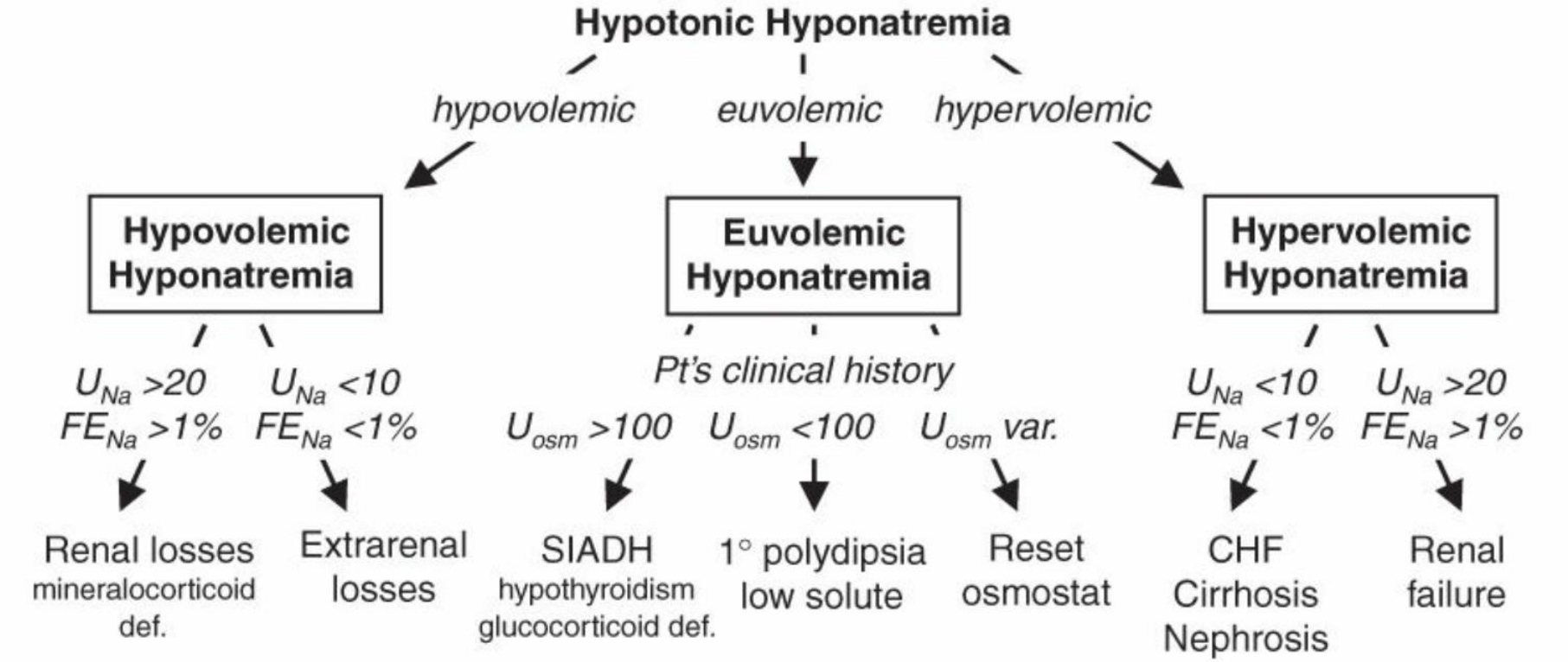


true

水多鈉少

PASSED

Washington: ch. 12 Fluid and Electrolyte Management



甲、固醇、水**腦**　心肝**腎**

腦: reset, ADH

Excess H2O relative to Na, usually due to ↑ ADH

↑ ADH may be appropriate (eg, hypovolemia or hypervolemia with ↓ EAV)

↑ ADH may be **inappropriate (SIADH)**

Hypovolemic hypotonic hyponatremia

(ie, ↓↓ total body Na, ↓ TBW)

Renal losses: esp. **thiazides**

Extrarenal losses: GI loss (diarrhea or vomiting), third-spacing (pancreatitis)

Euvolemic hypotonic hyponatremia (ie, ↑ TBW relative to total body Na

SIADH

Endocrinopathies: glucocorticoid deficiency/ **hypothyroidism**/myxedema coma

SIADH: 3C+藥物-CNS用藥

過度分泌ADH

Cancer: **Lung (SCLC),** GI, pancrease,lymphoma

CM: Pneumonia,abscess, TB, Aspergillosis, Resp failure, PEEP

CNS: Brain tumor, abscess, meningitis, encephalitis, trauma, SAH

Drug: **clorpropamide,** clofibrate, **narcotic, anti-psychi,** carbamazepine(Tegretol), **vincristine**, nicotine,

SIADH特色: euvolemic!!!!

$FE_{UricAcid}$ as >12% suggests SIADH

U_{Na} is usually above 40 mEq/L

$[K^+]$ is normal /no acid-base disturbance

[Uric acid] is frequently low

18.下列有關低血鈉症（hyponatremia）的描述，何者正確？

(A)如果血漿滲透壓（osmolality）偏低，應考慮是否有高血糖

(B)心臟衰竭可能造成細胞外體液（extracellular fluid）增加及低血鈉

(C)低血鈉及細胞外體液減少的病人，若尿液鈉離子濃度低於10 mmol/L，代表有Na+ wasting nephropathy

(D)抗利尿激素不適當分泌（SIADH）的病人通常血漿滲透壓正常，但細胞外體液減少

104-37
ADH:抗利尿—就是吸水！
抗利尿激素(anti-diuretic hormone) 不適當分泌症候群 (SIADH)，會造成下列何種電解質異常？

- A.高鈉血症
- **B.低鈉血症**
- C.高鉀血症
- D.低鉀血症

105-34

- 下列何種引起低血鈉的情況，最適合以注射0.9%生理食鹽水來治療？

- A.高血糖（ hyperglycemia）
- B.甲狀腺機能低下（ hypothyroidism）
- **C.皮質醛酮素缺乏（ aldosterone deficiency）**
- D. SIADH（syndrome of inappropriate ADH secretion）

要supple Na，最好是真的缺鈉

22 有關抗利尿激素分泌不當症候群（syndrome of inappropriate secretion of ADH）的敘述，下列何者錯誤？

(A)診斷必須排除腎上腺或甲狀腺功能不足

(B)通常是某些疾病的併發症，如肺結核、肺癌等

(C)病人出現低鈉血症，是由於體液過多所致

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(D)病人的尿液鈉濃度通常大於 10 mmol/L，且滲透壓大於 100 mOsm/kgH2O

Hyponatremia: therapy

Acute sx: initial rapid correction of [Na]serum (**2 mEq/L/h** for the first 2–3 h) until sx resolve

Asx or chronic symptomatic: **≤0.5 mEq/L/h**

Rate ↑ Na should not **exceed 6 (chronic) to 8 (acute) mEq/L/d**

Complication: **CPM/ODS:** paraplegia, dysarthria, dysphagia

Hypovolemic hyponatremia:

volume repletion with **isotonic 0.9% saline**

SIADH: **fluid restrict** + treat underlying

hypertonic saline ± loop diuretic: for sx

NaCl tabs or urea if chronic and no CHF

aquaresis: vaptans

Demeclocycline: causes nephrogenic DI, ↓ Uosm (rarely used)

Hypervolemic hyponatremia: free water restrict

w/ loop diuretics (avoid thiazide)

vasodilators to ↑ CO in CHF, colloid infusion in cirrhosis

一位56歲的男性病人因半夜發生端坐呼吸至急診就診，理學檢查發現血壓為164/98 mmHg，**不規則心跳120/min**，兩側肺部**有囉音**，心尖部有III/VI之全收縮期雜音及S3奔馬音，抽血檢查**Na+: 120 mEq/L**, K+: 4.5 mEq/L, Cl: 92 mEq/L。下列何種處置**最不適當？**

A. 再驗其尿中Na+濃度以釐清病因

B. 立即給予靜脈滴注3% saline以矯正其血中[Na^+]

C. 立即給予靜脈注射furosemide

D. 立即給予靜脈注射digoxin

99-11: hypervolemic hypoNa → diuretics

下列有關抗利尿激素不當分泌症候群（ syndrome of inappropriate antidiuretic hormone secretion, SIADH）的敘述，何者錯誤？

1.為體液量低下之低血鈉 hypovolemic hyponatremia

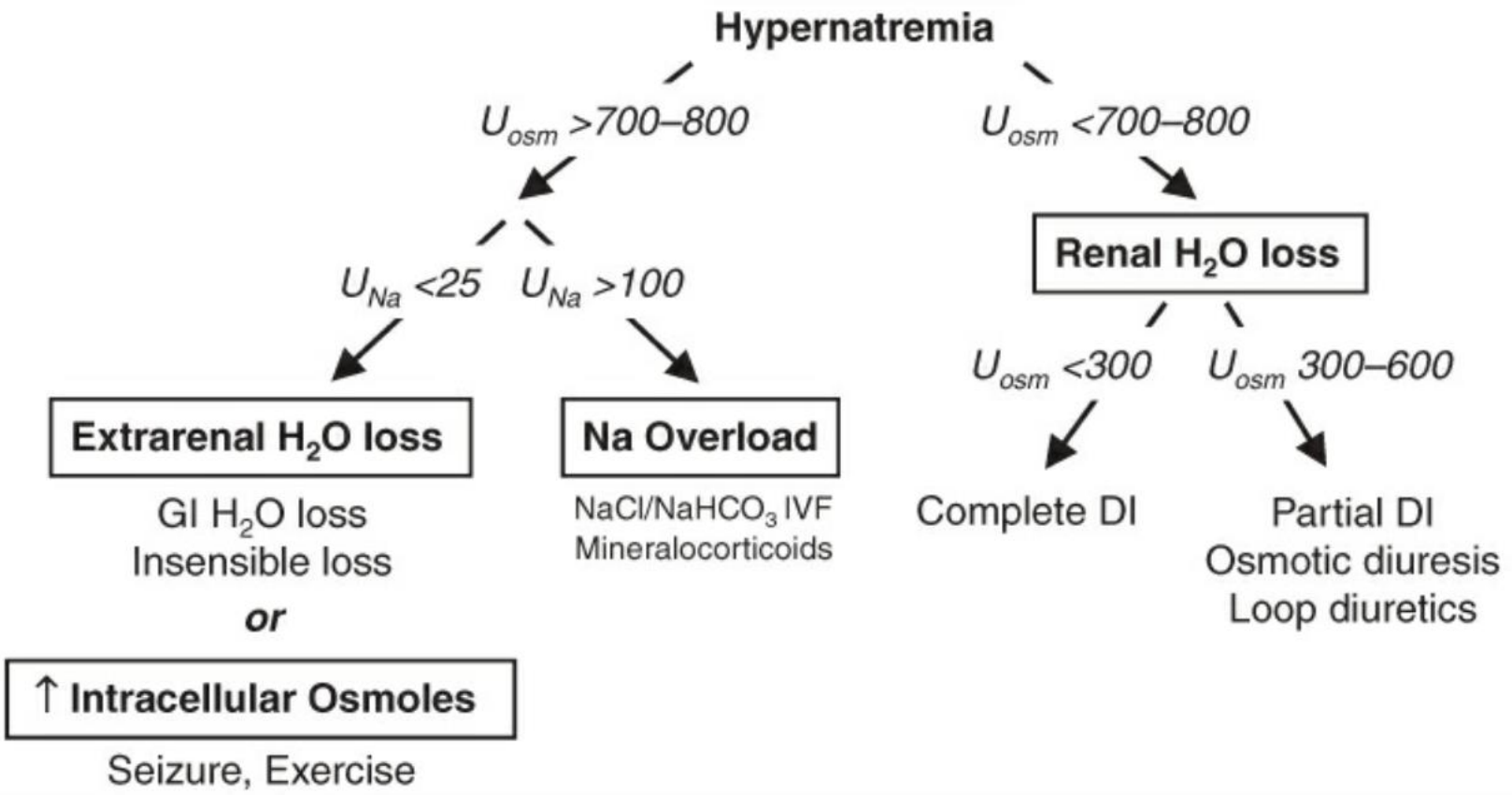
2.血清滲透度（ osmolality）低下

3.常合併血清低尿酸值

4.不適當之濃縮尿液，尿液滲透度 urine osmolality）常大於 100 mosmol/kgH2O

94-27: euvolemic


娛樂
Hypernatremia是指濃度高
Excess Na relative to H2O
就是這麼簡單


Hypernatremia
U_{osm} >700–800
U_{osm} <700–800
U_{Na} <25
U_{Na} >100
Renal H_2O loss
U_{osm} <300
U_{osm} 300–600
Extrarenal H_2O loss
GI H_2O loss
Insensible loss
or
↑ Intracellular Osmoles
Seizure, Exercise
Na Overload
NaCl/$NaHCO_3$ IVF
Mineralocorticoids
Complete DI
Partial DI
Osmotic diuresis
Loop diuretics


PASSED

Cause of hyperNa

Hypovolemic hypernatremia — **Water loss**

- **Renal H_2O losses** (U_{osm} 300-600): loop diuretics, osmotic diuresis (glc, mannitol, urea)
- **Extrarenal H_2O losses** (U_{osm} >600): diarrhea, insensible loss (fever, exercise)

Euvolemic hypernatremia

- **Diabetes insipidus** (U_{osm} <300-600): ADH defic. (central) or resist. (nephrogenic)
  - **Central**: congenital, trauma/surgery, tumors, or infiltrative disease of hypothalamus or posterior pituitary; also idiopathic, hypoxic encephalopathy, anorexia
  - **Nephrogenic** (*Annals* 2006;144:186)
    - congenital
    - drugs: **Li**, amphotericin, demeclocycline, foscarnet, cidofovir
    - metabolic: **hypercalcemia**, severe hypokalemia, protein malnutrition, congenital
    - tubulointerstitial: **postobstruction, recovery phase of ATN**, PKD, sickle cell, Sjögren's, amyloid, pregnancy
  - DI usually presents as *severe polyuria* and *mild hypernatremia*
- Seizures, exercise (U_{osm} >600): ↑ intracellular osmoles → H_2O shifts → transient ↑ $[Na]_{serum}$

Hypervolemic hypernatremia — **Excess Na**

- Hypertonic saline administration: eg, cardiac arrest resuscitation with $NaHCO_3$
- Mineralocorticoid excess: usually mild hypernatremia caused by ADH suppression

$$\textbf{Free } H_2O \textbf{ deficit (L)} = \frac{[Na]_{serum} - 140}{140} \times TBW$$

TBW = wt (kg) × 0.6 (♂) or 0.5 (♀);
if elderly use 0.5 (♂) or 0.45 (♀)

Rate of correction depends on acuity of onset and risk:
chronic (>48 hr): ~12 mEq/d appears safe w/o risk of **cerebral edema**

acute (<48 hr): may ↓ Na by 2 mEq/L/h until Na 145
hyperacute (min-hrs) & life threatening (ICH, seizure): rapidly infuse D5W ± emergent HD

2.一位35歲的男性，因為意識不清被家人送至急診就醫，抽血檢查發現血鈉過高(160 mEq/L，參考值135 ~ 145 mEq/L）。有關高血鈉(hypernatremia）的處理，下列描述何者最適當？

A.估算全身水量(total-body water）:女性是體重的60%，而男性是體重的50%

B.此病患若體重70公斤，計算free water缺乏量(free-water deficit）約5000 c.c.

C.不易感知的水分流失(insensible losses）約5 mL/kg/day

D.血鈉的矯正儘量不超過10 mM/day，以避免腦部水腫(cerebral edema）

108年第一次專 醫學(三)　70*0.6*(160-140)/140= 6L

Polyuria>3L/day

Definition and pathophysiology

- **Polyuria** defined as >3 L UOP per day
- Due to an *osmotic* or a *water diuresis*; almost always due to osmotic diuresis in inpatients

Workup

- Perform a timed urine collection (6 h sufficient) and measure U_{osm}
- 24-h osmole excretion rate = 24-h UOP (actual or estimate) × U_{osm}
  - >1000 mOsm/d → osmotic diuresis
  - <800 mOsm/d → water diuresis

Osmotic diuresis

- Etiologies
  - Glucose (uncontrolled diabetes mellitus)
  - Mannitol
  - Urea: recovering ARF, ↑ protein feeds, hypercatabolism (burns, steroids), GI bleed
  - NaCl administration
- Treatment: address underlying cause, replace free-water deficit and ongoing losses

Water diuresis

- Etiologies: DI (Na_{serum} >140) or 1° polydipsia (Na_{serum} <140)
  - see "Hypernatremia" above for list of causes of central and nephrogenic DI
- Workup of DI: U_{osm} <300 (complete) or 300-600 (partial)
  - water deprivation test: deprive until P_{osm} >295 and U_{osm} <300, then administer vasopressin (5U SC) or dDAVP (10 μg intranasal):
    - U_{osm} ↑ by >50% = central DI
    - U_{osm} unchanged = nephrogenic DI
- Treatment of DI: see "Hypernatremia" above

Euvolemic hypernatremia

- **Diabetes insipidus** (U_{osm} <300-600): ADH defic. (central) or resist. (nephrogenic)
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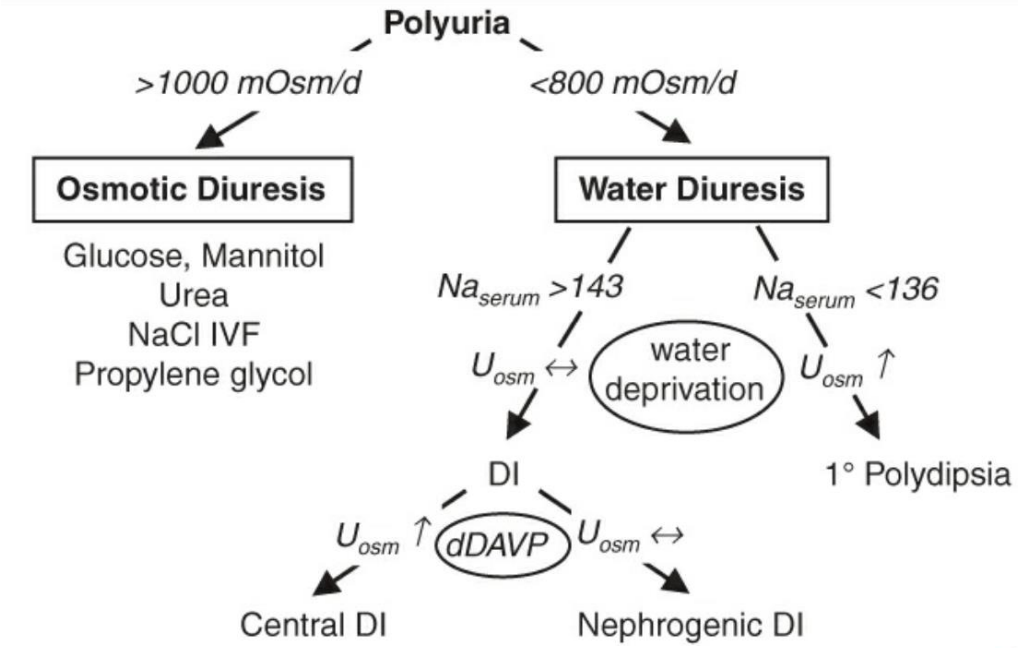


Treatment

- **1° polydipsia:** treat psychiatric illness, check meds, restrict access to free H_2O
- **Osmotic diuresis:** address underlying cause, replace free H_2O deficit (see "Hypernatremia" for formula to calculate) and ongoing losses
- **DI:**

  Central DI: desmopressin (dDAVP, 1st line), low Na/protein diet + HCTZ, chlorpropamide

  Nephrogenic DI: treat underlying cause if possible; Na restriction + HCTZ (mild volume depletion → ↓ delivery of filtrate for free H_2O absorption), consider amiloride for Li-induced DI (*Kid Int* 2009;76:44), indomethacin (*NEJM* 1991;324:850) or trial desmopression

  Pregnancy-induced DI: due to vasopressinase from placenta, ∴ Rx w/ dDAVP

一位 21 歲男性因多尿及夜尿多年至門診就診，血液檢查發現：鈉 158 mmol/L，鉀 3.7 mmol/L，氯124 mmol/L，尿液檢查發現鈉 12 mmol/L，鉀 6 mmol/L，肌酸酐 32 mg/dL， 滲透度 60 mosm/kg H2O， desmopressin （ DDAVP ）測試發現尿液滲透度上升至 500 mosm/kg H2O，下列敘述何者正確？

1. 病患為 primary polydipsia

2. 病患為腎因性尿崩症

3. 病患為中樞性尿崩症

4. 病患為滲透性利尿症（ osmotic diuresis ）

下列何種利尿劑可以用來治療先天性腎性尿崩症（ congenital nephrogenic diabetes insipidus）所引起之多尿症？① thiazides ②loop diuretics ③ acetazolamide ④amiloride

A. ①②

B. ②③

C. ②④

D. ①④

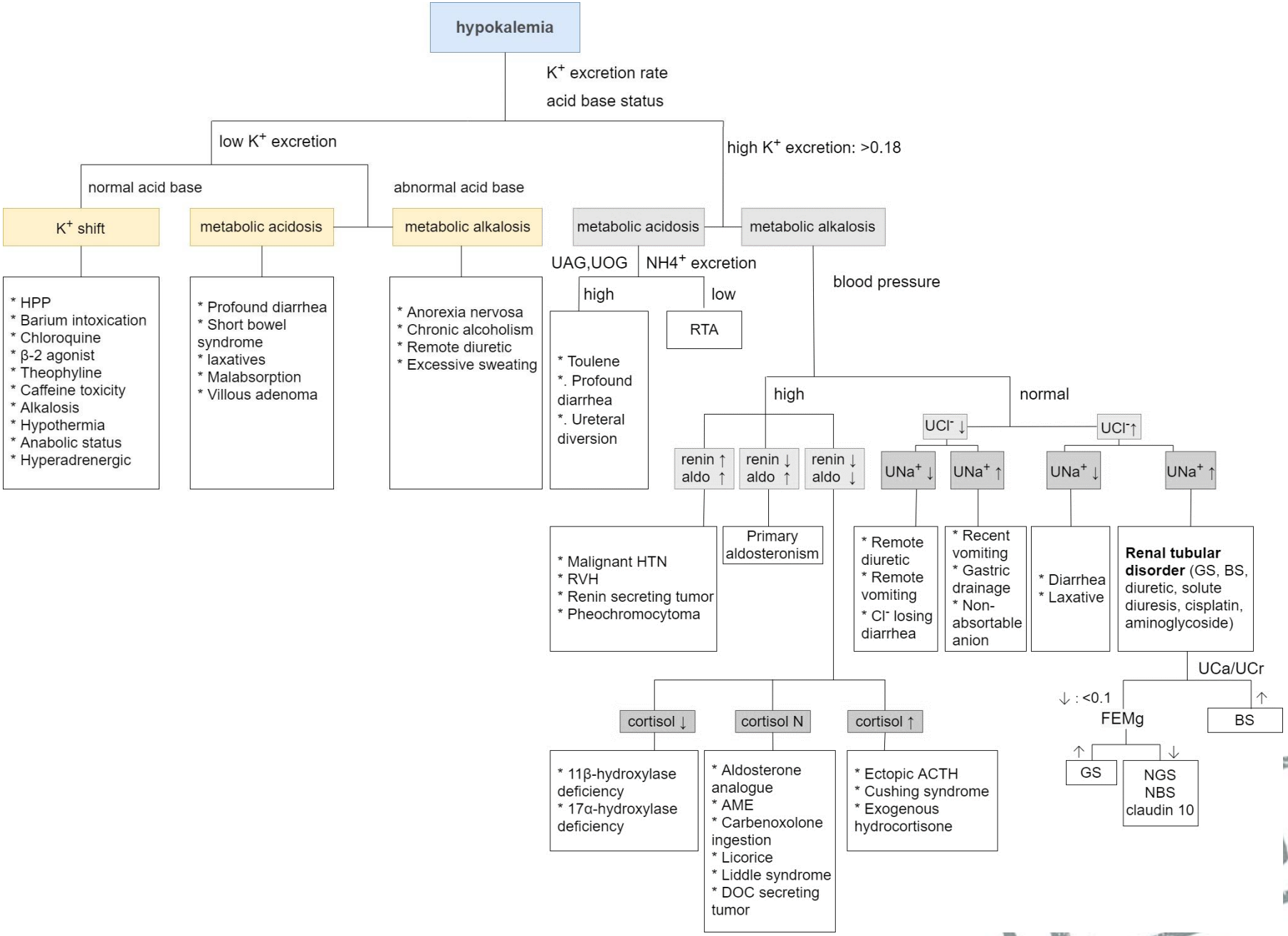
102-25

Potassium Homeostasis

低鉀鑑別診斷、對應治療

高鉀鑑別診斷、對應治療

EKG change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* Anorexia nervosa
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* Remote diuretic
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UCa/UCr
↓ : <0.1
FEMg
BS
GS
NGS
NBS
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* 11β-hydroxylase deficiency
* 17α-hydroxylase deficiency
* Aldosterone analogue
* AME
* Carbenoxolone ingestion
* Licorice
* Liddle syndrome
* DOC secreting tumor
* Ectopic ACTH
* Cushing syndrome
* Exogenous hydrocortisone

表二 K^+ Shifting 疾病與 K^+ wasting 疾病臨床特徵與治療之比較

快速區分 K^+ Shifting 疾病與 K^+ wasting 疾病之要點		
	K^+ Shifting 疾病	**K^+ wasting 疾病**
發生時間	快速	漸進性
低血鉀原因	K^+ 移入細胞內	K^+ 流失
尿 K^+ 排出	低	通常高
Divalent abnormalities	少	常常
酸鹼平衡	正常	異常
急性治療	少劑量 K^+ 非選擇性乙型阻斷劑#	大劑量 K^+ $MgCl_2$$
慢性治療	acetazolamide* loop diuretics[a]	K^+ citrate&
K^+ Shifting 疾病及 K^+ wasting 疾病之治療比較		
	K^+ Shifting 疾病	**K^+ wasting 疾病**
症狀恢復時的血清 K^+濃度	高	低
恢復時間	較短	通常較長
需 K^+ 補充劑量	較少量	較大量
矛盾性低血鉀 (%)	1/4	1/2
反彈性高血鉀 (%)	40~60	~0
矛盾性低血鉀之危險因子	嚴重交感興奮(hyperadrenergism)	低體液容量

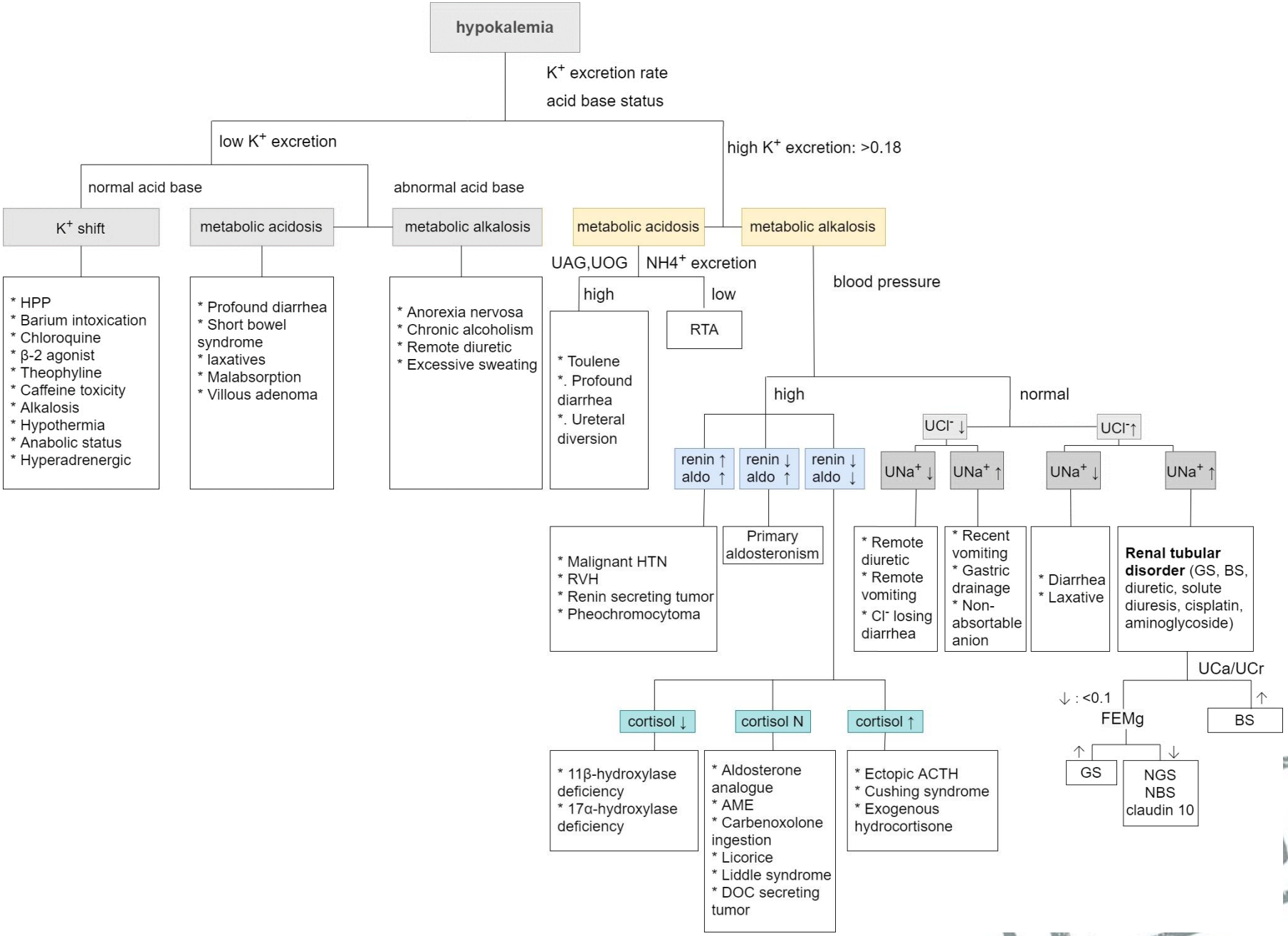
對於明顯高腎上腺皮質功能症(hyperadrenergism)及對鉀治療反應不佳之病人或許有幫助

$ 合併低血鎂的病人

* 對 familial hypoKPP 病人(有 R/X Cav1.1 突變)較有幫助

[a] 仍須隨機分配試驗證實

& 用於合併 metabolic acidosis 之病人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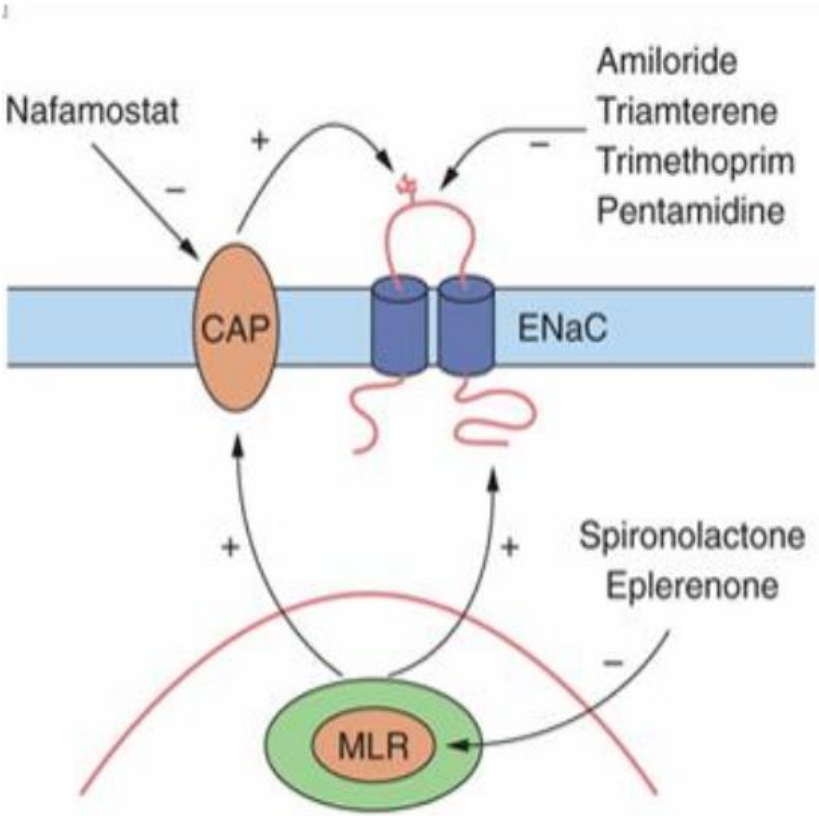
Renin-angiotensin-aldosterone-ENaC system



Interstitial nephritis, HIV

Renin ← NSAIDS, COX-2 inhibitors, beta-blockers

← Renin inhibitors

Angiotensin-I

ACE ← ACE inhibitors

Angiotensin-II ← ARBs

Aldosterone ← Spironolactone, canrenone, eplerenone

Heparin:aldo分泌↓

Renal tubule ← Amiloride, triamterene Trimethroprim, pentamidine Nafamostat

Calcineurin inhibitor: cyclosporin, tacrolimus

Hyper-RAAS

renin ACE

Angiotensinogen→angiotensin I→angeiotensin II→aldosterone

Suspect Hyperaldosteronism

Plasma renin & aldosterone a.m. collection

其他來源高起來的~cortisol

濃度太高，會作用在
aldosterone receptor

↓ renin ↓ aldosterone	↓ renin ↑ aldo (>15) aldo:renin >20	↑ renin ↑ aldosterone aldo:renin ≤10
Non-aldosterone mineralocorticoid excess	salt suppression test	2° Hyperaldosteronism
Cushing's syndrome CAH (some forms) 11β-OHSD deficiency Licorice (chronic ingestion) Liddle's syndrome Exogenous mineralocorticoids	⊕ 1° Hyperaldosteronism	Renovascular disease CHF, cirrhosis, nephrotic Hypovolemia & diuretic use Bartter's syndrome Gitelman's syndrome Renin-secreting tumor

Captopril test確診

AME ← 甘草

slide47

Volume 少

Adrenal CT or MRI

lesion *no lesion*

Adenoma / Carcinoma ← *localize* adrenal vein sampling → *no localization* Hyperplasia or GRA

11βHSD-2

Cortisol ⇄ Cortisone

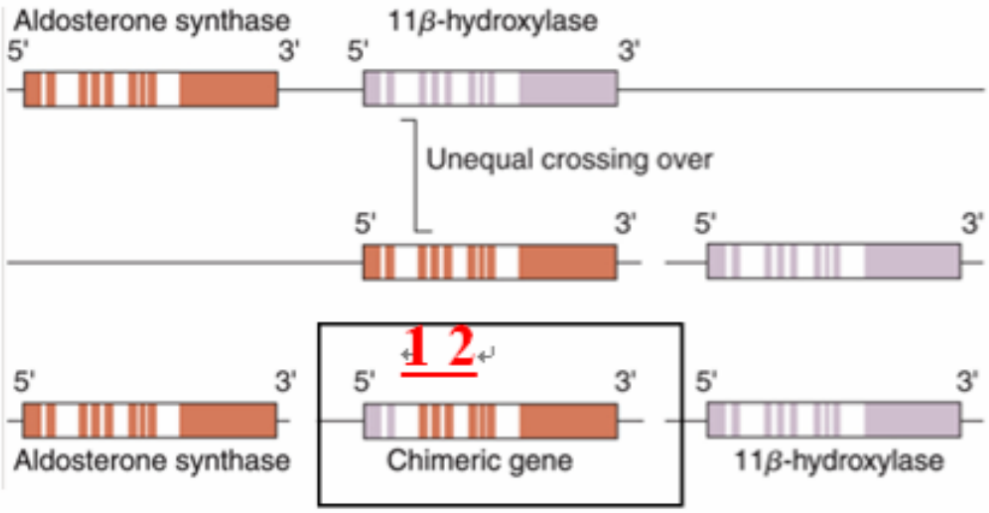
11βHSD-1

會去作用到aldosterone receptor

Glucocoritoicd remediable hyperaldosteronism (GRA)

- **Chimeric gene**：aldosterone synthase被11β hydroxylase控制，一直製造**aldosterone**
- Steroid可以抑制11β hydroxylase，故用steroi

CYP11B2 CYP11B1



Liddle's syndrome

• 99-24 下列關於 Liddle's syndrome 的敘述，何者錯誤？

1. 為自體顯性遺傳， amiloride-sensitive sodium channel 發生 activating mutations
2. 臨床表現有高血壓，低血鉀及代謝性鹼中毒
3. **血清 renin 濃度高， aldosterone 濃度高**
4. 低鈉飲食及 amiloride 可矯正其高血壓及電解質異常

46 下列那一項檢查結果最符合Primary aldosteronism的診斷？

A. 尿液pH值偏酸性

B. 血鉀濃度增加

C. 舒張性高血壓（diastolic hypertension），但無水腫

D. 站立時，腎素（renin）的分泌增加

治療下列引起低血鉀之疾病，何者較容易產生反彈性高血鉀（rebound hyperkalemia）？

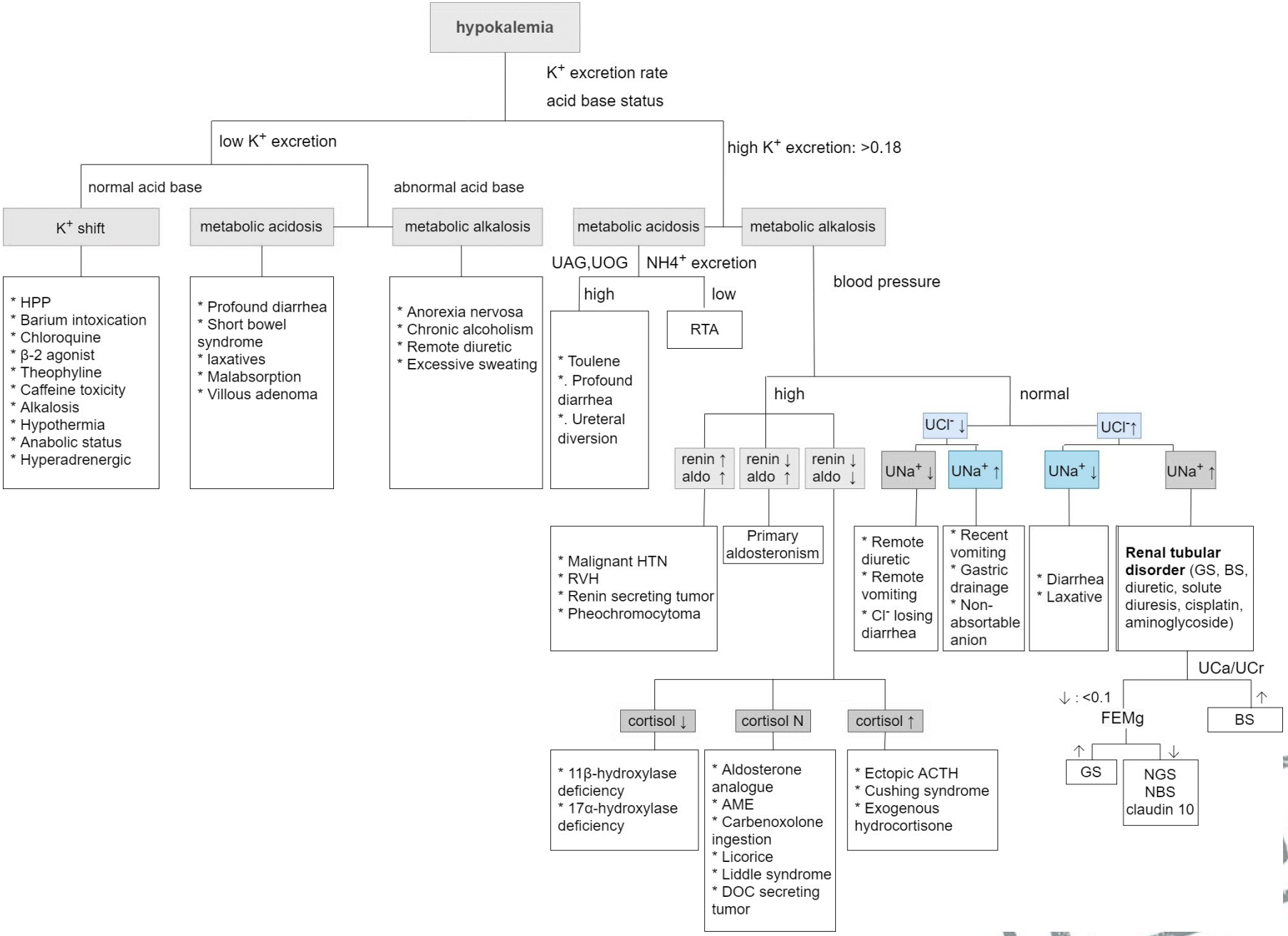
1. 低血鉀週期性無力症（hypokalemic periodic paralysis）

2. Liddle’s 症候群（ Liddle’s syndrome）

3. 糖尿病酮酸血症（ diabetic ketoacidosis）

4. 腹瀉（ diarrhea)

95-20:反彈，表示不是真的loss,為shift而已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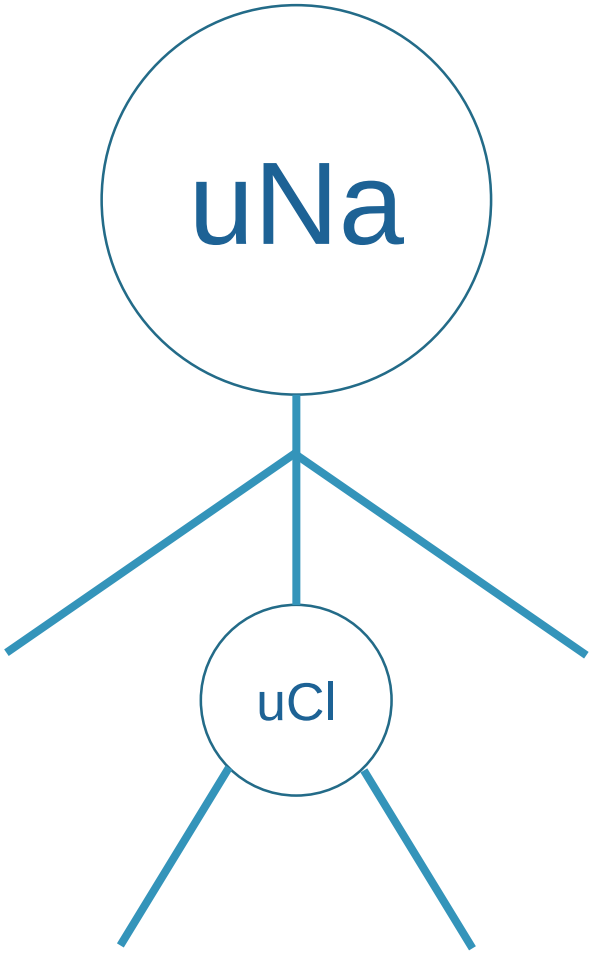
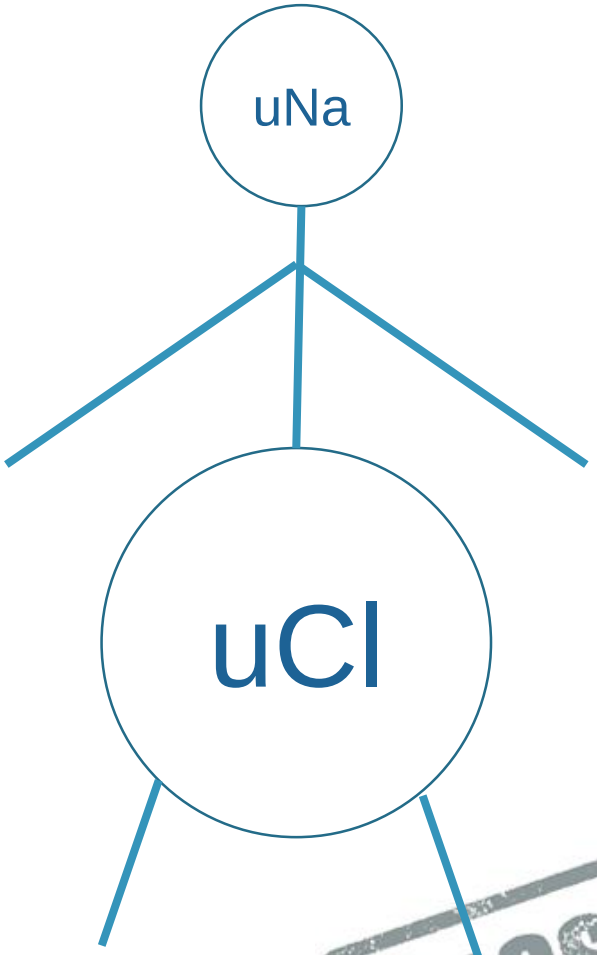
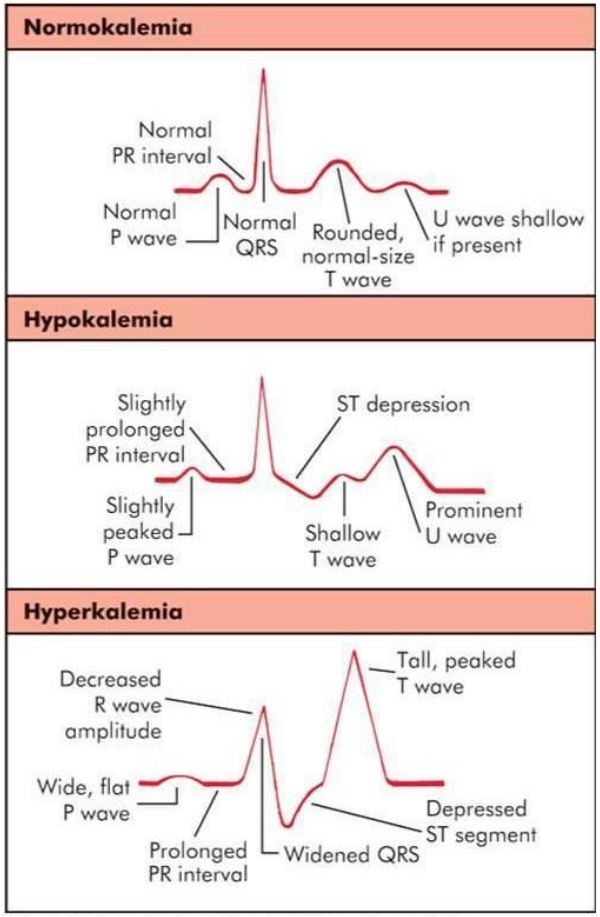
Upper GI:
uNa/ uCl>1.6



lower GI
uNa/ uCl <0.7



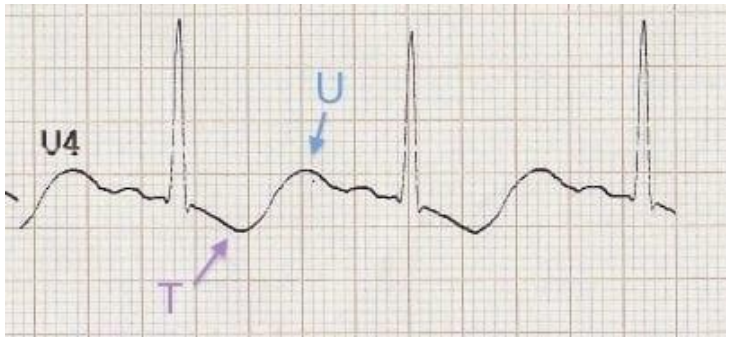
ECG Changes with Potassium Imbalance



ECG EKG Changes in Hypokalemia and Hyperkalemia

Treatment (*JAMA* 2000;160:2429)

- *If true potassium deficit:* **potassium repletion** (↓ 1 mEq/L ≈ 200 mEq total body loss)
  Dosage: 40 mEq PO q4h, 10 mEq/h (IV), 20 mEq/h (central line), 40 mEq in 1L IVF
- Replete K^+ to >3 or >4 mEq/L if high-risk (HTN, CHF, arrhythmias, MI, digoxin, cirrhosis)
- Beware of excessive potassium repletion if transcellular shift cause of hypokalemia
- Treat underlying cause (if ↓ vol: avoid dextrose as ↑ insulin → intracellular potassium shifts)
- Consider Rx that ↓ K loss: ACEI/ARB, K^+-sparing diuretics, βB
- Replete Mg if <2 mEq/L: IV Mg-SO_4 1–2 g q2h (oral Mg-oxide poorly tolerated b/c diarrhea)
  Causes of low Mg: GI loss (diarrhea, bypass, pancreatitis, malnutrition, PPI); renal loss (diuretics, nephrotoxic drugs, EtOH, ↑ Ca, 1° wasting syndromes, volume expansion)



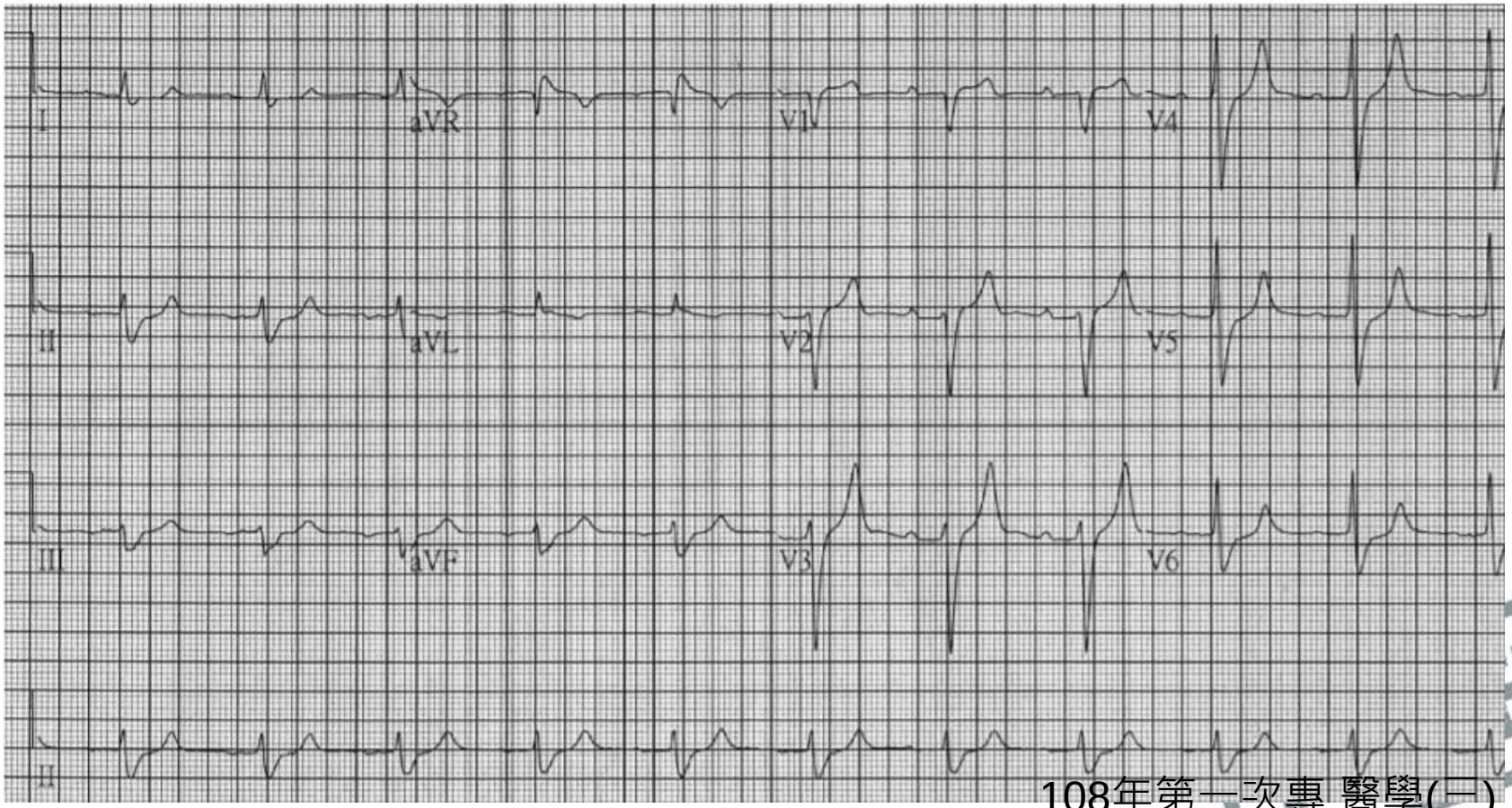
Therapy of Hyperkalemia	
Stabilization of the Membrane	
IV calcium	Works within min; action lasts up to 1 hr Ca gluconate 3 amp (3 g, 10% 30 mL, 14 mEq Ca) via peripheral line or $CaCl_2$ 1 amp (1 g, 10% 10 mL, 13.6 mEq Ca) via central line over 2–3 min Avoid in digitalis toxicity
Drive Extracellular Potassium into the Cells	
Insulin + glucose	Bolus 5–10 units of insulin R + 50 mL of 50% dextrose Effect starts in 10–20 min, peaks at 30–60 min, lasts for 4–6 hr K drops 0.5–1.2 mEq/L
Albuterol	Lowers the serum potassium concentration by 0.5–1.5 mEq/L 10–20 mg nebulizer; peak effect in 90 min Used as adjuvant to insulin + glucose
Sodium bicarbonate	Beneficial mainly in metabolic acidosis in acute ↑ K; and in chronic ↑ K in CKD 150 mEq in 1 L of 5% dextrose in water
Removal of Potassium from the Body	
Diuretics	Can be used for both acute and chronic hyperkalemia Dosage depends on the renal function
GI cations exchangers	Patiromer (*NEJM* 2015;372:211) 8.4–25.2 g qd; give 3–6 hr after or before other po meds Exchanges K for Ca in the colon s/e: constipation, ↓ Mg Sodium zirconium cyclosilicate (*NEJM* 2015;372:222) Sodium polystyrene sulfonate (SPS) 15–60 g single dose po (+/− sorbitol); 50 g enema (without sorbitol) Avoid in postop, ileus or bowel obstruction (s/e: intestinal necrosis)
Dialysis	HD is more efficacious in K removal than PD CRRT can be used subsequently in patients with ongoing K release Dialysate K <2 a/w sudden cardiac arrest (*KI* 2011;79:218) Rebound ↑ K: K shift to serum after HD. More pronounced after albuterol, insulin and high Na+ dialysate (*JASN* 2000;11:2337), post-HD ↓ K should not be corrected unless clinically indicated

一位59歲接受腹膜透析腎病患者，血壓為125/80 mmHg，心跳為76次/min，12導程心電圖發現有peaked T waves，血鉀值為7.0 mmol/L，無溶血。下列何種處置最不優先？

6. 一位82歲男性，有高血壓與慢性腎臟疾病，主訴最近疲倦無力，血壓152/90 mmHg，呼吸15次/分，心跳65 次/分，體溫36.5℃，心電圖顯示如下圖，其最可能為何種電解質異常？

A.高血鈣症(hypercalcemia ） B.低血鈣症(hypocalcemia)

C.高血鉀症(hyperkalemia) D.低血鉀症(hypokalemia ）



血鉀異常: 人名 syndrome大彙整

低血鉀 + HTN

Liddle syndrome: ENaC ↑

Conn’s syndrome: primary hyperaldo

低血鉀 + normal BP

Bartter / Gitelman: two diuretics

高血鉀 + HTN

Gordon syndrome (↔ Gitelman)

Renal Failure/ other electrolyte

AKI鑑別診斷、對應治療

CKD 分期/ ESRD及治療

Sodium and Water Homeostasis
Potassium Homeostasis
Renal Failure/ other electrolyte
Glomerular Disease
Urinalysis
Nephrolithiasis

Renal Failure/ other electrolyte

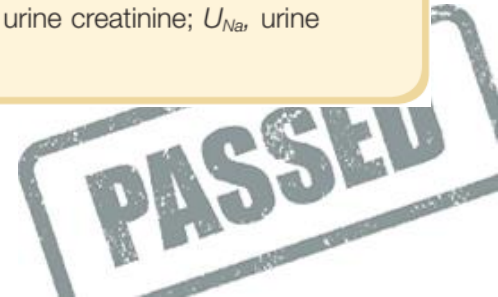
AKI Staging (KDIGO AKI 2012)		
Stage	**Creatinine**	**Urine Output (UOP)**
1	↑ 0.3 in 48 hr or ↑ to 1.5–1.99x baseline w/i 7 d	<0.5 cc/kg/hr for >6–12 hr
2	↑ to 2.0–2.99x baseline w/i 7 d	<0.5 cc/kg/hr for ≥12 hr
3	↑ to ≥3x baseline w/i 7 d; OR increase in Cr to ≥4.0; OR need for RRT	<0.3 cc/kg/hr for ≥24 hr or anuria for ≥12 hr

Table 29.5 Urine Indices Used in the Differential Diagnosis of Prerenal Acute Kidney Injury and Acute Tubular Necrosis

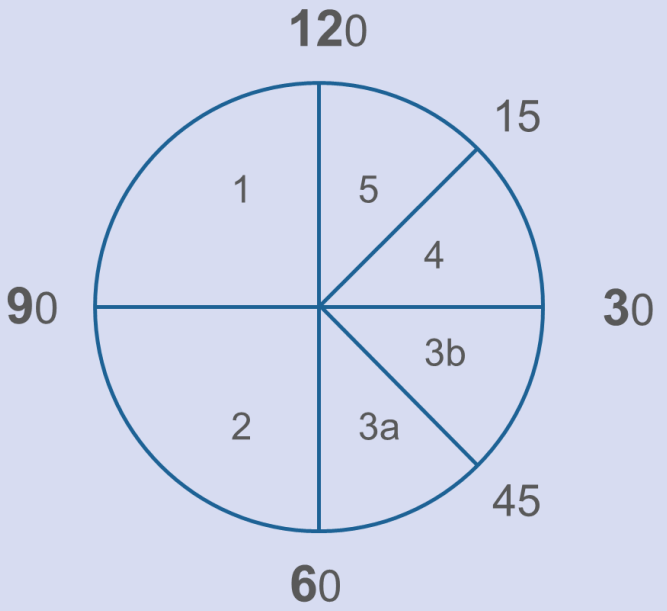
Diagnostic Index	Prerenal AKI	ATN
Fractional excretion of sodium (%)	<1[a]	>2[a]
Urine sodium concentration (mmol/L)	<20	>40
Urine creatinine-to-plasma creatinine ratio	>40	<20
Urine urea nitrogen-to-plasma urea nitrogen ratio	>8	<3
Urine specific gravity	>1.018	~1.010
Urine osmolality (mOsm/kg H_2O)	>500	~300
Plasma BUN-to-creatinine ratio	>20	<10-15
Renal failure index, $U_{Na}/(U_{Cr}/P_{Cr})$	<1	>1
Urine sediment	Hyaline casts	Muddy brown granular casts

[a]FE_{Na} may be >1% in prerenal AKI associated with diuretic use and/or the setting of bicarbonaturia or chronic kidney disease; FE_{Na} often <1% in acute tubular necrosis caused by radiocontrast or rhabdomyolysis.

AKI, Acute kidney injury; *ATN*, acute tubular necrosis; *BUN,* blood urea nitrogen; P_{Cr}, plasma creatinine; U_{Cr}, urine creatinine; U_{Na}, urine sodium.



KDIGO慢性腎臟病分期



GFR clock

持續白蛋白尿的分期		
A1 正常到 輕度升高	A2 中度升高	A3 重度升高
<30 mg/g	30-300 mg/g	>300 mg/g
<3 mg/mmol	3-30 mg/mmol	>30 mg/mmol

綠色：低風險；黃色（中度風險）；橙色（高風險）； 紅色（非常高的風險）

資料來源:恩主公醫院腎臟科張嘉峯醫師

22. 72歲男性為慢性腎病病人，血中肌酸酐(creatinine）6.3 mg/dL，下列何種酸鹼電解質狀態最少發生在此病人？

A.鈉(Na^+）148 mEq/L

B.鉀(K^+）5.6 mEq/L

C.磷(PO_4^{3-}）5.5 mg/dL

D.鈣(Ca^{2+}）8.0 mg/dL

108年第一次專 醫學(三)

65歲女性病人因長期糖尿病腎病變，接受規則血液透析治療已5年，透析前血中磷(PO_4^{3-}) 6.8 mg/dL、鈣 (Ca^{++}) 10.8 mg/dL、副甲狀腺賀爾蒙(PTH) 88 pg/mL。對此病人，下列何者為最適當治療？

A.使用磷結合劑(phosphate binder)

B.使用維他命D3(vitamin D3)

C.使用擬鈣劑(calcimimetic)

D.使用鈣濃度3.0 mEq/L透析液

108年第一次專 醫學(三)