110 年度國考複習

電解質/酸鹼異常

必拿考題!!!

- **酸鹼診斷判斷**
- 鉀離子**(低-診斷；高-診斷、處置)**
- 鈉離子與水分恆定

聯想、關鍵字(疾病)!!!

	91	92	93	94	95	96	97	98	99	100	101	102	103	104	105	106	Total
第一篇　腎臟內科	25	33	33	38	14	9	21	16	16	18	15	18	14	12	16	12	310
甲、酸鹼平衡	1	3	4	5	2			1	2	2	1	3	1	1	2	1	29
乙、離子平衡	7	8	8	9	3	3	6	1	2	2	5	9	6	5	5	6	85
丙、急性腎損傷	4		2	7	2		5	1	4	6	1	1	2	1	2		38
丁、腎臟疾病各論	7	12	9	8	5	1	3	5	4	6	2	3	3	1	4	3	76
戊、慢性腎臟病	4	8	4	6		2	1		2	2	5	1		2	1		38
己、透析治療	2	1	3	2	2	3	1	4	2				1	2	1	1	25
庚、腎臟移植		1	2	1			1	1								1	7
辛、多囊性腎病			1				1	2			1		1				6
其他未分類題目							3	1				1			1		6

Acid-Base Disturbances

Gas 判斷:

Acid-**B**ase ⟹ **C**ompensate

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

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

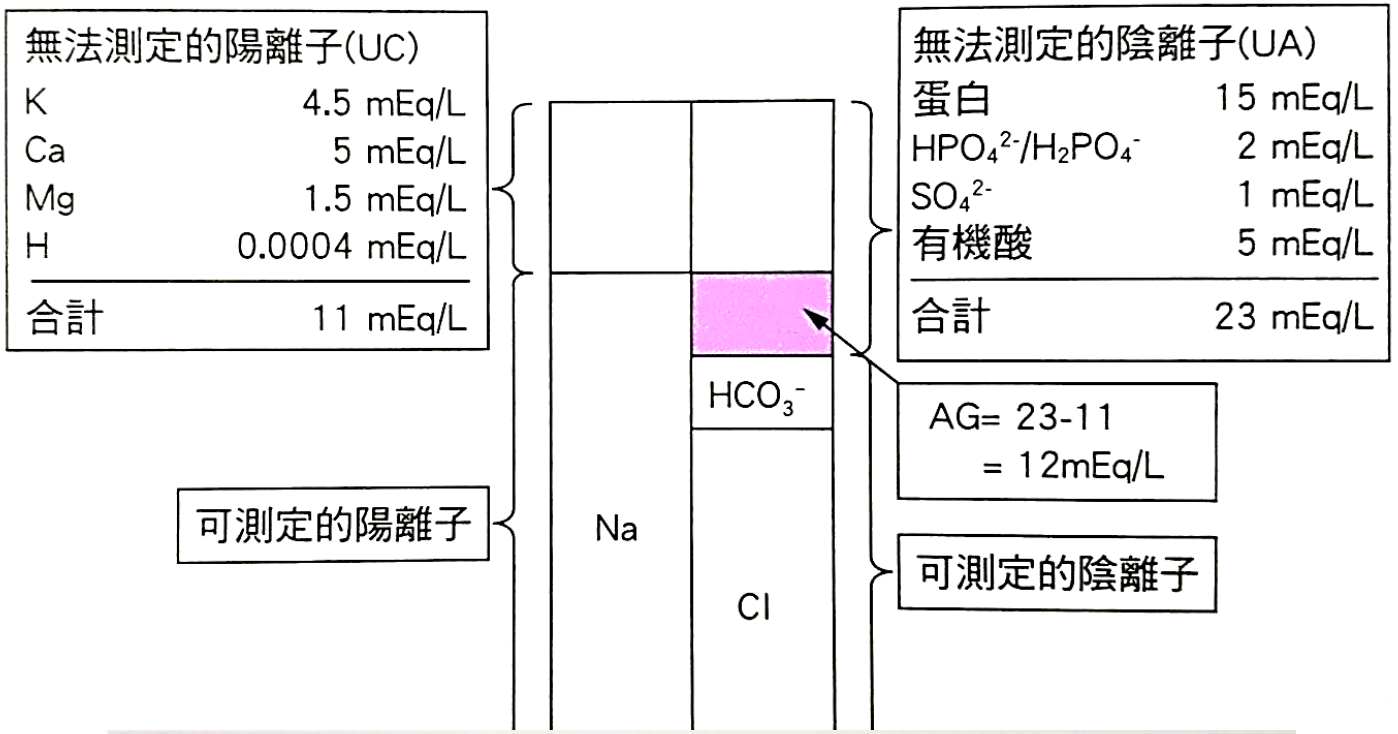
身體代償會使HCO_3^-, CO_2 同方向

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

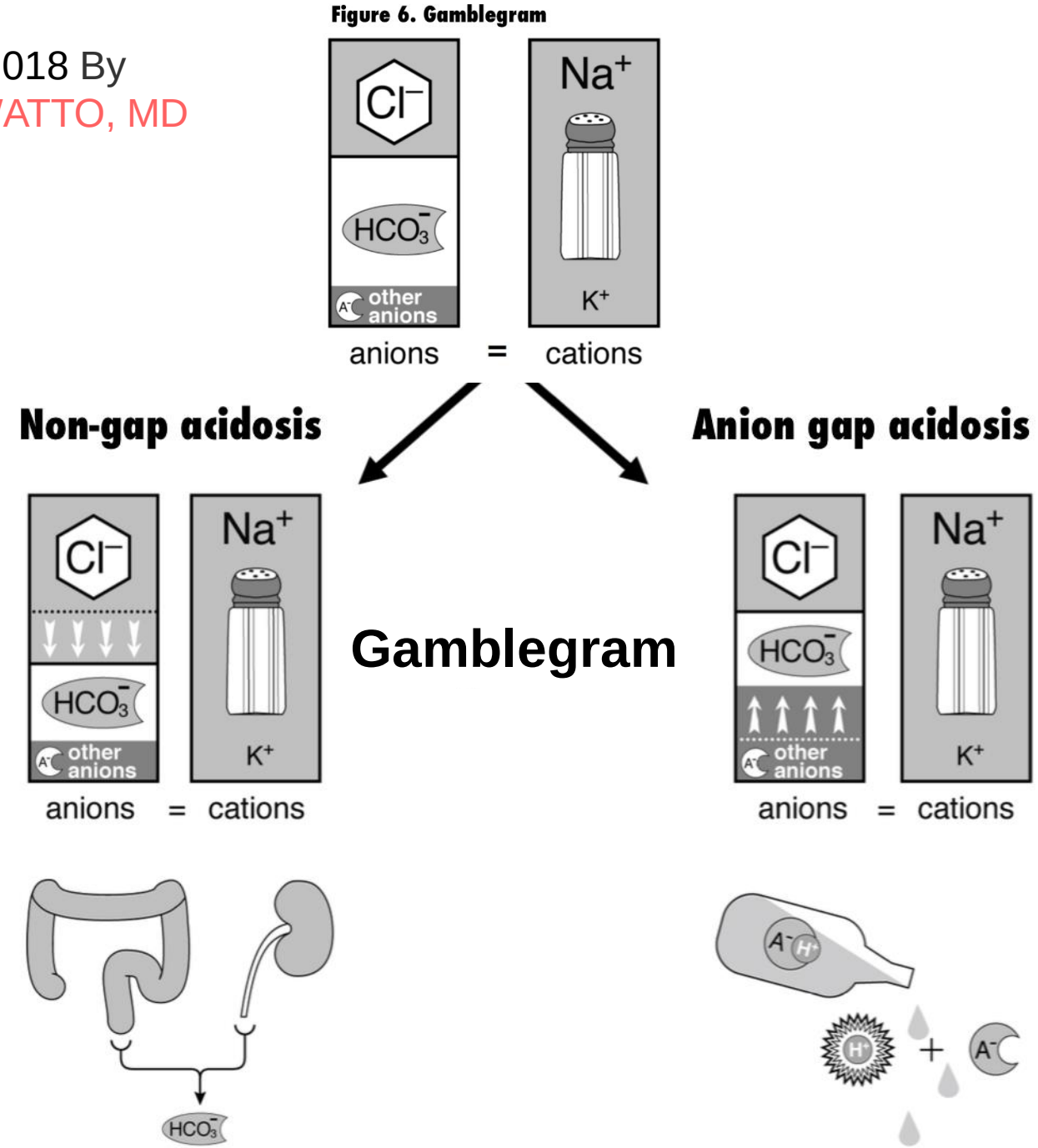


可測定的陽離子 [Na^+] ＋無法測定的陽離子 [UC]
＝可測定的陰離子 [Cl^- ＋ HCO_3^-] ＋無法測定的陰離子 [UA]
※UC: unmeasured cation, UA: unmeasured anion

- 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

MARCH 25, 2018 By
MATTHEW WATTO, MD



Metabolic acidosis

AG →Δ/Δ → KILR or OG

NAG →GI, (NEP → UAG)

High anion gap

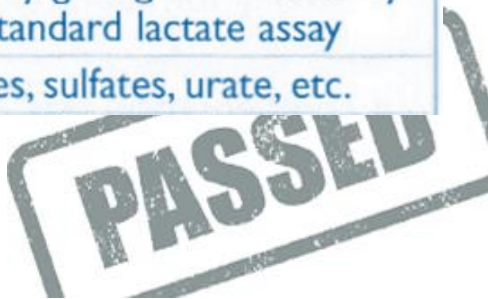
K

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)

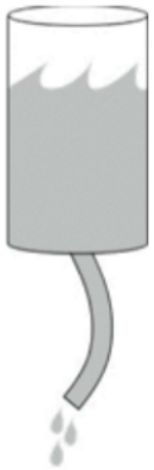
104-19

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?

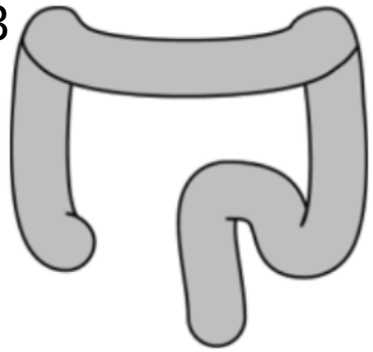
- A. lactic acidosis
- B. ketoacidosis
- **C. diarrhea**
- D. salicylate intoxication

Three buckets of Non gap metabolic acidosis

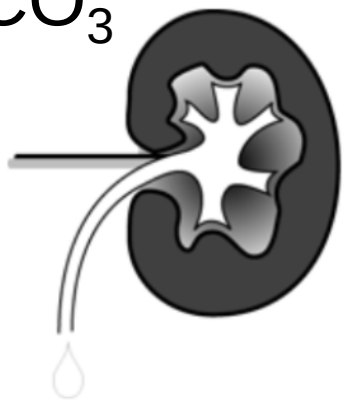
給 Cl



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

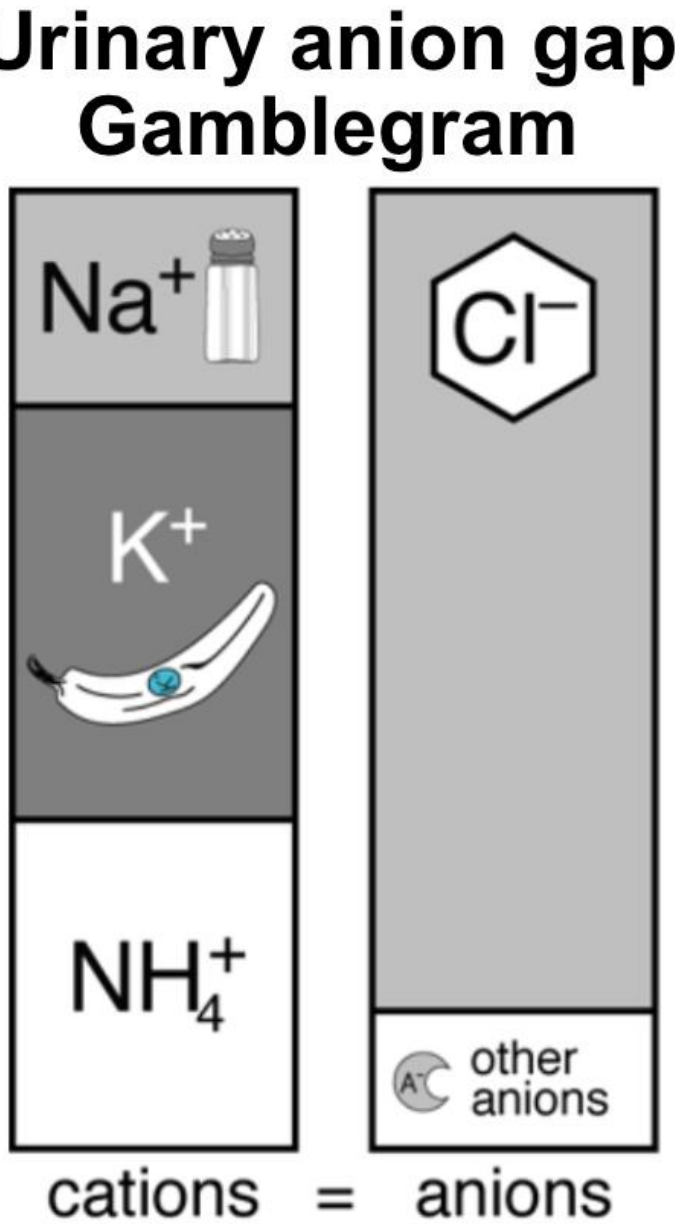
MARCH 25, 2018 By MATTHEW WATTO, MD

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+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$NH4^+$**應該是上升的
- 而$NH4^+$屬於未測得cation，故**UAG<0**



metabolic acidosis

UAG= uNa+uK-uCl <0

MARCH 25, 2018 By MATTHEW WATTO, MD

RTA (UAG>0)

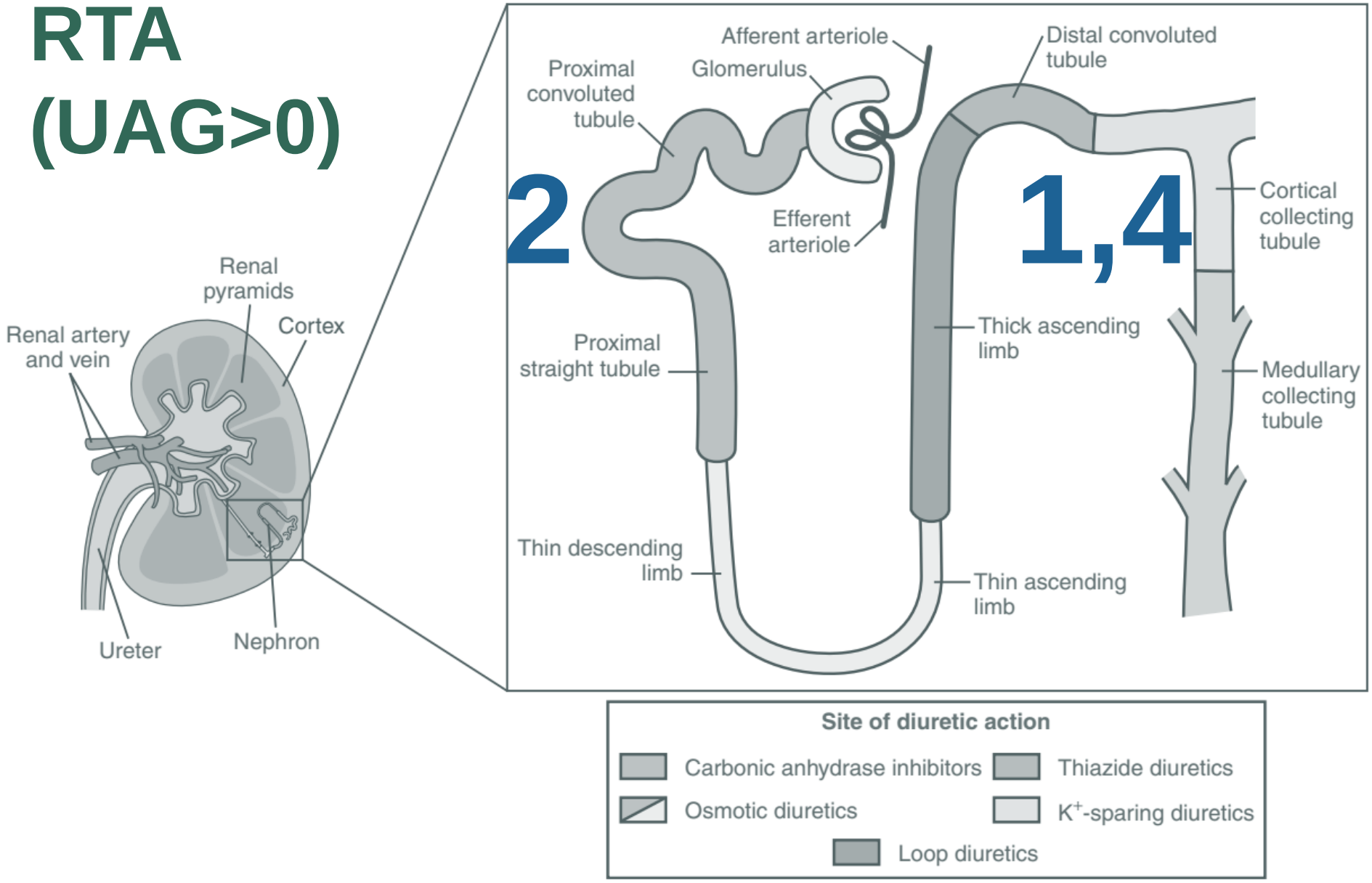


FIGURE 21–1 The nephron, depicting its location, segments, and the sites of action of different classes of diuretics.

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

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**

AG

KILR

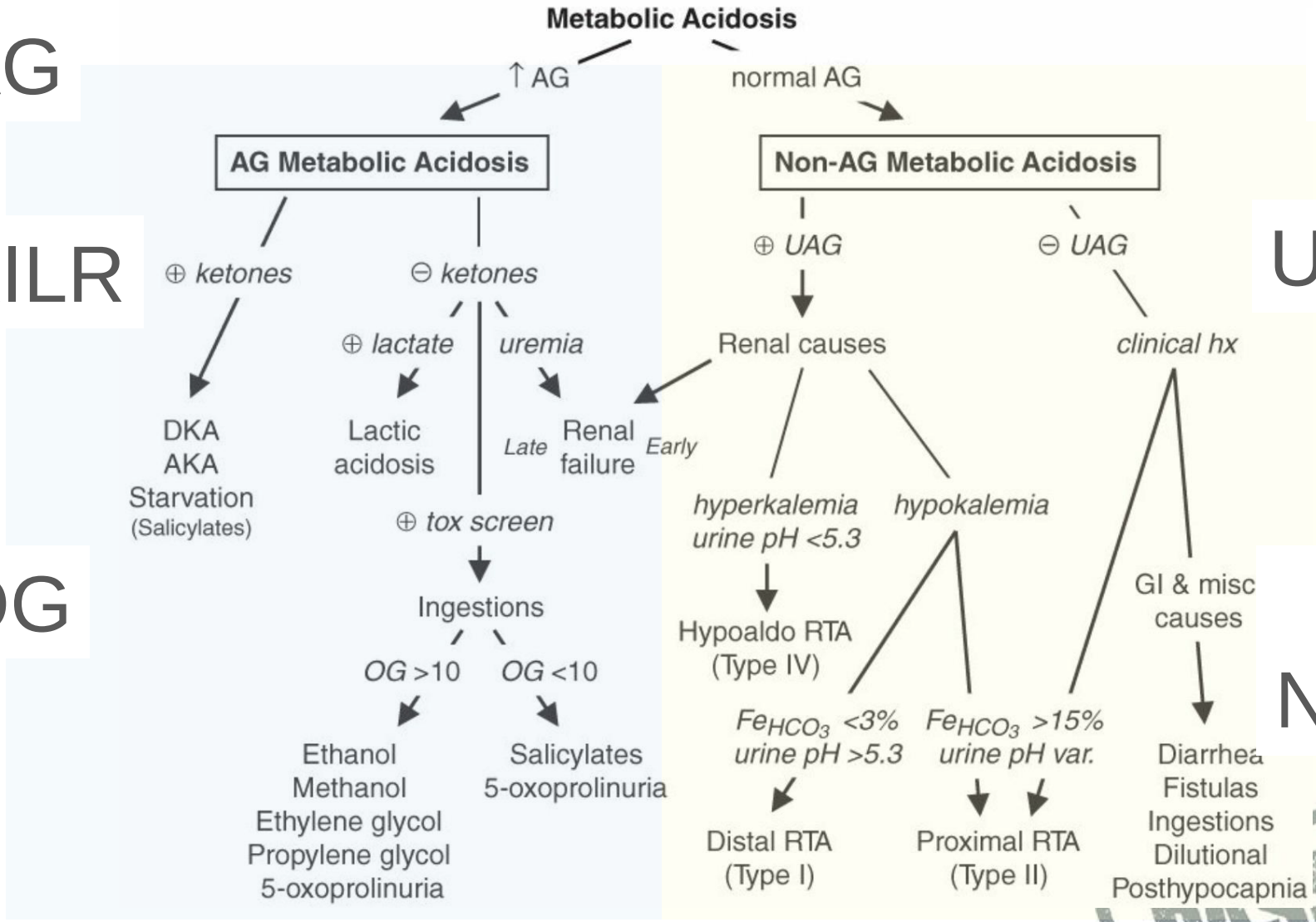
OG

AG

UAG

GI

NEP



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

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

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)

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

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

Metabolic alkalosis

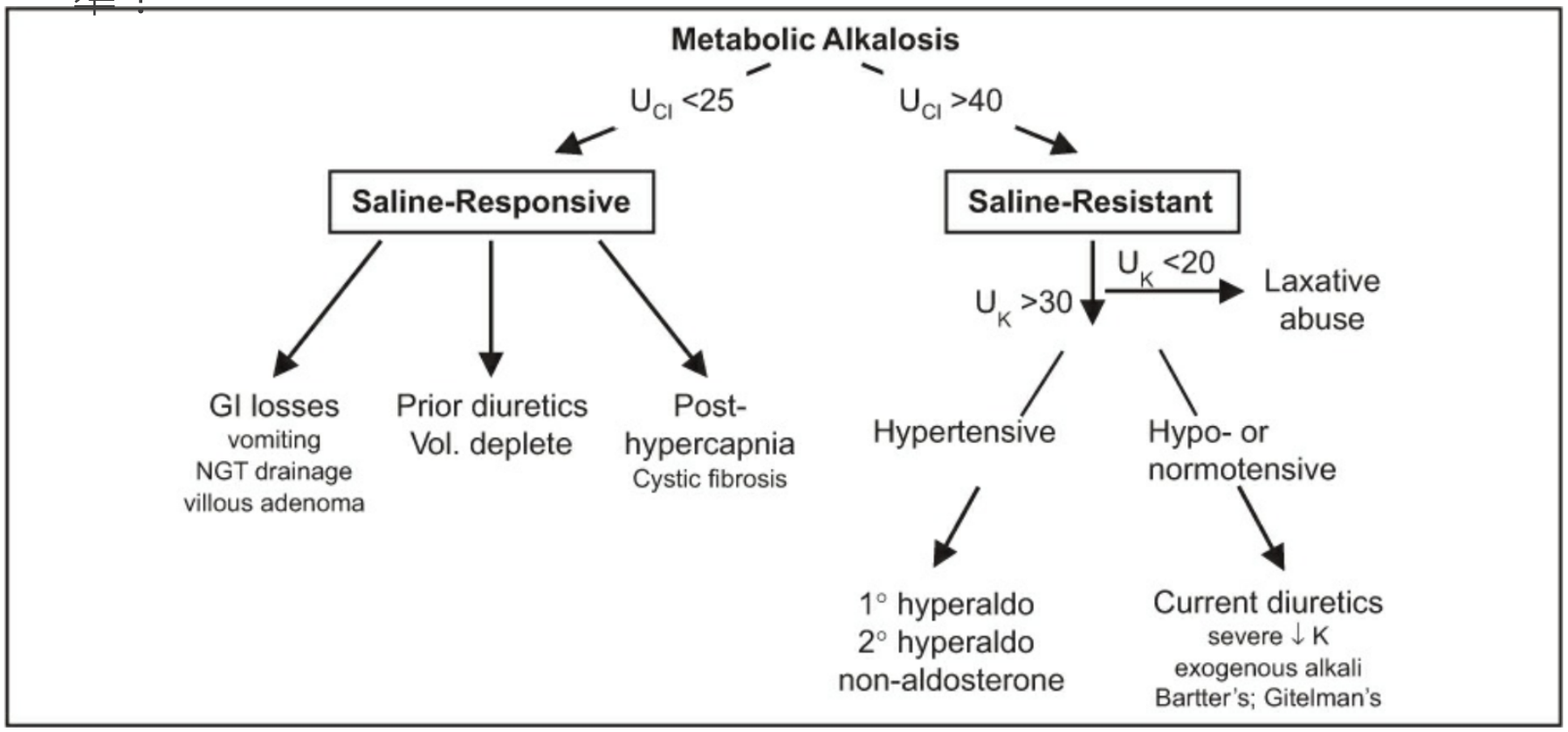
20 or 25

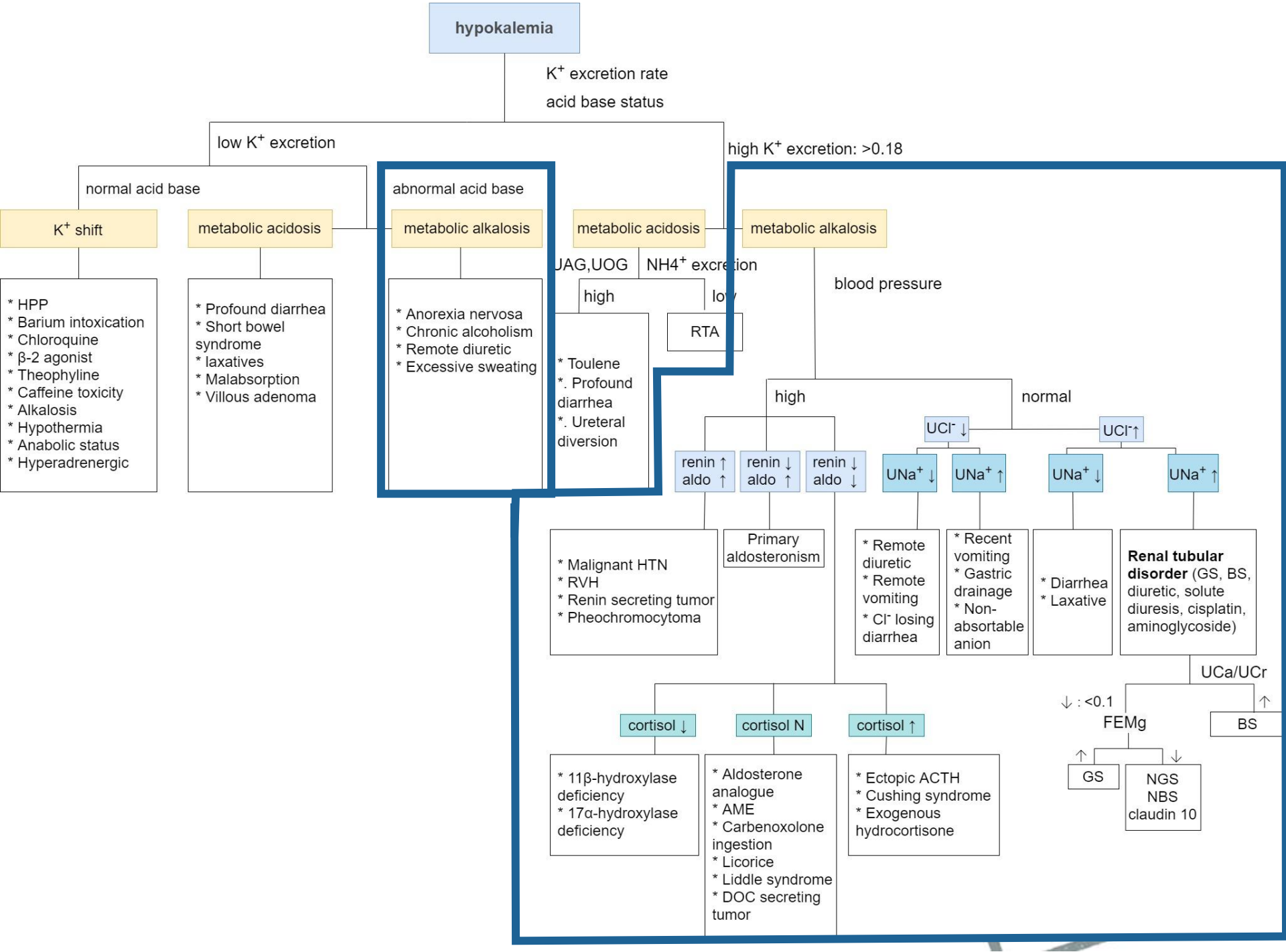
Etiologies of Metabolic Alkalosis	
Saline responsive UCl < 25 **Remote diuretic**	**GI loss of H^+**: emesis, NGT suction, villous adenoma, chloridorrhea **Renal loss**: loop/thiazide, ↓ Cl intake, milk-alkali, Pendred syndrome Posthypercapnia, sweat losses in cystic fibrosis
Saline resistant UCl >40 **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)

previous

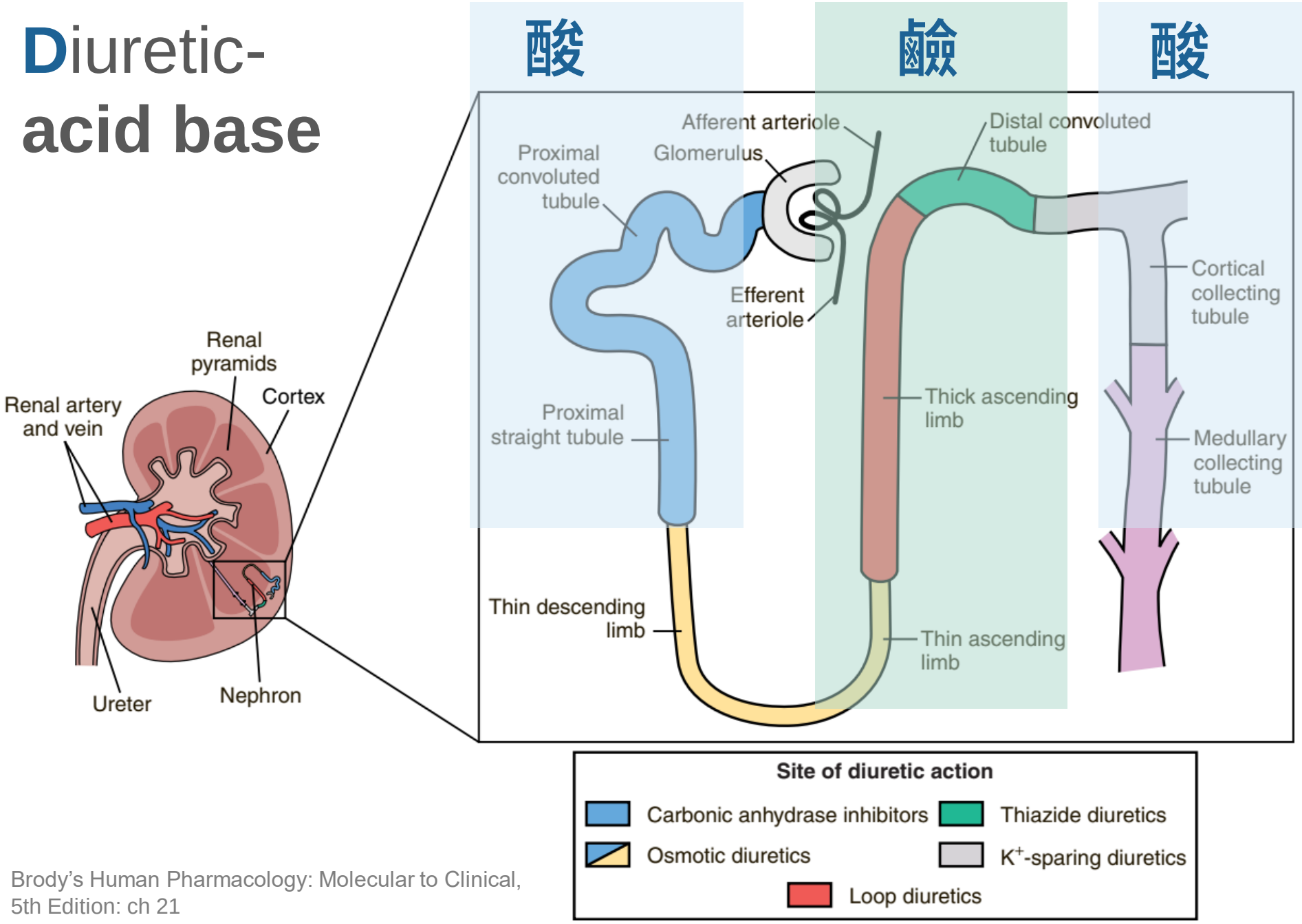
Metabolic alkalosis

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

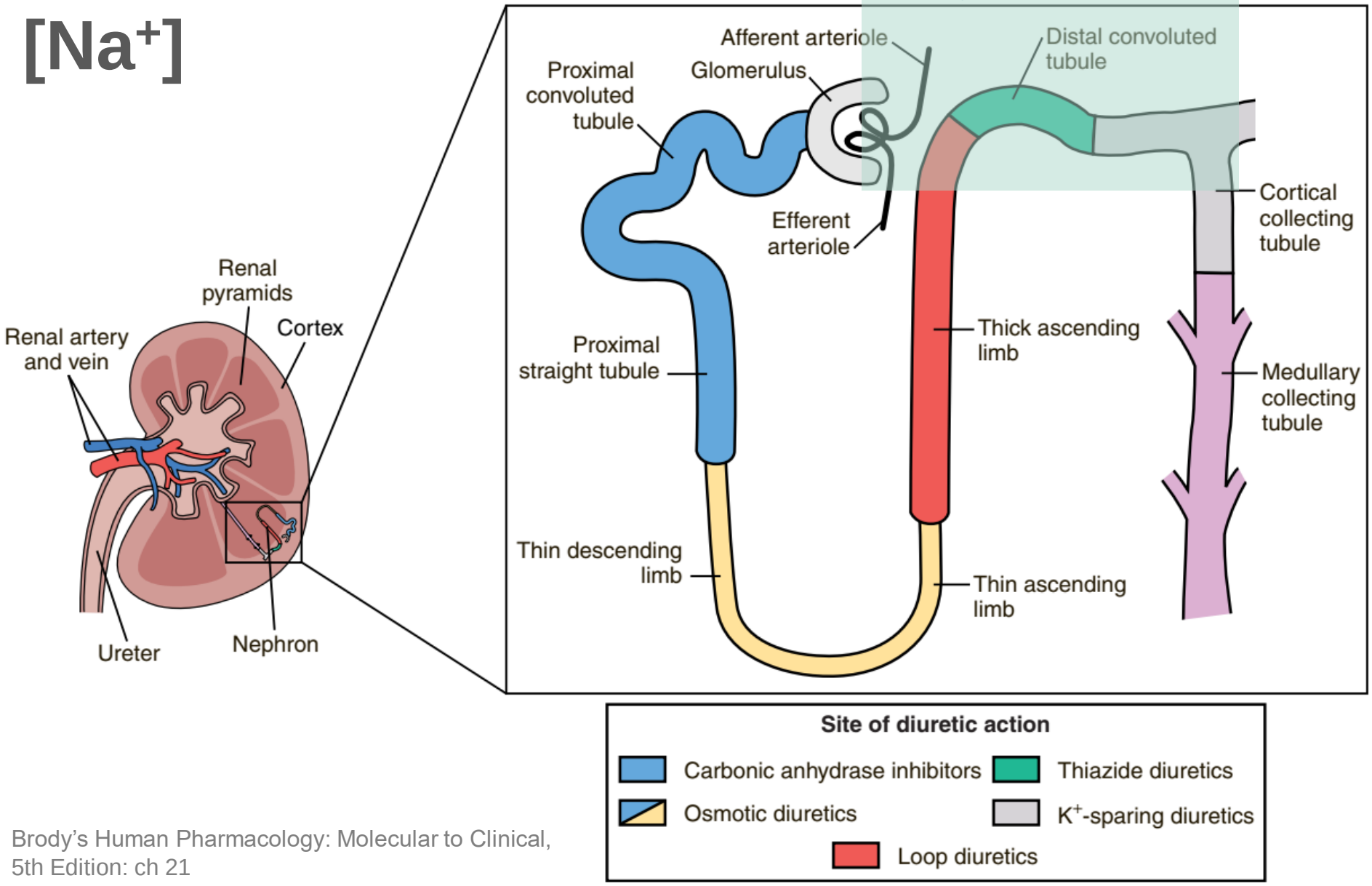


Brody's Human Pharmacology: Molecular to Clinical, 5th Edition: ch 21

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

Diuretic-
[Na^+]

↓血鈉



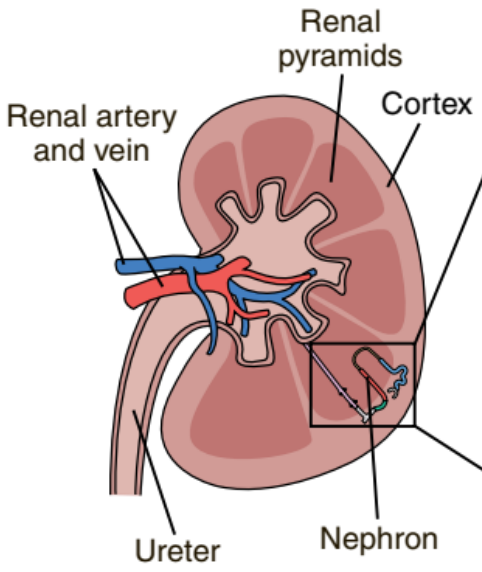
Brody's Human Pharmacology: Molecular to Clinical, 5th Edition: ch 21

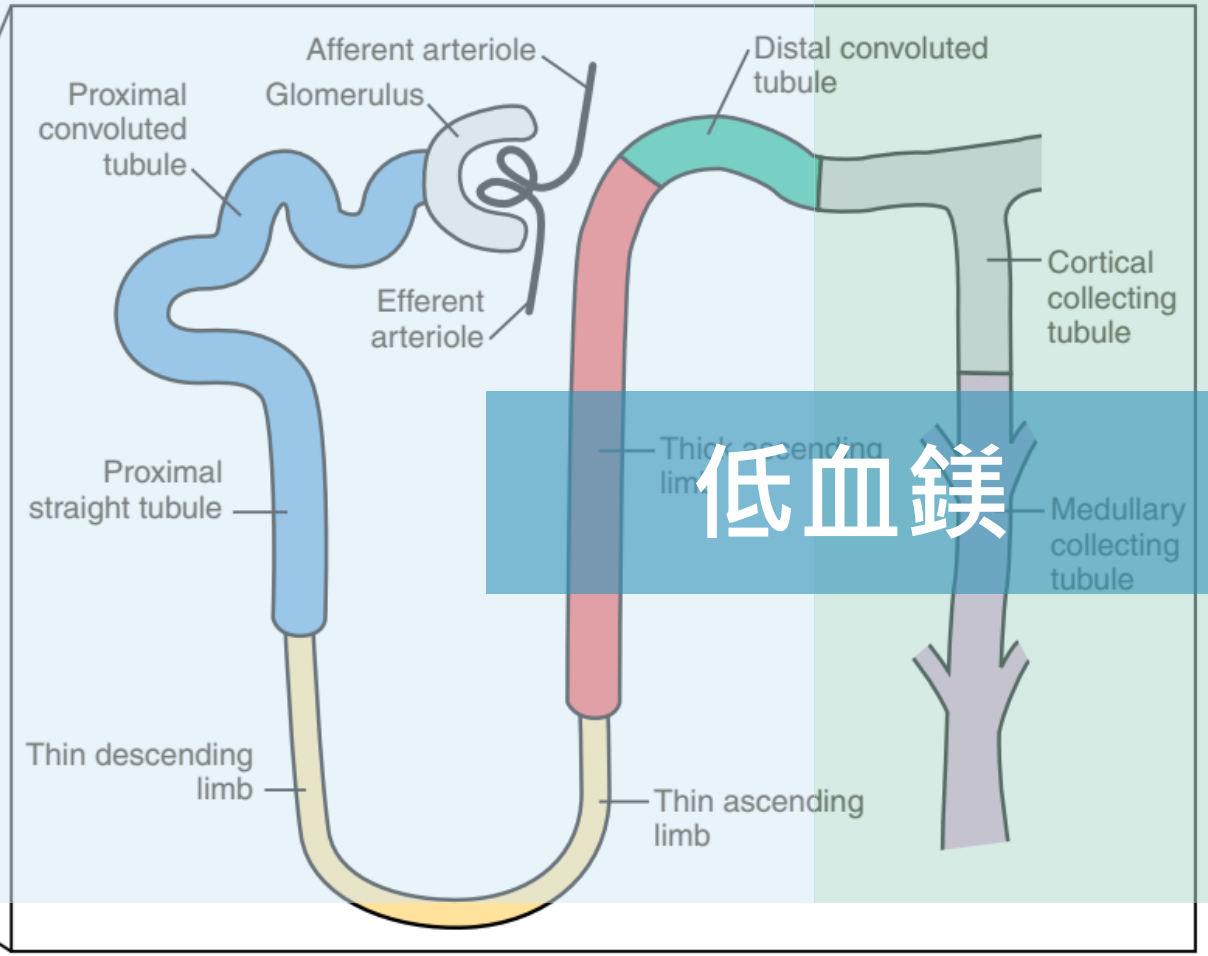
FIGURE 21–1 The nephron, depicting its location, segments, and the sites of action of different classes of diuretics.

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

低鉀

保鉀



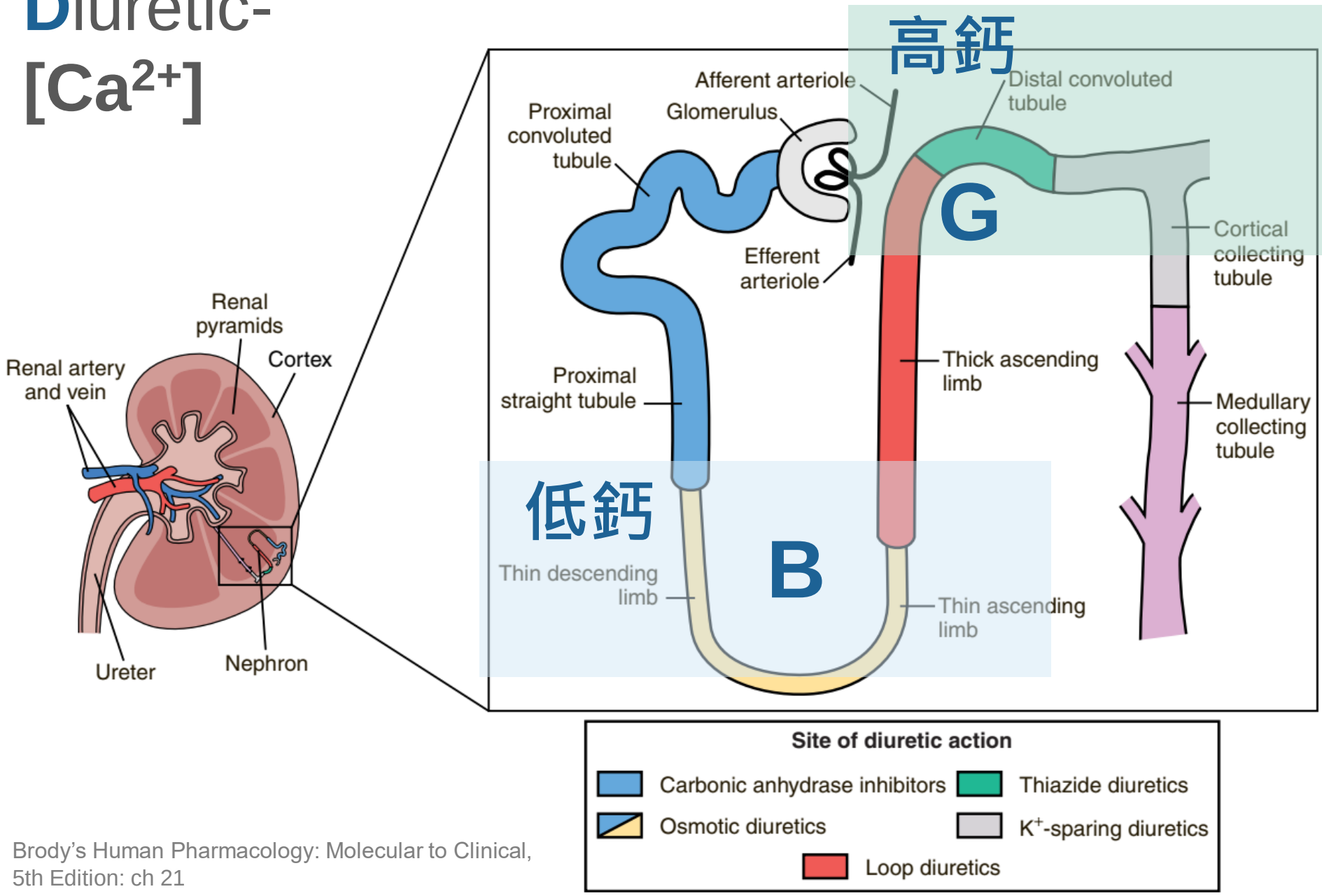


低血鎂

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.

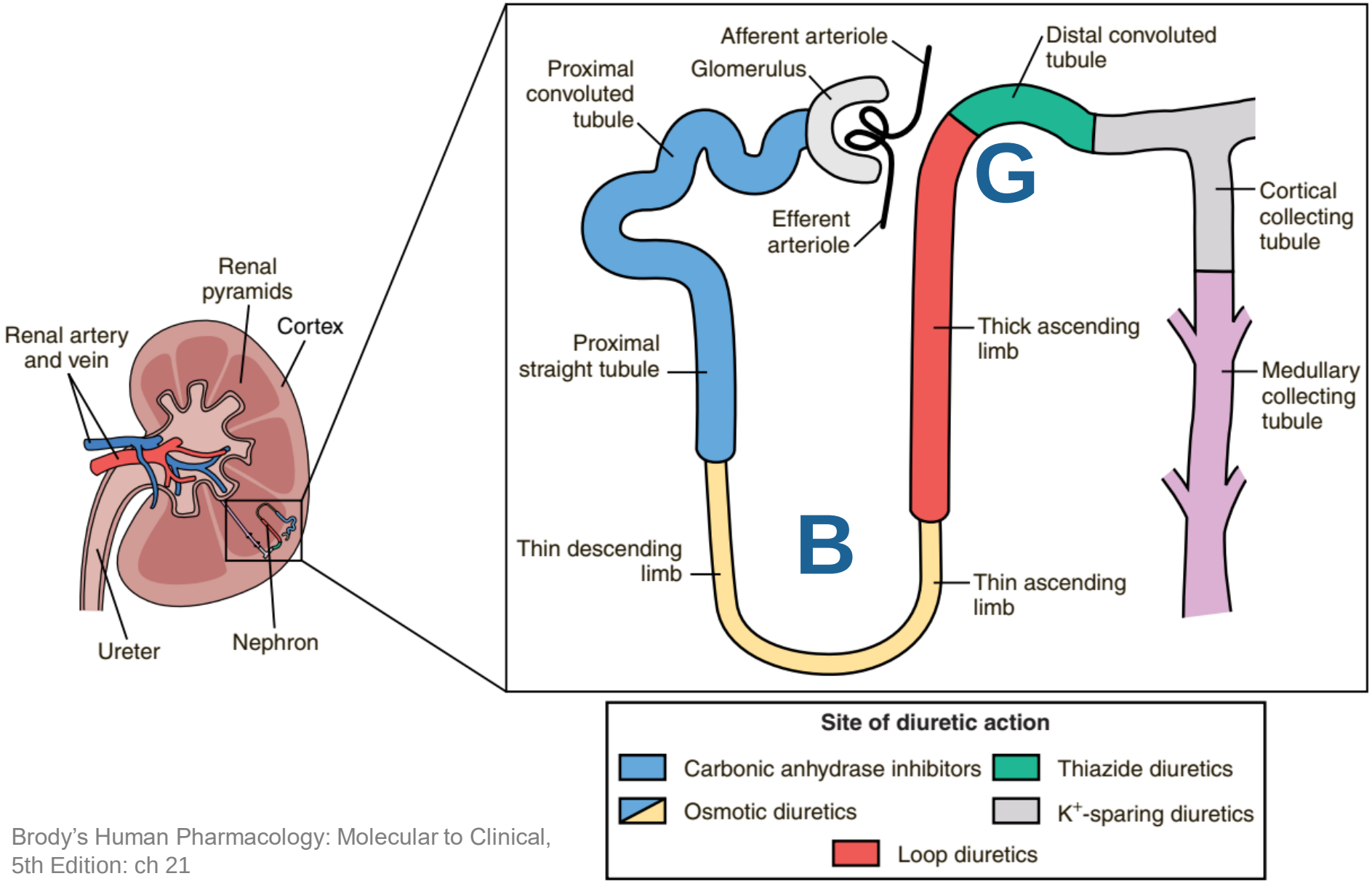
Diuretic-
[Ca^{2+}]



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”; $PaCO_2 = VCO_2/VE(1-VD/VT)$; $VE=RR \times VT$)

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）

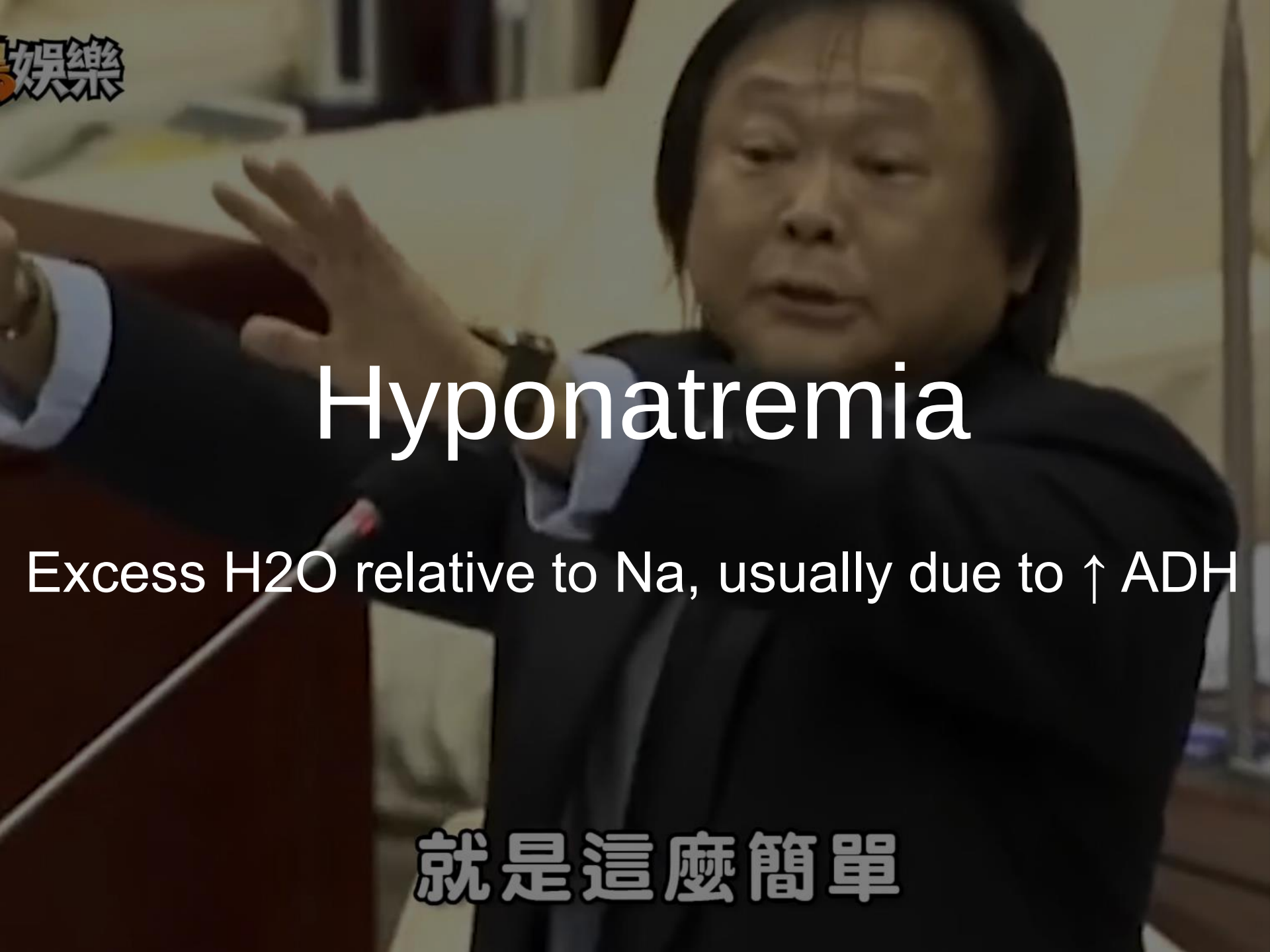
108年第二次專 醫學(三)

Sodium and Water Homeostasis

低鈉鑑別診斷、對應治療

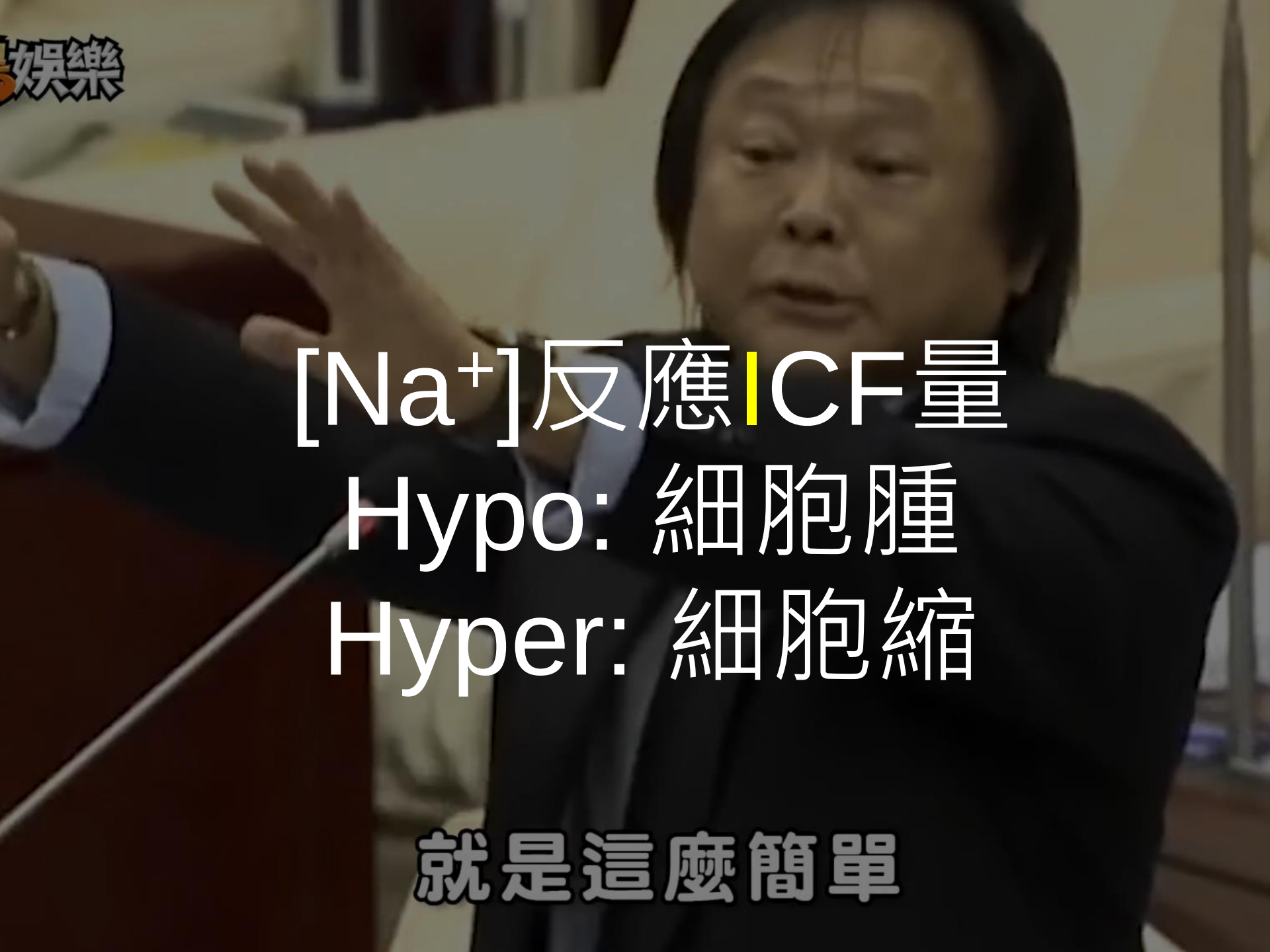
高鈉鑑別診斷(尿崩)、對應治療

Overcorrection- complication



Hyponatremia

Excess H2O relative to Na, usually due to ↑ ADH


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

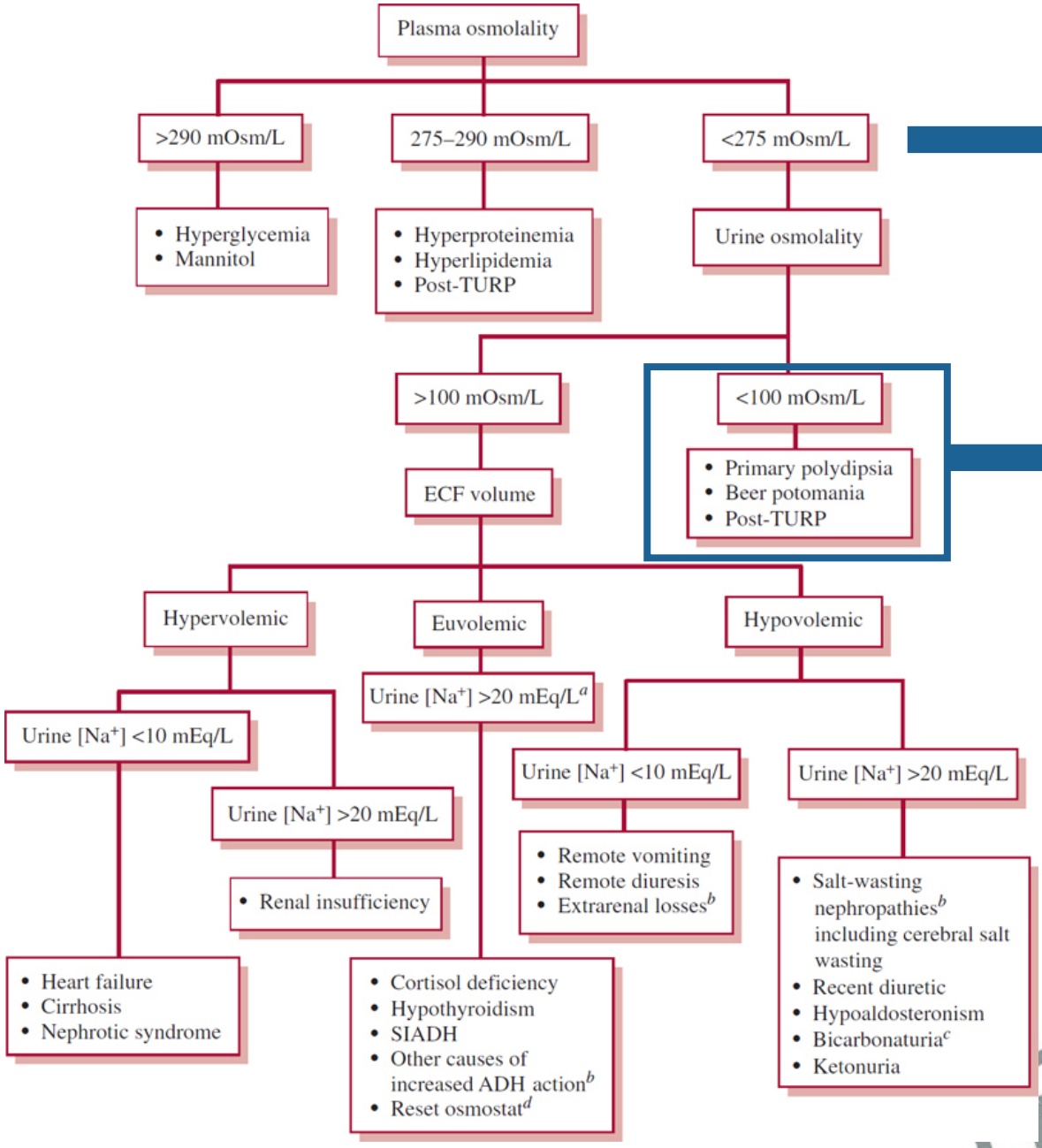
Hyponatremia

- 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

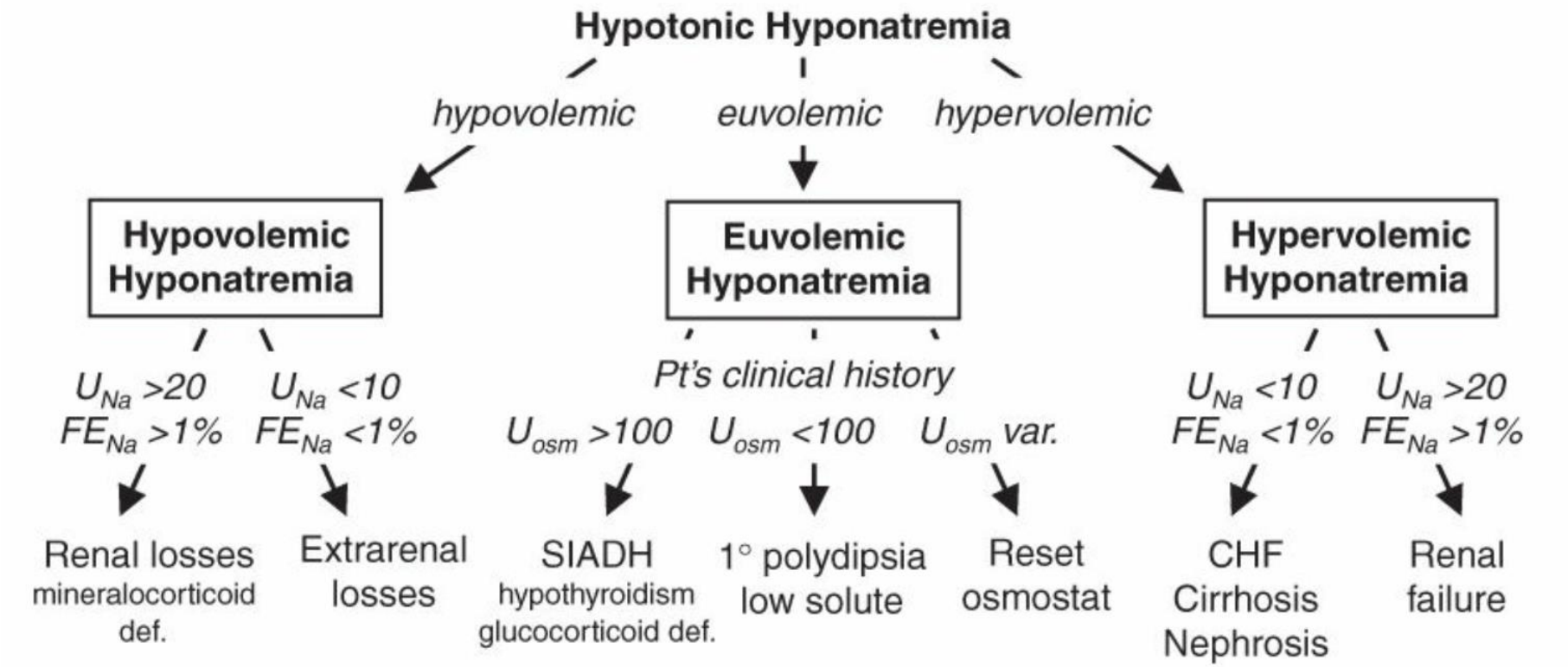


true

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 hyponatremia

(ie, ↓↓ total body Na, ↓ TBW)

Renal losses: esp. **thiazides**

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

Euvolemic 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

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

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

(C)病人出現低鈉血症，是由於體液過多所致

(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

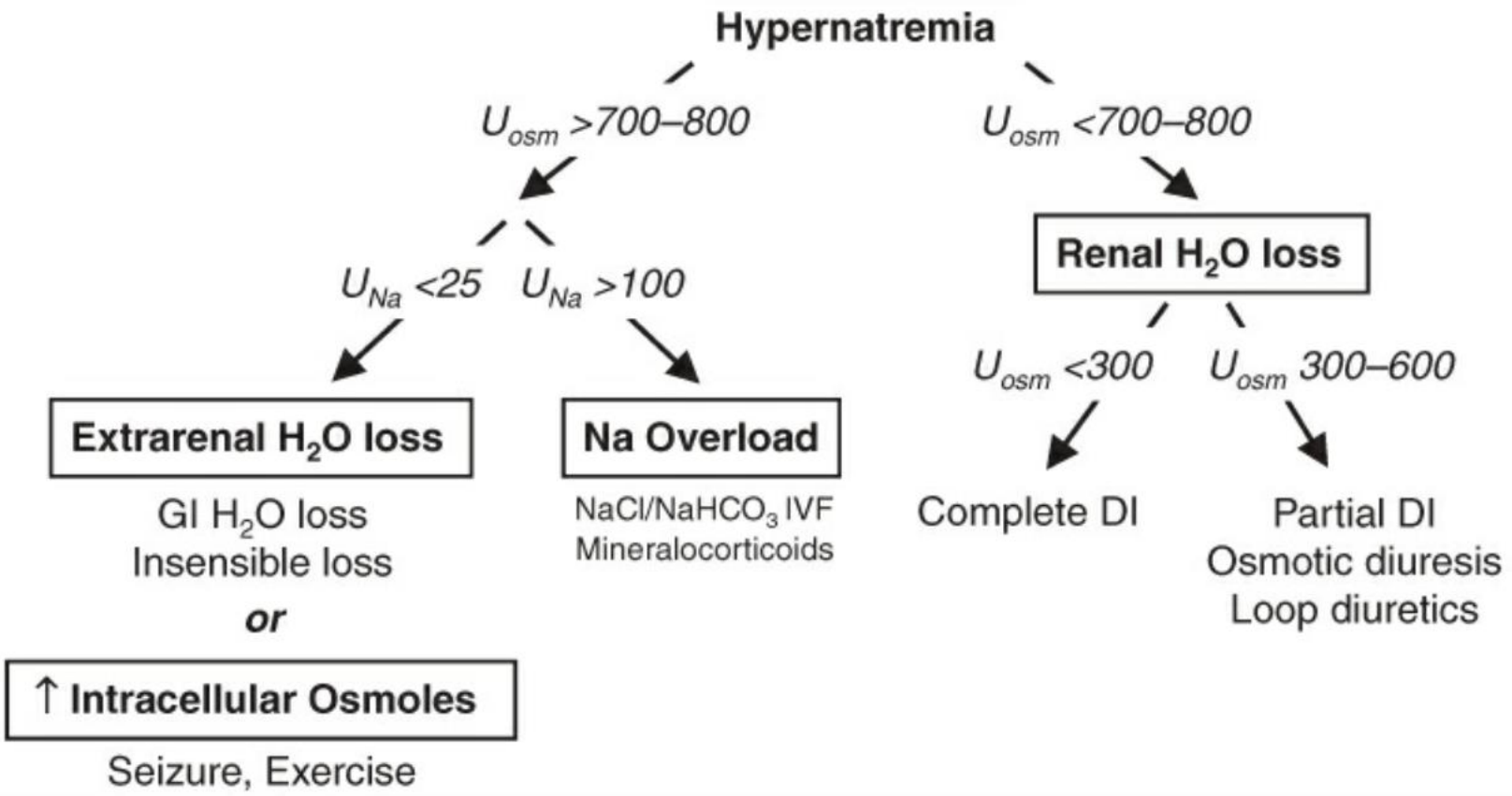
B. 立即給予靜脈滴注3% saline以矯正其血中[Na^+]

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


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
Na Overload
GI H_2O loss
Insensible loss
or
↑ Intracellular Osmoles
Seizure, Exercise
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

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

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

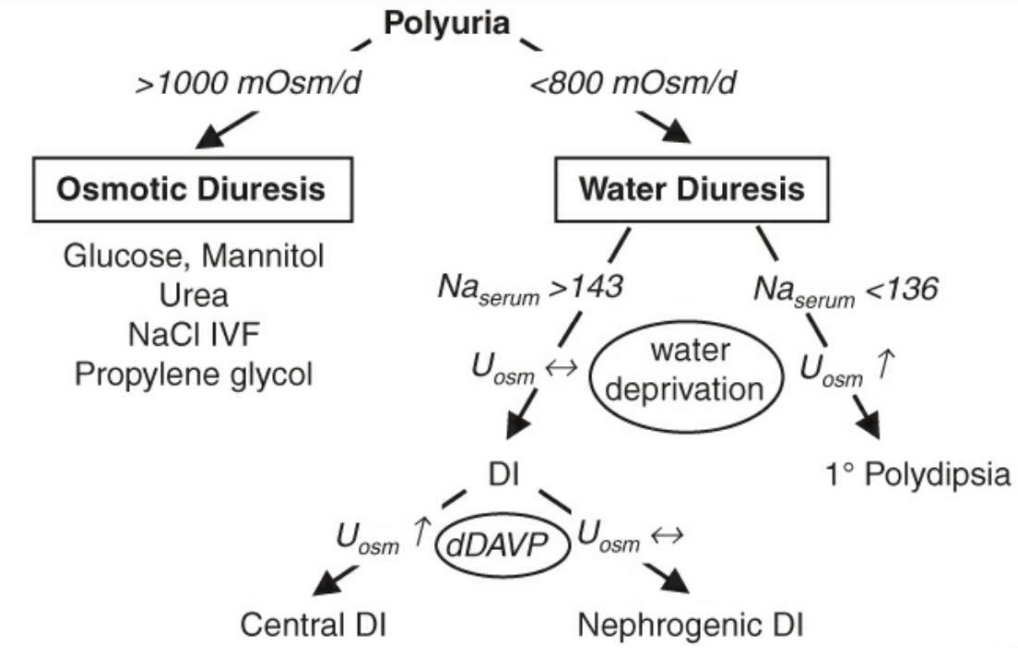
- Etiologies
  - Glucose (uncontrolled diabetes mellitus)
  - Mannitol
  - Urea: recovering ARF, ↑ protein feeds, hypercatabolism (burns, steroids), GI bleed
  - NaCl administration
- Treatment: address underlying cause, replace free-water deficit and ongoing losses

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)
  - **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*



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

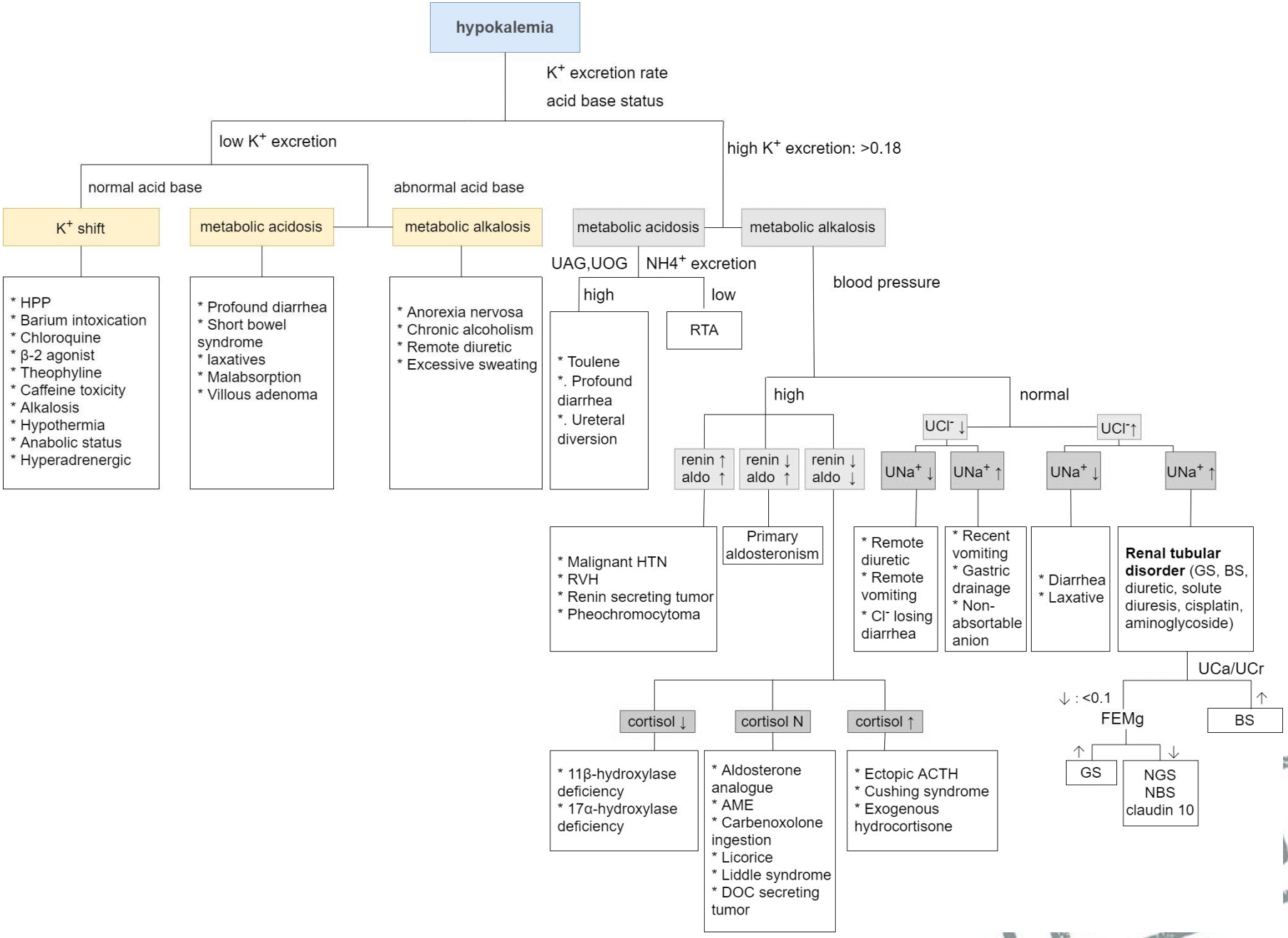
4. 病患為滲透性利尿症（ osmotic diuresis ）

Potassium Homeostasis

低鉀鑑別診斷、對應治療

高鉀鑑別診斷、對應治療

EKG change


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
* 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
UAG,UOG
NH4+ excretion
high
low
* Toulene
*. Profound diarrhea
*. Ureteral diversion
RTA
blood pressure
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

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

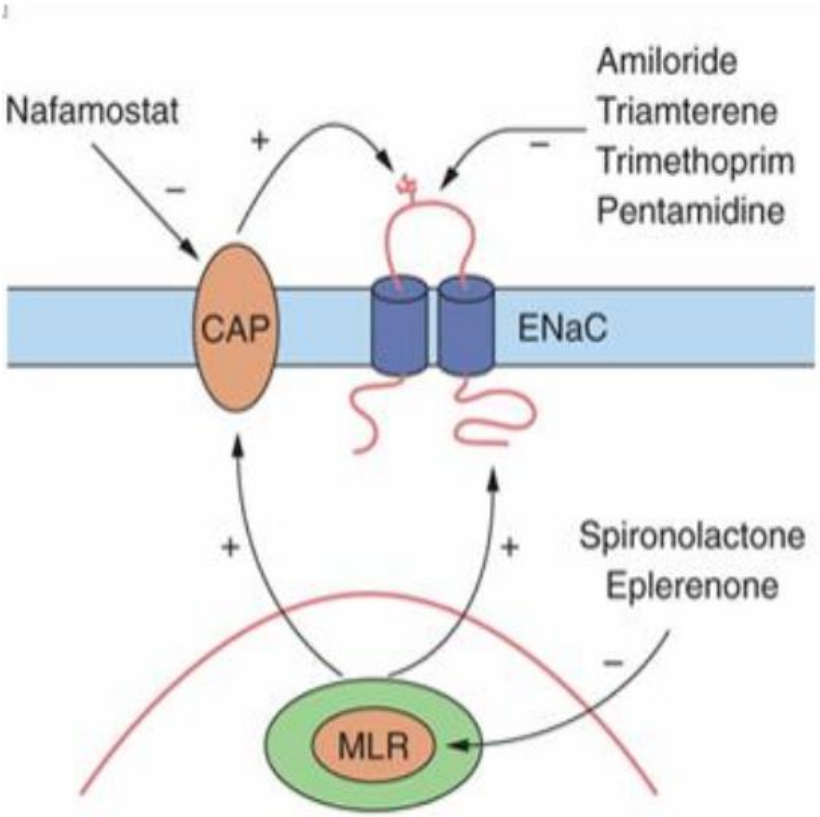
$ 合併低血鎂的病人

* 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

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

其他來源高起來的~cortisol

濃度太高，會作用在
aldosterone receptor

Suspect Hyperaldosteronism

Plasma renin & aldosterone a.m. collection

↓ 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

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）

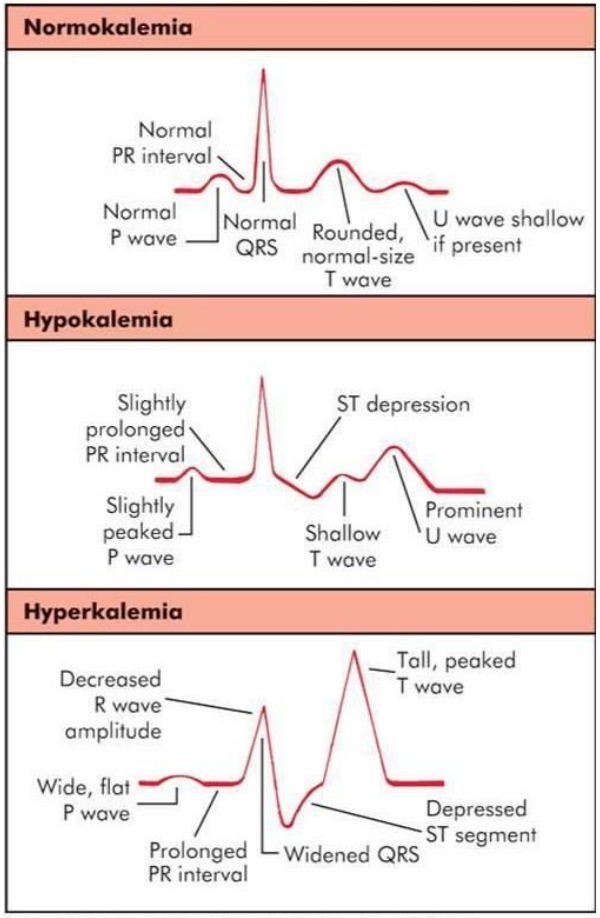
2. Liddle’s 症候群（ Liddle’s syndrome）

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

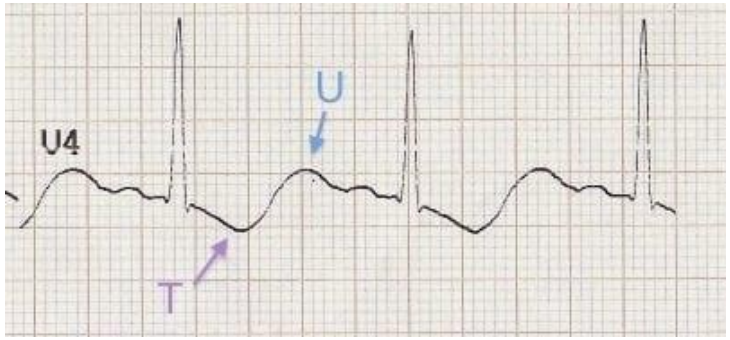
ECG Changes with Potassium Imbalance



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)

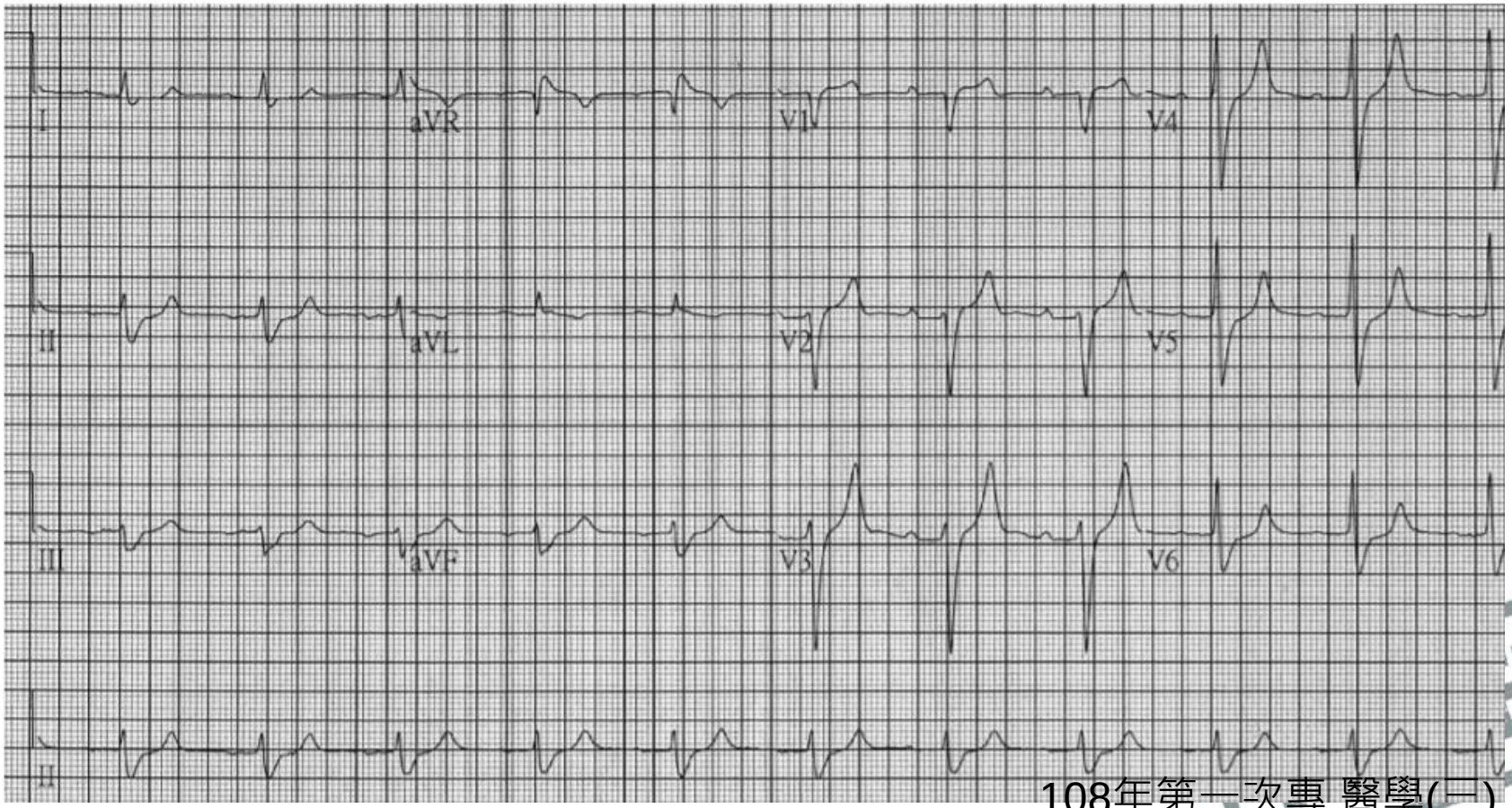


Therapy of Hyperkalemia	
Stabilization of the Membrane	
IV calcium	Works within min; action lasts up to 1 hr Ca gluconate 3 amp (3 g, 10% 30 mL, 14 mEq Ca) via peripheral line or $CaCl_2$ 1 amp (1 g, 10% 10 mL, 13.6 mEq Ca) via central line over 2–3 min Avoid in digitalis toxicity
Drive Extracellular 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

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 ） .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)

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)

109-01
一位25歲男性，突然發現下肢無力、無法站立與走路、需家人攙扶，遂在隔天早上由家屬陪同就醫。實驗室檢查發現低血鉀（2.0 mEq/L）及低血磷（1.8 mg/dL），而血清肌酸酐（creatinine 0.6 mg/dL）、鈉（139mEq/L）、鈣（2.2 mmol/L）及血清滲透壓（serum osmolality 290 mOsm/kg）則在正常範圍。同時尿液中的鉀與滲透壓分別為10 mEq/L與580 mOsm/kg。下列敘述何者最適當？

A.TTKG（transtubular potassium gradient）計算後等於2.0
B.此病患低血鉀是因腎臟排出過多鉀離子
C.需加驗甲狀腺功能
D.經由周邊靜脈補鉀時，應將氯化鉀加在葡萄糖水裡，鉀離子濃度儘量不超過20～40 mmol/L

109-27
某70歲女性患者近2週嘔吐、腹痛漸增而至急診；過去病史：糖尿病10多年，一個月前腎功能正常，目前使用之藥物包括metformin 1.5 g daily，losartan 50 mg daily，以及simvastatin 20 mg daily。實驗室檢查發現：Na+139 mmol/L，K+ 4.9 mmol/L，Cl- 103 mmol/L，glucose 216 mg/dL，cholesterol 220 mg/dL，Ca2+ 8 mg/dL，Cr 6.5 mg/dL；動脈血氣體pH 7.0，HCO3- 4.0 mEq/L，PCO2 14 mmHg，尿液無異常。下列何者為最可能之診斷？

A.diabetic ketoacidosis
B.rhabdomyolysis
C.lactic acidosis
D.type 4 renal tubular acidosis

110-25巴特氏症候群（Bartter’s syndrome）的腎小管上皮細胞功能失調出現在：

1.近端腎小管

2.亨利氏環粗上升支

3.遠端腎小管

4.集尿小管

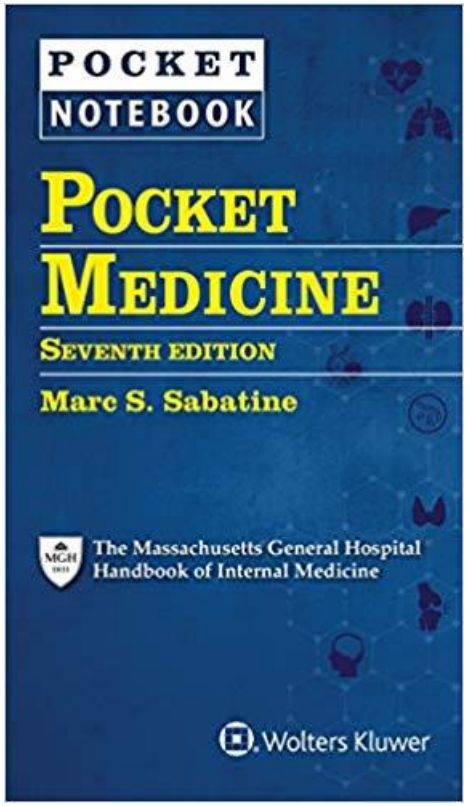
110-50
35歲女性病患，體檢時發現**血壓偏高**。她主訴近2個月來覺得頭痛、全身無力和倦怠。理學檢查除了血壓高（160/100 mmHg）以外，並無特殊發現。血液檢驗發現血中肌酸酐（creatinine）1.1 mg/dL，鈉離子137mmol/L，**鉀離子 2.6** mmol/L，動脈血液氣體檢查發現**代謝性鹼中毒**（metabolic alkalosis），血清中腎素（renin）為0.04 ng/mL/hr（參考值，1～5 ng/mL/hr）；血中皮質醛酮濃度（plasma aldosterone concentration）為39.2 ng/dL（參考值，5～30 ng/dL）； 血中腎上腺刺激素（adrenocorticotropin）為12 pg/mL（參考值，10～65 pg/mL）。此病患最有可能的診斷為何？

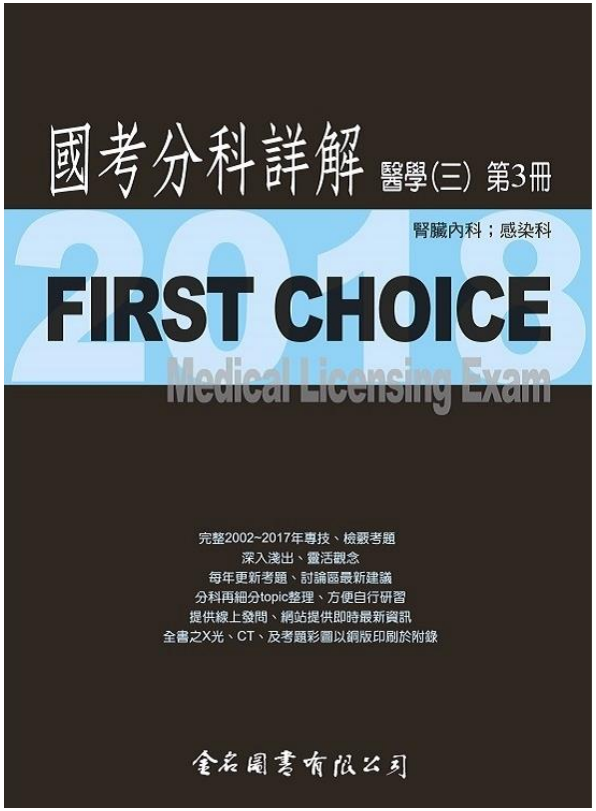
A.腎動脈狹窄（renal artery stenosis）
B.甲狀腺亢進（hyperthyroidism）
C.原發性皮質醛酮症（primary aldosteronism）
D.庫欣氏症候群（Cushing's syndrome）

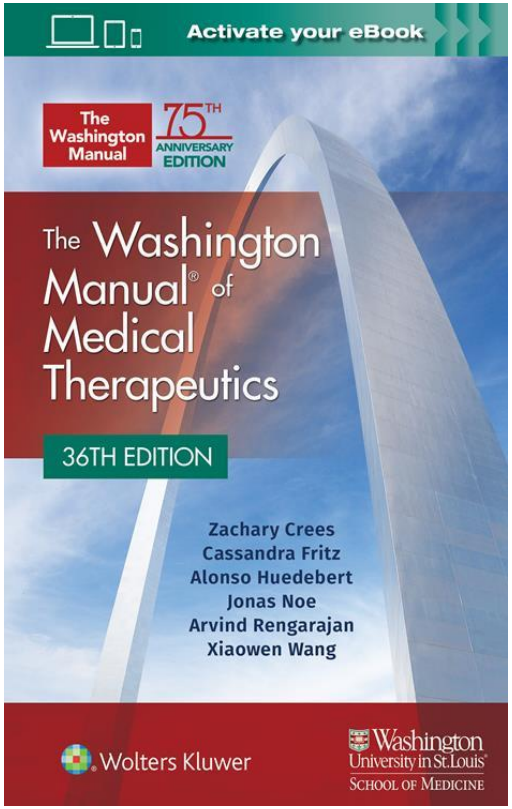
110-62

一位55歲男性兩天來嘔吐不止送至急診處。病人過去有十二指腸潰瘍，兩天前開始噁心嘔吐，但無腹痛或發燒。身體診察，脈搏每分鐘118，血壓88/50 mmHg，無貧血或黃疸；胸腔和心臟正常，上腹部稍有壓痛但無反彈疼痛，腸動聲稍減，血清電解質（mmol/L）：Na+ 136，K+ 2.9，Cl- 89。動脈氣體分析如下：pH 7.49，PaCO2 45 mmHg，PaO2 98 mmHg，HCO3- 32 mEq/L。下列有關對此病人的敘述何者最為正確？

A.尿液的氯離子應該<20 mmol/L
B.尿液的滲透壓應該<300 mOsmol/kg H2O
C.血液的滲透壓應該<270 mOsmol/kg H2O
D.尿液的鉀離子應該<10 mmol/L


POCKET
NOTEBOOK
POCKET
MEDICINE
SEVENTH EDITION
Marc S. Sabatine
The Massachusetts General Hospital
Handbook of Internal Medicine
Wolters Kluwer


國考分科詳解 醫學(三) 第3冊
腎臟內科；感染科
2018
FIRST CHOICE
Medical Licensing Exam
完整2002~2017年專技、檢覈考題
深入淺出、靈活觀念
每年更新考題、討論區最新建議
分科再細分topic整理、方便自行研習
提供線上發問、網站提供即時最新資訊
全書之X光、CT、及考題彩圖以銅版印刷於附錄
金名圖書有限公司


Activate your eBook
The Washington Manual
75TH ANNIVERSARY EDITION
The Washington Manual® of Medical Therapeutics
36TH EDITION
Zachary Crees
Cassandra Fritz
Alonso Huedebert
Jonas Noe
Arvind Rengarajan
Xiaowen Wang
Wolters Kluwer
Washington University in St. Louis
SCHOOL OF MEDICINE


PASSED