YAQEEN BATCH

PHYSIOLOGY

lecture : 6

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Lecture 6- Renal physiology

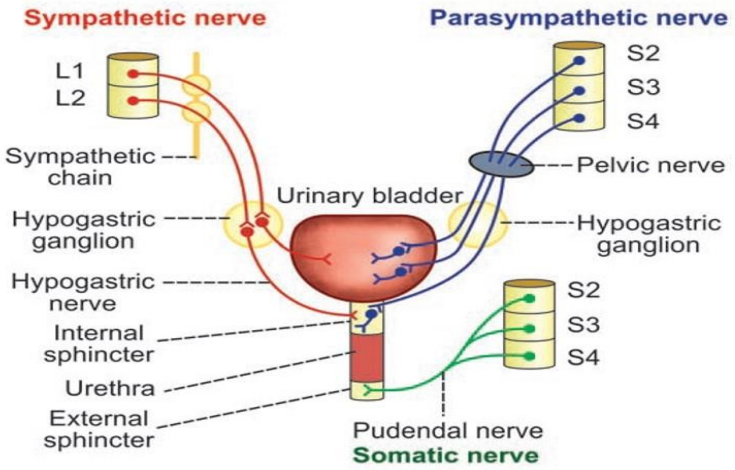
Sympathetic : from the lumber region >> preganglionic fibers >> synapse in the hypogastric region >> post ganglionic sympathetic fibers innervate (detrusor muscles , internal sphincter)

Parasympathetic :from the sacral region the pelvic nerve innervates (detrusor muscles , internal sphincter)

somatic :pudendal innervates the external voluntary sphincter (which is normally contracted and relaxes when the micturition occurs

الجدول مهم جدا نركز انه الجهاز العصبي الودي بعبي المثانة وبقلل التبول يعني بسكر ال internal sphincter وبرخي عضلات المثانة وعلى عكسه تماما الجهاز العصبي الودي بشجع عملية التبول فبرخي internal sphincter وبتنقبض عضلات المثانة لكن عملية التبول النهائية بتعتمد على ارادة الشخص نفسه وال higher centers

Innervation of urinary bladder



Pelvic nerve has sensory fibers→ impulses from stretch receptors in urinary bladder and urethra →CNS

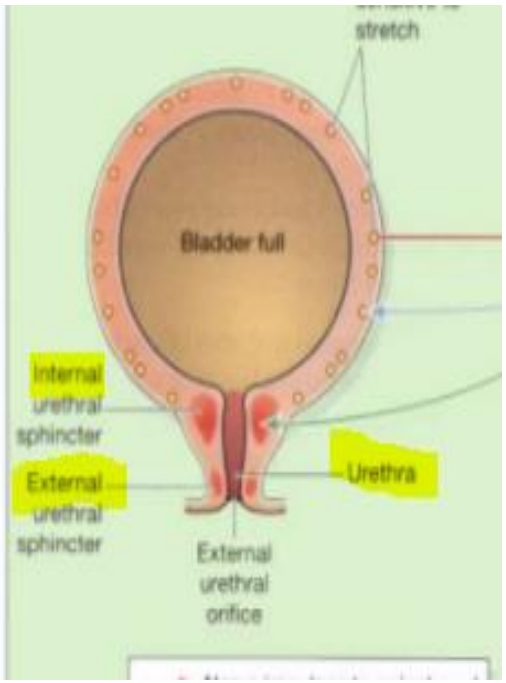
Nerve	On detrusor muscle	On internal sphincter	On external sphincter	Function
Sympathetic nerve	Relaxation	Constriction	Not supplied	Filling of urinary bladder
Parasympathetic nerve	Contraction	Relaxation	Not supplied	Emptying of urinary bladder
Somatic nerve	Not supplied	Not supplied	Constriction	Voluntary control of micturition

اذا هي عملية اني افضي البول من المثانة
لازم كمية البول توصل حد معين threshold بحيث بعد هاي الكمية العضلات تتوسع :

stretching and the stretch receptors detect this tension >> signals by the afferent fibers to the spinal cord (the whole reflex is on this level) and the afferent parasympathetic pelvic fiber contracts the detrusor m.s and relaxation of internal sphincter(the urine flow throuth the uerethra (when there is a desire the parasympathetic inhibits the pudendal nerve (relaxation of external sphincter >> urine >> emptying

Micturition

- Emptying the Urinary Bladder when it becomes filled →tension in its walls > threshold level→ micturition reflex
- Contraction of detrusor muscle→↑pressure in bladder to 40-60 mm Hg → emptying the bladder
- Internal sphincter→ prevents emptying of bladder until pressure in bladder > threshold level
- External sphincter→ voluntary skeletal muscle, used to consciously prevent urination



The basal volume after which recognized increase in pressure occurs is 150 MML
At this volume until 200- 300 mml the micturition waves created and their intensity increases with urine volume after 400 mml the micturition contractions increase rapidly

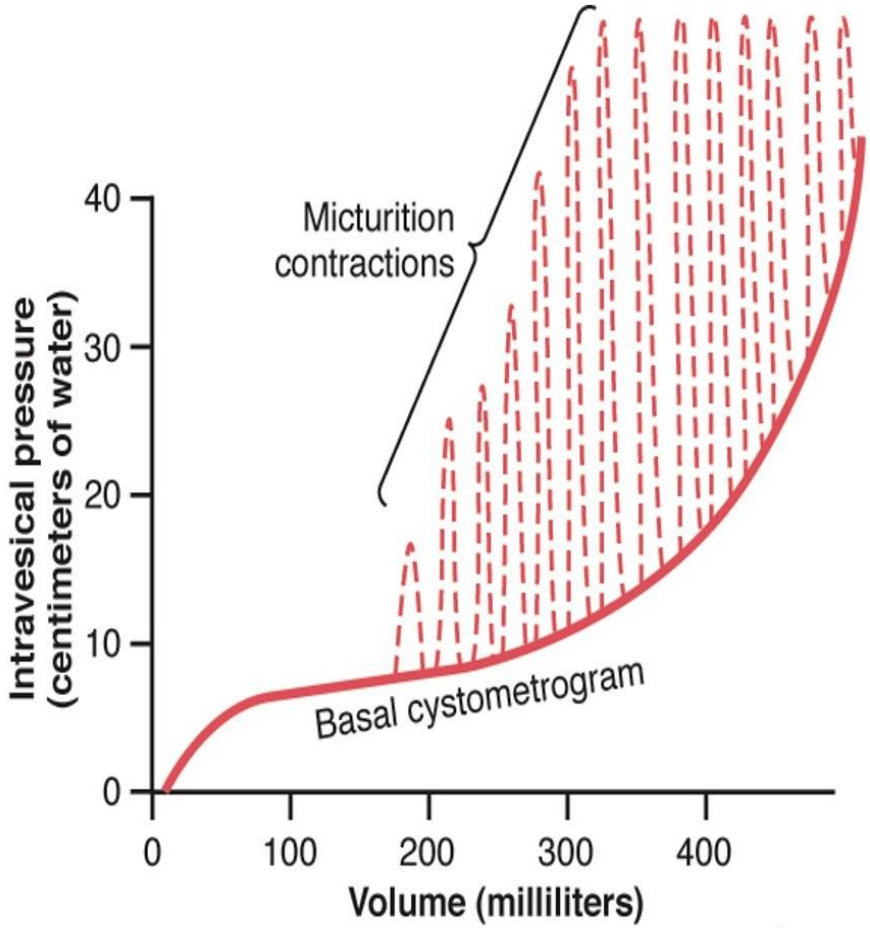
Filling of Bladder and Bladder Wall Tone; Cystometrogram

Tonic pressure changes

- No urine in bladder→intravesicular pressure is about 0
- 30-50 ml of urine →pressure rises to 5 -10 cm H2O
- 200-300 ml—only small additional rise in pressure; caused by **intrinsic tone** of the bladder wall.
- Beyond 300-400 ml→ pressure rises rapidly

Micturition waves

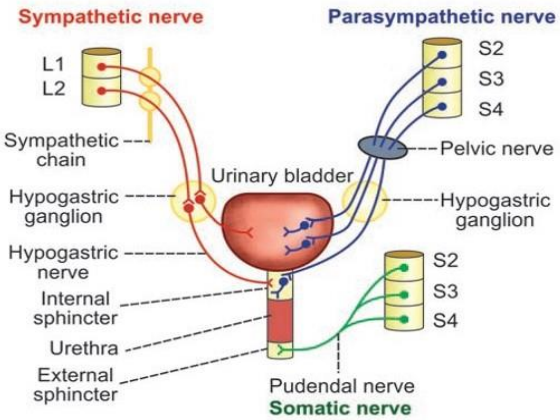
- Superimposed on the **tonic pressure** changes during filling
- Periodic acute increases in pressure
- Caused by the **micturition reflex**.

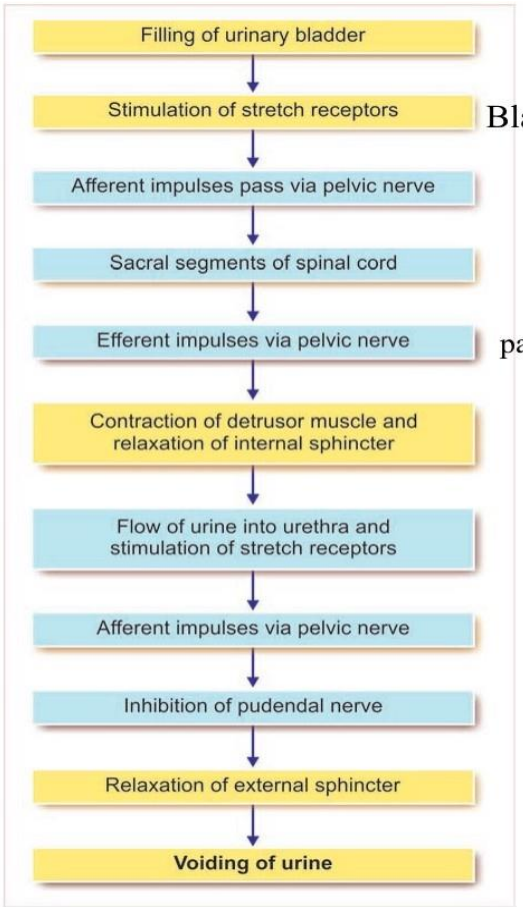


Micturition Reflex

- Autonomic spinal cord reflex
- Contraction of Detrusor muscle
- Inhibited / facilitated by brain

- When bladder is partially filled →micturition contractions usually relax.
- As bladder continues to fill → Micturition reflexes become more **Frequent** and **Powerful**





امرات بتكون كمية البول بالمثانة قليل مش لدرجة يعمل رغبة بالتبول بس مثلا الدكتور بطلب من مريض عينة بول كيف رح يقدر يتبول ؟ من خلال انه بضغط من عضلات البطن بزيد الضغط داخل البطن وبتالي داخل المثانة فبنزل البول والدماغ بسهل هاي العملية
امرات بتتعبى المثانة شوي وخلص بطلع موجات micturition بس بتختفي اما ازا ضلت تعبي لا بتزيد وبتصير اقوى واكثر

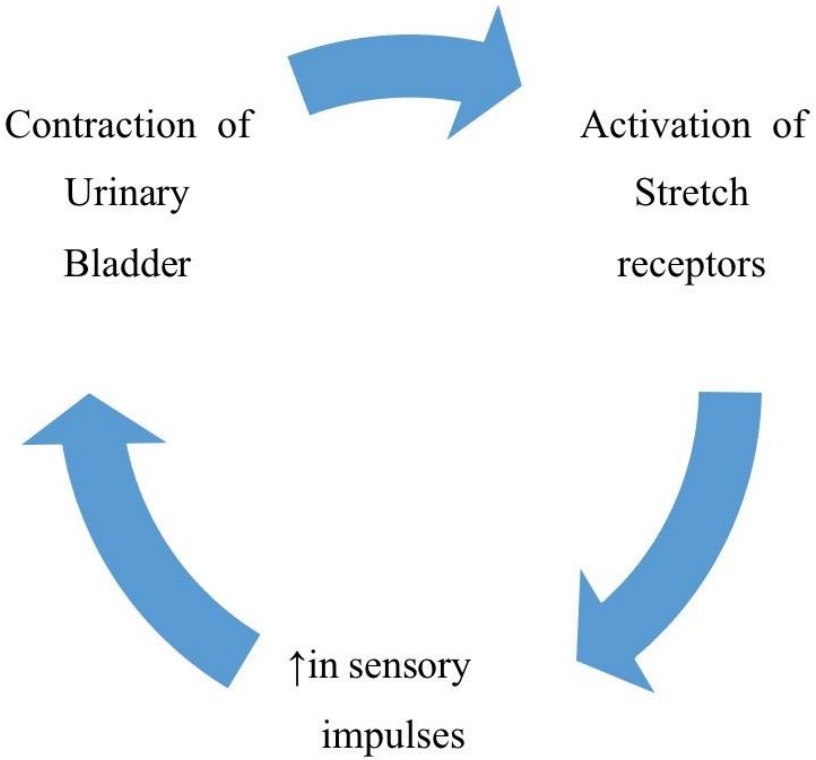
Self-Regenerative Reflex:

a single complete cycle of:

i.Progressive and Rapid increase of Pressure

ii. Sustained Pressure

iii. Relaxation



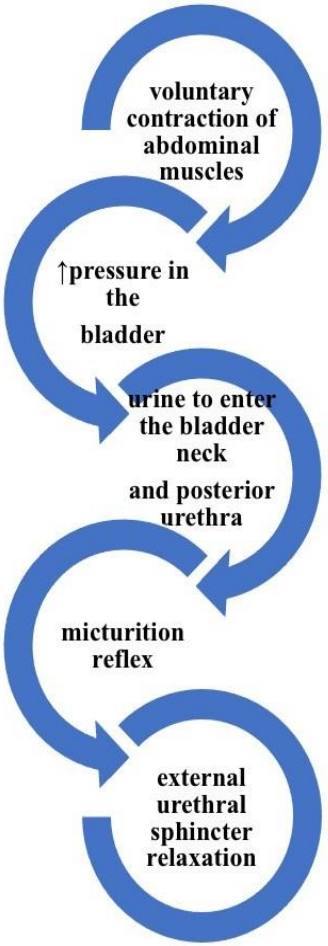
The higher centers control the micturition in 2 ways :
1)Partial inhibition to the whole micturition reflex
2) Inhibition to the external sphincter
Or a voluntary micturition may occur even and lower volumes by the pressure of the abdominal muscles which increases the intravesical pressure in the bladder

Control by Higher Centers

Higher centers normally exert final control of micturition

- Pon & cerebral cortex
- Partial **inhibition of micturition reflex**, except when micturition is desired.
- Prevent micturition, even if micturition reflex occurs, by **tonic contraction of external urinary sphincter** until a convenient time presents itself.
- Cortical centers can facilitate sacral micturition centers to initiate micturition reflex & relax external sphincter

Voluntary urination



the peristalsis is the strongest at the ureter:
The sympathetic inhibits micturition not only in the bladder level by the (relaxation of ms. And constriction of the internal sphincter .but also by lowering peristalsis in the ureter >> lower urine delivery to the bladder
parasympathetic والعكس صحيح بل

Transport of urine to urinary bladder

- No change in composition
- Urine from Collecting Duct→ Calyces (↑Pacemaker activity → peristalsis) →Pelvis→ Ureter →Urinary Bladder

Sympathetic stimulation:
↓Peristalsis
Parasympathetic stimulation:
↑Peristalsis

هلا منعرف قبل ما الحالب يفوت على المثانة بمشي 2 سم بشكل مائل مش رأسي وهناك في زي تضيق بمنع البول في حالد المثانه انقبضت يرجع على الجالب والكلية

عند الاطفال الحالب اقصر ومش مائل بشكل كافي ممكن فعلا البول يرجع على الحالبvesicoureteral reflex يدمر الكلية يرفع الضغط فيها ويزيد خطر العدوات

Flow of urine from ureter into urinary bladder

- Peristalsis in ureters forces urine into urinary bladder
- Oblique course+ compressed by detrusor muscle tone→Prevents Vesicoureteral Reflux
- Reflux →enlargement of ureters+ ↑pressure in renal calyces & medulla →damage

الشخص اللي عنده حصوات علقت في الحالب بكون الالم النتاج عنها شديد فبصير عنها ureterorenal reflex انه الجهاز العصبي الودي بيعمل انقباض في شرييناات الكلية فبقل معدل الترشيح والبول فببطل عندي كمان بول نازل من الكلية يضغط على حصوات الحالب يزيد المه

Pain sensation in Ureters

- Well supplied with pain nerve fibers
- Irritation/ block (e.g. stone) → intense stimulation of pain nerve fibers → Intense contraction of ureters (severe pain)

↓

Sympathetic reflex back to kidney→ vasoconctriction of renal arterioles→ ↓the urine output

=**Ureterorenal reflex→ preventing excessive flow of fluid into pelvis**

Acid-Base Regulation

Chapter 31
Unit V

Dr Iman Aolymat

Introduction

acid-base buffering mechanisms involved in maintaining normal H+ concentrations in ECF & ICF:

1-blood
2-cells
3-lungs
4-kidneys

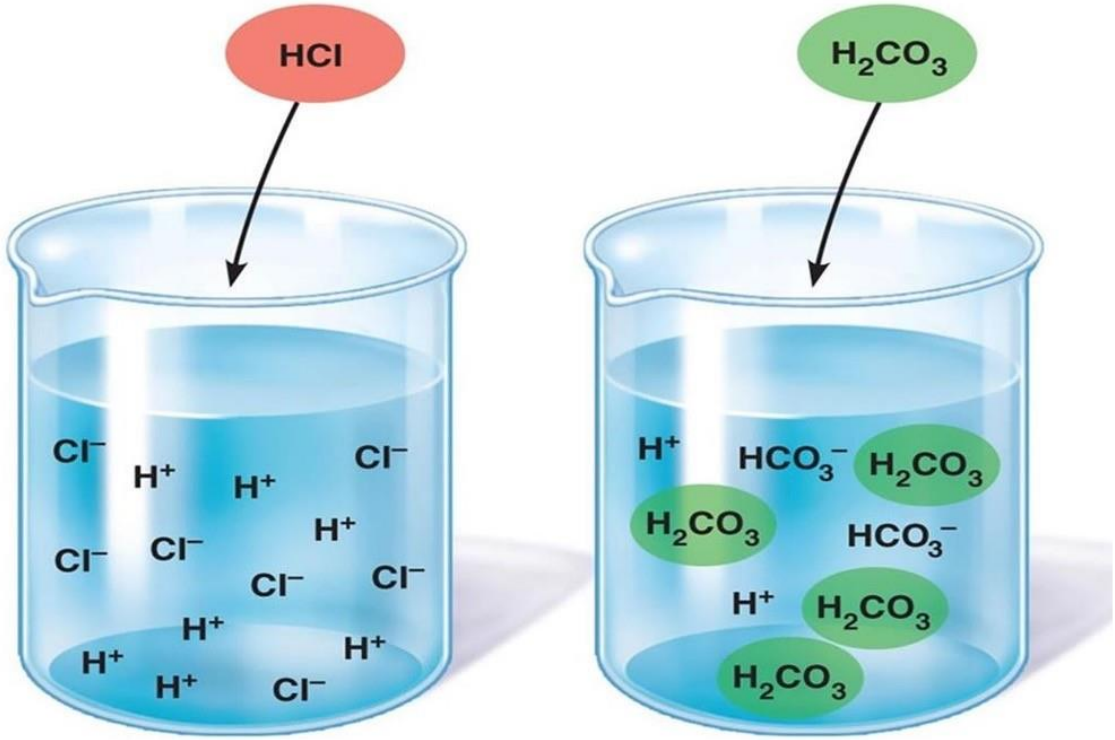
Acid-Base Fundamentals

- ***An Acid*** = a molecule that can release H^+ in a solution.
  - H2CO3 (carbonic acid) Weak
  - HCl (hydrochloric acid) Strong

- ***A base*** = a molecule that accepts H^+ in a solution.
  - Bicarbonate ions (HCO_3^-).
  - Hydrogen phosphate (HPO_4^{-2})
  - Proteins

There is amino acid in these protiens this increases the negative charge that accept the proton and strongly lower acidity

Strong vs weak Acid/Base



Strong acids **dissociate rapidly and release large amounts of H^+ in solution**

Weak acids **dissociate incompletely and less strongly releasing small amounts of H^+ in solution**

A **strong base** is one that reacts **rapidly** and strongly with H+ → quickly removing H+ from a solution.
Example is OH− + H+ → H2O

weak base e.g HCO3− because it binds with H+ much more weakly than does OH−.

Most acids and bases in ECF that are involved in normal acid-base regulation are **weak** acids and bases

Acidosis = excess addition of H+

Alkalosis = excess removal of H+ from the body fluids

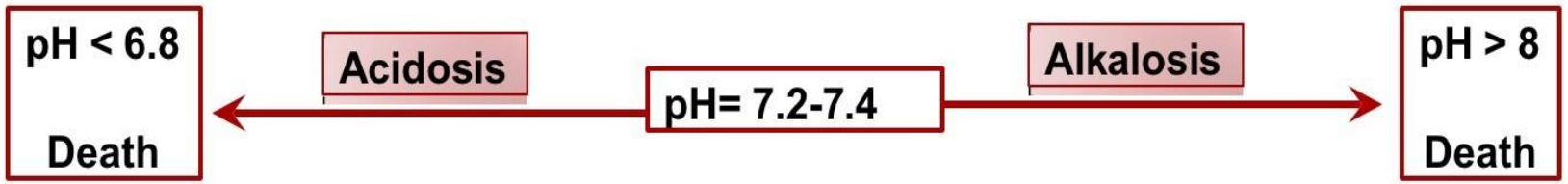
معدل الحموضة مهم واي تغير بسيطفيه بأثر على عمل الانزيمات وبكسر البروتينات
طبعا بعتمد على تركيز الهيدروجين وضروري نحفظ الرقم المحدد
طبعا علاقة ال ph عكسية مع تركيز الهايدروجين

[H^+] & the pH

- [H+] is precisely regulated at 0.00004 mEq/L (important for enzyme functions)
- H^+ ion concentrations are expressed as pH.

- pH ∝ - Log [H^+]
  - If the [H^+] increase → pH will decrease (more acidic)
  - If the [H^+] decrease → pH will increase (more alkaline)

Normally pH= 7.2-7.44



طبعا لما يقل التزيد الدموي على نسيج معين بقل الاكسجين فبلش اكسر الجلوكوز وانتج طاقة عن بطريقة لاهوائية بطلع فيها احماض مثل ال lactic acid فبتزيد الجموضة
كمان ال ph داخل الخلايا حمضية اقل من البلازما بشوي بسبب انه جوا الخلية عندي عمليات ايض واحماض بتنتج

Table 31-1 pH and H^+ Concentration of Body Fluids

	H^+ Concentration (mEq/L)	pH
Extracellular fluid		
Arterial blood	4.0×10^{-5}	7.40
Venous blood	4.5×10^{-5}	7.35
Interstitial fluid	4.5×10^{-5}	7.35
Intracellular fluid	1×10^{-3} to 4×10^{-5}	6.0-7.4
Urine	3×10^{-2} to 1×10^{-5}	4.5-8.0
Gastric HCl	160	0.8

Intracellular pH usually is < plasma because the **metabolism** of the cells produces acid especially (H2CO3).

Hypoxia and **poor blood flow** to tissues → acid accumulation and ↓intracellular pH.

Acid Production by the Body

- The body produces large amounts of acids on daily basis as by products of metabolism.
  - Metabolism of dietary **proteins**.
  - Anaerobic metabolism of **carbs and fat**.

- Acids in the body are of two kinds:
  1. Volatile (CO_2) الغازات بشكل عام
  2. Non-volatile “fixed” (sulfuric acid, lactic acid)

The Body's Defense Against Changes in $[H^+]$

Three main systems:

1. ***Body fluid buffers.***

   Works within seconds (bind acid/base→ weaker).

هاي الحوامض والقواعد الضعيفة خط الدفاع الاول ما بتغير اشي بتركيز الحمض او القاعدة هي بس بتمنع حدوث تغير حاد ومُفاجئ بالحموضة

2. ***Lungs***

   Works within minutes (eliminate CO_2).

عن طريق انها بتزيد معدل التنفس او بتقلله وبما انه ثاني اكسيد الكربون حمض زيادته او تقليله بساعدة بالتحكم بالحموضة لحد معين

3. ***Kidneys***

   Works within hours-days (EXCRETE ACID/BASE).

   The most powerful of the three.

الكلية اقوى واخر خط دفاع بشتغل بتمسك المادة المأثرة على الحموضة وبتطردها بالبول وكمان هي اللي بتنتج ال buffers

Chemical Buffer Systems in the Body

- ***There are 3 chemical buffers in the body;***

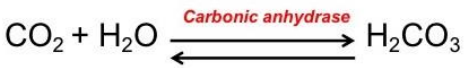
1. The HCO3 buffer system.
2. The PO4 buffer system.
3. Proteins.

- They are the 1st line of defence against changes in pH i.e. $[H^+]$, act within seconds.

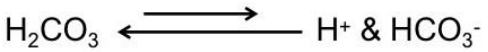
HCO3 Buffer System

- The main ECF buffer system
- Composed of:
  - A weak acid (H_2CO_3).
  - Its conjugated base ($NaHCO_3$).

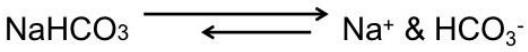
1. ***H_2CO_3 forms in the body by the reaction of CO_2 & H_2O***



2. ***H_2CO_3 ionizes weakly to form small amounts of H^+ & HCO_3^-***



3. ***The second component is $NaHCO_3$ which dissociates to form Na^+ & HCO_3^-***



HCO3 Buffer System

Putting it all together;

$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + \underbrace{HCO_3^-}_{+ Na^+}$$

Adding ACID (HCl)

$$(HCl \rightarrow H^+ + Cl^-)$$

$$+ HCO_3^- \rightarrow H_2CO_3 \rightarrow CO_2 + H_2O$$

lung

Weak acid

Adding base (NaOH)

$$CO_2 + H_2O \longrightarrow H_2CO_3 \longrightarrow \uparrow HCO_3^- + H^+$$

$$+ \qquad\qquad +$$

$$NaOH \qquad\qquad Na$$

+oH =H2o

Other Buffering Systems

The phosphate buffer:

- Plays a major role in buffering intracellular & renal tubular fluid.

- Composed of;
  - $H_2PO_4^-$ (dihydrogen phosphate/ACID)
  - HPO_4^{-2} (Hydrogen phosphate/BASE)

Proteins: PLENTIFUL

- Contributes to buffering inside cells→ H+ /HCO3- diffusion to the cell.
- E.g. Hb.

Summary of Body's Buffering Systems

- Buffer systems do not work independently in body fluids but actually work together.
- A change in the balance in one buffer system, changes the balance of the other systems.
- Buffers do not reverse the pH change, they only limit it.
- Buffers do not correct changes in $[H^+]$ or $[HCO_3^-]$, they only limit the effect of change on body pH until their concentration is properly adjusted by either the lungs or the kidney.

The Henderson-Hasselbalch Equation

What is the HHE?

- It is an equation that enables the calculation of pH of a solution.

What is it?

$$pH = pK + log \frac{HCO3^-}{0.03\ X\ PCo_2}$$

K = dissociation constant, pK = 6.1

0.03 = solubility of CO_2

من المعادلة تركيز البايكربونات طردي مع ال ph
وثاني اكسيد الكربون عكسي معاه

The Henderson-Hasselbalch Equation

$$pH = pK + \log \frac{[HCO3^-]}{0.03\ X\ PCO2}$$

- ***What do we understand from this equation?***

1. pH ∝ $\frac{HCO3-}{PCO2}$ **Regulated by kidneys** / **Regulated by lungs**

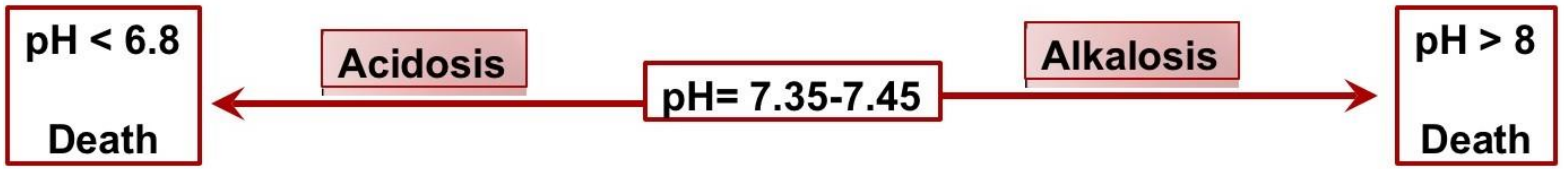
Each element of the buffer system is regulated

- ↑↑ HCO_3^- will ↑↑ pH
- ↑↑ PCO2 will ↓↓ pH

Respiratory regulation of acid-base balance

Acidosis= excess addition of H+

Alkalosis= excess removal of H+ from the body fluids

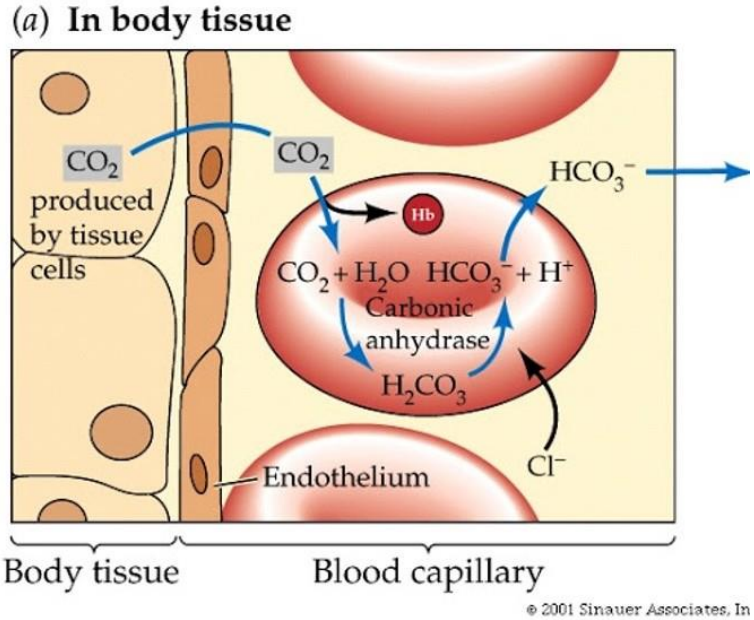


ثاني اكسيد الكربون حمض فلما يصير عندي حموضة بالجسم الرئة بتزيد معدل التنفس وبتغسله من الجسم وكونه حمض لما يقل بقلل الحموضة وبرجع الph طبيعي والعكس لما يصير عندي قاعدية بقلل معدل التنفس لحد معين طبعا عشان الاكسجين اهم فبزيد معدل ثانكي اكسيد الكربون بالجسم فبنقلل الحموضة

Respiratory Regulation of A/B

- 2nd line of defence against acid-base disturbances in the body.
- By modulating CO_2 excretion.
- Normally, PCO2 = 40 mmHg (35-45 mmHg).
- ↑CO2 formation→ ↑ECF CO2 → ↑ ECF PCO2 & Vice versa.

↑ECF CO2→ ↑H2CO3→ ↑H+



$$pH \propto \frac{HCO3}{PCO2}$$

Respiratory regulation of A/B

$$pH \propto \frac{HCO3}{PCO2}$$

- Response occurs within 3-12 minutes.
- ↑↑ ventilation (RR) → ↓↓ P_{CO_2} → ↑PH
- ↓↓ ventilation (RR) → accumulation of CO2→↑↑ P_{CO_2}→ ↓PH

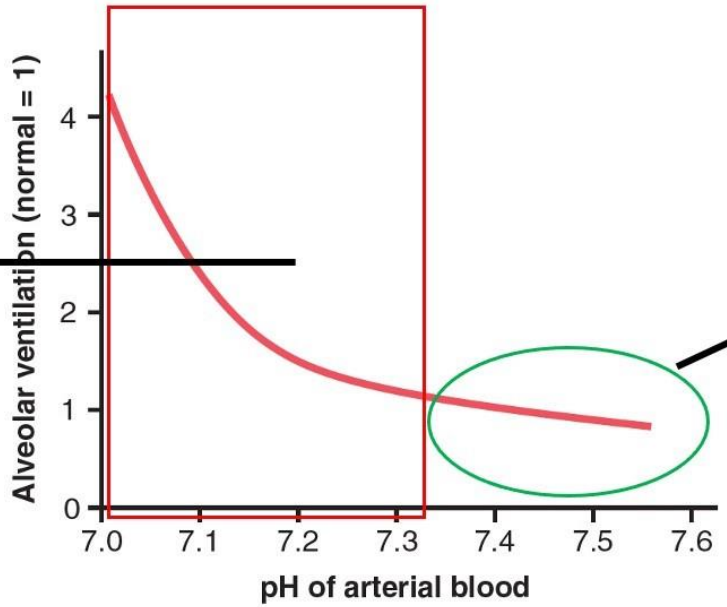
Respiratory Regulation of A/B

$$pH \propto \frac{HCO3}{PCO2}$$

- ↓↓ $[H^+]$ → ↑pH → ↓↓ ventilation (RR) → accumulation of CO_2 → ↑↑ P_{CO_2}.
- ↑↑ $[H^+]$ → ↓pH → ↑↑ ventilation (RR) → ↓↓ P_{CO_2}

More changes in RR at low pH

الفكرة بهاد السلايد انه الية الرئة بتنظيم حموضة الجسم فعالة اكثر في حال ال ph قليل لانها بتزيد معدل التنفيس وبتطلع ثاني اكسيد الكربون بس في حال كان ال Ph عالي وعندي قاعدين الكلية بتقلل معدل التنفس بس مش بدرجة كبيرة لانها بالنهاية مستوى الاكسجين اهم وما بقدر اقلل التنفس بشكل كبير



Less change (↓) in RR with high pH

↓

↓PO2

↓

↑↑ ventilation

Respiratory compensation is less effective at pH increase

The End
Thank you