بسم الله الرحمن الرحيم

# Acid-base balance

اللهم علمنا ما ينفعنا وانفعنا بما علمتنا

# Acid-base balance

- The data are used to assess patients in life-threatening situations
- Blood hydrogen ion concentration [H+] is maintained within tight limits in health. Normal levels lie between 36 and 44 nmol/L (pH 7.35-7.45)
- Any H+ values outside this range will cause alteration in the rates of chemical reactions within the cell and affect many metabolic processes of the body
- Values greater than 120 nmol/L or less than 20 nmol/L are usually incompatible with life

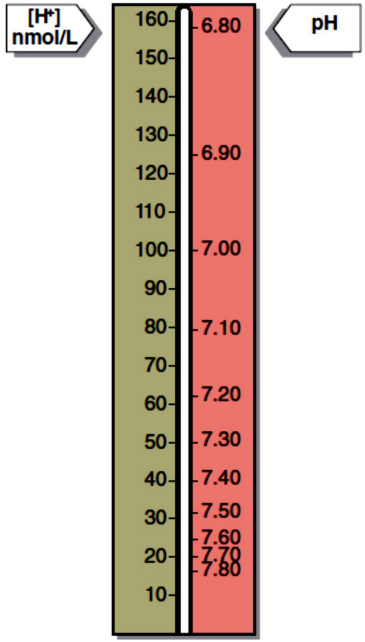



Fig 20.1 **The negative logarithmic relationship between [H+] and pH.**

# H+ Production

* مصادر $H^+$ من عمليات Metabolism في جسمنا
بناكلهم من البروتينات الحيوانيه ← Sulfer containing amino acid
هدول بكسر لهم Met و بنتجوا $H^+$
as Metabolic product

- Hydrogen ions are produced in the body as a result of **metabolism** (from the oxidation of the sulphur-containing amino acids of protein ingested as food)

اي حدا ماكل بروتينات حيوانيه رح يكون عنده Acidic Urine

- The total amount of H+ produced each day in this way is of the order of 60 mmol but all the H+ produced are efficiently excreted in urine. Everyone who eats a diet rich in animal protein passes an acidic urine

- Large amounts of **CO2** are produced by cellular activity each day with the potential to upset acid-base balance

- Under normal circumstances all of this CO2 is excreted via the **lungs**. Having been transported in the blood, Only when **respiratory function** is impaired do problems occur

بالوضع الطبيعي كل $CO_2$ بطلع من خلال الرئة لكن إذا التنفس صار ضعيف أو فيه مشكلة، ثاني أكسيد الكربون ما بطلع كويس ← برتفع في الدم
← فبصير مشكلة بال acid-base balance

# Buffering and buffers

- Buffer is a solution of the salt of a weak acid that is able to bind H+. Buffering does not remove H+ from the body but mop up any excess H+ produced (as a sponge)
- **Buffering** is only a short term solution to the problem of excess H. Ultimately, body must get rid of H by renal excretion
- The body contains a number of buffers to correct sudden changes in H production
- **Proteins** can act as buffers and the haemoglobin in the erythrocytes has a high capacity to bind H

# Buffers

$CO_2 + H_2O$ يتفكك لـ $H_2CO_3$ ويكوّن $H^+$ يرتبط بـ $HCO_3^-$
يطلع بالتنفس
carbonic acid

- In the ECF, **bicarbonate buffer** is the most important. In this buffer system, bicarbonate (HCO3-) combines with H+ to form carbonic acid (H2CO3)

- The association of H with bicarbonate occurs rapidly, but the breakdown of carbonic acid to CO2 and water happens relatively slowly.

$HCO_3^-$ — $H_2CO_3$

- The reaction is accelerated by an enzyme, **carbonic anhydrase**, which is present particularly in the erythrocytes and in the kidneys.

يحفز العمليات السابقه
الدم RBCs

- Only when all the bicarbonate is used up does the system have no further buffering capacity

لما يتم استنفاذ كل $HCO_3^-$ يعني يبطل في كمان capacity
لا buffering انه يعمل $HCO_3^-$

- The acid base status of patients is assessed by consideration of the bicarbonate system in plasma

# Buffers

لو الشخص كان عنده Acidosis رح نلاحظ انه عنده $HCO_3^-$ منخفض كونه حاول يعمل Compensation اللازم بس كان اكثر من قدرته [Capacity] فبيكون عنده متخربط (acid-base balance) انخفاضها برجع لعدم حد رتها

- The **bicarbonate buffer** system is unique in that:
  - The (H2CO3) can dissociate to water and CO2 allowing CO2 to be eliminated by lung
  - Changes in CO2 modify the ventilation rate
  - HCO3- concentration can be altered by the kidney

تحل Compensation باتجاه اللازم لانه تم استنفاذ نصفها

- **Phosphate buffer** system (HPO4= - H2PO4-) plays a role in plasma and RBC's and is involved in the exchange of Na/H+ ion in the urine filtrate

الفوسفات برتبط بال H+ وبطلعلي بالبول اكثر دوره بالـ urine

Urinary Tract بدون ما يعمل irritation تهيج بـ

- **Plasma proteins**, especially the imidazole groups of histidine, forms important buffer system in plasma. Most circulating proteins has net negative charge capable of H+ binding

Hb Hemoglobin

اي زيادة في $H^+$ رح تتجمع عند هم وترتبط بـ $H^+$

# Regulation of the acid-base balance

- In plasms at 37oC, the value for the combination of the solubility constant for PCO2 and the factor to convert mm Hg to mmol/L is 0.0307 mmol L-1. mm Hg-1

$$pH = pK' + \log \frac{cHCO_3^-}{0.031 \times PCO_2}$$

0.0307 بالزبط - للدقة -

هاد factor حتى أحول من mm Hg لـ mmol/L

أي زيادة بالـ $H^+$ بتؤدي إلى excretion in urine وبالطبع $HCO_3$ من الجسم

وأي نقص بالـ $H^+$ بيرفع الـ pH، فالكلى بترجع $HCO_3^-$ عشان تعدّل التوازن

excretion in urine أي زيادة عن بصيرة $H^+$

أي deficiency بالـ $H^+$ بسبب مرتفعة pH بجاوب الجسم

سيحب $HCO_3$ يرجعها من الكلى

# H+ excretion in the kidney

- All the H+ that is buffered must eventually be excreted from the body via the kidneys, regenerating the bicarbonate used up in the buffering process and maintaining the plasma bicarbonate concentration within normal limits.

- Secretion of H+ by the tubular cells serve initially to reclaim bicarbonate from the glomerular filtrate so that it is not lost from the body

خلايا الكلية (خاصة الأنابيب الكلوية) بتفرز $H^+$ إلى داخل البول (الأنبوب).

هاي $H^+$ بتتفاعل مع $HCO_3^-$ اللي نزل مع البول من الدم. $H^+ + HCO_3^- \rightarrow H_2CO_3$

فينتج عنهم $H_2O + CO_2$ بعدين $CO_2$ يرجع يدخل للدم ويتحول مرة ثانية إلى $HCO_3^-$.

الهدف: ما نخسر (أو $HCO_3^-$) من bicarbonate من الجسم.

بالعربي: الكلية ترجع أو $HCO_3^-$ بدل ما يضيع مع البول.

- When all bicarbonate has been recovered, any deficit due to the buffering process is regenerated.

- The mechanisms for bicarbonate recovery and for bicarbonate regeneration are very similar and sometimes confused.

الكلية أولاً بتسترجع الـ $HCO_3^-$ اللي نزل باليول.

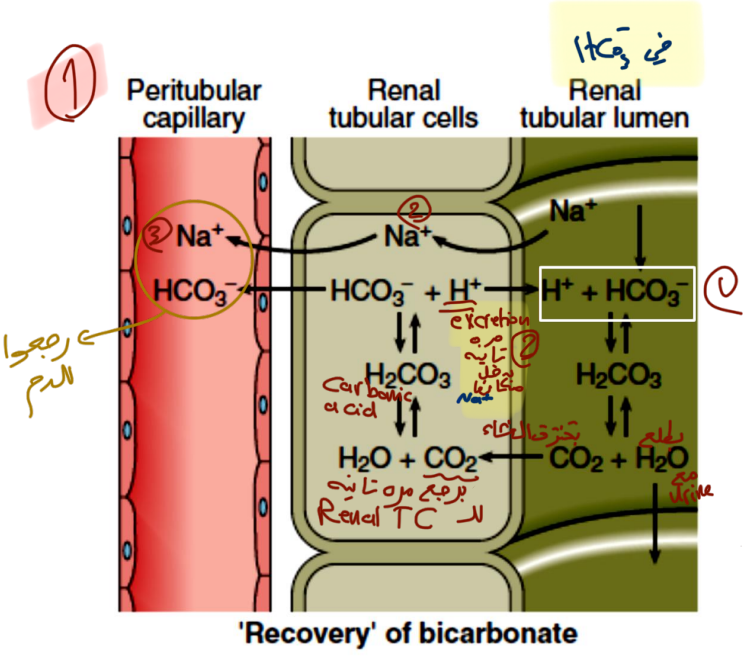
بعدين تصنع $HCO_3^-$ جديد إذا في نقص بالدم.

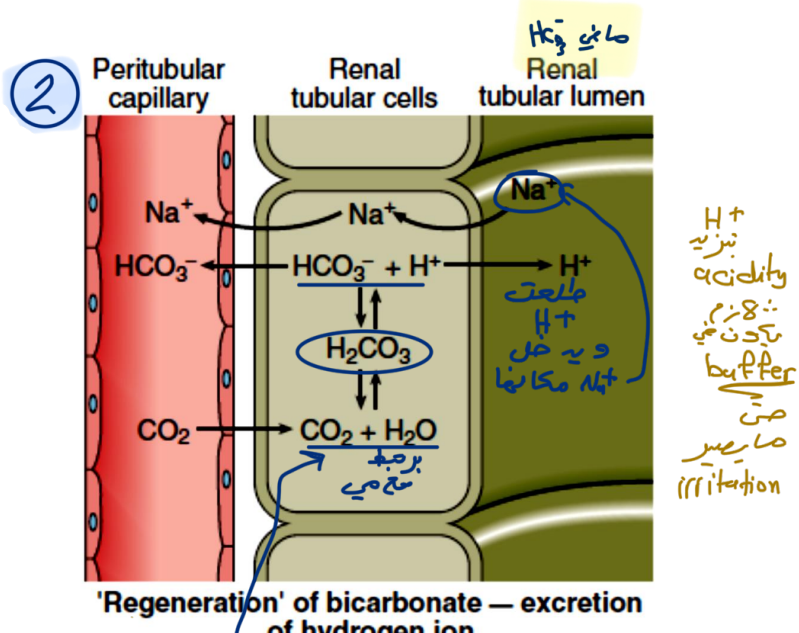
والـ $H^+$ اللي طلعته باليول بتعادل مع الفوسفات أو الأمونيا حتى ما يصير اليول حمضي جدًا.

- The excreted H+ must be buffered in urine or the [H+] would rise to very high levels, phosphate acts as one such buffer, while ammonia is another

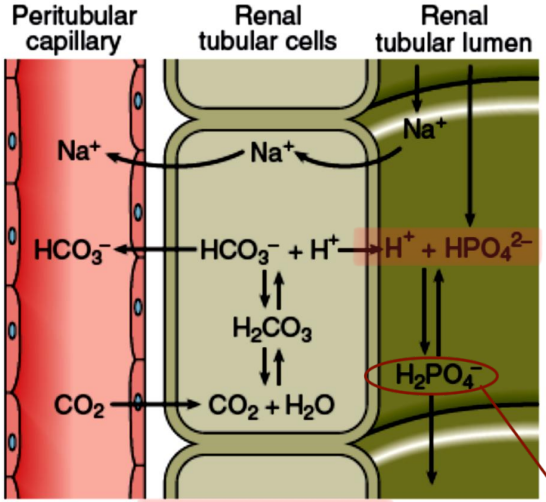
بالـ kidney في [Na⁺ – H⁺ pump] بتطلع H⁺ وبترجع الصوديوم Na⁺ والـ HCO₃⁻ رح يرجعوا الدم

# H+ excretion in the kidney

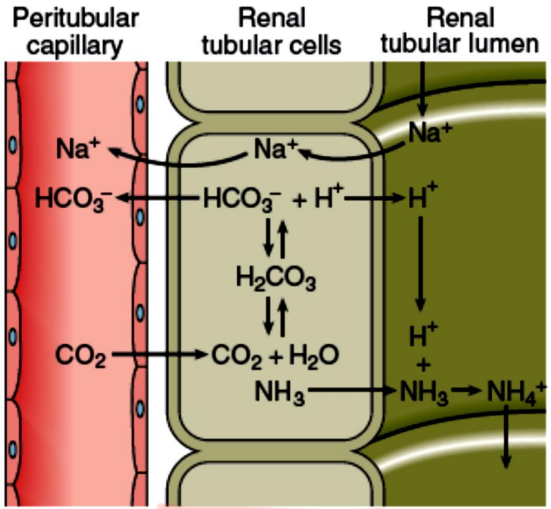


# H+ excretion in the kidney




هيك رح نتخلص من H+ مع الفوسفات buffered H+ بسيطة




هون رح ترتبط H+ مع ammonia [NH3] فرح تطلع مع Urine بدون ما تعمل irritation للقناة البولية

# Assessing status

- The carbonic acid ($H_2CO_3$) component is proportional to carbon dioxide, which is in turn proportional to the partial pressure of the $CO_2$
- Because the body's cellular and metabolic activities are pH dependent, the body tries to restore acid-base homeostasis whenever an imbalance occurs (**Compensation**)
- The body accomplishes this by altering the factor not primarily affected by the pathologic process. For example, if the imbalance is of **non-respiratory** origin, the body compensates by altering **ventilation** (fast response).
- For disturbances of the **respiratory** components. The **kidneys compensate** by selectively excreting or reabsorbing anions and cations. The kidneys are slower to respond (2-4 days)

# Assessing status

- The H concentration in blood varies as the bicarbonate concentration and pCO2 change. If everything else remains constant.

- Adding H, removing bicarbonate or increasing the pCO2 will all increase [H+]

زيادة Acidity

- Removing H, adding bicarbonate or lowering pCO2 will all cause the [H+] to fall.

1 2 3

- An indication of the acid base status of the patient can be obtained by measuring the components of the bicarbonate buffer system

القيم الطبيعية لـ pH

# Normal ranges

**TABLE 16-1 ARTERIAL BLOOD GAS REFERENCE RANGE AT 37°C**

| | |
|---|---|
| pH | 7.35–7.45 |
| $pCO_2$ (mm Hg) | 35–45 |
| $HCO_3^-$ (mmol/L) | 22–26 |
| Total $CO_2$ content (mmol/L) | 23–27 |
| $pO_2$ (mmol/L) | 80–110 |
| $SO_2$ (%) | >95 |
| $O_2Hb$ (%) | >95 |

# Causes of metabolic acidosis

- Metabolic acidosis with an **elevated anion gap** occurs in:
- **Renal disease.** Hydrogen ions are retained along with anions such as sulphate and phosphate.
- **Diabetic ketoacidosis.** Altered metabolism of fatty acids, as a consequence of lack of insulin causes endogenous production of acetoacetic and β–hydroxybutyric acids
- **Lactic acidosis.** Particularly tissue anoxia. In acute hypoxic states such as respiratory failure or cardiac arrest. It can be caused by liver disease. The presence of lactic acidosis can be confirmed by the measurement of plasma lactate concentration.
- Certain disease of overdosage or **poisoning**. As in **salicylate** overdose where build-up of lactate occurs, or **methanol** poisoning when formate accumulates, or **ethylene glycol** poisoning where oxalate is formed.

$HCO_3^-$ بطلع من الجسم فبحاول الجسم يعوضه بـ $Cl^-$

# Causes of metabolic acidosis

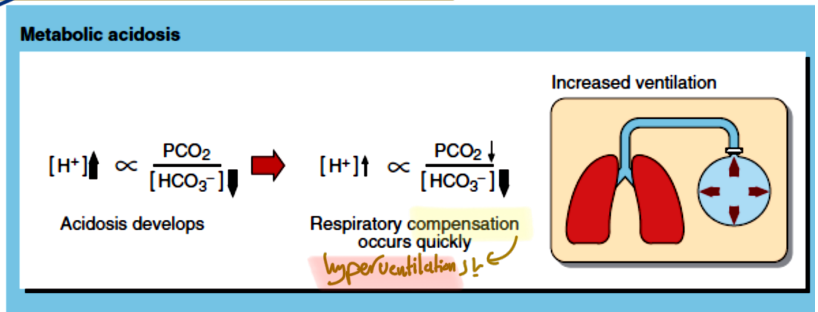
- Metabolic acidosis with a normal anion gap is sometimes referred to as **hyperchloraemic acidosis** because a reduced HCO3 is balanced by increased Cl concentration. It is seen in chronic diarrhea or intestinal fistula. Fluids containing bicarbonate are lost from the body

الكلى ما بتقدر تطلع $H^+$

- **Renal tubular acidosis**: Renal tubular cells are unable to excrete hydrogen ions efficiently and bicarbonate is lost in urine

يتراكم الـ $H^+$ في الدم ويقل الـ $HCO_3^-$ ← فيصير الدم حامضي (metabolic acidosis)

Urine بطلع صح $HCO_3^-$ فالجسم مش قادر يسترجعه




في RTA:
الخلايا الأنبوبية فيها خلل، سواء في مضخات $H^+$ أو في آلية إعادة امتصاص $HCO_3^-$.
نتيجة لذلك: الهيدروجين ما بطرح بكفاءة ← يضل في الجسم.
والبيكربونات ما تستعاد للدم بل تخرج مع البول ← الجسم يخسر القاعدة

# Clinical effect of acidosis

- The compensatory response to metabolic acidosis is hyperventilation, since the **increased [H+]** acts as a powerful stimulant of the respiratory centre.

- The deep rapid and gasping respiratory pattern is known as Kussmaul breathing. Hyperventilation is the appropriate physiological response to acidosis and it occurs rapidly.

- The raised [H+] leads to increased neuromuscular irritability. There is a hazard of arrhythmia progressing to cardiac arrest and this is more likely by the presence of hyperkalemia, which will accompany the acidosis.

- Depression of consciousness can progress to coma and death