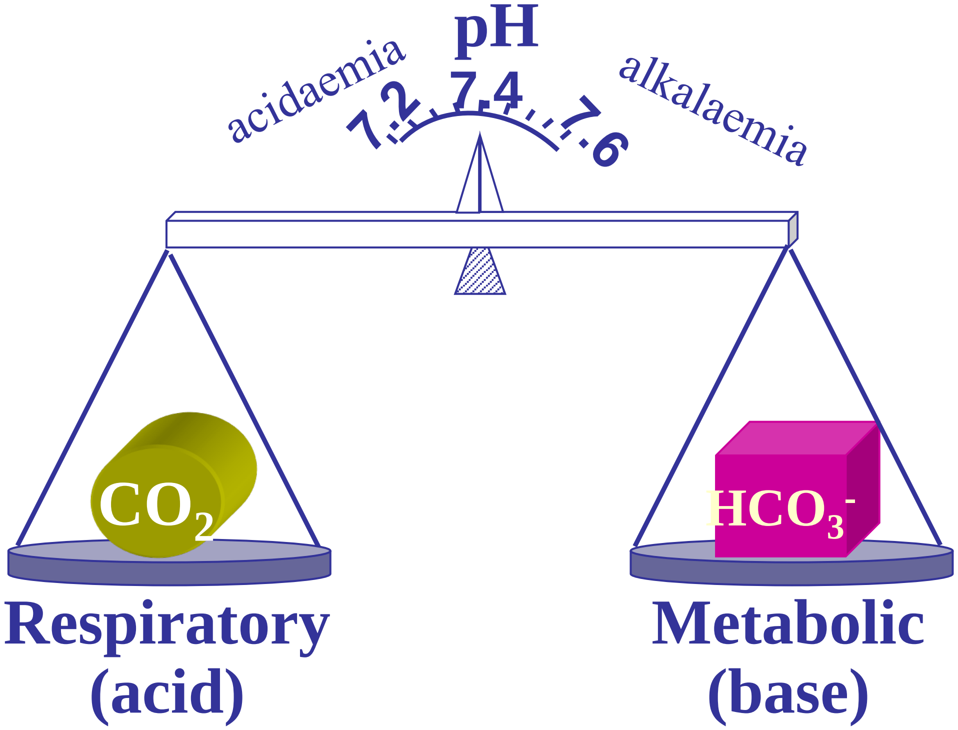
Acid-Base Balance



Outline

- H-H equation
- Interpretation
- Sample collection & pre analytical errors
- Respiratory Acidosis
- Respiratory Alkalosis
- Metabolic Acidosis
- Metabolic Alkalosis
- Mixed disturbances

Henderson Hasselbalch equation

$$H^+ + HCO_3^- \longleftrightarrow H_2CO_3 \longleftrightarrow H_2O + CO_2$$

$$pH = 6.1 + \log_{10} \frac{[HCO_3]}{pCO_2 \times 0.23}$$

Relationship of pH and H^+

pH **$[H^+]$**

$$pH = -\log_{10} [H+]$$

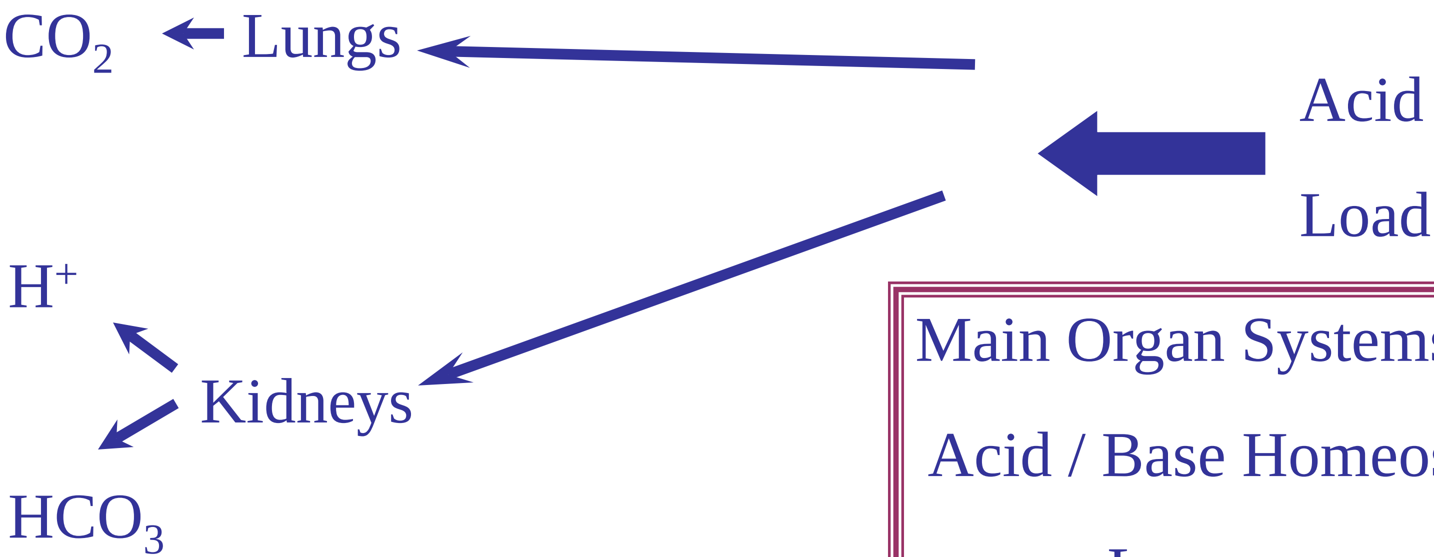
pH 6.0	[H+] = 1000 nmol/L
pH 7.0	[H+] = 100 nmol/L
pH 7.35	[H+] = 45 nmol/L
pH 7.45	[H+] = 35 nmol/L
pH 8.0	[H+] = 10 nmol/L

Main Buffers

Buffer	Equation	RBC	[Conc]	% Total
Hb	$H^+ + Hb^- \longrightarrow HHb$	40	53 mmol/L	
HCO3	$CO_2 + H2O \longrightarrow H_2CO3 \longrightarrow H^+ + HCO_{3-}$	1	25 mmol/L	60
PO4	$H_2PO_4 \longrightarrow H^+ + HPO4$	0.3	1.0 mmol/L	Low
Protein	$H^+ + Pr^- \longrightarrow HPr$	8	7.7 mmol/L	Low

RBC = relative buffering capacity

What happens when we get an increased acid load?



Compensation

Main Organ Systems Involved in Acid / Base Homeostasis.

- Lungs
- GI tract
- Kidney

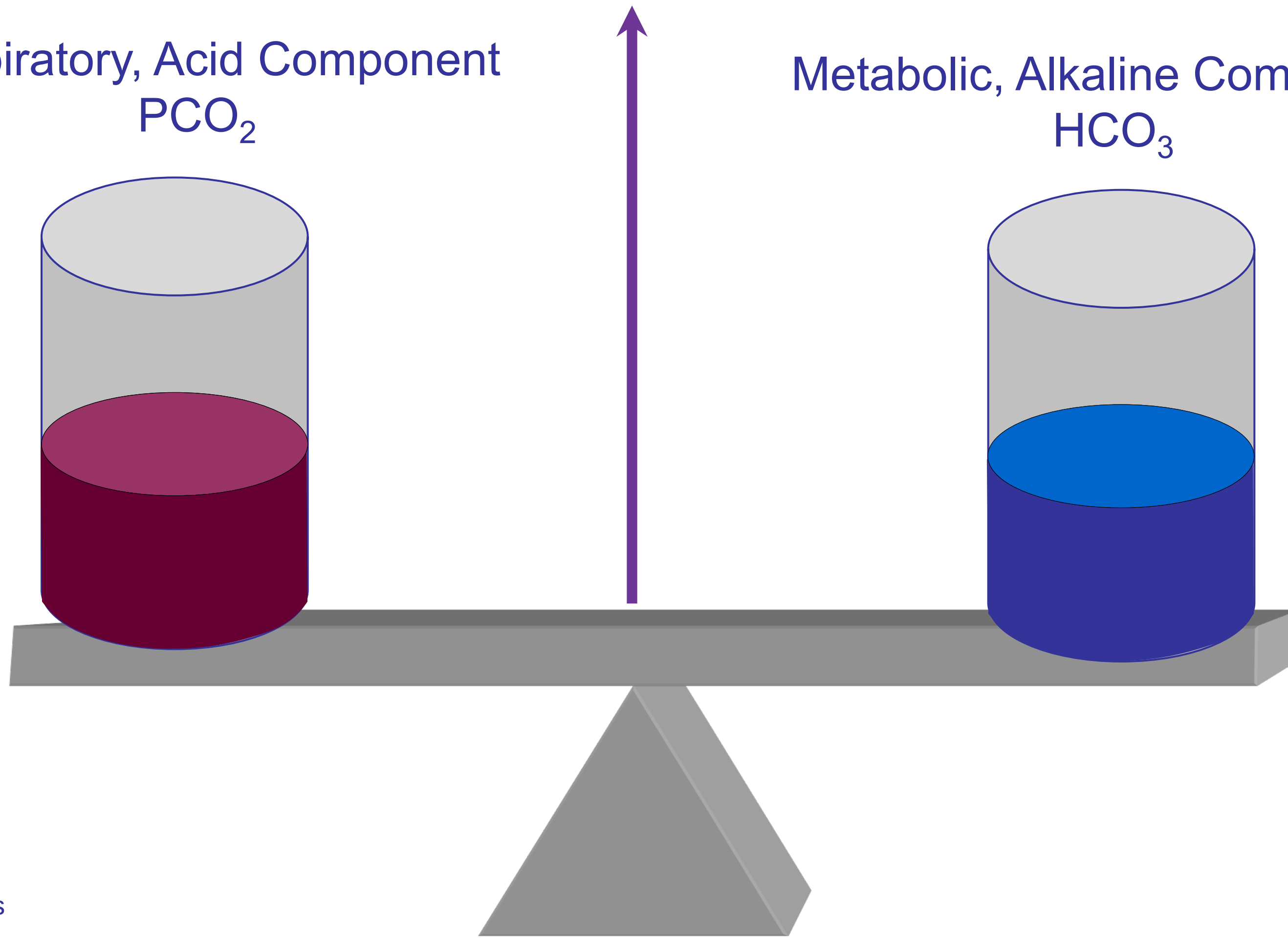
Ronda Greaves

Interpretation

pH µ $\frac{[HCO_3^-]}{PCO_2}$

The Acid-Base Balance

Acidosis Alkalosis



Respiratory

Choking

Broncho - pneumonia

COAD

A C I D O S I S

A L K A L O S I S

Hysterical overbreathing

Mechanical over ventilation

Raised intracranial pressure

Metabolic

Impaired H^+ excretion →	ACIDOSIS	ALKALOSIS	← Loss of H^+ in vomit
Increase H^+ production or ingestion →			← Alkali Ingestion
Loss of HCO_3^- →			← Potassium deficiency

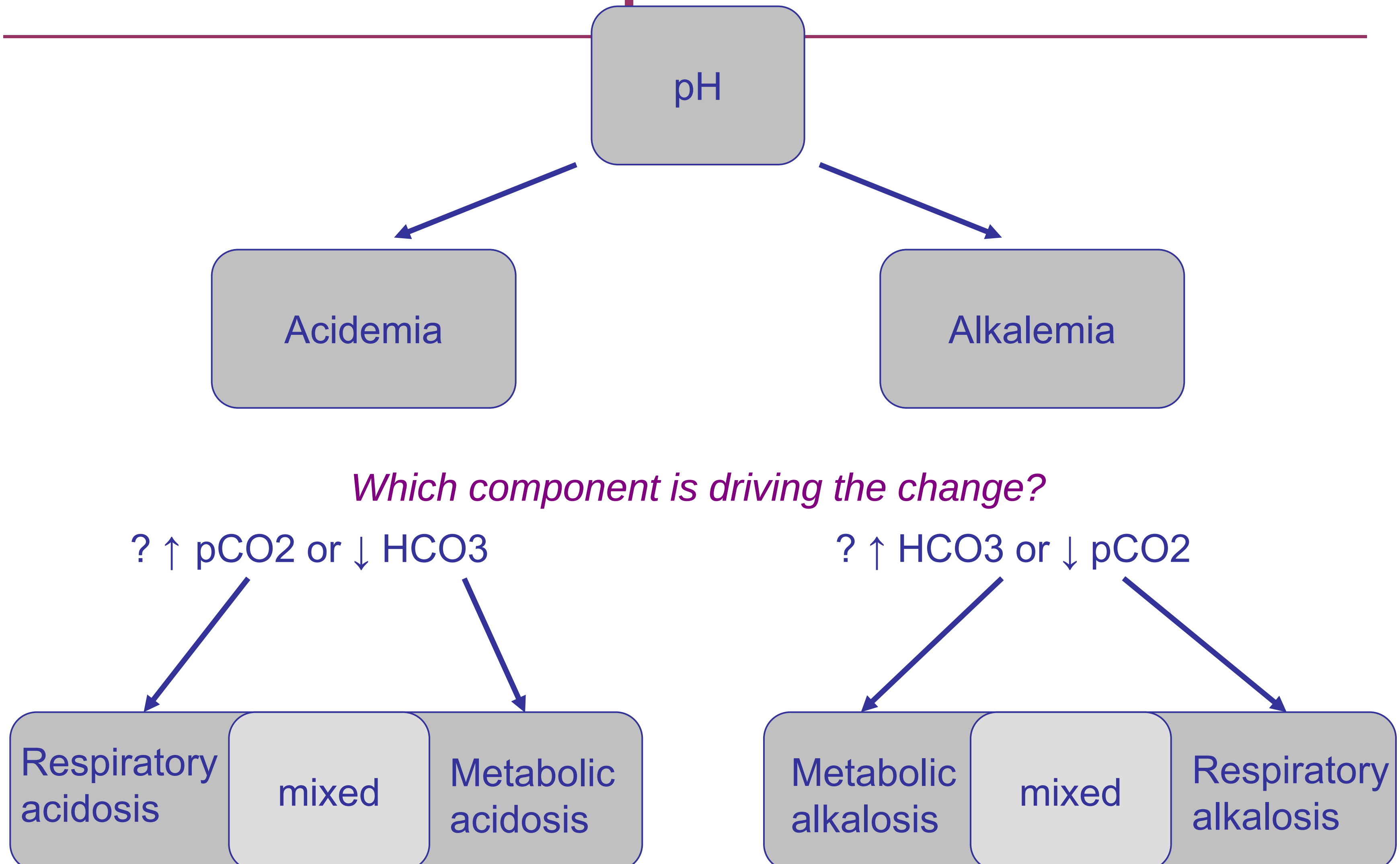
COMPENSATION LIMITS

- Primary disorders are corrected by compensation using the non-disordered component

- Renal compensation for abnormal pCO_2 :
  - HCO_3^- can fall to 10 if pCO_2 low
  - HCO_3^- can rise to 45 if pCO_2 high
  - max compensation 2-4 days

- Respiratory compensation for abnormal HCO_3^- :
  - pCO_2 can fall to 10 if HCO_3^- low
  - pCO_2 can rise to 60 if HCO_3^- high
  - max compensation 12-24 hours

Interpretation



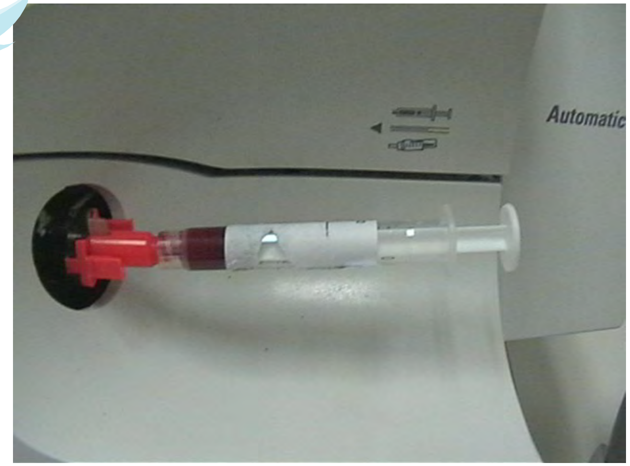
Case Study 1: Normal

pH	7.40	(7.35 - 7.45)	
pCO_2	40	(35 - 45)	mmHg
pO_2	90	(75 – 100)	mmHg
HCO_3	25	(21 - 30)	mmol/L
Lactate	1.0	(0.2 - 1.8)	mmol/L
Ketones	Neg		
Glucose	5.0	(3.6 - 7.7)	mmol/L
Creatinine	0.09	(0.07 - 0.11)	mmol/L
Urea	5.0	(2.5 - 8.3)	mmol/L

Sample Collection & Pre analytical errors

Pre analysis!

- Transport on ice
- Analyse stat (<15 min)
- Avoid air bubbles



- False results from:
  - Delayed analysis
  - Choice of collection device
  - Air in syringe
  - Air bubbles on electrode

CLSI blood gas and pH analysis and related measurements Approved guidelines (C46-A2) 2009.

Case Study 2: Collection tube

- 16 month old boy
- ICU
- Previous suspicious results - ??lab error

- Blood gas collected
  - Two samples collected from arterial line
  - 1. Capillary tube
  - 2. Blood gas syringe with liquid heparin

Case Study 2: Collection tube

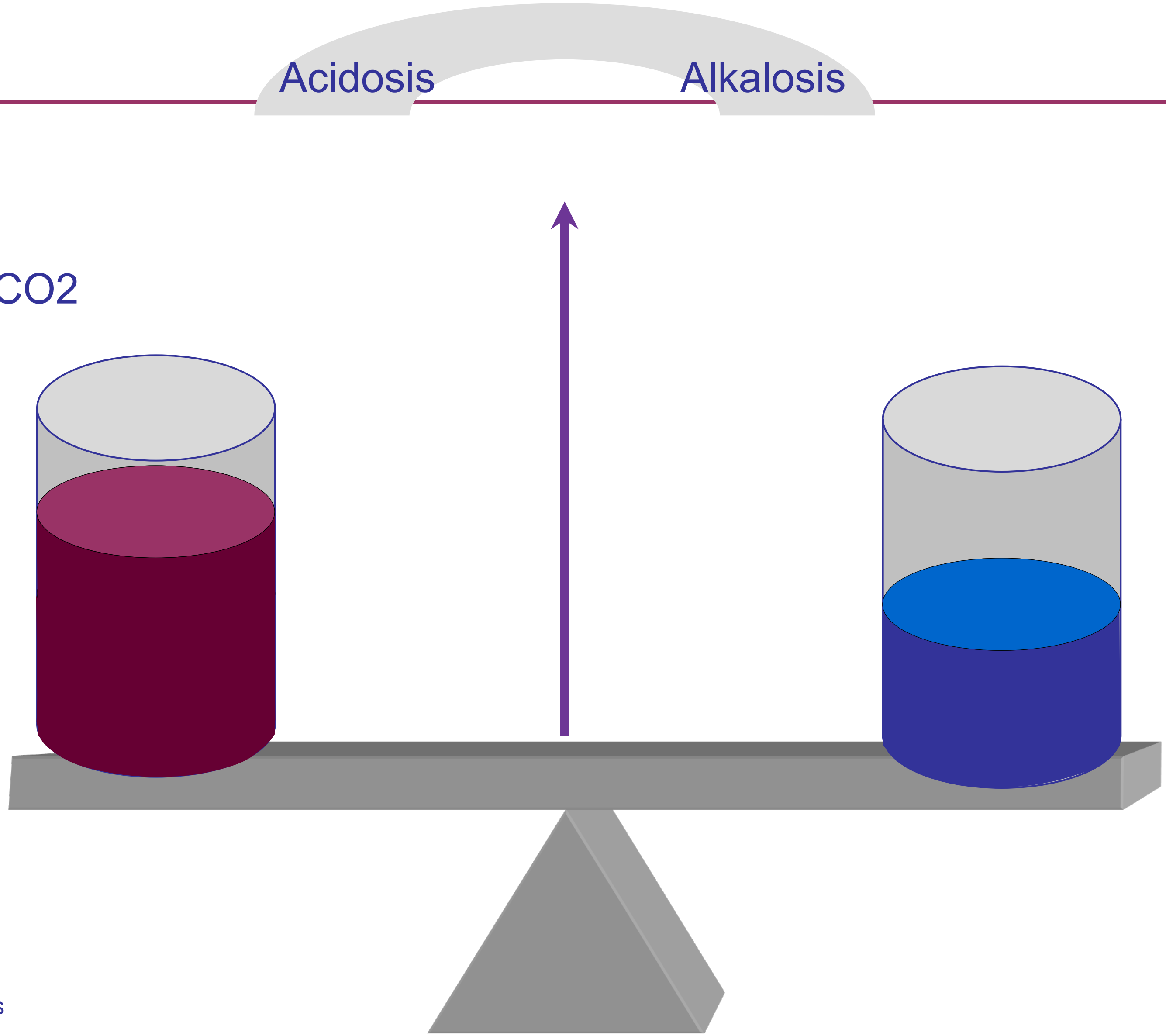
	Cap tube – analyser 1	Syringe – analyser 1	Syringe – analyser 2
pH (7.35-7.45)	7.49	7.41	7.41
pCO2 (35-45)	29	24	22.8
pO2 (80-100)	97	97	104.5
HCO3-	22.1	15.2	14.0
hct	36	21	
Ica++	0.79	<0.10	
Na+	135	137	
K+	2.6	1.3	
lactate	1.6	1.0	

Respiratory Acidosis

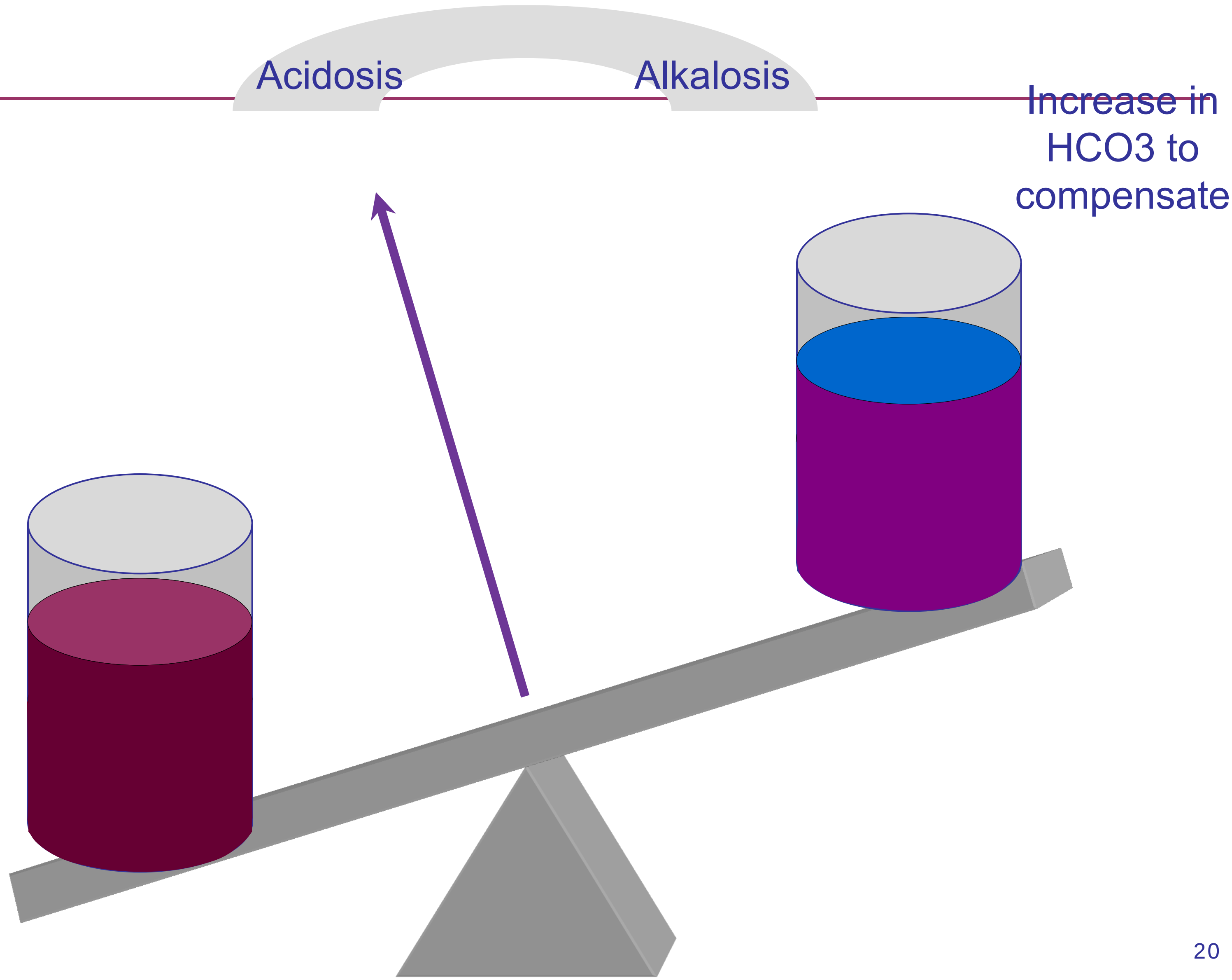
- **Lung disease**
- e.g. fibrosis, oedema, tumours, bronchitis, severe asthma, pulmonary embolism.
- **Mechanical**
- e.g. myopathies, trauma, pleural effusions, pneumothorax
- **Neurological**
- e.g. CNS depression (e.g. drugs) CNS disease

Respiratory Acidosis

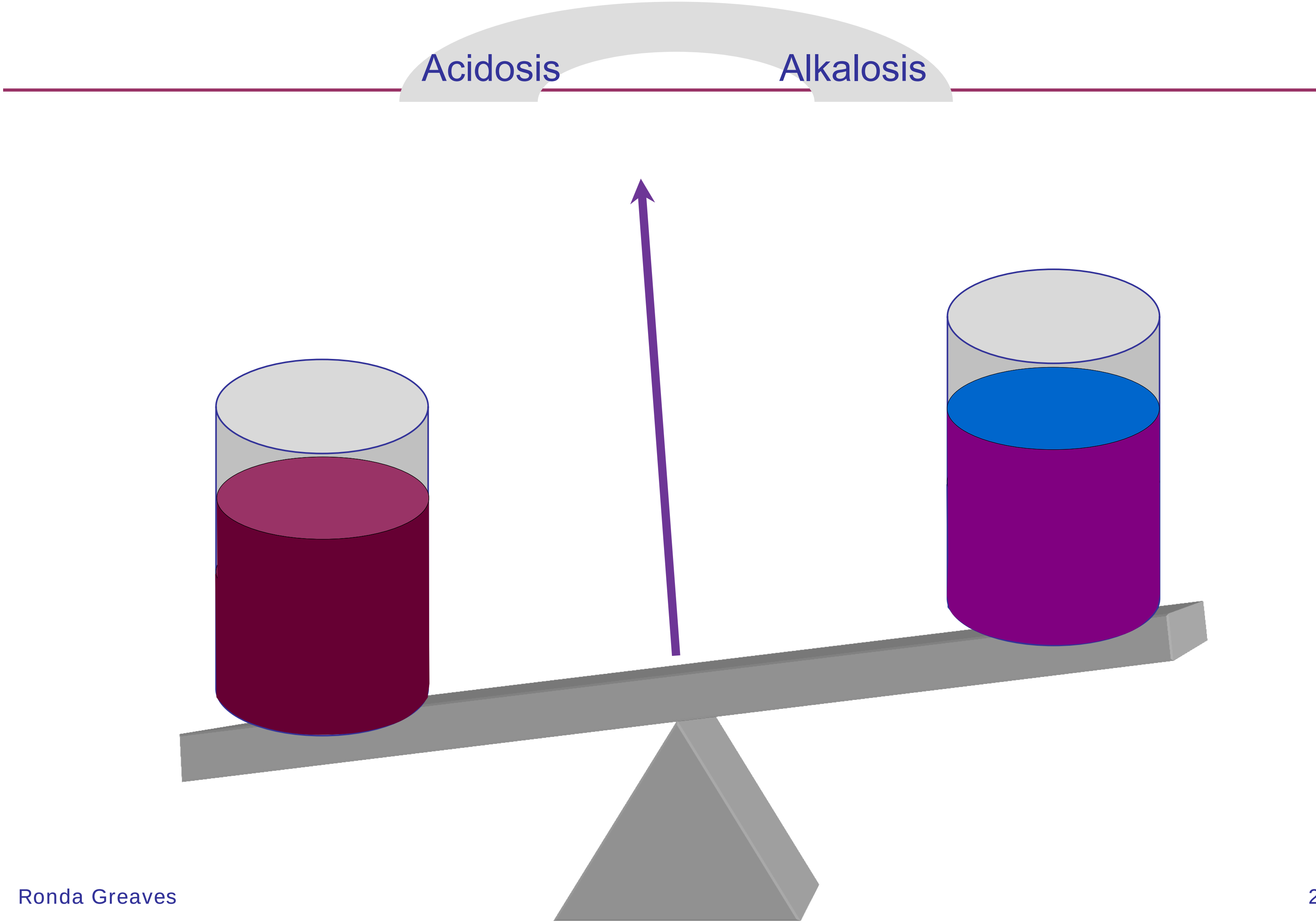
Increase in CO2



Respiratory Acidosis



1° Respiratory Acidosis compensated by 2° Metabolic Alkalosis



Case Study 3:
59 year old male with emphasema

pH	7.36		(7.35-7.45)
H^+	43	nmol/L	(35-45)
PCO_2	63	mm Hg	(35-45)
PO_2	52	mm Hg	(80.110)
HCO_3	35	mmol/L	(23-33)

Case Study 4: Infant Respiratory Distress in a newborn (Twin 1)

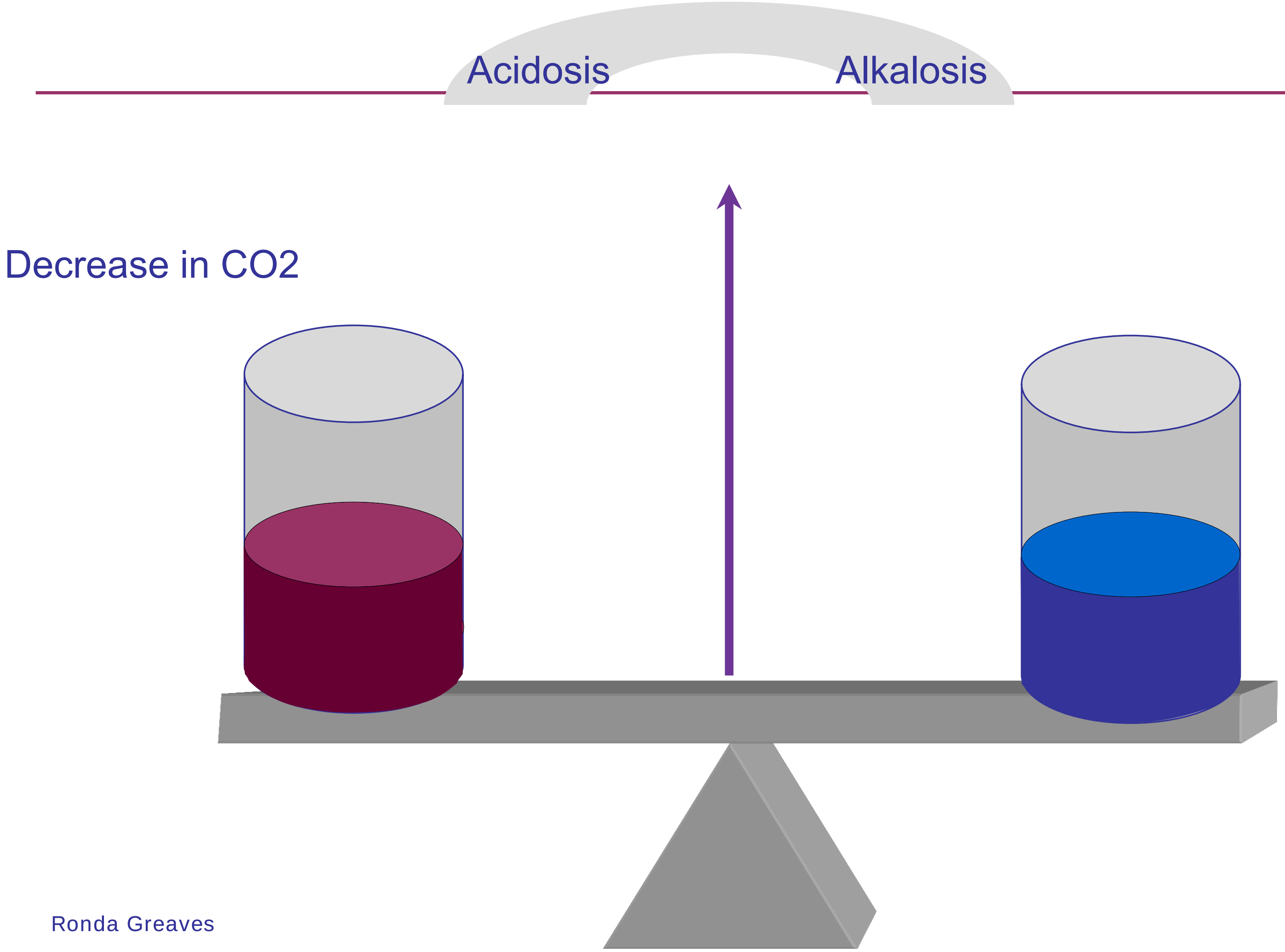
	Day 3 0500	Day 3 1345	Day 4 0220	Day 4 0800	RR
pH	7.36	7.29	7.25	7.21	7.32-7.46
pCO_2	36	49	54	64	31-42 mmHg
pO_2	38	45	50	44	55-105 mmHg
ABIC	20	23	23	25	20-26mmol/L
BE	-5	-4	-5	-4	-5-+5mmol/L

Is there compensation?

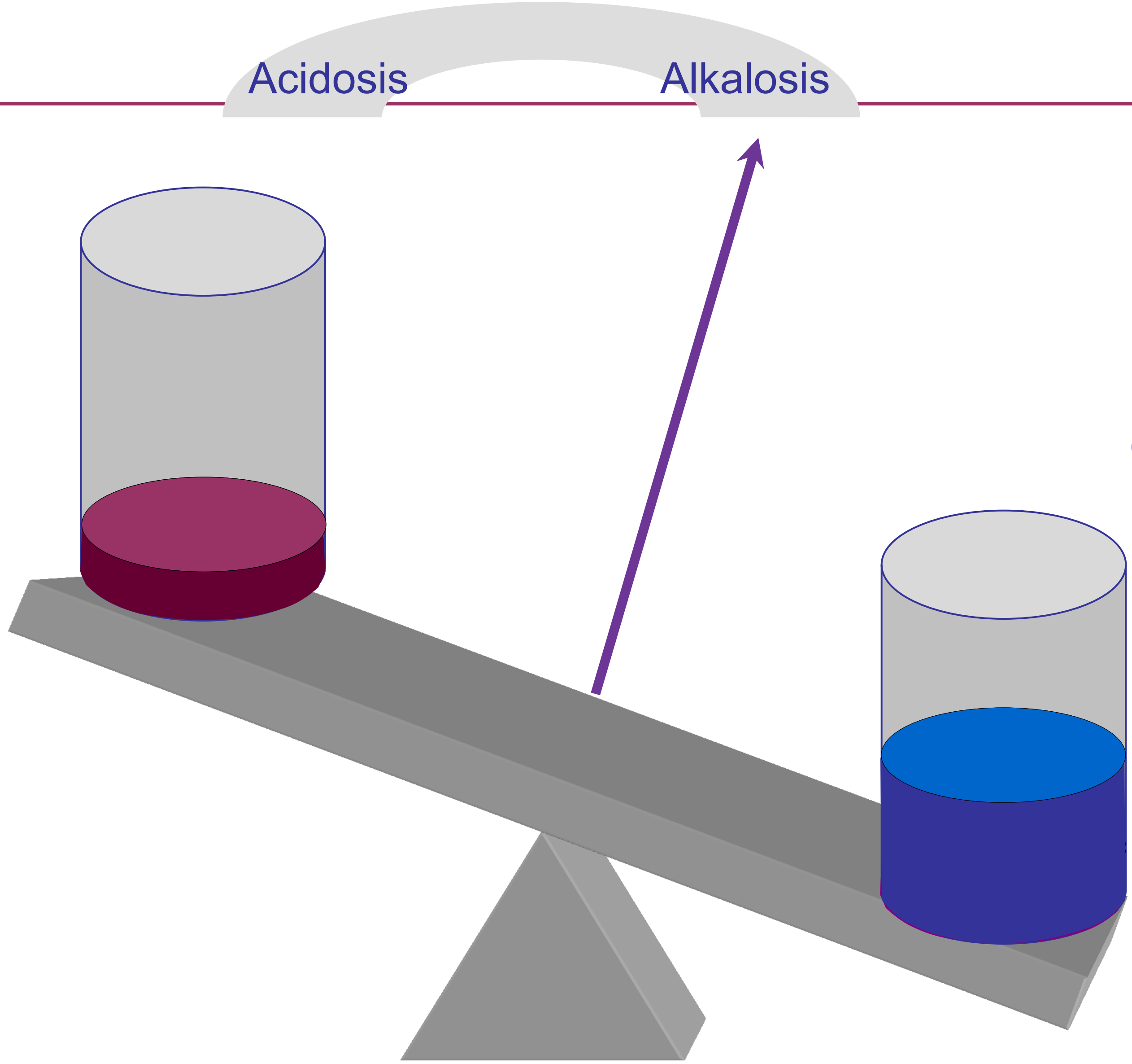
Respiratory Alkalosis

- **Hypoxia**
- Since oxygen diffuses less easily across the lungs than carbon dioxide, hyperventilation due to hypoxia can reduce pCO_2
- **Hyperventilation**
- e.g. anxiety/pain, CNS stimulation (e.g. salicylate OD)

Respiratory Alkalosis

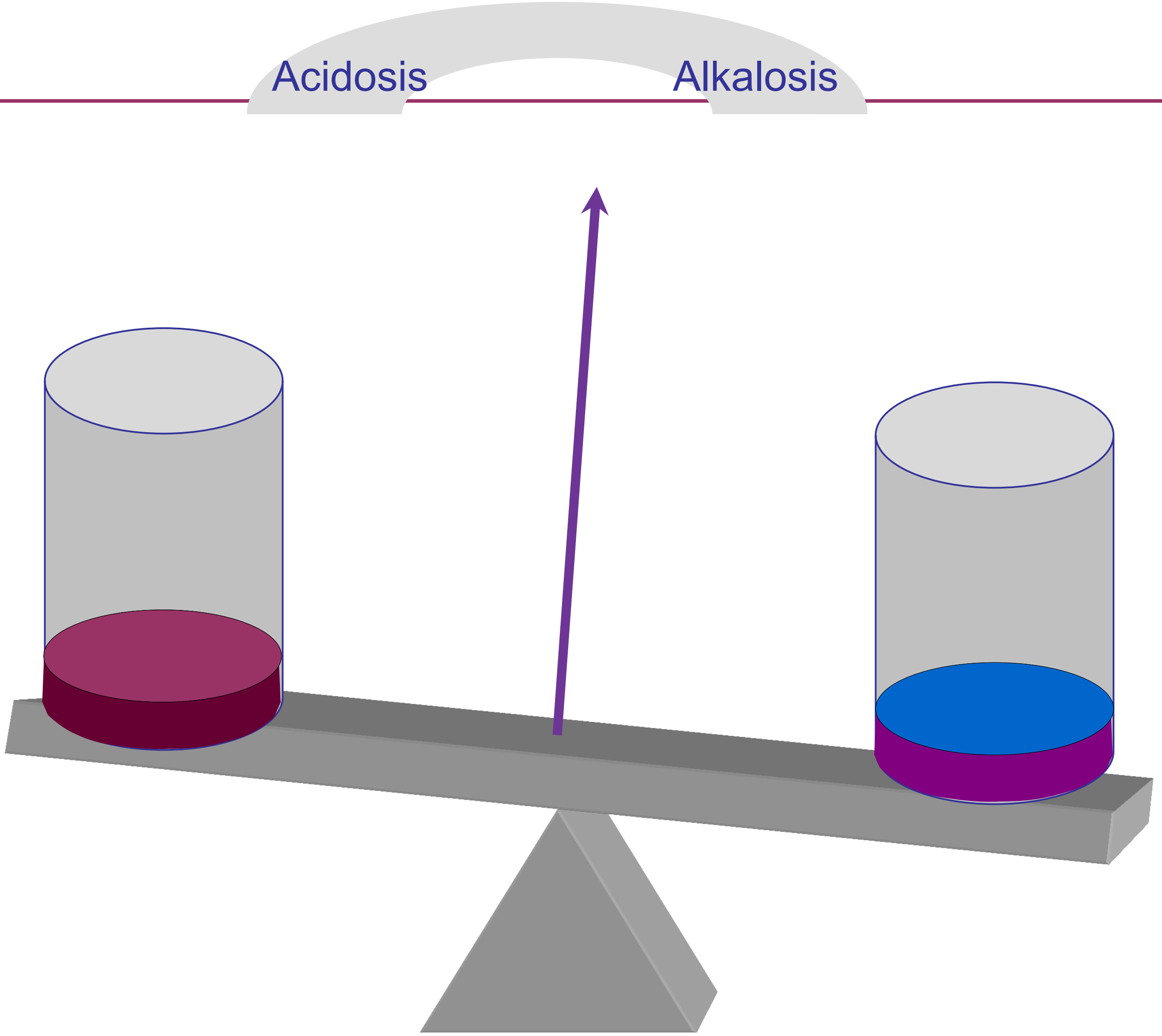


Respiratory Alkalosis



Decrease in HCO3 to compensate

1° Respiratory Alkalosis compensated by 2° Metabolic Acidosis



Case Study 5: 2 year old screaming during an arterial stab

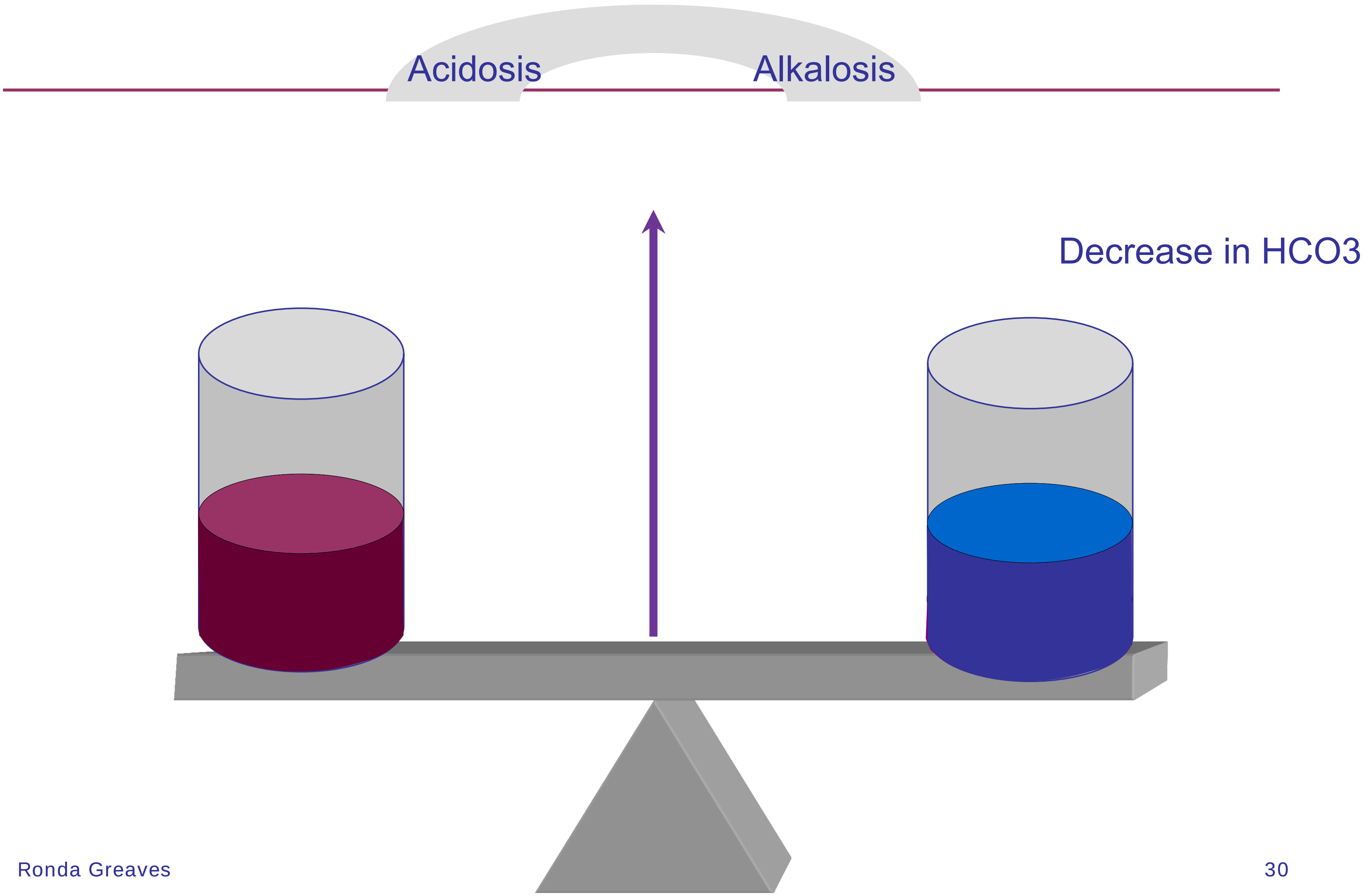
pH	7.47	(7.35 - 7.45)	
pCO_2	30	(35 - 45)	mmHg
pO_2	99	(75 - 100)	mmHg
HCO_3	28	(21 - 30)	mmol/L
Lactate	1.0	(0.2 - 1.8)	mmol/L
Ketones	Neg		
Glucose	5.0	(3.6 - 7.7)	mmol/L
Creatinine	0.04	(0.03 - 0.06)	mmol/L
Urea	5.0	(2.5 - 8.3)	mmol/L

Metabolic Acidosis

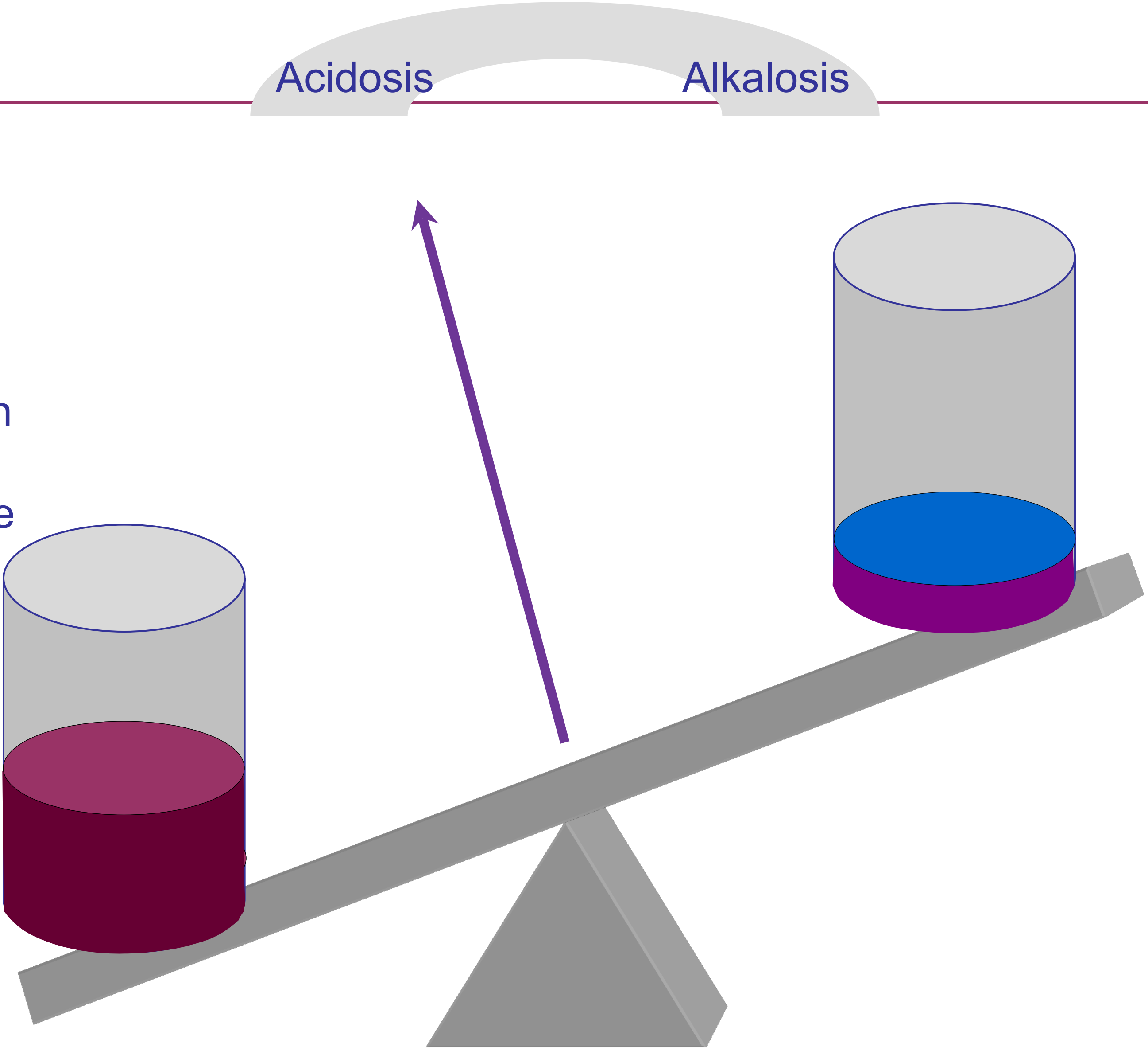
- **Increased rate of H+ production**
  - e.g diabetic ketoacidosis, lactic acidosis due to hypoxia
- **Decreased H+ excretion**
  - e.g. renal failure, renal tubular acidosis
- **Loss of bicarbonate**
  - e.g. diarrhoea, pancreatic fistula, renal tubular acidosis

Note: in newborns there is commonly a combined respiratory/metabolic acidosis

Metabolic Acidosis

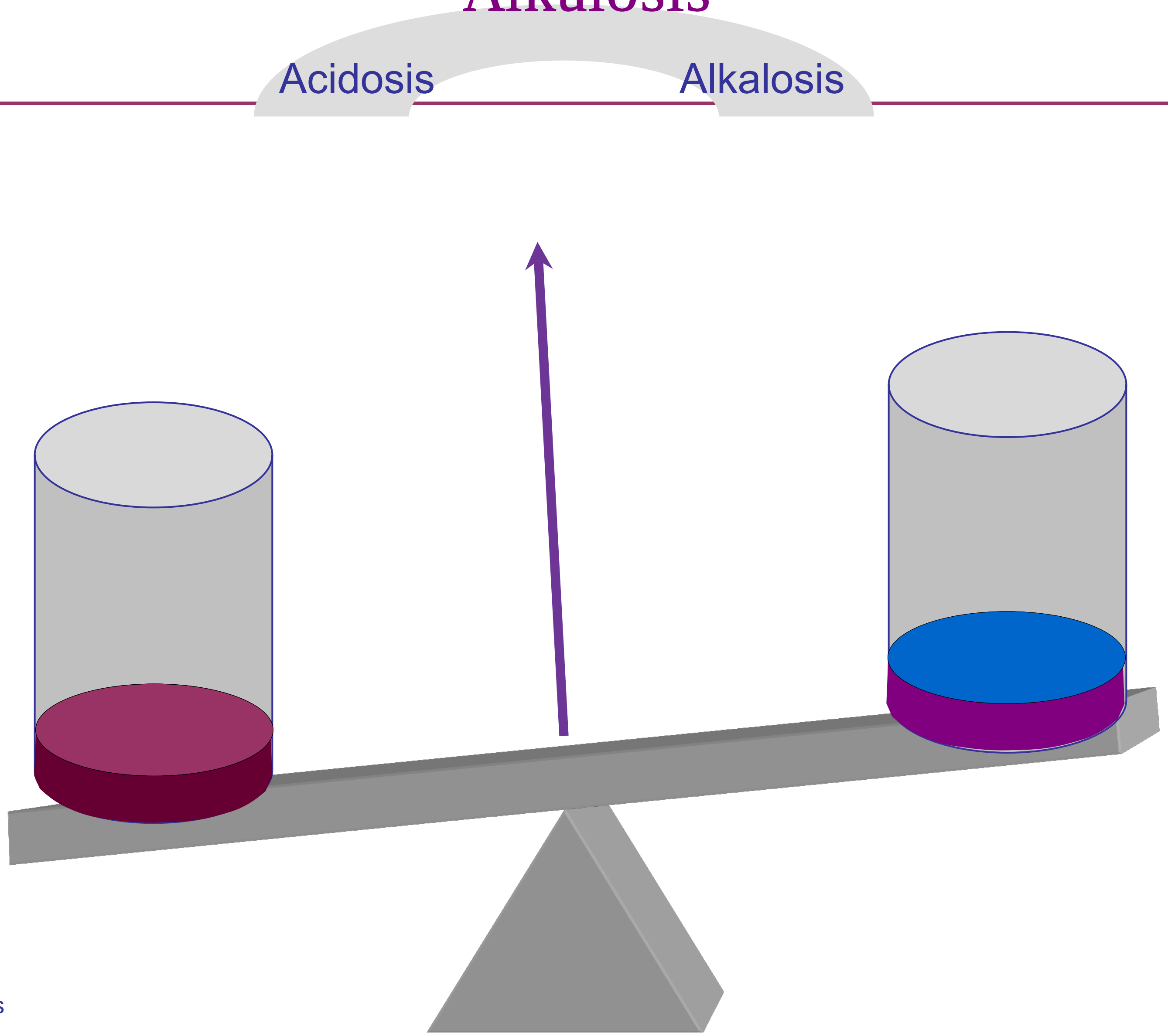


Metabolic Acidosis



Decrease in pCO2 to compensate

1° Metabolic Acidosis compensated by 2° Respiratory Alkalosis



Case Study 6: 45 year old female with chronic renal failure

	pH	7.28		(7.35-7.45)
	H^+	52	nmol/L	(35-45)
	PCO_2	26	mm Hg	(35-45)
	PO_2	100	mm Hg	(80-110)
	HCO_3	12	mmol/L	(23-33)
Plasma	Sodium	143	mmol/L	(137-145)
	Potassium	5.7	mmol/L	(3.1-4.2)
	Chloride	106	mmol/L	(98-106)
	Urea	75.0	mmol/L	(3.0-8.0)
	Creatinine	0.83	mmol/L	(0.05-0.12)

Once GFR falls below 20-30 ml/min (creat ~ 0.3-0.4 mmol/L), hyperkalaemia and metabolic acidosis develop. Below these values, other causes should be considered e.g. inc intake, aldosterone def

Case 7: A 4 year old child with diarrhoea and dehydration

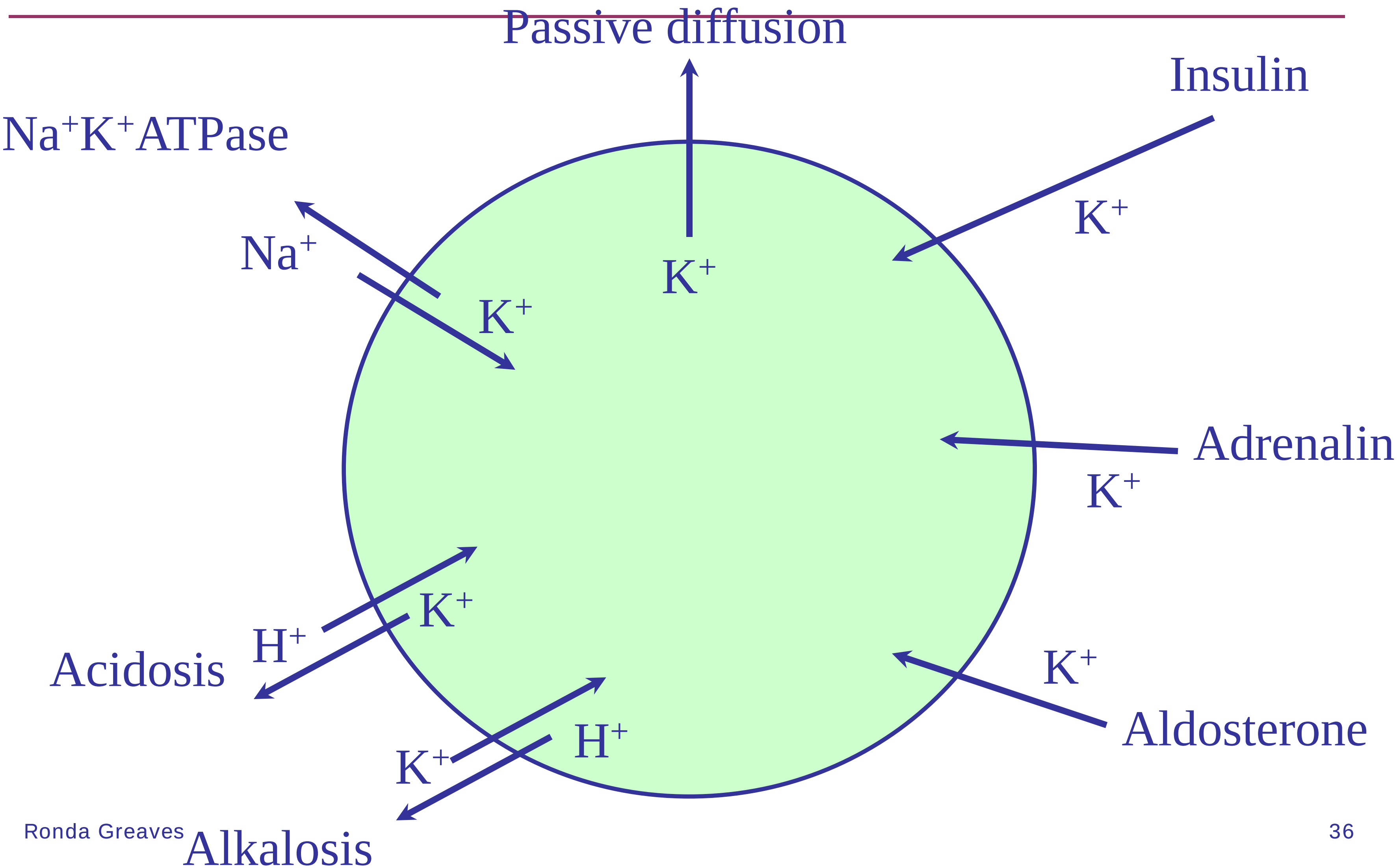
Plasma	Na	145	mmol/L	(132-144)
	K	1.9	mmol/L	(3.2-4.8)
	Cl	110	mmol/L	(98-108)
	HCO3	14	mmol/L	(23-33)
	Urea	3.2	mmol/L	(3.0-8.0)
	Creat	0.07	mmol/L	(0.06-0.12)
Urine	Na	<10	mmol/L	
	K	6	mmol/L	
	Cl	<5	mmol/L	

Diarrhoeal fluid contains large amounts of bicarbonate and potassium (metabolic acidosis). Extra-renal K loss with renal retention of Na and Cl

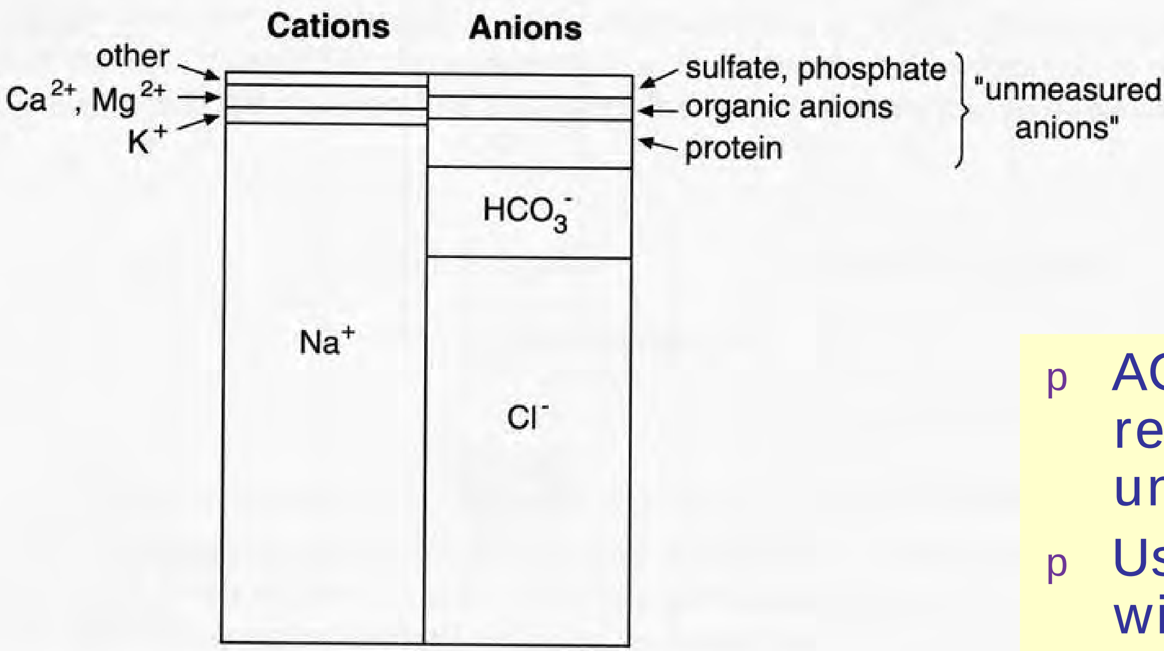
Case Study 8: DKA

pH	7.04	(7.35 - 7.45)	
pCO_2	7	(35 - 45)	mmHg
pO_2	125	(80 – 100)	mmHg
HCO_3	2	(21 - 28)	mmol/L
Sodium	141	(135-145)	mmol/L
Potassium	4.2	(3.5-5.0)	mmol/L
Chloride	106	(98-110)	mmol/L
Urea	10.9	(2.0 - 8.3)	mmol/L
Creatinine	0.09	(0.07 - 0.11)	mmol/L
Lactate	1.0	(0.2-1.8)	mmol/L
Ketones	POS		
Glucose	25.0	(3.6 – 7.7)	mmol/L

The cell wall



Anion Gap



Anion Gap = Na + K – Cl – HCO3$^-$

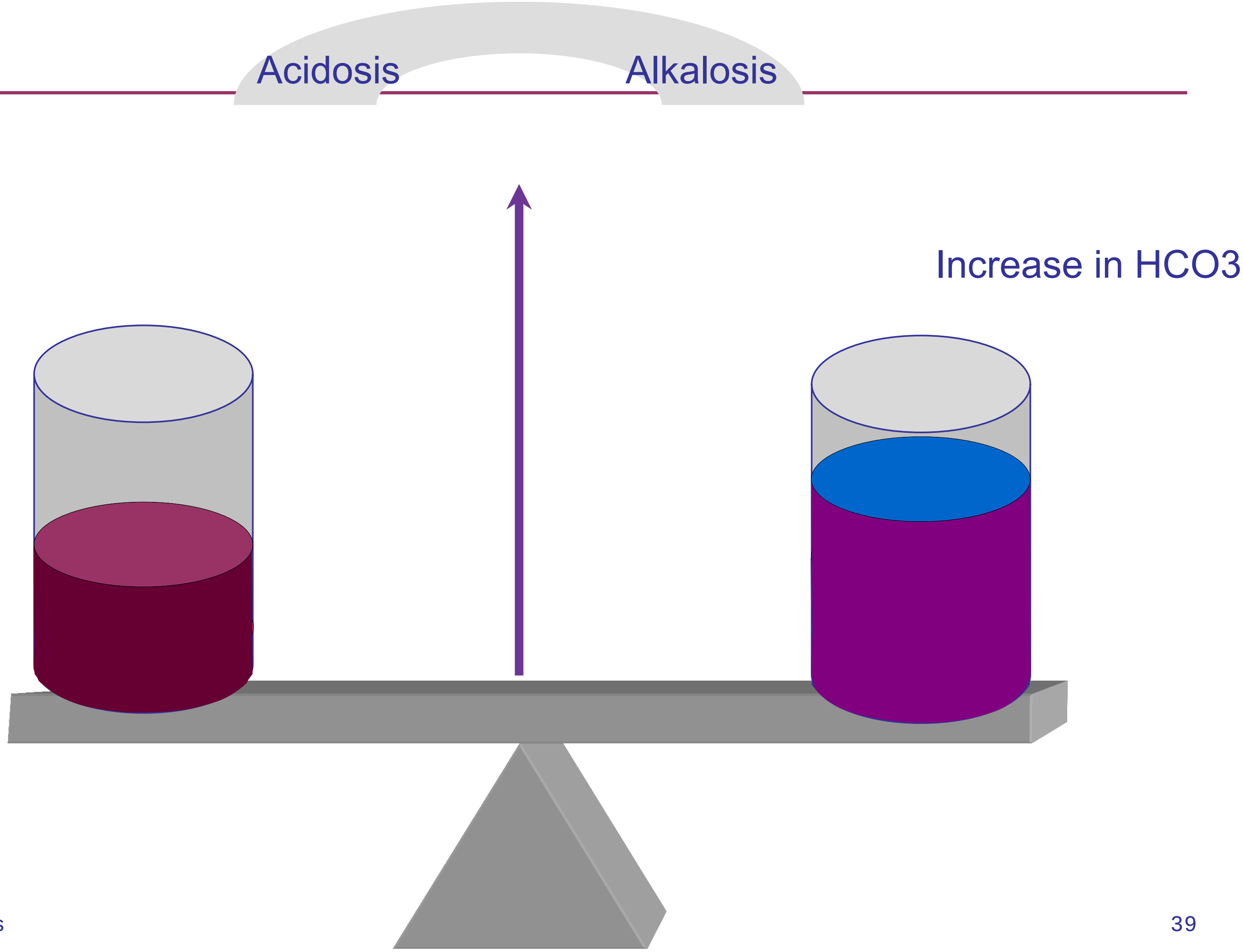
37 = (141 + 4.2) – (106 +2)

- AG is a measure of the relative abundance of unmeasured anions.
- Used to evaluate patients with metabolic acidosis.
- The result should be between about 12 – 20 mmol/L of unmeasured anions.

Metabolic Alkalosis

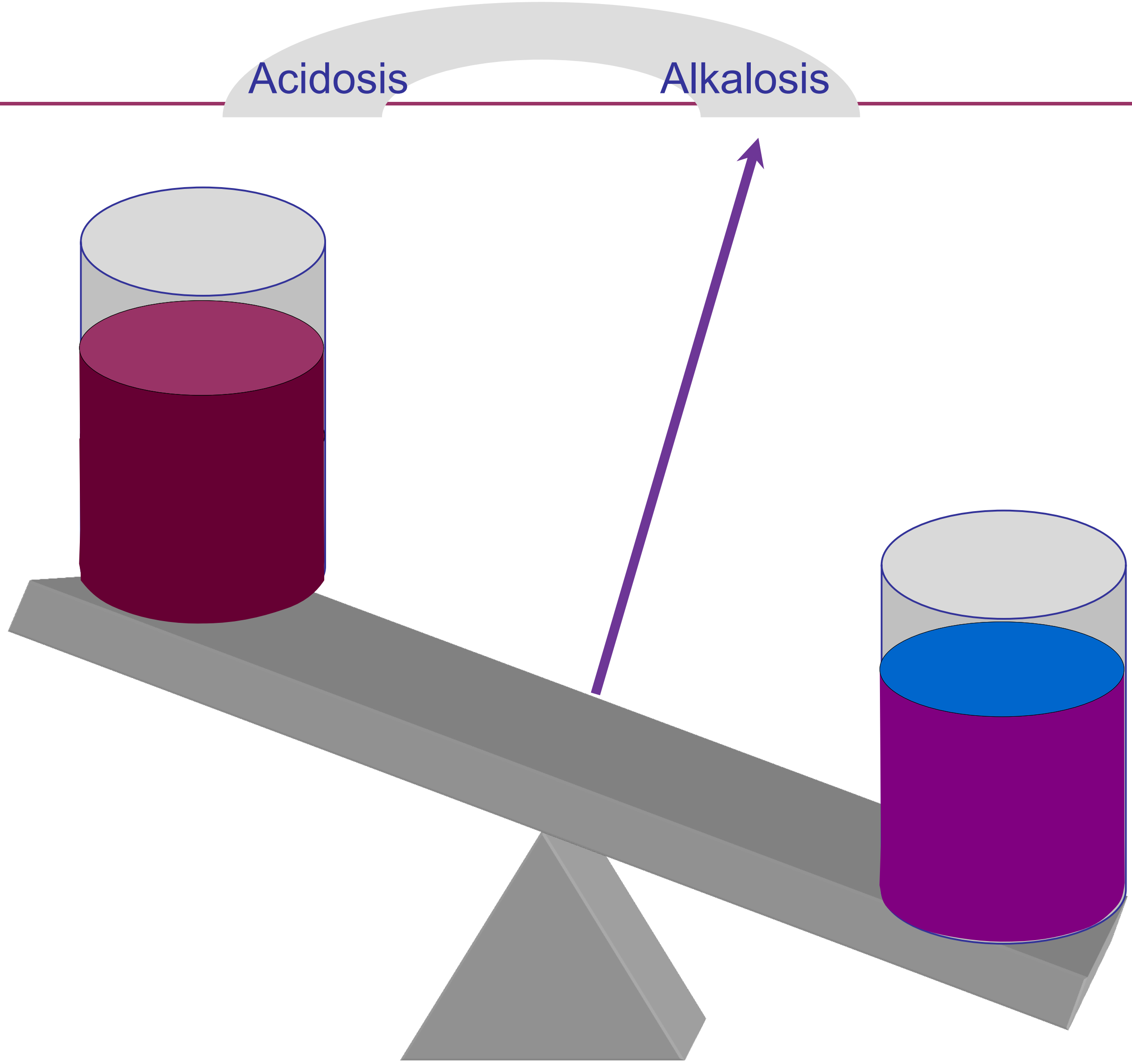
- Loss of H+
  - e.g. vomiting, potassium deficiency, hyperaldosteronism, excess alkali administration

Metabolic Alkalosis

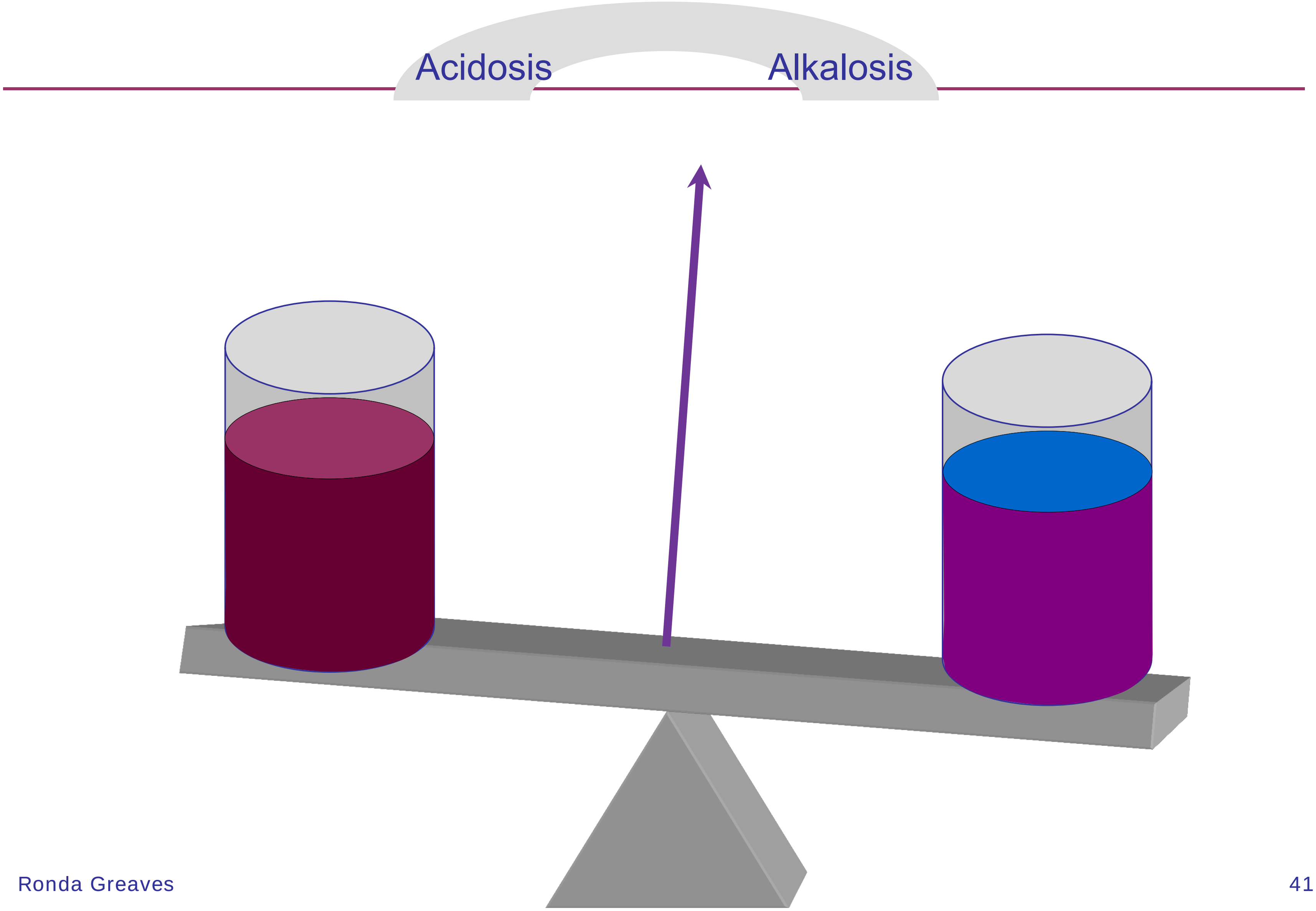


Metabolic Alkalosis

Increase in CO_2 to compensate



1° Metabolic Alkalosis compensated by 2° Respiratory Acidosis



Case Study 9: 6 week old boy presents to emergency department dehydrated and projectile vomiting

		initial	+1 d	+2 d	+3 d	RR
Na		133	132	139	138	135-145
K		3.3	4.6	6.2 H+	5.6	4.0-6.2
Cl		67	82	102	107	98-110
pH		7.54	7.60	7.49	7.45	7.34-7.43
pCO_2		60	36	36	40	32-45
ABIC		51.3	35.5	27.0	27.0	18.0-25.0
BE		>22.0	12.3	3.8	3.0	-4.0-+3.0
Urea		12.0	8.4	2.2	<1.0	1.7-6.7
Creat		0.07	0.04			0.01-0.03

Pyloric Obstruction

Constriction to the outlet of the stomach.

- Cause:
  - Contraction of an ulcer
  - Malignancy
  - Congenital abnormality.
- Symptoms:
  - Vomiting (often projectile)
  - Abdominal distension
  - Loss of HCl
- Biochemically:
  - severe hypochloreamic metabolic alkalosis.

- Commonest form of metabolic alkalosis in newborns is associated with Pyloric Stenosis
  - Congenital malformation of pyloric sphincter
  - “Projectile” vomiting at 3-4 weeks of age
  - Hypochloraemic metabolic alkalosis

Pyloric Stenosis

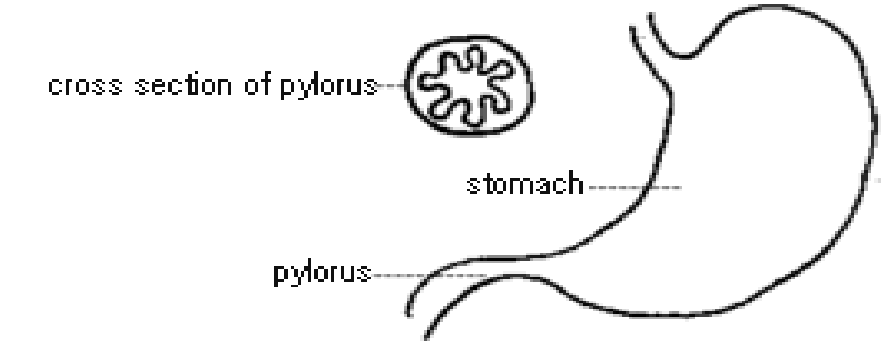


Diagram of normal stomach & pylorus.
Note the cross-section showing normal pyloric opening.

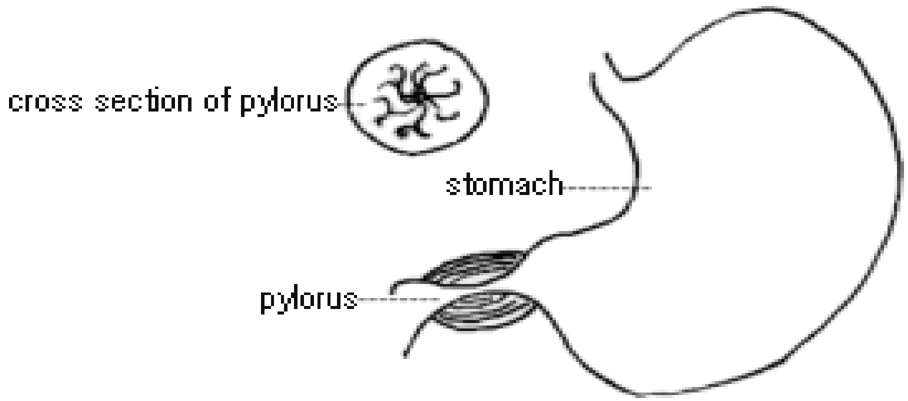


Diagram of stomach with pyloric stenosis.
Note the cross-section showing how the pyloric opening is very narrowed.

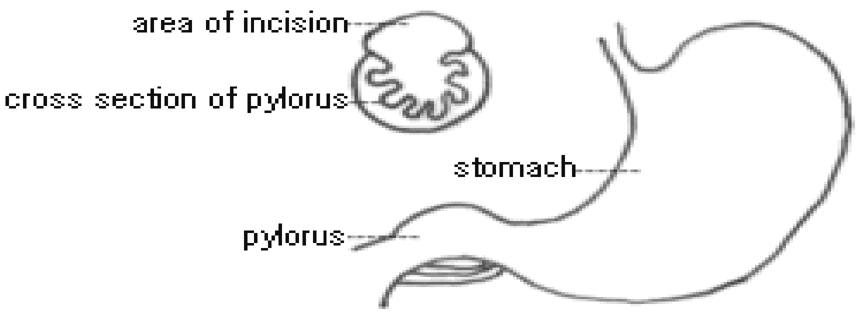


Diagram of stomach after repair of pyloric stenosis.
Note (in the cross-section) how an incision has been made in the muscle, enlarging the pylorus and relieving the obstruction.

http://www.pedisurg.com/PtEduc/Pyloric_Stenosis.htm

Diagnosis of Pyloric Stenosis

- Clinical Examination
- Radiology – Ultrasound &/or X-ray
- Laboratory – Blood gases and EUC's

Barium meal x-ray
Here is a very abnormal X-ray, showing a massively dilated, air-filled stomach, with very little bowel gas elsewhere. Suggests a high-grade gastric outlet obstruction.

http://www.vh.org/Providers/Lectures/PedsGIDisease/Case3.html

Mixed Disturbances

Case Study 10: Salicylate overdose - A 78 year old woman was admitted comatose after her daughter complained that her conscious state had deteriorated over the last few hours. 2 empty bottles of Red Flower Oil were found by her bedside. On examination, she was found to be tachypnoeic with a respiratory rate of 55.

pH	7.50	(7.35 - 7.45)	
pCO_2	17	(35 - 45)	mmHg
pO_2	103	(80 – 100)	mmHg
HCO_3	13	(21 - 28)	mmol/L
Sodium	152	(135-145)	mmol/L
Potassium	3.5	(3.5-5.0)	mmol/L
Chloride	118	(98 - 110)	mmol/L
Creatinine	0.12	(0.07 - 0.13)	mmol/L
Urea	13.0	(2.0 - 8.5)	mmol/L

Metabolic acidosis + Respiratory alkalosis

Case from Dr CS Ho – Hong Kong

Golden rules for interpretation

1. Follow the CLSI guidelines for collection and handling of samples
2. The patient's clinical history is the most important factor in determining the nature of the acid base disturbance.
3. Always start with pH to interpret blood gases i.e. acidosis or alkalosis
4. Only then look at the pCO_2 and bicarbonate i.e. respiratory or metabolic
5. Has compensation occurred?