

Neurological emergencies 1

Status epilepticus
Respiratory Failure in Neuromuscular Weakness

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Objectives

STATUS EPILEPTICUS

Initial investigations and components of management

including airway protection and use of anti-epileptic drugs

NEUROMUSCULAR EMERGENCIES

Clinical signs which point to neuromuscular ventilatory compromise

Bedside respiratory test of most use

Describe the findings on arterial blood gas which reflect type II respiratory failure

> As an F1 Recognise these emergencies, initiate management and call for expert help



Case



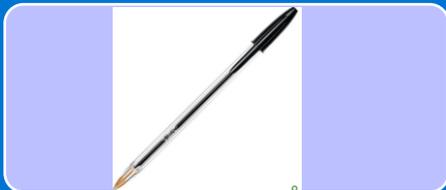
35 male on acute medical ward admitted overnight with seizures
Not yet fully assessed



It is 05.30 : 2 more tonic-clonic seizures lasting 6 minutes in last 30 minutes



Buccal midazolam 10mg given by nurse 10 minutes ago
He is GCS. E1 V2 M 5 You are called as F1



What do you think about as you arrive? Write down some key steps



Status epilepticus

Requires speedy systematic simultaneous assessment

ABCDE: Airway - Breathing - Circulation - Disability - Exposure

Termination of seizures

Search for cause

Follow the NUH protocol

New seizures **v** Status in known epilepsy has differences

Convulsive status epilepticus is the focus here; non-convulsive status more subtle and you have more time



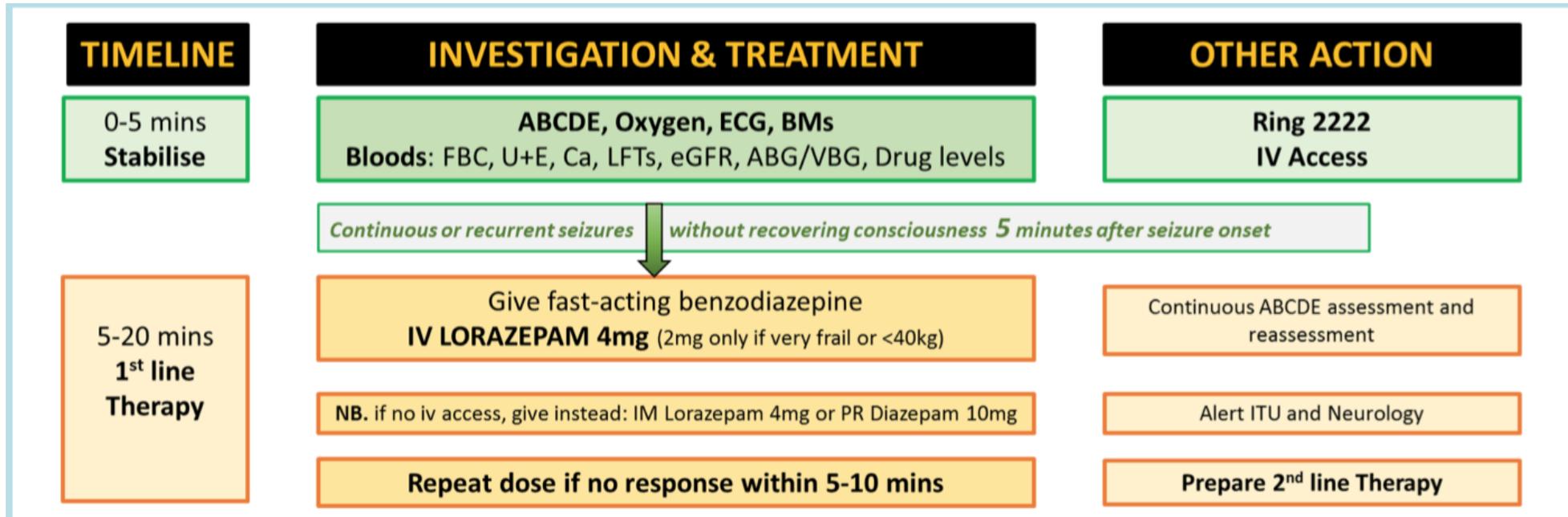
Step 1 - assume low GCS due to repeated seizures



1. Secure the airway - this may be obstructed due to low GCS (seizure or drugs)
2. If needed use nasopharyngeal / oropharyngeal airway (if GCS very low)
3. Give oxygen via mask
4. Measure Pulse and BP and O2 Saturation
5. IV access
6. Measure blood glucose
7. Send blood for Na, Ca, Mg, venous gas sample, FBC, LFTs



Step 2 assume he starts having convulsive seizures again

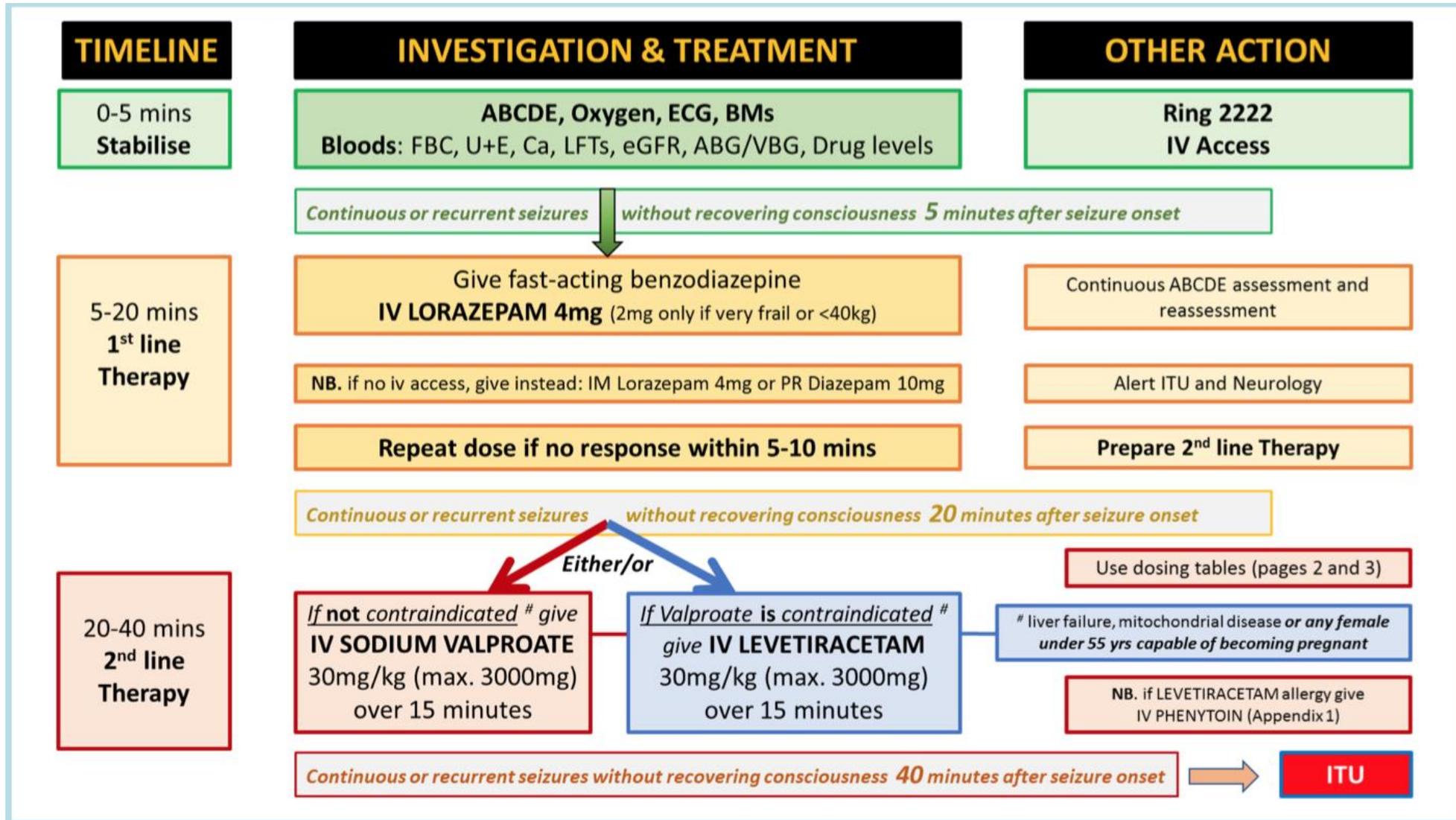


IV Lorazepam 2 - 4 mg over 60 seconds

If this is the second benzodiazepine → definitive second line drug added



Step3 Full NUH protocol



Note doses may increase
 LEV 60mg/kg
 SVP 40mg/kg
 After ESETL



Step 4. Assess response

1. Are seizures controlled?
2. If not seek help! – is this epilepsy or non-epileptic attacks?
3. Have serious causes been sought?
4. Alert ITU if seizures not controlled



Finding the cause

Previously known epilepsy

1. Low AED levels (compliance)
2. Infection

Often appropriate to reload IV with previous AED:

Phenytoin, Valproate, Levetiracetam, Lacosamide can be given IV

Not possible with lamotrigine, carbamazepine and several other



Finding the cause of SE

New epilepsy

1. Viral Encephalitis
2. Structural cause
3. Toxic (drugs and alcohol)
4. Metabolic
5. Autoimmune

POST STATUS ALGORITHM

IDENTIFY AND TREAT CAUSE OF STATUS EPILEPTICUS

Potential Causes are listed on Page 5

Investigations

- Brain imaging (CT quicker)
- CSF examination (if there is no history of seizures or obvious cause)

Also consider:

- Anti-epileptic drug levels
- MRI brain / MR venogram
- EEG
- Septic screen
- CXR if suspected aspiration
- Pregnancy test in women of childbearing age
- Toxicology screen, alcohol levels
- Serum ammonia
- Serum lactate
- Other more specialist tests e.g. VGKC antibodies (seek Neurology advice first)

PLAN REGULAR ANTI-EPILEPTIC DRUGS

Start maintenance anti-epileptic drugs within 4-8 hours of loading:

- Sodium Valproate* - Oral, NG and IV doses are 1000mg twice a day
- See Appendix 2, pg.8 for details
- Levetiracetam - Oral, NG and IV doses are 1000 mg twice a day; reduce doses in renal failure
- See Appendix 2 pg.8 for details

Always continue the patient's existing anti-epileptic drugs

- Many of these have liquid or dispersible formulations if there is no oral route (see Appendix 3).

Please Refer all patients with Status Epilepticus to the on-call Neurology Registrar

Referrals are made

via Switchboard 9am - 5pm
or via Medway/Notis out of hours
using the order term 'SEIZURE'



POTENTIAL CAUSES OF STATUS EPILEPTICUS



Infection:	Infection/sepsis, encephalitis (most commonly herpes virus), meningitis and cerebral abscess
Vascular:	Ischaemic stroke, intracerebral or subarachnoid haemorrhage, cerebral venous sinus thrombosis, hypertensive encephalopathy, posterior reversible encephalopathy syndrome (PRES)
Inflammatory:	Limbic encephalitis, demyelinating diseases or immune-mediated disorders
Metabolic:	Acute metabolic disturbances (most commonly sodium, calcium, magnesium and glucose), hypoxia/cardiac arrest
Trauma:	Head injury
Neoplasia:	Cerebral tumour (primary or secondary)
Paraneoplastic:	Some types of encephalitis
Degenerative:	All dementia syndromes
Congenital:	Idiopathic epilepsy, developmental anomalies of cerebral structure (e.g. focal cortical dysplasias)
Iatrogenic:	Non-concordance (forgetting or omitting medication)
Lifestyle:	Alcohol, illicit drugs, 'legal highs'



What if seizures do not stop?

ITU

Intubation and anaesthesia aiming for "burst suppression" under EEG



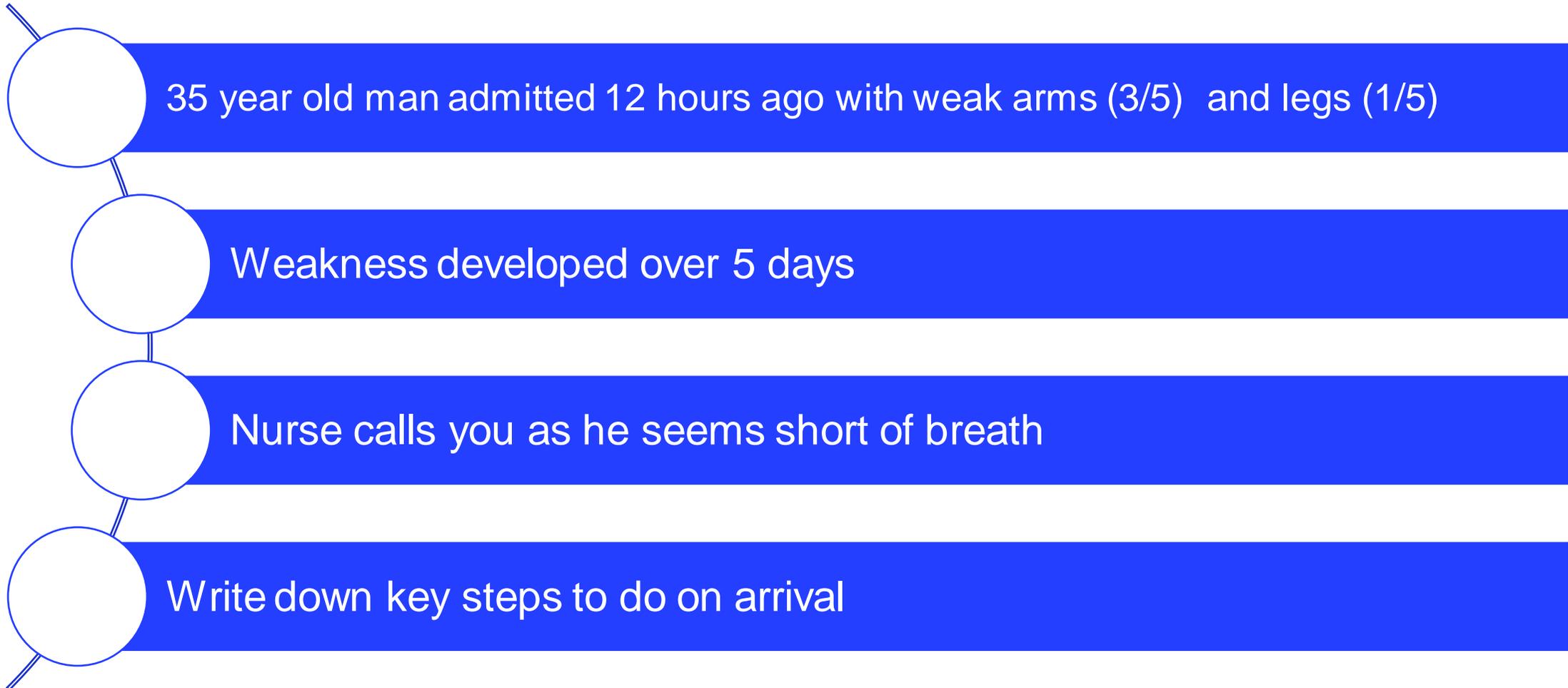
Mid point check: status epilepticus



1. What are the things to do on arrival in SE?
2. What are the drugs + doses of choice after benzodiazepines?
3. List 4 causes of new onset SE



Case



Type 1 and Type 2 Respiratory failure

Important Normal values

PaO₂ 10.6 – 13.3 kPa

PaCO₂ 4.8 – 6.1. kPa

pH 7.35 – 7.45

Bicarbonate 22 – 26 mmol/l

Respiratory rate 12- 20/min

Vital capacity (max inspiration-max expiration)

50ml/kg (3-5L average)

Drop VC upright – supine 10%

Type 1

Low O₂ Respiratory Failure

A failure of gas exchange at alveoli

e.g. acute lung pathology, PE

Type 2

High CO₂ and low O₂ respiratory failure

A failure to ventilate the alveoli

Common in COPD and Neuromuscular weakness



Neuromuscular respiratory weakness

1. Respiratory weakness kills
2. Acute and chronic neuro muscular respiratory failure differ

Acute

Guillain Barre syndrome, Myasthenic crisis

Chronic

MND, Myotonic dystrophy and other chronic myopathies



Acute v Chronic

Acute

Signs of acute neuromuscular respiratory failure

$\text{PaCO}_2 > 6 \text{ kPa}$ and $\text{pH} < 7.35$ normal or low bicarbonate

Chronic

Signs of chronic hypercapnia and nocturnal hypoventilation

$\text{PaCO}_2 > 6$ normal pH and bicarbonate $> 26 \text{ mmol/l}$



Acute neuromuscular respiratory failure

Diaphragm is the main muscle of ventilation

Respiratory failure may not be evident until diaphragm 30% function

Weakness of Diaphragm → breathless when flat + use of accessory muscles

Weak internal intercostal and abdominal muscles → weak cough

Weak bulbar muscles → Cannot clear secretions

Infection can precipitate respiratory failure



Signs of acute neuromuscular respiratory failure

Breathless and increased respiratory rate (>20)

Accessory muscle use (SCM, chest wall muscles, Intercostal)

Cannot speak in sentences or count 1 to 20 slowly

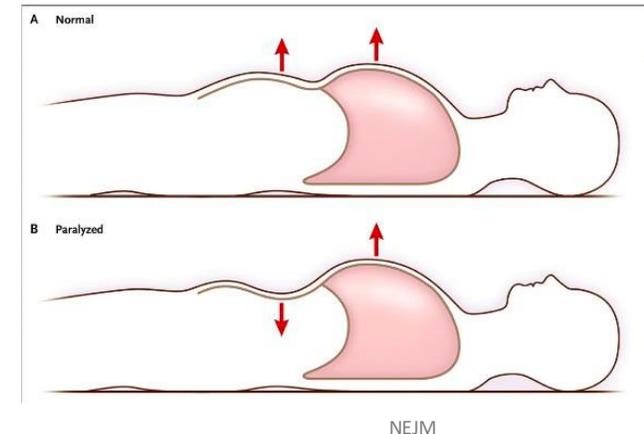
Cannot lie flat

Paradoxical diaphragm movement

Associated weak face, palate, neck, sniff, cough

Tachycardia, sweating and may have flap

If very severe: confusion due to hypoxia ($<8\text{pKa}$) and high CO_2 ($>7\text{pKa}$)



Assessment

Vital capacity the best predictor

Should be above 20ml/kg:

For 70 kg person: **1.5l or less is a serious concern**

Drop of >20% upright to supine suggests diaphragm paralysis

Practical Problems: accessing a VC machine and getting good measurement (lip seal)

If you can count 1 to 20 out loud 1/s probably OK for now

Gases: high CO₂ +/- hypoxemia with acidosis. Bicarbonate low or normal



Summary of signs



Weak limbs, face, neck, swallow

Cannot count 1 – 20

Anxious, sweaty

Cannot lie flat and diaphragm paradox

VC < 1.5 liters

Saturation may be OK until late



Management

Call for help: Critical care

Ensure airway is safe

Sit up, Oxygen mask monitoring HR and Saturations. (90-92%)

Suctioning if secretions++

Nil by mouth

Blood gases

IV access

Chest X-ray for ? Infection

Further management for critical care and neurology



Blood gas results

Acute neuromuscular respiratory failure

pH < **7.35** Pa O₂ < **8** pKa PaCO₂ > **6** pKa Bicarbonate low or normal

Chronic

pH normal PaCO₂ > **6** pKa Bicarbonate >26mmol/l



Chronic neuromuscular failure

Usually in patients with known neuromuscular disease

Myotonic dystrophy, acid maltase deficiency or MND

Symptoms of nocturnal hypoventilation:

Morning headache and lethargy

Daytime hypersomnolence

Resting Saturation <95%

Assessed by overnight oximetry



Understanding check

Called to patient with acute neuromuscular failure

- What are the two commonest causes?
- What is the best assessment of risk of needing ICU?
- What steps would you take at the bedside on arrival?

