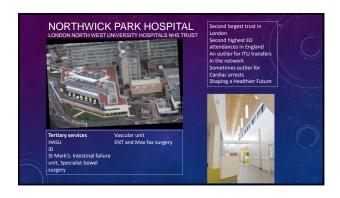
Scope – as requested

- 09.00 09.05 Introduction
- 09.05 09.45 MET calls: practical management of the deteriorating patient (Kath Fawcett)
- 09.45 10.20 Management of acute kidney injury. (Chris Laing)
- 10.20 11.00 Ward NIV: practical management, and its impact on patients and staff (Sussannah Bloch)
- 11.00 11.30 Coffee
- 11.30 12.00 Practical shock and sepsis management (Kath Fawcett)
- 12.00 12.35 Medical frailty and ICU admission. (Jamie Gross)
- 12-35 1300 Doing the right thing: who should we admit, and what are we committing them to? (audie nce/panel debate) Chaired by Ganesh all presenters able to participate





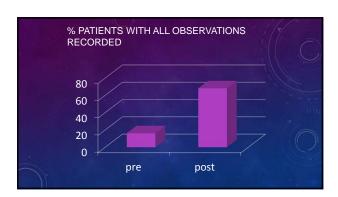


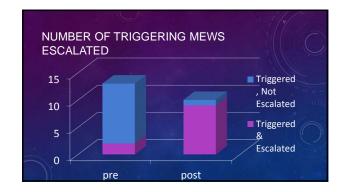


	1/4		
BACKGROUND			
Flexi-trainee in Acute medicine			
Started at NPH Oct 2010 Working on HDU, on call weekly or so,			
NPH MEWS obs charts Only 1 CCOT – who faced resistance from ward nurses	in in		
 Referrals for HDU from doctors, acute colleagues, nurses, physios, CCOT Often at 4.30pm 			
 (No cover for HDU after 5pm – only a med reg in ED until 9am) Mortality reviews, QUIP work 			
	\ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \		
BACKGROUND CONTINUED			
		-	
1 F/T registrar, 2 doing job share, 1 SHO			
 1 Consultant: who would do a 30-45min ward round before seeing her Respiratory pa We registrars were all sharing mortality audit work 	atients.		
Notes v difficult to find Left just looking at the scanned notes			
Trust proforma distracting Annoying and boring!			
	1-/		
THEMEO			
THEMES			
Most useful just reading the notes through	The second		
Get an overall sense of the failings Themes will then emerge	ring		
A focus for quality improvement			



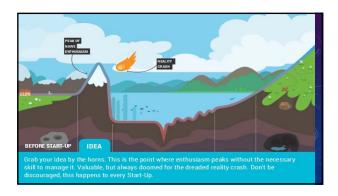






IIMIA	NG HIGH: INTRODUCING THE MET CALL We needed a team to be called early on in escalation, to:	
	Take it seriously	
	Stay involved as long as needed	
	Be Voice called like cardiac arrest	
3	Links to ITU and medical HDU	
	And to change over to national EWS (NEWS) still in development: the best EWS possible with SBAR for making escalation calls	
	We had a ready made team at NPH: skilled acute medical registrars already running the HDU	
X	Used to taking deterioration seriously, already trained up	
No. of Contract	Gatekeepers for HDU	
	And with the outreach nurses who had links to ITU	

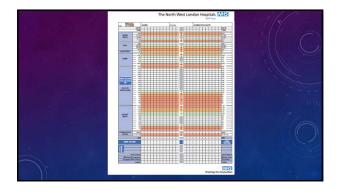
AN IDEA IS BORN	
Presented findings at clinical governance	
Declared the need for a Medical Emergency Team to respond to deteriorating patients	
Sceptical response: "Won't work" "Team will be inundated"	
"Teams won't want other doctors to interfere with their patients"	
2 CCOT nurses who had just taken over were there	
We pledged to make it work	



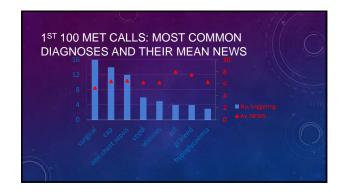
HOW DOES A REGISTRAR TAKE ON A PROJECT LIKE THIS?
Time
Stakeholder "buy in"
Established strong links with CCOT – who are the 'beating heart' of the hospital – know everyone, are trusted by everyone, know everything in terms of patients, SI's, how to get things done etc
Thick skin, belief vs delusional?

"POLI POLI"	
Took about 9 months from idea to trial	
Spent that time	
Writing Observations policy	
Introduced SBAR system at same time for staff escalating deteriorating patients	
Getting it ratified by the Board	
Presenting the plan to different clinician groups: medical, surgical, critical care	
Training the juniors	
Training the nurses	
Adding the Medical Emergency Call bleep to switchboard voice calls	
Waiting for the National Early Warning Score to be released formally	
'Trial' on 2 acute wards and the gastroenterology ward	

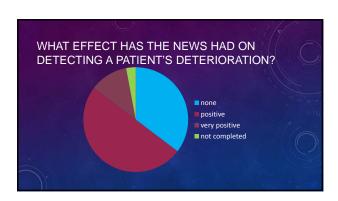


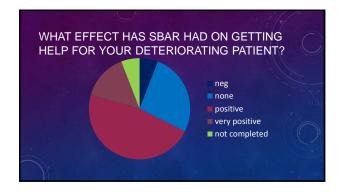






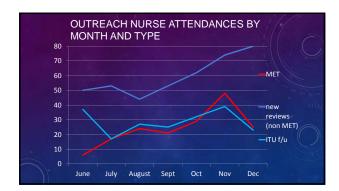










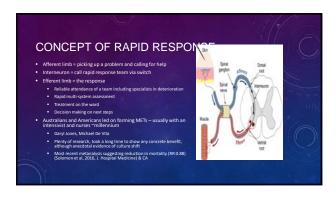


	ROLL OUT TO THE ENTIRE HOSPITAL AUGUST 2013
· .	Expansion was cautious
•	From August 2013 (when I returned as a consultant), we opened it up to the whole hospital.
•	Data collection initially the SBAR stickers, then electronic database
•	MET calls have steadily increased
	Data showing numbers of calls out of hours justified a ward registrar as well as registrar on take, and 24hr outreach nurse cover
	Now we have 2 outreach nurses on day and night, 7 days a week
	Reaching around 600 MET calls per month last Winter: 20/24hrs
	Downsides: winter months are extremely busy for MET team, sometimes 3 calls at once – but patients rescued earlier in general. SI panels and senior management have re-iterated the need to follow escalation policy.

	\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\
RISK SCORING	
Aggregate score (NEWS)	Risk
1-4	LOW
5-6 (or 3 in 1 parameter)	MEDIUM
7 OR MORE	HIGH

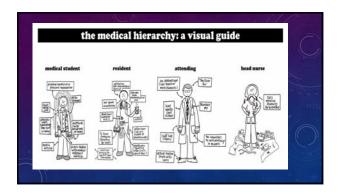


















MET CALLS: SITUATIONAL AWARENESS Now your team: swap mobile numbers at beginning of shift check competencies Check bleeps are all getting MET calls What will you do if there are 2 or more MET calls at once? Safety brief at beginning of shift: Discuss patients of concern Discuss how you will communicate.

CEILINGS (OF CARE
 Patients shouldn' management. 	t be for MET calls if they are for conservative or palliative
	ward based care, it is the decision of the team's consultant as talls would be of benefit compared to the team's own led.
	tients are not for MET – are they still for escalation to the comfort measures only? Are they for observations?

FUNCTIONAL BASELINE	
Generally rather cursory	
Not just 'can they' but DO they?	
Do they leave the house often?	
Do they walk around a supermarket? Go up and down the stairs?	
Do they walk a mile?	
 BiPAP for T2RF tends to be offered to COPD patients who are very limited – generally successful, but I would not give such patients BIPAP and vasopressors 	

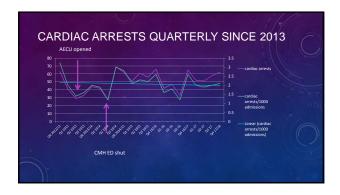
NEWS 2 now offers separate scale for CO2 retainers
Such patients – on LTOT, high b/l RR, will still score 3 at baseline
In such patients, difficult to decide on appropriate trigger for deterioration
Change in NEWS may be more relevant than absolute number
However, have less capacity to tolerate further changes
Other patients with altered baseline include disordered consciousness (brain injury etc), acute tachycardias
Best NOT to alter NEWS scores or chart
Change the ESCALATION – write it clearly in the notes and ensure nurses know and understand the plan
Maintain safety net: nurses can always call if worried no matter what score is, add 1 or 2 to usual trigger, review the patient after a few hours and include a bleep number to contact.

TREAT THE PROBLEM EFFECTIVELY • Work out the Whys and the Whats • Why is the patient in respiratory distress? • Secretions & sputum plugging • Chest physo, suctioning, nebulaires • PE – easy to overlook • Pneumonia vs Pulmonary Oedema • Be sparing with oxygen – no one needs sats 100% • Often FLOW which is needed-NHF, CPAP, humidified wall O2

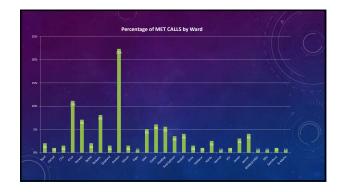
Wheeze – back to back nebs,

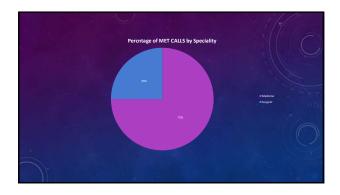
CRITICISM OF THE CALLER • Incredibly damaging to escalation • Try to pretend to be grateful that the team called you! • It is terrifyingly easy to turn people off calling

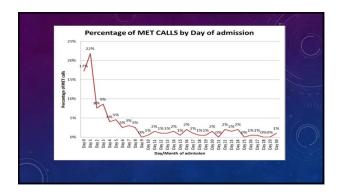


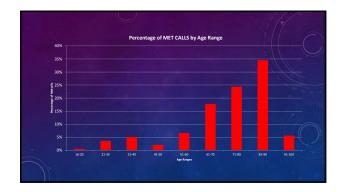


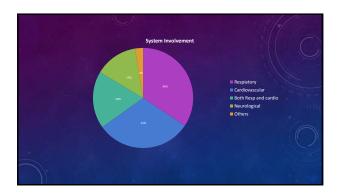


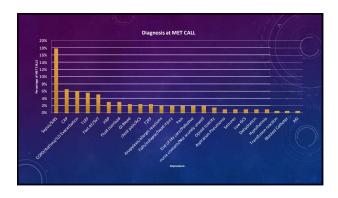




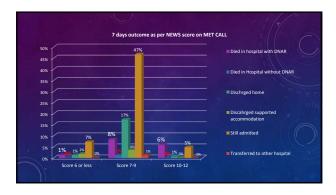


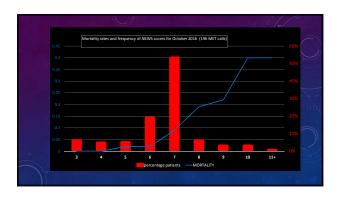


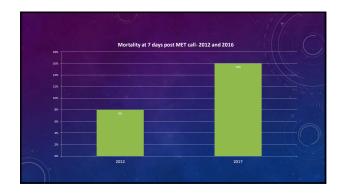


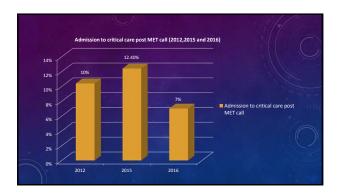


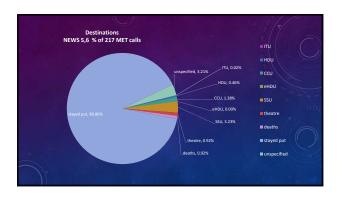


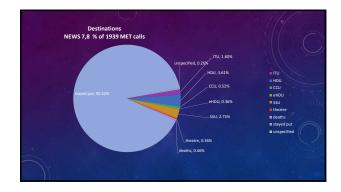


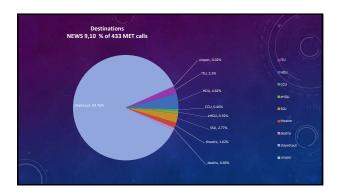


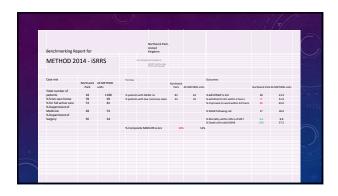


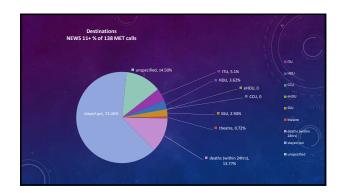


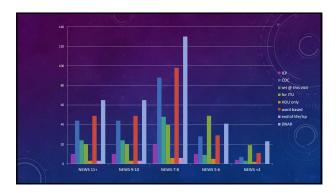


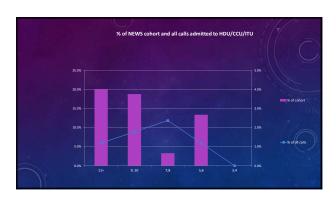


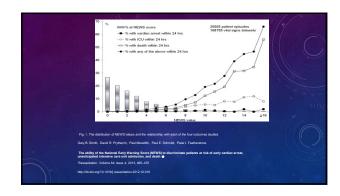


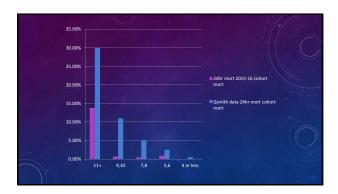


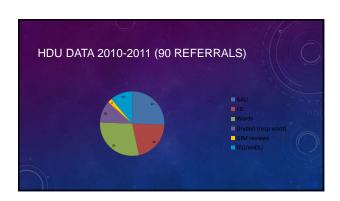


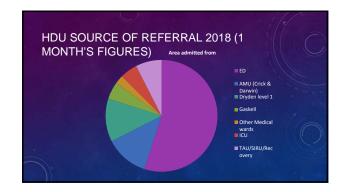


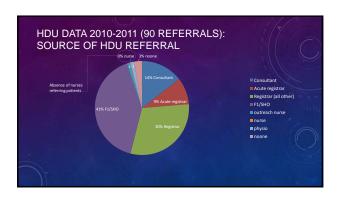


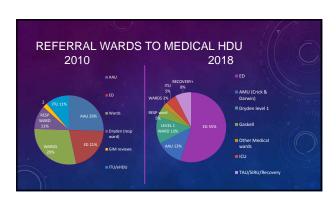






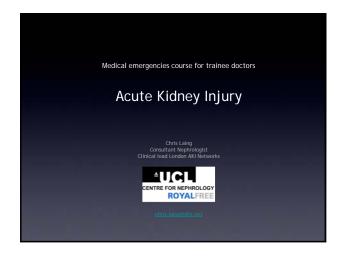






HDU MORTALITY	COMPARISONS	
Mortality 2010: 25%		
Mortality 2018: 12%		
Vast majority of referrals	via ED and MET* calls in 2018 (>95%)	
*from NURSES		

I١	N SUMMARY
٠	NEWS works: sensitive measure which conveys the risk of that patient deteriorating <4 = v low risk
	MET calls work: skilled team deal with raised NEWS across the hospital as it occurs (in general)
	Rarely calls made which should have been Crash calls (low GCS/fits/airway problems)
ı	Risk of 'call fatigue' has not been significant despite high call rates
۰	Risk of team burn out a concern
٠	Metrics of success difficult to pin down
	Some issues will improve with e-obs and NEWS 2
	Cultural change takes time
	Morale is shaky, frighteningly easy to deter MET calls being made
	We need ward based teams to respond to deterioration better, and earlier





Bywaters and Beal British Medical Journal 1941 Acute kidney injury associated with crush injuries in bombing victims ...signs of renal damage soon appear, and progress even though the crushed limb be amputated... ...the urine output, initially small, owing perhaps to the severity of the shock, diminishes further..... ...oedema, thirst and incessant vomiting soon develop. The blood urea and potassium, raised at an early stage, become progressively higher..... ...the patient is alternately drowsy and anxiously aware of the severity of his illness...

	-
Case 1	
	-
 78 year old CKD (Cr 130), peripheral vascular disease, diabetes, hypertension 	
On ACE, diuretic	
Fractured neck of femur	
NSAID analgesia	
Dyspnoea ?PE - CTPA	
Cr 220 on admission, day 8 800, K 6.8, Bic 12	
Dialysis, cr on discharge 200	
MULTIFACTORIAL AKI (high risk with several acute insults)	_
 Risk assessment, prevention, monitoring, recognition and early therapy all deficient 	

Case 2	
	-
19 year old femaleFever, malaiseBP 85/60	-
 Petechial rash Cr 60, 90 on admission Lactate 5 	
 AKI SECONDARY TO SEPSIS/HYPOPERFUSION Even mild AKI is a marker of illness severity 	

Case 3	
• 56 year old male	
Baseline Cr 82Attends GP surgery, malaise, cough	
• Cr 550	
Referred local acute medicine team	
Urine dipstick blood and protein +++	
Anuric, continued rise in creatinine (>700)	
Falling Hb to 7.6	
 Rise in K to 6.9, acidosis, 4 x doses of insulin dextrose 	
Emergency transfer Control of the control	
Same day biopsy - crescentic vasculitis	



 ANCA 	+ve ((pAN	CA)
--------------------------	-------	------	-----

- MPO IgG Ab 47
- raised KCO
- Anti-GBM, ANA, RF negative. Complement normal
- Randomised PEXIVAS trial (steroids, cyclophosphamide, plasma exchange)

 The Committee of Acid Memory and Consecution for the Committee of Acid Memory and Consecution of Acid Memory (MCA) Associated Paracellis (PEXIVAS)

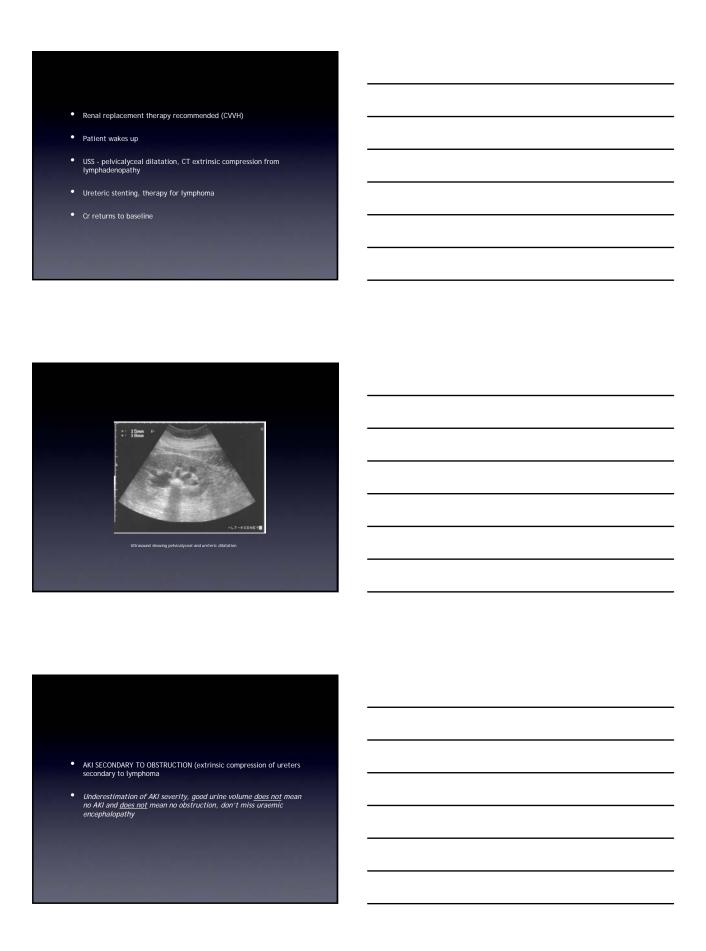
 RECOGNIZION

 RECO
- Dialysis independence Cr 200

AKI SECONDARY TO PARENCHYMAL KIDNEY DISEASE (VASCULITIS)

 Delayed referral to nephrology services, inappropriate repeated use of insulin an dextrose, acute renal disease may be systemic

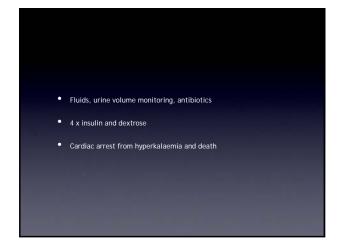
Case 4	
Case 4	
And the last of th	
2 -	
Case 5	
22 year old female	
New diagnosis of Hodgkins lymphoma	
Progressive decline in conscious level	
Intubated, CT, MRI, LP no abnormality	
Urine volume 200mls/hr, Cr 180 (baseline 55), urea 38	

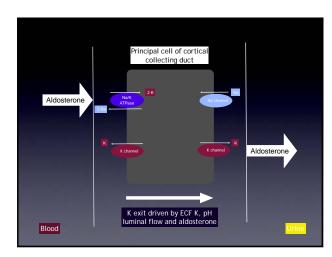


Case 6	
Case 0	
The second secon	
	-
 35 year old crohns disease, sepsis, small bowel resection, high dose steroids, TPN, TPN and NG feeding 	
steroids, TPN, TPN and NG feeding Baseline Cr 40, now 170	
Phosphate 3.1, Bic 15, urea 30	
and the second second	
The State of the S	
the state of the s	
_	
AKI SECONDARY TO SEPSIS	
 Don't underestimate degree of AKI, need for RRT dependent on compensation 	

Case 7	
	-
 46 year old lymphoma, chemotherapy 2 days post chemotherapy LDH 2000, Cr rise 90 to 140, PO4 4.1, Ca 1.7 	
	-
Urate high also	
Emergency dialysis to reduce phosphate	
Avoided IV calcium	

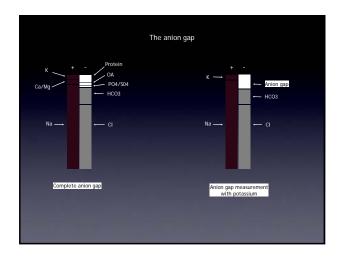
AKI SECONDARY TO PARENCHYMAL KIDNEY DISEASE (tumour lysis	
syndrome)	
 Anticipate AKI and prevent, need for RRT not dependent on level of renal impairment but on decompensation 	
Case 8	
	-
• 73 year old male	
Background ischaemic heart disease, LVF, CKD (baseline Cr 140)	
ACE, spironolactone, frusemide, B-blocker	
Admitted pneumonia, Cr 180	
K 7.8 with ECG changes, not acidotic	-

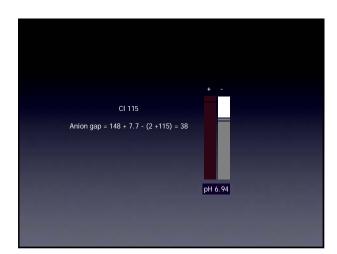


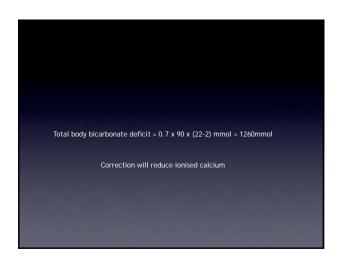




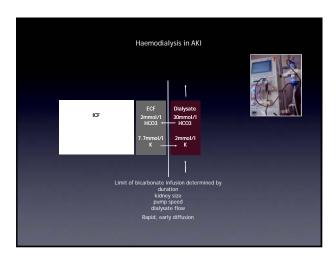
Case 9	
 45 year old male Diabetic, mild CKD (Cr 120) 	
 Generally unwell, malaise, lethargy, dyspnoea Dry, normotensive 	
Urea 120 pH 6.94 Cr 3472 PCO2 1.9	
Na 148 K 7.7 Bic 2 Ca 1.87 PO2 17.6 HCO3 3 BX2 -26 Lactate 1.8	
PO4 2.8 Alb 40	

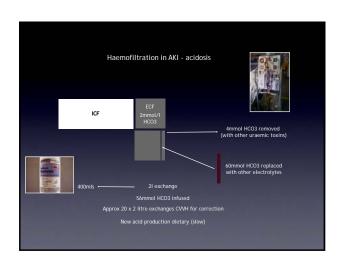


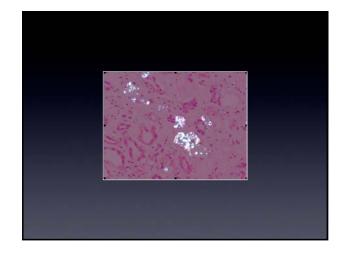


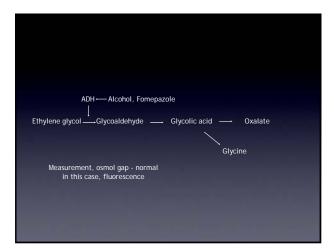




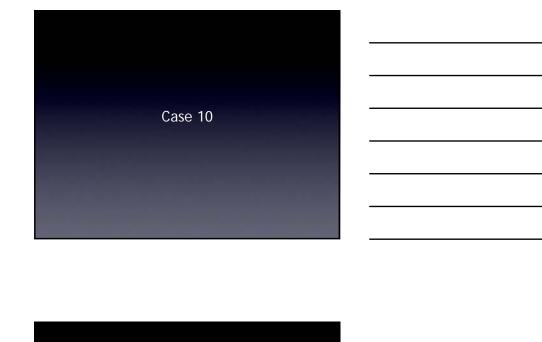






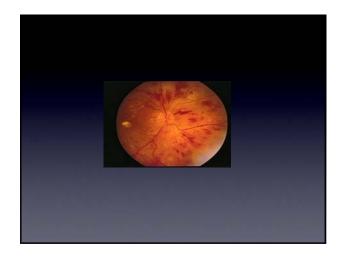


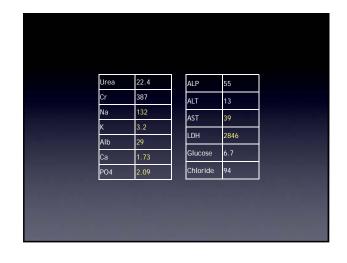
•	AKI SECONDARY TO TOXICITY (ETHYLENE GLYCOL POISONING)
۰	Beware the very high anion gap, always watch out for poisoning in acute medical presentations

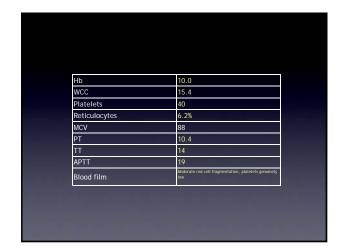


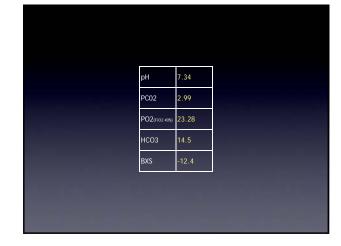
	38 year	old with	maiaise,	letnargy,	vaginai	bleeding

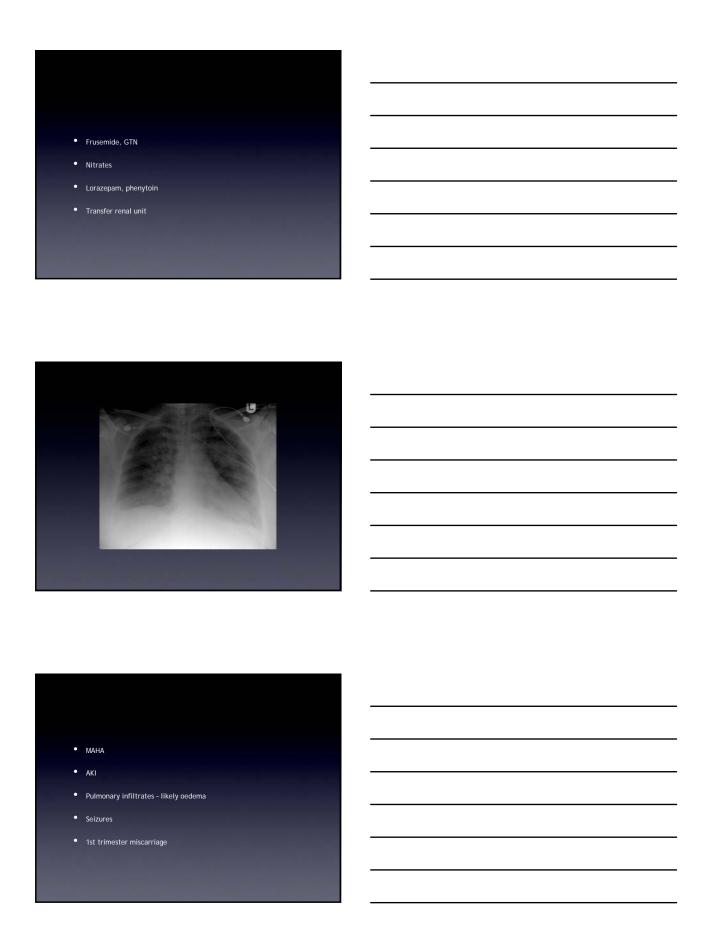
- Trying for a child, positive kit test
- Gynaecology review open cervical OS, bleeding
- BP 240/147mmHg
- Grand mal convulsion
- 'Medical opinion please'

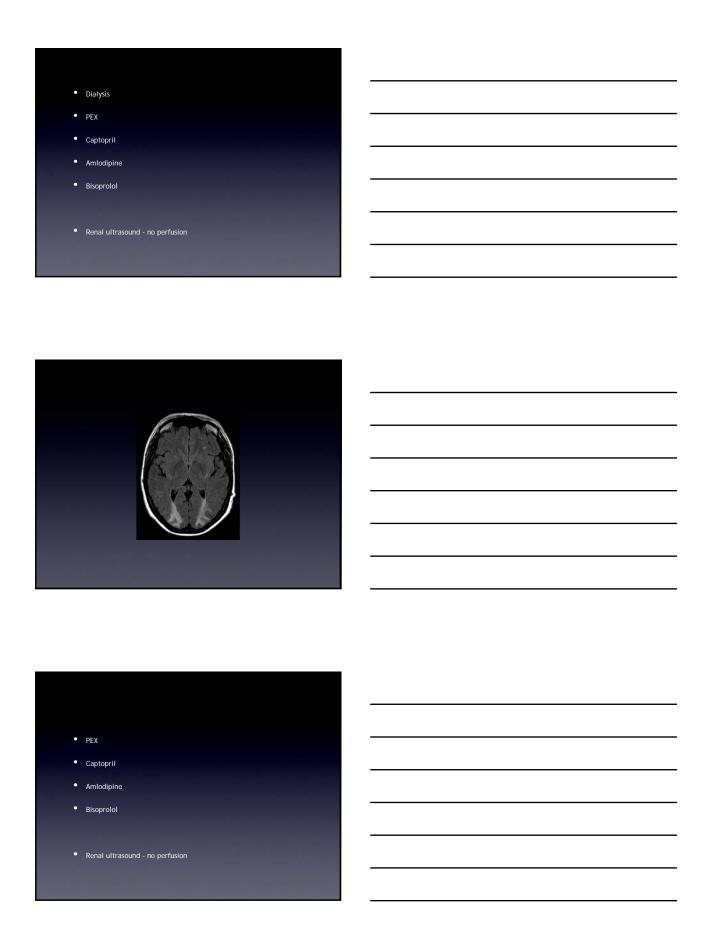
















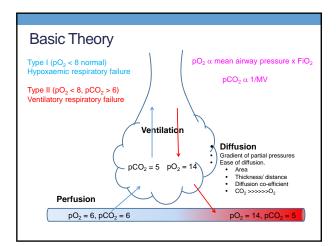
NON-INVASIVE VENTILATION: PRACTICAL MANAGEMENT AND IT'S IMPACT ON PATIENTS

Vicky Mummery/Susannah Bloch



Aim

- Theory of Non-Invasive Ventilation
- Indications/contraindications
- Guideline Update
- Setting up NIV
- Trouble shooting
- NIV treatment failure
- Key take home messages
- V60



1

Causes of respiratory failure Type I V/Q mismatch Rt to Lt intrapulmonary shunt Capillary blood supply Impairment of diffusion at level of alveolar-capillary membrane Rib fractures Pain Type II Reduced ventilatory drive Reduced tidal volume Dead space Increased CO₂ production Changes to alveoli NMD/CWD/OSA Remark of the control for the control fo

NIV explained

Non Invasive Ventilation (Bipap/ BiLevel)

Provision of ventilatory support through the patients upper airway via a mask or similar device.

Used to provide ventilatory support i.e. Type II Respiratory Failure.

Advantages of NIV over Ventilation

- Avoids intubation & assoc. problems
- Shorter hospital stay
- Avoids sedation
- Patient able to interact
- Patient able to take breaks for meals
- Able to administer in non-ICU setting
- BUT IT DOES NOT TREAT THE UNDERLYING CAUSE

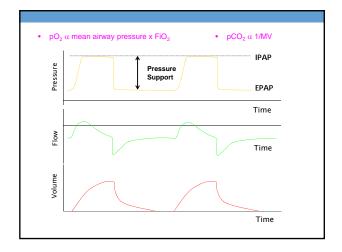
MAXIMISE MEDICAL TREATMENT FIRST

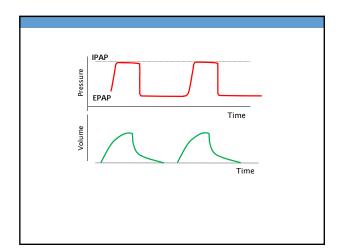
NIV	Theory
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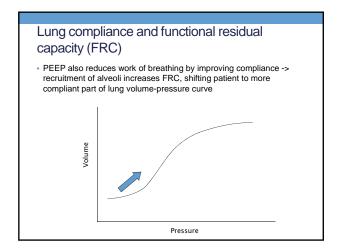
The aim of NIV is to support ventilation
There are 2 parts to NIV

Together they improve gas exchange and support respiratory function NIV machines allow a higher FiO2 delivery

- PS or IPAP
- Positive pressure support to inspiration
 - Increase TV (tidal volume)
 - Also Improve recruitment and compliance
 - Reduce work of breathing
- PEEP/ EPAP
- Positive end expiratory pressure throughout the ventilatory cycle
- Improves alveolar recruitment
- · Increases mean airway pressure
- Improves Recruitment and reduces shunt
- Unloads respiratory muscles and reduces the work of breathing
- Positive effect on cardiac afterload
- Stents open upper airway







This is the important slide!

- $pO_2 \alpha$ mean airway pressure x FiO_2 Therefore to improve oxygenation increase the FiO2 or PEEP
- $pCO_2 \alpha 1/MV$ Therefore to improve pCO2 increase the ventilation - increase the RR or the TV (PS)

Indications for NIV

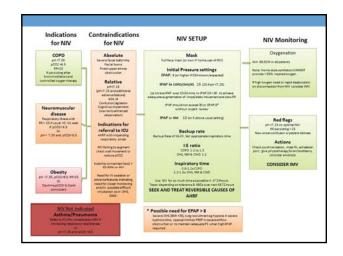


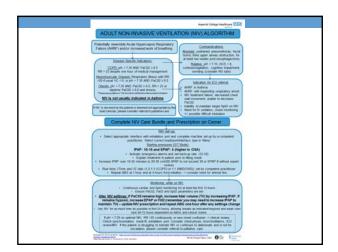
- Acidotic exacerbations of COPD
 Pulmonary oedema unresponsive to CPAP
 Acute-on-chronic hypercapnic respiratory failure in chest wall deformity / NM disease / OSA
- Weaning from ventilation
- Chronic
- Neuromuscular & chest wall diseases, COPD, OHS

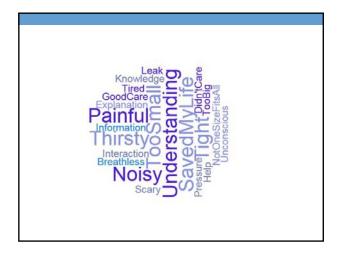
Contraindication to NIV

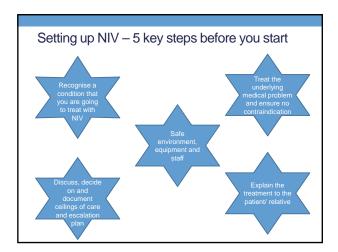
- Unconscious patient, inability to maintain own airway * Confusion/agitation *
- Vomiting
- High risk of aspiration Facial trauma/burns

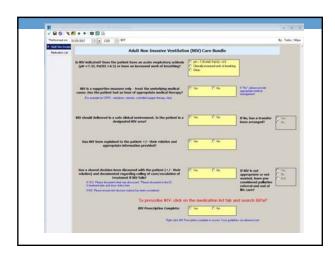
- Fixed upper airway obstruction
- Bronchial +/- pleural fistula
- Haemodynamic instability *
- Copious secretions *
- Suspected/confirmed undrained pneumothorax *
 Bowel obstruction *
- Lire-threatening hypoxamia *
 Severe co-morbidity *

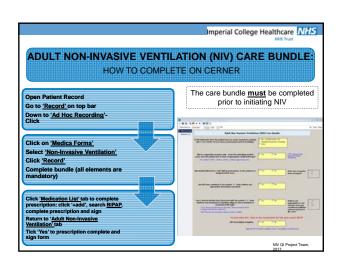












Н	ow	to	set	up	NIV
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- Initially hold the face mask in place.
- Then secure the head straps
- STAY WITH THE PATIENT
- Starting settings (12 and 4) increase every few minutes as tolerated.
- Aim for
 - Sats 88-92% (or >94%)
 - IPAP of 20
 - PEEP of 4 upwards
- Make sure the back up rate is on (12-14)
- Make sure the apnoea alarm is on
- Show the patient how to call for help and remove the mask
- Check that the chest wall is moving (coordination/ synchronisation), mask fit, comfort, heamodynamics, GCS

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- Baseline
- Response to maximal medical therapy
- At initiation of NIV
- One hour
- Frequency then depends on response
- ABG at 1 hour, then at 4-6hrs if stable
- If settings are changed, ABG 30-60 minutes after change is implemented
- CXR in event of any acute deterioration

Length of treatment

- NIV should be almost continuous for first 24 hrs (breaks allowed for meals, PHYSIO, nebs)
- Next 24 hours, at least 12 hours
- Switch to nocturnal NIV
- Consider withdrawal day 3 4

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Inadequate ventilatory support (persisting acidosis/high CO₂)

$pCO_2 \alpha 1/MV$

- Check for leaks
- Check expiratory port is not blocked
- Clinical re-evaluation (may include CXR)
- Wean FiO2 if SpO2 is >94% Increase IPAP in increments of 2-4cm $\rm H_2O$ & repeat ABG after 30-60 minutes
- Ensure optimal additional medical management

Troubleshooting

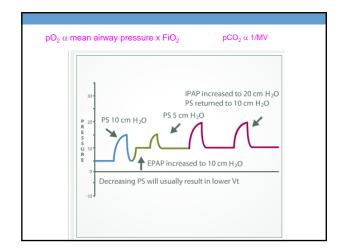
Persisting hypoxia

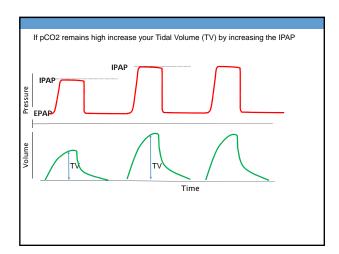
$pO_2 \alpha$ mean airway pressure x FiO_2

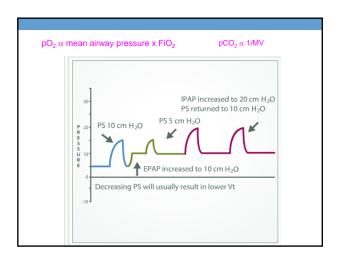
- Check for leaks
- Clinical re-evaluation (may include CXR)
- Increase EPAP in increments of 2cm H₂O (& IPAP by the same amount to maintain PS) & repeat ABG after 30-60 minutes
- \bullet Increase $\mathrm{FiO_2}\,\&$ repeat ABG after 30-60 minutes
- Ensure optimal additional medical management

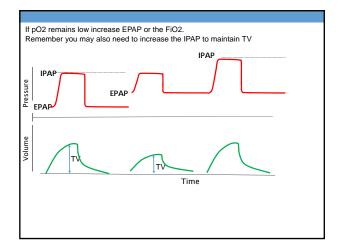
You only need to know 4 things

- pO₂ α mean airway pressure x FiO₂
- Therefore to improve oxygenation increase the FiO2 or PEEP
- pCO₂ α 1/MV
- Therefore to improve pCO2 increase the ventilation - increase the RR or the TV (PS)









Troubleshooting

- Leak from mask
 - Adjust straps
- Consider pressure dressing to cheeks/nose
- Nasal and forehead damage
 - Apply padded dressing
- Non-co-operation/aggression
 - IF sedation necessary do it carefully
- Swallowing air
 - Insert NG
- Eye irritation
 - Adjust mask fittings
- Asynchrony
- Consider different mode or machine discuss with Respiratory, it may be necessary to adjust the pressure rise time or the inspiratory

Treatment failure

- · Deterioration in patients condition
- Failure to improve ABGs
- Complications: pneumothorax, facial bridge erosion, sputum retention
- IntoleranceFailure to alleviate symptoms
- Deteriorating conscious level
- Patient / carer wish to withdraw treatment
- If the patient is for escalation of treatment start the referral process early!

Key points

- TREAT UNDERLYING CAUSE
- Ensure adequate monitoring and support
- Make a plan for treatment failure
- Ensure NIV is prescribed and the bundle is completed before initiation

 - $\rm pO_2~\alpha$ mean airway pressure x $\rm FiO_2$ Therefore to improve oxygenation increase the FiO2 or PEEP

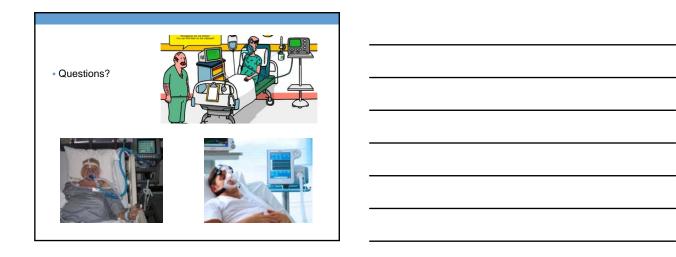
 - $\rm pCO_2~\alpha~1/MV$ Therefore to improve pCO2 increase the ventilation increase the RR or the TV (PS)

Further reading and competencies

- BTS/ICS Guidelines (2016)
- ATS/ERS Guidelines (2017)
- NCEPOD report (2017)
- Acute Adult NIV Competencies:

 $\underline{\text{http://source/cs/groups/extranet/@clinical/@guidelines/doc}}$ uments/ppgs/sid 100819.pdf









Watch our new video: Non Invasive Ventilation (NIV) for patients

NIV is often used in the treatment of acute respiratory failure, this video explains the treatment from a patient's perspective. It explains why you might need NIV and what will happen if you do.

The video includes interviews with patients who explain how the experience of using acute NIV felt to them.



https://www.youtube.com/watch?v=RSgwIEzi WO0&t=2s





Non Invasive Ventilation: An educational video designed for medical practitioners describing the patient centred approach to acute NIV.

https://www.youtube.com/watch?v=a1R3hsus MAA&t=8s



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Part II - Practical management of shock	
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• Shock	
Bedside assessment	-
 4 stages of shock management Fluids 	
Volume assessment	
Vasopressors	_
	<u>.</u>
The state of the s	
What is shock?	
High lactate	
Organs not working: brain, kidneys Handard (Could have a MAD (C but at ill in charle))	-
Hypotension (*could have a MAP >65 but still in shock)	
	-
	•
Other causes of high lactate	
Endogenous or exogenous adrenaline	
Asthma Vasopressor infusions	
Bowel ischaemia Liver failure, renal failure	
Convulsions	
Drugs (ARTs, aspirin, ETOH, methanol, metformin	

Different types of	shock & Presentation
Туре	Common Features
Hypovolaemic Cardiogenic	COLD peripheries, HR↑ BP↓ DIAPHORETIC
Distributive Septic Anaphylactic (Neurogenic)	• WARM peripheries, HR ↑BP↓
Obstructive Embolic, Tamponade Tension pneumothorax (Hyperinflation -asthma) Combined causes	Diaphoretic, JVP↑ HR↑ BP↓
• Endo/Severe acidosis/↓Ca ²⁺	

Diagnosis	
Clinical examination, examination, examination	
BP, pulse, diaphoresis, RR, sats	
Peripheral temperature and its change as you move up the arm	
• JVP	
ABG: lactic acidosis	
(Oxygenation may be low purely because lungs hypoperfused)	
Central capillary refill time >3 seconds	
Peripheral oedema	

Assess, Act, Assess, Ask	
A: airway intact/self ventilating? B: RR, sats, chest exam, ABG (lactate), CXR C: peripheral temperature & change, HR, BP, postural BP drop, JVPI D: CCS/AVPU/new Change in mental status/glucose E: rash, mottling, oedema	
F: Urine output/balance, U&E G: abdo examination, LFTs H: bloods, clotting I: temp, source, antiBs, culture results And repeat (ASK: Dxg function, allergies, last ate, collateral, ceilings/wishes)	

Cardiogenic and Obstructive	
Tend to look much worse	
VERY sweaty, greyRemember tension	
 Obstructive - quicker deterioration to cardiac arrest (Massive PE - continue CPR 1hr after thrombolysing!) 	
Septic shock	
 Can present with hypothermia Peripheries warmer > proximal limbs, 	
Sepsis 6: the earlier antibiotics given, after blood cultures, the lower the mortality	
Expanded circulation, more insensible loss from sweating/breathing, or vomitting/diarrhoea, - can be very negative fluid balance on arrival Segulation lightens again, vesspreader of specific segulation, patient	
As circulation tightens again - vasopressors or sepsis resolving, patient may well become overloaded, slow wean, ARDS etc* After 2-41 fluid, need to assess fluid responsiveness, be ready to start	<u> </u>
 After 2-4L fluid, need to assess fluid responsiveness, be ready to start vasopressors and give smaller fluid challenges "mortality increases if patients become boggy! 	
Chook requiring impropriate an eifice exting	
Shock requiring immediate specific action	
Apaphylavic	
Anaphylaxis Tension pneumothorax	
Addisonian crisis	
Embolism 'Silent chest' Asthma	

Other points	
Deranged electrolytes - K, Mg - AF, Hypocalcaemia (esp in severe acidosis) - Calcium is an inotrope in this scenario Toxicology: BB/CCB overdose - don't forget glucagon, insulin infusion (high dose), calcium, filtration	
Pregnant: remember to put pillow under R buttock IVC will be compressed by gravid uterus	

4 stages of shock	management - ROSS(D)	
 Require different app Different electrolyte. Fluid status 		
• Rescue • Optimisation	Stabilisation Stepdown/De-escalation	

Stage 1 - Rescue Scenario: New arrival in resusc or ward MET call or cardiac arrest Goal: achieve adequate cardiac output as soon as possible Secondary goal: establish differential diagnosis Fluids: N. Saline, Blood Rate: bolus - i.e. 500mls 'stat', need large bore cannulae etc Clinician presence: at bedside, continuous

Stage 2 - Optimisation

- Scenario: Hour 1 Hour 2
 Goal: Continue to aim for adequate Cardiac output, using bedside parameters
 Peripheral temperature
 Lactate
 Urine Output
 AVPU
 Secondary and
- Secondary goal: confirm type of shock(s), consider further diagnostics & treatment
- Consider advanced volume assessment* to assess for volume responsiveness

Stage 3: Stabilisation

- Goal: minimising harm from fluid and vasopressors
- Think about nutrition NG placement giving water enterally,
 This helps to maintain muscle mass & strength for weaning and rehabilitation
- Fluids: water NG, saline with drugs (antibiotics) & electrolytes, blood, albumin,
- Holistic assessment including fluid status
 Doctor: 2 assessments (at least) per day, but can be away from ward

Stage 4: Stepdown or De-escalation

- Scenario: Day 1 or 2 up until stepdown from level 2 or 3 care
- Goal: Weaning and diuresis to clear excess fluid
 Secondary goal: Fine tuning medical treatment, addressing issues holistically and using MDT interventions especially PT
- Fluids: minimal orally or NG only, Diuresing with frusemide, or potassium sparers

Filling - What? • N. Saline for rescue phase. • Eventually chloride levels will rise, causing acidosis • Not a problem in the early stage • Blood for bleeding (+FFP/Cryo/platelets prn) • Albumin - not contraindicated for use in sepsis, but place not defined yet • Save for very low albumin states, generally later stages of shock management • Nephrotic syndrome, Cirrhosis

Fluid resuscitation	
What How? Saline Blood (if bleeding) - remember MAJOR HAEMORRHAGE protocol) Just not dextrose Even cirrhotic patients - resuscitate them, worry about the ascites much later If young: give a litre stat If old/chronically hypertensive - be aware their LV won't have the capacitance/stretch to take large volumes fast - either slower or smaller volumes stat	

Filling: How?	
Deficit Diagnosis Disease: Anuric ESRF Tachyarrythmia (less filling time) L/T hypertension, Aortic stenosis HOCM Systolic heart disease Advanced age	

Shock management

- International Surviving Sepsis: 1.4L stat fluids (70kg) for septic shock
- Hypovolaemic shock but no bleeding source apparent
 Retroperitoneal haemorrhage
 AAA rupture
 Most difficult to identify source without scanning but too unstable to scan
 Major Haemorrhage call need blood, personnel, senior decisions
 Don't forget Tranexamic acid to control all bleeding
- If variceal haemorrhage: aim for SBP 100 not too high

Next stage... HDU

- A few hours/Litres of fluid down the line...
 Still shocked, not peeing, BP dropped down again
- Try fluid challenge- 250-500mls stat (pressure bag/pump)
 Insert lines arterial line, CVC
 Look for signs patient is still fluid deplete/still responsive

Starling's curve

High tech or Low tech? Fancy top only as goed as the care taken to set up and calibrate Trust your clinical skills! Measures of fluid responsiveness - venous Control second solurations: take a venous gas from the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in line and one of the central lines - measures sets in lines - the central lines - measures sets in lines - the central lines - measures sets in lines - the central lines - measures sets in lines - the central lines - measures sets in lines - the central lines - measures sets in lines - the central lines - measures sets in lines - the central lines - measures sets in lines - the central lines - measures sets - the central lines - measures - the central lines						
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Aiming for 80% Can also look at peripheral venous sats aiming >66% Caveats: more 'validated' in cardiogenic shock	Central venous saturations: take a venous gas from the central line - measures sats in blood in PA					
Delays in analysing sample will reduce accuracy Delays in analysing sample will reduce accuracy	Aiming for 80% Can also look at peripheral venous sats aiming >66%					
	Delays in analysing sample will reduce accuracy					

CVP measurements

Relies on transducer being placed at midaxillary height
"normal" = 8-12 - actually could be 3, could be 15
Large inter and intra-personal variability
Out of vogue- pretty useless
If using, make a dynamic measurement - compare pre and post fluid bolus or PLR
Beware: many resp patients have pulmonary HTN and TR - CVP will be permanently raised.
In practice, clinical examination/U/O/lactate etc more useful

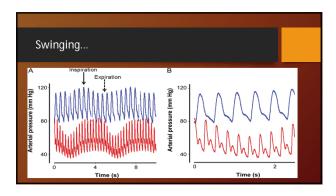
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Passive leg raise

- Equivalent to about 300ml fluid bolus, but reversible
 Need patient's head at 30 degrees and legs flat at start
 Ideally use bed controls rather than lifting legs as pain will mitigate effects
- Lower patient's head to flat and lift legs to 45 degrees or more.
- After a minute, remeasure your chosen parameter
 In theory should be oesophageal doppler of aortic pressure, or CO measurement
 In practice, arterial line trace may show an improvement

Arterial line trace analysis

- Arterial pulse wave can be analysed
- Area under the curve equivalent to stroke volume
- Diachrotic notch: placement on the down stroke gives info on fluid status
- Relies on a good trace (not attenuated) , good flow in the cannula
 Actual BP recorded relies on accurate alignment of the transducer at the height of the patient's RA = the patient's midaxillary line if the transducer is higher then it will seem like the patient is hypotensive



Arterial wave form + dilution = high tech analysis

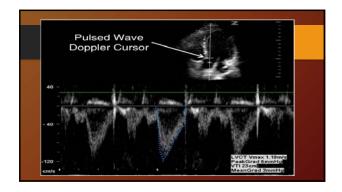
- Can use computer analysis of arterial wave forms to calculate cardiac output beat by beat
- In steady state if capacitance and systemic resistance constant then
- Pulse pressure can derive the SV and CO.
 PICCO and LiDCO uses thermodilution, lithium dilution + wave analysis
- \bullet Other measures coming through eg end tidal CO2 variation with filling,

Echo Can look at IVC diameter - better to look at before/after bolus Subcostal view, measure IVC diameter 0.5-3cm distal to RA If <1cm - fluid responsiveness likely If 1-2cm look for >50% collapse - fluid responsive

Other volume responses on Echo

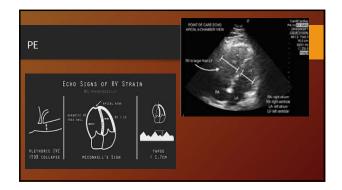
Apical views: Look at LVOT VTI (5 chamber view, apical) - trace around Pulsed wave doppler trace (quality) - if >20 = good SV or look for >15% increase after filling/Passive Leg Raise

Left atrium : if remains circular throughout cycle (ie bowing into RA) then ${\bf pt}$ is well filled.

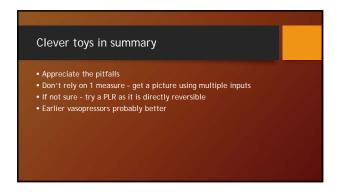




Echo: PE and tamponade	
PE: RV dillated - D shape on short axis parasternal or similar size to LV on 4 chamber apical (should be 0.5 the volume) May also see paradoxical septal motion (septum moves to L on systole) LV may well be small, collapsing on end systole (less filling)	
Tamponade: effusion + RA wall systolic collapse for >1/3 of cycle RV wall collapse in diastole IVC dilated >2cm	

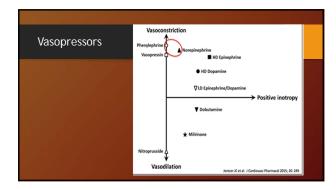






Marie Contract of the Contract	
Vasopressors	
Temporising measure - need to treat the cause Timing: give some fluid first - patients need it Don't be afraid of fluids Don't give patient 10L either!! Markers: lactate and U/O not responding Use peripheral temp as a guide + consider it once 3L+ start VP when they're warm" Generally started during stage 2 of Shock treatment	

Which one?					
	B ₁ receptors	B ₂ receptors	A ₁ r.	A ₂ r.	DA ₁ and DA ₂
Effects:	• +HR, inotropy • +Blood flow	• Vaso+ broncho- dilation • + inotropy	• +SVR • +inotropy	+Systemic Vascular Resistance	Natriuresis Splanchnic vasodilator
Adrenaline	++ to +++	+ to ++	+ to ++++ (dose)	+/- to +++ (dose)	-
Noradrenaline	++ (higher doses)	-	+++ (reflex bradycardia)	+++	-
Isoprenaline	***	+++	-	-	-
Dopamine	+/- to +++ (dose)	- to ++ (dose)	+/- to +++ (dose)	+	+
Dobutamine	++	+	+/-	??	0



	Agent	VASOPRESSIN	LEVOSIMENDAN	MILRINONE
_	Class	Endogenous peptide	Calcium sensitiser Inodilator	Bipryidine Inodilator
	Mechanism	V1=vasoconstriction V2=renal + endo V3= pituitary OxyT	Modulates troponin C Vascular ATP-K channels	
	Effects	Antidiuresis +SVR +platelet aggreg Less splanchnic flow	Inotropy Less SVR Less PVR +CO	Inotropy Less SVR Less PVR +CO
	Cons	\$\$\$ PHT Splanchnic ischaemia Uterine contraction Digital ischaemia	\$\$\$\$\$ Tachycardia, low BP Can't use in liver/renal F	\$\$\$ Low BP, may need NA too Little evidence
	Uses	Added in for septic shock (Terli - Decomp cirrhosis)	HF, can use with BB	HF, supports RV, can use with BB

In practice	
Most patients have a combination of sepsis + hypo or cardio shock Practically: Start NA first, achieve a MAP of 65 and urine output If not achieving aims and using lots of NA Look for other cause: bleeding/third space losses/cardiac failure/addisonian If cardiac failure: add in dobutamine If patient is a candidate for ITU: refer if steadily increasing req Dobutamine on its own frequently causes hypotension NA can cause reflex bradycardia - switch to adrenaline instead	

Saline for filling in rescue stage Once 3-4 L given look for volume responsiveness Look to start vasopressors Use repeated clinical examination to guide you Low tech volume assessment is useful: PLR Arterial line swing Central venous saturations Opn't rely on one parameter, use a variety Vasopressors: Noradrenaline Ad in dobutamine if HF Metaraminol can be useful

ICU: To admit or not to admit?

Jamie Gross Consultant in Intensive Care Northwick Park Hospital



@GrossJamie



Case 1

- 72 year old gentleman
- Type 2 respiratory failure secondary to pneumonia
- Multiple comorbidities
 - IHD and CCF with Dual chamber PPM
 AF (on warfarin)
 COPD on inhalers
 Type 2 DM and Hypertension
 Stage 3 CKD

 Functionally at baselines COP on wall
- Functionally at baseline: SOB on walking 5-10 metres
- Social history: lives with wife who is the main carer; doesn't leave house; Can wash/dress self but needs help showering. Wife does housework/cooking

	_
Observations and bloods	
Alert but slightly confused	
Tachypnoeic (RR 40) and using accessory muscles	
On FiO2 1.0; SpO2 82% HR 105 (AF) BP 90/40 (normally 130 systolic)	
Bloods: Creat 250 (B/L 140)	
Lactate 3.2	
Rx: Already started on broad spectrum cover antibiotics	
]
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Family want "everything to be done"	
"she's a fighter"	
Modical consultant do tur	
Medical consultant does not know the patient "let ICU decide"	

How far would you escalate this patient?

- 1. "Full escalation" if needed on ICU
 - mechanical ventilation
 - inotropes
 - Renal replacement therapy
- 2. "partial escalation" on HDU
 - NIV/optiflow
 - Inotropes
 - +/- renal replacement therapy
- 3. Ward care only

Case 2

- 90 year old lady
- Type 2 respiratory failure secondary to pneumonia
- PMH: F+W. Lifelong non-smoker
- Functional status:
 - "independent" (Weight 50 Kg)
 - lives on own but "slowed up a bit" recently. Manages ADLs
 - Gets out to the shops 2-3 times/week
 - Has a cleaner

Alert but confused and disorientated Tachypnoeic (RR 35) and using accessory muscles On FiO2 1.0; SpO2 82% ABGs on 100% O2: pO2 6.1, pCO2 8.4 pH 7.31 (normal Bicarb/BE) BP 140/84

Bloods: Nil significant

Lactate 1.1

Rx: Already started on broad spectrum cover

antibiotics

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Medics "She is a good 90 year old"

Family: "patient doesn't want a quality of life that is dependent on others"

How far would you escalate this patient?

- 1. "Full escalation" if needed on ICU
 - mechanical ventilation
 - inotropes
 - Renal replacement therapy
- 2. "partial escalation"
 - NIV/optiflow
 - Inotropes
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- Doing Good

NON MALEFICENCE

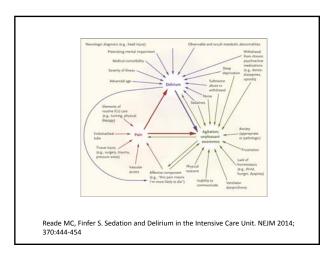
- Avoiding harm

AUTOMONY

- Informed consent

BENEFICENCE - Doing Good NON MALIFICENCE - Avoiding harm AUTOMONY - Informed consent	
	1
Beneficence v Non malificence Beneficence (doing good): - Longer term survival + - Left with a quality of life that is acceptable to the patient Non Malificence (avoiding harm) -> ICU IS HARMFUL TO MANY PATIENTS (particularly those intubated and ventilated)	
The Harms of ICU	



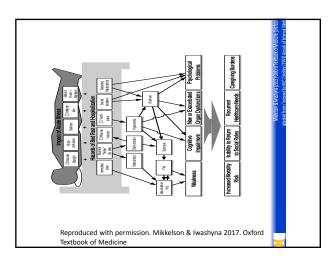




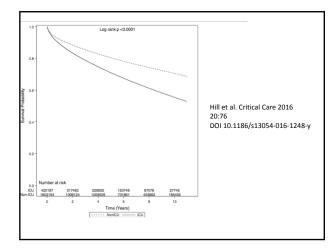
Intermediate/Long term physical and psychological sequalae

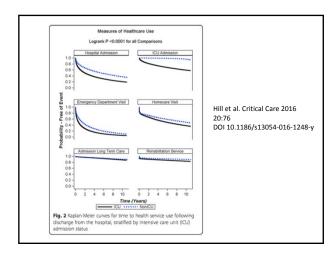
- Profound muscle weakness*
 - Difficulty weaning from ventilator
 - Prolonged period of rehabilitation
- Amnesia and cognitive impairment
- Anxiety/depression
- Post Traumatic Stress Disorder (PTSD)

→ May not ever get back to functional baseline and can be left with significant disability, reduced psychosocial function and poor QOL



Cuthbertson et al. Critical Care 2010, 14 :96 http://ccforum.com/content/14/1/R6	C CRITICAL CA
RESEARCH	Open Acce
Quality of life in the five year care: a cohort study	rs after intensive
Brian H Cuthbertson ^{1*} , Siån Roughton ² , David Jenkinson ³ , Graeme	MacLennan ³ , Luke Vale ^{2,4}
Abstract	
Introduction: Data on quality of life beyond 2 years after intensive explore this area further. Our objective was to quantify quality of lintensive care discharge.	
Methods: A prospective longitudinal cohort study in a University Inform the period before ICU admission until 5 years and quality adj	
Results: 300 level 3 intensive care patients of median age 60.5 yes recruited. Physical quality of life 611 to 3 morths (P = 0003), rose to again from 2.5 to 5 years after intensive care (P = 0.002). Mean phy all time points but the mean mental scores after 6 months were six value measured using the EuroQCU-50 quality of life assessment to years after intensive care unit, the cumulative quality adjusted life 5 for the general population (P < 0.001).	pack to pre-morbid levels at 12 months then fi ysical scores were below the population norm imilar to those population norms. The utility pool (EQ-5D) at 5 years was 0.677. During the fir
Conclusions: Intensive care unit admission is associated with a hig low quality adjusted life years gained compared to the general poy critical illness associated with ICU admission should be treated as a mortality, morbidity and the requirement for ongoing health care	pulation for 5 years after discharge. In this group life time diagnosis with associated excess



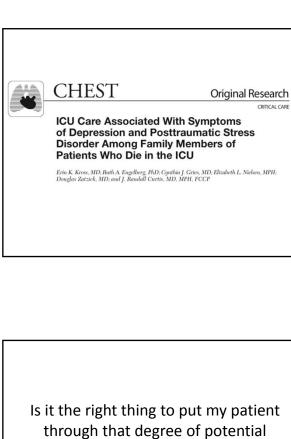


One-Year Outcomes in Caregivers of Critically Ill Patients

CONCLUSIONS
In this study, most caregivers of critically ill patients reported high levels of depressive symptoms, which commonly persisted up to 1 year and did not decrease in some caregivers. (Funded by the Canadian Institutes of Health Research and others; ClinicalTrials.gov number, NCT00896220.)

N ENGL J MED 374:19 NEJM.ORG MAY 12, 2016

Cameron JI et al. NEJM 2016;374:1831-41



through that degree of potential harm in the context of critical illness?

Are they likely to survive?

Are they likely to be left with a quality of life that is acceptable to them?

ANALYSIS

Intensive care: balancing risk and benefit to facilitate informed decisions

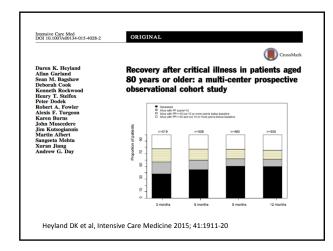
More efforts are needed to engage with the wider healthcare community and the public about what intensive care can—and cant—achieve, say Jamie Gross and colleagues

Jamie Gross consultant in intensive care medicine¹, Barry Williams patient and public representative², Premila Fade consultant geriatrician¹, Stephen J Brett consultant in intensive care medicine and professor of critical care³

How do we make benefit v burden assessment?

Patient's that don't do well in ICU

- Severe chronic respiratory disease
- Severe cardiovascular disease
- Cirrhosis of the liver with portal hypertension
- Those on long term dialysis
- Immunocompromised state (e.g. haematological malignancy, AIDS etc)
- ? Age
 - → Frailty



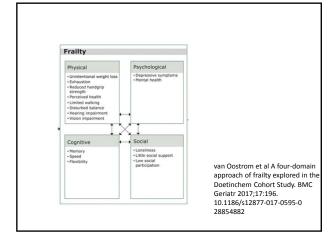
Variables	Single predictor		
	OR (95 % CI)	ca	P value
Age (per 5 years)	0.77 (0.64, 0.93)	0.53	0.006
Sex (male vs. female)	0.79 (0.60, 1.04)	0.53	0.09
APACHE II score (per 10 points)	0.55 (0.38, 0.80)	0.62	0.002
Marital status (married or living as married vs. other)		0.52	0.36
Baseline SOFA score (per 5 points)	0.82 (0.64, 1.05)	0.54	0.12
Admission type (medical vs. surgical)		0.64	< 0.0001
Surgical elective vs. medical	4.37 (2.82, 6.76)		
Surgical emergency vs. medical	1.99 (1.18, 3.35)		
Primary ICU diagnosis		0.64	< 0.0001
CABG/valve vs. cardiovascular/vascular	5.60 (3.32, 9.43)		
Gastrointestinal vs. cardiovascular/vascular	1.25 (0.59, 2.65)		
Neurologic vs. cardiovascular/vascular Other vs. cardiovascular/vascular	2.10 (0.88, 5.00)		
Respiratory vs. cardiovascular/vascular	0.73 (0.29, 1.83) 0.96 (0.51, 1.80)		
Sepsis vs. cardiovascular/vascular Sepsis vs. cardiovascular/vascular	0.85 (0.36, 1.80)		
Stroke vs. cardiovascular/vascular	0.18 (0.02, 1.53)		
Trauma vs. cardiovascular/vascular	0.70 (0.29, 1.70)		
Baseline PF score (per 50 points)	1.00 (0.73, 1.37)	0.51	0.98
Charlson Comorbidity Index (per 2 units)	0.61 (0.49, 0.77)	0.60	<0.0001
IQCODE at baseline (per 0.5 point)	0.75 (0.57, 0.98)	0.59	0.04
Frailty index (per 0.2 point)	0.48 (0.35, 0.66)	0.63	< 0.0001
Family preferences for life sustaining treatment	0140 (0120) 0100)	0.00	40,000
Comforts measures vs. other	0.69 (0.29, 1.65)	0.53	0.40
Total model degrees of freedom/events	1-8/123		
c-Statistic ^a	0.51-0.64		

McDermid et al. Critical Care 2011, 19301
http://ccforum.com/content/15/1/901

CRITICAL CARE

VIEWPOINT

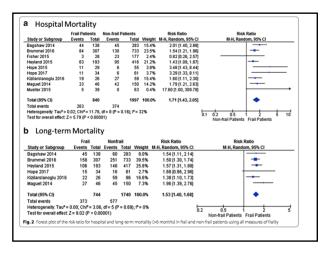
Frailty in the critically ill: a novel concept
Robert C McDermid', Henry T Stelfox' and Sean M Bagshaw'*

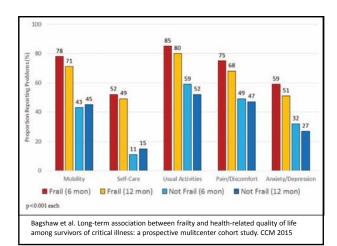


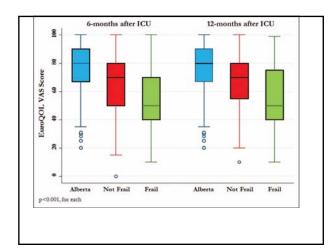


The impact of frailty on intensive care unit outcomes: a systematic review and meta-analysis

John Muscedere^{1,50}®, Braden Waters³, Aditya Varambally³, Sean M. Bagshaw⁶, J. Gordon Boyd¹, David Maslove¹, Stephanie Stbley¹ and Kenneth Rockwood⁵









- Doing Good

NON MALEFICENCE

- Avoiding harm

AUTOMONY

- Informed consent



- Doing Good

NON MALEFICENCE

- Avoiding harm

AUTOMONY

- Informed consent

Are elderly patients' opinions sought before admission to an intensive care unit? Results of the ICE-CUB study @

Julien Le Guen ☎; Ariane Boumendil; Bertrand Guidet; Aline Corvol; Olivier Saint-Jean; Dominique Somme

ONLY 12.7% (of a total of 2115) PATIENTS WERE CONSULTED ABOUT WISHES FOR ICU

Consent: patients and doctors making decisions together General Medical Council Making decisions when a patient lacks capacity 75 In making decisions when a patient lacks capacity 75 In making decisions when a patient lacks capacity 97 In making decisions when a patient lacks capacity 18 a make the care dyour patient your first concern 18 treat patients as individuals and respect their dignity 19 c support and encourage patients to be involved, as far as they want to and one able, indictions about the treatment and care to patients with respect and not discriminate against them. 76 You must also consider: a whether the patient's lack of capacity is temporary or permanent which reports of treatment used provide overall clinical baselfs for the patient's foure choices which option, including the option not to treat, would be lesst restrictive of the patient's fourer choices an explored statement or decision.



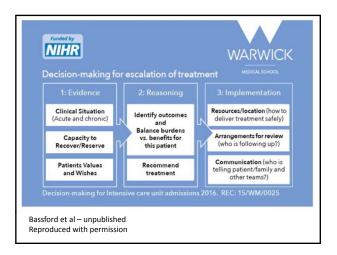
- Doing Good

NON MALEFICENCE

- Avoiding harm

AUTOMONY

- Informed consent



Back to our 2 patients with pneumonia

- 72 year old gentleman

- /2 year old gentleman
 Type 2 respiratory failure secondary to
 pneumonia
 Multiple comorbidities
 Poor exercise tolerance
 Suggests limited physiological reserve
 Social history, housebound and dependent
 on some ADLs
- High chance of not surviving current illness therefore risks of admitting to ICU likely to outweigh liklihood of survival let alone a return to the quality of life acceptable to the patient.
- 85 year old lady

- 85 year old lady
 Type 2 respiratory failure secondary to
 pneumonia
 No comorbidities
 "independent"
 But poor muscle mass and frail
 2 limited physiological reserve in view of age
 and frailty
 Will she we more than 34 days?
 Will she be able to ever get back to her
 current baseline in the event of significant
 critical illness?
- Possibly better chance of surviving acute pneumonia in the short term but may be difficult to weaning from a ventilator and concern about long term functional status if she does survive may need prolonged rehab. Will she be able to return home to independent living?
- Ascertain patients wishes

Don't forget....

PATIENTS NOT FOR INTUBATION/MECH **ANICAL** VENTILATION/ICU



Take home messages

- The goal of ICU is patient survival with a QOL that is acceptable to them
- ICU can be harmful to both patients and relatives
- Ethically guided decision making process should way up benefits v burdens
- Patient wishes should form part of decision making process where possible
- Frailty = increased vulnerability = decreased capacity to recover → ? Burdens > benefit

Thank you	