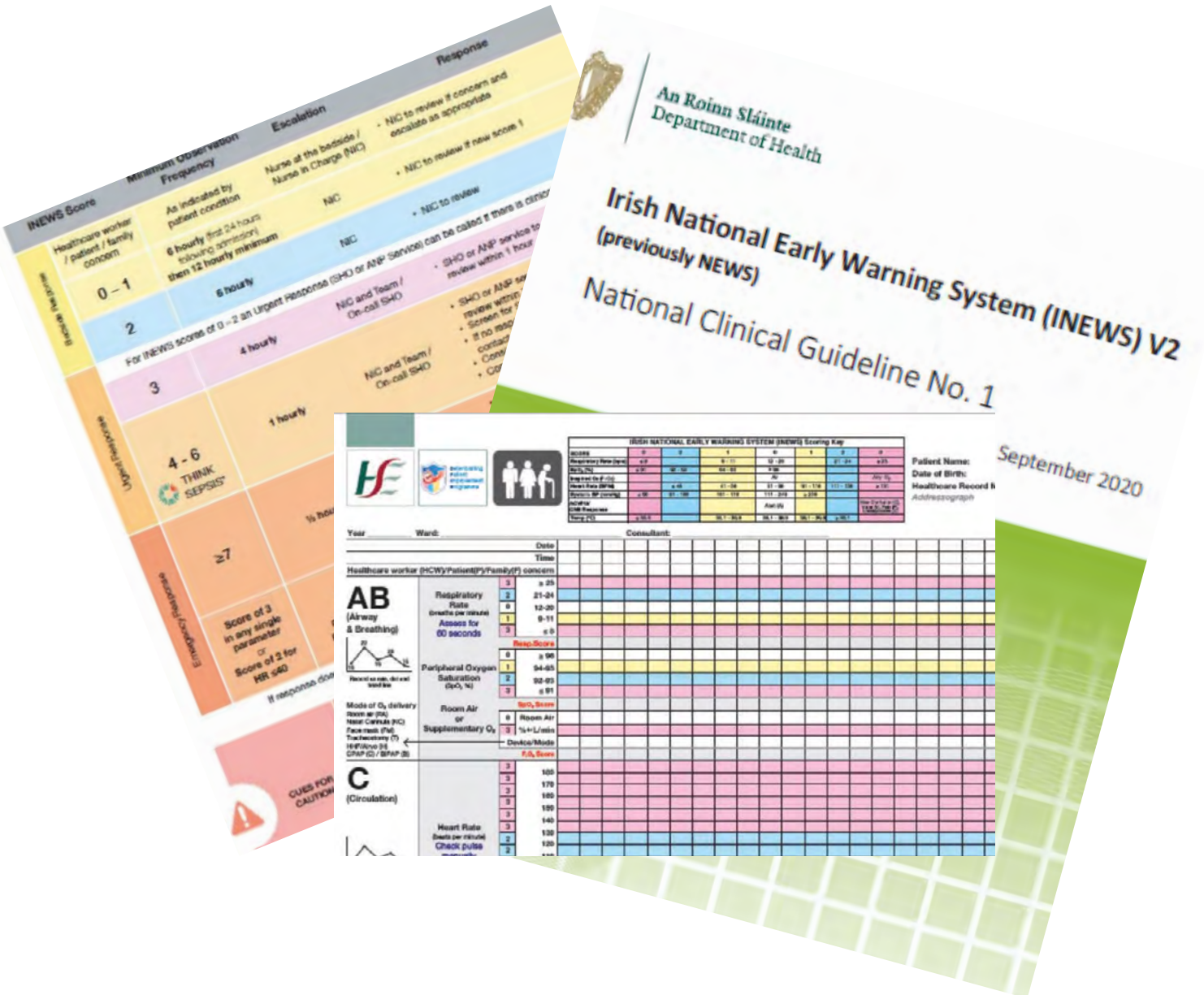
Deteriorating Patient Improvement Programme

Clinical Design & Innovation
Person-centred, co-ordinated care

The Irish National Early Warning System (INEWS) V2 User's Guide incorporating COMPASS©



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Disclaimer

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Neither the INEWS system nor the INEWS National Clinical Guideline replace professional judgement on particular cases, whereby the clinician or health professional decides that the system or individual INEWS guideline recommendations are not appropriate in the circumstances presented by an individual patient, or whereby an individual patient declines a recommendation as a course of action in their care or treatment plan. In these circumstances the decision not to follow a recommended course of action should be appropriately documented in the patient's healthcare record or nursing notes.
This INEWS User's Guide incorporating COMPASS is intended to support health care professionals in the provision of care to non pregnant adult in-patients in HSE, Voluntary and Private acute hospital services.

The NCEC National Clinical Guideline No. 1 INEWS V2 is available at the following link: https://www.gov.ie/en/collection/cc5faa-national-early-warning-score-news/

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Section 1 covers:

- Introduction to the Irish National Early Warning System (INEWS) V2

Introduction to the Irish National Early Warning System (INEWS) V2

Introduction

Acute physiological deterioration is a time-critical medical emergency and failure to detect and treat patient deterioration in a timely manner poses a threat to patient safety which may lead to adverse patient outcomes such as unanticipated cardiopulmonary arrest and death. Deterioration in a patient's condition in hospital is frequently preceded by measurable physiological abnormalities, often up to eight hours prior to deterioration. Regular measurement and documentation of vital signs and other observations is an essential requirement for recognizing clinical deterioration. Early Warning Systems (EWSs) use routinely recorded patient observations to assist in the recognition of patient deterioration.

Early Warning Systems (EWSs) were originally introduced as track and trigger tools used to quantify the severity of acute illness. In these earlier EWSs the focus was on the EWS as a score. EWSs have since evolved internationally. It has been recognized that, to be effective, a whole system approach is essential. This involves the original track and trigger tool, a clearly outlined escalation pathway, a consistent competent clinical response and evaluation at both clinical and organisational levels. INEWS V2 introduces a precursory step to the system – anticipation – whereby healthcare professionals operate from the perspective of expecting patients in hospital to deteriorate taking a 'situation awareness' approach using a series of 'cues for caution' to assist in patient assessment. This change from a score to a system is reflected in the new title - the Irish National Early Warning System (INEWS) V2.

INEWS V2 also highlights a concept known as 'the cycle of clinical futility'. The 'cycle of clinical futility' can be described as repeated patient reviews and/or interventions by (usually junior) nursing and medical staff with limited or no improvement in patient condition. Escalation of care for senior clinician review is indicated but not called for. Evidence shows that this failure to escalate care to more senior colleagues is common amongst healthcare staff and as such puts patients at risk of a serious adverse event.

Failure to escalate care occurs for a number of reasons including fear of appearing incompetent, fear of reprimand and a hospital culture where seeking help is not the norm (see pages 83 – 91 for more information).

A hospital culture which supports and encourages staff to seek advice and input from senior colleagues is vital to ensure an effective and robust patient safety culture.

KEY CLINICAL POINT
Acute physiological deterioration is a time-critical medical emergency

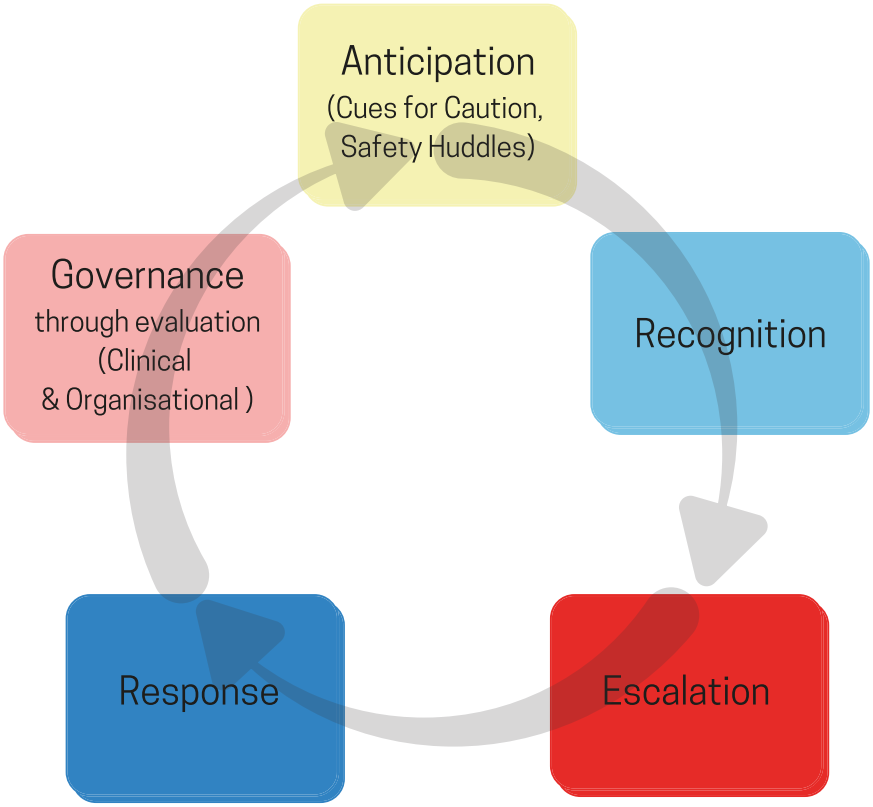


Figure 1 - INEWS System of Safe Care

The evidence-based, validated INEWS track and trigger tool, based on the ViEWS VitalPac© tool, uses six routinely recorded physiological observations plus supplemental oxygen to quantify the severity of acute illness. The INEWS observations are:

- Respiratory rate
- Oxygen saturation
- Room air or supplemental oxygen
- Heart rate
- Systolic blood pressure
- Level of consciousness (ACVPU)
- Temperature

INEWS outlines normal and abnormal ranges for each of the seven observations (Figure 2). A numerical score of between '0' and '3' is allocated to each of the seven parameters. A score of '0' represents the least risk and a score of '3' represents the highest risk. The INEWS values for each of the seven observations are added giving the patient's INEWS score.

IRISH NATIONAL EARLY WARNING SYSTEM (INEWS) Scoring Key							
SCORE	**3**	**2**	**1**	**0**	**1**	**2**	**3**
Respiratory Rate (bpm)	≤ 8		9 - 11	12 - 20		21 - 24	≥ 25
SpO_2 (%)	≤ 91	92 - 93	94 - 95	≥ 96			
Inspired O_2 (F_iO_2)				Air			Any O_2
Heart Rate (BPM)		≤ 40	41 - 50	51 - 90	91 - 110	111 - 130	≥ 131
Systolic BP (mmHg)	≤ 90	91 - 100	101 - 110	111 - 249	≥ 250		
ACVPU/ CNS Response				Alert (A)			New Confusion (C), Voice (V), Pain (P), Unresponsive (U)
Temp (°C)	≤ 35.0		35.1 - 36.0	36.1 - 38.0	38.1 - 39.0	≥ 39.1	

Figure 2 - INEWS Scoring Key

Anticipation

INEWS V2 emphasises an anticipatory approach to the management of patient deterioration. This approach means highlighting the role of situation awareness in the detection of clinical deterioration with a subsequent increased focus on those patients with low or 'no' INEWS scores. These patients are often the most vulnerable to unrecognized deterioration. Anticipatory care involves the use of situation awareness by staff, that is, 'knowing what is going on' for each patient so that the potential for deterioration can be detected and acted upon.

To support this anticipatory approach to deterioration a series of prompts, listed as 'Cues for Caution', have been included on the front of the INEWS patient observation chart (Figure 3) to assist staff in patient assessment. The clinical setting will determine other relevant 'cues for caution'.

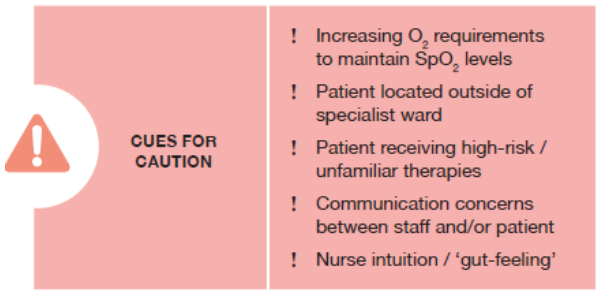


Figure 3 - Cues for Caution

A prompt to remind clinicians to think about the possibility of sepsis for INEWS scores of ≥ 4 (or ≥ 5 on oxygen) where there is a suspicion of infection is also included on the front of the INEWS patient observation chart (Figure 4).

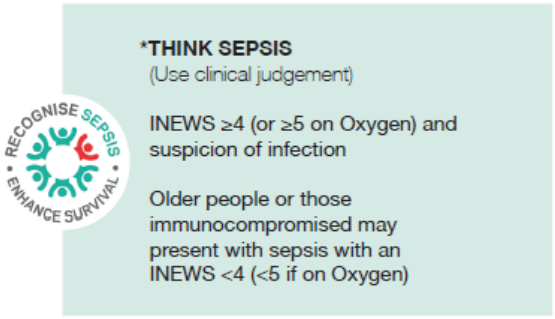


Figure 4 - Think Sepsis

The use of safety huddles in clinical practice is recommended in INEWS V2 to support this anticipatory approach to the management of deterioration.

Huddles are a goal oriented brief meeting of staff working together in order to share relevant information and agree actions. It is important that huddles remain focused and have a systematic process.

Huddles can enable teams to identify patients who have the potential to deteriorate and to flag concerns proactively.

It is important to monitor the effectiveness of Huddles.

Recognition

INEWS is a clinical assessment tool designed for use as an adjunct to clinical judgement. INEWS does not replace clinical judgement. The INEWS track and trigger tool is not used in isolation. If a healthcare worker is concerned about a patient even in the context of a low or 'no' INEWS score, care should be escalated for senior nursing or medical review as appropriate. As highlighted in the Paediatric Early Warning System (PEWS) User Manual 'there is no precise or specific threshold for any physiological parameter to identify deterioration. Rather, a trigger in one parameter or a gut feeling on the part of a healthcare professional should prompt information seeking from other parameters' (2017, p. 8).This emphasizes the need for clinical judgement on the part of the healthcare professional.

A colour-coded patient observation chart is used for documentation of physiological observations. Trend lines are documented between each set of observations facilitating the recognition of trends in patient observations (Figure 5).

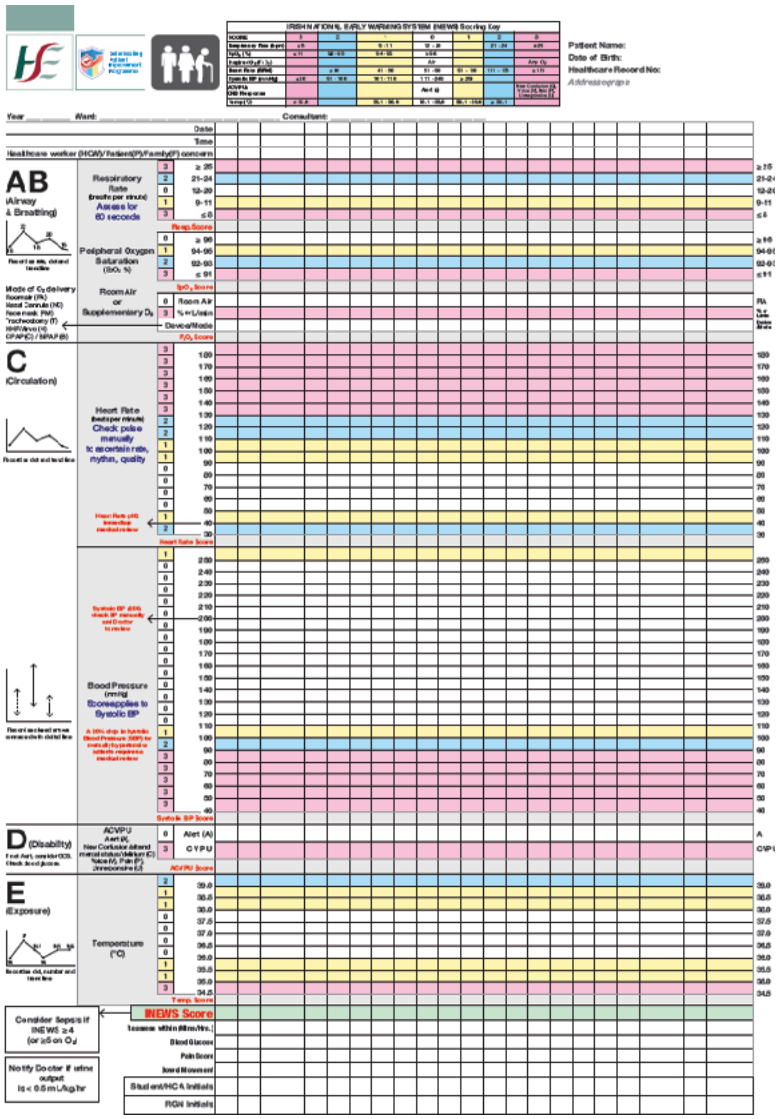


Figure 5 - INEWS patient observation chart

KEY CLINICAL POINT
INEWS does not replace clinical judgement; it is an adjunct to clinical judgement designed to aid clinical decision-making

Both the Glasgow Coma Scale (GCS) and a Numerical Pain Scale are now included in the INEWS patient observation chart. These were included after a review of patient observation charts in use in 29 of the acute hospitals across the country revealed that the majority of hospitals had added the GCS and pain scale to their charts. During the review it was observed that there can be differences between neurological observations charts in use in different hospitals. The neurological observations chart included in the national INEWS patient observation chart can therefore be replaced by charts in use locally.

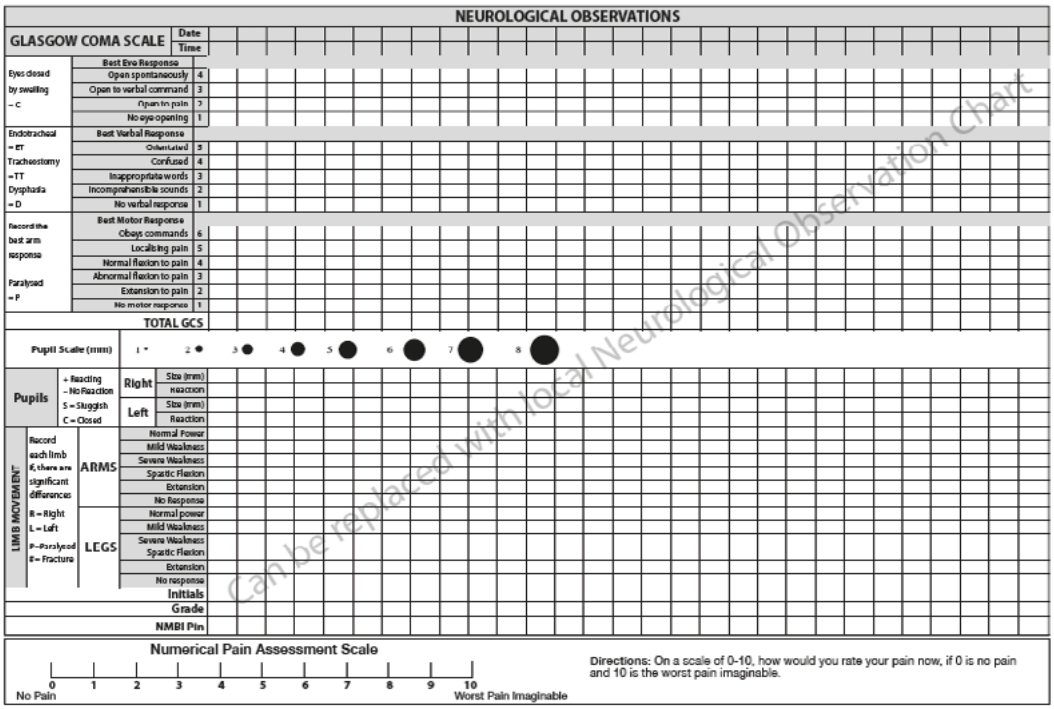


Figure 6 - Neurological observations chart and numerical pain scale

Escalation and Response

The INEWS Escalation and Response Protocol provides guidance on minimum frequency of observations monitoring, the level of escalation required for INEWS scores and the appropriate clinical response (Figure 7).

INEWS Escalation & Response Protocol

	INEWS Score	Minimum Observation Frequency	Escalation	Response
Bedside Response	Healthcare worker / patient / family concern	As indicated by patient condition	Nurse at the bedside / Nurse in Charge (NiC)	• NiC to review if concern and escalate as appropriate
	0 – 1	**6 hourly** (first 24 hours following admission) **then 12 hourly minimum**	NiC	• NiC to review if new score 1
	2	**6 hourly**	NiC	• NiC to review
Urgent Response	For INEWS scores of 0 – 2 an Urgent Response (SHO or ANP Service) can be called if there is clinical concern			
	3	**4 hourly**	NiC and Team / On-call SHO	• SHO or ANP service to review within 1 hour
	4 - 6 THINK SEPSIS*	**1 hourly**	NiC and Team / On-call SHO	• SHO or ANP service to review within ½ hour • Screen for Sepsis* • If no response to treatment within 1 hour, contact Registrar and/or ANP service • Consider continuous patient monitoring • Consider transfer to higher level of care
Emergency Response	**≥7**	**½ hourly**	NiC and Team / On-call Registrar Inform Team / On-call Consultant	• Registrar / Consultant / ANP service to review immediately • Continuous patient monitoring recommended • Plan to transfer to higher level of care • Activate Emergency Response System (as appropriate to hospital model)
	Score of 3 in any single parameter or **Score of 2 for HR ≤40**	**½ hourly** or **as indicated by patient condition**	NiC and Team / On-call SHO	• SHO or ANP service to review immediately • If no response to treatment or if still concerned, contact Registrar/Consultant • Consider activating Emergency Response System

If response does not occur as per protocol the CNM/NiC should contact the Registrar or Consultant

Figure 7: INEWS Escalation and Response Protocol

For the first 24 hours following admission to an acute hospital a patient should be monitored six-hourly at a minimum. This minimum frequency of observations monitoring acknowledges the vulnerability of patients in the early stages of admission. Patients admitted to the acute setting with exacerbations of their chronic conditions are in an acute phase of their illness therefore are acutely unwell and require close monitoring. A nurse or doctor can determine if more or less frequent monitoring is appropriate based on a patient's clinical condition.

KEY CLINICAL POINT
It is essential that staff familiarise themselves with their local hospital's INEWS Escalation & Response Protocol

KEY CLINICAL POINT
An urgent or emergency response can be initiated for patients with low or 'no' scores if the nurse at the bedside believes the patient's condition warrants it

Guidance is also provided in the INEWS Escalation and Response Protocol on when to call for a review of care and to whom a patient's care should be escalated, that is, nursing or medical review. A three-tiered response is outlined – bedside, urgent and emergency. An urgent or emergency response can be initiated for patients with low or 'no' scores if the nurse at the bedside believes the patient's condition warrants it.

Research evidence suggests that many barriers exist to the escalation of care by staff.These barriers include but are not limited to fear of reprimand by senior personnel, fear of looking stupid, behaviour of response team members during previous escalations of care, lack of clear policies and protocols for escalation of care and/or staff lack of knowledge of these protocols, over-confidence of own clinical ability leading to disregarding of INEWS score, and inexperience. A clinical environment which supports the psychological safety of staff is necessary to enhance the appropriate escalation of care.

KEY CLINICAL POINT
The INEWS parameters or a patient's INEWS score must not be altered

Both the INEWS Guideline Development Group (GDG) and the Consultant Advisory Group (CAG) considered the issue of parameter adjustment and INEWS score adjustment which had become accepted practice in the majority of sites after the introduction of NEWS in 2013. A Quality Assurance and Verification (QAV) healthcare audit of nine hospitals (73 healthcare records) in 2017/2018 demonstrated that when INEWS scores or parameters were adjusted these were rarely – if ever – reviewed with subsequent poor patient outcomes. Adjusting INEWS parameters and/or scores effectively means taking a patient off the early warning system leaving them vulnerable to unrecognized deterioration. Therefore the CAG made the decision to strongly recommend that INEWS parameters or scores must not be adjusted in INEWS V2.

Given that INEWS parameters and scores cannot be adjusted the CAG acknowledged the potential for over-triggering and alarm fatigue in some situations. The solution to address this challenge for practice was to include the option for a Registrar or Consultant, that is, a senior clinical decision maker, to document a Modified Escalation and Response Protocol in instances where they deem it appropriate to the patient's clinical condition.

The Modified Escalation and Response Protocol should not be used in the first 24 hrs following admission. The rationale for use, along with time and date of next review (no greater than 24 hourly) should be documented. A Modified Escalation and Response Protocol is documented on the back of the INEWS patient observation chart (Figure 8). For the majority of patients however the standardized INEWS Escalation and Response Protocol will be appropriate.

Modified INEWS Escalation and Response Protocol (to be completed by Consultant or Registrar only)
Not for use within first 24 hours of admission

	Date Year:	Time (use 24hr clock)	Rationale and Instructions/Interventions	Next medical review	Doctor (Signature and MCRN)
Start	20/05	0400	Imp: Chronic COPD, admitted > 24 hours ago Stable with RR 22, SpO_2 92%, O_2 2L/min (INEWS score 7) Escalate if change in RR or increased O_2 requirement to maintain SpO_2 treatment target of 92%.*	Maximum 8 hours (10am) or at ward round or sooner if concern	Dr. A Medical Registrar MCRN 1234567
End	20/05	1000			
Start	20/05	1000	Reviewed – continue as above.	24 hours or sooner if concern	Dr. A Medical Registrar MCRN 1234567
End	21/05	1000			
Start	/				
End	/				
Start	/				
End	/				
Start	/				
End	/				

*Text within sections above is provided as example only - please write over the watermark

Figure 8: Modified Escalation and Response Protocol

During the review process of the NEWS (2013) guideline it became apparent, through evidence from the literature and focus groups that there was a perception that the introduction of NEWS undermined clinical judgement. As a result the role of clinical judgement was emphasized in INEWS V2 and is reflected in several key INEWS V2 recommendations. One such recommendation allows for a Registered General Nurse (RM/ RCN /RPN/ RNID) with the appropriate clinical competencies to defer escalation for a short period of time (maximum 30 minutes) if the cause of patient discomfort or a new INEWS score is likely to be easily remediable. The rationale for the deferral of escalation, along with the timeframe for review must be clearly documented in the appropriate section of the INEWS patient observation chart (Figure 9). If the patient's symptoms do not resolve, or the INEWS score remains unchanged or increases escalation occurs as per protocol.

Deferred Escalation (to be completed by Registered General Nurse (RGN))

Date/Time (use 24hr clock)	Rationale and Interventions	Review at 30 minutes	Nurse (Signature and NMBI PIN)
25/05/20 @ 0400	Imp: Decrease in SpO_2 to 91%, on 2L/min O_2 via n/prongs, patient lying flat, patient states they feel okay Intervention: patient repositioned and n/prongs adjusted. Repeat observation and review decision at 30 minutes. NIC informed.*	0430 hours: SpO_2 back up to 96% on 2L/min O_2. No need for escalation	Nurse Brown (PIN 12345)
/ / @			
/ / @			
/ / @			

*Text within sections above is provided as example only - please write over the watermark

Figure 9: Deferred Escalation by an RGN

KEY CLINICAL POINT
The Modified Escalation and Response Protocol should not be used in the first 24 hrs following admission

Governance

Closed loop governance completes the INEWS system of care. This involves:

Bedside governance -

- Post-escalation/awaiting response team: maintain monitoring and surveillance of patient
- Post-response: re-evaluate interventions to determine effectiveness
- Avoid 'cycle of clinical futility' – escalate to more senior clinician if no response or limited response to intervention(s)

Organisational governance –

- A Consultant clinical champion providing clinical leadership
- A Deteriorating Patient Committee reporting to Hospital Executive Management Team
- Agreed schedule of audit
- Measurement of clinical outcomes e.g. Unplanned admission / readmission to ICU, cardiopulmonary arrest, incidence of non-invasive ventilation
- Agreed education and training schedule

Section 2 covers:

- Documenting physiological observations on the INEWS Patient Observation Chart (INEWS Track and Trigger Tool)

Section Two

Documenting physiological observations on the INEWS Patient Observation Chart (INEWS Track and Trigger Tool)

Documenting physiological observations on the INEWS Patient Observation Chart (INEWS Track and Trigger Tool)

Introduction

Poor recording of patient observations and lack of escalation of care when physiological observations are abnormal may result in poor patient outcomes, such as unplanned admission to ICU, unanticipated cardiopulmonary arrest and/or death. Measuring, monitoring and surveillance of patient observations is an essential element of patient assessment. Physiological observations are interpreted in the context of presenting clinical features and assist clinicians in determining the level of care required by a patient. When planning workloads time allocated to monitoring of patient's observations must be prioritized.

KEY CLINICAL POINT
INEWS does not replace clinical judgement; it is an adjunct to clinical judgement designed to aid clinical decision-making

The INEWS patient observation chart is the track and trigger tool within the INEWS system of care.
A track and trigger tool facilitates the:

- Recording of physiological observations
- Trending of the patient's physiological observations
- Calculation of a patient's INEWS score
- Triggering of escalations of care as appropriate

This section provides detail on how the INEWS physiological observations should be recorded on the INEWS patient observation chart.

While the focus on this section is on recording INEWS physiological observations it is important to assess the patient holistically.

Healthcare worker, Patient or Family Concern

We begin with a new addition to the INEWS chart – *healthcare worker, patient or family concern*. While 'concern' is not assigned an INEWS score, documentation of 'concern' on the INEWS patient observation chart acknowledges that concern is present and acts as a prompt for subsequent escalation of care as appropriate.
The inclusion of 'Healthcare worker, patient or family concern' is new to INEWS V2 and the presence or absence of concern should be recorded with each set of INEWS observations.

How to assess HCW, Patient or Family Concern

If a healthcare worker is concerned about the patient or the patient themselves or the patient's family express concern this should be recorded and escalated appropriately

How to record HCW, Patient or Family Concern

Record 'no concern' as '0'

Record Healthcare worker concern as 'HCW' or 'H'

Record patient concern as 'P'

Record family concern as 'F'

Healthcare worker (HCW)/Patient(P)/Family(F) concern	0	H	F	P	0

All 7 INEWS physiological observations are completed and scored to obtain a patient's INEWS score. The INEWS physiological observations are detailed below.

Respiratory Rate

A change in a patient's respiratory rate (RR) is one of the key early indicators of clinical deterioration yet RR is the most neglected vital sign. RR is more often estimated than counted by healthcare staff even where the primary diagnosis is respiratory. An increase of 3- 5 breaths per minute (bpm) can indicate acute deterioration. An increase of 3 to 5 bpm within the normal parameter range of 12 to 20 bpm could indicate acute deterioration.

How to assess Respiration Rate

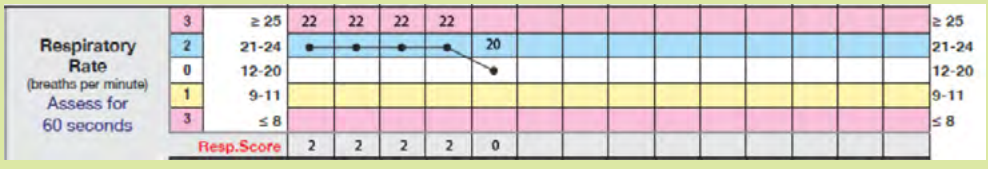
Count breaths for 60 seconds.

How to record Respiration Rate

Enter RR count in the corresponding row.

Place a dot in the corresponding row & join the dot to the previous dot using a line to indicate RR trend.

Enter the INEWS value (0, 1, 2 or 3) for RR in the grey row below the RR section.



KEY CLINICAL POINT

A change in a patient's respiratory rate (RR) is one of the key early indicators of clinical deterioration

Peripheral Oxygen Saturation (SpO_2)

Peripheral oxygen saturation or SpO_2 is an approximation of the level of oxygen in the blood. Specifically SpO_2 is the percentage of oxygenated haemoglobin (Hb) in the blood compared to the total amount of haemoglobin in the blood. SpO_2 is an estimate of arterial oxygen saturation levels. It should be noted patients with a low Hb might have a "normal" SpO_2 reading, however, may not have adequate tissue oxygenation.

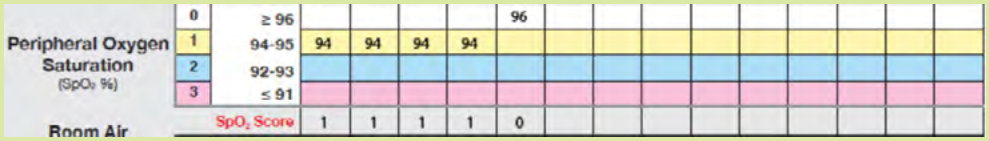
How to assess Peripheral Oxygen Saturation

Use pulse oximeter to measure oxygen saturation.

How to record Peripheral Oxygen Saturation

Enter % value in the corresponding row.

Enter the INEWS value (0, 1, 2 or 3) for SpO_2 in the grey row below SpO_2 section.



Peripheral Oxygen Saturation (SpO_2 %)	0	≥ 96					96								
	1	94-95	94	94	94	94									
	2	92-93													
	3	≤ 91													
Room Air		SpO_2 Score	1	1	1	1	0								

KEY CLINICAL POINT

A patient's new requirement for supplemental oxygen **or** a need for increasing levels of supplementary oxygen to maintain their SpO2 level is a clear sign of acute clinical deterioration and requires urgent medical review

Room Air or Supplementary Oxygen (FiO_2)

A patient's new requirement for supplemental oxygen or a need for increasing levels of supplementary oxygen to maintain their SpO_2 level is a clear sign of acute clinical deterioration and requires urgent medical review. This is often not recognized as a sign of acute deterioration as the patient's oxygen saturation level may be maintained due to the increase in supplemental oxygen.

How to assess FiO_2

Is the patient on room air?

Or do they need supplementary oxygen?

How to record FiO_2

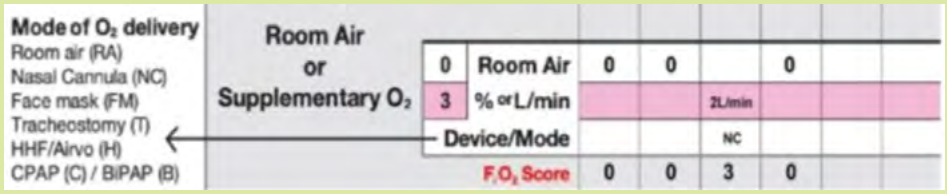
If on 'room air' record "0" into Room Air Box

If on Supplementary O_2 enter either

- L/min of O_2 being delivered or
- % of O_2 being delivered

Record the device in use as per legend e.g. 'RA' for 'Room Air' or 'NC' for 'nasal cannulae'

Enter the INEWS value (0, 1, 2 or 3) for FiO_2 in the grey row below FiO_2 section



Heart Rate

The section for documenting heart rate is now located before the blood pressure section as a result of chart review and staff feedback.

How to assess Heart Rate

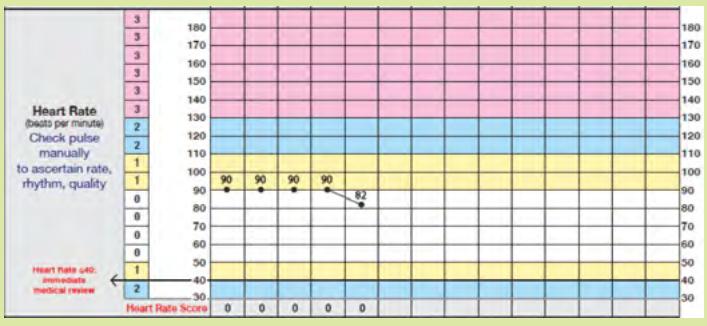
Palpate and count the patient's pulse for 60 seconds.

How to record Heart Rate

Record the HR by placing a dot in the corresponding row

Join the dot to the previous dot using a line to indicate HR trend

Enter the INEWS value (0, 1, 2 or 3) for HR in the grey row below HR section



KEY CLINICAL POINT
Manual palpation of pulse is recommended to ascertain rate, rhythm and quality

Blood Pressure

In INEWS the systolic blood pressure determines the INEWS value for blood pressure. The INEWS parameter range for a '0' score systolic blood pressure is broad. This is not to say that a systolic blood pressure of, for example 240mmHg is 'normal'. Rather the focus in INEWS is on hypotension as being the more likely scenario in a clinically deteriorating patient. If a patient's systolic blood pressure is higher than normal for that patient, or more specifically is greater than 200mmHg the blood pressure should be checked manually and a medical review undertaken

KEY CLINICAL POINT
A 20% drop in Systolic BP for normally hypertensive patients requires a medical review

How to assess Blood Pressure

Record blood pressure measurement. Record both systolic and diastolic readings

The systolic blood pressure (SBP) provides the INEWS value.

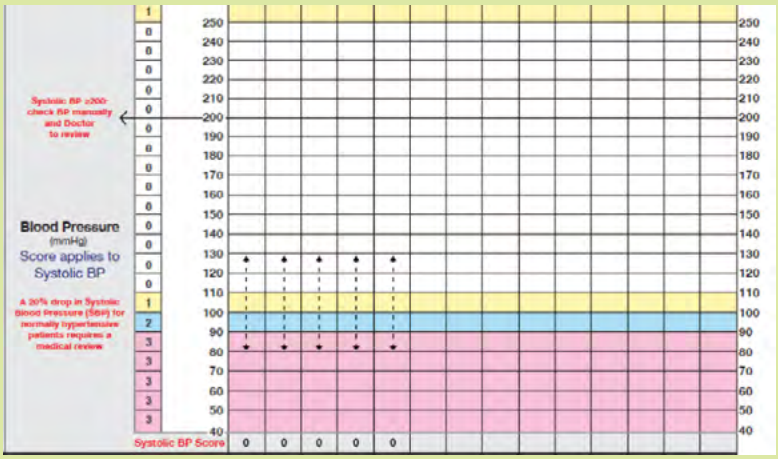
How to record Blood Pressure

Symbol

Systolic Reading

Diastolic Reading

Enter the INEWS value (0, 1, 2 or 3) for SBP in the grey row below BP section



ACVPU (previously AVPU)

In INEWS V2 changes in a person's mental status such as delirium or new confusion are recognized as key early signs of deterioration. A change in a patient's mental status requires an immediate medical review. In INEWS V2, AVPU becomes ACVPU where 'C' represents 'new confusion, altered mental status or delirium. Confusion should be considered to be 'new' until proven otherwise. A worsening confusion should always prompt concern and warrants urgent medical review. As acute confusion is a potent marker of clinical risk and acute illness severity, the possibility of 'new confusion' should be routinely considered (Williams 2019) ie with every set of INEWS observations completed.

How to assess ACVPU

Assess the neurological response using the ACVPU scale, only one outcome is recorded:

- If fully awake and talking to you the patient is alert , record A (Alert).
- If the patient is experiencing new confusion, altered mental status or delirium record C (New Confusion).
- If the patient is not fully awake and responds to voice only record V (Voice)
- If the patient does not respond to voice, administer a painful stimulus such as a trapezium squeeze and check for a response (eye opening, verbal such as moaning, or movement); if the patient responds to the painful stimulus record P (Pain).
- If the patient is unresponsive record U (Unresponsive).

How to record ACVPU

Record associated letter (ACVP or U) in relevant box.

Enter the INEWS value (0 or 3) for ACVPU in the grey row below ACVPU section.

Confusion should be considered to be 'new' until proven otherwise (Record 'C' and score '3').

If the patient's confusion is known to be their 'lived baseline' and they are 'Alert' (ie not considered to be 'V', 'P' or 'U') then record 'A' and score '0'.

If on admission it is unknown whether the confusion is new or a 'lived baseline' record 'C' and score '3'. However, if it is subsequently determined that the confusion is the patient's 'lived baseline' revert to 'A' for 'Alert' (ie if not considered to be 'V', 'P' or 'U') and a score of '0'.

ACVPU Alert (A), New Confusion/altered mental status/delirium (C) Voice (V), Pain (P), Unresponsive (U)	0	Alert (A)	A	A	A	
	3	C V P U				C
		ACVPU Score	0	0	0	3

KEY CLINICAL POINT
Changes in a person's mental status such as delirium or new confusion are recongised as key early signs of deterioration

KEY CLINICAL POINT
The possibility of *'new confusion'* should be routinely considered with every set of INEWS observations completed

Temperature

INEWS parameters identify a temperature of between 36.1^{O}C to 38.0^{O}C as being within the normal range for an adult patient. Extremes of temperature (high or low) are sensitive markers of acute illness severity. Sepsis can also present with a low or very low temperature

KEY CLINICAL POINT
If you are concerned about a patient escalate care even if patient's INEWS score is low or where there is no score

How to assess Temperature

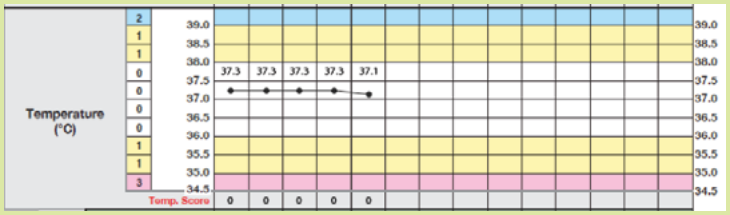
Record temperature measurement as per local hospital policy.

How to record Temperature

Record the value by placing a dot and exact temperature reading in the corresponding row

Join the dot to the previous dot using a line to demonstrate trend.

Enter the INEWS value (0, 1, 2 or 3) for temperature in the grey row below Temperature section.



INEWS Score

Calculate the patient's INEWS score by adding the INEWS values for each of the seven observations. To obtain an accurate INEWS score a full set of observations must be recorded on all occasions. Once the INEWS score has been calculated follow the INEWS Escalation and Response Protocol for appropriate action.

Below is an example of four complete sets of documented INEWS observations. The INEWS score has been calculated for each set of observations (Figure 10).

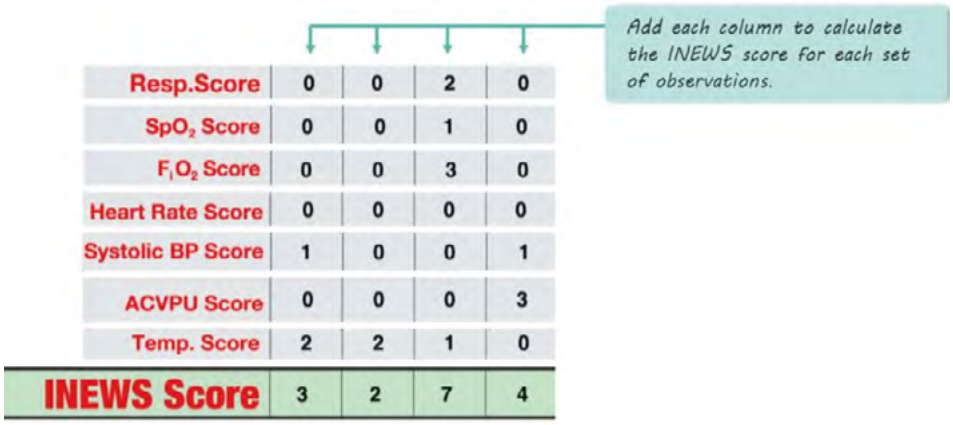


Figure 10 - Calculation of the INEWS score

KEY CLINICAL POINT

If you are concerned about a patient escalate care even if patient's INEWS score is low or where there is no score

KEY CLINICAL POINT

Some medications may affect physiological parameters, know your patient's medications and the potential a drug may have to impact on their parameters

Summary

THE INEWS OBSERVATIONS ARE:	• Respiratory rate • Oxygen saturation • Room air or Supplementary oxygen • Heart rate • Systolic blood pressure • Level of consciousness • Temperature
OBSERVATIONS MONITORING	• Is fundamental to patient assessment • Assists in identifying deterioration or improvement in a patient's condition • Helps to determine the level of care required
A FULL SET OF INEWS OBSERVATIONS SHOULD BE:	• Considered an essential element of patient assessment • Documented clearly and accurately • Part of the track and trigger element of INEWS • Inclusive of documentation of healthcare worker, patient or family concern if present • Measured 6 hourly X 24 hours after admission and 12 hourly thereafter if patient's condition dictates • Recorded on admission and thereafter to provide patient's baseline and trend

Section Three

Respiration

This section covers:

- Overview of Anatomy and Physiology of Respiration
- Respiratory rhythm and chest movement
- Baseline Respiratory Rate
- Effect of ill health on baseline Respiratory Rate
- Measuring Respiration Rate
- Physiological Oxygen Delivery
- Oxygen Delivery Systems for Supplementary Oxygen
- Monitoring and Titrating Oxygen Therapy
- Measuring Arterial Oxygenation
- Escalation of Care & Clinical Judgement
- Automated Respiratory Rate Monitoring
- Oxygen Delivery In Chronic Obstructive Pulmonary Disease (COPD)

The following section is based, with permission, on a series of six articles from the Nursing Times and COMPASS©

Introduction

Respiratory rate (RR), or the number of breaths per minute, is a clinical sign that represents ventilation (the movement of air in and out of the lungs). A change in RR is often the first sign of deterioration from any number of causes as the body attempts to maintain oxygen delivery to the tissues.

KEY CLINICAL POINT
A change in respiratory rate is one of the earliest signs of deterioration

Failing to recognise this early sign of deterioration can result in poor outcomes for patients. A HRB-CICER systematic review of the literature on early warning systems found that more than half of patients who had a serious adverse event could have been identified as high-risk of clinical deterioration up to 24 hours prior to the event based on changes in vital signs. Studies also showed that increased RR can be used to help predict patients at risk of cardiac arrest and emergency admission to ICU. (HRB-CICER 2019)

Overview of Anatomy & Physiology of Respiration

To understand the role of respiration in clinical deterioration a brief review of the anatomy and physiology of respiration is useful.

KEY CLINICAL POINT
Respiratory rate should be interpreted alongside other aspects of respiration (for example the depth, pattern and effort of breathing), other vital signs and presenting clinical features

Breathing has two essential components – ventilation and gaseous exchange:

- Ventilation is the process of physically moving air in and out of the lungs (measured by RR); and
- Gaseous exchange is the process of getting oxygen (O_2) into the body and carbon dioxide (CO_2) out.

The lungs are made up of large and small airways. The trachea is the largest airway and the bronchioles are the smallest. The bronchioles terminate in tiny air sacs called alveoli. Alveoli are the site of gas exchange and their presence increases as the airways become smaller. This allows for the total surface area of the lung to increase exponentially allowing maximum opportunity for gas exchange. Central and peripheral chemo receptors sensitive to hypoxia (low O_2 levels) and hypercapnia (increased CO_2) control the drive to breathe (Davies and Moore 2010).

Ventilation

Air naturally moves from an area of high pressure to an area of low pressure. During normal breathing, inspiration occurs by the contraction and flattening of the diaphragm and the contraction of the external intercostal muscles, causing a rise and outward movement of the ribcage. This increases the size of the thoracic cavity, the lungs expand, drawing air in. Expiration at rest is a largely passive process; inspiratory muscles relax and there is elastic recoil of the lungs giving rise to a state of pressure equilibrium before the cycle begins again (Bourke and Burns 2015). This movement of the chest wall (ventilation) is observed when respiratory rate (RR) is measured. Changes in RR occur in response to exercise, emotions and during sleep; those changes in RR associated with exercise and anxiety may be greater than 25 breaths per minute but will usually return to normal in a resting, calm state.

KEY CLINICAL POINT
A change in a patient's normal respiratory rate of even 3 to 5 bpm may be an early sign of deterioration

Gaseous exchange

The process of ventilation delivers air to the alveoli where gaseous exchange occurs by a simple process of diffusion. A gas will move from an area of high concentration to an area of low concentration. Oxygen moves into the bloodstream while carbon dioxide is removed and exhaled. Once oxygen is in the alveoli, it diffuses across the thin alveolar capillary membrane into the blood, and attaches to haemoglobin. From here, it is dependent on the pulmonary and then the systemic blood circulation to move the oxygen to the tissues and cells where it is required.

KEY CLINICAL POINT
RR measures ventilation while pulse oximetry measures oxygen saturation

An increase in a patient's respiratory rate can reflect either a drop in arterial blood oxygen saturation level or reflect compensation for the presence of metabolic acidosis. Respiratory rate may therefore be an important indicator of inadequate oxygen delivery to the tissues and therefore a marker of a deteriorating patient. As oxygen delivery to the tissues is reduced, cells revert to anaerobic metabolism. This increases the lactate production, resulting in the accumulation of lactic acid. This accumulation of lactic acid stimulates an increase in respiratory rate (tachypnoea).

The decrease in oxygen delivery to the tissues, which results in tachypnoea, can be due to problems at any point in the oxygen delivery chain (Figure 11).

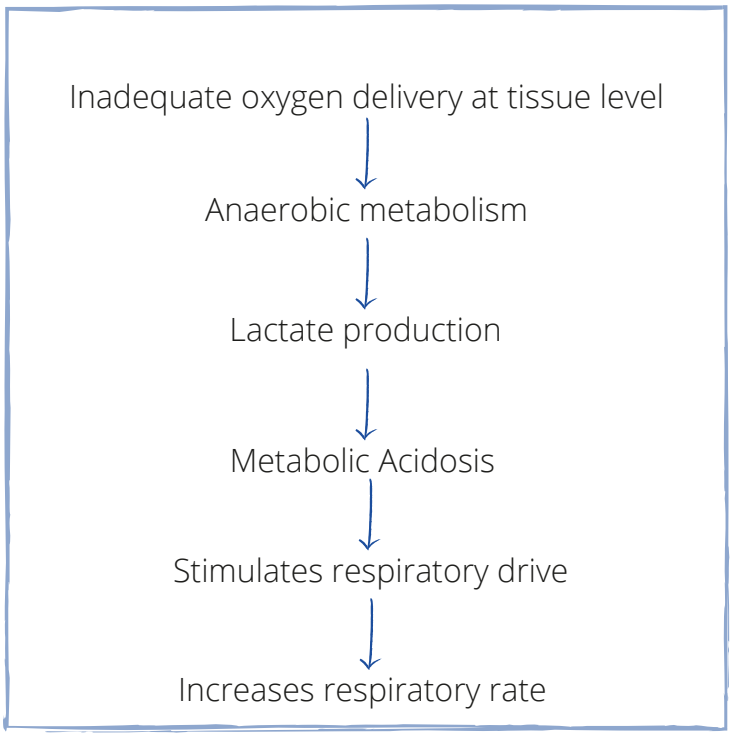


Figure 11 - Oxygen Delivery Chain

Respiratory rhythm and chest movement

Breathing rhythm and chest movement provide key information on a patient's condition and are observed in conjunction with counting the RR. It is important to observe the RR and to examine the rhythm of breathing and movement of the chest when conducting a respiratory assessment. This observation can aid rapid diagnosis and treatment particularly in patients who are acutely ill.

When there is an increased demand on the respiratory system from an acute episode, such as a chest infection, or long-term conditions, such as chronic obstructive pulmonary disease, the respiratory rhythm and chest movement change. These changes are compensatory mechanisms as a direct result of a chemical imbalance; the primary cause may be:

- mechanical
- metabolic
- neurological

The changes result in an increase or decrease in RR, depth of breathing and pattern of breathing. Changes in rhythm and chest movements are made through feedback mechanisms to the central respiratory control centres of the brain.

A range of receptors provide information that is interpreted in the higher respiratory centre, modulating RR and chest movement (Feldman and Del Negro 2006); these receptors are:

- Peripheral chemoreceptors found in the carotid artery which detect changes in PaO_2 in the blood as well as $PaCO_2$ and pH;
- Central chemoreceptors in the ventral medullary surface of the medulla oblongata in the brain which detect pH changes;
- Mechanoreceptors are stretch receptors located in the smooth muscle of the main airways and parenchyma. They respond to excessive stretching of the lung during inspiration and send signals to the apneustic centre of the pons (located in the brain stem); the pons controls inspiration and expiration.

KEY CLINICAL POINT
Breathing has two essential components – ventilation and gaseous exchange

Normal Respiration

In relaxed normal breathing the RR is 12-20 breaths per minute (bpm) (DoH 2020). Chest expansion on inspiration should be the same or similar on each breath. The chest wall is symmetrical, accessory (neck and shoulder) muscles are not used, diaphragm muscles are functioning, and there is no paradoxical movement – the chest and abdomen move in the same direction on inspiration and expiration.

KEY CLINICAL POINT
A person's baseline RR is the number of breaths per minute at rest

Abnormal Respiration

There are several reasons why respiratory rhythm and chest movement may change. Abnormality in respiratory rhythm may be related to changes in the patient's metabolic state; for example, a patient with diabetic ketoacidosis may exhibit signs of rapid, deep breaths. Such breathing (often called Kussmaul's breathing) aims to reduce the level of CO_2 in the blood to maintain a normal pH and re-establish a homoeostatic state. Patients with chest pain may have rapid but shallow breaths because deep breaths cause discomfort; in patients with rib fractures adequate pain relief is paramount to restore a normal depth and rate of breathing.

Table 1 outlines common respiratory / breathing rhythm patterns, monitoring of which will become more readily available with the introduction of automated respiratory rate monitoring. Table 2 details key respiratory changes and possible causes.

Table1: Breathing Patterns

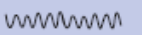
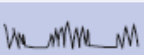
Pattern	Condition	Description
	Eupnoea	Normal breathing rate and pattern
	Tachypnoea	Increased respiratory rate
	Bradypnoea	Decreased respiratory rate
	Apnoea	Absence of breathing
	Hyperpnoea	Increased depth and rate of breathing
	Cheyne-Stokes	Gradual increases and decreases in respirations with periods of apnoea
	Biot's	Abnormal breathing pattern with groups/clusters of rapid respiration of equal depth and regular apnoea periods
	Kussmaul's	Tachypnoea and hyperpnoea
	Apneustic	Prolonged inspiratory phase with a prolonged expiratory phase

Table 2: Causes of Abnormal Respiratory Rate

Observation	Respiratory Changes	Possible causes
Chest Asymmetry	One side of the anterior chest moves more with normal tidal breaths than the other	Unilateral consolidation Pneumothorax Pleural effusion Fractured ribs (flail chest) Blocked chest drain Partial diaphragmatic paralysis Sputum plugging
Paradoxical chest and abdominal movement	Chest moves in opposite direction to the abdomen during normal tidal breathing	Neuromuscular disorder Spinal injury Diaphragmatic paralysis
Rapid and increased depth of breathing	Tachypnoea and deep inspiratory breaths	Metabolic acidosis such as diabetic ketoacidosis (Kussmaul's breathing) or renal failure Sepsis After exercise
Rapid and shallow depth of breathing	Tachypnoea and shallow inspiratory breath	Chest pain Abdominal pain Fractured ribs (pain) Sleep-disordered breathing pattern Cerebral lesion Shock Anxiety/stress Medication
Slow and increased depth of breathing	Bradypnoea with deep tidal breath, for example: • Apneustic breathing • Cheyne-Stokes respirations • Biot's respiration	Brainstem lesion, impending death Damage to the pons (respiratory centre in the brainstem that controls breathing) Congestive heart failure, neurological insult (after, for example, a stroke) Elevated intracranial pressure, for example, meningitis Sleep apnoea
Slow and shallow depth of breathing	Bradypnoea with shallow tidal breath	Neuromuscular disorders Opioid toxicity Hypopnoea (a partial blockage of the airway resulting in airflow reduction of >50% for ≥10 seconds) Hypothyroidism

Baseline Respiratory Rate

A person's baseline RR is the number of breaths per minute at rest. In INEWS the normal adult RR is considered to be between 12 and 20 breaths per minute. When ventilation and gas exchange occur as the result of normal respiration, the normal range of oxygen saturation of the blood (SpO_2) is 94-98% (O'Driscoll et al. 2017). The normal range for SpO_2 in INEWS, based on the validated, evidence-based prediction model for clinical deterioration, that is, ViEWS, is ≥ 96%.

Effect of ill health on baseline Respiratory Rate

In respiratory conditions where ventilation and/or gaseous exchange is impaired at rest, the hypoxic and hypercapnic drives will increase RR to maintain SpO_2.

- Poor gas exchange, as seen in conditions such as pulmonary fibrosis or emphysema (caused by a thickening of the alveolar wall and destruction of the lung tissue respectively), results in a higher resting RR. It is therefore important to consider the patient's 'normal, lived' baseline.

- Common obstructive lung conditions such as chronic obstructive pulmonary disease or asthma are characterised by an increased resistance to airflow as the small airways are narrowed, reducing oxygen delivery to the aveoli. During acute exacerbations their resistance is increased leading to a rise in RR. Administering bronchodilators relaxes the smooth muscle in the wall of the airways reducing resistance and returning the RR to normal ranges.

- Neuromuscular conditions affecting the lungs often lead to hypoventilation as the mechanisms needed for normal ventilation do not function properly. In this case a low RR (bradypnoea) can lead to respiratory failure.

- During surgery and post-operative recovery, RR must be monitored closely as anaesthetic agents, which usually contain opioids, can depress respiration and reduce RR (Koo and Eikermann 2011). They act on the central chemoreceptors suppressing the drive to breathe.

KEY CLINICAL POINT
Pulse oximetry is less accurate when SpO_2 is <80% (Jubran 2006)

Measuring Respiratory Rate

Measuring RR is acknowledged to be a core nursing skill, yet evidence suggests that it is an under-reported sign and is often estimated rather than accurately counted by nurses (Flenady et al. 2016). A study of 211 post-operative patients found that over 15% of RR measurements were undocumented (McGain,2008). Inaccurate recording or assuming that a patient's RR is within normal range is another issue. Semler et al (2013) found a significant difference between actual RR and the one that was recorded; out of 368 recordings, nearly 72% showed 18 or 20 breaths/min despite the fact that only 13% were actually within that range. From the evidence it appears that staff do not accurately measure and record patients' RRs leading to RR being referred to as 'the neglected vital sign' (Elliott 2016).

The normal accepted RR range for an adult is 12-20 breaths/min (DoH 2020) however this can vary according to a patient's age and medical condition. Changes in RR can be seen on activity, due to emotion, illness and in the older person population. Higher RRs can be due to age-related changes that affect the body's ability to meet physiological demands and is why RR must be part of a holistic patient assessment

A respiration rate of >25 breath/min (bpm) or an increasing RR (even by as little as 3 to 5 bpm) can be an indication that a patient may be clinically deteriorating (DoH 2020). Likewise, a RR of < 12 breaths/min, or a decreasing RR can also signify deterioration.

Respiratory rate monitoring should always be part of a holistic patient assessment. Patients whose RR is outside of the normal range should be closely monitored using the frequency of monitoring guidance in the INEWS V2 Escalation and Response Protocol. Respiratory rate should be interpreted alongside other aspects of respiration (for example the depth, pattern and effort of breathing), other physiological observations and presenting clinical features.

KEY CLINICAL POINT

Measurement of physiological observations over time (as opposed to a once off measurement) is a much more useful indicator of decline or recovery

KEY CLINICAL POINT

Pulse oximetry is often incorrectly used as a replacement for RR measurement to evaluate respiratory dysfunction

Pulse oximetry is often incorrectly used as a replacement for RR measurement to evaluate respiratory dysfunction. However, pulse oximetry measures oxygen saturation while RR measures ventilation. During the early stages of clinical deterioration a patient's SpO2 may appear to be in the normal range, but the RR will increase in response to inadequate tissue oxygenation (Mok et al. 2015).

Physiological Oxygen Delivery

Oxygen is essential for the adequate production of adenosine triphosphate (ATP) by cell mitochondria (Figure 12). Adenosine triphosphate (ATP) is required as a source of energy for all intracellular functions. ATP is formed in the mitochondria via phosphorylation. A phosphate is added to adenosine diphosphate (ADP) via a high-energy bond, thus forming ATP. This stores energy on a temporary basis. When energy is needed by the cell, ATP is dephosphoryladed to ADP, releasing the energy from the bond (Figure 13).

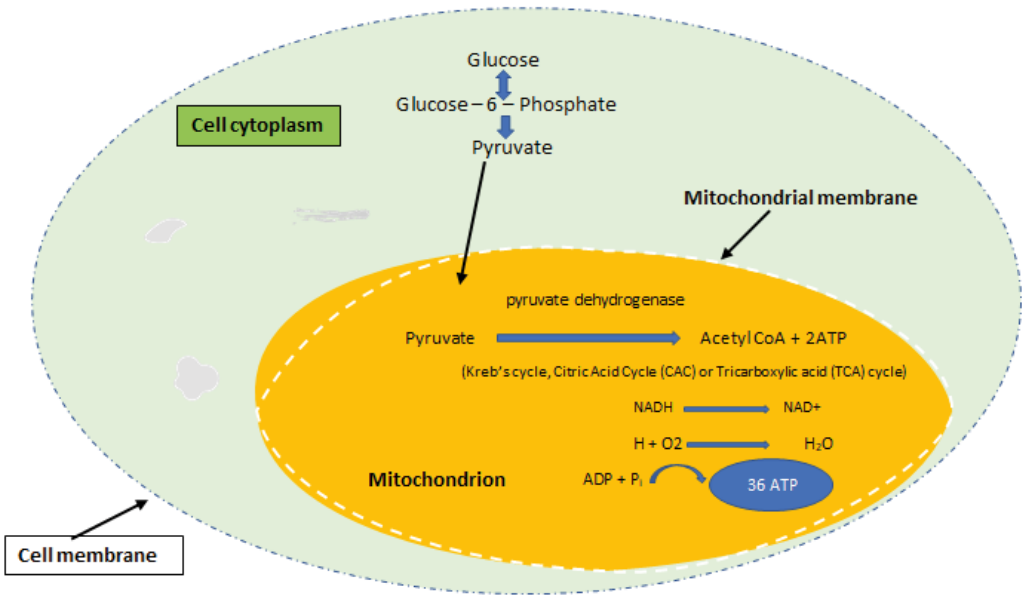


Figure 12 - Aerobic Metabolism i.e. metabolism in the presence of oxygen

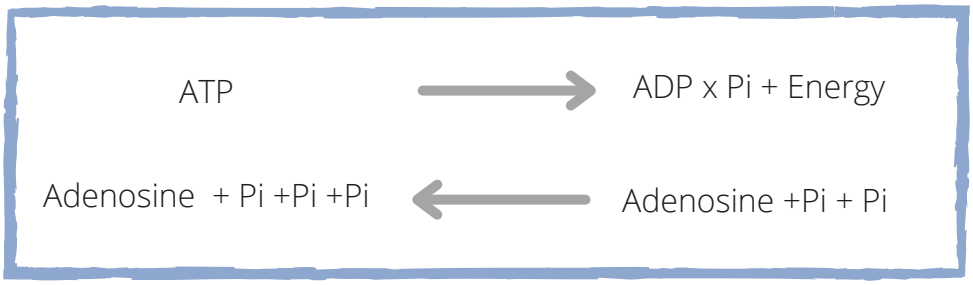


Figure 13 - Energy Release

If there is inadequate oxygen supply, ATP production falls, and cellular function is then depressed (Figure 14), through lack of energy. Therefore, reduced levels of oxygen supply at cellular level may cause organ failure, which at best leads to the unplanned admission of a patient to ICU and at worst a patient's death.

Therefore, if oxygen delivery is maintained, this may reduce the incidence of unplanned ICU admissions and unexpected deaths.

Hypoxia refers to insufficient *supply* of oxygen to the tissues of the body or part of the body.

Hypoxaemia refers to an abnormally low *concentration* of oxygen in the blood

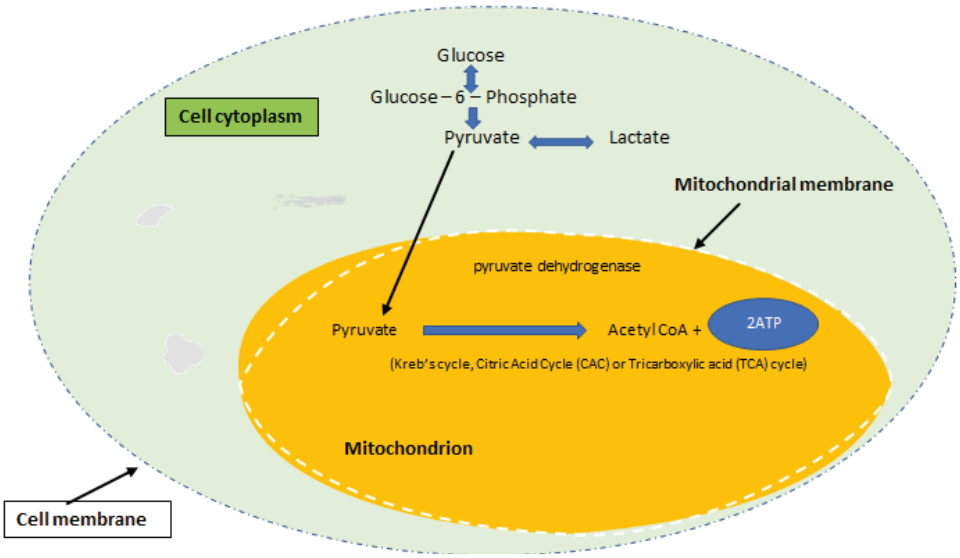


Figure 14 - Anaerobic Metabolism i.e. metabolism in absence of oxygen

Oxygen supply to the cells is described in Figure 15 in relation to airway, breathing and circulation.

Oxygen Delivery = Cardiac Output x Arterial Oxygen content

Thus oxygen delivery requires:

Arterial oxygen content:

- haemoglobin concentration (Hb)
- haemoglobin oxygen saturation (SaO_2)
- partial pressure of oxygen (PaO_2)

Cardiac output (see section on "CIRCULATION")

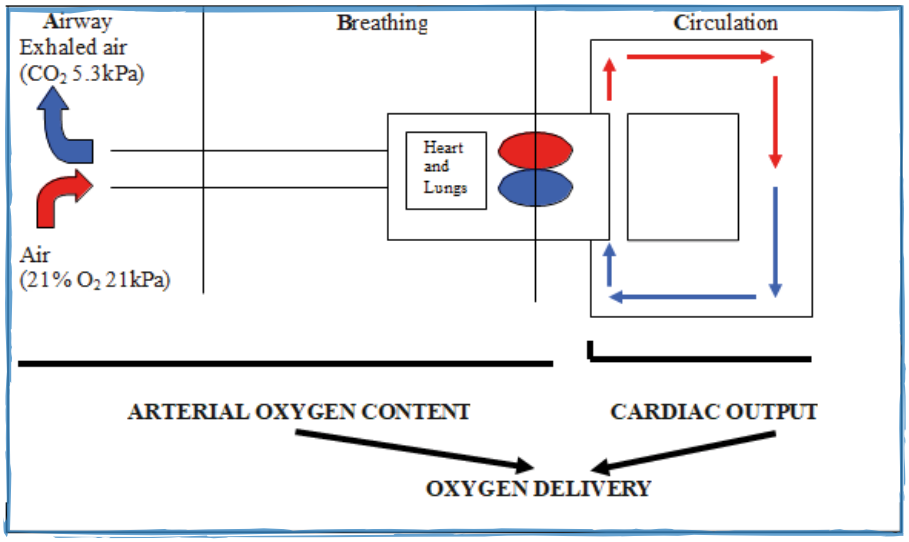


Figure 15 - ABC and the Oxygen Delivery

Oxygen Delivery Systems for Supplementary Oxygen

Oxygen delivery systems are classified into fixed and variable performance devices. They are able to deliver a wide range of oxygen concentrations.

Fixed Performance Devices

Provide gas flow that is sufficient for all the patient's minute ventilation requirements. In these devices, the inspired oxygen concentration is determined by the oxygen flow rate and attached diluter (see Table 3) e.g. the Venturi mask (Figure 16).

Figure 16 - Venturi Mask

In patients at risk of hypercapnia from a too high inspired oxygen, a Venturi system is more accurate in delivering the oxygen concentration desired.

Table 3: Relationship between Inspired Oxygen & Oxygen Flow Rate with Venturi Mask

Diluter Colour	Diluter Setting (Inspired oxygen)	Suggested Oxygen Flow Rate (Litres/min)
Blue	24%	2 l/min
White	28%	4 l/min
Yellow	35%	8 l/min
Red	40%	8 l/min
Green	60%	15 l/min

Variable performance devices

These do not provide all the gas required for minute ventilation, they entrain a proportion of air in addition to the oxygen supplied.

The inspired oxygen concentration will depend on:

- Oxygen flow rate
- The patient's ventilatory pattern (if the patient has a faster or deeper respiratory rate, more air will be entrained, reducing the inspired oxygen concentration).

These devices include nasal prongs, simple facemasks, partial re-breathing and non-rebreather masks.

Nasal prongs:

The dead space of the nasopharynx is used as a reservoir for oxygen, and when the patient inspires, entrained air mixes with reservoir air, effectively enriching the inspired gas. Oxygen flow rates of 2-4L/min (Figure 17).

High flow nasal prongs:

These use warm humidified oxygen at higher flow rates 4-8L/min (Figure 17)

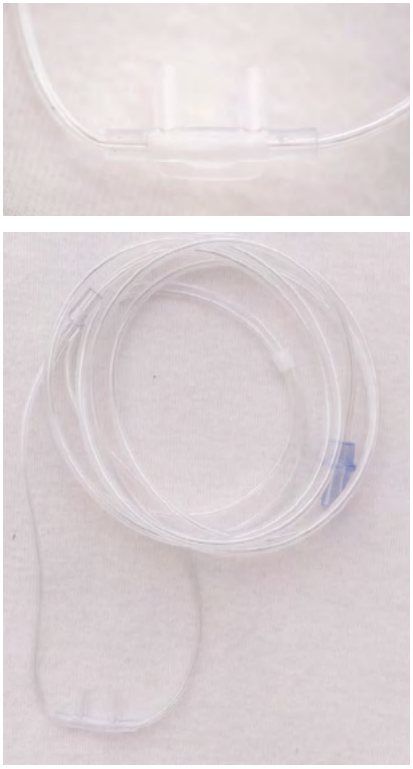


Figure 17 - Nasal Prongs

Oxygen facemask

Reservoir volume of oxygen is increased above that achieved by the nasopharynx , thus higher oxygen concentration can be achieved in inspired gas (max 50-60%) (Figure 18).

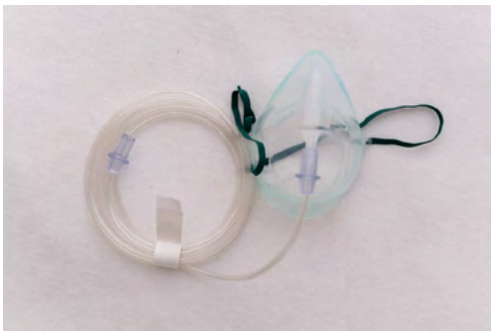


Figure 18 - Oxygen Face Mask

Non-re-breather mask

A simple face mask with the addition of a reservoir bag, with one or two-way valves over the exhalation ports which prevent exhaled gas entering the reservoir bag (permits inspired oxygen concentration up to 90%). Oxygen flow rate of 12-15L/min (Figure 19).

Figure 19 - Non re-breather Mask

All deteriorating patients should receive oxygen, before progressing to any further assessment. The aim is to deliver supplemental oxygen to achieve an SpO_2 of ≥ 96% in those patients not at risk for hypercapnoeic respiratory failure and the PaO_2 as close to 13kPa as possible, but at least 8kPa (SaO_2 90%) is essential. In most patients this can be achieved by sitting them upright and applying 12-15 litres/min of oxygen via a non-rebreather mask .

If the patient does not improve they will require review by an anaesthetist.

KEY CLINICAL POINT

Metabolic acidosis can increase the respiratory rate even though the arterial oxygen saturation may be normal

KEY CLINICAL POINT
In urgent situations, where oxygen is applied or the amount increased, a doctor must review the patient and prescribe accordingly

Monitoring and Titrating Oxygen Therapy

Oxygen therapy can be monitored clinically (patient's colour, respiratory rate, respiratory distress), or by measuring arterial oxygenation with pulse oximetry or arterial blood gas.
The advantage of measuring an arterial blood gas is that both oxygen and carbon dioxide are measured, as well as the metabolic status (including lactate).

If the carbon dioxide tension rises in someone with acute respiratory failure, it can be a sign that they are tiring and may require ventilatory support. If CO_2 begins to rise in a patient with COPD, it may be prudent to reduce the inspired oxygen concentration, however always remember that the arterial oxygen tension should not be allowed to fall below a PO_2 of 8 kPa.

Patients do not die from a raised CO_2 alone: they die from hypoxaemia.

Be aware that if a patient is maintaining a normal saturation, but their oxygen demands have increased (that is, they need more oxygen to maintain an normal SpO_2) then the patient is deteriorating.

In an acute setting, when taking an arterial blood gas sample, do not remove the oxygen. It is unnecessary to remove the oxygen and removing it may precipitate sudden deterioration. As long as the concentration of oxygen being delivered is recorded, the degree of hypoxaemia can be calculated. The blood gas machine can calculate this for you as long as the correct inspired oxygen concentration is recorded.

SaO_2 refers to the directly measured in vitro measurement of arterial oxygenation usually via ABG. SpO_2 refers to the indirect assessment using the oximeter.

Directly Measured Arterial Oxygenation

Arterial blood gases can be sampled and analysed in a standard fashion. These provide the gold standard method of measuring oxygenation. The ratio of oxygen carrying haemoglobin compared to the total amount of haemoglobin can be accurately measured. This value can be presented as a percentage which is termed SaO_2. Arterial blood gas measurement is invasive.

Indirectly Measured Oxyhaemoglobin

Pulse oximetry measures the presence of oxyhaemoglobin indirectly by measuring the absorption of light at certain frequencies. The absorption of light can be related to the presence of oxyhaemoglobin. This is usually done with a finger probe. It is possible to provide an estimate of indirectly measured oxyhaemoglobin concentration as a percentage of total haemoglobin. This is defined as the SpO_2. Pulse oximetry is non-invasive but can be affected by variables such as movement, the presence of unusual haemoglobin varieties, including carboxyhaemoglobin, and nail varnishes.

Oximeters can be unreliable in certain circumstances , e.g. if peripheral circulation is poor, the environment is cold, cardiac arrhythmias, or if the patient is convulsing or shivering

Although pulse oximetry provides good monitoring of arterial oxygenation, it does not measure the adequacy of ventilation, as carbon dioxide levels are not measured nor does it determine the adequacy of oxygen delivery to the tissues.

Oxygen saturation may be “normal” but the PCO_2 may be high which reflects inadequate minute ventilation and hence respiratory failure. Arterial oxygen saturation being “normal” does not rule out acute respiratory failure.

KEY CLINICAL POINT

SaO_2 *directly* measures arterial oxygenation usually via ABG. SpO_2 *indirectly* measures arterial oxygenation using oximeter

Arterial blood gases remain the gold standard for assessing respiratory failure. It measures arterial oxygen, arterial saturation and arterial carbon dioxide. It also provides information on the metabolic system (i.e. bicarbonate concentration, base excess and lactate) an approximate haemoglobin, electrolytes and blood glucose.

ABGs should be measured in patients who:

- Are critically ill;
- Have deteriorating oxygen saturations or increasing respiratory rate;
- Require significantly increased supplemental oxygen to maintain oxygen saturation;
- Have risk factors for hypercapnoeic respiratory failure;
- Have poor peripheral circulation and therefore unreliable peripheral measurements of oxygen saturation.

When assessing a patient remember to incorporate a full set of INEWS observations, do not just look at one parameter in isolation. If you suspect failing oxygen delivery, consider where in the oxygen delivery chain the problem might be (Figure 20).

KEY CLINICAL POINT
Remember to incorporate all INEWS physiological observations in your assessment

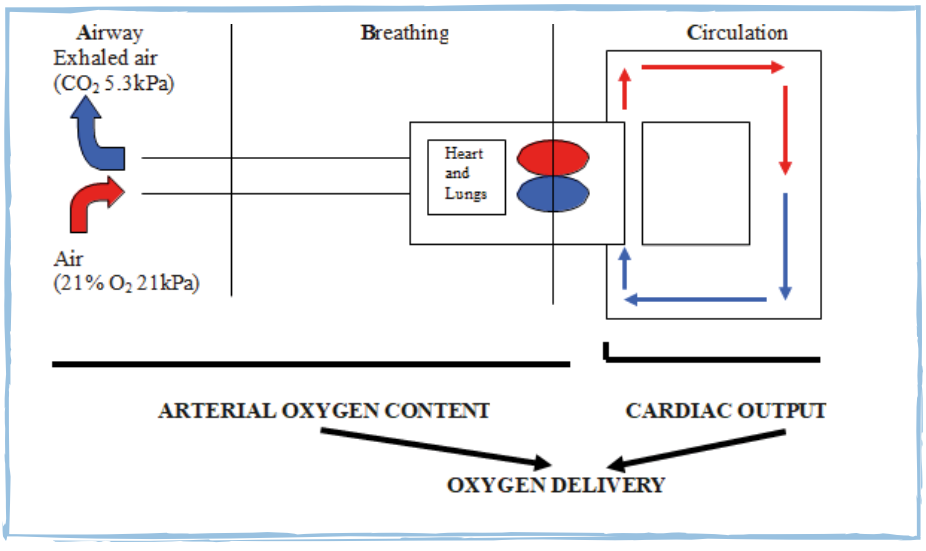


Figure 20 - ABC - Oxygen Delivery Chain

Escalation of Care & Clinical Judgement

Clinical judgement and knowledge of a patient's baseline RR (and other physiological observations) are key determinants used to guide monitoring frequency and escalation of care. If a clinician is concerned about a patient it is important to complete and document a full set of INEWS observations and consider these measurements in the context of the patient's observations trend. Measurement of physiological observations over time (as opposed to a once off measurement) is a much more useful indicator of decline or recovery.

Changes in mentation such as new confusion or delirium may be a sign of hypoxia and should be urgently investigated.

INEWS assists clinicians in standardising the approach to the recognition and response to clinical deterioration in Irish acute hospitals. However it is essential that both the patient's normal 'lived' baseline and the clinical judgement of the clinician (nurse, doctor, HSCP) is taken into account. INEWS V2 enables this individualisation of care through the use of the Modified Escalation and Response Protocol by a Consultant or Registrar (see INEWS Recommendations 16b and 17, page 42 INEWS NCG and pages below).

KEY CLINICAL POINT

Pulse oximetry is often incorrectly used as a replacement for RR measurement to evaluate respiratory dysfunction

Automated Respiratory Rate Monitoring

Continuous monitoring of vital signs has been recognised by clinicians as a way of identifying trends that might otherwise be missed by intermittent monitoring (i.e manual). A patient's condition can markedly change between routine sets of manual observations and these 'gaps' may cause signs of deterioration to be missed (Prgomet 2016).

During the COVID-19 pandemic an Automated Respiratory Rate Monitoring (ARRM) system was introduced into a significant number of acute hospitals in Ireland. When applied ARRM accurately measures RR. This accurate measurement of RR can enhance the predictive ability of INEWS. Consideration should be given to how ARRM can be used routinely in clinical practice i.e. on non COVID-19 patients.

Automated Respiratory Rate Monitoring (ARRM) enables the digital monitoring of a patient's respiratory rate – one of the seven INEWS parameters and the most neglected vital sign, usually estimated rather than counted (Flenady et al. 2016).

The introduction of ARRM will not alter the current standard of care for monitoring respiratory patients which includes conducting a full set of INEWS observations and calculating the INEWS score.

The monitoring and recording of RR is part of a full patient assessment. It should be interpreted in the context of other presenting clinical features, together with other aspects of respiration, for example the depth, pattern and effort of breathing (Kelly 2018). The purpose of ARRM is to accurately measure and record a patient's respiratory rate. Introduction of ARRM is a first, early step in digital monitoring of clinical deterioration. ARRM is an adjunct to INEWS which in turn is an adjunct to clinical judgement.

The surveillance and interpretation of data generated through continuous monitoring still requires expert nurses and clinicians to inform patient care.

Oxygen Delivery In Chronic Obstructive Pulmonary Disease (COPD)

In a small subgroup of patients who have Chronic Obstructive Pulmonary Disease (COPD) and are "CO_2 retainers" or their risk factors for hypercapnoeic respiratory failure are increased (e.g morbid obesity, chest wall deformities or neuromuscular disorders), high concentrations of oxygen can be disadvantageous by suppressing their hypoxic drive.

However, these patients will also suffer end-organ damage or cardiac arrest if their blood oxygen levels fall too low. The aim of care for these patients is to achieve PaO_2 of 8kPa or saturation of greater than 90% on pulse oximetry. Therefore in a patient with COPD who has a PCO_2 > 8kPa, but is also hypoxic, PO_2< 8kPa, do not turn the inhaled O_2 down, however, do not leave the patient unattended. If their PO_2 is >8kPa then you can turn the inhaled O_2 down to maintain SaO_2 > 90%

KEY CLINICAL POINT
In urgent situations, where oxygen is applied or the amount increased, a doctor must review the patient and prescribe accordingly

KEY CLINICAL POINT
Delivery of high concentrations of oxygen to achieve a PaO_2 within the normal range may suppress the respiratory drive in COPD patients causing the PCO_2 to rise

Q. Will a high concentration of inspired oxygen have an effect on the respiratory drive of my patient with Chronic Obstructive Pulmonary Disease?

A. This is a common concern. Many patients with COPD rely on a "hypoxic" respiratory drive. Their usual PaO_2 may be lower than the normal range e.g. 8kPa (normal 11-15kpa).

Delivery of high concentrations of oxygen to achieve a PaO_2 within the normal range may indeed suppress the respiratory drive in these patients causing the PCO_2 to rise.

However, when patients with underlying COPD deteriorate, e.g. due to pneumonia, high concentrations of inspired oxygen may be required to treat hypoxia, the aim being to return the PaO_2 to what is considered an acceptable level for this individual patient - usually 8kPa. Patient care and management includes:

- Prompt treatment of severe hypoxia is vital, failure to do so will result in cardiac arrest earlier than a raised PCO_2.
- Administer enough oxygen as prescribed to achieve an SpO_2 >90% and a PaO_2 of 8kPa. If the PaO_2 rises above 8kPa, you may turn down the inhaled oxygen as prescribed to achieve an SpO_2 of >90%.
- It is very important not to leave the patient unattended during this time and continuous monitoring and or transfer to a higher level of care may be required.

Q. Should I discontinue oxygen therapy temporarily while the deteriorating patient has a sample of arterial blood taken for Arterial Blood Gas (ABG) analysis?

A. No.

Q. What should I do if a COPD patient develops respiratory acidosis?

(pH ≤ 7.35 and respiration > 25 breaths per minute)

A. Consider institution of Early Non-invasive Ventilation

Patient Case Study

Mr. Murphy is 80 years old and presented to his GP with a five-day history of a productive cough, dyspnoea (shortness of breath), tachypnoea (rapid breathing) with some chest discomfort on deep breathing, and fever. He has a past medical history of hypertension treated with antihypertensives and is an ex-smoker. He lives with his wife in a bungalow and has had no recent travel abroad and has had a 'not detected' test result for COVID -19.

Mr Murphy's signs and symptoms suggested that he had community-acquired pneumonia (CAP). The very young and older people are more susceptible to CAP and the average length of stay in hospital is six days with mortality at 30 days being approximately 18% (Lim and Woodhead 2011).

Table 4 - Mr. Murphy's Physiological Observations

INEWS Physiological Observation	Normal Values	Baseline Assessment	Reassessment on Admission
Respiratory Rate	12 - 20	20	25
SpO_2	≥ 96%	94%	91%
FiO_2	Room Air	24% via Nasal Prongs	40% via Venturi Mask
Heart Rate	51 - 90	88	112
Blood Pressure	111 -249	140/70	130/80
ACVPU	Alert (A)	Alert (A)	Alert (A)
Temp	36.1 - 38.0	37.8	38.0
INEWS Score	0	4	11

Baseline vital signs

Mr Murphy was referred by the GP to the Acute Medical Assessment Unit (AMAU) to determine if admission to hospital was required.

In AMAU his baseline observations were recorded as part of a holistic assessment; his RR was at the upper end of normal – 20 breaths per minute (bpm) measured over a full 60 seconds, the depth of his breathing was shallow and the rhythm was regular. His oxygen saturation (SpO_2) was 94% (Table 4). He was sitting on a chair but leaning slightly forwards, with his hands on his knees and reported chest discomfort on the right side.

On further examination his chest movement appeared symmetrical and there was no paradoxical movement observed. A chest X-ray showed radiological shadowing at the lower section of his right lung. Mr. Murphy was commenced on 24% O_2 via nasal prongs. The decision to admit Mr. Murphy was made. Monitoring and observation continued. Mr Murphy's INEWS score was 4 on admission.

Reassess on admission

On admission Mr Murphy's INEWS physiological observations (Table 4) showed an increase in RR to 25bpm; although the rhythm was regular, the depth of chest movement remained shallow. The increase in RR and heart rate was largely due to metabolic demand for oxygen delivery and removal of lactic acid (a by-product of anaerobic cellular respiration). Anaerobic respiration occurs when there is an inadequate oxygen supply to the tissues.

Mr Murphy's SpO_2 had decreased to 91% on 24% O_2 via nasal prongs, indicating that insufficient oxygen was diffusing into the blood. Supplementary oxygen was, therefore, increased to 40% O_2 via venturi mask.
Mr Murphy's chest movement was asymmetrical, with reduced movement on the right side of the chest.

Mr Murphy is alert, however, responded with one-word answers, and was clearly fatigued. He was using accessory muscles to help in an attempt to increase inspiratory capacity.

These changes in physiological observations indicated that Mr Murphy had acutely deteriorated; his INEWS score was now 11 and required an immediate emergency review by Registrar/ Consultant / ANP, continuous monitoring and transfer to a higher level of care (DoH 2020).

As Mr. Murphy's INEWS score is > 5 on oxygen and infection is present, sepsis is suspected and the Sepsis Pathway is commenced (DoH 2014). The treatment interventions included antibiotics and oxygen to maintain SpO_2 within target range.

This case study illustrates how RR is an early sign of deterioration. It remains one of the physiological observations that is often poorly assessed and its importance not recognised.

KEY CLINICAL POINT

Abnormal respiratory rate is the single strongest predictor for a severe adverse event, yet it is the most neglected vital sign

Summary

- A change in RR is an important indicator of inadequate oxygen delivery to the tissues and therefore a marker of a deteriorating patient.
- RR should be counted for a full 60 seconds.
- A change in RR (even by as little as 3 to 5 bpm) can be an indication that a patient may be clinically deteriorating.
- Pulse oximetry measures oxygen saturation while RR measures ventilation.
- During the early stages of clinical deterioration a patient's SpO_2 may appear to be in the normal range, but the RR will increase in response to inadequate tissue oxygenation
- Be aware that if a patient is maintaining a normal saturation, but their oxygen demands have increased (that is, they need more oxygen to maintain an normal SpO_2) then the patient is deteriorating
- A change in one parameter only is highly unusual; assess all INEWS physiological observation and monitor trends when assessing a patient.
- Patients with a respiratory rate of 25 or more are at high risk of deterioration
- When the patient is acutely ill, it is unnecessary to remove the oxygen mask when taking an arterial blood gas sample, as it may precipitate sudden deterioration.
- Oxygen saturation may be "normal" but the PCO_2 may be high reflecting inadequate minute ventilation and respiratory failure.
- Oxygen is essential for the adequate production of adenosine triphosphate (ATP).
- If there is an inadequate oxygen supply, ATP production falls, and cellular function is then depressed.

This section covers:

- Importance of Oxygen: a recap
- Why pulse rate and blood pressure are “vital signs”, and the importance of measuring them
- The mechanisms which generate blood pressure
- Causes and consequences of, and compensation for hypotension
- What is meant by shock
- Management of hypotension in the deteriorating patient

The Importance of Oxygen: Recap

Oxygen reaching the cells and mitochondria is dependent upon adequate amounts of oxygen being delivered via the blood circulation. Without oxygen being delivered to the mitochondria, inadequate amounts of ATP are generated and cellular dysfunction occurs.

Oxygen delivery's key components are (Figure 21):

- Cardiac output = Stroke Volume (volume of blood pumped by each contraction of the ventricles) x Heart Rate (Heart beats per minute)
- Arterial oxygen content = Haemoglobin concentration x Arterial Oxygen Saturation

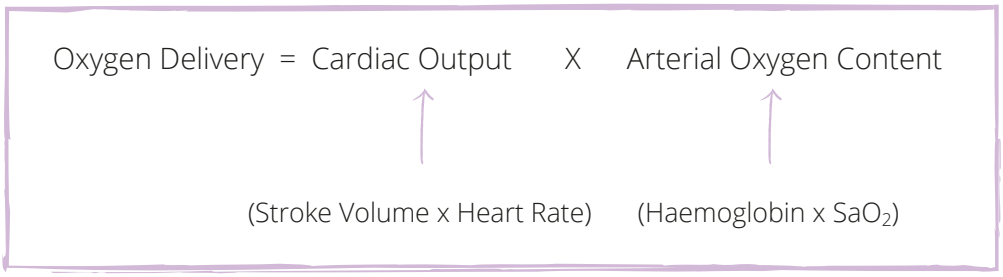


Figure 21 - Oxygen Delivery

Blood Pressure

The arterial blood pressure is the result of the discharge of blood from the left ventricle into the already full aorta.
When the left ventricle contracts and pushes blood into the aorta the pressure produced is known as the systolic blood pressure.
When complete cardiac diastole occurs and the heart is resting following the ejection of blood, the pressure within the arteries is termed the diastolic blood pressure.
Blood pressure is the product of cardiac output and total peripheral resistance, also known as systemic vascular resistance.

Blood Pressure = Cardiac Output X Total Peripheral Resistance

- The measurement of pulse and blood pressure are important surrogate markers of whether there is adequate cardiac output and hence oxygen delivery to the tissues.
- A decrease in blood pressure can reflect a decrease in cardiac output which can lead to a reduction in the amount of oxygen getting to the tissues.
- An increase in heart rate may reflect a decrease in stroke volume, which may reflect a decrease in cardiac output which may lead to inadequate amounts of oxygen getting to the tissues.

KEY CLINICAL POINT
A 20% drop in SBP for normally hypertensive patients requires a medical review

Blood Pressure and Maintenance of Organ Function

There are some organs that require an adequate blood pressure for their optimal function as well as adequate oxygen delivery. The brain and kidney are two examples of these organs. Inadequate blood pressure and oxygen delivery may present as new confusion / delirium or acute kidney injury (AKI).

The body's organs adapt over time to a person's "normal" blood pressure. If blood pressure is always elevated, e.g. chronic hypertension, the brain and the kidney adapt and will require a greater blood pressure in order to function normally. Therefore, it is important to know what your patient's "normal" or "lived baseline" blood pressure was prior to their current illness.

Definition of Hypotension

The generally accepted definition of hypotension in adults is:

- A drop of more than 20% from 'usual' blood pressure (e.g. a drop from 160 / 80 to 128/80 i.e 20% drop) or
- Systolic blood pressure (SBP) of less than 100mmHg

It is important to remember that someone who is normally hypertensive may be relatively hypotensive even when their systolic blood pressure is above 100mmHg (*see example above).* Do not always use 100mmHg as your CRITICAL Systolic Blood Pressure cut off!

Possible Causes of Hypotension

If blood pressure is the product of cardiac output and total peripheral vascular resistance, blood pressure can fall because of either:

a) A fall in cardiac output;
b) A fall in peripheral vascular resistance.

It is important to understand how cardiac output and total peripheral resistance are determined and what can affect them. Having understood these principles, it is then easier to know what interventions to initiate.

Cardiac Output

Cardiac output is the product of stroke volume (volume of blood pumped by each contraction of the ventricles) and heart rate (heart beats per minute).
Factors affecting stroke volume:

- Cardiac contractility
- Pre Load
- After Load
- Heart Rate

Cardiac contractility:

The ability of the heart to contract in the absence of any changes in preload or after load reflects cardiac muscle strength.

KEY CLINICAL POINT
Decreases in intravascular blood volume can decrease cardiac output and therefore decrease blood pressure

Major **positive** influences (inotropy) include:

- Sympathetic nervous system;
- Sympathomimetics (noradrenaline, adrenaline);
- Calcium;
- Digoxin.

Major **negative** influences (negative inotropy) include:

- Myocardial ischaemia;
- Acidosis;
- Drugs (e.g. beta-blockers, anti-dysrhythmic).

Pre-load

Pre - load is the volume of blood in the ventricles before they contract (end diastolic volume). Diastole refers to the ventricles at rest.
So, how well filled is the heart at the end of diastole? i.e. the end diastolic volume. Increases in end diastolic volume will result in an increase in stroke volume although if the end diastolic volume overstretches the heart muscle, the stroke volume can start to decrease.

The major effect of pre-load is venous return to the heart, which is influenced by both intravascular blood volume and intrathoracic pressure.

Intravascular blood volume can be *absolute* or *relative*.

Absolute intravascular blood volume: A decrease in intravascular blood volume (bleeding, electrolyte, water loss, diarrhoea, vomiting, diabetes insipidus) will cause a decrease in venous return and hence a decrease in stroke volume.

Relative intravascular blood volume: There is no actual loss of intravascular blood volume but with vasodilatation and pooling of blood (vasodilators, epidurals, sepsis) a decrease in venous return to the heart occurs and hence a decrease in stroke volume.

Intrathoracic pressure: An increase in intrathoracic pressure (e.g. asthmatic attack, positive pressure ventilation) will restrict the amount of blood returning to the heart, decreasing venous return and therefore reduce stroke volume.

KEY CLINICAL POINT
Increase in intrathoracic pressure can decrease cardiac output and therefore decrease blood pressure

Afterload

Afterload is the pressure the myocardial muscle must overcome to eject blood out of the ventricles during systole (i.e contraction).
This resistance can either be caused by an outflow resistance from the heart (aortic stenosis) or resistance to flow in the systemic circulation. This resistance is determined by the diameter of the arterioles and precapillary sphincters. As resistance rises, stroke volume is reduced.

KEY CLINICAL POINT
An increase in peripheral vascular resistance can decrease cardiac output and hence oxygen delivery

Heart Rate

The sinoatrial (SA) node is the inherent pacemaker situated in the upper right quadrant of the right atrium. Cardiac conduction begins in the SA node and from there the electrical impulse travels to the ventricles via the atrioventricular (AV) junction, bundle of His, the left & right bundle branches and Purkinje fibres. This spread of the electrical impulse through the cardiac conduction system is known as depolarisation. The depolarisation wave causes the atria and subsequently the ventricles to contract.

Heart rate is determined by the rate of spontaneous depolarisation at the sinoatrial node. The rate can be modified by the autonomic nervous system:

- Parasympathetic stimulation: **SLOWS** the heart rate via the vagus nerve e.g. vasovagal response, parasympathomimetics e.g. Anticholinesterases (Neostigmine);

- Sympathetic stimulation: **QUICKENS** the heart rate via the sympathetic cardiac fibres e.g. stress response, temperature, sympathomemetics (adrenalin, noradrenalin, isoprenaline).

In the absence of conduction through the atrioventricular node (Complete Heart Block), the ventricle will only contract at its intrinsic rate of 30-40 beats per minute.

Any change in heart rate can affect cardiac output. A faster heart rate can increase the cardiac output and this often occurs when the stroke volume is falling, while any reduction in heart rate can cause a decrease in cardiac output.

Does a fast heart rate always increase cardiac output and blood pressure?

No. There are situations when an increase in heart rate may reduce the cardiac output. If the ventricle does not have adequate time to fill with blood, this reduces the end diastolic volume and therefore stroke volume. Cardiac output reduces as a result and may cause a drop in blood pressure. A good example is atrial fibrillation with a rapid ventricular response.

Does a slow heart rate always decrease cardiac output and blood pressure?

No. Sometimes when the heart rate slows there may be no reduction in cardiac output. As the ventricle has a longer time to fill, the end diastolic volume is increased with each beat, it stretches the myocardial fibres and increases the stroke volume per beat. This mechanism may then compensate for the reduction in heart rate. Therefore, there may be no change or even an increase in cardiac output and blood pressure. A good example of this phenomenon is a very healthy athlete.

Peripheral (or Systemic) Vascular Resistance

Changes in peripheral vascular resistance (the cumulative resistance of the thousands of arterioles in the body) can increase or decrease blood pressure. Peripheral vascular resistance is a function of the Autonomic Nervous System. Autonomic Nervous system control of the peripheral vascular system occurs through either an increase or decrease in peripheral resistance.

An increase in peripheral vascular resistance involves:

- Stimulation of Sympathetic Receptors:

Sympathetic stimulation (alpha 1 receptor) of the arterioles can cause vasoconstriction and a subsequent increase in blood pressure. This often occurs in response to a fall in blood pressure (perhaps as a result of falling cardiac output), which is detected by baroreceptors situated in the carotid sinus and aortic arch, reducing the stimulus discharged from them to the vasomotor centre with a resultant increase in sympathetic discharge e.g. Sympathomimetics that stimulate the alpha 1 receptor will cause vasoconstriction of the arterioles, examples include noradrenaline, adrenaline.

- Direct action on arteriole smooth muscle:

Examples include vasopressin, angiotensin, methylene blue (a vasoconstrictor by inhibiting nitric oxide action on the vasculature).

A decrease in peripheral vascular resistance involves:

- Blockade of the Autonomic Sympathetic Nervous System

Anything that causes a reduction in the sympathetic stimulation of the arterioles will result in vasodilatation, reducing vascular resistance and blood pressure.

Influences include:

- Increasing the stimulation of the baroreceptors from a rise in blood pressure, which causes a reduction in the sympathetic outflow causing vasodilatation;
- Any drug that blocks the sympathetic nervous system can cause vasodilatation and a fall in blood pressure, e.g. 2 agonists (clonidine, epidurals)

Compensatory Mechanisms for Hypotension

An adequate blood pressure is important for the function of vital organs including the brain, heart and kidneys. Any reduction in blood pressure will trigger the body to respond in order to maintain homeostatis.

The body's compensatory response to a fall in blood pressure is dependent on the cause of the drop in blood pressure

If the drop in BP is due to a reduction in Cardiac Output i.e. through a reduction in either stroke volume or heart rate the body's compensatory response will be as follows:

- A reduction in stroke volume (SV) will result in a compensatory increase in the heart rate (tachycardia) and a compensatory increase in peripheral vascular resistance (cool, blue peripheries).

- A reduction in heart rate (HR) will result in a compensatory increase in total peripheral vascular resistance in an attempt to maintain a near-normal blood pressure.

If the drop in BP is due to a reduction in Peripheral Vascular Resistance the body's compensatory response will be as follows:

- There will be a compensatory increase in cardiac output. Cardiac output will be increased by an increase in the heart rate (tachycardia) and by an increase in the contractility of the heart which results in an increase in the stroke volume. Cardiac output is further increased following treatment with intravenous fluids to improve venous return.

Table 5: Clinical Features for Stroke Volume/Heart Rate /Peripheral Vascular Resistance

Stroke Volume	Reduction in Pre-Load (hypovolaemia):	○ Hypotension with a postural drop, ○ Tachycardia ○ Cool, mottled peripheries
	Reduction in contractility (cardiac failure):	○ Hypotension, ○ Tachycardia ○ Cool, mottled peripheries with signs of heart failure
HR	Reduction in Heart Rate (HR):	○ Hypotension, ○ Bradycardia ○ Cool mottled peripheries
PVR	Fall in Peripheral Vascular Resistance (PVR):	○ Hypotension ○ Tachycardia ○ Warm peripheries

KEY CLINICAL POINT
Inadequate oxygen delivery leading to lactate formation results in an increase in respiratory rate (RR) which is an earlier indicator of clinical deterioration than changes in either HR or BP

Consequences of Hypotension

The greatest concern is that hypotension may suggest that there is an inadequate amount of oxygen getting to the tissues because of a falling cardiac output, which is described as SHOCK.

Relationship between blood pressure (BP) and oxygen delivery (DO2):
BP = Cardiac Output x Peripheral Vascular Resistance
DO2= Cardiac Output x Arterial Oxygen Content

Inadequate Cardiac Output

- Cardiac output is integral to the amount of oxygen being delivered to the tissues. If the cardiac output falls, it is likely that oxygen delivery will fall.

- If there is inadequate oxygen delivery to the tissues, there is a reduction in the amount of ATP generated which is vital for cellular function.

- Inadequate oxygen delivery to the tissues in turn leads to organ failure, lactate formation and shock.

- Inadequate oxygen delivery leading to lactate formation results in an increase in respiratory rate (RR) which is an earlier indicator of clinical deterioration than changes in either HR or BP. Thus, it is important to monitor RR accurately.

Inadequate Pressure Gradient

- Clearly without a pressure gradient across the vasculature (from high pressure to low pressure) there can be no flow of blood and its constituents including oxygen.
- Some organs are able to maintain blood flow, despite changes in blood pressure (autoregulation) e.g. brain and kidney. However, the body reaches a point when this compensation mechanism can no longer occur, that is, if the blood pressure remains too low. Once this point is reached there is reduced blood flow to the organs resulting in a reduced amount of oxygen being delivered to the tissues and organs.
- Inadequate blood flow to the organs results in inadequate oxygen delivery to the organs resulting in reduced generation of ATP. This process will lead to organ failure (oliguria and altered mentation), lactate formation and shock.

When is hypotension not shock?

In order to demonstrate that there is shock there needs to be evidence that organs are failing and/or that there is evidence of anaerobic respiration by the presence of lactate.

For example: If patient is hypotensive post anaesthetic and has warm hands (suggesting good blood flow to the hands i.e. good cardiac output), is not confused, has a good urine output with no signs of heart or respiratory failure and has a normal lactate level (≤ 2 mmol/L) , then the patient is currently not shocked. However, it is important to continue regular monitoring of the patient's vital signs and to continually monitor for evidence of organ failure.

KEY CLINICAL POINT
A systolic blood pressure of less than 90 mmHg in an adult requires immediate review by doctor

Can a patient with a normal or high blood pressure have shock?

The key components of adequate oxygen reaching the tissues are cardiac output and arterial oxygen content. If either of these two elements are reduced there is a fall in oxygen transport to the tissues and this results in shock. Sometimes, the compensatory mechanisms for a fall in cardiac output, such as an increase in total peripheral resistance, can result in there being a normal or even high blood pressure reading for a short period of time.

Therefore, despite there being a "normal" blood pressure reading, there are other clinical signs of organ failure and anaerobic respiration i.e. the patient is shocked with a seemingly normal blood pressure.

For example: An elderly lady presents with an inferior myocardial infarction and complete heart block. On examination she has dark blue fingers, a heart rate of 40 beats per minute, her blood pressure is 210/100mmHg and she has evidence of pulmonary oedema and oliguria. Her lactate measurement is 10mmol/l (normal < 2mmol/l).

Despite a high blood pressure reading due to the increase in vascular tone to try and compensate for the fall in cardiac output, there is evidence of organ failure and anaerobic respiration. This patient IS shocked despite the high blood pressure.

Management of acute Hypotension

It is important to remember what generates a blood pressure i.e. Cardiac Output (stroke volume x heart rate) and Peripheral Vascular Resistance.

It is essential to determine from the patient's medical history and clinical examination, which of these two factors have decreased leading to a fall in blood pressure.

Hypotension due to a fall in Cardiac Output

There are two predominant causes of a fall in cardiac output - a fall in preload and a fall in cardiac contractility . Both have very different presentations and are described below.

Fall in cardiac output due to a fall in Pre-Load

Common causes include bleeding, loss of fluids and electrolytes.

History:

- A history relevant to bleeding, loss of fluid and electrolytes (diarrhoea, vomiting and polyuria from hyperglycaemia), loss of water (diabetes insipidus);
- Look at fluid balance chart and determine recent fluid balance;
- Can also describe symptoms of postural hypotension (feels faint when standing up, has actually "fainted").

Examination:

- Signs that are relevant to the fluid loss (bleeding into drains, melaena, nasogastric losses);
- Cool, mottled hands, tachycardia, hypotension with a postural drop (a drop more than 10mmHg in Systolic BP from lying to sitting).

Laboratory Investigations:

- Evidence of bleeding (fall in haemoglobin);
- Evidence of renal dysfunction (rising creatinine)
- Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling)

Management:

- Correct the cause of loss of fluid (call for urgent review of ongoing bleeding, may need to correct coagulopathy);
- Replace whatever fluid has been lost (blood if bleeding, saline if gastrointestinal losses, 5% Glucose if diabetes insipidus);
- Estimate how much has been lost by looking at the fluid balance chart, how much is in the drains, how far the haemoglobin has fallen;
- In the first instance in adults rapidly administer 500-1000mls of normal saline via a blood pump set through a large bore intravenous cannula;
- Observe response (tachycardia should be reduced and blood pressure increased);
- Continue to administer fluid rapidly until there is the desired response:
    - Blood pressure return to normal;
    - Heart rate returning to normal;
    - Improvement in organ function, particularly urine output.
- Intensive care should be alerted especially if there are no signs of improvement despite administering 3L of fluid;
- Continue to monitor observations using the INEWS Escalation & Response Protocol as a guide.

Fall in cardiac output due a fall in cardiac contractility

Common causes include myocardial ischaemia or infarction.

H*istory:*

- May describe history of chest pain suggesting ischaemia;
- May describe previous symptoms of heart failure i.e. orthopnoea (breathlessness in the recumbent position), swollen ankles, breathlessness
- Describe palpitations (suggesting a tachycardia- atrial fibrillation, ventricular tachycardia) or symptoms related to cardiomyopathy;

Examination:

- Cool, blue hands, tachycardia and hypotension;
- Signs of right heart failure (swollen ankles, raised jugular venous pressure);
- Signs of left heart failure (tachypnoea, fine inspiratory crackles that do not clear on coughing, third heart sound, low arterial oxygen saturation).

Clinical Investigations:

- Evidence of renal dysfunction (rising creatinine);
- Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling (base deficit, rising lactate);
- ECG – signs of ischaemia, infarction, dysrhythmia.

Management:

- If the patient is hypotensive and has signs of organ failure including heart failure (cardiogenic shock), the patient will require inotropic support and referral to either the coronary care unit or intensive care unit;
- Stop all intravenous fluids as the patient is by definition fluid overloaded;
- Continue strict monitoring of fluid balance;
- Myocardial infarction protocol if MI confirmed.

KEY CLINICAL POINT
When assessing a patient remember to incorporate all the of the INEWS physiological observations and not just an individual parameter

Hypotension due to a fall in Peripheral Vascular Resistance

Common causes include infection / sepsis, and vasodilating drugs.

History:

- Chills, fever, symptoms of infection,
- ingestion/inhalation of vasodilators.

Examination:

- usually accompanied by warm hands (a vasodilated vasculature) and tachycardia. There may be signs of organ failure (confusion, oliguria, tachycardia).

Laboratory investigations:

- Evidence of infection (rise or significant fall in white cell count)
- Evidence of renal dysfunction (rising creatinine)
- Evidence of lactate formation (metabolic acidosis on arterial blood gas sampling, a base deficit, a lactate > 2mmol/l)

Management of hypotension :

In the absence of tachycardia, organ failure and /or lactate formation i.e. no clinical evidence of:

- organ failure (not oliguric, not confused),
- anaerobic metabolism (lactate formation) and /or
- no associated tachycardia

There may be no need to do anything medically other than close monitoring of the patient's physiological observations as per INEWS escalation and response protocol to ensure that there is no downward trend in blood pressure.

In the presence of tachycardia, but absence of organ failure and lactate formation, the tachycardia could be in response to a fall in venous return (due to pooling in the vasculature) and fall in stroke volume that has not yet affected the amount of oxygen going to the tissues. It is important to improve venous return and stroke volume to maintain adequate cardiac output and oxygen delivery to the tissues. Therefore:

- Administer an intravenous fluid bolus (500-1000 mls of normal saline (0.9% NaCl) for adults) *(consider co-morbidities where use of smaller boluses may be indicated)*
- Continue to monitor observations as per INEWS escalation and response protocol, document any trends and escalate any concerns;
- If there is an improvement in the tachycardia and blood pressure then the fluid bolus has been adequate to restore venous return at this time. (NOTE – this may only be a compensatory response and therefore temporary, for that reason continue to closely monitor the patient's observations);
- If the tachycardia is not resolved repeat the fluid challenge;
- Continue to observe response – monitor and record observations;
- If the patient continues to have hypotension, tachycardia and warm hands, further fluid can be administered (check amount with doctor as this will be the third fluid challenge) particularly if there are no signs of heart failure;
- An intensive care review should be requested if three litres of fluid have been administered and the tachycardia and hypotension have not been resolved.
- Think Sepsis where infection is suspected (Inpatient Adult Sepsis Algorithm). Refer to the "Fluid Resuscitation Algorithm for Adults with Sepsis" for fluid resuscitation in the adult patient with sepsis.

KEY CLINICAL POINT

An intensive care review should be requested if three litres of fluid have been administered and the tachycardia and hypotension have not been resolved

Hypotension and evidence of organ failure:

- Administer intravenous fluid bolus (500-1000 mls of Normal Saline (0.9% NaCl) for adults)
- Continue to perform frequent observations monitoring, document any trends in the patient's condition (as per INEWS escalation and response protocol)
- If there is an improvement in the patient's tachycardia and blood pressure, then the fluid bolus has been adequate to restore venous return
- If the tachycardia, hypotension and organ failure remain, repeat the fluid challenge
- Call for an intensive care review particularly if the patient has received three litres of fluid or signs of organ failure persist
- Continue to monitor observations to ensure that the trend of blood pressure, pulse and level of consciousness in response to interventions are improving

Summary of the management of hypotension in adults:

- Hypotension and warm hands:
    - administer fluids;
- Hypotension, cool hands, no signs of heart failure:
    - administer fluids;
- Hypotension, cool hands, signs of heart failure:
    - Cease fluids.
    - Refer to CCU/ICU for inotropic support.

Patient assessment following the acute stage of spinal cord injury ans.

Neurological conditions such as Spinal Cord Injury or conditions affecting the brain such as Traumatic Brain Injury may disrupt the integrity of the body's autonomic nervous system such that a patient's baseline parameters may be different and their ability to respond to acute illness may be severely altered.

Following the acute stage of spinal cord injury (tetraplegic and high paraplegic patient) there are a group of patients that continue to suffer from cardiovascular dysfunction. (Ahmed et al 2019, Asafu-Adjaye et al 2015). It is important to establish in these patients individual baseline physiological values.

Autonomic dysreflexia can arise in spinal cord injury and result in rapid rises in blood pressure to levels which may be fatal In a patient with a spinal cord injury a rise in systolic blood pressure of ≥ 20 mmHg above their know baseline systolic blood pressure is a medical emergency and potentially fatal condition if not treated early (Asafu-Adjaye et al 2015).

Refer to local hospital policy for the appropriate escalation and response protocol.

KEY CLINICAL POINT
A 20% drop in Systolic Blood Pressure for normally hypertensive patient requires a medical review

KEY CLINICAL POINT
A Systolic Blood Pressure ≤ 90mmHg in adults requires immediate review by a doctor

Summary

- Blood pressure = Cardiac Output x Peripheral Vascular Resistance
- Hypotension:
  - A combination of tachycardia and and hypotension may be reflective of low oxygen delivery to the tissues.
  - Hypotension can be a marker of a deteriorating patient with an increased risk of death;
  - Hypotension is often a later sign of clinical deterioration (changes in respiratory rate and new confusion / delirium are earlier signs)
  - It is important to remember that a patient who is normally hypertensive may be relatively hypotensive even when their systolic blood pressure is above 100mmHg;
  - In adults do not always use 100mmHg as your CRITICAL Systolic Blood Pressure cut off;
  - A "shocked" patient has signs of organ failure which may or may not accompany hypotension.
- Decrease in cardiac output can be caused by:
  - Decreases in intravascular blood volume
  - Increases in intrathoracic pressure
  - Increase in peripheral vascular resistance
- Any decrease in cardiac output can cause a decrease in oxygen delivery.
- A SYSTOLIC BLOOD PRESSURE ≤ 90mmHg in adults requires immediate review by a doctor, consider activation of the Emergency Response System (ERS) as appropriate to hospital model.
- Remember to incorporate all observations in your assessment!

This section covers:

- Causes of decreased urinary output;
- When to be concerned about low urinary output;
- Acute Kidney Injury (AKI)
- Management of Pre and Post - Renal Oliguria

Section Five
Urinary Output

Introduction

A reduced urinary output (oliguria) is one of the most common triggers for a patient review. The kidney is an “end-organ”; thus poor urinary output can be an indicator of patient deterioration due to many different causes, and is a sign of overall decline. It is important that the cause of a reduced urinary output is correctly diagnosed.

Pathophysiology

Normal urine flow requires:

- Adequate oxygenation of the kidneys;
- Adequate kidney perfusion pressure;
- Normal function of the kidneys;
- No obstruction to urine flow e.g. prostatomegaly, renal calculus, blocked catheter, urethral valve disorders, ureterocele.

Oxygen Delivery

In order to function, renal cells require adequate oxygen delivery, just as all the other cells in the body do. Oxygen delivery depends on cardiac output and arterial oxygen content.

If oxygen delivery to the kidney is decreased, urinary output will fall. If oxygen delivery is insufficient for renal function, it probably reflects inadequate oxygen delivery to other tissues as well. Therefore, urinary output can be a sign of the adequacy of whole-body oxygen delivery.

Perfusion Pressure

Renal blood flow is auto-regulated (i.e. kept constant) throughout a wide range of Mean Arterial Pressures (MAP) (70-170mmHg). The MAP is the perfusion pressure experienced by the organs. This range is increased in chronically hypertensive patients, who then require a higher blood pressure to maintain normal kidney function.

$$MAP = \frac{(2X\ Diastolic\ BP) + Systolic\ BP}{3}$$

Figure 22 - Perfusion Pressure Experienced by Organs

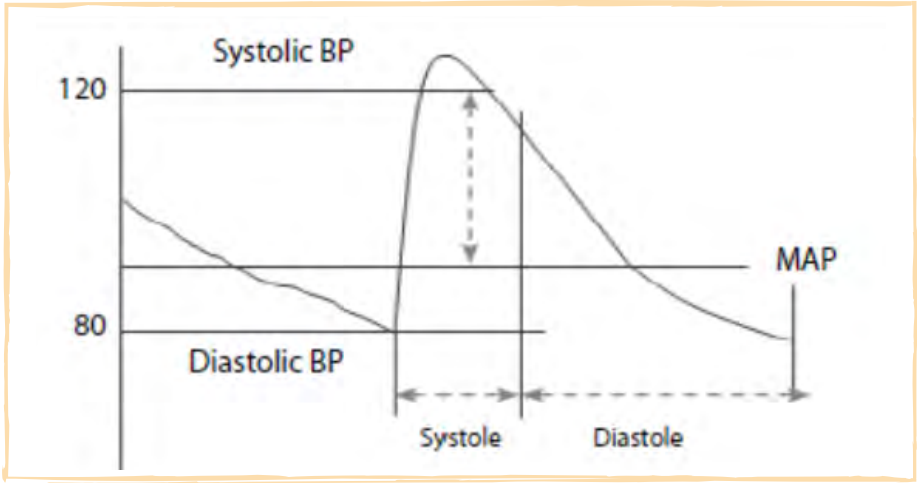


Figure 23 - Mean Arterial Pressure Diagram

If the mean arterial blood pressure falls below the lower limit for auto-regulation, then the renal perfusion pressure will decrease and thus urinary output will fall.

KEY CLINICAL POINT
Remember to always incorporate all the INEWS physiological observations in your assessment

Management of Low Urinary Output

The cause of the decreased urine output needs to be determined, This can be due to either decreased renal blood flow (in the face of decreased blood pressure, cardiac output or tissue oxygen delivery) or obstructed urine flow (important to diagnose early as it requires urgent correction).

KEY CLINICAL POINT
In adults urine output should be > 0.5mls per kg per hr i.e. 35mls per hour for a 70kg person.

Acute Kidney Injury (AKI) (Adapted from Makris & Spanou 2016)

Acute Kidney Injury (AKI) is a term used to describe the clinical syndrome that occurs when renal function is acutely decreased to a point that the body accumulates waste products and becomes unable to maintain electrolyte, acid-base and water balance. AKI is associated with poor clinical outcomes for hospitalised patients.

The pathophysiology of AKI is multifactorial and complex. The most common cause of AKI is ischaemia, which can occur for a number of reasons (Table 6). Physiological adaptations, in response to the reduction in blood flow can compensate to a certain degree, but when delivery of oxygen and metabolic substrates becomes inadequate, the resulting cellular injury leads to organ dysfunction.

Table 6: Pre-renal causes for Acute Kidney Injury (Makris & Spanou 2016)

Abnormality	Possible causes
Hypovolaemia	Haemorrhage Volume depletion Renal fluid loss (over-diuresis) Third space (burns, peritonitis, muscle trauma)
Impaired cardiac function	Congestive heart failure Acute myocardial infarction Massive pulmonary embolism
Systemic vasodilation	Anti-hypertensive medications Gram negative bacteraemia Cirrhosis Anaphylaxis
Increased vascular resistance	Anaesthesia Surgery Hepatorenal syndrome NSAID medications Drugs that cause renal vasoconstriction (i.e. cyclosporine)

Management of Pre-Renal Oliguria

When oliguria is due to decreased perfusion i.e. decreased blood pressure or cardiac output, it is potentially reversible. In this circumstance, the most important initial management is to exclude hypovolaemia (decrease in cardiac preload) being the cause. If hypovolaemia is likely (relative or absolute) give an intravenous fluid bolus of 500mls of Normal Saline (adults). Frusemide is not to be given unless you have ruled out all other possible reasons for low urinary output and the patient is clinically fluid overloaded.

Giving a fluid bolus will increase circulating volume, thus increase pre-load, and ultimately increase cardiac output. This will result in increased blood pressure, increased renal perfusion pressure, and ultimately increase the patient's urinary output.

Management of Post-Renal Oliguria

Absolute anuria should be seen as a sign of urinary tract obstruction until proven otherwise:

- Assess bladder size;
- Check catheter patency;
- If there is no catheter in-situ, the patient may need one inserted.

Timely reversion of pre-renal or post-renal causes of AKI usually results in prompt recovery of function, but late correction can lead to kidney damage (Makris & Spanou 2016).

KEY CLINICAL POINT

There is a small window of opportunity to prevent acute kidney injury (AKI)

SUMMARY

- Adult urine output should be >0.5mls/kg/hr i.e. 35mls /hr for a 70kg adult.
- There is a small window of opportunity for reversing oliguria and preventing acute kidney injury (AKI).
- Do NOT give Frusemide to oliguric patients unless you have ruled out all other possible reasons for low urine output and the patient is clinically fluid overloaded.

This section will cover:

- Physiological causes of a depressed level of consciousness
- Common causes of decreased level of consciousness
- New confusion/delirium or altered mentation as a sign of acute illness

Section Six

Level of Consciousness (LOC)

Introduction

In INEWS V2 changes in a person's mental status such as delirium/new confusion are recognized as key early signs of deterioration. A change in a patient's mental status requires an immediate medical review. In INEWS V2, AVPU becomes ACVPU where 'C' represents 'new confusion/delirium or altered mental status' ("confusion / delirium").
Confusion/delirium should be considered to be 'new' until proven otherwise.

KEY CLINICAL POINT
ACVPU replaces AVPU where 'C' represents 'new confusion, delirium or altered mental status'

Confusion/delirium, altered mentation or a depressed level of consciousness are common findings in acute illness. Causes can be considered as follows:

Intracranial disease	Meningitis, encephalitis Epilepsy Cerebrovascular disease, sub-arachnoid haemorrhage Head injury CNS infection

Systemic conditions	Hypoxia or hypercapnia Hypoglycaemia/hyperglycaemia Infection (e.g UTI or RTI) / Sepsis Hypotension, hypo/hyperosmolarity Hyponatraemia Hypo/hyperthermia Hypothyroidism, hypopituitarism, Addison's disease Sedative drugs Hepatic encephalopathy, uraemic encephalopathy

Additional common causes	Pain Constipation Dehydration Urinary retention Medications started or changed Noisy or busy environment Using recreational drugs Heavy alcohol consumption Lack of sleep Recent change in environment

Often the exact cause of the new confusion/delirium is unknown and is likely to be due to a combination of factors.

The physiology of new confusion/delirium/changes in mentation

Inadequate oxygen delivery

Neurons in the CNS, like all other cells in the body, are highly dependant on oxygen. Adequate oxygenation allows for the formation of large amounts of ATP 'energy packets' which are required for all cellular functions (Figure 11). When oxygen supply is inadequate insufficient ATP is produced (Figure 13) which leads to failure of some cellular functions especially in the brain and CNS. This results in the patient developing symptoms of new confusion, altered mentation, delirium or a depressed level of consciousness.

Oxygen supply to the cells in the brain depends on the same factors as oxygen supply to all other tissues in the body. Thus new confusion/delirium, altered mentation or a decrease in level of consciousness can result from a decrease in oxygen delivery due to:

- A decrease in arterial oxygen content
- A decrease in cardiac output
- A decrease in blood pressure.

Inadequate substrate delivery for metabolism

Cells require a substrate in order to form pyruvate which enters the Kreb's Cycle in the mitochondria to produce ATP. Many cells in the body can use glucose, fats or proteins as substrates for energy production. However, neurons can only use glucose as their substrate for energy production. Therefore, if serum glucose levels fall too low neurons will stop producing ATP and cellular function will be compromised. Thus new confusion, altered mentation, delirium or a decrease in level of consciousness can also result from hypoglycaemia.

New confusion/delirium/altered mentation

Delirium is an acute [new] confusion due to illness (Bracken-Scally et al. 2020). Delirium is reversible if recognised and treated early. New confusion/delirium/altered mental status is an early sign of acute illness and clinical deterioration. This is captured on the INEWS patient observation chart as ACVPU where 'C' represents 'new confusion, delirium or altered mental status'.

KEY CLINICAL POINT
New confusion, delirium or altered mental status is a key early sign of clinical deterioration and requires urgent medical review

The Second Irish National Audit of Dementia Care in Acute Hospitals (INAD-2) describes delirium as follows:
"*Delirium is an acute confusion due to illness, typically with reduced ability to focus and sustain attention, and altered alertness (e.g. drowsiness or hyper-vigilance). Many people with delirium develop psychosis (e.g. distressing hallucinations). During an episode of delirium, a person is prone to falls, dehydration, aspiration, iatrogenic events, and death. Delirium is an independent risk factor for mortality in hospital, and in the subsequent years (Bracken-Scally et al. 2020)*".

Who is at risk of developing new confusion/ delirium?

Anyone can develop delirium but it is more common in people who:

- Are acutely unwell or sick
- Have had recent surgery
- Are older (over 65 years)
- Are physically frail
- Have a diagnosis of dementia
- Have had a previous brain injury, stroke or Parkinson's disease
- Are on medications, particularly anticholinergics, psychoactive drugs, and opioids

What are the signs of new confusion / delirium?

A person may experience some or all of the following:

- A sudden change in behaviour and thinking occurring over hours or days
- Confusion i.e. things are mixed up or do not make sense to the person
- Easily distracted, unable to pay attention to what is happening or being said
- Difficulty remembering names and important information
- Seeing and/or hearing things which are not real (hallucinations)
- Thinking or believing things which are not true (delusions)
- Restlessness – not able to stay still, climbing out of bed repeatedly
- Changing levels of energy – from very drowsy to very alert and agitated
- Speech is sluggish or slow
- Day and night may become mixed up

KEY CLINICAL POINT
Check blood glucose in any patient showing signs of new confusion, delirium, altered mentation or a reduced level of consciousness

Delirium in dementia

New confusion/delirium can develop in patients with pre-existing dementia. In fact, the biggest pre-disposing factor for developing delirium is a baseline cognitive impairment, vision or hearing impairment, advanced age and frailty. Delirium can also precipitate or accelerate dementia. Indeed, delirium is one of few currently preventable causes of dementia. Thus, in considering the care of a person with known or suspected dementia in an acute hospital, we must have a particular focus on delirium prevention, detection and reduction (Bracken-Scally et al. 2020).

KEY CLINICAL POINT
Delirium is one of the few currently preventable causes of dementia

KEY CLINICAL POINT
Confusion should be considered 'new' until proven otherwise

KEY CLINICAL POINT
"The possibility of 'new confusion' should be routinely considered with every set of INEWS observations completed"

How do I score a patient using ACVPU on the INEWS patient observation chart ?

- If the patient is considered to have new confusion/delirium they score a 'C' = 3
- If it is unknown whether the patient's confusion/delirium is new or their lived baseline it should be considered to be new confusion/delirium until proven otherwise - they therefore score a 'C' = 3
- If / when it is determined that the level of confusion is the patient's lived baseline and they are otherwise alert (ie they do not score a V, P or U) then their ACVPU score returns to 'Alert' and they are recorded as an 'A' and score a '0' thus reflecting the patient's status.

This section covers

- ISBAR Communication Tool
- Documentation
- Communication & Safety Huddles
- Failure to Escalate Care
- Cycle of Clinical Futility

Section Seven
Communication

Introduction

One of the most important factors in determining an acutely ill patient's outcome is the quality of the communication among the clinicians involved.

In a review of serious incident investigation reports Mullen (2013) identified communication between clinical specialties as problematic with communication of unexpected clinically significant or urgent findings relating to deteriorating patients highlighted as warranting particular attention.

A systematic evaluation of the quality of reports of serious incidents investigation in Irish hospitals identified problems with both individual and team communication as causal factors leading to death or serious harm (McCaughan 2016).

KEY CLINICAL POINT
Where a patient's condition and /or a situation is deemed to be critical, this should be clearly stated at the outset of the conservation

The ISBAR clinical communication tools should be used when communicating information verbally and in writing between healthcare professionals. Where a patient's condition and /or a situation is deemed to be critical, this should be clearly stated at the outset of the conservation (DoH 2020).

ISBAR Communication Tool

The **I**dentify, **S**ituation, **B**ackground, **A**ssessment and **R**ecommendation (ISBAR) technique is an easy, structured and useful tool to help communicate concerns, and call for help or action.

It helps to prevent vital information being missed, provides a recognised framework within which to organise what you want to say and give the opportunity for you to state what outcome you desire from the conversation.

Table 7: ISBAR Communication Tool

ISBAR Communication Tool	
I **Identify**	**Identify:** Yourself, the person you are contacting & the patient
S **Situation**	**Situation:** Why are you calling? (Identify your concern) *Where a patient's condition and /or a situation is deemed to be critical, this should be clearly stated at the outset of the conservation*
B **Background**	**Background** What is the relevant background?
A **Assessment**	**Assessment:** What do you think is the problem
R **Recommendation**	**Recommendation** What do you want to do?

For example:

A 75 year old lady with a history of Ischaemic Heart Disease is admitted to hospital with a fractured neck of femur. Twelve hours post-operatively she complains of chest pain and her arterial oxygen saturation has fallen to 88% on 2L oxygen via nasal prongs and she has an INEWS score of 6. As the nurse caring for this patient you are concerned that she is acutely unwell and needs urgent attention (call SHO as per escalation protocol). The ISBAR communication technique would proceed as follows:

IDENTIFY:
"This is Sarah calling from 7 East about Mrs Smith, is this Dr. Jones?"

SITUATION:
"She is a 75-year-old lady who has an INEWS score of 6 due to a drop in her SpO2 to 88% on 2 L/min of O2 via nasal prongs, she is also tachycardic and tachypnoeic. She is also complaining of chest pain."

BACKGROUND:
"She is twelve hours post-op following surgery for a fractured neck of femur and she has a history of ischaemic heart disease."

ASSESSMENT:
"I think she is acutely unwell and may have ....."
In this case she may have a pulmonary embolus, a myocardial infarction, pneumonia or a fat embolus. If you are not sure what the cause of the problem is you can say that you think the patient is unwell and that you are concerned about them.

RECOMMENDATION:
"This patient requires an immediate medical review. I have increased her inspired oxygen in the meantime to 15 L/min on a non-re-breather mask."

You have effectively communicated the reason why you are calling, given the person some background information that may help in identifying the cause of the problem, given an idea of how ill you think the patient is and identified that you feel the patient needs review.

Documentation

Good documentation is key to effective communication and has the following purposes:

- Helps convey vital patient information between healthcare professionals
- Helps the flow of information from one shift to the next
- Helps to clarify your own thought processes
- Is a medico-legal requirement.

It is important to identify who needs to be informed about a deteriorating patient, what information needs to be communicated, when & how often and document appropriately.
For further guidance see https://www.hse.ie/eng/about/who/qid/quality-and-patient-safety-documents/v3.pdf

Communication and Safety Huddles

Safety huddles are one way of communicating key patient information within the multidisciplinary team at agreed time(s) during the day.
A safety huddle is a short multidisciplinary briefing, held at a predictable time and place, which focuses on the patients most at risk.

Effective safety huddles are:

- multidisciplinary
- of 10 - 15 minutes duration or less
- held at predictable time and place
- focus on patients most at risk e.g. cues for caution (DoH 2020)
- clear on agreed actions and who will complete them
- use visual feedback of data e.g. whiteboard
- enhance teamwork through communication and co-operative problem-solving;
- share understanding of the focus and priorities for the day
- improve situational awareness of safety concerns.

https://www.england.nhs.uk/atlas_case_study/improving-patient-safety-by-introducing-a-daily-emergency-call-safety-huddle/

KEY CLINICAL POINT
A safety huddle is a short multidisciplinary briefing, held at a predictable time and place, and focuses on the patients most at risk

Failure to escalate

Escalation of care enables staff to have a patient reviewed by a senior clinician (Nursing and /or Medical). Failure to escalate care when indicated puts a patient at risk of a serious adverse event. Evidence shows that healthcare professionals frequently fail to escalate care (O'Neill et al 2021). This occurs for a number of reasons. Chief among these reasons are:

- a feeling by medical and nursing staff that they should be able to manage a deteriorating patient's care without seeking help
- staff over-confidence in their clinical experience
- fear of looking incompetent if they do call for help
- fear of reprimand or other negative responses from senior colleagues and/or the emergency response team.

Other barriers and facilitators to escalation of care by healthcare professionals can be seen in Figure 24.

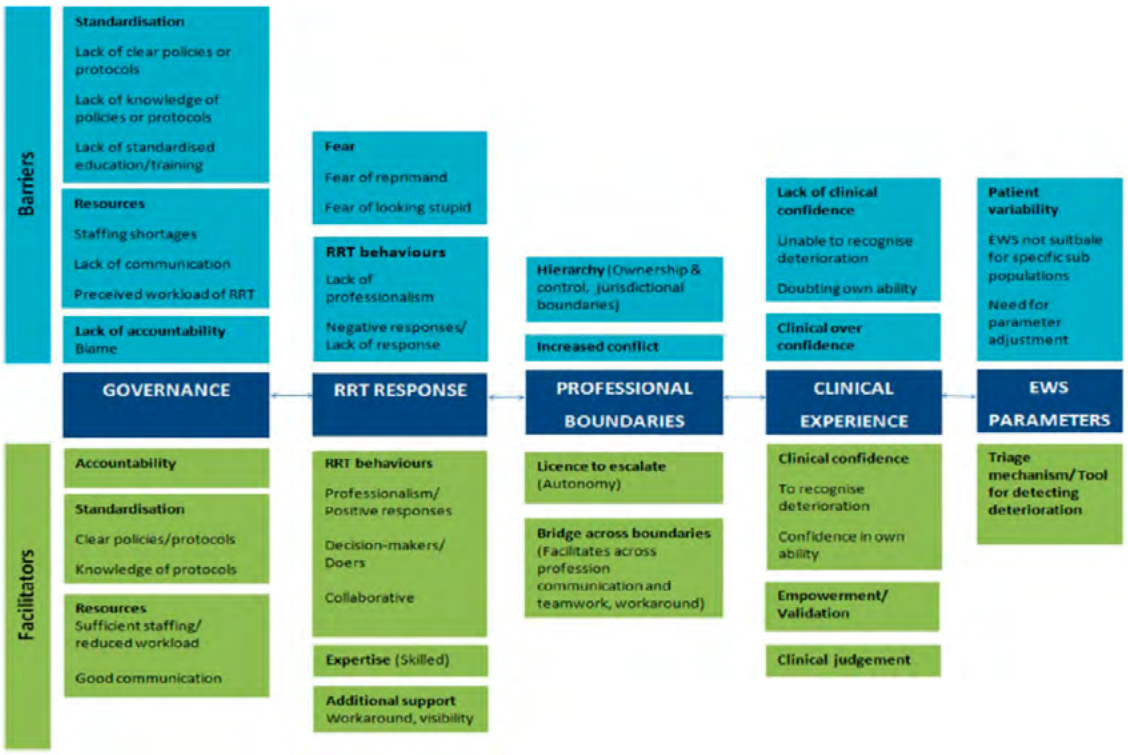


Figure 24 - outlines other barriers and facilitators to escalation of care (O'Neill et al. 2021).

This fear of escalating care – of calling for assistance from more senior colleagues – can result in what is known as a 'cycle of clinical futility'. The cycle of clinical futility can be defined as repeated reviews and/or interventions by (usually junior) nursing and medical staff with limited monitoring of response to interventions, limited clinical response to interventions or a decline in the patient's condition. Not calling for senior input early in a patient's deterioration trajectory can result in a serious adverse event for that patient ie cardiopulmonary arrest, unplanned admission to ICU or even death.

Buist (2018), writing about clinical futile cycles, describes three human factor issues at the patient interface – competency, cognition and culture – set within the traditional hierarchical referral model of care in hospitals. He writes that "it is at the bedside that staff are trapped in a clinical futile cycle, unable to get out of it due to either clinical incompetency (not able to recognise and act) and/or culture whereby calling for help may not be considered the norm in that ward or at that time" (Buist 2018).

A culture which supports and encourages staff to seek advice and input from their senior colleagues is vital to ensure that the patient is seen by the right person at the right time.

Reassess

When caring for a deteriorating patient, health care staff must always review the patient to ensure that the plans or actions have made a difference to the patient. It is NOT adequate to say you have informed someone, discharge your responsibility and forget about the patient. It is as much your responsibility to ensure that something is done, as it is the responsibility of the person you informed to come and attend to the patient.

KEY CLINICAL POINT
A continuous cycle of review is required until the patient improves

For a change of shift, all concerns and outstanding issues in relation to patients must be documented and verbally conveyed to the health care professional taking over the care of the patient to ensure continuity of care and follow-up.

Where the patient is not improving consider Immediate Senior Clinician Review. The need for critical care review either on the ward by the critical care outreach team or by transfer to higher level of care. A higher level of care maybe delivered at the bedside on the ward or by transferring to HDU/ICU.

Summary

- It is important to recognise when there is an abnormality in vital signs and make sure someone more senior knows about it and that someone is attending the patient appropriately.

- Use ISBAR when communicating.

- When documenting a medical entry always document:
  H - History
  E - Examination
  I - Impression/diagnosis
  P - Management plan

- Management plans should include:
  a. Observation orders
  b. Nursing orders
  c. Physiotherapy orders
  d. Change in therapy orders
  e. Investigations/interventions orders
  f. Notification orders

This section covers:

- Deteriorating Patient/ EWS Clinical Governance Group Aim & Objectives
- Membership of the Deteriorating Patient / EWS Clinical Governance Group
- Responsibilities of the Deteriorating Patient / EWS Clinical Governance Group
- Available Dataset to support performance review

Section Eight
Governance

Introduction

Success and sustainability of the Irish National Early Warning Systems (e.g. INEWS, PEWS) requires executive and clinical leadership, and structured organisational governance.

Within an organisation an effective governance framework for EWS provides the structure from which the key activities of risk identification, monitoring and evaluation for improvement of the early warning system can occur.

Each organisation is accountable for continuously improving the quality and reliability of their services and ensuring high standards of care. The factors known to contribute to this aim are; transparency regarding accountability and responsibility for maintaining standards, creation of a learning organisation, supports to enable clinical excellence to flourish, and use of quality improvement methodologies.

Governance of EWSs must form part of the hospital's overall safety and quality framework. These structures should have the capacity to monitor and continuously improve operation of the deteriorating patient early warning systems and care to the patient.

Regulated healthcare professionals are guided in their day-to-day practice by their codes of professional conduct which helps them to understand their professional responsibilities in caring for patients in a safe, ethical and effective way. A variety of healthcare professionals are involved in developing, implementing and sustaining EWSs governance. The responsibility to ensure the governance is established, transparent and operating effectively lies with the executive board/management team.

Organisations may develop their own standalone governance framework or they may be embedded within a broader structure e.g. Hospital Group. Regardless of the size of the governance group, oversight of the EWS must be multidisciplinary. This shared responsibility and accountability (executive, managers, healthcare professionals and consumers) for providing safer, effective, reliable person centred care for deteriorating patients, underpinned by continuous quality improvement forms the basis for an EWS clinical governance framework.

Clinical governance can be described as a framework through which healthcare teams are accountable for the quality and safety of care delivered to patients in their care. It is built on the model of the chief executive officer/general manager or equivalent working in partnership with the clinical director, director of nursing/midwifery and service/professional leads. A key characteristic of clinical governance is a culture and commitment to agreed service levels and quality of care to be provided (HSE February 2012).

The Department of Health, National Clinical Effectiveness Committee (NCEC) National Clinical Guideline (NCG) No 1 INEWS V2 recommendations 28 to 33 establish the standards on which clinical governance frameworks for INEWS should be developed. These standards also provide guiding principles for other early warning systems to be clinically governed. In today's environment where resources are limited and challenged to meet all competing demands, it is considered reasonable to bring all individual early warning systems governance together under one clinical governance umbrella for deteriorating patients.

KEY CLINICAL POINT
Clinical governance can be described as a framework through which healthcare teams are accountable for the quality and safety of care delivered to patients in their care

Deteriorating Patient / EWS Committee

Aim:

The Governance committee should oversee the ongoing performance and improvement of the anticipation, recognition, escalation, response and evaluation elements of the INEWS system locally.

Objectives:

- Be accountable for its decisions and actions
- Monitor effectiveness of clinical and quality improvement interventions
- Monitor effectiveness of education delivered
- Have a role in reviewing clinical outcome data and healthcare audits
- Provide advice about allocation and prioritisation of resources
- Include service users, clinicians, managers and executives in the membership of the group
- Develop quality improvement plans and report on progress.

Membership of the Deteriorating Patient / EWS Committee

Recommended membership of the Hospital-level Deteriorating Patient/EWS clinical governance group:

- Consultant Lead, EWS
- Senior Nurse Manager
- Frontline nursing
- Patient, family, carer
- NCHD Lead(s)
- Health and Social Care Professionals
- Critical Care Outreach RANP/CNS/CNMII
- Sepsis Lead
- Resuscitation Officer
- Palliative Care
- Quality and Patient Safety
- Educators/Practice Development
- Admin support

KEY CLINICAL POINT
Patients, families and carers have experiences of recognition and response systems that can inform planning and improvement activities

The above is not an exhaustive list, depending on clinical context other specialties may need to be included. Administrative support for the group would be of benefit.

Where practical this forum should seek to align governance for sepsis, cardiac arrest, resuscitation, INEWS, PEWS, IMEWS, EMEWS, Mortality & Morbidity, ICU admissions and discharges.

Role of patients, family members and/or carers

There is increasing evidence that health systems are safer when patients, families and carers are involved in healthcare design and delivery. Patients, families and carers have experiences of recognition and response systems that can inform planning and improvement activities. The role of patients, family members and/or carers ("patients") on EWS Governance Committees is new. The contributions of patients on the national INEWS Guideline Development Group and other national DPIP projects significantly enhanced programme outputs. To ensure meaningful participation of patient representatives the role of patients on the governance group will need to be facilitated and supported by the governance committee Chair and senior personnel. Detailed guidance can be found on Person and Family Engagement - HSE.ie

Responsibilities:

Senior hospital management should:

- Nominate an executive sponsor at senior management level with overall responsibility for the ongoing performance and improvement of EWSs

- Designate a Consultant Lead for Deteriorating Patient/EWSs as Chair of the group with responsibility for the ongoing performance and improvement of EWSs and associated clinical outcomes e.g sepsis, cardiac arrest

- Ensure the EWS Governance Group reports directly to the Executive Management Board (or equivalent) to appraise of progress and escalate risks

The hospital-level Deteriorating Patient/EWS clinical governance group has responsibility to:

Governance for Quality	• ensure the implementation of current relevant national clinical guidelines (EWSs, Sepsis, Clinical Handover etc)
	• ensure the development of local EWS implementation policies. These may relate to:
	  ▪ escalation processes (internal and external)
	  ▪ protocols to provide bedside, urgent & emergency response
	  ▪ monitoring and documenting observations
	  ▪ clinical communication
	  ▪ advance care directives, treatment-limiting and end-of-life decision-making
	• liaise with Palliative Care colleagues on EOLC pathways and their relationship to EWSs
	• authorise and support healthcare workers to escalate care until satisfied with outcome for patient by promoting an environment of psychological safety
	• actively support safety initiatives such as safety huddles in clinical areas
	• review and address poor adherence to local EWS policies and procedures

Education	Monitor the effectiveness of related education programmes, examples include education related to: ■ basic and advanced life support ■ monitoring and documenting observations ■ safety huddle / safety pause (anticipatory care) ■ recognition and escalation protocols ■ bedside, urgent & emergency response competencies ■ Sepsis Recognition & Management ■ Critical Care Outreach ■ physical assessment ■ clinical communication ■ simulation, mentorship and education principles ■ use of quality improvement methodologies
Communication	Maintain and communicate a focus on patient safety and high-quality care by: • encouraging and acknowledging clinical effectiveness and high-performing teams. • focusing on learning and improving systems by communicating information about risks and successful strategies throughout an organisation. • engaging with clinicians to identify and resolve problems (e.g. executive leadership clinical rounds or forums). • reporting EWS improvement projects and outcomes via newsletters, minutes of meetings, staff meetings or alternative forums. • Submit projects to National Excellence / Innovation Award or similar.

Resource Management	• Consider and plan resource needs based on performance and outcome data. This includes ensuring that the resources required to provide bedside, urgent & emergency response (such as equipment, pharmaceuticals and personnel) are available through, for example, service planning processes. • contribute to relevant business cases based on evidence for new technologies for digital EWS systems and digital monitoring
Quality & Patient Safety	Collaborate with designated Risk Management/Quality & Patient Safety personnel to • Report, investigate and analyse incidents, including near misses relating to the deteriorating patient. This could include any component of an EWS e.g. clinical handover processes, rapid response system use, escalation of care etc • Identify, analyse and respond to consumer, patient, family and carer complaints about recognition and response to clinical deterioration e.g. escalation of care and clinical communication • Review incidents related to recognition and response systems, and consider resource needs • Incorporate information from incidents and complaints into planning processes for EWS • Identify and acknowledge good practice and incorporate learning into planning processes for EWS

Measurement for Quality & Improvement

Oversee the ongoing performance of EWSs by:

- developing clinical audit schedule
- identifying and agreeing measurable clinical outcomes e.g. number of unanticipated cardiopulmonary arrests, unplanned admissions/readmissions to ICU
- Collecting and reviewing data in all acute care areas to enable evaluation of key activities such as:
    - monitoring and documenting observations
    - escalation of care and use of systems to provide emergency response. eg Resus Team / Critical Care Outreach
    - Clinical Outcomes
    - clinical communication
    - education and training
    - morbidity and mortality
    - advance care directives, treatment-limiting and end-of-life decision-making
- Prioritise and/or undertake continuous improvement plans / projects in response to performance data. This should include regularly reviewing key performance indicators, developing action plans, prioritising improvement areas, and providing support when implementing improvement projects.
- Engage and involve service users in design, planning and delivery of all care demonstrates a commitment to person centred care.
- Engage with staff to ensure they are valued, listened to and provided with tools, resources and skills to do meaningful work.

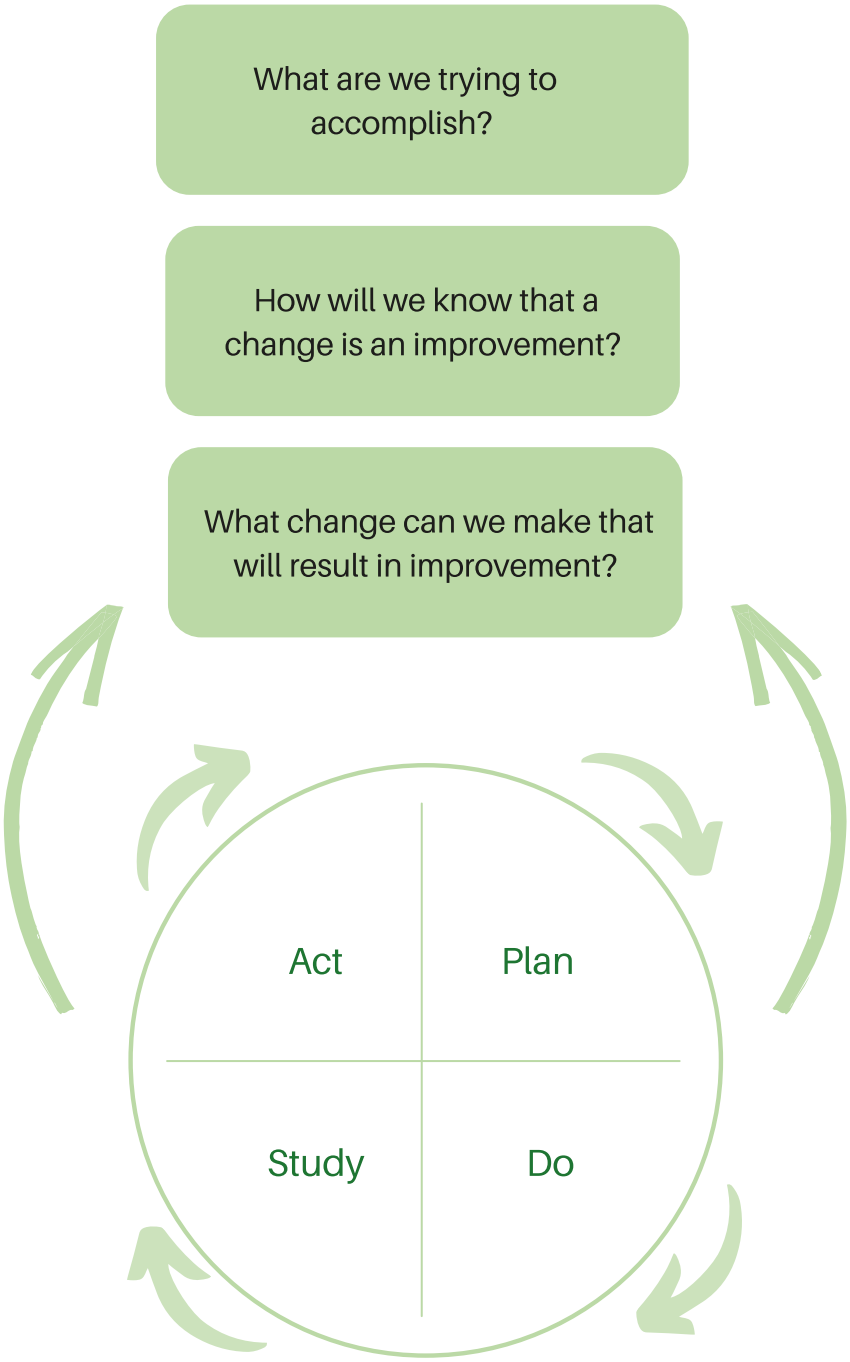


Figure 25 - Model for Improvement

The National Quality Improvement Team's 'QI Method Toolkit' is available as a resource to support staff working on QI projects or initiatives at the following link: QI Method Toolkit

Accountability Reporting Structure

The illustration below suggested reporting structure for the Deteriorating Patient / EWS Governance.

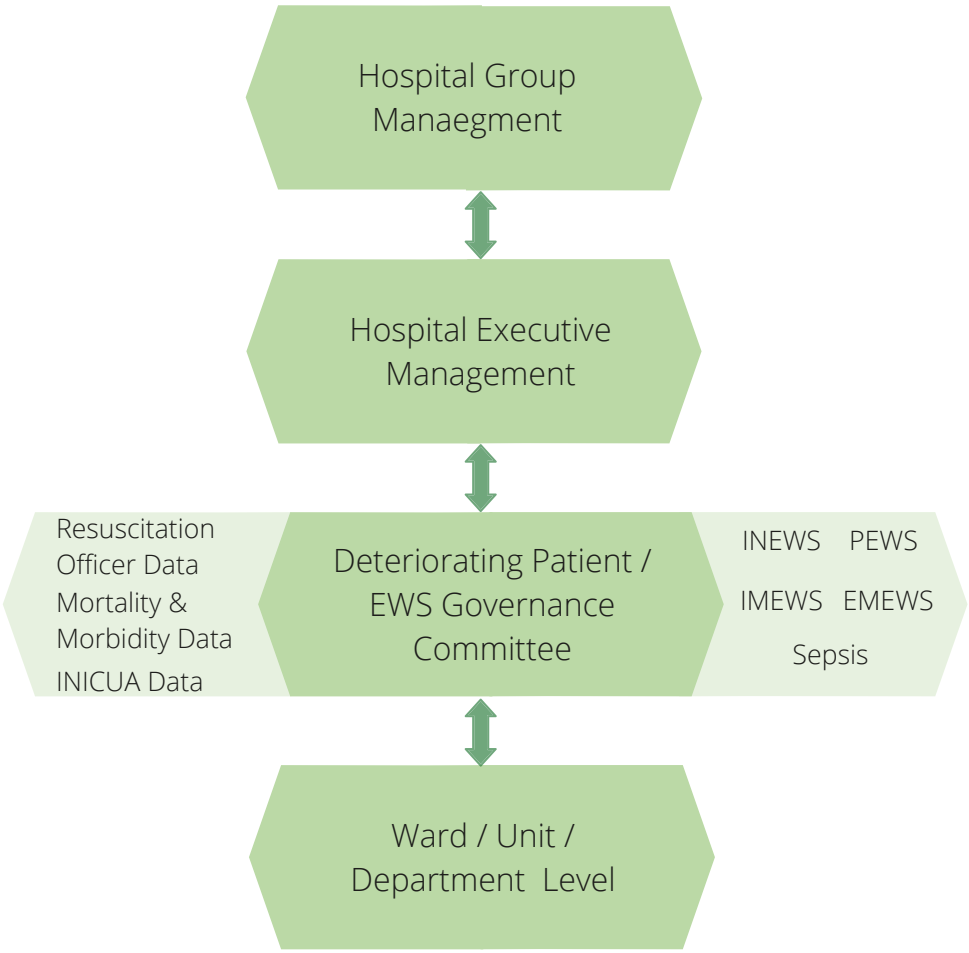


Figure 26 - Reporting structure for the Deteriorating Patient / EWS Governance

Frequency of Meetings:

Meeting should be held quarterly at a minimum or more frequently as required.

Reports:

Quarterly EWSs performance and progress reports to the hospital executive.

Table 8 Available Data to support performance review

Performance	Available Data
Monitoring and documenting observations	• NMQCM 'Patient Monitoring and Surveillance' (on Acute Care Questionnaire) Metric • EWS Audit Templates (INEWS, PEWS, IMEWS)
Escalation of care, and use of systems to provide emergency response.	• EWS Audit Templates (INEWS, PEWS, IMEWS) • Critical Outreach Data
Clinical Outcomes (total number of cardiorespiratory arrests, unplanned admission & readmissions to ICU, Sepsis)	• Local Resuscitation Dataset • Irish National ICU Audit (INICUA) • NQAIS Clinical Data • Sepsis Audit
Clinical Communication	• ISBAR3 Communication Tools (inter-departmental and shift clinical handover) • ISBAR Communication Tool for communication in relation to a deteriorating patient
Education and Training	• HSELand Compliance Reports • National Employment Records (NCHDs) INEWS Record – local Medical Manpower
Morbidity and Mortality	• National Audit of Hospital Mortality
Advance care directives, treatment-limiting and end-of-life decision-making	• Palliative Care Dataset (Acute Hospital)

Section Nine

Supplementary Material

Adult Airway

Oxygen cannot move into the lower respiratory tract unless the airway is patent. Causes of airway obstruction can either be mechanical or functional.

Causes of airway obstruction:

- Functional airway obstruction – may result from decreased level of consciousness, whereby the muscles relax and allow the tongue to fall back and obstruct the pharynx

- Mechanical airway obstruction – may be through aspiration of a foreign body or swelling/bleeding in the upper airway (e.g. trauma, allergy and infection). Mechanical obstruction may also be caused by oedema or spasm of the larynx.

Examination of the Airway

Recognition of airway obstruction is possible using a "look, listen, feel" approach.

Look: Complete airway obstruction can cause paradoxical chest and abdominal movements ("see-saw" like movement, where inspiration is associated with outward movement of the chest, but inward movement of the abdomen). Other signs of airway obstruction include use of accessory muscles (neck and shoulder muscles) and tracheal tug.

Listen: In complete airway obstruction, there will be no breath sounds at the mouth or nose; in incomplete obstruction, breathing will be noisy (stridor = inspiratory wheeze) and breath sounds are reduced.

Feel: Placing your hand immediately in front of the patient's mouth allows you to feel if there is any air moving in or out of the airway.

Management of the Obstructed Airway

In the majority of patients in hospital, the patient's airway obstruction is functional, i.e. due to a depressed level of consciousness. Simple clinical manoeuvres may be required to reopen the airway (see Figure 27).

1. Head tilt
2. Chin lift
3. Jaw thrust
4. Insertion of an oropharyngeal or nasopharyngeal airway.

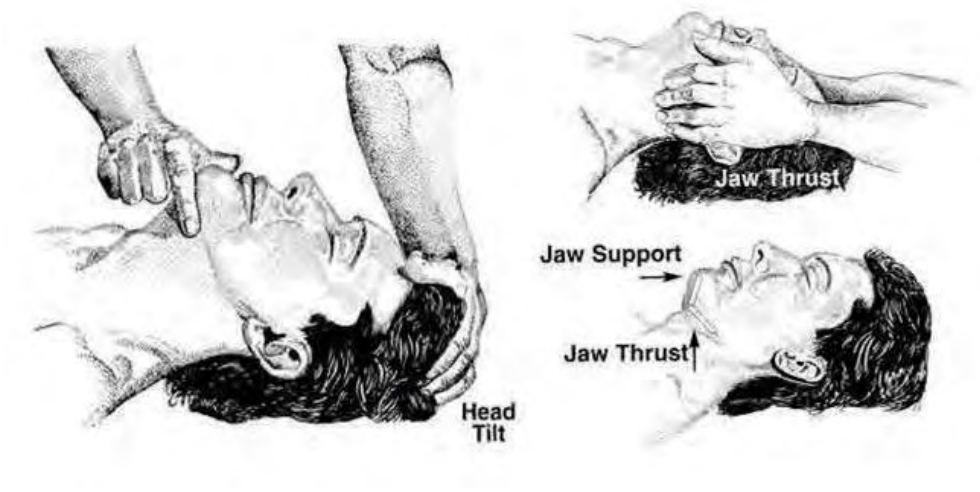


Figure 27 - Simple Airway Manoeuvres

Suctioning of the airway using an oropharyngeal sucker may be required to remove any vomitus or secretions, which may be contributing to airway obstruction. Due care must be taken when performing oral suctioning so as not to further compromise the patient's airway.

If the patient continues to have a depressed level of consciousness and is unable to protect his/her own airway, endotracheal intubation may be required. Endotracheal intubation may only be performed by experienced staff.

In all patients with an airway obstruction or patients who are unable to maintain an adequate airway, activate the cardiac arrest system (including anaesthesia) or ERS. In rare cases, the airway obstruction may be due to mechanical factors, which are not so easily treated, e.g. airway swelling, post-operative haematoma, infection. This is a medical emergency, therefore activate the cardiac arrest system or Emergency Response System.

A surgical airway may be required if endotracheal intubation is not possible (called a cricothyroidotomy), and this should only be attempted by competent, experienced staff.

Breathing

Breathing is required to move adequate oxygen into, and carbon dioxide out of the lungs.

Breathing requires:

- An intact respiratory centre in the brain;
- Intact nervous pathways from brain to diaphragm and intercostals muscles;
- Adequate diaphragmatic & intercostals muscle function;
- Unobstructed air flow (large and small airways).

Examination of Breathing

The "look, listen, feel" approach is a practical method of quickly determining causes of abnormalities in breathing.

Look

Respiratory rate is an important marker of a deteriorating patient. When you walk into a room and the first thing you notice is the patient's breathing, there is a significant problem with the patient.

Look for signs of respiratory failure which can include:

- Abnormal respiratory rate;
- Use of accessory respiratory muscles;
- Sweating/pallor;
- Central cyanosis;
- Abdominal breathing;
- Shallow breathing;
- Unequal chest movement.

Listen

Initially listen for:

- Noisy breathing which may indicate secretions in the upper airways;
- Stridor or wheeze which may indicate partial airway obstruction.

Then auscultate with a stethoscope to assess breath sounds:

- Quiet or absent breath sounds may indicate the presence of a pneumothorax or a pleural effusion;
- Bronchial breathing may indicate the presence of consolidation.

Feel

1. Palpation

Palpate the trachea and chest wall:

- tracheal deviation indicates mediastinal shift which may be due to:
  -a pneumothorax or pleural fluid-tracheal deviation away from the lesion;
  -lung collapse-tracheal deviation toward the lesion.
- chest wall crepitus (subcutaneous emphysema) is highly suggestive of a pneumothorax, oesophageal or bronchial rupture;
- asymmetrical chest wall movement may indicate unilateral pathology e.g. consolidation, pneumothorax.

2. Percussion:

- Hyper-resonance indicates pneumothorax;
- Dullness indicates consolidation or pleural fluid.

Measuring Respiratory Rate (RR)

- Counting RR and observing depth, symmetry and pattern of breathing.
- If a patient has been prescribed oxygen, ensure the oxygen mask or nasal cannula is correctly positioned and recorded on the observation chart before recording RR.
- Observe their respiratory function, for example, whether they can talk in full sentences (Dougherty and Lister 2015). Taking a breath mid-sentence or one-word answers may be a sign of respiratory distress.
- Note whether the patient is alert and orientated to time and place (Dougherty and Lister 2015). Changes in cognitive status, such as confusion, may be due to hypoxia, cerebral injury or side-effects of medication, such as opiates .
- Using a watch with a second hand, count breaths (number of times the chest moves up and down) for a full minute. This length of time is needed as changes can occur in the respiratory pattern and rate.
- While observing the RR, note the rhythm, which may indicate signs of underlying illness. Respirations should be regular with equal pause between each breath.
- Observe the patient's lips for signs of cyanosis (blue tinge), which may indicate hypoxia (low oxygen saturation [SpO_2]). Pulse oximetry is a valuable tool to measure SpO_2 but it has limitations when a patient's peripheral circulation or condition is compromised – for example, through tremor, shivering, hypovolaemia, hypothermia, heart failure or vaso-constriction (Elliott and Coventry 2012). Pulse oximetry is less accurate when SpO_2 is <80% (Jubran 2006).

Glasgow Coma Scale

Another common method of measuring CNS function is the "Glasgow Coma Scale" (GCS). The GCS is not included in the NEWS calculations but may be indicated for specific patients or on specific wards. The GCS is divided into three sections - best motor response, best verbal response, and best eye-opening response (see figure 28).

Patients with GCS <8 will almost certainly require intubation, as they are unable to protect their own airway. Further assistance will be required with anyone who has this level of consciousness.

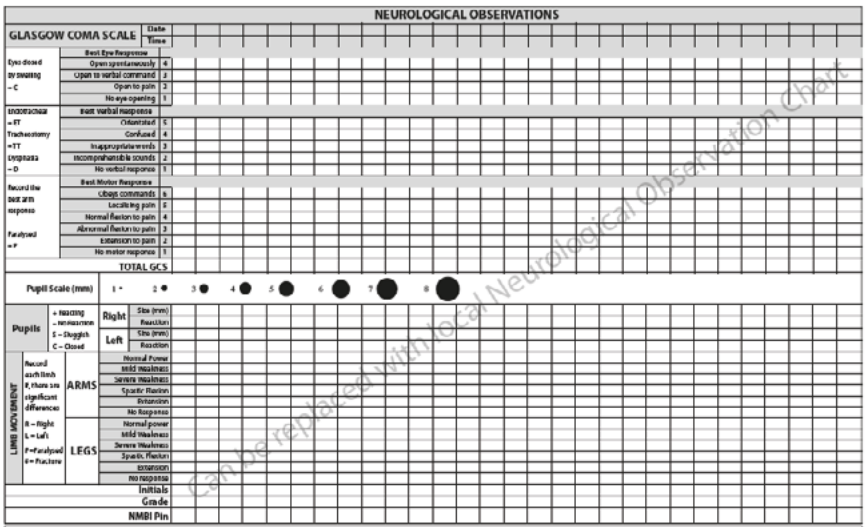


Figure 28 - Glasgow Coma Scale (GCS)

Pupillary Size

Pupils should be checked as part of the neurological observations, and when there is any reduction in the patient's level of consciousness. Any change in the size, equality or reactivity of the patient's pupil is an important clinical sign of a change in the patient's neurological condition. This can provide important diagnostic clues.

Bilateral pupillary dilatation
Causes:

- Sympathetic over-activity e.g. fear, stress, anxiety, hypoglycaemia;
- Sympathomimetic medication administration e.g. administration of adrenaline in an arrest situation;
- Anticholinergic medication activity e.g. atropine, tricyclic antidepressants, ipratropium nebulizer.

Bilateral pin-point pupils
Causes:

- Opioids/opiates;
- Cholinergic drugs-neostigmine, organophosphates;
- Brainstem CVA.

Unequal pupils
Causes:

- Previous surgery;
- Prosthetic eye;
- Eye drops;
- Brain lesions, aneurysms, infections;
- Glaucoma.

Management of Decreased Level of Consciousness

1. Check airway and breathing: ensure airway is patent:
    i. Head tilt, chin lift, jaw thrust
    ii. Insert oropharyngeal or nasopharyngeal airway if required.
2. Apply high-flow oxygen.
3. Measure blood glucose and correct if ≤ 3mmol/l (administer 50mls of 50% glucose intravenously) *(Check local policy for strength of glucose to be used)*.
4. Immediate medical review if GCS falls > 2 points.
5. If respiratory rate or arterial oxygen saturation is decreased, the patient may need ventilatory assistance using self-inflating bag and mask.
6. Ensure intravenous access; 500mls intravenous fluid bolus may be required if patient is hypotensive.
7. Reverse any drug-induced CNS depression e.g. Naloxone for opioid overdose (requires a medical order).
8. If the airway is patent, and the patient is breathing, place the patient supine in lateral recovery position.

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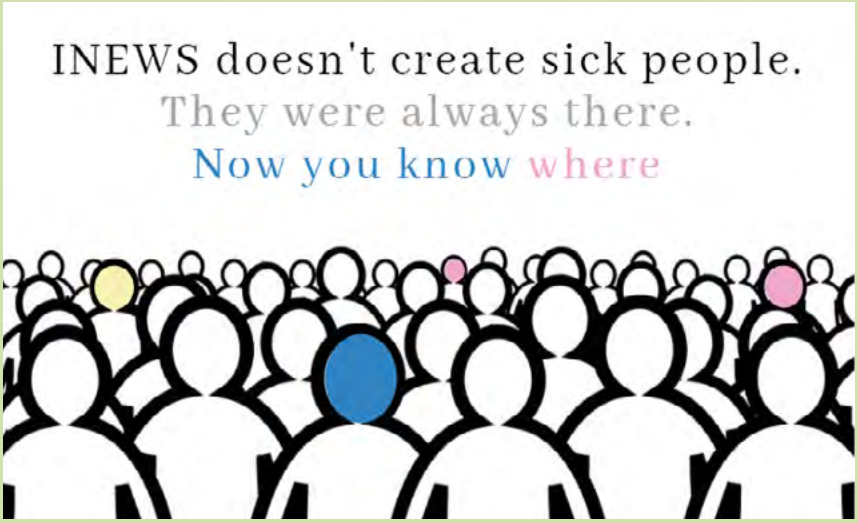

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