

## ICU

**Intensive care units (ICUs) are specialist hospital wards that provide treatment and monitoring for people who are very ill**

They're staffed with specially-trained healthcare professionals and contain sophisticated monitoring equipment.

ICUs are also sometimes called critical care units (CCUs) or intensive therapy units (ITUs).

Intensive care has been defined as “a service for patients with potentially recoverable conditions who can benefit from more detailed observation and invasive treatment than can safely be provided in general wards or high dependency areas.”

### **Who to admit**

Intensive care is appropriate for patients requiring or likely to require advanced respiratory support, patients requiring support of two or more organ systems, and patients with chronic impairment of one or more organ systems who also require support for an acute reversible failure of another organ. Early referral is particularly important. If referral is delayed until the patient's life is clearly at risk, the chances of full recovery are jeopardised.

### Indication for admission in ICU

- Threatened airway
- All respiratory arrests
- Respiratory rate  $\geq 40$  or  $\leq 8$  breaths/min
- Oxygen saturation  $< 90\%$  on  $\geq 50\%$  oxygen
- All cardiac arrests
- Pulse rate  $< 40$  or  $> 140$  beats/min
- Systolic blood pressure  $< 90$  mm Hg

- Sudden fall in level of consciousness (fall in Glasgow coma score >2 points)
- Repeated or prolonged seizures
- Rising arterial carbon dioxide tension with respiratory acidosis
- Any patient giving cause for concern
- Neuromuscular diseases
- Head injury
- Tetanus
- Multiple organ failure

### **Categories of organ system monitoring and support**

#### **Advanced respiratory support**

- Mechanical ventilatory support (excluding mask continuous positive airway pressure (CPAP) or non-invasive (eg, mask) ventilation)
- Possibility of a sudden, precipitous deterioration in respiratory function requiring immediate endotracheal intubation and mechanical ventilation

#### **Basic respiratory monitoring and support**

#### **Circulatory support**

- Need for vasoactive drugs to support arterial pressure or cardiac output
- Support for circulatory instability due to hypovolaemia from any cause which is unresponsive to modest volume replacement (including post-surgical or gastrointestinal haemorrhage or haemorrhage related to a coagulopathy)
- Patients resuscitated after cardiac arrest where intensive or high dependency care is considered clinically appropriate

- Need for more than 50% oxygen
- Intra-aortic balloon pumping

- Possibility of progressive

deterioration to needing advanced  
respiratory support

- Need for physiotherapy to clear  
secretions at least two hourly

- Patients recently extubated after  
prolonged intubation and  
mechanical ventilation

- Need for mask continuous positive  
airway pressure or non-invasive  
ventilation

- Patients who are intubated to  
protect the airway but require no  
ventilatory support and who are  
otherwise stable

### **Neurological monitoring and support**

- Central nervous system depression, from whatever  
cause, sufficient to prejudice the airway and protective  
reflexes

- Invasive neurological monitoring

### **Renal support**

- Need for acute renal replacement therapy  
(haemodialysis, haemofiltration, or haemodiafiltration)

### **Factors to be considered when assessing suitability for admission to intensive care**

- Diagnosis
  - Severity of illness
  - Age
  - Coexisting disease

- Physiological reserve
- Prognosis
- Availability of suitable treatment
- Response to treatment to date
- Recent cardiopulmonary arrest
- Anticipated quality of life

### **Basic monitoring requirements for seriously ill patients**

- Heart rate
- Blood pressure
- Respiratory rate
- Pulse oximetry
- Hourly urine output
- Temperature
- Blood gases

### **Respiratory support**

All seriously ill patients without pre-existing lung disease should receive supplementary oxygen at sufficient concentration to maintain arterial oxygen tension  $\geq 8$  kPa or oxygen saturation of at least 90%. In patients with depressed ventilation (type II respiratory failure) oxygen will correct the hypoxaemia but not the hypercapnia. Care is required when monitoring such patients by pulse oximetry as it does not detect hypercapnia.

A few patients with severe chronic lung disease are dependent on hypoxic respiratory drive, and oxygen may depress ventilation. Nevertheless, life threatening hypoxaemia must be avoided, and

if this requires concentrations of oxygen that exacerbate hypercapnia the patient will probably need mechanical ventilation.

Any patient who requires an inspired oxygen concentration of 50% or more should ideally be managed at least on a high dependency unit. Referral to intensive care should not be based solely on the need for endotracheal intubation or mechanical ventilation as early and aggressive intervention, high intensity nursing, and careful monitoring may prevent further deterioration.

Endotracheal intubation can maintain a patent airway and protect it from contamination by foreign material such as regurgitated or vomited gastric contents or blood. Putting the patient in the recovery position with the head down helps protect the airway while awaiting the necessary expertise for intubation. Similarly, simple adjuncts such as an oropharyngeal airway may help to maintain airway patency, although it does not give the protection of an endotracheal tube.

Breathlessness and respiratory difficulty are common in acutely ill patients. Most will not need mechanical ventilation, but those that do require ventilation need to be identified as early as possible and certainly before they deteriorate to the point of respiratory arrest. The results of blood gas analysis alone are rarely sufficient to determine the need for mechanical ventilation.

Several other factors have to be taken into consideration:

*Degree of respiratory work*—A patient with normal blood gas tensions who is working to the point of exhaustion is more likely to need ventilating than one with abnormal tensions who is alert, oriented, talking in full sentences, and not working excessively.

*Likely normal blood gas tensions for that patient*—Some patients with severe chronic lung disease will lead surprisingly normal lives with blood gas tensions which would suggest the need for ventilation in someone previously fit.

*Likely course of disease*—If imminent improvement is likely ventilation can be deferred, although such patients need close observation and frequent blood gas analysis.

*Adequacy of circulation*—A patient with established or threatened circulatory failure as well as respiratory failure should be ventilated early in order to gain control of at least one major determinant of tissue oxygen delivery.

### **Circulatory support**

Shock represents a failure of tissue perfusion. As such, it is primarily a failure of blood flow and not blood pressure. Nevertheless, an adequate arterial pressure is essential for perfusion of major organs and glomerular filtration, particularly in elderly or hypertensive patients, and for sustaining flow through any areas of critical narrowing in the coronary and cerebral vessels. A normal blood pressure does not exclude shock since pressure may be maintained at the expense of flow by vasoconstriction. Conversely, a high cardiac output (for example, in sepsis) does not preclude regional hypoperfusion associated with systemic vasodilatation, hypotension, and maldistribution

### **Signs suggestive of failing tissue perfusion**

- Tachycardia
- Confusion or diminished conscious level
- Poor peripheral perfusion (cool, cyanosed extremities, poor capillary refill, poor peripheral pulses)
- Poor urine output (<0.5 ml/kg/h)
- Metabolic acidosis
- Increased blood lactate concentration

### **Neurological considerations in referral to intensive care**

- Airway obstruction
- Absent gag or cough reflex
- Measurement of intracranial pressure and cerebral perfusion pressure
- Raised intracranial pressure requiring treatment
- Prolonged or recurrent seizures which are resistant to conventional anticonvulsants
- Hypoxaemia
- Hypercapnia or hypocapnia

### **Neurological support**

Neurological failure may occur after head injury, poisoning, cerebral vascular accident, infections of the nervous system (meningitis or encephalitis), cardiac arrest, or as a feature of metabolic encephalopathy (such as liver failure). The sequelae of neurological impairment may lead to the patient requiring intensive care. For instance, loss of consciousness may lead to obstruction of airways, loss of protective airway reflexes, and disordered ventilation that requires intubation or tracheostomy and mechanical ventilation.

Neurological disease may also cause prolonged or recurrent seizures or a rise in intracranial pressure. Patients who need potent anaesthetic drugs such as thiopentone or propofol to treat seizures that are resistant to conventional anticonvulsants, or monitoring of intracranial pressure and cerebral perfusion pressure must be referred to a high dependency or intensive care unit.

Patients with neuromuscular disease (for example, Guillain-Barré syndrome, myasthenia gravis) may require admission to intensive care for intubation or ventilation because of respiratory failure, loss of airway reflexes, or aspiration.

### **Renal support**

**Renal failure** is a common complication of acute illness or trauma and the need for renal replacement therapy (haemofiltration, haemodialysis, or their variants) may be a factor when considering referral to intensive or high dependency care. The need for renal replacement therapy is determined by assessment of urine volume, fluid balance, renal concentrating power (for example, urine:plasma osmolality ratio and urinary sodium concentration), acid-base balance, and the rate of rise of plasma urea, creatinine, and potassium concentrations. In ill patients hourly recording of urine output on the ward may give an early indication of a developing renal problem; prompt treatment, including aggressive circulatory resuscitation, may prevent this from progressing to established renal failure.

#### **Indications for considering renal replacement therapy**

- Oliguria (<0.5ml/kg/h)
- Life threatening hyperkalaemia (>6 mmol/l) resistant to drug treatment
- Rising plasma concentrations of urea or creatinine, or both
- Severe metabolic acidosis
- Symptoms related to uraemia (for example, pericarditis, encephalop

#### **The components of invasive monitoring in the ICU are as follows:**

- Invasive blood pressure (BP) monitoring
- Transesophageal Doppler (TED)
- Measurement of central venous pressure (CVP)
- Pulmonary artery catheterization
- Arterial blood gas (ABG) analysis
- Intracranial pressure (ICP) measurement
- Intra-abdominal pressure (IAP) measurement



## **TETANUS**

Tetanus is a rare and often fatal neurological disease that causes increased tone and muscle spasms due to the bacteria.

### **Systemic Involvement**

#### **Cardiovascular:**

- Hyperkinetic circulation is a result of elevated basal sympathetic and muscle activity.<sup>[8]</sup>
- Tachycardia with hypertension
- Increased stroke volume
- Thromboembolus

#### **Respiratory:**

- Muscular spasms: chest wall, diaphragm, abdomen, pharyngeal, and laryngeal tract (can lead to an obstructed airway which can be life threatening)
- Atelectasis, pneumonia, and aspiration: inability to cough secondary to muscular rigidity, spasms, and being sedated.
- Hyperventilation: common because of fear and autonomic disturbances.

#### **Renal:**

Renal involvement is only found in severe cases of tetanus.

- Dehydration
- Urinary stasis and infection
- Renal failure
- Altered renal blood flow secondary to catecholamine surges

#### **Gasto-intestinal:**

- Gastric stasis
- Ileus
- Diarrhea
- Hemorrhage
- Weight loss: As a result of the inability to swallow

#### **Neuromuscular:**

- Autonomic dysfunction: Leads to excess sympathetic activation and catecholamine levels

#### **Musculoskeletal**

- Muscle rigidity and spasms
- Lock-jaw (trismus)
- Risus sardonicus
- Opisthotonos (reactions of the head)
- Tendon avulsions: a result of spasms
- Fractures: a result of spasms

#### **Physiotherapy Management**

Cardiopulmonary physical therapy can be used to help in the prevention of respiratory complications. Breathing exercises, suctioning and airway clearance techniques are useful.

Physical therapy can also be used to help with muscle rigidity and spasms.

#### **PULMONARY OEDEMA**

Pulmonary edema is an abnormal buildup of fluid in the air sacs of the lungs, which leads to shortness of breath.

Pulmonary edema is often caused by congestive heart failure. When the heart is not able to pump blood to the body efficiently, it can back up into the veins that take blood through the lungs to the left side of the heart.

As the pressure in these blood vessels increases, fluid is pushed into the air spaces (alveoli) in the lungs. This fluid reduces normal oxygen movement through the lungs. This and the increased pressure can lead to shortness of breath.

Congestive heart failure that leads to pulmonary edema may be caused by:

- Heart attack, or any disease of the heart that weakens or stiffens the heart muscle (cardiomyopathy)
- Leaking or narrowed heart valves (mitral or aortic valves)
- Sudden, severe high blood pressure (hypertension)

Pulmonary edema may also be caused by:

- Certain medications
- High altitude exposure
- Kidney failure
- Narrowed arteries that bring blood to the kidneys
- Lung damaged caused by poisonous gas or severe infection
- Major injury

### **Symptoms**

Symptoms of pulmonary edema may include:

- Coughing up blood or bloody froth

- Difficulty breathing when lying down (orthopnea) -- Feeling of "air hunger" or "drowning" (wakes from sleep and causes to sit up and try to catch breath, it's called "paroxysmal nocturnal dyspnea")
- Grunting, gurgling, or wheezing sounds with breathing
- Inability to speak in full sentences because of shortness of breath

Other symptoms may include:

- Anxiety or restlessness
- Decrease in level of alertness (consciousness)
- Leg swelling
- Pale skin
- Sweating (excessive)

### **Signs and tests**

- Abnormal heart sounds
- Crackles in lungs, called rales
- Increased heart rate (tachycardia)
- Pale or blue skin color (pallor or cyanosis)
- Rapid breathing (tachypnea)

Possible tests include:

- Blood chemistries
- Blood oxygen levels (oximetry or arterial blood gases)
- Chest x-ray
- Complete blood count (CBC)

- Echocardiogram(ultrasound of the heart) to see if there are problems with the heart muscle (such as weakness, thickness, failure to relax properly, leaky or narrow heart valves, or fluid surrounding the heart)
- Electrocardiogram (ECG) to look for signs of a heart attack or problems with the heart rhythm

Physiotherapy also play an important role in the management of acute pulmonary oedema. Assist the patient through a variety of ventilatory exercises which greatly lessen the symptoms of the patient.

**CPAP** is one technique used by physiotherapists to lessen the symptoms experienced by the patient.

**CPAP** assists patients in three ways:

1. Decrease Work of Breathing

-by preventing alveoli collapse

2. Enhanced Gas Exchange

-improves VE/Q mismatch & lung compliance

3. Improved Hemodynamics

-decrease venous return to LV and decreases pre-load (1)

**Active Cycle of Breathing Exercises**

-reduces anxiety and increases O<sub>2</sub>

**Incentive Spirometry**

-increases lung vol and encourages basal expansion to reduce the work of breathing (7)

**Respiratory Muscle Exercises**

-decrease WOB

### **PEP device**

-maintains lung volume and facilitate clearance

### **Oxygen Therapy**

-improves perfusion and VE/Q mismatch (4)

### **Patient positioning (sitting up)**

-decreases WOB, reduce venous return & increases FRC (2)

### **Gravity Assisted Positioning with Percussions**

-enhances airway clearance

### **Suction & Manual Hyperinflation**

-used in intubated patient

### **MULTIPLE ORGAN FAILURE**

Multiple organ failure (MOF) is the commonest cause of death in the intensive care unit (ICU).

Multiple organ dysfunction syndrome is the presence of altered organ function in acutely ill patients such that homeostasis cannot be maintained without intervention. It usually involves two or more organ systems

The condition usually results from infection, injury (accident, surgery), hypoperfusion and hypermetabolism. The primary cause triggers an uncontrolled inflammatory response. Sepsis is the most common cause in operative and non-operative patients. Sepsis may result in septic shock. In the absence of infection, a sepsis-like disorder is termed systemic inflammatory response syndrome (SIRS).

The European Society of Intensive Care organized a consensus meeting to create the "Sepsis-Related Organ Failure Assessment (SOFA)" score to describe and quantitate the degree of organ

dysfunction in six organ systems. Using similar physiologic variables the Multiple Organ Dysfunction Score was developed.

Four clinical phases have been suggested:

- **Stage 1** the patient has increased volume requirements and mild respiratory alkalosis which is accompanied by oliguria, hyperglycemia and increased insulin requirements.
- **Stage 2** the patient is tachypneic, hypocapnic and hypoxemic; develops moderate liver dysfunction and possible hematologic abnormalities.
- **Stage 3** the patient develops shock with azotemia and acid-base disturbances; has significant coagulation abnormalities.
- **Stage 4** the patient is vasopressor dependent and oliguric or anuric; subsequently develops ischemic colitis and lactic acidosis.

### **Neurological conditions**

Various cardiorespiratory complications may be encountered in severely head injured patients due to inability to maintain airway, hypoventilation and direct injuries to the chest. Pneumonia can occur in about 60% of the patients with severe head injuries due to prolonged intubations, mechanical ventilation and inability to maintain airway

### **SMOKE INHALATION**

**Smoke inhalation** is the primary cause of death for victims of indoor fires.

Smoke inhalation injury refers to injury due to inhalation or exposure to hot gaseous products of combustion. This can cause serious respiratory complications

Deaths are the result of smoke inhalation injuries, including burns to the respiratory system. The hot smoke injures or kills by a combination of thermal damage, poisoning and pulmonary irritation and swelling, caused by carbon monoxide, cyanide and other combustion products.

Symptoms range from coughing and vomiting to nausea, sleepiness and confusion. Burns to the nose, mouth and face; singed nostril hairs; and difficulty breathing / carbonaceous sputum (burned saliva) are also signs of smoke inhalation injury. Approximately one third of patients admitted to burns units have pulmonary injury from hot smoke inhalation<sup>1</sup>

Advanced medical care may be necessary to save the life of the patient, including mechanical ventilation, even if the person is conscious and alert.

reatment consists of humidified oxygen, bronchodilators, suction, endotracheal tube and chest physiotherapy. There is no role for routine treatment of smoke inhalation with either antibiotics or steroids. Treatment depends on the severity of the smoke inhalation.

#### **Nebulized heparin and acetylcysteine**

Inhalation therapy with nebulized heparin and acetylcysteine is usually started and continued for five to seven days during the hospital stay.

#### **Oxygen therapy**

Carbon monoxide (CO) is always presumed to be a complication in smoke inhalation. The initial approach to presumed CO poisoning involves administering supplemental oxygen at a fraction of inspired oxygen (FiO<sub>2</sub>) of 100 percent and then the use of hyperbaric oxygen (HBO) therapy is evaluated by physicians

### **INTENSIVE CARE UNIT-ACQUIRED WEAKNESS**

Intensive care unit-acquired weakness (ICUAW) is observed in a substantial proportion of patients receiving MV for more than 1 week in the ICU. The etiology includes deconditioning and disuse atrophy due to prolonged bed rest and immobility, and critical illness polyneuropathy and/or myopathy, known as critical illness neuromyopathy. Other risk factors for ICUAW



include the systemic inflammatory response syndrome, sepsis, and multiple organ dysfunction syndrome; hyperglycemia; and medications, such as use of corticosteroids and neuromuscular blocking agents. As a consequence, recommendations to avoid these risk factors have been suggested.

## **PHYSIOTHERAPY MANAGEMENT IN ICU**

Implementation of an early mobilization program is feasible in most ICUs and provides benefits if started no later than 1 or 2 days after MV initiation. Such programs must be delivered after cardiorespiratory and neurological stabilization. This approach, together with specific muscle training, can improve functional outcomes and cognitive and respiratory conditions

### **Rotational Therapy**

Continuous rotational therapy uses special beds to turn patients along the longitudinal axis up to 60° on each side, with preset degree and speed of rotation. It has been hypothesized that this modality can reduce the risk of sequential airway closure and pulmonary atelectasis, resulting in reduction of the incidence rate of lower respiratory tract infection and pneumonia, and the duration of endotracheal intubation and length of hospital stay

### **Early Mobilization**

Early mobilization can be performed also in unconscious or sedated patients. Protocols include semirecumbent positioning with the bed head positioned at 45°, frequent changes in postures, daily sessions of joint passive movement, and passive bed cycling and electrical stimulation

### **Management of Airway Secretions**

Mechanically ventilated patients in the ICU may suffer from retained secretions due to many causes. The mucociliary system may be disturbed by endotracheal intubation, with increased infection susceptibility and mucus volume and tenacity.

Furthermore, immobilized patients may suffer from atelectasis, impaired cough mechanism, and related inability to expel secretions. Associated expiratory muscle weakness decreases cough strength; in addition, fluid restriction contributes to secretion retention. Helping airway clearance in patients under MV includes different techniques.

**Postural drainage.** Postural drainage traditionally includes gravity-assisted positions, deep breathing exercises, chest clapping, shaking or vibration, and incentivized cough to move airway secretions toward the upper airways

**Intrapulmonary percussive ventilation.** Intrapulmonary percussive ventilation (IPV) is a high-frequency ventilation modality that can be superimposed on spontaneous breathing. Intrapulmonary percussive ventilation may reduce respiratory muscle load and help to move airway secretions. This tool creates a percussive effect in the airways, thus enhancing mucus clearance through direct high-frequency oscillatory ventilation able to help the alveolar recruitment. Positive effects from this technique have been shown in patients with respiratory distress, neuromuscular diseases, and pulmonary atelectasis

**Positive expiratory pressure.** Positive expiratory pressure (PEP), first introduced in the 1970s, consists of a one-way valve through a mask or a mouthpiece connected to one or more small-exit orifices and adjustable expiratory resistor to enhance and promote secretion removal by stenting airways, increasing intrathoracic pressure, or increasing functional residual capacity.

A new modality to deliver a low level PEP at the mouth during spontaneous breathing is called temporary PEP, which has been recently proposed to treat patients with chronic mucus hypersecretion. This modality produces a 1 cm H<sub>2</sub>O increase in airway pressure along the respiratory cycle until immediately before the end of expiration

The level of applied pressure is several times lower than that (5 to 15 cm H<sub>2</sub>O) commonly used and considered effective with other PEP and/or oscillatory-PEP devices. Preliminary results show that an expiratory pressure less than or equal to 1 cm H<sub>2</sub>O applied for a fraction of the expiratory phase may improve the distribution of alveolar ventilation and prevent mechanical stress injury, which is expected to occur in the bronchial tree or lung parenchyma at a higher pressure.

**Manual hyperinflation.** Manual hyperinflation (MH) is commonly applied in patients under MV. It may stimulate cough and move the airway secretions toward the larger airways, from where they can be easily suctioned. Manual hyperinflation can prevent airway plugging and pulmonary collapse, and improve oxygenation and lung compliance. This technique is widely used, though the practice varies across different ICUs. The possible physiological side effects of delivered air volume, flow rates, and airway pressure must be carefully considered—especially in patients under MV. When performed by experienced and trained physiotherapists in stable, critically ill patients, MH is associated with short-term and probably nonrelevant side effects like reduction in cardiac output, alterations in heart rate, and increased central venous pressure. Nevertheless, other studies failed to show MH benefits in intubated and mechanically ventilated patients.

**Insufflation-exsufflation.** Methods to improve cough are important in critically ill patients because cough effectiveness is a determinant in weaning success and patient outcomes. Cough assist such as a mechanical insufflator/exsufflator clears secretions by gradually applying a positive pressure to the airway then rapidly shifts to negative pressure, producing a high expiratory flow.