97. Tachyarrhythmias in ICU
Nikhileshwar Prasad Verma

98. POCUS in Medical ICU
Ayush Bansal, Jai Bhagwan, Himanshu Sikri

99. Disease-specific Ventilation—Strategies and Evidences
RT Shriram Ganesh, S Tamilselvan, Rengarajan, Namavigayam, Sowmiya, Mukesh, Pragadeesh G

100. Prone Ventilation for Adults with ARDS
A Bhagwati

101. Respiratory Emergencies: Golden Hour Rules in the First Hour
T Geetha, Ramesh Duraisamy

102. Basics of Mechanical Ventilation—Must Know for All Physicians
Rudrajit Paul

103. ARDS in Viral Infections
Gagan Gunjan, Mohd Saif Khan, Krishna Kumar, DP Singh

104. ABG Analysis: Physicians’ Perspective
Ravindra Kumar Das

105. Hyperkalemia in ICU
SV Ramana Murty, TVSP Murthy, DJK Chakravarthy
Abstract
Tachyarrhythmia in ICU is very common. Their recognition and management are of paramount importance.

Introduction
It is not uncommon to see patients admitted in ICU to develop various tachyarrhythmias (HR >100 bpm) as they are on multiple drugs and in a state of stress. Tachyarrhythmias are more common than bradyarrhythmias and are well tolerated by some patients. But some may become unstable and manifestations in these patients could be:
- Dyspnea
- Hypotension
- Ischemic chest pain
- Altered sensorium

Tachyarrhythmias have profound effect on morbidity and mortality of the patient. Tachycardias result from enhanced automaticity, triggered activity or reentry and could be supraventricular or ventricular. There are various types of tachycardias, but the most common forms with whom an intensivist is confronted with are:
- Sinus tachycardia
- Supraventricular tachycardia (AT, AVNRT, AVRT)
- Atrial fibrillation
- Atrial flutter
- Multifocal atrial tachycardia (MAT)
- Ventricular tachycardia (monomorphic and polymorphic)

Assessment of Patient
An intensivist should first evaluate whether the patient is stable or not. Unstable patients must be urgently cardioverted. One should look for precipitating factors like electrolyte imbalance, hypovolemia, abnormal temperature, sepsis, abnormalities in gases, and drugs. When QRS complex is less than 0.12 second, it is termed as narrow complex tachycardia (NCT) and when QRS is more than 0.12 second, it is termed as wide complex tachycardia (WCT). The rhythm may be regular or irregular. A 12-lead ECG is mandatory to evaluate the arrhythmia. Response to vagal maneuvers as carotid sinus massage (CSM), valsalva maneuver and response to adenosine help in differentiating supraventricular tachycardia from ventricular tachycardia. Adenosine (6 mg) is given rapidly and 12 mg may be repeated after 2 minutes as second dose. Adenosine may cause severe bronchospasm and sometimes atrial fibrillation (AF). After giving adenosine it should rapidly be flushed with normal saline. Bedside, echo will tell us about the status of heart.

NCT with Regular Rhythm
Sinus Tachycardia
It is the most common tachycardia seen in the ICU and the HR is between 100–180 bpm. It rarely requires
treatment. In ICU, most of the time it is an appropriate response to underlying causes like fever, hypovolemia, pain, and hypotension. There could be iatrogenic causes too. Over enthusiastic treatment may sometimes backfire. Ischemia induced sinus tachycardia may be treated with beta blockers. It is difficult to differentiate from atrial tachycardia. The morphology of non sinus p wave of AT is the clue, which is different from sinus p wave in differentiating it from AT. The maximum sinus HR for a person is 220 – Age.

Atrial Tachycardia

Three continuous non-sinus APBs originating from the same focus make AT. If arrhythmia lasts for more than 30 second, it is called sustained. Mostly in ICU AT is non-sustained. The atrial rates range 100–260 bpm. Unstable patients are treated with synchronized DC shock. In stable patients AV nodal blocking agents are used to control the ventricular rate.

Atrioventricular Nodal Reentrant Tachycardia (AVNRT)

More than 60% of SVT is due to AVNRT. Here AV node has a dual conducting pathway. Since atria and ventricles are stimulated simultaneously p wave is usually submerged in QRS or sometimes appear just after QRS. It may also be seen as pseudo R’ in V1. The HR is usually between 140–220 bpm. Patients complain of palpitation and lightheadedness. Majority of the patients tolerate it well and arrhythmia can be terminated by vagal maneuvers or AV nodal blocking agents. Long-term preventive therapy is catheter ablation or use of AV nodal blocking agents.

Atrioventricular Tachycardia (AVRT)

There is an accessory pathway between atria and ventricles. If the impulse travels through AV node from atria to ventricles and go to atria from ventricles through accessory pathway (orthodromic conduction) the QRS complex is narrow and the tachycardia would be NCT. But if antegrade conduction is through accessory pathway and retrograde through AV node (antidromic conduction) tachycardia would be WCT. The activation of ventricle and atria take place one after another so RP interval is long and p wave is seen on ST-T wave. Orthodromic AVRT responds to vagal maneuvers and AV nodal blocking agents. But these drugs are harmful in antidromic AVRT. In such cases amiodarone is preferred drug. It is rare for a patient to become unstable with SVT but if this happens so DC shock with 25–50 J is sufficient.

Atrial Flutter

Saw tooth flutter waves are best seen in II, III, AVF at the rate of 300 bpm (250–350 bpm) with 2:1 block in the setting of structural heart disease in most of the patients. Sometimes 4:1 block is also seen. Rarely atrial flutter with 1:1 AV conduction may occur in high catecholamine states as sepsis, shock, and with the use of flecainide in the presence of fast conducting accessory pathway and becomes an emergency requiring urgent cardioversion. 50J is sufficient with the success rate of above 90%. AV nodal blocking agents and digoxin (especially in heart failure) can control ventricular rate. Flecainide is good for its prevention but should be used in combination of AV nodal blocking agent. Anticoagulant is started if atrial flutter is of more than 48 hours. Atrial flutter should always be thought if ventricular rate is 150 bpm and regular.

NCT with Irregular Rhythm

Irregular NCT is seen in AF, MAT, and sinus tachycardia with frequent atrial premature beats.

Atrial Fibrillation (Fig. 1)

The chaotic atrial activities with the rates ranging 400–600 bpm are seen as coarse or fine fibrillatory waves

![Fig. 1: Atrial fibrillation](image-url)
without discernible p waves on surface ECG. It is very common in ICU. Advancing age, valvular heart diseases, thyrotoxicosis, large left atrium, sleep apnea, and chronic kidney disease are some of the important predisposing factors for AF. It is pertinent to identify and treat the cause.

Management has three aspects:

- Rhythm control,
- Rate control, and
- Anticoagulation.

Urgent cardioversion is done if patient is hypotensive, there is ongoing ischemia, acute pulmonary edema, or underlying preexcitation with rapid ventricular rate. Synchronized shock of 200J is appropriate. Repeated shocks may be needed with increased energy up to 400J as success rate of cardioversion is less than 30% in ICU. If DC shock fails 300 mg amiodarone iv in 1 hour may be given and repeated as needed.

If onset of AF is within 48 hours, cardioversion is a common practice even if patient is not on anticoagulant, at risk of stroke and there is no mitral stenosis or large left atrium.

If duration of AF is not known or more than 48 hours, patient is anticoagulated for 3 weeks before cardioversion and for 4 weeks after cardioversion. The other approach is to rule out thrombus by transesophageal echocardiography start anticoagulation cardiovert the patient and continue anticoagulation for 4 weeks. In stable patient pharmacological cardioversion can be done by ibutilide, flecainide, and amiodarone.

Rate control can be achieved by AV nodal blocking agents IV diltiazem at a rate 5–15 mg/hr maintains the ventricular rate below 100 bpm in 90% cases during 24 hours infusion. Digoxin is seldom used as it takes longer time to slow the rate.

If AF persists for more than 48 hours iv heparin should be started but one should keep in mind that chances of bleeding increases with anticoagulants in sepsis without any benefit.

Wolf-Parkinson-White (WPW) Syndrome

WPW syndrome is triad of short PR interval, slurring of QRS (delta wave) and broadened QRS. Orthodromic conduction will produce NCT while antidromic conduction produces WCT. AF is very common in WPW and is life threatening as it causes rapid ventricular rate usually more than 200 bpm. QRS has a changing morphology in width and height unlike AF with fixed BBB where ventricular rate is not that fast and QRS morphology is always the same. It is the accessory path that propagates the impulses from atria to ventricles. Use of AV nodal blocking agents will be dangerous in this situation. Ibutilide, flecainide, and amiodarone are safe drugs in this condition. For unstable patient DC shock of 150–200 J is required.

Multifocal Atrial Tachycardia (MAT)

Three or more non-sinus consecutive p waves of different contours beating at a rate 100 or more per minute constitute multifocal atrial tachycardia. The P-P & P-R intervals are variable and ventricular rate is irregular. At times it is difficult to differentiate it from AF. In more than 50% cases of MAT the underlying cause is chronic obstructive lung disease. Coronary artery disease and valvular heart disease are frequently present along with chronic obstructive lung disease. Other precipitating factors may be low potassium and magnesium. Treatment should be directed toward underlying cause. DC shock is ineffective due to multiple atrial foci. AV nodal blocking agents are used to control the ventricular rate. Empirically magnesium is used with high success rate. MgSO₄ + 50 ccNS (2 gm) iv in 15 minutes is given followed by 6 gm MgSO₄ + 500 ccNS iv in 6 hours.

Wide Complex Tachycardia

When QRS duration is more than 0.12 second it is called wide complex. About 85% of wide complex tachycardia (WCT) are due to VT. About 10% are due to supraventricular tachycardia with aberrancy (SVT-A). The aberrancy could be fixed bundle branch block, functional or rate dependent block, or accessory pathway. The other causes of WCT could be pacemaker induced tachycardia where rate will never go beyond the upper limit fixed for the pacemaker, artifact where rate is very very fast but patient is hemodynamically stable and QRS complex march with regularity. Hyperkalemia where QRS is very very wide but rate is not so fast usually below 120 bpm and drugs like tricyclic antidepressants, CLASS IA and IC antiarrhythmic drugs may cause WCT.

Ventricular Tachycardia (Fig. 2)

When there are three or more consecutive ventricular premature complexes present at a rate more than 100/ min it is called ventricular tachycardia (VT) and if it persists for 30 or more seconds it is termed sustained.
VT could be monomorphic where morphology of all ventricular complexes are same and polymorphic where there is continuous change in morphology of ventricular complexes.

There are certain features though not always but if present suggest VT while differentiating it from SVT. They are:

- H/o heart disease or previous MI.
- QRS width >0.16 second.
- AV dissociation (ventricular rate more than atrial).
- Presence of capture and fusion beats.
- RBBB with left axis and LBBB with right axis, extreme north west axis.
- Presence of concordance.
- Presence of Brugarda’s sign (onset of QRS to nadir of S is >100 msec) and Josephson’s sign (notch in S wave).
- All precordial leads showing QS pattern.
- Presence of RBBB like morphology (dominant R wave in V1-2 & R/S<1 in V6).
- Presence of LBBB like morphology (initial R >0.04 second RS interval>0.07second in V1-2 & any Q in V6).

**Monomorphic VT**

This may occur in structurally normal or diseased heart. If it is non-sustained and heart is not diseased no treatment is needed. AV nodal blocking agents are used to prevent its recurrence. But patients who have structural heart disease (coronary heart disease, dilated cardiomyopathy, valvular heart disease) need further evaluation. Hemodynamically unstable patient with monomorphic sustained VT DC shock of 100–200 J is given and increment may be done up to 360 J, followed by lidocaine 1–4 mg/min iv infusion. In stable patient either shock or drugs can be given. In patients with preserved LV function procainamide, amiodarone, lidocaine, and sotalol may be given but preferred drug is amiodarone. In patients with compromised LV amiodarone or lidocaine may be used. 0.5–1 mg/kg lidocaine is given iv bolus followed by iv infusion at 1–4 mg/min lidocaine is more effective if VT is due to ischemia. One should never forget to correct hypokalemia and hypomagnesemia as they are precipitating factors. When VT is unstable, recurrent, and nonresponsive to cardioversion iv amiodarone may be given.

**Polymorphic VT**

Polymorphic VT may occur with normal or prolonged QT interval. PMVT with normal QT is mostly due to ischemia and almost always causes significant hemodynamic instability. Therefore, patient should be shocked.
Medication that predisposes to ischemia should be withdrawn. Electrolytes should be corrected. Beta-blockers may be used to prevent recurrence. PMVT with normal QT is treated in the same way as monomorphic VT.

The prolongation of QT (QTc >460 msec) may be:
- Congenital or
- Acquired due to drugs (antibiotics, antihistamines, antiarrhythmic drugs, antidepressants, etc.), electrolyte abnormalities (hypokalemia, hypocalcemia), etc.

**Torsades De Pointes**

It is a form of polymorphic VT with QT prolongation seen in 12 lead baselines ECG. There is beat to beat variation and appears to be twisting around the isoelectric line of ECG. This is treated with 1–2 gm of MgSO4 iv over 15–20 minute iv atropine and isoproterenol or overdrive pacing are also used for acquired form as they increase the heart rate and thus shorten the QT interval. Correction of electrolytes and removal of offending factor is a key to treatment. Congenital QTc prolongation is adrenergic mediated; hence beta-blockers are used.

**Pulseless VT/Ventricular Fibrillation**

Most of defibrillators in use these days are biphasic. Along with CPR and vasopressor (epinephrine) asynchronous shock of 120–200 J is given. Energy of shock is increased in stepwise manner.

**Antiarrhythmic Drugs Commonly Used in ICU**

- Adenosine 6 mg iv fast followed by rapid saline flush if no response repeat 12 mg iv fast.
- Esmolo 500 mcg/kg iv bolus then infusion at 50 mcg/kg/min. If needed increment of dose 25 mcg/kg/min every 5 minute maximum up to 200 mcg/kg/min.
- Diltiazem 0.25 mg/kg iv in 2 minutes bolus if needed second bolus dose after 15 minutes at 0.35 mg/kg and maintain the infusion at 5–15 mg/min.
- Metoprolol 2.5–5 mg iv in 2 minutes. If needed may be repeated every 5–10 minutes up to three doses.
- Amiodarone 150 mg iv bolus over 10 minutes. Repeat if needed, then 1 mg/min for 6 hours followed by 0.5 mg/min for 18 hours. Total dose in 24 hours should not exceed 2.2 gm.
- Lidocaine 0.5–1 mg/kg bolus instant followed by 1–4 mg/min.
- MgSO4 1–2 gm iv in 20 minutes. A continuous infusion at the rate of 2–4 mg/min may also be administered after bolus, if needed.

**DC Shock and Pacing**

A critically ill patient has many precipitants of tachyarrhythmia such as infection, high catecholamine state, involvement of multiorgan so response to shock is a challenge. Moreover, critically ill patient does not tolerate shock well. Taking care of precipitants is necessary to get a successful response of shock. Serial DC shocks are not appropriate for self limiting recurrent tachyarrhythmias.

Overdrive pacing is done by pacing the heart at higher rate than its native rate. It is done to treat SVT, atrial flutter, VT, who have either failed to response or recurrent. Rhythm of sinus tachycardia, AF, and VF cannot be controlled by this.

**Conclusion**

Although some patients are admitted in ICU because of tachyarrhythmias majority develop them in ICU because of multiple illness and treatment. The adverse effect of tachyarrhythmia is well established and treating them in ICU is difficult also. So a very judicious approach is needed to address this problem.

**Suggested Readings**

Abstract

Point-of-care ultrasound (POCUS) is a useful imaging modality in the era of modern evidence-based medicine. This article will summarize the historical development, basic physics, advantages of POCUS, and its applications in the medical intensive care unit (ICU). Based on improved accuracy and reduced time in narrowing diagnosis, it positively influences patient outcomes. Structured training programs with hands-on practice can equip the doctors acquire the necessary knowledge and skill in the field of POCUS.

Introduction

Point-of-care ultrasound (POCUS) literally refers to the point-of-care ultrasound, which is usually performed by non-radiologists in close proximity of the patient. The scope and goals are limited to specific clinical questions, which help in narrowing the diagnosis and guiding clinical therapy. It is an extremely useful imaging modality particularly for the critically ill patients. The advantages of POCUS of being quick, accurate, reproducible, radiation free, and available bedside for serial monitoring make it stand-out from other imaging modalities. In the western world, POCUS has become an integral part of the emergency medicine department and critical care. Moreover, Royal College of Emergency Medicine has made POCUS a mandatory part of the curriculum of emergency medicine training.¹ Though the use of ultrasound by emergency medicine physician and by an intensivist requires the same instrument and skill, still there are important differences in their application. POCUS is primarily used by an intensivist in the medical ICU for focusing on heart, lung, pleura, procedural guidance, and for vascular access. The intensivist does serial examinations to monitor the response to treatment, which aids in patient’s management. On the other hand, the emergency medicine physician does a single extended evaluation of the whole body including heart, lungs, abdomen, testes, obstetrics, musculoskeletal, and eyes for making a provisional diagnosis.²

Advantage of POCUS over Routine Ultrasound

POCUS in ICU is performed by the intensivist bedside who has full knowledge of the patient’s issues and there is rapid implementation of these results to patient’s management. There is no time lag in decision-making, provided the intensivist has the appropriate skill at image acquisition and interpretation. On the other hand, routine ultrasound is done by a radiologist or a cardiologist who are not in constant touch with the patient and there is always a time lag due to logistic issues. Moreover, serial monitoring of the patient is difficult with routine ultrasound.²

History

The use of POCUS is not new and can be traced back to 1990s when the first paper supporting the use of POCUS
was published by The American College of Emergency Medicine. It was followed by a series of publications highlighting POCUS to be one of the important tools in dealing critically ill patients. The use of ultrasound for central venous access was made compulsory by the National Institute for Clinical Excellence in 2002. Today, there are many fellowship programs in POCUS giving training to the intensivists. In 2016, the Society for Acute Medicine published the first POCUS curriculum for the physicians in the United Kingdom. Though there is no national certification course for POCUS in India, there is still a lot of potential in increasing the use of this modality. Further, the residents have an easy access of doing bedside POCUS with the advent of small portable ultrasound machines. We, therefore, need to overcome the inertia of using POCUS routinely for patient management.

**Basic Physics**

A clear mental picture of the three-dimensional anatomy and basic understanding of the physics of ultrasound are the prerequisites for optimal image acquisition and interpretation. The ultrasound transducers send ultrasound waves (1–15 MHz) through their piezoelectric crystals and receive reflected waves. A two-dimensional image is formed on the screen which can be optimized by using the correct transducer and adjusting its position. Higher frequency transducers produce high resolution images of superficial structures, while lower frequency transducers produce low resolution images of deeper structures. There are basically three types of transducers used in POCUS—linear, curvilinear, and phased array (Table 1).

Denser media reflect most of the sonographic waves, generating a white image. On the other hand, rarer media transmit most of the waves and reflect back very few waves, generating an echogenic black image. Thus, fluid appears black, soft tissue (liver) appears gray, fibrous tissue appears white without a shadow, and bones or stones appear white with a shadow (Figs. 1A and B). Air is very hyperechoic and thus prevents visualization of deeper structures.

**Components of POCUS**

POCUS usually involves evaluation of the heart, lung, pleura, lower limb Doppler, screening abdomen and for venous access. Still, the most important aspect in medical ICU is the cardiorespiratory evaluation to identify the cause of dyspnea or hypoxia.

**Heart**

A quick goal directed echocardiography is the hallmark tool for evaluation of the cause of shock or acute onset dyspnea. A combination of basic two-dimensional echocardiography combined with color flow mapping and Doppler imaging of the heart and great vessels gives a basic idea of the structure and function of the heart. All the views including parasternal long and short axis views, apical views, and subcostal views must be imaged wherever possible, for addressing specific abnormalities. The intensivist can promptly look for an immediately life threatening cause of hemodynamic collapse like major valve failure, massive pulmonary embolism, cardiac tamponade, acute cor pulmonale, or regional wall motion abnormality, which needs prompt treatment. With the addition of speckle tracking, one is able to negate the subjective error in assessing regional wall motion abnormality. Serial monitoring of inferior vena cava and its collapsibility by the subcostal view helps in assessment of the fluid status of the patient.

**Thoracic Ultrasonography (Lung and Pleura)**

Evaluation of the cause of dyspnea can never be complete without having a look at the lungs and pleura. It is not uncommon to miss the diagnosis of a small posteriorly located consolidation in an obese patient on clinical examination. Even chest X-ray in a critically ill ventilated patient does not give accurate information as there can be motion artifacts, rotation, coexisting fluid overload, and the views are usually anteroposterior. However, ultrasound has a similar accuracy as compared to computed tomography and can appreciate pathologies like consolidation, pleural effusion, pneumothorax, diaphragmatic dysfunction, and one should spend time to look at both sides of the chest. Thoracic ultrasound has a sensitivity and specificity of more than 90% for diagnosing these pathologies.

Some of the important findings in a thoracic POCUS are:

- **Seashore sign** (Fig. 2A) refers to the normal aeration pattern, which consists of lung sliding with A-lines.
### TABLE 1  Types of transducers used in POCUS

<table>
<thead>
<tr>
<th>Transducer type</th>
<th>Linear</th>
<th>Curvilinear (convex)</th>
<th>Phased array</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Frequency range (MHz)</strong></td>
<td>5–10</td>
<td>2–5</td>
<td>1–5</td>
</tr>
<tr>
<td><strong>Imaging depth (cm)</strong></td>
<td>9</td>
<td>30</td>
<td>35</td>
</tr>
<tr>
<td><strong>Applications</strong></td>
<td>Arteries/veins • Eyes • Skin/soft tissue • Pleura • Procedures – central venous catheterization, lumbar puncture</td>
<td>Abdominal viscera like • Liver • Gall bladder • Kidney • Urinary bladder</td>
<td>Heart • Inferior vena cava • Great vessels • Pleura • Lungs</td>
</tr>
</tbody>
</table>
Figs. 1A and B: (A) Appearance of fluid, soft tissue, fibrous tissue, and solids on ultrasound screen. (B) Propagation of ultrasound waves through various media.

Figs. 2A to C: (A) Seashore sign demonstrating lung sliding with A-lines in M-mode ultrasound. (B) Two-dimensional image of vertically oriented B-lines or lung rockets in a thoracic ultrasound. (C) Two-dimensional image showing a clear pleural effusion with lung consolidation (echogenicity similar to that of liver).
Lung sliding refers to the opposition of visceral and parietal pleura. Absence of lung sliding indicates presence of air or fluid between the visceral and parietal pleura. A-lines constitute the horizontal, hyperechoic reverberation artifacts created by repetitive reflection of ultrasound waves between the pleural line and transducer.

- **B-lines or lung rockets (Fig. 2B)** are the vertically oriented discrete hyperechoic lines, which extend to the sonographic window edge. They represent widened fluid-filled interlobular septa and indicate pulmonary edema, pneumonia, acute lung injury, or fibrosis, depending upon their distribution and characteristics.

- Consolidations appear with an echogenicity similar to that of liver, and thus called hepatization. Infective consolidations need to be differentiated from basal lung atelectasis (Fig. 2C).

- **Pleural effusion** as small as 5 mL can be picked up. Anechoic, clear fluid indicates transudate while hyperechoic, debris-filled fluid indicates exudate. Also, pleural effusion can be differentiated from pleural thickening.

### Abdomen

The importance of abdominal assessment cannot be undermined especially in cases of acute abdomen. Appreciation of intra-abdominal fluid, any septic foci, bladder distension, hydronephrosis can be done by the intensivist rapidly without waiting for the radiologist. Free fluid is most commonly detected in the perisplenic space and in the Morrison’s pouch in the supine position. Ultrasound can be used therapeutically in cases of drainage of abscesses.

### Lower Limbs

Patients in ICU remain bed-ridden for prolonged periods and require regular screening of their lower limb veins. Early identification of deep vein thrombosis and subsequent prevention of pulmonary embolism is one of the important aspects in managing complications of such patients.

### Ocular Ultrasound

The most important aspect of ocular ultrasound in medical ICU is to assess the intracranial pressure by the measurement of optic nerve sheath diameter. A cut-off value of 5 mm has 100% sensitivity of raised intracranial pressure which is comparable to a non-contrast CT. Serial monitoring of optic nerve sheath diameter helps in optimizing therapy. Pathologies such as globe rupture, traumatic detachments, lens dislocation, and vitreous hemorrhage can also be easily picked up but these are uncommon issues encountered in medical ICU.

### Ultrasound for Procedural Guidance

A variety of procedures can be performed in the ICU for routine management of patients for both diagnostic and therapeutic purposes. Ultrasound greatly increases the success rate and reduces the complication rate in cases of thoracocentesis, paracentesis, abscess drainage, regional anesthesia, lumbar puncture, and central venous catheter insertion. Upper extremity deep vein thrombosis secondary to central venous catheters can also be readily picked up.

### Miscellaneous

Several other uses of POCUS include looking for ectopic pregnancy, testicular torsion, epididymo-orchitis, tendon injury, etc., but these are uncommon issues encountered in the medical ICU.

### Conclusion

The enthusiasm and interest in using POCUS is rising across the globe owing to its speed and accuracy in diagnosis, in an acutely unwell patient. It acts as an aide to the traditional clinical and radiological techniques in reducing the morbidity and mortality of the patients. However, there is still a huge gap in expanding the utility of this modality due to lack of training programs and trained supervisors. Therefore, this rapid and reliable diagnostic tool should be included in routine medical curriculum to make it an integral part of the patient care.

### References

CHAPTER 99

Disease-specific Ventilation—Strategies and Evidences

RT Shriram Ganesh, S Tamil Selvan, Rengarajan, Namasivayam, Sowmiya, Mukesh, Pragadeesh G

Abstract

- Normal respiration is NEGATIVE (Table 1) Pressure, and Ventilator driven is POSITIVE Pressure Ventilation.
- Disease-specific ventilation strategies and evidences is done in respiratory failure as evidenced by clinical symptomatology and ABG.
- Whatever mode selected, the treating physician should maintain SYNCHRONY between the ventilator and the patient. Then the mode NIV or Invasive and finally to TROUBLE SHOOT. based on the specific diseases and the ventilator alarms.

Introduction

- The topic disease specific ventilation deals in brief about the basic steps in ventilation and then disease-specific strategies in patients presenting with respiratory failure
- This could be achieved by mechanical ventilation (MV)
- There is wide variety of MV available for use in modern pulmonary care
- The principles in MV are:
  - Disease-specific ventilation strategies
  - Lung protection strategy by applying safe ventilation practices

Respiratory Failure: Four Types

- The respiratory system helps to maintain adequate oxygenation.
- Respiratory failure results in either inadequate oxygenation or CO₂ removal or both.
- It’s one of the most common medical emergencies that warrant ICU admission.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Negative and positive pressure ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal respiration/negative pressure ventilation</strong></td>
<td>Ventilator generated/positive pressure ventilation</td>
</tr>
<tr>
<td>Inspiration done by respiratory muscles by sucking of air into the lungs, increasing the negative intrapleural pressure (-6 to -8 cm H₂O)</td>
<td>According to disease status, ventilator generates pressure in the airways to drive air into the lungs, pushing the chest wall outward</td>
</tr>
</tbody>
</table>

Type 1/Hypoxemic Respiratory Failure (Table 2):
- Most common type
- PaO₂ <60 mm Hg (Table 3)
- PaCO₂ <45 mm Hg
- Patient presents as tachypneic, hypoxic, and hypocapnic

Type 2/Hypercapneic Respiratory Failure:
- Seen in airway diseases (COPD), neuromuscular diseases, and chest wall abnormalities
- Patient presents with low-respiratory rate, hypoxia, hypercapnia, and acidic
Type 3/Perioperative Respiratory Failure: Due to atelectasis following GA

Type 4/Shock Related Respiratory Failure: Due to hypoperfusion

- Presentation of RF
  - Acute Respiratory Failure: Develops before compensation
  - Chronic Respiratory Failure: Compensated to buffer respiratory acidosis

Diagnosis
Always by clinical, supported by ABG, PFT, pulse oximetry, CXR, ECHO.

Clinical Features
- Type 1:
  - Mild to moderate: Hypoxia, tachypnea, tachycardia, diaphoresis
  - Severe hypoxia: Bradycardia, vasodilation, hypotension, infarction, arrhythmia, cardiac failure
- Type 2:
  - CNS disturbances: Depression, lethargy, headache, seizure
  - ABG: To confirm and differentiate the types of RF
  - Pulse oximetry: Assessing O2 status
  - CXR and ECHO: To differentiate between cardiogenic and noncardiogenic pulmonary edema

Treatment
- To correct arterial hypoxemia and to maintain target SpO₂ >89–90% and PO₂ >55–60 mm Hg
- It is done by:
  - Oxygen supplementation usually HFNO/NIV/Invasive ventilation
- HFNO: Supplies as much as 40 L/min to maintain constant O₂ flow
- NIV: Nowadays useful in acute respiratory failure as an initial tool especially in COPD exacerbation, weaning after extubation in COPD, in immunosuppressed patients in ARF, cardiogenic pulmonary edema
- NIV avoids the need for intubation, nosocomial infection
- A successful NIV depends upon properly fit interface & optimal ventilator settings
- Full face masks for ARF, nasal mask for COPD
- Noninvasive ventilation within 1–4 hours of MV, the response is observed and assessed for failed NIV trial
- In acute severe asthma, if NIV fails, requires endotracheal intubation
- Finally NIV is beneficial in COPD and cardiogenic pulmonary edema

Noninvasive positive pressure ventilation: Used in COPD, ARDS, cardiogenic pulmonary edema
Noninvasive negative pressure ventilation: Used in neuromuscular disease, central hypoventilation and chest wall abnormality

TABLE 2 Causes of RF (respiratory failure)

<table>
<thead>
<tr>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1: Acute pulmonary edema</td>
<td>Type 1: ILD-sarcoidosis</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>Idiopathic pulmonary fibrosis</td>
</tr>
<tr>
<td>ARDS</td>
<td>Pulmonary artery hypertension</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>Chronic heart failure</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>Cyanotic congenital heart disease</td>
</tr>
<tr>
<td>Type 2: Acute exacerbation of COPD</td>
<td>Type 2: Obesity hypoventilation syndrome</td>
</tr>
<tr>
<td>GBS</td>
<td>Neuromuscular disorders</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>Kyphoscoliosis</td>
</tr>
<tr>
<td>Poisoning head injury/raised ICP</td>
<td>Ruptured esophagus</td>
</tr>
<tr>
<td>Type 3: Upper abdominal emergencies</td>
<td>Type 4: Cardiogenic shock</td>
</tr>
<tr>
<td>Obesity/ascites</td>
<td>Septic shock</td>
</tr>
<tr>
<td>Prolonged GA</td>
<td>Hypovolemic shock</td>
</tr>
<tr>
<td>Peri-operative</td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
</tr>
</tbody>
</table>

TABLE 3 Respiratory parameters

<table>
<thead>
<tr>
<th></th>
<th>Normal people</th>
<th>Respiratory failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂ (Kpa)</td>
<td>10.5–13.5</td>
<td>&lt;7</td>
</tr>
<tr>
<td>PaCO₂ (Kpa)</td>
<td>4.7–6</td>
<td>&gt;7</td>
</tr>
<tr>
<td>RR (/min)</td>
<td>10–25</td>
<td>&gt;30</td>
</tr>
<tr>
<td>Tidal volume (mL/kg)</td>
<td>5–8</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Vital capacity (mL/kg)</td>
<td>30–70</td>
<td>&lt;15</td>
</tr>
</tbody>
</table>
Other uses of NIV:
- NIV weaning or postextubation
- Facilitation technique: For early extubation
- Rescue or curative technique: For avoiding reintubation who fails extubation

**Invasive Mechanical Ventilation**
- Done in failed O2 therapy and NIV trial[^22] and for airway protection:
  - Always start with a volume ACM[^9] (Table 4) (assist control mode of ventilation), then if the patient improves, shift to Pressure Support Ventilation[^23] (PSV)
  - To avoid ventilator induced lung injury (VILI), a low-tidal volume and high PEEP is always set to reduce mortality in ARDS—ARDS NETWORK STUDY[^24]

**Mechanical Ventilation[^26]**
(Basics and Modes)
Mainly indicated in:
- Depressed respiratory drive: Sedatives, stroke, head injury
- Excessive respiratory workload: ALI, ARDS, pulmonary embolism
- Ventilatory pump failure: Flail chest, tension pneumothorax

**Four Phases of Breath[^28]**
- Inspiration
- Change from inspiration to expiration
- Expiration
- Change from expiration to inspiration:
  - Triggering of a breath:
    - It describes the phase variables that changes from expiration to inspiration
    - Trigger is what initiates a breath
  - Time triggered—inspiration begins when a certain time has lapsed
  - Pressure triggered—ventilator senses the inspiratory effort reflected by drop in airway pressure and initiates inspiration
  - Volume triggered—when the baseline flow variable drops below the fixed flow (5–20 L/min), the machine initiates inspiration:
    - Limiting a breath:
      - It’s the set flow in pressure beyond which the variable never goes in inspiration or expiration.
    - Cycling of a breath:
      - Time cycled ventilation—common cycling mechanism. Inspiratory flow is terminated once the preselected time interval has lapsed after the start of inspiration and expiratory phase is started.
      - Pressure cycled ventilation—useful in SIMV/IRV modes. When a preselected air pressure has been reached, expiratory valve opens and ends the inspiration.
      - Volume cycled ventilation with pressure limiting valve—When a preset volume is delivered, inspiration is terminated, delivers constant VT.
      - Flow cycled ventilation—Here a preset flow ends the inspiratory flow useful in PSV.

**Patient Ventilator Interactions**
A patient interacts[^31] with the ventilator based on the ventilatory drive, when inspiration starts and ventilator

**TABLE 4** Parameters and goals of invasive[^25] MV in different respiratory diseases

<table>
<thead>
<tr>
<th>Prototype</th>
<th>Restricted lung-ARDS</th>
<th>Obstructed lung-asthma</th>
<th>Neuromuscular diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mode</td>
<td>V-ACMV</td>
<td>V-ACMV</td>
<td>V-ACMV</td>
</tr>
<tr>
<td>VT [per KG]</td>
<td>4–6 mL/kg</td>
<td>4–6 mL/kg</td>
<td>6–8 mL/kg</td>
</tr>
<tr>
<td>RR [per minute]</td>
<td>18–35</td>
<td>8–12</td>
<td>14–18</td>
</tr>
<tr>
<td>PEEP</td>
<td>High PEEP based on FiO2</td>
<td>5–8 cm H2O</td>
<td>Up to 5 cm H2O</td>
</tr>
<tr>
<td>I:E ratio</td>
<td>1:1 or 1:2</td>
<td>1:3–1:6</td>
<td>1:2–1:3</td>
</tr>
<tr>
<td>Plateau pressure</td>
<td>&lt;30 cmh 20</td>
<td>&lt;30 cm H2O</td>
<td></td>
</tr>
<tr>
<td>PaO2</td>
<td>55–60 mm Hg</td>
<td>55–60 mm Hg</td>
<td>60–80 mm Hg</td>
</tr>
<tr>
<td>PH</td>
<td>7.2–7.4</td>
<td>7.2–7.4</td>
<td>-</td>
</tr>
</tbody>
</table>
requirement, to satisfy metabolic demands whereas the ventilator interacts with the patient based on the inspiratory trigger, delivery of gas, and cycling (Fig. 1).

- **CMV:**
  - Patient cannot trigger the ventilator, but PCV always ensures Pplat < 30–35 cm H₂O—used as a lung protective strategy in ARDS.³²

- **ASSIST CONTROL MODE (ACM) (Fig. 2):³³**
  - Commonly used. It delivers controlled breath as well as assists patient triggered breath (Fig. 3); hence the patient continues to do inspiratory work even on ventilator.³⁴

- **SIMV:**
  - Ideal mode for weaning providing partial ventilatory support.

- **PSV:²³**
  - Allows breathing, providing full support making the patient fully comfortable used for weaning. Ultimately, SIMV and PS—an ideal choice of ventilatory support facilitating quick weaning as well.

No mode is superior one over the other. The initial settings:—FiO₂ 0.5–1, tidal volume—8–12 mL/kg, RR—8–12/min, inspiratory flow rate of 40–60 L/min.

---

**Fig. 1:** Graphical representation of breath²⁹

**Fig. 2:** Basics of mechanical ventilation²⁷
Management of Patients with Severe Airflow Obstruction

- **Acute Severe Asthma**
  AC mode or SIMV mode—settings:
  - Small VT: 5–7 mL/kg
  - RR: 12–15/min
  - Peak flow: 60 L/min
  - FiO₂: 0.5
  - To prevent Barotrauma auto PEEP should be <10 cm H₂O

- **COPD**
  SIMV or AC mode—settings:
  - VT: 5–7 mL/kg
  - RR: 24–28/min
  - FiO₂: 0.4
  - They have expiratory flow limitation, and hence auto PEEP, which is balanced by setting extrinsic PEEP, which reduces patient’s work of triggering

**ARDS—Advances in Diagnosis and Treatment**

- Berlin definition of ARDS

Based on the severity of hypoxemia:
- **Mild**: 200–300 mm Hg with PEEP >5 cm H₂O
- **Moderate**: 100–200 mm Hg with PEEP >5 cm H₂O
- **Severe**: <100 mm Hg with PEEP >5 cm H₂O
- (Berlin definition has greater predictive validity for mortality)
- Timing of onset of symptoms: within 1 week of clinical insult.
- Chest X ray: bilateral opacities.
- Origin of edema: not fully explained by heart failure.

**Early spontaneous breathing** in mild and late ARDS—by ACM, PSV, or APRV similar to PCM pressure control mode.

**Advantages of spontaneous breathing (Fig. 4):**
- to reduce ventilation induced diaphragmatic dysfunction (VIDD)
- to improve tidal ventilation, O₂ exchange, delivery

**THE STRATEGY OF VENTILATION IS:**
- **Minimal distension strategy**—low VT <6 mL/kg
- **Increased recruitment strategy**—open lung approach—high PEEP >10 cm H₂O
- **Goal of plateau pressure** <30 cm H₂O to avoid shunt and improve oxygenation

---

**Fig. 3:** Machine and patient triggering graph
This lung protective strategy ends with permissive hypercapnia (treated with a higher respiratory rate of 24–28 breaths/min) and benefited by decreased mortality.

Prone Positioning in ARDS
Earlier and prolonged prone positioning for more than 16 hours in severe ARDS improved 28 day, 90 day mortality.

Other therapies:
- Inhaled Nitric Oxide—used in refractory ARDS
- Surfactant recombinant protein C
- Steroids—methyl prednisolone 1–2 mg/kg in early severe ARDS
- ECMO—useful in severe refractory ARDS

In any settings

Manipulations to Increase Oxygenation
Increase:
- FiO₂
- PEEP
- Ti

Settings to Improve Ventilation
Increase:
- RR <35/min
- VT up to 10 mL/kg
- PIP (peak inspiratory pressure)

Complications of mechanical ventilation:
- Always whatever mode is set, see to that LPS is adapted—low VT is set even in normal and diseased lungs to avoid VILI

### Trouble Shooting

**Clinically**
- Breath stacking/dyssynchrony:
  - Increase inspiratory flow rate
  - Sedation
- Hypotension:
  - Minimize PEEP/sedation
- Altered sensorium:
  - Especially in NIV adjust IPAP, EPAP, and increase backup RR

**Based on ABG**
- Low PaO₂:
  - Adjust flow rate or FiO₂
  - Increase:
    - EPAP/PEEP
    - Ti
- High PaCO₂:
  - Increase:
    - IPAP/PS
    - Backup RR
- Low PaCO₂:
  - Decrease:
    - IPAP
    - Backup RR

### CARDS—COVID-19 with ARDS
- Management of COVID-19—Respiratory distress—Vasocenteric problem
- CARDS 2 types:
  - L-CARDS [low lung elastance—high compliance]
  - H-CARDS [high lung elastance—low compliance]
    - Typical ARDS lung

### Ventilatory Strategies for CARDS
- L-CARDS [mild cases]
  Before intubation:
  - NIV:
    - HFNO
    - CPAP
    - BIPAP
    - Awake prone positioning
After intubation:
- Lower PEEP [<10 cm H₂O]
- Liberal VT [7–9 mL/kg]
- Prone positioning

### H-CARDS
- Treated as typical ARDS lung, requiring invasive ventilation:
  - Higher PEEP [<15 cm H₂O]
  - Lower VT [5–7 mL/kg]
  - Prone positioning

## Abbreviations

ACM, Assist Controlled Mode
ALI, Acute Lung Injury
ARDS, Acute Respiratory Distress Syndrome
CARDS, COVID-19 with ARDS Patients
HFNO, High Flow Nasal Cannula Oxygenation
IRV, Inverse Ratio Ventilation
MV, Mechanical Ventilation
NIV, Noninvasive Ventilation
PIP, Peak Inspiratory Pressure
PEEP, Positive End Expiratory Pressure
RF, Respiratory Failure
SIMV, Synchronized Intermittent Mandatory Ventilation
VCM, Volume Control Mode
VT, Tidal Volume

## Conclusion

Ultimately one should know the basics of MV and then to decide NIV/INVASIVE VENTILATION according to specific diseases like COPD, ALI/ARDS, CCF, etc. and also see to that you always follow LPS (lung protective strategy) in ventilation.

## References

Prone Ventilation for Adults with ARDS

A Bhagwati

Abstract
Prone ventilation for adult respiratory distress syndrome (ARDS) patients is a resurrected concept, initially proposed in 1970s and widely used in 1990s for treatment of ARDS. However, it was not popular due to some studies and meta analysis. But the practice was reintroduced in 2015 Sepsis guidelines and currently is very popular particularly in patients with lung involvement due to COVID-19. The principle is to recruit the posterior lungs which are affected to improve oxygenation and FIO2/PaO2 ratio. This article briefly introduces the concept of prone position ventilation and also highlights the problems that are associated with it.

Introduction
Adult respiratory distress syndrome (ARDS) is a condition in which the lungs suffer severe widespread injury, interfering with their ability for gas exchange. Increasing pulmonary edema and alveolar collapse create a physiologic dead space, where no gas exchange can take place in the pulmonary capillaries, causing ventilation-perfusion mismatch. ARDS is a life-threatening respiratory condition characterized by hypoxemia, and stiff lungs ventilation. ARDS represents a stereotypic response to many different inciting insults and evolves through a number of different phases: Exudative phase, Fibrotic phase, and Proliferative phase. ARDS is a syndrome with multiple risk factors that trigger the acute onset of respiratory insufficiency and respiratory failure.

Causes of ARDS
Direct Lung Injury
- Aspiration of gastric contents or other causes of chemical pneumonitis
- Lung contusion, penetrating injury of the thorax affecting the lung tissues, fat emboli
- Near drowning
- Inhalation injury, pulmonary vasculitis
- Pulmonary edema due to repercussion due to transplant of lungs.

Indirect Lung Injury
- Sepsis
- Pancreatitis
- Shock with hypoperfusion
- Multiple blood transfusions (TRALI)
- Major trauma
- Burns, drug overdose
- Non-cardiogenic pulmonary edema.

Management of ARDS (Prone Ventilation)
In the intensive care unit (ICU), different types of patients requiring mechanical ventilation (MV) are admitted. It is challenging for a clinician when patient develops ARDS. Several strategies are employed, the currently
practiced and quite popular is prone positioning of a patient with ARDS on MV. Several studies and practical experience in recent times have shown improvement of oxygenation when a patient is placed in prone position. Prone ventilation was practiced in the 1995–1997 by me in Mumbai at Bhatia Hospital along with another center in Mumbai with excellent results. Unfortunately studies and meta-analysis did not evoke scientific justification for use of this protocol in MV, and fell out of use with other modalities.

However, in 2015, Surviving Sepsis guidelines revived the interest and recommended use of prone ventilation in ARDS as a part of recruitment maneuvers and thereafter it is now an accepted modality in management of ARDS patients, including finding its way in management of COVID pneumonia and COVID-induced ARDS.

Pathophysiology
In supine position, the positive pressure from the ventilator raises the anterior chest wall, but hardly has any effect on the posterior chest wall. When patient is placed in prone position, the posterior chest wall, which is less compliant, is elevated leading to more diaphragmatic excursion, which leads to increased recruitment of the posterior lung. This causes improved overall aeration of the alveoli of the posterior lungs thereby improving the lung compliance. This causes improvement of ventilation-perfusion ratio, which is compromised in ARDS patients.

Flowchart 1: Strategies in prone ventilation
Prone Position Protocol

Best Practice in Implementing Prone Position for Patients with ARDS
- Take written consent for prone ventilation, explaining to relatives in details advantages and risks involved.
- Assess the hemodynamic status and oxygenation to determine the eligibility of the patient.
- Administer sedation and muscle paralytic drugs, with sedation holiday.
- Provide care of eyes and tape eyelids, if indicated.
- Maintain adequate oral hygiene. Secure the patients tongue with oral airway to prevent tongue bite. Oral suction as and when required.
- Secure endotrachael tube and monitor to ensure that extubation or ventilatory disconnection does not occur.
- Maintain proper skin care and place hydrocolloid dressings on bony prominence, chin, and forehead.
- Position the patient’s face away from the ventilator to prevent tube kinking. Reposition the patient’s head hourly to prevent skin breakdown.
- Place ECG electrodes on the patient’s posterior chest.
- Empty the drainage bags.
- Appoint dedicated inter-professional team to take care (airways/ventilator tubing/line) of patient in prone position.
- Monitor effectively the hemodynamic status, blood gases within 1 hour of pruning the patient and then 4 hourly intervals.

Contraindications
- Burns, open wounds on face or ventral body surface
- Spinal instability
- Facial and pelvic fractures
- Life-threatening circulatory shock
- Increased intracranial pressure
- Pregnancy
- Abdominal trauma or surgery
- Acute bleeding
- Shock

Complications
- Facial and periorbital edema
- Pressure sores
- Accidental loss or displacement of ET, thoracic or abdominal drains, and central venous catheters
- Airway obstruction
- Hypotension
- Arrhythmias
- Vomiting

Summary of Strategies in Prone Ventilation

See Flowchart 1.

Conclusion

Prone positioning was first proposed in the 1970s as a method to improve gas exchange in ARDS. However, translation of this application in clinical practice is challenging as discussed. Prone position ventilation has been effective in management of moderate to severe ARDS patients, begun early in the course of the disease within 36 hours of the onset, for a period ranging 12–18 hours per session. However, prone positioning is not without harm and problems as discussed earlier.

Suggested Readings
Respiratory Emergencies are most common and sometimes life threatening. The Spectrum may range from Mild Respiratory Distress to Respiratory Arrest. The “Golden Hour” management is crucial to save the life. We divide the topic into Immediate, Emergent, and Urgent management with time frame. “Every Breath is like a little rebirth” as quoted by Cristen Rodgers. So, the Emergency Team should act promptly using clues from the Brief History, Quick Physical Examination with Essential Investigations.

**Introduction**
“A-B-C” is the unofficial mantra of any emergency care, which describes airway, breathing, and circulation. The first two letters in this mantra relate to the patient’s respiratory status. Compromise of the airway and respiration is the leading cause of death in many conditions in emergency setting. Respiratory emergencies are generally identified by “Acute dyspnea” as a presentation. Dyspnea can result from respiratory, cardiac, neurological, traumatic causes, etc. (Table 1).

**Spectrum of Respiratory Emergencies**
In emergency department (ED), the patients with respiratory emergencies present with a spectrum, that can range from mild respiratory distress to respiratory failure and finally respiratory arrest, which leads to death (Fig. 1). Respiratory emergencies are generally identified by “Acute dyspnea” as a presentation. Dyspnea can result from respiratory, cardiac, neurological, traumatic causes, etc. (Table 1).

**Management of Respiratory Emergencies**
For effective management of respiratory emergencies, we must have the following basic principles:

- Rapid assessment of respiratory distress to arrive at the correct working diagnosis.
- With the working diagnosis, aggressive targeted treatment without delay.
- Continuous and frequent reassessment to ensure response to the appropriate treatment.

With the above principles, we can divide the Golden Hour management of respiratory emergencies into the following three parts:

- Immediate management—to be done in 0–5 minutes (Table 2)
- Emergency management—to be done in 5–15 minutes (Table 3)
- Urgent management—to be done in 15–60 minutes (Table 4)

**Indications of Noninvasive Positive Pressure Ventilation**
The following clinical and laboratory clues give an idea for indication of noninvasive positive pressure ventilation (NIPPV) in respiratory emergencies:

- Respiratory distress (dyspnea, tachypnea, and the use of accessory muscles of respiration)
- Acidemia (pH <7.35)
Critical Care Medicine

Fig. 1: Spectrum of respiratory emergencies

TABLE 1 Common causes of respiratory emergencies

<table>
<thead>
<tr>
<th>Respiratory:</th>
<th>Cardiac:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper airway obstruction</td>
<td>Pulmonary edema</td>
</tr>
<tr>
<td>Croup</td>
<td>Arrhythmias</td>
</tr>
<tr>
<td>Supraglottitis</td>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>Epiglottitis</td>
<td>Neurological:</td>
</tr>
<tr>
<td>Neck abscess</td>
<td>Guillain-Barré syndrome</td>
</tr>
<tr>
<td>Ludwig’s angina</td>
<td>Myasthenia gravis</td>
</tr>
<tr>
<td>Angioedema</td>
<td>Snake bite envenomation</td>
</tr>
<tr>
<td>Subglottic stenosis</td>
<td>Metabolic:</td>
</tr>
<tr>
<td>Tumor</td>
<td>Diabetic Keto Acidosis</td>
</tr>
<tr>
<td>Foreign body</td>
<td>Psychogenic:</td>
</tr>
<tr>
<td>Other:</td>
<td>Anxiety</td>
</tr>
<tr>
<td>A/E Asthma &amp; COPD</td>
<td>Panic attacks</td>
</tr>
<tr>
<td>Obstructive Sleep Apnoea</td>
<td>Others:</td>
</tr>
<tr>
<td>Tracheomalacia</td>
<td>Anaphylaxis causing Angioedema</td>
</tr>
<tr>
<td>Tension Pneumothorax</td>
<td>SVC obstruction</td>
</tr>
<tr>
<td>Trauma causing Hemotorax</td>
<td>OPC poisoning</td>
</tr>
<tr>
<td>Acute Pneumonitis (Bacterial/Viral)</td>
<td></td>
</tr>
<tr>
<td>Hypersensitivity pneumonitis</td>
<td></td>
</tr>
<tr>
<td>Drug induced</td>
<td></td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td></td>
</tr>
</tbody>
</table>

- Hypercapnia (PaCO₂ >45 mm Hg)
- Hypoxemia (PaO₂/FiO₂ <200)

Most frequently used types of NIPPV are CPAP and BiPAP:
- CPAP—in acute pulmonary edema, CCF, pneumonia, acute respiratory distress syndrome (ARDS), and chest wall trauma.

### Indications for Converting Noninvasive Ventilation to Invasive Ventilation

- Inability to tolerate NIV
- Excessive secretions

- BiPAP—in COPD with type 2 respiratory failure, neuromuscular disease, and sleep apnea.
TABLE 2 Immediate management (0–5 min)\textsuperscript{5}

<table>
<thead>
<tr>
<th>History</th>
<th>Examination</th>
<th>Investigation</th>
<th>Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AMPLE H/o:</strong> (Allergies, Medications, Past H/o, Pregnancy, Last meal, Events before the presentation) Also ask for: Lip/tongue/throat /Neck swelling Stridor/Grunting Voice change Trauma Chest pain Emesis</td>
<td>Ability to speak, aspiration, oropharyngeal edema, trauma Breath sounds, Hypoxia, posturing/work of breathing/tracheal shift Check for shock (BP, extremities, weak pulse, sweating, tachycardia) Disorientation/ Delirium, signs of trauma</td>
<td><strong>SpO\textsubscript{2}</strong> Capillary glucose</td>
<td><strong>General Measures:</strong> Pt Positioning IV-line placement Airway repositioning—Jaw thrust, chin lift Suction O\textsubscript{2}/bag ventilation Urinary catheterization <strong>Specific Measurements:</strong> Choking maneuvers Airway in case of altered mental status NIPPV—for conscious pt with Pulmonary edema, OSA, COPD Needle decompression for pneumothorax Nebulized Salbutamol—for severe asthma Adrenaline—for anaphylaxis Call for help</td>
</tr>
</tbody>
</table>

Look for red flag signs (Box 1)

TABLE 3 Emergency management (5–15 min)\textsuperscript{5}

<table>
<thead>
<tr>
<th>History</th>
<th>Examination</th>
<th>Investigation</th>
<th>Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset/progression Similar episodes/Asthma/ COPD Fever/cough Chest pain Unilateral leg swelling Renal/Cardiac disease Allergy H/O</td>
<td><strong>Vital signs</strong> <strong>Focused Respiratory Exam</strong> Look: Pt posture Diaphoresis, cyanosis, pallor, ability to complete a sentence, single breath count, neck veins Listen: Breath sounds/Wheeze/crepts Heart sounds/murmur/rub Feel: Pulses, pedal oedema, peripheries Trauma Red flag signs</td>
<td><strong>ECG</strong> Urine Acetone USG—for effusions, pneumothorax ABG/VBG</td>
<td><strong>General Measures + Specific Measures:</strong> Intubation/NIPPV Chest tube—for Pneumo/ hemothorax Percardiocentesis—for tamponade Salbutamol/adrenaline—As per indication Fluid challenge for hypotension Antibiotics for infection Atropine for OPC poisoning ASV—for snake bite Diuretics for pulmonary edema/CCF NTG—for accelerated Hypertension/Pulmonary edema Call for help</td>
</tr>
</tbody>
</table>

- Ill fitting mask/leak due to facial abnormalities
- Progressive fatigue or impending respiratory arrest
- Hypoxia despite adequate positive airway pressure and high FiO\textsubscript{2}
- Progressive and persistent hypercapnia (>1 hour)
- High airway pressure requirement (>20 cm H\textsubscript{2}O)

Drugs Commonly Used in Respiratory Emergencies

- Nebulized Salbutamol—Severe asthma
- Adrenaline—Anaphylaxis/Asthma (Nebulization)
- Steroids—Asthma/COPD/COVID-19 pneumonia
TABLE 4  Urgent management (15–60 min)\(^5\)

<table>
<thead>
<tr>
<th>History</th>
<th>Examination</th>
<th>Investigation</th>
<th>Intervention</th>
</tr>
</thead>
</table>
| Detailed H/o including past H/o, Family H/o                            | Vital Signs                                                                 | CBC                                 | General Measures + Specific interventions: Depending upon on the diagnosis arrived:
| CCF: orthopnea, PND, leg swelling                                       | Complete Systemic examination                                               | RFT, Electrolytes                   | Needle thoracentesis in cases of pleural effusion                         |
| H/o asthma/ COPD—medication H/o, past ICU admissions for the same      | Repeat clinical examination if there are abnormal findings in lab investigations | Ketones                             | Steroids/NTG/Salbutamol/Bronchodilators/Inotropes depending on the condition |
| Cardiac ailments/ HT/Diabetes/ Dyslipidemia, etc.                       |                                                                             | RBC                                 | NIV/Intubation: for Neuromuscular/envenomation                              |
| Chest pain: Anginal/pleuritic                                          |                                                                             | ECG                                 | Call for help                                                               |
| DVT: Leg pain and swelling, H/o immobilization                         |                                                                             | Chest X-ray                         |                                                                             |
| HIV/TB status                                                          |                                                                             | HIV                                 |                                                                             |
| Smoking H/o                                                            |                                                                             | Echo                                |                                                                             |
|                                                                          |                                                                             | Cardiac enzymes                     |                                                                             |
|                                                                          |                                                                             | CT Chest                            |                                                                             |
|                                                                          |                                                                             | D Dimer                             |                                                                             |
|                                                                          |                                                                             | ABG/VBG                             |                                                                             |

General Measures + Specific interventions:
- Needle thoracentesis in cases of pleural effusion
- Steroids/NTG/Salbutamol/Bronchodilators/Inotropes depending on the condition
- NIV/Intubation: for Neuromuscular/envenomation
- Call for help

BOX 1  “Red flag signs” of acute dyspnea\(^2\)

- Altered mental status
- Shallow breathing
- Tachypnea (RR >40/min)
- Hypotension
- Hypoxia
- Cyanosis
- Stridor
- Tracheal deviation
- Absent breath sounds (Unilateral/Bilateral)
- Unstable arrhythmia

- Antibiotics—Infective exacerbations of COPD/asthma, Sepsis, Aspirations (Foreign body/Secretions), GBS, Trauma
- Diuretics—Pulmonary edema, CCF
- Nitroglycerin—Pulmonary edema
- Atropine—Organophosphorus poisoning
- Anti-snake venom—Cobra/Krait bite

Venous Blood Gases in ED

Arterial blood gas analysis is what is generally expected to be done in emergency setting. But venous blood gas (VBG) analysis is equally valuable, and has now become routine in the initial work up of cases presenting to the ED. There are many advantages of VBG analysis: arterial puncture which causes bleeding and pain can be avoided. VBG analysis can be done with the blood drawn for other lab analysis. VBG is easy to process by modern analyzers. A venous blood gives accurate measurement of glucose, K\(^+\), lactate, HCO\(_3^-\), hemoglobin, and COHb. In addition, a normal venous pCO\(_2\) will exclude hypercarbia. Venous lactate level gives early diagnosis of sepsis. Treatment response can be established by serial VBG in terms of lactate and K\(^+\).\(^8\)

Ultrasonogram in Respiratory Emergencies\(^5\)

- The Bedside Lung Ultrasonography in Emergency (BLUJE) protocol plays a major role in ED to diagnose various causes of respiratory failure.
- Focused Assessment with Sonography in Trauma (FAST) to diagnose pneumothorax and hemothorax.
- Bilateral increased B-line density in pulmonary edema.
- Unilateral B-lines in pneumonia with edema, or foreign body with lung collapse.
- Cardiac USG: to r/o cardiac failure, pericardial effusion and tamponade.
- Right heart strain indicates pulmonary embolism.
- A normal lung USG may be seen in bronchospasm, pulmonary embolism, anemia, or acidosis.

Conclusion

Respiratory emergencies are very common in the ED and it is imperative that all the team members be prepared to stabilize patient’s oxygenation and ventilation. Using clues from the history and quick physical examination, an emergency physician can guide the work up and intervene in the golden hour. Early use of bedside testing, including USG may limit unnecessary tests and save time in determining the best treatment course.
References


Mechanical Ventilation (MV) has come to the limelight over the last 1 year due to the Covid-19 pandemic. Ventilation has saved numerous lives all over the world. Clinicians have been forced to familiarize themselves with this technology.

In India, the government has supplied ventilators to many health facilities as an emergency. After the pandemic subsides, these machines will remain and there will be a huge demand for their use in different diseases. Thus, this is a good time for internists to learn about the basics of mechanical ventilation. This article describes the basic functions of the ventilator. This is not meant to be an exhaustive text on the topic. Rather, this article is meant to be a ready reckoner for the busy clinician.

Essential topics like FiO₂, I:E ratio, PEEP, and plateau pressure have been discussed. Some tips for tracheal intubation have also been touched upon. Towards the end, the overall ventilator strategies for common diseases like COPD, DPLD, and, of course, COVID-19 have been discussed. The importance of arterial blood gas report in a ventilated patient has also been discussed. The various output parameters in a modern ventilator have been mentioned. So, overall, this article is meant to help the clinician during a busy shift.
cardiopulmonary stability, then MV may be started as benefit of doubt. It must be remembered that MV shows the greatest advantage if used early. So, it is better to err on the side of overzealous ventilation than to wait longer when the underlying pathology has already gone into a tailspin.

- **Acute or chronic respiratory failure**
  - Central respiratory depression
    - Poisoning
    - Stroke
    - Post-neurosurgery
  - Peripheral respiratory compromise
    - Guillain-Barré (GB) syndrome
    - Myasthenia
    - Severe myositis
    - High cervical myelopathy
  - Lung pathology
    - Parenchymal pathology
    - Severe pneumonia
  - Acute respiratory distress syndrome (ARDS)
  - Severe pulmonary edema
    - Airway pathology
  - Acute chronic obstructive pulmonary disease (COPD)/Asthma
  - Airway edema in toxic gas inhalation
    - Vascular pathology
  - Pulmonary embolism

- **Severe shock**

- **Prophylactic ventilation**
  - Patient with recurrent vomiting when pharyngeal reflexes are deemed inadequate
  - After poisoning, especially if patient is anticipated to deteriorate
  - Severe cerebral edema, if hyperventilation is planned in the future
  - Severe sepsis with hyperventilation, when respiratory muscle fatigue is anticipated
  - Recurrent refractory seizures, when aspiration is a possibility or where the use of anesthetic drug is planned

This list is not exhaustive and other pertinent indications may be there. A study in the USA found that COPD, heart failure, and pneumonia were the main indications for the use of MV. Another international study found that in the intensive care, acute respiratory failure was the commonest indication of MV, followed by acute chronic COPD and coma. Acute respiratory failure can occur in a variety of indications from opiate poisoning to head injury.

### Some Notes on Intubation

Endotracheal intubation is one skill which every physician must acquire. This is a lifesaving procedure even if ventilator is not available. For example, in a patient with poisoning and laryngeal edema, immediate intubation can prevent airway closure. The steps of intubation will not be discussed here. They may be found in great details in anesthesia texts. But only certain relevant points will be mentioned:

- There must be a breathing circuit (Bain’s circuit) in the CCU. This will help in preoxygenation of the patient before intubation. Also, if during intubation, there is sudden hypoxia, the procedure may be temporarily abandoned and breathing circuit used to improve oxygenation.
- The physician must ensure that the laryngoscope is in working condition. In personal experience of the author, the battery of the laryngoscope is not checked properly in many Indian hospitals and often, at the times of crisis, the laryngoscope light source is found defunct.
- *An IV cannula must be in situ before the procedure.* There may be sudden hypotension or hypertension during the procedure and then, IV drugs may be needed as emergency.
- Suction apparatus must be ready at the bedside.
- Do not insert the tube until you can see the vocal cords well.
- A bougie must be present in the intubation tray. This often makes the procedure easier.
- Size of tube: adult male: 7.5–8.5; adult female: 6.5–7.5.
- If the procedure gets prolonged, either call expert help or abandon the procedure temporarily; sedate the patient, oxygenate him/her; then try again.
- Do not overinflate the cuff.
- It is a good practice to perform portable X-ray after the procedure.
- If prone ventilation is planned (e.g., Covid-19), a flexometallic tube may be used.
- Please remember that laryngeal manipulation can cause sudden sympathetic surge and hypertensive emergency. Thus, if difficult intubation is anticipated,
IV fentanyl/beta-blocker may be used beforehand to prevent this surge.

**Initial Settings**

Often a physician is able to diagnose respiratory failure early and connect the patient to the ventilator. But then, the problem arises: how to control the settings? The touchscreen of a modern ventilator has numerous options, many of which seems daunting to a non-trained observer ([Figs. 1 and 2](#)). However, the following basic steps are easy to follow and can be adequate in most cases:

- **Mode:** After intubation, it is best to put the patient in assist-control (A/C) mode, at least for some time. This is the safest mode, especially if the patient is unstable.
- **FiO\textsubscript{2}**: This indicates the fraction of inspired oxygen. It can vary from 21 to 100. Just after intubation, especially if the intubation has been prolonged or difficult, it is best to keep the FiO\textsubscript{2} at 100 for a few minutes. Then, once the SpO\textsubscript{2} stabilizes, the FiO\textsubscript{2} should be quickly reduced. It must be remembered that one should not aim for a SpO\textsubscript{2} of 100%. This is unnecessary and harmful. Any SpO\textsubscript{2} more than 94% is considered adequate. If the SpO\textsubscript{2} is consistently above this level, the FiO\textsubscript{2} should be reduced.
- **Tidal volume:** Usually, the tidal volume is kept at 8–10 mL/kg. The body weight used for this calculation is the Ideal Body Weight (IBW). But in ARDS or restrictive lung disease, the tidal volume is kept low at 5–6 mL/kg.
- **I/E ratio:** Normally, this ratio is 1:2. In COPD, this may be changed to 1:2.5 or 1:3. Conversely in ARDS or diffuse parenchymal lung disease (DPLD), this ratio may be 1:1.7 or even 1:1. The principle is that in obstructive airway disease, there is air trapping. So, the expiratory time should be increased to allow more lungs emptying and prevent auto-positive end expiratory pressure (PEEP). Some ventilators have an option, inspiratory flow rate, which is an indirect control of the inspiration:expiration time (I:E) ratio. Normal flow rate is kept around 60 L/min. If the inspiratory flow rate is higher, the time of inspiration is

---

**Fig. 1:** Touchscreen of a typical ventilator [1—the mode (A/C: Assist Control); 2—tidal volume; 3—maximum inspiratory flow rate; 4—pause time (a short period between inspiration and expiration when there is no air flow. This pause time allows more gas exchange); 5—the shape of flow-time curve. This is usually kept decelerating to prevent barotrauma; 6—trigger for inspiratory flow; 7—IBW, based on which tidal volume is calculated; 8—FiO\textsubscript{2}; 9—PEEP; 10—respiratory rate]. Each of these 10 parameters can be changed independently.

**Fig. 2:** Control panel of ventilator in PCV mode [1—inspiratory pressure; 2—inspiratory time (this determines the I:E ratio); 3—rise time (this means the fraction of time during inspiration after which the set inspiratory pressure is reached. Here it is set at 50%. This means after half of inspiration, the peak pressure of 15 is attained). Rests are same as Fig. 1.
decreased, thus expiration time increases. In general, in air trapping (like emphysema), expiration time is prolonged; and in decreased compliance or severe hypoxia (like ARDS), inspiration time is prolonged. Altered I:E ratio or inverse ratio ventilation is not physiological. Thus, the patient may be in distress. Hence, in these cases, sedation is needed. Sedation may be given with midazolam, fentanyl, the preceding two drugs in combination, propofol, etc.

- **Respiratory rate:** Normally, in adults, this is kept 10–14/min. Only in ARDS cases, when tidal volume is kept low, the respiratory rate may have to be increased to maintain the minute volume. If respiratory rate and inspiratory flow are controlled, the I:E ratio automatically becomes fixed. Some ventilators have “Inspiratory Time” option (for PCV mode). This is also a way to fix the I:E ratio.
- **PEEP:** The PEEP is normally kept at 3–5 cm of H\(_2\)O. However, in ARDS, the PEEP needs to be higher (discussed latter).
- **Volume cycled (VCV) or pressure controlled (PCV)?** This is another issue where intensivists are divided into different opinionated groups. In general, the physician should remember that pressure controlled ventilation is much more difficult from maintenance point of view. Often, there is need of deep sedation with or without N/M blockade. A Spanish study, done in 2000, found that VCV versus PCV modes did not have any difference in mortality in ARDS patients.\(^6\) So, the physician should use the ventilation mode in which he/she is comfortable.

Besides these, there are numerous other settings (like trigger sensitivity or rise time). But those intricate details are best left to the intensivists.

**Recruitment Maneuvers**

After the Covid-19 experience, the use of recruitment maneuvers has become the buzzword in critical care. This is a specialized technique, mostly done by intensivists well versed in respiratory care. Some common procedures include:

- Airway pressure release ventilation
- Continuous positive airway pressure (CPAP) maneuver (putting the ventilator in CPAP mode and increasing the pressure to 30 cm H\(_2\)O for 30–40 seconds)
- Prone positioning

Prone positioning is a good method and if used early, it can have considerable benefit in ARDS. But the enthusiastic young medicine resident who plans to use prone positioning must remember the following:

- Prone positioning has to be maintained for 12–16 hours/day. For this to be successful, 1:1 nursing care is a must
- Usually these patients need continuous sedation
- Proper preventive care for decubitus ulcers must be given

**Observation Panel**

The parameters mentioned in the previous section are input variables. Once we have set these parameters, we have to observe the patient data from output section of the touchscreen (Fig. 3). This will tell us whether the respiratory mechanics are appropriate. There are many output variables like respiratory rate, peak pressure, and plateau pressure (in case of VCV) and minute ventilation. Also, the curves (pressure time, volume-time, flow-time, and flow-volume) have to be observed (Fig. 4).

In addition, modern ventilators have special procedures to measure certain parameters like static compliance (by inspiratory hold).

**Trouble Shooting**

This has been depicted in Table 1.

**Weaning**

While connecting a patient to a ventilator for lifesaving, proper weaning is equally important. Delayed weaning may cause VAP, tracheomalacia, critical care induced neuropathy, and other problems.

The most important point that must be remembered for weaning is that the underlying disease must be quelled. There should not be any significant cardiac or pulmonary dysfunction at the time of weaning. For example, if the pneumonia is controlled but still, the patient is on vasopressor support, then weaning is unlikely to be successful.

The modern technique is to put the patient on spontaneous mode of ventilator from A/C mode. In spontaneous mode, the \(\text{FiO}_2\) is slowly brought down to 21 and the pressure support is also reduced (Fig. 5). If this is sustained, that is oxygen saturation is maintained and
Fig. 3: The observation screen of a modern ventilator [1—peak inspiratory pressure; 2—plateau pressure; 3—work of breathing; 4—I:E ratio; 5—dynamic compliance (Normal: 50–70); 6—respiratory rate; 7—rapid shallow breathing index (this is important for weaning); 8—minute ventilation. This should be less than 10. Very high values indicate hyperventilation]. The recent ventilators may have even more parameters. Physicians should familiarize themselves with their machines.

Fig. 4: The various graphs visible on ventilator screen [1—pressure-time; 2—volume-time; 3—flow-time; 4—flow-volume]
hemodynamic stability is attained, then T-piece trial can be given. The T-piece trial can be given for 30-60 minutes on the first day and then increased on subsequent days. If this is successful, extubation can be done. In some places, it is routine to put the patient on BiPAP or HFNC after extubation for some time. But this is not universal.

**Difficulty in Weaning**

In some cases, there is repeated weaning failure. Some common causes are: Hypokalemia, sepsis, underlying heart failure, hypothyroidism, severe anemia, massive ascites, etc. Thus, in such cases, these extra-pulmonary causes should be ruled out. Some rules of the thumb to assess suitability for weaning:

- RSBI<105 (RSBI=Respiratory rate/tidal volume in liters)
- 5<Respiratory rate<35
- Absence of significant acidosis (respiratory or metabolic)
- Good mentation
- MV<10 L. Very high minute ventilation means the patient is hyperventilating and respiratory muscles are likely to be fatigued. In such cases, extubation should be delayed.

**Ventilator Strategy in COVID-19**

Any discussion about MV in 2020 is not complete without a few words about Covid-19. Covid-19 is associated with rapidly progressive ARDS like picture and MV is the lifesaving measure in such cases.

Although there are some data on pulmonary thrombosis in Covid-19, the gross ventilator strategies remains similar to the ARDS protocol. Thus, low tidal volume (4–8 m/kg), plateau pressure less than 30 cm of H₂O and conservative fluid strategy are advised. Also, a high-PEEP strategy is preferred and in severe hypoxemia, especially that which does not respond to optimum oxygenation, prone ventilation (12–16 hours/day) is recommended. A corollary of this last strategy is autoproning, which is now commonly practiced. Recruitment maneuvers can be done as needed, including staircase maneuvers (incremental PEEP).

**Ventilator Strategy in COPD**

In COPD, there is obstructive airway disease. The patients have chronic respiratory acidosis. The ventilator strategy involves using an FiO₂ to target SpO₂ of 88–92%. The respiratory rate is kept at 10–14/min, not higher. This is because due to chronic respiratory acidosis, the HCO₃ levels are elevated as compensation. If respiratory rate is kept high, there will be quick CO₂ washout. This will precipitate metabolic alkalosis. The flow trigger is kept around 2 L/min. If this is lower, tachypnea may occur. I:E ratio is kept at 1:2.5 to 3.5. PEEP is started at 5 and then

---

**TABLE 1**

<table>
<thead>
<tr>
<th>Observation</th>
<th>Possible causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refractory hypoxia</td>
<td>Oxygen source disconnected</td>
</tr>
<tr>
<td></td>
<td>Lung atelectasis</td>
</tr>
<tr>
<td></td>
<td>Airway block</td>
</tr>
<tr>
<td>Sudden hypotension</td>
<td>Development of pneumothorax due to VILI</td>
</tr>
<tr>
<td></td>
<td>Too high PEEP</td>
</tr>
<tr>
<td></td>
<td>VAP with sepsis</td>
</tr>
<tr>
<td>Tachypnea</td>
<td>Patient-PEEP dys-synchrony</td>
</tr>
<tr>
<td></td>
<td>Sepsis</td>
</tr>
<tr>
<td></td>
<td>Airway block</td>
</tr>
</tbody>
</table>

---

**Fig. 5:** Ventilator in spontaneous pressure support mode [1—the support pressure during inspiration; 2—expiratory sensitivity: this means when inspiratory flow drops to 25% of the peak value, the ventilator will switch to expiration]
increased as needed. Expiratory sensitivity can be kept above the default setting of 25% to increase the time of expiration. Tidal volume is adjusted to maintain MV at less than 6 L. This prevents overinflation in emphysema.

**Ventilator Strategy in DPLD**

At the outset, it must be explained to the patients' relatives that DPLD has a very poor prognosis in the CCU. The usual ventilator strategies are low tidal volume and high respiratory rate. If VCV is unable to maintain adequate minute ventilation (6–10 L/min) at acceptable plateau pressure (<30–35), then PCV mode may be used. High PEEP may be used to recruit the collapsed alveoli. I:E ratio may be kept at 1:1 or even 2:1. Prone ventilation may be used. Here, unlike COPD, high FiO2 may be used to improve oxygenation but increasing the PEEP can also achieve that objective. Inspiratory hold technique may be used to determine lung compliance and if this is very low, recruitment may be tried (as described earlier). Failure to improve oxygenation by any means warrants the use of ECMO.

**Interpretation of ABG in a Ventilated Patient**

Often, the physician is asked to review the arterial blood gas (ABG) report of a ventilated patient. This is a crucial test to check the adequacy of ventilation. In an ABG report, the first parameter to check is the PaO2. Low values indicate hypoxia and the need to increase FiO2 or PEEP. Ideally, the PaO2 should be around five times the FiO2. That is, if FiO2 is 30%, the PaO2 should be around 150. PaO2/FiO2<300 (where FiO2 is expressed in decimal) is the cut-off for defining ARDS. The next parameter to be checked is PaCO2. Low values indicate hyperventilation and the need to decrease minute ventilation. High PaCO2 indicates inadequate ventilation. Other reasons for increased PaCO2 are a high-carbohydrate diet and severe shock. ABG should be done 30–60 minutes after any change in ventilator settings.

**Conclusion**

- In VCV, the clinician should always look at the peak and plateau pressures during daily rounds. Persistent high pressures increases risk of barotrauma. But persistent high pressures can also mean the patient is biting the tube (thus sedation may have to be increased), or there is secretion clogging the tube (thus suction may be needed). Also, very high inspiratory flow rate may increase the peak pressure.
- A ventilated patient needs good nutrition. Otherwise, weaning will be difficult.
- If prolonged ventilation support is anticipated, early tracheostomy should be done.
- The alarm settings of a ventilator should be checked periodically to ensure that proper thresholds are set (Fig. 6).
- In PCV mode, frequent suction should be avoided because it releases the alveolar pressure and can precipitate atelectasis.
- Apnea settings should always be active (Fig. 7).
Acknowledgment: The pictures taken with the help of a highly skilled medical technologist, who wishes to remain anonymous for professional reasons.

References

Abstract

Introduction: Acute respiratory distress syndrome (ARDS) is an inflammatory lung parenchymal injury caused by various direct or indirect insults to pulmonary alveoli leading to exudative non-hydrostatic pulmonary edema. Ashbaugh and colleagues were the first to give a clinicopathological description of the ARDS in twelve adult patients. The ARDS Definition Task Force defined ARDS in 2012 (Berlin's criteria).

Pathophysiology: Pathophysiology of ARDS in patients with viral infections is not entirely clear. This is due to variable interaction between host factors and host immune response to viral antigen, which governs the severity of pneumonia or ARDS. Damage to alveolar endothelium results in increased endothelial permeability and recruitment and infiltration of leucocytes. Organ cross talk can occur through common language of pro-inflammatory cytokines and subsequently multi-organ dysfunction syndrome occur.

Management: Prognosis of ARDS in viral illness is poor with significant mortality and morbidity. Management is mainly supportive. Specific management includes anti-viral drugs, immunotherapies, and steroids. Supportive treatment has an important role to allow some time for specific treatments.

In this chapter, a focused review of ARDS caused by a variety of respiratory and non-respiratory viruses is presented along with cutting-edge developments in diagnostic and treatment modalities.

Introduction

Acute respiratory distress syndrome (ARDS) is an inflammatory lung parenchymal injury caused by various direct or indirect insults to pulmonary alveoli leading to exudative non-hydrostatic pulmonary edema. In 1967, Ashbaugh and colleagues were the first to give a clinicopathological description of the ARDS in 12 adult patients. Clinically, ARDS is characterized by acute onset of dyspnea, hypoxemia, and appearance of bilateral diffuse radiological infiltrates, which are not explained by volume overload or cardiac dysfunction. Severe sepsis is the most common cause leading to ARDS in about 50% of cases. These infections can affect lungs directly (pulmonary ARDS) or indirectly (extrapulmonary ARDS).

Respiratory viruses that can cause ARDS can be of two types:

- Pandemic and
- Non-pandemic respiratory viruses.

Influenza viruses (H5N1 and H1N1), Severe Acute Respiratory Syndrome Corona viruses (SARS-CoV), and Middle East Respiratory Syndrome (MERS) Corona viruses are notable examples of pandemic respiratory viruses, which can result in severe form of ARDS compared to non-pandemic respiratory viruses. SARS was reported first time in 2002, originated in southern China and eventually involved more than 8,000 persons worldwide. H1N1 influenza pandemic started in 2009 from California, through Mexico, the United States spread...
globally affecting mostly children and young adults. Recent addition to the list of pandemic respiratory virus is the SARS-CoV-2, which has been attributable to Coronavirus Disease-19 (COVID-19). Exact incidence of ARDS caused by viral diseases is unknown. Nevertheless, with the advent of multiplex nucleic acid amplification assays for the detection of viral pathogens, increasing numbers of viral etiologies are elucidated among the critically ill patients. In this chapter, a focused review of ARDS caused by a variety of respiratory and non-respiratory viruses is presented along with cutting-edge developments in diagnostics and treatment modalities.

Epidemiology

Incidence of ARDS varies extremely from country to country, being lowest in Brazil (10.1 per 100,000 person-years) to highest in the USA (78.9 per 100,000 person-years). Unfortunately there is no population survey based data published from India showing incidence of ARDS. According to a multinational observational study representing a large data from 29,144 ICU patients across 50 countries, ARDS was present in 10.4% of patients admitted to ICU and 23.4% of patients on mechanical ventilator.

Infections are the most common causes of ARDS, in which bacterial especially Gram negative microbial pathogens are the most common isolates from microbial cultures described in ARDS patients. Exact incidence of ARDS caused by viral infections is not known. The most common viruses detected from viral pneumonia were Influenza and Rhinovirus. Respiratory syncytial virus infection is common in pediatric age group. Despite viral detection in critically ill patients, their role in pathogenesis in ARDS is debatable.

Etiology and Risk Factors of ARDS Caused by Viral Illness

Respiratory viruses can cause ARDS in those who are immunocompromised, in extreme of ages, having severe comorbidities, hematopoietic cell transplant recipients, and acquired human deficiency syndrome and in those with exaggerated host response. Most common types of viruses causing pneumonia are divided according to genetic material they contain and their pandemcity (Table 1).

Pathophysiology of ARDS Due to Viral Infections

Pathophysiology of ARDS in patients with viral infections is not entirely clear. This is due to variable interaction between host factors and host immune response to viral antigen, which governs the severity of pneumonia or ARDS. Respiratory virus first invades nasal and bronchial epithelium, this invasion causes injury to respiratory airway and alveolar endothelium, cytokine release syndrome, which in worse case can take the form of cytokine storm. There is disruption in surfactant synthesis, which leads to alveolar collapse. Organ cross talk can occur through common language of proinflammatory cytokines and multi organ dysfunction syndrome occurs. In lungs, diffuse alveolar damage (DAD), a pathological hallmark of ARDS, has been observed in direct viral invasion of cells and lytic effects. Damage to alveolar endothelium results in increased endothelial permeability and recruitment and infiltration of leukocytes, which stimulate production of reactive oxygen species and nitric oxide that damage the epithelial-endothelial barrier. Few components of coagulation and fibrinolytic system are also activated in viral induced lung injury. These pathological events are translated to high incidence of both hemorrhagic and venous thromboembolic events. Another unique difference in viral and bacterial etiology of ARDS is the small lymphocytes as predominant immune cells at the site of lung injury. Due to cytokine-induced lymphocytic sequestration, lymphopenia ensues.

Few viruses (HSV, VZV, and CMV) remain in dormant state in ganglia or reticuloendothelial tissue and may become reactivated in the state of immunosuppression, especially in later stage of prolonged sepsis, characterized by immune-paralysis stage. Interestingly, in cases where ARDS results from inflammatory host response rather than direct cytopathic effect of virus, the antiviral therapy alone has limited value as has been observed in case of COVID-19 where SARS CoV-2 primarily injures the vascular endothelium.

Clinical Features

Clinical features of virus induced ARDS are often nonspecific, vague, and overlapping. It is not uncommon for such patients to present with features of both ARDS as well as underlying cause. Symptoms appear within
few days after the exposure to inciting factor and is often characterized by fever, cough, sore throat, myalgia, chills, or rigors. Watery diarrhea is also common along with abdominal pain, nausea, and vomiting, especially in illness caused by enterovirus and corona viruses.

Progressive symptoms of breathlessness, escalating requirement of oxygen, increased work of breathing, and alveolar infiltrates on chest imaging within 6–72 hours of an inciting event, ARDS should be highly suspected. On examination, patients may have tachypnea, tachycardia, and diffuse crackles, wheeze. In severe case, confusion, respiratory distress, cyanosis, and diaphoresis may be evident.

### Differential Diagnosis and ARDS Mimics

There are a number of conditions that may present as acute hypoxic respiratory failure with bilateral alveolar opacities and mimic ARDS. Some of the important mimics are being mentioned here.

- **Acute cardiogenic pulmonary edema**: It usually involves left ventricular systolic or diastolic dysfunction, but it may also occur due to fluid overload, hypertension, or severe renal disease. It can be distinguished from ARDS by evidence of cardiac dysfunction, elevated right-sided filling pressures, or related radiographic abnormalities. BNP or NT-proBNP is usually elevated.
In doubtful cardiac involvement, bedside transthoracic echocardiography may be performed to seek evidence of cardiac dysfunction.

- **Diffuse alveolar hemorrhage (DAH):** Almost two-thirds of patients with DAH will present with hemoptysis. On fiber optic bronchoscopy (FOB), frothy hemorrhagic secretions are visible throughout the airways. Bronchoalveolar lavage (BAL) cytology, though nonspecific, may show hemosiderin-laden macrophages.

- **Inflammatory or autoimmune conditions:** Several specific acute inflammatory conditions may mimic ARDS, which mainly include acute eosinophilic pneumonia, pulmonary vasculitis, acute interstitial pneumonitis, acute fibrinous organizing pneumonia, cryptogenic organizing pneumonia. These conditions can be ruled out by serology, BAL specimens, ANA, C-ANCA, P-ANCA, or lung biopsy.

- **Malignancy:** Hematological malignancies, such as leukemia, lymphoma, and pulmonary secondaries of solid tumors may be mistaken for ARDS.

- **Others:** Few embolic syndromes (fat embolism syndrome and amniotic fluid embolism syndrome) may present with hypoxemia and bilateral opacities. BAL examination may reveal fat or amniotic fluid debris. Air embolism can be suspected when patients experiencing sudden-onset respiratory distress in the setting of a known risk factor, intravenous catheter insertion, or trauma.\(^{24}\)

**Diagnostic Criteria**

The ARDS Definition Task Force has defined ARDS in 2012 known as Berlin’s criteria,\(^{25}\) which consist of rapid onset (within 7 days) of ARDS, impaired oxygenation status (PaO\(_2\)/FiO\(_2\) ratio <300 mm Hg with positive end expiratory pressure, PEEP ≥5 cm H\(_2\)O), characteristic radiological opacities (diffuse, bilateral, and fluffy), and non-cardiac origin of pulmonary edema (not explained by cardiac dysfunction or fluid overload). Out of these four criteria, oxygenation status defines grading of severity of ARDS. Mild ARDS is said when PaO\(_2\)/FiO\(_2\) ratio is in between 200 and 300 mm Hg, moderate ARDS is signified by a PaO\(_2\)/FiO\(_2\) ratio between 200 and 100 mm Hg. Value of PaO\(_2\)/FiO\(_2\) ratio ≤100 mm Hg corresponds to severe ARDS, which is associated with highest mortality rate.

**Microbiological Tests**

In the past, viral infections were purely clinical and the diagnosis of exclusion. Association of ARDS to viral etiology is confirmed by isolation of intact virus particles from cell culture or viral antigen detection by immunofluorescence, or multiplex RT-PCR. Among all these, most commonly performed and most rapid test is the multiplex RT-PCR, which gives comparable diagnostic accuracy compared to cell line culture (gold standard) and immunofluorescence techniques.\(^{26}\) The best sample for highest diagnostic yield is BAL collected during FOB, which is not always possible due to highly infectious nature. Bacterial and fungal culture, lung histopathology should also be performed to find coexisting infections leading to ARDS.

**Laboratory Tests**

Hematological profile may reveal leukopenia, lymphopenia and thrombocytopenia, which are markers of severity of viral illness. Arterial blood gas (ABG) analysis shows hypoxemia, which is often initially accompanied by acute respiratory alkalosis (due to compensatory hyperventilation), which later is replaced by hypercapnic respiratory acidosis (due to contraction of baby lung of ARDS). Metabolic acidosis (HCO\(_3\) <15 mEq/L) with hyperlactatemia may also develop due to the precipitating sepsis or associated organ injury.

Routine biochemical examination may manifest the evidence of organ injury (acute kidney injury or liver dysfunction) reflective of severe hypoxemia or associated shock and systemic inflammation (hypoalbuminemia and raised C-reactive protein). The prothrombin time (PT) and activated partial thromboplastin time (aPTT) may be prolonged, and D-dimer is elevated in severe viral illness and ARDS, suggesting prognostic importance of coagulation parameters.\(^{27}\)

**Imaging Studies**

Radiological findings in viral ARDS are variable and nonspecific and graded according to the severity of ARDS. The initial chest radiograph may be normal or may reveal typically bilateral diffuse alveolar opacities with dependent atelectasis. Computed tomography (CT) of the chest may show widespread patchy and/or coalescent airspace opacities that are usually more apparent in the dependent lung zones. The opacities can be subtle (e.g.,
patchy ground glass), particularly in early ARDS, but can become consolidative in appearance as severity worsens. Lung ultrasound has emerged as noninvasive, radiation free, reproducible and promising tool with reportedly higher sensitivity (83–92%) for the diagnosis of ARDS compared with CT chest.28

**Management**

Management of ARDS in viral illness is mainly supportive. Specific management includes antiviral drugs, immunotherapies, and steroids. Supportive treatment has an important role to allow some time for specific treatments.

**Respiratory Support**

Main issue in ARDS is worsening gas exchange and hypoxemia due to development of intrapulmonary shunt. Therefore, reversal of hypoxemia is the key principle in the management, which can be accomplished by respiratory support in the form of various oxygen delivery devices.

*Role of NIV and HFNOT:* Both NIV and HFNOT are noninvasive oxygen delivery devices, which are associated with improved outcomes in acute hypoxic respiratory failure (AHRF) patients compared to invasive mechanical ventilation. Before intubating an ARDS patient who has moderate hypoxemia, a trial of NIV or HFNOT may be warranted.

Ventilatory settings for ARDS lung include low tidal volume (TV) protective lung ventilation (TV, 4–8 mL/kg of predicted body weight) and frequently monitoring and maintaining plateau pressure (Pplat) below 30 cm H2O to ensure safety to baby lung. Severe hypoxemia in ARDS (<150) should trigger institution of more aggressive approach such as prone position ventilation, extracorporeal membrane oxygenation (ECMO), and use of lung recruitment maneuver. Proning improves PaO2 and decreases CO2 retention in patients with severe ARDS. A multicentric trial (PROSEVA) has shown significant mortality improvement with proning when performed for the duration of 16 hours a day.29-31

**Fluid Management in ARDS**

The Society of Critical Care Medicine, 2020, guidelines recommend to use restricted fluid and to use dynamic tests of fluid responsiveness to determine the need of fluid.

**Role of Corticosteroid**

In H5N1 and H1N1 influenza ARDS, routine use of steroid was discouraged citing higher rate of nosocomial infections. Similarly, corticosteroids did not show any benefit but led to delayed viral clearance in a multicenter study performed in patients (n=309) with the MERS. Recently Oxford University released findings of RECOVERY (Randomized Evaluation of COVID-19 thERapY) trial in which dexamethasone reduced the mortality by one-third in the patients on mechanical ventilator.32

**Antiviral Treatment**

Antiviral treatment can reduce the viral load, viral shedding, and reduce the disease transmission to close contacts. It has also shown proven benefit in radiologic clearance in ARDS. However, the efficacy of antiviral is maximized if administered within 48 hours of symptom onset. Specific antiviral drugs have been listed in Table 2.

Following respiratory virus is detected in respiratory or blood sample, antiviral treatment may be started. In pandemic times, empirical antiviral without waiting for result is warranted to improve outcome.

**Antimicrobial Treatment**

In critically ill patients, due to presence of various indwelling catheters, prevention and treatment of nosocomial infection is of equally importance while managing virus induced ARDS. Therefore, considering local antibiogram and host risk factors, empirical antibacterials may be indicated.34

**Other Pharmacological Treatment in ARDS Patients**

**Anti-cytokine Drug**

Two drugs, Aviptadil (concentrated in the lung 40%) and Remestemcel-L are being repurposed and investigated as therapeutics in COVID-19 ARDS (CARDS). Both down-regulate the synthesis of proinflammatory cytokines in the lung.

**Immunotherapies**

Palivizumab (monoclonal antibody) with intravenous immunoglobulin (IVIG) has been recommended in preventing Respiratory Syncytial virus (RSV) induced
severe pneumonia in high-risk infants and young children. Interferons-alpha-2a with ribavirin has been shown to improve survival in severe MERS-CoV infection. Convalescent plasma, due to its immunotherapeutic potential, has been successfully used in SARS, MERS, Ebola virus disease, and COVID-19.

Conclusion

Prognosis of ARDS in viral illness is poor with significant mortality and morbidity. Mortality increases with poor oxygenation status (46% with severe ARDS), late presentation, requirement of mechanical ventilation, presence of comorbidity, and higher age. However, survival has improved with advances in critical care management and early supportive measures. Patients, who survive ARDS, suffer long-term consequences, which include significant weight loss, poor functional status, and poor quality of life.

Recent viral pandemics have deepened our understanding of pathophysiology and management of ARDS. Clinical features are nonspecific and overlapping with viral illness as well as ARDS, but helpful in diagnosis. Multiplex RT-PCR of respiratory sample is confirmatory for viral ARDS. Supportive management include NIV or HFNOT initially and invasive lung protective ventilation in later more severe stage of ARDS.

Specific management includes antiviral and corticosteroid, which should be started early in the course of viral illness for the maximum therapeutic benefit. Other therapies are adjunctive and still investigational.

References


TABLE 2

<table>
<thead>
<tr>
<th>Antiviral drug</th>
<th>Indication</th>
<th>Further information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ganciclovir</td>
<td>CMV induced ARDS</td>
<td>Combination therapy with immunoglobulins can reduce the mortality in ARDS</td>
</tr>
<tr>
<td>Acyclovir or Valacyclovir</td>
<td>HSV or VZV induced ARDS</td>
<td>Dose of acyclovir is 10 mg/kg IV q8h for 7 days. Combination with varicella-zoster immune globulin (VZIG) therapy may be considered</td>
</tr>
<tr>
<td>Oseltamivir</td>
<td>H1N1 ARDS</td>
<td>Oral dose of 150 mg twice daily for 10 days is recommended</td>
</tr>
<tr>
<td>Zanamivir</td>
<td>Oseltamivir resistant H1N1 ARDS</td>
<td>Compassionate use of intravenous Zanamivir or add Ribavirin for Oseltamivir resistant cases</td>
</tr>
<tr>
<td>Peramivir</td>
<td>Investigational use in H1N1 ARDS</td>
<td>Compared to Oseltamivir, it has higher affinity for the influenza virus, and only single dose intravenous infusion is enough. Evidence is limited in critically ill patients</td>
</tr>
<tr>
<td>Favipiravir</td>
<td>H1N1 resistant to Neuraminidase inhibitors (NAIs) and SARS CoV-2</td>
<td>Favipiravir is an oral antiviral, approved in Japan for the treatment of influenza. Trials are ongoing for its use in COVID-19</td>
</tr>
<tr>
<td>Ribavirin</td>
<td>Respiratory Syncytial virus (RSV), Metapneumovirus, Parainfluenza virus, Measles, and Hantavirus</td>
<td>Intravenous formulation is available for use in critically ill patients. Ribavirin nebulization may be used in ARDS</td>
</tr>
<tr>
<td>Remdesivir</td>
<td>SARS CoV-2</td>
<td>It inhibits viral RNA polymerases broad-spectrum antiviral activity, emergency use authorization (EUA)</td>
</tr>
<tr>
<td>Cidofovir</td>
<td>Adenovirus induced ARDS</td>
<td>Dose of Cidofovir is 5 mg/kg/wk for 2 weeks, then every 2 weeks. Another dosing schedule is 1 mg/kg IV thrice a week</td>
</tr>
<tr>
<td>Lopinavir–Ritonavir</td>
<td>SARS CoV-1</td>
<td>400 mg and 100 mg, respectively twice a day for 14 days. No impact on SARS CoV-2</td>
</tr>
</tbody>
</table>


Abstract

Background: Analyzed report of Arterial Blood Gas is indicated in almost all patients admitted through emergency department (ED). ABG measurements are widely used in hospitals nowadays. Its use is particularly confined in ICU as monitor due to lack of test of accuracy and availability of simple method of analysis, management if started after correlating the clinical diagnosis with that of ABG diagnosis, mortality is reduced and discharge is improved.

Method: Prospective randomized controlled trial had been done over 136 patients of ED. Allocation ratio was 1:1. One group was managed in the background of analyzed ABG measurements and the control group was managed according to the traditional method. The ABG measurements were analyzed according to “rkdas Indian 2017 method of ABG interpretation”. The primary and secondary outcomes were assessed statistically. Patients and outcome access were blinded.

Result: The percentage of death in the study group is significantly less than the control group (p-value 0.22) with 95% confidence interval (3.08–17.52). The percentage of discharge is significantly more in study group than control group (p-value 0.036) with 95% confidence interval (50.25–73.35).

Conclusion: Management in the background of interpreted ABG decreases the mortality and improves the number of discharge.

Introduction

Arterial blood gas (ABG) is the most accurate test possible without restriction. It is widely used in hospitals particularly in ICU as a monitor. For physicians it is indicated in almost all patients who are admitted through emergency department (ED). Analyzed report is the ultimate representation of patients’ clinical status. Correct interpretation can lead to quicker and reliable changes in the plan of care. A sensitive physician is always interested to have analyzed ABG report before planning of management.

It helps the clinician in diagnosis and in some cases prognosis. Management started in the background of ABG significantly decreases the mortality. Failure to improve and deterioration of ABG parameters during management suggests early referral of the patients. In mixed disorder (multi-organ involvement) where mortality is high and complete clinical diagnosis at a glance is difficult, analyzed report of ABG helps the physicians. Wide utility of ABG is restricted in ICU as a monitor due to lack of absolute method of analysis and reliable test of its accuracy. Whenever clinical diagnosis does not match with ABG analysis, clinician often categorizes it imprecise. This difficulty has been overcome by new method of ABG interpretation.

A randomized control trial has been done and outcome is enthusiastic. Mortality is significantly decreased and
discharge is improved when management is started after ABG correlation of clinical diagnosis.

### Indications
- Organ Failure *(Flowchart 1)*
- Multi Organ Failure
- Poisoning/Drug Toxicity/Alcohol Intoxication
- Clinical Findings *(Flowchart 2)*
- Different Diseases *(Flowchart 3)*
- Monitor in Ventilated Patient

### Method
It was a prospective randomized control trial that was done in ED of Darbhanga Medical College, Laheriasarai. After getting ethics approval and consent form duly signed. Patients were randomized in two groups by simple
random allocation. Around 136 patients admitted in ED were randomized in 17 blocks and each block were allotted 8 patients with allocation ratio 1:1. Patients and outcome accesses were blinded. Arterial blood from 68 patients were taken before start of management and measurement were done by cobas b 121 machine.

Primary outcome (mortality and discharge) and secondary outcome (total days of stay in hospital) were recorded. Referral and LAMA were also considered. Statistical analysis was done by SPSS and standard method applied. Analysis of measured ABG data were done according to the method mentioned below.

“rkdas Indian 2017 Method of ABG Interpretation”

- **Accuracy of ABG:**
  - Base excess (BE) method
  - Relation of pH to H⁺ as already established
  - Relation of Hb% measured through CBC and ABG
  - Relation of SpO₂% derived by machine and measured by pulse oximeter

- **Gas analysis:**
  - SpO₂%
  - PaO₂ mm Hg
  - Relation between PaO₂ & SpO₂ from Hb% dissociation curve
  - PaCO₂ mm Hg
  - Decide type of respiratory failure
  - Derivation of PaO₂ = 150 – 1.25 × PaCO₂ mm Hg
  - P (A–a) O₂ value mm Hg
  - Cause of hypoxemia decided by algorithm
  - P/F, i.e., PaO₂/FiO₂, i.e., hypoxemia index to decide ARDS and its severity
  - Oxygen content (CaO₂) in patients having anemia
  - CaO₂ = Hb (gm/L) × 1.34 × SPO₂/100 + 0.003 × PaO₂

- **Electrolyte analysis:**
  - Osmolality = 2 × Na (mEq/L)
    + Plasma glucose (mg/dL) / 18
    + BUN (mg/dL) / 2.8
  - BUN = Blood urea (mg/dL) / 2.14
  - Blood volume = 2L / Hct (%) in cases of having no anemia or polycythemia

- The cause of K⁺ derangement is correlated with pH
- Chloride generally changes in parallel with plasma Na⁺
- Free calcium level
- AG = (Na⁺ + K⁺) – (Cl⁻ + HCO₃⁻) mmol/L
- Delta Gap (Δ gap) = anion gap – 12
- Gap-Gap ratio = delta gap/HCO₃⁻ deficit (24-HCO₃⁻)
- Δ gap + HCO₃⁻

- **Acid-Base analysis:**
  - pH & H⁺ value
  - HCO₃⁻ value
  - Direction of movement of H⁺ and HCO₃⁻ to know about primary cause from rule of thumb
  - Find out the compensatory change from knowing the primary cause
  - Movement of PaCO₂ and HCO₃⁻
  - Calculate delta gap, gap-gap ratio, and delta gap + HCO₃⁻ in a patient of high anion gap metabolic acidosis to decide associated non-anion gap metabolic acidosis and/or metabolic alkalosis
  - In case of primary respiratory cause decide:
    - \[ \frac{\Delta H^+}{\Delta \text{CO}_2} \]
    - if < 0.3 – Chronic cause
    - If > 0.8 – Acute cause
    - If 0.3–0.8 – Acute on chronic respiratory cause
  - Decide complex disease:
    - If PaCO₂ and HCO₃⁻ moves in opposite direction
    - In a respiratory cause if there is associated metabolic cause or vice versa
    - In a respiratory cause if anion gap is >20
    - In a respiratory cause if base excess is ≥±5
    - In a high anion gap metabolic acidosis there is associated non-anion gap metabolic acidosis and/or metabolic alkalosis
    - In ABG, if pH = 7.4 ± 0.04; PaCO₂ = 36–45 mm of Hg; HCO₃⁻ = 22–26 mmol/L then no acid base disorder
    - Diagnosis of acid-base analysis.

- **Complete diagnosis**

**Result**

In the study and control group female to male were 31(45.6%) to 37(54.4%) and 36(52.9%) to 32(47%), respectively, which were not significant. Also (mean age
TABLE 1  Clinical characterization

<table>
<thead>
<tr>
<th>Case N (%)</th>
<th>Control N (%)</th>
<th>p-Value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
<td>10(14.71)</td>
<td>9(13.24)</td>
<td>0.805</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>3(4.41)</td>
<td>3(4.41)</td>
<td>1.000</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>6(8.82)</td>
<td>5(7.35)</td>
<td>0.753</td>
</tr>
<tr>
<td>Sepsis</td>
<td>1(1.47)</td>
<td>7(10.29)</td>
<td>0.026</td>
</tr>
<tr>
<td>Cerebrovascular accident</td>
<td>5(7.35)</td>
<td>12(17.65)</td>
<td>0.066</td>
</tr>
<tr>
<td>Cardiopulmonary</td>
<td>4(5.88)</td>
<td>6(8.82)</td>
<td>0.510</td>
</tr>
<tr>
<td>Multiorgan disease</td>
<td>9(13.24)</td>
<td>0(0)</td>
<td>0.001</td>
</tr>
<tr>
<td>End stage renal disease</td>
<td>1(1.47)</td>
<td>2(2.94)</td>
<td>0.559</td>
</tr>
<tr>
<td>CKD with HF</td>
<td>1(1.47)</td>
<td>1(1.47)</td>
<td>1.000</td>
</tr>
<tr>
<td>Hepatobiliary disease</td>
<td>2(2.94)</td>
<td>3(4.41)</td>
<td>0.648</td>
</tr>
<tr>
<td>DM</td>
<td>1(1.47)</td>
<td>1(1.47)</td>
<td>1.000</td>
</tr>
<tr>
<td>Diabetic complication</td>
<td>9(13.24)</td>
<td>4(5.88)</td>
<td>0.142</td>
</tr>
<tr>
<td>Others</td>
<td>16(23.53)</td>
<td>15(22.06)</td>
<td>0.838</td>
</tr>
</tbody>
</table>

± SD) between the two groups were (49.34±17.29) and (49.50±16.99), which were also not significant. So far as clinical characterization is concerned, in both the groups the differences in percentage of systematic involvement in all the categories were not significant except in sepsis of study group and multi-organ disease in control group (Table 1).

On analysis of ABG measurement it was found that 12(17.6%) ABG were inaccurate by BE method. When the relation between pH&H+ were matched in table, all 12 ABG were found accurate (Table 2).

Moderate to severe hypoxemia were found in 10(14.72%) of patients. Type I respiratory failure were present in 5(7.35%) of patients. Type II respiratory failure were found in 9(13.2%) patients out of which 4(5.9%) were due to hypoventilation alone, 4(5.9%) with severe hypoxemia and 1(1.5%) with moderate hypoxemia.

Hyponatremia was present in 58(85.3%) patients and conspicuously one had hypernatremia. 13(19.17%) patients had hyperkalemia. Hypocalcaemia was a common finding. Hypochloremia was present in 4(6.2%) of patients.

Discussion

In my study, 17.6% of ABG were inaccurate by BE method while in general population difference between measured and derived HCO3 is 4.5% only.6

In my study, only 42.6% of patients had correct provisional diagnosis in ED. While in 80.9% of patients ABG diagnosis matched with the final clinical diagnosis. Around 19.1% of ABG were not compared to final clinical diagnosis because the patient either left or referred to higher center (Table 3).

Different clinical characterizations of both groups were not significant except in sepsis and multi-organ disease group. Sepsis is a condition where there is multi-organ involvement commonly and in both the conditions mortality is high and almost equal (Table 1).

In 5.9% of severe hypoxemia pulse oxymeter was not useful.

The common acid-base disorders were respiratory 42(61.8%) among which the most common findings were acute or chronic respiratory alkalosis 21(50%). In the metabolic cause, acidosis was more than alkalosis (Table 2). One (1.5%) of patients had no acid base disorder.

Acute exacerbation of chronic obstructive pulmonary disease (COPD) was provisionally diagnosed in 4.4% of patients, but only 1.5% had acute on chronic respiratory acidosis (acute exacerbation of COPD). Around 8.8% of patients were clinically diagnosed as COPD/cor pulmonale in which 3% had acute or chronic respiratory acidosis (acute exacerbation of COPD).
### TABLE 2  ABG analysis

<table>
<thead>
<tr>
<th>Accuracy of ABG (BE method)</th>
<th>Respiration Failure Type I</th>
<th>Gas analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accurate</td>
<td>Not accurate</td>
<td>Hypoxemia</td>
</tr>
<tr>
<td>Total</td>
<td>No</td>
<td>Mild</td>
</tr>
<tr>
<td>Mod.</td>
<td>Severe</td>
<td>Hypoventilation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percentage</th>
<th></th>
<th>Frequency</th>
<th>Percentage</th>
<th></th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accurate</td>
<td>56</td>
<td>82.4</td>
<td>Not accurate</td>
<td>12</td>
<td>17.6</td>
<td>Hypoxemia</td>
<td>58</td>
<td>100%</td>
</tr>
<tr>
<td>Total</td>
<td>68</td>
<td>100</td>
<td>Yes</td>
<td>6</td>
<td>9.5</td>
<td>Total</td>
<td>7.35</td>
<td>100</td>
</tr>
<tr>
<td>Type I</td>
<td>Mod.</td>
<td>5</td>
<td>7.35</td>
<td>Severe</td>
<td>1</td>
<td>1.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>4</td>
<td>5.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type II</td>
<td>Hypoventilation</td>
<td>6</td>
<td>8.82</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mod.</td>
<td>9</td>
<td>13.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypoxemia</td>
<td>1</td>
<td>1.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>4</td>
<td>5.9</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Electrolyte analysis

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Na(^+) (mmol/L)</th>
<th>K(^+) (mmol/L)</th>
<th>Ca(^{++}) (mmol/L)</th>
<th>Cl(^-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>58</td>
<td>10</td>
<td>0</td>
<td>68</td>
</tr>
<tr>
<td>Percentage</td>
<td>85.3</td>
<td>14.7</td>
<td>0</td>
<td>100</td>
</tr>
<tr>
<td>Low</td>
<td>68</td>
<td>32</td>
<td>13</td>
<td>68</td>
</tr>
<tr>
<td>Normal</td>
<td>32</td>
<td>23</td>
<td>13</td>
<td>68</td>
</tr>
<tr>
<td>High</td>
<td>23</td>
<td>13</td>
<td>1</td>
<td>100</td>
</tr>
<tr>
<td>Metabolic</td>
<td>32</td>
<td>13</td>
<td>1</td>
<td>100</td>
</tr>
<tr>
<td>Metabolic</td>
<td>97.1</td>
<td>2.9</td>
<td>0</td>
<td>100</td>
</tr>
<tr>
<td>Total</td>
<td>97.1</td>
<td>2.9</td>
<td>0</td>
<td>100</td>
</tr>
</tbody>
</table>

#### Acid-base analysis

<table>
<thead>
<tr>
<th>pH</th>
<th>Low</th>
<th>Normal</th>
<th>High</th>
<th>Total</th>
<th>Metabolic</th>
<th>Res.</th>
<th>No defect</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>13</td>
<td>23</td>
<td>32</td>
<td>68</td>
<td>25</td>
<td>42</td>
<td>1</td>
<td>68</td>
</tr>
<tr>
<td>Percentage</td>
<td>19.1</td>
<td>33.8</td>
<td>47</td>
<td>100</td>
<td>36.8</td>
<td>61.8</td>
<td>1.5</td>
<td>100</td>
</tr>
</tbody>
</table>

#### Primary Resp. Cause

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>0</td>
<td>4</td>
<td>6</td>
<td>8</td>
<td>21</td>
<td>42</td>
<td>3</td>
<td>2</td>
<td>11</td>
<td>8</td>
<td>1</td>
<td>25</td>
</tr>
<tr>
<td>Percentage</td>
<td>0.95</td>
<td>7.1</td>
<td>14.3</td>
<td>19</td>
<td>50</td>
<td>100</td>
<td>12</td>
<td>8</td>
<td>44</td>
<td>32</td>
<td>4</td>
<td>100</td>
</tr>
</tbody>
</table>
In 13(19.1%) of patients, ABG diagnosis were not matched with the final clinical diagnosis due to LAMA and referral of patients.

Death in study group were 7(10.3%), while in control group it was 17(25%), p-value 0.222, significant. Around 42(61.8%) of patients discharged in study group while 30(44.1%) were discharged in control, p-value 0.036, significant. LAMA were 10(14.75%) and 7(10.3%) in study and control group, respectively. Around 9(13.2%) and 14(20.6%) patients were referred in study and control group respectively which is not significant (Table 4).

In 10.3% patients died during therapy compared to 25% in control group (Fig. 1). ABG predicted in almost all cases about the seriousness of the disease. In 5.9% patients of acute heart failure 2.9% had metabolic acidosis and died, 6 1.5% had normal pH and 1.5% had alkalosis, they survived. 8 There was 1.5% case of AMI and pH was 7.25. In acute coronary syndrome if pH < 7.3, mortality is expected to be 100%. 9 Around 2.9% had arrhythmia with acidosis, they succumbed during therapy. Acidosis contributes to the development of arrhythmia, and the patient is resistant to therapy in acidosis. One patient had CVA with altered sensorium and PaO₂ was 50 mm Hg. Commonly in such type of patients with CVA there is hyperventilation and PaO₂ should not be low but this was due to associated aspiration pneumonia. Around 1.5% of patients who died were a case of complex disease with pH 7.50, mortality is high in complex disease and pH > 7.55.

There were 4.4% of cases of acute exacerbation of COPD, 1.5% had pH 7.32 managed successfully with low FiO₂ and with Bi-Pap machine; 2(2.9%) patients had pH

<table>
<thead>
<tr>
<th>Match of ABG diagnosis to</th>
<th>Frequency no.</th>
<th>Percent (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Provisional diagnosis</td>
<td>29</td>
<td>42.6</td>
</tr>
<tr>
<td>Final clinical diagnosis</td>
<td>55</td>
<td>80.9</td>
</tr>
<tr>
<td>Not available to match the clinical diagnosis</td>
<td>13</td>
<td>19.1</td>
</tr>
<tr>
<td>Total</td>
<td>68</td>
<td>100</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Case</th>
<th>Frequency</th>
<th>Percent</th>
<th>95% Conf. Interval</th>
<th>Control</th>
<th>Frequency</th>
<th>Percent</th>
<th>95% Conf. Interval</th>
<th>p-Value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>7</td>
<td>10.3</td>
<td>3.08–17.52</td>
<td>17</td>
<td>25</td>
<td>14.71–35.29</td>
<td>0.022</td>
<td>Significant</td>
<td></td>
</tr>
<tr>
<td>Discharged</td>
<td>42</td>
<td>61.8</td>
<td>50.25–73.35</td>
<td>30</td>
<td>44.1</td>
<td>32.3–55.9</td>
<td>0.036</td>
<td>Significant</td>
<td></td>
</tr>
<tr>
<td>LAMA</td>
<td>10</td>
<td>14.7</td>
<td>6.28–23.12</td>
<td>7</td>
<td>10.3</td>
<td>3.08–17.52</td>
<td>0.436</td>
<td>Not significant</td>
<td></td>
</tr>
<tr>
<td>Refer</td>
<td>9</td>
<td>13.2</td>
<td>5.15–21.25</td>
<td>14</td>
<td>20.6</td>
<td>10.99–30.21</td>
<td>0.25</td>
<td>Not significant</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 1: Bar diagram of outcome in percentage
7.12 and 7.21 in which 1 patient (1.5%) was associated with non-anion gap metabolic acidosis and treated with Bi-pap and improved; while the other needed ventilator but refused to give consent and left against medical advice.

Around 14(20.6%) patients were suffering from type I and type II respiratory failure together. Their causes were decided by algorithm and managed accordingly. ABG acted as monitor in all the 75% mixed disorder patients. The outcome of the two groups shows that patients managed in the background of ABG findings had mortality less by 14.7% than control group, which is significant. The overall stay of the discharge patients were less in study group.10

**Conclusion**

Accuracy of ABG should be tested by different methods, if BE method fails. In this study, all ABG measurements were accurate. At ED in the hand of even an expert physician only about 50% of provisional diagnosis is correct. Around 81% of final clinical diagnosis matched with ABG diagnosis. From the time of admission proper management is started with ABG support in almost all cases, which may be a cause of better outcome. Almost all patients admitted through ED had ABG changes and thus indicated in all patients. In multi-organ disease (complex disease), the mortality is high. Measuring SpO2 through pulse oxymeter become difficult in severe hypoxemia and only ABG can say about hypoxemic state of patients.

Respiratory alkalosis is common ABG finding. Around 21% patients were of respiratory failure and their cause is easily decided by algorithm and thus managed accordingly. Around 13% of patients admitted in ED are of COPD out of which 33% is diagnosed as acute exacerbation of COPD and when ABG matching is done 33% of them were wrong. ABG is accurate method to decide acute exacerbation of COPD.

ABG also acts as monitor even outside of ICU, particularly in mixed disorders. Management in the background of analyzed report of ABG reduces mortality significantly.

Analysis report of ABG in ED is physician’s necessity for management. In other word physician can proclaim ABG as “gold standard in ED.”

**References**

Abstract
Hyperkalemia is a medical emergency and potentially life-threatening electrolyte disturbance. It usually presents in clinical setting of acute kidney injury, tumor lysis syndrome, major tissue damage like road traffic accidents or rhabdomyolysis. People above 60 years and those taking drugs like NSAIDs, Betablockers, and ACE inhibitors are at high risk. High index of clinical suspicion is required to diagnose hyperkalemia, since it may often be asymptomatic. All care should be taken while sending blood sample for serum potassium levels, since we may encounter pseudohyperkalemia. ECG changes usually correlate well with severity of hyperkalemia. Urgent measures are required when serum potassium is more than 7.5 mEq/L. Measures include immediate stabilization of myocardium with intravenous calcium gluconate and shift of Potassium from ECF to ICF by salbutamol nebulizations. Oral administration of ion exchange resins will reduce the serum potassium levels but slowly. We may have to go for hemodialysis in acute and severe cases. Simultaneously we have to address the original cause.

Introduction
Hyperkalemia is a medical emergency and potentially life-threatening electrolyte disorder especially in patients with renal disease, heart failure, and people using certain drugs like angiotensin converting enzyme (ACE) inhibitors, angiotensin-II receptor blockers (ARBs), beta-blockers, or non-steroidal anti-inflammatory drugs (NSAIDs). It is rare in healthy general population. Certain groups are at greater risk.

Why is It to be Addressed?
Acute severe hyperkalemia would become a life challenging and leads to dangerous cardiac arrhythmias and death if not attended to earliest.

Historical Background
- Sir Humphry Davy (1807), a British Chemist, coined the element, Potassium. It is called kalium in (Latin).
- However, Kalium is renamed as potassium. But in Periodic Table its symbol is retained as “K.”
- Atomic number: 19; Element classification: Metal; Period number: 4; Molecular weight: 39.098 g/mol; and Valance: +1.

Etymology
- Derived from modern Latin word “potassa”
- Potassa + ium

Definition
- Hyperkalemia is defined as a plasma potassium level more than 5.5 mmol/L.
- There is no internationally accepted definition for hyperkalemia; The “European Resuscitation Council” defines hyperkalemia as a plasma level more than 5.5 mmol/L and severe hyperkalemia as more than 6.5 mmol/L.
Basics
- Potassium is the principal intracellular cation 140–150 mEq/L.
- The plasma (serum) potassium is 3.5–5.0 mEq/L, while the whole blood contains 50 mEq/L.
- Dietary potassium is absorbed cent percent through GIT and 90% is excreted through urine and 10% through faces.
- It maintains intracellular osmotic pressure.
- It is required for regulation of acid base balance and water balance in the cells.
- It is required for the activity of the enzyme "Pyruvate kinase."
- It is essential for transmission of nerve impulse, cardiac muscle activity, and biosynthesis of proteins by ribosomes.

Note:2
- The factors that enhance cell uptake of potassium from ECF compartment include: insulin, alkalosis, beta catecholamines, hyperosmolality, and cell damage.
- The factors that impair cell uptake of potassium from ECF compartment include: acidosis, chronic kidney disease, diabetes mellitus, and alpha catecholamines.

The Clinical Spectrum of Hyperkalemia Presenting in ICU, Requiring Immediate Care
Acute kidney injury, acute on chronic kidney disease, MODS (Fig. 1). Acute necrotizing pancreatitis (Fig. 2), septic abortion, burns, snake bite, muscle injury (rhabdomyolysis), Tumorlysis syndrome, sepsis, disseminated intravascular coagulation, metabolic acidosis, and hemolysis.

Epidemiology
- Most cases in hospitalized patients are due to chronic drug use and renal compromise.
- Risk factors include advanced age, significant prematurity and the presence of diabetes mellitus, heart failure, and sepsis.
- Hyperkalemia has been reported in 1.1–10% of all hospital admissions.3

Pseudohyperkalemia4-6
- Pseudohyperkalemia represents an artificially elevated potassium concentration in the plasma. This is due to movement of potassium (K+) from the cells immediately prior to or following venepuncture.
- Pseudohyperkalemia is suspected when hyperkalemia is reported by laboratory in an asymptomatic individual with no obvious underlying cause and no ECG abnormalities.
- Faulty blood collection procedure, like excessive clenching of fist, prolonged tourniquet application during blood drawing, moisture in storage and delay in testing the blood sample, and gross change in room temperatures are some of the responsible factors.
Causes of Hyperkalemia

- Increased intake:
  - High potassium containing foods
  - Potassium containing drugs
  - Intravenous fluids containing potassium
- Tissue breakdown:
  - Catabolic state
  - Hemolysis
  - Rhabdomyolysis
  - Tumorlysis syndrome
  - Bleeding into soft tissues, body cavities
- Shift of potassium into extracellular fluid:
  - Metabolic acidosis
  - Tissue damage
  - Uncontrolled diabetes mellitus
  - Hyperkalemic periodic paralysis
- Impaired excretion:
  - Acute kidney injury
  - Potassium sparing diuretics
  - Reduced tubular excretion: Addison’s disease
  - Reduced circulatory volume

Common Risk Factors

- Premature infants probably due to renal prematurity
- Males are more affected because of excess muscle mass
- Elderly people >60 years
- Military recruits, drug abusers, Sickle cell anemia; conditions with high risk for rhabdomyolysis
- Peripheral vascular disease

Drug use:

- Excess and chronic use of NSAIDs
- Use of potassium sparing diuretics, beta-blockers, ACE inhibitors, ARBs especially with renal compromise
- Direct renin inhibitors (e.g., Aliskiren)
- Cyclosporine or tacrolimus
- Antibiotics (e.g., pentamidine and cotrimoxazole)
- Oral contraceptive pills
- Heparin therapy in bed ridden people

Foods Rich in Potassium (Fig. 3)

Vegetables: Potatoes, tomatoes, pumpkin, cooked spinach, Brussels sprouts
Fruits: Orange, banana, honey dew, prunes, raisins, other dried fruits

Other foods: Chocolate, nuts, seeds, peanut butter, onion, yogurt, bran products

Grading of Severity of Hyperkalemia

Hyperkalemia can be graded according to the serum concentration of potassium into:

- Mild 5.5–6.5 mmol/L
- Moderate 6.5–7.5 mmol/L
- Severe >7.5 mmol/L

Clinical Presentation

- Hyperkalemia is often asymptomatic, hence it can be called silent killer.
- Most patients are relatively asymptomatic with mild and even moderate hyperkalemia.
- Presentation may be nonspecific: with fatigue and vague muscular weakness. The patient may complain of tingling around lips or in fingers.

Physical Examination Findings

Physical signs vary as per the etiology and also serum potassium concentration:

- Pulse slow and usually irregular
- Hypertension and edema in the setting of renal disease
- Signs of hypoperfusion
- Shortness of breath, palpitations, chest pain, nausea, vomiting, and paresthesias
- Distension of abdomen and absent bowel sounds in case of acute necrotizing pancreatitis
- Muscle tenderness may be present in patients with rhabdomyolysis
- Jaundice may be seen in patients with hemolytic conditions, acute cholecystitis

Note:

- Symptoms usually develop at levels of 6.5–7 mEq /L
- But the rate of change is more important than the numerical value
- Individuals with chronic hyperkalemia may be asymptomatic relatively at higher level
- Whereas patients with a sudden rise of plasma potassium may develop severe symptoms relatively at lower values of potassium
- Neuromuscular manifestations include paresthesias and fasciculations in the arms and legs
Fig. 3: Fruits and vegetables rich in potassium.
As the serum K+ continues to rise, an ascending paralysis with eventual flaccid quadriplegia supervenes. Classically, trunk, head, and respiratory muscles are spared. Cranial nerves least affected. However, respiratory failure can rarely occur.

**ECG Manifestations of Hyperkalemia (Figs. 4A and B)**

Typical ECG findings in hyperkalemia include progress from tall, "peaked" T waves and a shortened QT interval to lengthening PR interval and loss of P waves, and then to widening of the QRS complex culminating in a "sine wave" morphology and death if not treated.

**Diagnosis**

Diagnosis of hyperkalemia depends on:
- Clinical suspicion
- Serum potassium concentration
- Characteristic ECG manifestations

*Note:*
- Beware of pseudohyperkalemia
- Then etiology should be recognized

Hence the investigations should envisage:
- Whether true hyperkalemia is present?
- How much severe it is?
- What is its etiology?
- Risk factors if any?

**Investigations**

- Serum electrolytes: Sodium, potassium, magnesium, calcium, chloride
- Blood urea, serum creatinine, bicarbonate (when Tumorlysis syndrome is suspected)
- Creatine kinase (especially when Rhabdomyolysis is suspected)
- Serum amylase, liver enzymes
- Baseline 12 lead ECG, and later repeat ECGs
- Serum cortisol levels
- Arterial blood gas report
- Ultrasound scan of abdomen
- CT scan abdomen, MRI abdomen depending on the clinical setting
- Urine complete analysis, urine electrolytes
- Complete blood counts

- Microbiological tests depending on suspected multiple organ failure or sepsis
- Blood hematology, if disseminated intravascular coagulation is suspected

*Note: Clinical setting along with comorbid history, should guide the investigations required for workup of a case toward assessing the severity of hyperkalemia, and clinical background*

**Transtubular Potassium Gradient (TTKG)**

- Estimation of TTKG can reveal whether hyperkalemia is due to renal cause or extra renal cause and mineralocorticoid bioactivity in people with hyperkalemia
- TTKG value <6 suggests renal cause
- TTKG value >6 suggests extra renal cause
- Typical TTKG on normal diet is 8–9
- TTKG <5 in presence of hyperkalemia may indicate mineralocorticoid deficiency

\[
\text{TTKG} = \frac{\text{Urinary K}^+}{\text{Serum K}^+} \times \frac{\text{Serum osmolality}}{\text{Urine osmolality}}
\]

**Emergency Management**

- Acute rise in serum potassium is a medical emergency
- Urgent treatment is required in cases of potentially fatal hyperkalemia, that is, serum K+ >7.5 mEq/L

Basic principles of acute management of severe hyperkalemia in ICU:
- Stabilization of myocardium
- Shift of extracellular fluid (ECF) potassium to intracellular fluid (ICF)
- Removal of the excess potassium from the body *Stabilization of myocardium* (to be done under cardiac monitoring) to prevent dangerous ventricular arrhythmia.
- Inj. Calcium gluconate (10% solution) 10–20 mL to be given iv over 5–10 minutes, or
- Inj. Calcium chloride 5 mL of 10% solution over 2 minutes
- However, calcium chloride can cause tissue necrosis if it extravasates and requires a central line
- The dose can be repeated after 5–10 minutes, if there is no change in ECG
Calcium antagonizes the cardiac and neurological effects of hyperkalemia and prevents cardiac toxicity and dangerous cardiac arrhythmias.

It should be noted that calcium gluconate does not reduce plasma potassium.

**Shift of ECF potassium to ICF:**
- Insulin and glucose infusion
- Beta 2 adrenergic agonists
- Alkalinizing agents

**Insulin and glucose infusion:**
- Administer regular insulin 10 units as iv bolus + 50% dextrose 50 mL as iv bolus
- Onset of action is within 15–30 minutes and action lasts for 2–6 hours
- Initial bolus of glucose insulin should be followed by continuous infusion of 5% dextrose at 100 mL/hour to prevent late hypoglycemia
- Insulin—dextrose is treatment of choice in case hyperkalemia with end stage renal disease (ESRD)
The plasma potassium concentration will fall by 0.5–1.5 mEq/L. This effect begins in 15 minutes and usually lasts for 4–6 hours.

In diabetic patients avoid extra dextrose infusion.

Alkalinizing agents:

- One ampoule of sodium bicarbonate 7.5% providing 44.6 meq is given over 5 minutes or better added to 5% glucose infusion.
- Onset of action is within 5–10 minutes and the effect lasts for 1–2 hours.
- However, the use of alkalinizing agent is controversial and contra indicated in patients with ESRD.

Note:

- When there is associated metabolic acidosis like in Tumorlysis syndrome alkali therapy can be used.
- It should not be advocated in cases of ESRD.
- Beta 2 adrenergic agonists (Salbutamol, Ventolin, or Albuterol): Salbutamol is given in a nebulized form.
- 5 mg of salbutamol mixed with 3–4 mL of normal saline and administered through a high flow nebulizer over 10 minutes.
- It generally becomes effective in 30–60 minutes and its effect persists for 2–4 hours.
- It lowers serum potassium by 0.5–1.5 mEq/L.

Rationale

Beta 2 agonists such as salbutamol promote cellular uptake of potassium and effectively lowers serum potassium level.

Removal of the Excess Potassium from the Body

- Loop or thiazide diuretics
- Cation exchange resins
- Dialysis

Loop diuretic:

- Fruesimide 40–80 mg iv, the onset of action within 15 minutes, lower value of K+ maintained for 2–3 hours.
- Hydrochlorothiazide can be given in mild cases.
- Potassium sparing diuretics contraindicated.

Cation exchange resin:

- Cation exchange resins, such as sodium polystyrene sulfonate (SPS) (Kayexalate) promote the exchange of sodium for potassium in gastrointestinal tract.
- Each gram binds 1 mEq of potassium and releases 2–3 mEq of sodium.

- When given orally the usual dose is 25–30 gm mixed with 100 mL of 20% sorbitol 3–4 times daily (sorbitol prevents constipation).
- Onset of action more than 2 hours, and maintained for 4–6 hours.
- It can also be given as retention enema consisting of 50 gm of resins and 50 mL of 70% sorbitol mixed in 150 mL of water every 4–6 hourly.
- In general each enema can lower the plasma potassium concentration by 0.5–1.0 mEq/L within 1–2 hours and effect will last for 4–6 hours.

Adverse effects of resins:

- Anorexia, nausea, vomiting, and constipation.
- Intestinal necrosis typically of colon or ileum is a rare but fatal complication of SPS. Intestinal necrosis is more common in patients with reduced intestinal motility (in postoperative state).

Note: Patiromer is a non-absorbed polymer provided as a powder for suspension which binds potassium in exchange of calcium.

Dialysis

Emergency dialysis is a final measure for lethal hyperkalemia that has not responded to more conservative measures or for patients who had chronic renal failure.

- The most rapid and effective way of lowering the plasma potassium concentration is hemodialysis, which can produce a 1 mEq/L drop in serum potassium after 1 hour, and 2 mEq/L drop after 3 hours.
- Peritoneal dialysis also removes potassium but is only 15–20% as effective as hemodialysis.
- Patients with acute kidney injury require temporary but urgent venous access for hemodialysis.

Conclusion

In ICUs, we often come across electrolyte disturbances. Hyperkalemia may sometimes present silently. Hence, one should have a high index of clinical suspicion and act accordingly. Calcium gluconate, for membrane stabilization and β2-agonists, and insulin with glucose to be used in the treatment of hyperkalemia to induce a redistribution of K+, and dialysis to be reserved for cases with severe hyperkalemia and renal compromise. The workup depends more so on the clinical background.
Hyperkalemia in ICU

CHAPTER 105

653

References
