Marmara Medical Journal 2016; 29 (Special issue 2): 36-48
DOI: 10.5472/MMJsi.2902.09

REVIEW ARTICLE

Improvements in intensive care units

Sait KARAKURT, Aslihan YALCIN, Huseyin ARIKAN

ABSTRACT

We have had some important improvements in the intensive care units (ICU) like high flow oxygen system (HFOS), therapeutic hypothermia, extra corporeal membrane oxygenation (ECMO), extra corporeal carbon dioxide removal (ECCOR), echocardiography (ECHO) and ultrasonography (US).

HFOS gives oxygen to the patients at rates of flow higher (up to 60 L/min) than that delivered traditionally in LFOS (up to 16L/min). It is obtained 1 cmH2O PEEP for every 10L/min of flow delivered by HFOS. HFOS serves as an important alternative to noninvasive mechanical ventilation especially in the management of the patients with hypoxemic respiratory failure.

Post-resuscitation care consists of optimization of oxygenation and ventilation, avoiding hypotension, treating immediate precipitants of cardiac arrest such as acute coronary ischemia and initiating therapeutic (induced) hypothermia. Therapeutic hypothermia decreases cerebral metabolic rate, blood volume, and intracranial pressure, prevents reperfusion injury. So hypotermia protects cerebral functions.

ECMO, the type of cardiopulmonary support, has become an essential tool in critical care patients with severe respiratory and cardiac failure, refractory to conventional therapy methods.

Critical care ultrasonography (CCUS) and echocardiography have utility for intensivist-performed, immediate diagnoses of life threatening diseases, with no need to transport patients to radiology or cardiology departments or wait for radiologist or cardiologist on a consultative basis. CCUS and echocardiography should be an essential part of training of every ICU physician.

Keywords: Intensive Care Units, Oxygen inhalation therapy; Extracorporeal membrane oxygenation; Extra corporeal carbon dioxide removal; Hypothermia, Induced; Echocardiography; Ultrasonography

Sait Karakurt (X), Aslihan Yalcin, Huseyin Arikan
Department of Pulmonary and Critical Care Medicine, School of Medicine, Marmara University, Istanbul, Turkey
e-mail: saitkarakurt@hotmail.com

High flow oxygen system

Traditionally, oxygen delivery systems can be classified as low flow oxygen system (LFOS) and high flow oxygen system (HFOS). If the patient's respiratory demand is met completely by the system, the system is called as high flow oxygen system such as venturi mask. If the patient ventilator demand cannot meet by the oxygen delivery system, the system is called as low flow oxygen system such as nasal cannula, simple mask (Table I).

Table I. Comparison of Low-Flow and High-Flow Oxygen Systems

	LOW FLOW OXYGEN SYSTEMS	HIGH FLOW OXYGEN SYSTEMS
Patient's all respiratory requirements	Cannot meet, part of the VT must be supplied by breathing room air	Meet
Examples	Nasal cannula, simple mask, mask with reservoir bag (partial or non-rebreathing mask)	Venturi mask, face tent, aerosol mask, T piece tracheostomy collar
Constant and predictable FiO2	May not provide	Provide
Change in patient ventilator pattern	Affect FiO2	Does not affect FiO2
Reservoir capacity	Affect FiO2	Does not affect FiO2
Cost	Cheap	Expensive

It is accepted that the adult patient whose tidal volume is between 300-700 mL and whose respiratory rate below 25/min and whose ventilatory pattern is regular may use LFOS, because the patient will receive consistent and predictable FiO_2 on a LFOS. If any one of these three criteria is not met, the patient should be on HFOS.

HFOS gives oxygen to the patients at rates of flow higher (up to 60 L/min) than that delivered traditionally in LFOS (up

to 16L/min). Oxygen flow only shows total gas flow, does not show FiO_2. FiO_2 delivered by an oxygen flow is determined by the apparatus and the patients. So it can be also got high FiO2 by using LFOS such as mask plus reservoir bag.

The high gas inlet flow prevents secondary room-air entrainments. So, all of the patient's respiratory requirements can be met by HFOS. The air is warmed to 37 °C and humidified. Humidification prevents mucus plugging. Mucociliary function remains well. The warmed and humidified oxygen can be better tolerated by the patient, so patient comfort increases.

High flow washes out CO_2 in anatomic dead space; create high FiO_2 in the anatomic reservoir (nasal cavity, nasopharynx and oropharynx). The warm, humidified constant flow improves gas exchange and reduces respiratory work. PEEP can be generated by HFOS. It is obtained 1 cmH_2O PEEP for every 10 L/min of flow delivered by HFOS. This may help to prevent and/or decrease atelectasis [1].

HFOS serve as an important alternative to noninvasive mechanical ventilation especially in the management of the patients with hypoxemic respiratory failure. Noninvasive mechanical ventilation cannot be used in patients with facial, esophageal or gastric surgery, impaired swallowing and cough. A high flow oxygen system uses special nasal cannula thus HFOS does not interfere with eating and speaking. If the patient receives FiO_2 of greater than 60% and more than 48 hours, the patient may at risk of oxygen toxicity. Positive pressure ventilation improves oxygenation and CO_2 removal by alveolar recruitment, increasing alveolar volume and redistribution of extracellular lung water, So it is reasonable to use positive pressure in the patient on high O_2 like this level (Figure 1) [2].

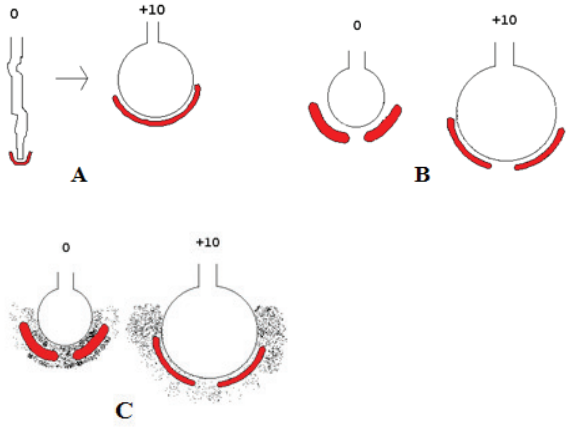


Figure 1. Positive pressure ventilation improves gase exchange. A-Alveolar recruitment, B-Increasing alveolar volume, C-Redistribution of extravascular lung water.

HFOS contains blender connected to the wall outlet, a humidifier, heated tubing and nasal cannula (Figure 2).

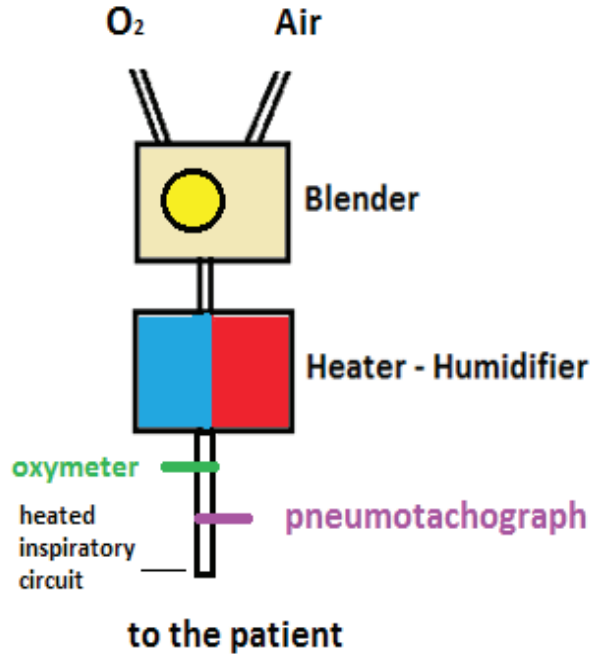


Figure 2. Schematic representation of a High-Flow Oxygen System

Epistaxis, base of skull fracture, surgery to the nose or upper aero-digestive tract, nasal obstruction; e.g. nasal fracture, tenacious secretions, tumor are main contraindications for HFOS therapy.

High-flow oxygen has been shown to result in better comfort and oxygenation than standard oxygen therapy delivered through a face mask in patients with acute respiratory failure of various origins. In acute cardiogenic pulmonary edema, providing low levels of PEEP, HFOS proposed to function as CPAP through opening collapsed alveoli and enhancing cardiac function through afterload reducing effect of PEEP. A cohort study showed promising results [3]. In postoperative hypoxemic respiratory failure HFOS have shown improved oxygenation, reduced respiratory rate and need for noninvasive mechanical ventilation[4-6]. Two randomized trials comparing standard oxygen therapy and HFOS have shown a better patient comfort but no significant difference in gas exchange in post-extubation patients [7, 8]. Two retrospective analysis of HFOS in Do-Not-Intubate patients with cancer and respiratory distress have shown that nearly 85% of patients

treated with HFOS improved or remained stable but 18% of patients still needed escalation of therapy to NIMV [9, 10].

It has been reported that treatment with high-flow oxygen improved the survival rate among patients with acute hypoxemic respiratory failure, as compared with standard oxygen therapy or NIMV. The hazard ratio for death at 90 days was 2.01 (95% confidence interval (CI), 1.01 to 3.99) with standard oxygen versus high-flow oxygen (p=0.046) and 2.50 (95% CI, 1.31 to 4.78) with NIMV versus high-flow oxygen (p=0.006).Intubation rates were not significantly different among three groups (38% in the high flow O_2 group, 47% in the low flow O_2 group and 50% in the NIMV group [11].A prospective trial in patients with hypoxemic respiratory failure, HFOS applied sequentially with non-invasive mechanical ventilation (NIMV) and shown that HFOS improved oxygenation but not as much as NIMV compared to standard oxygen therapy [12].

In conclusion, HFOS have become standard care in several clinical situations for infants, children and preterm neonates. It has a number of physiologic benefits over conventional therapy such as greater comfort and tolerance, improved oxygenation. So it is gaining clinical use in adult population.

Therapeutic hyperthermia

Therapeutic hypothermia after cardiac arrest

Cardiac arrest is a catastrophic, life threatening event. Survival and neurologic recovery vary widely. Fortunately over the last decade there are improvements in survival and neurologic outcomes due to increased education and awareness of basic cardiac life support and may be more importantly concept of post-resuscitation care.

Post-resuscitation care consists of optimization of oxygenation and ventilation, avoiding hypotension, treating immediate precipitants of cardiac arrest such as acute coronary ischemia and initiating therapeutic (induced) hypothermia (TT or IT). Current guidelines uses the term "Targeted Temperature Management" (TTM) [13-15].

During cardiac arrest, hypoxia and eventually anoxia develops. In brain tissue, within minutes ATP and glucose are depleted and cells lose their integrity. This results in apoptosis. Restoration of oxygenation as a result of successful resuscitation stops anoxic injury but death form reperfusion injury occurs. Reperfusion injury is a result of increased reactive oxygen species and with other inflammatory cascades further aggravates endothelial and vasomotor dysfunction, edema, hypoxia at tissue-level in spite of adequate arterial oxygenation, and sequential neurological damage. Hypothermia slowdown inflammatory cascade and inhibits apoptosis pathways by downregulating excitatory amino acids and free radicals. Also, hypothermia decreases cerebral metabolic rate, blood volume, and intracranial pressure [16].

The Hypothermia After Cardiac Arrest Study Group have shown that in patients who have been successfully resuscitated after cardiac arrest due to ventricular fibrillation, therapeutic mild hypothermia increased the rate of a favorable neurologic outcome and reduced mortality. 55 % of patients had better neurologic outcome and mortality at six months was 41 % in the hypothermia group as compared with 55 % in the normothermia group (RR: 0.74; 95 % CI: 0.58 - 0.95) [17]. Also another trial on comatose survivors of out-of-hospital cardiac arrest suggests that treatment with moderate hypothermia appears to improve outcomes in patients. 49 % patients treated with hypothermia survived and had a good outcome as compared with 26 % treated with normothermia (p=0.046). The odds ratio for a good outcome with hypothermia as compared with normothermia was 5.25 (95 % CI: 1.47 - 18.76; P=0.011) [18].

On the basis of these 2 studies, TH/IH/TTM is now considered standard of care in the treatment of patients successfully resuscitated from a ventricular tachycardia (VT/VF) arrest and is recommended as a reasonable option for patients with CA from a non-shockable rhythm.

Current practice guideline recommendations of The American Heart Association, the International Liaison Committee of Resuscitation, and the European Resuscitation Council are summarized in Table II.

Therapeutic hypothermia after traumatic brain injury

Fever aggravates outcome after stroke and head injury, probably by worsening secondary brain injury [19]. TH has become an experimental treatment for traumatic brain injury due to its intracranial pressure reducing and neuroprotective effect [20]. A systematic review of TH following traumatic brain injury has indicated a small decrease in mortality (RR 0.76, 95% CI 0.60-0.97) or poor neurologic outcome (RR 0.69, 95% CI 0.55-0.86) [21]. Later reviews found parallel results for death and neurologic outcome as well as an increased risk for pneumonia [20, 22-26].

Table II. Summary of Practice Guideline Recommendations for Therapeutic Hypothermia

American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care
• comatose (ie, lack of meaningful response to verbal commands) adult patients with ROSC after cardiac arrest should have TTM (Class I, LOE B-R for VF/pVT OHCA; Class I, LOE C-EO for non-VF/pVT (ie, "nonshockable") and in-hospital cardiac arrest).
• selecting and maintaining a constant temperature between 32°C and 36°C during TTM (Class I, LOE B-R)
• maintained for at least 24 hours after achieving target temperature (Class IIa, LOE C-EO)
• We recommend ***against*** the routine prehospital cooling of patients after ROSC with rapid infusion of cold intravenous fluids (Class III: No Benefit, LOE A)
• reasonable to actively prevent fever in comatose patients after TTM (Class IIb, LOE C-LD).
European Resuscitation Council Guidelines for Resuscitation
• TTM is recommended for adults after OHCA with an initial shockable rhythm who remain unresponsive after ROSC (strong recommendation, low-quality evidence)
• TTM is suggested for adults after OHCA with an initial non-shockable rhythm who remain unresponsive after ROSC (weak recommendation, very low-quality evidence).
• TTM is suggested for adults after IHCA with any initial rhythm who remain unresponsive after ROSC (weak recommendation, very low-quality evidence)
• Maintain a constant, target temperature between 32 ^{0}C and 36 ^{0}C for those patients in whom temperature control is used (strong recommendation, moderate-quality evidence)
• If targeted temperature management is used, it is suggested that the duration is at least 24 h (weak recommendation, very low-quality evidence).
International Liaison Committee on Resuscitation
• We recommend targeted temperature management as opposed to no targeted temperature management for adults with OHCA with an initial shockable rhythm who remain unresponsive after ROSC (strong recommendation, low-quality evidence).
• We suggest targeted temperature management for adults with OHCA with an initial nonshockable rhythm who remain unresponsive after ROSC (weak recommendation, low-quality evidence).
• We suggest targeted temperature management for adults with IHCA with any initial rhythm who remain unresponsive after ROSC (weak recommendation, very low-quality evidence).
• We recommend selecting and maintaining a constant target temperature between 32°C and 36°C for those patients in whom targeted temperature management is used (strong recommendation, moderate-quality evidence).

"ROSC: return of spontaneous circulation; TTM: therapeutic temperature management; VF: ventricular fibrillation; pVT: pulseless ventricular tachycardia; OHCA: Out of Hospital Cardiac Arrest; IHCA: In Hospital Cardiac Arrest; LOE: level of evidence; "

Therapeutic hypothermia in acute liver failure

Cerebral edema is a rare but fatal complication of acute liver failure. Its incidence increases with grade of encephalopathy and nearly 75 % of patients with grade IV encephalopathy have cerebral edema [27]. Cerebral edema leads to the most common causes of death in acute liver failure which are elevated intracranial pressure, brain ischemia, and brainstem herniation [28]. Liver transplantation remains the only definitive treatment option for cerebral edema. Animal models of liver failure have shown that hypothermia decreases cerebral edema [29]. A systematic review of case series has shown that induction of moderate hypothermia may have a benefit but only with intracranial pressure monitoring [30]. However, more recent studies have had variable results [31, 32]. In spite of the potential beneficial effects, hypothermia can also increase the risk of infection, cardiac dysrhythmias and bleeding [33]. Consequently, more studies are needed before TH/IH/TTM can be routinely used. However, it is a reasonable option as a bridge to transplantation as we used previously successfully in our center [34].

TH consists of 4 stages: initiation, maintenance, rewarming and return to normothermia (Figure 3). There are multiple methods to induce and maintain TH [35].

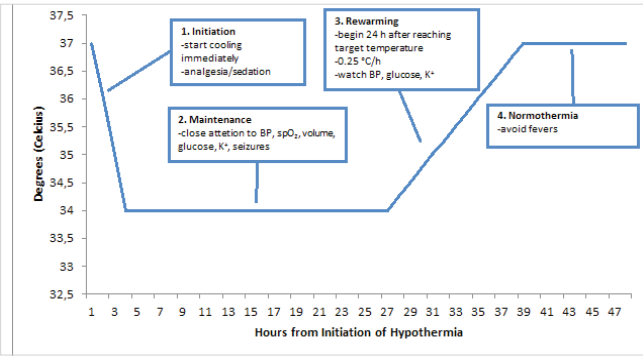


Figure 3. Stages of hypothermia. BP indicates blood pressure; K^+, serum potassium concentrations; spO_2, oxygen saturation.

Conventional Cooling Systems: Ice bags and cold saline infusion are the easiest and cheapest ways to induce hypothermia. They have been shown to be effective in inducing hypothermia but not in maintenance [36]. Also they can be used with other cooling methods [37]. But most important disadvantage of these systems is the uncontrolled cooling which may result in body temperatures below target range.

Surface Cooling Systems: Circulating cold air or cold fluids inside blankets or pads surrounding body is an effective way of cooling. Advantages include ease of use and rapid initiation of treatment. Also these systems have a computer controlled feedback system and allows user to interfere according to trends. Possible disadvantage of these systems is that skin burns and irritations. Shivers also seen more commonly [35].

Internal Cooling Systems: These systems use central venous catheters through which cool or warm saline circulates in a closed loop. By means of heat exchange with blood catheters achieve hypothermia and maintain it precisely. In spite of these advantages, compared to surface cooling there is no significant difference in outcome [38].

Extra Corporeal Membrane Oxygenation (ECMO) and Extra Corporeal Carbon Dioxide Removal (ECCOR)

ECMO has become an essential tool in critical care patients with severe respiratory and cardiac failure, refractory to conventional therapy methods.

The first heart-lung machine was used for cardiovascular surgery in 1953[39]. Soon after successful use of extracorporeal membrane oxygenation (ECMO) outside the operating room was reported in 1972, by Robert Bartlett [40]. This work led to technologic innovations and use of extracorporeal life support (ECLS) in management of patients with severe acute respiratory distress syndrome (ARDS). 2009 to 2010 influenza (H1N1) pandemics resulted in an increasing use of ECLS for cardiopulmonary failure after traditional treatment options have failed [41].

ECMO is a type of cardiopulmonary support, where blood is drained from the vascular system via cannula connected to a mechanical circuit outside the body and then reinfused into the circulation with oxygenation and removal of pCO_2.

Depending on indications there are two types of ECMO with cardiopulmonary (venoarterial VA-ECMO) or pulmonary support (venovenous VV-ECMO). Support can work partially or completely by preference. A typical circuit is composed of cannulas and tubing, a membrane or oxygenator, a blood pump and a heat exchanger. Venous blood drained from a major vein, passes through a pump and a membrane for gas Exchange and then returns a major artery (VA-ECMO) or vein (VVECMO) fully saturated with oxygen through an oxygenator, cleaned from CO_2 via a membrane heated by an exchanger (Figure 4).

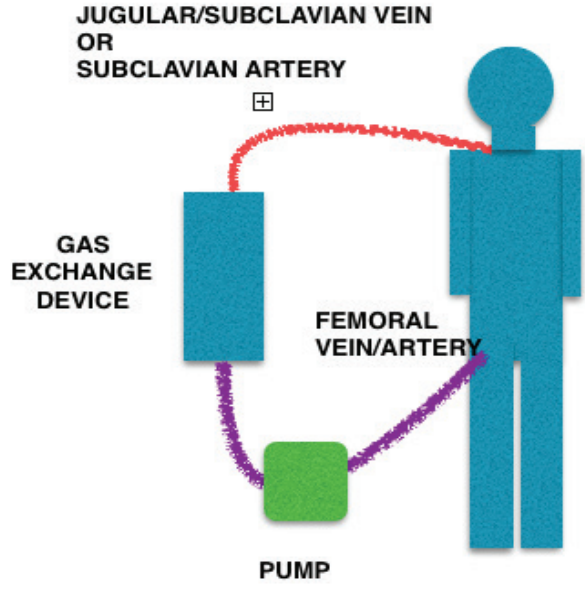


Figure 4. Essential components of extracorporal support systems. Venoarterial systems support heart and lung, venovenous systems support lung during ECMO. ECCOR (venovenous, lower blood flow rates system with or without pump) supports only lung

ECCOR is provided to remove CO_2 and, unlike ECMO, does not provide significant oxygenation. In contrast to ECMO, where the need for oxygenation requires high blood flow rates, ECCOR allows much lower blood flow rates (500-1000mL/min) and needs smaller access cannulas. So, ECCOR promises to improve safety and ease of use. If the patient has sufficient arterial pressure (arteriovenous pressure gradient ≥ 60 mm Hg), a pumpless system may be used in ECCOR. Otherwise, we have to use mechanical pump [42].

Vascular access and management

VA is preferred if cardiac function is depressed and cardiac support is also required. Patients with severe respiratory failure and secondary cardiac failure may improve on VV support alone. VV access may be by femoral and jugular veins with 2 cannulas or a double lumen cannula via only jugular vein [43].

VA access may be central with intrathoracic cannula through ascending aorta in postcardiotomy and percutaneous peripheral with femoral artery to femoral vein or femoral artery to jugular vein in non-postcardiotomy patients [43]. Femoral to femoral access is preferred for VAECMO because insertion is faster and easier in cardiogenic shock patients.

Although depends on the indication and type of ECMO, management based on circuit and patient related factors. Anticoagulation is needed, with a target value ACT or aPTT, to maintain the blood flow in circuit. Hemodynamics is important especially in VV-ECMO, inotropes, vasodilators; blood volume replacement may be needed. Lung protective strategies are used with minimizing support of mechanical ventilator to achieve lung recovery. Minimal to heavy sedation is required according to the stage of the procedure. Positioning and activity, infection control, renal replacement, nutrition and temperature management require special attention.

ECMO is a bridge to; recovery till the resolution of underlying reversible cause (for example; resolve of thrombus in massive pulmonary embolism with severe hypoxemia), implantable circulatory support ventricular assist device **(**VAD**)** or transplantation of heart or lung (Table III).

Table III. Indications and contraindications for VA-ECMO

Indications vor VA-ECMO	Contraindications for VA-ECMO
1- Inadequate tissue perfusion manifested as htpotension and low cardiac output despite adequate vascular volume	Absolute
2- Shock persists despite volume administration, inotropes and vasoconstrictors and intraaortic balloon counterpulsation if appropriate	1- Prolonged CPR without adequate tissue perfusion
3- Typical causes: Acute Myocardial infarction, myocarditis, peripartum cardiomyopathy, decompensated chronic heart failure, post cardiotomy shock.	2- Unrecoverable heart and not a candidate for transplant or VAD
4- Septic Shock is considered as an indication in some centers.	3- Advanced age
5- Biventricular failure, refractory malignant arrhythmias, heart failure with severe pulmonary failure	4- Chronic organ dysfunction (emphysema, cirrhosis, renal failure)
	5- Compliance (financial, cognitive, psychiatric, or social limitations)
	Relative
	1- Contraindication for anticoagulation
	2- Advanced age
	3- Obesity

There is no absolute contraindication. Each cause depends on every patient individually with respect to risks and benefits. However, conditions associated with poor outcome despite ECMO can be considered as contraindications (Table IV).

Table IV. Indications and contraindications for VV-ECMO

Indications for VV-ECMO	Contraindications for VV-ECMO
1- In hypoxic respiratory failure due to any cause ECLS should be considered when risk of mortality is 50% or greater and is indicatedwhen the risk of mortality is 80% or greater	1- Mechanical ventilation at high settings (FiO_2 > .9, Pplat>30 for 7 days or more.
a- 50% mortality risk is associated with a PaO_2/FiO_2 <150 0n FiO2 > 90% and/or Murray shore 2-3	2- Major pjarmacologic immunosuppression (absolute neutrophil count <400/mm^3)
b- 80% mortality risk is associated with a PaO_2/FiO_2 < 100 on FiO2 >90% and/or Murray score 3-4 despite optimal care for 6 hours or more.	3- CNS hemorrhage that is recent or expanding
2- CO_2 retention on mechanic ventilation despite high Pplat. (>30 cm H_2O)	4- Non-recoverable co-morbidity such as major CNS damage or terminal malignancy
3- Severe airleak syndromes	5- Age: not specifically
4- Need for intubation in a patient on lung transplant list	
5- Immediate cardiac or respiratory collapse PE, Blocked airway, Unresponsive to optimal care	

Complications of ECMO

Problems could be related to the underlining disease or ECMO mechanics itself. The most common complication is hemorrhage [44]. Surgery required bleeding was reported up to 34% with VA-ECMO and 17% with VV-ECMO [45]. Mainly anticoagulation and hemodilution rarely catheter malposition cause bleeding [46].Bleeding may occur at cannula side as well as any side of the body and may increase mortality. Mostly pulmonary or intracerebral hemorrhage was reported thorough ARDS patients on ECMO [47]. Treatment requires stopping anticoagulants and infusion of platelets and clotting factors [46]. Hemolysis of blood in the circuit, thrombus or clot formation and systemic thromboembolism and cerebral infarction were also reported in the literature [48, 49].Catheter and circuit related infections and septic complications may be higher through patients on ECMO. Increased volume of distribution and filtering effect of membranous part of the circuit may alter serum concentration of drugs. Dose alterations may be necessary when narrow therapeutic drugs are administered [50].

Weaning from ECMO

ECMO support is decreased together with the recovery of underlying disease. When gas flow is minimum at lowest ventilatory settings with improving organ function, weaning protocol is started. As a rule weaning is started at levels less than 30% of total support. Ventilatory settings and vasopressor-inotropes are adjusted support is interrupted for a while and patient is checked for stability. This procedure is called as 'trial off'. After a successful trial off, anticoagulant therapy is stopped and decannulation should be done. If patient seems to need ECMO further again, cannulas can be left 24 hours or more. Decannulation must performed after a successful trial off [43].

Prognosis

According to data reported by ELSO in January 2015, 65.171patients received ECLS, of total 71% were weaned and59% were discharged or transferred [50]. Underlying cause for ECMO has an importance on survival. In a study, 67%ofARDS patients were reported to be treated successfully with ECMO and survival to hospital discharge was52 % [51]. CESAR study demonstrated that referral to an ECMO center significantly improves recovery and survival from severe ARDS [52]. H1N1-relatedARDS was shown to have lower hospital mortality when referred to an ECMO center (23.7% *vs.*52.5%) [53]. It has been shown that ECMO performed cardiopulmonary resuscitation was associated with increased survival with minimal neurologic impairment compared to conventional method [54].

In conclusion, experience with ECMO has been

improving with better survival rates and decreasing morbidity. Considering ECMO for patients with respiratory failure or heart failure when conventional therapies failed, if the underlying pathology is reversible or as a bridge to heart and/or lung transplantation is important.

Ultrasound in critical care

Advances in technology have led ultrasound (US) use through non-radiologist physicians at bedside. A non-invasive, dynamic-real time tool that has no radiation risk may be valuable for critically ill for whom timely, efficient bedside diagnostics are essential.

Critical care ultrasonography (CCUS) has utility for intensivist-performed, immediate diagnoses of life threatening diseases, with no need to transport patients to radiology departments or wait for radiologist or cardiologist on a consultative basis. Because of the principle of focused assessments, it differs from traditional US examinations where time-consuming full regional scans are performed.

Additionally, the intensivist can repeat the examination to evaluate the effects of therapy and direct the management of the critical condition. Goal-directed and repeated US allows Intensive Care Unit (ICU) physician to diagnose and monitor the condition of the patient on a methodical basis.

Components of critical care ultrasonography

The American College of Chest Physicians/La Socie´te´ de Re´animation de Langue Franc¸aise Statement on Competence in Critical Care Ultrasonography (ACCP/SRLF Statement) defines 5 modules of CCUS: Cardiac: Basic and Advanced Levels, Thoracic: Lung and Pleura, Abdominal, Vascular Access and Vascular Diagnostic: Examination for Deep Venous Thrombosis(DVT) [55]

According to American Committee on Graduate Medical Education; US is a mandatory part of critical care fellowship training in the United States as from July 1, 2014 [56].

Consequently, CCUS and echocardiography should be an essential part of training of every ICU physician.

Thoracic ultrasonography

A recent field of CCUS is examination of the lung and pleura. Portability, absence of radiation, ability to repeat at bedside make lung US more applicable than chest x-ray or computed tomography (CT).

A variety of pathologies like pleural effusion, pneumothorax, atelectasis, pneumonia and alveolar-interstitial syndrome can be detected easily and quickly by sonographic examination of thorax. Compared with chest x-ray, US has high sensitivity and specificity rates for detection of pleural [57, 58] and parenchymal [59-62] diseases of lungs. (Table V) (Figures 5, 6, 7, 8, 9).

Table V. Sensitivity and specificity of US compared with chest x-ray

	Chest X ray (%) sensitivity	specificity	US(%) sensitivity	specificity
Pleural effusion	39	85	92	93
Pneumothorax	50	99	100	98
Alveolar-interstitial syndrome	60	100	98	88
Alveolar consolidation	68	95	93	100

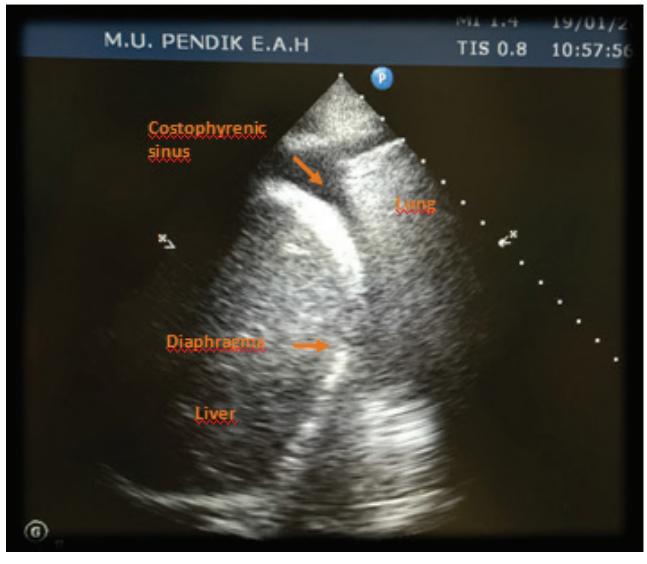


Figure 5. Normal lung US

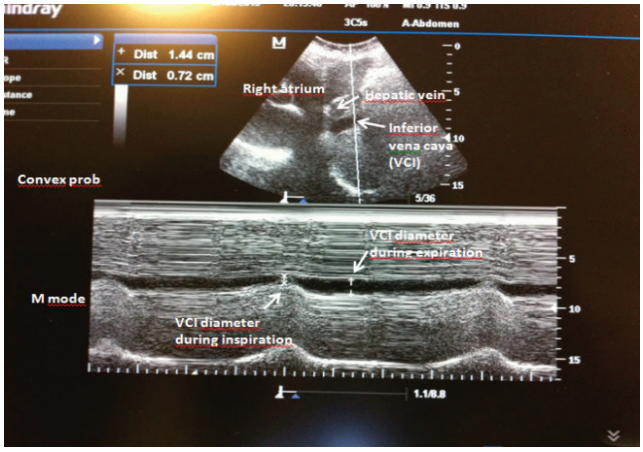


Figure 6. US assessment of VCI diameter. Collapse (decrease in VCI diameter more than 50%) with inspiration shows fluid responsiveness

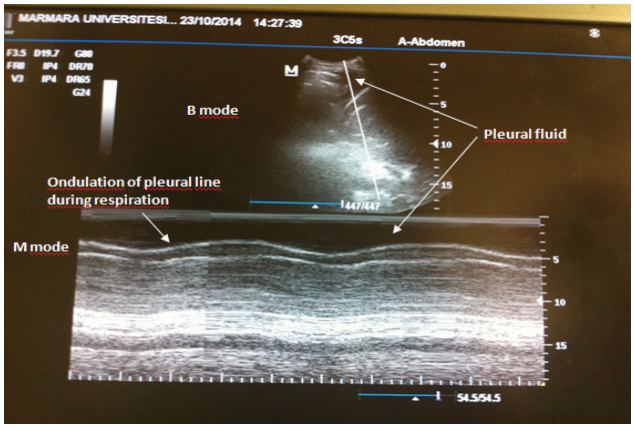


Figure 7. Pleural fluid seen in black. Also ondulations of pleural line during respiration in M mode on US are seen.

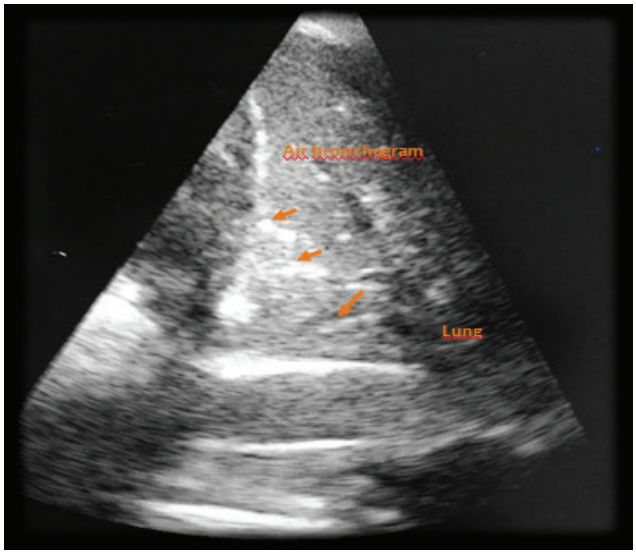
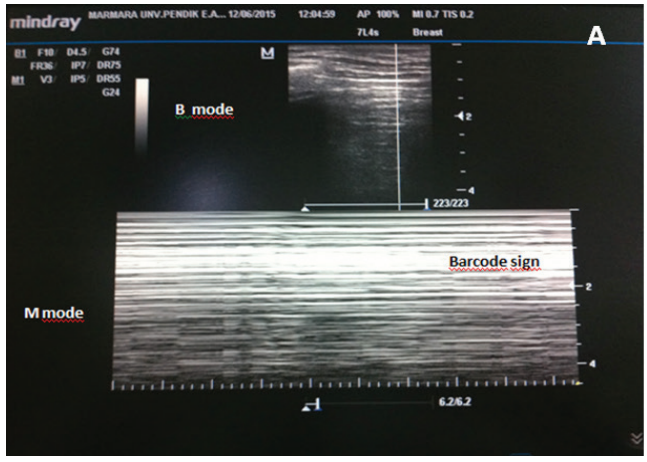


Figure 8. Air bronchogram is shown as white lines/dots on US in a patient with pneumonia



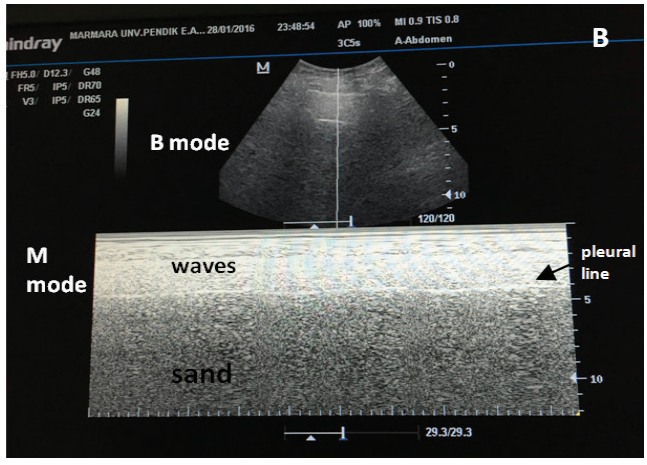


Figure 9. A-Pneumothorax. Parallel horizontal lines above and below the pleural line, resemble a 'barcode.(Barcode signs, stratosphere sign) B-Normal lung in B mode and in M mode, M-mode illustrating the 'seashore sign.' The pleural line divides the image in half: The motionless portion above the pleural line creates horizontal 'waves,' and the sliding line below it creates granular pattern, the 'sand'seeshore sign.

On the other hand, there are some disadvantages of performing thoracic US. It is notably that, almost all of the pathologies detected on US are superficial. When it comes to deeper parenchymal pathologies, US may be an inadequate tool compared with CT. Since results are operator dependent, different diagnoses between physicians may be confusing. This disadvantage itself reflects the importance of an adequate, standard and supervised training program for ICU physicians.

Abdominal ultrasound

Abdominal free fluid, causes of post-renal acute renal failure and however rarely, pathologies of retroperitoneum and major abdominal vessels can be detected early by focused

US assessments, especially in hemodynamically unstable critically ill. It has been shown in the critically ill that renal vasoconstriction can reduce renal blood flow to 50% of normal [63]. Daily monitoring renal perfusion via Doppler US in shock is a recent, ongoing research.

Transcranial Doppler (TCD)

Cerebral blood supply can be calculated using the Doppler US to support the diagnosis of brain death, vasospasm or ischemic stroke. TCD also helps to monitor the effects of therapeutic intervention of intracranial pathologies. It may help to diagnose brain death with a sensitivity of 76% and specificity of 74.3 % [64].

Ocular US

A direct relation has been documented between the subarachnoid space of the optic nerve and the chiasmal cistern of the brain. When ICP increases, cerebrospinal fluid flows towards the perineural subarachnoid space and results in expansion of the optic nerve sheath diameter (ONSD). Measurement of ONSD is useful to monitor intracranial pressure (ICP). A diameter of >5 mm has been shown to correlate with increased ICP shown on CT with a sensitivity of 100% and specificity 95%, respectively (Figure 10) [65].

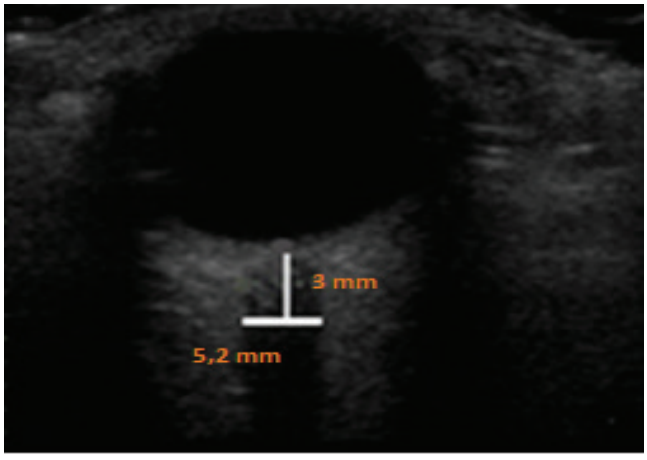


Figure 10. Optic Nerve Sheath Diameter of 5,2 cm in a 56 year-old male with intracranial hemorrhage (N<5 mm)

Other uses of critical ultrasound ara başlık

Additionally US helps to detect; DVT, muscle wasting due to critical illness, edema of tracheal wall with smoke inhalation, position of endotracheal tube, gastric volume assessment in patients with nasoduodenal enteral feeding in critically ill.

It also can be used to guide vascular access, thoracentesis, pericardiocentesis, paracentesis, percutaneous tracheostomy and regional anesthesia.

Critical care echocardiography

According to the ACCP/SRLF Statement, critical care echocardiography (CCE) can be performed on two levels methodically: basic and advanced [55]. While basic CCE can be learned in a relatively short training period with a limited number of transthoracic echocardiography (TTE) views, advanced CCE requires an improved skill level in both TTE and transesophageal echocardiography (TEE) and a much longer duration of training.

Reversible causes of shock or cardiac like cardiac tamponade, left ventricular failure, ventricular wall motion abnormalities, massive pulmonary embolism, infective endocarditis, intracardiac thrombi or aortic dissections can be detected immediately via ‘’ basic’’ or “focused” or “goal-directed” echocardiography, in critically ill patients. (Figure 1) Additionally, fluid responsiveness of patients on admission can easily be determined in shock states. Goal-directed echocardiography has been proven to, not only improve clinical diagnosis and but also management of acute critical illness (Table VI) [66].

Table VI. Basic TTE education

Pericardial space: Assessment of pericardial effusion and tamponade
Left ventricular (LV) and right ventricular (RV) dimensions and function:
- Cardiac pump failure
- Valvular failure
- Wall motion abnormalities with acute coronary syndrome
Acute cor pulmonale with massive pulmonary embolism
Severe hypovolemia with end systolic effacement of LV
Inferior vena cava (IVC) diameter: Assessment of fluid responsiveness and volume status.
Appropriate initial management strategy for shock: Need for vasopressors, inotropes, fluids, thrombolytics
Response to therapy: Effects of fluid resuscitation or thrombolytics on ventricular function.
Setting levels of positive end expiratory pressure according to RV systolic functions on mechanically ventilated

A detailed assessment of stroke volume (SV),pulmonary pressures, left atrial pressure, valvular function and the great vessels (aorta and pulmonary artery) requires an advanced examination and recognition of an abnormal situation should prompt a detailed assessment by a cardiologist.

Since noninvasive estimation of LV filling pressures with echocardiography is possible, invasive placement of pulmonary artery catheter has decreased. A Doppler index, named E/Ea correlates significantly with invasively derived mean pulmonary capillary wedge pressure. Generally, an E/Ea>10 is predictive of a mean pulmonary capillary wedge pressure above 15 mmHg with a sensitivity and specificity of 92 and 80 percent, respectively [67].

Diameter of VCI may help to have an idea about volume status of critically ill. Although it does not accurately reflect central venous pressure; respiratory variability IVC diameter has been described as a noninvasive method for determining fluid responsiveness [68] (Figure 2) For example, If the VCI diameter is less than 2.1 cm and there is 50% collapse during respiration, it means that the patient has low volume status.

In conclusion, portable, repeatable, relatively inexpensive, painless, x-ray free and safe US examination is a practical approach to diagnose, manage and follow up of the critical illness. Sonographic examination should be a part of daily routine work of intensivists to improve morbidity and survival rates.

References

1. Spoletini G, Alotaibi M, Blasi F, Hill NS. Heated humidified high-flow nasal oxygen in adults: Mechanisms of action and clinical implications. Chest 2015;148: 253-61.
2. Ward JJ. High-flow oxygen administration by nasal cannula for adult and perinatal patients. Respir Care 2013; 58: 98-122.
3. Carratala Perales J M, Llorens P, Brouzet B, et al. High-Flow therapy via nasal cannula in acute heart failure. Rev Esp Cardiol 2011; 64:723-5.
4. Corley A, Caruana LR, Barnett AG, Tronstad O, Fraser JF. Oxygen delivery through high-flow nasal cannulae increase end-expiratory lung volume and reduce respiratory rate in post-cardiac surgical patients. Br J Anaesth 2011;107:998-1004.
5. Parke R, McGuinness S, Dixon R, Jull A. Open-label, phase II study of routine *high*-flow nasal oxygen therapy in cardiac surgical patients. Br J Anaesth 2013,111:925-31.
6. Parke R L, McGuinness SP, Eccleston ML. A preliminary randomized controlled trial to assess effectiveness of nasal high-flow oxygen in intensive care patients. Respir Care 2011;56:265-70.
7. Rittayamai N, Tscheikuna J, Rujiwit P. High-flow nasal cannula versus conventional oxygen therapy after endotracheal extubation: a randomized crossover physiologic study. Respir Care 2014;59:485-90.
8. Tiruvoipati R, Lewis D, Haji K, Botha J. High-flow nasal oxygen vs high-flow face mask: a randomized crossover trial in extubated patients. J Crit Care 2010;25:463-8.
9. Epstein A S, Hartridge-Lambert SK, Ramaker JS, Voigt LP, Portlock CS. Humidified high-flow nasal oxygen utilization in patients with cancer at Memorial Sloan-Kettering Cancer Center. J Palliat Med 2011;14: 835-9.
10. Peters S G, Holets S R, Gay PC. High-flow nasal cannula therapy in do-not-intubate patients with hypoxemic respiratory distress. Respir Care 2013;58: 597-600.
11. Frat J P, Thille A W, Mercat A, et al. High-flow oxygen through nasal cannula in acute hypoxemic respiratory failure. N Engl J Med 2015;372:2185-96.
12. Frat J P, Brugiere B, Ragot S, et al. Sequential application of oxygen therapy via high-flow nasal cannula and noninvasive ventilation in acute respiratory failure: an observational pilot study. Respir Care 2015;60:170-8.
13. Callaway C W, Donnino M W, Fink E L, et al. Part 8: Post-cardiac arrest care: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2015;132(18 Suppl 2): S465-82.
14. Donnino M W, Anderson L W, Berg K M, et al. Temperature management after cardiac arrest: An Advisory Statement by the Advanced Life Support Task Force of the International Liaison Committee on Resuscitation and the American Heart Association Emergency Cardiovascular Care Committee and the Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation. Resuscitation 2016;98: 97-104.
15. Nolan JP, Soar J, Cariou A, et al. European Resuscitation Council and European Society of Intensive Care Medicine Guidelines for Post-resuscitation Care 2015: Section 5 of the European Resuscitation Council Guidelines for Resuscitation 2015. Resuscitation, 2015;95:202-22.
16. Polderman KH. Mechanisms of action, physiological effects, and complications of hypothermia. Crit Care Med 2009;37(7 Suppl): S186-202.
17. The Hypothermia after Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med 2002;346:549-56.
18. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med 2002;346:557-63.
19. Andrews PJ, Sleepman DH, Statham PF, et al., Predicting recovery in patients suffering from traumatic brain injury by using admission variables and physiological data: a comparison between decision tree analysis and logistic regression. J Neurosurg 2002;97:326-36.
20. Safar P, Kochanek PM. Lack of effect of induction of hypothermia after acute brain injury. N Engl J Med 2001;345: 66.
21. Sydenham E, Roberts I, Alderson P. Hypothermia for traumatic head injury. Cochrane Database Syst Rev 2009;(2): CD001048.
22. McIntyre LA, Fergusson DA, Hébert PC, Moher D, Hutchison

JS. Prolonged therapeutic hypothermia after traumatic brain injury in adults: a systematic review. JAMA 2003;289: 2992-9.
23. Henderson WR, Dhingra VH, Chittock DR, Fenwick JC, Ronco JJ. Hypothermia in the management of traumatic brain injury. A systematic review and meta-analysis. Intensive Care Med 2003;29:1637-44.
24. Harris OA, Colford JM, Good MC, Matz MG. The role of hypothermia in the management of severe brain injury: a meta-analysis. Arch Neurol 2002,59: 1077-83.
25. Georgiou AP, Manara AR. Role of therapeutic hypothermia in improving outcome after traumatic brain injury: a systematic review. Br J Anaesth 2013,110:357-67.
26. Crossley S, Reid J, McLatchie R, et al. A systematic review of therapeutic hypothermia for adult patients following traumatic brain injury. Crit Care 2014;18: R75.
27. Lee W M. Acute liver failure. N Engl J Med 1993; 329:1862-72.
28. Stravitz R T. Critical management decisions in patients with acute liver failure. Chest 2008;134: 1092-102.
29. Traber P, Dalcanto M, Ganger D, Blei AT. Effect of body temperature on brain edema and encephalopathy in the rat after hepatic devascularization. Gastroenterology 1989;96: 885-91.
30. Dmello D, Cruz-Flores S, Matuschak GM. Moderate hypothermia with intracranial pressure monitoring as a therapeutic paradigm for the management of acute liver failure: a systematic review. Intensive Care Med 2010;36:210-3.
31. Karvellas CJ, Todd Stravitz, R, Battenhouse M, et al. Therapeutic hypothermia in acute liver failure: a multicenter retrospective cohort analysis. Liver Transpl 2015;21: 4-12.
32. Jalan R, Olde Damink SW, Deutz NE, Hayes PC, Lee A. Moderate hypothermia in patients with acute liver failure and uncontrolled intracranial hypertension. Gastroenterology 2004;127:1338-46.
33. Stravitz R T, Larsen F S. Therapeutic hypothermia for acute liver failure. Crit Care Med 2009; 37(7 Suppl): S258-64.
34. Yalcin, A., et al., Therapeutic hypothermia a treatment choice for cerebral oedema in acute liver failure report of two cases, in 24th Scientific Meeting of the Asian Pacific Association for the Study of the Liver (APASL)2015: Istanbul, Turkey.
35. Vaity C, Al-Subaie N, Cecconi M. Cooling techniques for targeted temperature management post-cardiac arrest. Crit Care 2015;19:103.
36. Kliegel A, Janata A, Wandaller C, et al. Cold infusions alone are effective for induction of therapeutic hypothermia but do not keep patients cool after cardiac arrest. Resuscitation 2007;73:46-53.
37. Kliegel, A., Losert H, Sterz F, et al. Cold simple intravenous infusions preceding special endovascular cooling for faster induction of mild hypothermia after cardiac arrest--a feasibility study. Resuscitation 2005;64:347-51.
38. Gillies MA, Pratt R, Whiteley J, Borg J, Beale RJ. Therapeutic hypothermia after cardiac arrest: a retrospective comparison of surface and endovascular cooling techniques. Resuscitation 2010;81: 1117-22.
39. Cohn L H. Fifty years of open-heart surgery. Circulation 2003,107: 2168-70.
40. Bartlett, R.H., Gazzanika AB, Thomasian J, et al., Extracorporeal membrane oxygenator support for cardiopulmonary failure. Experience in 28 cases. J Thorac Cardiovasc Surg 1977,73:375-86.
41. Ventetuolo C E, Muratore C S. Extracorporeal life support in critically ill adults. Am J Respir Crit Care Med 2014;190:497-508.
42. Cove ME, MacLaren G, Federspiel WJ, Kellum JA. Bench to bedside review*:* Extracorporeal carbon dioxide removal, past present and future. Crit Care 2012;16:232.
43. Extracorporeal Life Support Organization. (ELSO), E.L.S.O., ELSO Adult Cardiac Failure Supplement to the ELSO General Guidelines, 2013.
44. Bartlett R H, Gattinoni L. Current status of extracorporeal life support (ECMO) f*or* cardiopulmonary failure. Minerva Anestesiol 2010;76:534-40.
45. Aubron C, Cheng AC, Pilcher D, et al. Factors associated with outcomes of patients on extracorporeal membrane oxygenation support: a 5-year cohort study. Crit Car 2013;17: R73.
46. Makdisi G, Wang I W. Extra Corporeal Membrane Oxygenation (ECMO) review of a lifesaving technology. J Thorac Dis 2015;7: E166-E176.
47. Wittenstein B, Ng C, Ravn H, Goldman A. Recombinant factor VII for severe bleeding during extracorporeal membrane oxygenation following open heart surgery. Pediatr Crit Care Med 2005;6:473-6.
48. Lidegran MK, Mosskin M, Ringertz HG, Frenckner BP, Lindén VB. Cranial CT for diagnosis of intracranial complications in adult and pediatric patients during ECMO: Clinical benefits in diagnosis and treatment. Acad Radiol 2007;14:62-71.
49. Madershahian N, Nagib R, Wippermann J, Wahlers T. A simple technique of distal limb perfusion during prolonged femoro-femoral cannulation. J Card Surg 2006;21:168-9.
50. Extracorporeal Life Support Organization. (ELSO), E.L.S.O., *ECLS Registry Report*. 2015.
51. Hemmila MR, Rowe SA, Boules TN, et al. Extracorporeal life support for severe acute respiratory distress syndrome in adults. Ann Surg 2004,240: 595-605; discussion 605-7.
52. Peek GJ, Mugford M, Tiruvoipati R, et al. Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial. Lancet 2009;374:1351-63.
53. Ramanathan K, Cove ME, Caleb MG, Teoh KL, Maclaren G. et al. Ethical dilemmas of adult ECMO: emerging conceptual challenges. J Cardiothorac Vasc Anesth 2015;29:229-33.
54. Chen YS, Lin JW, Yu HY, et al. Cardiopulmonary resuscitation with assisted extracorporeal life-support versus conventional cardiopulmonary resuscitation in adults with in-hospital cardiac arrest: an observational study and propensity analysis. Lancet 2008;372: 554-61.
55. Mayo PH, Beaulieu Y, Doelken P, et al. American College of Chest Physicians/La Societe de Reanimation de Langue Francaise statement on competence in critical care ultrasonography. Chest 2009;135:1050-60.

56. (ACGME), A.C.G.M.E. Program Requirements for Graduate Medical Education in Critical Care Medicine, 2014.
57. Gryminski J, Krakowka P, Lypacewicz G. The diagnosis of pleural effusion by ultrasonic and radiologic techniques. Chest 1976;70:33-7.
58. Yu CJ, Yang PC, Wu HD, Chang DB, Kuo SH, Luh KT. Ultrasound study in unilateral hemithorax opacification. Image comparison with computed tomography. Am Rev Respir Dis 1993;147:430-4.
59. Lichtenstein D, Meziere G. A lung ultrasound sign allowing bedside distinction between pulmonary edema and COPD: the comet-tail artifact. Intensive Care Med 1998;24:1331-4.
60. Lichtenstein D, Méziére G, Biderman P, Gepner A. The comet-tail artifact: an ultrasound sign ruling out pneumothorax. Intensive Care Med 1999;25:383-8.
61. Lichtenstein D A, Loubieres Y. Lung sonography in pulmonary embolism. Chest 2003;123:2154: author reply 2154-5.
62. Lichtenstein D A, Meziere G A. Relevance of lung ultrasound in the diagnosis of acute respiratory failure: the BLUE protocol. Chest 2008,134:117-25.
63. Barozzi L, Valentino M, Santoro A, Mancini E, Pavlica P. Renal ultrasonography in critically ill patients. Crit Care Med 2007.;35(5 Suppl): S198-205.
64. Chang JJ, Tsivgoulis G, Katsanos AH, Malkoff MD, Alexandrov AV. Diagnostic accuracy of transcranial Doppler for brain death confirmation:Systematic review and meta-analysis. AJNR Am J Neuroradiol 2016;37:408-14.
65. Blaivas M, Theodoro D, Sierzenski P R. Elevated intracranial pressure detected by bedside emergency ultrasonography of the optic nerve sheath. Acad Emerg Med 2003,10:376-81.
66. Lau G, Swanevelder J. Echocardiography in intensive care--where we are heading? Anaesthesia 2011;66:649-52.
67. Ommen SR, Nishimura RA, Appleton CP, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: A comparative simultaneous Doppler-catheterization study. Circulation 2000;102: 1788-94.
68. Mohsenin V. Assessment of preload and fluid responsiveness in intensive care unit. How good are we? J Crit Care 2015;30: 567-73.