Πρόγραμμα Προκαταρκτικής Κλινικής Εκπαίδευσης (ΠΚΕ)

Αναζωογόνηση (Υπόταση-Διαχείριση Αεραγωγού)

Εφη Πολυζωγοπούλου

Επίκουρη Καθηγήτρια Επείγουσας Ιατρικής ΕΚΠΑ

Πανεπιστημιακή Κλινική Επειγόντων Περιστατικών ΠΓΝ ΑΤΤΙΚΟΝ

Πρόεδρος Τομέα Επείγουσας Υπερηχογραφίας της Ευρωπαικής Εταιρείας Επείγουσας Ιατρικής

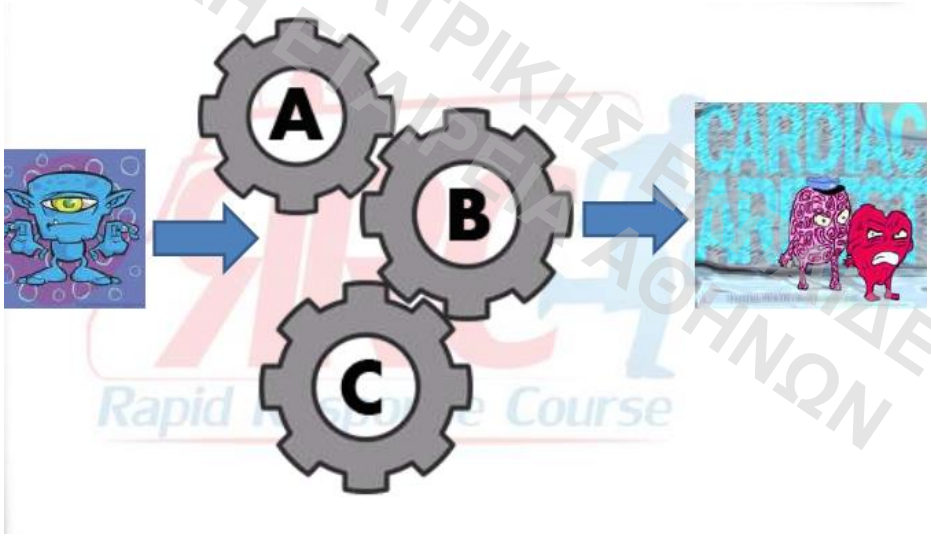
Εκπαιδευτικά Αντικείμενα-Ερωτήματα

- Αναγνώριση βαρέως πάσχοντος
- Βασικές αρχές προσέγγισης βαρέως πάσχοντος
- Βασικές αρχές αναζωογόνησης βαρέως πάσχοντος

Who is the critically ill patient?

At high risk or

with potentially life-threatening condition



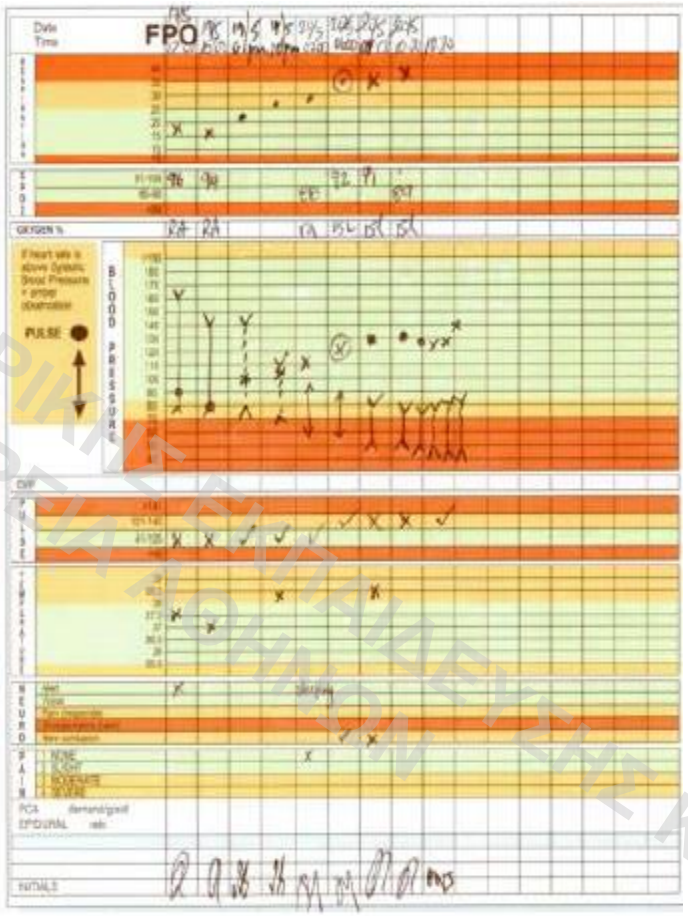
Why early recognition of the critically ill patient is so important?

Early recognition prevents:

- Cardiac arrests and deaths
- Admissions to ICU
- Inappropriate resuscitation attempts

Why early recognition of the critically ill patient is so important?

- Most arrests are **predictable**
- Deterioration prior to **50 - 80%** of cardiac arrests
- **Hypoxia** and **hypotension** are common antecedents
- Delays in **referral** to higher levels of care



Why early recognition of the critically ill patient is so important?

50% had serious vital sign abnormalities (abnormal BP, RR, HR) documented in the 8 hours before death

Internal Medicine Journal2001; 31: 343–348

even when there were documented physiological abnormalities and the criteria for calling the MET were met, the team was called for only 30% of patients prior to their unplanned admission to ICU

Lancet. 2005;365:2091-97

How to recognize the critically ill?

- Initial assessment
- Monitoring
- Initial diagnostic tests
- Management

Who is critically ill?

Case 1

65 yo

Dyspnea-chest pain (PMH: ICD)

BP: 85/58 mmHg

RR 28/min

SpO2: 85% (3 lt/min)

HR 111/min

T: 37.8^{o}C

P/E: NIL

ECG: sinus tachy

Lactate: 5 mmol/L (<2.0)

TnI:2.5 ng/ml (<0.7)

WBC: 13x10^{9}/L

Case 2

55 yo

Dyspnea

BP: 81/54 mmHg

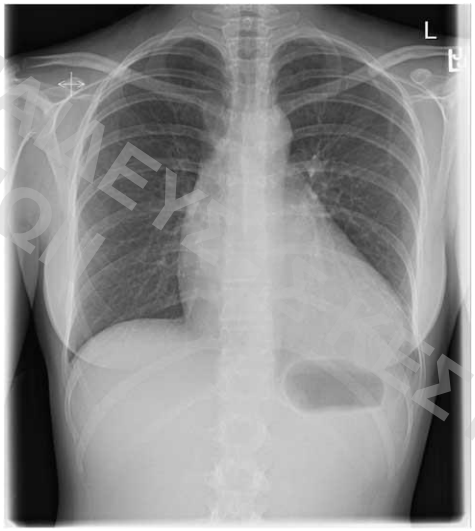
RR 24/min

SpO2: 82% (FiO_2 21%)

HR 131/min

P/E:NIL

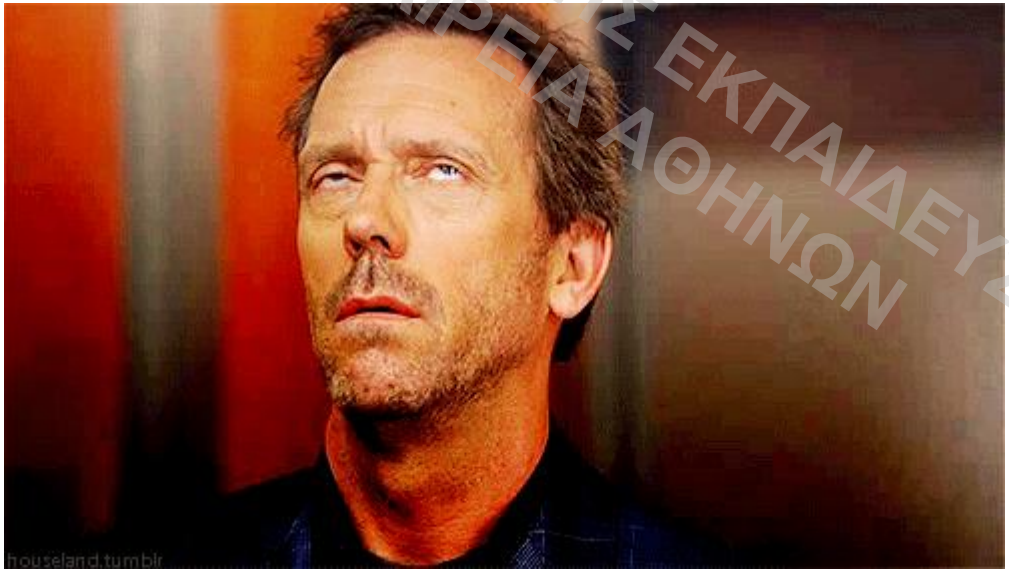
Labs: unremarkable



Hand over the patients and go home????

Septic?
Cardiogenic?
Obstructive?
Hypovolemic?
Distributive?

Fluids?
Antibiotics?
Pressors?
Thrombolytics?
Cath lab?



EMERGENCY THINKING

What does this patient need?

Think of Diagnoses that can kill

In Minutes

In Hours

In Days

what is the
most common thing?

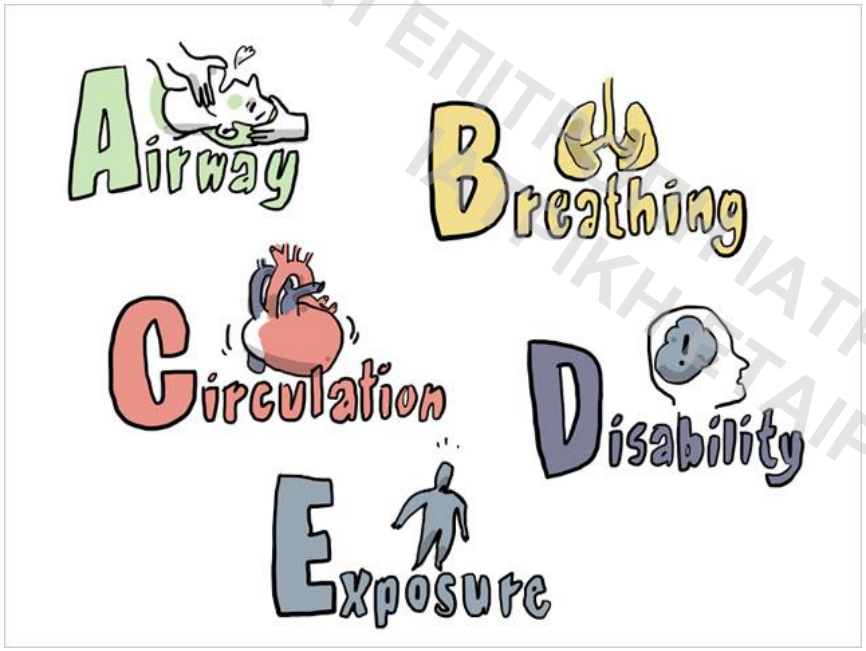
What is the most dangerous thing that this could be?
What is the most life-threatening
thing this could be?”

what is the most common thing this could be

- Blood sugar<80 mg/dl
- Sodium <120 or >150 Meq/l
- Potassium<2.5 or > 6 Meq/l
- pH<7.2
- Spo2<90%
- Bicarbonate <18 mmol/l
- Lactate >4 mmol/l

- **Elderly**
- **Immunocompromised**
- **Multitrauma**

INITIAL APPROACH



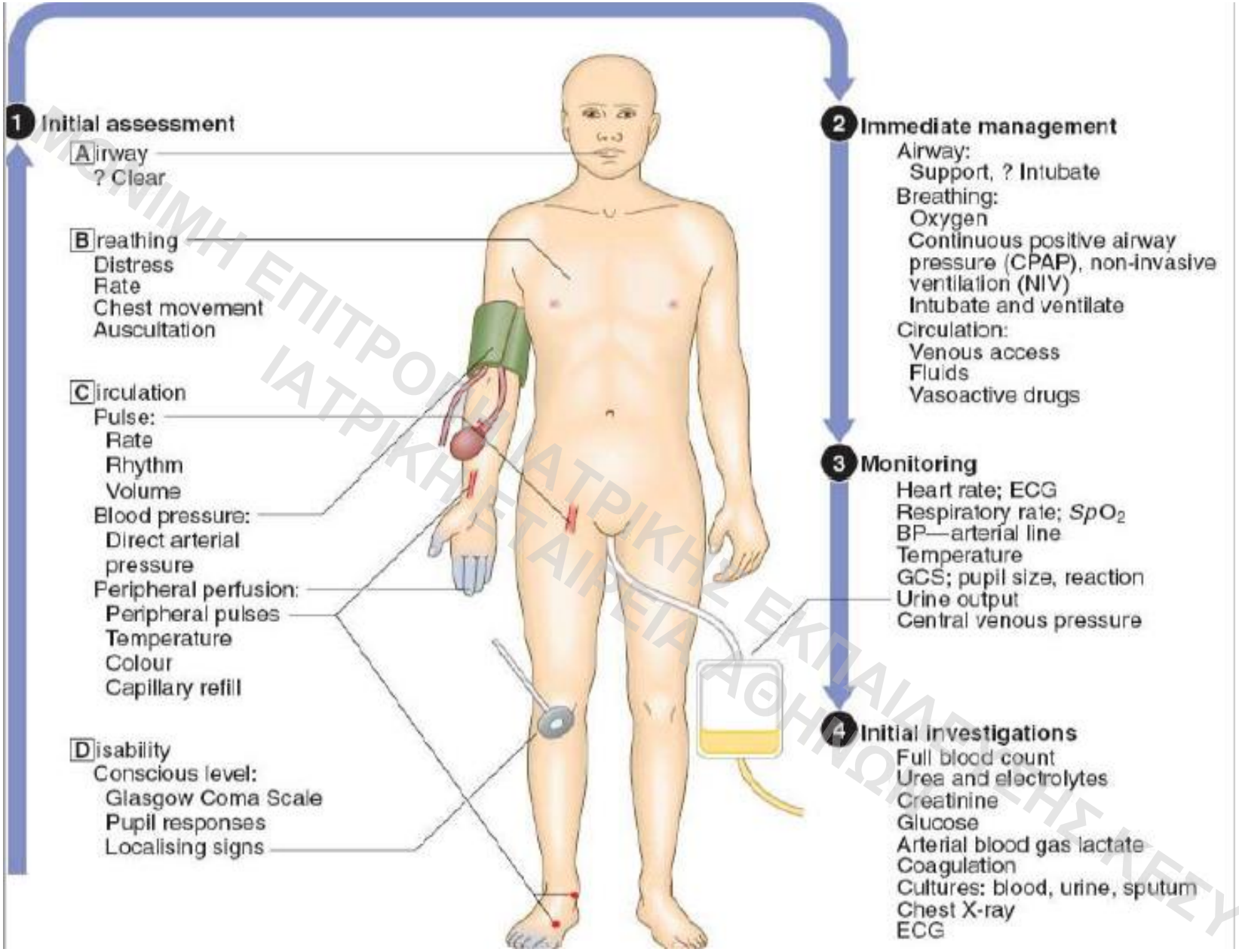
Monitor

Oxygen

Vital Signs

Iv

Exposure/draping/lighting


1 Initial assessment
A irway
? Clear
B reathing
Distress
Rate
Chest movement
Auscultation
C irculation
Pulse:
Rate
Rhythm
Volume
Blood pressure:
Direct arterial pressure
Peripheral perfusion:
Peripheral pulses
Temperature
Colour
Capillary refill
D isability
Conscious level:
Glasgow Coma Scale
Pupil responses
Localising signs
2 Immediate management
Airway:
Support, ? Intubate
Breathing:
Oxygen
Continuous positive airway pressure (CPAP), non-invasive ventilation (NIV)
Intubate and ventilate
Circulation:
Venous access
Fluids
Vasoactive drugs
3 Monitoring
Heart rate; ECG
Respiratory rate; SpO_2
BP—arterial line
Temperature
GCS; pupil size, reaction
Urine output
Central venous pressure
4 Initial investigations
Full blood count
Urea and electrolytes
Creatinine
Glucose
Arterial blood gas lactate
Coagulation
Cultures: blood, urine, sputum
Chest X-ray
ECG

The problem of I.V access in the Critically ill

Flow Rates in IV/IO Access

Gauge	Approximate Flow Rate to Gravity (mL/min)	Time to Infuse 1L (min)
14G	250	4
16G	150	7
Cordis	130	8
18G	100	10
15G Humeral IO	80	13
16G Distal Port Triple Lumen	70	15
15G Tibial IO	70	15
20G	60	17
22G	35	29
18G Prox Port Triple Lumen	30	34

PMID: 20581377; 20157465

Case 1

65 yo

Dyspnea-chest pain (PMH: ICD)

BP: 85/58 mmHg

RR 24/min

SpO2: 90% (3 lt/min)

HR 111/min

T: 37.8^{o}C

P/E: NIL

ECG: sinus tachy

Lactate: 5 mmol/L (<2.0)

TnI:2.5 ng/ml (<0.7)

WBC: 13x10^{9}/L

Case 2

55 yo

Dyspnea, fatigue

BP: 81/54 mmHg

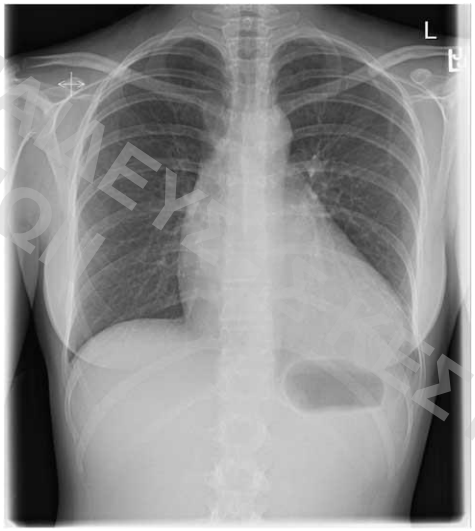
RR 20/min

SpO2: 95% (FiO_2 21%)

HR 121/min

P/E:NIL

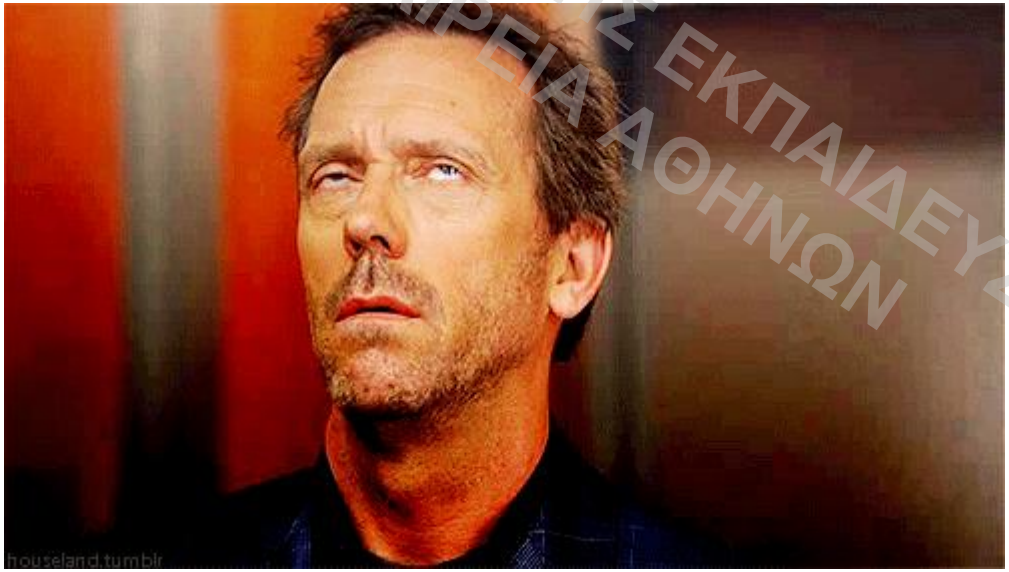
Labs: unremarkable



Hand over the patients and go home????

Septic?
Cardiogenic?
Obstructive?
Hypovolemic?
Distributive?

Fluids?
Antibiotics?
Pressors?
Thrombolytics?
Cath lab?



HYPOVOLEMIC

low circulating volume

TYPES OF SHOCK

CARDIOGENIC

poor pump function

DISTRIBUTIVE

Vasodilatory- ↓↓ SVR

OBSTRUCTIVE

extracardiac obstruction

to blood flow

EM in 5

http://www.emdocs.net/em-in-5-shock/

Fluid resuscitation

Balanced-Isotonic Crystalloids

Shock types-vasoactive drugs

		Pre-load	Pump Fn	After-load	Perfusion	
		PCWP JVP	CO	SVR	O2 Sat	TX
Hypovolemic	- Intravascular vol loss - hemorrhagic - fluid loss	↓	↓	↑	↓	Fluids
Cardiogenic	- Arrhythmia - AMI, valve failure - cardiomyopathy - pericarditis/PE	↑	↓	↑	↓	Dobutamine (5-20mcg/kg/min)
Distributive	Vasodilatory-↓↓ SVR -septic shock/SIRS/TSS - Anaphylaxis - neurogenic shock - Drug/toxin - Addisonian crisis	↓/-	↑	↓	-/↑	Norepi (neurogenic, septic) Epi (anaphylaxis) Phenyl (neurogenic) Dopamine
Obstructive	- Tension PTX - Tamponade - PE	↑	↓	-/↑	-/↓	Thoracostomy, pericardiocentesis

http://www.emdocs.net/em-in-5-shock/

Vasoactive agents

Rough properties of various vasopressors

Drug Typical dose range	Target	Effect on - Heart rate - Inotropy - Ectopy	Effect on systemic vascular resistance	Effect on cardiac output	Effect on blood pressure	Effect on pulmonary vascular resistance	Main uses	Safe for peripheral use?
Inodilators								
Dobutamine 2-20 mcg/kg/min	αββββ	⇑⇑⇑	⇓	⇑⇑⇑	Variable	⇓	Cardiogenic shock	
Milrinone 0.375-0.75 mcg/kg/min	cAMP	⇑⇑⇑	⇓⇓	⇑⇑⇑	Variable	⇓⇓	Cardiogenic shock	
Isoproterenol 2-10 mcg/min	ββββ	⇑⇑⇑⇑⇑	⇓	⇑⇑⇑	Variable		Bradycardia	Yes
Pure Vasopressors								
Vasopressin 0.01-0.06 U/min	V1 & V2	⇓	⇑⇑⇑	⇔/⇓	⇑⇑⇑	⇓	Distributive shock, Pulmonary HTN	No.
Phenylephrine 40-180 mcg/min	αααα	⇓	⇑⇑⇑	Variable	⇑⇑⇑	⇑⇑	Distributive shock	Yes
InoPressors								
Norepinephrine 0-40 mcg/min*	aaaβ	⇑	⇑⇑⇑	⇔/⇑	⇑⇑	⇔	Shock (most types)	Yes, for short period with monitoring
Epinephrine 0-20 mcg/min*	αββββ	⇑⇑⇑	⇑	⇑⇑⇑	⇑⇑		Bradycardia, cardiogenic shock, sepsis, anaphylaxis	Yes
Dopamine, low 1-4 mcg/kg/min	Dopa-R	⇔	⇓	⇑	⇓		Zombie apocalypse (absence of better agents).	Probably not
Dopamine, medium 4-10 mcg/kg/min	αββββD	⇑	Variable	⇑⇑	Variable			
Dopamine, high 10-20 mcg/kg/min	aaaβD	⇑⇑	⇑⇑	⇑	⇑⇑⇑	⇑		

*Listed ranges are typically used doses in the United States, but there is no true "maximal" dose. Some countries may tend to use higher doses than others. At very high doses, pressors may lose some receptor specificity. The best dose is the dose required to keep the patient alive – in some cases very high norepinephrine or epinephrine doses may be needed.

-The Internet Book of Critical Care, by @PulmCrit

https://emcrit.org/ibcc/pressors/

Adjuncts

- **Prefer P.O.C testing**
- **Rapid diagnosis**
- **Avoid patient transfer before admission**

Hs – Troponins
(well established for ACS)
Nt-proBNP
(Mostly prognostic value for HF)

Under research: MR-proANP, galectin-3
Multi-marker scores superior

Troponin
BNP/NT-proBNP
(high=> complications & 30-day mortality)
D-Dimers
(***Only rule out*** low probability pts)
IMA
(better (+) pred. Value vs d-dimers)

OTHERS:
NGAL => early AKI detection
S100B => Head trauma

Lactate
(>4 higher in-hospital mortality)
CRP
(low sens.& spec)
PCT
(bacterial vs non bacterial ***antibiotic stewardship***
spec 81%, sens 77%)
suPAR
(diagnosis & prognosis – higher spec. vs PCT – Sepsis vs SIRS differentiation)

Under Research: MR-proADM, LBS

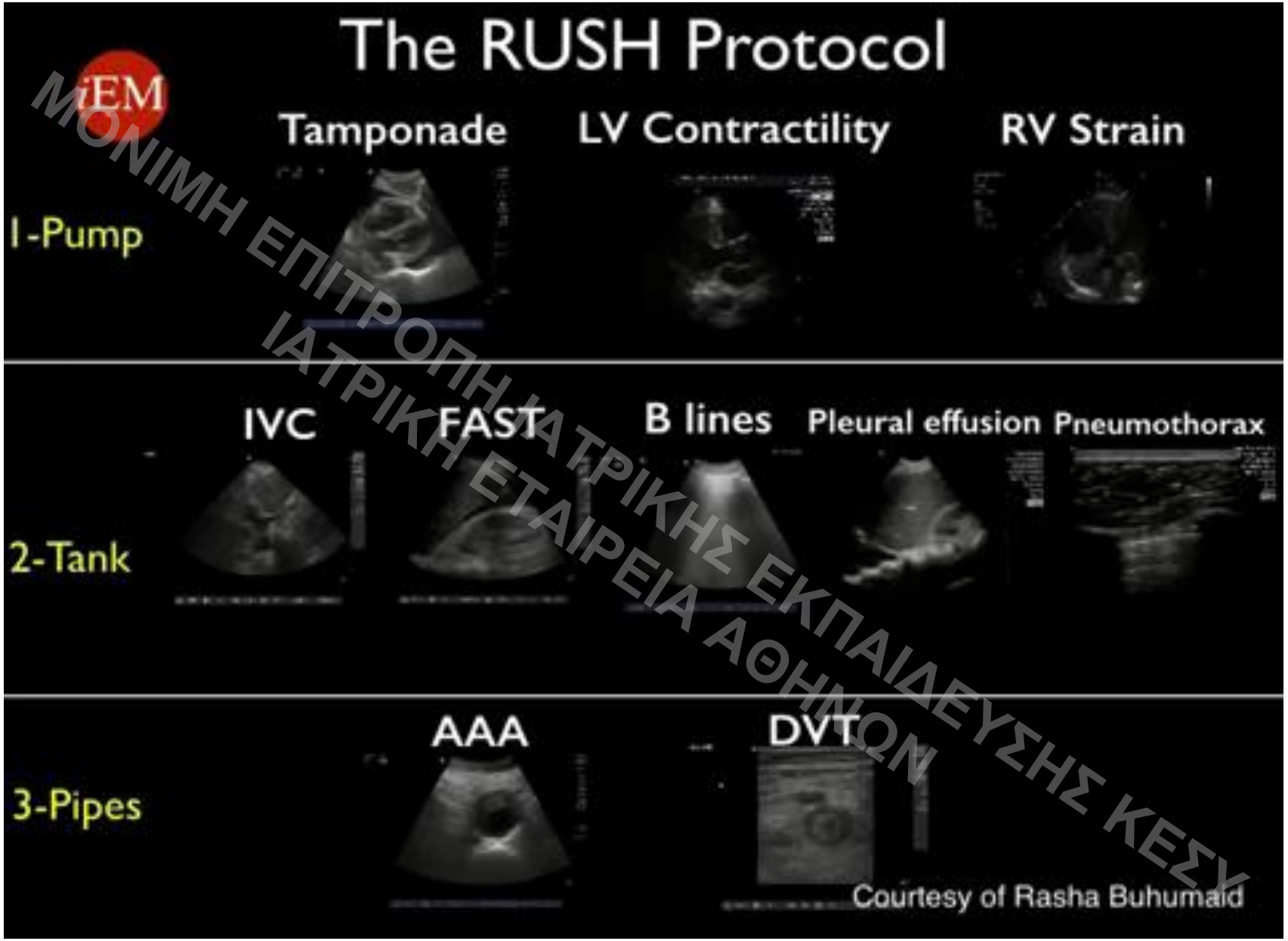
http://dx.doi.org/10.1136/bmjopen-2020-042989
DOI: 10.5772/intechopen.94509
SHOCK, Vol. 53, No. 4, pp. 416–425, 2020

Rapid

Ultrasound for

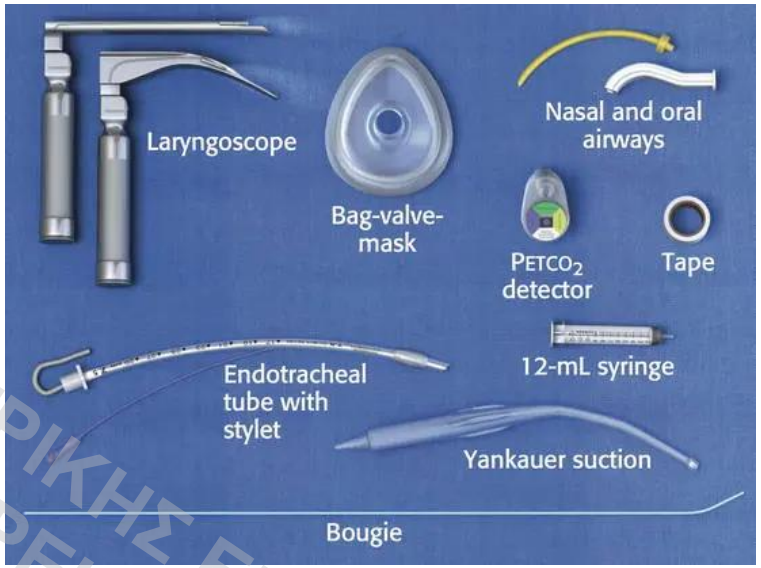
Shock and

Hypotension

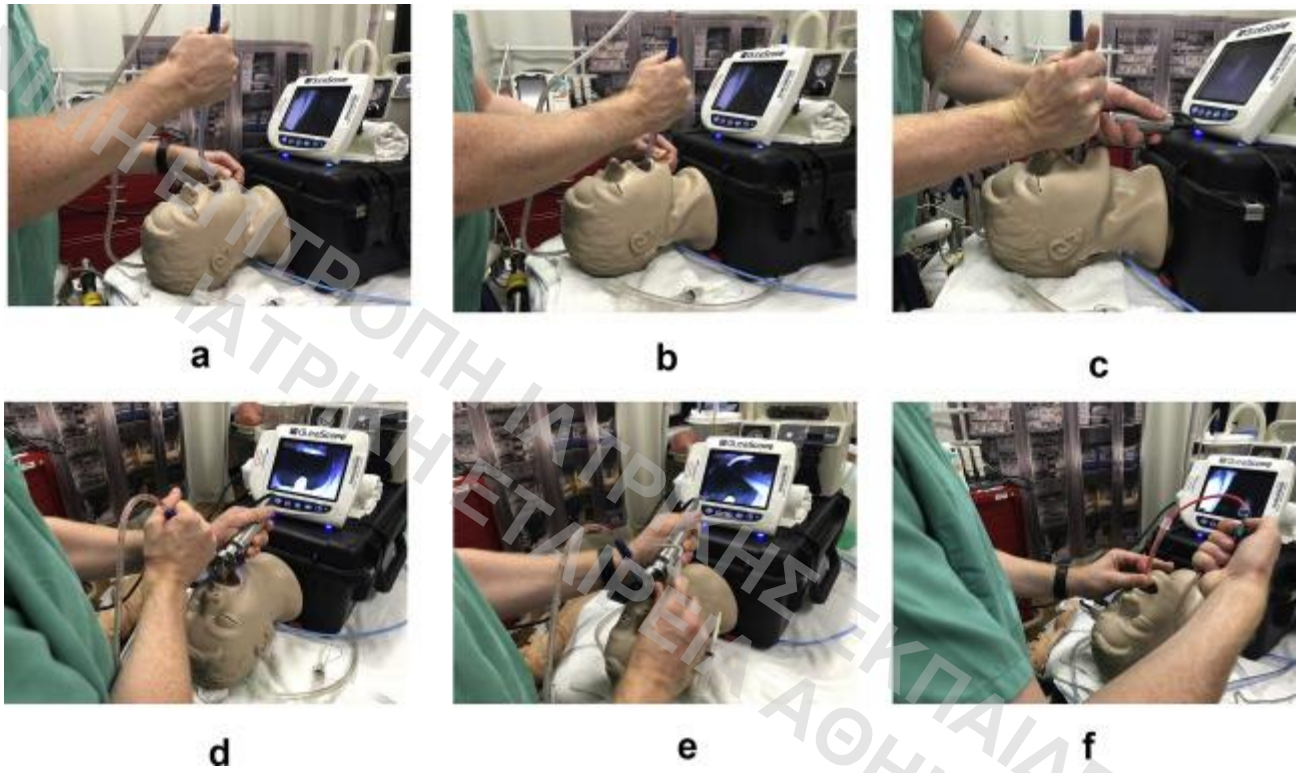


Anatomically and physiologically difficult airway

Equipment



Technique



Suction Assisted Laryngoscopy and Airway Decontamination (SALAD)

https://doi.org/10.1016/j.resplu.2020.100005

Basic principles of resuscitation in shock

The goal of the interventions is to restore adequate tissue perfusion identification and treatment of the underlying etiology

- Resuscitate before intubate
- Aggressive airway control (endotracheal intubation)
- Associated interventions such asmedications (ie, sedatives can exacerbate hypotension) and positive pres sure ventilation may reduce preload and cardiac output and may contribute to hemodynamic collapse.
- Shock patient requires lower dose of induction agent (except etomidate) and higher dose of paralytic
- Arterial oxygen saturation should be restored to > 93% and ventilation controlled to maintain a $PaCO_2$ of 35 to 40 mm Hg. Circulatory hemodynamic stabilization begins with IV access through largebore peripheral venous lines.
- US guidance has proven helpful
- Central venous access is the preferred route for the longterm administration of vasopressor therapy.
- Isotonic or balanced crystalloid intravenous fluids (0.9% NaCl, Ringer lactate) in the initial resuscitation phase
- Blood products in case of hemorrhagic shock
- Acidosis should be treated with adequate ventilation and fluid resuscitation.
- Sodium bicarbonate use is controversial. Use only in the setting of severe refractory acidosis

http://www.emdocs.net/intubating-critically-ill-patient/

Evaluation and Management of the Physiologically Difficult Airway: Consensus Recommendations From Society for Airway Management

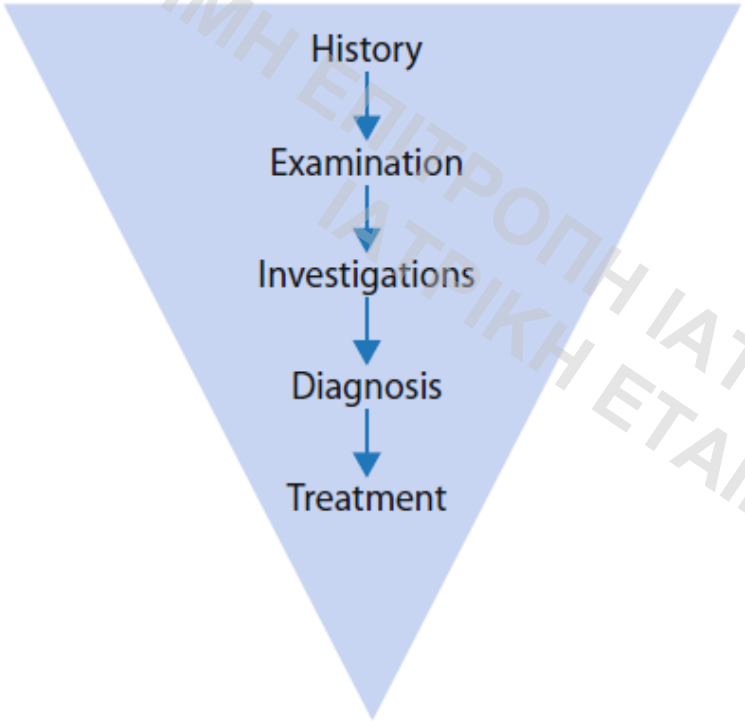
Rebecca L. Kornas, MD,* Clark G. Owyang, MD,† John C. Sakles, MD,‡ Lorraine J. Foley, MD, MBA,§ and Jarrod M. Mosier, MD,‡‖ on behalf of the Society for Airway Management's Special Projects Committee

Table. Top 10 Society for Airway Management Key Recommendations for Evaluation and Management of the Physiologically Difficult Airway

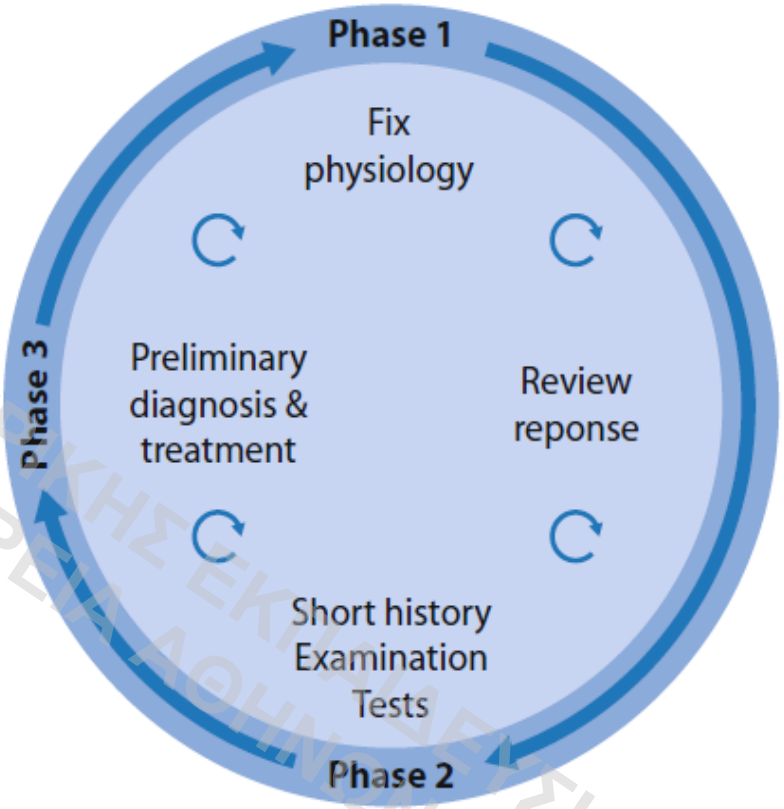
Physiology	Recommendations
Hypoxemia	1. Preoxygenation should be performed using high-flow oxygen for at least 3 min, or 8 vital capacity breaths. 99% agreement (SD 4.5%)
	2. Desaturation is the biggest risk factor for cardiopulmonary arrest. 87% agreement (SD 29%)
	3. If the patient has significant shunt physiology or reduced functional residual capacity (eg, pregnancy, obesity, ARDS), preoxygenation should be performed with PEEP using NIPPV. 98% agreement (SD 7.5%)
	4. Patients should be preoxygenated in the upright position when possible. 98% agreement (SD 6.3%)
	5. Delayed sequence intubation is an option for patients who cannot tolerate preoxygenation with NIPPV or HFNO. 100% agreement (SD 0%)
Hypotension	6. Risk factors for decompensation include vascular and cardiac effects of induction agents and effects of positive pressure ventilation. 99% agreement (SD 3%)
	7. Peri-intubation hypotension is independently associated with poor outcomes, including mortality, length of stay, and end-organ injury. 96% agreement (SD 12%)
	8. Patients should be screened for high risk of hemodynamic collapse with intubation. Those with a shock index >0.7 are at increased risk. 99% agreement (SD 1.5%)
	9. Fluid-responsive and fluid-tolerant patients should be fluid resuscitated before intubation, or at least during the intubation attempt. 99.5% agreement (SD 1.5%)
	10. When possible, vasopressor infusions should be started before intubation in patients that are not volume responsive or fluid tolerant. 99.5% agreement (SD 1.5%)

Anesth Analg . 2021 Feb 1;132(2):395-405

Structured Approach to Early Recognition and Treatment of Acute Critical Illness



Traditional



Recommended

DOI 10.1007/978-3-319-03746-2_51

- **Management of critically ill patients is most trying, time & resousce consuming in the ED**
- **Critically ill patients are not always in common view**
- **Our role is to identify them in time**
- **First assessment with ABCDE approach allows for simultaneous recognition & treatment of potentially fatal conditions**

- **POCUS can facilitate diagnosis & treatment of critically ill pts by reducing time to diagnosis, aiding targeted treatment and avoiding unnecessary movements**
- **POC testing & biomarkers are preferable and can be useful for diagnosis**
- **A circular working diagnose-fix physiology-reevaluation approach is preferable to the classic pyramid medical approach**

ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
Εθνικόν και Καποδιστριακόν Πανεπιστήμιον Αθηνών
ΙΔΡΥΘΕΝ ΤΟ 1837

ΙΑΤΡΙΚΗ ΣΧΟΛΗ ΑΘΗΝΩΝ

ΙΑΤΡΙΚΗ ΣΧΟΛΗ ΑΘΗΝΩΝ • ATHENS MEDICAL SCHOOL • 1837

Εφη Πολυζωγοπούλου
Επίκουρη Καθηγήτρια
Επείγουσας Ιατρικής ΕΚΠΑ
Πανεπιστημιακή Κλινική Επειγόντων Περιστατικών ΠΓΝ ΑΤΤΙΚΟΝ

EUSEM
EUROPEAN SOCIETY FOR EMERGENCY MEDICINE

ASS PROF. EFFIE POLYZOGOPOULOU
CHAIRPERSON OF EUSEM ULTRASOUND SECTION

effiep@live.com

effiep@med.uoa.gr