UNIVERSITY OF SZEGED

Basics of Emergency Medicine

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Tartalomjegyzék

Abbreviations

ACS	Acute Coronary Syndrome
ACCP/SCCM	American College of Chest Physicians/Society of Critical Care Medicine
ADORA	Antidepressant Overdose Risk Assessment
AED	Automatic External Defibrillator
AF	Atrial Fibrillation
AIDS	Acquired Immune Deficiency Syndrome
ALS	Advanced Life Support
AMI	Acute Myocardial Infarct
AMP	Adenosine Monophosphate
APTT	Activated Partial Thromboplastin Time
ARDS	Adult (Acute) Respiratory Distress Syndrome
ASY	Asystole
AV	Atrial - ventricular
BE	Base Excess
BLS	Basic Life Support
BZD	Benzodiazepine
CABG	Coronary Artery Bypass Graft
CAMP	Cyclic Adenosine Monophosphate
CBF	Cerebral Blood Flow
CEDIS	Canadian Emergency Department Information Systems
CK	Creatinine kinase
CNS	Central nervous system
CO	Cardiac output
CoHb	Carboxyhaemoglobin
COPD	Chronic Obstructive Pulmonary Disease
COX	Cyclooxygenase
CPP	Cerebral Perfusion Pressure
CPR	Cardio-Pulmonary Resuscitation
CRP	C-reactive Protein
CRT	Capillary Refill Time
CSF	Cerebrospinal Fluid
CT	Computer Tomography
CTAS	Canadian Triage and Acuity Scale
CVI	Cerebrovascular Insult
CVP	Central Venous Pressure
DAMP	Damage Associated Molecular Pattern
DBP	Diastolic Blood Pressure
DIC	Disseminated Intravascular Coagulation
DNOC	Dinitroorto Cresol
EDTA	Ethylenediaminetetraacetic Acid
EKG	Electrocardiograph
ERCP	Endoscopic retrograde cholangio-pancreatography

ES	Extrasystole
ETI	Endotracheal Intubation
ET	Endotracheal Tube
FFA	Free Fatty Acid
FFP	Fresh Frozen Plasma.
FiO2	Fraction of inspired O2
GABA	Gamma Amino Butyric Acid
GCS	Glasgow Coma Scale
GFR	Glomerular Filtration Rate
GI	Gastrointestinal
GM	Grand mal
GOT	Glutamate Oxaloacetate
GPT	Glutamate Pyruvate Transaminase
GRACE	Global Registry of Acute Coronary Events
hCG	Human Chorionic Gonadotropin
Htc	Haematocrit
Hgb	Haemoglobin
HHS	Hyperglycaemic Hyperosmolar State
HgA1c	Haemoglobin A1c
HTAS	Hungarian Triage and Acuity Scale
IABP	Intra-aortic Balloon Pump
IBD	Irritable Bowel Syndrome
ICD	Implantable Cardioverter Defibrillator
ICH	Intracerebral Haemorrhage
ICP	Intracranial Pressure
INH	Isonicotinic Acid Hydrazide
IPAT	Initial Pain Assessment Tool
ICU	Intensive Care Unit
LBBB	Left Bundle Branch Block
LD	Lethal Dose
LF	Liver Function
LMA	Laryngeal Mask Airway
LMWH	Low Molecular Weight Heparin
LSD	Lysergic Acid Diethylamide
MAP	Mean Arterial Pressure
MICU	Mobile Intensive Care Unit
MRI	Magnetic Resonance Imaging
MOF	Multi Organ Failure
MSOF	Multiple System Organ Failure
NAPQI	N-acetyl-para-benzoquinone-imine
NIBP	Non-invasive Blood Pressure
NO	Nitric Oxide
NSAID	Nonsteroidal Anti-inflammatory Drugs
NSTE-ACS	Non-ST Elevation Myocardial Infarction Acute Coronary Syndrome
NSTEMI	Non-ST Elevation Myocardial Infarction
PAD	Public Access Defibrillator

PAMP	Pathogen Associated Molecular Pattern
PCI	Percutaneous Coronary Intervention
PCP	Phencyclidine
PCT	Procalcitonin
PEA	Pulseless Electrical Activity
PoCT	Point of Care Testing
POSS	Prehospital Cincinnati Stroke Scale
PT	Prothrombin time
PTX	pneumothorax
PVT	Pulseless Ventricular Tachycardia
RBC	Red Blood Cell
RDE	Rectal Digital Examination
ROSC	Return of Spontaneous Circulation
ROTEM	Rotational Thromboelastography
RR	Respiratory Rate
RSI	Rapid Sequence Induction
SAH	Subarachnoid Haemorrhage
SBP	Systolic Blood Pressure
SIADH	Syndrome of Inadequate Antidiuretic Hormone Secretion
SIRS	Systemic Inflammatory Response Syndrome
STEMI	ST Elevation Myocardial Infarction
SVPT	Supraventricular Paroxysmal Tachycardia
TAI	Thrombocyte Aggregation Inhibitor
TAT	Turnaround Time
TB	Tuberculosis
TBI	Thyroxin Binding Index
TCA	Tricyclic Antidepressant
TCT	Thrombocyte
TEE	Transoesophageal Echocardiography
TI	Thrombin Time
TIVA	Total Intravenous Anaesthesia
TNF	Tumour Necrosis Factor
TP	Total Protein
TTE	Transthoracic Echocardiography
US	Ultrasound
VCI	Vena Cava Inferior
VES	Ventricular Extrasystole
VF	Ventricular Fibrillation
VT	Ventricular tachycardia
WBC	White Blood Cell
WHO	World Health Organisation

1. The Basics of Physical Examination and Documentation in the ED byDr.PetőZoltán

Working as an emergency physician means a challenging job for the ones who can cope with unpredictable situations under always changing circumstances. Lots of personal skills are needed to become a good emergency physician. For most of the patients contact healthcare professionals first in the ED, it is essential to build a good rapport with them. Because of crowded waiting rooms and misconceptions about the ED's function people might get angry with the medical staff. To prevent conflicts excellent communication skills, well-organised protocols and evidence based treatment strategies are required.

NB: Always treat the patient as you would want to be treated!

The ABCDE of Patient examination:
A: Airway
B: Breathing
C: Circulation
D: Disability
E: Environment, Anything Else

NB: Always insist to ABCDE, which focuses on the most life-threatening conditions!

Before examination always make sure, that the patient's dignity is preserved. Medical condition of the patient is always confidential.
Introduction is the simplest way to start building a good rapport, defusing a volatile situation and evaluating the patient's condition at the same time. The patient who can response in whole detailed sentences no serious ABCD problem is probable.
During examination all of the senses are used. It is extremely important to search for the absence of *normal* and the presence of *not normal*.
Note keeping is indispensable. It is essential to record the patient's history, every symptoms and as much objective parameters as you can to help documenting what happened to the patient. It gives also a good basis to explain the therapeutic decisions, what have been made. But keep in mind write nothing judgemental what might stigmatise the patient (body habitus, clothing, appearance, illicit drug use if there is no consequence for the recent condition). Do

not write either critics about other healthcare providers, or messages for them. Avoid speculations, suspicions or hunches.

Taking the patient's history:

- Why the patient is seeking help?
- What is the patient's chief complaint? Are there any modifying factors?
- Current medication
- Any allergies
- Previous history

Focus on the recent problem.

History of trauma patient: The finer details may have to wait until later, but:

Record the mechanism of injury.

In case of suspected major trauma the following should be recorded:

High speed road collusions

Another person died in the same vehicle

Patient felt more than 2 m

Pedestrian run over or thrown up by vehicle

NB: Avoid abbreviations, write the documentation for the patient!

After taking the history, examine the patient: ABCDE

A: Airway

Free, open

Partially obstructed

Completely obstructed

Examine the oral cavity and the nose. Search for bleeding, wounds and foreign body.

With auscultation can be heard: -put the stethoscope on the larynx

Normal breathing sounds: free airway

Stridor: partial obstruction

Absence of breathing sounds (with breathing movements): complete obstruction

Life-threatening condition: airway obstruction

Treatment: clear the airways, and maintain them

Document the physical findings whether they are physiological or not!

B: Breathing

Use all senses and search for absence of normal and presence of not normal:

Inspection:

Determine the respiratory rate and depth

Look for chest wall asymmetry. Paradoxical chest wall motion

Look for bruising, seat belt or steering wheel marks, penetrating wounds

Look for abnormal breathing pattern

Palpation:

Feel for the trachea for deviation

Assess whether there is adequate and equal chest wall movement

Feel for chest wall tenderness or rib 'crunching' indicating rib fractures

Feel for subcutaneous emphysema

Auscultation:

Listen for normal, symmetric breath sounds on both sides. Search for absence of breathing sounds,asymmetric breathing sounds or crepitation.

Percussion:

Percuss both sides of the chest looking for dullness or resonance

Record the followings:

SpO2, FiO2, Resp. Rate, arterial blood gas

Add further details if the patient is ventilated mechanically (position of ET, mode of ventilation, tidal volume, minute volume, PEEP).

Potential life-threatening conditions: PTX, tension PTX, haemothorax, flail chest, opened thoracic wound, respiratory failure.

Document the physical findings whether they are physiological or not!

C: Circulation

Look for signs of shock:

Fast shallow breathing

Cold, cyanotic, sweaty skin

Altered mental status

Low blood pressure

Tachycardia, with barely palpable pulse

Record the followings:

HR, NIBP or IABP, ECG description, Capillary Refill Time

Document the physical findings whether they are physiological or not!

Between C and D we describe the abdominal examination (in case of trauma patient C contains the abdominal status, and the pelvic stability too).

Abdominal examination:

Look for tenderness, pain, muscular defense

Look for bowel sounds

Palpate the liver, spleen

Do not forget rectal digital examination!

Document the physical findings whether they are physiological or not!

D: Disability

Examine and record GCS, mental status (disorientation, drowsiness, stupor or coma), pupils (width, symmetry and pupillary light reflex), muscle strength (symmetry, paresis), vegetative disorders, sensory disorders and pathologic reflexes.

For detailed description see Chapter 9.

Document the physical findings whether they are physiological or not!

NB: Measure blood glucose if the patient has altered mental state!

E: Environment, Anything Else:

Look for other important signs and symptoms: body temperature, blood glucose measurement. Also record any additional and relevant information.

After the physical examination the applied treatment has to be written, then the laboratory and imaging findings.

At the end of the documentation give a short, clear and logic summary about what happened to the patient in the ED and give an understandable conclusion.

An example of a normal physical status:

A: Free airways

B: Symmetric chest. Eupnoea. Symmetric, normal breath sounds. No congestion, no crepitation. No resonance, no dullness. No cyanosis.

RR: /min, SpO2: %

C: Well palpable pulse of all peripheral arteries. CRT < 4sec, Clear heart sounds without murmurs. Rhythmic heart function. No murmurs above carotid arteries. No oedema on the lower extremities.

ECG:

NIBP:

Abdomen: No tenderness. Presence of normal bowel sounds. Free renal regions. No fluid wave.

RDE:

Nasogastric tube:

D: GCS: E: 4, V: 5, M: 6 = 15. Alert, and orientated. Symmetric pupils, normal width on both sides with normal light reflexes. No nystagmus. No sign of meningeal irritation. Normal muscle strength. Intact sensory function. Intact vegetative function.Negative Romberg's probe. No sign of neurological disorders.

E: Normal skin, without jaundice. No rashes. Symmetric limbs. No adenomegaly.

Minor wounds:

Axial temperature: °C

Blood glucose: mmol/L

Parameters:

Scores:

Arterial/Venous Blood Gas: pH: , pO2: mmHg, SatO2: %, pCO2: mmHg, BE: , Na: mmol/L, K: mmol/L, Ca: mmol/L, Cl: mmol/L, Glu: mmol/L, Hgb: g/dL, Hct: %

Treatment:

Interventions:

Patient referral: It is another important role of the ED. If the patient need specialist's advice, or hospital admission it is essential being able to refer the patient to other healthcare professionals. Referral can be immediate (if specialist's help is needed promptly), urgent (in 30 minutes), less urgent (in 2hours), planned.

Role of patient referral:

Give a brief, clear, and detailed summary of the patient's history, physical findings, and any relevant information

Make a clear explanation, why the specialist's opinion is needed, or why the patient should be admitted

Be polite, and prepared to give any further information that would be asked for

Also write a formal referral letter with detailed information about the patient

After referral, document the followings:

The name of the specialist

The exact time of the referral

The conclusion

2. BLS – Basic Life Support by Dr. Cserjés Andrea

Definitions:

BLS - Basic life support: Protocolled managementto improve the chances of resuscitation of clinical death orimprove the chances of survival of life-threatening injuriestill advanced healthcare unit arrives. BLS is provided without special equipment (in ideal cases rescuers have personal protective equipment: gloves, and bag valve mask for ventilation). In public areas may be Public Access Defibrillators – PAD.

- Interventions used during BLS: CPR, use of AED (automatic external defibrillator), apply recovery position and management of suffocating.

Adulthood: Adult BLS should be performed in adults and in children presumably reached teenage.

Clinical death: reversible cessation of vital functions, resuscitation may be successful

Biological death: irreversible cessation of vital functions

Resuscitation: bring back reversibly ceased vital functions

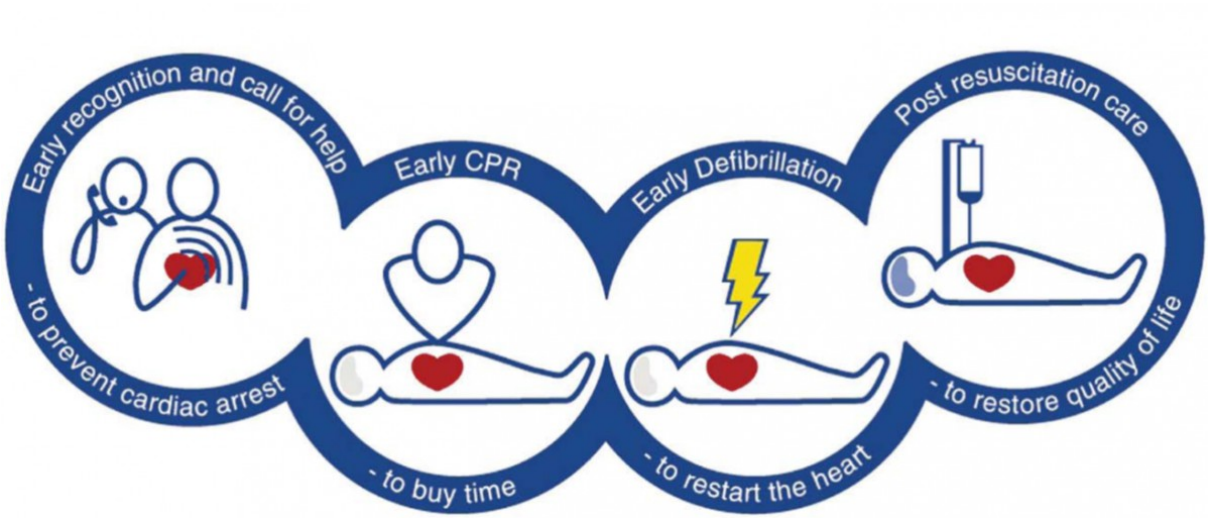
- Cardiopulmonary resuscitation CPR: perform chest compression, airway management and ventilation

Vital functions: spontaneous breathing, spontaneous circulation, proper brain function

Post-resuscitation care: special care, aimed for complete neurological recovery after resuscitation.

*Chain of survival:*order of a four-step protocol for the successful resuscitation:

- Early recognition and call for help
- Early CPR
- Early Defibrillation
- Early post-resuscitation care



from http://www.firstaidforfree.com

Early recognition and call for help:

Use ABCDE approach: if vital signs are present, cardiac arrest may be prevented.

Warning signs: sudden chest pain, dyspnoea…

If cardiac arrest occur (absence of vital signs) call for help (ambulance or hospital reanimation team).

NB: Always tell, that you ask help for resuscitation, and give the accurate address or localisation too!

Start good quality CPR.

ABCDE approach

General impression: Is the patient alive? Is there serious bleeding?

A	Free Partial obstruction Complete obstruction	Position: head tilt, chin lift Suction Oropharyngeal/Nasopharyngeal airways
B	Respiratory rate Respiratory work SpO2 Auscultation	A High flow Oxygen Ventilation
C	Heart Rate Peripheral Perfusion NIBP Assess Rhythm Dilated cervical veins, pulmonary oedema	A+B Monitor Insert IV cannula Give fluid Electric or pharmacologic therapy
D	AVPU Pupils Blood Glucose	A+B+C Give glucose, if hypoglycaemia present
E	Full body examination	A+B+C+D

	Body temperature Patient's history Regularly taken medicines Allergies	warming up

Steps of BLS

Safety	Approach carefully!
Evaluation of consciousness	AVPU - Unconscious
Call for help by shouting	Shout for help
Airway management	Position the patient, head tilt/chin lift
Vital signs	Absence of breathing
Call for help	Call for advanced help
Chest compression	Start compressions
Ventilation	Start ventilation
CPR	Resume 30:2 CPR

1. Safety: Make sure that the scene is safe: there is no electric, mechanical, explosive, toxic or chemical danger!

   Try to eliminate the dangerous element, if it is not possible, call for advanced help

2. Evaluation of consciousness
   a. the patient responds to verbal tactile or painful stimuli -> conscious
   b. the patient does not respond to stimuli -> unconscious

   NB: unconscious patients has relaxed muscles, tongue may fall back and obstruct the airways!

3. Cry for help: as loud as you can to get assistance till you can be with the patient and continue BLS. Assistance is essential for CPR, and for further call for advanced help.
4. Airway management: Move the patient into supine position, tilt the head back and lift the chin. Only clear the patient's mouth if something (vomit, food, foreign body…) obstruct the airways obviously.
5. Vital signs: after airway management examine the patient maximum 10 seconds
   a. Look, listen and feel for breathing!
      a.i. normal breathing: 2 breaths/10secs. no sign of dyspnoea, or rattle

a.ii. abnormal breathing: agonal or terminal breathing, dyspnoea, rattle

a.iii. no breathing

If the patient is unconscious or no signs of spontaneous breathing start CPR!

NB: in unclearcases start CPR!

6. Call for help:

    a. Unconscious patient with normal breathing: call advanced help and prevent suffocation:

        a.i. in case the rescuer can stay with the patient: head tilt, chin lift

        a.ii. in case rescuer has to leave the patient to call help: rescue position

        a.iii. in case the patient is vomiting: rescue position

    b. Unconscious patient with abnormal or no breathing: call advanced help

        b.i. in case the rescuer can stay with the patient: call for help on phone or send for help and provide CPR. If there is an opportunity to use AED send for it too.

        b.ii. in case rescuer has to leave the patient to call help: rescue position, then when return start CPR!

7. Chest compressions: CPR has to be started with chest compressions.

    a. Hands only CPR: rescuer does not want or unable to ventilate the patient, or rescuer is laic and has to be assisted through phone.

    b. In case of suspected prolonged hypoxic state ventilation ought to be performed.

    Perform chest compression:

        b.i. knee down next the patient (laying supine on hard ground) easily reach chest and head from there

        b.ii. put one hand's carpal region on the middle of the chest, then put the other hand on it with interlaced fingers

        b.iii. make sure that your hand carpal region is on the lower part of sternum

b.iv. push the chest 5-6cm deep with straight arms and fixed shoulders

b.v. then let the chest return to the starting position, but do not lift hands from chest

b.vi. do chest compression with the frequency of 100-120/min

b.vii. 1:1 ratio of the pushes and the releases

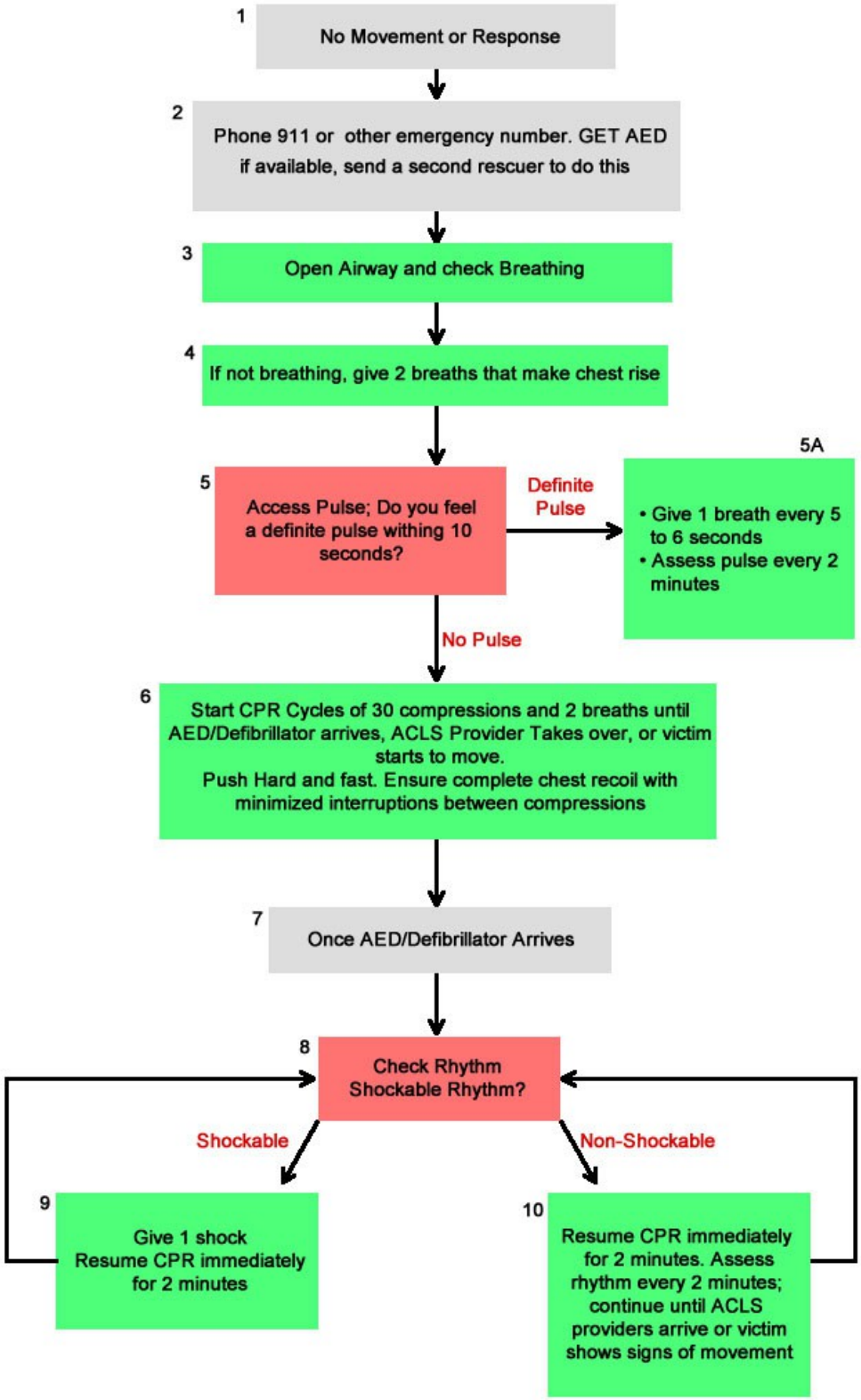
8. Ventilation: chest compression ventilation ratio in adults 30:2
    a. Hands only CPR requires no ventilation
    b. Proper way of mouth-to-mouth ventilation
        b.i. position the patient: head tilt, chin lift
        b.ii. hold the patient's nose, and open the patient' mouth
        b.iii. take a normal breath
        b.iv. cover the patient' mouth with yours, and blow air into it under 1 sec
        b.v. control the effectivity (look, listen and feel) and take a breath
        b.vi. repeat mouth-to-mouth ventilation
        b.vii. resume CPR
9. Only stop CPR for reassess vital signsif the patient starts to move
10. CPR should be performed continuously and good quality CPR is essential. To ensure good quality CPR rescuers must be changed in every 2 minutes. Before change the rescuer provides CPR has to count loudly, and the other rescuer has to get into position during the previous rescuer provide ventilation. The aim is to provide continuous CPR.
11. Stop the CPR if:
    a. advanced help arrived and started ALS
    b. ROSC: patient start to move, open eyes or start to breath
    c. rescuers have become exhausted

12. Early defibrillation with AED

    a. AED recognise shockable rhythm and deliver shock as the shock button pushed

    b. Follow the orders of the AED (“Do not touch the patient!” “Resume CPR!”)

    c. Special pads exists for children under the age of 8 (place one on the chest and one on the back) switch the AED to paediatric mode.



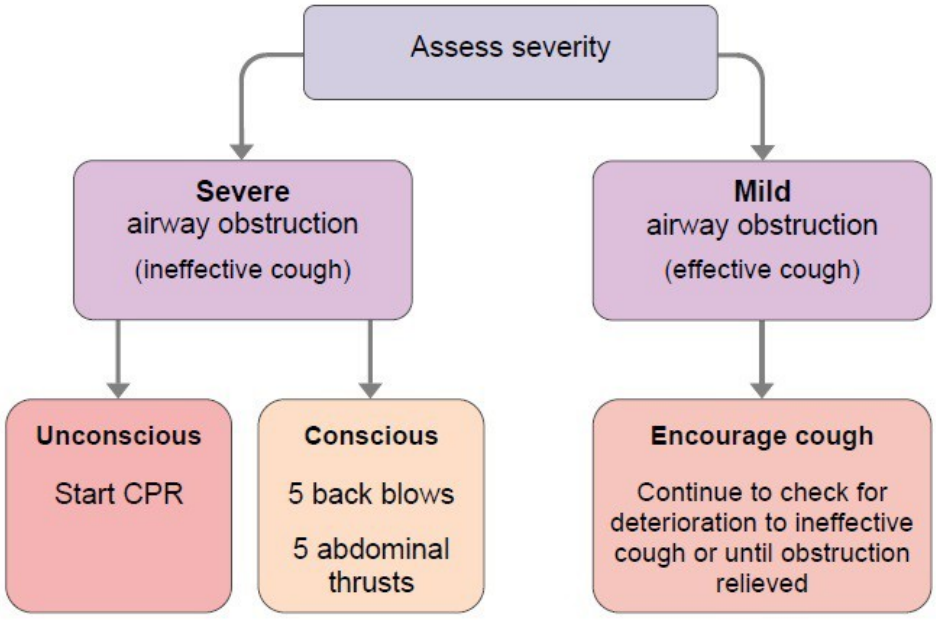
from http://blsalgorithm.com/

Other BLS Functions:

Rescue Position:

- CPR not required (maintained breathing and circulations)
- Tongue may fall back – airway obstruction
- Vomiting
- Patient has to be left to calling for help
- Regular reassessment of breathing (every minutes)
- If patient has abnormal or no breathing turn the patient into supine position and start CPR
- If the patient was in the same position for 30 minutes other sided rescue position should be taken into consideration.

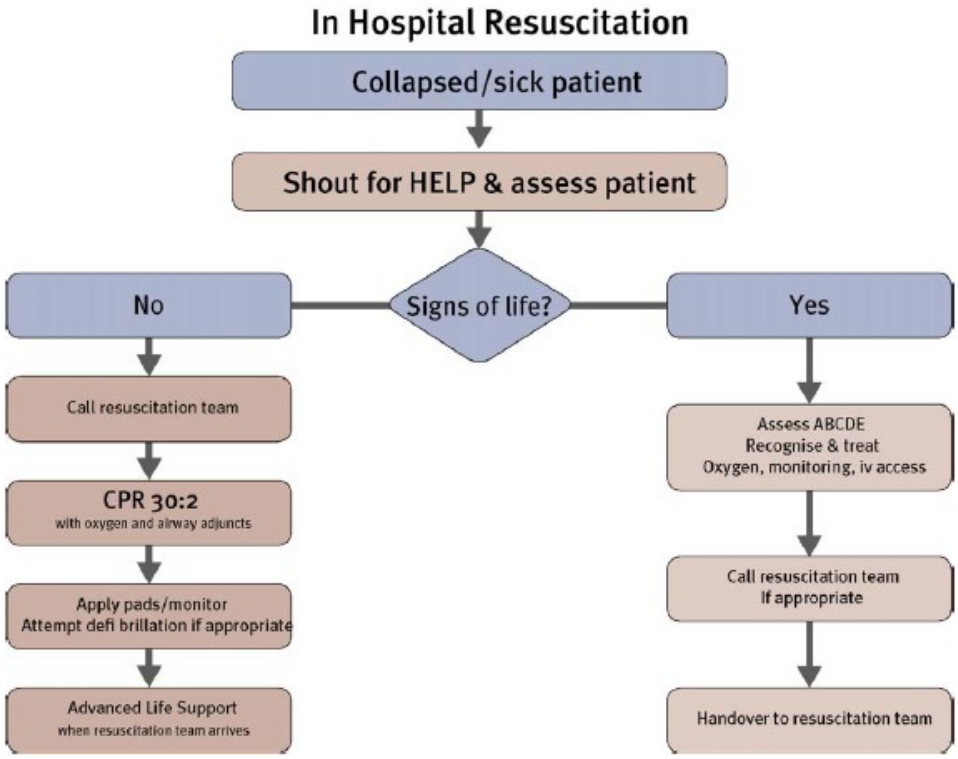
Foreign body caused suffocation: inhalation of small item or food may cause partial or complete airway obstruction.



from orchardtrainingservices.co.uk according to ERC Guidelines for resuscitation 2010

	Mild airway obstruction	Severe airway obstruction
General signs	occurs during eating sudden onset victim grabs his neck	
Responsiveness	Able to response.	Unable to response.
Other signs	forced coughing, maintained breathing	weak coughing, inspiratory and expiratory stridor UNCONSCIOUSNESS!
Management	encourage cough	conscious: 5 back blows, then 5 abdominal thrust unconscious: start CPR try to clear airways!

In hospital BLS:



from ERC Guidelines for resuscitation 2010

Early recognition and call for help (reanimation team)

Early CPR and defibrillation: rescuers work parallel:

1. rescuer ask for help
2. rescuer start chest compression
3. rescuer mechanical ventilation with mask, balloon and high flow oxygen

4. rescuer fetch the defibrillator

Refer the patient's case to the reanimation team when they arrive, and prepare for ALS.

Alternative airway management may be performed -> X-BLS

Post Resuscitation Care

- ROSC is not equal with successful resuscitation. Resuscitation is successful if patient recovers without neurologic deficit.
- Maintain assisted ventilation if spontaneous breathing is ineffective!
- Never leave the patient till the advanced help arrived!
- Organise patient transport to a healthcare facility for post resuscitation care!

Used Guidelines:

Hungarian Resuscitation Guidelines forCouncil Adult BLS and AED www.reanimatio.com

AHA Guidelines for CPR and ECC 2010 www.heart.org

3. ALS – Advanced Life Support by Dr. Cserjés Andrea

Advanced life support (ALS) is a supportive care in case of cardiac arrest provided by healthcare professionals. The primary goal of ALS is to restore spontaneous circulation secondary goals (post-resuscitation care) is to provide the best possible long term quality of life in survivors. In a broader sense ALS also considersperi-arrest states (in this note we cannot give details).

ALS algorithm:

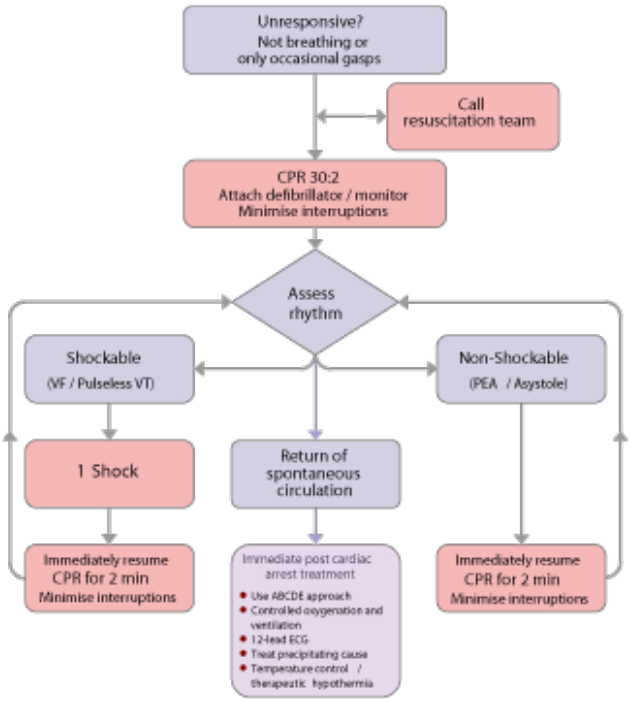
1. Unresponsive patient no sign of breathing or occasionally occurred gasping
2. Start BLS:
    - CPR: compression: ventilation ratio should be 30:2
    - Minimise interruption
    - change rescuer in every 2 minutes
    - frequency of chest compressions must be 100-120/min
    - if advanced airway has been performed, start ventilation with 10-12/min frequency
3. Attach defibrillator and monitor!
4. Assess rhythm! – shockable or non-shockable or return of spontaneous circulation

5/a. Shockable (VF, pulseless VT) 1 DC shock, then immediately resume CPR

5/b. Non-shockable (PEA, asystole) immediately resume CPR

5/c. Return of spontaneous circulation (ROSC): start immediately post cardiac arrest therapy

6. Assess rhythm after 2 minutes of CPR

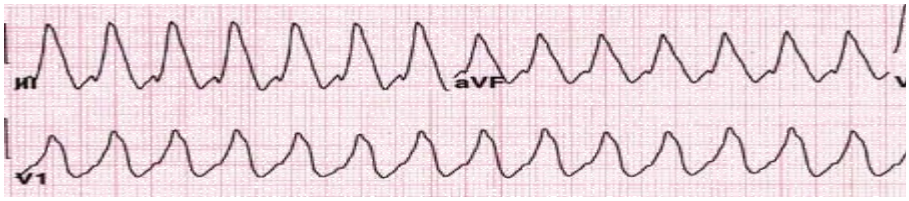


from https://lms.resus.org.uk

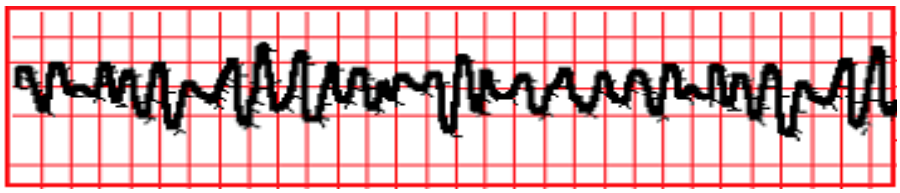
During ALS assessment of rhythm is the most important step to decide which type of arrhythmia occurs (shockable, non-shockable, pulse compatible rhythm). During the rhythm analysis CPR should be paused but only for 10 seconds.

Shockable arrhythmias:

Pulseless Ventricular Tachycardia (VT):

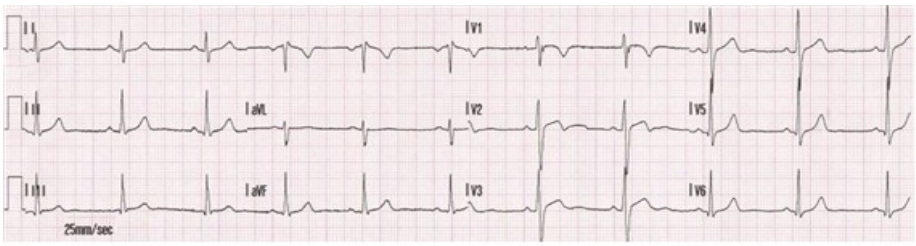


Ventricular Fibrillation (VF)

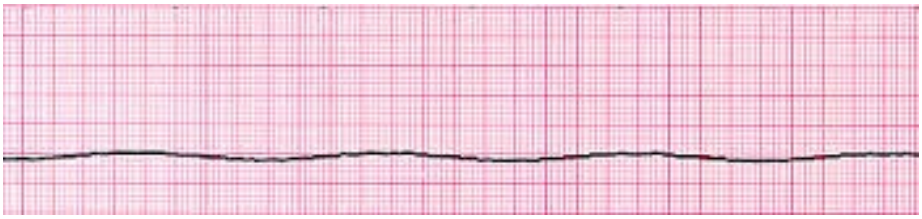


Non-Shockable arrhythmias:

Pulseless Electric Activity (PEA):



Asystole (ASY):



If pulse compatible rhythm is seen palpate carotid pulse.

- If no pulse can be felt resume BLS immediately!
- If pulse can be detected continue ROSC therapy

Management of Shockable Rhythm:

Deliver asynchronic shock with

- biphasic device: 150-200J then 150-360J
- monophasic shock: 360J then 360J

During shocks CPR must be paused, but not longer than 5-10 seconds

For the best conduction use electrode gel or stick-on defibrillator pads

No metal or wet thing should be near the site of defibrillation

Put the defibrillator pads more than 15cm from ICD or pacemaker

Safety:

- If metal electrodes used charge on the defibrillator!
- Order the rescuer handle oxygen mask to put the high flow oxygen 1m away!
- Order the rescuer provides CPR to stop compression!
- Before deliver shock check no one is in contact with the patient!
- Then finally check the monitor, and if the shockable rhythm is still present deliver shock.

Then continue CPR and ventilation immediately for 2 minutes.

Assess the rhythm again after 2 minutes of CPR.

If the monitor shows shockable rhythm deliver shock again with higher energy.

After the 3rd shock give 1mg adrenalin and 300mg amiodarone IV!

- Both of drugs facilitate ROSC and adrenalin improve coronary perfusion during CPR.

Give adrenalin in every 3-5 minutes if resuscitation continues.

Give 150mg amiodarone after the 4th shock then give 900mg/24h

Management of Non-Shockable Rhythm:

Continue BLS and perform IV access.

Give 1mg adrenalin IV then every 4 minutes.

During 2 minutes of BLS cycles:

- Insert peripheral IV or intraosseous cannula
- If indicated give IV drugs: magnesium, calcium, $NaHCO^3$, fluid, thrombolytic drugs.
- Perform advanced airway.

Pacemaker therapy if P wave asystole is seen on monitor.

Capnography

Ventilation with 100% FiO_2

Try to find reversible cause of the cardiac arrest 4H, 4T!

Lead the group (communication, planning, change rescuers, control CPR quality)

Reversible Causes of Cardiac Arrest: 4H, 4T

Hypoxia: the most common cause. Give high flow oxygen! Perform advanced airway!

Hypovolaemia: common cause in injured patients. Ask for previous events before cardiac arrest. Control bleeding if it is possible, give fluid and blood or blood derivates.

Hypokalaemia, Hyperkalaemia Acidosis: Previous events before cardiac arrest, and previous diseases, regularly taken medicines. Perform blood gas analysis if it is possible and treat electrolyte disturbances, give necessary therapy.

Hypothermia: environmental effect. Measure body temperature. Avoid unnecessary moving. Start to warm the patient up, and prepare for prolonged resuscitation!

Thrombosis, embolism: leading cause of death in Hungary.

- Patient history and previous events before cardiac arrest may indicate thrombosis as the underlying condition.
    - If AMI is proven on ECG after resuscitation (or during resuscitation) transport the patient to PCI laboratory!
    - If pulmonary embolism is suspected or identified thrombolytic therapy must be started and resuscitation has to be performed 60-90minutes!
    - If stroke is suspected thrombolysis is contraindicated!

Tension PTX: auscultation, percussion, and accompanying signs refer to tPTX like shifted trachea, subcutaneous emphysema. Perform immediate needle decompression, then insert a thoracic drain.

Tamponade: pericardial tamponade may be suspected according the patient's medical record, and it can be confirmed by US. Perform pericardiocentesis.

Toxins: the most difficult to identify toxins as reversible cause of cardiac arrest. Decontamination, then give antidote if it is possible!

Management after ROSC:

- ABCDE approach
- Perform advanced airway, and ventilate the patient under analgesia-sedation. The goal is normocapnia (EtCO2: 35-40 Hgmm), normoxyia (SatO2: 94-98% in COPD 90-94%).
- Monitor cardiac function, NIBP, Pulse, ECG, give catecholamines if the patient is hypotensive.
- Maintain renal function, monitor urine output. Regularly order blood gas analysis.
- Neurology: monitor blood glucose, prevent or treat seizures, therapeutic hypothermia (32-34°C)

Document accurately!

Used guidelines:

2010 AHA Guidelines for CPR & ECC

Post-resuscitation Care 2015 – Hungarian Resuscitation Council

2011 HRC Guidelines for adult ALS

4. Levels and Priorities of Emergency Care and the Rights of the Patient by Dr. Simon Marianna

The aging society, the educated and health conscious population and the developing medical interventions elevate the expenditures of modern healthcare, therefore worth considering and ranking have become inevitable factors in medical care. The boomingly elevating costs of healthcare creates imbalance between potential or real possibilities and growing social demands.

As a consequence of this imbalance came emergency medicine and emergency care into existence in the middle of 20th century. Optimal allocation of economic resources along professional considerations is the basic principle of emergency medicine.

The emergency medicine is characterised by:

Integration:patient centred care. Specialised medical teams, diagnostics and treatments assigned to different disorders.

Time dependency: the integrative approach guarantees rapid diagnosis and effective treatment of certain pathologies – particularly in emergency disorders with narrow time window.

Allocation: ensuring fair distribution of economic resources and preserving integrity need a professional allocation principle, which takes into consideration both the interests of the patients and the actual capacity of the healthcare system.

Emergency medicine approach along these principles unites the decision making procedure independently from its location. Different facilities (ambulance or emergency department) provide the same integrated and time dependent care based on the same approach.

Earlier therapy will result better outcome. Emergency care is limited in 24 hours.

The chain of emergency medicine is characterised by early recognition, early access, early differentiation, early stabilization and early causative therapy. The task of emergency medicine is the crisis intervention and the management of symptoms or groups of symptoms.

The allocation system of emergency medicine is called triage based on evaluation of chief symptoms, risk stratification and systemic risk management.

The main goal of emergency medicine is risk-orientated evacuation, the early recognition and therapy of high risk disorders. But emergency medicine has a preventive role too, recognise and treat the potentially high risk disorders avoiding aggravation.

Priorities of Emergency Care

1.A. Acute life-threatening conditions (without treatment: permanent disability or death)
1.B. Severe chronic diseases
2. Prevention, rehabilitation
3. Moderate acute or chronic diseases
4. Borderline cases
5. Medical care not related to current disease or injury

Time dependent priority order considering the severity of one's condition:

1. Medical Emergency
2. Possible Medical Emergency
3. Non-life-threatening condition, but without immediate treatment permanent disability may occur
4. Non-life-threatening condition, but the patient has severe pain and discomfort
5. Non-life-threatening condition, but requires immediate treatment for the patient's or social interest

The main goals of emergency care: decrease mortality, decrease organ damage, prevent permanent disability and release rapidly and effectively the patients' sufferings and pains.

The emergency medical services provides optimal patient care and navigation in 24 hours ensuring the equal opportunities. The goal of the emergency care is to stabilize the patient as soon as possible and navigate the patient to the optimal and definitive healthcare provider (to another department, to the general practitioner, to consultation or follow-up.) In some cases the emergency department can provide the definitive therapy too, and the patient may let home without further investigations.

To ensure the optimal function of the ED the best model is the "one-entering-point" model. Emergency care is exceptional comparing to other branches of medicine, because the patient's arrival is not prearranged but the patient's care cannot be delayed.

For the better care pre-hospital and in-hospital treatment should be integrated (common information and telecommunication system).

Difficulties and aspects of organisation of the optimal and equal emergency medical services:

- The number of the inhabitants are various in different areas.
- Ensure equal access to healthcare for every citizen.
- Effective communication between different healthcare provider facilities.

- Establishing minimum standards, conditions and facilities.

The levels of Emergency Medical Services:

Primary healthcare: everyone can seek medical care without referral letters. This form of healthcare relies on personal contact with healthcare professional. The main functions of primary healthcare: prevention, regular follow-ups, patient education, medical advice, treatment, rehabilitation and referral to ED or expert consultations. In urgent cases healthcare professionals have to provide **after-hour medical services**. In primary healthcare the diagnostic and therapeutic choices are limited.

If the patient requires acute medical care primary healthcare provider can refer the patient to outpatient care or emergency department. Ambulance is responsible for the patient transfer.

Outpatient care: Medical and surgical experts provide consultations (patients need referral). The main function of outpatient care: help primary healthcare, decrease the number in-hospital patients, but recognise the cases when in-hospital care is inevitable. Outpatient care has a wider access to diagnostic tests and treatment.

If the patient requires acute medical care primary healthcare provider can refer the patient to emergency department. Ambulance is responsible for the patient transfer.

Emergency medical services – Ambulance Services: provide medical emergency care at the scene and during the transport to the appropriate level of the medical services (outpatient care or ED).

A dispatcher centre receives the calls and order for every call a priority level and the required ambulance crew. OMSZ (Hungarian Ambulance Service – call: 104) provide 24 hours of equal ambulance care for everyone.The levels of priority: immediate, within 2 hours or over 2 hours.

Ambulance care is required: medical emergencies and possible medical emergencies, non-life-threatening conditions but with threatening permanent disability, conditions with severe pain, altered mental state, child delivery and in cases of psychiatric emergencies (suicidal behaviour, threatening behaviour). In case of natural disasters, catastrophes or mass accidents ambulance care has an essential role.

Ambulance service is also required formonitored patient transport between healthcare facilities.

The ambulance units of OMSZ:

- ambulance van with a driver and an ambulance assistant
- ambulance van with a driver, an ambulance assistant and an ambulance officer

- ambulance van with a driver, an ambulance assistant and an emergency physician
- ambulance motorbike
- ambulance car with a driver and a doctor
- helicopter emergency services
- mobile intensive unit
- neonatology ambulance unit
- ambulance boat
- mass accident unit

In-Hospital Emergency Care: Emergency Department (Level 1-3 depend on the resources of department)

Emergency department deal with all acute cases and critically ill patients not exclusively focuses on certain groups of disorders. Main goal is to maintain vital functions using ABCDE approach.

Emergency department is a legally defined department for outpatient and inpatient medical care of persons without prearranged appointment presenting on ED by their own means or transported by ambulance with acutely occurred symptoms or symptoms required acute care. The patient's stay in ED is limited to 24 hours. After 24 hours the patient has to be disposed from ED. In controversial cases the shift supervisor emergency physician makes the final decision.

If the patient's state surpasses the progressivity level of the emergency department, immediate disposition to higher progressivity healthcare facility is required.

Functions of ED:

- Patient admission and examination
- Stabilization of the patient's current condition
- Making primary diagnosis (it may not be the final diagnosis)
- Basic therapeutic intervention
- Decision making about the patient's further admission to other departments or discharge.

Healthcare professionals work in shifts in the same number at all time. Besides emergency healthcare professionals, diagnostic and laboratory background and many expert consultants guarantee the possibly most optimal emergency care of the always changing quantity of acutely ill or injured patients.

Specialized Emergency Centres exist in major cities and universities:

- with invasive cardiologic laboratory and cardiac surgery
- with toxicology
- with adult and paediatric burn surgery
- with neurosurgery and stroke unite
- with infectology

Patients' Rights:

The patients' rights could be adapted to a state's law in 3 different way:

- Exists an independent law for patients' rights (France, Denmark, Finland, Iceland, Norway, Greece, Latvia, Lithuania and the Netherlands).
- Regulations for patients' rights in different laws and ethical codex (Hungary and Belgium)
- Charter of the patients' rights (United Kingdom, Ireland, Slovakia, Portugal and Germany)
- European Charter of Patients' Rights (2002)

Act CLIV of 1997 on Health [from the website (www.ecoi.net) of European Country of Origin Information Network]:

Fundamental Principles:

(1) In the course of delivering healthcare services and measures, the rights of patients shall be protected. A patient's personal freedom and right of self-determination shall be restricted exclusively in cases and in a manner justified by his health status and defined in this Act.

(2) It shall be required to enforce equity throughout the utilization of healthcare services.

(3) The primary means of improving health are to promote health and to prevent disease.

(4) The set of fundamental professional requirements within the healthcare services shall not depend upon forms of ownership and operation, and shall be based exclusively upon the professional contents of the service.

(5) Structured by levels of care and focussing on man, the healthcare delivery system shall be designed so as to meet the needs as defined by the health status of individuals suffering from diseases of different types and severity; furthermore, it shall be based on evidences and cost-effective procedures.

In (emergency) cases the vulnerability of patients has to be decreased to the possible minimum level, and their dignity has to be preserved.

Right to medical care of good quality

Right to dignity

Right to communication

Right to leave the healthcare facility

Right to information

- The patient has right to ask any questions regarding his/her condition, to receive information about expected results of the planned diagnostic procedures and treatments, and about the possible complications.
- The patient has right to know the staff's name, rank and qualifications.
- The patient has the right to receive information in a way he/she can understand considering his/her age, qualification, religion, common knowledge and current mental and spiritual state.
- Access to interpreter or sign language interpreter
- Access to his/her medical records

The legally incompetent patient: the decisions are made with the patient but not by him/her. In these cases a previously authorised person will make the decisions in the name of the patient.

- If there is nobody authorised previously by the patient, his/her legal representative, his/her competent child who lives with the patient in the same household, or his/her parent, or his/her sibling, or his/her grand sibling, or his/her competent child who does not live in the same household, or his/her parent, or his/her sibling, or his/her grand sibling.

Right to self-determination

- All diagnostic procedures and treatments requires the informed consent of the adult competent patients.
- For invasive interventions the patient has the right to give or withhold consent. If the patient gives his/her consent, he/she must be give written and verbal consent and two witnesses have to sign it too and the patient has the right to withheld it whenever he/she want it without further explanation.

Right to refuse medical care if the patient does not mean a potential danger (infective diseases) to others.

- Life-saving diagnostics and therapeutic interventions against the patient's will can be performed only if
    - the patient is pregnant and presumably capable to deliver a new-born
    - the patient is incompetent.

Right to confidentiality

The patients' obligations:

- Shall give information about his/her condition, previous diseases and regular medications, about potentially harmful or contagious diseases and about his/her previous medical legal statements.
- Shall give valid and true information about his/her identity.
- Shall respect other patients' rights.
- Practising his/her rights shall not aggrieve the rights of healthcare professionals.

Patients' Rights in the Emergency Care:

In the emergency care prompt decision making and lack of time shall not aggrieve the patients' rights. Although, in acute life-threatening medical emergency the emergency physician may not take the patient's opinion into consideration. The patient cannot choose an other healthcare facility because of his/her acute condition. For the lack of time or extreme circumstances (mass accident) the patient will not receive fully comprehensive information about the planned treatment and interventions.

Act CLIV of 1997 on Health [from the website (www.ecoi.net) of European Country of Origin Information Network]: Section 125

In emergencies, irrespective of time and place, the healthcare worker shall provide first aid toany person in need, to the extent that said healthcare worker can provide such aid under givenconditions with the implements available, and/or shall immediately take necessary measures.In cases of doubt, the existence of an emergency shall be presumed.

Rights of healthcare professionals:

Act CLIV of 1997 on Health [from the website (www.ecoi.net) of European Country of Origin Information Network]: Section 139 – Protection of Healthcare Workers

A healthcare worker and all other workers employed by a healthcare provider qualify aspersons performing a public service.

Right and Obligation to Develop Professionally

Obligation to maintain confidentiality

Obligation to provide information

Obligation to document

Right to choice of methods of examination and therapy

Act CLIV of 1997 on Health [from the website (www.ecoi.net) of European Country of Origin Information Network]: Section 130

- The attending physician, in his area of responsibility, shall be authorized to issue instructions to healthcare workers participating in patient care. The instructions shall include a clear specification of the task to be completed, the place and time of completion, and, if necessary, the names and sphere of activity of additional healthcare workers to be requested to participate.
- The healthcare worker participating in the care shall
    - execute the instructions in accordance with the conditions set forth in them and in keeping with the code of practice of the profession,
    - immediately notify the attending physician, or if this is impossible, another physician participating in the care of the patient, if an unforeseeable event or event leading to a deterioration in patient condition occurs during implementation,
    - immediately make it known to the attending physician, or if this is impossible, to another physician participating in the care of the patient, if in his opinion, execution of the instructions would have an unfavorable influence on the condition of the patient, or if he has some other concern,

- refuse to execute the instructions, simultaneously notifying the attending physician, if, according to knowledge he is expected to possess, compliance would threaten the life of the patient or lead to permanent impairment to patient's health that would otherwise not be a necessary outcome of treatment.

- The participating healthcare worker, if instructed to execute the instruction despite the provisions set forth in Paragraph c) of Subsection (2), shall be authorized to request that said instructions be communicated in writing.

- Within the framework of the instructions, the healthcare worker, in keeping with his own professional competency and experience, shall make his own decisions on the manner and order of executing the tasks he is to complete.

Ethical Principles for Healthcare Professionals:

- The patient's health is elemental.
- Respect human rights, healthcare professionals shall not participate in torture, cruelty or humiliation.
- Respect the autonomy of the patient.
- Continuous development in profession.
- Truthfulness.
- Keep professionally independent.
- Respect confidentiality.
- Shall not use confidential information for personal interests.
- Insist to legal, moral and ethical principles, and make others aware too.

<u>Legal Background of Emergency Care in Hungary:</u>

- **THE FUNDAMENTAL LAW OF HUNGARY (25. April 2011.)**
- **Act LXIII of 1992 on the Protection of Personal Data and the Publicity of Information of Public Interest**
- **Act CLIV of 1997 on Health**
- **Act 1997 XLVII**

- **Regulation of Ministry of Welfare 19/1998. (VI. 3.)**
- **Regulationof Ministry of Health, Social and Family Affairs 60/2003. (X. 20.)**
- **Regulation of Ministry of Health 2/2004. (XI. 17.)**
- **Regulation of Ministry of Health, Social and Family Affairs 47/2004. (V. 11.)**
- **Regulationof Ministry of Health 10/2005. (IV. 12.)**
- **Regulationof Ministry of Health5/2006. (II.7.)**
- **Regulationof Ministry of Health 52/2006. (XII. 28.)**
- **Act 2011 CXXVIII**

5. Triage in the EDby Tóth Lajos

IT'S UNDER REVISION.

6. Management of Chest Pain in the ED by. Prof.Dr.RudasLászló

Chest Pain is a fairly common entity in ED. 25% of patients call ambulance because of chest pain. Chest pain is a symptom, which may associate with either a banal musculoskeletal disease or with a life-threatening medical condition like myocardial infarction, aortic dissection, tension pneumothorax, perimyocarditis, pericardial tamponade or pulmonary embolism. Any delay in these life-threatening conditions might be fatal.

To differentiate between conditions cause chest pain physical examination and certain diagnostic procedures are essentials.

Medical help-seeking behaviour of patients with chest pain:

Aetiology of Chest Pain	Patients' first choice Healthcare Provider		
	General Practitioner	Ambulance	Emergency Department
Cardiac	20%	69%	45%
Musculoskeletal	43%	5%	14%
Pulmonary	4%	4%	5%
Gastrointestinal	5%	3%	6%
Psychiatric	11%	5%	8%
Others	16%	18%	26%

According to the table above, although most patients go directly to ED with cardiac chest pain, many of them call ambulance or see the GP first. The mean delay in the definitive treatment of ischemic myocardial infarction is 1.5-2h both in the United States and Europe. Elderly patients, women, and disadvantaged patients seek help even later.

1. **Acute Coronary Syndrome – Ischaemic Chest Pain**

Coronary artery atherosclerosis is a disease of civilization with many risk factors (genetic, environmental and lifestyle factors). Coronary atherosclerosis leads to two major group of cardiac diseases:

Stable Angina is a chest pain due to narrowing coronary arteries. Symptoms of stable angina occur with the same amount of physical exertion and the pain is relieved by rest or medicine. Episodes of stable angina tend to be alike.

Acute Coronary Syndrome is a collective noun for ischaemic chest pain with rapid onset and significant discomfort. ACS may present in the form of worsening symptoms of stable angina (exaggerated pain or angina occurs in resting position) or newly present, severe cardiac chest pain.

Infarction means myocardial necrosis due to ischemia.

Diagnosis of Myocardial Infarction:

- Typical clinical presentation and symptoms.
- ECG changes
- Echocardiography: Hypokinetic or akinetic heart wall
- Laboratory findings

Types of Myocardial Infarction:

A) *Spontaneous Myocardial Infarction:*

- Elevated biomarker (Troponin) level without any previous cardiac intervention (coronary angiography, PCI, CABG).
- Clinical signs of ischemia (angina, or angina equivalent pulmonary oedema)
- New repolarization disorder: new alteration of ST-T, or new LBBB
- Presence of pathologic Q wave
- Echocardiography or MRI proved heart wall motion abnormality
- Thrombus is diagnosed in the coronary artery by coronary angiography (or post mortem autopsy)

Pathomechanism of Spontaneous Myocardial Infarction

- **Atherosclerotic plaque rupture:** the unstable plaque cracks up due to shear stress on arterial wall, initiates the blood clotting cascade and finally forms an occlusive or semi-occlusive thrombus.
- **Seconder infarction:** caused by the disproportion between myocardial oxygen supply and demand. If myocardial oxygen consumption is elevated (e.g. fever, sepsis, hyperthyroidism) an already existing narrowing on the coronary artery may not provide sufficient blood flow. This condition specially occurs in elderly patients with serious comorbidities but extreme strain may cause seconder myocardial infarction even in patient with normal arteries.

Atypical Symptoms: Patients may have Spontaneous Myocardial Infarction without typical symptoms (this form is usual in elderly female patient with diabetes mellitus)

- Dyspnoea

- Nausea and vomiting
- Presyncope/syncope

B) *Sudden Cardiac Death*

In some cases of SCD Myocardial Infarction is suspected due to anamnestic symptoms and ECG signs, but could not be proved by laboratory findings.

C) *Late Stent Thrombosis*

Myocardial Infarction caused by coronary stent reocclusion with the same criteria of Spontaneous Myocardial Infarction.

D) *Periprocedural Myocardial Infarction*

Myocardial Infarction due to coronary angiography, PCI or CABG.

Role of Troponins in the diagnosis of MI

Troponin-T and Troponin-I are highly specific markers of MI. Cardiac cell death causes an early release of Troponins into the blood.If blood sampling is performed too early, Troponin level will not beelevated, in these cases repeated Troponin measurement is needed 3 hours later to diagnose or exclude MI. Dynamic change of Troponin level is fairly specific to MI. AlthoughTroponin level may be elevated in other diseases apart from MI (See list below) but this elevation isconstant.

Non-MI diseases with elevated Troponin level:

- Chronic Renal Failure
- Severe Heart Failure
- Pulmonary Embolism
- Myocarditis
- Acute CNS disorder (SAH, cerebrovascular emergencies)
- Aortic dissection, aortic valve disease, Hypertrophic Cardiomyopathy
- Myocardial contusion
- Hyperthyroidism
- Taku-Tsubo Cardiomyopathy
- Infiltrative myocardial diseases
- Cardiotoxic drugs
- >30% burning
- Rhabdomyolysis

- Critical conditions like acute respiratory failure

ST Segment Elevation Myocardial Infarction (STEMI) and Non-ST Segment Elevation Myocardial Infarction (NSTEMI)

STEMI: total occlusion of an epical coronary artery causes transmural ischemia and consequently myocardial necrosis. ST segment elevation is the result of transmural ischemia. Time window for saving myocardium is narrow, therefore STEMI patients need prompt protocol treatment.

In non-ST segment elevation Acute Coronary Syndrome (NSTE ACS) laboratory findings confirm the myocardial infarction.

Unstable Angina Pectoris (UAP): NSTE ACS without Troponin elevation.

NSTEMI: NSTE ACS with Troponin elevation.

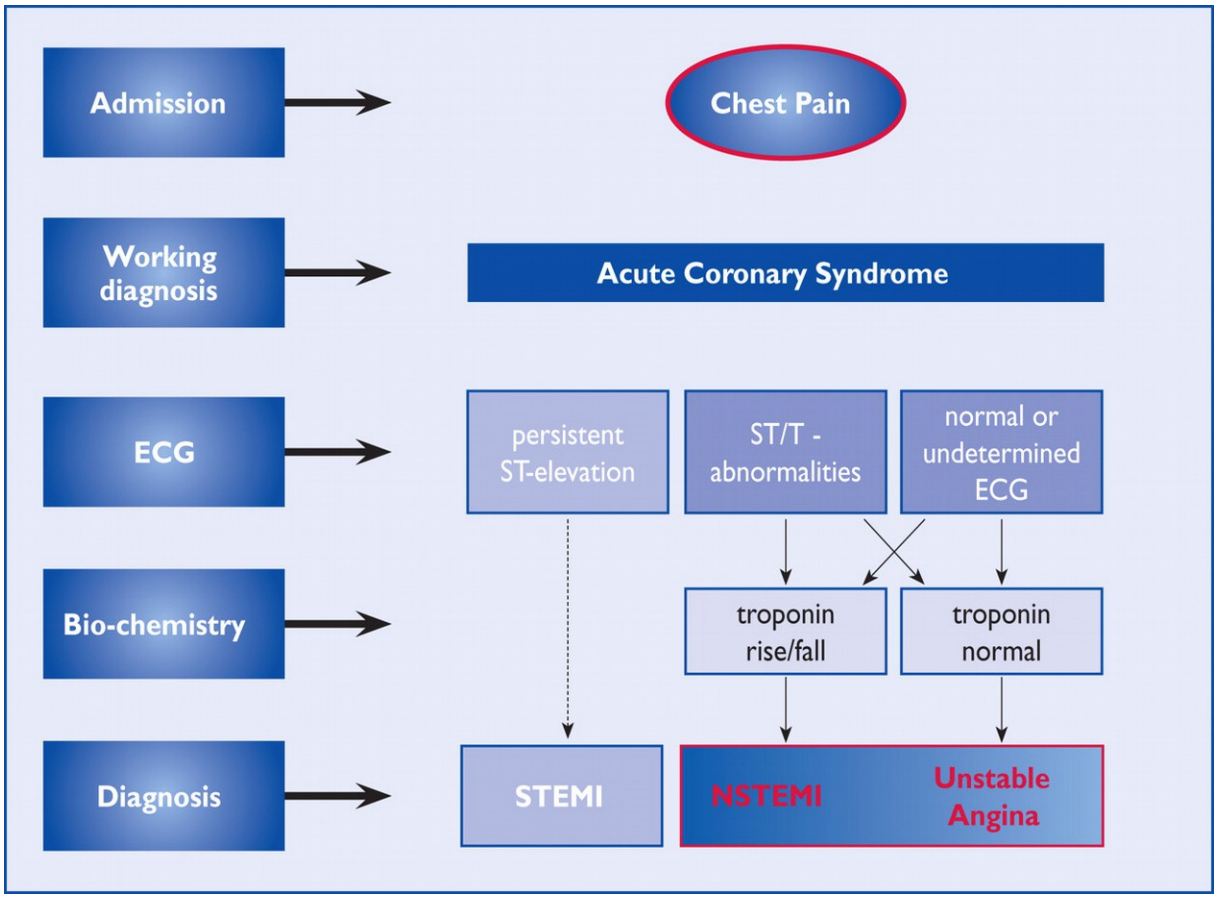


Figure 1. Acute coronary syndrome (ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation European Heart Journal (2011) 32, 2999–3054)

ECG Criteria of STEMI

- Timing of ST segment elevation:
    - ST segment elevation is seen more than 20 minutes
- Elevation of ST segment:

- Precordial leads (V2-3)
    - in males under 40 > 0,25 mV
    - in males above 40 > 0,2 mV
    - in females > 0,15 mV
- Other leads > 0,1 mV

- New-onset Left Bundle Branch Block with chest pain is STEMI equivalent
- Isolated posterior infarction (see ECG 1): occlusion of circumflex branch of left coronary artery is STEMI equivalent
    - V1-3 ST segment depression > 0,05 mV
- Left Main occlusion (see ECG 2):
    - ST segment elevation in aVR
    - multilead ST segment depression in at least 6

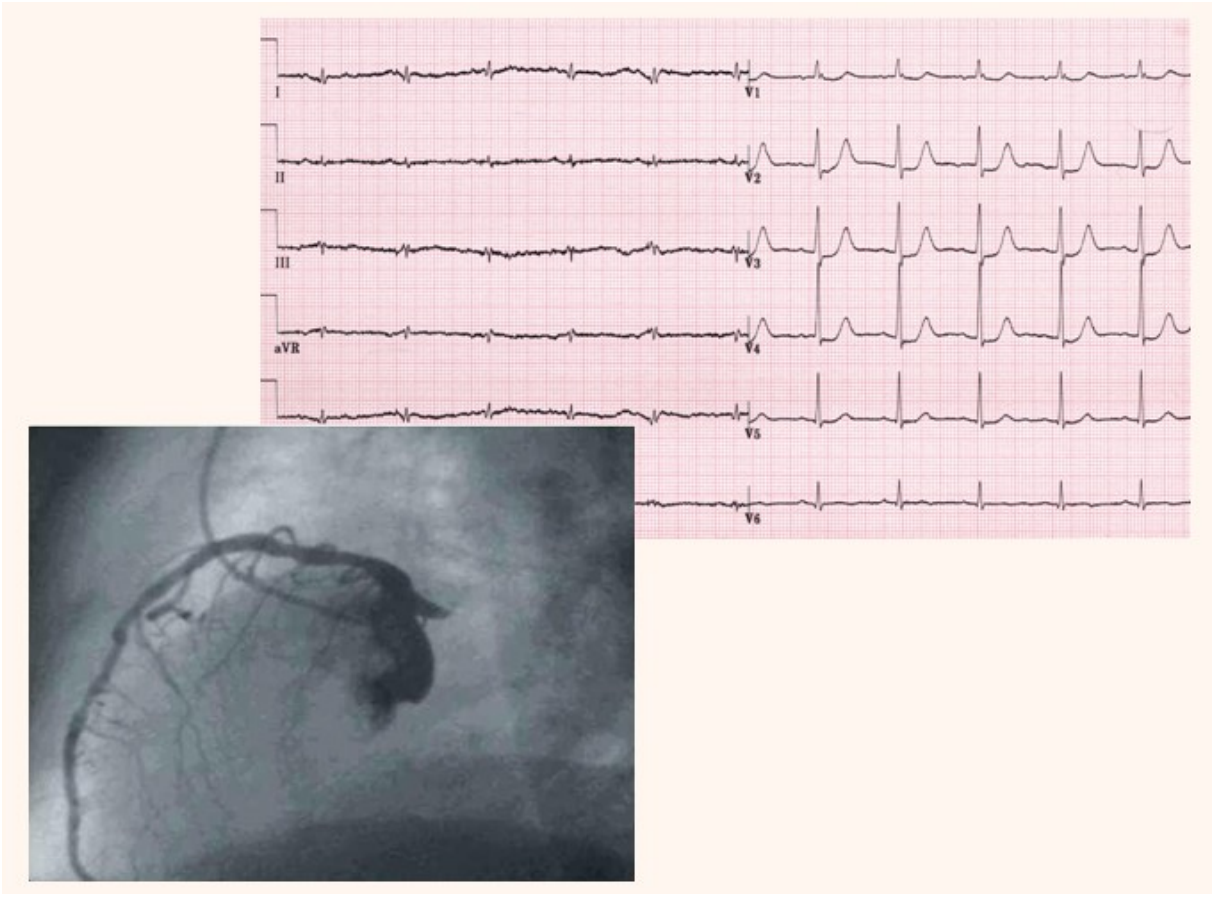


Figure 2. Isolated posterior AMI from KÖR-ITO archives, ECG and coronary arteriogram with occluded left circumflex artery

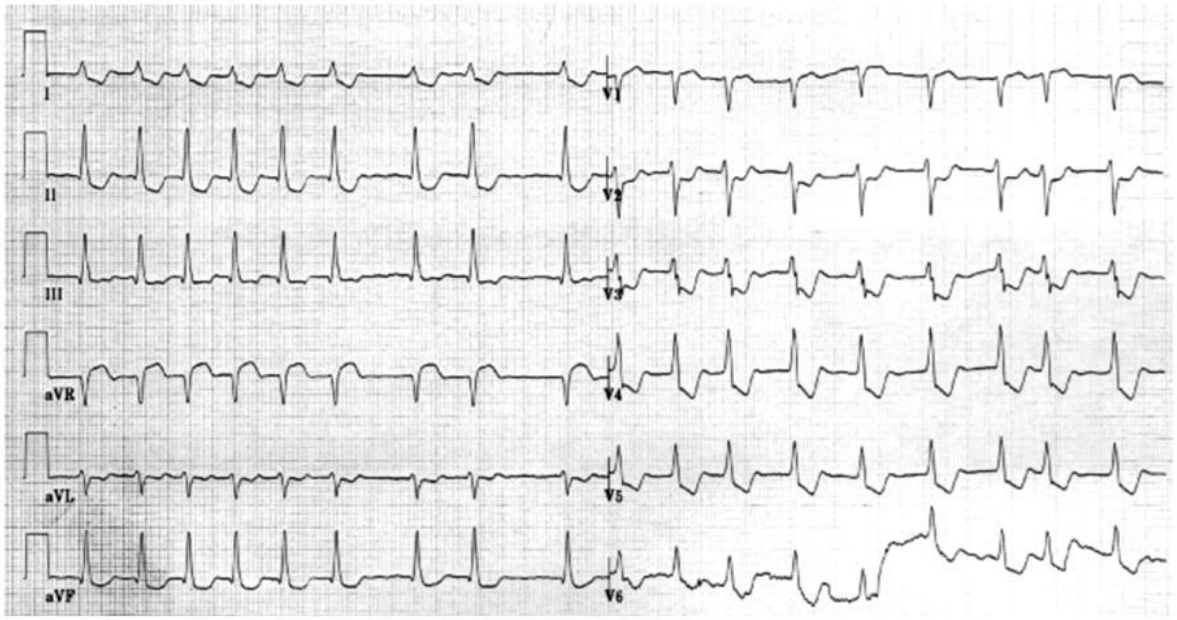


Figure 3. Critical Left Main stenosis and sever right coronary artery disease from KÖR-ITO archives, ECG with multilead ST depression and ST elevation in aVR

Management of STEMI

Like other emergency cases STEMI has a well protocoled treatment with narrow time windows. Myocardium necrotises after 6h of coronary artery occlusion. If the occluded coronary artery is reopened in 6 hours the myocardium (fed by the culprit artery) may be saved. If the symptoms are intermittent or the myocardium is preconditioned by previous ischaemic episodes time window may be wider.Reperfusion is indicated in every patient having symptoms less than 12 hoursif there is no contraindication. In patients having symptoms more than 12 hours reperfusion is indicated in two cases: symptoms recur or ECG changes. If symptoms occur more than 24 hours, and no ischaemic sign is present, reperfusion is contraindicated in stable patients.

Reperfusion Strategies

- Percutaneous Coronary Intervention (PCI): first choice in reperfusion therapy
    - effective reperfusion
    - stent implantation particularly the drug eluting stent implantation- is a long term solution for occluded coronary artery and prevents reocclusion
- Thrombolysis: if immediate PCI cannot be performed

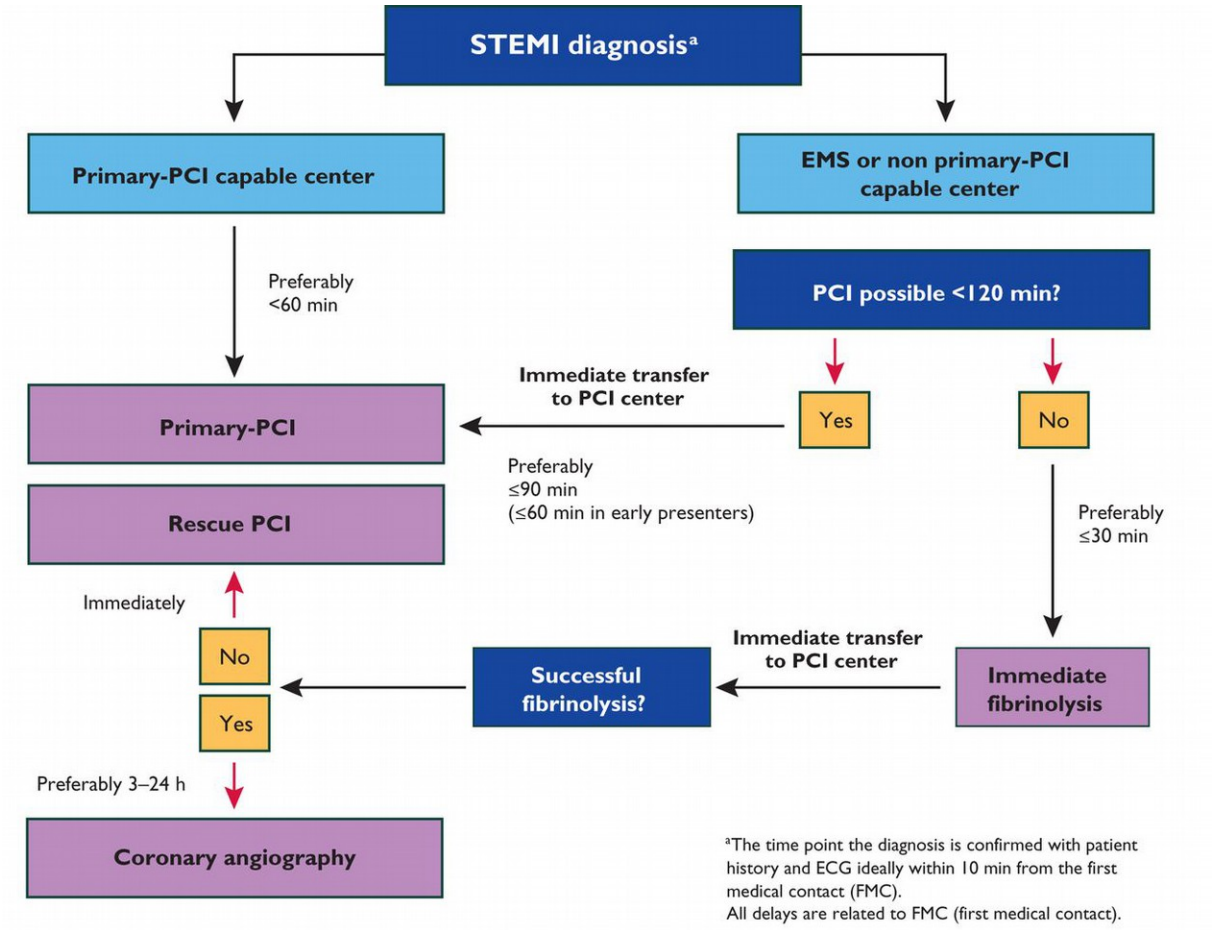
- after successful thrombolysis PCI is indicated within 3-24 hours for definitive treatment
- if thrombolysis was unsuccessful rescue PCI is indicated

For Best Practice STEMI Management a common protocol must be implemented at all level of healthcare (ambulance, ED, GPs). Time windows are used as the most frequently monitored quality indicators of STEMI Management.

Additional Therapy for PCI

- **Anticoagulants:**
  - Unfractionated Heparin (UFH): 70-100 IU/kg IV (UFH is used in all PCI centres in Hungary)
  - Low Molecular Weight Heparin (LMWH) – Enoxaparin 0,5mg/kg IV
  - Bivaluridin (0.75 mg/kg IV bolus, initially, followed by continuous infusion at rate of 1.75 mg/kg/h for duration of PCI) not used in Hungary
- **Thrombocyte Aggregation Inhibitors (TAI):**

  DOUBLE TAIAspirin+Clopidogrel/Plasugrel
  - thromboxane synthase inhibitors: Aspirin (acetyl salicylate) initially 150-300mg PO then 75-100mg /day
  - adp receptor/p2y12 inhibitors:
    - Clopidogrel initially 600mg PO, then 75mg/day
    - Plasugrel initially 60mg PO, then 10mg/day
  - GP iib/iiia inhibitors (not used routinely)
    - Eptifibatide or Tirofiban



Cath = catheterization laboratory; EMS = emergency medical system; FMC = first medical contact; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction.

Figure 4. Prehospital and in-hospital management, and reperfusion strategies within 24 h of First Medical Contact (from ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevationEuropean Heart Journal (2012) 33, 2569–2619)

Thrombolytic Therapy:

- Absolute contraindications:
    - Any previous history of haemorrhagic stroke
    - History of stroke within 6 months
    - Head trauma or brain surgery within 6 months
    - Known intracranial neoplasm or AV malformation
    - Suspected aortic dissection
    - Internal bleeding within 6 weeks
    - Active bleeding or known bleeding disorder
    - Major surgery, trauma, or bleeding within 3 weeks

- Traumatic cardiopulmonary resuscitation within 3 weeks

- Relative contraindications:
  - Oral anticoagulant therapy
  - Acute pancreatitis
  - Pregnancy or within 1 week postpartum
  - Active peptic ulceration
  - Transient ischemic attack within 6 months
  - Dementia
  - Infective endocarditis
  - Advanced liver disease
  - Uncontrolled hypertension (systolic blood pressure >180 mm Hg, diastolic blood pressure >110 mm Hg)

Thrombolytic agents:

- Alteplase (tPA):
  - 15mg IV bolus
  - Phase 1: 0,75mg/kg/30mins (max 50mg)
  - Phase 2: 0.5mg/kg/60mins (max 35)
- Tenecteplase (TNK-tPA):
  - <60kg 30mg IV bolus, 60-70kg 35mg IV bolus, 70-80kg 40 mg IV bolus, 80-90kg 45 mg IV bolus, >90kg 50 mg IV bolus

Additional drugs:

- UHF: 60 IU/kg (max 4000 IU), then according to aPTI (target range: 50-70sec.)
- Clopidogrel 300 mg PO, then 75 mg/day (older than 75 only 75mg PO)
- Aspirin 150-300 mg PO, then 75-100 mg/day

Additional Therapy of STEMI

- Morphine
- Oxygen supplementation
    - NB: Avoid hyperoxia!
- IV beta-blockers in patient with tachyarrhythmia and hypertension
- AVOID NSAIDs!

Management of NSTEMI

In patients with NSTEMI the urgency of the treatment is based on risk stratification. For risk stratification symptoms, vital signs and GRACE grading are considered.

Find GRACE calculator on:

http://www.outcomes-umassmed.org/grace/acs_risk/acs_risk_content.html

- High risk patient: therapy resistant severe chest pain, cardiovascular instability, life-threatening ventricular arrhythmias
    - ***PCI is indicated within 2 hrs!***
- Moderate risk patient: > 140 GRACE points without high risk signs
    - ***PCI is indicated within 24 hrs!***
- Low risk patient
    - ***PCI is indicated within 72 hrs!***

The additional treatment of anticoagulation and TAI is the same as for STEMI.

Cardiogenic Shock: A Complication of AMI

Shock is a life-threatening condition caused by acute, generalised circulatory failure, which leads to cellular dysoxia (laboratory findings: elevated lactate level).

Clinical Signs of Shock:

- Insufficient peripheral circulation: pale, cyanotic, cool, sweaty skin with mottling and prolonged capillary refill time
- Renal failure: urine output < 0.5ml/kg/h
- CNS symptoms: drowsiness, altered mental state

Classification of AMI related Heart Failure:

- Killip class 1: no clinical sign of heart failure
- Killip class 2: rales or crackles in the lungs, S3 gallop
- Killip class 3:acute pulmonary oedema
- Killip class 4: cardiogenic shock

Incidence of Cardiogenic Shock

- USA 50,000 cases/year, Europe 65,000 cases/year
- In STEMI patient the prevalence of Cardiogenic Shock is doubled
- 15% of Cardiogenic Shock can be seen on admission, 85% present in hospital phase
- Mortality rate: 40-80%

Factors lead to Cardiogenic Shock:

Mechanical Complications: acute mitral regurgitation or acutely present septal defect, free-wall rupture caused prompt pericardial tamponade.

Functional Complications:

1. Left Ventricular Insufficiency: (caused by left main occlusion, three-vessel disease, right coronary occlusion, LAD occlusion - anterior wall STEMI)
    - LV insufficiency is a result of:
        - Function lost by necrotised myocardium
        - Temporarily lost function of the stunned but not necrotised myocardium.
    - Pathomechanism of LV Insufficiency: decreasing filling pressure causes hypotension and consequently elevates vascular resistance. Decreasing perfusion of coronary arteries causes additional myocardial ischemia. Elevated LV filling pressure leads to pulmonary venous congestion and pulmonary oedema.
2. Right Ventricular Insufficiency: one third of inferior AMIs (caused by proximal occlusion of right coronary artery) affects RV function.
        - Signs of RV Insufficiency: hypotension, jugular vein distension, absence of pulmonary oedema

    NB: Give 500-1000 ml (not more) fluid replacement to maintain preload! Do not give vasodilators or diuretics!

Treatment of Cardiogenic shock

1. **PCI** may restore cardiac function, and improve the patient's life expectancy. Therefore in cardiac shock patient PCI must be performed as soon as it is possible (IA).
2. **Intra-aortic balloon pump (IABP)** decreases the afterload and improves the myocardial perfusion, but IABP has its limitation it cannot regenerate the lost cardiac output. IABP is used during PCI of an instable patient, CABG or as a temporary therapy for keeping the patient alive till the definitive therapy, cardiac surgery, is carried out.
3. **Vasopressors:**
    - Benefits: maintained coronary perfusion, positive inotropic effect
    - Disadvantages: elevated afterload increases transmural pressure and myocardial oxygen demand.

The most advantageous positive inotropic agent is norepinephrine.

Initial dosage: 0.05-0.5 mcg/kg/hrs

Positive inotropic agents increase the intracellular Calcium (Ca^{2+}) level with the stimulation of cAMP production (dobutamine) and the inhibition of the cAMP dissimilation (milrinone) and as a result positive inotropic agents enhance myocardial contractility. But elevated Ca^{2+}level causes relaxation disorders, and spontaneous depolarization, which lead to arrhythmias. Levosimendan does not elevate the intracellular Ca^{2+} level, but stabilise the bind between TroponinC and Ca^{2+} in systole, therefore levosimendan has no proarrhythmic effect. All positive inotropic agents have peripheral vascular effect - vasoconstrictors (norepinephrine), vasodilators (dobutamine).

NB: Avoid positive inotropic agents with vasodilator effect in severe hypotension ($BP_{systolic}$< 75mmHg)!

2. Non-ischemic Chest Pain:

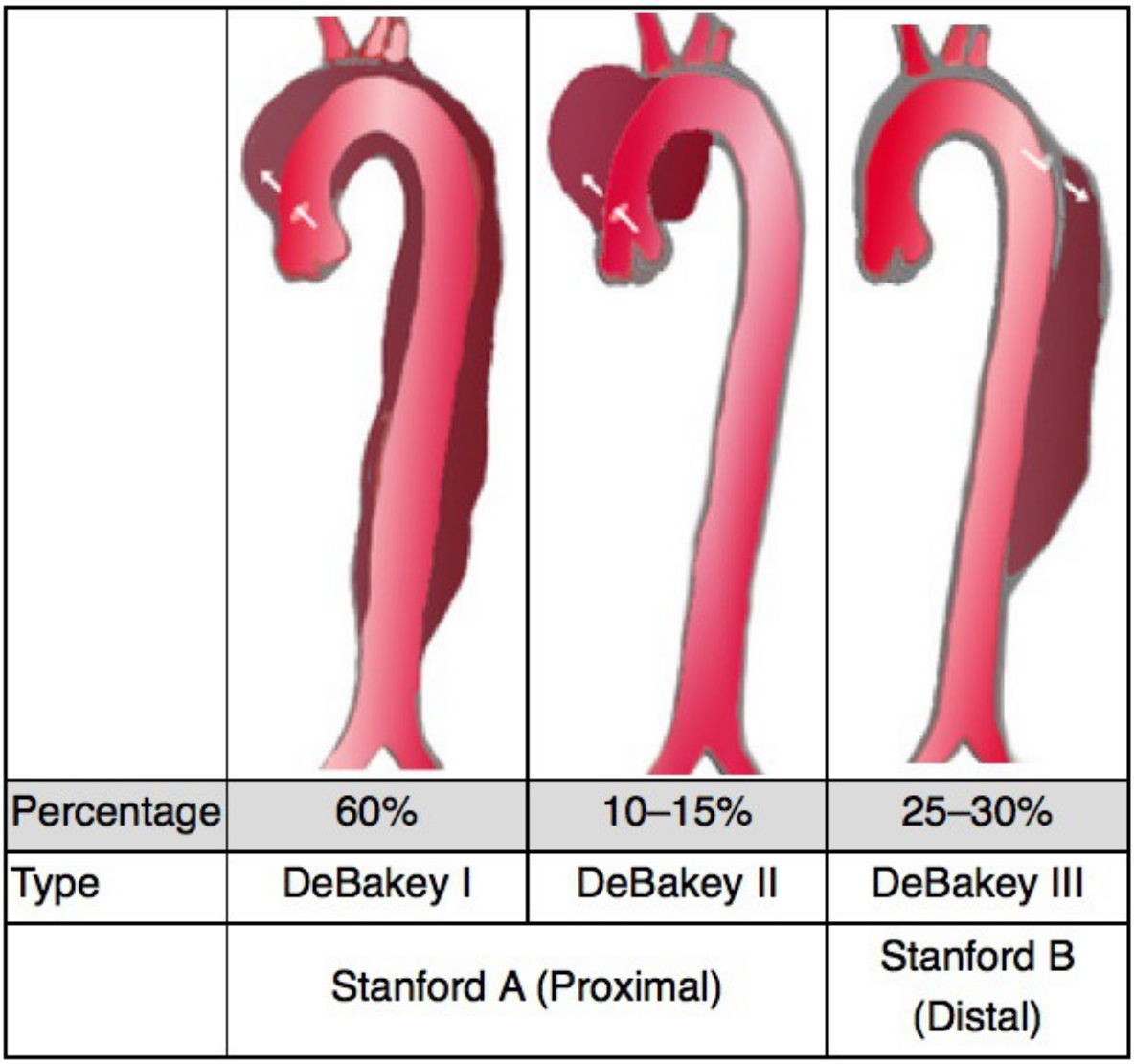
1. Acute Aortic Dissection
2. Acute Pulmonary Embolism
3. Boerhaave syndrome (Oesophageal rupture)
4. Pneumothorax
5. Acute Perimyocarditis

6. Pericardial Tamponade

1. ***Acute Aortic Dissection***

- Incidence: 2000 cases/year in the USA, mean age of patients with acute aortic dissection is between 50-70 years
- Mortality: 2-3% in the first 48 hrs, then it may be up to 75% without treatment in the next 2 weeks
- Pathomechanism: longitudinal splitting of the aortic intima and muscular media. The blood flows between two layers, and forces them apart forming a pseudo lumen. The most of the patients have history of hypertension and aortic dissection occurs more frequently in patients with Marfansy.
- Stanford Classification
    - Stanford A (DeBakeyi-ii): The tear originate from the ascending aorta and may reach the descending aorta too
    - Stanford B (DeBakey iii): The tear originate from the descending aorta distal to the left subclavian artery

Classification of aortic dissection



Percentage	60%	10–15%	25–30%
Type	DeBakey I	DeBakey II	DeBakey III
	Stanford A (Proximal)		Stanford B (Distal)

Figure 5. Classification of Aortic Dissection (source Wikipedia)

- Signs and Symptoms are vary with the location of the dissection (renal, neurologic or cardiac).
    - **Chest Pain:** Unbearable pain with a special tearing characteristic. The pain is the worst at the onset of the symptoms and it is difficult to alleviate even using IV opioids. The pain often radiates to the interscapular or abdominal region.
    - **Aortic regurgitation (diastolic) murmur** (in 30% of patients)
    - **Neurologic symptoms** (in 20% of patients) – SYNCOPE!
    - **Pulse deficit** (in 15-30% of patients)

- **>20 mmHg difference** between the blood pressure measured in both arms (not very specific might be normal variant)
- **Chest X-ray:** widened mediastinum, blurred aortic contour, dislocated aorta, pleural fluid
- Transthoracic Echocardiography first step diagnostic imaging
- Transoesophageal Echocardiography/**CT angiography**/MRI has high sensitivity and sensibility (CT angiography is used routinely)

- Complications:
  - AMI – dissection reaches the coronaries
  - Aortic Valve Insufficiency/Pericardial Tamponade – dissection reaches the aortic valve
  - Renal impairment – dissection reaches renal arteries
  - Stroke – dissection affects carotid perfusion

2. *Acute Pulmonary embolism*

   Pulmonary embolism (PE) is a fairly common emergency condition. Estimations suggest, that once in a lifetime almost 5% of the population get PE, and 5-10% of hospital inpatient mortality is related to PE. Although PE is a common condition it is often underdiagnosed or misdiagnosed.

Classification of acute PE: PE is classified by its effect on the circulation.

- Massive PE (5%): PE with signs of shock

  The sudden obstruction of the pulmonary arteries causes acute right heart failure. Systemic vasoconstriction occurs as a compensatory response to elevate the venous inflow to the right atrium to maximise the right ventricle stroke work. Massive PE often causes seconder myocardial ischemia by hypotension and consequently decreased coronary artery perfusion. ECG changing and Troponin level elevation is common.

- Submassive PE: PE in stable patient with signs of right heart dysfunction
- Non-massive PE:

- non-specific symptoms:
    - Dyspnoea (70%)
    - Pleural chest pain (40%)
    - Coughing (35%)
    - Malaise
    - fatigue
- presence of risk factors:
    - history of previous thrombosis/embolism
    - history of trauma, surgery, immobilization, pregnancy, contraceptives or malignant disease
- Well's Score for PE

Criterion	Points
Suspected DVT	3.0
An alternate diagnosis is less likely than PE	3.0
Heart rate > 100 beats/min	1.5
Immobilization or surgery in the previous four weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1.0
Malignancy (on treatment, treated in past six months)	1.0

Score range	Mean probability of PE	% with this score	Interpretation of risk
0-2 points	3.6%	40%	Low
3-6 points	20.5%	53%	Moderate
> 6 points	66.7%	7%	High

Figure 6. Wells criteria for PE

Diagnosis of acute PE:

- Gold Standard: CT angiography (CTA): sensitivity: 90-100%, specificity: 89-94% and 99% negative predictive value. With CTA RV/LV ratio can be measured to estimate RV dysfunction.

- Echocardiography: in absence of CTA it is recommended to use for exclusion of alternative diagnoses (aortic dissection, pericardial tamponade, AMI related heart failure), and check PE's secondary signs (RV dilatation, RV wall hypokinetic, left septal shift, decreased inferior cava vein respiratory variation, tricuspid valve regurgitation and > 30 mmHg pressure gradient above tricuspid valve). With echocardiography chronic and acute PE can be distinguished.
- D-dimer is a fibrin degradation product. With qualitative D-dimer ELISA test presence of fibrin can be detected. The elevated level of D-dimer proves thrombotic diseases (thromboembolism, trauma, malignant diseases and bleeding, necrotic and infectious conditions). D-dimer has a reassuring negative predictive value in suspected submassive or non-massive PE.
- ECG: S1Q3T3, non-specific ST segment changes, precordial T-wave inversion
- Chest X-Ray has no specific diagnostic value.

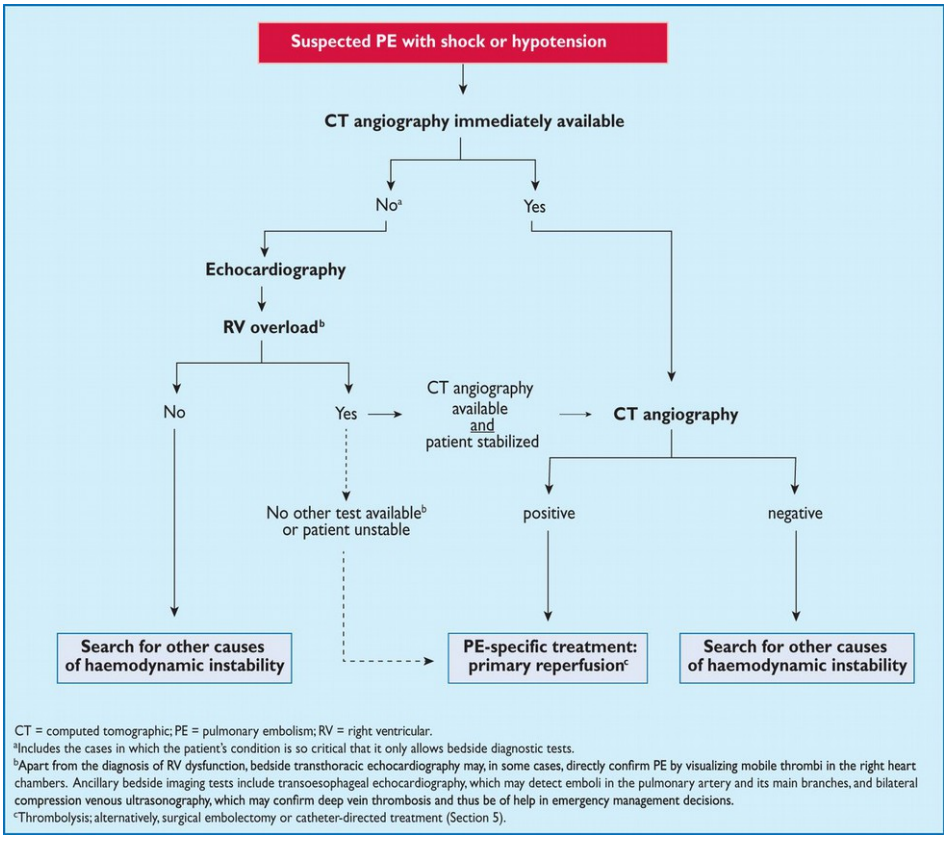


Figure 7. Diagnostic algorithm for suspected PE with shock or hypotension (from 2014 ESC Guidelines on the diagnosis andmanagement of acute pulmonary embolismEuropean Heart Journal (2014) 35, 3033–3080)

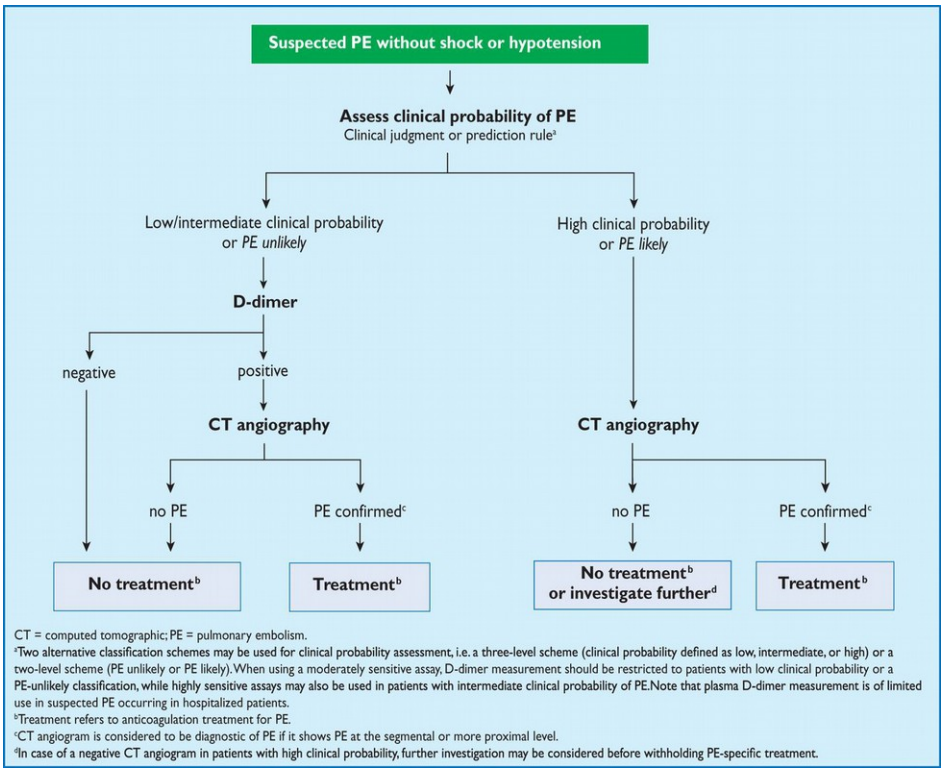


Figure 8. Diagnostic algorithm for suspected PE without shock or hypotension (from 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism European Heart Journal (2014) 35, 3033–3080)

NB: If the patient has signs of shock or hypotension with the suspicion of PE indicate CTA as soon as it is possible and if it is proved start treatment immediately!

Treatment of Massive PE:

- Thrombolysis with Alteplase 10 mg bolus, the 90mg/2 hrs with supplementary therapy. For contraindications of thrombolysis, see above!
- Catheter Embolectomy or Fragmentation of the embolus

The treatment of submassive or non-massive PE is not the role of ED.

3. *Boerhaave Syndrome:*

Oesophageal wall rupture as a complication of excessive vomiting, chest trauma, CPR or drinking corrosive agents. Nowadays Boerhaave syndrome has an iatrogenic origin (endoscopy). Bile, gastric acid and saliva rich in bacteria enter the mediastinum causing sever and fulminant infection and finally life-threatening septic shock.

Signs and symptoms:

- Vomiting is followed by sudden chest pain and breathlessness. (Emesis induced Boerhaave sy. is typical, but Boerhaave sy. has other aetiologies too)

- Subcutaneous emphysema
- Pleural fluid is suspected by percussion
- Chest X-ray: Mediastinal free air, widening of mediastinum
- CT: fluid or air in the mediastinum

4. *Pneumothorax*

Pneumothorax (PTX) is an emergency condition when air is present between the two layers of pleura (mostly caused by rupture of a subpleural bulla) and causes an uncoupling of the lung from the chest wall. PTX may have some predisposing factors male sex, asthenic body habitus,Marfansy or smoking.

Signs and symptoms:

- Pleural chest pain with breathlessness.
- Absence of breathing sounds above the collapsed lung
- Hyperresonance to percussion of chest wall
- Vocal Resonance
- Chest X-ray: air in the pleural cavity

Treatment:

- Small apical PTX without respiratory failure require no further treatment
- If the PTX cause respiratory symptoms, or it has great extension:
    - Air aspiration
    - Chest tube
    - Intercostal catheterisation
    - Heimlich valve insertion

Tension PTX: air is trapped in the pleural cavity under expirations, consequently intrathoracic pressure is elevated higher than atmospheric pressure, compresses the lung, and may displace the mediastinum and its structures toward the opposite side, with consequent cardiopulmonary impairment. If tension pneumothorax is suspected no further imaging (on site Chest X-ray is a possibility) is required but urgent needle decompression has to be done.

5. *Acute Perimyocarditis*

The inflammation of pericardium and myocardium. Acute perimyocarditis is not an uncommon entity in ED (0.1% of hospital admissions).

- Aetiology: viral, bacterial or fungal infections
- Predisposing factors: connective tissue disorders, renal impairment, malignant diseases previous aortic dissection.
    - Dressler sy is a special form of perimyocarditis 2-3 weeks after acute myocardial infarction.

Signs and symptoms:

- Chest pain with the characteristic of sharp and stabbing. Intensification of pain with deep breathing is typical. Pain decreases in intensity in sitting or forward bend position. Pain is not related to physical activity, and not ease with nitrates.
- Muscle ache, fatigue, malaise
- Fever: sudden high fever and chills are characteristics of bacterial infection.
- ECG: ST segment elevation and PR segment depression (except in aVR where ST segment does not change and PR segment is elevated) Later ST and PR segment changes recur and diffuse T wave inversion occurs.

6. Pericardial Tamponade

Pericardial effusion compresses the heart causing circulatory collapse (and in this situation immediate CPR has to be attempted). In most of the cases pericardial effusion is present without the symptoms of tamponade. 1-2 cm pericardial fluid has effect on circulation, but more than 2cm of pericardial fluid is a serious risk factor of pericardial tamponade. Certain warning signs can be observed before pericardial tamponade occurs. The pericardial pressure vary with the cardiac cycle, maximal pericardial pressure in tamponade occurs during end-diastole.

Signs and Symptoms:

- Transthoracic Echocardiography:
    - End-diastolic collapse of right atrium, then early diastolic RV collapse,

    - Dilated inferior cava vein with absence of inferior cava vein respiratory variation
    - Swinging heart
    - Circulatory collapse: the pleural pressure is equal to the intracardial diastolic pressure
- Dilated Jugular veins, blurred heart sounds
- Tissue hypoperfusion, but hypotension is not necessarily present
- ECG: low voltage, Electrical Alternans related to swinging heart phenomenon. (see Figure 9.)
- Paradox Pulse: peripheral pulse vanishes with inspiration end returns in expiration.

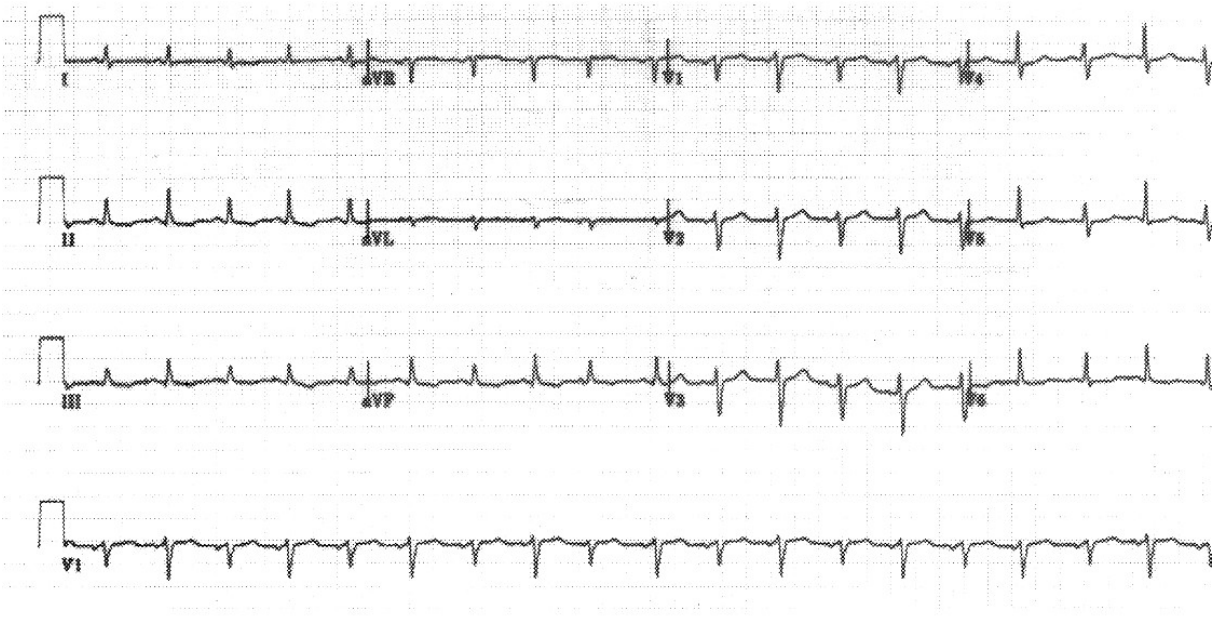


Figure 9. ECG low voltage and electric alternans (from KÖR-ITO archives)

Treatment of pericardial effusion: Pericardial Drainage (for scoring see Fig. 10)

Pericardial drainage may be a lifesaving intervention, but sometimes it is used as a diagnostic method (microbiological or cytological sampling).

Threatening Pericardial Tamponade ECHO findings:

- Right atrial collapse > 1/3 cardiac cycle
- Right ventricular collapse

- Respiratory flow variation across the mitral valve> 25%

Trauma or aortic dissection related pericardial tamponade or purulent pericardial fluid requires surgical management.

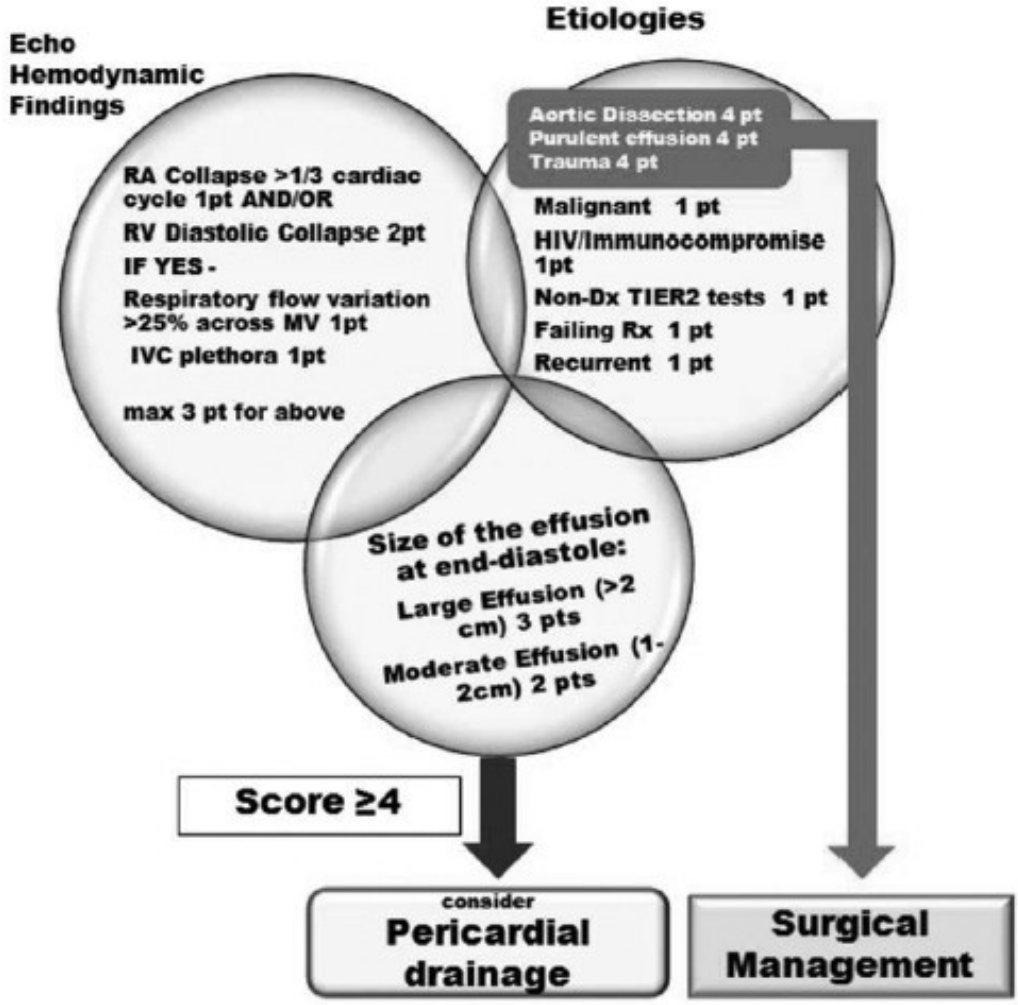


Figure 10. Pericardial effusion scoring index (from A Novel Pericardial Effusion Scoring Index to Guide Decision for Drainage CRITICAL PATHWAYS IN CARDIOLOGY · JUNE 2012)

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7. Management of Sepsis in the ED by Prof.Dr.MolnárZsolt

Although sepsis is receiving closer and closer attention – as it is the greatest challenge in intensive therapy (high mortality and expensive therapy), the concept behind sepsis is still undetermined. The fact is, sepsis has no definition, albeit in the clinical practice sepsis is mostly mentioned as a diagnosis. To this date no laboratory value or diagnostic procedure has been proven as definitive marker of sepsis. First time Bone at al. tried to create criteria for sepsis planning a multicentre investigation about the adjuvant therapy of metronidazole in septic shock. According to Bone's sepsis syndrome concept, sepsis is the systemic response to infection. The concept of sepsis syndrome was only a theory proposed in a Los Angeles hotel by a group of scientist in 1980 when they tried to work out a new protocol. As this definition was highly queried the ACCP/SCCM (American College of Chest Physicians/Society of Critical Care Medicine) Consensus Conference Committee laid down the definition of sepsis, infection, bacteraemia, severe sepsis, septic shock and a new concept, SIRS (systemic inflammatory response syndrome).

Although sepsis definition still need to be developed the current definitions are the followings:

- Infection: inflammatory response to presence of microorganisms
- Bacteraemia: presence of living bacteria in the bloodstream
- SIRS (systemic inflammatory response syndrome): inflammatory response to different ictus of the body. SIRS is proven if two from the followings occur:
    - Temperature < 36°C or >38°C
    - Heart Rate > 90/min
    - Respiratory Rate > 20/min or P_aCO_2< 30 mmHg
    - White Blood Count < 4,000cell/mm^3, or >12,000 cell/mm^3, or > 10% of immature form
- Sepsis: systemic response to infection with SIRS
- Severe Sepsis: sepsis + organ dysfunction, hypoperfusion or hypotension
- Septic Shock: sepsis-induced hypotension despite appropriate fluid resuscitation

    - BP_{sys}< 90 mmHg or more than 40 mmHg decrease respect the normal blood pressure values of the patient
    - Inotropes are required to maintain blood pressure
- MSOF (Multiple System Organ Failure): acutely occurred organ failure in critically ill patient requires organ support therapy

The pathophysiology of the inflammatory cascade:

The pathophysiology of sepsis and SIRS is not entirely known. Presumably d is independent from the type of the triggering factor. A random ictus (trauma, injury, infection, surgery) triggers proinflammatory cytokine production [tumour necrosis factor (TNF), interleukins (IL), platelet activating factor (PAF)] in monocytes. Proinflammatory cytokines activate leukocytes, trigger the production of neutrophil-endothelium adhesion molecules facilitating the neutrophil migration to the tissues. Inflammatory molecules (complements, prostaglandins and proteases) are released from leukocytes facilitating more cytokine release.

This cascade has two main mechanism: Damage Associated Molecular Pattern (DAMP) and Pathogen Associated Molecular Pattern (PAMP). The same cascade is triggered by both mechanisms for the similarity of genetic material in mitochondria and bacteria. This is the reason why the same pattern of ARDS is provoked in trauma, pancreatitis or an extra-pulmonary organ's severe infection. The severity of the condition depends on the given response (SIRS, sepsis, severe sepsis or without treatment MSOF) not on the culprit factor.

Diagnostics in sepsis:

Sepsis is a condition not a well-defined disease, therefore it might occur in various forms. Evaluation and early recognition of sepsis requires considerable experience.

There are many non-specific, and not really sensitive diagnostic signs or laboratory value like fever, CRP or WBC. These signs separately are not reliable, but important as alarming signs. Monitoring TNF, or IL-6, IL-1 and IL-8 is quite expensive, and used in research not in clinical practice. To this date the most sensitive marker of bacterial sepsis is serum procalcitonin level. Many trials examined the connection between the level of PCT and SIRS or sepsis. The elevated level or the increasing dynamics of PCT level is the most sensitive marker of bacterial sepsis.

NB: elevated PCT level is not enough for the diagnosis of bacterial sepsis, take the whole clinical picture into consideration!

Recognition of organ failure:

- Altered mental state or drowsiness: use Glasgow Coma Scale. Altered mental state is a significant – and in many cases the only – sign in suspected sepsis.
- Circulatory failure: tachycardia, elevated lactate level and increasing inotrope demand are reliable signs of circulatory failure.
- Respiratory failure: elevated respiratory rate, dyspnoea, arterial blood gas measurement:decreased Horovitz-quotient (Carrico index) PaO_2/FiO_2 the best marker of intrapulmonary shunt.
- Renal failure: measurement of urine output, BUN, seK^+, actual HCO_3^- and the most sensitive se. creatinine level. (Arterial blood gas measurement is one of the most important diagnostic procedures).
- Hepatic failure: GOT, GPT, GGT, ALP these enzymes might be elevated in many cases, therefore have low sensitivity and specificity. But albumin, coagulation factors and bilirubin are produced daily, therefore these markers are more sensitives and specifics in hepatic failure (bilirubin is the most sensitive and the most specific).
- Bone marrow failure: decreased platelet count.

Treatment of sepsis:

A non-specific but complex therapy. The main points of therapeutic intervention in sepsis:

- Resuscitation: restore normal DO2/VO2 ratio.
    - Oxygen therapy, mechanical ventilation.
    - Eliminate pain: give analgesics
    - IV fluid replacement therapy +/- vasopressor or inotrope therapy
- Antibacterial therapy: Broad spectrum antibiotics, and if it is necessary surgical intervention.
- Goal Directed Therapy: a controversial form of sepsis management. It has 3 different types and kindled the interest of the intensive care and emergency care 30 years ago.
- Specific therapy does not exist, albeit countless trial and research aimed specific TNF blocker or IL blocker or NO blocker development. All these scientific failures confirm the fact that sepsis is not a disease, but a condition.

Recommended Articles:

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8. Management of Neuroemergency in the ED by Dr.BorosIstván

One of the most common problems for which patients present to the ED are neurological disorders (10-20%). For the possible life-threatening or disabling outcome of neurological disorder prompt and well protocoled management is essential.

The most common neurological disorders in ED

- Stroke
- Epilepsy
- Neuromuscular disorders
- Vertigo
- Acute Pain

Stroke management in the ED:

Stroke is the most common neuroemergency, the third leading cause of death and the first leading disabling disorder in developed countries. Definitive (thrombolytic) therapy of patients with acute ischaemic stroke is possible within a narrow time window of 4.5 hrs after the first symptom presents. Unfortunately most of the patients arrive too late to the ED. Clinical practice guidelines and well protocoled management are required to avoid in-hospital delay, and by this improve the outcome of acute ischaemic stroke.

WHO definition of Stroke:

A clinical syndrome consisting of rapidly developing clinical signs of focal (or global in case of coma) disturbance of cerebral function lasting more than 24 hours or leading to death with no apparent cause other than a vascular origin.

Two major cause of stroke:

- Ischaemic Stroke: hypoxic cerebral injury due to decreased or stopped blood flow. The injured area has two parts: infarct core and penumbra. Penumbral area ha collateral blood supply, therefore in the penumbral area blood supply is decreased, but not fully stopped. Restoration of blood flow of the occluded artery within the above mentioned time window may prevent the penumbral area's permanent injury.
- Haemorrhagic Stroke:cerebral injury due to rupture of a vessel

Aetiology: 80 % of all stroke cases are related to ischaemia, and 20% to haemorrhage. As all cardiovascular diseases, stroke has the same risk factors:

- Vascular wall injury: atherosclerosis, hyalinosis, arteritis, dissection
- Blood related risk factors: altered cagulability, changes of blood quantity, changed blood components
- Circulatory status: arrhythmias, AMI, etc.

10-30% of ischaemic stroke have a thromboembolic aetiology. Atrial fibrillation is the most frequent underlying cause of thromboembolic stroke.
Haemorrhagic stroke has other aetiological factors, hypertension or anticoagulant are the most common ones. Intracerebral bleeding is caused by penetrating vessel injury. Rupture of an intracranial aneurism causes subarachnoid haemorrhage. One third of the stroke cases has unknown origin.

Signs and Symptoms of Stroke:

Stroke symptoms are varied according to the site of the lesion. The symptoms' characteristics advert the location of the lesion, hence physical examination is often enough to tell where the lesion is. The alarming signs of Stroke:

- Sudden one sided numbness, tingling, weakness, or loss of movement of face, arm, or leg.
- Sudden speech disorder
- Sudden confusion, altered mental state, drowsiness or coma (typical in haemorrhagic strokes)
- Sudden vision changes.
- Sudden confusion or trouble understanding simple statements
- Sudden problems with walking or balance with vegetative changes
- A sudden, severe headache that is different from past headaches

NB: Differentiate between stroke and stroke mimic states: hypoglycaemia, post epileptic paresis (Todd's paresis), migraine, Sclerosis multiplex

Prehospital Stroke Care:

The first step of immediate and effective stroke treatment is the high level of stroke awareness among the population. Rapid recognition of alarming sigs of stroke and calling ambulance immediately may save lives.

Stroke is responsible for 5-6% of all ambulance cases in Hungary. The prehospital treatment has to be done in 30 minutes, and the patient has to be transported to ED in 60 minutes.

For the field assessment of stroke Cincinnati Prehospital Stroke Scale is most widely used.

- Facial droop: Have the patient smile or show his or her teeth. If one side doesn't move as well as the other so it seems to droop, that could be sign of a stroke.
    - Normal: Both sides of face move equally
    - Abnormal: One side of face does not move as well as the other (or at all)
- Arm drift: Have the patient close his or her eyes and hold his or her arms straight out in front for about 10 seconds. If one arm does not move, or one arm winds up drifting down more than the other, that could be a sign of a stroke.
    - Normal: Both arms move equally or not at all
    - Abnormal: One arm does not move, or one arm drifts down compared with the other side
- Speech: Have the patient say, "You can't teach an old dog new tricks," or some other simple, familiar saying. If the person slurs the words, gets some words wrong, or is unable to speak, that could be sign of stroke.
    - Normal: Patient uses correct words with no slurring
    - Abnormal: Slurred or inappropriate words or mute

Prehsopital Stoke Assesment:

- ABCDE aprroach and treatment if it is necesarry.
- Define the accurate time of stroke symptoms’ onset.
- Evaluaion of contraindications.

Prehospital Stroke Treatment:

- Maintain vital functions according to ABCDE
- Give supplemental oxygen if the SpO2 < 95%

- Position the patient: the head of the bed elevated at 30 degrees
- Insert a large bore IV cannula
- Measure Blood Glucose (give glucose if BG < 2.7 mmol/L)
- Measure NIBP (maintain the MAP > 100 mmHg, give antihypertensive drug if NIBP > 220 mmHg)
- ECG to record the possible arrhythmias
- Rapid transport of stroke patient.

Emergency Department Stroke Management: The Stroke Patient Pathway

The goal for the acute management of stroke patients is to stabilize the patient and to complete initial evaluation and assessment. In the ED a stroke team (emergency physician, neurologist, radiologist, laboratory expert, clinical nurse specialist, nurse, orderly and administrator) is responsible to organise all the diagnostic and imaging procedures,which is essential for the definitive treatment within 4.5 hrs time window.

Investigations:

- ABCDE approach and treatment if it is necessary + rapid neurological assessment
- Monitor vital functions
- Record an ECG
- Check BG, electrolytes, quantitative blood count, prothrombin time, activated prothrombin time, INR, hepatic enzymes, BUN and creatinine.
- Arrange an emergency non-contrast CT scan of the brain

The imaging gold standard of acute stroke patient is CT scan. It provides immediate information about the type of stroke (ischaemic – no bleeding on CT scans, or haemorrhagic – bleeding on CT scans).

Treatment: in ischaemic stroke evaluate indications and contraindications of Thrombolysis

- Assess airway and breathing, if SpO2 < 95% give supplemental oxygen. Avoid hypoxia!
- Blood pressure:

    - Lower BP under 185/110 mmHg gradually if thrombolysis is planned
    - If BP > 220/120 mmHg lower gradually BP, target: lower the MAP 15-25% in the 1st 24hrs.
    - If BP < 100/70 mmHg, give crystalloid or IV vasopressors if crystalloid is insufficient.
- Control the body temperature, give antifebrile medication if T° > 37.5 °C
- Control BG, give IV insulin if BG > 10mmol/L, give glucose if BG < 4mmol/L
- Recurrent seizures: give carbamazepine or valproate or lamotrigine
- Give anticoagulants in ischaemic stroke if thrombolysis is not possible

Thrombolysis: thrombolytic agent – recombinant tissue plasminogen activator (rtPA) – breaks up the occlusive thrombus, and restore the cerebral blood flow.Thrombolysis is indicated if the onset of the symptoms is earlier than 4.5 hrs (in patients above 80 years with Diabetes the time window is narrower: 3 hrs.) and the following criterions are fulfilled:

- Diagnosis of ischemic stroke causing measurable neurologic deficit
- Neurologic signs not clearing spontaneously
- Neurologic signs not minor and isolated
- Symptoms not suggestive of subarachnoid haemorrhage
- No head trauma or prior stroke in past 3 months
- No MI in prior 3 months
- No GI/GU haemorrhage in previous 21 days
- No arterial puncture in noncompressible site during prior 7 days
- No major surgery in prior 14 days
- No history of prior intracranial bleed
- Systolic blood pressure under 185 mm Hg, diastolic blood pressure under 110 mm Hg
- No evidence of acute trauma or bleeding
- Not taking an oral anticoagulant, or if so, INR under 1.7
- If taking heparin within 48 hours, a normal activated prothrombin time (aPT)

- Platelet count of more than 100,000/μL
- Blood glucose greater than 50 mg/dL (2.7 mmol)
- No seizure with residual postictal impairments
- CT scan does not show evidence of multilobar infarction (hypodensity over one-third hemisphere)
- The patient and family understand the potential risks and benefits of therapy

If thrombolysis is indicated administer0.9 mg/kg rtPA IV (10% of the total dose in an initial IV bolus and the remainder in infusion over 60 minutes).

Intracerebral haemorrhage (ICH) is the main adverse effect of rtPA treatment. Arrange other non-contrasted CT scan of the brain 24 hrs after the thrombolysis to exclude ICH, and check for signs of other possible bleeding.

Treatment of haemorrhagic stroke:

Severity of bleeding associated brain tissue damage is depend on the extension and the localization of the bleeding. Minor and superficial haemorrhage requires only conservative treatment. Extensive haemorrhage or cerebellar haemorrhage indicates acute surgical intervention.

As a summary:

- Stroke patients need immediate treatment without any delay. Time is brain!
- Stroke has two major types: ischaemic and haemorrhagic stroke.
- Non-contrasted brain CT scan is the gold standard used to discriminate the two major stroke types.
- The time window of thrombolysis in ischaemic stroke is 4.5 hrs.
- Use ABCDE approach.
- Always monitor the patient's vital signs, and maintain the vital functions.

Management of Seizures in the ED

Besides maintaining the patient's vital functions, history taking both from the patient and the witnesses is essential for the accurate diagnosis and for the elimination of the triggering factors.

Definition: Seizure is a fulminant and transient brain dysfunction accompanied by motor, sensory and autonomic symptoms and loss of consciousness in most of the cases. Seizure itself is a symptom of abnormally synchronised neuronal function.

Epilepsy is characterised by spontaneously recurrent seizures. Seizures may be provoked by different triggering factors (fever, trauma, metabolic or electrolyte disorders).

Classification: primer (unknown origin) - seconder (known aetiology), partial (seizure localised to one limb)-generalised (seizure involve the whole body). Seizures also can be classified by the frequency of them: occasional seizures, cumulative seizures, and status epilepticus.

- Simple partial seizures (focal seizures): symptoms may be either motor, sensory, autonomic, or any combination of the three.
- Complex partial seizures (often with a preceding aura). This type of seizures is accompanied by different cognitive, affective, and psychomotor symptoms and loss or alteration of consciousness. Postictal drowsiness is usual.
- Grand mal epilepsy primarily generalised seizure, which is characterised by four phases: aura, tonic phase (rigid muscles), clonic phase (muscles alternately contract and relax), postictal state (patient regains consciousness slowly).
- Petit mal (absence) epilepsy lasts only a few seconds and has sudden onset with no aura or warning and no postictal symptoms. Seizures of this type usually affect children.

Occasional seizures occur only once and have a well-defined trigger factor. Elimination of the trigger factor is usually sufficient, no medication is needed. Fever or metabolic disorders might be trigger factors, but the in most of the cases alcohol withdrawal is the underlying cause of occasional seizure.

Cumulative seizures occur repetitively in a brief time. Between the seizures the patient's mental state is clear. This form of seizures may lead to status epilepticus, therefore the patient require immediate sedation.

Status epilepticus the convulsion least more than 5 minutes or drowsiness/confusion remains between cumulative seizures. This form of seizure require immediate treatment and if the patient does not respond to the therapy after 30minutes call for ICU.

Anticonvulsant therapy:

The goal of treatment in patients with seizures is to maintain vital functions according to ABCDE approach (maintain airways, give oxygen and maintain circulation) andachieve a seizure-free status with regained consciousness, prevent cerebral oedema and find the trigger factor to prevent further seizures.

A. Give diazepam 0.2-0.5 mg/kg IV, or lorazepam 0.05-0.1 mg/kg IV
B. Give Phenytoin 18 mg/kg with infusion pump 50 mg/min
C. Give Phenobarbital 8-20 mg/kg with infusion pump 60mg/min

Management of neuromuscular disorders in the ED

If the patient has muscle weakness as chief compliant neuromuscular disorders might be as underlying cause. These chronic disorders needs emergency care rarely, but sometimes neuromuscular disorders may occur acutely or affect breathing causing respiratory failure.

Guillain-Barré syndrome is an autoimmune neurological disorder affecting the myelin sheath of peripheral nerves usually following a virus or bacterial infection. After the infection Guillain-Barrésy.has a slow onset with distal muscle weakness, partial paralysis or pain. The symptoms progress proximally reaching cranial nerves and finally causing respiratory failure too. In the cerebrospinal fluid the protein level is elevated with normal or slightly elevated cell count.

Treatment of GuillanBarrésy

- Use ABCDE approach, maintain vital functions
- Tracheal intubation and mechanical ventilation
- Give LMWH to prevent DVT
- Plasmapheresis, IV immunoglobulin or corticosteroid administration should be considered.
- Monitor the patient! (If vagal nerve is affected arrhythmias or suddenly decreasing blood pressure may occur)

Myasthenia Gravis is an autoimmune disorder producing anti-acetylcholinesterase receptor antibodies. Main symptom in Myasthenia Gravis is painless weakness of muscles, which becomes progressively worse during physical activity. Proximal muscle weakness, ptosis, dysarthria, double vison and dyspnoea are characteristic symptoms. In Myasthenia Gravis only the motor function is affected, there is no sensory dysfunction, and the reflexes are also intact.

Treatment of Myasthenia Gravis

- Use ABCDE approach, maintain vital functions
- Tracheal intubation and mechanical ventilation
- Give pyridostigmine bromide (Mestinon) 30 mg initial dose, then repeat in every 4-6 hrs.
- Give corticosteroids 80-120mg once per day
- Plasmapheresis or IV immunoglobulin administration should be considered

Vertigo

Vertigo is a common symptom in the ED. Vertigo is a sensation of rotation, instability or movement of one's self. Vertigo may have peripheral or central nervous causes. The goal of the emergency treatment is to identify the underlying cause and give definitive therapy.

Discrimination of peripheral and central causes is essential.

- Peripheral vertigo: typically present with mild to moderate imbalance, nausea, vomiting, hearing loss, tinnitus, fullness, and pain in the ear, usually accompanied by severe autonomic symptoms.
- Central vertigo has accompanying neurologic deficits and pathologic nystagmus, vertigo is often so severe that many patients are unable to stand or walk.

Supportive therapy:

- Give antiemetic, diazepam, promethazine or betahistine.

Neurologic disorders with acute pain

We mention only the two most common condition: lumbar herniated disc and headache. The main goal of therapy is to ease the pain, prevent further complications, identify and treat the underlying cause.

Lumbar herniated disc:

Lumbar herniation is the most common type of disc herniation. The protruding disc causes nerve root compression with radiating nerve pain felt in the lower extremities or groin areaandprotruding disc can also cause bowel and bladder incontinence. Straight leg raise (also called Lasègue's test) has to be done, if the patient experiences sciatic pain when the straight leg is at an angle below 70 degrees the test is positive.

Treatment: bed rest, NSAIDs, muscle relaxants are often sufficiently ease the pain, but in some cases surgical intervention is indicated (in case of paresis, vegetative disorders or persistent pain despite administration of analgesic drugs).

Headache:

Headache is the most common neurological symptom, it is responsible for 1-2% of acute care. Although headache can result from benign causes, in some cases severe life-threatening condition might be the underlying pathology (encephalitis, meningitis, subarachnoid haemorrhage, subdural haemorrhage, elevated intracranial pressure, head injury). The main goal of emergency care is to discern these life-threatening conditions and treat them immediately.

Chronic pain often occurs in primer headache syndromes like migraine, tension-type headache or cluster headache, but changing in the characteristics of chronic pain may be an alarming sign. Other alarming signs are the followings: pain is unresponsive to treatment, patient suffers from worst ever headache or if the headaches is associated with worrying features like altered mental state, autonomic symptoms, fever, focal neurology.

NB: Consider subarachnoid haemorrhage (SAH) in any "worst ever" or sudden onset headache (usually described as a blow to the back head)!

Arrange emergency CT scan in every suspected case of SAH, if CT scan is normal carry out lumbar puncture!

If there is no sign of life-threatening pathology use a detailed history, use OPQRST formula to characterise headache. Look for accompanying or prodromal signs like blurred vision, photophobia, malaise, anorexia or vomiting.

Treatment of not life-threatening headache:

Minor analgesics possibly combined with adjuvant therapy (diazepam, droperidol, prednisolone, sumatriptan and high flow oxygen)

9. Management of Abdominal Pain in the ED by. Dr. Schneider Erzsébet

Abdominal pain is one of the most common presenting complaintsin the ED. Many disease provoke abdominal pain, therefore physical examination, patient history and accompanying symptoms must be taken into consideration to identify the underlying disease.
The characteristics of pain can refer to the disease, which has provoked it.

- sudden aching pain (e.g. perforation, nephrolithiasis or ectopic pregnancy)
- blunt, gradually intensifying pain (e.g. gastritis, pancreatitis or diverticulitis)

The localisation of pain:

- Typical localisation and symptoms:
    - appendicitis: McBurney's point, Blumberg sign, Rowsing sign, Psoas sign
    - nephrolithiasis: flank pain
    - diverticulitis: left iliac pain
    - biliary tract: right hypochondriac pain
    - stomach, duodenum: epigastric pain
- Extra-abdominal radiation of abdominal pain:
    - splenic rupture pain radiates to the left shoulder
    - pancreatitis: pain radiates to the left side of the chest
- Extra-abdominal pain radiates to the abdomen:
    - pneumonia, pulmonary embolism, myocardial infarction…

NB: patients with altered mental state or elderly patients may experience false symptoms, and symptoms may be modified by previously taken drugs!
Patient History - Take patient history as accurately as it is possible!

- Characteristics of pain (Onset, Provocation, Quality, Radiation, Severity, Timing)
- Accompanying symptoms(fever, nausea, vomiting and vegetative signs)
- Indiscretion of diet

- Characteristics of faces: colour (black, bloody, fair), alteration of defecation or bowel movement, diarrhoea or constipation
- Characteristics of urine: urge of urination, burning urine, altered colour, odour…
- Menstrual cycle
- Hernia
- Injury
- Previous diseases
- Regularly taken medicine

Physical examination of the abdominal pain:

- palpation (symmetric palpation all 4 abdominal quadrant)
- auscultation of bowel movement (absence, or high pitched tinkling sounds) abdominal splash!
- rectal digital examination
- abdominal percussion (flatulence, hepatomegaly, splenomegaly, free abdominal air)
- nasogastric tube

Other investigations: NIBP, ECG, Temperature, BGM, arterial blood gas analysis, bedside urinalysis, bedside qualitative beta hCG test.

Laboratory:

- Hepatic enzymes, renal function, urinalysis, drug tests, blood count, lactate, CRP, procalcitonin, calcium and cardiac enzymes

Radiologic investigations:

- X-ray, US, CT (if US and X-ray is insufficient to diagnose the condition!), endoscopy, special and rarely used radiological investigations:MR, angiography, isotopic imaging,

Acute Abdomen:

Progressive life-threatening condition.

Symptoms:

- Sever, intensifying abdominal pain
- Fulminant worsening condition

- Abdominal guarding (défense musculaire)
- Absence of bowel movement
- Accompanying vegetative symptoms: fever, nausea, vomiting, sweating, tachycardia
- Symptoms of massive bleeding if abdominal bleeding is the underlying condition.

Aetiology:

- Gastrointestinal perforation
- Intra-abdominal inflammation: appendicitis, cholecystitis, cholangitis, pancreatitis, abscess
- Intra-abdominal vessel occlusion: mesenteric thrombosis
- Intra-abdominal, retroperitoneal or gastro-intestinal bleeding
- Injury: hepatic rupture, splenic rupture
- Ileus
- Renal or biliary colic
- Ectopic Pregnancy
- Peritonitis

Investigations:

- abdominal X-ray, abdominal US, CT scan,
- arterial blood gas analysis
- laboratory: blood count, hepatic and renal function, pancreatic enzymes, calcium, INR, CRP, procalcitonin, lactate, urinalysis
- microbiologic sampling: haemoculture, urine bacteriology
- Bedside blood grouping
- Nasogastric tube
- Seek expert's help (surgeon, traumatologist, urologist, gynaecologist or gastroenterologist)

Therapy:

- maintain blood pressure, and vital functions
- management of pain
- transfusion
- adequate specific therapy according to aetiology

Abdominal emergency conditions according to their localisation

Right hypochondriac region:

- cholecystitis, biliary obstruction, biliary pancreatitis, right sided nephrolithiasis or pyelonephritis, appendicitis, costal fracture, hepatic congestion, pleuritis, PTX, pneumonia, pulmonary embolism, intestinal obstruction, tumour of colon, kidneys, gallbladder or liver
- Investigation: abdominal and chest X-ray, abdominal US, CT as occasion requires, ERCP
- Laboratory: blood count, hepatic and renal functions, inflammation parameters, INR, pancreatic enzymes, urinalysis D-dimer

Epigastric region:

- gastritis, gastric or duodenal ulceration, pancreatitis, intestinal obstruction, cardia spasm, AMI, colon tumour
- Investigations: abdominal and chest X-ray, abdominal US, CT as occasion requires
- Laboratory: blood count, hepatic and renal functions, inflammation parameters, INR, pancreatic enzymes, cardiac enzymes, D-dimer

Left hypochondriac region:

- left sided nephrolithiasis or pyelonephritis, splenic rupture, pancreatitis, intestinal obstruction, AMI, pulmonary embolism, PTX, pleuritis, pneumonia, tumour of: colon, pancreas or kidney.
- Investigations: abdominal and chest X-ray, abdominal US, CT as occasion requires
- Laboratory: blood count, hepatic and renal functions, inflammation parameters, INR, pancreatic enzymes, cardiac enzymes, D-dimer, urinalysis

Right iliac region:

- nephrolithiasis, ureter stone, urinary infection, appendicitis, adnexitis, ectopic pregnancy, ovarian torsion, intestinal obstruction, Crohn disease, hernia, orchitis, tumour of kidneys, colon, ovary or uterus
- Investigations: abdominal X-ray, abdominal US, CT as occasion requires
- Laboratory: blood count, hepatic and renal functions, inflammation parameters, INR, bedside beta hCG, urinalysis

Left iliac region:

- nephrolithiasis, ureter stone, urinary infection, appendicitis, adnexitis, ectopic pregnancy, ovarian torsion, intestinal obstruction, diverticulitis, hernia, orchitis, tumour of kidneys, colon, ovary or uterus
- Investigations: abdominal X-ray, abdominal US, CT as occasion requires
- Laboratory: blood count, hepatic and renal functions, inflammation parameters, INR, bedside beta hCG, urinalysis

Suprapubic or Hypogastric region:

- urinary infection, prostatitis, metritis, ovaritis, intestinal obstruction, tumour of uterus, ovary or colon
- Investigations: abdominal X-ray, abdominal US, CT as occasion requires
- Laboratory: blood count, hepatic and renal functions, inflammation parameters, INR, bedside beta hCG, urinalysis

Special conditions with abdominal pain:

Nephrolithiasis:

- Patient's history: sudden onset aching pain of the flank, which radiates to the iliac region, to testes or vulvar region. Accompanying symptoms: nausea, vomiting, other vegetative symptoms and haematuria.
- Physical findings: sensitive flank, tenderness along the ureter, absence of bowel sounds
- Investigation: renal X-ray, abdominal US, laboratory: renal and hepatic functions, amylase, lipase, blood count, urinalysis, CRP
- Therapy: analgesia, infusion, seek urologist help if it is necessary

Splenic rupture:

- Traumatic splenic rupture (blunt abdominal injury): may be immediate or two-stage splenic rupture
    - *NB: in trauma patients always search for signs of splenic ruptures, even if the trauma happened days before!*
- Spontaneous splenic rupture: splenic vein thrombosis, malaria, mononucleosis, haematological diseases or complication of pancreatitis.
- Patient's history: left hypochondriac, blunt, intense and continuous pain, which radiates to the left shoulder
- Physical findings: left hypochondriac tenderness, palpable splenomegaly, absence of bowel sounds, diffuse défense musculaire and in severe cases haemorrhagic shock.
- Investigation: abdominal X-ray and US, abdominal and pelvic CT scan, blood gas analysis, blood grouping, laboratory: electrolytes, glucose, blood count, renal and hepatic function, amylase, lipase, procalcitonin, CRP, calcium and INR
- Therapy: maintain vital function, analgesia and transfusion if it is needed, seek surgeon or traumatologist help for urgent surgical intervention!

Biliary colic:

Common and complex condition occurs in every age group. Biliary stones makes stop or inflammation. Acalculosus biliary painis uncommon.

- Patient's history: indiscretion of diet and consequent gradually intensifying right hypochondriac, cramping pain with nausea vomiting and fever.
- Physical findings: right hypochondriac tenderness radiates to epigastric region, palpable gall bladder, silent bowel movement and rarely acute abdomen
- Investigation: abdominal X-ray and US, abdominal and pelvic CT scan, blood grouping, laboratory: electrolytes, glucose, blood count, renal and hepatic function, amylase, lipase, procalcitonin, CRP, calcium and INR
- Therapy:
    - biliary colic – stop enteral feeding, start parenteral fluid replacement and analgesia

- cholecystitis – surgical intervention, time window: 72hrs, parenteral fluid replacement, analgesia, microbiological sampling then start antibiotic therapy
- cholangitis – ERCP (if cholangiolithiasis is identified) , parenteral fluid replacement, analgesia, microbiological sampling then start antibiotic therapy

Pancreatitis:

- Aetiology: 70% biliary stone, 20-25% alcohol induced and other diseases: lipaemic crisis, autoimmune pancreatitis, viral infection.
- Pancreatitis has a wide spectrum of severity from fast recovery without complications to rapidly worsening critical condition with ARDS, acute renal failure and circulatory collapse.
- Patient's history: indiscretion of diet or alcohol consumption followed by gradually intensifying epigastric and left hypochondriac cramping pain radiates to left part of chest. Accompanying symptoms: fever and vegetative signs.
- Physical findings: epigastric and hypochondriac tenderness on both sides, absence of bowel movement, abdominal splash, local défense
    - necrotising pancreatitis: Cullen sign, Grünwald sign, shock
- Investigation: abdominal and chest X-ray, abdominal US, abdominal and pelvic CT scan, blood gas, laboratory: electrolytes, glucose, blood count, renal and hepatic function, CRP, procalcitonin, amylase, lipase, calcium, albumin, INR, haemoculture
    - *NB: If the serum is lipaemic check blood triglyceride, and cholesterol level to rule out lipaemic crisis (cholesterol > 50 mmol/L, triglyceride > 40 mmol/L)!*
- Treatment: maintain vital functions, analgesia (IV spasmolytics, epidural analgesia), nasogastric tube, jejunal feeding, antibiotic therapy, treat complications like ERDS, acute renal failure.
    - In lipaemic crisis perform plasmapheresis!
    - In biliary pancreatitis ERCP should be performed!

Ileus:

Mechanical ileus:

External (strangulation, compression, invagination) or internal (tumour, fecolith, foreign body) obstruction of small or large intestine.

- Patient's history: gradually intensifying diffuse or local abdominal pain, bloating, altered defecation habits, odorous vomiting
- Physical findings: local or diffuse tenderness, meteorism, high pitched tinkling (metallic) bowel sounds, recurrent mass peristalsis, abdominal splash, through NG tube atonic or odorous gastric content empties
- Investigation: abdominal X-ray, abdominal US, abdominal and pelvic CT scan, blood grouping, blood gas, laboratory: electrolytes, glucose, renal and hepatic functions, amylase, lipase, blood count, INR
- Therapy: maintain vital functions, analgesia, nasogastric tube insertion, conservative activation of peristalsis (metoclopramide, physostigmine, enema, laxatives)
- Seek expert help if conservative therapy is insufficient!

Paralytic ileus:

Always a secondary condition provoked by severe diseases like peritonitis, metabolic crises, poisonings, acute abdominal catastrophes. Paralytic ileus occurs even in the most severe cases of mechanical ileus.

- Patient's history: diffuse abdominal pain with vomiting and vegetative signs.
- Physical findings: diffuse abdominal tenderness, meteorism, absence of bowel movement, abdominal splash, atonic gastric content empties from NG tube
- Investigations: abdominal X-ray, abdominal US, CT scan, blood grouping, blood gas, laboratory: electrolytes, glucose, renal and hepatic function, amylase, lipase, blood count, INR, beta hCG, urinalysis
- Therapy: maintain vital functions, specific therapy for the underlying condition, NG tube insertion, analgesia, seek expert help!

Appendicitis:

Occurs in every age group, always keep in mind the possibility of appendicitis!

NB: in elderly patients symptoms may be less characteristics!

- Patient's history: primarily epigastric pain, then intensifying, cramping pain in the right iliac region (McBurney's point), vegetative signs, fever.

- Physical findings: McBurney's point is the focus of abdominal pain + accompanying signs (Rowsing, Blumberg, Hendi and psoas sign), silent bowel movement or absence of bowel movement, défense musculaire, T_{rectal} - $T_{axillary}$> 1°C
- Investigations: abdominal X-ray and US, CT scan, blood grouping, blood gas, laboratory: electrolytes, glucose, renal and hepatic function, amylase, lipase, CR, PCT, blood count, INR, beta hCG, urinalysis
- Therapy: maintain vital functions, analgesia, seek surgical help, appendectomy

Diverticulitis:

Mostly occurs in elderly patients, localised to the sigma, but in rare cases diverticulitis may occur in the whole colon.

- Patient's history: slowly intensifying temporarily cramping left iliac pain, constipation, haematochezia, mucus in the faeces, fever
- Physical findings: left iliac tenderness, fever or sub-febrile temperature, silent bowel sounds, painful rectal digital examination, bloody stool
    - *NB in case of perforation or peritonitis acute abdomen or défense occurs!*
- Investigations: abdominal X-ray and US, CT scan, blood grouping, blood gas, laboratory: electrolytes, glucose, renal and hepatic function, amylase, lipase, CRP, PCT, blood count, INR, beta hCG, urinalysis, faecal culture for microbiological investigations.
- Therapy: maintain vital functions, analgesia, antibiotic therapy, if complications (perforation, peritonitis or abscess) occur, seek surgical help.

Gastro-intestinal perforation:

- Patient's history: sudden onset of cramping unbearable pain with a sudden easing, then gradually worsening condition leads to acute abdominal catastrophe.
- Physical findings: acute abdomen, défense musculaire, absence of bowel movement, shock, vegetative signs, liver cannot be detected by percussion
- Investigations: abdominal X-ray and US, CT scan, blood grouping, blood gas, laboratory: electrolytes, glucose, renal and hepatic function, amylase, lipase, CR, PCT, blood count, INR, lactate

- Therapy: maintain vital functions, analgesia, NG tube insertion, seek surgical help

Mesenteric thrombosis:

Mostly occurs in elderly patients. Risk factors: atherosclerosis and atrial fibrillation without anticoagulant prevention.

- Patient's history: gradually intensifying abdominal pain, nausea, vomiting, bloating, soft bloody stool with mucus in it. Atrial fibrillation and atherosclerosis in the medical history
- Physical findings: diffuse abdominal pain, meteorism, silent bowel movement or absence of bowel movement, raspberry jam-like stool, acute abdomen, shock
- Investigations: ECG, abdominal X-ray and US, CT-angio scan in every cases, blood grouping, blood gas, laboratory: electrolytes, glucose, renal and hepatic function, amylase, lipase, CR, PCT, blood count, INR, lactate
- Therapy: maintain vital functions, analgesia, NG tube insertion, seek surgical help

Abdominal aortic aneurysm rupture:

Always occurs in the form of acute abdomen.

- Patient's history: already known aneurysm or generalised atherosclerosis in the medical history. Sudden onset of abdominal pain, vertigo, loss of consciousness, haemorrhagic shock
- Physical findings: acute abdomen, défense musculaire, absence of bowel movement, pulsating abdominal bulge above the aorta, no pulse on the lower limbs, cold skin and mottling occurs, signs of shock.
- Investigations: ECG, in suspected aneurysm rupture CT-angio scan is the first choice, blood grouping, blood gas, laboratory: electrolytes, glucose, renal and hepatic function, amylase, lipase, CR, PCT, blood count, INR, lactate
- Therapy: maintain vital functions, analgesia, transfusion, seek angio-surgical help

Gynaecological conditions causing abdominal pain:

- Patient's history: sudden onset of suprapubic or iliac pain (ectopic pregnancy, ovarian torsion), or slowly intensifying blunt pain (adnexitis, perimetritis)
- Physical findings:

    - acute abdomen, local défense musculaire, shock -> ectopic pregnancy or ovarian torsion
    - suprapubic pain, sub-febrile temperature or fever -> adnexitis, perimetritis
- Investigations: abdominal and pelvic US, CT scan, blood grouping, blood gas, laboratory: electrolytes, glucose, renal and hepatic function, amylase, lipase, CR, PCT, blood count, INR, beta hCG
- Therapy: maintain vital functions, analgesia, transfusion, immediately seek gynecological help

10. Management of Gastrointestinal Bleeding in the ED by. Dr. Schneider Erzsébet

Gastrointestinal bleeding is a common, often critical emergency condition, challenges ED staff in the most severe cases.

Localisation of bleeding:

a. oral
b. oesophageal – tumour, varix
c. gastric – ulcer, tumour
d. duodenal – ulcer, pancreas tumour
e. small intestinal – IBD, tumour, angiectasia -> 2%
f. large intestinal – IBD, tumour, diverticulosis, haemorrhoid ->18%

Accurately taken history (lifestyle, alcohol consumption, smoking, defecation habits, weight loss, previous diseases and regularly taken medicines) has a great role in making diagnosis of gastrointestinal bleeding.

NB: Always ask for regularly taken anticoagulants!

Symptoms:

- hematemesis, coffee ground vomit
- melena, tarry stool, haematochezia
- sudden fatigue, feeling dizzy, collapse, loss of consciousness
- pain, dark diarrhoea

Physical examination:

- ABCDE approach! (the most important: NIBP, HR, pulse quality, shock index, level of consciousness, pallor, cyanosis…)
- Rectal digital examination (RDE)
    - tarry stool: upper-gastrointestinal subacute bleeding, HCl-haematin gives the black colour

    - melena: huge amount of soft, odorous and raspberry jam-like stool, sign of massive upper-gastrointestinal acute bleeding
    - haematochezia: normal stool mixed with fresh blood, sign of lower-intestinal bleeding
    - occult bleeding: digested blood in normal colour stool
- NG tube:
    - upper-gastrointestinal bleeding can be excluded if green-yellowish fluid (contains bile) passes through the NG tube.
    - If bloody fluid passes through NG tube, upper-gastrointestinal bleeding is confirmed

NB: if clear fluid passes through NG tube, post-pyloric bleeding may occur!

General investigations and therapy:

- Maintain vital functions (haemorrhagic shock therapy: crystalloids then blood transfusion)
- Physical examination, DO NOT FORGET RDE!
- NG tube insertion -> diagnostic and therapeutic at the same time. Giving cold water and drugs through NG tube may stop bleeding and prepare endoscopic examination too.
- Laboratory: electrolytes, glucose, hepatic and renal function, blood count INR
- Blood grouping
- Order blood derivates: RBC concentration, freshly frozen plasma, platelet concentration and prothrombin complex.
- Endoscopy: diagnosis, endoscopic coagulation, ligation or clipping

  NB: endoscopy only can be carried out in patients with normal vital functions!

Management of oesophageal varix bleeding: usually massive bleeding!

- Maintain vital functions (haemorrhagic shock therapy: crystalloids then blood transfusion)
- Physical examination: hepatic cirrhosis in the medical record, continuous hematemesis

- NG tube insertion:careful gastric lavage, NO SUCTION PERFORMED
- Laboratory: electrolytes, glucose, hepatic and renal function, blood count INR
- Blood grouping
- Order blood derivates: RBC concentration, freshly frozen plasma, platelet concentration and prothrombin complex.
- Give octreotide (prolonged effect somatostatin 50µg IV, then 25µg/h via infusion pump)
- Monitor the patient (NIBP, SatO2, GCS, gastric contents passed through tube, blood count)
- Endoscopy: varix ligation, sclerotherapy
- Balloon tamponade if endoscopy was unsuccessful
    - Sengstaken-Blakemore tube insertion (to the stomach through nose)
        - inflate the gastric part of the two-part balloon with 80-120 ml of air
        - pull the tube gently with a weight of 0,5-1kg
        - inflate the oesophageal part of the balloon with 80-100ml of air
        - perform gastric lavage for monitoring bleeding
        - deflate a little both balloon after 4 hrs.
        - DO NOT MAINTAIN tamponade more than 24hrs
- If both endoscopy and balloon tamponade were unsuccessful seek surgical help!

Management of gastric and duodenal bleeding:
The most common underlying condition is peptic ulceration, but bleeding may occur from tumour or angiectasy too.

- Symptoms:
    - hematemesis, coffee ground vomit
    - melena, tarry stool
    - hypoxia, shock

- Therapy:
    - maintain vital functions (give crystalloids and blood derivates)
    - insert nasogastric tube and perform careful gastric lavage
    - laboratory, blood grouping, order blood derivates
    - if the patient is stable, seek expert help to perform endoscopic treatment: infiltration with adrenalin, clipping, laser coagulation ortissue adhesive therapy.
    - Endoscopic Forrest Classification:
        - Forrest IA – squirting bleeding
        - Forrest IB – oozing bleeding
        - Forrest IC – leaking bleeding
        - Forrest IIA – non-bleeding, but visible vessel
        - Forrest IIB – ulcer covered with adherent clot
        - Forrest IIC – non-bleeding haematin covered ulcer
        - Forrest III – fibrin covered ulcer no sign of bleeding
    - Give IV PPI
    - Monitor the patient!

Management of large intestinal bleeding:
In most of the cases tumours or haemorrhoids but rarely IBD and diverticulitis are the source of large intestinal bleeding. Lower-intestinal bleedings requires only occasionally pharmacological therapy to maintain blood pressure or massive transfusion compared to the upper intestinal-bleedings.
Symptoms:

- haematochezia
- hypoxia, shock (rarely)

Management:

- maintain vital functions

- if the localisation of the bleeding is unsure insert NG tube and perform gastroscopy
- laboratory, blood grouping, order blood derivates
- monitor the patient
- urgent colonoscopy if bleeding cannot be stopped
- seek surgical help

Transfusion in gastrointestinal bleedings

The timing of transfusion is depend on the intensity of bleeding and the vital signs of the patient.

Blood (RBC concentration) transfusion: cross matched blood transfusion whenever possible (or in emergency situation – circulatory instability, uncross matched group specific or group O [universal RBC donor] blood transfusion maximum 2 unit). Warm up the RBC concentration to body temperature, use blood warmer.

Fresh Frozen Plasma transfusion: Used in the treatment of primary or secondary coagulation disorders. Use ABO cross matched FFP whenever it is possible, but in case of emergency or lack of group specific FFP give group AB FFP (universal FFP donor).

Platelet concentration transfusion: Used in thrombocytopenia, and in bleeding caused by thrombocyte dysfunction too (e.g. bleeding in patients take regularly thrombocyte aggregation inhibitor). Use ABO cross matched platelet.

Massive transfusion:

- more than 10 units of RBC transfusion in 12 hours
- replacement of the whole blood volume in 24 hours
- replacement of the 50% of the whole blood volume in 3hours
- more than 150ml/min blood loss

Possible complications of massive transfusion:

- hypothermia – use blood warmer
- hypokalaemia – caused by haemolysis, monitor potassium level and give potassium if it is needed
- citrate intoxication – give 500mg Calcium-gluconate for each 2-3 units of blood

- dilutional coagulopathy give RBC and FFP in 3:1 ratio and RBC and thrombocyte in 8:1 ratio

11. Metabolic Crisis in the ED by Dr. Börcsök Éva

Diabetes mellitus (DM):

- Metabolic crisis in patient with DM:
- Hypoglycaemia
- Diabetic Ketoacidosis (DKA)
- Hyperglycaemic Hyperosmolar State (HHS)
- Lactic acidosis

Hypoglycaemia:

1.) Definition:

- plasma glucose < 3mmol/L asymptomatic/biochemical hypoglycaemia
- plasma glucose < 3.5mmol/L clinical hypoglycaemia (trigger counter-regulatory responses)
- plasma glucose < 3.9mmol/L alert value of hypoglycaemia according to American Diabetes Association (ADA)
- the minimum therapeutic goal: 4-4.5 mmol/L (four is the floor)
- plasma glucose < 5.5 mmol/L with hypoglycaemia-like symptoms relative hypoglycaemia (Type 2 DM often accompanied by high HgA1c value)

2.) Classification:

- Severe hypoglycaemia:

  The patient cannot administer glucose or carbohydrates to himself, needs assistance. After glucose administration the patient's condition improved rapidly.
- Documented symptomatic hypoglycaemia:

  Hypoglycaemic symptoms and measured plasma glucose≤3.9mmol/L
- Asymptomatic hypoglycaemia

  No sign of hypoglycaemia, but measured plasma glucose≤3.9mmol/L
- Probable symptomatic hypoglycaemia

  Hypoglycaemic symptoms without low plasma glucose level but likely caused by plasma glucose ≤3.9mmol/L
- Pseudo-hypoglycaemia

  Hypoglycaemic symptoms with measured plasma glucose>3.9mmol/L but approaching that threshold

3.) Aetiology:

A) Inappropriate glucose supply

1.Endocrine disorders:

- hypopituitarism
- adrenal insufficiency

2. Enzyme deficiencies: glucose-6-phosphatase deficiency

3. Insufficient sugar intake:

- starvation and malnutrition
- infancy
- last trimester of pregnancy

4. Hepatic Disorders:

- congestive hepatopathy
- hepatic insufficiency
- hepatitis
- hepatic cirrhosis

5. Hypothermia

6. Uraemia

7. Intoxication

- insulin overdose
- sulfonylurea poisoning
- kinin intoxication
- disopyramide intoxication
- pentamide intoxication

8. Endotoxic shock

9. Pancreas tumour e.g. insulinoma

10. Low Carnitine Level

B) Reactive Hypoglycaemia

1. Alimentary Hyperinsulinism (postprandial elevated plasma insulin level with abnormally rapid gastric emptying)

2. Inherited Fructose Intolerance

3. Idiopathic

4.) Symptoms:

A) Symptoms related to Adrenal Response for Hypoglycaemia:

Epinephrine/Norepinephrine-related:

- Palpitation
- Quivering
- Anxiety

Acetylcholine-related:

- Sweating
- Sense of hunger
- Numbness in lips

B) Symptoms of neuroglycopenia:

- Concentration problems
- Headache
- Fatigue
- Tiredness
- Somnolence
- Shivering
- Double vision
- Vertigo
- Confusion
- Generalized or focal seizures

C) Coma

5.) Epidemiology:

- Type 1 DM: hypoglycaemia is fairly common among Type-1 diabetes patients, for the glucose elimination depends on the pharmacokinetics of insulin therapy and the counter-regulatory hormone secretion is impaired. Living with Type 1 DM for 5-10 years causes delayed glucagon-response, impaired catecholamine, human growth hormone (hGH) and cortisol secretion. Weakening neuroglycopenic alert signs are caused by the neuropathy of the Autonome Nervous System. It starts with the absence of the Adrenal response due to the damage of the Sympathetic Nervous System causing more sever hypoglycaemia.

More hypoglycaemic episodes have happened before, the greater the chance is for further episodes.

- Type 2 DM: Presence of hypoglycaemia is depend on the severity of Type 2 DM. Decreased beta-cell function, renal and hepatic insufficiency and increased physical

activity predispose hypoglycaemia. Anti-diabetic treatment overdose (insulin, sulphonylureas).

6.) Pathophysiology:

Physiologic glucose counter-regulation: low plasma glucose level has a negative feedback on pancreatic insulin secretion, and triggers the secretion of glucagon, epinephrine, cortisol and hGH, consequently plasma glucose level rises. In Type 1 DM this counter-regulatory mechanism is damaged: low plasma glucose level has less inhibitory effect on insulin secretion, less or no glucagon response is triggered and the epinephrine level remains lower, therefore plasma glucose level remains low or decreases further.

Generally:

- vegetative dysfunctions and neurologic symptoms occur: <3mmol/L plasma glucose
- cognitive dysfunctions occur <2.7mmol/L
- hepatic counter-regulation starts < 2mmol/L.

7.)Diagnosis: simple blood glucose measurement.

8.) Treatment:

CONSCIOUS PATIENT:

- 10-20g fast-absorbing carbohydrates + 10-20g slow-release carbohydrates
- 0.5-1.5dL regular (non-diet) soda or 2-3 lumps of sugar in a glaas of water + 10-20g slow-release carbohydrates

UNCONSCIOUS PATIENT:

- 10-40ml 40% glucose or 75-80 ml 20% glucose iv. Then 5% glucose drip. If the patient recovers consciousness → oral feeding (see above)
- Glucagon injection (route of administration: sc./im./iv.)
    - **Glucagon is contraindicated in patient with Type 2 DM !!!**

PROLONGED HYPOGLYCAEMIA:

Administer: diazoxide or octreotide (← insulin secretion inhibitors)

9.) Hypoglycaemia as a riskfactor:

- Higher cardiovascular risk
    - elevated risk of myocardial ischaemia and necrosis
    - elevated risk for arrhythmias:
        - higher heart rate variability
        - elevated sympathetic tone
        - changes in repolarisation

    - prolonged QT interval
    - changes in T morphology
    - ventricular arrhythmias occur
- neurologic signs can mimic and also cover the symptoms of stroke
- dementia
- trauma

Acidosis related to diabetes mellitus

1. Ketoacidotic states:

a.) Euglycaemic Ketoacidosis

b.)Alcoholic Ketoacidosis

c.)Starvation Ketoacidosis

2. Other metabolic Acidotic states

a.) Lactate Acidosis

b.) Hyperchloraemic Acidosis

c.) Salicylate Poisoning

d.) Uraemic Acidosis

e.) Drug induced Acidosis

A) Diabetic Ketoacidosis (DKA)

Mostly occurs in patients with Type 1 DM, (often this is the first sign of the Type 1 DM).

1.) Diagnostic criteria:

- plasma glucose > 13.9mmo/L
- pH < 7.35
- HCO3 < 18mmol/L
- High anion gap
- plasma ketone body +

2.)Pathophysiology:

As a result of the absolute insulin deficiency, the level of the counter-regulatory hormones (glucagon, cortisol, hGH, catecholamines) elevate consequently the glucose utilisation decreases. With increased lipolysis free fatty acid level elevates and ketogenesis appears causing ketoacidosis. With the elevated FFAs the triacil-glycerol level rises and causes hyperlipidaemia. As a compensatory mechanism HCO3 level decreases. Counter-regulatory response elevates protelysis and inhibits the protein synthesis therefore amino acid (AA) level

elevates. With the elevated level of substrates (FFAs and AAs) hepatic gluconeogeneis and glucogenolysis increases causing hyperglycaemia. When the hyperglycaemia reach the threshold of the kidneys glucosuria and osmotic diuresis appears and leads to dehydration and loss of electrolytes. Ketoacidosis causes nausea and vomiting which aggravates the dehydration and causes hyperosmolarity. Hyperosmolarity leads to renal impairment.

In DKA altered mental status is a result of hyperosmolarity and acidosis. The glucose supplementation of the neurons is independent from the insulin level, because glucose can filtrate through the blood brain barrier (BBB).

3.) Aetiology:

- Unknown in 20% of all cases
- Infection (source control!!!) 37%
- Maladministration of Insulin 21%
- Alcohol or Drug abuse 10%
- Endocrine diseases
- Pancreatitis or any other acute abdomen sy. 8%
- Acute Myocardial Infarction 5%

4.) Symptoms:

- Excessive urine output and excessive thirst
- Breath smells like acetone
- Nausea and vomiting, dehydration
- Loss of appetite loss of weight
- Muscle aches, stomach-ache (diabetic pseudo-peritonitis pH < 6.9)
- Kussmaul breathing (pH 7.0-7.2)
- Altered mental state → Coma
- Hypotension

5.) Laboratory Findings:

- elevated plasma glucose level (also from BMG, or arterial blood gas sample)
- metabolic acidosis
- high blood urea nitrogen (BUN)
- low plasma Na level, normal or high corrected Na
- normal or high K level
- leukocytosis (elevated WBC) (not exceptionally in infections)

- high amylase level (not exceptionally in pancreatitis)

6.) Other forms:

- sever hypo perfusion → elevated lactate and ß-hydroxy-butyrate level → high anion gap, low keton body level
- DKA in pregnancy: slightly elevated plasma glucose level but more progressive ketoacidosis (elevated cortisol, prolactin and placental lactogene hormone)
- DKA with normal or high pH (vomiting, antacids, Cushing sy.)

7.) Classification:

	Mild	**Moderate**	**Sever**
Plasma glucose	>13.9 mmol/L	>13.9 mmol/L	>13.9 mmol/L
pH	7.25-7.3	7.0-7.24	<7.0
Plasma $HCO3^-$	15-18 mmol/L	10-15 mmol/L	< 10 mmol/L
Ketone body plasma/urine	+/+	+/+	+/+
Anion gap	>	>	>
Osmolarity	Variable	Variable	Variable
Mental state	Alert	Drowsiness	Stupor/Coma

8.) Differential Diagnosis:

- Starvation: normal plasma glucose, normal plasma ketone body level, KETONURIA
- Uraemia
- Lactic acidosis
- Poisoning
  - methanol, ethylene glycol, salicylic acid → high AG + no ketonaemia
  - isopropyl alcohol → normal AG + ketonaemia
- Other forms of Metabolic Acidosis
- Prolonged alcohol consumption + vomiting → starvation + high ß-hydroxy-butyrate level

9.) Therapy:

1. Fluid replacement: fluid demand may be 5-8L!!!

The total fluid loss has to be supplemented in the first 12-24h!!!

- 1st h: 1000-1500 ml/h 0.9% Saline
- 2-3h: 500 ml/h 0.9% Saline

- 4-8h: 200-500 ml/h 0.9% Saline

Take into consideration:

- prolonged illness: higher demand
- chronic renal impairment: fluid retention
- elderly or patient with chronic heart failure: Aggressive fluid therapy may lead pulmonary oedema (CVP>10 cmH_20)
- Give glucose under 12-14 mmol/L
- In hypovolemic shock give 1000ml Saline
- Normal or high Na level give 0,45% Saline 4-14ml/kg/h
- Low Na level (pseudohyponatraemia): 0.9% Saline 4-14 ml/kg/h
- Patient monitoring (cardiogenic shock)

2. Insulin therapy:

- Rapid Acting Insulin 0.1 E/kg iv
- Infusion pump Rapid Acting Insulin 0.2 kg/kg/h iv. OR 0.1 E/kg/h im. (rarely)

goal: 3mmol/L/h decrease of plasma glucose (the dosage can be doubled)

- IF plasma glucose 12-14 mmol/L → give 5%-os Dextrose and continue Rapid Acting Insulin 0.05-0.1 E/kg/h.

3. Potassium supplementation:

Potassium supplementation is essential in all patients but patient with chronic renal impairment or patient with K > 5.0 mmol/L

Give potassium in the 2nd h of the treatment

Goal plasma K level: 4-5 mmol/L

- plasma K <3 mmol/L => 30 mmol/h
- plasma K 3.5-4.0 mmol/L =>40 mmol/h
- plasma K 4.5-5.0 mmol/L =>20-30 mmol/h

4. Sodium Bicarbonate supplementation:

Routinely given sodium bicarbonate is not recommended. Adequate Insulin and Fluid Regiments are usually sufficients. The given insulin inhibits the ketogenesis, and facilitates the oxidation of ketone bodies and the endogenous bicarbonate production.

Bicarbonate supplementation is indicated if the pH < 7.0

- pH 6.9-7.0 => 50 mmol NaHCO3 in 400 ml distilled water + 10 mmolKCl solution 200 ml/h iv.
- pH < 6.9 => 110 mmol NaHCO3+ 400 ml in distilled water + 20 mmolKCl

solution 200 ml/h iv.

Can be repeated in every 2 hours with regular K level control.

5. Phosphor supplementation is not recommended.

6. Additional therapy:

- eliminate the causes of DKA (in case of infection give antibiotics)
- Coma: intubation, O2 supplementation, nasogastric tube, monitor the urine output.

10.) Investigations:

- control plasma glucose every hour for 6 hours, then every 2 hours
- control plasma Na and K every hour for 6 hours, then every 2 hours
- arterial blood gas measurement every 2 hour for 6 hours, then every 6 hours
- BUN, Creatinine, amylase, haemoglobin, WBC, urine acetone, urine glucose measurement in every 4 h-s
- ECG monitoring
- Mental state evaluation
- Physical status
- Monitor: ECG, CVP, HR, NIBP, body temperature, Resp. Rate
- Fluid balance monitoring

B) Hyperglycaemic Hyperosmolar State:

1) Incidence: 1/1000 patient with DM/year

2) Clinical Appearence:

- hyperglycaemia with severe dehydration
- developed in days
- drowsiness, temporary lucidity then coma
- high HR, low NIBP, low CVP
- hypernatraemia
- uraemia (not renal origin)
- cerebral oedema
- high mortality 5-10%

3) Aetiology:

- elderly patient
- Type 2 DM
- acute stress:

- Infection
- STROKE
- Myocardial Infarction
- Excess glucose intake or parenteral feeding
- Diuretics
- Steroids
- Hydantoin
- Energy drinks (higher occurrence in children)
- Misuse of anti-diabetic medications

4) Pathophysiology:

Hyperglycaemia and extracellular hyperosmolarity are caused by relative lack of insulin (imbalance of insulin-glucagon ratio). Osmotic diuresis appears. Loss of water and sodium causes initially hypernatremia, then with the excessive dehydration (renal loss, sweating, etc.) causes normonatremia. Due to altered mental state water intake decreases. Finally hypernatraemia occurs. Intracellular water deficit causes cerebral dehydration facilitating intracellular osmo-active agent production which leads to intracellular rehydration then cerebral oedema.

5) Signs and symptoms:

- Patient History, Clinical appearance: (dehydration, altered mental status)
- Plasma glucose level of 25mmol/L or greater
- Effective serum osmolarity of 361 mOsm/L or greater
    - Calculation of Serum Osmolarity = (2 x (seNa + seK)) + BUN + pl. glucose
- Serum pH greater than 7.30
- Bicarbonate concentration greater than 15 mEq/L
- Small ketonuria and absent-to-low ketonemia
- Some alteration in consciousness

6) Therapy:

- EARLY STAGE OF HHS: hyperglycaemia, hyponatraemia, cerebral dehydration
    - 1000ml/h 0,9% Saline
    - 0,1 IE/kg/h Rapid Acting Insulin drip.

- SEVER HHS: hyperglycaemia, hypernatremia, cerebral oedema
  - mannitol/glycerine in every 6-8hs
  - Fluid therapy: 0,9% Saline then 0,45% Saline
  - Insulin therapy
- ADDITIONAL THERAPY
  - eliminate the causes of DKA (in case of infection give antibiotics)
  - Coma: intubation, O2 supplementation, nasogastric tube, monitor the urine output.

3. Lactic Acidosis

1) Definition: Lactate is the end product of glucose metabolism. Lactic acidosis is a form of metabolic acidotic states caused by elevated se. lactate and se. H+ level. Mitochondrial dysfunction leads to elevated lactate production (a negative ion) and higher anion gap.

Anion gap: = ([Na+] + [K+]) − ([Cl-] + [HCO3−])

2) Classification:

- Type A:Decreased perfusion or oxygenation
  - Shock (cardiogenic, septic, hypovolemic)
  - Heart Failure
  - Asphyxia (lack of oxygen)
- Type B:
  - 1: Underlying diseases:
    - DM
    - neoplasm
    - renal or hepatic diseases
    - convulsive diseases
    - malign hyperthermia
    - local tissue hypoperfusion or asphyxia
  - 2: Poisoning:
    - Carbon monoxide
    - Biuguanids
    - Ethyl or methyl alcohol
    - Salicylate
    - Fructose, Sorbitol, Xylitol
  - 3: Inborn metabolic disorders

3.) Signs and symptoms:

- Clinical signs: severe metabolic acidosis
- Laboratory Findings:
  - se Lactate > 5 mmol/L
  - HCO3- <15 mmol/L
  - pH < 7.3
  - AG > 16mEq/L (normally 8-16mEq/L)
  - Lactate/pyruvate Ratio > 10
  - Inorganic PO4-/se Creatinine Ratio > 3.

4) Treatment:

- Treat the underlying causes.

4. Thyroid Storm

1.) Definition: acute exacerbation of known or unknown hyperthyroidism

2.) Causes:

- Thyroxine overdose
- Thyroid gland surgery
- Thyroid adenoma or carcinoma (rare)
- Drugs contain iodine
  - expectorants
  - contrast medium
  - antiseptics
  - raidoiodine therapy
- Insufficient or stopped thyrostatic therapy
- Stress:
  - Infection
  - Trauma
  - Hypoglycaemia
  - Surgery

3.) Symptoms of Hyperthyroidism:

- fatigue
- loss of weight (without loss of appetite)
- sweating, heat intolerance

- shaking hands
- anxiety
- insomnia
- decreased libido
- widened eyes
- palpitation atrial fibrillation (among elderly)
- hot sweaty skin
- hair loss
- gynecomastia among men
- amenorrhoea
- osteoporosis
- thyrotoxic cardiomegaly, heart failure

Thyroid storm:

a.) Neurologic signs:

1. stage	2.stage	3.stage
Tremor, anxiety	Drowsiness	Coma
Hyperkinesia	Psychosis	
Fatigue		

b.) Cardiovascular signs:

1. stage	2.stage	3.stage
Tachycardia > 150/min	The same as in the 1.stage+	Hypotension
Tachyarrhythmia	Elevated R wave amplitude	Shock
Hypertension	Hypovolemia	

c.) Respiratory signs:

1. stage	2.stage	3.stage
High Respiratory Rate	Severe Dyspnoea	Respiratory Failure
Dyspnoea		
Hyperventilation		

d.) Gastrointestinal signs: nausea, vomiting, dehydration

e.) Urine output:

1. stage	2.stage	3.stage
Oliguria	Anuria	Uraemia

f.) Hyperthermia, excessive sweating

4.) Laboratory Findings: (There is no fast specific diagnostic measurement)

- T4 > 155nmol/L
- T3 >3nmol/L
- Thyroxine-Binding Index < 90
- High Leukocyte Count
- se Cholesterol < 3,8mmol/L
- High BUN, se Creatinine
- Low Urine Creatinine

5.) Investigations:

a) Monitoring:

- HR
- NIBP as soon as possible IABP
- CVP
- ECG
    - NB: VF is fairly common in severe cases of Thyroid Storm
- Body Temperature

b) Laboratory:

- Hormones: T4, T3, TSH, PBI (protein-binding index), TBI (thyroxine-binding index)
- Na, K, Ca, Mg
- BUN, Creat.
- Coagulation
- Blood count
- Hepatic enzymes
- CK
- Arterial Blood Gas Sampling

6) Therapy:

a) Fluid replacement:

- Give glucose and NaCl in 2:1 ratio
- KCl in case of low K
- MgSO4 1-6g/24h in infusion pump
- Amino Acid inf.: 50-100 ml/kg/24 h

b) Sedation:

- diazepam/midazolam iv.

c) Heart Rate Control:

- beta-blockers: metoprolol, propranolol, bisoprolol
- digitalis

d) Antibiotics: in case of suspected infection

e) LMWH

f) Vitamins, co-carboxylase

g) Hypercaloric Feeding

i) Organ Support Therapy

j.) Definitive Treatment:

- **Favistan/Methamyzol (thiamazole):** initially 80 mg iv. =>after 1-2hs 160-240 mg infusion pump.
- **Administeriodinecompounds**900-1400 mg Iodine
- **Lithium:** 1-1.5g/h oral => control Li level! (1mmol/L)
- **Hydrocortisone:**100-300 mg/24h infusion pump

If there is no change in symptoms of the 1st stage for 24 hours:

- **Plasmapheresis:** 1500 ml plasma from 3000 ml full blood
- **Hemoperfusion:** with carbon filter
- **Peritoneal dialysis**

5. Addison Crisis

Acute failure of the adrenal cortex with life-threatening symptoms. It could be the first sign of chronic adrenal failure.

1) Causes:

- TB
- Autoimmune adrenal disease
- Hereditary Diseases:
  - Adrenoleucodystrophy

    - Adrenomyeloneuropathy
    - Congenital Adrenal Hypoplasia
- Disseminated Mycotic Infections
- AIDS
- Amyloidosis
- Sarcoidosis
- Haemochromatosis
- Adrenal Lymphoma
- Adrenal metastasis
- Steroid Synthesis Inhibitors
- Surgical removal of both adrenal glands

Is there any information about current hormone therapy for adrenal failure?

Yes	No
Misuse (patient failed to take medicine)	Bilateral Adrenalectomy
Vomiting	After surgical removal of cortisol secreting adenoma
Increased glucocorticoid demand	Unknown Addison disease
Fever	Thyroid hormone supplementary treatment
Surgical intervention	Sever disease with fever
Drugs increase glucocorticoid use (barbiturate, rifampicin, phenytoin)	Trauma, surgical intervention
	Drugs increase cortisol metabolism, or steroid biosynthesis
	Bilateral Adrenal Haemorrhage (Waterhouse-Friedrichsen Sy.)

2. Signs and Symptoms:

- fatigue
- muscle weakness
- Painful joint and muscles
- Loss of weight
- Loss of appetite
- Abdominal pain

- Skin and Mucosal Hyper-pigmentation
- Dry skin
- Hypotension
- Hypoglycaemia
- Anaemia
- Hunger for salt
- Altered Mental State
- Amenorrhoea

In acute Adrenal Failure (like Waterhous-Friedrichsen Sy.) the chronic symptoms are absent.

Symptoms of acut Adrenal Failure:

- shock
- Acute abdomen
- fever
- nausea&vomiting
- Chest pain
- Drowsiness
- Malaise
- Anxiety
- Stupor
- Dehydration
- Hypotension
- Tachycardia
- Extra-renal Uraemia
- Electrolyte and Metabolic Disturbances

3.) Investigations:

a.) Laboratory Findings:

Se Na	<135 mmol/L
Se K	> 5 mmol/L
Se Ca	> 2.6 mmol/L (rare)
pH	Metabolic Acidosis
Renal function	Extra-renal uraemia

Se Glucose	low
Blood Count	Anaemia, high WBC, high Eosinphil count
Plasma Cortisol	<5mg/dL (<140nmol/L)
Plasma ACTH	>100 (often >1000pg/mL)
Short ACTH stimulation test show no or minimal elevation	Low plasma cortisol level

b.) Imaging :

- Abdominal CT
- Cranial MR
- If TB suspected: source control (chest X-Ray, Mantoux test, Bacteriological sampling)
- Autoimmune Investigations

4. Therapy: Unless prompt and effective therapy the mortality of Adrenal Crisis is high. But with effective therapy the life-threatening symptoms can be suppressed in 24-48hs.

High Dose of glucocorticoids:

- Hydrocortisone: slowly administered, 200-400 mg/day then in gradually decreased amount for 2-5 days (divided in 4 portions/day)
- Glucocorticoid + mineralocorticoid combined therapy: give long acting glucocorticoid before hydrocortisone e.g. prednisolone or dexamethasone.
- Fluid therapy: give 2000-3000 ml 0,9% Saline in the first 24hs.
- DO NOT replace Potassium!
- Give Glucose
- Temporary blood pressure support
- Give broad spectrum Antibiotics
- Treat underlying disease or trauma

8. Hyperlipidaemic Crisis

The third most common cause of pancreatitis

1) Classification:

a.) Primer hypertrygliceridaemia:

- Hereditary Dysfunction:
  - lipoprotein lipase deficiency
  - apo-C2 defect

b.) Secunder hyperlipidaemia

2) Causes:

- obesity
- DM insulin resistant type
- pregnancy
- excessive alcohol intake
- Drugs: beta-blockers, thiazide, oestrogene

3) Investigations:

4) Treatment:

- Fluid replacement
- Insulin therapy: Insulin elevates lipoprotein lipase activity 0.1-0.3IU/kg/h iv. pump
- Unfractionated Heparin: enhance endothelial lipoprotein lipase release
- Plazmapheresis: if se. Trygliceride> 40mmol/L
- Fibrates

12. Management of Intoxication in the ED by Dr.Hankovszy Péter

1. General Toxicology

Toxicology examines the effect of exogenous molecules on the human body.

Toxicology subspecialties: legal, clinical, environmental, analytical and ecotoxicology.

Clinical toxicology focuses on the treatment of poisoning/overdose of any toxic agent. Poisoning/overdose can be acute or chronic, deliberate or accidental.

Clinical signs and symptoms of poisoning and the factors affect the outcome.

- The patient's condition
- The characteristics of the toxic agent
- The dose of the toxic agent
- The timing of the exposure to the toxic agent
- The site of exposure to the toxic agent

Poisoning by toxic agents causes various symptoms or group of symptoms. The severity of the poisoning is depend on the previous health condition, comorbidities, the dose of toxic agent and the time of exposure. Drug poisoning may present typical symptoms.

Definitions:

- Poison: exert physical, chemical or physicochemical effect on the body even in small doses, causing temporary or permanent pathological disturbances in the vital functions.
- Poisoning: the pathological condition caused by the exposure to the poison. May be accidental but deliberate too.
- Toxin: poison produced by a living organism
- Toxicity: the effectivity (the poisonous capacity) of the poison (the lower the toxic dose is, the more toxic the poison is.)
- Dose: the quantity of the poison the body exposed to.
- Exposure time: the length of the poison exposed to the body (Haber's rule: brief exposure to high dose poison has the same effect as long exposure to low dose poison.)

- Cumulative capacity: multiple low doses of poison, which have solely no poisonous effect, but as they accumulated in the body their additional dose provoke symptoms of poisoning.
- Site of exposure: gastrointestinal, respiratory, dermal, ophthalmic, intravenous and urinary.
- Incubation period: the period between the exposure and the onset of signs and symptoms.
- Previous health condition: the severity of the poisoning is depend on the previous health condition.
- Sensitivity: children, elderly patients and red haired, pale skinned patients are more sensitive than others.
- The concentration of the poison: depend on the dose and the eliminatory processes.
- Acute poisoning: high dose poisoning with rapid onset of symptoms (1-2 hours - 1-2days)
- Subacute poisoning: lower repeated doses with later onset of symptoms (weeks, months)
- Chronic poisoning: low repeated or continuous doses with late onset of symptoms (months, years)

Toxicity of poisons:

LD50	lethal oral dose
extremely toxic	1 mg/kg
highly toxic	1-50 mg/kg
moderately toxic	50-500 mg/kg
mildly toxic	0,5-5 g/kg
slightly toxic	5-15 g/kg 0,5-1 litre
practically nontoxic	15 g/kg more than 1 litre

Defence mechanisms against poison exposure:

- Stop further exposure: glottal constriction, palpebral constriction andnausea

- Elimination of the poison: lacrimation, coughing, sneezing, sweating and vomiting
- Metabolism of the poison: hepatic detoxification
- Elimination from the circulation: via urine output, dermal and skeletal storing

Classification of different types of toxins and poisons:
Effect: selective or broad effect poisons
Origin: plants, animals, minerals and synthetic
Site of exposure: dermal, gastrointestinal and inhaled
Target: general cell poisons, corrosives, enzyme poisons and hematologic poisons
Effect of mechanism: summative poisons, cumulative poisons, concentrative poisons
Practical emergency categorization:

- inhaled poisons
- industrial solvents
- corrosives
- pesticides
- drugs
- other

Investigations, diagnostic procedures:

- Take accurate history from the patient and his relatives
    - What kind of poisons were at home?
    - Check the bin and the lavatory!
    - Check previous medical history! – previous suicidal attempt?
    - Actual suicide attempt/Suicide Note?
- Examination:
    - ABCDE approach!
    - common score system: Poison Severity Score
- ECG, Laboratory (special toxicology laboratory), Imaging and Endoscopy

Poisoning Severity Score:

ORGAN	NONE	MINOR	MODERATE	SEVERE	FATAL
	0 No symptoms or signs	1 Mild, transient and spontaneously resolving symptoms or signs	2 Pronounced or prolonged symptoms or signs	3 Severe or life-threatening symptoms or signs	4 Death
GI-tract		• Vomiting, diarrhoea, pain • Irritation, 1st degree burns, minimal ulcerations in the mouth • Endoscopy: erythema, oedema	• Pronounced or prolonged vomiting, diarrhoea, pain, ileus • 1st degree burns of critical localization or 2nd and 3rd degree burns in restricted areas • Dysphagia • Endoscopy: ulcerative transmucosal lesions	• Massive haemorrhage, perforation • More widespread 2nd and 3rd degree burns • Severe dysphagia • Endoscopy: ulcerative transmural lesions, circumferential lesions, perforation	
Respiratory system		• Irritation, coughing, breathlessness, mild dyspnoea, mild bronchospasm • Chest X-ray: abnormal with minor or no symptoms	• Prolonged coughing, bronchospasm, dyspnoea, stridor, hypoxemia requiring extra oxygen • Chest X-ray: abnormal with moderate symptoms	• Manifest respiratory insufficiency (due to e.g. severe bronchospasm, airway obstruction, glottal oedema, pulmonary oedema, ARDS, pneumonitis, pneumonia, pneumothorax) • Chest X-ray: abnormal with severe symptoms	
Nervous system		• Drowsiness, vertigo, tinnitus, ataxia • Restlessness • Mild extrapyramidal symptoms • Mild cholinergic/anticholinergic symptoms • Paraesthesia • Mild visual or auditory disturbances	• Unconsciousness with appropriate response to pain • Brief apnoea, bradypnoea • Confusion, agitation, hallucinations, delirium • Infrequent, generalized or local seizures • Pronounced extrapyramidal symptoms • Pronounced cholinergic/anticholinergic symptoms • Localized paralysis not affecting vital functions • Visual and auditory disturbances	• Deep coma with inappropriate response to pain or unresponsive to pain • Respiratory depression with insufficiency • Extreme agitation • Frequent, generalized seizures, status epilepticus, opisthotonus • Generalized paralysis or paralysis affecting vital functions • Blindness, deafness	

ORGAN	NONE	MINOR	MODERATE	SEVERE	FATAL
	0 No symptoms or signs	1 Mild, transient and spontaneously resolving symptoms or signs	2 Pronounced or prolonged symptoms or signs	3 Severe or life-threatening symptoms or signs	4 Death
Blood		• Mild haemolysis • Mild methaemoglobinemia (metHb ~10-30%)	• Haemolysis • More pronounced methaemoglobinemia (metHb ~30-50%) • Coagulation disturbances without bleeding • Anaemia, leukopenia, thrombocytopenia	• Massive haemolysis • Severe methaemoglobinemia (metHb >50%) • Coagulation disturbances with bleeding • Severe anaemia, leukopenia, thrombocytopenia	
Muscular system		• Mild pain, tenderness • CPK ~250-1,500 iu/l	• Pain, rigidity, cramping and fasciculation • Rhabdomyolysis, CPK ~1,500-10,000 iu/l	• Intense pain, extreme rigidity, extensive cramping and fasciculation • Rhabdomyolysis with complications, CPK ~>10,000 iu/l • Compartment syndrome	
Local effects on skin		• Irritation, 1st degree burns (reddening) or 2nd degree burns in <10% of body surface area	• 2nd degree burns in 10-50% of body surface (children: 10-30%) or 3rd degree burns in <2% of body surface area	• 2nd degree burns in >50% of body surface (children: >30%) or 3rd degree burns in >2% of body surface area	
Local effects on eye		• Irritation, redness, lacrimation, mild palpebral oedema	• Intense irritation, corneal abrasion • Minor (punctate) corneal ulcers	• Corneal ulcers (other than punctate), perforation • Permanent damage	
Local effects from bites and stings		• Local swelling, itching • Mild pain	• Swelling involving the whole extremity, local necrosis • Moderate pain	• Swelling involving the whole extremity and significant parts of adjacent area, more extensive necrosis • Critical localization of swelling threatening the airways • Extreme pain	

Persson H, Sjöberg G, Haines J, Pronczuk de Garbino J, Poisoning Severity Score: Grading of acute poisoning. Journal of Toxicology – Clinical Toxicology (1998) 36:205-13

General rules of detoxification therapy:

Intoxicated patient need prompt and complex therapy.

- Specific therapeutic choices:
    - Decontamination: elimination of the poison, which has not been absorbed yet.

- Elimination of the already absorbed poison.
- Giving Antidote.

- Non-specific Therapy: supportive therapy using ABCDE approach.

Decontamination:

External decontamination: pesticides, corrosives, parasites, radioactive substances and in every contaminated patients.

Internal decontamination:

Induced Emesis:

Indications: no other way of elimination, full stomach and less than 60 minutes after the exposure.

NB: Induced emesis can be performed only if the patient is absolutely conscious.

Contraindications: unconscious patient or in case of convulsions, collapse or shock, pulmonary oedema, trismus, oesophagus stricture. No pharyngeal reflex. Pregnancy.

Poisons contraindicate induced emesis: corrosives, soaps, foamy poisons, organic solvents, irritants.

Gastric Lavage:

Just in case of lethal poisoning!

Position the patient: Trendelenburg position on the patient's left side. Use 36-40Ch tube with lubrication. Lavage: 200-300 warm clear water in boluses till al the gastric content is eliminated.

NB: if the airways may not be free, perform tracheal intubation before gastric lavage!

Absolute indications of Gastric Lavage:

Poisons indicate Gastric lavage: organic phosphate-esters, paraquat, barbiturates, cyanide, ethylene glycol, poisonous fungi, methanol, nicotine and carbon tetrachloride.

Contraindications of Gastric Lavage:

Conditions contraindicate Gastric Lavage: aortic aneurism, oesophageal varix, oesophageal diverticulum, oesophageal tumour, oesophageal stricture, severe gastric ulceration, gastric tumours, threatening respiratory arrest, oedema of glottis or laryngospasm, pulmonary oedema, previous oesophageal surgery.

NB: In suspected corrosive poisoning Gastric Lavage is absolutely contraindicated!

Purgation: not used in management of intoxicated patients

Intestinal Lavage

1500-2000ml/hr polyaethylenglycol via duodenal or jejunal tube, additional IV metoclopramide is recommended. Intestinal lavage does not affect the electrolyte balance of the body.

Poisons indicate Intestinal Lavage: enterosolvent drugs, drugs contain iron, verapamil, theophylline, ampicillin, aspirin, lithium, paraquat, seeds and arsenic poisoning

Activated carbon

Activated carbon is a general antidote. Dosage: 1g/kg in 1every 2-4 hrs.

Poisons indicate Activated Carbon therapy: carbamazepine, digoxin, nadolol, barbiturate, glutethimide and theophylline.

Conditions contraindicate Activated Carbon therapy: GI bleeding, suspected perforation, unsafe airways and corrosive poisoning.

Forced diuresis – questionable effectivity.

Urine acidification with ammonium chloride, or arginine hydrochloride

Poisons indicate urine acidification: amphetamine, quinine, caffeine, lidocaine, theophylline and tricyclic antidepressants (TCAs)

Urine alkalization with sodium bicarbonate

Poisons indicate urine alkalization: barbiturates, methanol and salicylate.

Haemodialysis

Elimination of hydrophilic particles, smaller than 500D which has low endogenous clearance. Protein-bound poisons can be eliminated slowly with haemodialysis.

Poisons indicate haemodialysis: valproic acid, atenolol, propranolol, ethanol, ethylene glycol, methanol, isopropyl alcohol, salicylate, amphetamine, MAO inhibitors, amanitin, dicquat, DNOC (dinitro-o-cresol), and potassium or sodium salts.

Haemoperfusion

Elimination (with a filter) of low protein bound poisons, which has low endogenous clearance.

Poisons indicate haemoperfusion: barbiturate, glutethimide, promethazine, acetaminophen, INH, amitriptyline, amanitin, diquat, paraquat, diltiazem, aminophylline, carbon tetrachloride and carbamazepine.

Plasmapheresis:

Elimination of highly protein bound poisons.

Poisons indicate plasmapheresis: snake venoms, amitriptyline, calcium antagonists and amanita phalloides.

Antidotes:

An ideal antidote must be fast-acting, cheap, non-poisonous and it must have a long expiration date too. Unfortunately few poisons have antidotes and usually these antidotes are available only in greater clinics. Antidotes are mostly very expensive, but ordering antidotes when it is needed is too late in the most of the cases.

NB: Time is the main factor in antidote therapy!

Symptoms of Poisoning:

Specific signs are rare, poisoning may cause any symptoms.

NB if the patient has bizarre symptoms or unexplained worsening of the presenting symptoms always keep in mind the possibility of poisoning!

Skin and mucosal signs:

Organ	Sign	Poison
Lips, Tongue, Pharynx	swelling, mucosal lesion	corrosives or caustic poisoning, heavy metal
	yellowish lesion	picric acid
	orange lesion	chromate, potassium-bichromate
	brown lesion	nitric acid, iodine, bromide
Gingiva	purple	arsenic poisoning, antimony
	blueish	lead, mercury
	black	bismuth
	yellow	cadmium
Skin	pallor	cocaine, nicotine, opium, amphetamine, other stimulants
	flushed, dry and may be urticarial lesion	atropine, nicotine acid, scopolamine, nitro-glycerine
	cherry like	carbon monoxide
	yellow	picric acid
	jaundice	arsenic poisoning, phosphor, carbon tetrachloride
	ulceration	corrosives, bromide, iodine, fluor hydrogen acid
	suffusion	benzol, phosphor, carbon tetrachloride
	mottling	nitric gases, opiates, carbon dioxide

Eyes:

Pupils	dilated – mydriasis (+tachycardia)	sympathomimetics: amphetamine, cocaine, LSD… anticholinergics: antihistamine, atropine, TCA
	narrowed – miosis	sympatholytics: clonidine,

	(+bradypnoe)	opiates, valproic acid cholinergics: nicotine, carbamate, organic phosphates
Nystagmus		barbiturate, ethanol, carbamazepine, phenytoin, scorpion venom, PCP

Respiratory and Cardiac signs

Respiratory	bronchospasm	beta blocker, smoke inhalation, hydrocarbon inhalation, organophosphates, irritant gases
	hypoxia	inert gases, irritant gases…
Cardiac	tachycardia	stimulants (amphetamine, methamphetamine, cocaine) PCP, theophylline, atropine, antihistamine, thyroid hormones
	bradycardia	antiarrhythmic drugs (Ia,Ic), beta blocker, Ca-channel blockers, carbamate, clonidine, TCA, digoxin, quinidine, lithium, metoclopramide, opioids, organophosphates, physostigmine, propoxyphene
	hypotension	sympatholytic drugs, TCA, barbiturate, Ca-channel antagonists, meprobamate
	hypertension	amphetamine, cocaine, ephedrine, LSD, MAO inhibitors, anticholinergic drugs, nicotine
	broad QRS	beta blocker, digitalis, hyperkalaemia, TCA, phenothiazine, quinidine

Abdominal signs

Abdominal pain	cramps, diarrhoea (+hallucination, tachycardia)	ethanol, benzodiazepine, opiates, withdrawal sy.
	ileus (paralytic or mechanic)	sympatholytic drugs, anticholinergic drugs
	ischaemic bowel necrosis (due to vasospasm or)	amphetamine
Vomiting	hematemesis	corrosive (coffee ground) or caustic (bloody brownish)

		poisoning, heavy metal poisoning (obviously red)
	brownish	bromide, iodine, potassium permanganate
	turquoise	copper salts
Hepatic failure	direct hepatotoxicity	amanita phalloides, arsenic poisoning, copper poisoning, ethanol, phenol, halothane, nitrosamine, phosphor, thallium, valproic acid
	hepatotoxic metabolite	paracetamol
	hepatic vein thrombosis	comfrey alkaloids

Electrolyte and Acid Base Balance:

Osmolality normal value: 290mOsm/L 2[Na]+2[K]+[Urea]+ [Glucose]	Elevated osmol gap: $Osmolality_{measured}$- $Osmolality_{calculated}$	acetone, ethanol, ethyl ether, glycols, isopropyl alcohol, mannitol, methanol
Acid Base Disturbances	Metabolic alkalosis	diuretics, antacids, penicillin
	Respiratory alkalosis	salicylate, sympathomimetic drugs, drug induced hepatic respiratory or cardiac failure
	Metabolic acidosis with widened anion gap	formaldehyde, methanol, ethylene glycol, acetaminophen, beta adrenergic drugs, CO, cyanide, HS, iron, INH, salicylate, theophylline, ibuprofen, valproic acid, other acids
	Metabolic acidosis with narrowed anion gap	bromide or nitrate (elevated Cl level) lithium, Ca, Mg (decreased Na level)
	Respiratory acidosis	opioids, sedatives, barbiturate, bacterial toxins: botulin, tetanus
Electrolyte disturbances	Hyperglycaemia	beta adrenergic drugs, caffeine, corticosteroids, diazoxide, glucagon, theophylline, thiazide diuretics
	Hypoglycaemia	insulin, oral antidiabetics, salicylate, valproic acid, ethanol, propranolol
	Hyperkalaemia	alpha adrenergic drugs, ACEi, beta blockers, digitalis, fluoride, lithium, potassium

	Hypokalaemia	barium, beta adrenergic drugs, caffeine, diuretics, epinephrine, theophylline, organic solvents
	Hypernatraemia	lactulose, lithium, mannitol, valproic acid
	Hyponatraemia	beer intoxication, diuretics, amitriptyline, phenothiazine, oxytocin
Renal failure consequent metabolic acidosis and hyperkalaemia	Direct nephrotoxicity	amanita phalloides, ibuprofen, paracetamol, bromide, ethylene glycol, heavy metals
	Haemolysis	arsenic poisoning, naphthalene, ethylene glycol
	Rhabdomyolysis	amphetamine, cocaine, PCP, strychnine

Other signs

Body temperature	Hyperthermia	amphetamine, methamphetamine, cocaine, serotonin, salicylate, lithium, TCA, antihistamines
	Hypothermia	ethanol, barbiturate, TCA, hypoglycaemic drugs, opioids, phenothiazine, colchicine
Odours	acetone-like	acetone, isopropyl alcohol
	pear-like	paraldehyde, chloral hydrate
	almond	cyanide
	garlic	arsenic poisoning, tellurium, organophosphate
	addled egg	sulphides
	carrot-like	cow bane/water hemlock
Neurology	Cerebellar signs	ethanol, phenytoin, carbamazepine
	Extrapyramidal signs	phenothiazine, haloperidol, metoclopramide
	Seizures	TCA, theophylline, antihistamine, anticonvulsant drugs, INH, phenothiazine

Investigations:

- Physical examination – ABCDE approach
- ECG, SpO2, Blood glucose and Arterial Blood gas
- Monitor Urine output, cardiac signs

- Endoscopy
- Special toxicological laboratories has serious limitations: slow, expensive, has no negative predictive value, the result may altered by other medications or environmental effects. Special laboratory tests exist only for 40-100 types of poisoning therefore the other millions of possible poisoning agent cannot be detected with laboratory tests.

2. Specific Poisonings

A. Toxic Gases

B. Drug Poisoning

C. Industrial Solvent Poisoning

D. Corrosive and Caustic Poisoning

E. Pesticide Poisoning

F. Illicit Drug Poisonings

G. Food Poisoning

H. Fungal Poisoning

A. Toxic Gases

Inhalation of toxic gases causes hypoxic state via different mechanisms:

- Oxygen is replaced with the toxic gas in the inhaled air: CO_2, Methane
- Oxygen is prevented to bind to haemoglobin: CO
- Inhibition of mitochondrial cytochrome oxidase enzyme system: cyanide
- Toxic pulmonary oedema: Chloride, Phosphoric and nitric gases

Inhalation of toxic gases in high concentration may cause sudden reflexogenic respiratory arrest.

Certain toxic gases may cause as sever poisoning inhaled continuously in small quantity as they would be inhaled once in high doses. But some gases in small concentration has no poisonous effect, and these gases does not accumulate either (CO).

The first and most important rule in the therapy of toxic gases to stop further exposure to the toxic agent.

NB: Personal safety is first! Special protective equipment has to be worn for rescuing the patient!

Name and Characteristics of the Gas	Symptoms of Poisoning	Therapy
Ammonia (NH_3) Industrial gas used in fridges. Has a pungent odour. Solved in saliva or tear ammonia causes liquefactive tissue necrosis.	lacrimation, running nose and salivation, dyspnoea, coughing, bloody spit, pulmonary oedema, glottis and bronchial spasm, suffocation	• Transport in lying position, do not let the patient to speak! • Give Oxygen • Prevent coughing • Give steroids (40-80mg methylprednisolone) • Give Lidocaine drops to the eyes, and Lidocaine spray to the mouth • Wash the skin gently with water
Arsenic Hydride (AsH_3) Produced during metal work Very poisonous: 40-50ml is lethal. Has a garlic-like odour. Provoke symptoms with 2-6 hrs latency.	dizziness, headache, shivering, fatigue, vomiting, *8-12 hrs after the exposure: bronze skin* pulmonary oedema, oliguria, anuria, renal failure	• High dose fluid therapy (crystalloid) • Give $NaHCO_3$ 1mmol/kg IV
Cyanide (HCN) *gas form:* pesticide, smoke produced by organic materials burning *orally:* almond, galvanisation Inhibits the mitochondrial cytochrome oxidase.	flushed face, vertigo, headache, paralysis, lacrimation, angina, dyspnoea, vomiting, convulsion, unconsciousness, death (vividly red cadaveric lividity)	• Endotracheal intubation, FiO_2 100% • Gastric lavage • Give $NaHCO_3$ 1mmol/kg IV • 20-40ml Cobalt-EDTA 1,5% • 40% glucose • Use nitro-glycerine • 3-10ml methylene blue 1% IV • 20-40ml Sodium-Thiosulfate 10% IV • High dose Vitamin B_{12}
Phosphine (PH_3) Produced during phosphor production. Has a garlic-like odour. Used for pesticide, rodenticide, steeping	1st Phase: coughing, dyspnoea, diarrhoea, unconsciousness, convulsion, shock, pulmonary oedema, apnoea, death 2nd Phase (1-2days after survival of 1st Phase): petechial mucosal bleeding, icterus, anuria, dysproteinaemia, hyperglycaemia,	• Endotracheal intubation, mechanical ventilation • Give diazepam 10-20 mg for convulsions • Give steroids, and diuretics for pulmonary oedema.

	haemorrhagic shock, death	
Phosgene ($COCl_2$) Chemical industry, and medical industry. 1st WW. Freon poured on hot surface.	Inhaled in high concentration is lethal in 1-2minutes. Inhaled in low concentration: coughing, angina, lacrimation, colic abdominal pain, bronchitis 5-10hrs later toxic pulmonary oedema!	• Transport lying at 30 degree head-up-position. Do not let the patient to speak! • Gently wash the skin with water, remove all the clothes. • Endotracheal intubation, IPPV • Prevent coughing (Tramadol 50-100mg IV) • Prevent pulmonary oedema steroids • If Pulmonary Oedema occurs give Furosemide, nitrate, steroid, elevate the PEEP
Sulphur Dioxide (SO_2) Produced in rubber industry, cold stores, heavy metal industry Has a choky odour. Forms sulphuric acid solved in saliva.	glottis spasm, dyspnoea, cyanosis, running nose and lacrimation, angina, pulmonary oedema in high concentration: lethal	• Transport lying at 30 degree head-up-position. Do not let the patient to speak! • ET, ventilation • Prevent coughing (Tramadol 50-100mg IV) • Prevent pulmonary oedema steroids • If Pulmonary Oedema occurs give Furosemide, nitrate, steroid, elevate the PEEP • Lidocaine drops to eyes • Lidocaine spray to mouth • wash the skin gently with water
Sulphur Hydride (H_2S) Produced during organic protein catabolism -> sews, mines, caves, defecator pools Has an odour like addled egg. Forms Na_2S solved in saliva.	lacrimation, running nose, diarrhoea, dysuria, coughing, headache, vertigo, unconsciousness, death	• Give O2 or ET, ventilation • Give nitrate • Perform nasal, ocular and pharyngeal lavage!
Chloride (Cl_2) Chemical industry, disinfectant. Forms hydric chloride solved in saliva. Has a pungent odour.	“chloride-coughing”, dyspnoea, cyanosis, lacrimation, running nose, angina, pulmonary oedema, in high concentration it is lethal due to glottis spasm	• Transport lying at 30 degree head-up-position. Do not let the patient to speak! • Give O2 or ET ventilation • Prevent coughing (Tramadol 50-100mg IV) • Prevent pulmonary oedema steroids • If Pulmonary Oedema occurs give Furosemide, nitrate, steroid, elevate the PEEP • Lidocaine drops to eyes • Lidocaine spray to mouth

		• Wash the skin gently with water
Nitric Gas (NO, NO_2) explosions, chemical industry, celluloid burning, welding, silo Forms Nitric acid solved in saliva. **Shock Type:** direct acid effect -> suffocation, convulsion, death **Irreversible Type**: 2-3hrs agony in toxic pulm. oedema **Reversible Type:** (short exposure) dyspnoea, cyanosis, vomiting, unconsciousness, then recovery	In low concentration has no signs for 2-10 hrs, then coughing, yellow or brown spit occurs, dyspnoea, pulmonary oedema, tachycardia In higher concentration: + angina, vomiting, lacrimation	• Transport lying at 30 degree head-up-position. Do not let the patient to speak! • Give O2 or ET ventilation • Prevent coughing (Tramadol 50-100mg IV) • Prevent pulmonary oedema steroids • If Pulmonary Oedema occurs give Furosemide, nitrate, steroid, elevate the PEEP • Lidocaine drops to eyes • Lidocaine spray to mouth • Wash the skin gently with water • Remove all the clothes
Carbon dioxide (CO_2) Has no odour. Fill the space from below (heavier than air) wine cellar, defecator pool, silo	4-5% hyperventilation 20% blockage of the respiratory centre >50% 1 breath is lethal prompt death	• ET, ventilation, high flow oxygen • Hyperventilation • Give 1mmol/kg $NaHCO_3$
Carbon monoxide (CO) Has no odour. Fills the space from above (lighter than air) Penetrates through walls, ground and ceiling Produced during imperfect burnings Poisonous in every concentration! **Pulse oximeter shows normal SpO2!!!!!!**	1st Phase: drowsiness, headache, vertigo, nausea and vomiting, altered mental state, cherry red skin, fatigue, paralysis, AMI 2nd Phase: trismus, unconsciousness, convulsions (GM-like), arrhythmias, aspiration, hypotension 3rd Phase paralysis, apnoea, death 4th Phase coma 1-2days then recovery	• ET ventilation, Fio2: 100% for 20-30 minutes • For trismus give diazepam 10-20 mg IV • Give 1mmol/kg $NaHCO_3$ • 50% of CO eliminates in an hour on fresh air **PREVENTION WITH CO-DETECTOR!!!**

CO level can be monitored in arterial Blood Gas with COHb value:

- 10% COHb: no symptoms
- 20% COHb: headache, tachypnoea
- 30% COHb: + agitation, fatigue, nausea, palpitation

- 50% COHb: drowsiness, stupor, collapse
- 70% COHb: coma, dilated pupils, no light reflex, death
- 80% COHb: soon death
- >80% COHb: prompt death

B. Drug Poisoning:

Benzodiazepines:

Potency\Half-life	Ultrashort (<5h)	Short (5-15h)	Immediate (15-40h)	Long (>40h)
low		Gerdorm	Eunoctin	
medium				Seduxen
high	Dormicum	Xanax		Rivotril

Pharmacokinetics:

BZDs have wide therapeutic index, slow onset (orally taken), they are absorbed in duodenum and highly bound to proteins 80-90% (except Xanax – 70%). Benzodiazepines are metabolised in the liver (oxidation, glucuronide conjugation), and BZDs have active metabolites with long half-life. The conjugated metabolites are eliminated by kidneys.

Signs of BZD poisoning:

- Neurologic signs: nystagmus, dysarthria, stupor, delirium, coma, anterograde amnesia
- Cardiac signs (rare): slight hypotension and bradycardia, mild respiratory depression
- Neuromuscular signs (rare): ataxia, hypotonic muscles, slow movement
- Gastrointestinal signs (rare): nausea and vomiting, diarrhoea, incontinency

Therapy: -mainly supportive

- Decontamination with activated carbon
- Antidote: Flumazenil (Anaxate) 0,5mg IV bolus, onset of action 1-2 minutes, then 0,2-1mg/h
    - Contraindications of Flumazenil therapy: epilepsy, known BZD allergy, regularly taken BZD, after anaesthesia, if there is neuromuscular blockade, multidrug poisoning

Barbiturates:

Half-life:

- ultrashort: hexobarbital (Novopan)
- short: cyclobarbital (Hypnoval-Calcium)
- intermediate: amobarbital (Dorlotyn), butobarbital (Etovaletta)
- long: phenobarbital (Sertan, Sevenal, Sevenaletta, Mysoline)

Barbiturates may be combined with other drugs.

Pharmacokinetics:

Barbiturates have narrow therapeutic index. They are absorbed rapidly from the stomach, but metabolised slowly in liver (microsomal oxidation, glucuronidation, glycosylation), and they have inactive metabolites (eliminated by the kidneys). Phenobarbital is reabsorbed by enterohepatic cycle. Protein bound form is varying 20-86%.

Signs of Barbiturate Poisoning:

- cold skin, cyanosis, hypothermia, tachycardia, hypotension, hypovolaemia, bullous lesions, narrowed pupils, nystagmus

Therapy:

- Endotracheal intubation if GCS<9
- Manage hypotension, monitor the patient's vital signs
- Gastric lavage
- Activated Carbon
- Urine alkalization
- Forced diuresis

NB: Prevent and treat decubiti!

Beta blockers:

Beta blocker are competitive antagonist of catecholamines on beta adrenergic receptors, having negative inotropic, chronotropic and bathmotropic effect. They vary in beta-2 receptor selectivity, lipofilicity and intrinsic sympathomimetic activity. Consequently all their effects and side-effects are different.

Signs of Beta Blocker Poisoning

- Cardiovascular: hypotension and hypoperfusion sy. bradycardia, AV-block, congestive heart failure, cardiogenic shock

- Respiratory: the non-selective beta blockers may cause bronchospasm, dyspnoea, respiratory failure
- Neurologic signs: altered mental state, respiratory depression, seizures, **hypoglycaemia**

Therapy:

- Within 1-2 hours of exposure: decontamination
    - gastric and intestinal lavage, activated carbon
- Supportive therapy:
    - Fluid therapy (crystalloids)
    - Give glucose, monitor BG
    - In severe poisonings inotropic drugs may indicated
    - Endotracheal intubation if GCS<9
    - Specific therapy: atropine and/or glucagon
    - Glucagon therapy: 5-10mg IV bolus, 1-2mg/kg infusion pump.
    - Temporary Pacemaker Insertion

Digitalis

Digitalis has narrow therapeutic index: Digoxin (0.5-2.2 ng/mL), Digitoxin (10-15ng/mL). It affects the Na-K pump (has positive inotropic effect), vegetative CNS, AV-node and certain part of the cerebral cortex.

- Plants contain digitalis: yew tree, fox-glove, lily of the valley and oleander
- Animals produces digitalis: certain toads (bufotoxins)

Pharmacokinetics: digitalis is absorbed in the intestine, bound to skeletal and cardiac muscles (in a variable ratio). Recycled by enterohepatic cycle (activated carbon).

Mechanism of poisoning: 1st phase: therapeutic effects increased, 2nd phase noradrenalin release and arrhythmias. Digitalis poisoning may cause any type of arrhythmia depend on the quantity of myocardium bound digitalis and the myocardial sensitivity to digitalis.

Types and Severity of Digitalis Poisoning:

- Acute Poisoning: high serum Digoxin level, hyperkalaemia, vomiting and bradycardia

- Chronic Poisoning: myocardium is less sensitive to the digitalis, hypokalaemia, elevated or decreased serum digitalis level.
- In patients without heart disease: blocks, ventricular extra systoles, idionodal rhythm.
- In patients with heart disease: ventricular arrhythmias, accelerated rhythm, ventricular fibrillation
- COPD patients: atrial autonomy

Signs of Digitalis Poisoning:

- Nausea, vomiting, altered mental state, convulsion, delirium, loss of appetite, abdominal pain
- ECG signs: down-sloping ST segment depression, any type of arrhythmias (generally brady-arrhythmias + ventricular bigeminy, 2nd degree AV-block, slow AF, bidirectional VT, atrial tachycardia with AV-nodal block)

Therapy: goal of the therapy: prevent the digitalis bind to the myocytes and decrease myocardial sensitivity to digitalis.

- Decontamination: activated carbon (absorb intestinal digitalis, prevent reabsorption too).
- Supportive therapy: Mg, antiarrhythmic therapy.
- Antidote: Digoxin binding antigen fragments (Fab) Digibind
    - Indication: extreme hyperkalaemia, therapy resistant arrhythmias.
    - Extremely expensive

Ca-channel Blockers:

Ca is responsible for the activity of myocardium, vessels, sinus node, neuroendocrine cells and smooth muscle. In the SA and AV node the conduction of the electrical impulses is fully Ca-dependent. Therefor Ca-channel blockers cause SA inhibition, and they have negative dromotropic and inotropic effect.

Types of Ca-channel blockers:

- **Dihydropyridines:** amlodipine, felodipine, isradipine, nicardipine, nifedipine, nimodipine (used in SAH to prevent or treat vasospasm!), nisoldipine, nitredipine,

lacidipine, nilvadipine, manidipine, barnidipine, lercanidipinidine, clinidipine, benidipine. (main effect on the peripheral vessels -> dilatation, hypotension)

- **Phenylalkylamins:** verapamil, gallopamil (main effect on the AV-node -> bradyarrhythmia)
- **Benzothiazepine:** diltiazem (transient type between the dihydropyridines and phenylalkylamins)

Pharmacokinetics: absorbed in the GI tract, high first pass effect, 80% bound to proteins. Ca-channel blockers metabolised in the liver.

- *Signs of Ca-Channel Blockers:*
- Circulatory: bradyarrhythmias, AV-block, AV-dissociation, ECG: ST depression, U wave, inverse T wave. Myocardial depression: cardiogenic shock, hypotension, peripheral vasodilatation
- GI: nausea and vomiting, passage disturbances, ileus
- Others: hyperglycaemia, metabolic acidosis (elevated lactate), seizures, drowsiness, coma

Therapy:

- Supportive therapy and monitoring the patient's vital signs
- Decontamination: gastric and intestinal lavage, activated carbon therapy
- Fluid Therapy, keeping up Fluid Balance
- Plasmapheresis
- Calcium-chloride/Calcium-gluconate: 1-2g IV bolus repeated every 5-10 minutes (max 10g)
    - If the symptoms ease with calcium therapy, give Ca 20-50mg/kg/hr.
- Glucagon: if myocardial depression is present 2-10mg IV bolus, then 2-4mg/h
- Inotropic drugs (as second-line-therapy) e.g. dopamine 50 µg/h or noradrenalin.
- Amirone: Phosphodiesterase III inhibitor -> elevated cAMP production 1mg/kg IV bolus, then 6 µg/kg/min.

Antipsychotics and antidepressants:

Antipsychotics are used in treatment of: schizophrenia, schizophrenia-like disorders, psychoorganic syndrome, affective disorders with psychosis and behaviour disorders.
Antidepressants are used in treatment of (tri and tetracyclic antidepressants): major depression, bipolar affective disorder (in depression phase), dysthymia, organic depression, anxiety, obsessive-compulsive disorder (OCD), eating disorders and pain syndromes.

i. Antipsychotics:

- Typical antipsychotics:
    - Phenothiazine type: chlorpromazine, fluphenazine, thioridazine, promethazine
    - Thioxanthene-type: chlorprothixene
    - Butyrophenone: haloperidol
- Atypical antipsychotics: clozapine, risperidone, olanzapine

Antipsychotics' mechanism of action:
Limbic system and basal ganglions by dopamine receptor inhibition. Typical antipsychotics inhibits D2R, and slightly D1R too. Atypical antipsychotic has other target sites clozapine inhibits D5R, but risperidone inhibits 5-HT2R. Antipsychotics affect other receptors too (histamine-R, alpha-R …).
Antidepressant overdose – often as suicidal attempt

- Rare fatal outcome: might be lethal if arrhythmias, pneumonia, sepsis, MOF occur
- Cardiovascular: usually seen in typical antidepressant poisoning.
    - ECG: tachyarrhythmia, broad QRS, prolonged QT and PR, torsade de pointes and VT
- Anticholinergic effect: slow bowel movement, ileus, urine retention
- Altered mental state: agitation, drowsiness, swallowing disorders

Therapy:

- Supportive: treat electrolyte disturbances and arrhythmias (avoid 1a type antiarrhythmic use Lidocaine), maintain normal blood pressure, give fluid therapy.
- ET intubation if GCS<9

- Give BZD in seizures
- Alkalization with $NaHCO_3$

Special case of Antipsychotic Poisoning - Neuroleptic malign sy

- Sudden dopamine hyperactivity (typical in the first few weeks of antipsychotic therapy).
- hyperthermia, altered mental state, muscle rigidity, vegetative disorders, shock circulatory collapse, acute renal failure, DIC, pulmonary embolism and aspiration pneumonia
- Treatment: ET intubation, immediate cooling with icy pack, fluid therapy
- Bromocriptine, dantrolene, L-dopa, BZDs

ii. Tri and Tetracyclic Antidepressants

Mechanism of action: serotonin and noradrenalin reuptake inhibitors, anticholinergic, antihistamine effect, inhibition of GABA-R and alpha receptors. Fast Na-channel and K-channel inhibitors. Consequently Tri and Tetracyclic Antidepressants induce arrhythmias due to prolonged QRS, QT and PR.

Tri and Tetracyclic Antidepressants have high toxicity, narrow therapeutic index, rapid absorption and active metabolites produced in liver. They are reabsorbed by enterohepatic cycle. >90% bound to proteins. They have long half-life (8-92hrs). Renal elimination is low 3-10%.

Types: clomipramine, maprotiline, dibenzepin and amitriptyline.

Signs of Tri and Tetracyclic Antidepressant Poisoning:

- ECG:
    - QRS>100msec. in lead II
    - Right axis deviation
    - aVR: terminal R wave > 3mm and R/S ratio > 0,7
- ADORA (Antidepressant Overdose Risk Assessment) high risk if 1 or more criteria occue in the first 6 hrs.
    - QRS > 100 msec.

- arrhythmia or conductive disorders
- seizure
- altered mental state GCS < 14
- Resp.Rate< 8/min
- Hypotension $NIBP_{sys}$< 90 mmHg

Therapy:

- Supportive therapy (fluid therapy, ET intubation, ventilation)
- Decontamination: if >10mg/kg TCApoisoning, within 6 hrs.
- Activated carbon therapy
- TCA antibodies
- Ice pack

Arrhythmias:

- SVPT: alkalization, synchronic cardioversion DO NOT USE 1a, 1c or C-antagonist
- VT: alkalization, lidocaine 1mg/kg IV bolus, then, 2-4mg/min, cardioversion
- Torsade de Pointes: Mg, isoprenaline, pacemaker
- VF: defibrillation, alkalization, adrenalin, lidocaine, Beta blockers
- AV-block: alkalization, isoprenaline 0.5-5 μg/min, pacemaker
- Cardiac arrest: CPR at least 1hr, alkalization

Alkalization: NaHCO3 1-2mEQ/kg IV bolus, then with pump, pH target: 7.45-7.7!

Paracetamol

Mechanism of Action: weak COX inhibitor, rapid absorption, reaches peak concentration in 30-120 min. Short half-life: 1-3hr. 95% is metabolised in liver (60% glucuronidation, 30% sulfatation), 5% metabolized to 3-hydroxi-paracetamol and N-acetyl-p-benzoquinone imine (NAPQUI) by hepatic cytochrome P450.

Mechanism of Poisoning: Paracetamol overdose causes lack of liver enzymes of glucuronidation and sulfatation. Consequently all the paracetamol metabolised by cytochrome P450 producing extreme amount of NAPQUI. NAPQUI is a reactive substance, binds to

hepatic proteins' SH groups and makes the proteins inactive, therefore hepatic cell necrosis is induced.

Paracetamol also damages the kidney, but mechanism is unknown.

- Therapeutic dosage: 10-15 mg/kg max. 3-4g
- Toxic dosage: paediatric > 150mg/kg, adult > 250mg/kg

Signs of Paracetamol Poisoning:

1st Phase (30min-24h): non-specific symptoms: malaise, fatigue, loss of appetite, nausea and vomiting or sweating. May be asymptomatic.

2nd Phase (24-72h): elevating serum hepatic necroenzymes and decreasing renal function, pain in the right upper quadrant.

3rd Phase (72-96h): fulminant hepatic and renal failure may occur.

4th Phase: (4-14days): recovery

Therapy:

- GI decontamination: activated carbon 1g/kg in the first 4 hrs
- Gastric and intestinal lavage is indicated in retard paracetamol poisoning.
- Antidote: N-acetyl cysteine (Fluimucil Antidote 5g/25ml)
    - 150mg/kg IV in 200ml Rindex-5 in 15minutes
    - 50mg/kg IV 500ml Rindex-5 in 4hr
    - 100mg/kg IV 1000ml Rindex-5 in 16hr
- Elimination: haemodialysis, haemoperfusion
- Supportive therapy: antiemetic drugs, fluid therapy.

C. Industrial Solvent Poisoning

Gasoline poisoning:

Gasoline is a mixture of hexane, heptane and octane hydrocarbons. Inhaled or ingested gasoline has narcotic effect (lipophilic). Gasoline poisoning may be an accident (inhaled, ingested), but sometimes it is used to commit suicide ingested or IV. Gasoline is eliminated from body via airways, and independently from the site of exposure it provokes pneumonia.

Signs of Gasoline Poisoning:

Ingested gasoline causes fulminant vomiting – severe poisoning is rare.

Inhaled gasoline: headache, vertigo, drowsiness, bronchitis, conjunctivitis and mucosal irritation

Severe poisoning: deep coma, seizures, apnoea, VF, aspiration, haemorrhagic pneumonia.

IV gasoline: aching chest pain, coughing, cyanosis, pallor, sweating and sever poisoning.

Therapy:

- Stop further exposure, (evacuate the patient from closed rooms if further inhalation may occur)
- ET, ventilation
- 200ml liquid paraffin may stop further absorption of ingested gasoline.
- Gastric lavage (after paraffin has been administered)
- DO NOT INDUCE EMESIS!

Benzol Poisoning:

Benzol is used as an industrial solvent and paint thinner. Illicit use of benzol is "smoking" it. Benzol stops cell division.

Signs of Benzol Poisoning:

Burning pain of oesophagus and stomach, nausea and vomiting, enuresis, dyspnoea, fatigue, drowsiness, unconsciousness and apnoea

Therapy – the same as gasoline.

Ethanol

Ethanol poisoning is the most common form of intoxication, and many underage patient suffers from acute ethanol poisoning. Ethanol is metabolised in the liver.

Signs of Ethanol Poisoning:

Decreasing level of consciousness, stupor, somnolence, coma, bradypnoe, cold, sweaty skin, vomiting, aspiration and provoked seizures.

Levels of Ethanol Poisoning

~0,3‰		euphoria, calm, dissolved inhibitions, logorrhoea, decreased ability to concentrate on
0,8-1,2‰	influenced	Impairment of coordination and motor functions.
1,2-1,6‰	mild drunkenness	Worsening motoric functions,, unscrupulousness, slurred speech, loss of sense of danger, prolonged reaction time
1,6-2,0‰	moderate drunkenness	Symptoms mentioned above getting worst
~2,0‰	severe drunkenness	Drowsiness, depression, dysphoria with nausea.
2,0-3,0‰	Ethanol poisoning	Lost consciousness
3,0-4,0‰	drowsiness, stupor	
>4,0‰	coma	

Therapy:

- Call for ambulance, position the patient prevent aspiration. Keep the patient warm.
- Fluid therapy, monitor the patient's vital signs.
- Give folate and B12 Vitamin in suspected Wernicke encephalopathy
- ET intubation, and ventilation if GCS < 9.
- Warming.
- Gastric lavage may be performed.

Therapy of ethanol withdrawal sy (agitation, altered mental state, hallucination, hypotension, dehydration, electrolyte disturbances, hyperthermia)

Sedation, fluid therapy, antifebriles, BZD in seizures.

Methanol

Lethal dose: 10-100ml

Methanol causes narcosis, and its metabolite formaldehyde causes acidosis and retinal injury.

Symptoms of Methanol Poisoning

As Mild Drunkenness + abdominal pain, dyspnoea, headache, diarrhoea, lethargy. In severe cases: blindness, metabolic acidosis, delirium, seizures.

Therapy:

Give ethanol! (PO: 2-3spirits 3-4 hrs.)

IV ethanol therapy: give 10% ethanol

	Loading Dose	Maintenance Dose	Dose during dialysis
Non-drinker	2ml/kg/20mins	1ml/kg	4ml/kg
Occasional Drinker	2.5ml/kg/20mins	1.25ml/kg	5ml/kg
Regular Drinker	3ml/kg/20mins	1.5ml/kg	6ml/kg

Fomepizole: 4-methylpyrazole is a competitive inhibitor of alcohol-dehydrogenase.

- Under the age of 5 giving ethanol is absolute contraindicated.
- 15mg/kg in 100ml Saline under 30minutes, then 10mg/kg every 12hrs 4x.

Ethylene-glycol - antifreeze

Signs of ethylene-glycol poisoning:

12-36h after exposure: slurred speech, seizures, cerebral oedema, coma, metabolic acidosis, tachypnoea, Kussmaul breathing, nausea, vomiting

> 36h after exposure: cyanosis, pulmonary oedema, death. In urine oxalate can be detected.

>3days in survivors: acute renal failure, haematuria, crytalluria

Therapy: Ethanol therapy for 5 days!

- PO: 250-300ml spirits
- IV: 1.39 ml/kg/h 10% ethanol (regular drinkers: 1.95 ml/kg/h, non-drinkers: 0.83 ml/kg/h)
- Haemodialysis
- Thiamine: 100mg/day IV
- Pyridoxine: 100mg/day
- Alternative to ethanol: Fomepizole!

D. Corrosive and Caustic Poisoning

Acid poisoning:

Signs of acid poisoning:

- Coagulation necrosis of the skin and mucosa.
- The colour of the coagulum is characteristic of the type of acid. (H_2SO_4 – black, HCL – white, HNO_3 – yellow)
- Painful lesions of oral cavity, pharynx, oesophagus and stomach. Perforation of stomach may occur and septic peritonitis present as a consequence.

- Aspired acid may cause glottis spasm and pneumonitis
- Bleeding.
- Acidaemia, renal impairment.
- Long term effect: strictures.

Therapy:

- Physical examination: ABCDE approach, examine the eyes and the mucosa accurately.
- Decontamination, then wash the skin gently with water. (If the acid got into the eyes wash the eyes with clear water at least 20-30 min).
- If the patient has ingested the acid DO NOT INDUCE EMESIS! DO NOT PERFORM GASTRIC LAVAGE!
- Give water to drink.
- No alkali should be administered (the reaction will be exothermic and will cause further injuries).
- Supportive therapy: Fluid therapy, Analgesia!!! Early ET intubation and ventilation in severe cases.

Alkali poisoning

Signs of alkali poisoning:

- Liquefactive necrosis of skin and mucosa (deeper than coagulation type)
- Extremely painful lesion, collapse and shock can be seen.
- Perforation of stomach is more probable than in acid poisoning.
- Bleeding.
- Alkalosis.
- Long term complications: strictures.

Therapy: the same as acid poisoning.

Endoscopy: Perform endoscopy after stabilization of the patient's condition is very important to evaluate the damage.

What NOT to do: do not perform gastric lavage, do not give neutralizing agent, do not give milk, do not administer activated carbon. Do not induce emesis.

E. Pesticide Poisoning

Barium poisoning:
Occurrence: pesticide, rodenticide, X-ray contrast agent, sparkler
Mechanism of action: K-channel blocker, digitalis-like effect.
Signs of poisoning: salivation, vomiting, diarrhoea, hypertension, arrhythmias, VF, paralysis, respiratory paralysis.
Therapy:

- Give Na_2SO_4 PO, drotaverine, atropine
- KCl administration, monitor the patient!

Dicumarol poisoning:
Occurrence: Syncumar, Marfarin, Warfarin, rodenticides
Mechanism of action: inhibits the prothrombin formation.
Signs of poisoning: hematemesis, haematochezia, bleeding, suffusion, petechial lesions, hypotension, haemorrhagic shock.
Therapy:

- Gastric lavage
- 20-40mg Vitamin K + 500-1000ml Vitamin C
- Transfusion: blood, plasma and thrombocytes too
- Management of haemorrhagic shock

Dinitro-ortho-cresol and dinitrophenol poisoning

Occurrence: casual pesticides
Mechanism of action: methemoglobinemia
Signs of poisoning: headache, vomiting, colic abdominal pain, diarrhoea, hyperpyrexia, sweating, icterus, anxiety, tachycardia, dyspnoea, pulmonary oedema, metabolic acidosis, coma, convulsion, death (rapid onset of rigor).
Therapy: 200ml liquid paraffin,
Gastric lavage

- Ice packs

- Fluid therapy
- BZD if seizures occur.
- Methylene blue
- DO NOT ADMINISTER milk, ethanol and castor oil!!!

Alkyl-phosphate Poisoning (organophosphates, cholinesterase inhibitors)

Alkyl-phosphates contain sulphur	Phosphonate and Phosphonate esters	Carbamates
ANTHIO	AZODRIN	CHINOFUR
BASUDIN	BIO-STRIP	CINEB
Bi 58 EC	DIMEKRON	DITHANE
BUVATOX	UNIFOSZ	FURADAN
CIDIAL		GARVOX
NEVIFOS		

Absorption: dermal, mucosal, gastrointestinal and pulmonary absorption

Mechanism of action: Inhibition of cholinesterase enzyme (irreversibly - organophosphates, reversibly - carbamates)

Signs of poisoning:

Muscarinic symptoms: chest pain, dyspnoea, bronchoconstriction, increased bronchial mucus production, salivation, lacrimation, running nose, coughing, pulmonary oedema, pneumonitis, bradycardia, hypotension, AF, VT and myosis

Nicotinic symptoms: fasciculation, fatigue, sympathetic activity

CNS symptoms: agitation, seizures, coma

Latent poisoning: serum Cholinesterase activity 50% after 6hrs (good prognosis, usually asymptomatic)

Mild poisoning: serum Cholinesterase activity 20-50%, the patient is able to walk, but fatigue, headache, nausea, vomiting, and abdominal spasms are present.

Moderate poisoning: serum Cholinesterase activity 10-20%, the patient cannot walk, extreme fatigue, myoclonuses, myosis.

Severe poisoning: serum Cholinesterase activity< 10%, severe myosis, fasciculation, flaccid paralysis, depressed breathing, cyanosis.

Therapy:

- External decontamination, wear personal protecting equipment! Gastric lavage. Activated carbon.
- ET intubation, ventilation

- Antidote: Atropine 0.5-1mg IV, can be repeated antagonise muscarinic and CNS symptoms.
- Pralidoxime-chloride (Protopam) cholinesterase reactivator 2g loading dose, then 200-500mg/h for 18-24h.
- Haemoperfusion.

Nicotine Poisoning:
Occurrence: forensic cases, iatrogenic, industrial, pesticide poisoning or smoking
Lethal Dose: 40-60mg (from a cigarette 2-8mg absorbed, from a cigar 10-40mg)
Symptoms occur immediately:

- Headache, vomiting, shivering, fatigue, diarrhoea, sweating, hypertension, weak pulse, narrow pupils, loss of consciousness, seizures, shock, death.

Therapy:

- Supportive therapy: decontamination, gastric lavage, 5-10 ml Akineton (biperiden) IV, BZDs, ET intubation, ventilation.

Copper sulphate poisoning
Occurrence: pesticide, galvanization, chemical fertilizer
Mechanism of action: like iron poisoning
Signs of poisoning:

- Torques mucosal lesions in the mouth, hematemesis, colic abdominal pain, shock, renal injury, drowsiness, fatigue.

Therapy:

- PO administered egg white the albumin of the egg white forms copper albuminate.
- Gastric lavage
- Fluid therapy
- 50-100mg Dolargan IV
- 1 spoon of Potassium-ferro-cyanide 1% every 5 minute!
- 2-3 amp. Dicaptol
- $CaNa_2$-EDTA 10-20ml IV
- Methylene Blue

- AVOID castor oil, milk, fatty foods!

F. Illicit drug poisoning

Classification of illicit drugs:

Major analgesics	Stimulants	Hallucinogens	Solvents, Evaporating Drugs
opium, morphine, codeine, heroin, methadone, dextromorphan, buprenorphine, dextropropoxyphene	cocaine, amphetamine, methamphetamine, ephedrine, caffeine, nicotine, MDMA	LSD, hashish, cannabis, cannabinoids, mescaline, phencyclidine, ketamine, GHB, other plants	organic solvents, glues, lacquer

Opiate poisoning:

Signs of poisoning: respiratory depression, miosis, narrowed consciousness, clinical response to naloxone

Therapy: supportive therapy: ET intubation, ventilation, maintaining vital functions.

NB: 2-3h after heroin use pulmonary oedema, respiratory failure may occur!

Cocaine poisoning:

Signs of poisoning: general vasoconstriction leads to hypertension, tachycardia, hyperthermia, agitation, chest pain, stroke, mesenteric ischaemia

Therapy: cooling, BZDs

If rhabdomyolysis occur: urine alkalization, induced urination

Pulmonary oedema: diuretics, Morphine, nitrate, phentolamine, CPAP ventilation

Cocaine induced AMI: avoid the beta blockers, GIVE: Ca-channel blockers!

Amphetamine poisoning:

Signs of poisoning:

Sympathomimetic symptoms: tachycardia, arrhythmias, hypertension, hyperthermia, rhabdomyolysis, wide pupils.

CNS symptoms: anorexia, hallucinations, psychosis, seizures, stroke

Therapy: sedation, cooling, haemodialysis.

LSD:

Signs of poisoning: altered mental state, hallucinations, injuries

Therapy: sedation, supportive therapy.

Cannabinoids:

Signs of poisoning: tachycardia, agitation, hypertension

Therapy: sedation, beta-blockers

Organic solvents, glues:

Signs of poisoning: altered mental state, suffocation, unconsciousness, special odour, early dementia

Therapy: supportive.

G. Food Poisoning

Food poisoning is usually caused by bacterial infection (salmonella, shigella, streptococcus, staphylococcus, etc.) or bacterial toxins (botulin toxin).

Characteristic foods which may become source of poisoning: crèmes, ice creams, fish, mushroom, already opened conserve, raw meat, boiled pasta.

Common incubation period: 1-5hrs, Salmonella 8-12hrs.

Signs of poisoning:

- Abdominal pain type: epigastric spasm, nausea
- Gastritis type: epigastric spasm, nausea, vomiting
- Gastroenteritis type: epigastric spasm, nausea, vomiting, diarrhoea, fever, hypotension, headache, dehydration.
- Enteritis type: colic abdominal pain, diarrhoea, dehydration

Therapy: fluid therapy, astringents, calcium, antifebriles, Activated carbon, diet

Botulism:

Clostridium botulinum produces thermosensitive toxins: A, B, C, D, E, F and G

A-vegetables, B-meat, E-fish, F-wounds

Signs of Botulism: incubation time 6-48hrs, but sometimes it may be 10-20days!

- Neurologic signs: double vision, blurred vision, ptosis, wide pupils, loss of light reflexes
- No saliva production: dry mouth, and throat, hoarse speech, tongue paralysis
- Absence of bowel movements, no gastrointestinal secretion
- Urinary retention
- Skeletal muscle paralysis -> respiratory paralysis

Therapy:

- ET intubation, ventilation
- Laxatives (avoid laxatives with magnesium)
- Antitoxins: homolog monovalent or polyvalent antitoxins
- If the source was a wound: debridement

Fungal poisoning

Types of poisoning:

- Phalloides syndrome
- Orellanussyndrome
- Giromitrasyndrome
- Muscarinic syndrome
- Pantherinasyndrome
- Psylocibinesyndrome
- Coprinussyndrome
- Paxillussyndrome
- Gastrointestinal syndrome

Onset of symptoms:
Rapid onset: 1-2h
Slow onset: 5-24h
Rapid onset poisoning:

- Muscarinic poisoning causes parasympathetic activity: narrow pupils, sweating, salivation, lacrimation, running nose, nausea, vomiting, abdominal spasms, bradycardia,
- Muscaridine poisoningcauses atropine-like symptoms: hyperaemic, dry skin and mucosa, wide pupils, agitation, hallucination, tachycardia,
- Gastrointestinal poisoning: vomiting, diarrhoea, abdominal spasm, fatigue, headache

Slow onset poisoning (5-25h):

- Phalloides poisoning:

- 2 types of toxins:
    - amatoxin: toxic to liver and kidney
    - phallotoxin: gastroenteritis symptoms

1st Phase: nausea, excessive vomiting, diarrhoea, dehydration, fatigue, muscle spasms, hypotension

2nd Phase: increasing icterus, oliguria, anuria occur after a few asymptomatic days, then irreversible hepatic and renal failure present.

Therapy:

- Take samples from the foods
- Gastric and intestinal lavage!
- Excessive fluid replacement
- Antidotes: penicillin, acetyl cysteine
- Plasmapheresis
- Supportive therapy
- Liver transplantation

13. Analgesia, Anaesthesia and Sedation in the ED by Dr.Molnár Anna

50-60% of patients arrived to ED suffer from acute pain. We could differentiate three types of acute pain: somatic, visceral, and neuropathic. The diagnostic or therapeutic interventions may also cause pain, therefore analgesia, sedation and/or anaesthesia are required.

On one hand pain protects the body's integrity but on the other hand prolonged pain wastes too much energy. Early and adequate analgesia is a basic principal beside correct diagnosis and definitive therapy.

Definitions:

- **Pain** is an unpleasant feeling, perception and the subjective interpretation of the discomfort as a physiological response for tissue injury. The various conscious, unconscious and emotional responses to painful stimuli, depend on the actual physical and mental state.
- **Nociceptor** is a specific receptor for pain, stimulated by various kinds of tissue injury.
- **Hyperalgesia**is abnormally increased sensitivity for injuries, a nociceptive stimuli may causes more severe pain than it is expected.
- **Opioid:** any natural or synthetic narcotic that has morphine-like activities.
- **Opiate:** natural opioid
- **Procedural Sedation and Analgesia:** administration of sedatives or dissociative agents and analgesics to induce a blurredstate of the patient to tolerate painful procedures while cardiorespiratory function is maintained.
- **Anxiolysis/Minimal Sedation**: decreased reactivity and cognitive functions whileconsciousness is maintained.
- **Superficial/Conscious Sedation:**administration of sedatives with analgesics, while the patient can give adequate responses for verbal or tactile stimuli, but without stimuli the patient’s eyes are closed and has a minordifficulty of speaking. The airways are freeand spontaneous breathingand normal circulatory parameters are maintained.

- **Deep sedation:**the patient responds only to repeated or painful stimuli. Airway reflexes may be depressed and spontaneous ventilation may stop, but cardiovascular function is intact.

- **Dissociative sedation:**a dream-like state with total analgesia and amnesia. Airway reflexes, spontaneous breathing, and cardiovascular function are all maintained.

- **General Anaesthesia:** Hypnotic state with analgesia, amnesia, muscle relaxation. The patient does not response to any stimuli. For the muscle relaxation airway protection and assisted ventilation are required. Cardiovascular function may be affected too.

- **Local Anaesthesia** Local analgesic administration provides a reversible regional analgesia on the site of the planned intervention

Basic principles of analgesia

Believe the patient!

Identify pain

Specify characteristics of pain

Choose the most feasible treatment

Reassess pain

The perception of pain

- Nociceptive input:
    - nociceptors: peripheral receptors, which sense mechanical, thermal and multimodal stimuli
    - afferents: δ and C fibres
- Central processing:
    - Posterior spinal cord: pain modulation and integration
    - Supraspinal pain modulation and integration: the hypothalamus, the thalamus, the limbic system and the RAS give emotional and physiologic responses to pain.
    - Sensory cortex: The primary somatosensory area is located in the post central gyrus. It discriminates somatic pain, which can be located easily, but visceral pain causes a blurred sensation.

Acute pain:

- Acute pain is triggered by a nociceptive stimuli

- The severity of acute pain is proportional to the triggering stimulus
- Acute pain can be easily described and located by the patient
- Acute pain is gone with the disappearance of the triggering stimulus

Characteristics of acute pain:

Onset of pain

Provocation or palliation

Quality of the pain (sharp, dull, crushing, burning, tearing, in time pain may be intermittent or constant.)

Region and radiation

Severity

Timing

Modifying factors of pain sensation

Prevention of further nociceptive stimuli (e.g. positioning and fixation of broken limb)

Social factors (presence of family)

A few sips of water

Fight or flight response:

- Selective: defence, flight and learning
- Non-selective:
    - Neuroendocrine changes: sympathetic nervous system activation, catabolic hormone release, endorphin secretion
    - Inflammatory response: complement and cytokine activation
    - Psychological response: discomfort, anxiety

Estimation of pain: Pain is a subjective feeling. To standardise the estimation of pain objective measurements are required

Behaviour: it can help estimate pain in children, elderly, mentally disabled or patients who cannot speak the same language as the healthcare professional

Vital Parameters:

- elevated HR and NIBP, arrhythmias, AMI
- hyperventilation, decreasing SpO2
- elevated BG
- sweaty hands
- slower GI motility

Self-report Pain Scales: ask the patient to estimate his own pain:

1 dimension scales:

- Numeric scales: estimate pain in a 0-10 scale
- Visual Analogue Scale (VAS): patients point at a position along a continuous line between no pain and unbearable pain
- Verbal scales: No pain/Minimal pain/Moderate pain/Sever pain/Unbearable pain.
- For children Wong-Balers Faces rating score

0 1 2 3 4 5 6 7 8 9 10

no pain moderate pain severe/unbearable pain

Numeric pain scale

no pain unbearable pain

Visual Analogue Scale (VAS)

Multidimensional scales

Initial Pain Assessment Tool (IPAT):a questioner to specify the pain**.**

Management of pain:

- **Non-invasive technics:** cooling, verbal technics, relaxation, TENS (transcutaneous electrical nerve stimulation)
- **Medication:** minor/major analgesics
- **Invasive technics:** local or regional anaesthesia
- **General Anaesthesia**

Analgesics: the ideal analgesic has immediate effect, easy to dose and not modify the planned examinations.

1. Opioids:

- Opioids bind to κ, μ, δ receptors.
- Classification of opioids:
  - Opioid agonists: (See Table 1.) first choices in treatment of moderate/sever acute pain.
  - Agonist–antagonist opioids (buprenorphine, nalbuphine, pentasocin) Ceiling effect: increasing doses of a given medication has progressively smaller incremental effect.

    - Opioid antagonists (naloxone) effectively block the opioid receptor, inhibiting opioid effect (given in case of respiratory depression) 0.4-2mg iv/im/sc. repeat every 2-3minutes. Maximum dose: 10mg.
- Opioids' Side effects:
    - nausea and vomiting, urinary retention, pruritus, respiratory depression
    - *NB: stop administering opioids below 10/min respiratory rate in adult patients!*
- Opioid administration: iv. im. sc. buccal, transdermal
- Equipotent dosage:different doses of different opioids has the same effect. Switching opioid therapy requires accurate calculation of the equivalent doses.
- Cardinal rules in opioid treatment
    - opioids should be titrated with small repeated doses to achievefull analgesia but avoid side effects
    - Use opioids carefully in elderly patients, start with low doses
    - No addiction occurs as a result of acute pain management
    - Titrate opioids carefully if the patient has hepatic or renal failure
    - Titrate opioids carefully and monitor the patient with COPD, or any other respiratory disorders
    - Avoid opioid intoxication

Drug	Adult doses	Pharmacokinetics	Toxicity
Morphine	0.1 mg/kg iv. 10 mg im. 0.3 mg/kg p.o.	Onset: 5 min iv. 10-15 min im/sc. Maximal effect: 15-30 min Duration: 1-2 h (iv.)-3-4 h (im.,sc.)	histamine related anaphylaxis respiratory depression sedation

Fentanyl	0.5-3 μg/kg	Onset: prompt Maximal effect: 1-5 min Durtion:30-40 min	respiratory depression (>5μg/kg): chest rigidity
Pethidine	50-150 mg.iv/sc./im.	repeat every 3-4 h, max.600 mg/day	Seizures, ECG changes respiratory depression
Tramadol	50-100 mg iv	repeated once if required after 30 minutes Duration: 4-6 h400(-800) mg/day	
Codeine	30-60 mg p.o.	Onset: 4h often combined with paracetamol	nausea and vomiting has moderate effect

Table 1.

Non-Opioid Analgesics:

1. NSAIDs (carboxylates, enolates, pyrazolones) inhibit the excretion of inflammatory mediators. Solely NSAIDs can be effective against muscle pain, pelvic or gynaecologic pain and headache. Some of the NSAIDs have antipyretic effect too.
    - selective COX2 inhibitors/non-selective COX2 inhibitors (diclofenac)
    - side effects: thrombocyte dysfunction, GI bleeding, GI irritation, AKI

2. Paracetamol (acetaminophen) has no anti-inflammatory effect, and has no effect on thrombocytes.
    - Dosage: 500/1000mg 4-6 times a day. Maximum dose: 4g
    - No serious side effect, can be used in mild hepatic or renal impairment
    - Toxic dose: 140mg/kg/24h

Additional drugs:

1. Ketamine (phencyclidine): has analgesic and dissociative effect without any effect on respiration.
    - Used for minor interventions and administered to elderly patient with broken hip
    - Dose: 0,5-1mg/kg slowly

2. N2O (*nitrous oxide*): rapid onset, with short duration for minor interventions.
    - Side effect: nausea and vomiting.
    - DO NOT USE in patients with skull fracture, chest trauma, or suspected abdominal perforation!
3. Tricyclic antidepressants (e.g. amitriptyline, gabapentin) used against chronic neuropathic pain.
4. Steroids are used against neuropathic or articular pain.

WHO's Analgesic Ladderused for the management of acute and chronic pain (initially it was developed for cancer pain).For minor pain use minor analgesic, then I pain cannot be eased change drugs till total analgesia.

Local anaesthesia: for safe use of local anaesthetics basics of anatomy and pharmacology isrequired.

- Topical/Infiltrative local anaesthesia: EMLA/Lidocain, Ropivacaine, Bupivacain infiltration
- Peripheral nerve block: Anaesthetics is infiltrated near ulnar, radial, median or femoral nerves.

Sedation in the ED: the aim of the sedation is to eliminate anxiety and prevent the patient to remember the pain and theunpleasantfeelings.

GENERAL ANAESTHESIA is necessary for:

- Short surgical intervention
- Airway management
- Mechanical ventilation
- Patient with skull fracture to guarantee the proper PaCO2 level
- Status Epilepticus
- Cardioversion
- Patient with shock if lifesaving operation is need

Before general anaesthesia:

- Read patient's history (respiratory or circulatory disorders, epilepsy, GERD…)
- Current medication
- Allergies
- Mallampati scoring
- ASA scoring (If ASA is III-IV seek consultant's help)

Preparation:

- Prearrange every tool, which is necessary for General Anaesthesia
- Prearrange every drug required
- Monitor the patient

Induction: give analgesic, anaesthetic,sedative and/or muscle relaxant drugs.

- Analgesics:
    - Fentanyl: 1-2µg/kg iv.
    - Morphine: 0,1-0,15 mg/kg iv.
- Anaesthetics:
    - Propofol: Induction dose in healthy patients: 1,5-2,5mg/kg, in patient with shock, or in elderly patient titrate with 10-40mg. The patient is anaesthetised when verbal contact is lost. As the adverse effect of propofol respiratory depression and phlebitis on the site of the injection may occur.
    - Etomidate has a rapid onset, brief duration. Etomidate has cardio-depressive effect. Induction dose: 0,1-0,3 mg/kg
    - Ketamine Induction dose: 1-2 mg/kg may be repeated every 8-10 minutes if required in 1-1,5 mg/kg dose
- Peripheral muscle relaxants:
    - Suxamethonium 1mg/kg
    - Rocuronium: 1mg/kg

Airway management (basic or advanced)

Awakening: check the followingsat the end of general anaesthesia before pull out advanced airway:

- The patient co-operates.
- Muscle strength returned.
- Airway protective reflexes returned
- Spontaneous breathing returned

After general anaesthesia the patient has to be monitored at least 6 hours.

14. Laboratory tests in the EDbyDr.BorosIstván

Laboratory tests are essentials in the ED. Laboratory tests are used for evaluating the patients' condition and the efficacy of the given therapy, it is also inevitable in the medical decision making.Without laboratory tests, imaging or other diagnostic procedures Evidence Based Medicine would be inconceivable. To avoid useless or confusing mass of data but gain proper diagnostic results always take the followings into consideration:

- Why I order this laboratory test?
- What I expect from the laboratory test?
- Will the results change the diagnosis?
- Will the results affect the case's outcome?
- Will the patient have benefits from the results of the laboratory test?

NB: Treat the patient not the laboratory value!

1. Terminology:

- Serum: The fluid part of the blood obtained after removal of the fibrin clot and blood cells
- Plasma: The fluid part of the blood, in which blood cells and fibrinogen are suspended.
- Full blood count: is a panel of tests that evaluates the three types of cells that circulate in the blood
- Full blood count with differential: It identifies and counts additionally the number of the various types of white blood cells present
- Haemostasis: the blood coagulation
- Reference range: the variations of a measurement or value in healthy individuals, the prediction interval between which 95% of values of a reference group fall into
- Turnaround Time TAT: The interval between the ordering of a clinical laboratory test and the reporting of results
- Routine Blood test: depend on the local habits

2. Sampling for laboratory tests

Good practice in blood or urine samplingimprovesthe patient safety and provides reliable laboratory values. In ED improper blood or urine sampling may cause delay in diagnosis and consequently in definitive treatment.

General rules:

- Identify the patient, and label the collecting tubes
- Planning ahead, prepare all the items you will need!
- Using an appropriate location!
- Perform hand hygiene
- Always use gloves! Avoid any contact with the patient's blood or urine!
- Use disinfectant sprays e.g. 70% isopropyl alcohol and let the skin dry before drawing blood.
- Use closed systems for blood sampling
- After drawing blood discard the used blood-sampling device into a puncture-resistant container.

Venous blood sampling:

- Location: superficial veins (e.g. cubital veins), avoid the sites where any drug is administered
- Disinfect the site of the planned puncture
- Apply a tourniquet, about 5-10 cm widths above the selected venepuncture site. Ask the patient to make a fist
- Insert the needle at an angle of approximately 15-30 degrees
- Use vacuum blood collecting tube in the right order (leave last the tubes with anticoagulant), draw blood to the mark on the side of the tube, then carefully inverse the tubes a few times (do not shake).
- Ask the patient release the fist
- Remove the needle

- Apply pressure with gaze on the site of the puncture for 5 minutes

Arterial blood sampling:

- Location: superficial artery (radial or femoral)
- Disinfect the site of the planned puncture
- Use heparinised syringe for blood gas measurement
- Insert the needle at an angle of 45 degrees
- Draw minimum 1 mL blood
- Apply pressure immediately after drawing blood with gaze on the site of the puncture for 10 minutes or longer if the bleeding is not stopped
- Eliminate air from the syringe, carry out measurement as soon as possible

Capillary blood sampling:

- Location: lateral part of fingertips (avoid the index finger) or heels in infants
- Disinfect the site of the planned puncture
- Wipe the first drop of blood
- Do not apply too much pressure on the finger/heel
- Apply pressure with gaze on the site of the puncture for a few minutes

Urine sampling

- Cleansing the urethral area with clear water
- Use sterile urine collection container
- For the optimal measurement use sample of urine midstream in adults (in infants urine collection bag is used)
- Urine samples can be collected from catheters using a syringe, but let a small portion out before sampling. Alternatively in patients with permanent indwelling catheters use a needle to puncture the side of the catheter for sampling after disinfected the site of puncture.
- 10ml of urine is often enough for basic measurements
- Do not accept self-obtained samples

Laboratory tests used in the ED:

In the ED laboratory tests are used for:

- Rapid assessment
- Differential diagnostics
- Monitor the dynamics of the patients' condition
- Monitor the efficacy of treatment

The laboratory works effectively if TAT < 60 minutes, consequently emergency physicians have to rationalize the order of testing. For better laboratory performance the "shotgun" approach to ordering laboratory tests has to be replaced by a "rifle" (or targeted) approach.

Panels used in the ED:

- Electrolytes: Na^+, K^+, Cl^-, Ca^{2+}
- Liver function: ALP, GOT, GPT, GGT, Bilirubin, total serum protein, albumin
- Renal function: BUN, creatinine
- Pancreas function: serum amylase, lipase
- Cardiac enzymes: LDH, CK, CK-MB, Troponin T
- Metabolic function: serum glucose
- Inflammatory markers: CRP, PCT, Erythrocyte Sedimentation Rate (not used in emergency care)
- Toxicology, TDM (ethanol, common drug levels and illicit materialtests)
- Pregnancy: beta-hCG
- Full blood count
- Haemostasis panel: PT/INR, aPTT, TT, fibrinogen, D-dimer
- Routine urinalysis: specific gravity, pH, protein, ketone, glucose, bilirubin, urobilinogen, nitrite, haemoglobin, red blood cells, white blood cells, microscopic analysis

1. Electrolyte panel is ordered frequently as part of a routine screening or evaluation of patient withsuspected renal failure, cardiac arrhythmias, neuromuscular disorder, metabolic disorders, electrolyte or acid base imbalance.

    a. Na^+: Main extracellular cation. Has a role in regulation of neuromuscular junction, and Na^+ is responsible for maintaining the osmotic pressure gradient.

    Normal serum value: 135-145 mmol/L

    Hyponatraemia: low sodium intake, excessive perspiration, polydipsia, extreme physical activity, acidosis, diuretic overdose, vomiting, diarrhoea, bleeding, oedema, peritonitis, malignant diseases and poisoning.

    Hypernatremia:excessive sodium intake, dehydration, renal impairment, cardiac failure and burns

    b. K^+: Main intracellular cation. Has an important role in buffering system.

    Normal serum value: 3.5-4.5 mmol/L

    Hypokalaemia: low potassium intake, starvation, dehydration, vomiting, diarrhoea, diuretic overdose and dialysis

    Hyperkalaemia: starvation, physical exhaustion, alkalosis, dehydration, impaired renal function and salicylate poisoning

    c. Cl^-: The main inorganic anion of the extracellular space. Chloride (with sodium) is responsible for maintaining the osmotic pressure gradient.

    Normal serum value: 99-111 mmol/L

    Hypochloraemia: alkalosis, excessive perspiration, low chloride intake, hyperhydration, hypernatremia with oedema, emphysema, vomiting, diarrhoea, gastric tube and suction, renal and cardiac disorders.

    Hyperchloraemia: acidosis, dehydration, insufficient water intake, diarrhoea, pregnancy, anaemia, endocrine disorders and renal failure

    d. Ca^{2+}: is responsible for maintaining membrane potential, has an important role in contractile tissue function and membrane permeability. Calcium has a role in enzyme functions and in the excretion of enzymes. The role of calcium in the haemostasis is essential.

Hypocalcaemia: intestinal absorption disorders, decreased serum magnesium level, starvation, decreased level of vitamin D, pregnancy, alkalosis, alcohol use disorder, dialysis, diarrhoea, renal failure, malignant disorders, sepsis, rhabdomyolysis, massive transfusion chronic or acute pancreatitis and osteoporosis

Hypercalcaemia: high calcium intake, immobilisation, vomiting, dehydration, malignant diseases, endocrine diseases, acute pancreatitis, renal failure acute alcohol poisoning

2. Liver function: it is a frequently (routinely) ordered test in suspected cases of liver and biliary diseases, renal and cardiac diseases, sepsis, poisoning or tissue damage.

    a. ALP: alkaline phosphatase is responsible for ester hydrolysis. ALP is in high concentration in liver and biliary tract, bones, kidney and intestines.

    Normal serum value: 40-115 IU/L

    Lower ALP: starvation, alcohol use disorder, lack of vitamin B12, pregnancy, anaemia, inorganic acid poisoning and vitamin D poisoning.

    High ALP: pancreatitis, biliary tract obstruction, fever, acute renal failure, ileus, peritonitis, hepatic disorders, dialysis, alcohol use disorder

    b. sGOT/AST: serum Glutamic-Oxaloacetic Transaminase/Aspartate Transaminase microsomal and mitochondrial enzyme in the tissue of liver, heart, muscles, kidneys, brain, pancreas, spleen and lungs. sGOT is a good marker of tissue necrosis.

    Normal serum value < 40 IU/L

    Elevated sGOT: tissue necrosis, hepatic diseases, ileus, burns and glucose storage diseases

    c. sGPT/ALT Serum Glutamic-Pyruvic Transaminase/Alanine Transaminase is an enzyme of liver, muscle, kidneys an heart.

    Normal serum value < 40 IU/L

Elevated sGPT: alcohol use disorder, fever, hypoxia, haemolysis, infectious hepatic diseases, biliary, intestinal or pancreatic diseases, muscular damages, malignant diseases and poisoning

d. GGT: Gamma-GlutamylTranspeptidaseis responsible for peptide and amino acid transmembrane transport.

   Normal serum value 7-50 UI/L

   Low GGT: pregnancy, hypothyroidism

   High GGT: alcohol use disorder, obesity, postoperative state, hepatitis, biliary disorders, malignant diseases, renal or cardiac diseases (elevated GGT level can be seen for 2-4 days in patients with AMI), sepsis, poisoning.

e. Total serum protein: serum proteins has transport functions. This value is a good indicator of feeding and immune status. Proteins are responsible for maintaining intravascular osmotic pressure.

   Normal serum value: 60-80 g/L

   Hypoproteinaemia: starvation, malnutrition, plasmapheresis, massive bleeding, hepatic or renal failure, postoperative state, nephrotic sy., different enteropathies.

   Hyperproteinaemia: dehydration, infectious diseases, malignant diseases and haematological disorders.

f. Albumin: a protein produced in the liver. Albumin is a transport protein and is responsible for maintaining plasma osmotic pressure.

   Normal serum value: 35-53 g/L

   For hypoalbuminaemia and hyperalbuminaemia see hypoproteinaemia and hyperproteinaemia.

g. Bilirubin is produced during the degradation of haemoglobin in the liver and excreted into the bile. In the laboratory two different types of bilirubin is measured, conjugated and unconjugated bilirubin.

   Normal serum value: 3-17 μmol/L

Hypobilirubinaemia: severe anaemia

Hyperbilirubinaemia: haemolysis, starvation, hepatic disorders, biliary disorder, malignant diseases, infectious diseases, hepatic failure, cardiac failure and as a complication of transfusion.

3. Renal function is a frequently ordered routine laboratory test, used for evaluate the patient, monitor haemodialysis, differential diagnostics in suspected poisoning, assessing fluid status, renal impairment or failure. Estimated glomerular filtration rate (eGFR) is a widely used calculated value for estimation of renal function.

    a. Creatinine is the end product of the muscle protein metabolism. eGFR is calculated by an equitation based on creatinine level.

    Normal serum value for men: 62-106 μmol/L, for women: 44-97 μmol/L

    Low creatinine level: elderly patient, anaemia, muscle dystrophy, pregnancy

    High creatinine level: dehydration, renal failure (pre-renal, renal or post-renal), muscle injury, burns, diabetes, fever, endocrine disease, ileus and acetone poisoning

    b. Blood urea nitrogen (BUN): BUN is the degradation product of ammonia, produced during the protein metabolism.

    Normal serum value: 3.8-7.3 mmol/L

    Low BUN: low protein intake, starvation, pregnancy, fluid overload

    High BUN called uraemia: acute or chronic renal failure (pre-renal, renal and post-renal), muscle injury, burns, diabetes, hypovolaemia, sepsis and shock

4. Pancreas panel has an important role in the differential diagnostics of acute or chronic abdominal pain.

    a. Amylase is an enzyme produced in salivary/intestinal glands and in the pancreas.

    Normal serum value < 100 IU/L

    Low amylase level: permanent pancreatic injury, renal impairment and toxaemia of pregnancy.

High amylase level: pancreatitis, parotitis, abdominal trauma, peritonitis, ectopic pregnancy, after ERCP, acidosis, appendicitis and poisoning.

b. Lipase is an enzyme produced in pancreas, excreted into duodenum. Lipase is responsible for the metabolism of long chain fatty acids.

   Normal value < 70 IU/L

   Low lipase level: permanent pancreatic injury, hyperlipidaemia, pancreatic failure

   High lipase level: alcohol use disorder, pancreatitis, enteral disorders, after ERCP and in ectopic pregnancy.

5. Cardiac enzymes are essentials in the differential diagnostics of acute chest pain (see Management of Chest Pain in the ED). Besides diagnosis of AMI cardiac enzymes are feasible for monitoring cardiotoxic drugs, muscle disorders, rhabdomyolysis, compartment syndrome and other extracardial diseases.

   a. Creatine-kinase (CK) is an enzyme of all types of muscles, therefore CK is a perfect indicator of muscle injuries.

      Normal serum value: 24-195 IU/L

      Low CK level: immobilization, metastatic malignant diseases, pregnancy, thyroid diseases.

      High CK level: muscle injury, burns or any muscle diseases, arrhythmias, AMI, defibrillation, hypothermia, epileptic seizures, physical exhaustion, sepsis, shock, renal or cardiac failure.

      a.i. Creatine-kinase-MB CK-MB is an isoenzyme of CK, specific for myocardial injury.

      b. Troponin T: troponin is a myocardial structural protein one of three different types of troponin, troponin T is solved in myocardial cytoplasm, therefor Troponin T is a very specific (94%) marker of AMI.

      Normal serum value: 0.1 ng/mL

      High troponin T level: in AMI, unstable angina, cardiac trauma, cardiac surgery renal failure (Troponin T has renal elimination).

6. Metabolic parameters –serum glucose measurement is used for routine evaluation and monitoring diabetic patients.

    a. Serum Glucose (also can be measured with point of care technique)

        Normal value: 3.6-6 mmol/L

        Hypoglycaemia: extensive physical activity, starvation, insulin overdose, insulinoma, pancreatic, hepatic or renal failure, fever, sepsis, antidiabetic overdose, alcohol use disorder and beta-blocker poisoning

        Hyperglycaemia: obesity, metabolic X syndrome, diabetes, physical inactivity, pain, fever, stress, seizure, pheochromocytoma, ileus, pancreas failure, malignant diseases, surgical intervention, renal impairment, and poisonings: CO, theophylline…

7. Inflammatory markers are used for differential diagnostics of diseases with fever, sepsis or infection, if inflammatory diseases is diagnosed, for monitor treatment.

    a. Erythrocyte sedimentation rate is the rate at which red blood cells sediment in a period of one hour. Not used in ED because it has few specificity.

        Normal value < 20mm/hr

        ESR is elevated in many diseases: inflammatory, autoimmune, organ failures, pregnancy, poisoning, burns and trauma

    b. C-reactive protein (CRP) IgG type acute phase protein. CRP helps opsonisation, foreign body elimination and detoxification.

        Normal serum value < 5.0 mg/L

        High CRP level: tissue ischaemia, infection, surgical intervention, necrosis, burns, trauma and transplant rejection

    c. Procalcitonin (PCT) is the precursor of calcitonin, and it is absent in healthy peoples. Procalcitonin is elevated in bacterial infections but not in viral or sterile inflammation. 2-3 hrs after the onset of infection procalcitonin is elevated.

        Normal PCT level < 0.5 μg/L

PCT is elevated in bacterial, fungal or parasite infections, sepsis, fever, shock, multi organ failure, surgical intervention, trauma, burns and acute pancreatitis

8. Therapeutic drug management (TDM) and toxicology: TDM is the measurement of therapeutic drug levels. Toxicology measurements is used for detect illicit materials.

   From urine frequently used illicit drugs can be detected.

   Blood alcohol content:

~0,3‰		euphoria, calm, dissolved inhibitions, logorrhoea, decreased ability to concentrate on
0,8-1,2‰	influenced	Impairment of coordination and motor functions.
1,2-1,6‰	mild drunkenness	Worsening motoric functions,, unscrupulousness, slurred speech, loss of sense of danger, prolonged reaction time
1,6-2,0‰	moderate drunkenness	Symptoms mentioned above getting worst
~2,0‰	severe drunkenness	Drowsiness, depression, dysphoria with nausea.
2,0-3,0‰	Ethanol poisoning	Lost consciousness
3,0-4,0‰	drowsiness, stupor	
>4,0‰	coma	

9. Pregnancy: Beta hCG (human chorionic gonadotropin) is a sensitive test for pregnancy. In the ED diagnosing pregnancy is essential in the following cases:

   - differential diagnostics of abdominal pain in women of child-bearing age
   - abdominal trauma in women of child-bearing age
   - suspected ectopic pregnancy
   - monitor preterm delivery
   - diagnosis of trophoblastic tumours or germ cell tumours

Normal serum value in non-pregnant women < 3.0 IU/L

In the ED qualitative beta hCG measurement is sufficient to exclude or diagnose pregnancy or ectopic pregnancy, quantitative measurement is not used routinely. POCT qualitative beta hCG test is available in the ED and provides early diagnosis in the cases mentioned above.

10. Full blood Count: is one of the most common laboratory tests used in the ED. It provides information about bleeding (acute or chronic), inflammation, haematological diseases, malignant diseses or cellular haemostasis disorders.

    Full blood count:

    - WBC (white blood cell) Normal value: 4-10 G/L
    - RBC (red blood cell) Normal value: 4.5-5.5 G/L
    - Hgb (haemoglobin) Normal value: in males 13-17 g/dL in females 12-15 g/dL
    - HCT (haematocrit) Normal value: in males 40-50% in females 35-45 %
    - MCV (mean corpuscular volume) Normal value: 80-100 fL
    - MCH (mean corpuscular haemoglobin) Normal value: 26-34 pg
    - MCHC (mean corpuscular haemoglobin concentration) Normal value: 32-36 g/dL
    - RDW (red cell distribution width) Normal value: 12-15%
    - PLT (platelet) Normal value: 150-400 G/L
    - MPV (mean platelet volume) Normal value: 80-100fL

11. Haemostasis: this laboratory panel used for monitoring haemostasis in patients with bleeding or bleeding disorders, or in patients with regular anticoagulant therapy. Haemostasis panel provides sensitive information about liver function in every cases with suspected hepatic failure.

    - PT: prothrombin time monitor the extrinsic and the common coagulation pathway (Factor 1, 2, 5, 7, 9, 10, 11 and 12) Normal value: 12-15 sec.
    - INR: international ration the standardised form of PT. Normal value: 0.9-1.15
    - aPTT activated partial thromboplastin time monitors the intrinsic and the common coagulation pathway (HMWK, prekallikrein, fibrinogen, Factor 2, 5, 8, 9, 10, 11 and 12) Normal value: 25-35 sec.

- TT: thrombin time monitors velocity of the fibrinogen-fibrin transformation. The test is used in suspected DIC, lack of fibrinogen and for monitoring heparin therapy. Normal value: 18-22 sec.
- Fibrinogen is an acute phase protein and has an essential role in coagulation. The test is used in suspected DIC, bleeding disorders and for monitoring fibrinolysis. Normal value: 1.5-4.0 g/L

12. Routine urinalysis is used in patients with suspected urinary tract infection, renal impairment, urinary lithiasis or metabolic disorders.

    The measured parameters: urine specific gravity, pH, glucose, ketone bodies, bilirubin, urobilinogen, nitrite, haemoglobin, red blood cells, white blood cells and protein.

POCT Point of Care testing in the ED

Definition: A clinical laboratory measurement made at the bedside, in the clinic, or in the patient's home rather than at a centralized laboratory. The measurement may be carried out by healthcare professionals or in some cases the patient too.

POC testing has a short TAT, provides results almost promptly, therefore POC testing has a great importance in emergency care.

Commonly used POCT in ED:

- Serum glucose
- Blood gas analysis
- Cardiac markers (Troponin T/I, CKMB, BNP, Myoglobin)
- Haemostasis (INR, D-dimer, Thromboelastography)
- Urinalysis
- Toxicological panels

a. Blood gas analysis is commonly used in critically ill patients.

    a.i. Measured parameters: pO2, pCO2, pH

    a.ii. Calculated parameters: BE, HCO_3^-, c HCO_3^-, SpO2, O2

    a.iii. Metabolites: lactate, glucose

a.iv. Electrolytes: K^-, Na^+, Cl^-, Ca^{2+}

pH is the negative of the logarithm to base 10 of the activity of the hydrogen ion. Normal value: 7.35-7.4. Acidaemia: pH < 7.35, alkalaemia: pH > 7.45.

$PaCO_2$: partial pressure of carbon dioxide (CO_2) in arterial blood. Normal value: 35-45 mmHg. CO2 is produced during physiological metabolic processes and eliminated by alveolar ventilation.

Respiratory acidosis: pH↓, $PaCO_2$↑ (increased CO_2 production, decreased CO_2 elimination)

Respiratory alkalosis: pH↑, $PaCO_2$↓ (increased CO_2 elimination – hyperventilation)

PaO_2: partial pressure of oxygen (O_2) in arterial blood. Normal value: 8-100 mmHg. PaO_2 provides information about the body's oxygen supply. In case of imbalance of oxygen supply and demand the PaO_2/FiO_2 rate (Carrico index) provides information about the severity of the respiratory failure.

HCO_3^- is the main buffer of the human body. In the kidneys H_2CO_3 -> $H^+ + HCO_3^-$. The proton (H^+) can be eliminated via kidneys according to the acidity of the blood. Normal value: 22.26 mmol/L.

BE (base excess) is defined as the amount of strong acid that must be added to each litre of fully oxygenated blood to return the pH to 7.40. A base deficit (i.e., a negative base excess) can be correspondingly defined in terms of the amount of strong base that must be added. Normal value: −2 to +2 mEq/L.

Metabolic acidosis: pH↓, HCO_3^-↓, BE↓

Metabolic alkalosis: pH↑, HCO_3^-↑, BE↑

Thromboelastography: Thromboelastography (TEG) is a method of real time testing the dynamics of blood coagulation. It provides visual information about the clot formation in time. For the measurement only 15-20 minutes is needed. TEG is used in critically ill bleeding patients, during surgery with extensive blood loss or in cases when massive transfusion is needed. TEG has a fast growing significance in the emergency care.

Measured parameters:

- Clotting time
- Clot formation time
- Shear modulus strength
- Maximum clot firmness

- Elasticity constant
- Clot lysis index

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