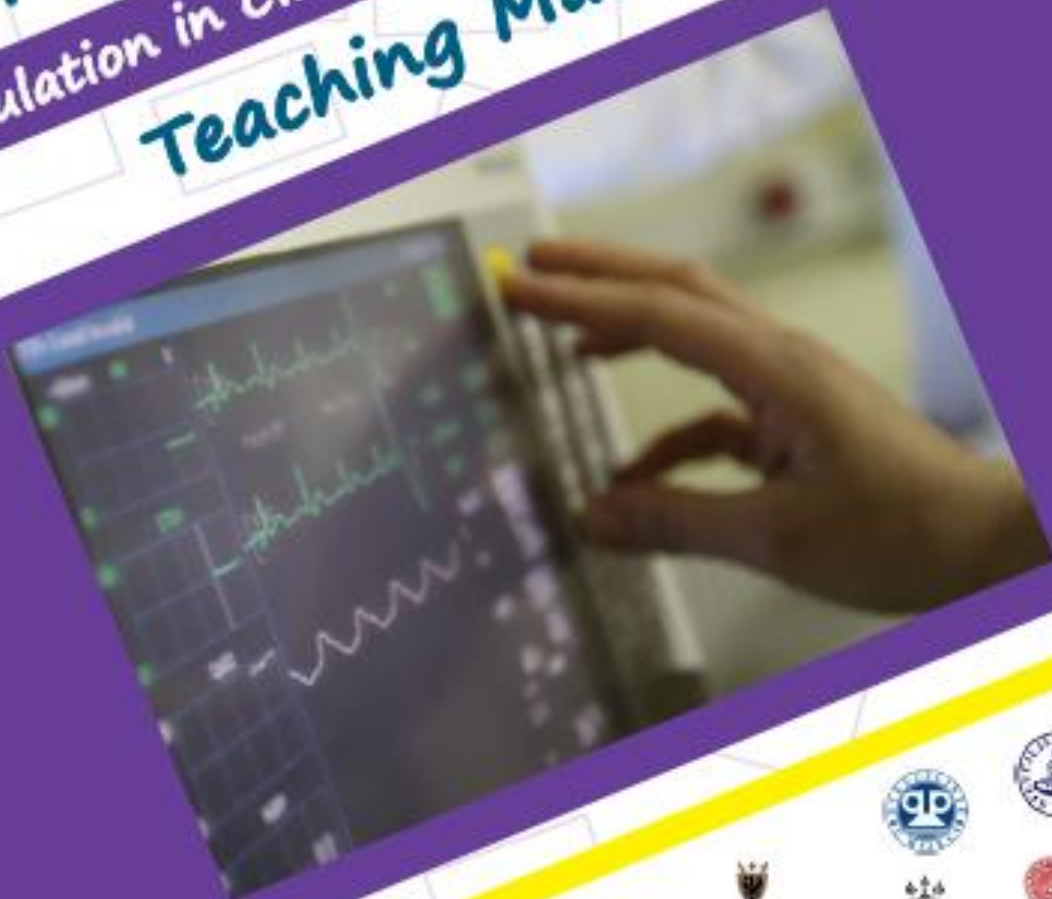




# ERASMUS INTENSIVE PROGRAMME

Simulation in Clinical Practice: Virtual Medicine  
Teaching Material



**Erasmus Intensive Programme: Simulation in Clinical Practice**

**TEACHING MATERIAL**

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## **TABLE OF CONTENTS**

LIST OF AUTHORS AND CONTRIBUTORS .....	4
TABLE OF CONTENTS .....	5
FULL-DAY WORKSHOPS .....	7
SECTION 1: ULTRASOUND IN EMERGENCY MEDICINE .....	8
1.1 Theoretical Background.....	8
1.2 Introduction to US Technology and Image Interpretation.....	8
1.3 Basic Applications of US in Emergency Medicine.....	9
1.4 Presentation of Simulation Scenario – Time Schedule.....	14
References.....	14
Correspondence .....	15
SECTION 2: AIRWAY MANAGEMENT .....	16
2.1 Theoretical Background.....	16
2.2 Advanced Tracheal Intubation Techniques.....	23
2.3 Presentation of the Simulation Scenario - Time Schedule .....	27
References.....	27
Correspondence .....	28
SECTION 3: ACUTE CORONARY SYNDROME AND ITS COMPLICATIONS.....	29
3.1 Theoretical Background of Acute Coronary Syndrome.....	29
3.2 Theoretical Background of Arrhythmias .....	32
3.3 ECG strips of Important Arrhythmias to be Recognized on Monitor .....	34
3.4 Presentation of the Human Patient Simulator .....	35
3.5 Presentation of Simulation Scenario – Time Schedule.....	36
3.6 Acute Coronary Syndrome and Arrhythmias Management Algorithms .....	37
References.....	40
Correspondence .....	40
SECTION 4: ADVANCED CARDIAC LIFE SUPPORT (ACLS) .....	41
4.1 Theoretical Background.....	41
4.2 Advanced Cardiac Life Support.....	41
4.3 Managing the Peri-arrest Patient.....	42
4.4 Presentation of Simulation Scenario – Time Schedule.....	42
4.5 Advanced Cardiac Life Support Basic Take Home Messages .....	43
References.....	43
Correspondence .....	43

SELECTED TOPICS IN SIMULATION .....	44
SECTION 5: SELECTED TOPICS IN SIMULATION – CARDIOLOGY SIMULATION .....	45
5.1 Theoretical Background.....	45
5.2 Presentation of the Simulator .....	46
5.3 Presentation of Simulation Scenario – Time Schedule.....	48
References.....	48
Correspondence .....	48
SECTION 6: SELECTED TOPICS IN SIMULATION – GYNAECOLOGY SIMULATION .....	49
6.1 Theoretical Background.....	49
6.2 Presentation of the Simulator .....	50
6.3 Simulation Scenario .....	51
References.....	51
Correspondence .....	51
SECTION 7: SELECTED TOPICS IN SIMULATION – OPHTHALMOLOGY SIMULATION.....	52
7.1 Theoretical Background.....	52
7.2 Presentation of the Simulators.....	52
7.3 Presentation of Simulation Scenario – Time Schedule.....	53
References.....	53
Correspondence .....	54
SECTION 8: SELECTED TOPICS IN SIMULATION – GASTRENEROLOGY SIMULATION .....	55
8.1 Theoretical Background.....	55
8.2 Presentation of the Simulator .....	55
8.3 Presentation of Portable Ultrasound Device.....	56
8.4 Basic Abdominal Ultrasound Examination Protocol.....	57
8.5 Presentation of Simulation Scenario – Time Schedule.....	57
References.....	58
Correspondence .....	58

# **FULL-DAY WORKSHOPS**



# **SECTION 1: ULTRASOUND IN EMERGENCY MEDICINE**

*Authors: Gregor Prosen, Matej Strnad*

<b>CONTENT</b>	
1.	<b>Theoretical Background</b>
2.	<b>Introduction to Ultrasound Technology and Image Interpretation</b>
3.	<b>Basic Applications of Ultrasound in Emergency Medicine</b>
4.	<b>Presentation of Simulation Scenario – Time Schedule</b>

## **1.1 Theoretical Background**

Emergency ultrasound (US) has become indispensable tool in armamentarium of every physician that takes care of (potentially) critically ill/injured patients, be it in prehospital arena, emergency departments (ED) or intensive care units (ICU). With its growing use, experience and evidence, utility of point-of-care-ultrasound (POCUS) has now expanded beyond emergency/critical care and small, portable, handheld devices can now be used in almost all fields of somatic medicine, from orthopaedics, sports medicine, gynaecology, paediatrics and off course internal medicine with its myriad sub-specialities (1).

Main aim and benefit of US use and incorporation in emergency medicine/critical care stems from the fact that it can greatly improve sensitivity and specificity of physical examination (2). Simply put, even limited ultrasound examination is in most applications superior to traditional, un-enhanced physical examination. However, in the same breath, we shall emphasize, that interpretation of POCUS can only make sense if put in greater clinical context of history, physical examination and wider clinical acumen. Being aware of this crucial point, using focused POCUS exam based on previous assessment of pre-test probability (history, classical propaedeutic), it becomes clear, that in order to use POCUS properly, we must be even more skilled in classical medical approach and clinical thinking, as otherwise, POCUS can give us totally misleading information and can ultimately even harm patients. It is thus of utmost importance to use POCUS as an *extension* and **not** a substitute of clinical examination (and clinical reasoning). Such use of US technology is conceptually *different* from US use by imaging specialists (radiologists, cardiologists, gynaecologists), who possess breadth and depth of knowledge that can interpret sonographic images mostly independent on its own.

## **1.2 Introduction to US Technology and Image Interpretation**

US in a form of mechanical waving with frequency over 20 kHz. Audible sound possesses frequencies from 20 Hz to 20 kHz. Since it is not a form of electromagnetic energy, it possesses none of risks associated with those diagnostic modalities (X-ray, CT) and can thus be safely used without fear of ionising radiation (especially important in paediatrics).

US or *sonography* generates images based on echoes that are reflected back to the probe. Reflection of emitted US waves is dependent on **echogenicity** of different tissues and fluids; namely, water and bodily fluids offer almost no resistance (and thus no reflection), thus US waves can pass through body fluids fully and reflect no echoes. Thus, water and body fluids (vessels, free fluid, and urine) are sonographically termed **an-echogenic**. On opposite side of spectrum are bone and air, which are ultimate reflectors of ultrasound, thus ultimately **hyper-echogenic** and for US impenetrable. Most parenchymatous organs are in large part composed of cellular water and thus are easily penetrable



to US and in some form **iso-echogenic**. Echoes, reflected back from organs, are “drawn” by US machine in forms of organs (gallbladder, kidney, heart), and thus enable clinician to recognise different organs and pathologies.

US image is a spectrum of 256 shades of grey, depending on different echogenicity. It is two-dimensional image, which is generated by operator himself. For clinical sono-operators it is of utmost importance to understand orientation of probe, different axis of image generation and how they relate to image interpretation on screen.

### **1.3 Basic Applications of US in Emergency Medicine**

**First** application of US in critical/emergency medicine was **FAST** protocol, aimed at identifying free intraperitoneal fluid (in context of trauma presumably blood). Since identification of internal bleeding is very unreliable by traditional clinical means, use of FAST protocol added major capability in recognising which patients are shocked due to internal bleeding and thus need urgent surgery (3). FAST protocol is perfect example of successful US application in critical care; used for **clear indication**/clinical question [is there free fluid/blood in peritoneal cavity and/or pericardium?], **clear technique** (clearly defined probe positions - **figure 1**) and **clearly defined sonographic patterns** and its **interpretation**, meaning and clinical decision-making (**figure 2**).

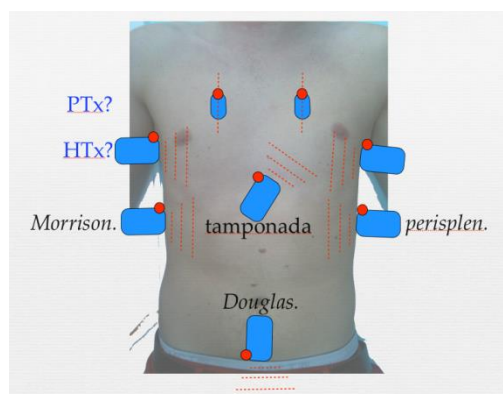


Figure 1: Probe positions.

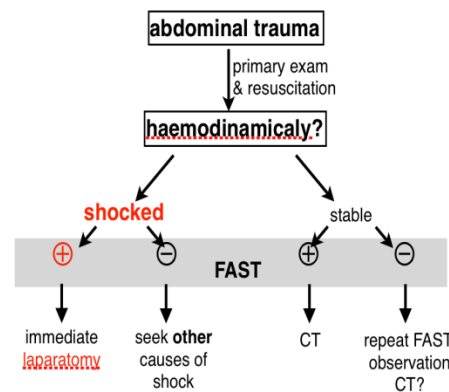


Figure 2: Algorithm of abdominal trauma management.

To sum up FAST protocol use, in shocked trauma patient, if free fluid in peritoneal cavity is found, it is presumed to be blood and such patient is resuscitated and transported to operation theatre urgently. **Figure 3** shows an example of free fluid (blood) in Morrison’s pouch (hepatorenal space).



Figure 3: Free fluid in Morrison’s pouch.

**Second** clear and relatively easy application of POCUS is assessment of **AAA** (aneurysm of abdominal aorta). Ruptured AAA is almost uniformly rapidly fatal and only prompt recognition gives patients even slight chances for meaningful survival. Yet, sonographically, measurements of abdominal aorta are fairly easy to make; we measure diameter of infrarenal aortic section in antero-posterior plane transversely and AAA is defined as diameter >3 cm or 150% local bulging compared to the rest of vessel diameter. **Figure 4** shows an example of AAA measured by POCUS.



**Figure 4: AAA measured by POCUS.**

With US, we cannot establish whether AAA has actually ruptured, we only measure diameter of abdominal aorta, knowing that probability of rupture increases with diameter size and vice versa, abdominal aorta of normal diameter *de facto* has a zero chance of rupture, thus confirming normal abdominal aorta size excludes rupture from differential diagnosis of acute abdomen/shock/syncope/back pain. As said before, it is crucial to establish pretest probability of rupture based on history. As AAA are fairly prevalent (cca. 6% in general male population over 60 years of age), clinician must know how to treat a patient in which AAA is found. If based on history, ruptured AAA is a possible differential diagnosis, US image of aneurismatically dilated abdominal aorta confirms this clinical suspicion and patient is stabilised as per ABCDE approach and transferred to CT angiography if hemodynamically stable and straight to operation theatre if unstable/shocked.

ACEP (American College of Emergency Physicians) defines **11 core applications of emergency US** (4):

1. **Trauma,**
2. **Intrauterine pregnancy,**
3. **AAA,**
4. **Cardiac tract,**
5. **Biliary tract,**
6. **Urinary tract,**
7. **Deep venous thrombosis (DVT),**
8. **Soft-tissue/musculoskeletal guidance,**
9. **Thoracic guidance,**
10. **Ocular guidance,**
11. **Procedural guidance.**

In our workshop, we will cover five basic, yet very useful applications: in addition to previously mentioned **FAST** protocol for free-intraperitoneal fluid/blood and **AAA**, we will also learn how to diagnose/exclude **DVT**. Furthermore, we will learn about basic emergency **cardiac** US (FOCUS;

**Focused Cardiac UltraSound**) and basic applications of **lung** US (pneumothorax [PTx], fluidothorax [FTx], pulmonary consolidations (eg. pneumonia) and “interstitial syndrome” eg. pulmonary oedema).

Assessment for proximal **DVT** (which carries highest mortality due to possibility of trombo-embolisation) is fairly easy. Basic premise of sonographic proximal deep venous leg examination is to compress deep veins and thus exclude the DVT if vein walls are completely coaptable and vice-versa; if deep vein does not collapse under graded pressure, then there must be “something” inside (that is thrombus, of course). Many times, we cannot visualise actual thrombus in the vein, but fortunately, this is not necessary, as specificity of un-collapsible vein is so high, that it automatically means it is a DVT.

Focused basic assessment of **heart (FOCUS)** can be crucially important in critically ill. At the basic level, we aim to assess truly necessary, basic aspects of heart function, compressed in four sonographic questions regarding FOCUS:

1. Is there pericardial effusion/tamponade present? (**obstructive** shock?)
2. What is a diameter and collapsibility of inferior vena cava (IVC) as the marker of **fluid responsiveness (preload)**?
3. How does **left-ventricular function** [ $f(x)$ LV] look like by eyeballing (**inotropy**)?
4. Is there evidence of *acute* **right-ventricular strain/function** (pointing to acute **pulmonary embolism**)?

These four major questions at least partially address major parts of hemodynamic assessment and heart function, namely *preload*, *inotropy* (function of both ventricles) and possible obstruction to flow (tamponade). Third parameter of afterload (to the left ventricle) can be measured by blood pressure.

These four major questions are being addressed by viewing the heart from **four standard projections/sonographic windows** (parasternal long axis [PLAX] - **figure 5**, parasternal short axis [PSAX], subcostal view [SC], apical four chambers view [A4C]).

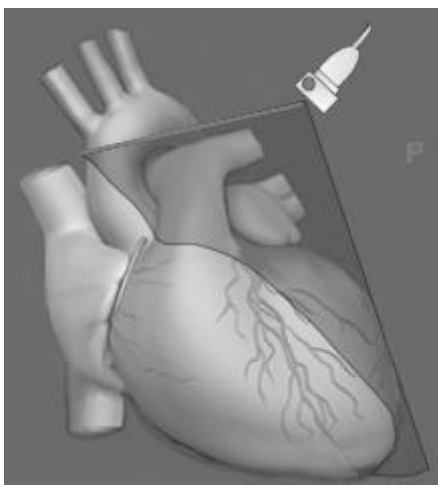


Figure 5: Parasternal long axis.

Aiming to answer four major hemodynamic questions above, we try to get the glimpse of facts regarding each question through each of the windows, thus all four standard cardiac windows are to be examined.

Similarly to four most critical questions to address cardiac function, we can systematise basic emergency **lung US** (LUS) use in four major pathologic patterns: pneumothorax [PTx], fluidothorax [FTx], pulmonary consolidations (e.g. pneumonia) and “interstitial syndrome” (e.g. pulmonary oedema).

Identification or exclusion of **PTx** can be fairly easy and quick, based on identification of **lung sliding**. Although air has historically been considered as “enemy of US waves”, doctor Lichtenstein has recognized, that although not visualising actual lung parenchyma (which totally scatter US beams), we can gain valuable information by deciphering different patterns of *artefacts* that are fairly specific for different pathologies.

**Lung sliding** is simple visualisation of visceral pleura sliding against parietal, thus giving appearance of motion. Since this motion artefact is dependent on absolute adherence of visceral pleura to parietal, visualising lung sliding very simply and confidently excludes PTx at that particular intercostal space! In contrast, finding specific sign of lung point confirms PTx and even approximates its size.

**FTx** can be identified with US very easily and much more accurately than on chest X-ray (CXR). **FTx** appears as anechogenic structure without structured borders. One cannot differentiate between transudate/exudate and haemothorax (HTx) conclusively; its presumptive diagnosis is based on clinical context (trauma? heart failure? signs of pneumonia?). Identification and localisation of FTx is especially useful to mark safest point for thoracocentesis.

**Consolidations** of lung parenchyma are mainly caused by lobar pneumonias or pressure/obstructive atelectasis. Lobar pneumonia exhibits sonographic pattern of isoechogenic structure (**figure 6**), not like liver or spleen, but with irregular borders that confluence into pattern of interstitial syndrome or normal pattern.



Figure 6: Sonographic pattern of isoechogenic structure.

**Interstitial syndrome**/pulmonary oedema is one of the latest and most exciting applications of LUS. It is classically taught that crackles, wheezing and rales cannot reliably differentiate between obstructive causes of dyspnoea (e.g. COPD and pulmonary oedema). On the other hand, with sonographic pattern of interstitial syndrome, we can reliably identify or exclude pulmonary oedema as a cause of dyspnoea. Lichtenstein et al. have recognized that with increasing interstitial oedema, normal pattern on lung US of horizontal (called “A”) lines, changes into vertical “B” lines.

Clinicians of many different specialties can learn to use any of these individual applications of **basic** lung, heart, abdominal and vascular ultrasound.

However, the major revolutionary impact of POCUS arises in sound use of different singular applications in problem-based, transversal assessment of patient in shock, respiratory failure, polytrauma, acute abdomen, etc. Many authors have proposed different protocols/algorithms and sequences to be used in different clinical scenarios.

Perhaps most clear example of such algorithmic approach is POCUS use in **cardiac arrest**. Every ALS provider is cognizant of “4H/4T” causes of cardiac arrest, but the fact remains, that it is very difficult to recognise cardiac tamponade, massive pulmonary embolism or tension PTx by clinical signs only. As written above, all these three causes are very easy to recognise by ultrasound.

After 25 years of pioneering work, Lichtenstein has condensed his concepts in landmark paper (5) and algorithm to address patients with **respiratory failure**, so called “BLUE” protocol (**figure 7**). It is easy, quick and reliable decision-making aid to identify critical causes of respiratory failure and can thus help getting proper treatment faster and more confidently.

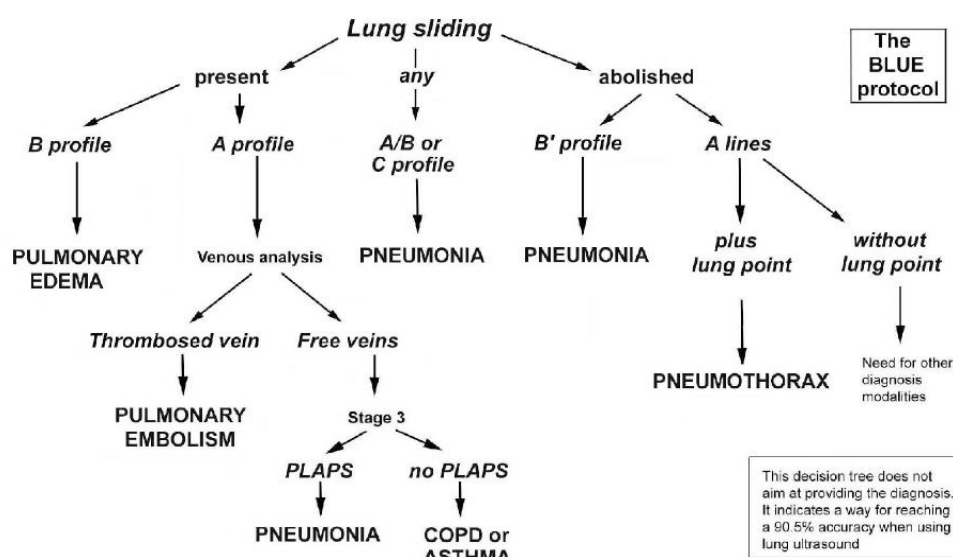


Figure 7: The BLUE protocol.

Perera et al. (6) have proposed another protocol called “RUSH” (Rapid Ultrasound in SHock), which systematically connects singular sonographic exams into quick and easy to comprehend sequence to identify major causes of shock: tension PTx, haemothorax, cardiogenic shock, massive pulmonary embolism, hypovolemia (IVC), hemoperitoneum (FAST), AAA and DVT.

POCUS in emergency medicine has become an indispensable tool in quality care for critically ill. There are many different US applications for singular pathologies, but true worth and synergy of POCUS can be harnessed when these singular “building blocks” are synthesised into quick and easy-to-use, problem-based, transversal protocols of use in respiratory failure/dyspnoea, shock, cardiac arrest, trauma and more.

## **1.4 Presentation of Simulation Scenario - Time Schedule**

<b>Part 1</b>	
00.00 – 00.40	Introduction to concept of POCUS and US technology (image acquisition, orientation artefacts).
00.40 – 01.00	Hands on training: getting to know the US machine
01.30 – 01.15	AAA lecture
00.15 – 01.30	Hands on training: AAA
01.30 – 01.45	DVT lecture
01.45 – 02.00	Hands on training: DVT
<b>Part 2</b>	
02.00 – 02.30	Lung US lecture
02.30 – 02.45	Hands on training: Lung US
02.45 – 03.10	FAST lecture
03.10 – 03.40	Hands on training: FAST
<b>Part 3</b>	
03.40 – 04.10	Focused cardiac US lecture
04.10 – 05.00	Hands on training: focused cardiac US
05.00 – 06.00	Putting it all together: case simulation and interpretation

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1. Moore CL, Copel JA. Point-of-care ultrasonography. NEJM 2011;364(8):749-57.
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## **SECTION 2: AIRWAY MANAGEMENT**

*Authors: Marko Zdravković, Mirt Kamenik, Dušan Mekiš*

<b>CONTENT</b>	
1.	<b>Theoretical Background</b>
2.	<b>Advanced Tracheal Intubation Techniques</b>
3.	<b>Presentation of Simulation Scenario – Time Schedule</b>

### **2.1 Theoretical Background**

#### **Introduction**

Maintaining a patent airway is crucial for human survival and is the first concern in many clinical situations, especially in medical emergencies with the well-known ABCDE approach in patient management. For ensuring the patient's safety, it is crucial to understand the airway anatomy, perform a (quick) airway assessment, be well trained in airway management and in using the available airway devices. These all are prerequisites to securing a patent airway in practice. One of the most stressful situations is when a patent airway is difficult to achieve by common measures and the ventilation of the patient is impossible (can't ventilate). If even intubation failed we have a can't ventilate-can't intubate situation, an even more drastic clinical scenario. More commonly ventilation is possible but definite airway management by intubation can not be achieved. In such cases it is critical to utilise the problem solving skills to achieve the desired outcome: patent airway, ventilation, oxygenation, and finally the definite airway secured from aspiration.

#### **Airway anatomy**

Having a solid understanding of the airway (functional) anatomy is crucial for every clinician performing airway management. The reader is advised to several online resources which visually present the anatomy of the airway:

- <http://www.youtube.com/watch?v=cK8fdHHyCPY>,
- <https://www.youtube.com/watch?v=wjRsa77u6OU>,
- <http://www.youtube.com/watch?v=DCVIEMNPe1E>,
- [http://www.youtube.com/watch?v=onbKgDn\\_maE](http://www.youtube.com/watch?v=onbKgDn_maE),
- <https://www.youtube.com/watch?v=fBHR1RjgLHA>.

In short, the upper airway consists of nasal and oral cavities, pharynx, larynx, trachea and principal bronchi. In normal breathing, air enters the nasal cavity where it is cleansed, heated and humidified (85% humidity, which increases to 95-100% in alveoli) by the cilia and the rich vasculature. Advancing to pharynx, which is in contrast to nasal cavities and larynx of soft tissue structure without cartilage or bony support, the airway continues to the larynx, which is composed of cartilages, muscles, ligaments and mucosa. Entry to the larynx is closed by epiglottis and for intubation purposes it is important to recognise the valleculae, space between the base of the tongue and the epiglottis. Visualisation of the rima glottidis by direct laryngoscopy is classified in Cormack-Lehane classification as: 1 - full visualisation of the larynx, 2 - partial, 3 - only epiglottis, 4 - no laryngeal structure is visible. For surface anatomical landmarks it is crucial to understand the location of the vocal cords behind



the thyroid cartilage and the location of cricothyroid membrane (usually around 9 mm in length). Trachea begins at the level of cricoid cartilage (C6) and continues for 9 - 15 cm before branching into principal bronchi.

### **Airway assessment**

By performing an airway assessment our goal is to identify individuals with a higher risk for difficult airway management, which would reflect in our approach in practice. We can define difficult airway management as a clinical situation in which an experienced operator has difficulties with larynx visualisation during laryngoscopy and/or endotracheal tube insertion (incidence of 0.13% - 18%) and/or bag-mask ventilation (incidence of 0.0001% - 0.02%). Incidence of failed intubation is reported to be 0.05% - 0.35%.

If the situation permits first take the history. One should ask about any past anaesthesia experience and any airway difficulties encountered, injury/neoplasm/surgery/radiotherapy of or around the upper airway, dentition/denture status, cervical spine injury/operation, rheumatological disease, temporomandibular joint mobility, pregnancy, stridor, dysphagia, obstructive sleep apnea and snoring. Also, the fasting period should be established.

While taking the history additional information can be obtained just by looking at the patient and listening to his/her voice: hoarseness, obesity, pregnancy, face shape, facial hair, visible changes on the head, neck and chest. Advancing the clinical examination, we also have available clinical measures/tests for predicting a difficult airway:

- poor flexion of the neck and/or poor extension of the head (< 35°),
- incisor distance of less than 3 cm,
- small mouth, big tongue and high arching palate,
- Mallampati score of 3 or more,
- poor mandibular protrusion (mandibular protrusion test),
- hyomental distance of less than 3 cm,
- thyromental distance of less than 6 cm,
- sternomental distance of less than 12.5 cm (objective measure of the neck length),
- body mass index > 25.

The sensitivity and specificity for difficult airway is very low for these indicators. However, combinations have a higher predictive value.

In emergency situations, where time is of an essence, a mnemonic LEMON has been established for a quick airway assessment:

- L: look externally for any physical cues (e.g. small jaw, facial hair, prominent/loose teeth, trauma, short neck, micrognathia, prior surgery etc.);
- E: evaluate using 3-3-2 rule (three fingers between incisors, three fingers between mentum of the mandible and the hyoid bone, two fingers between the hyoid bone and the thyroid cartilage);
- M: Mallampati classification;

- O: obstruction (soft tissue swelling, smoke inhalation, burns, broken neck, foreign bodies in the airway, trauma to the face or neck, excessive soft tissue from obesity, etc.);
- N: short and wide neck is not desirable, whereas neck mobility is a desirable setting.

There are many causes for difficult airway management, surely we can divide them into those related to the patient (congenital and acquired) and those related to the medical team (insufficient training in airway management techniques, poor airway assessment, lack of medical equipment or its malfunctioning). Physician taking care of the airway management should always be prepared for a difficult airway and have several alternative approaches in securing the airway at hand.

### Preparation for airway management

Operator and ideally the whole medical team taking care of the patient should always be prepared for difficulties in airway management and/or technical difficulties with the equipment. This includes sufficient training (knowledge and skills) as well as psychological preparedness. It is also better to have more falsely negative calls for help of more experienced operators than negative clinical outcomes. Also all the equipment should be at hand and prepared according to the local standards.

Patient positioning is important: sniffing position, Trendelenburg or anti-Trendelenburg. Consider manual in-line stabilization (MILS) technique for patients with suspected cervical spine injury and cricoid pressure application in non-fasted patients (more in rapid sequence induction section). Always employ proper patient monitoring, i.e. at least ECG, SpO<sub>2</sub>, blood pressure.

Before proceeding with airway management we have to render patient unconscious if he/she is still awake (unless awake fiberoptic intubation is indicated). Drugs used are preferentially short-acting with rapid onset of action and with known antagonists. This gives us an additional margin of safety as we have to avoid non-ventilation and non-oxygenation. Most common agents used are (Table 1): anaesthetics (midazolam, propofol, etomidate, ketamine, sevoflurane), opiate analgesic (e.g. remifentanyl, sufentanyl, fentanyl, alfentanil) and muscle relaxants (succinylcholine and rocuronium).

**Table 1: Commonly used drugs for airway management induction.**

agent	induction dose i.v.	agent	induction dose i.v.
midazolam	0.2 - 0.4 mg/kg	fentanyl	50 - 150 mcg
propofol	1,5 - 2,5 mg/kg	succinylcholine	1 - 2 mg/kg
etomidate	0.2 - 0.4 mg/kg	rocuronium	0.6 mg/kg
ketamine	1 - 2 mg/kg	remifentanyl	0.5 - 1.0 mcg/kg
alfentanil	10 - 20 mcg/kg	sufentanyl	10 - 20 mcg

### Basic airway management

In the unconscious patient the soft palate, base of the tongue and/or epiglottis can cause an airway obstruction at the level of the pharynx. Our approach is to establish the patent airway by either chin lift and head tilt manoeuvre, jaw thrust or the triple manoeuvre. In every situation also the presence

of a visible foreign body in the patient's mouth (e.g. chewing gum) should be ruled out and removed if present (by protected fingers, suction or Magill forceps).

If the patient is not breathing spontaneously, we should start with the ventilation of the lungs. By maintaining the head tilt and chin lift (use alternative technique in suspected cervical spine injury) we hold the face mask and ventilate the patient by using the hand bag. It is worth being aware of different modifications/adjuncts that can improve our efforts:

- oropharyngeal tube,
- nasopharyngeal tube (can cause bleeding from the upper airway and make the situation worse),
- two person technique (the more experienced one holds the face mask with both hands, the other ventilates the patient).

In practice, proper sizing of the adjuncts is very important and might be difficult for a novice to do so. Always consider proper face mask size and the level of its cuff inflation. Be careful not to press the mask against the eyes (vagal reflex, injury). Usually it is easier to ventilate with a face mask if dentures are still in place. On the other hand facial hair usually represents a more difficult situation. Always have a suctioning device ready and be aware of possibility of causing mandibular luxation during the face mask ventilation, especially in the elderly.

If we are successful in maintaining adequate ventilation, we buy ourselves time for a definite airway management. Here we can define adequate ventilation as a patent airway, face mask fit and seal, and effective ventilation with appropriate volume (5 -7 mL/kg) and frequency. Inspiration should take around one second (observe the chest rising) and always allow time for passive expiration. Be aware that the face mask is transparent, which enables you to observe the lips colour, presence of stomach contents, and water vapour (an additional indicator of effective ventilation).

### **Basic orotracheal intubation as a definite airway management**

Usually after bag-mask ventilation we proceed to definite airway management with orotracheal intubation. Direct laryngoscopy with intubation remains the golden standard for airway management as it enables not only ventilation and oxygenation but also secures the airway from aspiration. Since the 19th century, technological advances enabled us several approaches to intubation. Most commonly used is the MacIntosh laryngoscope and a properly sized endotracheal tube. There are also other laryngoscopes available (e.g. Miller with straight blades and McCoy laryngoscope) each with a spectrum of different blade sizes. As there are many devices available, one should be well trained in handling those that are available in your practice.

After choosing your laryngoscope it should be assembled and its proper function checked. Then a properly sized endotracheal tube should be selected, prepared (lubricated with local anaesthetic, cuff seal checked, 10 mL syringe, tube fixation tape) and adjuncts considered (e.g. different guide wires). Then you can approach the intubation. An assistant will hand you a laryngoscope while you maintain the head and neck in the sniffing position. With the right hand you open the patient's mouth (scissoring movement of thumb and index fingers) and carefully introduce the laryngoscope blade with the left hand in the right corner of the mouth. Then you displace the tongue to the left and keep advancing to the midline until you see the base of the tongue with epiglottis. Take care not

to compress the lower lip or upper teeth during this process. Then you pull the laryngoscope until the entry in the larynx is seen (Cormack-Lehane classification illustrates four different scenarios at this stage). A BURP (Backward, Up, Right-Pressure) manoeuvre on the thyroid cartilage can help us improve the larynx visibility. You proceed with a gentle endotracheal tube placement between the vocal cords. Do not advance too deep - the vocal cords should lie between the black marker lines on the tube (in any case, the cuff should be placed below the vocal cords). Remember the depth of insertion from the angle of the mouth. Remember that the first attempt at intubation is the best attempt as manipulation can cause oedema and haemorrhage from mucosa and lead to worse conditions during the next attempt (Table 2 lists some complications of endotracheal intubation). Assistant inflates the cuff and you use additional techniques to check the proper tube placement despite the most reliable one being the visual advancement of the tube through the vocal cords itself. We also observe chest movements, water vapour in the tube, auscultate the lungs and epigastrium, and if at hand observe the end tidal CO<sub>2</sub>. After proper placement is confirmed, the tube is secured and we continue with hand-bag or mechanical ventilation. We can summarise these steps as follows: patient assessment, positioning and monitoring; equipment and drug preparation; preoxygenation; induction; bag-mask ventilation; laryngoscopy; intubation and cuff inflation; proper tube placement confirmation; tube fixation; mode of ventilation selection.

**Table 2: Complications of endotracheal intubation.**

tube misplacement
damage to the: eyes, teeth, lips, tongue, soft palate, pharynx, vocal cords, trachea
cuff puncture and leakage
hypoxia, hypercapnia
aspiration of stomach contents, blood, part of a tumor
bronchospasm and laryngospasm
tachycardia / bradycardia / cardiac arrest
hypertension
myocardial ischemia

### Basics of mechanical ventilation

For use of any mechanical ventilator the most important technical issues to know are the on/off button, battery life, tidal volume setting (6-8 mL/kg), frequency setting (10-13/min) and the fresh gas flow with fraction of inspired oxygen selection. Additional feature might be anaesthetic gases selection (most commonly nitrous oxide and sevoflurane). If you are using oxygen tanks with limited quantity of gas, always check the pressure in the tank so you can establish when the reservoir will run out of oxygen. One can do so by using the following equation:  $pV/\text{flow} = t$ .

## I-gel

I-gel is the second generation supraglottic device available since 2007. In practice it has largely replaced the laryngeal mask and laryngeal tube. Consists of a breathing port, a smaller gastric port and has an anti-bite protection (newer version also has an oxygen port). Before its insertion, the device should be well lubricated, yet all larger pieces of the lubricant should be removed. I-gel is inserted in the sniffing position and usually does not require any additional manoeuvres than mouth opening (by pressing the chin down) for proper insertion. Fixation should be maxilla to maxilla. Through the breathing port also the endotracheal tube can be inserted (size 3 I-gel: 6 mm tube, size 4 I-gel: 7 mm tube, size 5 I-gel: 8 mm tube). Contraindication to I-gel use is a non-fasted patient. I-gel use is also discouraged in other conditions associated with an increased probability of aspiration (hiatus hernia, sepsis, diabetes mellitus, previous gastric surgery, obesity and pregnancy) or with pathology in pharyngeal/laryngeal structures. The peak airway pressures should not exceed 40 cm of H<sub>2</sub>O. More about the I-gel use is available at: <http://www.i-gel.com/igel-for-anaesthesia>.

## The vortex approach as a problem solving approach

The Vortex was designed by Nicholas Chrimes and Peter Fritz (<http://www.vortexapproach.com/Vortex Approach/Vortex.html>). It is a simple cognitive aid for managing an unexpected difficult airway situation (**Figure 8 and 9**). As it is very hard to recall complex existing algorithms in such stressful situation the Vortex actually emphasises simplicity of use and is designed to work in any setting. This is emphasised over anaesthesiology algorithms which can not be easily delivered in any setting as they are criticised of being too anaesthetist centred. The vortex also emphasises the importance of team work in managing an unexpected difficult airway situation. A shared mental model of difficult airway management within the team improves the effectiveness of management in crisis.

There are three basic airway management approaches, called non-surgical airway (NSA), surrounding the centred surgical airway in the vortex concept. One can start by either face mask ventilation, I-gel insertion or intubation to provide adequate ventilation and oxygenation. Then as one of these fails, despite suggested adjustments of the technique, one moves to another NSA technique. When all three fail despite taking into account possible adjustment of each technique (i.e. manipulation of the head and neck, use of adjuncts, size and type, suction, pharyngeal muscle tone) to deliver an optimal attempt of each NSA (we should strive that the second attempt at each NSA technique is the optimal one) then any further attempts will not change the outcome. The surgical airway is a reasonable next step, even if the SpO<sub>2</sub> is not failing yet. Actually the authors emphasise that the trigger for surgical airway is the inability to establish a patent airway following optimal attempts via each of the three NSA techniques and NOT the occurrence of oxygen desaturation. The concept of the green zone must also be emphasised. When by any of the NSA techniques we maintain proper ventilation and oxygenation we have time to think of the next steps and recruit additional personnel to the site. It is important to grasp, that every additional attempt may lead to can't ventilate can't oxygenate situation.

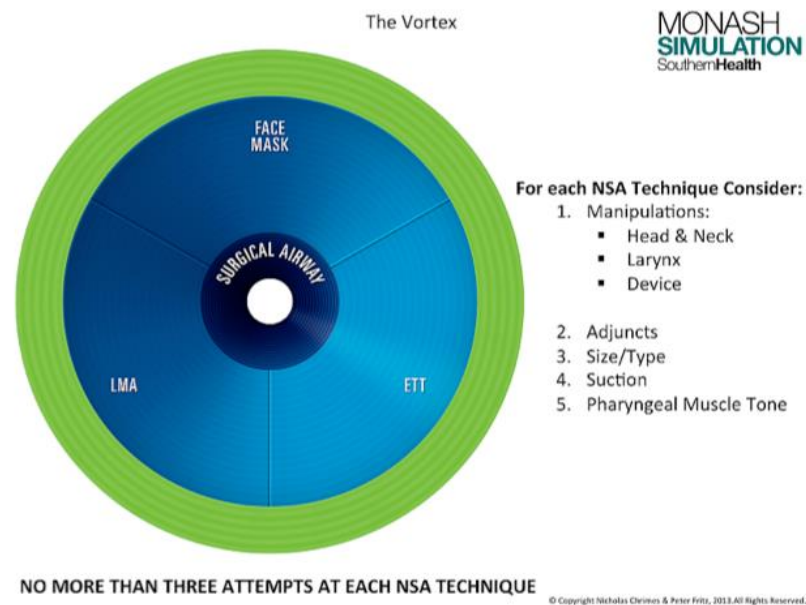


Figure 8: The Vortex approach.



**4 Step Approach to Failed Airway Management**

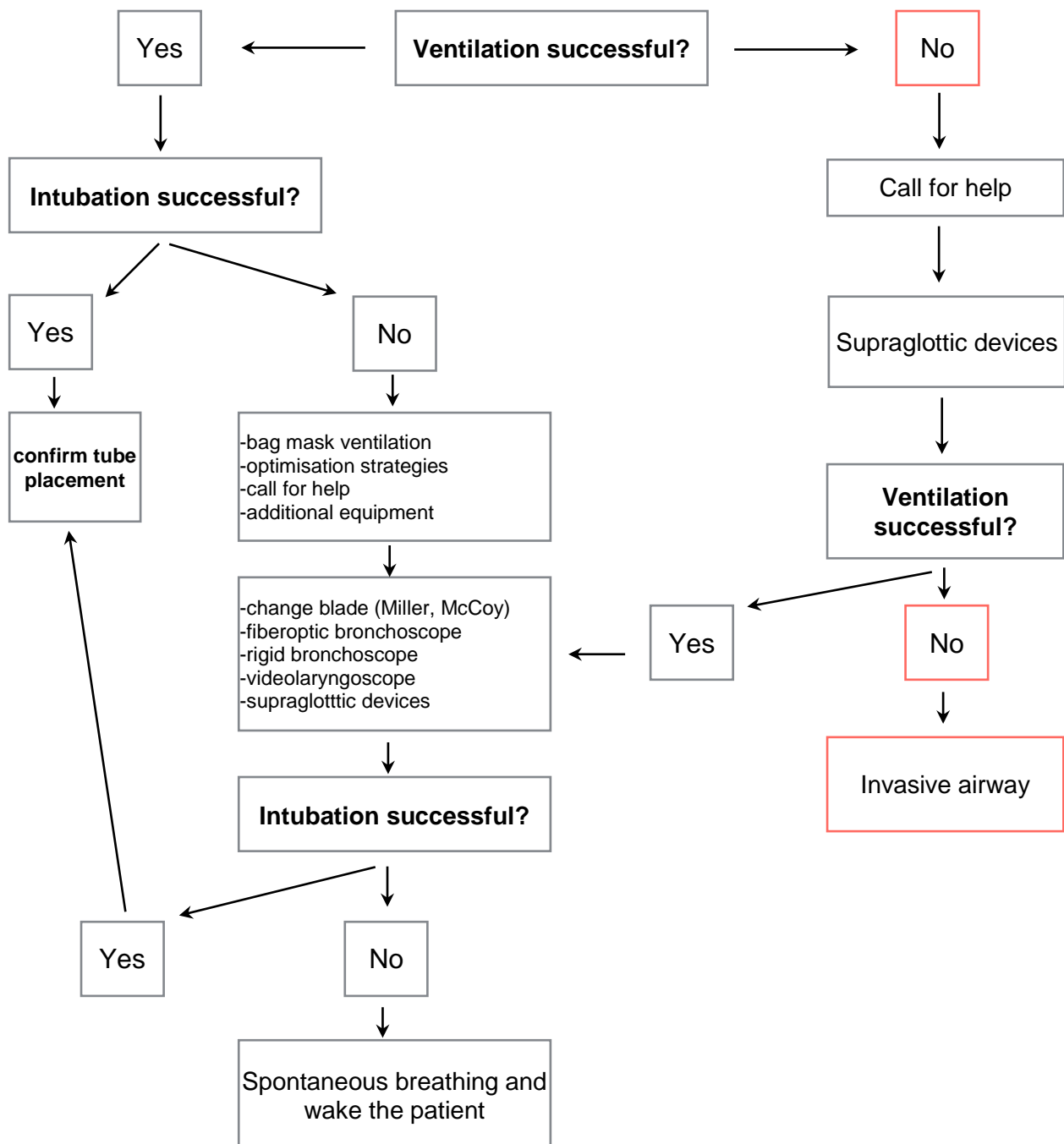
1. Optimal Attempts at Non-Surgical Airway
2. Emergency Surgical Airway
3. Green Zone: Optimise SaO<sub>2</sub>, Mobilise Resources, Consider Options
4. Definitive Airway

Figure 9: The Vortex approach.

**Anaesthesiology algorithms as a problem solving approach**

Two decision making points are crucial for difficult airway management. First, if a difficult airway is anticipated we can proceed to awake fiberoptic intubation or rarely for programmed surgical airway (tracheostomy) in local anaesthesia. Secondly, if a difficult airway was not anticipated and the patient is already anaesthetised, the most important question is whether ventilation is successful or not. The decision making tree hereafter is shown in algorithm below.

**Algorithm 1.** Unanticipated difficult airway algorithm at University Medical Centre, Department of Anaesthesia, Intensive Care and Pain Therapy, Maribor, Slovenia.



## **2.2 Advanced Tracheal Intubation Techniques**

**Airtraq.** Is a relatively simple form of video guided intubation technique. It improves the larynx visibility. At the tip of the Airtraq is a light source and optic system for picture generation. Device comes in four sizes and is for single use only. One decides upon the device size based on the tube needed to be placed in the patients trachea. Always check the device functionality before the

induction and turn the light on so that anti-fog system is fully activated (takes 30 - 60 sec). The battery life enables 40 min of light use. You have to lubricate the tube and properly place it in the side channel, so that tip is well aligned. Before introducing the Airtraq in the patients mouth you have to lubricate its surface (take care not to cover the lens with the gel). Device is introduced in the midline, pushing it against the hard palate - not the tongue and rotating up. Before it is vertical one uses the eye-piece to look at the airway anatomy. The tip should be placed either in the valleculae or grab the epiglottis (i.e. Miller technique). Then it is gently pulled up - vertically. We have to align the vocal cords with the centre of the image (twisting, rotation, forward/backward movement). Then we introduce the tube and observe its passage past the vocal cords. If we hit different laryngeal structures it is advised to withdraw the device and pull it vertically. We confirm tube placement via other means as well, and by lateral movement remove the device and the tube. Some clinical cases can be found at: [http://www.airtraq.com/index.php?option=com\\_content&task=view&id=234&Itemid=352](http://www.airtraq.com/index.php?option=com_content&task=view&id=234&Itemid=352). It is also possible to perform awake Airtraq guided intubation: <https://www.youtube.com/watch?v=iz6vhqQ9BVE>.

**Intubation laryngeal mask - Fastrach.** This mask designed for blind intubation (tube size 8) through its port. Before introducing the mask we have to lubricate its back side. Holding the rigid handle with one hand and lifting the lower jaw with the other we introduce the mask into the patients mouth, pressing against the hard palate and advancing into the pharynx. When it is placed the cuff should be inflated (not exceeding pressure of 60 cm H<sub>2</sub>O). Then the first Chandy manoeuvre follows to optimise mask position and ventilation. We gently rotate the mask in sagittal plane and leave it in the position which enables the best ventilation. The distal port of the mask is now best aligned with the vocal cords. Then the tube insertion follows. It is recommended to use the tube enclosed to the Fastrach mask which has special markers on its surface. On the mask is also a special device which lifts epiglottis as the tube is advanced through the mask. During the tube insertion it is recommended to use the second Chandy manoeuvre. As the tube is advanced, there might be resistance encountered. We use a special marking on the tube to ascertain what is the problem. This marking is placed at a distance at which the tube exits the distal port of the mask. Now if the resistance is encountered around 2 cm after the marking, we withdraw the tube to the marking followed by 6 cm withdrawal of the mask and retry the tube introduction. If the resistance is before or after the 2 cm from the marking, this means we have used an inappropriate size of the mask. There are three sizes of Fastrach available: size 3 (30 - 50 kg), size 4 (50 - 70 kg) and size 5 (70 - 100 kg). When we have confirmed the tracheal tube placement, it is recommended to withdraw the Fastrach mask as it exerts a high pressure on the pharyngeal mucosa. This is done by special obturator enclosed to the mask. We should pay attention not to dislocate the tube or not to damage its cuff inflation system. There are also other options, i.e. to use the intubation bougie (<https://www.youtube.com/watch?v=E7Lo1JD2Brk>) or Trachlight (<https://www.youtube.com/watch?v=rH8JPPz8hMQ>) over which we can introduce the tube or even fiberoptic intubation over the Fastrach mask.

**Nasal intubation.** This technique can be performed either blindly or with the use of laryngoscope/bronchoscope. Blind variation does not require any neck movement or any mouth opening. It has been reported to be successful in 92% of the emergency medicine departments. Three major contraindications are: coagulation disorders, skull base fractures, severe facial injuries. Common complications are: bleeding, laceration of the mucosa of the nose, pharyngeal injuries. One



lubricates inside of the both nostrils and checks which one is wider (awake patient can also tell us). The tip of the tube is turned backwards and to the septum. The most painful part is tube introduction in the oropharynx. Then as we approach the vocal cords the breathing sounds get louder. During the inspiration the tube is gently pushed in the trachea. The first attempt is successful in around 50%. Commonly the tube can hit epiglottis, valleculae, arytenoid cartilage, the cords itself, sinus piriformis and naturally oesophagus. Another technique is to use the nasal intubation in the unconscious patient. By using the laryngoscope one can observe the tip of the tube in the pharynx and visualise the glottic opening. Then by using the Magill forceps the tube can be placed in the trachea.

**Invasive airway management.** Surgical airway is one of the last resorts to establish a patent airway. There are several ways of performing the invasive airway. One is needle cricothyrotomy: using 5 mL syringe and a 12-14 gauge needle one puncture the cricothyroid membrane. Then there is a tube connect that can be inserted in the syringe after removing the plunger and via that can be limited high pressure ventilation established. Another way is cricothyrotomy using a scalpel where a vertical 1 cm incision is done between the thyroid cartilage and cricoid. Then one can cut the cricothyroid membrane horizontally and place any tube in the trachea. There are also different kits available (employing Seldinger technique) so it is advisable to get familiar with the one available at your institution. Some complications of surgical airway management are: bleeding, pneumothorax, subcutaneous emphysema, injury of larynx/trachea and oesophageal puncture. Also consider the tube obstruction with blood or mucus which can affect patient ventilation.

### **The fasted/non-fasted patient and rapid sequence intubation (RSI)**

When considering airway management the first check point should be the fasting status of the patient. If he/she ingested food in the last 6 hours the RSI is indicated. This technique aims to shorten the time from the absence of upper airway reflexes to endotracheal tube placement with cuff inflated. This is the period during which the stomach contents aspiration has the highest probability. RSI can be divided into 7 consecutive steps: preparation (t - 10 min), preoxygenation (t - 5 min), premedication (t - 3 min), paralysis with induction (t), protection (t + 30 s), placement (t + 45 s), postintubation management (t + 60 s).

**Preparation:** All the necessary equipment and drugs are prepared (do not forget to keep the suctioning device near and ready to use). Patient monitoring is placed (ECG, SpO<sub>2</sub>, blood pressure) and the i.v. access is essential. If circumstances permit a placement of nasogastric tube is encouraged and stomach emptied. Also the patient should be positioned in the Trendelenburg position (although some controversy exists in the literature).

**Preoxygenation:** with 100% oxygen at least 5 minutes (or 8 deep breaths).

**Premedication:** with fentanyl 1-2 mcg/kg or sufentanil 0.05-0.1 mcg/kg.

**Paralysis with induction:** sedative (propofol/etomidate/tiopental/ketamine), muscle relaxant (rocuronium 1.2 mg/kg or succinylcholine 1-2 mg/kg).

**Protection:** After the loss of consciousness - cricoid pressure should be applied (3 kg = 30 N; not too strong not too weak). Wait for 30 seconds and get prepared for the next step.

**Placement:** intubation in 15 - 20 seconds with immediate cuff inflation (before the attempt the lower jaw should be flaccid). If unsuccessful - gentle bag mask ventilation with cricoid pressure maintained.

**Postintubation management:** after the tube placement is confirmed the cricoid pressure is released. The tube is firmly attached.

In addition to the fasting status, there are also other indications for RSI e.g. patients with ileus, poisoning etc. In any case, if you are unsuccessful with the RSI you should immediately shift to the alternative airway management techniques to maintain proper oxygenation and provide definite airway as soon as possible (e.g. bag mask ventilation, l-gel, Airtraq, video laryngoscopy, and even invasive/surgical airway if needed). And always be prepared for alternative techniques when initiating the RSI.

### **Airway management in children**

When considering airway management in children similar techniques and approaches apply as in adults. However, special circumstances related to growth and development have to be taken into account as well as medication adjustments. In children most of the life threatening situations are of respiratory origin. Also 80% of all cardiac arrests are primarily caused by respiratory arrest. There are also anatomical airway differences between children and adults. The larynx lies more superior and anterior, they have a larger tongue relative to the mouth, epiglottis is bigger and differently shaped, the narrowest airway point is at the level of cricoid cartilage, they have big occiput and the vocal cords are angled (the tube might strike in the anterior commissure). We also have to be aware that basal metabolic needs in children are bigger - 6mL/kg/min - twice that of an adult and that the functional residual capacity is lower and the hypoxia develops faster (shorter time for intubation). In premedication atropin is suggested (0.02 mg/kg - minimal dose of 0,1 mg). Due to all of these reasons it is especially mandatory for health care professionals to have sufficient training in airway management in children.

In order to optimally place the paediatric patient for airway management we have to align the external acoustic meatus with the shoulders height. In infants we have to rise the chest with some soft clothing. Younger children need no such manipulation and in older ones we prepare in the same way as in adults (pillow). Preoxygenation and bag mask ventilation are performed in such positions with lifting the lower jaw and with oropharyngeal airway device if needed. Have in mind that hypothermia is rapidly developed in younger children. We use the Miller blade for laryngoscopy in children younger than 4 years and MacIntosh blade in older. The size of the tube can be selected by comparing its tip to the child's little finger or calculated by using the following formula (after one year of age):  $\text{age in years}/4 + 3$  for the cuffed endotracheal tube which is slowly substituting the uncuffed one in practice (as technical advances in tube production have allowed for better fit for children). Always prepare also one size smaller and one size bigger tube. For neonates we use tubes of size 2.5 - 3.0, infants up to 6 months of age 3.0 - 3.5 and from 6 to 12 months 3.5 - 4.0. Always be aware that head manipulations can dislodge the tube so repeatedly assess its position. And learn how to manage laryngospasm in children.

## Conclusion

Importance of airway management can not be overemphasised. Even in well controlled setting, i.e. operating room planned anaesthesia setting, the airway complications are the leading cause of death. It is very important for healthcare providers to be well trained in different approaches in airway management which is also the intent of this workshop. But always remember, that failed intubation does not lead to death, but improper ventilation and oxygenation do.

## **2.3 Presentation of the Simulation Scenario - Time Schedule**

<b>Part 1</b>	
00.00 – 00.15	Airway assessment
00.15 – 00.45	Basic airway management
00.45 – 01.15	Basic tracheal intubation
01.15 – 01.30	Basics of mechanical ventilation
01.30 – 01.45	I-gel
01.45 – 02.20	Vortex approach - 2 clinical scenarios
02.20 – 02.30	Break
02.30 – 03.30	Advanced tracheal intubation techniques: Airtraq, video laryngoscopy, fiberoptic intubation
<b>Part 2</b>	
00.00 – 01.00	Advanced tracheal intubation techniques: Fastrach, nasal intubation, invasive airway
01.00 – 01.30	Airway management in children
01.30 – 02.30	Scenarios on HPS (Human patient simulator): RSI, difficult airway management

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## **SECTION 3: ACUTE CORONARY SYNDROME AND ITS COMPLICATIONS**

*Authors: Nina Hojs, Tadej Zorman, Sebastjan Bevc*

CONTENT	
	1. Theoretical Background of Acute Coronary Syndrome
	2. Theoretical Background of Arrhythmias
3.	ECG Strips of Important Arrhythmias to be Recognized on Monitor
4.	Presentation of the Human Patient Simulator
5.	Presentation of Simulation Scenario – Time Schedule
6.	Acute Coronary Syndrome and Arrhythmia Management Algorithms

### **3.1 Theoretical Background of Acute Coronary Syndrome**

#### **Definition**

ACS is a group of clinical syndromes caused by coronary artery obstruction leading to acute myocardial ischemia. In the clinical setting ACS includes UAP, acute MI with ST-elevation (STEMI) or without it (NSTEMI) and sudden cardiac arrest. Acute MI is accompanied with elevated plasma concentrations of cardiac enzymes (troponin I or T, creatinine-kinase-MB), whereas in UAP those markers remain within normal ranges.

Regarding ECG changes two categories of patients are recognised:

- Patients with acute chest pain and persistent (>20 min) ST-segment elevation or new left bundle branch block (LBBB). This is termed ST-elevation ACS (STE-ACS) and generally reflects an acute total coronary occlusion.
- Patients with acute chest pain but without persistent ST-segment elevation; this is termed non-ST-elevation ACS (NSTEMI-ACS) and based on the measurement of troponins, will be further qualified as non-ST-elevation MI (NSTEMI) or UAP.

#### **Pathogenesis**

ACS is caused primarily by atherosclerosis of coronary arteries. Most cases of ACS occur from a rupture or erosion of a hemodynamically insignificant atherosclerotic plaque leading to platelet activation and aggregation, activation of the coagulation pathway and vasoconstriction. This process results in localised coronary intraluminal thrombosis and variable degrees of vascular occlusion, distal embolization may also occur. Consequently, coronary blood flow is reduced and oxygen and nutrition supply to the myocardium diminished. If oxygen demand exceeds oxygen supply, myocardial ischemia occurs and if being prolonged (more than 20-30 minutes), it causes myocyte necrosis (i.e. MI), accompanied with a rise of myocardial necrosis biomarkers.

Ischemia is associated with metabolic, mechanical and electrical changes in the affected myocardium leading to various ST segment and T wave abnormalities seen in the ECG, hemodynamic changes and arrhythmogenicity.

Stable CAD in the absence of plaque disruption may also result in ACS when physiologic stress (eg. trauma, anaemia, infection, tachyarrhythmia) increases demands on the heart.

Non-atherosclerotic causes of ACS are coronary occlusion secondary to vasculitis, coronary anomalies (congenital, aneurysm), coronary trauma, primary coronary vasospasm (variant angina), drug use (cocaine, amphetamines), hypoxia (carbon monoxide poisoning, acute pulmonary disorders), coronary artery embolism (secondary to cholesterol, air, sepsis), aortic dissection with retrograde involvement of the coronary arteries etc.

The severity and duration of coronary arterial obstruction, the volume of myocardium affected, the level of demand on the heart and the ability of the rest of the heart to compensate are major determinants of a patient's clinical presentation and outcome.

### **Signs and Symptoms**

The clinical presentation of ACS encompasses a wide variety of symptoms but the leading one is typically chest pain.

Patients experience retrosternal pressure or heaviness («angina») radiating to the left arm, neck, or jaw, which may be intermittent (usually lasting for several minutes) or persistent. These complaints may be accompanied by other symptoms such as diaphoresis (sweating), nausea, abdominal pain, dyspnoea, and syncope. However, atypical presentations are not uncommon. These include epigastric pain, indigestion, stabbing chest pain, chest pain with some pleuritic features, or increasing dyspnoea. Atypical complaints are more often observed in older (over 75 years) patients, in women, and in patients with diabetes, chronic renal failure, or dementia. Patient history and examinations are important to recognise other causes of chest pain.

Physical examination results are frequently normal in patients with ACS. If chest pain is ongoing, the patient will usually lie quietly in bed and may appear anxious, diaphoretic, and pale. Physical findings can vary from normal to any of the following – hypo- or hypertension, diaphoresis, tachycardia or bradycardia, cardiac murmurs, 3<sup>rd</sup> or 4<sup>th</sup> heart sound, pulmonary oedema and other signs of left heart failure, jugular venous distension and cool, clammy skin and diaphoresis in patients with cardiogenic shock.

### **Diagnostic Workup**

Beside patient history and physical examination, ACS workup requires at least a 12-lead ECG and laboratory studies (cardiac enzymes, complete blood count, basic metabolic profile, serum lipids, C-reactive protein). Further imaging techniques may be needed, i.e., echocardiography, chest x-ray, coronary scintigraphy, angiography, etc.

### **Treatment**

Initial treatment for ACS regardless of ECG changes should focus on stabilising the patient's respiratory and hemodynamic condition, relieving ischemic pain, and providing antithrombotic therapy to reduce myocardial damage and prevent further ischemia. The immediate general initial therapy of ACS includes antiplatelet therapy with aspirin, antiischemic therapy with nitroglycerine, oxygen to achieve arterial blood oxygen saturation of 94-98 % and pain relief with intravenous

morphine. Further management is based on ECG changes, patient's condition and risk stratification (**Algorithm 1**).

Beside initial therapy, every patient requires additional antiplatelet therapy (ticagrelor, prasugrel or clopidogrel) for 12 months, anticoagulant therapy (unfractionated heparin (UF), low molecular weight heparin, bivalirudin or fondaparinux) temporarily and antiischemic therapy ( $\beta$ -blockers). Angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) are given in case of heart failure or decreased left ventricular ejection fraction. Also important is secondary prevention with serum lipid lowering therapy (statins) and lifestyle changes (smoking cessation, regular physical activity, mediterranean diet). Other conditions, like arterial hypertension or diabetes mellitus, must be controlled and treated.

In general, patients with ACS can be treated either with invasive mechanical reperfusion treatment (primary percutaneous coronary intervention (PPCI), surgery) or with non-invasive pharmacological reperfusion treatment (fibrinolysis) or entirely conservative (dual antiplatelet, antiischemic and anticoagulant therapy) without reperfusion therapy. The aim of mechanical or pharmacological reperfusion is to recanalize infarct-related arteries to limit the extent of myocardial infarction and to salvage jeopardized ischemic myocardium.

Patients with STE-ACS or new LBBB should be treated with reperfusion therapy within 12 hours of symptom onset, preferably with PPCI. Patients with NSTEMI-ACS should first get maximum conservative therapy but in case of a high or intermediate risk of ischaemic events (GRACE score), dynamic ST segment and T wave changes, relevant rise or fall in troponin, worsening or on-going symptoms, deterioration with acute heart failure, hemodynamic instability or ventricular arrhythmias, reperfusion therapy should be initiated.

## Differential Diagnosis

### Chest pain:

Reflux esophagitis, oesophageal spasm, peptic ulcer, cholecystitis, pancreatitis, pulmonary embolism and infarction, pneumothorax, aortic dissection, pericarditis, pneumonia, pleuritis, costochondritis, rib fracture, cervical discopathy, muscle injury/inflammation, early herpes zoster, depression, anxiety, etc.

## Complications of ACS

Myocardial ischemia and/or infarct can lead to:

- cardiac pump failure with the development of acute heart failure ranging from mild (Killip class II) to severe (Killip class III) and shock (Killip class IV),
- mechanical complications with cardiac rupture (acute and subacute free wall rupture, ventricular septal rupture), mitral regurgitation and left ventricular aneurysm, and
- **arrhythmias** and conduction disturbances with ventricular arrhythmias (ventricular tachycardia, ventricular fibrillation, ventricular ectopic rhythms), supraventricular arrhythmias (mostly atrial fibrillation) and sinus bradycardia or heart block (AV block I-III degree).

## **3.2 Theoretical Background of Arrhythmias**

### **Definition**

Arrhythmia is an abnormality in heart rate and/or rhythm. Arrhythmia is every heart rhythm different from a sinus rhythm with a frequency of 60-100/min.

We can divide arrhythmias according to:

- heart rate: bradycardias (< 60/min) and tachycardias (> 100/min),
- rhythm: regular (e.g. sinus and ventricular tachycardia) and irregular (e.g. atrial fibrillation),
- source (origo): supraventricular (narrow QRS complex) and ventricular (broad QRS complex).

Cardiac arrest rhythms are pulseless ventricular tachycardia (VT), ventricular fibrillation (VF), pulseless electrical activity (PEA) and asystole.

### **Etiology**

Causes of arrhythmias are physiological (physical activity, respiratory arrhythmia), cardiovascular (coronary heart disease, myocardial infarction, myocarditis, cardiomyopathy), congenital (long QT syndrome, Brugada syndrome), metabolic (hypo/hyperthyrosis), electrolyte disorders, hypoxia, hypothermia, infections, drugs, abuse of substances (nicotine, cocaine, alcohol, coffee), etc.

### **Clinical Manifestations**

Arrhythmias can be asymptomatic or present with palpitations or signs and symptoms of hemodynamic compromise (dizziness, weakness, fainting or nearly fainting, heart failure, confusion, dyspnea, chest pain). Most dramatic presentation is with cardiac arrest.

### **Approach to the Patient**

Put the patient in a supine position or sitting position under an angle of 45°. Always use the ABCDE approach (Airway, Breathing, Circulation, Disability, Exposure) to assess the patient. Ensure oxygen given (if SpO<sub>2</sub> < 94 %), obtain an intravascular (i.v.) access, draw blood for laboratory tests, monitor SpO<sub>2</sub>, ECG, blood pressure, record a 12 lead ECG. Identify and treat reversible causes (4H – hypoxia, hypovolemia, hypothermia, hypo/hyperkalemia and other electrolyte and metabolic disturbances; 4T – tension pneumothorax, thrombosis, cardiac tamponade, toxins).

ECG is an important tool in arrhythmia recognition; therefore always use a structured approach to interpreting an ECG recording:

- Is there any electrical activity?
- What is the ventricular (QRS) rate?
- Is the QRS complex width normal or prolonged?
- Is the QRS rhythm regular or irregular?
- Is atrial activity present?
- Is atrial activity related to ventricular activity and, if so, how?



## Treatment

Arrhythmia treatment depends on the nature of the arrhythmia and the condition of the patient (adverse features). Adverse features are shock (hypotension, pallor, sweating, cold extremities, impaired consciousness), syncope, heart failure, myocardial ischemia (typical chest pain and/or typical ECG changes), extremes of heart rate (> 150/min, < 40/min).

Arrhythmia treatment can include:

- nothing,
- simple clinical interventions (vagal manoeuvres, fist pacing, cardiopulmonary resuscitation),
- drugs,
- electrical current (synchronised electrical cardioversion, pacing, defibrillation),
- catheter ablation,
- implantation of a pacemaker or implantable cardioverter-defibrillator (ICD).

## Bradycardias

Management of bradycardias depends on the presence of adverse features (**algorithm 2**). If the patient has adverse features, initial treatment is usually pharmacological. Give atropine 0.5 mg i.v. if necessary; repeat every 3-5 min to a total of 3 mg. Use cautiously in the presence of acute myocardial ischemia or infarction. Second-line drugs are adrenaline, isoprenaline, dopamine, glucagon (if beta-blocker or calcium channel overdose).

If drugs are ineffective in a patient with adverse features, use transcutaneous pacing. As an interim measure fist pacing can be performed by serial rhythmic blows with a closed fist over the lower edge of the sternum at a rate 50-70/min. For transcutaneous pacing apply electrode pads in the right pectoral-apical position. Select an appropriate pacing rate (60-90/min) and increase the current delivered (usually 50-100 mA, max. 200 mA) until electrical capture and palpable pulse is achieved. Since pacing is painful, use i.v. analgesia and sedation (midazolam-fentanyl). Contact a specialist for temporary intravenous pacing.

## Tachycardias

Management of tachycardias also depends on the presence of adverse features (**algorithm 3**). If they are present, synchronised cardioversion is used. Give conscious patients i.v. analgesia and sedation. Apply pads in the right pectoral-apical position. Switch on the synchronisation mode on the defibrillator. Choose shock energy according to type of defibrillator (mono/biphasic) and type of arrhythmia:

- broad-complex tachycardia or atrial fibrillation: 120-150 J biphasic shock or 200 J monophasic shock,
- narrow-complex tachycardia or atrial flutter: 70-120 J biphasic shock or 100 J monophasic shock.

Increase energy in increments, if this shock fails. Be aware, that there might be a slight delay before the shock is delivered. Always be careful performing synchronised cardioversion. While the defibrillator is charging, warn all rescuers (Stand clear!), remove oxygen delivery devices. If a second

shock is needed, reactivate the synchronisation switch if necessary. If electrical cardioversion fails, give amiodarone 300 mg i.v. over 10-20 min and attempt further synchronised cardioversions. Later you can give amiodarone 900 mg i.v. over 24 hours. Amiodarone is always given in 5 % glucose; an important acute side effect is hypotension.

If adverse features are not present in a patient with tachycardia, further management (vagal manoeuvres, drugs) depends on the type of arrhythmia (**algorithm 3**).

### **3.3 ECG strips of Important Arrhythmias to be Recognized on Monitor**



Figure 10: Ventricular tachycardia



Figure 11: Fine ventricular fibrillation.



Figure 12: Asystole.

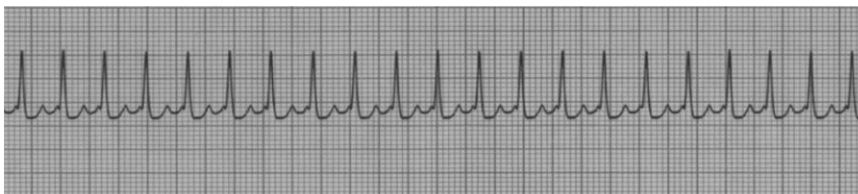


Figure 13: Supraventricular tachycardia.



Figure 14: Atrial flutter.



Figure 15: Atrial fibrillation.

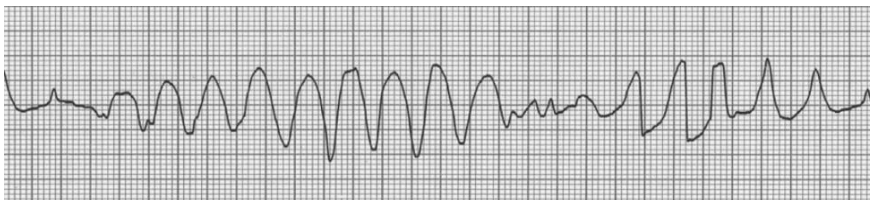


Figure 16: Polymorphic ventricular tachycardia - torsades de pointes.

### **3.4 Presentation of the Human Patient Simulator**

Human Patient Simulator (HPS) is the golden standard for medical training in simulations. HPS is an automated simulator mainly designed for training of emergency situations, anaesthesia and trauma. The simulator allows the exchange of respiratory gases, application of anaesthesia and real-time monitoring of the patient-model with physiologically-clinical devices. HPS possess an unique mathematically-technical model of the human physiology, which includes cardiac, respiratory, neurological and pharmacological components.

On the HPS, both uncomplicated clinical skills, as well as complex pathological scenarios from fields of internal medicine, reanimation procedures, anaesthesia, cardiology, neurology, trauma and pharmacology can be trained.

The simulator is suitable for administration of various drugs. By scanning bar codes, the physiological effect of various drugs can be studied. Simulation practice on a HPS is an opportunity for students, allowing them an overall treatment of a patient-model, emphasizing team work, thinking (clinical reasoning and decision making), learning from their own mistakes and allows repetition, result analysis and gaining experience without harmful effects for the patient.



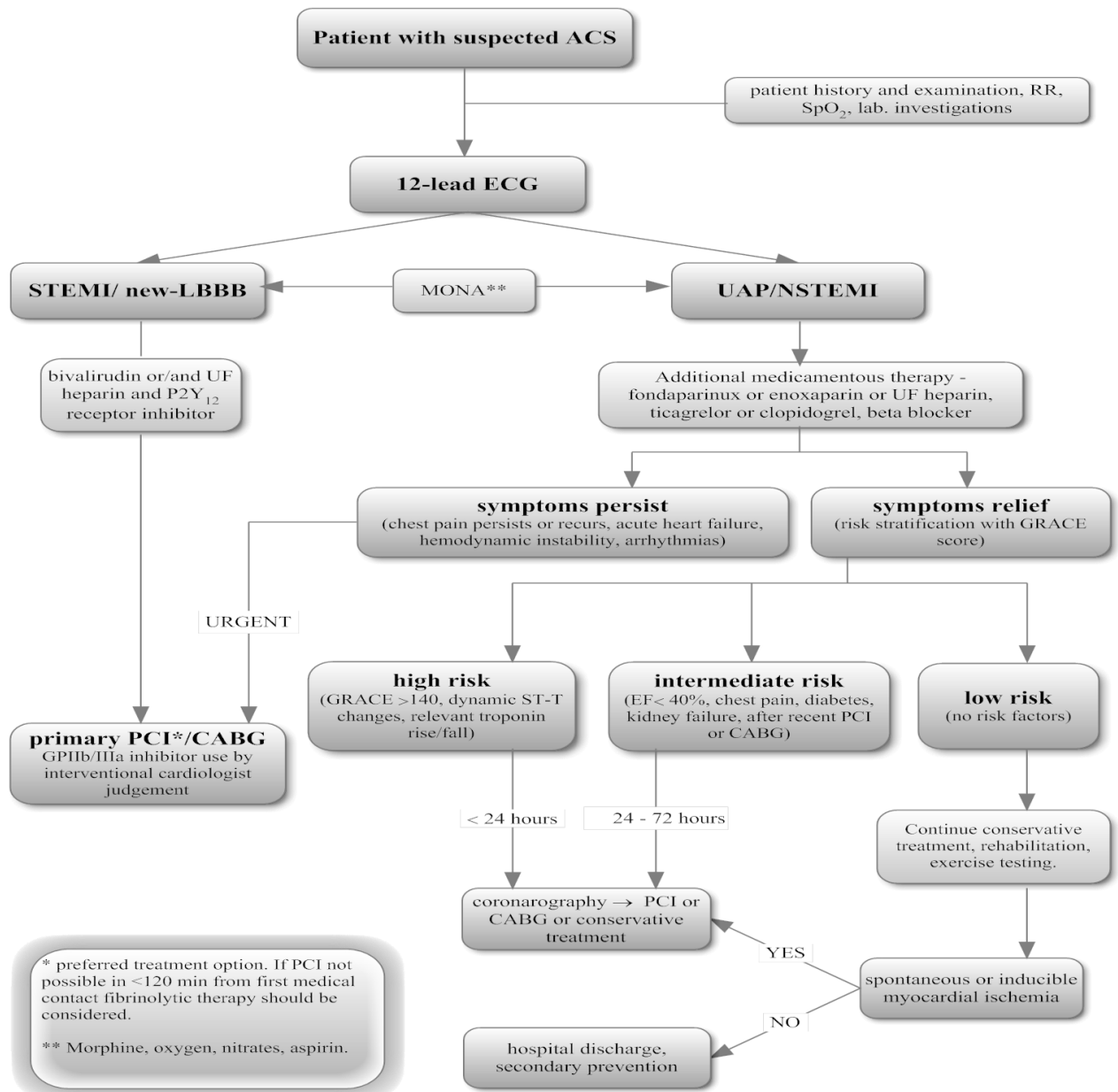
Figure 17: Human Patient Simulator in Action

### **3.5 Presentation of Simulation Scenario - Time Schedule**

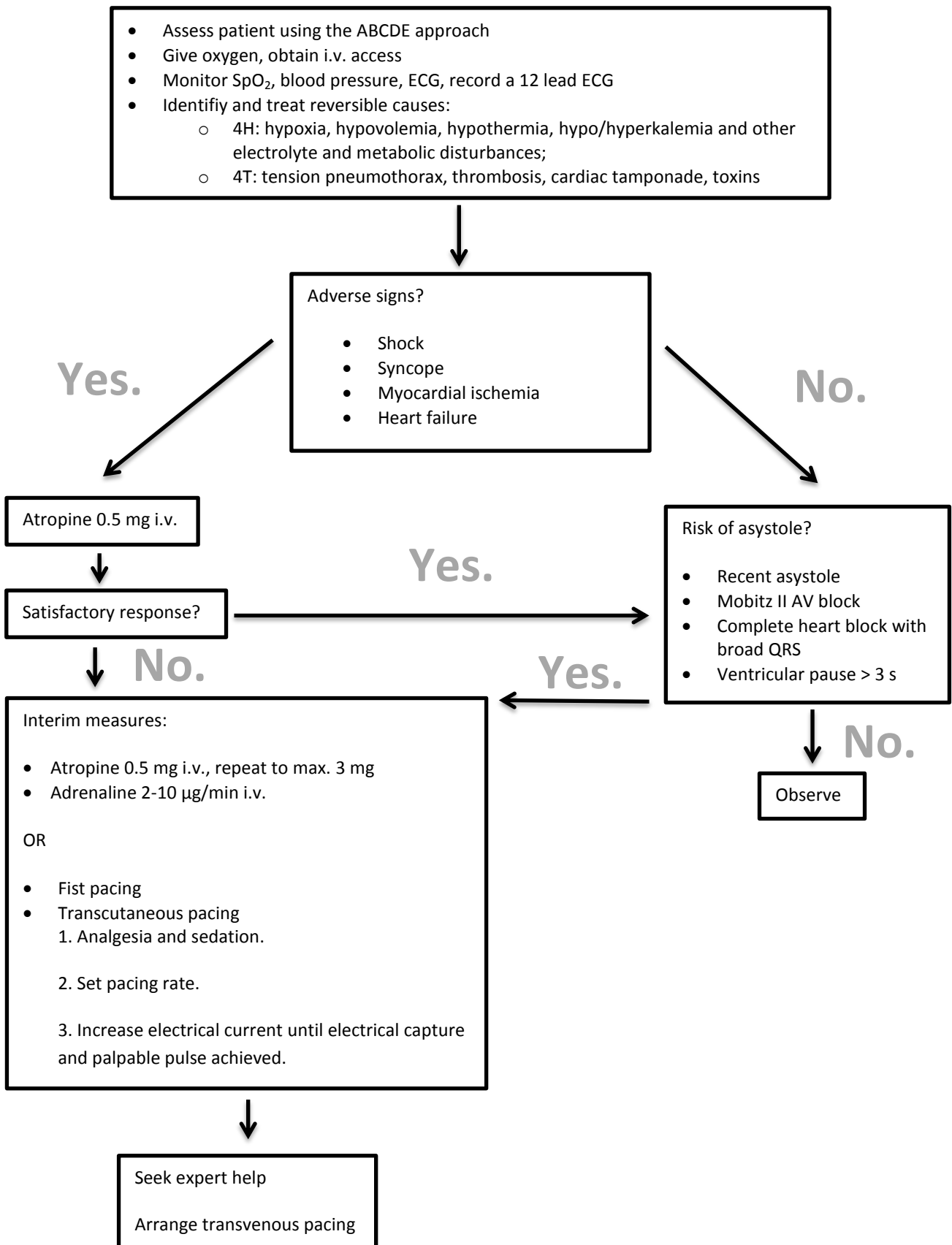
<b>Part 1</b>	
00.00 – 00.15	Acquaintance with the HPS
00.15 – 00.30	Acquaintance with components of the HPS monitoring (blood pressure measurements, pulse oximetry, ECG)
00.30 – 01.00	Acquaintance with medical equipment and materials (oxygen masks, blood pressure device, finger pulse oximeter, drugs, scanning bar codes, syringes, infusion sets, infusion pumps, intubation sets, defibrillator)
01.00 – 01.45	Acquaintance with drugs needed for the simulation scenario
01.45 – 02.00	Pop quiz about ACS knowledge
02.00 – 03.00	Performance of ACS simulation scenario: presentation of clinical case and practical training
03.00 – 03.30	Debriefing and evaluation
<b>Part 2</b>	
03.30 – 04.00	Acquaintance with drugs needed for the simulation scenario
04.00 – 04.30	Pop quiz about arrhythmias knowledge
04.30 – 05.30	Performance of arrhythmias simulation scenario: presentation of clinical case and practical training
05.30 – 06.00	Debriefing and evaluation

### 3.6 Acute Coronary Syndrome and Arrhythmias Management Algorithms

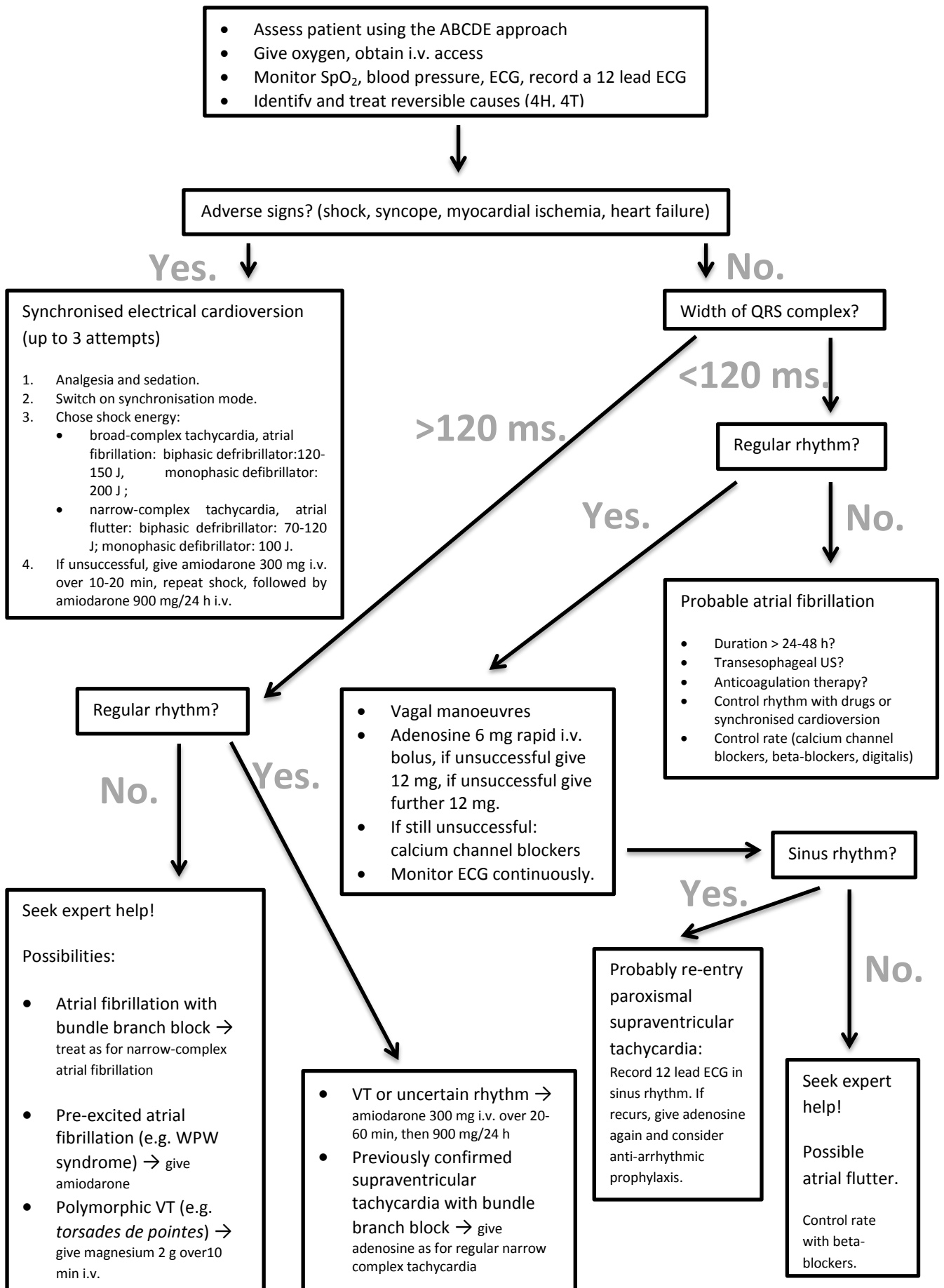
Algorithm 1. Acute coronary syndrome algorithm.



**Algorithm 2. Bradycardia algorithm.**



**Algorithm 3. Tachycardia algorithm.**



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## **SECTION 4: ADVANCED CARDIAC LIFE SUPPORT** **(ACLS)**

*Authors: Alenka Strdin Košir, Andrej Markota*

CONTENT	
1.	Theoretical Background
2.	Advanced Cardiac Life Support
3.	Managing the Peri-arrest Patient
4.	Presentation of Simulation Scenario – Time Schedule
5.	Advanced Cardiac Life Support Basic Take Home Messages

### **4.1 Theoretical Background**

Survival after cardiac arrest with good neurological outcome is about 5-10%. Poor survival, high incidence (about 100/100.000 in developed countries) and the complexity of treatment that follows once return of spontaneous circulation (ROSC) has been achieved highlights the importance of early coordinated action. The complexity of patients with ROSC also emphasizes the use of protocols. The actions that lead to survival are called “the chain of survival”. The first link emphasizes the importance early recognition of those at risk of cardiac arrest and calling for help. The second and third links advocate early cardiopulmonary resuscitation (CPR) and early defibrillation. The final link focuses on effective post-resuscitation care. The most widely used protocol when approaching patients who are at high risk of cardiac arrest is the “ABCDE” protocol. It recognizes the importance of achieving control over *airway, breathing and circulation*, as well as early neurological assessment (“*disability*”) and careful clinical examination (“*exposure*”). Approach to patient in cardiac arrest and peri-arrest situations will be briefly discussed in the following manuscript.

### **4.2 Advanced Cardiac Life Support**

The most widely used advanced cardiac life support (ACLS) guidelines are produced by International Liaison Committee on Resuscitation (ILCOR) in coordination with major national and international organizations that are dealing with cardiac arrest (American Heart Association, European Resuscitation Council, Australian and New Zealand Committee on Resuscitation, etc.). Current guidelines were published in 2010 and next revision of guidelines is scheduled in 2015. When discussing ACLS it is assumed that high-quality chest compressions and ventilation of patient’s lung are administered. The major algorithm in current ACLS guidelines is based on recognition of the electrical activity of the heart. In case of ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT) defibrillation and additional medication (amiodarone) is administered. In case of asystole or pulseless electrical activity (PEA) chest compressions, ventilation of the lungs and adrenaline are used in an attempt to achieve ROSC. Recognition of underlying rhythm needs to be performed as soon as possible.

In VF/pVT cardiac arrest immediate defibrillation is advised. Pulse checks are performed every 2 min and CPR is started immediately after the defibrillation (i.e. the success of defibrillation will be assessed after 2 min of CPR). Adrenaline and amiodarone are administered after the third successive defibrillation. Once started, adrenaline is administered every 4 min. Amiodarone can be repeated

after the fifth defibrillation. In asystole/PEA cardiac arrest immediate CPR and adrenaline are advised. Pulse checks are performed every 2 min and adrenaline is repeated every 4 min.

The only two mandatory medications used are adrenaline and amiodarone for VF/pVT cardiac arrest and only adrenaline for asystole/PEA cardiac arrest. Adrenaline is used as 1 mg intravenous injection. Amiodarone is used as 300 mg bolus (or 150 mg bolus for repeat bolus after the fifth defibrillation). Intraosseous injection is equivalent to intravenous injection if venous access is not possible.

Airway can be secured either by tracheal intubation or supraglottic devices. Repeated attempts of tracheal intubation can result in prolonged periods of no chest-compressions, which are not recommended. Supraglottic devices can be inserted without laryngoscopy and without interrupting chest-compressions; however, tracheal intubation remains the golden standard in airway protection.

In order to increase the likelihood of ROSC it is advised to actively look for and treat the reversible causes of cardiac arrest. They are summarized as the “4H/4T”, namely Hypoxia, Hypovolemia, Hypothermia, Hypo/Hyperkalemia and Tamponade, Toxins, Tension pneumothorax, Thrombembolism (including acute coronary syndromes).

### **4.3 Managing the Peri-arrest Patient**

Failure of multiple organ systems can occur after cardiac arrest. This can lead to further deterioration and cardiac arrest if unrecognized and untreated. In order to successfully treat patients after cardiac arrest a large number of health-care providers need to cooperate in a meaningful fashion (typically, prehospital emergency teams, emergency department and intensive care teams, radiology, interventional cardiology and laboratory teams). Protocolled treatment is therefore needed. The ABCDE approach is the most widely used. It focuses on a stepwise, targeted examination of organ systems. The problems with individual organ systems are solved immediately after detection. Airway is checked and secured first. Effective breathing is second, with the goal of achieving oxygen saturation of 94-98%. Circulation is third. Measurement and treatment in case of abnormal results of blood pressure, heart rate and ECG is performed. Venous access is secured and withdrawal of blood for laboratory tests is performed. Focused neurological assessment including consciousness and blood sugar determination is performed next. Whole body examination is performed last. Multiple cycles of ABCDE are performed until the patient is treatment goals are achieved.

In order to achieve survival after cardiac arrest with good neurological outcome the chain of survival and ABCDE approach need to be considered.

### **4.4 Presentation of Simulation Scenario - Time Schedule**

<b>Part 1</b>	
00.00 – 01.30	Cardiac arrest management Basic Life Support Resuscitation teams, equipment
01.00 – 03.00	Advanced life support algorithm: cardiac arrest simulations
<b>Part 2</b>	
03.00 – 04.00	Cardiac arrest in special circumstances
04.00 – 05.00	Tachycardia – cardioversion, drugs Bradycardia – pacing, drugs

05.00 – 06.00	ABCDE scenarios Prevention of cardiac arrest
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## **4.5 Advanced Cardiac Life Support Basic Take Home Messages**

Patients who are at risk of cardiac arrest need to be recognized early in an attempt to prevent cardiac arrest.
Call for help early.
Begin CPR immediately once cardiac arrest is diagnosed.
Assess rhythm and defibrillate as soon as possible.
Follow post-resuscitation treatment goals once ROSC is achieved.
Ensure rescuer safety during CPR.
Look for reversible causes of cardiac arrest.

## **References**

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# **SELECTED TOPICS IN** **SIMULATION**



## **SECTION 5: SELECTED TOPICS IN SIMULATION - CARDIOLOGY SIMULATION**

*Author: Franjo Naji, Samo Granda, Marko Zdravkovic, Sebastjan Bevc*

CONTENT
1. Theoretical Background
2. Presentation of the Simulator
3. Presentation of Simulation Scenario – Time Schedule

### **5.1 Theoretical Background**

#### **Introduction**

Percutaneous coronary intervention (PCI) is a non-surgical method used to open narrowed arteries that supply heart muscle with blood (coronary arteries). PCI is performed by inserting a catheter through the skin in the groin or arm into an artery. At the leading tip of this catheter, several different devices such as a balloon, stent, or cutting device (arterectomy device) can be deployed. The catheter and its devices are threaded through the inside of the artery back into an area of coronary artery narrowing or blockage.

#### **Types of Coronary Interventions**

Intervention means that even if the person is actively having a heart attack (myocardial infarction or MI), PCI can be used to intervene and stop the attack by opening up the narrow or blocked coronary artery. This allows blood to flow to the heart muscle. PCI began as percutaneous trans-luminal coronary angioplasty (PTCA), a term still found in the literature, and now encompasses balloons, stents (metal scaffolding expanded inside the artery lumen), and other modifications to the catheter tip, including devices that can cut out plaque and thus open up the narrowed artery. Although treatment of acute heart attack is a very important use of PCI, it has several other uses. PCI can be used to relieve or reduce angina, prevent heart attacks, alleviate congestive heart failure, and allows some patients to avoid surgical treatment (coronary artery bypass graft - CABG) that involves extensive surgery and often long rehabilitation time.

#### **Procedure**

Balloon angioplasty employs a deflated balloon-tipped narrow catheter that is inserted through the skin of the groin or arm into an artery. The catheter is threaded through the artery until it arrives in the coronary artery where there is narrowing or blockage. The catheter tip is then inserted through the narrowed area. Once in the narrowed area, the balloon is inflated, mashing the plaque into the vessel walls to reduce the narrowing.

The balloon is then deflated and the catheter removed. The process is viewed by injecting a dye that allows the cardiologist to view the flowing blood as it goes through the arteries. This viewing method (angiogram) can be used to assure that the artery has increased blood flow after the balloon is deflated and removed.

A stent is an extendable metal scaffold that can be used to keep open previously narrowed coronary arteries after angioplasty has been performed. The mechanism used to place the stent in a narrowed or blocked coronary artery is very similar to balloon angioplasty. The difference is that the unextended or collapsed stent surrounds the balloon. The stent surrounding the balloon is expanded when the balloon is inflated. After the stent surrounding the balloon extends, it locks into place against the plaque/arterial vessel wall. The stent stays inside the artery after the balloon is deflated. Stents are useful because they keep the coronary artery open when the balloon is deflated, preventing most arteries from narrowing again (termed elastic recoil) after the balloon is deflated. Recurrent narrowing (restenosis) sometimes may still occur after the stent is placed due to formation of scar tissue.

The newest stents are termed drug-eluting stents. These stents are covered in a drug that slowly comes off the stent and prevents cell proliferation (scarring or fibrosis) at the stent site more effectively than uncoated, bare-metal stents. There are many other stents beside coronary stents that are used for various other arteries and tissues. These include carotid artery stents (for stroke prevention), femoral artery stents, prostatic stents, oesophageal stents, and many others.

Patients usually recover well after PCI. They are monitored and observed after the procedure. About 4–12 hours later, any catheter equipment still in the skin and artery are removed and pressure is held by hand or by clamps or "sandbags" for about 20 minutes to prevent bleeding into the catheter insertion site. Alternatively, some patients may have the artery sutured shut where the catheter was placed. Blood clots can form at the PCI site that may cause blockage. Patients are treated with blood thinning anti-platelet agents such as clopidogrel bisulfate (Plavix) and aspirin. Most patients will be taking anti-platelet medication indefinitely. Patients are often discharged within 24 hours after percutaneous coronary intervention and are cautioned not to do any vigorous activity for about one to two weeks. Some patients may be referred to a rehabilitation centre, but most patients are not, and can go back to work (if work is not physically intensive) in about three days after PCI.

### **Complications of Coronary Intervention**

Although over 95% of PCI procedures are successful, there are a few patients that still have problems. For example, sometimes the catheter (or its guide wire) cannot get through the narrowed lumen, or a thrombus (blood clot) forms at the site if the inner lining of the artery tears at the balloon site. Although agents are used to chemically prevent clot formations, not all treatments are successful. About 1%-2% of current PCI procedures fail and may require emergent CABG surgery. The risk of a heart attack is about 1%-2% in people that have PCI.

Current PCI mortality is less than 1%, an incidence of 6.7% patients develop a hematoma at the catheter entry site (groin or arm). Some patients may develop an aneurysm in the artery at the catheter entry site. Most patients will experience some bruising and tenderness at the catheter entry site.

## **5.2 Presentation of the Simulator**

Simulator consists of a console, which represents the patient, and the angiographic part. There is already a vascular access in place at femoral artery site in the console and we can use original equipment and instruments that is used for angiography with this simulator. Hence, as handling the

instruments is in practical work the first major obstacle in training and the simulator provide efficient training of instrument handling. Angiographic part is consisted of two screens, foot-hold fluoroscopy commands, and a handle for roentgen arc command.

One screen shows the angiographic image and hemodynamic monitoring of the patient. The other screen is a command screen (i.e. touchscreen) for all the procedure that we do during the investigation/procedure. Very easily the type of instrument we want to use, give drugs, perform defibrillation etc. As there is no physical roentgen arc it is just graphically presented on this screen as well, whereas command handle actually moves it on the screen. There is also an added value in learning with anatomic display command.

This simulator can simulate several percutaneous diagnostic and therapeutic procedures, which are grouped into modules: coronary module, lower extremities module, pacemaker placement, resynchronisation therapy etc. In each module we can select from a list of clinical scenarios. After selecting the one, the simulator provides basic history and examination data of the patient and then we can start interventions. All users choices and activities (material selection, duration of the procedure, contrast burden etc.) are recorded in a special log which is reviewed after the procedure.

Learning invasive coronary interventions consists of several skills: artery access, instrument and material handling, drugs administration, use of roentgen projections/planes and the arc handling, recognition of the diseased vessels and distinction from normal anatomy. Only after all these skills are acquired the real interventions learning should start: coronarography, balloon dilatation, stent placement, clots aspiration etc. The simulator enables learning all the described procedures except vascular access. We currently have 10 different cases to teach the interventions on. In addition to technical skills acquisition the learner gains experience also in emergency situations when treating an unstable patient and quick decision making.



**Figure 18: Simulator of Coronary Intervention in Action.**

### **5.3 Presentation of Simulation Scenario - Time Schedule**

<b>Part 1</b>	
00.00 – 00.30	Acquaintance with the simulator and equipment
00.30 – 00.50	Demonstration of a simple coronarography imaging of RCA and LAD+LCX
00.50 – 02.00	Training in pairs
<b>Part 2</b>	
02.00 – 04.00	Acquaintance with the portable cardiac ultrasound device Presentation of basic cardiac ultrasound examination protocol on a volunteer Each participant performs basic cardiac ultrasound examination protocol on a colleague

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## **SECTION 6: SELECTED TOPICS IN SIMULATION - GYNAECOLOGY SIMULATION**

*Author: Tamara Serdinšek, Andraž Dovnik, Iztok Takač*

CONTENT
1. Theoretical Background
2. Presentation of the Simulator
3. Presentation of Simulation Scenario – Time Schedule

### **6.1 Theoretical Background**

#### **Emergencies in Gynaecology**

Emergencies in gynaecology comprise a large spectrum of diseases, which importantly affect the function of the entire body. Two leading symptoms of these conditions are bleeding and lower abdominal pain (1). In our workshop, we will take a closer look at one of the trickiest and sometimes very hard to diagnose gynaecological conditions, which can quickly evolve into a true gynaecological emergency: ectopic pregnancy.

#### **Ectopic Pregnancy**

Ectopic pregnancy (EP) occurs when the conceptus implants either outside the uterus (Fallopian tube, ovary or abdominal cavity) or in an abnormal position within the uterus (cornua, cervix). Combined tubal and uterine (heterotopic) pregnancies are uncommon, as are combined bilateral tubal pregnancies, and both present a huge diagnostic problem (1, 2). The estimated incidence of EP is 16 per 1000 pregnancies (1).

Basically, any mechanical or functional factors that prevent or interfere with the passage of the fertilized egg to the uterine cavity may be aetiological factors for an EP. Risk factors for EP include (1, 2, 3):

- higher maternal age,
- large number of sexual partners,
- use of intrauterine device,
- pelvic inflammatory disease (PID), gonorrhoea, chlamydia,
- previous tubal surgery,
- previous EP.

The risk of recurrence is about 10% and is increased in those with previous miscarriage or who have suffered tubal damage.

95-98% of EP occur in the Fallopian tube (and more than 50% of those are situated in the ampulla). As the tube is an inappropriate place for the development of the fertilized egg, the pregnancy usually terminates within couple of weeks either as tubal abortion or tubal rupture.

## Clinical features of EP

**A diagnosis of EP should be considered in any woman of reproductive age presenting with abdominal pain and/or vaginal bleeding who has a positive pregnancy test (3)!**

- Signs and symptoms

The classical triad of amenorrhoea, lower abdominal pain and vaginal bleeding is rarely seen as most women with an EP in modern practice are clinically stable and have non-specific symptoms (3). The most commonly reported symptoms in EP are vaginal bleeding (usually in small amounts) and pelvic pain (1). Acute signs and symptoms of intra-abdominal bleeding are seen with tubal rupture (1).

- General examination

General examination must include a record of pulse rate and blood pressure. Look for signs of shock (low blood pressure, fainting, dizziness, rapid heart rate), which can be present in patients with ruptured EP (intra-abdominal bleeding) (2).

- Gynaecological examination

Bimanual examination must be performed with great care as it can provoke the rupture of the tube (2). Uterus can be enlarged, adnexal palpation on the site of the EP can be tender, and movement of the portion of the cervix can be painful (1).

- Human chorionic gonadotropin (hCG) levels and transvaginal ultrasound (tUS)

hCG levels and tUS findings allow a minimally invasive evaluation of the patient with a suspected EP and they must be interpreted together. One of the most important parameters is the discriminatory hCG level above which the gestational sac (GS) of an intrauterine pregnancy should be detectable by ultrasound (usually above 1000 IU/L) (2).

The presence or absence of an intrauterine GS is the principal point of distinction between tubal and intrauterine pregnancy. In 10-20% of EP a pseudogestational sac is seen (small, centrally located endometrial fluid collection surrounded by single echogenic rim of endometrial tissue undergoing decidual reaction). The sonographic finding of an extrauterine sac with an embryo or embryonic remnants is the most reliable diagnosis of EP. An empty ectopic sac or a heterogenous adnexal mass is more common US feature. The presence of fluid in the pouch of Douglas is a non-specific sign of EP (2).

## Management

The classical approach to the treatment of EP has always been surgical salpingectomy or salpingotomy (by laparotomy or laparoscopy). With the wider use of ultrasound and early diagnosis, some non-surgical therapeutic approaches have also been introduced (methotrexate, local injections of prostaglandins, potassium chloride, puncture and aspiration of ectopic sac etc.) (2).

## **6.2 Presentation of the Simulator**

Low and high fidelity simulators will be used during this workshop. Laparoscopic skills will be trained using high fidelity haptic simulator Symbionix Lap Mentor, which can be used for training of basic and

advanced laparoscopic skills. It is particularly useful for surgical residents at the beginning of their training. Firstly, we will practice our basic laparoscopic skills while completing several different tasks (moving objects with grasp, eye-hand coordination etc.). Then we will proceed to the full procedure training (laparoscopic salpingectomy).

### **6.3 Simulation Scenario**

<b>Part 1</b>		
00.00 – 00.30	Getting to know each other, theoretical part	
<b>Part 2</b>		
00.30 – 02.00	<b>Group 1:</b> Clinical Cases	<b>Group 2:</b> Training of Basic and Advanced Laparoscopic Skills
02.00 – 03.30	<b>Group 2:</b> Clinical Cases	<b>Group 1:</b> Training of Basic and Advanced Laparoscopic Skills
03.30 – 04.00	Pop quiz, closure	

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## **SECTION 7: SELECTED TOPICS IN SIMULATION - OPHTHALMOLOGY SIMULATION**

*Author: Dušica Pahor*

CONTENT	
1.	Theoretical Background
2.	Presentation of the Simulator
3.	Presentation of Simulation Scenario – Time Schedule

### **7.1 Theoretical Background**

The quality of ophthalmic surgical training has increasingly changed in the last years. Technical difficulties of ophthalmological procedures have increased the need to perform training outside the operating theatre. Surgeons must control the psychological stress during surgery. Ophthalmic virtual reality (VR) simulators represent a satisfactory response to this request.

VR simulation offers a new approach for surgical training. VRMagic EYESi Ophthalmosurgical Simulator is one of the well-developed simulators available currently. Results of previous studies revealed positive transfer from the simulation environment to initial operating room procedures. Simulation technology must be used to improve patient safety for surgical procedures such as vitreoretinal and cataract surgery in modern surgical education. The structured curriculum including the VR training program must be added to the residence education program. A procedure-based training curriculum for ophthalmic surgery must begin by teaching the basic skills, such as hand-eye coordination, depth perception, anti-tremor program, etc. A standardized approach to surgical training is needed. Only with integrated approach it will be possible to deviate from the old model of practising on real patients.

VR simulation can be made available to trainees anytime and anywhere and does not require any additional supplies or animal tissue. It is the ideal training tool.

### **7.2 Presentation of the Simulators**

VRMagic EYESi Ophthalmic Surgical Simulator (Mannheim, Germany) is one of the well-developed simulators available in the market currently. Several reports about training on EYESi were published in the last few years. The modern equipment at Faculty of Medicine, University of Maribor with complete nowadays available ophthalmic simulators including:

**EyeSi Indirect ophthalmoscope simulator,**

**EyeSi Vitreoretinal simulator,**

**EyeSi Cataract simulator,**

offers a new opportunity to perform a new modern concept of ophthalmic surgical training.

EYESi Surgical was originally designed as a vitreoretinal training device. After some time an anterior segment module was developed for cataract surgery training as well as EYESi indirect and direct simulator. VRMagic EYESi ophthalmic surgical simulator allows repeated measurements of standardized surgical tasks. Feedback is provided in the surgical goal, surgeon efficiency, achievement of surgical goal, surgeon error or tissue injury and formative education feedback. Simulator consists of mannequin head with an electronic eye that rotates when manipulated by the surgeon. The simulation system is controlled by means of the touch screen. After login, the instruments can be configured and the courses selected and started. Original handheld instruments are inserted into the electronic eye and are freely movable. Instrument interaction with tissue and ocular structure is simulated in real time. A virtual operating microscope complete with zoom/focus foot pedal provides stereoscopic images of the eye and instruments to the surgeon. Each module has different difficulty levels to simulate increasingly complicated tasks. Required surgical skills can be isolated and train separately until they have been fully mastered. Each course combines training of basic skills with training of actual surgical procedures in structured course design, leading students step-by-step to expert performance.

### **7.3 Presentation of Simulation Scenario - Time Schedule**

00.00 – 01.30	Acquaintance with the simulator and equipment
01.30 – 04.00	Demonstration of a simple procedures

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## **SECTION 8: SELECTED TOPICS IN SIMULATION - GASTROENTEROLOGY SIMULATION**

*Author: Marjan Skalicky, Darja Lorber, Aleš Kodela, Miha Kodela*

CONTENT
1. Theoretical Background
2. Presentation of the Simulator
3. Presentation of the Portable Ultrasound Device
4. Basic Abdominal Ultrasound Examination Protocol
5. Presentation of Simulation Scenario – Time Schedule

### **8.1 Theoretical Background**

#### **Upper Endoscopy**

Upper endoscopy, also known as esophagogastroduodenoscopy (EGDS), is a procedure performed by passing a flexible endoscope through the mouth into the oesophagus, stomach and proximal duodenum. It allows real time assessment and interpretation of the findings encountered and is the best method of examining the upper gastrointestinal (GI) mucosa. EGDS can be used as diagnostic, screening or therapeutic tool. The most common indications for EGDS are unexplained dyspepsia, gastro-oesophageal reflux disease (GERD), persistent nausea and vomiting, bleeding from upper GI tract, dysphagia, pathologic or unclear findings with diagnostic imaging, removal of foreign objects, screening and surveillance of prior pathological findings. Possible complications of EGDS are rare and include complications due to sedation, bleeding, and perforation. Patients scheduled for elective EGDS are instructed to take nothing by mouth 4-8 hours prior the procedure.

#### **Colonoscopy**

Colonoscopy is performed by passing a flexible colonoscope through the anal canal into the rectum, colon and terminal ileum. This procedure is the gold standard for diagnosis of colonic mucosal disease. Most common indications for colonoscopy are screening/follow up for polyps or colon cancer, rectal bleeding, changes in bowel habits, chronic unexplained rectal or abdominal pain and pathologic or unclear findings with diagnostic imaging. During the procedure the examiner may take a biopsy or perform some therapeutic procedures such as removal of polyps. Serious complications of colonoscopy such as heavy bleeding or perforation are rare. Patient preparation is crucial for good visualization of the colon. Patients are instructed to avoid solid food one day before the test and drink only clear liquids up to several hours before the procedure. They also receive laxatives and copious amounts of fluids to clean their bowels.

### **8.2 Presentation of the Simulator**

Following the students' preferences the simulation system MENTOR-SIMBIONIX enables the acquisition of in-depth knowledge in endoscopic examinations (EGDS, colonoscopy, ERCP/EPT, EUS). If an invasive intervention is indicated (like all digestive endoscopies), the prescriber issuing a referral is obliged to obtain an informed consent from the patient. The prescriber is required to present the course and manner of the procedure, as well as the expected diagnostic therapeutic result in a

comprehensive manner. The prescriber also needs to point out the possible complications. By performing active work on simulators the students acquire the necessary information or knowledge.



Figure 19: Mentor-Simbionix Simulation System

### **8.3 Presentation of Portable Ultrasound Device**

Portable US device we use is called Sonosite NanoMaxx. It is a high quality portable US machine. It has an 8.4 inch touch screen and one knob button control. Light weight (2.7kg) and a build in battery make carrying it around and using it by patients' bed easy. It supports linear, curved and phased array transducers. Our machine has a 5-2 MHz C60n curved array transducer with a 30 cm scan depth, which is suitable for abdominal, gynaecology, nerve and obstetrics examination. Available scanning modes are 2D, colour Doppler, colour power Doppler mode and M-Mode. 2 GB of internal memory allows saving of up to 1800 images. If needed Wireless DICOM Image/Data Transfer and export of images to an USB device are also supported. Composite video output allows image projection on a bigger screen, which is great for learning purposes.



## **8.4 Basic Abdominal Ultrasound Examination Protocol**

(written by Robert Ekart, Sebastjan Bevc, Marko Zdravković)

Knock on the door before entering, enter and greet the patient.
Disinfect your hands.
Introduce yourself to the patient.
Explain the purpose of the exam to the patient.
Obtain patient's consent.
Confirm that that patient had not eaten for at least 6 hours.
Ask the patient to lie on his back.
Put some US gel on the patient's abdomen (warn him it may be a little bit cold), hold the transducer with your right hand and orient it properly (check a mark on the screen or help yourself with palpation of the tip of transducer while observing its movement on the screen).
Place the probe in the epigastrium and visualize abdominal aorta and inferior vena cava in the transversal plane.
Follow the transverse intersection of both vessels from the epigastrium caudally to their bifurcation.
Rotate the probe and follow the course of abdominal aorta and inferior vena cava in the longitudinal plane.
Place the probe under the right costal margin, ask patient to inhale deeply and hold his/her breath; visualize the liver parenchyma. To check all liver segments also place the probe in the epigastrium and intercostal.
Visualize the gallbladder (you may help yourself with colour Doppler).
Ask the patient to inhale and hold his breath. Search for the right kidney and visualize it in longitudinal and transverse plane. If you have problems visualizing the kidney, you may ask the patient to turn on his left side or on his abdomen.
Ask the patient to take a deep breath and hold his breath again. Check for spleen and left kidney under the left costal margin. Visualize the kidney in longitudinal and transverse plane. You may ask the patient to lay in his right side or abdomen if having problems.
Place the probe suprapubically and visualize the urinary bladder, prostate (male patient), uterus (female patient) in both planes.
Explain your findings to the patient.
Thank the patient for cooperation.
Disinfect your hands.

## **8.5 Presentation of Simulation Scenario - Time Schedule**

<b>Part 1</b>	
00.00 – 00.15	Introduction, getting to know each other
00.15 – 00.30	Acquaintance with the Symbionix GI Mentor simulation system
00.30 – 00.50	Demonstration of anatomy of upper and lower GI system and normal gastroscopy and colonoscopy
00.50 – 02.00	Each participant performs a normal EGDS and colonoscopy and one case of a pathologic gastroscopy and colonoscopy
<b>Part 2</b>	
02.00 – 02.15	Acquaintance with the portable ultrasound device
02.15 – 02.30	Demonstration of extended focused abdominal ultrasound on a phantom
02.30 – 03.15	Each participant performs extended focused abdominal ultrasound on a phantom
03.15 – 04.00	Each participant performs basic abdominal ultrasound examination protocol on a

colleague
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