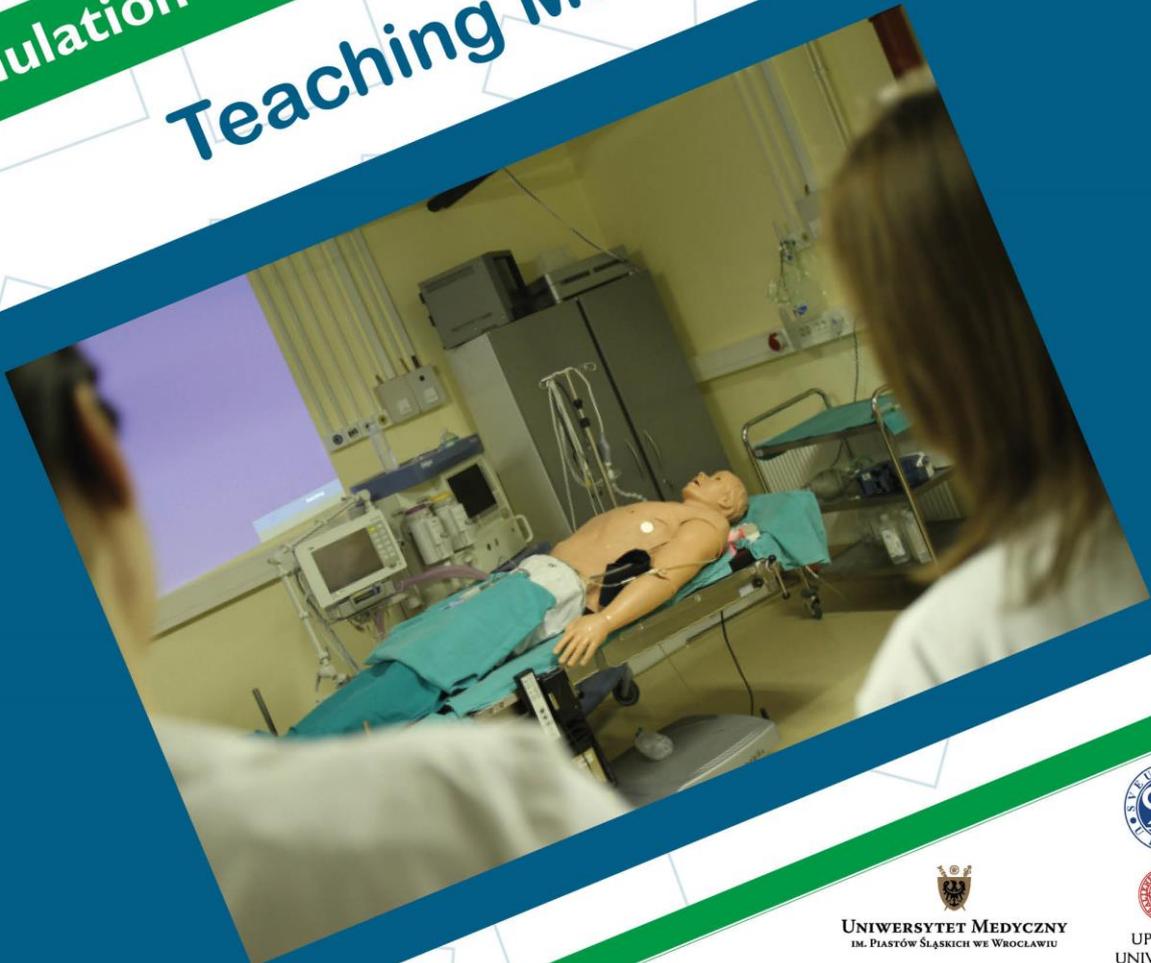


ERASMUS INTENSIVE PROGRAMME

Simulation in Clinical Practice: Virtual Medicine
Teaching Material



Erasmus Intensive Programme: Simulation in Clinical Practice

TEACHING MATERIAL

Faculty of Medicine, University of Maribor

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SECTION 1: ABDOMINAL ULTRASONOGRAPHY

Authors: Marjan Skalicky, Darja Lorber, Ales Kodela

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

1.1 Theoretical Background

Ultrasound (US) is a sound with a frequency above the human hearing level (>20000 Hz). The basic principle of diagnostic US is emitting the US waves into the human body and listening to the echoes. US machine analyzes the amplitude and time of the returning echoes and produces a real time image of the scanned tissue.

US waves are created in the US transducer probe that contains piezoelectric crystals. The piezoelectric crystals are capable of transforming electrical energy into mechanical oscillations that emit US waves. When receiving echoes, the piezoelectric crystals work the other way around, producing electrical current from which the real time image is generated. US probes that emit frequencies between 2 and 10 MHz are used in medicine. The probe is chosen depending on the tissue that has to be scanned. Higher frequency has shorter penetration and better resolution and vice versa. So we use high frequency (>5 MHz) probes when we want to see the structures that are right below the skin (neck, breast, scrotum, etc.), low frequency (1.6-2.25 MHz) probes are used for trans-cranial imaging and middle frequency (3-4 MHz) probes for abdominal US.

Echogenicity is a measure of acoustic reflectance (the extent to which a structure reflects ultrasonic waves). Structures seen on the US may be described as hyperechogenic, echogenic, hypoechogenic or anechogenic depending on their echogenicity. Hyperechogenic structures (fasciae, bones, artery wall, etc.) look completely bright on the screen because they reflect most of the sound waves, opposite to the anechogenic structures (fluids, blood, urine, cysts, effusions, etc.) that reflect almost none of the passing US waves and look almost completely black.

US examination can be enhanced with Doppler mode, which uses the Doppler effect to assess if fluid (blood) is moving towards or away from the ultrasonic probe. Fluids that are moving towards the probe are usually colored red, while fluids moving away are visible as blue color on the US machine screen.

1.2 Presentation of the Simulator

The current clinical propaedeutic abdominal examination includes palpitation, auscultation, and percussion. In order to establish the real anatomic condition of 28 organs or chemical systems, the use of complementary US examination is recommended to identify pathological conditions. During the study at the Faculty of Medicine in Maribor, ultrasonography training is conducted at the simulation center. First, the students get acquainted with US skills on phantoms, then they practice

among themselves simulating the physician-patient relationship, and eventually participate in clinical work on real patients. The transportable US device is called abdominal phonendoscope (USP). While performing the EFAS (extended focused abdominal US) program, the use of USP has become an additional tool in the black bag. Ultrasound phonendoscope (USP) simulations enable the acquisition of propaedeutic knowledge in abdominal anatomy using portable US. Deviations are listed in the medical report. There are 80 virtual patients with clinical data and planned algorithm available. The progress in acquiring simulation skills is assessed according to objective criteria, which also demonstrates the level of acquired knowledge.

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.

1.3 Presentation of Simulation Scenario – Time Schedule

Simulation Part (Marjan Skalicky, Darja Lorber, Ales Kodela)	
00.00 – 00.15	Introduction, getting to know each other.
00.15 – 00.30	Acquaintance with the portable ultrasound device.
00.30 – 00.45	Demonstration of extended focused abdominal ultrasound on a phantom.
00.45 – 01.45	Each participant performs extended focused abdominal ultrasound on a phantom.
01.45 – 02.00	Break.
Clinical Part (Marjan Skalicky, Darja Lorber, Ales Kodela)	
02.00 – 03.45	Practice of extended focused abdominal ultrasound on patients.
03.45 – 04.00	Break.
Simulation Part (Ales Kodela)	
04.00 – 04.15	Presentation of basic abdominal ultrasound examination protocol on a volunteer.
04.15 – 05.45	Each participant performs basic abdominal ultrasound examination protocol on a colleague.

1.4 Basic Abdominal Ultrasound Examination Protocol

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

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.

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SECTION 2: ENDOSCOPY SIMULATION

Authors: Marjan Skalicky, Darja Lorber, Ales Kodela, Miha Kodela

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

2.1 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.

Endoscopic ultrasound (EUS)

EUS uses high frequency US probe that is incorporated into the tip of the flexible endoscope. It obtains images of the gut wall and adjacent organs, vessels and lymph nodes. It is the most accurate preoperative local staging tool for oesophageal, pancreatic and rectal cancer. EUS can be useful for diagnosis of gallbladder disease, bile duct stones, submucosal GI lesions and chronic pancreatitis.

2.2 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.

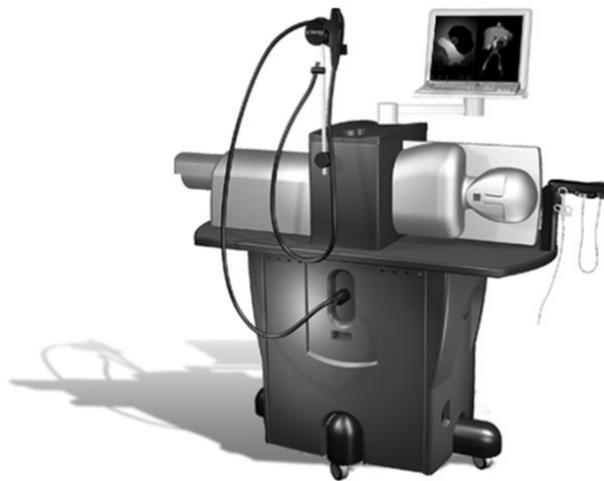


Figure 1: Mentor-Simbionix Simulation System

First Module for Lower Gastrointestinal Endoscopy (Colonoscopy)

The module is intended for trainees who have just begun the “hands-on” phase of learning and training in colonoscopy.

The module objectives are:

- Performing a complete survey of the lower GI tract with a forward viewing video-endoscope.
- Performing diagnostic and therapeutic procedures in “patients” with different colon anatomies.
- Recognition of typical lesions and abnormalities.
- Performing basic therapeutic procedures.

The module consists of 10 cases. The cases are arranged hierarchically from a simple diagnostic procedure to a difficult case, involving all training objectives of this module.

Available cases: Anatomy, normal tissue, melanosis, angiodysplasia, Crohn’s disease, diverticula, pseudomembranes, tumor, pedunculated polyp, ischaemic colitis, sessile polyp, pseudopolyps, diverticulum.

Second Module for Lower Gastrointestinal Endoscopy (Colonoscopy)

The module is designed for trainees who have just begun the “hands-on” phase of learning and training in Colonoscopy.

The module objectives are:

- Performing a complete survey of the lower GI tract with a forward viewing vide-endoscope.
- Recognition of typical lesions and abnormalities.
- Performing diagnostic and therapeutic procedures in “patients” with different colon anatomies and pathologies.

The module consists of 10 cases. The cases were created by Prof. Florent, Hopital Saint-Antoine, Paris, France.

Available cases: Anatomy, pedunculated polyp, ischaemic colitis, ulcerative colitis, diffuse angiomatosis, diverticulum, Crohn’s disease, diverticulitis, pseudomembranous colitis, benign tumor.

First Module for Upper Gastrointestinal Endoscopy (Gastroscopy)

This module is intended for trainees who have just begun the “hands-on” phase of learning and training in upper GI endoscopy.

- Performing a complete survey of the upper GI tract with a forward viewing video-endoscope.
- Performing diagnostic and therapeutic procedures in “patients” with major pathologies.
- Recognizing typical lesions.
- Performing basic therapeutic procedures.

The module consists of 10 cases. The cases are arranged hierarchically from a simple diagnostic procedure to a difficult case, involving all training objectives of this module.

Available cases: Anatomy, normal tissue, diverticulum, ulcer, inflammation, sessile polyp, tumor, bleeding ulcer, varices, leiomyoma, pedunculated polyp, AV malformation.

Second Module for Upper Gastrointestinal Endoscopy (Gastroscopy)

This module is designed for trainees who have just begun the “hands-on” phase of learning and training in Gastroscopy.

The module objectives are:

- Performing a complete survey of the Upper GI track with a forward viewing video-endoscope.
- Recognizing typical lesions.
- Performing diagnostic and therapeutic procedures in “patients” with major pathologies.

The module consists of 10 cases. The cases were created by Prof. Florent, Hopital Saint-Antoine, Paris, France.

Available cases: Oesophageal varices, bleeding ulcer, celiac disease, cystic dilation of fundic glands, degenerated gastric polyp, ulcer of oesophagus, pseudo-Whipple, watermelon stomach, haemorrhagic gastritis, portal hypertensive gastropathy.

First Module for Bleeding Situations

The module is designed to provide skilled endoscopists, trainees and nurses with a training model for emergency bleeding situations that require an urgent treatment.

The module objectives are:

- Performing complete survey of the upper GI tract throughout an emergency endoscopy.
- Performing diagnostic procedures in “patients” with symptoms of bleeding lesions.
- Performing therapeutic procedures using a variety of appropriate accessories (tools) intended for bleeding pathologies.

The module consists of 10 cases which include all the indicated training objectives. The cases were created by Dr. Sven Adamsen and Dr. Soren Meisner, Bispebjerg Hospital, Copenhagen, Denmark.

Available cases: Bleeding ulcer, ulcer with a clot, Dieulafoy’s lesion, Mallory-Weiss Tear, small cancer, duodenal ulcer, pre-pyloric ulcer.

2.3 Presentation of Simulation Scenario – Time Schedule

Simulation Part (Jure Auda, Miljenko Krizmaric)	
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.
00.45 – 01.00	Each participant performs a normal EGDS and colonoscopy.
01.45 – 02.00	Break.
Clinical Part (Darja Lorber, Jure Auda)	
02.00 – 03.45	Visit of the Endoscopy Section at Department of Gastroenterology.
03.45 – 04.00	Break.
Simulation Part (Marjan Skalicky, Darja Lorber, Jure Auda)	
04.00 – 04.15	Presentation of pathologic gastroscopy and colonoscopy.
04.15 – 05.45	Each participant performs one case of a pathologic gastroscopy and colonoscopy.

2.4 Case Presentation

Colonoscopy

Case 1 (First Module): A 30 year old female, with irregular bowel movements, alternated between constipation and diarrhoea. Stool is negative for parasites. She was referred for colonoscopy.

Biological tests: Hgb 11.3 g/dl (12-18), Hct 39 % (36-54), Alb 4,2 g/dl (3.5-5.5), Ca 9.6 mg/dl (9.0-10.5), TSH 2.0 mU/dL (0.2-3.0), FT4 17 pmol/dl (11.7-28).

Case 1 (Second Module): Mr. Cochet, 59 years of age, was hospitalized for diarrhoea that developed during the past 3 months, with a weight loss of 5 kg. Physical and rectal examinations were normal. The patient's medical history includes a cholecystectomy, which was performed 10 years ago, for cholelithiasis. The patient is not taking any medication.

Biological tests: RBC $4.3 \times 10^{12}/L$ ($4-5 \times 10^{12}$), Hgb 10.7 g/dL (>14), Hct 35 % (40-48), serum iron 5.6 $\mu\text{mol/l}$ (10.7-26.9), platelets $235 \times 10^9/L$ ($100-300 \times 10^9$).

Gastroscopy

Case 1 (First Module): A 30-year old male suffering from epigastric pain for the past 6 months. Physical examination does not show anything unusual. No response to omeprazole. He was referred for gastroscopy.

Biological tests: Hgb 14 g/dl (12-18), Hct 48 % (36-54), Alb 5.0 g/dl (3.5-5.5), Alk Phos 100 U/L (45-115), AST 26 U/L (0-35), ALT (30 U/L).

Case 1 (Second Module): Mr. Dupond, 40 years of age, was transferred to the Intensive Care Unit, due to hematemesis and melena. He is a bank clerk. He underwent a surgery at the age of 8 for an umbilical hernia. He has smoked 30 cigarettes per day and has consumed 30 g of alcohol (beer) daily for the past 20 years. He has periumbilical collateral venous circulation, splenomegaly and abdominal meteorism.

Biological tests: RBC $4.3 \times 10^{12}/L$ ($4-5 \times 10^{12}$), MCV 109 fL (85-95), Leucocytes 11.7×10^9 ($5 - 10 \times 10^9$), platelets $95 \times 10^9/L$ ($100-300 \times 10^9$), Hgb 8.5 g/dL (>14), ALT 95 IU (<30), Alk Phos 80 IU (<100), PT 65 % (<75).

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SECTION 3: ACUTE CORONARY SYNDROME

SIMULATION

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CONTENT	
1.	Theoretical Background
2.	Presentation of the Human Patient Simulator
3.	Presentation of Simulation Scenario – Time Schedule
4.	Case Presentation
5.	Acute Coronary Syndrome Management Algorithm

3.1 Theoretical background

Introduction

Cardiovascular disease is the leading cause of death in industrialized countries. Among these, coronary artery disease (CAD) is the most prevalent manifestation of cardiovascular disease and is associated with high mortality and morbidity. The clinical presentations of CAD include silent ischemia, stable angina pectoris (AP), unstable angina pectoris (UAP), myocardial infarction (MI), heart failure, and sudden cardiac arrest. Acute coronary syndrome (ACS) is a sudden and potential life-threatening manifestation of CAD. Chest pain is the leading symptom that initiates the diagnostic and therapeutic cascade, but the classification of patients and treatment strategies are based on the electrocardiogram (ECG).

It is very important that signs and symptoms of ACS are fast recognised and appropriate prompt action is undertaken by the patients themselves, eyewitnesses and medical personnel in order to reduce the risk of cardiac arrest and death (chain of survival).

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) 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. As a consequence, 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 arrhythmogeneity.

Stable CAD in the absence of plaque disruption may also result in ACS when physiologic stress (eg. trauma, anemia, 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, 3rd or 4th 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.

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 (β -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.

Possible non-acute coronary syndrome causes of troponin elevation:

Chronic or acute renal dysfunction, severe acute or chronic congestive heart failure, hypertensive crisis, tachy- or bradyarrhythmias, pulmonary embolism, severe pulmonary hypertension, myocarditis, acute neurological disease (e.g. stroke, subarachnoid haemorrhage), aortic dissection, aortic valve disease or hypertrophic cardiomyopathy, cardiac injury (contusion, ablation, pacing, cardioversion, or endomyocardial biopsy), hypothyroidism, Tako-Tsubo cardiomyopathy, infiltrative diseases (e.g. amyloidosis, haemochromatosis, sarcoidosis, sclerodermia), drug toxicity (e.g. adriamycin, 5-fluorouracil, herceptin, snake venoms), burns affecting >30% of body surface area, rhabdomyolysis, critically ill patients, especially with respiratory failure or sepsis.

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 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 2: Human Patient Simulator in Action

3.3 Presentation of the Simulation Scenario – Time Schedule

Introduction (Miljenko Krizmaric, Tadej Zorman)	
00.00 – 00.15	Acquaintance with the HPS.
00.15 – 00.45	Acquaintance with components of the HPS monitoring (blood pressure measurements, pulse oximetry, ECG).
00.45 – 01.30	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).
01.30 – 1.45	Acquaintance with drugs needed for the simulation scenario.
01.45 – 02.00	Break.
Simulation (Tadej Zorman, Sebastjan Bevc, Miljenko Krizmaric)	
02.00 – 02.15	Pop quiz about ACS knowledge.
02.15 – 03.30	Performance of ACS simulation scenario: presentation of clinical case and practical training.
03.30 – 03.50	Debriefing and evaluation.

3.4 Case Presentation

A 55-year old Caucasian male presents to the emergency department (ED) with chief complaint of chest pain. The pain has begun 3 hours ago while walking upstairs to the first floor. It was pressure-like in character, radiating to the left arm and neck, and did not change with breathing or body position, visual analogue scale (VAS) score was 4/10. He immediately took one pill of Aspirin 500 mg and 30 min after the pain almost completely disappeared. So far he has never felt a pain like this.

One hour later, while he was watching TV, the same kind, but more severe, pain appeared again. He felt sick and out of breath. Despite another Aspirin pill, he did not feel better; therefore, he decided to seek medical help. While he was driving to the ED, the pain stayed unchanged.

Past medical history: arterial hypertension and hypercholesterolemia, both known for 2 years, otherwise healthy, does not exercise regularly.

Family history: mother has diabetes mellitus type 2, father has arterial hypertension, he had an ischemic cerebral stroke

Childhood diseases: mumps, chickenpox, scarlet fever

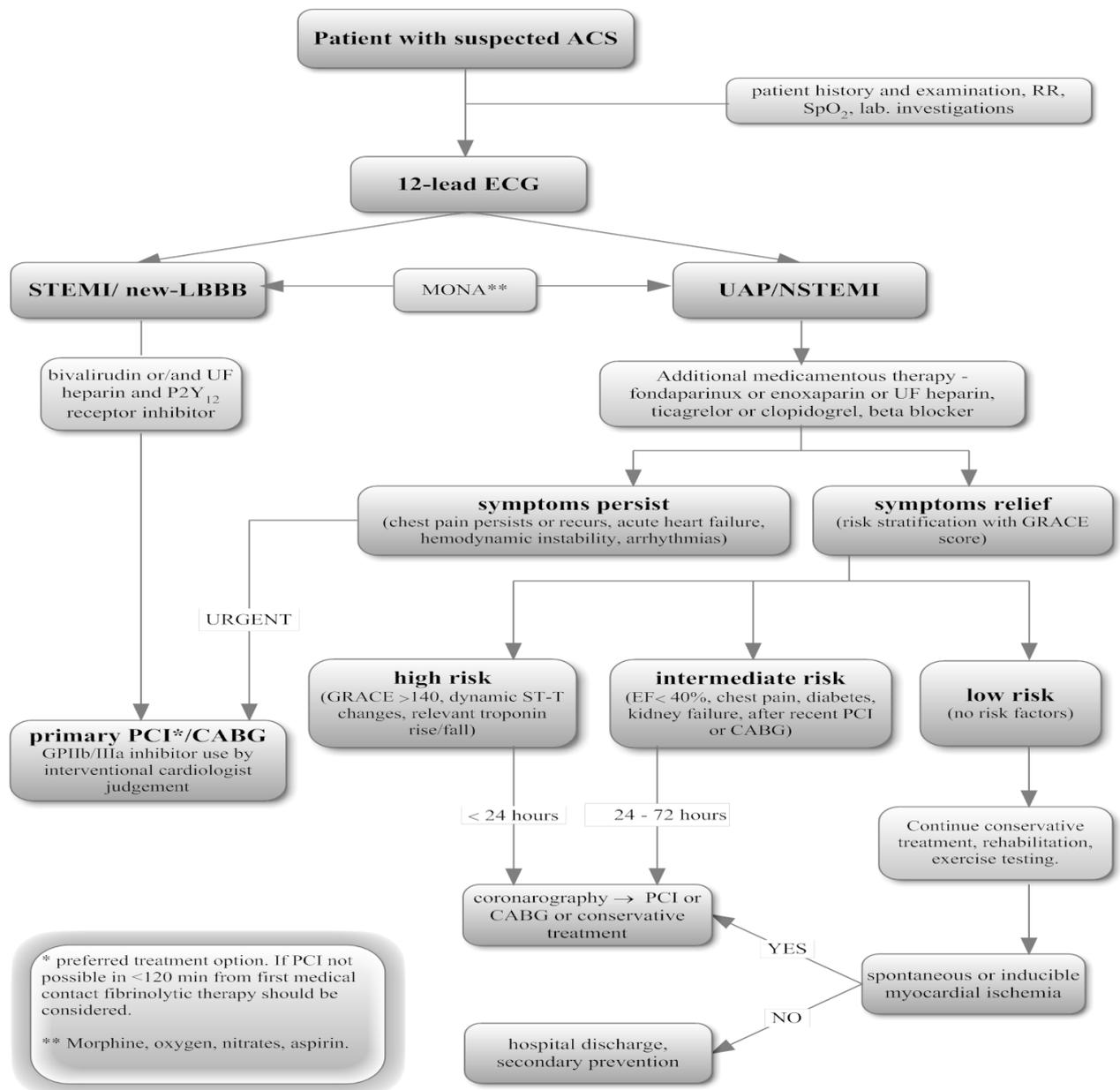
Allergies: penicillin

Medicine: atorvastatin 20 mg daily, perindopril 4 mg daily (does not take regularly)

Social history: married, one son (25 years old), works as a mechanic, smokes 20 cigarettes per day for 40 years, drinks 2-3 beers a month.

Physical Examination: Blood pressure 165/95 mmHg, pulse 85/min, SpO₂ 94%, height 185 cm, weight 100 kg (overweight, BMI 29,2 kg/m²), otherwise normal.

3.5 Acute Coronary Syndrome Management Algorithm



References

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SECTION 4: SIMULATION OF ARRHYTHMIAS

Authors: Nina Hojs, Tadej Zorman, Sebastjan Bevc

CONTENT	
	1. Theoretical Background
2.	ECG Strips of Important Arrhythmias to be Recognized on Monitors
3.	Presentation of Simulation Scenario – Time Schedule
	4. Case Presentation
5.	Arrhythmia Management Algorithm

4.1 Theoretical Background

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: 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/hyperthyroidism), 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 SpO₂ < 94 %), obtain an intravascular (i.v.) access, draw blood for laboratory tests, monitor SpO₂, 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 1). 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 pectorial-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 2). If they are present, synchronised cardioversion is used. Give conscious patients i.v. analgesia and sedation. Apply pads in the right pectorial-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 2).

Cardiac Arrest

After the recognition of cardiac arrest (no breathing, no pulse), early cardiopulmonary resuscitation (CPR) is important. Give 30 chest compressions followed by 2 ventilations. Attach a defibrillator and assess rhythm (stop chest compressions).

If the rhythm is shockable (VF, pulseless VT), deliver 1 shock (use the same precautions as in synchronised cardioversion). Without reassessing the rhythm or feeling for a pulse, restart CPR for 2 min. Then pause and check the rhythm, if the rhythm is shockable, deliver a shock and resume CPR. If a shockable rhythm persists give adrenaline 1 mg i.v. and amiodarone 300 mg i.v. after 3 shocks. Give further adrenaline 1 mg i.v. after every alternate shock (every 3-5 min).

If the rhythm is non-shockable (PEA, asystole), continue with CPR and give adrenaline 1 mg i.v. as soon as intravascular access is achieved. Recheck the rhythm every 2 min. If non-shockable rhythm persists give adrenaline every 3-5 min.

During CPR treat reversible causes (4H, 4T); obtain a secure airway and vascular access.

If signs of life (respiratory effort, movement) or readings from patient monitors compatible with the return of spontaneous circulation (ROSC) appear during CPR, stop CPR briefly and check rhythm on the monitor. If an organised rhythm is present, check for a pulse. If a pulse is palpable, continue post-resuscitation care and/or treatment of peri-arrest arrhythmia if appropriate. If no pulse is present, continue CPR.

If attempts at obtaining ROSC are unsuccessful, the cardiac arrest team leader should discuss stopping CPR with the resuscitation team. The decision requires clinical judgment and assessment of the likelihood of achieving ROSC.

4.2 ECG strips of important arrhythmias to be recognized on monitor



Figure 3: Ventricular tachycardia

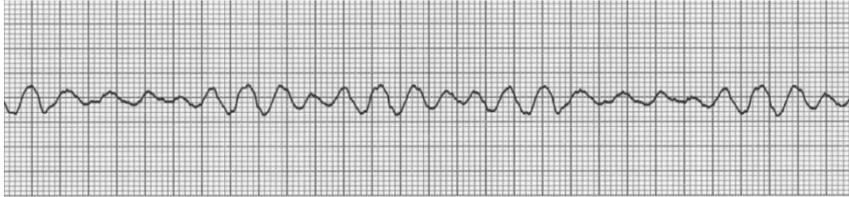


Figure 4: Fine ventricular fibrillation.



Figure 5: Asystole.

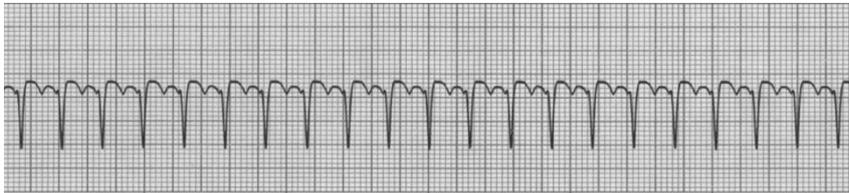


Figure 6: Supraventricular tachycardia.



Figure 7: Atrial flutter.



Figure 8: Atrial fibrillation.

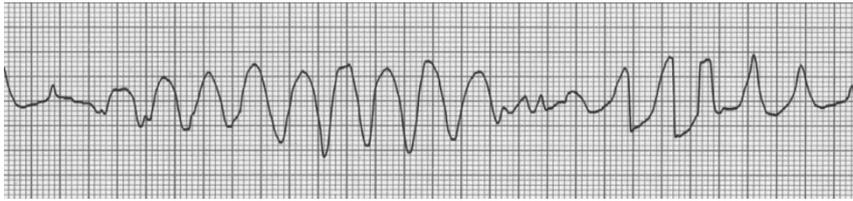


Figure 9: Polymorphic ventricular tachycardia - torsades de pointes.

4.3 Presentation of Simulation Scenario – Time Schedule

Introduction and Simulation (Nina Hojs, Sebastjan Bevc, Miljenko Krizmaric)	
00.00 – 00.15	Acquaintance with drugs needed for the simulation scenario.
00.15 – 00.30	Pop quiz about arrhythmias knowledge.
00.30 – 01.30	Performance of arrhythmias simulation scenario: presentation of clinical case and practical training.
01.30 – 02.00	Debriefing and evaluation.

4.4 Case Presentation

A 55-year old male comes to the internal emergency department.

Present complaints: the last few days he feels weak, he is out of breath when physically active, his heart sometimes beats hard and fast, he sweats more. He denies chest and abdominal pain, fever, headache, leg swelling, abnormal voiding and abnormal stool passage.

Allergies: none

Drugs: one for arterial hypertension

Past medical history: He has arterial hypertension for approximately 8 years, blood pressure at home is around 160/90 mmHg.

Childhood diseases: none

Family diseases: his father died of stroke

Social history: he is an economist, married, father of 2 children. He does not smoke; he drinks 4 beers per week.

Weight: 90 kg (198 lbs)

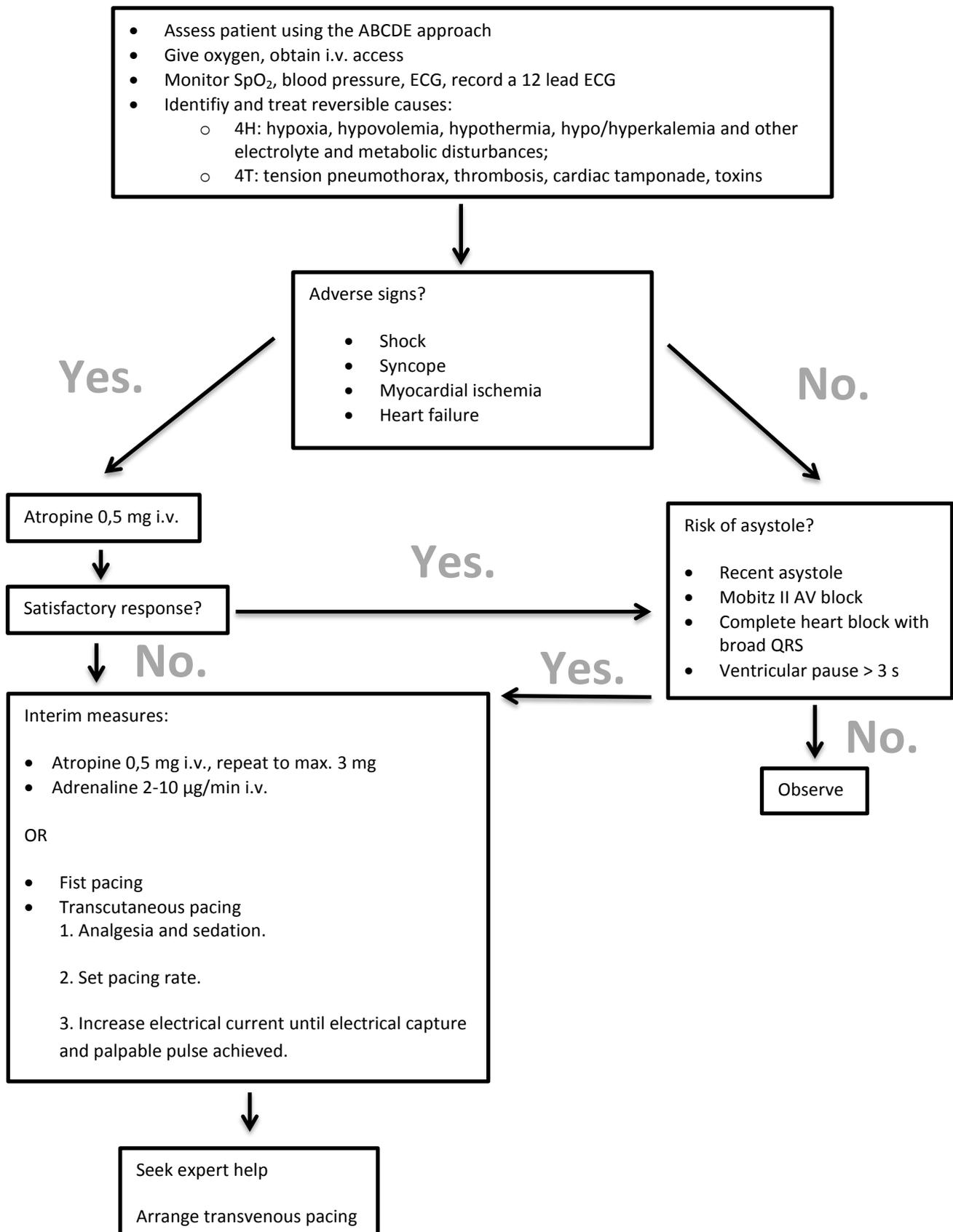
Height: 185 cm (72.8 in)

Body mass index: 26.3 kg/ m²

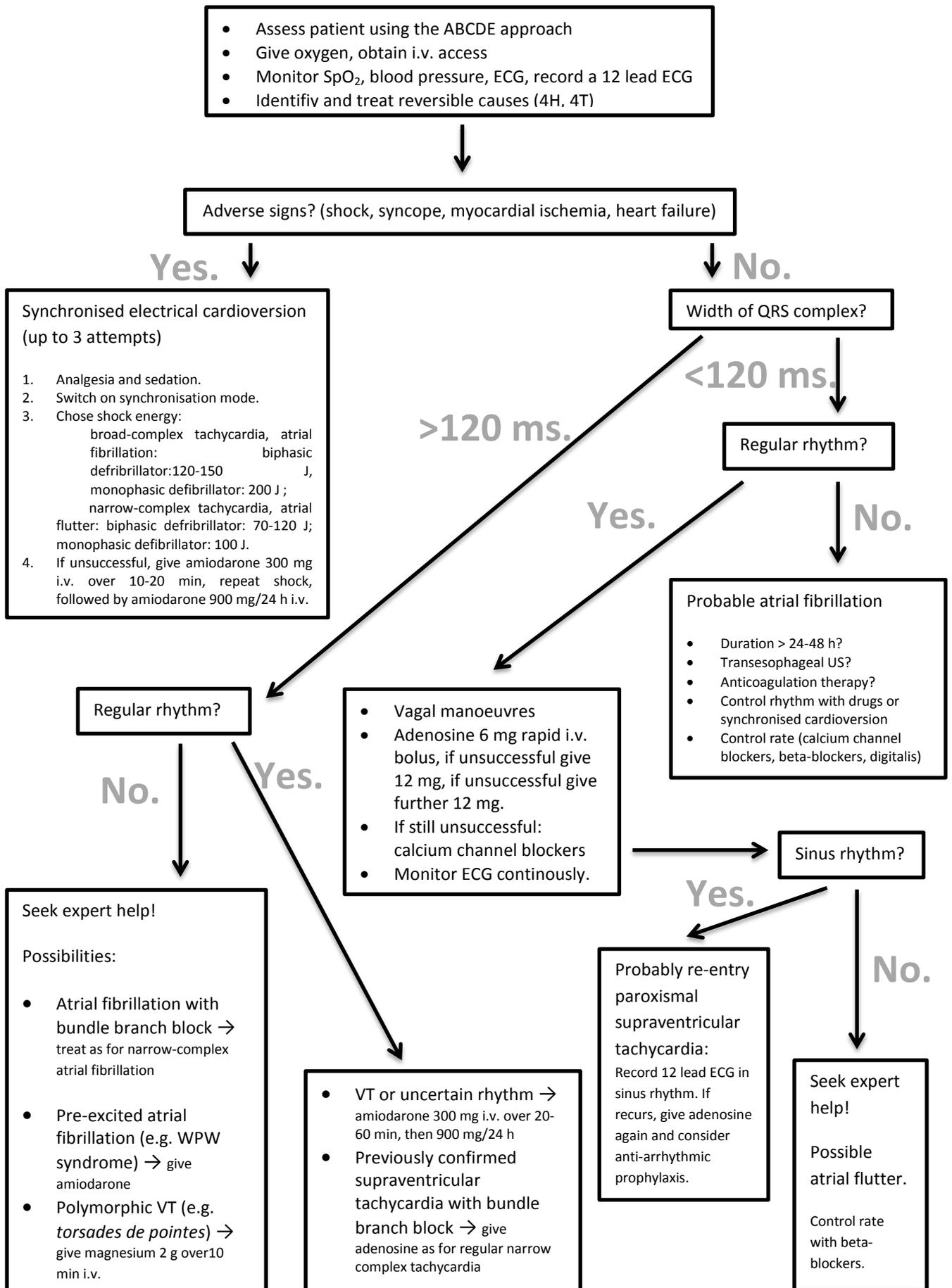
Physical Examination: Blood pressure 165/95 mmHg, pulse 85/min, SpO₂ 94%, height 185 cm, weight 100 kg (overweight, BMI 29,2 kg/m²), otherwise normal.

4.5 Arrhythmia Management Algorithm

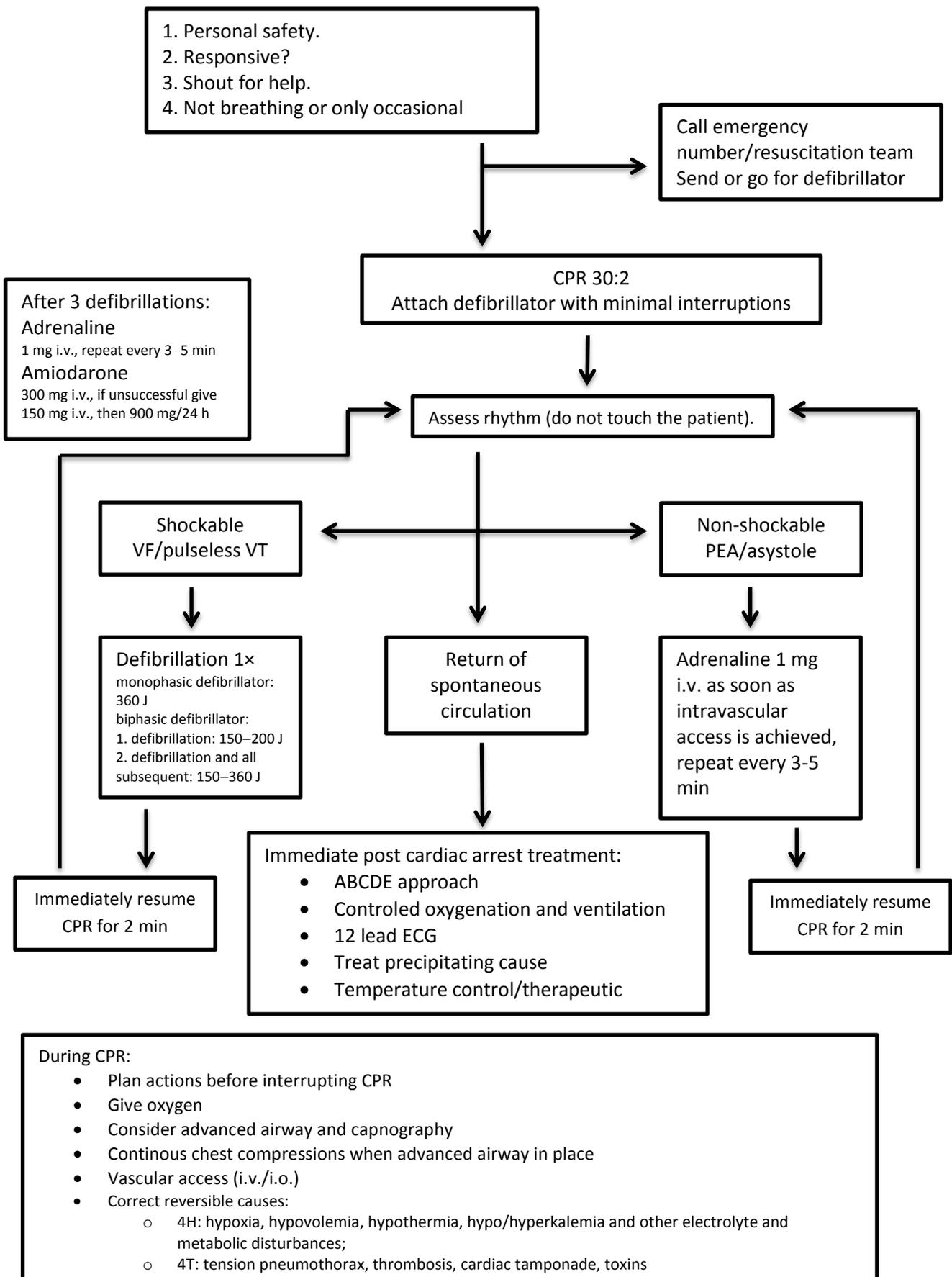
Algorithm 1. Bradycardia algorithm.



Algorithm 2. Tachycardia algorithm.



Algorithm 3. Adult advanced life support.



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SECTION 5: BASIC CLINICAL SKILLS

Authors: Tamara Todorovic, Karmen Zeme, Marko Zdravkovic, Sebastjan Bevc

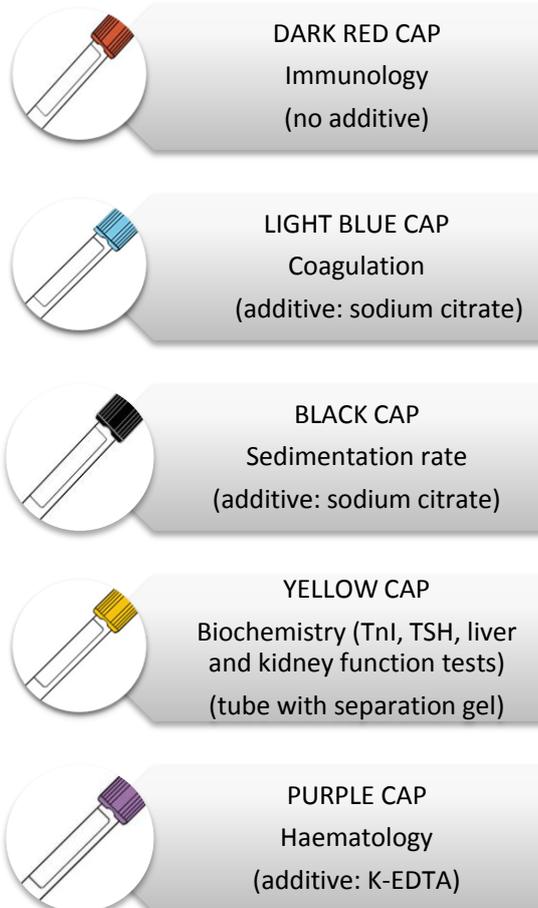
CONTENT	
1.	Theoretical Background
2.	Presentation of Simulation Scenario – Time Schedule
3.	Clinical Skills Protocols

5.1 Theoretical Background

Venipuncture

Venipuncture is an everyday medical procedure that can be performed by different types of medical personnel - doctors, nurses, medical students etc. When performing a venipuncture, always remember to confirm patient's identity, choose the appropriate tube(s) and to label the tube(s) correctly.

Tubes are selected according to the desired laboratory tests. Following tubes are commonly used in our environment:



Female urethral catheterization

In our environment, nurses tend to catheterize female patients if they are able to do so. If not, doctors have to step in, mostly in cases when catheterization is rather difficult. This is why female urethral catheterization should be learned by medical students and performed as many times as possible.

Always check for catheter size which is clearly marked on the wrapper of the catheter. For female patients, sizes 12 or 14 F usually suffice. A Foley catheter consists of the following parts:

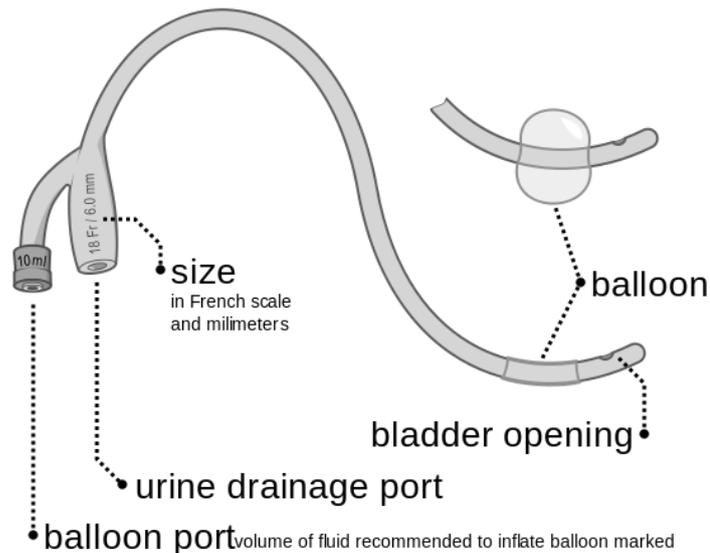


Figure 10: Foley catheter (source: www.wikimedia.org).

You should always work in an aseptic manner as catheters represent an important source of in-hospital infections.

Peripheral intravenous cannulation

Peripheral intravenous (IV) cannulation is a clinical skill that is usually taught early in the course of medical education.

When inserting a peripheral IV cannula, following things should be taken into account:

- Place a cannula as distal on the limb as possible. In this way, you can “save” the proximal portion of the vein for further cannulation in case of phlebitis, which is a common complication of the cannula insertion.
- In patients with dialysis AV-fistula, make sure to insert the cannula on the other hand or limb.
- There are several different sizes of IV cannulas:
 - yellow (24 gauge) – used in small children
 - blue (22 gauge) – used in children, adults with thin veins, cancer treatment patients
 - pink (20 gauge) – most commonly used cannula in adults
 - green (18 gauge) – surgical patients

- white (17 gauge) – used when there is a need of fast infusion of great amounts of fluids
- grey (16 gauge) – fast transfusion of blood and blood derivatives in case of a life threatening emergency
- orange (14 gauge) - fast transfusion of blood and blood derivatives in case of a life threatening emergency or reanimation
- Remember – the greater the cannula size (in gauge), the smaller the cannula diameter.

Intramuscular injection

Several different medicines can be injected via intramuscular route: haloperidol, chlorpromazine, diazepam, lorazepam, olanzapine, codeine, morphine, methotrexate, streptomycin, penicillin, interferon beta 1a, testosterone, ketamine, naloxone, vitamin B12, vitamin K.

General contraindications for intramuscular injection include:

- thrombocytopenia,
- haemophilia,
- anticoagulant treatment,
- local inflammation, hematoma etc.

Possible complications of intramuscular injection:

- infection,
- hematoma,
- pain,
- paraesthesia.

Subcutaneous injection

Insulin and low molecular weight heparin are usually injected via subcutaneous route.

General contraindications on the site of injection include:

- skin infection,
- skin oedema,
- skin scarring,
- hematoma.

5.2 Presentation of Simulation Scenario – Time Schedule

Introduction (Tamara Todorovic, Karmen Zeme, peer tutors)	
00.00 – 00.45	Demonstration of the procedures.
Simulation (Tamara Todorovic, Karmen Zeme, peer tutors)	
00.45 – 03.00	Practice in small working groups.

5.3 Clinical Skills Protocols

Venipuncture protocol (written by Sebastjan Bevc and Tamara Todorovic):

1	Disinfect your hands.
2	Introduce yourself to the patient.
3	Explain the purpose of the procedure to the patient.
4	Obtain patient's consent.
5	Put on the gloves.
6	Apply the tourniquet to the arm (10 cm above the chosen puncture site).
7	Identify a suitable vein by palpation and release the tourniquet.
8	Clean the puncture site using three cotton balls soaked with 70 % alcohol solution (using each once only and cleaning inside out).
9	Wait for the puncture site to dry.
10	Apply the tourniquet and ask the patient to clench a fist.
11	Attach the needle onto the holder and unsheathe it.
12	Warn the patient that you are about to start with the venipuncture.
13	Gently anchor the skin just below the puncture site using the thumb of your non-dominant hand.
14	Approach the skin and insert a needle at an angle of 20-40 degrees, about 1 cm deep (lumen of the needle is faced upwards).
15	Once the needle is in the vein, introduce the appropriate tube into the holder.
16	As soon as blood starts to flow into the tube, remove the tourniquet.
17	When blood flow ceases, gently disengage tube from holder and put the tube on the stand.
18	Place a clean cotton ball to the puncture site as the needle is gently withdrawn. Put the needle into sharps bin.
19	Pressure should be applied to the site until hemostasis occurs (app. 1 minute).
20	Check for any residual bleeding and apply sticky tape over the cotton ball.
21	Label the tube correctly.
22	Dispose used equipment and remove your gloves.
23	Thank the patient for cooperation.
24	Disinfect your hands.



Time limitation: 6 minutes

Female urethral catheterization protocol (written by Sebastjan Bevc and Marko Zdravkovic):

1	Introduce yourself to the patient.
2	Explain the purpose of the procedure to the patient.
3	Obtain patient's consent.
4	Place the patient's in an appropriate position: supine with hips abducted, knees flexed and heels together.
5	Unwrap the catheterization kit in an aseptic fashion (cotton balls, a kidney dish, a forceps).
6	Disinfect your hands and put on the sterile gloves.
7	Ask your assistant to unwrap a lidocaine gel, a catheter, a catheter bag and a 10 mL syringe onto your aseptic field in an aseptic fashion.
8	Ask your assistant to pour some saline into the kidney dish in an aseptic fashion. Fill the 10 mL syringe with a sterile liquid and also wet the cotton balls. Coat the distal portion of the catheter with sterile lidocaine gel.
9	Apply the sterile drape over the patient so the genitalia are exposed.
10	Use your non-dominant hand to hold the labia apart.
11	Clean the external genitalia with the wet cotton balls (using each once only) in a pubis-anus direction. Use forceps to hold the cotton balls.
12	Discard cotton balls into the kidney dish.
13	Apply some lidocaine gel to the external urethral orifice.
14	Insert the catheter into the urethral meatus and advance slowly until urine starts to drain. After that, insert the catheter for some centimeters further to ensure the balloon is beyond the urethra.
15	While performing previous procedure ask the patient if she experiences any pain.
16	Clamp the catheter at the urine drainage port.
17	Inflate the balloon with 10 mL of sterile liquid (use the 10 mL syringe) via the catheter side-arm (balloon port) and withdraw the catheter until the resistance is felt.
18	While inflating the balloon ask the patient whether she experiences any pain.
19	Remove the drape.
20	Connect the catheter to the catheter bag.
21	Remove the forceps to allow flow of the urine into the catheter bag.
22	Place the patient in a comfortable position.
23	Dispose used equipment.
24	Remove your gloves.
25	Thank the patient for cooperation.
26	Disinfect your hands.



Time limitation: 10 minutes

Peripheral IV cannulation protocol (written by Sebastjan Bevc and Karmen Zeme):

1	Disinfect your hands.
2	Introduce yourself to the patient.
3	Explain the procedure and its purpose to the patient.
4	Obtain patient's consent.
5	Place the patient in a comfortable position and seek for appropriate veins, preferably at a distal part of a non-dominant hand.
6	Place a towel under the chosen arm.
7	Put on the gloves.
8	Remove the appropriate cannula from its packaging.
9	Place the tourniquet 15 cm above the puncture site.
10	Identify a suitable vein by palpation and release the tourniquet.
11	Clean the puncture site using three cotton balls soaked with 70 % alcohol solution (using each once only and cleaning inside out).
12	Wait for the puncture site to dry.
13	Apply the tourniquet.
14	Unsheathe the needle.
15	Gently anchor the skin just below the puncture site using the thumb of your non-dominant hand.
16	Warn the patient that you are about to start with the procedure.
17	Approach the skin and insert a needle at an angle of 20-40 degrees (lumen of the needle is faced upwards).
18	Once the blood flashback is seen, continue with the insertion for approximately 3-5 mm, then lower the angle to 10 degrees and slide the cannula of the needle into the vein.
19	Release the tourniquet.
20	Place a gauze under the cannula.
21	Fix the cannula in place with a sticky dressing.
22	Gently unbind the cap from the needle.
23	With the forefinger of your non-dominant hand press over the vein at the tip of the cannula.
24	Remove the cap from the needle and then remove the needle itself while pressing over the vein at the tip of the cannula.
25	Put the cap on the cannula.
26	Dispose the needle into the sharps bin.
27	Flush the cannula with 5 mL of saline using the port on top.
28	Clean the skin area around the cannula.
29	Place date and hour of cannula placement on the sticky dressing.
30	Dispose used equipment and remove your gloves.
31	Thank the patient for cooperation.
32	Disinfect your hands.



Time limitation: 7 minutes

Intramuscular injection protocol (written by Karmen Zeme and Sebastjan Bevc):

1	Disinfect your hands.
2	Introduce yourself to the patient.
3	Explain the procedure and its purpose to the patient.
4	Obtain patient's consent.
5	Expose the intended site of the injection (upper outer quadrant of the gluteal muscle, upper outer brachial site, middle anterior third of the femoral area lateral from the middle line).
6	Place the patient into a comfortable position.
7	Put on the gloves.
8	Place a needle (e. g. 21 gauge) on a syringe.
9	Carefully unsheathe the needle.
10	Clean the medication bottle with an alcohol swab and draw up the medication.
11	Expel any air in the syringe.
12	Replace the needle with a smaller one (e. g. 25 gauge). Dispose the previous needle into the sharps bin.
13	Choose an appropriate injection site (without infection or a hematoma).
14	Clean the puncture site using three cotton balls soaked with 70 % alcohol solution (using each once only and cleaning inside out).
15	Carefully unsheathe the needle.
16	Hold the syringe between the thumb and the forefinger of the dominant hand.
17	Ask the patient to relax the muscles at the injection site as much as possible.
18	Insert the needle at full depth at a 90 degree angle.
19	Hold the syringe with your non-dominant hand and move your dominant hand to the plunger.
20	Draw back on the syringe to ensure you are not in vein.
21	Slowly inject the medication.
22	Place a clean cotton ball to the puncture site as the needle is gently withdrawn.
23	Put the needle into sharps bin.
24	Press a cotton ball on the injection site, gently massage it and apply pressure for approximately 1 minute.
25	Apply a sticky tape over the cotton ball.
26	Place the patient into a comfortable position.
27	Dispose used equipment and remove your gloves.
28	Thank the patient for his cooperation.
29	Disinfect your hands.



Time limitation: 7 minutes

Subcutaneous injection protocol (written by Karmen Zeme and Sebastjan Bevc):

1	Disinfect your hands.
2	Introduce yourself to the patient.
3	Explain the procedure and its purpose to the patient.
4	Obtain patient's consent.
5	Expose the intended site of the injection (upper middle brachial area, abdominal area between the navel and the upper iliac spine, middle outer third of the thigh).
6	Place the patient in a comfortable position.
7	Put on the gloves.
8	Choose an appropriate injection site (without infection or a hematoma).
9	Clean the puncture site using three cotton balls soaked with 70 % alcohol solution (using each once only and cleaning inside out).
10	Wait for the injection site to dry.
11	Carefully unsheathe the needle.
12	Hold the syringe between the thumb and the forefinger of the dominant hand.
13	Pinch a fold of skin with your non-dominant hand.
14	Inject the needle at 90 degree angle.
15	Hold the syringe with your non-dominant hand and move your dominant hand to the plunger.
16	Draw back on the syringe to ensure you are not in vein.
17	Slowly inject the medication.
18	Place a clean cotton ball to the puncture site as the needle is gently withdrawn.
19	Dispose the needle into the sharps bin.
20	Apply the pressure to the injection site with the cotton ball for approximately 1 minute.
21	Apply a sticky tape over the cotton ball.
22	Place the patient in a comfortable position.
23	Dispose used equipment and remove your gloves.
24	Thank the patient for cooperation.
25	Disinfect your hands.



Time limitation: 7 minutes

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SECTION 6: ELECTROCARDIOGRAPHY BASICS

Author: Andrej Markota, Miha Kodela, Tamara Todorovic, Karmen Zeme, Sebastjan Bevc

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

6.1 Theoretical Background

Electrocardiography is non-invasive, transthoracic recording of electrical heart activity detected by electrodes attached to predetermined positions on limbs and chest wall. The recording that is produced is called electrocardiogram. Electrocardiography is essential in measuring heart rate, determination of heart rhythm, diagnosis of acute coronary syndromes and contributes significantly to diagnosis of many other diseases (e.g. conduction abnormalities, pulmonary embolism, intoxication with many different toxins, congenital heart defects, heart failure, cardiac arrest). 3- or 5-lead electrocardiography is used to monitor heart rate and rhythm abnormalities. Diagnosis of acute coronary syndromes can only be made based on a 12-lead electrocardiogram. The leads used for 12-lead electrocardiogram can be divided into limb leads (I, II, III), augmented limb leads (aVL, aVL, aVR) and precordial leads (V1-6). Electrocardiographic recording of one heartbeat typically consists of a P wave, a QRS complex and a T wave. The orderly succession of P-QRS-T waves signifies organized passage of electrical current from sinus node, through atria, to atrioventricular node and then spreading all over the ventricles in a highly organized way. The isoelectric line of the electrocardiogram (a horizontal line denoting 0 mV) is the portion of electrocardiographic recording connecting the P wave and the following QRS complex, called the PR interval. Deflections of other parts of electrocardiographic recording are analyzed according to the PR interval, most importantly the deflections (either upward, termed elevations, or downward, termed depressions) of ST segment, which are crucial in the diagnosis of acute coronary syndromes. The speed at which the recording is made is usually 25 mm/s, and the vertical deflections are usually 10 mm/mV. Successful interpretation of a 12-lead electrocardiogram is needed in order to treat medical emergencies.

Step by step approach to the ECG interpretation

Following five major sections should be considered:

1. Measurements:
 - heart rate (state atrial and ventricular rate, if different),
 - PR interval (from beginning of P wave to beginning of QRS),
 - QRS duration (width of most representative QRS),
 - QT interval (from beginning of QRS to end of T),
 - QRS axis in frontal plane.
2. Rhythm analysis:
 - regular or irregular (map P-P and R-R intervals),
 - state basic rhythm (e.g. normal sinus rhythm, atrial fibrillation etc.) ,

- identify additional rhythm events if present .

3. Conduction analysis:

"Normal" conduction implies normal sino-atrial (SA), atrio-ventricular (AV), and intraventricular (IV) conduction.

- SA block,
- AV blocks: 1st, 2nd, and 3rd degree,
- IV blocks: bundle branch, fascicular, and nonspecific blocks.

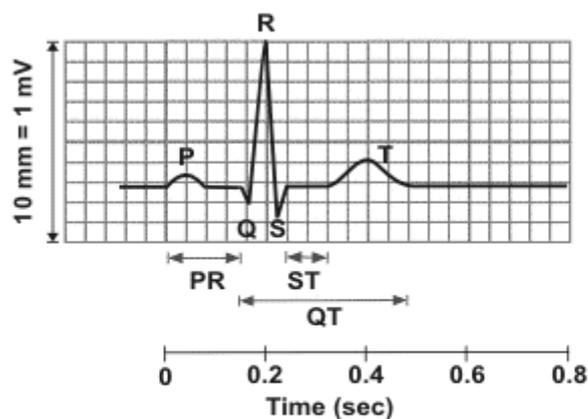
4. Waveforms description:

- P wave: if present, one per QRS, shape, duration, voltage,
- QRS complexes: look for pathologic Q waves, voltage,
- ST segments: look for abnormal ST elevation and/or depression,
- T waves: shape, look for abnormally inverted T waves,
- U waves: look for prominent or inverted U waves.

5. ECG abnormalities:

- life threatening arrhythmias (ventricular tachycardia/fibrillation, asystole),
- acute, subacute or old myocardial infarction,
- left ventricular hypertrophy.

If there is a previous ECG in the patient's file, the current ECG should be compared with it to see if any significant changes have occurred. These changes may have important implications for clinical management decisions.



P wave (0.08 - 0.10 s) QRS (0.06 - 0.10 s)
 P-R interval (0.12 - 0.20 s) Q-T_c interval (≤ 0.44 s)*

$$*QT_c = \frac{QT}{\sqrt{RR}}$$

Figure 11: EKG waves and intervals (source: www.cvphysiology.com).

6.2 Presentation of Simulation Scenario – Time Schedule

Introduction (Andrej Markota)	
00.00 – 01.00	Electrocardiography (ECG) basics – short lecture.
01.00 – 01.15	Break.
Simulation (Andrej Markota, Miha Kodela, Tamara Todorovic, Karmen Zeme, Sebastjan Bevc)	
01.15 – 03.00	ECG interpretation in small working groups.

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SECTION 7: CORONARY INTERVENTION

SIMULATION

Authors: Franjo Naji, Samo Granda, Marko Zdravkovic, Sebastjan Bevc

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

7.1 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 (atherectomy 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.

7.2 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 12: Simulator of Coronary Intervention in Action

7.3 Presentation of Simulation Scenario – Time Schedule

Simulation Part (Franjo Naji, Samo Granda, Marko Zdravkovic)	
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.
Clinical Part (Franjo Naji, Samo Granda)	
02.00 – 04.00	Visit of the coronary unit in the hospital.
Simulation Part (Franjo Naji, Samo Granda, Marko Zdravkovic)	
04.00 – 08.00	Further training in pairs on dilatation and stenting of the narrowed coronary vessel.

7.4 Case Presentation

Case 1: A 50 year old male smoker with typical history of stable angina (functional class II) was referred to the hospital for elective coronary angiography.

Cuff blood pressure: 147/76 mmHg

Heart rate: 85

Weight: 90 kg

References

1. Charles P. Davis, William C. Shiel Jr. MedicineNet.com. Coronary Angioplasty. MedscapeReference.com. Percutaneous Coronary Intervention. Available on: http://www.emedicinehealth.com/percutaneous_coronary_intervention_pci/page10_em.htm
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SECTION 8: LAPAROSCOPIC CHOLECYSTECTOMY **FOR CALCULOUS CHOLECYSTITIS**

Authors: Andrej Bergauer, Vid Pivec, Stojan Potrc, Vojko Flis

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

8.1 Theoretical Background

Introduction

A gallstone (cholelithiasis) is a benign condition which represents one of the most common “surgical” abdominal conditions. More than 70% of gallstones are formed by precipitation of cholesterol and calcium. The rest represent pigment stones, caused by precipitation of concentrated bile pigments (breakdown products of haemoglobin). The vast majority of gallstones are asymptomatic, often being identified at a time of abdominal imaging for other reasons or during abdominal surgery for another indication. Only 20%-30% of patients with asymptomatic gallstones will develop symptoms within 20 years. Because of the high incidence of this condition and increase in incidence with age (it is estimated that more than 60% of females over age of 70 years harbour gallstones), you will often encounter a case of symptomatic cholelithiasis or one of its sequel during your surgical/ medical clerkship or residency.

Acute calculous cholecystitis (acute inflammation of the gallbladder due to gallstones) is initiated by a gallstone, which obstructs the gallbladder’s outlet (either by obstructing cystic duct or by causing spasm of the musculature above the inflow to the cystic duct). If the obstruction is temporary and the stone is quickly dislodged and the obstruction alleviated, the result is a clinical picture of so-called biliary colic. An intense pain under right costal margin that can radiate to the back, under right shoulder blade or to the right shoulder is reported by the patient. Patient usually describes the pain being continuous, without interruptions. Thus, biliary colic is considered by some authors to be a misnomer. The pain can last from minutes to hours and disappear when the obstruction is alleviated. At the beginning of the biliary colic, vomiting is not uncommon. The patient is afebrile and tries to change position often in order to find a posture that would reduce the pain – a feature common to other spastic pain.

Clinical presentation and diagnostic procedures

Persisting impaction of the gallstone or obstruction in bile drainage from the gallbladder produces gallbladder distension and inflammation. Acute calculose cholecystitis ensues, the most common complication of gallstones. The gallbladder can also get inflamed without harbouring gallstones; however this is rare, 90% of all cases of acute cholecystitis are associated with cholelithiasis. It is also

associated with special subgroup of patients (polymorbid, immunocompromised, after severe injury or burns or patients with severe illness, receiving prolonged parenteral nutrition).

In the beginning stages, the inflammation is initially chemical – sterile. Gradually gut bacteria invade the inflamed gallbladder and infection supervenes. The combination of distended gallbladder with resulting ischemia of the wall and superimposed infection may result in a gallbladder empyema, necrosis, perforation, pericholecystitic abscess or even bile peritonitis.

The clinical presentation of biliary colic and acute cholecystitis is very similar in the early stages. It is of high importance to be able to differentiate between biliary colic and acute cholecystitis.

The classic clinical picture of acute cholecystitis is well summarized in almost any general surgery textbook. Fever, right upper quadrant pain, tenderness to palpation and possible guarding in the right upper quadrant with positive Murphy's sign and radiation of the pain to the epigastrium is something you know well. Let us concentrate on certain less well known features that might be of help.

The best discriminator between biliary colic and acute cholecystitis is time. Symptoms of biliary colic are self-limited and disappear in a matter of hours, while in acute cholecystitis, symptoms relentlessly persist. Acute cholecystitis is accompanied by local (local peritonitis or tender mass) and systemic (fever, leukocytosis) evidence of inflammation, while biliary colic is not. The patient is usually less restless than in biliary colic and tries to lie without much changing the position, in order to avoid pain with moving around (local peritonitis).

By performing a laboratory testing you might be able to underpin diagnosis of acute cholecystitis or to consider alternative diagnosis. Laboratory findings of leukocytosis, elevated C-reactive protein and slight elevation of bilirubin and liver enzymes may back up the diagnosis. Do not be tempted to dismiss the diagnosis of acute cholecystitis in absence of laboratory changes, especially with suggestive clinical presentation. The "labs" can be negative at the time of sample collection, but may be much different only a couple of hours later. It is the same as with acute appendicitis.

Mild to moderate elevation of bilirubin and hepatic enzymes is a relatively common feature of advanced acute cholecystitis (especially serum bilirubin and alkaline phosphatase). It is caused by reactive inflammation of the hepatic pedicle and the surrounding liver parenchyma. Unless there are also clinical and imaging features of ascending cholangitis or bile duct stones, you should not be tempted to simply attribute it to the cholangitis and thus refrain your patient from the best surgical management. The similar holds true to mild elevation of serum amylase (or lipase) – it does not automatically mean biliary pancreatitis unless there is a very suggestive clinical picture and imaging features.

Nowadays, the most simple and widely accepted imaging for acute calculose cholecystitis is transabdominal ultrasonography. Distended gallbladder with visible stones is a paramount sign. Combined with ultrasonographic Murphy's sign, it has a very high positive predictive value. Additional features are thickened gallbladder wall (with or without mucosal separation), pericholecystitic fluid collection, intramural gas or minute amount of free fluid in abdomen. The additional features are not necessarily present or pivotal to come to the diagnosis of acute calculose cholecystitis. Ultrasound scanning is good, since it can point out to pathology – e.g. detecting distended extra or intrahepatic bile ducts suggesting choledocholithiasis and initiating a different choice of further imaging and therapeutic interventions.

The other option is radionuclide scanning (hepatic iminodiacetic acid scan; so-called HIDA scan). The failure to fill the gallbladder with isotope within 2 hours after the injection of the isotope

demonstrates the obstruction of the cystic duct and is suggestive for acute cholecystitis. Positive scan means non filling of the gallbladder with the isotope. There are quite a few reasons for false positive test, however a negative scan (isotope entering the gallbladder within 2 hours) practically excludes acute cholecystitis.

Computed tomography scanning (CT) or magnetic resonance imaging has a controversial role in a non-complicated acute calculose cholecystitis and should not be the first choice for diagnostic imaging. It becomes important when the complications are suspected (magnetic resonance cholangiopancreatography (MRCP) for suspected choledocholithiasis – a non-invasive option to endoscopic retrograde cholangiopancreatography (ERCP) – if available).

Whichever test you will use, it is important to remember the fact, that you cannot diagnose acute cholecystitis when the gallbladder is not distended.

Treatment Plan

The simplest plan that is well suited to most of the patients (and surgeons alike) is to proceed to laparoscopic cholecystectomy. In majority of cases this is the optimal (and simplest) treatment plan. The inflamed and infected gallbladder is to be regarded as a “resectable infection” and treated as such. However medicine (or surgery) is not that simple and certain caveats should be considered.

Upon initial diagnosis of acute cholecystitis, initial therapy is directed towards general support of the patient, including fluid and electrolyte replacement, correction of metabolic imbalances and antibiotics. *Nill per os* (NPO) is instituted and nasogastric tube is indicated in severe vomiting. In most of the cases, the NGS is placed just prior to the operation in order to minimize gastric distension and facilitate operative procedure.

Considering the selection of antibiotic, it is important to know, that gram negative aerobe bacteria are the most commonly involved microorganisms. Anaerobes are detected in more than 15% of patients, but are rarely sole isolates. Selection of antimicrobial therapy is based on probable biliary pathogens and severity of the disease process. Broad spectrum antibiotics are warranted in the initial management. The usual selection would be cephalosporin of I, II or III generation (cefazolin, cefuroxime or ceftriaxone) or fluoroquinolone (ciprofloxacin, levofloxacin) as a monotherapy. In more severe infection piperacilin-tazobactam or imipenem-cilastatin or other carbapenems are an option. If the patient is severely debilitated or there are obvious signs of anaerobic infection on imaging, anaerobic coverage is warranted in the initial management (e.g. metronidazole). The length of the antibiotic coverage/ treatment is also linked to the extent of the disease and the patient condition. In cases with simple calculose cholecystitis there is only need for perioperative antibiotic coverage (usually 3 doses). In cases of gangrene of the gallbladder or empyema, the usual duration of antibiotic treatment would be 3-5 days postoperatively. In more severe cases with perforation or pericholecystitic abscess or biliary peritonitis, longer postoperative course of antibiotic treatment is warranted, but should be no less than 5 days (usually 7-10 days).

The cornerstone of treatment of acute cholecystitis is operative removal of the inflamed gallbladder. It is the optimal procedure since it eradicates the inflammation and prevents the recurrence. The timing of the operation can be either emergency procedure – “surgery tonight” (rarely needed), early procedure – “surgery tomorrow” (within 24-48 hours after admission; most of the cases) or delayed/ interval procedure (after approximately six weeks); once a popular option, in light of evidence based medicine only subgroup of patients benefits from an interval approach; patients with decompensated cardiac failure, gross coagulation disturbances or other co-existent medical illness that is decompensated.

Emergency cholecystectomy is to be performed following short resuscitation of the patient with clinical evidence of diffuse peritonitis and SIRS or frank sepsis. On imaging important clue is gas within gallbladder wall – features suggesting complicated cholecystitis (perforation, necrosis or empyema of the gallbladder). In this setting most of the surgeons would attempt laparoscopic cholecystectomy, but would consider early conversion to open cholecystectomy.

Early cholecystectomy (within 24-48 hours after admission) is the optimal choice for the patients that do not need an emergency operation. In this time-frame the conversion rates (from laparoscopic to open operation) are as low as 1.8% (compared to the delayed approach ranging from 11-32%). In the early phase of inflammation, operative dissection (open or laparoscopic) is easier – the inflammation and infection did not have time to produce fibrosis of tissue yet.

Delayed or interval cholecystectomy is reserved for the patients who have significant medical comorbidities or present late in the course of inflammation (they come to hospital 5 or even more days after onset of symptoms). The decision has to be individually made since approximately 20% of those patients fail medical treatment and need emergency surgical intervention before completing 6 weeks interval. Under those conditions, the operative procedure is challenging even to the experienced surgeon and carries higher risk for morbidity and mortality.

About 4-10% of patients who present with acute cholecystitis also have stones in the bile ducts. Acute cholecystitis is rarely associated with other complications of choledocholithiasis. In other words, acute cholecystitis combined with acute pancreatitis, ascending cholangitis, or pronounced jaundice is unusual. The emphasis is therefore on the treatment of acute cholecystitis, ductal stones are addressed as a secondary target.

The management of the patient with acute cholecystitis and suspected choledocholithiasis is directed by the severity of acute cholecystitis, imaging of the bile ducts and the general condition of the patient.

In the case of severe acute cholecystitis, mild elevation of bilirubin and hepatic enzymes and no dilatation of the bile ducts on sonography, laparoscopic cholecystectomy with intraoperative cholangiography is a reasonable option. If the stones are big and occlusive, conversion to open surgery is indicated and exploration and removal of common bile duct stones has to be performed. If the stones are small, they can be left in place and postoperative ERCP with sphincterectomy performed.

In the case of clinically mild acute cholecystitis, frank dilatation of bile ducts on sonography and liver function disturbances, the best is to initially treat cholecystitis conservatively (only antibiotics, delayed operation), MRCP or ERCP should be performed prior to operation, and the stones removed by endoscopic sphincterectomy.

In the severely ill/ septic patient the operation is performed on the emergency basis and the bile duct stones are preferably extracted via ERCP and EPT. Stent or nasobiliary drainage is usually placed additionally. Another less favorable option is intraoperative choledochotomy, cholangiography, stone extraction and placement of a T-drain.

Laparoscopic Procedure

Laparoscopic cholecystectomy is the procedure of choice for the acute calculose cholecystitis. Being minimally invasive (there are no big incisions and transection of the muscles) it offers several advantages over open cholecystectomy. After the laparoscopic procedure the length of stay is shorter, recovery is quicker, there is less postoperative pain and return to full activity is faster than in

open procedure. The mortality for the procedure is low (< 0.4%), the rate of iatrogenic complications is about the same as in open procedure and the length of surgery is not prolonged. (Providing that the concept of early operative treatment is adhered to, and conversion to open procedure is liberal in the setting of unfavorable conditions.)

The procedure was pioneered in Germany by Dr. Erich Mühe in 1985, followed by Dr. Phillippe Mouret of Lyon, France in 1987. The procedure was quickly embraced by the surgical society. The procedure is performed by first inserting a trocar or Veres needle into the abdominal cavity and insufflating the abdominal cavity with carbon dioxide to achieve pneumoperitoneum. After that, the optic system with camera is introduced and two or three more trocars are placed to insert the instruments. The instruments differ from classical surgical instruments since they are introduced into the abdominal cavity through small opening (trocars) and are manipulated from the outside of the body. After observing the anatomy through the camera and palpating the gallbladder with the grasper instrument, the decision is made whether to proceed with the laparoscopic procedure or convert to open operation. When the decision is made to go ahead with the laparoscopic procedure, the gallbladder is grasped at the fundus and retracted to the right shoulder by the grasper intended for the assistant. With the grasper in the left hand the infundibulum of the gallbladder is retracted laterally to the right in order to visualize the Calot's triangle (area bound by the cystic duct, common hepatic duct, and the liver margin). The dissection of the peritoneum is achieved by using a hook electrode or harmonic dissector). It should be always close to the gallbladder and above the Rouviere's sulcus of the liver (Rouviere's sulcus is a fissure on the liver between the right lobe and caudate process) in order to minimize the possibility to damage the common bile duct or right hepatic artery. The Calot's triangle is dissected in order to visualize the cystic duct and cystic artery – forming so called “two windows” – again safety is of the highest importance. After identification of the cystic artery and duct, both are closed off by using metallic clips. With clip applier, the clips are placed first on the cystic artery, one close to the gallbladder wall and two more approximately 1 cm distally. After that, cystic artery is divided by laparoscopic scissors. The cystic duct is clipped in the same fashion – one clip proximally, two distally, and divided by laparoscopic scissors. The gallbladder is then dissected free of its liver bed, observing that the dissection is done as close as possible to the gallbladder wall without puncturing it. Finishing the dissection of the gallbladder, the liver bed is checked for haemorrhage or bile leakage. The drain can be left in place in cases where bile leakage is of concern or the haemostasis was less than perfect. It is however not a routine to leave the drain in place. The gallbladder is placed in the endoscopic bag and retrieved through the umbilical port.

Complications of laparoscopic cholecystectomy

The complications of the laparoscopic surgery can be life threatening or cause severe mortality. It is important to be suspicious, especially where there are deviations from the normal course after laparoscopic cholecystectomy – recovery should be prompt, usually within first 24 hours, and the pain should be minimal. For a surgeon it is hard to consider that he might cause also harm, not only swift recovery from a potentially devastating disease. It is very important to remember that only surgeons who do not operate do not produce complications.

Hemodynamic instability, continuing pain or nausea/vomiting, abdominal distension, fever, and jaundice are all signs of complications. The abdominal pain could be localized (excluding trocar sites) or diffuse. If you have left a drain behind, any significant blood or anything bilious or resembling enteric content might suggest disaster.

Biliary leakage can originate from cystic duct, where the clip was misfired or dislodged, because of the unidentified and therefore unclipped accessory cystic duct or because of the iatrogenic damage to the common bile duct.

Common bile duct injury can be achieved either through malpositioning of the clips (so called tenting – when you clip both the cystic duct and the common bile duct – en masse), reckless electrocautery or sharp/blunt iatrogenic trauma. Either symptoms of obstruction or bile leakage can develop.

Bleeding can ensue after the operation, and can originate at the trocar sites, damage to the mesentery or major blood vessels upon initial access (positioning of the first trocar or Veres needle). Also inadvertent damage to right hepatic artery or dislodged clip from the stump of the cystic artery could happen. The liver bed, where the gallbladder was dissected from can be a source of the bleeding.

Inadvertent injury to the bowel may be a cause of complications that can be fatal if not considered early. Peritonitis which develops can be atypical, free air in the abdomen seen on plain X ray attributed to the insufflation with carbon dioxide, and free fluid seen on sonography also interpreted as a normal postoperative finding (as it can be).

8.2 Presentation of the Simulator

We will use two laparoscopic simulators in our Intensive Programme. The first one is low fidelity simulator, with pure mechanic parts and its indented use is to get acquainted with real laparoscopic instruments. Also, the basic technique for grasping and moving objects will be trained. The initial hand – eye coordination will be trained on that simulator. The main task will be to move coloured beads from one vessel to another first with right, and then with left hand.

The basic laparoscopic skills will be further trained on haptic laparoscopic simulator with force feedback (Simbionix LAP Mentor). There will be several tasks to complete – hand – eye coordination, clip application, clip application with tension and moving the objects with grasp – re/grasp technique. After mastering the basic skills and relevant anatomy, we will proceed to the whole procedure simulation, first with intensive tutoring, second with as little as possible of mentoring input.



Figure 13: Simulator of Laparoscopy Procedures in Action

8.3 Presentation of the Simulation – Time Schedule

Simulation Part (Andrej Bergauer, Vid Pivec)	
00.00 – 02.00	Basic laparoscopic techniques on low fidelity and high fidelity simulators; 2 pairs with rotation. Acquaintance with the simulator and equipment.
Clinical Part (Andrej Bergauer, Vid Pivec)	
02.00 – 04.00	Relevant anatomy refresh by using high fidelity simulator and video of the actual laparoscopic procedure; 2 pairs with rotation.
Simulation Part (Andrej Bergauer, Vid Pivec)	
04.00 – 08.00	Training of the whole procedure on high fidelity simulator with intensive tutoring and further training of basic techniques on low fidelity simulator; 2 pairs with rotation. Simulation of the whole procedure on high fidelity simulator with minimal tutoring and clinical case solving; 2 pairs with rotation.

8.4 Case Presentation

Case 1: A 42 years old Caucasian woman presents to the emergency department with pain in the right upper quadrant of the abdomen. The pain started in the evening the day before. She vomited twice, since the morning the sickness is gone, but the pain persists and radiates to the epigastrium. She had similar pain in the past, but it terminated by itself. She measured her body temperature at home and it was 37.6 centigrade. She passes urine and stool without any problems, does not describe any pain or burning sensation with micturition. The colour and the consistency of the stool did not change. Past medical history is only minor trauma with no pre-existent medical condition. She does not take any medication or hormone contraceptives. The menses is irregular; she might miss the last one. She does not smoke or ingest alcohol. She reports no allergies.

Case 2: A 56 years old male presents to the emergency department with chief complaint of severe pain in the epigastrium and right upper quadrant of the abdomen. The pain began 4 hours ago while walking upstairs to the first floor. He vomited yellowish slime once after the pain appeared. He describes pain as severe and incapacitating, he feels sick and out of breath. The pain is constant. He measured his body temperature at home and is 36.4 centigrade. He has a known cholecystolithiasis that produced colicky pain in the past, however, it was self-limiting. He rejected cholecystectomy since he was advised by his friends not to, because some of them have a lot of problems after the operation, not tolerating certain foods (he likes to eat well). He is concerned that he might have inflammation of gallbladder since his GP pointed him out in the past that this is a possibility. He smokes for at least 30 years, 20 cigarettes a day, has high blood pressure and hypercholesterolemia. He drinks occasionally; yesterday he might have a couple of drinks more than usually. He should take medication for hypertension and lipid lowering drugs; he takes them only occasionally since antihypertensive drugs made him cough and feel dizzy, after drug he was given for cholesterol, he had a lot of loose stools. He reports no allergies.

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