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O001 Indications for non-invasive and invasive mechanical ventilation

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Mechanical ventilation (MV) is a cornerstone in managing acute and chronic respiratory failure, providing full or partial support for respiration. MV is broadly classified into non-invasive ventilation (NIV) and invasive mechanical ventilation (IMV), with the decision-making process guided by clinical indications, the severity of respiratory compromise, and the potential for complications. This review explores the clinical indications for non-invasive and invasive mechanical ventilation while addressing the underlying pathophysiological rationale, the modalities employed, and the critical importance of appropriate monitoring.

Non-invasive ventilation (NIV) is primarily indicated in patients who retain their airway reflexes and do not require invasive airway management. It is commonly employed in the management of acute exacerbation of chronic obstructive pulmonary disease (COPD), acute cardiogenic pulmonary edema, and mild-to-moderate hypoxemic respiratory failure. NIV serves as a first-line intervention in these conditions to avoid intubation and its associated risks, such as ventilator-associated pneumonia (VAP) and trauma to the upper airway. Moreover, it plays a critical role during the weaning phase of mechanical ventilation, especially in patients transitioning from invasive ventilation. It facilitates the discontinuation of full ventilatory support while minimizing the risks of reintubation and prolonged ICU stays. The application of NIV significantly reduces complications associated with invasive techniques, shortens ICU stays, and lowers healthcare costs, thus representing a practical therapeutic approach for a subset of patients with respiratory failure.

In contrast, invasive mechanical ventilation is indicated when non-invasive methods are inadequate or contraindicated. This includes patients with severe respiratory failure where the ability to maintain adequate oxygenation and ventilation is compromised, as well as patients requiring airway protection due to altered mental status or an increased risk of aspiration (e.g., due to gastrointestinal bleeding or neurological deficits). Invasive ventilation is essential in the management of critically ill patients with acute respiratory distress syndrome (ARDS), severe hypoxaemia, or refractory hypercapnia. Furthermore, invasive MV is often required when non-invasive methods fail or are insufficient to support gas exchange, as in cases of progressive respiratory muscle fatigue or impending respiratory arrest.

The key non-invasive and invasive ventilation indications include respiratory pump failure, alveolar dysfunction, and complex respiratory pathologies, such as cardiogenic and non-cardiogenic pulmonary edema, airway obstruction, and vascular injuries (e.g., pulmonary embolism). MV alleviates hypoxaemia and hypercapnia by facilitating gas exchange through positive pressure ventilation, thereby reducing respiratory muscle workload and preventing further deterioration.

Both NIV and invasive MV require a careful selection of ventilation modes to meet the patient's specific needs. In NIV, pressure support ventilation (PSV) is frequently used to assist spontaneous breathing by delivering a set level of inspiratory pressure, reducing the patient's work of breathing. In invasive MV, volume-controlled ventilation (VCV) and pressure-controlled ventilation (PCV) control critical parameters such as tidal volume, airway pressure, and respiratory rate. The choice of ventilation mode is influenced by the underlying pathology, focusing on minimizing the risk of ventilation-induced lung injury (VILI), including barotrauma, volutrauma, and atelectotrauma. Appropriate adjustment of ventilator settings is critical to avoid exacerbating lung injury, particularly in patients with compromised lung mechanics, such as those with ARDS or COPD.

To ensure optimal outcomes, comprehensive monitoring is essential in non-invasive and invasive mechanical ventilation. In NIV, close monitoring of oxygen saturation, respiratory rate, and arterial blood gases (ABG) is required to assess the intervention's efficacy and detect early signs of NIV failure. More advanced monitoring is often necessary in patients receiving invasive MV, including continuous assessment of lung compliance, airway resistance, and detailed hemodynamic parameters. Imaging techniques such as chest radiography and lung ultrasound are valuable adjuncts in evaluating pulmonary function and detecting complications such as pneumothorax or pulmonary edema. In patients with COPD, careful attention must be given to avoid dynamic hyperinflation and auto-PEEP, conditions that can exacerbate respiratory compromise and cardiovascular dysfunction.

The weaning process from invasive mechanical ventilation is pivotal in patient recovery. Spontaneous breathing trials (SBT) assess the patient’s readiness to be weaned off mechanical support. NIV during this phase, particularly as a bridge following extubation, can mitigate the risk of extubation failure and reintubation in patients with borderline respiratory function. Successful weaning requires a thorough evaluation of respiratory mechanics, gas exchange, and the patient's ability to sustain spontaneous breathing without undue effort.

Moreover, ventilator-associated pneumonia (VAP) prevention and management are critical in patients undergoing invasive MV. VAP, which occurs in a significant proportion of patients on prolonged ventilation, is associated with increased morbidity and mortality. Strict adherence to infection control measures, regular oral hygiene, and timely initiation of antimicrobial therapies based on microbiological surveillance are essential in minimizing the incidence of VAP.

In conclusion, the indications for non-invasive and invasive mechanical ventilation are determined by the patient's clinical presentation, the severity of respiratory failure, and the need for airway protection. Both modalities are critical in managing respiratory insufficiency, carefully selecting the appropriate approach, ventilation settings, and monitoring strategies vital to optimizing patient outcomes. Future research should focus on refining weaning protocols, enhancing NIV efficacy, and

reducing complications associated with prolonged mechanical ventilation, particularly in high-risk populations.

O002 Who's still afraid of electricity?

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Sudden cardiac death (SCD) is a leading cause of death in the world (15-20%) with the majority of cardiovascular deaths (more than half). Malignant rhythms (such as ventricular fibrillation (VF) and pulseless ventricular tachycardia (pVT)) are causing SCD in patients with myocardial infarction in 3-12%, but the actual number is much bigger. Also, there are other reasons for SCD due to VF or pVT. Defibrillation is an indispensable treatment method in patients with SCD.
Methods: A review of current literature and European Resuscitation Council (ERC) protocols was performed.
Results: Early started resuscitation using defibrillation is the best treatment for patients with SCD and malignant arrhythmias (MA). Malignant rhythms are also called shockable rhythms based on defibrillator usage. Following ERC protocols one defibrillation shock is indicated in shockable rhythm, then immediately continuing chest compression for 2 minutes, and then new assess rhythm. After 3 shocks, amiodarone is indicated (300mg after 3rd shock, and another 150mg after 5th). If amiodarone isn't available (or local decision is made), lidocaine may be used (100mg after 3rd shock, and another 50mg after 5th). In the presence of refractory VF escalating shock energy and/or alternative defibrillation pad position is indicated (dual defibrillation is not recommended). We should always try to deliver safe and efficient defibrillation which is achieved by placing pads in the correct anterior-lateral position and more than 8cm away of some implantable device, minimizing the fire risk by placing oxygen mask or nasal cannula at least 1m away from patient's chest. Today we have two different groups of defibrillators based on shock techniques: monophasic and biphasic, with roughly the same efficacy in saving someone's life. Monophasic defibrillation sends an electric current in one direction (from a negative to a positive electrode) and delivering energy of 200–360 J. Biphasic defibrillation sends a current in two directions (from one electrode toward the other and then returns it back to the originating electrode) and delivering energy of 120–200 J. There are five main types of defibrillators: automated external defibrillators (AED), advanced life support defibrillators, implantable cardioverter-defibrillators, manual external defibrillators and wearable defibrillators. Most of these five types are biphasic defibrillators.
Conclusions: There are no contraindications for defibrillation. Early defibrillation is one of the most important survival factors for patients with SCD and MA.

O003 Mechanical circulatory support

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For patients presenting with refractory cardiogenic shock, hemodynamic stabilization and ensuring adequate perfusion of vital organs are often not possible after pharmacological therapy (inotropic and vasopressor stimulation), mechanical ventilation and treatment of the underlying cause of cardiogenic shock. In that case, for the purpose of maintaining adequate organ perfusion, myocardial relief and decongestion, mechanical circulatory support can be used as a treatment option, which can represent a bridge to recovery or a final decision, a bridge to permanent mechanical circulatory support or heart transplantation. The introduction of mechanical circulatory support in addition to the standard treatment of cardiogenic shock has opened up new possibilities for the treatment of cardiogenic shock.
Several devices are available for temporary mechanical circulatory support, which differ according to the achievable hemodynamic characteristics, the method and place of insertion, the size of the catheter and/or cannula, and whether they have an integrated gas exchanger (oxygenator). Among the devices used as temporary mechanical circulatory support are the intra-aortic balloon pump, percutaneous extracorporeal left heart support (Impella), extracorporeal mechanical circulation with oxygenator (ECMO) and iVAC 2L. The mentioned devices can be used in various configurations - independently or in combination, and their choice is based on clinical experience, the clinical condition of the patient, the amount of minute volume that needs to be ensured and their availability.

O004 "In the beginning, it was…" (case report)

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EIn patients with ST-segment elevation acute myocardial infarction (STEMI), primary percutaneous coronary intervention (pPCI) is the reperfusion treatment of choice, while fibrinolysis is usually reserved for cases where pPCI cannot be done due to the time frame of patient presentation (>120 min) or because it is not immediately available.
However, specific anatomical problems can result in failed pPCI, so "rescue" fibrinolysis remains an option in these patients, even though this type of treatment is not represented in the current medical literature.
A woman aged 64 was admitted to the emergency ward due to chest pain that began 2 hours before admission. Her risk factors for coronary artery disease (CAD) were hypertension, dyslipidemia, and smoking. Physical findings showed arterial hypotension and jugular vein distension.

The first obtained ECG showed first-degree AV block with marked ST elevation in leads II, III, aVF, V5, and V6 and ST depression in leads I, aVL, and V2.
The diagnosis of inferior STEMI was made, and she was referred to the cath lab for the treatment strategy of angiography with pPCI.
Due to the poorly palpable radial pulsations, the operator chose a transfemoral approach. In the cath lab, the patient developed a third-degree AV block, and a temporary pacemaker was implanted via the right femoral vein. Angiography of the left coronary artery (LCA) system showed no significant stenoses. However, even after aortography, the operator could not cannulate the right coronary artery (RCA) with several guiding catheters.
The patient developed further hemodynamic deterioration, and the team decided to introduce urgent thrombolysis by STEMI protocol with alteplase. In the hours after, she had two episodes of ventricular fibrillation that were terminated with cardiopulmonary resuscitation with defibrillation. Subsequently, the patient was rhythmically stable, and the temporary pacemaker was removed.
The echocardiography showed akinesia of inferoposterolateral walls of the left ventricle with reduced systolic function and right ventricle dysfunction.
After patient stabilization, coronary computed coronary angiography was obtained, and it showed a rare coronary anomaly – the origin of the RCA from the left sinus of Valsalva with critical stenosis in the proximal segment of the artery, which was completely patent. Using this knowledge, a second angiography and ad hoc PCI of RCA with implantation of one drug-eluting stent was performed.
The patient began rehabilitation and was discharged after 12 days, with no symptoms on the next check-up.
The anomalous origin of RCA is a rare anomaly with an estimated prevalence of less than 0.5%. Clear recommendations and treatment strategies for STEMI in these patients remain obscure. Although Rescue/"Bailout" fibrinolysis as a treatment strategy for STEMI after "failed" PCI is not represented in current recommendations, in some cases, it can be lifesaving.

O005 High-sensitivity cardiac troponin I as potential biomarker of cardiovascular risk for primary prevention

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Cardiovascular disease (CVD) remains the leading cause of mortality in developed and developing countries. Cardiovascular (CV) risk assessment based on the utilization of CV risk scores, enable clinicians to assess an individual's risk to experience CV adverse events. These risk scores that mainly include gender, age, smoking, systolic blood pressure, cholesterol levels, history of diabetes, have many limitations. We have witnessed that in recent years there is growing interest and discussion that the measurement of cardiac biomarkers could improve CV risk stratification. Recently, cardiac troponine I (cTnI) have been suggested and evaluated as an important independent risk factor for future adverse cardiac events, especially among asymptomatic ambulatory adults without a history of prior CVD (primary prevention). It is enabled by development of newer generations of high-sensitivity cardiac troponine (hs-cTn) assays, with improved analytical sensitivity and imprecision profile. A large number of meta-analysis and studies assessed the ability of hs-cTnI to detect individuals at higher CV risk in a general population. Proposed algorithms for the use of hs-cTnI in CV risk stratification in general population, with special focus on individuals with intermediate CV risk according to SCORE2 was recently published. The health economic consequences of using hs-TnI for assessing CVD risk in a general population will be discussed. Taking into account all the evidence so far available, it is necessary before the wider implementation of CV risk stratification and prevention strategies that incorporate hs-cTn, research should be conducted to define the optimal target populations, timing of measurement, and what should be the interventions in people with increased risk.

O006 Acute myopericarditis mimicking myocardial infarction: Case report

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The prevalence of acute myocardial infarction without coronary artery obstruction (MINOCA) that presents with ST elevation and is actually mimicked by acute myopericarditis is very low. Suspecting acute myopericarditis, given the wide variety of clinical presentations, and especially in the case of mimicking myocardial infarction, represents a significant clinical challenge.
CASE REPORT: A 46-year-old patient came to the emergency room due to pressure in the chest that occurred 90 minutes before admission, during the night, without accompanying symptoms and signs, without an infectious syndrome and exposure to significant noxia in the previous period. Risk factors for the development of coronary artery disease were smoking and obesity. Physical examination was normal. Electrocardiographically, ST elevations were seen in the inferior and anterolateral leads with reciprocal ST depressions with a negative T wave in leads aVL and V2. According to the protocol for acute coronary syndrome with ST elevations (ACS-STE), he was referred for urgent coronary angiography, which did not show significant stenosis on the coronary arteries. Upon admission to the hospital, laboratory analyzes showed the dynamics of myocardial necrosis markers indicative of acute myocardial injury, while other laboratory parameters, including inflammatory markers, were within the reference range. An echocardiographic examination showed

hypokinesia of the basal segments of the inferior, posterior and lateral wall and the apex itself with the limit values of the ejection fraction of the left ventricle. Given that all criteria were met for MINOCA, an entity that requires further evaluation of the underlying cause of myocardial injury, cardiac magnetic resonance (CMR) was performed. On the cine sequences, the left ventricle was seen with slightly reduced ejection fraction (EFLK 46.3%) and hypokinesia of the apical segment of the lateral wall. On T2-weighted Black Blood sequences, the existence of focal myocardial edema was determined, and on Late gadolinium enchancement sequences, focal accumulation of gadolinium in the subepicardial layer of the myocardium as well as in the corresponding segments of the pericardium was determined, without pericardial effusion. By correlation with the other clinical findings, the CMR imaging of the heart indicated acute myopericarditis as the main cause of acute injury and dysfunction of the myocardium.
CONCLUSION: Acute myopericarditis is a significant imitator of diseases of the cardiovascular system, if we suspect it and direct the diagnostic protocol in the right direction, we will be able to make an accurate diagnosis in a timely manner and menage the patient in proper manner.

O007 Akutni koronarni sindrom kod boelsnika sa preuranjenom koronarnom bolesti

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Premature coronary disease (PCD) is not rare among patients who are hospitalized due to acute coronary syndrome (ACS) and according to data from studies, as many as 25% of hospitalized patients with ACS have PCD. Today, it is recommended that coronary artery stenosis of more than 50% in men younger than 50 and women younger than 55 is considered under PKB.
Patients with PCD are more likely to smoke, have hyperlipidemia (including familial forms), as well as a family history of coronary disease compared to older patients. Also, in younger patients there are more often so-called "non-traditional" risk factors for coronary disease such as obesity, thrombophilia, and in women (in the reproductive period) sometimes the causes can be disturbed, usually lower, estrogen levels. In patients with PCD, the first presentation of coronary disease is usually myocardial infarction with ST segment elevation and/or sudden cardiac death.
In general, mortality in patients with PCD is significantly lower compared to older patients, but many studies have shown that the incidence of non-fatal adverse ischemic events does not differ significantly compared to the older population. Also, in patients with PKB, repeated non-fatal ischemic events are much more often conditioned by the progression of atherosclerosis and the appearance of stenosis on other coronary arteries, compared to older patients in whom changes in the same blood vessel (e.g. stent restenosis) are a more frequent cause of repeated recurrent ischemic events. These findings indicate the enormous importance that secondary, and above all primary, prevention should have in young people.

O008 Varieties of anticoagulant therapy in the treatment of patients with myocardial infarction

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Timely recognition of different forms of acute coronary syndrome and complications arising from the nature of the disease itself or previous therapy and comorbidity requires appropriate anticoagulant therapy.
The standard application of unfractionated (UFH) and low molecular weight heparins (LMWH) is still the basic anticoagulant therapy for patients with myocardial infarction. Recent findings and recommendations suggest the use of an intravenous bolus of enoxaparin 0.3 mg/kg in those whose last LMWH s.c. dose was 8 hours before percutaneous coronary intervention (PCI). Particular attention is drawn to reducing the dose of LMWH in patients with renal insufficiency with eGFR<30 ml/min by 50%, i.e. to the use of a single dose instead of a therapeutic dose prescribed twice a day.
In non ST elevation myocardial infarction (NSTEMI) in which PCI is not planned in the first 24 hours, the administration of fondaparinux is recommended.
It is encouraged to perform PCI without delay, in patients who were previously treated with direct oral anticoagulants (DOAC) with the note that i.v. UFH bolus 70–100 IU/kg must be applied before intervention. Intravenous bolus UFH is also used in those on vitamin K antagonist therapy (VKA) whose INR is <2.5.
Enoxaparin is the parenteral anticoagulant of choice in patients treated with fibrinolytic therapy, with a note that the dose must be adjusted to age (>75 years<) as well as the degree of renal function (<30 ml/min/1.73m2). For those younger than 75 who have been treated with fibrinolytic therapy, i.v. bolus 30 mg with further subcutaneous administration at a dose of 1 mg/kg every 12 hours for 8 days.
Special attention is paid to the therapy of detected left ventricle parietal thrombi, in whom, in addition to standard therapy with heparin preparations (UFH,LMWH) and VKA,

DOAK anticoagulant therapy gets an increasingly important place.
The use of anticoagulant therapy with heparin derivatives requires regular monitoring of the number of platelets, clinical and laboratory monitoring for the detection of heparin-induced thrombocytopenia (HIT). Bivalirudin and argatroban represent valid and globally available therapeutic options in patients with HIT treated with PCI.
The introduction into practice of an increasing number of new anticoagulant drugs with all their advantages and potential disadvantages obliges the clinician to establish an optimal antithrombotic regimen adapted to each patient individually, taking into account all his comorbidity.

O009 Elevated D dimer – what do we do next?

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D-dimer is a biomarker of fibrin formation and degradation. It is commonly used in clinical practice to exclude a diagnosis of pulmonary embolism or deep vein thrombosis. Also, high values of D-dimer might indicate physiologic conditions, including aging and pregnancy. In addition, some pathological conditions have been associated with increased D-dimer levels, such as disseminated intravascular coagulation, infectious diseases, hemorrhages, trauma and cancers.Primary cardiac tumors are very rare with an incidence rate less than 0.03%, and they can be separated into benign (75%) and malignant (25%).Sarcomas represent almost all primary malignant tumors. The most common is angiosarcoma (33%), then rhabdomyosarcoma (20%), mesothelial sarcoma and malignant fibrous connective histiocytoma.The prognosis of malignant tumors is poor, and the survival time is only a few months to years.A 69 year old female was hospitalized due to high fever and positive acetone in the urine, and also having palpitations. In her laboratory results, one of the parameters which stood out was the high value of D-dimer.CT pulmoangiography did not show signs of pulmonary embolism. Transthoracic echocardiography showed large cardiac mass, that fills almost the entire right atrium. Patient was represented to the Cardiac Surgery Medical Advisory Board of IKVB „Dedinje“. The patient consented to surgery, and the specimen was sent for pathological examination. Histology and immunohistochemistry showed the definite diagnosis, and chemotherapy was further treatment. One month after the operation, repeat echocardiography showed a mass, high likely a relapse.Unfortunately, 10 months after the diagnosis, the patientdid not survive. This case demonstrates the importance of considering a primary cardiac malignancy in the differential diagnosis.

O010 MINOCA vs. myocarditis - from diagnosis to therapy

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MINOCA (Myocardial Infarction with Non-Obstructive Coronary Arteries) is characterized by acute myocardial infarction signs without significant coronary artery stenosis (<50%). It represents approximately 6% of all acute myocardial infarction cases. The clinical presentation and risk factors are similar to coronary heart disease, except that MINOCA predominantly occurs in younger individuals and more frequently affects women.Pathophysiological mechanisms include coronary artery spasm, spontaneous artery dissection, embolization, thrombosis and microvascular dysfunction. Myocarditis, a common differential diagnosis of MINOCA, is an inflammatory condition often caused by infections or autoimmune disorders and is frequently associated with elevated myocardial necrosis biomarkers.
Case report: In this study, we present the case of a 32-year-old woman with acute myocardial infarction without coronary artery obstruction and symptoms suggestive of myocarditis. One month postpartum, she experienced fatigue, palpitations, and chest pain radiating to the spine and left arm. Her medical history included a recent upper respiratory infection with fever and no known risk factors for coronary disease. Blood tests revealed elevated hsTnI (maximum 1329 ng/l), mild monocytosis, and lymphocytosis, with other parameters normal. Electrocardiogram showed sinus rhythm (HR 80/min), without ST or T changes. An echocardiogram revealed a normal-sized left ventricle with preserved systolic function, no segmental motion abnormalities, and a hyperechogenic pericardium. Given her age, absence of typical risk factors for myocardial infarction, recent infection, monocytosis, lymphocytosis, and mildly elevated hsTnI, we initially considered myocarditis as the diagnosis. However, cardiac MRI (Magnetic Resonance Imaging) showed focal subendocardial necrosis of the inferior wall of the left ventricle, likely caused by MINOCA. The final diagnosis of MINOCA was confirmed by CT coronary angiography, which showed no coronary artery narrowing.
Conclusion: MINOCA and myocarditis present with similar symptoms, which can complicate diagnosis, especially in younger adults where myocarditis may be more commonly considered. As demonstrated in this case, where MINOCA was not initially suspected, it is crucial to use cardiac MRI to differentiate between these conditions. Cardiac MRI should be performed in all cases suspected of myocarditis to exclude MINOCA and other potential diagnoses, ensuring accurate treatment and preventing complications.

O011 STEMI - Do we always have control? A case report of a patient with cardiogenic shock as a complication of acute myocardial infarction with ST elevation in the anterolateral localization

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Cardiogenic shock is one of the most significant and potentially fatal complications of myocardial infarction. The incidence of cardiogenic shock as a complication of acute coronary syndrome is 4-12%. The aim of this paper is to present a patient with a myocardial infarction who, after surviving cardiac arrest, underwent revascularization within the optimal time frame, despite which cardiogenic shock developed immediately upon admission to the coronary unit.
A 55-year-old patient was admitted due to a myocardial infarction with ST elevation in anterolateral leads. The admission to our hospital occurred approximately one hour after the onset of anginal symptoms. During transportation in the ambulance car, cardiopulmonary arrest occurred, and resuscitation measures were successfully carried out. Upon admission, emergency coronary angiography was performed, revealing a thrombotic occlusion of the ostial left anterior descending artery. Manual thromboaspiration was performed, followed by pPCI (primary percutaneous coronary intervention) of the LM-LAD with the implantation of a drug-eluting stent. Despite revascularization being performed within the optimal time frame, shortly after admission to the coronary unit, the patient presented with symptoms and signs of pulmonary edema and cardiogenic shock. Repeated ECG recordings showed no evolutionary changes. With timely revascularization and well-controlled medication support, significant clinical improvement was achieved in the patient.
Cardiogenic shock is a complex clinical syndrome with an exceptionally high mortality rate. In our patient, given the occurrence of cardiac arrest and the location of the "culprit" lesion at the bifurcation of the left main coronary artery, there was a high risk of developing cardiogenic shock as well as a fatal outcome. However, thanks to the pPCI performed within the optimal time frame and well-controlled medication support, a fatal outcome was prevented, and a good therapeutic response was achieved, resulting with recovery of myocardial function.

O012 Periprocedural complications in cath-lab

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Invasive cardiology diagnostics and percutaneous coronary interventions play a major role in the timely diagnosis and treatment of coronary artery disease. However, these procedures carry a certain degree of risk, including complications during or immediately after the intervention, known as periprocedural complications.
It is important to point out the wide range of periprocedural complications that can occur in the cath lab, from complications related to vascular access, such as hematoma and pseudoaneurysm formation, complications on the coronary arteries themselves, such as dissection and perforation, to embolic events such as periprocedural myocardial infarction, stroke, early stent thrombosis, etc. Various strategies to alleviate these complications are being considered, including advances in procedural techniques and new intravascular imaging techniques. What is important to point out is that adequate patient selection and thorough preprocedural treatment play a major role in reducing the incidence of periprocedural complications in interventional cardiology.
Understanding and solving periprocedural complications are essential for improving the safety and efficiency of percutaneous coronary interventions, which will be explained in the lecture and through a case presentation.

O013 Elevated troponin levels – are they always an alarm for myocardial infarction?

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Troponin is a protein complex that is involved in the contractile process of skeletal and cardiac muscle. Cardiac troponins are very sensitive and specific markers of myocyte necrosis and as such present a diagnostic method that is widely used for detection of even small amounts of myocardial necrosis. The new high sensitivity troponin methods allow detection of very minor damages of the heart muscle, increasing the number of patients with elevated troponin levels and thus hampering the interpretation of troponin results. Besides acute myocardial infarction (AMI), there is a wide specter of potential diseases with troponin release, including non-ischaemic causes such as dilated non-ischaemic cardiomyopathy (DCM). DCM is characterized by the presence of left ventricular dilatation and systolic dysfunction in the absence of coronary artery disease or abnormal loading conditions. Several pathophysiological factors (neurohormonal activation, inflammation, metabolic dysfunction, microvascular ischaemia) have been advocated to explain subtle ongoing necrosis in DCM, creating a myocardial injury and thus elevated troponin levels. Here is an illustrative case of a 50-year old patient with non-ischaemic dilated cardiomyopathy who presented himself with symptoms and signs of acute myocardial infarction with maximum hsTnI levels of 2006 ng/L. Transthoracic echocardiography showed enlarged left ventricle with reduced ejection fraction and multiple wall motion abnormalities, after which a coronary angiogram was done that showed coronary arteries without obstructions. Having done a cardiac MRI, his condition was characterized as alcohol-induced non-ischaemic dilated cardiomyopathy.

O014 Initial approach to cardiogenic shock

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Cardiogenic shock is a condition of critical tissue hypoperfusion resulting from primary ventricular dysfunction.

A number of multicenter randomized clinical trials have defined cardiogenic shock (CS) in different ways. The official recommendations of the European Society of Cardiology (ESC) from 2021 define cardiogenic shock as systolic blood pressure < 90 mmHg with clinical and/or laboratory signs of hypoperfusion. Clinical signs include: presence of cold extremities, oliguria, mental confusion, weakness, and narrow pulse pressure, while laboratory signs include: metabolic acidosis, elevated serum lactate, and elevated serum creatinine.

Despite progress in the diagnosis and treatment of CS, in-hospital mortality, regardless of the cause of CS, is 21-50%, while 30-day mortality is 30-50%. In advanced stages of CS, the in-hospital mortality rate is up to 70-80%.

Similar results were obtained in a sample of patients treated at the Institute for Cardiovascular Diseases of Vojvodina due to CS during 2022/2023, where in-hospital mortality in patients with CS caused by acute myocardial infarction (AMI) is 49.4%, while in CS caused by other etiologies is 88.9%. Mortality rate increases in advanced stages of CS up to 98.4% in refractory CS.

The etiology of CS is diverse, and myocardial disease, with acute coronary syndrome, accounts for 81% of all causes of CS. Other causes include: diseases of native and artificial heart valves, electrical disturbances, extracardiac (obstructive) and other causes. According to the time of occurrence, CS can occur as a consequence of an acute cardiac event or as a decompensation of chronic heart failure.

In 2018, the Society for Cardiovascular Angiography and Intervetions (SCAI) shock classification was published, which classifies CS into 5 stages (A-E) depending on its severity. Due to the inconsistencies in the definition, which was descriptive, in 2022 this classification was supplemented with clinical and laboratory parameters, which clearly set the definition of each of the stages of CS and talk about the necessity of applying mechanical circulatory support.

According to this classification, patients who are hemodynamically stable but who are at risk of developing CS, such as patients with a large AMI or decompensated heart failure, belong to stage A CS. Patients who are hemodynamically unstable, who do not require hemodynamic support and do not have clinical signs of hypoperfusion belong to stage B CS, and intensive monitoring is recommended for them. Patients in stage C CS have a developed clinical picture of CS, are hypotensive and hypoperfused, which is defined by values of mean arterial pressure (MAP) < 65 mmHg and values of serum lactate 2-5 mmol/l without or with one vasopressor. Stage D CS develops in patients who, despite the intensification of therapy, experience clinical deterioration, which is defined by MAP values of 50-65 mmHg and serum lactate values of 5-10 mmol/l with the use of two or more vasopressors/inotropes. Stage E represents refractory CS with MAP values < 50 mmHg and serum lactate values > 10 mmol/l or with refractory cardiac arrest. In stages C, D and E, mechanical circulatory support either in the form of an axial pump or venoarterial (V-A) ECMO may be appropriate.

In evaluating and prognosticating patients with CS, the SCAI proposed a three axis model, which uses three separate entities. The first entity represents the severity of CS, which is determined by the SCAI shock classification, hemodynamic parameters, metabolic disorders and vasopressor toxicity. The second entity is the phenotype and etiology of CS and is determined by acute heart failure or exacerbation of chronic heart failure, clinical etiology, left/right or biventricular heart failure, cardiac or cardiopulmonary decompensation, congestion profile, and biochemical phenotype. The third entity is represented by risk modifiers which include risk factors that cannot be influenced, such as gender, age, patient comorbidities, cardiac arrest with comatose state of consciousness, presence and reversibility of organ failure, activation of systemic inflammatory response and weakness and risk of developing complications.

In the initial approach to a patient with CS, the recommendations for clinical practice are that, in addition to a clinical examination, all patients with CS should undergo an electrocardiogram, chest radiograph, echocardiographic examination, computed tomography and transesophageal echocardiographic examination (if there are indications for them), and from mandatory laboratory tests: serial high-sensitivity troponin, complete blood count, electrolytes, creatinine, liver function tests, arterial gas analysis and serum lactate.

With the aim of timely diagnosis and initiation of therapy, a model of centers specialized in CS was recently introduced, which by definition would represent tertiary hospitals or cardiovascular centers with a large number of patients and which have intensive cardiac care units with a sufficient number of staff available, continuous monitoring of all vital parameters, mechanical ventilation, invasive hemodynamic monitoring, administration of vasoactive drugs, the possibility of placing temporary pacemakers, continuous hemodiafiltration. Centers specialized in cardiogenic shock should have 24 hours/7 days available percutaneous coronary intervetions (PCI), 24h available echocardiography and mechanical circulatory support options, which include intra-aortic balloon pump, V-A ECMO, as well as axial pumps (Impella) and availability of cardiac surgery. These centers must have a number of expert consultants: cardiologists, intensivists, interventional cardiologists, pulmonologists, nephrologists, neurologists, palliative care professionals. Professional consultants in CS centers include: pharmacologists, social workers, respiratory and physical therapy, dietitian, and CS centers should also meet academic standards in terms of participation in national and international registries for CS, as well as medical staff education.

In its latest recommendations from 2021, the ESC suggests that in the initial treatment of patients with CS, the etiology of CS (acute coronary syndrome or other) and the presence/absence of mechanical complications should first be

determined. In the case that acute coronary syndrome (ACS) is the cause of CS, urgent PCI or surgical revascularization is advised, and in case of other causes, identification and treatment of them. Regardless of the etiology of CS, in all patients it is necessary to consider the use of oxygen therapy and ventilatory support, to consider the use of vasopressors and/or inotropes, and to consider the use of short-term mechanical circulatory support. In the event that hypoperfusion and organ dysfunction are corrected, it is advised to wean off mechanical circulatory support and/or vasopressors/inotropes, and if despite the applied therapy, further progression of CS occurs, long-term mechanical circulatory support and hemodialysis should be considered, while the the possibility of palliative care should always be considered (4). The American Association of Cardiologists gave similar recommendations as the European one.

Current ESC recommendations do not advise the routine use of IABP in CS, especially in patients with ACS and without mechanical complications, while short-term mechanical circulatory support may be considered in patients with severe/refractory CS.

The results of recently published studies in the field of CS resulting from AMI, ECLS-SHOCK and ECMO-CS do not support the routine application of short-term mechanical circulatory support in this group of patients. Much is expected from the results of numerous randomized prospective clinical studies that are currently underway, with the aim of improving the long-term survival of patients with CS.

O015 Assisted modes of ventilation

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Assisted/spontaneous modes of mechanical ventilation constitute a crucial phase in the progression towards resuming spontaneous, independent breathing in mechanically ventilated patients. In rare instances, these modes may serve as the sole applied modes of ventilation (when maintenance of the patient's spontaneous breathing is necessary). The primary objective is to transition the patient from controlled to assisted ventilation at the earliest opportunity, once conditions permit, and subsequently to wean the patient from mechanical ventilation as soon as feasible, while avoiding premature discontinuation. Timely intubation, appropriate mechanical ventilation, and prompt weaning and extubation are critical components in the recovery process of any intubated and mechanically ventilated patient, regardless of whether the underlying condition involves heart failure, surgical intervention, septic shock, neurological or neurosurgical disease, or trauma.

In addition to adequate ventilation, which ensures satisfactory gas exchange and is achieved by optimizing ventilation parameters, precautions must be taken to prevent adverse effects from mechanical ventilation (e.g., pneumothorax, pneumomediastinum, volutrauma, barotrauma, biotrauma). Therefore, it is essential to ensure maximum synchronization between the patient and the ventilator to reduce the patient's respiratory effort and achieve the predetermined objectives of mechanical ventilation. This necessitates continuous monitoring and real-time adjustment of ventilation parameters to achieve optimal outcomes (adequate gas exchange, appropriate airway pressures, and patient-ventilator synchronization). This process is dynamic and requires substantial dedication, carrying significant responsibility for the physician, a phenomenon particularly observed during the COVID-19 pandemic.

Sedation, and particularly muscle relaxation, should be avoided when feasible to prevent patient anergy, prolongation of mechanical ventilation, ventilator-associated infections (pneumonia), and secondary septic conditions in treated patients. Of paramount importance is the prevention of diaphragmatic atrophy, as the primary objective is for the patient to overcome the crisis with minimal discomfort and return to independent, spontaneous breathing, which encompasses coughing.

Patients are typically initially ventilated utilizing one of the controlled modes, subsequently transitioning to one of the spontaneous/assisted ventilation modes as a step in the weaning process. In some instances, following a period of assisted ventilation, the patient may necessitate a return to a controlled mode, contingent upon their overall clinical status and the efficacy of treatment, as well as the objective prognosis for recovery.

The most frequently utilized modes of assisted ventilation, providing optimal comfort and synchronization, are Pressure Support Ventilation (PSV) and Proportional Assist Ventilation (PAV). The patient initiates each breath (inspiration), which is supported by the ventilator (in pressure support) to achieve optimal tidal volume and minute ventilation. The respiratory rate (frequency of breathing) is also determined by the patient. In contrast to PSV, where each breath is supported by the same pressure support (predetermined by the physician to achieve optimum tidal volume through monitoring and titration), in PAV mode, the ventilator delivers proportional support (contingent upon the patient's effort and work of breathing). The work of breathing (WOB) is calculated by the software based on predefined values. Consequently, if the WOB increases (patient's effort increases), ventilator support increases proportionally. This mode of ventilation is highly comfortable for the patient and is utilized for weaning. However, it is essential to monitor and clinically evaluate each ventilated patient: hemodynamic stability, respiratory rate, breathing pattern and mechanics, signs of distress, agitation, fatigue, or excessive perspiration - in order to adjust ventilation parameters or transition to an alternative mode of ventilation. This is particularly crucial for patients on prolonged mechanical ventilation. It is also imperative to assess the appropriate timing for tracheostomy, as there is a risk of tracheal wall damage in intubated patients on prolonged mechanical ventilation. Over time, pressure

support is decreased, along with FiO2 and PEEP, and when optimal conditions are achieved, it is safe to extubate the patient.

Volume Support Ventilation (VSV) utilizes Psupp for spontaneous breaths; however, the optimal tidal volume (VT) is predetermined by the physician and is ensured. Based on software calculations performed during the previous respiratory cycle, the ventilator adjusts the necessary support for the patient's effort to achieve the specified tidal volume.

Airway Pressure Release Ventilation (APRV) is frequently employed for recruitment and for the ventilation of patients with ARDS and polytrauma. Spontaneous breathing occurs at two levels of pressure (similar to BILEVEL). The release of pressure enhances the elimination of CO2. The I:E ratio exceeds 10. This mode is contraindicated in patients with COPD and in cases of bronchopleural fistula. In certain instances, it can significantly improve ventilation. By preventing alveolar collapse, the pressure required for alveolar reopening is reduced. This is particularly significant considering that surfactant production is diminished in mechanically ventilated patients. Conversely, care must be taken to avoid overdistension of healthy alveoli and prevent barotrauma.

Adaptive Support Ventilation (ASV) is arguably the most advantageous mode for physicians less experienced with mechanical ventilation. It is applicable for both controlled and assisted ventilation, including weaning processes. The system employs breath-by-breath evaluation of compliance, resistance, and work of breathing (WOB) for optimization. The software calculates optimal minute volume based on the input parameters. Patient height is utilized to determine the ideal body weight. The parameters adjusted by the physician include the percentage of WOB performed by the ventilator (percentage of assistance), positive end-expiratory pressure (PEEP), and fraction of inspired oxygen (FiO2). In cases where the patient is not breathing spontaneously, the ventilator assumes full responsibility for the WOB, delivering optimal respiratory rate (RR) and tidal volume (VT). Real-time plotting is displayed on the ventilator screen. For patients breathing spontaneously, the physician gradually reduces the percentage of ventilator assistance. WOB, clinical parameters, and breathing pattern are continuously monitored. Prior to patient extubation, arterial blood gas analyses are performed and evaluated.

The significance of understanding and executing mechanical ventilation effectively became evident during the COVID-19 pandemic. It is imperative to include as many physicians as possible to perform mechanical ventilation on patients appropriately. Continuing education is essential for this process to optimize outcomes and minimize the incidence of adverse events.

O016 Acute massive pulmonary thromboembolism masked by ST-segment elevation: Case report

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Acute massive pulmonary thromboembolism, masked by ST-segment elevation in leads oriented towards the right ventricle (RV) is exceedingly rare. The presence of ST-segment elevation can complicate the diagnostic process. Recognizing this uncommon electrocardiographic (ECG) pattern is crucial for facilitating prompt and appropriate therapeutic management. Although a few case reports describe the ECG presentation of pulmonary embolism as ST-segment elevation in precordial leads, the exact mechanism remains uncertain.

CASE REPORT: A 60-year-old male presented to the emergency room with sudden dyspnea and loss of consciousness on two occasions, denying chest pains. He reported a history of prostate cancer currently on biological and corticosteroid therapy. Physical examination was normal. The initial electrocardiogram revealed right bundle branch block (RBBB) with ST elevation in leads V1-V3 and elevation in aVR, accompanied by ST depression in V4-V6, DI, and aVL. Subsequent ECG showed elevation only in lead V1. An urgent echocardiographic examination showed a D-shaped left ventricle, right ventricle dilatation and dysfunction, McConnell's sign, suspected thrombus at the bifurcation of the pulmonary trunk, and dilated inferior vena cava, suggestive of pulmonary embolism (PE). The patient underwent MSCT pulmonary angiography, confirming the diagnosis of a massive pulmonary embolism with a thrombus present at the bifurcation of the pulmonary trunk. A CT of the endocranium revealed no pathological changes. Laboratory analyses showed significantly elevated levels of myocardial necrosis markers and D-dimer, along with elevated total prostate-specific antigen and moderate hypoxemia in gas analyses. The patient was treated with fibrinolytic therapy (alteplase) and low molecular weight heparin in accordance with the severe pulmonary embolism protocol. Subsequent echocardiography indicated improved right ventricular function with preserved movement of the right ventricle's free wall. A Bubble test confirmed the presence of a patent foramen ovale (PFO) with low risk of causing blood clots. Ultrasound of the lower extremity veins revealed normal results, and screening tests for thrombophilia were within normal limits. The patient declined a coronary angiography. The patient remained hemodynamically and rhythmically stable throughout the hospital stay, with satisfactory gas exchange.

CONCLUSION: The differential diagnosis for ST elevation is wide, including early repolarization syndrome, Brugada syndrome, LBBB, Prinzmetal’s angina, Takotsubo cardiomyopathy, left ventricular aneurysm, hypothermia, hyperkalemia, acute pericarditis, and pulmonary embolism. Pulmonary embolism can present with abnormal EKG and biomarkers that mimic an acute coronary syndrome, even showing ST elevation. It's important to be aware of this EKG pattern in PE, as it should prompt earlier bedside echocardiography, which is crucial for diagnosis. This should then lead to a computed tomography pulmonary angiogram

to confirm PE. Early diagnosis reduces the mortality rate associated with massive PE, which can be as high as 25–65% despite thrombolytic therapy.

O017 Role of cardiac magnetic resonance imaging differential diagnostic of acute coronary syndrome

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Acute coronary syndrome (ACS) is a life-threatening manifestation of atherosclerosis, which usually occurs in the setting of sudden plaque erosion or rupture with intracoronary thrombosis and partial to complete cessation of the downstream myocardial perfusion.
The diagnosis, management, and treatment of the various forms of ACS, which include persistent ST-segment elevation myocardial infarction (MI), non-ST-segment elevation MI, and unstable angina (UA), have rapidly been evolving in recent years with subsequently a significant decrease in early and late mortality.
The aim of this case is therefore to elucidate the role of cardiac magnetic resonance imaging (CMR) in this group of cardiac high-risk patients.
In our work, we present two patients with a similar clinical presentation of acute MI, without the presence of occlusive disease of the coronary arteries, with very similar laboratory parameters.The CMR helped us in making a correct diagnosis and determining the appropriate therapy.
CMR is a key in differentiating ACS from non-ACS related causes of chest pain. CMR is increasingly used to evaluate efficacy of novel treatment strategies in ACS patients.

O018 MINOCA from A to Z

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Myocardial infarction without coronary artery obstruction (MINOCA) is represented by 6% of all myocardial infarctions. The female gender predominates. Age is less than in myocardial infarction with significant stenosis, traditional risk factors or concomitant cardiovascular diseases are less present, except for hypertension. The prognosis was thought to be benign, but long-term studies show an increased risk of death and new cardiovascular events. A meta-analysis (44 studies) indicated that mortality was 2.0%, and that maximal hs-troponin was consistent with increased risk.
The definition of MINOCA must meet three criteria: 1. that the universal criteria for acute myocardial infarction are met, 2. that there is a non-obstructive lesion on the coronary artery (<50%), 3. that there is no clinically obvious, specific cause for the acute presentation of the patient.
It is important to determine the pathophysiological mechanism of MINOCAe in order to determine the optimal therapy for these patients. This is not easy in everyday clinical practice. The interpretation of previous studies is hindered by: different definitions of MINOCAe, studies on a small and selected number of patients, lack of suitable comparison populations, lack of randomized clinical trials on a population of suitable size. Properly designed prospective studies are needed to gain better insight into the pathophysiology of MINOCAe such as imaging studies, and into the efficacy of different treatment modalities e.g. therapeutic studies. When asked if we answered all the questions and reached Z in this entity, the answer is no.

O019 Possibilities of anticoagulant therapy in patients with thrombosis of artificial valves

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Dysfunction of the mechanical artificial valve (MV) can occur due to thrombosis, ingrowth of fibrous pannus, degeneration or endocarditis. The aforementioned conditions can occur simultaneously, so it is crucial to determine the etiology of the dysfunction of the mechanical heart valve and start appropriate treatment as soon as possible. Mechanical artificial heart valves, although longer-lasting than biological ones, carry with them a significantly higher risk of thrombosis and require lifelong use of anticoagulant therapy. The estimated annual rate of thrombosis of artificial heart valves is 0.1 to 5.7%, with higher rates in specific types of valves, those in the mitral or tricuspid position and if less than 3 months have passed since surgical implantation. The main cause of thrombosis is inadequate use of anticoagulant therapy.
The clinical presentation of MV thrombosis can vary from asymptomatic incidentally detected during echocardiographic examination to cardiogenic shock or thromboembolic events including massive stroke. Therefore, a high level of clinical suspicion and quick and timely diagnosis are necessary. According to the currently valid recommendations of the European and American cardiology associations, when a diagnosis of thrombosis is made MV therapy can include: optimization of anticoagulant therapy, application of thrombolytic therapy, surgical treatment and application of transcatheter interventions. Small non-obstructive thrombi of size 5-10 mm can be treated by optimizing anticoagulant therapy: using unfractionated and low molecular weight heparin with vitamin K antagonists until therapeutic INR values are reached. In the case of obstructive thrombosis and the presence of a larger thrombus with optimization of anticoagulant therapy, if the clinical condition of the patient allows it, the application of thrombolytic therapy, surgical treatment or transcatheter release of the artificial valve

leaflet is considered. According to the recommendations of the European Association of Cardiologists, urgent surgical intervention is recommended in critically ill patients with obstructive thrombosis in the absence of major comorbidities. American Heart Association recommendations favor low-dose slow infusion thrombolytic therapy or surgical treatment depending on clinical factors (including clinical and surgical experience).
Long-term prevention of thrombosis and thromboembolism after implantation of an artificial valve includes the use of effective antithrombotic therapy as well as modification of risk factors for thromboembolism.

O020 Is cardiac magnetic resonance necessary in acute coronary syndrome

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Acute coronary syndrome (ACS) represents a term of three types of coronary artery disease that describes a range of conditions related to sudden, reduced blood flow to the heart. These conditions include a myocardial infarction (MI) with ST elevation (STEMI), MI without ST elevation (NSTEMI), and unstable angina.
It often causes severe chest pain, discomfort, dyspnea, dizziness but some other symptoms can also be seen such as nausea, vomiting, heavy sweating, fainting, fatigue. ACS can occur suddenly when atherosclerotic plaque tears or splits open causing narrowing of the coronary vessel leading to ischemia. Sometimes ACS is caused by coronary artery embolism, coronary artery spasm and spontaneous coronary artery dissection.
Cardiac magnetic resonance (CMR) has shown to be an essential first step in the diagnosis of patients with ACS because it has a crucial role in differentiating the etiology of that syndrome. Imaging characteristics are used in clinical practice to discriminate etiologies of myocardial damage which may have similar clinical presentation.
Acute MI, Takotsubo syndrome, myocarditis and MI with non-obstructive coronary arteries all have some overlap regarding clinical symptoms as well as laboratory analysis. Early CMR within two weeks was shown to be an important diagnostic tool for assessing the etiology of patients presenting with troponin elevation and myocardial injury.
It has a unique capability to accurately assess both ischemic and non-ischemic etiologies for myocardial injury in order to ensure prompt treatment that is very important to ease symptoms and prevent complications.

O021 Nurse activities in patients with heart failure

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Heart failure is a frequent, progressive and deadly disease and one of the most significant problems today, both in the world and in our country. It is one of the leading causes of hospitalization in people over 75 years old. Heart failure is a serious condition that requires professional treatment and monitoring. Nursing activities include measuring vital signs, monitoring symptoms, implementing physician orders, monitoring medication intake, education for lifestyle changes, and providing emotional support to the patient. Nursing activities have an impact on the patient's quality of life, reducing the number of hospitalizations and improving the general state of health.
The aim of the work was to assess the impact of nursing activities, with special reference to patient education, on health outcomes in patients with heart failure.
Material and methods: The paper used a standard strategy of reviewing the available literature by searching electronic databases: Cohran, PubMed and Scindeks, according to key words: heart failure, nursing activities, health care, education
Results: Nursing activities in the education of patients with heart failure, which are related to lifestyle changes, correct taking of therapy and pointing out the importance of regular check-ups, improve the health outcomes of these patients.
Conclusion: Nursing activities in patients with heart failure have very positive effects, not only on patients, but also on society as a whole. They influence positive health outcomes for patients, reducing the number of readmissions to the hospital, improving quality of life and reducing mortality, as well as reducing the costs of health services.

O022 Recurrent MINOCA and the role of mutimodality imaging: case report

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The prevalence of myocardial infarction with non obstructive coronary arteries (MINOCA) is 6%, approximately half of them belongs to recurrent MINOCA.
CASE REPORT: A 53 year old woman arrived at emergency department due to first occurrence of typical chest pain that started at rest and lasted for approximately 30 minutes. Beside smoking for more than 25 years she was without significant previous medical history. Her physical examination at admission was unremarkable, her ECG showed sinus rhythm with incomplete right bundle branch block (RBBB) and horizontal ST depressions up to 1 mV in leads V4-V6. Her hs Troponin I was slightly elevated, so she was admitted to Cardiac Care Unit (CCU). On emergency ECHO there was discrete hypokinesia of left ventricular apex with preserved global systolic function. Patient underwent invasive coronary angiography which revealed no significant stenosis in coronary arteries, but with slow coronary flow (SCF). Further laboratory

analysis showed the rise and fall of markers of myocardial necrosis typical for acute myocardial lesion. She was discharged after five days with final diagnosis of NSTEMI without obstructive coronary artery disease (MINOCA). Three months later patient came again to our ED with atypical chest pain that occurred after several days of intensive emotional stress. Her physical findings were normal, with slight elevation of troponin level. On ECG there was sinus rhythm with incomplete RBBB, with discrete depression of ST segment in leads from V4 to V6 and with negative T wave in leads D3 and aVF. Considering previous medical history, patient was readmitted to our department. On repeated ECHO there were no visible regional wall motion abnormalities. Serial laboratory analysis showed the rise and fall of markers of myocardial necrosis with pattern typical for minimal ischemic lesion. In order to further characterize the type of myocardial lesion we performed cardiac magnetic resonance (CMR). On SSFP cine sequences on CMR there were no abnormal findings. On T2 weighted black blood sequences there was discrete subendocardial increase of signal intensity in the segments 10 and 11 of left ventricle corresponding to tissue edema. After 10 minutes of application of gadolinium there was clearly visible enhancement of signal intensity in subendocardial layer in segments 7, 9, 13, 14 and 15. Also on 4 and 2 chamber views there was subendocardial enhancement of signal in apical segment of septum and inferior wall along with medial and apical segment of anterior wall. Our definitive diagnosis was recurrent myocardial infarction with no obstructive coronary artery stenosis probably caused by microvascular disease.

CONCLUSION: This case represented a patient with recurrent MINOCA which prevalence is not negligible. Case is also pointing on essential role of multimodality imaging, especially on CMR when the final diagnosis after invasive coronary angiography is not clear.

O023 Changes in left ventricular function in SCAD

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Long-term follow-up of patients with spontaneous coronary artery dissection (SCAD) is of great importance. Repeated events occur more often in another blood vessel (29% in 10 years). Therefore, it is important to find clinical and imaging parameters as predictors for recurrent events but also death, stroke, need for revascularization (POBA, PCI), ventricular arrhythmias and sudden cardiac death (SCD). It is not clear whether the control of SCAD patients should be carried out by monitoring only with non-invasive imaging methods, or whether repeated invasive coronary angiography is also required. Myocardial injury/infarct size and left ventricular function after SCAD can be variable parameters and are sometimes of crucial importance and strong predictors of prognosis in the follow-up of these patients. Echocardiography and magnetic resonance imaging of the heart are imaging methods to monitor these changes. Younger women, but also middle-aged women with SCAD, tend to reduce infarct size and improve left ventricular systolic function during follow-up. This fact is consistent with the higher prevalence of TIMI 3 flow on angiography in SCAD-STEMI patients. However, if we have a more complex type of SCAD with total coronary artery occlusion, multisegmental and multivessel changes, this is not relevant.

O024 Optimization of life habits in prevention of coronary disease in women during menopause

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Vascular dysfunction, hypertension, and macrovascular obstructive coronary artery disease show a significant increase in women starting around their 50s. The more noticeable age-related cardiovascular risk seen in postmenopausal women is often resulting from withdrawal of protective estrogen. Direct effects of estrogen on the vascular system are supported by the presence of estrogen receptors on endothelial, smooth muscle and adventitial cells, and include increased nitric oxide release resulting in vasodilation and reduced oxidative stress. Cardiovascular diseases arises from a complex interaction of genetic, modifiable epigenetic, lifestyle and environmental factors. A serious approach is necessary, to prevent coronary disease in women, and it includes: optimization of body composition, consumption of a predominantly plant-based, Mediterranean-like diet, Intermittent fasting and time-restricted eating, and regular exercise, especially when initiated early in life. Habitual exercise appears to alleviate age-related decline in cardiac performance. Sedentary ageing is connected to degradation in cardiovascular function. Within the framework of changing life habits, it is recommended to avoid or limit alcohol consumption and abstinence from all forms of smoking. Insufficient sleep duration and disrupted sleep patterns are developing risk factors for the beginning and progression of hypertension, atrial fibrillation, ischemic heart diseases. Mindfulness-focused stress management and cognitive therapy, particularly when combined with slow breathing exercises and artistic/philosophical spiritual practices, can effectively diminish psychological and emotional stress, anxiety, and depression and promote human flourishing. Nurturing deep connections with family and friends through insight, empathetic communication, and forgiveness, marked by altruism and compassion, are paramount in enhancing the plasticity of well-being. Air pollution, even at low levels of fine particulates like PM2.5, is associated with higher risk of mortality from cardiovascular, respiratory, and cancer. Exposure to PM2.5 and ozone reduces heart rate variability, activates the immune system, and heightens oxidative stress, leading to endothelial dysfunction in both the vasculature and

brain. Physical activity in clean environments, especially parks and forested areas, offers both cardioprotective and psychological benefits.

O025 Myocardial infarction, myocarditis or Takotsubo cardiomyopathy - MRI to the rescue

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Until the past few years, all patients with a working diagnosis of myocardial infarction without coronary artery obstruction were treated as myocardial infarction or were not given therapeutic attention. Today, that has been changed with the cardiac magnetic resonance (CMR).

It is recommended that CMR shoud be performed in those patients within 14 days from the onset of symptoms. Signs of myocarditis, acute myocardial infarction or cardiomyopathy can be detected and proved by CMR. In a small number of cases, the diagnosis for these patients remains unclear.

In the detection of these conditions, PSIR (phase sensitive inversion recovery) sequences are used, that is sequence for the detection of late gadolinium enhancement (LGE), acquired 10 min. after contrast application.

Subendocardial or transmural LGE in coronary distribution is a typical pattern for infarction. In contrast to infarction, in myocarditis, LGE is subepicardial in non-coronary distribution and usually in the inferolateral wall, while in Takotsubo cardiomyopathy, LGE is absent.

In all three conditions in the acute phase, edema can be seen on the T2w sequence with fat suppression, which confirms the diagnosis; in the case of infarction, it is more often focal, and in the case of myocarditis and takotsubo cardiomyopathy, it is more often diffuse.

O026 The significance of lipidomics in cardiology emergencies

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Cardiology emergencies include acute coronary syndrome, myocardial infarction, sudden cardiac death, and heart failure. This presentation will focus on the importance of lipidomics in diagnosing and treating cardiovascular disorders in which ischemia-reperfusion injury (IR) plays a significant role. Lipidomics is a part of metabolomics that monitors the overall lipidome of individual tissues and organs (e.g., myocardium). It has been developing for the last two decades, primarily with the improvement of analytical methods such as Liquid Chromatography (LC)-Based Techniques (LC-MS), Shotgun Lipidomics (ESI/MS) and Nuclear Magnetic Resonance (NMR). The Framingham Risk Score (FRS) has already shown that LDL-c and HDL-c serum levels can predict 75% of cardiovascular risk. However, so far, attention in cardiology has been focused on macromolecules (e.g., LDL or LPa), but small molecules have proven to be more reliable biomarkers in the early stages of the development of adverse cardiovascular events. Thus, it has recently been shown that the Lipidomic Risk Score (LRS) is better in risk assessment for patients in the intermediate group, according to the FRS. Also, platelet aggregability is higher if the content of lysophosphatidylethanolamines in the platelet membrane is increased, and it decreases with an increase in acylcarnitine. It is essential to monitor the levels of certain saturated, monounsaturated, and polyunsaturated fatty acids (for example, palmitic or arachidonic acids) to assess the risk of ischemic events and IR injury. Finally, blocking the transport of long-chain fatty acids into the mitochondria and shifting the cell's energy metabolism from fatty acids to glucose can also prevent lipotoxicity, which is involved in cell death by various mechanisms.

O027 Cardiologist and pulmonologist on a joint mission

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Chronic obstructive pulmonary disease (COPD) is strongly associated with cardiovascular disease, in particular acute myocardial infarction (AMI). The prevalence of COPD amongst AMI populations ranges from 7% to 30%, which is possibly even an underestimation due to underdiagnoses of COPD in general.

Several pathological mechanisms underlie the relationship between COPD and ischaemic heart disease. COPD and ischaemic heart disease have common risk factors, of which smoking and increasing age are most important. In the acute phase of COPD, systemic inflammation develops with increased secretion of proinflammatory cytokines, which on the one hand accelerates atherosclerotic process, and on the other hand, systemic inflammation increases the number of platelets and their reactivity, which increases the risk of thrombotic events. Also, the level of coagulation factors increases in these patients. Hypoxia is associated with activation of the renin angiotensin aldosterone system which increases the risk of AMI.

Prognosis of patients with COPD after AMI is markedly worse compared to patients without COPD, both during short- and long-term follow-up. Particularly, all-cause mortality rates are significantly higher after correcting for known prognostic factors and smoking. In a large population-based registry in the UK, including 34019 COPD patients and 266142 patients without COPD, the 6-month mortality rates were significantly higher among COPD patients with non-ST-segment elevation myocardial infarction (non-STEMI) and ST-segment elevation myocardial infarction (STEMI) compared to non-COPD patients. These results were confirmed in subsequent studies

reporting double mortality rates for COPD patients compared to patients without COPD at long follow-up.

Following AMI, the rate of adverse events is higher in COPD patients when compared to patients without COPD, which might in part be due to a delay in recognition and subsequent revascularisation. Echocardiography has shown that COPD patients might be more vulnerable to ischaemia, leading to worse cardiac function when compared to patients without COPD. Advanced imaging techniques could play a pivotal role in risk stratification after AMI in patients with and without COPD.

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