PROGRAM

9. kongres Hrvatskog PULMOLOŠKOG DRUŠTVA HLZ-a

s međunarodnim sudjelovanjem

1. - 4. listopada 2026.

Hotel Excelsior, LOVRAN

ORGANIZATOR

Hrvatsko pulmološko društvo
Hrvatskoga liječničkog zbora

9. kongres Hrvatskog PULMOLOŠKOG DRUŠTVA HLZ-a

s međunarodnim sudjelovanjem

ORGANIZACIJSKI ODBOR

Neven Miculinić
Gordana Pavliša
Nevenka Piskač Živković
Marko Banić

ZNANSTVENI ODBOR

Sonja Badovinac
Fedja Džubur
Marko Jakopović
Mateja Janković Makek
Ozren Koluder
Marta Koršić
Đivo Ljubičić
Robert Marčun
Giovanni Batista Migliori
Suzana Mladinov
Ivan Pavić
Gordana Pavliša
Nevenka Piskač Živković
Aleš Rozman
Jasna Tekavec Trkanjec
Luka Vrbanić
Žarko Vrbica
Andreja Vukić Dugac

Poštovane kolegice i kolege,

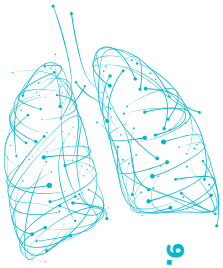
dobro došli na 9. kongres Hrvatskog pulmološkog društva HLZ-a s međunarodnim sudjelovanjem koji ove godine neće biti, kao prethodni kongresi, u Dubrovniku. Kao što slutite, glavni su razlozi financijske prirode te nemogućnost industrije da sponzorira veći broj sudionika na ekskluzivnim destinacijama (troškovi smještaja i prijevoza, ograničenja koja nameće udruga inovativne farmaceutike). Jasno je da bi Dubrovnik privukao brojne strane predavače, no siguran sam da će i u Lovranu izbor tema, predavanja i predavača itekako privući Vašu pažnju. **Opstruktivne bolesti pluća** su glavna tema praktički svake godine, ali sa svrhom. Pojavom novih bioloških terapija šire se mogućnosti liječenje kako astme, tako i KOPB-a. **Hitna stanja** u pulmologiji obogatit će naše znanje o indikacijama za prijem u JIL i osnovnim žurnim metodama liječenja. **Onkologija** se ponovno vraća u glavne teme naših skupova – svake godine novi onkološki lijekovi i nove, šire indikacije imunoterapije omogućuju dulje i kvalitetnije preživljavanje bolesnika s tumorima pluća. Uz glavnu temu koja se bavi **bolestima pleure**, sudionicima Kongresa se pruža mogućnost da usavrše svoje vještine u dvjema **radionicama**: „Pleuralne tehnike“ i „UZV pluća“. Čim se registrirate, požurite s prijavom – subota od 15.30 sati! Novost je da ćemo ove godine imati temu koja povezuje pedijatra-pulmologa i pulmologa: **Nasljedne bolesti**: Korak od dječje prema adultnoj pulmologiji. Saznajmo više o cističnoj fibrozi, nedostatku alfa-1-antitripsina i važnosti suradnje s kolegama pedijatrima. Ove se godine obilježava **15 godina od prvog endobronhalnog ultrazvuka u Hrvatskoj** pa je tome posvećen i Simpozij. Standardno su prisutni **Interaktivni prikazi slučajeva** iz kliničke prakse kao i **slobodne teme** na posterima. Najbolji će poster biti i nagrađen.

I pri kraju, ali ne i manje važno: **2. listopada 2026. (petak) u hotelu Excelsior/Lovran u 19.15 je Izborna skupština Društva** pa Vas molim da joj nazočite! Prijedlozima i savjetima poboljšajmo rad našeg društva.

prim.mr.sc. **Neven Miculinić**, dr. med.
predsjednik Hrvatskog pulmološkog društva HLZ-a

ČETVRTAK 1. listopada 2026.

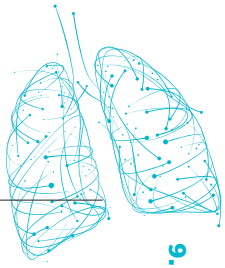
15.00	Registracija sudionika
16.10	I. Huljev Šipoš: When More Is Less: „High Positive Airway Pressure and Intermittent Hypoxia - Friend or Foe?"
16.30	K. Lalić: When even the viruses go ZEP! Tezepelumab joins the asthma fight
17.00-19.00	**RESPIRATORY EMERGENCIES: From Triage to the Intensive Care Unit** ***Chairs: F. Džubur, S. Mladinov***
17.00	A. Keranović: From Admission to the Ward: ED and Pulmonologist Collaboration in the Triage and Evaluation of Patients
17.20	G. Glodić: Hemoptysis in the Emergency Department
17.40	M. Pevec: Acute Interstitial Pneumonia vs. Severe Infection: Pitfalls in Differential Diagnosis
18.00	I. Bunjevac: Surgical Approach to Pulmonary Emergencies: When to Consult a Thoracic Surgeon?
18.20	F. Džubur: Admission of the Oncology Patient to the ICU: A Rational Approach and Supportive Care Options
18.40	DISCUSSION and Q&A
19.00	**Mini simpozij - Boehringer Ingelheim** A. Hećimović: Nerandomilast – novo poglavlje u liječenju plućne fibroze
19.30	M. Fležar: Preoperativna obrada pulmološkog bolesnika
20.00	**SVEČANO OTVORENJE**
20.30	**Večera**



PETAK, 02. listopada 2026.

8.30-10.50	**OBSTRUCTIVE LUNG DISEASES** ***Chairs: G. Pavliša, Ž. Vrbica***
8.30	S. Škrgat: The Asthma Patient Behind the Symptoms: Who is at Risk for Future Exacerbations and Airway Damage?
8.55	S. Hromiš: Beyond Control: Can Severe Asthma Enter Remission?
9.20	M. Vergles: Dual biologic therapy in severe asthma: justified strategy or overtreatment?
9.45	Ž. Vrbica: Plastic bronchitis
10.10	RASPRAVA
10.20	**Mini simpozij - AstraZeneca** D. Muršić, E. Vučak: Epitel u središtu nezadovoljenih potreba u KOPB-u: može li IL-33 promijeniti paradigmu liječenja? ***Moderatorica: A. Vukić Dugac***
10.50	**Pauza uz kavu**
11.20	G.B. Migliori: The future and Challenges of Shorter TB treatment regimens: findings from a global NIH study
11.50	TB Reloaded: Novi režimi, novi izazovi – PANEL RASPRAVA M. Janković Makek, M. Hajduk Ribić, D. Baričević, V. Trkeš

12.25	**Mini simpozij - Berlin Chemie Menarini** **Evolucija u liječenju astme i KOPB-a** Đ. Ljubičić: Kada kontrola astme zahtjeva korak više G. Pavliša: Evolucija liječenja KOPB-a
12.55	**Mini simpozij - Berlin Chemie Menarini** V. Popović: Kada je vrijeme za eskalaciju? Mjesto dualne bronhodilatacije u suvremenom liječenju KOPB-a
13.15	**SASTANAK Sekcije za opstruktivne bolesti pluća**
14.00	**Ručak**
15.10	T. Tokić Vuksan-Ćusa, R. Zrilić: Možemo li ranije prepoznati KOPB? Iskustva oportunističkog probira pušača kombinacijom LDCT-a i testova plućne funkcije
15.30	**Mini simpozij - Swixx Biopharma** S. Mladinov, M. Gomerčić Palčić: Je li smanjenje broja egzacerbacija dovoljno?
16.00-18.00	**PLEURAL DISEASES** ***Chairs: M. Ćurin, L. Vrbanić***
16.00	S. H. Skaarup: Thoracic ultrasound - present and future
16.30	S. Onyancha: Thoracoscopy and beyond
17.00	E. Vučak: Treating pnemothoraxophobia with current guidelines
17.25	I. Kovačević: Pleural infections - still wandering in the dark or seeing the light
17.50	L. Vrbanić: Pleurology?

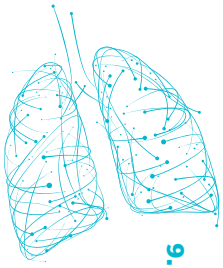


18.00	Pauza uz kavu
18.15	N. Sviličić: Anatomija dojma ili komunikacija između percepcije i stvarnosti
19.15	**IZBORNA SKUPŠTINA HRVATSKOG PULMOLOŠKOG DRUŠTVA**
20.30	Večera

SUBOTA, 03. listopada 2026. - DVORANA A

8.30-10.20	**NASLJEDNE BOLESTI: Korak od pedijatrijske prema adultnoj pulmologiji** ***Voditelji: A. Vukić Dugac, I. Pavić***
8.30	I. Mihatov Štefanović: Bronhopulmonalna displazija – dugoročne posljedice ranog početka
8.50	A. Vukić Dugac: Cistična fibroza – od simptomatskog liječenja do ciljane terapije
9.10	Lj. Odak: Genetska dijagnostika – kada posumnjati, koga testirati?
9.30	I. Pavić: Primarna cilijarna diskinezija – kako ne propustiti dijagnozu?
09.50	**Mini simpozij - Medison Pharma** A. Vukić Dugac, I. Bambir: Što kada dijete odraste – prijelaz iz pedijatrijske u odraslu skrb
10.20	Pauza za kavu

10.45-12.45	**NOVOSTI U ONKOLOGIJI** ***Voditelji: M. Jakopović, S. Badovinac, V. Popović***
10.45	S. Badovinac: Sveobuhvatni algoritam dijagnostike raka pluća: Između kliničke realnosti i vremenskog pritiska
11.10	M. Jakopović: Perioperativni zaokret kod NSCLC-a: Pozicioniranje kliničke prakse u RH
11.35	S .Mladinov: Precizna onkologija danas i sutra: Trenutačni dometi i evolucija ciljane terapije
12.00	V Popović: Redefiniranje paradigmi kod SCLC-a: Klinički proboji i nova era u liječenju
12.25	RASPRAVA
12.40	**Mini simpozij - MSD** K. Krpina: Od dijagnoze do terapije: NSCLC iz prakse pulmologa
13.15	**Ručak**
15.00-16.30	**PRIKAZI SLUČAJEVA IZ KLINIČKE PRAKSE** ***Voditelji: J. Tekavec Trkanjec, O. Koluder***
15.00	M. Kneussl: Tuberculosis and Pregnancy
15.25	I. Folnožić, M. Gomerčić Palčić: Kada u diferencijalnoj dijagnozi nedostaje točan odgovor
15.55	J. Tekavec Trkanjec: Silikoza - tihi okidač nekih drugih bolesti
16.30	**Pauza za kavu**
17.00-18.30	**SIMPOZIJ: 15 godina EBUS-a u Hrvatskoj** ***Voditelji: N. Piskač Živković, S. Badovinac, Đ. Ljubičić***
17.00	N. Piskač Živković: 15 godina EBUS-a u Hrvatskoj: Iskustva, postignuća i pogled u budućnost.



17.10	S. Badovinac: 15 godina EBUS-a u dijagnostici medijastinuma u Hrvatskoj: Korak naprijed kroz nove ERS/ESGE/ESTS kliničke smjernice i 9.TNM klasifikaciju
17.35	A. Rozman: EBUS - Guided Cryobiopsy: Our Experience and Recent Advances
18.00	A. Szlubowski: Clinical Application of Thin Convex Probe EBUS in Pulmonary Diagnostics
18.25	RASPRAVA

SUBOTA, 03. listopada 2026. - DVORANA B

9:00-10:30 pauza 10:45-13:15 pauza 15:00-16:30 pauza

***RADIONICA PLEURALNIH VJEŠTINA – „Hands-on“**

***RADIONICA ULTRAZVUKA PLUĆA – „Hands-on“**

Voditelji: L. Vrbanić, R. Marčun

***Za radionice je potrebno prijaviti se prilikom registracije.**

21.00 **Večera**

NEDJELJA, 04. listopada 2026.

8.30-10.30 **SLOBODNE TEME – posteri**

Voditelji: I. Barković, Đ. Ljubičić, N. Miculinić

11.00 Odjava i odlazak iz hotela

OPĆE INFORMACIJE

MJESTO ODRŽAVANJA SKUPA

Excelsior**** / Šetalište maršala Tita 15, 51415, Lovran

KOTIZACIJA

- Rana kotizacija za liječnike do 28.8.2026. ..... **500 €**
- Kasna kotizacija za liječnike od 29.8.2026. ..... **700 €**
- Kotizacija za sponzore ..... **600 €**
- Kotizacija za umirovljenike i specijalizante ..... **450 €**

Kotizacija za sve kategorije uključuje: sudjelovanje u znanstvenom programu, pristup izložbenom prostoru, materijale skupa, prisustvovanje otvorenju skupa s koktelom dobrodošlice, zajedničkoj večeri 3.10., ručak 2. i 3. 10.206., prisustvovanje satelitskim simpozijima, kavu u stankama.

HOTELSKI SMJEŠTAJ

Za sudionike Skupa osiguran je smještaj po posebnim cijenama u hotelu Excelsior

- hotel Excelsior - cijena paketa HB 3 noćenja u jednokrevetnoj sobi **465 €** po osobi
- hotel Excelsior - cijena paketa HB 3 noćenja u dvokrevetnoj sobi **323 €** po osobi

Cijena smještaja uključuje: doručak, večere 1. i 2.10.2026., te boravišnu pristojbu.

9. kongres Hrvatskog PULMOLOŠKOG DRUŠTVA HLZ-a

SAŽETCI

BEYOND CONTROL: CAN SEVERE ASTHMA ENTER REMISSION?

Hromiš S.

Institute for Pulmonary Diseases of Vojvodina, Sremska Kamenica, Serbia,

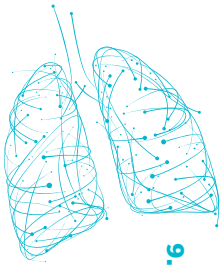
Faculty of Medicine, University of Novi Sad, Novi Sad, Serbia

Biologic therapies have significantly improved treatment outcomes in severe asthma and, in a subset of patients, have resulted in an almost complete resolution of all disease manifestations.

These outcomes have led to a new, increasingly accepted treatment goal, shifting from disease control to clinical remission. This concept was first formalised by Menzies-Gow et al. using a Delphi consensus based on experience from other chronic inflammatory diseases, and comprises sustained absence of significant symptoms, exacerbations and the need for systemic corticosteroids, with optimisation or stabilisation of lung function and patient and physician agreement, for at least one year.

Since then, the definitions used in real-world studies and expert consensus statements vary in required criteria, treatment status and duration. These differences reflect the still insufficiently understood mechanisms leading to remission and the need to define a state that predicts favourable long-term outcomes rather than only short-term improvement.

Better asthma control, higher biomarker levels (eosinophils and FeNO), absence of maintenance oral corticosteroids and the presence of rhinonasal comorbidities are known to be associated with a better response to biologics. However, individual prediction of treatment response is still not possible, and currently available inflammatory biomarkers have limited predictive value.



Severe asthma can enter clinical remission, which moves the treatment goal beyond disease control, but remission is currently achieved only in a subset of patients. Establishing clinical remission as a treatment goal requires harmonised criteria, so that rates are comparable and have prognostic value. This lecture will discuss criteria, predictors and limitations of clinical remission.

"WHEN MORE IS LESS" HIGH POSITIVE AIRWAY PRESSURE AND INTERMITTENT HYPOXIA: FRIEND OR FOE?

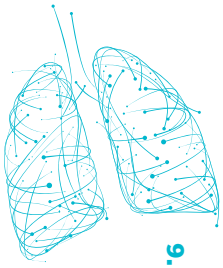
Huljev Šipoš I.

KB Dubrava, Zagreb, Croatia

OBJECTIVE: To examine whether complete suppression of respiratory events and intermittent hypoxia should always be the therapeutic goal in sleep-disordered breathing, and to discuss the balance between the benefits and adverse effects of high positive airway pressures.

METHODS: A narrative synthesis of the physiological and clinical effects of high positive airway pressure and intermittent hypoxia was combined with our clinical approach to patients requiring high therapeutic pressures. Particular attention was given to high-intensity non-invasive ventilation in chronic hypercapnic respiratory failure, hypoxic burden, dose-dependent effects of intermittent hypoxia, and strategies for reducing required expiratory positive airway pressure.

RESULTS: High-intensity non-invasive ventilation using higher inspiratory pressures can improve alveolar ventilation and reduce arterial carbon dioxide tension in selected patients with chronic hypercapnic respiratory failure. In sleep-disordered breathing, however, escalating positive airway pressure may increase mask leak, aerophagia, discomfort and treatment-emergent central sleep apnea and may impair adherence. Conversely, untreated intermittent hypoxemia contributes to oxidative stress, sympathetic activation, endothelial dysfunction and cardiovascular and metabolic complications. Experimental and clinical observations suggest that the biological effect of intermittent hypoxia is dose-dependent: controlled low hypoxic burden may activate adaptive pathways, whereas severe repetitive hypoxia is harmful. In patients requiring high expiratory positive airway pressure, identification of the predominant site of upper-airway collapse may enable adjunctive treatment. In selected patients with retrolingual obstruction, individualized 3D-printed intraoral appliances used with positive airway



pressure therapy have, in our experience, reduced required expiratory positive airway pressure by approximately 2–6 cmH_2O, with improved tolerance.

CONCLUSIONS: Neither positive airway pressure nor intermittent hypoxia is intrinsically a friend or foe. Their effects depend on dose, duration and clinical context. Treatment should therefore aim not merely at normalization of respiratory indices, but at an individualized therapeutic optimum that achieves effective disease control with the lowest treatment burden and best long-term adherence.

KEYWORDS: Sleep Apnea; Positive-Pressure Respiration; Intermittent Hypoxia; Noninvasive Ventilation; Treatment Adherence and Compliance

HYPOXIC BURDEN AND CORONARY COLLATERALIZATION IN PATIENTS WITH NON-ST-ELEVATION MYOCARDIAL INFARCTION

Šipoš K.[1], Huljev Šipoš I.[2]

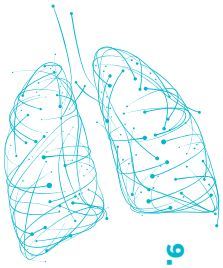
[1] *Magdalena Clinic for Cardiovascular Diseases, Krapinske Toplice, Croatia, Department of Cardiology*

[2] *University Hospital Dubrava, Zagreb, Croatia, Department of Pulmonology*

BACKGROUND: Intermittent hypoxia associated with sleep-disordered breathing has traditionally been considered an important contributor to cardiovascular injury. However, repeated hypoxic exposure may also activate adaptive responses, including hypoxia-inducible pathways involved in angiogenesis and vascular remodeling. Hypoxic burden provides a more comprehensive measure of nocturnal hypoxic exposure than the apnea-hypopnea index alone. We aimed to investigate the relationship between hypoxic burden and the degree of coronary collateralization in patients with non-ST-elevation myocardial infarction (NSTEMI) undergoing elective coronary angiography.

METHODS: In this ongoing prospective observational study, patients with NSTEMI scheduled for elective coronary angiography undergo overnight respiratory polygraphy to characterize sleep-disordered breathing and quantify hypoxic burden. Coronary collateral circulation is assessed angiographically using the Rentrop and Werner classifications. The relationship between hypoxic burden and the extent of coronary collateralization is subsequently evaluated. Twenty patients have been enrolled to date. Formal statistical analysis is ongoing.

PRELIMINARY RESULTS: Preliminary observations suggest that higher hypoxic burden is associated with a greater degree of coronary collateralization. Patients with more pronounced nocturnal hypoxic exposure appear to demonstrate more developed collateral coronary circulation according to angiographic assessment. Although these observations require confirmation by formal statistical analysis, they



raise the possibility that repetitive nocturnal hypoxia may, under certain conditions, be associated with activation of adaptive angiogenic mechanisms and development of coronary collateral vessels.

CONCLUSIONS: Our preliminary observations suggest a potential positive association between nocturnal hypoxic burden and the degree of coronary collateralization in patients with NSTEMI. These findings support the hypothesis that the cardiovascular effects of intermittent hypoxia may depend on its intensity, duration, and pattern of exposure and may include both detrimental and adaptive responses. Whether higher hypoxic burden independently predicts more extensive coronary collateral circulation remains to be determined after completion of recruitment and statistical analysis.

KEYWORDS: Non-ST-Elevation Myocardial Infarction; Hypoxic Burden; Intermittent Hypoxia; Coronary Collateral Circulation; Sleep-Disordered Breathing; Angiogenesis

IDIOPATHIC INTERMITTENT HYPOXEMIA

Vrbanić L.[1], Goršeta B.[1], Vučak E.[1], Gomerčić Palčić M.[1,2], Folnožić I.[1], Brkić K.[1]

[1] *Department for Pulmonology, Divison for Internal Medicine, University Hospital Center "Sestre Milosrdnice", Zagreb, Croatia*

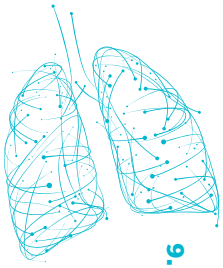
[2] *School of Medicine, University of Zagreb, Croatia*

BACKGROUND: Hypoxemia of unknown etiology is a broad clinical entity whose differential diagnosis includes airway diseases, pulmonary parenchymal disorders, pulmonary circulation abnormalities, sleep-related breathing disorders, chest wall and respiratory muscle diseases, as well as cardiovascular diseases.

AIMS: A multidisciplinary approach is essential in the diagnostic workup to enable identification of the underlying cause of hypoxemia.

METHODS: We report a 55-year-old woman evaluated for intermittent hypoxemia over three years. She initially presented with leg weakness, fever, cough and dyspnea. She subsequently experienced recurrent desaturation episodes (down to 85%) of variable frequency and duration (up to 24 hours). Systemic and inhaled corticosteroids led to transient symptomatic improvement.

RESULTS: Pulmonary funcion tests were normal, except slight intermittent reduction of diffusing capacity. Methacholine challenge was negative. Cardiopulmonary exercise testing revealed no desaturation or spirometric abnormalities, with cardiovascular limitation. Chest X-rays were unremarkable. Serial CT showed stable mild bilateral posterobasal ground-glass opacities, expiratory air trapping and small non-progressive nodules, without interstitial lung disease or pulmonary vascular pathology. Lung scintigraphy excluded pulmonary embolism. Bronchoscopy was unremarkable. Polysomnography excluded sleep disorders. Transthoracic and transesophageal echocardiography, coronary angiography and myocardial scintigraphy excluded pulmonary hypertension, intracardiac shunting and significant coronary



disease. Immunological findings were inconclusive. Assesment of the autonomic nervous and muscular system found no abnormalities.

CONCLUSION: Despite extensive pulmonary, cardiological, immunological and neurological evaluation, no definitive cause of hypoxemia was identified. Non-classical mechanisms of unresolved intermittent hypoxemia should be considered and further investigated.

RESIDUAL CHEYNE–STOKES RESPIRATION DESPITE EFFECTIVE CONTROL OF OBSTRUCTIVE EVENTS WITH CPAP IN HFrEF

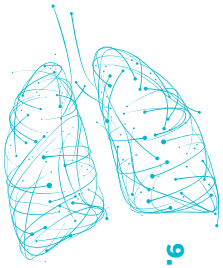
Huljev Šipoš I.[1], Šipoš K.[2]

[1] *Department of Pulmonology, University Hospital Dubrava, Zagreb, Croatia*

[2] *Department of Cardiology, Magdalena Clinic for Cardiovascular Diseases, Krapinske Toplice, Croatia*

BACKGROUND: Sleep-disordered breathing is common in patients with heart failure with reduced ejection fraction (HFrEF) and frequently comprises a combination of obstructive sleep apnea and periodic breathing, including Cheyne–Stokes respiration (CSR). While obstructive respiratory events can be effectively treated with continuous positive airway pressure (CPAP), the clinical significance of persistent CSR after adequate CPAP titration remains incompletely understood.

CASE SERIES: We present a series of nine patients with chronic HFrEF and mixed sleep-disordered breathing. All patients underwent polygraphy followed by CPAP titration using respiratory polygraphy, aiming to eliminate obstructive respiratory events. Following titration, patients were treated with fixed CPAP pressures of 6–10 cmH_2O. Obstructive apneas and hypopneas were successfully eliminated in all patients, with no residual obstructive events. Supplemental oxygen at 1–2 L/min achieved stable nocturnal oxygenation without significant desaturations. None of the patients reported significant daytime sleepiness after treatment initiation. Despite effective control of obstructive events and nocturnal hypoxemia, Cheyne–Stokes respiration persisted throughout the night. This finding suggests that elimination of upper-airway obstruction and intermittent hypoxia does not necessarily normalize the underlying ventilatory pattern. Persistent CSR may reflect ongoing instability of ventilatory control, prolonged circulation time, and oscillations in arterial carbon dioxide tension, with sustained sympathetic activation.



CONCLUSION: CPAP combined with supplemental oxygen can effectively eliminate obstructive respiratory events and nocturnal hypoxemia in patients with HFrEF, thereby reducing the burden associated with intermittent hypoxia. However, persistent Cheyne–Stokes respiration may indicate residual pathophysiological instability and potentially ongoing cardiovascular risk despite successful treatment of the obstructive component. These patients may therefore require further individualized assessment and optimization of treatment.

KEYWORDS: Cheyne–Stokes Respiration; Heart Failure with Reduced Ejection Fraction; Sleep-Disordered Breathing; Continuous Positive Airway Pressure; Central Sleep Apnea

PULMONARY CRYPTOCOCCOSIS PRESENTING AS AN INCIDENTAL PULMONARY NODULE: A CASE REPORT

Jelinčić P.[1], Kovačić J.[1] ,Radman L.[2], Pevec M.[2], Džubur F.[1, 2], Glodić G.[1, 2]

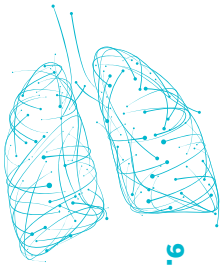
[1] *Sveučilište u Zagrebu, Zagreb, Croatia – Medicinski fakultet*

[2] *KBC Zagreb, Zagreb, Croatia – Klinika za plućne bolesti Jordanovac*

Pulmonary cryptococcosis is an opportunistic fungal infection that occurs in immunocompromised patients, including solid-organ transplant recipients. Its clinical and radiological presentation can vary widely, making diagnosis challenging, especially in asymptomatic patients. We present a case of a lung transplant patient with pulmonary cryptococcosis presenting as an incidental pulmonary nodule.

A 66-year-old male patient underwent bilateral lung transplantation for end stage COPD 8 years ago. He remained stable during post-transplant follow up. In June 2026, during a routine follow-up examination, chest CT revealed newly developed incidental pulmonary nodules; the larger measuring 28 mm in the right lower lobe (RLL). Bronchoscopy with radial endobronchial ultrasound identified an eccentric homogeneous pathological lesion measuring approximately 15 mm in the RLL. Bronchial aspirate and lavage cytology demonstrated numerous neutrophils and macrophages containing round-to-oval intracellular structures, raising suspicion of fungal infection, particularly histoplasmosis or cryptococcosis. PET/CT demonstrated bilateral pathological FDG uptake within the pulmonary nodules, with a maximum uptake of 13.4, while no pathological uptake was identified elsewhere in the body. After final microbiological analysis confirmed cryptococcosis, the patient was hospitalized and treated with liposomal amphotericin B for two weeks. Following completion of induction therapy, consolidation therapy with fluconazole 400 mg daily was initiated.

This case highlights the importance of considering opportunistic fungal infections in immunocompromised patients such as lung-trans-



plant recipients since they may present with minimal or nonspecific clinical manifestations, such as incidental pulmonary nodules. Despite the indolent clinical presentation, aggressive therapy is warranted to prevent systemic disease.

CENTRAL SLEEP APNEA AFTER ACUTE STEMI: A TRANSIENT POST-REPERFUSION PHENOMENON?

Šipoš K.[1], Huljev Šipoš I.[2]

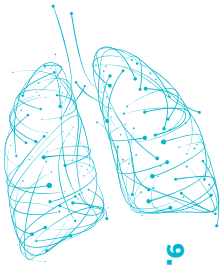
[1] *Magdalena Clinic for Cardiovascular Diseases, Krapinske Toplice, Croatia, Department of Cardiology*

[2] *University Hospital Dubrava, Zagreb, Croatia, Department of Pulmonology*

BACKGROUND: Sleep-disordered breathing is common in patients with acute myocardial infarction, but its phenotype and evolution following successful reperfusion remain incompletely understood. Central respiratory events and Cheyne–Stokes respiration (CSR) may occur during the acute phase of myocardial injury and potentially reflect transient alterations in cardiac function, autonomic regulation, and ventilatory control. We aimed to characterize sleep-disordered breathing immediately after reperfusion in patients with ST-segment elevation myocardial infarction (STEMI) and to assess its evolution during subsequent recovery.

METHODS: In this ongoing prospective observational study, consecutive patients with acute STEMI successfully treated with reperfusion undergo overnight respiratory polygraphy during the early post-reperfusion period. Follow-up respiratory polygraphy is performed approximately two weeks after the index event and again at six months to evaluate changes in the frequency and phenotype of sleep-disordered breathing. Approximately 15 patients have been enrolled to date. Formal statistical analysis is ongoing.

PRELIMINARY RESULTS: Preliminary observations indicate a high frequency of central respiratory events and periodic breathing consistent with Cheyne–Stokes respiration during the immediate post-reperfusion period. At the two-week follow-up, a reduction in central respiratory events and CSR is already apparent, with further improvement observed at six months. These preliminary findings suggest a progressive evolution of the respiratory pattern following acute STEMI, with



central sleep-disordered breathing being most prominent during the acute phase and tending to regress during cardiovascular recovery.

CONCLUSIONS: Central respiratory events and Cheyne–Stokes respiration appear to be frequent immediately after reperfused STEMI but may represent a dynamic and potentially transient phenomenon. Their early improvement at two weeks and further regression at six months highlight the importance of timing when assessing sleep-disordered breathing after acute myocardial infarction. Sleep studies performed exclusively during the acute phase may therefore overestimate the long-term burden of central sleep-disordered breathing. Longitudinal reassessment may be important for accurate phenotyping and for avoiding premature decisions regarding long-term treatment. Final conclusions will require completion of recruitment and statistical analysis.

KEYWORDS: ST-Segment Elevation Myocardial Infarction; Cheyne–Stokes Respiration; Central Sleep Apnea; Sleep-Disordered Breathing; Reperfusion

IZRAŽENA PERIFERNA EOZINOFILIJA KAO NUSPOJAVA ANTIMIKROBNE TERAPIJE

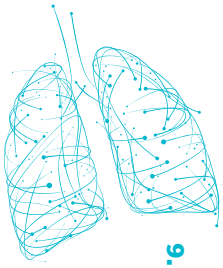
Beljo Kosanović G.[1], Ignjatović K.[1], Banić M.[2], Moćan A.[2], Lovrić T.[2], Janković Makek M.[1,2]

[1] Medicinski fakultet, Sveučilište u Zagrebu

2 Klinika za plućne bolesti Jordanovac, Klinički bolnički centar Zagreb

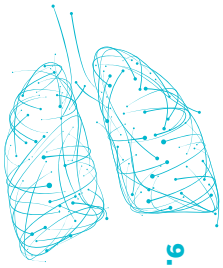
POZADINA: Eozinofilija uzrokovana lijekovima prepoznata je nuspojava antimikrobne terapije, iako je izražena periferna eozinofilija rijetka. Prikazujemo slučaj teške periferne eozinofilije tijekom terapije levofloksacinom i vankomicinom.

PRIKAZ SLUČAJA: Bolesnica stara 35 godina, liječena zbog depresije, hospitalizirana je zbog respiratorne insuficijencije (kod prijema potreba za terapijom visokim protocima kisika; FiO2 60%) i obostrane pneumonije. Na odjelu je nastavljena započeta terapija levofloksacinom, a CT angiografijom je isključena akutna embolija i potvrđena obostrana pleuropneumonija i mali perikardijalni izljev. Drugi dan liječenja ordiniran je vankomicin zbog nalaza gram-pozitivnih koka u hemokulturi, no terapija je prekinuta već nakon prve doze zbog pojave osipa na prsima i leđima. U laboratorijskim je kontrolama zamjećena značajna periferna eozinofilija (porast s inicijalnih 1.9% na čak 28%) te je razmatrana mogućnost eozinofilne pneumonije. Međutim, kako je uz antibiotsku terapiju bolesnica imala kliničko i radiološko poboljšanje uz deeskalaciju oksigenoterapije, postavljena je sumnja na eozinofiliju uzrokovanu antimikrobnom terapijom i eventualni DRESS (drug reaction with eosinophilia and systemic symptoms) sindrom. Učinjena je sternalna punkcija (normocelularna koštana srž) i dodatna obrada kojom je isključena zahvaćenost ostalih organa (na prvom mjestu srca i jetre). Zahvaljujući brzom kliničkom oporavku i gotovo potpunoj radiološkoj regresiji, antibiotska terapija je potpuno ukinuta nakon 8 dana liječenja, a u laboratorijskim nalazima se kroz iduća dva mjeseca postupno pratila normalizacija vrijednosti eozinofila.



ZAKLJUČAK: Izražena eozinofilija nakon kratkotrajne izloženosti antibioticima iznimno je rijetka. U prikazanom slučaju eozinofilija je dosegnula 28% tijekom terapije levofloksacinom i nakon samo jedne doze vankomicina te se postupno i spontano normalizirala nakon prekida antimikrobne terapije.

notes / bilješke

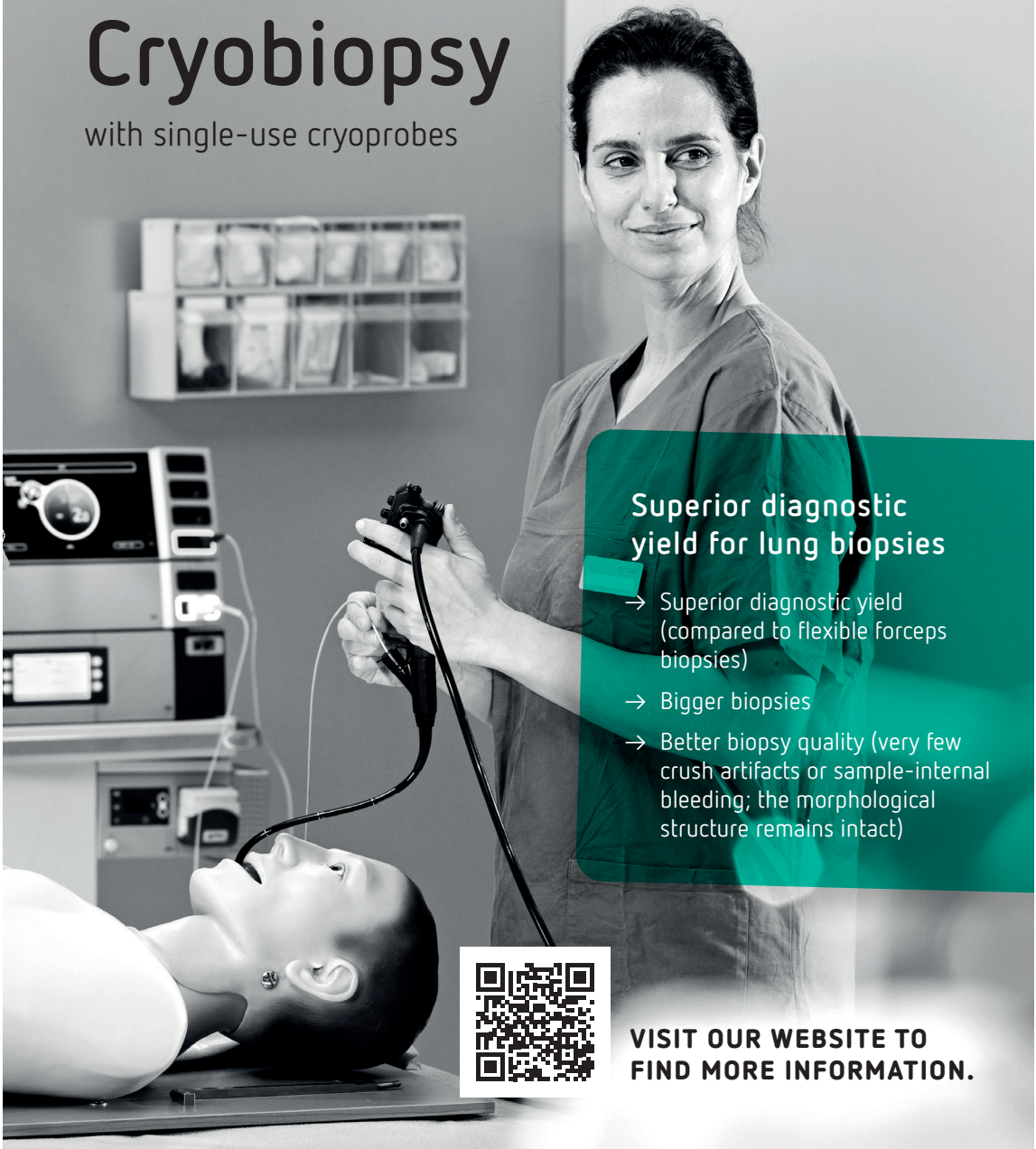


notes / bilješke

erbe
power your performance.

Cryobiopsy

with single-use cryoprobes

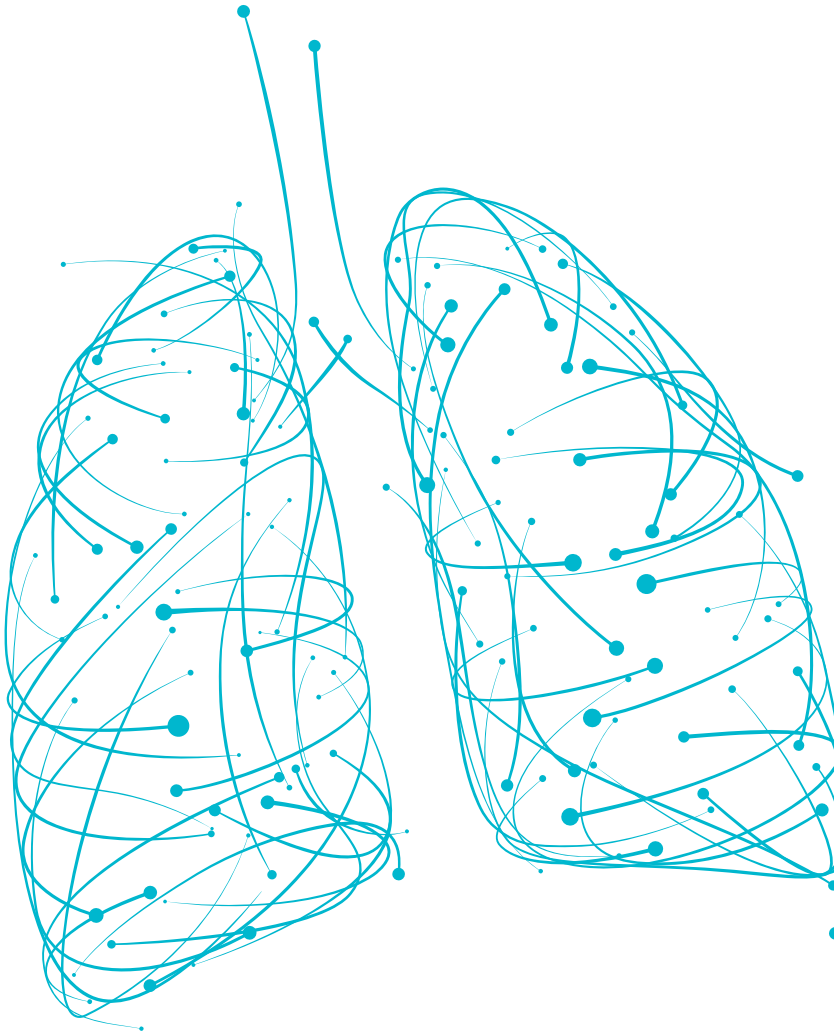


Superior diagnostic yield for lung biopsies

- → Superior diagnostic yield (compared to flexible forceps biopsies)
- → Bigger biopsies
- → Better biopsy quality (very few crush artifacts or sample-internal bleeding; the morphological structure remains intact)

VISIT OUR WEBSITE TO FIND MORE INFORMATION.

MEL-Medikal d.o.o Vinka Međerala 4a HR-42000 Varaždin 042-203 953 info@mel-medikal.hr



TEHNIČKI OGANIZATOR
conTres projekti d.o.o.
www.contres.hr
E-mail: info@contres.hr