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RECORD

Содржина

Воведно

Почитувани колеги,

Имам чест во овој број на електронското списание за кардиоваскуларна медицина да го најавам Професорот Филипо Креа, како Уредник на European Heart Journal, кое е официјално списание на Европското Здружение по Кардиологија. Креа, кој последниот период освен уредување на овој престижен журнал има за цел едуцирање на младите кардиолози во насока на пишување и објавување на научни трудови, одговори на мојата покана и го испрати тематскиот ревијален труд со наслов Како да се напише труд.
Во вториот дел местото го најде базичен труд во областа на кардиоваскуларната медицина од колега кардиолог од странство, кој предава патолошка физиологија, а кој ја помина позитивната рецензија. Со овој труд практично списанието се отвара за испраќање и на трудови од базичната медицина.
Ми останува да Ви посакам Вам и на Вашите семејства среќни претстојни Велигденски и Рамадански празници.

Проф. д-р Маријан Бошевски, FESC

How to write a scientific paper

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Извадок

Овој ревијален натпис има за цел да понуди практични совети за концептот на истражувањето, стилот на пишување и вообичаените стапици во процесот на објавување. Наменет е да ги охрабри младите кардиолози во придонесот во научната литература.

Introduction

Writing a scientific paper is essential for cardiologists aiming to contribute to the body of knowledge within their field. Whether publishing original research, case studies or review articles, effective writing is critical in communicating complex ideas clearly and persuasively. The process can be daunting, but by following a structured approach, researchers can effectively navigate the stages of manuscript preparation, achieving high-ranking journals with a wide resonance for their research. This review article aims to offer practical advice on research conceptualization, writing style, and common pitfalls in the publication process, empowering young cardiologists to contribute meaningfully to the scientific literature.

Why publish a research paper

Publishing a scientific paper is essential for researchers as it allows them to share their findings, contribute to the advancement of knowledge, and engage with the scientific community. For cardiologists, publishing is also a way to provide insights into clinical practice and ultimately improve patient care. Moreover, publication increases visibility and fosters collaboration with peers and institutions, expanding the impact of the research.

One key metric that evaluates a researcher's performance is the *h-index*. The h-index is a quantitative measure that reflects both the number of publications a researcher has and the number of citations these papers receive. A high h-index suggests that the researcher has produced a body of work that has been widely recognized and cited by other researchers in the field. For cardiologists, achieving a higher h-index can enhance their professional reputation, increase opportunities for research funding, and improve their chances of securing academic positions.

How to write a research paper

Formulating a research question

The first step in writing a scientific paper is choosing a relevant and novel topic. The scope of potential subjects is vast, ranging from basic science to clinical studies. The selected topic should align with your expertise, and be achievable within practical and ethical constraints, while also filling a gap in current knowledge. A research topic should not only be novel but can also address existing controversies in literature. Once the topic is decided, crafting a precise research question will guide the design and structure of your paper.

Study design selection and methodology

The methodology section is one of the most critical parts of writing a scientific paper. It includes the choice of study design, which will depend on the research question and the available resources. Study designs in cardiovascular research include randomized controlled trials (RCTs), cohort studies, case-control studies, and systematic reviews. RCTs are considered the gold standard for evaluating clinical interventions due to their ability to minimize bias and establish causality, whereas observational studies are valuable for examining associations in real-world settings, particularly when randomization is impractical. Determining an appropriate sample size through statistical power analysis is essential to detect clinically meaningful effects while balancing resource use. Equally important is the transparent and detailed description of data collection and analysis procedures.

Structure and content of a scientific paper

A scientific paper follows a structured format, usually organized into the following sections: Title, Abstract, Introduction, Methods, Results, Discussion, and Conclusion.

Title

The title of a research paper should be clear, concise, and accurately reflect the study's focus while capturing readers' attention and interest.

Abstract

The abstract is the first part of your paper that readers will see. As such, it must offer a succinct yet comprehensive summary of the study, allowing readers to quickly grasp its purpose, methods, key findings, and clinical implications.

Introduction

The introduction sets the foundation by providing background and context for the research. It gives an overview of the existing literature, summarizes relevant studies, and highlights gaps or controversies that justify the study. It concludes with a clear statement of the study's objective, outlining the specific aim and direction of the research.

Methods

The methods section details how the research was conducted. This section must clearly describe the study design, participant selection, and data collection. It should also address the statistical analysis methodology, discuss the chosen tests for evaluating relationships, comparing groups, or testing hypotheses, and define the criteria for statistical significance. This transparency allows readers to assess the reliability and robustness of the study’s results.

Results

The results section presents the main findings of the study, which should be explained with clarity in a logical, coherent order. To facilitate the understanding of complex data, the use of visual aids such as tables and figures is highly recommended. In addition to presenting raw data, it is essential to report the statistical measures that underpin the findings, including p-values, confidence intervals, and effect sizes. These measures enable readers to evaluate the reliability and robustness of the results.

Discussion

The discussion section interprets and contextualizes the study’s findings within existing literature, addresses the strengths and limitations of the study, and highlights the potential clinical implications to improve patients’ outcomes.

Conclusion

The conclusion briefly summarizes the study’s main findings, highlighting their clinical relevance and potential impact on cardiovascular care. It also suggests directions for future research, particularly where new questions or uncertainties have emerged.

Choosing the right journal

Selecting an appropriate journal is crucial for the successful publication of your manuscript. Researchers should target journals that align with the scope of their research and reach the intended audience that can benefit from or build upon their findings. Before submitting a manuscript, the author may consider if there are upcoming conferences that make the paper timely. Another key factor to consider is the journal's impact factor, which can indicate the reach and influence of the publication within the scientific community. Higher-impact journals generally have wider circulation and greater citation potential, thus enhancing the visibility and recognition of research work. Finally, authors should always review the submission guidelines for specific requirements regarding manuscript formatting, word limits, and referencing style of the journal to ensure compliance and avoid delays in the submission process.

Revising and Refining the Paper

Writing the first draft of a scientific paper marks the beginning of a lengthy process. Once the draft is complete, it’s essential to engage in revisions to refine the manuscript and enhance its clarity, precision, and overall impact. Revising a paper is a critical step that ensures the research is communicated effectively to the target audience, with attention to detail that improves readability and scientific rigor. Ensuring clarity is crucial during manuscript revision. To achieve this, authors should employ direct, concise language that remains scientifically accurate but avoids excessive jargon. Peer review constitutes another vital component, as feedback from colleagues, mentors, or domain experts often improves the structure, argumentation, and overall readability of the research.

Conclusions

In conclusion, writing a scientific paper is essential for cardiologists and researchers aiming to make a meaningful contribution to cardiovascular medicine. A well-structured manuscript, careful journal selection, and thorough revision process ensure effective communication and impact. Publishing research advances clinical practice, improves patient care, and fosters professional growth, leaving a lasting influence on the field.

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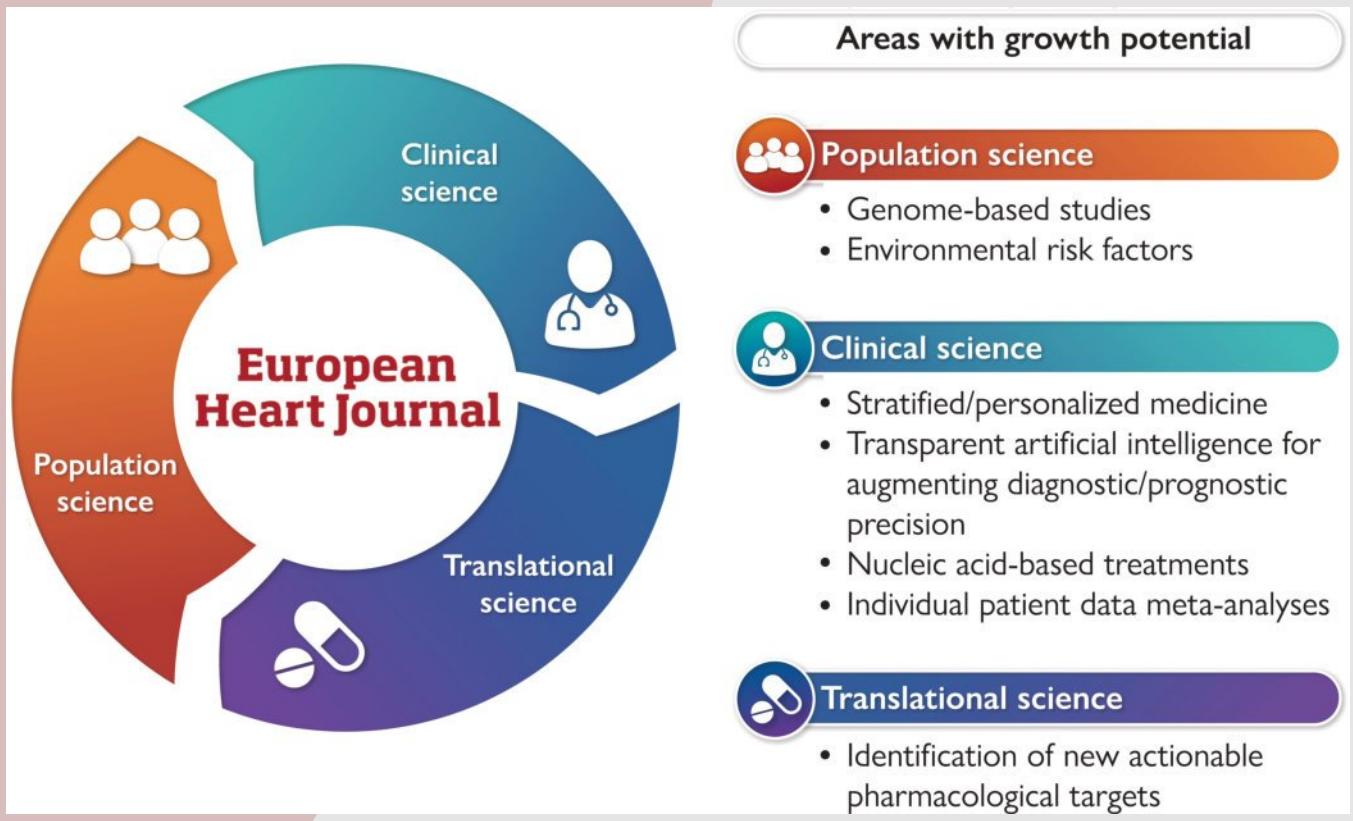

Figure 1. European Heart Journal. Area of Interest

Obstructive sleep apnea syndrome, cardiovascular risk and adropin – What is the connection?

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Summary

Obstructive sleep apnea (OSA) syndrome is the most common organic sleep disorder that can induce multi-organ dysfunctions. The developed hypoxia in the course of the disease leads to oxidative stress and inflammation, with subsequent vascular damage that is at the base of the development of different cardiovascular complications. The polysomnography (PSG) test, as a gold standard for the diagnosis of OSA syndrome, is not always available and applicable, which requires searching for other approaches for supporting the diagnosis and predicting the severity of OSA. Considering the anti-inflammatory properties of adropin, its importance for the maintenance of endothelial homeostasis and that hypoxia is a factor for inflammation and endothelial dysfunction in OSA patients, a relationship between adropin levels and OSA could be supposed.

Key words: Sleep apnea, cardiovascular complications, endothelial dysfunction, adropin

Извадок

Синдромот на опструктивна ноќна апнеја (OSA) е најчестото органско нарушување на спиењето кое може да предизвика дисфункции на повеќе органи. Развиената хипоксија во текот на болеста доведува до оксидативен стрес и воспаление, со последователно васкуларно оштетување кое е во основата на развојот на различни кардиоваскуларни компликации. Тестот за полисомнографија (PSG), како златен стандард за дијагноза на OSA синдром, не е секогаш достапен и применлив, што бара пребарување на други пристапи за поддршка на дијагнозата и предвидување на тежината на OSA. Со оглед на антиинфламаторните својства на адропинот, неговата важност за одржување на ендотелната хомеостаза и дека хипоксијата е фактор за воспаление и ендотелијална дисфункција кај пациенти со OSA, може да се претпостави врска помеѓу нивоата на адропин и OSA

Клучни зборови: Sleep apnea, кардиоваскуларни компликации, ендотелна дисфункција, адропин

OSA syndrome and its prevalence

Obstructive sleep apnea (OSA) is a chronic respiratory disease and is considered the most common organic sleep disorder that affects the quality of sleep and health of more than 100 million people of the adult population worldwide. It occurs with repeated pauses in breathing during sleep, alternating with loud snoring [1, 2, 3]. OSA syndrome is characterized by repeated series (lasting more than 10 s and a desaturation ≥ 4%) of total absence (apneas) or reductions of respiratory flow of more than 50% (hypopneas) during sleep due to obstruction of the upper respiratory tract [3, 4]. The interruption of breathing puts the organism in conditions of oxygen starvation and hypoxia. This increases the number of awakenings during the night and leads to insufficient sleep with subsequent excessive daytime sleepiness, reduced work capacity, and disorders in memory, attention, and concentration. As a consequence, all organs and systems are affected: primarily the cardiovascular and endocrine ones. Obesity and male gender appear to be the strongest risk factors for OSA [5].

Pathophysiology of OSA

The pathophysiological mechanisms of OSA are complex: due to partial or complete obstruction of the upper airway during sleep, the airflow in the lungs is obstructed and this leads to apnea, hypopnea and/or increased respiratory effort. The developed hypoxia in the course of the disease leads to oxidative stress, activation of the sympathetic part of the autonomic nervous system and inflammation [6].

OSA and elevated sympathetic activity

Patients with OSA syndrome are characterized by dysfunction of autonomic cardiovascular regulation both at night and during the day. They have high sympathetic activity when awakening and increased arterial pressure and sympathetic tone during sleep. During episodes of apnea, there is an increase in efferent sympathetic nerve activity. Hypoxia itself also leads to a disturbance in energy processes in cardiomyocytes and a decrease in heart contractility. Consequently, the sympathetic nervous system is activated, which increases heart rate and provokes peripheral vasoconstriction, further increasing left ventricular preload [7]. The stimulated sympathetic activity is provoked by the chemoreflex stimulation due to a drop in partial oxygen pressure in the arterial blood and hypercapnia during the apnea episode and is one of the main factors responsible for the increase in arterial pressure and heart rate, which accompanies the recovery of ventilation after each apneic episode.

Oxidative stress and inflammation in OSA

Transient hypoxia during apneic/hypopneic cycles in patients with OSA was considered a factor that provokes an excessive formation of reactive oxygen species (ROS). The developed oxidative stress (OS) leads to disorders in DNA, proteins, lipids and overall cell structure, as well as to systemic inflammation and apoptosis [8, 9]. Studies with animal models of OSA confirm elevated OS biomarkers: parameters of lipid peroxidation, antioxidant enzyme levels and free oxygen radicals producing enzymes [10, 11].
In addition, the intermittent hypoxia and the accumulation of ROS in OSA stimulate the production of proinflammatory factors, like C-reactive protein (CRP), tumor necrosis factor (TNF) and some interleukins, thus further exacerbating the severity of the disease [12].

Cardiovascular risk in patients with OSA syndrome

Recently, lots of studies have reported the association between OSA and various cardiovascular disorders (such as hypertension, myocardial infarction, etc.), thus presenting the importance of OSA syndrome for the cardiovascular risk [13]. In a 4-year study [14], our team diagnosed OSA syndrome in 106 patients using a polygraph test at home (SOMNO check Cardio, Loewenstein medical, Germany): a multi-channel device measuring respiratory flow, snoring, saturation, pulse rate, plethysmography, and pulse wave analysis (Fig 1). A large number of these OSA syndrome patients were polymorbid with concomitant cardiovascular diseases (Fig.2). The significance of vascular lesions has been confirmed in the pathogenesis of multiple cardiovascular and cerebrovascular complications in OSA [15].
Endothelial dysfunction is also a feature of patients with OSA, from studies examining forearm blood flow, intima-media thickness, carotid-femoral pulse wave velocity, number of circulating progenitor endothelial cells, and vascular endothelial growth factor. As this disorder is associated with a transient decrease in oxygen saturation at night, endothelin (ET-1) is a potent vasoconstrictor, released in response to hypoxia [1, 14, 15].
The polysomnography (PSG) test is accepted as a gold standard for the diagnosis of OSA syndrome. With the help of PSG, numerous parameters are monitored and recorded during sleep. However, one of the biggest disadvantages of this method is that it requires the use of complex equipment and an overnight stay in a specialized laboratory, where the sleeping conditions are different from those in the patient's home. Therefore, the results of the PSG test often show the current state for a particular night only and do not always correspond with the true condition of the patient [9, 14].
In search of supporting the diagnosis of OSA syndrome and the associated cardiometabolic complications, a variety of biomarkers have been studied. Some authors present in their research several parameters of inflammation that show a correlation with the severity of OSA. The majority of researchers suggest reliable indicators related to oxidative stress, while others - some cardiovascular biomarkers like natriuretic peptides [8, 9, 12, 16]. It could be supposed that when PSG is not available, these parameters and clinical data would be helpful in the diagnosis of the disease and prediction of its severity.

Adropin

Adropin was identified in 2008 by Kumar et al. in a study of gene expressions in melanocortin-3 receptor-deficient mice [17]. Secreted adropin is a peptide that consists of 43 amino acids with an identical sequence in rats, mice, pigs and humans [18]. The gene that encodes adropin is an energy homeostasis-related gene (Encho) that is predominantly expressed in the brain and liver but is also detected in many other tissues like the heart, lungs, kidneys, etc. [18, 19]. Nowadays, there is enough evidence indicating that adropin production and secretion are nutrition-dependent, revealing the role of the peptide in glucose and lipid metabolism and its connection to obesity and diabetes. However, new findings show that adropin plays a role in many other processes in the body, incl. cardiovascular functions, especially that of endothelium [18, 19]. Several studies reveal an association between adropin and cardiovascular diseases, indicating lower serum adropin levels in patients suffering from coronary artery disease, atrial fibrillation, hypertension and atherosclerosis. Probably downregulation of adropin levels in these conditions could serve as a potential marker for early diagnosis and the prognosis for possible cardiovascular complications [19, 20].
It has been proven that adropin administration improves heart function and coronary flow and has a protective effect on endothelial cells [21, 22]. A study with rats on a high-fat diet low dose of adropin reduced hyperlipidemia and decreased mRNA expression of pro-inflammatory cytokines, showing that this peptide could play an anti-inflammatory role in many organs including heart and vessels [23]. It was shown that adropin could modulate the anti-atherosclerosis process by inhibiting TNFα-induced adhesion molecules of endothelial cells, suggesting that adropin is involved in controlling the functions of endothelial cells [18]. By stimulating the expression of endothelial nitric oxide (NO) synthase, responsible for the production of vascular NO in the endothelium, adropin exerts a protective role in maintaining endothelial homeostasis [20].

Adropin and OSA syndrome

Considering the anti-inflammatory properties of adropin, its importance for the maintenance of endothelial homeostasis and that hypoxia is a factor for inflammation and endothelial dysfunction in OSA patients, a relationship between adropin levels and OSA could be supposed (Fig.3). Unfortunately, research done in this direction is still scarce.
A study by Bozic et al. 2018 [24], performed on 50 adult male patients with OSA, reported a significant correlation between adropin plasma levels and parameters of inflammation and data from the PSG test. Since the decrease in adropin levels was dependent on the severity of OSA, the authors suggest that adropin could play a role in the pathophysiology of the disease and peptide levels could serve as a predictor for OSA intensity. Chelikhisar et al. 2020 have found that adropin changes proportionally depending on OSA status, with adropin levels determined to be elevated in moderate or severe OSA patients [19]. A strong correlation between adropin and parameters of endothelial dysfunction (ET-1, NO) in OSA syndrome was found by Fan et al. [25]. The authors report more reduced adropin levels in OSA

patients who had endothelial dysfunction in comparison to those OSA patients without that vascular damage. A study by Kong and Liu [13] concerning vascular adhesion protein-1 and adropin levels in 50 male patients with PSG proven OSA reveals that the most elevated markers of inflammation (IL-6, TNF-α, and high sensitive CRP) were in patients with severe OSA, accompanied by the most significant drop in plasma levels of adropin, respectively.

Conclusions

Based on the current data, it could be assumed that circulating adropin levels may be used as a biomarker in predicting endothelial dysfunction in OSA syndrome patients before the clinical manifestation of cardiovascular complications. Yet, more studies will need to be performed to reveal the cardiovascular risk of OSA syndrome and the possible relation with adropin levels.

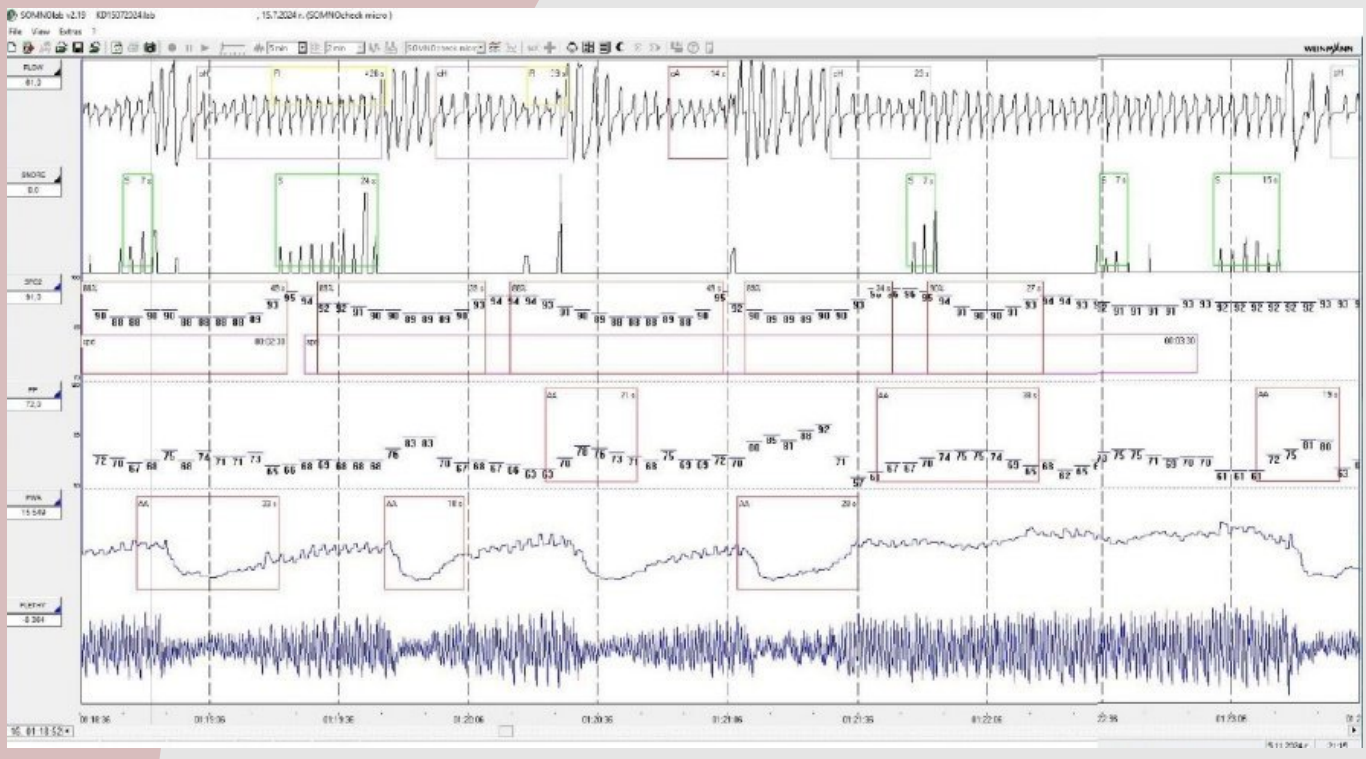

Figure 1. Multi-channel polygraph test (SOMNO check Cardio, 6s оњн дата): device measuring respiratory flow, snoring, saturation, pulse rate, plethysmography, and pulse wave analysis.

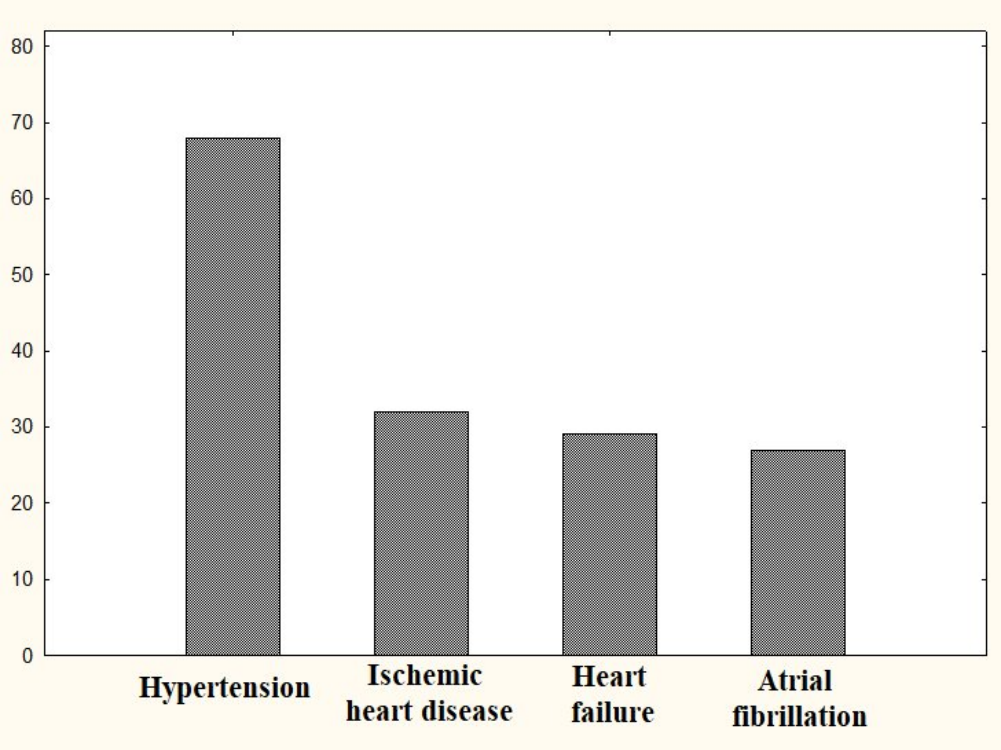

Figure 2. Concomitant cardiovascular diseases in a 4-year study study among 82 patients diagnosed with OSA syndrome4

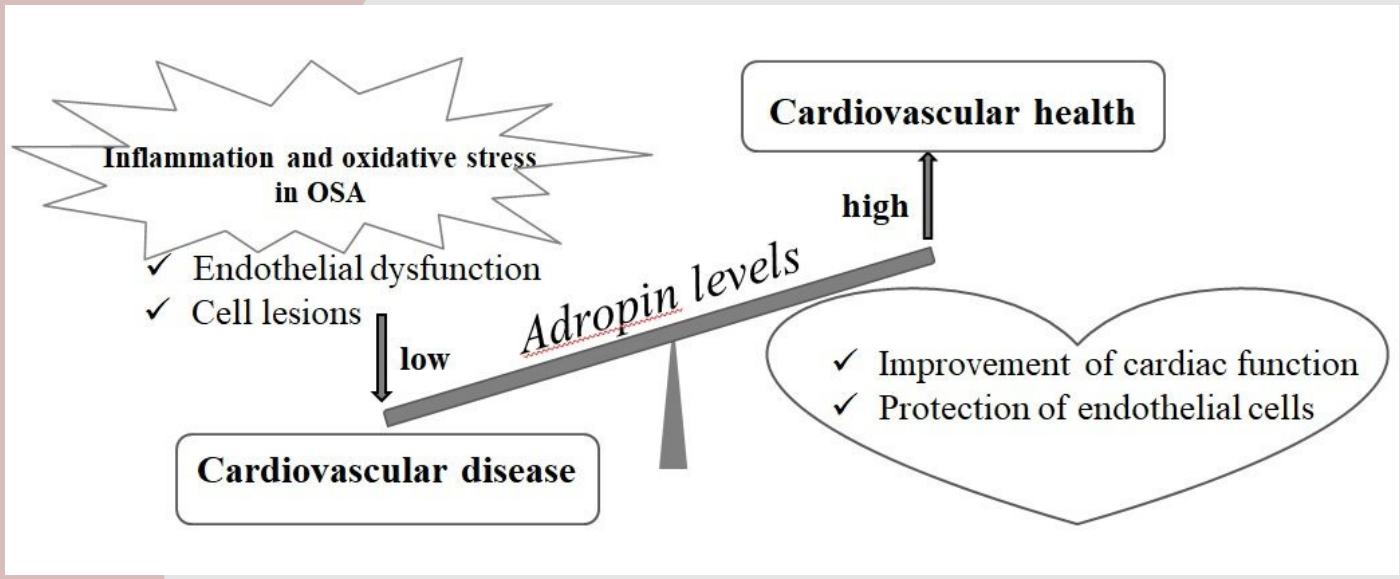

Figure 3. Relationship between adropin levels, cardiovascular health and OSA 2

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Art is medicine / Medicine is art

Сашо Поповски. Ева