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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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INFLUENCE OF ESSENTIAL HYPERTENSION ON RIGHT VENTRICULAR MORPHOLOGY AND FUNCTION

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Abstract.

Objectives: Right ventricular (RV) morphologic and functional changes still remain a mystery in patients with AH. The aim of this study was to evaluate the influence of essential hypertension on RV function and morphology.

Materials and methods: 75 nonsmoker hypertensive male patients (mean age 57.13± 7.27) and 25 normotensive control subjects (mean age 57.56±7.55) were recruited in a study. All participants underwent 24-hour ambulatory blood pressure monitoring. Heart ultrasonography was performed to assess RV morphology and its systolic and diastolic function.

Results: In comparison with normotensive subjects, hypertensive patients had significantly higher RV wall thickness and significantly lower TAPSE (5.36±0.98 and 19.86±2.68 vs 4.11±0.50 mm and 22.52±2.02, P<0.0001). RV hypertrophy was found in 38.66% of hypertensive subjects. EF of RV in normotensive subjects was significantly higher than in hypertensives (62.73±12.81 vs 57.58±7.53%, respectively). RV mean E/A was significantly lower in hypertensive group (1.41 ± 0.13 vs 0.89 ± 0.15, P < 0.001). RV diastolic dysfunction was found in 54.6% and systolic dysfunction in 7% of hypertensive subjects. The RV E/e′ ratio was increased in hypertensives (4.84 ± 0.97 vs. 3.88 ± 0.32 in the control group, P < 0.05). Tricuspid and mitral E'/A' ratio was decreased in hypertensive group (0.79 ± 0.13 and 0.90 ± 0.19 in hypertensive vs. 1.21 ± 0.15 and 1.29 ± 0.15 in the control groups, respectively, P < 0.001 for both).

Conclusions: According to study data, AH affects both ventricles simultaneously and causes concentric remodeling, hypertrophy, and functional disturbances in both ventricles; hence, in comparison with systolic dysfunction, existence of diastolic dysfunction was more prevalent in hypertensive population.

Key words. Right Ventricle, Heart Remodeling, Arterial Hypertension, Heart dysfunction.

Introduction.

Arterial hypertension (AH) is one of the major risk-factors of cardiovascular morbidity and mortality. It is responsible for 8·5 million deaths from stroke, ischemic heart disease, other vascular diseases, and renal disease worldwide [1,2]. Historically, an enormous attention was paid to the assessment of left chambers of the heart in patients with AH. Hence, it is well proven, that AH is associated with left ventricular hypertrophy (LVH) and remodeling, causing diastolic, as well as systolic heart failure. The importance of right ventricular (RV) functional and anatomical changes has long been neglected; consequently, less studies were addressed to the investigation of RV anatomy, morphology, and function in patients with AH [3]. The importance of RV was first described by Sir William Harvey in his treatise "De Motu Cordis" in 1616. Therefore, till 1950th, assessment of right heart sizes, geometry and function was lacking, because of underestimation of its meaning and absence of adequate investigation methods.

Ferlinz et al. were the first who hypothesized that in hypertensive patients' RV function may be affected by chronic left ventricular pressure overload and revealed an augmented RV afterload due to the increased pulmonary pressures in hypertensive patients, which leads to the higher end systolic volume index (ESVI) and lower ejection fraction (EF). Their study data showed, that in comparison with normotensive subjects, hypertensive patients had significantly increased right-sided heart pressures and volumes [4,5]. Atkins et al. revealed increased pulmonary vascular resistance in patients with AH [6]. Myslinski et al. via echocardiography assessed RV function and structure in 59 hypertensive patients. Study results showed existence of the impaired diastolic filling of left and right ventricles, hypertrophy of interventricular septum and thickening of a free wall of RV [7]. Akintunde et al. revealed changes in diastolic wave velocities and RV wall and inner dimensions [8]. Cuspidi et al. showed that 20% of hypertensive patients who visit specialist because of AH have biventricular hypertrophy [9,10]. Involvement of the RV and the development of cardiac hypertrophy in response to AH in both ventricles was reported by Iliev et al. in 2019 as well [11]. Hence, we can suggest that AH affects the whole heart and both ventricles physiologically represent a single functional unit. Moreover, right ventricle has found to be a significant contributor to heart hemodynamics and cardiac output in patients with different cardiovascular pathologies [12,13]. Multiple studies showed RV function and geometry changes in patients with AH, heart failure, vascular heart disease, mitral and aortic valve diseases, shock, sepsis, etc. [14-20]. Other studies revealed strong relationship between RV changes and cardiovascular mortality and morbidity [21-23]. According to Sanders et al., a decrease in RV systolic function is an independent predictor of adverse outcomes, including heart failure-associated hospitalization and mortality [24].

Despite clinical studies support the idea that the RV is affected by AH along with LV, right heart remodeling and its assessment principles are not even mentioned in the world's leading guidelines of AH management [15,25-31]. The basics of RV dimensions and function were partly and quite superficially described in the recommendations for chamber quantification published in 2005 by the American and European Societies of Echocardiography, which mainly focused on the left heart assessment [32]. Relatively complete recommendations of RV assessment were published in 2010 in the "Guidelines for the Echocardiographic Assessment of the Right Heart in Adults" [33]. Hence, because of the importance of RV function for prognosis, there is a need for further improvement of

diagnostic methodology as well as standardization of the RV echocardiographic and other imaging findings.

The importance of right ventricular behaviour and its changes still remain a mystery in patients with AH. Published data regarding hypertension mediated RV changes are scarce, heterogeneous, insufficient, and sometimes contradictory. Hence, the aim of this study was to evaluate influence of essential hypertension on right ventricular function and morphology.

Methods.

75 male patients (mean age 57.13± 7.27) with documented AH for at least 1 year and 25 normotensive subjects (mean age 57.56±7.55) were recruited in a study. All study participants were either lifetime nonsmokers or quit smoking at least for 5 years. All 100 individuals gave informed consent for participation in a study. All the participants underwent to the 24-hour ambulatory blood pressure monitoring. AH was diagnosed as an average 24-hour BP ≥130/80 mmHg, or an average daytime BP ≥135/85 mmHg or an average nighttime BP ≥120/70mmHg. For BP levels in calculations were used mean 24-hour systolic and diastolic BP. None of the study participants had a history of pulmonary disorders, congestive heart failure, cerebrovascular disease, or significant valvular disorders. It can be suggested that some of the study participants may have had latent coronary artery disease, but cannot be proofed, because coronary angiography was not performed in any of them. Therefore, none of the subjects had ECG or ultrasound changes typical for coronary artery disease and a history or symptoms indicative on it. All study participants had regular sinus rhythm without any specific changes and/or conduction abnormalities. All the study participants were undergone to the respiratory functional test, i.e., spirometry to exclude existence of obstructive lung disease. Some of the hypertensive patients were on antihypertensive treatment with recommended medications. These were stopped at least 2 weeks before the study and were substituted by short acting medications, which may be applied only in the case of high blood pressure numbers as a symptomatic treatment and not for control. None of the patients developed neither potentially dangerous acceleration of hypertension, nor developed any new symptoms or hypertension complications.

Echocardiographic examinations were performed using Hitachi EUB-450 (Hitachi Medical Corporation, Tokyo, Japan) ultrasonography with 3,5 MHz transducer. Evaluation was performed in accordance with the "Guidelines for the Echocardiographic Assessment of the Right Heart in Adults" [33]. Patients were in left decubitus position. M-mode presentation in the parasternal view was used to measure posterior wall diastolic diameter (PWD), interventricular septum diastolic diameter (IVSD), right ventricular wall (RVW) end-diastolic diameter, RV outflow tract diameter (RVOTD), left atrium diameter (LAD) and LV end-diastolic and end-systolic diameters (LVEDD & LVESD). RVW thickness ≥ 5 mm was considered as RV hypertrophy [34]. M-mode measurements were used to calculate LV ejection fraction by the Teichholz formula.

Mitral and Tricuspid flow velocities, namely, early diastolic peak flow velocity (E), late diastolic peak flow velocity (A) and the ratio of early-to-late flow velocity peaks (E/A ratio) were assessed by Pulsed Doppler from a four-chamber view. Tissue Doppler was used to measure mitral and tricuspid annular plane velocities, namely early diastolic (E') and late diastolic (A') velocities.

According to the American Society for Echocardiography and the European Association of Cardiovascular Imaging, for evaluation of diastolic function of left and right ventricles we used: a) The ratio between **E-wave** and **A-wave** (**E/A ratio**); and b) Estimating left and right ventricular filling pressure via **e'. Apart from it, we used** E'/A' ratio, which is one of the recommended indices approved by the American Society of Echocardiography for assessing RV diastolic dysfunction [35]. The American Society of Echocardiography suggested that patients with an E/A ratio ≤0.8 should be considered as having an early stage of LVDD [36]. Consequently, we defined E/A ratio abnormality as E/A ratio ≤ 0.8, and normal E/A ratio as 0.8 < E/A ratio < 1.5.

LV systolic function was assessed via calculation of EF%. Its meaning more than 56% was considered as normal. For the assessment of systolic function of RV, we used Tricuspid Annular Plane Systolic Excursion (TAPSE) and right ventricular EF. TAPSE, we obtained by placing the M-mode cursor through the lateral portion of the tricuspid valve annulus in the apical four-chamber view, as it is indicated by guidelines. The excursion of the tricuspid valve from the base of the heart towards the apex is measured as the distance from the annulus to the apex at end diastole minus that distance at end systole. TAPSE does not depend on the geometry of the RV and assesses longitudinal function of RV. TAPSE value < 17 mm indicates systolic dysfunction. RV EF was calculated with equation: RVEF % = 2.9*TAPSE, suggested and later on proofed by Guzmán-Sánchez in 2017 [37].

Using conventional two-dimensional echocardiography, we assessed left and right ventricular end-systolic (ESV) and end-diastolic volumes (EDV). The ESV is referred to as the volume of blood in the left or right ventricle at the end of the systolic ejection phase immediately before the beginning of diastole or ventricular filling. For assessment of EDV and ESV Indexes (EDVI & ESVI), we corrected the volumes for the body surface area (BSA). For body surface area calculation, we used Du Bois formula: BSA(m2) = 0.20247 x height (m) 0.725 x weight (kg) 0.425.

Echocardiographic LVH was defined by left ventricular mass (LVM) indexed for BSA (LVM/BSA) as suggested by Zhang et al. [38]. For assessment of LV mass was used formula proposed by Devereux et al. [17,39]. LV mass (g) = 0.8(1.04{(LVIDD + IVST + PWT)3 - LVIDD3}) + 0.6g, where LVIDD is LV internal end-diastolic dimension, IVST is the end-diastolic interventricular septal wall thickness, and PWT is end-diastolic LV posterior wall thickness [40]. Normal values of LV mass indexed to body surface area were considered to be 70±9g/m2 in men [41]. According to the ESH/ESC guidelines, as a cut-off value used to define LVH, based on the relationship between ventricular mass and body surface, was 115 g/ m2 for men and 95 g/m2 in women [42,32].

According to the Relative Wall Thickness (RWT) and LVMI, all the patients we classified into 4 categories, namely:

- If the LVMI exceeds 115 g/m2:
- RWT > 0.42 – Concentric Hypertrophy
- RWT ≤ 0.42 – Eccentric Hypertrophy
- If the LVMI ≤115 g/m2
- RWT > 0.42 – Concentric remodeling
- RWT ≤ 0.42 – Normal heart.

The American Society of Echocardiography criteria were used to categorize subjects based on LV internal dimension in diastole (LVIDD) [43]. These criteria classify the LV size as *normal* (men: 42 to 59 mm; women: 39 to 53 mm), *mildly dilated* (men: 60 to 63 mm; women: 54 to 57 mm), *moderately dilated* (men: 64 to 68 mm; women: 58 to 61 mm), or *severely dilated* (men: ≥69 mm; women: ≥62 mm).

Statistical analysis.

Data was analyzed by using SPSS Statistics for Windows, Version 16.0. Quantitative data were summarized using means ± standard deviation. Qualitative data were summarized using percentages and proportions. Comparisons between groups were made using independent t-test and chi-square as appropriate. Pearson R statistical test was done for measurement the strength between the different variables and their relationships. Statistical significance was taken as $p < 0.05$.

Results.

Age, body mass index and heart rate were similar in both groups. All study participants were males and nonsmokers. Systolic (SBP) and diastolic blood pressures (DBP) were significantly higher in hypertensive subjects compared to the controls. General characteristics of the hypertensive and normotensive groups are listed in Table 1.

Table 1. *General characteristic of the examined population.*

Characteristic	Hypertensives (n=75)	Normotensives (n=25)	P-value
Age (yrs)	57.13± 7.27	57.56±7.55	NS
BMI (kg/m2)	28.7 ± 3.9	28.5 ± 3.3	NS
SBP (mm Hg)	153.93±8.60	125.04±7.83	P<0.001
DBP (mm Hg)	94.6±8.16	77.8±5.15	P<0.001
Heart rate, bpm	70.89±8.64	72±7.46	NS
History of diabetes	22 (29.3%)	7 (28%)	NS
History of dyslipidemia	58 (77.33%)	16 (64%)	NS

BMI: body mass index; DBP: diastolic blood pressure; SBP: systolic blood pressure; NS: non-significance.

In comparison with normotensive subjects, hypertensive patients had significantly higher RVW and IVS thickness in diastole and left atrium diameter (LAD) (5.36±0.98 mm, 1.25±0.16 cm and 4.11±0.50 cm vs 2.88±0.64 mm, 0.88±0.2 cm and 3.45±0.06 cm in normotensive controls, $P < 0.001$ for all). There was no significant difference in right atrial septal-lateral diameter, aortic bulb, and pulmonary trunk diameter between hypertensive and normotensive groups (3.70±0.61, 3.38±0.53 and 2.97±0.12 vs 3.5±0.56, 3.41±0.11 and 2.91±0.07, consecutively. P=NS).

All study participants had normal range of LV internal dimension in diastole (LVIDD) (5.19±1.76 vs 4.44±1.81 in hypertensive and normotensive groups respectively; P=0.0699). Only 5 patients had mild dilatation of left ventricle from the hypertensive group and none from the normotensive individuals.

Left ventricular concentric hypertrophy was found in 36 hypertensive patients, 21 patients had concentric remodeling and 18 normal left ventricular mass index without evident remodeling or hypertrophy. Among those with concentric hypertrophy, severely enlarged left ventricular mass was detected in 9 patients, moderately enlarged in 17 patients, and mildly enlarged in 9 patients. None of the control group subjects had echo findings, indicative for LVH. LV mass indexed for BSA, LV mass index and relative wall thickness of hypertensive and normotensive study participants are given in table 2.

Table 2. *LV mass index and relative wall thickness of study population.*

Characteristic	LV mass indexed for BSA, g/m2	LV mass index	Relative Wall Thickness
Hypertensive patients (n=75)	116.75±26.83	242.38±58.10	0.466±0.07
Hypertensive patients with LV concentric hypertrophy (n=39)	139.76±17.55	283.76±45.4	0.498±0.05
Hypertensive patients with LV concentric remodeling (n=18)	98.23±14.87	210.54±32.38	0.500±0.053
Hypertensive patients with normal heart (n=18)	91.09±8.05	184.56±22.01	0.361±0.0547
Normotensive individuals (n=25)	72.22±14.67	153.82±32.3	0.383±0.04

Table 3. *M-Mode echocardiography characteristics of the study populations.*

	Hypertensives (mean ± s.d.)	Normotensives (mean ± s.d.)	Significance
RVW thickness	5.36±0.98 mm	2.88±0.64 mm	P < 0.001
IVS thickness in diastole	1.25±0.16 cm	0.88±0.2 cm	P < 0.001
Left atrium diameter	4.11±0.50 cm	3.45±0.06 cm	P < 0.001
Right atrial septal-lateral diameter	3.70±0.61	3.5±0.56	NS
Aortic bulb	3.38± 0.53	3.41±0.11	NS
Pulmonary trunk diameter	2.97±0.12	2.91±0.07	NS
LV mass indexed for BSA	116.75±26.83	76.72±14.56	P < 0.005
Ejection fraction of left ventricle	61.02±5.06	58.08±2.46	NS
LV internal dimension in diastole (LVIDD) cm	5.05±0.43	4.44±1.81	NS

NS – not significant

16% hypertensive patients (n=12) had bi-ventricular hypertrophy, 8 patients - LV concentric remodeling with RV hypertrophy, 24 (32%) patients - isolated LV hypertrophy, 6 patients - isolated right ventricular hypertrophy. Both ventricles were normal in 12 patients. We found a positive correlation between LV mass index and body mass index (r=0.792, P=0.03).

Hypertensive patients had significantly higher LV mass, relative wall thickness and LV mass indexed for BSA in comparison with normotensives (P<0.05 for all). Moreover, in comparison with normotensives, hypertensives without LV hypertrophy or remodeling still had significantly higher LV mass indexed for BSA (P<0.05). In hypertensive subjects, LV mass and LV mass indexed for BSA significantly increased from normal heart to concentric remodeling and concentric hypertrophy (P<0.05). There was no difference between relative wall thickness of hypertensive patients with LV concentric remodeling and hypertrophy (P=NS).

In comparison with hypertensive patients, where was found a significant large positive correlation between SBP level and interventricular septal and posterior wall thickness (r=0.816 and r=0.669, respectively; P<0.001 for both), normotensive subjects did not show relationship between these parameters SBP level and / posterior wall thickness (r=0.24, P=0.236 for SBP and interventricular septum thickness and r=0.18, P=0.372 for SBP and posterior wall thickness). Therefore, DBP level did not show significant positive or negative correlation with interventricular and posterior wall thickness among hypertensive and normotensive subjects (r=0.038, P=0.742 and r=-0.006, P=0.958 for hypertensive vs. r=-0.065, P=0.756 and r=-0.190, P=0.362; respectively).

SBP value was independently associated with LV hypertrophy and its diastolic dysfunction (all p<0.01). With the increase of SBP, LV mass index and E/e′ stepwise increased and e′ stepwise decreased significantly (all p<0.05). In the whole population, SBP was independently correlated with LVMI, LVEDD, e′, and E/e′ (all p<0.01).

Ejection fraction of left ventricle was not different between the study groups (61.02±5.06 for hypertensives vs 58.08±2.46 for normotensives; P=NS). Similar to EF%, LV internal dimension in diastole were not different between the groups (Tab 3).

RV hypertrophy was found in 38.66% of hypertensive subjects (n = 29). Mean value of TAPSE in hypertensive population was significantly lower than in normotensive subjects (19.86±2.68 vs 22.52±2.02; P<0.0001). According to recommendations, TAPSE <16mm, was considered as an abnormal [44]. Only 5 patients had mild systolic dysfunction of RV with an average TAPSE 16 in hypertensive patients and none in normotensive control group.

EF of RV in normotensive subjects was significantly higher in comparison with hypertensive patients (62.73±12.81 vs 57.58±7.53%, respectively). Therefore, RV ejection fraction less than 44% was considered as an abnormal systolic function [44]. There was no significant positive relationship between left and right ventricular EF in hypertensive and normotensive groups (r=0.159, P=0.171 and r=-0.036, P=0.826; respectively).

In comparison with normotensive subjects, hypertensive patients had significantly increased right-sided heart volume indexes (84.57±7.30 vs 68.76±7.45, P<0.05 for EDVI and 35.61±6.67 vs 22.28±2.85, P<0.05 for ESVI).

Mitral E/A ratio was 1.18 ± 0.09 in controls and 0.84 ± 0.14 in hypertensive groups, respectively (P < 0.001). E/A <0.8 was found in 48 hypertensive patients (0.75±0.055); among them 12 had LV concentric remodeling and 36 concentric hypertrophy (0.76±0.06 vs 0.74±0.05; P=NS). According to normal E/A level (>0.8), 18 hypertensive patients had normal heart and 9 patients - concentric remodeling. We found negative significant correlation between left ventricular mass index and E/A (r=-0.454, P<0.001).

LV mean e′ in hypertensives was significantly lower than in normotensives (6.63±1.027 vs 8.5±1.78; P<0.0001). We did not reveal correlation between e′ and SBP, DBP or LV mass index (r=0.095, P=0.413 and r=0.024; P=0.833, r=0.026, P=0.823; respectively).

RV diastolic dysfunction defined as E/A < 0.8 was found in 54.6% (n=41) of hypertensive patients. Right ventricular mean E/A was 1.41 ± 0.13 in control and 0.89 ± 0.15 in hypertensive participants (P < 0.001). Therefore, none of the control group subjects had RV diastolic dysfunction. Positive significant correlation was found between mitral and tricuspid E/A ratio in hypertensive patients and control subjects (r = 0.282, P < 0.001 for HTN and r=0.558, P<0.001 for normotensive controls).

The right ventricular E/e′ ratio, which is a reflection of RV filling pressure and is considered as a surrogate of right atrial pressure (RAP) was increased in hypertensive group (4.84 ± 0.97 vs. 3.88 ± 0.32 in the control group, P < 0.05). Tricuspid and mitral E'/A' ratio was decreased in hypertensive group (0.79 ± 0.13 and 0.90 ± 0.19 in hypertensive vs. 1.21 ± 0.15 and 1.29 ± 0.15 in the control groups, respectively, P < 0.001 for both).

There was a significant small negative relationship between left and right ventricular E/A ratio and positive correlation between left and right sided E/E' ratio (r=0.218, p=0.037 and r=0.246, P=0.03, respectively).

Discussion.

Our study revealed an increased RV wall thickness, significantly thicker IVS and PW, and higher peak atrial velocity in hypertensive individuals. Cuspidi et al. assessed heart morphology in 330 untreated and treated uncomplicated essential hypertensive patients. Similar to our study results, RV hypertrophy was found in 33.6% (vs. 34.6% in our study) and bi-ventricular hypertrophy in 15,7% of patients (vs. 16% in our study) [45]. Our study results point out that hypertensive subjects in comparison with normotensive controls have higher RV mass and RV wall thickness. Similar data were obtained by Todiere et al via analyzing results of cardiac magnetic resonance imaging [46]. RV hypertrophy in essential hypertension could be explained via three major mechanisms. First, an overstimulation of the sympathetic and the renin-angiotensin-aldosterone systems, which are the cornerstone for hypertension pathogenesis and could be responsible for increased pulmonary arteriolar resistance and, RV hypertrophy. Second, the mechanical interaction between the right and left ventricles through the interventricular septum and third, oxidative stress and endothelial dysfunction could induce changes in pulmonary circulation leading to RV hypertrophy.

Previous studies have reported that patients with impaired LV relaxation, i.e., diastolic dysfunction have an increased risk of sudden cardiac death [47]. According to published literature, the early to late diastolic transmitral flow velocity (E/A ratio) is considered as the key marker for assessment of left ventricular

relaxation impairment [48,49]. By experts, E/A ratio <0.8 has been recognized as a strong indicator of LVDD and related to the poor prognosis of heart failure [50,51]. That's why in our study we used E/A ratio to assess LV diastolic function and indirectly predict prognosis. Similar to Hanboly et al., who showed a 60% prevalence of RV diastolic dysfunction, in our study RV diastolic dysfunction was found in 54.6% hypertensive patients [52]. Both studies revealed decreased TAPSE in hypertensive population. In comparison with normotensive control subjects, hypertensive patients in our study had decreased tricuspid and mitral E'/A' ratio. These data are in accordance with the data obtained by other investigators [18]. Hence, our study revealed significant correlation between mitral and tricuspid E/A ratio.

Zhou et al. aimed to investigate the association of tissue Doppler E/e' with cardiac events in hypertension patients and retrospectively enrolled 222 asymptomatic nonischemic patients with AH. They showed that E/e' ratio represents an early, effective tool for cardiovascular risk stratification in hypertensive population and is the strongest predictor of cardiac events. Namely, they showed, that compared with patients whose E/e' ≤ 14, patients whose E/e' □14 the hazard ratio of cardiac event is significantly increased [53]. In our study, Mitral E/e' was significantly higher in hypertensive individuals and 5 of them had E/e' >14.

Like Yongtai et al., our study revealed an independent association between LV hypertrophy and LV diastolic dysfunction (all P<0.01) [54]. Similar to the results obtained by Eliakim-Raz et al., we clearly showed positive correlation between SBP and interventricular septal and posterior wall thickness (P<.001, r= 0.121) [55]. Jung et al. via using multivariate logistic regression analyses, assessed the odds ratios for LV hypertrophy and increased relative wall thickness and found a significant correlation between elevated BP and LVH, LV mass index and relative wall thickness [56]. Similar results were obtained by Hendriks et al, who using Mendelian randomization showed that each 10 mm Hg increase in SBP was significantly associated with 4.01 g increase in LV mass (P=0.002) [57].

In a large cohort of primary care patients with normal LVEF and free from HF, Nistri et al. showed, that low septal e' is an independent and incremental predictor of overall mortality and cardiovascular hospitalizations [58]. Hence, we found out that hypertensive patients had significantly lower septal e' in comparison with normotensives. Therefore, in our study we could not revealed correlation between SBP or DBP level and e'.

In comparison with normotensives, our study showed significantly lower level of right ventricular ejection fraction in hypertensive individuals, which is in complete accordance with the data obtained by Koutsampasopoulos et al. [15]. We believe that RV wall and interventricular septum thickening may play an important role in RV diastolic dysfunction in patients with AH. Moreover, it should be considered that lower than normal RV ejection fraction in hypertensive patients might be indicating the impairment of RV contractility. Thus, we can suggest that the assessment of RV performance in patients with AH, may be an additional, sensitive indicator of the course of disease.

Hence, we can agree with the data obtained by other investigators that arterial hypertension impacts RV morphology and its diastolic and systolic function. We may suppose that impairment of RV systolic function in hypertensive population could be related with right ventricular hypertrophy and ventricular interaction. Tadic et al. explained the deterioration of RV diastolic function as happening due to the increased stiffness of the RV caused by hypertrophy, retrograde transmission of increased LV filling pressure to the pulmonary circulation and ultimately to the RV and negative influence of renin-angiotensin-aldosterone and sympathetic nervous system on pulmonary circulation and the RV [18]. Abdeltawab et al. demonstrated that RV diastolic dysfunction is not only an early marker that is correlated to the presence of essential hypertension but also showed it to be a marker of its severity and degree [53].

Conclusion.

The study data point out that AH affects left and right ventricles simultaneously and causes morphological and functional disturbances in both ventricles. AH causes left and right ventricular concentric remodeling and later on ventricular hypertrophy. Hypertension affects the diastolic function of the left ventricle, and these changes are accompanied by similar changes in the right ventricle. Hypertension affects RV systolic function as well, but less frequently.

Despite the fact that we proved interaction between the left and right ventricles while AH, question regarding prognostic value of RV function and its association with clinical outcomes is still opened and unanswered. Hence, further studies are still needed to completely understand mechanisms of biventricular morphologic and functional changes in patients with arterial hypertension, to determine the clinical utility and prognostic value of RV functional and morphological changes in patients with AH. Whether these right heart changes occur early, at the same time as left heart changes due to the interdependence of the two structures, or whether it is a secondary phenomenon possibly related to pulmonary vascular changes remains to be proven by further studies. It is immensely important to standardize and improve the diagnostic methodology of RV evaluation, which might have a crucial meaning for professionals involved in hypertension management to improve patients' prognosis.

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