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A Comparative Quantitative Analysis of the Postnatal Changes in the Myocardium of the Left and Right Ventricles in Rats

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Background: The growth of the heart during the foetal and early postnatal development takes places mainly due to hyperplasia. The late postnatal development is characterised by cardiomyocytic hypertrophy in response to normal physiological mechanisms and increased load. To study the cell size most authors measure the diameter either directly or indirectly.

Aim: The aim of the present study was to make a comparative quantitative analysis of the postnatal changes observed in the left and right ventricles in rat by evaluating the changes in three morphometric parameters – thickness of the free wall, transverse section of the cardiomyocytes and cardiomyocytic density in the left ventricle and right ventricle.

Materials and methods: In the present study, we used histological material from the hearts of 15 male Wistar rats, distributed in five groups aged 2 weeks, 1 month, 3 months, 6 months and 12 months, respectively.

Results: In both ventricles, the wall thickness and the transverse section of the cardiomyocytes increased with age, while the cardiomyocytic density decreased. Changes were identical in both ventricles; however, they were more dynamic and pronounced in the left ventricle.

Conclusions: The studied morphometric parameters reveal that age-related hypertrophy and the gradual loss of cardiac muscle cells take place in both ventricles but have a more dynamic pattern of progression in the left ventricle as compared with the right ventricle.

BACKGROUND

Myogenesis and the development of the heart has been broadly discussed over the last 100 years.¹ Until recently, there was a consensus in literature that the growth of the heart during the foetal and postnatal development of the rat takes place mainly due to hyperplasia. However, during the early period of postnatal development of the heart, cardiomyocytic proliferation ceases and heart growth during the late postnatal period is a result of hypertrophy of the cardiac muscle cells. The mean size of cardiomyocytes increases in conjunction with body growth during the postnatal development. They also possess the ability for additional hypertrophy in response to increased load.²⁻⁴

When studying cell size, most authors measure the cell diameter in transverse histological slides by

adopting a cylindrical model of the cell.⁵ The diameter is measured either directly at every individual cell or indirectly. In case of direct measurement difficulties arise from the fact that cells are not always cut perpendicularly to their long axis making their transverse section appear elliptical rather than round. Therefore, some authors measure only the shortest diameter, while others measure at least two diameters for each cell.⁶⁻⁸ Often, longitudinal slides are used to measure the diameter, however in this case not all cells are cut precisely along their longitudinal axis, which makes it possible for the diameter to appear smaller.³ Indirect evaluation is based on the measurement of the total area covered only by cardiomyocytes on histological slides; this area is then divided by the number of cells in this slide.⁹

AIM

The aim of the present study was to make a comparative quantitative analysis of the postnatal changes observed in the left ventricle (LV) and right ventricle (RV) in rat by evaluating the changes in three morphometric parameters – thickness of the free wall, transverse section of the cardiomyocytes and cardiomyocytic density in the LV and RV.

MATERIALS AND METHODS

In the present study, we used histological material from the hearts of male Wistar rats (WR) with the approval of the University Committee on Animal Resources. All animals received humane care in compliance with the “Principles of laboratory animal care” formulated by the National Society for Medical Research and the “Guide for the care and use of laboratory animals” prepared by the National Institute of Health (NIH publication No. 86–23, revised 1996). The morphometric results were received from the hearts of 15 male rats, distributed in five age groups, each containing three animals: two-week-olds, one-month-olds, three-month-olds, six-month-olds, and twelve-month-olds, respectively. The animals were anaesthetised with ether and cervical dislocation was performed. The hearts were removed from the chest cavity and were placed in physiological saline in order to rinse the blood in the cardiac cavities. After a short fixation in 10% neutral buffered formalin, a transverse section was performed through the middle of the heart, a little below the level of the cardiac valves, thus dividing the heart into two halves. The hearts were then placed again in 10% neutral buffered formalin for immersion fixation. Afterwards, they were fixed in paraffin blocks from which we prepared 7- μ m-wide paraffin tissue slides. These slides were a series of transverse cross-sections through the ventricles, which were stained with haematoxylin and eosin.

The morphometric analysis was performed on five slides from the heart of each animal. Quantitative data were obtained with a computerised system for image analysis NIS-Elements Advanced Research (ver. 2.30). The areas of interest in each slide were first found on low magnifications (x100, x200), taking into account the respective age group. Afterwards, at magnification x400 we photographed an area of the myocardium, containing longitudinally oriented cardiomyocytes cut transversely, and a zone with surface area of 0.04 mm² was marked. The borders of the cardiomyocytes within the zone were outlined. Due to the fact that car-

diomyocytes are elongated, the contours frequently pass through the cells; therefore, in order to make a good comparison of the data, it was decided that the marked zone should include these cells which are adjacent, on the inside or outside, to two perpendicular contours, and also should not include cells which are adjacent, on the inside or outside, to the other two borders. After outlining the borders, the following morphometric parameters were obtained automatically: thickness of the free wall of the left and right ventricle (μ m); transverse section of the cardiomyocyte (μ m²); cardiomyocytic density – calculated as number of cells per unit of surface area of the slide (number of cells / mm²); the cardiomyocytes were determined as a general population.

The obtained quantitative data were graphically presented in diagrams and were statistically evaluated using a Student-T-test. Statistically significant differences were read in the case of $p < 0.05$. Microsoft Office Excel 2010 was used to process the data and to demonstrate the obtained results in an adequate way.

RESULTS

Quantitative data on the morphology of the free wall of the LV and RV, as well as the individual cardiomyocytes were obtained by analysing randomly selected areas without large and massive ruptures of the myocardium caused by the laboratory processing of the material (**Figs 1-3**).

Changes in the parameter thickness of the free wall of the LV and RV in the different age groups of rats showed significant differences. The thickness of the free wall of the LV showed a dynamic pattern of increase during the postnatal period; the increase in the values of this parameter was most

Figure 1. Orientation photomicrograph of a transverse section through the heart, representative of the thickness of the free walls of the left and right ventricles, H&E.

Figure 2. Photomicrograph of transversely cut cardiomyocytes in the left ventricle, H&E. Magnification x400.

Figure 3. Photomicrograph of transversely cut cardiomyocytes in the right ventricle, H&E. Magnification x400.

significant and intensive in the age groups after the third month. A thorough study of the changes in the thickness of the free wall of the RV revealed that no significant increase in the quantitative characteristics of this parameter was noted before the sixth month. To some extent, an increase in

the values of the parameter was observed in the period between the six- and the twelve-month-old animals (**Fig. 4, Table 1**).

Being one of the basic morphometric parameters, the transverse section of the cell reflects thoroughly the changes in the size of the cardiomyocytes dur-

Figure 4. Changes in the free wall thickness of left (LV) and right ventricles (RV).

Table 1. Numerical presentation of changes in the free wall thickness of left and right ventricles

Left ventricle (μm)			Right ventricle (μm)		t-test
Wistar rats	Wall thickness (μm)		Wall thickness (μm)		
Age	Mean value	SD	Mean value	SD	
2 weeks	1006.0	22.6	455.7	2.1	p<0.001
1 month	1362.0	21.2	540.7	114.5	p<0.001
3 months	1702.7	123.7	561.3	96.8	p<0.001
6 months	2596.3	60.1	571.3	2.12	p<0.001
12 months	2848.3	47.3	989.8	21.2	p<0.001

ing various periods. As a whole, the values of this parameter among the different groups from the postnatal period gradually increased, which was reflected in the ascending curves. In the cardiomyocytes from the LV, an intensive increase of the transverse section of the cardiac muscle cells was noted in all age groups, while in the cardiomyocytes of the RV, the changes were most pronounced during the third, sixth and twelfth month. A thorough investigation of the changes in the transverse section of the cardiac muscle cells revealed that in two-week-old rats, the value was almost identical in cardiomyocytes from both ventricles. After that, it gradually increased in the period up to the third month in cardiomyocytes from the RV, while in the cells of the LV, the values increased significantly during all periods. The diagram in **Fig. 5** makes it evident that after the third month, the transverse section of cardiomyocytes in the RV also increased in a more dynamic pattern (**Table 2**).

Changes in the cardiomyocytic density in the LV and RV were inversely proportional to those

observed for the transverse section of the cells. Over the whole postnatal period, the values of the cardiomyocytic density in both ventricles decreased slowly, however in the cardiac muscle cells in the LV, a significant decrease was noted between the one- and three-month-old rats as compared to the same age groups in the RV. The cardiomyocytic density in the group of the twelve-month-old animals in both ventricles had almost identical values (**Fig. 6, Table 3**).

The changes observed in the parameters outline a tendency for age-related hypertrophy. They are more dynamic and have an earlier beginning in the LV and happen significantly slower in the RV.

DISCUSSION

In general, several parameters were analysed in the present work (wall thickness, transverse section of the cardiomyocytes, cardiomyocytic density), in five age groups of animals. The changes in time observed in the cellular parameter – cardiomyocytic density – were studied for the first time in literature

Figure 5. Changes in the transverse section of the cardiomyocytes in the left (LV) and right ventricles (RV).

Table 2. Numerical presentation of the changes in the transverse section of the cardiomyocytes in the left and right ventricles

Left ventricle (μm ²)			Right ventricle (μm ²)		t-test
Wistar rats	Transverse section of the cardio- myocytes (μm ²)		Transverse section of the cardio- myocytes (μm ²)		
Age	Mean value	SD	Mean value	SD	
2 weeks	73.6	8.1	54.2	10.1	p<0.001
1 month	214.7	35.3	65.8	12.6	p<0.001
3 months	354.2	41.1	107.7	16.6	p<0.001
6 months	490.1	55.9	223.6	39.2	p<0.001
12 months	568.5	62.4	391.6	45.5	p<0.001

Figure 6. Changes in the cardiomyocytic density in the left (LV) and right ventricles (RV).

Table 3. Numerical presentation of the changes in the cardiomyocytic density in the left and right ventricles

Left ventricle			Right ventricle		t-test
Wistar rats	Number of cells/mm ²		Number of cells/mm ²		
Age	Mean value	SD	Mean value	SD	
2 weeks	2193.5	168.6	1502.2	113.3	p<0.001
1 month	1987.3	150.9	1468.6	55.9	p<0.001
3 months	950.5	54.1	1281.3	114.6	p<0.001
6 months	880.7	65.2	1237.1	208.1	p<0.001
12 months	800.6	73.7	743.9	143.1	p<0.05

and no correlations were found. Postnatal changes in the wall thickness and the transverse section of the cardiomyocytes have also been studied by other authors, which allowed us to make comparisons.²⁻⁴

According to our own data and those of other authors, the hypertrophy of the ventricular cardiomyocytes in mammals is a continuously advancing process, which takes place throughout the whole life of the animal.¹⁰⁻¹² Due to the fact that life expectancy in most rodents reaches or surpasses 2 years, the analysis of animals only from younger age groups can mask potentially critical changes occurring as aging advances, which probably take place simultaneously with hypertrophy.^{13,14} The differences in the transverse section rather than the cellular length are the most accurate parameter of hypertrophic variations in the cardiomyocytic size.¹⁵ During the full development of hypertrophy (6-12 months), the thickness of the wall of the LV in rats remains constant but the transverse section of the cardiomyocytes increases progressively. The parameter wall thickness in the LV increases without a significant increase in the transverse section.¹⁶ These data are consistent with the results of the

present study where an insignificant increase of the wall thickness, correlating with increase in the transverse section of cardiomyocytes was observed in both ventricles. The part of the LV, occupied by cardiomyocytes remains significantly high, while the amount of interstitial tissue is significantly low. As hypertrophy progresses (12-18 months) a constant value is reached (a plateau), however the wall thickness in the LV and the transverse section of the cardiomyocytes continue to increase.¹⁶ This period of hypertrophy probably represents a secondary phase of ventricular growth, related to the muscle cells, as well as to other cellular types and the extracellular matrix associated with them.¹⁷ Due to the fact that the transverse section of the cardiomyocytes doesn't decrease throughout this period, the morphometric analysis reveals that the smaller area, occupied by muscle fibres probably indicates a decrease in the cellular density as a result of larger tissue mass being taken by the interstitium. The biochemical analysis reflects the increased collagen accumulation in the ventricles as aging progresses.¹⁸

During the end stage of hypertrophy (18-24

months) the myocardium of the ventricles shows morphometric signs of degeneration. The thickness of the free wall of the LV decreases significantly, while the transverse section of the muscle cells remains relatively constant. These results show that the reduction of the thickness of the wall of the LV is a result of the loss of cardiomyocytes, as well as the formation of fibrous cicatrices.¹⁶ Olivetti et al. also report the loss of cardiac muscle cells in the LV of rats as aging progresses.¹⁹

The growth of the RV during the early postnatal period is a result of the process of eccentric hypertrophy, as opposed to the growth of the LV, which happens due to both eccentric and concentric hypertrophy.²⁰ According to our data, the difference in the rate of postnatal growth between the two ventricles in rats leads to a higher increase in the number of cardiomyocytes in the LV. In spite of this, the cardiomyocytes from both ventricles show an almost identical mean value of cellular hypertrophy. The results of the studies of Anversa et al. show that as aging progresses, a significant loss of muscle fibres is observed in both ventricles.²¹ The comparative analysis of the cardiomyocytic density in the present study also confirms the presence of age-related loss of cardiac muscle cells. This phenomenon is almost identically manifested in both ventricles but persists longer on the left. Over the period 4-12 month, a 19% decrease in the total number of cardiomyocytes in both ventricles was observed.²¹ In the later age intervals, a similar decrease was observed in the LV, while in the RV, the initial loss underwent full reverse progress up to the twelfth month. The studies of Anversa et al. support the hypothesis that a similar phenomenon is observed in human hearts.²² It is well known that as myocytic proliferation ceases during the process of postnatal development, both the physiological and the induced growth of the myocardium are a result mainly of hypertrophy of the cardiac muscle cells.³ This fact was confirmed by the results of our morphometric analysis of the cardiomyocytic density.

As aging progresses, the total number of cardiomyocytes decreases. The maximum volume that cardiomyocytes can reach is $90,000 \mu\text{m}^3$ and the maximum surface area of the transverse section is approximately $600-900 \mu\text{m}^2$.²³ Cardiomyocytic hypertrophy is, in itself, a physiological process, which constitutes a response to increased demand on the heart. This is why age-related hypertrophy can be considered as developing through a simi-

lar mechanism. Unlike physiological hypertrophy, age-related hypertrophy is a result of a loss of cardiomyocytes due to aging and the decrease of their stability, which increases the demand on the remaining healthy cells.²⁴ Although the development of hypertrophy takes place as aging advances, it is important to note that the ability to increase the cellular surface area is lower in aged cardiomyocytes.²³ For this reason, the ratio between aged and young cardiomyocytes, together with the total number of cardiomyocytes can be used as a prognostic marker of the ability of the myocardium to preserve or worsen its function.²⁴ In support of this, our previous studies²⁵⁻²⁸ have shown that aging of the myocardium is characterised by an increase in the amount of collagen, a decrease in capillary density and changes in the activity of enzymes of the NOS group, which may explain the onset of dysfunction in the aging myocardium. These data and the results of the present study, in line with evidence from the relevant literature lead to the conclusion that as aging advances, the morphology and the structure of the heart become impaired both at the level of the organ and at the level of the individual cardiomyocyte.²⁴

CONCLUSIONS

The conducted study and the obtained quantitative data which were traced as they changed dynamically over the different age periods, reveal a detailed and elaborate assessment of the changes in the myocardium and the cardiomyocytes in the LV and RV, which result from and represent a reflection of the age-related hypertrophy. The thickness of the free wall and the transverse section of cardiomyocytes in both ventricles increased as aging progressed – changes, which reflected the process of hypertrophy and were more dynamic and pronounced in the LV than in the RV. In line with this, the values of the cardiomyocytic density decreased with aging, the LV showing a more dynamic pattern of changes, which indicated the loss of cardiac muscle cells in the later periods of postnatal development.

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Сравнительный количественный анализ постнатальных изменений в миокарде левого и правого желудочков у крыс

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Введение: Рост сердца во время эмбрионального и раннего постнатального развития происходит в основном из-за гиперплазии. Позднее постнатальное развитие характеризуется кардиомиоцитарной гипертрофией в ответ на нормальные физиологические механизмы и повышенную нагрузку. Чтобы изучить размер клеток, большинство авторов измеряют диаметр прямо или косвенно.

Цель: Целью настоящего исследования являлось проведение сравнительного количественного анализа постнатальных изменений, наблюдаемых в левом и правом желудочках у крыс, путём оценки изменений трёх морфометрических параметров - толщины свободной стенки, поперечного сечения кардиомиоцитов и плотности кардиомиоцитов в LV и RV.

Материалы и методы: В настоящем исследовании мы использовали гистологический материал из сердец 15 самцов крыс Вистар, распределённых в пять групп в возрасте 2 недель, 1 месяца, 3 месяцев, 6 месяцев и 12 месяцев.

Результаты: В обоих желудочках толщина стенки и поперечный разрез кардиомиоцитов увеличивались с возрастом, а плотность кардиомиоцитов уменьшалась. Изменения были одинаковыми в обоих желудочках; однако они были более выраженными и динамичными в левом желудочке.

Заключение: Изученные морфометрические параметры показывают, что связанная с возрастом гипертрофия и постепенная потеря сердечных мышечных клеток проявляется в обоих желудочках, но имеют более динамичную картину прогрессирования в левом желудочке в отличие от правого желудочка.