Mazur E. S.[1], Mazur V. V.[1], Rabinovich R. M.[2], Myasnikov K. S.[2]

[1] Tver State Medical University, Tver, Russia

[2] Regional Clinical Hospital of the Tver Region, Tver, Russia

Right Ventricular Longitudinal Strain in Acute Pulmonary Embolism and Right Ventricular Myocardial Infarction in Patients With McConnell's Sign

Aim	To study the right ventricular (RV) myocardial longitudinal systolic strain in patient with RV myocardial infarction (MI), and pulmonary embolism (PE) with and without McConnell' phenomenon.
Material and methods	This study included 53 patients with PE (mean age, 59.0±15.1 years; men, 58.5%) and 30 patients with RVMI (mean age, 61.8±10.9 years; men, 90%). Longitudinal strain of basal, medial and apical segments of the RV free wall (RVFW) and the interventricular septum (IVS) was determined in the mode of two-dimensional speckle tracking. Ratio of the IVS apical strain to the RVFW strain (apical ratio) was calculated. Systolic excursion of the RVFW apical segment (apical excursion) was measured in the anatomical M-mode from the apical four-chamber view.
Results	The McConnell's sign was observed in 23 (43.4%) of 53 patients with PE and in 16 (53.3%) of 30 patients with RVMI ($p>0.05$). Irrespective of the cause for the RV damage, patients with the McConnell's sign had higher values of the apical ratio (1.69±0.50 vs. 0.95±0.22; $p<0.001$; cutoff point, 1.18) and apical excursion (7.9±1.7 vs. 2.6±1.4 mm; $p<0.001$; cutoff point, 5.0 mm). Apical excursion closely correlated with the value of apical ratio ($r=0.65$; $p<0.001$) but not with the RVFW apical segment strain ($r= –0.07$; $p>0.05$).
Conclusion	Incidence of the McConnell's sign was similar in patients with PE and RVMI. McConnell's sign is based on a passive systolic shift of the RVFW apical segment, which develops during contraction of the IVS apical segment. The greater the ratio of IVS apical segment to RBFW global strain the greater the amplitude of this shift. With the ratio value of 1.18 or more, the systolic shift of RVFW apical segment was >5 mm, which was visually perceived as the McConnell's sign.

Keywords	Right ventricular myocardial strain; right ventricular myocardial infarction; pulmonary embolism; McConnell's sign
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Corresponding author Mazur E. S. E-mail: mazur-tver@mail.ru

In 1996, McConnell et al. [1] described an echocardiographic observation that was found to be highly specific for pulmonary embolism (PE). This phenomenon, called McConnell's sign, is a combination of medial hypokinesia of the right ventricular free wall (RVFW) and normal apical contraction (Figure1). Patients with right ventricular (RV) dysfunction and without McConnell's sign had hypokinesia of the entire RVFW, including the apical segment (Figure 2).

The high specificity of McConnell's sign for acute RV overload was confirmed by several trials [2–4] and acknowledged by experts of the European Society of Cardiology [5]. However, Casazza et al. [6] showed in 2005 that McConnell's sign is detected equally often in patients with PE (70%) and in patients with RV myocardial infarction (RVMI) (79%). Several observations of McConnell's sign in non-PE related RV dysfunction appear in the literature, particularly in inverted stress-induced cardiomyopathy [7, 8].

There is much evidence that McConnell's sign is a non-specific phenomenon with a mechanism yet to be clarified. Notably, little research has been undertaken on the relationship of McConnell's sign with the specifics of the RV strain in patients with PE and RVMI. Thus, the objective of this study was to assess RV longitudinal systolic strain in patients with RVMI and PE, with and without McConnell's sign.

Material and methods

The study included patients hospitalized in the Tver Region Clinical Hospital during 2017–2019. PE was present in 53 patients at high (n=15) or intermediate (n=38) risk and RVMI in 30 patients. All patients signed an informed consent to participate in the study. The

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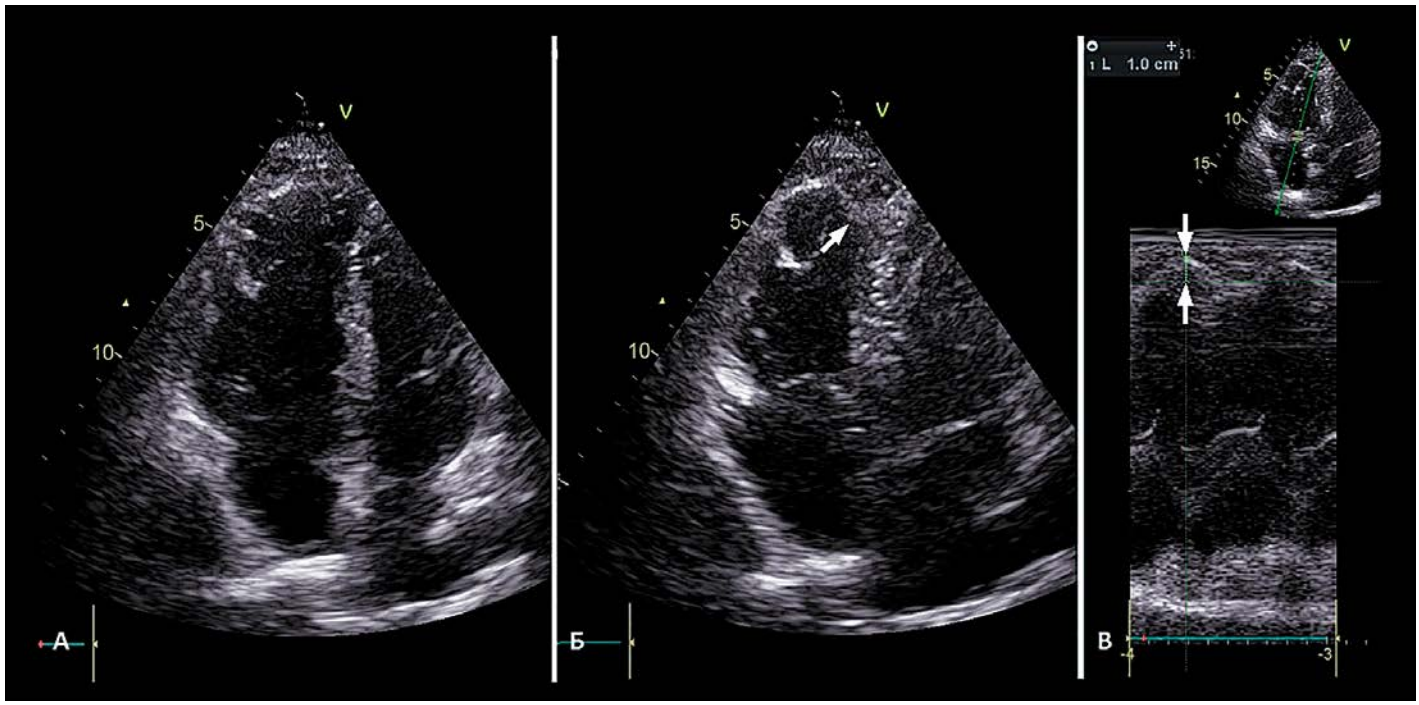
Figure 1. Presence of McConnell's sign in a patient with pulmonary embolism



A – diastole; B – systole: the arrow shows the normal movement of the apical right ventricular free wall; B – the apical systolic excursion in the anatomical M-mode is 1.0 cm.

study was cross-sectional and observational, and it was approved by the ethics committee of the Tver State Medical University.

The study included only patients in whom RV strain was assessed within the first 24 hr after the diagnosis of PE or after RVMI had been verified. The diagnosis of PE was verified by a multislice contrast-enhanced computed tomography scan of the pulmonary artery (PA). Right coronary artery (RCA) occlusion, RV dysfunction, and ST-segment elevation in the V3R and V4R electrocardiographic leads were the criteria for diagnosis of RVMI. The study did not include patients with a history of myocardial infarction (MI) or coronary artery intervention, or patients with PE who had no signs of RV overload.

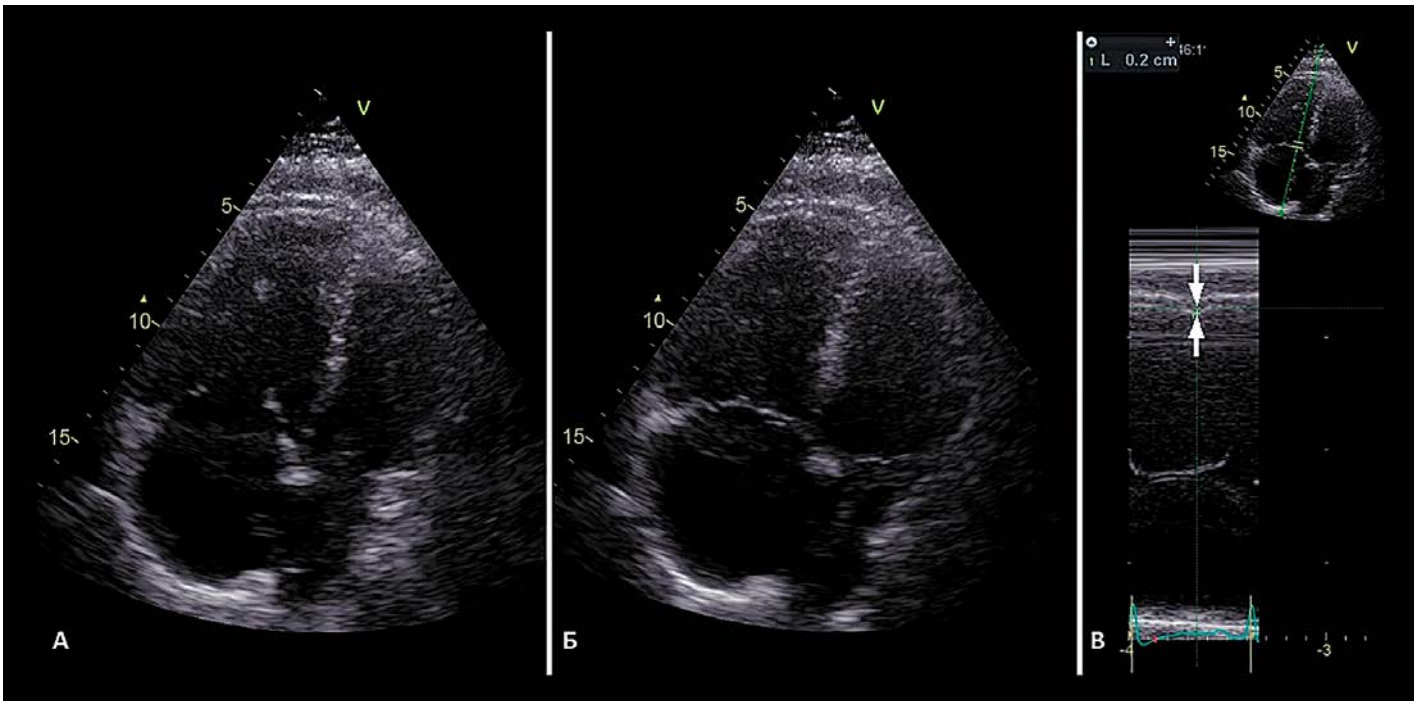
The age of patients with RVMI or PE was similar, 61.8±10.9 and 59.0±15.1 yr, respectively. Male patients prevailed in both groups, 90% and 58.5%, respectively. Concomitant hypertension was reported in 63.3% and 50.9%, diabetes mellitus in 16.7% and 15.1%, and risk factors for PE in 13.3% and 64.2% of RVMI and PE patients, respectively.

Patients with RVMI underwent echocardiography (Vivid S70, GE) within 24 hr after percutaneous coronary intervention, patients with PE of high risk within 24 hr after the stabilization of hemodynamics, and patients with PE of intermediate risk on the same day that computed angiopulmonography was performed. PA systolic pressure (PASP) was calculated from the tricuspid pressure gradient. The systolic apical excursion (AE) of the RVFW was measured in the apical four-chamber view and anatomical M-mode [9] (see Figures 1 and 2). The number of left ventricular (LV) segments with impaired contractility was counted in patients with RVMI. The structures of interest were visualized, and the ultrasound parameters were calculated according to American and European guidelines [10, 11].

RV longitudinal systolic strain was defined in 2D speckle tracking mode and RV-focused four-chamber view [12]. The basal, medial, and apical strain of RVFW and interventricular septum (IVS) was estimated. The absolute values of strain were used according to available guidelines [10, 13]. The RV global strain was calculated as the mean strain of all six RV segments, and the RVFW and IVS strain was calculated as the mean strain of the three corresponding segments. The apical ratio (AR) was calculated as the quotient of the IVS apical strain divided by the RVFW strain. The LV longitudinal systolic strain was measured in patients with RVMI in three apical views, two-chamber, four-chamber, and the LV outflow tract views. The LV global systolic strain was calculated as the mean strain of all 16 segments.

Statistical analysis was performed with the SPSS Statistics v. 15.0 software suite. Mean values and standard deviations (M±SD) were calculated. Student's t-test, Mann-Whitney-U test, or Kruskal Wallis H test was used to estimate the statistical significance of inter-group differences, depending on the number of groups

Figure 2. Absence of McConnell's sign in a patient with pulmonary embolism



A – diastole; B – systole: hypokinesia of the apical right ventricular free wall;
B – the apical systolic excursion in the anatomical M-mode is 0.2 cm.

compared and on the nature of the distribution. Mean values of strain at different levels of RVFW or IVS were compared using the Friedman test. The chi-square test or Fisher's exact test were used to analyze ratios. Correlations of quantitative variables were identified by paired and multiple correlation analyses. The cut-off points for patients with and without McConnell's sign were determined using receiver operating characteristic (ROC) analysis. Results were considered significant at $p<0.05$.

Results

McConnell's sign was identified in 23 (43.4%) patients with PE and 16 (53.3%) patients with RVMI ($p >0.05$). Patients with PE and McConnell's sign more often experienced shock at the start of the disease, and they had lower RV global strain than those in the RVMI group (Table 1). Differences between the mean values of RV global strain were due to an exceptionally lower value of the RVFW strain, given that the mean values of the IVS strain did not differ between the groups.

RVFW strain decreased significantly from basal to apical segments in both groups. The IVS apical strain was lower than the basal and medial strains in patients without McConnell's sign, and it was higher than the baseline strain in patients with McConnell's sign. Due to those differences, the mean value of AR in patients with McConnell's sign was 1.7 times greater than that in patients without this sign. Differences in AE were even more pronounced. The mean AE value was three times higher in patients with McConnell's sign than in the comparison group. According to the ROC-analysis, the cut-off points for PE patients with and without McConnell's sign were AR and AE values of 1.18 and 6 mm, respectively.

Correlation analysis showed a close relationship between AE and AR ($r=0.72$; $p<0.001$), but no relationship between AE and RVFW apical strain ($r= -0.16$; $p>0.05$). This means that the amplitude of the systolic RVFW apical excursion depends on the contractility of the surrounding heart muscle rather than on its own contractility. In other words, the systolic RVFW apical excursion is a passive movement caused by the contraction of the RVFW apical segment. The larger is the IVS apical strain and the smaller is the RVFW total strain, the greater will be the amplitude of this displacement. Correlation analysis showed also a close relationship between RV global strain and RVFW strain ($r=0.90$; $p<0.001$) and between RV global strain and IVS strain ($r=0.78$; $p <0.001$). There was a less close but significant relationship between RVFW strain and IVS strain ($r_{xy}=0.44$; $p<0.001$). According to multiple correlation analysis, the relationship of RVFW strain and IVS strain is mediated by their individual relationships with global strain. The partial correlation coefficient showed that a significant and very strong, but negative relationship, existed between RVFW strain and IVS strain ($r_{xy(z)}= -0.96$; $p<0.001$). In terms of physiology, this

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Table 1. Results of examination of patients with PE with and without McConnell's sign

Parameter	McConnell's sign		p
	negative (n=30)	positive (n=23)	
High-risk PE, abs. (%)	4 (13.3)	11 (47.8)	<0.05
PASP, mm Hg	58.2±16.8	64.5±18.2	>0.05
RV strain, %	15.7±3.6	13.7±2.8	<0.05
RVFW strain, %			
• general	14.7±4.9	11.2±3.5	<0.005
• basal	16.8±6.4	13.5±5.0	<0.05
• medial	15.4±5.4	11.1±4.0*	<0.002
• apical	12.9±4.8*, **	9.7±4.5*, **	<0.02
IVS strain, %			
• general	16.8±3.2	16.3±2.9	>0.05
• basal	18.2±3.8	15.0±4.0	<0.005
• medial	18.5±4.0	16.9±3.6*	>0.05
• apical	13.6±4.2*,**	16.5±3.6*	<0.01
Apical ratio	0.95±0.19	1.57±0.46	<0.001
Apical excursion, mm	2.57±1.38	7.96±1.58	<0.001

Data are M±SD or absolute and relative values (abs. (%)).
p <0.05: * vs basal strain; **, vs medial strain. PE, pulmonary embolism; PASP, pulmonary artery systolic pressure; RV, right ventricle; RVFW, right ventricular free wall; IVS, interventricular septum.

Table 2. Results of examination of patients with RVMI with and without McConnell's sign

Parameter	McConnell's sign		p
	negative (n=14)	positive (n=16)	
Global RV strain, %	12.9±2.8	12.7±2.7	>0.05
RVFW strain, %			
• general	13.9±3.0	11.6±3.6	>0.05
• basal	10.9±4.0	8.4±4.8	>0.05
• medial	14.4±4.1*	10.3±4.4	<0.02
• apical	17.1±4.2*,**	16.6±4.1*,**	>0.05
IVS strain, %			
• general	11.2±3.1	13.1±2.3	>0.05
• basal	9.9±3.1	9.1±3.1	>0.05
• medial	10.9±3.5	11.4±2.8*	>0.05
• apical	13.6±5.1*,**	19.9±4.2*,**	<0.001
Apical ratio	0.97±0.28	1.85±0.54	<0.001
Apical excursion, mm	2.79±1.37	7.81±2.01	<0.001
Left ventricular strain, %	14.3±2.21	14.8±2.92	>0.05
Number of involved segments, n	4.9±1.6	4.8±1.1	>0.05
Posterior apical akinesia, abs. (%)	8 (57.1)	1 (6.3)	<0.005

Data are M±SD or absolute and relative values (abs. (%)).
p <0.05: *, vs basal strain; **, vs medial strain. RVMI, right ventricular myocardial infarction, RVFW, right ventricular free wall, IVS, interventricular septum.

means that the decrease in RVFW strain is accompanied by a compensatory increase in IVS strain.

It should be also noted that while PASP in patients with PE is low, it is significantly correlated with RV global strain (r= –0.33; p<0.02) and with RVFW strain (r= –0.34; p<0.02), but it does not correlate with IVS strain (r= –0.21; p>0.05). Thus, the increase in pressure load causes a decrease in RVFW strain and a compensatory increase in IVS strain, including in its apical segment. AR increases and is accompanied by an increase in AE and the appearance of the visually defined McConnell's sign, which is a marker of severe RV overload in the case of PE.

RVMI patients with or without McConnell's sign did not differ in mean values of global, RVFW, and IVS strains (Table 2). Unlike in patients with PE, in whom RVFW and IVS strains decreased from the basal to the apical segments, a significant increase in RV strain was observed from the basal to the apical level in patients with RVMI. The involvement of the basal and medial segments in RVMI is mainly because they are supplied by the RCA, whereas the apical segments of the RVFW and IVS are supplied by the left anterior descending artery (LAD) and are not stunned in case of the RCA occlusion [7, 8].

It should be noted that the IVS apical strain was significantly higher in patients with RVMI and McConnell's sign than in those without this sign. Thus, the mean value of AR in patients with McConnell's sign was almost twice that of the other group. AE in patients with McConnell's sign was 2.8 times higher than in the other group. According to the ROC-analysis, the cut-off points for RVMI patients with and without McConnell's sign were AR and AE equal to 1.47 and 5 mm, respectively. The amplitude of AE was correlated with AR (r=0.58; p<0.002), but it did not correlate with RVFW apical strain (r= –0.08; p>0.05). Thus, the RVFW systolic apical excursion is a passive movement caused by the contraction of the IVS apical segment in both patients with RVMI and PE.

The size of the involved area and LV global strain in the RVMI patients with and without McConnell's sign did not differ. However, LVPW apical akinesia was almost one-tenth as frequent in patients with McConnell's sign as in the comparison group. This difference deserves special attention since the involvement of the posterior apical segment makes the onset of McConnell's sign virtually impossible. McConnell's sign was actually observed in only 1 (11.1%) of the 9 patients with affected posterior apical LV wall. However, the preserved contractility of the LV posterior apical segment does not necessarily lead to the onset of McConnell's sign in patients with RVMI. Six (28.6%) of twenty-one patients with RVMI and without the involvement of the LV posterior apical segment did not have the sign.

Table 3 shows the comparison of patients with RVMI divided into 3 groups depending on the nature of the LV lesion and the presence of McConnell's sign. Group 1 included nine patients with affected LV posterior apical segments. The two other groups were comprised of patients without the involvement of this segment, 6 subjects without McConnell's sign (Group 2) and 15 subjects with McConnell's sign (Group 3). The most extensive LV lesions and the lowest values of LV global strain were observed in Group 1, along with the lowest values of RV global strain and IVS apical strain. As a result, the group mean of AR was close to 1, and the mean AE was less than 3 mm. As mentioned previously, McConnell's sign was only detected in 1 (11.1%) patient in this group. In should be noted that only in this group, did IVS apical strain not differ from its basal and medial strains. Since the IVS is not supplied by the RCA, the IVS is not affected by RCA occlusion. However, the LV posterior apical segment, which was involved in the MI of Group 1 patients, is adjacent to the IVS apical segment. Thus, LV posterior apical akinesia may limit IVS apical contractility, which explains the pronounced decrease in its strain.

Group 2 patients had the smallest LV lesions and the largest RVFW strains, especially at the basal and medial levels. The IVS apical strain was comparable to the RVFW strain, which is why the AR was close to 1 mm, and the mean AE was slightly more than 3 mm. LV lesions were significantly larger in Group 3 than in Group 2 and close to those in Group 1. From the mean values of the RVFW total strain, it appears that the RV involvement was comparable also in Groups 1 and 3. However, the values of the global strain of both LV and RV in Group 3 were significantly higher than in Group 1, and they did not differ from that of Group 2. In other words, the functional state of the ventricles in Groups 2 and 3 was similar, even though the size of MI was significantly larger in patients of Group 3. The fact that there were no differences in the ventricular functional state in patients with extensive or with non-extensive myocardial involvement is obviously due to hyperfunction of the unaffected segments, namely the IVS apical segment. There the mean strain was significantly higher than in other groups. The increased IVS apical strain caused a sharp increase in the AR and AE values.

Thus, the onset of McConnell's sign in RVMI is associated with extensive myocardial involvement in both ventricles except for the LV posterior apical segment. In this case, a compensatory increase in the contractile activity of the IVS apical segment is possible. It results in an increase in AR and AE, which is visually defined as McConnell's sign. Comparison of the findings in Tables 1 and 2 shows that regardless of the reason for the RV dysfunction, patients with and without McConnell's sign have high and low values of AR and AE, respectively. The mean AR in patients with and without McConnell's sign was 1.69±0.50 and 0.95±0.22, respectively ($p<0.001$), and the mean AE was 7.9±1.7 and 2.6±1.4 mm, respectively ($p<0.001$). AR and AE equal to 1.18 and 5.0 mm, respectively, are the cut-off points for patients with and without McConnell's sign. AE was closely correlated with AR ($r=0.65$; $p<0.001$), but did not correlate with the RVFW apical strain ($r= – 0.07$; $p>0.05$).

Table 3. Findings in patients with RVMI divided into groups depending on the features of the LV lesion and the presence of McConnell's sign

Parameter	Groups of patients with RVMI		
	Group 1 (n=9)	Group 2 (n=6)	Group 3 (n=15)
Number of involved segments, abs.	6.1±0.3	3.2±0.4#	4.7±1.1#,##
Left ventricular strain, %	13.9±1.5	15.3±2.7	14.9±3.1
Global RV strain, %	11.7±3.5	13.7±2.1	13.1±2.3
RVFW strain, %			
• general	12.2±4.6	15.0±1.4	12.1±3.0
• basal	8.8±5.0	12.5±2.7	8.8±4.7
• medial	11.8±5.8*	16.3±2.1	10.9±3.9##
• apical	15.8±5.0*,**	17.5±4.3*	17.2±3.5*,**
IVS strain, %			
• general	10.7±2,9	12.0±3.2	13.3±2.3
• basal	9.9±2.9	9.8±3.4	9.1±3.2
• medial	10.2±3.0	11.8±4.0*	11.5±2.9*
• apical	11.9±4.4	16.0±4.9*,**	20.5±3.8#,##,*,**
Apical ratio	1.12±0.74	1.07±0.31	1.78±0.46#,##
Apical excursion, mm	2.8±1.9	3.3±0.8	7.9±2.0#,##

Data are M±SD. $p<0.05$: #, vs Group 1; ##, vs Group 2;
* vs basal strain; **, vs medial strain. LV, left ventricle;
RVMI, right ventricular myocardial infarction, RV, right ventricle;
RVFW, right ventricular free wall, IVS, interventricular septum.

Discussion

McConnell's sign is a categorical sign, the presence of which is established by subjective perception of the RVFW apical systolic movement. In this study, McConnell's sign was visually defined when the amplitude of the RVFW systolic displacement exceeded 5 mm. This criterion allowed us to objectify the definition of McConnell's sign, but its clinical significance remained to be clarified.

According to the hypothesis put forward by McConnell et al. [1] to explain the mechanism of the phenomenon described by them, the distinct RVFW

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ПОКАЗАНИЯ К ПРИМЕНЕНИЮ:** первичная гиперхолестеринемия по классификации Фредриксона (тип IIa, включая семейную гетерозиготную гиперхолестеринемию) или смешанная гиперхолестеринемия (тип IIb) в качестве дополнения к диете, когда диета и другие немедикаментозные методы лечения оказываются недостаточными; семейная гомозиготная гиперхолестеринемия в качестве дополнения к диете и другой липидснижающей терапии (например, ЛПНП-аферез) или в случаях, когда подобная терапия недостаточно эффективна; гипертриглицеридемия (IV тип по классификации Фредриксона) в качестве дополнения к диете; для замедления прогрессирования атеросклероза в качестве дополнения к диете у пациентов, которым показана терапия для снижения концентрации общего ХС и ХС-ЛПНП; первичная профилактика основных сердечно-сосудистых осложнений (инсульта, инфаркта, нестабильной стенокардии, артериальной реваскуляризации) у взрослых пациентов без клинических признаков ишемической болезни сердца (ИБС), но с повышенным риском ее развития (возраст старше 50 лет у мужчин, старше 60 лет у женщин, повышенная концентрация С-реактивного белка (≥ 2 мг/л) при наличии как минимум одного дополнительного фактора риска, таких как артериальная гипертензия, низкая концентрация ХС-ЛПВП, курение, семейный анамнез раннего начала ИБС). **ПРОТИВОПОКАЗАНИЯ.** Для суточной дозы 5 мг, 10 мг и 20 мг: повышенная чувствительность к розувастатину или любому из компонентов препарата; заболевания печени в активной фазе, включая стойкое повышение активности «печеночных» трансаминаз, а также любое повышение активности «печеночных» трансаминаз в сыворотке крови более чем в 3 раза по сравнению с верхней границей нормы (ВГН); тяжелые нарушения функции почек (КК менее 30 мл/мин); миопатия; одновременный прием циклоспорина; беременность, период грудного вскармливания; применение у пациентов, предрасположенных к развитию миотоксических осложнений; дефицит лактазы, непереносимость лактозы, синдром глюкозо-галактозной мальабсорбции (препарат содержит лактозу); возраст до 18 лет (эффективность и безопасность не установлены). Для суточной дозы 40 мг: повышенная чувствительность к розувастатину или любому из компонентов препарата; заболевания печени в активной фазе, включая стойкое повышение активности «печеночных» трансаминаз, а также любое повышение активности «печеночных» трансаминаз в сыворотке крови более чем в 3 раза по сравнению с верхней границей нормы (ВГН); наличие факторов риска развития миопатии/рабдомиолиза: почечная недостаточность умеренной степени тяжести (КК < 60 мл/мин), гипотиреоз, миопатии в анамнезе, включая наследственные; миотоксичность на фоне приема других ингибиторов ГМГ-КоА-редуктазы или фибратов в анамнезе, чрезмерное употребление алкоголя; состояния, которые могут приводить к повышению плазменной концентрации розувастатина, одновременный прием фибратов, применение у пациентов монголоидной расы; одновременный прием циклоспорина; беременность, период грудного вскармливания; применение у пациентов, предрасположенных к развитию миотоксических осложнений; дефицит лактазы, непереносимость лактозы, синдром глюкозо-галактозной мальабсорбции (препарат содержит лактозу); возраст до 18 лет (эффективность и безопасность не установлены). **СПОСОБ ПРИМЕНЕНИЯ И ДОЗЫ.** Внутрь. В любое время суток, независимо от приема пищи. Таблетку не разжевывать, не измельчать, проглатывать целиком, запивая водой. До начала терапии препаратом Сувардио® пациент должен начать соблюдать стандартную гипохолестеринемическую диету и продолжать соблюдать ее в течение всего периода терапии. Дозу препарата Сувардио® подбирают индивидуально с учетом целевых показателей концентрации холестерина и индивидуального терапевтического ответа на проводимую терапию. Рекомендуемая начальная доза препарата Сувардио® составляет 5 мг или 10 мг 1 раз в сутки как для пациентов, ранее не принимавших статины, так и для пациентов, переведенных на прием данного препарата после терапии другими ингибиторами ГМГ-КоА-редуктазы. При выборе начальной дозы следует руководствоваться концентрацией холестерина и возможным риском развития сердечно-сосудистых осложнений у данного пациента, а также следует оценить потенциальный риск развития побочных эффектов. При необходимости через 4 недели можно скорректировать дозу препарата. В связи с возможным развитием побочных эффектов при приеме дозы 40 мг по сравнению с более низкими дозами препарата окончательное титрование до максимальной дозы 40 мг следует проводить только у пациентов с тяжелой формой гиперхолестеринемии и высоким риском возникновения сердечно-сосудистых осложнений (особенно у пациентов с наследственной гиперхолестеринемией), у которых при приеме дозы 20 мг не была достигнута целевая концентрация холестерина и которые будут находиться под врачебным наблюдением. При назначении дозы 40 мг рекомендовано тщательное наблюдение врача. Не рекомендуется назначение дозы 40 мг пациентам, ранее не обращавшимся к врачу. **ПОБОЧНОЕ ДЕЙСТВИЕ.** Со стороны нервной системы — часто: головная боль, головокружение; нарушения со стороны эндокринной системы — часто: сахарный диабет 2-го типа; со стороны пищеварительной системы — часто: запор, тошнота, боль в области живота; лабораторные показатели: повышение активности креатинфосфокиназы (КФК), концентрации глюкозы, гликозилированного гемоглобина, билирубина в плазме крови, активности гамма-глютамилтранспептидазы, щелочной фосфатазы, нарушение функции щитовидной железы; прочие - часто: астенический синдром, гинекомастия, периферические отеки; нарушения со стороны мочевыделительной системы — при приеме розувастатина может наблюдаться протеинурия. Изменения содержания белка в моче (от отсутствия до наличия следовых количеств до уровня ++ и выше) наблюдаются менее чем у 1% пациентов, принимающих розувастатин в дозе 10 мг и 20 мг, и примерно у 3%, принимающих препарат в дозе 40 мг. Нарушения со стороны опорно-двигательного аппарата и соединительной ткани — часто: миалгия. **ОСОБЫЕ УКАЗАНИЯ.** Через 2—4 недели после начала лечения и/или при повышении дозы препарата необходим контроль показателей липидного обмена (при необходимости требуется коррекция дозы). Розувастатин, как и другие ингибиторы ГМГ-КоА-редуктазы, следует с особой осторожностью назначать пациентам с имеющимися факторами риска миопатии/рабдомиолиза. Рекомендуется проинформировать пациентов о необходимости незамедлительно сообщать врачу о случаях неожиданного появления мышечных болей, мышечной слабости или спазмах, особенно в сочетании с недомоганием или лихорадкой. Определение показателей функции печени рекомендуется проводить до и через 3 месяца после начала лечения. Возможны взаимодействия с другими лекарственными препаратами (см. соответствующий раздел инструкции). **ВЛИЯНИЕ НА СПОСОБНОСТЬ УПРАВЛЯТЬ ТРАНСПОРТНЫМИ СРЕДСТВАМИ, МЕХАНИЗМАМИ:** необходимо соблюдать осторожность при управлении автотранспортными средствами, занятиях потенциально опасными видами деятельности, требующими повышенной концентрации внимания и быстроты психомоторных реакций (риск развития головокружения).

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4. Согласно данным базы ООО «АЙКЬЮВИА Солюшнс» «Розничный аудит ГЛС и БАД в РФ» средняя розничная цена на национальном уровне в сентябре 2019 г. для лекарственного препарата Сувардио® таблетки, покрытые пленочной оболочкой 10 мг № 28 ЗАО «Сандоз» составляет 481,24 руб., для лекарственного препарата Сувардио® таблетки, покрытые пленочной оболочкой 10 мг № 90 ЗАО «Сандоз» составляет 970,48 руб., для лекарственного препарата Сувардио® таблетки, покрытые пленочной оболочкой 20 мг № 28 ЗАО «Сандоз» составляет 604,20 руб., для лекарственного препарата Роксера® таблетки, покрытые пленочной оболочкой 10 мг № 30 ООО «КРКА-РУС» составляет 596,32 руб., для лекарственного препарата Роксера® таблетки, покрытые пленочной оболочкой 10 мг № 90 ООО «КРКА-РУС» составляет 1296,60 руб., для лекарственного препарата Роксера® таблетки, покрытые пленочной оболочкой 20 мг № 30 ООО «КРКА-РУС» составляет 884,71 руб.

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GO FOR GOAL cholesterol control for life = ДВИЖЕНИЕ К ЦЕЛИ. Контроль холестерина в течение жизни.
Реклама.

apical systolic movement is associated with the passive displacement toward the contracted LV rather than the active contraction of this segment. The present study showed that the RVFW apical systolic displacement is caused by contraction of the IVS apical segment, which is a part of the LV, both structurally and functionally. The higher is the value of AR, i.e., the ratio of IVS apical strain to STR strain, the greater is the amplitude of the RVFW apical systolic displacement. If AR is 1.18 or more and the amplitude of the RVFW apical systolic displacement exceeds 5 mm, this is visually defined as McConnell's sign.

The decrease in the RVFW strain in patients with PE is associated with an increase in PASP, i.e., the severity of RV overload. The increase in the IVS strain, including its apical segment, is a compensatory reaction supporting the general systolic function of the RV. Thus, high values of AR and associated McConnell's sign are indicative of severe RV overload, which is evidenced by the fact that McConnell's sign was identified in 31.6% of the subjects with PE of intermediate-risk and 73.3% patients of high risk ($p<0.05$). Pruszczyk et al. [14] reported similar results. They identified this phenomenon in 18% of the general group of subjects with PE and in 43% of patients in a high-risk subgroup. Kurnicka et al. [15] identified McConnell's sign in 19.8% of patients with PE and in 75% of high-risk patients. McConnell's sign may have predictive value in patients with PE, but this is a subject that requires further study.

The onset of McConnell's sign in RVMI depends on the size and location of the infarction, which, in turn, depends on the coronary anatomy and the level of the RCA obstruction. We established that involvement of the LV posterior apical segment makes McConnell's sign almost impossible, since akinesia of this segment prevents the compensatory increase in the contractile activity of the adjacent IVS apical segment, hyperkinesia of which results in a pronounced systolic displacement of the RVFW apical segment.

If the RCA supplies the posterior apical segment, in case of RCA occlusion, this segment will be involved in the infarction. This variant of blood supply is observed in about 20% of cases, whereas the LAD supplies the posterior apical segment in 80% of cases [10]. Akinesia of the LV posterior apical segment was observed in 9 (30%) of the 30 patients with RVMI. This is approximately the same proportion as having RCA blood supply to this segment. In the intact LV posterior apical segment, McConnell's sign appears if the contractile activity of the IVS apical segment must be compensated, i.e., in extensive LV and RV myocardial damage associated with obstruction of the proximal RCA [16–18]. Thus, McConnell's sign in RVMI shows extensive myocardial damage that does not involve the LV posterior apical segment.

Conclusion

McConnell's sign was defined in 50% of patients with PE and RV overload and in 50% of patients with right ventricular myocardial infarction. Thus, it impossible to use McConnell's sign for the differential diagnosis of these conditions. McConnell's sign appears due to the passive systolic displacement of the apical right ventricular free wall, which occurs when the apical interventricular septum contracts. The higher is the ratio of the apical ventricular septum to the right ventricular global strain, the greater is the amplitude of this displacement. When the ratio is 1.18 or more, the systolic apical strain of the right ventricular free wall exceeds 5 mm, which is visually defined as McConnell's sign.

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