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Spectrum of Phenomena of Multiple Myocardial Contractions during Each Single Cardiac Cycle

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A B S T R A C T

The phenomenon of multiple myocardial contractions during each single cardiac cycle, triggered by a single sinus node impulse, is the most intriguing echocardiographical topic among detectible modalities of irregular kinetics of interventricular septum. Tissue Doppler imaging based methods gave proofs of active contractions in cases presented herein. Broad spectrum of this phenomenon, which can be found both in healthy subjects and in patients is presented. Forms of multiple myocardial contractions during the same cardiac cycle presented in this analysis are: secondary systolic contraction, postsystolic and late postsystolic contraction, multiple (three- and fourfold) contractions with combination of secondary systolic and postsystolic contraction, and combination of regular systolic, secondary systolic or postsystolic contraction with pre-contraction as a part of possible preexcitation. Explanation of mechanism generating these phenomena could be found in possible existence of accessory, concealed slow pathways of cardiac conduction system.

Key words: *systole, diastole, postsystolic contraction, myocardium, tissue Doppler imaging*

Introduction

A biomedical phenomenon of multiple myocardial contractions during each single cardiac cycle, which occurs in a considerable segment of population, has been identified thanks to echocardiographical methods, among which tissue Doppler imaging leads[1]. Ultrasound analysis of cardiac performance precisely detects periods and phases of cardiac cycle: ventricular systole and diastole, with isovolumic contraction time and ejection, and isovolumic relaxation time and ventricular filling. Normal myocardial contraction is presented by ultrasound as a thickening of myocardial walls with their synchronous movement toward the centre of left ventricle cavity. Routinely used echocardiographical parasternal M-mode displays synchronous movement of interventricular septum (septum) and left ventricular free wall toward each other in such a case (Figure 1a). In fact, normal, physiological contraction is asynchronous in part, because depolarisation of basal septum precedes approximately 70 ms to depolarisation of posterior wall (Figure 1b).

Besides physiological kinetics, different models of irregular septal kinetics exist. Paradoxical septal movement (systolic movement toward the centre of right ventricle) occurs after pericardiotomy or with right ventricular pressure overload. Asynchronous septal contraction occurs with left bundle branch block (a short movement toward the centre of left ventricular cavity, after which occurs a movement predominantly toward the centre of right ventricular cavity). Premature (presystolic) contraction occurs with existence of aberrant conduction pathways. These models of septal kinetics have been well explained so far and are out of interest of this analysis.

An unexplained phenomenon of secondary systolic movements of myocardial walls can often be seen in routine echocardiographical work both in patients as well as in healthy. These secondary systolic movements occur after regular myocardial contraction, during the same cardiac cycle and they are triggered by the same sinus node impulse, as different from premature ectopic beats. Intense discussions are being held over last years about a part of this phenomenon, named postsystolic contraction[2] (or according to established terminology postsystolic movement or thickening or shortening), which is characterized by secondary contraction occurring at the

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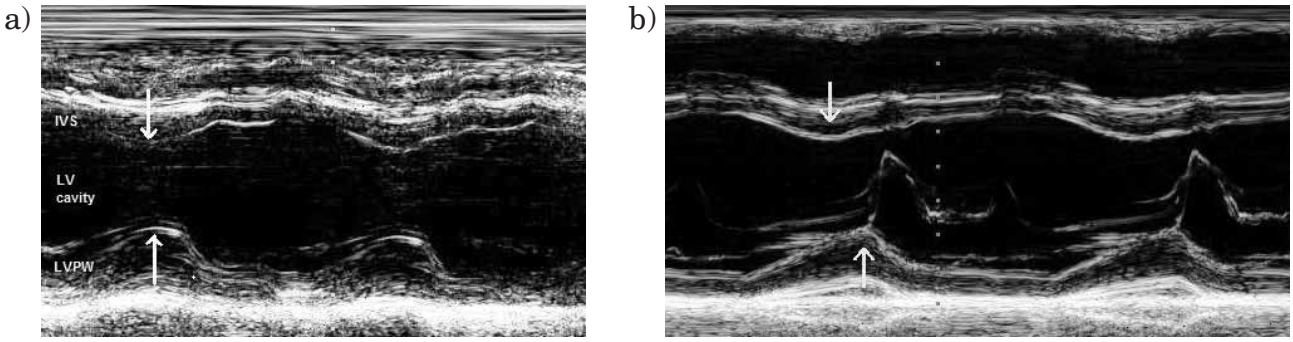

Fig. 1a) and b). Normal contractility of myocardial walls, with synchronous (Figure 1a) or asynchronous (Figure 1b) movements of interventricular septum and left ventricular free wall. Legend: arrows mark systolic movements of walls, IVS – interventricular septum, LV cavity – left ventricular cavity, LVPW – left ventricular posterior wall.

beginning of diastole of the same cardiac cycle, in isovolumic relaxation phase (Figure 2).

This phenomenon has been detected by echocardiography, or more specific by tissue Doppler imaging methods[3], in patients with ischemic heart disease where it has been most thoroughly analyzed[4], but has also been described in healthy population[4]. Some evidences point to postsystolic motion as an active contraction instead of passive movement of myocardial wall caused by hemodynamical changes, as some opinions suggest, but these evidences are still not conclusive enough[5]. Different from postsystolic contraction, a similar secondary systolic movement that occurs in phase of systole has not yet been described by scientific or professional auditorium. Although this phenomenon can be observed in everyday routine work when attention is devoted, it is possible it is not detected due to very short temporal distinction between end-systolic and early postsystolic contraction. Multiple myocardial contractions during the same cardiac cycle also exist, but no scientifically defined opinions or reports have yet been published on such topic.

To elucidate these phenomena we performed a study on modalities of irregular systolic kinetics of interventricular septum with preliminary findings presented herein.

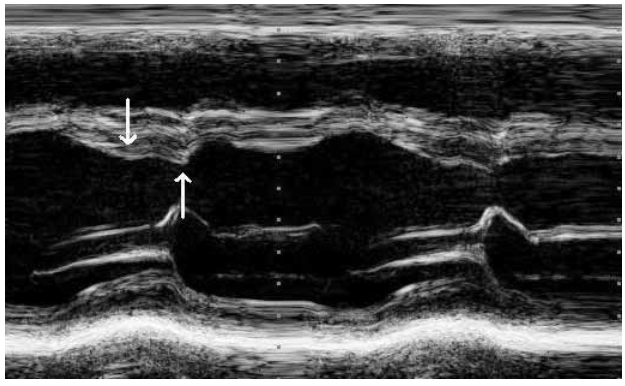

Fig. 2. Postsystolic contraction – a double septal movement during the same cycle, one before end of systole (upper arrow), second in diastole (bottom arrow).

Methods

Sample

Our ongoing study 'Modalities of irregular systolic kinetics of interventricular septum' is oriented not only on problem of multiple myocardial contractions but also on other forms of asynchronous or paradoxical movements of septum already mentioned in Introduction. Apart from echocardiographical measures presented here, it also analyzes hemodynamical and electrophysiological variables. We used a narrower spectrum of analyses for a segment of the study shown in this paper because it describes only cases of healthy examinees and patients with no hemodynamical disturbances which could influence myocardial contractility.

In this paper we describe representative cases of mentioned phenomenon who had presence of multiple contractions during each single cardiac cycle detected at a routine echocardiographical examination. We included 7 examinees, 4 females and 3 males, aged from 22 to 56 years (mean 38.2 years). One was a healthy volunteer, while others were referred to an echocardiographical examination due to various clinical conditions: arterial hypertension (two), arterial hypertension and left bundle branch block (one), possible ischemic heart disease (one), heart murmur (one) and palpitations (one).

An informed consent on the study protocol was obtained from each examinee. The study protocol has been approved by local ethics committee and all procedures were conducted in accordance with the revised declaration of Helsinki (Hong Kong, 1989).

Echocardiographical examination and measurements

Echocardiographical examinations were performed in a standardized manner, with patients in left supine position. ECG tracing was obtained continuously in one lead. All echocardiographical examinations were performed by the same investigator, using Philips iE33 ultrasound machine with a 5–1 MHz transducer.

After the routine examination, intervals of opening and closing of the valves during cardiac cycle (used for

defining timing of cardiac cycle phases) were measured. Timings of occurrence and peak of additional contractions were estimated after that. Finally, recording of data for offline strain and strain rate analysis of exact timings of occurrence and peak of contractions was performed. Only patients in sinus rhythm with no arrhythmias during the examination were included in the study.

M-mode grey scale and colour M-mode images, colour tissue Doppler M-mode and pulse wave Doppler images of the apical and parasternal long and short axis views were acquired for defining timings of aortic valve opening and closure, and mitral valve opening and closure and for defining cardiac cycle phases. Mechanical systole was defined as the time from aortic valve opening to aortic valve closure, while electromechanical systole was defined as the time from beginning of QRS signal to aortic valve closure. Measurements of intervals were conducted on 15 cardiac cycles due to possible respiratory sinus arrhythmia, so that we could use measured intervals gained in cardiac cycle with the closest RR interval for subsequent offline tissue Doppler analysis.

Timings of occurrence and peak of additional contractions, compared with cardiac cycle intervals, were roughly measured in the first phase of analysis by colour tissue Doppler M-mode in parasternal short axis views at the basal level of left ventricle, from 3 consecutive cycles recorded during breath hold. By colour tissue Doppler M-mode, movements of walls were analyzed by change in colour where blue depicted movement away from the transducer and red depicted movement toward the transducer. Timings of multiple septal movements were compared with the end of anterior movement of posterior wall (end of systolic contraction).

Five consecutive cardiac cycles were recorded from parasternal short axis view at the basal level of left ventricle during breath hold for the final confirmation of timing of contractions by colour tissue Doppler velocity analysis. During recording care was taken of aligning septum and left ventricular posterior wall perpendicular to ultrasound beam. We used the narrowest possible image sector angle, with frame rate 65+/–14 frames per second and appropriate velocity scale to avoid aliasing. Offline analysis (QLAB software, Philips) defined myocardial velocities profile, strain and strain-rate curves which showed timings of occurrence and peak of each single deformation (contraction). Region of interest was maintained at the same position on septum or posterior wall during cardiac cycle by adjusting its position frame to frame. Default width of region of interest was 2.5 mm and the length was adjusted depending on width of myocardial wall. Strain correction was set to correct each cycle. Strain rate was estimated from tissue Doppler regional velocities profile for each myocardial segment by calculating the velocity gradient. It is defined as a temporal course of tissue deformation and is expressed in s^{-1}. Strain is a measure of tissue deformation and is expressed in %. Strain data are provided by temporal integration of strain rate data. Magnitude of peak strain and strain rate for systolic and additional myocardial contractions, and timing from R-wave (beginning of electromechanical systole) to peak of strain and strain rate for every contraction were measured.

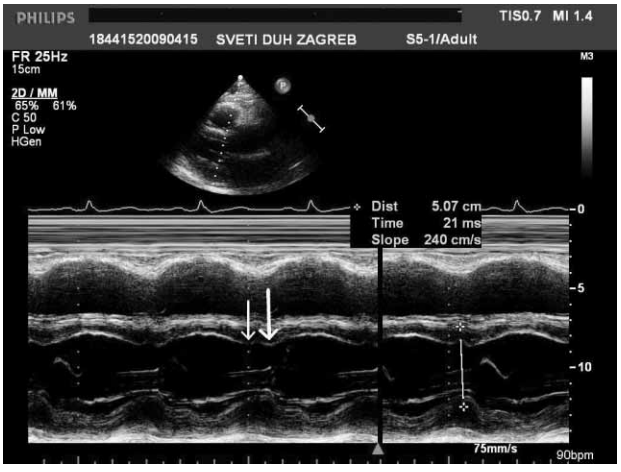

Fig. 3. Double systolic movement of interventricular septum. Legend: thinner arrow – regular systolic movement, thicker arrow – secondary systolic movement. Oblique line connecting crosses shows that the onset of secondary septal movement precedes peak movement of posterior wall for 21 ms.

Results

Case 1 – Double myocardial contraction during the same cardiac cycle – case of a secondary systolic contraction

A 33-year-old male with mild arterial hypertension was referred to a routine echocardiographical examination. Besides borderline left ventricle hypertrophy (13 mm) and a very mild mitral regurgitation examination showed otherwise normal findings.

As a co-finding, two posterior septal movements during each cardiac cycle were observed from parasternal long axis M-mode view (Figure 3) – resembling double septal contraction. This was shown even clearer using colour tissue Doppler in M-mode view (Figure 4). While posterior wall moves only forward (red) and backward (blue) during this cycle, septum moves in posterior direction (blue), then for a short time in anterior (red), and then again in posterior direction (blue marked with an arrow).

Confirmation that the second movement was an active contraction rather than passive movement was obtained by measurement of septal thickness at the time of peaks of first and second movement, and at the time between those movements (Figure 5). Thickness of septal wall was 16.0 mm during peak of first contraction, then it decreased to 14.8 mm, and at peak of secondary contraction it thickened again to 15.4 mm. This finding of repeated myocardial thickening during secondary posterior movement strongly suggested that there was a secondary contraction during the same cardiac cycle.

Analysis of timing of onset of additional septal contraction showed that it preceded the peak of posterior

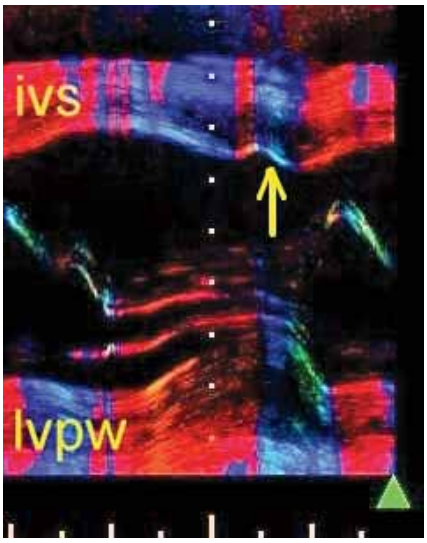

Fig. 4. Double movement of the septum shown using colour tissue Doppler imaging in M-mode view. Legend: ivs – interventricular septum, lvpw – left ventricular posterior wall, arrow – secondary systolic movement.

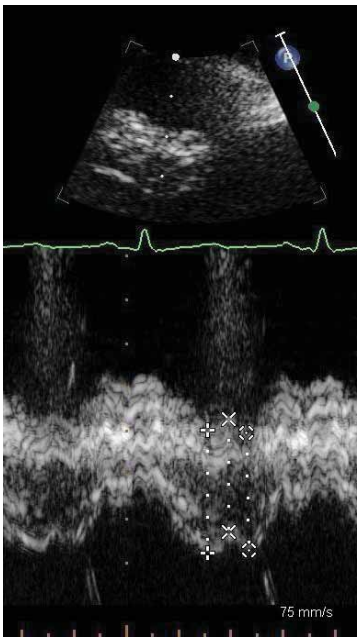

Fig. 5. Secondary myocardial thickening during the same cardiac cycle (septum enlarged). Legend: septal thickness between two points: + 16.0 mm, × 14.8 mm, ※ 15.4 mm.

wall contraction for at least 21 ms (Figure 3, right segment), in other words it occurred during systole. For the definitive confirmation that it was a secondary systolic contraction of septum, and not a postsystolic one, tissue deformation analysis based on tissue Doppler imaging methods was performed.

Strain rate analysis of septal radial deformation showed peak of regular systolic contraction (yellow label 1 in Figure 6a) 173 ms after the onset of electrical systole (R wave) with peak strain rate 1.3 s^{-1}. Secondary contraction began 319 ms after R-wave (yellow label 2 in Figure 6a), with peak at 335 ms after R-wave (yellow label 3 in Figure 6a), with peak strain rate 0.9 s^{-1}. Considering that the duration of the systole was 365 ms, which means peak of secondary contraction preceded to aortic valve closure, it was clear this was the case of secondary systolic (intrasystolic) contraction.

Strain analysis (Figure 6b) showed corresponding results, proving that additional septal contraction occurred during systole. Peak strain of first systolic contraction occurred 245 ms after R-wave (peak strain 14.1 %), while peak strain of second contraction, which began 314 ms after R-wave, occurred 366 ms after R-wave (peak strain 14.2 %). This confirmed that secondary contraction occurred during systole and ended by its end.

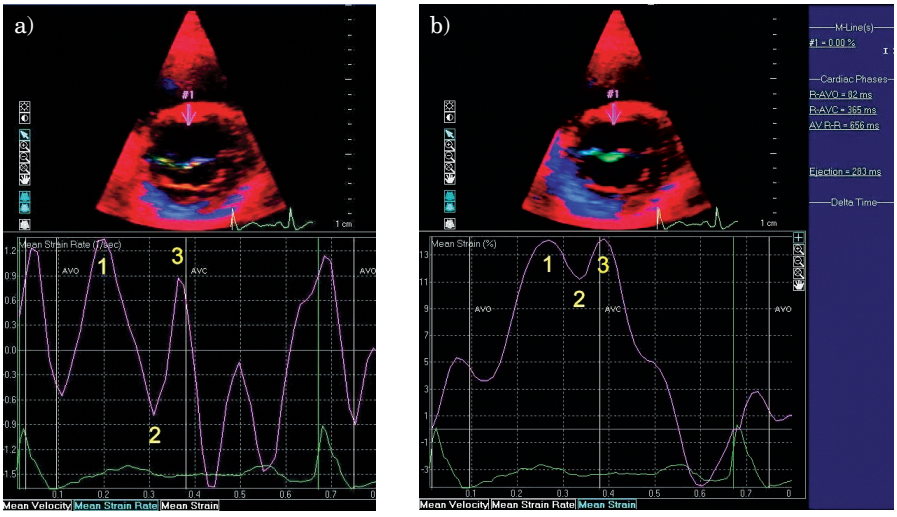

Fig. 6 a) and b). Strain rate (Figure 6a) and strain (Figure 6b) analyses of double systolic contraction (purple lines). Please see text for explanations.

Case 2 – Double myocardial contraction during the same cardiac cycle – case of a postsystolic contraction

A 45-year-old male with probable ischemic heart disease was referred to an echocardiographical examination. His electrocardiogram showed incomplete right bundle branch block, and routine echo examination showed mild aortic root dilatation (46 mm). Long parasternal views of colour tissue Doppler M-mode revealed a double posterior movement of septum (Figure 7). The beginning of the secondary septal movement was synchronous with the peak of anterior movement of posterior wall, thus suggesting that the secondary movement, or quite possibly contraction, was a postsystolic one.

A confirmation that the secondary thickening was a postsystolic contraction, was obtained by strain analysis of septal radial deformation (Figure 8a). Peak strain of regular systolic deformation (contraction) occurred 253 ms after R-wave (peak strain –1.2 %). Secondary contraction began 360 ms after R-wave, that is after aortic valve closure which occurred 300 ms after the beginning of electrical systole. This contraction had peak 430 ms after R-wave (peak strain –7.6 %), late in diastole. This confirmed that it was a postsystolic contraction.

Corresponding results were obtained by strain rate analysis (Figure 8b). Peak systolic strain rate of the first contraction occurred at 202 ms after R-wave (peak strain rate –1.0 s^{-1}), while secondary contraction began 315 ms after R-wave with peak at 379 ms after R-wave (peak strain rate –2.2 s^{-1}). This finding also confirmed that the contraction was postsystolic.

Case 3 – Double myocardial contraction during the same cardiac cycle – case of an asynchronous postsystolic contraction of opposite myocardial walls

A 22-year-old healthy female was referred to an echocardiographical examination due to evaluation of heart murmur. A mild mitral regurgitation was found, with otherwise normal findings. Double posterior movement of septum was displayed from parasternal long axis M-mode view, as well as double anterior movement of left ventricular posterior wall (Figure 9).

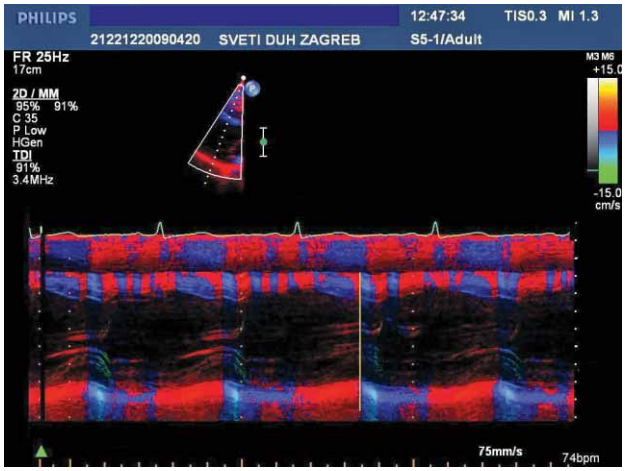

Fig. 7. Secondary postsystolic movement of the interventricular septum. Legend: yellow line indicates simultaneous onset of the secondary septal thickening with the peak thickening of the posterior wall, at the end of systole.

Radial deformation analysis was performed on septum and posterior wall. Duration of electromechanical systole was 395 ms. Septal strain analysis (Figure 10a) showed peak systolic deformation 170 ms after R-wave (peak strain –2.5 %), secondary deformation that began 480 ms after R-wave with peak strain 550 ms after R-wave (peak strain –4.2 %). In other words, secondary contraction happened in whole after aortic valve closure, as a postsystolic contraction.

Posterior wall radial deformation analysis was simultaneously performed (Figure 10b). It showed unusually short interval between two contractions. Peak of regular systolic deformation was noted 300 ms after R-wave

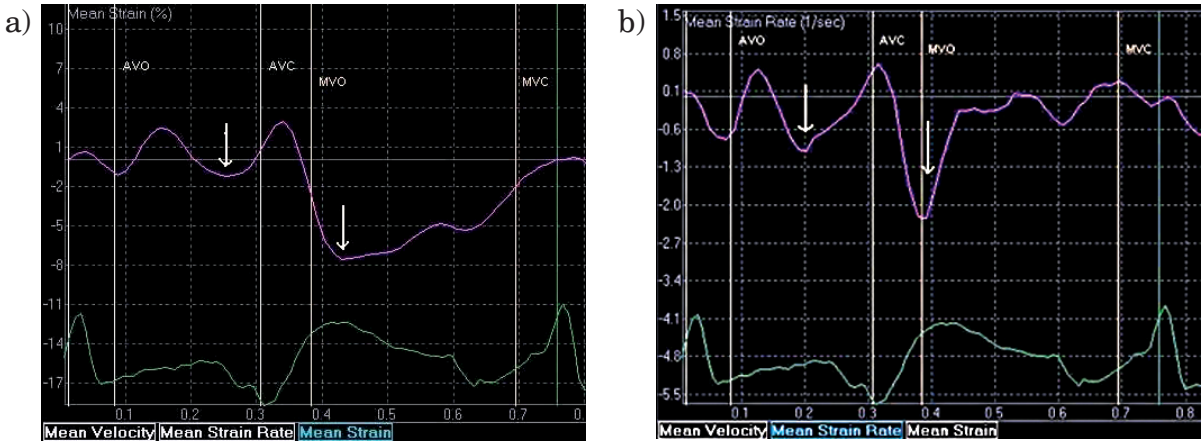

Fig. 8 a) and b). Postsystolic contraction shown by strain (Figure 8a) and strain rate (Figure 8b) analysis. Legend: arrows mark peaks of systolic and postsystolic deformation, AVO – aortic valve opening time, AVC – aortic valve closure time, MVO – mitral valve opening time, MVC – mitral valve closure time.

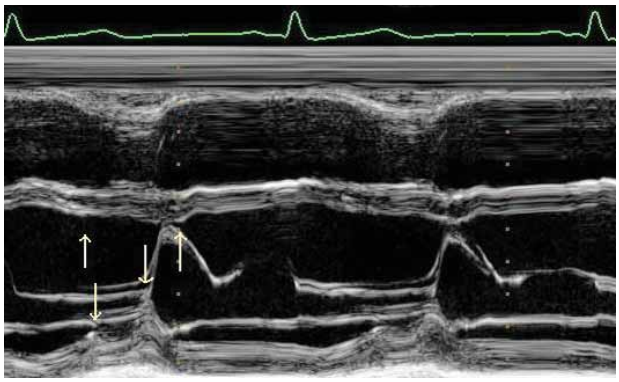

Fig. 9. Double movement of both interventricular septum and left ventricular posterior wall. Legend: Arrows mark posterior movements of septum (arrows directed upward) and anterior movements of posterior wall (arrows directed downward).

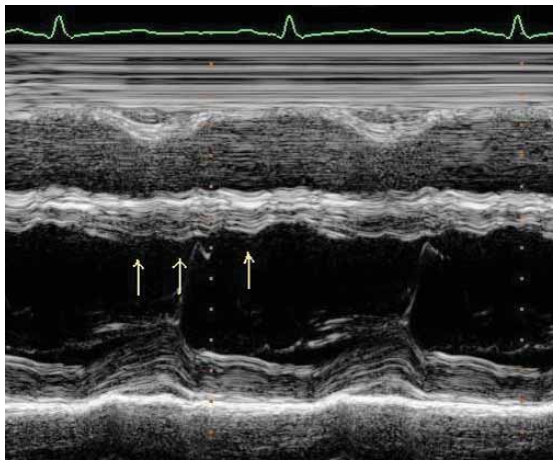

Fig. 11. Multiple movements of interventricular septum during the same cardiac cycle (marked with arrows).

(peak strain 13%), while secondary deformation began 400 ms after R-wave with peak strain that occurred 450 ms after R-wave (peak strain 11%). This contraction began simultaneously with the aortic valve closure, which means it was also postsystolic. However, the onset of secondary postsystolic contraction of the septum was considerably late, thus resulting in double asynchronous contraction of opposite myocardial walls.

Case 4 – Multiple myocardial contractions during the same cardiac cycle – case of a double postsystolic contraction

A 36-year-old male with history of palpitations and incomplete right bundle branch block underwent echocardiographical examination which was completely normal, except for finding of multiple movements of the septum. Two additional movements after the first systolic one were noted (Figure 11).

Analysis of radial deformation of septum was performed (Figures 12a and b). Peak systolic contraction occurred 260 ms after R-wave by strain analysis (peak strain –6.9 %) and 240 ms after R-wave by strain rate analysis (peak strain rate –1.5 s^{-1}). Beginning of the secondary contraction was synchronous with the aortic valve closure, 340 ms after R-wave in both analyses, with peak strain 490 ms (peak strain –9.3 %) and peak strain rate 440 ms (peak strain rate –1.2 s^{-1}) after R-wave. These analyses showed that the postsystolic contraction began in the earliest diastole at the beginning of isovolumic relaxation time. Third contraction was a late postsystolic contraction. It began 530 ms after R-wave, with peak 600 ms after R-wave (peak strain –8.2 %).

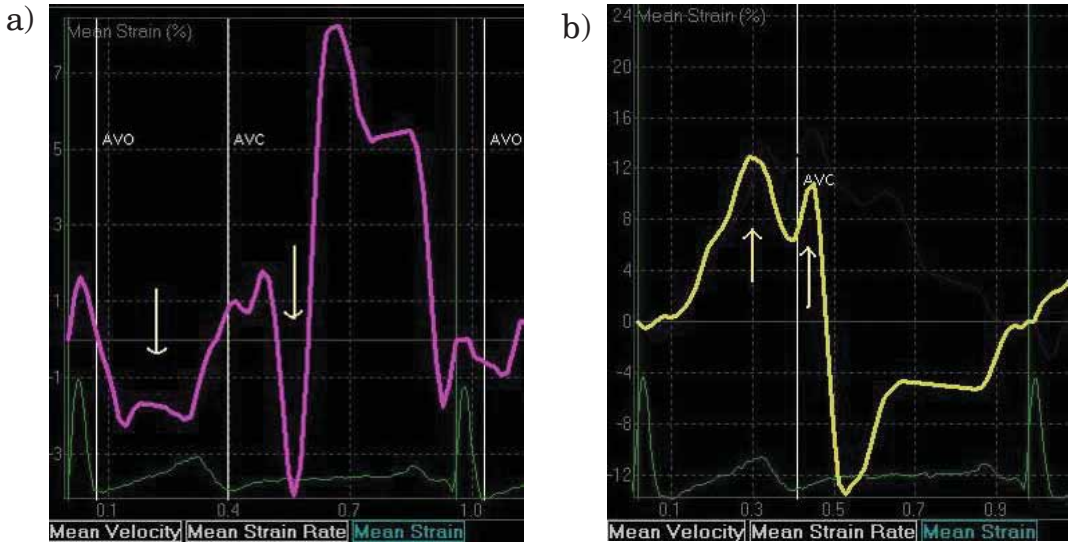

Fig. 10 a) and b). Asynchronous systolic and postsystolic contraction of interventricular septum (Figure 10a) and left ventricular posterior wall (Figure 10b). Legend: arrows mark systolic and postsystolic myocardial deformation, AVO – aortic valve opening time, AVC – aortic valve closure time.

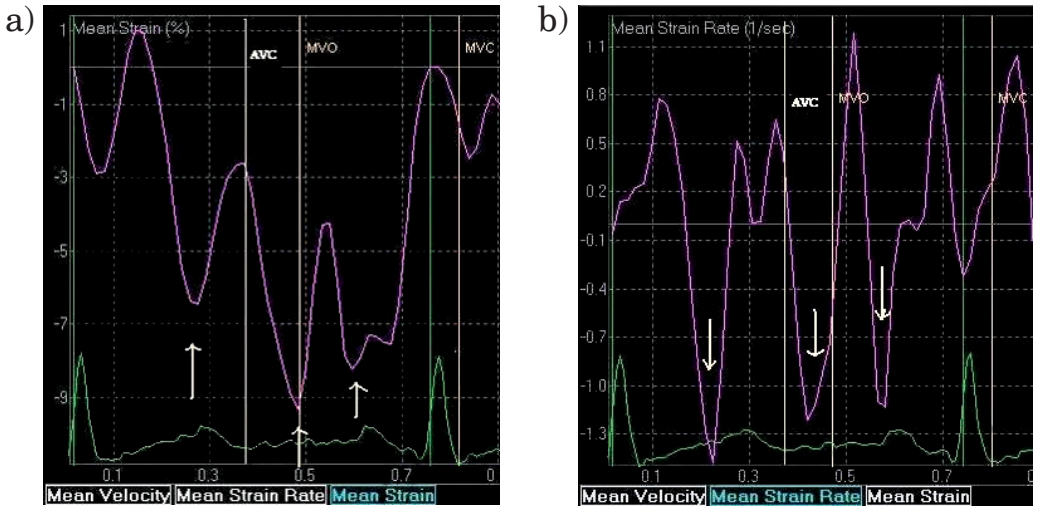

Fig. 12 a) and b). Multiple contractions of interventricular septum displayed by strain (Figure 12a) and strain rate (Figure 12b) analysis.Legend: arrows mark peaks of contractions: systolic, early postsystolic and late postsystolic, AVC – aortic valve closure time, MVO – mitral valve opening time, MVC – mitral valve closure time.

Case 5 – Multiple myocardial contractions during the same cardiac cycle – case of a secondary systolic contraction followed by a postsystolic contraction

A 56-year-old female was referred to an echocardiographical examination due to evaluation of arterial hypertension. Mild aortic and mitral valve regurgitation were found. Two additional posterior movements of septum were detected using tissue colour Doppler M-mode in parasternal long axis view. Onset of first additional movement appeared to precede peak of systolic contraction of posterior wall, suggesting it might be 'intrasystolic' (Figure 13).

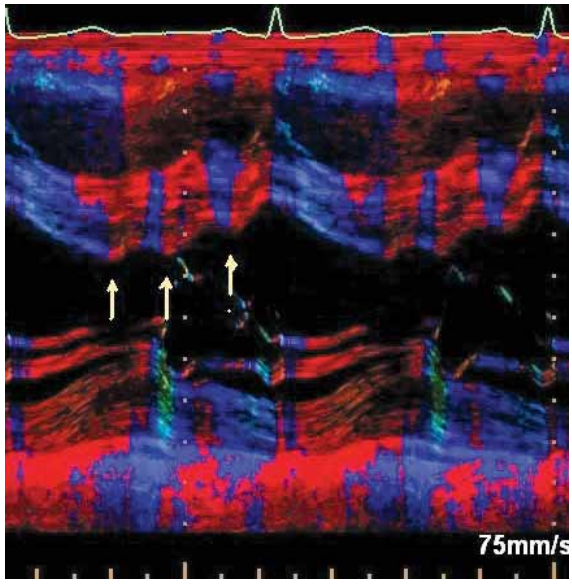

Fig. 13. Multiple myocardial contractions, with possible secondary systolic and additional postsystolic contraction.

Three deformations were clearly seen by strain and strain rate analysis (Figures 14a and b). Beginning of secondary contraction was 302 ms after R-wave by strain analysis, and peak of this contraction was 371 ms after R-wave (peak strain –35.7%), which was almost synchronous with the aortic valve closure that happened 367 ms after R-wave. This confirmed that second septal contraction occurred during systole. Third deformation was late diastolic, with onset 550 ms after R-wave and peak 630 ms after R-wave.

By strain rate analysis, peak systolic contraction began 199 ms after R-wave (peak strain rate –1.1 s^{-1}), beginning of secondary contraction was 319 ms after R--wave, with peak 369 ms after R-wave (peak strain rate –1.9 s^{-1}). This again confirmed that the secondary contraction was intrasystolic, or 'secondary' systolic. Third contraction was clearly late postsystolic.

Case 6 – Multiple myocardial contractions during the same cardiac cycle – case of a pre-contraction, systolic and postsystolic contraction

A 48-year-old female patient with left bundle branch block (of unknown time of onset) and newly diagnosed arterial hypertension was referred to an echocardiographical examination. Besides borderline left atrium dilatation (44 mm), finding was nearly normal. However, parasternal long axis view showed multiple posterior movements of septum during each cardiac cycle, with the first one appearing already during QRS signal, before the onset of expected mechanical systole (Figure 15).

Strain analysis of septal radial deformation (Figure 16a) showed peak of regular systolic contraction 380 ms after R-wave (peak strain –8.5 %). Secondary deformation began 540 ms after R-wave, with peak 620 ms after R-wave (peak strain –11 %). Considering systolic duration (440 ms) it was clear that secondary deformation was postsystolic contraction. Third deformation (first

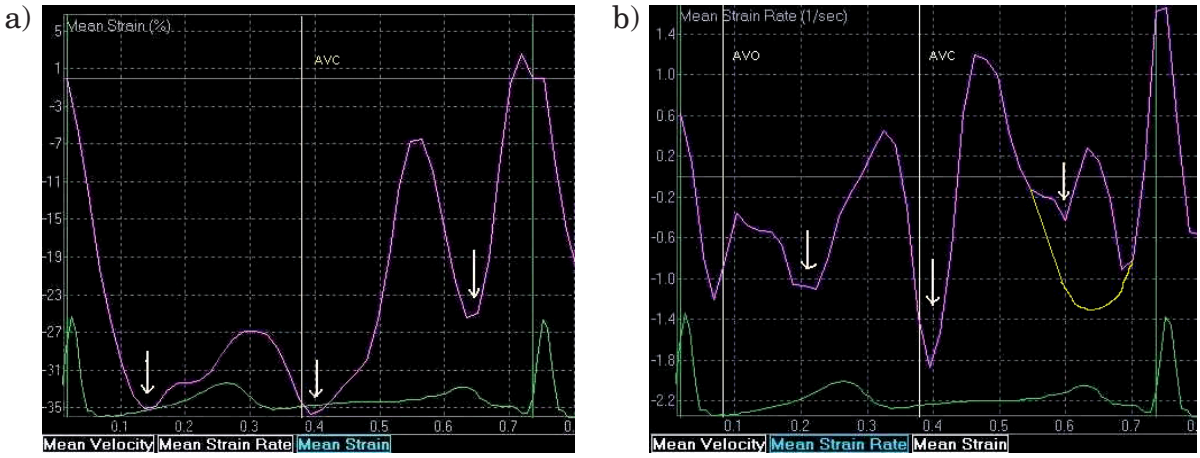

Fig. 14 a) and b). Multiple – systolic, secondary systolic and postsystolic – myocardial contractions, as seen by strain (Figure 14a) and strain rate analysis (Figure 14b). Legend: arrows mark peaks of three contractions. Yellow line in the right figure is an attempt to approximate normal strain rate curve (by excluding probable artefact). AVO – aortic valve opening time, AVC – aortic valve closure time.

and fourth in Figure 16a) began just before the onset of QRS complex (about 50 ms), reaching peak (peak strain –4.2 %) during ventricular depolarization (QRS).

Corresponding results were provided by strain rate analysis (Figure 16b): regular systolic contraction with peak 180 ms after R-wave (peak strain rate –1.6 s^{-1}), secondary systolic deformation that began 480 ms after R-wave, reached peak 550 ms after R-wave (peak strain rate –1.6 s^{-1}), which means it was a postsystolic contraction. Third deformation began, by this analysis, even more before the onset of QRS (100 ms) and lasting 160 ms (peak strain rate –1.3 s^{-1}).

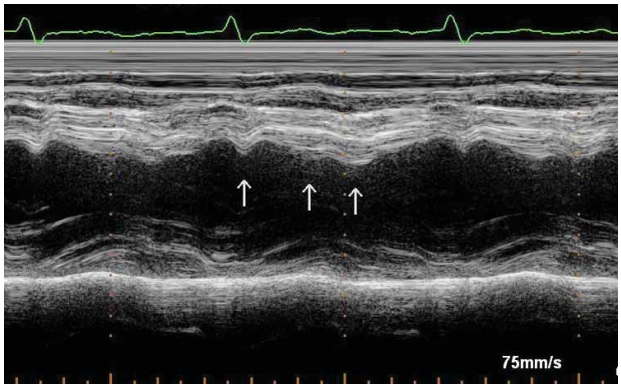

Fig. 15. Multiple posterior movements of interventricular septum – early (»presystolic«), systolic and postsystolic.

Case 7 – Fourfold myocardial contraction during the same cardiac cycle – case of a pre-contraction, double systolic and postsystolic contraction

A 28-year-old female was a subject of an echocardiographical examination as a participant of healthy control group in our ongoing study mentioned previously. Echo findings, as well as rest of the status and tests, were nor-

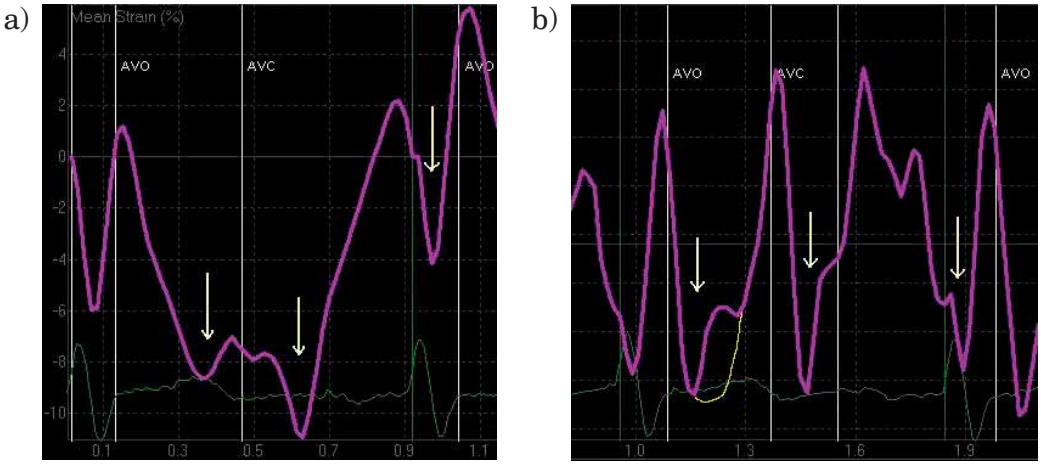

Fig. 16 a) and b). Multiple deformations of interventricular septum by strain (Figure 16a) and strain rate analysis (Figure 16b). Deformation which occurs just before the onset of QRS complex ends before the QRS ending. Legend: AVO – aortic valve opening time, AVC – aortic valve closure time. Yellow line in the right figure is an attempt to approximate normal strain rate curve (by excluding artefact).

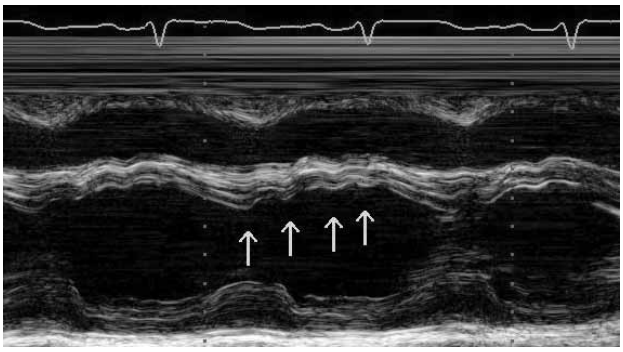

Fig. 17. Fourfold posterior movement of the interventricular septum.

mal besides multiple posterior movements of interventricular septum. Four posterior movements of septum were detected by grey scale M-mode (Figure 17) and confirmed by colour tissue Doppler imaging. The fourth deflexion began at the onset of QRS complex.

On the other hand, analysis of deformation did not firmly confirm this finding. Three deformations were clearly defined in all analyzed cycles (arrows facing downward), while the fourth deformation (arrow facing upward) remained doubtful (Figure 18). Conclusion could not be set up whether that was an abortive pre-contraction (as can be seen in preexcitation) or an artefact.

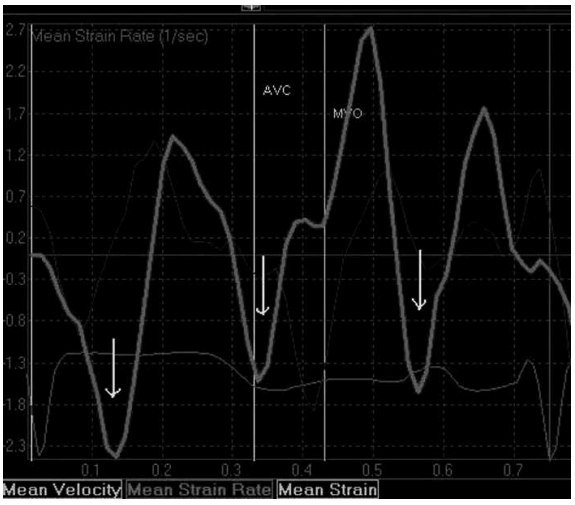

Fig. 18. Three- or fourfold (?) contraction of interventricular septum. Legend: arrows facing downward mark peaks of three myocardial deformations (systolic, secondary systolic, postsystolic), while arrow facing upward marks possible fourth, 'abortive' pre--contraction.

Discussion

Phenomenon of multiple myocardial contractions during each single cardiac cycle is poorly recognised. It looks for extensive further investigations and explanation of possible pathophysiological implications. This is the first paper which shows a significant spectrum of different modalities of this phenomenon. It also provides evidences that these unusual myocardial movements are active contractions.

Our first case in this series shows, for the first time as far as we know, a secondary systolic myocardial contraction during the same cardiac cycle. This phenomenon differs from already known phenomenon of postsystolic myocardial contraction by the timing of its occurrence during cardiac cycle. Impact of this fact would be negligible if the mechanism of an origin of systolic and postsystolic contraction is the same (which is quite possible), but we believe these two phenomena should be distinguished. We found systolic contraction far less often than postsystolic one, and always in healthy subjects. On the other hand, postsystolic contraction, which is often found in healthy subjects, is even more often found in patients with ischemic heart disease and cardiomyopathies. It is even presumed that there is a difference between postsystolic contractions occurring early during beginning of diastole and those occurring late during left ventricular filling, latter ones being suggestive of more serious ischemic pathophysiological substrate[4]. So, it is possible that these two phenomena indirectly separate physiological from potentially or clearly pathological conditions.

Postsystolic myocardial contraction has been well described during last decade, but less well explained. The existence of simultaneous postsystolic contractions of opposite myocardial walls of left ventricle has also been mentioned[4,6]. An asynchronicity of multiple contractions of opposite walls of left ventricle, shown in the third case, is even more interesting by our opinion. Peak of systolic contraction of septum precedes here 130 ms the peak of contraction of posterior wall, but furthermore secondary contraction of posterior wall, that begins immediately after primary contraction, considerably precedes postsystolic contraction of septum. A resultant asynchronicity of myocardial walls did not have any hemodynamical implications, and by our diagnostic methods none pathological elements to support a hypothesis on different pathophysiological significance of secondary systolic and 'late' postsystolic contraction were found in this case.

Although multiple myocardial contractions during the same cardiac cycle have not yet been described, they can sometimes be found. As far as we know, a case of combined normal systolic, secondary systolic and postsystolic contraction, as is our case 5, has not ever been reported. Such examples add to the quantum of knowledge on the spectrum of multiple myocardial contractions during the same cardiac cycle, but the threefold contraction further confuses when thinking about possible explanation of mechanism underlying this phenomenon.

Cases of multiple (three- and fourfold) contractions (case 6 and 7) were added to the spectrum of cases presented, opening the question of echocardiographical finding of ventricular pre-contraction, which precedes electrical systole and ends before ending of ventricular depolarization. A contraction triggered by a preexcitation seems

to us to be the most likely mechanism which could explain this phenomenon. As no signs of preexcitation were found in electrocardiogram, concealed accessory pathway could be an explanation for the phenomenon. This contraction began, according to our measurements, before the onset of QRS complex, which almost without a doubt dismisses the possibility that the phenomenon is a very early systolic contraction called 'septal flash', which is often found in bundle branch block[7]. Case 7 depicts a contraction that could be speculated about, whether it is an 'abortive' pre-contraction or an artefact, while findings of all echocardiographical analyses in case 6 support existence of clear pre-contraction, more specifically a contraction that precedes ventricular systole.

Considering possible technical limitations of tissue Doppler imaging related to obtaining samples for radial deformation analysis, it is possible that heart movement and additional respiratory movements change position of echocardiographical samples. So, recorded sample could change position during contraction analysis from interventricular septum at mid-diastole, to a more proximal segment – interatrial septum at the moment of late diastole – atrial contraction. By this, an atrial contraction could be recorded instead of ventricular pre-contraction. In such a case, findings obtained by parasternal long axis M-mode view (Figures 15 and 17) would be unexplained.

Although the width of spectrum of multiple contractions was the focus of interest in this paper, question that had to be resolved was whether these were active contractions, or passive movements of myocardial walls caused by hemodynamical changes, as was suggested by some. First 'evidence' of active contractions is given by findings obtained in M-mode view (Figure 5) which show thickening, followed by thinning, and then again by thickening, during septal movement toward posterior, anterior and then again posterior direction, consecutively. That strongly suggests contraction-relaxation-contraction during the same cardiac cycle. A more exact evidence of contraction is given by newer method of tissue Doppler data analysis, herein used strain and strain-rate analyses, which detect active myocardial deformation[8]. These analyses proved a multiple contraction in each of cases described.

Mechanism of multiple myocardial contractions during the same cardiac cycle is not clear. We believe that additional 'protecting' pathways of conduction system connecting atria with ventricles exist in a significant part of population. The electrophysiological characteristics of such pathways would include slower conduction of impulses than through 'normal' pathways. So, they would be activated after regular conduction pathways in case of inadequacy of 'main' conduction system or in case of insufficient myocardial contraction. We suppose that the activation of such pathways in healthy population manifests itself as a secondary systolic or as an 'early' postsystolic contraction, while in case of ischemia, with slower myocardial conduction, they cause 'late' postsystolic contraction. If this hypothesis is correct, a question whether such pathways exist in all population or are a genetic variant, still remains unanswered. An extensive epidemiological analysis should be carried out to answer this question, and the results could change the established perception of physiological myocardial kinetics. In fact, this paper is presented to open some new questions on mechanisms of basic cardiovascular physiology and pathophysiology as well.

Main limitation of this study can be found in the lack of a 'golden standard' for non-invasive detection of myocardial contraction, which could correct problems arising from tissue Doppler analysis. Tissue deformation analyses used herein is still far from perfect, although certain improvements are done through newer methods, as currently developed speckle tracking analysis is[9]. Considering convincing results of simpler echocardiographical methods (M-mode, especially enhanced with colour tissue Doppler imaging), a common sense suggests accuracy of here presented findings, and, naturally, a need for further investigations. Less important for the topic, the question of pre-contractions as a reflection of preexcitation (cases 6 and 7), lacks the confirmation obtained by electrophysiology. Nevertheless, very convincing ultrasound findings inspired us of presenting our results in front of a scientific auditorium.

In conclusion, the spectrum of phenomenon of multiple myocardial contractions that occur during the same cardiac cycle is presented. Proofs are given that the movements were active contractions. Questions on mechanism, causes and pathologic significance of the phenomenon are to be resolved.

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SPEKTAR FENOMENA VIŠESTRUKE KONTRAKCIJE MIOKARDA TIJEKOM ISTOG SRČANOG CIKLUSA

S A Ž E T A K

U ehokardiografskoj analizi modaliteta iregularne kinetike interventrikularnog septuma najinteresantniji je fenomen višestruke kontrakcije miokarda koja se događa unutar jednog te istog srčanog ciklusa, izazvan jednim jedinim sinusnim impulsom, repetitivno. U ovom je radu metodama tkivnog Dopplera dokazano da se u svim ovdje prikazanim oblicima ovog fenomena radi o aktivnim kontrakcijama. Prikazan je široki spektar ovog fenomena, koji se može naći i u zdravih ljudi i u bolesnika. Oblici višestruke kontrakcije miokarda u istom srčanom ciklusu prikazani u ovoj analizi su: sekundarna sistolička kontrakcija, postsistolička (i kasna postsistolička) kontrakcija, višestruka (tro- i četverostruka) kontrakcija s kombinacijom sekundarne sistoličke i postsistoličke kontrakcije, te kombinacija regularne sistoličke, sekundarne sistoličke ili postsistoličke kontrakcije s pre-kontrakcijom u okviru vjerojatne preekscitacije. Objašnjenje nastanka ovih fenomena nalazimo u mogućem postojanju akcesornih, skrivenih, sporih segmenata provodnog sustava srca.