Effects of acute ischaemia on intramyocardial contraction heterogeneity

New ultrasound technologies to study an old phenomenon

P. Colonna, R. Montisci, L. Galiuto*, L. Meloni and S. Iliceto

Institute of Cardiology, University of Cagliari, Italy; *University of San Diego, CA, U.S.A.

Pathophysiology of transmural heterogeneity

The diagnostic approach of patients with coronary artery disease normally relies on the assessment of global and regional perfusion and contraction in the normal and pathological areas of the myocardium. However, experimental pathophysiological studies have clearly shown that microvascular flow, metabolic consumption and the strength of contraction do not behave homogeneously in the different transmural layers of the myocardium.

In dogs with normal coronary arteries, for example, the consumption of oxygen \cite{1} and the concentration of high energy substrates such as kinase phosphate and ATP \cite{2} is greater in subendocardial layers. This transmural gradient is also preserved during increased myocardial consumption, such as in high rate atrial pacing \cite{3}. The myocardial blood flow behaves similarly, with a perfusion gradient favouring the subendocardium \cite{4}.

In addition myocardial contraction has a transmural heterogeneity \cite{5,6}. When investigating myocardial contractility the anatomical arrangement of myocardial fibres has to be taken into account. Subendocardial and subepicardial fibres are mostly parallel to the long axis of the left ventricle and the midwall fibres are oriented circumferentially. During systole, these fibres contract and the longitudinal and transverse axes of the left ventricle shorten, while the ventricular wall thickness increases \cite{5,6}.

Several methods have been developed over time in experimental laboratories to measure contractility in the different myocardial layers. The first method used to investigate the heterogeneity of intramyocardial contraction utilized paired sonomicrometers, implanted in the ventricular wall, with the ultrasonic crystals aligned to the anatomical direction of the myocardial fibres. The superficial pair, aligned along the longitudinal fibres in the subepicardium, measured ventricular long axis shortening \cite{7,8}. The inner-intermediate pair, aligned circumferentially in the subendo- mid-myocardium, investigated circumferential shortening. Thanks to this first method it was possible to demonstrate that subendocardial fibres contract much more than subepicardial ones.

To resolve the confounding effect of different fibres shortening orientation, Gallagher et al. \cite{7} studied myocardial thickening of different intramyocardial layers. They used two adjacent pairs of crystals implanted along the thickness of the myocardium: one pair to measure overall left ventricular thickening and the other to measure only the outer half of myocardial thickening. This technique showed that the subendocardial half of the myocardial wall contributes 83% of total systolic thickening \cite{7,9,10}.

Subsequent experimental studies, using a pulsed Doppler transducer attached to the epicardium to measure myocardial contraction velocity in different myocardial layers, confirmed the previous findings of a greater subendocardial contribution to overall myocardial thickening \cite{11}.

Further confirmation of the concept of heterogeneous myocardial contraction has been provided by other experimental studies in which traditional M-mode echocardiography was used to measure, at high temporal resolution, the distance between sutures placed at various distances within the thickness of the myocardium. The sutures act as a target and divide the thickness of the wall into three different layers (Fig. 1) \cite{12,13}.

Key Words: Myocardial contraction, echocardiography, ischaemia, myocardial stunning, transmural heterogeneity.


Correspondence: Dr Paolo Colonna, MD, Institute of Cardiology, PO S. Giovanni di Dio, Via Ospedale 46, 09124 Cagliari, Italy.

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The data collected with this method showed that, during the increase in contractility due to physical exercise or inotropic dobutamine stimulus, the subendocardium contributes most of the wall thickening\textsuperscript{12,13}.

**Ischaemia and transmural contraction**

In basal conditions, vascular tone in the microvasculature of the subendocardium is much lower and coronary reserve is physiologically lower in this myocardial layer, due to greater oxygen demand in baseline conditions. Therefore, when an acute reduction of coronary blood flow occurs, the subendocardium is the first layer to be vulnerable to ischaemia. In fact, when producing progressive coronary occlusions\textsuperscript{14–18} or when dogs performing physical exercise have a significantly narrowed coronary artery\textsuperscript{19,20}, a severe reduction in perfusion and kinesis occurs in the subendocardium, but only a trivial reduction of both factors can be detected in the subepicardium. These experimental studies have also demonstrated that, during partial coronary occlusion the reduction in flow is limited to the deepest layers and the contractility is mainly depressed in the subendocardium. These results were also confirmed with the pulsed Doppler\textsuperscript{21} and the intramyocardial suture technique described above\textsuperscript{22}.

**Heterogeneity in stunned myocardium and infarct size**

After the release of 15 min coronary occlusion, the ischaemic myocardium progressively recovers its contractility. This post-ischaemic recovery in contractility has been demonstrated to be heterogeneous along the myocardial wall, but much quicker in the subepicardium than in the subendocardium, with a ‘subendocardial stunning’ phenomenon\textsuperscript{21}. Several mechanisms have been hypothesized to explain this delay in recovery by the subendocardium: (a) the relatively more severe reduction of flow affecting the subendocardium, (b) a delay in restoring subendocardial blood flow (microcirculatory stunning), (c) a change in the local geometry and curvature of the post-ischaemic wall, (d) heterogeneity of accumulation of reperfusion-derived metabolites (free oxygen radicals).

Following a longer period of ischaemia, necrosis also progresses heterogeneously from the endocardium to the epicardium\textsuperscript{20,23,24} and this wavefront phenomenon is not dependent on the collateral circulation\textsuperscript{24}. Liebermann et al. showed that if the histological necrosis, measured 48 h after coronary occlusion, affects less than the subendocardial 20% of the myocardial wall thickness, a proportional reduction in myocardial thickening occurs; if the necrosis exceeds the threshold of 20%, a total absence of myocardial thickening is observed (Fig. 2)\textsuperscript{25}.

In another experimental study, the histological transmural extension of necrosis across the myocardial wall and echocardiographic myocardial thickening were studied 2 weeks after myocardial infarction\textsuperscript{26}. There was a direct correlation between myocardial thickening and the percentage of necrosis in those myocardial infarctions involving subendo- and mid-myocardium, up to a value of about 60% of histological necrosis\textsuperscript{26}. This different ‘necrosis thickness threshold’ for akinesia, found in the two studies, is probably due to the timing of echocardiographic measurements of myocardial thickening. In fact the echocardiograms performed by Liebermann, 48 h after coronary occlusion, could have erroneously considered ‘akinetid because of necrosis’ some segments with a considerable amount of stunned myocardial wall thickness. Conversely, the echocardiograms performed after 2 weeks showed a correlation closer to the real pathological findings, because a percentage of the wall thickness had probably recovered from transient subendocardial stunning.

The clinical importance of the above data is underlined by some interesting recent findings. In a group of animals with considerable infarct...
transmurality, there were large diastolic and systolic volumes and considerable regional and global left ventricular dysfunction over 6 weeks. In this group of dogs, there was also a great number of complications, such as left ventricular thrombus, arrhythmias and deaths[27]. Moreover, Sklenar et al. showed that the poor correlation between histological infarct size and myocardial thickening at rest remarkably improved during dobutamine infusion[28]. The dobutamine did not improve the correlation if the segment was supplied by a coronary artery with residual stenosis. This better correlation was explained by the fact that the dobutamine infusion improved the contractility in the segments with a basal akinesia, but without transmural necrosis (viability in the mid-superficial myocardium). In the presence of residual stenosis of the infarct-related artery, dobutamine infusion did not ameliorate myocardial contractility because of a biphasic response to dobutamine of the stunned myocardium, thus correlation remained poor[28,29].

**Evaluation of intramural heterogeneity in humans**

At present there is a lack of information concerning the heterogeneity of myocardial contraction in the human heart. This is because it is impossible to use invasive techniques to calculate the transmural differences of myocardial contraction and ischaemia. In recent years, the development of ultrasound technology has pointed to the feasibility of investigating myocardial heterogeneity. These new techniques, such as tissue characterization with integrated backscatter, tissue Doppler imaging, and myocardial contrast echocardiography look very promising, although they are still not ready to be used in the bedside clinical decision.

**Integrated backscatter**

Ultrasonic tissue characterization of the heart is a new application of echocardiography to evaluate the structural and functional state of myocardium. This method provides quantitative indexes of the heart’s physical properties and variations due to pathophysiological changes. Tissue characterization at present appears capable of analysing different layers along the myocardial transmural thickness, while conventional echocardiography, although capable of segmental quantitative analysis, cannot differentiate the relative role of the different layers of the myocardium.

Conventional echocardiography identifies the boundary between blood pool and endocardium thanks to the specular reflection from the ultrasonic signal wavelength which is smaller than the boundary dimension. On the other hand, quantitative tissue characterization detects intramural tissue structural components: with this technique the wavelength of the incident beam is much greater than the boundaries between the different components of the tissue, so the ultrasound become scattered in a multidirectional phenomenon[30].

Since it is not possible to place the myocardium between a transmitting and a receiving transducer, the actual software for tissue characterization measures the waves that are scattered and redirected back to the same transducer. The amount of this ultrasonic backscattered energy depends on the inhomogeneities within the tissue (the number and density of intramural scatterers) and on the ultrasound frequency. Basically, it is possible to measure the complete ultrasound frequency spectrum of the backscatter signal or the frequency average of it, called integrated backscatter. This integrated backscatter is calculated by processing the signal directly in the time domain, producing real-time integrated backscatter two-dimensional images. It is then possible to select a region of interest to calculate the integrated

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Figure 2  The threshold effect of necrosis thickness. The reduction in myocardial thickening, measured 48 h after coronary occlusion, is proportional to the percentage of wall thickness histological necrosis if the necrosis affects less than the subendocardial 20% of myocardial wall thickness. If the necrosis exceeds the threshold of 20%, the above correlation is lost because of total akinesia of myocardial wall beyond this threshold. Adapted from Liebermann et al.[25].
backscatter values in a particular segment or layer of myocardium.

During contraction and relaxation of normal myocardium, a consistent and reproducible cardiac cycle-dependent pattern with higher values of integrated backscatter near end-diastole and lower values (around 6 dB) near end-systole exists[31]. These cyclic variations of integrated backscatter are an expression of regional intramural myocardial contractile performance[32,33] and are related to contractility[34], but are not directly dependent on inotropic state changes[32,33,35]. In dog models, the behaviour of integrated backscatter cyclic variations were studied in the different layers, placing the region of interest at different depths in the myocardial wall. These studies demonstrated that normal myocardial segments, at rest and during inotropic stimulation, show a 2 dB greater amplitude of integrated backscatter cyclic variations in the subendocardial myocardial layer than that of the subepicardial one[32,36]. We and others have shown a similar finding in human beings of integrated backscatter cyclic variations in transmural gradients, by means of transoesophageal[37] and transthoracic[38,39] two-dimensional echocardiography.

**Effects of ischaemia on integrated backscatter**

Experimental studies showed that during acute myocardial ischaemia, provoked by coronary artery ligation, not only myocardial thickening but also integrated backscatter cyclic variations were abolished[40,41]. More recently Vitale et al.[42] showed similar behaviour in integrated backscatter cyclic variations during stress tests in patients, but did not investigate the recovery modalities or myocardial thickening.

Interestingly, ischaemia-induced blunting of integrated backscatter cyclic variations is related to the duration of coronary artery occlusion. If reperfusion is achieved very early (after 5 min occlusion) recovery of integrated backscatter cyclic variations is immediate and complete; if reperfusion is delayed (after 30 min) the recovery period lasts several hours[43]. Surprisingly, in several experimental studies, integrated backscatter cyclic variations in the reperfusion phase recovered much faster and more completely than other conventional regional left ventricular functional parameters (myocardial thickening, systolic shortening, wall motion score index)[44,45].

Similar to these experimental studies, we measured integrated backscatter cyclic variations with transoesophageal echocardiography during atrial pacing stress tests in 36 patients with suspected coronary artery disease. In segments related to a significantly narrowed coronary artery, myocardial thickening decreased at peak-pacing, was still reduced 5 s later, and recovered 2 min after atrial pacing interruption; backscatter cyclic variations were blunted at peak-pacing and immediately recovered after pacing interruption (Fig. 3)[46]. Picano et al. observed a similar dissociation between blunting of conventional video image gray level cyclic variations and segmental dysfunction, in patients studied immediately after coronary artery occlusion during angioplasty or intra-operative ischaemia[47,48].

**Figure 3** Myocardial thickening (Th%, broken line) and integrated backscatter cyclic variations (IBScv, solid line) in myocardial segments supplied by coronary arteries with significant (>50%) stenosis narrowing. Both myocardial thickening and integrated backscatter cyclic variations are significantly reduced at peak atrial pacing. Five seconds after atrial pacing interruption (Post) recovery to baseline values is already present in integrated backscatter cyclic variations, but not in myocardial thickening. Adapted from Iliceto et al.[46].

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Potential of integrated backscatter cyclic variation analysis in the early detection of stunned myocardium

To evaluate the potential of integrated backscatter cyclic variation analysis in the detection of stunned myocardium, Milunski et al.[34] studied, after an acute myocardial infarction, patients with and without infarct-related artery patency (which were supposed to have a greater amount of viable myocardium). In those without infarct-related artery patency, neither wall motion score index nor integrated backscatter cyclic variations recovered from early (within 24 h of chest pain onset) or late (pre-discharge) studies; in those with infarct-related artery patency, integrated backscatter cyclic variations improved in the late study, while wall motion score index did not[49]. Assuming that this disparity between techniques was due to post-ischaemic dysfunctioning, but still viable (‘stunned’) myocardium, the analysis of backscatter cyclic variation recovery could be a sensible and early index with which to identify viability[34,50].

These results have very recently been validated in the setting of acute myocardial infarction treated with primary coronary angioplasty. In these patients, a normalization of integrated backscatter cyclic variations was observed 3 days after coronary angioplasty, mostly in the akinetic segments that showed functional recovery only 3 weeks after the acute phase[50]. At present, the behaviour of backscatter cyclic variations is not known in the different transmural layers of the stunned or infarcted myocardial segments.

However, some actual limitations restrict the bedside use of this tissue characterization technology. First of all, this is still a non-automatic and time consuming method. Secondly, the most precise cyclic variations curves are obtained when the insonification angle of the ultrasonic beam is perpendicular to the myocardial segment. When this angle is much smaller than 90° the strength of the backscatter is sometimes low, resulting in images which are difficult to be correctly analysed[51]. Thanks to the transoesophageal approach and the new facilities these problems could be partially solved[52].

Effects of ischaemia on backscatter endo-epicardial gradient

The above mentioned transmural gradient of cyclic variations has been demonstrated to be abolished during coronary occlusion[37], mainly because of blunting of subendocardial integrated backscatter cyclic variations, and to recover after 15 min of reperfusion. During this experiment there was a reduction in the subendocardial blood flow comparable to the reduction of integrated backscatter cyclic variations.

In patients with coronary artery disease we demonstrated during atrial pacing stress-induced ischaemia that the integrated backscatter cyclic variations were blunted only in the subendocardium. They remained substantially unchanged in the subepicardium and in all the layers of myocardial segments supplied by non-stenotic coronary arteries (Fig. 4)[37].

Subendocardial post ischaemic stunning

Bolli et al. have demonstrated that after 15 min of coronary occlusion followed by reperfusion, the contraction in subendocardial layer recovery was much slower than that in the subepicardium. Therefore, after acute myocardial ischaemia it is possible to observe subendocardial stunning[51]. Since the subendocardial layer is responsible for a great part of the overall transmural contraction, the loss of its contribution due to ischaemia can provoke a reduction in overall transmural thickening during two-dimensional echocardiography[13,22].

As regards experimental[43] and clinical[46] data, the post-ischaemic dissociation between integrated backscatter cyclic variations and myocardial thickening recovery time could be explained as follows: the overall thickening (mainly determined by subendocardial layer contractility) did not recover immediately after ischaemia because of functional impairment of the subendocardial layer, and conversely, at the same time, the mid-inner myocardium (which does not play an important role in the overall thickening) had already partially recovered. However, integrated backscatter cyclic variations appear to return to normal immediately after atrial pacing interruption, probably because the region of interest is positioned in the mid-myocardium, where myocardial stunning lasts for less time than in the subendocardial layer. This hypothesis underlines the relevant impact of the positioning of the region of interest inside the myocardium and of the spatial definition of the echocardiographic integrated backscatter modules and analysis packages. Our preliminary data with integrated backscatter calculated in the different myocardial layers did not demonstrate subendocardial stunning, probably because of the limited severity of the atrial pacing-induced ischaemia or the spatial resolution of the echocardiographic integrated backscatter machine[53].

Tissue Doppler imaging

Technical background

In conventional echocardiography, the Doppler principle has been utilized to evaluate velocity and direction of blood flow non-invasively. Accordingly, the standard Doppler system is set in order to analyse only high-frequency, low-amplitude signals, derived by blood flow, while all the other reflected echoes are removed by a high-pass filter, before signal processing. The acoustic characteristics of Doppler signals derived from ventricular wall motion are peculiar and different from those derived from blood flow: (1) wall motion velocity is much slower than blood flow velocity, approximately 10 cm . s⁻¹ or less; (2) the Doppler signal amplitude from wall motion is 40 dB larger than that coming from red blood cells.

In tissue Doppler imaging, the routine colour flow mapping instrumentation has been modified to analyse and measure ventricular wall motion: the low-frequency high-amplitude Doppler signals derived from...
Figure 4  Normal segment showing heterogeneity of integrated backscatter cyclic variations in different transmural layers. Panel (a) Graphs showing subendocardial and subepicardial cyclic variations of integrated backscatter: the region of interest is placed in both the subendocardial and the subepicardial layers of the posterior myocardial wall. Panel (b) Enlarged picture which shows that subendocardial layer backscatter cyclic variations are greater than subepicardial ones.
wall motion bypass the high-pass filter and are put
directly into the autocorrelator. Moreover, an accurate
calculation of low velocity has been improved to be able
to measure very low velocities, to a lower limit of
0·2 cm. s$^{-1}$[54].

Tissue motion velocity signals are displayed in
real time in an adequate colour display format. Different
velocity maps are available: by convention, myocardial
cardiac walls moving toward the transducer are colour-coded
in red, while walls moving away from the transducer are
colour coded in blue. Furthermore, red and blue
colours have different brightness levels, to enhance low
velocities. Colour Doppler information derived from
myocardium are usually superimposed on the B- and
M-mode two-dimensional echocardiographic image,
thus facilitating the determination of endo-epicardial
boundaries[55].

Myocardial velocities may be calculated using
the software package included in echocardiographic
machines, by using an off-line computer program[56,57]
In vitro and in vivo studies have demonstrated that
tissue velocities measured with tissue Doppler closely
correlate with the actual velocity of an acoustic reflector
(r=0·99). In a clinical setting, Miyatake et al.[58] and
Donovan et al.[59] independently validated, in a group of
normal subjects, the tissue Doppler velocity measure-
ment in comparison to the conventional M-mode
echocardiography and the correlation coefficient ranged
from 0·87 in the Donovan study to 0·99 in the Miyatake
study.

The most important limitation of this technique
is related to the translocation movement of the heart: the
transducer is stationary on the chest wall and the heart
moves in the thoracic cavity along the cardiac cycle. The
translocation is responsible for the different velocities
calculated in posterior and anterior segments or when
analysing the same segment from different echocardio-
graphic views[55,60,61]. Moreover, mostly when using the
apical views, there could be an angle greater than 30°
between the transducer ultrasonic beam and the vector
of the movement of the left ventricle walls. This angle
can be responsible for velocity values significantly lower
than the real ones.

Myocardial velocity gradient
When considering a normally contracting myocardial
wall studied by tissue Doppler imaging, the presence of
a gradation in velocity along each scan line is evident,
with the endocardium moving faster than the epicar-
dium and therefore reflecting the rate of increase in wall
thickness (Fig. 5). This pattern has been described as
myocardial velocity gradient, and can be defined as
the difference in myocardial velocity between the endo-
cardium and epicardium. Two different groups of
researchers developed their own computer software to
convert the digital representation of colours into velocity
data, obtaining the velocity gradient of the wall in any
image.

To validate this technique, Fleming et al.[56]
compared the myocardial velocity gradient calculated
from tissue Doppler M-mode images and the rate of
change in wall thickness from conventional M-mode
images. A strong correlation between the two methods
was demonstrated, suggesting that velocity gradient had
potential for the assessment of myocardial contractility.
This calculation appears particularly useful for two main
reasons. Firstly, it gives a reliable estimate of the myo-
cardial velocity of contraction and relaxation originating
from the difference between endo and epicardial vel-
ocity, independent of translocation of the heart, which,
however, affects absolute velocity measurements[62,63].
Secondly, it offers the unique opportunity of studying,
in vivo, the intramyocardial mechanical activity,
through a parameter, the velocity of motion, capable of
discriminating between different wall layers.

The first step in the study was to obtain a range
of normal values from normal volunteers. An interesting
observation was that the velocity gradient was not
uniform throughout the cardiac cycle, but showed dif-
ferent values during specific cardiac phases; further-
more, during the same cardiac phase, the velocity
gradient was different in different myocardial walls[62].
Thus, myocardial mechanical activity, expressed as
velocity of motion, behaves heterogeneously with differ-
ences expressed in three main areas: (1) between endo-
cardium and epicardium; (2) between different cardiac
phases; (3) between different myocardial walls. Once
normal velocity values and normal patterns of myocar-
dial heterogeneity were well studied, tissue Doppler
imaging was applied to different pathological myo-
cardial states, involving left ventricular myocardial
function.

In patients with old myocardial infarction and
left ventricular regional wall motion abnormalities,
studied by Uematsu et al.[63], dysfunctioning infarcted
regions were colour coded at tissue Doppler, but there
was little change in colour brightness between the endo-
cardium and epicardium, compared with that of
non-infarcted regions (Fig. 5). The velocity gradient
calculation, in fact, showed a marked difference between
infarcted and non-infarcted regions both in the antero-
septum and in the posterior wall. The velocity gradient
in the infarct region was also significantly lower than the
one in control subjects, in both the antero-septum and
posterior wall. In dilated cardiomyopathy with impair-
ment of left ventricular contraction, the range of colour
brightness at tissue Doppler was still maintained, but
the endo-epicardial change was reduced. In both the
antero-septal and posterior wall, the myocardial velocity
gradient was demonstrated to be significantly reduced
in patients with dilated cardiomyopathy, compared to
normal subjects. This reduction in velocity gradient is
evident in all systolic cardiac phases, but, during
diastole, only during atrial contraction[64].

Another useful clinical application of tissue
Doppler imaging, is in the assessment of intramural
function in patients with left ventricular hypertrophy of
different aetiologies. The analysis and quantitation of
myocardial velocity gradients, in fact, has shown a
significant reduction in this parameter in patients with
Figure 5  Inversion of myocardial velocity gradient with tissue Doppler imaging. Panel (a) Normal myocardium gradient favouring subendocardial velocity in both anterior septum (subendo$= -1.5 \text{ cm} \cdot \text{s}^{-1}$, subepi$= -0.3 \text{ cm} \cdot \text{s}^{-1}$) and posterior myocardial wall (subendo$= 4.2 \text{ cm} \cdot \text{s}^{-1}$, subepi$= 2.2 \text{ cm} \cdot \text{s}^{-1}$). Panel (b) Inverted myocardial velocity gradient in a ischaemic posterior myocardial wall (subendo$= 3 \text{ cm} \cdot \text{s}^{-1}$, subepi$= 3.3 \text{ cm} \cdot \text{s}^{-1}$).
hypertrophic cardiomyopathy, while patients with left ventricular hypertrophy secondary to hypertension had values comparable to normal subjects. Thus, myocardial velocity gradient measurement has potential in the identification of patients with hypertrophic cardiomyopathy within the group of hypertrophies of different origins[65].

Myocardial contrast echocardiography

The use of contrast media in conjunction with new technological tools implemented in echocardiographic equipment could have a unique potential in providing information on coronary blood flow in the different transmural layers[60]. Some studies using intracoronary injected contrast with conventional echocardiography obtained controversial results in the demonstration of transmurally heterogeneous distribution of blood flow[67-69]. It was not possible to demonstrate a significant difference in myocardial opacification between subendocardial and subepicardial areas in both the transmural distribution of myocardial contrast, with conventional echocardiography, in normal conditions and during myocardial ischaemia[67,68]. Conversely, Perchet et al. observed the subendo/subepicardial ratio before and after coronary angioplasty and demonstrated the ability of myocardial contrast echocardiography to detect transmural differences[69]. With the advent of new resonating echocontrast agents and of the second harmonic echocardiography it has been possible to demonstrate myocardial perfusion with intravenous contrast injection and transthoracic echocardiography[70,71]. These new techniques seem to ameliorate the resolution of the images and will perhaps permit a separate measurement of contrast opacification in the different myocardial layers.

Intramyocardial heterogeneity: clinical implications

With the advent of effective therapeutic reperfusion strategies (thrombolysis, percutaneous transluminal coronary angioplasty) in the acute phase of myocardial infarction, myocardial salvage is frequently achieved. This salvage occurs in a heterogeneous way and involves, in a progressive manner, different layers of the myocardium. Thus, after prolonged myocardial ischaemia followed by reperfusion, different myocardial layers can present different anatomical and/or functional characteristics. This heterogeneity has an important influence on overall myocardial thickening that, on the other hand, not only represents the final result of intramyocardial disorders, but is also the major determinant of overall left ventricular dysfunction and size. This latter is the most powerful predictor of prognosis after myocardial infarction. Regional thickening is influenced mainly by the extent of non-reperfusion, scarred myocardium (usually confined to subendocardial layers in case of successful and timely reperfusion); an important influence, however, is the extent and functional status (alive and normo-contracting; alive but stunned and temporarily dysfunctioning; alive but chronically dysfunctioning) of salvaged myocardium beyond the scarred area. These different anatomical and functional intramyocardial patterns represent the basis for different functional outcomes of regional and hence global left ventricular function[10].

In the light of the potential impact of intramyocardial heterogeneity on functional and consequently clinical outcome, the importance of new non-invasive technologies capable of explaining these effects of myocardial contractility is obvious. These new techniques will probably open a window on an almost totally unexplored world of cardiac pathophysiology, even if it is still necessary to investigate this phenomenon in different aspects of pathology. The impact of these new investigations could be of great relevance not only for a better understanding, but also for an improved and more appropriate treatment of patients after acute myocardial infarction.

References


