Analysis of the Influence of the Electrical Asynchrony on Regional Mechanics of the Infarcted Left Ventricle Using Electromechanical Heart Models*

Feng LIU**, Ling XIA** and Xin ZHANG***

Asynchronous electrical activation, as induced by myocardial infarction, causes various abnormalities in left ventricle function. The influence of the electrical asynchrony on regional mechanics of the left ventricle is simulated using a mechanical heart model and an electrical heart model. The mechanical model accounts for the ventricular geometry, the fiber nature of the myocardial tissue, and the dependency of the activation sequence of the ventricular wall. The electrical model is based on a heart-torso model with realistic geometry, and different action potential waveforms with variables in duration are used to simulate the abnormal electrical activation after myocardial infarction. Regional deformation, strain and stress are calculated during systole phase. The preliminary results show that asynchronous electrical activation, as an important factor, significantly affects regional mechanical performance of the infarcted left ventricle, it indicates heterogeneous contraction pattern and elevated systolic stresses near the injured region. The simulated results are compared with solutions obtained in the literature. This simulation suggests that such coupled heart models can be used to assess the mechanical function of the left ventricle with diseases such as myocardial infarction, and more realistic models of cardiac function are essential for clinical evaluation of heart disease.

Key Words: Human Left Ventricle, Myocardial Infarction, FEM Analysis, Mechanical Model, Electrical Model

1. Introduction

The human heart is a neuroelectrically actuated mechanical pump. Before mechanical contraction, the electric excitation is generated and transmitted throughout the heart wall via special transmission system. When myocardial ischemia or infarction (MI) occurs, the electrical transmission progress or pattern is much more heterogeneous and asynchronous⁽¹⁾⁻⁽³⁾, which causes various abnormalities in left ventricle (LV) function⁽⁴⁾. For example, the influence of the electrical asynchrony on regional mechanics of LV is obvious in such pathological condition, but the knowledge of the excitation-contraction coupling mechanism in three-dimensional

infarcted ventricular wall is limited⁽⁵⁾. The purpose of this paper is to investigate the interactions between electrical conduction and mechanical function in the infarcted LV using mathematical modeling method.

Information about LV mechanical performance is of critical importance in understanding the etiology of heart disease such as MI⁽⁶⁾. Regional wall stress and strain are often used to assess LV function in the presence of ischemia and MI. Myocardial strain distributions can be measured experimentally and clinically⁽⁷⁾, while regional wall stresses must be computed from mathematical models because reliable measurement of ventricular wall stress is practically impossible. A number of mechanical heart models have been developed to approximate wall stress and some of them were presented for the ischemic ventricle⁽⁸⁾⁻⁽¹³⁾. Bogen et al.⁽⁸⁾ used a symmetric membrane model of the infarcted ventricle, in which infarction was modeled by a change in tissue stiffness, to investigate the relation between ventricular function and

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** Institute of Biomedical Engineering, Zhejiang University, China. E-mail: feng.liu@cmr.uq.edu.au

*** Department of Bioengineering, University of Illinois at Chicago, U.S.A.

infarct size. Larry et al.⁽⁹⁾ presented an ellipsoidal shell model for the infarcted LV during end diastole and end systole based on the nonlinear shell theory. Finite element method (FEM) may be the most powerful numerical tool to analyze cardiac mechanics since FE models can include 3-D ventricular geometry, fiber and connective tissue architecture, the complicated constitutive properties of the myocardium and the boundary conditions. Perl et al.⁽¹⁰⁾ developed a FE model to simulate the mechanics of the infarcted LV, where they applied an inverse calculation procedure in which active stresses were chosen so that the calculated change in ventricular pressure and volume with time coincided with experimental data. Recently, a number of sophisticated 3-D FE models of ischemic ventricle have been developed. For example, VanCampen's model⁽¹¹⁾ considered many important aspects such as muscle fiber orientation, redistribution of intracoronary blood in the ventricular wall, viscoelastic behavior of myocardial tissue, and regional decrease of myocardial perfusion, resulting in an ischemic region. Bovendeerd's model⁽¹²⁾ considered the thick-walled ventricular geometry, the fibrous nature of the myocardial tissue, and the dependency of active muscle fiber stress on time, strain, and strain rate. Mazhari et al.⁽¹³⁾ combined a FE model to study the structural basis of regional dysfunction under conditions such as acute MI and ischemia-reperfusion. The functional border zone was also scrutinized and they found that regional wall stresses affect the functional border zone much more than anatomic variations.

Although these models are reasonable enough to reproduce some complex features of regional myocardial mechanics, the influence of the asynchronous electrical activation after MI is usually simplified or neglected. For example, in Bovendeerd's model⁽¹²⁾, the sequence of depolarization of the ventricular wall was assumed to be rotationally symmetric. The activation pattern was assumed to remain unaffected by ischemia. Ischemia was modeled by disabling the generation of active stress while leaving passive material behavior and activation sequence unchanged. In fact, experimental studies of the myocardial action potentials following coronary artery occlusion have shown that the resulting regional ischemia is reflected by characteristic changes in the shapes of the action potentials in the ischemic region⁽¹⁾. The local conduction velocity and activation sequence in ventricular wall could be changed by ischemia or MI. In this investigation, we attempt to evaluate the influence of the electrical asynchrony on cardiac contractility of the infarcted LV during systole phase. The analysis is based on an earlier developed electrical heart model⁽¹⁴⁾⁻⁽¹⁵⁾ (E-model) and a mechanical heart

model⁽¹⁶⁾ (M-model). At first, the activation series of the infarcted LV are simulated by the E-model, and then the active force is calculated based on the results from the E-model, finally the cardiac mechanics were calculated by the M-model and verified by other mathematical model results and imaging solutions.

2. Model Description

The computer heart model is a great assistance in the investigation of physiological and pathological properties of human heart. We have ever developed a M-model and an E-model of the human heart. Here we provide a brief description of these two models (for more details see Refs.(14)-(16)), in which the parameter values, are identical to those used in this study).

2.1 Electrical heart model⁽¹⁴⁾⁻⁽¹⁵⁾

Based on the image data of the human body and heart sections, the E-model was reconstructed as a 3-D array of approximately 65 000 cell units spaced 1.5 mm apart. An action potential waveform with variable in duration is assigned to each unit. One layer of Purkinje fibers was sprinkled in the innermost endocardial surfaces of the ventricles, and four simultaneous starting units (the earliest excited units in the ventricles, which assumed to be the junctions at the terminations of the left and right bundles, one in the right ventricle and three in the left) were set in the endocardial surfaces. The right ventricle starting unit was set at the right low septum, and the LV starting units were set in the right low septum, the LV starting units were set at the left midseptum, the left low anterior, and the left posterior, respectively. The distribution of the Purkinje fibers and the four starting units were adjusted repeatedly until the activation sequence of the E-model resembles the pattern reported by Durrer⁽¹⁷⁾. The conducting velocity of the Purkinje fibers was assumed to be five times faster than that of the normal ventricular cells. In the simulation, the action potential waveforms with different duration can be set according to the electrical property of the heart to each unit. The excitation propagation algorithm is a hybrid of the rule-based algorithm and the wavefront-based algorithm. In the simulation, the initial excitation time of all myocardium units is determined first. For each unit, the time elapsed since its excitation is obtained by subtracting the initial excitation time of the unit from the current simulation time. The excitation wavefront of a simulation step is a set of myocardium units that are excited in a certain time interval. The simulation step time is set at the smallest excitation propagation time between an adjacent node pair. It was proved that this propagation algorithm is efficient with reasonable

accuracy.

2.2 Mechanical heart model⁽¹⁶⁾

The 3D M-model of LV developed earlier was based on the geometry of LV, fibrous structure (see Fig. 1, for more details see Ref.(16) and reference therein) and activation series on the ventricular wall. The model was implemented in a in-house finite element package.

The myocardium is a kind of optimal laminated composite and so the theory of laminated composite was used in this model. The myocardium is considered as a composite composed of a number of layers of different directional, fiber-reinforced laminas. At any point in the myocardium the material can be considered as transversely isotropic. This method deals well with the dependence of local LV wall mechanics on myocardial fibrous structure.

In the M-model, 3-D 8 nodes isoparametric element was the basic element, and there were total 760 brick elements, 1 005 nodes and 3 015 degrees of freedom. We developed a special isoparametric method to discretize the LV, whose major feature was that each element can accommodate several layers having different ply orientations. It could provide accurate solutions using less degrees of freedom than conventional FEMs (those methods using a layer as a element), and it saved both in computer memory and computational time (in the order of 10 times).

According to the FE discretization, the active force vectors are unidirectional in a layer. After the activation sequences were determined by E-model, the active forces per element could be determined. It assumed that the muscle fibers generate active force parallel to the fiber direction. The fiber contractile

Fig. 1 The fibrous architecture of the ventricular myocardium. The myocardium is considered as a composite composed of a number of layers of different directional, fiber-reinforced laminas.

behavior was described using the model presented in paper 18 and references therein, which was mainly based on the fiber length and time (the elapsed time since the moment of activation). The velocity-force relation was neglected. The calculation of the active force is in the appendix. Using this M-model, the regional strain, stress and deformation of LV could be calculated.

3. Myocardial Ischemia Simulation

Myocardial ischemia or MI is an imbalance between the supply of oxygenated blood and the oxygen requirements of the myocardium. It is caused by a reduction in the blood supply of the coronary vessels. With insufficient oxygen supplement, the muscles lose their ability to contract. According to the degree of myocardial disease, the condition can be identified as myocardial ischemia, injury, or necrosis. Myocardial ischemia makes activation process slow and it does not effect the depolarization; the electrical activity of the muscle cells in the injury zone is weakened; necrotic tissues have no electrical activity. The asynchrony of electrical activation on the infarcted LV is simulated by the E-model first and then the abnormal mechanical behavior of LV is simulated by the M-model.

3.1 Electrical simulation

The ischemia/MI can change the myocardial action potentials⁽¹⁾. The main changes are decreases in the magnitude of the resting potential and in the action potential duration. Action potentials with prolonged duration have been observed in the infarcted regions of experimental animals after the development of inverted T waves in the surface electrocardiogram (ECG)^{(1),(2)}. Such abnormal action potentials are used in the E-model to study the effects of acute myocardial ischemia and infarction. That is, the evolution of infarctions is simulated by progressively modifying the abnormal action potentials assigned to the injured region.

Before electrical simulation, the model parameters such as myocardial muscle element (healthy element or injured one), action potentials should be set up first. In this simulation, MI is assumed to be in the position encoded with gray-scaled colors in Fig. 2 (a). Figure 2(b) shows the action potentials in different region of the infarcted LV. The infarcted zone may be divided into necrotic tissue, acute ischemia tissue (the corresponding action potential curve is (③-④), and mild one (the corresponding action potential curve is ① and ②).

Thus, the activation sequences of the diseased LV are simulated and shown in Fig. 2(d). In this figure, gray-scaled colors encodes the associated time lag of

(a) The setup of MI elements. In the injured region, the myocardium is divided into necrotic tissue, acute ischemia tissue and mild tissue. Here the layer No. 9 (12 mm from the base, see also Fig. 2 (d)) is shown.

(b) Assumed action potentials in the E-model. The injured degree of the muscle become more severe from the action potential curve ① to ④, and curve ⑤ is the prolonged action potential curve caused by ischemia (adapted from Miller's results⁽¹⁾).

(c) The activation sequence on normal LV and right ventricle

(d) The activation sequence on the infarcted LV and right ventricle. Notice the asynchrony of the electrical activation after MI.

Fig. 2 The simulation of MI based the electrical model (E-model)

electrical excitation. Figure 2(c) depicts the activation sequences on the normal heart, the difference between them is obvious, especially near the injured region.

3.2 Mechanical simulation

When the active forces are determined based on the electrical simulation, the stress, strain and displacement during systole could be calculated. Myocardial diseases such as MI may change the stiffness of myocardium, activation sequences and local contraction forces. Some assumptions for these changes have to be introduced when considering MI. It was realized by adjusting the model parameters

according to some data or ideas from references^{(8),(9),(11),(19)}. The model editor (a Man-Machine interface which is easy for the set up the model parameters and visualization⁽²⁰⁾) can deal with three kinds of parameters: myocardial fiber type (normal and diseased muscle) and position, material parameters (elastic modulus) and external loads (active force, cardiovascular loads, etc.). Equation (2) in the appendix depicts the alternation of the active force, w is the MI factor, in the boundary zone of the MI region w varies between 1.0 (healthy situation) and 0.0⁽¹¹⁾. In the remaining part of the MI region it takes the value 0.0. In the systolic period, the elastic

modulus of the diseased zone is set to be several times larger than that of normal zone in this M-model since it is usually found that the chronic infarcts are relatively stiff^{(8),(9),(19)}.

Once model parameters are determined, the simulation can be started. In the mechanical simulation, the time incremental step of simulation is 7 ms, and the activation period $T_e=353$ ms.

4. Results and Discussion

4.1 Deformation

The pumping function of the heart is characterized by a highly temporally ordered periodic change of the 3-D spatial geometry of the heart's chambers. Some ventricular function that can be measured is the relative timing of the events in contraction. A movie sequence of the LV during systole is shown in Fig. 3. The deformation is taken over three time sequences ($T_1 < T_2 < T_3$, which comes near end-diastole, middle-systole and end-systole, respectively). It shows that the contraction procedure of LV is complicated, it includes axial shortening, radial contraction and twisting at different degrees. The deformations near the infarcted zone are relatively unpredictable than those near the healthy region. The diseased zone suffered high pressure and the action of adjacent tissues can bulge outside of the LV wall. This typical deformation, showing a bulging of the ischemic region, has also been found in experiments⁽²¹⁾. The reduction of mechanical function in the border zone may be due to "constrained" motion of the border zone. It is found that although the MI deteriorated the deformation of the wall, the estimation of the size and location of the ischemic region from single deformation pattern of the LV is not straightforward and so a time series motion should be studied. The simulation also predicts that the local contraction patterns are considerably more sensitive to abnormal conduction than measures of global cardiac function.

Fig. 3 A movie sequence of the LV during systole. The deformation is taken over three time sequences ($T_1 < T_2 < T_3$, which comes near end-diastole, middle-systole and end-systole, respectively).

The wall-motion abnormality of the diseased LV has also been detected in the Cardiac-tagged MRI⁽²²⁾. Using tagging technique, it was found that the first indication that there is low blood flow to the heart is the onset of wall-motion abnormality described above. It was found that the mechanical activation times correlated linearly with the electrical activation⁽²⁴⁾, therefore it is essential to consider electro-mechanical coupling problem when studying ventricular mechanics.

In addition, comparison of our simulation solutions and the deformation results of LV obtained from tagged MRI⁽²²⁾ showed that the displacement field obtained from our model of normal LV was similar with the result of medical image analysis. The amplitude and direction of major axis contraction, minor axis contraction, heart wall distortion, and the variation tendency of anti-clock twisting angle are roughly congruent⁽¹⁶⁾. It also showed that local wall mechanics were strongly dependent on the transmural variation in muscle fiber orientation.

4.2 Stress field

In this section, the principal stress is only considered and its distribution relates to the activation sequences. When the MI disturbs the electrical excitation regionally in the ventricular wall, the time lag of active force is changed compared with the normal one. From Fig. 4, it is easy to find that the maximum principal stress near the diseased tissue is larger than that of normal tissue. The predicted peak stress in the infarcted LV is compressive and has a magnitude about eight times that in the normal LV. Since different models used different parameters, the predicted stress values differed from each other, but all the models concluded that the stress values in the diseased zone were greater than those far away from the infarct zone^{(8),(9),(12)}. Some therapy aimed to alter

Fig. 4 The maximum principal stress distribution in the middle-layer (in radial direction) of LV wall with MI. Notice the large value near the injured region.

local stress distributions may be tried in clinical application.

Some investigation indicated that myocardial wall stress might be one of the primary determinants of blood flow in coronary artery⁽¹³⁾. The difference of the stress distribution between the endocardium and the epicardium may make the muscles on the inner wall more sensitive to insufficiency of oxygenated blood. This may be the reason why the inner wall of LV tends to suffer injury first. Moreover, in the border zone surrounding the MI, systolic stresses are elevated. This zone has the potential of progressing toward infarction.

4.3 Strain distribution

Figure 5 shows the maximum principal strain (left panels) and minimum principal strain (right panels) for the epicardial nodes. In this figure, the lengths and directions of the line segments describe the deformation magnitudes and directions respectively. In the figure, the upper two panels are from the front view, the lower two are from the view of apex of LV. The diseased zone is located at the left side of LV in the above panel and left corner in the lower panel.

Strain is a quantity of interest because it can be used to quantify the amount of tissue contraction. In the normal zone, our simulation indicates that the maximum principal strain is in the radial thickening direction (positive), and the minimum one is roughly in the fiber direction (negative). These results are

Fig. 5 The strain on the epicardium of diseased LV. In the normal zone, the maximum principal strain is in the radial thickening direction (positive), and the minimum one is roughly in the fiber direction (negative). Notice that in the abnormal region (left corner), the minimum principal strain is in the radial direction, indicating that the heart wall is being compressed rather than thickened.

similar to those from tagged MRI^{(22),(23)}, and it coincides with the LV physiological phenomena of radial thickening and shortening of fibers during the systole. Notice that in the abnormal region, the minimum principal strain is in the radial direction, indicating that the heart wall is being compressed rather than thickened. For comparison, the minimum principal strain of nodes on the epicardium of normal LV is also given (see Fig. 6).

Figure 7 (a) shows the evolution of strain in the ischemic zone for three patients with inferior ischemia, the data was obtained by tagged MRI⁽²⁵⁾. Figure 7 (b) is the strain-time curve from simulation. Figure 8 shows the normalized wall thickening (WT) data from some experiment and Larry's mathematical model data (Fig. 8 (a) adapted from paper 9 and references therein) and the simulated results (Fig. 8 (b)). From these two pictures, it is obvious that the model could predict the evolution of strain throughout the diseased heart during systole. The discrepancy may be due to model parameters such as the position, size of the ischemic region. These influences of the asynchrony on the strain were also found in some other experiments⁽²⁾.

The muscle shortening and wall thickening relate not only to the contraction ability of muscle itself but also local loads and stress distribution^{(8),(26)}. The muscle tissues in the MI zone sustain the normal pressure and under increased local wall stress. This enlarges the impedance to coronary blood flow. The insufficiency of blood supplement may exacerbate the muscle contraction dysfunction.

Finally, the regional mechanics of the infarcted LV is calculated without considering the activation asynchrony after MI. These calculation shows that the difference between these two data (those considering the asynchrony and those without considering asynchrony) could be up to 25% in the disease zone but no much difference in the healthy zone. So in the mechanical modeling of the infarcted LV, more attention should be paid to the electrical asynchrony problem and so the introduction of the E-model is needed.

Fig. 6 The minimum principal strain of the nodes on the epicardium of the normal LV

(a)

(b)

Fig. 7 The evolution of strain of the infarcted LV during systole phase. Figure (a) shows the evolution of strain in the ischemic zone for three patients with inferior ischemia, the data was obtained by tagged MRI⁽²⁵⁾. Figure (b) is the normalized strain-time curve from simulation.

All in all, the regional mechanical properties are rather heterogeneous across the wall because of time and spatial difference in the moment of activation after MI. With the activation asynchrony, the early-activated fibers are relatively short and unable to shorten substantially further, whereas the late-activated parts are able to shorten considerably since they have relatively large initial length⁽¹²⁾. Hence the heterogeneity of the cardiac mechanics is obvious after MI. The stress and strain pattern study is helpful for quantitative diagnosis of MI or other heart diseases. This method will be of advantage in assessing clinically relevant manifestations of heart diseases such as MI.

4.4 Model limitation

Many aspects of ventricular mechanics are incorporated in the present M-model: the 3D ventricular

(a)

(b)

Fig. 8 The normalized wall thickening (WT) data at the end of systole. Figure (a) shows the data from the experimental data and Larry's model data (paper 9 and references therein). Figure (b) is the WT data from simulation.

geometry, the varying muscle fiber orientation across the wall, the anisotropy of the passive myocardial tissue, and the dependency of the activation sequence in the wall. Obviously, not all-physiological aspects were taken into account. For example, model simplifications concerning material properties and constitutive equations were introduced. This simplification is motivated by the fact that the stresses associated with these properties are relatively small compared with the active fiber stresses during systole⁽¹²⁾. But in diastole phase, these simplifications could introduce large error. Focus is on the electromechanical coupling since it is one of the dominant factors determining the ventricular mechanics. The entire mechanical model feature (advantage and disadvantage) was discussed in detail in the previous paper⁽¹⁶⁾. Ongoing and future work includes adopting more sophisticated continuum biomechanical models,

such as those of VanCampan's model⁽¹¹⁾. The spatial resolution should be enhanced later.

In the view of electrical heart model, a number of 3-D models of ventricular electrophysiology have already been developed. With these models it is possible to simulate the spread of excitation within the ventricles and to compute an equivalent body-surface electrocardiogram^{(20),(27)}. In our E-model, the algorithm of the excitation process of the heart is isotropic, and anisotropic property should be considered in further simulation later⁽²⁰⁾.

The cardiac cycle is initiated by an electrical stimulus resulting in fiber shortening, causing contraction, known as excitation-contraction coupling. Most of the detailed information lies at the single cell level. The interactions between electrical conduction and mechanical function in the heart is mostly unknown so 3-D models of cardiac electromechanical coupling or suitable experimental approaches for studying these interactions in the whole heart are needed⁽⁵⁾. It is not yet practical to implement a fully comprehensive model that includes all known electromechanical interactions; Here preliminary integrate models are used to study specific interactions. More sophisticated coupled models of the heart could be used to simulate complex interactions between electrophysiology, cardiac contraction, haemodynamics, etc. To illustrate the potential of this approach, we have simulated electrically-induced changes in cardiac mechanics. This preliminary simulation can quantify the electrical and mechanical status of LV, e.g., quantifying the regional myocardial stress and strain field to detect myocardial mechanical difference to provide information in diagnosis.

5. Conclusion

The regional mechanics of the infarcted LV during systole phase is studied using a mechanical heart model and an electrical heart model. The investigation focuses on the influence of electrical asynchrony on ventricular wall stress, strain and deformation. The preliminary simulation results show considerable disruption of the contraction pattern near the diseased zone, the minimum principal strains in local regions with MI were significantly smaller compared with those in corresponding regions of the normal hearts, while the stress is larger than normal tissues. So asynchronous electrical activation after MI deteriorates the regional mechanical performance of LV. This simulation suggests that such electromechanical model provides a way to investigate the biomechanical function of the LV with diseases such as MI, and more realistic models of cardiac function are important for clinical evaluation of heart disease.

Appendix

In the fiber-coordinate system (one axis is chosen to coincide with the local muscle fiber direction, another one is determined by the epicardium surface normal vector.) in Fig. 1, the expression of the active force along the fiber is:

$$\sigma'_e = \begin{cases} \sigma'_0 \sin\left(\frac{t-\tau}{T_e} \pi\right), & 0 \leq t \leq T_e \\ 0, & t > T_e \end{cases} \quad (1)$$

Here, t is the time; τ is the time lag of active stress, T_e is the activation period. σ'_0 is the active force of a myocardial fiber and which is a function of fiber length, time t , fiber directions, etc.

In the calculation, the stress and strain vector is expressed with respect to two coordinate frame of reference: global coordinate system X - Y - Z and local coordinate system ξ - η - ζ . The nodal force equivalent to the active contractile force is calculated based on Eq.(1) and the isoparametric transformation:

$$\{F_f\}^e = -w \sum_{l=1}^{L_e} \int_{\xi^{l-1}}^{\xi^l} \int_{\eta=-1}^1 \int_{\zeta=-1}^1 [B]^T \times T \{0, 0, \sigma'_e, 0, 0, 0\}^T |J| d\xi d\eta d\zeta \quad (2)$$

Here, $[B]$ is geometric matrix of element, l , L_e stand for the number and total number of layers in an element respectively, T is the transformation matrix between the fiber coordinate and global coordinate. $\{0, 0, \sigma'_e, 0, 0, 0\}^T$ is the active force row vector in the fiber-coordinate system, $|J|$ is the determinant of the Jacobian matrix. ξ , η , ζ are the local coordinate system with the magnitudes ranging from -1 to 1 ; ξ^l and ξ^{l-1} represents individually the coordinate values along ξ direction on the front-side and back-side of layer l in an element. When $l=1$, $\xi^{l-1}=-1$, when $l=L_e$, $\xi^l=1$. w is a MI factor. It represents the ability of the tissue to generate active force and varies between 0 (completely ischemic tissue) and 1 (normally contracting tissue). The velocity-stress relation is neglected here. Moreover, the cardiovascular loads, blood pressure is distributed load which is perpendicular to the endocardium, its equivalent nodal force can be expressed as integral form with intensity of pressure perpendicular to ξ in the local coordinate system.

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