# Analysis of Elasticity Image of Chronic Hepatitis Based on Dynamic Model of Fibrosis Progression

肝線維化の力学的モデルに基づく肝エラストグラフィの解析

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## 1. Introduction

Precise evaluation of the stage of chronic hepatitis C with respect to fibrosis has become an important issue to prevent the occurrence of cirrhosis and to initiate appropriate therapeutic intervention. Real-time elastography enables us to evaluate the stiffness of tissue and is a new method for noninvasive staging of liver fibrosis combing of some features from elasticity image. We developed a dynamic model of fibrosis progression in order to quantitatively evaluate the relation between these values and the progression. This paper presents a model and the analysis of an elasticity image based on it.

## 2. Method

## 2. 1. Modeling of Fibrosis Progression

The human liver is composed of many hexagonal structures termed liver lobules and containing a central vein. Fibrosis progression is accompanied by changes of tissue structure. When fibrosis grows, the lobules are destroyed and replaced by regenerative nodules.

We can model the liver tissue structure using the potential distribution <sup>1)</sup>. With this model, we can monitor and determine the change of liver structure. To prepare the model, we first scattered the central points of the potential distribution corresponding to the central veins and then located the potential distribution around each central point as in eq. (1).

$$p(r) = p_{\max} \sin\left(\frac{\pi}{2}k^{r}\right) \tag{1}$$

In eq.(1),  $p_{\text{max}}$  is the maximal value and k is the form parameter that determines the distribution defined as the value of 0.8 to 1.2 randomly as an

spread. The parameter  $p_{\text{max}}$  of all the potentials is initial condition and other parameters are set to experimentally estimated values as discussed in <sup>1</sup>).

We simulated the fibrosis progress by combining the potentials and making the nodules and fibers as fibrosis progresses; we assumed that the stage of the model is determined by the number of central points. F0 is the initial state; F1, F2, F3, and F4 are 1/2, 1/4, 1/8, and 1/16 of the initial central number, respectively.

## 2. 2. Young's modulus distribution

To simulate tissue deformation, values of Young's modulus were assigned to this model composed of parenchyma and fibers. We then assigned Young's modulus [kPa] to the parenchyma based on eq. (2) and allocated it to the fibers based on eq. (3).

$$E_0 = p_{\text{max}} - \bar{p}_{\text{max,normal}} + E_{\text{normal}}$$
(2)  
$$E_1 = 2p_{\text{max}} - \bar{p}_{\text{max,normal}} + E_{\text{normal}}$$
(3)

In eq.(2) and eq.(3),  $\bar{p}_{max,normal}$  is the average of the maximal value of each potential for a normal liver model, and  $E_{normal}$  is the average Young's modulus on parenchyma of a normal liver model. We established the equations, coefficients, and  $E_{normal}$  of 4 [kPa] with reference to practically measured Young's moduli<sup>2)</sup>.

## 2.3. Strain distribution

The tissue deformation caused by compression is simulated by applying the finite element method (FEM) to the tissue model. The Young's modulus distribution model was compressed from upper with pressure of 50kPa (about 1%) and each scatterer

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Fig. 2 Young's modulus distribution assigned to deformation model



Fig.3 Strain distribution images reconstructed using simulated RF signals

was moved assuming the two-dimensional stress state. Displacement along axial direction was calculated by comparison of two frame data before and after compression. Then, the strain is calculated by differential processing of the displacement.

In addition, strain distribution was obtained by the same way as ultrasound equipment. RF signals were generated from the distribution of scatterers. Then, the strain distribution is calculated by combined autocorrelation method (CAM), which we have developed<sup>3)</sup> and applied to ultrasound equipment(the Real-time Tissue Elastography, Hitachi Aloka Medical Ltd). Finally, we created the color images of this strain distribution (**Fig.3**)

## 3. Results

Figure 1 illustrates the fibrous structure of normal and chronic hepatitis obtained based on the fibrous progression model. Two-dimensional model was applied and 2538 central points were distributed within area (40mmx40mm). The number of iteration does not indicate the physical characteristic of the liver directly, but is selected to only determine the size of nodules. Fig 1 (a) presents the normal liver, and (b) and (c) depict the liver tissue when fibrosis progresses and the number of the central points has decreased to 1/4 and 1/16 compared to the normal state. We can



Fig. 4 The relationship between the stage of fibrosis and the mean strain of each stage.

observe that the fiber area increases as the disease progresses.

**Figure 2** illustrates the Young's modulus distribution, and Figs. 2 (a) through (c) are Young's moduli assigned to the models in Figs. 1 (a) through (c). As fibrosis progresses, the maximum potential becomes higher, so Young's modulus increases accordingly due to eqs. (2) and (3).

Figures 3 (a) through (c) present strain distribution images obtained by using the CAM. As fibrosis progresses, the blue area increases, indicating that the area has become stiffer than the area around it. In addition, the strain distribution becomes more and more complex. Figure 4 presents the average strain of each stage. We can see that the average strain decreases as the stage progresses.

#### 4. Conclusion

This study proposed a dynamic model of fibrosis progression. From the strain distribution, we could see that the area of low strain increases, the average strain decreases, and the strain distribution becomes more and more complex as fibrosis progresses. These changes are actually seen in elastography. In the future, we will examine the relationship between the models and the clinical data and confirm whether the models are correct. We will then need to establish new staging using a dynamic model so that we can quantitatively diagnose the fibrosis progression.

#### Acknowledgment

This work was supported by Grant-in-Aid for Science Research[22103507], MEXT, Japan.

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