Consideration of mechanical filtering system of the Meissner corpuscle

Shinobu Kuroki[1], Hiroyuki Kajimoto[2], Takaaki Nara[3], Naoki Kawakami[1] and Susumu Tachi[1]

[1]Graduate School of Information Science and Technology, The University of Tokyo, Tokyo, Japan
(Tel : +81-3-5841-6917; E-mail: Shinobu Kuroki@ipc.i.u-tokyo.ac.jp, kawakami, tachi@star.t.u-tokyo.ac.jp)
[2]Department of Human Communication, The University of Electro-Communications, Tokyo, Japan
(Tel : +81-42-443-5445; E-mail: kajimoto@hc.uec.ac.jp)
[3]Department of Mechanical Engineering and Intelligent Systems, The University of Electro-Communications, Tokyo, Japan
(Tel : +81-42-484-3327; E-mail: nara@mce.uec.ac.jp)

Abstract: We are researching the natural timing of mechanoreceptor activity with the aim of developing improved artificial tactile presentation systems. In order to develop strategies for manufacturing noninvasive devices, we focused on peripheral level. We have presented a hypothesis on the energy conversion system of the Meissner corpuscle, a type of mechanoreceptor. It was proposed that these corpuscles encode the normal strain, especially by detecting the horizontal normal strain. We also observed an experimental evidence consistent with our hypothesis. In this study, we focused on the virtual stiffness of the mechanoreceptors and developed a motion model to verify why the horizontal stretching preferentially induces activity in the Meissner corpuscle.

Keywords: Mechanoreceptor, strain, nerve activity, human information processing

1. INTRODUCTION

To provide natural sensation by artificial means, many kinds of displays were developed to date. One of the most important point of the design of devices and signals is spatiotemporal densities. In tactile fields, this kind of problem is not quite as simple as those in other senses. Here, all we know is two-point discrimination threshold. The problem is that we don't even know the requirements of designing; the parameters of the major proportions of tactile informations cannot be determined yet. As a result, we were unable to demonstrate sensations clearly. Consequently, ad hoc stimulations have been used for the tactile devices. To somehow break through this situation, we intend to figure out the human tactile perceptual mechanisms partially.

To make noninvasive devices, we focused on peripheral level. When you touch an object with your finger, the skin surface is deformed, the mechanoreceptor deformation is followed, and then nerve activities arise. By using a simulation of dynamics of elasticity, the prediction of elastic transfer property from skin to mechanoreceptor already has a certain accuracy. The left-behind subject is the relation between mechanoreceptor modification and nerve activity.

We have been proposing an explanation for the energy conversion system of Meissner corpuscles which exist in the superficial layer of the skin as shown in Fig. 1. The response of this receptor is supposed to be important especially for active touch. Our proposed explanation was experimentally-supported in our previous work. In this paper, we tried to describe this explanation i.e. the relation between the skin modification and a nerve activity of Meissner corpuscle by using a spring model. This discussion will help us determine an effective strategy for signal design of tactile displays.

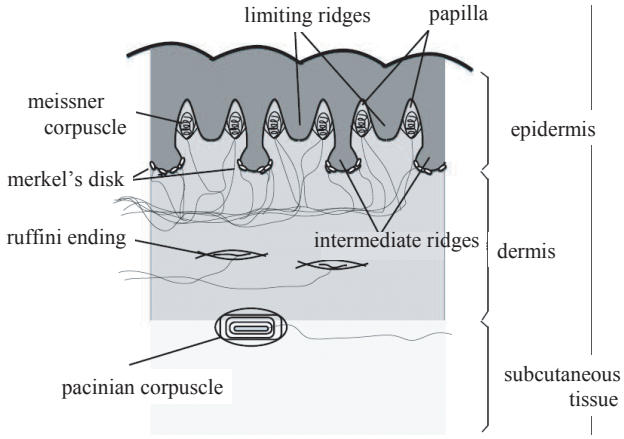

Fig. 1 pattern diagrams of the finger skin (reconstructed from [1])

2. RELATED RESEARCH

Tactile sensations are generated by mechanoreceptor units. Mechanoreceptors are classified into four groups based on their receptive fields and speeds of adaptation. Meissner corpuscles are known to have small receptive fields and to be rapidly adapting mechanoreceptors. It is well known that they have a relatively low characteristic frequency of around 40 Hz, localized in the papillary dermis with lengths of around 150 μm and diameters of 40-70 μm.

Many electrophysiological experiments involving the application of vibration to a fingertip have been conducted [2]. Fig.2 shows the RA nerve activity recorded in the arm on the application of a sinewave vibration to a

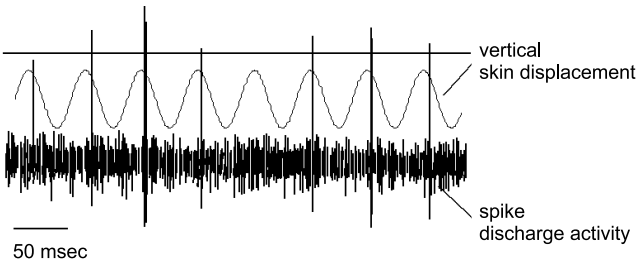

Fig. 2 Nerve activity of a single RA skin afferent fiber to mechanical vibration (reconstructed from [2])

fingertip. The nerve activity is synchronized with the mechanical vibration. It occurs in a specific phase with one or two pulses per cycle of the applied vibration. Since the interval of the nerve activity is sufficiently long, it is considered that this synchronization is not caused by a refractory period. This receptor is offset-compensated receptor, so this finding suggests that Meissner corpuscles must have some mechanical filtering system. This suggestion is a good basis for analyzing the nerve activation timing of Meissner corpuscles.

3. HYPOTHESIS

We reviewed the structure of the Meissner corpuscle and presented a hypothesis on the strength of two structural reasons [3].

3.1 Axonal structure

Generally, when using the finite element method, mechanoreceptors are approximated to encode the strain energy density of the skin. This approximation is reasonable if the receptor is an ideal elastic body with axons that spread omnidirectionally such that they become equally sensitive in all directions. However, observations have revealed that some part of an axon runs tangential to the skin surface, most of it is regarded to run parallel to the skin surface. We consider that if nerves encode the line integral of the strain, horizontal stretching should preferentially generate nerve activity.

3.2 Periaxonal structure

Iwanaga et al. reported that each corpuscle is linked with the basal aspect of the epidermis by dermal collagen fibers[4]. Their findings indicated a deformation in the epidermis is followed by a distortion of the dermal collagen fibers, which continue into the corpuscles. With existence of these fibers, skin can expand and contact nonlinearly. Motivated by Iwanaga's findings and her hypothesis of Meissner corpuscles, we proposed a detection mechanism, which is illustrated in Fig. 3. (The top and middle diagrams are based on Iwanaga's hypothesis.) If the skin surface is pushed in or if it is stretched from its basal phase (middle diagram), the epidermal basement membrane under the stimulated point will open and the Meissner corpuscles will be extended along their minor axes. As a result, nerve axons are stretched, which leads to nerve activity. In contrast, when some part of the skin is pulled or shrunk (lower diagram), the collagen fibrils bend, and as a result, the deformation of the skin surface will not reach the Meissner corpuscles directly. Sripati et al. recorded nerve activity with 15 can-

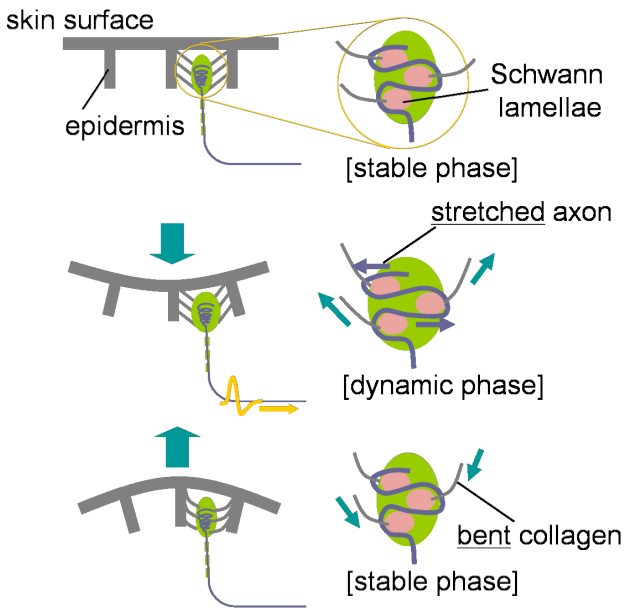

Fig. 3 Proposed nerve activity mechanism of the Meissner corpuscle

didate variables for the relevant receptor deformation [5]. They concluded that maximum tensile strain likely drives SAI mechanotransduction, whereas changes in receptor surface area drive RA transduction. However, we propose that Meissner corpuscles encode the normal strain; that is, tensile strain likely drives RA transconduction. Still, their finding that the physical quantities closely related to local membrane stretching were most predictive of the observed afferent responses is consistent with ours. According to this hypothesis, nerve activity does not occur when the skin surface is shrunk, but when the skin is stretched. Thus, it is possible to realize nerve activation in a specific phase that is synchronized with mechanical vibration.

With these two reasons, we proposed the following hypothesis: Meissner corpuscles encode the normal strain, particularly detecting the horizontal normal strain. To verify the proposed detection mechanism for the Meissner corpuscle, we took experimental approach and reached a positive conclusion. In next section we developed a motion model to verify why horizontal stretching preferentially induces activity in the Meissner corpuscle.

4. MODEL

Here, we use a spring model to describe the stretching mechanism of an axon. In order to discuss the significance of collagen fibrils in this mechanism, we describe the relation between the deformation of the finger skin and the stretching of an axon. We attempt to model the nonlinearity between the direction of the ex-

ternal force and the structural stiffness of an elastic body. Consider how a small volume under only a vibrator deforms when perpendicular vibrations are introduced in an elastic body. For the sake of simplicity, we consider an elastic truss in two-dimensional space. Small rotations and small strains are assumed.

The Meissner corpuscle, dermal layer, epidermal layer as a bridge girder, and collagen fibril are partially - modeled. Consider the cell as an existence region of the Meissner corpuscle. In a cell that is surrounded by the epidermal layer as a bridge girder, the collagen fibrils link the corpuscle and epidermis, as shown in Fig. 4. This cell

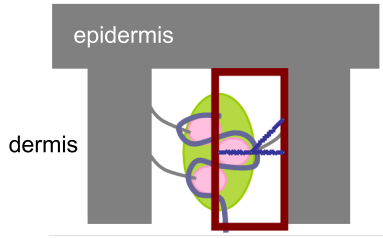

Fig. 4 Modeled cell region (surrounded by red box)

elongates and contracts with the deformation of the skin surface. In this section, the changing of the virtual stiffness of this cell is predicted. There are three components in the cell,-the Meissner corpuscle and dermal layer have a relatively soft stiffness;, while the collagen fibril has a hard stiffness. Naturally, these materials stiffnesses do not change; however, the virtual stiffness of the entire cell structure can change. We predict that the collagen fibril has two modes, namely, rotation and translation, and that the virtual stiffness changes due to the switching between these modes.

4.1 Three-element model

Consider a plane three-bar truss with the geometry shown in Fig. 5. This model comprises three elements in four nodes. Elements A, B, and C are springs represent-

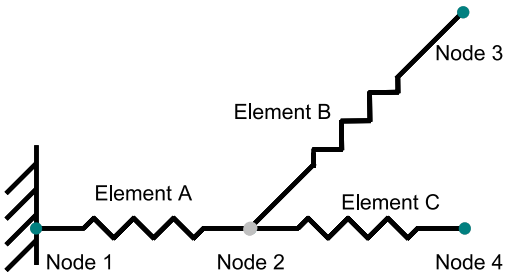

Fig. 5 Three-element model

ing the axon terminus, collagen fibril, and dermal layer, respectively. The X-and Y-axes are considered to be parallel and perpendicular to the skin surface, respectively. We extend this model by U along the X-axis and calculate the stretching of element A. In other words, node 1 as the center of the Meissner corpuscle is a fixed end while nodes 3 and 4 as the epidermal basement membrane (i.e., at the boundary of the dermis and the epidermis) are displaced along the X-axis only.

$$(u_1, v_1) = (0, 0) \tag{1}$$
$$(u_3, v_3) = (U + \delta U, 0) \tag{2}$$
$$(u_4, v_4) = (U, 0) \tag{3}$$

Here, u and v represent the displacement of each node along the X- and Y- axes, respectively. We set different displacements for nodes 3 and 4. This displacement describes the elastic body depth difference between these two nodes. Node 3 is shallower than node 4; therefore, it will be stretched horizontally to a greater extent by a vertical force.

In this model, node 2 is not externally forced.

$$(p_2, q_2) = (0, 0) \tag{4}$$

Assume $U > 0$, considering a horizontally stretched dermal papilla. Set the default positions of elements A and C to the horizontal, with element B angled at θ_b. Applying Eqs. (1),(2),(3), and (4) to the equilibrium equations of node 2, the displacement of node 2 (u_2, v_2) is given by

$$u_2 = \frac{1}{2}U \tag{5}$$
$$v_2 = \frac{(U + 2\delta U)\cos\theta_b}{2\sin\theta_b} \tag{6}$$

When nodes 3 and 4 are moved in the positive direction, node 2 exhibits a positive displacement along both axes. Note that $u_2 < U$. This shows that both elements A and B rotate so that θ_a asymptotically approaches θ_b. As a result, the corpuscle (element A) runs parallel to the collagen (element B). Conversely, when adding

$$(u_3, v_3) = (-U - \delta U, 0) \tag{7}$$
$$(u_4, v_4) = (-U, 0) \tag{8}$$

we anticipate symmetrical results.

$$u_2 = -\frac{1}{2}U \tag{9}$$
$$v_2 = \frac{(-U - 2\delta U)\cos\theta_b}{2\sin\theta_b} \tag{10}$$

It shows that both elements A and B rotate such that they are orthogonal to each other. As a result, the corpuscle (element A) is at right angles to the collagen (element B).

In the following truss analysis, only small rotations and small strains are assumed. The next step is to consider element A as an angled bar. We vary the angle and check the resulting effect. Here, we set the default position of element C to the horizontal, and elements A and B are angled at θ_a and θ_b to the horizontal. The displacement of node 2 (u_2, v_2) is given by the equilibrium equa-

tion.

$$u_2 = \frac{K\left(s_b^2 + s_a^2 + s_{b-a}^2\right) + 2ks_a^2}{2K\left(s_b^2 + s_{b-a}^2\right) + 2ks_a^2}U + \frac{K\left(-s_b^2 + s_a^2 + s_{b-a}^2\right)}{2K\left(s_b^2 + s_{b-a}^2\right) + 2ks_a^2}\delta U \quad (11)$$

$$v_2 = \frac{K\left(s_b c_b - s_a c_a + s_{b-a} c_{b-a}\right) - 2ks_a c_a}{2K\left(s_b^2 + s_{b-a}^2\right) + 2ks_a^2}U + \frac{K\left(3s_b c_b - s_a c_a + s_{b-a} c_{b-a}\right)}{2K\left(s_b^2 + s_{b-a}^2\right) + 2ks_a^2}\delta U \quad (12)$$

where c_a and s_a represent $\cos a$ and $\sin a$, $(k_a, k_b, k_c) = (k, K, k)$, and $k < K$. Here, elements A and C have the same spring constant and the fibril has sufficient stiffness.

First, we assume that θ_a is small. We set the approximate values of $\cos a \cdot \sin a = 1$ and $\sin^2 a = 0$ in the equilibrium equation.

$$u_2 = \frac{1}{2}U - \frac{s_b^2 - s_{b-a}^2}{2\left(s_b^2 + s_{b-a}^2\right)}\delta U \quad (13)$$

$$v_2 = \frac{K\left(s_{2b} - s_a + s_{2(b-a)}\right) - 2ks_a}{2K\left(s_b^2 + s_{b-a}^2\right)}U + \frac{3s_{2b} - s_a + s_{2(b-a)}}{2\left(s_b^2 + s_{b-a}^2\right)}\delta U \quad (14)$$

Here, $v_2 > 0$ because $K \gg k$, and $u_2 < U$ so that θ_a approaches θ_b. At the default position, the corpuscle (element A) is parallel to the collagen (element B). By inputting $-U$ to this expression, symmetrical results can be calculated.

Second, consider the situation where θ_a is nearly equal to θ_b. We set the approximate values of $\cos(b-a) = 1$ and $\sin^2(b-a) = 0$ in the equilibrium equation.

$$u_2 = \frac{K\left(s_b^2 + s_a^2\right) + 2ks_a^2}{2Ks_b^2 + 2ks_a^2}U - \frac{K\left(s_b^2 - s_a^2\right)}{2Ks_b^2 + 2ks_a^2}\delta U \quad (15)$$

$$v_2 = \frac{K\left(s_{2b} - s_{2a} + s_{b-a}\right) - 2ks_{2a}}{2K\left(s_b^2 + s_{b-a}^2\right) + 2ks_a^2}U + \frac{K\left(3s_{2b} - s_{2a} + s_{b-a}\right)}{2Ks_b^2 + 2ks_a^2}\delta U \quad (16)$$

In this equation, v_2 becomes negative when θ_a becomes larger than θ_b. As a result, θ_a becomes smaller and approaches θ_b again.

These equations indicate that when $U > 0$ is added to this cell, θ_a approximates θ_b and the mode of the collagen fibril switches from rotation to translation. Next, we attempt to describe an axon elongation with this model and verify the effectiveness of these mode conversions. If an axon elongation δL is much smaller than the natural length of an axon (i.e.; element A) L, it can be described as such

$$\begin{aligned}\delta L =& \sqrt{(L+u_2)^2 + v_2^2} - L \\ =& L\left\{\sqrt{\left(1+\frac{u_2}{L}\right)^2 + \left(\frac{v_2}{L}\right)^2} - 1\right\} \\ \doteq& L\left\{\sqrt{1+\frac{2u_2}{L}} - 1\right\} \\ \doteq& L\left\{1 + \frac{u_2}{L} - 1\right\} \\ =& u_2 \end{aligned} \quad (17)$$

Among the axon elongation characteristics that one may consider, a controversial characteristic is the coefficient value of u_2. Therefore, we restrict the argument to the coefficient value of u_2. Here, we focused on the major displacement U's coefficient value. From Eq.(11), it can be written as

$$\begin{aligned} u_2 =& \frac{K\left(s_b^2 + s_a^2 + s_{b-a}^2\right) + 2ks_a^2}{2K\left(s_b^2 + s_{b-a}^2\right) + 2ks_a^2}U + \alpha\delta U \\ =& \{1 + \frac{s_a^2 - s_b^2 - s_{b-a}^2}{2\left(s_b^2 + s_{b-a}^2\right) + \frac{2k}{K}s_a^2}\}U + \alpha\delta U \end{aligned} \quad (18)$$

When adding $U > 0$, θ_a asymptotically approaches θ_b so that s_a increase while s_b and s_{b-a} decrease. Assume that K is sufficiently larger than k, then the denominator of the first term in Eq.(18) decreases. On the other hand, considering the numerator of the first term in Eq.(18), $s_a^2 - s_b^2 - s_{b-a}^2$ increases. Note that θ_b must be smaller than $\frac{\pi}{2}$ and it must decrease, while θ_a increases from zero. Because the rate of increase of s_a^2 is larger than that of $-s_{b-a}^2$, $s_a^2 - s_b^2 + s_{b-a}^2$ increases on the addition of $U > 0$. Therefore, the coefficient value of u_2 now increases monotonically when adding $U > 0$, and θ_a asymptotically approaches θ_b. With this tendency, the horizontal displacement U is converted to u_2 efficiently on the addition of $U > 0$ as opposed to the addition of $U < 0$.

If there is no fibril in the skin-mechanoreceptor system, node 2 would not be displaced along the Y-axis. With input $(u_4, v_4) = (U, 0)$,

$$u_2 = \frac{U}{2} \quad (19)$$

$$v_2 = 0 \quad (20)$$

This u_2 without collagen is smaller than u_2 with collagen.

5. DISCUSSION

After the skin surface has been pushed, the axon endings, which were horizontal by default, run parallel to the angled collagen fibril. It was shown that this tendency is not disrupted by an additional displacement. Conversely, after the skin surface has been pulled, the axon endings

run at right angles to the collagen fibril. It was shown that the coefficient value of axon elongation monotonically increases on the addition of a horizontal stretch deformation. Since the collagen fibril has two modes, namely, rotation and translation, the virtual stiffness changes due to the switching between these modes. The elongation increases on stretching and decreases on shrinking. Pushing deformation results in direct stretching from the epidermis to the mechanoreceptor. With this tendency, an axon that is sufficiently softer than the collagen fibrils will distend suitably when stretched horizontally. As a result, an external force is converted to axon elongation efficiently on pushing as opposed to pulling. This effect leads to a nonlinearity in the displacement magnitude and horizontal elasticity of the mechanoreceptor.

The axon endings are folded along the minor axis. This changing of the virtual stiffness corresponds to a lengthening of the minor axis of the mechanoreceptor, i.e., when calculating the strain of the axon not relative to the length of the mechanoreceptor minor axis but relative to the size of the dermal papilla on stretching. In other words, the integral strain along the width of the axon increases when it is stretched horizontally.

6. CONCLUSION

The objective of this study was to determine the mechanisms behind human tactile perception. We focused on the transitive problem and attempted to describe the timing of nerve activity. In this study, we verified a mechanical filtering system for Meissner corpuscles by using a structural model.

Based on a previous research, we considered that the Meissner corpuscles tend to stretch in the horizontal direction, and proposed the following hypothesis: Meissner corpuscles encode the normal strain, especially by detecting the horizontal normal strain.

To explain our hypothesis, we predict the changing of the virtual stiffness of a cell, which is existence region of the Meissner corpuscle. In order to describe the relation between the deformation of the finger skin and the stretching of an axon, we created a spring model. Although the materials stiffnesses do not change, the virtual stiffness of the entire cell structure can be changed. This changing may be due to the switching of the two modes of the collagen fibril, namely, rotation and translation. The virtual stiffness of the dermis increases and decreases on stretching and shrinking, respectively. An external force is converted to axon elongation efficiently on pushing as opposed to pulling. This effect leads to a nonlinearity in the nerve activity-, as explained by our normal strain hypothesis. A design is required to verify this model. Once the physical quantity that is encoded by the mechanoreceptors has been clarified, we can easily calculate that quantity either by a theoretical formula or by means of simulation. If it becomes possible to describe the spatiotemporal distribution of the activity of tactile receptors, it will be possible to obtain a signal design that exhibits a natural tactile sense. This will lead to the development of better tactile displays.

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