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## A Neural Network Model of Pain Mechanisms: Functional Properties of the Network Cells Providing the Power Law

HARUYUKI MINAMITANI

**Abstract**—Model simulation of a neural network providing the conduction mechanisms of pain and tactile sensations was undertaken, and the functional relations between neural activities of the network cells and stimulus strength applied on peripheral receptive fields were obtained. The functional relations of thalamocortical cells fall close to a straight line indicating a simple power law. The exponents of the power function are more than three for pain perceptive neural cells, while those for tactile sensation are about 1.5, which agrees well with the counterpart physiological data. The results are instructive for covering the gap between physiologically and psychophysically investigated somatosensory functions.

### I. INTRODUCTION

Studies elucidating somatosensory mechanisms are undertaken by two fundamental means. The electrophysiological method is the proper way to investigate neural receptive fields and neural pathways of somatic sensations such as touch and pain. On the other hand, the psychophysical method has served to obtain the subjective functions between any applied stimulus and the consequent sensibility perceived in human subjects, which present no inner mechanism of neural connections. Boundary studies covering the gap between physiological and psychological observations have not been largely reported, except to correlate the psychophysical power law with primary afferent or central neural activities [1]–[7]. Although some models and theories concerned with pain and tactile sensations have been proposed in conformity with the physiological aspects, none of them deal with the mathematical analysis and the synthesis as well. Model simulation may be expected to establish quantitatively the interrelation of both studies, viz., the numerical description connecting the psychophysical sensory mechanisms with the counterpart physiological evidence.

In a previous paper [8], a neural network model of pain mechanisms was proposed, and the model simulation was carried out to investigate inner mechanisms essential for pain and tactile sensations. The modeling was based on various assumptions

drawn from the results of physiological and anatomical studies in the literature [8]. The overall simplified model was constructed of 18 neural units, three afferent neural fibers ( $A_\beta$ ,  $A_\delta$ , and  $C$  fibers), six spinal cord cells, three brainstem cells, three thalamic cells, and three cortical cells, the response activities of which are provided by solving 18 simultaneous differential equations with two sets of linear differential equations for the adaptation effect of peripheral afferents. Thalamic ventral posterolateral nuclei (VPL) on the dorsal column-medial lemniscus tract (DCLT), being organized as a large myelinated  $A_\beta$  fiber and rapid tempo system, serves tactile sensation and terminates the axons in the first and second somatic sensory area (SI, SII) of the cerebral cortex. On the other hand, the extralemniscal reticular tract (ELRT) system plays an essential role in protopathic sensory modalities such as pain and thermal sensation, the evoked responses of which are transmitted by small  $A_\delta$  and  $C$  fibers. The centromedian parafascicular complex (CM-Pf) on ELRT serves the perception of slow burning pain and projects directly as well as indirectly on the associated area with SI and SII in the cerebral cortex ( $H$ ). Additionally, the thalamic posterior nuclei group (PO) on the neospinothalamic tract (NSTT) plays an important role in pain as well as mechano-perception, of which information are mainly transmitted by small myelinated  $A_\delta$  fibers. PO projects the information on the somatic sensory area of cerebral cortex, probably on SII and partly on SI. The temporal modality of the neural responses mimics the somatic sensation as follows.

Fast elevation of the initial bursts of VPL and PO cells are elicited by the  $A_\beta$  fibers' facilitation but are decreased drastically by the adaptation of  $A_\beta$  fibers, inhibition from the adjacent neural cells, and the negative feedback from the upper brain. This modality at the *initial phase* ( $\sim 50$  ms) seems to be the warning signal for the noxious stimuli succeeding the rapid reflex withdrawal movement. After the initial phase, activities of VPL and PO cells are again facilitated by the succeeding impulses transmitted on  $A_\beta$  and  $A_\delta$  fibers. Particularly, the firing rate of PO cells increases up to the second maximum at about 75 ms, which suggests that the fast stinging pain may be evoked in PO cells and projected on the cerebral cortex. When low stimulus intensity is applied, the firing rate of PO cells does not increase, and no fast pain but only tactile sensation occurs at this *second phase* (50–250 ms). If stimulus intensity is considerably high, CM-Pf cells are also facilitated by the transmitted impulses of  $C$  fibers at the *third phase* (250 ms  $\sim$ ) and the sensation of slow burning pain occurs and is projected on the cortical  $H$  and SII cells. Therefore, pain sensibility can be estimated by the firing activities of PO and CM-Pf cells as well as cortical SII and  $H$  cells, while tactile sensibility is estimated by those of VPL and cortical SI cells.

Corresponding to a previous paper [8], the response characteristics of the neural cells to stimulus intensity and the frequency of repetitive pulse sequences are investigated. Some stimulus-response relations characterized by the Stevens' power law are obtained from the specific features of temporal modality and compared with those of physiological data. As can be seen, this model simulation provides valuable information for elucidating the central neural mechanisms for sensory magnitude estimation, which may in turn present the analytical cue to discussing the physiological correlates of psychophysical functions. Computer simulation of the overall model has been made on a HITAC 8700/8800 digital computer (Hitachi Corp.), using the fourth-order Runge-Kutta method to solve the nonlinear differential neural equations. The simulation program has been written in Fortran. Parameter determination has been carried out on a trial-and-error basis by using the iterative method of model simulation. The precise procedure and the parameters have been presented in [8].

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The author is with the Department of Electrical Engineering, Biomedical Engineering Division, Faculty of Science and Technology, Keio University, Hiyoshi 3-14-1, Kohoku-ku, Yokohama 223, Japan.

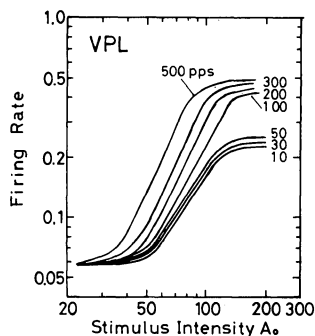


Fig. 1. Relation between stimulus intensity and firing rate of VPL cells where stimulus frequency is considered as parameter. Ordinate scale represents peak firing rate of VPL cells at second phase of temporal modality.

## II. FUNCTIONAL PROPERTIES OF THE NEURAL NETWORK CELLS PROVIDING THE POWER LAW

The maximum firing rate of each neural cell is normalized to be 1.0 because of the property of the neural equations used. All of the simulation results are obtained for the repetitive stimuli with 1-ms pulsewidth. Fig. 1 shows the relation between stimulus intensity and the firing rate of VPL cells where the stimulus frequency is considered as a parameter. The abscissa scale is stimulus intensity, while the ordinate scale represents the peak firing rate of VPL cells at the second phase. Both scales are plotted in log-log function. The initial- and second-phase bursts of firing discharges in VPL cells are mainly evoked by the tactile conduction on  $A_\beta$  fibers, while the third-phase firings are often influenced by  $C$  fibers if high stimulus intensity supramaximal for  $C$  fibers is applied. Since the initial-phase activity is considered as a transient response to the suddenly applied stimulation, the magnitude of tactile sensation should be represented by the peak second-phase response which follows the transient initial burst. As can be seen in Fig. 1, spontaneous activity of VPL cells is about 0.05. The application of low stimulus intensity, below  $A_0 = 30$ , evokes the activity of very few VPL cells. When stimulus intensity is increased, the firing activity of VPL cells is positively accelerated and the firing rate increases dramatically. The relation between the two variables falls close to a straight line, indicating a simple power function of the form  $FR = KA_0^n + C$ , where FR is the firing rate of VPL cells,  $K$  is a constant of proportionality,  $A_0$  is the stimulus intensity, and  $C$  is a constant representing the level of prestimulus spontaneous activity.

For higher intensity, above  $A_0 = 100$ , the acceleration of firing decreases as saturation is approached. As can be seen in the figure, the higher stimulus frequency results in the higher rate of firing discharges. Note however, that the stimulus-response relation for 50 pps and the lower frequency describes the different characteristics of those for 100 pps and higher. The maximum firing rate approaches 0.5 above 100 pps, but it descends to 0.25 below 50 pps. The response for the intermediate stimulus frequency (50–100 pps) is critical and amounts to the medium firing rates (0.3–0.4). The difference of maximum saturation of the second-phase firings mainly depend on the relation between the membrane time constant and the stimulus frequency, as well as stimulus pulsewidth. In the case of small membrane time constant, the firing rate of VPL cells increases even if the stimulus frequency and the pulsewidth are constant. Physiologically, virtual neural response in VPL cells may be accelerated by the recruitments of the neural fiber population and the raising of stimulus intensity and frequency [5]. Since the membrane time constant used in the model may be physiologically adequate, neural recruitment must be considered in the model in order to increase the maximum saturation for the lower frequency stimulation. The exponent  $n$  of the power function of VPL cells are about 1.7–2.0 for high-frequency stimulation (above 100 pps) and about 1.45–1.5 for low-frequency stimulation (below 50 pps).

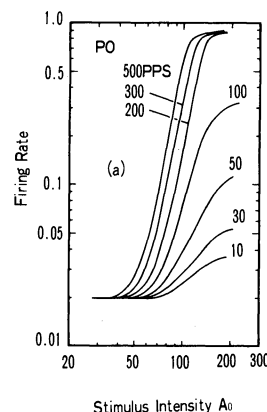


Fig. 2. Relation between stimulus intensity and firing rate of PO cells. Ordinate scale represents peak firing rate of PO cells at second phase of temporal modality.

Fig. 2 shows the stimulus-response relation of PO cells in log-log coordinates, the ordinate scale of which represents the peak firing rate in the second phase at about 80–100 ms. This activity is elicited by the  $A_\delta$  fibers' facilitation and is regarded as fast stinging pain responding to stimulus intensity supramaximal for  $A_\delta$  fibers. The firing rate of PO cells decreases somewhat at the latter half of second phase, and firing modality rather similar to that of VPL cells lasts during the stimulation if stimulus intensity lower than the  $C$  fibers' threshold is applied. Provided the stimulus intensity supramaximal for  $C$  fibers, the firing rate of PO cells increases owing to the  $C$  fibers' excitation at the third phase. As can be seen in Fig. 2, the firing rate of PO cells cannot be elicited so much in the low range of intensity as well as stimulus frequency. Increasing the stimulus intensity provides the positively accelerated firing activity in PO cells, and the stimulus-response relation is characterized by the form of the power function,  $FR = KA_0^n + C$ . Further increasing the stimulus intensity results in a decrease in the acceleration of firing as saturation is approached for each stimulus frequency. As a matter of course, the firing rate increases progressively along with stimulus frequency. The exponent  $n$  has shown about 3.7–4.8 for 100–500 pps of stimulus frequency; however, it decreases to 1.8 or less for frequency lower than 50 pps.

In contrast, Fig. 3 shows the stimulus-response relation of the third-phase activity in PO cells, the ordinate scale of which represents the peak firing rate of PO cells at the steady state after 280 ms ( $PO^*$ ). This activity is positively accelerated by the peripheral stimulation supramaximal for  $C$  fibers. If stimulus intensity lower than the  $C$  fibers' threshold accompanied by low stimulus frequency is applied, no facilitation on  $C$  fibers and no succeeding elevation of firing activity in PO cells are evoked at the third phase. PO cells only hold the lasting low firing rate elicited by  $A_\delta$  fibers at this phase. However, the high-frequency stimulation with low-intensity subliminal stimulus for  $C$  fibers brings the accumulation effect of short interval repetitive pulses on spinal cord cells (lamina V), which in turn evokes high firing activity of PO cells lasting over the second and third phases. Thus the stimulus-response relation regarding the third-phase activity involves the rather complex components, depending on both  $A_\delta$  fibers' and  $C$  fibers' facilitations. As can be seen in Fig. 3, the response relation for higher frequencies shows a quite similar characteristic to those of Fig. 2, but a little higher intensity is needed for eliciting the same degree of third-phase activity as for the second-phase activity. When low stimulus frequency of 10–30 pps is applied, high intensity supramaximal for  $C$  fibers is needed to elicit the sufficiently high firing rate of PO cells. The response to high intensities ( $A_0 > 120$ ) shows the steeply arising characteristic elicited by  $C$  fibers' excitation. These functional relations are also represented by a power function of  $FR = KA_0^n + C$ , the exponents  $n$  of which are about 4.4–4.6 for 50–500 pps and

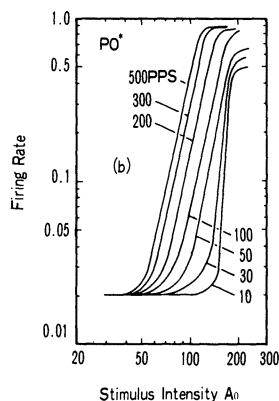


Fig. 3. Relation between stimulus intensity and firing rate of PO cells. Ordinate scale represents peak firing rate at steady state of third phase (PO\*).

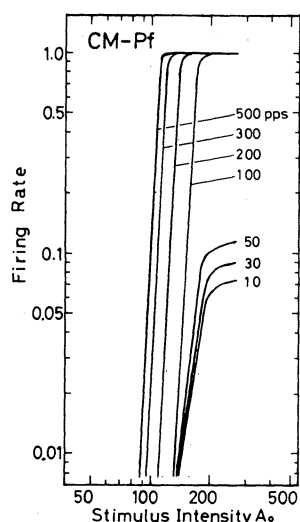


Fig. 4. Relation between stimulus intensity and firing rate of CM-Pf cells. Ordinate scale represents peak firing rate at steady state of third phase.

about 9.8–10.0 for 10–30 pps. The firing rate of spontaneous activity is 0.02, while the maximum firing rate of PO cells reaches to about 0.95.

Fig. 4 shows the stimulus–response relation of CM-Pf cells. The ordinate scale represents the peak firing rate of CM-Pf cells at the steady state of third phase. Activation of the cells is mostly elicited by C fibers and evoked by higher intensity than those for VPL and PO cells. Even a slight increasing of stimulus intensity provides a precipitous elevation of the firing rate, and it readily reaches maximum saturation. About 1.0 of the maximum firing rate is evoked by the high-frequency stimulation, while the maximum saturation reduces to less than 0.12 for frequencies lower than 50 pps. These characteristics may be caused by the accumulation effect of pulse sequences relating the membrane time constant; that is, the long interval of stimulation does not provide the facilitation lasting enough to the succeeding input, and the higher frequency of stimuli results in the more accumulation of firings in the cells. The functional relation of CM-Pf cells is also characterized by the form of power function, and the exponents  $n$  are about 18.0–21.0 for 100–500 pps and about 6.5–8.0 for 10–50 pps. The firing characteristic of CM-Pf cells seems to represent pain generation; the small derivative of input intensity brings a large increase of the firing rate which may correspond to light pain, heavy pain, intolerable pain, and so forth. This relation can give rise to the conjecture that the discrimination of the magnitude of pain sensation is unreliable, just as we cannot

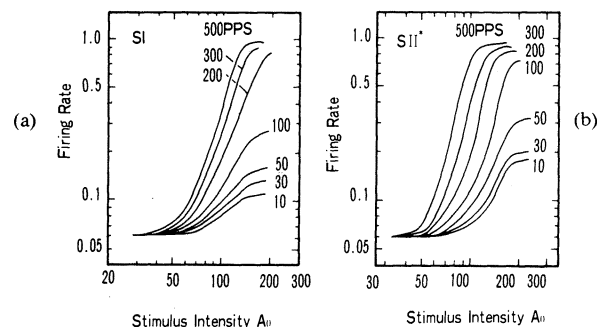


Fig. 5. Relations between stimulus intensity and firing rates of cortical SI and SII cells. (a) Peak firing rate of SI at second phase. (b) Firing rate of SII at steady state of third phase.

clearly perceive the psychophysical quantitative values of pain sensation.

Regarding the response functions of cortical SI, SII, and H cells, rather similar relations to those mentioned above have been obtained. Fig. 5(a) shows the functional relation of the peak firing activity of SI cells at the second phase while Fig. 5(b) is the third-phase activity of SII cells (SII\*). The spontaneous activities of both cells are the same, about 0.06; however, the firing rate of SI cells is elicited by lower stimulus intensity than that for SII cells. In the middle range of stimulus intensity, both relations are approximately followed by the form of power function, the exponents  $n$  of which are 0.9–2.5 for SI cells and 1.8–3.6 for SII cells, respectively. The functional relation of SI cells represents the tactile sensation, which resembles the characteristic of VPL cells. On the other hand, the relation of SII cells represents the pain sensation projected from PO cells. Similar relations of H cells and the second-phase activity of SII cells are omitted here, but they are depicted in the following section.

### III. COMPARISON OF THE SIMULATION RESULTS WITH PHYSIOLOGICAL DATA

Fig. 6(a) shows the relation between the frequency of stimulation and the firing rate of VPL cells at the second phase, which estimates the sensation magnitude of tactile mechanism. This relation is transformed from the result described in Fig. 1, and the stimulus intensity is considered as a parameter. The abscissa scale is the frequency of stimulation, while the ordinate scale represents the firing rate of VPL cells, minus a constant 0.07 (FR-0.07). A very low firing rate of 0.07, involving the spontaneous activity of VPL cells, is assumed as the minimum magnitude of tactile sensation. The functional relation between two variables is represented by a power function, the exponent  $n$  of which is 0.75 for  $A_0 = 80$  and 0.6 for  $A_0 = 100$ , respectively. In lower as well as higher ranges of stimulus frequency, the curves deviate slightly from the power law. For comparing the simulation result with physiological data, the relation between the frequency of stimulation and that of response is shown for a typical ventrobasal thalamic VPL neuron in Fig. 6(b). This relation is from the result reported by Poggio *et al.* [9], in which the responses of VPL cells to electrical repetitive stimulation have been extracellularly recorded from unanesthetized monkeys. Rectangular pulses of sufficiently supramaximal intensity have been applied on the peripheral receptive field. The condition of the physiological experiment which is from that of model simulation is that the pulsewidth of 0.1 ms has been used. The abscissa scale is the frequency of stimulation, while the ordinate scale represents the mean frequency of neuronal discharge in log–log coordinates, less a constant for the mean values of the prestimulus rate of discharge. The responses follow the stimuli to rates of 100–200 Hz with great fidelity. At or above these rates, the cells fail to respond to every stimulus; however, with a further increase in the frequency of stimulation, no further increases occur in the

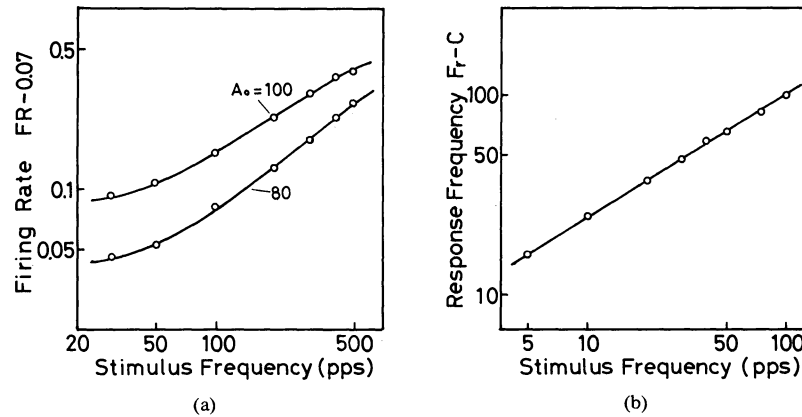


Fig. 6. Functional relations between frequency of stimulation and firing activity of VPL cells relating to tactile sensation. (a) Simulation result transformed from Fig. 1. (b) Physiologically obtained data referred from Poggio and Mountcastle [9].

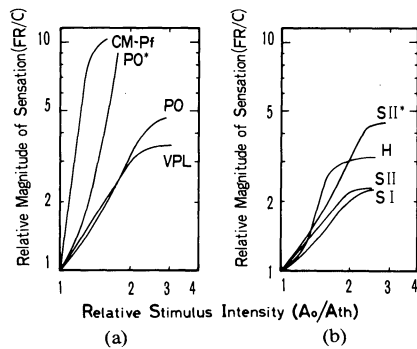


Fig. 7. Functional relations between relative stimulus strength and relative magnitude of pain and tactile sensations, modified from Figs. 1-5. (a) VPL, PO, PO\*, and CM-Pf. (b) SI, SII, SII\*, and H cells.

frequency of nerve cell response. The functional relation between the frequency of neuronal discharges and the frequency of stimulation shows a power function the exponent  $n$  of which is about 0.64. Both results in Fig. 6(a) and (b) represent the quite similar power functions regarding tactile sensation; however, the values of coordinates are not coincident between the model and the physiological data.

In contrast, Fig. 7(a) and (b) show the relations between relative stimulus strength and the relative magnitudes of pain and tactile sensations, which are modified from the results described in Figs. 1-5. The ordinate scale represents the firing rates of various neural cells, normalized by a constant  $C$  involving very little firing activity and the spontaneous response of each neural cell.  $C$  is arbitrarily determined on the assumption that it represents the minimum magnitude of each sensation which may be perceived with difficulty. The abscissa scale is also normalized by the threshold intensity  $A_{th}$  necessary to maintain the constant firing rate of  $C$ . Characteristics in these figures are only for stimulus frequency of 50 pps. The relative magnitude of each neural activity is followed by a power function with an increase in stimulus intensity as  $FR/C = (A_0/A_{th})^n$ . The exponents  $n$  for the neural cells associated with pain sensation become larger than those for tactile sensing neural cells. Regarding the functional relations of PO and SII cells, the exponents of second-phase activities are less than those of third-phase activities; the former may provide the tactile or fast pain modality, while the latter represent slow burning pain. The described stimulus-response relations mostly mimic the Stevens power law; however, the relative firing rates approach the maximum plateau when high intensity is applied. The exponent  $n$ , the minimum sensation magnitude  $C$ , and the threshold intensity  $A_{th}$  of each neural cell are tabulated in Table I. As can be seen the threshold intensities for tactile neural cells are low relative to those of pain sensing neural cells.

For verifying the results of model simulation, some physiological data are represented in Fig. 8. These data are from the study by Toda *et al.* [7] and are modified a little for comparison with this study. The relations between intensities of electrical noxious stimuli applied to the tooth pulp and the magnitudes of central evoked responses have been investigated with lightly anesthetized rats. Constant current pulses of 0.1-ms duration at 1 or 2 Hz have been delivered for stimulation, and micropipette electrodes have been used for recording intracellularly the evoked potentials from SI, SII, PO, and the ventrobasal complex (VB) cells. The abscissa scale is relative stimulus intensity, while the ordinate scale represents the relative amplitude of the evoked potential. Both are normalized by the minimum amplitude and the corresponding stimulus intensity. From this figure, the stimulus-response function in each neural cell can be said to follow the power function only in the range of lower stimulus intensities; however, it deviates from the power function in the higher ranges. The exponents of the characteristics represented in linear relationship on the log-log coordinate are also tabulated in Table I. In spite of the different stimulus conditions between model and physiological experiments, comparison of both results shows rather similar functional values regarding the power law. In particular, the stimulus-response relations of VPL, SI, and the second activities of PO as well as SII cells depicted in Fig. 7(a) and (b) closely resemble the physiological ones.

On the other hand, Fig. 9 shows the relation between relative stimulus intensity and the relative magnitude of cortical evoked potential in human subjects. In the study made by Spreng and Ichioka [1], electrical noxious stimuli with rectangular pulse sequences (0.5 Hz, 0.3-ms pulsewidth) have been applied to the tooth pulp of human subjects, and the stimulus-locked cortical potentials have been measured by using special electronic averaging equipments. The stimulus-response relation has also been represented by the form of the power function, the exponent of which is  $3.12 \pm 0.4$  for two subjects. In the figure, the intensity course of the equivalent psychophysical result regarding pain sensation obtained by Stevens [2] is also represented for comparison with the dependence on stimulus strength of the amplitude of the evoked potentials. In his experiment, 60-Hz sinusoidal electrical shock was applied through human fingers, and the sensation magnitude was determined by the psychophysical matching method. Although the stimulating conditions of both experiments are quite different, respectively, the exponents of the function are in good conformity. As can be seen in Fig. 9, the exponent of the power function related to pain are higher than those for tactile sensation.

From the results mentioned, all functional relations between relative stimulus intensity and the relative magnitude of pain and tactile sensations are tabulated in Table I. Experimental conditions and experimental mammals are quite different among the four studies; however, the results of model simulation mimic the

TABLE I  
FUNCTIONAL VALUES OF MAJOR NEURAL CELLS WITH COUNTERPART PHYSIOLOGICAL AND PSYCHOPHYSICAL DATA<sup>a</sup>

	Model Simulation								Electrophysiological Results				Psychophysical Result	
	VPL	SI	PO	SII	PO*	SII*	CM-Pf	H	VB	PO	SI	SII	Cortical cell	
n	1.5	1.1	1.8	1.15	4.6	2.2	8.0	3.6	1.66	1.6	1.55	1.43	3.12 ± 0.4	3.5
c	0.07	0.07	0.023	0.07	0.023	0.07	0.01	0.07	—	—	—	—	—	—
A <sub>th</sub>	53	65	70	75	80	80	150	125	76 (μA)	62	50	52	—	—
Remarks	50 pps, pw = 1 ms. rectangular pulse								1~2 Hz, 0.1 ms. rectangular pulse				0.5 Hz, 0.3 ms. rectangular pulse	60 Hz sinusoidal electrical shock
	PO, SII: second-phase activity (tactile or fast pain sensation)								rat				human subj.	human subj.
	PO*, SII*: third-phase activity (slow burning pain)								(Toda et al. [7])				(Spreng & Ichioka [1])	(Stevens [2])

<sup>a</sup>n-exponent of the power function, C-minimum sensation magnitude; A<sub>th</sub>-threshold intensity.

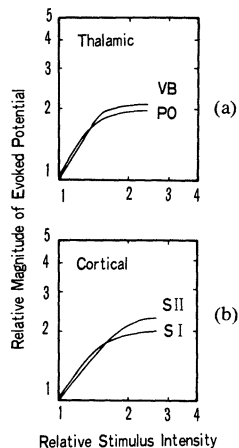


Fig. 8. Functional relations between relative stimulus strength and relative magnitude of firing responses of major neural cells relating to pain and tactile sensations physiologically obtained. (a) VB and PO cells. (b) SI and SII cells. Data are from Toda *et al.* [7].

characteristics of central neural functions investigated by physiological and psychophysical methods. The power functions of neural activities in tactile sensing VPL, SI, as well as of the second-phase activities in PO and SII cells, are similar to those obtained by electrophysiological study. The exponents of the third-phase activities of PO and H cells also mimic the pain characteristics obtained by Spreng and Ichioka as well as by Stevens, while the response of CM-Pf cells becomes larger than those.

#### IV. DISCUSSION

The aim of this study was to present a neural network model providing the conduction mechanisms of pain and tactile sensations and to obtain the functional relations between neural activities of the network cells and the applied stimulus intensity. The relations between the two variables fall close to a straight line indicating a simple power function, particularly in the middle range of stimulus intensity. For lower or higher intensity of repetitive stimulation, the neural response is commonly observed to deviate from the straight line, and the gradient becomes smaller. This is consistent with the basic evidence of physiology that the neural response to low-intensity subliminal stimulation of the neural cells is very slight but positively accelerated, while at very high intensity it is intense but slightly accelerated as it approaches the maximum saturation. Regarding individual sensi-

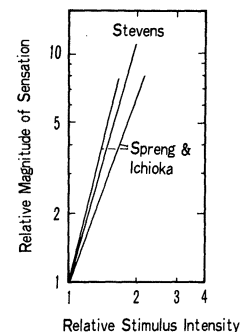


Fig. 9. Functional relations between relative stimulus strength. (a) Relative magnitude of cortical evoked potential in human subjects, from [1]. (b) Psychophysical sensation magnitude obtained by Stevens [2].

bility, the log-log slope of the straight line is greater, and the firing threshold intensity is much higher, for pain than for tactile sensation. The exponent of the power function obtained by model simulation is related to the sensitivity coefficient  $\nu_i$  and the coupling coefficient  $iC_j$  of neural cells defined in the neural equations [8]. Since  $\nu_i$  is the same for all neural cells, the differences in sensibility mainly depend on  $iC_j$ . Physiologically, the differences in sensibility may be determined by differences in peripheral and central innervations. Furthermore, the magnitude of sensation may depend on the recruitment of new units progressively brought into action with the increasing amplitude of peripheral stimulation [5]. How the neural recruitment grows up in correlation with sensation modality is not yet specified; however, it must be closely related to the extent and strength of stimulation as well as the density and threshold of peripheral and central neural populations. This suggests that the exponent of the power function varies largely within the individual subjects and their test points stimulated.

Knibestöl and Vallbo [6] investigated the stimulus-response functions of primary afferents and psychophysical intensity estimation on mechanical skin stimulation in the human hand. Their findings, obtained from 46 test points in 18 subjects, showed that the neural as well as psychophysical stimulus-response relations were individually fitted to the power function, but the exponents of the power function showed a rather large range of variation within the two sets of data. Most of the neural functions had exponents below 1.0 ( $n = 0.3-1.7$ , mean = 0.72) while a large proportion of the psychophysical functions had exponents above 1.0 ( $n = 0.4-2.1$ , mean = 1.2). This reveals that the distributions of neural and psychophysical functions are

clearly different, which in turn suggests that the power function of peripheral response is inconsistent with the corresponding central neural function. Particularly, some discrepancies exist in the subjective magnitude of sensation between the various psychophysical reports, which may be due to methodological factors [3], [4]. Different experimental conditions as well as different experimental animals may yield different power functions relating to the magnitude of identical sensation, even if the subjects are in well-trained human groups. It will be not so much a question of whether the exponent of the power function is consistent with those of a specified group. The essential thing is that the stimulus-response function closely fits the form of the power function or of the logarithmic function or log-tanh function [6], and that the threshold intensity as well as the exponent of the power function are much larger for pain than for tactile sensation. Judging from this point of view, the results of model simulation seem to represent the functional relation between stimulus intensity and the neural responses of pain and tactile sensations in accordance with those of physiological and psychophysical studies. However, the exponent of the power function of CM-Pf cells' activity, regarded as slow burning pain in the model, is too large compared with the physiological and psychophysical magnitude estimations of pain sensation. Model parameters such as the coupling coefficient and the firing threshold of CM-Pf cells must be smaller than the present values in order to obtain the most optimal fitting of simulation data to the empirical characteristic of pain modality.

The other improvements which should be made in the proposed model are as follows. 1) Neural recruitment must be considered in the model by introducing the coupling coefficient which increases in quantity with increasing input intensity evoking the interacted neural cells. 2) The distribution of firing thresholds in a neural group integrating many homologous neurons must also be considered in the model to realize the recruitment of new units progressively brought into action with the increasing amplitude of stimulation. 3) It is probably a common notion that the sensitivity coefficients  $\nu_i$  of neural cells are not uniform, but the differences in  $\nu_i$  would be considerable between the cells participating in pain and the other somatic sensations. Thus the sensitivity coefficient should be different for each neural cell. 4) As can be perceived in human subjects, tactile sensation is well localized, but pain is diffuse and poorly localized, so that the localization of somatic function may also come into question, and the distribution of receptor population should be given for the model realization. Such problems still await solution.

The results of model simulation have been obtained by applying rectangular pulse sequences of 1-ms pulsewidth. In contrast, most physiological experiments used rectangular pulse sequences with short duration (0.1–0.3 ms). Differences in the stimulating condition cannot provide the close identification between both results. In the model, however, time scaling allows easy identification of the condition by substituting the variable  $t$  as  $t = Dt^*$  in the used neural equations [8] as follows:

$$\mu^* \frac{dF_i}{dt^*} = -F_i + (1 - r_i F_i) \frac{1}{1 + \exp\{-\nu_i (C_i F_j - \theta_i)\}}$$

where  $\mu^* = \mu/D$ . As can be supposed from the equation,  $t^*$  is scaled by  $1/D$  which readily makes the pulsewidth short, and the firing response is closely related to the scaled membrane time constant  $\mu^*$  as well as the scaled stimulus frequency. If scale factor  $D = 5$ , time variable  $t = 1$  ms, and membrane time constant  $\mu = 5$  ms, then  $t^*$  and  $\mu^*$  become small as  $t^* = 0.2$  ms and  $\mu^* = 1$  ms.

This study assumes that the functional relation of tactile sensation is represented by the second-phase activities of VPL and SI cells, while pain is represented by the second-phase activities of PO and SII cells for fast stinging pain and by the third-phase activities of CM-Pf and  $H$  cells for slow burning pain. It is probably adequate to assume the both activities of NSTT and

ELRT cells as for pain sensation, but a question arises as to whether the tactile sensation should be represented by the initial-phase activity or by the second-phase activity of DCLT cells. Thinking of the psychophysical magnitude estimation of the tactile function, second-phase activity would better represent the tactile function than the initial-phase activity of DCLT cells because the latter is a transient activity adapting quickly after the beginning of stimulation. The functional relation of the maximum initial-phase activity of VPL cells also falls close to a straight line of the power function; however, the exponents are relatively large: about 1.9–3.8 for 10–500 pps of stimulus frequency. It seems to reveal no tactile but a warning response eliciting the rapid reflex withdrawal movement. From this point of view, representing tactile function by using the second-phase activities of VPL and SII cells may be adequate.

#### IV. SUMMARY AND CONCLUSION

In this correspondence, a neural network model of pain mechanisms has been proposed, and the model simulation has been carried out to investigate inner mechanisms essential for pain and tactile sensations and to obtain the stimulus-response relations of the network cells. Despite the simplification of neural pathways and the arbitrary determination of parameter values, the present data of model simulation seem to show a general agreement with corresponding physiological data. The following general conclusions may be drawn from these studies.

1) The magnitude of tactile sensation can be estimated from the second-phase firing activities of thalamic VPL cells and cortical SI cells. The functional relation between stimulus intensity and the firing rate was indicated by a power function, the exponents of which were in the range of 0.9–2.5. An initial burst of firing activity may be considered as a warning signal for the suddenly applied stimulus succeeding the reflex withdrawal movement.

2) The sensibility of fast stinging pain is estimated by the second-phase activity in thalamic PO cells, while that of slow burning pain is simulated by the third-phase activities in PO and CM-Pf cells. They are innervated by high-threshold  $A_\delta$  and  $C$  fibers, respectively, and projected in cortical SII cells and the associated area with SI and SII cells. The pain function was also represented by the power law, the exponents of which indicated almost more than three.

3) In the lower or higher range of stimulus intensity, the stimulus-response relation of each neural cell deviates from the straight line of the power function, and the log-log gradient becomes smaller. It is physiologically proper that the magnitude of evoked responses is not much facilitated by the low stimulus intensity supraliminal for the neural cells. In contrast, saturation of the firing activity may require that each neural unit is supramaximally stimulated at the onset of saturation and, therefore, transmitted the maximum information from the periphery.

4) High stimulus intensity inducing the maximum saturation in tactile VPL cells is equivalent to a noxious input, which in turn follows the facilitation of high threshold CM-Pf cells and may yield the pain generation.

5) As indicated in the tactile and pain sensibilities, temporal summation is confirmed in the growth of sensation; that is the proportionality between stimulus frequency and the increase in the firing rate was observed on the wide range of middle stimulus intensity. Such a specific feature was theoretically predicted by Zwislocki [10] and experimentally confirmed by Verrillo [11].

6) The growth of sensation may be discriminated by the associated number of receptors, as well as the upper neural cells excited and the amount of evoked responses in thalamocortical neural system. An increase in the magnitude of sensation may be related to an increase in the number of sensory units, that is, the number of afferent neural pathways and synaptic connections. The coupling coefficient between neural network cells corresponds to the number of such neural connections which may be

dominant on the exponent of the power function. Regarding pain sensation, it is suggested that many synaptic connections, viz., a large coupling coefficient, may result in an exponent larger than that for tactile function.

Although a number of physiological studies are now underway to elucidate the central neural mechanisms of somatic sensations and the behavioral responses, how the magnitudes of pain and tactile sensation are discriminated in the central neural system and why they follow a power function is not easily explained. As mentioned, some problems still remain in the present study; however, the proposed model provides a good simulation of the magnitude estimation of pain and tactile sensation relating to the power law. Mathematical model study is instructive in elucidating the interacted sensation mechanism and in bridging the theoretical gap between neural functions physiologically investigated and the subjective estimation of psychophysical sensory magnitude. Further improvement and characterization of the model should be done on the basis of well-designed controlled electrophysiological experiments.

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### Computer Coordination of Limb Motion for Locomotion of a Multiple-Armed Robot for Space Assembly

CHARLES A. KLEIN AND MARK R. PATTERSON

**Abstract**—Robotics is expected to play an important role in the construction of large space structures. One possible system would be a

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C. A. Klein is with the Department of Electrical Engineering, The Ohio State University, Columbus, OH 43210.

M. R. Patterson was with the Department of Electrical Engineering, The Ohio State University, Columbus, OH. He is now at Battelle Columbus Laboratories, Columbus, OH 43201.

multiple-armed manipulator vehicle which could use general-purpose arms to walk over the structure to a construction site and then use the arms for assembly. The simulation of locomotion of such a vehicle with arms modeled after a currently available industrial manipulator is described.

#### I. INTRODUCTION

For the coming years, plans are being started for the construction of large structures in space. Such structures include solar space stations, large multibeam antennas such as reflectors and lenses, and space factories [1]. Space construction techniques being studied include at-site manufacture of beams from rolls of material and assembly of stackable control beams [1]. Robotics is expected to have an important role in the construction, inspection, and maintenance of these structures in view of the alternative astronomical cost of supporting men as construction workers in space [2], [3].

Although at this time exact requirements for a space robotic system are far from definite, several conceptual designs have been proposed [4]. One proposed robot is a free-flying teleoperator which could carry building materials from the bay of a space shuttle to a remote construction site with propulsion units. A number of independent arms would be useful for manipulating the payload and fine positioning the vehicle with respect to the framework of the large space structure. One can foresee the need for a multiple-armed vehicle without the propulsion units of the free-flying teleoperator; it would depend on its general-purpose arms to walk over the girders of the structure to the current construction site and, having arrived, would use its arms for assembly or maintenance. Such a system, which could be called a multiple-armed manipulator vehicle (MAMV), would be ideal for unattended maintenance since electric motors could be powered from solar rechargeable batteries, whereas propulsion units would need additional propellant.

A key problem for such a space robot is the control of its arms/legs for walking across a structure. Ideally, the lower level portions of that control task would be performed automatically by a computer. For instance, a supervisor, either human or computer, could specify the body's position, orientation, velocity, and angular velocity, and a walking algorithm could generate the necessary foot motions to be executed by even lower levels in the hierarchical control structure. This control philosophy is incorporated in the concept of supervisory remote control [5], [6].

A considerable amount of research has already been done on terrestrial legged walking vehicles in hopes of developing an efficient means of locomotion over irregular terrain [7]. Many studies have considered the leg sequence or gait selection problem for the special case of straight-line constant-speed locomotion over level ground. Although combinatorially large numbers of possible gaits [8] exist, stability under gravity has been used to select certain optimal gaits [9]. At the Ohio State University, a hexapod vehicle has been built which demonstrates each of these wave gaits [10], accepts high-level joystick commands from an operator to perturb these gaits for turning control [11], and uses force information for limited adaptation to irregular terrain [12]. Other legged vehicles are the General Electric quadruped [13], the JPL Mars Rover [14], and a number of Russian vehicles [15].

However, for space locomotion, gravity is not a consideration, and since footholds must be grasped, only a limited number of footholds are possible, eliminating regular gaited locomotion. Gaited locomotion has been extended to nonperiodic leg sequences known as "free gaits" which allow a vehicle to traverse regions containing subregions unsuitable for support [16], [17]. Again, however, the heuristic algorithms feature stability of support in a gravitational field and make simplistic assumptions about the properties of the individual arms.

Although making a complete design of a space robotic system is premature, determining which engineering problems are crucial