醫工學會誌 : 第 2 卷 第 1 號, 1981
J. of KOSOMBE: Vol. 2, No. 1

研 究 論 文
81—1—06

Realization of the Dynamic Control System for the Neural Network Analysis of the Cerebellum*

Myoung-Ho Lee

=Abstract=

This paper deals with a new approach to the modelling of neural interactions in the cerebellar cortex to construct a general purpose electronic simulation model. Since physiological data show that cerebellar neural activity changes in an approximately pulse manner in response to pulse stimulation, the differences in timing between excitation and inhibition of cerebellar cells will be treated as pure time delays and the transfer functions of the cells will be presented by pure gains.

The parameters to be discussed in this paper are the coupling coefficients between a cell and its several inputs, the magnitude of a coupling coefficient which is presented as a measure of how much influnce a particular has on its target cell. And also this paper has been proposed that the cerebellum engaged in improving the overall performance of the motor control system, i.e., the cerebellum is a compensator.

1. Introduction

The cerebellum is the principal organ concerned in the control of movement. The physiological functions of the cerebellum can be divided into several portions, each of which acts as a coordinator for a certain type of motor action.

In recent years, a wealth of knowledge has been obtained concerning the neural compositions and activities in the cerebellum and related structures. Usually there have been opened the three following new approaches to the mechanisms of the cerebellar coordination. The first approach is to analyze the neural network in the cerebellar cortex and to suppose what sort of computation may occur there. The second approach is to observe impulse activities in the cerebellar cortical neurons and to speculate what sort of information processing occurs there. The third approach is to analyze how the cerebellum is linked with subcerebellar motor centers and to grasp the significance of the cerebellar control of various motor functions.

Especially, there are only five different types of cells in the cerebellar cortex and their interconnecions are homogeous throughout. In theory, therefore, one need only analyze a small population of cells in order to understand how the cerebellum processes incoming information. For these reasons, the cerebellum has attracted the attention of neural modelers in recent years.

Several models based on known numerical parameters have been proposed. Marr[1] descri-

<1981. 4. 27 접수>
*본 논문은 1980년도 문교부 학술연구 조성비에 의한 것임.
연세대학교 전기공학과
Dept. of Electrical Eng. Yonsei University

bed the cerebellum learning devices in which output is modified until it is appropriate to the contexts represented by the input. Mortimer[2], using automata theory, simulated a small patch of cerebellar cortex containing several cells. The result of his simulation were in substantials agreement with the physiological data obtained by Eccles at el[3]. The model of Cavert and Meno[4] predicted that the cerebellar cortex would inhance the detail of input patterns.

This paper only deals with the first approach to construct a general purpose electronic simulation model for the neural network in the cerebellar cortex. On the other hand, this paper has been proposed that the cerebellum engaged in improving the overall performance of the motor control system, i.e., the cerebellum is a compensator[5].

2. Neural Network of the Cerebellar Cortex

The cerebellar cortex has five kinds of neurons: granule cells, Purkinje cells, Golgi cells, basket cells, and stellate cells. Only granule cells are excitatory and the other four kinds of cells are inhibitory. It also has two kinds of input fibers called mossy fibers and climbing fibers. Both of them are excitatory. Output fibers of the cerebellar cortex are axons of Purkinje cells, and hence are inhibitory. Main connections between these cells and input fibers are shown in Fig. 1. All inhibitory neurons are shown in black.

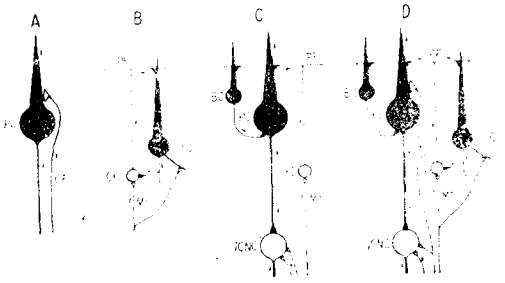

Fig. 1. Diagram of the most significant cells and their synaptic connections in the cerebellar cortex. CF, a climbing fiber:MF, a mossy fiber: PUU, a Purkinje cell: GRC, a granule cell: GOC, a Golgi cell: BAC, a basket cell: STC, a stellate cell: PF, a parallel fiber.

The climbing fiber(CF) has been shown to make an enormously powerful excitatory synapse with the Purkinje cells(PUC) that fires several times to a single climbing fiber impulse. By contrast the mossy fiber(MF) is very diversified, each branching so as to contribute excitation to about 400 granule cells, and 80,000 granule cells via their parallel fibers (PF) contribute to the excitation of each Purkinje cell. The basket cells(BC) are also excited by parallel fibers and their action go transversely to end as inhibitory synapses on the Purkinje cell somata, which are in this way encased in a basket-like structure. The Golgi cells(GOC) are excited by parallel fibers and also by mossy fibers, and their inhibitory synapses on the granule cells complete a simple negative feedback loop.

3. The Dynamic Control Model of the Cerebellar Cortex

The main signal flow diagram is shown in Fig. 2. As can be seen in Fig. 2, Golgi cells have two input signal paths, that is, feedforward path and the feedback path. The feedforward path transmits mossy fiber input signals and the feedback path transmits outputs of granule cells to Golgi cells. The Purkinje cells provide the only output from the cerebellar cortex, all other cells in the cortex are interconnections. The granule cells are excited by the mossy fibers and in turn,

excite the Purkinje cells, Golgi cells, basket cells and stellate cells. The Golgi cells, which also receive direct input from the mossy fibers, inhibit the granule cells, while the basket and stellate cells inhibit purkinje cell activity.

The model to be developed in this paper takes advantage of the differences in timing between the excitation and inhibition of the various cells in the cerebellar cortex. There is, however, an important distinction between the two models. Calvert and Meno's model used the excitatory(EPSP) and inhibitory (IPSP) post-synaptic potentials of the purkinje cell to find different excitatory and inhibitory time constants.

However, the time constants they chose were larger than indicated by the data of Eccles, et al.[3]. When the proper time constants for the EPSP and the IPSP of appoximately 5 and 20 ms, respectively, are inserted into their model, the interesing dynamics of their model occur at frequencies greater than 20 Hz. But one encounters another difficulty with the approach used by Calvert and Meno[4].

The EPSP and IPSP of the Purkinje cell follow exponential time courses in response to pulse stimulation of the mossy fiber input. However, the spike activity of the purkinje cell, its true output, responds to the same pulse stimulation with a short increase in firing rate followed by a silent period. These spike frebuency changes occur in an approximately step manner and, therefore, a nonlinear relationship exists between the postsynaptic potentials and the spike activity of a purkinje cell. Thus, the linear transfer functions derived by Calvert and Meno are not physiologically meaningful.

A new approach to the modelling of neural interactions in the cerebellar cortex will be presented. Since physiological data show that cerebellar neural activity changes in an approximately pulse manner in response to pulse stimulation, the differences in timing between excitation and inhibition of cerebellar cells will be treated as pure time delays(d) and the transfer functions of the cells will be presented by pure gains(g,r,b,p).

The last parameter to be discussed is the coupling coefficient(k_i) between a cell and its several inputs. The magnitude of a coupling coefficient is a measure of how much influnce a particular input has on its target cell. For example, the Golgi cell has both mossy fiber and parallel fiber inputs. If the mossy fiber input is stronger than the parallel fiber input due to either a larger number of active fibers or more efficacious synaptic contacts, then the

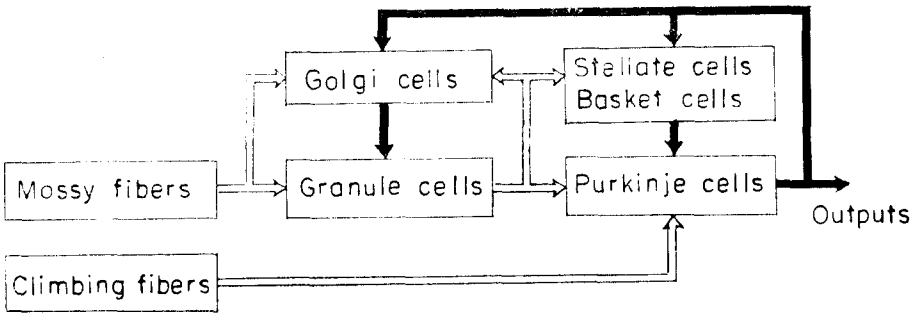

Fig. 2. Diagram showing main signal flows in the cerebellar cortex. ⊸and ➡are excitatory and inhibitory signals, respectively.

mossy fiber input will have larger coupling coefficient with the Golgi cell than will the parallel fiber input.

The dynamic control model of the cerebellar cortex is shown in Fig. 3. In order to avoid further complications of the model, the basket and stellate cells have been lumped together in one block. It should also be noted that the time delays and the coupling coefficient are not absolute measures but relative measures of the excitatory and inhibitory influnces present. For these reasons, some pathways do not any parameters associated with them, since their parameters have been absorbed into other pathways.

The straight-through time delay between mossy fiber input and Purkinje cell output is not shown. A reasonable value of 5 ms for this delay would a total phase lag to the system of 14° at 10 Hz and 1.8° at 1 Hz. The dynamics of this model can be more easily understood if the simple network $H_1(s)$, $H_2(s)$ inside the dotted block are examined for mossy fiber and climbing fiber inputs, respepectively:

From the signal flow graph in Figure. 3, if $H_1(s)$ and $H_2(s)$ are the transfer functions for input F(s) and input C(s), respectively, then

$$G_F(s)=H_1(s)F(s) \quad (1)$$

$$G_C(s)=H_2(s)C(s) \quad (2)$$

Therefore,

$$G_F(s)+G_C(s)=H_1(s)F(s)+H_2(s)C(s) \quad (3)$$

$$H_1(s)=\frac{p(1-bk_3e^{-sd_3})}{1-bpk_4e^{-sd_4}}F(s) \quad (4)$$

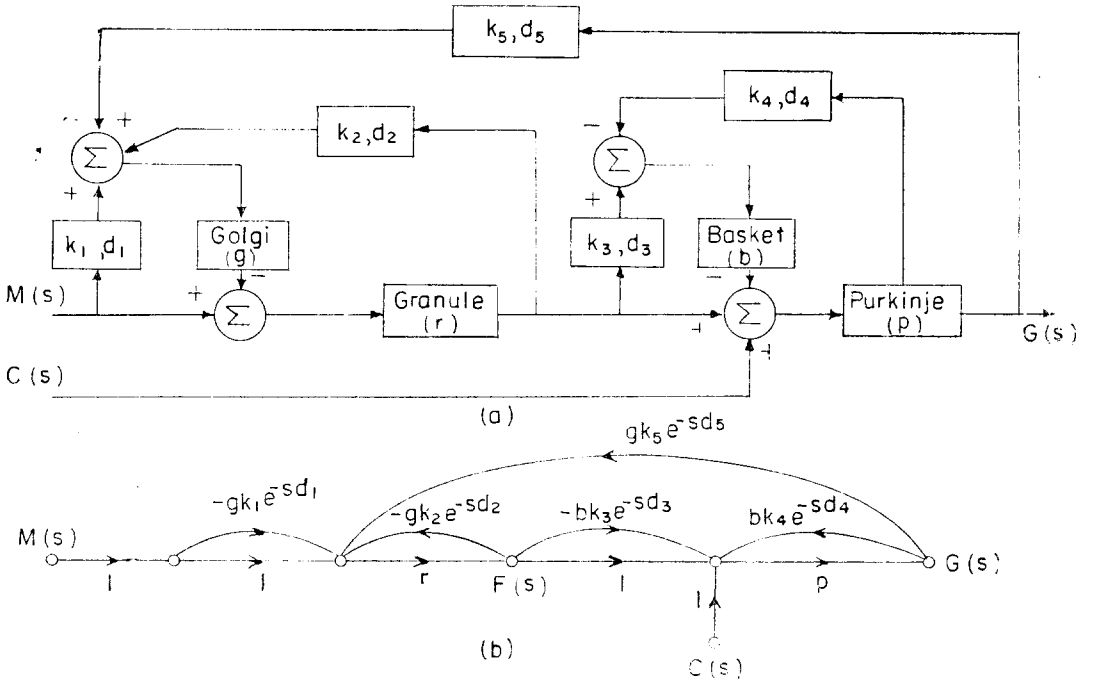

Fig. 3. Block diagram of the cerebellar cortex and its signal flow graph. M(s), mossy fiber input: C(s), climbing fiber input: F(s), parallel fiber input: B(s), basket and stellate cell output: G(s), purkinje cell output: k, coupling coefficient: d, time delay: b,p,r,g, gains of neural elements.

$$H_2(s)=\frac{p}{1-bpk_4e^{-sd_4}}C(s) \quad (5)$$

Therefore, from(3), (4), and (5),

$$G_F(s)+G_C(s)=\frac{p(1-bk_3e^{-sd_3})}{1-bpk_4e^{-sd_4}}F(s)+\frac{p}{1-bpk_4e^{-sd_4}}C(s) \quad (6)$$

$H_1(s)$ and $H_2(s)$ can be simplified by using the expanded from of e^{-sd} and neglecting higher order terms:

$$e^{-sd}=1-sd+\frac{(sd)^2}{2!}-\frac{(sd)^3}{3!}+ \quad (7)$$

Since $d=5ms$, $sd\ll 1$ for $\omega<100$ rad/s($f<16$ Hz) where $s=j\omega$. Therefore, for movement less than 10 Hz, e^{-sd} can be approximated as follows:

$e^{-sd}=1-sd$. So substituting $1-sd$ for the exponential terms in(6), we get

$$G_F(s)+G_C(s)=\frac{p(1-bk_3(1-sd_3))}{1-bpk_4(1-sd_4)}F(s)+\frac{p}{1-bpk_4(1-sd_4)}C(s)$$

$$=\frac{k_3d_3}{k_4d_4}\cdot\frac{s+(1-bk_3)/bk_3d_3}{s+(1-bpk_4)/bpk_4d_4}F(s)+\frac{1}{k_4d_4}\cdot\frac{1}{s+(1-bpk_4)/bpk_4d_4}C(s) \quad (8)$$

$$=K_1\cdot\frac{s+\alpha}{s+\beta}F(s)+K_2\cdot\frac{1}{s+\beta}C(s) \quad (9)$$

$H_1(s)$ can be recognized as the transfer function of lead-lag network. Purkinje cell activity will lead granule cell output if $\alpha<\beta$ and will lag granule cell output if $\alpha>\beta$, we assume that $d_3\doteqdot d_4$, these relationships will reduce to:

lead network if $k_3>pk_4$ and

lag network if $k_3<pk_4$

On the other hand, $H_2(s)$ can also be recognized as the transfer function of lead-lag network. When the climbing fiber input stimulate to the Purkinje cell directly, the transfer function has only a real zero, we assume that $d_4\doteqdot d_6$.

Therfore, the model predicts that if purkinje cell collaterals have a weak effect on the basket and stellate cells, this output stage of the cerebellar cortex will act as a lead compensator and, if they have a strong effect, it will act as a lag compensator. In fact, if the effective coupling(k_4) between Purkinje axon collaterals and basket cells is very small, then the fiber order pole in(10)

$\beta=\frac{1-bpk_4}{bpk_4d_4}$ would be quite large and would lie well outside the physiological range of movement. In this case, high frequency behavior of this system would be domained by the membrane dynamics of the various cells, which have been neglected in this model. On the other hand, since electrical stimulation of parallel fibers F(s) leads to an increase in the discharge rate of Purkinje cells followed by a prolonged pause in Purkinje cell activity[3], the inhibitory coupling between parallel fibers and Purkinje cells(bk_3) is very effective and zero of $H_1(s)$ from(10)

$\alpha_1=\frac{1-bk_3}{bk_3d_3}$ would be quite small as bk_3 approach 1. Significant low frequency dynamics in this system could, therefore, be produced by the zero of $H_1(s)$. The climbing fiber input is very effective and the zero of $H_2(s)$ from(10)

$\alpha_2=-1/d_6$ would be a real zero constant. Especially, if the effective coupling(k_4) between Purkinje axon collaterals and basket cells approaches to the infinite, then the transfer function approaches 1.

So we feel that at low frequencies the dynamics produced by time delays are more significant than those produced by membrane phenomena. The transfer function of the entire cerebellar cortical model is shown below.

$$G_1(s)=\frac{rH_1(s)-rgk_1e^{-sd_1}\cdot H_1(s)}{1-(-rgk_2e^{-sd_2}+H_1(s)(rgk_5e^{-sd_5}))}M(s) \quad (11)$$

$$G_2(s)=\frac{k_6e^{-sd_6}\cdot H_2(s)(1+rgk_2e^{-sd_2})}{1-rgk_2e^{-sd_2}+rH_2(s)gk_5e^{-sd_5}}C(s) \quad (12)$$

From(11) and (12),

$$G(s)=G_1(s)+G_2(s)$$
$$=\frac{rH(s)-rgk_1e^{-sd_1}H_1(s)}{1-(-rgk_2e^{-sd_2}+H_1(s)(rgk_5e^{-sd_5}))}M(s)$$
$$+\frac{k_6e^{-sd_6}H_2(s)(1+rgk_2e^{-sd_2})}{1-(-rgk_2e^{-sd_2}+rgk_5H_2(s)e^{-sd_5})}C(s) \quad (13)$$

From using the approximate from of e^{-sd}, so that

$$G(s)=H_1(s)\cdot\frac{r-rgk_1(1-sd_1)}{1+rgk_2(1-sd_2)-H_1(s)rgk_5(1-sd_5)}M(s)+$$
$$H_2(s)\cdot\frac{k_6(1-sd_6)(1+rgk_2(1-sd_2))}{1+rgk_2(1-sd_2)-rgk_5H_2(s)(1-sd_5)}C(s) \quad (14)$$
$$=H_1(s)\cdot rgk_1d_1\cdot\frac{s+(1-gk_1)/gk_1d_1}{1+rgk-rgk_2d_2s+H_1(s)rgk_5d_5(s-1/d_5)}M(s)$$
$$+k_6H_2(s)\cdot\frac{(1+rgk_2-rgk_2d_2s-d_6s-rgk_2d_6s+rgk_2d_2d_6s^2)}{1+rgk_2-rgk_2d_2s-rgk_5H_2(s)+rgk_5d_5H_2(s)s}C(s) \quad (15)$$

and since $d_2s\ll1$, $1+rgk_2-rgk_2d_2s\doteqdot1+rgk_2$. Therefore,

$$G(s)=H_1(s)\cdot rgk_1d_1\cdot\frac{s+(1-gk_1)/gk_1d_1}{1+rgk_2+H_1(s)rgk_5d_5(s-1/d_5)}M(s)+$$
$$\frac{-k_6d_6H_2(s)(1+rgk_2)\cdot(s-1/d_6)}{1+rgk_2+rgk_5d_5H_2(s)(s-1/d_5)}C(s) \quad (16)$$

If the Purkinje cell feedback to the Golgi cell is very weak, i.e., $k_5\to0$, then the transfer functions, $G_1(s)$ and $G_2(s)$, reduce to as follows, respectively.

$$G_1(s)=\frac{rgk_1d_1}{1+rgk_2}\cdot(s+(1-gk_1)/gk_1d_1).H_1(s).M(s) \quad (17)$$

which is a lead network cascaded with the lead-lag network $H_1(s)$. The amount of lead that is added is determined by g, k_1, and d_1.

$$G_2(s)=-k_6d_6(s-1/d_6)\cdot H_2(s)\cdot C(s) \quad (18)$$

The amount of lead that is added is determined by d_6. From(17) and(18),

$$G(s)=\frac{rgk_1d_1}{1+rgk_2}\cdot(s+(1-gk_1)/gk_1d_1)\cdot H_1(s)$$
$$M(s)+(-k_6d_6(s-a/d_6).H_2(s)\cdot C(s) \quad (19)$$

If the collateral feedback is very large, i.e., $k_5\to\infty$ from(16), then the transfer functions, $G_1(s)$ and $G_2(s)$, reduce to as follows, respectively.

$$G_1(s)=\frac{k_1d_1}{k_5d_5}\cdot\frac{s+(1-gk_1)/gk_1d_1}{s-1/d_5}M(s) \quad (20)$$

which, although unstable, can act as a non-minimum phase compensator when it is used in a feedback circuit. If k_5 is somewhere between these two extremes, the model predicts a double zero, double pole transfer function for the cerebellar cortex.

$$G_2(s)=\frac{-k_6d_6(1+rgk_2)}{rgk_5d_5}\cdot\frac{(s-1/d_6)}{(s-1/d_5)}C(s) \quad (21)$$

From(21), transfer function, $G_2(s)$, approaches to 1 if $d_5=d_6$. This means that pole and zero are always constant value, 1. Therefore, there is no effective on lead-lag network to cerebellar cortex.

From(20) and (21),

$$G(s)=\frac{k_1d_1}{k_5d_5}\cdot\frac{s+(1-gk_1)/gk_1d_1}{s-1/d_5}M(s)$$
$$+\frac{-k_6d_6(1+rgk_2)}{rgk_5d_5}\cdot\frac{(s-1/d_6)}{(s-1/d_5)}C(s) \quad (22)$$

4. Discussion and Conclusion

It can be seen that the predicted values will be well fit, except at low frequency from the respective transfer functions, $H_1(s)$, $H_2(s)$, (11), (12), (16), (19), and (22). At low frequencies, predicted phase data lead the normal value. This phase discrepancy can probably be adequately explained by the phase lead, due to cupular dynamics, observed at the endorgan at low frequencies of stimulation[5]. It should be noted that absolute identification of unit type, Purkinje, granule, mossy fiber, etc., could not be made. However, we may safely eliminate some unit types from this discussion.

On the other hand, the time constants due to the membrane dynamics of cerebellar cells are short compared to the input frequencies used. At high frequency used in this study (1.0 Hz), the straightthrough time delayed neglected in the model would add less than 2° of lag and should, therefore, not influnce the parameter estimation significantly.

Finally, the cerebellum could match its dynamics to a particular control system it is involved in by varing the strengths of its coupling coefficients. We are now in position to examine the sensitivity of the break points, for example, $\omega=(1-bk_3)/bk_3d_3$, to changes in the strength of the inhibitory coupling, in this case bk_3.

Assuming a delay(d_3) of 5.0 ms, the variation in as bk_3 changes from 0.99 to 0.90, and also, according to the model, Purkinje axon collaterals would add poles to the cerebellar network, which would decrease the phase lead seen in this study.

REFERENCES

1) D. Marr, "*A Theory of the Cerebellar Cortex,*" *J. Physiol., Vol.22, pp.88-93, 1969.*
2) J.A. Mortimer, "*A Cellular Model for Mammalian Cerebellar Cortex,*" *Comput. and Commun. Sci. Dept., Univ. Michigan, Ann Arbor, Rep. 032967-T, October, 1970.*
3) J.C. Eccles, M. Ito and J. Szentagothai, *The Cerebellum as a Neuronal Machine. New York: Springer-Verlag, 1967.*
4) T.W. Calvert and F. Meno, "*Neural Systems Modeling Applied to the Cerebellum,*" *IEEE Trans., Syst., Man, Cybern., Vol. SMC-2, pp. 363-374, July, 1972.*
5) D.C Higgins, L.D. Partridge and G.H. Glaser, "*A Transient Cerebellar Influnce on Stretch Responses,*" *J. Neurophysiology, Vol. 25, pp. 684-692, 1962.*
6) D. Larsell, "*The Cerebellum of Reptiles: Chelonians and Alligator,*" *J. Comp. Neurol., Vol. 56, pp.299-345, 1932.*
7) J.V. Walsh, J.C. Houk and E. Mugnaini, "*Identification of Unitary Potentials in Turtle Cerebellum and Correlations with Structures in Granular Layer,*" *J. Neurophysiol., Vol. 37, pp.30-47, 1974.*
8) J.V. Walsh, J.C. Houk, P.L. Atlieri and E. Mugnaini, "*Synaptic Transmission of Single Glomeruli in the Turtle Cerebellum,*" *Science, Vol. 170, pp.881-883, 1972.*
9) J.C. Eccles, "*The Cerebellum as a Computer: Patterns in Space and Time,*" *J. Physiol., Vol. 229, pp.1-32, 1973.*
10) T. Nagano and O. Ohmi, "*Plausible Function of Golgi Cells in the Cerebellar Cortex,*" *Biol. Cybernetics, Vol. 29, pp.75-82, 1978.*

□ 국문초록 □

소뇌의 신경회로망 해석을 위한 운동제어계의 실현

이 명 호

소뇌의 운동제어특성을 해석하기위한 시뮬레이션 모델을 소뇌의 소뇌피질을 중심으로 하는 두 입력방법의 소뇌 운동제어시스템으로 구성하였다. 소뇌피질의 다섯신경세포의 이득을 임의의 상수계수로 하고 이들 신경세포와 두 입력신호에서 비롯되는 결합계수와 그 크기를 정의하고 이들이 target cell에 미치는 영향을 전달함수로 유도하고 이에 필요한 보상방법을 제시하였다.