MODELLING THE CONTROL PROPERTIES OF THE BASAL GANGLIA

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INTRODUCTION

The precise role of the basal ganglia in human motor activity is not yet fully resolved. A popular school of thought advocates the notion of learned motor "programs" which are hypothesised to be recorded in the basal ganglia (Tatton and Bruce, 1981; Marsden, 1982). The complexity of these "programs" is, however, a subject of controversy. This chapter is an attempt to analyse simple motor activity and to infer the possible controller role of the human basal ganglia.

The tool used for the quantitative assessment of motor ability, a computer-based visual tracking device, will be described. A mathematical model of the relevant control loops involved in the control of simple motor activity will then be introduced. Finally, the results of measurements with a control group and with patients suffering from Parkinson's disease will be presented and discussed.

QUANTITATIVE ASSESSMENT OF MOTOR ABILITY

The standard neurological examination is the classical method of assessing the sensory and motor system functions (De Jong, 1967). This examination consists of subjective evaluations of strength, sensory and motor co-ordination, equilibrium, gait, sensory perception, mental state, language, reflexes and the cranial nerves. The examiner studies the patient for signs and symptoms of disease and judges the extent of impairment. The overall impression the examiner acquires from each neurological category is the so-called neurological function of the patient.

It has to be stressed that the judgments of factors like speech, facial expression, touch and pressure sensation are necessarily subjective. A given function is then assigned a vaguely defined category, such as "mildly abnormal" or "moderately abnormal" to assess sensory and motor functions better and each category is enumerated. This kind of assessment is called an ordinal scale rating. It is simple and very quick to perform. Although such a scale can be used for initial evaluation and differential diagnosis of a neurological disorder, the rating categories are often too broad and vaguely defined to detect effects of medication or small changes in the
patient over a longer period of time. It also has to be mentioned that, in addition to differences in the rating techniques of different neurologists, the rating style of the same examiner may change over time and with experience. Therefore, direct comparisons— one of the primary motivations behind rating scales— may become worthless or even impossible to carry out.

Many rating scales have been developed and employed to assess parkinsonian deficits over the years. The most commonly used rating scales include the evaluation methods introduced by Hoehn and Yahr (1967), Webster (1968), Duvoisin (1971), and Marsden et al. (1973).

One of the methods of measuring the neurological and motor functions is to evaluate the tracking performance of patients (Velasco and Velasco, 1973; Flowers, 1976; Baroni et al., 1983; Wing and Miller, 1984). Tracking can be defined generally as the problem of making the output of a controlled process correspond as closely as possible to the reference input (Poulton, 1974; Sheridan and Ferrel, 1974). Two principal types of tracking task can be defined:

(i) closed loop systems, where the behaviour of the process which is to be controlled is fed back to the human operator visually and/or through somatosensory feedback; and

(ii) open loop systems, where the process output is not fed back to the human operator. In many cases the absence of visual information about the behaviour of the process is enough to interrupt this feedback.

Differences between closed loop and open loop tracking performance can represent valuable diagnostic data in many cases. Therefore, these two principal types of tracking tasks reflect the neurological tests used in clinical examination to assess sensory and motor function as well as co-ordination.

A VISUAL TRACKING DEVICE

The configuration depicted in Fig. 1 is a simple device with which open and closed loop pursuit tracking tests can be performed (Hacisalihzade, 1986).

Since motor tasks can be performed using different motor strategies, that is the co-ordinated activation of different muscles, measurements using movements with several degrees of freedom are usually difficult to analyse. The measurements discussed in this chapter use the displacement of the end phalanx of the thumb to perform the tracking tasks. This movement has a single degree of freedom and the peripheral control mechanisms have been analysed in detail (Marsden et al., 1976).

A single digital process computer (PDP 11/23) is used in the measurements

- to drive the stimulus of the tracking task (real time/on line);
- to record the tracking performance of the subject (real time/on line); and
- to analyse the recorded tracking data (off line).

The end phalanx of the thumb is tightly strapped to the shaft of a direct current motor. The angular position of the shaft is measured by a potentiometer. An individual plaster of Paris cast is used to ensure that the hand rests in its most relaxed form. Potentials of relevant muscles can be checked by means of EMG to ensure that the tracking is performed by the flexion and extension movements of the thumb. Through adjustment
Fig. 1. Picture of the device with which tracking tasks can be performed. (a) armrests, (b) hand platform, (c) motor, (d) thumbscrew, (e) potentiometer measuring the position of the thumb, (f) button to start the experiment, (g) power amplifier, (h) overheating surveillance, (i) D/A and A/D converters.

of the shaft's position with respect to the fixed position of the hand in the cast, it is made sure that this movement is executed through the activation of the extensor and flexor pollicis longus muscles alone and not by thenar and forearm muscles.

The reference input of the tracking task is a small target which moves on the vertical axis of an oscilloscope screen. The task is to move a point of light up and down on the screen so that it stays in the middle of the target. The position of the point of light corresponds to the angular position of the shaft which can be rotated by the movements of the end phalanx of the thumb.

The motor can produce disturbances of the thumb position by applying a variable torque. It is possible to perform measurements with this configuration with or without disturbing torque as well as measurements in which the actual position of the thumb is visible only for a fraction of the time (visual or somatosensory open loop conditions). Fig. 2 shows the functional diagram of the measurement configuration.

A MATHEMATICAL MODEL OF HUMAN MOTOR CONTROL MECHANISMS

The model of the major afferent and efferent connections shown in Fig. 3 assumes a control loop which has the block-diagram depicted in
Fig. 2. Schematic diagram depicting the experiment configuration.

Fig. 3. Major afferent and efferent connections in the brain.
Fig. 4. Block diagram of the control loop which is affected in Parkinson's disease.

Fig. 4. In this model the "idea" for a motor action is processed in the cortex with a time delay and first order dynamics. It is then further processed in the basal ganglia and conveyed to the periphery (which is also modelled as a delay with a first order low-pass) for the execution of the desired action. The input of the cortical processing is modified continuously during the execution of the action by comparing the "idea" with the visual and somatosensory feedback of the present state of the execution. The basal ganglia itself is also modelled as a control loop, where both the pars reticulata of the substantia nigra and the striatum are modelled as first order systems and where the pars compacta of the substantia nigra has a PD-controller role over the striatum.

Obviously this highly complex and high order model cannot be verified. Therefore, it makes sense to simplify it. The result of this simplification is shown in Fig. 5. In this simplified model, the "idea" is processed with a delay in the cortex and then further processed in the basal ganglia before being sent to the periphery, which is also modelled as a simple delay. The input of the cortical processing is modified continuously also in this model during the execution of the action by comparing the "idea" with the visual and somatosensory feedback of the present state of the execution. The basal ganglia itself is again modelled as a control loop, where the substantia nigra acts as a PD-controller on the striatum.

RESULTS

It is now possible to verify the model in Fig. 5 and to identify the parameters of this system. A series of measurements was conducted with nine controls and eleven parkinsonians. In each measurement session, tracking performances of the subjects were recorded as step responses to pseudo-random square waves. The resulting step responses (42 for each subject) were then averaged for every individual.

Fig. 6 shows the comparison of the measured and modelled step responses of (a) an average control and a typical parkinsonian (b) with and (c) without
Fig. 5. Simplified model showing the controller characteristic of the substantia nigra.

Fig. 6. Modelled and measured step responses of (a) a control, (b) a parkinsonian in "off" state and (c) a parkinsonian in "on" state.

medication ("on" and "off" states). The dashed curves in (a) and (c), as well as the dotted curve in (b), are the results of simulation with identified values of parameters substituted in the model shown in Fig. 5. The continuous curves in (a) and (c), as well as the dash-dotted curve in (b), are the results of averaged measurements.

Table 1 shows the results of identification of parameters of the model shown in Fig. 5. The standard deviations are shown in brackets below the mean values of the identified parameters.

It was observed that the time constant of the striatum ($T_s$), which was modelled as a first order low-pass, was not significantly different in the controls and the parkinsonians with or without medication. This is in agreement with the fact that the striatum is not influenced in Parkinson's disease. It was also observed that the total delay in cortical processing and in the periphery ($T_1 + T_2$) (which cannot be identified separately) is
Table 1. Identified Parameters of the Model Depicted in Fig. 5 for Nine Controls and Eleven Parkinsonians

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Controls &quot;off&quot;</th>
<th>Parkinsonians &quot;on&quot;</th>
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<tbody>
<tr>
<td><strong>$T_S$</strong> [s]</td>
<td>0.069 (0.019)</td>
<td>0.071 (0.018)</td>
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<tr>
<td></td>
<td></td>
<td>0.067 (0.022)</td>
</tr>
<tr>
<td><strong>$T_1 + T_2$</strong> [s]</td>
<td>0.251 (0.087)</td>
<td>0.257 (0.094)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.243 (0.117)</td>
</tr>
<tr>
<td><strong>$K_p$</strong> [-]</td>
<td>0.018 (0.007)</td>
<td>0.157 (0.054)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.049 (0.027)</td>
</tr>
<tr>
<td><strong>$K_d$</strong> [s]</td>
<td>0.195 (0.071)</td>
<td>1.243 (0.347)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.277 (0.121)</td>
</tr>
<tr>
<td><strong>$K_f$</strong> [-]</td>
<td>0.001 (0.001)</td>
<td>- (0.001)</td>
</tr>
</tbody>
</table>

about the same in all groups, which is also in agreement with previous findings about the reaction time in parkinsonians not being affected by the disease.

However, the significant differences in the proportional and the derivative feedback gains ($K_p$ and $K_d$) between the control group and the parkinsonian population supports the conclusion that the basal ganglia actually have a PD-controller characteristic and that the neuronal depletion which causes Parkinson's disease actually increases the values of these gains significantly (5 to 10 times). This makes fast tracking impossible, thus rendering the parkinsonians bradykinetic. Furthermore, the effect of the medication is such that the controller gains get closer to their normal values with adequate medication, which is also reflected in Fig. 6.

Two important questions which have to be answered by further research concern whether a correlation exists between the increase in the values of controller gains and the stage of the disease, and whether a decoupled parallel controller model - in which a separate simple model, as described above, can be employed for every single degree of freedom - can be used for movements combining several degrees of freedom.

CONCLUSIONS

Starting with a physiological model, a mathematical model of the afferent and efferent connections relevant to the control of motor activity has been derived. This model has then been simplified. Measurements based on simple visual tracking tasks with a single degree of freedom have been performed on controls and parkinsonian patients with defective basal ganglia. These measurements have been used to verify the derived mathematical model which explains the control properties of the basal ganglia. Parameter identification of this model has shown no significant difference in the parameters of the striatum, cortical processing and periphery between both groups, whereas significant differences were observed in the substantia nigra parameters between the controls and the parkinsonians. These results confirm a simple PD-controller role of the substantia nigra over the striatum during motor action with a single degree of freedom.
ACKNOWLEDGEMENT

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REFERENCES