UNIVERSITY OF MANITOBA

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Ying Cheng

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BY

YING CHENG

A dissertation submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements of the degree of

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To Dr. Terrence P. Hogan, whose dedication to education helped shape the course of my pursuit for knowledge.

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ABSTRACT

Using the concept in control theory, conditioned behaviour in general and the delayed conditioned approach behaviour in particular are viewed as optimally controlled processes. As such learned habits are subject to quantitative analysis as continuous processes in the time domain. The characteristics of a behavioural system are described by its relative error to a baseline level, its phase velocity and the phase plane diagrams. It enables the analysis of behaviour as on-going active processes, rather than as discrete correlational entities.

The delayed conditioned approach paradigm is essentially a servomechanism-like paradigm. It can readily be studied in the context of control theory analysis. It was used to study the role of the frontal striatal system and the cerebellar control system in temporal and delay conditioning.

64male adult Albino rats were trained in the delayed conditioned approach behaviour and then divided into 9 groups. Four experimental groups received bilateral electrolytic lesions either to the frontal pole, the head of the caudate nucleus, the posterior caudate nucleus or and the dentato-interposed nuclei respectively. There were a corresponding 4 sham lesioned groups and 1 normal control group.

Results showed that bilateral electrolytic lesions in the head and the posterior aspect of the caudate nucleus produced temporary impairment in the initiation

of conditioned approach paradigm. Subsequently, the head of caudate nucleus lesioned group evolved into a hard limit cycle, no longer sensitive to changes in reinforcement contingencies. In contrast the posterior caudate nucleus lesioned group showed functionary recovery from an initial impairment on the DCA. The group with frontal pole lesions showed temporary shift toward shorter response latencies, suggesting the posibility of hyperreactivity to CS presentations. Lesions in the dentato-interposed nucleus produced permanently ataxic Ss, but their performance on the conditioned approach paradigm was essentially the same as those of the normal control Ss.

The results were discussed with references to the concept of act inhibition, the initiation and control of conditioned behaviour and of possible motivational variables.

Chapter 1

Review of Literature

Traditionally, the relationship between brain structures and behaviour relies mainly on techniques which assume that behavioural systems are discrete, static entities. Inter actions between the central nervous system (CNS) and behaviour are described in correlational terms.

Alternatively, the application of control theory and techniques allows behavioural systems to be studied as servomechanism-like systems which are in the continnual process of establishing active stability and equilibrium.

while correlational data yield abundant information on the modes of CNS function and behaviour , they do not provide adequate information where behaviours are essentially viewed as on-going, active processes. The study of timing function,ie. temporal discrimination and delay conditioning (eg. Skinner, 1938, 1946; Wilson and Keller, 1953; Laties, Weiss, Clark and Reynolds, 1965; Reynolds , 1966) is one of the topics in point.

Review of recent literature suggests the existence of two major hypotheses concerning the neurophysiological substrates underlying delay response and temporal discrimination. One hypothesis states that 'timimg' is a function of the cortical inhibitory processes (hypothesis I), while the other postulates that 'timimg' is a function of the cerebellar postural mechanisms (hypothesis II).

The present study attempts to study the relative role of the two proposed neural mechanisms. Using the control theory approach anaylsis, it attempts to evaluate the characteristics of the cortical and cerebellar neuronal systems as active, on-going processes in their adjustments to various levels of demand in ' timing ' of behaviour.

Historical Background

Literature supporting hypothesis I : delayed respon and temporal discrimination as a function of cortical inhibitory processes.

The 1936 paper published by Jacobsen precipitated the search for the neurological mechanisms for prefrontal lobe-lesion-induced impairment on delayed response tasks. After bilateral prefrontal lobectomies, his monkeys showed a severe and permanent impairment on Hunter's delayed response test in the Wisconsin General Test Apparatus(WGTA), delayed alternation, go-no-go tests, sensory discrimination, discrimination reversal, and to a lesser extent, avoidance training. The majority of results favors the concept of the "frontal lobe system" proposed by Rosvold and Szwarcbart (1964)which included the dorsolateral prefrontal cortex, the anteriodorsal aspect of the head of the caudate nucleus, the subthalamus (and possibly the hippocampus) as components of the cortical inhibitory system responsible for delayed

response performance (Battig ,Rosvold and Mishkin, 1962;Divac,1968(a),(b); Divac ,Rosvold and Szwarcbart, 1967; Fox,Kimble & Lickey,1964; Gross,1963(a), hompsom,(d); Gross et al,1962,1964; Hansing Schwartzbaum and Thompscn,1968; Konorski ,1959,1961,1964,1967; Mishkin ,Prockop and Rosvcld ,1962; Pribrim,1955; Rosvolt & Szwarcbart,1962,1964; Stamm,1968,1969,1970; Stamm and Rosen, 1969; elc). It should be noted, however, that Hunter's "delayed response" involves restraint of S by the experimenter as well as self-restraint. It thus is somewhat different from Pavlovian DR.

Anatomical evidence

Anatomical studies have shown the existence of neuronal pathways among these cortical and subcortical structures. In the young and adult albino rats, Webster (1961) performed localized cortical ablations via suction or thermoccagulation and subsequently traced the pattern of retrograde degeneration traced, using the Golgi rapid method, Marchi's method and the Nauta method. Webster showed that all rats with cortical ablations exhibited striatal degeneration. "Changes in the position of the lesion produce changes in the locations of the region of the striatum found to contain degeneration. The striatal degeneration is more widespread dorsally than nearer the pallidum". There were no axonal termination in the pallidum. In addition,the striatal degeneration was considered terminal because the Nauta & Gygax (1954)

method showed complex nests of degenerating axons around striatal cells. Some of the cortico-striate fibers were collaterals of axons in the internal capsule. Most importantly, Webster described that the cortico-striate projection is topographically well organized in both anterior posterior and mediolateral planes, and is derived from widespread areas of the cortex, probably including the auditory and visual areas. With cats Webster (1965) identified a similar arrangement with the exception that the rat the striatum is not divided into caudate nucleus and putamen, while the cat striatum is. In both studies the author failed to observe striate-cortical projections. Similar results were found in monkeys by Nauta(1964), and Burandt (1961). Kamp and Powell(1970) studied the cortico-striate connections in adult macaque monkeys. Using Nauta technique, they found that: (1) all parts of the cortex send fibers to the striatum in topographically well organized fashion. In the frontal lobe the cortex of the medial surface projects dorsally in the striatum, that of the lateral surface laterally and the orbital surface medially. (2) Posteriorly the arrangement is modified in relation to the development of the temporal lobe. (3) Projections from the somatosensory and motor area is large while that from the visual area is the smallest. (4) The sensorimotor and frontal asscciation cortex are related to the caudate nucleus (and, in particular, to the large part of the head and the parietotemporal lobe to the body and

tail of the nucleus. Kemp specifically pointed out that "there is topcgraphic projections but multiple innervation from different cortical areas upon one striatal region". Kemp et al's (1970;1968) studies are in agreement with thcse by Carman et al (1965), Nauta (1964) and Divic et al (1967).

Indirectly, Mettler, Hovde and Grundfest (1952), and Harman (1954) found a decrease in the volume of the caudate nucleus in monkeys after extirpation of the premotor and orbital cortex. But Ermolenko(1969) was the first to report direct striato-cortical projections. Using cats as subjects, Ermolenko performed electrocoagulaticn of the rostral part of the head of the right caudate nucleus. The brains were fixed in 10% formaline and sectioned ten days later with Nissl's method. He found that there is direct projection from the rostral part of the head of the caudate nucleus to the prefrontal orbital, anterior limbic 32 and secondary somatosensory cortical areas. Preterminal degeneration of axons from the head of the caudate nucleus was found in the ipsilateral cortical area 6. Destruction of the rostrocentral part of the head produced degeneration in the anterior part of sigmoid gyrus. Destruction of the rostro-medic-ventral part of the head produced degeneration in the anterior part of area 4. Projection of the dorso-medial and central areas of the rostral part of the head of the caudate on the frontal cortex partially

overlap. The caudate part of the head had more extensive cortical connections as destruction of the area caused degeneration of large number of fibers of medium and thin caliber and of the thinnest preterminals in the premotor, motor orbital, secondary somatosensory and limbic cortical areas. But the author felt that a more extensive study of the caudal part is needed. These findings suggested that impulses from the caudate nucleus may reach the cortex directly by-passing the intermediate subcortical structures ,ie., putamen, globus pallidus and nonspecific thalamic nuclei. "It definitely extends the concept of integraton between caudate nucleus and cortex" (Ermolenko,1969).

Anatomical evidence for the existence of frontothalamic and of frontosubthalamic projections were also present. Nauta(1964) observed prefrontothalamic degeneration in the nonspecific thalamic nuclei, the reticular nucleus and the midline region of the intralaminar thalamic complex. Johnson (1961); Johnson & Clemente (1959), and Szabo (1962) all showed that the pallidum projects to the ventral lateral thalamic nucleus. However the existence of a direct caudato-thalamic connection is not well documented at present (Buchwald and Hull,1967). From the subthalamus there is a fiber passage reaching the rostral midbrain tegmentum especially these anatomical considerations, I shall turn to behavioural studies concerning the effects on delayed

response via various experimental manipulations of these cortical and subcortical structures. More specifically, emphasis shall be on the "inhibitory processes" and delayed responses.

Behavioural Task

Konorski and his colleagues have devoted over ten years in studying the relationship between frontal lobe damages and performance in the delayed response paradigm (Lawika, 1957, 1959; Konorski and Lawika, 1959; Lawika and Konorski, 1959; Konorski 1961a, 1961b; Konorski and Lawika, 1964; Konorski, 1967). The general procedures of the delayed response paradigm of this method are as follow: in the first phase the animal is taught to receive food in two or more places; and in the second phase, the location of food is signalled to the subject prior to the start of the trial. Both the accuracy and the speed of responding are controlled by the temporal interval (ie. the delay) between the offset of the signal and the onset of the trial. In most delayed response procedures the animal is confined in the starting platform. A stimulus is given indicating the location of reward. In some experiments such a stimulus is provided by baiting one of the food bowls in front of the animal. Alternatively visual, auditory or kinesthetic cues are utilized in the delayed alternation procedure in which the preceding response is a cue for the next response. Konorski's analysis of the delayed response paradigm asserts that

the instrumental response of approaching the appropriate food source is controlled by compound CSs. One element of the compound CS is a trace of the preparatory stimulus while the other elements are the releasing stimulus common for all instrumental responses. The most important facts obtained can be summarized into five points: (1) in normal dogs and cats the correct response occurs regardless of assumed bodily orientation during the delay interval; (2) the correct delayed responses in normal animals generally can occur after a delayed period of as long as three minutes for cats and of ten minutes or more for dogs. (3)distractions introduced during the delay period do not generally impair the delayed response performance; (4) the delayed response remain correct when there is a screen around the starting platform to block the subject(s)'s view of the reward cups; and (4) in the double signal test in which two feeders are signalled in immediate succession in the same trial, Ss usually approach the more recently signalled feeder first. Frontal lobe lesions in the area of prefrontal poles rostral to the presylvian sulcus produced severe impairment of the delayed response. Correct performance became secondary to a correct body orientation during the delay period, and with introduction of distraction, correct responce is reduced to chance level. Until 1961, Konorski considered the delayed responses a form of Pavlovian trace conditioning, and he attributed the impairment to the destruction of brain area concerned with recent

memory of the directional cues. In addition to discussing the role of prefrontal-lesion-induced damage to Pavlovian inhibitory CR's (1957,1959), Konorski assumed that profrontal lesions also lead to a impairment of the neuronal mechanism of reverberating circuits in general and therefore of all cortical inhibitory processes (Konorski, 1961 b). Thus, before the end of 1961, Konorski's hypothesis concentrated on the cortical inhibitory processes, frontal lobe lesion and delay response. Since then, Konorski and colleagues have developed another hypothesis to account for the effect of frontal lobe lesion on delay response, namely that of kinesthetic gnosis of spacial relations (1967). This hypothesis shall be discussed in a later section.

Lesion Experiments

In North America, the concept of act inhibition was first put forth by Stanley and Jaynes (1949) and they attributed the frontal-lobe lesion induced behavioural impairment to the damage in the neural mechanism concerning act inhibition. As quoted by Brutkowski (1963, 1964, 1965), B. Shustin showed that in dogs with frontal lobe damage the previously trained CRs are often replaced by a variety of spontaneous motor unconditioned reflexes. In Brutkowski's experiment, he trained dogs to place the right forelimbs on the food tray on presentation of a CS in a conditioned reflex room. After large frontal lesion involving the precruciate area, the S executed the CR

continuously even in the absence of the CS. "Presumably a release mechanism accounts for these motor manifestations, since they all were clearly observable in the preliminary period of testing" (Brutkowski, 1965).

Konorski (1961) summarized the work on act inhibition from their laboratory. Dogs were trained to perform various types of excitatory and inhibitory CRs. The prefrontal lesion including the frontal poles rostral to the presylvian sulcus (i.e., gyrus proreus, gyrus subproreus and the anterior part of gyrus orbitalis and the subjacent white matter) were then removed. Postoperative testing showed that positive CRs were either normal or enhanced while inhibitory CRs were disinhibited. This was the case for both alimentary and defensive CRs. This effect is attributed to impairment of internal inhibition, which, according to Konorski, was based on the formation of inhibitory connections between the central representations of the CS and UCS, alongwith the excitatory connections formed earlier . Also prefrontal lesions may either produce relatively pure drive disinhibition, when the lesion is limited to the frontal poles, or to motor response disinhibition when the lesion encroaches upon the precruciate area.

Brush et al, (1961) trained rhesus monkeys on a series of 11-trial object discrimination task presented at the rate of three problems per day under two different conditions of testing in a modified Wisconsin General

Test Apparatus. In the "baited" condition, the choice of one object of the pair on the first trial was always associated with a reward, and on the remaining 10 trials the S was required to choose the same object. In the "unbaited" condition the choice of an object of the pair in the first trial was never associated with a reward, and on the subsequent 10 trials the S was required to choose the other object. While no impairment occurred in the "baited" condition, the frontal Ss performed poorly on the "unbaited" problem. "On the assumption that the object choices on the first trials were determined by aversions and preferences for one object over the other, the poor performance in the "unbaited" condition was ascribed to the frontal animals' great persistence or inertia of their initial sets". Brush et al., thus suggested that animals with frontal ablations had difficulty in withholding any strong approach tendency resulting from training or preference, thereby perseverating on one response to the exclusion of others. Impairment on differentiation and delayed response tasks were also attributed to the same effects (Battig et al, 1960, 1962; Brush et al, 1961; Brutkowski et al, 1963; Mishkin, 1964).

Battig, Rosvold and Mishkin (1960) tested monkeys with caudate and frontal lesions in delayed alternation and delayed response tests. The delayed response training consisted of two phases. In phase I there was

neither a delay nor a screen. A peanut was placed in the left or right foodwell. Then both wells were covered with neutral-gray cardboard plaques and the animal was allowed to make a choice. In phase II, there was no delay but a screen was introduced and in phase III, both screen and delay were introduced. Delay was gradually increased from two to ten seconds in steps of 1 seconds. Bilateral lesion of dorsolateral frontal cortex and of the head of the caudate nucleus both produced impairment on the two tests employed, but the magnitude of deficit was greater with the frontal group. In addition tests on the alternation in the automatic apparatus were most severely impaired, an effect which the authors attributed to the relatively smaller number of stimulus cues in the test situation. Ahsimilar trend of results was observed in a go-no-go discrimination task (Battig et al, 1962). In the 1962 study the authors studied the comparative effects of bilateral frontal and of the head of caudate nucleus lesion on WGTA go-no-go auditory discrimination in adult rhesus monkeys, with a view to find out if discrimination and delayed response were mediated via the same neural mechanism. In the testing situation Ss were presented with a positive 1000 Hz tone or a negative white noise. The subject was trained to reach for reward covered under a 3 in. square gray cardboard within 5 sec. or to refrain from reaching with presentation of the negative CS. On colour discrimination, red signaled reward while green signaled non-reward. The results

showed that (1) both bilateral ablation of the dorsolateral prefrontal cortex and of the head of the caudate nucleus impaired DR and discrimination tasks; and (2) the former type of lesion produced greater impairment.

Mishkin (1962, 1964) studied effects of orbital frontal and lateral lesions on visual and auditory discrimination, learning set, discrimination reversal, one-trial learning and spatial reversal. Their results showed that the performance of the lateral frontal group was better than that of the orbital group on all tasks except on DR, although performance deficits were detected for both groups. The author postulated that frontal lesion produced loss of act inhibition. The greater impairment produced by the lateral lesion was an effect of both weakened act inhibition and spatial elements.

Brutkowski (1963, 1964, 1965) studied the sequelae of partial lesions in dogs. The perseveration of response was produced by lesions of the dorsolateral portion of the prefrontal cortex only. His dogs were trained in a conditioning experiment in which both positive and inhibitory trials were presented on a schedule of either 15 sec. or 1 min. intertrial intervals. After the lesion, impairment occurred only in the short interval schedule. He suggested that deficits reflected perseveration since perseveration should be greater at shorter intervals. As support for the hypothesis that the frontal cortex is an essential structure

in act inhibition, Burtkowski quoted Lukaszewska (1963, 1965). His studies showed that normal rat which had relatively little frontal cortex also had a remarkable perseveration tendency. Such a tendency, however, could be overcome with long term training, suggesting that the frontal area in rat differs from other species by exerting a suppressing action on the mechanism expressing perseveration. Furthermore, this was confirmed by the bilateral removal of the anterior tip of forebrain which produced permanent perseveration.

In addition Divac (1968) in a comparison of the relative importance of prefrontal cortex and of the head of caudate nucleus on delayed response, studied the effects of lesion in 15 adult cats. The Ss were trained to approach either the left or right feeder according to Gellerman series for reinforcement. In the delay problem the preparatory auditory CS lasted for 3 sec., after which the delay began. The S was released upon termination of the delay, and was rewarded with a correct response. The delay was introduced in steps of 0, 5, 15, and 30 sec. Upon achieving a criterion of 90% correct responses they were tested with distractions of (1) lowered hood which prevented visual contact with the surrounding, during the middle 20 sec. of the 30 sec. delay, and (2) delivering minced meat for 10 sec. during the middle of the 30 sec. delay. Ss then underwent surgery in prefrontal; anterior caudate; anterior caudate

and prefrontal; posterior caudate or no operation. Retention sessions conducted 20 days after the operations showed that (1) both frontal and caudate lesions significantly increased the number of errors in the DR test; (2) effects of the caudate lesion were greater in magnitude than those of the frontal lesion. This result challenged Rosvold's (1958), Battig's (1960, 1962) and Divac's (1967, 1968) statement that the caudate nucleus subserves the same function as the frontal input area but to a lesser degree. This apparent discrepancy was attributed by Divac to species differences -- the prefrontal cortex plays a less important role in the cat, with regard to the delayed response performance. (3) Both control and experimental Ss showed significant improvement in the course of post-operative testing sessions. Body orientation during the delay was shown to be related to error in performance as almost all incorrect orientation ended in erroneous response. However, position habit might be a consequence rather than the cause of an S's inability to solve the task (Divac, 1968; Cross and Weiskrantz, 1964). Divac further showed that even a correct body orientation did not necessarily lead to a correct choice, contradicting Lawicka and Konorski's observation in dogs (1959).

In their attempt to clarify the role of caudate nucleus in response inhibition, Fox, Kimble and Lickey (1964) performed two stage bilateral electrolytic lesion in the cats to determine if lesions of caudate nucleus

would produce performance impairment. The experimental animals were substantially inferior to the normals in their ability to inhibit the instrumental feeding response in the passive avoidance situation as indicated by failure of Ss to withhold entering the feeding box. Since the caudate Ss were not different from the normals in acquisition of an instrumental feeding response, the deficit was not in learning ability. Neither were there deficits on active avoidance behaviour. They concluded that the caudate plays a role mediating frontal and anterior limbic inhibition in passive avoidance tasks.

Chorover and Cross (1963) studied the behavioural effect of bilateral electrolytic lesion of the head of the caudate nucleus in hooded rats. Basically animals were trained in a two-lever box for food. Each lever press that followed a response to the opposite lever resulted in delivery of food pellet. In the acquisition group, the caudate Ss were markedly inferior to both posterior cortical lesioned and the normal control Ss. The same type of results were obtained with the retention group. However, the caudate animals were apparently normal in a Hebb-Williams maze learning. Thus the head of the caudate nucleus was considered a necessary structure for adequate alternation performance.

Schwartzbaum and Donovick (1968) studied the effect of septal and caudate lesions on brightness and spatial discrimination and reversal. They attempted to provide a

more precise description of the perseverative disorder subsequent to disruption of the "response inhibition". All experiments were conducted with rats in the shuttle-box type of device. The authors found that septal lesions selectively impaired reversal of spatial but not brightness, discrimination. Caudate lesions, on the other hand, disrupted brightness discrimination. That the caudate-lesioned Ss showed a higher incidence of response in the incorrect compartment was considered evidence of spatial orientation disruption. While the data agreed with their earlier results using stimulation, the authors mentioned that the tests could not rule out some commonality of operation of the two structures (Donovick and Schwartzbaum, 1966, 1968).

Goodman , Rosvold and Mishkin (1970) studied the effects of prefrontal lobectomy in infant and juvenile rhesus monkeys on the delayed response tasks. These ages were selected so that the infants were operated well before while the juveniles were operated well after the maturation of delayed-response capacity which develops in the normal monkeys between 4 - 8 months of age (Harlow et al, 1968; Harlow et al, 1960). The Ss were trained in a Wisconsin General Test Apparatus, containing a stationary test tray with two recessed wells, 13 in. apart. All subjects were trained on delayed response, delayed-alternation and visual-pattern discrimination tasks. The results showed that prefrontal lobectomy caused learning

deficits on all tasks for both groups. In the case of the delayed-alternation task, the operation produced a greater effect in the older Ss, but performance was better in the older normal Ss. The authors attributed the pattern of damage to impairment in a function mediated by the orbital cortex, which, in contrast to dorsolateral function (Akert, et al, 1960) is not compensated by early removal. Harlow et al, (1968; 1964) have showed that dorsolateral prefrontal lobectomy produce delayed-response deficits only in monkeys 5 months old or over.

To study the neural mechanisms concerned with regulation of responsivity and lateralization in neural organization, Hansing, Schwartzbaum and Thompson (1968) studied the effects of unilateral and bilateral caudate nucleus lesion on rats in 15-sec. VI and 1-min FI schedules. Ss were trained to press lever in two-lever box for 8% sucrose solution. Daily sessions were given alternatively with each of the two lever. The results were as follows: Unilateral caudate lesions caused a complete cessation of lever pressing with the contralateral paw. Retraining was not successful. Even when attracted to the lever, the S did not reach pre-operative level of performance. With a second-stage caudate lesion of the opposite side, some Ss failed to respond with either forepaw, while some Ss showed a recovery from an odd pattern of depressed response with the forepaw

contrary to the second lesion. The cortical lesion which included the sensorimotor areas although sufficient to induce transient paralysis of contralateral limbs and disturbance in placing reactions, did not impede lever pressing with the contralateral forepaw. One-stage bilateral caudate lesions did not produce any systematic behavioural changes on the VI schedule but showed a significant shift in the temporal distribution of responses during the first half of each FI. Unilateral lesion of the same subcortical area did not produce any demonstrable changes in performance. On the other hand, while unilateral caudate lesion produced a significant decrease in response rate with the contralateral limb, bilateral caudate lesion produced no systematic shift in topography of response, though it did increase the changes in response pattern in an absolute sense. The authors attributed the changes to lesion-induced disturbance in the initiation of behaviour, which is a function of the striato-pallidal system (Hansing, et al, 1968; Denny-Brown, 1962).

Electrical Stimulation Experiments

Turning to the experiments using electrical stimulation, one is impressed by the relative consistency in the results obtained from different laboratories. The work of Buchwald, myers, Lauprecht and Heuser (1961) showed that low frequency stimulation of the caudate nucleus (70V, .01 msec. at 5 sec. intervals) activated

inhibitcry processes manifested both by behavioural changes (eg. from alertness tc drosiness and sleep) and by the occurrence of "caudate-spindles" in the electrogram, which is a train of high voltage, rhythmical oscillation, lasting 3 sec. in duration, with a latency of 150 - 200 msec.). Both the behavioural changes and the electrical recordings could be disinhibited by novel stimulation. 40 cats were used in acute and chronic experiments to study the cortical loci of the caudle-spindle (Buchwald et al, 1961a). With a low intensity single shock, 0.01 msec. duration, at 5 sec interval, of the caudate nucleus, spindle bursts were picked from several cortical and subcortical sites. The most prominent place for spindle activity is the ipsilateral precruciate (anterior sigmoid gyrus) region of the cortex. It can also be obtained from contralateral anterior sigmcid gyrus, bcth posterior gyri, most of the thalamus, globus pallidus, contralateral caudate nucleus, entopeduncularis, and septum. The caudate spindle is elicited at a frequency of 10-12 Hz and can be obtained from resting or sleeping animals and in preparations exhibiting relatively slcw rhythmical electrocorticograms. It is thus antagonistic to functioning of the central arousal system. In relation to other brain areas, the authors showed that electrocoagulation of the thalamic nucleus ventralis anterior ipsilateral to the site of caudate stimulatien suppresses "caudate-spindles" at the ipsilateral cortex but contralateral spindling

remains. With prolonged stimulation, the Ss exhibited decreasing sensitivity to peripheral stimulation and gradually passing into a state of drowsiness and sleep. Simultaneously, the caudate spindle threshold decreased while that of duration increased. However, at any point in the sequence, both the electrical and behavioural changes can be reversed by arousal stimulation of sufficient magnitude. Further, the electrophysiological effects of central arousal can be compensated for by an increase in the parameters of caudate stimulation. "These results suggest that the 'caudate-spindle' is related to neurological processes mediating general behavioural inhibition and acting antagonistically to reticulc-thalamic arousal processes" (1961, I).

Heuser et al,(1961) stimulated the caudate nucleus of cats before and after separation of the thalamus from the caudate. The results showed that the caudate-induced cortical spindles depend primarily on anterior ventral nucleus of the thalamus. The same was true with caudate-induced cortical desynchronization. They suggested the existence of a caudate-loop (thalamus-caudate-anterior ventral nucleus). Depending on its frequency, this feedback loop can function in inhibitory or facilitory fashion. But under the normal circumstance the caudate acts in a inhibitory manner to produce inhibition both electrographically and behaviourally eg. loss of alertness .

Buchwald et al, 1961 (Buchwald, Wyers, Lauprecht and Heuser) implanted chronic electrodes in cortical and subcortical loci to study the behavioural effects of stimulating the caudate nucleus and ventral anterior nucleus of the thalamus in cats. Depending on the stimulating parameters, the behavioural consequences varied. All cats were trained to bar press on FR-1, FR-4, and FR-12, and stimulus threshold was established for each S. Bilateral stimulation of the caudate nucleus was then presented at frequencies ranging from 1 pulse/5 sec. to 5 pulse/sec. At these frequencies there is a stimulus-induced slowing and cessation of response. While the lower frequencies caused a more gradual reduction in response rate before cessation of bar pressing , the higher frequency stimulation could cause abrupt termination of responsing in the operant situation. However, these Ss remained alert and oriented, though they seemed unconcerned with the lever. Upon termination of the stimulation, Ss resumed lever press with a post-stimulation pause without any change in response rate as compared to pre-stimulation baseline rate. High frequency stimulation of the caudate at 300 pulse/sec. on the other hand, caused desynchronization of the caudate-spindle without changes in overt response pattern, and it never increased rate of bar pressing in hungry Ss. Stimulation of an S previously exhibited low-frequency-stimulation induced cessation of response caused the S to resume control rate of lever press.

Effects of the stimulation on the anterior ventral thalamic nuclei produced essentially the same results. The authors thus concluded that the caudate nucleus is concerned with two separate opposing functional systems; a low frequency inhibitory system and a high frequency facilitatory system. Normally the caudate functions as a part of the negative feedback sending modified signals from the diffuse thalamic nucleus to the anterior ventral nucleus of the thalamus, and then to the cortex.

In the 1964 paper, Buchwald, Horvath, Soltysik and Romero-Sierra further showed that low frequency stimulation induced cessation of lever press was not motor dysfunction. Rather, it was the consequence of behavioural inhibition -- in contrast to caudate stimulation of the internal capsule which caused the S to become very awkward or press too lightly to actuate the manipulandum, often showing tremor of the contralateral paw and head, and general unresponsive to external stimulation.

Buser, Rougeul and Perret (1964) trained cats to press lever for food with one foreleg. A repetitive 2 pulse/sec. sequences of flashes or clicks served as the CS. The Ss were implanted cortically with recording electrodes and subcortically with stimulating electrodes. Essentially the same behavioural observation was reported, with bilateral stimulation of the head of the caudate nucleus and of the midline thalamic nuclei. The optimal frequency was 3/sec. for the caudate and 7/sec.

for the thalamus. While stimulation produced cessation of conditioned response, thalamic stimulation had much less effect on the more 'natural' movements such as food-seizing , head turning, chewing and walking. Caudate stimulation showed no effect on these 'natural' movements. The authors also reported a close correlation between electrophysiological and behavioural effects. Threshold for suppression of the conditioned movement and for cortical 'spindling' appeared to be of the same magnitude. Thus, similar to the previously quoted experimenters, the authors considered the caudate nucleus and the midline thalamic nuclei the important centers for response inhibition, of conditioned behaviour. Buchwald, Romero-Sierra, Hull and Wakefield (1967) showed further that stimulation of both the caudate nucleus and of the ventrolateral thalamic nucleus could serve as a discriminative stimulus for either the presence or absence of reinforcement. Such CS quality of central stimulation could oppose the innate inhibitory effects of stimulation upon overt behaviour. Based on these observations,they argued that there is a considerable overlap of neural elements stimulated by the inhibitory stimulus and by the learned stimulus.

In the 1967 paper, Buchwald, Hull and Trachtenberg correlated between unit activity, EEG and effects of stimulation of the head of the caudate nucleus and the thalamus. In "chronic" cats, parallels between beha-

vioural and electrophysiolcgical activity were observed. Stimulatien of these sites induced an inhibition of the unit activity whose duration is closely associated with the onset of EEG spindles, with a concomitant extracellular positive slow wave. The inhibition was abolished when afferent inputs were paired with the inhibitory stimulus.

Stamm and Rosen (1969) studied the relation between electrical stimulation and steady potential shifts in prefrontal cortex in four monkeys during delayed response performance. Bipolar stimulation consisting of 2 sec. trains of 1 msec. pulses at the rate of 50 pulses/sec. was delivered to the principal sulcus contralateral to the responding hand. Correct response for each S was disrupted if the stimulatien train was presented during the first second of delay. The recording showed that the magnitude of negative steady potential shift from prefrontal cortex was related to the rates of correct and incorrect response, the higher magnitude was related to higher correct response rates, suggesting that the shifts reflected the state of excitation of the underlying neural element. They concluded that the cortical segment of pricipal sulcus in contralateral hemisphere was crucially implicated in the DR task only during the brief period before and after the beginning of the delay interval.

Deadwyler and Wyers (1970) studied the effects of interpolated septal and caudate stimulation on DRL performance in albino rats (1970), with a view to determine the point within the delay interval where stimulation of either structure maximally influences DRL performance. The experiment consisted of six daily sessions to each of three interpolated stimulation conditions. The three interresponse time classes were : the early (0 - 4.3 sec.) the middle(4.4 - 8.7 sec.) and the late (11.0 - 15.4 sec.) interresponse time. Daily sessions consisted of four counterbalanced blocks of 18 reinforcements. The stimulating parameters were 60 1/2 ms /sec of 20 - 100 microamps for the septal Ss and 3.0 1/2 ms/sec at 700 microamps for the caudate Ss. While the substantial difference in stimulation intensities cloud the interpretation of the results, the results are in part consistant with other studies (Buchwald et al ,1964 ; Buser et al , 1964). Specifically, results showed that with septal stimulation facilitation of response inhibition in the 'early' and 'late' time classes of the DRL delay interval did not enhance overall DRL performance. Neither did it significantly disrupt normal timing behaviour, indicating that such stimulation did not act as a reversible lesion. With caudate animals, on the other hand, there was a significant reduction in responsiveness during all three interresponse time intervals. Besides, the 'middle' and 'late' interpolation conditions affected a significant increase in responses during interresponse times immedi-

ately following time out of the DRL delay. All three interpolated caudate stimulation facilated the efficiency of DRL performance by significantly increasing the mean number of reinforcements per response. The authors thus concluded that there was a stimulation-induced disruption of normal timing process. Caudate stimulation "not only inhibited responses during its occurrence but also may have disrupted the 'mnemonic' device which the animals normally utilized to process information about the passage of time" (Deadwyler and Wyers, 1970).

Chemical Stimulation Experiments

Direct application of cholinergic compounds into the head of the caudate nucleus produced effects similar to those elicited by electrical stimulation. Hull et al, (1967) compared the effects of direct cholinergic stimulation of the head of the caudate nucleus with those based on electrical stimulation of the same area. They found that parallel to electrical stimulation studies, behavioural consequences of carbachol were a function of the total dose. Two qualitatively different behavioural patterns were observed. Application of small doses (less than 4 mg) of carbachol elicited responses which included inhibition of on-going learned performance although the animal remained alert and responsive to sensory stimuli. Higher doses (ranging from 4 to less than 10 mg) on the other hand, produced behavioural effects which included intense rage, severe autonomic discharge and sharp drop

in cerebral cortical temperature. These effects are different from those produced by high frequency electrical stimulation. The experimenter attributed the effect to high resistence of carbachol to hydrolysis by cholinesterase. Similar effect was also obtained with local application of the chemical tc lateral ventral thalamic nucleus. The same neuronal pcol was considered to have been excited by both electrical stimulation and by the chemical to produce the inhibitory behavioural patterns.

Neill and Grossman (1970) also compared the effects of lesicn and chemical compound scopolamine on caudate functicning. Local application of chemical was done by bilateral implantation of dcuble-walled cannulas into the dorsal and ventral aspects cf the head of caudate nucleus of 40 rats. 5 - 15 mg of crystalline scopolamine methyl nitrate was tamped into the tip of cannula 5 minutes before shuttle-box avoidance training. The results showed that both dorsal and ventral lesions in the head of the caudate nucleus significantly impaired acquisition of avoidance training. But a blockade of cholinergic component cf the dorsal and ventral caudate nucleus produced directly opposite effects on avoidance training.

Administration of scopolamine tc the ventral aspects reliably facilitated, while to the dorsal aspect reliably inhibited shuttle-box learning. The experimenters suggested that both aspects of the head of the caudate nucleus are inhibitory in action and cholinergic, but

they inhibit different behavioural functions. "While dorsal cholinergic mechanisms appear involved in the inhibitory aspects of activity in a noval environment, ventral chclinergic mechanisms are involved in the inhibitory ccmponents of shuttle-box avoidance learning". The absence of facilitatory effects of the ventral lesion was probably due to a mcre predominent effect of other systems in this same general area. Indeed, Neill and Grossman's study corresponds well with Mclennan and York's wcrk on cholinergic mechanisms in the caudate nucleus and its response to electrical stimulation of ventral anterior nucleus of thalamus in cats. In 1966 paper, Mclennan and York described the results of central application of the caudate nucleus by iontophoresis from five-barrelled electrodes. They found that neurons responding by excitation fcllowing ACh and VA (ventral anterior thalamic nucleus) stimulation and responding by depression fcllowing ACh and VA stimulation have been observed. The two types of neurons are located in different regicns of the nucleus, with the excitatory neurons forming a lamina surrounding the depressed neurons. At greater depths near the ventralateral surface and near the medial edge of the nucleus other groups of ACh excitable cells are located. Together with their earlier cbservations (Mclennan, 1964) that the release of ACh frcm the caudate can be enhanced by VA stimulation, it is suggested that the 1966 results indicate a final cholinergic link in the pathway from VA to caudate

nucleus. Shute and Lewis (1963); Ordy et al,(1969) and Macintosh (1941) lend support to the hypothesis that the caudate nucleus is an important structure in the cholinergic system.

Carlton (1963) stated that "...there are inferential grounds for supposing that a cholinergic system selectively antagonizes the effect of activation on certain behaviour and that the basis of this selectivity is the extent to which that behaviour is rewarded". Pavlov (1927) analyzed delayed responsing into two phases of initial inactivation and activation. According to his analysis, the phase of activation is closely related temporally to the onset of reinforcement (Pavlov, 1927), while the delay is established via gradual lengthening of time interval between the onset of CS and of reward. The logic consistency between behavioural and pharmacological studies can be readily seen. And we can say that, together with studies using stimulation and lesion techniques, the frontal lobe system is cholinergic and inhibitory in function under normal circumstances, and it is crucially involved in delayed response and temporal delay.

Literature supporting hypothesis II : delayed respons and temporal discrimination as a function of mediating postural mechanisms

While literature supporting hypothesis 1 (that

delayed response and temporal discrimination are a function of the frontal-caudate -thalamic subsystem) is abundant, there are studies which support the second hypothesis that delayed response and temporal discrimination are a function of mediating postural mechanisms. These studies will be reviewed below.

Wilson and Keller (1953) studied the behavioural pattern of male albino rats on the spaced responses (i.e., differential-reinforcement-of-low-rate schedule; DRL). After preliminary training in Skinner box, the Ss were reinforced with food pellets for lever presses separated from the previous presses by progressively longer delay intervals, viz., 10, 15, 20, 25 and 30 sec. They observed that as the delay intervals increases the rate of bar pressing decreases in a fashion linear to the delay intervals used. The median time between successive responses increases slowly , but the number of reinforcements decreases quite rapidly. More important for our purpose, he noticed that each S developed clearly defined collateral behaviour during the intervals between successive responses. "This change in the rate of the conditioned response results from the partial extinction of bar pressing and the strengthening of behaviour that is incompatible with it". As these responses which gained precedence over lever press due to nonreward are not specified in the reinforcing contingency each S developed its own pattern. With increase in delay intervals, more

links were conditioned to the chain of collateral behaviour. The strength of such collateral chains was thought to be maintained through conditioned reinforcers provided by the stimulation attending the response chain.

Laties, Weiss, Clark and Reynolds (1965) examined in detail the phenomenon of mediating collateral behaviour in the DRL schedule. An adult albino rat was trained on DRL 22-sec EXT 2-min FR 30-EXT-2-min. After 35 hours of training the experimenters observed that there was a very regular pattern of behaviour during the pauses between the DRL component and only at that time. The rat appeared to be biting its tail and moving its mouth over the surface from one end to the other while holding the tail in the front paw. Recording on this behaviour showed that (1) the rat nibbled on its tail longer during pauses preceding reinforced responses. "...the amount of mouth-tail contact could have served as a discriminative stimulus for lever pressing". Application of cyclohexiimide to rat tail supressed mouth-tail contact and also the amount of reinforcement. Intraperitoneal injection of amphetamine sulfate and removal of lever also induced variation in the mediating behaviour chain and subsequent reduction in reinforcement rates. These results supported the hypothesis that the collateral behaviour chain is important in the temporal spacing of responses. Essentially the same type of behavioural patterns were observed by Hodos et al, (1962), using monkeys as

subjects.

Miles (1964) reported his study in squirrel monkeys with frontal lesions. He emphasized the importance of spacial factors in delayed response performance. Lesion induced hyperactivity would then reduce delayed-response proficiency (French, 1959, and Miles, 1964). He also mentioned the importance of bodily orientation and a learned pattern of behaviour in bridging the delay interval. Thompson (1959) studied the effects of frontal lesion on avoidance learning in cats. He postulated that frontal and striatal lesions interfered with proprioceptive and tactile stimulation essential for the integration of response chains. "The severity of postoperative deficit consequently would depend upon the extent to which the behaviour is under the control of discriminative and reinforcing stimuli produced by the posture and movements of the organism" (Miles, 1964).

Based on experimental reports by Dabrowska who studied the effects of prefrontal lesions in rats, Konorski postulated that postural habits are a major factor in delayed response performance (Konorski, 1967).

Dabrowska (1959, 1963) studied prefrontal lesion in rats under food reinforcement in a four-unit-quadruple-choice apparatus. He found that the lesion disrupted the S's capacity to integrate performance into a unitary act and they had to learn the proper way in each unit

separately. Normal rats showed considerable saving in learning from task to task. But the performance of a frontal rat was much more superior if only one aspect of the last unit was changed. Using a reverse T maze to study the return response in normal, normal blinded, frontal blinded rats, as well as rats trained with one black and one white arm in the maze , he found that only prefrontal lesions impairment in return response. Consequently Konorski argued that the prefrontal region modulated performance that was presumably controlled by kinesthetic cues.

Following Konorski's hypothesis, Stamm (1970) also attributed the normal performance on delayed alternation the function of kinesthetic inputs. He trained monkeys on DA and visual discrimination tasks either in WGTA, restraining chair or locomotor maze. Half of each group received dorsolateral prefrontal cortical lesion. The results were (1) frontal lesioned monkeys performed much poorer in maze learning task than in the restraining chairtask ; (2) the frontal monkeys' errors in responding were a consequence of strong positional habits; (3) the frontal ss were most handicapped in acquiring any visual discrimination task and (4) the frontal Ss which reached criterion also developed postural habits. performance dropped to chance levels if positional habits were interrupted. Thus he concluded that the rate of delayed alternation is a function of the degree of motor involve-

ment in the task. The severity of prefrontal impairment is a function of the degree of motor involvement in the response processes.

If the collateral mediating behaviour and kinesthetic inputs play a critical role in temporal discrimination and delayed responses, then disruption of neural substrate essential to patterned response events would also cause disruption, temporary or permanent, of timing behaviour and/or delayed response. This is one of the major interests of the present paper. For this purpose the cerebellar pathways and nuclei which are implicated in motor/postural events were studied.

Anatomical Considerations

Anatomically, the cerebellum can be divided into three major sections, following Jansen and Brodal's concept of longitudinal corticonuclear organization of cerebellum (1940,1942). This concept stressed the importance of localization in the efferent corticonuclear projections rather than distribution of afferent fibers in the cerebellar cortex in the interpretations of results obtained by stimulation and ablation. Using cats as subjects, Jansen and Brodal showed that there is a medial zone (vermal cortex and fastigial nucleus) regulating the posture, tone, locomotion and equilibrium of the entire body. The intermediate zone (paravermal cortex and interpositus nucleus) regulated the spacially

organized movement and skilled movement, tones, and posture associated with movements of the ipsilateral limbs. Each lateral zone (hemispheric cortex and dentate nucleus) is involved in the same skilled and spacially organized movement of the ipsilateral limbs without any apparent regulation of their posture and tone. Goodman, Hallett and Welch(1963) used 37 female adult albino rats to study the organization of cerebellum. Their study confirmed Jansen and Brodal's work, with the exception that the intermediate and lateral zones in the rat are not as clearly defined as those in the cats. Such subdivision of the cerebellum into longitudinal zones is also associated with a localization of the corticonuclear projections in a rostro-caudal direction.

Roberts (1967) traced the outgoing pathways from the dentate nucleus. The greater part of the outflow was reported to run from the cerebellar cortex to the deep nuclei of the cerebellum. The dentate nucleus receives its afferent input from the lateral zone and from parts of the extrapyramidal system, and from the red nucleus, the pathway in each case relaying in the inferior olive. The outflow of the dentate nucleus runs in the superior cerebellar peduncle, back to the red nucleus of the opposite side and to the basal ganglia. Thus it forms a relay station for at least two loops in the extrapyramidal system. However, it does not necessarily mean that these structures operate in feedback and stabilizing

fashion. Roberts stated that the extrapyramidal system, including the lateral hemisphere of the cerebellum appeared to be concerned with the detailed coordination of all parts of the body to provide a steady base for precise voluntary movements.

Snider (1950) stated that the newer concept of the cerebellum included cerebellar action in dampening and in potentiating associated sensory and motor centers. It is the "greater modulator" of neurologic function. He reported that the tactile, auditory and visual areas of the cerebellum have a dual projection, one from the end organ and another from similar areas within the cerebrum. These cerebellar areas project back to the projection areas on the cerebrum. Similar arrangement also exists between cerebellum and thalamus and the basal ganglia. Thus it is a self-regulating feed-back circuit, adding or subtracting total effect of afferent volley to functional areas. However ,Evarts and Thach(1969) on the other hand, favor more the hypothesis that the lateral zone of the cerebellum does not form reciprocal connection with its cerebral afferents. Instead, it sends output to the sensorimotor cortex and possibly initiate activity there. On the whole the output pathways of the lateral cerebellar zone are still not well understood.

In his Croonian lectures, Holmes (1922) presented the hypothesis that cerebellum plays two distinct though closely allied roles in movement. Firstly, it is a part,

and probably a controlling part of the central mechanism concerning maintaining, regulating, and distributing postural tones in muscles. Secondly, during movement it regulates and coordinates the modification of tones that accompany the relaxation and contraction of all muscle groups directly or indirectly concerned in the movement. In the first case, the cerebellum has a continuous and unvarying function while in the second case, its activity is discontinuous and variable (Holmes, 1922).

Functional Considerations

In the 1939 paper, Holmes described in detail the effects of destructive lesion of the cerebellum, in man and higher primates. The most obvious effect was a lesion-induced disorder in voluntary movements, usually called ataxia. The fundamental disturbances in voluntary movement are (1) a delay in starting a movement, and (2) irregularity in its acceleration. The latter disturbance was attributed to irregular innervation of the prime movers. In compound movements there may be a lack of synchronicity in the separate components of the movement, due to delay in the initiation of one component relative to the other, and excessive range of one element of the movement. Defective postural fixation was another factor. The predominance of symptoms of cerebellar defects involves the components of voluntary movement. This phenomenon indicates that "it influences directly cerebral mechanisms which excite and integrate such movements

rather than the reflex apparatus of the brain stem and spinal cord; the latter, when isolated from the fore-brain, are in fact unaffected by ablation of the cerebellum"(Holmes,1939). It is important to mention that cerebellar symptoms may gradually decrease in intensity or disappear over time.

Thach's(1968; 1969; 1970a; 1970b; 1975) recent work relied heavily on the reports of Holmes as well as on his theoretical framework. Thach(1968) studied the discharge of dentate and interposed neurons in monkeys during rapidly alternating movements of primate arms. He found that neurons in both nuclei changed discharge frequencies over a wide range in relation to movement. In an attempt to study the temporal relation between Ss movement and dentate-interposed discharge pattern Thach (1969) trained monkeys to grasp a handle and hold it against one of two barriers until the onset of the discriminative stimulus. Reinforcement was delivered if s moved the handle to the opposite barrier within 300 msec. of the onset of the discriminative stimulus. Discharge patterns of these neuronal cells were studied during the maintained posture and for the first index of movement. The results showed that many cells showed modified discharge between CS and movement, and changes were time-locked to movement. For most cells, discharge changed before the EMG response preceding movement. "Thus, cerebellar output may help initiate as well as 'regulate' movement".

The same type of results were obtained by Thach(1970,a) when monkeys were trained to perform two prompt movements and to maintain two different postures. The behavioural sequence was (1) to hold the lever against a stop, (2) when a light came on, to move quickly to the opposite stop, (3) to hold there long enough for a light to be on, and (4) to move quickly to the first stop. The period of holding varied to eliminate anticipatory responses. Ninty-one nuclear cells were studied. Of these 5 dentate cells and 41 interposed cells, again there is a change in nuclear discharge time-locked to movement rather than CS onset. While patterns of change in discharge is different for flexion and extension they are also time-locked to movement, or preceding it. However, the peak of time-of-change distribution is earlier for the dentate neurons than for the interposed neurons. The discharge of Purkinje cells in the intermediate zone of the anterior lobe of the cerebellum was also recorded (Thach,1970,b). These Purkinje cells also exhibited the same type of properties as the interposed nuclear cells to which they project. Evarts and Thach(1969), Herrick(1924) and Holmes (1922,1939) all lend support to the assumption that the interposed and lateral nucleus of the cerebellum are involved in initiation as well as maintenance of movement.

Gambarian et al (1969) studied the effects of sectioning the medial lemniscus in dogs trained to

perform certain tasks. They showed that a bilateral transaction of this pathway dces not end ability to perform voluntary movement, nor does it interfere with the transmission of peripheral stimulation to the sensorimctcr cortex. Adrain (1943) showed that the body surface is prcjected tc the cerebellum in the same way as the topcgraphical arrangement in the cerebral cortex. Russian study by Bekaia and Moniava(1963) have determined electrophysiclogically the pathway between the "sensorimotor area" of the cerebellum and of the cerebrum. It is shown that these pathways originate in the paramedial and anterior lobules of cerebellum and reach the cerebral cortex via the ipsilateral dentate nucleus, the anterior pedencle of the cerebellum, the mesencephalon, the thalamus and the red nucleus. Thus,instead of considering the pyramidal tracts the scle servant of voluntary movement (Gambarian,1966), the cerebellum is also an area of termination of the afferent pathway of the motor analyzer. Granit(1955) showed that motor function is greatly served by extrapyramidal pathway via which the cerebral structures exert control over voluntary movements by selectively ccntrolling the gamma efferent system.

The brain functions in an integrative manner. Destruction of any brain area may influence other brain centers and neural circuits afferent to ,or efferent from the area. The delayed response and temporal discrimination are very complicated tasks consisting of specific

tasks organized in time. Not surprisingly, the experimental results are variable, often reflecting the different methodologies used in studying response timing. From the preceding review, however, one may draw two conclusions. First, selective lesions of the frontal-caudate-thalamic areas will produce impairment in delayed response, spatial or temporal, with little damage to the motor apparatus. On the other hand, selective lesions of motor efferent systems will produce clinic symptoms (eg. ataxia), but the S will still be capable of performing the tasks, though with difficulty.

CHAPTER II

THE INVESTIGATION

The present study proposed to examine the relative role of the frontal-lobe system and the cerebellar efferent system in the temporal and delay conditioning in rats . For this purpose the delayed conditioned approach procedure (DCA) will be used, since it separates the effects of treatment which interferes with the S's ability to perform a task in general from those on accuracy of timing (Halasz,1968, Halasz & Cheng,1967, Cheng & Halasz,1968). It provides us with a tool which can separate the general effects due to hyperactivity or disturbance in response apparatus from the more specific effects such as impairment of response inhibition or timing of the response (a more detailed account of the theoretical framework is presented in Appendex I).

The delayed conditioned approach paradigm (DCA) is a behavioural paradigm which includes 'timing' of response as an essential component. Based on Halasz' definition (1969; 1970; 1972), the DCA has both 'inhibition of delay ' and the operant differential-reinforcement-of -low-rate components. Essentially, it is a discrete-trial, discriminated interval response with within and between trial DRL, a servomechanism- like paradigm aimed at keeping response latencies within a specified range, via reciprocal processes of extinction and reconditioning (Halasz,1968; Halasz & Cheng,1969). A schematic diagram

of the DCA is presented in figure 2. The different components of the DCA paradigm are described and explained in the section on 'method' under the heading of 'experimental design'.

The DCA has several unique features which render it a useful tool for behavioural assay of CNS drugs (Halasz & Marrazzi,1964; Halasz,1966;Cheng & Halasz,1967); for stability of an organism (Halasz, 1966;1967a;1967b;1968;1969) and for effects of radiation (Cheng & Halasz,1968;Wiseman & Halasz,1968; Halasz et al,1970). (1) Via trial-abort feature the DCA differentiates effects due to hyperactivity from those due to more systematic effects of various experimental variables. As shown by Halasz' work on drugs, pharmacological agents which produced increased level of general activity tend to produce increased intertrial lever presses and increased variation in response latencies, without significant changes in the mean response latency. Systematic effects on timing on the other hand,tend to show a drug-induced temporal shift toward shorter (LSD study) or longer (adult radiation) latencies,without significant changes in response variation. Neither were there significant changes in intertrial interval lever presses. (2) The trial-abort period can differentiate the effects of hyperreactivity to CS signal from the effects of hyperactivity. While the former will cause increased lever presses with short latency of response,

Figure 2. A schematic diagram of the delayed conditioned approach diagram (DCA), (1d). The five training stages are presented in a,b,c,d,e consecutively. S is the water availability period.

1a. CS -SD

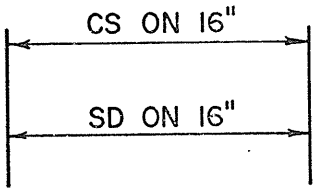
1b. CS -SDand trial abcrt period

1c. institution of 5" delay and 1" penalty

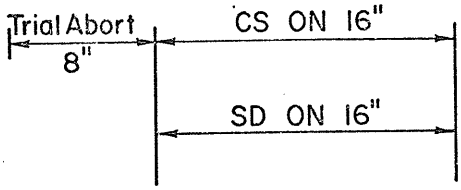
1d. step increment of delay to 9" and of penalty to 5",the DCA paradigm.

1e. Another step increment of the delay to 13" and of penalty

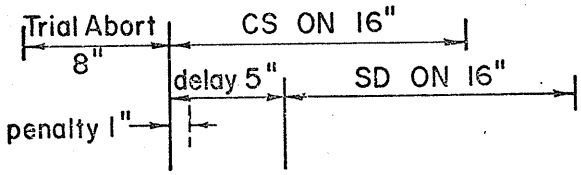
to 9",activation of the integral/optimal control.


CS ON 16"
SD ON 16"

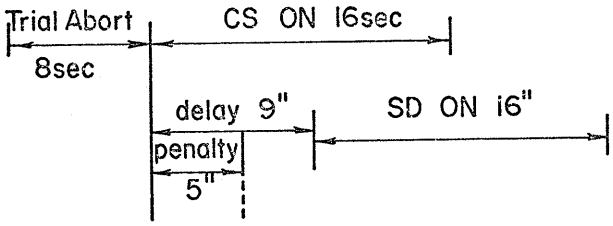
1a


Trial Abort
8"
CS ON 16"
SD ON 16"

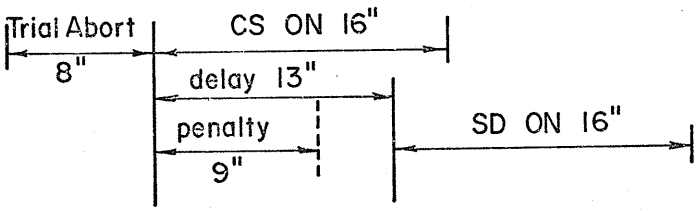
1b


Trial Abort
8"
CS ON 16"
delay 5"
SD ON 16"
penalty 1"

1c


Trial Abort
8sec
CS ON 16sec
delay 9"
SD ON 16"
penalty
5"

1d


Trial Abort
8"
CS ON 16"
delay 13"
penalty
9"
SD ON 16"

1e

the latter will cause a increase in trial-abort counts not only during the between -trial DRL and during a CS trial. (3) Penalty count can reflect the effect of disruption in timimg. Ss who have lesions that affect response timing may have shortened latencies and therefore an increase in the penalty count. If it be so,it can be reflected in shcrtened response latencies in a relatively systematic fashicn. (4) Impairment on DCA performance due tc motoric defects will manifest itself through lengthened response latencies with decreased reinforcement counts,because injury of the motor defects will manifest through lengthened responses latency with decreased reinforcement ccunts,because injury of the motor apparatus affect smocth execution of an act. (5) Changes in the stability cf DCA behaviour will result in changes in the number of switchings of the correctional programme. Lcnger than required response latencies will result in little correction while very short response latencies will cause much increased penalty counts due to the correctional input. (6) The DCA is a servomechanism-like paradigm. Its response latency is "driven" to values demanded by the reinforcement schedule. As such it is a self-regulating behavioural system,responsive to the fluctuating demand imposed by the schedules of reinfcrcement. By probing the behavioural system via changes in ccntrcl policies, the resultant history of that behaviour provides insight into the governing equations for that behaviour (Halasz & Cheng,1969; Halasz,

1970, Halasz,1972). (&) Due to the servomechanistic characteristics of the DCA behaviour, it lends itself as a useful paradigm via which one can apply sensitive probe methodology for exploring behavioural abnormality in brain-lesioned animals. In this context the interrelationships between the brain "centers" and the behaviour can be studied in an active equilibrium system approach, rather than as static correlational entities.

METHOD

Subjects:

Adult male albino rats of the Holtzman strain were used in the experiment. Training sessions began when the animals reached 60 days of age. In total, 64 rats were used in the experiment of which 50 survived throughout the whole experiment on the DCA paradigm. Assignment of the subjects to the various treatment groups were randomized via the aid of a random table.

Deprivation Schedule:

Ss were placed on 3 days of 23-hour water deprivation schedule before the commencement of the experimental procedures. Thereafter, all subjects were maintained on 23 hour water deprivation throughout the experiment. A 20 minute watering period was given for each S, one hour after the daily session. All Ss were put on food and water ad lib for 3 days prior to brain surgery. Wet mesh food was provided for the first post-operative week for

all Ss (Whittier& Orr,1962), followed by one week of food and water ad lib, before reinstallment of the 23-hour water deprivation schedule.

All subjects were housed in individual wire cage measuring 6"x6"x8" in dimension .The ambient temperature was maintained at 70 F by air conditioning. A 12-hour light and 12 hour darkness cycle waa used. All experiments were conducted during the light phase of the cycle.

Lesions were performed when subjects were between 120 to 150 days of age.

Apparatus

(i) Operant chamber: All training and testing sessions were conducted in a sound- attenuated two-lever Skinner Box with the right lever programmed to deliver water reinforcement. A lever press of 24 gm force or more produced water reinforcement. The CS,a 1KHz, 80db. pure tone was generated by a Eico Model 377 sine-square wave Audio Generator.IVE (Lehigh Valley Electronics) 1530 print-out counter registered the response latency of first response to each CS. BRS precision probability unit (Model No.PP-1)ensured randomization of DRL duration. Experimental contingencies were programmed via BRS logic units. Precise adjustments of logic were monitored by a Heathkit Electronics oscilloscope. The integral/ optimal programme was programmed by a Heath Amplifier System Model EUW-19-14.The activity of the S in the experimental situation was monitored by a model MT 100

Electrocraft Movement Transducer, whose field sensitivity ranges from 1/4" to 8" above and around the sensor. Counter speed is 2/sec. Activity level of Ss was also measured by a running wheel for rats.

(ii) DCA: As shown in figure 3a the required minimum delay of response is represented by the adjusted duration of a 1-shot pulse, which was triggered by the onset of CS. The pulse was led to one input of a summing amplifier. The duration of the inverted output of a flip-flop;which was set by CS onset and reset by bar press,coded the actual response latency on every CS trial. The output of this flip-flop constituted the other input of the summator. The output of the summing operational amplifier (p) was then fed to the input of the integrator operational amplifier. The output of the integrator was the cumulated error (p) and was plugged into an adjustable-level Schmitt-trigger. If the p exceeded the set value, the Schmitt- trigger set a flip-flop to close the contact point of the schedule correction relay which added the correctional capacitor Cc to the CB controlling the baseline DCA schedule. After integration over 8 consecutive CS trials, a shorting relay then discharged the feedback capacitor Ci. The integrated p was reset to zero.

In the event that the correction flip-flop was set by a prior integration and had not exceeded tolerance level, an and-gate assured the reset of flip-flop to restore to baseline schedule. To guard against extinc-

tion , a failure to respond during the CS-SD period led to immediate reset of schedule.No reset took place if p was unacceptable once the correctional flip-flop was set.

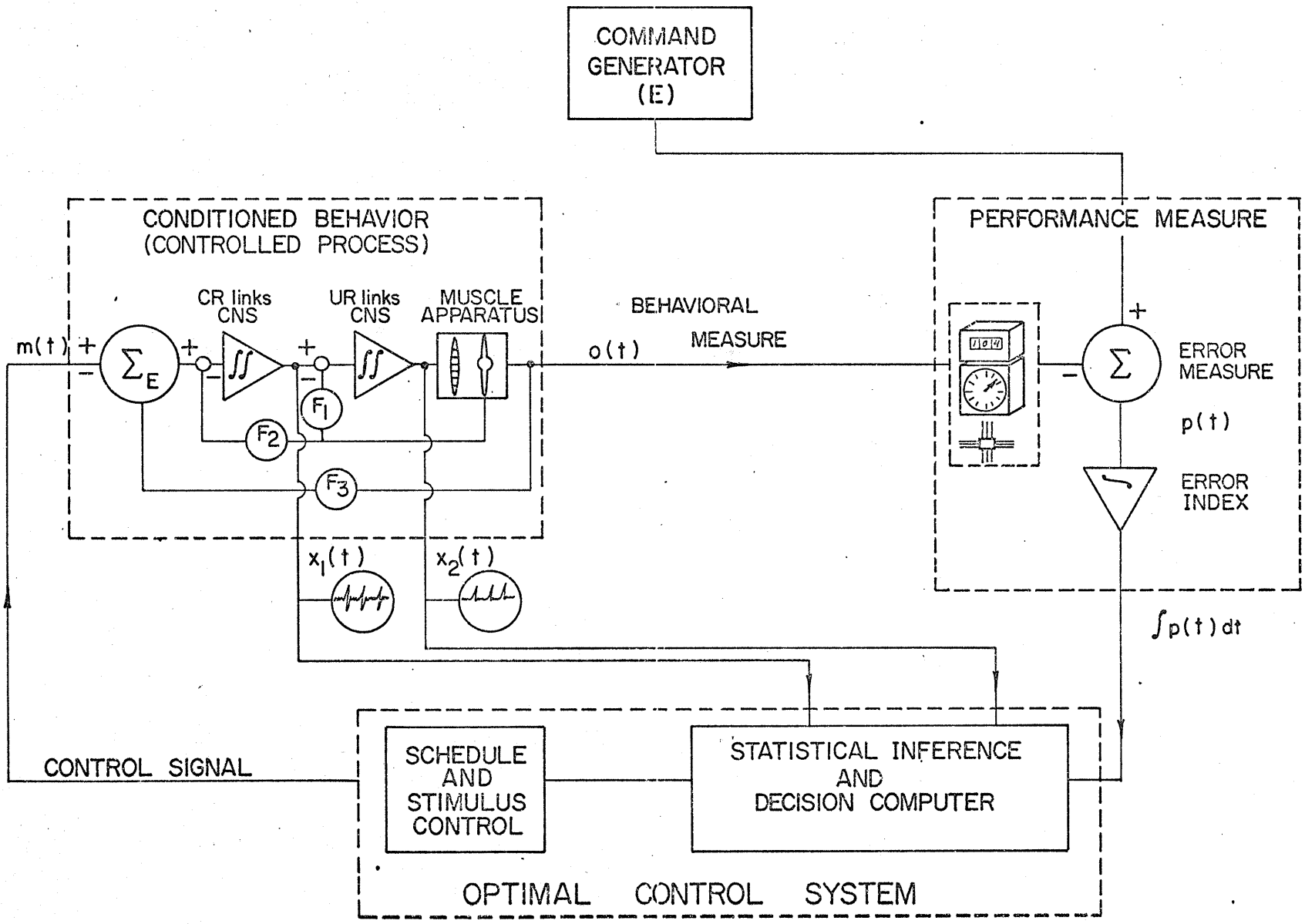
(iii) The Integral/optimal control

The integral/optimal control (which will be subsequently referred to as a correctional input) was programmed via a Heathkit Operational Amplifier System and was programmed and controlled by BRS units. Where computation and mathematical calculations were required for the optimal/integral control, we constructed an analog computing system. Heathkit chopper stabilized operational amplifiers were used for integration and subtraction of the error term (p). Motorola 4-quadrant Multipliers were essential in controlling the variance of response measure distribution. By way of Schmitt triggers and Rovetti windows, the analog computor interfaced with the regular BRS programming equipment (fixed limb of feedback diagram) The schematic flow chart for the O/I program is presented in figure 3 (Halasz & Cheng,1969).

(iv) Surgery Equipment

A C.H. Stoeling Co. stereotaxic, Krieg- Model No 51220 for the rat was used. A C.H. Stoeling Co. Lesion Producing Device (Cat. No. 58040) for the rat was used for the passage of lesioning current. The lesion electrode was made from tungsten wire which was insulated at all points except for the tip.

Figure 3. A schematic representation of the integral/ optimal control (Halasz & Cheng, 1969).


COMMAND GENERATOR (E)
CONDITIONED BEHAVIOR (CONTROLLED PROCESS)
CR links CNS
UR links CNS
MUSCLE APPARATUS
m(t)
Σ_E
F1
F2
F3
$x_1(t)$
$x_2(t)$
BEHAVIORAL MEASURE
o(t)
PERFORMANCE MEASURE
Σ
ERROR MEASURE
p(t)
ERROR INDEX
$\int p(t)\,dt$
CONTROL SIGNAL
SCHEDULE AND STIMULUS CONTROL
STATISTICAL INFERENCE AND DECISION COMPUTER
OPTIMAL CONTROL SYSTEM

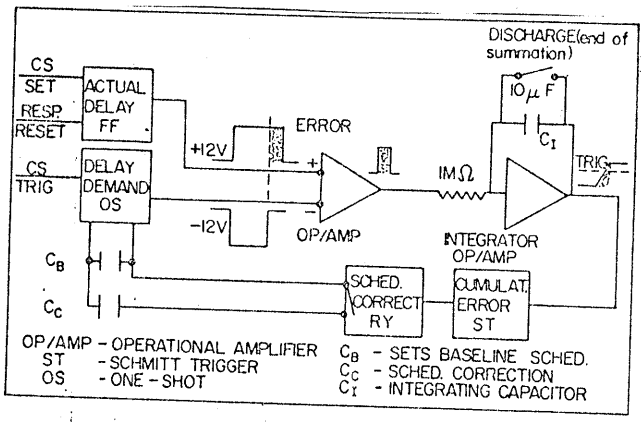


Fig 3a. A simplified flow-diagram of integral/optimal program (Halasz & Cheng, 1969)

Experimental design: the Delayed Conditioned Approach (DCA)

"Structurally" the DCA can be described as follows: It is a conditioned approach response in which a subject is required to press a lever for water reward, but the reward is not available until a certain time interval has lapsed since the CS onset. This time interval between the onset of CS and of Reinforcement is the delay. A penalty period (DRL component), the punishing component of the paradigm starts with the onset of CS but is usually shorter in duration than the delay component. Any lever presses during the penalty period will cause immediate termination of the CS and the scheduled reinforcement period for that trial. Immediately preceding the CS onset, there is a trial-abort period. Indescriminate lever presses during the intertrial interval and within the trial-abort period will result in postponement of the next scheduled trial. This is the other DRL component of the paradigm. Typically, at the base line schedule the CS is 16 sec.; penalty 5 sec; delay 9 sec.;reinforcement 16 sec; and trial abort 16 sec. Figure 2 is a schematic diagram of the DCA.

In addition, a correctional schedule was used for DCA "shaping" and as a "probe"to explore the behavioural abnormality in lesioned rats. This was done by means of correctional changes of reinforcement schedule (Halasz &

Cheng, 1969). Based on bang bang control theory the integral optimal program strategically increased or decreased the demanded value of the delay. In detail, the optimal/integral control was programmed in the following steps (1) The error term (p=required latency-actual latency) was formulated (2) Integration of p over 8 consecutive trials to obtain a (p) This p constituted a sample of the level of DCA performance (3) The final error integral p was assessed and its acceptability was evaluated. (4) if p was below the lower tolerance level, a correctional schedule, which was a "lumped" unit increment of 4" in delay and penalty was in effect. When the p exceeded the upper tolerance the correctional increment was removed. (5) reset of P for next integration.

The stability of the DCA system was evaluated in terms of the following behavioural indices:

(a) the mean response latency of response to the DCA trial was used as an indicator of the absolute level of S's performance on the DCA.

(b) The relative error

A ratio, P=(required latency - actual latency)/ change of required latency(ie. a unit increment of 4 seconds), was used as an indicator of the state of the DCA system. As a dimensionless quality, the P provided the ratio of behavicural adjustment to the changes in demand levels of the reinforcement schedule. Each

experimental session comprised of 40 actual daily trials. Since there were usually much stochastic fluctuations of a subject for every daily session,it was our practice to use the P of each 40 daily trials as one data point against a time point.

(c)the $\overline{P}$ plot--the time response

The relative error of the DCA was plotted against time (days). P=1.000 corresponds to behaviour perfectly adjusted to baseline contingency (delay=9,penalty=5) P=0.000 corresponds to behaviour perfectly adjusted to the correctional input (delay=13,penalty=9).Basically the P plot transforms the P values into the standard form for the time-domain analysis of the servomechanism-like control systems. It renders the behavioural data available for quantitative analysis.

(d) $\overline{P}(0)-\overline{P}(T)$ Plot

Using the last day before the installation of the optimal/integral control as the baseline,this difference plot gives the day-to-day changes in the $\overline{P}$ of the DCA performance.

(e) the phase-plane

The optimal control of operant behaviour is best understood by representing it in a phase plane (Halasz, 1972). The stage-space of the DCA behaviour system has as its coordinates the error index $\overline{P}$ and its time derivative DP/DT. The trajectory so derived represents

both the level of controlled behaviour and its tendency to increase or decrease that level.

PROCEDURE

A. Delayed Conditioned Approach (DCA) Procedure:

(1) DCA training sessions:

Training on the DCA was done in the following stages for a total of 6 to 8 weeks (figure 2):

(a) Rats with 23-hour water deprivation were trained to press a lever for water reinforcement. Each dipper of water contained approximately 2 to 3 drops. All Ss were put on CRF for 3 days. Some Ss were trained by the method of successive approximation.

(b) SD-S^{Δ} training: A CS of 16 sec. duration, 84 db.,10 KHz pure tone was introduced .Water reinforcement was contingent on lever presses during CS duration, Ss were put on this schedule for 5 days. Each daily session had 40 trials. A 8 sec trial abort period was instituted on the 3rd day to reduce intertrial lever presses. Any indescriminate lever presses during this period postponed the next secheduled trial.

(c) A 5 sec delay and 1 sec penalty period were instituted. 5 daily sessions were run under this procedure.

(d) A lumped increment of delay and penalty period at a 4 sec step fashion was implemented. Ss were trained on this schedule for approximately 4 to 6 weeks.

(e) Superimposition of the integral/optimal input was in effect. Ss were put on the paradigm for 10 days. Each daily session consisted of 40 daily trials. Upon

termination of the training sessions,Ss werer randomly assigned to the treatment groups.

(2) Ss were put on food and water ad lib for 3 days. Then brain surgery was performed. All subjects were given two weeks rest period post-operatively.

(3) Post-operative Testing Sessions: A total of 16 daily sessions were run:

1. Retention sessions:All Ss were put back on 23 hour water deprivation schedule and their performance on the DCA were studied for ten consecutive testing days. The integral/optimal programme was superimposed on the base-line DCA contingencies during this phase of testing. Motoric activity in the operant chamber, and the activity level in the rotational running wheel were monitored.

2. Retention sessions with on intergal/optimal control: Upon termination of the 10 testing days on the DCA with the integral/ optimal control,the Ss were then returned to the baseline contingencies of the DCA for 5 testing sessions.

3. Extinction sessions:All Ss were put on 2 days of extinction on the DCA paradigm.

B. Procedure for Lesioning:

All lesions and sham lesions were electrolytic and bilateral in nature. Operations were performed with subjects under ether anesthesia.

During surgery, a scalp was used to make a midline incision. A high speed dental drill was used to make a small opening through the skull at points as guided by the stereotaxic coordinates. The lesioning electrode was lowered in the brain sites. An anodal current of 3.5ma. was passed between the lesion electrode and the rectal ground for 30 or 45 seconds to produce the desired lesion. The muscle flaps were then closed with autoclips,.2cc terramycin I.P. was injected immediately following the surgery to control for infection.

(i) 4 lesioned groups:

(1) Head of Caudate Nucleus Lesioned Group (H CD)

Six rats were assigned to this group. The coordinates for this group was based on de Groot's Atalas for the rat (1959). The lesion electrode was lowered into the brain at 1.7 mm anterior to the bregma,2.7 mm lateral to the midline and 5.5 mm from the surface of the skull.

(2) the Dentato-interposed nuclei of the Cerebellum

Seven Ss were used in this group. Lesions were made at 11 mm posterior to the bregma,2.2 mm lateral to the midline and 7 mm from the surface of the skull. These coordinates were based on Bures,Petran & Zachar's atlas for the rat.

(3) the Frontal lesioned group (Ft)

Seven rats were used. The lesion coordinates were 5.2 mm anterior to the bregma,1,5 mm to the midline and 2 mm from the surface of the skull.

(4) the posterior caudate nucleus lesioned group (P

CD C)

Six Ss were assigned to this group. The lesion coordinates were.2 mm posterior to the bregma, 3.5 mm lateral to the midline and 3.5 mm from the surface of the skull.

(ii) the Normal Control Group (N); Ten subjects were used, of which seven completed the whole experiment.

(iii) four operated control groups:

(1) the Head of the Caudate Nucleus lesioned control group (H CD C)

Six Ss were assigned tc the group. Same coordinates as those of the H CD were used,except no electrolytic lesion current was passed through the brain loci.

(2) The Frontal Operated Ccntrol Group(Ft C)

Five Ss were aSsigned to this group.

(3) the Dentato-interposed nuclei of the Cerebellum Operated Ccntrcl Group (D-I C)

Five Ss were aSsigned to this group.

(4) the posterior caudate necleus operated control group. Three subjects were assigned to this group.

C. Histology Procedure:

The brain spcimen were cut into blocks , embedded in paraffin and sectioned on a microtone. Sections were of 11 or 6 in thickness. The sections showing the location of the lesicn sites and the passage of the electrode tracts were stained with cresyl violet. (All histologic-

al procedures were done in the department of physiology, the Medical Faculty, the University of Manitoba.)

CHAPTER III

RESULTS: BEHAVIOURAL DATA

The behavioural data are described in terms of graphic representations However, it should be stated that statistical analyses were done on all the groups and on all data which are presented in the text. All data were evaluated at .05 level of significance.

GROUP I the group with bilateral electrlytic lesion in the head of the caudate nucleus (H CD):

A. general observation:

Upon awakening from ether after the surgery, Ss bearing bilateral electrolytic lesion in the H CD showed hyperreactivity to the external stimulation. All Ss showed strong startle reaction to a light puff of air.On the whole they were more active in the home cage. Ss showed no impairment in their motoric functions. The hyperreactivity subsided by the end of the first post-operative week.

B. rotatioal activity running wheel:

Figure 4 shows the activity level of the Ss (n=6) before and after the surgery. Each point on the graph represents the mean of the total number of rotations tested on that day. Data from 3 pre-operative days and 5 post-operative days were included. As shown on the graph,there was no change in the mean of the total number of rotation over the 30 minute testing sessions between

the last pre-operative day and the first post-operative day ($p > .05$). Over time, however, there was a tendency towards decreasing of activity in the running wheel. Similar trend was found for the H CD and the H CD C groups (figure 4). The normal Ss maintained a stable level of activity through out the 8 testing sessions.

C. perfcrmance on the DCA

(1) Figure 5 shows the the means of the total number of a) total lever presses, b) reinfirced lever presses, c) penalty count and d) trial abcrt count of all 29 daily sessions. For clarity the 29 daily sessionss were grouped into six time blocks. Of which, the first and the last blocks were on the baseline DCA contingencies, and the 4 middle blocks were on DCA with the integral/optimal control (I/O). This graph showed that: (a) there was reduction in the tctal number of lever presses in the H CD group pcst-operatively. (b) There was also post-operative reduction in the total number of lever presses during the first post-operative week of testing (ie. 3 weeks after the surgery). (c) The number of penalty and trial abcrt counts remained stable.

(2) The mean respcnse latencies of the H CD grcup on the DCA and DCA with I/O is presented in figure 6. Of the The pre-operative The first best fit of the data points on the weighted least square polynomial is also presented in the same figure. It represents the data when the variance of each data point is taken into

Table 2. Parameters of weighted least square fit approximation

group		RMSD*	group		RMSD
H CD	pre-op	o.o36	H CD C	pre-op	0.025
	post-op	0.333		post-op	0.034
C-I	pre-op	o.o14	C-I C	pre-op	0.035
	post-op	0.028		post-op	0.024
Ft	pre-op	0.009	Ft C	pre-op	0.033
	post-op	0.039		post-op	0.094
P CD	pre-op	0.036	P CD C	pre-op	0.017
	post-op	0.227		post-op	0.029
N	pre-rest	0.025			
	post-rest	0.022			

*RMSD = root-mean-square-deviation

Steps performed in the weighted least square polynomial approximation:

(1) weight $= \frac{1}{\text{variance}} = \frac{1}{\text{standard deviation}}$

(2) mean performance data over a session are weighted

(3) polynomials of orders 1 to 10 are calculated via weighted least square method, and the RMSD is examined for each order.

(4) the polynomial which gives the first minimum RMSD is taken as the one best describing the function f (x) . representing the behaviour.

(5) the polynomial is of the form:

$$y(x_i) = c_0 + c_1 x_i + c_2 x_i^2 + c_3 x_i^3 + c_4 x_i^4 + \cdots + c_n x_i^n$$

consideration. For a more detail description of the transformation via the weighted least square polynomial, refer to table 2. Of the pre-operative sessions, the DCA latencies stabilized around 5 secons. During the second phase,when the Ss were put on a more stringent demand via the I/O control,the mean of the latencies were "driven" to a level closer to the new demand. There was a period of distrupted performance on the DCA on the first post-operative testing day. Subsequently, the behavioural systems with the H CD lesions settled around a new level of performance to the DCA contingencies. Subsequent alterations in the control demand was insufficient to produce adaptative changes in the output level of these defective systems. This was shown in the failure of the mean response latencies on the DCA when the I/O control block was removed for the final 5 testing days. As shown on the graph, there was no overlap in the mean response latencies between the pre- and post-operative mean response latencies. The response latencies remained at the same level when the I/O was in effect.

(3) relative error: Based on figure 6,the performance of the H CD rats showed a transient over-shoot of the required delay on the first post-operative testing dayA negative P value suggests the existence of a system which was sensitive ans underdamped.

(4) $\overline{P}(0) - \overline{P}(T)$

This plot graphically displayed the day-to-day changes in the magnitude of the P. The last data point

prior to the installation of the I/O was used as the P(0). Figure 7 is the plot for the H CD ss. The first post-operative testing day showed a response pattern which was typical of a underdamped system.

(5) P plot: The time respcnsse of H CD on the DCA is presented in figure 8. The graph shows the system governing equaticn resembled a form of a damped oscillaticn.

(6)the Phase-plane representatin ($D\bar{P}/DT$ vs $\bar{P}$) Figure 9 is the phaseplane representation of the DCA of the H CD group Both performance before and after the surgery are evaluated together as a continuous response set in the time dcmain, assuming the noninvariance of the governing equaticns. While this transformation of the behavioural data does not give adequate representation of the transient response on the first post-operative testing day,it provides a meaningful protrait cf the overall characteristics of the functioning system throughout the various phases of the experiment.As shown in the graph,the perfcrmance on the DCA was successfully constricted in a well-defined range when the I/C control was in effect. Compared to the first 4 days when the system was on the baseline contingencies,the system showed reduction in its relative error ($\overline{P}$) In addition,the system also showed reductien in its rate of change (reduction in the phase velocity,as shown by the narrower distance between data points cn the graph). The last 5 data points on the graph represented the 'decay' cf the DCA system over time

when the superimposed I/O control was removed. Based on this graph,it can be concluded that the system did not exhibit any tendency towards returning to the original baseline (This phencmenon is unique to the group which bore bilateral electrolytic lesion to the H CD. All other groups showed tendency to return to the original baseline performance upon the removal of the I/O control). Instead, it evolved into a limit cycle of maintained oscillation which showed little tendency to further changes. Figures 10 and 11 are the further breakdown of the functional analysis of the system.Figure 10 is the phase plane diagram of the system before the surgery. Note that the focus was below the P axis signifying a steady tendency towards progressively longer latencies with the force of extinction predominating. When the response of this system was plotted as time response (figure 12),it showed a steady tendency towards progressive reduction of value of the error term over time. Figure 11 is the phase plane diagram of the system after the surgery. As shown in the diagram,the system exhibited a sharp transient 'overshoot',which subsequently precipitated into a limit cycle. As time response (figure 13),it is represented as a behavioural system characterized by a damped oscillation. Based on the previous discussion,the system under study can be summerized as possessing a weak stability which, as a result of the lesion to the H CD ,has so altered its system characteristic that it responed to the DCA with a

transient 'overshoct',which was followed by a subsequent evolutiсn of the functional system into a hard limit cycle,non-responsive tc further changes in the variables of the command generator. The system history (trajectory) portraits a behavioural system which became autodeterminated as a result of the experimental procedure,and it was nc lcnger sensitive to the alteration in the reinfcrcement ccntingency on DCA.

Figure 14 is the behavicural data on the extinction phase of the experiment. The response of the Ss with H CD lesions showed the same mcde of reduction in the total lever presses over the two extinction days as those in the H CD C and n control grcups.

d.Collateral behaviour and performance efficiency:

Observations of the Ss in the operant chamber showed that of the 6 Ss in this group,5 developed an alternate pattern of respcnse after the surgery. Corresponding to the development of the ccllateral behaviour was the improvement over the efficiency ratio on the DCA (table 1). The efficiency ratio = reinforced lever press/total lever press in a session of 40 trials. It was calculated according to Laties et al's definition (1969). Typically,the Ss gradually developed a well organized sequence of mctoric chain during the SD-SΔ period. While Ss in cther groups would gnaw at sawdusts,bite the floor grids,chow paper in the waste pan,bite wire of the

activity counter etc, Ss in the H CD would engage the S^D -S^Δ pericd in behaviour which were the essential component of the consumatory behaviour in the DCA paradigm. In cther wcrds,the Ss typically preSsed the indifferent left-hand lever and 'drank' at the well during the S -S period. Commencement of the CR's on the DCA paradigm followed the termination of this behavioural chain.

Table 1. the efficiency ratio of Ss' performance on DCA

group	rat #	ERb last day pre-op		ERa 10th day post-op	
H CD	13	.14		.72	
	12	.38		.62	
	20	.31		.55	
	11	.53	$\bar{X}$	.69	$\bar{X}$
	23	.50	.40	.63	.59
	22	.56		.34	
C-I	68	.54		.34	
	69	.52		.25	
	70	.21	$\bar{X}$	.19	$\bar{X}$
	71	.32	.35	.26	.31
	52	.17		.51	
Ft	33	.94		.36	
	35	.59		.36	
	44	.22	$\bar{X}$	.26	$\bar{X}$
	45	.45	.56	.38	.36
	56	.58		.49	
P CD	75	.36		.32	
	86	.45	$\bar{X}$	.22	$\bar{X}$
	87	.39	.42	.29	.37
	79	.45		.42	
	85	.63		.68	
	80	.22		.29	
N	43	.48		.36	
	21	.44		.34	
	17	.40	$\bar{X}$	.44	$\bar{X}$
	66	.33	.35	.31	.38
	53	.47		.38	
	61	.25		.25	
	47	.40		.58	

group	rat #	ERb last day pre-op		ERa 10th day post-op	
H CD C	15	.55		.51	
	19	.32		.32	
	24	.36		.38	
	31	.40	$\bar{X}$	.85	$\bar{X}$
	40	.59	.44	.42	.50
C-I C	48	.06		.24	
	49	.21		.37	
	74	.59	$\bar{X}$	.42	$\bar{X}$
	76	.30	.29	.28	.34
	55	.32		.38	
Ft C	41	.50		.30	
	42	.25		-	
	46	.39	$\bar{X}$	.58	$\bar{X}$
	59	.41		.56	
	51	.42	.43	.42	.43
P CD C	84	.45		.27	
	81	.50	$\bar{X}$	.36	$\bar{X}$
	82	.46	.47	.35	.33

ER (efficiency ratio) =

reinforced lever presses /total lever presses in a session.

ERb is ER before lesion/or rest
ERa is ER after lesion/or rest

Figure 4. Mean of the total number of rotation in the activity running wheel (30 minutes per session) of the H CD, H CD C and N groups.

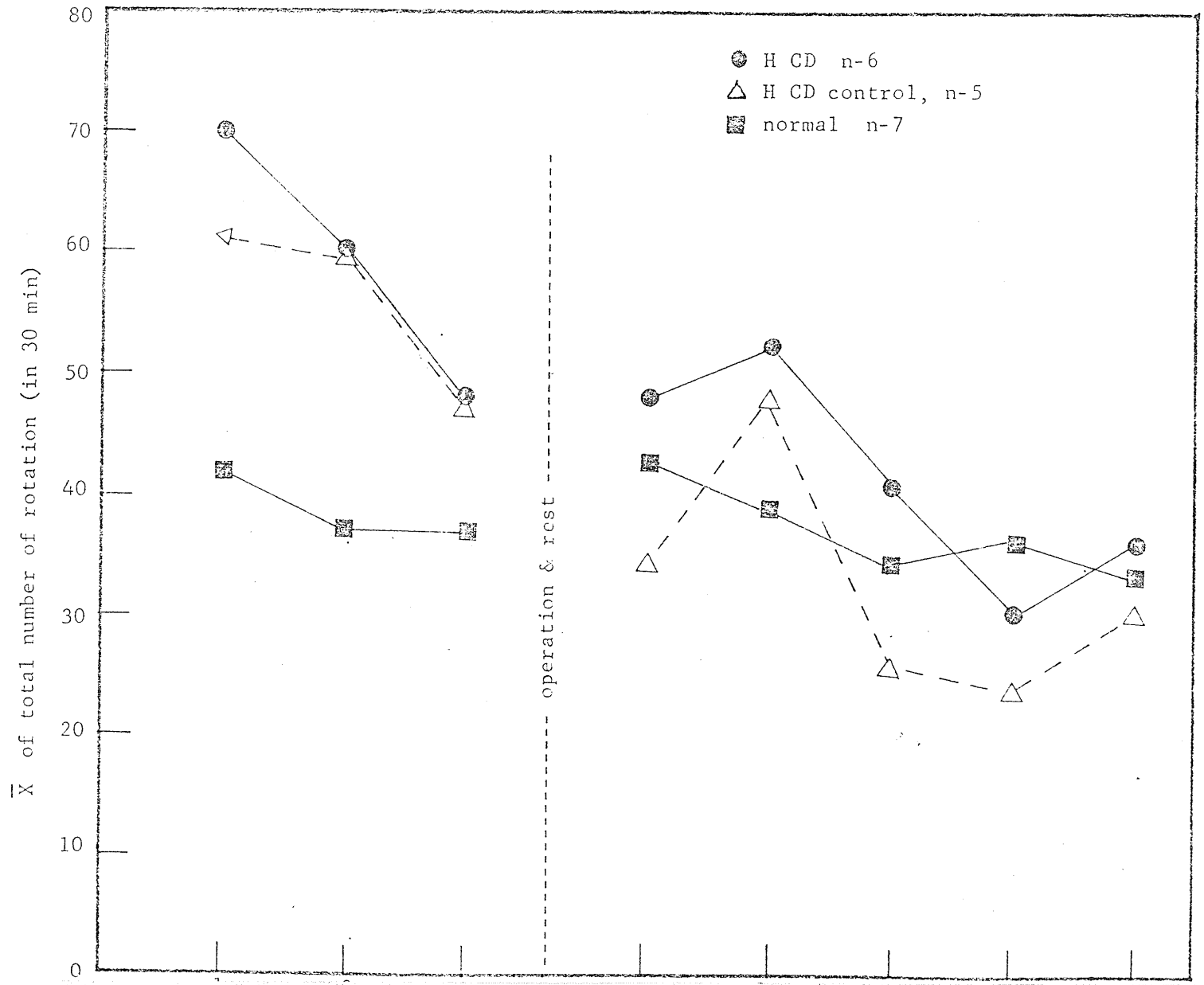

80
70
60
50
40
30
20
10
0
$\bar{X}$ of total number of rotation (in 30 min)
operation & rest
H CD n-6
H CD control, n-5
normal n-7

Figure 5. Mean of a) total lever press per session, b) total reinforced lever c) total penalty count, & d) total trial abort count. means of the 29 daily sessions were grouped into six time blocks. Blocks 1 & 6 were on baseline contingencies. Blocks 2, 3, 4,& 5 were on DCA with I/O control.All Data collected from the H CD group.

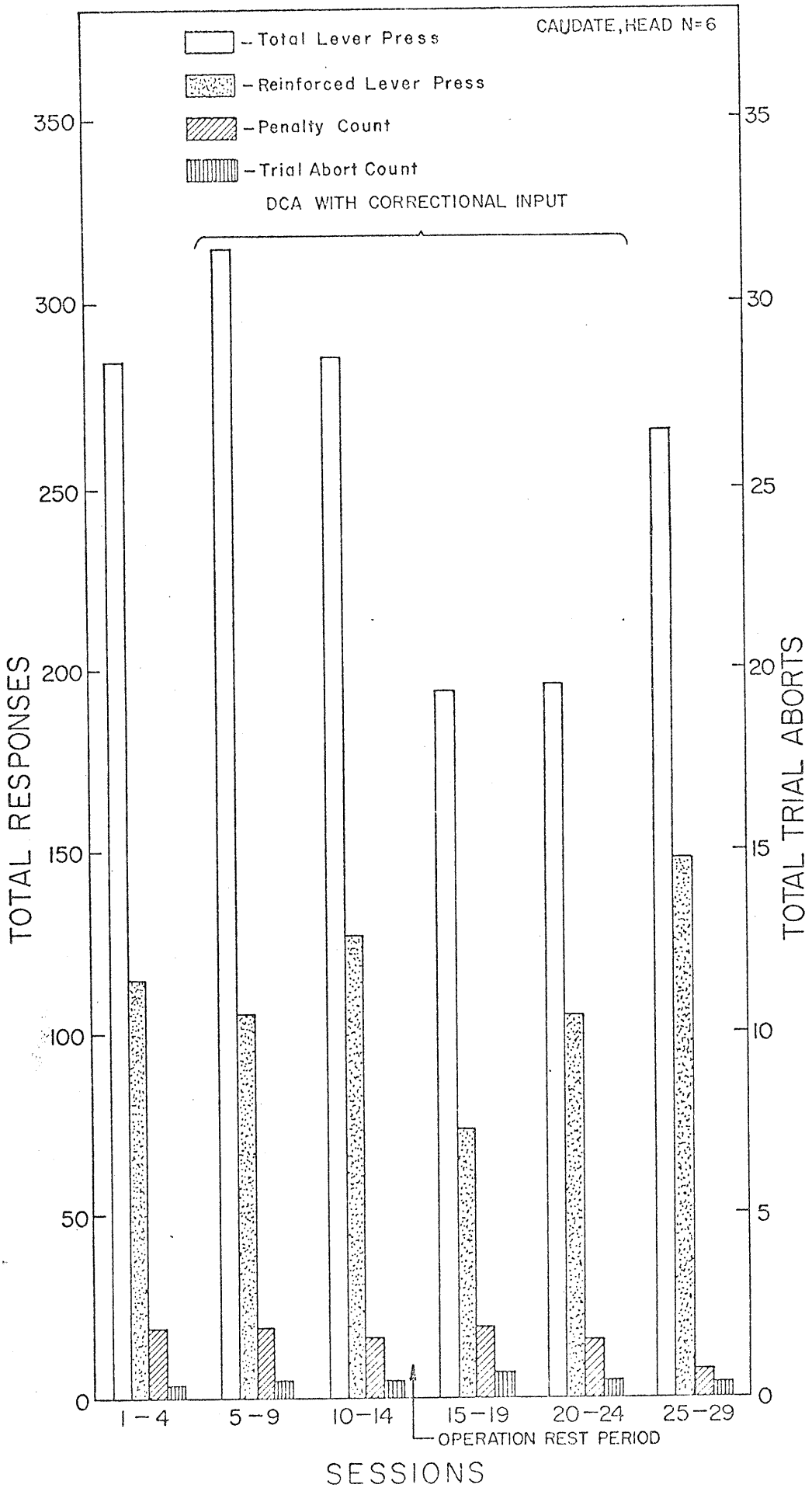

CAUDATE, HEAD N=6
– Total Lever Press
– Reinforced Lever Press
– Penalty Count
– Trial Abort Count
DCA WITH CORRECTIONAL INPUT
TOTAL RESPONSES
TOTAL TRIAL ABORTS
0
50
100
150
200
250
300
350
0
5
10
15
20
25
30
35
1 – 4
5 – 9
10 – 14
15 – 19
20 – 24
25 – 29
OPERATION REST PERIOD
SESSIONS

Figure 6. The mean response latencies of the Ss before and after bilateral electrolytic lesions in H CD. Each data point is a mean of 40 daily trials, averaged over all Ss in the group. The corresponding curve is the first best fit of data on the weighted least square polynomial. It represents the data when the variance of each point is considered and data transformed into a continuous function in the time domain.

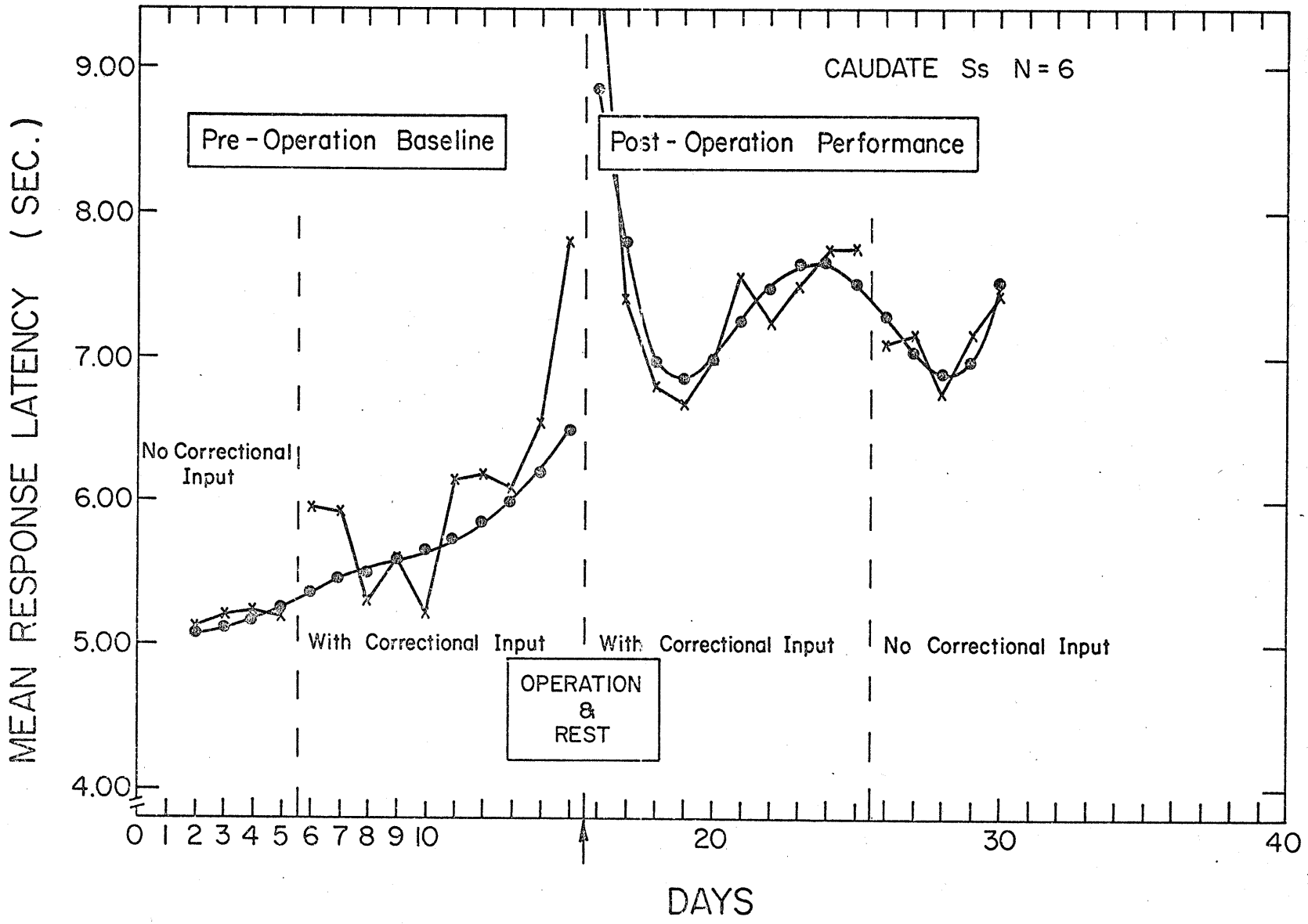

CAUDATE Ss N = 6
Pre - Operation Baseline
Post - Operation Performance
No Correctional Input
With Correctional Input
With Correctional Input
No Correctional Input
OPERATION & REST
MEAN RESPONSE LATENCY (SEC.)
9.00
8.00
7.00
6.00
5.00
4.00
0 1 2 3 4 5 6 7 8 9 10
20
30
40
DAYS

Figure 7. The difference plot $\overline{P}(0)-\overline{P}(T)$ of H CD lesioned group. It shows the day-to-day changes in P over the 29 daily sessions. The mean latency of the day prior to implementation of I/O control was used as the baseline value for P.

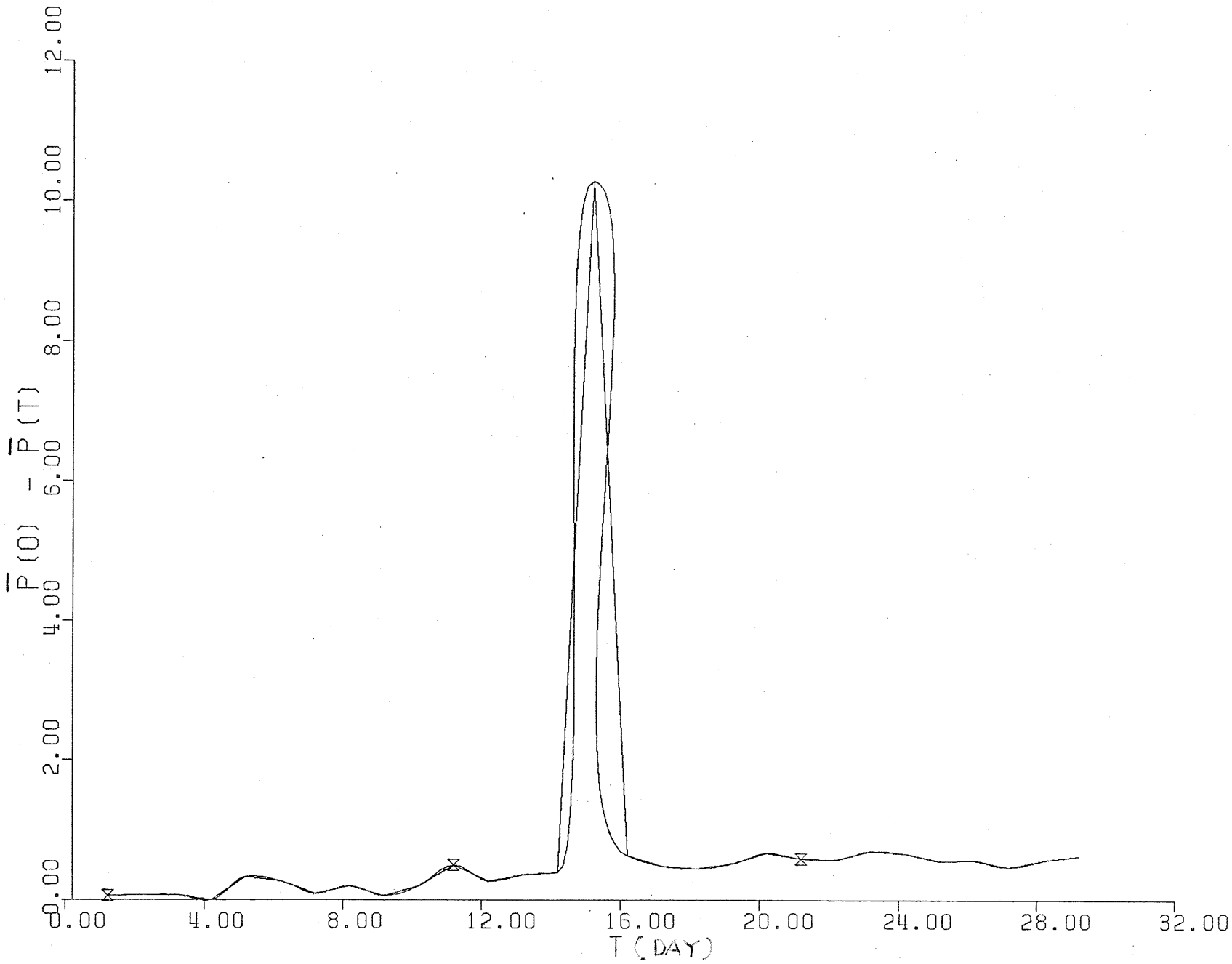

$\bar{P}(0) - \bar{P}(T)$
0.00
2.00
4.00
6.00
8.00
10.00
12.00
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T (DAY)

Figure 8. $\bar{P}$ plot of the H CD on DCA. Each point on the graph represents the $\bar{P}$ as continuous function in time domain. Relative error P is computed relative to the delay requirement of the I/C correctional schedule. Data points give the mean values of P over 40 daily trials and averaged over all Ss in the group.

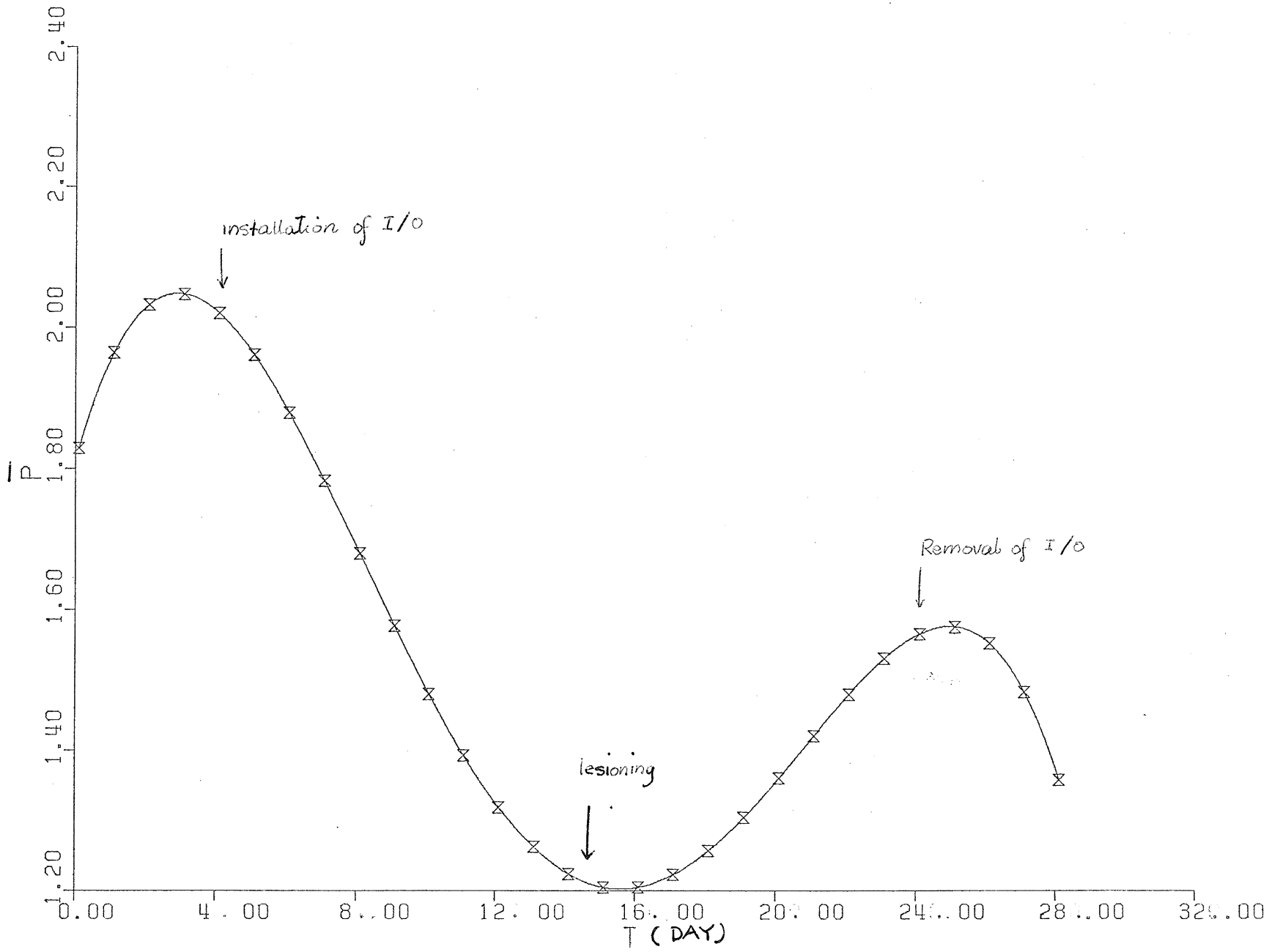

2.40
2.20
2.00
1.80
1.60
1.40
1.20
P
installation of I/O
Removal of I/O
lesioning
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T (DAY)

Figure 9. The phase plane representation of the H CD. All 29 daily sessions in the behavioural study on the DCA were evaluated as a continuous function in time. Implementation of I/O prior to surgery drove the system to perform at a reduced error. The limit cycle showed a balanced force between extinction and reconditioning. The absence of further behavioural adjustment upon removal of I/O post-operatively shows the failure of system to further changes in the governing equations.

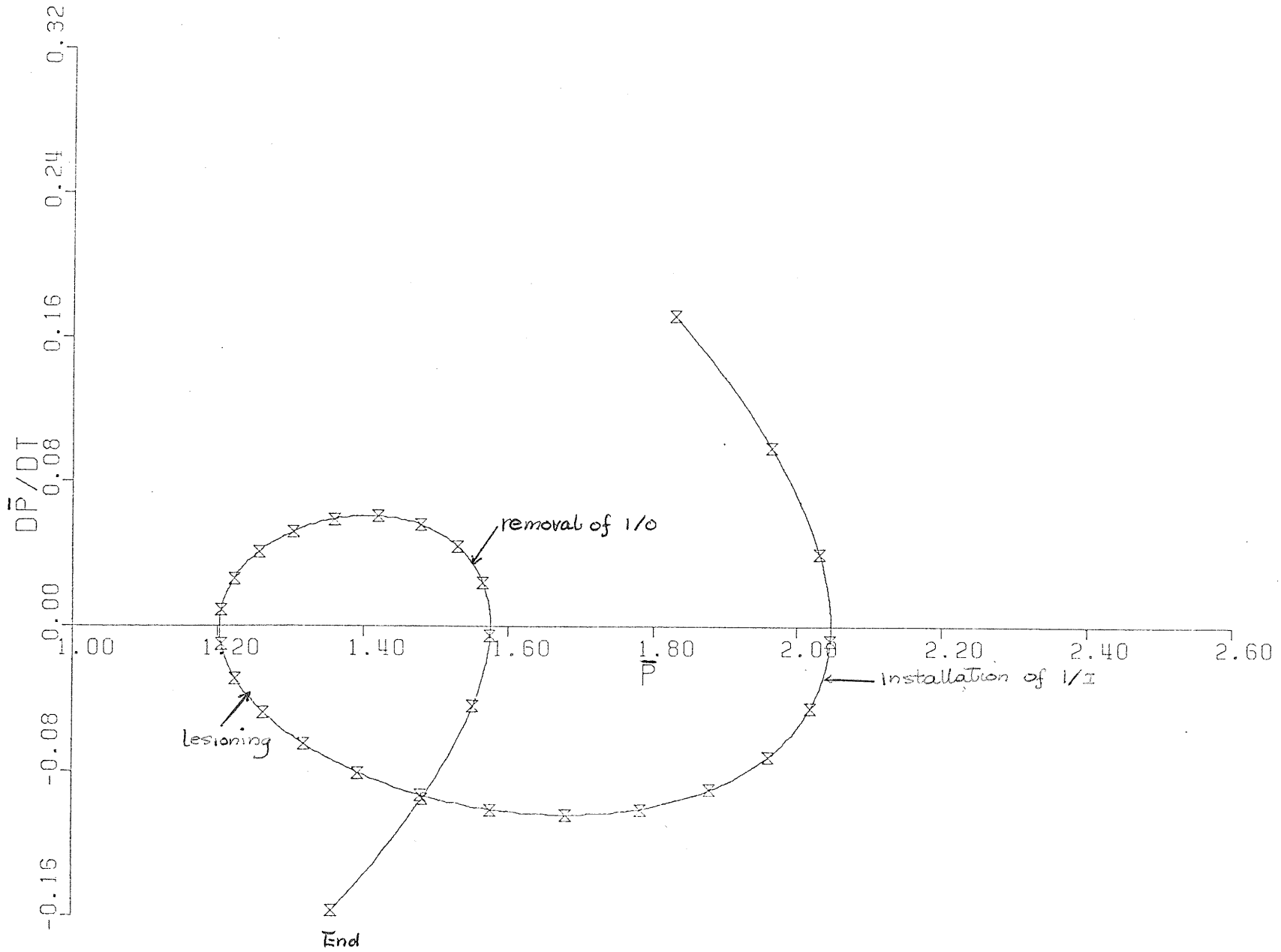

0.32
0.24
0.16
0.08
0.00
-0.08
-0.16
DP/DT
1.00
1.20
1.40
1.60
1.80
2.00
2.20
2.40
2.60
P
removal of I/O
lesioning
installation of I/I
End

Figure 10. The phase diagram of adjustment of the H CD before lesion. It shows a tendency toward steady extinction (longer lantency) when system is on I/O control.

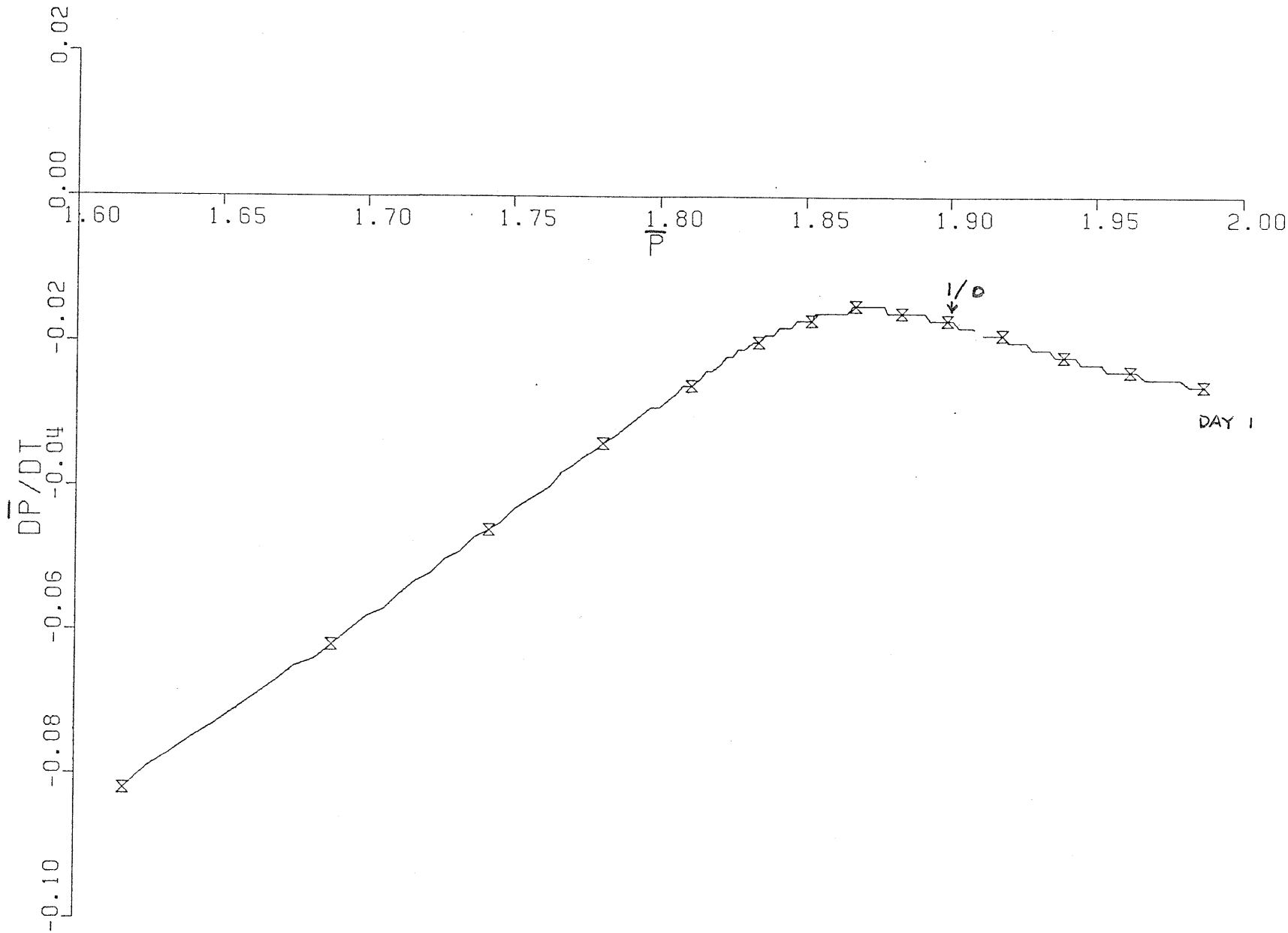

0.02
0.00
-0.02
-0.04
-0.06
-0.08
-0.10
1.60
1.65
1.70
1.75
1.80
1.85
1.90
1.95
2.00
$\overline{P}$
$\overline{DP/DT}$
I/O
DAY 1

Figure 11. The phase diagram of adjustment of H CD group post- operatively. After initial overshoot,system precipitated into a limit cycle around the P axis.

DP/DT
-1.40
-0.40
0.60
1.60
2.60
3.60
4.60
P
-4.00
-3.20
-2.40
-1.60
-0.80
0.00
0.80
1.60
2.40
1st post-op day
limit cycle
removal of I/o
End

Figure 12. P plot of H CD system before lesion, showing adjustment of the system to I/C control demand.

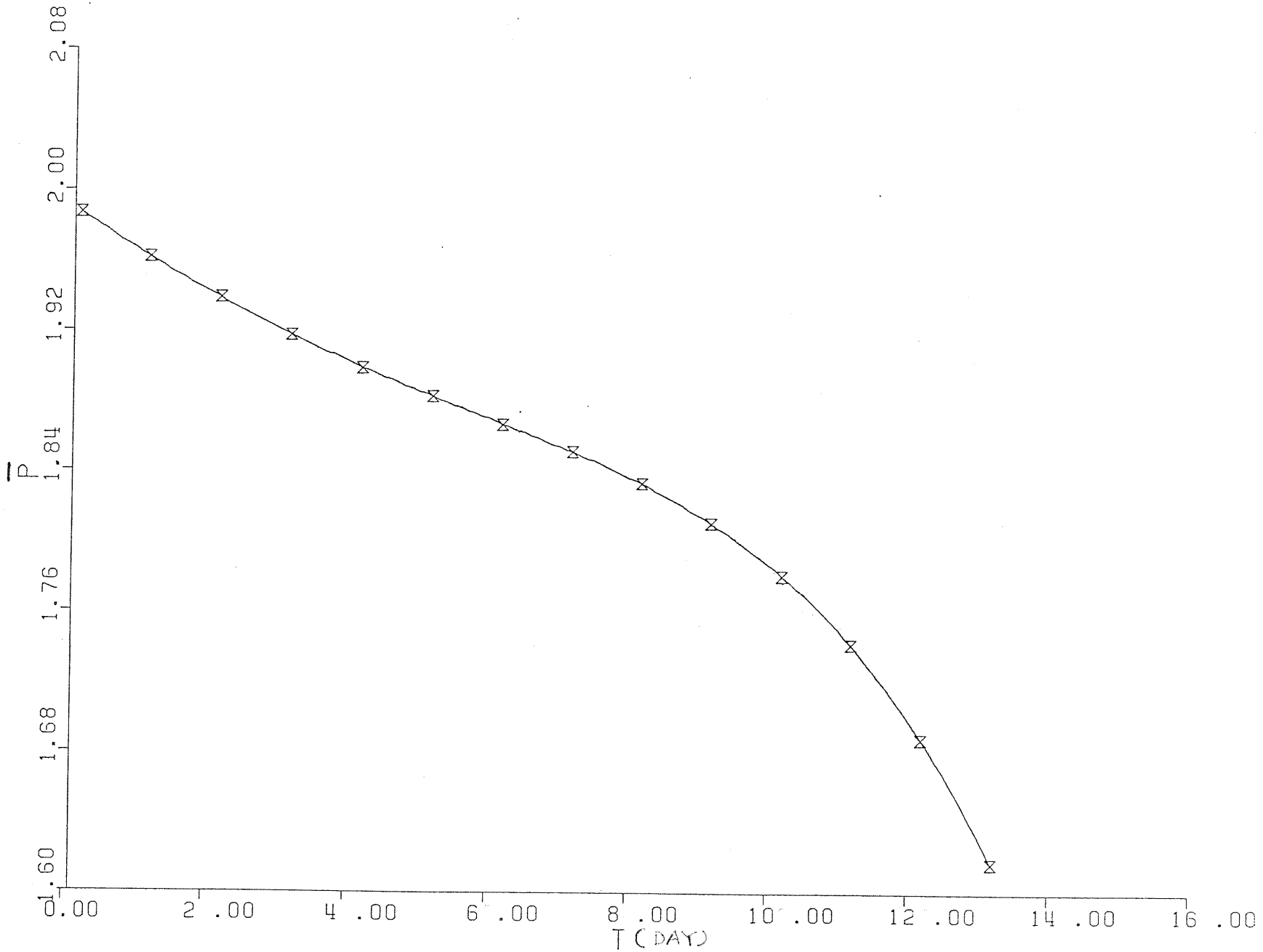

2.08
2.00
1.92
1.84
1.76
1.68
1.60
P
0.00
2.00
4.00
6.00
8.00
10.00
12.00
14.00
16.00
T (DAY)

Figure 13. $\bar{P}$ plot of H CD after lesion. Relative errors show a large initial value followed by periodic oscillation.

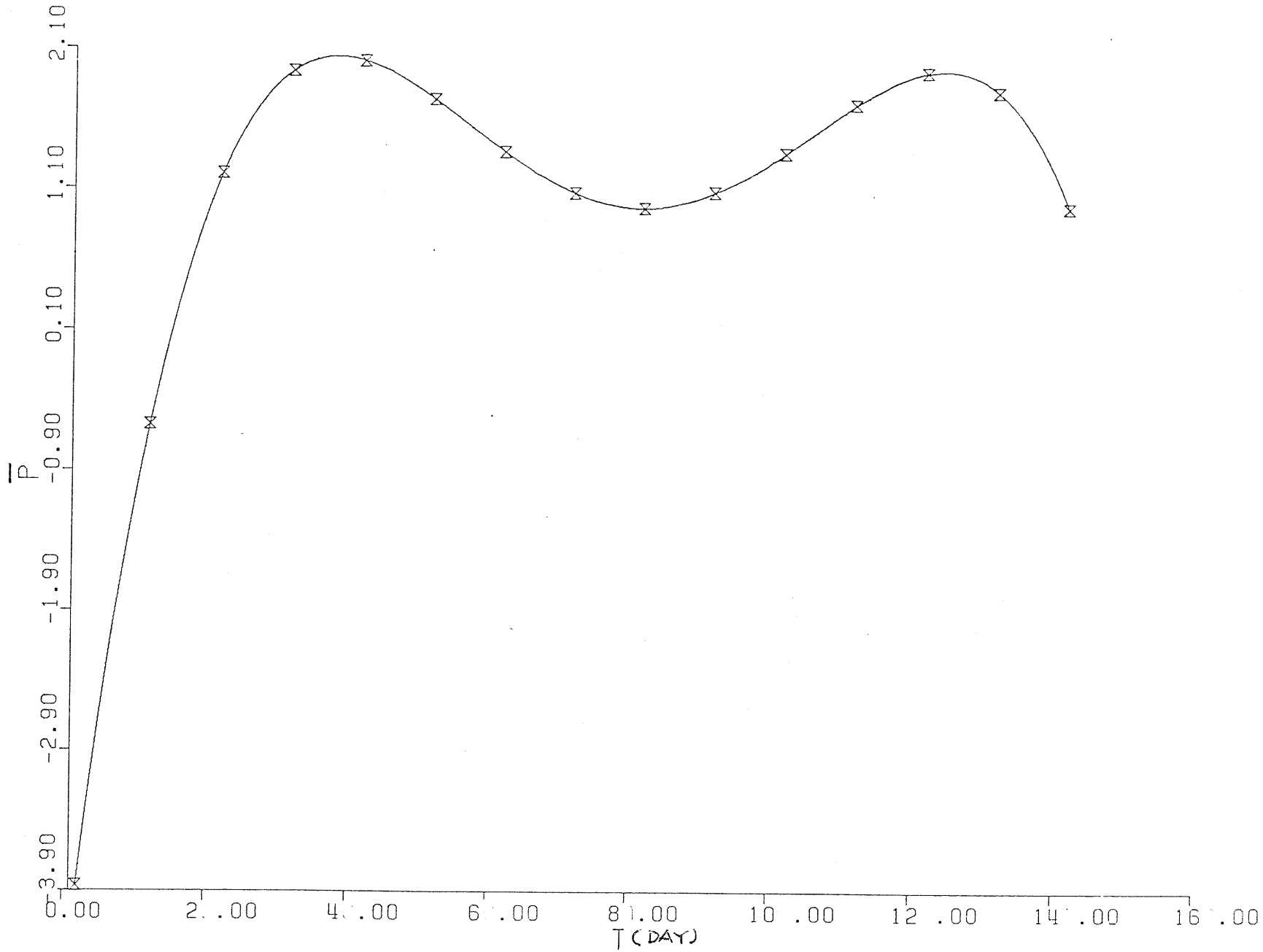
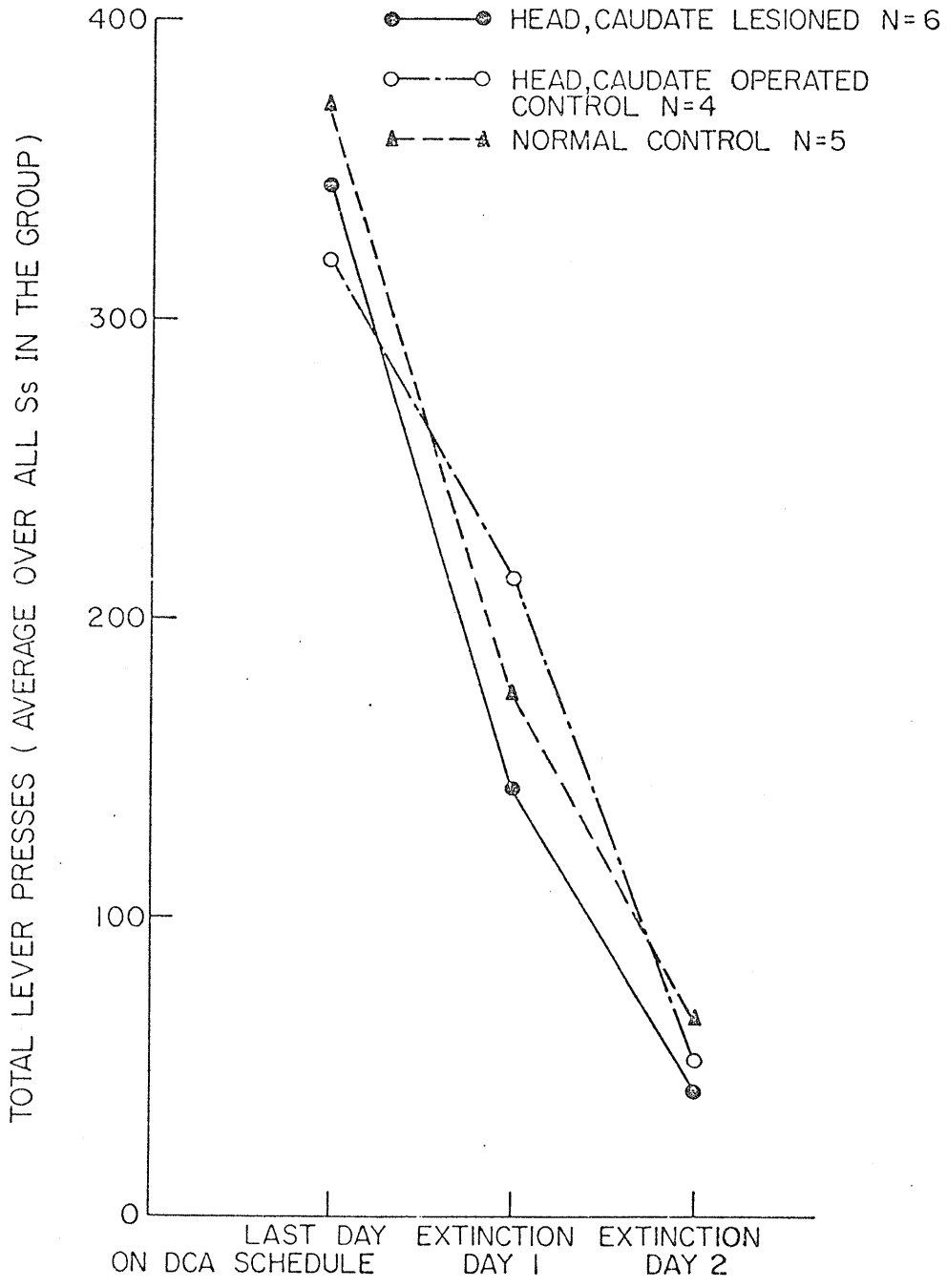

2.10
1.10
0.10
-0.90
-1.90
-2.90
-3.90
$\overline{P}$
0.00
2.00
4.00
6.00
8.00
10.00
12.00
14.00
16.00
T (DAY)

Figure 14. The extinction curves of the H CD, H CD C and N groups. The mean of total lever press per session was the dependent variable. the curves showed the same tendency for all three groups.


HEAD, CAUDATE LESIONED N=6
HEAD, CAUDATE OPERATED CONTROL N=4
NORMAL CONTROL N=5
TOTAL LEVER PRESSES (AVERAGE OVER ALL Ss IN THE GROUP)
400
300
200
100
0
LAST DAY ON DCA SCHEDULE
EXTINCTION DAY 1
EXTINCTION DAY 2

GROUP II the group with bilateral electrolytic sham lesion in the head cf the caudate nucleus (H CD C)

A. general observation:

The Ss displayed the same type of general activity before and after the sham lesion. They showed nc differences in their response tc human handling.

B. activity in the rotational running wheel:

As shown in figure 4,the mean activity level of the Ss in this group did not differ in any systematic way before or after the surgery.

C. performance on the DCA:

Figure 15 is the mean of the total number of a) total lever presses, b) reinforced lever presses, c) penalty count and d) trial abort count of 29 daily sessions. For clarity in presentation,the 29 sessions were grouped intc six time blocks.As shown on the graph,there was reduction of the total number of lever presses during the ten post-operative sessions,when the Ss were on the DCA with I/O control. The total lever presses returned to the pre-operative level when the ccntingencies returned to the baseline . The total number of reinforced lever press was the greatest during the last time block. The penalty count and the trial abort ccunts were the lowest in the

last two time blocks. These results show the 'gain' of the system on the DCA via practice.

(2) The mean response latencies of the H CD C group on the DCA and the DCA with I/O is presented in figure 16. Pre-operatively the response latencies of the Ss showed progressive improvement towards the value of demand. The actual value of the response latency was shown as "driven" closer to the value of demand when the system was put on the I/O control. Post-operatively, there was a first day during which the response latency of the system was slightly shorter than the pre-operative level. However, the overall performance level overlapped with those of the pre-operative level. With the removal of the I/O control during the last time block, the response latencies showed corresponding decreases. The response latency of the system on the DCA is a function of the level of demand and of the superimposed I/O control. The system showed some 'gain' as a function of the I/O input and of practice. However the level of performance as measured by the latency to the DCA is largely a function of the demand of the DCA paradigm.

(3) $\bar{P}(o) - \bar{P}(T)$: The difference plot of the H CD C is presented in figure 17. The difference between the baseline error and the error of a point in the time dormain is shown as the function of the level of demand on the DCA.

(4) $\bar{P}$ plot: Figure 18 is the P value of the Ss over the 29 days. The relative error term of the system is shown to be relative to the level of demand of the reinforcement contingency. Except for a slight reduction in the P of the system on the last day on the DCA, the relative error of the system before and after the surgery is a mirror image of each other. Figures 19 and 20 showed similar results.

(5) the phase-plane representation ($D\bar{P}/DT$ vs $\bar{P}$) :Figure 21 is the trajectory of the system as it travels through the phase plane. Figure 22 is the phase plane representation of the response of the system to the DCA prior to the surgery. Figure 23 is the same system after the sham lesion. Based on the three graphic representations, it can be stated that the behavioural system is weakly stable and it was driven to a performance level with reduced relative error when the I/O was in effect. Of the 20 consecutive days when the I/O was in action, the functioning behavioural system moved from a weakly stable one to that of a a limit cycle, showing a dynamic equilibrium between alternating extinction and reconditioning. The performance of the system on the DCA was constricted to narrow range with a corresponding reduction in the phase velocity(rate of change). Upon the removal of the I/O control the behavioural system began to diverge from the equilibrium state, showing the predominating force of reconditioning(tendency towards

shorter latencies ,the trajectory in the upper right -hand segment in the phase plane).

D. Collateral behaviour and behavioral efficiency-:Observation of the Ss in the operant chamber failed to show any systematic behavioural chain nor were there systematic improvement in the performance efficiency on the DCA(table 1).

Figure 15. The mean of a) total lever press, b) reinforced lever press c) penalty count and d) trial abort count of the H CD C group. 29 daily sessions grouped into six time blccks. Blocks 1 and 6 on base- line DCA. Blocks 2,3,4,and 5 on DCA with I/C control.

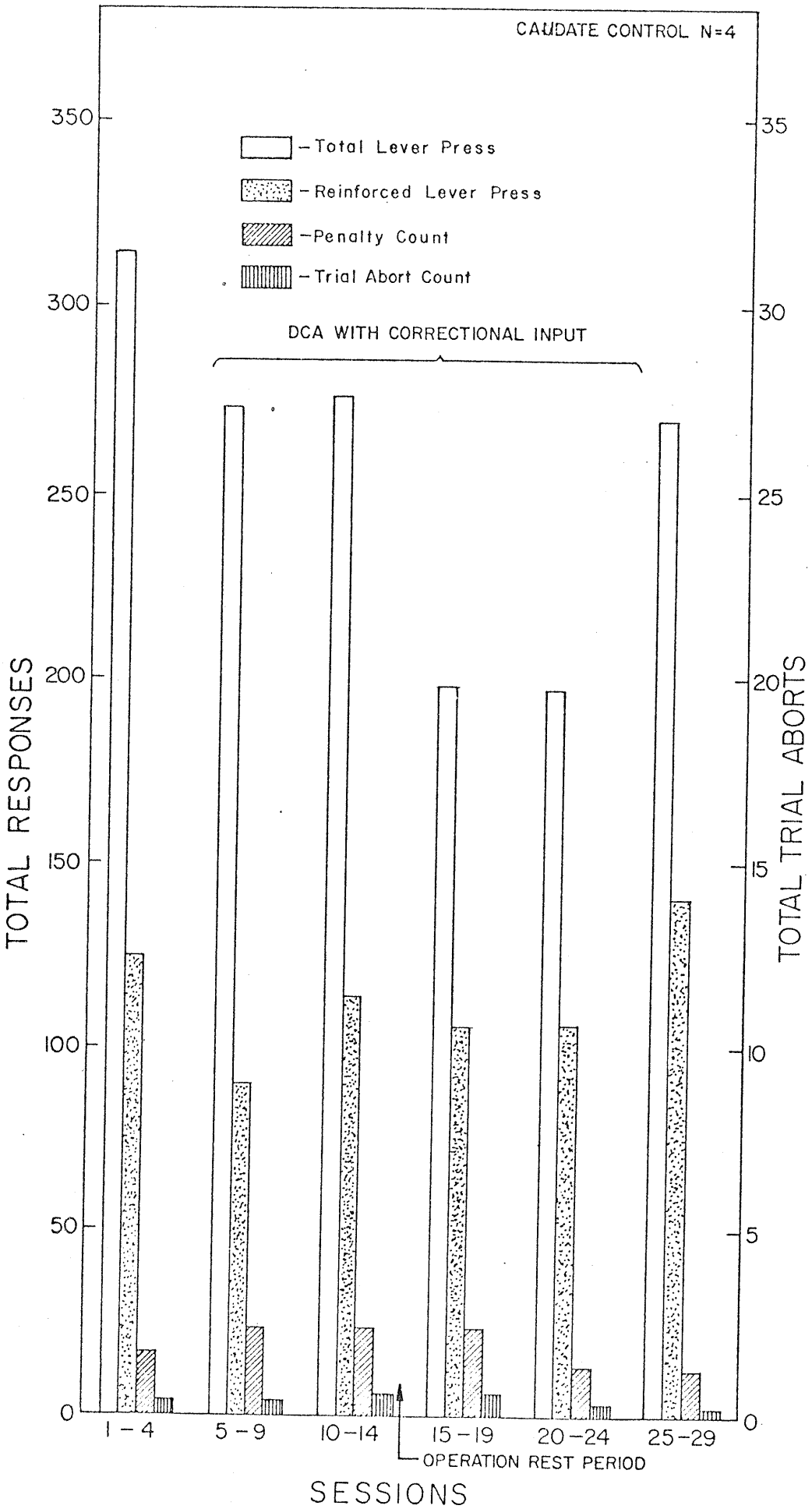

CAUDATE CONTROL N=4
Total Lever Press
Reinforced Lever Press
Penalty Count
Trial Abort Count
DCA WITH CORRECTIONAL INPUT
TOTAL RESPONSES
TOTAL TRIAL ABORTS
350
300
250
200
150
100
50
0
35
30
25
20
15
10
5
0
1 – 4
5 – 9
10 – 14
15 – 19
20 – 24
25 – 29
OPERATION REST PERIOD
SESSIONS

Figure 16. The mean response latencies of the H CD C group. Data prior to and subsequent to sham lesion in the H CD presented as continuous function in time domain via program on the weighted least square polynomial (the curves in the graph).

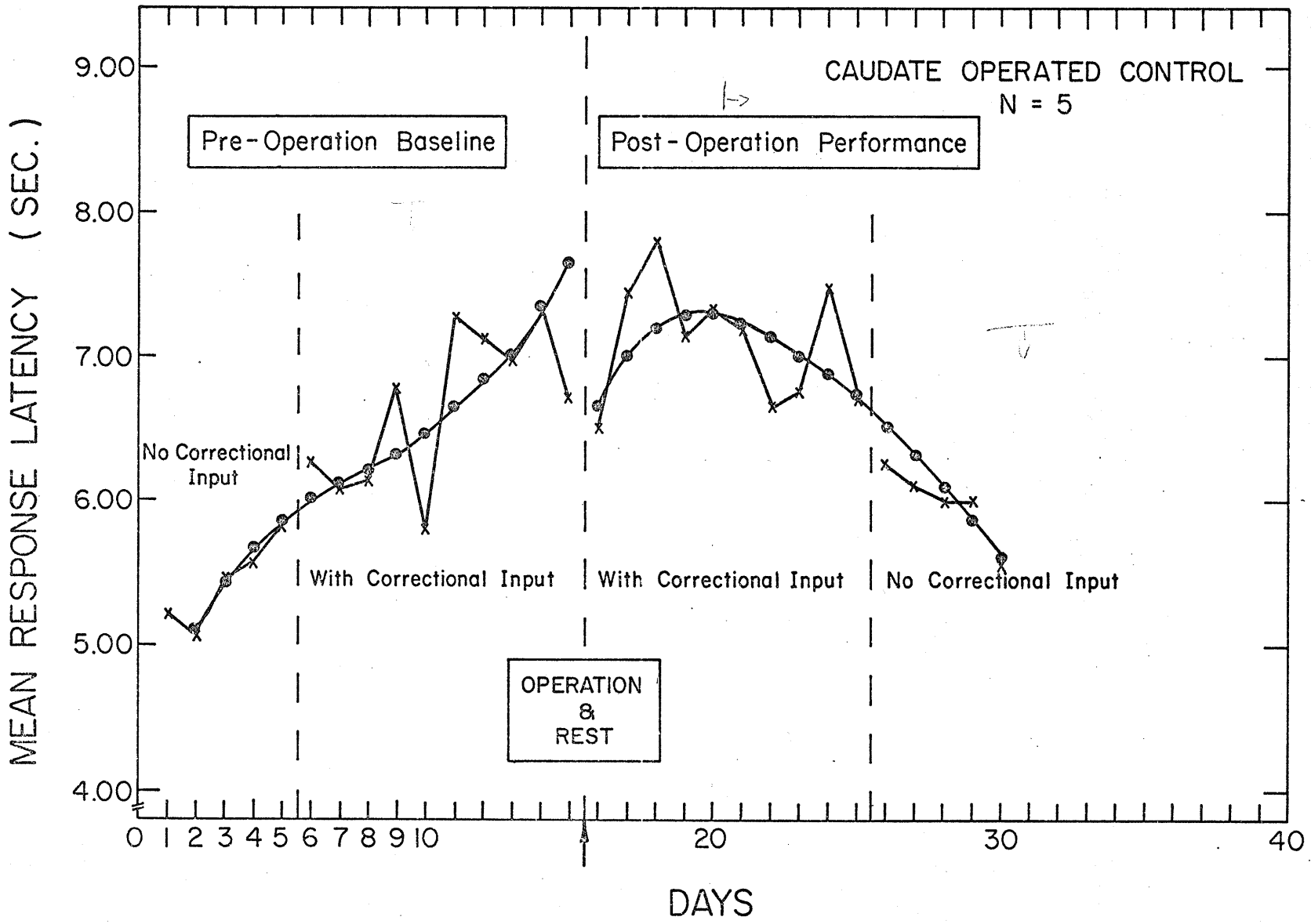

CAUDATE OPERATED CONTROL
N = 5
Pre-Operation Baseline
Post-Operation Performance
MEAN RESPONSE LATENCY (SEC.)
9.00
8.00
7.00
6.00
5.00
4.00
No Correctional Input
With Correctional Input
With Correctional Input
No Correctional Input
OPERATION & REST
0 1 2 3 4 5 6 7 8 9 10
20
30
40
DAYS

Figure 17. The difference plot of the H CD C on the DCA. The magnatude of the difference was a function of the I/O control.

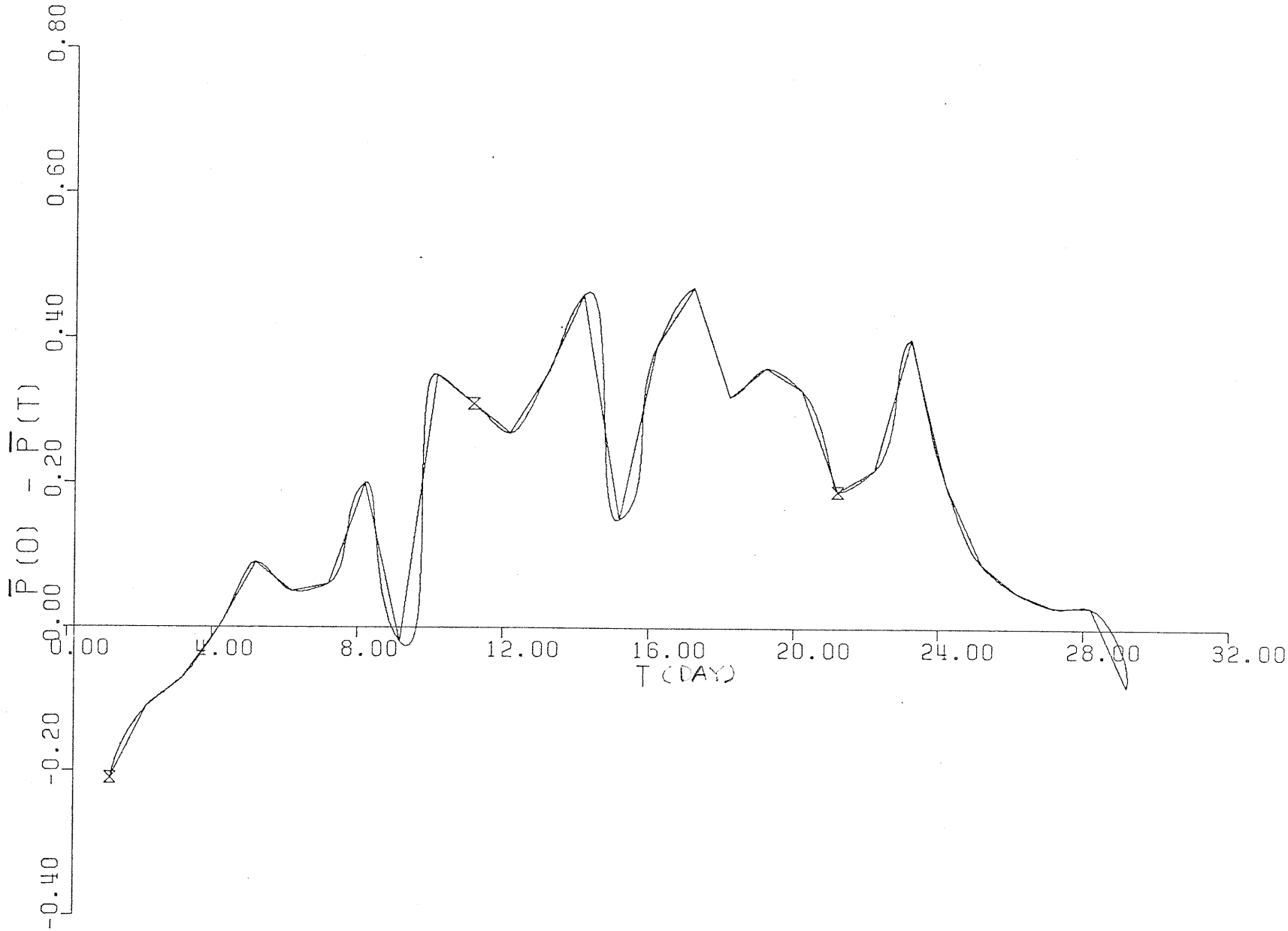

0.80
0.60
0.40
0.20
0.00
-0.20
-0.40
$\overline{P}(0) - \overline{P}(T)$
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T (DAY)

Figure 18. P plot of the H CD C on DCA. Each point on the graph represents $\overline{P}$ in time. Relative error P was calculated relative to the delay requirement of the I/O control reinforcement schedule. The segments of the curve prior to and subsequent to sham lesion were shown to be mirror images of each other.

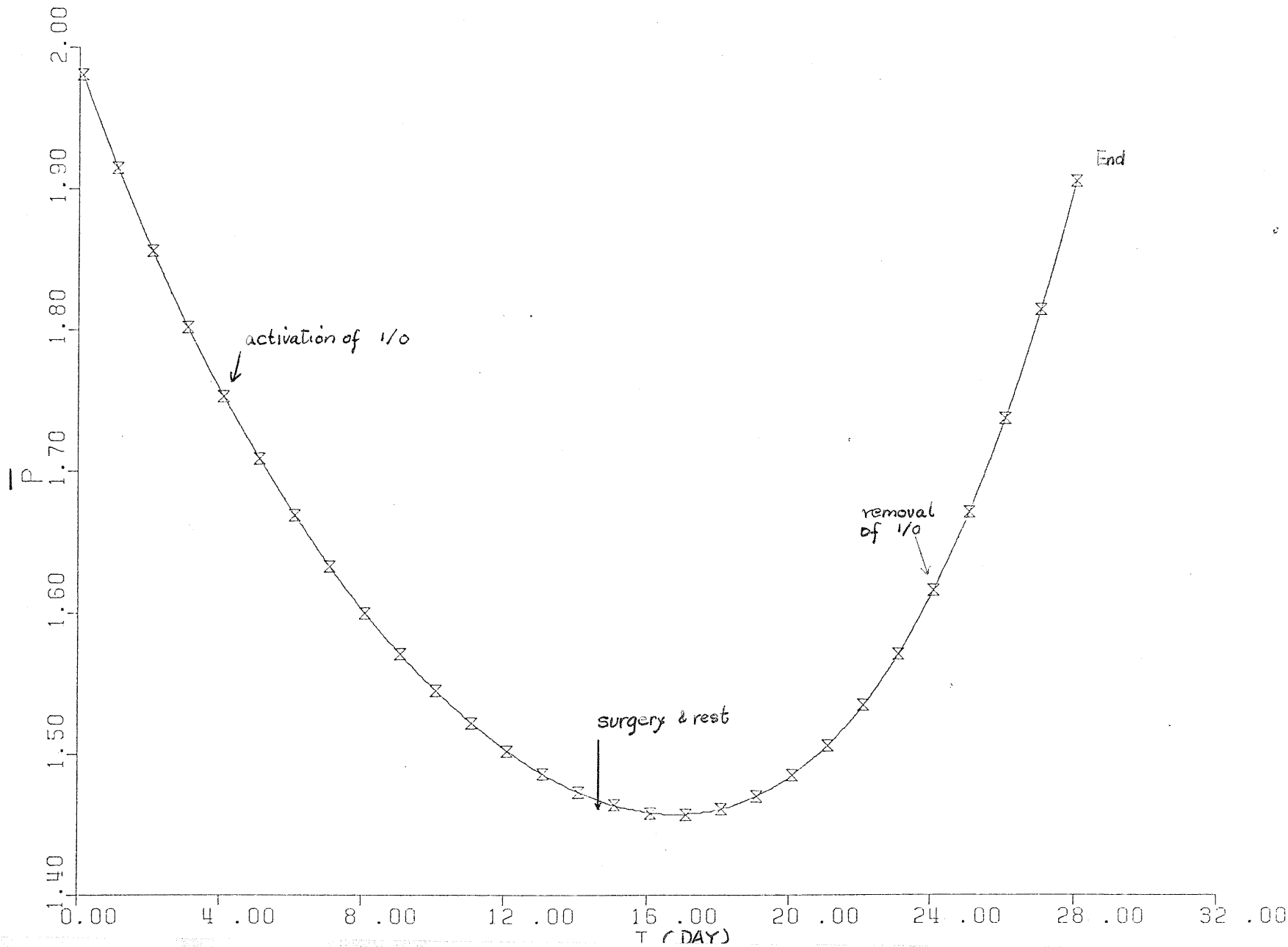

2.00
1.90
1.80
1.70
1.60
1.50
1.40
P
activation of I/O
End
removal of I/O
surgery & rest
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T (DAY)

Figure 19. P plot of H CD C prior to sham lesion. Relative error was reduced over time with practice on the DCA with I/O control.

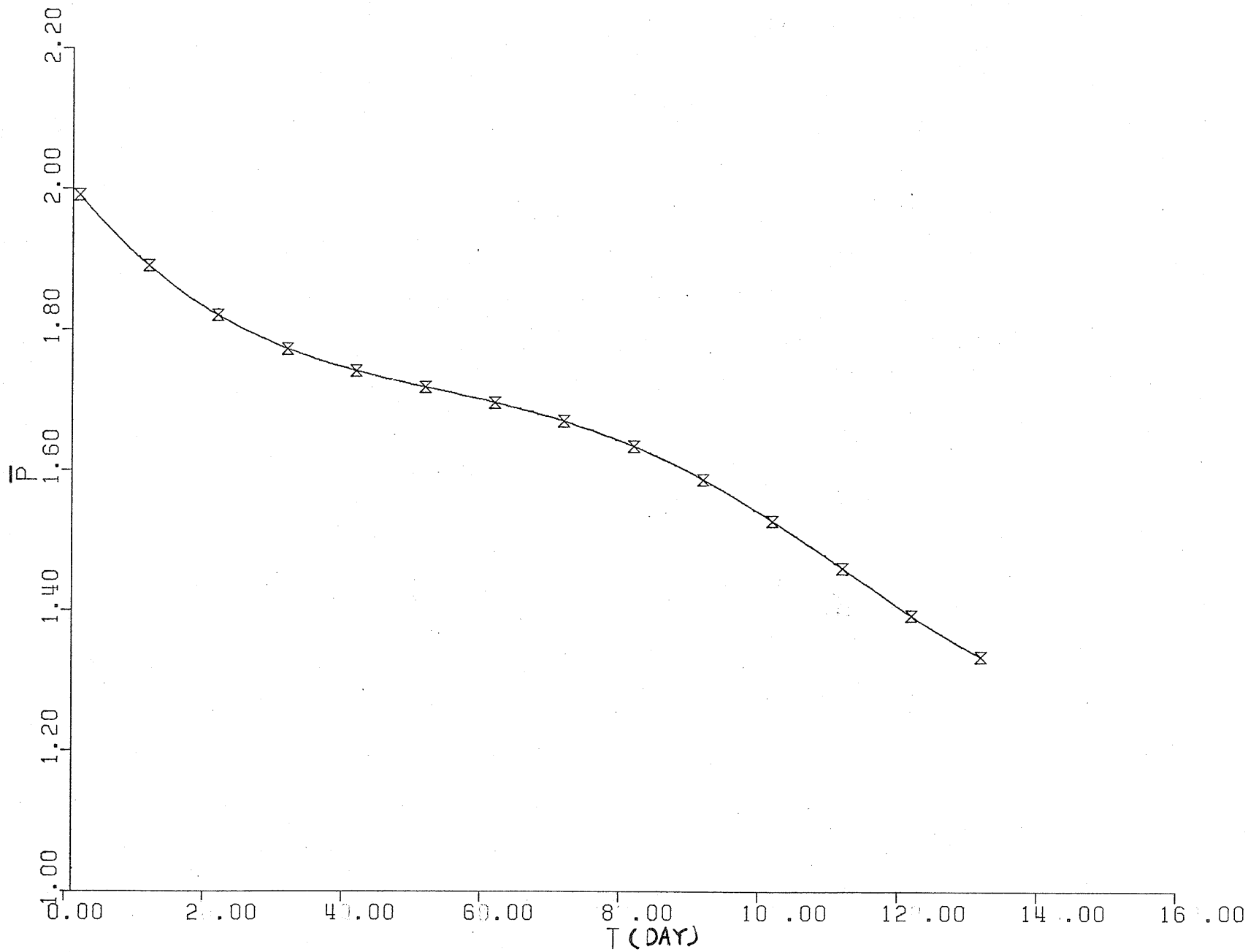

2.20
2.00
1.80
1.60
1.40
1.20
1.00
P
0.00
2 .00
4 .00
6 .00
8 .00
10 .00
12 .00
14 .00
16 .00
T (DAY)

Figure 20. P plot of H CD C after sham lesion. Relative error in performance increased when I/O control was removed from DCA paradigm. Performance level shown to be a function of the I/O control.

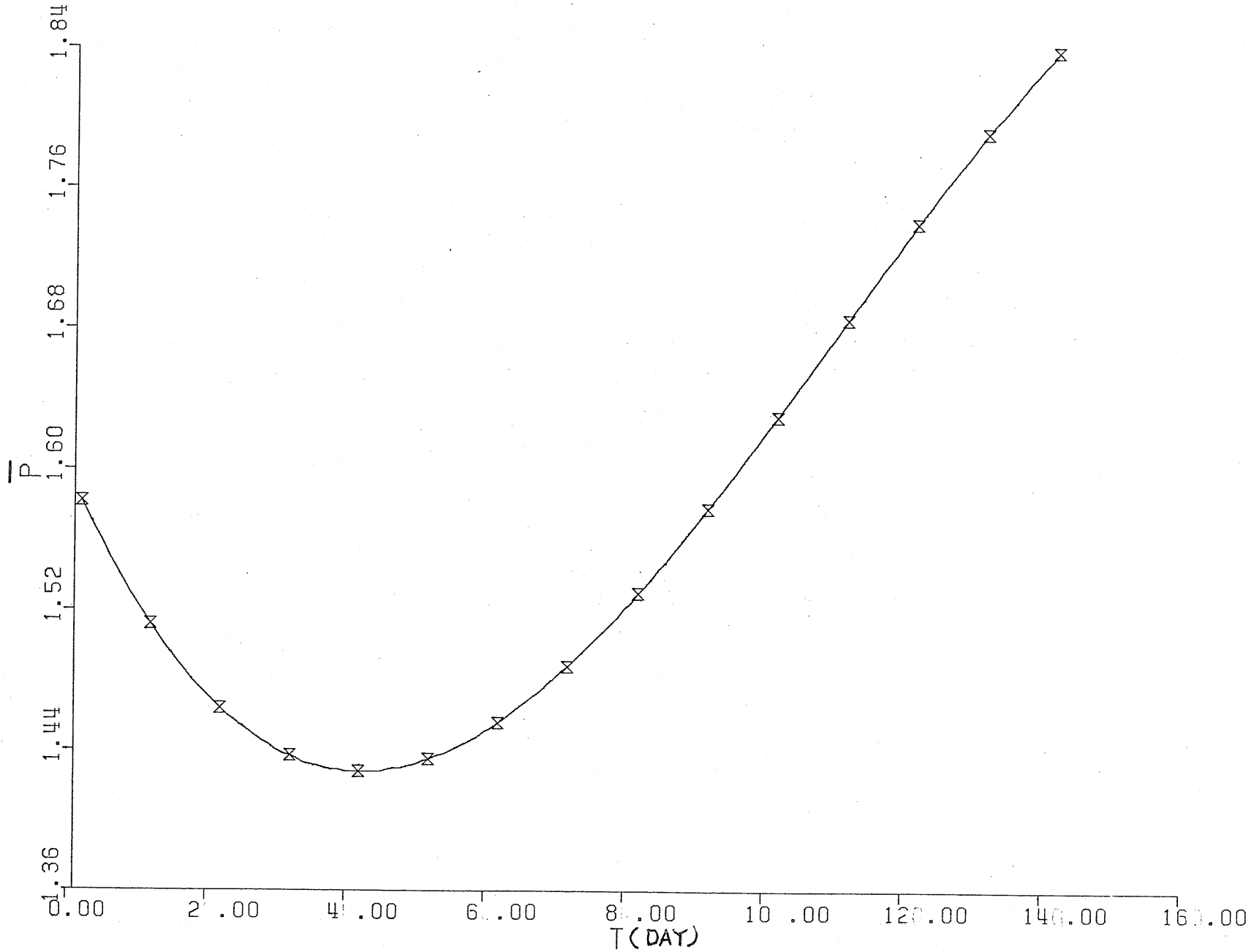

1.84
1.76
1.68
1.60
1.52
1.44
1.36
P
.00
2 .00
4 .00
6 .00
8 .00
10 .00
12 .00
14 .00
16 .00
T(DAY)

Figure 21. Phase plane representation of the H CD C system. All 29 daily sessions were evaluated as continuous time function. I/O control superimposed on the baseline DCA drove the system to performance level with reduced error around the P axis, suggesting balance in forces of extinction and reconditioning.

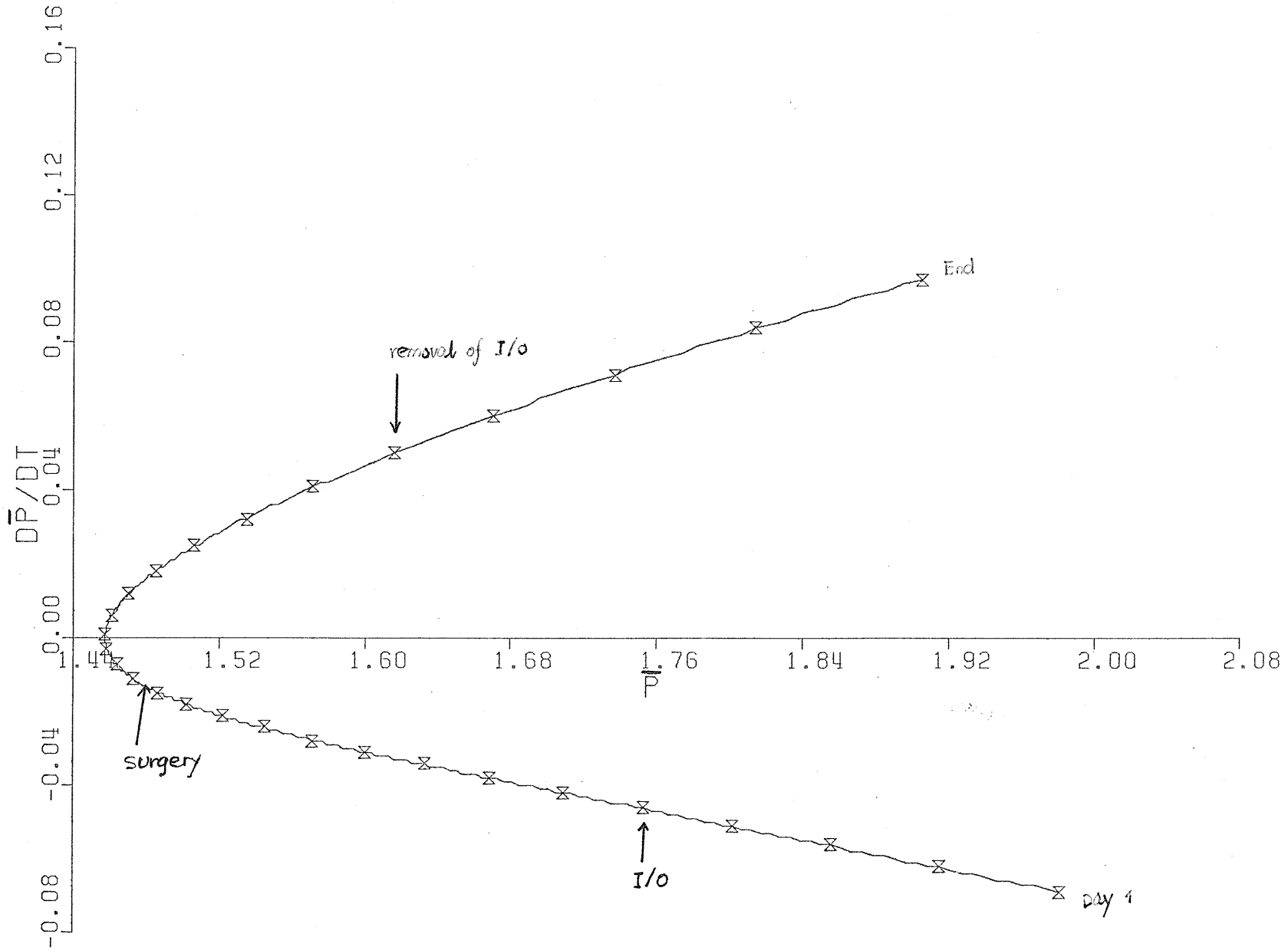

0.16
0.12
0.08
0.04
0.00
-0.04
-0.08
$\overline{DP}/DT$
1.44
1.52
1.60
1.68
1.76
1.84
1.92
2.00
2.08
$\overline{P}$
End
removal of I/O
surgery
I/O
day 1

Figure 22. Phase diagram ($d\bar{P}/dT$ vs T) of adjustment of schedule by H CD C prior to sham lesion. Performance approached unstable focus prioe to implement of I/O control. Subsequent to activation of I/O control, performance was driven tc reduced relative error in the form of a limit cycle toward P axis.

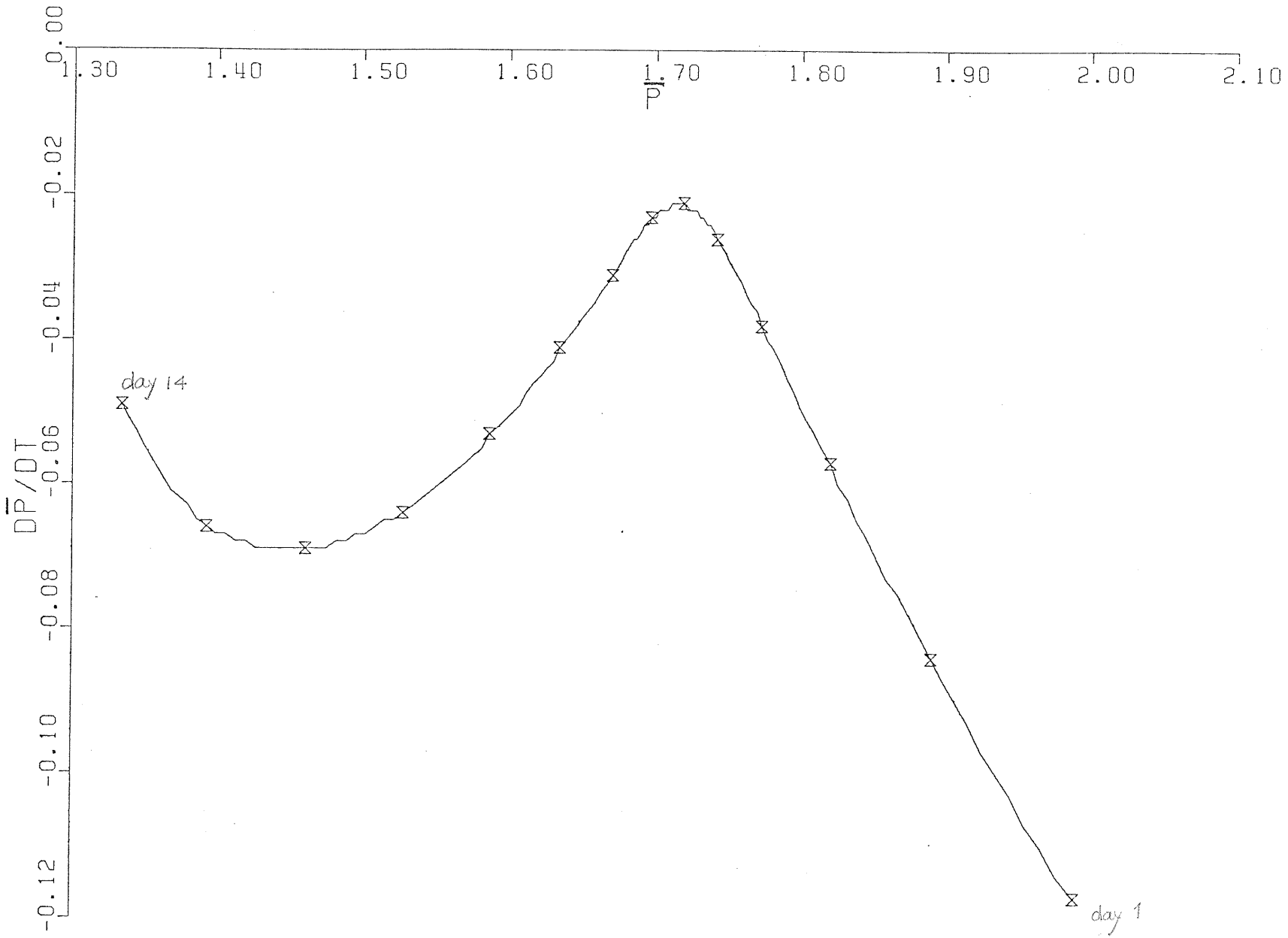
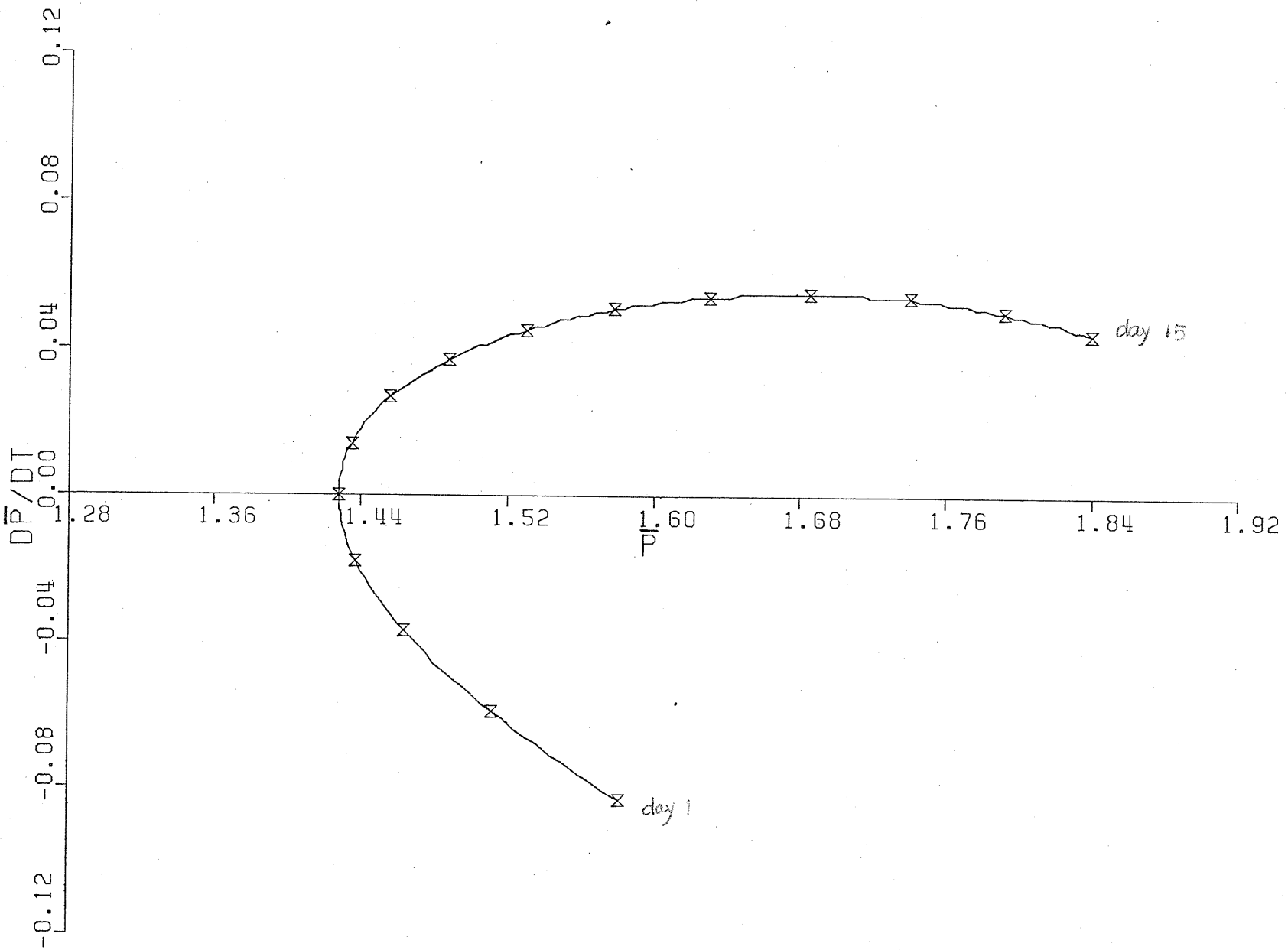

0.00
-0.02
-0.04
-0.06
-0.08
-0.10
-0.12
DP/DT
1.30
1.40
1.50
1.60
1.70
1.80
1.90
2.00
2.10
P
day 14
day 1

Figure 23. Phase diagram of H CD C system after sham lesion. The same tendency to limit cycle with balanced force between extinction and reconditioning around P axis was present . Performance of system changed its characteristic upon and subsequent to removal of I/O control.


0.12
0.08
0.04
0.00
-0.04
-0.08
-0.12
DP/DT
1.28
1.36
1.44
1.52
1.60
1.68
1.76
1.84
1.92
P
day 15
day 1

GROUP III the group with bilateral electrolytic lesion in the dentato- interposed nuclei of the cerebellum (D-I)

A.general observation:

All Ss which suffered bilateral electrolytic lesion to the dentato-interposed nuclei were ataxic. Upon awakening from the surgery,ss were with no observable motoric signs. Movement of the eye muscles were the only elicited sign of movement. By the second post-surgical day,animals showed severe signs of ataxia. The degree of mobility gradually returned and improved over time,but all Ss remained permanently ataxic.

Subjects avoided human handling and had to be fed wet mesh food during the first post-operative week. They began to eat regular laboratory rat chow during the second post-operative week,but they were unable to retrive food from the conventional food bins, Ss showed signs of intentional tremor when approaching the water sprout. Intentional tremor was also evident when the Ss approached the lever in the operant chamber. These signs of ataxia corresponded to the reports by Carrea and Mettler (1947), and by Zervas (1967) which showed that interposed nucleus lesion resulted in devastating ataxia.

One of the subjects,no. 52 suffered asymmetrical lesion due to some technical difficulty. This S was also

permanently ataxic,though the behavioural signs were more pronounced to the right side of the body limbs.

B. activity measured by the rotational running wheel: Figure 24 shows the mean of the total number of rotations in the running wheel over sessions of 30 minutes. The results from the D-I group,D-I C group and the normal control group are presented. Based on this graph,it is obvious that the D-I lesion 'successfully' and permanently suppressed the running activity to a very low level. In addition,the behaviour showed no tendency to recover over time. When the measurement of rat no. 52 was deleted,the activity of the other 5 rats were shown to be nil.

C. performance on the DCA:

(1) Figure 25 is the mean of the total number of a) total lever presses, b) reinforced lever presses, c) penalty count, and d) trial abort count of the Ss over 29 daily sessions. For clarity the sessions were divided into six time blocks. As shown on the graph, there was general reduction in the total lever presses when the I/O control was in effect. There was also a slight reduction in the mean of the total reinforced lever presses. The mean of the total reinforced lever press was the highest during the 6th block,when the I/O control was removed post-operatively.

(2) The mean response latencies of the D-I group on the DCA and on the DCA with I/O control is presented in figure 26. As was the case with the previous groups, the mean response latencies of the system was a function of the level of demand of the reinforcement contingency. Post-operatively, when the behavioural system was severely ataxic, the mean response latencies of the system was again shown to be a function of the stringency in control demand. With subsequent removal of the I/O control, the mean response latencies of the system showed a corresponding decrease. The D-I nuclei did not produce any apparent disruption in the p performance of the behavioural system on the DCA either with or without the superimposed I/O control.

(3) $\overline{P}(0) - \overline{P}(T)$: The difference plot of the D-I is presented in figure 27. Again the main factor in this analysis is the controldemand

(4) P plot:

Figure 28 is the P value of the group over the 29 experimental sessions. Each data point is the mean of the 6 Ss" performance in the same time point. Again the behavioural system is shown to be servomechanismic-like in its response to the level of control demand as denoted by the reinforcement contengencies. Using the day of the surgery as the midpoint the response of the system before and after the surgery are shown to be a mirror image of each other. When the I/O control was in effect, the

response of the system was confined to a narrow range, showing little day-to-day variations (reduced phase velocity). When the I/O control was removed, the behavioural system showed a corresponding increase in its relative error as well as in its rate of change in its error.

(5) the phase-plane representation ($D\bar{P}/DT$ vs $\bar{P}$):

Figure 32 is the phase-diagram of the adjustment of the DCA to the superimposed I/O control before the system underwent lesioning to the D-I nuclei. The system showed tendency towards a limit cycle which exhibited a equilibrium force between the processes of extinction and of reconditioning. This process of a balanced equilibrium between forces of extinction and of reconditioning continued post-operatively. As shown in figure 33, the system spiraled around the P axis. However the system diverged from the limit cycle towards a larger relative error in its performance when the I/O control was subsequently removed. When the performance of the system on the DCA is evaluated as a continucus process in the time dormain, the adjustment could be seen as approaching asympotically towards a limit cycle which spiraled around theh P axis. The behavioural system then returned to the previous level of performance, with predominately the force of reconditioning at work, when the I/O control was removed. The lesion to the controlled system again did not cause any apparent disruption to the systen under study.

(6) Figure 34 is the graphic representation of the D-I during extinction phase of the experiment. The ss showed a response pattern which was similar to the D-I control and normal control ss.

Figure 24. Mean of total number of rotations in running wheel activity. Graph shows the activity level of the D-I,D-I C and N groups. Each data point is an average of all Ss in the group. Bilateral electrlytic lesion in D-I nuclei reduced activity to almost nil. D-I nuclei sham lesion showed opposite effect.

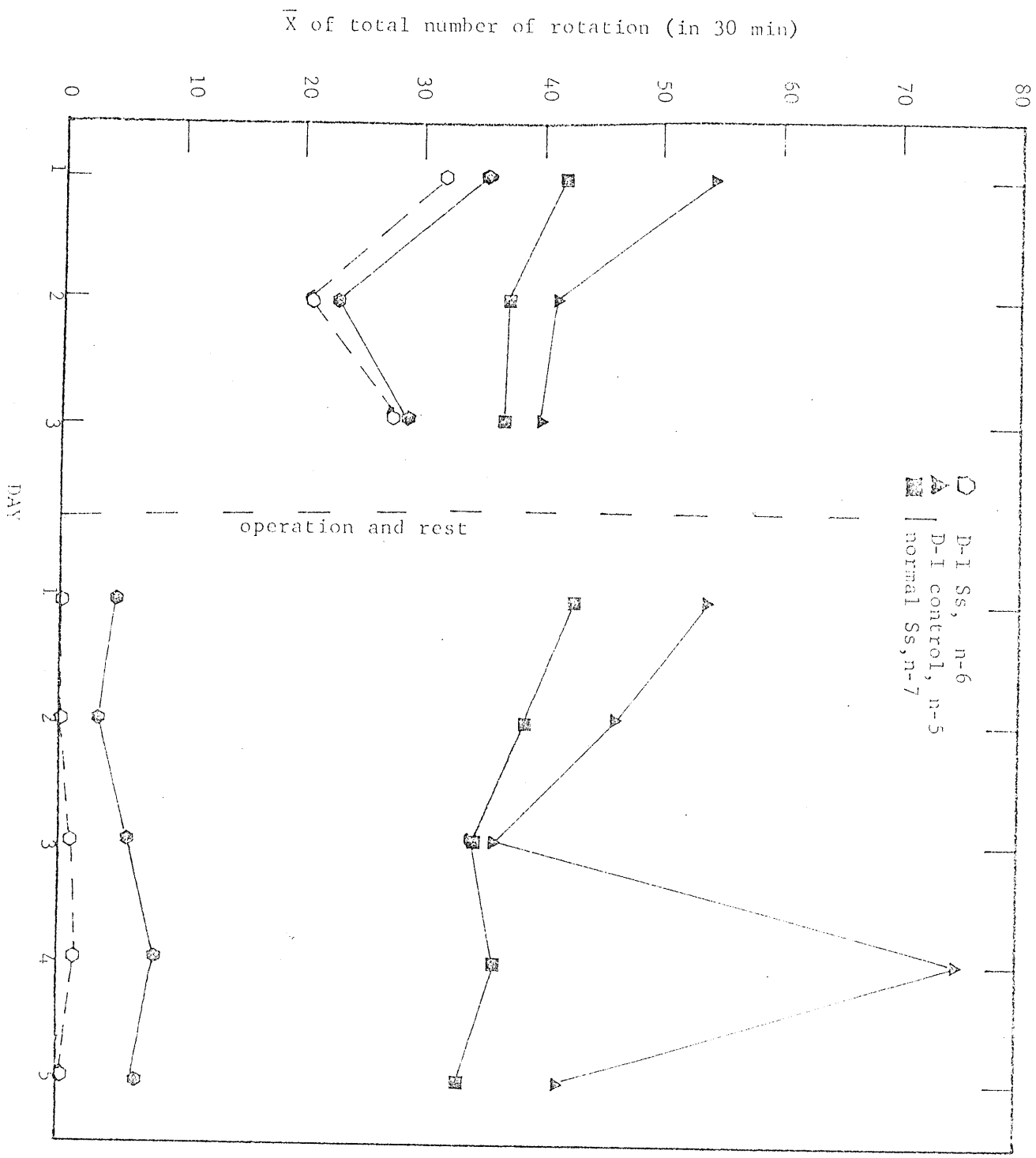

$\bar{X}$ of total number of rotation (in 30 min)
0
10
20
30
40
50
60
70
80
1
2
3
operation and rest
1
2
3
4
5
DAY
D-I Ss, n-6
D-I control, n-5
normal Ss, n-7

Figure 25. The mean of a) total lever b) reinforced lever press c) penalty count and d) trial abort count for every daily session. Each data point is a mean of all Ss in D-I group. 29 daily sessions grouped into six time blocks.

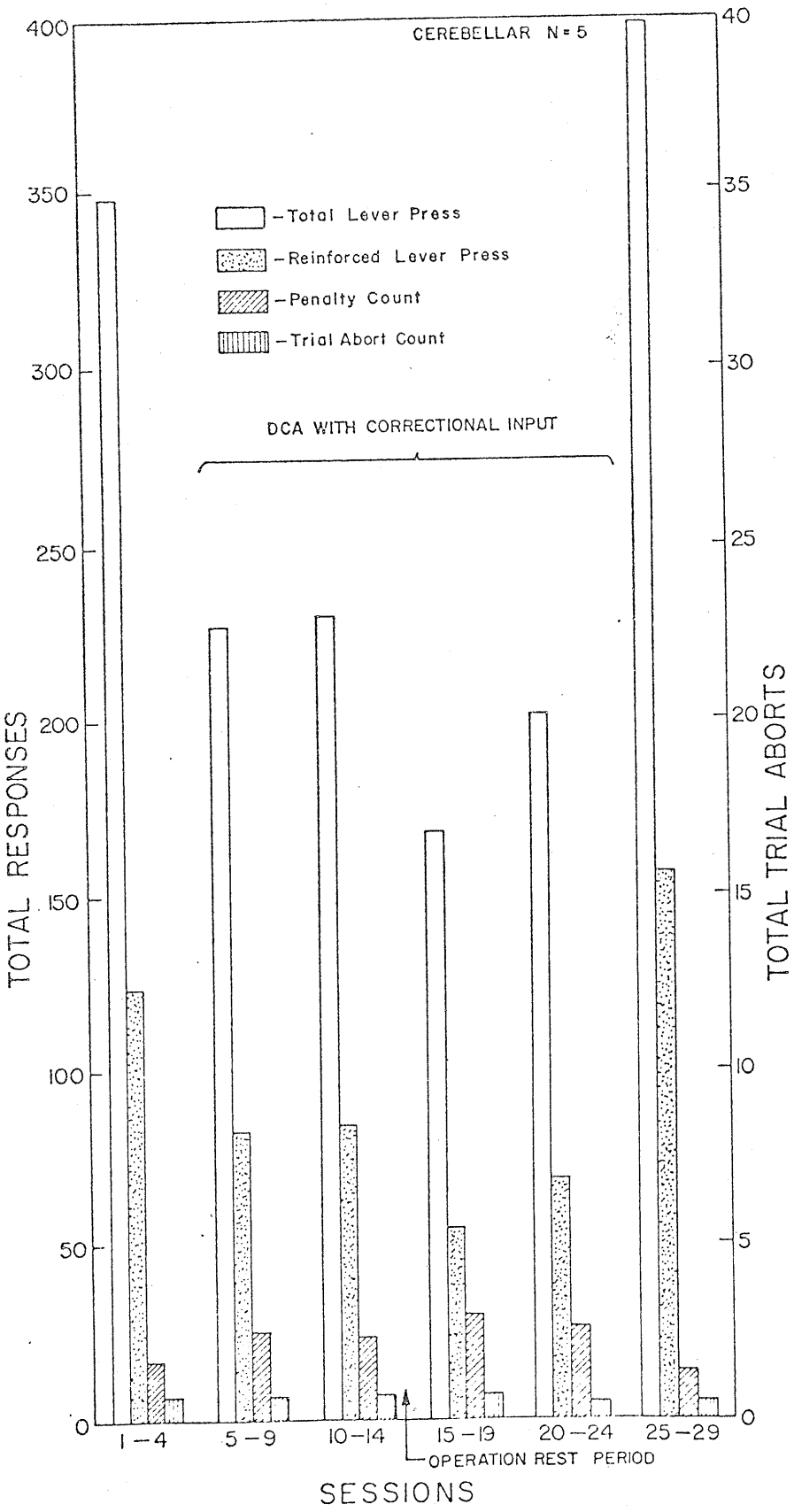

CEREBELLAR N=5
Total Lever Press
Reinforced Lever Press
Penalty Count
Trial Abort Count
DCA WITH CORRECTIONAL INPUT
TOTAL RESPONSES
TOTAL TRIAL ABORTS
400
350
300
250
200
150
100
50
0
40
35
30
25
20
15
10
5
0
1–4
5–9
10–14
15–19
20–24
25–29
OPERATION REST PERIOD
SESSIONS

Figure 26. The mean response latencies of D-I lesioned system before and after surgery. Data presented as response latencies over 29 daily sessions and as continuous time function both suggested that lesion did not produce observable changes in the performance on DCA.

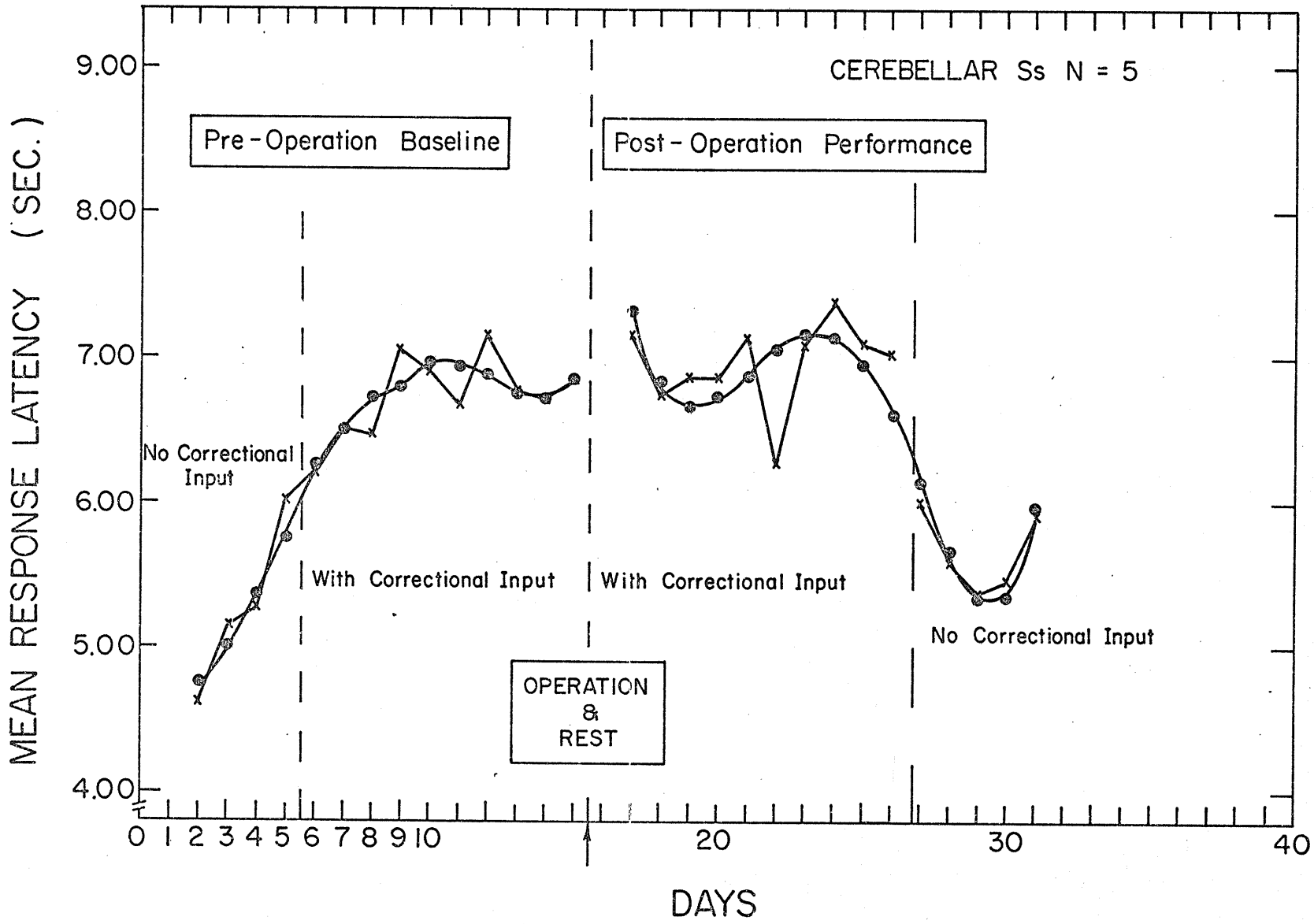

CEREBELLAR Ss N = 5
Pre-Operation Baseline
Post-Operation Performance
MEAN RESPONSE LATENCY (SEC.)
9.00
8.00
7.00
6.00
5.00
4.00
No Correctional Input
With Correctional Input
With Correctional Input
No Correctional Input
OPERATION & REST
0 1 2 3 4 5 6 7 8 9 10
20
30
40
DAYS

Figure 27. The difference plot $\overline{P}(0)-\overline{P}(T)$ of the D-I lesioned group. Magnitudes of difference was a function of the reinforcement contingencies.

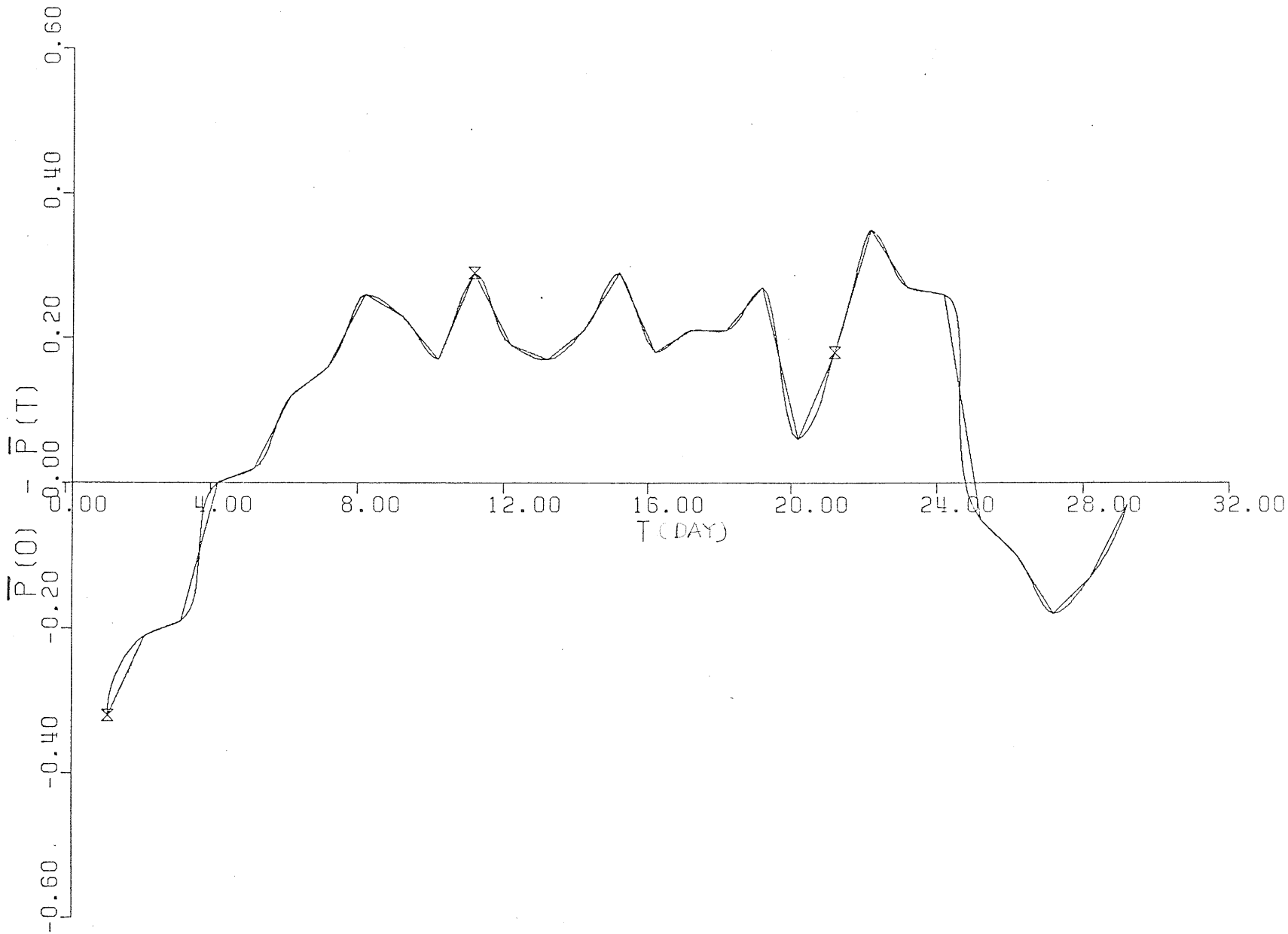

0.60
0.40
0.20
0.00
-0.20
-0.40
-0.60
$\overline{P}(0) - \overline{P}(T)$
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T (DAY)

Figure 28. $\bar{P}$ plot of D-I system on DCA. 29 daily sessions were treated as continuous time function. I/O control reduced relative error term of the system. Lesion produced no observable effect on the performance of the system on the DCA.

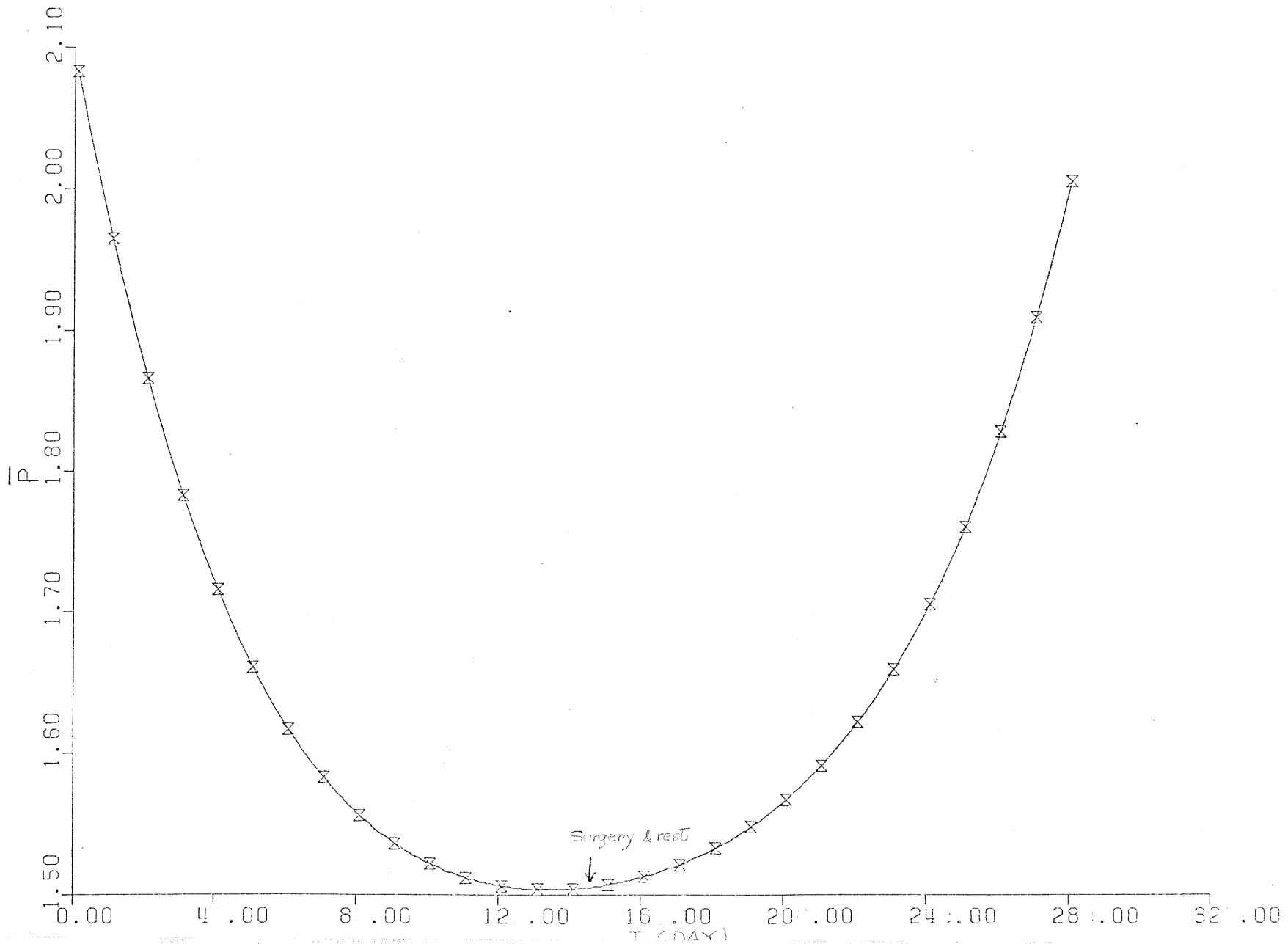

$\overline{P}$
2.10
2.00
1.90
1.80
1.70
1.60
1.50
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T (DAY)
Surgery & rest

Figure 29. $\bar{P}$ plot of the D-I lesioned system prior to surgery. Performance of system showed the noninvariance of governing equations when the system was functioning at different levels of control demand.

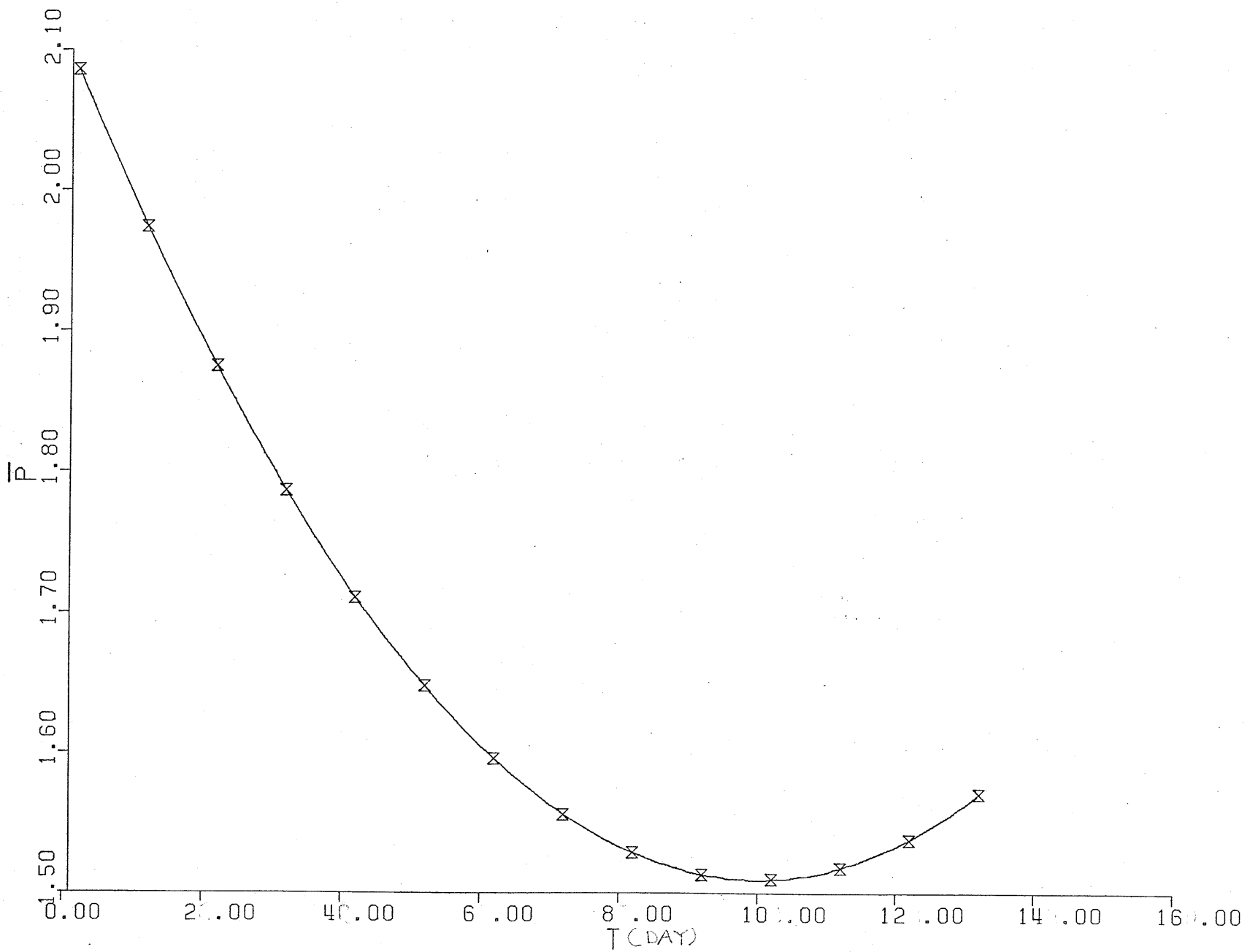

2.10
2.00
1.90
1.80
1.70
1.60
1.50
$\overline{P}$
0.00
2 .00
4 .00
6 .00
8 .00
10 .00
12 .00
14 .00
16 .00
T (DAY)

Figure 30. $\overline{P}$ plot of D-I system after lesion. Performance on the DCA and DCA with I/O control showed characteristics essentially identical to that of the normal.

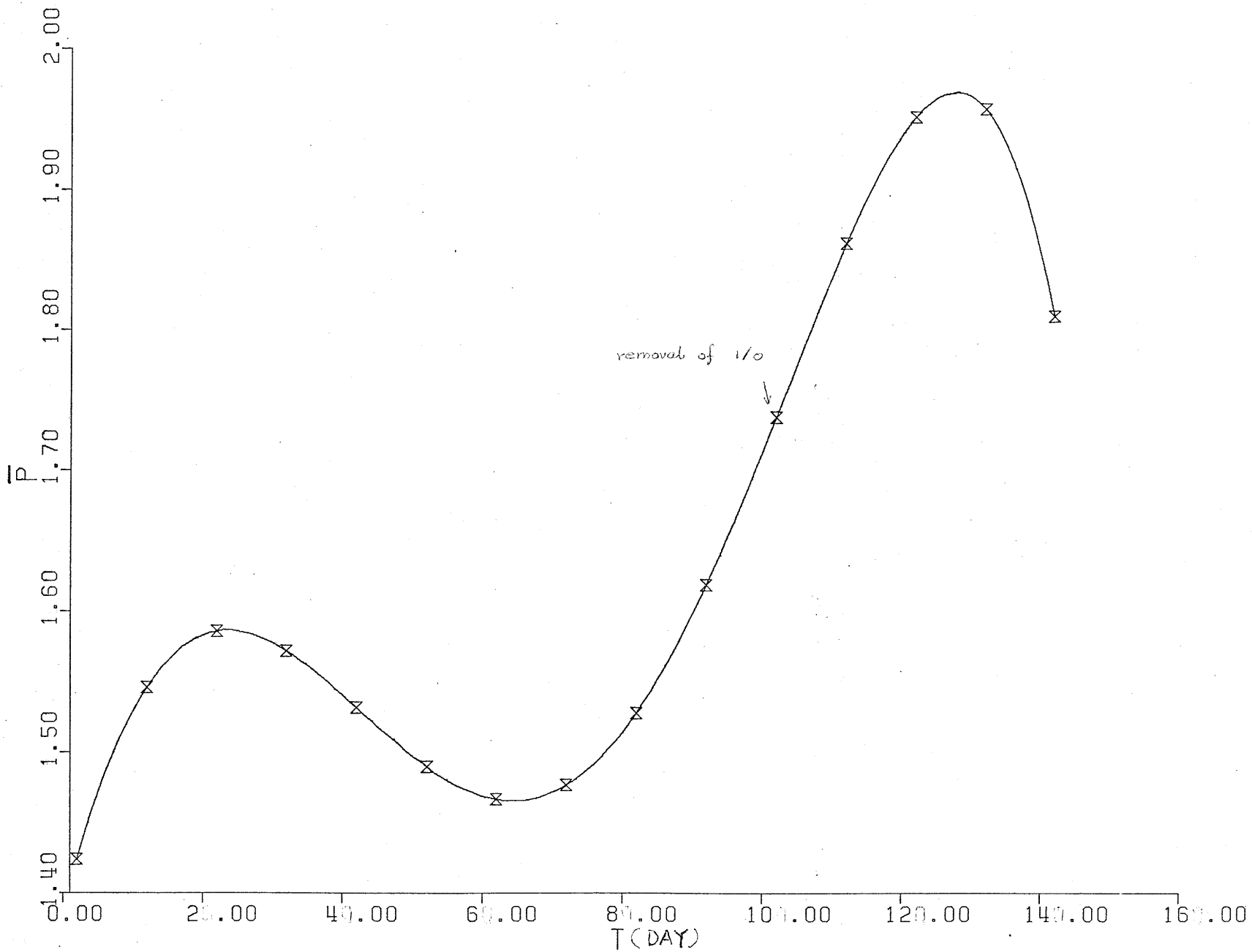

2.00
1.90
1.80
1.70
1.60
1.50
1.40
$\bar{P}$
removal of I/O
0.00
40.00
120.00
T(DAY)

Figure 31. Phase plane representation of D-I lesioned system. All data points were evaluated as continuous time function. I/O control drove system to perform with reduced P and reduced dP/dT.

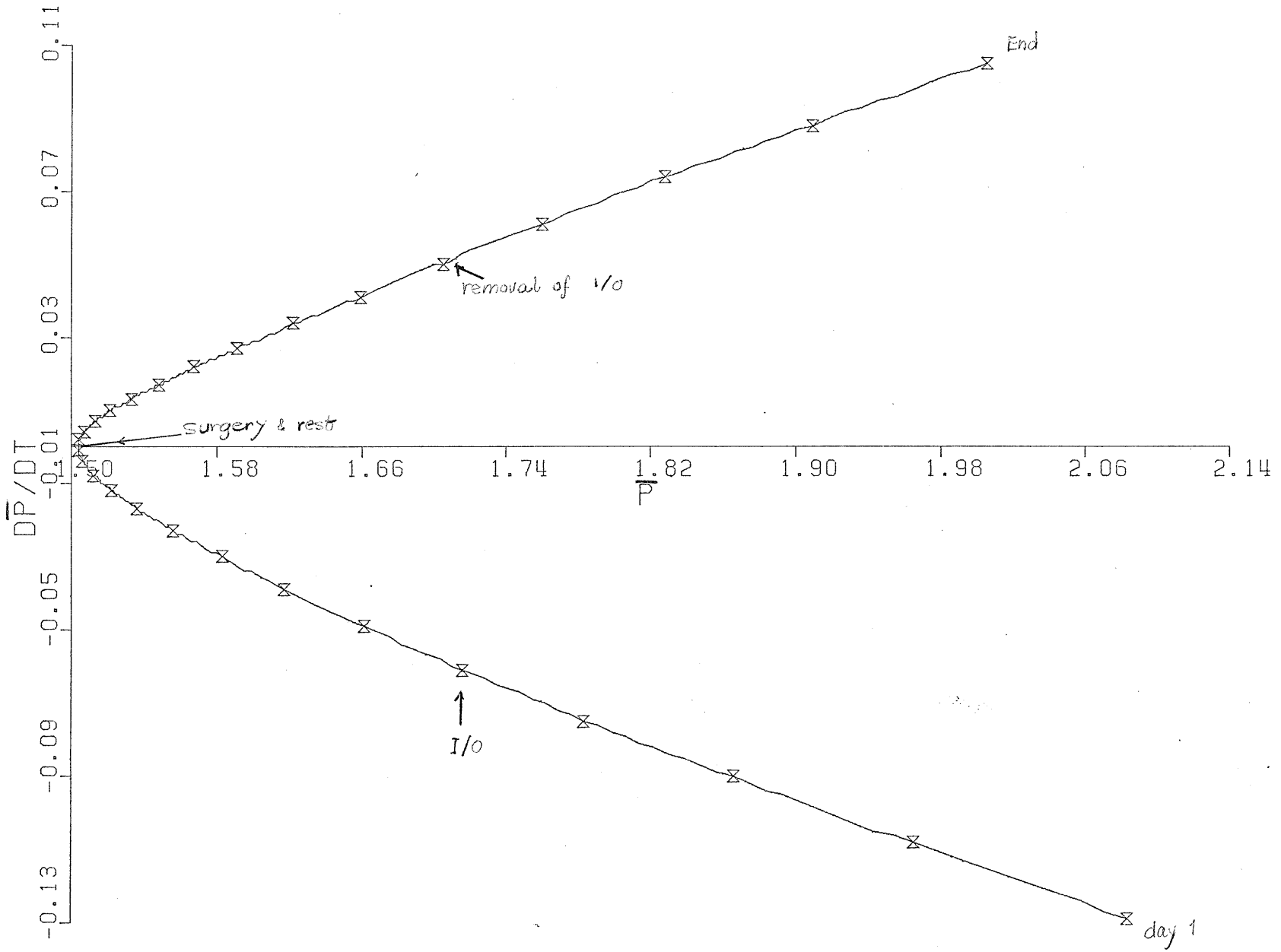

0.11
0.07
0.03
-0.01
-0.05
-0.09
-0.13
DP/DT
1.50
1.58
1.66
1.74
1.82
1.90
1.98
2.06
2.14
P
End
removal of I/O
surgery & rest
I/O
day 1

Figure 32. Phase diagram of D-I system prior to surgery. System showed servomechanism-like adjustment to changes in schedule of demand. While on I/O control, system tended to approach limit cycle around P axis.

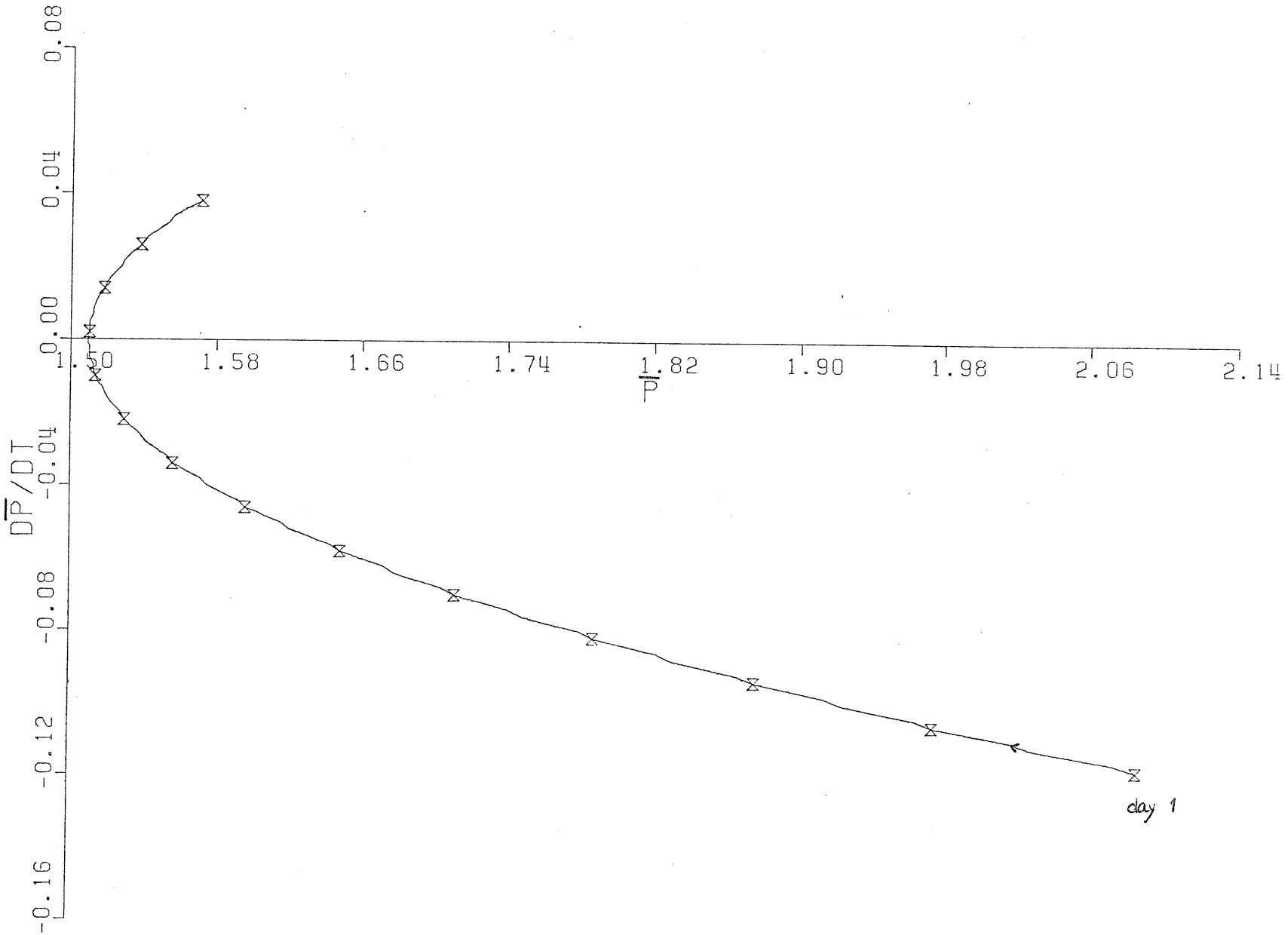

0.08
0.04
0.00
-0.04
-0.08
-0.12
-0.16
DP̄/DT
1.50
1.58
1.66
1.74
1.82
1.90
1.98
2.06
2.14
P̄
day 1

Figure 33. Phase diagram of D-I system post-operatively. System continued behabioral functioning as a limit cycle while on I/O control. Behavioral trajectory changed its $\overline{P}$ and $d\overline{P}/dT$ when returned to base line DCA.

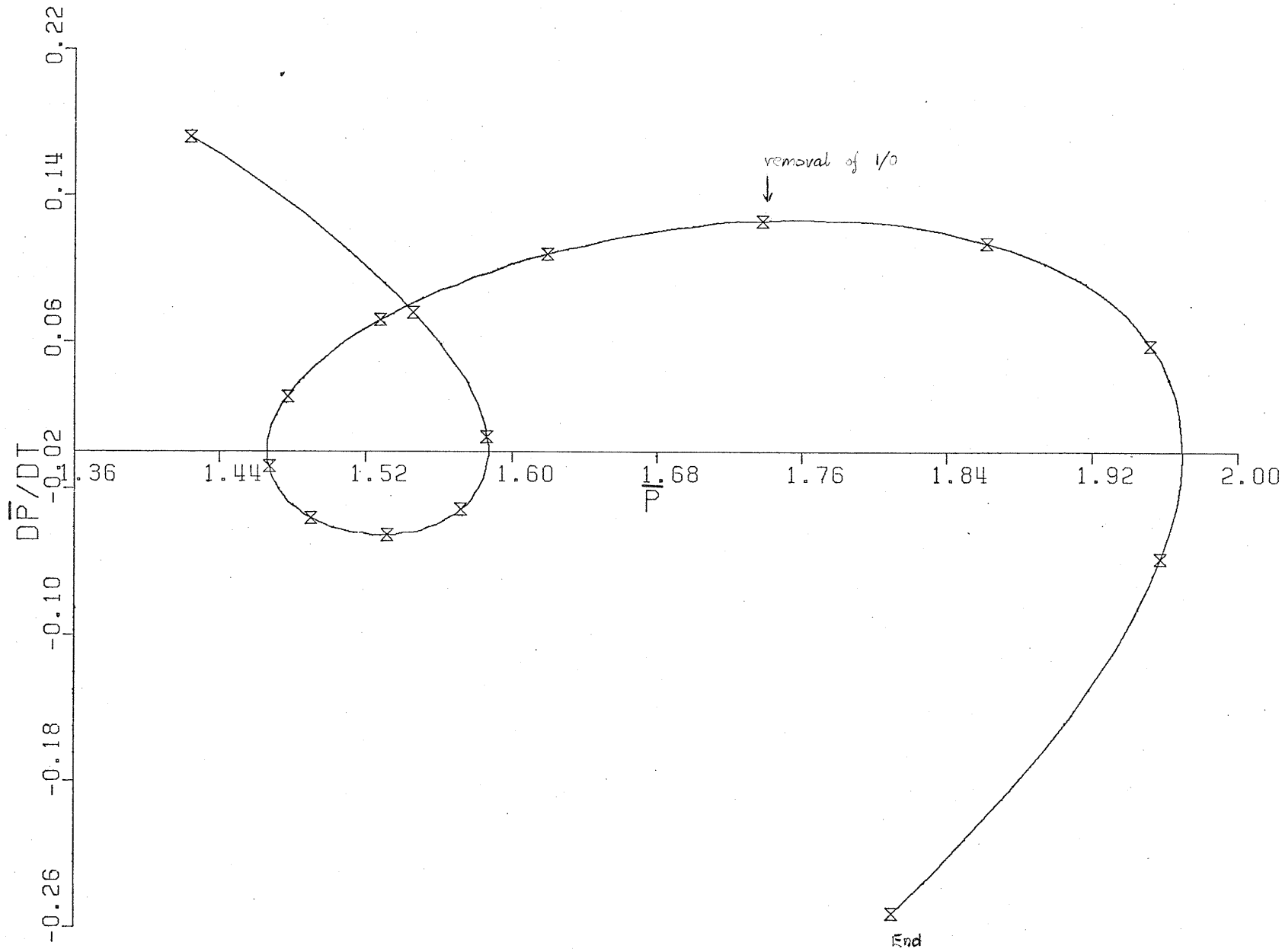
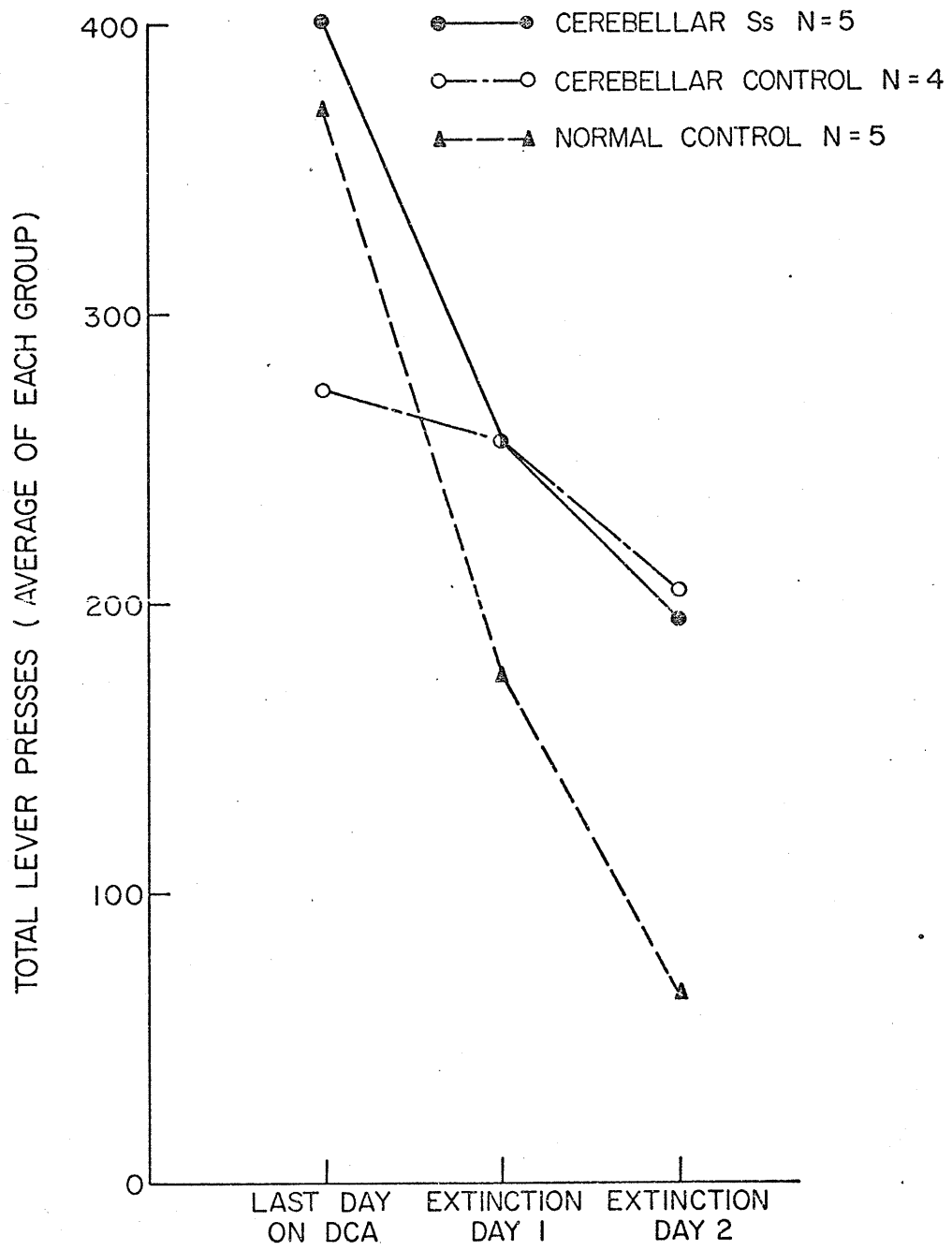

0.22
0.14
0.06
-0.02
-0.10
-0.18
-0.26
$\overline{DP}/DT$
1.36
1.44
1.52
1.60
1.68
1.76
1.84
1.92
2.00
$\overline{P}$
removal of I/O
End

Figure 34. Extinction curves of D-I ,D-I c, and N groups. Mean of total lever presses per session was used as behavioural index. All groups showed same type of curve.


CEREBELLAR Ss N=5
CEREBELLAR CONTROL N=4
NORMAL CONTROL N=5
TOTAL LEVER PRESSES (AVERAGE OF EACH GROUP)
400
300
200
100
0
LAST DAY ON DCA
EXTINCTION DAY 1
EXTINCTION DAY 2

GROUP IV the group with sham lesion in the dentato interposed nuclei of the cerebellum (D-I C)

A. general observation:

Ss in this group showed essentially the same general pattern of behaviour before and after the sham lesion. There was slight reduction in activity during the first post-operative week. Like all animals, they sowed signs of irritation to the autoclips on the head. Overall, the behavioural level of the Ss returned to normal by the second post-operative week.

B. activity level as measured by the rotational running wheel: was essentially the same before and after the sham lesion. Other than for the 4th post-operative testing session when the mean response level showed a sudden increase (caused by the oscillatory behaviour pattern of one S), there was considerable overlap in the activity level of the Ss before and after the sham lesion.

C. performance on the DCA:

(1) Figure 35 is the mean of the total number of a) total lever presses, b) reinforced lever presses, c) penalty count, and d) trial abort count of the 29 daily sessions. There was a reduction in the total number of the lever presses during the two post-operative time blocks without any corresponding changes in the total

number of reinforced lever presses. This improved efficiency in the behaviour on the DCA corresponded to an increased response latencies during the same time period in figure 36. This improvement was not accompanied by the development of a motoric chain post-operatively.

(2)Figure 36 is the mean response latencies of the D-I C group before and after the sham lesion. The performance of the system showed steady improvement,as driven ib the I/O control. The process continued post-surgically, exhibiting a behavicural system whose relative error and phase velccity were kept within a narrow range. With the removal of the I/O control,the response latencies returned to a level ccmparable to that of the pre-operative,pre-I/O contrcl baseline.

(3) P(0)-P(T): The difference plot of the D-I C group is presented in figure 37. The graph showed that the 'gain' of the functioning system was the greatest during the ten consecutive time points which corresponded to the ten post-surgical days when the Ss were on the I/O control.

(4) P plot:

When the response of the system is cast into time domain (figures 38,39 & 40), the P's gave the ratio of behavioural adjustment to that demanded by the change in the reinforcement contingencies. In this case,the P's were ccnstricted tc a narrow range by the superimposed

I/O control. Behavioral adjustment was also present when the demand was returned to the baseline level.

(5) $D\overline{P}/DI$ vs $\overline{P}$:

Pre-operatively the phase plane diagram of the performance on the DCA showed that the system moved toward a semi-stable focus around the P axis when the I/O was in iffect (figure 42). The same trend was in effect post-operatively(figures 41 &43) when the I/O was applied. When the I/O was removed the behavioural system deviated from the previously established active equilibrium state,suggesting the noninvariance of the governing equation in control policy.

Figure 35. Means of a) total lever press, b) reinforced lever press, c) penalty count, and d) trial abort count of Ss in the D-I sham lesioned group. Values of a) and b) seen as a function of values in reinforcement contingencies on DCA.

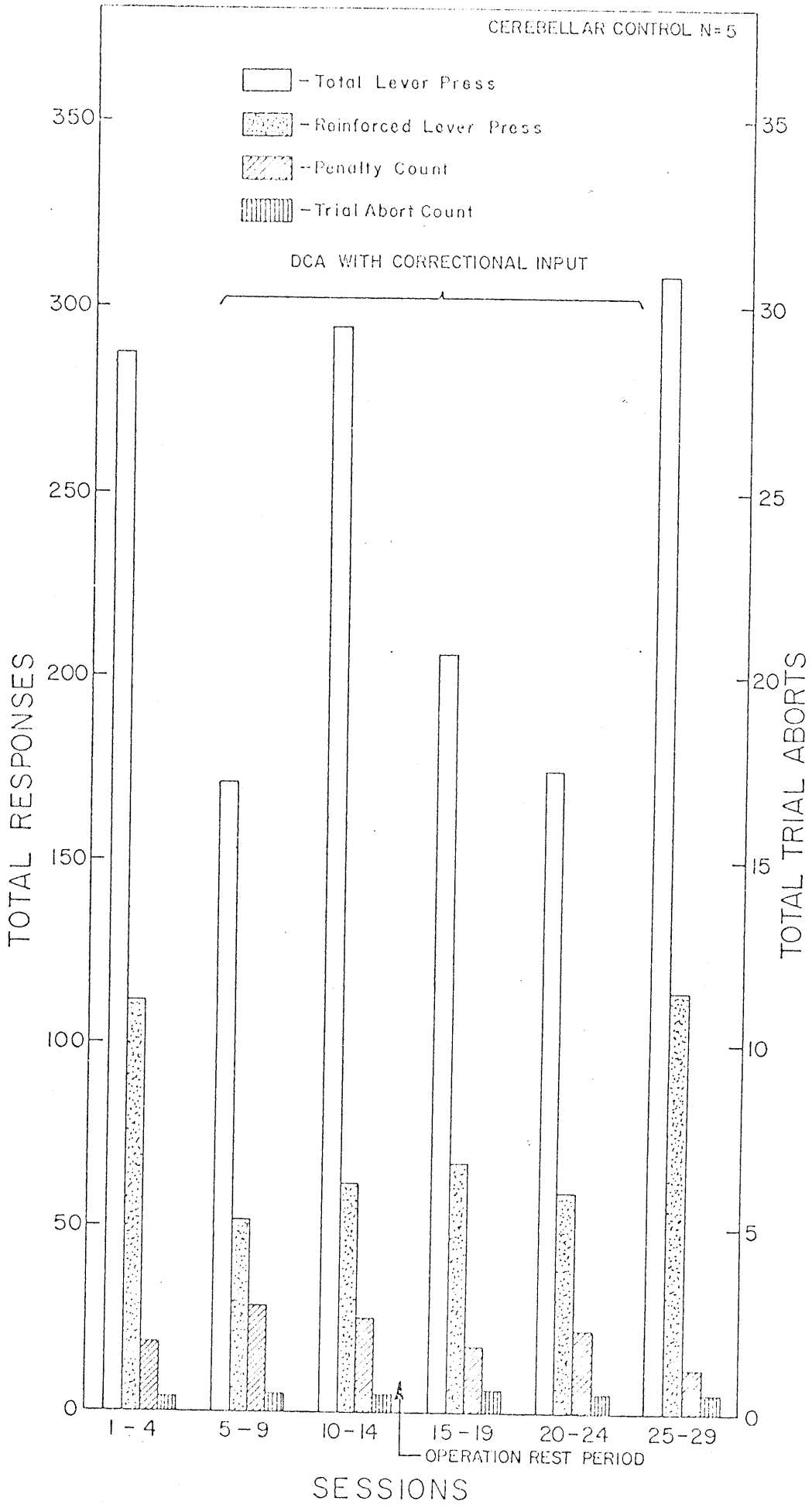

CEREBELLAR CONTROL N=5
- Total Lever Press
- Reinforced Lever Press
- Penalty Count
- Trial Abort Count
DCA WITH CORRECTIONAL INPUT
TOTAL RESPONSES
TOTAL TRIAL ABORTS
0
50
100
150
200
250
300
350
0
5
10
15
20
25
30
35
1 - 4
5 - 9
10 - 14
15 - 19
20 - 24
25 - 29
OPERATION REST PERIOD
SESSIONS

Figure 36. The mean response latencies of D-I sham lesioned group. Behavioral system resembles that of optimally contrclly process, adjusting performance level relative to levels cf demand. Behavioral efficiency improved over practice and post-operatively.

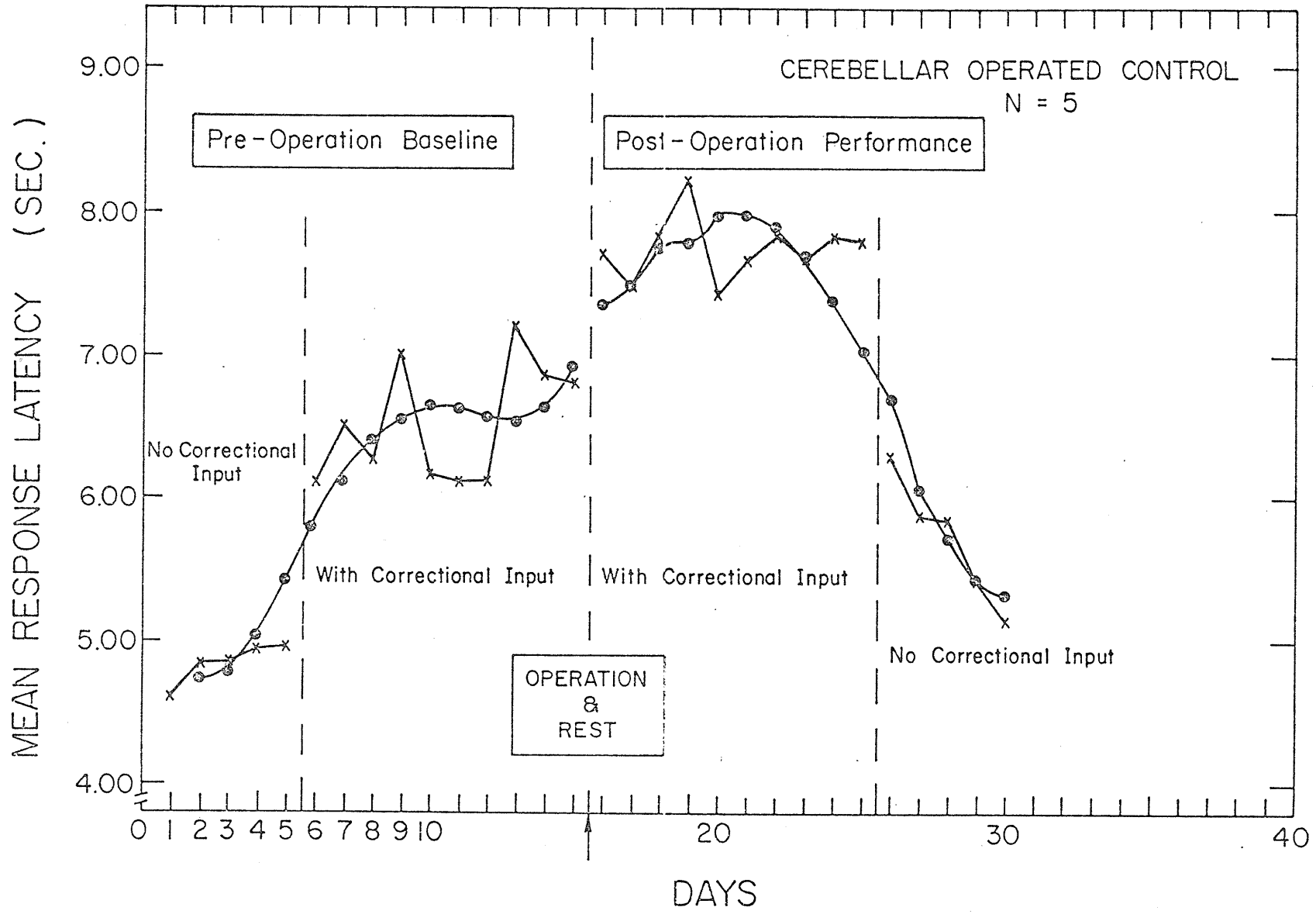

CEREBELLAR OPERATED CONTROL
N = 5
Pre-Operation Baseline
Post-Operation Performance
MEAN RESPONSE LATENCY (SEC.)
9.00
8.00
7.00
6.00
5.00
4.00
No Correctional Input
With Correctional Input
With Correctional Input
No Correctional Input
OPERATION & REST
0 1 2 3 4 5 6 7 8 9 10
20
30
40
DAYS

Figure 37. P(0)-P(T) plot of D-I c group. Difference between daily sessions and the pre-I/O control baseline is a function of level of demand of DCA.

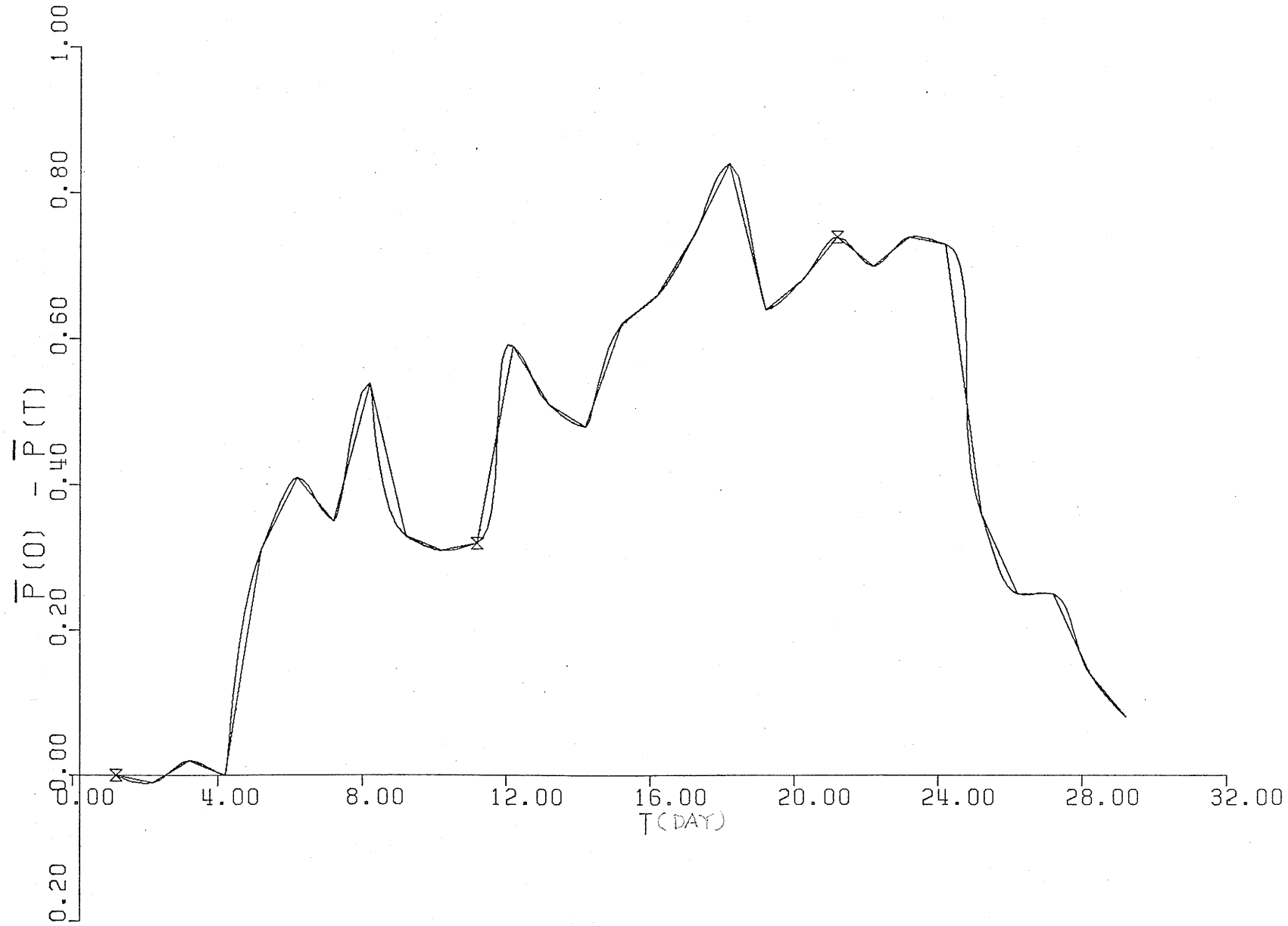

1.00
0.80
0.60
0.40
0.20
0.00
-0.20
$\overline{P}(0) - \overline{P}(T)$
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T(DAY)

Figure 38. P plct of D-I C cn DCA. Relative error computed relative tc 13" of delay on I/O ccntrol. Performance showed continual imprcvement post-operatively while system on DCA with I/C ccntrol. System returned to baseline perfcrmance when I/O ccntrol removed.

0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
1.20
1.40
1.60
1.80
2.00
2.20
2.40
P

Figure 39 P plot of D-I C group before sham lesion.

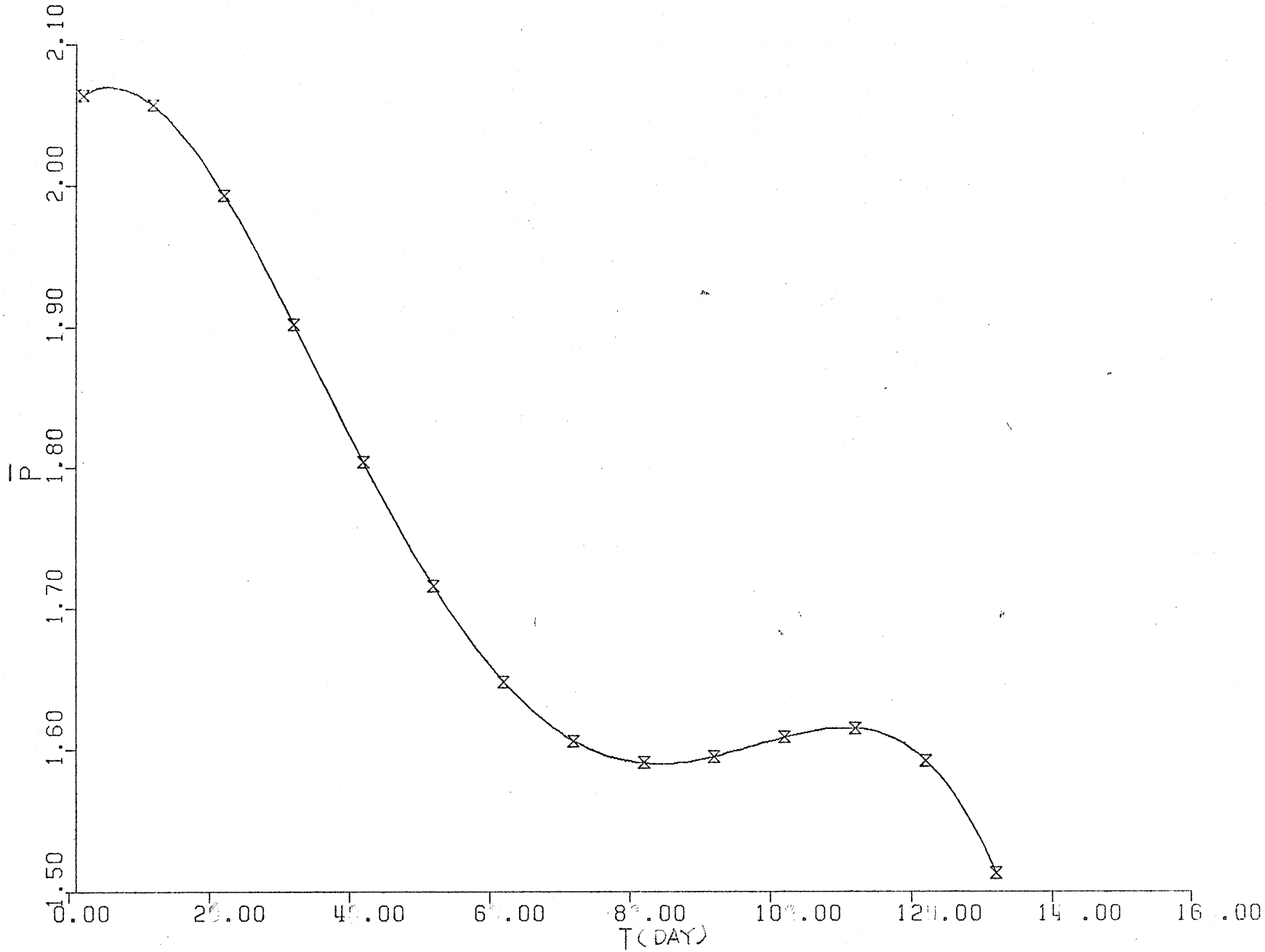

2.10
2.00
1.90
1.80
1.70
1.60
1.50
P
0.00
T(DAY)

Figure 40 P plot of D-I C group after sham lesion.

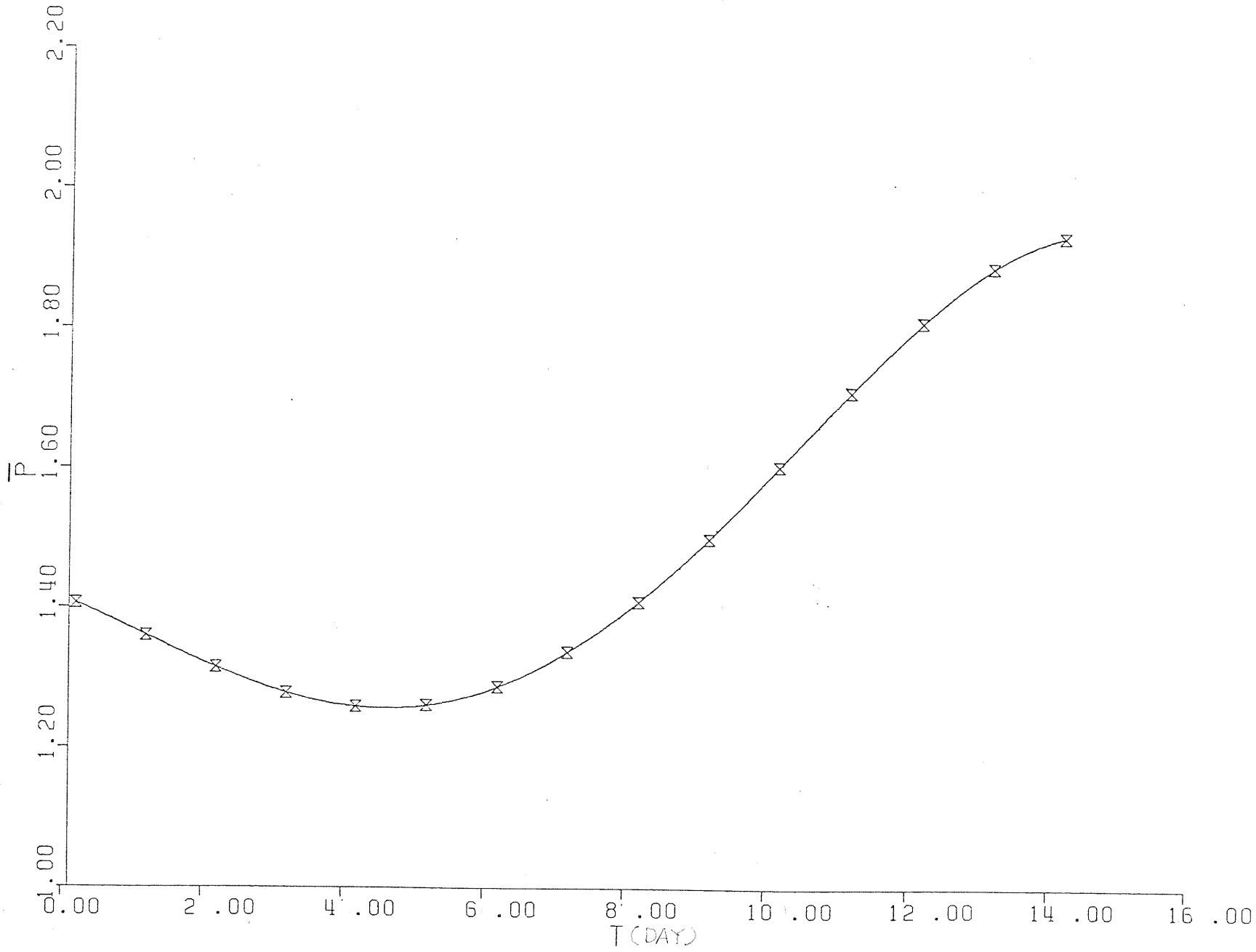

$\overline{P}$
1.00
1.20
1.40
1.60
1.80
2.00
2.20
0.00
2.00
4.00
6.00
8.00
10.00
12.00
14.00
16.00
T (DAY)

Figure 41. Phase representation of D-I C group. 29 daily sessions treated as continuous function in time domain. System response suggested servomechanism-like behaviour on DCA and DCA with I/O control.

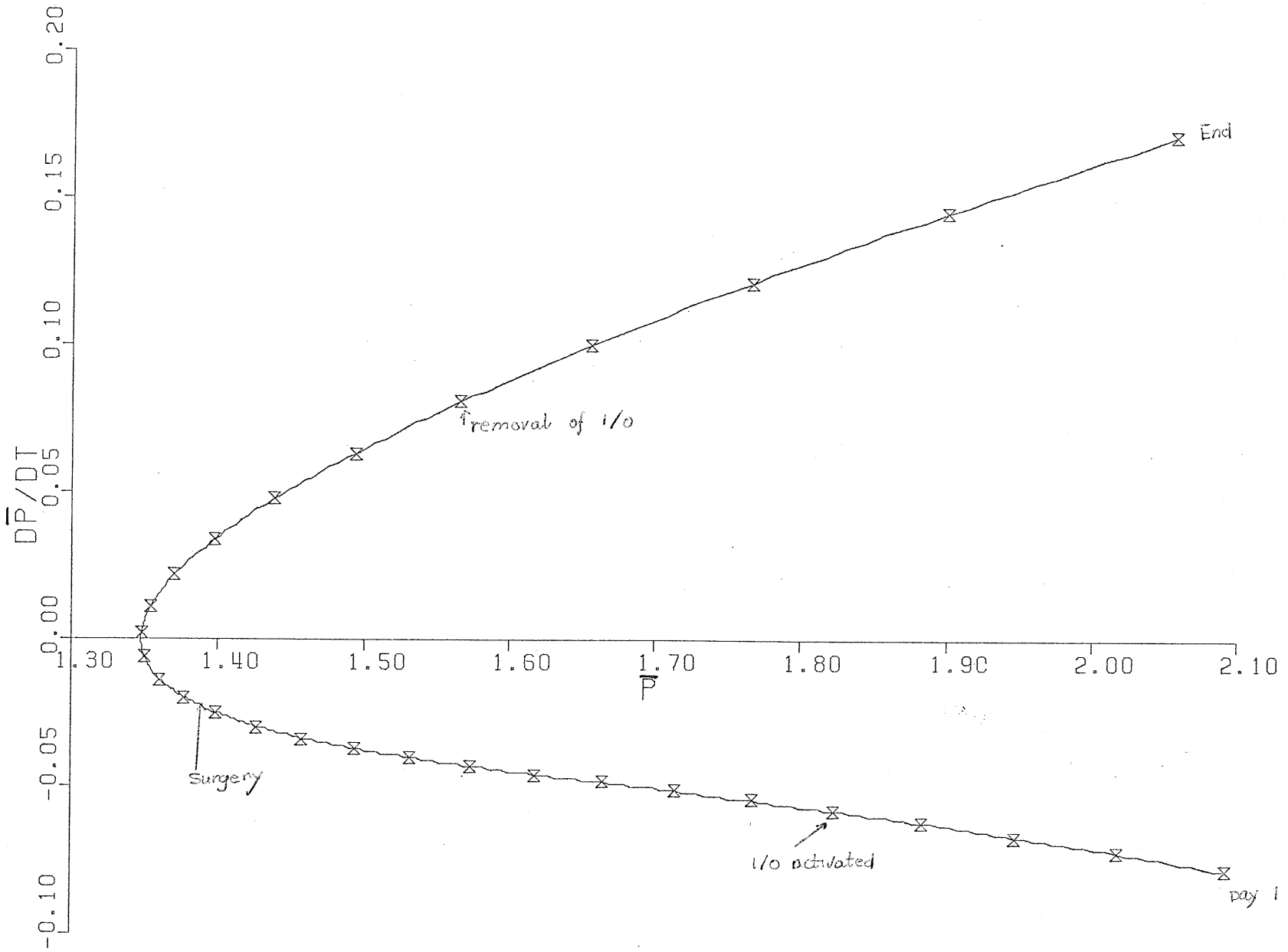

0.20
0.15
0.10
0.05
0.00
-0.05
-0.10
$\overline{DP}/DT$
1.30
1.40
1.50
1.60
1.70
1.80
1.90
2.00
2.10
$\overline{P}$
End
removal of i/o
Surgery
i/o activated
pay 1

Figure 42. Phase diagram of D-I C system prior to sham lesion. System was 'driven' to function towards a semi-stable focus by the superimposed I/O control, with reduction in $\overline{P}$'s and in dP/dT.

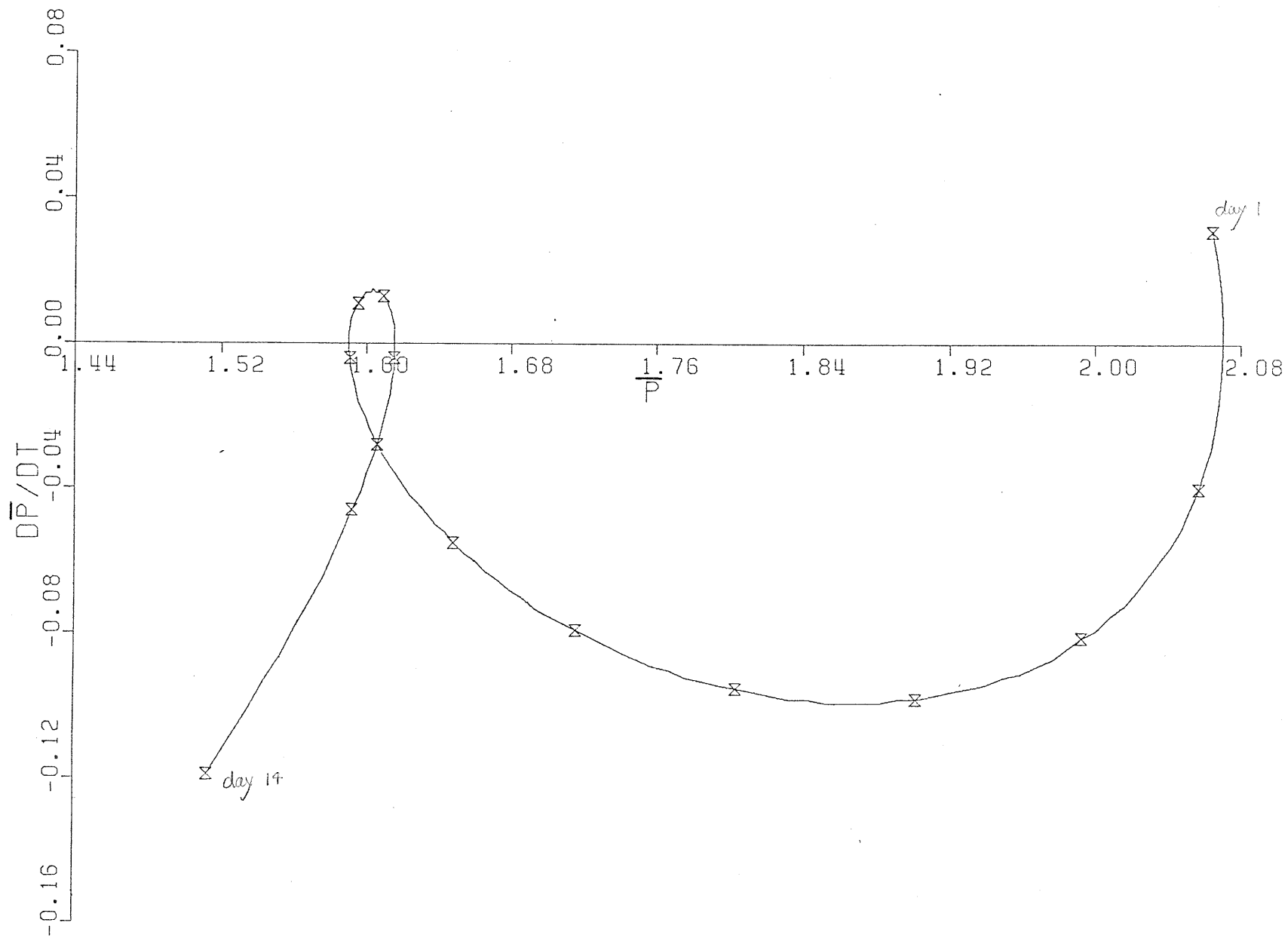
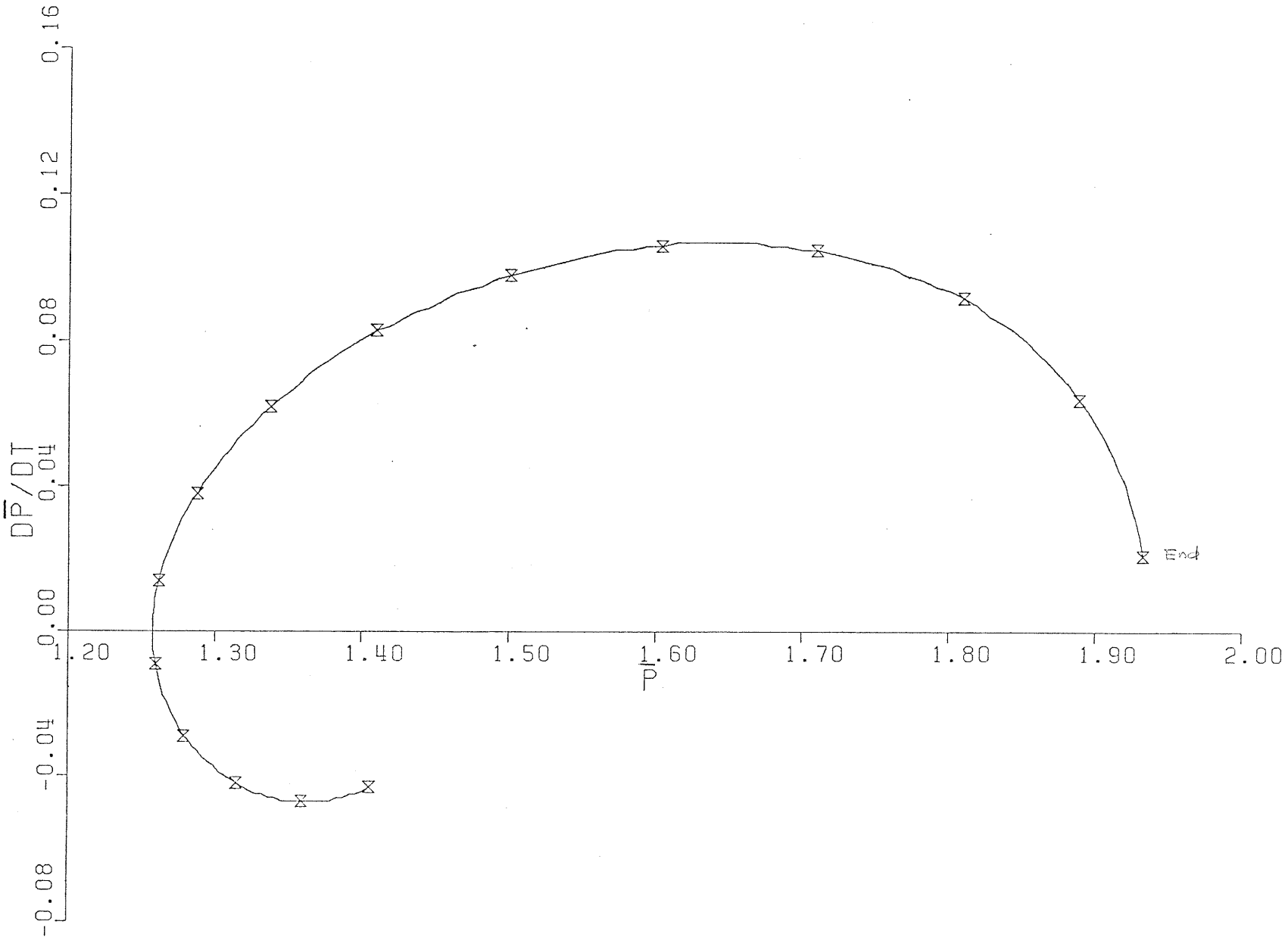

0.08
0.04
0.00
-0.04
-0.08
-0.12
-0.16
DP̄/DT
1.44
1.52
1.60
1.68
1.76
1.84
1.92
2.00
2.08
P̄
day 1
day 14

Figure 43. Phase diagram of D-I C system after sham lesion. System showed same mode of behaviour on DCA with I/O control :limit cycle. System readjusted to baseline DCA when I/O ccntrol was removed


0.16
0.12
0.08
0.04
0.00
-0.04
-0.08
DP/DT
1.20
1.30
1.40
1.50
1.60
1.70
1.80
1.90
2.00
P
End

GROUP V the group with bilateral electrolytic lesion in the frontal pole (Ft)

A.General observation:

ss in this group exhibited similar general pattern of behaviour prior to and subsequent to the lesioning. Animals showed no specific signs of hyperre- activity to human handling. Unlike the Ss in the H CD group, they did not show strong startle reaction when a puff of air was blown into the cage.

B. activity in the rotational running wheel:

Figure 44 shows that, as compared to the pre-operative level and the activity level of the sham lesioned grcup,the frontal Ss showed an increase in the first three post- lesioned testing sessions. The activity level returned to the same general level by the 4th post-lesioned testing sessicn.The effect of 2.5 mg/kg of d-amphetamine cn the frcntal animals were studied as a pharmacological 'verificaticn' cf the lesion site. The effect of the drug was measured in the activity running wheel at 10 minute interval fcr 100 minutes after ip injecticn of the drug. As shown in figure 45, the frontal Ss displayed the double peak effect of the drug,which is unique to the frontal lesioned animals (Iversen,1971,Lynch,1969).

C.performance on the DCA:

(1) Figure 46 is the mean of a) total lever press b) reinforced lever press, c) penalty count and d) trial abort count of the 29 sessions which were grouped into six time blocks. Reinforced lever press was related to the schedule on the DCA. Trial abort count remained at the same level throughout the six time blocks. Penalty count was the highest during the second time block when the I/O was first introduced. It remained at the same level in the three subsequent time blocks.

(2) Figure 47 is the mean response latencies of the Ft Ss before and after the lesion. Pre-operatively the performance of the system showed steady improvement as a result of practice and was further driven to approximate the demand imposed on it via the I/O control. Post-operatively, the performance of the system showed a systematic shift towards that of a shorter latencies on the DCA. The mean response latencies returned to the baseline level as shown in block one, when the I/O was removed.

(3) The graphs representing the P(0)-P(T) and the P vs T (figures 48 &49) verified the same trend in the response of the 'lesioned' system to changes in the control policy.

(4) P plot:

Pre-operatively the response of the system in the time dcmain is similar to the systems described in previous sections. The relative error of the system was reduced over time as a function of the I/O control and of practice(figure 50). Post-cperatively,there was an increase in the relative error when compared to the pre-operative level. However, the general forms of the trajectcries were still mirror images of each other Figure 51 is the P plot of the Ft system before the lesion. The I/C control effectively constricted relative error of the system to a narrow range. Post-operatively the response of the system showed a tendency to oscillate towards shorter latencies (figure 52). However this tendency was held under check by the I/O control. With the removal of the I/O control the behavioural system exhibited a shift toward the pre-operative pre-I/O base-line level.

(5) $D\overline{P}/DT$ vs $\overline{P}$:

Pre-operatively the system moved toward an unstable focus and then diverged toward progressive legthening of the response latencies,showing the predominating force of extinction during this phase of the experiment (figure 53). Post-operatively, the system showed a slight tendency to overshoct,a characteri- stic of a slightly underdamped system (figure 54). But the I/O control quickly restricted the error and the phase velocity of the trajectory of the system was precipitated into a

limit cycle around the P axis. Upon the removal of the I/O control the system readjusted its response characteristic to the baseline level of demand.

(6) Figure 55 is the extinction data on the Ft, FT C and normal ss. As with the previously cited groups, the Ft system showed the same pattern of response reduction over the two sessions.

Figure 44. Activity level of frontal lesioned,frontal sham lesioned and normal groups measured by rotational running wheel. Data points represented mean of all Ss in each group. 3 pre-operatvie days and 5 post-operative days were presented. Frontal group showed temporary increase in activity count post-operatively while sham lesioned Ss showed temporary decrease in same time period.

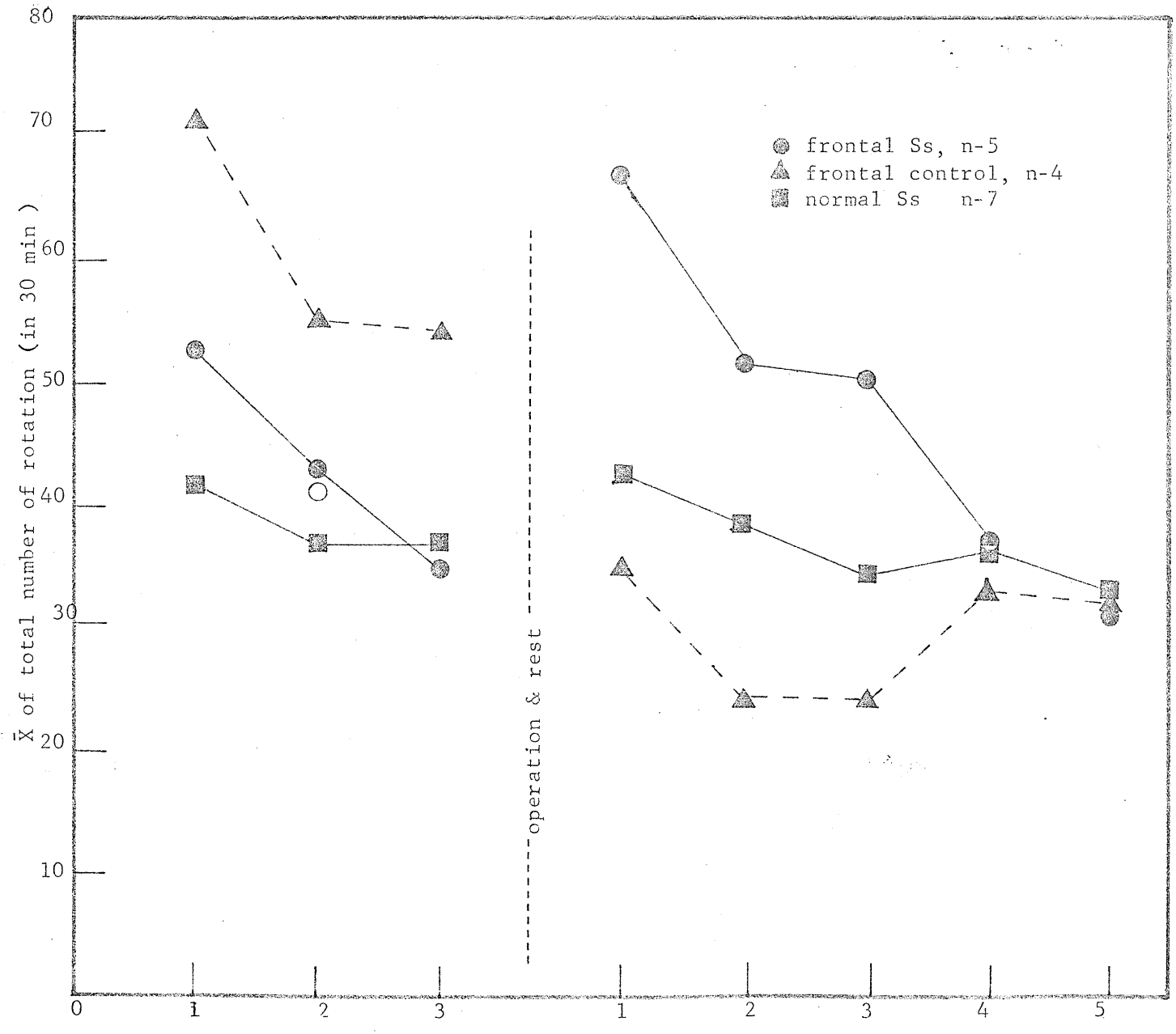

80
70
60
50
40
30
20
10
X̄ of total number of rotation (in 30 min)
frontal Ss, n-5
frontal control, n-4
normal Ss n-7
operation & rest
0
1
2
3
1
2
3
4
5

Figure 45. Means of total number of a) total level press, b) reinforced lever press, c) penalty count and d) trial abort count in each daily session. 29 daily sessions were grouped into six time blocks.

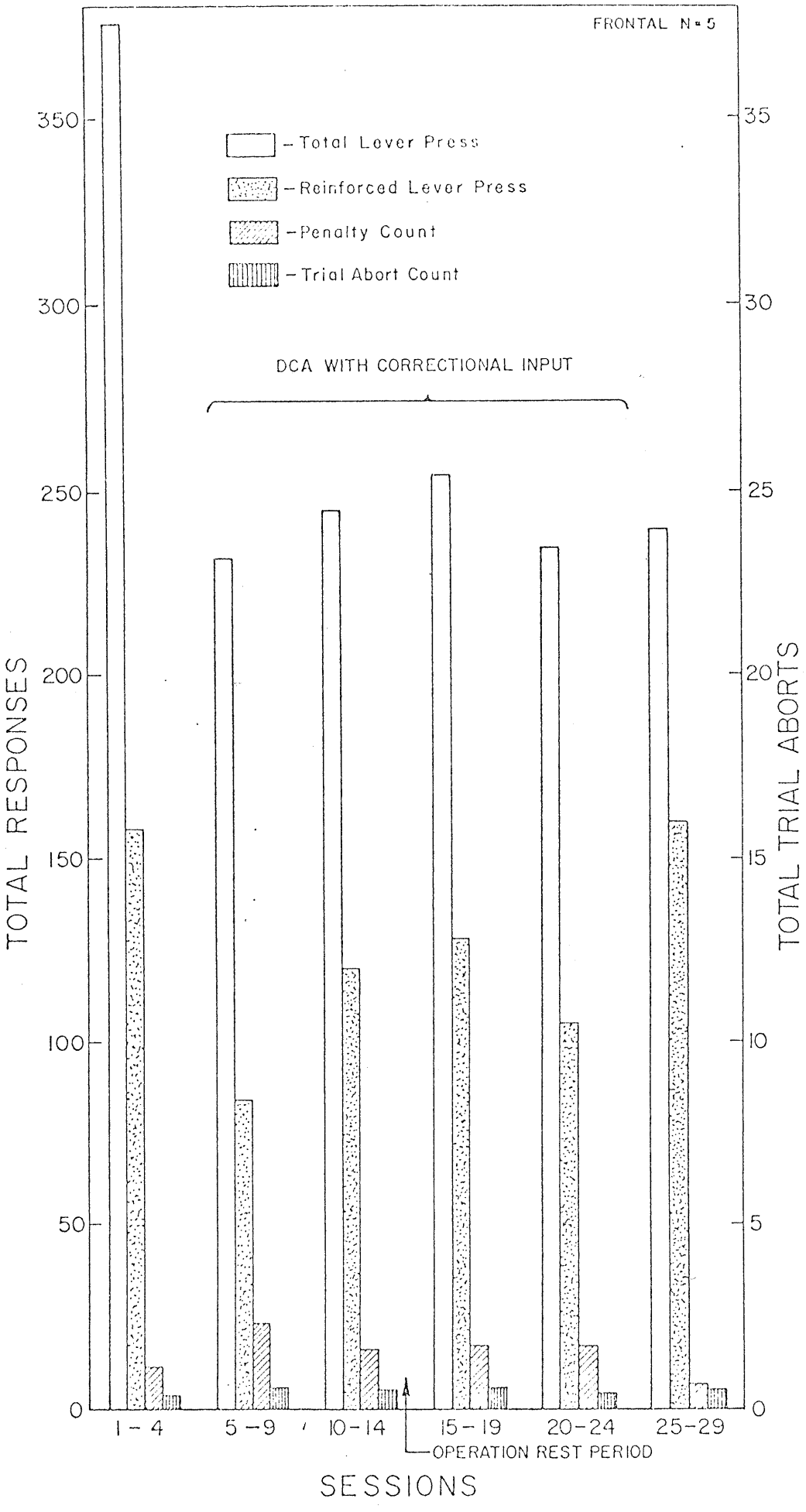

FRONTAL N=5
Total Lever Press
Reinforced Lever Press
Penalty Count
Trial Abort Count
DCA WITH CORRECTIONAL INPUT
TOTAL RESPONSES
TOTAL TRIAL ABORTS
0
50
100
150
200
250
300
350
0
5
10
15
20
25
30
35
1-4
5-9
10-14
15-19
20-24
25-29
OPERATION REST PERIOD
SESSIONS

Figure 46. Effects of 2.5mg/kg d-amphetamine on running wheel activity in frontal, frontal operated control, and normal rats. Exper imental session started immediately after intraperitonial injection of drug. Number of rotations were counted at 10minute interval for 100 minutes. Note the second peak exhibited by frontal animals dur ing the second half of the session.

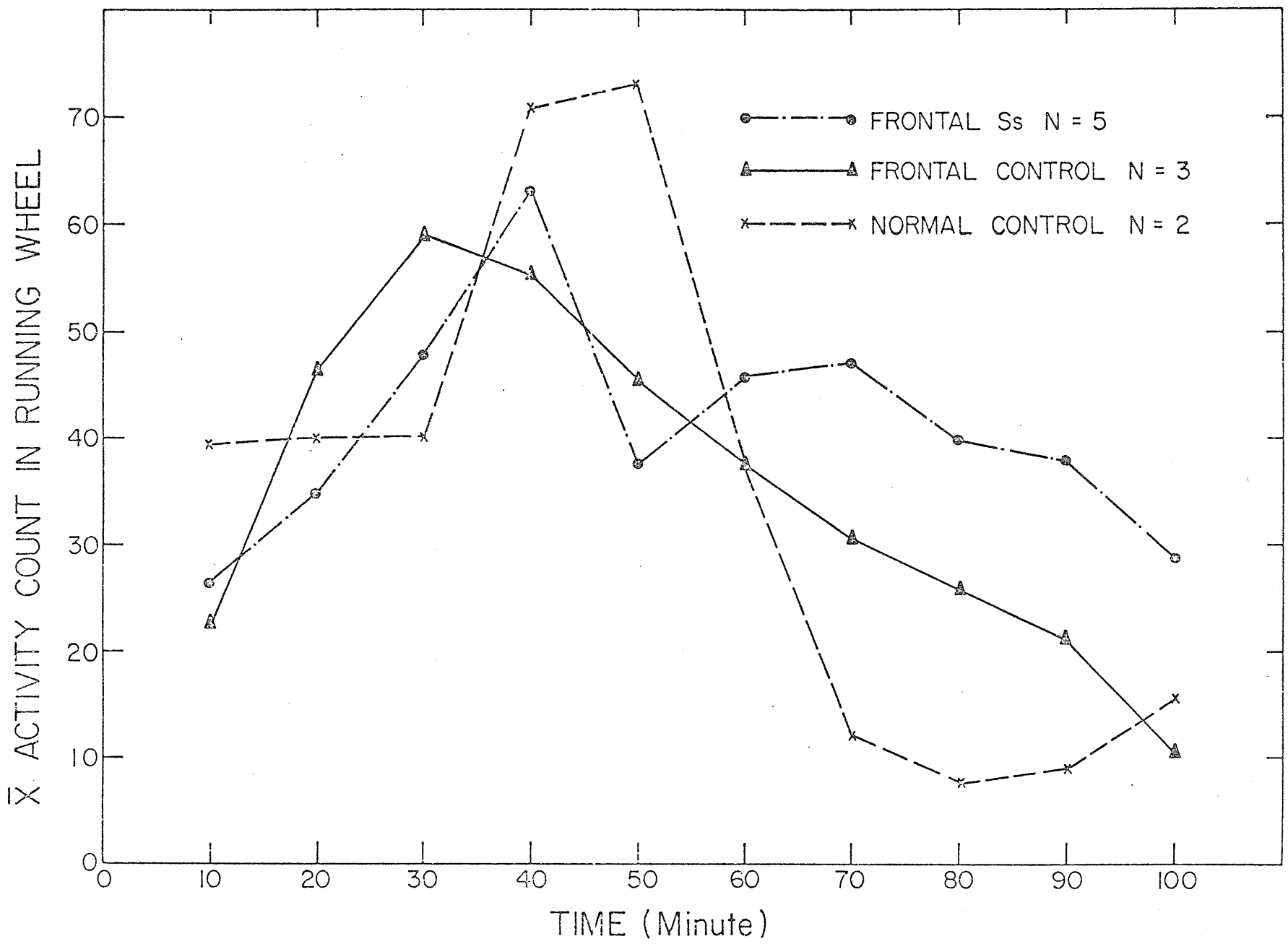

FRONTAL Ss N = 5
FRONTAL CONTROL N = 3
NORMAL CONTROL N = 2
X̄ ACTIVITY COUNT IN RUNNING WHEEL
TIME (Minute)
0
10
20
30
40
50
60
70
80
90
100

Figure 47. Mean response latencies of frontal Ss before and after bilateral electrolytic lesions in the frontal area. Each data point was a mean of 40 daily trials averaged over all Ss in that group. Post-operatively system showed reduced response latencies on DCA with I/O control.System returned to pre-operative pre-I/O control level in response latencies during last 5 days when I/O control was removed. Data was transformed to continuous function via weighted least square fit polynomial.

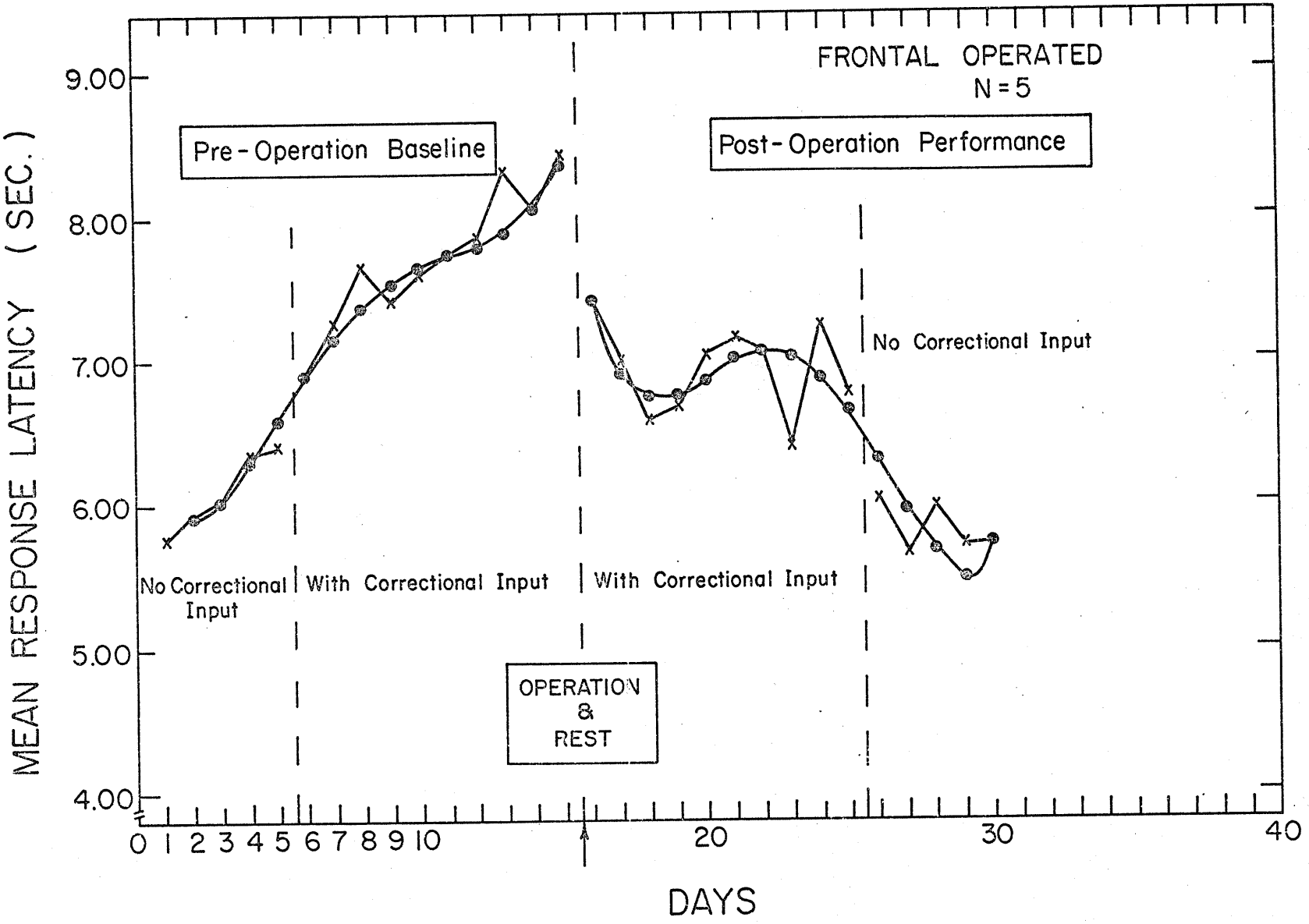

FRONTAL OPERATED
N = 5
Pre-Operation Baseline
Post-Operation Performance
No Correctional Input
No Correctional Input
With Correctional Input
With Correctional Input
OPERATION
&
REST
MEAN RESPONSE LATENCY (SEC.)
9.00
8.00
7.00
6.00
5.00
4.00
0 1 2 3 4 5 6 7 8 9 10
20
30
40
DAYS

Figure 48. Difference plot P(0)-P(T) of the frontal group. system showed tendency to perform at level which resembled that of pre-operative pre-I/O control input, resulting in reduced difference The peak of the curve suggested attempt of the system to regain performance level as demanded by DCA with I/O control.

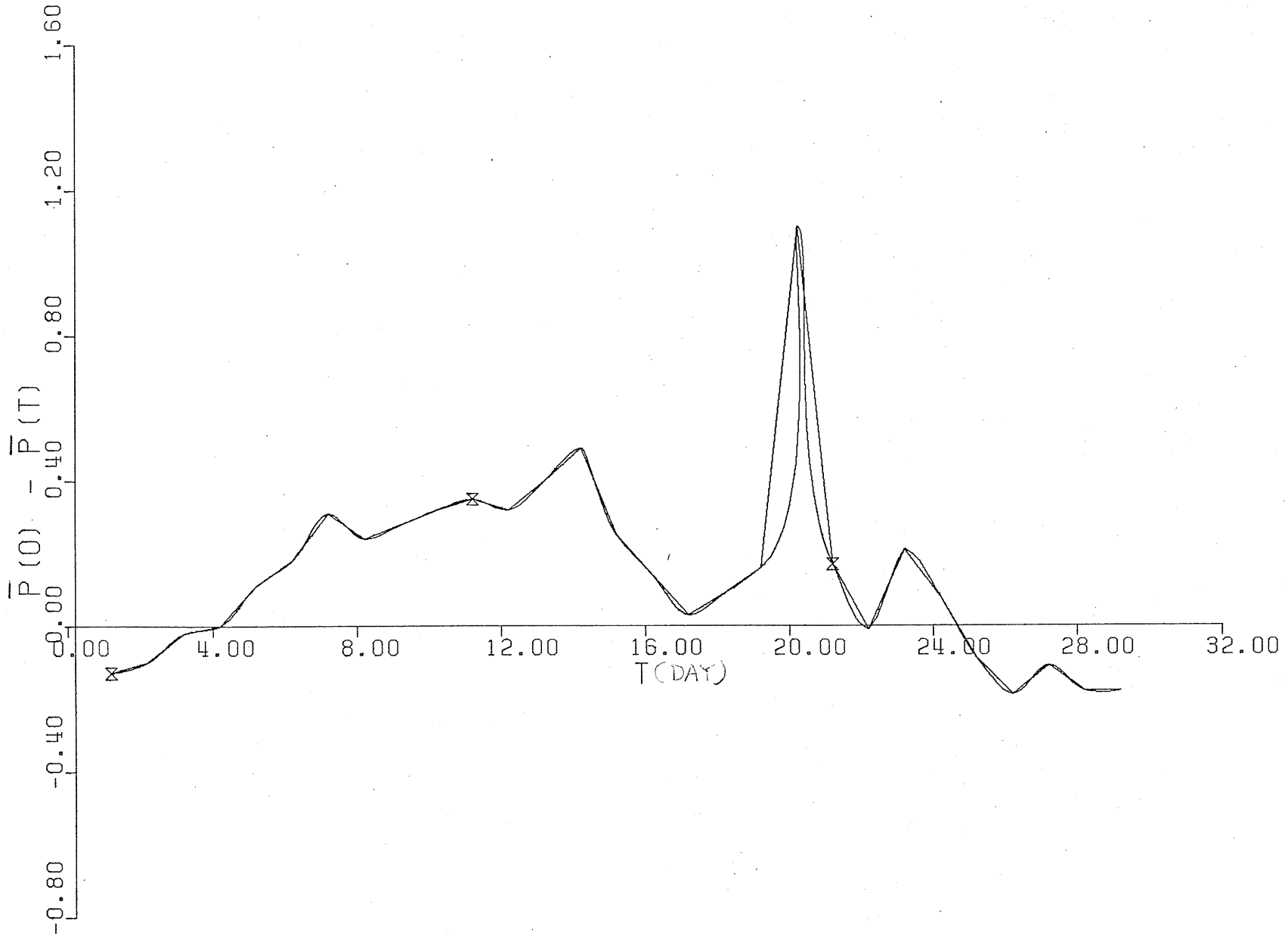

1.60
1.20
0.80
0.40
0.00
-0.40
-0.80
$\overline{P}(O) - \overline{P}(T)$
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T(DAY)

Figure 49. $\overline{P}$ plot of the frontal system over 29 daily sessions. Graph showed $\overline{P}$ as continuous time function.

1.30
1.40
1.50
1.60
1.70
1.80
1.90
pI
32.00
28.00
24.00

Figure 50. Phase plane representation of the frontal system. All 29 data points were considered in description of the behavioural history. Prior to lesion, the superimposed I/O control drove the system to perform at reduced vlaues for $\bar{P}$ and $d\bar{P}/dT$. Post-operatively the system showed tendency to deviate from the previously achieved state (increased $\bar{P}$ values).

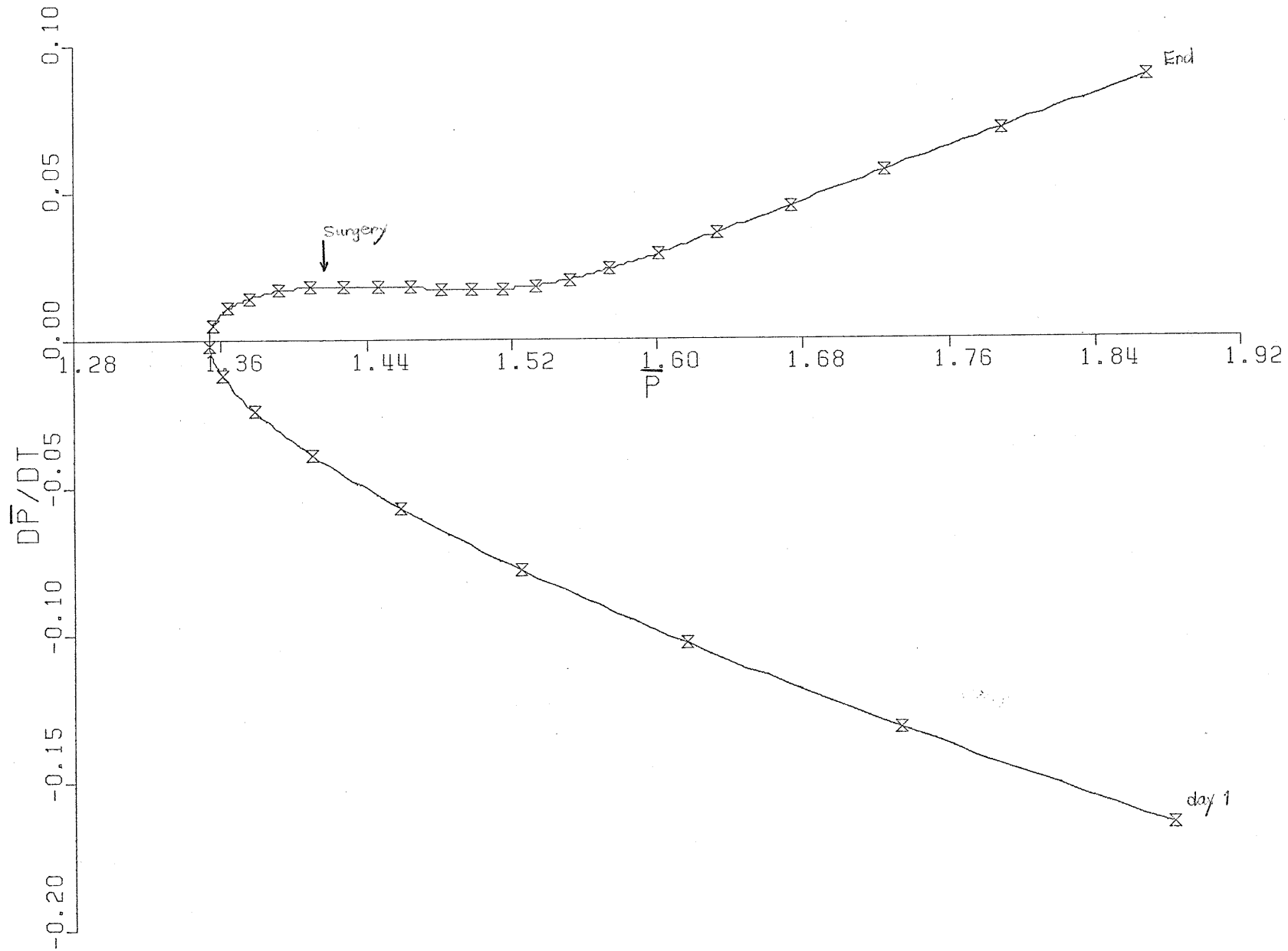

0.10
0.05
0.00
-0.05
-0.10
-0.15
-0.20
DP/DT
1.28
1.36
1.44
1.52
1.60
1.68
1.76
1.84
1.92
P
Surgery
End
day 1

Figure 51. $\overline{P}$ plot of frontal system prior to lesion. System showed reduction in P's with practice on DCA with I/O control.

FT Pre-op
S.

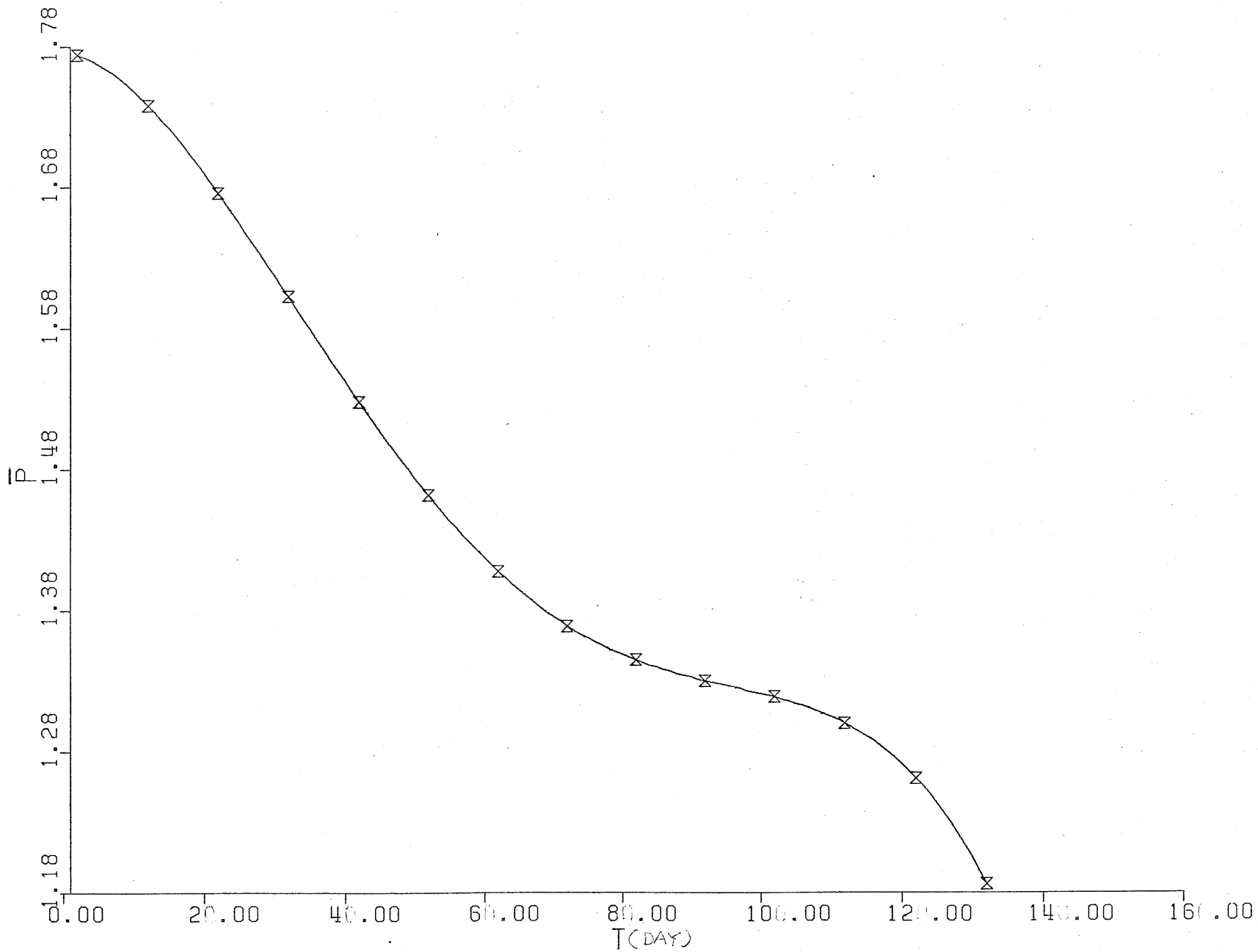

1.78
1.68
1.58
1.48
1.38
1.28
1.18
$\bar{P}$
0.00
20.00
40.00
60.00
80.00
100.00
120.00
140.00
160.00
T(DAY)

Figure 52. P plot of frontal system subsequent to lesioning. Behavior on ICA with I/O control and on baseline DCA showed damped oscillation.

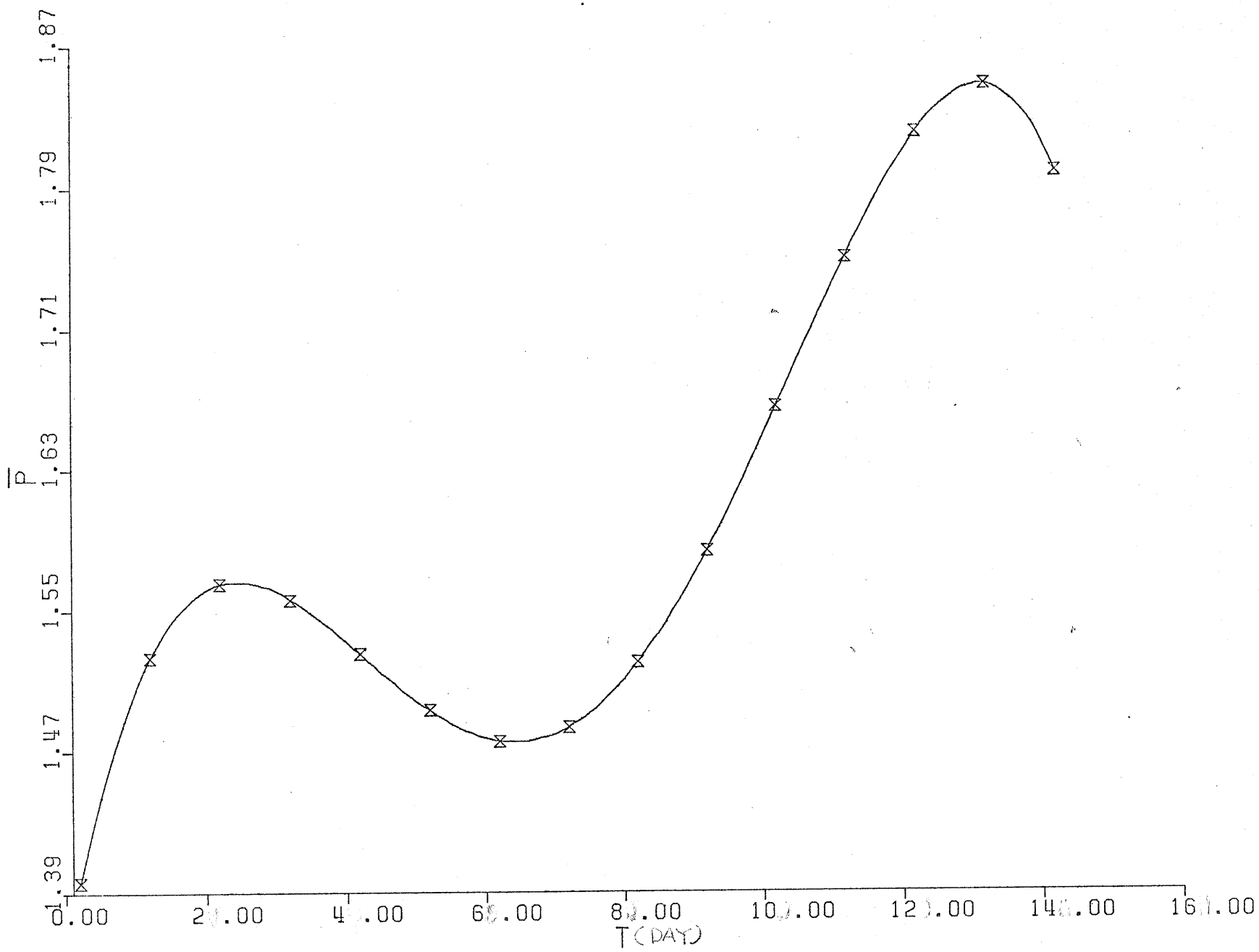

1.87
1.79
1.71
1.63
1.55
1.47
1.39
$\overline{P}$
0.00
T(DAY)

Figure 53.Phase diagram cf frontal system prior to lesion. System approached unstable focus when system put on DCA with I/O control.It then mcved toward still smaller p values with accelerated speed with further practice on the schedule.

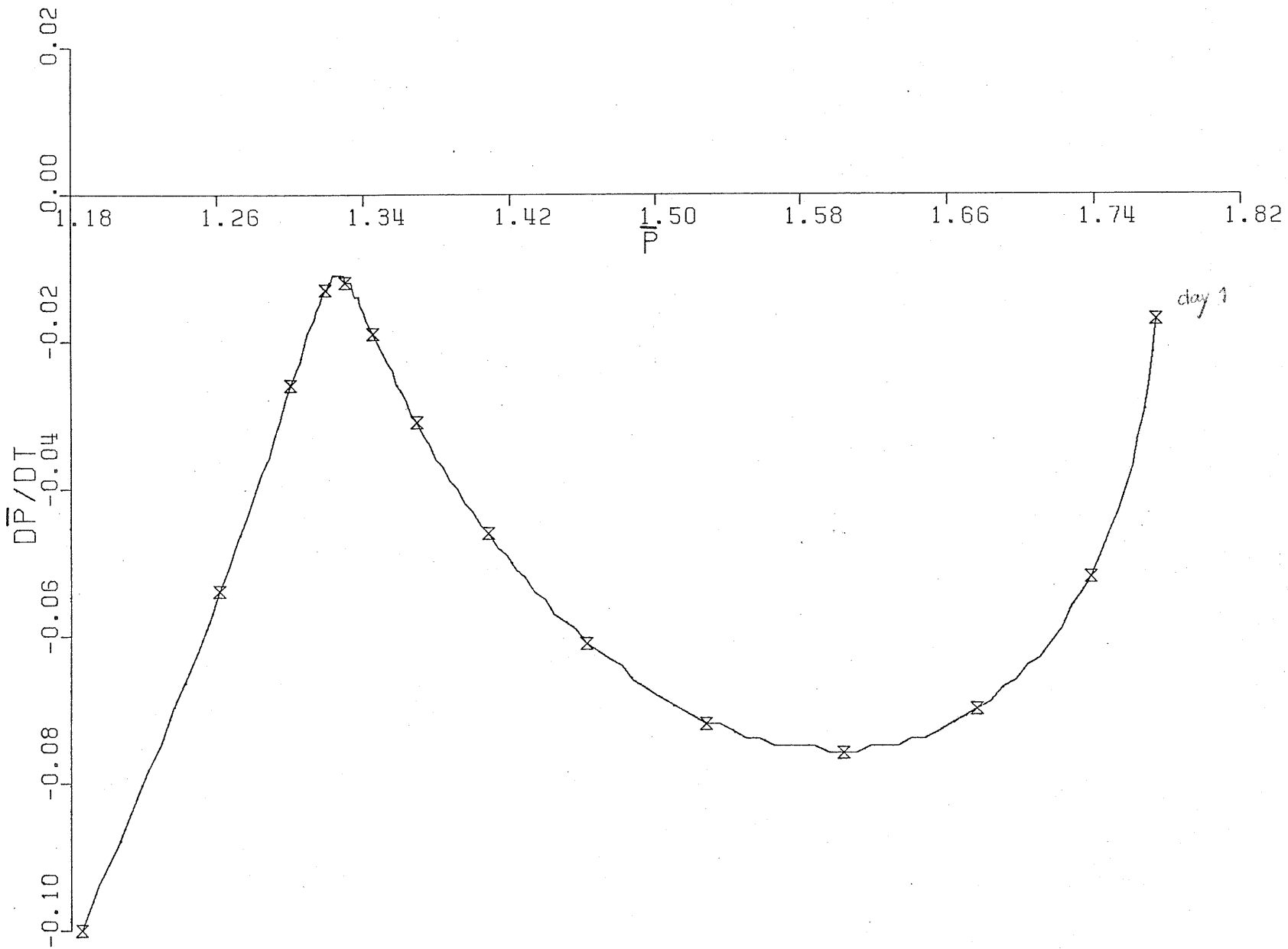

0.02
0.00
-0.02
-0.04
-0.06
-0.08
-0.10
1.18
1.26
1.34
1.42
1.50
1.58
1.66
1.74
1.82
$\overline{P}$
$D\overline{P}/DT$
day 1

Figure 54. Phase diagram of frontal system subsequent to lesioning. System executed a limit cycle with center of focus around P axis. It moved toward increased P's when I/O control was removed.

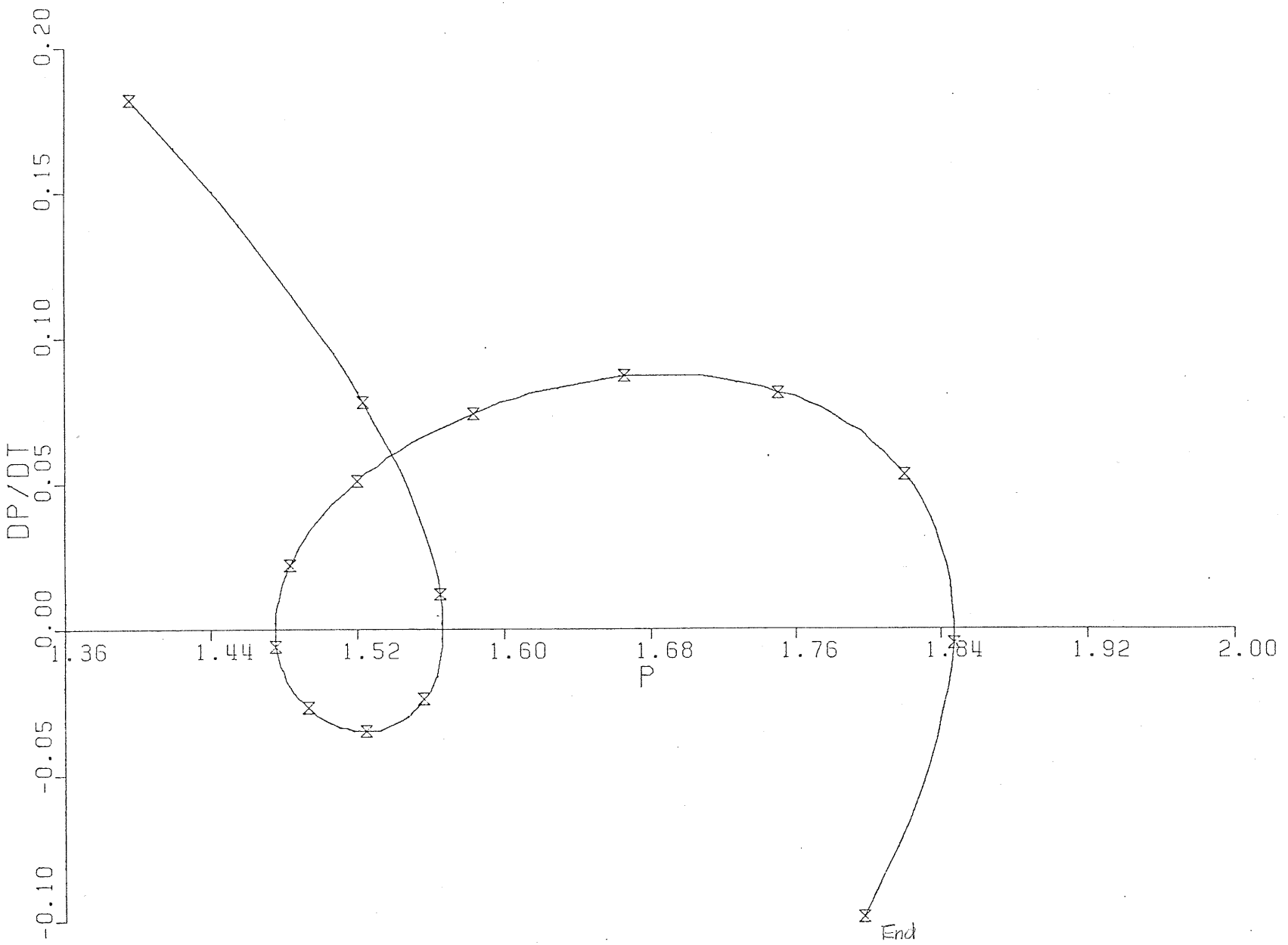
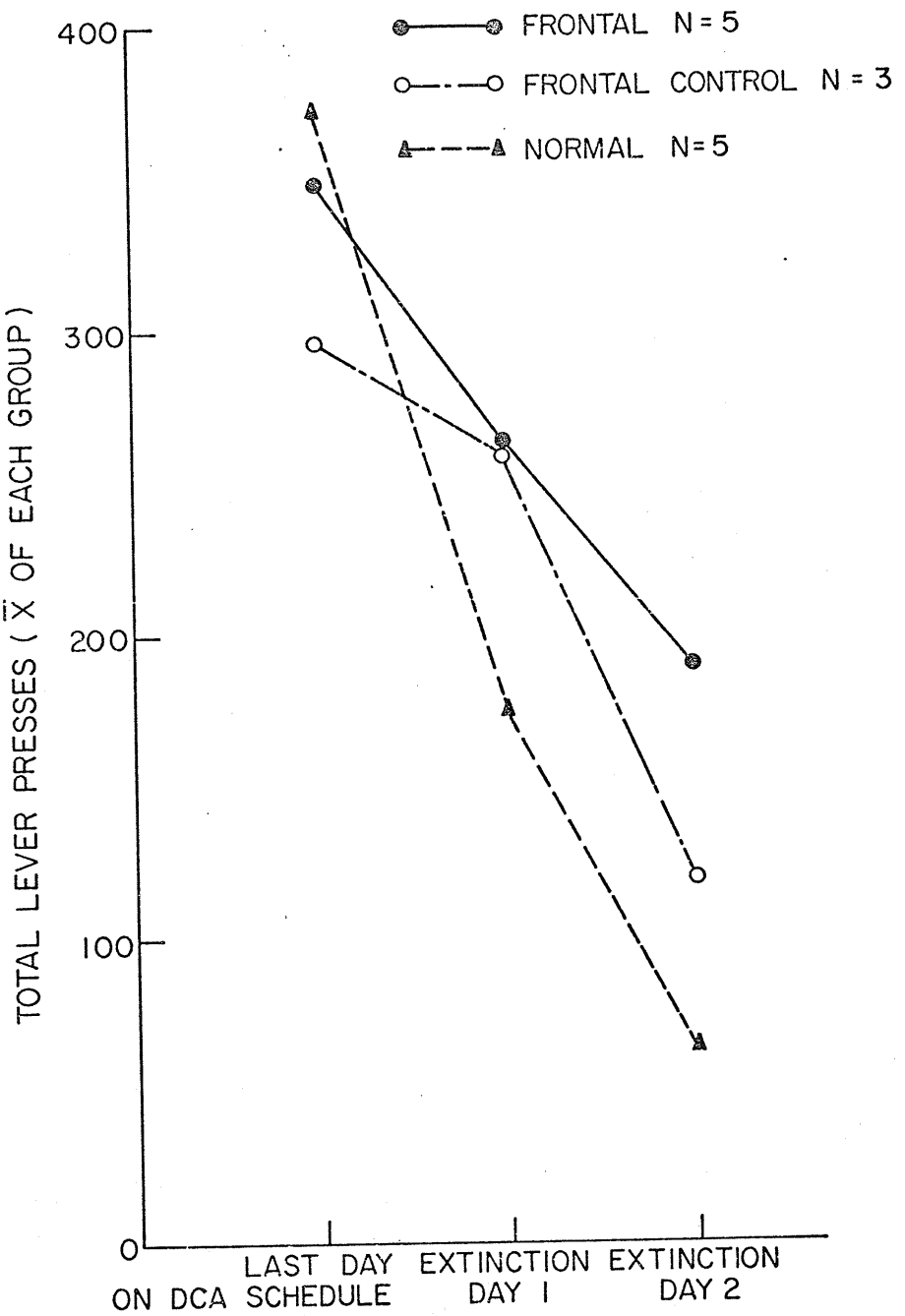

DP/DT
0.20
0.15
0.10
0.05
0.00
-0.05
-0.10
1.36
1.44
1.52
1.60
1.68
1.76
1.84
1.92
2.00
P
End

Figure 55. Extinction curves of the frontal, frontal control and normal groups on DCA. Similar trend observed for all froups.


FRONTAL N = 5
FRONTAL CONTROL N = 3
NORMAL N = 5
400
300
200
100
0
TOTAL LEVER PRESSES ($\bar{X}$ OF EACH GROUP)
LAST DAY ON DCA SCHEDULE
EXTINCTION DAY 1
EXTINCTION DAY 2

GROUP VI the group with bilateral sham lesion in frontal pole (Ft C)

A.general observation: The behaviour pattern of the Ft C ss were not different before and after the sham lesion.

B.activity in the rotational running wheel: Unlike the Ft Ss which showed a period of heightened activity in the running wheel,the Ft C Ss showed a post-operative reduction in this behaviour. This group differd from the control in its pre-operative level,ie. it had a higher level of activity as measured by the wheel prior to the sham lesion. Post-operatively the activity level was not different from that of the control (figure 44).

C.performance on the DCA:

(1) Figure 56 is the mean of a) total lever presses per session,b)reinforced lever press c)penalty count and d)trial abort count of the 29 daily sessions which were grouped into six time blocks. The overall picture is similar to the other control groups. On the whole,the mean of the total lever press was reduced when the system was on the I/O control.The number of total reinfirced lever press was the greatest,while the total number of the penalty and trial abort count were the lowest in the last block.

(2)Figure 57 is the mean response latencies of the

Ft group over the 29 experimental sessions. Post-operatively, there was slight reduction in the mean response latency in the I/O phase of the testing. Still, there was ccnsiderable overlap in the response samples.

P(0)-P(T):The difference between the pre-I/O bade-line and the subsequent sessions was a function of the schedule of demand (figure 58). Upon removal of the I/O control, the P(0)-P(T) returned to the same level as pre-I/O sessions.

(4) P plot:Figures 59,60 & 61 are the p plot of the system's adjustment in the time domain. As with other operated ccntrcls, these Ss ' behavioural adjustment prior and subsequent to the sham lesion were mirror images cf each other.

(5) DP/DT vs P: In gifure the system was charac-terized by an unstable focus which subsequently diverged towards a lengthening of response latencies,with the force of extinctcn pre-dominating,just as was the case with the pre-operative system in the FT group.Post-operatively, the system moved to a limit cycle around the P axis. But the limit cycle was a soft one . The behavioural system deviated frcm the tendency to a limit cycle when the I/O control was removed(figure 62).

Figure 56. The means of a) total lever press, b) reinforced lever press, c) penalty count and d) trial abort counts of the frontal sham lesioned group. Data from 29 daily sessions were averaged over all Ss in respective groups. For explanation, see text.

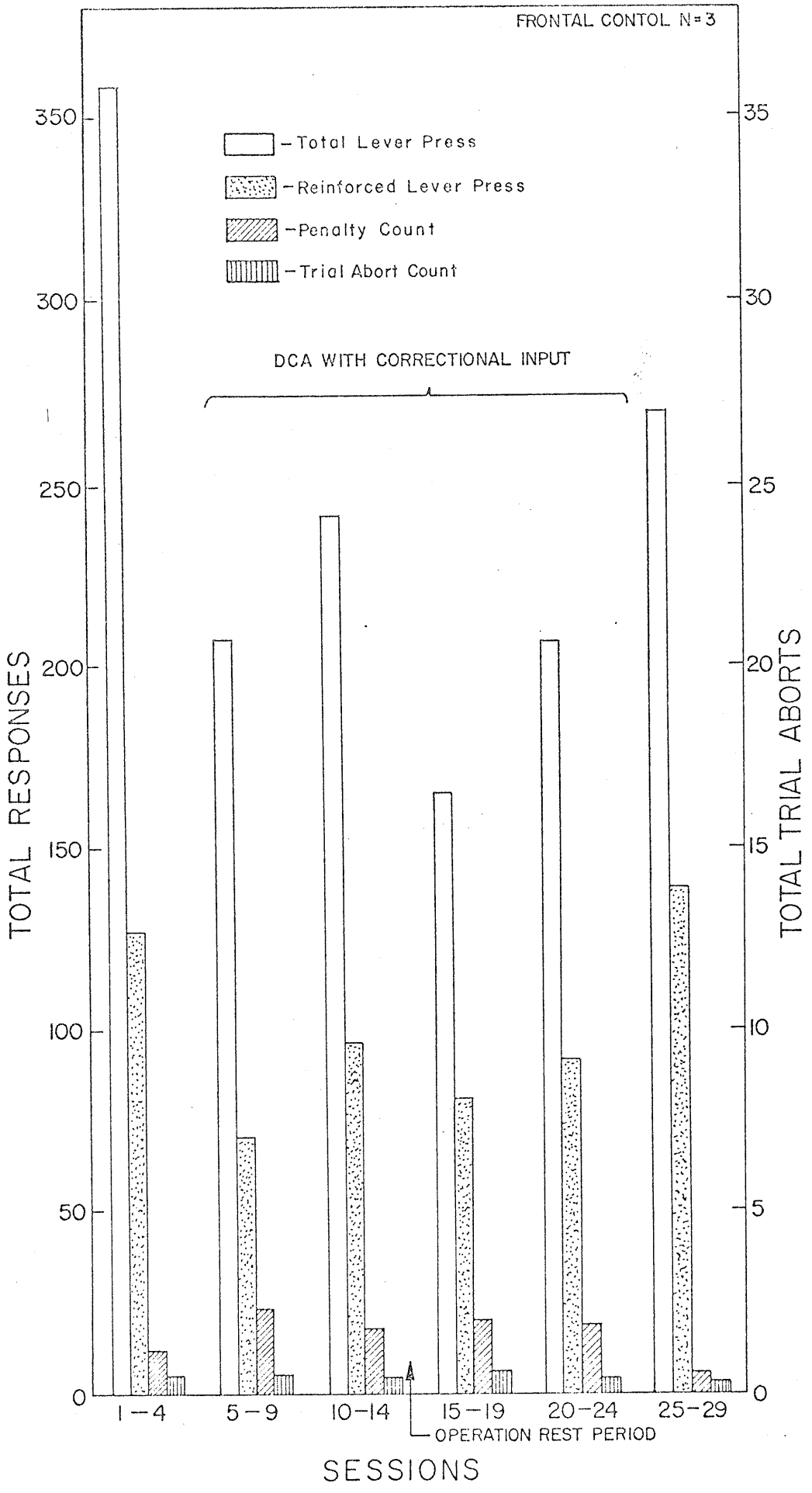

FRONTAL CONTOL N=3
-Total Lever Press
-Reinforced Lever Press
-Penalty Count
-Trial Abort Count
DCA WITH CORRECTIONAL INPUT
TOTAL RESPONSES
TOTAL TRIAL ABORTS
350
300
250
200
150
100
50
0
35
30
25
20
15
10
5
0
1-4
5-9
10-14
15-19
20-24
25-29
OPERATION REST PERIOD
SESSIONS

Figure 57. Mean response latencies of frontal sham lesioned group. Each data point was a mean of 40 daily trials averaged over all ss. Corre sponding curve was the first best fit on the weighted least square polynomial.

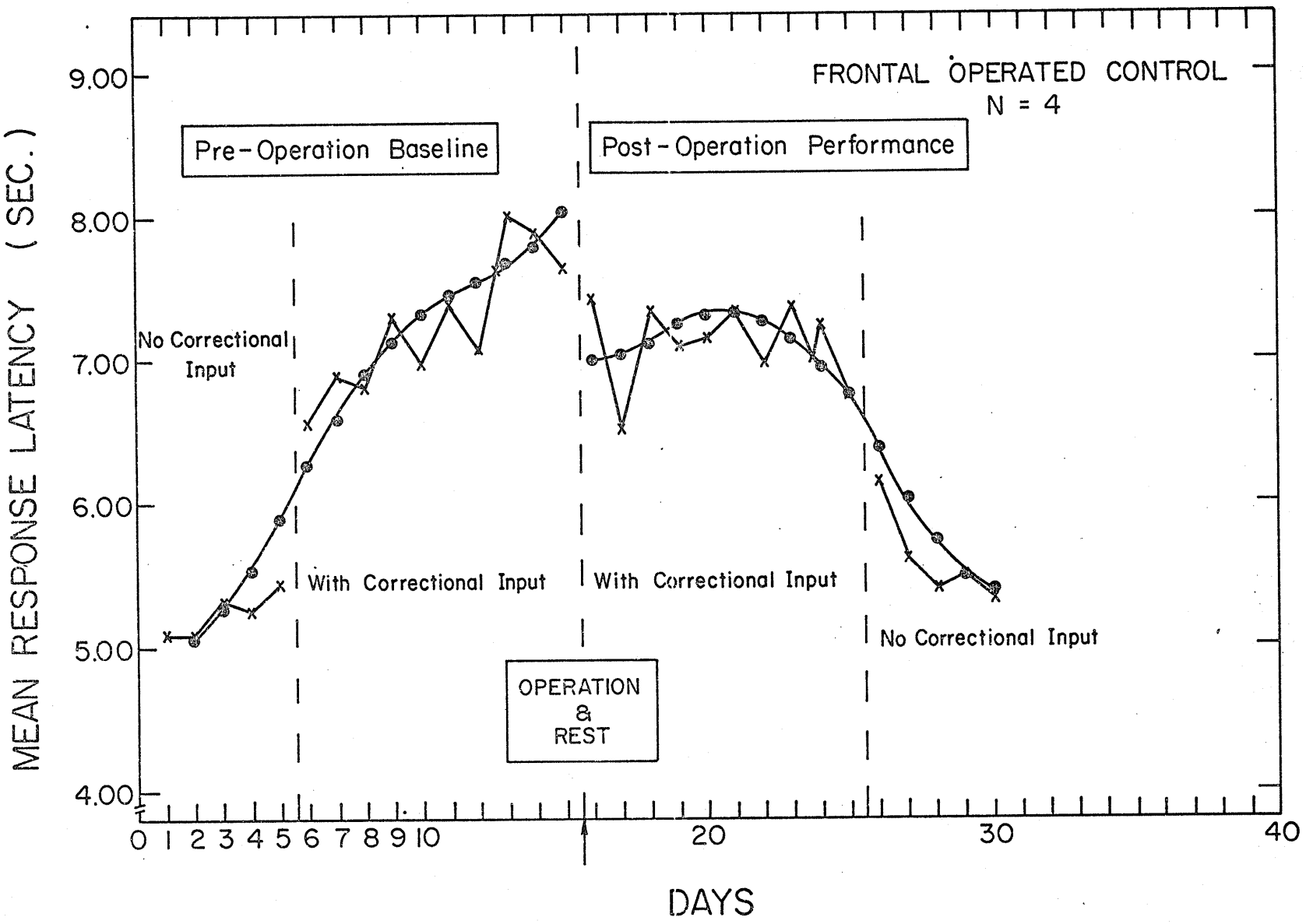

FRONTAL OPERATED CONTROL
N = 4
Pre-Operation Baseline
Post-Operation Performance
No Correctional Input
With Correctional Input
With Correctional Input
No Correctional Input
OPERATION & REST
MEAN RESPONSE LATENCY (SEC.)
9.00
8.00
7.00
6.00
5.00
4.00
0 1 2 3 4 5 6 7 8 9 10
20
30
40
DAYS

Figure 58. Difference plot of the frontal sham lesioned group. Unlike the frontal system, It maintained performance level after the surgery.

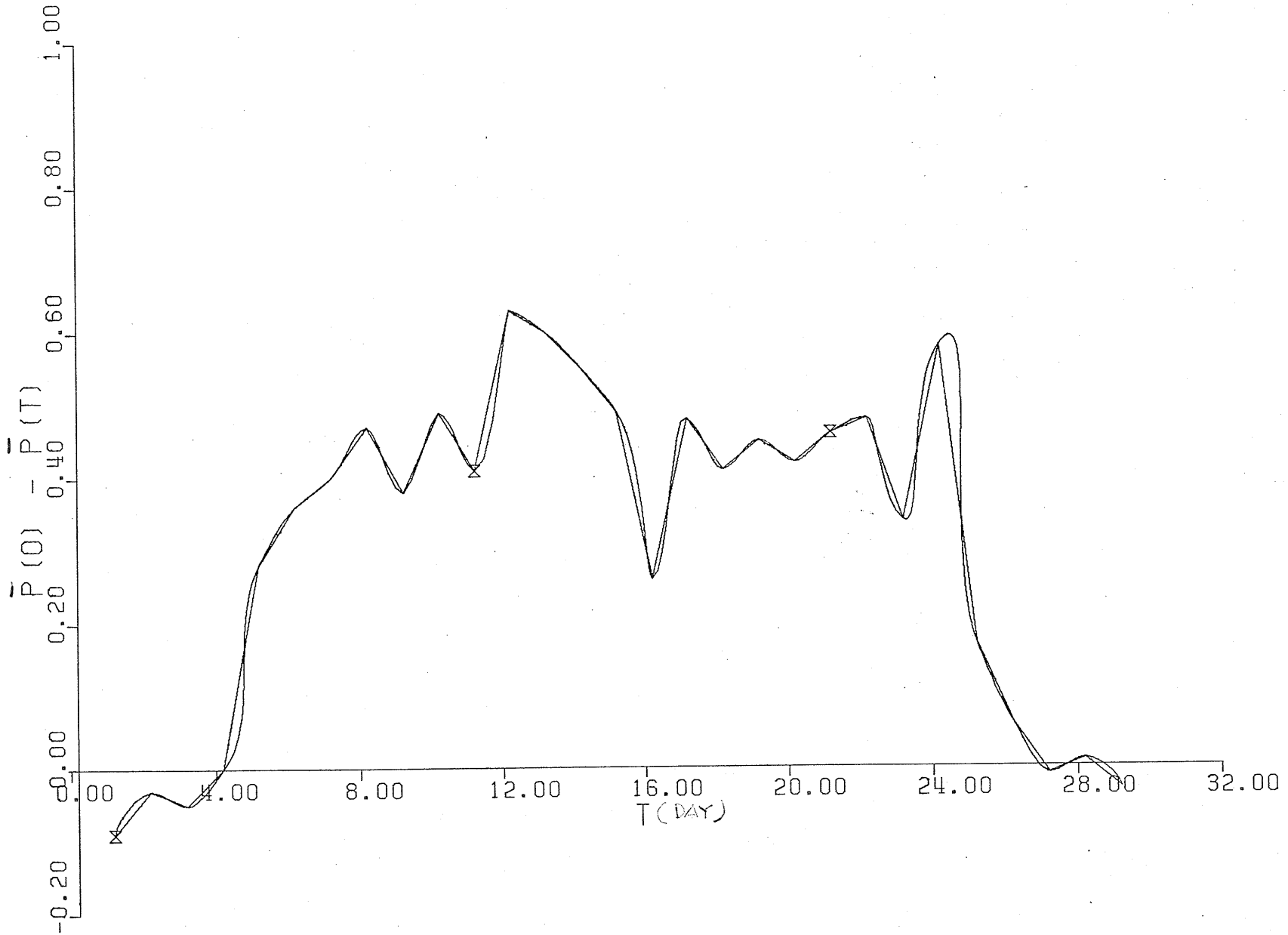

1.00
0.80
0.60
0.40
0.20
0.00
-0.20
$\bar{P}(0) - \bar{P}(T)$
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T (DAY)

Figure 59. P plot of the frontal sham lesioned prior to surgery.

FT C Pre-OP

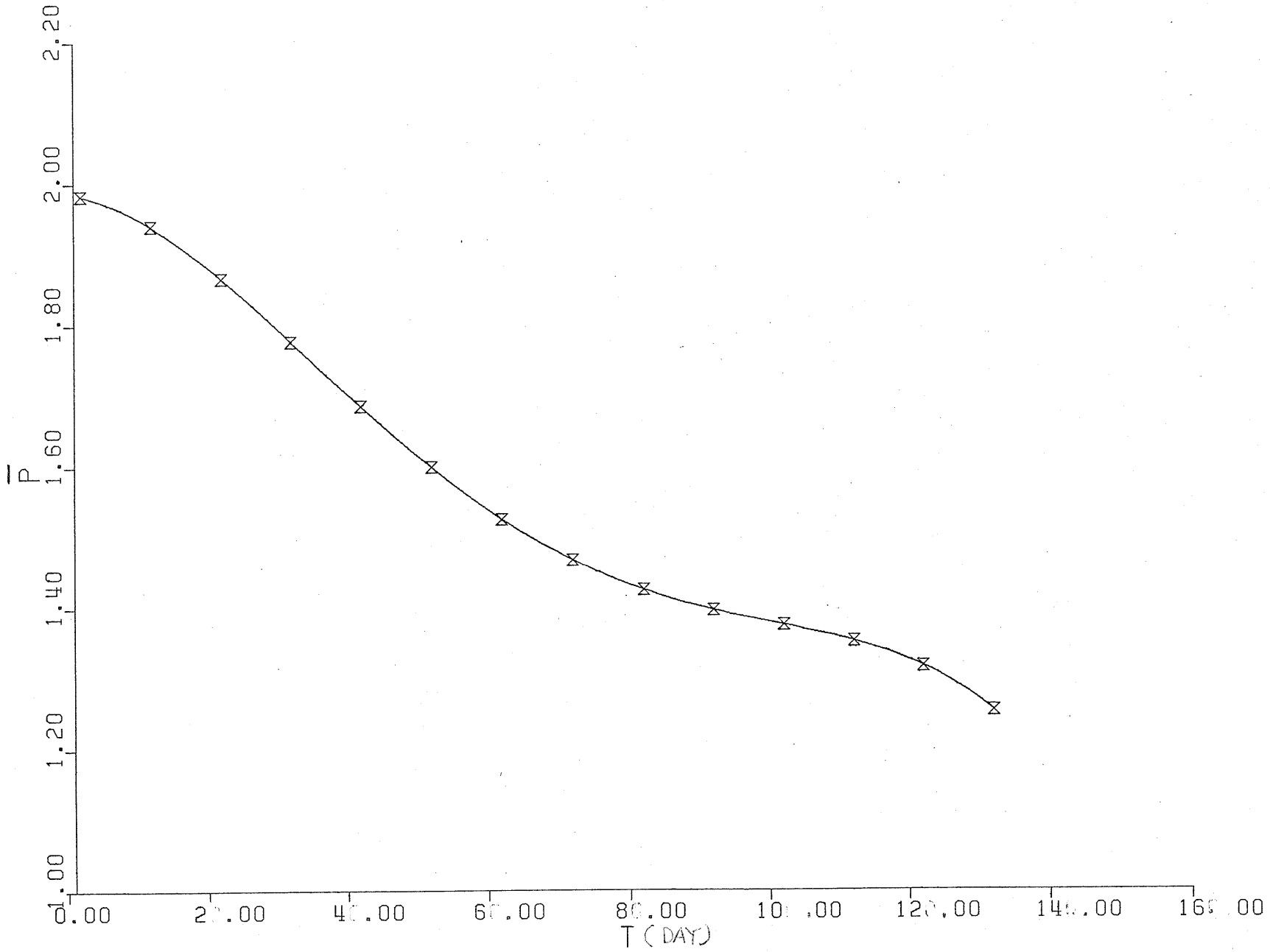


Figure 60. P plot of the frontal sham lesioned group subsequent to surgery.

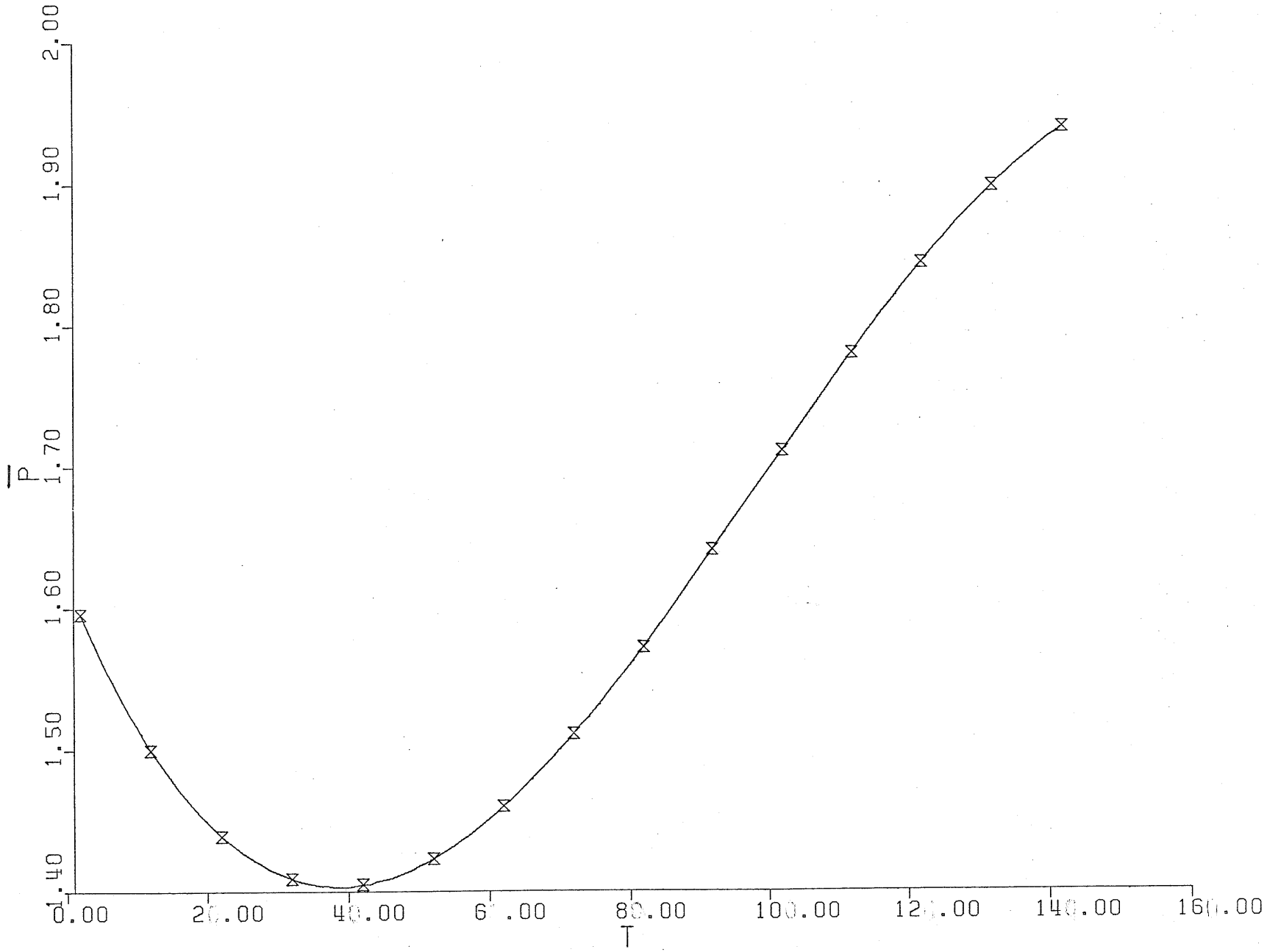

2.00
1.90
1.80
1.70
1.60
1.50
1.40
P
0.00
20.00
40.00
60.00
80.00
100.00
120.00
140.00
160.00
T

Figure 61. P plot of the system with frontal sham lesion.Perfo rmance before and after the surgery were mirror images of each other.

P
1.20
1.40
1.60
1.80
2.00
2.20
2.40
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T

Figure 62. Phase diagram of the group with sham lesion in the frontal pole.

2.10
2.00
1.90
1.80
1/P
1.70
1.60
1.50
1.40
1.30
0.12
0.08
0.04
0.00
-0.04
-0.08
-0.12
DP/DT

Figure 62 a. phase diagram of the Ft C system prior to surgery. System approached unstable focus and then moved to performance at still reduced P values.

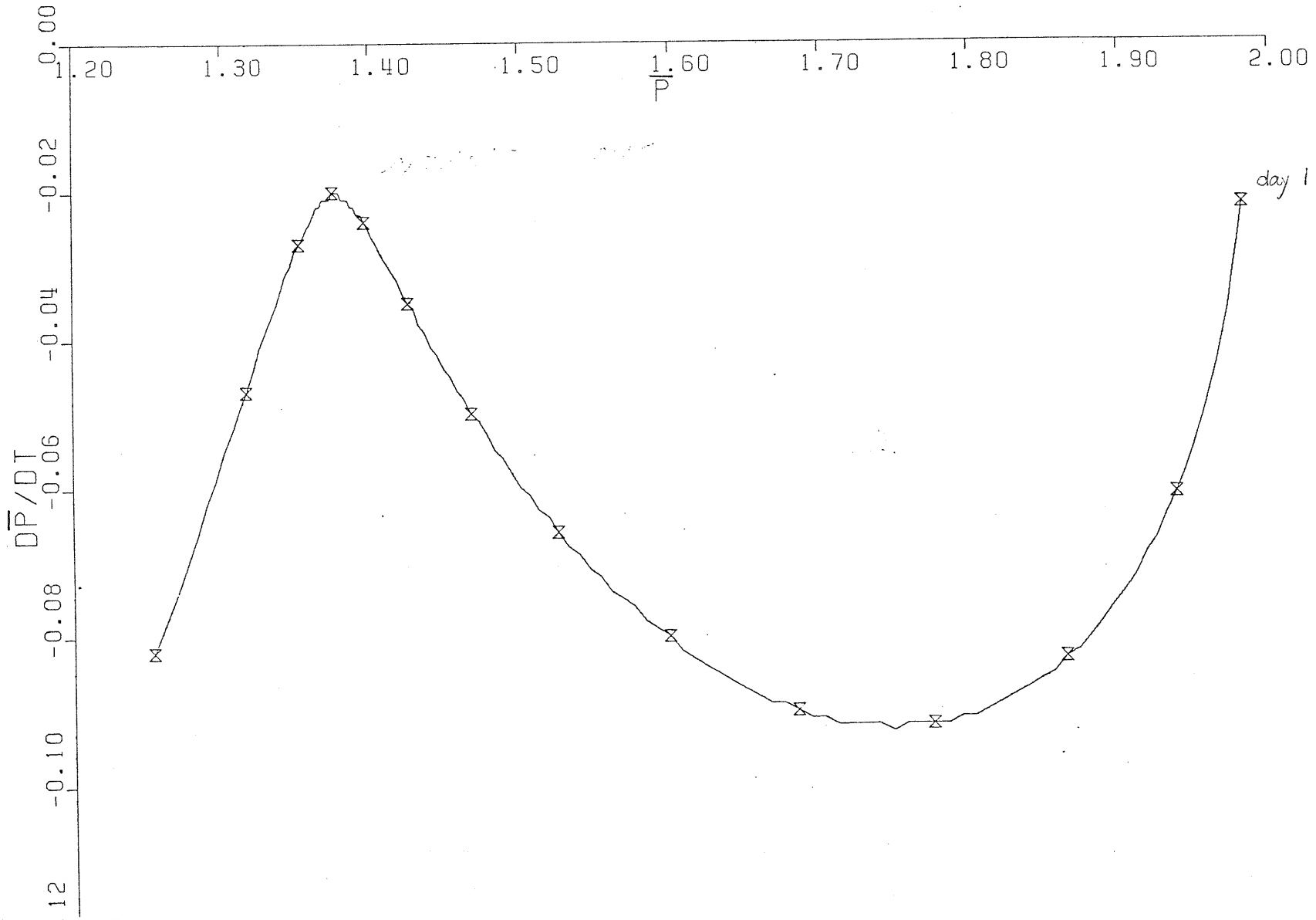

0.00
-0.02
-0.04
-0.06
-0.08
-0.10
12
DP/DT
1.20
1.30
1.40
1.50
1.60
1.70
1.80
1.90
2.00
1/P
day 1

Figure 62 b. Phase diagram of Ft C system after sham lesion. System functioned in limit cycle before ajdustment to change in DCA.

2.00
1.92
1.84
1.76
1.68
1/P
1.60
1.52
1.44
1.36
-0.12
-0.08
-0.04
0.00
0.04
0.08
0.12
DP/DT

GROUP VII the group with bilateral electrolytic lesion in the posterior aspect of the caudate nucleus (P CD)

A.general observation:Post-operatively, the Ss were generally sluggish in appearance.They spent much of the time in the home cage away from the front of the cage. They showed signs of irritation when handled. These signs of irritation to human handling gradually subsided by the 2nd post-operative week.

B. activity level measured by the rotational running wheel: Figure 63 presented the mean of the total number of rotations over 30 minute period. Data of three pre-operative and 5 post-operative sessions are presented. As shown on the graph, There is no difference in the pre- and post-operative level of running wheel activity.

C. performance on the DCA:

Figure 64 is the graphic presentation of the mean of a) total lever press,b)reinforced lever press,c) penalty count off and d)trial abort count of the 29 daily sessions. The mean number of total lever press and of reinforced lever press were the highest in the last time block . Post-operatively there was slight reduction in the mean total of reinforced lever press,which corresponded with a reduction in the penalty count and of total lever press. In other words the effect of the lesion was a general reduction in the response of the system to the

DCA. The depressed response of the system to the DCA was not present in the second post-operative time block.

(2) Figure 65 is the mean response latencies of the P CD on the DCA. The weighted least square fit of the pre-operative mean latenciessuggested the adjustment of the system to the changed level of demand to be linear. Post-operatively there was an initial period of increased response latencies during which the behaviourwas characterized by much nonresponses within a daily session. The response of the system then returned to a level comperable to that the the pre-operative baseline. Upon subsequent removal of the I/O control,the response of the system adjusted accordingly.

P(0)-P (T):The difference plot which used the day prior to the I/O control input as the P(0) showed that there was a large discrepancy between the baseline performance and that of the first and second post-operative testing sessions (figure 66).

P plot: when the responses of the P CD system wast ransformed into time respcnsethe response pattern of the system before and after the operation is a mirror image of each other. (Figure 67). When the pre-operative and post-operative sessions were analyzed separately, the pre-operative behavioural response was shown to be linear in its adjustment to the changed levels in demand.(figure 68). Post-operatively,(figure 69)the system showed large

initial negative relative error followed by a reduction in the value of both the relative error and the rate of change in the value of the error.

DP/DT vs P: When the system is seen as a continuous function in the time domain over the 29 daily sessions, the phase -diagram of the P CD group is shown as a behavioural system which evolved from the process of predominately reconditioning into that of extinction. Through practice the system then evolved into a limit cycle, maintaining a active equilibrium between the forces of extinction and recond itioning around the P axis (figure 70). While the phase diagram of the system pre-operatively did not differ from any other groups(figure 71) the phase diagram of the post-operative behavioural system was that of an underdamped system which responded to the control demand with a transient overshoot . Subsequently, it evolved into a an unstable limit cycle which moved away from the focus when the demand of the control signal was changed by the removal of the I/O control(figure 72). The response of the P CD post-operatively differed from that of the H CD in that the latter did not respond to the removal of I/O control.

Figure 73 is the results on the extinction phase of the experiment. The extinction curves of the P CD ,P CD C & normal C groups showed the same tendencies.

Figure 63. Mean number of revolution in the rotational running wheel in the P CD, P CD C and N groups. Each point on the graph was a mean of all Ss in that group. All groups maintained same levels of activity before and after the surgery or rest.

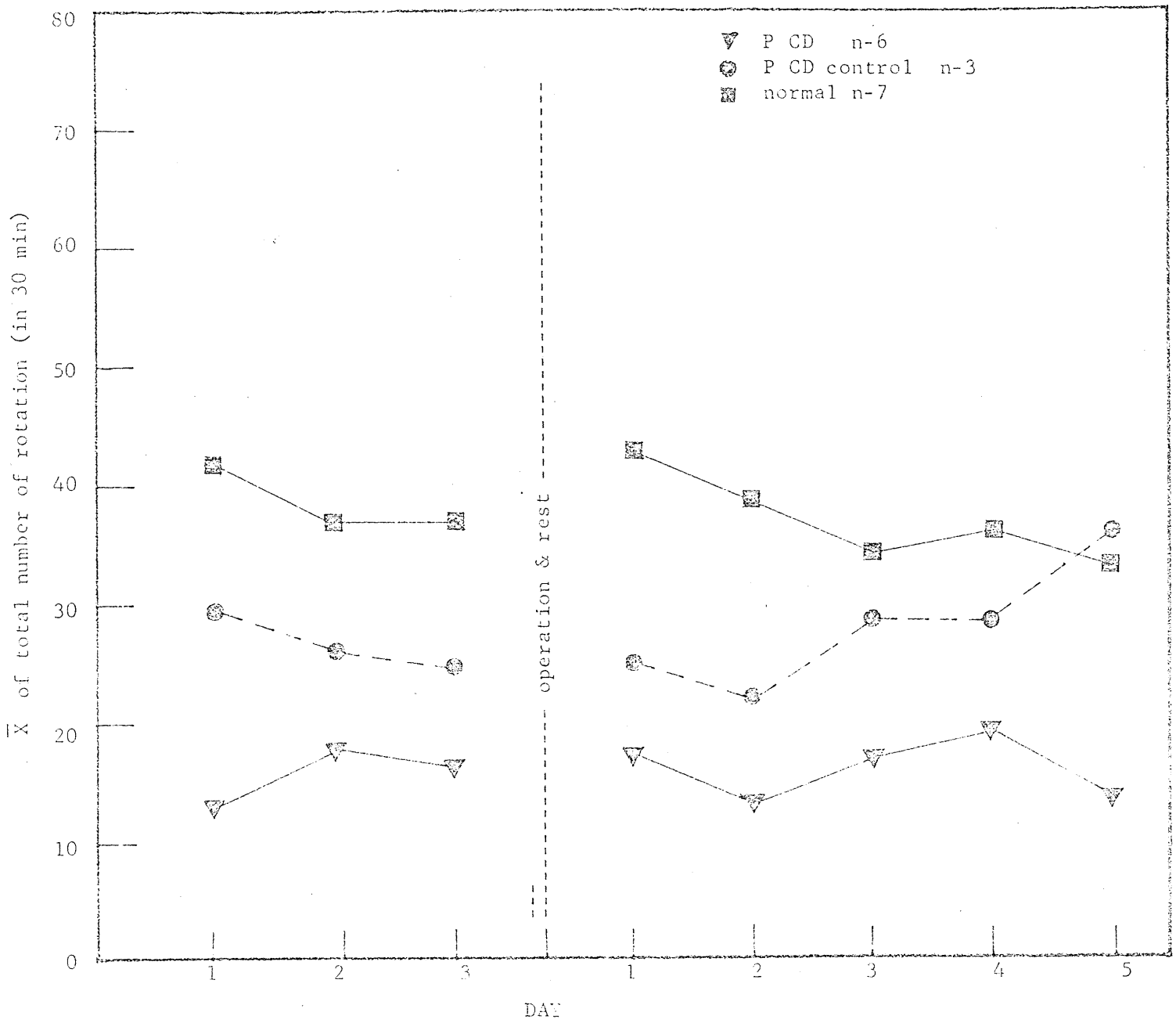

P CD n-6
P CD control n-3
normal n-7
operation & rest
$\bar{X}$ of total number of rotation (in 30 min)
80
70
60
50
40
30
20
10
0
1
2
3
1
2
3
4
5
DAY

Figure 64. Means of a) total lever press, b) reinforced lever press c) penalty count and d) trial abort count of the P CD group. Each value was an average of all Ss in the time block.

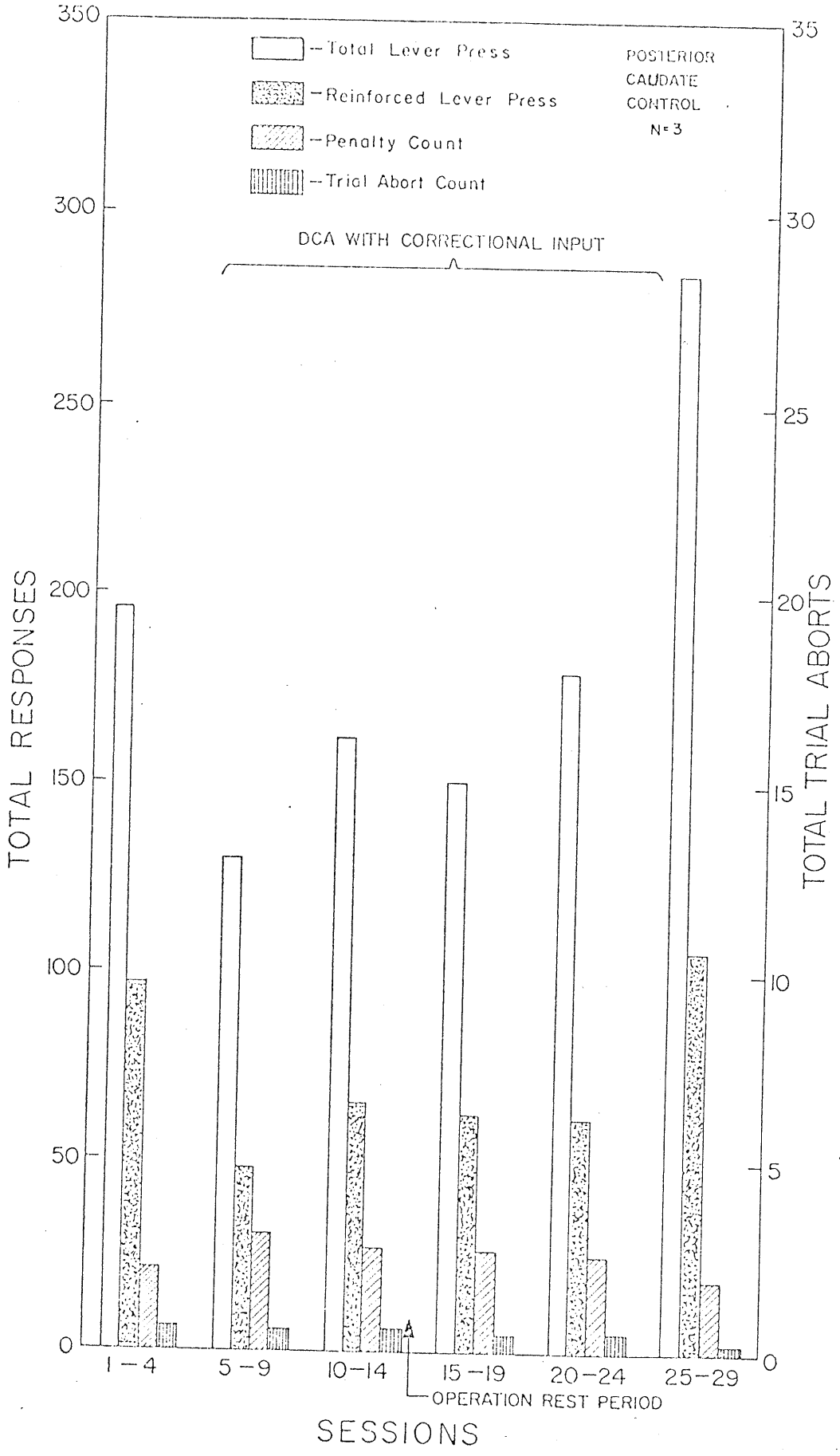

Total Lever Press
Reinforced Lever Press
Penalty Count
Trial Abort Count
POSTERIOR CAUDATE CONTROL N=3
DCA WITH CORRECTIONAL INPUT
TOTAL RESPONSES
TOTAL TRIAL ABORTS
350
300
250
200
150
100
50
0
35
30
25
20
15
10
5
0
1–4
5–9
10–14
15–19
20–24
25–29
A
OPERATION REST PERIOD
SESSIONS

Figure 65. The mean response latencies of the P CD on DCA. System showed increased response latencies during initial segment of post- operative sessions. Response latencies showed corresponding adjustment when system returned to baseline DCA.

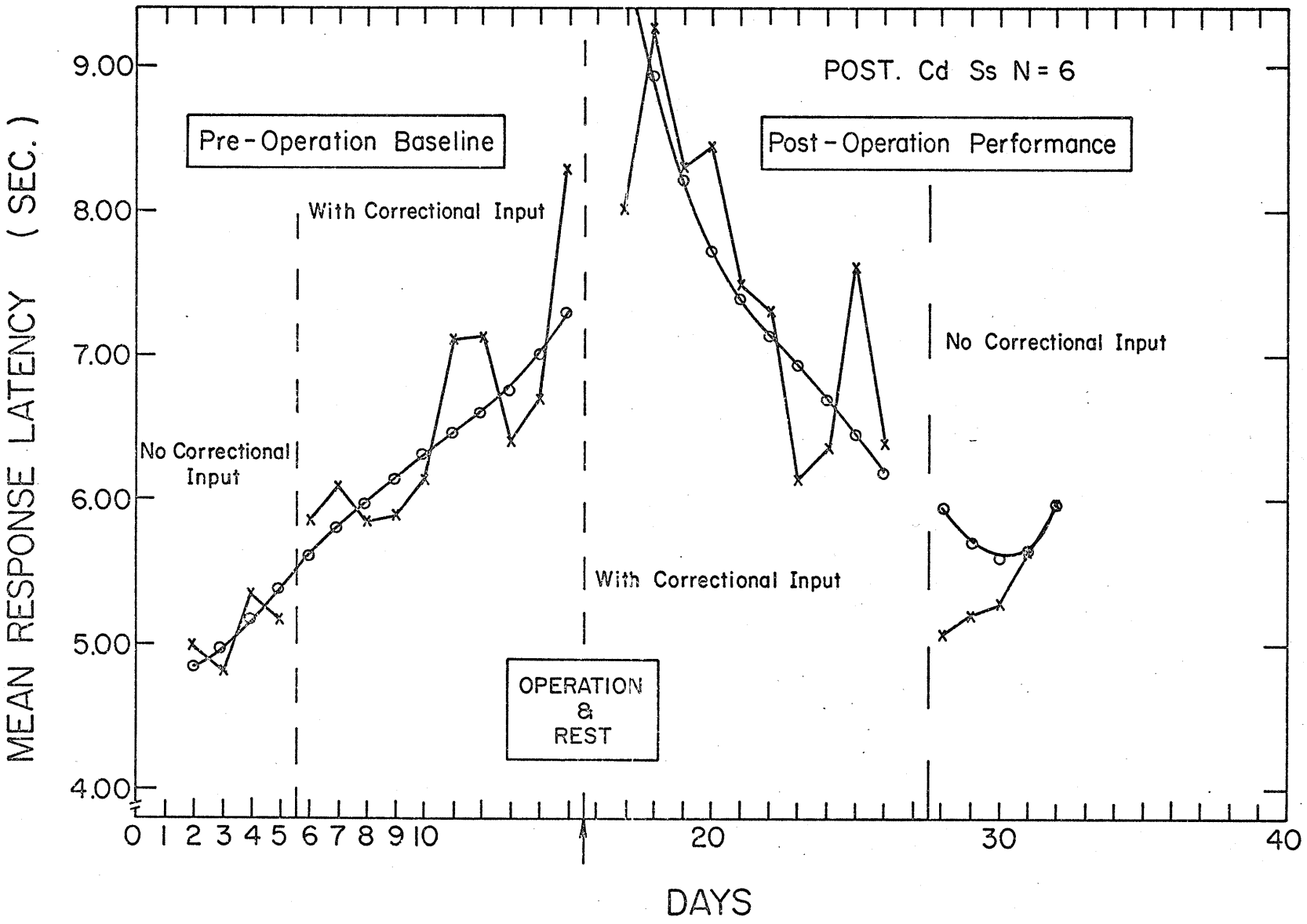

POST. Cd Ss N = 6
Pre-Operation Baseline
Post-Operation Performance
With Correctional Input
No Correctional Input
No Correctional Input
With Correctional Input
OPERATION & REST
MEAN RESPONSE LATENCY (SEC.)
9.00
8.00
7.00
6.00
5.00
4.00
0 1 2 3 4 5 6 7 8 9 10
20
30
40
DAYS

Figure 66. Difference plot of P CD system. The peak of the curve was during the initial segment of the post-operative test sessions.

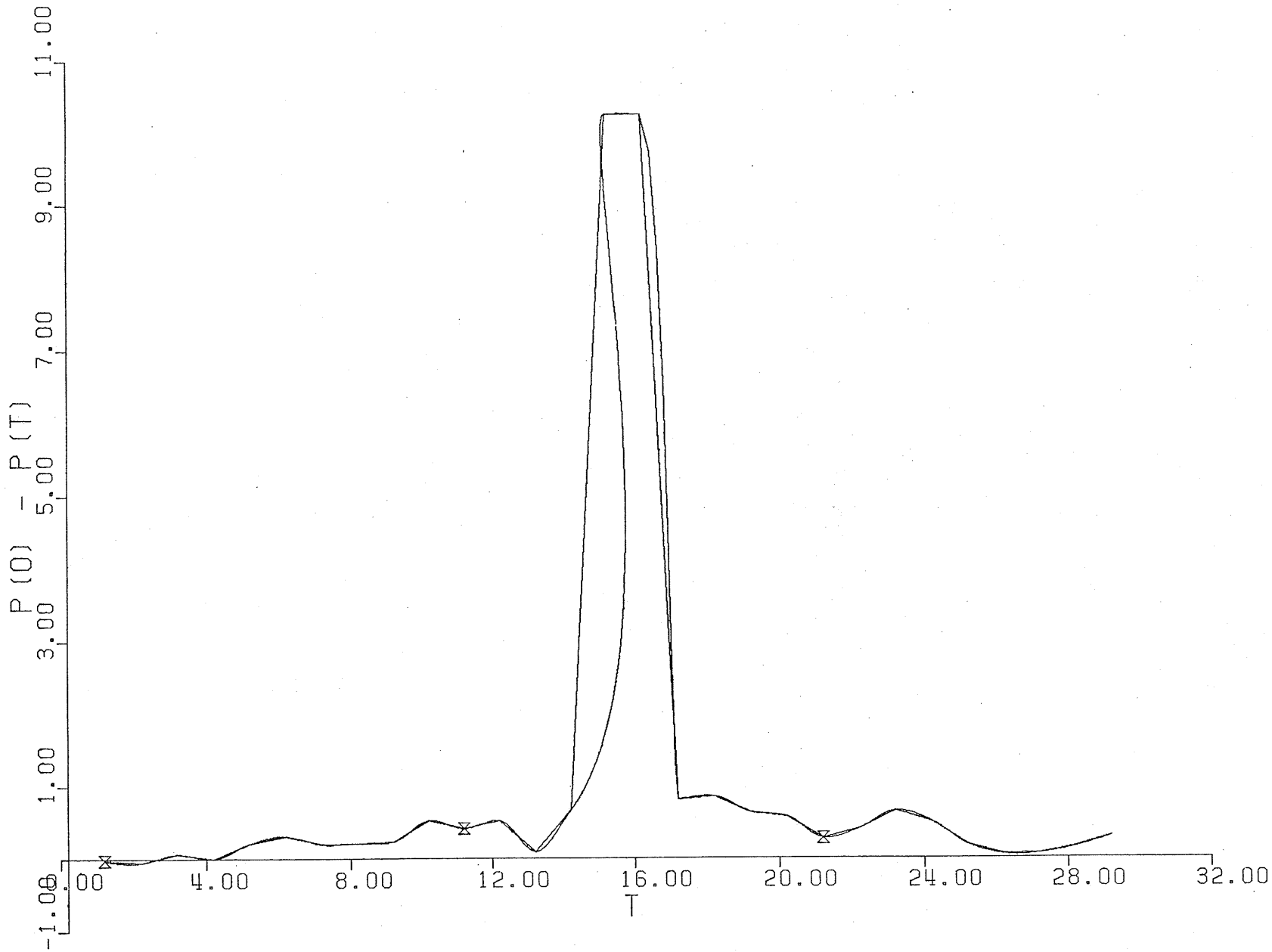

P(O) - P(T)
11.00
9.00
7.00
5.00
3.00
1.00
-1.00
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T

Figure 67. P plot of P CD system in time domain. All 29 daily sessions were considered continuous time function. Curve showed system's adjustment to levels of demand as damped oscillation.

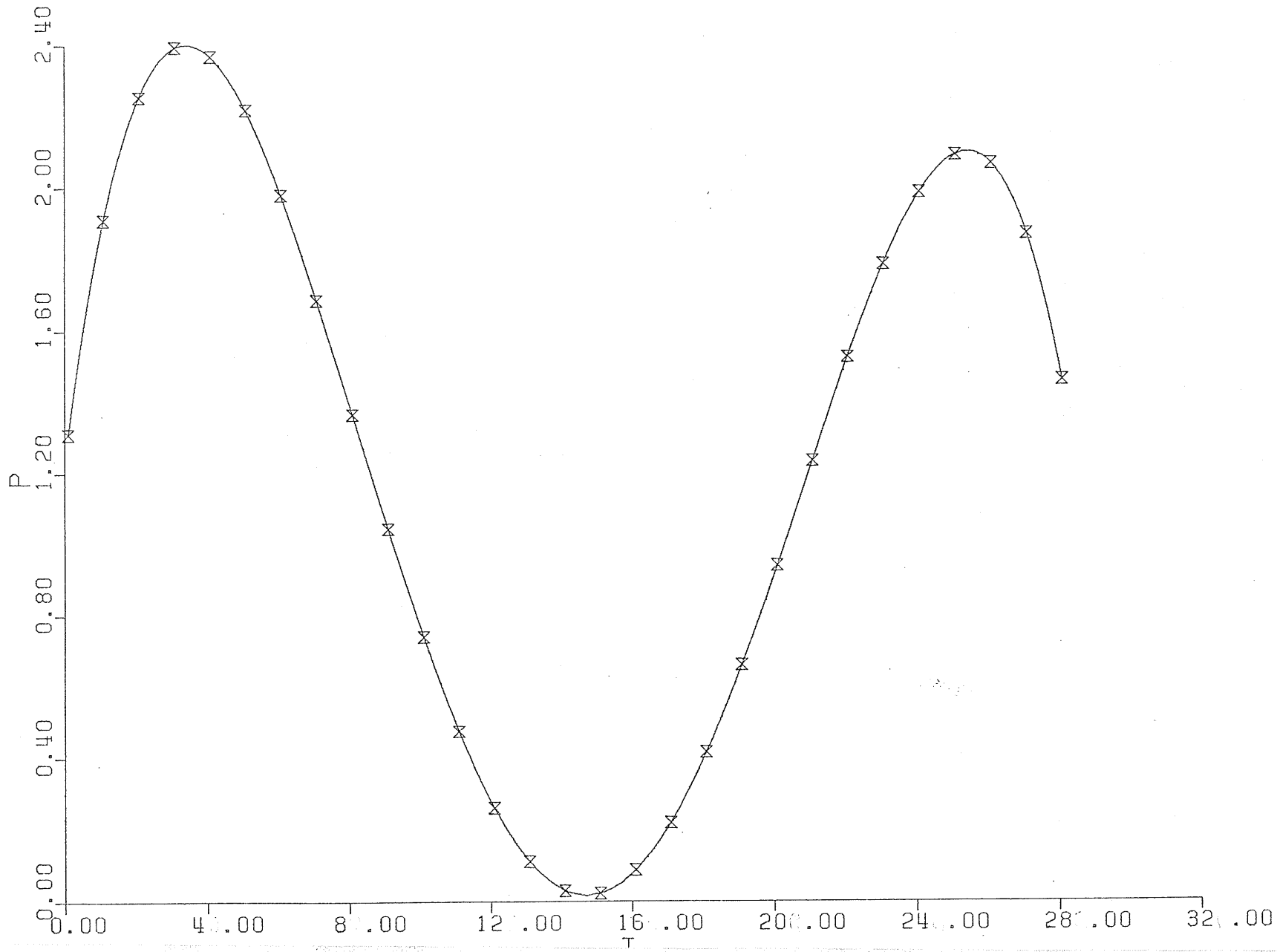

P
0.00
0.40
0.80
1.20
1.60
2.00
2.40
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T

Figure 68. P plot of P CD system prior to lesioning.

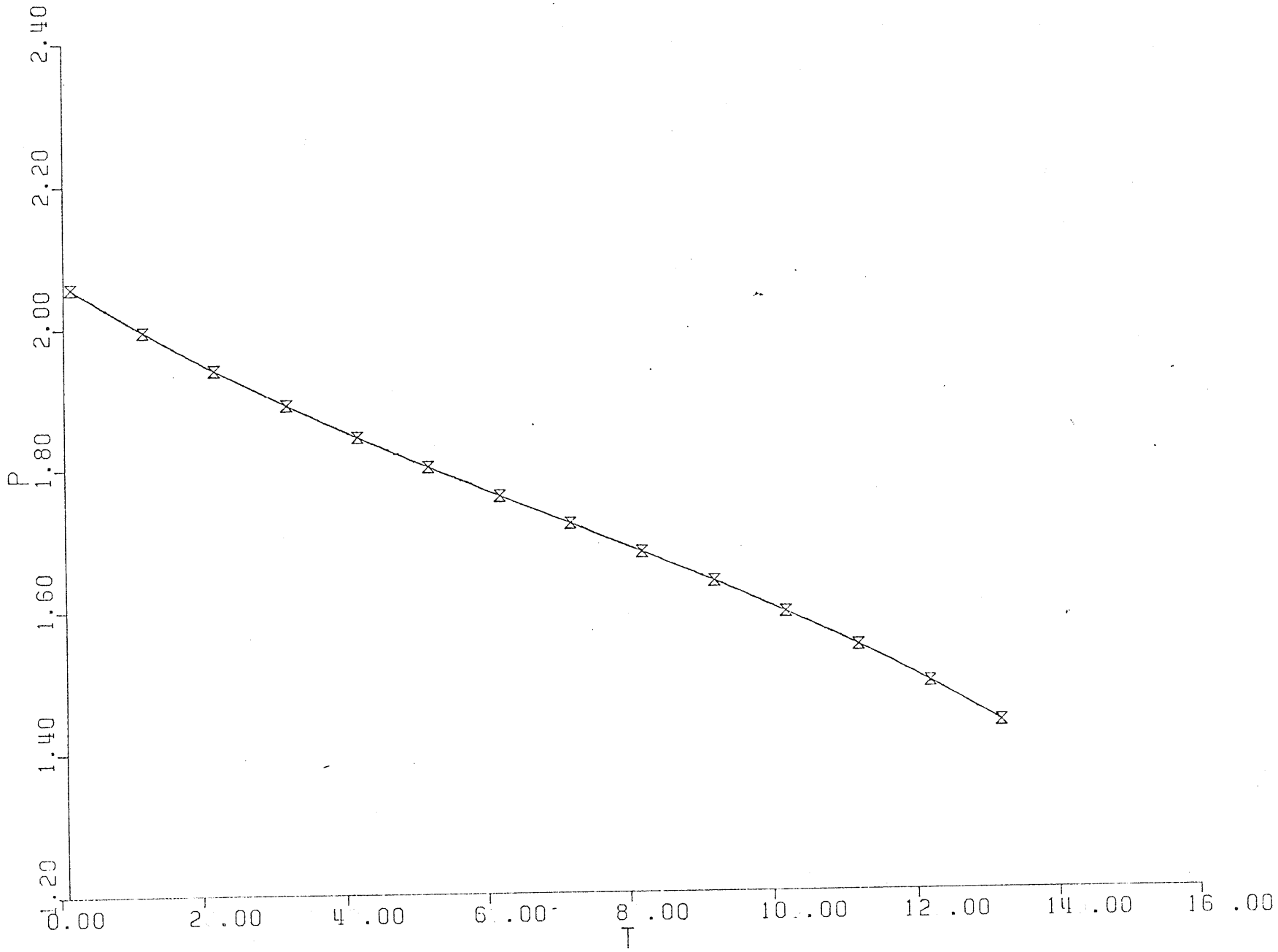

P
2.40
2.20
2.00
1.80
1.60
1.40
1.20
0.00
2.00
4.00
6.00
8.00
10.00
12.00
14.00
16.00
T

Figure 69. P plot of P CD system subsequent to lesioning.

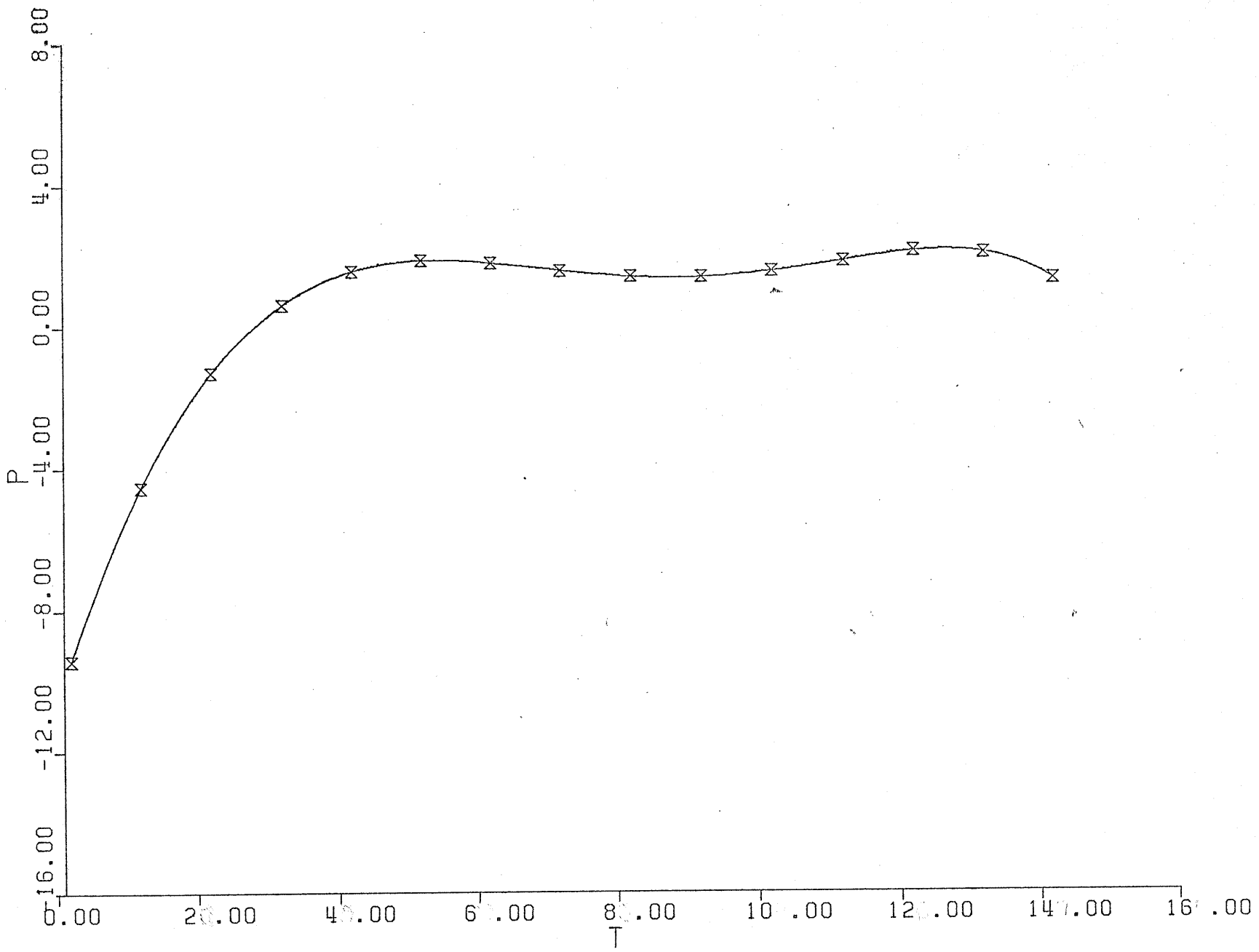

8.00
4.00
0.00
-4.00
-8.00
-12.00
16.00
P
0.00
T

Figure 70. Phase plane representation of the P CD system .All performance on DCA were evaluated as continuous. System functioned toward a reduced P when put on I/O control but evolved away from the control was subsequently removed. Adjustment of system to changes in demand observed.

3.20
2.80
2.40
2.00
1.60
1/P
1.20
0.80
0.40
0.00
-1.20
-0.80
-0.40
0.00
0.40
0.80
1.20
DP/DT

Figure 71. Phase diagram of P CD after bilateral electrolytic lesion. System showed a transient overshoot before precipitated inot a limit cycle. System showed adjustment to removal of I/C control in moving away from the limit cycle.

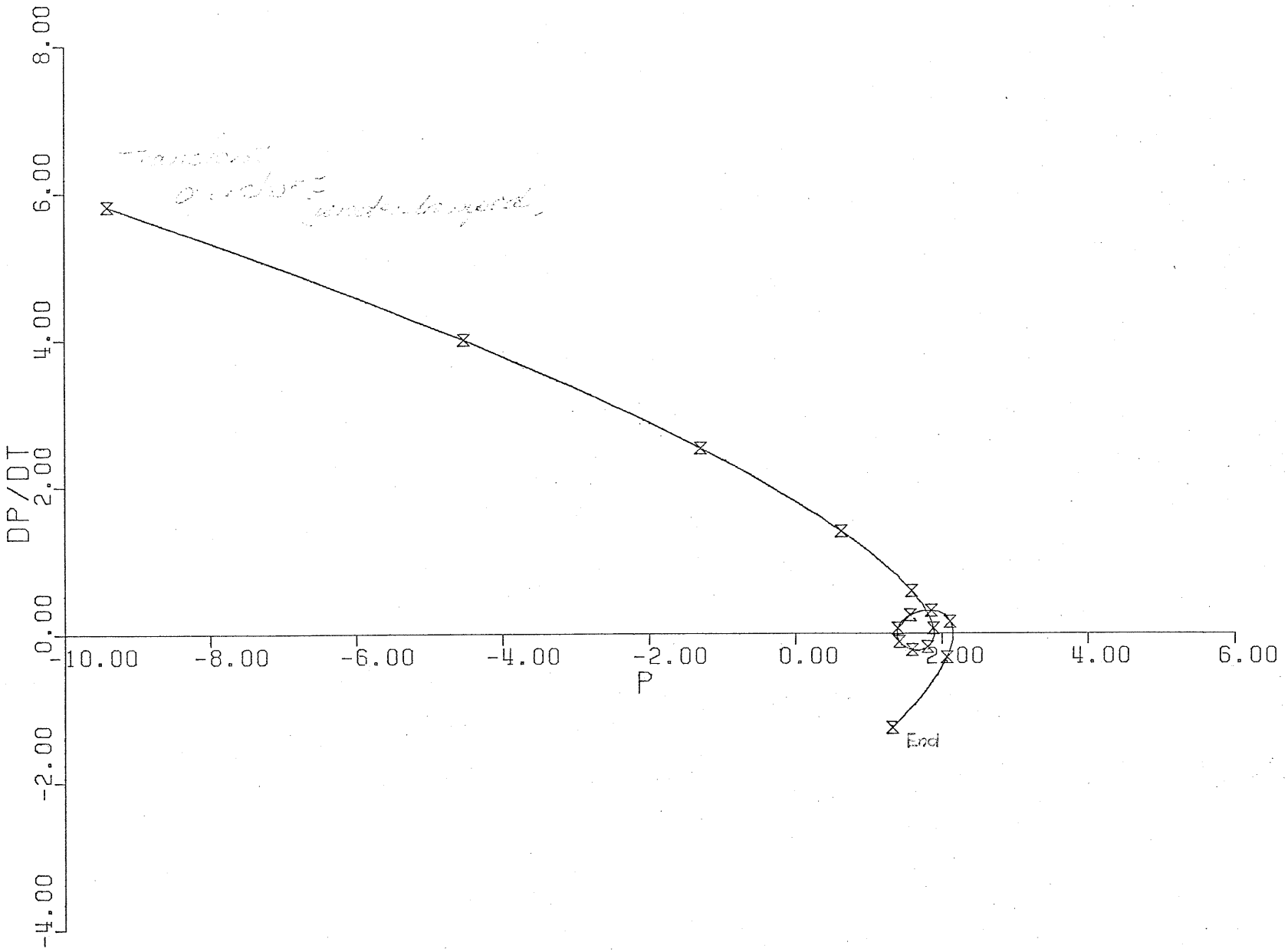

8.00
6.00
4.00
2.00
0.00
-2.00
-4.00
DP/DT
-10.00
-8.00
-6.00
-4.00
-2.00
0.00
2.00
4.00
6.00
P
End

Figure 72. Phase diagram of P CD prior to lesioning.

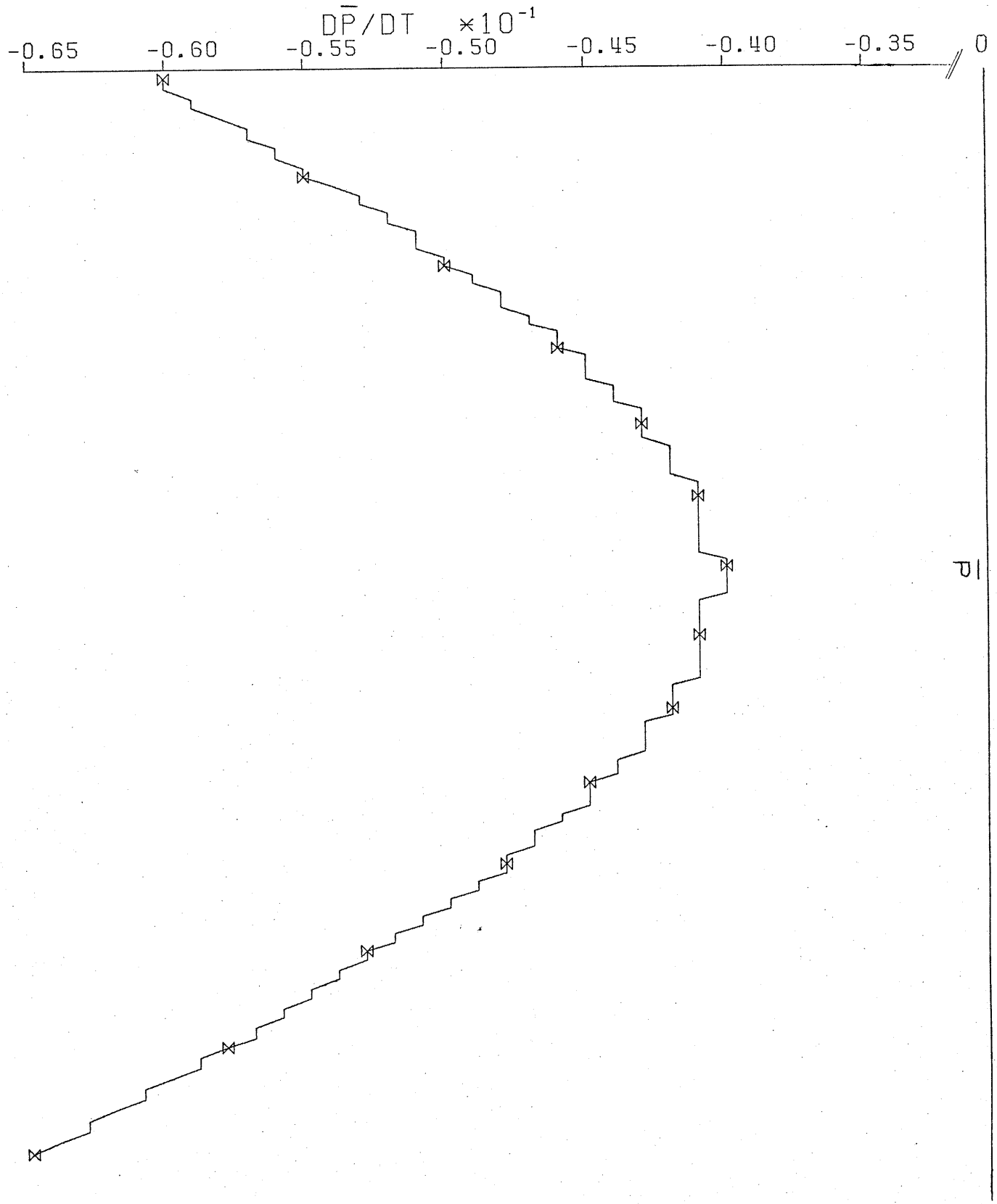
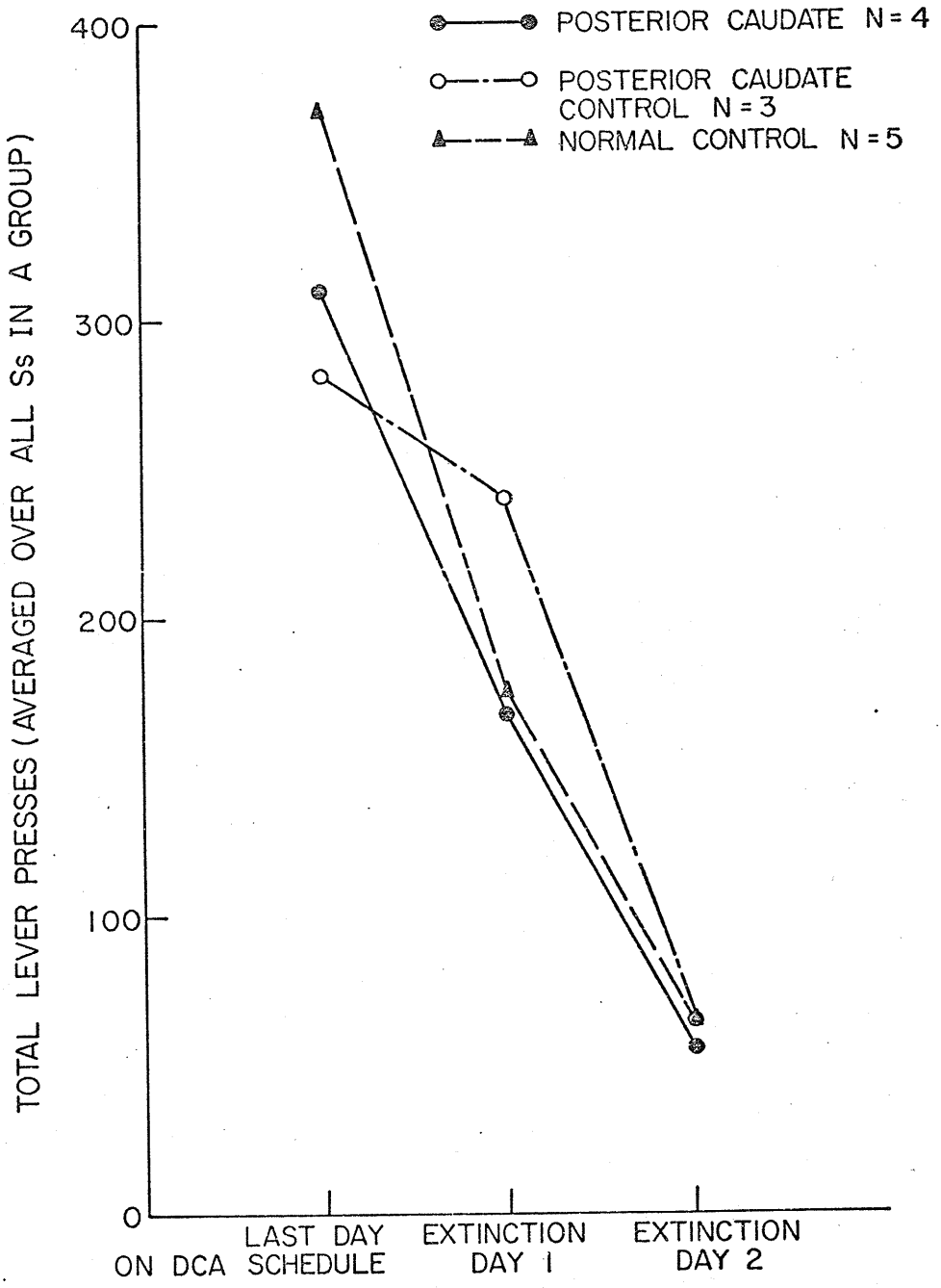

$D\bar{P}/DT \quad *10^{-1}$
-0.65
-0.60
-0.55
-0.50
-0.45
-0.40
-0.35
0
IR

Figure 73. Extinction curves of the P CD, PCD C and N to DCA.


POSTERIOR CAUDATE N = 4
POSTERIOR CAUDATE CONTROL N = 3
NORMAL CONTROL N = 5
TOTAL LEVER PRESSES (AVERAGED OVER ALL Ss IN A GROUP)
400
300
200
100
0
LAST DAY ON DCA SCHEDULE
EXTINCTION DAY 1
EXTINCTION DAY 2

GROUP VIII the group with sham lesion in the posterior aspect of the caudate nucleus

A.general observation:The pre-operative and post-operative behaviour of the Ss with sham lesions did not differ in any observable manner. Other than signs of slight irritation due to the autoclips on the forehead, the Ss appeared normal.

B. activity level in the rotational running wheel-:As shown in figure 63, the post-operative activity in the running wheel was essentially the same as that of the pre-operative data.

C.performance on the DCA:

(1)Figure 74 presented the mean ofa)total lever press b)reinforced lever press,c)penalty count, and d) trial abort count of the P CD C group. Data from the 29 daily sessions were grouped into six time blocks. The behavioural data displayed in this graph resumbled closely those of the other operated control groups.

The mean response latencies of the P CD C on the DCA with and without the I/O control is presented in figure 75. Of the pre-I/O baseline performance,the system stabilized around 5" of delay. With the I/O control input, the system was driven over time to perform at a longer response latencies. Post-operatively the same type of behavioural pattern is displayed. The data

points on the graph showed the oscillatory type of performance on tha I/O control. Mean latency of response returned to that of the pre-operative pre-I/O control baseline upon the removal of the I/O control. The gain of the system is seen in the increase in the mean of the total number of reinforced lever presses and reduction in the total penalty and trial abort counts (figure 74).

(3) P(0)-P(T) vs T: The difference plot of this group is similar to the other operated control(figure 76).

(4) P plot:The response of the P CD C on the DCA in the time domain is presented in figure 77. When the 29 daily sessions were fitted via weighted least square polynomial,the post-operative section of the data is a mirror image of its pre-operative section. When the pre- and post-operative sections were presented as separate data sets (figures 78 & 79), similar results were obtained.

(5) the phase-plane representation (DP/DT / P):

Figure 82 is the phase diagram of the system before it was subjected to the sham lesion. Before the I/O control was superimposed the system stabilized around the P axis with a relative error close to 2.1. As the I/O control was applied the system was driven to function with a much reduced error term. the trajectory told of a system which travelled across the phase plane with a

predominating force of extinction during the middle section of the system's 'attempt' to stabilize on a new demand. The later phase of the trajectory showed that the force of reconditioning had entered at this stage of the system's active adjustment. Figure 80 is the phase diagram of the system post-operatively.Initially while on the I/O control, the system exhibited a relatively stable performance with a balanced processes of extinction and reconditioning at work. The phase diagram is a limit cycle with the focus around the P axis. Upon the removal of the I/O control, the system began to show decay of the DCA on I/O control. During this phase the force of reconditioning (shorter response lat- ency) became predominating, and the system showed a rapid swing to increased relative error over time. When the P CD C is considered as a whole the phase diagram is that of a active servomechanis- like system which traversed acrossed the behavioural phase plane in the form of a limit cycle (figure 81).

The extinction data presented in figure 73 showed that the P CD C showd the same trend on the extinction phase of the experiment.

Figure 74. Mean of a) total lever press, b) reinforced lever press , c) penalty count and d) trial abort count of Ss in the P CD C group. Values were means of each daily sessions ,averaged over all Ss in each time block.

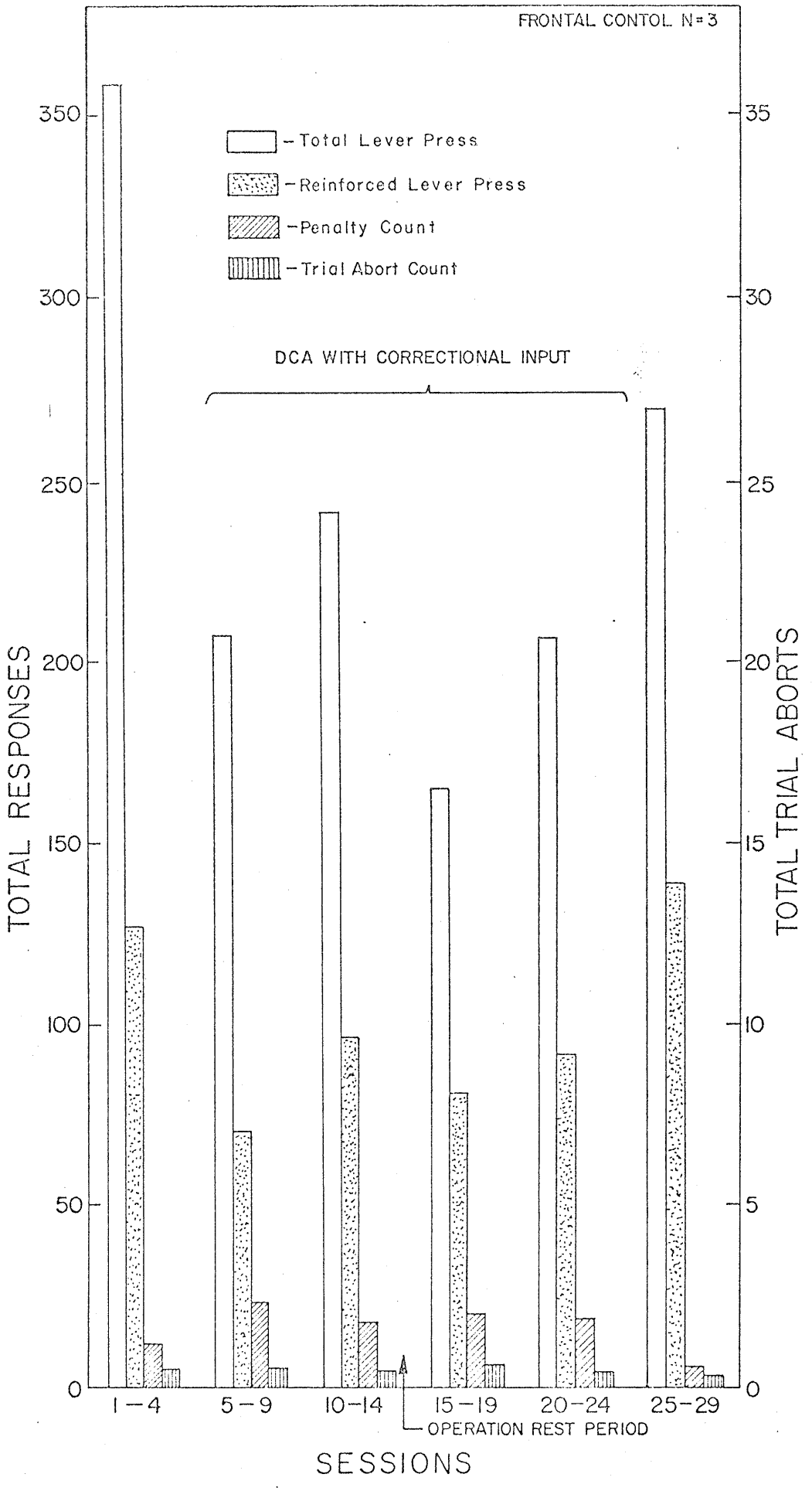

FRONTAL CONTOL N=3
Total Lever Press
Reinforced Lever Press
Penalty Count
Trial Abort Count
DCA WITH CORRECTIONAL INPUT
TOTAL RESPONSES
TOTAL TRIAL ABORTS
350
300
250
200
150
100
50
0
35
30
25
20
15
10
5
0
1–4
5–9
10–14
15–19
20–24
25–29
OPERATION REST PERIOD
SESSIONS

Figure 75. The mean response latencies of the P CD C group. Corresponding curve was the first best fit via the weighted least square polynomial.

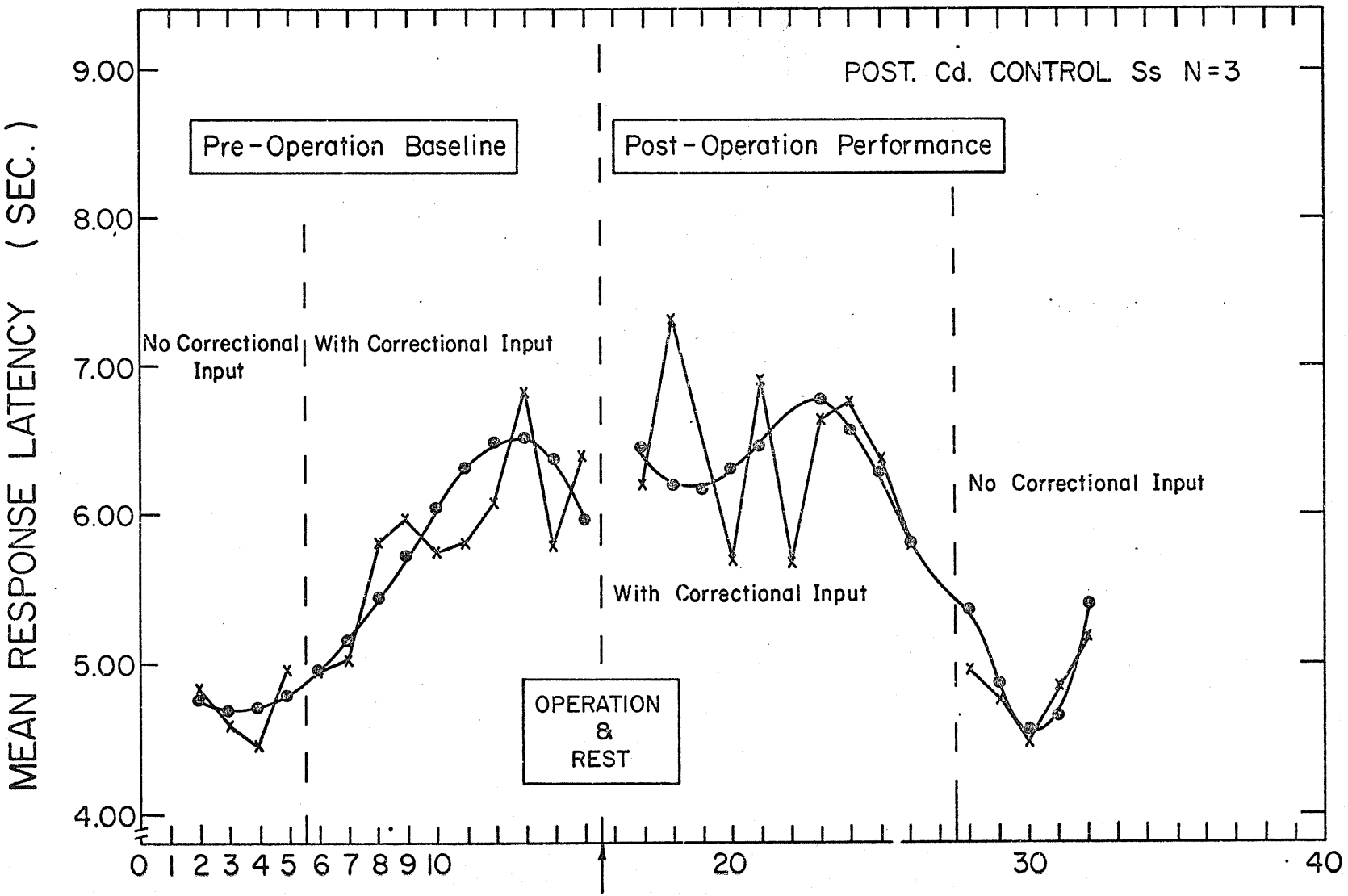

POST. Cd. CONTROL Ss N=3
Pre-Operation Baseline
Post-Operation Performance
No Correctional Input
With Correctional Input
With Correctional Input
No Correctional Input
OPERATION & REST
MEAN RESPONSE LATENCY (SEC.)
9.00
8.00
7.00
6.00
5.00
4.00
0 1 2 3 4 5 6 7 8 9 10
20
30
40

Figure 76. The difference plot of the P CD C group.

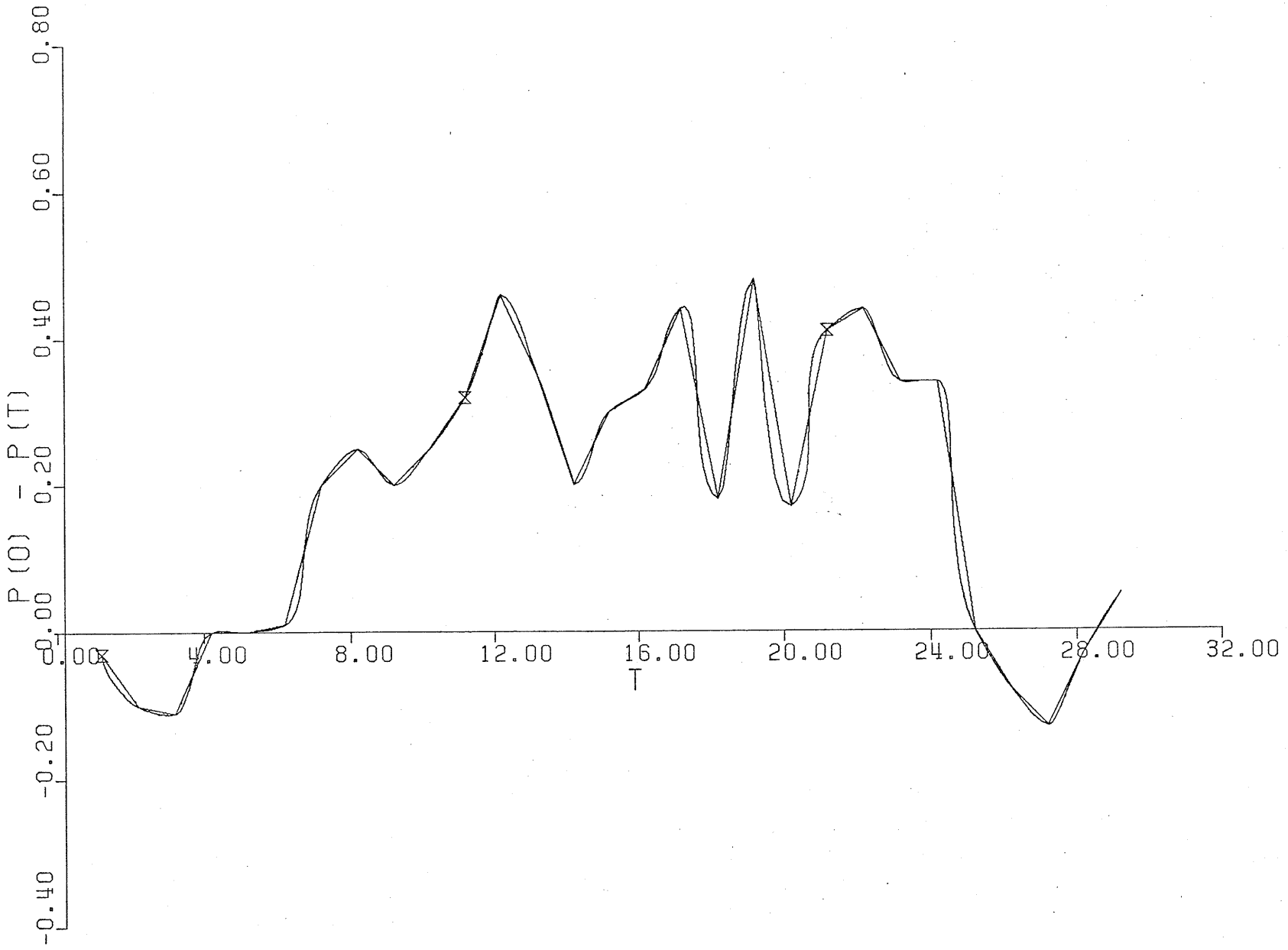

0.80
0.60
0.40
0.20
0.00
-0.20
-0.40
P(0) - P(T)
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T

Figure 77. P plot of P CD C before and after the sham lesion. Performances before and after sham lesion were mirror images of each other.

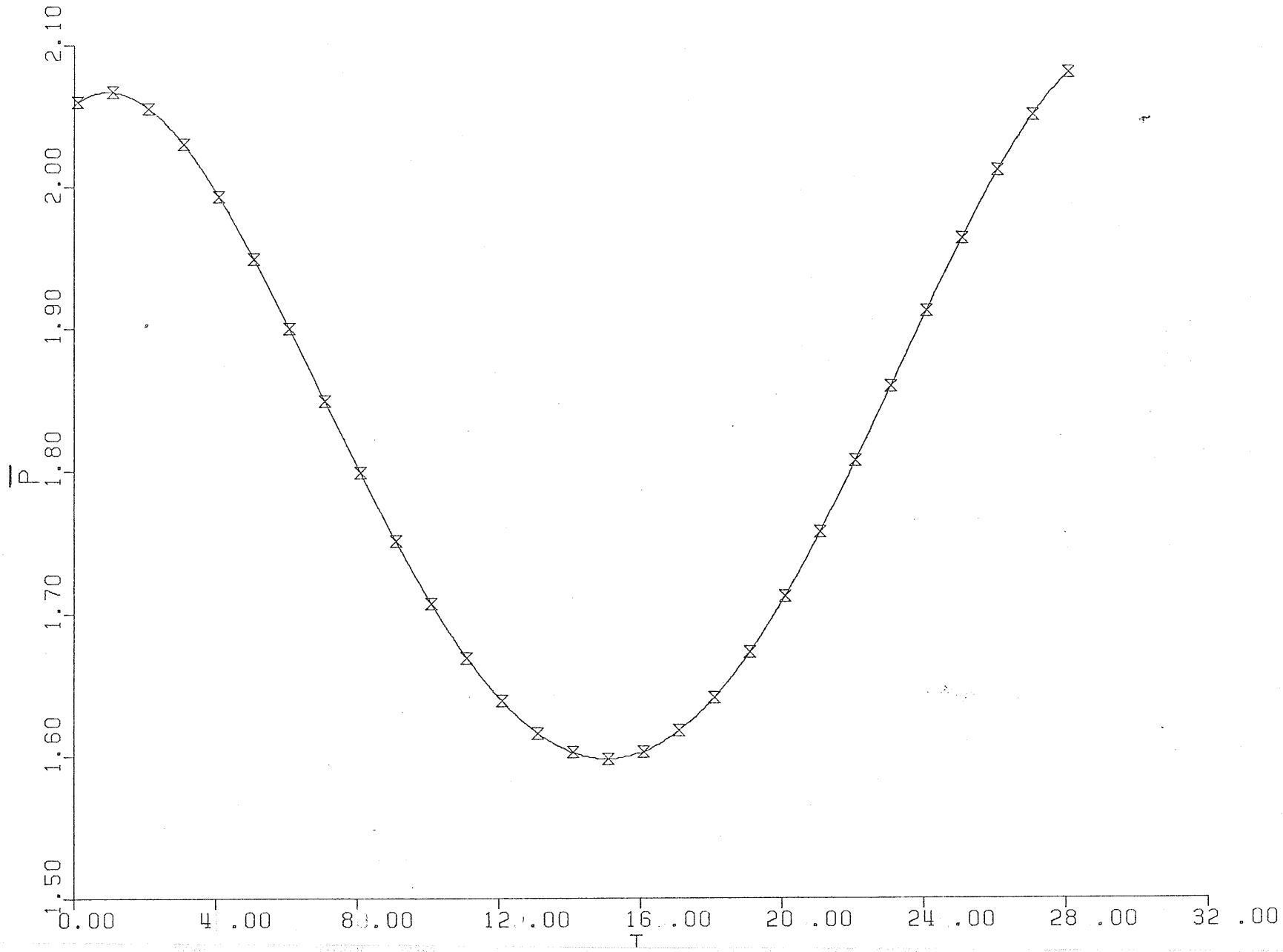

2.10
2.00
1.90
1.80
1.70
1.60
1.50
$\overline{P}$
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T

Figure 78. P plot of P CL C prior to sham lesion. System showed adjustment to changes in demand.

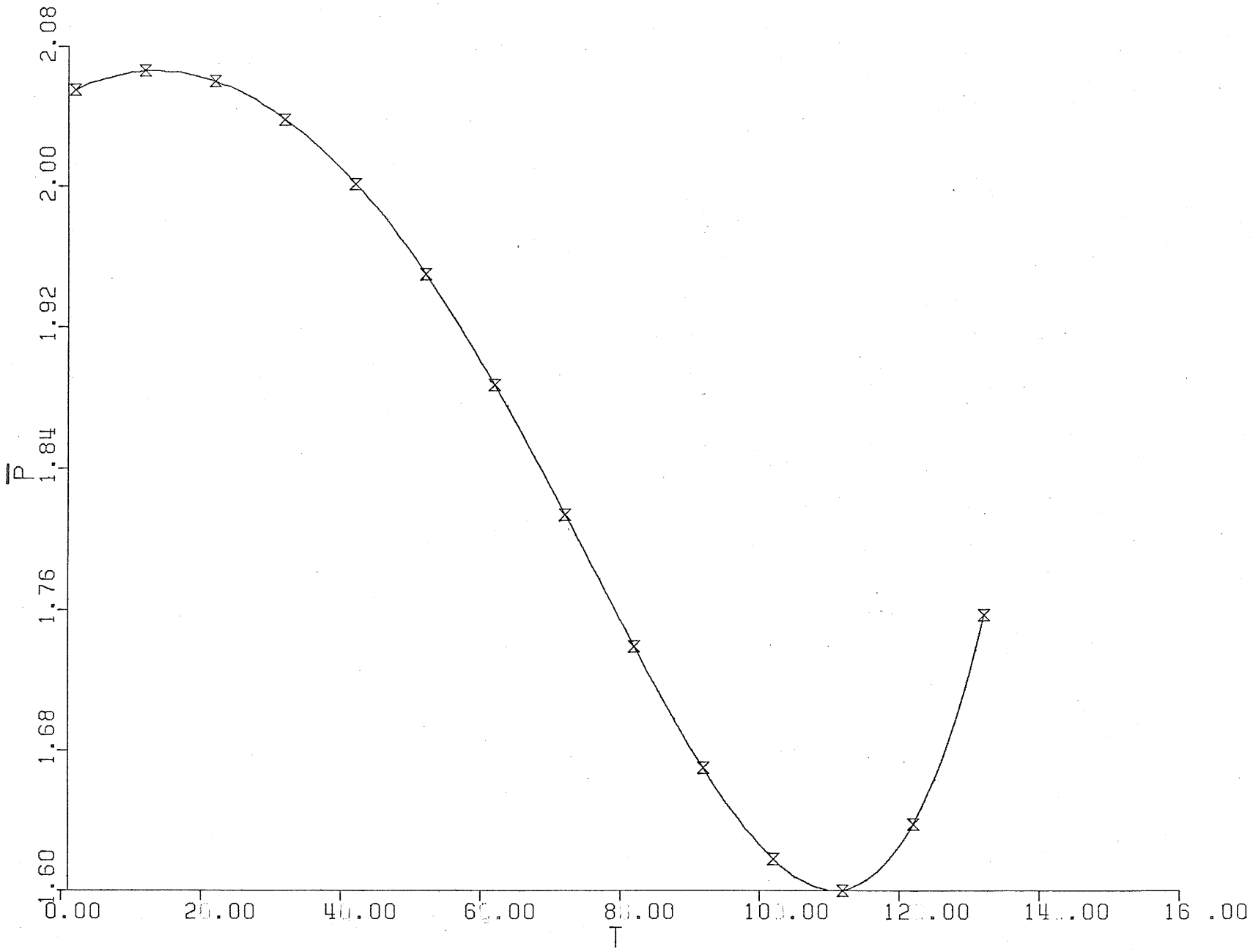

2.08
2.00
1.92
1.84
1.76
1.68
1.60
$\overline{P}$
0.00
20.00
40.00
60.00
80.00
100.00
120.00
140.00
160.00
T

Figure 79. P plot of P CD C subsequent to sham lesion.

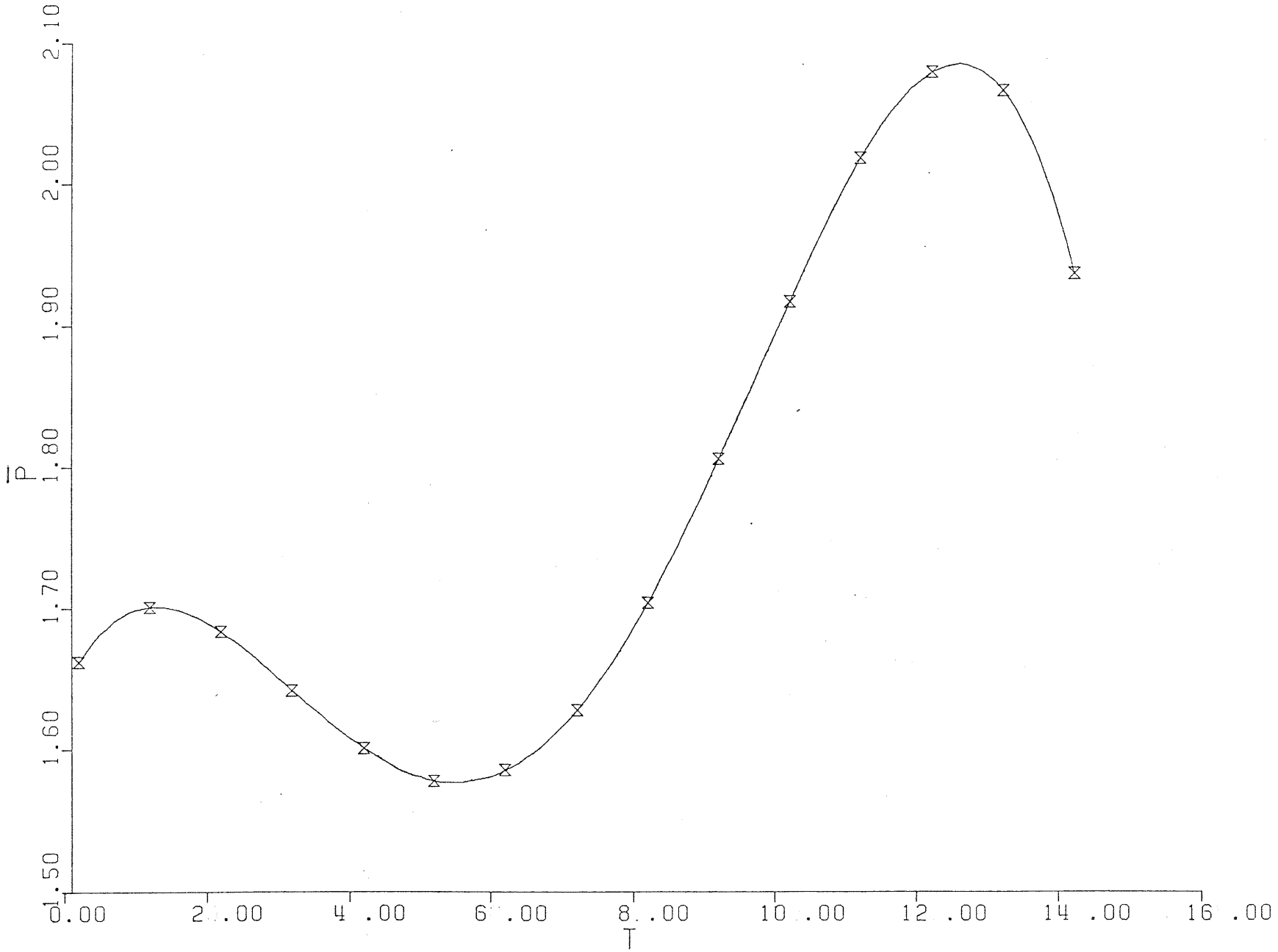

2.10
2.00
1.90
1.80
1.70
1.60
1.50
$\overline{P}$
0.00
2.00
4.00
6.00
8.00
10.00
12.00
14.00
16.00
T

Figure 80. Phase diagram cf P CD C system prior to sham lesion. System was driven to performance around limit cycle by I/O contrcl.

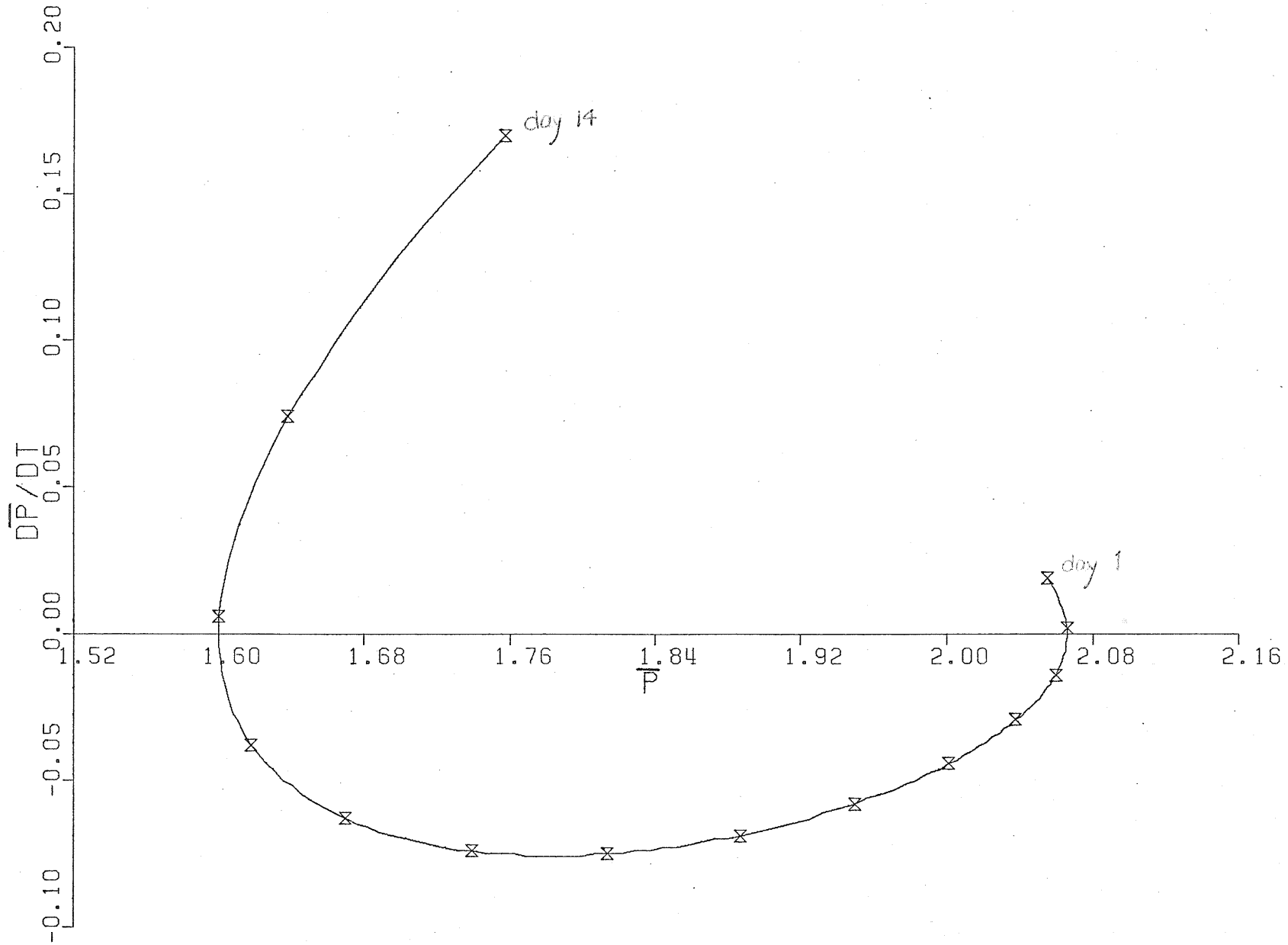
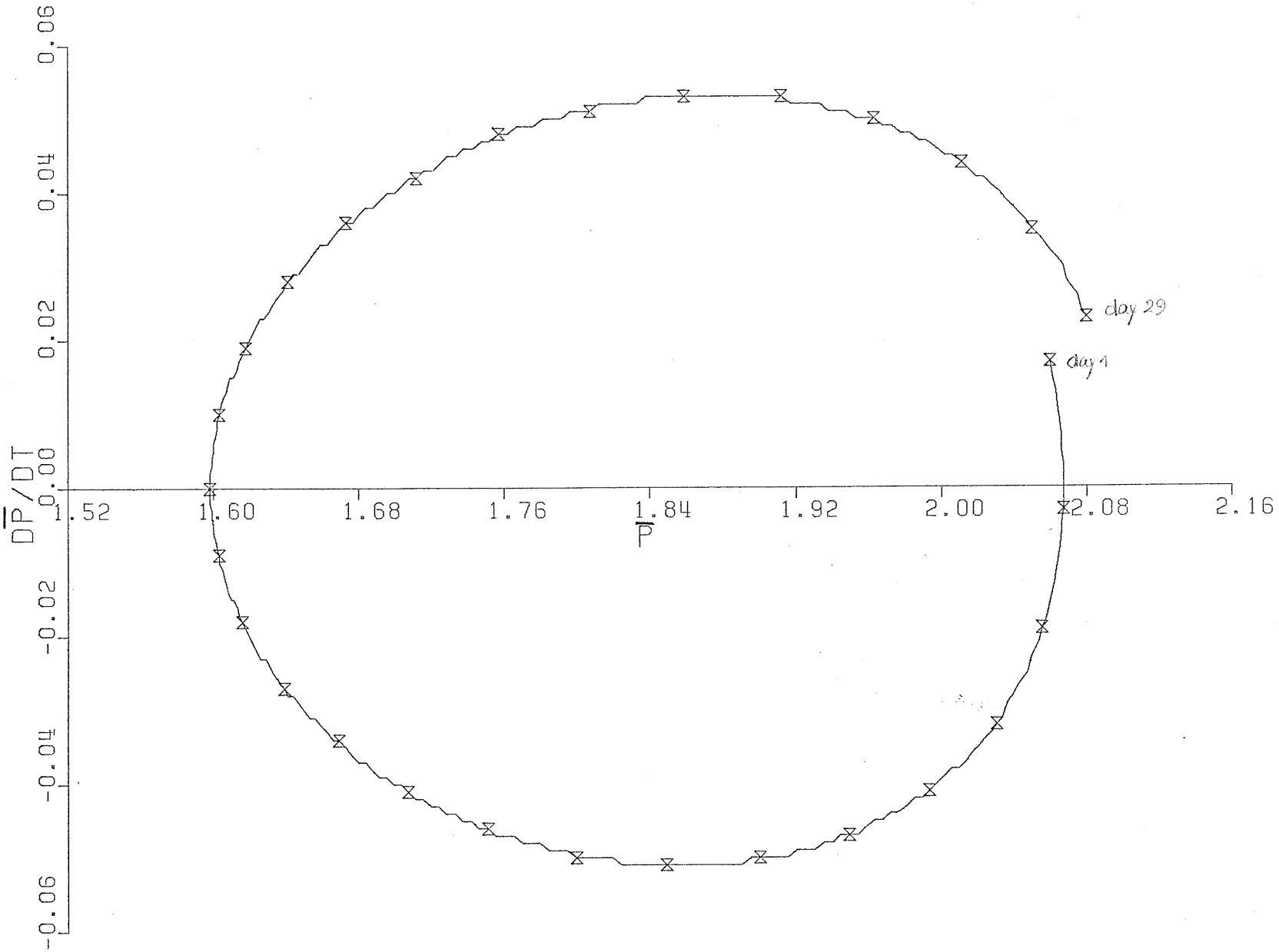

day 14
day 1
0.20
0.15
0.10
0.05
0.00
-0.05
-0.10
DP/DT
1.52
1.60
1.68
1.76
1.84
1.92
2.00
2.08
2.16
P

Figure 81. Phase diagram of P CD C system after sham lesion. Similar behaviour observed as before sham lesion.

2.16
2.08
2.00
1.92
1.84
1.76
1.68
1.60
1.52
1/P
End
-0.24
-0.16
-0.08
0.00
0.08
0.16
0.24
DP/DT

. Fig.82 Phase plan representation of P CD C system when all behavioural data on DCA were evaluated as continuous in time. Trajectory told of a system which showed balanced forces between extinction and reconditioning on the DCA.


0.06
0.04
0.02
0.00
-0.02
-0.04
-0.06
DP/DT
1.52
1.60
1.68
1.76
1.84
1.92
2.00
2.08
2.16
P
day 29
day 1

GROUP VIIII the normal control group (N)

A. general observation:

The general behaviour of the normal control Ss varied little throughout the different phases of the exper- iment. During the 19 days while the experimental groups underwent surgery,the Ss in N were put on rest with food and water ad.lib. While all Ss showed weight gain during this period,the body weights returned to the pre-rest level via 3 days of 23-hour water depriva tion prior to the onset of the post-rest testing phase.

B. activity measured by the rotational running wheel:

Based on figure 63,the Ss in the normal group maintained a uniformed level of activity through the 8 daily sessions.

C.performance on the DCA:

(1)Figure 84 presents the mean of a) total number of lever press, b) total reinforced lever press, c) penalty count , & d) trial abort count of the 29 daily sessions. For clarity data were grouped into six time blocks. The mean of total lever press and of reinforced lever press were the hightet during the final time block, while the penalty count was the lowest during the same block. This shows the 'gain' of the behavioural system via its experience on the DCA with the super- imposed I/O control. The I/O control reduced the mean of the total

lever press per session. (The same general phenomena were reported by results from the other 8 groups).

(2) The mean response latencies of the N on the DCA with and without the I/O control is presented in figure 85. Based on this figure the response latency of the system prior to the implement of the I/O control stabilized around 5" of delay. the system was driven to respond at a level around the 7" range when the I/O control was in effect. The system responded at the same general level after the rest period, suggesting that DCA showed little deterioration over time. Performance then returned to a level close to the original baseline latency when the superimposed I/O control was removed.

(3) P(0)-P(T) vs T: Figure 86 showed that the gain of the functioning system was the greatest when the I/O control was in effect.

(4) P plot: Figure 87 is the time response of the DCA system. It showed that the performance of the system was linear over the 20 daily sessions when the superimposed I/O was in effect. The behaviour of the system departed from the original level of baseline performance, showing reduction in the P values. The system then returned to the performance level com parable to that of the original baseline upon subsequent removal of the I/O control. This characteristic manifested the servomechanism- like behaviour of the DCA system. Figures 88 & 89 are the P

plots of the N group before and after the rest period. These graphs exhibited similar behavioural patterns and are a mirror images of each other. Its basic form of behaviour on either level of demand over time is that of a damped oscillation.

(5) DP/DT vs P:Figures 90 & 91 are the phase diagram of the N before and after the rest. As shown in figure 90,the trajectory of the system moved toward a reduced relative error with the inplementation of the I/O control. While on the DCA with I/O control, the system showed reduction both in the relative error and the rate of change of the relative error The control command effectively constricted the behavioural system to a more 'stable ' performance level over time and with a reduced relative error term. The system adjusted to the changed demand in a limit cycle around the P axis. The same limit cycle was maintained after the period of rest (figure 91). The system then moved away from the limit cycle when the superimposed I/O control was removed. When the behavioural system cn the DCA before and after the rest was considered as a continuum in the time domain (feasiable as the system shcwed virtually no changes in its performance over the period of rest) the DCA adjusted to the changes in the level of demand in the form of a very tightly knit limit cycle,showing virtually no changes in the values cf DP/DT over time. The values spiralled closely around the focus which was on the P

axis (figure 92). The system then moved anay from the L.C. when the I/O control was removed. That the system showed the tendency to stabilize at two different loci in the state- space of behavioural phase plane suggests the noninvariance of the governing equations at the two different levels of the reinforcement contingencies of the DCA paradigm.

(6) As shown in figure 73, the extinction curve of the N group was similar to those of the other groups.

Figure 84. The means of a) total lever press, b) reinforced lever press ,c) penalty count and d) trial abort count in each session. of all Ss from a particular seSsion. Data were then grouped into six time blocks.

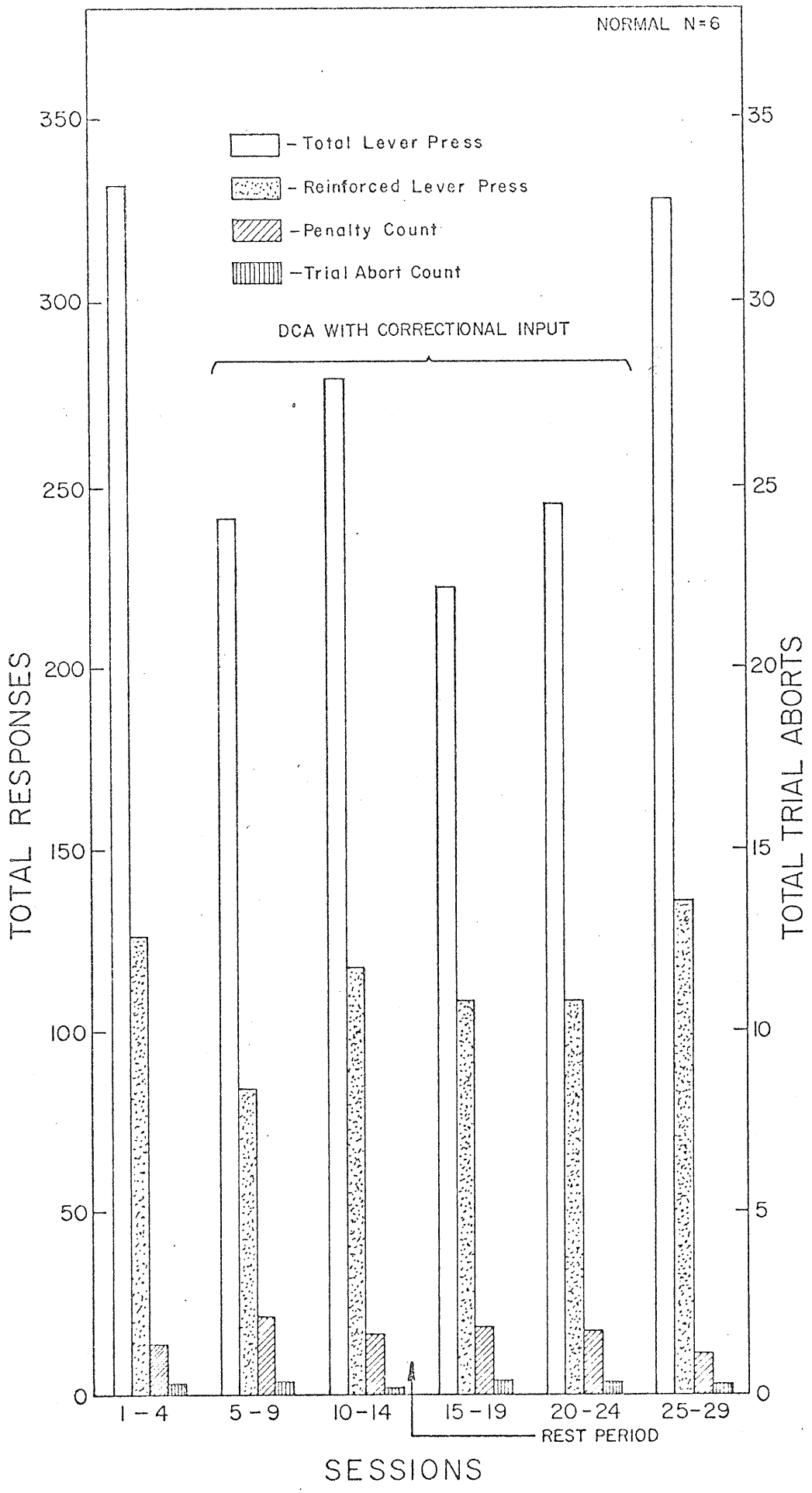

NORMAL N=6
- Total Lever Press
- Reinforced Lever Press
- Penalty Count
- Trial Abort Count
DCA WITH CORRECTIONAL INPUT
TOTAL RESPONSES
TOTAL TRIAL ABORTS
0
50
100
150
200
250
300
350
0
5
10
15
20
25
30
35
1 - 4
5 - 9
10 - 14
15 - 19
20 - 24
25 - 29
REST PERIOD
SESSIONS

Figure 85. The mean response latencies of the normal group on DCA and DCA with I/O control/ Each data point was a mean of 40 daily trials, averaged over all ss. The corresponding curve was the first best fit via the weighted least square polynomial.

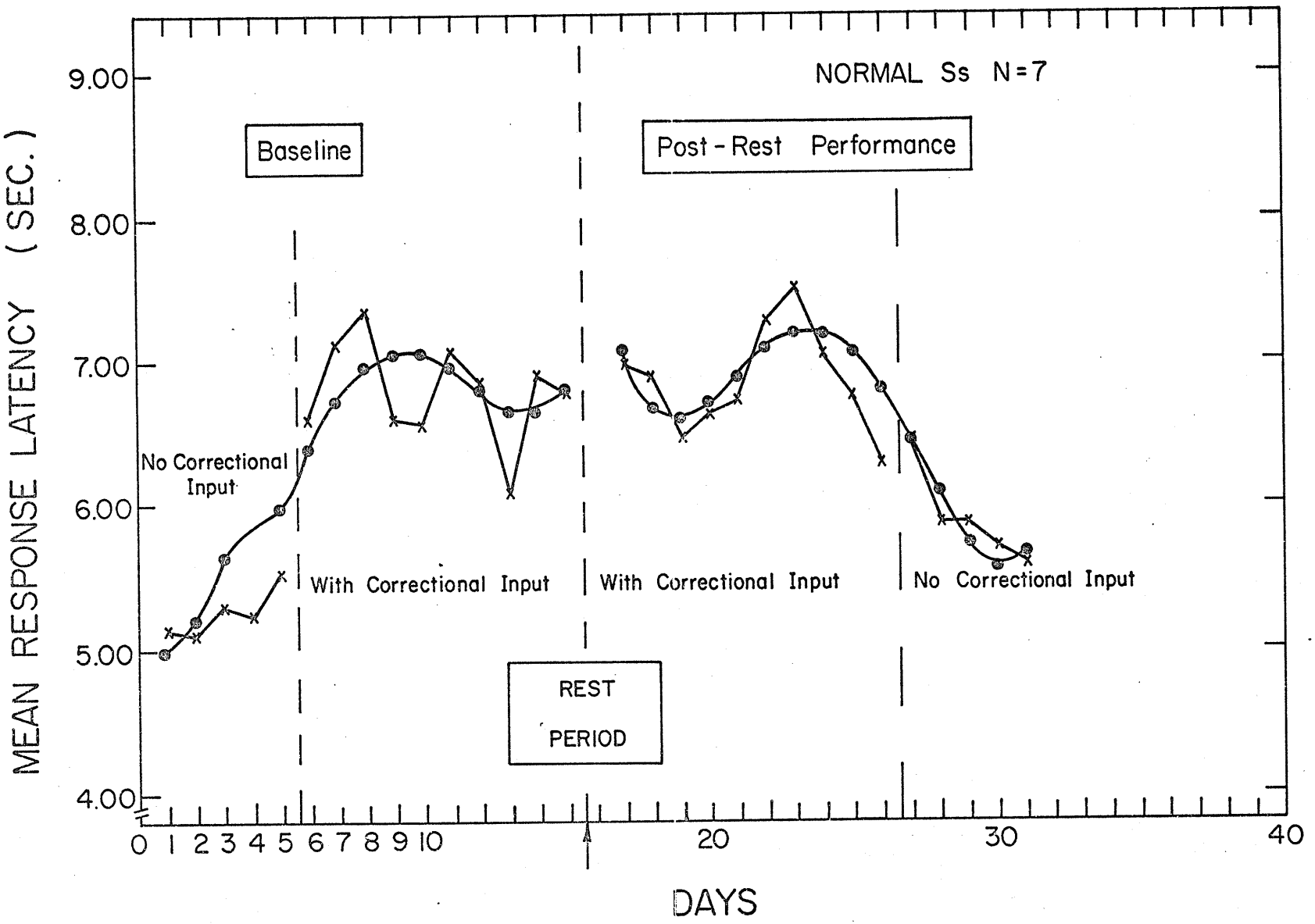

NORMAL Ss N=7
Baseline
Post-Rest Performance
MEAN RESPONSE LATENCY (SEC.)
9.00
8.00
7.00
6.00
5.00
4.00
No Correctional Input
With Correctional Input
With Correctional Input
No Correctional Input
REST PERIOD
0 1 2 3 4 5 6 7 8 9 10
20
30
40
DAYS

Figure 86. The difference plot P(0)-P(T) of the normal group. The magnitude of the difference is a function of the reinforcement contingencies.

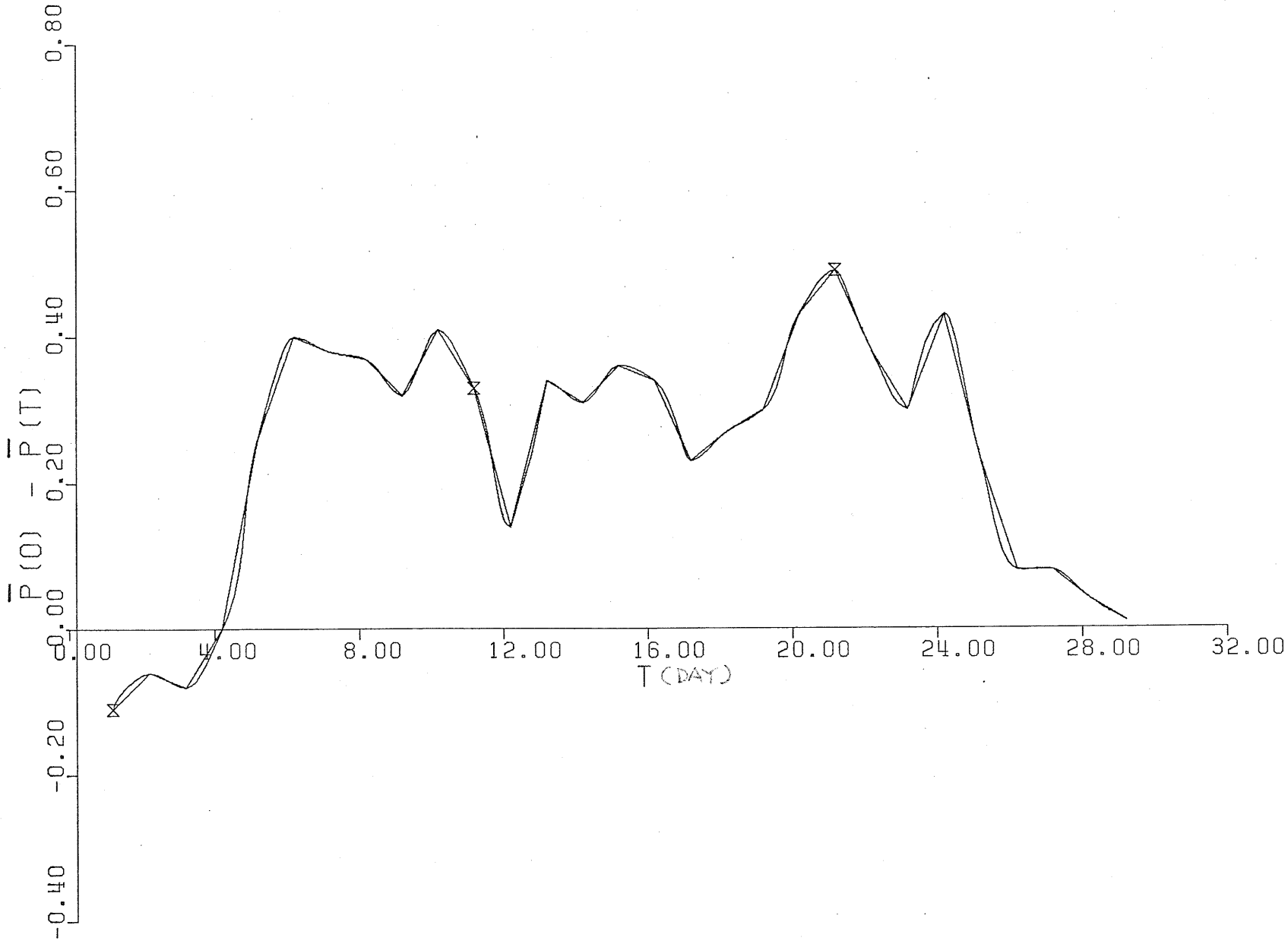

0.80
0.60
0.40
0.20
0.00
-0.20
-0.40
$\bar{P}(0) - \bar{P}(T)$
0.00
4.00
8.00
12.00
16.00
20.00
24.00
28.00
32.00
T (DAY)

Figure 87. P plot of the normal system.

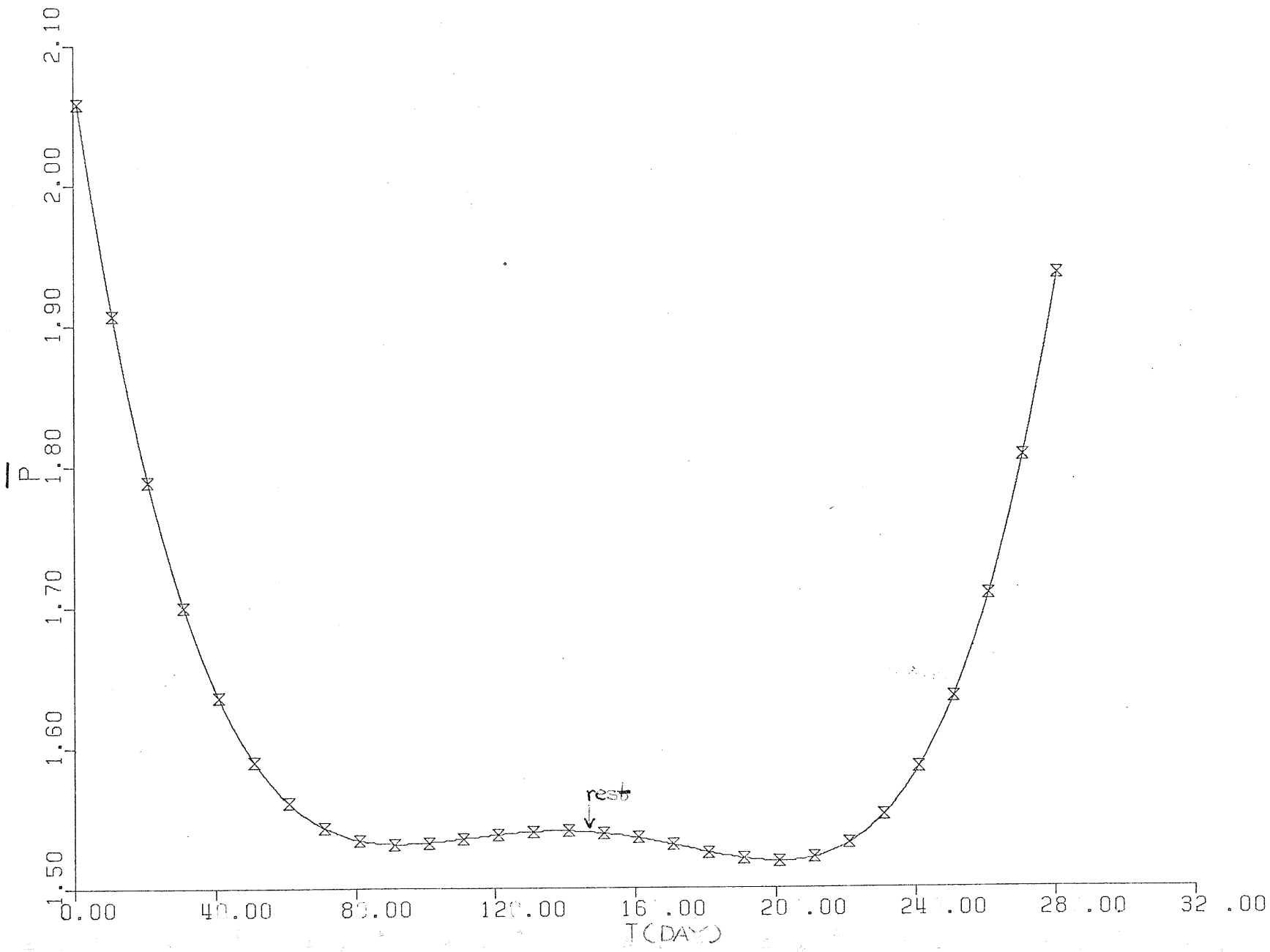

2.10
2.00
1.90
1.80
1.70
1.60
1.50
P
rest
0.00
40.00
80.00
120.00
160.00
200.00
240.00
280.00
320.00
T(DAY)

Figure 88. P plot of the normal system prior to rest period.

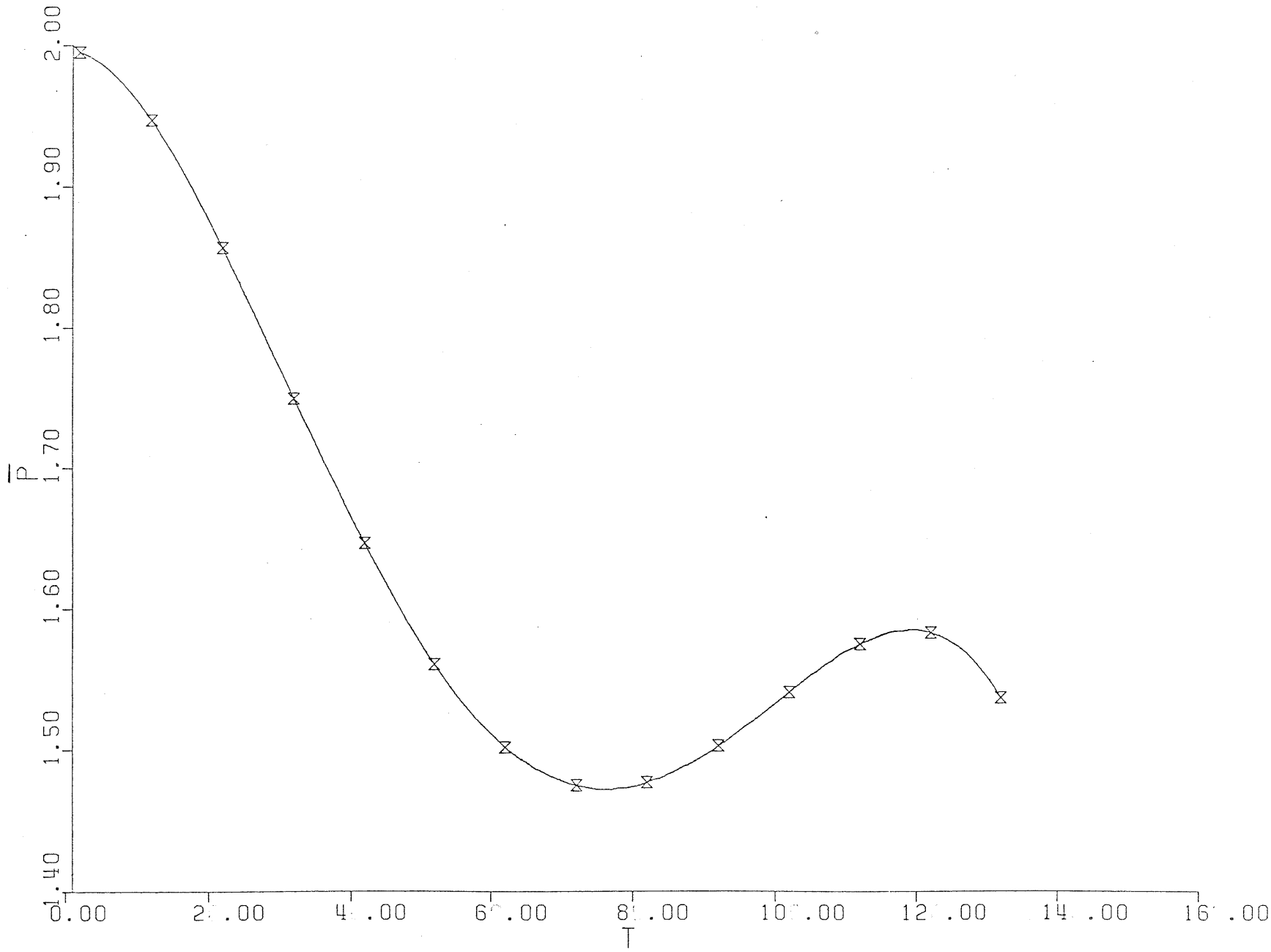

2.00
1.90
1.80
1.70
1.60
1.50
1.40
$\overline{P}$
0.00
2.00
4.00
6.00
8.00
10.00
12.00
14.00
16.00
T

Figure 89. P plot of the normal system after the rest period.

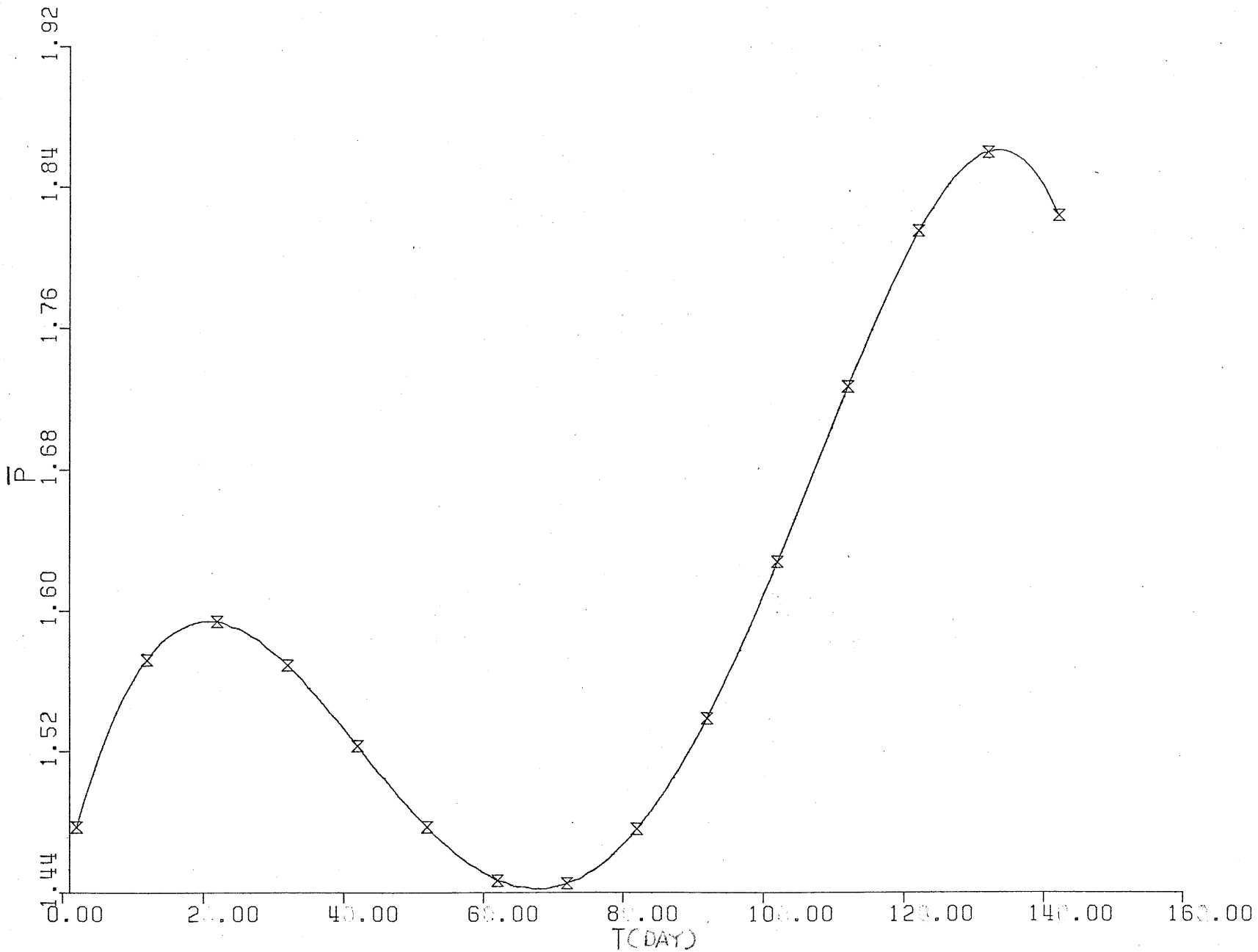

1.92
1.84
1.76
1.68
1.60
1.52
1.44
$\overline{P}$
0.00
40.00
T(DAY)

Figure 90. Phase diagram of the normal prior to rest period. System driven to reduced P's ,functioning around $\bar{P}$ axis in a limit cycle.

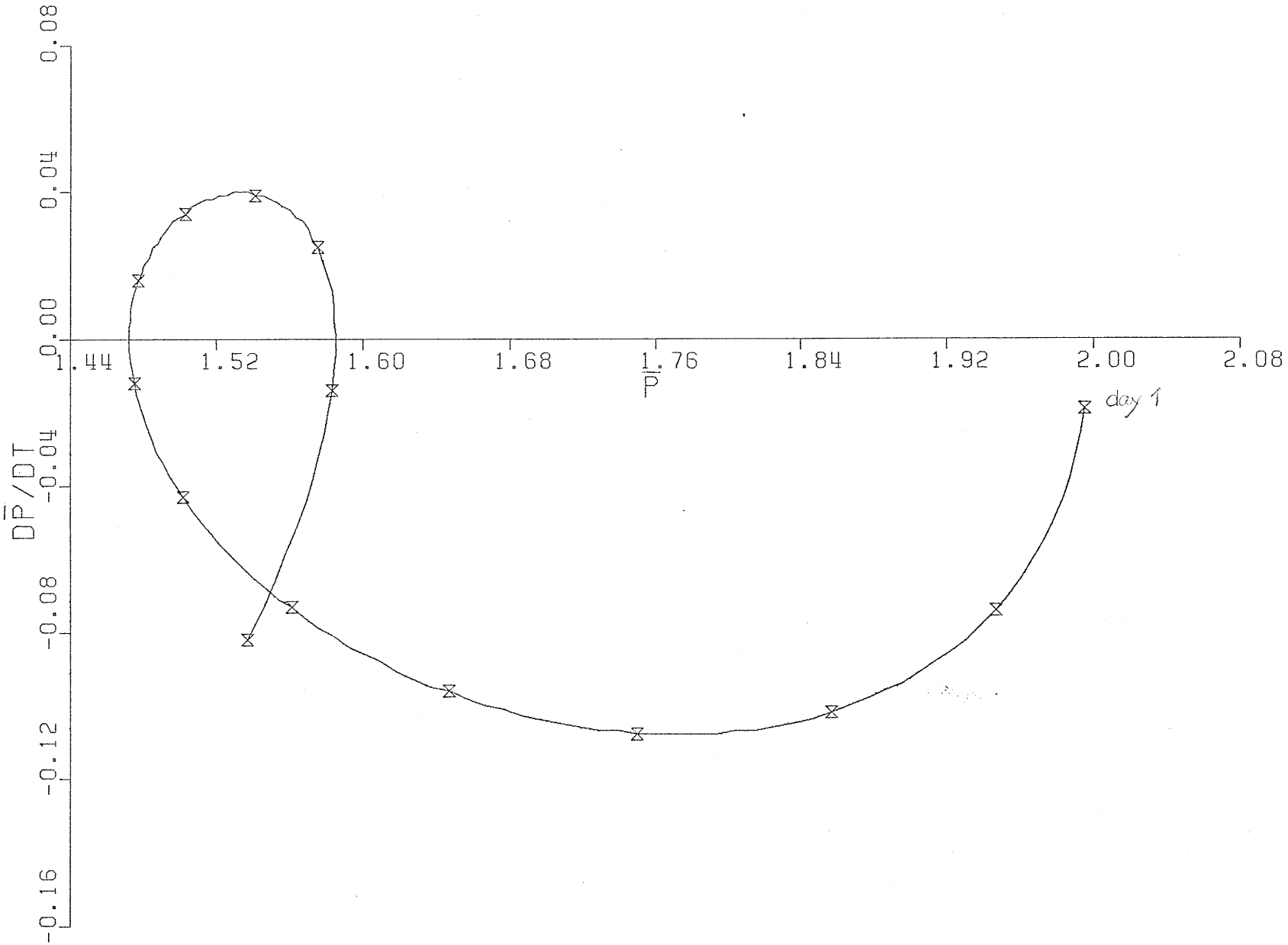
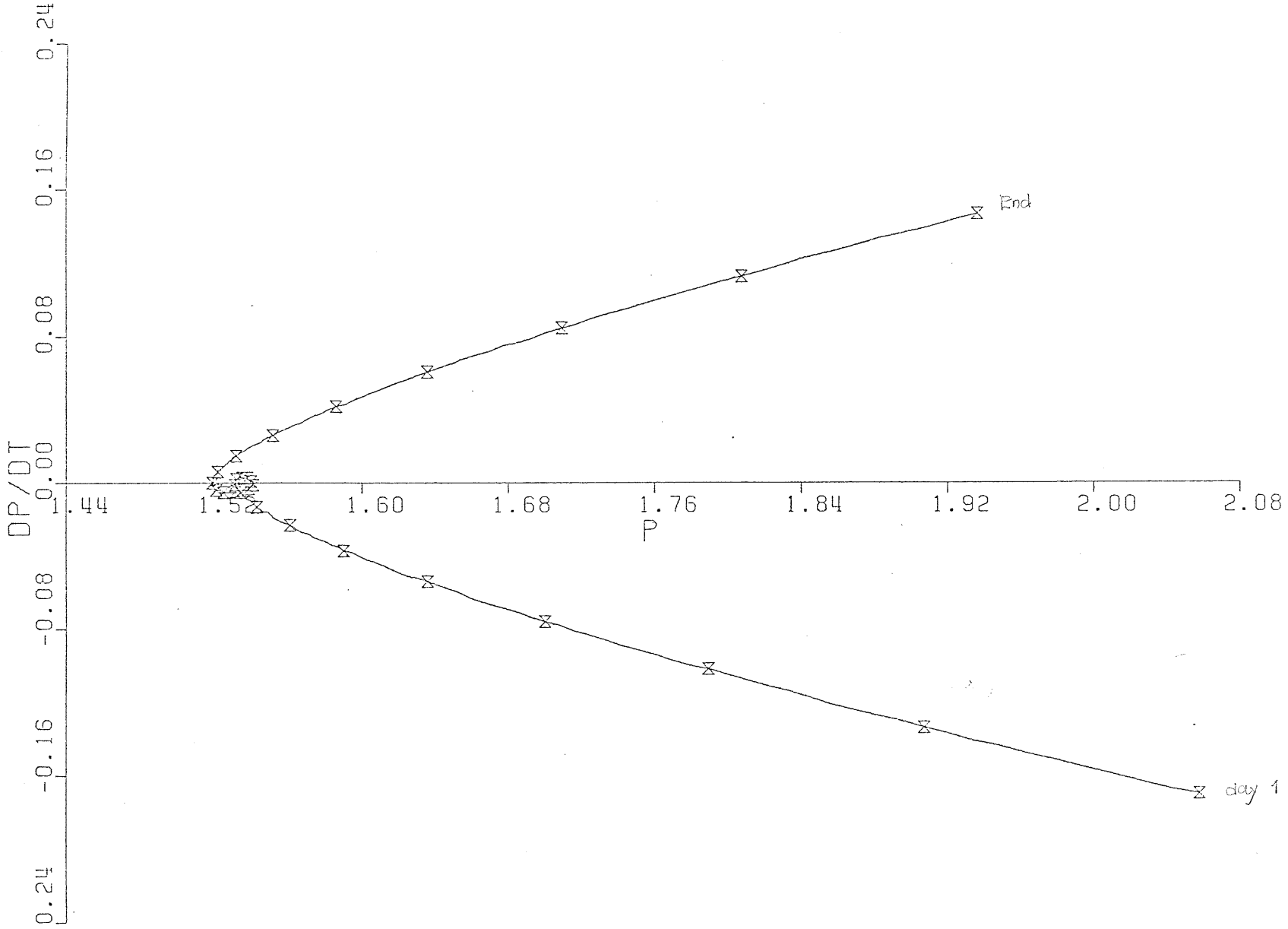

0.08
0.04
0.00
-0.04
-0.08
-0.12
-0.16
DP̄/DT
1.44
1.52
1.60
1.68
1.76
1.84
1.92
2.00
2.08
P̄
day 1

Figure 91. Phase diagram of the normal system subsequent to rest period. Figures 90 and 91 are mirror images of each other.

2.00
1.92
1.84
1.76
1.68
1.60
1.52
1.44
1.36
1/P
End
-0.10
-0.05
0.00
0.05
0.10
0.15
0.20
DP/DT

Figure 92. Phase plane representation of the normal system. System was driven to perform at reduced $\overline{P}$ and dP/DT when I/O control was superimposed on the DCA. System continued to spiral around special value on the $\overline{P}$ axis after the rest period. Behavior stabilized with $\overline{P}$'s and dP/DT approaching a constant, while on the DCA with I/O control. System evolved away from the focus when the I/O control was removed.


0.24
0.16
0.08
0.00
-0.08
-0.16
-0.24
DP/DT
1.44
1.52
1.60
1.68
1.76
1.84
1.92
2.00
2.08
P
End
day 1

RESULTS: THE HISTOLOGICAL DATA

I. brain specimen of subjects bearing bilateral electrolytic lesions in the head of the caudate nucleus:

A) GENERAL OBSERVATIONS:

The damaged brain tissues corresponded well with the area as defined by the stereotaxic atlas of deGroot(1954), ie. anterior -1.7mm from Bregma, + 2.7 mm lateral to,the midline, and 5 mm from the surface of the skull. The brain specimen within this boundary showed damage to the cortical tissues around the area where the lesioning electrodes entered the brain. However,due to the extreme thinness of the sections (6u) and of the long period of time between the surgery and the sacrifice of the animals,there were no distinct tracts observable.

The lesioning current produced a constricted area of damage to the head of the caudate nucleus. The area of maximal damage was confined to the section medial to the lateral aspect of the corpus callosum, dorsal to the anterior commissure and ventral to the medial aspect of the corpus callosum. Scar tissues and destroyed areas (space) were readily observable to the normal eye and under the microscope (4x & 10x). Disarrangement of the corpus callosum due to the penetration of the electrode was also observed.

B) AREA OF MAXIMAL DAMAGE:

The area of maximal damage overlapped to some extent with the space occupied by the lateral ventricles, creating a picture of much enlarged ventricular space.

As shown by the following diagram, the ventricular space was approximately 4 to 5 times that of a normal brain specimen. With the enlargement of the ventricular space, there was a corresponding deduction in the total area normally occupied by the caudate nucleus. In addition, there was much damage to the neurons of the caudate nucleus in the lesioned area, leaving only a very small portion of the caudate tissues free of any scar tissues.

Damage to the section of the corpus callosum by penetration of the electrode was comparable from brain section to brain section across all subjects.

The anterior commissure and other nearby structures escaped injury. The following is a serial reconstruction of brain sections in the area of maximal damage via bilateral electrolytic lesion of HCD. All brain sections were 6u or 11u in thickness and every 5th serial section was presented.

II. brain specimen of subjects served as operated control group for the HCD lesioned ss:

A) GENERAL OBSERVATIONS:

As with the hcd lesioned ss, the brain specimen within the confine of the lesioning coordinates showed damage to the cortical tissues. Such damage was bilateral and symmetrical. As well, the amount of damage to the surface of the cerebral cortex was comparable to those in the lesioned group. In general the damage was a result of the penetration of the electrode through the brain tissue.

The sections of the corpus callosum where the electrode passed showed disarrangement of the fiber tracts. The amount and type of damages were also comparable to those in the lesioned group. There was no identifiable electrode tracts found in the sections. Again an observation comparable to those in the lesioned group.

There was no observable damage to the HCD either with 4x or 10x of magnification under the microscope. The lateral septal nuclei and the caudate nuclei were of normal sizes. There was no enlargement to the lateral ventricular spaces. Postoperatively, there were no temporary hyperreactivity or overt sensitivity to blowing air, nor increased resistance to handling in the form of urination and defecation, which were quite pronounced in the HCD lesioned ss behaviourally, the data verified the intactneSs of the septal area.

Comparison of the HCD and HCD operated control Ss would give the selective effects of damages to the head section of the caudate nucleus,and to the lateral ventricle The maximal damages were sustained by the head of the caudate nucleus which showed reduction both in the total area and in the number of neurons.

III. Brain specimen of Ss bearing bilateral electrolytic lesions in the dentato-interposed nuclei of the cerebellum:

A) GENERAL OBSERVATIONS:

The area bearing tissue damage via passage of

electrical currents centered around the section as defined by the lesioning coordinates by Bures et al (1967), ie. ap-11mm frcm Bregma, lateral- 2.2 mm from the midline and vertical -7mm frcm the surface of the skull. However, the majority cf the lesioned areas were slightly ventral to dendate nucleus area with most De neurons. Consequently, only the ventral portion of the dentate nucleus (DE) was affected, resulting in the sparing of much DE neurons.

In general, there were bilateral symmetrical indent on the surface of the cerebellar cortices where the electrodes were lowered into the brain. The amount of tissue damage was consistant from specimen to specimen. Again, there was no distinctly identifiable electrode tracts left by penetration cf the electrodes into the brain tissue.

B)THE AREA OF MAXIMAL DAMAGES:

maximal damages were sustained in the ventral portion of the dentate nucleus in the section lateral to the fourth vewntricle. There were also damages to the acoustic tubercle, restiferm body, and trigeminal nerve, in the vicinity of the DE. The only exception was rat 52 which bore lesicns in the ventral portion of the DE while escaping damage to both the CR and CSV.

Involvement of Deiters nucleus and Bechterew nucleus were possible but very slight. More anteriorly, damage to the brachium conjunctivim (BC) was also present.

Due to the closeness of the lesioned sites to the lateral aspect of VIV, the specimen with lesions created an illusion of larger than normal ventricular spaces.

The following diagrams are samples of a typical DE lesion. Serial reconstruction of the sections suggested the extent of damage to be from AP11 to AP9.0 of Bures et al's atlas.

IV. brain specimen of subjects bearing bilateral sham lesion in the caudato-interposed nuclei of the cerebellum

(A) GENERAL OBSERVATIONS:

As with the DE lesioned ss, the brain specimen showed slight bilateral damages to the cerebellar cortices in the tissue block as defined by the lesion coordinates of Bures et al (1967). As presented in the following diagram, the damages to both cortices were symmetrical, causing destruction of comparable magnitude on both sides. Such damages were quite comparable to those sustained by the lesioned group.

Again, as with the lesioned Ss there were no distinctly identifiable marks left by the passage of the electrodes, though some sections gave the appearence of increased glial cells.

On the whole, the DE area remained intact and were clearly identifiable under the microscope(4x).

v.The brain specimen of Ss bearing bilateral electrolytic lesions to the pre-frontal pole of the cerebrum:

A) GENERAL OBSERVATION:

The area of maximal damage wherein lesion sites were identified corresponded to the area designated by the lesion coordinates of deGroot(1954) ie. AP 5.2mm lateral to the midline and 2mm from the surface of the skull.

As shown in the following diagram, the lesioned sites were triangular in shape and the area of destruction was situated in the upper medial quadrum of the pre-frontal cortex. The destroyed area was approximately 1/8 of the total pre-frontal cortex.

B) AREA OF MAXIMAL DAMAGE:

The area bearing destructive electrolytic lesions were effectively confined to the pre-frontal cortex. No visible damages were observed either in the lateral ventricles or in the head of the caudate nucleus. The subcortical structures in the pre-frontal areas were also spared from damage.

C) ASYMETRICAL BILATERAL LESION:

in rat 45, the brain specimen showed the lesions to be asymmetrical. The lesion site was situated in the lower right quadrant of the left hemisphere. Comparable size of lesioned site was situated in the upper left

quadrant in the right hemisphere. As with all Ss samples described previously, there were no identifiable marks left by the passage of the electrodes, other than very slight reduction of cortical neurons along the tissues aligned with the damaged cortical surface and the lesioned sites.

In general, the lesioning current (3.5ma. for 30sec.) produced an elongated triangular shape or a tear shaped damage to the tissue. The space produced by destructive electrolytiic current showed presence of glial cells. Scar tissues were present in the vicinity of the lesioned area. The ventricular spaces posterior to the lesion sites were of normal sizes.the caudate nucleus was also spared.

VI.brain specimen of Ss bearing bilateral sham lesion in the pre-frontal cortex.

GENERAL OBSERVATION:

Both the left and right pre- frontal cortices showed slight indent on the surface of the cortex. Based on the location of the indentation, one could reasonably conclude that these damages were left by the penetration of the electrodes into the brain tissue. the extent and loci of these indentation were consistant in all sections of the brain specimen of Ss in this group.

VIII.brain specimen of Ss bearing electrolytic

lesions in the posterior aspect of the caudate nucleus

A) GENERAL OBSERVATION:

The area wherein lesioned damages to the tissue was located corresponed closely with the intended lesion sites. The site of maximal damage was within the confines of the lesioning coordinates of deGroot(1954), ie.AP .2mm from Bregma, 3.5mm lateral to the midline and 3.5mm from the surface of the skull.

Scar tissue was present from AP 7.8 to AP5.8mm.

In general, there were bilateralsymmetrical damages to the surface of the cerebral cortex where the electrodes entered the brain. As was consistant with all other groups, there were no distinctly identifiable marks left by the passage of the electrodes. On the whole there was much tearing in the brain tissues, making the verification of lesioned sites were difficult at times. brain sections of rat 80).

B) THE AREA OF MAXIMAL DAMAGE

The area sustaining maximal damage was located in the middle section of the CPU(nucleus caudate/putamen), medial to the corpus callosum, and lateral to the internal capsule. Two ss (rats 86&75) seemed to have sustained some damage to the lateral ventricles, causing a general enlargement of the ventricular space. All other Ss escaped damage to the ventricles. On the whole,

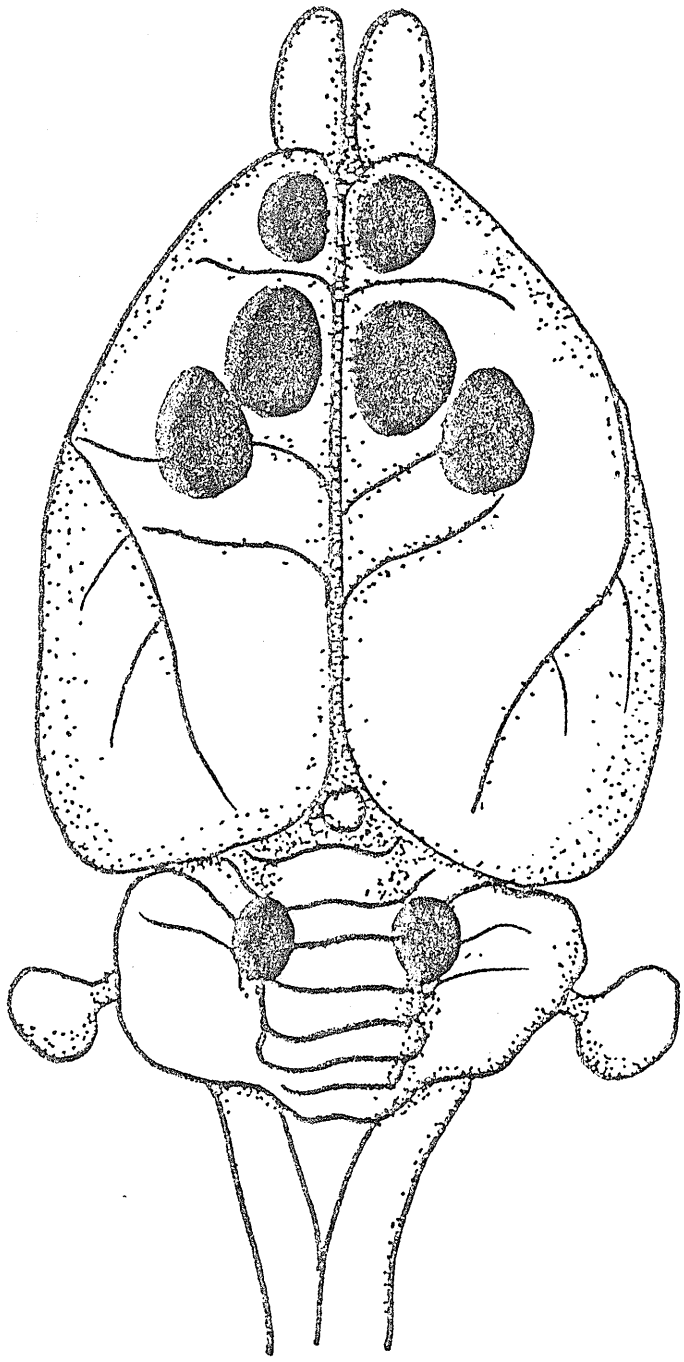
the lesioning current produced a constricted area of destruction within the confines of CPU. Total area destruction was approximately 1/8 of the CPU territory. The length of the destroyed area was approximately 1/4 of the length of the subcortical area.

Damages to the corpus callosum were limited to the medial sections where the electrodes passed through to reach the proposed lesion sites. Disarrangement of the fiber tracts were clearly observable under the microscope. The section of the corpus callosum which showed most disturbance was slightly anterior to the section with maximal damage to the CPU.

VIII. Brain specimen of Ss bearing sham lesions in the posterior aspect of the caudate nucleus

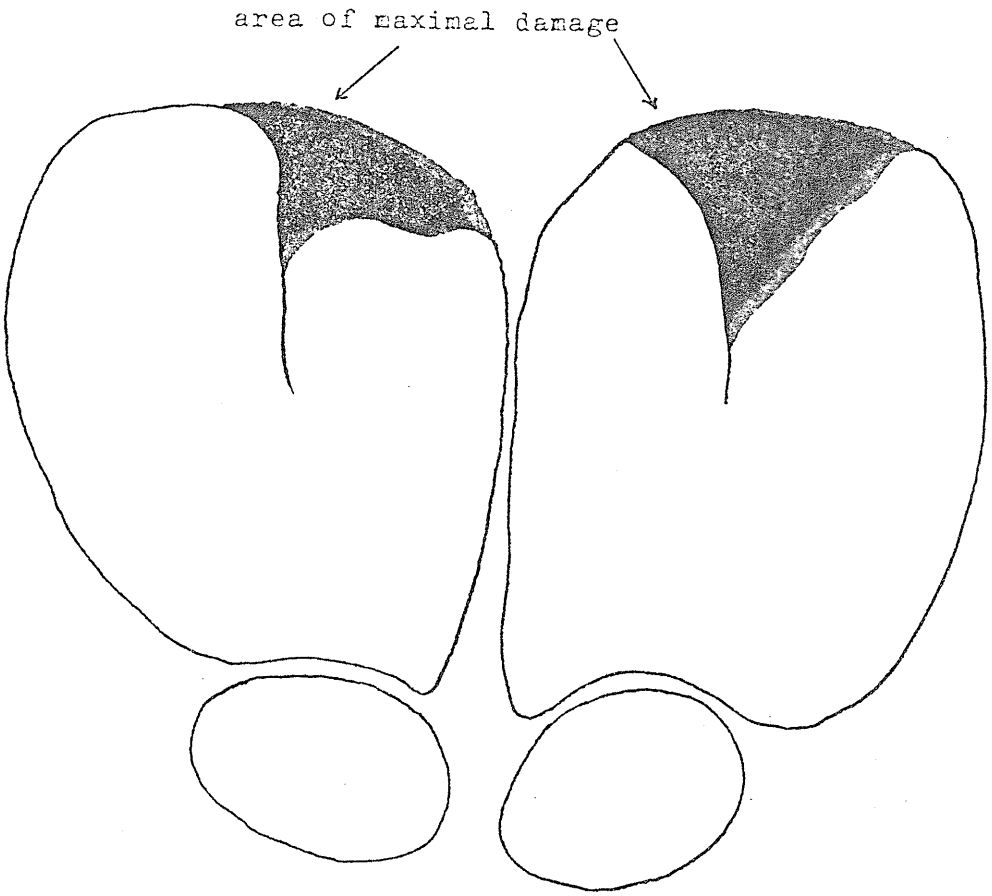
GENERAL OBSERVATIONS:

As was the case with two other sham operated groups, the brain specimen of Ss in this group showed the symmetrical indentation on the surface of the cerebral cortices. These indents were caused by the passage of the electrode through the brain tissue. Again there were no identifiable marks left. There was much tearing and breakage in the brain section, probably associated with the histological procedures.



Diagrammatic representation of the lesion sites in rat brains.

Figure 93. Diagrammatic representation of the lesicn sites in rat brains.



Rat 35, slide # W.

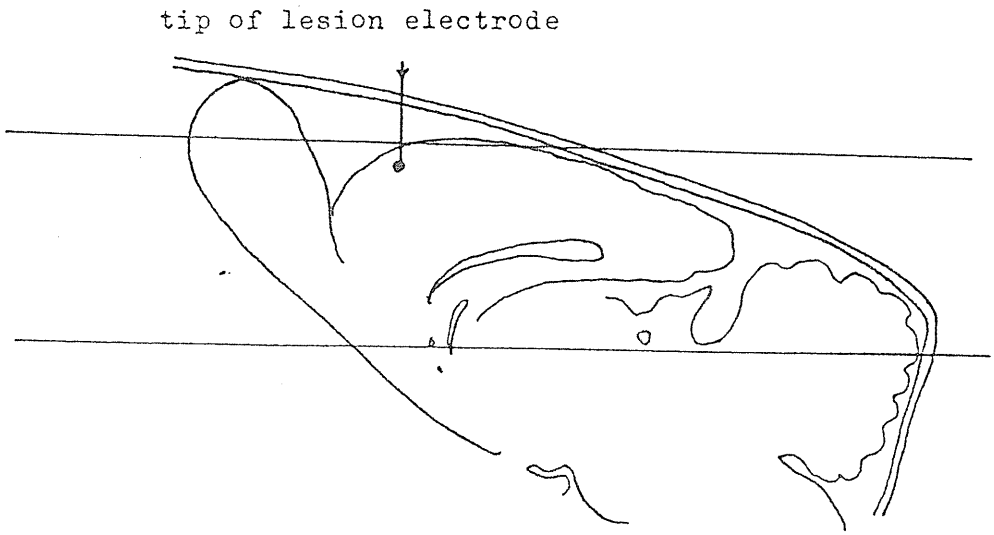
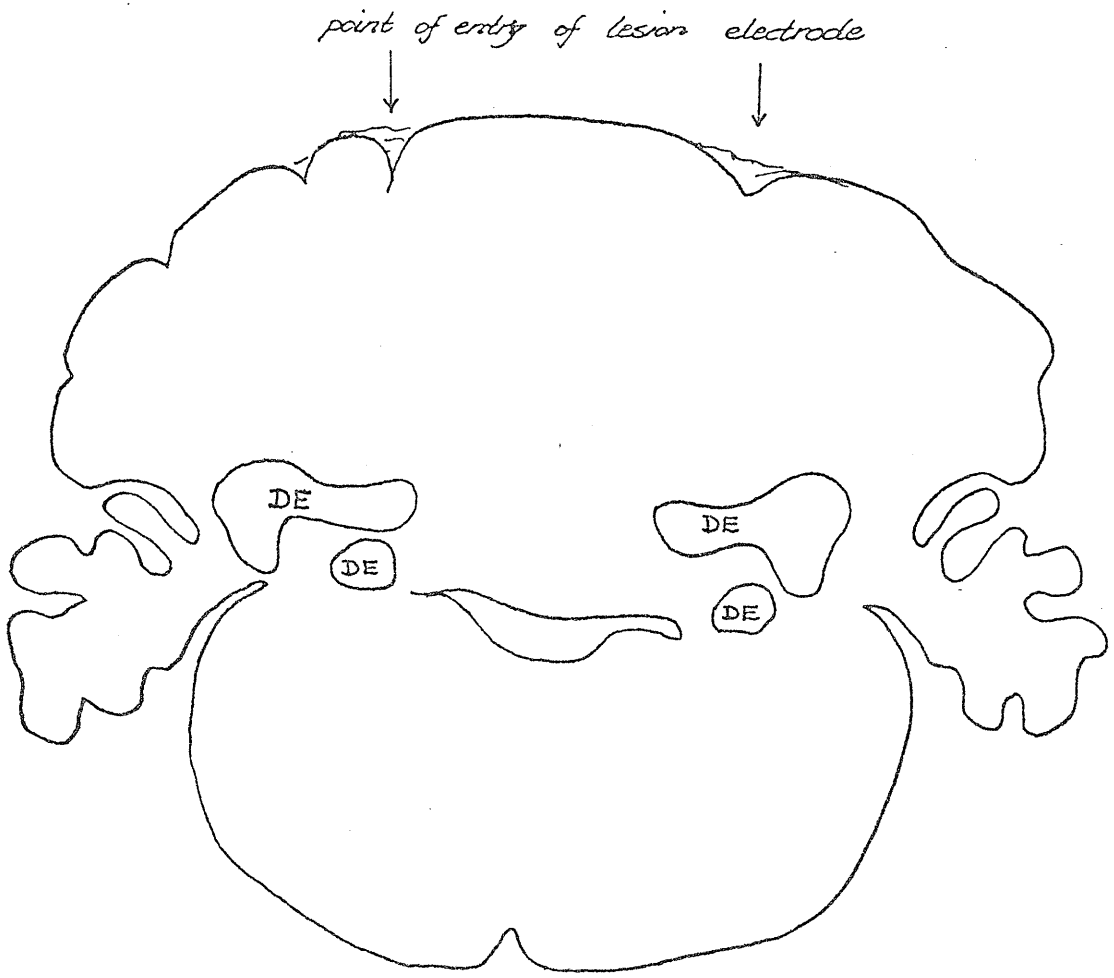
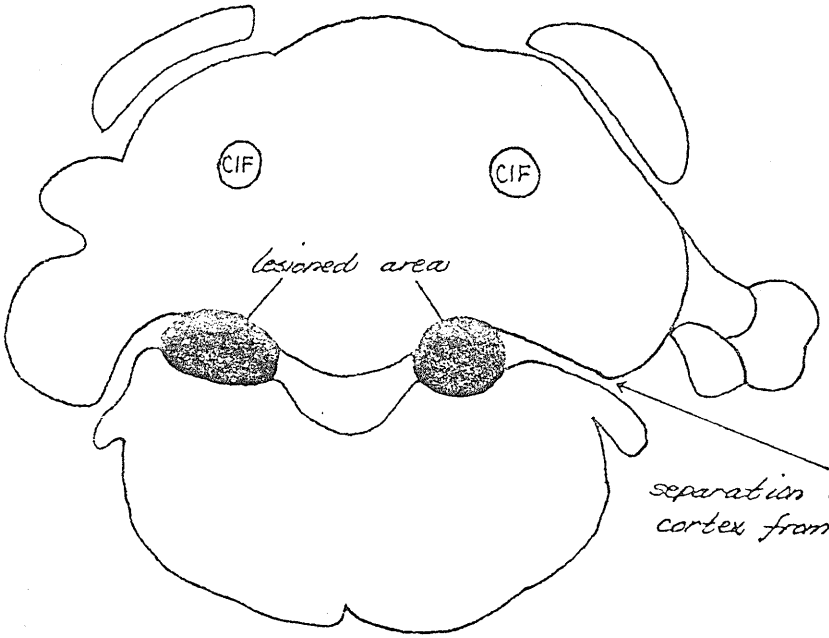


Diagram D. A diagram of a brain specimen bearing bilateral electro- lytic lesion to the frontal pole of the cerebral cortex.

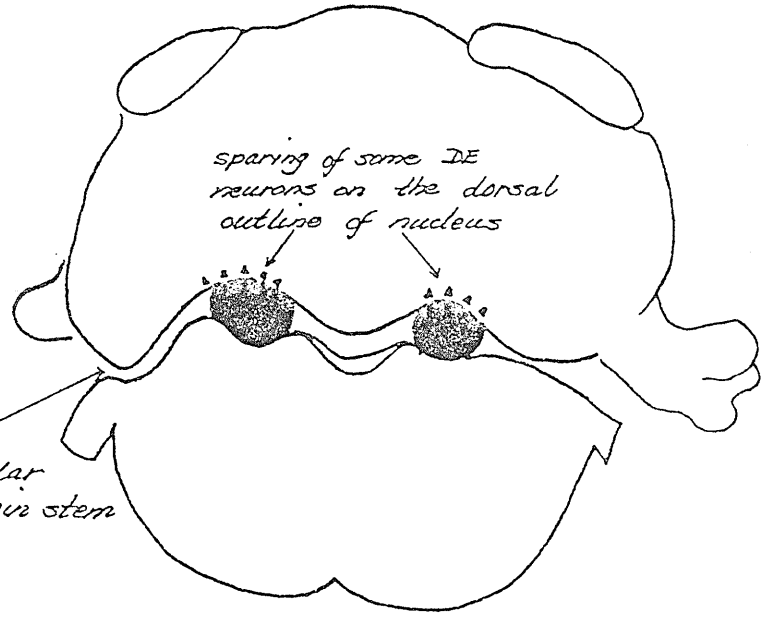


Rat # 55, slide # 10, AP 10.0

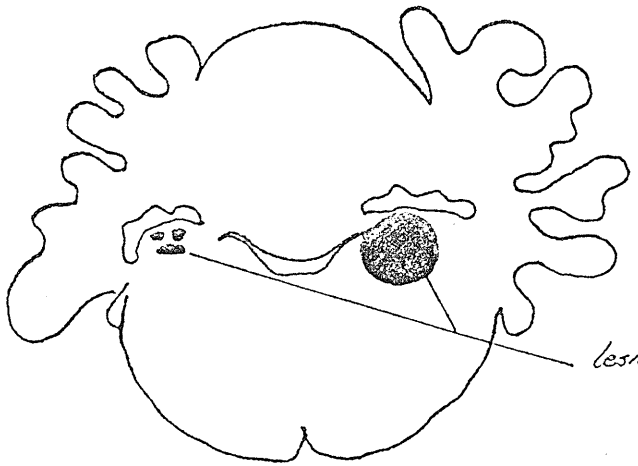
Diagram C.A diagram of a brain specimen bearing sham lesion in the dentato-interposed nuclei of the cerebellum.



Rat# 69, slide #3 AP 9

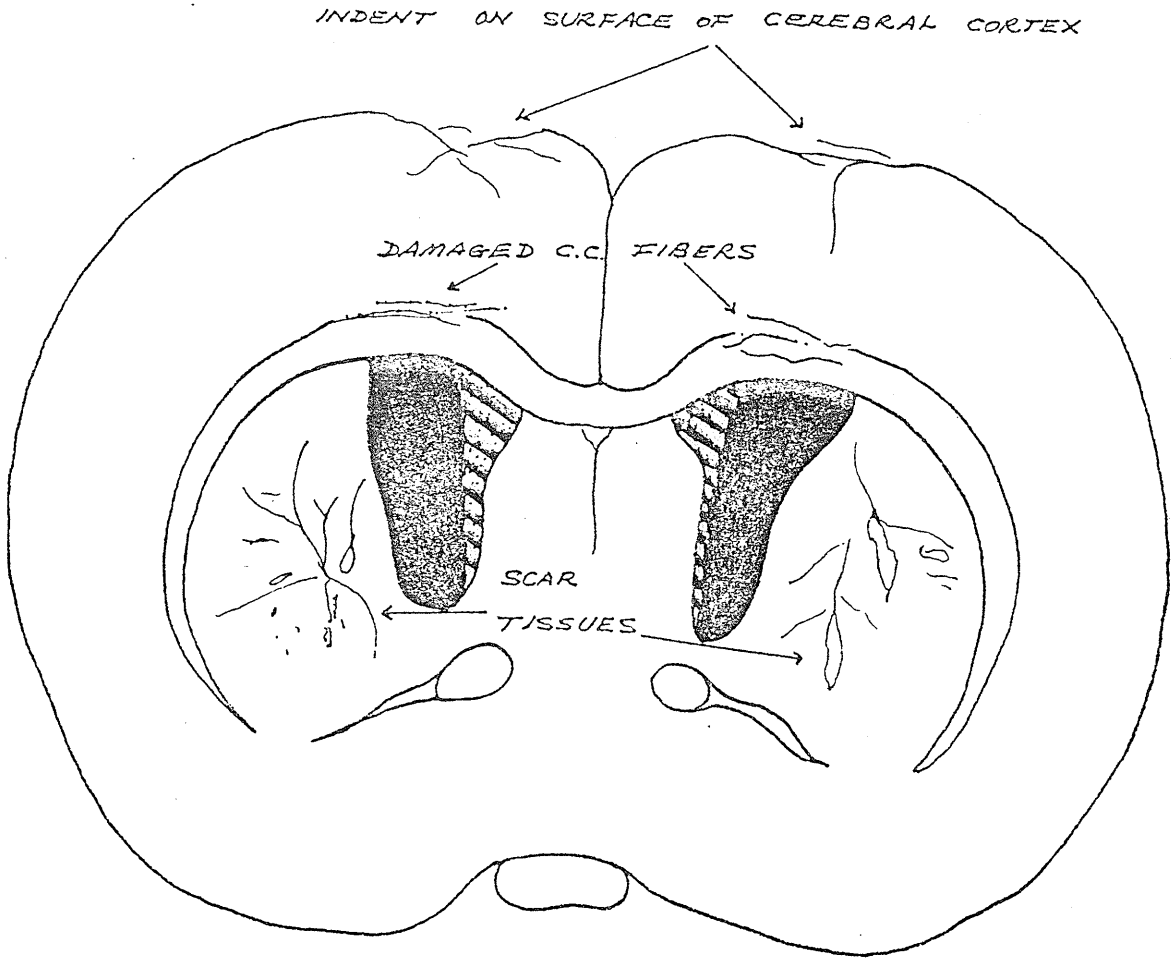


Rat # 69, slide # 4 AP 10



Rat# 52 slide #10

Diagram B. Diagrams of brain speciemn bearing bilateral electro- lytic lesions to the dentato-interposed nuclei in the cerebellum. Drawing done under 10X of a micrcsccpe.



RAT # 23

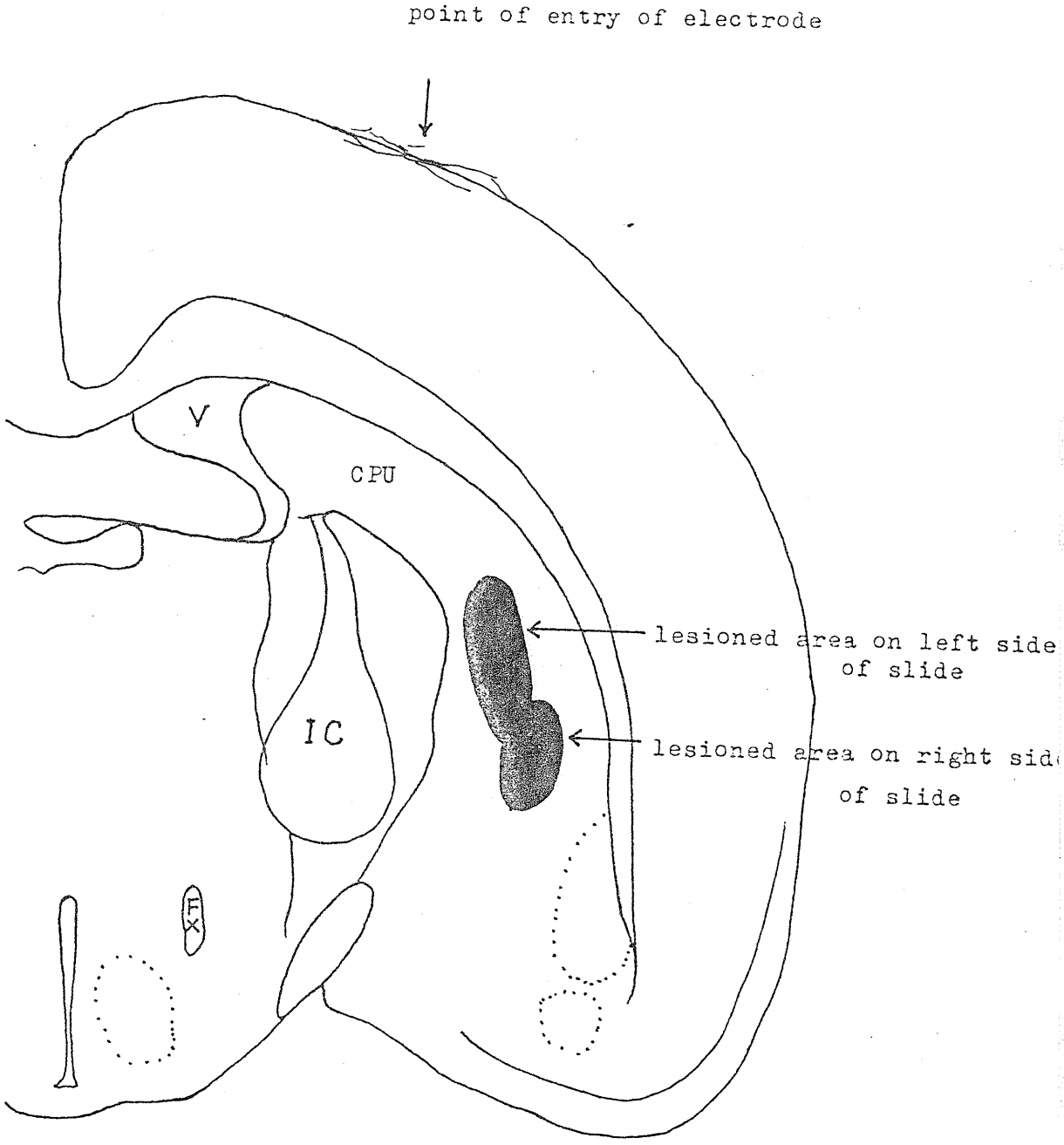
SLIDE #7

NORMAL SIZE & SHAPE OF LATERAL VENTRICLE

+ AREA OF MAXIMAL DAMAGE

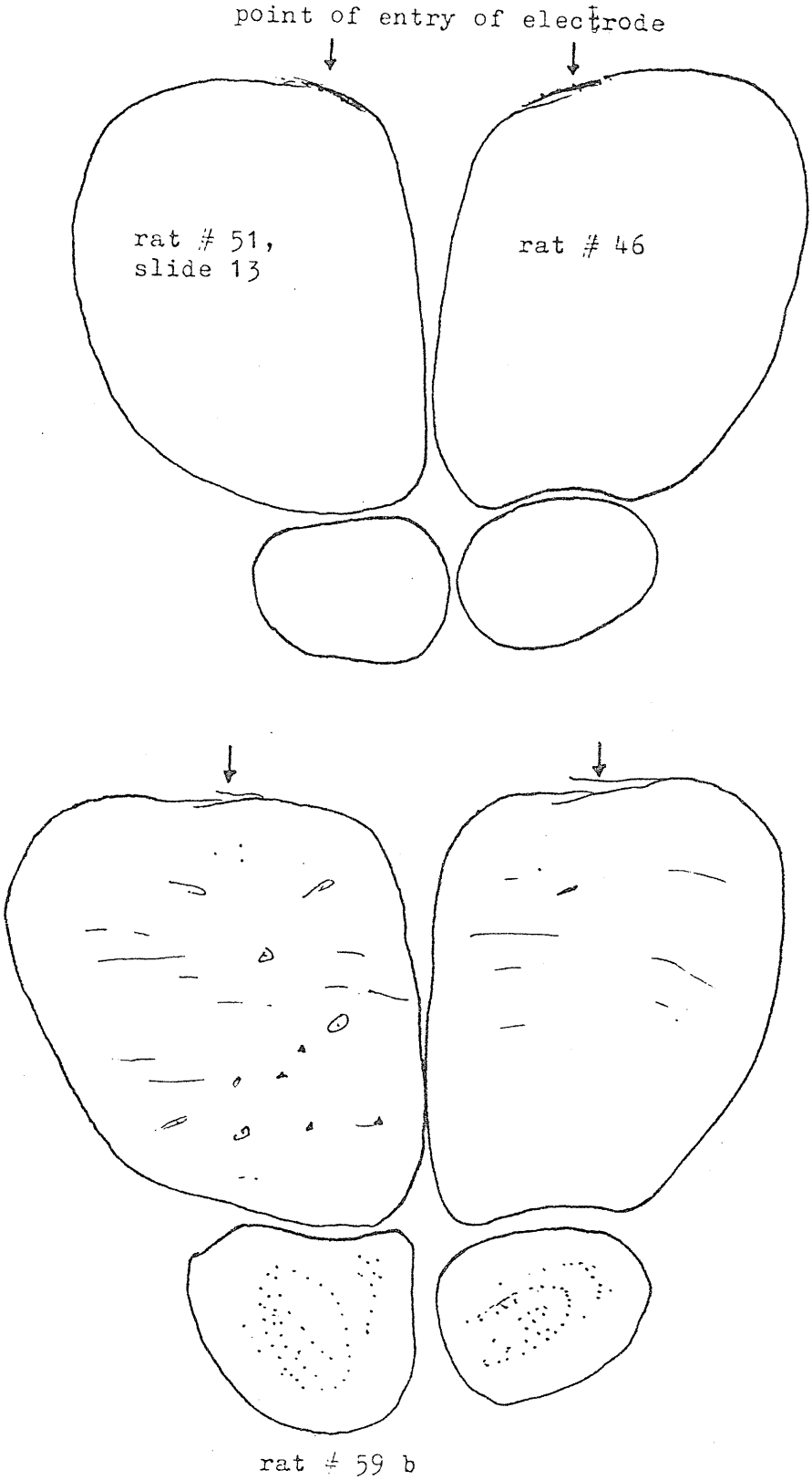
Diagram A. A diagram of a brain specimen bearing bilateral electrolytic lesion to the head of the caudate nucleus. Observation done under 10X in a microscope.

Diagram H. Diagrams of brain specimen bearing bilateral electrolytic lesion in the P CD, note the enlarged ventricular space.



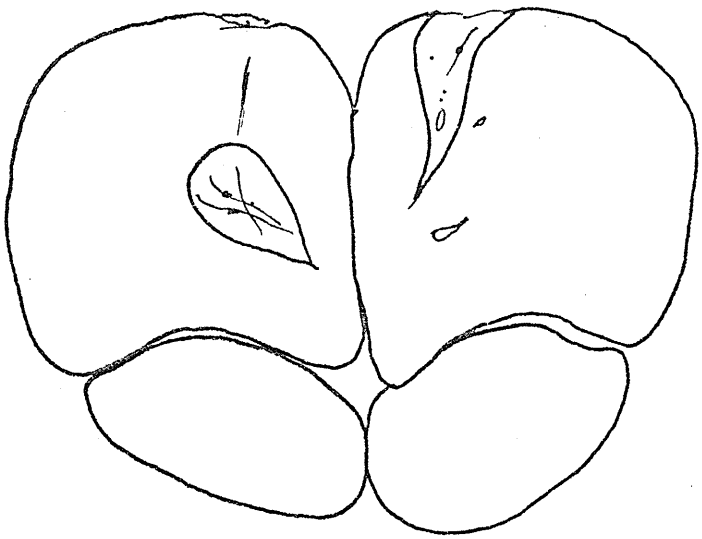
rat # 85, slide # 2, area of maximal damage corresponded to AP 6.2 to AP 5.8 of deGroot's atlas for the rat.

Diagram G. Diagram of brain specimen with bilateral electrolytic lesion in the posterior aspect of the caudate nucleus.



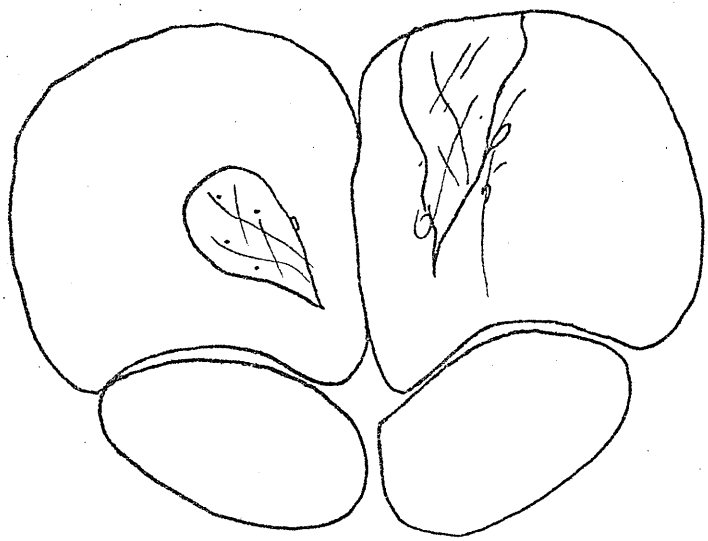
The extent of tissue damage due to penetration of the electrode.

Diagram F. Diagrams of brain specimen with sham lesion in the frontal pole of the cerebral cortex.



lesioned area

slide # 14

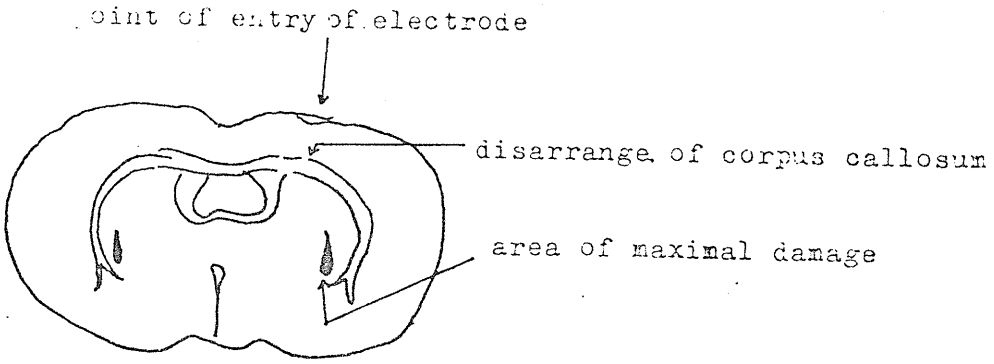


scar tissue

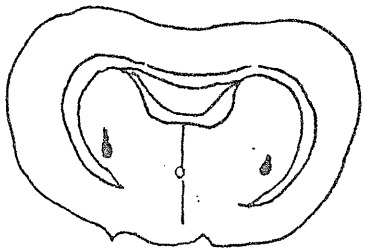
slide # 15 d

Rat # 45 with bilateral electrolytic lesion in the frontal area of the cerebral cortex.

Diagram E. Diagrams of brain specimen bearing asymmetrical bilateral electrolytic lesion to the frontal pole of the cerebral cortex.

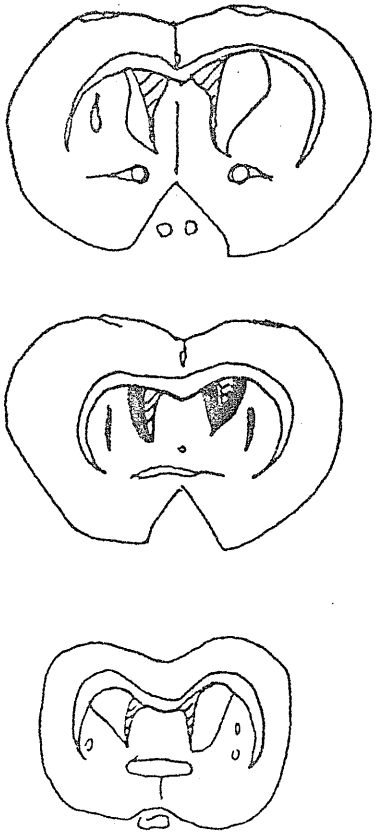


rat # 87, slide # 4 AP 6.6 mm



rat # 87, slide # 3 AP 6.2 mm

Ha. Two slides from rat # 87 which received bilateral electrolytic lesions.



Hb. Three slides from rat # 86 which received bilateral electrolytic lesions. In this S the ventricular area was affected.

CHAPTER IV

DISCUSSION

Initiation and Control of Movement and Response Inhibition

(A) the frontal-striatal system:

Initiation of voluntary movement: The present study suggested that both H CD and P CD lesions led to a temporary impairment of response on the DCA (c.f., Figures 7, 10, 13, 66, 69 & 71). This phenomenon lends support to Hansing (1968) and Denny-Brown (1962) hypothesis that the striato-pallidal system is involved in the initiation of behaviour. Electrolytic destruction of the areas led to a temporary disruption in the initiation of the CR's in the operant chamber. Responses in the same session showed increased response latencies (negative relative errors),interspersed with many non-responses to the 40 CS presentations. That the disruption was temporary attests to the phenomenon of behavioural recovery after electrolytic lesions in the brain. As shown by the work of Finger et al (1973), Raisman & Field (1973), Schultze & Stein (1975) and Wall & Egger (1971), there is collateral reinnervation and functional recovery in the CNS areas. In addition this phenomenon is quantitatively predictable and follows a rigid time-course and results in a characteristic pattern of synaptic formation. The lesioned areas in the present study were confined in well localized areas to avoid massive damage to nearby struc-

tures. Consequently there were some sparing of the caudate neurons. Therefore it is conceivable that to some extent there was functional recovery in the caudate neurons. That the recovery was not complete is shown by the failure of the H CD to return to baseline performance on the DCA after removal of the I/O control(c.f., Figure 13).

Caudate nucleus and act inhibition (Stanley & Jaynes' concept of act inhibition): If one accepts Halasz's (1969) arguement that Pavlovian inhibition of delay develcps in the DCA,then the original tone CS, together with the 5" or 9" of delay acquired an inhibitory quality. Thus the first 5" or 9" of the CS duration acted as a physiclogical stimulus for the subsequent period of activity. Lever presses should increase as the reinforcement availability period approached. The data from the H CD Ss showed that subsequent to the initial sessicn of many nonresponses to the CS after the lesion, Ss developed a patterned motoric chain to mediate the CS-SD interval. Such motoric chains were concommitent to improved performance on the DCA. As presented in the results sectien, the components of the motoric chain were 'anticipatory goal behaviour' which were present during the preliminary training but were later 'inhibited' in that Ss refrained from responding during the delay period in the DCA. Lesions in the dorsal and dorsomedial aspects of the H CD presumatly produced the disinhibition of the motcric events,as shownbby theppost-operative

development of the collateral behavioural chains (table 1). Presumably compensatory motoric chains were then developed, utilizing the disinhibited motoric sequences to mediate the delay. All these events took place when the I/O control was in effect. Perseveration of the conditioned behaviour was manifested when the Ss failed to readjust their response latencies upon the removal of the superimposed I/O control. The apparent improved performance of the H CD Ss on the DCA, and in particular, of maintained improved performance when the I/O control was removedcould be attributed to the failure of the defective system to overcome the initial behavioural set. The apparent facilitatory effect of the H CD lesion could thus be a masked form of behavioural perseveration. The post-operative performance of the H CD group could be motoric disinhibition which facilitated the formation of mediating behavioural chain during the delay period in the DCA.The absence of reduction in response latencies when I/O control was no longer in effect could be a result of impairment to the central neuronal feedback loops essential to proper adjustment of the system to changes in 'timimg'. This defective system responded to the control demand with an initial overshoot of an underdamped system and then precipitated into a hard limit cycle, which is an invariant state of the conditioned behaviour.

Post-operatively the P CD lesioned system also showed an initial underdamped adjustment on the DCA with

I/O control. However,the behavioural system quickly returned to the pre-operative level of performance on the third testing session,without the development of 'lesion-related' motoric chains. Therefore it could be assumed that behavioral data did not suggest disinhibition of previously inhibited motor behaviour.

Unlike the studies (eg. Rosvold,1958; Divac, 1967, 1968) which employed the more conventional tasks such as delayed response and delayed alteration frontal lesions did not produce behavioural effects which are similar to that of the caudate nucleus lesion. Rather, the frontal system showed a shift of the response latencies to a shorter value, resulting in an increase in the relative error of the behavioural system. It suggests the possibility of hyperreactivity to the presentation of CS.The absence of lesion-induced increase in trial abort count (Figure 45) would lend support to this arguement .That the shortened response latencies to DCA could not be attributed to an enhanced responsivity to rewarding stimuli supported the conclusions of Neill et al (1974).

(B) the cerebellar control system:

The conventional concept classsified the cerebellum as the a segment of the efferent motor system which is regulated by the 'higher' centers in the cerebrum (Brooks,1975). The most recent researches however tend to reconfirm Holmes' (1907) proposal that the cerebellum is involved in the initiation of voluntary movement. For

instance, Thach's work (1969,1973,1975) with primates suggested that the cerebellum output may help initiate as well as regulate behaviour. Thach's experiments studied the temporal relationship between changes in cerebellar activity and a simple learned movement, flexion and extension of ipsilateral wrist. Measurement of the discharge of single neurons in the dentate and interposed nuclei showed that many cells showed "modified discharge between signal and movement, and changes were time-locked to movement rather than signal.....for most cells discharge changed even before the EMG response preceding movement". Using the functional relationship in the vestibular-ocular reflex arc and the superposed cerebellum as an example, Ito (1970) considered the cerebellum to be a servomencanism of the feedforward control system which "having the aid of a computer having a learning capacity, can perform much more flexible and subtle controlling..." (p.). Based on his theory , the cerebellar cortex normally exerts a modulating function over the subcortical neurons which receive excitatory signals from the cerebellar afferent collateral fibers. The neocerebellar cortex is linked to the cerebral motor area by the cortico-spinal and cortico-bulbar impulses via the pontine nuclei and the inferior olive. The hemispheric output from the dentate nucleus returns to the motor area via the VL nucleus of the thalamus. Ito called this arrangement a model reference adaptative control system. During the learning phase of a complex motoric skill, the

voluntary unskilled movements were initiated by the association cortex which were first transfered to the motor cortex and then through the pyramidal tract down to the spinal motor centers. The final outcome was checked by the association cortex via the sensory pathways. This system of control is a large negative feedback loop "formed through the external world" (Ito,1970). During the learning phase,the cerebral cortex is continuously aware of the system's performance and adjustment are made as required. With sufficient training, the performance of the CR's become refined voluntary skilled movements. The large negative feedback loop via the external world is replaced by an internal one "passing through the cerebellum which would serve as a model of the combination of the spinal motor system,the external world and the sensory pathways". Thus the original negative feedback system is converted by learning into a feedford system which no longer depend on a straightford negative feedback loop from the output to the input. Additional input pathways to the cerebellum serves to adjust the internal model as required in a servomechanism-like system.

In the context of Ito's analysis,cerebellar dysmetria and intentional tremor were viewed as a result of loss or impairment of the internal model. The impairment would manifest itself in the overall performance of a system which utilized a large negative feedback loop with a complex transfer function. As is shown in the method

section, the DCA is a complex behaviour requiring 3 to 4 months of training to achieve relative consistency in performance. The results showed that intentional tremor was present in the conditioned behaviour of the D-I lesioned Ss in the operant chamber. However the performance on the DCA was not impaired by the 'impairment in the internal model in the cerebellum'. Data presented in the result section showed that the response curves of the D-I lesioned system was identical with those of the normal system. Several factcrs might have contributed buted tc the apparent nonexistence of impairment of DCA by the ataxic system. One such factor could be the servomechanism-like characteristic of the DCA paradigm, a simulated model of optimally contrclled processes. While bilateral electrclytic lesicn in the D-I nuclei effectively produced clinical symptcms of dysmetria and intentional tremor,the optimal process control paradigm provided sufficient parallel tc and an substitute for the neuronal feedback for the adequate performance of the conditicned behaviour on the DCA. Functional recovery could be another contributing factor for absence of impairment in the initiation of CR's in the D-I system. The histclcgical data showed that there were sparing of dentate neurons in the dorsal aspect of the nucleus. It is conceivable that the remaining dentate neurons were able to compensate for the function of the destroyed neurons.

It is possible that the nature of the task demand,

namely the ability to delay the conditioned behaviour relative to the onset of a CS presentation, enabled the system to readjust its response during the delay period so it could still adequately maintain its performance efficiency. The exact nature of the relationship between intentional tremor and performance efficiency awaits future experimental research. The results from the present study are at variance with Persinger's(1971) study. He examined the relationship between pre- and neo-natal exposure to X-radiation or electromagnetic fields and the DCA. His study showed that in the DCA situation greatest behavioural effects were obtained during transient states associated with the schedule changes , while performance in the steady states was little affected. Ascribing to Marr's(1969) theory of cerebellar cortex Persinger conceptualized temporal or timing function as mediated via the mossy fiber-granule-cell parallel fiber circuit. Based on this hypothesis, disruptive effects on the DCA would manifest itself when the context from the mossy fiber input varies from cerebral input. Yet in view of the two main behavioural data from the present study, namely, (1) the H CD lesion-induced perseveration on the DCA, which coincided with the post-operative development of motor mediating response and (2) the failure of cerebellar dysmetria and intentional tremor to impair DCA, the complex motor learning does not seem to take place in the cerebellum(Halasz, 1978 personal communication). Rather than sup-

porting the theory by Marr(1969), Eccles(1969,1973) and Ito(1970), the present study tends to give more support to Llinas'(1967, 1971, 1972, 1974), and Sotelo's(1975) position that complex motor learning does not take place in the cerebellum.

MOTIVATIONAL HYPOTHESIS:

Suggestions have be made (Sorensen & Ellison,1970) that the changes in the performance of the CD Ss could be secondary to a changed motivational state. Were this the case, the disrupted performanceon the DCA by the H CD and P CD Ss could be attributed to reduced level of thirst. In the present study this hypothesis is made plausible in view of the fact that histological data suggested the invasion of lesion damage to the lateral ventricles. The brain ventricular system has in turn been proposed as the active site for the dipsogenic response to angiotensin (Johnson,1972; Johnson & Epstein,1975). Their work showed that even insensitive tissues in the caudate nucleus could be made effective for arousal of drinking by angiotensin if the cannula for the peptide was angled to pass through the ventricles. However,several factors would suggest that the present effects of the caudate lesion on the DCA could not be explained by this motivational hypothesis. First, Buggs'(1974,1975,1976) work suggested that there was complete recovery of drug-induced drinking behaviour from 4 to 14 days after the lesion of the proposed active sites for angiotensin.

The post-operative testing sessions in the present study did not commence until 19 days after the lesion. By that time the possible disruption in the ventricular flow would have recovered. Second, Bugg's ((19767) study alsc implicated the antericr third ventricle at the preoptic and hypothalamic level as the active site for angiotensin. The fourth ventricle and the posterior third ventricle were withcut any active sites. And third, Neill et al (1974) compared the effects of frontal, striatal, and septal lesicns in paradigms thought to measure incentive motivaticn cr behavioural inhibition. The results showed that rats with frontal or striatal lesions which increased responding for rewards in many operant paradigms did not sufficiently increase their rate of water consumption,rate of acquisition of saccharine drinking, latency to eat in a novel environment or acquisition of runway responses. They concluded that "the behavioural changes seen after striatal cr frontal damage in the rat are not due to enhanced responsivity to rewarding stimuli". Thus, it is more likely that CD performance was due to disruption of brain structures concerned with temporal discrimination than to any indirect effect cn motivational systems.

CONCLUSION AND SUMMARY

Based on the more recent literature (Everts,1975; Thach, 1975; Brooks, 1975; Liu & Chambers, 1971) both the caudate nucleus and the dentato-interposed nuclei are proposed to be the essential structures in the initiation and control of learned behaviour. Eccles (1969,1973) , & Ito (1970) proposed that the channels for the neural traffic from the 'will' to actual movement inception run from the cerebral association cortex to the motor cortex with a detour for learning through the cerebellum. Kemp and Powell (1971) stressed the anatomical convergence from association cortex through the basal ganglia and cerebellum,via the thalamic funnel of VL, to the motor cortex. Therefore the major cerebral influence of the cerebellum and the basal ganglia is upon the motor area of the cortex. Efferently the entire cerebral cortex sends fibers to both the basal ganglia and the cerebellum. Thus " the input going into the cerebellum and into the basal ganglia may be coded in a more abstract and complex manner than the input into the motor cortex" (Everts, 1975). Within this context, motor control is now studied in terms of relations of different divisions of the central nervous system to one another and to parts of the body. Many motor functions are multiply represented and control of these functions invloves simultaneous action of all levels of the nervous system's hierarchical organization .The work of Thach , Everts,Ito

and Brooks all implied the existence of transcortical feedback loops in a servomechanism-like functional system (Everts 1973, Halasz, 1973). The DCA is a self-regulating behaviour system which is servomechanically driven and homeostatically stabilized (Halasz, 1972). It can be used as a simulated model of CNS control over conditioned behaviour. In the present study it was used as a "probe" methodology to study behavioural abnormalty which might have resulted from lesions in the striatal-frontal and/or dentato-interposed motor subsystems. The report in the result section suggested that the DCA with the I/O control successfully restricted the performance of the behavioural systems in a well-defined range in the state-space of behaviour. Prior to the surgery, the phase diagrams of all groups showed tendency to a soft limit cycle with the focus around the P axis. Post-operatively, the trajectory of the H CD system showed a temporary disruption in the form of an underdamped overshoot, then precipitated into a hard limit cycle, no longer sensitive to changes in the governing equations (eg. reinforcement contingencies). The P CD and the frontal systems also showed temporary disruption . The former responded in the form of an underdamped overshoot similar to the H CD system, while the latter responded in the form of a damped oscillation torward a larger relative error. Both system returned to the pre-operative response patterns characterized by a tendency to a soft limit cycle on the DCA with I/O control paradigm. The

systems behaviour showed corresponding adjustment when the noninvariance of governing equations were assumed (changes in reinforcement contingencies). The dentato-interposed system was permanently ataxic. However the system manifested the same behavioural characteristics as before the lesion. In addition ,its post-lesioned behavioural history was virtually identical with that of the normal system. Thus,using the DCA and the DCA with the I/O control,the present study differentiated betweem (a) the specific effects of CD lesion on initiation and control of conditioned behaviour which were constructed on Pavlovian inhibition of delay and (b) the effects of dentato-interposed terposed lesion on the same behaviour. In addition, it differentiated the general effects of cerebral/cerebellar lesions on motor activity such as running wheel from those which are concerned with central nervous system's initiation and control of learned complex motor act (praxis). The exact nature of the transcortical and transneuronal servo-loops awaits future research.

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APPENDIX I

THEORETICAL FRAMEWORK AND CONCEPTUAL MODEL

The present work developed after many years of experimental research in the laboratory of M.F.Halasz. Halasz' work leans heavily towards modern control theory in general, and more specifically the dynamical stability theory in which behavioural systems are seen as self-regulatory in the context of active stabilization. Using the DCA as a behavicural paradigm, he attempts to seek an improved balance between fcrmal adequacy and labcratory relevance in expounding a theory of the homeostatic stabilizatien of behaviour. Actions (or CR's) are seen as systems cf movements coordinated in the function of a result. Acquisitien of a CR comes about as a result of experience, and through the internal process of equilibrium, the expression cf a regulation or an acquired stabilization of coordinative processes. As such, he viewed behavioural oscillatien (and stability) via a qualitative dynamical system theory. That is, a control theory providing a basis for an experimental analysis of the stability of conditioning (Halasz,1968).

The Halaszian scheme is based on:(1) a modern physical model of a contrclled process, (2) an inclusive but explicitly defined set cf integro-differential equations postulated to govern statistical measures of behavioural performance, applied tc, (3) a paradigmatic kind of (neurcmuscular) conditioned activity. (p.2, Halasz, 1974).

Halasz used the elastic spring to personify the disturbance- resistence properties of behaviour. While Freud put more emphasis on the "spring like" restoration of behavioural patterns, Halasz made a finer differentiation between the inertia,the passive tendency of a system to maintain a given state of motion, and the "restoring force",an active vectorial process towards the equalibrium reference state. In addition, the damping constant of a behavioural system which determines the rate of change of a perturbed system was also given a prominant position in describing the characteristics of a behavioural system. When an active behavioural system is thus visualized,it can be analyzed and described by the second order linear differential equation; ie, i P+cP+ kP =0 (P=deviation of a behavioural system from the reference equilibrium i=inertia c=damping constant k= elastic constant)

In the biological and/or behavioural transients,the damped sinusoids or exponential are the solutions to this equation. as Halasz has shown, (1966,1967) the solution to this equation gives comprehensice account of transients in the latencies of the delayed conditioned responses due to disturbance in the CS-US intervals. As such,the generality of the dynamic stability theory renders possible the experimental analysis of behaviour. as on-going,active controlling/controlled processes.

Behavioral variability,as well as uniformity,is accepted as lawful functions of an active biological system.

With the more complex behavioural systems,Halasz employed the concept of the servomechanism. A servomechanism is a control device which adjusts a passive controlled element by means of a sensor which compares the actual output/condition of the element with that of the demand. Its activity is actuated by an error signal formed at the summing point when command and feedback information converge. The relevance of a servomechanism model to the general problem of the stability of biological form and function is at once obvious. It enables the application of concept of control to neurological, physiological and psychological studies.

the optimal process control is an extension of the servomechanism concept,which may be applied to the homeostatic regulation in biology and behaviour. in this context, the sensor,the feedback control and the demand input are all statistic in nature.in the conventional servomechanism framework,the controlled element is a passive device,but in the optimal process control it can be an "operating plant" containing feedback loops of a lower order.the present author considers this model most attractive in the analysis of function and behaviour which include inthem the "neuronal circuits" as a significant variable. in this framework, the sensor takes statistic samples of the performance of the plant. an

index is computed and fed back for control decisions. The variables describing the state of the control element is therefore distinguished from the index describing the condition of the plant. While this fine distinction is not always possible experimentally,it is nevertheless an important consideration in relation to the adaptative, noninvariant and nonlinear systems of control.

According to Halasz,this model gives the best possible physical picture for an essentially mathematical treatment of behavioural homeostasis,because it is the most concordant with neurophysiological knowledge,with the statistical nature of behavioural measurements and the plasticity and adaptivity of conditioned responses(Halasz,1973). It gives rise to the concept of control policy,ie,the "improvement of the behaviour of systems" which "best renders theoretically the characteristics of CR"s considered as motor skills or "praxis". The CR"s are conceptualized as the paradigmatic behaviour in the treatment of homeostatic stabilization. These behavioural systems can be studied mathematically by identifying them with "optimal process control & coordinated with control policy". In this framework,the controlled process block is the executive apparatus of the skill. It includes muscle fibers,spinal neurons,motor cortex ,anterior cerebellum,etc.,and is in turn subserved by lower order servomechanisms. The object of regulation is an activesystem including the CNS,the effectors and the environment. Environment refers to the fixed limb of the

reinforcement programming apparatus within which the motor skill is exercised.Perfcrmance measurement is the ongoing activity og the brainthrough sense organs,memory circuits etc. The internalized feedback processes are made explicit via the installation of experimental apparatus such as counters, timers, etc."While the anatomy and physiology of the brain's own performance measurement over it executive plant is so poorly understood.... it is the internalized content that corresponds to elements of 'praxis'"(Halasz,1973).The state space of a CR is "the conceptual theatre for the operation of habit as control policy"(Halasz,1973).It is the meeting ground of the condition of performance, the demands of the external environment and the available control actions. Eccles(1967) hypothesized the possible existance of such an area in the cerebellum at which exteroceptive, proprioceptive and executive pathways intersect. Halasz, on the other hand, devoted his energy to the construction of the conceptual theatre with electronic hardware and software. The principles thus derived were taken as to provide the theory to appropriate internal processes.

At the simple level, a state-space can be represented by a "phase-plane" which specifies the momentary state of performance by the level of a response and its tendency to change (ie., the first derivative).

At the more complex level a state-space is statistical because every point in the space is the status and

tendencies of a frequency distribution. This is so because the actual condition of skilled performance at any time'T' is a frequency distribution of that performance measure which can be described by a set of statistical movements Pi in an empirical performance sample. Mathematically is :

$$dpi/dT=fi(Pi,Pi) \qquad (1)$$

$dpi/dT=gi(Pi,Pi)$, $i=1,n$ (2) The trajectory (ie., the sclutions to these equations) describes the history of the performance. When the performance is not affected by the action of the demand generator, the equaticns represent the auto-determination of the behaviour as influenced by its cwn internal structure.

When the performance is driven by external demands $di(T)$, the equation becomes

$$dpi/di=gi(PiPi)+di(T) \qquad (3)$$

Because of the statistic nature of decision process, the $di(T)$ is likely a discontinuous step-wise increment of the reinforcement contingency.

When the performance measure is an error function, Pi is an error index. The origin of the (Pi,Pi) phase plane corresponds to performance perfectly adjusted tc the demand and at equilibrium there. The current condition of behaviour (as represented by the radius vector from the origin to the point) gives a display of discrepancy between the actual and desired state of the

praxis.

At the higher-crder ccntrol systems the control signal(mi) can be experimentaly applied or removed relative to the state- space related discrepancy display.This signal is applied over and abcve the restoring forces essential in the auto-determined stabilization of performance. Mathematically, it is

$$dpi/di=gi(Pi,Pi)+di(T)+mi \qquad (4)$$

mi is generally binary (0,cr 1),ccnforming with optimality principle cf ccntrcl cr "bang-bang" control (Tomvic, 1966). Briefly, the bang-bang control action (Pi,Pi) utilizes strategical switching in and out of mi in such a way as to make the conditicned action most rapid and stable.As the trajectory intersects the switching line, correctional mi is activated. The position and the angle of the switching line describes the ccntrol policy in force.(A vertical switching line means that mi will be applied when error index Pi has exceeded a tolerated level, an oblique line states that the derivative Pi, or anticipation is included in the process of control decision.The optimal control policy can be derived mathematically for a system if we know its governing equations. On the behaviour level, however, one is confronted with the inverse problem. Various control policies are empirically constructed to probe a behavicur system. The optimal policy is empirically derived via experimenatal manipulations to provide some insight in

the unknown equations.

When the application of mi (correctional actions) is made functionally dependent on a segment of trajectory (a finite history of a behavioural system),the principle of optimality plays a role in the control policy(that regulate performance about a reference pt. in its state-space). Under this condition, the quadratic criterion is used to introduece an integral equation which is necessary to compute the value of ranges of T over which mi is in action.(Note:The quadratic criterion states that a function

$$F(Pi+Pi)$$

of the squared radius vector described by the system about the origin should be minimized or held to a set value over a trajectory segment).

The concept of optimality provides a vital and qualitative understanding of system oscillations.

Based on the Halaszian model of behaviour, a conditioned habit is defined by a policy of control over skilled neuromuscular performance. As such,the equations (4) and (5) represent one of the states of the skill which may fluctuate over time. To describe more fully the skill, together with its variations, the forms of equations (4) and (5) must parametrically be dependent on state variables

$$sk=1....m$$

$$dpi/dt=gi(pi,pi,sk)+di(t)+mi(f(sk)dt) \quad (5)$$

the sk's are located in the control system block and specify the adaptative dimensions which the brain possesses over behaviour, and over the external stimulation. If the process cntrol of a CR is complete in the S the sk's are neurophysiological and are related to the plottei (reversible state changes associated with factors such as mctivational or circadian rhythms) CNS dimensions. If, on the other hand, the external apparatus is used to simulate the higher CNS control, sk's can mean the adjustable decision criteria in the regulatien (Halasz & Cheng,1969). In a S-(O)-R paradigm, the sk's belong with the O variables while the pi(the state of the controlled neurcmuscular process) belong to the R variables. Thecry cf nonlinear oscillations

As pointed out by Halasz the sk's and their equation

$$dpi/dt=gi(pi,pi,sk)+di(t)+mi(f(sk)di) \quad (6)$$

provide the dynamic basis for conceptual application of the thecry cf nonlinear oscillatien to the understanding of behavioural fluctuations and homeostasis. Fixed State of Praxis=perfcrmance oscillatien about an equalibrium poin. When the control policy is fixed,sk's being constant the equatien (6) maintain a specific form. The conditioned performance pi's may fluctuate around an equilibrium point (pi=0). Several types of fluctuations may occur: (a) The stockastic jitter is an artifact of

statistical estimation, which does not bear on the deterministic aspect of behavioural fluctuation, (b) Lawful oscillation of a behaviour about the equalibrium may take twc forms when the system is perturbed by a demand signal .If the restoring force is such that the trajectcry returns to the initial point, it is stable. If the trajectcry departs indefinitely from the initial point, it is unstable. Fixed state of Praxis - oscillation during transition between equilibrium points.

Due to a change in demand a formerly stable equilibrium point O' may beccme unstable. The behaviour diverges from O' and heads tcward a new level at O and gradually ccnverges tc it in a tightening spiral.This equilibrium pcint is called a focus. In the time domain this phase plct corresponds to a decaying oscillation. If the gi's are linear the spiral is a damped sinuscidal.

Fixed state cf Praxis- limit cycle :

The oscillation of a ccntrclled process which neither decay with the passage of time, nor result from any loss cf control are called the limited cycles. The limited cycles are fully deterministic, maintained cyclicities.In this condition, the statistic parameters of the response measure dc nct converge tp fixed values but to an oscillation with a definite period. If the cycling system resists attempts to disrupt it it is called a stable limit cycling. Like other forms of behavioural measure, it is a mode of operation of the behavioural system permitted by the regulatory policy in

action.

Variable damping terms, (pi,pi),gi's, retarded action by the mi, and the inertial nonlinearities (associated with the integral terms of the optimization criterion) may all contribute to the possibilities of LC oscillations. The initiation of a limit cycle can be soft or hard, and once a limit cycle is attained the antecedent history and manner of excitation no longer has consequence over subsequent events.

Evaluation of Praxis :

The solution portrait of a CR remains fixed in so far as the state of the skill remains fixed.If on the other hand, the skill itself undergoes evalution,the process of transformations has significant consequences over the potentiality and the actualities of conditioned performance. As the invariance of CR performance can not be assumed under all conditions, the variables relative to the evolution of a conditioned performance hold significant implications for the study of biological behavioural systems.

In the context of the systems theory, topological dynamics, bifurcation theory and parametric excitations are three of the significant concepts pertaining to the consequence of the noninvariance of the policy of control. As presented in Halasz' publications (1968,1970) all the three concepts are highly relevant to the study of behaviour in an operant lab.The present study is indebted to these theories for the conceptual understand-

ing of the behavioural systems both in fixed and in evoluting phases of a CE (a)The topological dynamics concept shows that in the domain of the evolution of behavioural possibilities, the solution portraits are qualitatively presented in the form of the topological profile of the indicated steady-states. For instance, the profile of the particular system may remain unchanged for a wide range of sk's. However, when certain critical value is exceeded the solution configuaration may involve a discontinuous transition into a different one.

(b) The bifurcation theory entails the rules according to which a differentiation equation evolves in a continuous way. It concerns with the general guidelines by which the new LC's and O's arise in pairs from old onesas sk's in equation (6) pass the critical values. For instance,as implied in Halasz' 1970 paper, a stable equilibrium point bifurcate into an unstable equilibrium pointand a LC as a parameter of the governing equation changes.

Parametric excitation deals with the phenomenon of forcing of oscillation by a periodic di(t) input under condition of de(6).

With a change in the state of the control system, oscillatory behaviour may occur for no apparent reason. The parameter excitation concept relates this phenonenon to the forcing of oscillation by a periodic di(t) input under condition of invariance in (6).One example is the periodic parameter variation may be interchangeable with

an external fluctuation of demand conditions.

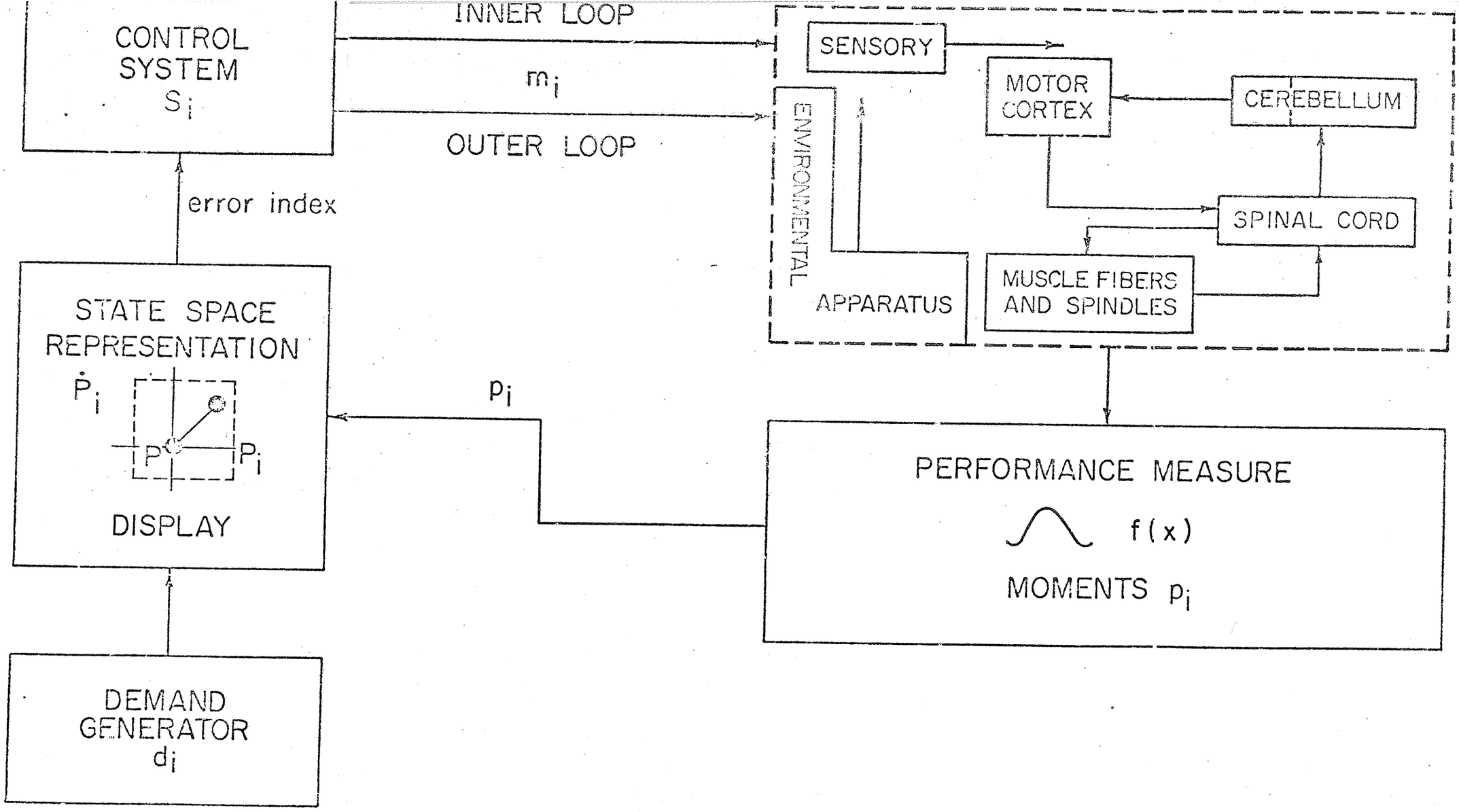
Based on these three concepts, stability is a continuous characteristic of solution profiles when the noninvariance of the governing equations are a possibility. More over, it is perforce a property of actual performance because the stability of an equilibrium point or cycle can be specified only with respect to what happens to an actual behavioural system occupying or or traversing it.

Based on the foregoing presentation, dynamic stability theory has significant bearing on the design, analysis, interpretation, and hardware of the behavioural experiment. For instance, within the frame of reference, the relevant trend in the mode of variation of experimental variables is the shift from static, factorial levels of treatment to dynamic, time dependent input forms. This shift comes easily in the operant lab because its approach is to study behavioural processes on a causal process in time (and not on a set of data crrelations).An experiment consists of a probe of the ongoing controlled process by demand inputs, alteration of state-parameters, changes in the optimalization criteria, etc. The hardware, the design and display of experiment on neuromuscular performance is also refined from manipulation of a single variable and that of a feedback loop. In analysis of data, stochastic/ deterministic partition and stable oscillation are taken as lawful functioning of systems rather than as

noises/contamanants.

Overall, using the control-theoretical model, Halasz re-apprcaches homeostasis in psychology as pertaining to the structure of behavicur rather than only to motivational cycles that energize it. Utilizing the mathematical definitiсn of habit, the CR's are seen as a way of management over performance rather than of performance itself. The praxis consists of a definite way to relate behaviour output to demand input.

Figure 1. Conditioned praxis as an optimally controlled process (Halasz,1973).


CONTROL SYSTEM
S_i
INNER LOOP
m_i
OUTER LOOP
error index
STATE SPACE REPRESENTATION
$\dot{P}_i$
P
P_i
DISPLAY
DEMAND GENERATOR
d_i
p_i
SENSORY
MOTOR CORTEX
CEREBELLUM
ENVIRONMENTAL
APPARATUS
SPINAL CORD
MUSCLE FIBERS AND SPINDLES
PERFORMANCE MEASURE
$f(x)$
MOMENTS p_i