A TREATISE ON PSYCHOLOGY AND VOLUNTARY MOTOR BEHAVIOR WITH AN INVESTIGATION OF BIOFEEDBACK EFFECTS ON HEMIPLEGIAS RESULTANT FROM CEREBROVASCULAR ACCIDENT

A THESIS
SUBMITTED TO THE FACULTY OF THE GRADUATE SCHOOL
OF THE UNIVERSITY OF MINNESOTA

by

Nancy Stephanie Mroczek

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

March, 1976

ACKNOWLEDGEMENTS

Dedication

To my mentor,

Dr. Halpern

At the outset of our relationship I thought that I could learn alot from you, but I underestimated how much that would be. Every student should be as fortunate as I was, to have had a teacher, counselor, and friend like you. There was no point on any issue or perspective which I could not bring to you, feeling welcomed, and, further, walk away from you, feeling inspired, guided, and corrected. What an error-feedback signal you were for me! (Fortunately, such a kind one too.) You educated me in physiology, the brain, and muscles, and attuned me to the concepts, interpretation, and meaning of biological phenomena. Besides the extensive education, you showed me how to treat people by your example.

It does not suffice to say that you bent over backwards for my project. I have watched you act similarly for all causes you thought worthy of pursuit. It was a period of vigorous growth and privilege to have worked with you. How indeed, I shall miss you.

Thank you for the countless opportunities to argue (you were always willing and inevitably adept with reason), listen, (I was often just absorbing loads of new things) and learn, (with each step, approximating better comprehension in my sphere of inquiry). Whatever the future problems, this knowledge and experience will avail me.

Thank you also for your willingness to engage in other people's enterprise and thank you for offering your work in the interests of other people.

Oh, let's just get down to it: thanks for the raps, and just plain thanks for everything, pal. We had a good time, heh?

Always and humbly,

Nancy

First and foremost--there are not even the words for it--acknowledgements to my parents. Thank you for everything you gave me. You gave me love, security, toleration, and cooperation, (though you did not show me how rough things could get at times!). You are my first source of humanity. I hope that I am able to consider, as you did, the needs of love, guardianship, and example, which are shared by both children and adults alike.

Together with my parents, acknowledgements to my sister, Alaina Mroczek, who upon my beckoning for help, came out to Minneapolis to give me a hand in the current undertaking. Initially the situation seemed to indicate a need for some aid now and then to facilitate a smooth, coordinated study. Well, studies don't run smoothly, especially at incipiency, there is no end to the work until the final paper, and there are weeks when the work is overwhelming. My sister arrived in such weeks and offered the free consultation that what I really needed was a full-time worker. She volunteered to be that worker and kept me abreast of the constantly proliferating nitty gritty details which involved multifarious

facets of project organization, tremendous amounts of data compilation, and small but heavy problems like patient transfers. She managed to endure, with true devotion to me, the study, and the patients, a couple months stay of long days of hard work. When she came, I was knee deep in the muddy. When she left, I was above water and sailing. What can I say?

Finally, a warm thank you to my many instructors in settings everywhere, who educated me, and who gave me pride and inspiration for my calling.

It is so true that I "get by with a little help from my friends ...". And at the University of Minnesota I met so many good people--people who opened their doors, cabinets, minds, sympathies, talents, and honest to goodness labor to my project:

Mary Moore, PM&R, who helped me with the beginning of a protracted search for patients;

Helen Barrett, PM&R, who directed the comings, goings, and in-between mishaps of patient traffic to my study;

Steven Haig, PM&R, who, as a favor, helped me with patient transfers;

Nancy Lutz, PM&R, who repaid my "patience" during her work, with extra hard help whenever time was available;

Mr. Hole, Audiology, and Suzannah Gorke, Neuropsychology, who tested my patients and who were both gracious to the patients as well as the many questions from E;

Larry Stradal, PM&R, who came, time and again, on a moment's SOS to help with equipment problems.

Elaine Burnhardt, EMG-Neurology--Elaine, I cannot say enough to you. You did everything, and you were with me all the way. First, but least importantly, producing the most competent work anyone could ask for; secondly, doing the many many favors; and thirdly, offering the shoulder on which I could unload my chronic "woes"; (I hope I lent you the same). It was nice to know you as a friend, Elaine. Thank you.

Bill Iacono, a student colleague, who in beautiful comaradarie, was a friend, and who also tried to aid my electronic savoir faire in a course long ago.

Lee Brownstown, a student colleague and friend, who helped me in so many ways. You were a sounding board and a feedback signal, listening and keeping me on base. You also extended many many favors, graciously. We also had those many pleasant walks together. Merci, Lee!

Finally, in consulting the many authorities whose expertise was invaluable to this study, I was fortunate to have intersected with the operations of men guided by principles, humanitarianism, and true clinical concern--men who seemed to hold the title, doctor, with dignity.

Dr. William Kennedy (with whom I "apprenticed" in the Neurology EMG lab for 3 months when the study was but an indefinite idea)--Dr. Kennedy, I am so happy for having had the experience of "apprenticing" with you. Being around you, I was able to observe a superb clinician as well as admire an implicit code of humanitarian treatment. Of course, I was also able to acquaint myself

with motor problems, neurology, and electrodiagnosis which enabled me to formulate a definite study. I enjoyed, benefited, and came away perhaps a bit more understanding in general, after my exposure to you. Thank you, Dr. Kennedy.

And Dr. Alan Rubens (who carried out the neurological examinations on my patients)--my acquaintance with you was brief, but again inspirational. From watching you work, my impression was one of great competence, integrity, and human concern. It is also in admiration that I acknowledge you and your help.

There were so many experts with whom I did not work, but who, all the same, gave me time, financial assistance, and their expertise in discussion on various facets of the study. They also pitched in to help when the help was needed.

Dr. Manfred Meier--thank you so much. You first invited me to carry out my proposed study on the CVA grant. You further consulted with me on MMPI's and other questions, and gave me your lab facilities for neuropsychological testing. Most importantly, however, thank you for your spirit and rapport. Every opportunity to talk with you and exchange ideas was a genial and satisfying occasion.

Dr. Paul Meehl--I came to you with an inquiring mind and when you listened, I felt I'd better scurry to tell you. And so I wrote some sections of my paper. To say the least, I feel fortunate to have come into contact with your intellective character--lamentably late, but better late than never.

Dr. Richard McHugh (with whom I collaborated on Statistics)--Oh, oh, I don't know what to say to you with all the bureaucratic

and fiscal problems you took on to see my project through. You also provided me with astute academic guidance, and I give you a hearty thank you.

Dr. Joseph Resch, CVA grant treasurer, who had confidence in E and who promoted the welfare of this study by endorsing the monies fundamental to its actualization. It was also a pleasure to know your thoughtful demeanor, Dr. Resch.

Dr. Robert Thomas--How many times did I call or see you about questions on neuropsychological tests? And how many times did you always answer my questions, as soon as you had time, in the most satisfactory and complete way? That was nice and it was important to me. Thank you.

Dr. Alan Roberts--Thank you for your advice and encouragement. Although we did not get to see each other much, I appreciate the concerned consultations you contributed to my efforts.

Last, yet figuratively and literally the first, to those of the experts who have helped me, I pay acknowledgements to Dr. Travis Thompson, my psychology advisor during my entire stay at the University of Minnesota. It happened that we rarely were able to chat as we were far afield each others research pursuits. But it also happened that I saw someone whom I could respect and who showed me by action, the idea of principle at work. You were very good to me; I think of the help you gave me both in terms of obligation and respect. I have high regard for you. Thank you.

And now, I have to add a perrenial plea. That is, that the "system", being what it is, did not make it possible for me to spend more time in discussion with each of you.

Finally, surveying the passage, I have garnered this: whenever I have come into contact with professionals who were exemplary, I could not help but mark their impression on me. I believe that learning some knowledge can best be brought about in an environment where it is possible to emulate exemplary people. Besides, there is much to learn in this world other than some specific knowledge, and the process of knowledge acquisition is most enjoyable when minds are in harmony. Furthermore, knowledge in action can be attractive, inimical, or just plain frustrating if words say more. In conclusion, I would also say that I learned more by experience--that is by living and talking with people both in and out of academic settings--than I learned from any textbooks, though I prize that which books have taught me dearly.

Finally, a thank you to my patients, each of whom was only too happy to help, and each of whom added to E's capacity as a psychologist and a human being.

Grant Acknowledgements

This investigation was supported in part by Research Grant NS-3364 from the National Institutes of Health, Public Health Service. It was carried out at the University of Minnesota Regional Rehabilitation Research and Training Center, RT-2. It was also supported in part by Rehabilitation Services Administration of the Department of Health, Education, and Welfare Grant 16-P-56810.

Table of Contents

PREFACE

Feedback properties of psychological and physiological systems have been receiving increasing attention of late. Feedback might be defined as information an organism receives as a result of some response. Information is used by the organism to the extent that skilled control or new contingencies are uncertain or not pre-programmed. Biofeedback (BF), a special feedback technique involving electronic monitoring of a subject or patient's bodily states in order to make such information available to him, is a fairly recent experimental and applied development. The mechanisms of BF in this and other studies, are, to a great extent, conveniently subsumed under general principles of feedback.

It is important, however, to emphasize that the current paper does not expose or utilize feedback to denote a fixed mechanism requiring literal acceptance as <u>the</u> explanation of motor behavior. It is, rather, a conceptualization or framework exploited to highlight the dynamics of organismic activity at <u>any</u> level and, further, to foster an interdisciplinary perspective of searching for relationships broadly perceived as those between input and output, and specifically in this paper, as relationships between the workings of the sensory and motor systems at the intra and extraorganismic levels of activity. In sum, feedback affords psychology, as well as other disciplines, a contemporary principle around which ideas about human motor behavior may be developed.

Sensory feedback, or sensation at some level, is required for muscular control. Hence, the cyclical, reafference, feedback frame of reference within which motor patterns may be studied. Sensory events which enter awareness are felt, recognized, apperceived, ideated. The motor is not perceived except through some sensation. It is performed. The sensory, at some level, is conscious behavior. The motor--i.e., the process of efferent activity--is unconscious behavior.[1,2]

At the subjective experiential level, the feedback process may be delineated as concrete physical receptor transmission to efferent pathways whose output establishes reafference, and so on. On the other hand, as oftentimes is the case, where the motor feedback process cannot be described by conventional concrete peripheral physical descriptors, it may only be inferred as internal corollary discharge.

---

[1]Pertaining to the ontogeny of sensori-motor experience, see, e.g., Held & Hein, 1963, or Nissen, Chow & Semmes, 1951.

[2]Conversely enough, a verbatim ascription of meaning to "sensory" or "motor" events is astutely allayed in a quotation from Magendie (1824) cited by Evarts (1971)--(The reader is also referred to Towe, 1973, on this matter.)--in an article on feedback and corollary discharge. (The latter concept is discussed in future sections.) Evarts sagaciously proposes to give "the last word to the man [Magendie] who said one of the first words on this topic" and quotes from Magendie as follows:

> The organs which concur in muscular contraction are the brain, the nerves, and the muscles. We have no means of distinguishing in the brain those parts which are employed exclusively in sensibility, and in intelligence, from those that are employed alone in muscular contraction. The separation of the nerves into nerves of feeling and nerves of motion is of no use: this distinction is quite arbitrary. (pp.111, 112)

Or, alternately, it may be, and often is, described as intention--intention followed by a comparison between what is intended with what is achieved. Corollary discharge is a concept, which like feedback, is currently in vogue, particularly in feedback-oriented discussions. Intention, on the other hand, is a description of human behavior which cannot be formally included as part of a physicalistic terminology. Yet it frequently appears in scientific writings, the goal of which is a deterministic analysis of motor behavior. The same is true of the words "voluntary movement".

Intention and voluntary movement are not mere vestiges of outmoded thought, nor the remnants of unexplored or unexplained areas of physical activity. In fact, as more is known about the nervous system, they are oftentimes the most fitting descriptions of behavioral phenomena. Such descriptions of behavior, of course, fall within the scope of teleological explanation. Woodburne (1967), a psychologist who has written a text on the neural basis of behavior, comments on teleological explanation as follows:

> Some readers may be disturbed by terms which impute purpose to the activity of the nervous system. The use of such terms is meant to imply the adaptation of an animal to its environment, or the natural selection of certain subfamilies which possess neural structures allowing better adjustment to particular environmental conditions...[In discussion of the central control or modulation of sensory input], no other basis has been found for the selective monitoring of the input which is allowed through to higher centers than what appears to be significant to the individual at that moment. This meaningful significance is certainly a teleological mechanism. (p. 10)

In relation to such a conclusion, it may become evident from the following discussion of motor control, feedback, and biofeedback, as well as case histories resultant from this study, that a source of control, or cognitive activity is important to motor behavior. Motor

skill in evolution involves "patterns which are progressively precise in their aim and purpose" (Ingram, 1970, p. 53). Furthermore, as practice increases, so does variability.

With respect to intended, voluntary movement and such variability, Luria (1973) has remarked as follows:

> The initial component of human voluntary movement and actions is the intention or motor task which, in man, is hardly ever a simple, direct response to an external stimulus (only the simplest forms of firmly established, habitual action still remain in this category), but always creates a 'model of the future need', a scheme of what must take place and what the subject must achieve or, to use Bernstein's term, the 'Soll-Wert'.
>
> This motor task or model of the future need, is constant or invariant, and it demands an equally constant, invariant result. For instance, if the motor task is to go up to a cupboard and to take a tumbler, or to hammer a nail, the fulfillment of these acts is the constant, invariant result by which the action is completed. However, it would be a mistake to imagine that the invariant motor task creates an equally constant and invariant programme for the fulfillment of the required action. It is a most important fact that the invariant motor task is fulfilled not by a constant, fixed set, but by a varying set of movements which, however, lead to the constant, invariant effect. This thesis applies both to elementary and to the most complex motor systems. (p. 248) (Author's quotation marks and underline)

The current paper quotes the above, with reservation; hopefully that the above does not render an exaggerated emphasis to efferent variability or response heterogeniety. (Nor does Luria intend such emphasis.) It is important, however, to emphasize variability as a counterpoint to equally valid and important observations of response generalization, inasmuch as the latter are often the sole aspects of movement which receive attention, or around which argument and experimental analysis proceed.

The current thesis does, however, accord high regard to the role of intention in voluntary movement, and, at the same time, esteems the analysis and description of behavior in terms of intention and voluntary movement as appropriate to psychological discipline. For

current purposes, this can be explicated in two ways from two examples of contemporary types of treatment of psychological phenomena: (1) attempts at description of psychological phenomena in terms of physiological phenomena or, (2) attempts at description of psychological phenomena directly patterned after description in physical science. To explicate:

(1) biological processes are correlates and not causes of psychological activity;

(2) physical science may serve as a model for psychology in the sense of exemplifying the process or procedure of how laws are reasoned, formulated, and established--for example, how facts are interpreted by means of substantial reason as well as the process of reason or inference, and the use of assumptions, logic, etc. to arrive at causality or explanation. But, physical science is not, however, a model for psychology in the sense (literally taken) of its treatment, description, and definition of the properties of the systems it studies.

Pertaining to (1), for example, neurophysiological explanation is appropriate to neurological and physiological processes which, although correlated with psychological processes, do not share identity with them. Pertaining to (2), for example, traditional physical science is appropriate for explanation of the behavior of nonliving materials. Explanation in psychology, patterned after traditional physical science in the sense of elucidating only the physical properties of a system, fails to explain the lawfulness or regularity of the behavior of living material, i.e., as pursued here, the psychological behavior of living material. The search for lawful relationships or observed

regularities of the behavior of inorganic vs. organic matter requires different levels of discourse as well as different qualitative description of the physical and psychological systems respectively.[3]

In conclusion, the foregoing posits an analysis by synthesis of current knowledge in motor learning. It also posits that such a synthesis illumines not only the role of an organismic processor, but also the fact that there is a processor capable of consuming information to continually effect and regulate consequences which are germane, reinforcing, or indigenous to organismic activity.

Words such as intention and voluntary movement are used in the description of psychological process--in the current research, the psychology of the learning process of motor control in brain-damaged adult victims of stroke--without any implication that a reductionist definition will eventually confer meaning to these terms. Hopefully, this thesis will approach some convincing descriptions of the essential attributes of intention and voluntary movement as well as have advanced, by example, a rudimentary argument as to the validity of the phenomena so symbolized by language.

---

[3]Computer or information processing descriptor terms and models are also currently receiving a good deal of attention. They are also at best metaphorical or analogical frameworks for the study of certain organismic behavioral functions. They cannot, however, provide, by mechanistic account, explanation of attitude, intention, concentration or self-generated, purposeful behavior.

SECTION I

INTRODUCTORY NOTE

The introductory review to the current research will consider voluntary movement, feedback, and biofeedback in motor function. The review entails (1) basic anatomy and neurophysiology of the motor system; (2) voluntary movement and sense of effort; (3) receptor modalities for voluntary movement; (4) levels and types of internal and exteroceptive feedback; (5) possible systems and mechanisms involved in motor control; (6) feedback approaches to the study of motor control; and (7) a survey of the biofeedback literature on muscle control. The latter is a group of recent studies antecedent to the current study which investigated the technique of biofeedback on stroke patient hemipareses of the upper extremity as a substitute link for interrupted feedback routes.

SECTION II

THE NEUROMUSCULAR SYSTEM

Peripheral Activity

The basic unit of the skeletal neuromuscular system is the motor unit consisting of an anterior horn cell neuron, the peripheral axon process of the neuron with its numerous terminal branches and the muscle fibers innervated by these branches (motor unit neuron cells supplying the head emerge from the brain stem). Those motoneurons making up the motor unit are classified as alpha motoneurons [(A) α]. (Motoneurons called gamma, which also leave the anterior root, will be discussed further.) Motor units are functionally distinguished from one another since all of the fibers innervated by one motoneuron contract practically synchronously. Furthermore, although muscle fibers within one muscle may differ histochemically, a given motor unit consists of muscle fibers which are histochemically identical.

The whole muscle is made up of many muscle fibers. The junction between an axon and a muscle fiber is called the motor end plate which is located in the middle of the muscle fiber. The muscle fiber is from a few mm to tens of cm long in man and less than 0.1 mm wide. The number of muscle fibers innervated by one motoneuron can range from 2-3 up to 2000. The ratio of fibers to motoneurons is generally increased for muscles involved in gross movement (e.g., posture muscles, calf muscle) and decreased for those involved in delicacy of movement (e.g., finger and ocular muscles). The functional unit of contraction within the muscle fiber is the myofibril which is composed of many

tightly packed thick and thin myofilaments of myosin and actin arranged in parallel. These are thought to slide into one another to cause contraction of the muscle fibers (Goodgold and Eberstein, 1972).

Conduction of the electro-chemical potential in muscle fiber is analogous to that of neuron conduction. The resting membrane potential of the fiber is -70 to -90 μv (interior negative, exterior positive). When a nerve impulse arrives at the motor end plate, acetylcholine is transmitted across the neuromuscular junction. If the membrane potential reaches threshold voltage, an action potential is propagated in the muscle fiber and muscle contraction results.

A given muscle is composed of many motor units. Muscle tension is due to the number of units firing as well as to the frequency of unit firing. Thus, the least possible tension involves only one motor unit firing; with increased effort an increasing number of units is brought into play. The latter phenomenon is termed recruitment. Fibers within a motor unit fire practically simultaneously while motor units within the muscle fire asynchronously to effect overall smooth muscle contraction. A contracted muscle fiber shortens to 57% of its resting length.

The electromyograph (EMG) recorder is an electronic unit which records muscle potential signals visually and also usually acoustically. The record of the EMG is commonly displayed on an oscilloscope. With very slight muscular effort, a single motor unit (SMU) action potential (intramuscular electrodes) may be seen on the oscilloscope as a di- or triphasic wave of 3-16 msec duration with an amplitude ranging from 300 μv to 5 μv. The rate of firing may be from 5-15/sec. If effort is increased substantially an increased number of motor units firing as well as an increased firing rate of individual units produces

what is called an interference pattern. Here SMU's are no longer distinguishable and the display consists of dense activity with multitudinous spikes. In a completely relaxed muscle, no activity is observed on the oscilloscope.

SMU activity is recorded from a S who is contracting weakly and attending to his effort. Some muscles are more amenable to SMU control than others (e.g., thenar eminence, thumb muscles, as compared to flexor carpi, hand flexor, muscles). The precise display of the motor unit potential wave form (shape, duration, amplitude) depends on several factors: size of electrode; type of electrode (surface, intramuscular, etc.); equipment used; distance of the electrode from active muscle fibers; and the number of active muscle fibers being recorded from. The frequency response range of EMG potentials is from 2-10,000 Hz.

When a muscle is stimulated by a nerve or directly, two types of muscle contraction may result--isometric or isotonic. "If the ends of muscle or its tendons are fixed so that the muscle cannot shorten, it will develop tension. This sort of contraction is called isometric" (Mountcastle, 1974, p. 85). During this type of contraction there is no measurable external work. If only one end of a muscle is fixed, it shortens when stimulated. "When the free end of the muscle is made to lift a weight or to oppose a resistance held constant during contraction, the contraction is isotonic" (Mountcastle, 1974, p. 85). During this type of contraction there is measurable external work.

Central Nervous System Activity

At the central nervous system (CNS) level, motor organization is hierarchical and mainly contralateral. Spinal, subcortical, and corti-

cal centers all play a role in effecting smooth motor performance. The cortex, basal ganglia, cerebellum, brain stem, and spinal cord are the basic functionally descript centers involved in neuromuscular control. It must be emphasized, however, that all CNS structures for motor function have both efferent and afferent connections. The same may be said for the sensory system so that sensory and motor systems are not functionally distinct. In fact, the extensive intercommunication between the two systems makes it difficult to distinguish them for experimental purposes. Further, increasingly refined analyses of either makes their distinction increasingly lacking in outline.

The motor area[4] (area 4) of the cortex, which contains the fifth layer of the giant Betz motor cells, is primarily responsible for the control of fine voluntary movements of single or small muscle groups. Its projections fade into the somesthetic area and vice versa. The motor cortex is the site of initiation and transmission of impulses to muscle. The pre-motor area of the cortex (area 6) just anterior to the motor area, seems to be responsible for integrated, dynamic functioning of motor elements of a rhythmic, serial skill nature (i.e., "kinetic melody" patterns of movement [Luria, 1966]). This area also seems necessary for evaluation of performance in conjunction with intended movement.

There are two motor pathways from the brain to muscle, the pyramidal tract and extrapyramidal system.[5] The former (40% of which originates from the two cortical motor areas) seems to be the more

---

[4]The whole cortex is largely sensorimotor there being no absolutely distinct sensory or motor areas.

[5]The pathways from brain to muscle are conventionally described as being organized into two systems. Hitherto clear distinctions between these systems are gradually being diminished by recent work.

direct route from cortex to spinal cord, innervating especially limb and hand muscles, and, in particular, those muscles which have few extrafusal fibers per motor unit. The extrapyramidal system has connections with many subcortical structures and is believed to organize automatic, postural, and highly skilled movements. It supplements the pyramidal system by refining and smoothing out movements and operates by integrating and modulating gross motor and postural reflexes.

The basal ganglia are a subcortical group of nuclei which, albeit poorly understood, are important in controlling automatic or rhythmic movements. The basal ganglia have connections with both the cortex and brain stem structures. Some abnormalities developing from destruction to parts of the basal ganglia are chorea, hemiballismus, athetosis, and Parkinson's disease.

The cerebellum, most notable in terms of feedback is best described as a mediator between other cerebral structures and the muscular system of the body. It receives input from the cerebral cortex, the brain stem, and sensory receptors. For example, there are cerebral-cerebellar feedback loops and brain stem-cerebellar feedback loops. The cerebellum monitors both intended movement and body position in space and can mediate or regulate the adjustment between efferent and afferent signals via its feedback loop channels. Damage to the cerebellum results in tremor, overshooting, ataxia, etc. which are assumed to be unmodifiable by voluntary control. (Where damage to the cerebellum has occurred, it would be interesting to see whether any progress might be made with biofeedback.)

Finally, both the brain stem and spinal cord are important in reflex motor function. The brain stem is an area of integration for

sensory and motor information. It is especially important for reflexive posture adjustments (i.e., of antigravity muscles). The reticular formation also seems to exert control (facilitory and inhibitory) on gamma efferents (gamma discussed in the next section). The reticular formation has several feedback loops. For example, excitatory sensory input to it feeds to the cortex and back to the reticular formation to re-excite the cortex. The reticular formation also has loops to the spinal cord to facilitate or inhibit muscular response where proprioceptive impulses, in turn feed back to the reticular formation. The spinal cord is the locus of many basic reflex behaviors as well as the final common pathway to motor output from higher centers.

Higher centers govern lower centers in motor function. However, as motor behaviors become well-learned they seem to be "stored" as subroutines at different levels of the motor hierarchy. Although higher centers may direct movements, their complete execution will depend on all motor levels functioning appropriately. Recent evidence (Evarts, 1974) suggests that both the cerebellum and basal ganglia are not necessarily to be considered "lower centers" in the motor hierarchy. Neurons in these "centers" fire prior to movement culling information from other impinging areas of the brain. It is quite plausible to take the view that these structures send commands and not only corrections to the motor cortex via their massive interconnections with it, whence pyramidal tract neurons consequently direct impulses downward.

The Muscle Spindle and Gamma

As already mentioned, somesthesis (and, in particular kinesthesis) is very important to normal motor development and function. There are

two kinds of somesthetic receptors. The first group is cutaneous receptors which are receptors for light touch, touch, and pressure. The second group of receptors is called proprioceptors from the word "proprioception" which was introduced by Sherrington (1906) to include all sensations arising from: a) muscles which contain receptors for muscle length and rate of change of muscle length, b) tendons which contain receptors for tendon tension and state of muscle contraction, c) joints which contain receptors for rate, direction, amount of joint extension and position of joint at stabilization, and d) the vestibular system which contains receptors for sudden movement changes and the position of the head in space. Proprioceptors transduce information on one's "insides"--the state of muscle contraction, tendon tension, joint angulation, and body movement and position in space. Kinesthesis is often used interchangeably (and confusingly) with proprioception in the literature. Kinesthesis is more equivalent to a psychological discriminative capacity dependent upon proprioceptive cues.

The muscle spindle, a proprioceptor, is the unique muscle receptor. An intramuscular structure, it is often manipulated or related to psychological and physiological aspects of motor behavior. At the same time it provides a rather explicit, relatively well-understood model of neurophysiological feedback.

The muscle spindle is a mechanism with both sensory and motor components. It is a fusiform structure, 7-8 mm long, attached in parallel to ordinary skeletal muscle fibers, hence to be called extrafusal fibers. The muscle spindle itself encapsulates 2-8 muscle fibers called intrafusal fibers. The intrafusal fibers are at the poles of the spindle, and receptor organs are in the middle of it. Intrafusal fibers are of two types: nuclear bag, whose nuclei are found in an

equatorial distended region, and nuclear chain, whose nuclei are arranged single file. Muscle spindle receptor endings are of two types: annulospiral (or primary) endings which innervate middle fiber regions and flower spray (or secondary) which are found close to but spread out from the former (Guyton, 1971).

The sensory nerves (Group IA and II afferent fibers) innervating the receptor endings enter the cord at the posterior roots. Motor nerves, called gamma (γ) motoneurons (Gamma I and Gamma II efferent fibers) innervate the intrafusal muscle fibers and leave the cord at the anterior roots. As mentioned previously, alpha efferent fibers also leave the cord at the anterior roots. In addition to their different functions, alpha fibers are larger and conduct more quickly than gamma fibers.

Muscle spindle function is not completely understood nor is a complete account of what is currently known possible here, yet some of the spindle's mechanism may be outlined (Frohlich, 1972). When a muscle is stretched, the spindle mediates reflex contraction as follows: the spindle is attached in parallel to extrafusal fibers, and the intrafusal fibers and receptor regions of the spindle stretch during muscle lengthening. Both the annulospiral and flower spray endings respond reflexly to stretch. The fibers of these endings relay impulses via the posterior root onto the anterior alpha motoneurons. The latter, in turn, send impulses to the extrafusal fibers to contract. This leads to a slackening of tension on the spindle receptors, decreased firing of its sensory fibers, and diminished facilitation of muscle contraction via the alpha fibers.

The muscle spindle mechanism is, however, involved in more than just reflex activity and this is where the gamma system comes into play.

Influences projecting onto gamma efferents originate in the motor cortex, subcortical nuclei, and cerebellum. Gamma firing causes the intrafusal fibers to contract thereby also causing the middle receptor region of the spindle to stretch. Thus, if gamma efferents fire during muscle contraction, they increase the sensitivity of the annulospiral and flower spray endings. This reactivates spindle receptor activity and continued contraction can be sustained via the above defined loop.

Properties of the gamma system are not well understood. It serves as a muscle length adjuster and is thought to be especially involved in muscle tone, postural adjustments, and exact positioning of muscle in complex movement. It has been thought that gamma efferents fire first in intentional movement, but the current view is that gamma and alpha efferents are coactivated.

The alpha-gamma linkage provides, in effect, a gamma bias which can "set" muscle length and allow for facilitation of continuous myriad muscle positionings at the peripheral spindle reflex level. A constant bias set keeps the spindle under constant tension even as load compensation (effort) requirements change. This frees the cortex from having to send continual impulses to the periphery concerning perpetual position changes. Furthermore, spindle receptors are capable of transducing static and dynamic properties of muscle movement. Flower spray endings and some annulospiral endings fire primarily in response to change in muscle length. But annulospiral endings on the nuclear bag intrafusal fibers fire in response to both change in muscle length as well as rate of change in length. Thus, for example, at stretch reflex initiation, annulospiral receptors can send rapid impulses such that changes in muscle length will give rise to, at changing moments

of time, muscle tonal changes, as conditions present within the spindle continue to change. This is a predictive function (reminiscent of feedforward control [Matthews, 1964] in servomechanisms) where known position and velocity provide adequate information on where the muscle will be in future time.[6]

Also, whereas annulospiral endings do not travel to the somesthetic cortex, it is believed that flower spray endings do. The latter may be involved in "muscle sense" during exertion (Paillard and Brouchon, 1968). On the other hand, some authors (Mountcastle, 1974; Merton, 1964) however, conclude that muscle proprioceptors are insentient.

The muscle spindle mechanism is another example of feedback loop activity. It involves a reverberative circuit capable of re-exciting anterior alpha motoneurons to keep contraction going. Furthermore, the alpha-gamma linkage mediated by the spindle loop is akin to a servomechanism. A servomechanism is an automatic control system which utilizes the direct effect or signal of a movement to act reciprocally in a closed loop cycle to regulate guidance, timing and force of an ongoing movement. (Other servomechanism analogues to motor function are discussed in Section IX.)

---

[6]In feedforward control, a fast or coarse movement is calculated centrally and run off in "open loop" fashion where there is no regulation of movement during its execution. Adjustment, if any, follows execution. In feedback control, there is continual regulation in "closed-loop" fashion where execution of movement is continually regulated by information on how well movement is being performed.

SECTION III

VOLUNTARY MOVEMENT

Defining Voluntary Movement

In the current research it is only voluntary movements of motor behavior which are of concern. Thus before systematically reviewing several approaches to feedback control in movement, some description of voluntary movement is in order. Neurophysiological and behavioral references to voluntary movement are abundant in feedback and biofeedback literature. Although the term is widely used, there is and can be no strict definition of what is meant by it. Definition is made all the more difficult because reflexes, programmed movement patterns and feedback are all involved in the control and regulation of voluntary movement. At the same time voluntary movement is the most important and numerous of motor movements, particularly for man and vertebrate animals. Thus, for purposes of the present exposition some criteria and views of voluntary movement will be discussed.

Movements can be viewed as continuous gradations of behavior hierarchically organized from reflexive to increasingly complex behaviors. One can distinguish involuntary or reflex behavior, at one end of a continuum, as that movement which is a direct consequence of a stimulus applied to a receptor organ. Although even here the reflex can be heightened or dampened, depending on the total psycho-physiological context in which it occurs. Neurophysiologically, sensory input arriving at the cord may produce a reflex but it is by no means exhausted or confined to a distinct portion of the cord. Sensory input spreads widely. Psychologically, there can be a simple reaction to a stimulus which

elicits the reflex response, or an aggrandizement of stimulus-response, or a diminution of a reflex response, all depending on the behavioral context in which the reflex activity occurs.

As Efron (1966) points out

> The reflex is only definable by reference to the concept of voluntary initiated action. [p. 498] [Also] the definition of "reflex" action contains . . . by implication, reference to a class or classes of action which are non-reflexive. This is necessarily so, since the purpose of a definition is to isolate the essential attributes which differentiate one phenomenon from another . . . Behavior which is automatic, innate, involuntary, and independent of consciousness needs to be isolated conceptually (i.e., defined) only because other behavior exists which is voluntary, learned, and dependent on conscious activity. [p. 491]

Consciousness however does not necessarily mean attentive or awareness activity to any specific, predetermined, interorganismically identical sensation. Voluntary movement is conscious action but consciousness is at a level of consequences to be achieved. If the consequences are to learn muscle control, then consciousness may be directed to some muscular affect of voluntary movement. And, if motor control is to be learned, the affect must ultimately be internal.

Conscious or voluntary behavior is the obverse of reflex behavior. The latter is activity which may be elicited from anesthetized, decerebrated, or spinalectomized organisms and whose characteristics such as latency, amplitude, duration and topography can be precisely defined. Such precision depends on a) ideal experimental conditions, and b) the simple direct chain of impulses at the segmental or transcortical (see footnote 7) level causing reflexes to follow stimulation with a probability approaching 1.0. There can be no operational definition of a "stimulus" or a "response" independent of the entire sequence; a specification of reflex behavior in terms of stimuli and responses requires a multiplicity of definitions for each token.

Reflex behavior is relegated to that behavior occurring at the spinal cord, and partly, brain stem and higher cortical levels.[7] It is behavior which is "wired in" and elicited stereotypically within a given species. It is, in its original manifestation, unlearned and simple, although it can be conditioned.

However, in addition to differences in response characteristics of conditioned reflexes, there is a question of whether conditioned reflex behavior is, in fact, reflex behavior. For example, the evidence to date on conditioning in spinal animals is weak (Milner, 1970; Paillard, 1960). Furthermore, the fact that conditioned responses do differ from reflexive ones suggests that they do not involve identical neurophysiological pathways of reflex behavior. Oftentimes measurements of their characteristics conform more closely to similar movements made voluntarily (Efron, 1966).

Voluntary behavior can range from simple to complex movements. It may involve the integration and elaboration of many reflexes or even completely new patterns of movement. Indeed, the infinite variability of response possibilities is often more striking than the similarities between responses. Voluntary behavior cannot be elicited directly by a stimulus. Originally, it must be learnt and it must involve supraspinal neurophysiological activity. Even though exteroceptive stimuli may precede it, voluntary behavior is behavior whose input is internally generated and not a directly determined stimulus consequence. It requires a conscious mental milieu or mental psyche action even if not

---

[7]Higher cortical reflex control is inferred from manipulations where reflex movements are induced, e.g., by opposing rhythmic breathing with an unexpected air volume reduction (increased airway resistance) in an S breathing through a tube, and latencies are observed which are longer than those of spinal cord muscular stretch reflex action yet shorter than automatic voluntary breathing action (e.g., Sears, 1974).

to explicitly direct the motor response. Habit or an operantly conditioned response may be correlated with an external stimulus but the same may be said of reflex behavior. Voluntary motor action as habit occurs in the context of a consciously functioning (awake, alert) organism, a state which can only be described as such phenomenologically or by physiological manipulations, but not descriptions, of it. It is difficult to determine where consciousness begins and what structures or neurophysiological activities it involves. Furthermore, consciousness is process at either physiological or psychological levels of description.[8]

As one criterion for voluntary movements from physiological psychology, Vanderwolf (1971) points to the fact that voluntary movements are not stimulus dependent. They "can be easily brought under the control of any one of a number of different motive states . . . and can be connected with different drives on different occasions" (pp. 102, 104). Motive states and external stimulations must interact at some supraspinal level to result in voluntary movement.

---

[8]It might again be added that physiological explanations of behavior are viewed here as correlates or underpinnings but not adductions of behavior at the psychological level. Psychology is, strictly speaking, the study of behavior processes as different from physiological brain processes. However, for one, e.g., neuropsychological study of the brain is a highly fruitful endeavor for psychology. It exposes a tremendous profusion of behaviors and makes them available for consideration and investigation. It also affords a working field of reciprocity for brain/behavior knowledge which may be accumulated for purposes of using behavioral activity to delineate brain lesion focus and extent (Meier, 1974), or, alternately, for assessing the effects of particular brain lesions on particular behaviors (Baker & Meier, 1970) and so on.

Also, to foster knowledge in either fields of physiology (or neurology) and psychology, as knowledge in any of the fields advances, psychology, e.g., must increasingly take into account organismic biology as physiology, e.g., must increasingly take into account organismic behavior.

Some attributes this author would ascribe to voluntary movements are as follows: voluntary, gubernatorial movements are relatively complex, variable, producible upon internal or external command, and dependent on the unique individual and his history of experience. Voluntary movement is a level of behavioral description superordinate to isolated elements of interoceptive and exteroceptive stimuli. The voluntary is produced to effect a change and is, furthermore, inhibitable (see p. 29).

Some Views of Voluntary Movement

One view of voluntary behavior, although by no means a definition, follows from operant behaviorism. Skinner, behaviorism's most notable proponent, says that voluntary behavior is "studied as an event appearing spontaneously with a given frequency" (Skinner, 1938, p. 21). Voluntary behavior, basically the equivalent of operant behavior, is treated as a biological given where the eliciting stimuli are presumed unknown. The issue of awareness or automaticity does not enter into the explication. Voluntary behavior is behavior emitted by an organism and conditioned by contingencies of reinforcement. It is operative on the environment in reaction to stimuli appropriate to an organism's operant conditioning history, and, finally, it involves, in the main, striate muscle movement. Reflex behavior is dichotomized as behavior which is elicited and classically conditionable. It is conventionally classified as behavior which involves primarily smooth muscle activity. There is an exception to this classification, for one, in the skeletomuscular reflex reaction to pain. There is another in the skeletal efferent activity resultant from arousal to stress. (The coordinated activity of the autonomic and skeletal systems are briefly outlined in Section VI.)

Segal (1972), pursuing the issue of operant or voluntary behavior further contends that there are some "minimal units" (Skinner's term 1969, pp. 175-176) of behavior of both phylogenetic and ontogenetic origin and that voluntary or "operant response classes appear to derive from topographies that are induced by reinforcement, by deprivation, by eliciting stimuli, by releasing stimuli, by emotional induction and probably by still other procedures not yet identified" (p. 22). Once induced the distinction between reflex and voluntary behaviors is a logical one based on category of conditioning into which the behavior falls. However, psychological phenomena do not conform to strictly formal statements of validity. Beyond this, the aggregate framework of behaviorism as a system of explanation or description of the behavior of animate matter has been shown wanting when subjected to a pure, completely logical analysis (Lambert, 1971).

An operant analysis cannot account for motor behavior as presented in this paper. Man does react and does learn to react; however, the psychological question of interest to this author is <u>how</u> does man react and <u>how</u> does he learn to react (questions of process). Further, comparisons are not completely appropriate since in the behaviorist domain operant and reflex behaviors are responses rather than movements. Indeed they are not meant to be conceptualized as anything other than responses[9] while here we are concerned with movement per se. In an operant analysis, salivation, which is parasympathetically innervated, and limb flexion, which is motorically innervated, may both fall

---

[9]The term "response" also ignores the distinction between conscious and unconscious behavior and abandons or renounces its significance.

into the category of reflex behavior. Here the problem is the genesis of voluntary behavior, and how it is self-produced--one outside the domain of category of conditioning, indeed, independent of the context of conditioning. The question here is how one is to differentiate movements by at least some descriptions of their essential differences.

A further problem arises from the blurring and alternate conceptualizations of operant--reflex distinctions (Vanderwolf, 1971). For example, autonomic responses can be both operantly and classically conditioned (Miller, 1969); conditioned reflex behaviors may become voluntary or be viewed as instrumental behaviors (Reynolds, 1968; Hebb, 1956); the "minimal units" out of which responses are shaped may, in fact, not be unlimitedly raw malleable behaviors strictly shaped by contingencies of reinforcement (Moore, 1973; Herrnstein, 1972); most importantly, a taxonomy of responses based on procedure can lead to a virtual identity of procedure with behavior study and thereby obscure actual behaviors. Inordinate concern for method or procedures of "conditioning" can lead to errors on the side of insufficient attention to the richness of an organism's repertoire of behavior. Also, it might be mentioned that if one is interested in prediction and control of motor output, a perspective of the vast stochastic base of voluntary movement shows it to be so complicated and enormous that organismic output described in terms of probability is close enough to infinity to amount to randomness, volition, intentional movement--however one may wish to label it. Thus each instance of it is, in effect, unique, and has no predictable frequency.

Kimble and Perlmuter (1970) have reviewed some classical theories of volition. They give ample treatment to James' theory of volition

which will be summarized here as the most influential of classical examples. According to James (1890), all voluntary behavior is learned. James held that stimulation of a receptor led to effector discharge and that kinesthetic stimuli were necessarily concomittant with motor excitation. The voluntary aspect of an action was caused by excitation of kinesthetic cells which produced an image of the consequence of an act. Thus, James considered kinesthesis to be critical for voluntary movement. Kimble and Perlmuter concur with James that there is an "image" of an end to be achieved, the physiological equivalent to central feedback loops (to be discussed further on). They cite, for one, deafferentation studies (also to be discussed further on) as supporting this point of view. Hefferline and Perrera's experiment with the stimulus fading technique should also be noted. Using human subjects, Hefferline and Perrera (1963) found that fading of an exteroceptive stimulus eventually resulted in that stimulus being internally driven (for details see pp. 72-73).

Kimbel and Perlmuter also stress the role of motivation and attention in voluntary movement. Their level of analysis applies specifically to the process of voluntary movements as they are being learned. Acts which have become automatized, e.g., well-developed skills and conditioned responses, are analyzed as acts for which motivation and attention have dropped out. All such automatic well-learned behaviors, besides unconditioned reflexes, are grouped as involuntary acts, and only those which are motivated and attended to are called voluntary ones. Although motivation and attention may not be specific to certain levels of ongoing activities which are being produced "automatically", motivation and attention cannot have dropped out from an acting, conscious organism. There is a difficult distinction about voluntary

movement to be made here, one which hedges the philosophical intent side of the issue and a psychophysiological mechanistic side of the issue. Sage (1971), a neuropsychologist, has the following to say about voluntary movement:

> Nearly all human movements can be voluntarily controlled. This control is a product of many parts of the nervous system functioning together. The term "voluntary control" in this respect may be misleading. Usually it refers only to a pattern of activity. Even very simple movements involve many muscles but normally we do not pay any attention to any of the component parts of a movement except the prime movers. For example, if you attempt to pick up a ball from the table, the fingers are the part of the body with which you are conscious. However, in order to get the fingers to the ball, the forearm and shoulder must be moved, and in order to do this, flexor muscles of the arm and elbow must be relaxed and the shoulder stabilizer muscles must be contracted. Furthermore, if there is movement of the total body, trunk and lower extremity muscles may be called into action. The activities are not done consciously. [p. 196]

The above is the view of the present paper, the important point being that while voluntary movement cannot be automatic and unconscious, movement of muscles themselves may be automatic and unconscious.

A point of view more befitting the view of this paper, stems from Efron (1966):

> An action is voluntary when we have consciously established its <u>purpose</u>, consciously initiated it, and consciously maintained and regulated it to insure that the purpose (the goal) is achieved. The setting of the purpose or goal of the movement is a mental action--an operation of the faculty of consciousness. The subsequent initiation of such movements is further mental action. [p. 492] [Author's italics]

While motivation or attention to a movement per se may be lacking as in a well-learned movement, intention at a more integrated executive level may still determine the movement without directly influencing many of its specifics. For example, the brain does not know about individual muscle activity yet it does know about movement. A movement may be performed for purposes of consequences (reinforcement), or, alternately, reinforcement may supply the motivation for a movement. For example, Woodworth (1906), cited in Kimble & Perlmuter (1970), remarked,

> If I wish to cut a stick, my intention is not that of making a certain back and forth movement of my arm, while simultaneously holding the fingers pressed tightly towards each other; my intention is to cut that stick. When I voluntarily start to walk, my intention is not that of alternately moving my legs in a certain manner; my will is directed towards reaching a certain place. I am unable to describe with any approach to accuracy what movements my arms or legs are to make; but I am able to state exactly what result I design to accomplish. [p. 369]

Thus, there is a difference between voluntary movements to a goal (means to an end) and movement in individual muscles. In the examples above kinesthesis may be submerged, at least at intermittant points of movement; such movements are called ballistic (see Section VIII). Physiologically, the muscle spindles could be "locked" at some phases by gamma efferents.

Recording from the precentral motor cortex, Evarts (1974) has shown that pyramidal tract neurons (PTN's) respond to intention to move before any muscular movement occurs. Monkeys presented with visual stimuli which "instruct" them to move one way or another, e.g., flex or extend, show different PTN discharge patterns for different "intended" movements. These discharges are related to "intended" movements as the movements are not enacted muscularly until a second, tactile stimulus is presented. Also, recordings from PTN's show that they fire in response to the particular muscle to be used rather than joint position to be achieved in movement. Evarts concludes that "the motor cortex may be thought of as a flexibly controlled center in which input-output relations can be changed" (p. 1395).

Luria (1973) has discussed the crucial importance of the frontal lobes in intentional movement. Massive lesions of these lobes can produce an apathico-akinetico-abulic syndrome.[10] Luria cites an hypothetical

---

[10]"Abulia" is a compound of "a" plus the Greek "boulē" which means "will" and is defined as "abnormal lack of ability to act or to make decisions" (Webster's Seventh New Collegiate Dictionary, 1963).

example of such syndromal behavior. If a patient who has suffered such a lesion is asked, for example, to "lift his hand" from a position of its resting on his bedclothes, he may begin slowly at first and continue with increasingly lower amplitude movements until he does not move at all. Yet he knows what he is asked to do (but only specifically that which is contained in the instructions). What he does not have is the intentional, voluntary aspect of movement ability to do it. The aforementioned is a simple motor task. If the task is increased in complexity, with identical instructions to "lift the hand" but the hand is now resting under the bed sheets, the patient does not do anything at all, for the motor program is more involved and he cannot begin to first pull his hand from under the sheet to, then, lift it. He again understands the command. What is lost is the regulatory function of intention in movement. Luria says,

> Characteristically in all these cases, by contrast with cases of deep-seated lesions of the premotor area involving the subcortical ganglia, there are no marked features of motor disinhibition or of uncontrolled superfluous strokes, and it is not individual movements but the general forms of action (for example, drawing closed geometrical shapes, or writing) which begin to exhibit pathological inertia, interfering with the performance of the required task . . .
>
> The essential feature of these cases is that patients with massive (most frequently bilateral) lesions of the frontal lobes not only lose their assigned programme, replacing it by 'basic' or 'echopraxic' action or by pathologically inert stereotypes, but they also fail to notice their mistakes. In other words, they lose not only control over their actions, but also the ability to check their results, although frequently they remember the task assigned to them perfectly well.
>
> This disturbance of the operation of comparing the result of an action with its original intention, or disintegration of the 'action acceptor' function (Anokhin) is one of the most important features of frontal lobe lesions. Special tests have shown that this defect is often restricted to the analysis of the patient's own actions. A patient with a massive frontal lesion who is unaware of his own mistakes will readily notice mistakes committed by somebody else if the test is carried out in such way that identical 'mistakes' in the performance of an action are made to appear to have been made by a 'third person'. [pp. 207, 210] [Author's quotation marks and underline]

Voluntary movement can be inhibited. It can be willed and not executed. There is an intentional aspect of voluntary behavior evidenced in the apraxia known as idiokinetic apraxia (Paillard, 1960, p. 1691). Here a patient may have suffered a lesion in a specific area of the parietal lobe[11] and can no longer control or intentionally carry out movements although these movements are still part of his repertoire and are objectively understood by him. Such patients may become very frustrated when, for example, they cannot perform gestures like salutes, pointing, and other descriptive gestures, but these same patients will perform appropriately under stressful conditions, where the movement falls into place automatically, unintended. Again there is an ideational apraxia where a patient is quite capable of performing component parts of a voluntary act but the temporal sequence of events is disturbed. For example, in lighting a cigarette, the patient may strike a cigarette to a matchbox, unaware of his error. It is, thus, as though a serial plan of action is lacking.[12]

Penfield's (1938) research on human cortical stimulation also shows evidence (from subjective report) of there being strong intention to make a response upon stimulation without the occurrence of the response itself.

Literal components of intentional movements do not have to be conscious. They may run off as automatic means to consequences of action, as, for example, in the higher order consequences (rules)

---

[11](Second convolution at the extremity of the Sylvian fissure, involving the region of the supramarginalis gyrus.)

[12]Here there seems to be involvement of rhinencephalic structures and anterior frontal regions of the cortex.

governing symbolic gestures. However, when a person is specifically attending to muscle action, "conscious effort" (Basmajian, 1963) or "sense of effort" (Merton, 1964) is likely to play an important role in movement and movement accuracy. [Such a theme was stressed in the current study (see METHOD: "Procedure Common to BF and PT").]

Merton (1964) has shown that human Ss can perform a movement without sensory cues (feedback) for it, that is, at least the sensory cues traditionally conceived of as those necessarily accompanying voluntary movement. Ss had their wrists tourniqueted in a way such that joint and skin sensations to the top joint of the thumb were blocked. Then blindfolded, Ss were able to move their thumbs with accuracy almost identical to that before anesthesia. When movements were blocked by E, Ss thought they had moved their thumbs anyway. Thus, there was no sensation from the IP joint distally, but effort to move produced the correct movement.

Conversely, another example presents itself from the current study. Patient 6, who had no finger movement prior to training, nor any experimental training for finger movement, was asked to concentrate and think of moving his fingers during physical therapy training. The patient could move his fingers but could not feel the movement. He had to rely on E's assurance (he did not watch his hand) that he had, in fact, moved his fingers. (Was such a patient tapping into "old", pre-stroke, "corollary discharge" routes?)

From his study, Merton (1964) concludes that the muscle spindle feedback mechanism is private--indigineous to movement but insentient as far as S's kinesthetic capacity is concerned. Or, it might be said that mind, or mental effort is not necessarily bound to the sensation of peripheral physical stimuli often inducted as necessary to movement.

SECTION IV

KINESTHESIS

As mentioned previously, kinesthesis is commonly assumed to be involved in voluntary movement. Kinesthesis has no one precise definition, but might be described as a psychological discriminative capacity. According to Howard (1968),

> Kinesthesis is the name for all judgements of a limb's position and movement depending on receptors in the limb itself and on the neural discharge to the muscles. The receptors in limbs which could serve this purpose include the muscle spindles, tendon receptors, and the joint receptors. These may be referred to collectively as the proprioceptive components of kinesthesis. [p. 27]

Kinesthesis implies psychological capacity for attention, judgment, and consciousness based on physiological sensations from proprioception. Merton (1964) and other authors to be discussed further, have shown that kinesthesis is not absolutely essential, (an effect which may be specific to certain movements studied) and that something more than kinesthesis, at levels higher in the motor hierarchy, is essential to voluntary movement.

Kinesthesis from all available sources including the vestibular apparatus, however, does play a fundamental role in normal motor performances. The importance of kinesthetic sources of feedback is particularly evident in studies of active and passive movement. Active movement is voluntary movement. Passive movements (provided S remains passive and does not take part,--i.e., does not involve gamma efference), is movement made by an external agent.

Lloyd and Caldwell (1965) found that the accuracy of positioning the leg (from knee joint with thigh resting on a table) was better in those Ss who actively moved their limbs than in those Ss whose limb was passively moved when degrees of movement were those common to the range of walking. The authors propose the effect of practice as a likely explanation. Paradoxically, Ss in the passive condition, however, did somewhat better at extremes of extension and flexion.

Paillard and Brouchon (1968) have conducted a series of experiments to determine the kinesthetic properties of manual reaching (motion and position) using only proprioceptive channels of information--(vision, audition, and touch are absent unless explicitly manipulated). Their most important finding is that self-induced or voluntary displacement of the hand is far superior to passive displacement in calibrating position. The authors view muscle spindle properties as most likely responsible for their results. Muscle spindles are the servants of higher order directionality.

In one series of experiments (Paillard and Brouchon, 1968), Ss moved a slide along a track with one index finger under conditions of active or passive movement. They then had to move, with the same finger, a slide on a parallel track to the same position by themselves. Ss were blindfolded with head and trunk fixed. Active movement was far superior to passive movement for calibration of position. (Accuracy of position sense during active or passive maintenance of the final position of the target finger was, however, poor in both conditions.)

In further experiments, Paillard and Brouchon (1974) used the same procedure and manipulated vision (of the target hand only), or tactile cues (electric shock to the tip of the target finger) under conditions

of active and passive movement or active and passive maintenance of the target finger. During both active and passive movement, only the tactile cues were important aids. During maintenance (stabilization) of the target finger, visual cues were important aids. Slowing the positioning movement led to deteriorated performance, while speeding it up improved performance. It is noteworthy that the tactile cues were important during movement; tactile cues are important to motion production. The visual cues were important during target finger maintenance; the eyes are important monitors of limb position. Cutaneous cues provide feeling and feeling is important to motion. The eyes, on the other hand, can check or control what is intended.

Another experimental procedure used by the authors (1968) involved Ss holding a concentric ring plaque with one index finger stabilized at target (under the center of the ring). This finger was then actively or passively displaced. The index finger of the other hand was connected to a stylus which could mark the plaque, and which was then moved by S to determine target position. Results confirmed those obtained in the above experiments that self-induced movement is far superior to passive movement for accuracy of positioning. Position maintenance performance was again poor under both active and passive conditions of movement.

The authors again used the above procedure (1974) with the target fixed to the finger so that only the wrist was allowed to move. The target forearm muscles and tendons acting on the target hand's wrist were cooled or vibrated, respectively. These manipulations again caused deterioration of performance.

From these combined results, the authors conclude that there must be a velocity signal contributing to accurate movement positioning.

Cooling and vibration manipulations are seen to affect this signal. Since joint receptors are seemingly activated in the same way under conditions of active and passive movements, the authors tentatively ascribe the velocity signal to spindle afferent fibers[13] and the gamma dynamic system. The gamma dynamic system is influenced by supraspinal and cortical levels of control.

Paillard and Brouchon (1968) also suggest the possibility of motor outflow, internally derived, (i.e., corollary discharge, see pp. 41-42) to explain the difference between voluntary, self-induced movements and passive movements. Yet they note that proprioception would have to be significant in the first weeks of life to build up the requisite neural substrate of information or to meet changing conditions during movement over and above organized internal suprasystems.

---

[13]Tendon receptors are also considered but not hypothesized as responsible, due to evidence that increased Golgi tendon organ activity by muscle loading does not seem to lend important cues to position sense.

SECTION V

RECEPTOR MODALITIES OTHER THAN KINESTHESIS IMPORTANT TO FEEDBACK AND VOLUNTARY MOVEMENT

As already noted, besides proprioception, other peripheral sensory controls are important in motor function. When kinesthetic feedback is lacking at the periphery due to the unusualness of the movement (as in biofeedback of single motor unit control), where it has been organically interrupted (as in stroke), or where it is not sufficient for movement accuracy (as in sewing, musical instrumentation, etc.), vision, audition and cutaneous stimuli all provide supplemental or even substitution of kinesthesis in movement. Thus, in moment to moment organismic functioning, proprioception is reinforced by all other sensory modalities resulting in fine movement patterning. Different modalities will be more or less efficient for appropriate motor control depending on the task and circumstances.

Vision

Vision, for example, is generally operative when movements are being learned, the final stages of learning being heavily dependent on proprioception, except where vision is still required for fine movement. Vision can also preclude proprioception as the important reafferent modality. Klein and Posner (1974), for example, found that in reproducing a complex (tracking) movement, after training with both kinesthetic and visual cues available, as opposed to training with visual cues alone, Ss did better after just visual cue presentation. The kinesthetic cues seemed to be somewhat of a hindrance and are perhaps not usually "consciously" monitored in such a pattern of movement.

Audition and Touch

Audition and touch also heavily reinforce proprioception in movement control. For fine movements, either of these can function as an executive modality over proprioception since they are, along with vision, the most attentive modalities of environmental input. For example, in playing a musical instrument, the superlative connection between stimulus and response is one between audition and motor efference. Furthermore, as will be seen, human biofeedback research thus far shows auditory cues to be the most suitable and salient cues for extremely fine movement control.[14] In the absence of auditory or visual channels of action feedback, as in setting the back of one's hair in rollers, or getting through an unfamiliar environment in complete darkness, tactual cues are adequate for guiding movement.

---

[14]There is a reflex connection between auditory stimuli (including subliminal auditory stimuli) and the EMG response. Involuntary EMG muscle action potential increases from auditory input have been elicited in the forearm, sternocleidomastoid, and masseter muscles (Goldstein, 1972).

SECTION VI

AUTONOMIC CONSIDERATIONS IN VOLUNTARY MOVEMENT

Evidence for the integration of autonomic and somatic activity within the CNS has been accumulating (Gellhorn, 1967; Germana, 1969, 1974) leading to a conceptualization of these systems as coordinated rather than disjoint in voluntary movement. Gellhorn (1967), for example, favors looking at two broad systems of ergotropic and trophotropic activity (terms introduced by Hess, 1954). The trophotropic system involves increased parasympathetic activity, decreased motor activity, and cortical synchronization. The two systems are tonically innervated and reciprocally related. Impinging afferent impulses may influence shifts in balance between these states.

Gellhorn suggests that there must be integration of sympathetic sensory and motor input. He regards the sympathetic division of the hypothalamus,[15] with its influence on motor cortex activity, as a probable source of causation of voluntary movement. The circuit of ergotropic activity is from proprioceptive impulses to increased sympathetic activity in the hypothalamo-cortical system. Increased hypothalamic sympathetic activity reciprocally inhibits the parasympathetic hypothalamic division and vice versa.

Working with rats and cats, Vanderwolf, et al. (Vanderwolf, 1971; Whishaw and Vanderwolf, 1973) have been studying hippocampal,[16] rhythmi-

---

[15]"referred to frequently as the center for regulation of the autonomic system" (Mountcastle, 1974, p. 808)

[16]The hippocampus is part of the limbic system which also plays an important role in autonomic system control.

cal slow theta (4-7 Hz, usually less than 20 mv in amplitude) activity (RSA) during movement. On the basis of their observations, they suggest a correlation between RSA and the initiation and continuation of higher level control voluntary movement. Voluntary movements are running, jumping, digging, shifting posture, walking, isolated movements of the head or one limb, etc. RSA apparently continues as long as such movement occurs. Voluntary movements are contrasted with movements which the authors refer to as automatic movements, which do not correlate with RSA. Such movements include licking, chattering the teeth, chewing, immobility, defecation, face washing, etc. The latter are viewed as clusters of behaviors which might be rigidly (innately) bound to particular motive states (Vanderwolf, 1971).

Klemm (1970) has simultaneously recorded from the hippocampus and the medullary and midbrain reticular formation (MRF and MBRF),[17] along with recordings of muscle electrical activity. Even during states of apparent immobility, theta activity was recorded when there was increased muscle tone. Klemm postulates that theta activity indicates a limbic system readiness to respond whereby sensory input to the MBRF influences limbic structures, and descending impulses to the MRF influence spinal motoneurons.

The issue of the significance of RSA is still a theoretical one. When viewed systemically in terms of an adapting organism, it is clear that autonomic responses are integrated with somatic responses. Thus, voluntary motor movement is composed of both motor output and motor "readiness". Germana (1969) summarizes such a viewpoint:

---

[17]The reticular formation is a mass of cells from the brain stem to thalamus, which, when activated, causes electrophysiological arousal. It can be divided into two functional systems, the MRF, part of a descending fiber system, and the MBRF, part of an ascending system.

1. Central activities independently give rise to experience and to the specific autonomic and behavioral responses which occur at the periphery.
2. These central activities constitute the organization of autonomic and behavioral responses.
3. Central efferent organization is initially reflected at the periphery as an anticipatory, preparatory state, or posture.
4. The autonomic and somatic responses which constitute such a preparation for specific behaviors should demonstrate high intercorrelation since they have common central origins and are concerned with the same end-product (adaptive behavior). (p. 80)

SECTION VII

FEEDBACK AND VOLUNTARY MOTOR CONTROL

Feedback loops may be considered as closed systems or circuits where there is input, output, and feedback from output to the system allowing for a measure of self-correction. When the input "terminal" is inside the skin, the loop function is that of an adjustor or regulator. When boundary demarcations fall outside the skin at external input or output "terminals", closed loop, self-regulatory principles still apply and are evidenced in the homeostatic, continual adjustor processes of the functioning organism. There is, in addition to closed loop systems, feedforward control, or open loop systems (see p. 17) where responses can be made on the basis of current systemic conditions into the future without regulation during response execution. This type of control is also evident at all levels of organismic functioning and may be viewed as directed, anti-entropic control.

Extrapolations from cybernetic principles of closed and open loop systems have been made to both physiological and psychological descriptions of life processes. (Although he is not discussed in this paper, reference must be made to Weiner [e.g., 1948] as the initial and influential expositor of cybernetics [feedback].) Organismic self-produced movement utilizes feedback while it also builds upon the consequences of perpetual motion and sensation. These are attributes of voluntary life. Psychologically, the distinction between closed and open loop systems means organismic capabilities of both activity and reactivity. Organisms can incorporate information as well as project activity onto and against external influences. Furthermore, to use feedback to achieve or maintain homeostatic conditions there must be voluntarism. In other words, loop connections (i.e., connection of loops, their sustenance, constitution, or forging) implicates more than "passive" phenomena composing organismic "activity".

Having reviewed some considerations of voluntary movement, the remainder of this section will be devoted to the concept of feedback in voluntary movement. Fitts and Posner (1967) distinguish two types of feedback: intrinsic feedback which is internal to S and mostly arises from response-produced cues, and augmented feedback which is supplied extrinsically to the response from the environment. Taub and Berman (1968) identify the same types of feedback as topographic and non-topographic feedback. Evarts (1971) makes an even finer distinction of intrinsic feedback which leads to three categories of feedback, a distinction which will be followed in the present paper: 1) internal feedback from motoneuron back onto its input sources; 2) response-produced feedback from muscle onto its input sources; and 3) knowledge of results--(the term exteroceptive feedback will be used here instead)--which is any source of feedback external to the physiological organism (e.g., Granit, 1972).

Internal Feedback

The possibilities on the numbers and types of internal feedback loops--motor to sensory, motor to motor, and sensory to motor--are great. Internal loop pathways (Oscarsson, 1970) can feed back onto CNS sources without direct peripheral input and are possible candidates for "corollary discharge" circuits in voluntary movement. There is descriptive and experimental evidence for something at least akin to corollary discharge (see Merton earlier, pp. 20-21, 1964; Sears, 1974; Granit, 1972). For example, if one intends to move a paralyzed eye his judgment is that his eye has moved. The result is an illusion of movement of the environment. There are multitudinous examples of such illusions (Granit, 1972).

The term "corollary discharge" was introduced by Sperry (1950), "correlator-comparator" by Held (1961), and, initially, "efference

copy" by von Holst and Mittelstadt (1950). These concepts are related in that they share the notion that there is more than proprioceptive input guiding a response. That is, there are anticipatory adjustments at higher levels of the CNS which can guide the response in feedforward control, a control which does not depend on moment to moment peripheral sensory input. Thus, movement information of central origin can monitor outgoing impulses and feed back onto central mechanisms as impulses reach the periphery. Movement will be either matched or mismatched to the corollary discharge, and an organism will respond or readjust according to the reafferent signals of his movement.

Response-Produced Feedback

Response-produced feedback is indigenous to response in the normally functioning organism; yet it may not be absolutely essential in learning an instrumental response. For example, the proceeding Section XI offers examples of Ss who are able to control single motor unit or extremely small motor potential activity. These Ss are, at the same time, unable to discriminate response-produced cues and, sometimes, the response itself. In another vein, Merton (1964; see p. 30) showed that Ss who were intending movement under conditions of obliterated response-produced cues, reported movement even when movement was blocked.

There is no doubt, however, that peripheral sensations of a response are valuable indices, cues, and aids to performance and learning. For example, it is usually the case that patients who have suffered motor impairment (such as stroke victims) are better equipped for physiatric rehabilitation when sensory damage has been minimal. (Section IV also describes several experiments on the active and passive positioning of upper limb or limb parts which highlight the importance of response-produced feedback.)

Deafferentation and Curarization Studies Related to Internal and Response-Produced Feedback--Ethics and Conclusions

A review of some deafferentation and curarization studies is presented as part of the Internal Feedback and Response-Produced Feedback sections. The review is proceeded by some considerations this author has given the following research.

Deafferentation studies show that organisms are able to use their limbs (although with great reluctance and inferior ability) in the absence of proprioceptive feedback, feedback presumed indigenous and essential to normal movement. Many authors have come to the conclusion that internal feedback loops, the physiological correlates of the corollary discharge concept, may still be functional, along with exteroceptive sources of feedback. First it must be emphasized that no matter how functional and purposeful movements are after deafferentation, they never regain the smoothness and precision of normal movement. Furthermore, Bossom (1974) has suggested the possibility that there are sensory fibers in the ventral roots which would not be transected in dorsal rhizotomy. A review of deafferentation studies provides some interesting data which must be taken into account.

Earlier investigators reported immobilization of affected limbs after deafferentation. Lassek (1953), for example, reported that brachial rhizotomy in the monkey (left dorsal roots C_2-T_4) resulted in flaccidity of the limb with almost total loss of voluntary movement. Many reflexes were affected as well. Lassek attributed the loss to the lack of sensory impulses which were viewed as essential to movement.

Twitchell (1953) partially or completely deafferented the upper limbs of monkeys. He found virtual paralysis in completely deafferented limbs (C_3-T_3 dorsal roots cut), extension and flexion being the only remaining movements. The latter were at first purposeless but their use, although crude and clumsy, was recovered in time. Such recovery required vision of the limb. The flexion and extension movements no longer occurred after section of the three uppermost dorsal roots of the cervical region and thus were believed to be related to the tonic neck reflex.

Partial deafferentation (e.g., of one dorsal root which innervated the hand--C_6, C_7, C_8, or T_1) resulted in some discernable defect, mainly in grasping. Here the deficit was reported to be due to lack of muscle proprioceptors, this lack contributing the greater deficit. With cutaneous receptors present, there was very little deficit in grasping, but without them, the deficit was severe. Grasping was possible without muscle proprioceptive input. Twitchell's conclusion was that those movements abolished by partial deafferentation were those which rely on adequate stimuli for output.

Recently, it has been shown by several investigators that dorsal rhizotomy of upper extremities does not necessarily result in great loss of voluntary movement. Quite sophisticated and purposeful movements can be preserved if post-operative care is excellent and procedures to test for movements are adequate (Bossom, 1974). In cases where only one side is transected, having the normal limb bound facilitates efferent activity in the deafferented limb.

Taub and associates[18] have carried out a number of studies on movement and learning in deafferented monkeys. A summary of their findings appears in a report by Taub and Berman (1968). For example, Knapp, et al., (1958, 1963) trained monkeys with one deafferented forelimb and without vision to voluntarily flex the deafferented limb to a buzzer which both terminated it and avoided shock. (The contralateral unaffected limb was tied to the apparatus.) Animals trained pre-operatively were able to relearn the response to criterion post-operatively. Pre-operatively naive animals were also able to learn the response post-operatively. When a click too short to be terminated by movement was used as the CS, the monkeys again learned to avoid shock by responding. With the intact limb immobilized, food-deprived monkeys also learned to reach out of their cages for food using their deafferented limbs.

In all of the above learning situations, exteroceptive feedback was available--most obviously the auditory conditioned stimulus and visual conditioned or unconditioned stimuli. Such feedback was virtually eliminated when Taub, Ellman and Berman (1966) trained monkeys, with neither view of their limbs nor any auditory stimulus, to perform the voluntary movement of grasping a polyvinyl cylinder (taped to the hand of the deafferented limb, which was immobilized in a halter) to avoid shock.

In all of the above studies of single forelimb deafferentation, Ss were not observed to use the affected limb in any purposeful way in the free ranging situation. However, Taub, et al., (1964) deafferented both limbs of three monkeys and found the opposite to be the case for these Ss. One or two weeks after the operation, there was progress to excellent coordination and a fair rate of ambulation continued over the course of two to six months. Some monkeys could even pick up a raisin. Although vision was not controlled at post-operative outset, later blindfolding showed that Ss were capable of similar movement without vision.

Total spinal deafferentation was then carried out on three monkeys (verified histologically as complete in two and practically complete in the third), none of whom survived very long. These Ss were able to use their upper limbs in the free situation as well as those animals with bilateral forelimb deafferentation. There was, up through survival time, no recovery of lower extremity function. Blindfolding these monkeys and restraining them in isolation for 12 hours with a random white noise background resulted in intense restless movement.

Taub and Berman (1968) hypothesize that their results indicate either a) a central mechanism which has feedback of central origin

---

[18]Knapp, Taub, and Berman, 1958; Knapp, et al., 1963; Taub and Berman, 1964; Taub, Ellman, and Berman, 1966.

feeding into it before impulses of intended movement reach the periphery (corollary discharge) or b) a central mechanism which relies on no feedback whatsoever and which directly sends centrifugal impulses to the periphery. The authors favor the former explanation and conclude that "the concept of self-produced or voluntary movement must now be exhumed and examined experimentally" (p. 190).

Taub and Berman (1968) make particular note of the fact that total spinal deafferented monkeys could still respond with virtually no sensory input. Even in a case where the cranial parasympathetic system was blocked through the vagus to interrupt all but 5% of the ANS interoceptive pathways, the animal remained awake and performed accurately for a series of 10 avoidance responses. When this final 5% was blocked, somnolence did result but S could still be awakened to respond with intense auditory and facial somatic input.

Proprioception (and also touch) is important to movement but apparently not crucial. Furthermore, even with virtual elimination of exteroceptive feedback (including vision) save shock in an experimental situation (Taub and Berman, 1963, 1968), Ss can still self-produce movement. Barring a ventral root sensory pathway, it might be concluded that central efferent monitoring of self-produced movement is possible and, in fact, may be most important. Normally, proprioceptive and other ancillary exteroceptive feedback sources play an important role in voluntary movement. Yet such sources are apparently not absolutely essential.

Another technique which has been used specifically to block response-produced feedback is the administration of paralyzing agents to the peripheral neuromuscular system. These studies show that an organism can execute movement or operate on his environment in a purposeful manner without having to train a particular muscle movement in advance of making the movement. That is, learning can occur in a paralyzed state such that transfer cannot be somatically mediated.

Solomon and Turner (1962) trained dogs to avoid shock by panel pressing to S^0 (light out stimulus). They then curarized (with d-tubocurarine) the animals and carried out classical discriminative conditioning sessions pairing shock and no shock with different stimuli, S+ and S- (2 tones). After recovery from paralysis, the dogs were returned to the initial training situation and all three stimuli were presented. Panel pressing responses to S^0 and S+ were far more frequent than panel presses to S-. Responses to S+ were also far more resistant to extinction than responses to S-. The authors concluded that learning was possible in the absence of skeletal movement or response-produced feedback. They also suggested the possibility of ANS mediation of learning. (Several authors have reported autonomic classical conditioning in curarized animals with transfer of these responses to a normal state.)

Buchwald et al. (1964) used flaxedil to paralyze adult cats and carried out classical conditioning sessions using a tone as CS and shock to leg as US. Flaxedil was used at a depth of paralysis presumed to block gamma motoneuron activity. Ss did not show immediate overt

leg flexion conditioned responses (CR's) in a normal state but they did show quicker learning (first 10 trials) of the CR compared with naive cats (which required at least 3 sessions for learning to occur).

During the paralyzed state, Ss were not only unable to move but presumably also getting no proprioceptive feedback from gamma motoneurons. The authors suggest that a blockade mechanism during flaxedil training explains their results. They note that conditioned flexor responses have been elicited in a normal state from cats who were trained during bulbocapnine catatonia. Thus, in addition to lack of movement preventing learning in the paralyzed state, they also suggest that lack of the effect of gamma motoneuron spindle feedback can prevent learning. In a flaxedil state conditioned gamma motoneuron activity is postulated as available (along with other supraspinal sources of conditioning and pupillary response) but the reafference or afferent feedback mediated by these neurons is unavailable. When the normal state is restored, the added effect of afferent feedback is seen to lead to faster responding in Ss who have been exposed to pairings in the flaxedil state.

Whatever the mechanism responsible for the transfer of learning from a paralyzed to a non-paralyzed state, peripheral response-produced feedback can apparently be by-passed to effect efficient motor movement. Even an instrumental or operant response can be trained without the response having to be made. In a paralyzed state, the organism is functioning at the CNS level only and somehow puts together efferently, the meaning of components of stimuli. Curariform agent studies suggest a central systemic state supplying conditioning "conditions" during which Ss can learn signal significance without making a movement.

Preliminary Comments:[19,20]

A. On the content of the above studies:

1. The deafferentation and curarization studies cited derive from the fields of behavioral neurophysiology, psychology, etc. It is the behavioral results of the research which are germane to certain current psychological approaches or fields of inquiry.

---

[19]Any references to, or comments on, the corollary discharge concept in this section are predicated on the above and only the above studies. The author does not dispute the conceivable potency or pregnancy of the corollary discharge concept for psychological endeavor.

[20]This section does not address itself to a multiplicity of tangential issues: e.g., whether much of what is studied as above can be obtained from consenting human Ss who are organically impaired or who voluntarily consent to experimental manipulation; or, e.g., the suitability of many artificial experimental conditions which do not simulate nor resemble natural environments and conditions of existence; and so on.

B. On ethics and conclusions:

1. The ethics to be posited are the author's stance and proferred opinion.
2. Conclusions posited about behavior are also the author's thesis and responsibility.
3. The author means to confine her opinions to the discipline of psychology.

Deafferentation studies prove in an experimental way that organisms are able to use their limbs (although with great reluctance and inferior ability) in the absence of proprioceptive feedback, feedback presumed indigenous and essential to normal movement. Hence, many authors have come to the conclusion that internal feedback loops, the physiological correlates of the corollary discharge concept, may still be functional, along with extroceptive sources of feedback. Needless to say, some internal neurophysiological activity, of course, must be functional for bodily movement to occur.

Bossom (1974) has reviewed the general stages and nature of recovery in single limb deafferented animals. At first there is great dependence on vision. To extend the deafferented arm (to reach for food) the animal must have both hand and foot in view. From prior experience, it would seem that the animal cognizes or can make the connection between the drive for food and a limb for prehension of food. To grasp a piece of food, the animal opens his palm over it and then must be helped to cup his hand. Use of thumb and forefinger opposition to pick up small pieces of food must be carefully trained. When a pinching stimulus is applied, no attempt is made to remove it unless it is seen. (Interestingly, animals have little difficulty localizing

the mouth without vision, to bring food to it, relative to the initial difficulty in reaching and grasping. Perhaps this is because the mouth is an experiential part of the body schema which has not been disturbed.) If total deafferentation procedures were carried out on higher level infant organisms who had not experienced the sensory through and concomitant with motor exploration, or vice versa, i.e., the motor with sensory reafferentation, the above results would likely be impossible.

Vision is an extremely important factor in re-establishing movement. Vision guides the organism to realize that there is an appendage once used to purpose, which, with organismically intended actions, can again effect afferent activity.

Deafferentation and curarization studies invade the living organism with increasingly radical physiological alterations of the sensory system and sometimes painful stimuli to test the tenets of conjectured mechanistic models of organismic activity. The result of such procedures is an infinite regress to some level of "unexplained" voluntary activity eluding peripheral physical stimuli.[21] The effect is replacement of the latter with new inferred higher order internal reafference pathways and the corollary discharge concept.

The procedural extremes of deafferentation and curarization studies and the resultant paucity of concrete neurophysiological knowledge about motor control and physical properties of organismic conditioned response is perhaps best exploited here for its implications of voluntary movement to psychology and ethics. To this avail, such evidence is used here to assist the higher psychological and ethical issues at hand.

---

[21]In operant S-R theory, these stimuli are sometimes surmised hypothetical inferences for questions which invariably give rise to such formulations (for further reference see, e.g., Kelleher, 1966).

A rigorous definition and interpretation of the objective of deafferentation and curarization studies as applied to psychological science interposes a conclusion lacking force, and secondarily, an heuristically weak conclusion. A greater number of physical elements can increasingly be eliminated--both nerve pathways from the peripheral to the central nervous system and exteroceptive cues can be blocked or cut--and, unless the final result is comatose or stereotyped behavior, voluntary movement is still obtainable. In some way, an alive, i.e., non-demolished, even minimally sentient, animal survives; it moreover wills or does not so will to survive. That is to say, there is something essential in being (the living) which is not manifest in non-being (the non-living).

The fixed yet circuitous and divergent course of much investigation, formulation, estimation and methodizing, as well as an indefinite convergence of influences, collides in the knowledge such as that resultant from deafferentation and curarization studies--one of them the formidable maintenance of implicit positivistic attitudes which are not, in general, subject to enduring scrutiny. Many premises, in fact, would not be feasible, nor brought to experimental analysis, if there were a concurrent inquiry pertaining to the validity of their status, an inquiry fostered through reason and evidence.

In addendum, an organism survives, avoids pain, eats to maintain himself, fights for his being, wills to exist, (i.e., to survive); and such phenomena or manifestations are all the more marked by emphasis, and the conclusions all the less salient, considering the damaged or impaired physical condition of many of the organisms subjected to this type of experimentation.

Psychologically, within the limits of some corporeal substrate other than reflex neural activity, plus a modicum of environmental sustenance, organisms are capable of meeting new conditions with adaptive efferent activity. Deafferentation and curarization studies reinforce the role of living brain and "higher" organisms as processors. The means to this end acknowledgement is of greater priority than the end result. Life is a process and as such it is the vitalism (holistic function), rather than the fragmentation (atomistic function) of organisms which is decisive. It seems that as long as there is viable amount and kind of living neurological tissue, there will be plastic, adaptive, proactive organisms.

Exteroceptive Feedback

Movement coordination, then, is dependent on reafferent central stimulation and reafferent peripheral stimulation.[22] Finally, all movement is to be observed within the context of an environment full of external consequences which provide S with a third kind of feedback, exteroceptive feedback of events. Exteroceptive feedback, often called knowledge of results, has been widely used in experiments on motor learning. Exteroceptive feedback manipulations are various: clicks, reports of error, visual displays, etc. Actually, outside of the laboratory exteroceptive feedback such as praise, gifts, corrections, etc. often accompanies many types of motor skill training. Experimentally, the conclusion for exteroceptive feedback and motor skill is unequivocal. Feedback greatly enhances performance, and, depending on the task, learning. The following points to be made about exteroceptive feedback are broad generalizations obtained across many kinds of motor tasks, experimental designs, and types of exteroceptive feedback.

---

[22]The vestibular apparatus relies on teleostimulation (gravity).

In a review of informational feedback, I. Bilodeau (1966) points out that many terms have been used to describe exteroceptive feedback. The common ones are knowledge of results, feedback, reinforcement, and information feedback. However termed, (the present paper will use the term exteroceptive feedback), it plays a definite role in improving performance, sustaining performance, and eliminating undesirable responses.

Bilodeau also points out that three properties have been ascribed to feedback by various authors, properties which are not easily distinguishable. They are a) information (stimulus property), b) motivation (drive property), and c) reinforcement (reinforcer property).

Exteroceptive feedback is motivational in that a great percentage of feedback or constant feedback is superior to intermittent or variable feedback. Yet for humans, even intermittency of feedback is not too important provided that no other motor tasks intervene. (Neither is temporal delay of feedback very important for humans provided no other motor tasks intervene.) The motivational property of feedback is also evident in a situation where two groups are well trained on a task, one group receiving feedback, the other not receiving any. The former group will perform better. (This might also be viewed as a qualitative reinforcement function of exteroceptive feedback.)

Within an operant paradigm, exteroceptive feedback can serve as both a stimulus and a reinforcer. One can shape a desired response with appropriate applications of feedback. In established responding, motor behavior sustained by constant feedback will decrease rapidly in proficiency when feedback is withheld. On the other hand, motor behavior supplied with intermittent feedback may or may not result in more stable

responding once feedback is withdrawn. For particularly fine, discriminate motor tasks, there is apt to be a greater and longer dependency on feedback. Withdrawal of once important feedback with no decrement in performance would suggest a pre-emption of exteroceptive feedback by other feedback sources--e.g., Kimble & Perlmutter's "image" of an end to be achieved, or other extrinsic or intrinsic (inner mediated) feedback loops.

The fact is that once many motor skills are learned they no longer depend on any observable parameters of control or even on conscious control (e.g., walking). Also, whether skills will be maintained or retained depends on the exigencies of learning, the utility of the response. Vital or uniquely relevant skills are self-generated.

Bruner (1970) points out that intentionality precedes skill. Observing the behavior of infants one can see that there is a preparedness to interact, touch, manipulate, put to the mouth, etc., objects in the environment. The way the skill develops is not temporally confined to parameters of stimulus, then response. Rather, there is an innate movement reference which is the substance out of which skills develop, proceeding with progressive spatio-temporal differentiation of gross movements as behavior develops (correct serial timing coordination within movement run-offs at efficient moments). Bruner stresses that the initial reflexes infants are born with are not "chained together"[23] as the material for later skills, (although, of course, they are inextricably woven into the human repertoire). Rather, movements which can segment and develop into component parts come from athetoid-like[24]

[23]As Bernstein (1969) notes, "a reflex is not an element of action, but rather an elementary action," (p. 443). (author's italics)

[24]From athetosis, "a condition in which there is a constant succession of slow, writhing, involuntary movements of flexion, extension, pronation, and supination of the fingers and hands and sometimes of the toes and feet" (Stedman's Medical Dictionary (22nd ed.). Baltimore: The Williams and Wilkins Co., 1972.)

diffuse activity of the infant, shaped by experience with the environment and initially brought into service by intention to do, act, touch, see, reach, etc.

Several authors distinguish an informational property of feedback. O'Brien and Azrin (1970), for example, distinguish between reinforcement and informational properties of a stimulus, both of which may operate to strengthen a response. They advocate the latter as having greater practical advantage in behavior modification. If an individual is motivated, he can usually utilize some kind of information to program his responses.

O'Brien and Azrin (1970) treated slouching problems using information feedback. (Ss were chosen as available rather than in accordance with such a problem.) The feedback was a vibro-tactile stimulator which Ss wore attached to their shoulders under their clothes. Slouching points were uniquely determined for each individual, the stimulator being energized when such a point was reached. A miniature elapsed-time meter, which was part of S's harness but unknown to S, recorded slouching durations. Ss wore the harness for four hours. The first and last hours were control periods, the second and third, feedback periods. Slouching was found to be significantly reduced during hours two and three vs. hours one and four.

A follow up study sought to determine whether the vibro-tactile stimulus might have been aversive. Ss were instructed either to slouch (and thereby presumably motivated to do as told, say the authors), or Ss were not instructed at all. For all Ss the stimulator was activated on one side of the back when slouching occurred and on the other side when there was no slouching. Thus the quality and intensity of stimulation were constant. It was found that Ss instructed (motivated) to

slouch did so while non-instructed Ss decreased slouching. A decrease in slouching was not due to stimulus aversiveness since the same stimulus could increase and decrease slouching.

In conclusion, the authors suggest that information feedback procedures can be used fruitfully on patients who are motivated. They also caution the futility of such procedures with non-motivated individuals. This caution, of course, follows from the fact that patients who do not wish to change their behavior, or who cannot be induced to do so, will not process information. It is probably also true, however, that in environmental settings which offer ample opportunity for patient option, effective procedures may arouse hope or desire and thereby entice patient response. In environmental settings which offer standard, homogeneous, limited contingencies, rewards--which are based on contingencies--will also necessarily be limited (i.e., rewards will be primary [fundamental in the hierarchy of existential needs], or of minimal, circumscribed intrinsic merit, and consequently, unlikely incorporative of serious, esteemed personal value). "Rewards" in such settings may compel (and conceivably repulse) patient response.

SECTION VIII

SKILLED AND BALLISTIC MOVEMENTS

A departure from strict kinesthetic reflex or locomotor control may provide a key to what is meant by skilled movements. Paillard (1960) defines skilled movements as

> a particular category of finely coordinated voluntary movements, generally engaging certain privileged parts of the musculature in the performance of various technical acts which have as common characteristics the delicacy of their adjustment, the economy of their execution and the accuracy of their achievement. (p. 1679)

Again Bruner (1970) writes:

> Skilled activity is a programme specifying an objective or terminal state to be achieved, and requiring the serial ordering of a set of constituent, modular subroutines. Functionally equivalent variations in serial order and substitution rules for constituent subroutines both are features of skilled activity and render skill productive in the sense that language is productive. Variations in serial order assure flexibility or productivity by making possible appropriate changes in the order with which constituent subroutines are used. The more a skill is linked in real time with such physical requirements as gravitation, constraining velocities, etc., the fewer the functionally equivalent variations in order that will be possible . . . (p. 65).

Some skilled movements will always depend on attention, feedback, and practice (e.g., piano playing) while others may become automatized (e.g., bicycling). Much has been written about skilled movement but there is a paucity of scientific data on how skills develop. Even a viable method of approaching the issue is wanting.

The common assumption that kinesthesis must be involved in voluntary movement is not necessarily the case. This does not mean that kinesthesis is not involved in normal movement. However, even during normal movement, the extent to which peripheral, response-produced feedback subserves a given response, once an intentional movement has

begun, can vary. Bouisset and Lestienne (1974), for example, found that the sole determiner of the kinematics of forearm flexion or extension (index finger movement) aimed at a fixed target was instructions to S. In one experiment, S was instructed to move at slower or faster speeds and it was the initial speed of the movement which seemed to account for the kinematics of movement. Since the movement always corresponded to instructions given to S regardless of inertia conditions, the authors emphasize the adaptivity, rather than stimulus bound properties, of the neuromuscular system.

Higgins and Angel (1970) studied performance of pursuit tracking (joy stick movement to left or right to match a target line on an oscilloscope), comparing error correction time (ECT) with proprioceptive reaction time (PRT). Visual feedback of tracking movement was available in the form of a cursor movement on an oscilloscope corresponding to ongoing tracking movements. In the first part of the experiment, S was to align the cursor directly over a target and change his errors as quickly as possible. In the second part of the experiment, S was to hold the joy stick against any counter force (which was 5 lbs. of force suddenly pulling to the right or left). Mean ECT was shorter than mean PRT. The authors concluded that a central mechanism rather than sensory feedback was responsible for ECT. A central movement mechanism does seem a likely explanation for the results. However, the logic of the conclusion must equally emphasize the fact that a) vision, although a slow system, was more critical in directing error correction than proprioception and b) that the proprioceptive response to force probably involved transcortical loop relay time rather than just peripheral receptor reflex time, as S had to first realize the pull and then react.

The above sorts of movements which require less than continual proprioceptive feedback, are often referred to as pre-programmed or ballistic movements. "The ballistic principle means that the elementary units of action, if one may so speak legitimately, are essentially timed and phased sequences of muscular contractions and relaxations which are initiated as wholes" (Wellford, 1974, p. 382)--e.g., the music phrase, tennis stroke, karate punch, etc. Study of such movements has shown that peripheral feedback may be important at initial and/or terminal phases of movement, or intermittently. Units are stored centrally and modified a) only as necessary to match external events or b) to give greater coherence to units by increasing the usage of storage capacity.

There must be some programmed storage, since, if this were not the case, motor movement would be a matter of discrete, intermittent, oscillatory performances, where a stimulus would appear, a time interval elapse, and a response be made. More importantly, the nervous system is, in effect, prepared for eventualities in anticipation of consequences--or as Bernstein (1969) refers to it, a capacity for "running into the future" (p. 446). Such is the case when the experienced baseball player hits a ball accurately, well in advance of seeing where the ball goes (Kay, 1970). (Kinesthetic and visual cues at certain points are likely very salient cues here.)

Bernstein, a Russian cybernetician, criticizes viewing an organism as merely reacting to his environment (Russian reflexology) and takes, instead, a view in line with current physiological knowledge, of the organism acting upon his environment. He has the following to say about motor movement:

> If we analyze the basis for the formation of motor acts, we find that each significant act, whether reactive or spontaneous, is a solution (or an attempt at a solution) of a definite problem of action. But the problem may be considered to be the result which the organism is attempting to achieve, in the sense of something which should be, but still is not, the case. Thus, the problem of action is some sort of model of the required future, encoded or otherwise represented in the nervous system (Bernstein, 1969, pp. 444-445). (Author's italics)

Again, here we see the necessity of some notion akin to corollary discharge. Furthermore there is an irreconcilability between an elementary S-R view of an organism and the actual psychological-physiological, adapting, and to a great extent, internally monitored organism (see pp. 41-42). In sum, in skilled movement, there is fluidity and continuity and one can react to predicted outcomes with increased sequencing of action (larger units of behavior).

SECTION IX

SERVOMECHANISTIC ANALOGUES

Self-adjustment of human motor response implies error detection capabilities through feedback to achieve correct motor output. Paillard (1960) writes:

> The formerly classic concept of a motor projection containing all the detailed elements of the patterning of command, like the perforated music rolls of a mechanical piano, now appears clearly untenable. It would necessitate on the part of the originating structures and mnemonic functions a gigantic task very difficult to conceive in terms of nervous mechanisms. The most perfect pre-existing plan could not account for the astonishing capacity of the nervous system to adjust our movements to the ever-changing and most unforeseen circumstances of their achievement. (p. 1698)

Paillard, quoting from Weiss (1941), defines two types of adaptive plasticity of the motor system:

> 1) elasticity within a given qualitative performance admitting of quantitative adaptation to what, for the given species, is a normal range of variability of the environment. [Paillard calls this the] "flexibility of the usual or inborn forms of action".
> 2) the ability of an organism to cope with emergency situations lying beyond the normal range of elasticity, by creating new performances previously not even formerly latently in existence--[Paillard calls this the] "adaptive learning of new forms of action". (p. 1698)

Such characteristics of motor behavior have led bioengineers, neurophysiologists, and experimental psychologists to analyze motor performance as analogous to servomechanistic feedback control systems (see p. 17), (Parsegian, 1973). The latter are machines capable of continually monitoring final aim in phase advance (timing in advance of some consequence). Feedback circuits into output command can modify the input command of the system. Error can be detected within a certain range rendering the system flexible within those limits. Again, apropos of servomechanistic models, Paillard (1960) writes:

> A principle of circular regulation seems to govern the fundamental mode of relation which ties the efferent to the afferent portions of the nervous system either inside the body or through the external medium. Thanks to its self-regulating mechanism, each working unit of this system assumes, in a given range of flexibility, its own functional balance in accordance with the requirements of the general equilibrium which results in this internal unit contributing to the effectiveness of the organism as a whole. (p. 1702)

Although there are many physiological and psychological properties which cannot be accounted for by servomechanistic models they do afford some approximation to the feedback loops involved in motor behavior. Apropos the inadequacies of servomechanistic models, e.g., in higher organisms, sensory input is very difficult to quantify in its full complexity or describe with precision in terms of its attributes (there are operational problems, subjective differences, etc.); also there may be, over a given time span, observable input without observable output and vice versa. In any event, the motor, output side of the model may be more concrete, observable, and measurable.

SECTION X

CYBERNETIC APPROACHES TO LEARNING AND PERFORMANCE

Closed loop systems analyses of learning are currently receiving a great deal of attention. For example, Adams (1971), looking at motor behavior in terms of error processing, has put forth a closed-loop view of behavior where central emphasis is given to the role of response-produced feedback and practice. Error processing, aided by both feedback and practice, (along with a learned "standard reference mechanism" against which an S can judge subsequent movements and express their identity with or deviation from the standard), are viewed as fundamental to learning. Sensory modes more or less add up as channels for data processing, uninformative stimuli being considered contextual or unregistered.

Following the "closed-loop" theory of behavior, Adams and Goetz (1973) designed an experiment to assess the role of feedback and practice on error detection and correction for learned movement. The movement was a self-paced displacement of the arm which was to move a slide along a track a certain number of inches. In learning trials, S had to move the slide to where it stopped (a point fixed by E). On test trials S had to choose which of two successive lengths (the criterion or an error length) was the practiced movement, (called error detection trials). A second part of the experiment was identical except that in test trials S had to move the slide to a fixed error point and then, adjusting for correction, move it to the practiced length (called error correction trials).

Feedback was maximal or minimal in three modes: 1) visual, where S's sight of his hand movements only was illuminated vs. no movement visible at all; 2) auditory, where S could hear his response-produced sounds vs. white noise masking any sounds; 3) proprioceptive, where a spring tension on the slide provided heightened feedback vs. no spring tension. Practice was either high or low: ten trials vs. two trials over a series of eight criterion movements.

On test trials, as the amount of error increased, all Ss showed an increase in ability to discriminate the criterion from the error length. It was also found that maximal feedback was important. Furthermore practice was to little avail when feedback was minimal. However, when feedback was maximal, high practice groups performed significantly better than low practice groups for error detection and approached significance for error correction.

Adams and Goetz say that the correct movement was learned without errors during error detection before any error correction was possible, i.e., when literal trial and error kinesthetic, etc. stimulation was not possible. When error correction was demanded the authors interpret an established standard reference mechanism of movement as responsible for movement accuracy.

Adams and Goetz's study again points to the significant effect of feedback in motor performance as well as enhanced effects of feedback with practice. Adam's closed loop theory of motor behavior is, however, too simple. Any elaborate theory of what is involved in skilled motor behavior is, at this point, premature and the most one can do is work within a theoretical framework (e.g., of closed loop systems), none of which will be completely satisfactory. The simplicity of the above theory might be criticized on grounds of supposing an additive

function of stimulus input sources where information stimuli "count" and uninformative stimuli are negligible. Also, motivation and reinforcement are not taken into account. Finally, Adams means to say that there is a standard reference mechanism which S can consciously tap into to detect error, while such a correct movement standard is only figurative and may not at all be "accessible" to S.

Karl Smith, a cybernetician, also takes the view that motor performance and learning is closed loop and incorporates neurophysiological evidence into his psychological model of motor behavior (Smith, 1966). Smith describes a fundamental difference between closed and open loop conceptualization as follows: Closed loop systems

> 1) are . . . self-governed; 2) they operate in terms of reciprocal closed-loop circuits of activity composed of dynamic response and sensing processes; 3) they involve continuous control of such closed-loop circuits of activity; 4) they are directly guided in both sensing and response in terms of feedback control; 5) different component activities of the closed-loop system are integrated through feedback processes; 6) the feedback control of such response systems can be transformed by instruments and communication (symbolic) systems of specialized design. (pp. 426, 427)

Open loop systems, on the other hand, which have an analogue in current reinforcement learning theory, are governed by reactions which are a direct function of external or hypothetical internal stimuli, and are a matter of a linear relationship between input and output without continual regulation of behaviors via intrinsic or external feedback. Motor adjustments, if they occur, are made after the fact, as a result of consequences of reinforcement.

Smith's primary research concern has been variation of exteroceptive feedback as in delays or distortions of it in experimental situations. In general, he has found that abnormal and distorted feedback in any modality is very deleterious to motor tasks of an ongoing,

continuous nature (e.g., speech, tracking, drawing, singing, playing music, etc.).

In 1962, K. Smith and W.U. Smith reported several feedback experiments on the space-structured characteristics of motion. The authors proposed that the primary movement systems were firstly postural, secondly transport, and thirdly, manipulative in both order of development and importance to behavioral regulation. In some experiments, distorted visual feedback was manipulated by televised inversion and reversal of ongoing handwriting, maze tracing, and target tapping activity. Since the postural (up-down) reference system was analyzed as more crucial to organismic function than the transport (right-left) reference system, it was expected that visual inversion of stimulus feedback would be more deleterious to perceptual-motor function than reversal or inversion-reversal conditions. These views were confirmed for durations of both contact and travel movements of handwriting and maze-tracing activity. Ss could usually achieve a normal efficiency level over several days practice, particularly for simpler tasks and under reversal feedback conditions.

Other research on delayed feedback (Smith, 1966) for tasks like writing, speaking, breathing, maze-tracing, musical instrumentation, etc. has shown by and large, that delayed feedback is deleterious to the point of a S's being unable to continue his task, or being able to make only limited improvements. This, it would seem, is to be expected as what in fact is happening is that at any given moment, S is performing some response with resultant inaccurate or inappropriate feedback. Performance should not be expected to be normal if reafference can never match efference. The feedback S is receiving is not in keeping with movements in time and thus, if S attends to such feedback,

he is subject to stimulation out of step with the temporal movement of his response. In such instances, S would have to perform without attending to the feedback in order to avoid assimilation of conflicting and confusing cues. Delayed feedback during ongoing continuous tasks is like "throwing a wrench" into the system, where no feedback is better than delayed feedback.

SECTION XI

BIOFEEDBACK AND VOLUNTARY MOTOR CONTROL

A primary interest of this paper and research is EMG biofeedback (BF). BF may be taken to mean exteroceptive feedback on bodily states. Owing to technology, it is a specific feedback technique which involves the monitoring, via electronic instrumentation, of bioelectrical potentials emanating from different bodily sources with immediate provision of the corresponding information to S. The feedback may be direct (e.g., an oscilloscopic display with acoustics) or transformed (e.g., a tone or light correspondingly proportional in pitch or brightness [or color] to response intensity).

One unique aspect of BF is the extreme precision of the exteroceptive feedback it provides. Another is the sheer availability of such artificial feedback. It is, in most cases, normally not available to S at all even through intrinsic channels. BF enables Ss to master controls which indeed are so difficult that they may often not carry over from the laboratory situation unless constantly or intermittently supplied with BF for maintainence.

There are many types of BF--cortical wave, intestinal contraction, blood pressure, etc. The only one to be considered here is muscle (EMG) BF. Physiological and psychological aspects of motor response have been discussed. Integration of the results of EMG BF and motor control will inevitably entail a resolution of a complex array of intricate relationships. To report on EMG BF is to report on a series of successes, yet both experimental and applied research is relatively recent. Viewed with caution, however, implications of the success of BF would suggest

finer levels of psychophysiological control by humans than is normally possible. They would suggest self-control which can readily be applied, with jurisprudence, to existing or new therapeutic procedures.

Experimental Studies

Single Motor Unit Control. EMG biofeedback takes its departure from a study done by Harrison and Mortenson (1962). These authors researched the possibility of training single motor unit (SMU) firing and obtained results which subsequent research has repeatedly confirmed. They recorded from motor units of the tibialis anterior (shin) muscle (both needle and surface electrodes). Ss could discriminate different pitch qualities of different SMU's from the EMG acoustics as well as observe them on an oscilloscope. With voluntary effort, most Ss could fire at least one unit, primarily by slight isometric contraction. In the more astute cases, Ss could fire different rhythms of one SMU (single, double, triple or quadruple contraction patterns). One S could keep up to six independent units firing simultaneously. This feat required a good degree of concentration. As in subsequent studies, the auditory and visual feedback seemed essential aids to control, the former a noticeably more salient cue than the latter.

Leibrecht, et al., (1973) investigated the effects of auditory vs. visual feedback on SMU training in the tibialis muscle as well as the effect of feedback (binary light with or without a raw EMG acoustical signal, needle electrodes) on retention. They compared two groups (one from a previous study). The previous group had received only binary visual feedback and another group now received binary visual plus augmented auditory feedback. The authors found that the latter group learned much faster, the effect being confined to the speed of learning

and not one of ultimate performance. Thus, the augmented dual-modality feedback made a quantitative difference in learning.

Carlsöö and Edfeldt (1963) compared the ability of Ss to control SMU activity with no exteroceptive feedback vs. activity with exteroceptive auditory (raw EMG acoustical signal) and visual (EMG oscilloscopic display) feedback. They found that learning, both in terms of time for S to search for a SMU, and time he is able to maintain SMU activity, is clearly superior for Ss with exteroceptive feedback. Their data also seemed to suggest that the auditory stimulus was more effective for learning than the visual stimulus. In addition, when some Ss received random noise or light flashes while trying to perform, the noise was by far more disturbing to performance.

Basmajian (Basmajian, 1967a, b, c; Basmajian and Simard, 1967; Basmajian, 1963; Basmajian, 1972; Scully and Basmajian, 1969) has worked extensively on SMU training through EMG feedback. In Basmajian's studies, Ss are supplied with an oscilloscopic display of motor units along with loudspeaker acoustics. Electrodes are intramuscular (Basmajian, 1967b). To test the extent of SMU control under varying conditions of distraction, Basmajian and Simard (1967) studied control of SMU's in the tibialis anterior muscle during contralateral limb movements, varying fixed ipsilateral limb positions, movements of the ipsilateral limb, and segmental body movements of head, neck and upper extremities. It was necessary to train SMU control during the various distractions.

The easiest control to maintain was that during movements in parts of the body other than the ipsilateral limb. Nearly all of the 32 Ss studied were successful at this. Maintenance of SMU control while making movements in the same limb was more difficult, requiring active

assistance from a technician in positioning the limb, preventing normal motor recruitment, etc. during training. Yet 1/2-2/3 of the Ss were successful in maintaining some joint positions and SMU activity. It was easiest for a distal (toe) joint, harder for a proximal (hip) joint, and hardest for a crossed (ankle) joint (where the muscle studied crossed the joint).

In another study, Scully and Basmajian (1969) induced massive muscle contraction of a muscle by electrical stimulation simultaneous with an S's ongoing conscious control of SMU activity in the same muscle. This did not interfere with SMU control.

Basmajian (1972) reports that almost all of the hundreds of Ss who have been tested can learn control of at least one SMU. Some few Ss can control up to six independent SMU's and/or produce different rates and rhythms of firing them. The most highly accomplished Ss (one in 20) can perform SMU control without any exteroceptive feedback. Recall in this task is "semiconscious"--i.e., there is no introspective knowledge of how one literally does it but rather an anticipation or imagination of sight and sound of the motor unit kept functional by subsequent feedback from E (exteroceptive feedback as knowledge of results).

Basmajian (1967c) notes that the mechanism of SMU control can be described as a loop system where the proprioceptive stimuli, integrated with auditory and visual stimuli, form the sensory side of the loop to reinforce learning. This leads to motor output in a final common pathway. The loop starts in a specific SMU activity and ends in the same response, but the sensory side (or integration of cues and output [current author's insert]) remains complex.

Wagman, et al., (1965) closely observed some of the essentials of SMU control. They reported that proprioceptive feedback to S mainly through muscle position, a pattern of muscle contraction, or even pressure on the skin (as when the muscle was put in a cast) was necessary to learn control. For example, SMU activity in the gastrocnemius (calf) muscle might appear only when there was dorsiflexion of the ankle joint. Or it might appear in the abductor pollicis brevis (thumb abductor) muscle only at a certain position and while other muscles controlling the thumb simultaneously contracted in a certain way. For all muscles studied, once learning was established, there was greater independence of proprioception of position or contraction pattern maintaining the response, and, as the authors say, more conscious control of an SMU. For example, in the case of the abductor pollicis brevis muscle, when S gained control of an SMU, he no longer had to produce the training position or overall pattern (detectably at least). Instead, S reported relying on recall of the position and pattern to reproduce the response. Finally, if two SMU's in the same thumb muscle were learned, each in a different thumb position, once well established, both could be fired in succession from one position.

In sum, studies of SMU control show that a) proprioception plays a crucial role in learning, and SMU control is entirely possible yet difficult to maintain without exteroceptive feedback; b) auditory and visual cues are important as exteroceptive feedback, the former seeming to be the more salient cue; c) a good deal of concentration and care is required to maintain the control beyond one SMU or in the context of other movement and noise distractions; d) control is not direct but an indirect adjunct to other position and contraction pattern movements and imagined EMG sights and sounds. It also involves substantial conscious effort.

Operant Procedures. Hefferline and co-workers were also early researchers in EMG feedback. The feedback was used to control covert muscle responses. Hefferline et al., (1959) and Hefferline and Keenan (1963) recorded an integrated potential from the thenar eminence (thumb) muscles of one hand (surface electrodes). (They also placed dummy electrodes elsewhere.) A positive reinforcement group consisted of monetary pay offs per numerical score increases visible to S. Negative reinforcement groups got instructions that a noise which was turned on over music a) concerned the effects of body tension on movement, or b) that an invisible response could turn off or avoid the noise, or c) that S could twitch his thumb to turn it off.

After a 10-minute baseline period, E reinforced S whenever some criterion response magnitude was observed (one which occurred not more than once in one or two minutes but which was higher than responses at baseline resting level--e.g., approximately from 25-100 μv vs. 10-20 μv respectively). A 10-minute extinction period followed. For all groups the frequency distribution of covert response magnitudes before and after conditioning was skewed towards the high amplitude end, whereas the frequency distribution of responses during conditioning showed that the distribution tended towards normality. For every S the reinforced response became most frequent usually with an abrupt increase in rate once conditioning began.[25]

Hefferline (1958) placed electrodes over the masseter (jaw) muscle and instructed Ss to move the muscle in accordance with keeping a meter before them midscale. The meter had a range of 0-5.0 μv and read in

---

[25]With the exception of those Ss told that a small muscle twitch could turn off the noise. These Ss were occupied with gross movement of the thumb and did not condition.

values of 0-1.0. Ss received a 10-second feedback period, a 10-second no feedback period, and a 10-second rest period over daily sessions. Performance with feedback immediately became a skill, implying that Ss already have good control of the masseter within their motor repertoire. Performance without feedback continued to improve over four days, finally to the level of performance with feedback. After a few days all Ss were able to respond tightly around midscale without feedback. Hefferline suggests that Ss learned a tension scale for themselves which did not need to rely on exteroceptive feedback. This is suggested in another part of the study where other Ss were trained with feedback to respond to three different values on the scale. Performance with feedback for the three values was not as accurate as performance under a single value. When these Ss were then required to approximate three slightly different values within .1 meter points of the trained values without feedback, they tended to somewhat undershoot or overshoot these values.

Hefferline and Perrera (1963) used an ingenious method of training covert response discrimination. The authors instructed S to key press (overt response) whenever he heard a tone imposed on background noise. S was rewarded with 2¢ per correct press. The authors simultaneously recorded covert thumb twitches and occurring covert key presses. They conditioned covert key presses to occur between thumb twitches and overt key presses. That is they explicitly shaped a behavior of covert key press preceding overt key press, without S's knowledge. At first the tone was presented as quickly as possible after a thumb twitch. Between the thumb twitch and tone (which led to the rewarded key press), there occurred a covert key press. Paradigmatically: thumb twitch → sub key

press → tone → overt key press → 2¢. In time, only those thumb twitches which were followed by sub key presses within two seconds were followed, through E's manipulation, by the tone. Both thumb twitches and correct sub key presses increased. The tone became functionally reinforcing for the sub key press as well as a discriminative stimulus for the overt key press.

Next the authors used a procedure of fading (after Terrace, 1963). The tone was slowly reduced in intensity to no tone at all. Still, with no tone, 72% of the thumb twitches were followed within two seconds by an overt key press. 80% of the overt key presses followed thumb twitches. Ss reported that they still heard the tone--an example of transfer of control from an exteroceptive stimulus to internal control.

In summary of his results, Hefferline notes

> Assumptions apart, we are nevertheless without clear evidence on a question whose status is belied by the term "augmented sensory feedback"--namely, whether it is afferent information which is augmented, as the term implies, or whether as recent reports suggest . . ., it is efferent information which is just as likely to be involved. On this point, we can only suggest that a study of the interactions between exteroceptive feedback and behavior might well provide a convenient model from which to make further assessments of the mechanisms of response discrimination. (Hefferline, 1970, p. 264) (Author's italics)

Sasmoor (1966) conditioned the muscle response of the right thenar eminence using EMG biofeedback (surface electrodes, some placed as dummy electrodes). Direct muscle potentials were fed to an apparatus which read response amplitudes and triggered a digital counter when a criterion response of a certain amplitude (20-30 μv) was encountered. The digital counter enabled E to count the number of reinforceable responses during a session. The apparatus was also connected to a counter which S had in view as well as a cumulative recorder.

For six Ss the instructions were to see how readily the pointer they had in front of them could move. Fifteen minutes were alloted for settling down, then 15 minutes for observation of operant level by E. The counter was then illuminated and 30 minutes of conditioning criterion responses ensued. The reinforcement was a movement of a pointer 30° around 360° of rotation. There was then 45 minutes of extinction: 30 minutes with counter lit and 15 minutes with counter dim. Four more Ss were similarly trained except that for two Ss criterion responses were those within the 10-20 μv range. There were two control Ss for whom the pointer was driven by responses from an experimental S.

It was found that reinforcement of responses abruptly increased the frequency of responses in general and in the conditioning range in particular soon after conditioning began. Yet for five of the six experimental Ss the highest response rate was in the 10-20 μv range. Before training, operant levels were markedly skewed on the low end of the response frequency distribution. During conditioning, the relative frequency shifted towards the reinforced range. Sasmoor suggests that a generalization gradient set up in conditioning may have interacted with the operant response gradient to produce these results. At extinction the distribution was again similar to operant level. The two control Ss increased their response rate during pointer movement although not nearly as dramatically as experimental Ss. Extinction for all Ss was abrupt.

Sasmoor refers to pointer movement as reinforcement. One could also call it exteroceptive visual feedback. The author reports that all experimental Ss said they were trying the move the counter and felt they did exhibit some control over it. They did not, however know what their

controlling responses were, which may have obscured contingencies and resulted in the less defined effect on the precisely manipulated range. Or again, judging from operant level distribution, the Ss may have been working within the confines of a range of responses where there were more low amplitude response-producing motor units or where fine discriminations were difficult (a 20-30 μv response vs. an operant level of 5-10-20 μv range). For example, response amplitudes conditioned by Hefferline et al., (1959) and Hefferline and Keenan (1963) were usually 45 μv and higher vs. a resting baseline of about 10-20 μv.

In general, evidence from the review of both Single Motor Unit Control Studies and Operant Procedures suggest that feedback enables Ss to make very fine motor discriminations.

Therapeutic Contributions

Miscellaneous Applications. The following therapeutic procedures, as well as several of the experimental studies, use augmented feedback, i.e., feedback proportional to the state of muscle contraction. However, in most of the following studies, the signal has been clarified--for example, the auditory signal by conversion into a proportional rate of clicks or a tone pitch instead of the direct acoustical sound of the EMG. There is evidence that auditory feedback is a good stimulus signal for state of muscle contraction (see Single Motor Unit Control, p. 67).

However, there has been no research on the type of auditory feedback most suitable for EMG BF under different experimental or applied conditions. Furthermore, when tone pitches or clicks are used, the sound quality of either may not be comparable among studies. Some tones could, for example, be aversive in which case they may be more useful in achieving a decrease in contraction for a corresponding decrease in tone pitch, etc.

A final point to be made is that in relaxation studies to be discussed, the frontalis (forehead) muscle is most often used as the target muscle. The assumption is that it is a most difficult muscle to relax (Budzynski and Stoyva, 1969) as well as a major site of tension (Malmo and Smith, 1955). Thus if success in relaxation can be achieved with the frontalis, total deep relaxation presumably can follow relatively easily and completely.

Budzynski and Stoyva (1969) report a between-group comparison of the extent of muscle relaxation possible with and without an auditory EMG feedback training procedure. The frontalis was used as the target muscle (surface electrodes). There were three groups and all were told to relax. An experimental group was also told that the pitch of a tone would vary with muscle tension. Most importantly, a shaping procedure was used for this group so that the S had to decrease his muscle action potentials (MAP's) more and more to keep the tone low. Each of the other groups were control groups and received either silence or a steady low tone. The final levels of frontalis MAP's were measured against S's original baseline. The experimental group did significantly better. Mean MAP decreases were 50% for the experimental condition, 24% for the silence condition and 28% for the steady tone condition, so that training with BF seems to have had a considerable effect on relaxation.

Again Budzynski, et al., (1973) conducted a study of EMG feedback (proportional clicks, surface electrodes) to alleviate tension headache. Tension headache involves sustained contraction of scalp and neck muscles. It was reasoned that relaxation of the frontalis muscle should result in tension headache relief through greater voluntary control over muscle tension. Ss were screened for severe psychological problems.

Ss also carefully charted their headaches two weeks prior to training to obtain baseline rates. Feedback training was carried out in the laboratory over 16 bi-weekly, 20-minute sessions. Ss also practiced the relaxation at home (no specific instructions given).

Compared with control groups of noncontingent feedback and no feedback, only the experimental contingent feedback group showed a significant decrease in tension headaches. The latter were able to reduce tension 50-70% after two to three weeks of training. There was also decreased drug usage for headache pain in this group as reported in a three month follow-up. Furthermore, the three month follow-up and then an 18 month follow-up of the experimental group revealed that most Ss were still benefiting from the feedback training. Besides headache alleviation, Ss reported being more generally relaxed at the 18 month follow-up.

Wickramasekera (1973) also used EMG feedback (proportional tone pitch, surface electrodes) to lessen frontalis muscle activity in five tension headache patients (again carefully screened for psychological problems and charted for baseline levels). Ss had been unable to benefit from other kinds of therapy. The author introduced a within-group control for placebo effect into his design by using a contingent feedback session, a non-contingent feedback session, and then another contingent feedback session. Ss were always under the impression that the feedback was "true". Baseline and non-contingent feedback sessions showed no significant difference. Contingent feedback sessions did, however, show a significant decrease in tension level. Thus, it is suggested that the contingent feedback effects the change.

Raskin, et al., (1973) monitored EMG levels of the frontalis muscle (surface electrodes) of patients with recalcitrant chronic

anxiety. Ss were given augmented auditory feedback (tone pitch) training to induce deep muscle relaxation proceeding with the frontalis muscle. Areas of primary concern were anxiety, sleep difficulties, and headaches. The authors confirmed the findings of Budzynski, et al., (1973) of markedly reduced tension headaches with feedback training.

The authors were conservative, however, as to their conclusions concerning how helpful EMG feedback therapy, used alone, might be in relieving chronic anxiety. The major finding was that although patients can definitely learn to relax, the effect is largely transient so that the training does not generalize to an anxiety-provoking situation. Relaxation must somehow be incorporated into the patient's life. Insomnia, in those of the patients who suffered from it, was amenable to some modification in that the S could get to sleep and did not need sedatives but the sleep was one of frequent awakenings, early risings, etc. Subjective reports indicated that Ss experienced states of tranquility during deep muscle relaxation.

Cleeland (1973) employed a design combining EMG feedback (surface electrodes; augmented tone pitch) and shock avoidance for purposes of treating torticollis, a disturbance of posture control in the head and neck muscles. The cause of torticollis is unclear. Cleeland used an experimental procedure tailored to individual cases. Ss were being seen medically for their condition. A type of treatment procedure was, for example, first providing S with auditory feedback on the activity of the problematic sternocleidomastoid (neck) muscle. S was required to progressively relax this muscle and upon each step of relaxation, move his head toward the midline. When movement was possible all the way to the midline, sustained posture may not yet have been possible because of muscle spasm. Cutaneous shock was then applied to the sternocleido-

mastoid immediately and throughout the duration of spasm until S learned to keep his head straight. Eight of the 10 patients studied reduced spasm frequency during laboratory trials. In a mean follow-up of 19 months, six of these Ss were still benefiting (three markedly and three moderately).

Another area where EMG feedback has augured potential benefit is in systematic desensitization therapy. The assumption here is that deep muscle relaxation implies a calm, relaxed state (e.g., Schultz and Luthe, 1959). There is some evidence that a relaxed skeletal musculature is associated with a non-aroused, parasympathetic, "trophotropic" mode of reactivity (see Gellhorn, 1968, this paper, p. 37). Systematic desensitization requires that the S be in a relaxed state in order to effect a non-anxious re-conditioning of responses to arousing stimuli.

Wickramasakera (1972) reported a case where EMG feedback was used as an adjunct to systematic desensitization therapy. The case concerned a written test phobia. Except for some initial and intermittent guidance, the patient was mostly self-trained in relaxation and therapy. Before desensitization, there were three sessions of verbal instruction on muscle relaxation. These were followed by daily 30-minute laboratory sessions with EMG augmented auditory feedback on the frontalis muscle and 30-minute relaxation periods at home. The therapist then went through the first three of 20 desensitization scenes with the patient after which the patient rehearsed the remaining 17 herself, all the while with the aid of EMG feedback. The treatment, which had a 21 day duration altogether, was apparently quite successful. Interestingly, Wickramasakera reports a regular relationship between increased EMG levels at imaginary scene onset and decreased levels at scene termination.

Budzynski and Stoyva (1972) have also incorporated EMG feedback into the behavior therapy procedure and have found it, thus far, to be quite useful. Before therapy begins, S is trained to relax his muscles deeply through EMG-augmented auditory feedback, starting with extensor forearm or preferably frontalis muscle if not too difficult. When a relaxed level is achieved, systematic desensitization begins, each session starting with a 10-minute feedback relaxation period. During therapy, E has a monitor before him which signals differently colored lights for EMG levels a) below criterion, b) somewhat above criterion, c) and "too much" above criterion. E thus monitors S's anxiety level and can terminate the imagined scene when EMG level goes too high. In between scenes, S receives feedback to bring him back to relaxation level. As the authors suggest, this precise physiological monitor of an S's supposed anxiety level can benefit many cases in behavior therapy where a lack of success may depend upon imperfect relaxation at the outset. Support for the importance of muscle relaxation comes from therapies which have met with a good deal of success like Jacobson's (1938) progressive relaxation technique and Schultz and Luthe's (1959) autogenic training method both of which revolve around a state of deep muscle relaxation.

However, muscle relaxation may not represent a state of non-anxiety for all Ss (Lader and Matthews, 1968). Although most Ss report tranquility during deep muscle relaxation, exactly how muscle tension fits in with the organismic response in its entirety for all Ss at any given moment is as yet unknown. Gellhorn (1968), for example does cite the possibility of an uncoupling of muscle tension from the ergotropic system via cortical control.[26] Individual differences in response

[26]Also, e.g., if one looks at muscle length and tension at the periphery alone, the two are inextricably related. However, tension in muscle can be independent of length under CNS influence and control. (The stroke patient motor problems in the current study are illustrative of pathological CNS influence on muscle tension.)

specificity suggest that EMG feedback may be used as one of many techniques to engender relaxation. Other physiological targets might include heart rate, blood pressure, or brain waves.

EMG BF has been used to control subvocalization during silent reading. Hardyck, et al., (1966) fitted Ss in a speed reading class with laryngeal muscle electrodes (surface) and headphones which fed back the acoustics of muscle response. The authors compared what was a clearly detectable difference between relaxation baseline EMG levels and subvocalization levels during silent reading. Ss were first told to swallow and listened to the resultant sound. They then trained to control the sound against relaxation under various conditions of talking, turning the head, etc. Ss then trained to control the noise of subvocalization responses while they silently read. Most Ss were able to control subvocalization within five minutes and all Ss did so by the end of a 30-minute session. A one and three month follow-up showed no subvocalization in any Ss during tests of silent reading.

Applications to Neuromuscular Problems, Particularly Stroke.

Marinacci (1972), reporting on his clinical experience, claims that paralyzed, weak or atrophied muscles may be amenable to re-use through auditory EMG feedback training. Lost voluntary control over the skeletal neuromusculature can result from a variety of causes. These can be upper and lower motor neural and/or muscular destruction, sensory aphasia, local edema, disorganization of motor engrams (e.g., from disuse because of pain), etc. Marinacci says that just how much training of voluntary control with EMG feedback can provide depends on a) the amount of irreparable physiological damage and b) the voluntary effort of the patient.

Intensive electromyographical study (needle electrode) of patients who have lost muscular control shows that one can often detect some neuronal firing, possibly due to impulses leading to reflexive activity or impulses elicited through voluntary effort. In the former case, auditory feedback may lead to eventual transfer of neural activity to some voluntary control. In the latter case, it may lead to full voluntary control. The progressive success with EMG therapy often appears to be due to an increase in the frequency of neuromuscular activity which leads to hypertrophy of the muscle from overuse. This hypertrophy facilitates the generation of giant motor units which can somewhat compensate for lost neural tissue. Also, surviving units can sprout and thereby innervate extra, formerly de-innervated muscle fibers. Exactly how this happens is unknown.

Marinacci points out that therapy can be arduous and patience and perserverence on the part of both the examiner and the patient are required. Also the re-educative process will not cure all neuromuscular ills, nor does it necessarily contain the regenerative steps outlined above.

Johnson and Garton (1973) used an EMG feedback technique (raw EMG visual signal and acoustics) on 10 hemiplegic patients to effect gross dorsiflexion of the ankle with a criterion aim of improved ambulation. The study was purposely very general in terms of procedure and evaluation, and consequently, informal in ultimate conclusions about return of muscle function. The authors wished to avoid "a highly regimented training routine".

Etiology of hemiplegia was eight cerebrovascular accident patients and two traumatic head injury patients. Pre and post experimental

muscle function was assessed in terms of muscle grade: (0 [no function], T [trace], P [poor], F [fair], G [good]); and ambulation: (+ [improved] or - [unimproved]). Dorsiflexion was trained by applying surface electrodes to the anterior tibialis muscle. (Some patients required a few needle electrode sessions and some patients were trained on the peroneus group when anterior tibialis training resulted in inversion of the foot during dorsiflexion.) The terminal training point for any patient was ambulation without a leg brace. During therapy, gross dorsiflexion was induced by massive flexion, gross facilitation, or reflex methods. The latter techniques are physical therapy (PT) techniques. Thus, the study was an implementation of BF and PT training.

Duration of training in the laboratory was a 30-minute session on each of three separate days. Following this, patients practiced at home with a portable EMG unit. Instructions were to practice a minimum of two 30-minute sessions per day. Patients were seen at one to two week intervals to assess function and to monitor development under the self-training program. The total time patients were involved in the training ranged from 2 to 16 weeks. Poorly motivated patients dropped out, another moved away, etc.

Results showed that all patients improved on muscle grade, even those with only two weeks of training. Distributions were one patient from 0 to P, five patients from 0 to F, one patient from T to F, one patient from P to G, and one patient from F to G. Five of the 10 patients showed no or little functional improvement in terms of overall gait.

Brudny, et al., (1974) have reported a large scale study of sensory feedback treatment for patients with disorders of voluntary movement.

Etiology of illness in the patient group was varied: 10 stroke, one hemiatrophy, one meningioma, one cerebral palsy, three torticollis, and five dystonia patients (four of whom were treated for torticollis). Disabilities treated were torticollis (13), dystonia (5), facial spasm (3), quadriparesis (2), and hemiparesis (13).

All but four of the patients (patients with hemiparesis) had been disabled for longer than nine months and some up to 25 years. (As spontaneous remission of symptoms is difficult to determine for the four early patients, they are eliminated from this review). All other patients, even those who had undergone some other therapy, had reached a plateau in terms of improvement.

The technique of feedback the authors used involved an audiovisual display of an integrated EMG potential (surface electrodes). Visual feedback was a dial pointer on a calibrated scale, or an oscilloscopic display of muscle action potentials, or a digital readout of integrated muscle potential measured in μv/sec. Auditory feedback was the raw acoustical sound of muscle action potentials or a proportionally augmented clicking rate. Performance was shaped by changing electromyographic sensitivity to increment standards of performance. Supplementary feedback was often used by having the patient view his progression in a mirror.

Ss were videotaped before and after treatment. Physiological parameters of relaxed and maximal EMG and integrated EMG of the target muscles and contralateral muscles were also recorded before and after treatment. Criteria for improvement were dependent on disability. For example, the authors cite the following criteria for hemiparesis and torticollis: for hemiparesis, upper extremity function gains were evaluated in terms of 1) relief of spasms with no functional component,

2) assistive function of the extremity, and 3) actual prehension. For torticollis, gains were evaluated in terms of how long a patient could maintain a neutral neck position with and then without feedback.

Duration of therapy for all patients ranged from 4-12 weeks with variable results for each category of disability. By and large, the results were impressive. For example, of the stable hemiparetic patients, four improved to prehension stage for upper extremity, four to assistive capacity, and one to spasticity control. For the torticollis patients, three maintained a neutral neck position over months with monthly reinforcement sessions--(in one case therapy was discontinued by E), six over several hours--(four had therapy discontinued), and four over minutes--(one had therapy discontinued). The two quadriparetic patients showed improvement from some degree of non-functional movement to some functional movement (e.g., one total care patient progressed to self care and driving a wheelchair). Two of the patients with facial spasms showed marked improvement, enough to return to work. Therapy seemed least successful for dystonia patients with the exception of one of the five who showed marked improvement of a left lower extremity dystonia.

It is very important to note that patients were screened for minimal involvement of aphasia, organic mental syndrome or any mental deficiency which might impair ability to follow instructions. Furthermore, all Ss had some minimal form of voluntary control in the affected limbs.

Brudny, et al., report that Ss quickly apprehended the changes which could be affected through biofeedback signals. The authors interpret various patient problems as an interruption of normal physiological feedback control at varying anatomical levels of the CNS. Biofeedback,

as an indirect supplement to normal feedback operation, is seen to result in transfer of control to direct systemic monitoring of voluntary function.

Brudny, et al., are now training patients with a modified EMG machine which has a single-beam (a dot moving up and down across the scope) oscilloscope display that rises and falls according to the state of contraction over a visible 10-second time scan across the screen. There is again a simultaneous digital printout of muscle action potentials measured in μv/sec and a simultaneous augmented tone.

Basmajian, et al., (1975) compared the effects of conventional PT vs. a combination of PT and BF on two groups of hemiplegic patients (10 patients in each group) over a five week training period. All patients had suffered stroke and the condition under treatment was footdrop. Patients were a minimum of three months poststroke and were able to ambulate with or without a cane and/or short leg brace. Prior to training, patients were rated as belonging to one of three basic recovery stages. Dependent measures were strength of dorsiflexion measured in kilograms by a spring dynamometer device attached to the foot, active ROM measured in degrees of motion, and improvement in gait pattern ranked according to one of five categories (ranging from no dorsiflexion to normal gait).

Training consisted of 40 minutes of PT for one group, and 20 minutes of PT plus 20 minutes of BF for another group. All patients showed improvement on strength of dorsiflexion and active ROM measures. The group receiving PT plus BF showed twice as much improvement on these measures over the group receiving PT alone. Gait changes were most notable for three patients of the PT plus BF group. This was in

terms of both a more normal gait pattern and the length of time over which these patients could maintain control. Three patients in the PT only group made gains in gait pattern in terms of their immediate initial response to walking, but they were not able to control the pattern over even a slightly significant amount of time. The latter patients, however, started from a lower recovery stage than did the more successful patients.

Follow-up of patients for their gait patterns at 4 to 16 weeks revealed that two of the three PT plus BF group patients who had made notable gains (one of them was not followed) showed no regression; also, one patient from that group who had not made much progress in gait immediately following training, had further improved at follow-up. All other patients in both groups maintained a status of gait function similar to that at the termination of training.

Kukulka, Brown, and Basmajian (1975) have used their own model of a mini-EMG muscle trainer on three patients to facilitate functional return of the long finger flexor muscles after surgery on the finger flexor tendons of the hand. Patients first received 15 minutes of whirlpool bath and 20 minutes of therapeutic exercise. This was followed by 20 minutes of EMG therapy (raw visual signal and acoustics, surface electrodes). Patients practiced various hand motions, starting with non-specific massive motion to more isolated movement at the finger joints. Patients were assessed on active ROM of the index, long, and ring fingers at the MP, PIP, and DIP joints. EMG therapy resulted in good return of motion, whereas the usual prognosis for return of motion in patients with such lacerations has been moderate to poor.

Considerations in the Current Study. The current study also set out to investigate BF on a stroke patient population. PT, a program with known validity, was incorporated into experimental design for purposes of a within patient control. The objective of the present study was to assess 1) the viability of BF as a technique of neuromuscular education for hemiparetic victims of stroke; 2) the efficiency and limitations of such a procedure in clinical practice; 3) the use of BF as a technique for investigation and analysis of the role and mechanism of motor behavior in the human being.

The aforementioned studies using BF training for hemiplegic conditions resultant from stroke addressed themselves to global qualitative or functional assessments of motor control as defined by neurological or physiatric criteria. In contrast, the current study was primarily concerned with an investigation of the learning process involved in BF training. Thus, this study focused on isolated training of a specific muscle corresponding to a particular movement for experimental purposes of delineating the learning process with respect to one muscle and motion. The study incorporated a) fundamental quantitative parameters which would be affected by learning; b) qualitative measures of patient capacities for learning and motor control; and c) descriptive evaluation of process through detailed observation of patient behavior.

Of the studies cited above, Brudny et al., (1974) rated patient gains on a neurological global scoring basis. Basmajian et al. (1975), rated patient gains in terms of muscle strength and functional improvement in ambulation. (They also measured active ROM which was a measure used in the present study.) Strength and functional improvement in ambulation are extremely important clinical measures and ultimately

most relevant to personal patient experience. Yet, for example, for the present study, strength was not chosen as a measure. The reasons are two-fold. First, in patients with spasticity, as in the current and above samples, there is a certain proportion of strength which is involuntary, a non-learning result of spasticity; there is no way of identifying involuntary and voluntary components of this strength. Second, using strength as a dependent measure, it cannot be assumed that a target muscle is the only one contracting. Strength is the resultant of the force of an agonist minus that of its antagonist muscle contraction. Furthermore, there are actions in which more than one muscle contributes synergically to the total force of the contraction. With spasticity, the contributions of each is difficult to assess. Without experimental control over muscles other than the target muscle, specificity of the muscle contraction contributing to strength is lost.[27] Thus, change in strength does not permit definitive conclusions about learning related to a target muscle contraction. On the other hand, active range of motion, for example, is more precise and accurate as it reflects the total excursion of a single muscle.

It might also be mentioned, that the above cited studies on BF and stroke hemiplegias did not aim for a statistical treatment of outcome criteria (dependent measures). The present study was designed to subject quantitative parameters to statistical analysis. There were as many controls as feasible in design, technique, and training. Yet, as in the above studies, qualitative descriptive statements of individual patient progress are the most valuable indices of process and efficiency,

---

[27]An EMG measure obtained from surface electrodes on a target muscle also entails the difficulty of isolating a specific muscle contraction; but observation of a muscular contraction pattern with explicit attention to the contraction of one muscle group minimizes the contribution of extraneous synergic contraction.

from any point of view. That is, the learning process is best evaluated by attention to the necessarily heterogeneous nature of patient syndromes and behavior.

SECTION XII

WHAT IS STROKE?

Before proceeding to the METHOD section, this study will address itself to a final issue--the question of what is stroke. The answer is a complex and multi-faceted one. Thus, the following is, at best, a very general outline. Furthermore, the outline refers to the effects of stroke damage on primarily the gray matter structure or cortex of the brain but effects reported may also include damage to deeper brain structures such as the limbic system, basal ganglia, and internal capsule.

Stroke is the result of insufficient blood perfusion to the brain for a critical period of time--a time long enough to cause necrosis of brain cells. The physiologic causes of stroke are as follows: narrowing of blood passages due to arteriosclerotic disease can lead to thrombosis or embolism of cerebral vessels. Or, hypertension or aneurysm of cerebral vessels can cause hemorrhage by leading to the rupture of vessel walls. There can also be inadequate blood flow to the brain as a result of an alternation of the hemodynamics of the circulatory system. Such events, in turn, cause ischemia, which means insufficient blood nutrients supply (particularly oxygen) to an area of brain cells, whereby the cells are impaired or killed. Actual sensory, motor, or other behavioral impairment depends on the area and amount of brain tissue destroyed.

Sensory impairment and motor impairment are contralateral to the brain lesion site. Sensorily, vision, touch and proprioception can be impaired by stroke. Motorically, muscle function can be impaired with

weakness ranging from severe flaccidity to excessive tone with varying degrees of spasticity and possibly contractures (the latter is a result due to lack of movement because of increased muscle tone).

Aphasia commonly occurs with left (i.e., usually dominant) cerebral hemisphere lesions. Aphasia is an impairment of language function which can affect the generation and/or understanding of spoken-verbal or graphic-verbal symbolic activity. It does not necessarily imply generalized intellectual impairment. The latter is the result of destruction to areas subserving other intellectual functions, as well as higher abstractive functions.

Visual-perceptual, spatial-perceptual, or visual-motor function, and associative-abstractive intellectual processes are more commonly impaired with right (i.e., usually non-dominant) cerebral hemisphere lesions. Altogether, stroke can have debilitating effects on communication, higher level thought processes, and emotions.

Thought processes affected by stroke are for example: orientation, memory, planning, reasoning, judgment, abstract thinking, concentration, mathematical ability, etc. The emotional effects of direct organic impairment may be emotional lability (e.g., frequent lacrimation to the slightest, even inconsequential, stimulus, and, less commonly, inappropriate laughter); or inattentiveness (lack of attention span), irresponsibility, unresponsiveness to major cues, irascibility, rigidity of thinking, etc. All of the above outlined functions can be altered separately or in combination, and can vary in degree by virtue of the systemic role, as well as the geographic location, of the lesioned tissue.

Emotional reactions secondary to stroke manifest themselves as individualized, realistic, pragmatic reactions against a sudden, over-

whelming debilitation and dependency, which almost inevitably results in feelings of insecurity or profound sorrow. Psychologically, a stroke patient must face himself as a different, sometimes drastically different, person. If there is a loss of abilities which were highly prized by an individual before he had his stroke, his acceptance of his poststroke status is all the more traumatic for him.

A stroke victim, furthermore, must sustain himself in a society where he is handicapped and where the handicapped person is an isolated or ostracized individual. Environmental influences surrounding the handicapped stroke victim are often less than encouraging or secure, particularly where disability leads to confinement in a nursing home, where there is no supportive family, or where friends fall away, etc.

Barring a severe, global impairment or direct organic emotional impairment, a person who has suffered the precipitous, debilitating effects of stroke, and who eventually tries to compensate against irrevocable depression, or who tries to re-educate himself, or who even simply tries to maintain a sense of self-dignity--especially when there is little or no environmental sustenance--must be a person of fortitude, resolution and courage.

In sum, disability from stroke presents a multitude of problems, all of which may effect learning ability. If there is aphasia, communication problems can be greatly frustrating and language rehabilitation is a long, arduous process.

Lost sensory function is essentially irreversible after any gradual, "spontaneous recovery" period. It is commonly assumed that there is no change in sensory function after such a period. A tentative suggestion presents itself as follows. If one looks at sensory processing in a

similar way as movement processing--i.e., as an integrative function involving broad areas of the brain, then it is perhaps conceivable that it may be possible to reorganize sensory processing at central levels just as it is possible to learn new motor function in a similar way. Where there is sensory, in addition to, motor impairment, new sensory processing at some level, may occur concomitant with new motor processing. Such sensory change may be revealed, in some instances, as peripheral, observable change occurring concurrent with observable motor change.

It is generally agreed that six months is the time span for spontaneous recovery of motor function after stroke. In general, the lesser the sensory impairment, the better the prognosis for motor return. If motor return through spontaneous recovery or rehabilitation is not forthcoming, any regained motor function is considered maximal. That is, the residual motor loss is diagnosed as virtually "permanent".

All of the factors outlined above enter into any work with stroke victims. Further, as a result of brain damage, many stroke patients suffer a diminished level of activation, as distinct from motivation, which interferes with central energizing from higher cerebral central control over reticular activating centers. A therapist must deal with all of the above. Needless to say, the therapeutic situation requires a therapist who is proficient and empathetic.

Neuromuscular education in stabilized stroke conditions is an impressive example of learning. Motor cells of the pyramidal and extrapyramidal systems have been destroyed and a patient has organically diminished or interrupted motor output. At the risk of redundancy, it is re-emphasized that brain cells have been destroyed and pathways interrupted. The process of acquiring new function is a process of

learning; the evaluation of the behavioral development of new function must be viewed within a learning framework, as what is being evaluated is the process of new learning as applied to available, employable physiological structures. The learning process in this study is signified by 1) the unlearning of self-defeating poststroke patterns; 2) the possible relearning of the use of old mechanisms which may yet be accessible; or 3) the learning of completely new repertoires along new pathways utilizing available intact brain structures which can accommodate such phenomena. (Observations of the psychological processes, learning phenomena, and an evaluation of the statistical outcome of dependent measures indicative of the establishment of motor control for patients in this study are considered in future sections.)

In conclusion, the damage sustained by the patients in this study, except for Patients 4, 6, and 8 (see Neurological Examinations for these patients in Section XIV), was varying degrees of unilateral cortical damage involving pyramidal or extrapyramidal regions of the cortex with varying degrees of sensory loss, oftentimes some degree of aphasia, and other behavioral disturbances. These impairments are described as completely as possible by sensory, motor, and neuropsychological assessment. Prime attention, of course, is given to baseline motor function and the ensuing learning process in discussion of both the case histories and statistical results.

SECTION XIII

METHOD

Subjects

Patients (n = 10) were chosen from several Twin Cities hospital files, nursing homes, or institutes for the handicapped. They were initially screened by physiatric examination by Daniel Halpern, MD, at the Department of Physical Medicine and Rehabilitation, University of Minnesota Hospitals. Many candidates were seen for initial screening. The determining criteria were a) an hemiparesis of the upper extremity resultant from cerebral vascular accident (CVA) with significant room for improvement in one muscle or muscle group; b) a relatively uncomplicated medical history besides the CVA; c) a minimum of one year poststroke status; d) a workable amount of cooperation, motivation, and attention on the part of the patient. All patients met the above criteria.

Patients chosen had sustained varying amounts and distribution of cellular damage to the cortex of either the right or left hemisphere. Thus, the extent of impeded motor activity and sensory and cognitive incapacitation was variable across patients.[28] In all patients, hemiparesis was the result of basic sensori-motor impairment except for Patient 7 whose motor deficiency included an apraxia.

The actual duration of poststroke status was one year, zero months to twelve years, eight months. Patient age range was 39 to 75, which

---

[28]There was suggestion in the neurological examination of deep infarction (capsular and basal ganglia) in Patient 6; extensive thrombosis in Patient 4; and possible bilateral damage in Patients 4 and 8.

included one 39 year old patient, seven patients from 50 to 64 years of age, and two patients 75 years of age. There were three females and seven males. Hemisphere distribution of the CVAs was three right CVAs and seven left CVAs.

Choice of Target Muscles

Specific target muscles were chosen as follows: surface electrodes were to be used. Selection of a target muscle was limited to easily accessible superficial muscles. (Thus, finger muscles were not even considered.) The degree of function in the possible different muscles of the upper extremity was evaluated for each patient. Subject to the above constraints, a muscle of the arm or forearm was chosen as the target muscle if the patient had some, even minimal, voluntary control over that muscle and, if there were significant room for measurable improvement in that muscle.

Actual target muscles chosen for patients were the wrist extensors in eight of the cases, seven of which the activity trained was greater contraction and one the activity was greater relaxation or inhibition; and the biceps in two cases, one in which the activity emphasized greater contraction and one relaxation.

Preliminary Tests

Neurological Examination. Prior to any treatment, patients were given neurological examinations (reported in Section XIV; also, patient Sensory Function summarized in Table 1). The neurological examination was carried out by Alan B. Rubens, MD, at the Hennepin County Medical Center, Minneapolis. The neurological examination consisted of assessment of motor performance and sensory impairment as well as testing for apraxia and a brief description of aphasia. The motor examination

Table 1

Sensory Function

Weights Assigned 3 Groupings of Sensory Function[a]			Patient #									
			1	2	3	4	5	6	7	8	9	10
	1	Pin	0	1	1	1	1	0	0	0	1	0
		Touch	0	1	1	1	1	1	0	1	1	1
		Vibration	1	1	1	1	1	1	1	1	1	1
	2	Position Sense	0	0	1	1	1	1	0	0	1	1
		Two Point Discrimination	1	0	1	0	0	0	0	1	1	0
	3	Graphesthesia	1	1	1	0	1	0	0	0	1	1
		Stereognosis	0	0	1	0	1	0	0	0	1	0
Maximum Score Possible	13	Patient Score	6	6	13	5	11	4	1	4	13	7

Impaired[b] = 0
Intact = 1

[a]For purposes of correlational analysis, sensory function was divided into a "hierarchy" of three categories and each category was assigned a weighted score. Category weight values are 1, 2, and 3. A patient's score is derived by adding his scores within a category and multiplying them by the category weight value.

[b]"Impaired" indicates a sensory impairment which can range from mild to severe. Sensory function was recorded as impaired only when impairment included the paretic upper extremity.

included: 1) a description of muscle tone and spasticity on the hemiplegic side; 2) bilateral testing of deep tendon reflexes (DTRs); and 3) an evaluation of motor weakness tested as apparent muscle strength against external Examiner resistance, graded on a scale of zero to five where 0 = no evidence of contraction; 1 = Trace (anything from visual or tactile evidence of slight contraction, palpable movement of the tendon, or muscle contraction including some joint motion); 2 = Poor (complete range of motion with gravity eliminated); 3 = Fair (complete range of motion against gravity); 4 = Good (complete range of motion against gravity with moderate resistance); 5 = Normal (complete range of motion against gravity with full resistance).

Sensory examination included bilateral testing of responses to seven categories of sensation and sensory function as follows:

Pin: Patient sensation of pin prick applied to the affected and non-affected sides. The patient indicates whether he can feel the sharpness of the stimulus and whether it is felt as the same or different on his affected compared against his unaffected side.

Touch: Patient sensation of a cotton wisp applied to the affected and non-affected sides. The patient indicates whether he can feel the stimulus and whether it is felt as the same or different on his affected compared against his unaffected side.

Vibration: Patient sensation of a vibrating tuning fork applied to the skin over a bony prominence of the distal upper and lower extremities and responded to as above.

Position sense: With patient's eyes closed, patient appreciation of the direction of motion at joints passively moved by the Examiner. The directions and joints tested were usually upward and downward motions of the distal great toe and thumb or index finger joints.

Two point discrimination: With eyes closed, two points of an esthesiometer were applied lightly to the skin starting with a standard distance of 1 mm and increased by 1 mm intervals. The patient reports whether he feels the sensation of one or two points. Two point discrimination was recorded as the minimal distance at which two points were identified as separate by the patient. The areas usually sampled were the pulp of the index or middle fingers.

Graphesthesia: With eyes closed, patient identification of letters and numbers traced with a pencil over the palm and the dorsal surface of the wrist and hand.

Stereognosis: With eyes closed, patient recognition of objects placed in palm of the hand. Representative objects are a pen, key, spoon, coin, etc. In cases where paresis interfered with movements of palpation, the Examiner moved the object over the hand, particularly the thumb and index fingers, to maximize the opportunity for tactile identification.

Apraxia testing - Apraxia testing consisted of requiring a patient to attempt, upon command, specific motions in the paretic and contralateral limb as well as several gestures involving the facial musculature--for example, whistling, sipping through a straw, wrinkling the nose, etc. When patients were unable to respond upon command only, the Examiner demonstrated the gesture, and then had the patients try it.

Aphasia - Patient's ability to verbalize was observed; any gross defect in language ability was recorded as aphasia.

Neuropsychological Testing. Patients were also tested on a neuropsychological battery under the direction of Manfred J. Meier, PhD, at the Neuropsychological Laboratories, University of Minnesota Hospitals. The inventory is tabulated in Table 2 and included the following:

A. Wechsler Adult Intelligence Scale (WAIS) (Wechsler, 1955) (seven subtests):

Table 2

Neuropsychological Inventory

	Patient #									
	1[a]	2	3	4	5[b]	6	7	8	9[c]	10[d]
WAIS Full Scale IQ	83	92	108	97	114	85	95	87	78	84
WAIS Verbal IQ	77	86	106	96	117	92	107	87	114[e]	80
WAIS Performance IQ	92	100	109	98	108	78	80	88	94	91
WAIS Subtests:										
Comprehension	7	8	12	9	14	10	12	10	NA[f]	6
Mathematics	8	5	9	9	13	8	10	6	4	8
Digit Span	7	8	9	8	10	9	13	8	NA	4
Vocabulary	4	11	13	10	13	9	10	7	NA	10
Block Design	9	8	13	11	12	8	6	10	7	10
Picture Arrangement	8	10	9	10	10	5	8	8	12	8
Object Assembly	9	11	12	7	12	4	5	6	7	7

[a]Vocabulary score difficult to interpret as English was a second language, relatively imperfect to native tongue poststroke for this patient.

[b]

[c]Verbal scores not applicable--complete expressive aphasia in this patient.

[d]All verbal scores confounded by expressive aphasia for this patient.

[e]Shipley Hartford score substituted for WAIS Verbal IQ.

[f]NA = Not Applicable.

- CONTINUED ON NEXT PAGE -

Table 2(Continued)

	Patient #									
	1	2	3	4	5	6	7	8	9	10
Wechsler Memory Scale (WMS)	94	96	116	80	143	79	92	79	NA[f]	96
WMS Subtests:										
General Information	5	5	6	2	5	6	3	5	NA	5
Orientation	5	5	5	3	4	5	3	4	NA	5
Mental Control	7	2	7	4	9	6	9	0	NA	0
Memory Passages	6	10	12	4	17	.5	5	7.5	NA	10.5
(↑ 60' delayed recall)	7	7	12	0	15.5	0	0	2.5	NA	9.5
Digits Control	8	8	10	8	10	9	12	9	NA	6
Visual Reproduction	7	13	11	4	13	1	2	4	NA	4
Associates Learning	12	7.5	18	7	20	7.5	9.5	9	NA	16
(↑ 60' delayed recall)	9 6e,3h[g]	5 5e	10 6e,4h	4 4e	10 6e,4h	5 5e	6 6e	3 3e	NA	8 6e,2h

[g] e = "easy" associations; h = "hard" associations.

- CONTINUED ON NEXT PAGE -

Table 2(Continued)

	Patient #									
	1	2	3	4	5	6	7	8	9	10
Porteus Maze	7.5	16.5	16	9	13.5	10.5	8.5	17	9	10
Trail Making, Part A[h]	71"-0	146"-0	42"-0	98"-0	44"-0	105"-0	180"-0	71"-0	170"-0	77"-0
Trail Making, Part B[h]	205"-3	365"-3	63"-0	500"-0 Incom-plete	89"-0	371"-4	244"-5	244"-5	230"-5	132"-0
Bender-Gestalt Recall	5	6	7	1	6	0	1	3	1	3
Aphasia Screening Test (AST)[i]	8 1v,7nv	4 3v,1nv	0	8 7v,1nv	0	7 6v,1nv	0	4 4v	25	15 15v
Tactile Formboard I[j]										
LH[k]	20"-10	29"-10	33"-10	20"-10		43"-10		28"-10	31"-10	30"-10
RH[k]		134"-10	18"-10		18"-10		33"-10			
Tactile Formboard II[j]										
LH	210"-10	300"- 7	300"- 8	300"- 5		300"- 2		290"-10	232"-10	257"-10
RH			62"-10		282"-10		300"- 1			

[h]Score indicates time and number of objects placed.

[i]v = verbal; nv = non-verbal errors

[j]Score indicates time and number of objects placed.

[k]LH = Left Hand; RH = Right Hand

1. WAIS Verbal Intelligence Quotient (VIQ)--derived from Comprehension, Digit Span, Arithematic, and Vocabulary subtest scores.
2. WAIS Performance Intelligence Quotient (PIQ)--derived from Picture Arrangement, Object Assembly and Block Design subtest scores.
3. WAIS Full Scale IQ--Prorated from the above subtests.

Subtest scores reported are age-scaled: average is 10; one standard deviation is three points.

B. Aphasia Screening Test (AST) (Halstead & Wepman, 1949)--measure of verbal and related perceptual abilities. Scored as number of errors. Range of possible errors is 0 to 32.

C. Porteus Maze Test (Porteus, 1959)--maze tracing requiring visuomotor ability, foresight and planning. Reported as a Test Age (TA) score. A TA of 13 is equivalent with a mean IQ of 100; a year in either direction from 13 is equivalent with approximately 5 IQ points.

D. Trail Making Test, Parts A & B (Reitan, 1958)--serial tasks requiring visuomotor ability and concentration on extended sequencing (connecting numbers to numbers [Part A] and numbers to letters to numbers, etc. [Part B]). Scored as performance time in seconds and number of errors. Median performance for aged normals for Part A is approximately 90 sec, 0 errors; for Part B, approximately 120 sec, 0 to 1 errors.

E. Tactile Formboard Test, I & II (e.g., Meier and Resch, 1967)--visuomotor-tactual performance task of object recognition and placement executed with vision (Part I) and blindfolded (Part II).

Scored as performance time in seconds and number of objects placed (10 objects is maximum). With vision, average for aged normals is approximately 20 sec, 10 objects placed; blindfolded, average is approximately 200 sec, 10 objects placed.

F. Bender-Gestalt Recall (Bender, 1938)--Test of visuomotor-spatial ability to copy designs, and visual-motor memory of the designs. Scored as number of recalls in reproduction of copied designs. Of 9 possible recalls, median is a score of 5.

G. Wechsler Memory Scale (WMS) (Wechsler, 1945):

1. WMS Memory Quotient--Age-corrected memory quotient. The mean quotient is 100 and the standard deviation is approximately 15, but varies as a function of a S's age. The WMS was based on the following subtest scores:

   a. General Information
   b. Orientation
   c. Mental Control
   d. Logical Memory
   e. Memory Span
   f. Visual Reproduction
   g. Associate Learning

Subtest scores reported are raw scores where the maximum scores are 6, 5, 9, 23, 15, 14, and 21, respectively. Patients were also tested for 60 minute delayed recall on the Logical Memory, and Associate Learning subtests. Maximum scores for the latter are 23 and 10, respectively.

Other tests administered included the following:

H. Minnesota Multiphasic Personality Inventory (MMPI)--The MMPI was administered to determine possible pathological profiles. Profiles showed no marked deviations and all configurations were well within acceptable limits for a stroke patient population. In addition, the relationship of ego-strength (Es), depression (D), and psychosthenia (Pt) to clinical outcome was of interest (MMPI scores tabulated in Table 3).

Audiometry Testing. Patients were also tested in audiometry under the direction of Frank M. Lassman, PhD, at the Audiology Clinic, University of Minnesota Hospitals, to determine auditory acuity, speech audiometry, and ability to judge pitch and intensity differences analagous to those used as an auditory stimulus in BF training. There were no significant deficits in any patients on the audiometric tests. (See Appendix for details of the testing.)

Design

After preliminary inventories, baseline measures (described below) on target muscles and movements were begun. Four measures were taken over four different days of a two week period (two days in each week). On the first and last of baseline days, similar measures were taken in the contralateral, unaffected arm. Following baseline, an AB, BA Cross Over design was employed. A will refer to BF and B to PT. Five patients received BF following PT and five were trained in the opposite order. Patients were randomly assigned to one of the two possible groups. The exception to the randomization is the fact that three of the ten patients were right CVAs and seven were left CVAs. It was deemed essential that all three right CVAs did not fall into any one

Table 3

MMPI Scores

	Patient #									
	1	2	3	4	5	6	7[a]	8	9	10
L	46	44	50	53	50	50	44	50	56	56
F	96	60	46	50	55	60	62	66	58	66
K	35	62	55	59	62	53	49	35	42	44
Hypochondriasis (Hs)	65	82	52	70	77	62	62	44	46	62
Depression (D)	68	80	34	70	48	56	72	57	63	72
Hysteria (Hy)	51	62	54	76	78	62	67	43	49	44
Psychopathic Deviate (Pd)	46	53	57	53	71	55	62	50	60	53
Masculinity-Femininity (Mf)	59	47	34	69	57	51	39	63	61	67
Paranoia (Pa)	76	62	59	47	59	53	56	53	59	73
Psychasthenia (Pt)	66	60	50	69	60	58	52	65	56	64
Schizophrenia (Sc)	88	80	51	63	78	73	65	63	57	74
Hypomania (Ma)	75	35	60	55	78	70	63	63	73	65
Social Introversion-Extroversion (Si)	69	55	50	57	42	49	49	66	62	61
Ego-Strength (Es)	24	56	58	43	46	43	35	34	43	32

[a]This patient left 98 questions randomly unanswered throughout the MMPI and thus his profile is of questionable validity.

group.[29] Thus, the three were randomly assigned to both groups and the other seven patients were randomly assigned to the remaining slots (Group distribution tabulated in Table 4).

BF and PT training were begun following baseline. Training progressed a total of eight weeks, consisting of two 4 week blocks, Phase I and Phase II--BF first and PT second, or vice versa. Patients were seen at thrice-weekly intervals and performance tests identical to those at baseline were taken consecutively at the 4th, 7th, 10th, and 12th sessions of two consecutive 4 week periods. This provided four tests in each training phase. (For experimental protocol see Table 5.) (Also for a synopsis of patient prior therapies and pre-experimental functional status see Table 6.)

Measures

Test measurement consisted of two parts, always in the same order. First, a Command situation, also referred to as Part A, where the patient responded to five repetitions of two commands to 1)"raise the wrist" and 2) "let it go" (in the cases where wrist extensor contraction was required), or 1) "bend the elbow" and 2) "let it go" (in the one case where biceps contraction was required), or 1) "straighten the elbow" and 2) "bend the elbow" (in the one case where biceps relaxation was required). Secondly, the patient was instructed in a Rate situation, referred to as Part B, to contract and release the target muscle on his own, trying to move repeatedly as correctly and rapidly as possible during a one-minute period. Emphasis here was both on correct movement and rate. Sample instructions: "You should make the movement as fast as you can but do not forget to make each movement as good as possible."

---

[29]Right and left CVAs classically evince different neurobehavioral symptoms.

Table 4

Training Protocol and Patient Characteristics

Patient	Phase I	Phase II	Age	Sex	Hemisphere Lesion	Poststroke Duration	Hemiparesis of Affected Upper Extremity	Aphasia	Amount of Practice at Home[a]
1	PT	BF	54	M	Left	6 yrs 1 mo	Spastic; slightly flaccid	None	Considerable
2	PT	BF	56	M	Left	3 yrs 1 mo	Spastic; normal tone	Anomia; expressive aphasia at complex syntactical levels	Limited
3	PT	BF	39	F	Right	12 yrs 8 mos	Dystonia of hand & forearm muscles	None	Considerable
4	PT	BF	75	M	Left[b]	2 yrs 4 mos	Spastic; slight increased tone	Dysphonia & Spastic dysarthria	Moderate
5	PT	BF	54	M	Right	1 yr 9 mos	Flaccid as to amount to flail	None	Considerable

- CONTINUED ON NEXT PAGE -

Table 4(Continued)

Patient	Phase I	Phase II	Age	Sex	Hemisphere Lesion	Poststroke Duration	Hemiparesis of Affected Upper Extremity	Aphasia	Amount of Practice at Home[a]
6	BF	PT	62	M	Left	1 yr 6 mos	Spastic; excessive tone	Mild Dysarthria	Limited
7	BF	PT	62	M	Right	1 yr	Severe flaccidity	None	Limited
8	BF	PT	50	F	Left[b]	1 yr 6 mos	Spastic; increased tone	Dysarthria	Moderate
9	BF	PT	75	F	Left	2 yrs 6 mos	Spastic; increased tone	Complete expressive (Broca's) aphasia	Considerable
10	BF	PT	64	M	Left	10 yrs	Spastic; rigid-excessive tone	Moderate Broca's aphasia; mild Dysarthria	Considerable

[a]"Amount of Practice" during the experimental period was categorized as:
(1) Limited amount - very little practice, or for several experimental days, none at all;
(2) Moderate amount - fair amount of practice relative to patient's energetic ability;
(3) Considerable amount - full use of practice potential by a patient.

[b]Possibly bilateral lesion (see Neurological Examination for this patient)

Table 5

Schedule of Training and Testing

	Week	Group 1 n = 5	Group 2 n = 4
Baseline	1	△ △	△ △
	2	△ △	△ △
Phase I	3	□ □ □	○ ○ ○
	4	⊡ □ □	⊙ ○ ○
	5	⊡ □ □	⊙ ○ ○
	6	⊡ □ T	⊙ ○ Ⓣ
Phase II	7	○ ○ ○	□ □ □
	8	⊙ ○ ○	⊡ □ □
	9	⊙ ○ ○	⊡ □ □
	10	⊙ ○ Ⓣ	⊡ □ T

Sessions:
- △ Baseline
- □ Biofeedback
- ○ Physical Therapy
- t Testing with Training
- T Testing Only

Table 6

Prior Therapies and Patient Pre-Experimental Status

Patient	Time Elapsed Since Last Stroke	Prior Therapies[a]	Extremities Treated	Duration of Individual Therapies	Total Prior Therapy Time	Pre-Experimental Status[b]
1	6 yrs 1 mo	PT	leg	6 wks (U of M Hosp)	1-1/2 mos	Plateaued
2	3 yrs 1 mo	PT	arm & leg	1 mo (MMC Hosp) 1 yr (Courage Ctr)	1 yr 1 mo	Stopped therapy at Courage Ctr approx 2 yrs ago
3	12 yrs 8 mo	PT,OT,CT	arm & leg	6 mos (St.Marys Hosp) 1-1/2 mos (Sr. Kenney Institute) 3 mos (Courage Ctr) 3 mos (Private) 2 yrs (Private)	3 yrs 1 mo	Plateaued 3 yrs ago after extensive PT for upper extremity; pre-experimental residual was 3 yrs long-standing
4	2 yrs 4 mos	PT	arm & leg	3 mos (St.Joe's Hosp) 1 yr (Private)	1 yr 3 mos	Plateaued
5	1 yr 9 mos	PT,OT	arm & leg	8 mos (VA Hosp) 6 mos (Nsg Home) 2-1/2 mos (Courage Center)	16-1/2 mos	Plateaued; therapy of arm began in last 2 wks of 8 mo period up until final 2-1/2 mos at Courage Ctr; in final therapy, evaluated as plateaued
6	1 yr 6 mos	OT,PT,CT	arm & leg	1 mo (Ramsey Hosp) 3 mos (VA Hosp)	4 mos	Ongoing therapy discontinued for patient to participate in experiment

- CONTINUED ON NEXT PAGE -

Table 6(Continued)

Patient	Time Elapsed Since Last Stroke	Prior Therapies[a]	Extremities Treated	Duration of Individual Therapies	Total Prior Therapy Time	Pre-Experimental Status[b]
7	1 yr	PT,CT,OT	arm & leg	1 mo (VA Hosp) after first stroke 2 mos (VA Hosp) after second stroke	1 mo & 2 mos	Plateaued; after 2nd stroke, therapy was on leg only. At that time patient left hospital using Lofstrand cane for ambulation. Pre-experimentally, he could not walk with a cane unless assisted
8	1 yr 6 mos	None	---	---	---	---
9	2 yr 6 mos	PT,OT	arm & leg	2 mos (MMC Hosp)	2 mos	Plateaued
10	10 years	PT	arm & leg	4 mos (Nsg Home) 1 yr (VA) simultaneous with 10 yrs (Courage Ctr)	10 yrs; (from 2-1/2 mos after stroke until experiment	Plateaued; but, to keep active, continued therapy for gait at Courage Center

[a]PT indicates physical therapy; OT, occupational therapy; and CT corrective therapy.

[b]"Plateaued" indicates therapist or medical doctor appraisal of patient progress.

Measures taken during the Command situation (Part A) were:

A. Averaged electromyographic (EMG) response--the peak value of the averaged EMG of each response.

B. Range of angular motion--the difference of angle of flexion minus angle of extension which is equivalent to the total number of degrees of movement.

C. Latency of movement--time to respond to a command to move; measured from a mark on a paper recorder made by E's pressing a button simultaneously with her command to move, to the beginning of a rise in the EMG.

Measures during the Rate situation (Part B) were the EMG and range measurements as above, without latency, as well as:

D. Repetition of Movement--the number of times the patient moved his wrist or elbow during one minute.

Unique Measures:

E. Lowest EMG activity--for the patient who could not relax her wrist extensors (Patient 3, a patient with dystonic posturing in the muscles of the forearm and hand), a unique measure was obtained because of its significance. This was the "lowest level of EMG activity" achieved when the patient relaxed the wrist and consciously allowed it to fall into flexion.

In addition to (E), there was a qualitative change in Phase II for the same patient. This was wrist extension with finger extension. In all patient cases of Phase I training for wrist extension, the wrist was raised with fingers flexed. By Phase II, the above patient had a relaxed extensor EMG level over all four tests of Phase I and a range of motion by the end of Phase I equivalent to that of her contralateral

wrist during baseline. Thus, in Phase II, she began training on a qualitatively more difficult movement--raising the wrist with fingers extended. Dependent measures were identical for this movement. Because of this qualitative change in Phase II for Patient 3, she was not analyzed in the group statistics.

Equipment and Apparatus

A Medic Flexline EMG machine was used which recorded a raw EMG signal on a small oscilloscope. An averaged signal was deemed simpler and more appropriate for patient viewing (to be used as part of BF training). Thus, the EMG machine was connected to a signal averager built in our labs which fed to an American Optical monitor oscilloscope screen, Model 10765, to project an averaged EMG signal display to the patient. The scope had its own gain settings to magnify the signal above the setting on the EMG machine thereby allowing more subtle manipulations of signal size. The EMG signal was seen as a dot (leaving a trace) which rose and fell with contraction and relaxation of the target muscles. The EMG machine contained a loudspeaker which provided the auditory signal for BF training.

A Honeywell 2106 Visicorder Ultraviolet paper recorder unit was used to record responses. There were three channels: 1) an Event marker--activated by E pressing a button specifically with commands to move; 2) an EMG (averaged signal) channel; and 3) a goniometer channel to record angular movement.

Surface electrodes were used to record the EMG. Placements of the active electrodes were over the bellies of the wrist extensors or biceps. Ground and reference electrodes were placed over a nearby bony prominence and the tendons of the muscle, respectively. The skin was

marked with carfusin dye at each placement site to keep placement of electrodes consistent over sessions.

The goniometer consisted of a potentiometer connected to two hinged moveable metal strips. The apparatus was placed over the axis of the wrist on the ulnar side or the lateral epicondyle at the elbow. The limb surface above and below the joint was first covered with Dycem plastic cloth to insure against goniometer slippage.

During testing, both electrodes and the goniometer were applied to the patient; during BF training, only the electrodes were applied; during PT training the patient was free of any apparatus.

During BF training, as well as during testing under both BF and PT training, patients were seated in a slightly padded conventional office chair without arm rests. The chair was positioned on a platform to allow for greater height so that patients' arms could rest on an adjustable hospital bedside table which was wheeled next to the patient at a comfortable height. The table was positioned at a standard height and position for each patient and arm position on the table was held constant. The table was necessary for wrist extension target muscle movement only. Of the two patients who worked on the biceps muscle, one patient who required biceps relaxation (Patient 10) was seated in the chair alone where his upper extremity was free to relax, and the other patient who required biceps contraction (Patient 7) was seated in a wheelchair where his arm rested on the wheelchair seat. (The latter was the only patient who sat in a wheelchair.) During PT training the patients sat in the chair or wheelchair as above but movement was, of course, not confined to any standardized limb position on the table.

Procedure for PT

Patients were seated and physical therapy using range of motion, facilitation, resistance, inhibition, etc., was practiced for 30 minutes. Responses were shaped in that patients received immediate positive reinforcement, for example, "good", for any approximations to improved movement. Therapy for the muscle or muscle group in question required focus of attention on antagonist and other muscles of the forearm and arm which contributed to impaired motor ability, as well as target muscles. Impressed malfunctional stereotyped patterns had to be broken in order to establish new repertoires. Also, patients were frequently instructed to refer to the practiced movement in the unaffected limb to get the feel for what the correct movement entailed. At times, in the beginning of PT, patients did movements with both arms at the same time, having the unaffected limb move only as far as the affected one could, aiming for synchrony of the affected limb with the unaffected one. Some patients were told to look in a mirror to eliminate shoulder elevation, internal rotation, or abduction during elbow flexion, etc. Training was highly specific and goals were to shape and trim an appropriate, correct response, adding and subtracting any undesired features which made movements less "pure", isolated and exact.

Patients' limbs were moved passively at the shoulder, elbow, and wrist, and they were told to pay attention to the feel of it: to feel where it was moving and which muscles were pulling. There was enhancement of sensation at appropriate muscles and tendons by tactile stimulation concomitant with saying "Here and here and here--do you feel it?", and telling the patients "Now pay attention to . . . ", etc. Movements which could not be initiated by patients, or which had minimal excursion

or strength, were assisted by E moving the joints passively through the range (passive ROM) with the above instructions as well as instructions to "hold" at a position the patients were well able to maintain; in addition, patients were then told to actively relax the agonist ("Don't let it fall, bring it back down with control"),--i.e., to maintain a voluntary motion of the agonist muscle as much as possible. This procedure was also followed to provide guidance for the correction of incorrect movements by interspersing such passive movements with patient-initiated attempts, progressively reinforcing for successively correct approximations. Where non-voluntary antagonist contraction interfered with a desired motion, there was training of voluntary relaxation of the antagonist using the same principles of proprioceptive feedback, etc. as were used for activation of a muscle, but attention was directed to relaxation of the antagonist.

When patients did initiate movement, as in wrist extension, for example, to the command "raise your wrist", they were told "That's it, good", etc., "hold", if, and as they could, "bring it back down", and "let it go". Since most patients could move target muscles somewhat, attempts were also made at greater angular excursions and/or longer holds as feasible. For movements which could not be initiated, or which were initiated poorly, it was stressed that "Even a little bit, this much, is great", demonstrated by E on herself for example, barely extending the wrist (and, as necessary, e.g., without wrist supination or without shoulder retraction, elevation, etc.), demonstrated again on the patient himself, or for example, gestured symbolically by E motioning "just a little bit" with her fingers. For all movements, it was continually stressed "Remember correct movement is better than

any old kind of motion, etc." The latter stress about correctness of movement was a common constant theme to shape pure, isolated movements.

There were three parts to any movement, as feasible. They were instructed as, for example: 1) "raise the wrist", 2) "bring it back down", and 3) "relax it" or "let it go". Concerning (3): most of the time when patients were able to carry out a movement, they were able to carry it out actively contracting the agonist, but very often they would develop a contraction of agonist and antagonist muscles which continued for a long period or they would not completely relax the agonist at movement termination. In order to get good target muscle control, it was necessary to give specific instructions to relax or discontinue contraction. In other words, bidirectional ability to contract concentrically and eccentrically--to energize and inhibit--was always stressed so that if the patient were extending the wrist, it was equally stressed to bring the hand down voluntarily, instead of letting it fall, as possible, and then relax, that is, "let go" when the whole movement was completed. E repeatedly served as an example of correct movement and had the patients try movements appropriately on unaffected limbs. Demonstrations from E of what the specifics were in a particular movement were done both by facing the patient and moving, as well as by placing the limb in an orientation similar to the patient's and moving with instructions of "See, now you try it". E would also point and touch herself and patients on the places muscles were moving and the joints around which movements were taking place. If there were things patients were doing incorrectly, E would call attention to the difference between the incorrect movement and the correct one, pointing out salient features both on herself and the patients.

E also touched the skin over the muscles concerned a great deal to determine muscle tightness or looseness during movements to better ascertain how to shape, or what to inhibit, what to increase, etc. For inhibition of excessive tone there was a great deal of explanation and touching. For example, for tight wrist flexors during wrist extension, E would touch and say "Do you feel how tight this is?", etc. Then, for example, she would say "You have to stop making both these (wrist flexors and extensors) muscles tight at the same time if you are to raise the wrist better". E even sometimes explained in very simple terms how muscles on one side did one thing and those on the other the opposite, so that simultaneous excitation would result in, for example, a wrist that would not raise all the way. Tactile stimulation helped patients feel and attend to excessive tone. The moment tone diminished in the problem muscle E said "That's it, right", etc.

For the flaccid cases in particular, (two patients in this study), facilitation was used which consisted of skin friction or percussion of the muscle to facilitate and reinforce as movement occurred. In other cases of patients with increased tone, facilitation was used only as necessary and beneficial. Most patients did not need to have the target response elicited as they could execute it to some degree, and facilitation was used only as a springboard for a more firmly founded repertoire of voluntary control. Antagonist muscles of the target group, and, on occasion, other muscles were also facilitated. Movements in muscles other than the target agonist or antagonist were usually initiated by passive ROM as described above.

Resistance was a good part of PT. It was used mostly on the target muscle group but sometimes on antagonists of that group and occasionally on other muscles. Resistance was never applied to a

point where the patients did not feel successful pushing against E. Patients derived great satisfaction and immediate, physically apparent feedback from the sensation of resistance. (On the other hand, for example, while relaxation [inhibition] training is of equal importance, it is difficult to accomplish, and difficult for patients themselves to experience comparable sensory feedback for relaxation in PT.) E made certain patients knew how to practice resistance training themselves for purposes of work at home.

For many patients there was general relaxation training of the paretic upper extremity. This involved instructing the patients to go to the opposite, unaffected limb, tense all the muscles "tight, tight, tight, tight", and then "let go", illustrated by E all the while. Patients were then asked to do the same. E then said

> Do you feel the relaxation? Now it is the same feeling you want in your other arm because now it is all tight. (They would know it and agree.) So when you go home to practice, do this (again, show me how you do it) and then try to get the same relaxation feeling in the affected (right or left arm). Also practice (demonstrated) relaxing in both limbs starting with the good one, feeling heavy, loose, loose as a goose, like spaghetti, first your neck muscles, then your upper arm muscles, through your elbow, into your lower arm, through your wrist, into your hand, down to your finger tips.

This was said in a monotonous, hypnotic tone and E was simultaneously getting loose and heavy too. Another relaxation method was instructed as follows: "Try letting your arm go as a dead weight". This was tried on the affected and unaffected limb singly and together. To demonstrate, E would say "Okay, feel my arm and move it", "there is nothing coming from me", etc. E's limb remained passive and the only way it would move was if the patient pushed it, after which it would fall.

As patients learned relaxation, those with excessive tone received emphasis to execute movements with the arm first relaxed to facilitate

appropriate, smooth, correct motion. The technique was used as part of the total plan of giving resistance, stressing relaxation, etc., keeping target muscles improving without losing achievements to date or disturbing coordinated smooth movement.

All methods used were used to the extent and times at which the patient could feel successful. In other words, E was careful not to ask more of the patient than he could do, yet much was required in terms of work and attention to subtle, gradual changes. PT was quite dynamic in terms of what was desired and how hard patients were to try. There was constant verbal feedback, touch, stimulation, explanation, example, corrections, praise, and direction of attention to immediate particulars. Constant coaching, in other words, in every way, shape, or form. Also, there was endless repetition of important particulars to be sure that patients understood E and that E understood the patients. Patients were repeatedly asked to try on their own, movements in practice, to verify that they were going about the movement correctly. When patients would first arrive for a session, E would say "Okay, show me what you have been doing", or, "what we have been practicing", if they did not do homework, to see how well they remembered, executed, or even if they understood the movement pattern in the first place. The patients, who were an alert and aware group, needed a lot of repetition--not only because they were impaired, but also because motor control, particularly of the precise variety demanded, is an extremely difficult feat.

Procedure for BF

For the first session, electrodes were connected to the target muscles on both arms. The patients were first told to try movement on the

unaffected side to experience the sound and TV display corresponding to movement. Instructions were as follows (using the wrist as an example):

> We are going to begin by showing you how this machine works. First, we will look at your right (or left) arm to see what happens. Please move your wrist up and down like this. (Demonstrated) Now go ahead and move it slowly, etc. and watch how the dot on the screen goes up and down and listen to how the noise gets louder and softer. Move your wrist some more, paying attention to the dot going up and down and the noise getting louder and softer. The dot goes up and down depending on what you are doing with your wrist. So does the sound get louder and softer depending on what you are doing with your wrist. When your wrist is relaxed the dot is low. When the dot moves higher, it is because your wrist is moving more. You can make it high or low with your movements. Also listen to the noise. When your wrist is relaxed the noise is quiet. When the noise gets louder it is because your wrist is moving more. You can make it go quiet or loud with your movement.
>
> Now from here we're going to try it with your other hand to see what we can do. Note the difference in the dot and sound for this arm compared with your other side. Pay attention to the dot and try to make it go higher (or lower). Also you will make the noise go louder (or softer). That's the whole point. The higher (or lower) you can make the dot go and the louder (or softer) you can make the noise, the better you are doing. You must put your mind to it to try to make the dot high (or low) or it will not move. The noise will also get louder (or softer) as you try to move (or relax). The way you get the dot and sound to work is by raising your wrist (or relaxing your wrist), that is, bringing your wrist up as much as you can (or letting your wrist go as completely as possible).

After several patient attempts following the above instructions, additional instructions were given. For the eight patients requiring greater target muscle contraction:

> Now, although we are most concerned with your getting the dot to go higher and the noise to get louder, you should always bring your wrist (or elbow) down to the point where the dot is just a smooth, flat line and the noise is absolutely quiet. Do not start another movement until you reach this quiet, relaxed point. This is because we want to make the muscle go well in both directions--first to make it work and then to shut it off or relax it.

For the patient who required wrist extensor relaxation (Patient 3), directions were as above with reversed emphasis. For the patient who required biceps relaxation (Patient 10), bidirectional control was not stressed at first as the patient was extremely spastic and had virtually

no range of motion around a static flexed elbow position. Thus any and all relaxation was emphasized. About two weeks into BF training, when relaxation was apparent, bidirectionality was instructed.

Patients readily understood how the stimuli corresponded to contraction and relaxation. After one session of limb comparison and acquaintance with the apparatus, only the affected limb was connected and the patients were left alone to train for 30 minutes with occasional visits from E. The visits were to monitor performance and ascertain that movements were being executed as correctly as possible. The necessity and content of such visits is elaborated in patient case histories (Section XIV). The training room was daylight dim (shade drawn) and the door was kept slightly ajar so that E could view both the patient and the TV during practice.

Procedure Common to BF and PT

Patients were given a great deal of verbal guidance in both BF and PT. That is, e.g., if a patient were elevating the shoulder to contract the biceps or flexing the elbow to extend the wrist, he was continually reminded that the movements in question were not being executed or not being executed appropriately--that, in fact, many movements were occurring at once, movements were incorrect, or that movements incorporated features which were not part of the training. Thus, there was a great amount of verbal guidance to isolate a single correct response, necessarily a more intricate part of PT training than BF due to the constant mutual efforts of both the patient and E during PT rather than the intermittent visits from E during BF. Also, E made generalized appraisals of patients' performances after each session of both BF and PT, noting how the patient was doing, adding careful statements of encourage-

ment as necessary or warranted. Exhortation to greater effort was subdued in order to prevent patients from meeting with too much failure should they not succeed. Patients were highly motivated to work. There were few absenteeisms and subjective reports of a good deal of practice at home from most patients (Amount of practice tabulated in Table 4).

Concentration was also a common theme stressed to both groups by E. Frequent statements were made to the effect to "concentrate now", "you must concentrate", and "put your mind to it."

SECTION XIV

RESULTS

Statistical Results

First, consideration will be given statistical results. A diagram representing the experimental design, as well as possible statistical comparisons derivable from such a design, is presented in Table 7. The major dependent variables analyzed statistically were averaged EMG activity, range of motion, and latency. The repetition of movement variable is given descriptive rather than statistical treatment.

An exhaustive description of all comparisons is not presented. Many comparisons, especially intergroup, were insignificant due to the limited degrees of freedom (e.g., 6 df for intergroup comparisons). Hence, statistically insignificant comparisons are recorded only as they are logically of critical importance or of supplementary interest to group performance analyses.

Related independent variables of sensory function and neuropsychological assessments obtained at experimental outset, have yet to be submitted to analyses to determine their possible correlation with patient performance on dependent variables. It was not possible to include these elements in the current study.

Patient 3 was omitted from all statistical analyses. The primary reason for this omission is the fact that a qualitatively different response feature was incorporated into Patient 3's response requirements for Phase II (for details see "Measures", pp. 115-116 and the case history, pp. 229-247).

Table 7

Experimental Design and Lay Out (Current Page) Incorporating Illustrations of Possible Statistical Comparisons (Current and Next Page) for all Dependent Variables

Inter and Intragroup Comparisons											
Part A (Command)							Part B (Rate)				
		Treatment						Treatment			
		Baseline	Phase I		Phase II			Baseline	Phase I		Phase II	
Group			BF	PT	BF	PT	Group		BF	PT	BF	PT
6, 7, 8, 9, 10 Subjects	Group 1 (BF then PT)	✓	✓			✓	6, 7, 8, 9, 10 Subjects Group 2 (BF then PT)	✓	✓			✓
1, 2, 3, 4, 5 Subjects	Group 2 (PT then BF)	✓		✓	✓		1, 2, 3, 4, 5 Subjects Group 2 (PT then BF)	✓		✓	✓	

- CONTINUED ON NEXT PAGE -

Table 7(Continued)

Pooled (Groups 1 and 2) Comparisons

Part A (Command)				Part B (Rate)			
	Treatment				Treatment		
Group	Baseline	Phase I & II BF	Phase I & II PT	Group	Baseline	Phase I & II BF	Phase I & II PT
Subjects 1 2 3 4 5 6 7 8 9 10 — Pooled Groups 1 and 2	✓	✓	✓	Subjects 1 2 3 4 5 6 7 8 9 10 — Pooled Groups 1 and 2	✓	✓	✓

As previously noted, the purpose of this study, was to assess BF training. However, a concurrent control was necessary, and for this purpose, a Cross Over design was employed with PT as a control treatment. There were, thus, two groups, 1 and 2, which got two Phases, I and II, of training in reversed order. Group 1 received BF, Phase I and then PT, Phase II. Group 2 received PT, Phase I and then BF, Phase II.

Test sessions consisted of two parts always in the same order. The first part, referred to as Part A, was a Command situation where a patient responded to five commands to, e.g., "raise the wrist" and "let it go". The second part, referred to as Part B, was a Rate situation where the patient was instructed to respond on his own as many times as possible as well as correctly as possible over a one-minute period.

It was expected that response characteristics--i.e., averaged EMG activity and range of motion--might differ in either situation as follows: During the former situation, external stimuli to movement were controlled by E and the patient may have had to have been more attendant to literal response characteristics or specifics; in the latter situation, stimuli to each movement were not controlled by E and the patient may have performed with a greater degree of "automaticity", attending to cues of moving faster and well, somewhat abstracted from attending to response specifics. Thus, patients might presumably have exhibited less EMG activity or range of motion in overall response during Rate.

As it happened, training was quite focused on accuracy of movement; and, instructions in Rate highlighted accuracy as well as rate.

Hence, relative to baseline, patients moved slowly, within the confines of response accuracy during Rate. It is surmised that, in the end, the training time course was too brief for Rate to approximate a paradigm in which responses were being executed "automatically". The results of Part A (Command) and Part B (Rate) are presented together in Statistical Results.

Averaged EMG Activity[30]--using mean values in microvolts (uv)

Pooled Group Comparisons: (see Table 7 and Figures 1 and 2)

A pooled group analysis of BF Part A ($\bar{x}$=69.1) and BF Part B ($\bar{x}$=69.2) over baseline Part A ($\bar{x}$=42.5) and baseline Part B ($\bar{x}$=43.2) showed statistically significant (correlated t) differences ($\bar{x}$=26.5 and $\bar{x}$=26.0, respectively) with associated p values of .01 and .009. To obviate the problems connected with studying the effects of BF training alone, a concurrent control of PT was incorporated into the experimental design. A pooled group analysis of PT Part A ($\bar{x}$=60.4) and PT Part B ($\bar{x}$=58.6) over baseline Part A ($\bar{x}$=42.5) and baseline Part B ($\bar{x}$=43.2) also showed statistically significant differences ($\bar{x}$=17.9 and $\bar{x}$=15.3, respectively) with associated p values of .04 and .03. The differences over baseline for Parts A and B showed consis-

[30]Patient 10 was excluded from the EMG analysis. This is because when either the affected or unaffected limb was in a position requiring a relaxed biceps muscle (i.e., a position of an extended elbow), there was a certain amount of EMG activity in both limbs during baseline and even an increased amount of EMG activity in the unaffected limb during Rate at baseline. Furthermore, the decrease in averaged EMG activity for the affected limb in a position requiring a relaxed biceps was minimal during both BF and PT training. In light of Patient 10's case history (see pp. 346-363), the constant residual EMG activity in both limbs under conditions of elbow extension seems most likely due to a psychological "non-relaxed" attitudinal bias. As the history reports, Patient 10 exhibited behavioral characteristics antithetical to absolute relaxation, which, for current purposes, would be manifest as utterly limber elbow joints representative of completely relaxed biceps muscles. An attitudinal bias seems a more likely rationale for the residual activity evidenced in both limbs under the condition requiring biceps relaxation than, e.g., bilateral brain damage.

Table 8

Pooled Group Comparisons for AVERAGED EMG ACTIVITY

(Computed using mean values [in microvolts] of averaged EMG activity)

	Part A					Part B				
	Δ	SE	df	t	P	Δ	SE	df	t	P
Pooled Group Comparisons (Groups 1 and 2)						Pooled Group Comparisons (Groups 1 and 2)				
BF vs PT						BF vs PT				
$\bar{x}$=69.1 $\bar{x}$=60.4	8.6	7.6	7	1.13	.295	$\bar{x}$=69.2 $\bar{x}$=58.6	10.7	5.6	7	1.91	.098
BF vs Baseline						BF vs Baseline				
$\bar{x}$=69.1 $\bar{x}$=42.5	26.5	7.8	7	3.41	.01	$\bar{x}$=69.2 $\bar{x}$=43.2	26.0	7.3	7	7.3	.009
PT vs Baseline						PT vs Baseline				
$\bar{x}$=60.4 $\bar{x}$=42.5	17.9	7.1	7	2.5	.04	$\bar{x}$=58.6 $\bar{x}$=43.2	15.3	5.9	7	2.61	.03
Overall Treatment (Phases I & II) vs Baseline						Overall Treatment (Phases I & II) vs Baseline				
$\bar{x}$=64.8 $\bar{x}$=42.5	22.3	6.4	7	3.46	.01	$\bar{x}$=63.9 $\bar{x}$=43.2	20.8	6.0	7	3.47	.01

Figure 1

Averaged EMG Activity in Microvolts (SE indicated)

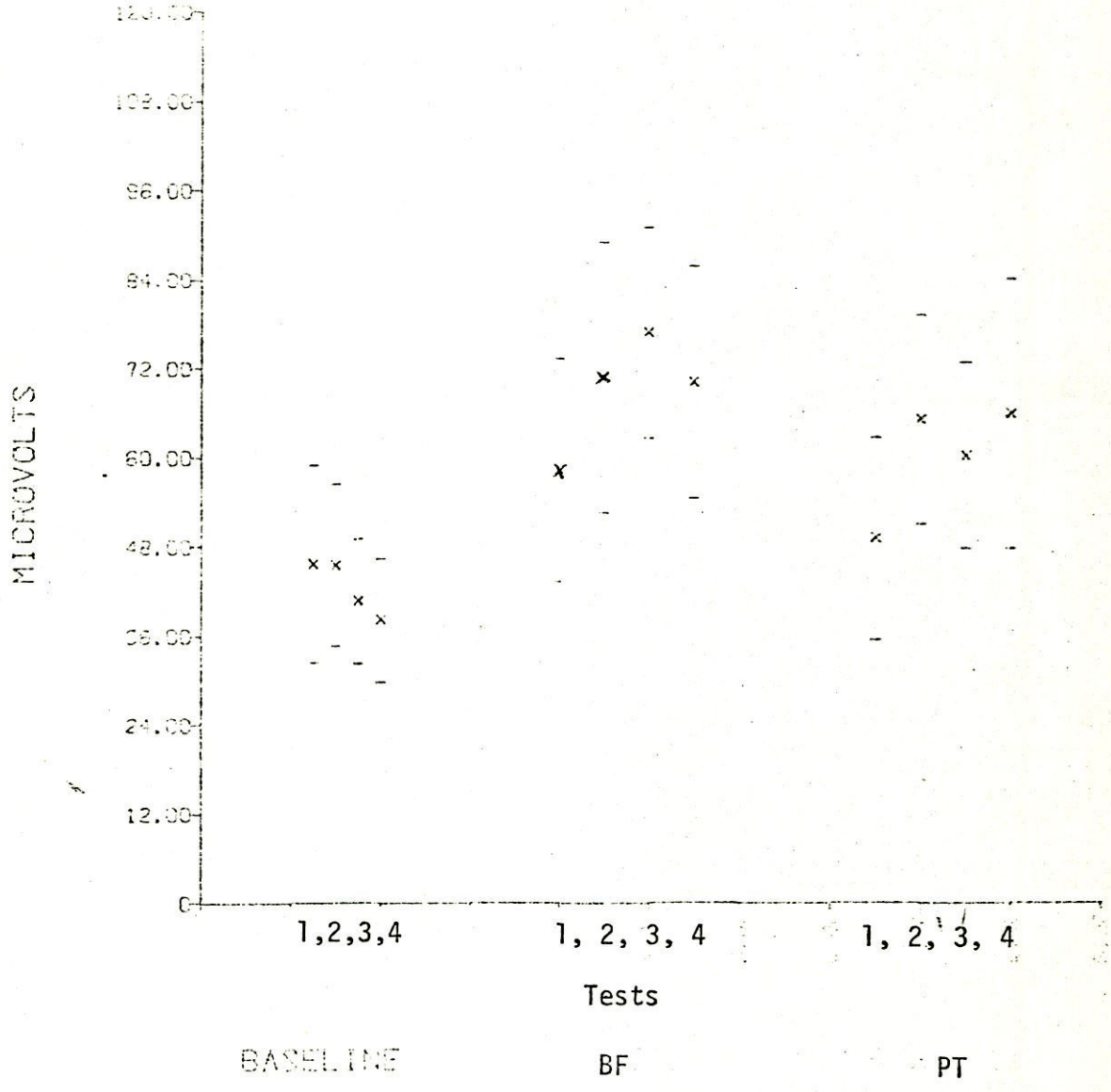
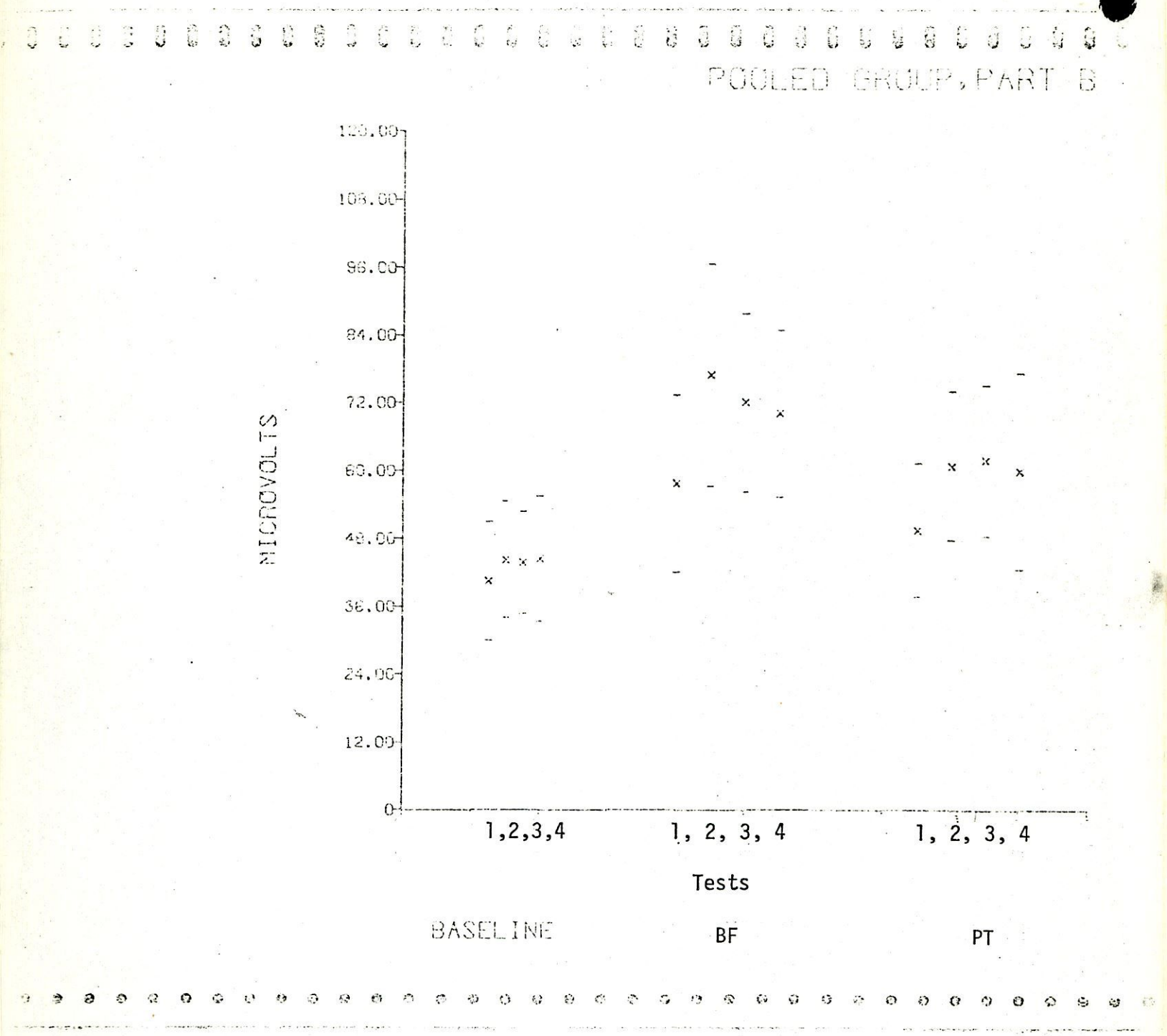


Figure 2

Averaged EMG Activity in Microvolts (SE indicated)



tently greater gains in EMG activity for pooled BF training relative to the pooled, control group, PT training. For example, there was an average net gain of 17.9 μv in Part A and 15.3 μv in Part B for PT vs. an average net gain of 26.5 μv in Part A and 26.0 μv in Part B for BF.

A further pooled group comparison between BF and PT training Part A (mean difference=8.6) and BF and PT training Part B (mean difference= 10.7) supplemented the effect of higher net gains over baseline for BF training. A suggestive trend for this comparison in the direction of greater increments of EMG activity under BF training was indicated by p values of .295 and .098 for Parts A and B, respectively. Thus, there were suggestive trends that BF was more effectual in producing EMG increments than PT. But, as is indicated below, BF was more operative in precisely conditioning such activity than was PT.

Qualifications Pertaining to the Above Results

Although BF did significantly increase that which it specifically conditioned, i.e., EMG activity, the results of BF superiority over PT for EMG conditioning, belie a comparison of BF vs. PT. This is because the procedural emphasis of PT training for three of the four patients in Group 1 was general limb and target muscle relaxation training. The latter specifically shapes inhibition of the intensity of muscle contraction in favor of (a) eliminating detrimental antagonistic or synergistic muscle contraction patterns and (b) obtaining the most proficient and efficient target movement. Thus, while BF was conditioning EMG increases for all analyzable patients, PT was conditioning, as a consequence of procedure, EMG decreases or steady patterns of contraction, particularly for Patients 6, 8, and 9. The latter patients were all in Group 1 and all exhibited a considerable

degree of spasticity. (For complications involved in Group 1 vs. Group 2 comparisons see "Intragroup Comparisons" pp. 140-148.)

Unlike BF then, PT assessment is constrained by the sensitivity and even the tractability of the EMG variable to PT training. Hence, what actually only can be concluded is that BF, relative to baseline, and relative to some control, i.e., PT, did increase that which it conditioned, namely, EMG activity. Interpretation of any foregoing comparisons between BF and PT for averaged EMG activity is confined similarly as above. In addition, although not germane here, there are other problems involved in comparisons between BF and PT which will be discussed fully in the next section (Discussion).

A slope analysis of pooled BF vs. pooled PT, Parts A and B, showed no significant differences. Finally, a comparison of the pooled overall treatment Part A ($\bar{x}$=64.8) and Part B ($\bar{x}$=63.9) over baseline Parts A and B was statistically significant for both Parts A and B (mean differences=22.3 and 20.8, respectively) at the .01 and .01 levels.

As previously noted, Part A and B comparisons (i.e., Command vs. Rate) did not statistically represent different--i.e., quantitatively different--learning situations. Clinical observations made by E of learning under Part A and Part B would also seem to suggest that Parts A and B entailed similar learning and performance in general, with the rather trivial exceptions of differences at baseline performance for the range of motion variable (discussed further on) for three patients (for further details see the case histories for Patients 2, 6, and 9), and again for the range variable during training for one patient (for further details see the case history for Patient 2).

Intergroup Comparisons: (see Table 9)

There were two groups in this study. Group 1 received BF first and then PT training, while Group 2 received PT and then BF training. A critical intergroup comparison is the equality of Group 1 Part A ($\bar{x}$=36.2) and Part B ($\bar{x}$=37.2) vs. Group 2 Part A ($\bar{x}$=49.0) and Part B ($\bar{x}$=49.3) at baseline. Such a comparison was, as would be desirable, not statistically significant (p=.56 and .59, respectively). The standard error terms between the groups also did not differ at baseline. However, there was an approximate 12 point difference between the group means, Parts A and B, at baseline which may be clinically significant. Group 1 started at a lower baseline level and percentage-wise made overall greater gains. Furthermore, once Group 1 began initial training, which was BF, the standard error term for the group remained the same while that for Group 2, which began PT training first, doubled during both PT and BF training. This was due to the fact that, relative to each other, three of the four analyzable patients in Group 1 showed good gains (save in Phase II where one of the former individuals [Patient 6] suffered a distinct loss due to general relaxation training [overall group performance was also lower because of general relaxation training]), while the fourth individual of Group 2 showed relatively moderate gains in Phases I and II. In Group 2, on the other hand, when patients were compared against each other, there was one out of four analyzable individuals who showed great gains, one individual who showed good gains, one individual who showed relatively moderate gains (in terms of EMG only--[for current purposes of exposition]), and one "non-learner" (Patient 5) who made no gains whatsoever. Hence there was a double standard error term for Group 2 in Phases I and II

Table 9

Intergroup Comparisons for AVERAGED EMG ACTIVITY

(Computed using mean values [in microvolts] of averaged EMG activity)

	Part A					Part B				
	Δ	SE	df	t	P	Δ	SE	df	t	P
Intergroup Comparisons (Group 1 vs Group 2)										
Baseline 1 vs Baseline 2										
$\bar{x}$=36.2 $\bar{x}$=49.0	12.8	20.6	6	.61	.56					
Intergroup Comparisons (Group 1 vs Group 2)										
Baseline 1 vs Baseline 2										
$\bar{x}$=37.2 $\bar{x}$=49.3						12.1	21.2	6	.56	.59

which perhaps represents "inequalities" in learning potential in terms of information extracted as averaged EMG activity in conjunction with the interactive effects of the latter parameter with different training modalities and different patient problems.

In light of the above especially, direct comparisons between Groups 1 and 2 are to be viewed with circumspection in terms of external validity. Of course, for inferences to external validity, overall results are subject to the constraints of a small n, the circumstances under which patients were available for study, patient characteristics necessary for inclusion in the study, and other extraneous but important patient characteristics, etc. These considerations are covered in Section XIII and Case Histories and some will be revisited in the Discussion section. For purposes of internal validity, group differences at baseline were not statistically significant for any dependent variables. At the same time, however, it is true that chances of equal groups are less than optimal with a total n of four in each group. In the final analysis of the data obtained one might search for group patient differences in terms of their possible influence on group performances.

Ancillary Aspects of the Data: a Post Hoc Look at Patient Characteristic Differences

It is to be noted that the muscle tone of the analyzable individuals assigned to Group 1 contained three individuals with increased tone and one with flaccidity; and all four individuals analyzed in Group 1 had decreased, or poor position sense. The muscle tone of analyzable individuals in Group 2 contained one individual with slightly increased tone, one individual with practically normal tone, one with

slight flaccidity, and one with extreme flaccidity; and position sense was assessed as intact for all of the four individuals analyzed in Group 2. Three of the four patients in Group 1 had relatively considerable spasticity and one had minimal spasticity while two of the patients in Group 2 had relatively moderate spasticity and two had mild spasticity. Also, generally speaking, poststroke duration was shorter for Group 1 than for Group 2. Finally, on a scale of 5, muscle grade strength was 2 for three individuals, and 3 for one individual in Group 1; whereas it was 1 and 1-2 for two individuals, 2 for one individual, and 3-4 for one individual in Group 2. The latter two individuals of Group 2 were those who showed the greatest overall gains for EMG recruitment in Group 2. Although Groups 1 and 2 were not statistically significantly different at baseline on any of the dependent quantitative parameters of averaged EMG activity, range of motion, or latency (see pp. 156 and 171 for baseline comparisons of range of motion and latency), in retrospect, matching on some of the aforementioned patient characteristics may have provided a more adequate design.

Intragroup Comparisons: (see Table 10 and Figures 3, 4, 5, and 6)

Group 1

Further insight into the above comparisons is obtained from intragroup comparisons similar to the above pooled group comparisons. Group 1 comparisons of BF Part A ($\bar{x}$=59.6) and Part B ($\bar{x}$=58.5) over baseline Part A ($\bar{x}$=36.2) and Part B ($\bar{x}$=37.2) (mean differences=23.4 and 21.3, respectively) were statistically significant with p values of .03 and .008. Group 1 comparisons of PT Part A ($\bar{x}$=57.3) and Part B ($\bar{x}$=55.4) over baseline Part A and Part B (mean differences=21.1 and

Table 10

Intragroup Comparisons for AVERAGED EMG ACTIVITY

(Computed using mean values [in microvolts] of averaged EMG activity)

	Part A					Part B				
	Δ	SE	df	t	P	Δ	SE	df	t	P
Intragroup Comparisons: Group 1 (BF then PT)										
BF vs PT										
$\bar{x}$=59.6 vs $\bar{x}$=57.3	2.3	6.4	3	.37	.74					
$\bar{x}$=58.5 vs $\bar{x}$=55.4						3.1	6.8	3	.46	.68
(Phase I) BF vs Baseline										
$\bar{x}$=59.6 vs $\bar{x}$=36.2	23.4	6.0	3	3.90	.03					
$\bar{x}$=58.5 vs $\bar{x}$=37.2						21.3	3.4	3	6.3	.008
(Phase II) PT vs Baseline										
$\bar{x}$=57.3 vs $\bar{x}$=36.2	21.1	9.0	3	2.35	.10					
$\bar{x}$=55.4 vs $\bar{x}$=37.2						18.2	7.8	3	2.35	.10
Intragroup Comparisons: Group 2 (PT then BF)										
BF vs PT										
$\bar{x}$=78.5 vs $\bar{x}$=63.6	14.9	14.3	3	1.04	.37					
$\bar{x}$=80.0 vs $\bar{x}$=61.7						18.3	7.9	3	2.31	.10
(Phase I) PT vs Baseline										
$\bar{x}$=63.6 vs $\bar{x}$=49.0	14.6	12.3	3	1.19	.31					
$\bar{x}$=61.7 vs $\bar{x}$=49.3						12.4	9.8	3	1.27	.29
(Phase II) BF vs Baseline										
$\bar{x}$=78.5 vs $\bar{x}$=49.0	29.6	15.5	3	1.91	.15					
$\bar{x}$=80.0 vs $\bar{x}$=49.3						30.7	14.8	3	2.07	.13

Figure 3

Averaged EMG Activity in Microvolts (SE indicated)

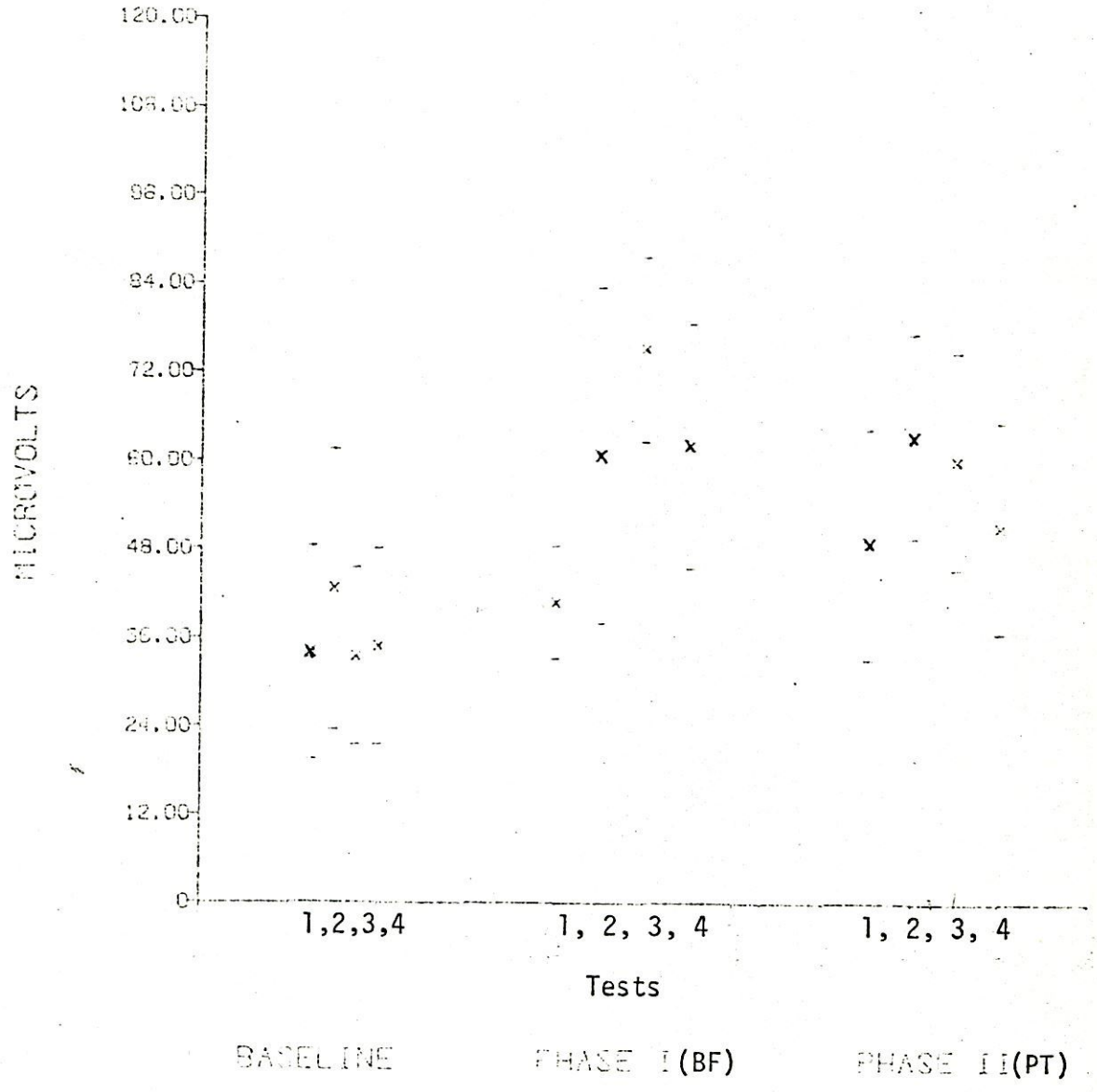


Figure 4

Averaged EMG Activity in Microvolts (SE indicated)

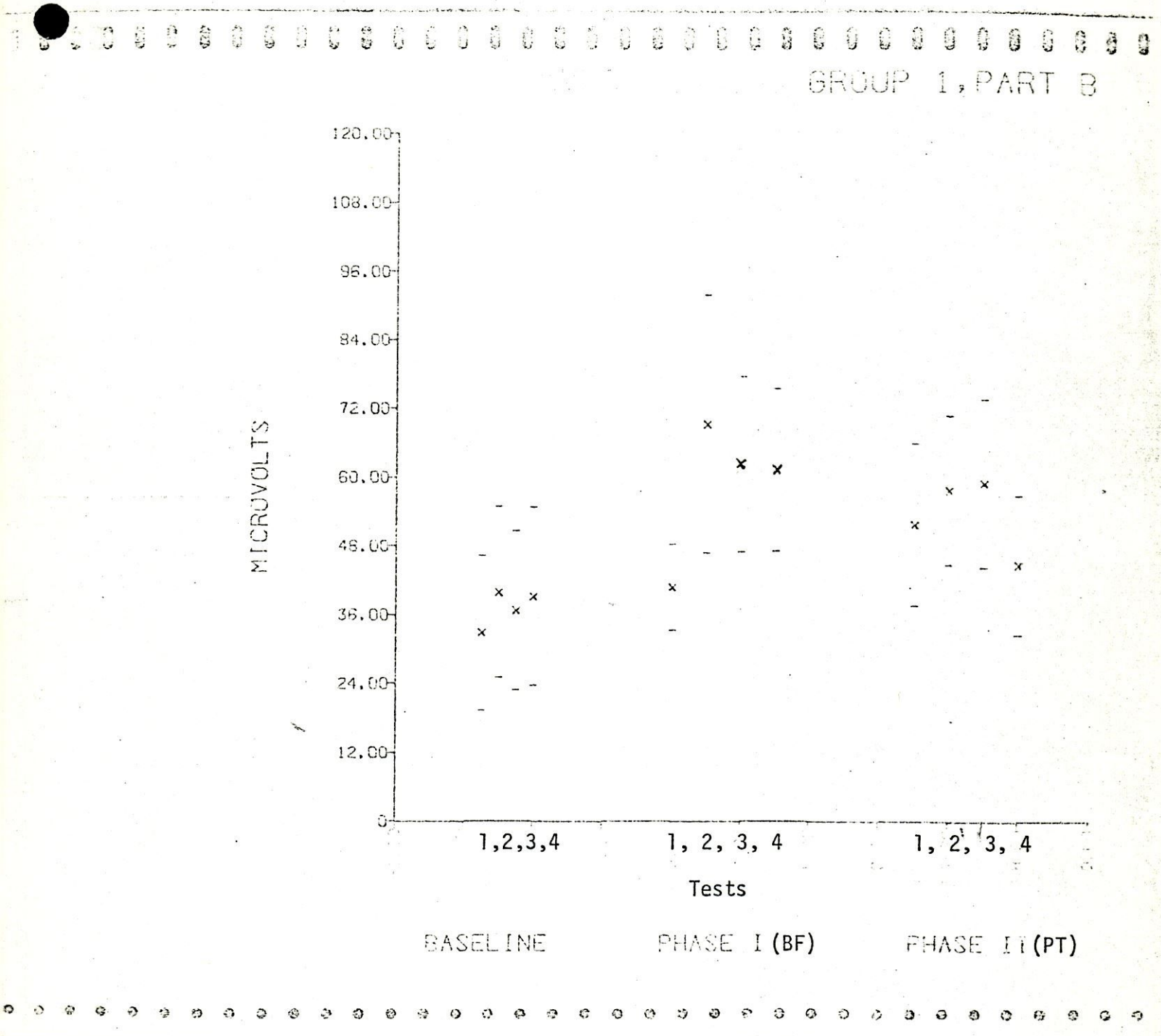


Figure 5

Averaged EMG Activity in Microvolts (SE indicated)

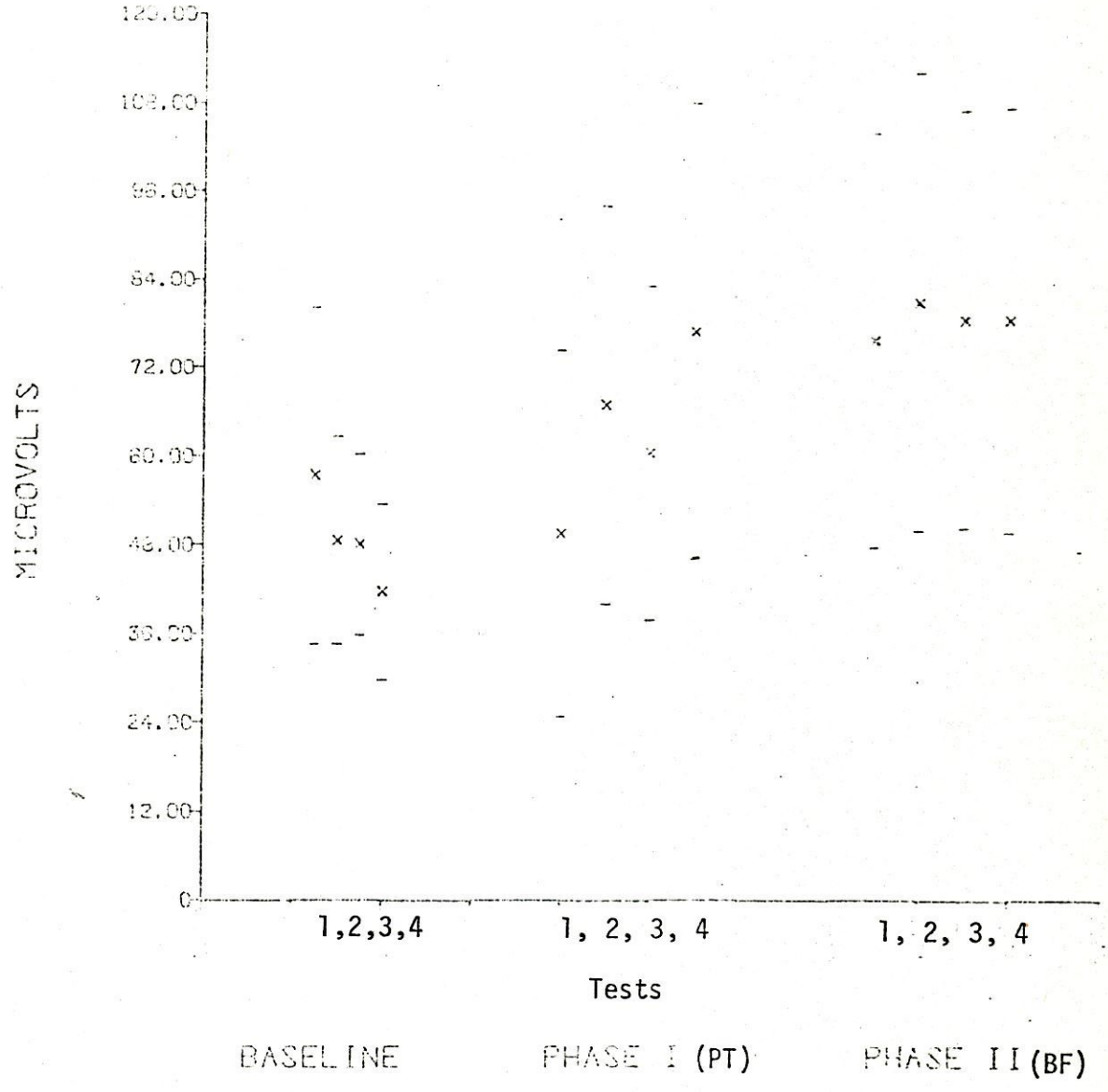
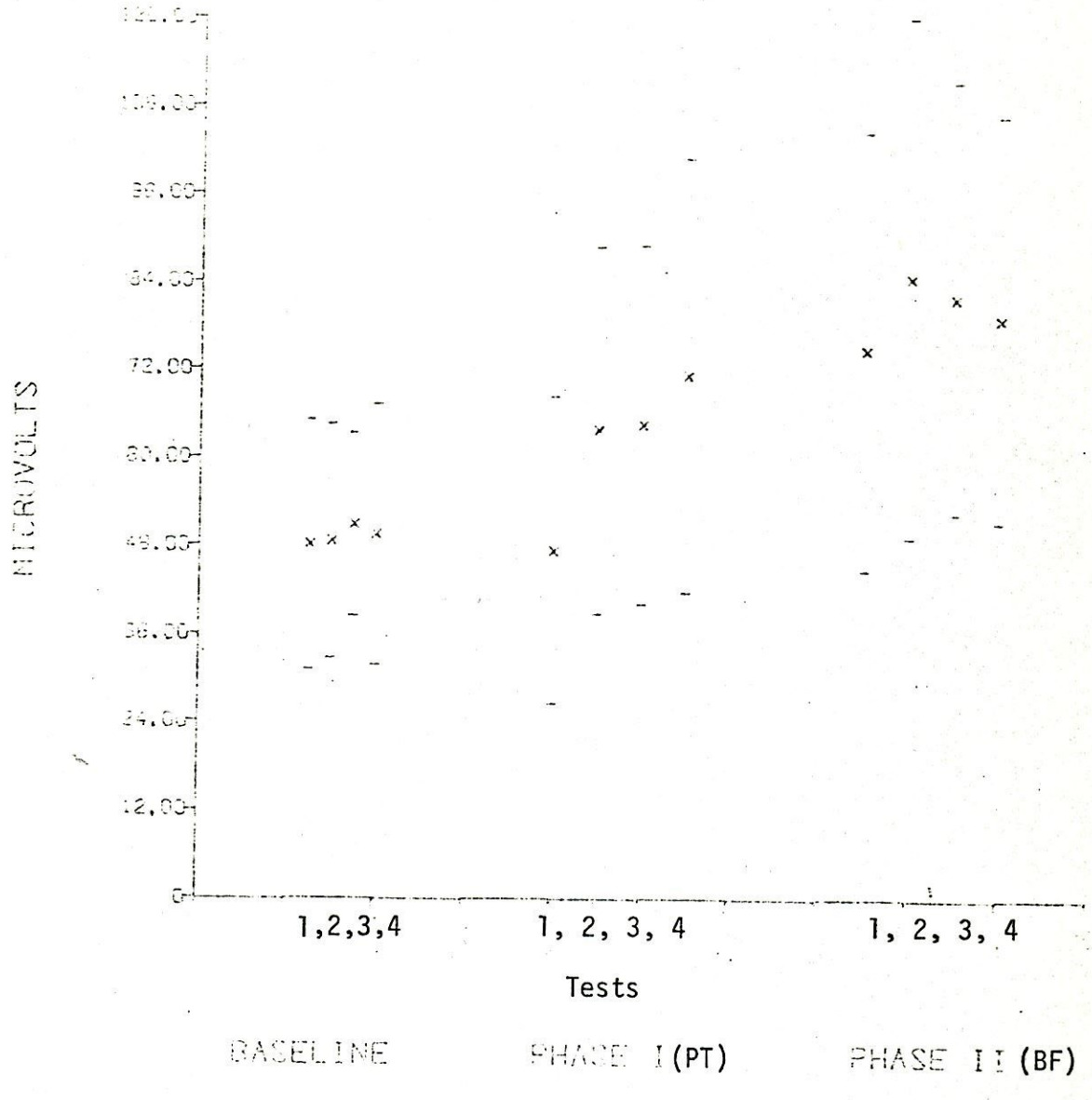


Figure 6

Averaged EMG Activity in Microvolts (SE indicated)



18.2, respectively) approached but did not attain significance with p values of .10 and .10, respectively. Group 1 intragroup comparisons of BF vs. PT, Parts A and B, (mean differences=2.33 and 3.1, respectively) were not significant with p values of .74 and .68.

Group 2

Group 2 BF Part A ($\bar{x}$=78.5) and Part B ($\bar{x}$=80.0) over baseline Part A ($\bar{x}$=49.0) and Part B ($\bar{x}$=49.3) (mean differences=29.6 and 30.7, respectively) suggested a trend toward greater EMG recruitment under BF training with p values of .15 and .13. A similar comparison of PT Part A ($\bar{x}$=63.6) and Part B ($\bar{x}$=61.7) over baseline (mean differences= 14.6 and 12.4, respectively) did not suggest such a trend with p values of .31 and .29. The large standard error term (discussed on pp. 137 and 139) for Group 2 rendered low t, and hence, p values well above conventional levels. Group 2 intragroup comparisons of BF vs. PT, Parts A and B, (mean differences=14.9 and 18.3, respectively) were not significant for Part A (p=.37) but approached significance in the direction of a greater effect under BF training for Part B with a p value of .10.

A comparison of BF vs. PT, for Group 2, e.g., looking at Part B for the following contrast, is also what seems to account for the trend toward significance in the pooled group comparison of BF vs. PT where p=.098. In actual numbers, the Group 1 (BF then PT) mean was 58.5 for BF, which was the first training for this group, and 55.4 for PT, which followed BF training for this group. For Group 2 (PT then BF) the mean was 61.7 for PT, which was the first training for this group, and 80.0 for BF, which followed PT training for this group. In summary, there was an initial 21.3 μv increment for Group 1 over baseline in BF training and an 18.3 μv difference over baseline (i.e.,

a slight decrease) for Group 1 in subsequent PT training. On the other hand, there was an initial 12.4 μv increment for Group 2 over baseline in PT training and a 30.7 μv difference (an additional 18.3 μv increment) for Group 2 in subsequent BF training. The overall pooled group increment favoring greater BF net gains (see "Pooled Group Comparisons") seems particularly bolstered by Group 2 gains in BF where BF was the second training regimen. That is, for Group 2 a PT gain of 12.4 μv over baseline was increased by an additional 18.3 μv over PT during BF training. Combining this result for Group 2 with a 21.3 μv net gain in Group 1 for BF over baseline with a no μv net gain (i.e., a 3.1 μv loss) for Group 1 in PT after BF, the suggestive trend where $p=.098$ in the pooled BF vs. PT comparison (in this example, for Part B), seems due to a greater difference for BF in both Groups 1 and 2, where the Group 2 contribution to the pooled group difference favoring BF is particularly strong, even though the intragroup comparison of BF over baseline for Group 2 merely approached significance ($p=.10$).

As explained earlier (see pp. 135-136) three patients in Group 1 (Patients 6, 8, and 9) received a great deal of relaxation training during PT, their second training regimen. The fourth patient, Patient 7, did not require such training. The group, thus, held steady, i.e., made no increments, on averaged EMG activity during PT after its initial mean increment (e.g., Part B) of 21.3 μv over baseline. Whereas three of the four patients in Group 1 were trained to inhibit excessive muscle contraction in the target and other limb muscles during PT, the opposite was the emphasis for the individuals in Group 2. That is, three patients in Group 2 received a good deal of resistance training

for the target muscle and no general relaxation training for the limb or specific relaxation for the target muscle within the total program of therapy. The fourth patient, Patient 5, received some resistance and no relaxation training.

In sum, while general relaxation training was critical for the patients in Group 1 during PT, the patients in Group 2 did not need any general relaxation training during PT. The data for PT, Groups 1 and 2, reflect this difference: Group 1, as a whole, making no gains in EMG during PT, and Group 2, as a whole, making good gains in EMG during PT.

Range of Motion[31,32]--in percent scores, using the value of attained range over maximum range possible--i.e., the range variable was calculated as range achieved relative to the maximum possible for a given patient.

Pooled Group Comparisons: (see Table 11 and Figures 7, 8, 9, and 10).

A pooled group analysis of BF Part A ($\bar{x}$=38.5) and BF Part B ($\bar{x}$=36.8) over baseline Part A ($\bar{x}$=27.3) and baseline Part B ($\bar{x}$=20.8)

---

[31]The maximum range a patient could achieve was subject to individual variation because of contractures (shortening of soft tissue around a joint) in some patients. In fact, less than maximum range boundaries due to contractures signified additional pathology and therefore the same absolute change in range in patients with contractures vs. patients without contractures represented more change for patients with contractures. Hence, the most suitable measure was that of attained range relative to the maximum range possible for a given patient. Maximum range possible was determined by passively stretching a joint and measuring the angles obtained.

[32]Patient 7 was excluded from the range analysis. This is because he had an apraxia (for details, see this patient's case history pp. 296-312). Briefly, the target movement for Patient 7 was elbow flexion which was accomplished by a gross swing of an adducted shoulder and included crossed extensor reflex activity. Although he had a limited capacity for movement in his paretic upper extremity, Patient 7 did not know how to accomplish any specific target motion as he did not know how to "put it together". There was no change in degree of angular motion in either BF or PT training and no true control over joint movement at baseline or in Phase I (BF) or Phase II (PT). In sum, elbow angulation was always accomplished by the aforementioned stereotyped pattern.

Table 11

Pooled Group Comparisons for RANGE OF MOTION

(Percentage scores--computed using mean values of the range attained relative to the maximum range possible for a given patient)

	Part A					Part B				
	Δ	SE	df	t	P	Δ	SE	df	t	P
Pooled Group Comparisons (Groups 1 & 2)										
BF vs PT										
$\bar{x}$=38.5 $\bar{x}$=39.8	1.3	2.3	7	.56	.59	.64	2.2	7	.29	.78
BF vs Baseline										
$\bar{x}$=38.5 $\bar{x}$=27.3	11.2	2.3	7	4.82	.002	16.0	3.0	7	5.40	.001
PT vs Baseline										
$\bar{x}$=39.8 $\bar{x}$=27.3	12.5	3.3	7	3.79	.007	16.7	4.1	7	4.03	.005
Overall Treatment (Phases I & II) vs Baseline										
$\bar{x}$=39.2 $\bar{x}$=27.3	11.9	2.6	7	4.65	.002	16.4	3.4	7	4.81	.002

Figure 7

Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

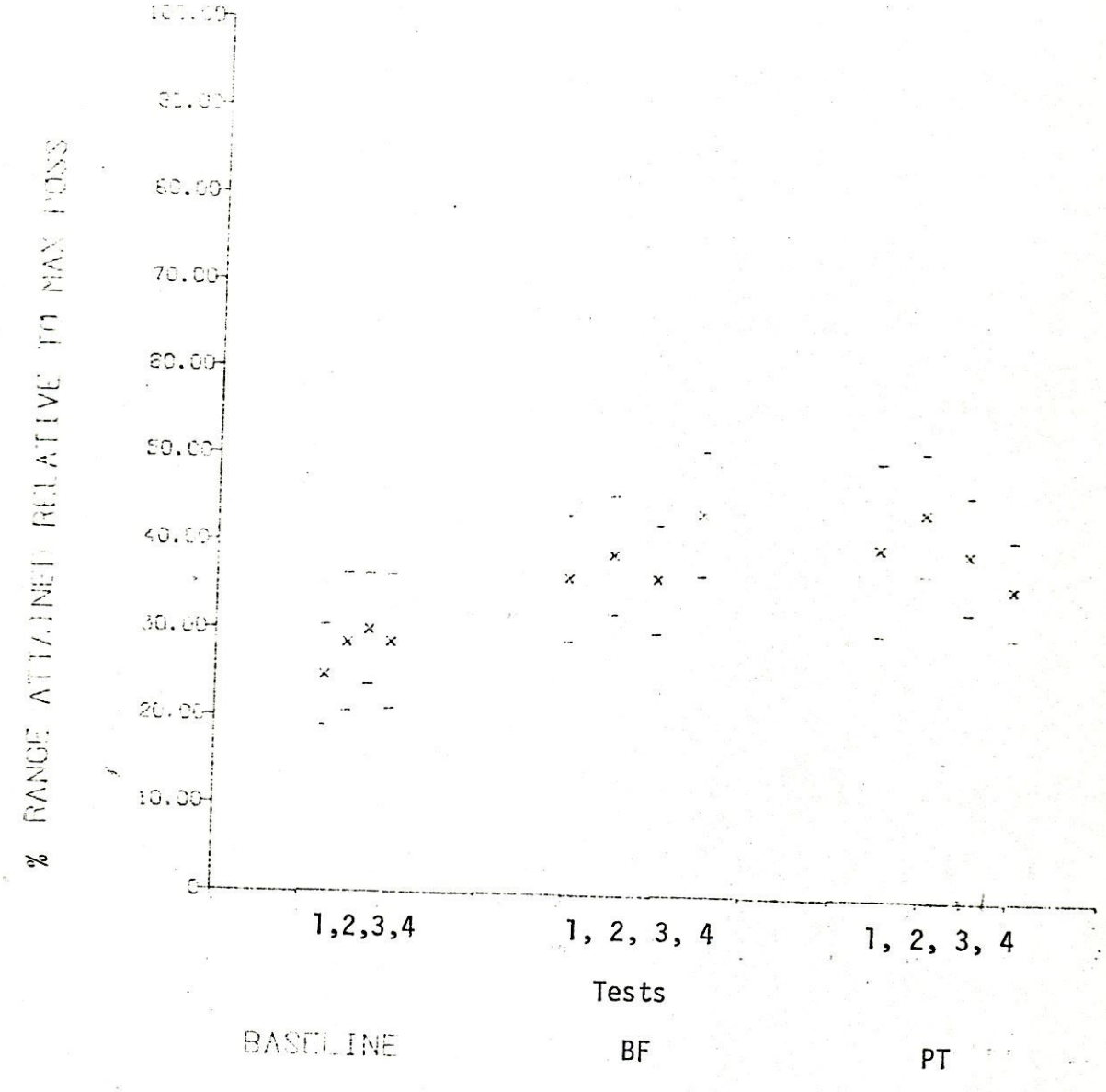


Figure 8

Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

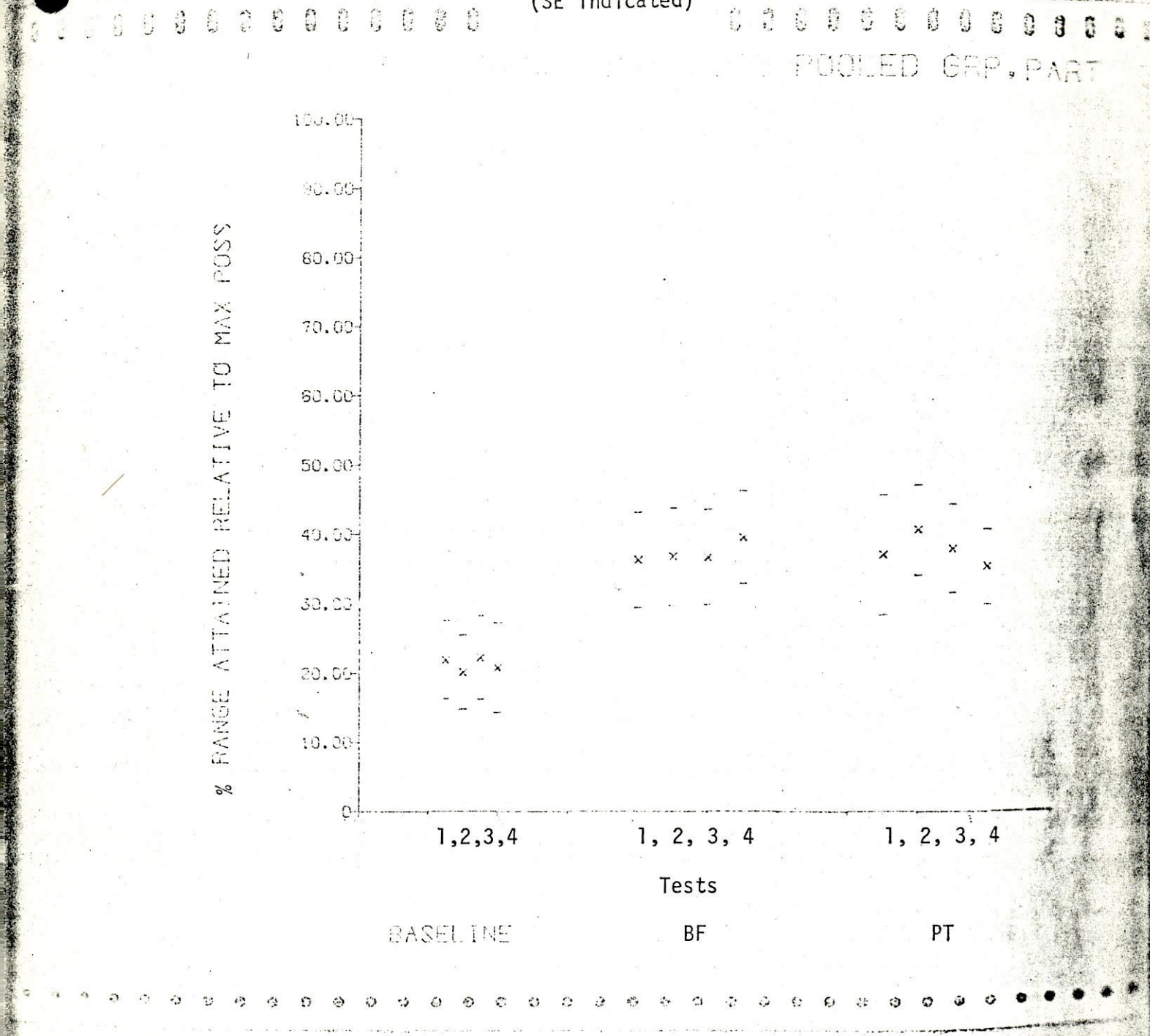


Figure 9

Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

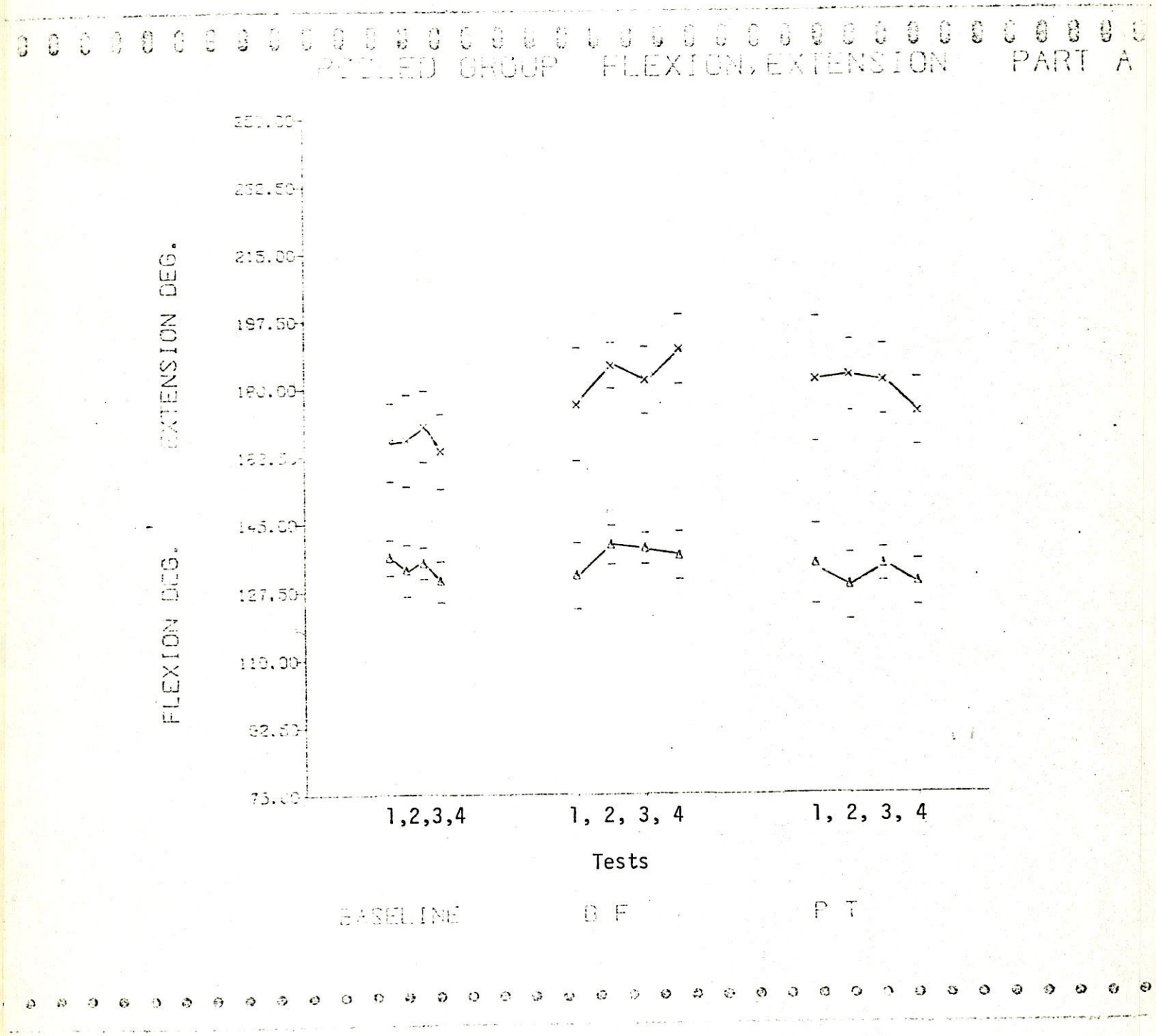
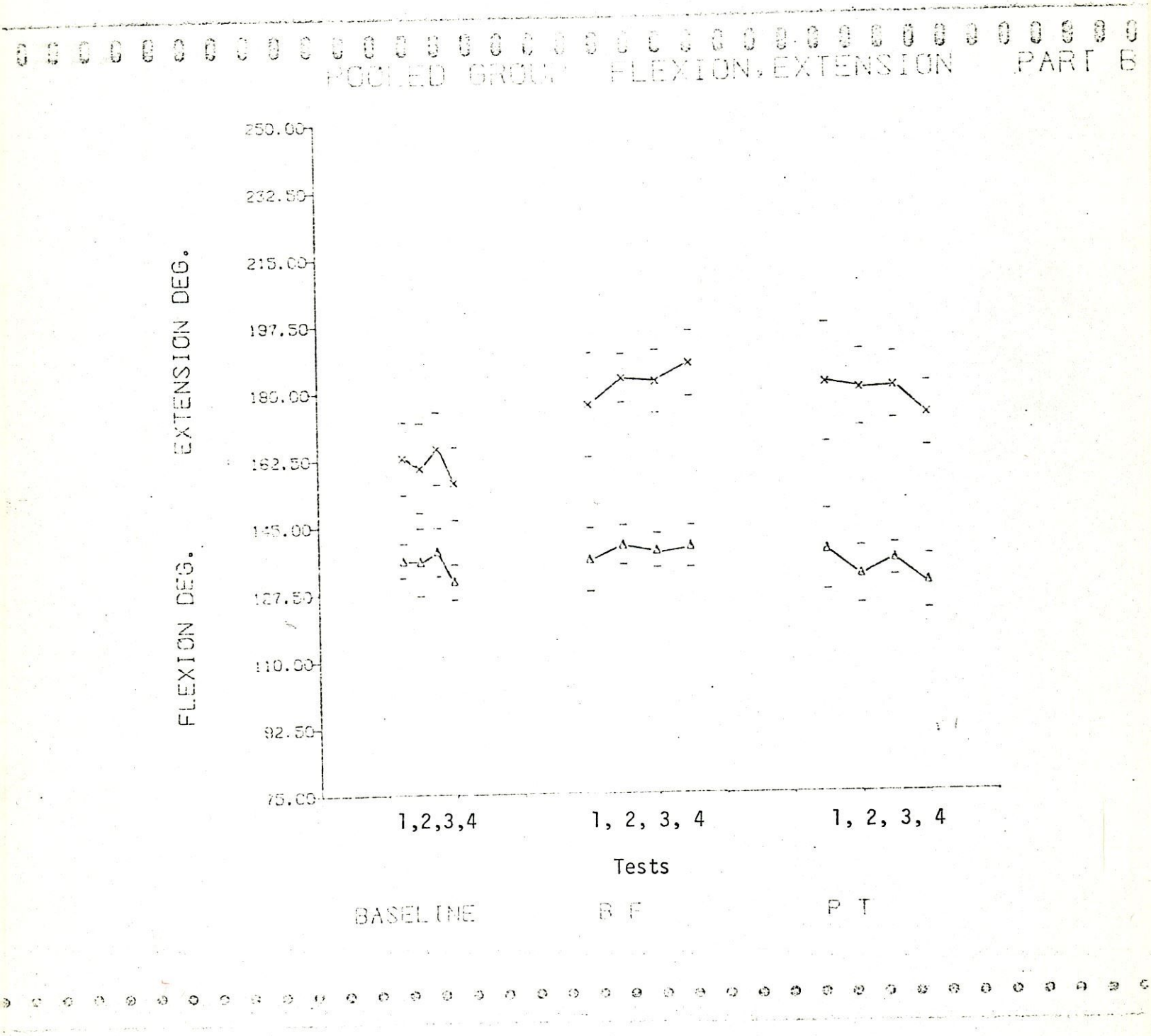


Figure 10

Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)



showed statistically significant (correlated t) differences (means of 11.2 and 16.0, respectively) with associated p values of .002 and .001. A pooled group analysis of PT Part A ($\bar{x}$=39.8) and PT Part B ($\bar{x}$=37.5) over baseline Part A ($\bar{x}$=27.3) and baseline Part B ($\bar{x}$=20.8) also showed statistically significant differences (means of 12.5 and 16.7, respectively) with associated p values of .007 and .005. A comparison between BF vs. PT Part A showed no significant difference (mean difference=1.3) with a p value of .59. A similar comparison of Part B showed an analogous mean difference of .64 with a p value of .78.

A slope analysis of pooled BF vs. pooled PT, Parts A and B, showed no significant differences. Finally, a comparison of the pooled overall treatment for range Part A ($\bar{x}$=39.2) and range Part B ($\bar{x}$=37.2) over baseline Parts A and B was statistically significant for Parts A and B (mean differences=11.9 and 16.4, respectively) at the .002 level and .002 level. In sum, it can be concluded that there were significant increments in range under both BF, and the control PT training, Parts A and B.

Intergroup Comparisons[33] (see Table 12)

The following are comparisons of Group 1 which received BF first and then PT training, and Group 2 which received PT first and then BF

---

[33]There were a few patients (i.e., 2, 6, 8, and 9) who had lower scores on range Part B vs. range Part A during baseline. This was due to the fact that Part B (Rate) required complete but continuous responding. In this situation, the above patients failed to either extend or flex at baseline to the full extent they were capable while simultaneously concentrating on rate, etc. After training ensued, however, and patients were presumably always attending to several aspects of their movements, the disparity between range Parts A and B disappeared. Patient 2 had, however, somewhat less range during Part B, for Phases I and II. Also, interestingly, except for Patient 9, patients did not attain maximum range in the unaffected limb when it was tested during baseline. This is because a person does not, in normal movements, e.g., extend his wrist to the absolute maximum possible, as the latter would entail a forced "unnatural" contraction to the utter joint limit of movement.

Table 12

Intergroup Comparisons for RANGE OF MOTION

(Percentage scores--computed using mean values of the range attained relative to the maximum range possible for a given patient)

			Part A							Part B				
			Δ	SE	df	t	P			Δ	SE	df	t	P
Intergroup Comparisons (Group 1 vs Group 2)								Intergroup Comparisons (Group 1 vs Group 2)						
Baseline 1 vs Baseline 2								Baseline 1 vs Baseline 2						
$\bar{x}$=26.2	$\bar{x}$=28.4		2.2	14.4	6	.15	.89	$\bar{x}$=17.2	$\bar{x}$=24.4		7.3	11.9	6	.61	.56

training. An intergroup comparison of the equality between Group 1 Part A ($\bar{x}$=26.2) and Part B ($\bar{x}$=17.2) and Group 2 Part A ($\bar{x}$=28.4) and Part B ($\bar{x}$=24.4) at baseline (mean differences=2.2 and 7.3, respectively) showed baseline scores to be not statistically different with associated p values of .89 and .56.

The two groups were, at baseline, in terms of absolute differences, more equivalent on the range variable than they were on the averaged EMG variable. The standard error terms between the groups also did not differ at baseline. Nor did a doubling of the standard error term over baseline occur for Group 2 as training ensued for both Phases I and II vs. no change in the standard error term for Group 1, as did occur for Group 2 vs. Group 1 on the averaged EMG variable.

The gains made for individuals within Groups 1 and 2 were relatively similar irrespective of their starting levels for range at baseline. Also, the gains for Group 2, as a whole, were less over baseline than the gains for Group 1 over baseline. Furthermore, the two individuals in Group 2 who contributed most to the training effect for the averaged EMG variable over baseline in Phases I and II, did not contribute a similar large amount to the range gains for Group 2 relative to the two individuals who contributed little or nothing to the overall Group 2 gains on the averaged EMG variable.

Intragroup Comparisons:[34] (see Table 13 and Figures 11, 12, 13, 14, 15, 16, 17, and 18.)

[34]All subjects (save Patient 7 who could not be included in the range calculations [see p. 148]) made a change in range. Patient 5 who is presented in "Case Histories" as the only non-learner and who showed essentially no increments in EMG recruitment, showed increments in range (e.g., Part A) by 7 units in PT over baseline and then by 5 units (i.e., a loss of 2 units) in BF over baseline. However, during the eight weeks of experimental training, Patient 5 would always produce movements which had a considerable amount of range on the first few attempts but, after the first few attempts, the response would fatigue, diminish, and could not be replicated. As fatigue was rapid, some of Patient 5's initial responses in a test session were much greater than his mean score. (For details of this patient's overall performance see his case history, pp. 264-279.)

Table 13

Intragroup Comparisons for RANGE OF MOTION

(Percentage scores--computed using mean values of the range attained relative to the maximum range possible for a given patient)

	Part A					Part B				
	Δ	SE	df	t	P	Δ	SE	df	t	P
Intragroup Comparisons: Group 1 (BF then PT)										
BF vs PT										
$\bar{x}$=40.7 vs $\bar{x}$=44.5	3.8	4.0	3	.97	.40					
$\bar{x}$=39.7 vs $\bar{x}$=41.8						2.1	4.2	3	.50	.65
(Phase I) BF vs Baseline										
$\bar{x}$=40.7 vs $\bar{x}$=26.2	14.4	4.0	3	3.66	.03					
$\bar{x}$=39.7 vs $\bar{x}$=17.2						22.6	2.7	3	8.35	.003
(Phase II) PT vs Baseline										
$\bar{x}$=44.5 vs $\bar{x}$=26.2	18.3	5.0	3	3.62	.04					
$\bar{x}$=41.8 vs $\bar{x}$=17.2						24.6	5.6	3	4.37	.02
Intragroup Comparisons: Group 2 (PT then BF)										
BF vs PT										
$\bar{x}$=35.0 vs $\bar{x}$=36.3	1.2	2.4	3	.49	.66					
$\bar{x}$=33.1 vs $\bar{x}$=33.9						.80	1.8	3	.45	.68
(Phase I) PT vs Baseline										
$\bar{x}$=35.0 vs $\bar{x}$=28.4	6.7	1.7	3	4.04	.03					
$\bar{x}$=33.1 vs $\bar{x}$=24.4						8.7	2.3	3	3.71	.03
(Phase II) BF vs Baseline										
$\bar{x}$=36.3 vs $\bar{x}$=28.4	7.9	1.5	3	5.25	.01					
$\bar{x}$=33.9 vs $\bar{x}$=24.4						9.5	2.3	3	4.13	.02

Figure 11

Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

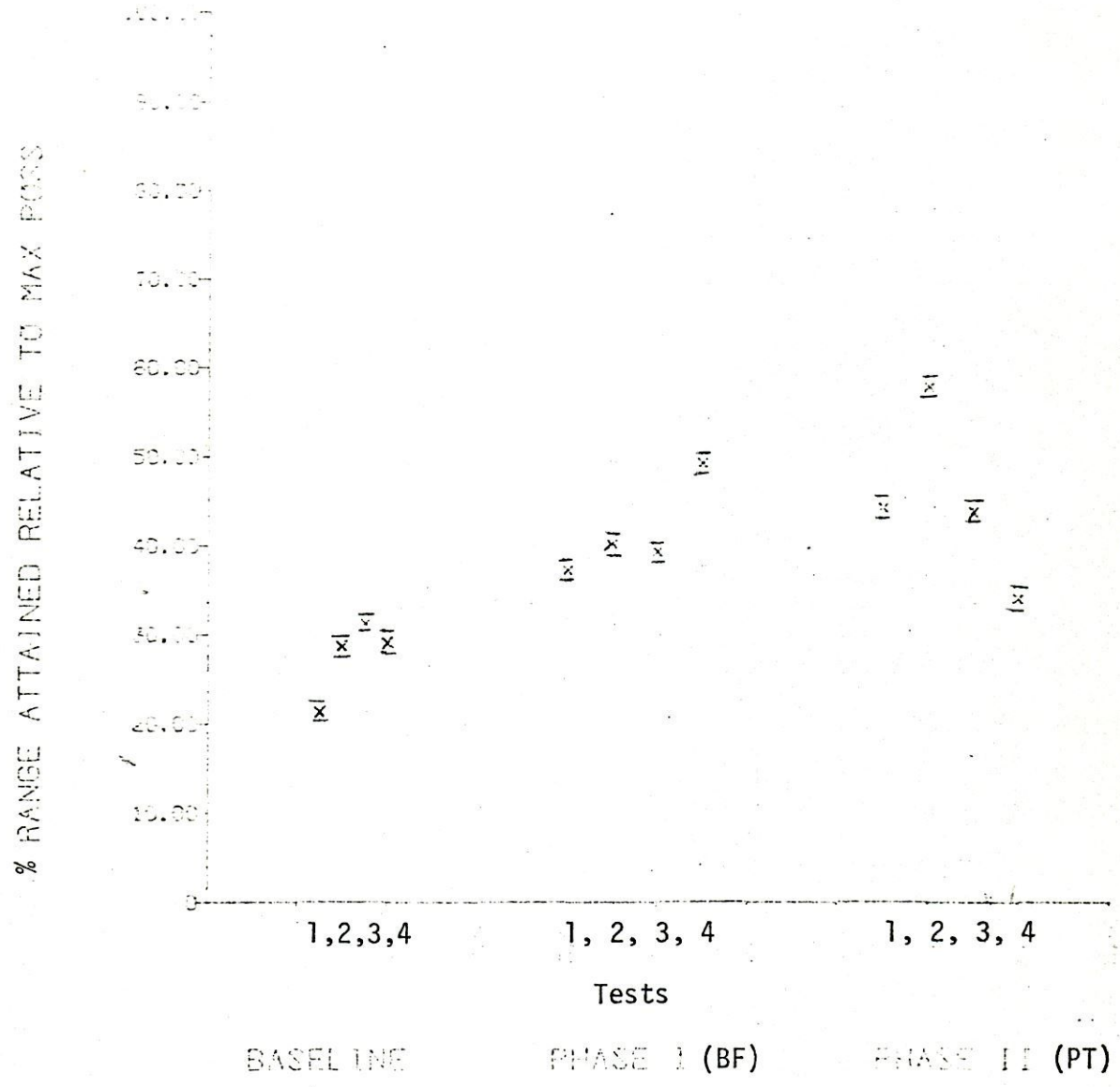


Figure 12

Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

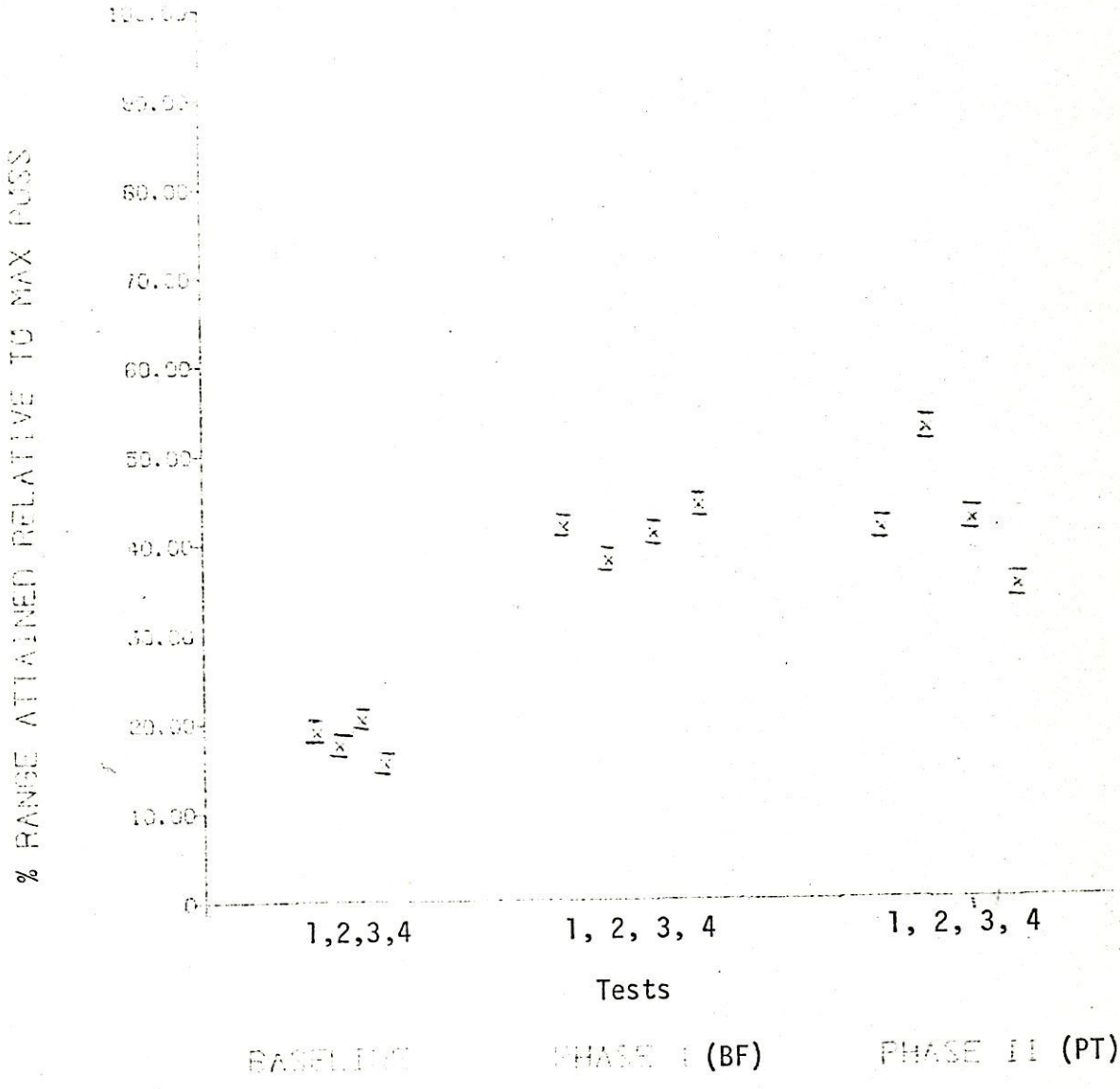


Figure 13

Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

Figure 14

Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

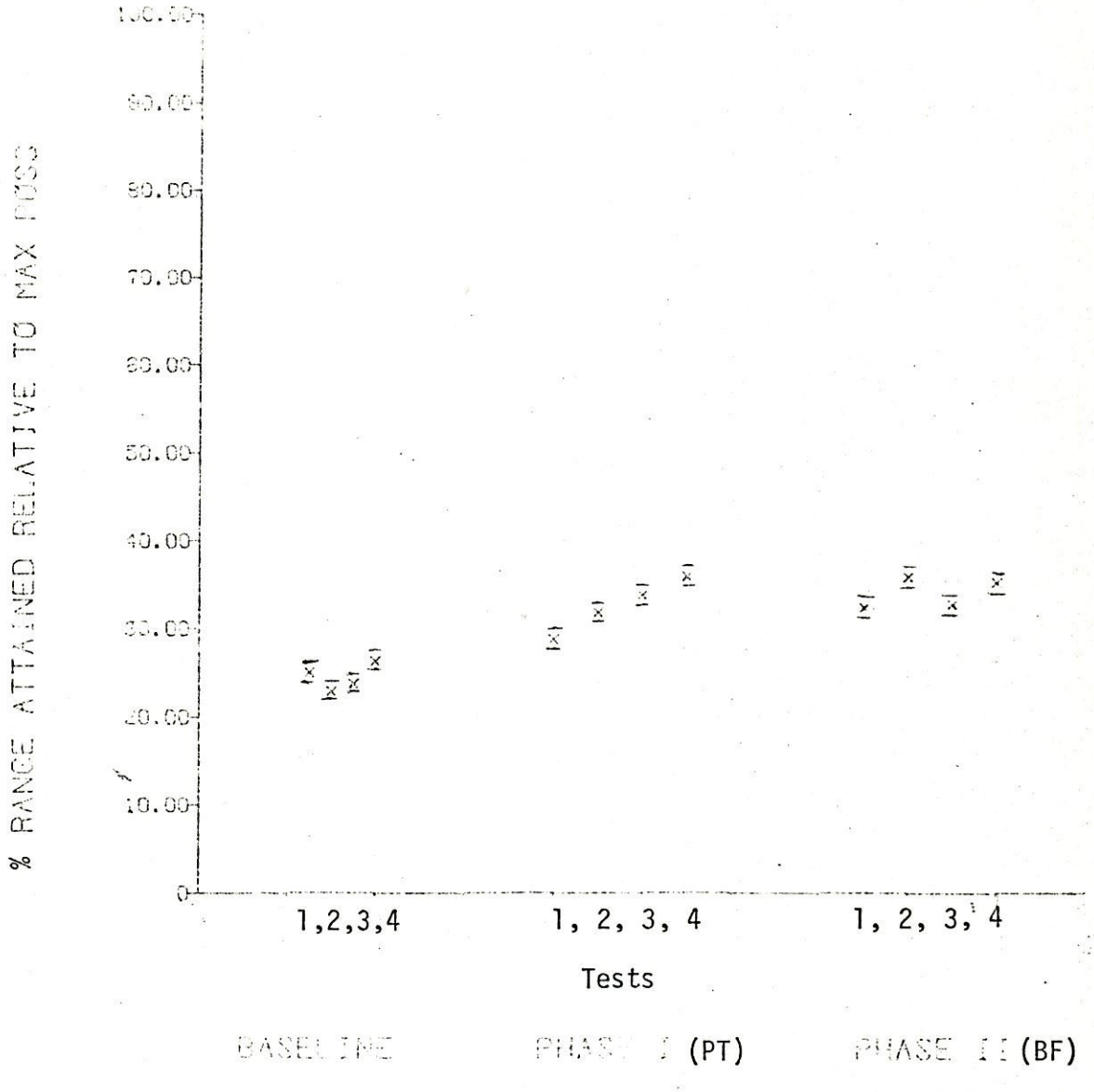


Figure 15

Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

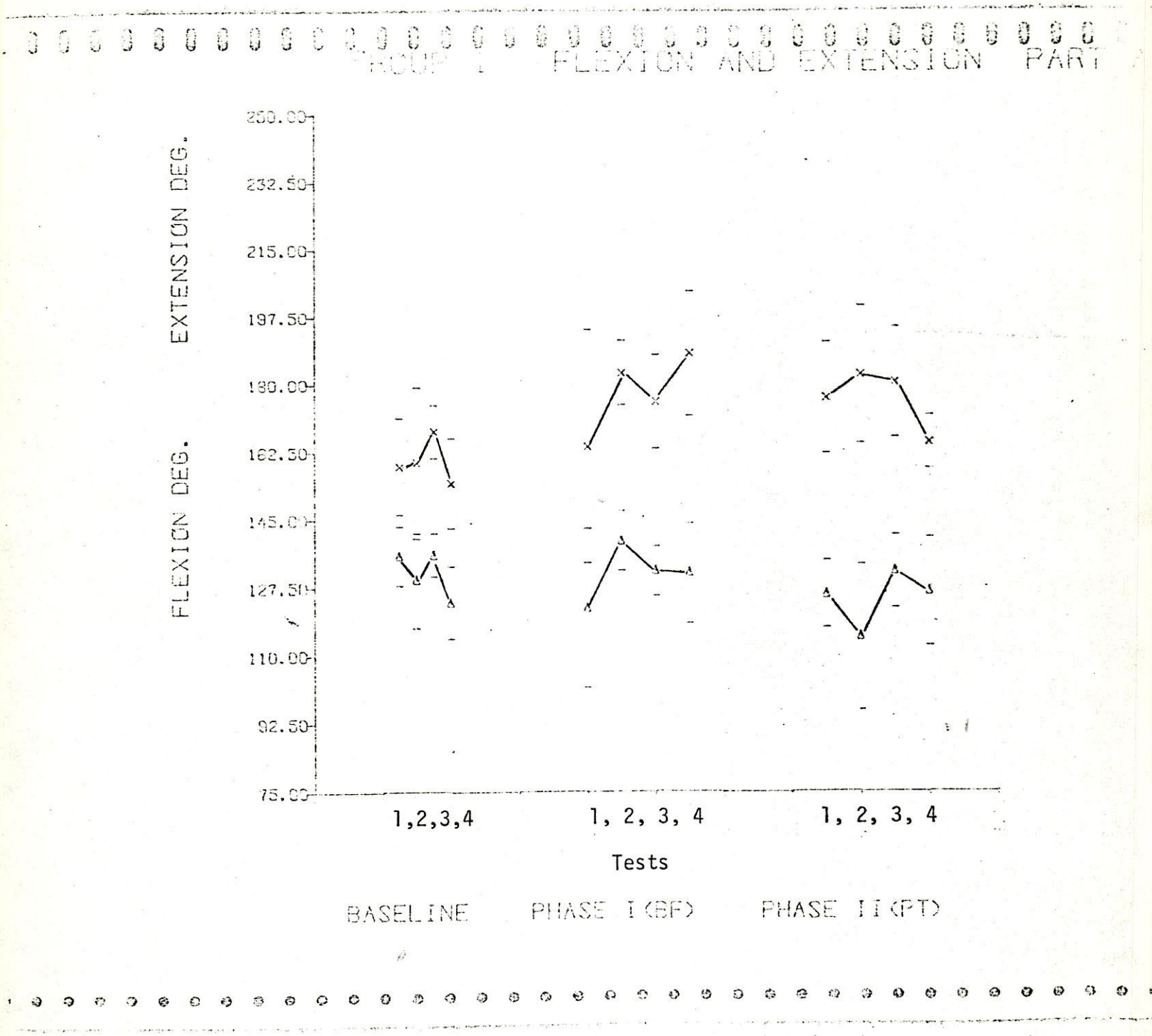


Figure 16

Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

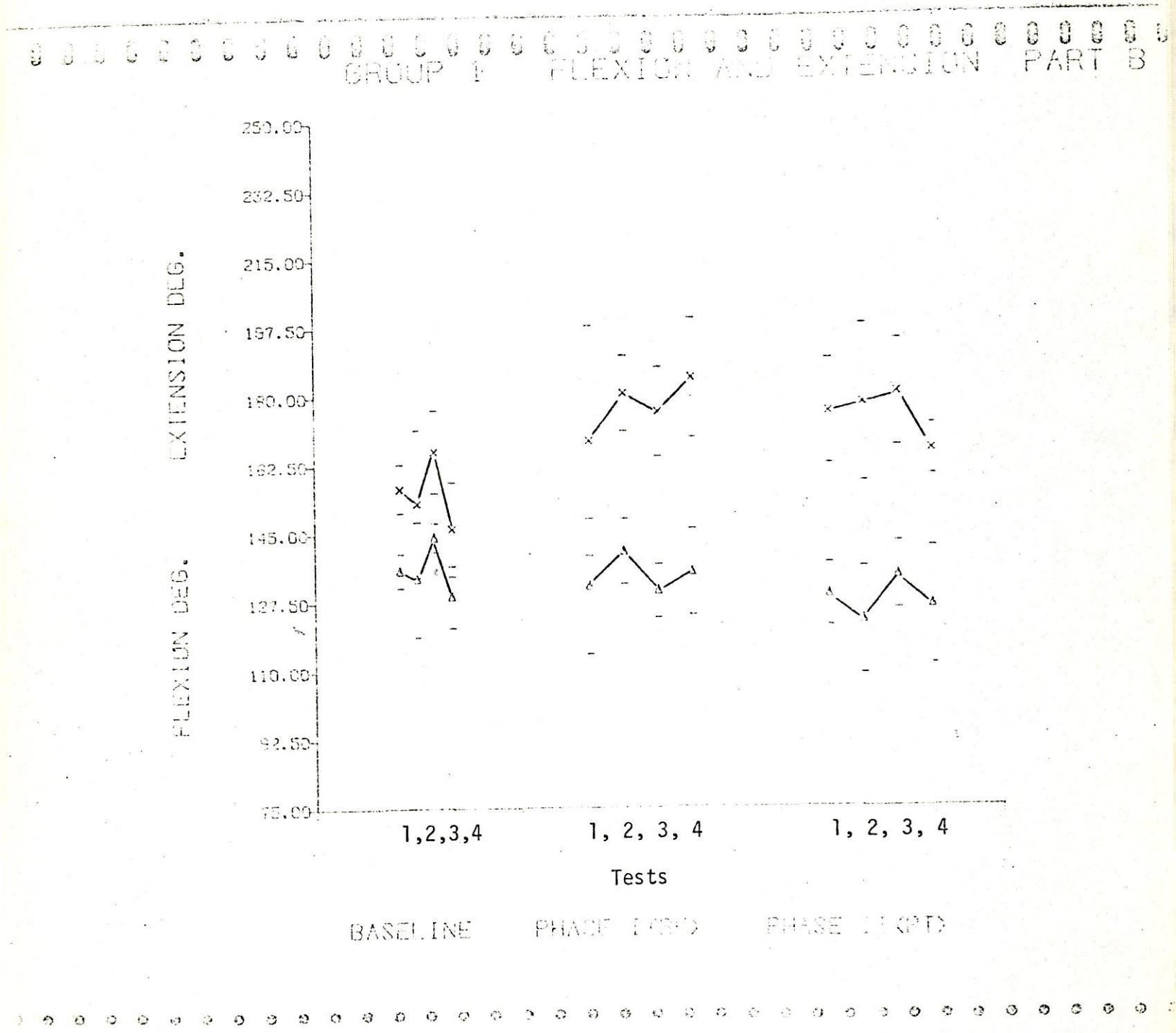


Figure 17

Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

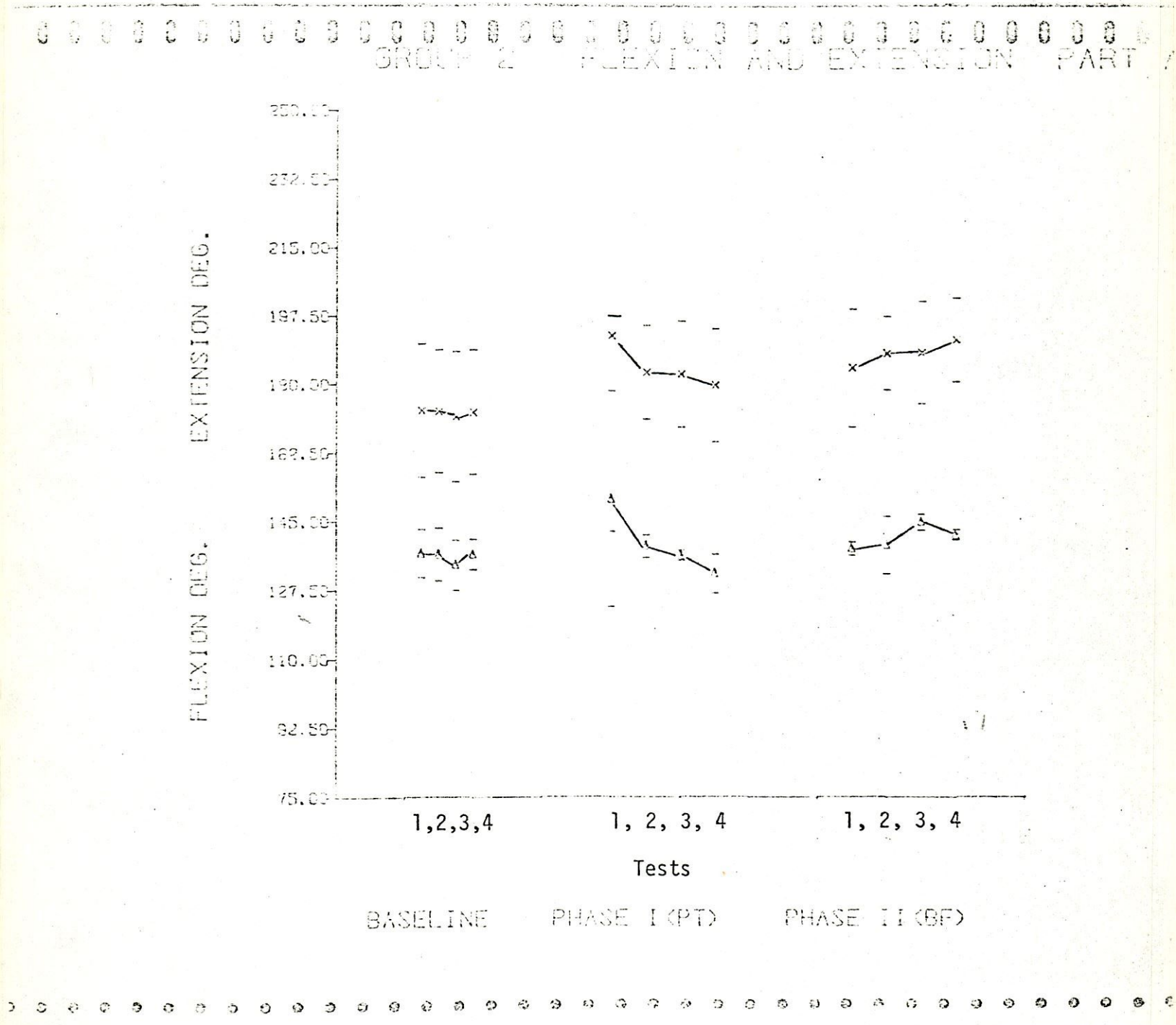
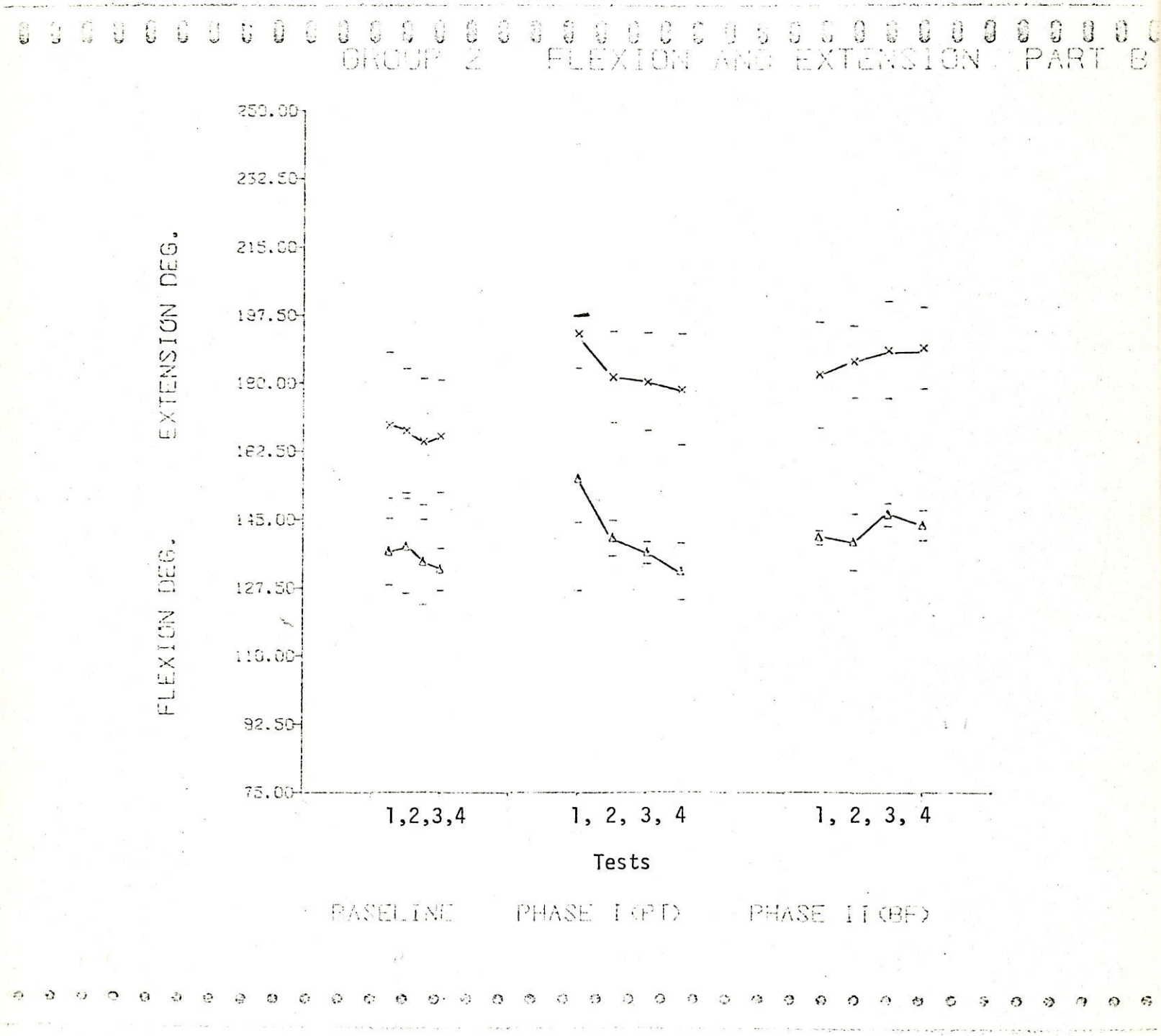


Figure 18

Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)



Group 1

The following are intragroup comparisons similar to the above pooled group comparisons. Group 1 comparisons of BF Part A ($\bar{x}$=40.7) and BF Part B ($\bar{x}$=39.7) over baseline Part A ($\bar{x}$=26.2) and baseline Part B ($\bar{x}$=17.2) showed statistically significant differences (means of 14.4 and 22.6, respectively) with p values of .03 and .003. Group 1 comparisons of PT Part A ($\bar{x}$=44.5) and PT Part B ($\bar{x}$=41.8) over baseline Part A ($\bar{x}$=26.2) and baseline Part B ($\bar{x}$=17.2) also showed statistically significant differences (means of 18.3 and 24.6, respectively) with p values of .04 and .02. Group 1 intragroup comparisons of BF vs. PT, Parts A and B (mean differences=3.8 and 2.1, respectively) were not significant with p values of .40 and .65.

Group 2

Group 2 comparisons of PT Part A ($\bar{x}$=35.0) and PT Part B ($\bar{x}$=33.1) over baseline Part A ($\bar{x}$=28.4) and baseline Part B ($\bar{x}$=24.4) (mean differences=6.7 and 8.7, respectively) also showed statistically significant gains in PT training with p values of .03 and .03. A similar Group 2 comparison of BF Part A ($\bar{x}$=36.3) and BF Part B ($\bar{x}$=33.9) over baseline Part A ($\bar{x}$=28.4) and baseline Part B ($\bar{x}$=24.4) (mean differences=7.9 and 9.5, respectively) again showed significant increments in range with p values of .01 and .02. Group 2 intragroup comparisons of BF vs. PT, Parts A and B (mean differences=1.2 and .80, respectively) were not significant with p values of .66 and .68.

Ancillary Aspects of the Data

One might expect, as did this author, that averaged EMG activity would, by and large, covary with range. Yet inspection of the range data for particular mean individual performances within a specified

test session seems to indicate a bit of an anomalous relationship between averaged EMG activity and range. For example, individuals, could increase averaged EMG activity and decrease or maintain range and vice versa. The data for the following individuals, Patients 1, 2, and 8, are comparatively great (Patients 1 and 8) and comparatively slight (Patient 2) examples of the manner in which averaged EMG activity and range could vary more or less independently of each other within a test session's performance. For example, for Part A (Phase I or II as indicated), there was simultaneous variation as follows:

(Averaged EMG activity was analyzed in terms of absolute μv of activity; while range of motion was analyzed in terms of percentage of range attained relative to the individual maximum range possible. For purposes of comparison, range is presented both in terms of percentage scores and in terms of absolute number of degrees.)

Patient 8, Phase I (BF)

	Tests			
	1	2	3	4
Averaged EMG Activity	49.0 μv	107.1 μv	92.4 μv	98.0 μv
Range of Motion (Degrees)	64.4^0	70.0^0	73.0^0	90.1^0
(% $\frac{\text{Range Attained}}{\text{Maximum Range Possible}}$)	60%	60.8%	63.1%	78.3%

Patient 2, Phase I (PT)

	Tests			
	1	2	3	4
Averaged EMG Activity	120.4 μv	134.4 μv	116.4 μv	158.2 μv
Range of Motion (Degrees)	64.2^0	58.3^0	74.5^0	69.5^0
(% $\frac{\text{Range Attained}}{\text{Maximum Range Possible}}$)	49.4%	44.8%	57.3%	53.4%

Patient 1, Phase II (BF)

	Tests			
	1	2	3	4
Averaged EMG Activity	36.4 μv	34.3 μv	49.7 μv	39.2 μv
Range of Motion (Degrees)	18.2^0	37.9^0	21.2^0	37.0^0
$(\% \frac{\text{Range Attained}}{\text{Maximum Range Possible}})$	14.0%	29.2%	16.3%	28.4%

What the above seems to indicate is that averaged EMG activity and range can increase or decrease more or less independently with respect to each other. This observation cannot be accounted for here, but it might be remarked that the sample in this study contains many pathological factors contributing to total response. Ideally these factors should be accounted for but are impossible to account for with current knowledge and the given sample. Some pathological factors which do contribute to response patterns are outlined below. However, these factors vary from patient to patient, both in terms of the amount and kind of an individual problem at a given time in training, as well as in terms of individual patient variation from one response to the next or, more generally, from one session to the next.

Contributory pathological factors are co-contraction of agonists and antagonists or co-contraction of synergists to, e.g., increase EMG activity without increasing range; substitution of a target motion with synergistic contraction of muscles or an increase of tone in the antagonist to, e.g., result in a steady amount of EMG activity and a decrease in range; or relaxation of an antagonist or diminished contraction of synergist muscles to result in greater range of motion with less EMG activity, etc. In sum, all of the above and more are

operative in motor responding which is altered by pathology. It might be added that case histories are helpful here to the extent that they portray idiosyncratic patient pathological problems and progress and, to some extent, the quantitative counterparts of these problems and progress.

Again, viewing the data from a slightly different perspective, looking at the mean total net gains over baseline for individuals in Phases I or II on the averaged EMG and range variables, somewhat similar observations can be made. As was noted in intragroup comparisons for range (see pp. 156-166), the overall gains in range for the two highest averaged EMG variable performers in Group 2 (Patients 4 and 2) were relatively small compared to gains on the averaged EMG variable for these individuals. In addition, in Group 1 there was one individual (Patient 9) who showed practically no gains in averaged EMG activity yet who made an overall gain in range close to the mean gain in range for Group 1. The following results for these three responders are presented as:

Percent scores representing the absolute difference change over baseline in Phases I and II (e.g., Part A)

Patient 2

	Phase I (PT)	Phase II (BF)
Averaged EMG Activity	59.9%	47.4%
Range of Motion	23.2%	14.0%

Patient 4

	Phase I (PT)	Phase II (BF)
Averaged EMG Activity	21.1%	110.6%
Range of Motion	4.2%	18.6%

Patient 9

	Phase I (BF)	Phase II (PT)
Averaged EMG Activity	13.1%	7.5%
Range of Motion	6.5%	25.5%

The above figures again illustrate averaged EMG activity varying somewhat independently of range. For Patient 4 and Patient 9, furthermore, they also show that BF and PT produced different kinds of changes. Patient 4 increased his averaged EMG activity by 110.6% over baseline on BF (89.5% over PT) while he only increased his range by 18.6% over baseline on BF (14.4% over PT). Patient 9 increased her averaged EMG activity by 7.5% over baseline on PT (a decrease of 6.6% from BF which only showed a 13.1% increase over baseline) while she increased range on PT over baseline by 25.5% (a 19% increase over BF). Patient 4 could thus increase his averaged EMG activity on BF to a great degree without similarly increasing range; and Patient 9 could increase range on PT to a good degree while decreasing slightly on averaged EMG activity. For Patient 9, averaged EMG activity barely increased on either BF or PT and range also barely increased on BF, but range increased a good amount on PT.

Summary for Range of Motion

In summary, results for range of motion showed significant increases in range over baseline under both BF training and the control PT training. Furthermore, pooled group differences between gains made under either BF or PT training, Parts A and B, showed no significant differences. Both BF and PT were effectual in increasing range.

Latency[35]--using mean values of time to respond in seconds. Latency was obtainable from Part A (Command) only. (The reader is referred to "Measures" for the method of latency calculation.)

There were not the multitudinous significant or suggestive trends for latency as there were for the major variables, averaged EMG activity and range of motion. Hence, any interesting or important comparisons are treated together (see Tables 14, 15, and 16, and Figures 19, 20, and 21). There was a significant difference between the pooled group comparison of BF ($\bar{x}$=.62) vs. PT ($\bar{x}$=.80) (mean difference=.18) with an associated p value of .059, so that pooled group PT training latency was longer. The same increase in latency for PT training held for an intragroup comparison of Group 2 BF ($\bar{x}$=.48) vs. PT ($\bar{x}$=.70) training (mean difference=.22) with a less than conventional but suggestive p value of .09. Also, although a comparison of baseline latency for Group 1 vs. Group 2 was not statistically significant, the mean latency for Group 1 ($\bar{x}$=.71) was .23 seconds more than the mean latency for Group 2 ($\bar{x}$=.48). A pooled group comparison of PT ($\bar{x}$=.80) over baseline ($\bar{x}$=.60) had an associated p value of .17.

In general, what seems to be the case (Figure 19) is that pooled BF performance (Group 1 and 2) had a latency which was not significantly different from baseline and which remained relatively the same throughout BF training sessions. On the other hand, pooled PT performance (irrespective of the phase of PT training) showed a relatively very long latency in the initial test session of the first of four test sessions and continued to decline over the four sessions.

---

[35]Patient 10 was omitted from a latency analysis as his latency was inordinately long relative to that of the rest of the sample population and followed a unique course of change with respect to his particular problem (see his case history, pp. 346-363).

Table 14

Pooled Group Comparisons for LATENCY*

(Computed using mean values [in seconds] of latency)

	Part A*				
	Δ	SE	df	t	P
Pooled Group Comparisons (Group 1 & 2)					
BF vs PT $\bar{x}$=.62 $\bar{x}$=.80	.18	.08	7	2.24	.059
BF vs Baseline $\bar{x}$=.62 $\bar{x}$=.60	.02	.09	7	.23	.82
PT vs Baseline $\bar{x}$=.80 $\bar{x}$=.60	.20	.13	7	1.52	.17
Overall Treatment (Phase I & II) vs Baseline $\bar{x}$=.70 $\bar{x}$=.60	.10	.10	7	1.06	.32

*Latency obtained from Part A only.

Table 15

Intergroup Comparisons (Group 1 vs Group 2) for LATENCY*

(Computed using mean values [in seconds] of latency)

	Part A*				
	Δ	SE	df	t	P
Intergroup Comparisons (Group 1 vs Group 2)					
Baseline 1 vs Baseline 2					
$\bar{x}$=.71 $\bar{x}$=.48	.23	.23	6	1.01	.35

*Latency obtained from Part A only.

Table 16

Intragroup Comparisons for LATENCY*

(Computed using mean values [in seconds] of latency)

	Part A*				
	Δ	SE	df	t	P
Intragroup Comparisons: Group 1 (BF then PT)					
BF vs PT					
$\bar{x}$=.90 $\bar{x}$=.76	.14	.15	3	.98	.40
(Phase I) BF vs Baseline					
$\bar{x}$=.75 $\bar{x}$=.71	.04	.11	3	.37	.73
(Phase II) PT vs Baseline					
$\bar{x}$=.90 $\bar{x}$=.71	.19	.22	3	.85	.45
Intragroup Comparisons: Group 2 (PT then BF)					
BF vs PT					
$\bar{x}$=.48 $\bar{x}$=.70	.22	.09	3	2.50	.090
(Phase I) PT vs Baseline					
$\bar{x}$=.70 $\bar{x}$=.48	.22	.18	3	1.19	.32
(Phase II) BF vs Baseline					
$\bar{x}$=.48 $\bar{x}$=.48	.00	.16	3	.00	1.0

*Latency obtained from Part A only.

Figure 19

Latency (SE indicated)

POOLED GROUP, PART A

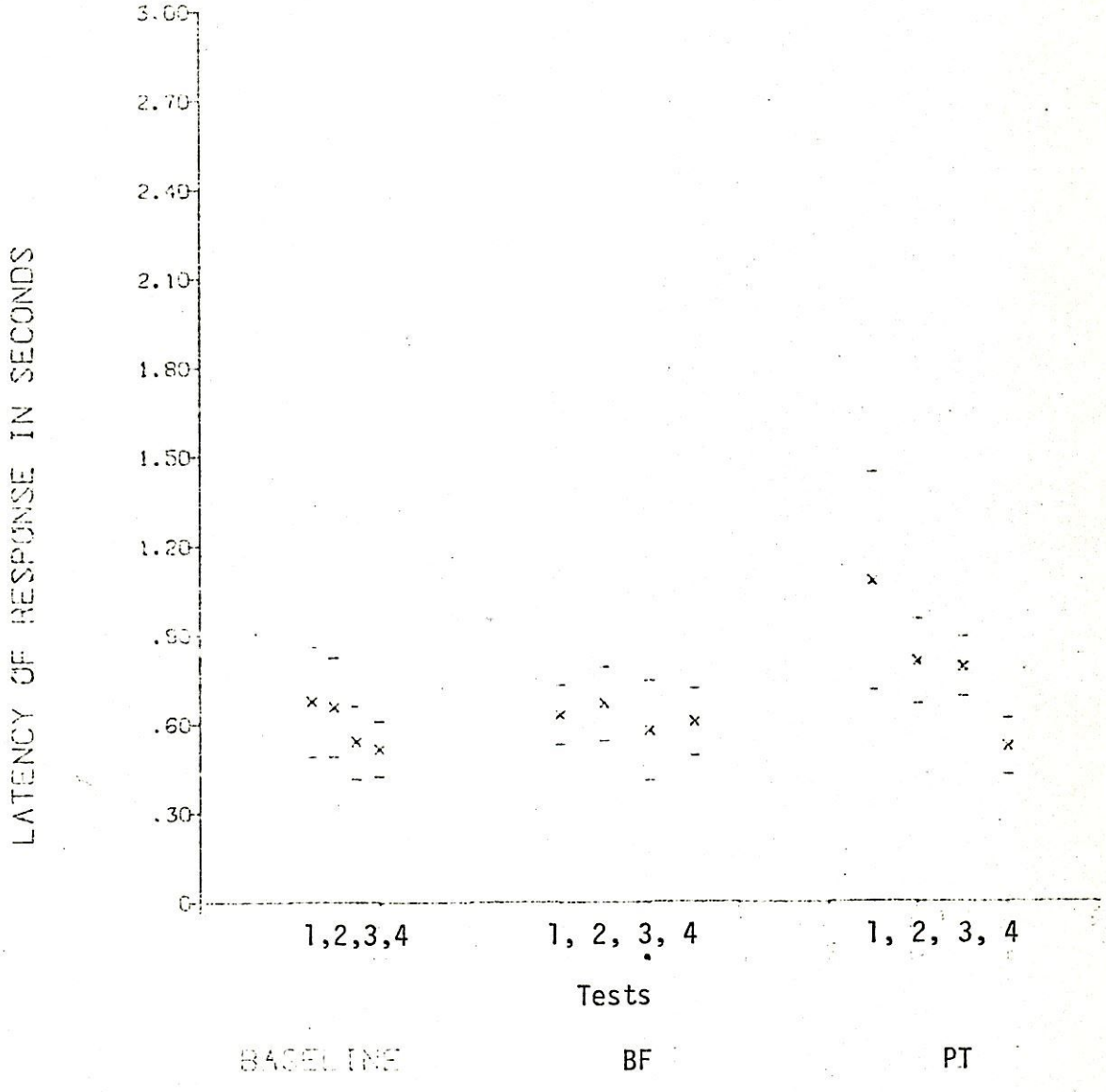


Figure 20

Latency (SE indicated)

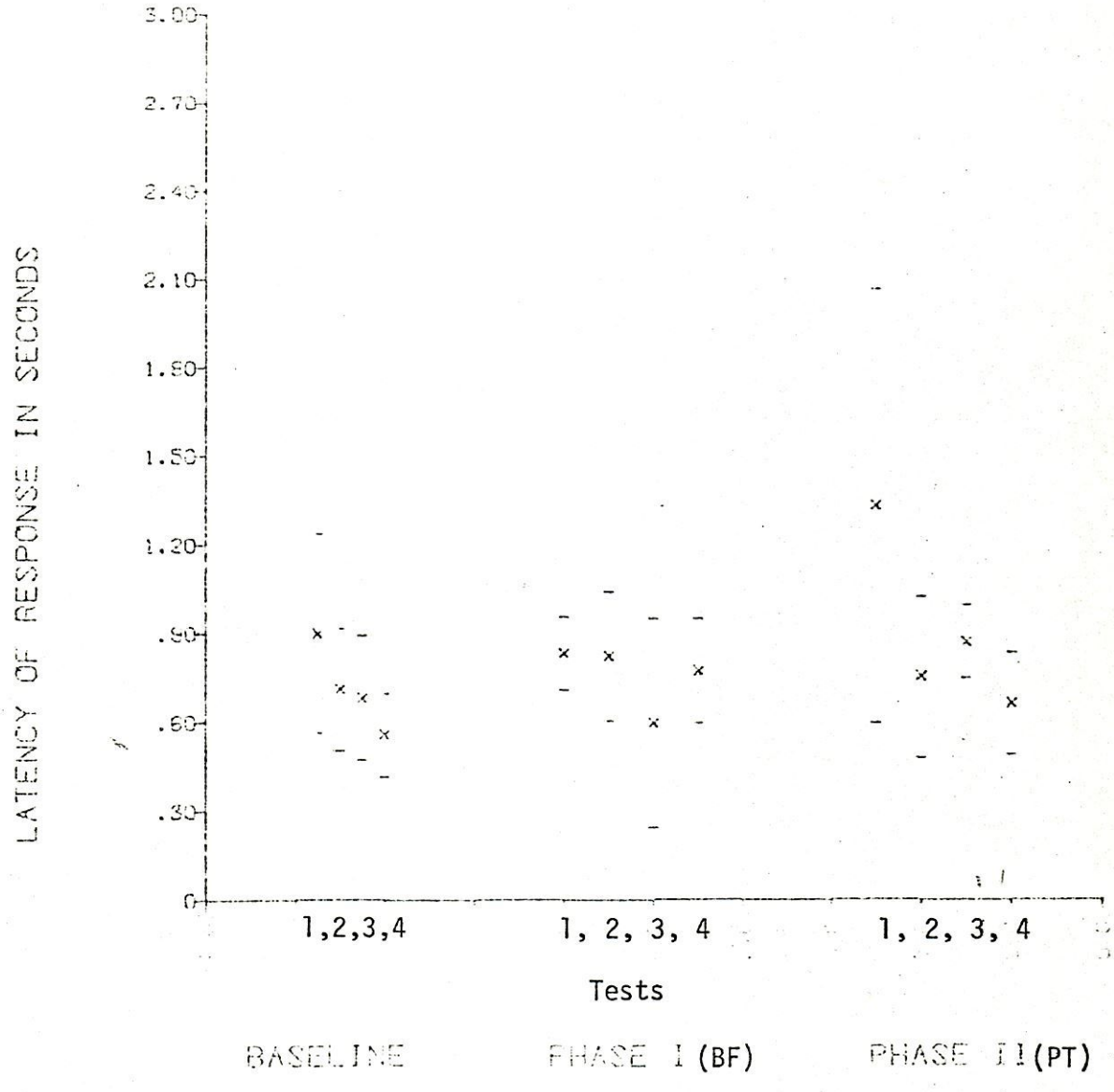
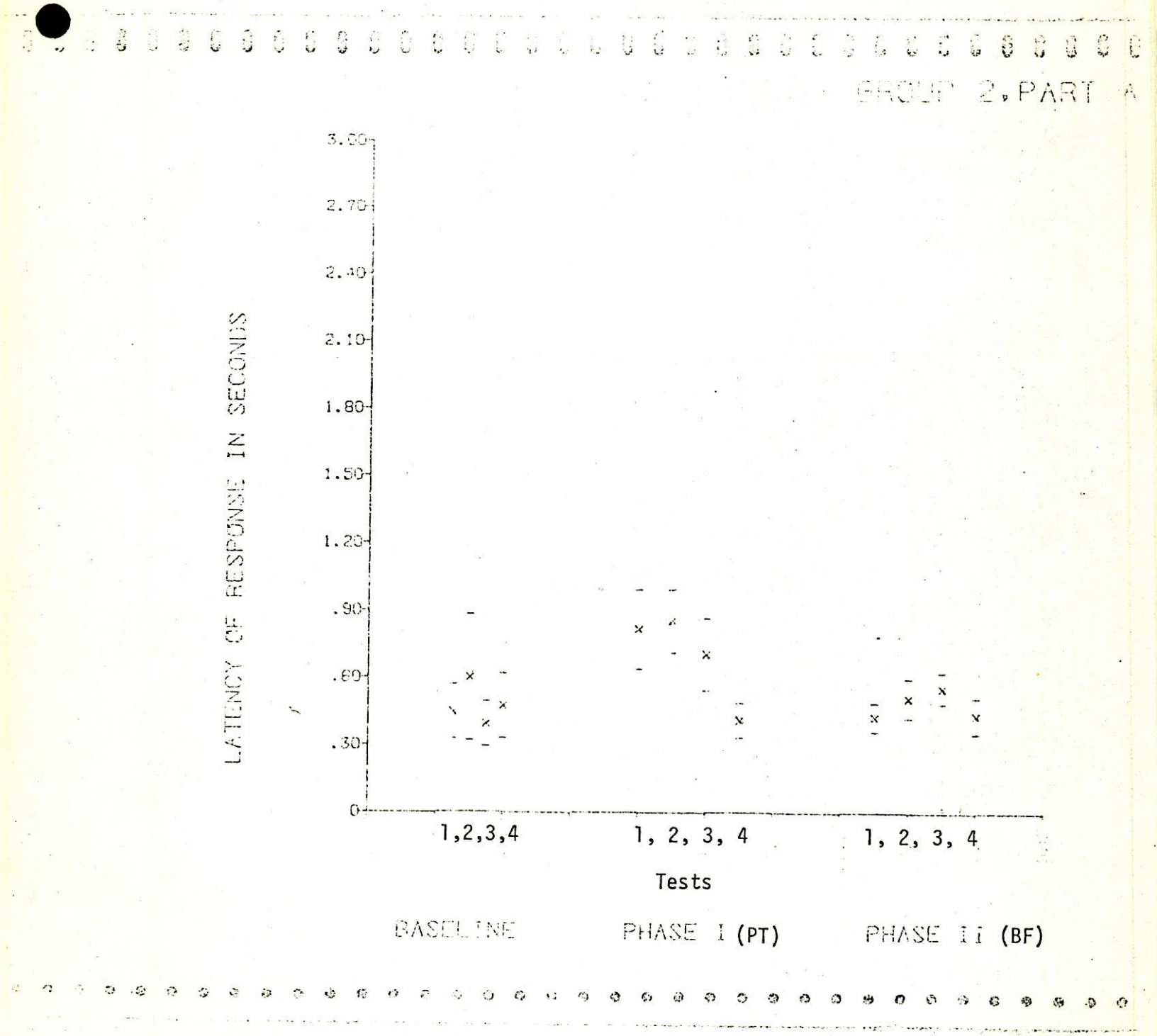


Figure 21

Latency (SE indicated)



At baseline, pooled group latency continually declined. It might have been expected that latency would increase once training ensued under either BF or PT, as the patient would be less yielding to the most facile or spontaneous movement patterns which he had repeatedly "practiced" at baseline. He would now conceivably have to attend to and integrate new features in his movements and therefore would begin a movement less quickly. As training sessions progressed, however, latency would be expected to decrease with increased practice or learning. Although group latencies did initially increase over baseline in the first sessions of both BF and PT training conditions, latency for pooled group PT training started at a much higher level than the latency for pooled group BF training at the first session over baseline, and continued to decline to a low level (a lower level than BF) by the last PT session (regardless of the phase in which PT occurred). Pooled BF training, on the other hand, showed no decline in latency over sessions. A conjectural statement might be made to the effect that the learned efferent activity controlled by visual input (BF) resulted in less rapid responding, than did the learned efferent activity controlled by proprioceptive input (PT).

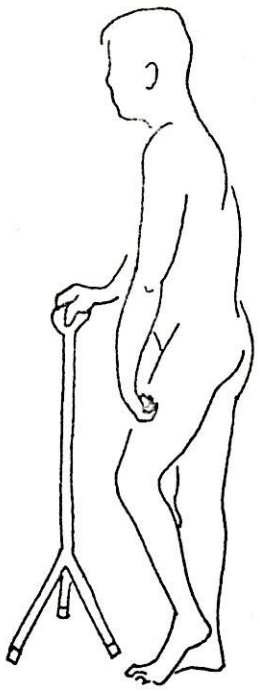
Repetition of Movement

Repetition of movement was not analyzed statistically, as a descriptive statement of it was deemed sufficient. Repetition as a measure was obtainable from Part B (Rate) only where the patient moved repeatedly over a one-minute period. The number of times he moved in this situation was his score on the repetition variable.

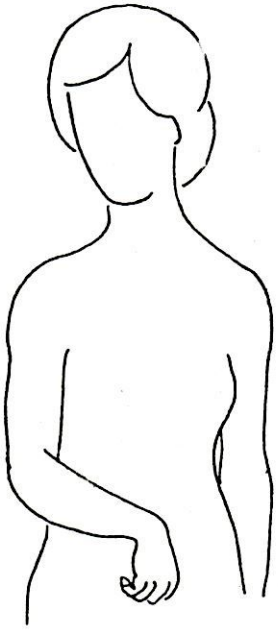
As individual graphs demonstrate (see patient case histories), relative to baseline all patients showed a definite decrease in repeti-

tion of movement during training. In many cases, movement repetition during training did again increase some, but overall, it remained less than at baseline. The "explanation" for this trend would seem to be that once training began, responses were modified by the training procedure so that patient attempts at incorporating whatever specifics were being trained for more accurate, skillful responding, resulted in an overall decrease in rate of movement. That is, the patients did not move without attending to the accuracy as well as to the rate of their movements, and hence, the number of movements decreased. The longer intervals between responses and the longer response durations underlying the decrease in the number of responses might be inferred to represent increased patient attention to response specifics.

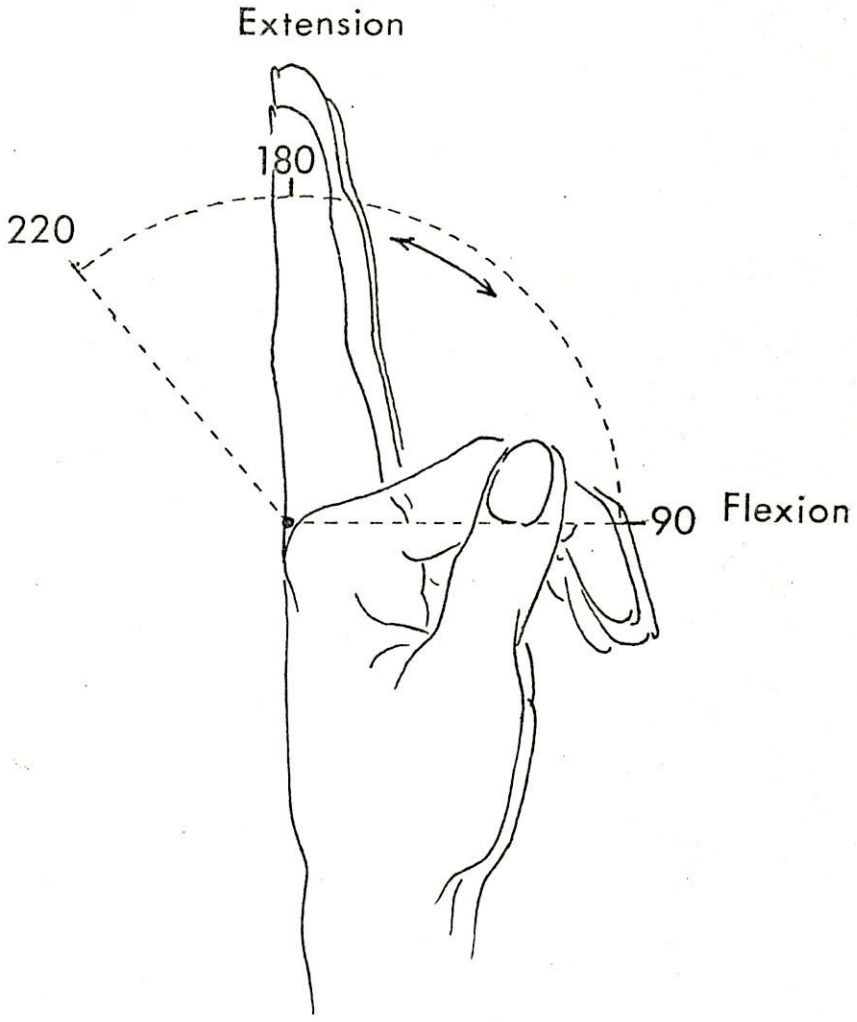
The following graphics and actual patient pictures precede case histories and illustrate movement patterns as well as some of the types of patient problems involved in this study. Also, a Glossary to the terminology used in case histories is provided on pp. 403-410.



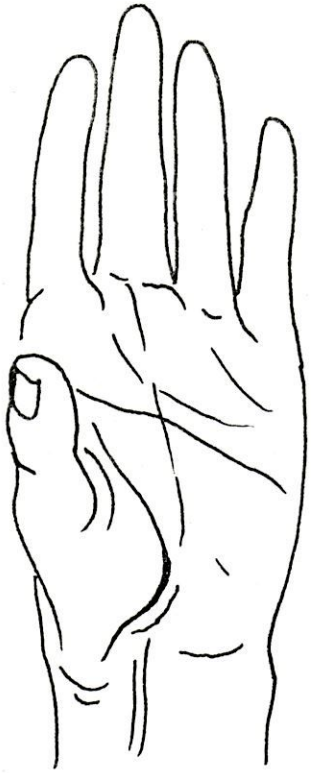
Typical Hemiplegic Posture



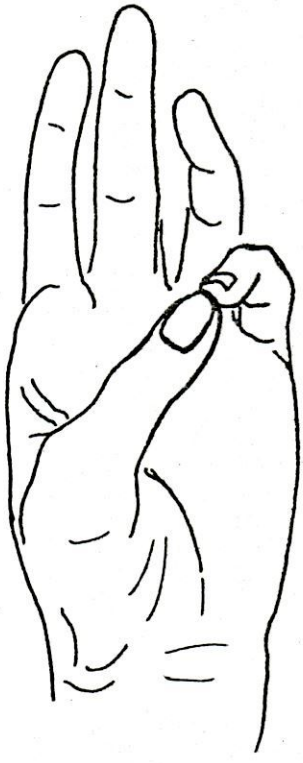
Typical Hemiplegic Posture
(upper extremity)



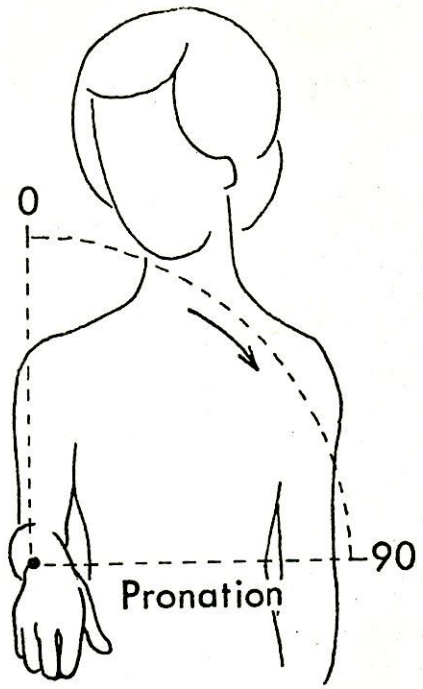
Finger (MP)



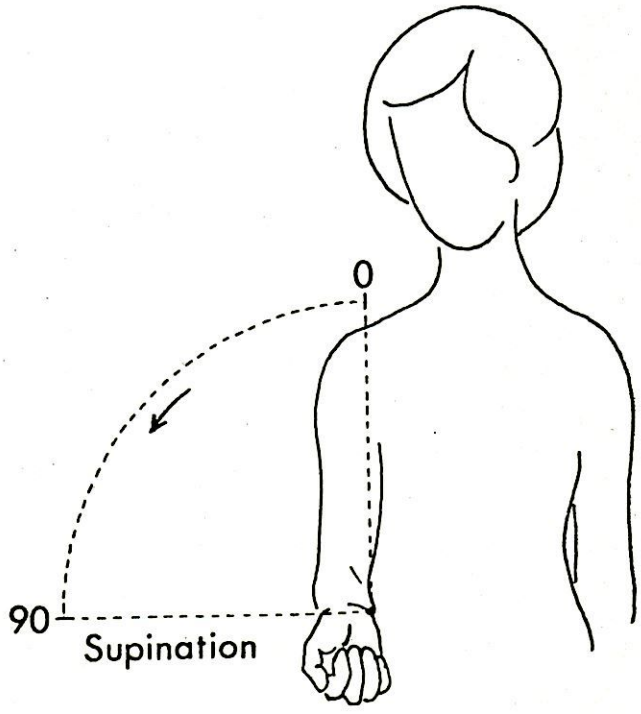
Abducted
Thumb



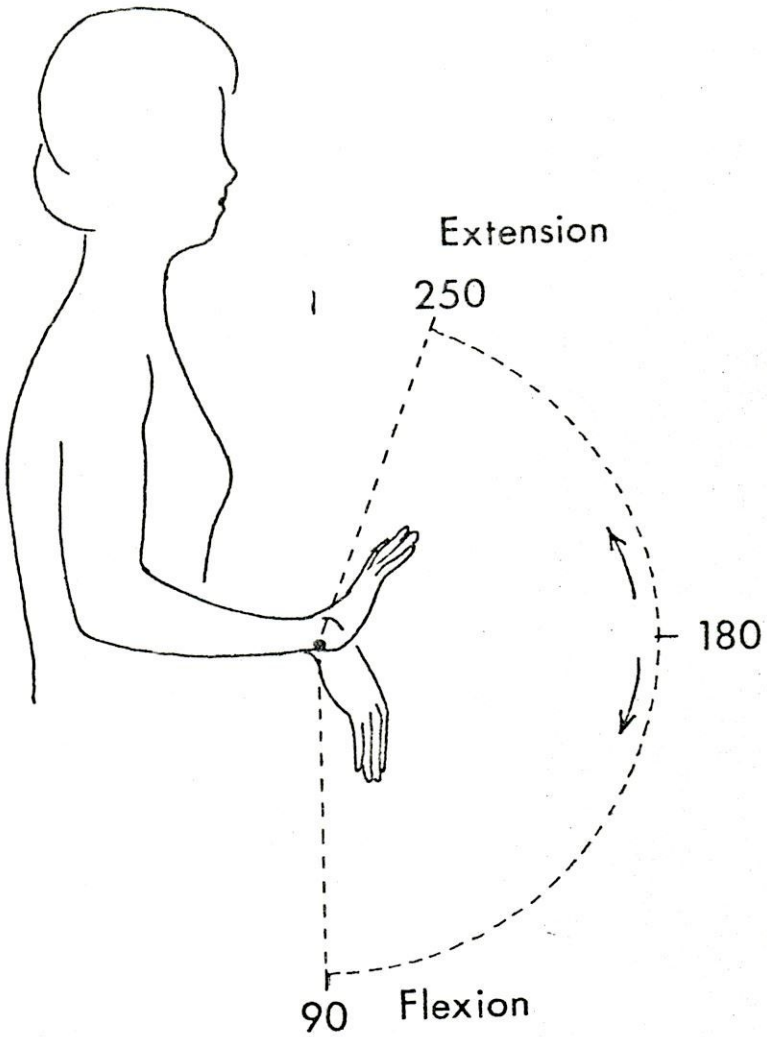
Abducted–
opposed
Thumb



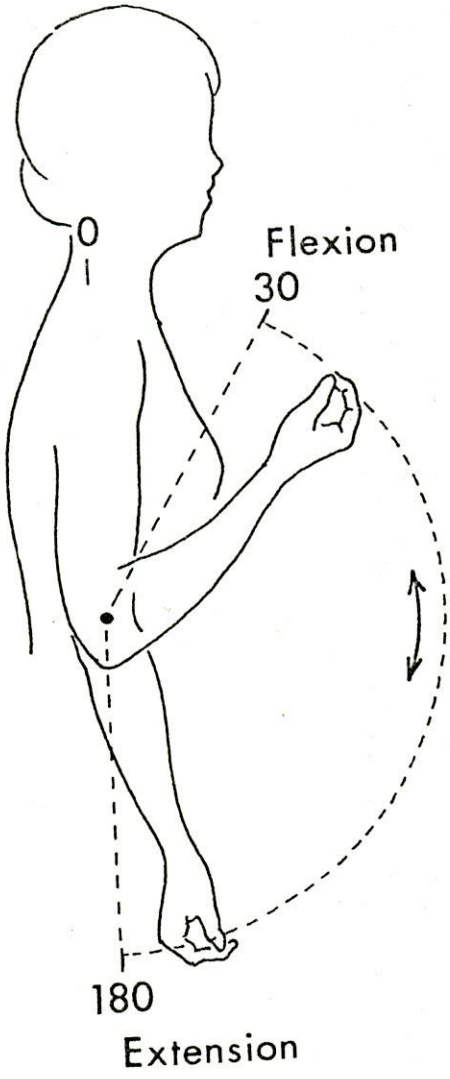
Forearm



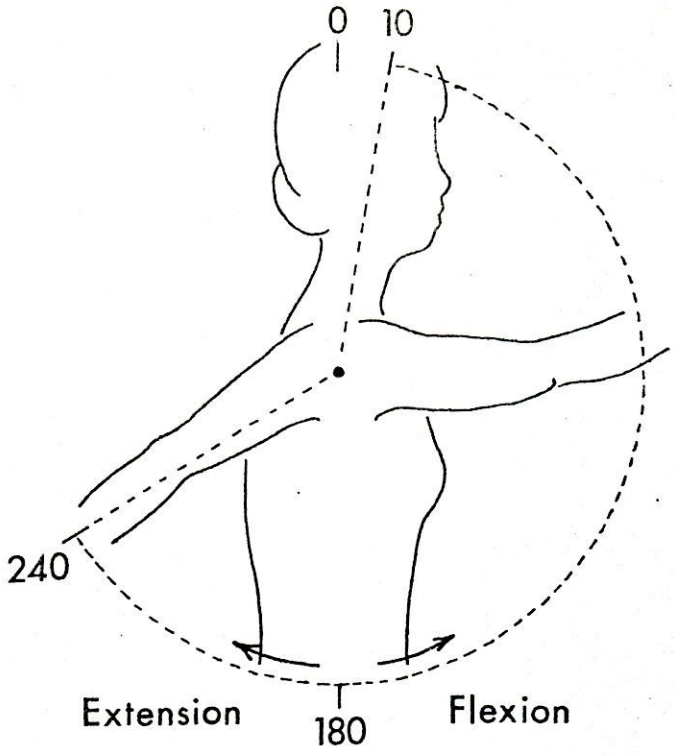
Forearm



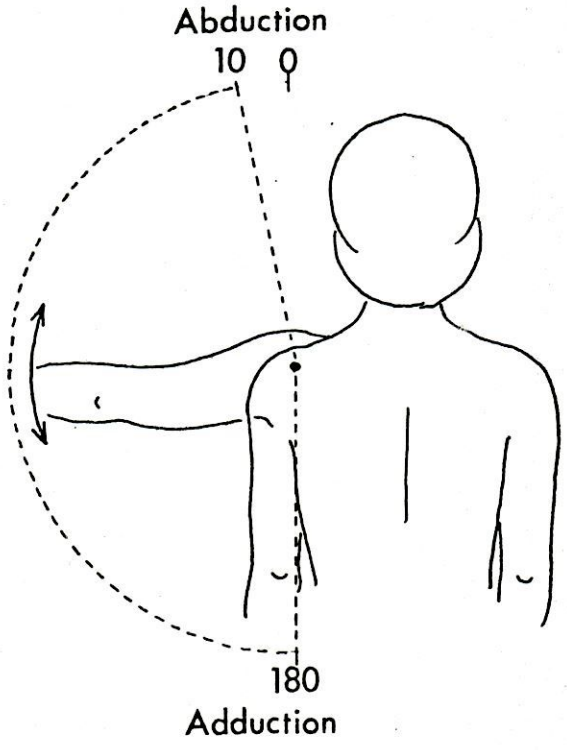
Wrist



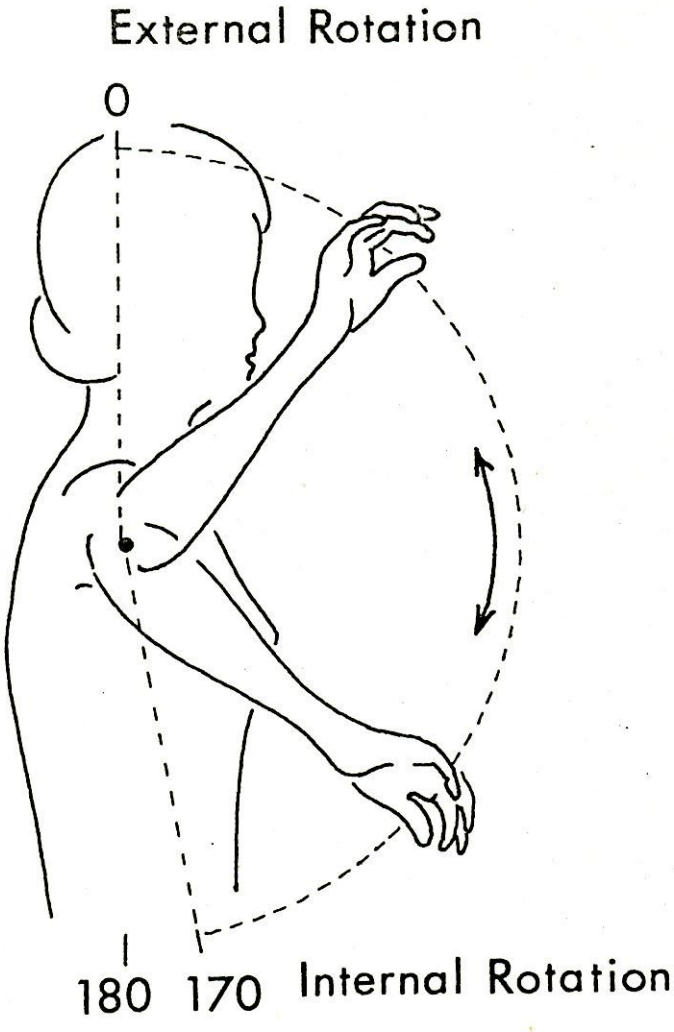
Elbow



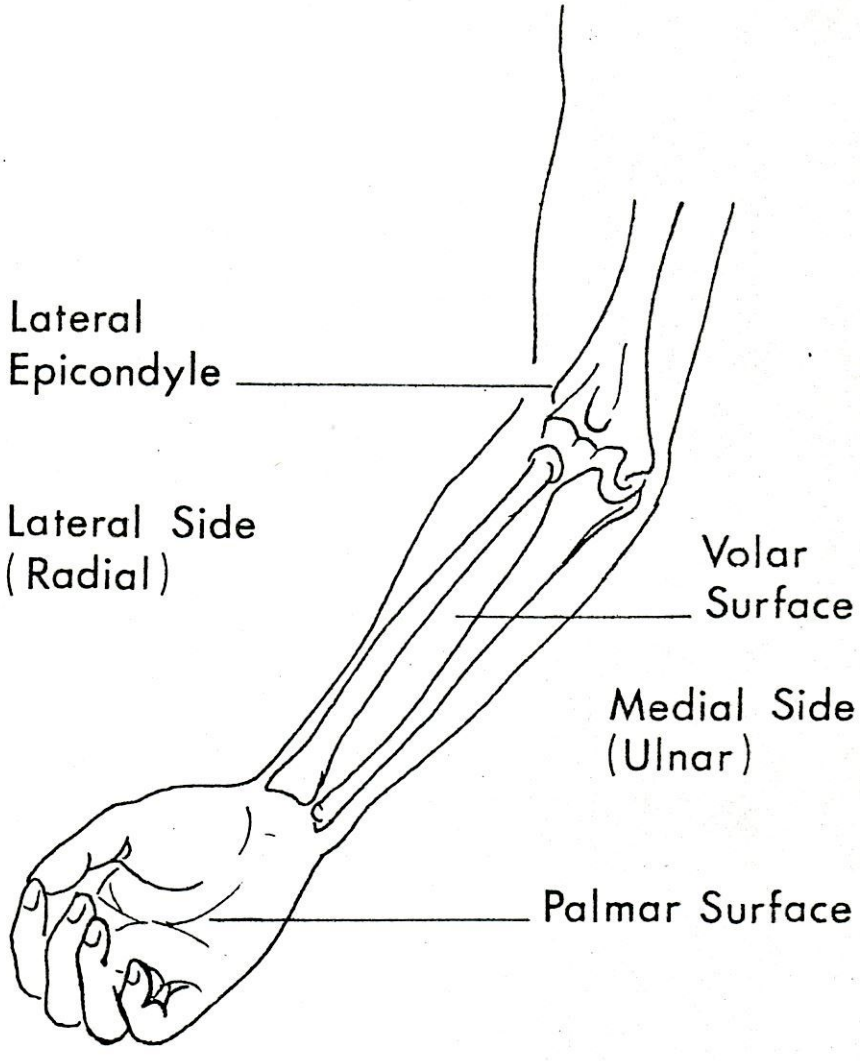
Shoulder

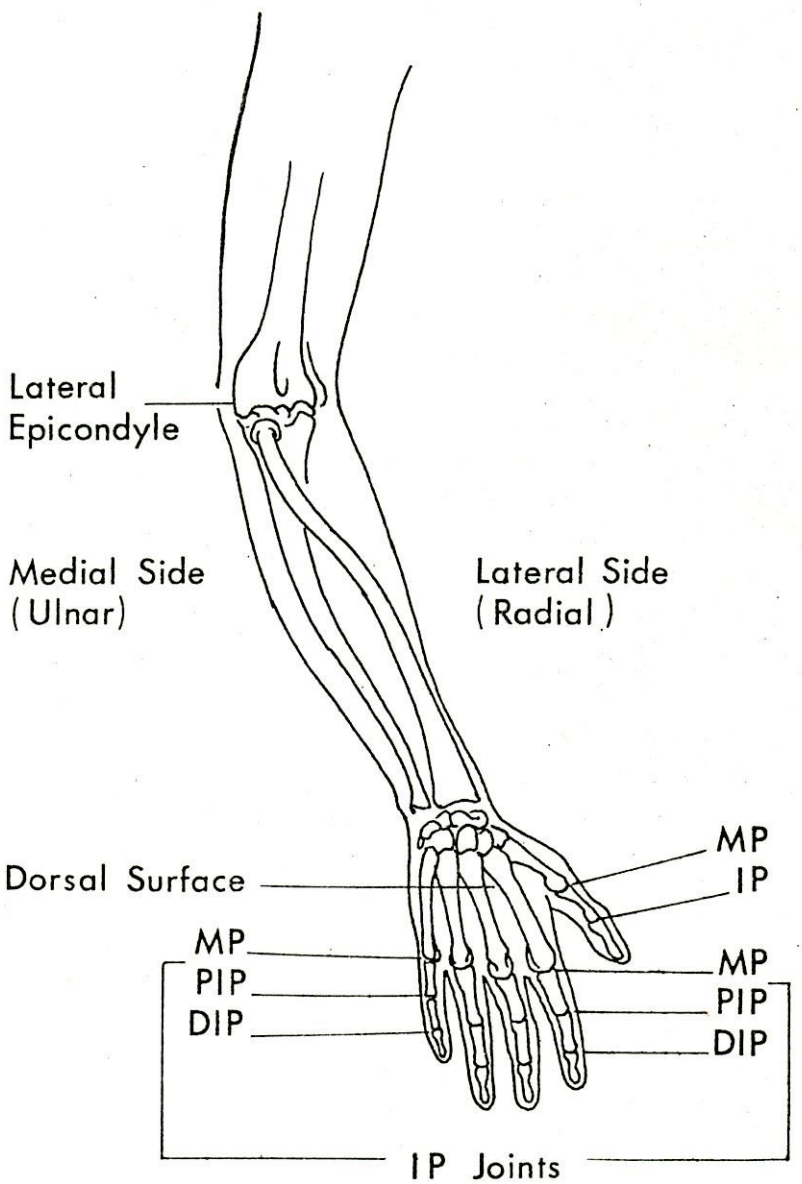


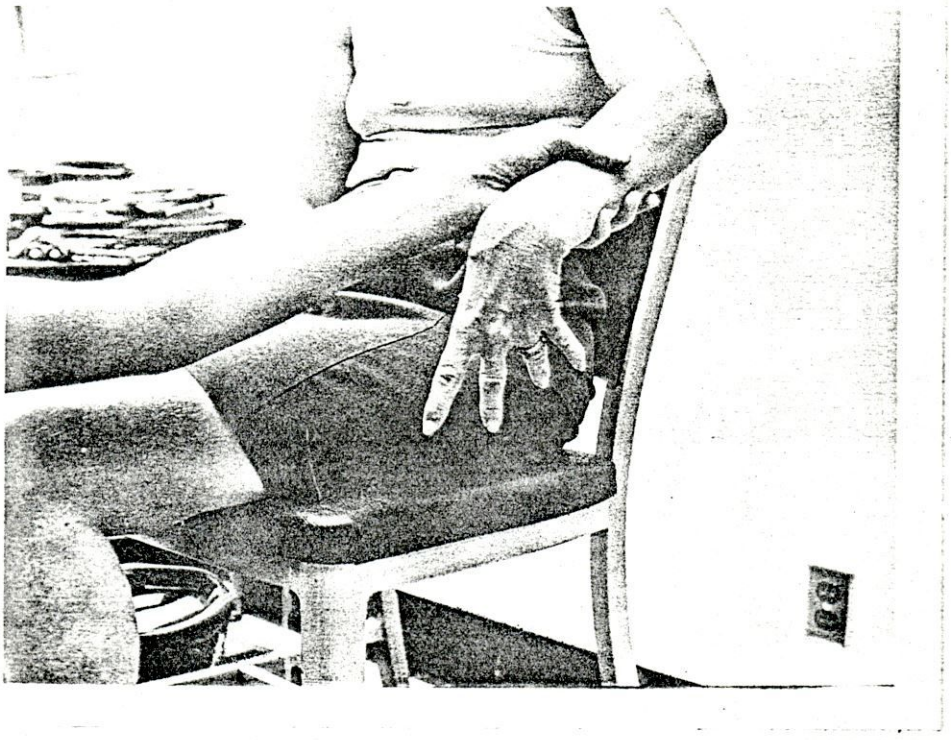
Shoulder



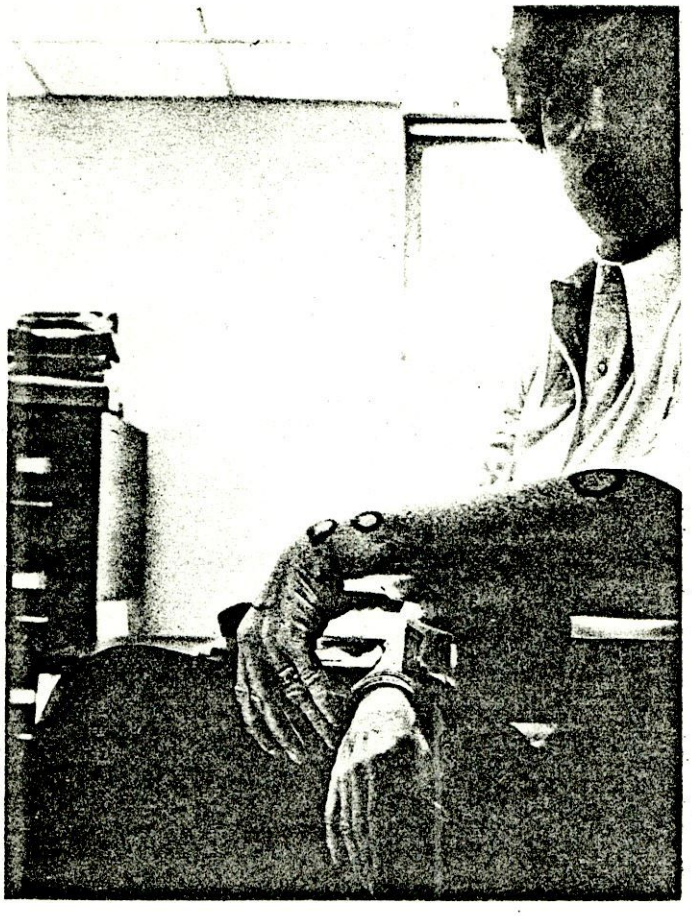
Shoulder


Lateral
Epicondyle
Lateral Side
(Radial)
Volar
Surface
Medial Side
(Ulnar)
Palmar Surface


Lateral
Epicondyle
Medial Side
(Ulnar)
Lateral Side
(Radial)
Dorsal Surface
MP
IP
MP
PIP
DIP
MP
PIP
DIP
IP Joints



Patient 3 at baseline: Illustration of the splaying motion of the fingers of the dystonic hand as Patient 3 attempts to keep her hand open.

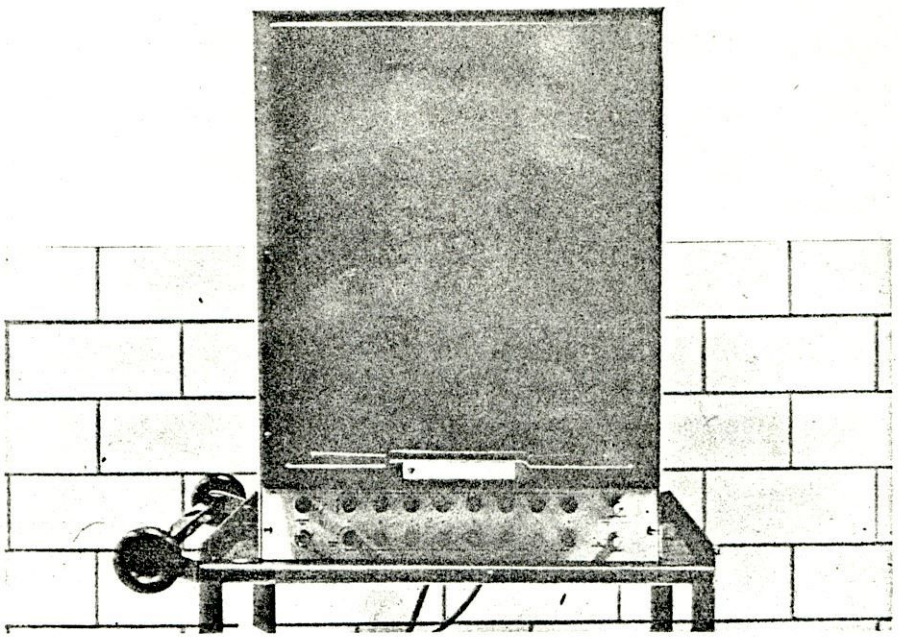


Patient 3 at the end of Phase I (PT): Illustration of Patient 3 relaxing the wrist and fingers of her dystonic hand. (Circles on skin are dye markings for electrode placements).

Patient 7 at the end of Phase I (BF): Illustration of Patient 7, who was apractic, flexing the elbow as a component of the stereotyped motion of shoulder adduction.

Patient 10 at the end of Phase II (PT): Illustration of Patient 10 in the process of "waiting" and concentrating on relaxing his biceps to achieve maximum elbow extension possible (approximately 160^0, and, at times, up to 170^0).

Patient 1 at the end of Phase II (BF): Illustration of Patient 1 concentrating and responding with great effort to achieve maximum wrist extension (an average of approximately 180^0) together with inhibition of wrist supination. (Circles on the skin are dye markings for electrode placement. The patient also had tatoos).



The EMG BF monitor oscilloscope for patient viewing of a dot (leaving a trace) moving up and down in proportion to averaged EMG activity. (Unfortunately, the TV was photographed after it had been dismantled from the EMG machine and signal averager equipment. It could not be conveniently reconnected to the equipment in order to photograph a picture containing a typical TV display.)

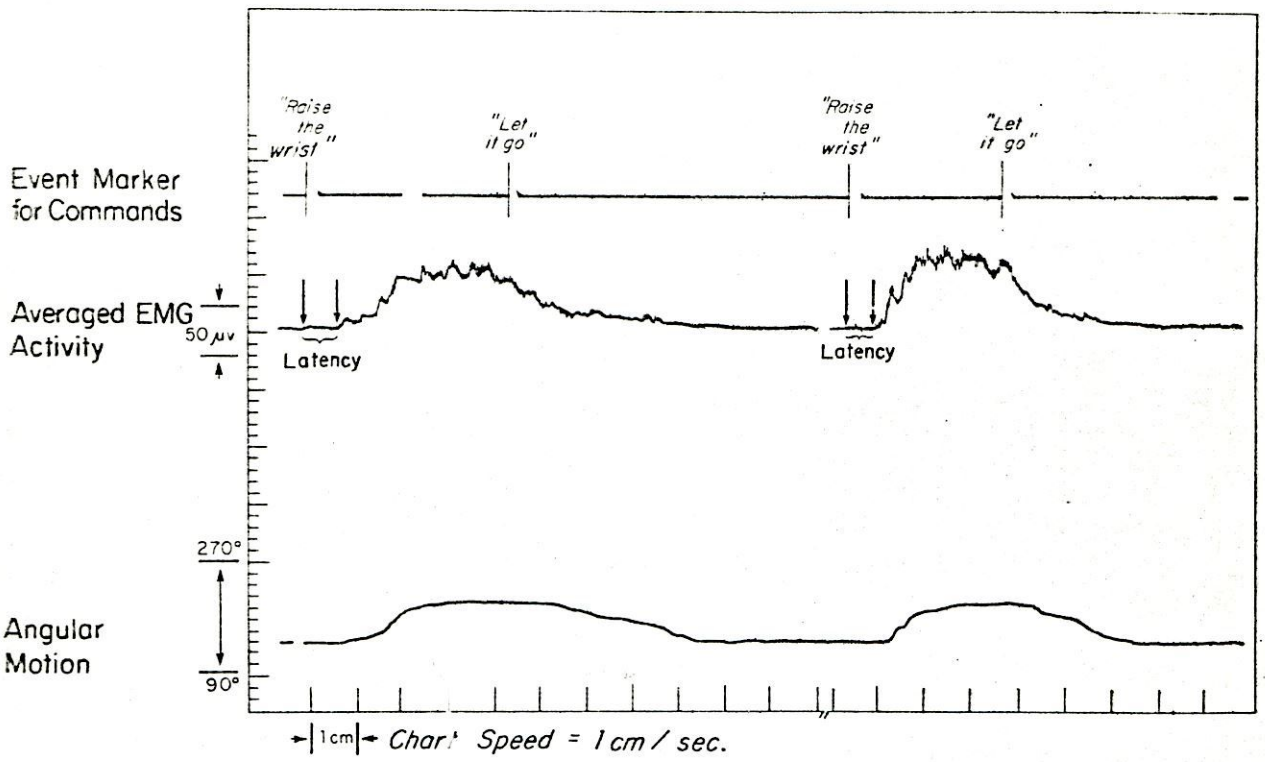


Illustration of the paper recorder raw data display on three channels (including the event marker channel for Latency, Part A).

Case Histories

A Note on Case Histories

The author would like to comment on how this study was accomplished. There was abundant observation and notetaking, the result of which is the individual case histories. These "cumulative records" of patient behavior are, it is felt, the true handle to what has been studied. It is hoped that the interested reader will refer to the case histories as the primary data source, as opposed to, e.g., statistical results. Statistical results, gloss over many important aspects of the learning process which are reflected, but not taken into account by, the quantitative parameters. The most direct information is to be found in case histories.

Note: The following was overlooked in the Case History reports of Patients 1, 2, and 8. The forearm of these patients was sometimes strapped to the working table during BF in order to assist posture and localization of responses to the target muscles. The restraint was removed as the patients improved.

Patient 1

MMPI

L	46
F	96
K	35
Hs	65
D	68
Hy	51
Pd	46
Mf	59
Pa	76
Pt	66
Sc	88
Ma	75
Si	69
Es	24

Code

8"69'271-'(59)^{6}3,24,4

Patient 1[36]

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	83
Verbal IQ (VIQ)	77
Performance IQ (PIQ)	92
WAIS Verbal Subtests:	
Comprehension	7
Mathematics	8
Digit Span	7
Vocabulary	4
WAIS Performance Subtests:	
Block Design	9
Picture Arrangement	8
Object Assembly	9
Porteus Maze Test	7.5
Trail Making Test, Part A	71"-0 errors
Trail Making Test, Part B	205"-3 errors
Bender-Gestalt Recall	5
Wechsler Memory Scale (WMS) Quotient	94
WMS Subtests:	
General Information	5
Orientation	5
Mental Control	7
Memory Passages	6
Digits Control	8
Visual Reproduction	7
Associates Learning	12
Aphasia Screening Test (AST)	8-1 verbal, 7 non-verbal errors
LH Tactile Formboard I, Standard RH	20"-10 objects placed
LH Tactile Formboard II, Blindfolded RH	210"-10 objects placed

---

[36] Interpretation of tests requiring proficiency in vocabulary for this patient (i.e., especially the WAIS Vocabulary subtest) is complicated by the fact that English was a second language for him and was relatively imperfect poststroke compared with his native tongue.

Patient 1, 54 yr old white male, 1 stroke (6 yrs, 1 mo poststroke)

Clinical Diagnosis:
(1) Hypertension
(2) Left heart strain
(3) Slight obesity
(4) Left middle cerebral artery thrombosis
(5) Right hemiparesis

Prior Handedness: Right

Prior Therapy: 1-1/2 mos; leg only

Comments: Dysgraphia

NEUROLOGICAL EXAMINATION

Description of Weakness:

Spastic right hemiparesis with moderate facial involvement. Hemiparesis most extensive distally in the right upper extremity with minimal flexion and extension at the wrist, slightly better, 1-2/5 for elbow flexion, with good elbow extension. 4/5 abduction at shoulder, 3/5 right hip, 4/5 knee, 3/5 (flexion) and 4/5 (extension), 2/5 ankle and toes, dorsi and plantar flexion. Unsustained ankle clonus. Right sided hyperreflexia and Babinski sign with increased tone. Arm hangs down as he walks but fingers are kept in mild spastic grasp.

Description Sensory Impairment:

Pin: Slight decrease of sensation to pin prick on right side.

Touch:	Intact
Vibration:	Intact
Position Sense:	Intact
Two Point Discr:	Increased to 6 mm on right hand.
Graphesthesia:	Intact
Stereognosis:	Impaired on right hand.

Aphasia:

Normal

Apraxia:

None

Comments: None

Training Objective: Wrist extension

Phase I: PT

Phase II: BF

Introduction:

Patient 1 had a right spastic hemiparesis. His upper extremity hung at his side, fingers flexed. Spontaneous movements were non-functional, consisting of a stereotyped pattern of shoulder elevation, abduction, and internal rotation, elbow flexion, with some wrist extension and flexed fingers. He was able to flex the elbow only insofar as the rest of the pattern occurred. Apparent triceps strength on command was quite good. Movement to the specific command to "raise the wrist" resulted in a range of wrist extension (e.g., Part A) from

136^0 to 149^0 in the standard position with forearm resting on the table and hand flexed over the edge. Wrist extension from this position involved approximately 40^0 of supination from the fully pronated position, some elbow flexion, and external rotation of the shoulder towards termination of the movement. Wrist extension could also reach 180^0 if he held the shoulder flexed forward and the elbow extended at straight line, or when wrist extension was allowed to occur as part of a stereotyped movement. He could not move the fingers, except by tenodesis, nor could he flex the shoulder. The patient exhibited a great deal of clonus in his movement which was readily inhibitable by voluntary attention and slower motion.

Patient 1 lived with his wife who worked a full-time job. He did everything for himself. At the onset of training, he considered his right arm as "heavy", "asleep", mentioned that it should be "cut off" and had little hope of improvement. The patient was willing to try with a cautious, realistic attitude toward probable improvement. A combination of imposed discipline and noticeable progress readily overcame his reluctance to imagine improvement as feasible. Patient 1 ceased to mention the heaviness and became more energetic in improving his function. He practiced a great deal at home and subtle changes were always evident. He typically shut his eyes during PT and experimental tests in order to concentrate on movements. He would be quite fatigued and perspiring at a session's end but his endurance continually increased. He remarked once after BF practice that it felt like "pins and needles" in his arm which had previously been referred to as "asleep" and often noticed that his limb got "hot" distal to the elbow after BF practice. This man had a definite experience of his upper extremity "coming alive".

Phase I: PT -

The target movement for Patient 1--wrist extension--was complicated by a number of undesired features. He supinated a great deal during extension and could not isolate wrist extension from the entire gross pattern described above. Thus, focus was on isolating wrist extension, and elbow and shoulder flexion, together with trying to eliminate supination during wrist extension. Facilitation and resistance was applied to the wrist extensors. Wrist extensor activity was maximal when the synkinetic movement described above was allowed to come into play. Thus, as training for isolated wrist extension without supination began after baseline, Patient 1 was performing at somewhat less than his baseline level on dependent measures. As PT progressed, his overall gains were tremendous, however. He could flex the shoulder with elbow extended from a baseline of 180^0 to approximately 100^0. Elbow flexion also increased although Patient 1 was never able to flex without abducting the shoulder. Wrist extension stayed the same for averaged EMG but range increased (e.g., Part A) from approximately 13^0 at baseline to 24^0. Elbow flexion during wrist extension was inhibited but supination at the wrist could not be inhibited. Clonus was nonexistant as long as Patient 1 did not move excitedly. The length of time Patient 1 could practice wrist extension under applied resistance was continually increasing.

Phase II: BF -

Progress on wrist extension essentially continued as above with one major improvement. By weeks 3 and 4 of BF, Patient 1 had eliminated supination during wrist extension to a great degree. How this was

accomplished is unknown to E except that, as before, frequent attention was given supinator inhibition. During BF, however, this attention was solely verbal. The final two tests during BF training were recorded from wrist extension movement with minimal supination. At week 3 of BF, Patient 1 remarked "I feel something different today", referring to the way he moved without supination. This change was accompanied by a disproportionate increment in endurance level over prior sessions. Also, interestingly, the EMG voltage attained by Patient 1 on tests during the BF phase, where he had no exteroceptive feedback available to him, was approximately half the voltage he attained as he watched and listened to BF signals during BF training.

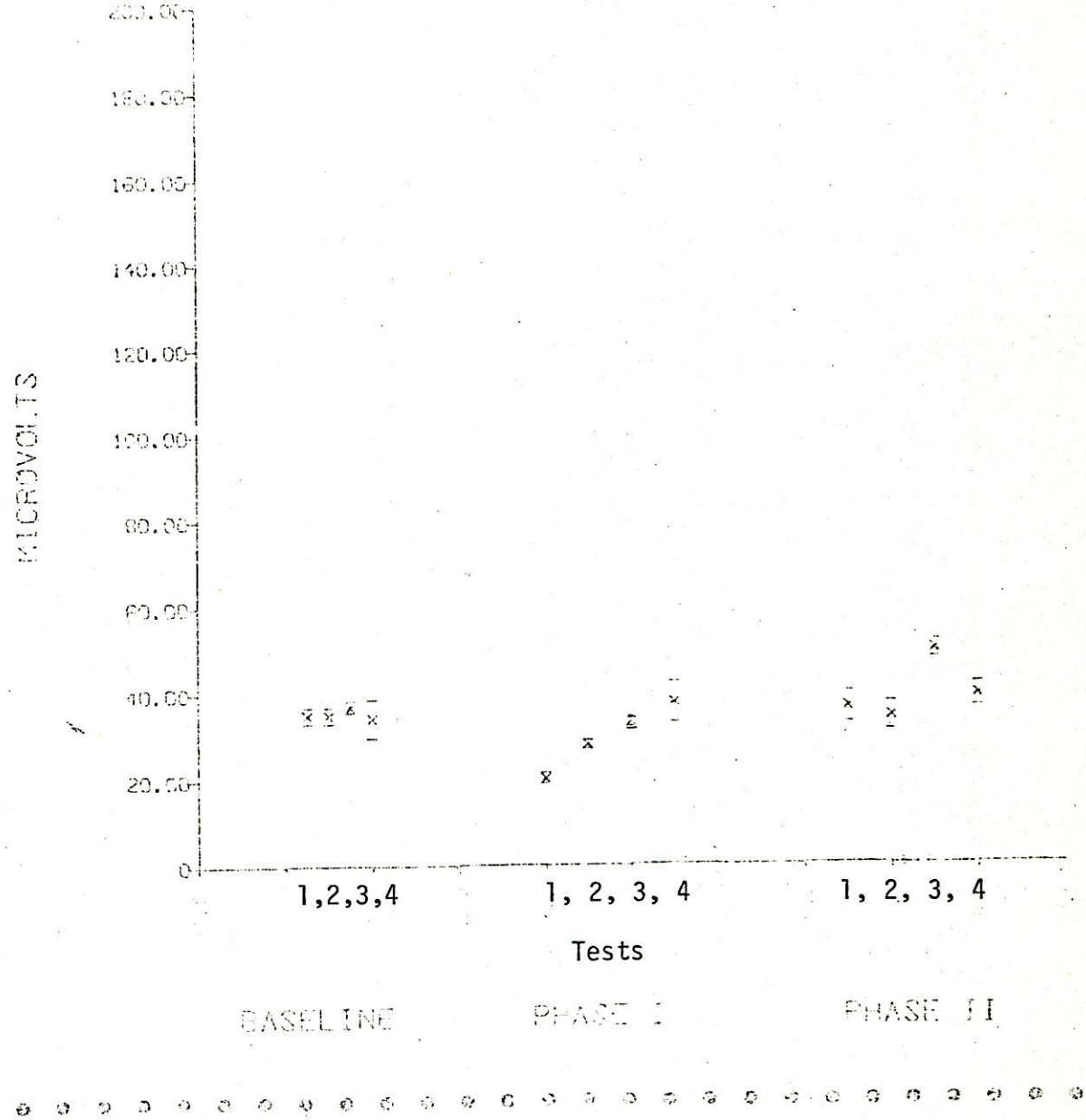
Comments:

At the end of overall training, Patient 1 also had some finger flexor control--an increase from not being able to grip a dynomometer at all prior to training, to gripping it at 12.2 kilograms at the end of training.

Patient 1 is missing a score for test 1 of Phase I on graphs of angular motion because of a goniometer equipment failure.

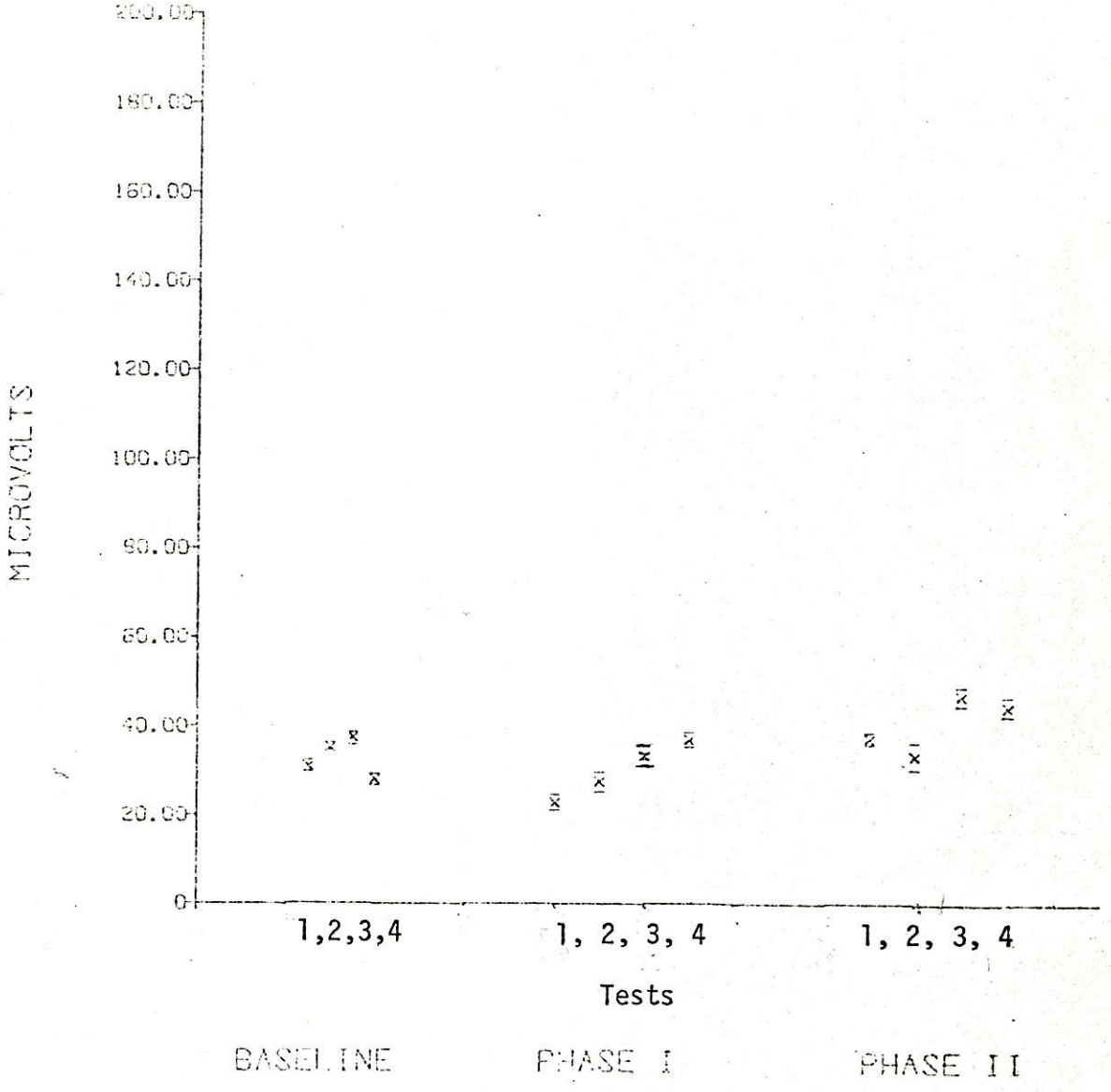
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 1 PART A (DPP, LINE 228 - 221)

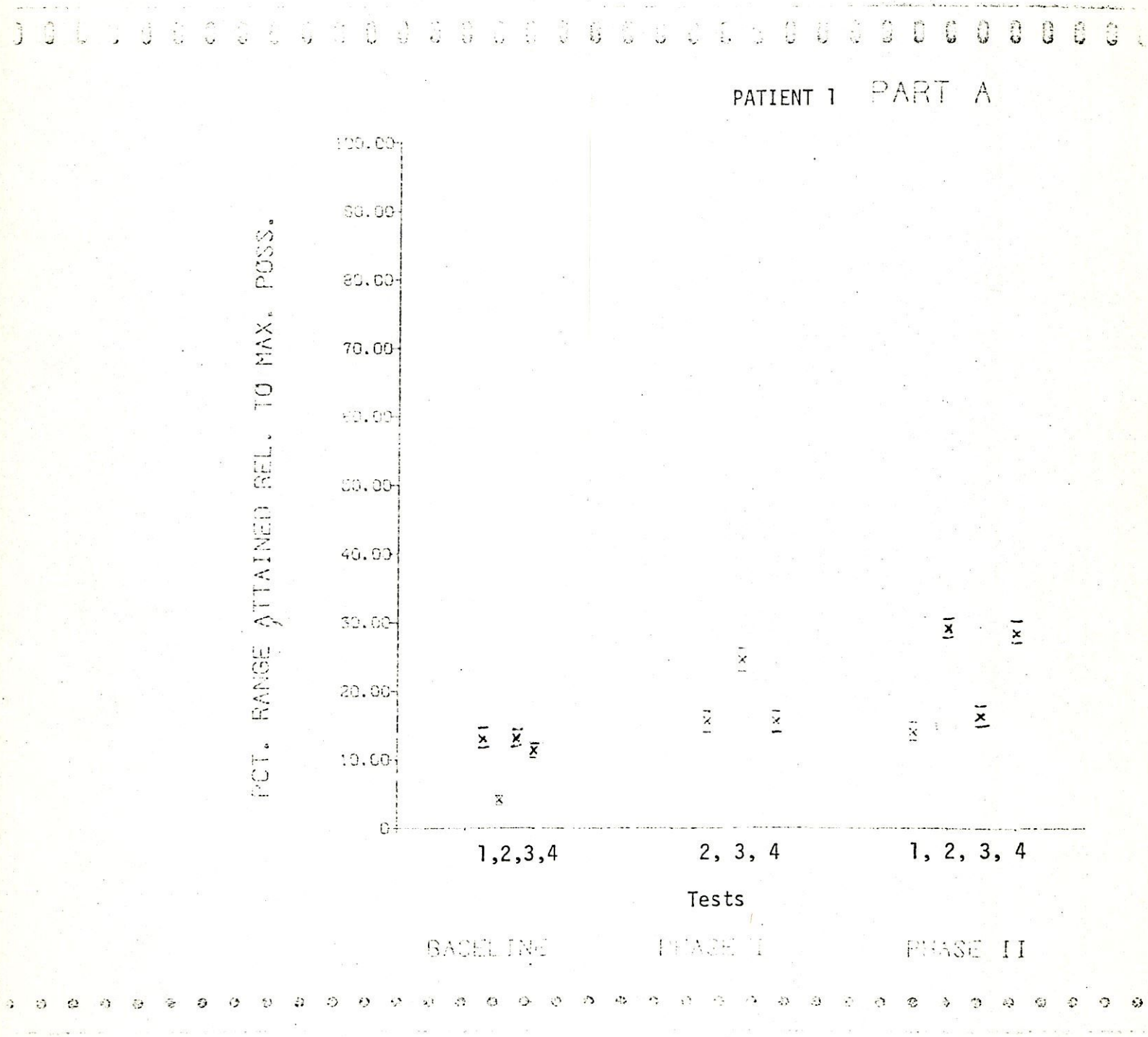


Averaged EMG Activity in Microvolts (SE indicated)

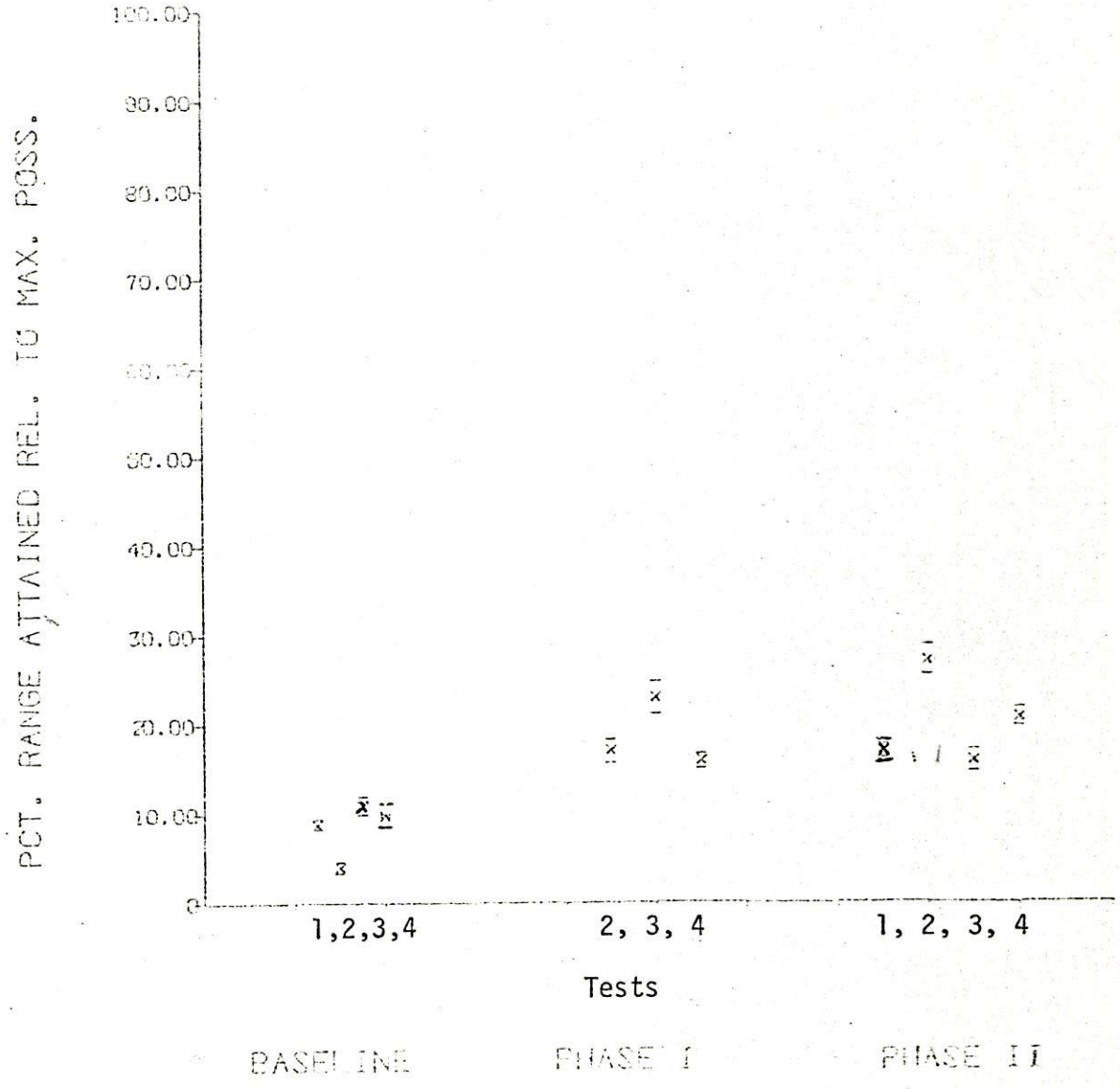
PATIENT 1 PART B (OPP. LIMB 203 , 249)



Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

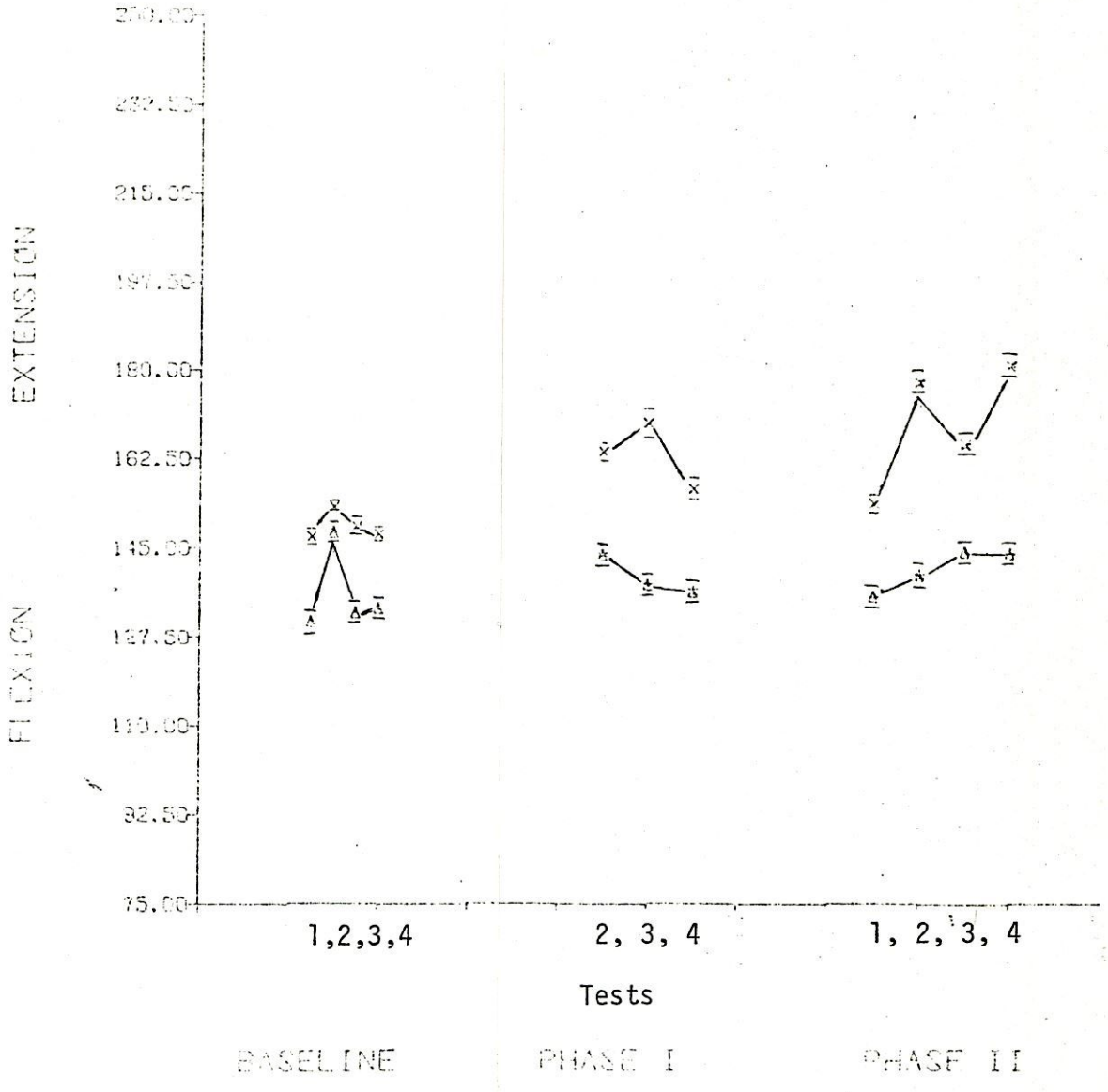


Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)



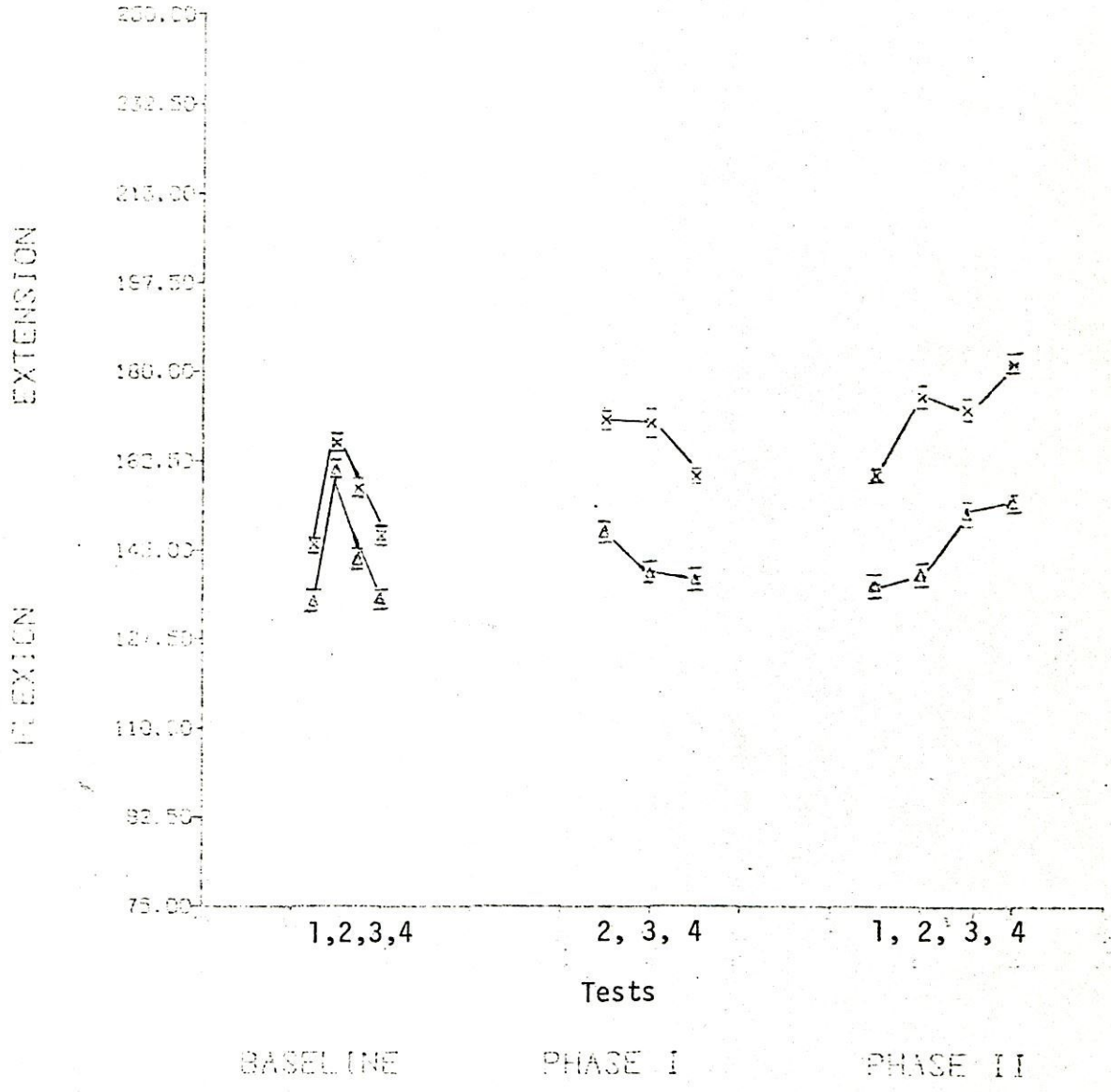
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 1 PART A



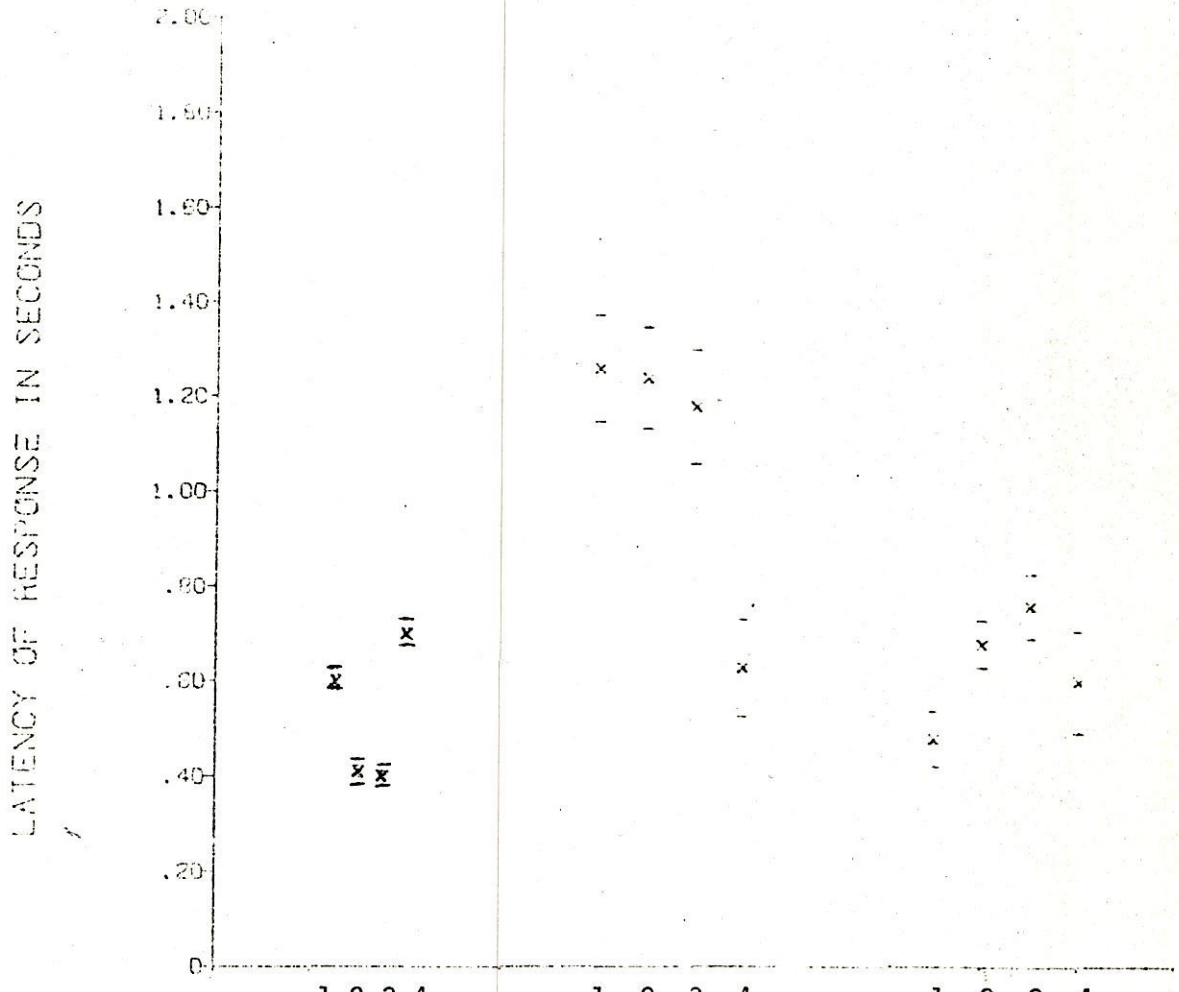
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 1 PART B



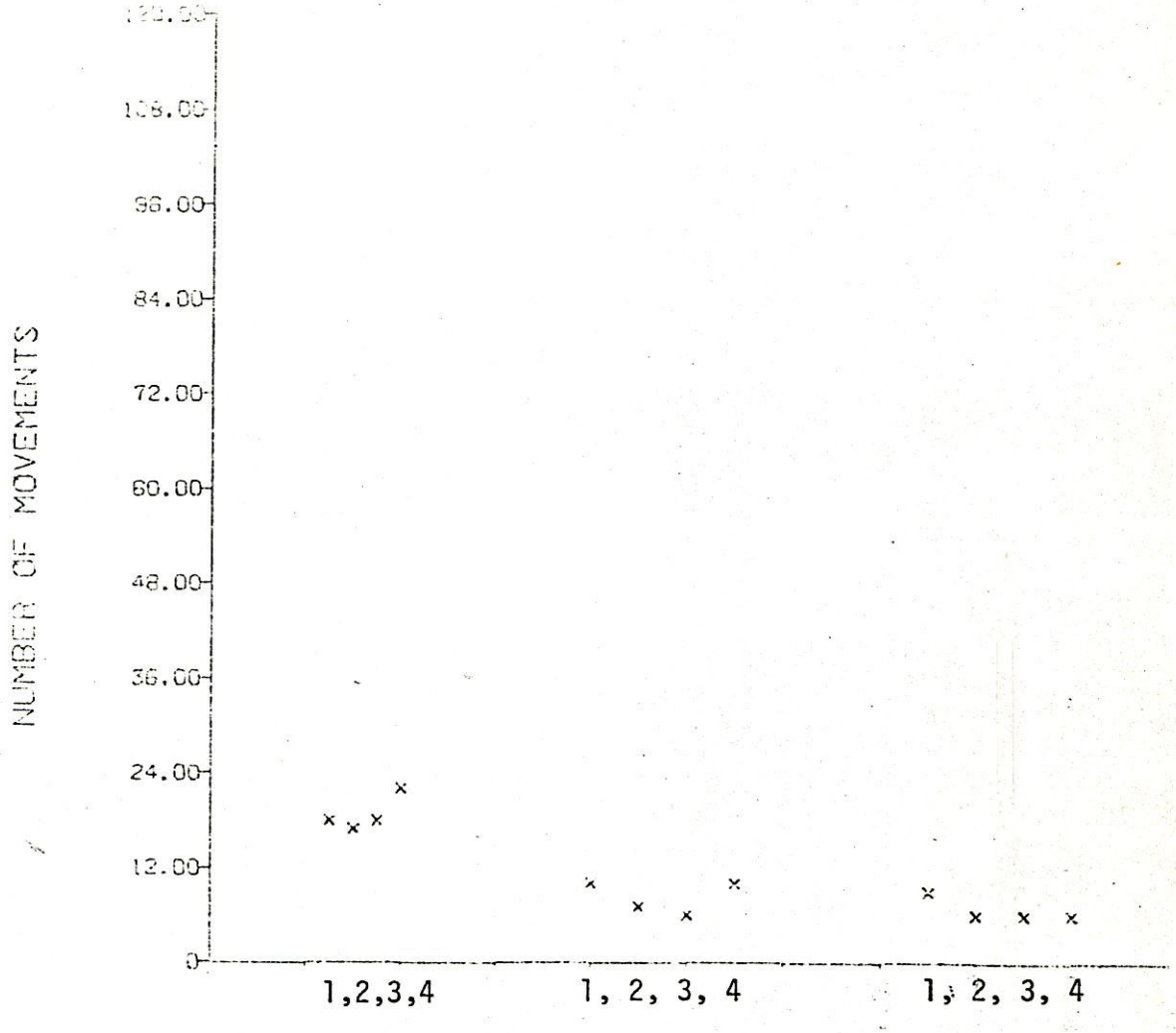
Latency (SE indicated)

PATIENT 1 PART A (AFF. LIMB .4 , .3)



Repetition of Movement

PATIENT 1 PART B (OFF. [illegible] 115 . 125)



Patient 2

MMPI

L	44
F	60
K	62
Hs	82
D	80
Hy	62
Pd	53
Mf	47
Pa	62
Pt	60
Sc	80
Ma	35
Si	55
Es	56

Code

128"367-"9(47)^{0}2,7,19

Patient 2

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	92
Verbal IQ (VIQ)	86
Performance IQ (PIQ)	100
WAIS Verbal Subtests:	
Comprehension	8
Mathematics	5
Digit Span	8
Vocabulary	11
WAIS Performance Subtests:	
Block Design	8
Picture Arrangement	10
Object Assembly	11
Porteus Maze Test	16.5
Trail Making Test, Part A	146"-0 errors
Trail Making Test, Part B	365"-3 errors
Bender-Gestalt Recall	6
Wechsler Memory Scale (WMS) Quotient	96
WMS Subtests:	
General Information	5
Orientation	5
Mental Control	2
Memory Passages	10
Digits Control	8
Visual Reproduction	13
Associates Learning	7.5
Aphasia Screening Test (AST)	4-3 verbal, 1 non-verbal errors
LH Tactile Formboard I, Standard	29"-10 objects placed
RH	134"-10 objects placed
LH Tactile Formboard II, Blindfolded	300"- 7 objects placed
RH	

Patient 2, 56 yr old white male, 1 stroke (3 yrs, 1 mo poststroke)

Clinical Diagnosis: (1) Thrombosis, left carotid artery
(2) Right hemiparesis

Prior Handedness: Right

Prior Therapy: 1 yr, 1 mo total; arm and leg

Comments: Memory was good. Ability to express what was reasoned in a syntactically appropriate manner was very poor. Patient lost math ability which was quite good prior to stroke.

NEUROLOGICAL EXAMINATION

Description of Weakness:

Spastic right hemiparesis, worse in leg, minimally involving the face. Strength in right upper extremity is 4/5 except for elbow flexion and arm extension which are 3/5. Spasticity limits elbow and wrist movement particularly on extension. Much greater weakness in right knee extension and all movement of the foot and toes which show 2/5 strength. DTRs are increased on the right with a Babinski sign but no clonus.

Description Sensory Impairment:

Pin: Minimally decreased sensation to pin prick over right foot.

Touch: Intact

Vibration: Intact

Position Sense: Decreased position sense in right foot.

Two point discr: Intact

Graphesthesia: Intact

Stereognosis: Intact

Aphasia:

Mild anomia.

Apraxia:

None

Comments: None

Training Objective: Wrist extension

Phase I: PT

Phase II: BF

Introduction:

Patient 2 had a mildly spastic hemiparesis. The tone of his left upper extremity was normal. He was better at all movements of the upper extremity than any other patient. However, his spontaneous movements did consist of an hemiparetic pattern of shoulder elevation, abduction, and internal rotation, supination of the forearm to 0^0, fingers loosely flexed, thumb adducted. He had a fair degree of finger dexterity particularly in the thumb and index fingers. With the limb resting on the table, maximal wrist extension was (e.g., Part A)

approximately 200^0 at baseline, and invariably included supination to almost 0^0, elbow flexion, shoulder abduction, and a shift of body position with Patient 2 leaning heavily on his hemiplegic side over the table. Wrist extension averaged EMG and range were fair--(e.g., Part A) approximately 83 μv and approximately 54^0, respectively. Range, Part B, tended to be less than range, Part A, as Patient 2 tended to both extend and relax the wrist less in the former (Rate) vs. the latter (Command) situation. Patient 2 had a slight equilibrium problem in his movements due to poor coordination and a "vague" position sense. Although position sense in the upper extremity was assessed as intact, the patient did not seem to be keenly aware of this sensation. He referred to his paretic limb as having "one half the feeling" and not really feeling his affected side as he did his contralateral one. Without vision of his limb, he had a poor appreciation of wrist supination or shoulder abduction. Patient 2 liked to look at his wrist during wrist extension. Of those studied, this patient was the most "advanced" in terms of motor return. He could flick light switches and hold bottles with his right hand, as well as use both hands to put on his belt and tie his shoes.

Patient 2 lived with his wife who worked all day. He attended to himself completely. The most severe part of his loss was mental--a difficulty in name finding and expressing concepts with appropriate syntax and choice of words at levels above trite discourse. Also, it took several different explanations of one point for him to truly understand an idea, yet once he understood, his comprehension and retention was very good. He was in good health physically. Although motivated

to learn, he practiced only a poor to fair amount at home as he had a good deal of company staying at his house during the weeks of the study.

Phase I: PT -

Patient 2 was given a lot of resistance training in both the wrist extensors and flexors. There were also repeated and somewhat successful attempts to train wrist extension without supination. Other movements practiced were shoulder and elbow flexion without abduction. Patient 2 viewed himself in a mirror at home to try to eliminate supination or abduction in the above movements. Both of the latter flexion movements improved in range and abduction was decreased a great deal from approximately 110^0 to 160^0. Shoulder flexion increased dramatically--from approximately 135^0 to 30^0 (with abduction supervening at approximately 90^0 of flexion). For wrist extension, averaged EMG increased (e.g., Part A) from approximately 83 μv at baseline to approximately 132 μv, and range (Part A) increased from approximately 54^0 at baseline to approximately 67^0. Although not exhibiting as great a difference as at baseline, range continued to be somewhat less during Part B, the Rate, than Part A, the Command situation. Supination patterns during wrist extension could not be inhibited but there was more focus on shoulder abduction during this phase of training.

Phase II: BF -

Wrist extension, averaged EMG, and range remained about the same as during PT, with one critical difference to be described below: the patient was constantly reminded to "try not to turn your wrist". (This

verbal instruction, along with more vigorous instructions to eliminate abduction, was a theme in PT too.) One day during the second week of BF, the patient produced an isolated wrist extension without supination while observing the TV display. E remarked on the difference. The patient was now able at times (especially at the start of sessions) to extend the wrist without supination. After several attempts, supination would return to the movement. It is surmised that the success at eliminating supination could be due to general increased concentration and increased attention given the supination problem during BF, as well as E's instructions noted above, and other instructions to, for example, sit straight while practicing, or, for example, to not abduct the elbow over the table, etc. The patient first claimed he was not able to feel absence vs. presence of supination without vision of his hand. However, as he became successful an increased number of times, this comment disappeared. The patient remarked instead that it takes a while to learn "how to concentrate", but that "I'll get there". When he began to appreciate presence vs. absence of supination, Patient 2 said "I believe I got the message to keep this muscle loose", and touched the wrist supinator area of the forearm.

The last three tests of BF were taken on wrist extension without supination. Because of this, the first of these three tests showed a drop in range. The last two tests, however, showed range approximating values similar to those where supination was a component of wrist extension in testing. Interestingly, the last day of BF the patient wanted to try shoulder flexion for E. Upon trying, he could not do it at all whereas he had done it up to 20^0 during PT. This surprised him a great deal, since he was still practicing shoulder flexion at home.

Comments:

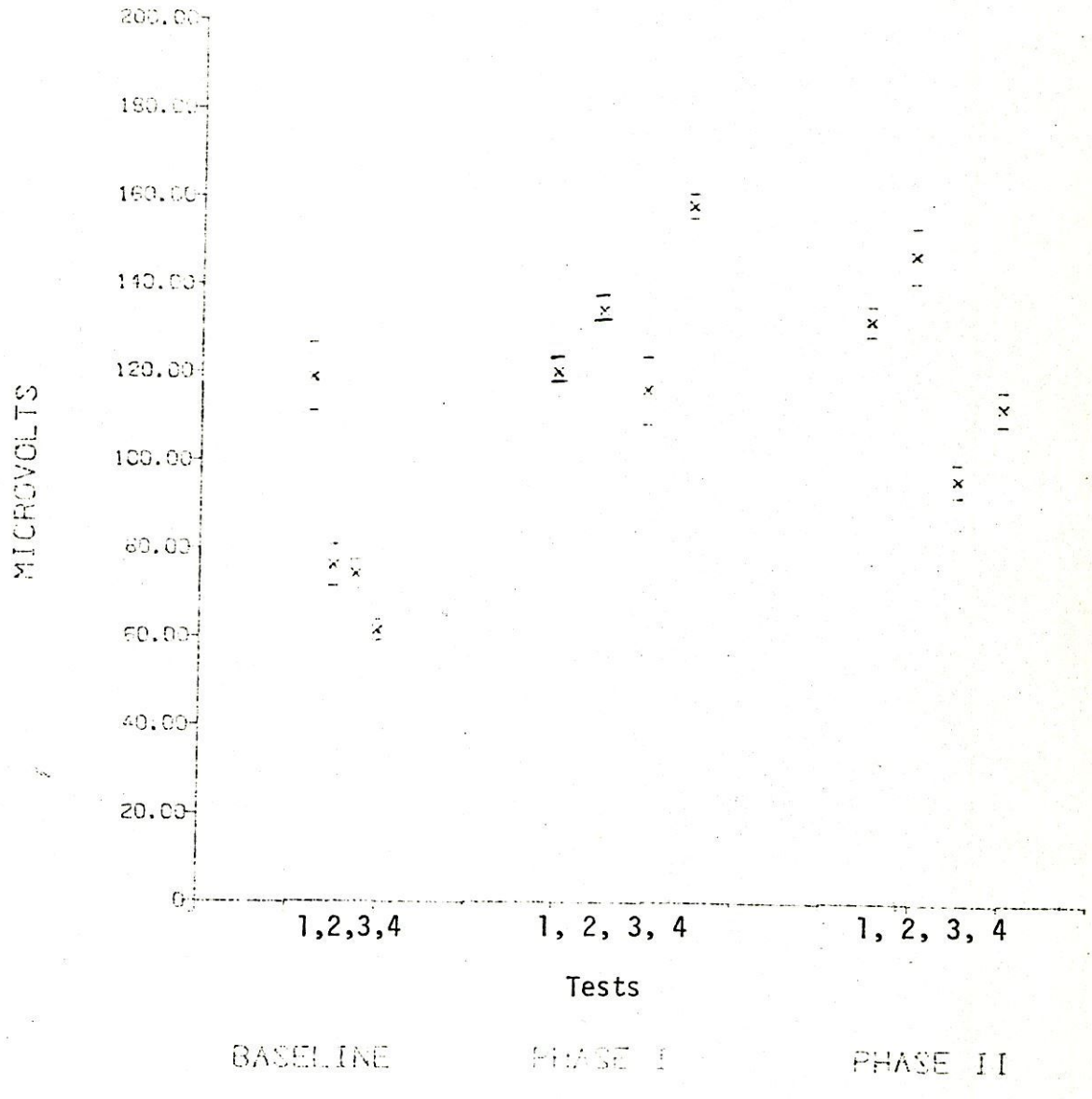
When Patient 2 demonstrated greater increments in the averaged EMG during either PT or BF, he did not experience different "feelings" in his arm. This lack of subjective experience could be due to a large "just noticeable difference" (JND) at an already established, moderately good level of function.[37] Basic "function" was not lacking. Refinement of "function" to skill would be a more appropriate way of viewing Patient 2's training.

---

[37]The same comment applies to Patient 4.

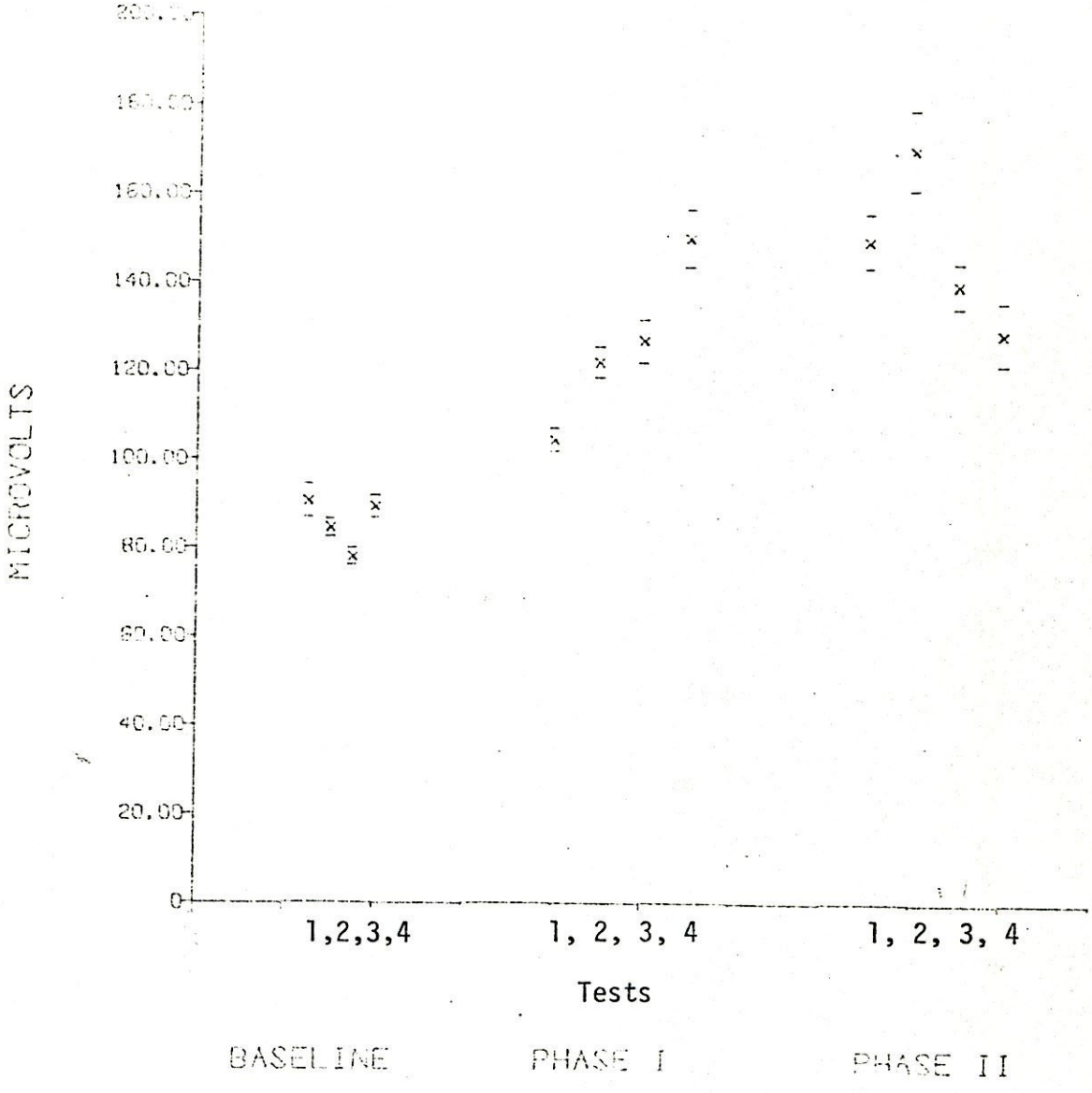
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 2 PART A (OPP. LIMB 262 , 273)

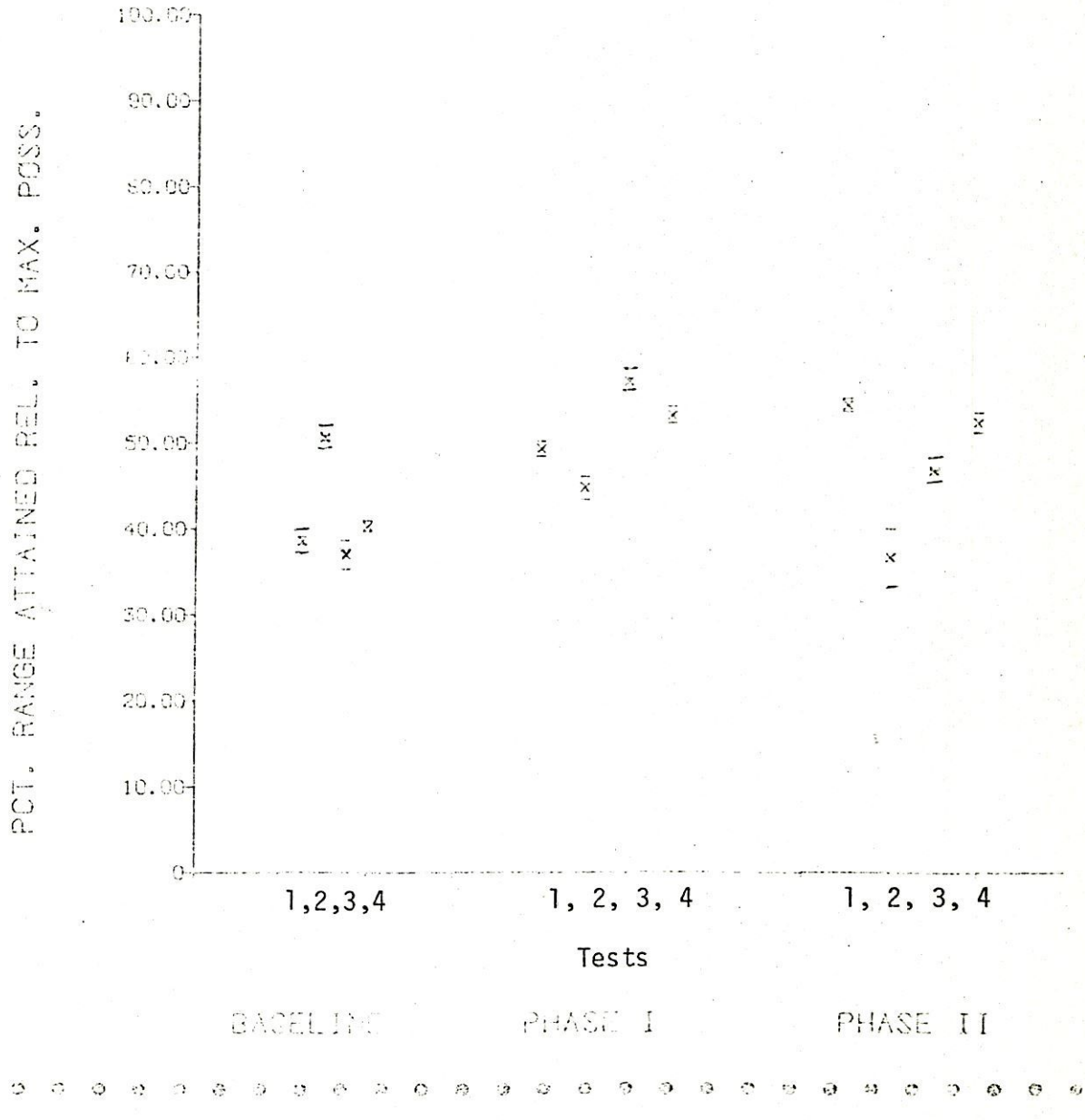


Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 2 PART B (UPP. LIMB 243 , 216)

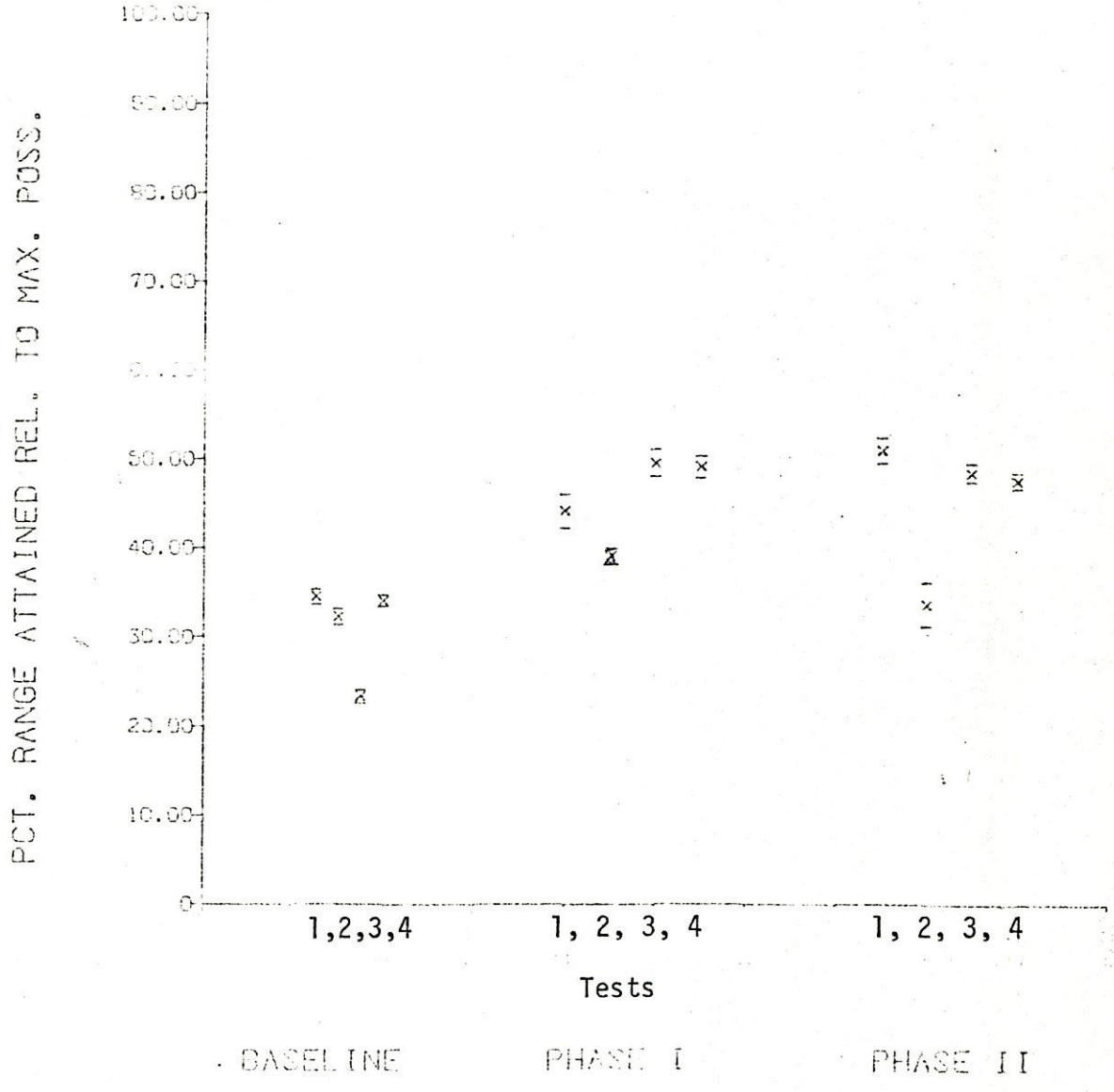


Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)



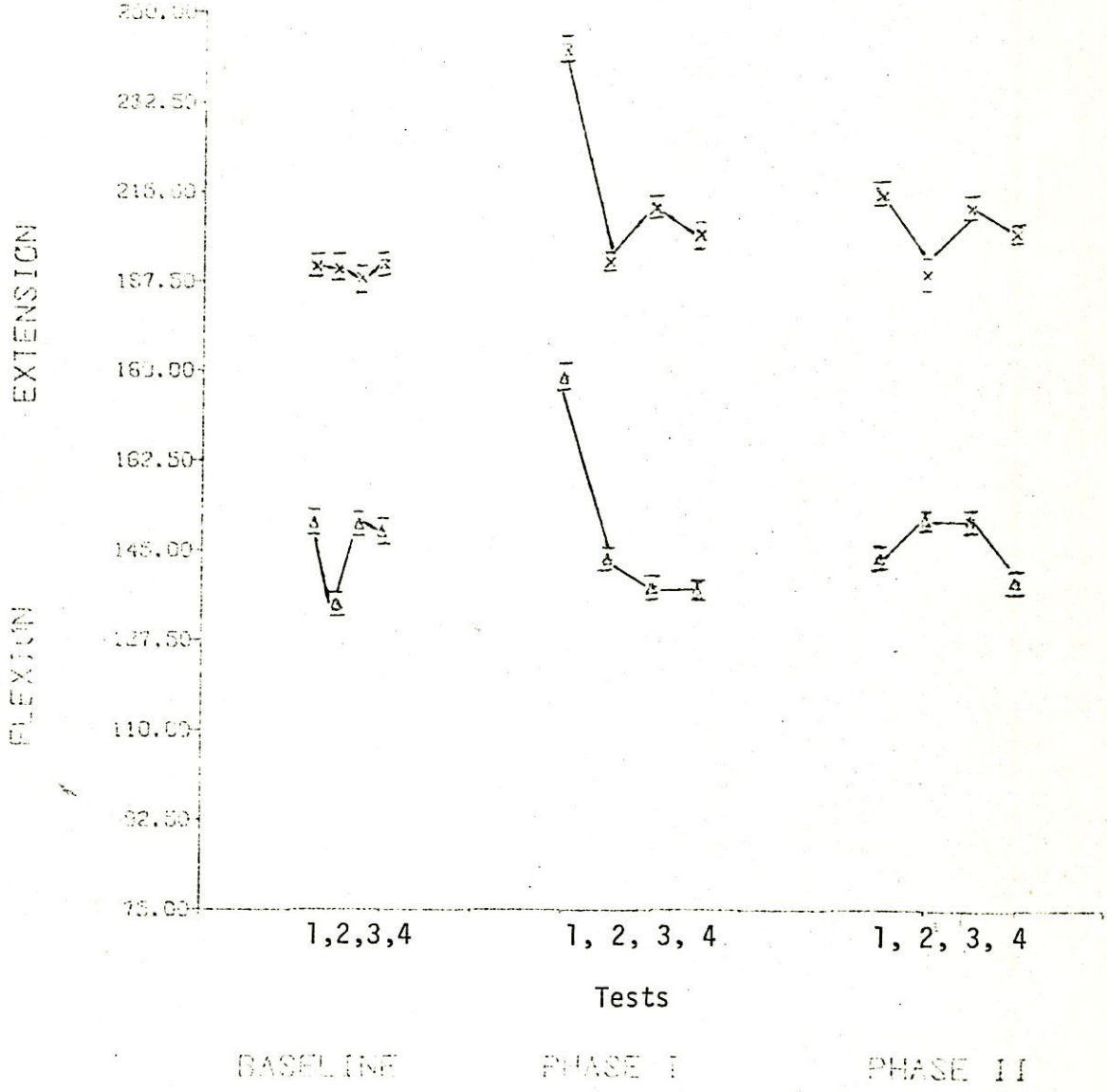
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 2 PART B



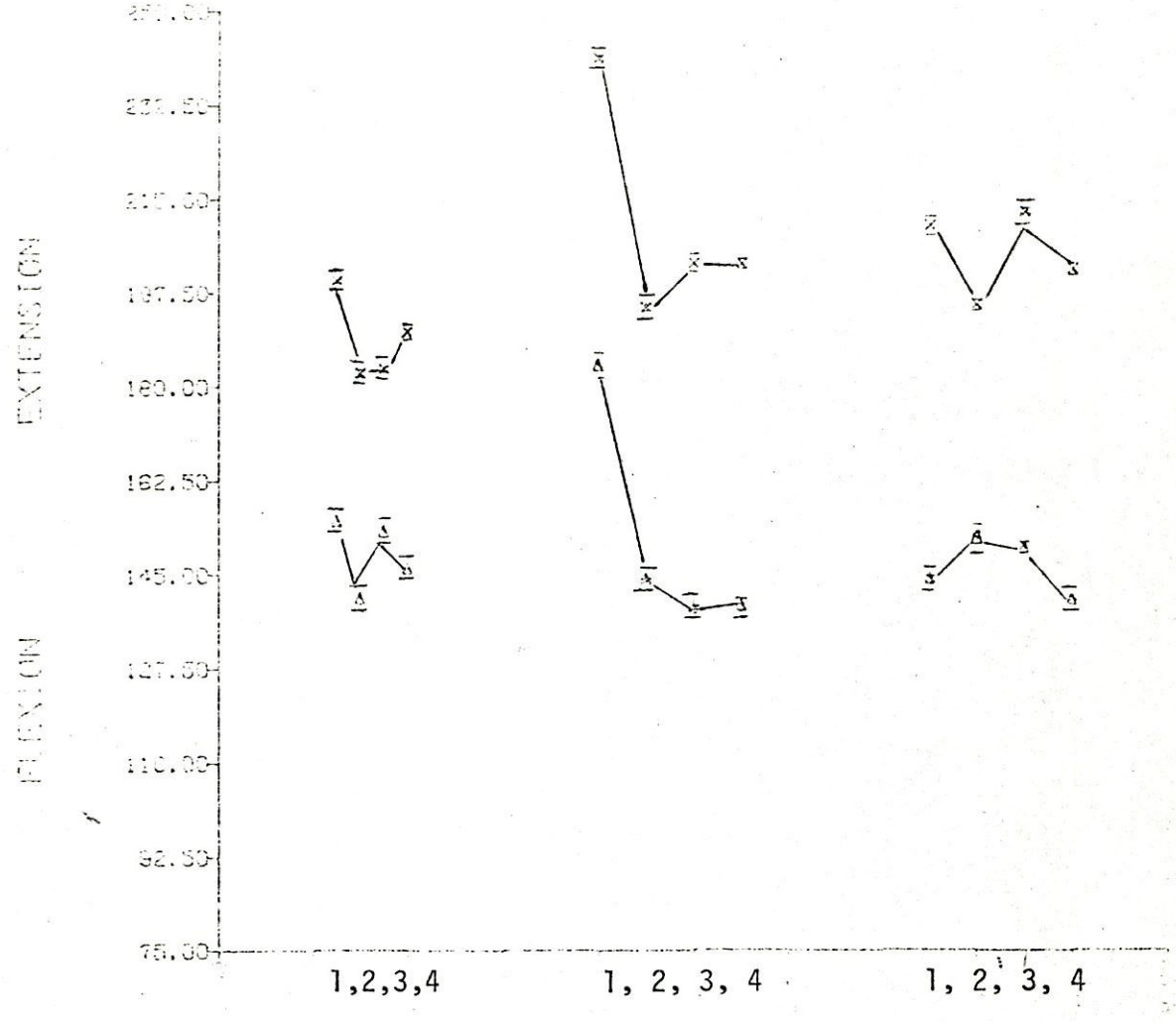
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 2 PART A



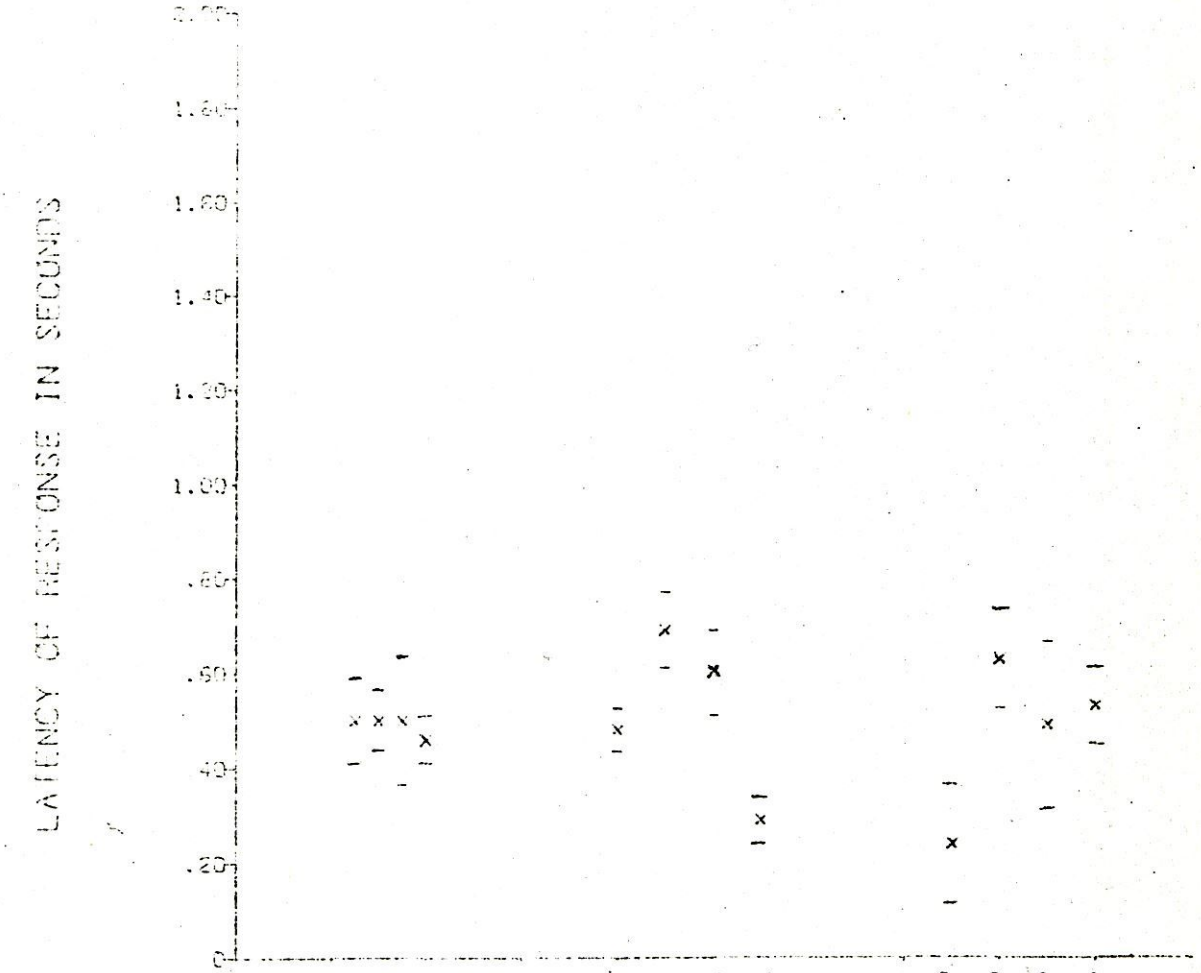
Extension ($\bar{X}$) and Flexion ($\bar{\Delta}$) in Degrees (SE indicated)

PATIENT 2 PART B



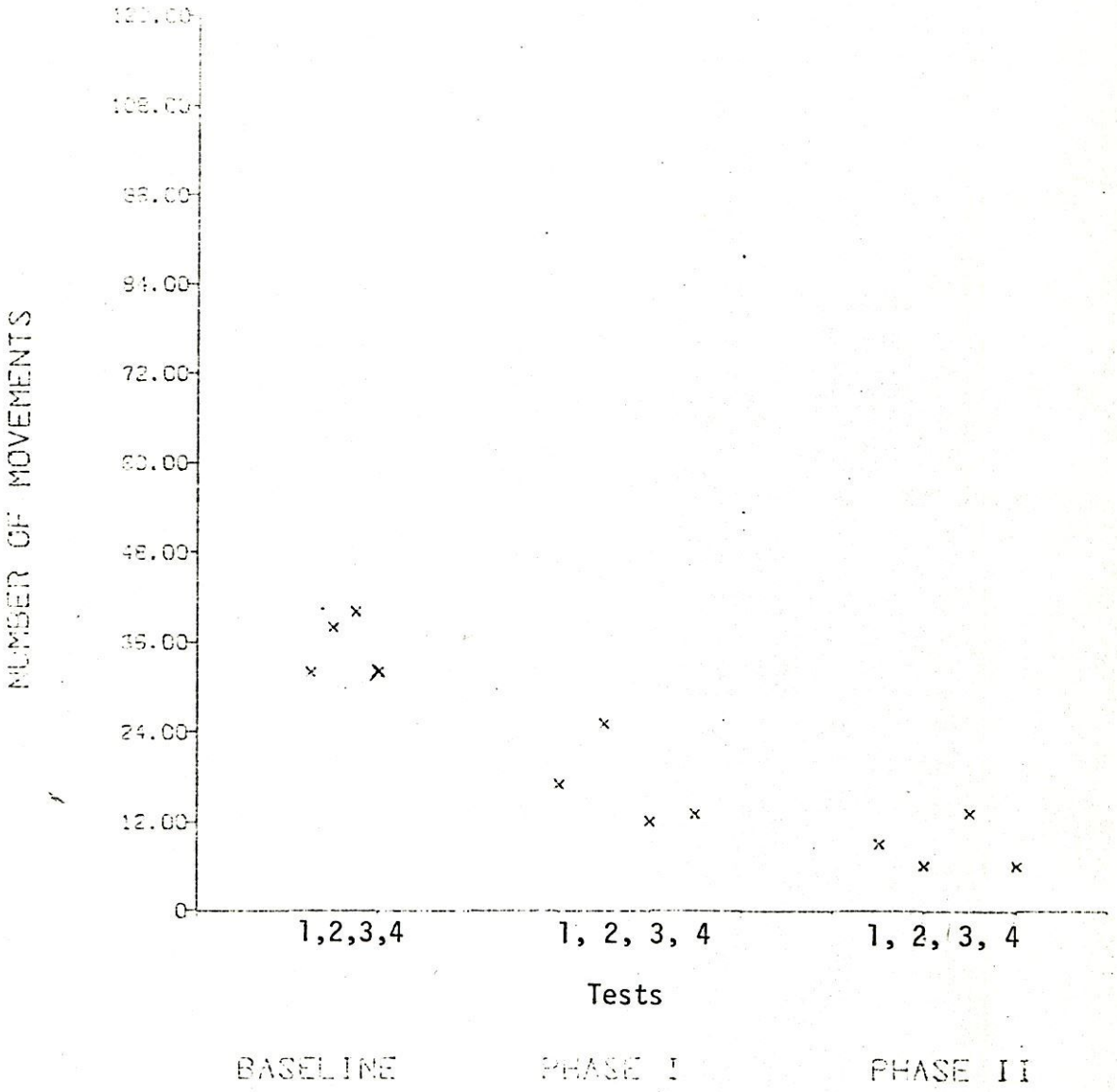
Latency (SE indicated)

PATIENT 2 : ART A (OPP. LIMB .5 . .3)



Repetition of Movement

PATIENT 2 PART B (UPP. LIMB 32 , 128)



Patient 3

MMPI

L	50
F	46
K	55
Hs	52
D	34
Hy	54
Pd	57
Mf	34
Pa	59
Pt	50
Sc	51
Ma	60
Si	50
Es	58

Code

862 '971-'3(67)^{0}6,10,9

Patient 3

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	108
Verbal IQ (VIQ)	106
Performance IQ (PIQ)	109
WAIS Verbal Subtests:	
Comprehension	12
Mathematics	9
Digit Span	9
Vocabulary	13
WAIS Performance Subtests:	
Block Design	13
Picture Arrangement	9
Object Assembly	12
Porteus Maze Test	16
Trail Making Test, Part A	42"-0 errors
Trail Making Test, Part B	63"-0 errors
Bender-Gestalt Recall	7
Wechsler Memory Scale (WMS) Quotient	116
WMS Subtests:	
General Information	6
Orientation	5
Mental Control	7
Memory Passages	12
Digits Control	10
Visual Reproduction	11
Associates Learning	18
Aphasia Screening Test (AST)	0-0 errors
LH Tactile Formboard I, Standard	33"-10 objects placed
RH	18"-10 objects placed
LH Tactile Formboard II, Blindfolded	300"- 8 objects placed
RH	62"-10 objects placed

Patient 3, 39 yr old white female, 1 stroke (post-operative) (12 yrs, 8 mos poststroke)

Clinical Diagnosis: (1) Probable right middle cerebral artery embolism
(2) Left upper extremity dystonia of hand and forearm muscles

Prior Handedness: Right

Prior Therapy: 37 months total; arm and leg

Comments: None

NEUROLOGICAL EXAMINATION

Description of Weakness:

Mild spastic left hemiparesis minimally involving the face. Weakness does not reach 4/5 in any joint. Dystonic posturing of right upper extremity. Difficulty forcing fingers open when hand is closed. Thumb often held in an abducted opposed position. Minimal left hemiparesis with severe dystonic posturing, with flexion of the fingers and wrist. Left hyperreflexia and Babinski signs.

Aphasia:

Normal

Apraxia:

None

Comments:

None.

Training Objective: Wrist extensor relaxation

Phase I: PT

Phase II: BF

Introduction:

Patient 3 was a relatively young (39 year old) stroke patient who suffered a right CVA after lung surgery for a benign adenoma. She had made great gains over the years in various therapies. For several months poststroke, she had a completely paralyzed left side. At the time of the experiment, she had a three-year standing residual of highly dystonic finger and wrist extensor muscles in her left upper extremity. There were no residuals in the lower extremity. The patient's wrist extension movement was highly dystonic and the extensor muscles were easily evident to touch (and at times even sight) as being very tight and overactive. When the patient let go of the wrist, passively or after extension, the extensors remained excited. Wrist extension was an intermittently interrupted motion resembling cogwheeling in Parkinsonism. The patient's fingers were extremely dystonic and her hand was always clenched tightly, fist-like in appearance. She could pull open the fingers with great difficulty with her opposite hand and maintain them extended, albeit the whole while the fingers moved athetoid-like in a splaying motion, and tended to flex back within a short period of time. Finger extension was facilitated by rubbing the dorsal surface of the hand and finger flexion by touch to the palmar

surface. The patient could extend the wrist with fingers flexed ([e.g., Part A] approximately to 209^0), but not with the fingers extended unless the fingers were first pulled apart. Range of wrist extension under the latter condition was half the range during extension with fingers flexed. The wrist extensors and finger muscles seemed an inextricable problem,--i.e., relaxation in either would likely have effects on both.

Over the course of baseline determination, Patient 3's affected limb reached values on all dependent measures equivalent to those of her unaffected limb (except for range, Part B, where extensor relaxation was always a good deal less than maximal under the requirements of continuous responding in the Rate situation). Thus, one unique measure was obtained for her which reflected the true difference between limbs. This was the lowest point of EMG activity following wrist extension during the test Rate situation. This measure was called "lowest EMG activity" (after eccentric contraction). Due to the above, as well as a qualitative change introduced into movement in Phase II (see METHOD), learning for Patient 3 is presented and evaluated apart from group statistics.

In order to conceal her crippled hand, Patient 3 always wore clothes with belts or pockets so that she could keep her hands in them, away from public scrutiny. She also draped coats, sweaters, etc. over her arm to further camouflage the hand. She furthermore wanted to protect her arm from injury and this positioning helped. Consequently, her elbow was always flexed as she walked in public, and, from habit, it was flexed a good deal of the time at home, too. When E first observed Patient 3, she was hard pressed to realize any paresis until

attention was brought to the completely obscured dystonic hand. The patient's positioning of the arm was not conducive to relaxation education in the wrist. Thus, the patient was urged throughout training to take steps to eliminate it and let her arm go.

Patient 3 lived with her husband and four children. She bore one of her four children after her stroke, while still quite hemiplegic, in order to reassert womanhood to herself. She led a very active life--doing all the housework, keeping a part-time job, and involving herself in school and civic affairs.

Patient 3 was well motivated and practiced a lot. While for five years poststroke her hemiplegic condition was one of flaccid, spastic paresis, her residual was an extreme tension, subjectively tiring and uncomfortable.

The patient at first tended to anticipate E in the test Command situation. During PT, she was also anticipatory at touch to her arm and she almost inevitably joined in with E to move her wrist as E was determining tension or trying to passively move it. The patient also had difficulty letting her whole limb go passively. During the course of training, Patient 3 explained that she probably was afraid of letting her limb go in order to protect it.[38] At the beginning of BF, Patient 3 stated some doubts about the acoustics to be used as she was afraid of losing her hearing only inasmuch as she was afraid of losing anything else health-wise. Despite the fact that the dystonia was a real physical handicap in that her hand was completely out of voluntary control, the above demonstrates a good deal of psychological overlay. The

---

[38] Patient 8 exhibited similar tendencies with one similar underlying psychological reason, protection of the limb.

patient was also highly inquisitive about rather sophisticated details of experimental design, neuromuscular anatomy, etc. In fact, although she made significant gains in training, E would infer that her intellectualism at times stood in the way of even greater gains.

Phase I: PT -

The greatest part of PT was devoted to general relaxation training of the limb moving specifically into the wrist extensor group, pursuing a goal of relaxed extensors and a limber wrist. The wrist was tight even when the patient was not actively extending. Furthermore, after extension, E could often feel a "catchpoint". As the wrist was let go, the extensors relaxed somewhat and then towards the end of "letting go" there was a slight increase in tightness. Patient 3 became very aware of this and all other subtle gradations in muscle tension. She would point them out to E or alternately, verify E's sensation of her arm. A veteran of several physical therapies, Patient 3 was not dismayed by unsuccessful attempts at control. In order for Patient 3 to counter her experience and fear of flaccidity in relaxation, she was told to view relaxation as a discipline, an active inhibitory process, subjectively the opposite of tension. Throughout PT, Patient 3 was never able to let her wrist go limber for any great amount of time during passive movement by E. She did, however, improve on the number of times she was able to achieve relaxation at the wrist and reported increasingly longer periods of a relaxed distal extremity particularly at home when alone and in a relaxed atmosphere. The patient realized that isolated relaxation in a conducive setting had to be incorporated into a daily routine. She thus tried taking time out to relax her wrist and hand at the office, when walking, etc. The wrist extensor and

finger muscles loosened together. The patient was encouraged to let the limb hang loosely in public, to try to push elevator buttons with a relaxed finger, etc. She began to open her fingers voluntarily and sometimes maintained them extended without athetosis for a short period of time.

At the end of the first week of PT, the patient had her fingers in a relaxed semi-flexed position and rubbing on the dorsal surface by E did not produce finger extension as it had during baseline. The patient reported her wrist and hand as feeling "more right". A problem during both PT and BF was occasional days of increased tone due to a hectic day or some sort of aggravation. (Pre-menstrual days always meant increased tone prior to training but this effect ceased during experimental months.)

PT training proceded with alternate practice at relaxed extensors (limber wrist, smooth motion during extension and release, quiet extensors after relaxation) and occasional voluntary extension of the fingers. The wrist flexors were never tight during wrist extension. Some resistance was applied to the flexors. As mentioned previously, if the wrist relaxed, the fingers were also relaxed. At times the patient would be doing quite well and then fatigue in her efforts, and report losing concentration. By the last week of PT, wrist motion was less saccadic, and relaxation of the wrist extensors and fingers was more frequent and complete. There was also no significant discrepancy now between range Parts A and B. The patient had attained a degree of wrist extensor relaxation ("lowest EMG activity") comparable to that of her unaffected limb on the four consecutive tests given during PT. Since her fingers were simultaneously relaxed, it was decided that she would raise the

wrist with fingers extended during BF. Thus, Phase II signified a qualitatively different response for this patient.[39]

Phase II: BF -

Patient 3 now practiced wrist extension with extended fingers. Her response to BF was "it's facinating". At first she was full of detailed questions at the end of sessions until E and she agreed that a non-analytical approach would be best. The second week of BF, Patient 3 reported having buttoned a shirt cuff with her left fingers and, when she realized it, she told herself to not think about it, just do it. She next reported keeping her arm on the arm rest of a chair at a theater with the fingers relaxedly extended throughout an entire movie. She began to walk around with relaxed fingers in public but her elbow would still be slightly flexed until she realized it and let it go. She also reported that wrist extension was becoming easier even when she felt that the number of her successes at relaxation (on the EMG TV) was holding steady. Finally, she reported having gone dancing with her husband and having held her right hand around his shoulder with fingers extended. At the end of BF, Patient 3 had a remarkably limber wrist at times (E would notice it when applying electrodes), was able to extend the fingers more rapidly, and could keep the fingers in a relaxed or semi-extended position for short periods of time without athetosis. However, as remarked earlier, there were still bouts of the "more spastic days".

---

[39] Patient 3 was the only patient for whom the target response incorporated a new feature in any Phase.

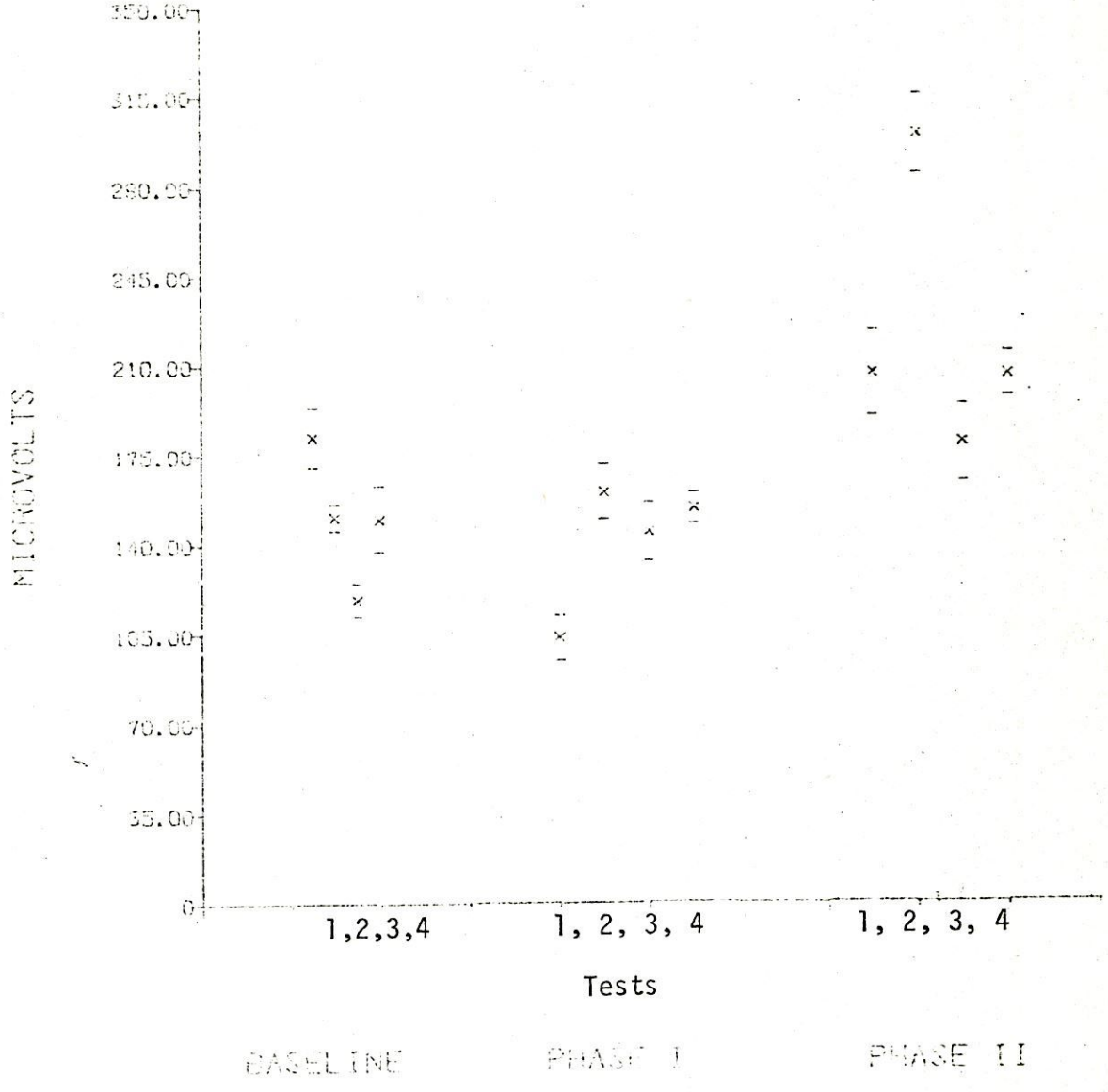
Comments:

E would evaluate Patient 3's progress as a continual alteration of motor control over time, with neither PT nor BF standing out as a more important mode of treatment. PT, however, was probably more effective for this patient in laying the acutely necessary foundation of relaxation for the progressive development of voluntary, coordinated motor control.

Also it was not mentioned in the Introduction that this patient, like Patient 5, but unlike all other patients studied, had suffered little, if any, cognitive loss due to stroke.

Averaged EMG Activity In Microvolts (SE indicated)

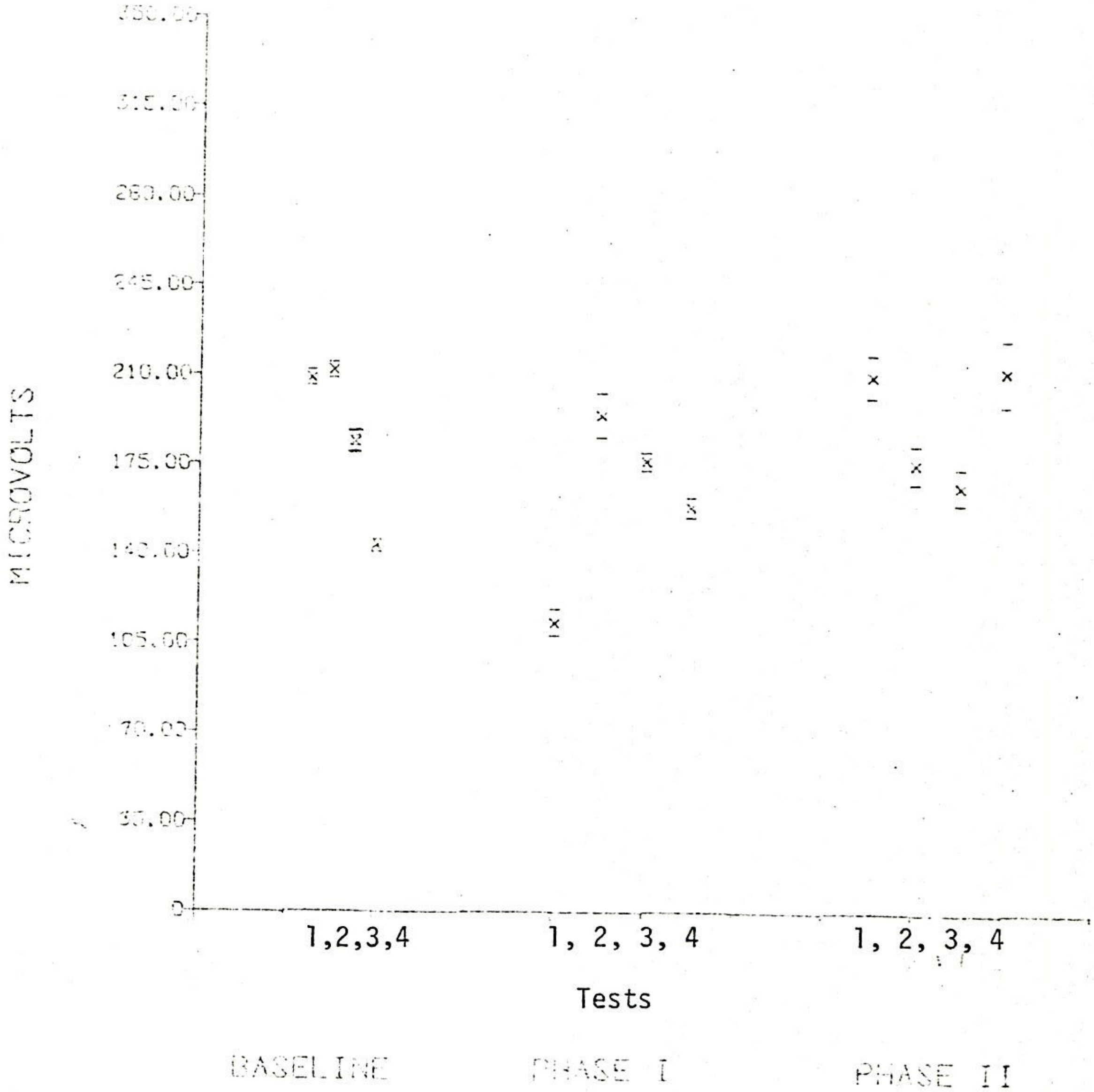
PATIENT 3* PART A (OPP. LIMB 175 , 115)



*Baseline and Phase I are composed of tests of responses of extending the wrist with fingers flexed. Phase II is composed of tests of extending the wrist with fingers extended.

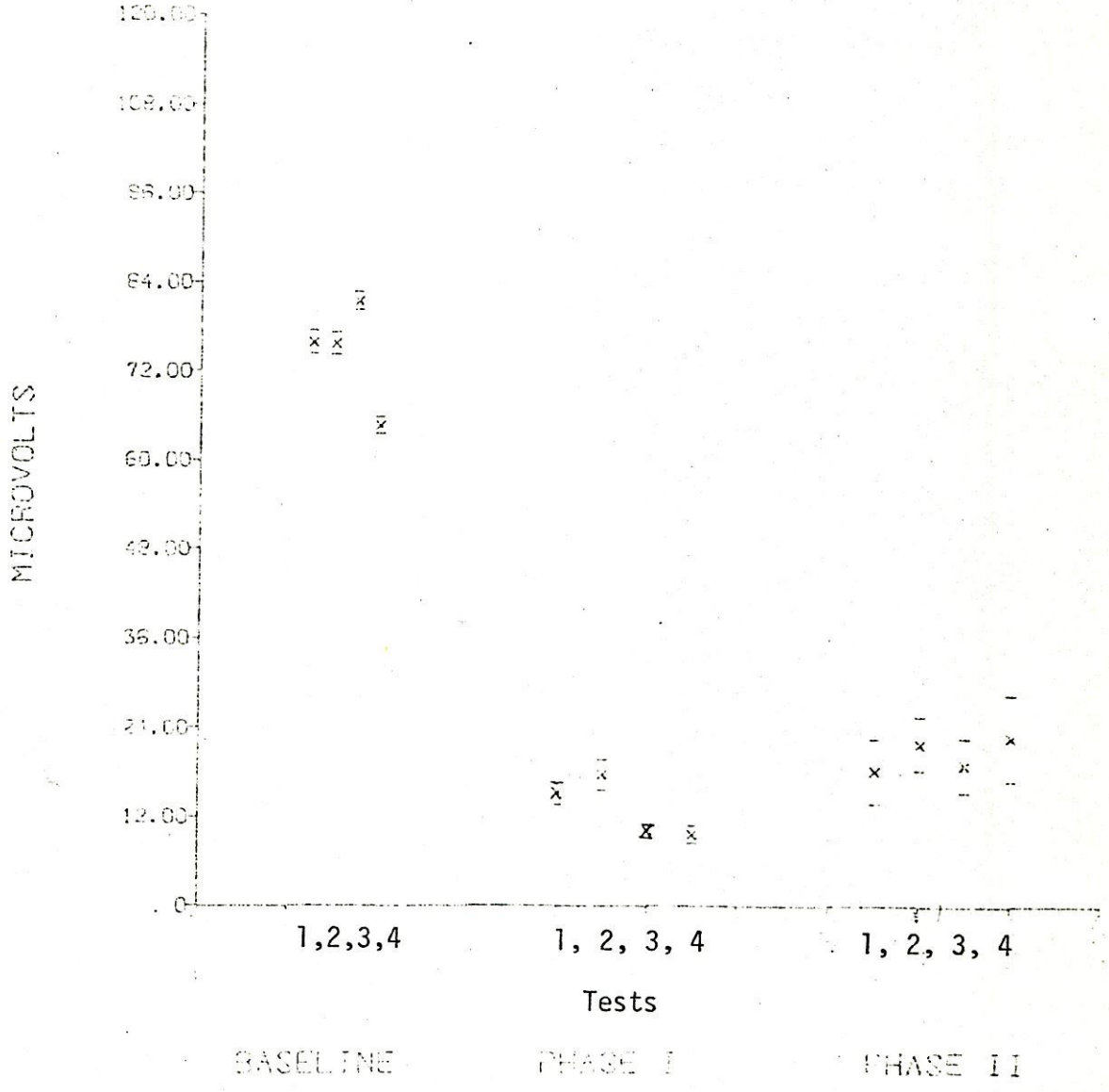
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 3*



*Baseline and Phase I are composed of tests of responses of extending the wrist with fingers flexed. Phase II is composed of tests of extending the wrist with fingers extended.

Lowest Averaged EMG Activity in Microvolts (SE indicated)

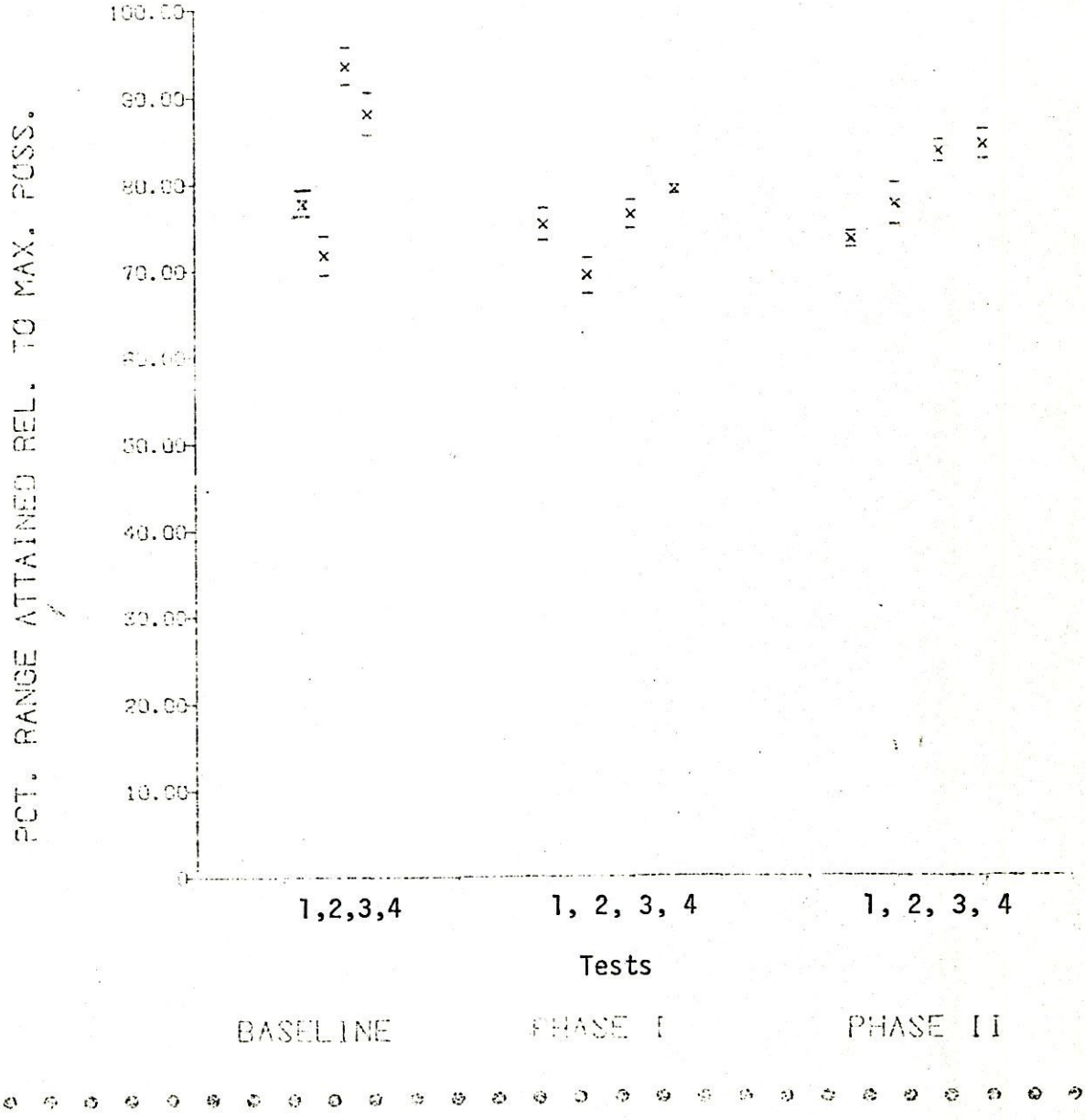


This graph is unique to Patient 3. It reflects the lowest amount of averaged EMG activity during wrist extensor relaxation, Part B. (For further details see the case history for Patient 3.)

*Baseline and Phase I are composed of tests of responses of extending the wrist with fingers flexed. Phase II is composed of tests of extending the wrist with fingers extended.

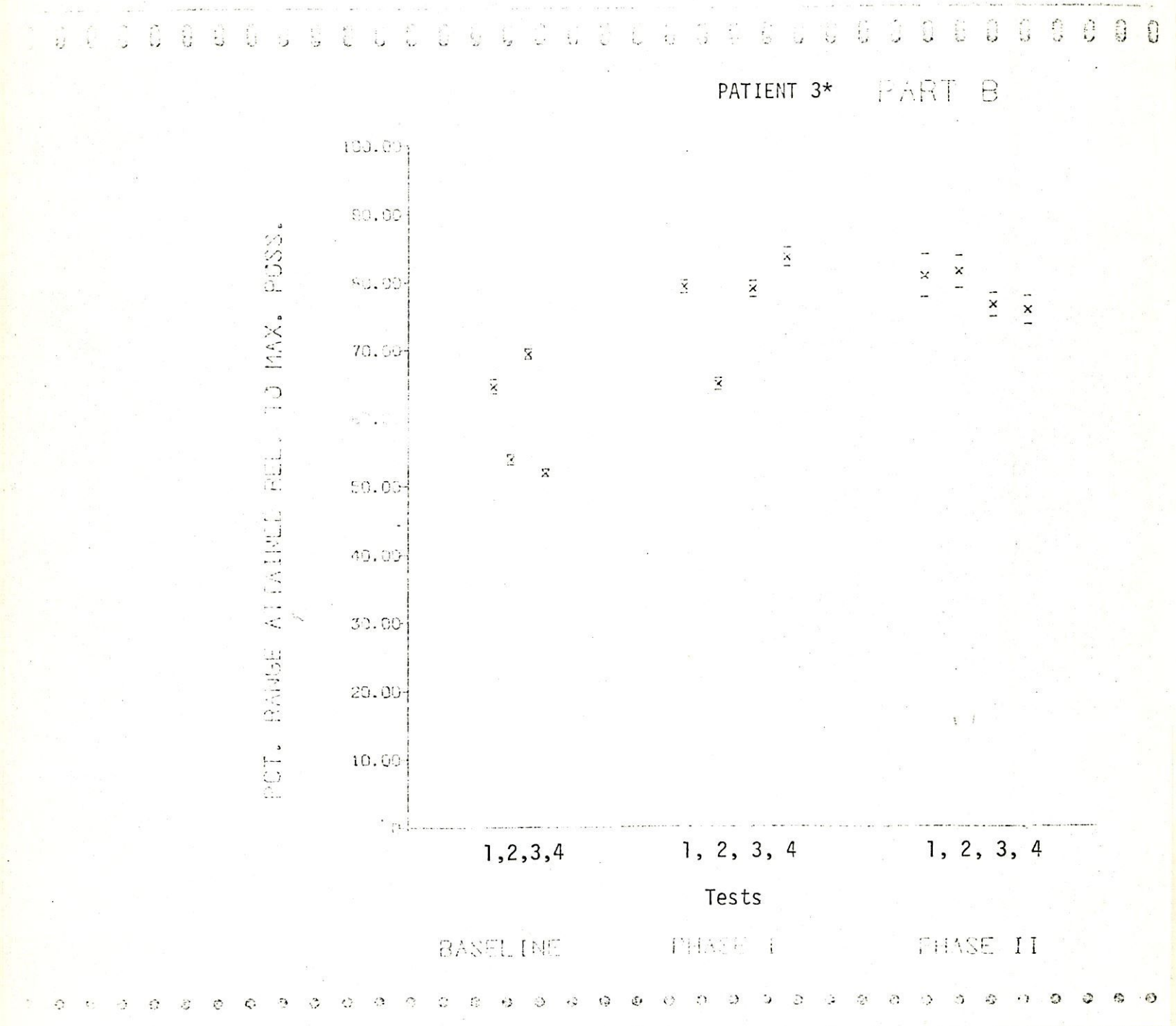
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 3* PART A



*Baseline and Phase I are composed of tests of responses of extending the wrist with fingers flexed. Phase II is composed of tests of extending the wrist with fingers extended.

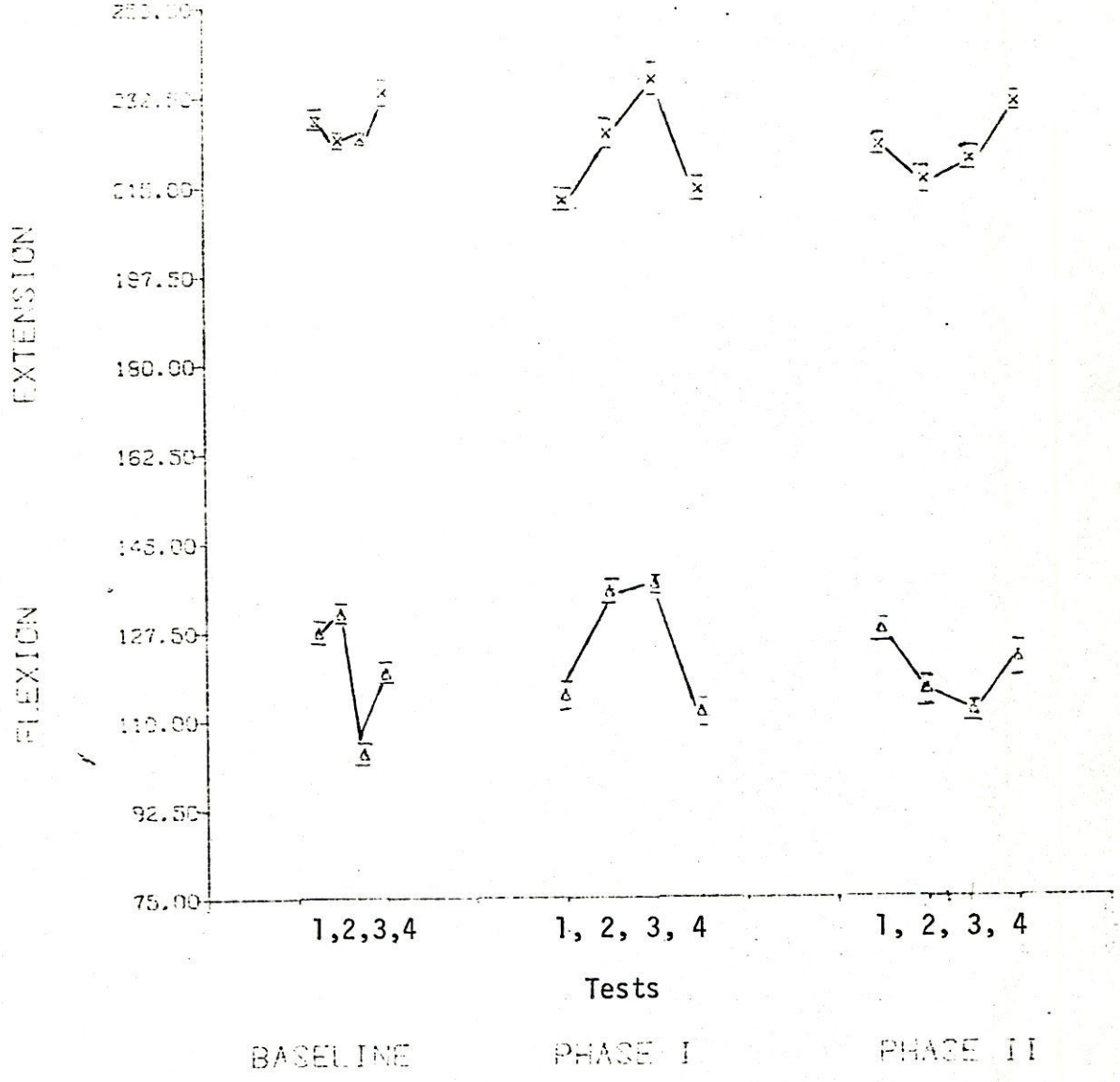
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)



*Baseline and Phase I are composed of tests of responses of extending the wrist with fingers flexed. Phase II is composed of tests of extending the wrist with fingers extended.

Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

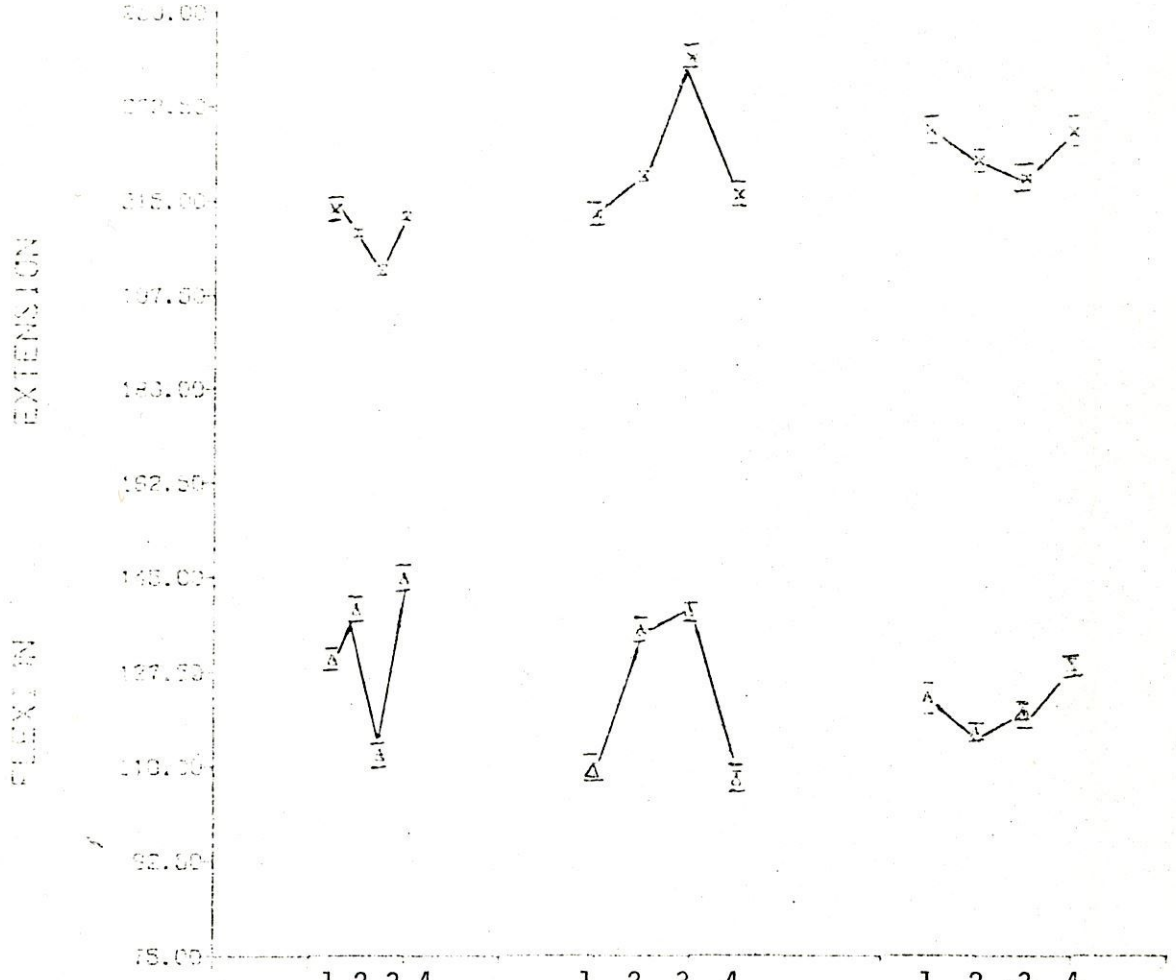
PATIENT 3* PART A



*Baseline and Phase I are composed of tests of responses of extending the wrist with fingers flexed. Phase II is composed of tests of extending the wrist with fingers extended.

Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

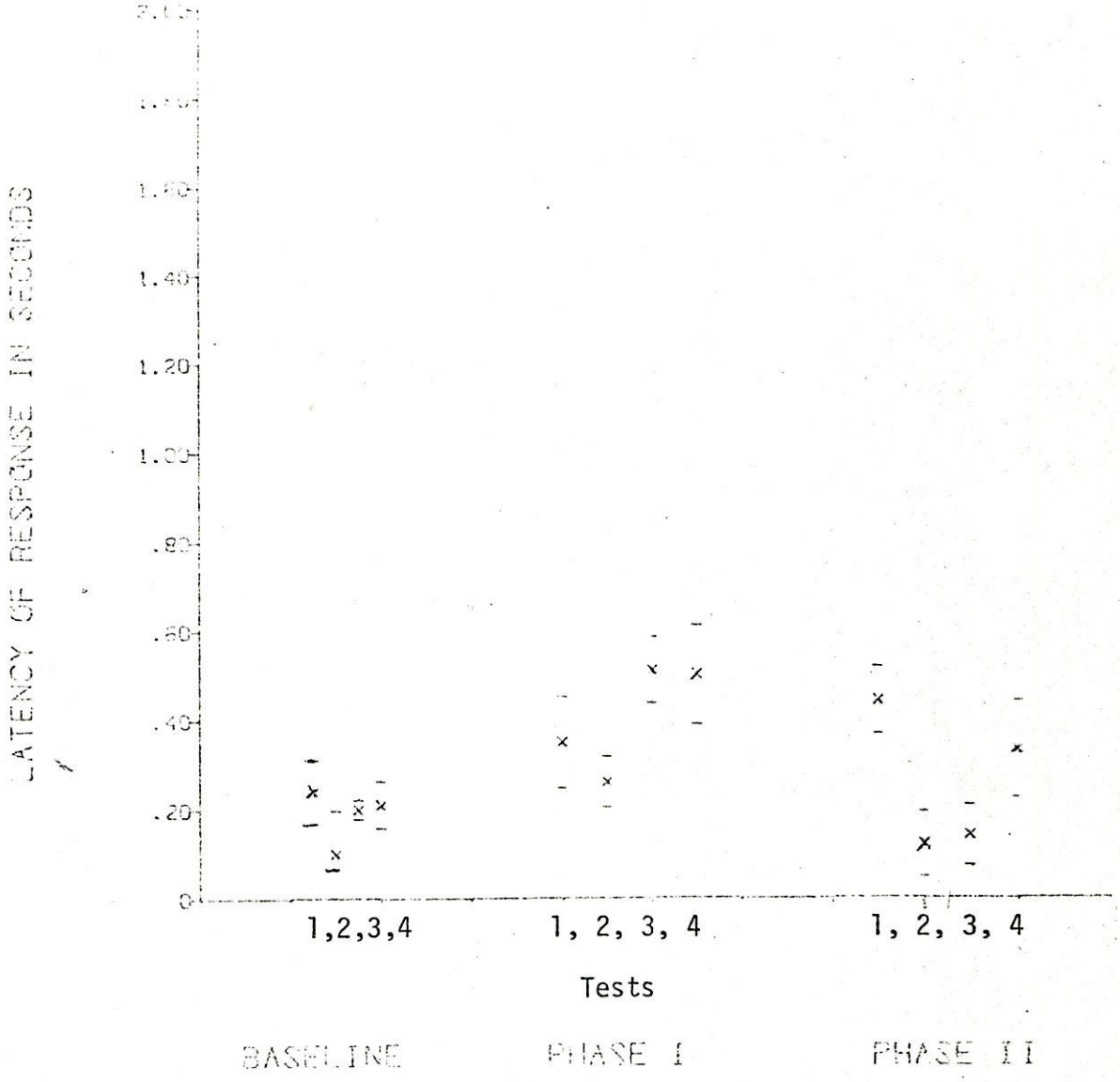
PATIENT 3*



*Baseline and Phase I are composed of tests of responses of extending the wrist with fingers flexed. Phase II is composed of tests of extending the wrist with fingers extended.

Latency (SE indicated)

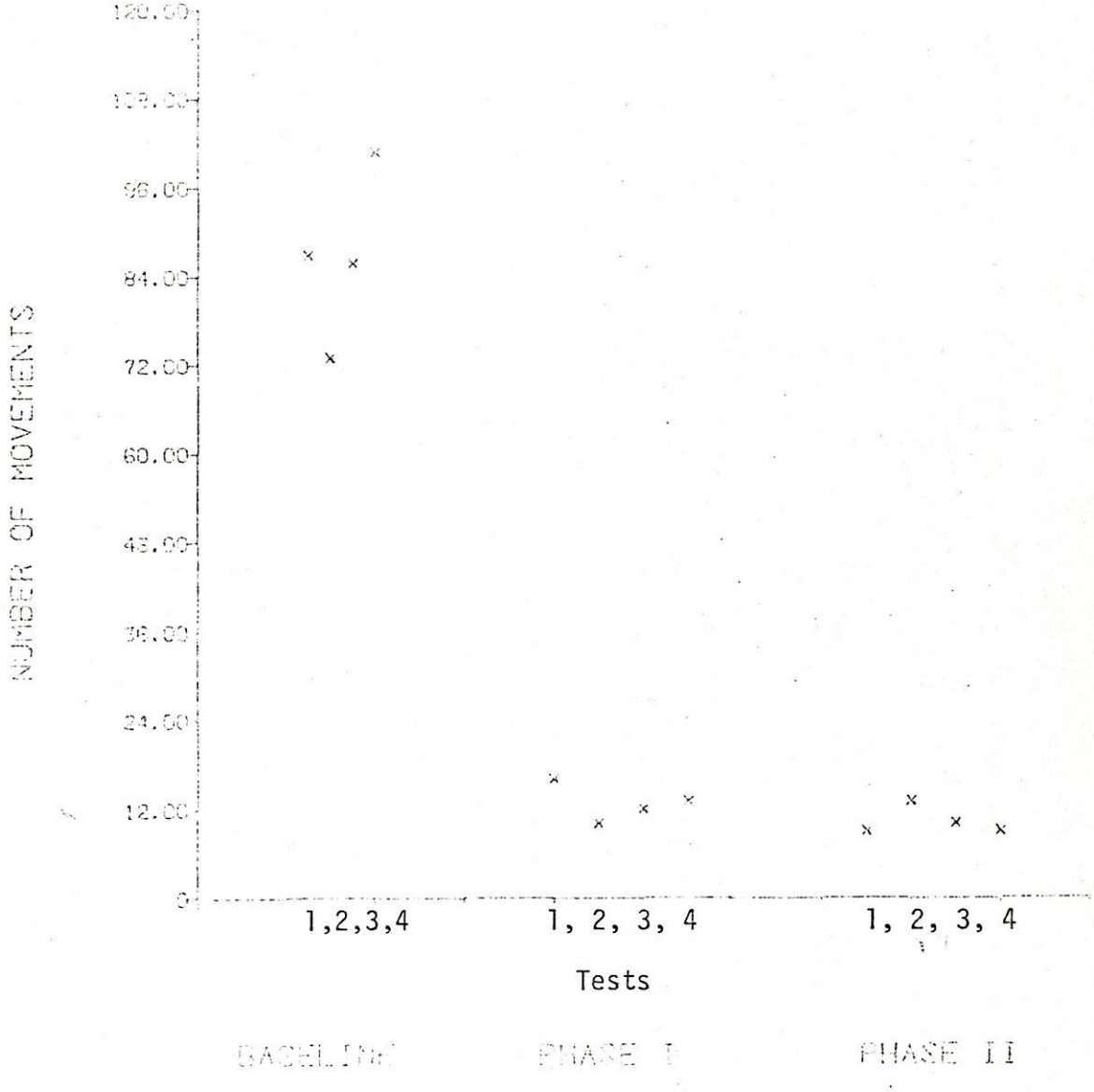
PATIENT 3* ART A (OPP. LIMB .3 , .1)



*Baseline and Phase I are composed of tests of responses of extending the wrist with fingers flexed. Phase II is composed of tests of extending the wrist with fingers extended.

Repetition of Movement

PATIENT 3*



*Baseline and Phase I are composed of tests of responses of extending the wrist with fingers flexed. Phase II is composed of tests of extending the wrist with fingers extended.

Patient 4

MMPI

L	53
F	50
K	59
Hs	70
D	70
Hy	76
Pd	53
Mf	69
Pa	47
Pt	69
Sc	63
Ma	55
Si	57
Es	43

Code

3 12'789-'(69)'5,3,17

Patient 4

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	97
Verbal IQ (VIQ)	96
Performance IQ (PIQ)	98
WAIS Verbal Subtests:	
Comprehension	9
Mathematics	9
Digit Span	8
Vocabulary	10
WAIS Performance Subtests:	
Block Design	11
Picture Arrangement	10
Object Assembly	7
Porteus Maze Test	9
Trail Making Test, Part A	98"-0 errors
Trail Making Test, Part B	500"-0 errors, incomplete at 500" limit
Bender-Gestalt Recall	1
Wechsler Memory Scale (WMS) Quotient	80
WMS Subtests:	
General Information	2
Orientation	3
Mental Control	4
Memory Passages	4
Digits Control	8
Visual Reproduction	4
Associates Learning	7
Aphasia Screening Test (AST)	8-7 verbal, 1 non-verbal errors
LH Tactile Formboard I, Standard RH	20"-10 objects placed
LH Tactile Formboard II, Blindfolded RH	300"-5 objects placed

Patient 4, 75 yr old white male, 1 stroke (2 yrs, 4 mos poststroke)

Clinical Diagnosis: (1) Arteriosclerotic heart disease
(2) Probable extensive thrombosis
(3) Right hemiparesis

Prior Handedness: Right

Prior Therapy: 1 yr 3 mos total; arm and leg

Comments: None

NEUROLOGICAL EXAMINATION

Description of Weakness:

Spastic right hemiparesis with marked right facial involvement. Limb weakness is greatest at the right shoulder and right hip with 2/5 strength at the shoulder, 2/5 biceps, 4/5 triceps, 4/5 wrist extensors and flexors, and finger extensors and flexors; 2/5 at hip, 4/5 knee, ankle, and toes. Tone increased on right particularly extensor tone at elbow. Hardly any elbow flexor tone; however, there is flexor spasticity of the fingers and wrist. Hyperreflexia on right with sustained right ankle clonus. Left sided reflex also brisk. No Babinski signs.

Description Sensory Impairment:

Pin: Intact

Touch: Intact

Vibration: Intact

Position Sense: Intact

Two point discr.: Decreased on left hand.

Graphesthesia: Impaired bilaterally.

Stereognosis: Impaired on right hand.

Aphasia:

Dysphonic and dysarthria of spastic nature.

Apraxia:

None

Comments:

Hemiparesis involves proximal upper and lower extremity suggesting possible anterior cerebral artery lesion or watershed distribution lesion bilaterally; suggested by speech and possible bilateral sensory signs and reflex signs.

Training Objective: Wrist extension

Phase I: PT

Phase II: BF

Introduction:

Patient 4 had a right spastic hemiparesis. Following the stroke, return started distally in both the upper and lower extremities. The patient could extend the wrist and fingers moderately well although movement was weak and fatigued easily. Wrist extension tended to

include elbow flexion. Elbow flexion was a "favorite" movement for Patient 4 and he walked holding his arm in a flexed position of about 90^0. Spontaneous movement consisted of minimal shoulder flexion, extension, or abduction with flexion and extension particularly of the elbow and fingers, and secondarily, the wrist. He tended to move his chest and abdominal muscles as well as the lower extremity of the paretic side along with any movements of the paretic upper extremity. Aside from the latter, any obligatory stereotyping of a gross movement pattern in response to general or specific commands was relatively quite minimal.

The patient lived with his wife who was an extremely caring nursemaid. Thus, he was able to sit all day and got help in hygiene and transfers, although he could, in fact, very easily do these himself. Because of the available help, he used his paretic upper extremity to a minimal degree. He was very cooperative and increasingly motivated as he saw training "doing something" for him. Under his wife's tutelage, he practiced a moderate amount at home.

Patient 4 seemed to have suffered some receptive language loss. He would sometimes be momentarily perplexed at what, for example, "raise the wrist" meant, or, for example, he would bend the elbow to that command or vice versa. Patient 4 was instantly frustrated at the slightest of his misunderstandings. He was also frustrated by an expressive aphasia which left him unable to express himself at a normal rapid rate (the words came out garbled). He had very little acceptance of his general condition. He was, however, in very good physical health. In sum, Patient 4 had a good deal of mental loss especially at other than primitive levels of reasoning; his concentration was poor

and he was easily prone to cry over his "rotten" condition (his report). He also once blurted in reference to his paretic upper extremity, "ah, cut it off".

There was a good deal of uncertainty on the part of the patient in processing or integrating the idea of which movement was being asked of him verbally. He would say e.g., "Which movement is that?", or "I forgot", etc. E thus often showed him the movement and he then said "oh ya, ya, ya". Thus, he had trouble translating language into action but he usually initiated a movement very well when it was visually demonstrated. His behavior of forgetting movements might also be characterized as a "latent" motor ability which was confounded by an overriding disability of the patient's being unable to make specific connections to movements via external verbal or "spontaneous" internal control. (Except for elbow flexion, a latter type of"guidance" was, hence, in effect, non-existent.) Once Patient 4 started a required movement, the understanding of how to do it was no problem. However, execution was usually less than optimum because the specifics of correct movement, which he could well attain, were not part of his automatic repertoire. Thus, they were constantly verbalized and/or illustrated to him.

Patient 4 could not attend to one, let alone several, details of a movement for any length of time. Thus, he would extend his wrist but he limited his range because his natural tendency was to work in less than his maximum angle of eccentric or concentric extensor contraction. His motor and mental memory were very limited. Thus, during PT, carry-over changes in performance from one session to the next had to be reiterated before performance would resume at a given level. Similarly, during BF, it took many sessions before Patient 4 was able to

simultaneously extend his wrist maximally, aside from simple focus on raising the TV dot which would go very high, though not peak, with less than maximum extension. In addition to this, until repeatedly reminded after many training sessions, he did not bring the TV dot back down to zero (maximum extensor inhibition) before attempting to raise the wrist again. Patient 4 favored looking at his wrist during movement.

Phase I: PT -

PT focused on resistance to wrist extension, attention to maximal wrist extension, and practice of shoulder flexion. Wrist extension was best when the limb rested on the table and much reduced when the limb rested in other positions (for example, hanging by the patient's side). Over PT sessions, Patient 4's wrist extensor movements became more "natural" and motor unit recruitment increased ([e.g., Part A] from approximately 62 μv at baseline to approximately 76 μv during PT). Range remained approximately the same. Patient 4 also made a lot of progress on flexing the shoulder, with the elbow extended (from 180^0 at baseline to approximately 130^0 during PT).

Phase II: BF -

Patient 4 was frequently interrupted by E during BF, since, as already explained, he could not concentrate on, or remember, the details of movement for very long. Thus, he had to be checked to be reminded to 1) relax the wrist to zero on the TV; 2) raise the dot by extending the wrist maximally; and 3) keep his chest and abdomen muscles at ease. It seemed that there was a greater ability to attend to all three features as BF progressed. The most important result of BF was a great

increase in the averaged EMG ([e.g., Part A] to approximately 132 μv). Range, which was (e.g., Part A) approximately 63^0 at baseline and 65^0 during Phase I, increased to approximately 74^0. E would surmise, however, that while performance was good when sustained by BF and verbal instructions of specifics, ultimate storage was minimal. That is, there was probably little transfer of external stimulus to internal stimulus control for the target movement.

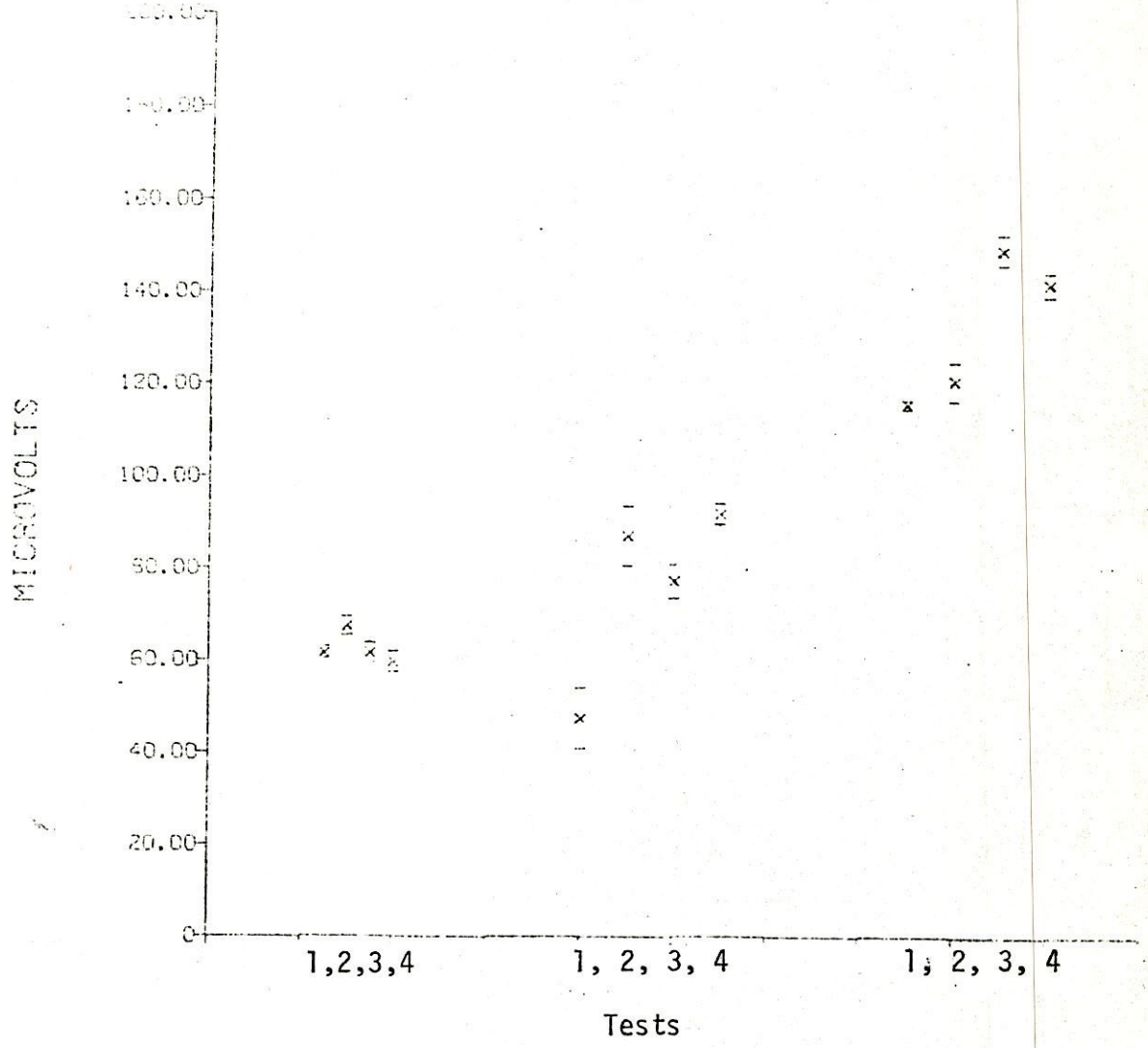
Comments:

1) As a result of overall training, the patient started to wash his face and almost successfully tried to plug his razor into a wall socket with his paretic hand. 2) In addition to a deficient motor repertoire, this patient suffered a great lack of attentional capacity.

Patient 4 is missing a score for test 1 of Phase I on graphs of angular motion because of a goniometer equipment failure.

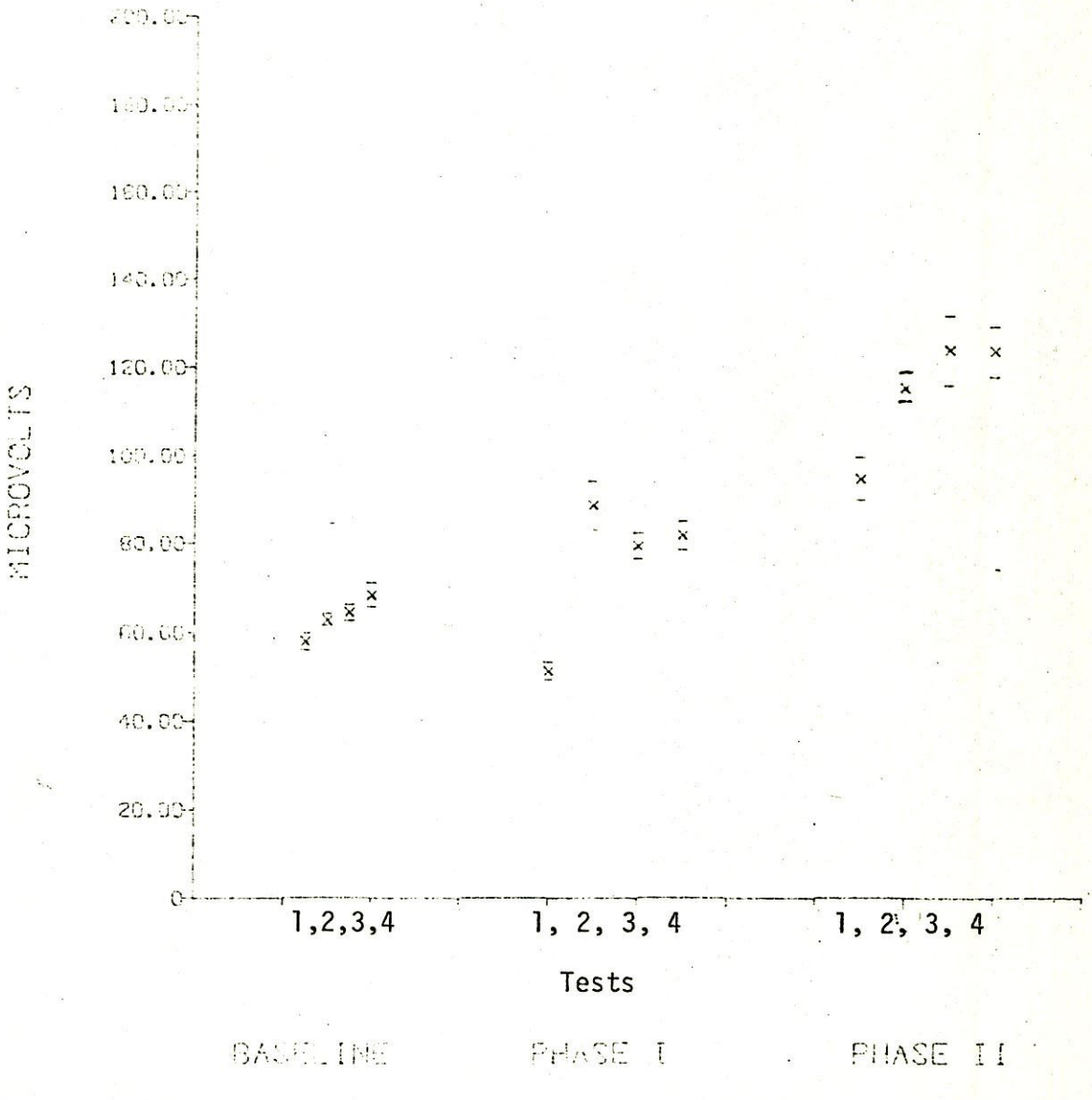
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 4 PART A (DPP. LIMS 231 , 241)



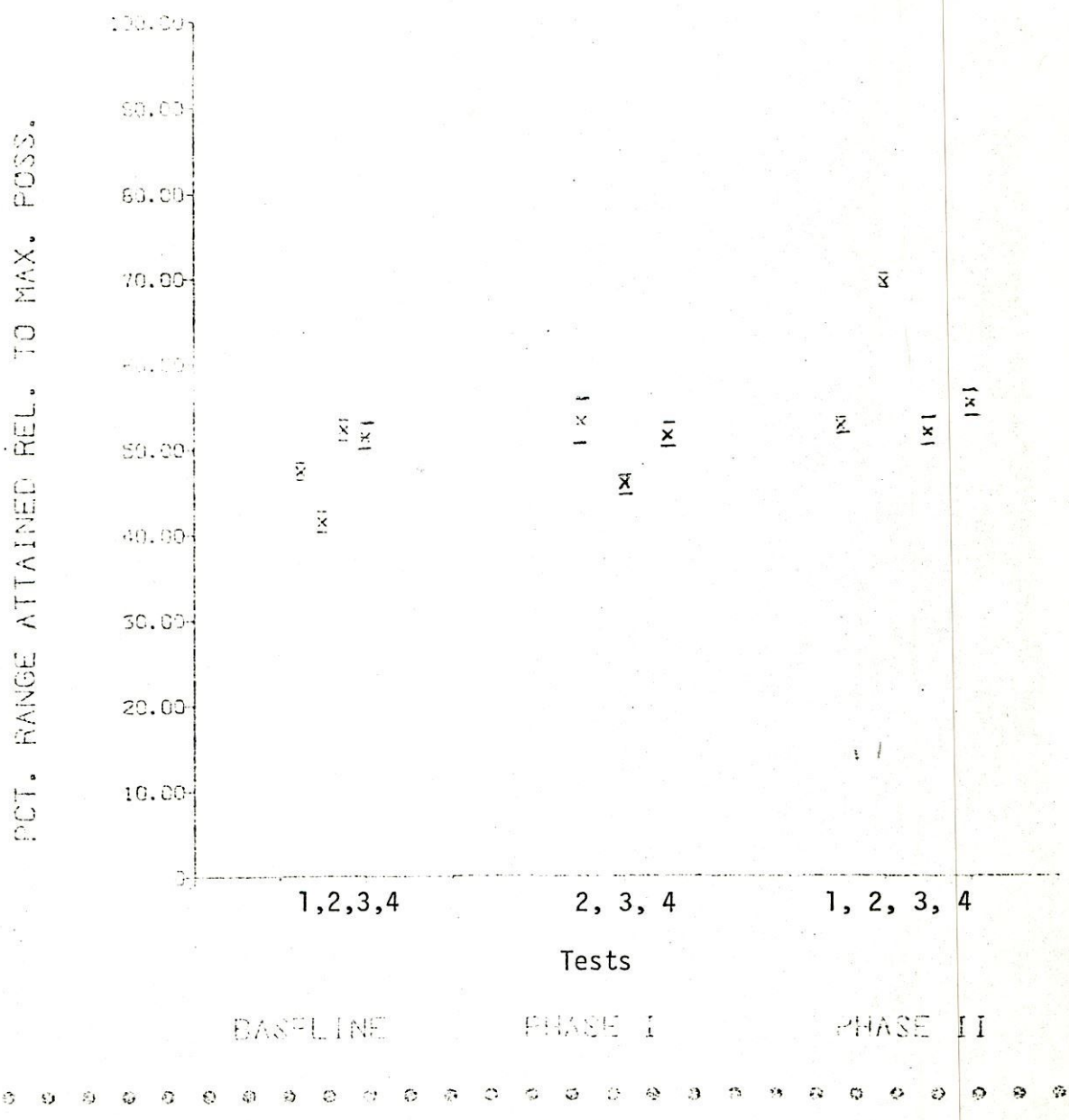
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 4 PART B (OFF. LIMB 212 , 261)

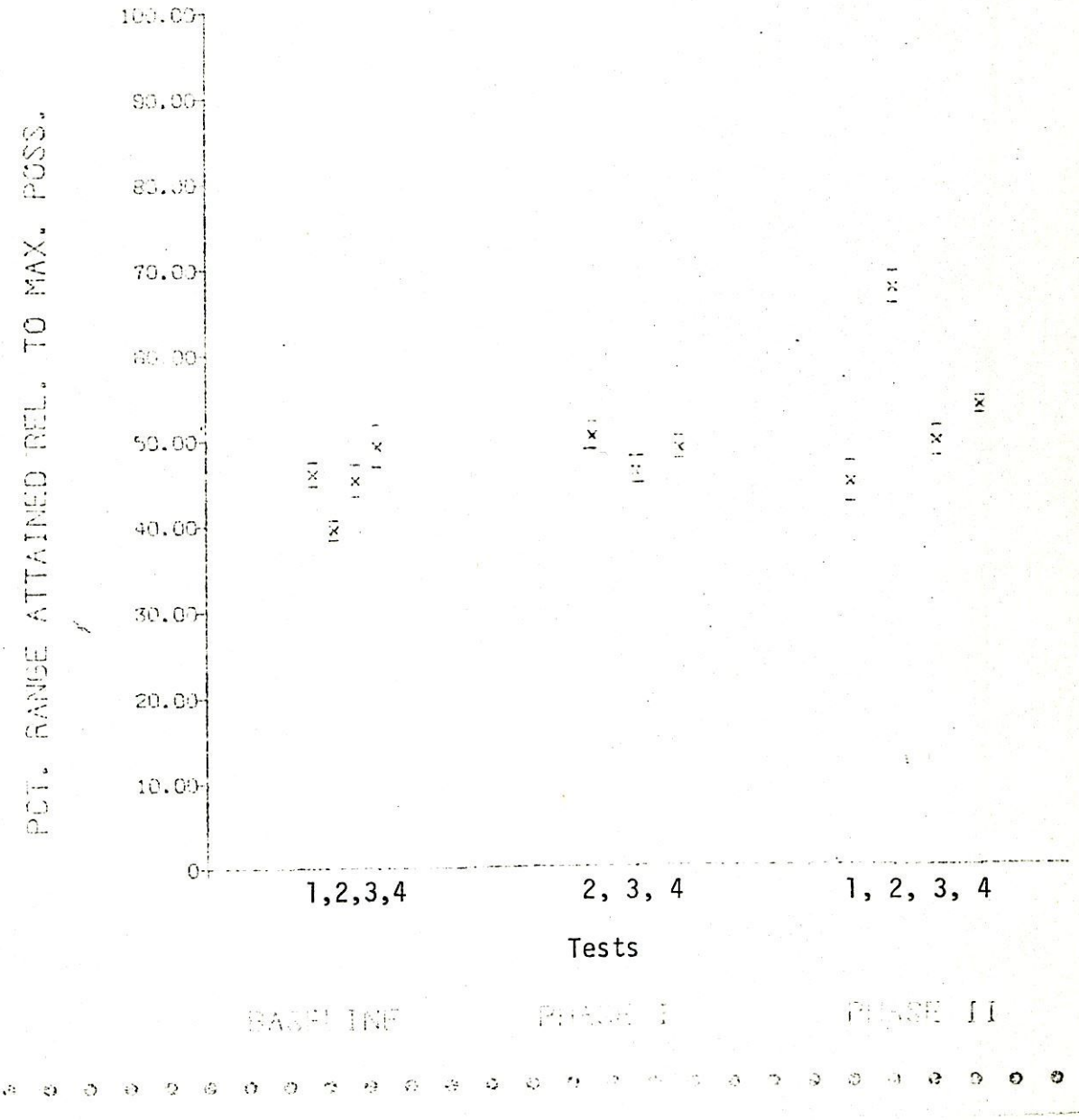


Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 4 PART A

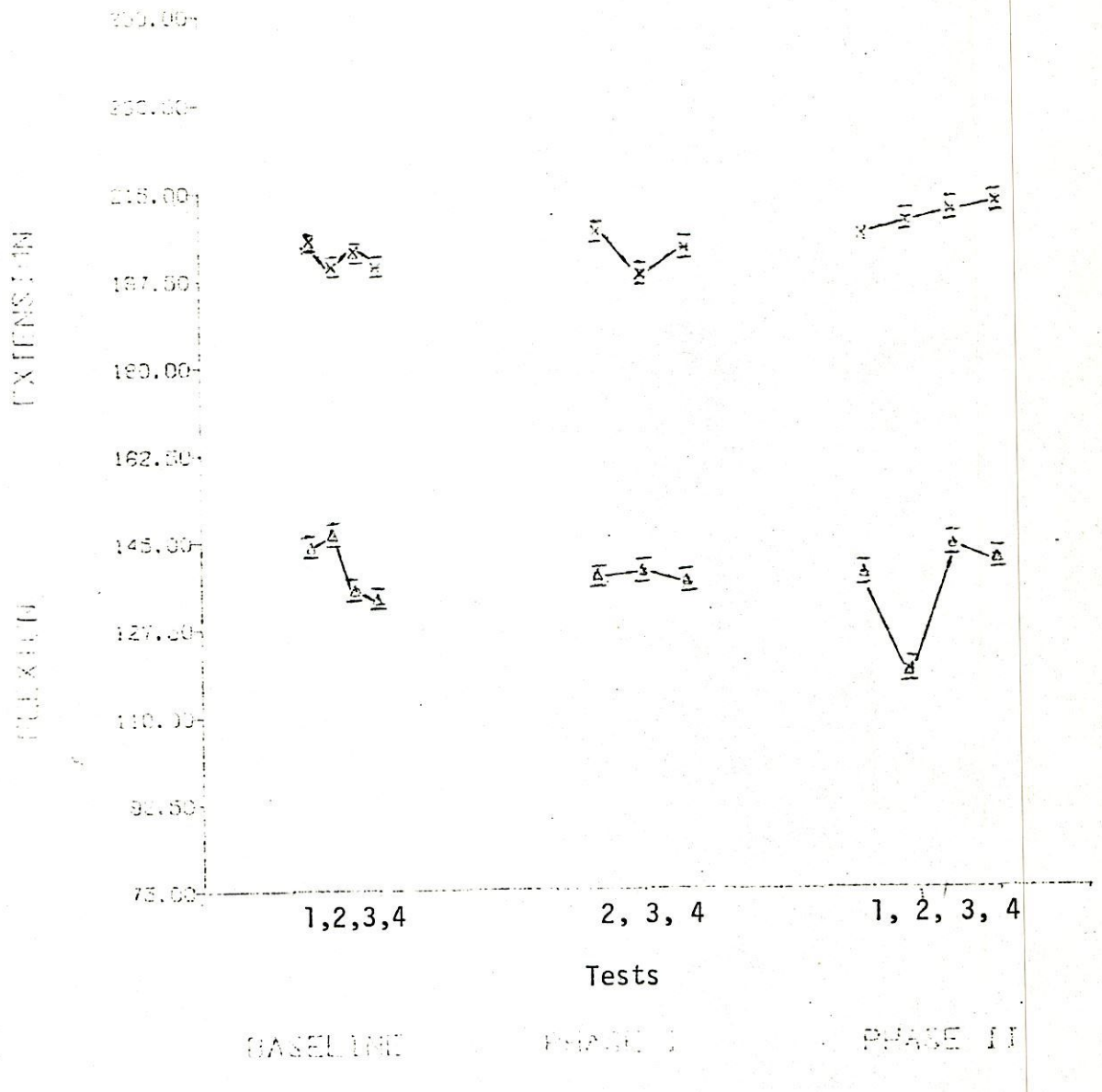


Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)



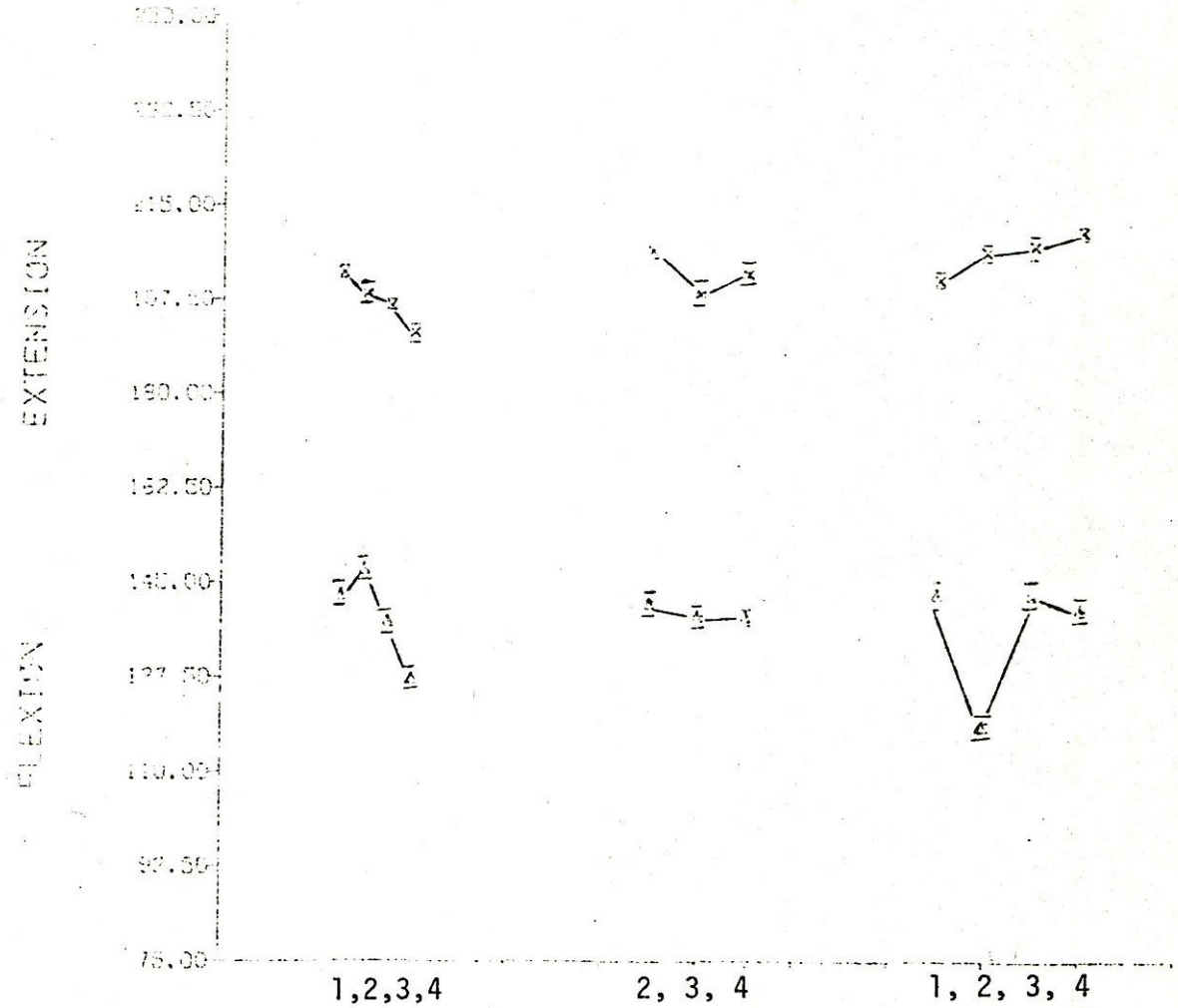
Extension ($\bar{X}$) and Flexion ($\bar{\Delta}$) in Degrees (SE indicated)

PATIENT 4



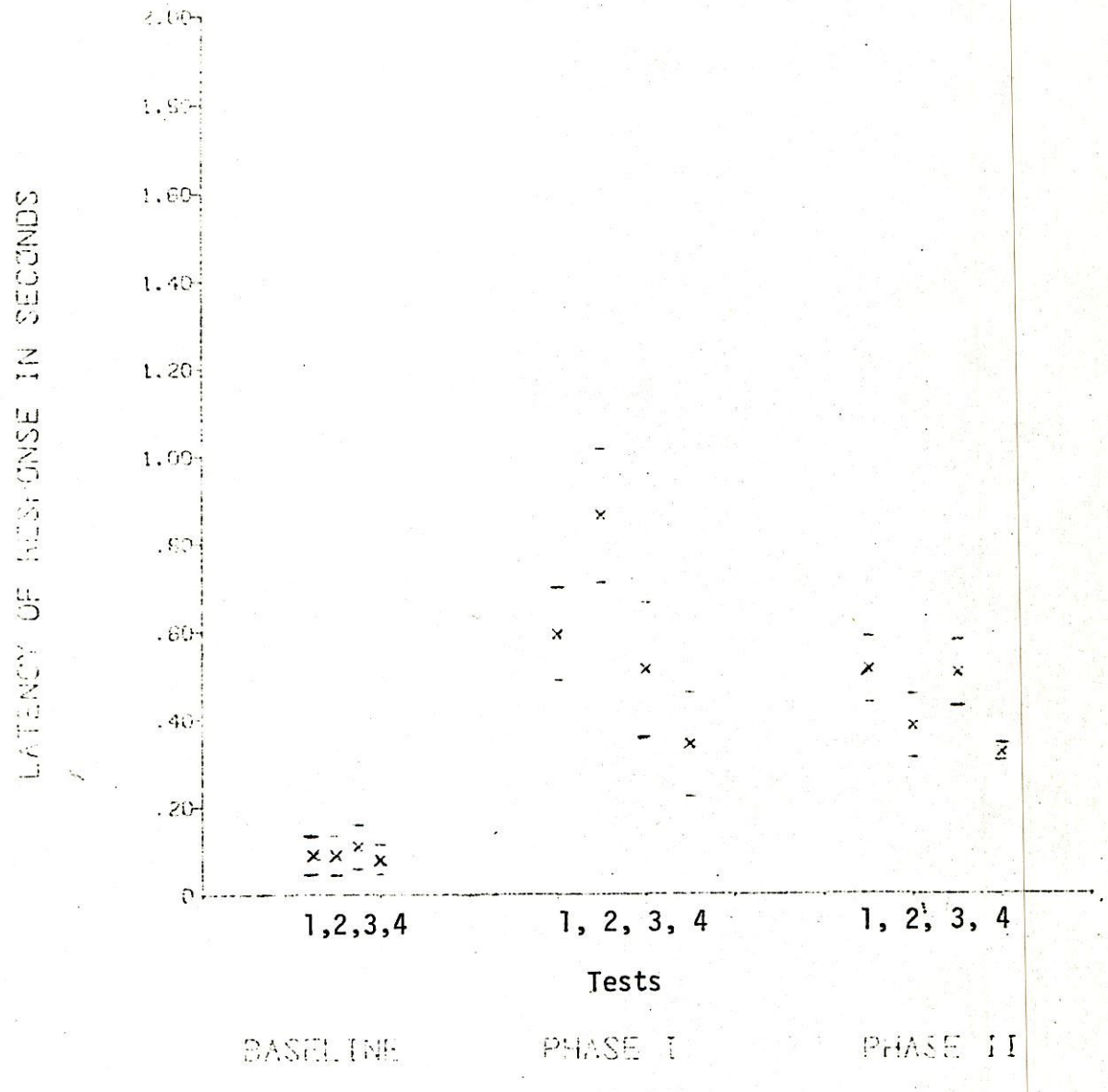
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 4



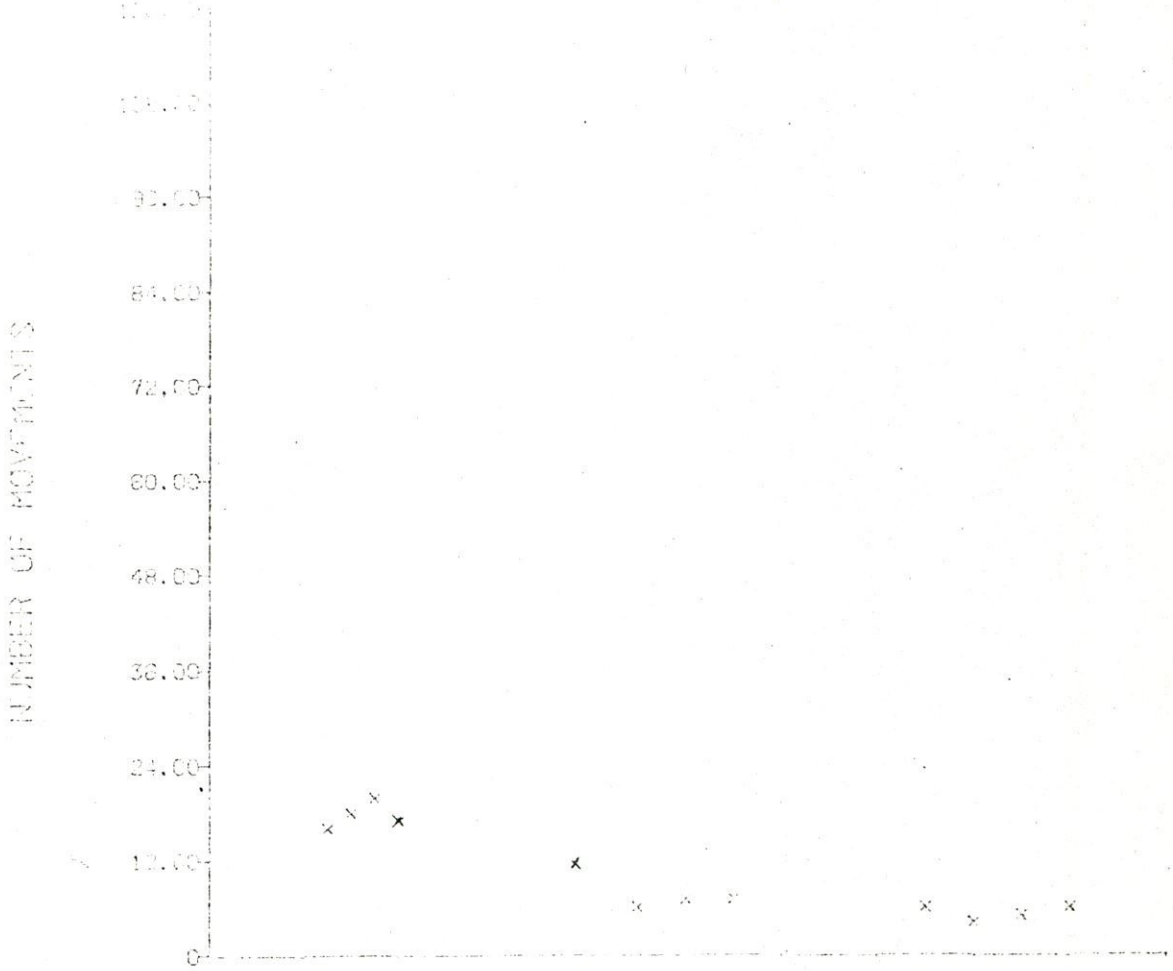
Latency (SE indicated)

PATIENT 4 P-F-P A ([illegible] LIMB .4 , .4)



Repetition of Movement

PATIENT 4 (



BASELINE PHASE I PHASE II

Patient 5

MMPI

L	50
F	55
K	62
Hs	77
D	48
Hy	78
Pd	71
Mf	57
Pa	59
Pt	60
Sc	78
Ma	78
Si	42
Es	46

Code

38914'76-'(57)^{0}4,5,19

Patient 5

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	114
Verbal IQ (VIQ)	117
Performance IQ (PIQ)	108
WAIS Verbal Subtests:	
Comprehension	14
Mathematics	13
Digit Span	10
Vocabulary	13
WAIS Performance Subtests:	
Block Design	12
Picture Arrangement	10
Object Assembly	12
Porteus Maze Test	13.5
Trail Making Test, Part A	44"-0 errors
Trail Making Test, Part B	89"-0 errors
Bender-Gestalt Recall	6
Wechsler Memory Scale (WMS) Quotient	143
WMS Subtests:	
General Information	5
Orientation	4
Mental Control	9
Memory Passages	17
Digits Control	10
Visual Reproduction	13
Associates Learning	20
Aphasia Screening Test (AST)	0-0 errors
LH Tactile Formboard I, Standard	18"-10 objects placed
RH	
LH Tactile Formboard II, Blindfolded	282"-10 objects placed
RH	

Patient 5, 54 yr old white male, 1 stroke (1 year, 9 mos poststroke)

Clinical Diagnosis: (1) Hypertension
(2) Obesity
(3) Right cerebrovascular accident
(4) Left flaccid hemiparesis

Prior Handedness: Right

Prior Therapy: 16-1/2 mos total; first 7-1/2 mos, leg only, remaining 9 mos, arm and leg

Comments: None

NEUROLOGICAL EXAMINATION

Description of Weakness:

Left hemiparesis with increased reflexes and Babinski sign. Left arm abducts at 60^0 but is very weak both at elbow and wrist. Minimal spastic flexor positioning in the upper extremity with minimal spastic extensor positioning of the left lower extremity. No dorsiflexion movement at foot although extension at the knee and hip are present.

Description Sensory Impairment:

Pin: Intact

Touch: Intact

Vibration: Intact

Position Sense: Intact

Two point discr: Slight decrease of two point discrimination on the left hand.

Graphesthesia: Intact

Stereognosis: Intact

Aphasia:

Normal

Apraxia:

None

Comments:

No comment.

Electromyography Report (done by Tsing Yun Chiang, M.D., Veterans Hospital, Minneapolis):

EMG of sampled muscles from the left arm show impairment of recruitment of the motor units proportional to the strength of contraction. Such impairment is more severe distally than proximally. Surface EMG of the left extensor indicis fails to reveal abnormal increase in frequency of low amplitude MUAP. Test of neuromuscular transmission is normal. The distal motor latency and the motor conduction velocity of the left ulnar nerve are likewise normal. There is nothing in the findings of the electrodiagnostic test which is not accounted for by the old stroke.

Training Objective: Wrist extension

Phase I: PT

Phase II: BF

Introduction:

Patient 5 had a spastic hemiparesis. His left upper extremity was so flaccid as to amount of flail and his paresis severe. He had subluxation of his muscles at the glenohumeral joint. His paretic upper extremity hung loosely and swung freely like a pendulum when he walked (as opposed to a coordinated, rhythmic arm swing natural to walking). His fingers were held in a semiflexed spastic position with the thumb adducted parallel. Movement to general command consisted of an excessively effortful response with a great deal of overflow to facial and back movements. The paretic limb exhibited a slight mass movement pattern of elevation against gravity. For the most part, however, the limb was moved by passive activity of gravity in succession to a general series of other body movements. Movement to specific commands resulted in a similar collection of movements to produce some slight evocation of the desired movement. Ability to flex the elbow and extend the wrist was slight and was executed in the gross fashion described, with arm abducted and body tilting to the right. There was no ability to flex the shoulder. Patient 5 could not sustain a movement and fatigued very rapidly with respect to movement frequency. Extension of the wrist caused some finger extension and greater thumb abduction. Throughout training for wrist extension, the patient could feel "pulls", but often in inappropriate places--for example, the shoulder, the wrist flexor muscles, or fingers. He sometimes claimed to feel pulling in the wrist extensors.

Patient 5 was an unusual case. During Phase I (PT), the patient could isolate specific (pure) movements without much training, and E surmised that this argued well for improvement. As it happened, Patient 5 did not improve under either PT or BF training.

Patient 5 lived alone and was completely self-sufficient, although everything was difficult for him. He was quite obese and walked with a Lofstrand cane in the left hand. He managed a sling for his left limb with his teeth and right limb. He had many devices for self-care which he himself designed and had set up around his apartment--for example, a file attached to a table to file his nails. Although he would ordinarily have been classified as needing help in hygiene, transfers, etc., he managed alone.

It appeared that Patient 5 did not suffer much loss, if any, of cognitive function. He was quite active in politics and organizations for the handicapped. The patient claimed to have good "reflex action" with his left arm in emergency situations--for example, grasping something very well when about to fall to the extent that he had to pry his gripped hand open afterwards. The patient practiced a good deal.

Phase I: PT -

When he started PT, Patient 5 had a slight ability to flex the elbow and raise the wrist while abducting the arm and tilting his head and body to the right. A couple of sessions of PT sufficed for him to zero in on three discrete, pure movements: wrist extension, elbow flexion, and shoulder flexion. He was also able to keep the elbow extended during shoulder flexion and continually improved on range of motion in the three movements. This "improvement" was always evident only on the first one to three tries. He then could do no more and frequently referred to movement as trying to "move a dozen boxcars". E was always perplexed by the patient's genuine skill in movements specifics but his terrible lack of power. Where and how, the question

recurred, could Patient 5 recruit only the same amount of motor units for a fair amount of range of motion in the first place, and secondly, repeatedly fail to reproduce the response after the first couple of tries. The patient seemed to have excessive inhibition (which did not prevent up to 38^0 of wrist extension) and a rapidly fatiguing motor output.

Resistance training could not be used much as the patient would have met with a lot of failure. His wrist extension response was always barely anti-gravity, particularly after a couple of tries. Facilitation by poking the extensors (which had to be forceful due to the patient's obesity which made the extensor muscles difficult to percuss) caused black and blue marks. The patient also had poor circulation on the left side, for which he took medication; the problem was sometimes evident as a bluish skin tone.

The patient sometimes felt his "fingers in spasm" as he tried to extend the wrist, and his wrist flexors were simultaneously active with attempts at wrist extension. By the third week of PT, Patient 5 was able to extend his wrist approximately to 180^0 as he flexed the shoulder or placed his arm on his lap. He could also keep his fingers semi-extended during wrist extension. At the end of PT, Patient 5 could flex the shoulder to about 130^0, flex the elbow approximately to 90^0, and raise the wrist (e.g., Part A) to approximately 152^0, which was approximately 12^0 over baseline, but his power remained the same. While wrist extension had almost doubled in range, averaged EMG remained about the same (approximately 15 μv at baseline and 17 μv Phase I [e.g., Part A]). He could also grip a bottle with his left hand to uncork it with his right.

Phase II: BF -

There was no change during BF training. The EMG TV display and accoustics reflected the very severe lack of power in the limb (gain settings were, of course, maximal). There was essentially no difference in range of motion, but there was a slight increase in rate for the last two tests of BF.

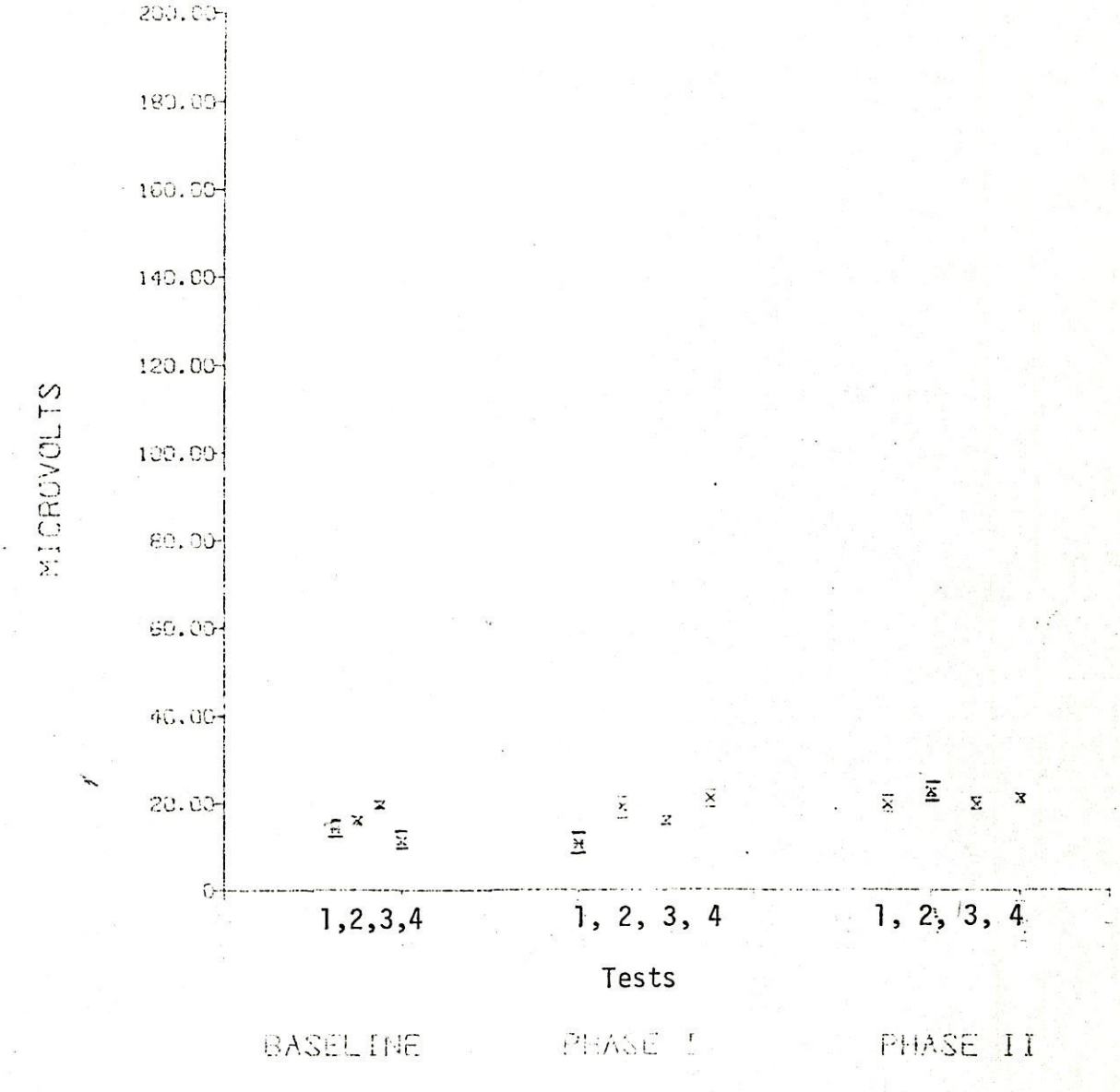
Comments:

Patient 5 claimed to have improved overall in that he could now hold his paretic limb abducted to wash it, turn water faucets with the left hand, pick up small articles like a pencil, use the shift key on his typewriter with his left thumb, hold paper down when writing longhand, and control an envelope to put a letter in it, all with the paretic side.

The patient often felt a "pull" in the "wrong" place during wrist extension--for example, along the inner arm, the wrist flexors, or fingers. He also had days of severe pain in both the hemiparetic upper and lower extremities. A needle electrode EMG examination (see p. 267) ascertained that there was no contributing neuropathy or myopathy to account for poor motor performance.

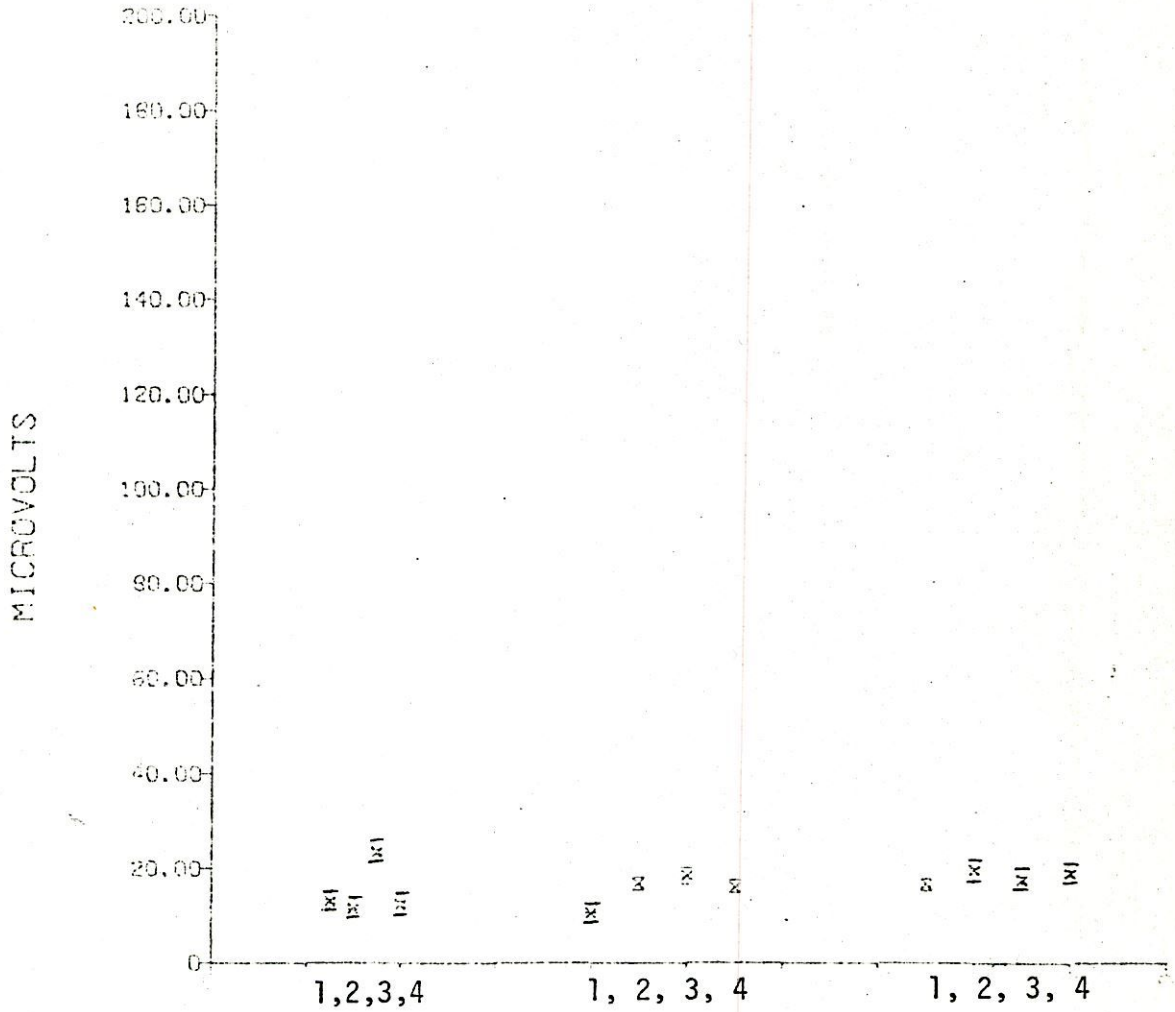
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 5 PART A (OPP. LIMB 211 , 188)

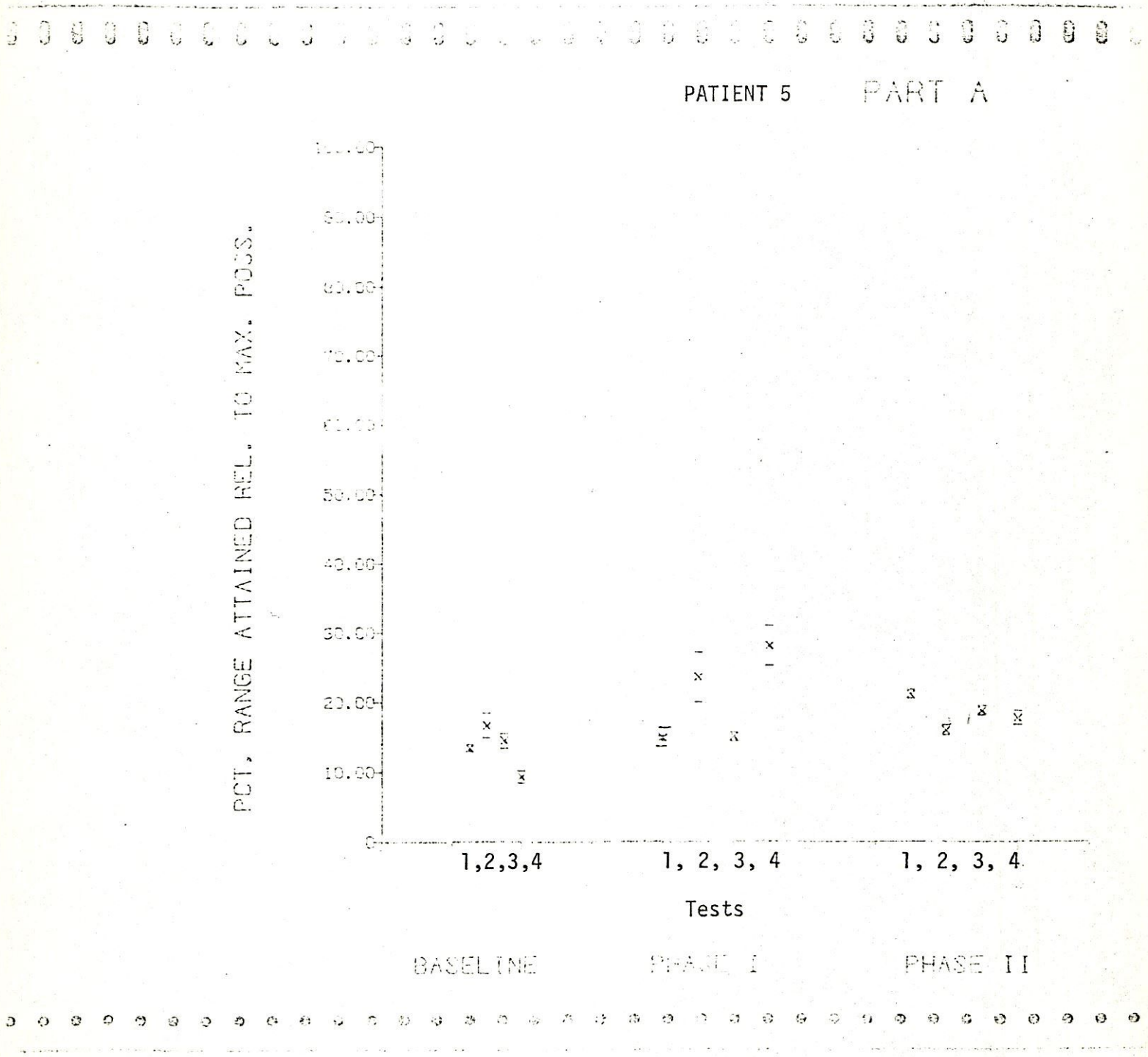


Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 5 PART B (OPP. LIMB 179 , 171)

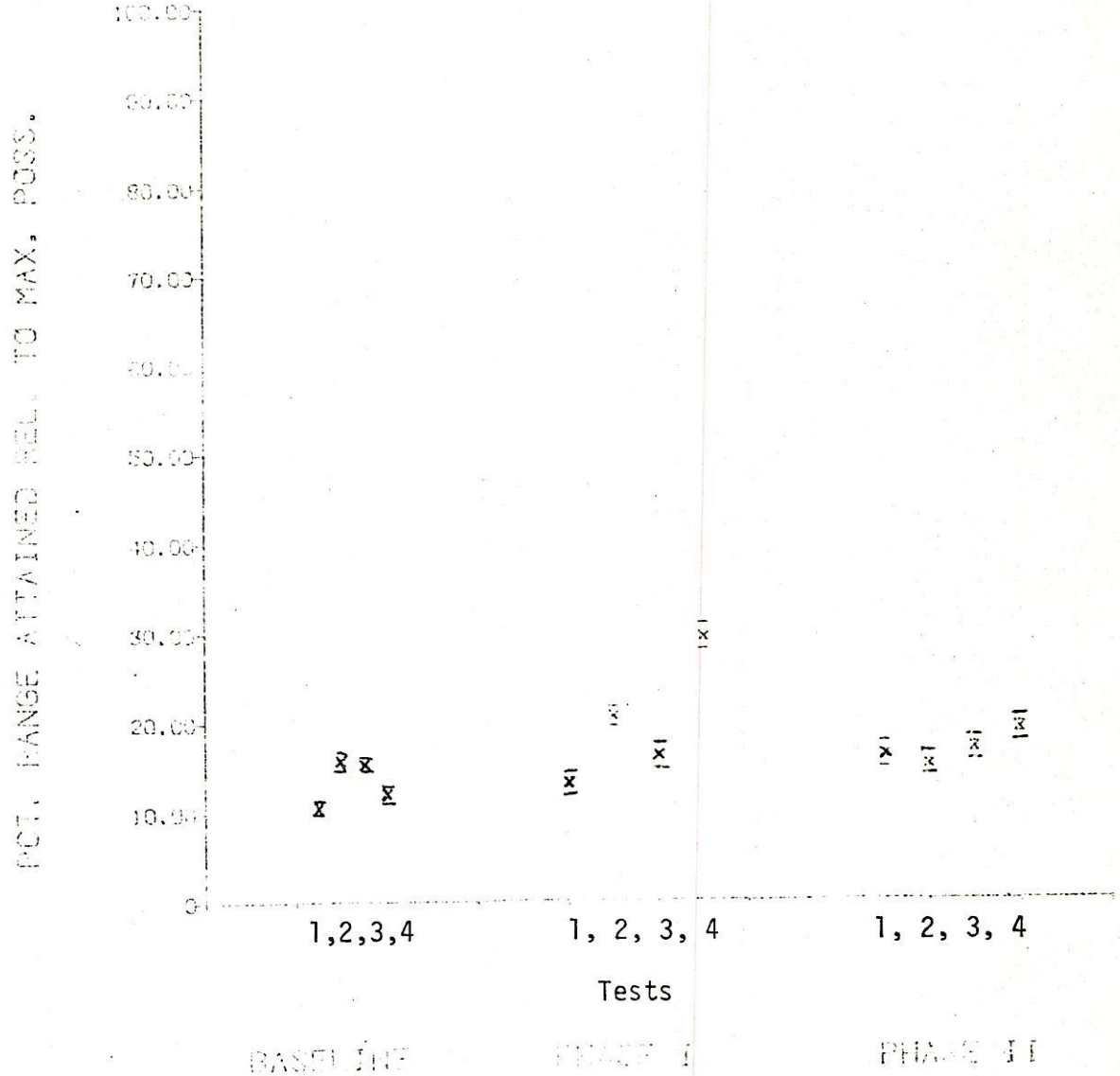


Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)



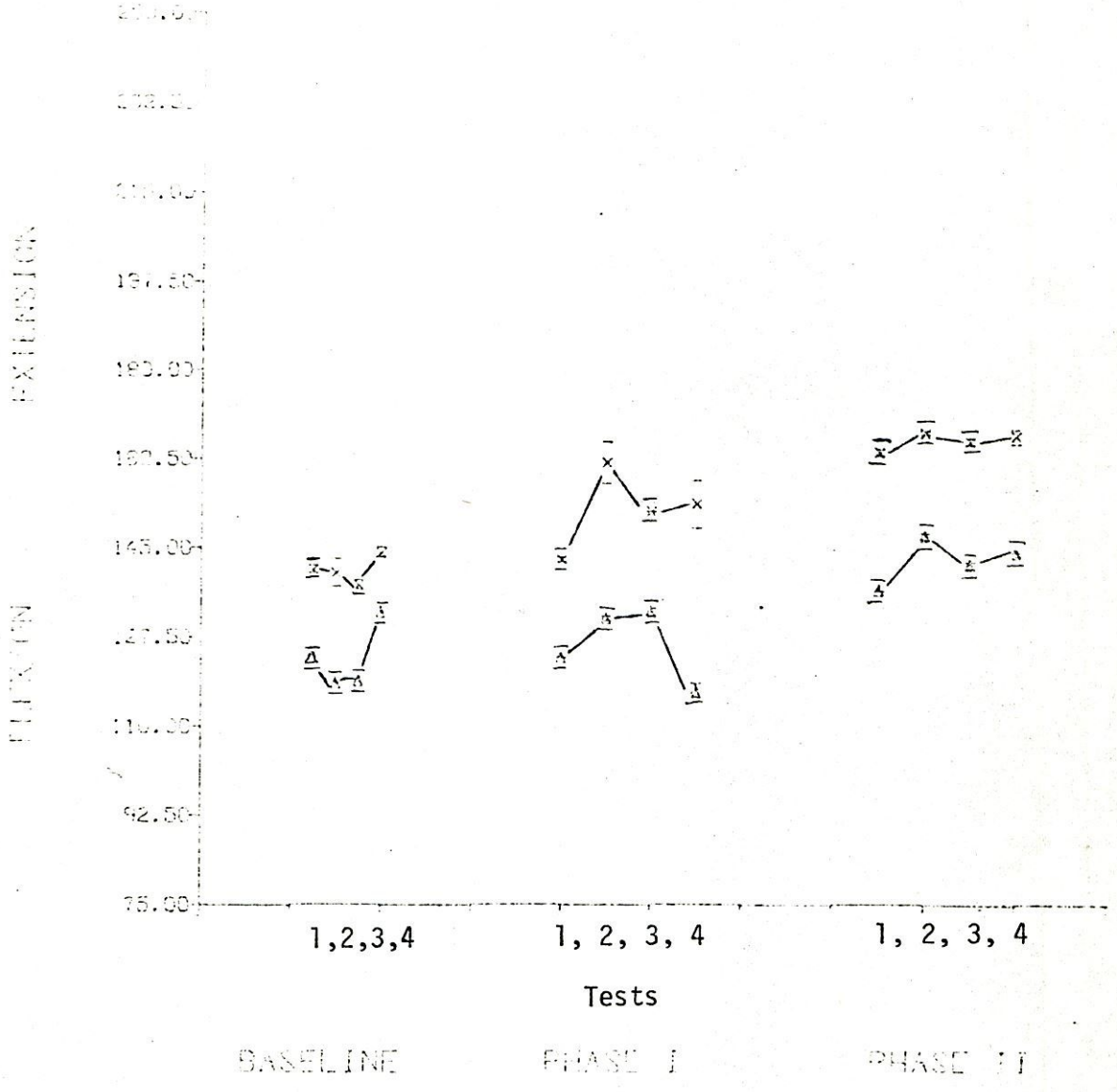
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 5 PART B



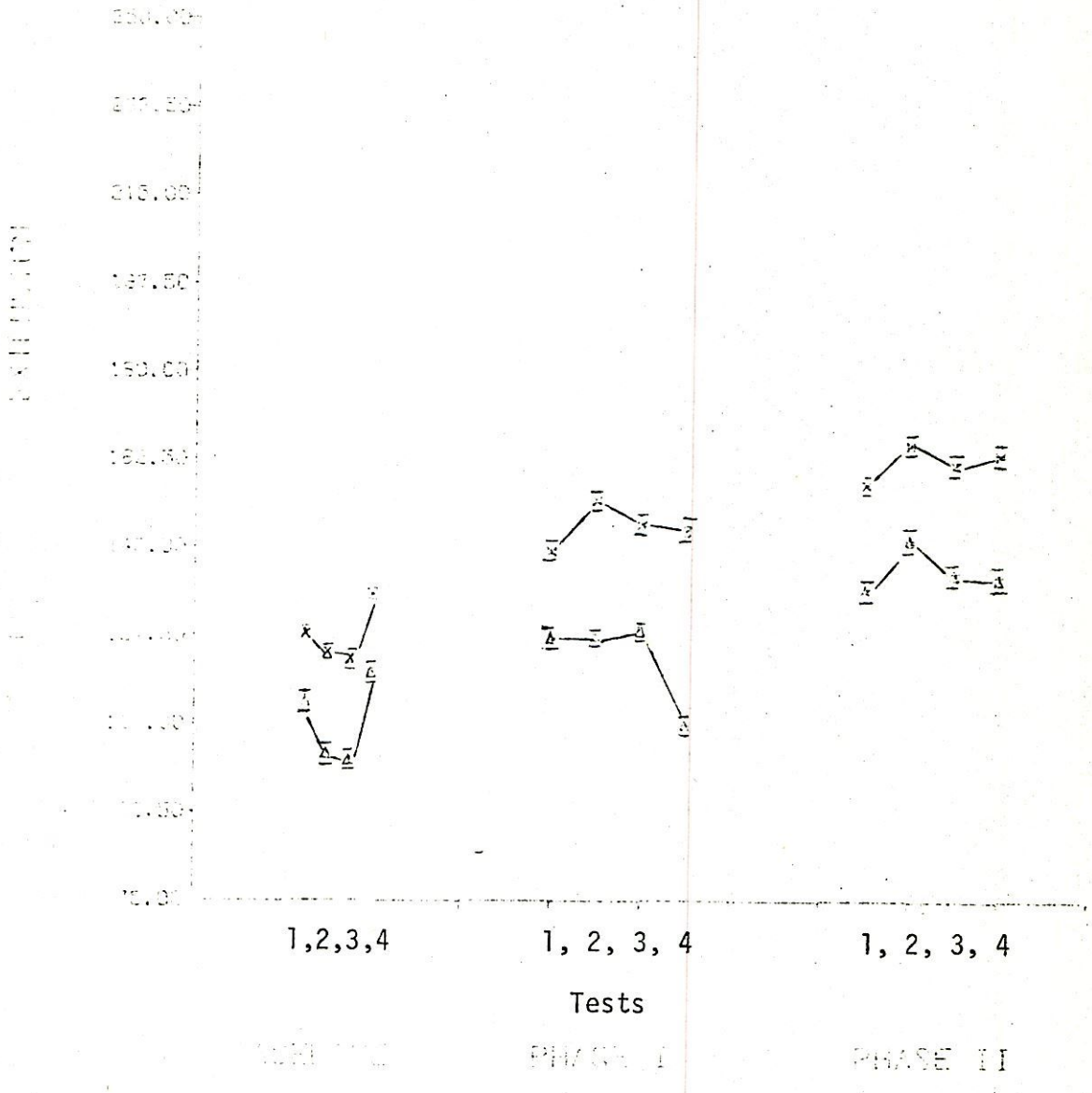
Extension ($\bar{X}$) and Flexion ($\bar{\Delta}$) in Degrees (SE indicated)

PATIENT 5 PART A



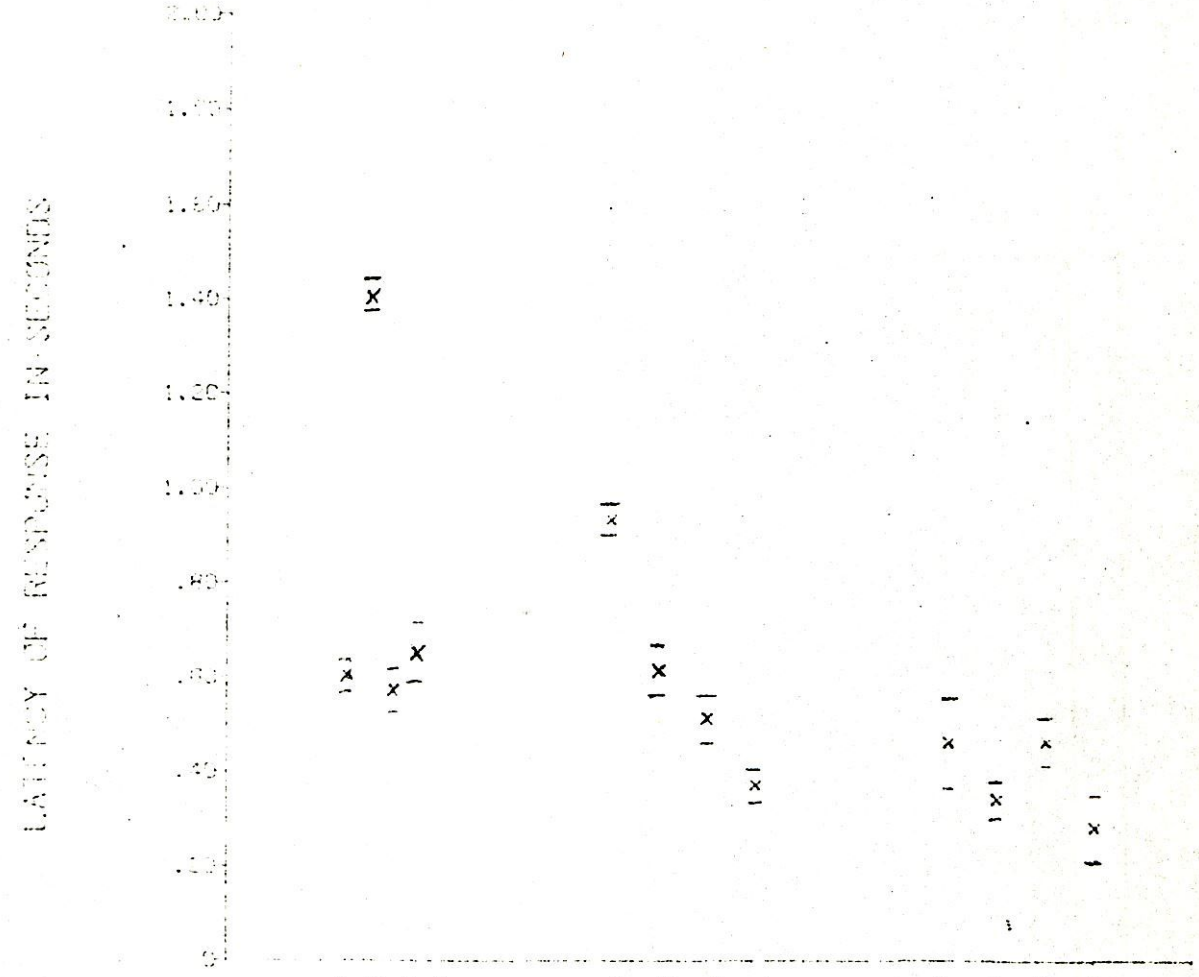
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 5



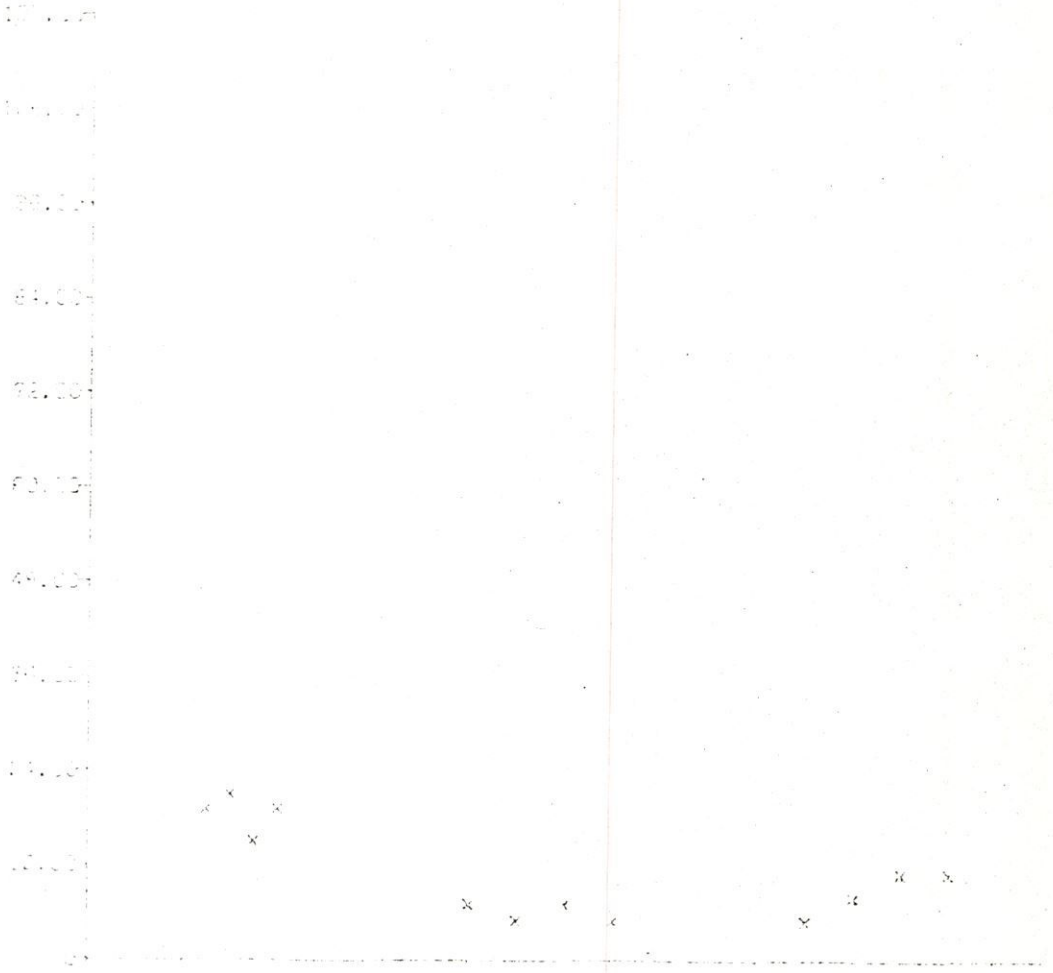
Latency (SE indicated)

PATIENT 5



Repetition of Movement

PATIENT 5



Patient 6

MMPI

L	50
F	60
K	53
Hs	62
D	56
Hy	62
Pd	55
Mf	51
Pa	53
Pt	58
Sc	73
Ma	70
Si	49
Es	43

Code

89'13724-'(51)^{0}4,7,14

Patient 6

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	85
Verbal IQ (VIQ)	92
Performance IQ (PIQ)	78
WAIS Verbal Subtests:	
Comprehension	10
Mathematics	8
Digit Span	9
Vocabulary	9
WAIS Performance Subtests:	
Block Design	8
Picture Arrangement	5
Object Assembly	4
Porteus Maze Test	10.5
Trail Making Test, Part A	105"-0 errors
Trail Making Test, Part B	371"-4 errors
Bender-Gestalt Recall	0
Wechsler Memory Scale (WMS) Quotient	79
WMS Subtests:	
General Information	6
Orientation	5
Mental Control	6
Memory Passages	0.5
Digits Control	9
Visual Reproduction	1
Associates Learning	7.5
Aphasia Screening Test (AST)	7-6 verbal, 1 non-verbal errors
LH Tactile Formboard I, Standard	43"-10 objects placed
RH	
LH Tactile Formboard II, Blindfolded	300"-2 objects placed
RH	

Patient 6, 62 yr old black male, 2 strokes; first stroke 1 yr, 6 mos ago; second stroke 1-1/2 weeks later [1 yr 6 mos poststroke (second and last stroke)]

Clinical Diagnosis:
(1) Moderate obesity
(2) Hypertension
(3) Embolism (or thrombosis?) middle cerebral artery
(4) Right spastic hemiparesis

Prior Handedness: Right

Prior Therapy: 4 mos total; arm and leg

Comments: None

NEUROLOGICAL EXAMINATION

Description of Weakness:

Spastic right hemiparesis with little or no movement at the finger and wrist which shows severe spasticity and contracture. 2/5 right elbow and right shoulder, 2/5 knee, ankle and toes. Sustained ankle, knee and elbow clonus on the right. Minimal right facial weakness.

Description Sensory Impairment:

Pin: Mildly decreased pin of right hand and foot.

Touch: Mildly decreased touch in right hand and foot.

Vibration: Normal

Position Sense: Poor in right hand.

Two point discr: Intact

Graphesthesia: Normal

Stereognosis: Significant loss in right hand.

Aphasia:

Dysarthria.

Apraxia:

None.

Comments:

Impression: Severe spastic right hemiparesis. Maximal right hand and fingers with severe spasticity and contracture. Absence of severe speech disturbance in presence of severe hemiparesis suggests deep seated infarction rather than cortical.

Maximum angle of wrist extension due to wrist flexor contractures: approx 190^0 to 200^0.

Training Objective: Wrist extension

Phase I: BF

Phase II: PT

Introduction:

Patient 6's right upper extremity had excessive tone and was severely spastic with a highly obligatory stereotyped pattern. He carried his arm in a flexed elbow position of 90^0 with a pronated wrist,

fingers flexed, thumb adducted. Any attempts at movement resulted in an invariable pattern of back extension, rotation of the body to the hemiplegic side, extreme shoulder elevation and abduction, some external rotation (approximately 30^0), elbow flexion, a pronated wrist, and a tightly clenched fist. When he tried to extend the wrist in the standard position with the forearm resting on the table and the hand flexed over the edge, all the above came into play. The wrist extended (e.g., Part A) to approximately 153^0 and supinated to mid-position. Range, Part B (Rate), at baseline was somewhat limited relative to range, Part A (Command), as the patient did not achieve as good a relaxed flexion position during the Rate situation before initiating a new extension response. Patient 6 had limited abduction of the shoulder and contractures of the wrist. Maximum angle of passive wrist extension was approximately 190^0 to 200^0. The elbow dropped to extension when he was in a quiet sitting or standing position and he could voluntarily and slowly flex the elbow approximately to 90^0 but was unable to extend it. He was unable to "hold" (maintain) a movement to any degree. Any general body movements caused hyperreflexive spastic activity in the upper extremity. Patient 6 wore a splint for his hand a good deal of the time.

Patient 6's stroke was a severe blow to his mental and motor energy. Although alert, oriented, perfectly comprehending, and perfectly able to get about in transfers, dressing, hygiene, and eating, the amount of effort he could expend on any activity was limited. In other words, he was slowed down to a level of a man 30 years his senior. He lived comfortably in his home with a lady friend and had every need attended to. He did not practice at all at home for the first five to six weeks of

training partly because he felt "therapy" was sufficient and partly because of his easy fatigue and limited strength. Patient 6 favored looking at his wrist.

Phase I: BF -

The first approach was to allow Patient 6 to raise the wrist freely from a standard flexed position over the edge of the table. However, due to the pattern described above there was, in fact, very little wrist extension. Instead, movement amounted to the repeated occurrence of the stereotyped pattern consisting largely of shoulder abduction and retraction which forced the table to roll away each time. Thus, Patient 6 was strapped at the shoulder and forearm and the table was strapped to his chair. Even the strappings did not prevent complete disarrangement of the arm, wrist, and table as Patient 6 continued to do all else instead of, or in addition to, wrist extension. Extension was thus only a terminal component embedded in a gross motor response. Wrist extension was also very transient. He fatigued immediately upon each attempt as well as rapidly over time. Patient 6 was given repeated instructions to not pull back but rather only to raise the wrist. The whole procedure of wrist extension through BF was an illogical approach as so much else had first to be abolished before the patient could be in a position to extend the wrist in an optimal way. Yet BF training progressed, along with verbal stress on the fact that Patient 6 was not extending the wrist so much as pulling his arm up, back, and away at every attempt. In spite of this pattern the averaged EMG and range of motion for wrist extension continued to increase significantly during BF. There was also now no significant discrepancy between range Part A and B, but rate of movement during

the latter (Rate) situation was very much reduced. Wrist extension was developing into a single movement, isolating itself apart from the stereotyped pattern. This is all the more notable in the face of the obligatory nature of the stereotyping of his motion. Patient 6 was always well aware of a "pull" in his wrist extensor muscles when they contracted (his report).

Due to E's instructions, by the time Patient 6 had completed BF, he had eliminated shoulder elevation and abduction and did not even need his limb strapped. This overall change paralleled a slight decrease in his average EMG at test 4 of BF, but the movement was, to a much greater extent, the explicit, required one. At the end of BF, he could also flex the elbow to a greater degree, and extend it, albeit weakly.

Phase II: PT -

By the time PT began, Patient 6 was in somewhat better condition to extend the wrist. However, his muscles were still hypertonic so that one element of PT was general relaxation. After the second week of PT, it was carefully explained to Patient 6 that he would have to practice at home. By the beginning of the third week of PT, Patient 6 had made significant gains with practicing at home. The arm was highly relaxed, and hung at his side as he walked. He was able to swing the shoulder in a relaxed anteroposterior motion involving rapid alternation of shoulder flexor and extensor muscles. After passive ROM at the shoulder from E, he could hold his arm extended at approximately 90^0 without flexing the elbow, and continue extension of the arm to 180^0, weakly, but voluntarily. He could also flex and extend the elbow as

before and had some voluntary finger flexion, to the point of controlling the thumb somewhat apart from the other four digits. Finger movement did not exist prior to training. Patient 6 could not feel the finger movement.

In spite of the above changes, wrist extension, although the focal point of therapy, remained transient and fatigue was extremely rapid, as before. The extensors always fatigued the moment peak angulation occurred and the wrist fell slowly into flexion. Facilitation and resistance were used for wrist extension. Resistance, however, could be carried on, although quite forcefully, only for a relatively short period of time. Resistance was thus used intermittently with other procedures and it was also used on other muscles. At the end of PT, Patient 6's limb was relaxed and he was evaluated, ironically, as only now being ready for functional neuromuscular re-education.

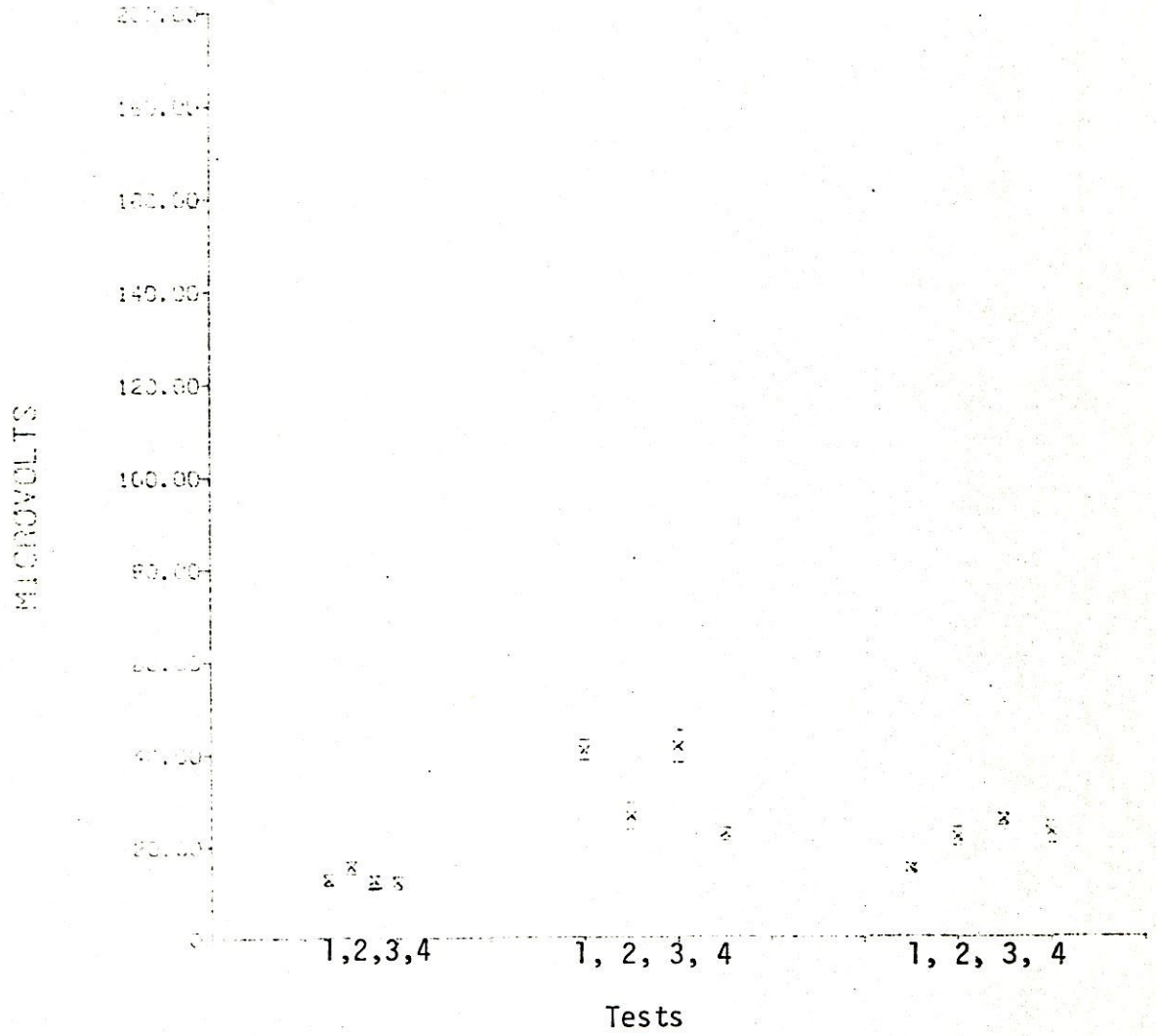
Comments:

At the beginning of the experimental program, in retrospect, Patient 6 really appeared to have an extremely poor prognosis for learning any improved motor control. He showed distinct improvement in wrist extensor activity isolation during BF training. However, during the PT period, a greater opportunity was presented for dealing with the many other aspects of his motor control problems in addition to the wrist extensor deficit. Thus, the most salient features of this case are 1) the relative efficacy of BF when used together with intensive guidance and instruction in this case for isolated wrist extensor contraction--though the BF stage was fraught with insurmountable problems of the obligatory stereotyping; and 2) the abolition of a highly obligatory pattern during the PT phase, leaving the arm "cleared" for new habits or repertoires.

Patient 6 is missing scores for test 1, Phase I, and test 2, Phase II on graphs of angular motion because of a goniometer equipment failure.

Averaged EMG Activity in Microvolts (SE indicated)

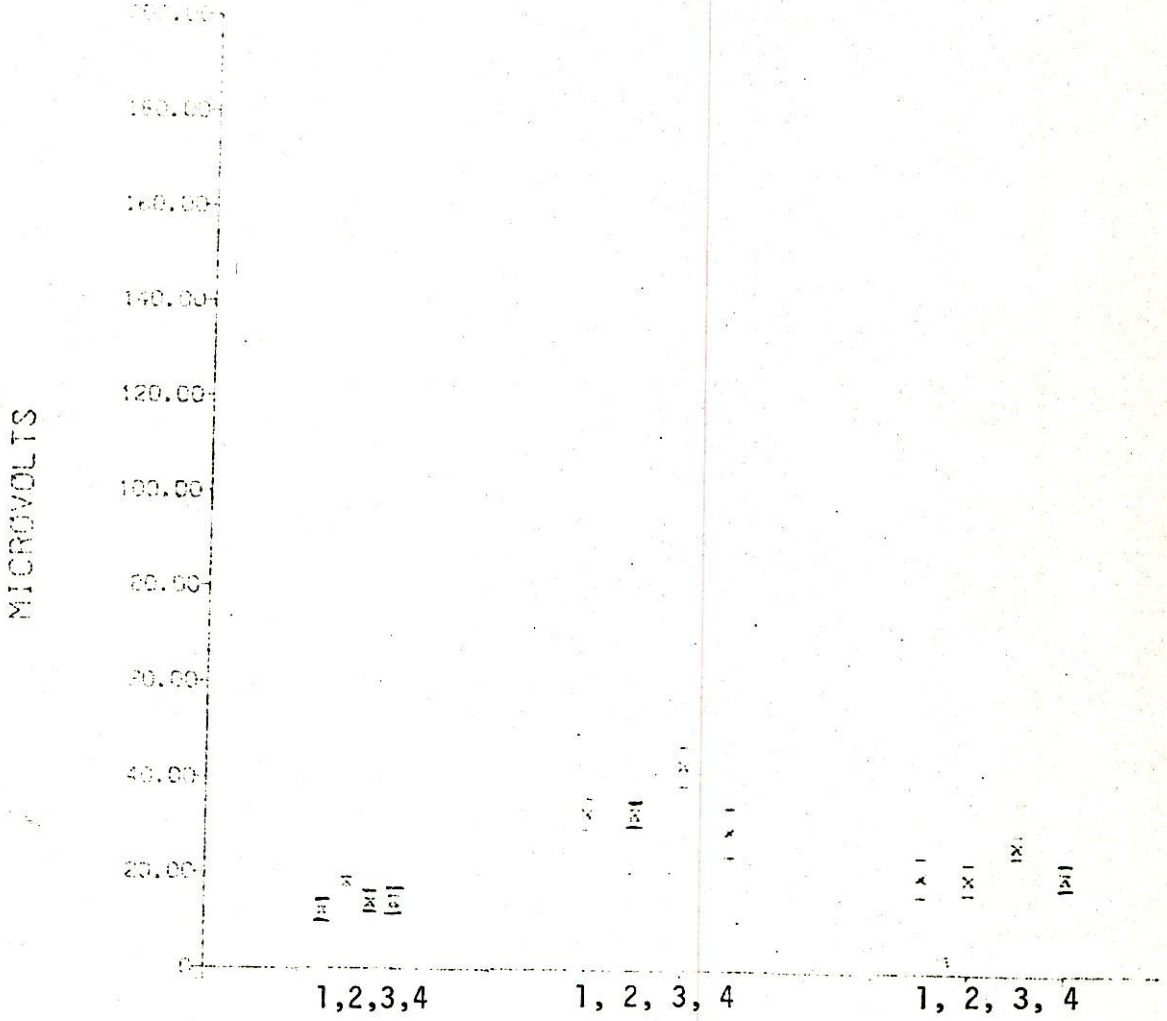
PATIENT 6 PART A ([illegible] 172 : 175)



[illegible] PHASE II

Averaged EMG Activity in Microvolts (SE indicated)

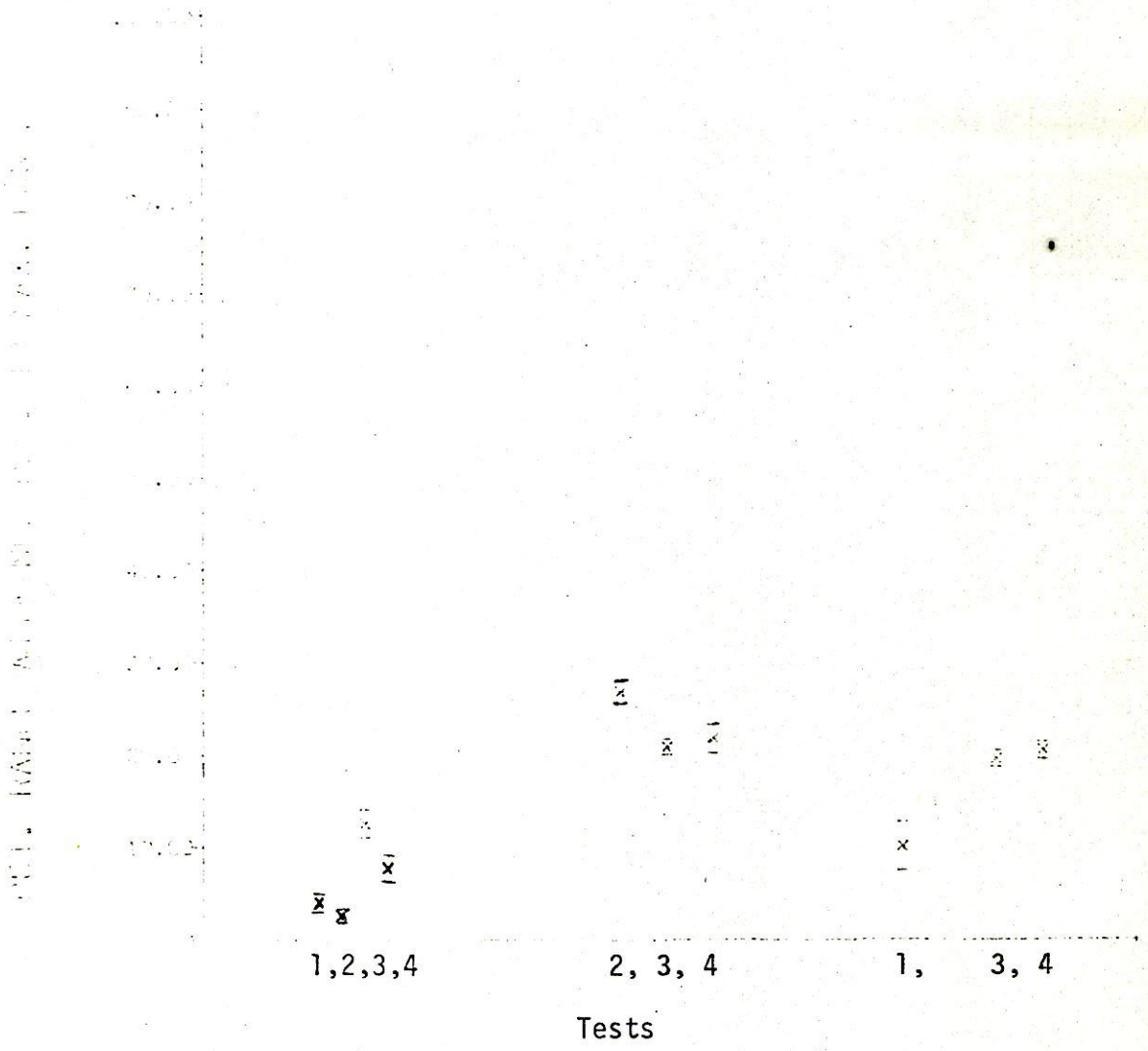
PATIENT 6



PHASE II

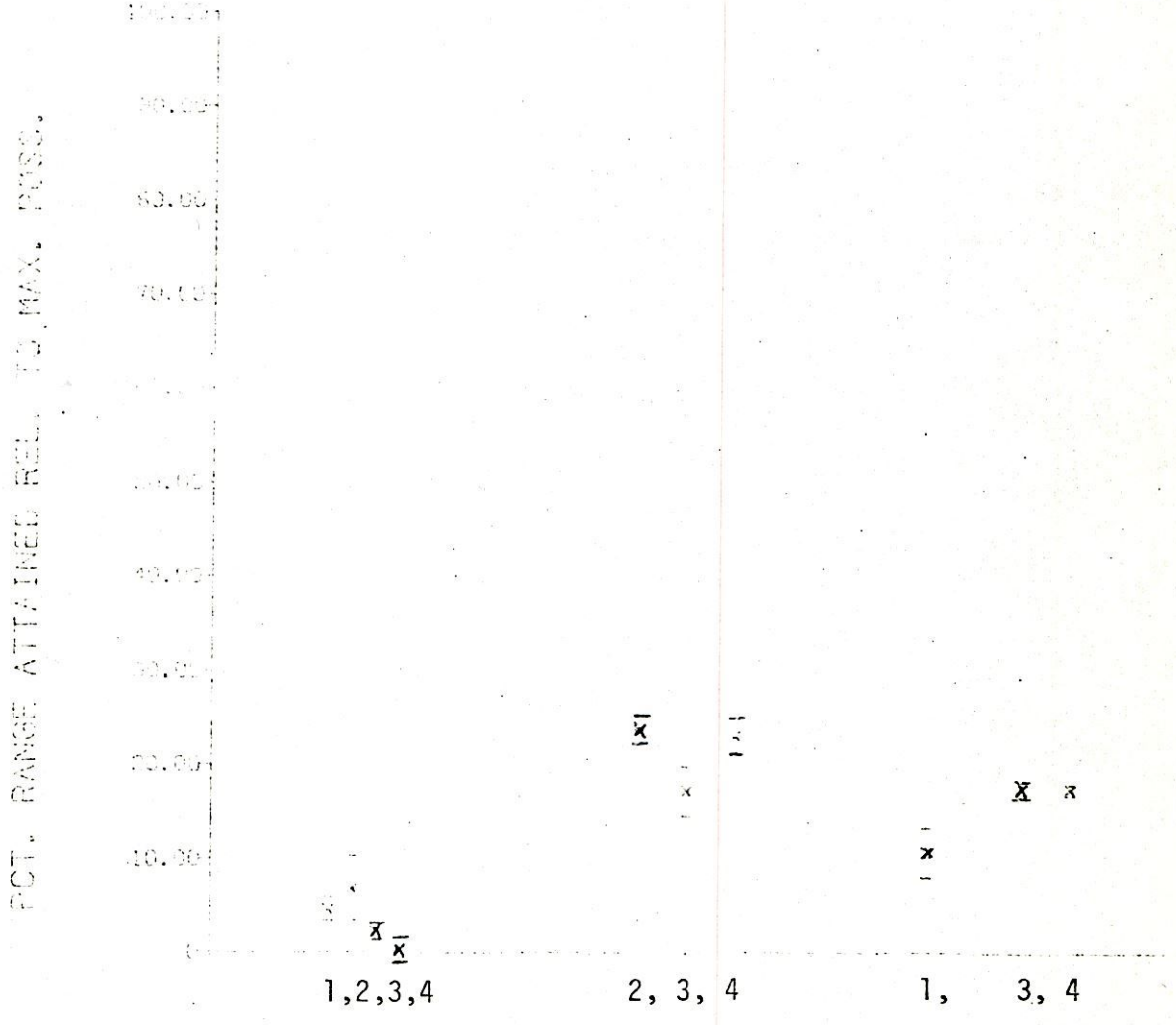
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 6 PART A



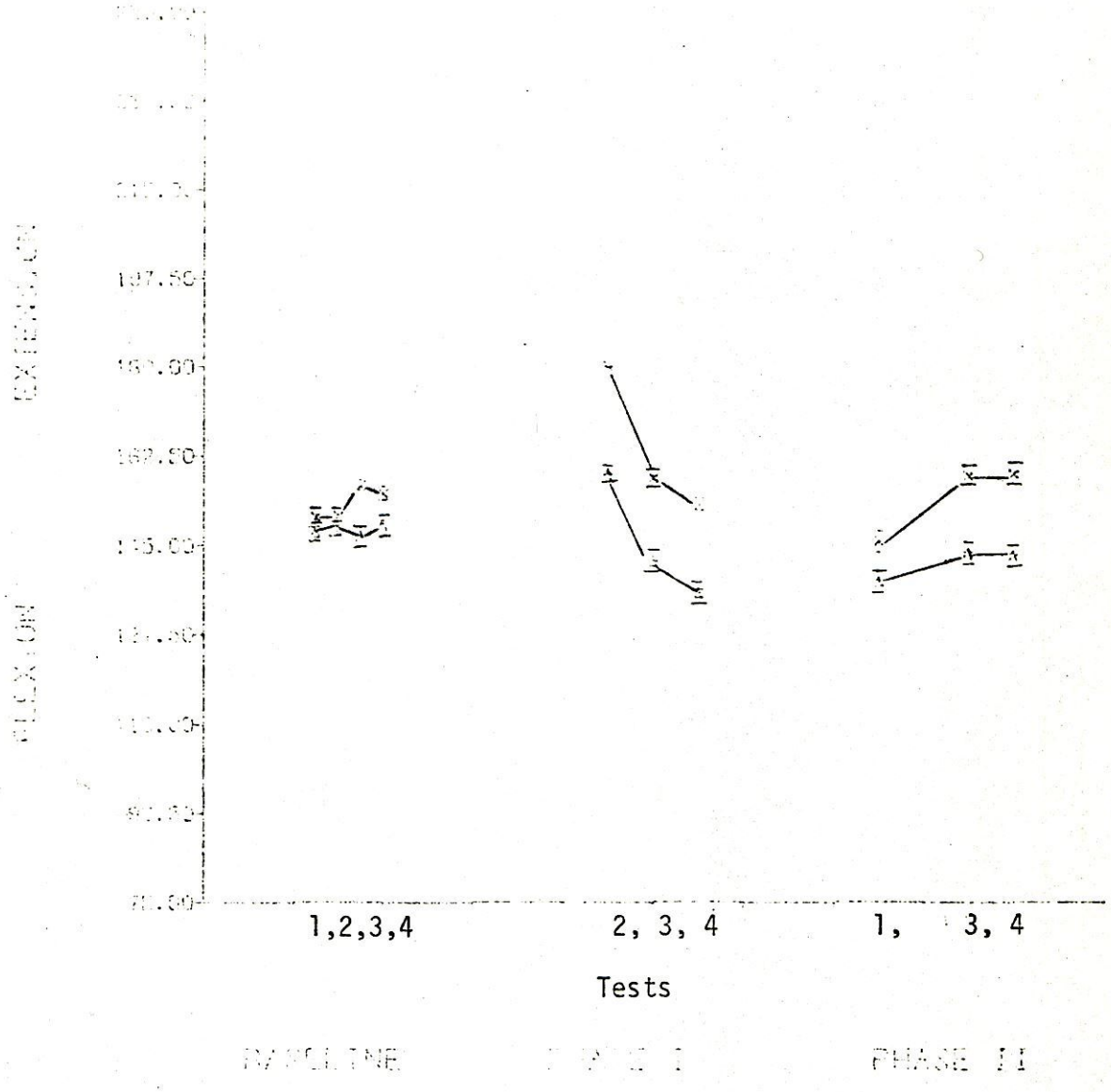
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 6 PART B



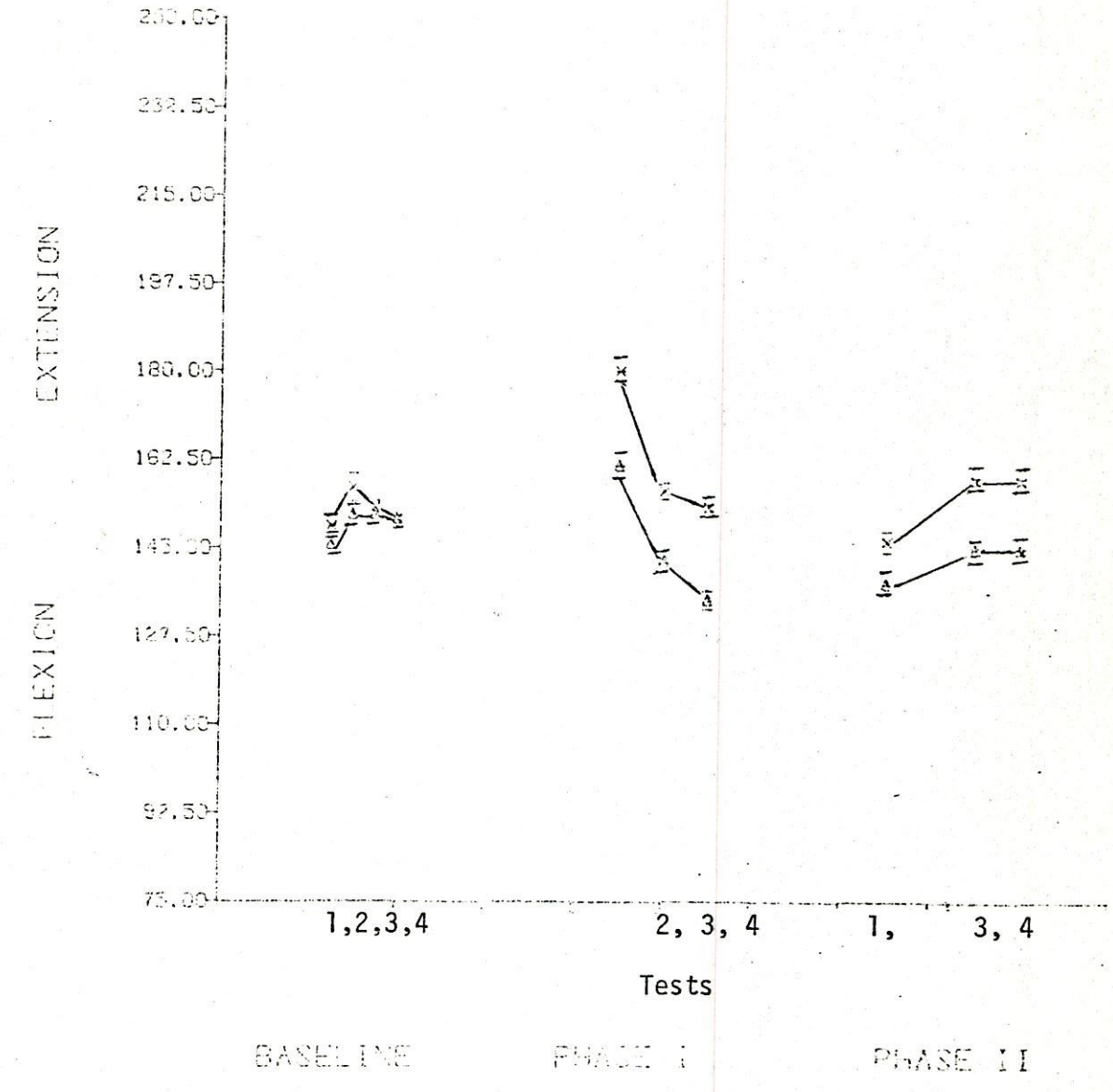
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 6



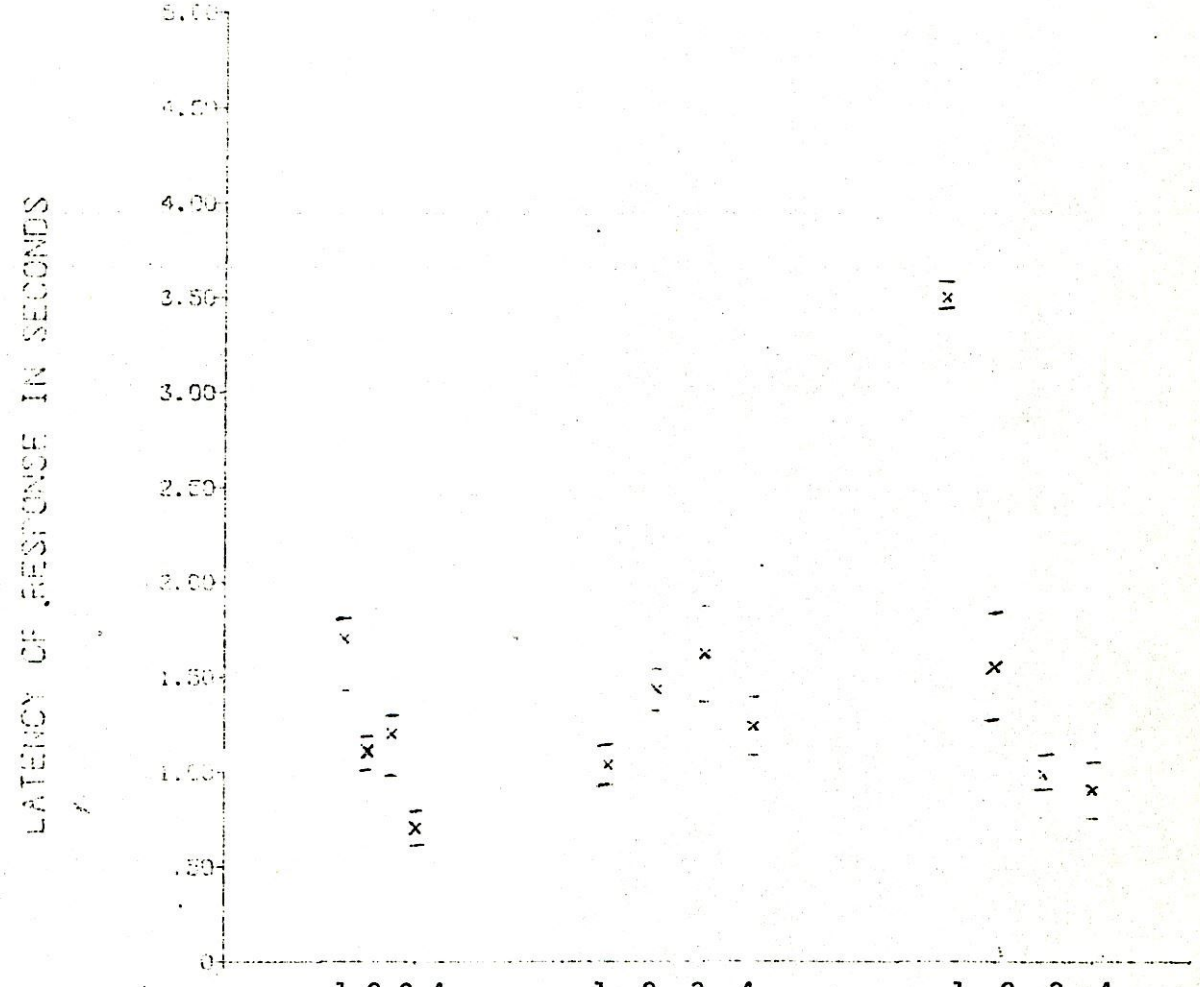
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 6 PART B



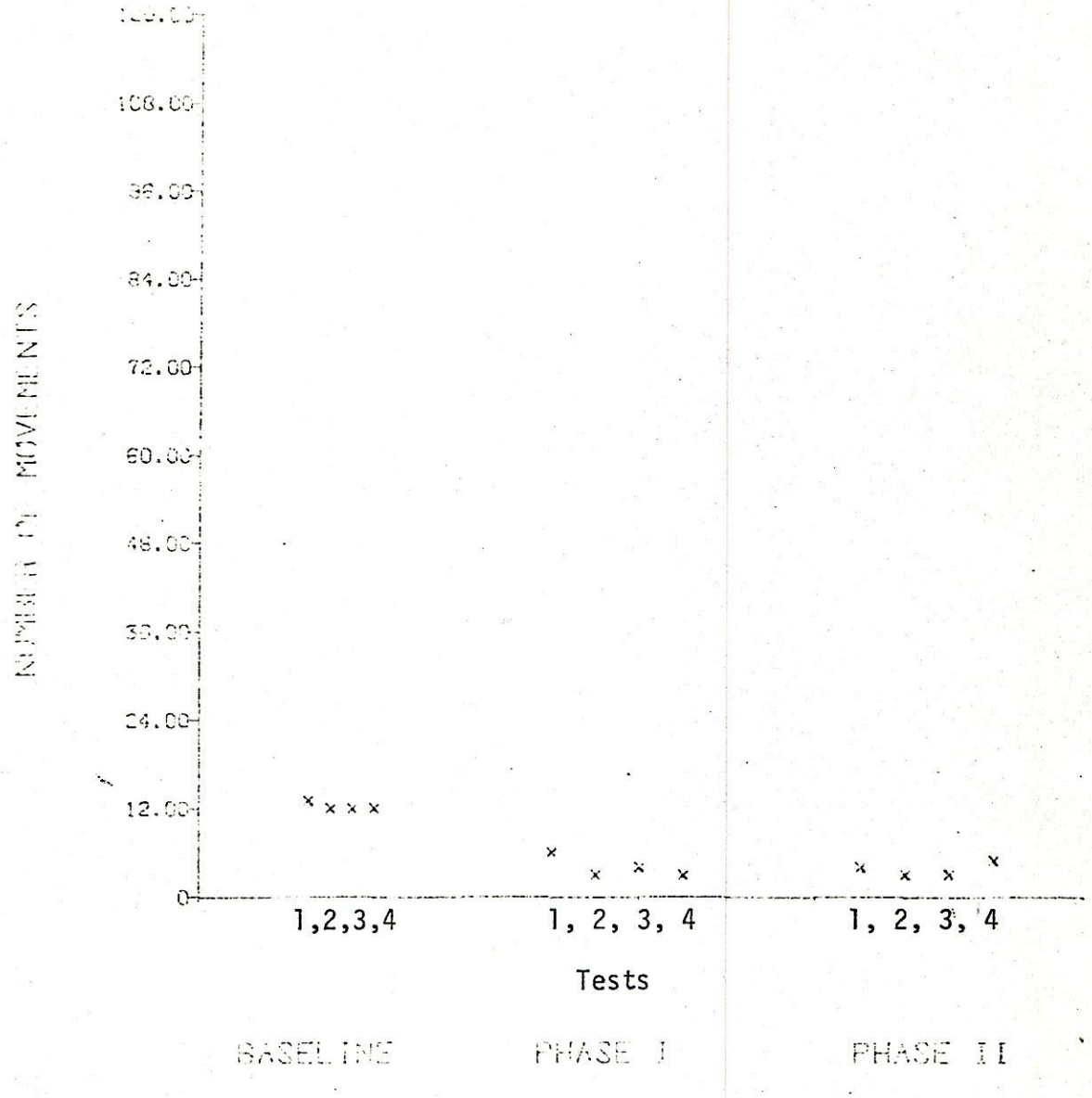
Latency (SE indicated)

PATIENT 6 [illegible] A ([illegible] .5 , .4)



Repetition of Movement

PATIENT 6 - A (CH. LIMB 81 , 84)



Patient 7

MMPI[40]

L	44
F	62
K	49
Hs	62
D	72
Hy	67
Pd	62
Mf	39
Pa	56
Pt	52
Sc	65
Ma	63
Si	49
Es	35

Code

2' 389146-'(39)[98]2,8,12

[40] This patient left 98 questions randomly unanswered throughout the MMPI and thus his profile is of questionable validity.

Patient 7

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	95
Verbal IQ (VIQ)	107
Performance IQ (PIQ)	80
WAIS Verbal Subtests:	
Comprehension	12
Mathematics	10
Digit Span	13
Vocabulary	10
WAIS Performance Subtests:	
Block Design	6
Picture Arrangement	8
Object Assembly	5
Porteus Maze Test	8.5
Trail Making Test, Part A	180"-0 errors
Trail Making Test, Part B	244"-5 errors
Bender-Gestalt Recall	1
Wechsler Memory Scale (WMS) Quotient	92
WMS Subtests:	
General Information	3
Orientation	3
Mental Control	9
Memory Passages	5
Digits Control	12
Visual Reproduction	2
Associates Learning	9.5
Aphasia Screening Test (AST)	0-0 errors
LH Tactile Formboard I, Standard	33"-10 objects placed
RH	
LH Tactile Formboard II, Blindfolded	300"- 1 object placed
RH	

Patient 7, 62 yr old white male, 3 strokes; first stroke, 2 yrs ago, second stroke 10 mos later, third stroke 1 mo later--(1 yr poststroke [third and last stroke]).

Clinical Diagnosis: (1) Hypertensive arteriosclerotic cerebrovascular disease
(2) Right middle cerebral artery thrombosis

Prior Handedness: Right

Prior Therapy: 3 mos total; arm and leg after first stroke; leg only after second and third stroke

Comments: Left limb apraxia, upper and lower extremities

NEUROLOGICAL EXAMINATION

Description of Weakness:

Hypotonic left hemiparesis predominantly of the left upper extremity. Strength in left upper extremity does not exceed 2/5; strength is 3/5 at the hip, 2/5 at knee extension, and flexion. Bilateral hyporeflexia; the left knee and ankle reflex are about the only ones which are elicitable. Hypotonia on left with strong left Babinski sign.

Description Sensory Impairment:

Pin: Mild decrease of sensation to pin prick over left upper extremity from elbow down.

Touch:	Mild decrease of sensation to touch over left upper extremity from elbow down.
Vibration:	Intact
Position Sense:	Impaired in left upper and lower extremities.
Two point discr:	Increase in two point discrimination sensitivity from 1 mm to 6 mm on the left side.
Graphesthesia:	Impaired on the left side.
Stereognosis:	Impaired on the left side.

Speech:

Hoarse

Apraxia:

Marked apraxia of left upper extremity.

Comments:	There is no neglect of hemiplegia; severe flaccid left hemiparesis.
Training Objective:	Biceps contraction.

Phase I: BF

Phase II: PT

Introduction:

Patient 7 had a dense left hemiparesis. His upper extremity hung flaccidly, fingers clenched. The elbow flexed when he yawned. Initially, elbow flexion was elicited with facilitation. There was, however, no ability to hold the elbow flexed. When Patient 7 did initiate elbow

flexion himself it usually took a great while for him to "warm up" to it. Active range of motion was always the same (approximately 20^0), the greater part of the movement being extreme shoulder elevation and some adduction. Movement components of flexion and adduction included crossed extensor and tonic neck reflex activity. Patient 7 could also abduct the shoulder approximately to 50^0. At first during training, shoulder elevation and adduction were the only true aspects of movement. Instructing Patient 7 to inhibit either was to no avail. He practiced elbow flexion very little, if at all, at home. Once early in training, Patient 7 observed himself "flexing" the elbow in a mirror. The undesired features of his response seemed to make little impression on him. It was very difficult for him to understand the slightest suggestions of what was required of him in flexing the elbow other than his perception of <u>any</u> motion of his upper paretic side which is what bend the elbow meant to him behaviorally.

Patient 7 had difficulty locating his left arm (poor position sense) when he had to feel for it with his right hand behind his back. There was some neglect of his left body side resultant from the right sided brain lesion. For example, he was observed on one occasion to have his eyeglasses sitting diagonally on his face with the right lens over the right eye and the left lens over the left forehead area. Patient 7 was oblivious to this disarrangement.

Patient 7 also exhibited a sense of futility and lack of motivation for attentiveness to a "useless" side. He lived in a nursing home where he got minimal care and attention. He had no relatives. There were no facilities in the home to walk or use the left arm. He had sunk into a position of being crippled, at the mercy of his environment,

and at experimental outset, was most concerned with being maltreated by anyone as little as possible.

Phase I: BF -

Patient 7 was at first (during baseline) too cautious to hope for improvement. He was motivated yet suspicious and without hope at the same time. During BF, he got great joy at seeing things happen from his body on TV. It surprised and satisfied him to even see any electrical activity ("aliveness") come from his biceps. This spurred him on to increment the EMG dot and he tried until fatigued during experimental sessions. At home he practiced little due to new diversions, lack of sustenance, improper setting, etc. His attention span was excellent during experimental sessions except about three or four times in the first two weeks of BF when days were August hot and he was "under the weather". His head drooped and he was mentally fuzzy. He also was incontinent on one or two of those days (at his nursing home), where he may have had minor seizures. During weeks 3 and 4 of BF he "came around" (the weather got cooler too) and his averaged EMG increased to a degree he had never approached before ([e.g., Part A] from approximately 20 μv and 17 μv, weeks 1 and 2, to approximately 71 μv and 49 μv, weeks 3 and 4). In general, Patient 7 was highly variable in his EMG response. In spite of the averaged EMG increase, Patient 7 did not invariably flex nor increase his range incrementally over time, as this was a more difficult thing for him to do than to recruit motor units. That is because he had a limb apraxia. To get Patient 7 to angulate more, E told him to "make power", a "muscle" as in biceps flexion, or punch his own nose, etc. Often, however, his flexions were minimal, particularly at warm up.

By the third and fourth weeks of BF, Patient 7's replies to queries by E of whether he noticed differences in his arm was "ya, it's easier" and he knew good attempts from bad because the muscle "gets tight" and "has more umph". Yet he did not know how to bend the elbow per se. During BF training, it wasn't until the fourth week that he said (as his averaged EMG really shot up during one response) "ok, I think I finally got the idea". His muscle tone to touch was gradually increasing in the whole arm. He had a particular, large motor unit (repeatedly noticeable as the same one) which caused a good voltage and noise increase and which at first came into play after many attempts, and then came into play more quickly and easily. It could fire even if the elbow were not flexed (isometric biceps contraction), but Patient 7 were trying. Range of elbow flexion was variable and remained essentially unchanged. Patient 7 had trouble shutting off his motor units once he got them going. With his attention diverted the motor units would stop firing (i.e., he would go back to a "dead" neglected arm without realizing it). In sum, Patient 7 could make the connection to increase motor unit firing but he could not control biceps relaxation nor necessarily use his motor potential to flex the elbow as he was limb apractic.

Phase II: PT -

Gains made during BF carried over to Phase II. The patient may have continued to improve more had he remained on BF. During PT he got very specific instructions on what to do with his arm. He held steady with his gains. E taped a cup to his chest or put her finger on it and said "look at the goal and hit there and then look at the chair where your

arm should come down to rest and move the arm over and then down. Pay attention to where the arm needs to go." So the pattern was 1) "bend the elbow", 2) "now over", 3) and "down". E also had him abduct his arm specifically by looking to a goal in the desired direction. One session during the third week of PT, E examined wrist extension of which the patient had the very slightest amount, and only with facilitation, before any training. He could now voluntarily extend the wrist to about 50^0. This was accompanied three-quarters of the way through movement by arm extension. The patient could also protract and depress the shoulder with a fair amount of strength. (These movements were not evaluated before training.) Thus, these movements were done once in a while in PT but mostly there was intensive continuation of elbow flexion training until fatigue which got less rapid as time went on. Changes which occurred during the third and fourth weeks of PT were the patient's starting to move more slowly and controlledly, on occasion, during elbow flexion towards the target on his chest.

Comments:

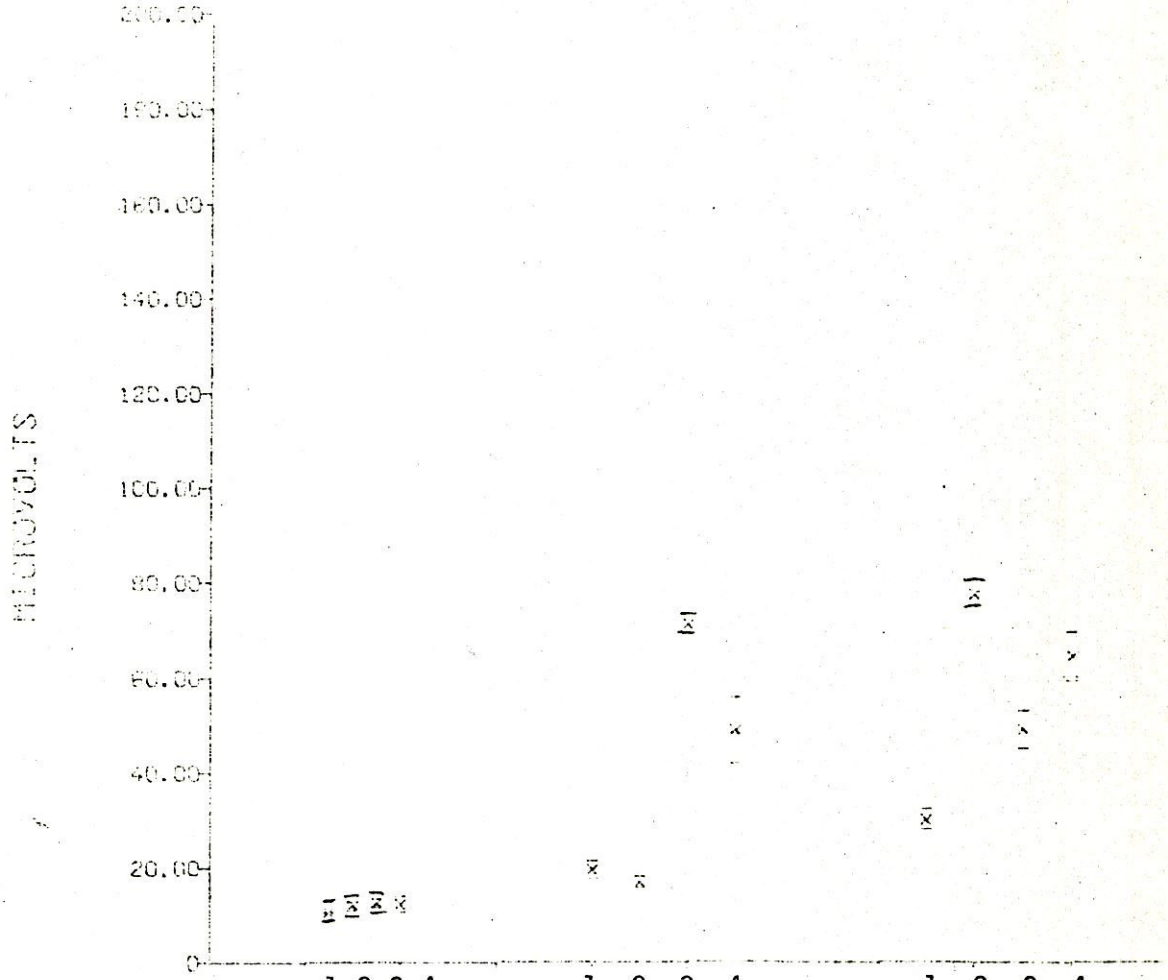
Patient 7 was confined to a wheelchair. Initially he answered that he could do many things, when questioned, that he was clearly unable to do, but was loathe to admit to himself more than anyone else. The patient needed help in transferring, dressing, hygiene, and any manipulations requiring more than his unaffected upper extremity. As training progressed, he "admitted" more about his arm and said, yes, he had trouble bending per se irrespective of motor power. Also, as training progressed, he would move his left arm for E without the aid of his right arm to put on his jacket, something he did not do initially. He

was unaware of this change. Once he realized that training would be ending, he reverted to assisting his left arm with his right on this task (again unaware of the change).

E would evaluate Patient 7, together with Patient 10, as one of the most logical types of patients likely to benefit from BF training. Patient 7 had virtually no movement in a flaccid limb. Minimal responses were elicitable by the PT technique of facilitation. Concomitant kinesthetic sensation at this level of output was, it would seem, essentially nil, thus providing little foundation upon which to build a motor repertoire (particularly as this patient had very poor sensory function). BF, on the other hand, seemed to afford a viable, alternative reafference channel for building motor control. The compounding problem of apraxia, however, if surmountable, seemed perhaps more so via a goal-directed movement technique learned in the context of mutual therapist-patient interplay.

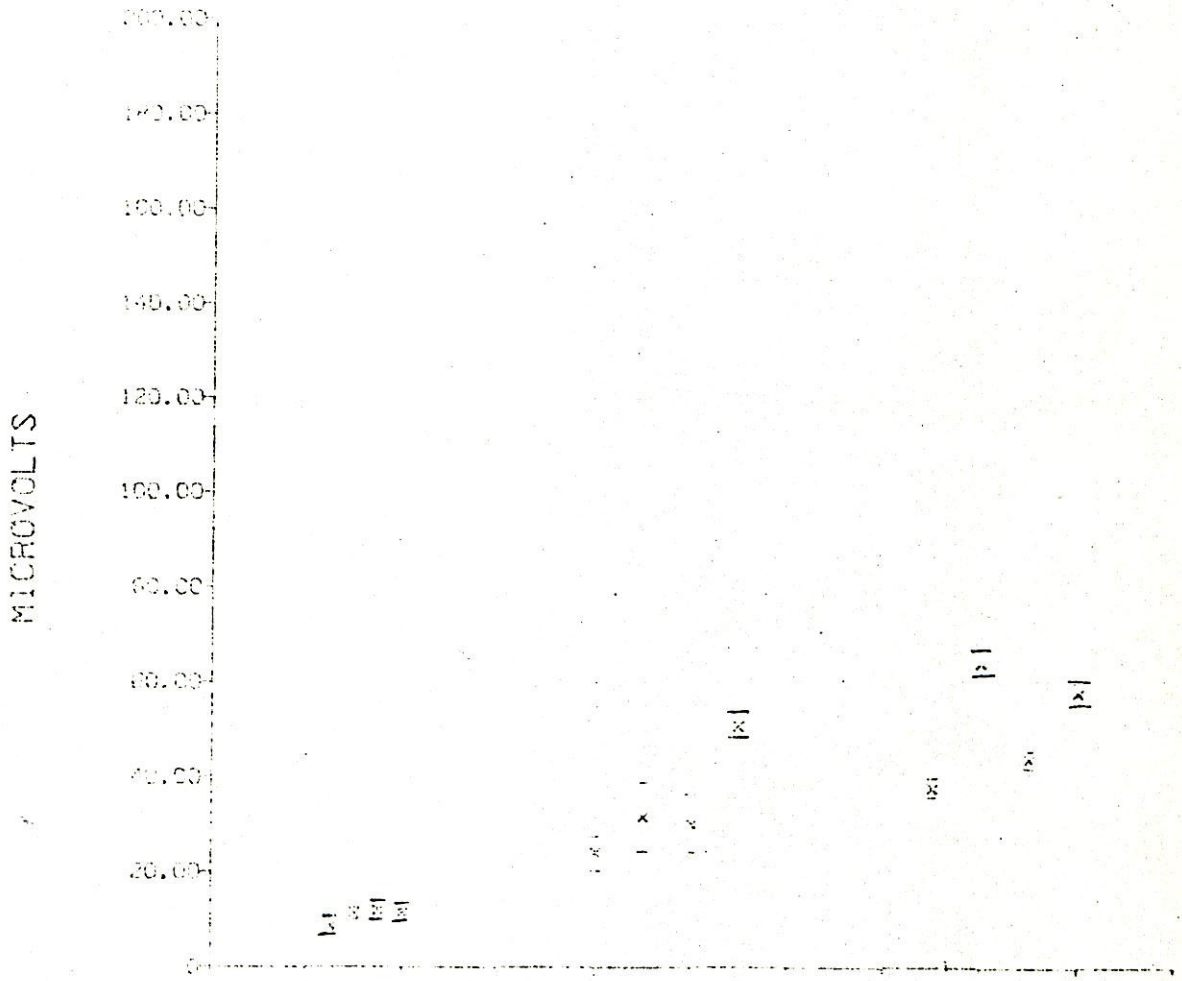
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 7

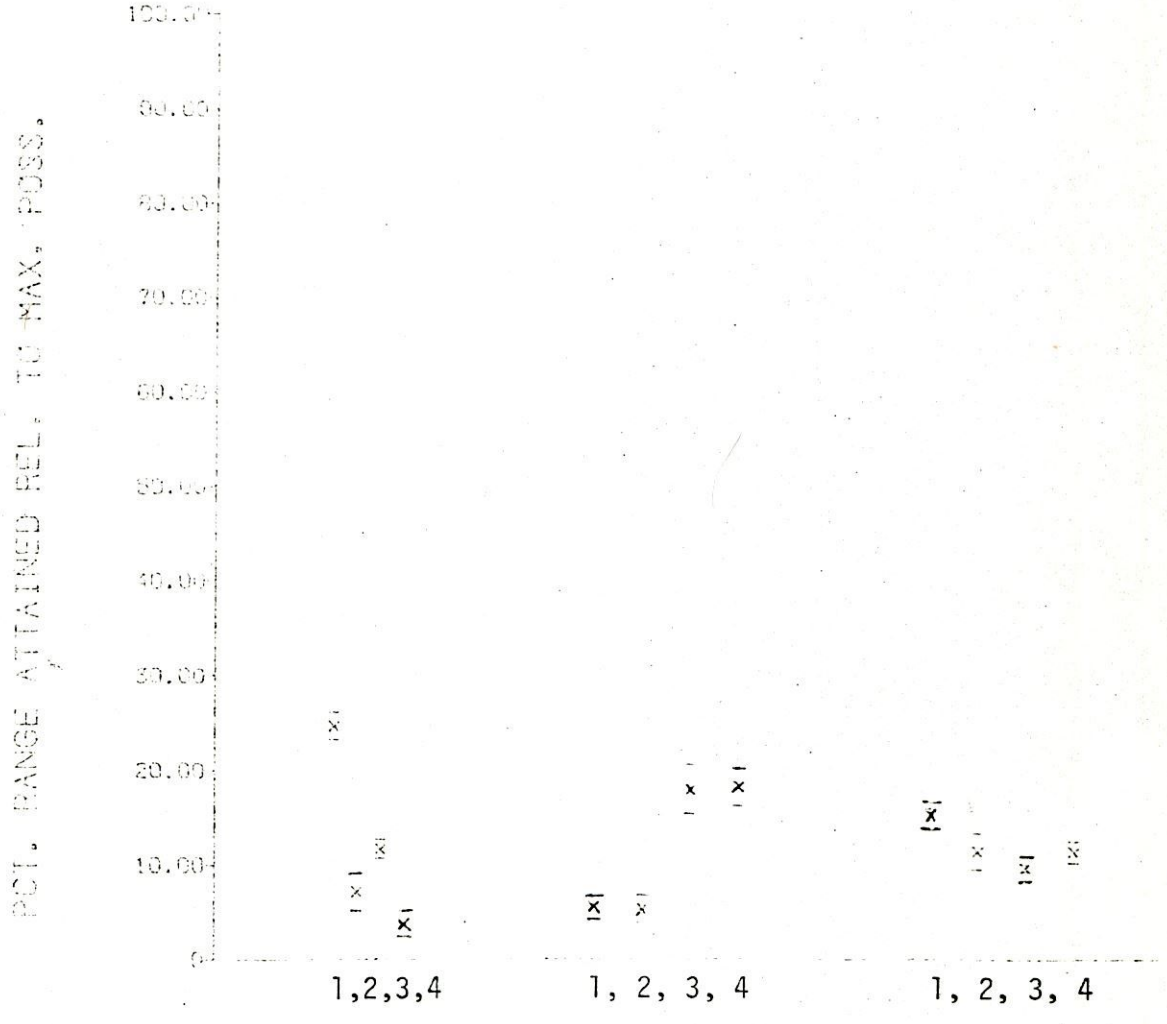


Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 7



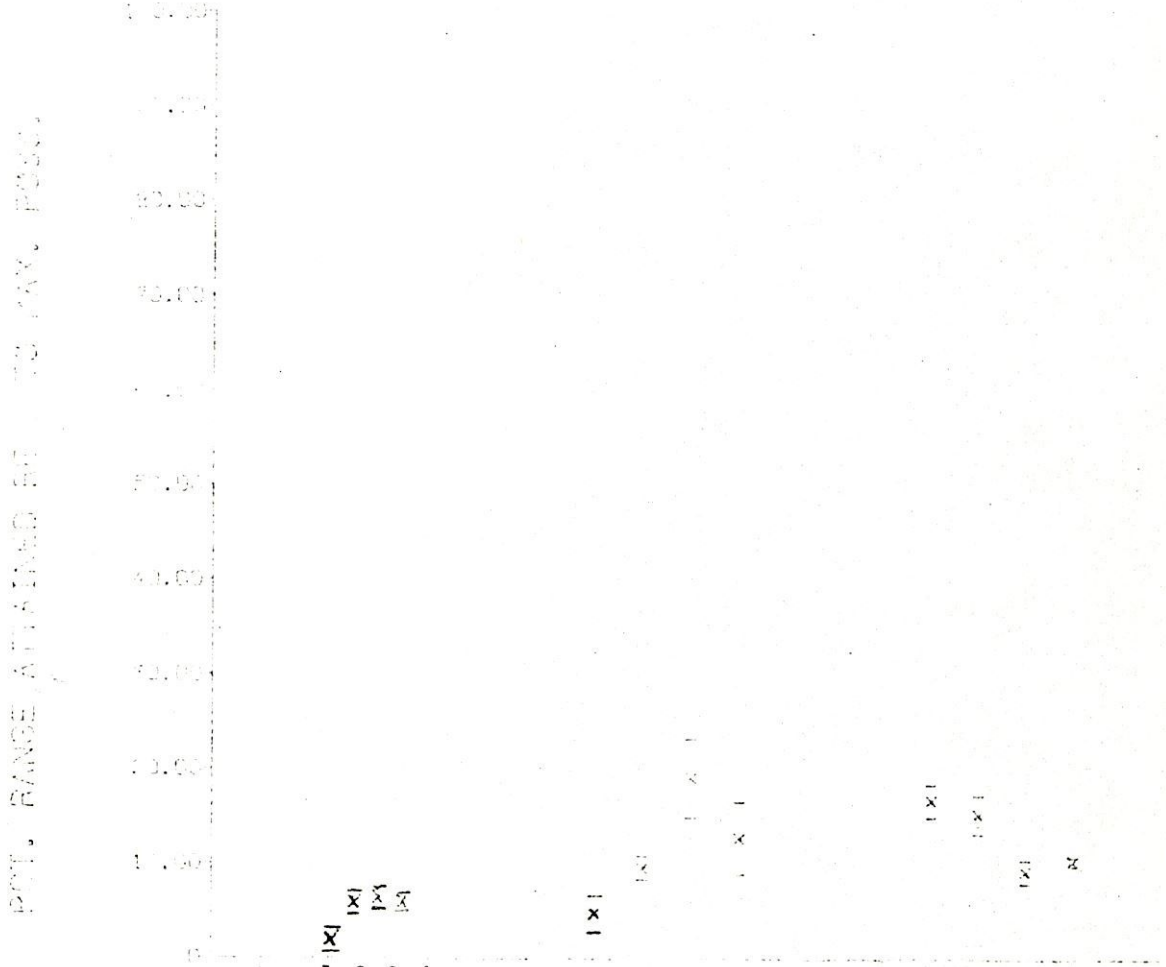
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)



*Graphs related to range of motion are not indicative of learning for Patient 7, as he was apractic, but are presented for thoroughness. (For further details, see Patient 7's case history.)

Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

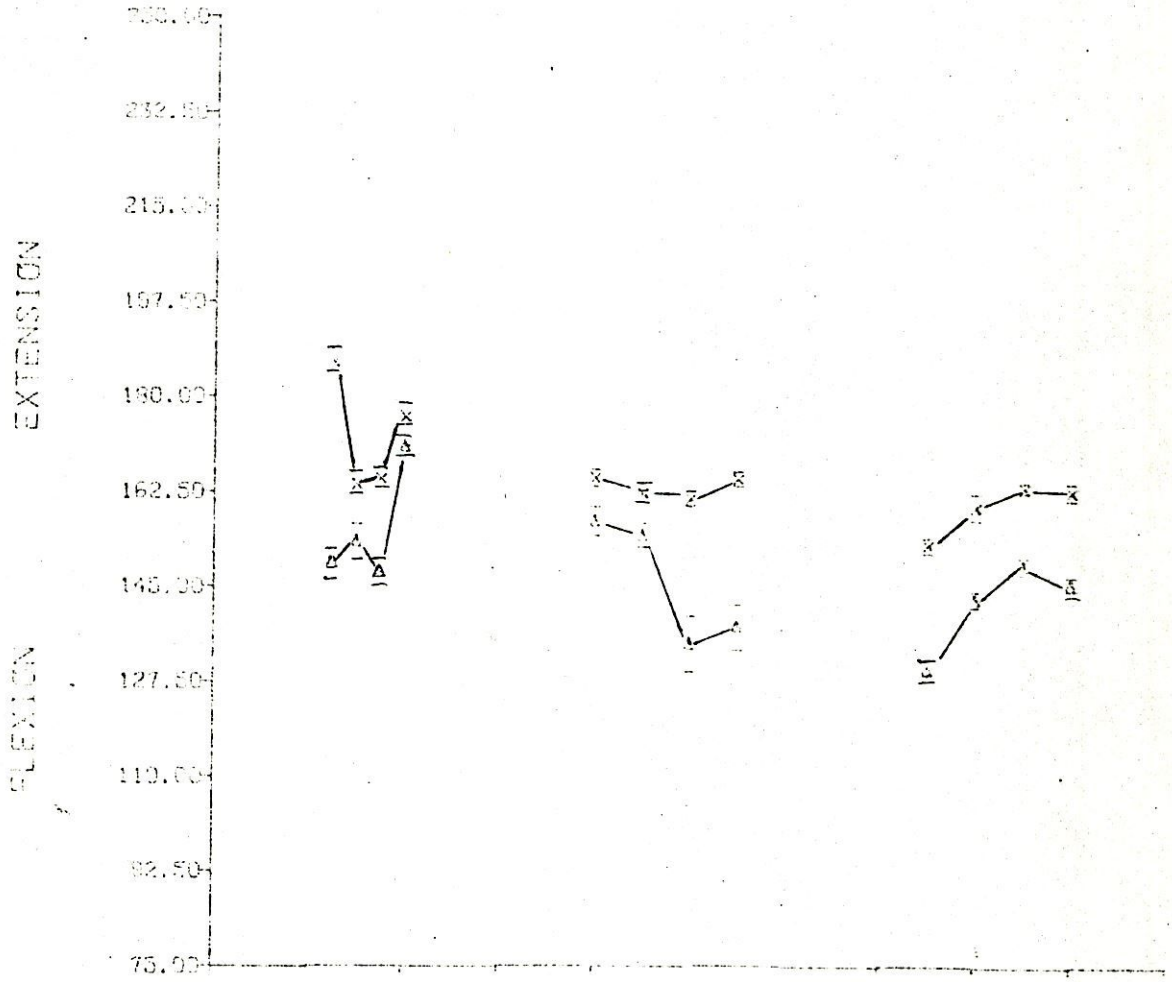
PATIENT 7*



*Graphs related to range of motion are not indicative of learning for Patient 7, as he was apractic, but are presented for thoroughness. (For further details, see Patient 7's case history.)

Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

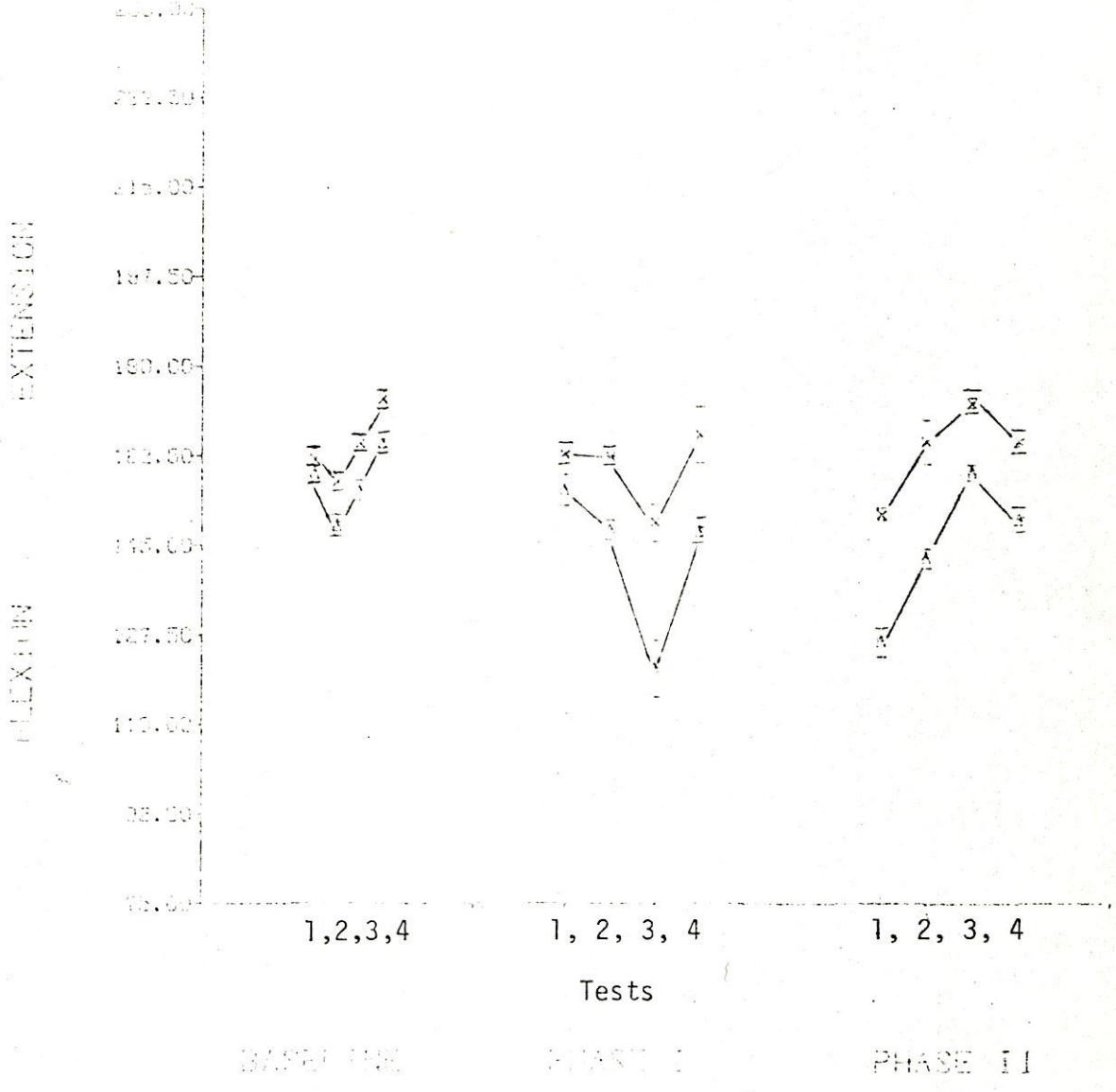
PATIENT 7*



*Angle of conditioning was, for Patient 7 only, flexion instead of extension. Graphs related to angular motion are not indicative of learning for Patient 7 as he was apractic, but are presented for thoroughness. (For further details see Patient 7's case history.)

Extension ($\overline{X}$) and Flexion ($\underline{\Delta}$) in Degrees (SE indicated)

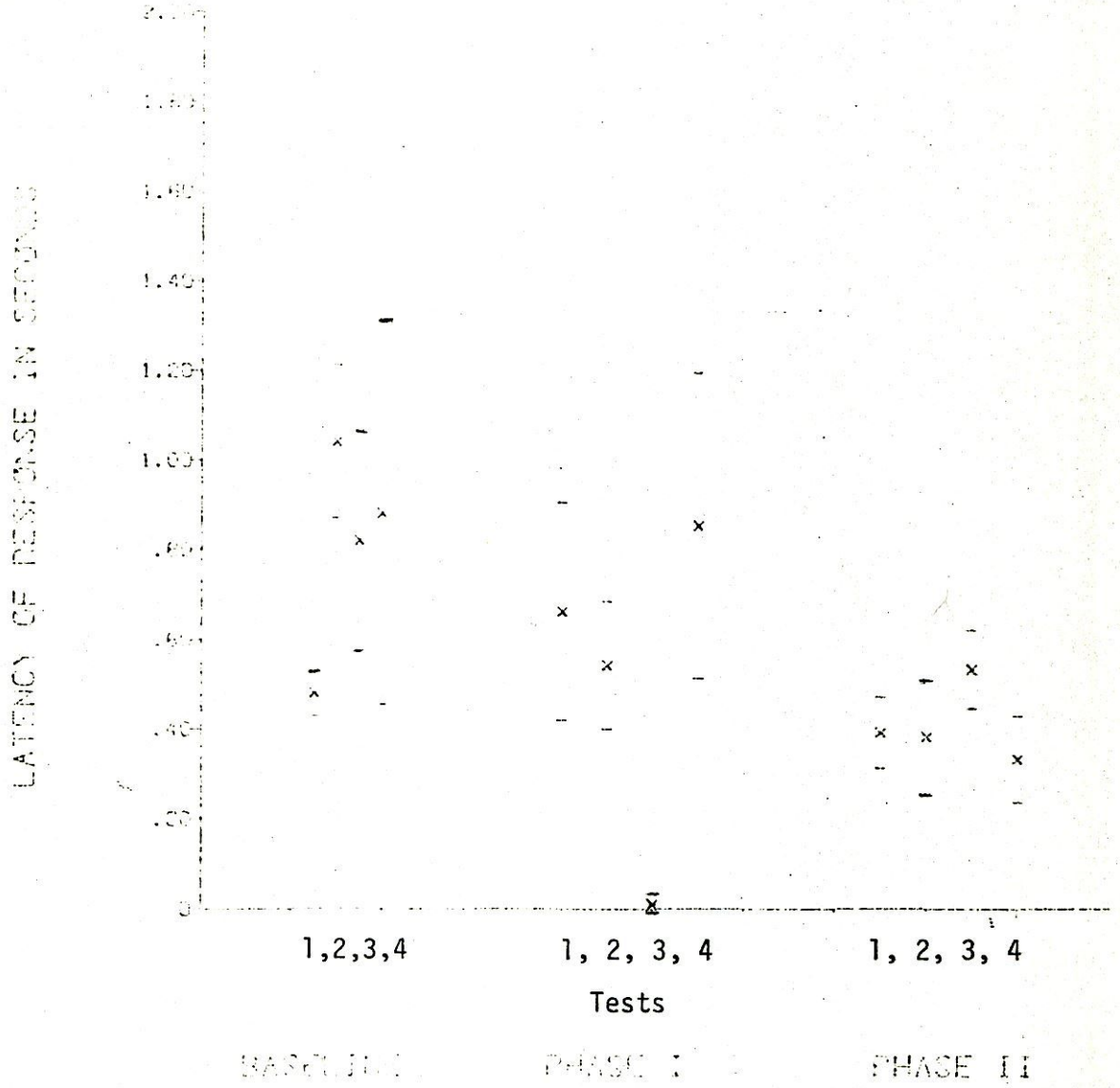
PATIENT 7*



*Angle of conditioning was, for Patient 7 only, flexion instead of extension. Graphs related to angular motion are not indicative of learning for Patient 7 as he was apractic, but are presented for thoroughness. (For further details see Patient 7's case history.)

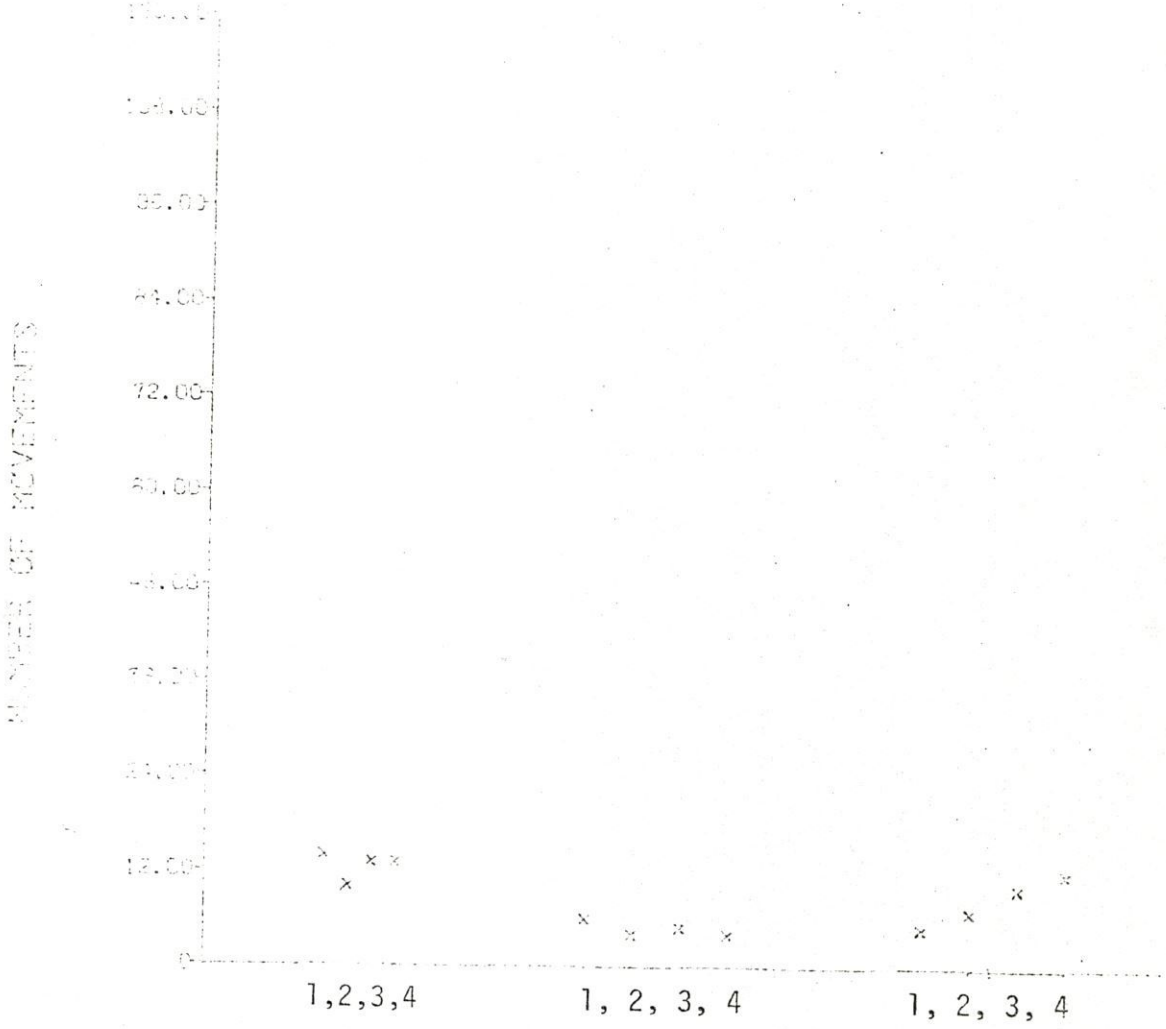
Latency (SE indicated)

PATIENT 7 [illegible] (UPP. LIMB [illegible] .1 [illegible] .1)



Repetition of Movement

PATIENT 7 ( )



Patient 8

MMPI

L	50
F	66
K	35
Hs	44
D	57
Hy	43
Pd	50
Mf	63
Pa	53
Pt	65
Sc	63
Ma	63
Si	66
Es	34

Code

'789 2-'31(63)'4,10,4

Patient 8

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	87
Verbal IQ (VIQ)	87
Performance IQ (PIQ)	88
WAIS Verbal Subtests:	
Comprehension	10
Mathematics	6
Digit Span	8
Vocabulary	7
WAIS Performance Subtests:	
Block Design	10
Picture Arrangement	8
Object Assembly	6
Porteus Maze Test	17
Trail Making Test, Part A	71"-0 errors
Trail Making Test, Part B	244"-5 errors
Bender-Gestalt Recall	3
Wechsler Memory Scale (WMS) Quotient	79
WMS Subtests:	
General Information	5
Orientation	4
Mental Control	0
Memory Passages	7.5
Digits Control	9
Visual Reproduction	4
Associates Learning	9
Aphasia Screening Test (AST)	4-4 verbal errors
LH Tactile Formboard I, Standard RH	28"-10 objects placed
LH Tactile Formboard II, Blindfolded RH	290"-10 objects placed

Patient 8, 50 yr old white female, 1 stroke (1 yr, 6 mos poststroke)

Clinical Diagnosis:

(1) Hypertension

(2) Obesity

(3) Occlusion of left carotid artery, middle cerebral artery

(4) Right hemiparesis mainly involving right upper extremity

Prior Handedness: Right

Prior Therapy: None

Comments: Patient 8 suffered another stroke prior to the last experimental week.

NEUROLOGICAL EXAMINATION

Description of Weakness:

Spastic right brachial monoparesis. Spastic flexion of fingers with springy contractures. Strength: 3/5 biceps, 2/5 triceps, 3/5 extension and flexion of wrist. All DTRs increased on right with increased DTRs of the left upper extremity. Right Babinski.

Description Sensory Impairment:

Pin: Slight decreased sensation of pin over the face, right upper and lower extremity.

Touch: Intact

Vibration: Intact

Position Sense: Severe loss of position sense in right upper extremity.

Two point discr: Intact

Graphesthesia: None

Stereognosis: None

Aphasia:

Mild dysarthria.

Apraxia:

Minimal buccofacial apraxia.

Comments: Increased left upper extremity reflexes suggest possibility of bilateral cerebral dysfunction.

Maximum angle of wrist extension with finger contractures: approx 235^0.

Training Objective: Wrist extension

Phase I: BF

Phase II: PT

Introduction:

Patient 8 had a spastic monoparesis of her right upper extremity. She fractured this extremity at the surgical neck of the right humerus about 12 months after her stroke. The arm rested in a sling while the fracture was healing. After the fracture, maximum elbow extension was approximately 160^0. Patient 8 walked with her elbow flexed at 90^0, somewhat because of the fracture, but largely due to spasticity. She

furthermore reported that holding her arm bent was a protection mechanism against hurting it any further. She had contractures at the shoulder joint and in the long finger flexor muscles. She normally held her fingers in relaxed semi-flexion at all joints, thumb abducted parallel to the plane of the palm. Finger flexion increased when she extended the wrist. She could voluntarily extend the fingers at the MCP joints although the muscles were very tight and spastic. She had voluntary parallel abduction of the thumb and was able to flex the thumb at the IP joint. Active wrist extension in the standard position over the table was (e.g., Part A) approximately 186^0 during baseline. (With associated shoulder movements, the patient achieved better angles for this movement.) Wrist extension was jerky and inevitably accompanied by clonus. There was simultaneous activation of the wrist extensors and flexors during wrist extension, so that baseline range of motion was from 133^0 to 186^0 to equal 53^0. Wrist extension was also accompanied by elbow flexion of about 20^0 from the flexed 90^0 position of the arm resting on the table. Maximum wrist extension possible for Patient 8, due to the wrist contractures, was approximately 235^0.

Patient 8 was an anxious, shy, cooperative, and amiable woman. A widow, she lived with two teen-aged daughters and a 10 year old son. She was capable of most activities other than those which required bimanual fine dexterity. She could hold down a jar to open it and flick light switches with her right hand. She practiced alot at home. Patient 8 was socially anxious and was anxious when having to perform in front of E. In addition she was anxious about further injury to her arm and she anticipated E in her movements. In other words, she was constantly "set" to move. For example, she would anticipate E's command in the test Command situation. She was told she must wait rather

than anticipate and she inhibited this tendency. She also could never resist co-moving her limb with E during PT, or resist moving it to touch. She had a difficult time "letting go" to E's instructions. She could, however, remain passive with E, but she was presumably more capable of relaxing when alone. She was keenly aware of relaxation vs. tension in her paretic upper extremity.

Phase I: BF -

During BF, Patient 8 would start extending the wrist and end with slightly raising the forearm off the table. She was reminded not to let her wrist leave the table and she eliminated raising the forearm after one week of BF training. She was also told to inhibit the accompanying clonus ("don't move to a point where you shake, move smoothly", etc.). The clonus stopped within a couple of sessions but would reappear with fatigue at the very end of a session. Wrist extension became a smooth motion (less jerky) and "natural", more cosmetic in appearance, over time.

The greatest problem to surmount, it would seem, was the simultaneous flexor excitation during wrist extension. This problem was explained to Patient 8 with specific references to the opposing functions of the wrist flexors and extensors. Within the first three BF sessions, she ceased to co-activate these muscles. This was quite dramatic as it was accomplished strictly by verbal instruction. One final problem was that the wrist extensors did not relax completely when active wrist extension was terminated and flexion supervened. When this happened, range of motion was decreased (e.g., Part A) from a maximum of 74^0 to 53^0. Bidirectional control on the EMG TV and acoustics was correlated with inhibition of the extensors for greater range of motion along with

smoother, correct, better appearing, coordinated wrist movement. The latter suggested greater ability to relax the flexors as well as the extensor muscles.

The amount of averaged EMG recruitment almost doubled during BF ([e.g., Part A] from approximately 49 μv to approximately 87 μv). Patient 8 was completely engrossed in making the TV peak when left on her own and E's visits soon became superfluous interruptions. They were thus less frequent as Patient 8 had good powers of attention, concentration, and comprehension of required specifics.

Phase II: PT -

NOTE: The third testing session for PT was one session early as Patient 8 appeared ill with what seemed to have been a protracted upper respiratory infection. Treatment was terminated thereafter because of the onset of a second cerebrovascular accident.

During PT, Patient 8 was urged to relax her arm and "let the elbow go" (extend it). She practiced general arm relaxation and was told to incorporate an extended, relaxed arm into her daily routine. Training consisted of manual resistance to the wrist extensors, and, secondarily, wrist flexors. Patient 8 practiced the resistance at home. Some attempts were also made at active shoulder flexion with instructions to consciously relax the muscles resisting this motion. Spasticity in the latter prevented complete relaxed shoulder flexion to 10^0. Patient 8 flexed the shoulder to approximately 30^0 but remained tight. She once released tension during the movement but neither time nor attention to this movement was sufficient to see gains. At the third week of PT, the maximum angle of wrist extension was evaluated as "holding steady".

The patient, however, appeared to be reverting to more jerky movements and a greater degree of clonus during movement. From the day the patient learned to inhibit the wrist flexors (week 1, BF) wrist flexors were never seen or felt to tighten during wrist extension. It did, however, seem that the wrist extensors were not relaxing as well during PT as during BF.

Comments:

When asked, the patient reported liking BF better because "I can see what I am trying to do". Probably as a result of general arm relaxation, it was noticed during PT that the patient had more finger function in both finger extensors and flexors although the digits were still flexed at the PIP and DIP joints. Near the end of the experiment, the patient suffered a second stroke (see Note, p. 319).

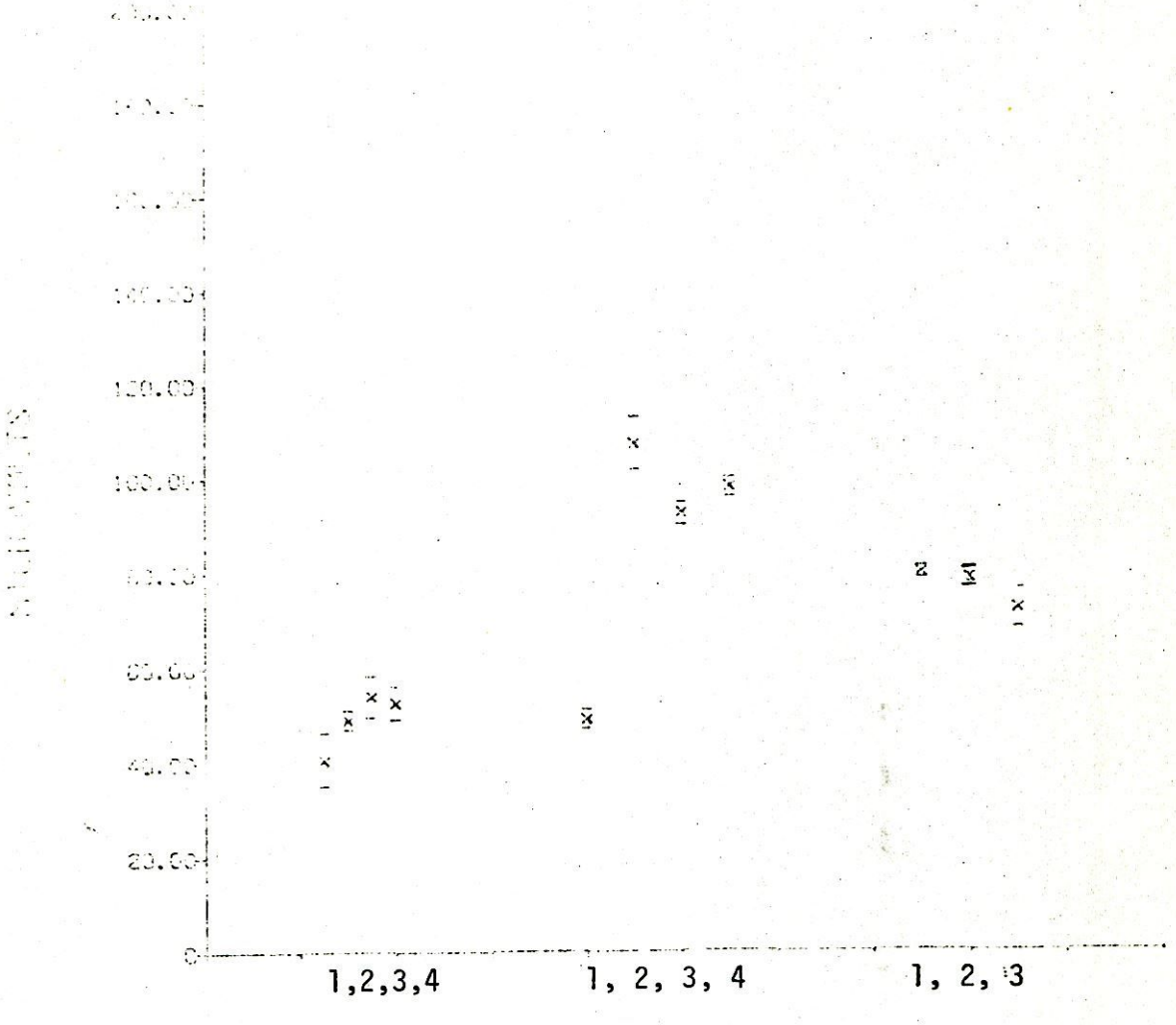
Although Patient 8 achieved a significant increase in averaged EMG activity in Phase I, BF training, that trained level of motor recruitment dropped overall in Phase II, PT training. Such an effect could be due to a certain amount of extinction[41] when the response was not sustained by EMG feedback or it could be due to relaxation training in PT, etc. Considering that Patient 8 had poor position sense, a response of high averaged EMG recruitment, if not well ingrained, might very well rapidly fall away, particularly if it is not related to limb function. Again, too, relaxation training (i.e., PT) does not necessarily incorporate a contraction which consists of "maximal" averaged EMG activity.

---

[41]Extinction here implying that the response was learned, i.e., "stored" during BF.

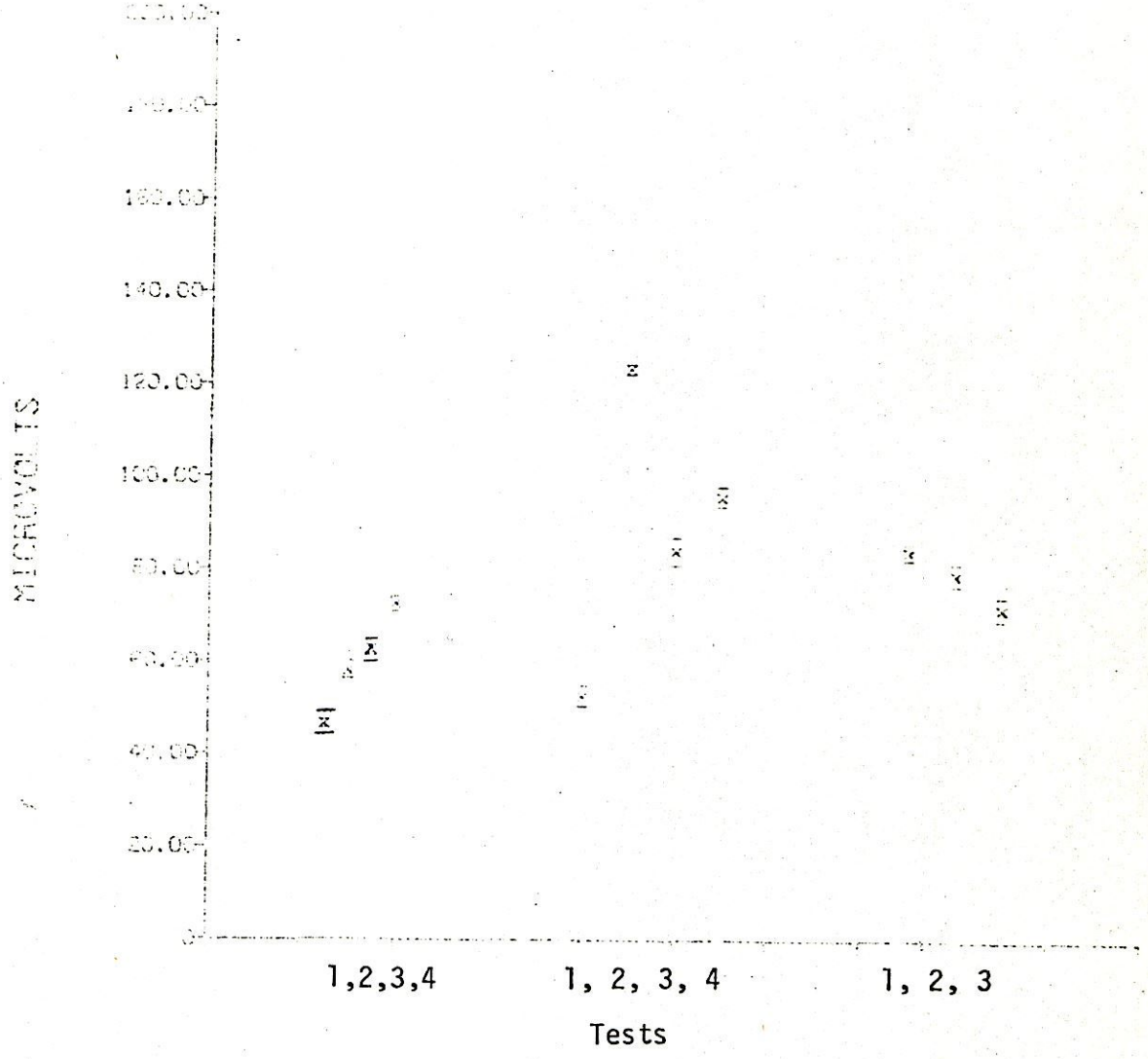
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 8



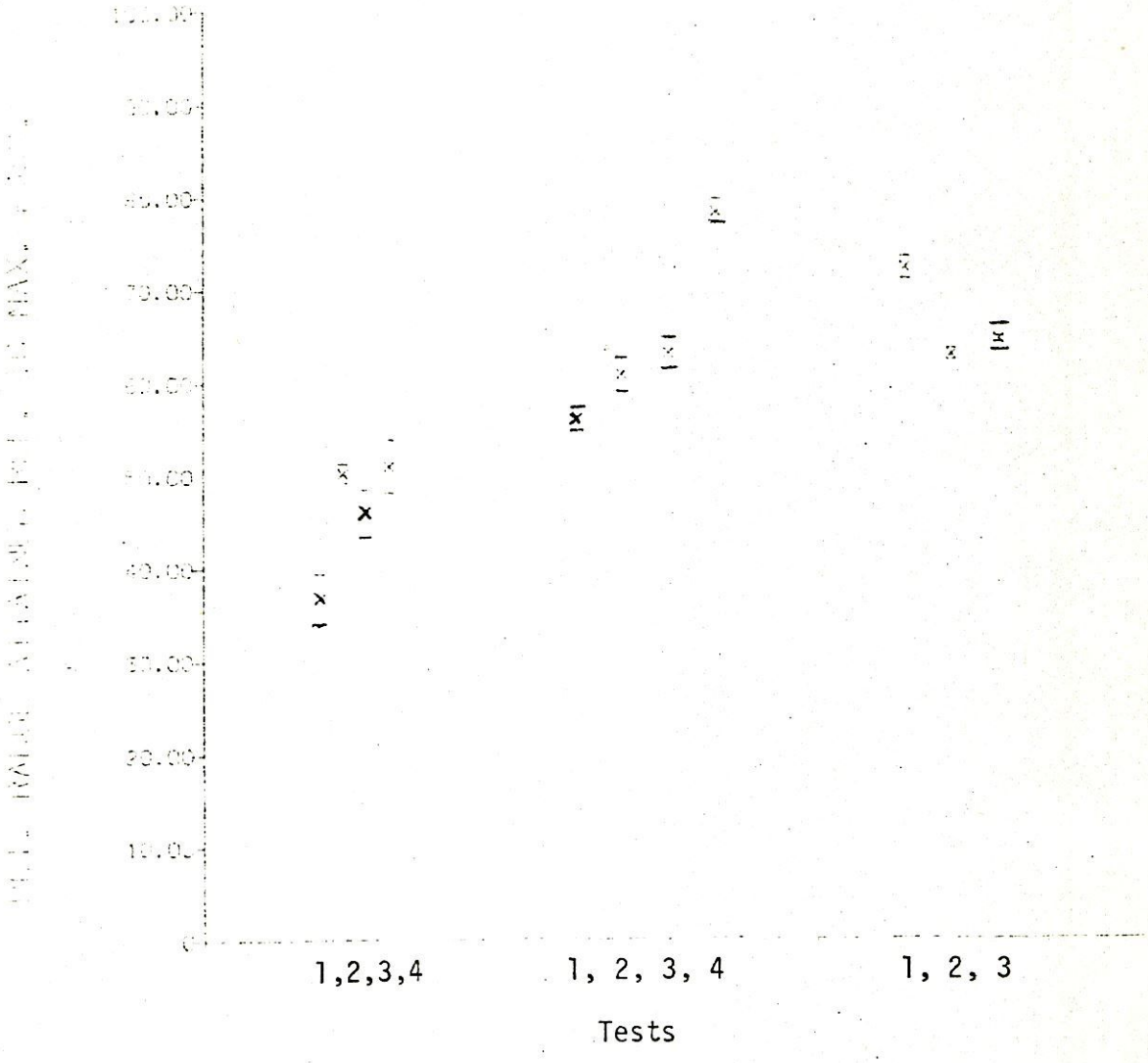
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 8 PART 5 (, LINES 2302 , 2395)



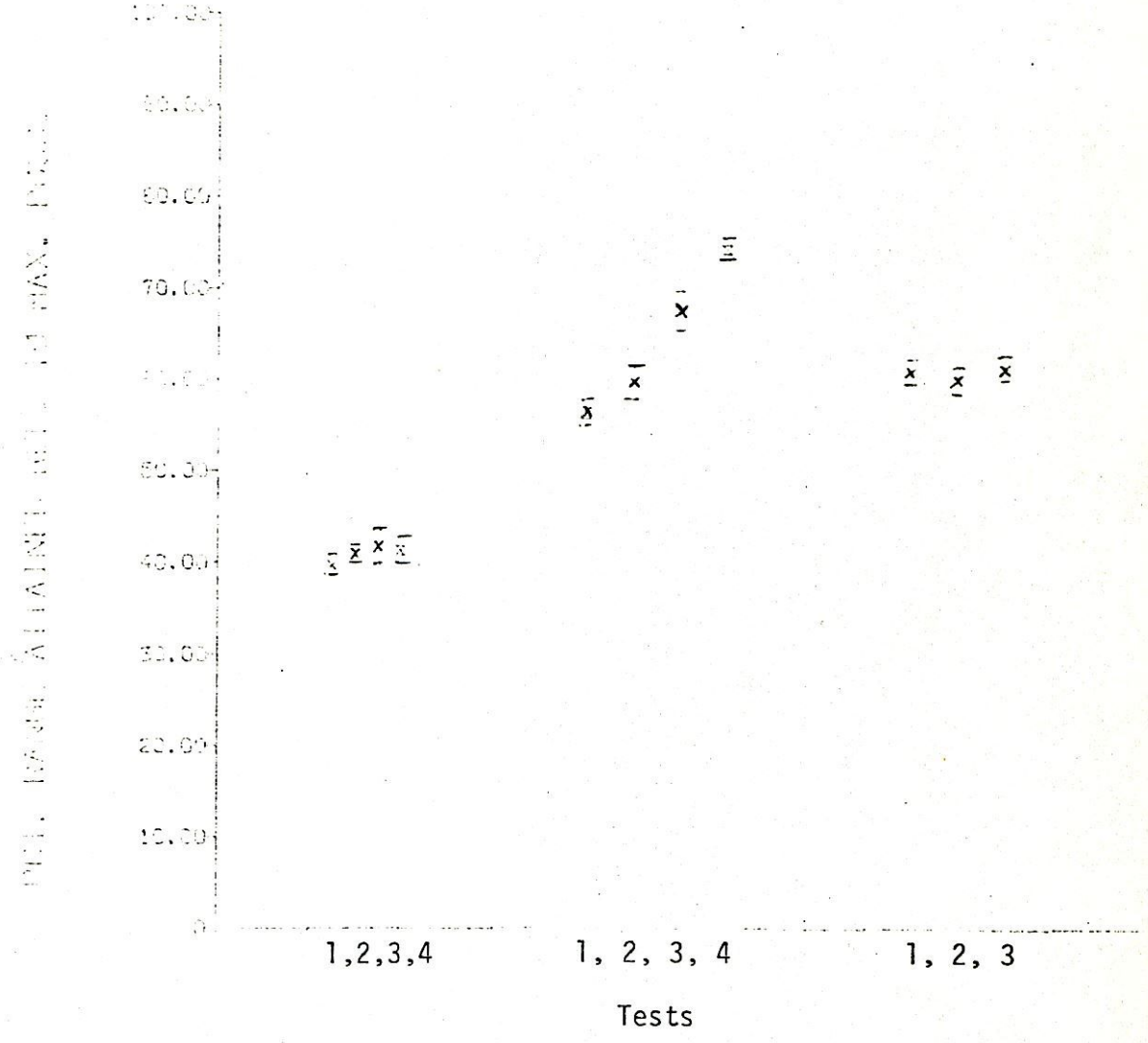
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 8



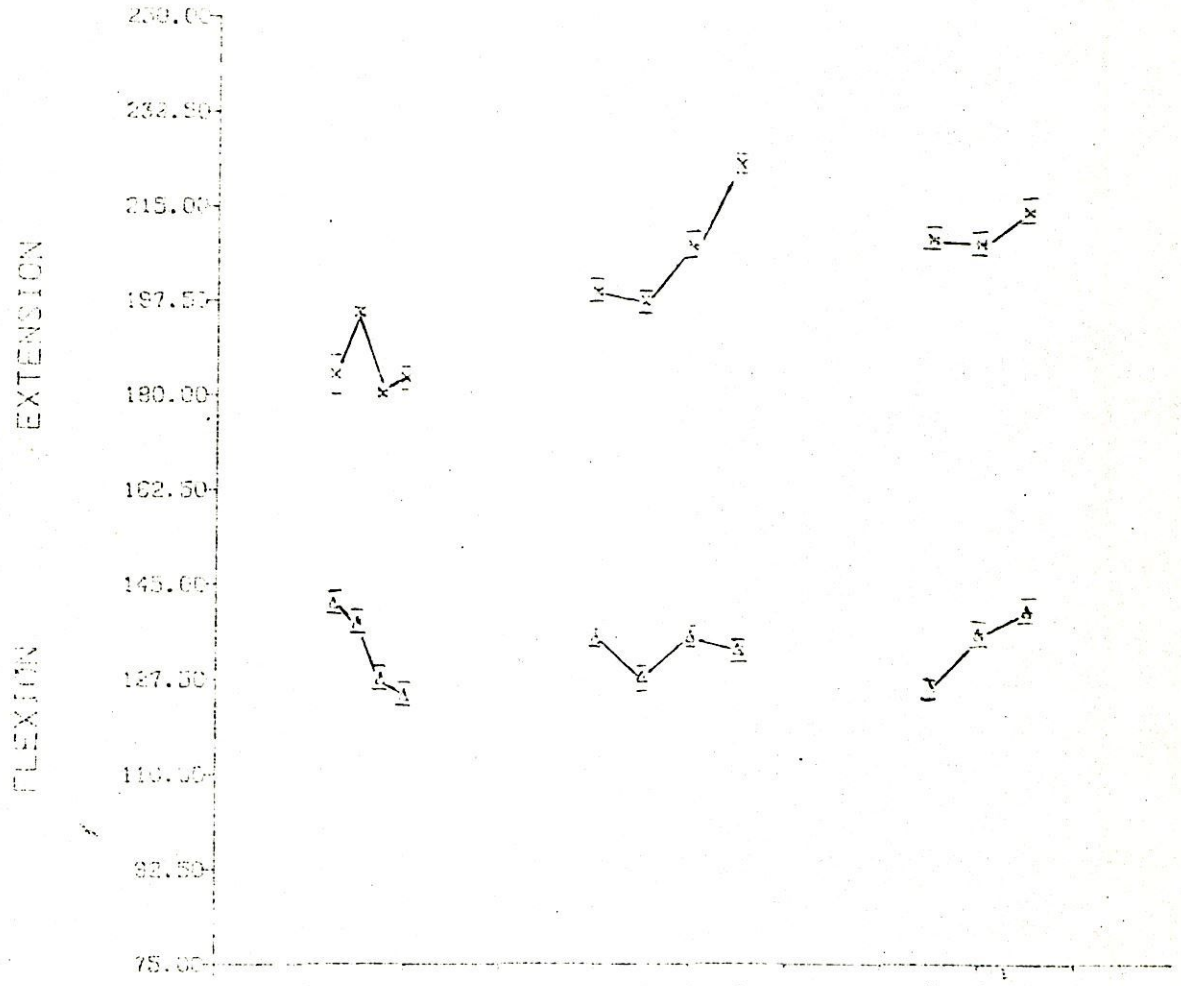
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 8



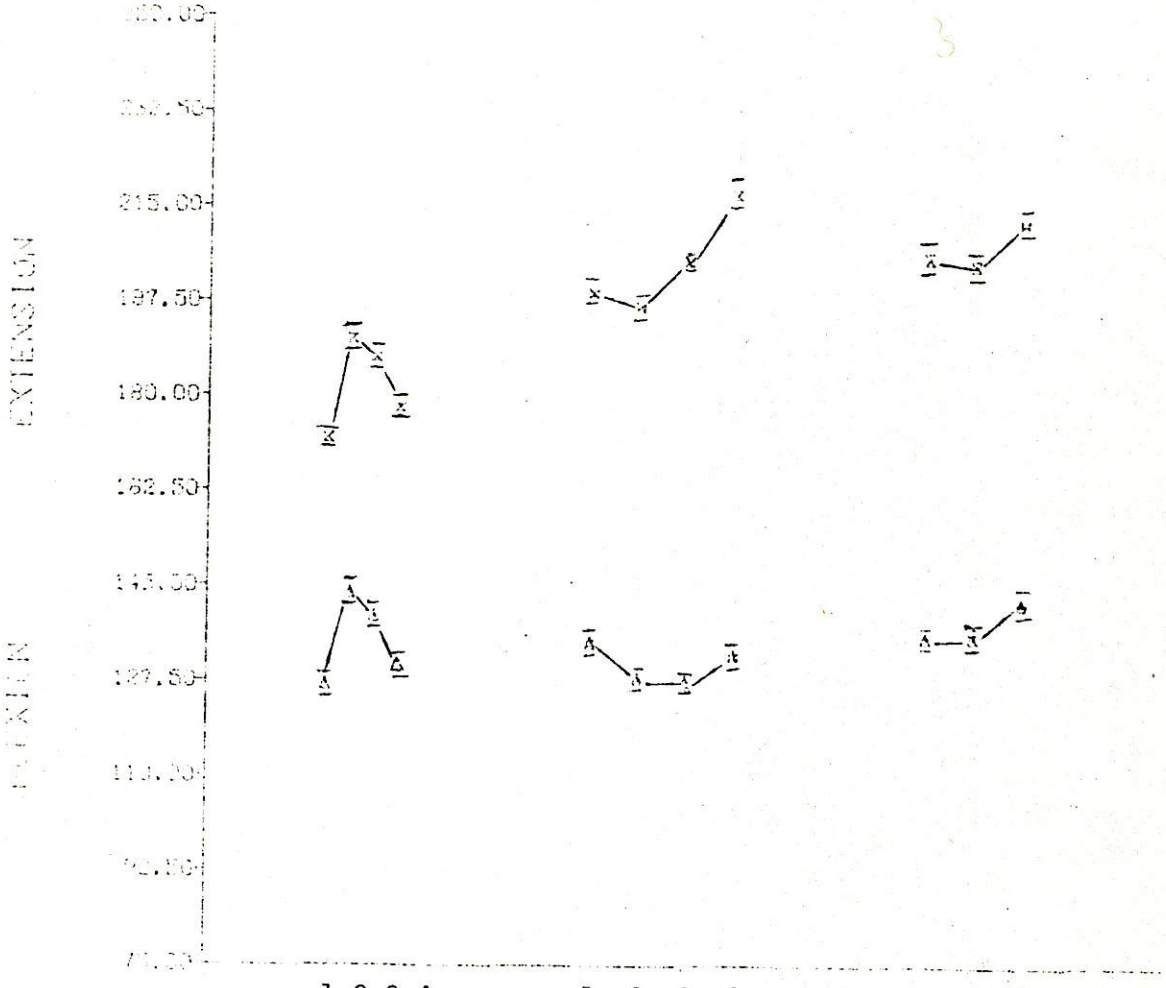
Extension ($\bar{X}$) and Flexion ($\bar{\Delta}$) in Degrees (SE indicated)

PATIENT 8 PART A



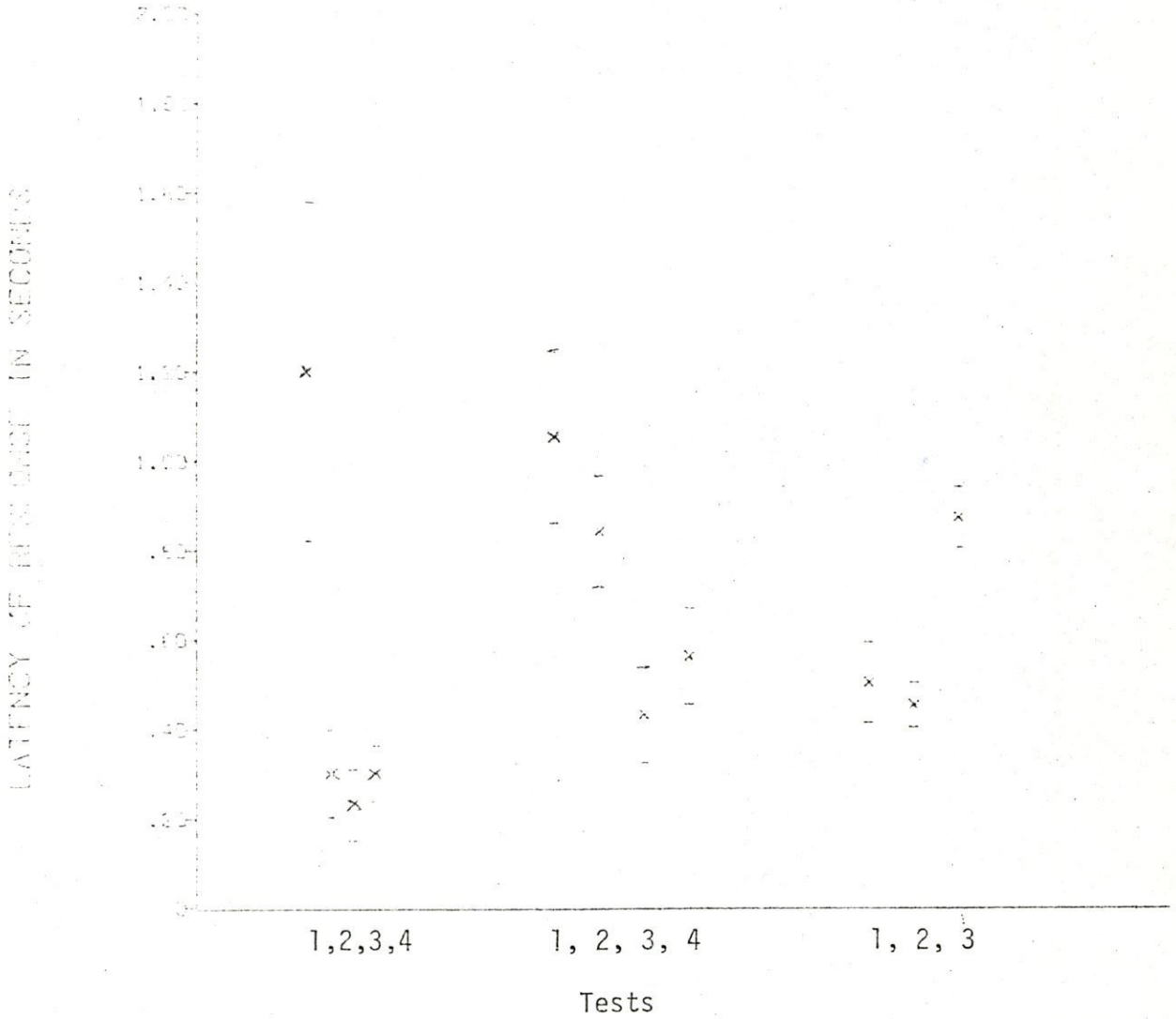
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 8



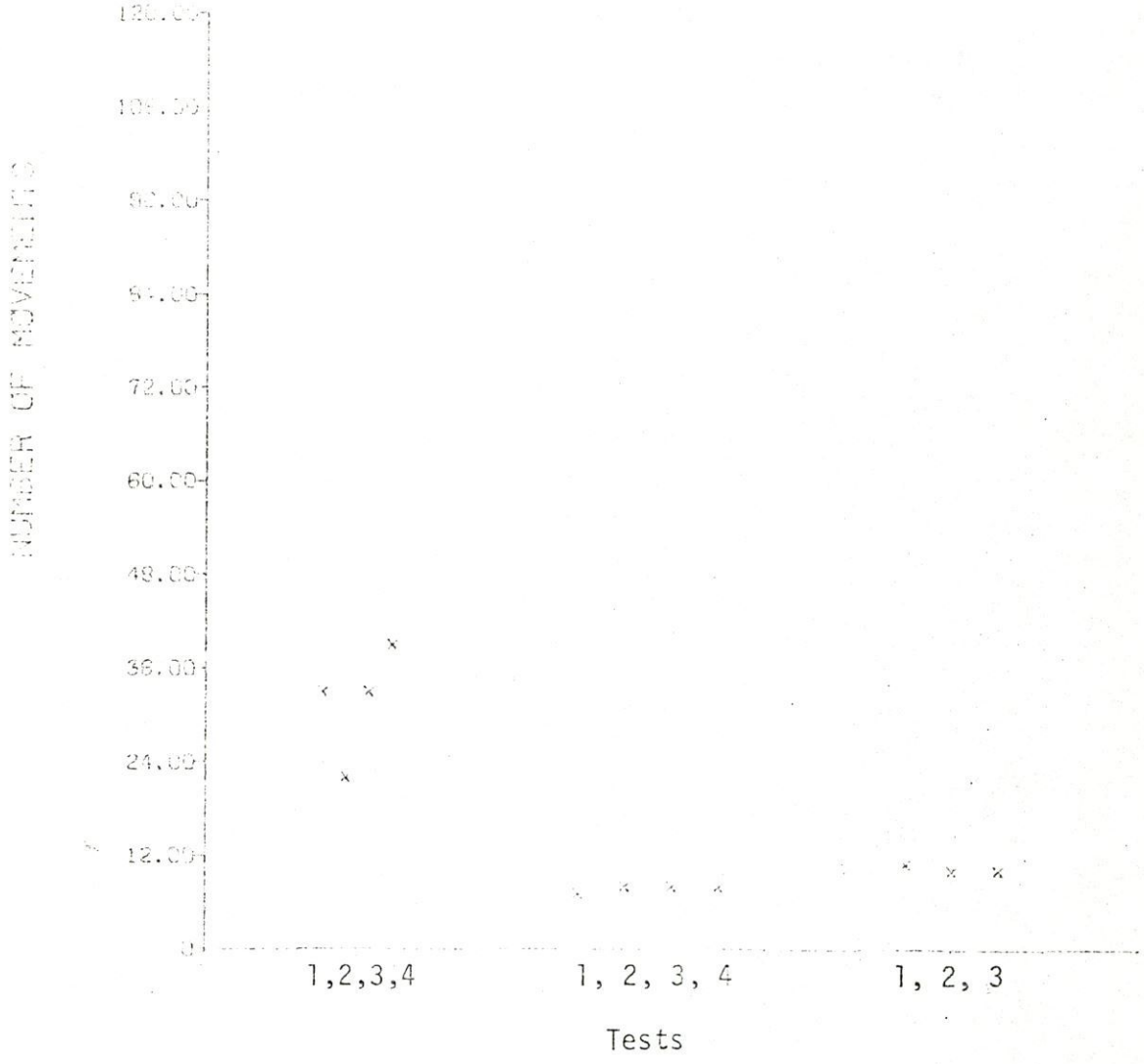
Latency (SE indicated)

PATIENT 8



Repetition of Movement

PATIENT 8 (



BASELINE PHASE II

Patient 9

MMPI

L	56
F	58
K	42
Hs	46
D	63
Hy	49
Pd	60
Mf	61
Pa	59
Pt	56
Sc	57
Ma	73
Si	62
Es	43

Code

9'246 87-'(61)^{5}6,6,8

Patient 9

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	78[42]
Verbal IQ (VIQ)	68[42]
Performance IQ (PIQ)	94
Shipley Hartford Test	114[42]
WAIS Verbal Subtests:	
Comprehension	NA
Mathematics	4
Digit Span	NA
Vocabulary	NA
WAIS Performance Subtests:	
Block Design	7
Picture Arrangement	12
Object Assembly	7
Porteus Maze Test	9
Trail Making Test, Part A	170"-0 errors
Trail Making Test, Part B	230"-5 errors
Bender-Gestalt Recall	1
Wechsler Memory Scale (WMS) Quotient	NA[43]
WMS Subtests:	
General Information	NA
Orientation	NA
Mental Control	NA
Memory Passages	NA
Digits Control	NA
Visual Reproduction	NA
Associates Learning	NA
Aphasia Screening Test (AST)	25-25 errors (unable to respond verbally)
LH Tactile Formboard I, Standard RH	31"-10 objects placed
LH Tactile Formboard II, Blindfolded RH	232"-10 objects placed

[42]This patient had a complete expressive aphasia. The Mathematics subtest was the only Verbal subtest which could be used. Hence, the VIQ score, prorated on the Math test only, and the Full Scale IQ, prorated on the Verbal and Performance subtests, are not truly representative scores. The Shipley Hartford Test (Institute of Living, 1939) was administered as a substitute for the WAIS VIQ. The latter score was 114.

[43]NA = not applicable (due to complete expressive aphasia).

Patient 9, 75 yr old white female; 1 stroke (2 yrs, 6 mos poststroke)

Clinical Diagnosis: (1) Chronic hypertension
(2) Thrombosis, middle cerebral artery
(3) Right brachial monoparesis at time of experimental neurological examination

Prior Handedness: Right

Prior Therapy: 2 mos total; arm and leg

Comments: None

NEUROLOGICAL EXAMINATION

Description of Weakness:

Spastic right brachial monoparesis with general flexor spastic rigidity of a springy nature at the wrist and fingers and some at the elbow joint. Reflexes and tone are increased on the entire right side. There is a Babinski sign on the right. Strength is 2/5 for finger extension, 1/5 for elbow extension, 4/5 elbow flexion, 3/5 arm abduction.

Description Sensory Impairment:

Pin: Intact

Touch: Intact

Vibration: Diminished vibration below ankles bilaterally.

Position Sense: Mild diminution of position sense at the right thumb and toe.

Two point discr: Some impairment in the right upper extremity.

Graphesthesia: Intact

Stereognosis: Impaired in the right upper extremity.

Aphasia:

Limited to repetitive utterances of na-na-na with good comprehension.

Apraxia:

Severe bucco-facial apraxia.

Comments: None

Maximum angle of wrist extension with wrist and finger flexor contractures: approx 215^0

Training Objective: Wrist extension

Phase I: BF

Phase II: PT

Introduction:

Patient 9 had a spastic monoparesis of the right upper extremity. Spontaneous movement consisted of rotatory activity at the shoulders with maintenance of an adducted position and minimal flexion, extension or abduction, with flexion or extension of the elbow and wrist. In other words, most movements were movements at the elbow and wrist.

In command to general movement, Patient 9's movement was jerky due to spasticity and consisted of a pattern of shoulder abduction, elevation, and partial external rotation, elbow flextion, and wrist extension (approximately to 190^0) with clenched fingers. The angle of wrist extension was greater in gross movement than in the standard position (wrist over the table). She had contractures of the wrist and finger flexor muscles. She was not able to flex the shoulder without abduction, elevation, and external rotation at the shoulder joints. She could flex the elbow without abduction and habitually carried the elbow at 90^0 flexion. She was able to extend the elbow to approximately 135^0. At baseline, she could extend the wrist to approximately 170^0 (Part A)and 154^0 (Part B) in the standard position, and flex the wrist to approximately 129^0 and 138^0. The discrepancy in range in Part A (Command) and Part B (Rate) was due to the fact that the patient moved rapidly with lesser successive maximal angles of extension and relaxation during Rate. Increased spasticity during the uninterrupted Rate situation contributed to this effect and, in addition to being a contributory effect, was itself caused by the patient's trying to move rapidly. Contractures at the wrist and fingers permitted a maximum extension of approximately 215^0. Wrist extension consisted of simultaneous activation of wrist extensors and flexors as well as tenodesis and active contraction of the finger flexors three-quarters of the way through movement. Finger flexion was partly by tenodesis but especially by a forceful clenching (clawing) of the forearm finger flexors. There was a good deal of spasticity and increased tone in the whole upper extremity. The patient wore a splint for her fingers and wrist. Initially she moved the whole arm in the stereotyped pattern described above when attempting to extend the wrist. Through-

out training of the right upper extremity she tended to move her whole body--for example, heaving the chest and extending the thoracic spine with wrist extension or, for example, leaning towards the paretic side with passive ROM for shoulder flexion. The patient tended to undershoot in a finger tapping psychomotor test and was observed to have a difficult time approaching at target objects she wished to contact with her paretic hand.[44]

Patient 9 was highly motivated and practiced a good deal at home. She lived in a nursing home and could do everything for herself except manuevers which required bimanual dexterity. Although she had a complete expressive aphasia, she was very astute at getting her wishes made known via gestures and voice intonations. In fact, there was very little "communication gap". Patient 9 was extremely eager and quick to respond ("hyper") in general, slow to relax, and highly anticipatory. She was also stimulus bound to a degree. She had trouble inhibiting certain behavioral tendencies as well as inappropriate motor excitation patterns which needed sustained attention. For example, although told to listen and pay attention as E explained or demonstrated a movement to her (for example, during PT) she would start the movements immediately with the first stimulus of movement from E. The patient also found it very hard to stop either simultaneous wrist flexor

[44] Part A (Command) consisted of fine commands and response. However, averaged EMG response duration (i.e., time from initial ascent of averaged EMG activity for wrist extensor contraction to terminal descent of averaged EMG activity for wrist extensor relaxation) became quite long with training onset for Patient 9--e.g., response duration was typically 30 seconds in Part A initially during training compared with, e.g., 2 seconds during baseline. Although several patients exhibited an increase in response duration with training, Patient 9's increase was long relative to the performance of other patients (save Patient 10). Thus, to keep within the time and cost constraints of experimental testing, Patient 9 was given 5 commands in Part A (Command) during baseline, but 3-5 commands in Part A of Phase I and II of training. The great increase in response duration after training onset for Patient 9 is also operative in Part B (Rate) where her uninterrupted response rate decreased to 2-3 responses per the one-minute period vs. an average of 40 responses during Rate at baseline.

excitation or finger flexion during wrist extension. With repeated instructions, reminders, or stress on internal concentration, she was able to eliminate these behaviors and motor patterns. She was also extremely satisfied with any movement regardless of its appropriateness and wanted finger return more than any other movement. Thus, part of the initial problem was having the patient accustom herself to the importance of pure and isolated wrist extension instead of finger movement. Patient 9 was one of the more extreme cases of a patient who needed a lot of monitoring of responses by E to ascertain that she was performing and understanding what E had instructed. Patient 9 was moderately aware of the difference between relaxation and tension in both of her upper extremities.

Phase I: BF -

During this period, Patient 9 got very involved in the TV display and would work for the entire training period without fatigue. She needed frequent interventions from E to guide her verbally. E pointed out that along with wrist extension she was 1) raising the forearm; 2) simultaneously contracting the wrist flexors; and 3) clenching her fingers. The first was eliminated rapidly. The other two undesired features took practically all of the BF sessions to eliminate. At first E unobtrusively would observe the patient and find her persisting in activities she had just been asked to cease doing. Then as sessions progressed, the patient could individually inhibit one or the other of the remaining undesired movement components. Finally, the patient could extend the wrist without either wrist flexor excitation or finger flexion. Furthermore, movement was converted to smooth rather than

jerky motion. Averaged EMG at this more refined point was slightly diminished due to the patient's less forceful but more appropriate, harmonious movement. Grip strength also started to improve. With Phase I training, the discrepancy for range in the Command vs. the Rate situation disappeared, but rate of movement for the latter exhibited a dramatic decrease as Patient 9 became more attuned to internally monitoring her response.[45]

Phase II: PT -

As PT began, the patient was able to keep the fingers voluntarily semi-extended at the MCP joints in order not to flex them during wrist extension. Towards the end of PT the patient began to re-excite both wrist extensors and flexors. Intensive wrist extension training continued concentrating on overall arm relaxation together with wrist flexor inhibition during wrist extension and manual resistance applied to the wrist extensor muscles. Here E had to be careful not to excite spasticity or a return to the former pattern. Besides wrist extension, the patient practiced some shoulder flexion and general relaxation of the entire upper extremity,which, as a by-product, helped her to extend the elbow to 180^0. The patient held her arm more relaxed, elbow less flexed, and her grip strength kept improving without ever having had any specific attention paid to it. Her movements were altogether more "natural".

---

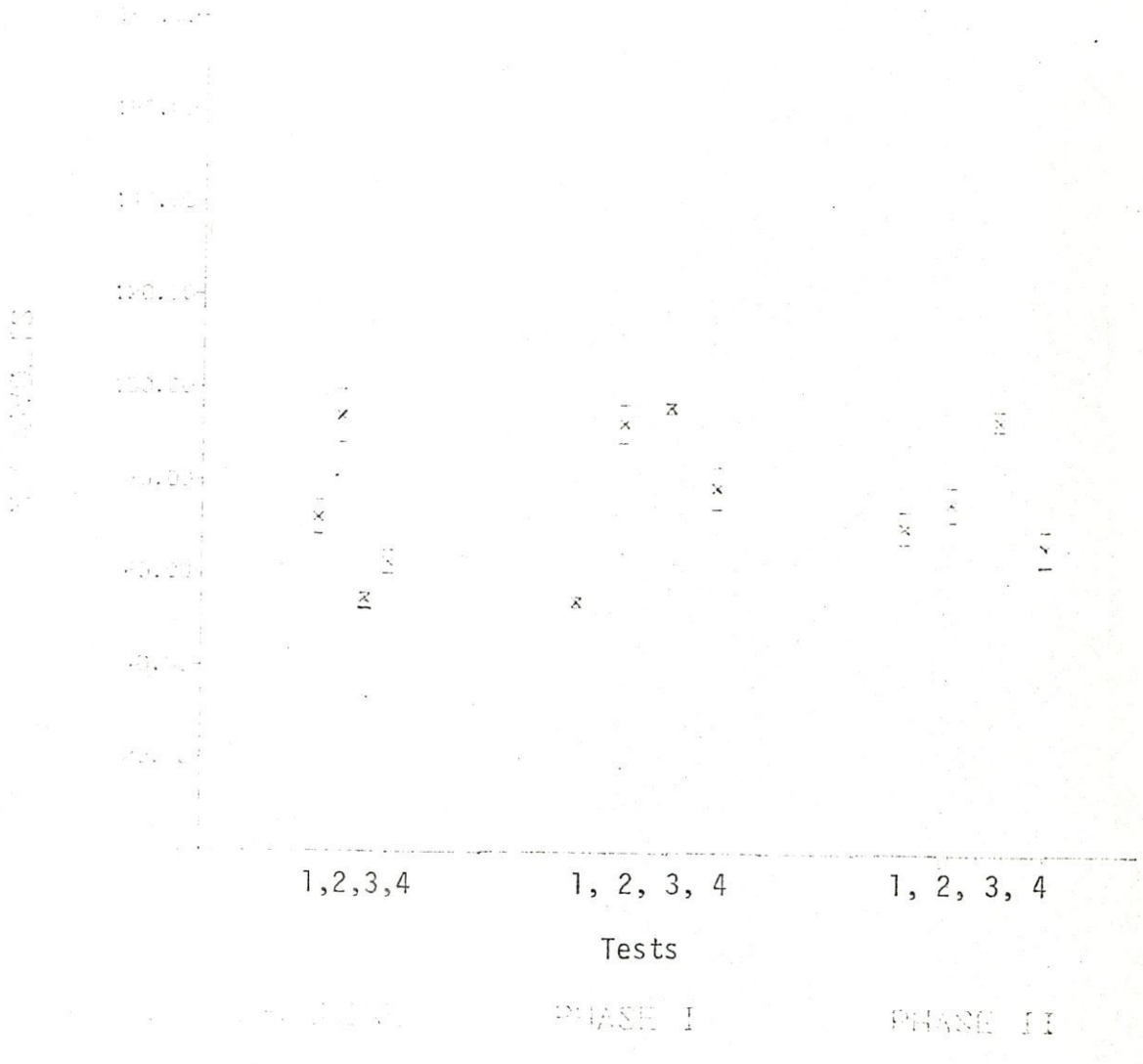
[45] Rate of movement during the Rate situation decreased for all patients in both Phase I and II over baseline, as training proceeded to incorporate attention to movement specifics.

Comments:

Due to overall training, Patient 9 could grip a pulley at home with her right hand whereas before she used her left hand to position her hand on the pulley. She could also use her right hand to remove a light jacket and could contact and pick up small objects without undershooting. When asked, the patient reported having liked BF over PT.

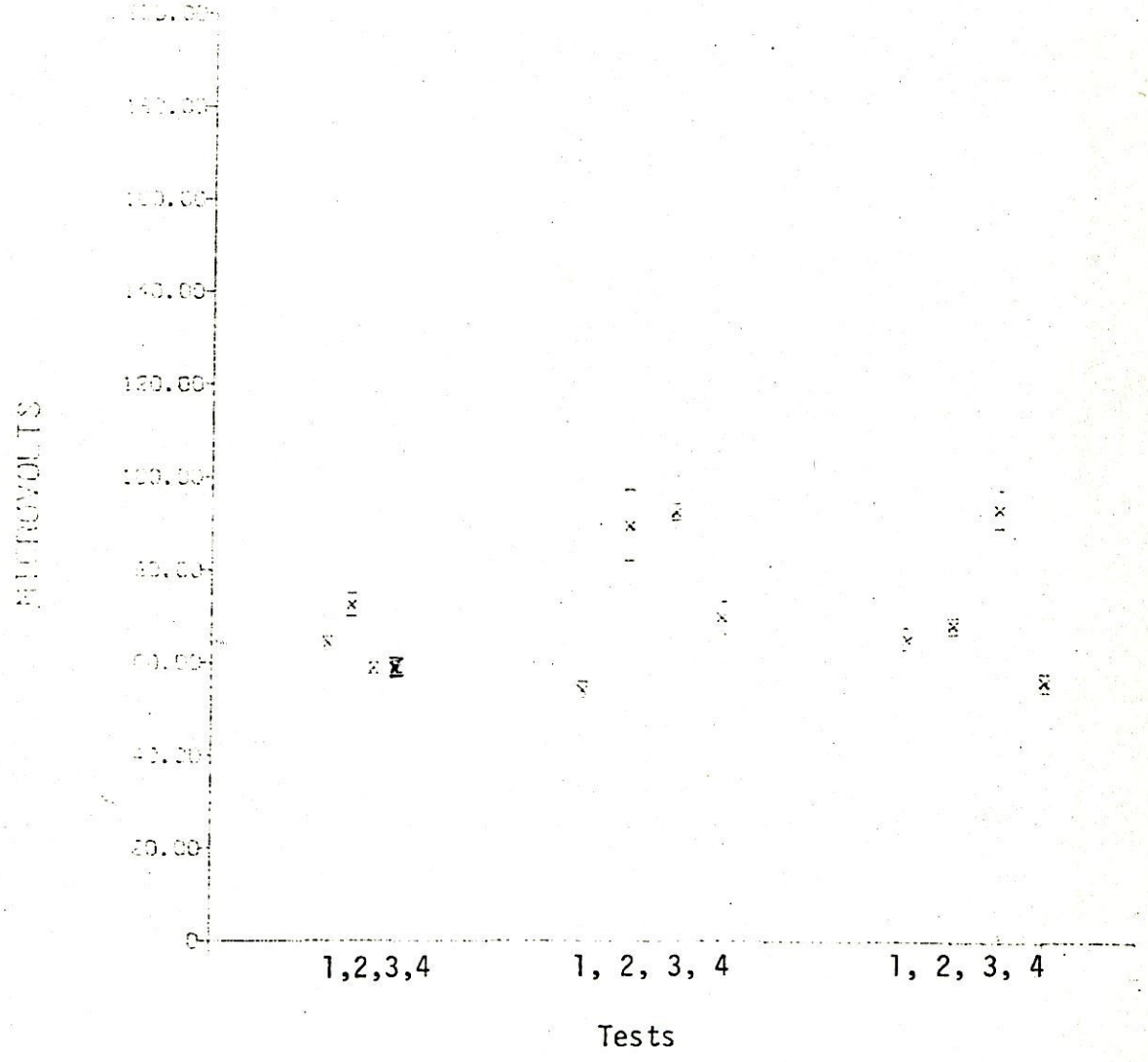
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 9



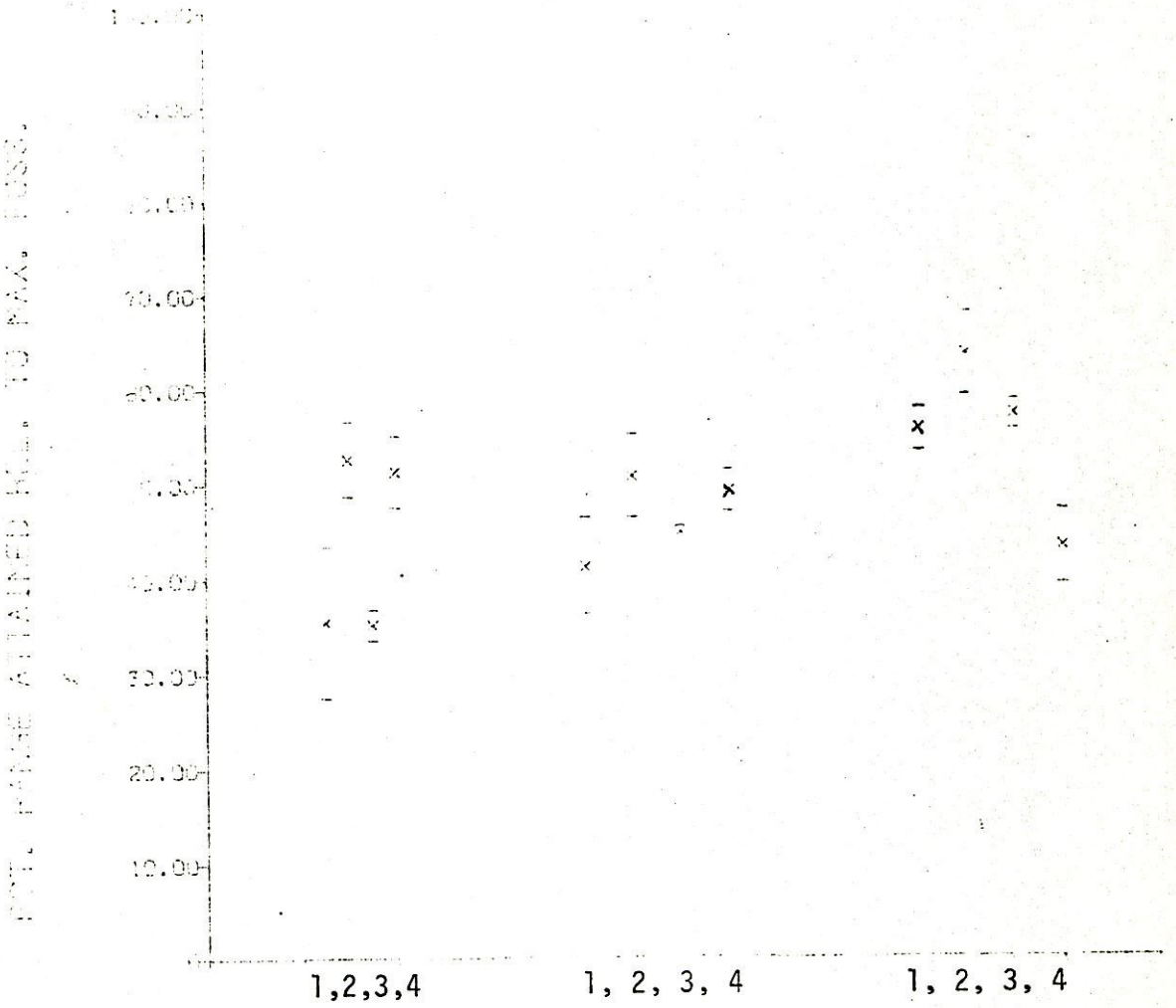
Averaged EMG Activity in Microvolts (SE indicated)

PATIENT 9



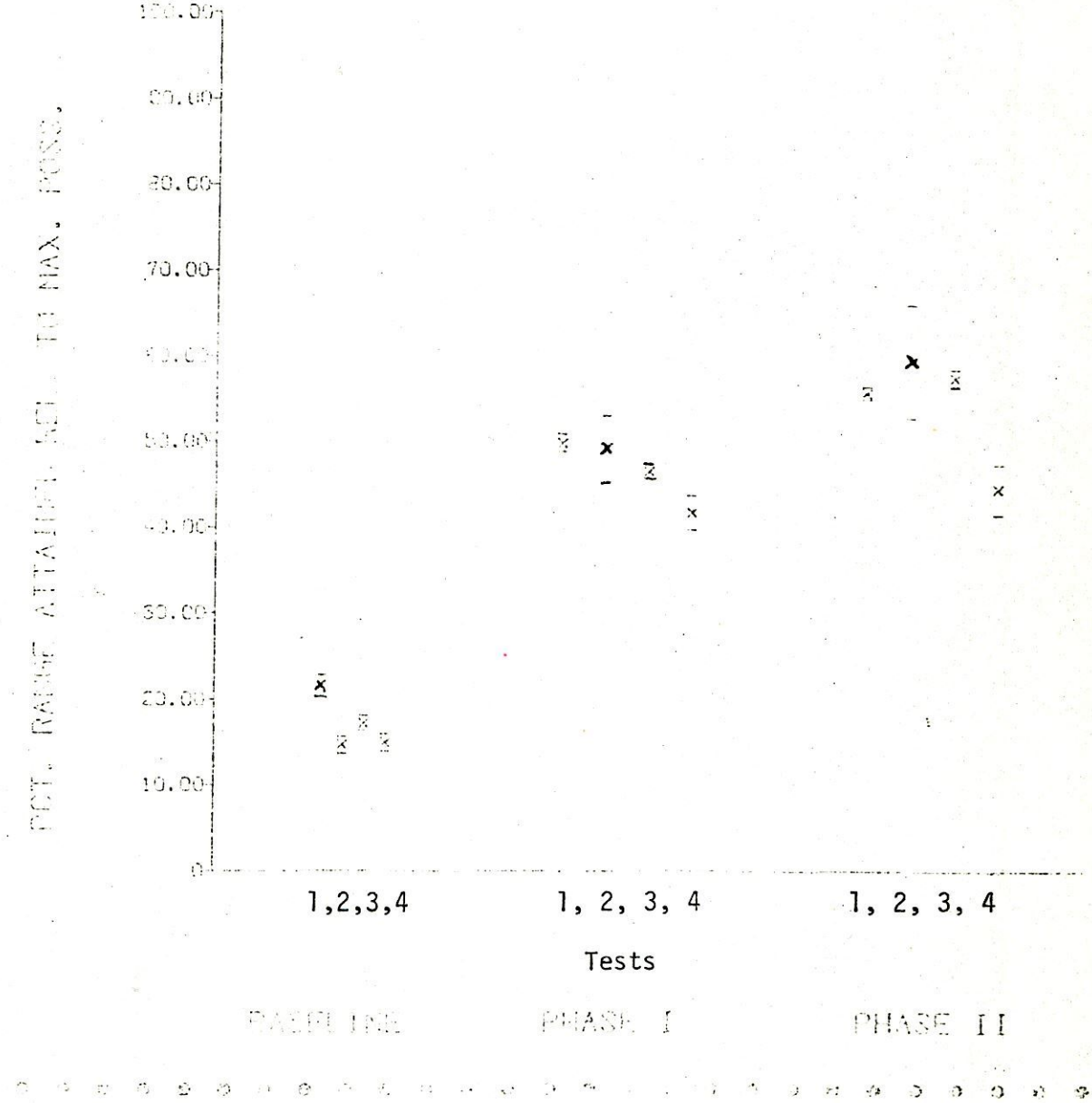
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 9



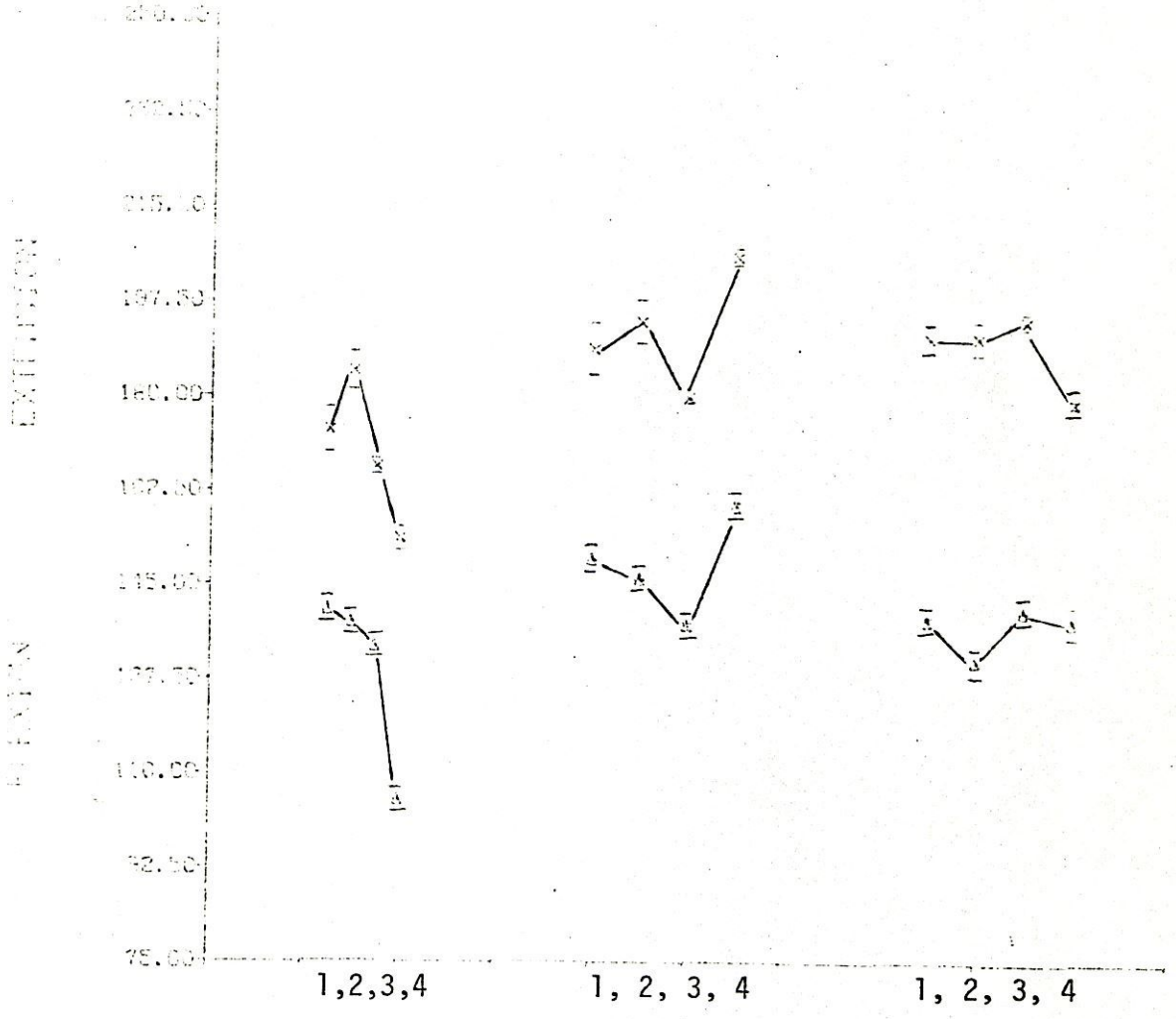
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 9 PART B



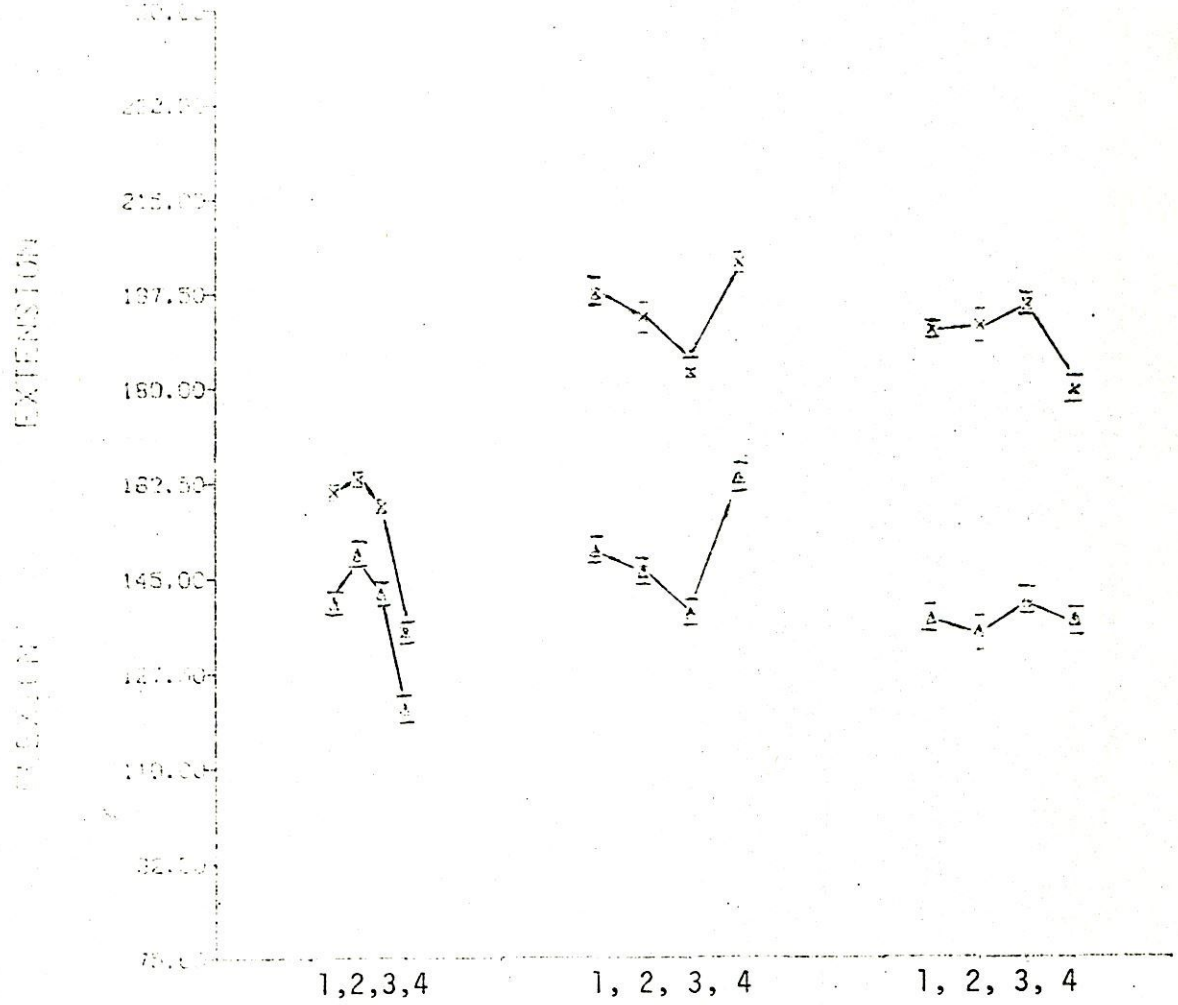
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 9



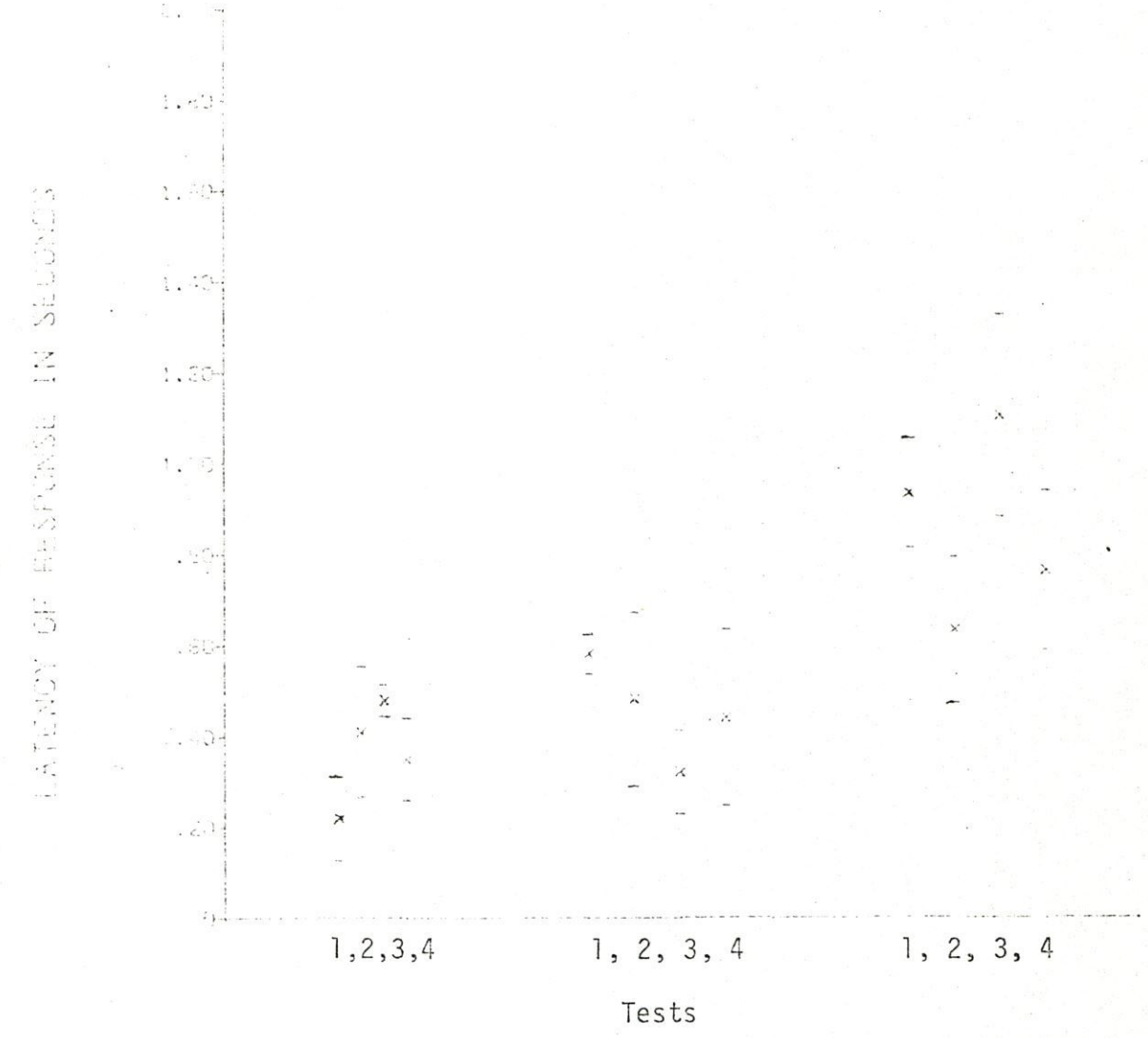
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 9



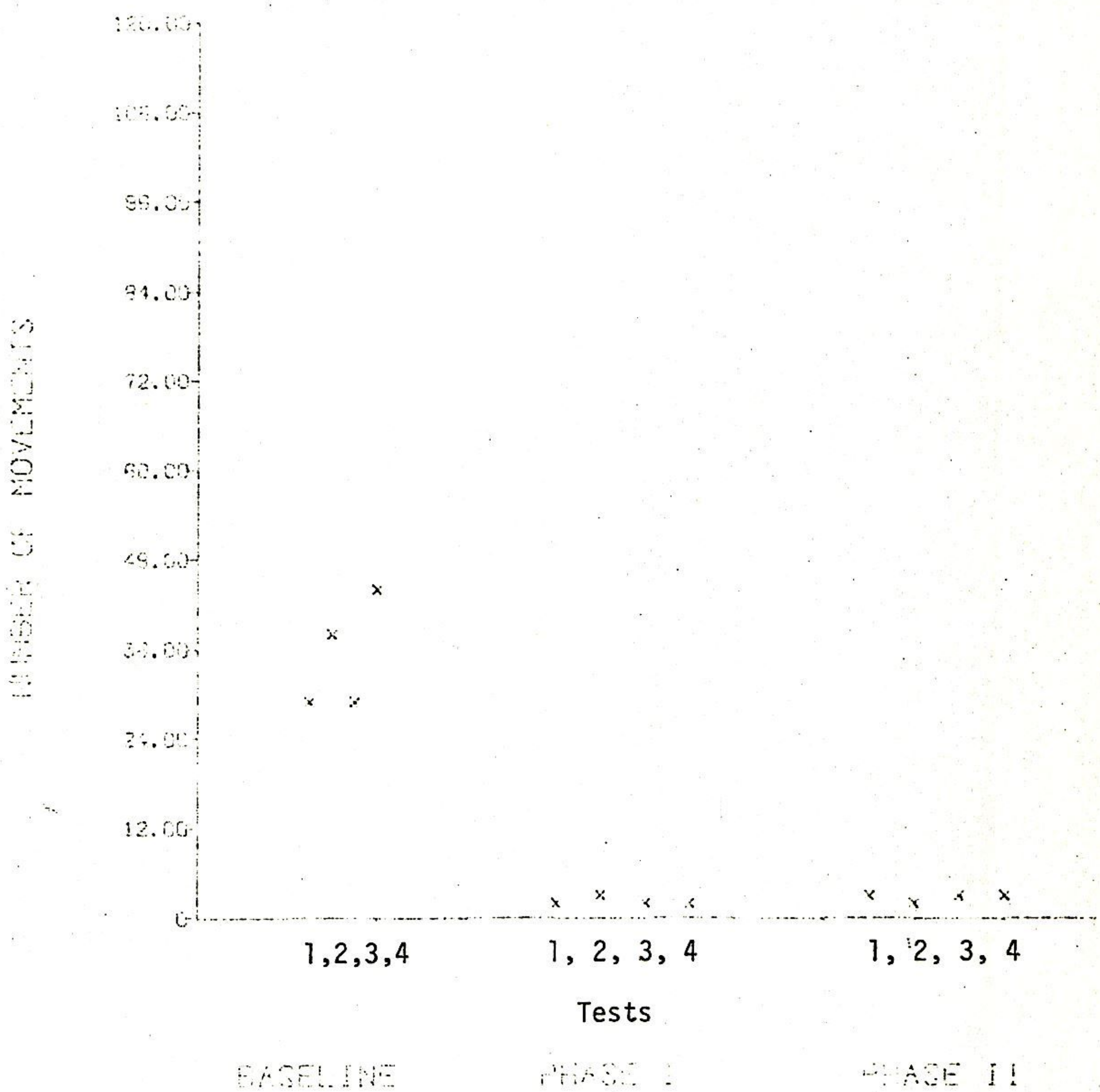
Latency (SE indicated)

PATIENT 9



Repetition of Movement

PATIENT 9



Patient 10

MMPI

L	56
F	66
K	44
Hs	62
D	72
Hy	44
Pd	53
Mf	67
Pa	73
Pt	64
Sc	74
Ma	65
Si	61
Es	32

Code

862'97 1-'3(67)^{0}6,10,9

Patient 10[46]

Wechsler Adult Intelligence Scale (WAIS)	
Full Scale IQ	84
Verbal IQ (VIQ)	80
Performance IQ (PIQ)	91
WAIS Verbal Subtests:	
Comprehension	6
Mathematics	8
Digit Span	4
Vocabulary	10
WAIS Performance Subtests:	
Block Design	10
Picture Arrangement	8
Object Assembly	7
Porteus Maze Test	10
Trail Making Test, Part A	77"-0 errors
Trail Making Test, Part B	132"-0 errors
Bender-Gestalt Recall	3
Wechsler Memory Scale (WMS) Quotient	96
WMS Subtests:	
General Information	5
Orientation	5
Mental Control	0
Memory Passages	10.5
Digits Control	6
Visual Reproduction	4
Associates Learning	16
Aphasia Screening Test (AST)	15-15 verbal errors
LH Tactile Formboard I, Standard	30"-10 objects placed
RH	
LH Tactile Formboard II, Blindfolded	257"-10 objects placed
RH	

---

[46]For this patient, all scores requiring verbal ability are somewhat confounded by an expressive aphasia.

Patient 10, 64 yr old white male; 1 stroke (10 yrs poststroke)

Clinical Diagnosis:
(1) Myocardial infarction
(2) Congestive heart failure
(3) Hypertension
(4) Obesity
(5) Left cerebrovascular accident
(6) Right hemiparesis

Prior Handedness: Right

Prior Therapy: 10 yrs total; therapy devoted to arm and leg for lengthy portions of that time; continual therapy for leg

Comments: None

NEUROLOGICAL EXAMINATION

Description of Weakness:

Spastic right hemiparesis with no movement at fingers or wrist. Trace of movement at the elbow and some at the shoulder. Shoulder does not go beyond 45^0 abduction with 2/5 strength. Springy spastic rigidity at elbow which is kept at 45^0 flexion and at the wrist which is kept flexed as the fingers. Hyperactive reflexes and Babinski sign with sustained ankle clonus on right.

Description Sensory Impairment:

Pin:	Decreased sensation to pin particularly over right hand and arm.
Touch:	Intact
Vibration:	Intact
Position Sense:	Absent in foot.
Two point discr:	Increase in two point discrimination sensitivity from 1 mm to 9 mm on left side and 7 mm on right side.
Graphesthesia:	Moderately impaired in right hand.
Stereognosis:	Mild impairment in right hand.

Aphasia:

Mild to moderate Broca's aphasia with good comprehension.

Apraxia:

Buccofacial apraxia for blowing and whistling. No limb apraxia.

Comments:	This patient is one of the few with overt aphasic symptoms.
Training Objective:	Biceps relaxation
Phase I:	BF
Phase II:	PT

Introduction:

Patient 10 had an extremely dystonic (excessive tone) right hemiplegia. His upper extremity was always held in elbow flexion at 45^0, shoulder abducted, wrist pronated, and fingers flexed, thumb slightly adducted by hyperextended at the MCP and IP joints. He had severe flexion contractures at the wrist. Any command to move, for example, to flex or to extend the elbow, resulted in shoulder elevation, with scapular and vertebral movements to simulate external rotation while the rest of the limb was held in the usual pattern as above. There was some slight ability to flex the elbow. However, during attempts at movement there was effectively no motion distal to the shoulder. Essentially, the rest of the body moved around a static arm.

Patient 10 was severely aphasic although his comprehension was very good. He was extremely well motivated, and cooperative, and practiced at home more than any other patient. He was overreactive in the sense of being extremely quick to respond to any situation. For example, he would be quick to tape and untape himself as E was doing the same to him, quick to get on and off the lab chair, and so forth. His overreactivity as well as a desire to be no bother to E was sometimes, in fact, interfering. Further, his activity level was completely contrary to relaxing a spastic, hypertonic upper limb. He was spoken to slowly and softly in order to minimize excessive arousal.

Patient 10 lived with his wife of three years, and her health was feeble. He attended to all the cooking and household chores, and got around quite well.

Phase I: BF -

During BF Patient 10 had to have his paretic upper extremity propped in a position whereby his adducted shoulder would not interfere with elbow extension. Thus, he had foam pillows fitted up under his arm and fastened around his chest by belts. His hand was wrapped in plastic to facilitate its sliding over the foam. Whenever Patient 10 attempted to straighten the elbow he would also elevate and abduct the shoulder. This could be so strong a pull that his strappings would come undone. E repeatedly told him to stop pulling the shoulder and only straighten the elbow instead. Finally, at the third week of BF, E suggested that he take a look at what he was doing in a mirror at home. Following this suggestion, within a session's time, the patient had completely eliminated shoulder elevation. By the third and fourth week of BF he also no longer needed his props.

Before continuing this history it is important to begin with the following note: the range achieved by Patient 10 when left alone over many minutes to practice with the EMG TV was usually greater than the range achieved in a Testing period. I.e., to achieve an "absolute" maximum amount of range at any one attempt required a very long time period (several minutes), particularly in the first two weeks of BF. In general, the patient could get a certain amount of range relatively quickly and, if left alone for great lengths of time, this range would further increase at sporadic intervals. The latter effect was not an invariable or "predictable" phenomenon. During Testing, range was determined from the terminal angle at which there was no forthcoming change after a one-minute "waiting" period. Also, Testing was not as conducive to increased range and decreased latency as was training

by the patient left alone to practice, free of any "performance" requirements (even though efforts were made to subdue any suggestion of "performance" requirements during Testing).[47]

During the first week of BF, Patient 10 achieved absolute zero on the dot and noise of the EMG TV which meant a relaxed biceps vs. a certain degree of incessant spastic biceps activity and a range of (e.g., Part A) approximately 22^0. This feat satisfied him tremendously. The reasons were two-fold: 1) he was pleased to see success on the TV; and 2) the movement was intrinsically reinforcing since he experienced it as a release of tension. Although able to achieve maximum relaxation, and hence an extended elbow, in the lab setting, the patient's over-reactivity--for example, his quickness at leaving his chair--as well as many general body movements, caused immediate elbow flexion. Thus he was instructed to try to incorporate biceps relaxation into his routine activities of walking and working about the house. The number of times Patient 10 achieved "quiet" on the EMG TV continued to increase during BF and his latency to terminal elbow extension, which was very long at first, continued at a variable but overall decline over sessions. The patient reported his arm as feeling "real good", "relaxed", and

---

[47] During baseline where his response was extremely limited (i.e., when there was minimal range), response duration (time from initial ascent of averaged EMG activity for elbow flexion to terminal descent of averaged EMG activity for elbow extension) was quite short (e.g., 1 second) and Patient 10 was given the usual 5 commands for Part A (Command). At training onset, however, except for Test 1 where he was given 4 commands, Patient 10 was given 1 or 2 commands in Part A during Phase I (BF) as his total response duration was inordinately long (e.g., one minute). During Phase II, he was given 1 command in Part A as response duration remained long in Phase II. The increase in response duration is quite evident in Part B (Rate) where Patient 10 inevitably could only achieve 1 response per the one-minute period.

generally speaking said there was more "feeling" in his arm (the latter was probably an effect due to the sensation of and attention to movement). He began to walk with his elbow extended at times (until he would jar himself up or onto or off of the lab chair and his elbow flexed). At the end of BF the patient used the paretic limb to straighten his newspaper and push plates off the table.

Averaged EMG activity over the four BF Test sessions, which was in the direction of decreased biceps activity, was comparable to or less than the level of activity in the biceps of the contralateral limb during elbow extension as tested at baseline. There was relatively very little room for improvement on the EMG measure. Range and latency are the outstanding dependent measures for this patient. Range showed continual dramatic improvement with (e.g., Part A) approximately 72^0 at Test 4 of BF vs. an average of 13^0 during baseline. As there was virtually no range during baseline, latency at baseline is quite short. Latency during Phase I, however, corresponding to the achievement of continual increments in range, started at 10.4 seconds and continued to decrease over sessions to 1.3 seconds at Test 4 of BF.

Beyond biceps relaxation, a critical problem for Patient 10 was greater voluntary control of elbow extension (that is, not having to "wait" for the biceps to relax as he always did, sometimes in a pensive, concentrating posture), or, in other words, shortening the latency at will. He also needed triceps activation to help push the elbow down rather than let it drop slowly by gravity to extension. It was noticed towards the end of BF that when the patient felt he had achieved maximum extension, but was still several degrees short of

180°, his inadvertant turning of attention to something E was doing would cause a final drop to 180°. In sum, although BF was an invaluable tool for biceps relaxation in this rather dramatic case, true inner, completely voluntary controls were only beginning. It might also be mentioned that E would surmise BF as particularly useful in cases such as those of Patient 10, where, a priori, agonists and antagonists contracted together with little motion resulting anywhere in the limb distal to the shoulder. Relaxation of a muscle in such a spastic extremity is perhaps a more easily accessible goal via BF than other methods. (Post-experimentally, E was contacted by a physical therapist who is currently working with Patient 10. The therapist commented on the practically insurmountable problem of relaxing a limb such as Patient 10's via conventional methods available to him. This further reinforces E's opinion that for a case such as Patient 10's, BF commends itself as a method to use initially as part of an individualized rehabilitation program to train some fundamental relaxation in a highly problematic target muscle before trying to obtain any active contraction activity in that muscle.)

Phase II: PT -

It was felt that Patient 10 could continue to benefit in three ways: 1) primarily by heavy emphasis on biceps relaxation; 2) perhaps an even further shortening of his latency through (1); and 3) triceps activation to achieve voluntary elbow extension. It was emphasized to the patient to continue practicing biceps relaxation in order to maintain the gains he had already made. Most attention therefore was given to biceps relaxation. Along with this he practiced general limb

relaxation. He was also given facilitation and resistance training to the triceps, but this was discontinued immediately because it caused biceps contraction. During PT, it shortly became evident that Patient 10 was losing ground in biceps relaxation in that latency started to increase although range of motion held steady for a longer period of time. Furthermore, it seemed that EMG voltage at elbow flexion was also lessening. As mentioned, in the PT phase, training aimed at relaxing the entire limb as well as the biceps. At the end of PT, Patient 10's fingers grew increasingly relaxed during total limb relaxation. His fingers could now be passively extended with little resistance, if done slowly, whereas this was not possible previous to PT. From a semi-flexed position he could now flex the fingers voluntarily. He had no finger movement prior to PT training.

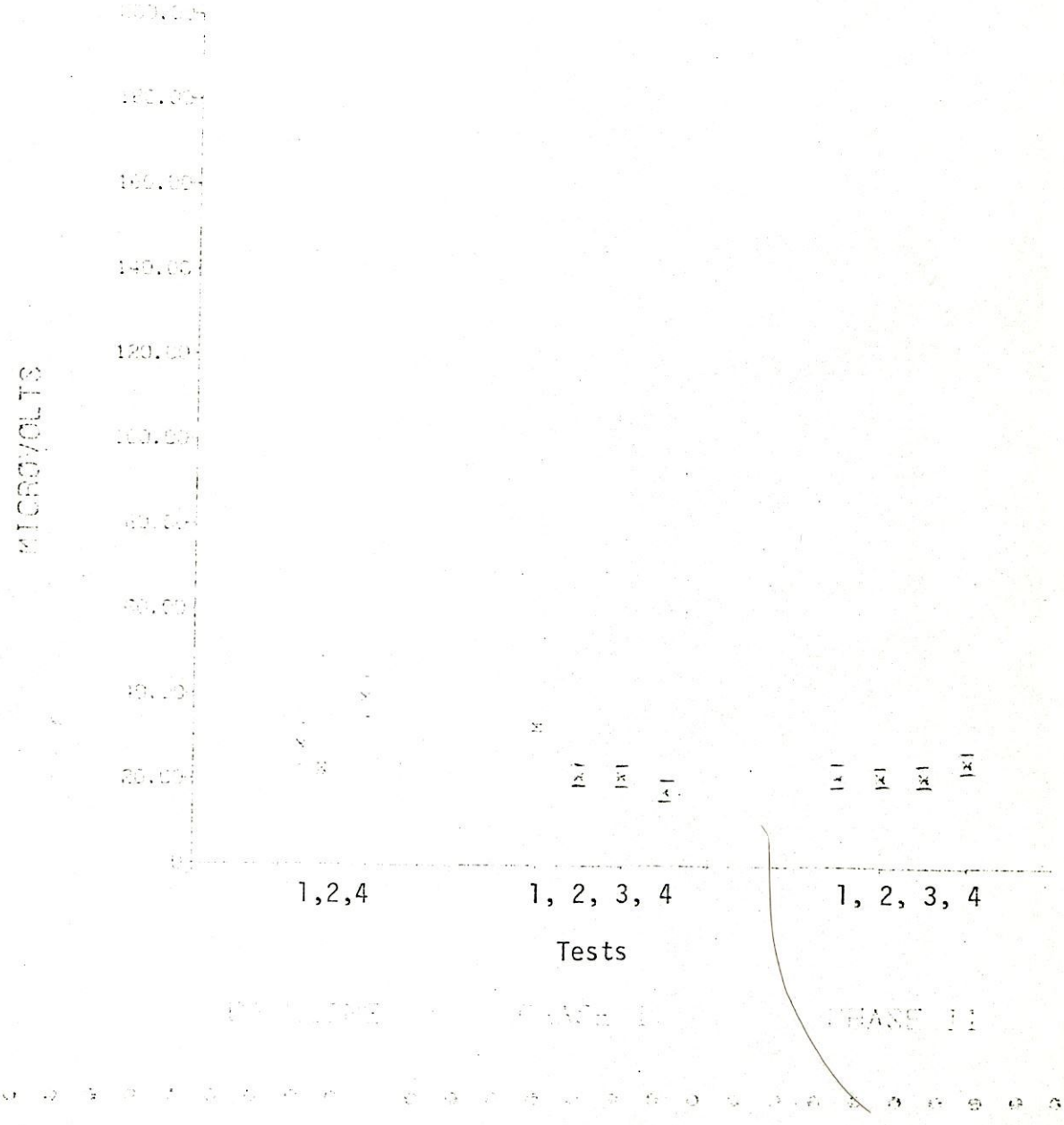
Comments:

Patient 10 was evaluated as having lost ground in biceps relaxation during PT, and it was felt that continued BF may have meant continued progress in this muscle. In other words, in the biceps, where he received BF prior to PT, he appeared to be losing what he had learned. At the same time, it is true that learning relaxation appeared possible via PT in areas which had no prior exposure to another modality, namely, finger relaxation and movement.

Patient 10 does not have any scores for test 3 of baseline as he missed this session and it could not be included within baseline time constraints.

Averaged EMG Activity in Microvolts (SE indicated)

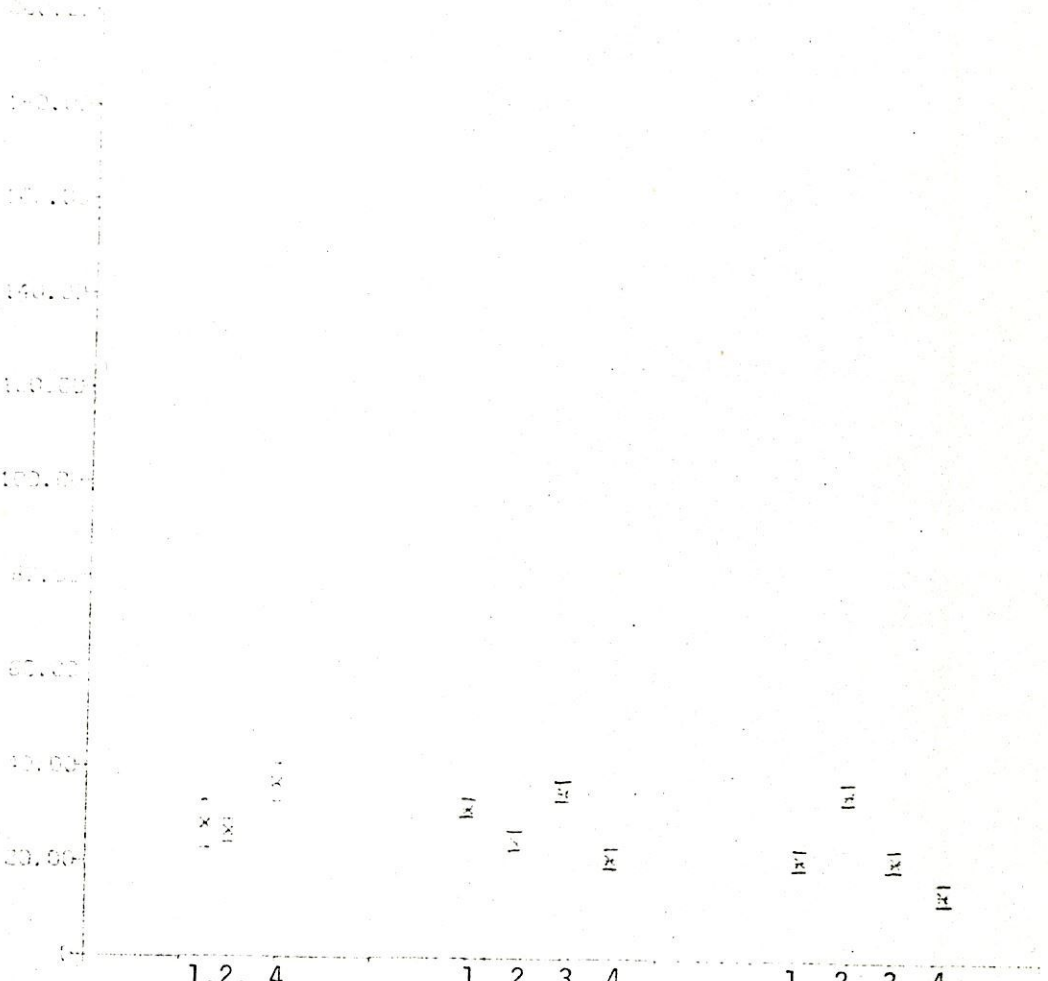
PATIENT 10*



*Patient 10 was training for a decrease in averaged EMG activity. All other patients were training for an increase in averaged EMG activity save Patient 3 (see graph of "Lowest EMG Activity" for Patient 3).

Averaged EMG Activity in Microvolts (SE indicated)

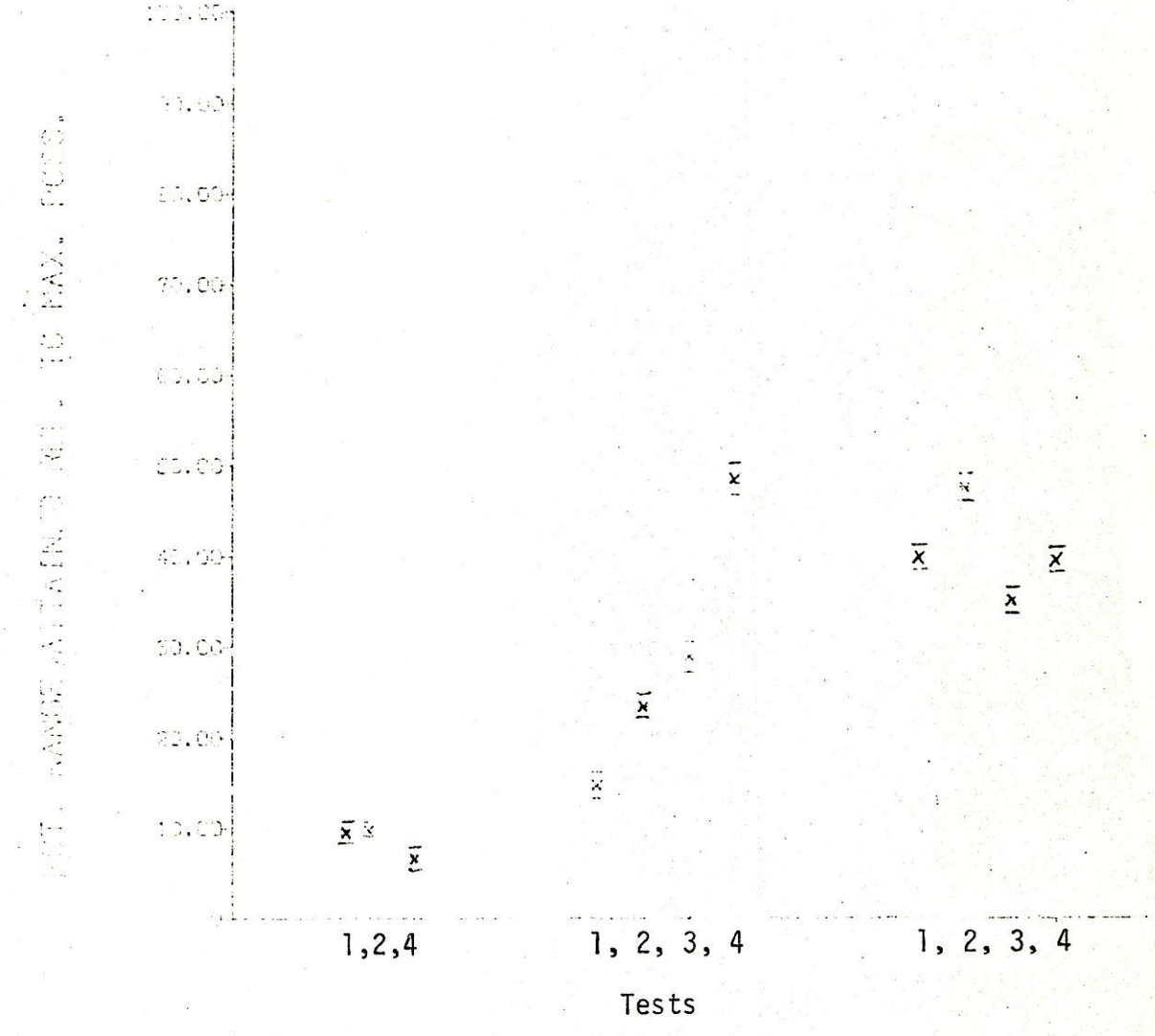
PATIENT 10*



*Patient 10 was training for a decrease in averaged EMG activity. All other patients were training for an increase in averaged EMG activity save Patient 3 (see graph of "Lowest EMG Activity" for Patient 3).

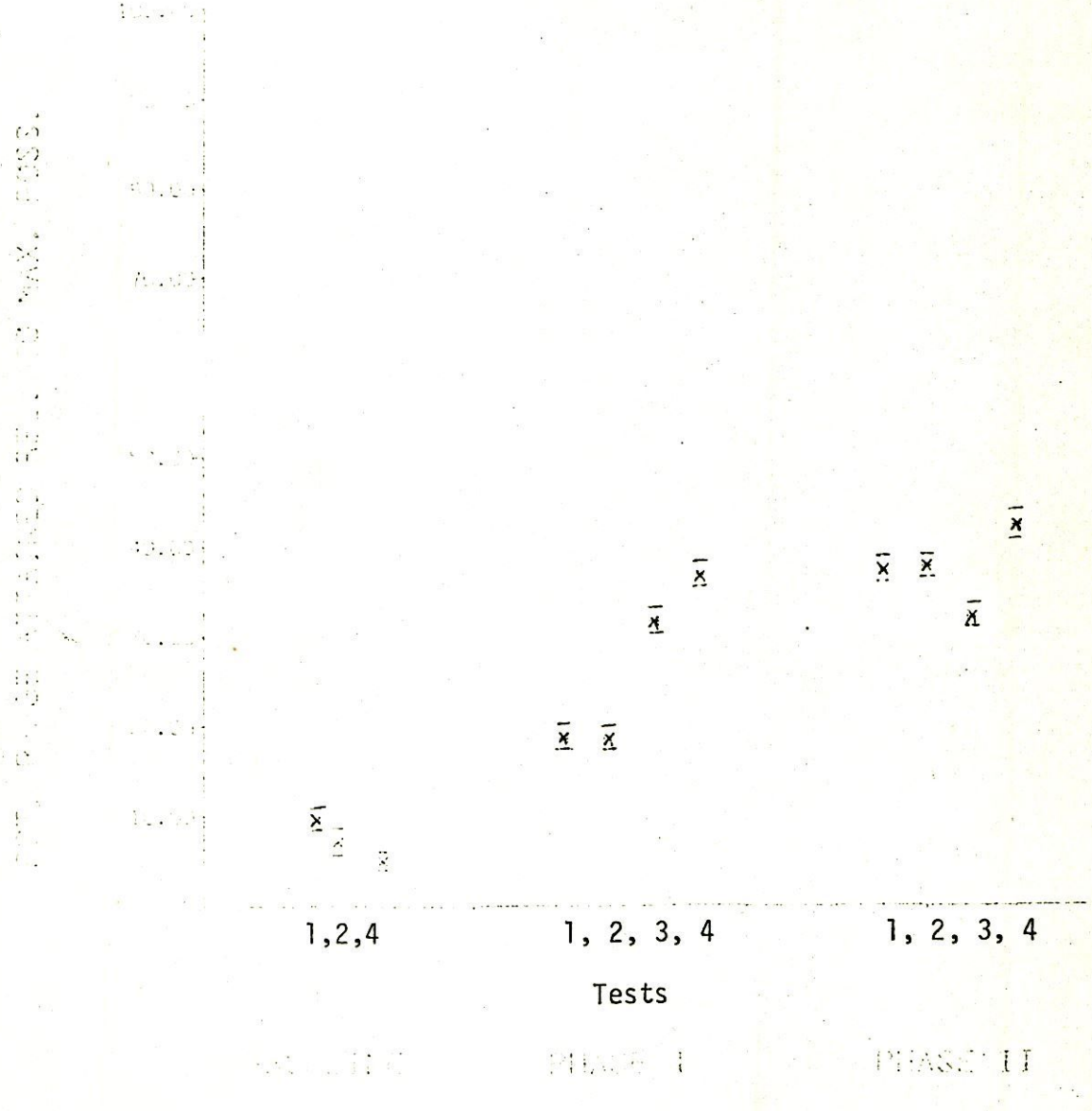
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 10



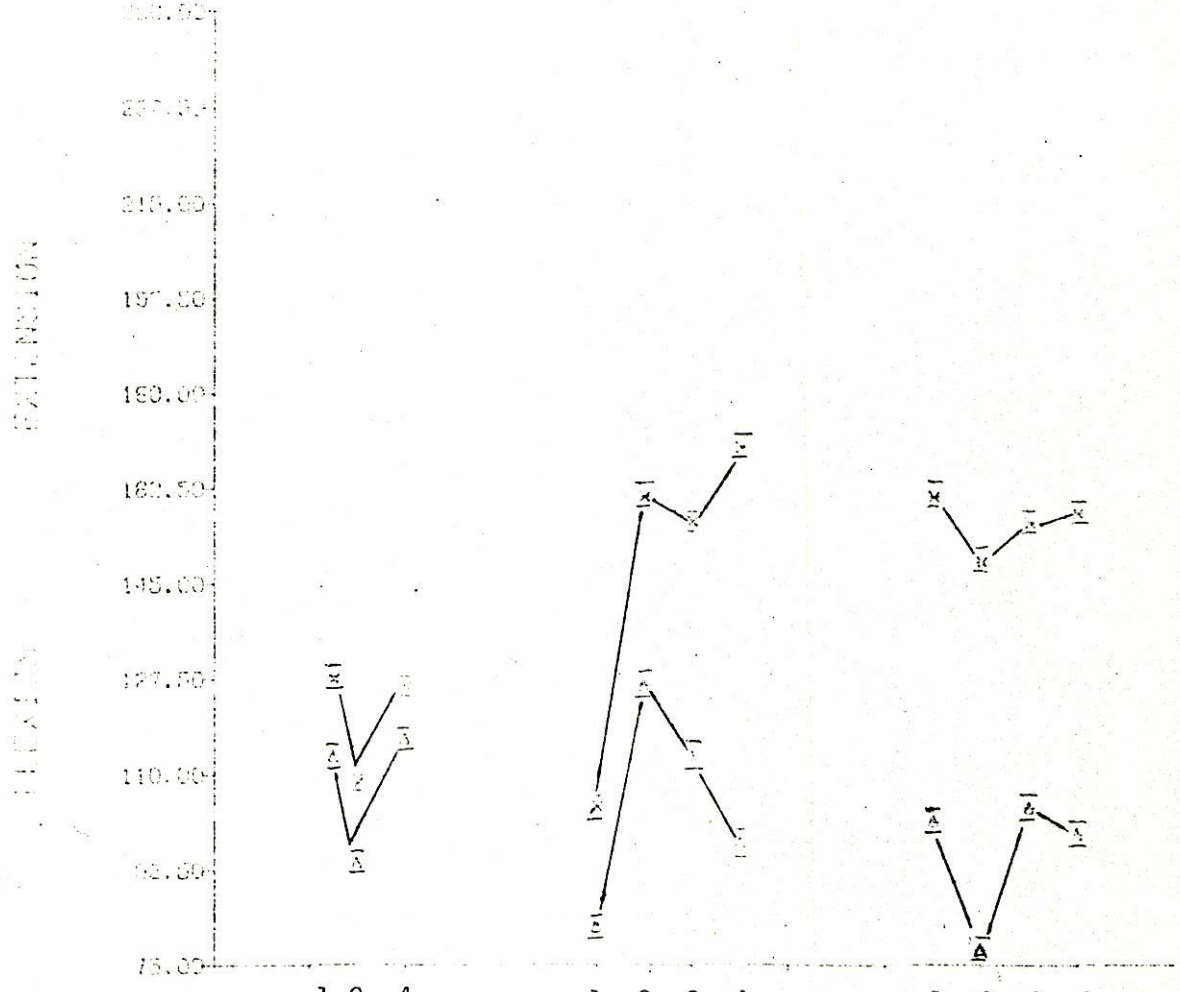
Range of Motion in Percent--
(Absolute Range Attained Relative to Maximum Range Possible)
(SE indicated)

PATIENT 10 PART B



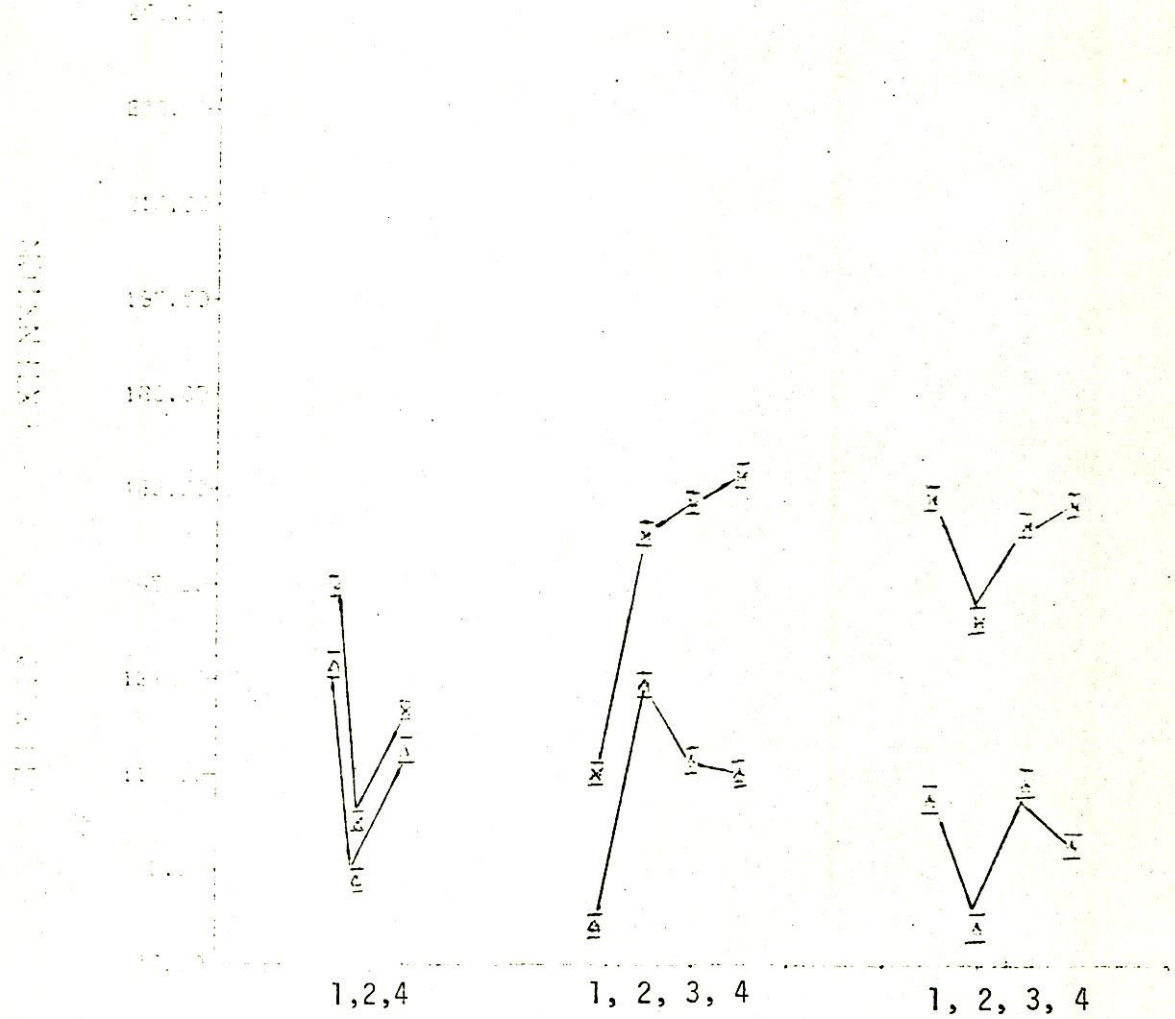
Extension ($\bar{X}$) and Flexion ($\bar{A}$) in Degrees (SE indicated)

PATIENT 10



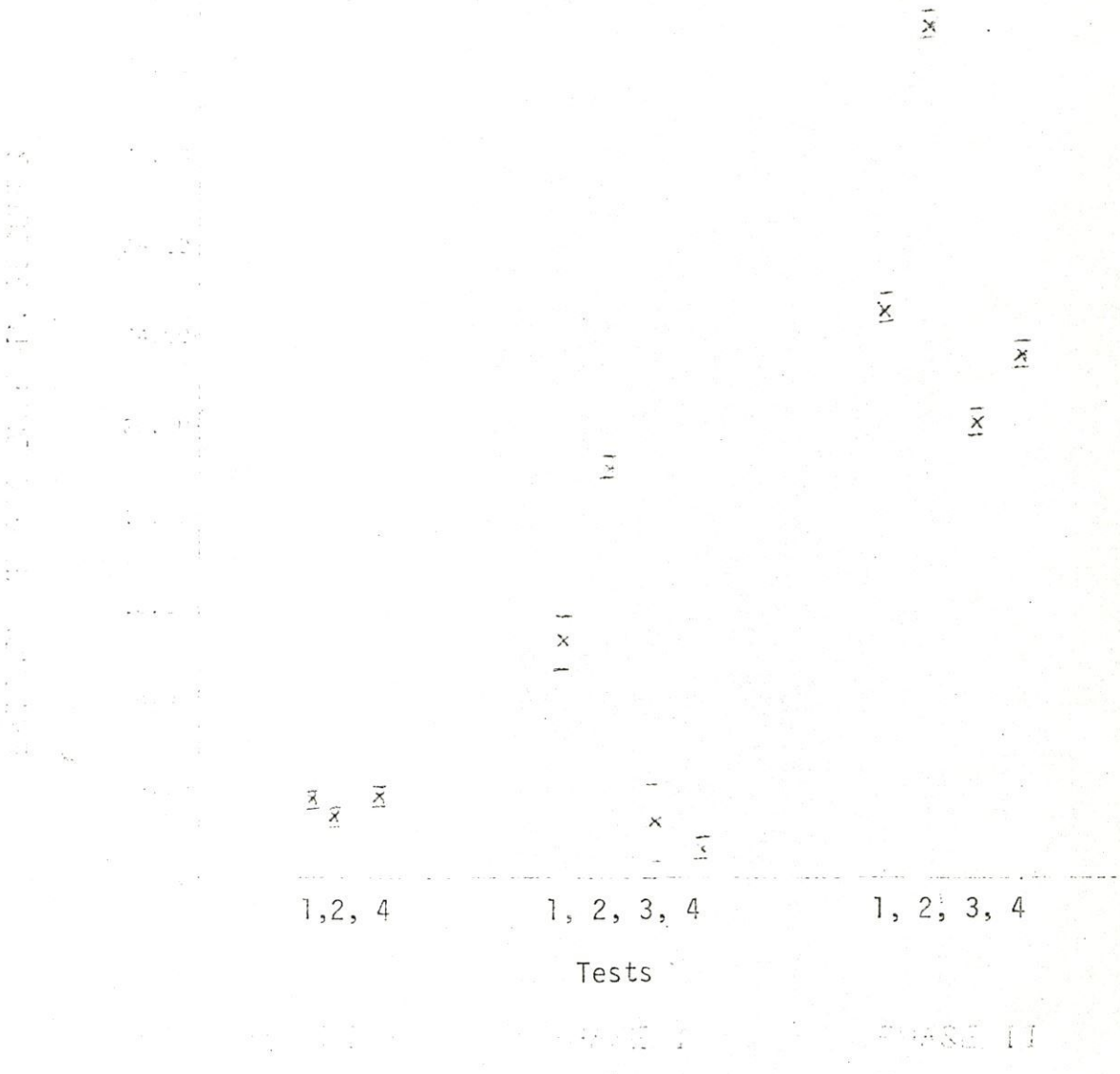
Extension ($\overline{X}$) and Flexion ($\overline{\Delta}$) in Degrees (SE indicated)

PATIENT 10



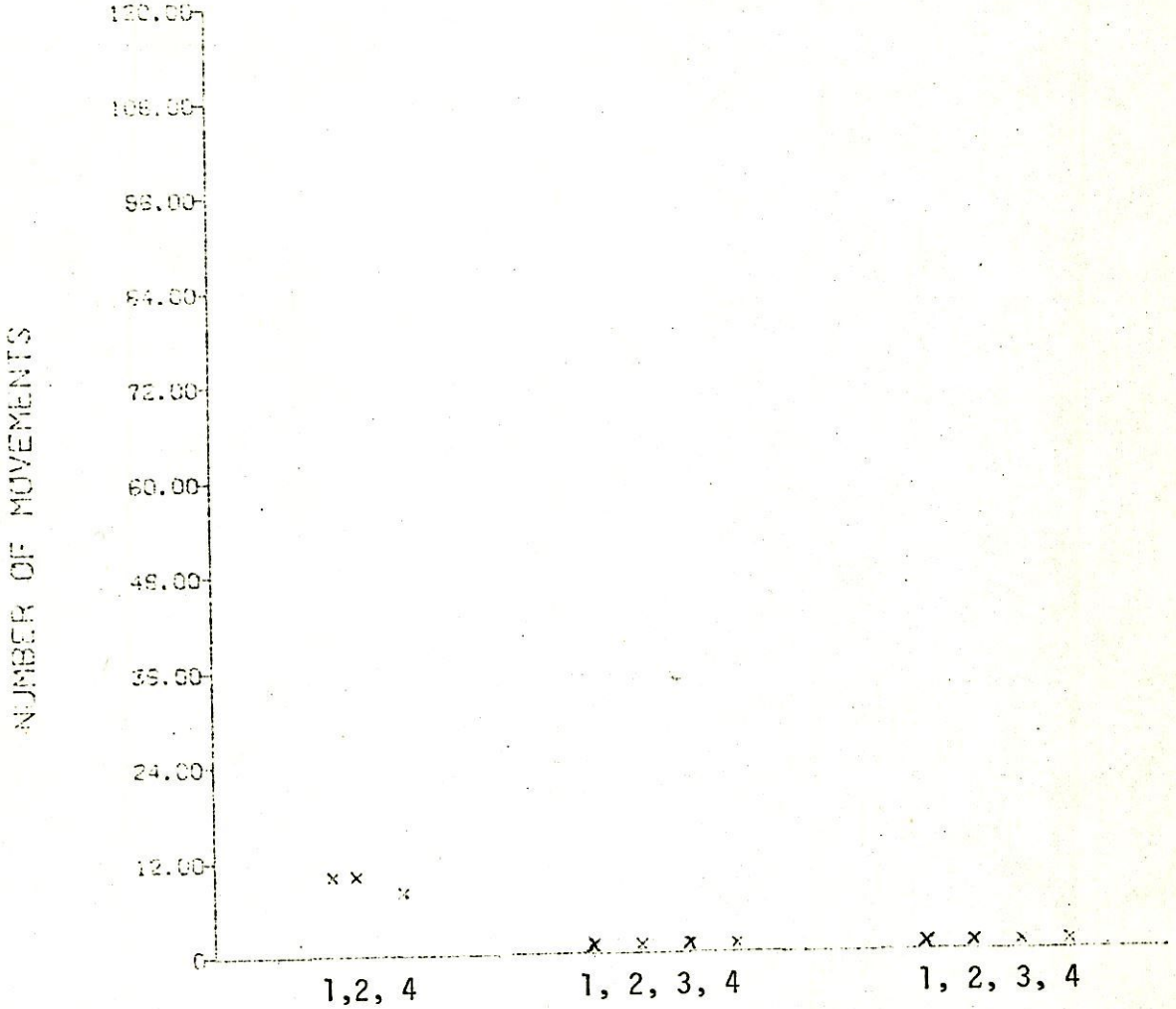
Latency (SE indicated)

PATIENT 10



Repetition of Movement

PATIENT 10 PART B (OrP. LIMB 133 , 131)



SECTION XV

DISCUSSION

Statistical results showed that there was no learning difference between the Part A (Command) and the Part B (Rate) situation for either averaged EMG activity or range of motion. That there was no difference between Parts A and B, is probably due to the fact that patients were instructed thoroughly and repetitively to execute a response in only the most correct and efficient manner, and hence replicated a typical performance under either the Command or Rate situations. The following comments apply to both Parts A and B for averaged EMG and range of motion performance.

For an analysis of the averaged EMG activity, characteristic differences between patients and the performance of patients comprising both groups were discussed in Section XIV under Averaged EMG Activity, Intergroup and Intragroup Comparisons. The discussion pointed to certain problems which had to be taken into account in any between-group comparisons.

One result obtained from an analysis of the averaged EMG variable was significant pooled group training effects for both BF and PT with, however, a suggestive trend or more EMG recruitment under BF training (pooled group) vs. PT training (pooled group). There was significant learning under either training for Group 1 (PT scores, however, carry the BF residuals for Group 1), and a suggestive trend for more learning under BF for Group 2 (BF scores, however,

carry the PT residuals for Group 2). Reasons for the superiority of BF over PT training were discussed in Section XIV with the summary conclusion that, because of gross procedural differences which were sometimes opposite in emphasis in terms of EMG recruitment, PT is not amenable to direct comparisons with BF for EMG recruitment. For averaged EMG activity, PT was, strictly speaking, a control treatment.

All patients included in the averaged EMG activity analyses were training EMG increments. The major conclusion which can be drawn from these patients is that BF training was effective in increasing averaged EMG recruitment.

Finally, Patient 10 trained for an EMG decrement in his biceps during elbow extension but was not included in the EMG analyses--(see footnote p. 131). For Patient 10, BF was a very helpful instrument for illustrating biceps relaxation. (See the case history for this patient). Also, Patient 3, who was not included in any statistical analyses (see p. 127), trained for an EMG decrement during wrist extensor relaxation. BF was a helpful modality but PT training seemed more effective for dealing with the problem. (See, e.g., the case history for Patient 3.)

The major result obtained from an analysis of range of motion was that BF and PT were equally effective in increasing range. Also, consideration of the apparent relationship between range and averaged EMG activity seemed to indicate that averaged EMG activity and range of motion could vary more or less independently.

The major result obtained from an analysis of latency was that in the first test session of BF and PT (regardless of phase) latency increased over baseline. Furthermore, the initial increase in latency

was greater for PT than BF. Latency was expected to increase immediately after baseline, and it was also expected to fall over the four sessions of a given phase. However, latency remained the same for BF while it continued to decline for PT. The decline for PT continued to a level which was less than that of BF at the last session of a phase.

Finally, repetition of movement for Part B (Rate) was much reduced during training as opposed to baseline. The reason for this decrease was discussed in Section XIV, "Repetition of Movement".

BF vs. PT in the Clinic

Initially this author intended to probe the psychological possibilities of self-conditioning of unconscious physiologic activity particularly in persons who had had some relevant components of their physiologic structural apparatus destroyed or traumatized. However, she is obliged to address the question of the clinical value of PT vs. BF. To begin this explication the reader must bear in mind that it is felt that the former goal, to be discussed in "EMG BF and Psychology", has been shown; but the study as a whole cannot present the isolated psychological ramifications of EMG activity conditioning and the circumscribed clinical uses of the EMG machine. It must deal with the difference between increased physiological EMG activity vs. increased function. It must furthermore, demonstrate that how function (motor) evolves and what ultimately is meant by the achievement of "function" is an entirely separate issue from the conditioning of motor unit recruitment in one target muscle as is, strictly speaking, the method and potential of BF training.[48]

EMG and Range as Related to Function

To begin, in this study, range is a more functionally, i.e., clinically, revealing variable, than, for example, the EMG variable, because it is to a greater extent, the result of a group of related activities contributing to an emergent property of motor control (skill). Although range is a quantitative parameter, it does not conceptually carry the same meaning of quantity as does the EMG

---

[48] And also as was, strictly speaking, the explicit design for BF training in this study.

variable. The former is a neutral number of degrees on an arc, which is actually the result of a synthesis of the activity of interrelated and often diverse elements contributing to ultimate movement; The EMG variable, on the other hand, is literally a matter of elements of EMG activity (motor unit action potentials) appending each other. Evolution of motor control is somewhat different for range of motion than for EMG recruitment. For range, if the agonist itself can contract or relax as necessary, and if the whole of muscles around the joints are functioning properly in that agonists and antagonists are being appropriately excited and inhibited, and non-specific or extrinsic motions are abrogated or in the process of being inhibited, range will be able to improve. Also if spasticity or clonus is not too interfering, if contractures are minimal, etc., again, range will be able to improve.

For EMG recruitment, greater target muscle contraction alone is distinct from many of the aforementioned "variables" which are important to the evolution of range. This is supplemented by the fact that range, in terms of function, depending on the individual patient, does not seem to be a necessarily proportionally incrementing quantity with different levels of EMG recruitment. As discussed in Statistical Results, range increases can be parallel with EMG decreases and EMG increases can occur in the absence of range increases or in the presence of range decreases. One clear case of the functional aspects behind the above events is presented below.

In BF (Phase I for Patient 9), Patient 9 changed her baseline EMG recruitment level (e.g., Part A) of 70.7 μv, to 80.0 μv--a gain of 9.3 μv--and increased her range to 44.1^0 over a baseline level of 41.4^0.

In PT, she decreased her μv level again by 4 μv to 76 μv but increased her range to 60^0 over the baseline level of 41.4^0. The change in range in PT and a slight EMG decrease was due to comprehensive instructions deriving from PT principles aimed at effecting the qualitatively most correct (i.e., functional) movement. The latter, for Patient 9, was not effected by EMG increases (which caused an array of detrimental response features [see the case history for this patient]) but rather by greater muscle relaxation (less or steady amounts of EMG recruitment) to achieve (within the limits of contracture) a greater and smoother range response. Patient 9 made general improvements in function during BF too. The improvements occurred, irrespective of very little EMG increment, because of the multiple instructions and attention given Patient 9's target movement. There is, then, an essential difference between motor evolution conceptualized in terms of sheer EMG recruitment vs. evolution of range increments which are part and parcel of functional change.

For EMG recruitment, theoretically, physiologically what is happening is the re-use or the re-making of pathways (i.e., structural changes in whichever sense such change may be formulated--for example, an increased or decreased neural inhibition, excitation, or use of new pathways, etc.). This literal process is not knowable to a patient especially in BF except through secondary, indirect means (i.e., feedback outside the body) which is not merely one step removed, as, e.g., is an exteroceptive cue which reinforces a skill or skill component of which a S has immediate realization or awareness. Rather, this physiologic increase in activity, is remotely related to S's primary, corporeal awareness, and hence, physical sensory mental

experience, except as post hoc peripheral sensory reafference is available for efference to be felt. The latter feature, if present, reinforces and gives validity to, for example, the BF type of informational stimuli and experience.

The Clinical Goal: Return of Function[49]

If a goal of physical therapy, i.e., therapy for the body, is motor skill, function then, ideally, is the fitting, efficient, synergically appropriate use of muscles as comprehensively applicable to, e.g., a limb. In other words, given function, a person who wished to tip his hat would move the required body parts to get his finger tips to his hat by specifically and automatically contracting his muscles at several body points in a particular direction at a particular speed. The sequence of contraction would be rhythmic and each muscle would contract with appropriate strength at the necessary moments in time. (For further discussion, see, e.g., pp. 26-27). For a person who has normal function, such movement is ballistic and the subsidiary elements are unconscious. The goal alone suffices to achieve the very complex interplay of muscle contractions, inhibitions, and accompanying joint movements, etc. If a person has less than complete functional control, he may have to concentrate on attending to his muscles, and limb, by some means, to get to his hat. A person with very poor function may, e.g., at best be able to raise his arm in an obligatory stereotyped motion (the nature of which is described in

---

[49] Skill is emphasized in function as function may connote any incompetent use of a limb to a goal. The view here is that function should aim for the most effective use of a body part. For example, one may raise his wrist as the end-product of a gross movement of shoulder retraction with elbow abducted, etc., vs. raise his wrist isolatedly with such patterns eliminated.

patient case histories), a motion which will not suit his purpose; or, if the motion is sufficient to touch the hat, it will be totally lacking in grace and coordination--in sum, lacking in the total design or pattern necessary for proficient, and efficient use.

Furthermore, to touch the hat, a person must have a primary knowledge, conscious or unconscious, of his limb in space, of his limb relative to his hat, of the degree to which he is attaining his goal moment to moment, etc. Enough has been said to suggest that the factors indigenous to good function are extremely complex and are ordered within a system of communication between mind and body that cannot be subsumed under simple, singular formulae or rules especially when a patient with his own unique sensorimotor handicaps presents himself for therapy--i.e., return of function.

Given the convergence of the necessary conditions for a certain amount of function as exemplified by, e.g., range, gains in function (and range) will be most extensive in a consistent individualized program of rehabilitation. Furthermore, once a new level gradient is achieved, there is apt to be a decrease in function when the training regimen is changed (e.g., especially see Case Histories to see how these changes may occur) and a new input system causes a disturbance of gains to date. In this study the new input could range from elimination of undesirable response features to the use of new cues.

Conclusion on the Evaluation of, in Particular, the Role of BF in Clinical Practice

If motor activity is known, i.e., experienced and guided, through sensory monitoring, theoretically, then, gains which are made via PT--i.e., gains made by virtue of knowledge or apperception of

primary peripheral sensory stimuli--would seem less subject to deterioration (i.e., better retained) if practiced and, more importantly, used, than gains made by bypassing such experience. Bypassing such experience would be the case in those instances where Ss feel no sensation from EMG BF, or where Ss have no sensory function (e.g., position sense) for purposes of PT.

If gains in motor output are made, the most fundamental and direct gains are those made, if possible, by physical therapy, that is, by physical sensation, the demarcation of which is the skin, joints, and muscles to the CNS. This is because such gains do not require translation in inner sensory experience--the experience is direct. The pathway to motor efference in PT is the peripheral sensory aspect of sensation where cues are immediate elicited, subjective experiences (i.e., feelings) (in addition to components of attention, comprehension, etc.). Corollary discharge, ideation and exteroceptive events, can feed back to an organism the cerebral knowledge of efferent activity which may or may not be sustained by translation to inner experience (i.e., be subjectively incorporated into a sensory schema of the body), to result in ultimate motor control (function) of, for example, a limb.

This is precisely why many investigators have questioned the retentivity of EMG BF skills when sustained only by technology or inadequate input. However, in a S who has adequate sensation, efferent activity which is propelled by EMG BF would seem accessible to sustenance and substantiation by the internal sensory experience of movement. In any event, immediate conscious or unconscious afference of efferent events is the most efficient, if feasible, mode of training, as the sensory and motor are but two aspects of one process.

In addition, the following points are of prime importance to this discussion. In any physical therapeutic training where BF is used as a stimulus modality, the most important feature is not the BF stimulus but rather how the BF stimulus is used. Myriad methods and maneuvers must be utilized by (a) the therapist to help the patient in every way possible to effect a specific, skilled type of neurophysiological change, and (b) the patient, to ultimately realize some sort of sensory awareness of his limb in order to achieve a durable, spontaneously available motor pattern. The latter aspects of therapy, regardless of stimulus input, constitute what is known as physical therapy. This paper contains voluminous reference in Method and especially Case Histories to all of the means used during both BF and PT training to effect stimulus perception, meaningfulness, and effectiveness.

During BF training, this study utilized, in essence, a new stimulus modality within a restrained method of physical therapy, where E was only permitted unceasing verbal and, on occasion, visual guidance to help the patient realize what he was doing in order to use the stimulus and use his limb. What the patient did use most of all from therapy to effect function was the guidance and feedback from E; and, if the patient truly established some new motor repertoire for himself, he did so by guidance and feedback from some sensory limb awareness as a supplement to the information he received from the BF stimulus.

In sum, BF is part of an entire therapeutic process, and, a more effective part if the procedure which is physical therapy is not constrained by experimental limits. Furthermore, as "Considerations and Comparisons Between the Current and Related Studies" will

point out, other studies which have used EMG BF for neuromuscular education in victims of stroke usually either report having utilized comprehensive PT or some PT methods in addition to BF input. From the current author's experience, strictly isolated BF input cannot produce rehabilitation.

Patients for Whom BF was a Valuable Training Modality

Patient 7 and Patient 10 are the two patients for whom BF was a very effective treatment modality. Each of these patients was at an opposite extreme of pathologic muscle tone. Patient 7 had a completely flaccid hemiparetic upper extremity while Patient 10 had an extremely hypertonic hemiparetic upper extremity. The one phenomenological characteristic these patients shared was barely minimal spontaneous voluntary movement anywhere in the wrist and elbow joints of the affected limb: the former due to very minimal muscle contraction ability and the latter due to extreme muscle tone and spasticity to the point of rigidity.

Patient 7

Patient 7 had severe impairment of sensory function and a very weak elbow flexion response (his target movement) which was elicitable only by, e.g., stimulation of the stretch reflex. There was, thus, both minimal motor, and presumably very minimal concommitant sensory reafference, associated with the elbow flexion response. Patient 7 had an inattentiveness to his affected upper extremity precisely because of poor sensation in the limb. With an electrode placed on his biceps and connected to the EMG TV during BF (Phase I for Patient 7), he was surprised to see some activity at all, and was, after approximately two weeks, able to increase his EMG activity from an initial

11.8 μv average of activity at baseline to an average of 71.2 and 49.0 μv of activity at weeks three and four of BF. Patient 7 reported feeling his response in conjunction with greater increases in EMG recruitment; hence, a cycle of informational, along with some sensory reafference cues, was effectively completing a loop upon which Patient 7 could build gains. During PT (Phase II for Patient 7), Patient 7 did not lose the amount of EMG activity he had accomplished in BF. Along with EMG increments, however, range increments for strict elbow flexion--i.e., the explicit, isolated use of the biceps and bracchioradialis muscles to flex the elbow--were not forthcoming. This is because Patient 7 was apractic. Throughout BF and PT, he did, however, use a greater total swing of an adducted shoulder of which elbow flexion was a component. EMG recruitment was "isometric" to the extent that the biceps would fire with or without a shoulder movement and also fail to terminate firing when elbow extension supervened. The reader is referred to this patient's case history for a number of elements which are either briefly or not at all reviewed here. From the foregoing, it is evident that Patient 7 had other problems which, if surmountable, needed other techniques of therapy to get ideal increased function. Yet BF served as a provocative entry modality into Patient 7's sensorimotor system and furthermore would conceivably have been a continuing invaluable tool to reach some acutely necessary modicum of a physiological substrate of EMG recruitment in any of the muscles of his flaccid limb.

Patient 10

Patient 10 had relatively good sensory function. However, his biceps (the target muscle) and other muscles were so spastic and hyper-

toned that his upper extremity is best described as rigidly set in a flexed elbow position of 45^0 with a pronated wrist and flexed fingers which, to the touch, had a very "hard" feel of hypertonicity. Patient 10 was 10 years poststroke and his condition appeared "permanent". Although he had good proprioception, the limb was practically "static" when he tried to move, in that there was very little motion around the elbow joint and hence a barely detectable amount of biceps relaxation around which to begin elbow extension. In the face of such a pragmatic problem, BF was an invaluable tool. An electrode on the biceps connected to the EMG TV, sufficed, in very few sessions, to cause decreased biceps activity and a range of motion of up to (e.g., Part A) 72.5^0 at test session four of BF vs. a mean of 8.6^0 at baseline. The biceps EMG activity never went to complete zero although adjustments were made at appropriate sensitivity levels whereby any progress would be readily apparent to Patient 10. The training time of four weeks on BF was hardly sufficient for full and facile control over biceps relaxation (for further details see the case history for this patient). Although Patient 10 still maintained good range of motion during PT, his latency to initiate elbow extension, as well as the actual time taken to achieve maximum elbow extension, which was decreasing during BF, began increasing during PT. In sum, for patient 10, BF was an excellent beginning for biceps relaxation and, most dramatically, for increased range. Patient 10 did not have voluntary triceps activity for elbow extension. The elbow fell to extension by gravity. Presumably, if BF input would ultimately have been sufficient for efficient control of biceps relaxation in achieving elbow extension by gravity, a total rehabilitation program of, e.g., eventual increased

triceps activity, or focus on other limb muscles, would have been indicated in order to achieve a significant functional change.

One might speculate as to whether BF would have been necessary to relax other muscles (e.g., the finger flexors) or to increase the activity of other muscles (e.g., the triceps). Patient 10's tightly flexed fingers were at times so decreased in tonicity during PT as to be non-resistive to passive stretch. Due to general limb relaxation, he also began to move the fingers somewhat during PT. Also, Patient 10 did have some triceps strength upon which he could have, in theory, been able to build more strength by exercise; but isometric use of the triceps during experimental PT sessions, where the biceps was not yet under control, caused immediate increased biceps activity. In sum, BF was a most efficacious input modality to deal with the major factor interfering with functional change in Patient 10's affected upper extremity--i.e., a severely spastic biceps muscle; however, were Patient 10 to overcome his lack of biceps relaxation control via BF, many other techniques--e.g., general limb relaxation and exercise--may have been eventually used in addition to BF as part of a total program of rehabilitation for ultimate return of function.

BF and the Eight Other Patients

For all other patients, E cannot say that BF was, in and of itself, a particularly efficacious modality for return of function. An unqualified statement that BF was more effective than PT for EMG recruitment is misleading on two counts. As explained in Statistical Results, PT trained general limb relaxation and inhibition of intense target muscle contraction in three of the four patients in Group 1 while BF specifically conditioned a greater amount of EMG recruitment in all

statistically analyzed patients. Furthermore, unqualified conditioning of EMG increments is not always the most necessary requirement for ultimate return of function. This is why PT often focused on inhibition of intense muscular contraction and, very importantly, as a corollary, inhibition of spasticity and clonus. It also helped inhibition of antagonist and synergist muscles, etc.

As reported earlier, nine of the ten patients were classified as learners. The one non-learner was Patient 5. Of the nine learners, two patients were shown to have benefitted by BF as an input modality. They were Patient 7 and Patient 10. There were six patients for whom PT seemed most effective. These were Patients 1, 2, 3, 4, 6, and 9. For the ninth patient, Patient 8, BF and PT were effective to the extent that verbal and instructional feedback was most important in the general training regimen. The reader is referred to the case histories for these patients.

The verbal guidance afforded patients during BF, in supplement to the BF modality itself, was an extremely important ingredient in BF training. Though the BF stimulus was rather parenthetical for many patients, BF training was helpful for all patients to the extent that continual verbal guidance during practice with the EMG TV for the elimination of undesirable response features or the reinforcement of correct response patterns, etc. effected general functional changes. Case histories reveal such functional changes as due to the rigorous verbal demands and continuous, immediate verbal appraisals (positive or negative feedback) given patients while they were using the BF machine.

In sum, BF training was not irrelevant but it was not as completely appropriate as it might have been when justifiably incorporated

into an entire system of therapy. Motor control (skill) is not gained by contracting (or relaxing) a muscle to full capacity. Hence, BF was not isolatedly, i.e., without verbal instructions of specifics, etc.--even for Patients 7 and 10--an automatic cause of therapeutic gains.

Johnson and Garton (1973) (see "Therapeutic Contributions", this paper) in their study of BF and neuromuscular control in stroke patients make the point that two important limitations to conventional PT are: "few therapists with the necessary training and motivation, and the great expense due to the enormous amount of time required in direct patient-therapist contact." Yet in reference to BF training, they conclude that "we did not hesitate to utilize any technique of facilitation or operant conditioning at our disposal. No attempt was made to eliminate the need for a sound working knowledge of facilitation and operant conditioning by the therapist. Indeed we suspect that the more proficient the therapist is in these areas and the greater the ability to pass along this information to the patients, the more successful will be the method," (pp.320-322).

The above is presented to reinforce the observations made by the present author. EMG therapy is not a matter of a patient virtually on his own with a machine which will effect a change. Probing, intelligent therapist care is the most vital element for maximizing the value of the EMG machine. In other words, the machine is a valuable instrument when used rationally and skillfully for selected patient cases under proficient therapist guidance. Irrational or unskillful use of BF is not valuable.

Conventionally, one role of the therapist is to instruct in motion, use non-voluntary methods of motion assistance (not employed

in the current study's PT regimen), and enhance the importance of sensation as related to learning. This role can be aided by BF. Another therapist role, however, is to direct attention to important particulars and provide feedback as to the appropriateness of a response. Finally, except in the relatively rare cases of indefatigable intraindividual motivation, of greatest importance is the foundation of patient trust and belief that therapist response is as honest and enterprising as (a) the patient condition, (b) methodology and, (c) technology permits. BF as a modality may be <u>required</u> as in patient problems such as those of Patient 7 and Patient 10; but an enterprising therapist, (besides patient motivation which a good therapist will elicit), is the superordinate constituent for functional change in a therapeutic situation. BF as an input modality is encouraged where it might be (a) an incentive, (b) a modality adding substance to a total therapeutic situation, or (c) the pragmatically most useful modality at a particular therapeutic junction.

Teuber (1968) states,

> Paralysis is not merely characterized by apparent absence of certain complex motor sequences, but often by changes in the stimuli adequate to provoke a movement. Whether we study altered motor performance or perception, our problem is not properly characterized by saying that we are searching directly for changes in response: what is needed is discovery of the stimulus. (p. 275)

BF, as this study has shown, can be a valuable stimulus for particular patient problems. It may at times be helpful to see one's EMG activity increase or decrease. In sum, BF is additional information which can be reasonably utilized when it makes meaningful sense to a patient's mental or subjective corollary sensory experience, or when it is one of the few possible input modalities which can successfully "make entrance" into a patient's sensory experience to effect eventual self-regulation of motor control.

Concentration in PT and BF

Both PT and BF require attention to input stimuli. In PT the stimuli are proximal, palpable sensations which directly stimulate an organism at the physical boundaries of his skin. In BF, the stimuli are distal, observed sensations which indirectly stimulate an organism through apprehension by the intellect. Thus, in PT the quality of sensation is imparted through touch and proprioception, while in BF it is imparted through cognition. In PT the literal initial input modalities--the "givens"--are inherently medical; in BF the literal initial input modalities--the "givens"--are inherently psychological. Above and beyond the input junction, however, there is a mutual interchange between the medical and psychological modes of operation in either PT or BF.

Both PT and BF training require, beyond stimulus input, the perception and attention to the stimuli comprising their training input modalities. Both also require the interpretation of stimuli into some type of subjective experience within which a S can effect an alteration of his behavior. Regardless of the different modes via which stimuli are initially registered in PT and BF, both require the conscious exertion of mental effort directed towards stimulus input and output.

In this study great emphasis was placed on mental effort for the attainment of desired outcomes. Within whatever perceptual limitations patients were acting, a most essential feature of both PT and BF was the direction of concentration to the feel and comprehension of stimulus events.

Retentivity in BF and PT

Input registration in PT is direct in the sense of being identical to stimuli through which corporeal sensory awareness or the result of motion is felt. Yet as PT had many stimulus events simultaneously

or successively highlighted within the time course of a single phase, conditioning of the specific isolated target response was occurring in many ways other than exclusive EMG recruitment. The total therapeutic matrix was effecting functional alterations at intricately woven merging levels of perceptual experience by continually building upon a given level of physically interpretable experience of the limb as part of the body schema. Such experience culminates in a total pattern of learned movement.

Input registration in BF is indirect in the sense of being removed, a priori, (in formal, unalloyed essence) as non-felt, cogitated information. But the conditioning of the target response by the BF stimulus alone (without E's verbal instructions to modify other aspects of a response) was effecting limited functional alteration by specific, intense, and direct conditioning of increased or decreased EMG activity. This conditioning may or may not have been incorporated into more than a time-locked training consumption of information. If the BF stimulus was utilized in a manner abstracted from corporeal experience, an experience which a patient did not carry away with him as engrammatically more than abstract information--information of which he was unaware in terms of long term consolidation to effect a response except via BF--then the information will not have been utilized in terms of transformation to some type of physical corollary discharge counterpart of a subjective sensory experience of movement and the part moving.

The latter is important because a patient is not forced to use an hemiparetic limb in his daily living and, he is not apt to use it except as he has internal sensory experience of it. The possibility of

limited retentivity of the ability to increase or decrease EMG activity outside of the BF training situation is important if the recruitment increase or decrease which is learned is necessary for the use of a limb (i.e., limb function). Other cues of course, inevitably supplement the BF experience so that they may operate to reinforce and sustain skills learned in BF. Supplementary cues are not only kinesthetic. They are also e.g., vision of a limb moving, or, e.g., the success of a movement to achieve some end purpose, etc.

The possibility of non-retentivity of essential BF skills is important because, for example, while Patient 7, who had severely impaired sensory function, maintained his EMG recruitment ability in PT training (Phase II for Patient 7), Patient 4, who had relatively good sensory function, had no sensory appreciation (subjective experience) of EMG increases which amounted to 100% increments over baseline in BF (Phase II for Patient 4). As is reported in Case Histories, ultimate storage (vs. performance) of EMG activity increments on BF was evaluated as likely minimal for Patient 4. (For specific details leading to this particular patient evaluation, the reader is referred to Patient 4's case history.)

Thus, there is the question of the retentivity of the specific ability to recruit EMG activity as well as the importance of recruiting high levels of EMG activity for muscle use as learned via BF, if there is no transformation to cues somehow proprioceptive to a patient--cues which a patient can call upon, consciously or unconsciously, without the aid of EMG BF. Whether the BF knowledge, which is intellectually appreciated, can be ultimately inwardly physically appreciated, is apt to vary from patient to patient. A long term follow-up of patients

who are trained on BF EMG recruitment with therapist aid and verbal instructions of response specifics, etc. might be able to help determine if most patients would be well able to ultimately "store" the BF experience in terms of the spontaneous recruitment of EMG increases or decreases as indigenous to motor output.

The features of a response besides EMG recruitment which were modified during BF in this study by repetitious instructions from E, were modified by stimuli common to any type of physical therapeutic situation--i.e., concentration and subjective physical experience to effect a qualitative change in a limb movement. Therapy for the body must ultimately be related to the body by a patient. The experience or feeling of physical movement is attained by concentration; and by concentration patients can either translate cognitive events to sensory experience as, e.g., "corollary discharge", or transfer concentration from cognitive events to some perceived reinforcing concomitant sensory events.

Reflections on the Current Study

The patients in the current study did not have much else to do besides come to therapy. Except for Patient 3, they either lived in nursing homes or whiled away their days at home. There was also no financial burden on patients as the experiment was a funded project. Two of the criteria for inclusion in the study were some motivation and some, even minimal, voluntary movement. Also, though most patients had sustained massive brain damage, all had relatively good comprehension skills, and all were at least one year poststroke. Thus, the patients represented a sample of available, motivated, and cooperative

individuals. None were completely paralyzed in the extremity of concern. Most patients were between 50 and 64 years of age.

The patients in the current sample did have massive brain damage; also although all were at least one year poststroke, (six months is the generally accepted period for spontaneous maximum recovery), some were several years poststroke (e.g., Patient 1 six years poststroke, Patient 10 ten years poststroke, and Patient 3 twelve years poststroke). Learning occurred in nine of the ten patients studied. Reference to the above patient case histories as well as other patient case histories indicated a fair amount of learning given the relatively short time course of training. As Basmajian et al. (1975) have noted, the potential for learning in longstanding stroke conditions has perhaps not been fully recognized.

The target response for all ten patients was not a functionally relevant response. That is, patients who were required to flex or extend the elbow or, extend the wrist, did not have finger control, and hence, the response being trained was "useless" for practical purposes in their daily routines. At the same time, however, if patients were motivated to practice the target response in spite of its inutility, those patients who were receiving PT training practiced it together with many of the other responses which were being trained at

that time. (See Table 4 for amount of patient practice.) Oftentimes it was the "other responses" (i.e., shoulder flexion in particular for the wrist extenders,or general limb relaxation for everyone who received it) which were practiced more, as they were understandably more relevant to the patient conceptualization of what functional (vs. experimental) goals should be.

During PT, patients were involved in target muscle function as part of a total program aiming for skilled movement. It is dubious whether attempts at home in the initial weeks of training were practiced in the proscribed experimental manner, since in the initial weeks of training, patients most definitely needed continual repetition and reminders of response specifics, especially in the beginning of a session's performance. Yet, by the end of a PT or BF phase, patients did generally have more understanding of those specifics.

Patients who received BF in Phase I of training had only the target response to practice, if they wished. They did not receive further training until PT ensued in Phase II. Patients who received PT in Phase I had the aggregate of movements and movement specifics which were being trained in PT to practice during both Phases I and II. The manner in which patients practiced at home cannot be known. As a result, any assessment of the effectiveness of practice would be difficult to establish.

Finally, as previously mentioned, careful consideration of case histories gives the most fruitful analysis of training specifics as well as the most accurate representation of results. The case histories are also valuable indices to the evolution of motor control in the pa-

tient population studied. Concerning case histories, E was more aware of looking for functional changes in Phase II vs. Phase I.

Patient Behaviors

Some general observations of specific interest or import are culled from case histories and presented here. However, the reader is referred to patient case histories for the full story.

Patient 5 is classified as a non-learner. He learned no EMG recruitment and a very few degrees of range. More importantly, although he was able to produce an ample amount of range at either the wrist, elbow, or shoulder joints in his initial few attempts of a session's performance, he could never reproduce the responses (he would fatigue) after the initial attempts. This was an unchanging pattern over eight weeks of experimental training. Furthermore, even though range was quite ample on the first few attempts, averaged EMG always remained the same. Patient 5 also had intact cognitive function, and practically completely intact sensory function. The neurophysiological conjecture for this patient's condition might be an overriding excessive inhibition at synaptic junctions of efferent pathways to the upper extremity. Neither PT nor BF training was successful in interrupting this condition.

Another point of behavioral interest is the difference in subjective sensory experience of motor response (seemingly irrespective of patient sensory function) between patients who were initially at low levels of averaged EMG output vs. patients who were initially at relatively high levels of averaged EMG output. Patients were carefully questioned at times as to whether they felt EMG increments that E objectively could observe via her instrumentation. The initially low

level responders usually reported definite sensory experiences of increased EMG recruitment. On the contrary, two patients at relatively high levels of averaged EMG activity (Patients 2 and 4) could not experience increments of activity of up to 50 μv (e.g., going from 80 μv to 130 μv for Patient 2) or again of up to 60 μv (e.g., going from 60 μv to 120 μv for Patient 4). In fact, although these patients had confidence in E, during BF training, both of the latter patients questioned the purpose or relevance of raising their wrists to make the EMG TV dot go high. On BF, they performed to their fullest capacities but under either BF or PT, when carefully questioned for subjective experiences of any difference in "feeling", as E observed increments in their averaged EMG activity, they replied that they had no awareness of any different feelings and asked whether they should feel something--to which E quickly replied, "No, just asking". These patients had, moreover, relatively good sensory function, and both reacted to PT as a more cogent training experience than BF.

Sheer EMG recruitment or cerebral knowledge of efferent activity was particularly not suitable for such patient types nor presumably for others like them in the general stroke patient population. Patient 7 and Patient 1, e.g., on the other hand, are outstanding examples of initially low level EMG recruiters who had definite sensory awareness of EMG increments in either PT or BF (see the case histories for these patients). In addition, Patient 7, was assessed as having overall very poor sensory function. On BF, the sight, and ultimately the feel, through sensation, of EMG activity were very motivating stimuli for Patient 7.

A final point of interest was the mesmerizing quality of the EMG TV stimulus for the patients in general. E would have to tip-toe in when approaching a patient practicing on the BF TV, for some patients would be startled by E's intrusion. Several patients seemed to prefer to disregard E's presence even when they heard her come in, because they were so absorbed and interested in maneuvers with the TV. They would patiently listen to E's instructions to movement specifics and proceed to get "right back to work". Patient 8 commented that she liked BF because she could "see what she was doing". The occasion to really "see" muscle activity and make it conform to cognitive demands was apparently satisfying to most patients (save Patients 4 and 2--although they too were satisfied to the extent that they were making the TV dot do what E said they should make it do).

Considerations and Comparisons Between the Current and Related Studies

Other researchers in the area of EMG BF with stroke patient populations have generally reported using methods fundamental to PT and functional experience in addition to EMG feedback to effect the practical utilization of a limb. They also generally have used some qualitative functional criteria to measure experimental outcome (see pp. 81-88). The means by which function may be evaluated were not, except for range, the parameters used in this study. Qualitative assessments of patients' skill were explicitly eliminated, although the latter, it is believed, are necessary (though difficult to regulate) to assess function. Strength, e.g., which might seem a good measure of function, was considered to pose too many problems for this study (see pp. 88-90). At the same time, it must be emphasized that case histories in this study are extremely valuable reports of function and functional progress.

This study was conceptualized and carried out in terms of a learning framework (again see pp. 88-90) focusing on one target muscle and movement. It was primarily interested in a controlled assessment of EMG BF within a learning paradigm and, of necessity, incorporated PT as a control. Thus, aside from range, the major dependent variable deemed most suitable for quantitative assessment was averaged EMG activity. As recurrently mentioned throughout the paper, the latter parameter was specifically sensitive to the methods and outcome of BF, as opposed to PT, training.

By consideration of the noted facts, and directed careful analytical attention, the current study provided an exploration and some clarification of EMG BF and neuromuscular learning in stroke patients. At the same time, it opened a forum for observation and discussion of the control training--PT--and learning, especially in terms of the evolution of motor control as function. Within the constraints explicitly outlined and examined throughout this paper, the study delineated at least some features of the clinical role and practice of EMG BF. It also arrived at the conclusion that BF is not a therapy but that it is, rather, a supplementary stimulus modality of therapy which can be used within a total therapeutic situation (see pp. 373-374 and pp. 377-380).

It was evaluated that beyond some necessary amount of EMG activity for muscle use, there would seem to be a point of diminishing return for sheer EMG recruitment vs. function in stroke-damaged individuals. Conditioning EMG activity is not the equivalent of conditioning motor control. The goal in BF training is EMG increase or decrease. The goal in PT is not specifically EMG increase or decrease; it is motor control (use). As a supplementary modality, proficient

use of the EMG machine in clinical settings would seem to be definitely helpful for certain types of patient problems. The patient problems in this study for which this was the case were those of Patient 7 and Patient 10, each of whom had individually characteristic reasons for such implementation (see "Patients for Whom BF was a Valuable Training Modality"). The problem types for which EMG BF may be critically useful in the general stroke population, are not known. Basmajian et al. (1975), in fact, have called for research on just this issue.

Finally, from the current study at least, it would seem that physical therapy (PT) is involved in neuromuscular therapeutic training whether such training does or does not incorporate a BF stimulus input modality. (See pp. 118-123 and pp. 367-374 for a discussion of the nature and methods of PT.) In this study, as the case histories indicate, the skilled aspects of function were arrived at via methods conventionally subsumed under methodology indigenous to PT.

As well as can be discerned from methods of other EMG BF research with stroke patient populations, response specificity (see Method, this paper) may not have been trained in other studies in the same manner or extent as in the current study. This study, in any event, was extremely specific in training subordinate components of a motion, or eliminating components of a motion interfering with precise movement control, so that the preliminary basics of motor control were attempted first. In general, the procedure was to make subordinate components of a motion available and useful, and then to integrate these into a behavior which would represent the optimum function possible. Response specificity (skill) requirements may lead to better ultimate control (one does not know), but in any event, they would seem to require a much

longer time course than that pursued in the current study. As both BF and PT required response specificity, BF training, again, was more a matter of concentrated therapy, as opposed to a machine and muscle pairing, which required continual verbal and, on occasion, visual, therapist guidance.

Also, via the method used in this study, range had ample opportunity to benefit in BF, particularly by virtue of the continual instructions which told patients to raise the wrist as high as they could and let it go until the TV display showed zero. Patients had to do this and nothing else intensely for 30 minutes. In PT, the intensity of attention given to the target muscle and joint was, in conformity with the method, much less, as the procedure was that of directed attention to the logically necessary components of training, in order eventually to attain skilled movement. At the same time, PT involved a great deal of relaxation training for some patients which automatically decreased EMG activity, but increased general function, as well as range, in the target muscle as part of the whole of functional improvement. As previously mentioned, ultimate maximal EMG activity may be antithetical to function, and the isolated pairing of a machine with a massively problematic patient condition can even impede function, as case histories (e.g., Patient 9 and Patient 4) reveal. Were PT and BF to be somewhat more equivalently compared in the current study, the above qualifications would have to be taken into account. Because of the many qualifications, they are not directly comparable.

As it is impossible to directly compare PT and BF within this very study, it is even more difficult to directly compare this study

against those of, e.g., Johnson and Garton (1973), Basmajian et al. (1975), and Brudney et al. (1974) (reported in full on pp. 81-87). This is because methods, goals (e.g., the extent of isolated movement training), and parameters, as well as the numbers and types of patients from whom conclusions are drawn, are confounded. For example, both BF and PT can differ as procedures from study to study; also, functional assessments were used as major outcome criteria in the above studies and there were differences among assessment protocols; functional assessments in the current study represent the very body of the entire investigation. Again, even for the one similar quantitative parameter, range of motion, which was used in both this study and that of Basmajian et al. (1975), the current study used a mean range value within a test session as a patient's score, while Basmajian, et al. used the highest range value attained within a test session as a patient's score. Finally, the n was relatively small in this study. However, although the n was small, the array of patients did include a good sample of stroke patient problem types.

The current study seems to draw somewhat different conclusions than the above studies which, by and large, concluded that very good results were obtained with EMG BF training. One difference is perhaps different and larger populations. Yet a critical difference would seem to lie in the current author's conceptualization of therapy, physical therapy, and biofeedback. The latter was conceptualized within the general context of the multiple factors assumed necessary to a therapeutic situation which deals with neuromuscular education for victims of stroke. From such a point of view, the conclusion is that therapy which is directed towards a change in motor activity is

mediated by the following factors:

(a) motivating a patient and keeping his attention focused and sustained;

(b) manipulating the therapeutic situation to conduce the proper instructive circumstances;

(c) teaching a patient to be aware of target body parts;

(d) conditioning a patient to respond to evidences of visual, auditory or haptic effects of motion;

(e) manipulating body parts either directly by E or by the patient himself through E's instructions; and so on.

All of the above factors are understood to fall under the rubric designated as PT (physical therapy). Within this framework, rather than a therapy, EMG BF is seen as a new, provocative, and valuable stimulus modality which can be utilized within a therapeutic program for certain patient types and specific patient problems.

EMG BF and Psychology

There is then the question of the amount of long term storage of EMG increment or reduction arrived at via BF without, eventually, the sustenance of the BF information stimuli. Regardless of the former, the paradigm of a BF teleostimulus, and a responsive patient processor is of intrinsic interest to some branches of psychology. The current study is seen to be an addition to the body of literature (some, e.g., in the introduction to this paper) which might cull from its results a psychological perspective of the living organism as a self-responsible agent. EMG BF is conscious self-conditioning of

unconscious neurophysiologic activity and certain tenets of relevance to psychology might be derived from such results.

In the current study, the patient had before him meaningless symbolic stimuli of a dot moving on the EMG TV and the sounds of the EMG acoustics. These stimuli had no intrinsic informational meaning except as imputed to them by the patient, initially through E's directions. The BF situation required that the patient be motivated--i.e., that he want to do something with the dot and the sound. Given the necessary motivation, the patient then had to impute informational properties to the visual and auditory stimuli in order to utilize--i.e., consume--the information the stimuli "conveyed". The informational feedback was, furthermore, feedback on neurophysiological efferent activity of which the patient had no direct, literal awareness except through the stimuli to which he could give meaning and utilize. Given these conditions, most patients were able to increase their neurophysiological EMG activity.

Thus, patients were self-programming themselves. The stimuli could not passively impress or infiltrate an organism who, in turn, reacted in some conforming way. The stimuli could only be effective if given meaning and controlled by the organism as an information processor and self-controller. As mentioned, the neurophysiological innervation being controlled was not accessible to a patient except via the external stimuli. Thus the patient was controlling both his external stimulus environment and his internal physiological environment. A good question which arises here is whether the patient really did control his physiology in as literal and direct a sense as he controlled the informational stimulus. In any event, to the extent that patients could alter efferent activity via feedback information,

they were doing so by making cognitive sense of varying gradations of exteroceptive cues; it was the patients and not the stimuli which were producing a change.

Also, the cues traditionally deemed essential for motor output are most commonly thought of as kinesthetic, or the naturally available sights and sounds of movement along with which a person has concomitant proprioceptive awareness. In EMG BF, customary input modalities are not operative. The sights and sounds are related only as symbolically germane to ongoing activity S presumes he is altering. Hence efferent activity is controlled by perceptions which are not the common modalities of control and the efferent activity is not apparent to S except through the machine he uses.

There is one additional feature. The patient has had relevant parts of his motor neurophysiologic apparatus destroyed. It has been lesioned and pathways are interrupted. He is, in spite of this equipment deficit, manipulating his motor efference in terms of EMG increase or decrease. In effect, his body has suffered a blow, and he is at a level of assumed stability in terms of spontaneous return of motor function; efference is organically depressed. A patient, however, concentrates and somehow increases efference, more or less a positive psychosomia.

The same alteration of efferent activity, of course, occurs in PT. The fact that learning and self-control is occurring in PT is, somehow, not as dramatic at face value as changes occurring in BF, yet it is perhaps even more dramatic in results than EMG increases or decreases. In PT, unfortunately, furthermore, it is somewhat too facile to conceptualize learning within a paradigm of input-output, or

sensory stimulus→motor response, bypassing, so to speak, the man behind the change. The BF paradigm just makes the matter of self-conditioning, via the use of a non-felt teleostimulus, a more blatant example of internal control over exteroceptive and interoceptive events.

In effect, EMG BF demands a view of the organism which is, at minimum, different from, e.g., mechanistic postulations of S-R theory relating to how an organism might be accommodated or regarded. As science or theory changes with the times, and as every science carries with it, advertently or inadvertently, an attitude or set of beliefs, the latter attitudes or beliefs, perspectives, etc. are susceptible to contemplation along with experimental results. The speculation that how psychology views an organism will ultimately determine its goals and application, might also be considered.

Within such a frame of reference, Skinner's (1966) statement that "the consequences of an action change an organism regardless of how or why they follow" (p. 14), is tenable yet incomplete. (The following is a suggestion as to why this is so, yet also does not presume to be complete.) In a hungry rat and lever press situation, e.g., it is true that a pellet delivered for a lever press is an external stimulus which can cause, in the Humean sense, an increase in lever pressing behavior. Yet more fundamental to an explanation of the rat's behavior is the fact that pellet delivery has an impact on lever pressing precisely because the rat is food deprived (i.e., hungry). Most fundamentally, the rat presses a lever in order to get food, a reversion to what has been termed "teleological" explanation.[50] The EMG BF

---

[50]The current author does not espouse teleology as an explanatory system (especially in terms of its generic characterization), but merely refers to it for purposes of briefly illustrating the above comments.

paradigm incorporates an organism using stimuli in order to achieve some goal. The auditory and visual experimental stimuli, furthermore, cannot have consequences on behavior unless an organism chooses for them to have consequences. Strict formulations of external manipulations and internal reactions do not suffice to explain the self-conditioning process. It is the patient who is adapting, molding, and modifying his response, and hence, it is the patient who is the producer, with help from E, of change.

Also, man can control his body, within, of course, the limitations of his physical status, and a man can control himself. By the same token, a man can be controlled.

The most parsimonious (see following paragraph) and lawful explanation of all such behavior is mind. Physiology is not sufficient to account for it, nor is neurology, physics, chemistry, or genetics, etc. The "leftover" (for psychology) is behavior--manifest as other than a physiological, neurological, physical, chemical, or genetic event.[51]

Parsimony, as above, was used purporting an exaggerated inclination. It is believed, in fact that, in some instances, parsimony as a principle has led to a frugality of behavioral explanation which has failed to appreciate the most logically consistent alternatives to the understanding of behavioral events. In terms of explanation in the sense of something known or arrived at, yet not seen (i.e., inference), and circumstantial evidence notwithstanding, the current author supports a postulation that if one admits a psychology, one takes into account a mind.

---

[51]For a somewhat related discussion, the reader is referred to the Preface of this paper.

For example, an overview of the entire paper, as well as the specific research reported in it, are felt to indicate that motor control, as the output side of an organism, is extremely complex. It depends to a great extent, on both properties of input stimuli as well as the ascription of property to stimuli input, leading to a determination of stimulus value, or percept, by the act of perception. The latter results in consumption or neglect of a stimulus depending on its relevance in terms of practical utility as related to the consequences of a desired goal. Such observable outward events might all be conceived as, e.g., falling within a nomenclature of voluntary behavior. (Some possible meanings of the latter phraseology were attempted in the first part of this paper.) The primary feature of voluntary behavior is the inner agency of the human being, or mind.

Many sciences, or branches of a particular scientific genre, attempt to explain the whole of human behavior from some aspects or perspectives on the total organism, with the result of precisely more knowledge on those aspects from a particular perspective. The view here is that no observable stimulus, receptor modality, or brain area, etc. can hold a completely adequate description of the whole person. Also, psychology is the discipline which can provide observation and <u>understanding</u> of human behavior (also more readily perhaps than explanation, especially if the latter is patterned after, e.g., cause and effect `a la Newtonian science, as such explanation does not seem a feasible goal).

As more is known, e.g., about the body or, e.g., about the observable physical properties of stimuli or responses, the mind can

appear (or appears)[52] increasingly elusive and enigmatic and, at the same time, a stronger and more likely supposition (or fact[53]).[52] The important point would seem to be to deny simple characterizations and conclusions about behavior, the "rule" instead being that of increasing complexity. Questions which may present themselves from such a point of view are (a) whether all observable behavioral events are to be ordered in one or a similar way, and (b) whether such order itself, as is discernable, increases in complexity.

---

[52]Depending on one's position and conceptualization within a given frame of reference.

[53]As in an actual event.

APPENDIX

Audiometry Examination--carried out by Mr. Richard Hole, University of Minnesota Hospitals.

The audiometry examination was primarily carried out to ascertain that patients could process the gradations of EMG feedback acoustics employed in the current study, and, secondarily, to ascertain patients' ability to make discriminations of speech shaped noise at the sensori-neural level. Each patient was given a routine audiologic evaluation. Three patients had hearing within normal limits. Seven patients exhibited moderate high frequency sensori-neural hearing losses, the nature and extent of which would have no effect on their perception of the EMG sound.

The amplified sound produced by the muscle contraction is complex in nature. The sound changes in tonal quality and in intensity as the strength of the contraction increases or decreases. To assess the patients' ability to perceive relatively fine intensity and tonal quality differences, each patient was asked to make same or different judgments of paired stimuli--consisting of electronically produced complex signals. The stimuli were presented to the patients via a loudspeaker with the patient seated in an acoustically treated test booth. The stimuli consisted of white noise vs. speech shaped noise and narrow bands of noise with center frequencies as follows: 900 Hz vs. 1000 Hz and 1750 Hz vs. 2000 Hz. The presentation level was 50 dB relative to audiometric zero (HL). (One patient, Patient 7, whose hearing dropped at 2000 Hz listened to narrow band noise of 1350 vs. 1500 Hz.)

Following the pitch judgment task, each subject listened to paired stimuli to judge same or different for loudness. Again complex signals were used, specifically a narrow band noise with a center frequency of 1000 Hz and a broad band noise (white noise). The fixed level was 50 dB HL and the probe signal was randomly presented at 2 dB intervals above the 50 dB level.

All subjects were able to make same or different judgments for pitch with 100% accuracy. The judgment for loudness produced greater variability across subjects. Five subjects were able to respond correctly 100% of the time when the intensity difference was 4 dB or more, two were able to do so when the difference was 6 dB or more and three required a difference of 8 dB or more. This was all well within judgment capabilities necessary to discriminate differences in the EMG sound as well as speech shaped noise.

GLOSSARY[54]

Abduction - the withdrawal of a part from the axis of the body (Dorland's, 1965).

Adduction - the act of drawing toward a center or toward a median line (Dorland's, 1965).

Adducted opposed - pertaining to the thumb, the act of drawing the thumb toward the median ray of the hand with simultaneous rotation of the thumb to face the other digits.

Agonist - the muscle under consideration, most often the prime mover of the joint (Dorland's, 1965).

A muscle opposed in action by another muscle, called the antagonist.

Anomia - loss of the power of naming objects or of recognizing and recalling their names (Dorland's, 1965).

Antagonist - a muscle which acts in opposition to the action of the agonist.

Aphasia - defect or loss of the power of expression by speech, writing, or signs, or of comprehending spoken or written language, due to injury or disease of the brain centers (Dorland's, 1965).

Apraxia - inability to carry out purposeful movements in contra-distinction to paralysis or other motor or sensory impairment, especially inability to carry out purposeful sequences of movement or to make proper use of an object.

---

[54]Many definitions or aspects of them are taken from Dorland's Illustrated Medical Dictionary (24th ed.). Philadelphia: W.B. Saunders Co., 1965. Such definitions are suffixed by (Dorland's, 1965). Other quoted definitions or aspects of them are appropriately referenced.

Arm - the part of the upper extremity between the shoulder and the elbow as distinguished from the forearm (Dorland's, 1965).

Arteriosclerotic heart disease - loss of elasticity, thickening and hardening of the coronary arteries (Dorland's, 1965).

Cerebrovascular accident - injury to the blood vessels of the cerebrum, or brain, from hemorrhage, thrombosis, embolism, etc. (Dorland's, 1965).

Clonus - spasm in which contraction and relaxation alternate in rapid succession.

Cogwheeling - motion manifesting itself in a series of little jerks when the muscle is passively stretched or actively moved.

Contracture - a condition of fixed high resistance to passive stretch of a muscle, resulting from fibrosis of the tissues supporting the muscles or the joints, or from disorders of the muscle fibers (Dorland's, 1965).

Deep tendon reflex (DTR) - rapid contraction of a muscle in response to rapid stretch of the tendon, as in being struck with a reflex hammer.

Distal interphalangeal joint (DIP Joint) - the joint between the second and third bones of each of the fingers.

Dysarthria - imperfect articulation in speech (Dorland's, 1965). Impaired production of phonemes or combinations of phonemes due to inadequate neuromuscular control.

Dysgraphia - Inability to write properly. Associated with organic disturbance of the central nervous system.

Dystonia - Excessive tone which is the result of uninhibited activity of anterior horn cells; manifest by abnormality of body posture and altered limb position due to excitation from supra-segmental mechanisms.

Embolism - the sudden blocking of an artery or vein by a clot or obstruction which has been brought to it's place by the blood current (Dorland's, 1965).

Epicondyle - an eminence upon a bone, above a rounded projection on the bone (Dorland's, 1965).

Extension - [in most cases] a movement which brings the members of a limb into or toward a straight condition (Dorland's, 1965).

Extensor - any muscle that extends a joint (Dorland's, 1965).

Facilitation - a maneuver to activate a muscle. The patient has attention directed to increase muscle tone or to change the angular position of a joint. Reinforcement is given for successful achievements. Techniques used were skin friction, muscle percussion, and rapid passive stretch of a muscle, with immediate command to activate the muscle just stretched. Also, passively placing the limb in a position requiring activation of the target muscle for maintenance with a command to "hold" the position, "don't let it fall", etc.

Flaccid Hemiparesis - hemiparesis with looseness of muscles.

Flaccidity - lack of muscle tone.

Flail - extreme looseness; total lack of muscle tone.

Flexion - the act of bending or condition of being bent (Dorland's, 1965).

Flexor - any muscle that flexes a joint (Dorland's, 1965).

Forearm - the part of the upper extremity of the body between the elbow and the wrist (Dorland's, 1965).

Glenohumeral joint - the shoulder joint.

Hemiparesis - a lesser degree of hemiplegia; see hemiplegia.

Hemiplegia - impairment of motion or impairment of ability to carry out specific voluntary activities on one side of the body. The impairment gives rise to abnormal muscle tone which may vary from excessive weakness to excessive tone.

Hemiplegia, contralateral - as in this study, the hemiplegia is on the side of the body opposite to the side of the brain lesion causing the dysfunction.

Infarct - an area of coagulation necrosis in a tissue due to local anemia resulting from obstruction of circulation to the area (Dorland's, 1965).

Infarction - the formation of an infarct (Dorland's, 1965).

Infarction, cerebral - an ischemic condition of the brain, producing a persistent focal neurological deficit in the area of distribution of one of the major cerebral arteries (Dorland's, 1965).

Infarction, myocardial - the formation of an infarct in the myocardium, as a result of interruption of the blood supply to the area, as in coronary thrombosis (Dorland's, 1965).

Inhibition, physiological - prevention of firing of presynaptic terminals of anterior horn cells or prevention of depolarization of anterior horn cells by the activity of specialized synapses at the axon hillock.

Inhibition, the technique of - a command to the patient to diminish the tone of a muscle by using such words as "relax", "make it softer", "leave it loose", etc. An attempt was always made to direct attention to the sensation of relaxation - for example, "Do you feel it looser?" "Do you feel it softer?" E also passively moved the part and demonstrated the difference in tone when a muscle felt loose. There was a constant comparison of the difference between

loose and tight. Also used during stereotyped movements to eliminate undesired components of movement, i.e., to aim for a single direction joint movement.

Interphalangeal joint (IP) - by convention, the distal thumb joint.

Ischemia - deficiency of blood-flow in a part, due to functional constriction or actual obstruction of a blood vessel (Dorland's, 1965).

Kinesthesis - sensation of vibration, position, or motion. Muscle-tendon-joint sense (Woodburne [1967], p. 358).

Limb - an arm or a leg with all its component parts (Dorland's, 1965).

Loftstrand cane - single ended cane with a handle with a grip at right angles to the stem and a metal cuff for retention around the forearm or upper arm joint above the elbow.

Metacarpophalangeal joint (MCP joint) - the joint at the part of the hand between the hand and the fingers.

Monoparesis - clinical assessment of deficiency in movement control or muscle tone confined to one extremity.

Movement to general command - in this study movement pattern of the paretic upper extremity in response to a non-specific command to for example "Move your arm as much as you can", "high as you can", etc.

Movement to specific command - in this study, movement pattern of the paretic upper extremity in response to a specific command--e.g., "bend your elbow", "raise your wrist", etc.

Occlusion - the act of closure or state of being closed. Obstruction to the flow of blood through a blood vessel as the result of spasm of the vessel or the presence of a thrombus [or embolus] (Dorland's, 1965).

Pronation - applied to the hand, the act of turning the palm backward (posteriorly) or downward, performed by medial rotation of the forearm (Dorland's, 1965).

Proprioception - sensation from muscles, tendons, joints and semicircular canals, utricle and saccule (Woodburne [1967], p. 360). Relating to the position or motion of the head or other segments of the body.

Proximal interphalangeal joint (PIP joint) - the joint between the first and second bones of each of the fingers.

Quadriparesis - impairment of motion of all four extremities.

Range of motion (ROM) - normal angular excursion possible in a joint.

Reflex - innate response to a particular stimulus (Woodburne [1967], p. 360).

Relaxation - lessening of muscle tension by decreased contraction or, isometrically, by decreased muscle tone.

Relaxation training - "Relaxation of muscle fibers, i.e. complete absence of all contraction, . . . seen as the direct physiological opposite of tension . . .", achieved by "systematically tensing and releasing various muscle groups and by learning to attend to and discriminate the resulting sensations of tension and relaxation . . . " (Bernstein and Borkovec, 1974, p. 3) (after Jacobson, 1938).

Retraction - the act of drawing back (Dorland's, 1965).

Rigidity - stiffness or inflexibility (Dorland's, 1965).

Rigidity, hemiparetic - rigidity of the paretic limbs in hemiparesis (Dorland's, 1965).

Spastic hemiparesis - hemiparesis with excessive motor tone characterized by deep tendon reflexes.

Spasticity - a condition of increased muscle tone characterized by hyperactive deep tendon reflexes.

Splay - a forceful wide spreading of the fingers.

Spontaneous movement - in this study, typical movement pattern of the paretic upper extremity during non-specific general patient activity.

Stereotyped movement patterns - simultaneous or nearly simultaneous contraction of several muscles in a limb to effect a uniform complex movement in response to a command for any single component of the entire movement. Most patients have a similar complex movement pattern with individual minor variations among them. Other terms used are associated or synergic patterns, and hemiplegic stereotyped movement pattern which refers to a pattern which includes abduction and internal rotation of the shoulder, flexion of the elbow, flexion at the wrist, and flexion of the fingers as a result of general somatic activity, or in an attempt to flex only the elbow.

Subluxation - an incomplete or partial dislocation of a joint (Dorland's, 1965).

Supination - applied to the hand, the act of turning the palm forward (anteriorly) or upward, performed by lateral rotation of the forearm (Dorland's, 1965).

Tenodesis - elicitation of a motion in a limb or portion of it, secondary to a relative shortness of the muscle or tendon fixed to the part.

Thrombosis - the formation, development or presence of a plug or clot in a blood vessel or in one of the cavities of the heart, formed by coagulation of the blood, and remaining at the point of it's formation (Dorland's, 1965).

Tone - ease with which a joint can be moved or a muscle can be stretched; measure of spontaneous muscle activity.

Voluntary movement - movement under organismic control; the obverse of reflex; characterized by the ability to respond to a command either by activating or terminating a motion at will.

Watershed - an area supplied by blood vessels coming from more than one direction.

References

Adams, J. A. A closed-loop theory of motor learning. Journal of Motor Behavior, 1971, 3, 11-149.

Adams, J. A. and Goetz, E. T. Feedback and practice as variables in error detection and correction. Journal of Motor Behavior. 1973, 5, No. 4, 217-224.

Baker, A. B. and Meier, M. J. Anatomical determinants of behavioral change: Presentation 3. In A. L. Benton (Ed.), Behavioral change in cerebrovascular disease. New York: Harper & Row, 1970.

Basmajian, J. V. Control of individual motor units. American Journal of Physical Medicine, 1967, 46:1427-1440.

Basmajian, J. V. Muscles alive: their functions revealed by electromyography (2nd ed.). Baltimore: Williams and Wilkins Co., 1967.

Basmajian, J. V. and Simard, T. G. Effects of distracting movements on the control of trained motor units. American Journal of Physical Medicine, 1967, 46, 480-486.

Basmajian, J. V. Electromyography comes of age. In D. Shapiro, Barber, T. X., Dicara, L. V., Kamiya, J., Miller, N. E., Stoyva, J. (Eds.) Biofeedback and self-control. Chicago: Aldine Publ. Co.: 1972.

Basmajian, J. V., Kukulka, C. G., Narayan, M. G. and Takebe, K. Biofeedback treatment of foot-drop after stroke compared with standard rehabilitation technique: effects on voluntary control and strength, The Archives of Physical Medicine and Rehabilitation, 1975, 56, 231-236.

Bender, L. A. A visual motor Gestalt test and its clinical use. New York: (Research Monograph No. 3) American Orthopsychiatric Association, Inc., 1938.

Bernstein, N. A. Methods for developing physiology as related to the problems of cybernetics. In M. Cole and Maltzman, I. (Eds.). A Handbook of contemporary Soviet psychology. New York: Basic Books, Inc., 1969.

Bernstein, D. A. and Borkovec, T. D. Progressive relaxation training: a manual for the helping professions. Champaign, Ill.: Research Press, 1974.

Bilodeau, I. McD. Information feedback. In E. A. Bilodeau (Ed.), Acquisition of skill. New York: Academic Press, 1966.

Bossom, J. Movement without proprioception. Brain Research, 1974, 71, 285-296.

Bouisset, S. and Lestienne, F. The organization of a simple voluntary movement as analyzed from its kinematic properties. Brain Research, 1974, 71, 451-457.

Brudny, J., Korein, J., Levidow, L., Grunbaum, B. B., Lieberman, A., and Friedmann, L. W. Sensory feedback therapy as a modality of treatment in central nervous system disorders of voluntary movement. Neurology, 1974, 24, No. 10, 925-932.

Brudny, J. Personal communication, January, 1975.

Bruner, J. S. The growth and structure of skill. In K. Connolly (Ed.), Mechanisms of motor skill development. New York: Academic Press, 1970.

Buchwald, J. S., Standish, M., Eldred, E., and Halas, E. S. Contribution of muscle spindle circuits to learning as suggested by training under flexedil. Electroencephalography and Clinical Neurophysiology, 1964, 16, 582-594.

Budzynski, T. H. and Stoyva, J. M. An instrument for producing deep relaxation by means of analog information feedback. Journal of Applied Behavior Analysis. 1969, 2, 231-237.

Budzynski, T. H., Stoyva, J. M., Adler, C. S. and Mulaney, D. J. EMG biofeedback and tension: a controlled outcome study. In N. E. Miller, Barber, T. X., Dicara, L. V., Kamiya, J., Shapiro, D. and Stoyva, J. (Eds.), Biofeedback and self-control. Chicago: Aldine Publ. Co., 1973.

Carlsöö, S. and Edfelt, A. W. Attempts at muscle control with visual and auditory impulses as auxiliary stimuli. Scandinavian Journal of Psychology. 1963, 4, 231-235.

Cleeland, C. S. Behavior techniques in the modification of spasmodic torticollis. In N. E. Miller, Barber, T. X., Dicara, L. V., Kamiya, J., Shapiro, D. and Stoyva, J. (Eds.), Biofeedback and self-control. Chicago: Aldine Publ. Co., 1973.

Efron, R. The conditioned reflex: A meaningless concept. Perspective in Biology and Medicine, 9, 1965-66, 488-514.

Evarts, E. V. Feedback and corollary discharge: A merging of the concepts. Neurosciences Research Program Bulletin, 1971, 9, 86-112.

Evarts, E. V. Minireview: Brain Mechanisms in Motor Control. Life Science, 1974, 1393-1400.

Fitts, P. M. and Posner, M. L. Human performance. Belmont, Calif: Brooks/Cole, 1967.

Frolich, E. D. Pathophysiology: altered regulatory mechanisms in disease. Philadelphia: J. B. Lippincott Co., 1972.

Gellhorn, E. Motion and emotion. Psychological Review, 1964, 71, 457-472.

Gellhorn, E. Principles of autonomic - somatic integrations. Mpls: University of Minnesota Press, 1968.

Germana, J. Central efferent processes and autonomic - behavioral integration. Psychophysiology, 1969, 6, No. 1, 78-90.

Goldstein, I. B. Electromyography: a measure of skeletal muscle response. In N. S. Greenfield and Sternbach, R. A. (Eds.), Handbook of psychophysiology. New York: Holt, Rinehart and Winston, Inc., 1972.

Goodgold, J. and Eberstein, A. Electrodiagnosis of neuromuscular disease. Baltimore: The Williams and Wilkins Co., 1972.

Granit, R. Constant errors in the execution and appreciation of movement. Brain, 1972, 95, 649-660.

Guyton, A. C. Textbook of medical physiology (4th ed.) Philedelphia: W. B. Saunders Co., 1971.

Halstead, W. C. and Wepman, J. M. The Halstead-Wepman aphasia screening test. Journal of Speech and Hearing Disorders, 1949, 14, 9-13.

Hardyck, C. S., Petrinovich, L. F. and Ellsworth, D. W. Feedback of speech muscle activity during silent reading: rapid extinction. Science, 1966, 154, 1467-1468.

Harrison, V. F. and Mortenson, O. A. Identification and voluntary control of single motor unit activity in the tibialis anterior muscle. Anatomical Record, 1962, 144, 109-116.

Hebb, D. O. The distinction between "classical" and "instrumental", Canadian Journal of Psychology, 1956, 10, 165-166.

Hefferline, R. F. The role of proprioception in the control of behavior. Transactions of the New York Academy of Sciences, 1958, 20, 739-764.

Hefferline, R. F., Kennan, B. and Harford, R. A. Escape and avoidance conditioning in human subjects without their observation of the response. Science, 1959, 130, 1338-1339.

Hefferline, R. F. and Keenan, B. Amplitude induction gradient of a small-scale (covert) operant. Journal of the Experimental Analysis of Behavior. 1963, 6, 307-315.

Hefferline, R. F., Perrara, T. B. Proprioceptive discrimination of an operant without its observation by the subject. Science, 1963, 139, 834-835.

Hefferline, R. F., Bruno, L. J. J., and Davidowitz, J. E. Feedback control of covert behavior. In K. Connolly (Ed.), Mechanisms of motor skill development. New York: Academic Press, 1970.

Held, R. Exposure history as a factor in maintaining stability of perception and coordination. Journal of Nervous and Mental Disease. 1961, 132, 26-32.

Held, R. and Hein, A. Movement-produced stimulation in the development of visually guided behavior, Journal of Comparative and Physiological Psychology, 1963, 56, 872-876.

Hernstein, R. J. and Loveland, D. H. Food avoidance in hungry pigeons and other perplexities. Journal of the Experimental Analysis of Behavior, 1972, 18, No. 3, 369-383.

Hess, W. R. Dienephalon: autonomic and extrapyramidal functions. New York: Grune and Stratton, 1954.

Higgins, J. R. and Angel, R. W. Correction of tracking errors without sensory feedback. Journal of Experimental Psychology. 1970, 84, 412-416.

Holst, E. von and Mittelstaedt, H. Das Reafferenzprinzip (Wechselwirkungen zwischen zentralnervensystem und peripheire). Naturwiss. 1950, 37, 464-476.

Howard, I. P. Displacing the optical array. In S. J. Freedman, The neuropsychology of spatially oriented behavior. Homewood, Ill.: Dorsey Press, 1968.

Ingram, T. T. S. The reflex substrata of voluntary activity: general discussion. In K. Connolly (Ed.), Mechanisms of motor skill development. New York: Academic Press, 1970.

Institute of Living. Shipley Institute of Living scale for measuring intellectual impairment. Institute of Living, Hartford 2, Conn.: 1939.

Jacobson, E. Progressive relaxation (2nd ed.). Chicago: University of Chicago Press, 1938.

James, W. Principles of psychology. New York: Holt, 1890.

Johnson, H. E. and Garton, W. H. Muscle re-education in hemiplegia by use of electromyographic device, The Archives of Physical Medicine and Rehabilitation, 1973, 54, 320-325.

Kay H. Analyzing motor skill performance. In K. Connolly Mechanisms of motor skill development. New York: Academic Press, 1970.

Kelleher, R. T. Chaining and conditioned reinforcement. In W. K. Honig (Ed.), Operant behavior: areas of research and application. New York: Appleton-Century-Crofts, 1966.

Kimble, G. A. and Perlmuter, L. C. The problem of volition. Psycho logical Review. 1970, 77, No. 5, 361-384.

Klein, R. M. and Posner, M. I. Attention to visual and kinesthetic components of skills. Brain Research, 1974, 71, 401-411.

Klemm, W. R. Correlation of hippocampal theta rhythm, muscle activity, and brain stem reticular formation activity. Communications in Behavioral Biology. 1970, Part A, 3, 147-151.

Knapp, H. D., Taub, E. and Berman, A. J. Effect of deafferentation on a conditioned avoidance response. Science, 1958, 128, 842-843.

Knapp, H. D., Taub, E., and Berman, A. J. Movements in monkeys with deafferented forelimbs. Experimental Neurology, 1963, 7, 305-315.

Kukulka, C. G., Brown, D. M. and Basmajian, J. V. A preliminary report: biofeedback training for early finger joint mobilization, The American Journal of Occupational Therapy, 1975, 29, No. 8, 469-470.

Lader, M. H. and Matthews, A. M. A physiological model of phobic anxiety and desensitization. Behavior Research and Therapy. 1968, 6, 411-421.

Lambert, K. Explanation and intention. In A. Jacobs and Sachs, L. B. The psychology of private events. New York: Academic Press, 1971.

Lassek, A. M. Inactivation of voluntary motor function following rhizotomy. Journal of Neuropathology and Experimental Neurology. 1953, 3, 83-87.

Leibrecht, B. C., Lloyd, A. J. and Pounder, S. Auditory feedback and conditioning of the single motor unit. In N. E. Miller, Barber, T. X., Dicara, L. V., Kamiya, J., Shapiro, D., Stoyva, J. (Eds.), Biofeedback self-control. Chicago: Aldine Publ. Co., 1973.

Lloyd, A. J. and Caldwell, L. S. Accuracy of active and passive positioning the leg on the basis of kinesthetic cues. Journal of Comparative and Physiological Psychology. 1965, 60, 102-106.

Luria, A. R. Human brain and psychological processes. New York: Harper and Row, 1966.

Luria, A. R. The working brain: an introduction to neuropsychology. New York: Basic Books, Inc., 1973.

Malmo, R. B. and Smith, A. A. Forehead tension and motor irregularities in psychoneurotic patients under stress. Journal of Personality, 1955, 23, 391-406.

Marinacci, A. A. The basic principles underlying neuromuscular re-education. In D. Shapiro, Barber, T. X., Dicara, L. V., Kamiya, J., Miller, N. E., and Stoyva, J. (Eds.), Biofeedback and self-control. Chicago: Aldine Publ. Co., 1972.

Matthews, P. B. C. Muscle spindles and their motor control. Physiological Review, 1964, 44, 219-288.

Meier, M. J. Some challenges for clinical neuropsychology. In R. M. Reitan and Davison, L. A. (Eds.), Clinical Neuropsychology: Current Status and Applications. Washington, D. C.: V. H. Winston & Sons, 1974.

Meier, M. J. and Resch, J. A. Behavioral prediction of short-term neurologic change following acute onset of cerebrovascular symptoms. Mayo Clinic Proceedings, 1967, 42, 641-647.

Merton, P. A. Human position sense and sense of effort. Symposia of the Society for Experimental Biology. 1964, 18, 387-400.

Merton, P. A. The properties of the human muscle servo. Brain Research, 1974, 71, 475-478.

Miller, N. E. Learning of visceral and glandular responses. Science, 1969, 163, 434-435.

Milner, P. M. Physiological psychology. New York: Holt, Rinehart and Winston, Inc., 1970.

Moore, B. R. The role of directed Pavlovian reactions in simple instrumental learning in the pigeon. In Hinde, R. A. and Hinde, J. Stevenson (Eds.), Constraints on Learning. New York: Academic Press, 1973.

Mountcastle, V. B. (Ed.), Medical physiology (13th ed.), Vol. 1., St. Louis: C. V. Mosby Co., 1974.

Nissen, H. W., Chow, K. L., and Semmes, J. Effects of restricted opportunity for tactual, kinesthetic, and manipulative experience on the behavior of a chimpanzee, The American Journal of Psychology, 1951, 64, No. 4, 485-507.

O'Brien, F. O. and Azrin, N. H. Behavioral engineering: control of posture by informational feedback. Journal of Applied Behavior Analysis. 1970, 3, 235-240.

Oscarsson, O. Functional organization of spino-cerebellar paths. In A. Iggo (Ed.), Handbook of sensory physiology: somatosensory system, Vol. 2. Berlin: Springer-Verlag, 1970.

Paillard, J. The patterning of skilled movements. In J. Field, Magoun, H. W., and Hall, V. E. (Eds.), Handbook of physiology: Neurophysiology, Sec. 1, Vol. 3. Baltimore: Waverly Press, Inc., 1960.

Paillard, J. and Brouchon, M. Active and passive movements in the calibration of position sense. In S. J. Freedman, The neuropsychology of spatially oriented behavior. Homewood, Ill.: Dorsey Press, 1968.

Paillard, J. and Brouchon, M. A proprioceptive contribution to the spatial encoding of position cues for ballistic movements. Brain Research, 71, 1974, 273-284.

Parsegian, V. L. This cybernetic world of men, machines, and earth systems. Garden City, N. Y.: Anchor Books, Doubleday and Doubleday, Inc., 1973.

Penfield, W. The cerebral cortex in man. Archives of Neurology and Psychiatry, 1938, 40, 417-442.

Porteus, S. D. The Maze Test and Clinical Psychology. Palo Alto, Ca.: Pacific Books, 1959.

Raskin, M., Johnson, G., and Rondestvedt, J. W. Chronic anxiety treated by feedback-induced muscle relaxation: a pilot study. In N. E. Miller, Barber, T. X., Dicara, L. V., Kamiya, J., Shapiro, D. and Stoyva, J., (Eds.), Biofeedback and self-control. Chicago: Aldine Publ. Co., 1973.

Reitan, R. M. Validity of the trail making test as an indicator of organic brain damage. Perceptual Motor Skills. 1958, 8, 271-276.

Reynolds, G. S. A primer of operant conditioning. Glenview, Ill.: Scott, Foresman and Co., 1968.

Sage, G. H. Introduction to motor behavior: a neuropsychological approach. Reading, Mass.: Addison-Wesley Publ. Co., 1971.

Sasmoor, R. M. Operant conditioning of a small-scale muscle response. Journal of the Experimental Analysis of Behavior. 1966, 9, No. 1, 69-85.

Schultz, J. W. and Luthe, W. Autogenic training. New York: Grune and Stratton, 1959.

Scully, H. E. and Basmajian, J. V. Effect of nerve stimulation on trained motor unit control. Archives of Physical Medicine and Rehabilitation. 1969, 50, 32-33.

Sears, T. A. The afferent regulation of learnt movements. Brain Research, 1974, 71, 465-473.

Segal, E. F. Induction and the provenance of operants. In R. M. Gilbert and Millenson, J. R. (Eds.), Reinforcement: behavioral analyses. New York: Academic Press, 1972.

Sherrington, Sir C. The integrative action of the nervous system. New Haven: Yale University Press, 1906.

Skinner, B. F. The behavior of organisms: an experimental analysis. New York: Appleton-Century-Crofts, 1938.

Skinner, B. F. Operant behavior. In W. K. Honig (Ed.), Operant behavior: areas of research and application. New York: Appleton-Century-Crofts, 1966.

Skinner, B. F. Contingencies of reinforcement: a theoretical analysis. New York: Appleton, 1969.

Smith, K. V. and Smith, W. M. Perception and motion. Philadelphia: W. B. Saunders Co., 1962.

Smith, K. V. Cybernetic theory and analysis of learning. In E. A. Bilodeau (Ed.), Acquisition of skill. New York: Academic Press, 1966.

Solomon, R. L. and Turner, L. H. Discriminative classical conditioning in dogs paralyzed by curare can later control discriminative avoidance response in the normal state. Psychological Review. 1962, 69, No. 3, 202-219.

Sperry, R. W. Neural basis of the spontaneous optokinetic response produced by visual neural inversion. Journal of Comparative and Physiological Psychology, 1950, 43, 482-489.

Taub, E. and Berman, A. J. The effect of massive somatic deafferentation on behavior and wakefulness in monkeys. Paper read at Psychonomic Society, Niagra Falls, Ont., Oct., 1964.

Taub, E. and Ellman, S. J. and Berman, A. J. Deafferentation in monkeys: effect on conditioned grasp response. Science, 1966, 151, 593-594.

Taub, E. and Berman, A. J. Movement and learning in the absence of sensory feedback. In S. J. Freedman, The Neuropsychology of spatially oriented behavior. Homewood, Ill.: Dorsey Press, 1968.

Terrace, H. S. Errorless transfer of a discrimination across two continua. Journal of the Experimental Analysis of Behavior. 1963, 6, 223-232.

Teuber, H. L. Perception. From B. Milner and Teuber, H. L. Alteration of perception and memory in man: reflections on methods. In L. Weiskrantz (Ed.), Analysis of Behavioral Change. New York: Harper and Row, 1968.

Towe, A. L. Motor cortex and the pyramidal system. In J. D. Maser (Ed.), Efferent organization and the integration of behavior. New York: Academic Press, 1973.

Twitchell, T. E. Sensory factors in purposive movement. Journal of Neurophysiology, 1954, 17, 239-254.

Vanderwolf, C. H. Mechanisms of voluntary movement. Psychological Review. 1971, 78, No. 2, 83-113.

Wagman, I. H., Pierce, D. S., Burger, R. E. Proprioceptive influence in volitional control of individual motor units. Nature, 1965, 207, 957-958.

Wechsler, D. A standardized memory scale for clinical use. The Journal of Psychology, 1945, 19, 87-95.

Wechsler, D. Manual for the Wechsler Adult Intelligence Scale. New York: The Psychological Corp., 1955.

Weiner, N. Cybernetics: or control and communication in the animal and the machine. New York: John Wiley and Sons, Inc., 1948.

Wellford, A. T. On the sequencing of action. Brain Research, 1974, 71, 381-392.

Whishaw, I. W. and Vanderwolf, C. H. Hippocampal EEG and behavior: changes in amplitude and frequency of RSA (theta rhythm) associated with spontaneous and learned movement patterns in rats and cats. Behavioral Biology, 1973, 8, No. 4, 461-484.

Wickramasekera, I. Instructions and EMG feedback in systematic desensitization: a case report. Behavior Therapy. 1972, 3, 460-465.

Wickramasekera, I. Electromyographic feedback training and tension headache: preliminary observations. In N. E. Miller, Barber, T. X., Dicara, L. V., Kamiya, J., Shapiro, D., and Stoyva, J. (Eds.), Biofeedback and self-control. Chicago: Aldine Publ. Co., 1973.

Woodburne, L. S. The neural basis of behavior. Columbus, Ohio: C. E. Merrill Books, Inc., 1967.

Suggested Readings

Eccles, J. C. Brain, speech, and consciousness. In J. C. Eccles, The understanding of the brain. New York: McGraw-Hill Book Co., 1973.

Green, A. M. and Green E. E. Biofeedback: research and therapy. In N. O. Jacobson (Ed.), In being well is a responsibility! London: Turnstone Books, 1975. (in press)

Segal, J. Biofeedback as a medical treatment, Journal of the American Medical Association, 1975, 232, No. 2, 179-180.

Sister Kenny Institute Staff. About stroke. Minneapolis: Sister Kenny Institute, 1975. (A very lucid, useful booklet for persons who have suffered stroke, as well as family, friends, and counselors, etc. of stroke patients.)