

BIOFEEDBACK

Principles and Practice for Clinicians

SECOND EDITION

Edited by

John V. Basmajian, M.D.



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Preface to the Second Edition

In a new field of science and therapy that is rapidly changing, significant books become either collectors' items or important landmarks that measure progress. The first edition of this work served its time well as a landmark and we are grateful for its generous reception by reviewers and readers. But we cannot ignore the passage of time. So this new edition was planned to record ideas and results that are new and provocative or new and accepted, while retaining and consolidating whatever is older and acceptable.

"Old" is really not very old, for biofeedback as a widely recognized discipline is barely a teenager. But in its short life it has been documented in some ten thousand professional articles and its practitioners and scientists number in the many thousands around the world. Perhaps its greatest success is the major impetus it has given to the growing field of behavioral medicine. By dramatically revealing the extent of self-regulation possible with suitable training techniques, biofeedback showed physicians, rehabilitation therapists, behavioral scientists and clinical psychologists that they all had responsibilities in the same health camp—at least those who were not sure that their current practices were perfect.

In this new edition, most of the old chapter headings have been retained while the contents were changed to varying degrees. The chapter devoted to the scientific basis of biofeedback is split by parthenogenesis into one on striated muscle (Chapter 2) and another on the autonomic nervous system (Chapter 3). New and authoritative chapters include: Chapter 4 by Joel Lubar on the theory and practice of electroencephalographic biofeedback; Chapter 22 by Herbert Johnson and Virgil Hockersmith on biofeedback in pain management; Chapter 21 by Andrew Cannistraci and George Fritz on the use of biofeedback in dental practice; Chapter 23 by Keith Sedlacek on Raynaud's disease; Chapter 26 by David Paskewitz on computers in biofeedback; and Chapter 28 by Gary DeBacher on the construction and uses of electrogoniometry for biofeedback retraining of articular functions in rehabilitation settings.

Several chapters have been completely rewritten by the previous authors to reflect changes of thought and practice. Others have been rewritten by new authors. A companion book by this same publisher obviated the need for intensive "how-to" chapters on the methodologies for psychotherapy (Gaarder and Montgomery: *Clinical Biofeedback: a Procedural Manual for Behavioral Medicine*, 2nd ed.). Our technical book on electrode placement for specific ("targeted") muscle retraining (Basmajian and Blumenstein: *Electrode Placement for EMG Biofeedback*) has been such a success that the publisher and Dr. Blumenstein agreed heartily with me to absorb most of it into this book to make it more comprehensive and available for all physicians, rehabilitation therapists, psychotherapists and behavioral scientists. We invite their suggestions for further improvements that may someday reflect the best of biofeedback theory and practice in a third edition. Meanwhile this book is the state of the art and science today.

Acknowledgments for this book are mostly deserved by the chapter authors. They responded to my urgent requests with easy grace and reasonable promptness. The publishers continued to be cooperative and responsive, making my editorial role as simple as that complex task can be made. To earlier authors, present authors, and all who contributed to the success of the first edition and the preparation of this second edition, my warmest thanks.

Preface to the First Edition

Practical biofeedback in medicine and psychotherapy has come of age. In its embryonic period during the 1960s and early 1970s some doubts as to its legitimacy and future prospects were widely expressed. As one of its putative fathers, I was deeply concerned that biofeedback—particularly myoelectric biofeedback—should get a fair start. Thus, along with many students, colleagues and with growing numbers of workers in other centers, we struggled to provide the research which nourished its growth. During the late 1960s this healthy growth was threatened by a popular sweep of interest in biofeedback and the accompanying host of entrepreneurs who began exploiting it—especially “alpha feedback”—for a variety of purposes.

Alpha feedback, after its glamorous and confused flirtation with the public press, has returned now to research where it needs to mature. Serious clinicians emerged from the early popularization phase relatively safely and the popular press is beginning to understand that biofeedback is a medical methodology. The more directly useful clinical applications have matured through validating research projects to the point where clinicians can safely apply some aspects to the treatment of well-defined conditions while they continue research in other aspects of biofeedback applications.

The authors of the various chapters all have had a commitment to research and/or application of research results in biofeedback. Several have been doing basic research in the field of electronic recordings of biological phenomena for many years; others moved from related clinical fields into biofeedback research and therapy with ease because of their strong clinical backgrounds. They are generally recognized as leaders in their special fields. Most important, they are expert therapists and diagnosticians who employ biofeedback techniques judiciously and selectively, not as a shotgun treatment.

We offer this book as a timely summary and explication of the best applications of biofeedback treatment techniques to neurological, psychosomatic and psychological disturbances. The techniques are not final and complete, but the underlying principle is quite clear and substantiated. Provided with various electronic instrumental displays of covert responses, patients can acquire substantial voluntary control of them. Where the responses are exaggerated, they can be reduced by an effort of will; where reduced (e.g., paresis), they sometimes can be restored or reinforced by retraining. Treatment of patients with stroke, cerebral palsy, severe recurring headaches, all distressingly difficult (sometimes hopeless) in the past, offers new hope to many patients. Other conditions not so impressive to an observer, but very important to the sufferer, also respond to biofeedback techniques. This book brings together in one volume the present state of the art.

Hamilton, Canada
1978

J. V. BASMAJIAN

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PART 1

INTRODUCTION AND

NEUROSCIENCE

CHAPTER 1

Introduction: Principles and Background

JOHN V. BASMAJIAN

Biofeedback may be defined as the technique of using equipment (usually electronic) to reveal to human beings some of their internal physiological events, normal and abnormal, in the form of visual and auditory signals in order to teach them to manipulate these otherwise involuntary or unfelt events by manipulating the displayed signals. This technique inserts a person's volition into the gap of an open feedback loop—hence the artificial name *biofeedback*, a name that some scientists and clinicians abhor for linguistic and other reasons. Unlike conditioned responses, the animal involved, here necessarily a human being, must want to voluntarily change the signals because they meet some goals.

Teaching patients to control a wide range of physiological processes occasionally has amazing therapeutic results, but legitimate clinical use of biofeedback as described in this book must be differentiated from the fad which caught the popular imagination in the late 1960s. While many self-serving promoters of general biofeedback are still at work, true clinical biofeedback has quietly taken a place as a genuine treatment for a growing number of neurological and psychosomatic ailments. Both scientific and practical studies provide us with sufficient concrete evidence that objec-

tive neurological signs and symptoms can be altered, particularly in patients with upper motor neuron paralysis and spasticity due to brain damage. The easiest form of do-it-yourself biofeedback—alpha brain wave biofeedback—is still not understood scientifically. "Alpha feedback" is still a mystery and it is not an acceptable treatment method. Other forms of EEG feedback have shown considerable promise in the experimental situation (Chapter 4).

Biofeedback control of cardiac rates and blood pressure is emerging slowly from research-oriented clinical studies. Whether it will have great practical usefulness with large numbers of hypertensive patients remains to be seen. Reports of patients who maintain continued lower blood pressure over months and other patients who can willfully elevate their blood pressure (to prevent, for example, orthostatic hypotension) provide striking evidence that biofeedback may have very practical results in changing cardiovascular function.

Among the most striking areas of biofeedback treatment has been the alleviation of migraine by teaching subjects to elevate the temperature of fingertips. While some of the results may arise from "placebo effect," the evidence is growing that the link between

peripheral blood flow and cranial blood flow provides a clear explanation of the phenomenon.

Another area in which placebo effects provide considerable underpinning of the results is in the treatment of chronic tension headaches with myoelectric biofeedback that is aimed at reducing general muscle tension. Deep relaxation strikes directly at the cause, i.e., muscle tension. There are other ways in which relaxation can be taught effectively, but biofeedback gadgets are useful in accelerating the treatment and good psychotherapists quickly wean their patients from the gadgets. They teach their patients self-control as part of their long-term treatment. General or total body relaxation also has proved to be very useful in the rehabilitation clinic along with targeted relaxation of specific muscles in spasm. Thus, in patients with dyskinesias (e.g., spasmodic torticollis) and cerebral palsy, and patients who have suffered stroke and have spasticity, relaxation therapy enhances their subsequent training of improved motor performance.

In rehabilitation, the most useful feedback has been myoelectric or "electromyographic" (EMG). "Electromyographic" is probably not a good adjective to apply to this form of biofeedback because the patient and the clinician both do not view electromyograms or an electromyograph. Instead, the myoelectric signals from the muscle are translated into acoustic and visual signals that are very simple to understand—buzzing sounds and lights. A premise of myoelectric biofeedback has always been that patients can respond to a physician's request during an EMG examination to alter the coarse level of activity of individual muscles as they are displayed as spikes on a cathode ray oscilloscope and also heard as popping noises on a loudspeaker. Indirectly this knowledge (gained in the period following the Second World War in many clinics including mine) led to my early studies. In the 1950s and 1960s we were advocating the possible application of tiny sources of muscle potentials for the development and control of myoelectric prostheses. This led to more intense studies of how this control is exerted and its exquisite nature.

Several streams springing from widely separated sources came together in the 1960s to form the broad but shallow river that was to become the clinical biofeedback technology of

today. This river is becoming deeper as scientists and clinicians add controlled research results to the wide scattering of case reports. One of the early sources was the work done by Jacobson (1) in the 1920s and 1930s. He developed and became an enthusiastic proponent of relaxation therapy in which he employed rather primitive electromyographic equipment to monitor the level of tension in the muscles of his patients. Limited by the apparatus available at that time, Jacobson developed methods of electrical measurement of the muscular status of tension and with these measurements he facilitated progressive somatic relaxation for a variety of psychoneurotic syndromes. Meanwhile, in Germany, Shultz (2) developed his related technique, autogenic training, which has been most widely popularized in Canada by his pupil Luthe (3). Although autogenic training employed no specific EMG equipment, nevertheless it was one of the early sources of much of today's biofeedback application in the treatment of psychosomatic and neurotic symptoms.

While medical specialists and psychologists who knew of these relaxation techniques kept them alive and growing, the field of diagnostic electromyography was spreading equally slowly. Electromyography grew out of studies of neuromuscular and spinal cord functions. It began with the classic paper in 1929 by Adrian and Bronk (4) who showed that the electrical responses in individual muscles provided an accurate reflection of the actual functional activity of the muscles. In subsequent years only a scattering of research papers kept up the flow of the stream that was to become myoelectric biofeedback alive. Thus, in 1934, Olive Smith (5) reported observations on human conscious control of individual motor unit potentials, their general behavior and their frequency. She showed that normally there is no proper or inherent rhythm acting as a limiting factor in the activity of motor units. Shortly after, Lindsley (6) confirmed Smith's findings and seems to have been the first scientist to emphasize that at rest "subjects can relax a muscle so completely that . . . no active units are found." Relaxation sometimes requires conscious effort and in some cases special training.

Lindsley found that complete relaxation was not difficult in any of his normal subjects. This finding has since been confirmed by thousands

of investigators using modern electrical apparatus. Gilson and Mills (7), Harrison and Mortensen (8) and I (9) continued this type of work. Using fine-wire electrodes which we had recently developed for other purposes, I was able to train normal subjects to isolate many separate motor units and to activate them consciously. This was a form of single spinal motor neuron training. Then a long series of studies with my students and colleagues led progressively towards a confluence with the other streams that were to form the modern biofeedback river.

The influence of the single motor unit research projects on the development of therapeutic techniques was not dramatic. As early as 1960, Marinacci and Horande (10) presented various case histories and discussed the effectiveness of the display of the EMG to patients in an effort to restore function for various neurological conditions. Later, Andrews (11) trained stroke patients to flex and extend a paralyzed elbow region. Then a growing number of investigators began to concentrate on the treatment of stroke patients with myoelectric biofeedback (12-14). The myoelectric control "stream" joined the muscle relaxation "stream" in the mid-1960s. Following my publications on single motor unit controls (9, 15), Budzynski *et al.* (16) developed a technique for inducing generalized body relaxation for the relief of tension headaches. This type of application of myoelectric feedback has grown to dominate the entire field. While medical rehabilitation groups emphasize targeted relaxation or targeted retraining of semiparetic muscles, clinical psychologists and specialists in psychosomatic medicine almost limit their clinical work to general deep relaxation, often combining Jacobsonian techniques and autogenic training along with instrumental relaxation.

Two other streams arrived in the mid-1960s. One of these was the increasing use of operant conditioning in animal psychological research. Thus, the cardiovascular conditioning of Miller and his colleagues (17) caught the imagination of many scientists and clinicians (18). At the same time experts in EEG began to report interesting correlations between the state of emotional set and consciousness on the one hand and the amount of alpha waves generated by the subject on the other. By the end of the 1960s all of the various streams had joined, leading to the formation in 1969 of the

Biofeedback Research Society (renamed in 1976 the Biofeedback Society of America).

In the 1970s the shape of biofeedback was changed dramatically. The most dominant form which received the greatest publicity in the late 1960s, alpha feedback, has virtually dried up as a scientifically defensible clinical tool. Where it is still used by serious clinicians, it is combined with other techniques to achieve relaxation. However, it has also returned to the research laboratory from which it probably should not have emerged prematurely. Through the next generation of scientific investigation, it may return as a useful applied technique.

Temperature and/or peripheral blood flow control of the extremities has become one of the major tools of clinical psychologists and their scientific associates. Considerable controversy continues as to whether direct blood flow measurements, photoplethysmography or skin-temperature sensing devices are preferable. These matters are considered elsewhere in this volume as are the possibilities for altering blood pressure and general tension.

The most dramatic application of biofeedback to large numbers of severely handicapped patients has been in the area of myoelectric biofeedback. Both for general relaxation, which alleviates many symptoms due to anxiety or excess muscular tension (e.g., tension headache, general anxiety, stress pain such as low-back pain, etc.), and for the treatment of neurological handicaps, myoelectric biofeedback has gained a solid following of clinical scientists. In the physical rehabilitation field, biofeedback has made its greatest medical strides. For example, we reported that 64% of our patients wearing a short leg brace for foot-drop following stroke could discard the device after several weeks of biofeedback training (19). This confirmed our earlier control studies which were performed more rigidly (14). We have also gained considerable experience with the retraining of patients with tendon transfers and surgery in the hand (20), and with spasticity control (19). These matters will be discussed in subsequent chapters.

To conclude this chapter, a quotation from my 1976 paper in *Modern Medicine* is still appropriate (21):

... a cautiously optimistic view of clinical biofeedback is justified. In rehabilitation medicine and neurology it offers the only hope for

the alleviation or reversal of many severe symptoms. In reparative tendon surgery and orthopedics, it expands the tools of the physical and occupational therapist. In psychiatry, psychotherapy, and general medicine it offers a tool for deep relaxation therapies that can lead to alleviation of many symptoms. In cardiology, it offers real hope of benefits for at least some hypertensive patients. By acquiring sound knowledge, physicians and therapists can ensure that the uses of biofeedback in treatment of patients are both scientifically correct and ethical.

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Neurophysiological Factors in Electromyographic Feedback for Neuromotor Disturbances

STEVEN L. WOLF

Significant strides have been achieved in both clinical and experimental biofeedback applications governing several anatomical systems. These advances may be considered remarkable in light of the relatively short time in which investigators have been examining the far-reaching implications of biofeedback training. From a clinical perspective, instances demonstrating appropriate physiological performance in patients or laboratory subjects have become known to the clinician, who has been able to successfully implement such findings and thus improve the status of his client. Nevertheless, any new technique is bound either to fall by the wayside or to become easily labeled as a technology if its roots are not quickly and correctly planted in soil tempered by scientific merit and credence. As Brown (1) has correctly noted:

Laboratory procedures are developed and modified to probe and uncover elements critical to the phenomenon being studied, and relatively few research studies are directed toward evaluating elements critical to successful application. This has been especially true in biofeedback where it has been assumed, often with considerable error, that the essential aspects for optimal clinical effectiveness have been adequately defined.

Indeed, references in the biofeedback literature are filled with studies which clearly define variables and statistically or empirically analyze results. Seldom have investigators extrapolated information contained within the annals of basic scientific research to explain

or hypothesize mechanisms to account for clinical observations. While a systems analysis explanation of biofeedback has been provided (2, 3), the purpose of this chapter is to provide (a) a general review of the essentially neurophysiological events underlying motor control and (b) mechanisms which might contribute to an understanding of reports supporting the efficacy of biofeedback treatments. It should be emphasized that the discussion which follows is not designed to unequivocally explain or interpret neural mechanisms invoked by virtue of biofeedback paradigms. Rather it is to encourage researchers and clinicians to think about and delve into the physiological bases of their observations. Discarding empiricism in favor of thought-provoking concepts can elevate the status and potential of biofeedback.

EMG FEEDBACK TO ACHIEVE MOTOR CONTROL

A primary objective of all electromyographic (EMG) biofeedback is to enable the patient to reacquire voluntary control over his striated musculature. Goals are directed toward an appropriate increase in activity of weak or paretic muscle and a reduction in activity levels of spastic muscles (4, 5). The anatomical and physiological factors underlying the processing of information from muscle feedback systems are complex and poorly understood. Yet, compared to feedback systems governing autonomic or behavioral functions, a measurement of treatment efficacy can be more easily assessed from muscle feedback.

Ultimately the acquisition of a motor skill can be manifest in any variety of ways—from the elimination of an assistive ambulatory device to the resumption of a vocation.

In clinical EMG feedback training a patient is made aware of minute or overexaggerated muscle contractions through visual and auditory feedback from that muscle. The information provided instantaneously about this covert activity is readily processed by the patient. An examination of patient behavior as the training procedure progresses enables the observer to offer some speculations on the neural mechanisms contributing to reacquisition of motor control. In the case of the patient with neurological deficits, processing the informational content of a feedback signal is confounded by the disruption of supraspinal neural influences upon muscle. Patients at first rely upon visual and auditory representations of muscle activity quite extensively. As the patient improves in ability to initiate and terminate muscle contractions he becomes less dependent on electronically generated cues and more reliant upon visual input from the moving limb segment.

Traditional Rehabilitation Procedures

EMG biofeedback as a muscle reeducation tool should not be employed at the expense of traditional therapeutic procedures, but it should, at least, serve as an adjunct. One should realize that rehabilitation personnel serve as exteroceptive stimulants for the patient. A therapist's ability, for example, to palpate a spastic muscle and instruct the patient to relax provides verbal feedback about motor behavior which the patient cannot adequately perceive. Specifically, most therapies are designed to provide appropriate proprioceptive input so that the patient may learn to make the proper motor response. While an EMG feedback machine can never substitute for a clinician, its advantages are obvious. Such a device provides exteroceptive cues which are accurate and instantaneous. Quantitatively the information available is always proportionate to the magnitude of muscle force. Realistically the feedback signal may substitute for inadequate proprioceptive signals and can be used to shape responses more precisely than signals generated by any clinician. This may enable the central nervous system to reestablish appropriate sensory-motor loops under the

volitional control of the patient. A discussion of this issue has been well presented in a monograph (6).

Clinical Efficacy Studies

The inclusion of EMG feedback applications among patients with purely musculoskeletal disorders has proven to be a valuable adjunct in treatment following meniscectomy (7) or chronic low back pain (8). In such cases the neural circuitry necessary to adequately process feedback signals is intact. Loss of kinesthetic cues is non-existent and, primarily, muscle weakness or reduced strength predominates. In such cases the patient uses visual and auditory representations of muscle activity in combination with increased motivational drive to accelerate corticospinal and parallel descending motor systems to induce increased motor neuronal drive.

The situation is exceedingly more complex among patients who are afflicted with central nervous system deficits. Not only must disinhibition of specific muscle groups be overcome through reduction of hypermotor responses, but a reappreciation of proprioceptive cues must be gained. Knowledge regarding the efficacy of feedback interventions to cause such improvements is inadequate primarily because of the limited number of controlled studies comparing feedback interventions to no treatment or other physical therapeutic procedures. Significant improvement in upper (9) and lower (10) extremity function in stroke patients exposed to EMG feedback paradigms has been reported. Results from these studies were based upon clinical grades rather than concrete data and lack appropriate controls. Basmajian and colleagues (9) attempted to demonstrate that superior strength and range of motion around the ankle joint can occur among stroke patients who receive feedback and therapeutic exercise as compared to individuals receiving exercise alone. Unfortunately, this work has been criticized because, (despite randomization in group determinations) those individuals receiving feedback and exercise were comparatively more recent in stroke onset than the group receiving exercise exclusively. The results from that study, however, were substantiated by a controlled, blind study among a chronic stroke population, undertaken by Binder and colleagues (11). Patients receiving a standardized exercise

program as well chronic stroke patients receiving feedback in conjunction with exercise showed improvements.

On the other hand, the work of Lee and colleagues (12) focused attention on the deltoid muscle in only three experimental sessions among stroke patients with hemiplegia from six months to seven years in duration. Results from that study indicated no differences with respect to changes in EMG levels among patients placed in a true feedback or false feedback group. Unfortunately, all patients were subjected to limited measurement trials of short duration. Of potential importance was the observation that older and less motivated patients did not do as well as younger patients. A crossover design that compared EMG feedback with physical therapy has been reported by Mroczek and colleagues (13). This experiment examined changes in wrist mobility. Biofeedback after physical therapy appeared less effective than the reverse sequence, which suggests that different strategies may be necessary, and the strategies employed by the patient during physical therapy may interfere with the biofeedback strategy.

Wolf and colleagues (13, 14) have attempted to make exacting neurophysiological measurements of improvement among stroke patients subjected to feedback training exclusively. At the conclusion of their study and at one-year follow-up the results remained consistent, which suggests that improvements learned during feedback sessions were retained. Of significance was the fact that improvements among chronic stroke patients could occur without respect to age, side of the lesion, duration of stroke, or duration of previous rehabilitation. These findings have subsequently been corroborated by other investigators (15-17). The greatest apparent deterrent to successfully interfacing stroke patients with feedback appears to be the magnitude of dyskinesia that exists among patients engaged in feedback training.

Few attempts have been made to explain changes in neuronal processing during feedback applications among patients with central nervous system pathology (18, 19). In fact, most information designed to provide explanations about how exteroceptive signals induce improved sensory-motor integration is exceptionally vague. This state of affairs is not terribly surprising, since neuronal circuitry for exe-

cution of simple placement movements among sub-human primates is uncertain. In summary, while feedback applications to neurological patients have appeared promising, further controlled clinical studies are required and neurophysiological correlates of movement must be obtained in animal models that simulate clinical neurological conditions.

MOTOR CONTROL AND FEEDBACK LOOPS

How do patients process feedback information? What factors account for the patient's ability to gradually become less dependent upon artificially induced information signals? Why might patients gain motor control using EMG feedback after failing to achieve significant gains using conventional rehabilitation procedures? Answers to these questions and others form the basis for our understanding of EMG feedback. By exploring these issues many of the anatomical and neurophysiological components necessarily associated with EMG feedback emerge. Even more questions are raised than answered. What follows below, then, is a discussion of several aspects of "feedback loops" involved with establishing motor control. It is indeed remarkable that a multitude of internal, modulatory neural networks exists, and, even more significant, that electronic feedback may evoke exteroceptive systems that ultimately engage these internal networks.

The information contained below, although broad in nature, relies heavily upon decades of intensive observations on motor control systems within sub-human primate models. Most of the neurophysiological data recorded from conscious animals have been acquired using operant conditioning paradigms.*

While one would be tempted to extract mechanisms from motor control studies that explain how feedback can restore motor func-

* Operant conditioning should not be confused with EMG biofeedback training techniques. The former refers to a condition in which a particular behavior is selectively reinforced so that its frequency of occurrence may be augmented or reduced. Frequently the motivational component of an operant conditioning paradigm is difficult to evaluate. EMG feedback provides a precise and continuous signal proportional to the subject's response. Operant reinforcement is often discontinuous, delayed or intermittent (6).

tion among neurological patients, such external validation must be excessively tempered. First, the simple act of executing a command, such as volitional grasp of an object, includes complex sensory and motor circuitry that has yet to be clearly elucidated. Second, changes in neuronal processing following experimentally induced central nervous system lesions very rarely mimics true pathology. For example, the neuronal damage following a cerebral vascular accident is often vague and non-specific. Even a disruption of a specific cerebral arterial component can produce highly variable neuronal losses. Therefore, naturally occurring neuropathology can rarely be equated to specific experimental lesions. Caution must be exercised in transferring mechanisms underlying sensory-motor integration in the presence of experimentally induced pathology to clinical situations. Nonetheless it is imperative to understand some conceptions regarding motor and sensory functioning during volitional activities and to speculate upon how exteroceptive cues might contribute to improved fluidity of movement and function.

An Overview

The general scheme involving essential components to execute volitional movement is shown in Figure 2.1, derived and modified from the work by Allen and Tsukahara (20). Information about the formulation of a voli-

tional motor act is conveyed from cortical association areas to the lateral cerebellum, basal ganglia and the premotor cortex (including the posterior parietal lobe and frontal lobe premotor areas). This information is converted into a command for movement that is organized into neuronal discharge patterns at appropriate efferent areas of the motor cortex (21-23). In this case the basal ganglia may reinforce motor cortical activity (24-26) or, if appropriate, cause a delayed motor response (27-29). The basal ganglia also function to integrate the motor command with appropriate visual, auditory, proprioceptive and other exteroceptive inputs. As a result, eye and body responses are oriented toward attention to a limb movement (24, 30, 31). The lateral cerebellum functions to provide appropriate temporal patterns of agonist-antagonist muscle activity so that the programmed movement is appropriate. In part this comparator function of the lateral cerebellum is based upon prior motor learning (32, 33).

The neuronal patterns emerging from the motor cortex may activate brain stem and spinal efferent systems for further elaboration on the execution of the desired movement as well as for provision of essential input to the intermediate cerebellum and somatic sensory areas. These areas subsequently may modulate their responses to sensory input that will be derived from the resulting movement (34-36).

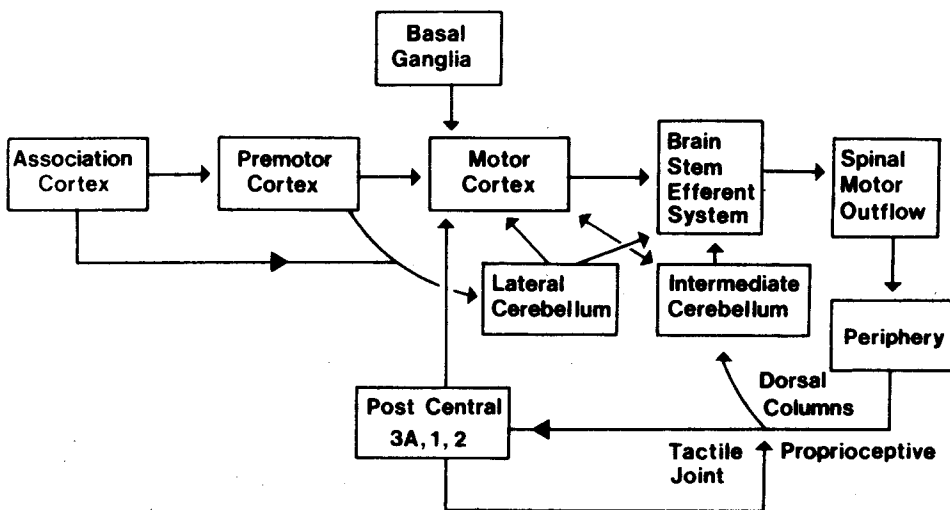


Figure 2.1. Essential central and peripheral nervous system components involved in the execution of a voluntary movement. Modified after Allen and Tsukahara (20). See text for details.