The Comprehensive Handbook of BEHAVIORAL MEDICINE

Volume 1: Systems Intervention

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Preface

Many of the greatest strides in medical care have neither been glamorous nor made the front page of *The New York Times*. They have been simple measures such as sanitation, immunization, and provision of clean, wholesome food. And even more glamorous medical breakthroughs and techniques like heart transplants are often last-ditch responses to largely preventable medical problems that required a lifetime to develop. Changing those life styles which may cause, worsen, or exacerbate disease and utilizing current medical knowledge may be the most important strides medicine will make in the next few decades. To meet this challenge, techniques have already been developed to change eating and nutritional patterns that may lead to obesity and heart disease. In addition, interventions are being developed for a wide variety of medical problems. Many of these techniques are based on behavioral principles.

Several years ago, one of the editors of this book gave a behavioral medicine seminar for psychiatry residents concerning behavioral principles and their application to medicine. As the seminar developed, it became evident that many of the important articles on the subject were scattered throughout a wide body of literature, which encompassed a variety of disciplines and journals. No single source was available to provide the state of the art of this emerging field. This book was spawned, in part, as an attempt to overcome this deficit. We wanted to provide a handbook to help health-care practitioners understand and use behavioral techniques appropriate to their areas of interest. However, we also wanted to develop a handbook that would be useful to researchers. To reach both audiences we decided to include a wide range of chapters, some focusing on practical and clinical issues, others focusing on theoretical or technical issues. We encouraged the contributors to speculate about future directions in their areas of specialty. We also decided to include several sections on basic physiology, to illustrate the potential for change in several systems.

Our contributors have been patient and supportive. They have watched as pages of "golden prose" disappeared under the hand of heavy editing. Many made last-minute additions to make their material as up-to-date as possible. And, we have been impressed with the many changes our contributors have undergone in their lives. Some have moved or received promotions, new degrees, and a variety of honors. They have married and

divorced, had children, suffered the deaths of loved ones, and died themselves. Especially in light of these many personal changes, we thank them for their help.

We have also been impressed with the tremendous changes in the field of behavioral medicine. Since the field will undoubtedly continue to change, we intend to add volumes to the Comprehensive Handbook to keep it as up-to-date as possible.

In this work, we would like to acknowledge the help of many individuals. We received editorial assistance from Patricia Benefiel, Ph.D., and Francis Filloux, and many suggestions about content from our colleagues. The manuscript has been typed, retyped, and re-retyped tirelessly by Dottie Pakus. Tome Tomisawa has also helped enormously with preparation of the book. The project has been generously supported by a grant from the Janss Foundation, which helped with the multiple and miscellaneous expenses associated with manuscript preparation, and psychologically supported by the enthusiasm of the Foundation's President, Mr. Joseph Legett.

Introduction

Research in the behavioral sciences has led to an explosive growth in information of potential use to the medical profession. This information includes basic and applied studies on the initiation, persistence, and treatment of psychophysiological disorders as well as numerous case reports and systematic investigations of successful behavioral interventions in medical disorders. The theoretical and practical descriptions of techniques and the case reports of interventions are spread widely throughout the medical and psychological literature, and the state of the art is advancing rapidly. It is almost impossible for those involved in the specialties of psychiatry and psychology to collect and read this material, and it is well beyond the realm of practical expectation for the medical practitioner to be more than aware that this literature exists. It has become popular to refer to at least one branch of this literature as behavioral medicine.

Behavioral medicine can be thought of as the systematic application of applied behavioral analysis and behavior therapy techniques to medical problems. This involves the detailed analysis of psychological problems accompanying organic disease process, for example, stress in gastrointestinal disorders, or the subjective symptoms that accompany organic disturbances, such as the pain that occurs subsequent to a serious burn. As part of a detailed analysis of these types of behaviors, the therapist specifies in as great detail as possible the behavior under consideration or the symptoms to be modified and then implements a modification process based on principles derived from operant and respondent psychology.

The scope of problems considered by behavioral medicine is large and growing. Studies have ranged from interventions for particular organic illnesses, like decreasing the frequency of PVC's by biofeedback control of heart rate, to large-scale interventions in communities. The field includes studies on changing the pathophysiology of organic illnesses, reducing complaints from functional pain patients, reorganizing psychosomatic wards to include token economy principles, increasing percentage of clinic visits kept, humanizing treatment facilities, and changing entire communities through media combined with limited face-to-face intervention. More specific examples of some of the studies are as follows:

The use of a behavioral approach to treat an organic problem: A patient learns to use gastric acid biofeedback to reduce gastric acid secretions. The procedure can significantly reduce gastric acid secretion in some patients and has the potential of aiding those patients in whom gastric acid secretion is a major factor in peptic ulcer disease (18).

The use of applied behavioral analysis to increase attendance at regular clinic appointments: The therapist determines the baseline rate of missed clinic visits. He introduces various strategies while he monitors the frequency of kept appointments. By introducing strategies systematically, and then removing them, he determines that telephone prompts are a useful way to increase kept appointments (16).

The application of a multi-factor behavioral program in a community to reduce cardiovascular risk: Farquhar provided media only and media plus limited face-to-face intervention to three large towns. Both the media only and the media plus limited face-to-face intervention show decreased risk reduction compared to a control (see Volume 3 of this Handbook, Chapter 13.)

Although behavioral medicine procedures are derived from an experimental analysis of behavior, many of the programs incorporate both traditional medical and psychological therapeutic procedures. Purists would argue that such programs or packages are not strictly products of applied behavior analysis, and future work may indicate that many components of such programs and packages are unnecessary to bring about the desired change. At this stage of development of the field, however, there is a tendency or even eagerness to combine procedures. Some of these combinations have proved useful, for instance, Azrin combined a number of behavioral procedures to develop a package to teach rapid toilet training to children (2). Health care practitioners can use the package with little training from behavioral therapists (3). Ferguson describes a program originating in a series of studies using strict behavioral analysis procedures that incorporate behavior modification with family and individual therapy in the context of a therapeutic community to treat anorexia nervosa (see Volume 2 of this Handbook, Chapter 6.)

The types of observation used in behavioral medicine are quite varied. Unlike traditional pyschosomatic medicine practitioners who dealt largely

with the symptom on a symbolic level, the behavioral medicine therapist attends to physiological data, for example, blood pressure, temperature, and weight; psychological data, for example, behavior paterns as in Type A coronary prone behavior; emotional material, for example, anxiety in stress management programs; mathematical models, for example, in cost-effectiveness analysis; community system interactions, for example, in care delivery systems, societal priorities, and prioritizing health care needs; the macroanalysis of behavior patterns, for example, making recommendations for community-wide dietary changes; and finally, epidemiology which provides the tools for analyzing societal changes, much as the observation of discrete behaviors in a single case design allows one to determine the effectiveness of behavioral interventions in a single subject.

Medical versus psychological approaches to psychophysiological problems

How the mind and body interact to produce disease is an age-old problem. The answer for a particular age has been more often dictated by the prevalent technology of psychophysiology than by facts. In the 17th, 18th, and 19th centuries, when effective medical treatments were sparse, physicians concentrated on the emotional aspects of disease, and doctors became exquisite observers of behavioral pathology and consolers of the sick and dying. With the advent of modern medicine and effective techniques for diagnosis and treatment, the pendulum has swung to a highly scientific and technological approach to the mind and body: The "whole patient" is lost in the specifics of compartmentalized and highly specialized care. On the other hand, modern psychosomatic medicine has taken a different, almost antithetical course in this century, to medicine as a whole. Before the 1930's there were large numbers of studies undertaken to examine the relationship between emotions and disease, but there was no systematic undertaking to provide a theoretical or empirical basis for observations.

With Dunbar's publication of *Emotions and Body States* (5), which reviewed psychosomatic medicine from 1900 to 1930, the field came into its own. Between the 1930's and 1960's there were two basic trends: On the one hand the personality theorists, led by Franz Alexander and his colleagues, attempted to determine the relationship between personality and disease; on the other hand, laboratory investigators, led by Wolfe and his colleagues, studied the relationship between laboratory stresses and the development of physiological processes. The main tool of personality theory was psychoanalysis. Over the years, the basic assumption of the personality theorists that personality or specific conflicts are related to specific diseases was rarely substantiated and, more important, the therapy was not effec-

tive. The laboratory investigators' research became engulfed in a flood of new techniques and developments in related fields: It became obvious that most diseases are determined by a variety of factors and that the concept of physiological events induced by a single stimulus is insufficient to explain diseases. One example is provided in Taylor's chapter on hypertension (this volume, Chapter, 4), in which it is clear that many factors can induce high blood pressure. Thus, modern psychosomatic researchers need to incorporate findings from psychophysiology, psychoendocrinology, pathology, and medicine, and with the advent of behavioral therapies we have some additional, perhaps more effective tools for modifying disease-related behaviors.

The behavior therapist might approach a particular problem with a different set of assumptions than the traditional psychotherapist. For instance, when dealing with a patient with rheumatoid arthritis, it is not assumed that the patient is "passive dependent" and "masochistic" with "self-sacrificing traits." Rather, the patient's pain and disability are considered operants and partly contingent on the reward system of that particular person. The patient might be taught practical ways of self-control of his pain, and even self-management of his disease process. Neither of these procedures is incompatible with other treatment processes which may be necessary, for instance, antidepressants for severe depression or family therapy if the family unit exhibits dysfunctional behavior.

ETHICS

There continues to be some discussion of the ethics of applying a behavioral technology to medical or other problems and concern about the amount of control that the therapist exhibits over an ill individual's free expression of feeling, etc. Eloquent arguments have been presented both pro and con. In practical terms the issues are frequently rather obvious, especially if the data is carefully collected: A mother responds to a child's head-banging with cuddling and affection, thus reinforcing the behavior; when she is instructed not to respond, the child continues head-banging, a mild shock stops the behavior, and the mother can respond to the child with cuddling and affection for appropriate behavior. But even this is an extreme case. (The reader will note that aversive techniques are rarely used or advocated in the many programs and procedures that follow.)

Another example: A woman suffering from pain for six years and addicted to medication is told, following analysis of pain sheets which she has prepared, that she reports more pain when her husband comes home at night. He is instructed not to respond to her pain but to respond to what she has done during the day and to any positive health statements she might

make. The therapist systematically introduces new activites into her life. He does not inquire about her pain but notes that she has resumed many activities previously abandoned and has cut down on medication. Another patient is brought to the psychiatric ward in a wheelchair because she cannot walk, after having been thrown out of another hospital for being too demanding and a faker. After determining through careful medical examinations that the patient is able to walk, access to ward activities is made contingent on short walks, which are carefully reinforced and then shaped into more extended walks. She moans that her therapists are not sympathetic to her woes; but she starts walking. More self-care behaviors are reinforced and she has soon resumed many of her previous activities and is sent home. Six months later, having managed her diabetes better than for the previous three years (no return to hospital, less insulin, adherence to diet, and careful monitoring of her urine), engaging in activities she had previously abandoned and given up hope of ever assuming again, she is extremely grateful.

Many other cases can be reported for similar "successes." Often, the problems of a behavioral technique are less with the scientist than with the practitioner. All treatments should be delivered with complete compliance to medical ethics and respect for the patient. Furthermore, the so-called "basic clinical skills," like appropriate communication with the patient, establishing a working relationship, and many other unspecified behaviors of the "good clinician" are as important for the behavior therapists as for any other therapist. Some day we may be able to specify these behaviors more clearly, but they remain today a crucial part of good care.

The goal of behavioral techniques is similar in many ways to the therapeutic goals in medicine. The surgeon defines a specific target symptom, hypothesizes a pathology, opens the individual up with a knife, and removes or repairs the diseased part. The behavior therapist, too, is interested in a specific pathology, makes a discrete intervention, and, like the good surgeon, he does not ignore the rest of the patient.

The interventions are done for the "good of the patient," and although in books such as *A Clockwork Orange* it is hypothesized that this technology could be used to totally control an individual's behavior, in fact people do not do what they do not want to do even under rather extreme operant conditions.

There is a danger in behavioral medicine for the non-medically oriented behavior therapist who is not enamored of the disease model which is the basis of medicine. Unlike psychological syndromes where it is difficult to demonstrate the specific underlying pathology that traditionally would be the object of therapeutic intervention, with medical disorders there is usually a specific underlying physical pathology. For the medically uninitiated,

this distinction is extremely important. One only has to consider the use of operant techniques in rehabilitating an individual who has had a myocardial infarction to see the obvious limitations. For example, if one were to suggest and implement a program of *vigorous* exercise two weeks after a complicated myocardial infarction, there would be a high danger of precipitating another MI. Similarly, attempting to restore normal intellectual function in an individual with a rapidly progressive brain tumor may be frustrating for the patient and the therapist alike. Behavioral medicine will be most useful when individuals with special training in medicine and behavioral medicine work closely together.

METHODOLOGY

When investigating the interface between behavior and physiological disease, it is necessary to consider the type of data or dependent and independent variables that the experimenter or clinician will be confronted with when he attempts to determine whether or not an intervention has been effective. This data can be given a "hardness value." At one end of the hardness spectrum is an event such as a myocardial infarction, the definition of which is quite explicit, and the occurrence of which would be agreed upon by most observers. Other examples of this type of data are whether or not a child was enuretic in the night, whether or not a patient with anorexia nervosa gained weight in a specified interval, etc. On the other end of the data spectrum is the self-report of internal state. In this case an experimenter or clinician is dealing with information that is not directly observable by anyone other than the patient, and is totally beyond the realm of interobserver validation. Examples of this type of data are the feelings of anxiety, stress, and pain.

When discussing treatment or change, the first type of data allows direct measurement—the parameter in question either changed or didn't change. For example, if an individual is being treated for neurodermatitis, one could measure the square inches of excoriation on his forearm at each clinic visit; in studies of compliance, one could measure serum drug levels in seizure patients taking Dilantin; and if one wants to reduce cardiac risk by helping a patient reduce his serum cholesterol, this can be measured directly in a sample of blood. This type of outcome data provides the clinician with direct measurement of the effect of a therapy on a target symptom. However, it should be carefully noted that none of these "hard" data measurements is of a behavior, and to use them as a dependent measure of the effect of a behavioral treatment program is risky. This danger of *post hoc ergo propter hoc* logic can lead one to implement a treatment program

on the assumption that a behavioral treatment implemented prior to noticing a change in a physiological dependent variable was the cause of the change in that variable. In practice, such changes have multiple determinants, and the behavioral change program may be irrelevant or secondary to an unrelated, and usually unspecified effect such as therapist enthusiasm. A prime example of this is the behavioral treatment for obesity. Individuals treated with these techniques reliably lose weight. However, when one looks at their eating behaviors, it is *not* usually the case that they eat in an "obese style" prior to treatment or that their eating style is significantly changed by their therapeutic experience. Behavioral researchers often use the methodology of applied behavioral analysis to determine cause and effect in such situations. These procedures will be described shortly.

In discussing "hard data" it should be remembered that what appears "hard" is not always so. For instance, a person's blood pressure or the presence of a peptic ulcer would be considered hard data. But blood pressure measurement is subject to a wide variety of variables and is often inaccurate when more scrupulous methods are compared to "clinical methods." Pflanz (13) has found that radiologists agree on the presence of an ulcer in only 60% of films. In fact, psychosomatic medicine has been limited by the difficulties of establishing hard data on many diseases commonly accepted as readily diagnosable and in determining the criteria of hard data for making the diagnosis of that particular disease.

The second type of data comes from observation of behaviors. For this type of data collection, the behavior must be well defined and agreed on by all observers. For some medical problems, it is easy to specify a discrete problem behavior such as scratching. In other cases, however, it is difficult to specify an observable maladaptive behavior related to the pathological process, for example, in the treatment of hypertension, there are few externally verifiable behaviors. Examples of direct behavior measurement are the frequency of pain complaints of a patient as observed by the nursing staff, the amount of time in an interview spent stuttering, and the number of bites of food per day consumed by a patient with anorexia nervosa. Behavioral observation data can be collected in several ways. On an inpatient service or in an experimental setting, trained observers can be employed to monitor behaviors on a continuous basis, or an interval schedule can be used where observations are made for a specified period of time periodically through the day. An example of interval observation would be checking a child's pants for wetness during the first ten minutes of each hour, and recording the number of intervals in which wetness or dryness is present. This type of data can be collected by family members, for example, the wife observes a patient take his medication as prescribed and makes a note of

this behavior in a notebook, or parents can make a note of how many observation intervals per day contain an incident of scratching during the treatment of a neurodermatitis. The data may also be kept by the patient, for example, miles per day jogged in a cardiovascular program, or calories consumed in an obesity treatment program. The direct measurement of behavior to some extent avoids the logical error of equating behavioral processes with physiological change. However, it is not without pitfalls. For a physician, the resolution of the physiological problem is of prime importance. For example, with an obesity patient, the goal is amelioration of the symptom, and it is quite irrelevant to the physician (and usually to the patient) how this happens; whether the weight loss is from nonspecific variables or from the treatment program is academic. For the clinician to pay attention only to his "hard" observable data and ignore the goals of the physican and the patient would not be very helpful.

A third type of data comes from self-report. These data vary from a description of physical events—for example, a patient may report that he decreased his rate of eating consistently throughout the week or that he practiced relaxation twice a day as prescribed—to a description of his internal events, like feeling and moods. Although various devices can be employed to make cheating less likely, for example, self-report diaries, graphing of daily results, etc., ultimately the report depends on the patient's veracity. It must be remembered that self-report alone leads to change in the frequency of many behaviors independent of other interventions that might be used. Many of the more innovative techniques used in behavioral medicine have focused on direct observation of data that had previously been considered in the bailiwick of self-report. For instance, by building a device into a tape recorder to note when the tape recorder is turned on and off, it becomes possible to determine how often a patient has turned on a relaxation tape.

The final type of data, and the second type of self-report, concerns the self-report of internal events. Behaviorists are most critical of the usefulness of self-report of internal states, since there is no way to verify the accuracy of the report. But as Skinner observed, verbal behavior is as meaningful as any other behavior and subject to the same principles of behavioral analysis. The conditions of self-report are frequently very important determinants of the self-report: For instance, a pain patient addicted to Talwin may give a very different pain report to his emergency room doctors than to physicians on the ward who have made it clear that reports of increased pain will not be rewarded with medication. Often a behavioral consultant is asked to change thoughts and attitudes concerning health care behaviors. For example, a juvenile diabetic may need counseling to help him improve his attitude about injecting himself with insulin and maintaining a steady

level of blood sugar. It is impossible to monitor attitude directly. Interviews and paper-and-pencil tests can be used to question the patient, and these usually show that attitude does change after instruction, or at least the patient is aware of the attitude he should have after instruction and complies with instructions to report the proper attitudes. This in turn may or may not correlate with a change in health-related behavior. A third type of self-report data describes feelings. In this case also, there is nothing the experimenter can observe to corroborate the patient's self-report. For example, in a stress reduction program it might be desirable to decrease the amount of time an individual feels anxious, and multiple interventions could be introduced towards this end. However, the demand characteristic of the experimental situation may be such that the patient continues to feel anxious but tells the experimenter, because he is a friendly guy, that he feels better and less anxious.

Community applications of behavior medicine techniques generally rely on data of the first class mentioned above: Observable and directly measurable events or states rather than behaviors. For example, in the Stanford Three-City Study, the measures included periodic checks of patient blood pressure, cigarette consumption, cholesterol consumption, and weight reduction. Although an aggressive behavior modification program was provided to the communities, it is only by inference that one can say that these pro-health behaviors increased as a result of specific elements of the program.

A variety of types of observation have been developed along with a small technology which aids the experimenter and clinician in monitoring progress. The simplest type of observation relates to the first type of data, measuring the symptom on a periodic basis when the client comes for a medical checkup. Familiar examples of this are taking blood pressures, weighing patients, measuring the amount of medication in blood, etc. This type of observation is symptom and clinically oriented, and may not relate directly or simply to the behaviors being modified. In observing behaviors, two basic schemata have been used: Interval and frequency observations. In interval observation, an observer or team of observers periodically spends a fixed amount of time in the proximity of the patient and indicates whether or not a behavior has occurred and/or the frequency of occurrence of the behavior during that period of observation. This type of behavioral observation can be carried out by the patient's family or the patient himself.

A number of aids have been developed to help the observer measuring behaviors. One of the most popular measuring devices is a golf counter which can be worn like a wristwatch. With this device, one can record frequency data in an unobstrusive way, either as an observer or as a patient observing his own behavior. Timers of many kinds have been employed

both as cueing devices and as aids to interval observation. A wide variety of forms have been developed to help individuals monitor their own behavior. Usually these include the time, a description of the behavior, its magnitude, and the environmental determinants (antecedents and consequences) of the behavior in question. Similar observation forms can be used by external observers, for example, nursing personnel on a pain unit. In this case a specified pain behavior such as demanding medication will be entered with the time of its occurrence, the antecedents to its occurrence, and the consequences, for example a shot of Demerol. Audio- and video-tape recordings have been used extensively in the observation of behavior. These tools provide perhaps the ultimate way of quantifying the frequency of behaviors under observation. However, they produce an enormous amount of data for which detailed analysis is often extremely time consuming and expensive. Other technological devices have appeared in the literature which range from small toilets for children that record the frequency of urination and reward the child, to sophisticated piezo-electric activity monitors which record one week of global bodily activity on an ultraminiaturized tape recorder worn like a wristwatch by a patient.

When one applies behavior therapy techniques to any medical problem, one is under a series of constraints not present in a pure experimental situation. The type of data collected will depend upon the resources available. In an outpatient clinic it may be impossible to observe behaviors. The behavior may be unspecifiable as in anxiety, or a clinical symptom such as square inches of excoriation which improves with treatment. In a research setting, it may be more interesting to make a fine grain analysis of problem behaviors such as caloric or bite intake with anorectic patients, and more closely selected dependent variables.

REVIEW OF BEHAVIORAL THEORY AND PRINCIPLE

In analyzing the role of the behavior-oriented therapist, Goldfried and Pomeranz (6) conceptualized their approach in terms of four types of variables: a) Antecedent stimulus variables which may have elicited or set the stage for the maladaptive behavior or symptom; b) Organismic variables whether of a psychological or physiological nature; c) The overt maladaptive behavior itself; d) The consequent changes in the environmental situation including the reaction of others to the maladaptive behavioral pattern. The therapist attempts to intervene at one or more of these stages and make therapeutic behavioral changes. The tools available to him come from a large body of experimental literature. These interventions can be classified as to the time of their application in relationship to the problem behavior: As antecedents to the behavior or consequences of the behavior.

Behavioral Antecedents

Respondent conditioning

Early in the days of psychology, Pavlov and others discovered a series of learning contingencies generally known as respondent or classical conditioning. They observed that many environmental stimuli such as food lead regularly to apparently inborn autonomic responses such as salivation. They termed the former an unconditioned stimulus (UCS) and the latter an unconditioned response (UCR). They then observed that if a formerly neutral object or event, for example, the sound of a buzzer, was presented to the animal at the same time or prior to the time when it was fed (presented with the UCS), it would elicit the same response, salivation. In this case the sound was termed a conditioned stimulus (CS) because it had taken on new meaning for the animal by pairing it with a stimulus that would normally produce an autonomic response. The response to this conditioned stimulus was then known as a conditioned response (CR); its appearance was conditional upon the presentation of the conditioned stimulus. Pavlov also demonstrated that the CS would generalize to stimuli of the same or similar properties, treatment settings, and other factors.

This type of learning is important because of the large amount of literature showing that many autonomic functions can be controlled or modified through respondent conditioning. An experimental demonstration of the classical conditioning of a physiologic variable was carried out by Whitehead et al. (17). The presentation of a conditioned stimulus (the sound of a buzzer) was followed by tilting a table on which the patient was resting to lower blood pressure. They found that after a few pairings of this type, the buzzer alone came to elicit small but consistent decreases in systolic blood pressure without tilting the table. This type of learning paradigm has been used extensively to explain the acquisition of psychosomatic symptoms of a wide variety. Although it is an appealing formulation of these problems, in many cases the experimental evidence to substantiate this view is lacking. In treatment paradigms, respondent conditioning has been used primarily to eliminate unwanted behaviors. For example, Kantorovich (9) paired the taste of alcohol with nausea and vomiting caused by an injection of apomorphine. After a few pairings, the taste and smell of an alcoholic beverage was sufficient to cause the autonomic response of nausea and in some cases vomiting. This was proposed by him and many others as a definitive treatment for alcoholism (See Volume 3 of this Handbook.). When a conditioned response has been produced, it is possible to eliminate this response through a process known as *extinction.* If an organism that is used to being fed after the sounding of a buzzer has been conditioned to salivate at the

sound of the buzzer, its salivatory response can be extinguished by presenting the conditioned stimulus without the undonditioned stimulus over a period of time.

Discriminative stimuli

In the environment there are many stimuli that signal to the organism that a certain set of contingencies is in play. For example, a stoplight signals to the driver that at the intersection there is a rule which states that if the light is red, one may receive a traffic ticket for driving through the intersection without stopping. The light in itself has no reinforcing properties, it is solely a means of informing the driver of a set of contingencies. In a medical setting, the presence on the ward of a friendly sympathetic nurse may signal to a patient that if he engages in pain behaviors, his efforts may be rewarded with a shot of morphine. The presence of other nurses may signal a different set of contingencies and result in less of this type of behavior being emitted. Metcalf (12) reports an asthmatic patient who monitored his attacks for 85 continuous days. The patient noticed that of the 15 days when he had asthmatic attacks, 9 occurred while visiting his mother's home. Talk about mother, projective tests, photographs of mother, etc., would not provoke an attack, but visiting with the mother reliably precipitated such attacks. The mother's presence then became the discriminative stimulus for asthmatic attacks in this patient. Similar types of discriminative stimuli are seen in many patients with eating disorders where an environmental event such as watching the six o'clock news signals to the patient that it is the time to emit hungry behaviors which will be reinforced by feeding.

The control of discriminative stimuli over behaviors can be modified by several procedures. The simplest of these is *environmental engineering:* If a discriminative stimulus is identified and can be eliminated from the environment, the consequent behavior might not occur. The patient can be instructed not to engage in the behavior governed by the discriminative stimulus, for example, he can be instructed that pain behaviors will not be attended to by the nurse who formerly reinforced these behaviors. Over a sufficient length of time, the discriminative stimulus will no longer designate the former set of contingencies, and the behavioral response to the environmental cue will be extinguished.

Consequences of Behavior

Positive reinforcement

An event is considered to be a reinforcer of a previous event if the frequency of occurrence of the previous event increases as a result of the con-

tingent application of the reinforcement. Many environmental stimuli are natural reinforcers, for example, food, water, sexual activity, and warmth. Other activities are social or symbolic reinforcers, such as attention, praise, money, and diamonds. When considering reinforcers, it is necessary to bear in mind that reinforcers are idiosyncratic, that is, what is reinforcing to one person may not be reinforcing to another. An example of this would be attempting to reinforce jogging behavior with cigarettes in an individual if the individual did not smoke. The reinforcer would have little power or meaning to the jogger. Likewise, using money as a reinforcer for a retarded child may not work if the child does not know the value of money.

Negative reinforcement

Negative reinforcement is defined as the contingent removal of an aversive stimulus which increases the frequency of the preceding behavior. For example, scratching will temporarily terminate an aversive stimulus, itching, and the termination of this stimulus increases the probability that the behavior, scratching, will be repeated. The cessation of itching is a negative reinforcer of the scratching behavior. Negatively reinforced behavior is often very difficult to extinguish.

Shaping

In some therapeutic occasions, it is desirable to establish a rather complex or extended repertoire of behavior. In this case a procedure known as shaping is used. During shaping, reinforcement is given for behaviors that *successively approximate* the therapeutic goal. Each time the organism makes a response that is closer to the desired behavior than a previous response, he is reinforced. For example, when one wishes to have a client engage in a jogging program, initially social reinforcement may be dispensed for walking a fixed distance, then for walking and jogging, for jogging alone, and then for jogging greater distances. The eventual behavior of jogging three miles a day may be thus shaped over a period of time by reinforcing successful completion of increasing performance demands.

Self-Reinforcement

Under some circumstances, reinforcement can be applied by the patient himself. With the use of self-reinforcement, the reward can be a tangible object such as a cigarette, or a covert mental reinforcement such as saying,

"I'm a good person." The former procedure has been investigated by Homme (8) in connection with relieving the symptoms of depression by reinforcing positive self-statements after cueing one's self by smoking a cigarette. The latter covert reinforcement is best shown by Mahoney's attempts to alter attitudes towards eating, and praising one's self covertly for successful performance (10).

Extinction

As with classical conditioning, operant conditioning often involves not only acquiring a behavioral repertoire, but also unlearning an undesirable behavior. Extinction is a process of systematically *not* reinforcing a maladaptive behavior. If the behavior is under the control of the reinforcement, by not allowing this reinforcement to occur, the behavior will weaken and vanish with time. For example, when one wishes to extinguish the behavioral response of eating in the presence of a cue such as television, the client is instructed to not reinforce the urge to eat in front of the television with food. The urge to eat gradually extinguishes over a several-month period until the television returns to the status of a neutral stimulus.

Aversive procedures

Several types of aversive stimulation can be used to alter behavior. The most commonly used but possibly least effective intervention known is punishment. In this case, an aversive stimulus is administered after the performance of the target behavior. The effect of this learning paradigm is to suppress the occurrence of the problem behavior, and often to train the patient to avoid the punishing situation and the punishing individual. Occasionally it will engender fear responses that generalize from beyond the clinical situation to other similar classes of individuals such as all doctors, or all health-giving institutions.

Escape learning and avoidance training

Escape learning and avoidance training are two additional aversive procedures that are effective in reducing the frequency of an unwanted behavior. In escape training, the individual can terminate an ongoing aversive stimulus by performing a specified behavior. In an avoidance training paradigm, the individual learns that if he engages in a defined behavior as an

alternative to an unwanted behavior, he will avoid the occurrence of an unpleasant stimulus. Once he has learned the new behavior, the aversive stimulus can be discontinued with little effect. He will continue to avoid indefinitely punishment that is now nonexistent, because on no occasion will he test the reality to see if the contingencies are still in play. Parenthetically, it is thought that many types of avoidance behaviors such as hand-washing compulsions may have their origin in this type of learning. Avoidance learning is the basis of much "superstitious" behavior. A patinet may take Vitamin C to prevent a cold and attribute the absence of colds to the Vitamin C. Every day he feels well reinforces the pill taking.

APPLIED BEHAVIORAL ANALYSIS

Common to many behavioral medicine studies is the use of applied behavioral analysis to determine if a particular treatment is causing a response. Applied behavioral analysis refers to the use of the experimental analysis of behavior, particularly as developed in laboratory investigations of operant behavior applied to socially or medically important behaviors. To fullfill the standards of design for such applied behavioral analysis, a study should employ an intrasubject replication design of some kind (e.g., reversal, multiple baseline, multiple schedule) and there should be a clear description of the reliability of the behavioral observations. The most popular intrasubject design is the so-called single case study. The work by Agras et. al. (1) on the factors influencing eating behaviors in anorexia nervosa patients illustrates the use of this design.

In this experiment, they were interested in the effect of reinforcement on caloric intake and consequently weight gain in patients with anorexia nervosa. Figure 1 demonstrates their experimental paradigm. During the baseline, *ad lib* eating was allowed in an inpatient setting, and both caloric intake and weight were measured daily. During the reinforcement phase, the patient was reinforced by allowing privileges such as radio, television, playing games with a nurse, etc. for gaining at least 0.1 kg per day. When privileges were made noncontingent, caloric intake sharply decreased and the rate of weight gain declined. Reinstitution of reinforcement contingencies caused the patients to resume their caloric intake and weight gain. It is notable that in this experiment negative reinforcement was controlled by contracting with the patient when he entered the hospital that he would stay for twelve weeks "for research purposes whether or not the patient gained weight."

This study also used a *reversal*, a procedure in which the supposed active intervention (the reinforcement of caloric intake) is removed while the tar-

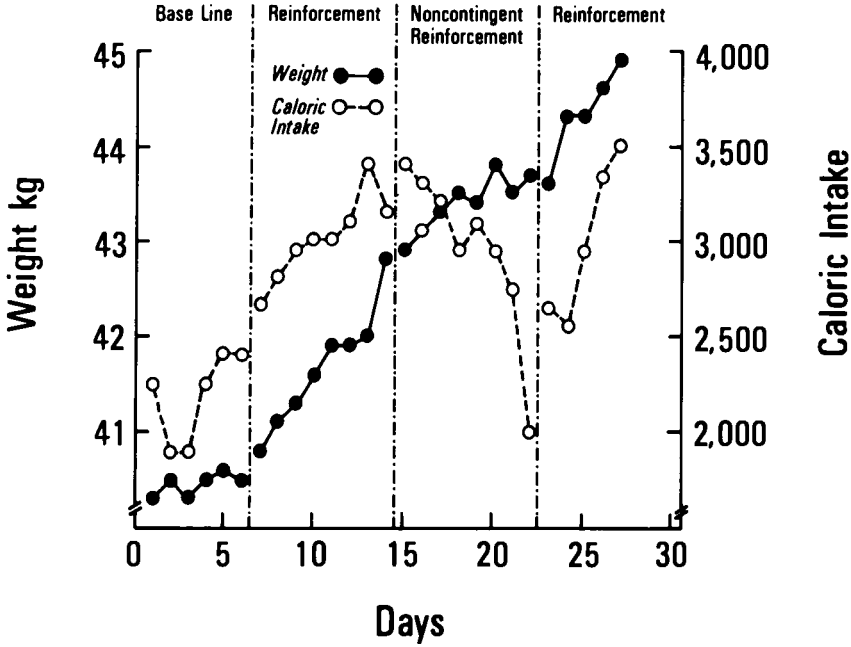

Figure 1: Weight gain and caloric intake in a patient with anorexia nervosa during a thirty day treatment period with varying reinforcement contingencies. (From Agras, W.S. et al., *Arch. Gen. Psychiat. 30*: 279-286, 1974.)

get behavior continues to be measured. It is assumed that if the behavior returns to baseline during reversal, the intervention was actively affecting the rate of the behavior in this case. Other authors have used *multiple baselines* to demonstrate the effects of a particular therapy. In this procedure interventions are introduced sequentially over time, and the results of the intervention should speak for themselves (especially because such procedures do not lend themselves to statistical analysis). Figure 2 illustrates the use of a multiple baseline design (15).

Group outcome studies are often useful in demonstrating the effectiveness of a particular therapy but are not the type of design which permit cause and effect determinations. If an intervention is introduced in a group, then removed and reintroduced, in the so-called ABAB design, cause and effect can be demonstrated. However, group outcome studies remain the most important design to demonstrate that a therapy has a specific effect as compared to the many nonspecific variables that can affect the rate of a particular behavior.

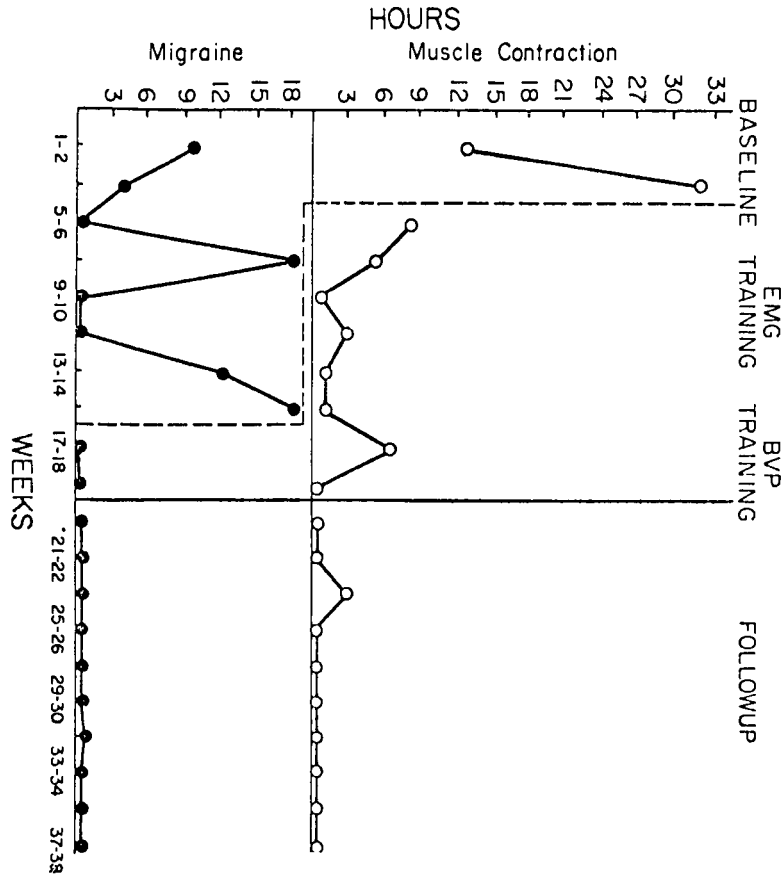

Figure 2: Combined migrane-muscle contraction headaches using BVP and EMG biofeedback (From Sturgis, E.T. et al., *TABA 11*:215-224, 1978.)

BEHAVIOR THERAPIES

We mentioned earlier that the use of applied behavior analysis procedures is only one way in which studies are classified as behavioral medicine. The other is the use of a behavior therapy technique supposedly derived from applied behavior analysis. Although there is considerable controversy about which techniques should be considered behavior therapy techniques, the following techniques, with a brief description of their use, are commonly considered behavior therapy techniques and will be referred to frequently in this text.

(a) *Relaxation* is a procedure aimed at achieving muscle and mental relaxation. Developed by Jacobsen in the 1930's and popularized by Wolpe for use with systematic desensitization, the procedure has entered behavior therapy in its own right. Relaxation has been used to treat most conditions in which anxiety appears to play an important role in the etiology, maintenance, or worsening of the condition. The most striking studies have centered on the usefulness of relaxation in reducing high blood pressure (See this volume, Chapter 4). Less certain, but promising are the effects of relaxation in tension headaches (See Volume 2 of this Handbook, Chapter 12.) insomnia, (this volume, Chapter 11) and perhaps asthma (Volume 2 of this Handbook, Chapter 1).

(b) *Systematic desensitization* is a procedure developed by Wolpe that uses anxiety reduction techniques (like relaxation) paired with anxiety provoking stimuli to reduce anxiety. Although systematic desensitization itself is rarely used with the various procedures mentioned in this book, many of the techniques are derived from systematic desensitization. However, many individual case reports using systematic desensitization continue to appear in the literature.

(c) *Token economies* are a special application of reinforcement and extinction procedures in which tokens, which can be exchanged for a variety of other reinforcers, are used to change various behaviors. For instance, the use of token economies on a psychosomatic unit was recently described by Wooley et al. (19).

(d) *Respondent conditioning* procedures include extinction and stimulus control conditions like generalization, discrimination, and fading. Examples of these procedures applied to medical problems are found throughout these volumes.

(e) *Operant conditioning* procedures, described previously, include shaping.

(f) *Aversive conditioning* procedures, also described previously, are mentioned throughout the book.

(g) *Implosion and flooding* are special techniques used to expose phobic subjects to a feared situation without relaxation.

(h) *Assertion training* includes a variety of treatment procedures to increase social or assertive behavior. There has not been wide application of this procedure to behavioral medicine except as it may be included in a treatment package. For instance, assertion training is frequently included in an anorexia nervosa treatment program.

(i) *Modeling* is a technique in which the therapist or a confederate models appropriate behavior for the client. Modeling of pain responses to injections was used, for instance, to prepare children for injections and has been shown to decrease postsurgical fear in children (11).

(j) *Feedback, including self-monitoring,* involves both therapist feedback of progress of observational data and biofeedback of physiological functions. Several studies have shown that feedback about progress is vital to obtaining behavioral change and maintaining change. This may occur from an individual monitoring his own behavior, having feedback provided by a therapist, or mechano-electrical device or having physiological information fed back to him (biofeedback).

(1) *Self-monitoring* has a certain active treatment effect itself. For example, in obesity control programs it has been found that keeping a food diary with intensive feedback about when, where, and under what circumstances the individual eats is an effective therapeutic tool in decreasing food intake.

(2) *Therapist feedback* is also important both as an active therapeutic component itself or as part of a package. For example, Agras et al. showed that with patients who have anorexia nervosa, positive reinforcement was without effect if the client was unaware that the reinforcement contingencies had been met (1).

(3) *Biofeedback* uses electronic monitoring of autonomic functions to provide feedback about basal states and change. A great deal of research has shown that with this type of monitoring, physiological systems that are normally not accessible to awareness can be controlled by cognitive strategies. For example, finger temperature with appropriate electronic feedback can be raised and lowered by clients when the behavior change is operantly reinforced. Many examples of biofeedback are provided throughout this book.

(k) *Instructions,* including therapist instructions and self-instructions, are important behavior change techniques.

(1) *Self-instruction* is a cognitive technology for investigating self-defeating instruction and replacing it with more self-supportive internal dialogue. Although applications of this technology are just beginning,

there is some evidence that this strategy is useful in the treatment of obesity, and possibly in stress management programs (see this volume, Chapter 1).

(2) *Therapist instruction.* In most behavioral paradigms, instruction plays an important part in setting the stage for change. Many studies have demonstrated that subjects unaware of contingencies, or the reasons or necessity for change, do not respond as well to reinforcement as do individuals instructed in the aspects of a program. In health care systems, basic elements of medical knowledge such as nutrition or the desirability of preventing hypertension, set the stage or the rationale for the client to engage in health-seeking behavior. The importance of therapist instruction is illustrated in Redmond's study (14). They found that subjects instructed to raise and lower blood pressure did as well as those given brief training in progressive relaxation or given biofeedback of their blood pressure.

(l) Cognitive behavioral approaches include cognitive instructions, corrections of magical thoughts; correcting maladaptive self-statements, which occur in a variety of settings; and contingency conditions. This area is reviewed by Mahoney in Volume 3, Chapter 5. Other cognitive therapies are logotherapy; paradoxical intention; and the self-management techniques of self-monitoring, self-cuing, self-reinforcement, and self-punishment.

THE FUTURE OF BEHAVIORAL MEDICINE

Behavioral medicine has a bright future. New measurement and evaluation techniques will continue to be integrated with behavioral observations to enhance our basic knowledge of the importance of psychological factors in the development, maintenance, and treatment of disease. In speculating on the future direction of behavioral medicine, we suspect that the preliminary observations reported by our contributors of the effectiveness of treatments will be pursued and examined. We will learn more about what constitutes a specific treatment and the limitations and usefulness of specific treatments. Questions of generalization and maintenance, desperately in need of attention from researchers, will be pursued more vigorously.

The use of behavioral techniques will extend into more areas of medicine. A few studies have already shown that techniques can be developed in laboratory and clinical research settings and transferred to health care practitioners who have no particular expertise in behavior therapy. One specific consultation/liaison program is discussed in Volume 2, Chapter 10. Other models will be developed and behavior therapists will play a greater role in the management of patients on the ward.

In an outpatient setting, the applications of behavioral medicine are numerous. Most physicians in their daily practice are confronted with patients with a wide variety of "psychosomatic" symptoms which are often treated by medication, for example, Diazepam. Rather than a prescription for a minor tranquilizer for the housewife who is moderately stressed, or an endless flow of Maalox for the patient with chronic gastritis, an analysis of the patient's behaviors, often in the form of a self-report diary, will help pinpoint the determinants of the symptomatology and allow the physician or consultant to prescribe a treatment program.

One of the areas of greatest potential application of behavior therapy techniques is in preventive medicine. It has been argued that most of the decrease in mortality in the past 50 years can be attributed not to medical intervention, but to disease prevention. Such firmly entrenched customs as washing one's hands, the use of sanitary facilities, and the refrigeration of foods, have had a marked effect on morbidity and mortality.

At the present time behavior therapy techniques are being applied to primary intervention programs which are designed to prevent the development of diseases. Farquhar and others report in their chapter (Volume 3 of this Handbook, Chapter 13) on the use of systematic intervention on a community-wide basis to effect cholesterol consumption, cigarette consumption, obesity, and blood pressure. On an individual basis, these techniques have been successfully used to reduce serum cholesterol, and blood pressure.

Based on the widespread acceptance and practical application of behavior therapy techniques in medical settings, one can speculate that behavioral medicine as a subspecialty will become increasingly important in the future. The determinants of this importance will lie in being able to offer the medical practitioner an effective intervention strategy for his patient. To facilitate this, much research is needed to look at the determinants of "ill health" behavior, and the specific strategies that appear to be useful in modifying or eliminating this type of behavior. The state of the art currently in most areas is clinical case description. In a few well-circumscribed areas such as anorexia nervosa and obesity, detailed investigative work has determined many of the antecedents and consequences responsible for the behaviors in question. Therapeutic programs have been implemented and replicated repeatedly. However, even here the work is not done. Ineffective treatments are still included in the therapeutic packages, and additional therapeutic variables, as yet unspecified, clearly influence the outcome.

REASON FOR THIS TEXT

We had three goals in mind in preparing this text: (a) To disseminate information on behavior medicine techniques, ideas, and findings to a wide

variety of disciplines; (b) to provide previously unpublished research data and theoretical discussions for practitioners and researchers; and (c) to provide a forum for discussion of speculative ideas in a format that will be periodically updated. To achieve these goals, we have included basic research and physiological information that may be of more interest to the academician or researcher and also as much clinically useful information as possible so that practitioners might pick up this book and use to to help them treat their patients. In the introduction to the various chapters we provide a guideline to the type of material and the direction of reading that individuals may take. Some readers may not find the physiological section of a chapter interesting, but may learn practical techniques in other sections. Where such dissections can be made, we indicate them in the introduction. In many cases we have asked authors not only to publish their research findings, but to speculate from them. In this way, we hope to provide a forum for discussion of ideas, a place for cross-fertilization that it is hoped will bear fruit in future editions of this text.

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The Comprehensive Handbook of BEHAVIORAL MEDICINE

Volume 1: Systems Intervention

The Comprehensive Handbook of Behavioral Medicine, Volume I

Introduction To The Cardiovascular System

When Miller and Dicara reported that blood pressure and heart rate could be influenced by instrumental, as well as classical conditioning procedures, the door seemed opened to a new era of psychosomatic research (1). The implication from their research was clear: Environmental events could directly influence physiological process, and the problem of how external events could influence internal process was opened anew. A flood of early studies sustained the general notion that external events, including feedback, could produce statistical effects, but the magnitude of change was generally small. This led Blanchard and Young, in 1973, to describe the self-control of cardiac functioning as "a promise as yet unfulfilled." (2) Over the next few years, however, other studies have produced more encouraging results. In the next review, Blanchard and Miller (3) note that biofeedback of blood pressure, EMG or GSR, and relaxation techniques all seem useful for blood pressure reduction; that heart-rate biofeedback has shown some utility in treating PVCs and sinus tachycardia, and temperature feedback seems helpful with some peripheral vasoconstrictive disorders. In this section, we present chapters which review the contributions of behavioral medicine to five main areas relevant to cardiovascular functioning: Pattern A Behavior, Raynaud's Disease, Arrhythmias, Hypertension, and Cardiac Rehabilitation.

The first chapter in this section reviews research into the defining characteristics and possible implications of the "Type A" personality. Many studies undertaken, mostly by Roseman and Friedman, have suggested that Type A personality should be considered a major risk factor in the development of cardiovascular disease. The notion of a behavior pattern associated with cardiovascular risk has great appeal to behavioral researchers; the behavior can be quantified; baseline measures can be obtained; and interven-

tions can be demonstrated. Suinn begins Chapter 1 with a review of studies on the relationship of stress to cardiovascular disease. Epidemiological prospective studies have suggested that stress may be related to the development of acute and chronic cardiovascular disease-other physiological studies have related stress-induced change in serum cholesterol and catecholamines as possible mechanisms for these changes. Furthermore, several recent studies have demonstrated physiological patterns for Type A behavior patterns which could lead to cardiovascular disease. However, the existence of a specific coronary behavior pattern and, if it does exist, its importance as a cardiovascular risk, is far from universally accepted. Suinn provides a scholarly discussion of these issues followed by a description of the behavioral interactions which have been used to modify Type A behavior, and the stress presumably related to this behavior.

One particular type of cardiovascular disease that has been the focus of many studies in Raynaud's disease, or episodic digital asphyxia of distal extremities. Conners reviews the physiology of peripheral vasoconstriction and the techniques which have been used to increase and decrease peripheral vasoconstriction in Chapter 2. As he notes, "The most impressive aspect of several similar dealings with these variables is the wide range of individual variation in responsiveness." He argues for assessment procedures which can account for individual physiological responsivity and instructed variables.

In the next chapter, Feuerstein and Ward provide an overview of the effects of psychological treatment for arrhythmias. Several recent reviews (4,5) have demonstrated that psychological factors influence arrhythmias. The sympathetic nervous sytem seems to play an important role in the development of such arrhythmias, but, as the authors note, our knowledge about the usefulness of behavioral interventions is still very tentative and has not moved much beyond the single case study stage. They point out that reduction of heart rate is the most promising biofeedback treatment for reducing PVCs, and that relaxation training also appears useful. Behavioral studies on arrhythmias have focused on methods of treating impulse conduction, and have neglected to study coping strategies and similar techniques to help individuals deal with life-stress problems which may precipitate these disorders.

Taylor, in Chapter 4, reviews one of the areas of behavioral medicine most solidly based on well-controlled experimental data: the behavioral contributions to the understanding and treatment of high blood pressure. This area had been more extensively studied than most, and the studies indicate that some behavioral procedures may be useful as adjunct treatments for high blood pressure. Taylor also explores the process by which phar-

macological agents become incorporated into medical practice and the implication this has for behavioral medicine researchers.

The next chapter concerns an extremely important area of cardiovascular disease that has not achieved sufficient attention from behavioral researchers. Despite minor trends in a downward direction, myocardial infarction remains the most common disabling disease in our country. Psychological interventions have not kept pace with medical knowledge: Between 10 and 50 percent of patients having suffered an MI do not return to work, despite medical evidence that their hearts are functioning at near-normal physiological levels. So, we have included Chapter 5 by DeBusk, which reviews many of the recent medical contributions to cardiac rehabilitation. As he notes, psychological, and not medical problems, are responsible for many patients' not returning to work—it behooves behavioral researchers to develop programs to maximize return to work as one major outcome variable. The accurate assessment of clinical functioning has potentially revolutionized this area of study. Until recent technological advances have permitted an accurate assessment of the patient's myocardial function, return to work was largely left to the idiosyncrasies of the physician in charge of a patient's care. For instance, three months became the standard time when people should start to think of returning to work; even though data suggests that the heart had largely healed by two months. Ten years ago in medical school, we were instructed not to give postmyocardial infarction patients rectal exams or to disturb them for fear of precipitating an attack. We were also told to keep them at strict bedrest. Even newer techniques which will provide direct visualization of the heart's function, like TEC scans, will provide more direct descriptions of the heart's function in postmyocardial infarction patients. It will also be possible to accurately characterize a patient's medical status and to look more closely at the psychological factors in his care.

Another area of major research in cardiovascular disease, risk factor intervention, can be found in other sections of this handbook: Pechacek and McAlister discuss smoking programs in Volume 3, Chapter 11; Hagen reviews the obesity literature in Volume 2, Chapter 3; Dunbar and Agras discuss compliance in Volume 3, Chapter 6; and Nash and Farquhar describe the Stanford Three Community Study in Volume 3, Chapter 13.

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The Comprehensive Handbook of Behavioral Medicine, Volume I

CHAPTER 1

Pattern A Behaviors and Heart Disease: Intervention Approaches

Richard M. Suinn

By far the major cause of death in the United States is cardiovascular disease. The estimate by the National Heart, Lung and Blood Institute in 1975 was that 1.3 million Americans would experience some aspect of coronary heart disease, with about 675,000 dying from it. Heart disease has another major impact in that about 26% of those dying will be under the age of 65. Thus, it becomes a substantial economic and social problem by taking away persons during their productive years. Heart disease across nations is highest among Americans, to the extent that it has sometimes been considered as reaching epidemic proportions. As a result of studies such as the Framingham Heart Disease Epidemiology Study, sponsored by the National Heart and Lung Institute (12), a number of factors have been implicated in the etiology of heart disease. Among the primary risk factors are: Elevated serum cholesterol, involving levels 250–275 mg per 100 ml or greater; obesity; hypertension, involving systolic and diastolic blood pressure over 160/95 mm Hg; excessive cigarette smoking, involving 20 cigarettes or more daily; diabetes mellitus; and lack of exercise. The presence of any one factor increases the risk of a heart attack, and adding further factors increases the chances measurably. For example, any combination of three of the factors can increase the risk up to 30 times. Other cited risk factors include age, genetic factors, dietary intake of animal fats and cholesterol, and the presence of some specific diseases. At the same time, it is recognized that these traditional risk factors still do not account for all of the variables associated with heart disease. Jenkins (32), in a review of the literature, suggests that

the best combination of these risk factors would still fail to identify most new cases. Using the combination of two or more such risk factors in a predictive formula does identify persons of high risk; however, such factors still predict only approximately half the incidence of new occurrences (25, 39). Hence, the search for other factors, such as personality variables, has been initiated.

The hope that specific personality types would somehow be identified as predisposed toward certain physical diseases has never been supported by research data. Dunbar (14) believed that personality traits acted as predisposing factors to coronary occlusion and angina. The former type of patient was characterized as being superficially calm, self-sufficient, suppressing of emotions, authoritarian, and in conflict over expressing hostility. In spite of such speculation, little research to date has confirmed any reliable relationship between personality clusters and incidence of physical disease. In comprehensive reviews of the literature, Jenkins (32, 33) found few valid associations between specific personality traits and disease processes; however, some suggestive factors other than personality constructs did appear promising. These two are psychological stress levels and the Type A coronary-prone behavioral pattern. These findings will be discussed next.

Stress may be defined as the state of a person faced with psychological or physical threat. It may be understood as involving stressors (i.e., stimuli prompting stress, such as a divorce or an imminent work deadline), and as measurable through self-report data, assessment of cognitions, observation of behaviors, or physiological measurements. Anxiety questionnaires are examples of the first type of data, while changes in heart rate may be examples of the last. Changes in such measures of stress may mean that stressors have been removed or that the subject is no longer reacting to the presence of these stressors. The evidence of the association between stress and coronary disease exists in two forms: Data relating stress to disease outcomes and data relating stress to specific physiological consequences. With respect to the former, a number of studies suggest that the risk or outcomes of coronary disease may be related to stressors such as life events. Job frustrations and economic difficulties seem to enhance the risk of heart disease. For example, House (29) reports that excessive responsibilities, overtime assignments, and occupational demands precipitate the development of coronary disease. Sales and House (60) found that occupational groups with greater measured job dissatisfactions showed consistently greater mortality rates from heart disease than others in the same occupations. Frustrations about status were found to be an important source of dissatisfactions. Kits van Heijningen, and Treurniet (40) studied workers in the Netherlands, finding that cases of infarction tended to be preceded by loss of prestige in work as-

signments. Similarly, Bruhn, McGrady, and duPlessis (4) interviewed 64 patients in Oklahoma and discovered that those with coronary disease reported lack of recognition by their superiors as compared with matched controls without heart disease.

Stressors of life dissatisfactions can come from sources other than the occupational environment. In a report on 32 pairs of Swedish twins discordant for heart disease, Liljefors and Rahe (41) found the affected twin experiencing greater unhappiness over working conditions, level of education, childhood experiences, and adult personal relationships. Both Blohmke, Schaefer, Abel et al. (3), and Bruhn, Wolf, Lynn et al. (5) discovered family and marital tensions among coronary patients. Since nearly all of these studies are retrospective, the interpretations should be viewed conservatively. Although they all suggest that life stressors as reflected in dissatisfactions contribute to heart disease, it is alternately possible that coronary patients have a lower tolerance to stressors and become dissatisfied more readily. One means of answering this problem of interpretation is to measure objectively the level of stress represented by various life events, and to study the relationship between such events and heart disease, independent of the patients' statements of dissatisfaction over these events. This has been made possible by the Social Readjustment Rating Scale (27, 28). The scale lists 45 life events (retirement, divorce, death of a spouse, marriage, change of job responsibilities, etc.), each assigned a value by judges. These values represent weights based on the intensity and length of time such events require for adjustment to the event. A subject completing the scale lists each life event that has occurred during certain time intervals, such as six months ago, six months to one year ago, etc. The weighted value for each life event is then multiplied by the number of times the event occurred, summed across all 45 events, and this gives a life change or life events score. Connolly (10) and Rahe, Romo, Bennett, and Siltanen (49) report elevated life change scores in patients six months or so prior to myocardial infarction. In the Connolly study, life events were categorized as illness-independent or illness-related, e.g., the degree to which loss of job might or might not have been caused by the employee experiencing angina symptoms. When compared with control employees matched for occupation and age, the coronary patients not only experienced a greater number of life event changes of all types, but this difference also held true when only those illness-independent changes were counted. Rahe and Romo (49) studied 279 survivors of infarction and found that increases in life stresses appeared to be associated with the onset of new myocardial infarction. Moreover, they discovered that such increased stress was also associated with sudden or delayed death following illness. These studies of life change events, and the previously reported research on life dissatisfactions and

frustrations, lend support to the premise that stress and heart disease are correlated. Although predictive studies are sorely needed, the hypothesis is rapidly forming that stress may precede the onset of coronary disease. Another body of literature is also building that relates stress to specific physiological and biochemical changes, which in turn could lead to vascular insufficiency and heart disease.

A most interesting topic is the apparent influence of stressors on cholesterol levels. A number of studies have identified environmental stressors and examined their impact on cholesterol through sampling over time. Friedman, Rosenman, and Carroll (20) took blood samples from accountants twice a month from January to June. Since April 15 is the final date for income tax returns, the stress experienced by these subjects might be considered at its maximum the week before, and data on cholesterol would be especially informative. With diet intake remaining constant, the results showed the serum cholesterol to be substantially higher in April than in February or March. Following the April 15 deadline, the cholesterol level fell sharply. Studying underwater demolition team trainees, Rahe, Rubin, Gunderson, and Arthur (50) found that cholesterol levels increased under the stress of learning new skills, or when subjects felt fearful, angry, or depressed. Clark, Arnold, Foulds, Brown, Eastmead, and Parry (7) examined new cadets admitted to the Air Force Academy, and found that the highest peaks in cholesterol level occurred during the weeks considered to be the highest in environmental stresses for these cadets. Following this stressful phase of training, the cholesterol levels decreased. Finally, animal research has replicated the human studies. Paré, Rothfeld, Isom, and Varady (46) fed rats a high lipid diet, then exposed them to unpredictable and unavoidable shock for two to eight days. In comparison with control rats fed the high lipid diet but without shock experiences, the shock-stressed animals showed a higher accumulation of serum cholesterol.

Stress may have other physiological impacts that contribute to the risk of heart disease. Threat from stressors triggers the sympathetic nervous system and its emergency responses. As a fight-or-flight answer to the threat, the adrenals discharge epinephrine and norepinephrine (26, 42). Heart rate increases and blood pressure rises. It is speculated that the specific catecholamine secreted may depend upon the emotional response to the stressor; thus, epinephrine has been said to be associated with fear (22), while norephinephrine is connected with anger (21). The release of these catecholamines induces certain changes which may in turn be significant in the pathogenesis of coronary disease. Thus, the increase in blood pressure may aggravate a hypertensive condition. Also, since the catecholamines facilitate the aggregation of blood platelets, this reaction may be a contributing factor to thrombosis (1, 22, 45). Finally, the catecholamines have been

implicated in the increased formation of lipids in the blood (15).

Thus far, the data reported have examined the contribution of stress on coronary disease. As mentioned earlier, a second physiological dimension that appears related to risk of heart disease is the Type A (also known as Pattern A) coronary-prone behavioral pattern. The Type A pattern is the most promising and well researched of the psychological factors. In the early 1950s, Drs. Friedman and Rosenman became convinced that coronary risk might be predictable from certain overt behaviors. The story is told that waiting-room behaviors initially captured their notice. They observed that coronary patients seemed to "wear out the front part of the chair" by always sitting on the edge. However, the observation was overlooked, and it took a more direct experience to set off the search. In the course of a study of Junior League women and their husbands which discovered no differences in cholesterol intake, the president of the Junior League offered the advice that "stress" was the guilty factor in husbands' heart attacks. As a result, a series of rigorous studies was begun which confirmed the relationship between the Type A coronary-prone behavior pattern and heart disease (15, 17, 19, 52, 53, 55, 56, 57). The Pattern A behaviors involve a chronic sense of time urgency, a competitive achieving, a sense of being driven, an involvement in multiple activities with self-imposed deadlines, a high pace in both mental and physical actions, and an aggressiveness and hostility. Subjects may display some of the pattern or the entire pattern may be fully developed. In the large-scale research with over 3,000 male employees, approximately 50% (51) were classified as Pattern A persons; the figures for women are still being investigated and appear to be lower, but gradually on the rise as the employment of women also rises (77).

Two methods have been used to identify the behavioral pattern; the Structured Interview (SI) (57) and the Jenkins Activity Survey for Health Prediction (JAS) (34, 35, 36). The SI is a standardized stress interview designed to measure both mannerisms and content associated with Type A signs; the JAS is an objective questionnaire, leading to a total Type A score, as well as ones for Speed and Impatience, Hard-Driving, and Job-Involvement (factors considered as part of the Type A pattern).

In early studies, Friedman and Rosenman (17) demonstrated that whereas 4% of Type B men exhibited signs of coronary disease, 28% of Type A men were victims. A corollary study of women (53) discovered that Type A women showed four times the incidence of coronary disease as Type B women, as well as a higher cholesterol level, a faster blood clotting time, and a higher incidence of hypertension. The Western Collaborative Group Study was an attempt to obtain prospective data, using a longitudinal approach of over eight years, and covering over 3,000 men aged 39 to 59. Once again the vulnerability of Pattern A persons to coronary disease

was proven. Within 2½ and 4½ years after the start of the research (55, 56), the originally healthy Pattern A men now showed between nearly two to six times the rate of heart disease as Pattern B men (the higher ration being for the 39-to 49-year-old group). The trend continued to hold at the 8½-year final follow-up (52); of 1589 Type A men, 178 developed coronary disease as compared with 79 of the 1565 Type B men. In other words, the Type A men experienced nearly twice the rate of new coronary heart disease than the Type B men. Additionally important is the fact that the behavioral pattern was found to contribute significantly as a risk factor, even when the data was recomputed to adjust for the traditional risk factors of cholesterol, smoking, blood pressure, age, etc.

In addition to the evidence that Pattern A persons have a higher incidence of coronary disease, data is also available regarding the greater presence of other risk factors in Pattern A persons. For example, serum cholesterol appears higher in those with fully developed Type A behavioral patterns. Friedman and Rosenman (17) obtained mean values of 253 mg per 100 ml for Type A men as compared with 215 mg per 100 ml for Type B men. Among the 39- to 49-year-old subjects of the 8½-year follow-up, 21.2% of the Type A's had cholesterol levels equal to or greater than 260 mg per 100 ml, as compared with 16.5% of the Type B subjects. Similarly, serum triglycerides have also been found to be higher in Type A persons (19).

Other studies suggest that Type A subjects as a group exhibit increased sludging (19), a faster clotting time (17), and a greater discharge of norepinephrine in response to stressors (16, 63). Although not confirmed by others, Howard, Cunningham, and Rechnitzer (30) reported a correlation between elevated systolic and diastolic blood pressure and development of Pattern A behaviors. In addition, Type A persons seem to react to a stress interview with an increase in diastolic blood pressure while Type B persons seem to show a decrease (61). Finally, there is some suggestion that the presence of Type A patterns may enhance the risk of coronary disease when elevated diastolic blood pressure is also present (56).

The concept of stress in the Type A pattern is an important one. This author postulates that Type A behaviors face the Type A person with circumstances which act as stressors, e.g., the self-imposition of deadlines across many activities faces the Type A person with the stressor of time. Additionally, the sense of time urgency accepted by the Pattern A individual makes him perhaps hyperresponsive or sensitive to such time cues, thereby increasing the intensity of the stress. Furthermore, it is hypothesized that the Pattern A person's reaction to stress is to cope by exhibiting overlearned responses which have proven successful in the past. But such overlearned responses turn out to be Type A behaviors, such as working harder to meet the deadlines, staying longer if time starts to run out, arriving at appointments earlier, taking fewer vacations.

In a similar conceptualization, Glass (23) has interpreted the Type A pattern as involving "a characteristic style of responding to environmental stressors that threaten an individual's sense of control" (p. 181). Matthews, Glass, Rosenman, and Bortner (43) also offered the view that the "Pattern A may indeed be interpreted as a response style for coping with threats to a sense of environmental mastery and control."

It should be pointed out, however, that the Pattern A picture is far from complete. It is true that solid research data appears to confirm the relationship between Pattern A behaviors and risk of heart disease, through both retrospective and prospective studies. It also appears true that the Pattern A person may be reliably identified through either a structured interview or an objective questionnaire, and that the pattern exists among both men and women. However, there is insufficient direct evidence yet to permit formulating the role which stress plays in the Pattern A heart disease sequence, or to determine the exact antecedents of the behavioral pattern (44, 58).

On the other hand, all formulations regarding the role of stress are inferences that seem logically meaningful. Thus, Pattern A subjects seem to exhibit or possess other risk factors (higher cholesterol, faster clotting, higher epinephrine) which also may be attributable to stress induction. The catecholamines may be an important mechanism triggering a chain of events that eventually lead to heart disease; this hypothesis seems sensible in light of the data that Pattern A subjects show a greater discharge of norepinephrine in response to stressors than Pattern B subjects. Also, there exists a series of psychological laboratory studies involving stress induction (24) that seems to support the contention that Pattern A subjects react differently than Pattern B subjects to various categories of stressors. Finally, the premise that stress management is important in the lives of Pattern A persons forms the basis for some of the intervention methods to be described next.

In 1974 we reported on a behavioral approach to Pattern A patients (66). The Cardiac Stress Management Program (CSMP) was developed based upon several premises. The first premise was that stress management had to be the early focus of training. It was assumed that any attempts to encourage patients to alter their overlearned Pattern A behaviors would lead to tension, inasmuch as these behaviors have been "tried and true" patterns for so long. Also, it was felt that the patients' response to increasing tension would be to continue to display Pattern A responses as coping behavior to the stressor. A second premise was that tension reduction had to come from stress management methods which would show gains quickly, as opposed to long-term psychotherapy approaches. With the characteristics, time urgency, and impatience typical of the Pattern A patients, brief intervention techniques seemed essential. Hence, an application of Anxiety Manage-

ment Training (AMT) (67, 69, 72, 73, 75) was adopted. A third premise was that patients needed not only to see that alternates to Pattern A behaviors existed, but that these alternate behaviors would be just as productive, and did not entail becoming apathetic. A fourth premise was that behavioral training that emphasized a self-control model would be important (such as AMT). And finally, it was believed that behavior change would be fostered only by an experience permitting patients to actually emit alternate responses to Pattern A ones. Hence, Visuo-Motor Behavior Rehearsal (VMBR) (65, 70) was modified for use with the patients.

The Cardiac Stress Management Program involved three sessions of AMT followed by two sessions of VMBR. Anxiety Management Training is a behavioral approach that trains patients in anxiety coping skills (e.g., relaxation), in recognizing early physical signs that stress is building up, and in initiation of self-control over the stress before it becomes dominant or overwhelming. Deep muscle relaxation exercises (31) are used in collaboration with gradual exposure to stressful imagery until the patient has developed skill both in recognition of stress cues and in self-reduction of stress. Since this study was the first attempt to train post-MI patients within two to four weeks after infarct, the apparent success of this aspect of training alone was an important gain. Although there was concern about the suitability of the AMT stress arousal aspect so soon after hospitalization, there were no reports of physical symptoms or pain resulting from the training.

Visuo-Motor Behavior Rehearsal is a method originally developed by the author for use with athletic competitors and applied to Olympic Team members (71). VMBR is a means of enabling persons to practice new adaptive responses under conditions nearly identical with real-life ones through use of imagery. Patients are first relaxed, then trained to visualize an interaction prompting Pattern A reactions, then to substitute alternative behaviors. In CSMP, the AMT approach deals with stress management and is followed by the VMBR method to encourage the adoption of non-Pattern A behaviors. In the first application of CSMP, Pattern A patients at the Spalding Rehabilitation Center, Cardiac Reconditioning Unit, were identified by medical personnel. Such patients were then divided into a CSMP group and a control group. Both groups received the same rehabilitation program of exercise prescription, physiological stress testing, dietary assistance, and smoking management; only the CSMP group received the behavioral training. Data showed that nearly all of the CSMP patients (83%) reported substantial reductions in daily tensions, with the same numbers also reporting changes in life styles. These changes included behavioral ones, such as delegating more responsibility to employees. Stress reduction reports included statements such as "I could accomplish as much or more without the previous stress that I put on myself."

Of great interest were the contrasting results on lipid levels for each group. Whereas the control patients (who nevertheless were still undergoing standard cardiac rehabilitation treatment) showed a reduction in serum cholesterol of 2.6 mg % (from 233.7 to 231.1), the CSMP patients experienced a drop of 15.0 mg % (from 229.1 to 214.1). The change for triglycerides was even more dramatic, with a slight gain for the controls (179.3 to 182.5) but a substantial decrease for the CSMP group (171.6 to 128.8). Similar results were subsequently obtained with a replication sample (68). The lipid reductions were consistent with previous reports that changes in stress levels affect levels of cholesterol, and consistent with the CSMP patients' reports of subjective decreases in stress. Thus, the research appeared extremely promising. However, since the measure of behavioral change did not include either the Structured Standard Interview or the JAS, the results could only be considered as providing indirect evidence that stress management could alter Pattern A behaviors.

A follow-up study has been completed with direct measurement of the Pattern A style (74). Subjects were healthy volunteers responding to an advertisement describing Pattern A characteristics. Subjects came from all walks of life including a municipal executive, a regional sales manager, a dental laboratory specialist, an executive secretary, a farmer, and a mathematician. Psychological tests administered were the JAS, the State-Trait Inventory-State (STAI-S), and the State-Trait Inventory-Trait (STAI-T) (64). The JAS has been discussed earlier as one of two standard approaches to measuring Pattern A behaviors. It is computer scored, and provides several different scores thought to be related to coronary risk: a total Type A score, a Speed and Impatience score, and a Hard-Driving score. The STAI-S is a measure of state anxiety, i.e., the level of stress precipitated or present during the period of time the questionnaire is being completed; the STAI-T measures trait anxiety, i.e., continuing stress reactions that appear to characterize the subject regardless of situational conditions. State anxiety may be considered more situational and hence changing, while trait anxiety may reflect a more chronic inclination of the person to experience stress in a variety of situations. Blood pressure and 24-hour fasting blood samples were also obtained. The premise was again that stress management was a crucial step: Without stress control, Pattern A subjects would resist change; with it, subjects might begin to initiate their own attempts to change.

The program was conducted for six sessions, meeting twice a week, session one identifying Pattern A behaviors and providing relaxation training, and the remaining sessions involving AMT with added emphasis on time urgency stressors. Significant decreases were found to occur in the AMT-trained group on JAS Speed and Impatience, and Hard-Driving scores, and on both state and trait anxiety scales, as compared with a wait-list control

group. Total Type A JAS scores did not reach significance, but the medians for the treated sample showed reductions in the predicted direction. Although not significant, the blood pressure data were promising in that 86% of the treated subjects showed reductions in systolic pressure (mean reduction = 14 mm Hg) and 71% had lower diastolic pressure (mean reduction = 2.4 mm Hg). Lipid-level changes did not reach significance. On the whole, the results appear to confirm the effectiveness of AMT in reducing stress levels as well as Pattern A characteristics. Such results also offer indirect support for the premise that stress is involved in the formation and maintenance of Pattern A behaviors, in that removal of stress appears to have permitted changes in Pattern A characteristics. As indicated earlier in this chapter, this author contends that Pattern A persons place themselves in situations that tend to be stress producing, then react by displaying further Pattern A behaviors; hence, the Pattern A responses are in a sense self-perpetuating, with the key being stress management.

The previous hypothesis does not deny the possibility that the early antecedents of Pattern A behaviors could be parental modeling and training, cultural reinforcement, direct occupational rewards through organizational structures, and perhaps even a small genetic component based upon activity level, and there are some suggestive studies in these directions. Matthews, Richins, and Glass (44) conducted an exploratory study with mothers and their children, discovering that the mother of a Type A child encouraged him to try harder as compared with the mother of a Type B child. Generally, however, the "study does not provide support for a coherent theory of Pattern A development" (p. 161) (23). Cultural data (9) suggest that among the Japanese, the disease effects of being Pattern A increases "in direct proportion to the degree of Westernization" (8). Regarding occupations, individuals in professional and managerial positions tend to have a higher frequency of Pattern A behaviors than individuals at lower levels of the occupational structure; however, this correlation is not a strong one and Type A patterns appear in all levels of education and occupation (6, 15, 55, 62). Finally, Rosenman, Rahe, Borhani, and Feinlieb (58) reported positive, modest correlations between Pattern A behaviors and traits (e.g., activity, impulsivity) considered heritable among twins. On the other hand, the Type A pattern did not itself show a significant heritability estimate. The major points are twofold: Studies of such antecedent conditions are still exploratory and results complex, while data on stress seems somewhat more coherent; and intervention methods dealing with stress are currently available and may prove useful for initiating changes now among the many healthy Pattern A adults before heart disease threatens, and among those Pattern A patients who run further risks of additional hospitalization if life styles do not change.

Other recent reports are adding further confirmation that behavioral change can occur through psychological methods. Roskies, Spevack, Surkis, Cohen, and Gilman (59) selected healthy volunteer Pattern A persons, judged on the basis of the Structured Interview to be fully developed Type A's. One group was treated under a psychotherapy approach, centering on the theme that Pattern A behaviors were a repeat of a family constellation of striving for mother's love and guilt about ambition from identification with father. The behavioral approach for the second sample trained subjects in deep muscle relaxation, in daily monitoring of tension level, and use of relaxation to cope with any signs of stress arousal (a similar approach to the AMT model). Both groups were seen once weekly for 15 weeks.

Significant reductions were discovered after treatment for both groups in cholesterol and systolic blood pressure; although not reaching significance, the serum cholesterol for the behaviorally treated group appeared to drop to a much greater degree (40.7 mg per 100 ml) than that of the psychotherapy group (16.1 mg per 100 ml). A health questionnaire showed lower psychological and physical symptoms with increased general life satisfactions for both groups (the latter scores coming from a scale based upon the life events approach of Rahe). Neither STAI-S or STAI-T values reached significance (possibly because of the low control pretest levels), although again the behavioral group showed somewhat greater reductions. Unfortunately, the authors did not reclassify the subjects with the Structured Interview to measure directly changes in Pattern A profiles. As a substitute, subjects were asked to report on the number of hours spent in overtime work or in recreation, level of responsibility at work, and a self-rating of felt time pressure. The time pressure data were significant, but not the others. Once again, although not significant, it appears as if the behavioral group contributed more to the statistical significance (this group showed a reduction of 1.0 as compared with 0.4 change for the psychotherapy group). In general, the authors conclude that the two brief intervention approaches significantly modified some of the major physiological risk factors among Pattern A volunteers, and reduced stress levels, as measured by health symptoms, sense of time pressure, and feelings of satisfaction about life events. They also conclude that cholesterol and blood pressure reductions were achievable without diet, exercise, or smoking habit changes. This report is an exciting and encouraging one as the search for suitable intervention procedures continues.

The group psychotherapy approach of Roskies et al. (59) had been recommended previously by Friedman and Rosenman (18, 54), but no published data has yet been released measuring actual changes in Pattern A behaviors. Rahe, O'Neil, and Arthur (47), in an unpublished report, used brief group psychotherapy of four to six sessions with postmyocardial in-

farction patients. The therapy also included an educational format in the form of a booklet describing the nature of heart attacks, its emergency treatment, and rehabilitation methods. This combination led to fewer rehospitalizations among the treated patients than among those not receiving therapy. Reports on Pattern-A-related behavioral changes were impressions gained by the researchers from therapy discussions, and not measured directly. The Roskies et al. data on the behavioral approach is also consistent with Suinn's pilot CSMP studies with patients and his AMT study with healthy volunteers. It is also in keeping with an unpublished dissertation by Yarian (78). College students scoring high on the JAS appeared to show decreases in Type A scores from 15.7 to 12.5 following a four-session biofeedback relaxation program. Jenni and Wollersheim (37) have also reported on the use of behavioral methods with Pattern A persons. Using advertisements, a mixed group of 32 subjects was recruited, some with a history of heart disease but most being healthy volunteers. Those rated as showing Pattern A behaviors through the Structured Interview were retained for treatment. Treatment was composed of anxiety management training or cognitive behavior therapy, the latter emphasizing changing irrational beliefs about time, competitiveness, etc. After six weeks of treatment, the subjects showed significant reduction in both trait anxiety and state anxiety, with the anxiety management training group continuing to show reductions on trait anxiety at follow-up. However, state anxiety scores tended to show increases at follow-up, although not reaching the exact pretreatment levels. A subgroup of subjects identified as "high Type A" were selected for separate study. On this group (of 5 in cognitive therapy and 5 in anxiety management training), self-ratings of perceived Type A behaviors (on the Bortner Scale) declined more for the cognitive treated group. As with the Roskies study, the Structured Interview was not readministered but a self-rating scale substituted to measure Pattern A behaviors. A different behavioral approach has been reported by Jonson (38). Using Pattern A subjects who also suffered from essential hypertension, he used self-monitoring methods. Subjects learned to estimate their blood pressures through daily monitoring with a portable sphygmomanometer. They then used cues of increase in blood pressure as signals to attend to what was happening in the environment that was stressful. Once such stressors were identified, then a behavioral program of stress management and problem solving was initiated. Initial examination of results suggests substantial reductions in systolic blood pressure. A self-rating questionnaire on Type A patterns was also administered. Preliminary inspection suggests some improvements being reported following the twenty-week program.

Curtis (11) selected Pattern A volunteers from a single industry using the Structured Interview. An Educational Intervention Program (EIP) was of-

fered, involving ten sessions covering autogenic training, stress management, relaxation, and role playing. Although not reaching statistical significance, the data showed treated Pattern A subjects as ranking highest in reduction in cholesterol (a decrease of 12.9 mg per 100 ml), with the nontreated Pattern A subjects being next (a decrease of 9.2 mg per 100 ml), and the untreated Pattern B subjects changing the least (a decrease of 4.6 mg per 100 ml). Serum triglycerides also showed the largest decrease for the treated Pattern A subjects. Finally, since behavioral stress management approaches often include relaxation training, a report by Swencionis (76) is relevant. Using the JAS to identify Pattern A subjects, he concluded that Pattern A subjects were as capable of learning relaxation methods as were Pattern B subjects.

Although it is too early to tell, and since the few reports available are all based on smaller sample sizes or fail to collect direct measures of Pattern A behavioral changes, some trends may be offered for speculation. First, brief intervention methods have been the rule in these studies, and rather than being ineffectual, they have produced results. This suggests that Pattern A behaviors are not immutable or resistant provided that appropriate intervention methods are designed. Another premise that may have substance is the idea that stress management must be a major part of the program; without it, Pattern A persons may be unwilling or unable to change since the Pattern A behaviors may be a direct response to stressors. Friedman and Rosenman (18) have emphasized the Pattern A behaviors as a response to an environmental challenge or stressor. Matthews, Glass, Rosenman, and Bortner (43) viewed Pattern A behaviors as a "response style for coping with threats." If these assumptions are accurate, then programs simply aiming at changing Type A patterns without regard to their coping and stress management functions are not likely to succeed. At the same time, programs with a stress management component may be viewed as more likely to succeed, whether the stress management is through direct training in stress arousal and control as in AMT, or cue-controlled relaxation, or indirect training such as in biofeedback-assisted relaxation. A third trend is that behavioral methods may be more effective than traditional therapy methods, although this speculation is tentative. In the only report comparing the two, significant differences were not obtained by Roskies et al.; however, on a number of variables, the behavioral group seemed to progress more favorably. The fact that six reports (37, 38, 59, 68, 74, 78) obtained some important results with behavioral procedures suggests that the efficacy of such methods is promising and should not be ignored. Although it could be argued that each of these also relied upon relaxation training, it is unlikely that learning to relax will alone lead to Pattern A changes. The isolated response of taking 15 or so minutes to rest and relax (a sort of psychological

coffee break) can and does have some benefits, as evidenced by reports on forms of meditation. These may be attributable to the recovery of bodily processes during the resting period and to the temporary reduction of stress as the person is physically isolated from stress cues (the presence of people, task demands, telephones, etc.). However, unless the individual is trained to apply the relaxation response as a stress coping behavior, then all the Pattern A person has accomplished is a brief interval of quiet before the environmental realities again prompt the striving, urgencies, deadline setting, impatience, acceptance of heavier work loads, etc. (13) Stated in another way, the use of stress management programs aims at both aiding the person in facing stress and also at providing the opportunity to acquire alternate coping skills. The Pattern A person must do more than take coffee breaks (then return to working harder to make up for the lost time); he must acquire a healthier behavioral style. A fourth speculation derives from thoughts about group therapy. In the Roskies et al. group, the therapist worked from a theory about the origins of striving and aimed at prompting changes related to this area. With Rahe et al., patients received direction from a manual on rehabilitation, and also had opportunities to discuss "changing their ways" regarding deadlines, rushing, work, etc. It is possible that such a more structured group therapy, with clearer behavioral objectives and a tone of commitment to discovering actions that reflect change, forms an important core for this traditional therapy model. Pattern A persons are often aware of their gratifications from being Pattern A persons, and often clearly state their occupational rewards and status as factors. They are also fully cognizant of the increased risk of further hospitalization if they do not alter their behavioral patterns. Traditional therapy based upon talk-therapy without direction or insight-therapy without commitment to behavioral alternatives may therefore be inappropriate. Research is sorely needed that isolates intervention methods based upon a conceptualization of Pattern A dynamics (we suggest stress management followed by behavior change), using larger sample sizes and including studies of both healthy Pattern A subjects and those suffering from coronary heart disease, utilizing standard measures of Pattern A characteristics and a battery of measures of dependent variables considered associated with the Type A style (such as lipids, behavioral diaries, life event reports, and anxiety scales) (see Tables 1 and 2 for summaries of studies using lipids or stress measures), and with follow-up to determine the stability of changes. Control groups should include placebo groups, or at least ones in which active rehabilitation treatment is underway and the patients are not subject to doubts based on their awareness of an existing psychological therapy group for other patients. At the start, smaller studies are needed to refine different intervention methods, which might then be examined in the more system-

Table 1: Pattern A Interventions and Lipids

Study *Group*	*N*	*Program (No. Length Sessions)*	*Serum Cholesterol Pre*	*Post*	*Change*	*Serum Triglycerides Pre*	*Post*	*Change*
Suinn, 1975* (67)								
Control A's	10	Wait List	233.7	231.1	- 2.6	179.3	182.5	+3.2
Behavior Therapy A's	10	5	229.1	214.1	-15.0	171.6	128.8	-42.8
Behavior Therapy A's	17	5	262.5	243.3	-19.1	270.9	205.8	-65.1
Roskies et al., 1978 (59)								
Psychotherapy A's	13	14	237.1	221.0	-16.1	119.6	119.0	- 0.6
Behavior Therapy A's without CHD	12	14	238.2	197.5	-40.7	138.5	134.0	- 4.5
Behavior Therapy A's with CHD	6	14	213.3	180.7	-32.6	130.2	114.8	-15.4
Curtis, 1974 (11)								
Control A's	11	Wait List	202.4	193.2	- 9.2	104.4	102.0	- 2.4
EIP** A's	12	10	181.3	168.3	-13.0	144.3	137.0	- 7.3
Control B's	7	Wait List	193.1	188.6	- 4.5	109.3	104.6	- 4.7
Suinn & Bloom, 1978 (74)								
Control A's	7	Wait List	222.0	206.2	-15.8	87.2	92.0	+4.8
Behavior Therapy A's	7	6	187.3	203.3	+16.0	113.0	128.0	+15.0
Jenni & Wallersheim, 1978 (37)								
Control A's	13	Wait List	235.1	241.5	+6.4	–	–	–
Cognitive Therapy A's	14	6	213.5	201.9	-11.6	–	–	–
Stress Management A's	11	6	225.1	253.7	+28.6	–	–	–

*Subjects of this study were heart disease patients; other studies used healthy volunteers.

**EIP included autogenic training, stress management, role playing.

Table 2 : Pattern A Interventions and Stress Measures

		STAI-State			*STAI-Trait*			*Other Measures*		
Study Group	*N*	*Pre*	*Post*	*Change*	*Pre*	*Post*	*Change*	*Pre*	*Post*	*Change*
Suinn & Bloom, 1978 (74)										
Control A's	7	47.7	68.0	+20.3	49.7	45.7	- 4.0	–	–	–
Behavior Therapy A's	7	51.3	37.2	-14.1	47.5	37.5	-10.0	–	–	–
Jenni & Wallersheim, 1978 (37)										
Control A's	13	41.2	43.8	+2.6	43.2	49.3	+6.1	–	–	–
Cognitive Therapy A's	14	40.4	32.8	- 7.6	42.0	36.6	- 5.4	–	–	–
Stress Management A's	11	40.6	37.4	- 3.2	51.6	44.0	- 7.6	–	–	–
Roskies et al., 1978 (59), 1979 (59a)								Health*		
Psychotherapy A's	13	32.0	31.5	- 0.5	34.3	33.5	- 0.8	4.6	2.9	-1.7
Behavior Therapy A's without CHD	12	36.1	33.7	- 2.4	41.4	35.8	- 5.6	11.0	1.5	-9.5
Behavior Therapy A's with CHD	6	36.2	35.2	- 1.0	32.8	33.5	+0.7	4.5	0.8	-3.7
Yarian, 1976 (78)								EMG†		
Control A's	10	–	–	–	–	–	–	7.12	7.80	+.68
Biofeedback A's	10	–	–	–	–		–	7.81	4.38	-3.43
Control B's	10	–	–	–	–	–	–	5.76	6.03	+.27
Biofeedback B's	10	–	–	–	–	–	–	6.46	5.13	-1.33

*The General Health Questionnaire–measures psychological and psychophysiological symptoms.

†EMG–from the frontalis muscle; may be interpreted to reflect stress versus relaxation levels.

atic and rigorous ways mentioned above. However, to prevent a proliferation of many "exploratory" studies, larger-scale ones should be encouraged as soon as there is the start of a foundation. Theory building that states the guidelines being pursued in the intervention method must be established, to avoid techniques being tried merely for techniques' sake. The relationship between the theory of Pattern A change and the goals of the intervention should be explicated. For example, there are a variety of general lines of attack possible. Therapy has the option of initiating emotional reconditioning to eliminate the ability of a cue to act as a stressor, of attempting to alter the person's perception or cognitions of what constitutes a threat or a stressor, of encouraging the person to leave environments that precipitate Pattern A behaviors, or of strengthening alternate stress-coping behavioral patterns.

Anxiety management training is a combination of emotional reconditioning plus self-management whenever stress is prompted in the future. Cognitive restructuring may be possible as a means of either altering values leading to behavioral shifts or permitting stressors to continue but without emotional impact. Leaving the stressors in the environment may not be as drastic as it sounds. This may include preventative steps such as learning better time management, carefully interspersed scheduling of stressful appointments during time slots when energy level is still optimal, and delegating tasks meaningfully. Of course, value shifts through career goal setting might also lead to major job changes that may reduce stressors. Intervention programs may be developed emphasizing the acquisition of alternate stress-coping patterns. For example, since Pattern A persons may be subject to accepting work overloads, learning how to gauge work assignments and refuse gracefully and without guilt is desirable. Of course, if the acceptance pattern relates to time urgency and impatience ("I can do this quicker; I hate to wait for them to finish it"), emotional stress management may be more appropriate. Acquiring a perspective on the proper role of relaxation and recreation-as-relaxation (rather than as striving) can be most effective. Identifying and using other persons as resources under stress conditions can be a valuable stress coping approach. Even learning to place behaviors under self-control can be stress reducing. The Pattern A individual is characterized by rapid striding, fast eating, emphatic speech, and quick movements. In turn, these very actions may be subtle triggers precipitating a sense of urgency, rush, stress, and tension. By deliberately walking at a slower rate, and talking at a calmer pace, the overall impact may be a greater sense of calm and control, perhaps through a psychophysiological-behavioral feedback loop. As these various types of approaches are researched, more broad intervention programs might be considered. Family programs might be valuable, on the premise that spouses contribute heavily to maintaining patterns of behavior through their own response patterns,

and may be obstacles to change. More studies are needed to fill the void in understanding possible childhood antecedents of Pattern A behaviors and to develop preventative steps. In the way of prevention, perhaps major changes are needed on the societal level. Industries and businesses can be influential in supporting non-Type A solutions to work tasks, in the same way that industry accepted the rest break as a contributor to enhanced efficiency rather than lost time. Stress management training might be introduced to protect the working lifespans of employees. Perhaps large-scale communications programs can begin to analyze the role of striving in the United States. Achievement appears to be synonymous with striving, and being driven with being successful. Yet alternate values and attitudes are present in other countries. Japan successfully manages to be a productive nation, while its employees avoid being overwhelmed by competition. The Japanese worker fits the mold of being hard working while not being driven. However, these large-scale types of intervention may be presumptuous to consider at this time. What is important is the fact that steps must be taken about the Type A pattern.

In conclusion, there are some structural elements which might be useful in the design of intervention programs for individuals. First, brief intervention seems almost necessary. Pattern A persons approach treatment as persons "in a hurry" to begin with. Unless hospitalized for physical reasons, they are unlikely to devote months to a program of change. Secondly, and in a related way, these brief programs must have a tangible impact in some form. Participants must experience concretely a new ability to relax, an insightful demonstration of their actual daily stress levels, a different perspective on their definitions of success, or some other aspect that is a convincing early product of treatment. From a theory, Glass has obtained a sequence of coherent studies supporting the premise that loss of control is stressful for the Pattern A subject (23). If we accept this premise (while reserving judgment on other sources of stress), a third element which might be suggested is the inclusion of self-control or self-management procedures. These procedures emphasize the goal of training the participant in acquiring methods and skills that are effective without the presence of a therapist. A fourth aspect relates to the inclination of Pattern A persons to point to their achievements as correlated with being Type A, and the corresponding fear that not being Type A would lead to loss of productivity and apathy. This fallacy must be understood and dismissed at once. Finally, rather than seeking immediate changes in the variety of behaviors that form the Type A pattern, ranging from multiple thinking to aggressiveness, early intervention steps might aim at the more salient ones.

Matthews, Glass, Rosenman, and Bortner (43), in factor-analyzing the Structured Interview, suggest tentatively that time urgency and impatience

and hard-driving factors seem most associated with coronary disease risk. The urgency and impatience response pattern may be viewed as an emotional stress pattern to time cues. Emotional reconditioning or emotional control programs appear most suited to this type of issue. The hard-driving component is still not altogether clear and may be a derivative of three elements: hard-working behaviors, aggressive competitiveness, and a high activity level. Some of these might be approached in programs aiming at better management of work loads, balance between work behaviors and recreational or nonwork tasks, development of satisfactions in nonwork activity, and engaging in work behaviors based on reality constraints rather than anxiety or tension cues (being driven to work). Finally, the reader by now has recognized that the soundest data on the Pattern A behaviors are those that identify the behaviors as a significant risk factor in coronary heart disease. Surprisingly enough, although such a finding may be traced back over 15 years, the development of intervention methods is still in its infancy. The intervention research reported in this chapter is provocative, promising, exciting, stimulating, but still exploratory. Many of the statements proposed by this author are tentative and are offered as speculative guideposts in yet uncharted territory. But the answers are there to be found. And given the high cost to man and society of heart disease, it is imperative that they be found.

NOTE

Since the writing of this chapter, Roskies et al., have published a six-month follow-up, showing behavior therapy for clinical CHD persons to be more beneficial than psychotherapy on follow-up, with behavior therapy for non-CHD persons showing intermediate level benefits.

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CHAPTER 2

Behavioral and Psychophysiological Aspects of Raynaud's Disease

C. KEITH CONNERS

Maurice Raynaud (39) provided the first accurate clinical description of the condition that now bears his name:

> Madame X, aged 26 years, has never been ill; but she has been the subject since childhood of an infirmity which makes her an object of curiosity to her acquaintances. Under the influence of very moderate cold, and even at the height of summer, she sees her fingers become exsanguine, completely insensible, and of a whitish yellow colour. This phenomenon happens often without reason, lasts a variable time and terminates by a period of very painful reaction, during which the circulation is re-established little by little and return to the normal state. Madame X has no better remedy than shaking her hands hard, or soaking them in lukewarm water.... This state, which I dare hardly call a disease, is local syncope in its simplest form. [As quoted by Birnstingl (1)]

This condition—seen principally in young healthy women, when characterized by episodic digital asphyxia due to an exaggerated reaction to cold in otherwise normal digital vessels and in the absence of other disease—has been termed *Raynaud's disease.* When any of a variety of pathological conditions lead to peripheral arterial insufficiency, for example, endocrine disorders, arterial disease, arterial compression, collagen disorders, etc. (28, 38), and result in episodic digital asphyxia, the condition is referred to as *Raynaud's phenomenon. Raynaud's syndrome* includes conditions in which there is general arterial insufficiency of the fingers, whether episodic or not,

and regardless of whether digital necrosis or gangrene is present (1). Although it is frequently thought that Raynaud's disease is rare, the phenomenon of reactive cold hands and feet, particularly among young women, appears to be quite common and probably has a wide distribution of severity and may not come to medical attention except in extreme forms. No accurate epidemiological surveys of incidence are available.

Attacks are usually limited to the upper extremities but may also occasionally involve feet and toes. Painful ulcers on the fingertips with eventual necrosis are generally thought to occur only in Raynaud's phenomenon, where disease of the peripheral vessels or blood supply are implicated (1). Many patients have well-defined sequential color change in the affected digits when a vasospastic attack occurs, often changing from pallor to cyanosis to red which is accompanied by pain. However, many times color changes are minimal or only one or two of the colors appear.

A number of techniques have been employed in an attempt to establish objective diagnostic criteria, including thermometry, thermography, calorimetry, plethysmography, regional isotope clearance, and arteriography (38). Neither basal digital temperature nor the absolute temperature decrease following cold exposure consistently discriminates between normals and patients with Raynaud's phenomenon. However, the time required for temperature recovery after cold exposure is often different in normals and patients with vasospasm (38).

Although medical intervention is effective when an underlying organic pathology can be corrected (such as relieving pressure on a subclavian artery), treatment of Raynaud's disease is frequently unsuccessful. At one time local sympathectomy was often prescribed to relieve the symptoms. Follow-up studies found poor long-term results. Neither pharmacologic nor surgical treatments are indicated in most cases. A noninvasive behavioral treatment would be potentially useful for this disorder. This chapter will summarize what is known of behavioral influences on peripheral circulation and vasomotor activity, and then discuss the current status of behavioral intervention with Raynaud's disease and phenomenon.

NEUROGENIC CONTROL

The control of peripheral vasomotor tone is quite complex; it is regulated by an intricate series of excitatory and inhibitory influences at all levels of the nervous system (41, 45). Raynaud proposed that the vasospasms were due to normal reflex responses to cold and were controlled by central sympathetic influences (40). Lewis (22) proposed a "local fault" explanation. He showed that when reflex vasodilation had been produced by warming the

body, vasospasm still occurred when the patient's hands were put in cold water. Conversely, vasospasm could not be produced by body cooling when the hands were kept warm. More recent explanations emphasize that both local mechanisms that provide autoregulation of blood flow and multilevel CNS influences (46) are important. Considerable voluntary cardiovascular control is possible (7), which suggests that higher cognitive processes may be implicated in peripheral vasomotor activity.

The anterior hypothalamus is an important brain center for temperature regulation. Animals deprived of hypothalamic structures have markedly altered thresholds of response to cold or heat. Although the hypothalamus appears to regulate reflex-patterned adjustments to maintain the body temperature in changing environmental conditions, various components of this regulatory pattern can be activated by other stimuli and can be behaviorally conditioned independently of thermal adjustment requirements (45). It is entirely possible that emotional stimuli from higher CNS pathways and the reaction to a cold environment could interact to produce an abnormal physiologic state in the peripheral vessels. Although it has been proposed that disturbances in serotonergic areas of the brain produce electroencephalographic (EEG) changes that reflect disturbed central control over peripheral vasomotor activity (19), careful EEG studies of Raynaud's patients fail to show any such abnormalities that might be indicative of a seizure-like basis for the disease (11).

RELATION TO SKELETAL MUSCLE ACTIVITY

Recent psychophysiological investigations have emphasized the close coupling of the somato-motor and cardiovascular systems. It is known that there is a sympathetic cholinergic vasodilator regulatory area in the motor cortex that regulates blood flow to peripheral muscle. This system produces vasodilatory responses in large muscles in *anticipation* of exercise and may be part of a generalized physiologic preparation for fight or stress (41, p. 102). Stimulation of the hypothalamic portion of this system produces rage behavior in cats, which raises the possibility that anger, hostility, and other action-oriented emotions might activate this system.

Isometric contractions of either the hand or the foot result in a significant decrease in hand temperature, and the effect is proportional to the strenuousness of the exercise (25). Precise vasomotor control can be learned with appropriate feedback even when there is no muscular contribution (24). Miller and colleagues (29) suggested that their curarized animal preparations, in which there was good operant control over peripheral circulation, may have benefited from the elimination of distracting cues from the skeletal muscle system. With one exception (12), reports of biofeedback-as-

sisted muscle relaxation indicate that decreased muscle tone is associated with increased digital skin temperature (33). Prior training in EMG biofeedback relaxation has been suggested as facilitating acquisition of feedback control over skin temperature (56).

ANXIETY AND STRESS

Digital vasoconstriction usually occurs as part of an anxiety or stress response (5; p. 189). Subjects show vasoconstriction when discussing topics that are stressful for them but no vascular changes when discussing neutral or positive topics (10). Cases have been reported where Raynaud's disease appeared to be a response to a disturbing life situation (30, 31, 14) or sheer drop in digital temperature occurred during a stressful interview (14). One author proposes that these patients are in a state of chronic anxiety (31). D. T. Graham suggests that "vasoconstrictive" emotions involve either overt action or preparation for action, e.g., states of hostility or anxiety (15). FPV (Finger Pulse Volume) is more immediately sensitive to stress than pulse rate (6). Subjects exposed to the threat of shock show decreased FPV, which correlates significantly with self-reported anxiety. These vasoconstrictive effects are opposite to the increase in blood flow in the forearm which occurs as a response to stress (32).

The prestress activity of the vasomotor system determines the direction of change. For example, anxiety increases vasodilation in the fingers of subjects whose hands were previously exposed to cold. Even higher levels of anxiety may lead to a reflex rebound and even more intense vasoconstriction (44). When vasoconstriction in the fingers is induced by body exposure to cold, a further lowering of hand temperature by sudden immersion in ice water (the cold pressor test) will cause vasodilation (23). The rebound latency of the immersed hand can be further affected by stress or threat of stress (55). These data indicate a centrally mediated contribution to peripheral vascular responses.

Lowered room temperature interferes with the ability of normal subjects to gain directional control over finger skin temperature during biofeedback, and some studies show that only attempts to decrease temperature are statistically reliable (54, 59). This may be the result of superimposing feedback upon a system already undergoing reflex vasodilation. These studies typically find large differences among subjects in ability to gain voluntary control in a cold environment. Unfortunately, many studies have not adequately controlled for the stress effect of the cold (which some subjects may perceive as quite aversive). Cold stress might trigger normal vasoconstriction and counteract any voluntary efforts to vasodilate.

EFFECTS OF ATTENTIONAL FOCUS

Heart rate undergoes a biphasic response when subjects are given an interval during which they prepare to make a motor response (34). This phenomenon appears to be an adaptive response that reflects adjustments facilitating sensory intake or rejection. Forearm and finger blood flow show a similar biphasic response to Preparatory Interval (PI), and when subjects are asked to perform a task requiring concentration on an external signal, both tonic heart rate level and blood flow decrease. Adding a PI accentuates the effect (58). These findings suggest that attention might alter peripheral vasomotor tone and the phasic responses of vasoconstriction. Subjects required to identify words—a task presumably leading to "sensory intake"—show a small decrease in forearm blood flow and a large increase in forearm vascular resistance. Apparently this type of sensory intake is associated with a vasoconstriction of skeletal muscle vasculature (57, 58). Large changes in digit pulse volume accompany external attention in an interview situation (2). A similar effect has been demonstrated in children given sensory intake or sensory rejection tasks. Autistic children respond as though in a chronic state of sensory rejection with increased digital blood flow compared with normals (9).

PERSONALITY FACTORS

No consistent personality characteristics have been associated with Raynaud's disease. Hostility and anxiety have been implicated in the disease in both neurophysiological and clinical studies. However, these data are impressionistic. There is some indication that subjects with external loci of control have higher basal skin temperatures and greater temperature stability than those with internal control loci (13).

VERBAL INSTRUCTIONS, SUGGESTION, AND HYPNOSIS

Biofeedback and other experimental manipulations of states, e.g., conditioning studies, often confound instructional variables with the experimental manipulations. For example, much of the directional heart rate effect attributed to biofeedback can be obtained by instruction alone (7). Similar effects are seen with the behavioral control of skin temperature. In one study (26), undergraduates were given training in hypnosis for about ten hours and while hypnotized they were asked to produce bilateral skin temperature changes. They were able to produce large decreases, as much as

7°C, and increases of up to 2°C. None of the awake subjects was able to achieve this magnitude of temperature change in the hands. This effect has not been replicated (42), and in addition it has been shown that neither capacity for absorbed attention nor hypnotic susceptibility is related to the ability to learn to control skin temperature (43). These studies support the view that generalized relaxation, removal of distracting stimuli, confidence of the subject in his ability to gain control, and motivation to persist in attempts to change hand temperature are important variables; hypnotizability or hypnotic trance state are not.

When subjects are trained to increase hand temperature by biofeedback, suggesting warmth by using thermal imagery, verbal suggestions, slides, and taped sentences all enhance acquisition of control (17, 18, 48). Early experiments that attempted to induce the typical attitude of Raynaud's syndrome patients by hypnosis and consequently produce digit vasoconstriction (16) have not been replicated. A carefully controlled study (37) showed that regardless of the experimental suggestion or attitude employed, hypnotized subjects vasoconstricted and unhypnotized subjects vasodilated (3).

BIOFEEDBACK AND CONDITIONING

Operant conditioning of finger blood volume in humans has been clearly demonstrated (49, 51). These studies have demonstrated that the effects are not due to conditioning of respiration or skeletal muscle. However, post-experimental questionnaire data reveal that most experimental subjects correctly hypothesize that the reinforcing signal is contingent upon their stimulating thoughts or bodily activity, whereas control subjects either think the signal is contingent on their relaxing or they do not know (51). In an experiment in which subjects were not informed of the purpose of the study, successful "increase" subjects reported either no thoughts or relaxing imagery while successful "decrease" subjects spontaneously reported being occupied with "tension, efforts, or lost love affairs, etc." (35). Interestingly, control subjects who were unaware of the purpose of the experiment became frustrated because of false feedback signals and also showed temperature decreases. In one study (21), these effects were significant after 12 training sessions but not after 4 or 8. More recently, significant within-session increases in finger temperature averaging 2°F were obtained after 5 training sessions with no further improvement in control up to 20 sessions (20).

BIOFEEDBACK WITH RAYNAUD'S PATIENTS

Surwit (52) described four successfully treated cases of Raynaud's syndrome. One case was treated with biofeedback, relaxation instruction, autogenic imagery, counseling, and assertive training. He felt that failure of maintenance at one-year follow-up was probably due to loss of motivation. He pointed out that in addition to relaxation, autogenic imagery, and in one case hypnosis, there was a large amount of therapist attention and enthusiasm. He suggests that the patient's life style and coping patterns may need alterntion to prevent inadvertent reinforcement of symptoms and to help the patient remain motivated to practice. Blanchard and Haynes (4) pointed out the lack of pretreatment data and confounding treatments reported in these studies. They conducted a single-case design study in which feedback training was alternated with self-control sessions. At follow-up two, four, and seven months after treatment, further self-control and feedback "booster" sessions were given. Not only was their patient, a 28-year-old female with moderately severe symptoms, able consistently to increase the temperature of her hand during the feedback phase, but the latency with which the maximum increase was obtained decreased during this phase. The patient's efforts at self-control were not consistent but appeared to improve over time. Although this study controls for baseline changes, expectancy, and instructional effects, the authors note that the patient spontaneously began to use thermal imagery, which may have contributed to the improvement in hand temperature control or the decreased symptoms from her Raynaud's syndrome. This case demonstrates the use of an adequate methodology in a clinical situation to elucidate scientific issues while providing treatment.

May and Weber (27) reported four case studies of patients with Raynaud's disease, and four with Raynaud's phenomenon caused by scleroderma or lupus. Treatment consisted of 16 50-minute temperature feedback sessions over eight weeks. Five normal controls were also studied. The patients with Raynaud's phenomenon showed the most consistent and the most effective control. Subjects with severe symptoms performed better and achieved more symptom reduction than those with mild symptoms, an effect the authors attribute to increased motivation. They comment that "the success of the training depends more on the subject achieving a state of relaxation, on the subject achieving mental contact with his fingers (perhaps easier for a Raynaud's subject since his disease is a sign of a connection, albeit a maladaptive one) and on the motivation of the subject... than it depends on the degree of physiological pathology present." The extent to which disturbed central control mechanisms in primary Raynaud's disease might contribute to these findings was not explored.

Stephenson (50) reports two successful cases in which biofeedback was combined with autogenic training, muscle relaxation, and psychotherapy. Both patients were suffering from Raynaud's phenomenon with unhealed lesions and areas of necrosis on the fingers. Temperature increases obtained were 10.1°F and 10.5°F. Large rapid increases in temperature have also been reported for three cases with pure Raynaud's disease (47).

In a recent unpublished report (36), three cases of Raynaud's phenomenon were treated with biofeedback alone. One case, a 51-year-old female with dermatomyositis, scleroderma, hypertension, and severe symptomatology failed to show improvement with ten sessions of biofeedback. She had multiple somatic complaints in addition to Raynaud's symptoms, and was the only one of the patients with overt psychopathology. She described herself as being a "tense person." Her MMPI was typical of an individual with a conversion reaction or one who is enjoying considerable secondary gain from the symptoms. For the two successful cases, maximum temperature increases were 15°F and 16°F. The largest changes occurred within the first 15 minutes of the sessions, and a 2.5°F increase was considered a successful response. Despite this temperature increase, only one of the two patients showed a consistent reduction in symptoms at follow-up.

Finally, in a recent controlled study (53), Surwit assigned 30 subjects with Raynaud's disease to four treatment groups: Biofeedback plus autogenic training and autogenic training alone, with half of each treatment group practicing at home and half in the laboratory. All treatment groups showed a significant increase in hand temperature between pre- and posttreatment sessions, and treated subjects were all significantly better than waiting-list controls. Moreover, a stress test consisting of decreasing the ambient room temperature from 25°C to 17°C caused fewer symptoms in treated patients than untreated controls. Neither the biofeedback nor the visits to the laboratory appeared to be essential for reducing self-reported vasospastic attacks or objective stress performance in a cold environment. It is worth noting that very few studies use objective criteria of symptomatic improvement; most rely on patient reports of vasospastic attacks. Although increased hand temperature should be a prerequisite for removing symptoms, hand-warming alone may be insufficient to produce objective changes. Clearing of dermal lesions, changes in hand color, changes in blood flow, and other factors need to be documented.

COMMENT

The important question for the clinician is what methods of therapy or combinations of treatments are most effective. Our review of the psycho-

physiology of this group of disorders that shares cold hands as a common symptom has shown a number of factors appearing to be therapeutic variables. Few case reports or experimental studies are rigorous enough to allow us to rule out alternative explanations. Only the recent report of Surwit et al. (53) has attempted to separate the effects of experimenter contact, biofeedback, and self-instruction. After reading this clinical literature, it is hard to avoid the conclusion that several *different* mechanisms affect peripheral circulation; multimodal assessment and therapy may be indicated.

Given the fact that decreasing muscle tension has been shown to facilitate acquisition of learned vasomotor control; that static muscle tension causes vasoconstriction; and that the motor cortex is closely linked to peripheral vasomotor adjustments, it seems that *some* patients with Raynaud's syndrome may benefit from EMG biofeedback or other relaxation techniques. Active exercise or isometric training might also produce improvement in peripheral circulation by eventually correcting chronic vasoconstrictive "overshoot" under cold conditions through homeostatic compensation.

It seems reasonably clear that thermal imagery, suggestion, and self-instruction can lead to clinically useful vasodilation. For those Raynaud's syndrome patients who have a vasoconstriction response to anxiety or stress, behavioral methods for anxiety reduction such as densitization or assertive training may be useful. It is unwise, however, to assume that any general trait (hence treatment) will characterize all patients. For example, external attending seems to be associated with marked digital vasoconstriction in some patients. This cognitive process may be a normal reflex that is adaptive for passive information-seeking situations, and may accompany a character style of extreme vigilance. This coping style might be treatable by "sensory rejection" attentional training, which is associated with vasodilation (2, 58). The "bare attention" method of Vipassana meditation might be useful in this case. To our knowledge, neither the assessment nor the training of the attentional component of the vasomotor defect has been reported with Raynaud's syndrome patients. Hypnosis, as a potent form of attentional focus and self-instruction, may be a useful treatment technique.

Although everyone agrees that a high degree of patient motivation is essential for gaining vasomotor control, no formal methods have been developed to assess this factor. It is interesting that very large finger temperature changes, 15° or more, are often reported with Raynaud's syndrome patients but seldom in studies with normal control subjects. Whether this relates to the intrinsic pathology or the added motivation of symptomatic patients is unclear. Although the profound homeostatic reflex adjustments of the peripheral vessels and basal blood flow rate to cold and stress are well known, no one has investigated a shaping procedure whereby patients would be ex-

posed to small gradations of progressively colder ambient temperatures during training with appropriate controls for the subjective response to cold as a stressor.

Biofeedback has been shown to be useful but not essential in the treatment of Raynaud's syndrome patients. In reviewing the studies presented in this paper, one is impressed by the variability among subjects in response to treatment. Each patient must be evaluated individually and treatment planned accordingly. Biofeedback is an informational approach to visceral learning to which some patients will respond more readily than others. Similarly, some patients will require more imagery-oriented intervention, stress reduction counseling, or attentional types of treatment programs.

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The Comprehensive Handbook of Behavioral Medicine, Volume 1

CHAPTER 3

Psychological Treatment of Cardiac Arrhythmias

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INTRODUCTION

Psychological factors have long been thought to play a role in the etiology of certain types of cardiac arrhythmias. However, not until recently have such psychologically based techniques as biofeedback, relaxation training, meditation, hypnosis, and psychotherapy been used in the treatment of certain patients with cardiac arrhythmias. The aim of this chapter is threefold: 1) To provide an overview of the role psychological factors may play in the etiology of cardiac arrhythmias; 2) to review the rationale, procedures, and outcome of the various psychological interventions used in the treatment of cardiac arrhythmias; and 3) to discuss future directions in research and clinical application.

PHYSIOLOGICAL BASIS FOR CARDIAC ARRHYTHMIAS

The normal cardiac impulse originates in the primary pacemaker in the sinoatrial node and is propagated in a regular sequence through the atria, the atrioventricular node, and the ventricles via the common bundle, right and left bundle branches, and the Purkinje fibers. This normal cardiac activity is dependent on a greater degree of automaticity in the sinoatrial node than in other potential pacemakers in the heart, and a synchronized conduction of all impulses resulting in coordinated ventricular contractions. When one or both of these functions is interrupted, a cardiac arrhythmia re-

sults. Arrhythmias can be categorized according to their underlying mechanisms as disorders of impulse formation, disorders of impulse conduction, or disorders resulting from abnormalities of both impulse formation and impulse conduction.

PSYCHOLOGICAL INFLUENCES ON CARDIAC ARRHYTHMIAS

Although coronary artery or other heart disease plays a major contributory role in most cardiac arrhythmias (1, 9), psychological factors have also been proven to be important. Several areas of research indicate that psychological factors can affect the rhythmicity and conduction of both the healthy and the diseased heart (14, 16, 22). It has been demonstrated that psychological stressors presented during a stress interview and the associated physiological arousal can result in blocks in atrioventricular conduction (2), atrial arrhythmias (11), ventricular extrasystoles (24), and atrial ventricular tachycardias (17, 27). Most of these findings have been based on a small number of patients, and therefore the relationship between stress and cardiac arrhythmias remains somewhat suggestive.

Research on classical and operant conditioning indicates that learning processes may play a role in the etiology and exacerbation of cardiac arrhythmias. For example, premature ventricular contractions have been classically conditioned in subjects with no history of heart disease (19). This finding suggests that various cardiac arrhythmias could be conditioned to a variety of environmental stimuli. Operant conditioning studies have demonstrated the ability of healthy subjects to learn to regulate their heart rate (6, 18). There are no studies showing that these techniques can establish arrhythmic patterns in nonpatient populations; however, single-case studies have demonstrated the ability to control the frequency of cardiac arrhythmias through operant conditioning or biofeedback procedures (e.g., 12, 21).

From an etiological perspective, research on stress and conditioning suggest the role of autonomic arousal and learning in the development or exacerbation of various cardiac arrhythmias. Although additional research on psychobiological mechanisms of cardiac arrhythmias must be completed to clarify the specific role of these psychological factors, their influence should not be ignored.

PSYCHOLOGICAL INTERVENTIONS FOR CARDIAC ARRHYTHMIAS

The psychological interventions that have been employed in the treatment of cardiac arrhythmias include heart rate biofeedback, relaxation, me-

ditation, hypnosis, and psychotherapy (5, 25). Two basic treatment rationales provide the foundation for the use of these interventions. The first is related to relaxation and hypnotic procedures. In general, these procedures are assumed to reduce high levels of arousal that may trigger or exacerbate arrhythmias in certain patients. Theoretically, by teaching patients to produce a relaxed state or a low level of arousal, arrhythmic activity may be modified. The second major treatment, biofeedback, is a more specific approach to arrhythmia control. Through biofeedback the patient is provided with information, usually in the form of a digital display, a light, or a tone that indicates the status of his heart rate or cardiac conduction. The patient uses this information to develop strategies to produce specific cardiac responses. The desired outcome of this training is the self-regulation of a cardiac response in the absence of feedback in the patient's natural environment. The literature in this section will be discussed in an order reflecting the physiological mechanisms responsible for specific arrhythmias.

Single Ectopic Beats

Pickering and Miller (21) report findings from two well-documented cases. The first patient was a 14-year-old boy with a 2½-year history of premature ventricular contractions (PVCs). Therapeutic trails of various antiarrhythmic medications had been unsuccessful. During 30 sessions over six months, he learned to maintain a normal sinus heart rate rhythm. Feedback consisted of an ECG displayed on an oscilloscope. Occasional trials without feedback were introduced to test his ability to detect the PVCs. The ability to correctly identify changes in heart rhythm was never consistent, and he was unable to suppress the PVCs during the nine sessions. Following the tenth session, he obtained some control and during the subsequent nine sessions, the amount of time in normal sinus rhythm increased from 5.2 to 32.7 percent. This patient, however, was unable to maintain normal sinus rhythm without feedback.

The second case concerned a 35-year-old man who had experienced PVCs for one year. The PVCs primarily occurred sporadically, although during occasional symptomatic attacks each normal beat was followed by a PVC, resulting in a bigeminal rhythm. Training sessions were carried out on two separate occasions, 5 months apart. Each period of training occurred for 4 consecutive days with 4 training sessions per day. The goal of training was increased heart rate and the mode of feedback was a meter display of the cardiotachograph output. During the initial 16 sessions, the patient learned to raise his heart rate by 20 to 25 beats per minute (BPM) for short periods of time. This increase was associated with a suppression of the bigeminal rhythm. During the subsequent five months, the patient practiced increas-

ing his heart rate using a cardiac monitor at home. When he was reevaluated in the laboratory he was able to increase his rate by 20 to 40 BPM with or without feedback and he was capable of suppressing the bigeminal rhythm approximately 50 percent of the time. With feedback, he could modify his heart rate to any given level between 70 and 110 BPM. Toward the end of the second training phase which followed five months of practice at home, he was able to supress his PVCs 100 percent of the time. While both cases suggest the potential utility of biofeedback in the management of cardiac arrythmias (the second case more so that the first) as Pickering and Miller indicate "it remains to be seen whether such training confers any sustained therapeutic benefit."

Pickering and Gorham (20) studied a 31-year-old woman with an eight-year history of PVCs. The PVCs were parasystolic in rhythm and occurred with a frequency of 0 to 15 per minute. The patient was trained to speed up and to slow down her heart rate during 16 one-hour sessions. Feedback of PVCs was provided using a heart rate meter. During the first six sessions she was able to increase her heart rate approximately 4.2 BPM. During the final session, she could raise her heart rate by 25 BPM with or without feedback. As the patient learned to increase her heart rate, the threshold for the appearance of arrhythmias shifted from 79 BPM to 94 BPM. She was much less successful at heart rate slowing and was only able to achieve decreases of 1 or 2 BPM. No follow-up was reported.

Engel and Bleecker (12) investigated the effects of heart rate conditioning in a 27-year-old woman diagnosed as having Marfan's Syndrome—an inherited disease with abnormalities of the skeletal system, the eyes, and the cardiovascular system—and a seven-year history of progressive heart failure which had not been responsive to medical treatment. The patient experienced up to 20 PVCs per minute during two days of baseline measurements. The patient was taught to lower her heart rate for four days, to raise the rate for two days, to differentially raise and lower the rate for four days, and to maintain her rate within a defined range for four days. She averaged 15 PVCs per minute during the pretraining baseline recording. There was an immediate decrease to 5 PVCs/minute at the onset of training. It is difficult to explain this reduction in arrhythmic activity on the basis of heart rate training, since it occurred before she learned heart rate control. During the 14 days of heart rate training, her average frequency of PVCs dropped from five per minute to almost none. Nine months after training, several ten-hour ECG recordings were collected which demonstrated the absence of PVCs. At the follow-up visit, the patient was reported to be working steadily and taking no antiarrhythmic medication.

The use of feedback techniques for single ectopic heartbeats was further demonstrated by Weiss and Engel (26) in a study of eight patients. The subject sample included two females and six males who ranged in age from 36

to 77 years. These patients were hospitalized during the course of the experiment and had up to three 80-minute heart rate feedback training sessions daily. Visual feedback consisted of binary heart rate information and a meter that indicated the amount of time the patient was demonstrating control. Heart rate increase followed by heart rate slowing and differential heart rate increase and decrease were taught to the subjects. They had approximately ten sessions of training in each of these conditions; then they were trained to maintain their heart rate within a defined range. During this training, feedback availability was progressively limited. This procedure was employed to "wean" the subjects from the need for feedback. All eight subjects learned some degree of heart rate control. Five were able to control the frequency of PVCs in the laboratory, and four subjects showed decreased PVCs at a follow-up which in certain cases was 21 months post training.

There are two reports of nonbiofeedback interventions for the treatment of patients with single ectopic beats. Collison (10) reports the use of hypnosis with a 16-year-old boy who had a four-year history of frequent ventricular ectopic beats. The subject readily entered a deep trance and was given suggestions of increased relaxation and coping abilities. The number and length of sessions are unspecified. The patient was reported to have experienced complete relief and he was asymptomatic at a three-year follow-up. Unfortunately, no data on specific ECG changes were reported.

An additional study suggests that the Benson relaxation response may be effective in decreasing the occurrence of PVCs. Benson, Alexander, and Feldman (3) taught 11 subjects—two females and nine males ranging in age from 48 to 70 years—the relaxation technique. All of the subjects had a diagnosis of ischemic heart disease of at least one-year duration and documented PVCs for which none of the subjects was taking medication. They had one treatment session during which they were taught the relaxation response. They were then instructed to practice the technique for 10 to 20 minutes twice a day for one month. The frequency of their PVCs was monitored before training and after the month's practice. Reduced PVC frequency was documented in eight of the eleven patients. The average PVCs per hour for all subjects dropped from 151.5 to 131.7, or an average decrease from 2.5 to 2.2 PVCs per minute. Although this average decrease of 0.3 PVCs per minute is small, the decrease in three of these patients was over 85%. During sleep the average rate of PVCs per hour in these subjects decreased from 125.5 to 87.9.

Multiple Consecutive Ectopic Beats

Scott, Blanchard, Edmunson, and Young (23) report data from two single-subject experiments. One subject was a 46-year-old male who had a

20-year history of chronic tachycardia. After two baseline recording sessions, he was trained to decrease his heart rate for 22 sessions (40 minutes in length with 20 minutes of feedback). During the first 13 sessions a meter showing the amount of time the patient was able to decrease his heart rate below a criterion level was used for feedback. For the next nine sessions, the performance criterion was continuously reset in the desired direction to shape the patient's heart rate change. During the last two sessions the patient was reinforced for his attempts to return his heart rate to the preexperimental baseline level. The constant criterion procedure produced very little change in the patient's heart rate; however, the shaping procedures led to a decrease of 17 BPM. The patient's preexperimental heart rate averaged 80 BPM; during the last six trials of the shaping procedure, his heart rate averaged 72 BPM. When he was instructed to return his heart rate to baseline, it stabilized at 77 BPM. Following training the patient experienced less anxiety, returned to work, and spontaneously decreased his dosage of a minor tranquilizer.

In the same paper, a similar case is described of a 50-year-old man who had a 26-year history of anxiety neurosis including tachycardia. After two baseline recording sessions, this patient was trained to decrease his heart rate during 19 sessions (40 minutes in length with 20 minutes of feedback) using the shaping procedure. He was then instructed for eight trials to return his heart rate to baseline levels. His average baseline heart rate of 96 BPM dropped 14 BPM after 19 shaping trials. During the last 8 trials when the patient was retrained to his preexperimental baseline, his heart rate stabilized at 78 BPM. The patient reported that he felt stronger and less anxious, and that he was able to perform more household chores without tiring rapidly.

Blanchard and Abel (4) report a single-subject experiment in which a 30-year-old woman with a 16-year history of episodic tachycardia received 33 sessions of heart rate biofeedback training (20 minutes of feedback per session). She also participated in group psychotherapy and was stabilized on Dilantin (100 mg per day for brief seizure-like attacks) during the course of the study. Preceding the initiation of biofeedback treatment, she had discovered that her episodes of tachycardia were elicited by specific stressful stimuli. She successfully learned to decrease her heart rate an average of five beats per minute with and without visual heart rate feedback. She was also able to maintain her heart rate decrease in the presence of new stressful stimuli. The patient reported that her episodes of tachycardia had virtually disappeared by the end of the 33 training sessions.

Engel and Bleecker (12) report data from single-subject experiments with three patients who had various supraventricular tachycardias. The first patient was a 41-year-old man with an eight-year history of constant paroxys-

mal atrial tachycardia and a heart rate that averaged between 130 and 140 BPM. Treatment with medications had been ineffective. During 25 30-minute sessions, the patient was taught to slow his heart rate. For the first 17 sessions, feedback was a display of his electrocardiogram on a calibrated oscilloscope. For the final eight sessions the patient trained with binary heart rate feedback. The patient was able to slow his heart rate somewhat with the oscilloscope feedback, but he was more effective at regulating his heart rate with the binary feedback. By the seventeenth session he had established a new resting heart rate 15 BPM below his pretreatment level. During the subsequent five months, the patient's heart rate ranged from 60 to 75 BPM and his symptoms of congestive heart failure disappeared.

Engel and Bleecker report a second subject who had a sinus tachycardia. This patient was a 53-year-old woman with an average heart rate of 106 BPM who had not been observed to have a heart rate below 80 BPM for the four years prior to the study. She was trained to decrease her heart rate over 21 sessions. For the first 11 sessions she received constant visual binary heart rate feedback. For the final 9 48-minute sessions, the percentage of time that feedback was available was progressively reduced to help her become less dependent on visual feedback to control her heart rate. She was equally successful in lowering her heart rate with and without feedback. Her pulse dropped from 86 BPM at the start of the study to 68 BPM in later sessions. Along with this change, her blood pressure decreased from 140/80 mm Hg to 115/75 mm Hg.

The third case was a 36-year-old woman with a two-year history of one episode of paroxysmal atrial tachycardia (PAT) per month and occasional periods of sinus tachycardia. This patient was trained to slow her heart rate for 20 17-minute sessions and to increase her heart rate during an additional 10 sessions. During 8 sessions of differential heart rate training she was instructed to raise and then lower her heart rate. Although this patient was only able to minimally slow her heart rate, she was able to consistently increase it from 7 to 20 BPM. Surprisingly, she reported less anxiety when she had a faster heart rate. At a six-month follow-up, she reported only one episode of tachycardia, which she was able to control voluntarily.

Bleecker and Engel (7) employed a similar experimental procedure with three women and three men who had chronic atrial fibrillation. They ranged in age from 28 to 62 years and had symptoms for 2 to 20 years. All of them had rheumatic heart disease which had been stabilized with digitalis for at least three months. They were taught to increase their ventricular rate (VR) for ten sessions, to slow it for ten sessions, and to alternately speed and slow it for another ten sessions. All biofeedback sessions were 17 minutes long with continuous visual binary feedback of ventricular rate. Two subjects were consistently able to increase their rate. Two were better

at slowing their rate, and the remaining two subjects were best at differentially increasing and slowing their ventricular rates. On the average, subjects learned to increase VR by 9.5 BPM and to slow it by 3.5 BPM. All subjects were able to differentially increase and slow their ventricular contraction rate during the alternation phase of training. An analysis of their R-R intervals indicated that all subjects generated significant differences in the distribution of ventricular beats independent of ventricular rate. Two subjects received extensive training to decrease ventricular rate variability. While both were able to reduce this variability with feedback, neither subject showed any reduction of VR variability in the absence of feedback. This study demonstrates that digitalized patients with chronic atrial fibrillation can learn to control ventricular rate.

Although biofeedback is the most frequently used psychological technique for the treatment of tachycardia, meditation, psychotherapy, and hypnosis have also been used. Lown, Temte, Reich, Gaughan, Regenstein, and Hai (15) studied meditation as a treatment in a 39-year-old man with two episodes of ventricular fibrillation related to "nervous actvity." The patient was taught a Transcendental Meditation-like procedure and practiced this meditation twice daily while viewing his ECG on an oscilloscope. While meditating, the patient attempted to suppress his PVCs. After one week he was able to reduce their frequency from 11 per minute to 3 per minute. Nine months later he had no PVCs. The effect of meditation, however, was confounded by the feedback procedure, instituting regular exercise, verbalization of dreams, and antiarrhythmic drugs.

Collison (10) describes the use of hypnosis for the treatment of various tachycardias. A 36-year-old man with a ten-year history of paroxysmal ventricular tachycardia, which was unsuccessfully treated with quinidine and sedatives, was given relaxing hypnotic suggestions. He was seen for hypnotherapy daily for an unspecified length of time as an inpatient, and then weekly and biweekly for a total of six months. After treatment the tachycardia occurred only sporadically, and at three-year follow-up the patient reported only mild recurrences of the symptom.

Collison also used hypnosis with three women (22 to 35 years old) who suffered from paroxysmal atrial tachycardia (PAT). These patients received relaxation suggestions designed to help them cope with various life situations. The number and length of sessions were unspecified. One patient experienced immediate relief, a second patient showed a decrease in the frequency of episodes of PAT from several weekly to one every 10 to 12 weeks, and the third patient reported no change in symptoms. The long-term effects of this procedure were not reported.

Rahe and Christ (22) used psychotherapy and family therapy to treat an 11-year-old boy who developed paroxysmal ventricular tachycardia. The

parents monitored their son's pulse and observed his emotional state. This monitoring presumably led them to an understanding of the psychological factors influencing the tachycardia. At the same time, the patient was seen weekly for individual play therapy, the patient's mother was seen for weekly individual psychotherapy, and the parents had monthly couples therapy sessions. After a year of this treatment along with various antiarrhythmic drugs, the investigators noted improvement in the patient's levels of enjoyment and relaxation and in the parents' ability to interact with their son with confidence and firmness. Other than one stress-related episode of tachycardia, the patient was reported to have remained in good health.

Gruen (13) compared a group of recent myocardial infarction (MI) patients who were seen for 30-minute daily psychotherapy sessions ($n=33$) with a similar group of patients who were not seen in therapy ($n=35$). The occurrence of arrhythmias recorded during two daily 5-minute samples (7 to 11 days post-MI) were tabulated for both groups of patients. It was found that 15% of the treated patients as compared to 29% of the controls experienced ventricular arrhythmias. During this time, 5% of the treated patients and 23% of the controls had supraventricular arrhythmias. This decrease in incidence of arrhythmias along with decreases in reported weakness and depression, days of intensive care, and less anxiety when measured at four-month follow-up in the treatment group, supports the use of psychotherapy during recovery from an MI. Because of the sporadic and limited collection of ECG data, definite conclusions regarding the impact of psychotherapy on cardiac pathology in this group must await replication incorporating appropriate cardiovascular measures.

Psychological Interventions for Cardiac Arrhythmias Resulting from Disturbance of Impulse Conduction and Combined Impulse Formation and Conduction

Engel and Bleecker (12) studied three subjects with third-degree heart block who were treated with biofeedback training to speed ventricular rate. None of the subjects were able to consistently increase their ventricular rate. Bleecker and Engel (8) also used biofeedback to assist a 29-year-old woman with a 10-year history of Wolff-Parkinson-White (WPW) Syndrome. She was taught to lower her heart rate (26 sessions), raise her heart rate (15 sessions), and alternately increase and slow her heart rate (21 sessions). Following this she was taught to increase the frequency of WPW conduction and the frequency of normal conduction. For 17 minutes of each session she received binary feedback, which was presented to her visually during heart

rate training and aurally during heart rhythm training. At the end of this treatment, she could decrease her heart rate 3.4 BPM, increase her heart rate 2.5 BPM, and successfully change from increased to decreased heart rate or vice versa during 20 of 21 test sessions. She was also able to increase the proportion of normally conducted beats by 13% with and without feedback. Follow-up after ten weeks showed that she could continue to differentially control her cardiac conduction.

CLINICAL IMPLICATIONS

The clinical research literature is based on a small number of patients treated with biofeedback, relaxation, meditation, hypnosis, and psychotherapy. Definitive conclusions regarding the efficacy of these techniques with cardiac arrhythmias cannot be made. The studies are limited by the use of multiple interventions, which prevents the indentification of the active treatment components. Many reports do not describe the treatment setting (inpatient or outpatient), the length of treatment sessions and the duration of treatment, or the use of medication, including antiarrhythmic agents. Many of the case reports do not provide sufficient information to allow replication. The most serious clinical limitation is the lack of clinical outcome data which must include a description of generalization of treatment effects to the patient's home environment. However, given these limitations, some tentative conclusions can be drawn from this body of research.

Heart rate control achieved through heart rate feedback training results in the elimination or reduction in the frequency of PVCs in approximately 50% of the cases. The training procedure that appears to be most effective is one including training sessions in heart rate deceleration, acceleration, combined deceleration and acceleration, and sessions that train the patient to maintain his heart rate within a fixed range. The use of analogue visual feedback of the patient's ECG is not as effective as binary feedback. The effects of hypnotic procedures on ectopic beats are unclear. An anecdotal report suggests remission of arrhythmias in one case at three-year follow-up. However, this conclusion is not supported with ECG data, and further research is clearly needed in this area. Relaxation training for treating PVCs appears to be promising. The reported efficacy is based on short-term follow-up studies. However, this technique does appear to be effective in reducing the frequency of PVCs in certain patients. The type of patient who will respond has not been specified. Given the simplicity and low cost, the clinical use of relaxation training warrants serious consideration.

Heart rate biofeedback appears to be useful in the treatment of tachycardia. In a series of case studies, it appears that training in heart rate decele-

ration assists in the management of stress-related tachycardia. It also appears that biofeedback training for stress-induced tachycardia may be facilitated by teaching heart rate regulation in the presence of stressful stimuli. A binary feedback approach to train heart rate deceleration has been the most common therapeutic strategy. A continuous visual display of ECG is not as useful. The effects of nonbiofeedback procedures for tachycardia are difficult to evaluate. Unfortunately, few specific data were provided to describe changes in the frequency of target problems. These studies do suggest, however, that the use of hypnosis, self-monitoring of pulse and emotional level, and psychotherapy may be useful in the treatment of some types of tachycardia.

Biofeedback and other psychological techniques do not appear to be very useful in the treatment of third-degree heart block. However, increases in normal cardiac conduction and decreases in heart rate have been reported in one patient with Wolff-Parkinson-White Syndrome.

A comprehensive assessment of the variability and psychophysiological basis of arrhythmias should be undertaken prior to any attempts at intervention. This assessment can be instrumental in determining appropriate intervention strategies. Following assessment, relaxation skills might be taught and the effects of this training on target arrhythmias carefully monitored. The initial use of relaxation training rather than cardiac biofeedback is suggested because of the simplicity of administration and the clinical observation that relaxation skills are more easily learned than self-regulation of heart rate. Also relaxation training may assist the patient in gaining biofeedback-assisted cardiac control in the future. If the arrhythmias persist after the patient has learned and practiced relaxation, training in specific cardiovascular regulation could be attempted. The effects of biofeedback on specific arrhythmias must be continuously evaluated and treatment plans modified if persistent arrhythmic activity is observed.

FUTURE DIRECTIONS

Large-scale, well-controlled, long-term outcome studies are needed to evaluate the differential effects of various techniques with and without medication. Future research should describe diagnostic criteria and outcome measures and incorporate multidimensional evaluations to specify changes in the arrhythmias as well as changes in psychosocial and behavioral indicators of health. To determine the degree of generalization of laboratory-based arrhythmia changes, patients should be studied with long-term ambulatory ECG monitoring.

Changes in arrhythmias and/or heart rate must be carefully analyzed. Attention should be paid to the learning process and associated cardio-

vascular changes. Since these types of data are not an integral part of outcome studies, the combined process-outcome study should be a routine research strategy. The use of homogenous clinical populations would also help define optimal treatments for specific types of arrhythmias. Investigations of different types of arrhythmias might be fruitful. To date, major emphasis has been placed on the disorders of impulse. Sophisticated exploratory studies could be performed on the effects of various psychological procedures on conduction and combined impulse formation/conduction disorders.

At the clinical level, the various case reports indicating that psychological stimuli, both pleasant and aversive, are associated with various cardiac arrhythmias may hold implications for future diagnosis and treatment. Specifically, environmental stimuli that are potentially related to an activation of sympathetic arousal could be identified in a clinical interview or by using daily self-monitoring. As these stimuli are identified, their *specific* relationship to target arrhythmias could be assessed by ambulatory ECG monitoring. Once the existence or nonexistence of a relationship between stressors and arrhythmias is determined, an appropriate treatment plan could be designed. For example, if cardiac arrhythmias are noted following exposure to stress, it may be appropriate to treat the stress response in relation to the specific arrhythmia. In a hypothetical case, the intervention could be threefold: 1) Decrease general sympathetic arousal by relaxation training, 2) reduce general arrhythmic activity by biofeedback, 3) reduce stress-induced arrhythmias by using biofeedback or other behavioral techniques in the presence of specific stressors. Some patients may require all three procedures, while others may require only one or two of them. Although this proposed management strategy for cardiac arrhythmias requires empirical verification, it is not unreasonable to assume that this type of approach may facilitate the psychological control of arrhythmias in the future.

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The Comprehensive Handbook of Behavioral Medicine, Volume I

CHAPTER 4

Behavioral Approaches to Hypertension

C. BARR TAYLOR

Hypertension is an important health problem. The National Health Survey (45) has indicated that about 20% of the adult population is hypertensive, and lowering blood pressure has been demonstrated to reduce the mortality and morbidity from high blood pressure (77, 78). Furthermore, blood pressure reduction seems beneficial even for individuals with borderline hypertension (72). Although antihypertensive medications have a proven effectiveness in reducing blood pressure, such treatment suffers from several drawbacks: a) compliance to antihypertensive medication is low (37, 40), b) the side effects of the medication are sometimes unpleasant, and c) the cost can be prohibitive for some patients. Thus, alternative or supplementary modes of treatment are desirable. In recent years many studies have been undertaken to determine the usefulness of behavioral approaches as supplementary or alternative modes of treatment to reduce high blood pressure. In this chapter we will review the status of these approaches.

Behavioral approaches to hypertension include biofeedback, relaxation and related techniques, environmental manipulation, and other "general strategies" designed to reduce stress, like cognitive coping strategies and anxiety reduction techniques. Since the last two categories, environmental manipulation and general strategies to reduce stress, have not been extensively investigated in relation to blood pressure, they will not be reviewed in this paper.

METHODOLOGICAL PROBLEMS OF BLOOD PRESSURE STUDIES

Any treatment approach to essential hypertension must be considered in light of various methodological problems. Failure to adequately evaluate treatment led to the situation colorfully described by Weiss (82) in an early review of the hypertension literature:

> What has been done in an effort to reduce the blood pressure? Because of an ill-founded idea that protein was responsible for hypertension and kidney disease the patient was denied meat and eggs, and especially red meat, which for some reason was looked upon with particular dread. His diet was rendered even more unpalatable by the withdrawal of salt. Sympathy would doubtless have been extended to this half-starved fellow except that he probably was not able to eat anyway, his teeth having been extracted on the theory that focal infection had something to do with hypertension. Even before this he had sacrificed his tonsils and had had his sinuses punctured because of the same theory. In case some food had been consumed, the slight colonic residue was promptly washed out by numerous colonic irrigations, especially during the period when the theory of auto-intoxication was enjoying a wave of popularity. To add to his unhappiness, he was often told to stop work and exercise. Of course, he was denied alcohol and tobacco as well as coffee and tea...."

The most important considerations in determining the effectiveness of a particular therapy are the following.

Unspecified Therapeutic Effects

The unspecified therapeutic effects on blood pressure are well known. Such variables as reassurance (60), therapist expectancy (60), new technology (26), suggestion (79), and instruction (52) have been demonstrated to lead to significant and sometimes long-lasting reductions in blood pressure. For instance, in a classic study on unspecified effects on hypertension, Goldring and associates (26) pointed an "electronic therapeutic gun" armed with impresive but innocuous electronic equipment at patients who were told that the gun would reduce their blood pressure. Blood pressures fell an average of 20/14 mm Hg; in eight of the patients diastolic blood pressure fell below 90 mm Hg, an average diastolic fall of 25 mm Hg. In another study, Volini and Flaxman (79) told patients that a particular surgery would reduce their blood pressure when in fact the surgery was totally unrelated to the patient's blood pressure. The study suffered many method-

ological flaws (for instance, blood pressure measurement techniques were not described) and the results must be viewed with skepticism, but the magnitude of effects suggests the power of unspecified effects: Postoperative systolic blood pressure fell 58 mm Hg (27%), while diastolic blood pressure fell 46 mm Hg (31%) and remained down 13% at an average follow-up 5.5 years later.

In attempting to determine the time course of unspecified therapies, Taylor (75) graphed the change in blood pressure over time for placebos given as controls of hypertensive medication trials. The maximum placebo effect occurred from seven to ten weeks and had mostly disappeared by four months. One implication of these data is that follow-up at six months after end of treatment is necessary to adequately assess the effects of a blood pressure treatment.

Measurement Problems

Blood pressure measurement involves a number of variables that must be taken into consideration. The following factors have all been demonstrated to have an effect on blood pressure measurement.

Measurement Factors

Factors include size and type of cuff, placement of cuff, size of arm, position of patient (standing, sitting, lying), point of inflation, rate of deflation, criterion of measurement (4th vs. 5th Korotkoff sounds), training of examiner, hearing of examiner, how and when blood pressure is recorded.

Site and Person Factors

Furthermore, blood pressure is subject to many other variables like site of measurement (differences have been noted between home and clinic measurements), familiarity with measurement site (blood pressures decrease as individuals become familiar with the measuring area), person measuring blood pressure, hospital vs. nonhospital, etc. Blood pressure tends to be lower when subjects are relaxed and have previously exercised.

Law of Initial Values

According to the law of initial values, there is a frequent correlation between initial level of a physiological variable and amount of change relative

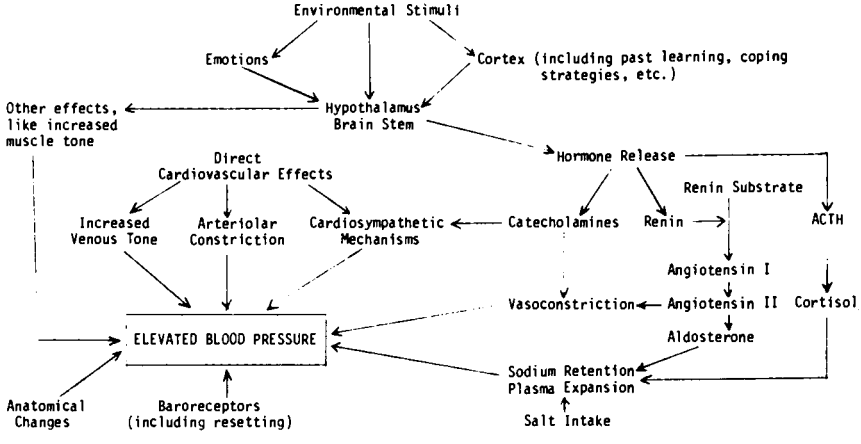
to a particular stimulus. For blood pressure subjects, this means that subjects with higher blood pressures would be expected to show greater changes. Jacob et al. (30) have recently developed a regression line for blood pressure changes by plotting decrease in systolic pressure against pretreatment systolic pressure and decrease in diastolic pressure against pretreatment diastolic pressure. In theory, any study demonstrating mean values "above" this regression line has a significant treatment effect, since most placebo studies fall below the regression line. This approach is a reasonable way to determine the "real" therapeutic effectiveness of a particular procedure.

Interacting Factors Controlling Blood Pressure

Blood pressure is regulated through a complex system of interactions between central and peripheral nervous system activities, endometabolic changes, and other processes. A summary of some of the important factors affecting blood pressure is presented in Figure 1. Many of the arrows are drawn in one direction only; in fact, feedback loops are present between many of these same variables. However, Figure 1 makes it clear how it is

FIGURE 1

Outline of Important Factors Leading to Elevated Blood Pressure*



*Adapted from Patel (49)

next to impossible to control all factors influencing blood pressure. While many of the neuroendocrinological factors can be measured, the environmental stimuli and other variables which also affect blood pressure are more difficult to measure. For instance, salt affects blood pressure, and while serum and total body salt can be measured, salt intake is more difficult to assess unless patients are in restricted research environments. A minor change in an individual's salt intake can dramatically affect his blood pressure. Similarly, other dietary factors, exercise, weight, stress, and habits all influence blood pressure and are difficult to specify. Medications not given specifically for blood pressure may affect blood pressure. Even discontinuing some blood pressure medications may have prolonged or paradoxical "rebound effects" on blood pressure.

Type of High Blood Pressure

The cause of the elevated blood pressure also influences the effect of a treatment. We would not expect a patient with renal vascular hypertension in which elevated renins are a major factor in causing the blood pressure elevation to show the same treatment response to a behavioral procedure as a patient with essential hypertension who has no identified medical problems contributing to the blood pressure elevation. Furthermore, many patients with essential hypertension suffer from undiagnosed medical problems that may be causing or contributing to their hypertension.

Given the many methodological problems facing blood pressure studies, it is not surprising that the results of studies using even the same procedures are sometimes significantly different or even contradictory.

BEHAVIORAL TREATMENT FOR HYPERTENSION

In this section, we will review the behavioral treatment of hypertension. We have divided the discussion into the following categories: 1) relaxation and related techniques; 2) biofeedback; and 3) combined treatments.

Relaxation and Related Techniques

Relaxation as an adjunctive treatment for hypertension has been recommended for many years. In 1930, Buck (16) wrote "we may conclude that sedation and relaxation are the two objectives to be sought in the treatment of hypertensive patients." He taught patients a five-minute relaxation pro-

cedure which he encouraged them to practice one to three times daily. His procedure did not, however, become incorporated into the standard treatment of hypertensives, and only recently have such procedures come under the scrutiny of careful investigation.

Relaxation and related techniques include: a) progressive relaxation and derivative programs, b) meditation techniques, including the "relaxation response," c) autogenic training, and d) hypnosis. Benson (8) has argued that all these techniques share similar procedures: The subject assumes a passive attitude in a comfortable position and quiet environment while using a mental device like repetition of a word, phrase, or image. For Benson this constitutes the "relaxation response." Benson suggests that the "relaxation response" elicits the so-called trophotropic response, a hypometabolic state that opposes sympathetic activity. We will review the evidence for this theory later.

Progressive relaxation and derivative techniques. This category includes techniques largely derived from Jacobson (31). In the 1930's, Jacobson devised a technique to help subjects relax which included extensive training in muscle tension and relaxation. Over the training period, sometimes lasting 50 sessions, subjects learned to relax most of the muscles in their body. Newer techniques use far fewer sessions, taped instructions, and other modifications to make Jacobson's technique more adaptable to current practice settings. Table 1A lists six studies in which progressive relaxation and derivative techniques have been used.

Jacobson first reported the effects of progressive muscle relaxation on blood pressure reduction (32). Of the three cases he reported, average drop in blood pressure within single sessions was 18 mm Hg systolic and 9 mm Hg diastolic. Deabler et al. (18) compared progressive relaxation with a combination of progressive relaxation and hypnosis. The progressive relaxation resulted in changes of about 10 mm Hg in systolic and diastolic blood pressures with the addition of hypnosis leading to slightly greater changes. Unfortunately, the design of this study was such that the effects of hypnosis plus relaxation were not directly compared with relaxation alone, confounding analysis of the additional benefits of hypnosis.

Brady et al. (13) used a metronome-conditioned relaxation procedure. In this study, subjects wore a miniaturized metronome set at 60 beats per minute. With each beat, the subjects were conditioned to relax. The patients undertook 18 to 75 30-minute sessions. Three of four patients studied showed significant reductions in blood pressure as compared to their baseline; one patient who continued practicing showed continued improvement six months later. Shoemaker and Tasto (66) used a taped progressive relaxation program lasting three sessions in a study comparing this procedure with a control and biofeedback. The progressive relaxation group showed

average systolic blood pressure reductions of 7 mm Hg and average diastolic changes of 8 mm Hg. No follow-up was obtained. In a subsequent study with normal volunteer patients, they found no significant changes in relaxation compared to controls told to sit quietly or to controls with no therapeutic activities (73). Since subjects only listened to a 30-minute relaxation tape three times over four days, they may not have had time to "learn" the relaxation technique. Taylor et al. (74) compared relaxation with a nonspecific control and a nontreatment control in medicated patients with essential hypertension. All groups spent an average of five 30-minute sessions with a therapist. The 11 patients in the relaxation group showed an average reduction in systolic blood pressure of 14 mm Hg and diastolic blood pressure of 4 mm Hg, which was significantly different from the nontreatment controls and from subjects given a supportive psychotherapy program matched for the same number of sessions. The relaxation group maintained its gain at six months, but the supportive therapy group showed continued improvement, and there were no significant differences at that time. Walsh et al. (80) compared pulse wave velocity biofeedback and relaxation with a tape using 24 essential hypertensive subjects. During the first phase of the study which lasted five weeks, the pulse wave velocity biofeedback group did better than the relaxation group. However, during the next phase in which both groups were given relaxation, the subjects first taught relaxation outperformed the subjects first taught biofeedback.

These studies suggest that progressive muscle relaxation can lead to systolic and diastolic blood pressure reductions of around 10 mm Hg, depending on the initial value of the blood pressure, and that these changes can be maintained at six months.

Meditation techniques: There have been a number of studies evaluating meditation techniques. In an early study, Datey et al. (17) used a yogic exercise called Shavasan to treat 47 hypertensive patients. The patients performed the exercise daily for 30 minutes and required up to three weeks to learn the technique. Twenty-two patients showed significant reduction in blood pressure, and the final blood pressure average was 148 mm Hg/86 mm Hg; 19 of 37 patients had drug dosages reduced by one-third or more. Ten of the 22 who did not respond were reported to have attended irregularly or failed to perform the exercise correctly. Furthermore, 65% of patients diagnosed as having essential hypertension and 42% of the patients with renal hypertension reponded, but none with arteriosclerotic hypertension responded.

Benson studied the effects of transcendental mediation on moderately hypertensive subjects (6). Subjects with blood pressure greater than 140 mm Hg or 90 mm Hg diastolic were recruited for the study. Patients continued to meditate daily for two weeks, using transcendental meditation proce-

Table 1A*: Relaxation

SOURCE	CLINICAL PROBLEMS	TYPE OF TREATMENT	DURATION OF BASELINE (B) AND TREATMENT (T)
Jacobson, 1939 (32)	Essential hypertension; average BP, 176/95 mm Hg	Progressive relaxation, outpatient	B: Values from single session of 45 min
Shoemaker & Tasto, 1975 (66)	Essential hypertension; average BP: E-1, 136/90 mm Hg; E-2, 132/90 mm Hg; C, 132/90 mm Hg	E-1: progressive relaxation E-2: biofeedback of BP C: monitoring, outpatient	B: 3 sessions T: 6 sessions of 80 min each in 2 wk
Brady et al., 1974 (13)	Essential hypertension; average diastolic BP, 104.6 mm Hg	Metronome conditioned relaxation, outpatient	B: 10 sessions over 4 wk T: 18—75 30-min sessions (50 additional sessions for 2 pts.)
Taylor et al., 1977 (74)	Essential hypertension; average BP, 146/95 mm Hg	E-1: progressive relaxation E-2: supportive psychotherapy C: medical management only (E-1 & E-2 also received medical management)	B: 2 mo. T: 5—6 wk (practice relaxation daily)
Tasto et al., 1976 (73)	Normotensive volunteers E: baseline 121/70 C: 120/70	C-1: no therapy C-2: read magazine art. E: progressive relaxation	C-1: measurement only C-2: 3, 30-min. sessions with tape E: 3, 30-min. sessions over 4 days
Walsh et al., 1977 (80)	24 hypertensive subjects. Half on medication. Overall baseline 147/94	Phase 1: E-1, biofeedback of pulse wave velocity; E-2, relax with tape Phase 2: E-1 & E-2, given both biofeedback and relaxation	B: 1 session Phase 1: E-1, once/wk for 5 wk E-2, once/wk for 5 wk Phase 2: E-1 & E-2, given 5, 30-min sessions

*These tables are modeled after those of Blanchard and Miller, 1977 (12).

NO. OF PATIENTS	TYPE OF STUDY	RESULTS	FOLLOW-UP
3	Case report	Average drop in BP in single session: systolic, 18 mm Hg; diastolic, 9 mm Hg	None
15:5 per condition	Controlled group outcome study	Average change in BP: Systolic: relaxation, -7 mm Hg; biofeedback, 0 mm Hg; control, -2 mm Hg; Diastolic: relaxation, -8 mm Hg; biofeedback, -1 mm Hg; control, 0 mm Hg; 4/5 relaxation subjects showed improvement	None
4	Single-subject experiments	3/4 patients showed significant reductions in treatment as compared to baseline, approximately 8 mm Hg reduction in diastolic BP	2 wk–6 mo Improvement maintained with continued practice
E-1: 10 E-2: 10 C: 11	Controlled group outcome	E-1: reduced both systolic BP ($p<.05$) & diastolic BP ($p=.054$) more than E-2 or C; E-2=C E-2: △BP: systolic, -14 mm Hg; diastolic, -5 mm Hg	6 mo: gains maintained by E-1, C reduced systolic BP by 7 mm Hg
C-1: 10 C-2: 10 E: 10	Controlled group outcome	Pre to post treatment BP -2/-1 -2/-2 -3/-0	None
Phase 1: 24 Phase 2: 16	Single group outcome	Phase 1: E-1: -24/-13 E-2: -7/-6 Phase 2: Biofeedback during Phase 1: -21/-10; pts. taught relaxation during Phase 1: -6/-5	1 year: subjects taught biofeedback during Phase 1: -18/-9; subjects taught relaxation during Phase 1: -10/-10

Table 1B: Meditation

SOURCE	CLINICAL PROBLEMS	TYPE OF TREATMENT	DURATION OF BASELINE (B) AND TREATMENT (T)
Datey et al., 1969 (17)	Hypertension, essential, renal, and arteriosclerotic; average BP: nonmedicated patients, 185/109 mm Hg; medicated patients 149/94 mm Hg	Yogic exercise emphasizing breathing, meditation, and passive relaxation, outpatient	B: not given T: 30-min daily sessions for approx. 3 wk
Benson et al., 1973 (4)	Hypertension; all pts. receiving antihypertensive medications; average BP, 150/93 mm Hg	Meditation & passive relaxation ("transcendental meditation")	B: 6 wk T: 2 wk (continued to meditate daily)
Benson et al., 1974 (6)	Hypertension; all pts. receiving medication: average BP, 147/92 mm Hg	Meditation & passive relaxation ("transcendental meditation"), outpatient	B: 5½ wk T: 2 wk (continued to meditate daily)
Blackwell et al., 1976 (9)	Essential hypertension; all receiving medication; average BP: home, 153/102 mm Hg; clinic 139/98 mm Hg	Meditation & passive relaxation ("transcendental meditation")	B: 10 wk T: 4 days (2 hr/day) continued to practice daily
Stone & DeLeo, 1976 (70)	Hypertension, newly discovered cases; average BP 147/94 mm Hg	Meditation & passive relaxation, outpatient	B: 14 days T: 5 daily sessions of 20 min, continued to practice daily
Pollack et al., 1977 (51)	Essential hypertension, 9 on medication, average BP, 115.0/96.2 mm Hg	"Transcendental meditation"	B: 3 mo T: 6 mo Freq?
Benson et al., 1974 (7)	Borderline hypertension, (newly discovered cases) average BP, 147/95 mm Hg	Meditation & passive relaxation (elicitation of "relaxation response"), outpatient	B: 6 wk T: 2 wk (continued to practice daily)
Peters et al., 1977 (50)	Normotensive volunteers	C-1: control C-2: sit quietly E: relaxation response	C-1: measurement only C-2: 2 wk (practiced 2 times daily) E: 2 wk (practiced 2 times daily)

NO. OF PATIENTS	TYPE OF STUDY	RESULTS	FOLLOW-UP
47:10 not receiving drugs; 37 receiving drugs	Single group outcome	22 pts. showed significant reduction in BP (including 9/10 not receiving medication); final BP average, 148/86 mm Hg; 19/37 had drug dosage reduced by 1/3 or more	None
30	Single group outcome	No effects on diastolic BP; systolic BP dropped to these values at various follow-up times; during treatment, 142 mm Hg; 6 wk, 140 mm Hg; 9 wk, 125 mm Hg; for 9 pts. who stopped meditating, BP returned to baseline levels in 4 wk	9 wk: continued improvement
14	Single group outcome	Postmeditation training change in BP (after 30 days); systolic, -10.6 mm Hg; diastolic, -5 mm Hg	20 wk: no further improvement after 30 days, but improvement maintained
7	Single group outcome	Postmeditation training changes in BP: systolic, home, -7.5 mm Hg; clinic, -4 mm Hg; diastolic, home, -6 mm Hg; clinic, -1.5 mm Hg; 5/7 showed significant improvement; 6/7 less anxious by measurement	6 mo: 3/6 had improved change in BP about 3-4 mm Hg
C: 5 E: 14	Controlled group outcome	After 6 mo of meditation; systolic, 144 mm Hg; E, 132 mm Hg; diastolic, C, 93 mm Hg, 84 mm Hg	6 mo
20	Single group outcome	Pre-post after 6 mo of therapy -4/-2	
22	Single group outcome	Postmeditation training BP (after 30 days), 140/91 mm Hg	
C-1: 39 C-2:39 E: 58	Controlled group outcome	From first 4 wk of baseline to last 4 wk of study: C-1, -0.5/-1.2 C-2, -2.6/-2.0, E, -6.7/-5.2	

dures. In the first study, all patients received antihypertensive medication. The initial blood pressure was 150 mm Hg/93 mm Hg. The meditation procedure had no effect on diastolic blood pressure; systolic blood pressure had dropped to 125 mm Hg at nine weeks for the 31 subjects who remained in the study at that time. In a subsequent study with borderline hypertensive patients who also practiced transcendental meditation for two weeks, blood pressures dropped from an average baseline of 147 mm Hg/92 mm Hg to 136 mm Hg/90 mm Hg. The improvement was maintained at 20 weeks.

Blackwell (9) replicated Benson's study using seven subjects on a stable course of antihypertensive medication. Blood pressures were measured both in the clinic and at home. After 9 to 12 weeks of beginning meditation, the mean decrease for the whole group was 1.5 from an average baseline of 139 mm Hg/98 mm Hg. At the follow-up six months later, three of four subjects showed significant decrease in diastolic or systolic blood pressures as measured in the clinic and at home. However, Pollack (51) studied nine hypertensive patients controlled on medication with an average baseline blood pressure of 115 mm Hg and found no change in blood pressure after three months of meditation.

Stone and DeLeo (70) studied a technique derived from Zen Buddhist meditation. The patients were told to sit in a chair in an upright position, loosen tight clothing, relax their muscles, and concentrate their minds on breath counting cycles. After five 20-minute instruction sessions, they were asked to repeat the technique twice daily. After six months of treatment, the treatment group showed a reduction in blood pressure of 15 mm Hg/11 mm Hg, reported to be statistically significant at the .05 level compared to both the control group and its own pretreatment. (Since the control group had only 5 subjects compared to 14 for the treatment group, the t-test procedure they used for statistical significance is misleading.)

Benson (8) has published a number of studies on the effects of his relaxation response. Twenty-two newly discovered hypertensives with an average blood pressure of 147 mm Hg/95 mm Hg were taught the relaxation response. They practiced daily for two weeks. Postmeditation training, blood pressure was 140 mm Hg/91 mm Hg. These changes were maintained at 25 weeks. In another study, Peters (50) taught relaxation response to normotensive volunteers who were instructed to practice the procedure. The blood pressure fell from a mean of 120 mm Hg/79 mm Hg to 113 mm Hg/74 mm Hg, a significantly greater change than controls who sat quietly for the same length of time, and untreated controls.

The results from the meditation studies are confusing. The same meditation procedure has a significant effect in one study and little effect in another. However, in comparing the two most disparate studies, Datey et al. (17) and Pollack (51), the differences in results can largely be accounted for

by the significant differences in initial blood pressure (Datey's mean for nonmedicated subjects was 185 mm Hg/109 mm Hg and for medicated subjects was 149 mm Hg/94 mm Hg, while Pollack's was only 115 mm Hg/96 mm Hg). The law of initial values would suggest that Datey's group would be expected to have far greater changes than Pollack's; in fact, none of the treated groups in any of these studies reached the mean systolic levels that Pollack's group started with. We might conclude that subjects with already well-controlled blood pressure may not be expected to show further changes, except that Peters et al. (50) found significant relaxation response effects in a normotensive population. The relaxation response may have greater effects on reducing blood pressure than practicing transcendental meditation, but this is unlikely since the two procedures are very similar. Some of the methodological issues discussed earlier may have led to the difference in results. For instance, we have no direct information on how much individuals practiced in the two groups.

Autogenic training. There have been several studies reported in the English literature on the effects of autogenic training on blood pressure. Luthe (39) reports changes on 10–20% systolic and 5–10% diastolic for individuals who practiced this technique, but the details of his study are not given. Klumbies and Eberhardt (35) treated 26 patients with essential hypertension with autogenic training only. Blood pressure dropped from a mean of 165 mm Hg/100 mm Hg to a mean of 130 mm Hg/80 mm Hg after four months of treatment. Since the study was only reported as an abstract, the details of the design are too scanty to assess the validity of these results.

Hypnotic techniques. Deabler et al.'s (18) study showed an added effect of hypnosis when added to progressive relaxation. Friedman et al. (22) reported that 13 subjects taught relaxation with hypnosis showed changes of 15 mm Hg/9 mm Hg at the end of treatment. At the end of one month, these changes were 11 mm Hg/7 mm Hg. However, there are too few studies to determine the specific effects of hypnosis on blood pressure.

Summary of Relaxation and Related Techniques

There have been an impressive number of studies focused on the effects of relaxation and related techniques in reducing blood pressure for both essential hypertensive and normotensive subjects. These techniques have been demonstrated to have small, albeit significant changes relative to various types of controls. However, only four of the studies used nontreatment controls and only two of these provided follow-up for six months or longer. Thus, the evidence is suggestive but not conclusive that relaxation has a specific effect on blood pressure reduction.

How Does Relaxation Work?

Since relaxation has a suggestive demonstrated effect on reducing blood pressure, the question is raised as to how this may occur. Theoretically, relaxation could reduce blood pressure through most of the factors listed in Figure 1, e.g., it could have direct effect on reducing muscle tension, the reaction of an individual to environmental stimuli, or the effect of the hypothalamus on releasing hormones that affect blood pressure. As mentioned previously, Benson suggests that the "relaxation response" elicits the so-called trophotropic response. Hess (28) first observed that parasympathetic and sympathetic branches of the autonomic nervous system were not confined to visceral targets but also influenced somatic functions, including skeletal muscle activity and cerebral cortical function.

Through direct stimulation of trophotropic centers in the cortex and hypothalamus, Gellhorn and others (23, 24) found that heart rate, blood pressure, and sweat were reduced, while pupillary constriction occurred and gastrointentinal motor and secretory functions increased. The EEG became synchronized, skeletal muscle tone was increased, and the shivering response was blocked. Frequently these were accompanied by inactivity, drowsiness, and sleep. On the other hand, stimulating ergotropic centers led to increased cardiac rate, blood pressure, and sweat secretion, pupil dilation, inhibition of gastrointestinal motor and secretory functions, dysynchrony of the EEG, increased skeletal muscle tone, arousal, heightened activity and emotional responsiveness—functions that seemed to be opposite to those of the trophotropic response. Furthermore, other studies have demonstrated reciprocal relationships between ergotropic and trophotropic systems. Thus, Benson's notion is that the relaxation response activates trophotropic responses which lead to the types of anatomic, somatic, and behavioral effects mentioned above. In fact, there is no direct evidence that relaxation or any related techniques actually elicit trophotropic responses.

In one study, Stone and DeLeo (70) showed declines in furosemide-stimulated peripheral renin activity (PRA) and in plasma dopamine betahydroxylase (DBH) in individuals who practiced a meditation technique, both responses consistent with a trophotropic response. However, Herxheimer (27) notes that in the Stone study, furosemide-stimulated plasma-renin activity decreased in both treated and control groups, and the mean decrease was actually greater in the controls than in the treated groups. The fact that PRA decreased in those controls whose blood pressure did not change argues against adrenergic changes as the mechanisms for blood pressure decrease. Furthermore, DBH is not a reliable measure of adrenergic function (81). Thus, the Stone article does not argue convincingly for meditation leading to peripheral adrenergic changes. In another study on the effects of

meditation on blood pressure, Pollack (51) found plasma renin activity constant for both controls and meditation subjects. Further studies will have to be undertaken to clarify the mechanisms of how relaxation may affect blood pressure and to test the relaxation response hypothesis.

Jacob et al. (30) reanalyzed Jacobson's data to determine if there was a correlation between EMG and blood pressure. They found a significant correlation between decrease in muscle microvoltage and decrease in systolic and diastolic blood pressures. Both coefficients were significantly different from zero ($p < .0024$ and $p < .01$ using Spearman ranks). However, this does not indicate that the muscle relaxation per se led to the change in blood pressure, for EMG changes may merely have accompanied other physiological changes. Several studies have demonstrated that muscle relaxation does not necessarily lead to reduced blood pressure, e.g., polio victims with muscle paralysis show elevated blood pressure. At this point, we can only speculate on how relaxation and related techniques may affect blood pressure.

How Much and How Long Should Subjects Practice?

A major flaw in all the studies reported thus far has been the lack of direct evidence that subjects have learned the technique and practiced it as prescribed. Lacking this data we can only guess how much and for how long subjects should practice. The relative minor changes in blood pressure achieved by one or two session programs suggest that subjects must, at least, practice longer than this.

Biofeedback

Since the recent interest in autonomic conditioning of the nervous system spurred by Miller's (43) early studies, there have been many studies comparing the effects of relaxation and biofeedback to determine the effects of biofeedback on blood pressure. A number of ingenious procedures have been developed to provide biofeedback training:

Constant pressure cuff system. In this system, developed first by Tursky and colleagues (76), binary (yes/no) feedback is provided for increases or decreases in pressure at each beat relative to the pressure of the cuff. The cuff is inflated to a level where 50% of heartbeats exceed cuff pressure; with the result that 50 percent of the heartbeats produce audible Korotkoff sounds.

Standard pressure cuff system. In this system, subjects are provided feedback on a minute-by-minute basis, with blood pressure readings obtained from a standard blood pressure cuff. The system has the advantage of being rapid to use and inexpensive, but repeated cuff inflation can lead to peripheral edema in the arm.

Pulse velocity biofeedback. In this procedure pulse velocity as measured by transit time between the R wave on the ECG and the uptake of ths pulse pressure measured at the radial or other pressure spot is used to provide biofeedback to subjects. Pulse wave velocity correlates with blood pressure according to some authors.

Subjects have been provided with verbal praise, monetary rewards, and pleasant scenes as rewards for lowering pressure, although the typical procedures involve merely verbal praise and support from the experimenter. A change in tone or a color displayed on a screen may also act as a reinforcer for blood pressure change.

The results from 14 studies of biofeedback procedures are reported in Table 2. Most of the studies have reported immediate or short-term effects; there are no long-term follow-up studies.

In an early study, using systolic blood pressure feedback, Benson et al. (4) demonstrated an average decrease in systolic blood pressure of 16.5 mm Hg for five or seven subjects. Also, in an early study, Miller (42) demonstrated immediate diastolic blood pressure decrease of 21 mm Hg. In other preliminary studies, Schwartz demonstrated systolic change of -7 mm Hg. Blanchard et al. (11) simply provided minute-by-minute feedback of subject's systolic blood pressure using a routine cuff and demonstrated changes of 9 mm Hg to 51 mm Hg with an average of 26 mm Hg with four subjects with essential hypertension. Follow-up was short, from one to four weeks, but three of the four patients maintained at least half of their posttreatment gains. This study is significant in that it shows a relatively simple procedure yielding the same results as do more sophisticated and complicated ones. None of these studies attempted to have the patients learn to generalize their blood pressure training from the laboratory or clinic to the home. However, Krist and Engel (36) taught their subjects to reduce their blood pressure at home and found that three or four patients maintained changes of systolic blood pressure of around 18 mm Hg and diastolic of around 7.5 mm Hg at two or three months. The small number of subjects in this study precludes any definitive statements about the effect of home practice but suggests that there may be an effect. The results of other studies on biofeedback alone or biofeedback compared with other procedures are presented in Table 2.

From this table it appears that biofeedback procedures can produce immediate systolic changes up to 25 mm Hg and immediate diastolic changes

of up to 16 mm Hg. Unfortunately, there are no long-term follow-ups and too few controlled studies to help us make a statement about the usefulness of this procedure or to compare it with other procedures.

The significance of biofeedback as a specific learning phenomenon has been questioned. Brener (15) argues that biofeedback elicits a general therapeutic response akin to any unspecified response. On the other hand, Schwartz et al. (59) have clearly demonstrated that patients can both raise and lower their blood pressure; if Brener's argument were valid, we would expect changes in one direction only. The necessity of blood pressure feedback as the active part of biofeedback has also been questioned. Redmond et al. (52) demonstrated that subjects can both raise and lower their blood pressure without biofeedback. This finding is consistent with other studies that have questioned the specific effect of feedback versus setting and other unspecified variables.

Combined Treatments

Combined relaxation techniques have shown the greatest long-term effects on blood pressure. In a series of studies, Patel has provided evidence for the effectiveness of yoga meditation technique combined with GSR biofeedback. In the first study (46), using a single group, patients were instructed to relax in the supine position and to concentrate on phrases like "relax with each exhalation." Patients also received feedback of their GSR by means of an auditory signal. Patients were also told the results of their blood pressure at the end of the sessions. They practiced these techniques three times each week with the therapists and were also told to practice at home. After treatment, three months later, the mean reduction of blood pressure was 25 mm Hg/14 mm Hg from a mean pretreatment of 159 mm Hg/100 mm Hg, a statistically significant change. In her next study, Patel evaluated the effects of her treatment using a controlled group outcome (47). The experimental group received the same treatment as above, the control group was asked to rest quietly for 30 minutes. The average decrease in blood pressure of the treated group was 20 mm Hg/15 mm Hg from a pretreatment level of 168 mm Hg/100 mm Hg, significantly different from the control group who showed changes of only 1 mm Hg/2 mm Hg at the end of treatment. Most significantly, the treated group maintained these changes at the end of 12 months. This study was criticized for not randomizing the subjects to treatment, so in a third study, Patel randomized her subjects and added a crossover condition in which the controlled group was treated after six weeks (48). The treated group showed changes of 25 mm Hg/15 mm Hg from a baseline of 168 mm Hg/100 mm

Table 2: Biofeedback

SOURCE	CLINICAL PROBLEM	TYPE OF TREATMENT	DURATION OF BASELINE (B) AND TREATMENT (T)
Brener & Kleinman, 1970 (14)	5 Normotensive	Systolic, biofeedback	B: None T: 2 sessions
Schwartz, 1972 (58)	40 Normotensive	Systolic rate pattern biofeedback	B: None T: 1 session
Benson et al., 1971 (2)	Essential hypertension; average baseline systolic BP, 165 mm Hg	Biofeedback of BP, outpatient	B: 5—16 daily sessions (av. 11) T: 8—34 daily sessions (av. 22)
Miller, 1972 (42)	Essential hypertension; average baseline diastolic BP, 97 mm Hg	Biofeedback of BP, inpatient & outpatient	B: 26 sessions in 6 wk T: 37 sessions in 3 mo
Elder et al., 1973 (19)	Essential hypertension; average baseline BP: E: 153/104 mm Hg; C-1: 148/108 mm Hg; C-2: 150/95 mm Hg	E: biofeedback of BP+ social reinforcement for lowering C-1: biofeedback of BP C-2: monitoring BP, inpatient	B: 1 session T: 7 sessions in 4 days
Schwartz & Shapiro, 1973 (59)	Essential hypertension; average baseline diastolic BP, 102 mm Hg	Relaxation instructions + biofeedback of BP, outpatient	B: 5 daily sessions T: 15 daily sessions
Blanchard et al., 1975 (11)	Essential hypertension; average baseline systolic BP, 154 mm Hg	Biofeedback of BP, outpatient	B: 4 sessions T: 5—13 sessions
Elder & Eustis, 1975 (20)	Hypertension; average baseline BP, 147/85 mm Hg	Biofeedback of BP, outpatient	B: 1 session T: 20 sessions in 12—82 days
Kristt & Engel, 1975 (36)	Essential hypertension; average baseline BP, 163/95 mm Hg	Biofeedback of BP + home monitoring of BP & practice, inpatient	B: 5 wk/4 day measures at home T: 3 wk

NO. OF PATIENTS	TYPE OF STUDY	RESULTS	FOLLOW-UP
5	Single group outcome	Pre-post systolic: -17	None
20	Single group outcome	Pre-post systolic: -7	None
7—6 receiving medication	Single group outcome	Average decrease in systolic BP, 16.5 mm Hg, 5/7 patients showed significant response	None
1	Systematic case study	Diastolic BP decreased to 16 mm Hg; antihypertensive medications stopped	None
C-1: 6 C-2: 6 E: 6	Controlled group outcome	Systolic BP: E=C-1=C-2 Diastolic BP: E<C-1<C-2 E: diastolic BP=80% of baseline C-1: diastolic BP= 93% of baseline 4/6 patients in E showed significant response	1 wk differential dropout rate; E group apparently maintained gains
7	Single group outcome	No overall change in diastolic BP; 1/7 patients showed reduction of 14 mm Hg	None
4; 2 receiving medication	Single subject experiments	All 4 patients showed decreases in systolic BP during feedback training (9-51 mm Hg; average 26 mm Hg)	1—4 wk, 3/4 patients maintained 65% of gains
22	Single group outcome	Average decrease: systolic BP, 7.8 mm Hg; diastolic BP, 6.5 mm Hg; 9/22 patients showed significant decrease	2 mo on 4 patients no maintenance of gains
5 single (baseline data on 4); all receiving medication	Single group outcome	Average decrease; systolic BP, 18 mm Hg; diastolic BP, 7.5 mm Hg; all 4 patients showed decreases in systolic or diastolic BP; all 5 patients showed ability to lower BP at home	2 or 3 mo: 3/4 patients maintained gains

TABLE 2 (continued)

SOURCE	CLINICAL PROBLEM	TYPE OF TREATMENT	DURATION OF BASELINE (B) AND TREATMENT (T)
Kleinman & Goldman, 1974 (33)	Essential hypertension (newly diagnosed); average baseline BP, 167/108 mm Hg	Biofeedback of BP, outpatient	B: None T: 9 sessions
Fey & Lindholm, 1975 (21)	Normotensive	E-1: Reinforced systolic BP increase E-2: Reinforced systolic BP decrease E-3: Random feedback C: No feedback	3 one-hour treatments over 3 days
Surwit & Shapiro, 1977 (71)	Essential hypertensive; E-1: 157/95 E-2: 155/94 E-3: 156/92	E-1: systolic biofeedback E-2: EMG biofeedback E-3: Relaxation response	10 sessions over 5 wk
Richter-Heinrich et al., 1975 (54)	Essential hypertension; 141.0	E: biofeedback with visual rewards C: given rewards noncontingent on BP but yoked with E	20 conditioning trials over 4 days
Goldman, et al., 1975 (25)	Male hypertensives 167/109	Systolic biofeedback	9 weekly sessions
Friedman & Taub, 1977 (22)	Essential hypertension, medicated and nonmedicated	E-1: hypnosis & biofeedback E-2: hypnosis only E-3: biofeedback of systolic/diastolic visual BP C: measurement only	B: none E-2&3: 7 training sessions

Also see Shoemaker & Tasto (66), Table 1A

NO. OF PATIENTS	TYPE OF STUDY	RESULTS	FOLLOW-UP
7, none receiving medication	Single group outcome	Average decrease: systolic BP, 6 mm Hg diastolic BP, 15 mm Hg	None
5 in each group	Controlled group outcome	E-2: -10 systolic E-1, E-3, C: No significant changes	None
E-1: 8 E-2: 8 E-3: 8	Single group outcome	Decreases, last training session to 1 wk follow-up: E-1: -0/-5.6 E-2: -2.1/-0 E-3: -0/-0 (All patients showed major drop during baseline before treatment began)	None
E: 20 C: 10	Controlled group outcome	Pre-post training E: 15% C: 8%	None
7	Single group outcome	Pre-post, -8/-15	None
E-1: 10 E-2: 13 E-3: 13 C: 11	Controlled group outcome	Decreases at end of 7 wk: E-1: -0.8/-1.5 E-2: -14.9/-9.3 E-3: -7.7/-5.8 C: -2.4/-2.6	Decreases at 1 mo: E-1: -0.3/-5.8 E-2: -10.6/-7 E-3: -9.6/-2.3 C: +1.9/-1.9

Table 3: Combinations

SOURCE	CLINICAL PROBLEM	TYPE OF TREATMENT	DURATION OF BASELINE (B) AND TREATMENT (T)
Deabler et al., 1973 (18)	Essential hypertension; average BP: E(no drug), 163/96 mm Hg C(no drug), 155/95 mm Hg E(drug), 58/95 mm Hg	Progressive relaxation + hypnosis, inpatient	B: 1 30-min session T: 9 30-min sessions in 5 days
Moeller & Love, 1974 (44)	Essential hypertension; average baseline BP: 150/100 mm Hg; all receiving antihypertensive medication	Biofeedback of frontalis EMG + autogenic training, outpatient	B: 2 sessions T: 17 (1/wk)
Love et al., 1974 (38)	Essential hypertension; average baseline BP, 162/106 mm Hg	Biofeedback of frontalis EMG + various relaxation training; controls: monitoring of BP for 4 wk, outpatient	B: 1 session T: 16 (1 or 2 sessions per wk)
Russ, 1974 (55)	Hypertension; average baseline BP: E-1: 138/92 mm Hg; E-2: 164/109 mm Hg; C: 162/103 mm Hg	E-1: progressive relaxation for 2 sessions then EMG frontalis feedback for 9 sessions E-2: BP biofeedback C: false feedback	B: 2 sessions T: 11 sessions
Patel, 1973 (46)	Hypertension, average baseline BP: 159/100 mm Hg; all receiving antihypertensive medication	Biofeedback of GSR + passive relaxation training + meditation, outpatient	B: variable, at least 3 sessions T: 36 half-hr sessions over 3 mo
Patel, 1975 (47)	Hypertension, average baseline BP: E: 159/100 mm Hg; C: 163/99 mm Hg; 18 of each group receiving medication	E: Same as above C: resting quietly for 30 min, outpatient	B: 3 sessions T: 36 half-hr sessions over 3 mo
Patel & North, 1975 (48)	Hypertension; average baseline BP: E: 168/100 mm Hg; C: 169/101 mm Hg; 18 of each group receiving medication	E: Same as above C: Same as above, outpatient (BP taken by "blind" examiner)	B: 3 sessions T: 12 half-hr sessions over 6 wk

See Table 2 - Friedman

NO. OF PATIENTS	TYPE OF STUDY	RESULTS	FOLLOW-UP
E(no drug) 6 C(no drug) 6 E(drug) 9	Controlled group outcome	C(no drug): no change E(no drug): greater reduction in BP with hypnosis than with relaxation training Reduction in BP: systolic: relaxation; no drug, 10%; drug, 13%; hypnosis: no drug, 16%; drug, 16%; diastolic: relaxation, no drug, 15%; drug, 5%; hypnosis: no drug, 19%; drug, 8%	None
6	Single group outcome	Average decrease; systolic BP, 18 mm Hg; diastolic BP, 12 mm Hg; 5/6 patients showed significant decreases	None
40(27 completed treatment); 10 controls	Controlled group outcome	Average decrease: systolic BP, 15 mm Hg; diastolic BP, 13 mm Hg; control group showed essentially no change in 4 wk	8 mo: (N = 23) further decreases in BP; systolic, -6.5 mm Hg; diastolic, -4 mm Hg
E-1: 5 E-2: 3 C: 2	Controlled group outcome	Groups too small for any significant difference; E-1: average decrease in BP: systolic, 8 mm Hg; diastolic, 8 mm Hg; 3/5 patients showed improvement	None
20	Single group outcome	Average decrease in BP: systolic, 25 mm Hg; diastolic, 14 mm Hg; 16/20 patients showed significant response; 12/20 also had reduction in medication	None
E: 20 C: 20	Controlled group outcome	Average decrease in BP: systolic, E, 20 mm Hg; C, 1 mm Hg; diastolic, E, 14 mm Hg; C, 2 mm Hg; 12/20 pts. in E had reduced medication	12 mo: E, 144/87 mm Hg; C, 164/98 mm Hg
E: 17 C: 17	Controlled group outcome study with crossover (controls treated at end of experiment)	Average decrease in BP: systolic: E, 26 mm Hg; C, 9 mm Hg; C after treatment, 28 mm Hg; diastolic: E, 15 mm Hg; C, 4 mm Hg; C after treatment, 16 mm Hg	4 mo for C; 7 mo for E: 146/86 mm Hg

Hg, significantly better than the nontreated group mean changes. After treatment, the control group mean changes were 15 mm Hg/16 mm Hg. These changes were maintained at seven months for the originally treated group and at four-month follow-up for the treated control group. Two other studies listed in Table 3 have been reported either at meetings or informally and too little data is available on the procedures to allow for any statement as to the meaningfulness of the results.

Deabler's subjects, given progressive relaxation and then hypnosis, showed changes (18) of 16% systolic blood pressure for subjects on no drugs and 19% systolic blood pressure in patients with essential hypertension for subjects on drugs. However, the study has several methodological flaws. The control group and the treated groups were of significantly different sizes, and the control group's blood pressure was not monitored for the same length of time as the treated group. Since relaxation and hypnosis were used during each session, the individual effects could not be parceled out.

In summary, combined relaxation procedures involving meditation, relaxation, and biofeedback induced relaxation lead to blood pressure changes from 8–28 mm Hg/8–19 mm Hg. These changes are significant even when initial blood pressures are accounted for (30). Furthermore, at

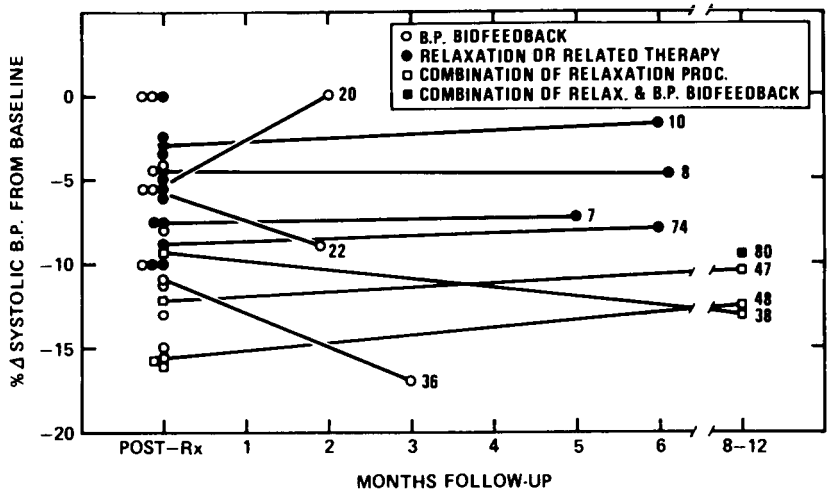


Figure 2. Percent change in systolic blood pressure from baseline to posttreatment and follow-up for studies involving blood pressure biofeedback, relaxation related therapy, a combination of relaxation procedures, or a combination of relaxation and blood pressure biofeedback. Numbers on the figure refer to the reference in the bibliography. The numbers for the studies reporting posttreatment only available from the author.

least two adequately designed studies suggest that these initial changes are maintained at six months or longer.

Comparison of Techniques

Figures 2 and 3 compare results from the studies listed in Tables 1A, 1B, 2, and 3. In these figures, the studies listed in the tables are graphed by percent of change from baseline. Definitive analysis of specific treatment effects can only be accomplished by using the types of regression analysis presented by Jacob et al. (30). As can be seen in the two figures, the greatest mean changes for systolic and diastolic blood pressures occur with biofeedback procedures or combined relaxation/meditation procedures. The contradictory results of studies are also evidenced in the figures—the various procedures show a wide spectrum of changes from none to more than 10% from baseline systolic or diastolic. Another striking feature of the figures is that no blood pressure biofeedback studies have more than three months of follow-up. The time course of the placebo suggests the importance of extending follow-up beyond three months. For the studies which have been followed up for more than six months, the combined procedures have the greatest sustained effect.

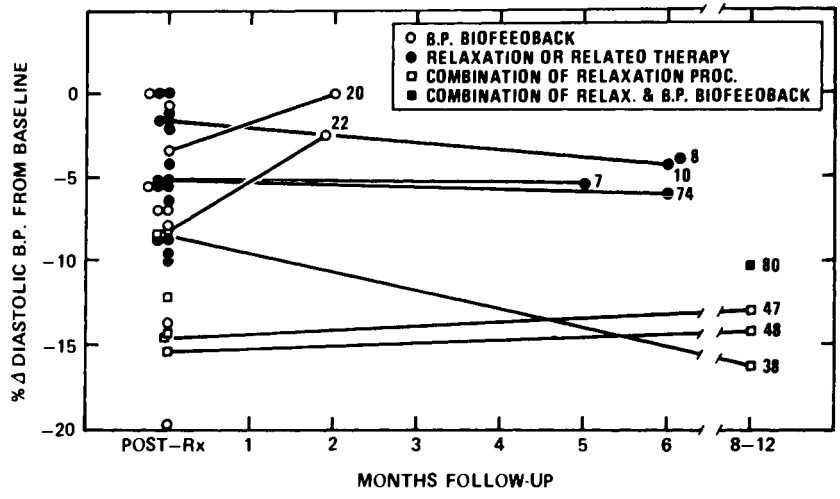


Figure 3. Percent change in diastolic blood pressure from baseline to posttreatment and follow-up for studies involving blood pressure biofeedback, relaxation related therapy, a combination of relaxation procedures, or a combination of relaxation and blood pressure biofeedback. Numbers on the figure refer to the reference in the bibliography. The numbers for the studies reporting posttreatment only available from the author.

Generalization of Effects to Other Settings

A concern of investigators in this field is that subjects may reduce their blood pressures only at the time blood pressure is being taken in the clinic or office but not at other times. Three studies have addressed this issue. Blackwell et al. (9) found good correlations between changes in blood pressure taken at home and blood pressure taken in the clinic. Krist and Engel (36) also demonstrate drops in both home and clinic settings. Kleinman and Goldman (34) used the Halstead Category subtest as an independent measure of generalization effects. They argued that if subjects showed improvement on the Halstead Category which correlated with improvements in blood pressure, this would prove that the blood pressure was being reduced in other settings. They found a significant positive correlation between systolic pressure and number of errors on the Category Test and between magnitude of decrease in systolic pressure and improvement in Category Test subsequent to training. Their argument is only valid if the Category Test improvement would only have occurred if blood pressure had remained down and not simply because, at the time of the test, the blood pressure was reduced. Since they failed to demonstrate this, their argument is not convincing. Only continuous or frequent blood pressure systems in controlled and natural environments will be able to determine if relaxation effects generalize to other settings.

Side Effects

Relaxation and related techniques, biofeedback, and combined treatment procedures are remarkably safe, but several adverse reactions have been reported. There have been several reported cases of psychosis developing in individuals practicing extended meditation procedures. Since meditation in some settings may vastly decrease proprioceptive feedback and occur in environments with minimal sensory input, these psychoses may be akin to those developing from sensory deprivation of any type. There have also been reports of depersonalization and other unusual states or feelings occurring with these practices. Wolpe (84), who has probably had the most experience with relaxation, reports that some patients become depressed or experience bothersome thoughts. Other patients complain of pain in various sites following practice. Finally, some patients prefer to use behavioral interventions for their hypertension control and will stop medications. In one reported case, this resulted in a potentially life-threatening hypertensive crisis (74).

REVIEW OF BEHAVIORAL APPROACHES TO HYPERTENSION REVIEWS

Behavioral medicine procedures have probably been most extensively studied in the area of hypertension. Several reviews, and the data produced here, suggest that relaxation and related techniques have real treatment effects on patients with essential hypertension and that these treatment effects may last six months or longer. These studies have also been extensively reviewed in the medical literature, and the comments made by largely nonbehavioral researchers in medical journals may reflect the issues of how behavioral procedures will be accepted by medical authorities. For this reason, we will review the comments made by medical reviewers of primarily behavioral articles.

After Stone and DeLeo's (70) article appeared in the *New England Journal of Medicine,* Abboud (1) wrote an editor on relaxation and autonomic control and hypertension. He listed many methodological problems with the studies: Nonrandomized and unequal control groups, failure to note if patients complied with procedures, etc. However, after reviewing the physiology of hypothalamic effects on blood pressure and their relationship to the development of essential hypertension, he ended with the optimistic note that, "if autonomic functions can be modified and trained in man, and if such training is effective in the early phase of hypertension, and, most importantly, if it can be maintained over a space of time, the positive feedback mechanisms at the level of both the central nervous system and the vasculature may be interrupted and sustained hypertension may be prevented." His criteria for favoring relaxation and related techniques are their simplicity, the fact that they do not require behavior modification, and that they can be practiced economically and easily. These criteria are what practitioners apparently find particularly desirable about prescribing drugs.

After a brief description of the results and several behavioral studies, a reviewer in the *American Heart Journal* (10) states that, "Taken together, these results indicate that... for TM... a minority (of subjects) show sustained benefit of blood pressure, some show transitory benefit, and others show none. The presence of psychological benefit, the absence of drug side effects and the experience of self-control may make TM an appealing therapeutic adjunct for some hypertensive individuals. Others may find the time commitment, mystical aspects, payment of a fee, and assumption of personal responsibility less appealing." After noting that the specificity of effects attributable to TM per se remain doubtful, he notes that "none of the studies on blood pressure have applied a meaningful control for the time,

interest or mystique of meditation," and notes the well-established effect of the placebo on high blood pressure. He recommends that patient's questions about meditation be taken seriously but makes no commitment as to whether or not doctors should recommend it.

In another review, "Meditation and Methyldopa," the editors of the *British Medical Journal* also address the state of the behavioral change art (41). After reviewing Patel, Blackwell, and Stone's studies, they arrive at the cautious statement, "these studies raise the possibility that control of blood pressure in some well motivated patients with mild hypertension may be possible without drugs. The scope of these techniques remains to be defined, the feasibility of their widespread application is uncertain, and there is no immediate suggestion that physicians' efforts to lower blood pressure with drugs will be replaced wholesale by relaxation techniques. But, in a few years, who knows?"

In a more extensive review of the area Steptoe (69), who is a psychiatrist and has studied the effects of biofeedback on blood pressure, concludes, "The effects cannot be attributed solely to the biofeedback or instructional methods employed... the contribution of nonspecific placebo factors has yet to be evaluated." He ends with a cautious statement, "It is probably that psychological treatments will be appropriate for only a proportion of hypertensives... methods of the type reviewed here may make a valuable contribution to the long-term management of high blood pressure." In the most extensive review of behavioral approaches to high blood pressure, Shapiro and others (63) who have been active in unspecific and biofeedback techniques as related to high blood pressure, indicate the steps behavioral techniques will probably have to undergo to become "accepted treatments." Shapiro notes that for pharmacological agents, evaluation and acceptance of new drugs into standard practice occur in three phases. In Phase 1 trials, individual patients receive the drug to determine its basic physiological effects, its dose-response relations, and its side effects; in Phase 2 trials, controlled comparisons are made of the drug to placebo or other agents in small groups of patients, usually with double-blind techniques, with further establishment of dose and toxicity; and then in Phase 3, broad clinical trials in large samples of patients are performed. He notes that most behavioral studies have been Phase 1 studies, and the lack of Phases 2 and 3 trials makes the specific contribution of behavioral methods uncertain. After reviewing many of the same studies mentioned in this chapter, he concludes, "All methods seem to produce modest, albeit usually statistically significant, falls in blood pressure." And he states, "In conclusion, we believe that widespread and uncritical application of behavioral methods to the treatment of high blood pressure is premature." This advice is well warranted and it behooves the behavioral researchers involved in this to begin to pur-

sue the types of issue raised by Shapiro and these other reviewers.

Future research may benefit from these types of studies related to Phases 1, 2, and 3 activities.

Phase 1

Basic Physiological Effects

The mechanisms of relaxation and related techniques, biofeedback, and combined effects should be studied. Basic biochemical and physiological data need to be obtained. Benson's hypothesis should be tested.

Dose-Effect Relations

There have been no formal studies on dose-response relationships between the behavioral procedures and the effects on blood pressure. Should subjects practice once or twice a day, and for five or ten minutes? Do the same dose-effect relationships apply across populations?

Side Effects

The occurrence or nonoccurrence of side effects should be systematically reported. As side effects are identified they should also be studied.

Generalization Effect

Issues of generalization of treatment effect from laboratory and clinic to natural settings should be undertaken.

Phase 2

Comparison Between Active Agents and Placebo

The modest effects on reducing high blood pressure demonstrated by some of the behavioral effects warrant comparison with active agents and placebos. Such trials require large number of subjects, but the advantages of nonpharmacological approaches would seem to justify the effort and costs.

Studies on the Interaction Between Types of High Blood Pressure and Treatment

Behavioral approaches may be more useful with some types of blood pressure than others. Labile and borderline hypertensive patients may do better than patients with long standing, reno-vascular or arteriosclerotic hypertension.

Phase 3

In general, Phase 3 studies should follow completion of Phase 1 and 2 studies. The current enthusiasm by the lay press for behavioral approaches may lead to the wide dissemination and use of these procedures prematurely.

CONCLUSION

Blood pressure control, in both normals and hypertensives, has been an important area of study for behavioral research. There have been many studies comparing behavioral treatments with control groups and each other; a few studies have been well designed and conducted with long-term follow-up. Taken together, these studies suggest that relaxation and related procedures, particularly in combination, have an active treatment effect. However, the mechanisms, dose-effect relationships, interactions with medication, comparison with medication, generalization, and practice effects have yet to be answered. It seems appropriate for researchers in this area to begin to address these questions.

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The Comprehensive Handbook of Behavioral Medicine, Volume 1

CHAPTER 5

Cardiac Rehabilitation: Current Status And Future Prospects

ROBERT F. DEBUSK

The term *rehabilitation* as used here encompasses restoration of a patient's medical, psychological, and social status toward normal following heart attack. Two major recent trends reflect a shift toward development of a data-based system of management for postinfarction patients: A greater emphasis on early prognostic stratification following infarction, and provision of earlier access to physical activity following infarction.

EARLY PROGNOSTIC STRATIFICATION

The extent to which patients return to normal activities following infarction and the rate at which they return are markedly influenced by the perceived likelihood of recurrent coronary events, i.e., by the prognosis. Traditionally, prognostication following myocardial infarction has been based upon "clinical evaluation" consisting of the hospital course and the posthospital history, physical examination, chest X-ray, and electrocardiogram. These features reflect cardiovascular function at rest or during restricted activity, and none provides information concerning the presence or absence of abnormalities during the kinds of activity which the patient encounters in his usual environment.

In recent years, a variety of techniques have been employed that extend the clinical evaluation. Invasive and noninvasive monitoring of ventricular function during and following hospitalization for infarction has permitted stratification of patients into groups with markedly different mortality rates (15). Two noninvasive techniques, ambulatory electrocardiography and treadmill exercise testing, have proved particularly useful. With ambulatory

monitoring a patient wears a battery-operated tape recorder capable of recording the electrocardiogram for 24 hours. The electrocardiogram can be readily transcribed onto computers for data analysis and processing. It then becomes possible to determine a patient's mean and maximal heart rate and the frequency and type of any ventricular arrhythmias which occur during the period of monitoring. Moss (14) found that 45% of patients dying within the first five months following hospitalization for infarction came from only 15% of the population. This "high risk" group had frequent premature ventricular contractions, i.e., > 20/hour by ambulatory electrocardiography, and in addition had hypotension or congestive heart failure within the Coronary Care Unit, or a history of limiting angina prior to hospitalization.

Treadmill exercise testing has only recently been applied to the evaluation of prognosis and functional capacity soon after infarction (12). In treadmill testing a patient walks on a moving belt at a variable speed and grade while his electrocardiogram and blood pressure are continuously monitored. The treadmill measures the maximal heart rate, blood pressure, and workload, and the appearance of symptoms, abnormal heart rhythms, a fall in blood pressure, or the appearance of an electrical signal indicating a lack of blood supply to the heart during exercise, i.e., myocardial ischemia.

We have found treadmill exercise testing to be useful in characterizing prognosis and functional capacity. Our patients, who are uniformly free of congestive heart failure and unstable angina pectoris at three weeks following infarction, represent approximately two-thirds of all males under the age of 70 who survive hospitalization. Nearly one-half had experienced significant ventricular ectopic activity, heart block, or clinical heart failure within the hospital. The results of early postinfarction exercise testing in other populations containing patients with clinical heart failure may well be different. The details of our protocol are described elsewhere (12).

What prognostic information is provided by treadmill exercise testing three weeks following infarction? Three major classes of abnormality are evident: *Myocardial ischemia,* manifest as ischemic ST segment depression or angina pectoris. The prevalence of myocardial ischemia was 40% in our series. Of patients with ischemic ST segment depression, angina was present in one-third. Conversely, patients with angina had ischemic ST segment abnormalities in two-thirds of cases. Survival free of coronary events viz. myocardial infarction, sudden cardiac death, or coronary artery bypass graft surgery was decreased in the presence of marked ischemic ST segment depression of 2 mm or more. *Mechanical damage,* abnormal ventricular function, reflected in a markedly diminished maximum treadmill workload, was associated with a diminished projected five-year event-free survival. *Electri-*

cal damage, manifest as ventricular ectopic activity. Ventricular premature contractions were present in 54% of tests, were rarely an endpoint, and were not associated with complications of testing even when of "high grade," and were not significantly correlated with prognosis.

It is important to emphasize that treadmill testing provides information *independent* of the clinical estimation of severity, even within a population whose one-year prognosis is already expected to be low due to the exclusion of patients with significant mechanical damage.

EARLY ACCESS TO PHYSICAL ACTIVITY

The second major trend in cardiac rehabilitation is provision of early access to physical activity following infarction. Restriction of physical activity was originally based on histological studies of Mallory et al. (11), who demonstrated that up to six weeks were required for complete scar formation. The clinical inference from these studies was that physical activity must be restricted for a like period, an inference that has impeded access to physical activity for 40 years. Mallory's study of patients dying within the first six weeks following infarction concerned only those with the most severe mechanical damage. For this reason, Mallory's conclusions cannot be applied broadly to all patients with infarction. Fears that physical activity might precipitate heart failure, aneurysm formation, or myocardial rupture have not been borne out in clinical trials of early ambulation and early hospital discharge following infarction (11). More recently, measurements of ventricular function have revealed marked abnormalities, of the kind probably found in Mallory's patients, in only about one-third of cases (9). Ventricular function as reflected in functional capacity by treadmill testing is not significantly impaired in patients without clinical heart failure: Exercise testing soon after infarction has revealed an unexpectedly high functional capacity in these patients.

Four aspects of postinfarction physical activity are of particular importance: 1) Mobilization within the hospital, 2) transition from hospital to home care, 3) exercise training, and 4) occupational work evaluation.

Early Mobilization Within The Hospital

Levine popularized the "armchair" management of myocardial infarction in the 1950's (10), the first major step away from strict bedrest for postinfarction patients. Many empirical studies since that time have indicated that limited physical activity within the hospital is safe in patients free of

clinical heart failure. Notwithstanding this experience, most patients presently managed in American hospitals spend most of their time in bed rather than in chairs and by the time of hospital discharge they have usually not achieved a level of physical effort commensurate with their activity upon their return home (16). We found that uncomplicated patients could safely perform physical activity equivalent to a workload of 2 to 3 METs within the coronary care unit as soon as three days following hospitalization (1). Patients free of significant electrical, mechanical, and ischemic abnormalities by the fifth hospital day may safely engage in ambulation within the hospital. Such patients comprise nearly half of all those hospitalized with myocardial infarction.

In uncomplicated patients performing treadmill exercise testing as soon as seven days following infarction, we have not noted myocardial ischemia or ventricular ectopic activity below our arbitrary heart rate endpoint of 110 beats/minute. Between 7 and 14 days following infarction we have provided heart rate-monitored ambulation programs programs in which the maximum heart rate was 100 or less. This has allowed a work intensity of 3 METs or less, which could be readily sustained for 30 to 60 minutes daily. At 14 days the exercise test heart rate limit was increased to 120. Patients continuing ambulation between the 14th and 21st days following infarction were given a peak heart rate limit of 110. If myocardial ischemia appeared at treadmill heart rates below 120, the heart rate at the onset of myocardial ischemia was used as the basis for subsequent activity guidelines. Thus, the treadmill exercise test has provided an objective basis for guidelines regarding physical activity in the earliest stages of recovery following infarction.

Transition From Hospital To Home Care

Until recently, this transition has been surrounded by uncertainty, owing to the lack of a standardized technique for establishing a baseline of functional capacity at the time of hospital discharge. More recent experience with treadmill exercise testing indicates that functional capacity at two weeks following infarction is sufficient to permit most normal activities upon return home. Using an arbitrary heart rate maximum of 120, we noted an average functional capacity of 4 METs, or multiples of resting energy expenditure, with two-week postinfarction exercise testing. This energy cost is roughly equivalent to that of walking at a pace of three miles per hour. Because selected patients are being discharged as soon as seven days following infarction, it is important to assess their capacity to engage in normal activities upon their release from the hospital. Low-level treadmill exercise testing is useful for this purpose; it provides quantitation of

functional capacity and detects major cardiac abnormalities such as advanced ventricular ectopic activity and myocardial ischemia which are often unaccompanied by symptoms.

Exercise Training

The natural history of recovery of functional capacity following infarction has only recently been examined. It is clear that by three months following infarction, most patients have attained a functional capacity equivalent to that of sedentary age-matched normal individuals. It is *not clear* that these individuals require a formal program of exercise training *if* the sole objective is to restore functional capacity to normal levels.

Traditionally, cardiac rehabilitation has been equated with group programs of high-level exercise training commencing no sooner than three months after infarction and continuing more or less indefinitely. Although this model has provided undoubted physical and psychological benefits, simpler and less expensive strategies for enhancing functional capacity and psychological status need to be explored. The following questions need to be answered:

a) What is the minimum *intensity* of physical activity that will restore functional capacity to normal levels following infarction? In our experience with exercise training between 3 and 11 weeks following infarction, no substantial improvement in functional capacity was achieved by programs of gymnasium or home exercise training compared to a "control" condition in which patients received no specific guidelines for physical activity. Significantly, the functional capacity of *all* patients of 7 METs at 3 weeks improved to 9 METs at 7 weeks and to 10 METs at 11 weeks (8). This level of functional capacity is not significantly different from that of sedentary middle-aged men without clinical heart disease and indicates that within our population the extent of infarction is not a major determinant of functional capacity. It is important to emphasize that performance of an exercise test at three weeks is likely to make patients more physically active than they would otherwise be, in part because of the demonstration of their ability to perform exercise without ill effects. Thus, our "control" group cannot be considered a control group in the purest sense. On the other hand, the increasing application of early functional testing is likely to alter the management of most patients, so that they will more closely conform to our group rather than a group in which no early exercise testing is performed.

b) How soon may exercise training commence following infarction? We have provided exercise training beginning at three weeks following infarction to 70 patients free of clinical heart failure or unstable angina. Guide-

lines for the intensity of early exercise training were similar to those used in training patients at a later phase of convalescence, i.e., at 10 to 12 weeks following infarction. The initial activity prescription was based on the maximum heart rate achieved with symptom-limited exercise testing at three weeks following infarction. No complications attributable to the exercise training program have been noted.

c) What is the role of home training programs? Ten patients underwent stationary bicycle exercise training at home, using guidelines for exercise intensity that were identical to those used by patients receiving gymnasium exercise training. These patients were free of exercise-induced myocardial ischemia. The electrocardiogram during stationary bicycle exercise, transmitted to the laboratory, was useful in documenting that exercise was being performed within preset heart rate limits. No complications were noted in the approximately 24 hours, i.e., three hours weekly for eight weeks, per patient participating in the study. Much larger experience is necessary to document the safety of home exercise programs. We feel that the safety of these programs will be enhanced by: 1) Selecting patients free of clinical heart failure and free of myocardial ischemia occurring spontaneously or induced by exercise testing; 2) basing the intensity of home exercise training on a symptom-limited exercise test performed prior to training; and 3) periodic monitoring of the electrocardiogram during exercise to detect myocardial ischemia or ventricular ectopy not disclosed by prior exercise testing (7).

d) When has maximum benefit of exercise training been reached? This decision is based upon the observation that functional capacity does not continue to rise with continued exercise training. Follow-up exercise testing is usually carried out between three and six months following the onset of training. The functional capacity of our patients who continued exercise training between 11 and 26 weeks did not increase significantly over the 11-week value, and there would have been no further benefit in their continuing physical exercise training *if* enhancement in functional capacity were the only objective. It may be that a 10 MET exercise capacity represents a maximum attainable level for most patients following infarction (4). Since few patients perform activities requiring a higher level of physical exercise, this ceiling does not restrict the activity of most patients following infarction.

Evaluation of Occupational Work Capacity

The mechanization of industry has diminished the importance of functional capacity as an important determinant of occupational work potential.

Objective assessment of functional capacity is potentially useful in evaluating the cardiovascular tolerance for occupational work, but few patients receive such an evaluation (2). Work classification units have made a contribution to the management of postinfarction patients. Their usefulness could be augmented in the following ways: a) Occupational work evaluation must be performed sooner following myocardial infarction. Most patients undergo this evaluation no sooner than 8 to 12 weeks following infarction, despite evidence that functional capacity has reached a level compatible with occupational work considerably before this time. Most of our patients could have returned to work before the seventh week after infarction. b) Greater emphasis must be placed on eliciting a *maximum cardiovascular response* with physical effort. It is myocardial oxygen consumption, not total body oxygen consumption, which determines the ability of postinfarction patients to perform physical work tasks. In normal individuals and in coronary patients, the heart rate-systolic blood pressure product correlates closely with myocardial oxygen consumption and with coronary blood flow (5). In fact, heart rate alone correlates nearly as well with myocardial oxygen consumption as does the product of heart rate and systolic blood pressure. The heart rate at the onset of ischemic ST segment depression or angina pectoris, the "ischemic threshold," has been shown to be reproducible upon repeat treadmill exercise testing.

Even though the heart rate during dynamic leg exercise such as treadmill walking or bicycle riding correlates closely with myocardial oxygen consumption, there has been doubt in the past that this same relationship would obtain under other circumstances in which patients were performing static or lifting work, dynamic work involving the arms simulated by cranking a bicycle with the arms, or when static effort was combined with dynamic effort involving the arms or legs. Under all these circumstances, we have found that the heart rate reliably indicates the onset of myocardial ischemia. For example, we found that neither the *peak* heart rate or blood pressure or the *ischemic threshold* of heart rate and blood pressure were significantly altered when static effort was added to treadmill exercise. Likewise, we have found the "ischemic threshold" of heart rate to be the same for dynamic exercise involving the arms and the legs (3).

Indeed, it may be that *any* factor capable of inducing myocardial ischemia will do so at a heart rate that is relatively constant for a given individual. For example, the ingestion of food is known to decrease the amount of physical effort required to produce angina pectoris, without altering the maximum heart rate or ischemic threshold. In other words, the addition of a postprandial stress to a physical work stress simply increases the *rate* at which the ischemic threshold is reached during treadmill exercise, without significantly altering the ischemic threshold or the peak heart rate. It ap-

pears likely that cigarette smoking, psychological stress, and exposure to cold, each capable of inducing myocardial ischemia, will have a similar effect. The few direct comparisons of psychological and physical stress reported in the literature indicate that the heart-rate response to psychological stress is far less than to maximum physical stress, and that the incidence of myocardial ischemia is proportionately lower (13). As a corollary, myocardial ischemia will appear at lower workloads and heart rates in patients with more severe disease than in patients with less advanced disease. Patients with severe coronary artery obstruction may demonstrate myocardial ischemia with *several* laboratory methods, including static effort and psychological stress, whereas patients with less advanced disease may manifest ischemia *only* with maximum treadmill exercise.

These observations have major implications for the management of postinfarction patients. If the heart rate threshold for ischemia is relatively constant during any combination of physical, environmental or psychological stress, it will a) obviate the need for blood pressure measurement to detect the onset of myocardial ischemia, thus considerably simplifying the formulation of guidelines for activity in patients with coronary disease, b) permit the ischemic "threshold" for a given individual to be readily determined during dynamic effort in the laboratory, thereby obviating the need to simulate the particular environment to which the postinfarction patient returns. For example, although static and combined static-dynamic work tasks are commonplace in business and industry, it may be unnecessary to simulate the actual occupational tasks involving these forms of effort, if the ischemic threshold can be determined by treadmill exercise testing. In our patients, static effort never elicited ischemic ST segment depression or angina pectoris, and was far less effective than symptom-limited treadmill exercise in eliciting ventricular ectopic activity. Once the heart rate threshold for ischemia is established by treadmill exercise testing, there is good assurance that ischemia will not be noted at significantly lower heart rates during almost any other circumstance.

Although the formulation presented above has a sound theoretical basis, *on-the-job confirmation* of cardiovascular responses to actual working conditions will provide assurance that significant cardiovascular abnormalities are not evident during actual circumstances. Such monitoring is in its infancy, and it has had the substantial drawback that a computerized method for assessing ischemic ST segment abnormalities by continuously recorded electrocardiographic tapes has not been available.

THE FUTURE OF CARDIAC REHABILITATION

Functional testing performed soon after the acute event has major implications for the management of postinfarction patients: 1) Such testing will

facilitate prognostic stratification following infarction and may favorably influence prognosis. Since the mortality rate following infarction is greater in the first few months than at any later time, it is important to perform the evaluation as soon as possible following infarction. Patients at high risk for recurrence can then receive potentially effective medical and surgical therapy. Patients at low risk for recurrence can be spared needless restriction of activity and the risk of toxic drugs and the performance of coronary angiography. In either case, uncertainty regarding the patient's true status will be resolved. 2) Access to physical activity is a powerful antidote to the depression that often follows myocardial infarction (6). The early demonstration to the patient that he can safely perform physical activity may dispel much needless depression and anxiety, not only for the patient but for his family. Further studies are needed to document the psychological impact of early exercise testing and ambulation programs. 3) Early determination of occupational work potential may enhance the work capacity of most post-infarction patients by permitting early return to work and by decreasing the rate of job reassignment, early retirement, and other costly dislocations of the work force.

Assuming that early testing and monitoring can achieve potential benefits, how would they be financed? The technology is simple, readily available, and relatively inexpensive. Occupational disability is so costly that any substantial reduction in its costs would release the necessary economic resources to underwrite the costs of functional evaluation following myocardial infarction (2).

SUMMARY

The humanistic use of advanced technology appears to have considerable potential for optimizing the management of patients following myocardial infarction. It is clear that psychosocial factors, not medical factors, pose the greatest obstacle to rehabilitation of most patients following myocardial infarction. Much needless psychological disability can be obviated when advanced technology is used to clarify the issues of prognosis and functional capacity following infarction.

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The Comprehensive Handbook of Behavioral Medicine, Volume I

Introduction to the Musculoskeletal System

This section includes three chapters of seeming great disparity; normal exercise physiology, behavioral approaches to exercise and athletic performance, and rehabilitation from strokes. The relationships among pathology, normalcy, and supernormalcy are less clear with striated muscles than with other organs. These muscles and their relationship to the body as a whole are on a continuum from dennervated or de-afferented tissues at one extreme and the hypertrophied muscles of a weight lifter on the other, the immobile quadriplegic at one end of the spectrum and the marathon runner on the other. Between these two poles are most of the rest of us with considerable muscle disuse, low levels of activity, and moderately developed coordination. In providing this range of discussions, we hope to both stimulate interest in neglected areas in desperate need of research, like the use of behavioral techniques to increase activity and exercise, and to report some important research findings on proven techniques, like biofeedback for muscle retraining.

In the first chapter, Sucec provides a fascinating discussion of major trends in exercise physiology and their implications for "perfection of the human organism." He notes that "extraordinary physical feats achieved under heightened circumstances or in unusually compelling situations are legendary." He then provides a description of the overriding physiology and biochemistry for this physical activity but adds that these physical explanations are only part of the picture. He quotes Ryder as saying, "A champion stops not on achieving a given rate of speed but on winning a given medal," an apparent example of delayed but real reinforcement. He concludes with speculations on the implications for perfection of the human organism, a process that should be generalizable to those with less perfect physical endowments or pathological impairment.

If the limits of physical conditioning are not physiological, if we have not achieved or even nearly achieved our physiological capacity, then psychological factors would seem to be the major limiting factor. Epstein and

Wing discuss the difficulty of developing successful exercise programs. Their chapter begins with a discussion of the relationship of activity to obesity, which they suggest is a neglected area of research both from the standpoint of basic physiology—e.g., how activity is related to caloric consumption and storage in humans—and of the use of activity enhancement in weight control programs, which to date have tended to focus on eating behaviors. A discussion of cardiovascular risk factors intimately links obesity and exercise: Weight loss itself may lead to a reduction in blood pressure in hypertensive individuals, and a decreased weight may facilitate activity; increased activity may lead to weight loss and independently affect blood pressure and perhaps lower serum cholesterol. Epstein and Wing discuss some of the problems involved in measuring activity and techniques that have been used to overcome these difficulties. Measures of activity are, in fact, difficult: Direct observation usually occurs only in special settings; self-report has severe limitations; and ambulatory devices all have limits. Furthermore, measures of physical conditioning, like the treadmill or the 12-minute test, are independent measures that may or may not relate directly to activity levels. For example, a person can significantly increase his treadmill performance by increasing his heart rate above 120 for 10 minutes three times a week—a trivial change in a week's activity. Following their discussion of exercise and obesity and the problems of measuring activity, they discuss studies designed to improve adherence to exercise prescription. The paucity of studies is striking given the importance of the subject, and Epstein and his group are to be credited for developing several key model studies pertinent to research in this area. This chapter ends with a discussion of behavioral sports psychology and thus comes full circle to Susec's chapter on exercise physiology.

The section on the musculoskleletal system concludes with Basmajian's excellent discussion of the state of the art of rehabilitation for stroke patients. He notes that little doubt exists as to the usefulness of behavioral and biofeedback technologies when applied wisely by trained therapists to appropriate cases. Although it is a literature largely of single case studies, at least one controlled study which focused on retraining the tibialis anterior muscle demonstrates an increase in both strength of dorsiflexion and range of motion twice as great in the patient receiving biofeedback training as in the control or traditional therapy group. He also presents data on early promising results for treating subluxation of the shoulder and restoring hand function. The specific use of biofeedback is different for each condition: With muscle retraining the focus is on feedback, with impaired hand function the goal is to relax spastic muscles.

Ironically, our ability to restore function in the damaged body seems greater than our knowledge of encouraging "normal" people to exercise.

The Comprehensive Handbook of Behavioral Medicine, Volume I

CHAPTER 6

Major Trends In Exercise Physiology: Recent Findings And Their Implications For Perfection Of The Human Organism

ANTHONY A. SUCEC

INTRODUCTION

The great German poet and biologist, Goethe, wrote "What you can do or dream you can, begin it, for boldness has genius, magic and power in it." In actual performance, human capacities probably far exceed what an individual imagines or dreams his physical and/or mental powers to be. And, while absolute limits to human potential are obvious (for example, those imposed by morphology of the species), cumulative evidence in the relatively new field of exercise physiology suggests that the limits of performance are more psychological in nature than physiological.

Extraordinary physical feats achieved under heightened circumstances or in unusually compelling situations are legendary. We can all at least vaguely recall news accounts of natural disasters or life-threatening accidents in which victims experienced unaccountable surges of strength or stamina and overcame incredible odds against their survival.

This phenomenon of unanticipated performance capacity in highly intense situations is most commonly exemplified during sporting events, particularly Olympic games or world-record qualifying events. Many track fans, for example, regard Billy Mills' 1964 record 10,000m win at the Tokyo

games to be among the greatest in Olympic history. His victory was remarkable in that Mills ran his fastest 5000m in the first half of the race and then went on to run the second 5000m as fast as he had run the first! Before the games, Mills had not been expected even to finish in the top ten. After the race, he described his win as the result of his genuine belief that he *could* win; he asserted that this psychological dimension had as much importance as training in this dramatic record-setting run (Personal communication to the author, December 1964).

World records for various athletic events may provide, as A. V. Hill suggested with remarkable foresight in 1926 (35), the best available indices for assessing the limits of human performance. Figure 1 depicts the progression

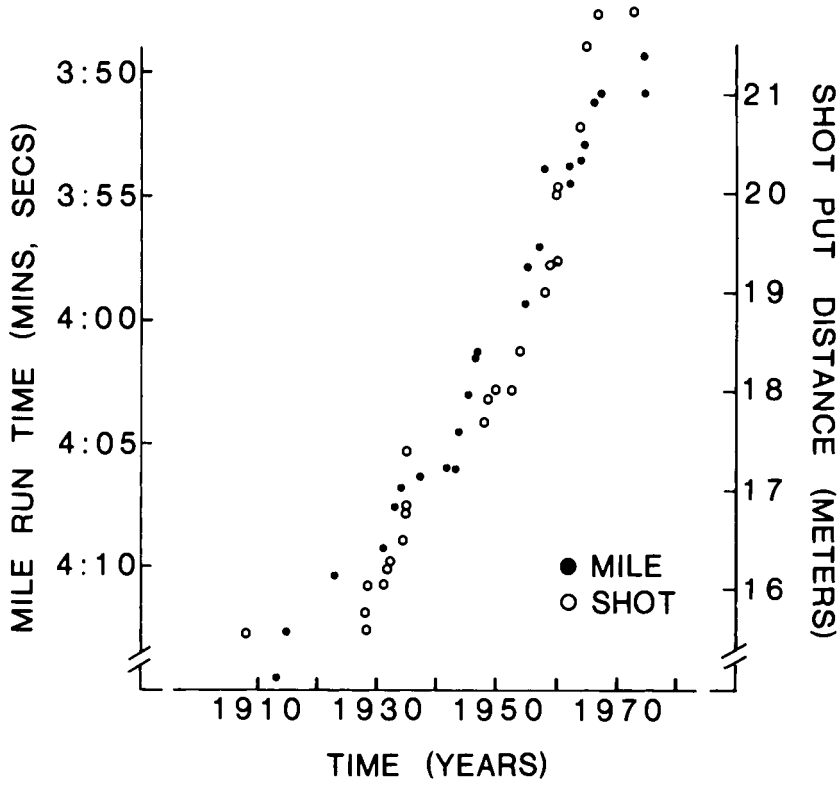

Figure 1. The progession of the world's records for the one-mile run and shot put (16 lb.) for men. Advances in the records have been attributed to increased and improved training, as well as to improvements in nutrition, medicine, and technology. Apparently, their summed effect has resulted in an almost identical linear rate of improvement; moreover, there is little to suggest a change in the rate of the past 65 years. Based on data from Quercetani (57).

of world records for the one-mile run and the shotput. The steady, almost exactly parallel improvements in the two events disclosed in the figure affirm Hill's notion about the abundant physiological data that records may yield. From the turn of the century (43), the literature contains repeated attempts to predict running performance. Elaborating on Hill's early suggestion, F. M. Henry (33) subjected physiological data to mathematical analysis and predicted the four-minute mile long before most other experts had thought it possible. Henry's achievement gave important impetus to the systematic evaluation of physiological adaptations resulting from athletic training. Although the preponderance of this research was undertaken to enhance accuracy in the prediction of performance in sporting events, it also provoked related inquiry and research into the nature of critical variables found in physical stress, such as circulation, metabolism, muscle biochemistry, and other fundamental mechanisms.

An Overview of Performance Factors

Generally speaking, athletic performance is the culmination of diverse traits and skills, training, and "given" factors. A survey of the literature reveals agreement that major determinants in athletic achievement (20) include genetic endowment, age, sex, and training (duration, frequency, intensity, etc.). More specifically, Figure 2 shows the complexity of factors contributing to one's ultimate performance. This model, as proposed by Astrand and Rodahl (2), imparts a sense of the intricate relationship between the various factors which have been demonstrated to exert either a direct or a modifying influence on physical performance. The model also focuses on service functions, such as fuel and oxygen consumption, and identifies several categories of modifying traits or conditions, such as: 1) Somatic factors (sex, age, physique, health status); 2) training adaptions (biochemical and histologic alterations); 3) psychological factors (mental attitude, motivation); 4) environmental influences (altitude, temperature, pollution); and 5) nature of the work (intensity, duration, rhythm). Thus the production of energy is dynamically dependent upon concerted influences of these factors; how this complex interchange facilitates or inhibits the service functions determines performance.

Overriding Considerations

Aerobic And Anaerobic Energy Sources

Ultimately all athletic performance, regardless of its nature, relies on an adequate energy source, e.g., to sustain speed in distance running or main-

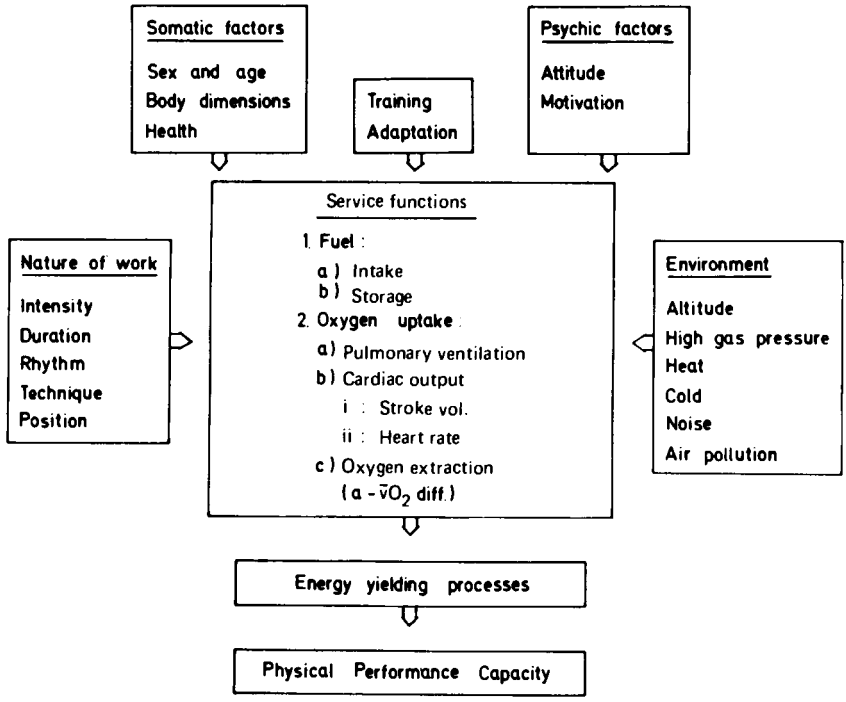

Figure 2. Factors influencing the capacity for muscular activity. Reproduced from Astrand and Rodahl (2) with permission.

tain tension levels in weight lifting. Energy is supplied to working tissues via two modes, either aerobic (via oxygen) and/or anaerobically (without oxygen).

The predominant energy source for prolonged activity is the aerobic pathway (oxidative phosphorylation), while the primary energy source for weight lifting is anaerobic metabolism (glycolytic pathways). In human skeletal muscle, two fiber types have been identified and categorized (37) according to contraction rate: Slow twitch fibers (ST) and fast twitch fibers (FT). The ST fiber has a higher oxidative capacity than FT fibers and a lower level of glycolytic activity. Therefore, the ST fiber predominates in aerobic exercise; the FT fiber functions primarily in anaerobic exercise, though both types can increase their oxidative capacities as a result of endurance training.

Age And Sex

These characteristics undeniably exert profound influences upon sports performance. Figure 3 indicates the primacy of age over sex in United

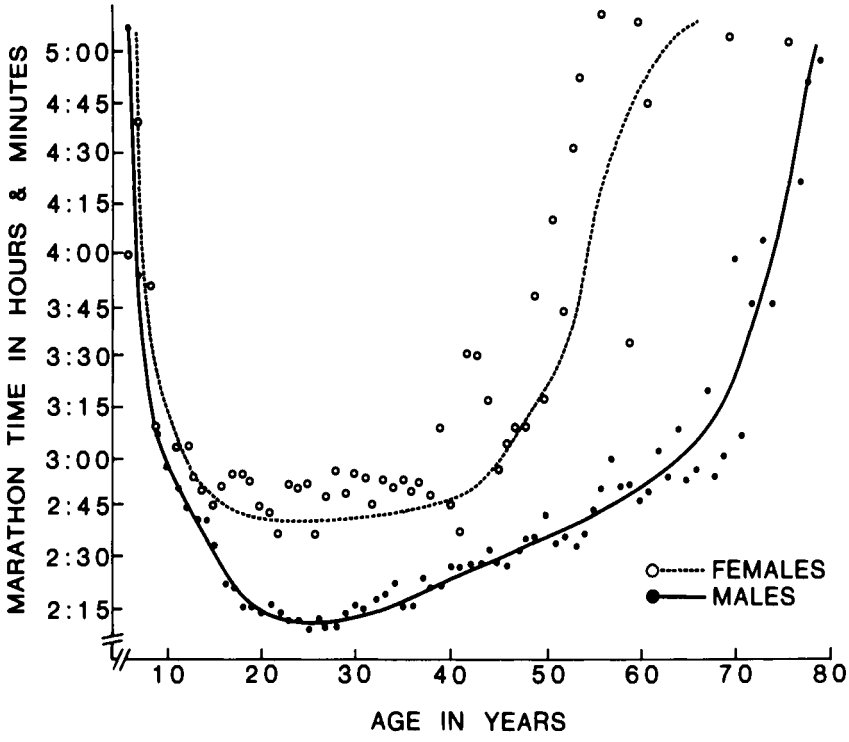

Figure 3. Age and sex records for United States citizens, as of January 1978. Based on data from *Runners World* 13:99, 1978.

States marathon scores, classified according to age records. Scores range from five hours for both males and females at 6 years to lows of 2.15 and 2.62 hours respectively for males and females in the mid-twenties; gradually scores climb back to five hours for females of 50 years and males of 70 years.

As in many athletic events, sex differences in marathon scores do not emerge until the onset of puberty. At this age, female scores no longer improve at the same rate as do male scores (cf. hemoglobin differences, p. 106); improvement continues at a decreasing rate until age 20. After this time, performances remain somewhat constant until age 40, after which they decrease rapidly. Males continue to improve until age 18, after which the rate of improvement decreases until mid-twenties. From this age until the mid-sixties, there is a gradual linear decline in performance.

During the peak performance years of marathon runners, the male/female differential averages 20%. That is, women in this age group can maintain a maximum running speed which is equivalent to about 80%

of the maximum speed men can maintain. This differential is fairly consistent in other capacities, such as oxygen uptake (aerobic capacity), body composition, and so forth; manifestations of this differential will be discussed further as they occur in the appropriate sections to follow.

Wide variations in muscular strength resulting from sex and age differences are obvious; quantification of these differences is discussed extensively in the literature and can be found in standard exercise physiology textbooks. For example, Ikai (40) reported that women are capable of approximately 87% of the maximal muscular strength of men in a leg extension task. However, females can only manage 53% of the averaged strength that men can lift in an arm flexion test.

Longitudinal curves for muscular strength parallel those observed in aerobic capacity and marathon performance. The strength decrement can exceed 20% when differences are compared between the ages of 20 and 60 years (2).

PRINCIPAL FACTORS OPERATIVE IN ENDURANCE AND STRENGTH PERFORMANCE

Space restrictions prohibit a satisfactory examination of any single factor and its interplay with other factors in several sporting events. Indeed, a comprehensive discussion of a given variable (e.g., sex or age in relation to oxygen uptake) would undoubtedly consume most of the pages allotted to the musculoskeletal system. Therefore, I have elected to confine the discussion to a few major factors mentioned above as they are operative in two types of performance: 1) *Endurance*–which relies on aerobic energy sources and requires low-to-moderate muscle tension; and 2) *strength*–which depends upon anaerobic energy reserves and generates high levels of muscular tension. Prototypical sporting events which illustrate these are *distance running* and *weight lifting.*

Both sports are highly responsive to conditioning; the influence of skill, thus of motor function, is limited in both. World class proficiency in distance running demands a hierarchy of factors and training which contrasts sharply, in fact almost inversely, to the hierarchy necessary for world-class weight-lifting performance; naturally, the effects of training vary widely, as do their broader implications for physical activity in health maintenance.

Respiratory, Circulatory, and Metabolic Factors in Endurance Training and Performance

Among several important factors in distance running, *aerobic capacity,* or maximal oxygen uptake ($\dot{V}O_2max$), has clear preeminence. This is the max-

imal ability of the respiratory and circulatory systems to transport oxygen to the site of contracting muscle cells and the corresponding cellular capacity to extract the oxygen for use by the mitochondria (37). Costill (14) and other investigators (64) have reported a strong correlation coefficient ($r \cong .90$) between $\dot{V}O_2max$ and running performances for three miles (64) or longer (14, 17).

The hallmark of the endurance athlete is, indeed, a large $\dot{V}O_2max$ (16, 25). Costill et al. (15) found a mean $\dot{V}O_2$ max of 77.4 ml/kg•min for 14 male world-class runners, while 18 male distance runners of college varsity caliber averaged 70.1 ml/kg•min. Varsity high school, college, and club runners respectively averaged $\dot{V}O_2$ max of 63.1, 68.7, and 65.5 ml/kg•min and had corresponding best times in the two-mile run of 9:42, 9:17, and 9:26 (64). Distance runners, as well as other endurance athletes, are known to possess $\dot{V}O_2$ max beyond 80 ml/kg•min (Figure 4). Steven Prefontaine, the American record holder for the three-mile and 5000m runs, was measured at 84.4 ml/kg•min (16). Female endurance athletes have had $\dot{V}O_2$ max scores in ex-

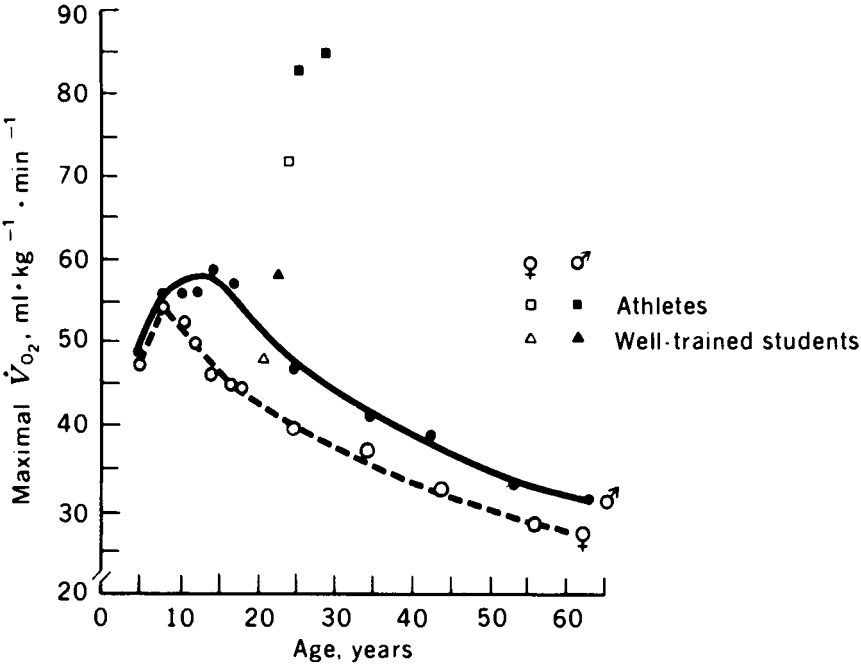

Figure 4. Aerobic capacity ($\dot{V}O_2max$) for normal males and females from the ages of 5 to 65 years. The data points on the upper portion of this figure represent values for elite endurance athletes. Reproduced from Åstrand and Rodahl (2) with permission.

cess of 70 ml/kg•min, while physically fit males and females in their twenties averaged 50 and 45 ml/kg•min respectively (2).

Limiting Factors in Endurance Performance

Astrand has identified six factors that may limit aerobic capacity: 1) Ventilation; 2) oxygen diffusion rate from the alveolar air to the blood; 3) cardiac output ($\dot{Q}$); 4) muscular blood flow; 5) oxygen diffusion from muscle capillaries to tissue; and 6) blood volume and hemoglobin content. However, other secondary factors may modify $\dot{V}O_2$ max, e.g., mass of muscles involved and mode of exercise (2). The assessment of $\dot{V}O_2$ max, as summarized by Åstrand and Rodahl (2), "is a measure of maximal energy output and the functional capacity of the circulation, since there is a high correlation between the maximal $\dot{Q}$ and the maximal aerobic power."

There is substantial avidence in the literature supporting the hypothesis that some physiological variables do in fact limit the rate of oxidative metabolism. The first, as suggested by Åstrand, may be lung capacity to transport oxygen to alveolar sacs. However, physiologists generally contend that $\dot{V}O_2$ max is not correspondingly restricted by this limitation; Robinson (58), and other investigators as well (50), have found that alveolar oxygen tension during maximal work is as high as or higher than it is at rest, while carbon dioxide tension is lower at work than at rest. However, a high *ventilation rate* may represent a substantial energy requirement (i.e., as much as 9% of $\dot{V}O_2$max has been attributed to respiratory muscles) (53). Furthermore, ventilation increases during exercise were equivalent to 26 and 18 times the rates during rest in trained males and females respectively (Figure 5). Compared to the increase in fit males (17 times) and fit females (14 times), the ventilation increases in trained males and females (over 40% more) elicit considerable curiosity about the causal factors determining their disproportionate increases.

In the normally healthy lung, the oxygen diffusion rate is not generally considered to represent an impasse to transport of oxygen, since most studies indicate only slight drops in main arterial oxygen saturation during maximal work (7, 51). However, since $\dot{Q}$ represents the total amount of blood circulated and thus the quantity of oxygen, there is little doubt that $\dot{Q}$ imposes an "absolute" or upper limit to endurance performance. The product of stroke volume (SV) and heart rate (HR) determines $\dot{Q}$. During maximal exercise, SV increases to about two times the SV at rest; and HR increases to about three times HR at rest. Their product, as calculated in

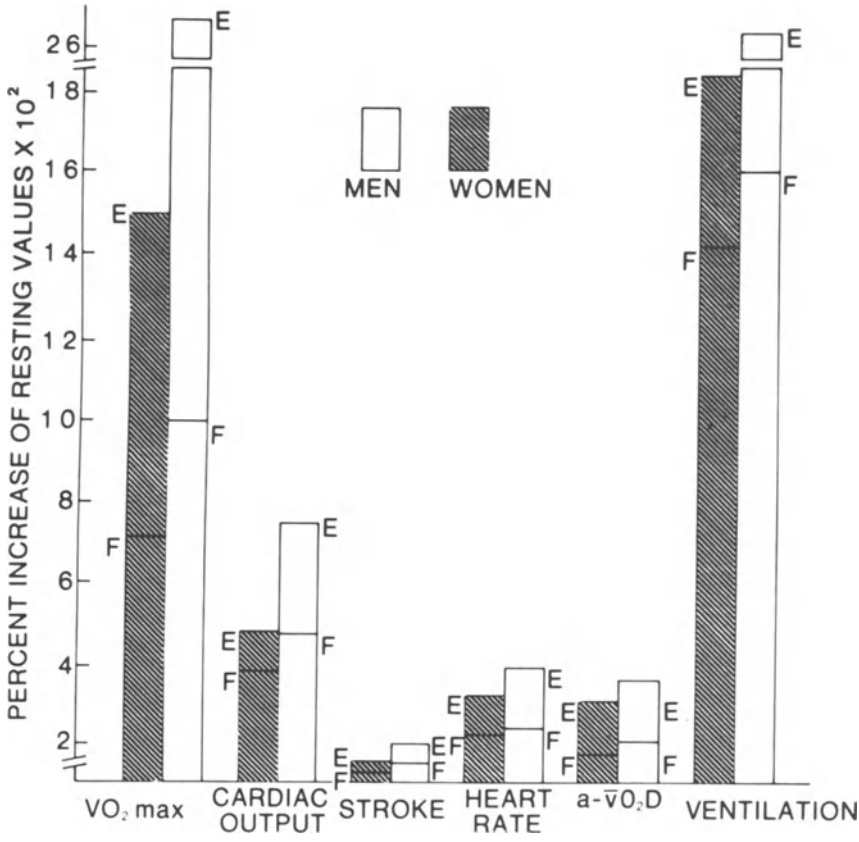

Figure 5. Percent changes in scores for selected circulatory and respiratory parameters of endurance performance at maximal oxygen uptake. Scores are expressed as a percent of the resting scores. The subjects are either fit or elite class male and female distance runners. Based on data from Åstrand (1), Åstrand and Rodahl (2), and Ekblom and Hermanson (25).

Figure 5, is a sevenfold increase for elite male athletes and a fivefold increase for elite female athletes. The differences in these values are quite striking (34%) when comparing the five- and fourfold increases in values for fit subjects. By ascertaining the product of $\dot{Q}$ by arterial-venous oxygen difference ($\dot{V}O_2 = \dot{Q} \cdot a\text{-}\bar{v}O_2D$), one may derive the $\dot{V}O_2$ score (as in Figure 5). $\Delta\dot{Q}$ for the elite male is 7.5 and $\Delta a\text{-}\bar{v}O_2D$ is 3.5, leading to a product of 26.3, which equals the $\Delta\dot{V}O_2$. The $a\text{-}\bar{v}O_2D$ variable signifies the tissue's capacity to extract oxygen from arterial blood, and the increase in this variable as an

adaption to training accounts for 50% of the increase in $\dot{V}O_2max$; the other 50% may be attributed to an increase in $\dot{Q}$, which increases primarily as a result of greater SV in response to exercise (2).

Hemoglobin (Hb) may represent a limiting factor in children, since they only average 78% per unit of weight as compared to adult males (2). An apparent relation exists between body size and Hb content; females have lower Hb levels than males (2). Since Hb transports oxygen, it seems probable that abnormally low levels could reduce aerobic energy production. Moreover, Ekblom et al. (24) have reported an increase in arterial oxygen content as well as $\dot{V}O_2max$ in three trained runners 28 days after the loss of 800 ml of blood followed by a reinfusion of their own packed red cells.

The sum effects of the above considerations upon $\dot{V}O_2max$ are modified in respect to sex and age. And, when $\dot{V}O_2max$ is expressed in ml/kg•min, it becomes clear that peak values are attained in the late teens (Figure 4). However, some of the changes associated with aging may be related to one's lifestyle. It would be tempting to assign such decrements to the aging process, and certainly aging (such as the loss of muscle mass) must be related to this process. It may be hypothesized that progressive physical inactivity related to aging contributes to the extent of such changes (46). Krause and Raab (46) term this chronic inactivity hypokinetic disease, which they define as "too little exercise" or "less than acceptable energy expenditure." They go on to state that such health problems as heart disease, hypertension, back pain, obesity, and diabetes are highly related to lifestyles devoid of physical activity. Although it appears that in most cases physical training and activity will increase $\dot{V}O_2max$ 10 to 20%, it may not significantly reduce the decline in $\dot{V}O_2max$ with age. However, athletes (male and female) who have continued endurance training through the years will average aerobic capacities as large as those for average normal adults in their twenties (13). In a ten-year longitudinal study reported by Kasch and Wallace (42), 16 middle-aged males, whose mean age at the start of the program was 45 years, showed virtually no change in $\dot{V}O_2max$ (3.38 vs. 3.31 l/min).

Findings reported recently (16, 25) have provoked serious doubt about the absolute dependence of endurance performance on $\dot{V}O_2max$. Moreover, it has been suggested that another physiological variable may be of considerable importance (16, 19, 69), namely, the onset of anaerobiosis or anaerobic threshold (AT), which designates the highest attainable $\dot{V}O_2max$ before a sustained increase in blood lactate occurs (69, 70). The import of this concept is that AT represents the approximate maximum speed that a distance runner may maintain for a prolonged period of time (cf. Biochemical adaptions below). Moreover, the longer the duration of the performance, the more critical an athlete's AT will become as a limiter of that performance (65). Costill et al. (16) reported that two of the world's leading ma-

rathoners, Frank Shorter and Derek Clayton, can run marathons at speeds which would exceed 85% of their $\dot{V}O_2max$. These two athletes appear to possess lower than average $\dot{V}O_2max$ when compared to other elite world-class distance runners, namely, 71.3 and 69.7 ml/kg•min, as compared to an average of 77.4 ml/kg•min for their peers. While there are no data yet available on AT for women, some data for men have been reported. Davis et al. (19) have found the AT to be near 60% of $\dot{V}O_2max$ for normal college-aged males. The AT for nonendurance athletes is somewhat higher at 70% (48), while two independent reports (48, 65) have found the AT of distance runners to average 86 and 84% of $\dot{V}O_2max$ respectively. While these findings strongly suggest that endurance training can increase AT, it is not clear, because of the experimental design, that this is due to training and/or genetic effects.

However, Williams et al. (71) report an average increase of 16% of AT for Bantu natives, who had participated in an intense aerobic/anaerobic training program for a period of 4 to 16 weeks on a daily basis.

Respiratory, Circulatory, and Metabolic Factors in Strength Training and Performance

Among the several factors affecting weight-lifting performance, the most important is the *capacity of muscle to develop high levels of tension* (i.e., strength). Generally speaking, there is a good relationship between the area of the cross section of a muscle and strength; this relationship is expressed as kg/cm^2 (12). Nonhuman mammal values range from 1.5 to 2.5 kg/cm^2, and there is evidence to indicate that human muscle may elicit somewhat higher values (12). One impediment to determining muscle strength in humans is the variation in individual strength performance from test to test (2). Åstrand and Rodahl consider the proportion of ST to FT fibers and the number of muscle fibers which an individual is capable of activating to be two major sources of this variability (2).

Trained male weight throwers who followed strenuous strengthening regimes averaged 60% higher strength scores than their untrained counterparts, while the trained female weight throwers showed a 42% advantage over untrained counterparts (40). A comparison of the trained men and women showed that leg extension differences increased, in that the females could only lift 77% of that averaged by the men. However, the female throwers increased to 65% of that averaged by trained males in arm flexion (40). Therefore, it appears likely that strength potential is determined, in large measure, by genetic predisposition, since the number of muscle fibers remains fixed after birth. Nonetheless, training clearly enhances maximal muscular strength while disuse will diminish strength (22).

Although a review of the literature by this author yielded no data for female weight lifters or throwers, the aerobic capacity, i.e., $\dot{V}O_2max$, for male weight lifters is near 55 ml/kg·min, which is only slightly higher than the 50 ml/kg·min for untrained males (2). Corroborating evidence that slight adaption of the respiratory and circulatory systems results from strength training has been reported by Roskamm (59), who found that male weight lifters have a smaller mean heart volume per kg of body weight (10.8 ml) than nonathletes (11.7 ml). It should be pointed out, however, that the weight-lifter's ratio is affected by the increased muscle mass as a result of strength training. The heart volume for endurance athletes ranges from 13.2 ml/kg (cross-country skiers) to 14.8 ml/kg for professional cyclists (59). As might be surmised, there is a high correlation between heart volume and $\dot{V}O_2max$ (2).

Thus, endurance training clearly is capable of increasing an athlete's oxygen transport capabilities. Moreover, there is an increase in ability to extract oxygen from the blood (see Biochemical factors, discussed below). The increase in $\dot{Q}$, which is the result of an increased SV, is the major adaption relevant to increased blood flow. The high heart volumes for endurance athletes relative to their body weight is another reflection of adaption to endurance training. In contrast, respiratory and circulatory adaptions to anaerobic training, especially short-lived, high-intensity effort, seem to be minimal.

Training for Distance Running

Typical training for distance runners consists of a combination of fast continuous running (6 minutes per mile for 10 miles or more) and intensive interval running (e.g., 55 seconds per 440 for 8 to 10 440's). For world-class runners, training usually entails an all-year regimen of twice-daily sessions (5). The critical aspect of training seems to be its intensity, that is, running speed (39). Coincidentally, intensity also seems to be the primary factor for strength training (12). The elite distance runners (e.g., marathoners) will commonly average in excess of 120 miles weekly on their training programs.

Training for Weight Lifting

Strength training may take several forms: *Isotonic* (range of motion), both concentric and eccentric types; *isometric* (no joint movement); and *isokinetic* (range of motion at a constant speed). Thistle et al. (67) compared

the effects of each technique after an eight-week training program. He reported an average strength increase of 27.5% in isotonic subjects; 9.2% in isometric; 35% in isokinetic; and a 9.4% increase in control subjects. The effectiveness of each technique in developing strength is coordinant with the demands of the training situation or event for which the training is intended; the underlying goal is to achieve hypertrophy (cf. discussion of biochemical factors, below). Muscle cells are not alone in their adaptions to strength training, since similar changes accrue to connective tissues, while ligaments and tendons are also strengthened (12).

Biochemical Factors and Effects in Endurance Performance

As a response to training for distance running (aerobic exercise), muscle fibers undergo an adaption which enhances endurance performance; this adaption, when coupled with increases in $\dot{Q}$ is reflected in an increase in $\dot{V}O_2max$ (39). In trained athletes, muscle lactate levels are reduced, (31) indicating a reduced anaerobic metabolism for a given rate of work. Contrary to earlier belief, the increase in lactate levels does not necessarily indicate a hypoxic state in the exercising muscle (41), but more likely an imbalance between the glycolytic rate and pyruvate uptake by the Krebs Cycle (37, 41). Another adaption in the trained individual is the greater production of energy from fatty acids and less from carbohydrates, thus sparing glycogen (32, 37).

Myoglobin has been found to increase as a result of aerobic training. The magnitude of this increase, as with the above changes, is related to the frequency, intensity, and duration of a training program (37). As a result of a 12-week endurance training program, an 80% increase in the myoglobin of dark muscle (i.e., ST fibers) in rats was observed (55). The importance of myoglobin muscle content is that it has been found to be highly correlated with the respiratory capacity content of the mitochondrial fraction of skeletal muscle (47), which also saw a twofold increase in the trained rat muscle's capacity to oxidize pyruvate (36).

Similar human results were reported by Morgan et al. (51), who found a 45% increase in the capacity of the quadriceps to oxidize pyruvate following two hours of daily training for a one-month period. In a study of ten male subjects, each exercised with one leg on a bicycle ergometer, with the work load being progressively increased from 300 to 900 kpm·min, specificity of muscle adaption was clearly demonstrated since no changes were found in the nonexercising leg.

Gollnick and King (30) found that the increase in mitochondrial enzymes was not as great as the increase in size and number of mitochondria. An in-

crease in the oxidation of fats (as high as twofold) which, as stated earlier, represents a major source of energy in prolonged exercise, has also been demonstrated (11). The question of why a trained individual will use more fat as a fuel source, compared to an untrained individual, may be partially answered by an increase in control of carbohydrate metabolism (37), which, according to Newsholme (52), is achieved by an unknown allosteric mechanism related to the rate of fatty acid oxidation in muscle. While the changes in mitochondrial enzymes may range from 100% to no change, the differences that occur result in a mitochondrial content and enzyme pattern more closely resembling that found in cardiac muscle (37). In contrast to the above changes found in skeletal muscle consequent to endurance training, no such changes are found in cardiac muscles with endurance training (39) when the activity of various mitochondrial enzymes are expressed in grams of heart muscle for trained rats. However, there is an increase in the rate of myocardial protein synthesis, which in turn results in physiological hypertrophy (39).

In summarizing the biochemical adaptions to endurance exercise, it may be concluded that the increase of $\dot{V}O_2max$ is due to the adaption of the mitochondrial content and respiratory capacity of skeletal muscle and physiological cardiac hypertrophy due to increased cardiac protein synthesis.

Biochemical Factors and Effects in Strength Training and Performance

Anaerobic energy supplies to skeletal muscle are considered to be adenosine triphosphate (ATP) from creatine phosphate (CP) and glycolysis (i.e., degradation of glycogen to lactate). ATP and CP are stored in muscle in the form of phosphogens and provide an anaerobic energy source during exercise (29). Phosphogen breakdown in exercise is proportionate to the intensity of exercise (34, 44) and appears to be regulated by the phosphorylase reaction (29). Since there is a considerable lag in oxygen transport at the onset of exercise (the half time being approximately 30 secs), these anaerobic energy stores are important to aerobic as well as anaerobic sport performance (39). Intense anaerobic training has generally produced no effect in enzyme activites of the glycolytic pathways (37). Holloszy et al. (38), arguing that earlier studies may not have employed training intensities sufficient to elicit changes in these enzymes, selected key rat enzymes of the glycolytic pathway but found no changes in the rats as a result of strenuous training programs.

Although protein synthesis accelerates in both endurance and strength training, there is a specific change related to the contractile proteins (21). It appears that actin and myosin are attached to the myofibrils until they en-

large and divide into two normally sized myofibrils (12). Thus, hypertrophy due to weight training is the result of enlarged muscle fibers due to an increase in myofibrils.

Differences in testosterone levels are thought to play an important role in muscle mass differences between men and women (27), since this hormone is known to be involved in protein synthesis and muscle hypertrophy (62). Moreover, there is evidence that testosterone levels are affected by maximal (i.e., intense) exercise (66). However, correlational analysis has failed to substantiate a significant relationship between changes in testosterone and strength as the result of a single training session (27).

A recently developed technique of muscle biopsy (4) has resulted in elucidation of ST and FT fibers in athletes. Gollnick et al. (28) have found that sprinters have a muscle composition which is 73% FT, while another study of three sprinters gives a mean value of 79% (15). Thorstensson et al. (68) have found relatively low FT percentages for endurance athletes, i.e., 41 and 33% for racewalkers and orienteers. The mean percentage of FT fibers for 14 elite distance runners was 21%, while good distance runners had 38% and a control group had 42% FT fibers (16). A moderate correlation coefficient ($r = .48$) has been reported between the percent of FT fibers and muscle strength (peak torque) in leg extension (68).

In summary, the adaption to anaerobic training of high-intensity exercise is a hypertrophy of muscle cells due to increased contractile protein which results in increased myofibrils. A higher percentage of FT fibers is common in athletes who practice high-intensity exercise and, conversely, a low percentage of FT fibers is common among endurance athletes. The role played by training in these differences is not clearly understood at this time. There appears to be no adaption of the anaerobic enzymes, however, since no changes have been reported in the glycolytic pathways as a result of intense anaerobic training.

Body Composition in Endurance and Strength Performance

The term body composition (BC) signifies the ratio of lean weight to fat in body weight. Generally, in all trained athletes this ratio is determined by the demands of a particular sport and/or the training necessary for high levels of competence in a sport (20). Two of the commonly used methods for determination of body composition are skinfold measurement and hydrostatic evaluation (54). These techniques dichotomize lean and fat weight, and the relative fat is then expressed as a percentage of the body weight.

While the average relative fat for young, healthy untrained adult males has been reported to be near 15% of body weight (72), it is known that for his average female counterpart it is around 26% relative fat (73). Exercise-dependent changes in body composition show relationships among such parameters of training as the frequency, intensity, and duration of the entire training regimen (10, 64). However, as Carter and Phillips (10) have reported, in relatively moderate endurance training most of the reduction in fat will occur during the first year of training. They found that seven sedentary middle-aged adults experienced virtually all of the 20% decrease in fat weight during the first year of a two-year program. Unfortunately, no longitudinal studies have yet been reported comparing body composition changes before and after long-term training for competitive distance runners. But Costill et al. (18) compared male marathon runners with a group of age-matched college teachers of similar weight. Using skinfold measurement, they estimated the body density and fat and found a relative fat of 16.3% for the teachers and 7.5% for the marathoners. Pollack et al. (56), also using a skinfold technique, have reported a mean relative fat of 4.7% for 20 male elite distance runners, and also reported an average relative fat of 6.1% for "good" male distance runners (14, 64). Using the hydrostatic technique to determine body composition, Sucec (64) reported a significant difference in mean relative fat between male high school ($n = 11$), college ($n = 10$), and club runners ($n = 8$), namely, 7.8, 6.3 and 5.2% respectively. These relative fat scores showed a relationship with running performance for the one-, two-, and three-mile runs, yielding correlation coefficients of .35 to .52. Wilmore et al. (74) have also reported a correlation between relative fat and performance level. Moreover, with continued intense competitive training, there seems to be a continued reduction in relative fat (64). Although it might appear that age is related to relative fat, it is unlikely since it has been reported that relative fat for untrained males is fairly constant from the ages of 9 to 29 years (74).

In a cross-section study using a hydrostatic technique, Wilmore et al. (74) reported an average relative fat of 8.4% for 70 world and national class female distance runners between the ages of 9 and 51 years. By comparison, they reported a mean value of 6.1% relative fat for sprinters and distance runners, a value considerably lower than the 19.3% reported by Malina et al. (49). Therefore, the differences between untrained males and females and the elite distance runners in relative body fat are quite dramatic. That is, both the male and female distance runners have relative fat percentages only one-third that of their untrained counterparts. Moreover, the elite male runners have 40% less fat on the average than the elite female runners.

While excess weight, or fat, can be and often is, crucial for distance running performance, it is of considerably less consequence for weight lifters and throwers. As an example, Wilmore et al. (74) found an average relative fat of 22% for female shotputters, discus, and javelin throwers, while Fahey et al. (26) and others (3) have reported means from 16.3 to 19.6% for relative fat scores in male throwers. The means of relative fat in male weight lifters, as reported in two separate papers (26, 63), were 9.8 and 15.6%. It seems obvious that the mean scores for relative fat for both the female and male weight lifters and throwers is similar to the mean in their respective untrained peers.

Somatotype in Endurance and Strength Performance

Somatotyping the anthropometric description of body size, shape, and composition has established clear distinctions among sports groups when applied to athletes from moderate (e.g., high school and college varsity) to outstanding (e.g., national and world class) performance levels (9, 45). Figure 6 is a somatochart and represents graphically the three components of a somatotype, as first devised by Sheldon (61). These three components are

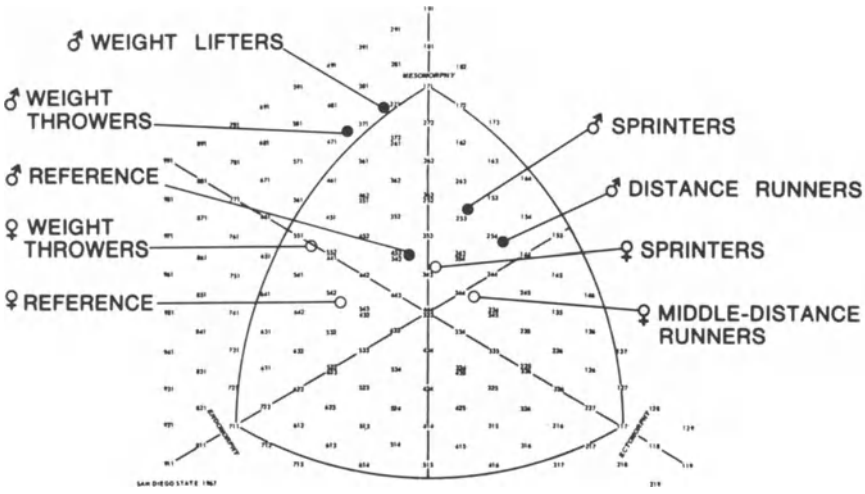

Figure 6. Somatochart devised by Heath and Carter (8), showing mean somatotypes for various sports groups of male and female athletes as well as reference groups. Somatochart and data adapted from de Garay, Levine, and Carter (20) with permission.

endomorphy, mesomorphy, and ecotomorphy; they are presented by axes, which divide the graph into three sectors. Consequently, an individual's somatotype is reported as a tripartite score, with the order being the same as the above components. The individual receives a rating based on a scale from 1 to 10 (i.e., 1 equals an extremely low score, while 10 represent an extremely high score), which indicates the dominance for a given component. Therefore a 1-4-4½, which is the somatotype of Bikila and Wolde, both Oympic marathon champions, indicates a very low endomorphy (20), while the somatotype for the male heavyweight silver medalist in the 1960 Olympics, Redding, is 4½-10-½ which, in contrast to the marathoners, is moderate in endomorphy, very high in mesomorphy, and very low in ectomorphy (20).

If, as de Garay et al. suggest, "one views the human body as a system which has certain functions to perform," and has basic materials such as fat, muscle, and nerve, etc., then it could be reasoned that these materials are largely genetically predisposed (20). Environmental factors such as diet and training could, of course, have profound effects on the physique. Therefore, these mean somatotypes reflect both genetic endowment and changes resulting from years of intensive training in each athlete's specialty.

As observed in Figure 6, it is clear that distinctly different somatotypes represent the general demands of the sport. The mean female somatotype for middle distance runners, 2-3.3-3.7, shows a dominance of ectomorphy (linearity) with mesomorphy slightly inferior, in contrast to the reference groups, whose mean somatotype of 5-4-2.5 is a considerably lower endomorphy (fatness) and high ectomorphy. However, when comparing the runners with the weight throwers, whose mean somatotype is 5.3-5.2-1.7, there is a dominance of endomorphy and mesomorphy with a low ectomorphy. Just the opposite pattern is seen in the runners. In short, there is slight increase in mesomorphy going from the distance runners to the weight throwers, as well as a shift from the dominance of ectomorphy to endomorphy. Thus, it is clear that the distance runners need a minimum of muscularity to perform at world-class standards while the weight lifters need maximum muscularity.

The Role of Psychological Factors in Performance

It is not within the realm of the physiologist to examine in detail the psychological factors clearly at work in human performance. That there is a role is virtually undeniable, for, as we have seen, the limits of the human organism in physiologically adapting to stress are not yet in sight. The most compelling evidence to support this contention is found in the progression

of world records (cf. Figure 1); likewise, some insight into the role of psychology in physiological adaption can be obtained by a brief look at this dimension. Indeed, Ryder et al. (60) state that "at present the factor limiting record performance may be pathological or psychological, but it is not physiological." Analyzing the progression of world records in various distances, and with the objective of projecting the percentage by which and in what time frame these records will be superseded in future performances, Ryder et al. (60) contend that the runner has two adaptive needs: "1) To induce changes that will enable him to better meet the demands of his external environment and 2) to maintain homeostasis, the essential constancy of his internal environment." What induces the distance runner to subject himself willingly to progressively increasing training stress, and make the necessary respiratory, circulatory, enzymatic, musculoskeletal, and endocrine adjustments which allow him to perform most efficiently under that stress? Ryder et al. (60) conclude that it is the psychological impetus of the records themselves: "Our investigations of footracing have led us to the conclusion that the barrier to be overcome by the runner who wants to be a champion is psychological: The last record set and the willingness of athletes to try to break it are the determining factors for the next record. Moreover, a champion stops not on achieving a given rate of speed but on winning a given medal." This statement contains the key to the particular contribution of exercise physiology to our knowledge and understanding of the integral, complex functioning of the human organism.

Implications for the Perfection of the Human Organism

In many ways, the question of whether or not the human organism can be perfected is unanswerable; however, the extent to which it can be perfected is just beginning to be explored. The findings to date are gratifying and inspiring; additionally, they have profound consequences for all investigators and practitioners connected with human health and well-being, as has been demonstrated in many of the studies cited in the body of this paper, as well as in ancillary studies specifically related to health aspects of exercise physiology (1, 6, 10, 23, 42, 46). Briefly summarized, some of the benefits of low-intensity long-duration (e.g., 30 minutes or more) exercise, as outlined above, include the improvement of cardiovascular function (42), along with the reduction of blood pressure in hypertensive adults (6); to the reduction of fat storage as well as overall weight (54); and, at the same time, maintenance of one's lean weight. Perhaps most importantly, habitual exercise may substantially reduce the effects of aging, which is of increasing concern in the sociological as well as in health and behavioral sciences: What, indeed, is the value in prolonging life if its vitality is diminished?

The overriding benefit accruing from a well-designed exercise training program is perhaps the least measurable, except in anecdotal terms: Namely, the increased sense of well-being and self-sufficiency (46) attested to by devoted endurance athletes as well as by individuals who undertake a training program for leisure recreation activity or on medical advice. In an age and culture that impinges significantly on the feeling of individual self-determination, the potential of such a training program in restoring to an individual control over his own life appears as boundless as the physiological adaptation of the organism to stress now seems in the light of exercise physiology.

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The Comprehensive Handbook of Behavioral Medicine, Volume 1

CHAPTER 7

Behavioral Approaches to Exercise Habits and Athletic Performance

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INTRODUCTION

Behavioral procedures have been used in the analysis and modification of numerous aspects of health, including obesity[45] and smoking[1]. However, one health behavior that has received relatively little research attention is exercise. The major purpose of this chapter is to provide an introduction to the use of behavioral principles for influencing exercise habits and athletic performance.

It is surprising that behavioral principles have not been extensively applied to exercise. Exercise behaviors comprise a large number of overt motoric responses that differ in topography, intensity, frequency, and duration, and thus are ideal for behavioral measurement techniques. In addition, exercise behaviors would appear to be responsive to stimulus and reinforcement control procedures[3]. A large literature has been developing which indicates the potential importance of exercise for physical[14, 17] and possibly mental health[13, 34, 35, 42].

This review is divided into four sections. The first section relates exercise or activity levels to obesity. This relationship is of interest because obesity is a common topic for study by behavioral investigators. Obesity is a disorder in which a person's input exceeds his output. A large majority of behavioral research on obesity has focused on the study of input. The data to be presented in this chapter show the importance of studying input-output, or

energy balance, relationships for a better understanding of obesity. A short overview of the measurement of exercise behaviors is presented in the second section. The third section reviews the scant literature on the acquisition of exercise behaviors. Some behavioral factors that may be related to acquisition and maintenance of exercise are discussed. The fourth section highlights studies in a new, rapidly growing area of exercise behavior, behavioral analysis of sports performance.

THE EFFECT OF EXERCISE ON CALORIC INTAKE AND WEIGHT

Physical activity exerts an important effect on body weight. Body weight is a function of the number of calories consumed minus the number of calories expended through basal metabolism and physical activity. Excess of body weight for obese persons may be due to a high caloric intake, low level of activity, or a slightly increased intake relative to output. Although the nature of the caloric imbalance may differ between individuals, research strongly suggests that differences in body weight between normal and obese individuals are more a function of differences in activity than differences in caloric intake. This finding runs counter to the idea that obesity is simply a function of overeating. While a decrease in food intake is certainly the quickest way to decrease weight, it is important to realize that the variables used to change behavior are not necessarily those that are operating in the acquisition of that behavior. For example, the importance of activity in the gradual accumulation of weight can be easily demonstrated. If one person walks two miles per day (one mile back and forth to work) more than a second person, and their metabolism, food intake, and other activity are approximately equal, then the first burns 200 calories per day more than the second, 1000 more per week, 50,000 per year, enough to account for over 10 pounds of weight difference per year.

The difference in activity level for normal-weight and overweight adults was studied by Chirico and Stunkard[3]. Pedometers were used to record the miles walked per day by pairs of adults matched for age and occupation but differing in weight. The leaner females averaged significantly more miles than the obese females. The differences were in the same direction, but less pronounced for males.

Obese adolescents also appear to be less active than their thinner peers. Bullen, Reed, and Mayer[2] used time-lapse photography to study the activity of normal-weight and overweight girls attending summer camp. The girls were observed at swimming, tennis, and volleyball, and an analysis was made of the percentage of picture frames in which the girls were inactive. Although both the normal-weight and obese girls were supposedly

playing tennis, the obese girls were inactive on nearly 60% of the picture frames compared to 15.2% for the normal-weight girls. While swimming, the obese girls spent much of the time floating in the shallow end of the pool, while the thinner girls stayed in the deep area and were actively swimming.

Johnson, Burke, and Mayer[24] studied 28 matched pairs of normal-weight and overweight adolescent girls. The obese girls consumed 600-700 calories per day less than the normal-weight girls, but also participated less in school athletics. Lower levels of caloric intake and differences in activity were also found for adolescent boys[43], but as with the adults, the differences in activity were less pronounced for males than for females.

Although it may be difficult to determine whether the inactivity is a cause or an effect of obesity, the tendency to inactivity appears to be present in the earliest stages of obesity. Rose and Mayer[39] measured the caloric intake, triceps skinfold, and physical activity of 31 six-month-old infants. Activity was recorded with an "actometer" attached to the infant. The thinnest infants actually ate more than the heaviest ones, but they also were considerably more active.

Physical activity affects weight directly by burning up calories, but it may also affect weight indirectly by influencing the number of calories that are consumed. The relationship between caloric intake and energy expenditure was specified in a cross-sectional analysis by Mayer, Roy, and Mitra[30]. Activity level of 300 workers in West Bengal was measured by the physical effort involved in their occupation, and caloric intake was obtained from dietary histories. For normal levels of activity, an increase in activity was associated with an increase in appetite, but weight remained constant. However, this energy balance relationship broke down at the most sedentary levels of activity, where people actually ate more, rather than less, and were heavier.

Although the energy balance among eating, exercise, and weight is widely quoted and is supported by extensive animal research[29], there has been little further research to test this relationship with humans, with the exception of research by Durnin and associates[6]. One reason for this has been the difficulty of making accurate measurements of activity level in the natural environment.[7] To avoid this difficulty, we thought that it might be possible to study the relationship between intake, expenditure, and weight by utilizing a sample of activity in a standardized situation rather than trying to measure the total daily activity. In our first study, we decided to use performance in an aerobic exercise program as a sample of activity[12]. Seventeen nonobese females participated in a five-week aerobics exercise program. They attended the exercise class five times per week during this period and were required to walk or jog one to two miles each day. The

average daily running time was used as an index of activity, with slow running times related to inactivity. Caloric intake was self-reported for each weekday over the five-week period and converted to calories by the experimenters.

According to Mayer's curve[30], the relationship for persons who are within the moderate activity levels should be that greater activity is associated with greater caloric intake, with weight remaining relatively constant across activity levels. The data for this study support this relationship. The correlation between weight and activity level was $r = .08$ ($dF = 15$, $p > .05$), while the correlation of the mean daily caloric intake with mean daily running time was $r = .69$ ($dF = 15$, $p < .01$), suggesting that fast running times were associated with larger caloric intake. The correlation between caloric intake and weight was $r = .14$ ($dF = 15$, $p > 0.5$).

Similar relationships were observed when performance on the 12-minute test was used. The 12-minute test, popularized by Cooper[4], is a field test of aerobic capacity in which people are asked to run or walk as far as they can in 12 minutes. Performance on the test correlates .90 with treadmill measurements of oxygen consumption[4] and has a test-retest reliability of $r = .94$[5]. Our subjects took this test prior to participation in the exercise program. Performance on the 12-minute test, a single determination of activity level, correlated highly with daily caloric intake, $r = .65$ ($dF = 15$, $p < .01$), and was again unrelated to weight, $r = .09$ ($dF = 15$, $p > .05$).

The correlations between performance on the 12-minute test and both caloric intake and weight were replicated in a second study. Nineteen female subjects were given the 12-minute test on two occasions, one week apart. The correlation between performance on the two tests was $r = .92$. During the intervening week, the subjects self-recorded their food intake. The data were converted to calories by the experimenters. Average performance on the first 12-minute test was correlated with daily caloric intake, $r = .48$ ($dF = 17$, $p > .06$), and with weight, $r = .05$ ($dF = 17$, $p < .05$).

These results suggest that the part of Mayer's curve pertaining to nonobese persons can be replicated using the performance on the 12-minute test as a sample of activity. Further research with more sedentary, obese individuals is needed to determine whether the entire energy balance curve can also be established using this measure.

The studies of the relationship between activity and intake discussed so far have been cross-sectional analyses in which the caloric intake of inactive individuals is compared to the intake of active persons. Although animal studies[10] suggest that the same results can be produced by varying the activity of a single subject, comparable human data are sparse. Epstein, Masek, and Marshall[9] studied the effect of an increase in prelunch activity on lunchtime caloric intake. Six obese Head-Start students were engaged in ac-

tivities such as relay races, tricycle races, dancing, and jogging for 10 minutes prior to lunch three days a week for two weeks and were praised for active participation. The behavior of the children was observed on a 10-second time-sampling basis during the activity period and categorized as active or sedentary. Food consumption during lunchtime was assessed by weighing the food before and after the subjects ate and converting to calories. Weight was measured at weekly intervals.

The activity manipulations produced a significant increase in prelunch activity and an overall significant decrease in caloric intake ($X = 380$ calories) compared to baseline caloric intake ($X = 487$). Moreover, the changes in activity were significantly related to the decreases in intake, $r = .90$[10]. Those subjects who had the greatest increase in activity also had the greatest decreases in intake. Since measures of evening caloric intake were not available, it is not known whether children compensated for the lunchtime decrease in intake by consuming more food than normal at dinner.

MEASUREMENT OF ACTIVITY

Activity in the natural environment has been assessed in one of two ways. First, self-report measures of activity have been used, in which persons estimate the duration and often the intensity of the activity, over intervals as short as a day[26] or as long as a year[49]. The second procedure is to estimate activity levels from job requirements, as Mayer[30] did when studying activity-obesity relationships, and by Morris[36] and Paffenbarger[37] in studying the relationship between activity and heart disease. Both techniques may give an estimate of activity level, but neither provide fine-grained analyses of activity topography, intensity, or duration. Behavioral methods for directly studying the class of exercise behaviors may be easily developed to fill this void.

In our investigation of the relationship between activity level, caloric intake, and weight a very simple measurement of activity was used, distance run/walked in 12 minutes. Since the relationships found using the 12-minute test were similar to those found for activity level during work[30], it seemed possible that performance on the 12-minute test might be related to general activity level. The second study by Epstein, Wing, and Thompson[12] examined relationships between the 12-minute test and two measures of natural activity level, the pedometer and daily logs of activity. Subjects were asked to wear a pedometer for seven days and to record the miles walked each day. Subjects also recorded the duration of specific activities engaged in during the day, and these data were converted to caloric output. Performance on the 12-minute test correlated significantly with

both daily pedometer readings (r=.49) and daily logging of specific activities (r=.54). The daily pedometer readings correlated r=.59 with daily logs converted to caloric output. These results suggest that performance on the 12-minute test may be providing a sample of overall naturalistic activity.

While pedometers have been used in the assessment of activity levels in several research studies on obesity[3, 44], they have not been used extensively in measurement of activity level. Pedometers provide a simple mechanical way to assess distance traveled during a day. However, there are certain limitations to the use of pedometers. They tend to break relatively easily, and must be placed at a specific spot on the body (usually the hip) and remain perpendicular to the ground to operate properly, and to adjust them to read actual distance walked, calibration adjustments must be made for each subject[15]. Unadjusted readings will provide estimates of relative increases and decreases in activity within one person, but may not be comparable across persons. LaPorte, Kuller, Kupfer, McPartland, Matthews, and Casperson[26] have validated an improved movement-activated measure of activity based on activity sensors originally used in psychiatric investigations[32]. The devices used by LaPorte et al. are piezo-electric units that sense movement of any part of the body the sensor is worn on, which have included wrist, trunk, or ankle. Since the activity sensors have not yet been calibrated for distance, measures across subjects may be difficult to compare but relative changes within subjects are possible.

Pedometers and activity sensors provide general estimates of activity, but they do not provide information on the topography or intensity of the activity. The most direct way to assess these factors would be to use behavioral observation procedures. Hovell, Bursick, Sharkey, and McClure[20] developed a behavioral observation procedure that rated the activity of children in three levels for arm and leg movement on a 5-second time sampling basis for 50 consecutive observations. Results of the measurement study showed that intensity of arm and leg movements could be reliably observed, that the children were active approximately 60% of the time, and that children were significantly less active than persons engaged in aerobic exercise.

Epstein, Masek, and Marshall[9] used a less comprehensive observation procedure to assess the activity level of obese children during a free play period. The children were observed on a 10-second interval basis and activities were scored as active or sedentary. The operational definitions of active or sedentary were made in relationship to the specific activities available to the children during free play, and all active responses involved movement transversed over space. Results showed that the children significantly increased activity during a structured exercise period compared to an unstructured free play period.

ADHERENCE TO EXERCISE

One of the major contributions that behavioral medicine can make to exercise research is to help develop technologies that promote adherence to exercise programs. Several studies have shown that compliance to exercise programs is typically very poor. Montoye, Van Huss, Brewer, Jones, Ohlson, Mahoney, and Ohlson[33] arranged a five day per week program of calisthenics and swimming to decrease serum cholesterol in adult males. They reported that subjects attended approximately one-half of the exercise sessions. Mann, Garrett, Farhi, Murray, and Billings,[28] found that only 58% of the subjects attended at least one-half of the scheduled five sessions of exercise per week over a six-month program. Almost half of the subjects that did drop out terminated by the end of the fifth week.

Adherence is equally poor in exercise programs that the person does on his own. Cooper[4] reports a dropout rate from jogging programs of 30 to 60%. Gwinup[16] found that less than a third of the obese female subjects in an exercise program were able to meet the criterion of maintaining 30 minutes or more of exercise daily for a year or longer.

The initial use of behavioral procedures to influence adherence to aerobic exercise was by Epstein, Thompson, and Wing[11]. Thirty-seven female college students participated five days per week in an aerobic program for five weeks. The subjects were assigned to five groups, three contract groups which differed according to exercise intensity, a lottery group, and a no-treatment control group. Subjects assigned to the contract condition were required to deposit $5.00 prior to the study and were refunded one dollar per week contingent on attendance at four of the five exercise sessions. Subjects in the lottery condition deposited $3.00 prior to the study and were able to earn a chance in the lottery by attending four of the five exercise sessions for a given week. The lottery prize was a $21.00 gift certificate or the equivalent cash prize. No contingencies were arranged for the control group.

Analysis of attendance data showed that mean sessions attended for the three contract groups and the lottery group were equivalent and superior to the attendance in the control group.

Much additional research is necessary to develop technologies for increasing adherence and maintenance of exercise habits over long periods of time. There are numerous obstacles hindering the acquisition of exercise behaviors. For example, many of the people who want to begin programs are in poor physical condition initially and need to begin an exercise program quite slowly. Starting a program with too-intense or too-long duration exercise can result in a cardiovascular or musculoskeletal accident, or at

least in numerous aches and pains that require considerable rest between exercises. Everyone knows the person who begins to exercise too vigorously each Monday, and has to take off the remainder of the week to recuperate for the next Monday. Starting slowly, however, is often frustrating to the would-be athlete. Another drawback to the successful initiation of an exercise program is the delayed nature of losing weight, or improving cardiovascular and respiratory function. As anyone who has begun an exercise program knows, there is much pain before there is pleasure.

Numerous behavioral techniques may be readily applied to the appropriate acquisition of good exercise habits. One useful technique to improve adherence to exercise may be self-monitoring of the frequency and duration of exercise. Self-monitoring has previously been found to be the single most important factor in weight loss[38], as well as being useful in improving medicine compliance[8]. After a program is developed, self-monitoring may provide feedback to the person about how well the program requirements are being met. A second useful technique may be modeling. By having the person exercise with an experienced individual or group of exercisers, the novice exerciser can be taught the appropriate preexercise "warm ups" and postexercise "cool downs." Modeling can also be used to teach people to pace their running, to ensure beneficial, but not too extensive exercise intensity. The exercise setting may also be important, as group support programs may facilitate acquisition.

After appropriate exercise habits are acquired, it is hoped that positive feedback from their body, physical changes, and relaxing time away from work will be sufficient to maintain exercise. However, these changes may not be sufficient, and other motivational factors may be considered.

Heinzelman and Bagley[18] report that the support of a wife is a critical determinant of a husband's continued participation in an exercise program. This variable, which has also been found important in weight-loss literature[23,27], deserves more systematic investigation in exercise programs. Motivation in exercise programs might also be improved by increasing the frequency of contact with the therapist[22] and by using larger financial contracts contingent on exercise performance in addition to attendance[21].

BEHAVIORAL SPORTS PSYCHOLOGY

The previous sections dealt with exercise as it relates to health and fitness and a healthy life style. Behavioral principles may also be applied to a specific subclass of exercise behaviors—competitive athletics. Performance in athletic events may be a function of several factors that can be influenced by behavioral principles.

Suinn[48] considers athletic skills to be a function of basic ability combined with learning, experience, and training. Performance in an athletic event is related to the strength of the correct athletic response, the degree to which performance can be transferred from practice to performance conditions, and the reduction of incorrect responses that compete with the correct performance in skill events or that utilize extra energy in endurance events. These components may be broken down into physical and psychological factors. Physical ability is a prerequisite for good performance, and this ability is responsive to shaping or training techniques. Fitness necessary for the event is also a function of training. However, it is well known that the most fit athlete does not necessarily do the best in a particular event, partly because of psychological factors that mediate his performance. The athlete may not be "up" for the event, he may be too aroused and engage in anxiety-related responses that interfere with correct performance, or the transfer of the skill from a practice site with no onlookers or competing stimuli to a noisy stadium/auditorium with numerous competing stimuli may prove difficult. The following section will review the behavioral procedures currently being developed to deal with these and related performance issues.

The basic fitness components of athletic performance are provided by training. While the importance of proper training is agreed upon, anecdotal reports of poor athletic performance due to inadequate or incorrect training or poor motivation are common, and it cannot be assumed that athletes who want to perform well will automatically do the necessary amount or type of training. Self-recording may be useful in regulation of training behaviors. McKenzie and Rushall[31] performed two studies to assess the effects of self-recording on training behaviors of competitive high school swimmers. In a first study three characteristics of participation in practice were manipulated in a multiple baseline fashion. Athletes were to publicly self-record attendance at practice, tardiness to practice, and leaving practice early in a sequential multiple baseline design. Introduction of the self-recording was associated with sequential changes in the targeted responses. In Study 2, eight swimmers publicly self-recorded the number of laps completed during practice according to preplanned coach's instructions. The self-recording was implemented in an ABAB withdrawal design. Rates of lap swimming increased during use of the first program board, decreased during the withdrawal, and subsequently increased with the second self-monitoring condition. Results of both studies suggest maintainence of the desired behavior change after and during follow-up. Lottery and contract procedures previously described[11] may also help regulate training behaviors.

While swimming is an endurance sport, and requires distance and speed as major training goals, skill sports require shaping of correct execution of

athletic behaviors. Komaki and Barnett[25] assessed the effects of behavioral specification and feedback on play execution for the center and four backfield members of a Pop Warner football team. The training package included instructing each of the five players in exactly what they were to do on three different plays and providing feedback on performance of each play. Results showed reliable increases in play execution for the young athletes, with marked individual performance changes observed for the quarterback.

Heward[19] performed an analysis of motivational procedures on the offensive performance of the Indianapolis Clowns, a barnstorming baseball team. These players were assumed to have necessary skills, but showed variable performance, which may have been related to motivational problems. Heward designed an efficiency average that reflected hits, runs, runs batted in, walks, sacrifices, and hit by pitches to assess offensive team performance. Three levels of meal money were provided to the three players showing the top three efficiency averages. Money was made available and withdrawn in an ABAB withdrawal design. Analysis of efficiency averages and runs scored showed large increases with the first meal money condition, no decrease in return to baseline conditions, and a subsequent decrease in both measures during the final meal money condition. Heward suggested that functional relationships were not observed during the last two conditions because of social support for improved performance after external reinforcement was removed, and the unfortunate coincidence of the final reinforcement procedure at the end of the season, when motivation and performance naturally decline.

Behavioral analyses of sport by Suinn[48] suggest that anxiety reactions may influence performance, as well as physical training and motivational factors. In an initial report[46], Suinn reported the use of desensitization and behavior rehearsal to improve competitive performance of college ski racers. The behavior rehearsal involved relaxation and then covert visualization of their performance on a course. The procedure was adapted to a slow-motion instructional technique which allowed skiers to covertly repeat a race to discover errors and practice correct responses. In 1977 Suinn[47] reported his experiences as Psychologist of the Nordic Ski Team and Biathlon Team at Winter Olympics in Innsbruck. A combination of procedures was reported, including desensitization, thought stopping, covert reinforcement, and the ingenious use of covert behavioral rehearsal in combination with in vivo practice. Although Suinn was not able to collect data in standard experimental designs to verify impressions of the utility of the treatments, he reports favorable anecdotal results. The development of similar procedures to reduce stress symptoms that interfere with performance may yet prove to be the most interesting aspect of behavioral sport psychology.

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The Comprehensive Handbook of Behavioral Medicine, Volume 1

CHAPTER 8

Stroke and Rehabilitation

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The need for good rehabilitation services becomes desperately obvious in most victims of stroke even before the unstable acute phase has passed. Yet, by and large (that is, on a statistical basis), current physical rehabilitative techniques applied at the acute stages have failed to influence the final outcome (10, 12). Thus, most effective rehabilitation—either "physical" or "behavioral"—probably occurs some weeks or months after the stroke. Many clinicians refuse to accept this dour outlook, and for them the improving methods of behavioral medicine offer the best hope for useful early intervention. Probably myoelectric (EMG) biofeedback has contributed most to the new optimism, despite the fact that the evidence for its effectiveness has emerged almost exclusively from treatment of the chronic stages, some months or years poststroke (5).

A complete account of stroke and current methods for rehabilitation is uncalled for here—in fact, it has filled a large book already (11). Stroke is a common condition. More than two million Americans alive today have survived an attack and now bear the burden of varying amounts of neurological deficit. Its classical name, apoplexy, is fighting a losing battle against the expressive term *stroke,* and the latter is now also beginning to win over the dubious term "CVA" (cerebrovascular accident) and "hemiplegia" (which is a bare symptomatic descriptor of unilateral paralysis). *Stroke* its name shall be—a sudden, or relatively rapid, blow that kills 200,000 and maims a larger number each year in the United States.

THE PATIENT AND MANAGEMENT

The stroke victim usually suffers moderate to marked unilateral motor loss and may experience, then recover from a loss of consciousness. Distur-

bances of speech, vision, and coordination are common, and slight to profound psychological changes are almost universal. Thus, the comprehensive multidisciplinary team approach has become almost the rule in the management of the various stages of the recovery period. A modicum of behavioral management, without that clear label, has always played a part in the treatment of the postacute stage. Unfortunately, until the 1940's some of the techniques were actually harmful, e.g., enforced rest, usually dangerous, was all too common. Now specialists agree that mobilization is extremely useful and perhaps essential in rehabilitation of the stroke patient with severe neurological losses.

THE THERAPIES

Physical therapy has become heavily involved in the battle to restore hemiplegic patients to optimal performance levels. Heavy reliance on techniques that purport to elicit natural, unconscious, and reflex neural responses is "high fashion." To the unbiased observer, it has become increasingly obvious that therapists have been indirectly reinforcing and training patients with what we would call behavioral methods. The addition of myoelectric (EMG) biofeedback has moved physical therapists much further into the growing group who practice behavioral management techniques relying on the patient's conscious effort and self-control.

Speech therapists dealing with the communication problems of the stroke patient are also recognizing the increasing role of behavioral techniques. While electronic biofeedback is only in its infancy in speech therapy, practitioners have used some of the techniques under other labels and with considerable success.

SIGNS AND SYMPTOMS IN THE POSTACUTE PERIOD

Motor Loss

Hemiplegia, the characteristic finding in nine out of ten surviving patients, occurs almost always in the upper limb and somewhat less frequently in the lower limb. The severity of paralysis may vary from area to area of the same limb, e.g., the distal muscles vs. the proximal muscles; extensors vs. flexors; and vice versa.

Disturbance of Muscle Tone

Most patients with motor loss pass from an early flaccid stage to a spastic state of the paralyzed limb(s) by the time rehabilitative therapy is begun in

the postacute stages. Spasticity often is a greater burden than the paralysis, especially in the restoration of hand function. It tends to diminish slowly, but if it persists for several months it probably will remain for years.

Cerebral Function

Aphasias and apraxias from damage to speech and cognitive areas of the cortex persist to some degree in about half of all recovering patients. Intellectual function is often slowed, but because many of the patients are debilitated, this may be a secondary consequence of the brain damage in such persons. The almost universal slump in the emotional status of stroke patients is a blend of understandable despair and pathological changes in cortical function; how much of each exists in the blend for a given individual is almost unfathomable. A clearer idea of these factors is mandatory if the approaches of behavioral medicine are to be fully successfull.

Other Symptoms

Stroke patients have a scattering of many other disturbances in the body economy, e.g., visual disturbances, cardiovascular problems which usually predate or cause the stroke, lack of bowel and bladder control, swallowing difficulties, gross sensory losses, severe pain, and contractures in joints, etc. These sometimes dominate the total picture in an individual patient.

THE ROLE OF BEHAVIORAL MEDICINE

As noted before, therapists assigned to treating stroke patients intuitively use behavioral methods even as they "lay on hands." Success cannot be achieved by the mechanical use of treatments, even those dependent on eliciting basic reflexes, without an intense psychosocial interplay between the patient and everyone who is nearby. Thus, depending on the existing emotional status and intellectual level of the patient, everyone who has responsibility or a personal regard for the patient should be alert to the possibilities of recovery as a result of stimulation of innate cognitive and motor-control mechanisms that may have survived the brain damage. Informal systems for doing this are used in many rehabilitation centers and long-term care facilities. However, the intensive loving care of a spouse who has learned how to force the patient to think, to talk, and to use paralyzed limbs to the point of frustration may have been the best behavioral medicine

available in the past. Perhaps the newer methods now under development for reeducating stroke patients are needed only to treat specific symptoms, e.g., foot-drop, which no amount of encouragement by a spouse or friend can alleviate as rapidly as myoelectric (EMG) biofeedback.

BIOFEEDBACK

Practical biofeedback therapy for the rehabilitation of stroke patients is expanding very rapidly. The limits, in practical terms, and expenses in time and effort (hence money), have yet to be determined. In a large proportion of the patients, some severely disabling symptoms of stroke can be relieved rapidly while others absorb many hours of professional therapy over many weeks or months. The massive damage to the brain cannot be repaired or restored to normal function overnight. The patterns of skilled movements and inhibitions acquired during maturation of a normal central nervous system are not going to be made whole by a few days of intensive behavioral therapy. Yet the circumscribed successes of biofeedback have been very impressive.

CONTROL OF MOVEMENT AND POSTURE

Elsewhere (2, 3) I have written at some length about the way in which the human organism acquires skilled behavior. The brain "learns" to pattern an economically spare form of recruitment of motor units by a process of trial and error that depends chiefly on inhibition. Progressive inhibition of the inefficient mass responses that are the basic motor reflexes is an innate capacity of the normal central nervous system (CNS). If the brain is seriously damaged at or around the time of birth, as in cerebral palsy, this capacity is grossly reduced; nonetheless, subsidiary pathways and neural mechanisms may survive to permit "trick movements" and other compensatory ways of achieving mobility and posture. The truly fantastic plasticity of the human brain allows it to shift some function to noncompromised areas. However, clever retraining is needed to imitate even a semblance of "normal" skilled behavior.

In the early 1960's my colleagues and I (1, 3) described a system of training subjects consciously to control with great precision the fine functioning of individual spinal motor neurons. Although patients with massive brain damage cannot be expected to demonstrate the same meticulous control, nevertheless the visual and acoustic displays provided by electronic biomonitoring devices provide them with cognitive and motor feedback on

which behavioral medicine can capitalize. With a motivated patient and a wise therapist, biofeedback allows full use of the conscious potential for retraining both motor activation and motor inhibition or relaxation of undesirable activity.

Training and retraining of motor skills generally occur at a subconscious level, partly by reliance on interoceptive and exteroceptive feedback to tell the brain and brain stem what has happened. To develop engrammatic patterns at the spinal level requires millions of repetitions during growth and development (9). Training, whether it is the child's learning of simple social motor responses or the athlete's and musician's preparation for a specific and complex skilled act, involves the progressive inhibition of many muscles, parts of muscles, and even single motor units that normally come into play when one first attempts to produce the skilled act (2, 3). The athlete and the musician drill and drill until the skilled movement is perfected; we often lose sight of the fact that many of our "simple" acts, such as standing upright or walking, had to be drilled into us over many years. The young animal has enormous amounts of reflex overactivity which must be overcome during training and maturation. Some of these overresponses do not fade out until the mid-teens; others are successfully suppressed during infancy. All or almost all primitive reflexes return when the brain is damaged, and any retraining must start over again with less functional capacity and fewer intact normal mechanisms. Biofeedback and other behavioral techniques can help dredge some of these out of dormancy (4, 7, 8).

Three major symptom complexes have been our targets: Footdrop, with or without spasticity; shoulder subluxation; and reduced hand function. Some of our patients were treated for more than one condition, but generally this was done serially rather than simultaneously. All of the patients reported here had had cerebrovascular accidents months or years before and were thought by their physicians to have reached a plateau in their recovery.

MYOELECTRIC (EMG) BIOFEEDBACK FOR STROKE PATIENTS

Foot Drop

The most dramatic application of electromyographic biofeedback in this decade has been in the treatment of footdrop in stroke patients. A number of clinics (7, 8) as well as my groups have been working with electromyographic biofeedback for footdrop over several years. Although most of these studies have been of a general or an uncontrolled nature, we reported a controlled study in which biofeedback training was compared with that of

conventional physical therapy in 20 hemiparetic patients with chronic footdrop (5). Twenty volunteers—ten men and ten women aged 30 to 63 years—were randomly assigned to one of two equal groups. The first group was treated with therapeutic exercises, while the second group underwent therapeutic exercises plus biofeedback training. The tibialis anterior muscle was selected for study because of its primary function as the chief ankle dorsiflexor which counteracts footdrop.

The increase in both strength or dorsiflexion and range of motion was about twice as great in the patients receiving biofeedback training as in the other group. More importantly, our patients in the biofeedback group achieved and retained conscious control of dorsiflexion in the swing phase of gait. At follow-up, three of these patients continued to walk without their previously needed short leg brace.

Subsequent to this demonstration of biofeedback superiority in a controlled study, we have treated a growing number of patients in a clinical rather than a pure research setting. All of these patients also have been seen as "research patients." All but a few have been highly motivated and their cooperation has been excellent.

A total of 39 patients in our routine treatment program were the subject of our second study of footdrop (6). Patients with and without short leg braces were included. The technique of biofeedback training has been described elsewhere (5). The apparatus employed was the Basmajian/Emory Myotrainer (Biofeedback Technology, Karlin Industries, 940 W. Port Plaza, St. Louis, MO 63141).

Patients Previously Treated With Short Leg Brace

Twenty-five patients had been treated reasonably efficiently before biofeedback training with a short leg brace. All but one of these patients had had their stroke many months or several years before biofeedback treatment, and their foot drop condition had stabilized.

Of these 25 patients, 16 were able to discard their short leg brace entirely following 3 to 25 biofeedback sessions (mean, 16.6 sessions). Each biofeedback rehabilitation training session lasted approximately one-half hour. The remaining 9 patients showed little or no improvement, sometimes for obvious reasons such as poor motivation, severe spasticity, intercurrent illness, and early discontinuance of treatment, e.g., only 3 or 4 treatments in 4 of these 9 patients. Some of the patients were even able to discard their cane for daily living activities. Several now use their short leg brace occasionally when they are on their feet for long periods of time.

Patients Not Previously Treated With Short Leg Brace

Fourteen patients with footdrop had reasonably good function at the ankle and had not been treated with braces. The aim of treatment was to significantly increase functioning. After 3 to 17 sessions of biofeedback rehabilitation training, ankle function failed to improve in only two patients, while six had moderate to excellent improvement of strength and range of motion, which greatly improved their gait.

Effects of Age, Sex, and Duration of Foot Drop

The patients' age apparently was not directly related to the effectiveness of biofeedback. Patients in both the 30- and 60-year-old age groups were among those who discarded short leg braces. The proportion of men and women in whom treatment was successful was the same as in the overall population studied. Neither failure nor success of treatment seemed to be related to the duration of foot drop. Failures occurred in patients with either recent or late stroke, while treatment was successful in patients who had had foot drop for periods ranging from three months to six and one-half years.

Shoulder Subluxation

Our experience with subluxation since 1974 has been uniformly good. Basing our approach on the hypothesis that subluxation is caused by an unlocking mechanism (3), we have concentrated on improving the mobility of the scapula, with an emphasis on restoring the proper orientation of the glenoid cavity. When the glenoid cavity is facing upward as well as forward and laterally (as in normal shoulders), subluxation is eliminated because of the normal tightening of the coracohumeral ligament and the adjacent superior capsule of the glenohumeral joint.

Many patients with subluxation have received muscle reeducation with EMG biofeedback from the muscles of the shoulder region. Clinical results have ranged from moderate to excellent reduction of subluxation and an accompanying improvement of scapular and glenohumeral mobility. Our initial general analysis shows clear evidence of radiographic improvement in almost all subjects (6).

Impaired Hand Function

Increasingly we have been successful in restoring hand function in stroke patients. Both relaxation techniques for spastic muscles and muscle reedu-

cation of paretic muscles are used for forearm and intrinsic hand muscles. Severe spasticity can be moderately modified by targeted relaxation techniques, including immediate acoustic and visual feedback of muscular activity. As our experience increased, we found that a growing number of patients could be trained to relax spastic muscles by direct conscious effort. This in turn allowed greater voluntary use of the hand and strengthened their grip and pinch without exuberant flexor synergies (6).

Our series of patients treated for hand spasticity is too short for statistical analysis; however, a gratifying number of patients have shown substantially improved hand function after approximately a dozen sessions of personally tailored biofeedback rehabilitation therapy. A great part of the effectiveness of this therapy may result from the improved body image and increased motivation that the patient gains from the therapist. However, part of the improvement is clearly the result of restored motor controls which use new or previously indolent cognitive and motor pathways.

RATIONALE

Biofeedback for foot drop in the hemiparetic patient is of undoubted value, especially if the patient has mobility. Particularly dramatic results are seen in those patients who have been forced to wear a short leg brace for footdrop for months or years prior to EMG biofeedback treatment. In our series, almost two-thirds of these patients are able to discard their braces after treatment. The neural pathways involved in this marked neuromotor improvement are unclear. One can suggest two possibilities: Either new pathways develop (highly unlikely), or old persisting cerebral and spinal pathways are mobilized by introducing the artificial feedback loop with EMG biofeedback. The latter explanation is highly probable; the artificial proprioception provided by acoustic and visual responses to peripheral motor activities appears to be a powerful training device. Undoubtedly, new forms of cognition at the cortical level also are utilized. The generalizations we can make about retraining paretic muscles are applicable to learning voluntary inhibition of spastic muscles. The inhibition patterning seems to arise in part from obscure processes in diffuse centers of the cerebral cortex. Since inhibition is a central phenomenon, one must consider the possibility that brain-stem centers, and perhaps the cerebellum, are critically important for this learning. It is too simplistic to consider a schema where an impulse begins in a tiny area of cerebral cortex and then passes directly along a facilitatory path to a desired set of motor neurons. The motor learning process probably involves a neuronal network, with the "main" pathway for motor activation being at most a small part of the whole.

Apparently, patients who succeed in inhibiting marked peripheral spasticity in hemiplegia use surviving pathways to increase the inhibition of overactive motor neurons. By using an "override mechanism," they appear to succeed in relearning how to dampen even the influence of powerful stretch reflexes; for example, our patients learn to move one muscle while inhibiting the usual reactive hypertonicity in its antagonists and synergists.

Relaxation therapy also has a major application, both targeted and generalized, in managing stroke patients who have a lot of emotional stress. While targeted relaxation of antagonists has always been part of our routine, as in foot drop cases, our group began to explore general relaxation for stroke patients only in the past five years. Although I cannot offer statistical evidence for success, I am confident that functional improvement can be obtained in stroke patients with general biofeedback and deep relaxation, much as psychosomatic ailments can be improved in the neurologically intact patient by using this technique. I encourage the widespread use of electromyographic motor retraining of "paretic" muscles, relaxation training of spastic muscles, and general relaxation training of the whole patient.

Our treatment of shoulder subluxation by biofeedback (6) is related to the above treatments, but employs a mechanism that is quite different. In this case, patients are trained to mobilize an area by the usual biofeedback technique, which results in the restoration of a passive, but effective, function of a joint. This treatment technique, which is much superior to the usual treatment of subluxation by the use of slings, relies on a thorough understanding of normal kinesiology (3). The usually bad effects of previous neglect of simple biomechanics can often be reversed.

Finally, although our experience with managing limited hand function in stroke patient is limited, the increasing ability of our patients to inhibit the muscle spasms that result when they attempt to grasp, or otherwise to use their hands, merits widespread testing and validation. Much of the problem in hand function is not only the obvious paresis, but in addition the muscle spasticity which is equally disabling. Combining inhibition training with neuromotor retraining of the weak hand and forearm muscles seems to be a logical approach that should be thoroughly investigated.

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The Comprehensive Handbook of Behavioral Medicine, Volume 1

Introduction To The Central Nervous System

Perhaps nowhere in medical science is there a greater distance between basic research and applied research than in disorders of the central nervous system. In the past 20 years many aspects of central nervous system physiology have been elucidated, and our understanding of psychophysiology, psychopharmacology, and basic learning has been greatly expanded. On a microscopic level we know which cells in the lateral geniculate nucleus and visual cortex respond to vertically striped lines, the adaptation that takes place in the visual system subsequent to ablation of specific cortical areas, etc. We know that inhibition of enzymes by the use of metabolic poisons can prevent memory from being permanently encoded, and that disruption of neural systems by electrical shock can similarly interrupt learning processes. There have been ample descriptions of the effect of enriched and simplified environments on basic structures of the brain such as cortical thickness and dendritic branching. Virtually an unlimited number of further examples could be given to illustrate the wealth of information in this area.

Applied research has attempted to alleviate symptoms of a disordered central nervous system, but in most cases without regard to basic neurophysiological research. It is too soon for a synthesis, but not too soon to look ahead, for each field to see what the other has discovered, and for each to use the implications of each others discoveries: They are working with the same organism but at different levels of abstraction. In this section, we have combined an article that reviews the basic physiology of the central nervous system in terms of "plasticity," a way of looking at the extent to which this system is malleable, adaptable, and hence treatable, and the following papers describe two common central nervous system complaints, insomnia and memory dysfunction.

We envisioned a shorter chapter on the plasticity of the nervous system, but Rosenzweig and Bennett have so superbly discussed the state of malleability in the area, and the implications are of such great importance to be-

havioral medicine researchers and practitioners that we feel that to reduce or simplify it would be a disservice to the reader. Rosenzweig and Bennett point out the transition in thinking about the central nervous system from the entirely plastic, *tabula rasa* physiology of Watson and Pavlov in the 1920's through 1940's, which was supported by the neurophysiology of the day that could find nothing to suggest any one area of the cerebral hemisphere could not perform the function of any other area. Between 1940 and 1960, Sperry and others reversed this opinion with the observation that when these were ablated, function was irrecoverably lost. Work subsequent to this has revived the concept of the physical plasticity of the central nervous system. The observation of dendritic branching and recovery after lesion in a wide variety of areas supports this notion. Observations made in the 1960's conclusively demonstrated that the environment has an extremely profound influence on the developing brain, and that enriching the environment of a developing animal leads to an increase in its brain weight; or more specifically, the thickness of its cerebral cortex. Later work extended this observation from the infant to other age groups, including senile animals, where similar differences are seen between animals in an enriched versus simple environment.

This positive response of the central nervous system not only to harsh intervention, such as central nervous system damage, but also to the mild experience of environmental complexity or deprivation has extremely profound implications. For example, is there sufficient reserve or plasticity in the system to entertain hope that with appropriate treatment procedures central nervous system damage might be even partially "cured," or compensation for damaged areas learned? Similarly, what are the factors that promote and limit the formation of neural connections subsequent to trauma, and what are the inherent limits of the physiological system in terms of performance or intelligence? Basic neurophysiological research appears to indicate that the inherent limits of the central nervous system have been overestimated, and to a large extent are still unknown. Likewise, the capacity of this system for growth, regrowth, and adaptation subsequent to trauma is unknown. There appear to be favored ways for the system to react to trauma, adapt, and relearn function and favored environmental contexts for these reactions; however, the specification of these factors is at this point impossible.

When looking at the common clinical symptom of memory disturbance, many issues arise. The complaint is widespread. As Poon points out, two-thirds of a sample of 600 elderly noninstitutionalized individuals have a complaint of memory disturbance, a percentage that was higher than those in institutions. However, many factors contribute to this complaint. These include lack of concentration, lack of attention, depression, anxiety, and

other psychological states. When one is approached by an individual complaining of poor memory, there are few diagnostic tools that will allow one to specify what the problem is. On the one hand there are a variety of clinical tests, e.g., the verbal mental status exam which gives the therapist not much more than a feeling that something is wrong, and on the other hand, there are very specific tests of memory function like short-term iconic storage which may not be relevant to a specific memory complaint. What is lacking is useful measures of memory related to everyday experience, such as names, facial recognition, recall of directions, etc. Poon points out that the methods for remediating poor memory are basically unchanged since Simonides in 477 B.C.

In addition to specific memory skill methods, care must be paid to the individual's daily needs, environment, and cognitive style. Along with a mnemonic approach to encoding and practical strategies for information retrieval, the patient has to develop a belief or attitude that he can change and that he will succeed tn his attempts to change. There needs to be a measure of progress and proof that skills work. Intrinsic and extrinsic reinforcers are necessary to motivate the patient, and there must be a routine built in to practice skills to ensure generalization and maintenance. A memory testing or training program must be individualized. Poon points out that some individuals are verbalizers, some visualizers, and that basic neurophysiological data indicates that attention must be paid to this difference: Someone who has a verbal memory style does not do well with a mnemonic test which relies on visualization. Finally, the vicious cycle that many elderly individuals enter of socially withdrawing because of the fear that people will realize they have a memory defect and label them as elderly, must be eliminated. Practice, feedback, charts, realistic goals, and reward contingencies are vital components of a treatment program for memory loss.

Insomnia is another pervasive complaint of central nervous system function disturbance. Thoreson et al. point out the subjective nature of this complaint, and its frequent contamination with other factors, for example, drug-induced insomnia, transient emotional disturbance, anxiety, drugs, coffee, alcohol, and the various sleep pathologies such as sleep apnea, nocturnal myoclonis, etc. As with memory, a taxonomy of insomnia is necessary to facilitate treatment. When the above causes for insomnia are eliminated, a population remains that has difficulty falling asleep or staying asleep. In these cases, arousal may be due to introceptive or extroceptive stimuli, phase lag shifts, or other physiological activities which lead to wakefulness. Treatment depends to some extent on diagnostic category. Clearly the individual suffering from anxiety will be treated differently from an individual who has drug-dependency insomnia, and these two will

be treated differently from an individual with ideopathic insomnia. Hence, diagnosis is quite critical. The behavioral techniques used to date rely primarily on modification of the antecedents of sleep activity. These include progressive relaxation, EMG relaxation, and stimulus narrowing techniques to eliminate distracting stimuli and activities in the sleep environment that otherwise lead to or are associated with wakefulness.

Other chapters of this book are relevant to the central nervous system. Neuromuscular re-education, discussed by John Basmajian in the section on rehabilitation of the nervous system, began with demonstrations that spinal motor neurons could be brought under conscious control. This work and subsequent clinical investigations have demonstrated a plasticity or ability of the motor cortex to adapt. Epstein et al. and Cinciripini et al. review the important diagnostic pathophysiology and treatment issues of tension and migraine headaches. Cataldo et al. discuss the application of behavior therapy techniques to two neurological syndromes, epileptic seizure disorders, dystonia movements and chronic pain in children. They point out that many types of seizure activity can be brought under operant control in humans, and that there is some indication that sensory motor rhythm in the motor cortex can be conditioned by the use of biofeedback techniques and that self-produced activity in this part of the brain can abort epileptic discharge.

A large number of case reports in the behavioral literature illustrate the application of behavior therapy techniques for a variety of other central nervous system disorders. Various authors have reported improvement in patients with spasmodic torticollis, tics, and more permanent neurological impairments such as Gilles de la Tourette Syndrome, and the tardive dyskinesias. In at least one of these conditions, Gilles de la Tourette Syndrome, medical treatment has proved superior to behavioral, but research is just beginning on the use of operant and respondent control techniques to help with these problems. Under some conditions and circumstances the nervous system is plastic; it remains for the behavioral researcher to demonstrate, for what and how their techniques may prove useful to enhance central nervous system function and minimize central nervous system disability.

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CHAPTER 9

How Plastic Is The Nervous System?

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INTRODUCTION

Dramatic evidence of plasticity of the nervous system has been flooding in from many sides in the 1960's and 1970's—in striking contrast to the evidence of fixity of neural structures and connections that prevailed in the 1940s and 1950s. Opinions on this topic have ebbed and flowed in the past and may do so again, but the present tide must be recognized. As examples, let us note some representative recent publications that are devoted in whole or in part to this subject: *Plasticity and Recovery of Function in the Central Nervous System* (100), *Neural Mechanisms of Learning and Memory* (79), *Elements of the Behavioral Code* (18), *Brain and Learning* (104), *Recovery from Brain Damage* (30), and *Neuronal Plasticity* (17). The foreword to a recent symposium on protein synthesis in the brain starts with this sentence, "It is only recently that the high degree of plasticity in the brain could be recognized" (74). Even if most of the research in this area is not yet at the point of being applied, many of the findings and concepts give hope of applications to such problems as those of enhancing intellectual ability and preventing some forms of mental retardation, preventing or alleviating senile decline of mental functioning, and aiding recovery of function after brain injury. Before going ahead to examine evidence and speculations about neural plasticity, let us first take up the plan and scope of this chapter.

Plan and Scope of this Chapter

At the request of the editors, this chapter is intended to be partly speculative in character. We have interpreted this charge to mean that we should

both review research in this area and also attempt to make some extrapolations from it. Some extrapolations will take the form of suggestions for further research, and others will predict possible applications of current and future research. We will try to label the speculations clearly to aid readers in separating fact from fancy, although such distinction is not always easy even in the case of research literature, as will be seen in some areas of controversy and dispute upon which we will touch. Some speculative material will be sprinkled throughout the chapter; other such material will be found in the final section. A catalog of plastic changes in the nervous system will be presented on pages 171–178.

We should note that most of the material in this chapter comes from research with animal subjects rather than observations on human beings. Most investigators of neural mechanisms take the use of animal models largely for granted. Much justification, both practical and theoretical, can be supplied for extrapolating from animals to human beings where brain-behavior relations are concerned, but it is well to remember that not all such extrapolations are found to work out. Rather than discuss these problems here, we will simply cite two current references (76, 112).

Some of the main topics and questions to be considered in the chapter are these: To what extent are neural connections predetermined by heredity, and to what extent can they be altered by postnatal determinants? We will consider effects of several factors, including damage to other parts of the system, sensory deprivation or distorted sensory input, and training and experience.

Can new connections be formed in the adult mammalian nervous system? Such connections have been reported when tissue from other organs is implanted into parts of the brain. Also, it has been shown in some brain regions that when one input tract to the region is removed, the endings from other inputs then sprout and occupy the vacated synaptic sites. Changes in neural connections have also been demonstrated when animals are given enriched experience or formal training.

Does recovery of function after brain damage involve the formation of new compensatory connections or only the learning of ways to use residual capacities to make up for the loss? (And if learning is involved, does this imply formation of new connections and/or modification of preexisting ones?)

Before starting our review of recent evidence and current speculations, let us look back briefly to gain some historical perspective.

Changing Viewpoints

Views concerning the plasticity of the nervous system have changed widely during this century. Up through the 1920s and 1930s the prevailing

assumption among psychologists and physicians was that the behavioral repertoire of the organism is largely a product of accumulated training or conditioning. This was partly based on the research of Pavlov and on the teaching of Watson that almost any form of behavior could be taught by appropriate techniques. It also appeared consistent with the inability of Franz, Lashley, and Goldstein to find any specific locus for memories in the brain in studies involving experimental lesions in animals or brain-injured human beings. Even localized cerebral representation of sensory-perceptual functions was questioned. Lashley suggested that if the surgery were feasible, the striate cortex could be circumscribed and undercut, lifted out, rotated 180° and then reconnected—probably without seriously disrupting visual perception. Experimental reports stated that after motor nerve connections to the muscles in a limb were reversed, normal coordinated behavior reappeared, presumably mediated by adjustments of central connections.

A strong challenge to the proponents of functional plasticity was presented by Roger Sperry in the 1940s. When he conducted experiments involving surgical transplantation of nerves to muscles, he found that the predicted readjustments did not occur. Sperry also conducted extensive experiments on regrowth of the visual or retino-tectal system of amphibians and fishes and found great specificity and rigidity of connections. For example, if the eye of such a subject was removed and reimplanted after a rotation of 180°, the connections of retina to tectum were reestablished precisely and the animal showed inverted vision; furthermore, this inversion was found to persist indefinitely without any correction by reeducation. Behavior reverted promptly to normal, however, upon surgical rerotation of the eyeball back to its original orientation. On the basis of such results, Sperry concluded that fiber pathways and connections in the nervous system are precisely ordered and established without regard to function; he suggested that the guidance of the precise connections should largely be ascribed to the operation of highly selective cytochemical affinity.

In the 1960s and 1970s, much evidence has become available that new connections can be formed in the adult nervous system. Some of this came from the work of our group at Berkeley showing that differential experience leads to anatomical and chemical alterations in the brain, including changes in numbers of dendritic spines (7, 8, 41, 80, 81, 82). Greenough (43) found that differential experience causes changes in dendritic branching. Schneider (90) showed sprouting of some connections of nerve cells when other connections of these cells are removed. Extensive sprouting was also found to take over empty sites when one neural tract to a structure is removed (64). When tissue from any of a number of organs is implanted into the brain, it attracts innervation (67). Hubel and Wiesel and their collaborators have shown in a rich program of experimentation that visual depri-

vation or distorted visual inputs early in the life of cats or monkeys leads to changes in visual receptive fields and also to measurable changes in the anatomy of the cortical receptive areas. Training or differential experience has been demonstrated in a number of laboratories to aid recovery of function after brain lesions in experimental animals (44, 91, 118). Several of these indications of neural plasticity will be taken up in some detail later in this chapter.

The rapid survey of indications of neural plasticity in the last paragraph indicates both the wealth of research in this area and the growing recognition of possibilities of plasticity even in the adult mammalian nervous system. Rather than considering this as a swing of the pendulum back to an earlier position, we would prefer the analogy of a spiral ascending pathway on which we have now reached a higher outlook whence we can see more clearly both the opportunities for plastic change and the limitations imposed by characteristics of the neural system.

PLASTICITY IN CENTRAL CONNECTIONS

Study of Retino-Tectal Projections

Although some of the most convincing demonstrations of predetermined specificity in connections of the nervous system were afforded by research in the visual system (5, 95, 97, 98), further research on retino-tectal connections has given indications of some plasticity in this system, and research with mammalian subjects has shown much evidence of both anatomical and electrophysiological plasticity.

Using amphibian embryos, experimenters found that when they made a compound eye by joining either two nasal half-retinas or two temporal halves, the fibers from the compound eye seemed to project not just to the corresponding half of the tectum but to the whole tectum, each half spreading its representation across the entire tectum in a mirror image pattern (37, 101). It was also reported that in goldfish when the posterior half of the tectum is removed, the whole retina will in time come to project in an apparently orderly but compressed pattern on the remaining half-tectum (38, 120, 121). Meyer and Sperry (66) have reviewed much of this work and have raised questions about accuracy and interpretation of results apparently indicating plasticity of projection. Further research along this line, however, appears to yield quite convincing evidence of plasticity. Thus Udin (106, 107) studied retino-tectal mapping in frogs after regeneration of fibers when the operations were performed in adults; the tests after regeneration included both recording of action potentials and behavioral tests of

accuracy of orientation to visual targets. The caudal half of the tectum was removed and in some experiments the optic nerves were left intact, whereas in other experiments one nerve was also transected. When the caudal half of the tectum was removed on one side of the brain but the optic nerves were left intact, the terminals displaced from ablated tissue formed a permanently disorganized projection superimposed on the unaltered representation of those parts of the retina that normally projected to the rostral half of the tectum. The new connections appeared in the electrophysiological records about seven weeks after operation; they could be discriminated from old connections because the action potentials of new connections were only about half as large as the old and the new occurred singly or in isolation while old connections were in multiunit clusters. The behavioral tests of turning toward a visual target showed lack of accuracy in the part of the visual field that projected to the ablated half of the tectum. When the caudal half of one tectum was removed and the optic nerve was also transected, then the whole visual field obtained a compressed representation on the remaining rostral half of the tectum. Electrophysiological mapping showed that all parts of the visual field were represented. The behavioral tests also showed representation of the whole field but with less accuracy in the affected side than in the control side.

Schneider and collaborators have carried out an extensive program of research on determinants of visual connections in the hamster and have found examples of sparing of function in spite of removal of tissue, loss of function, and maladaptive alterations of function after damage inflicted early in life (90). If the superficial layers of the superior colliculus are destroyed unilaterally at birth, axons from the eye contralateral to the lesion not only grow out to the area of early damage but, in most cases, they also form an abnormal decussation, crossing the midline of the tectum to form terminations in the undamaged colliculus. This projection to the "wrong" side of the midbrain increases greatly if the undamaged colliculus is deprived of its normal innervation by early removal of its contralateral eye. Hamsters with such anomalous connections show a behavioral deficit—they turn to the wrong direction in response to stimulation in a large area of the upper and temporal visual field. Some of the factors that control the formation of abnormal connections have been specified, based on a program of neuroanatomical experiments: 1) Growing or regenerating axons tend to invade vacated terminal spaces and to compete with other axons for occupancy. 2) Axons tend to conserve at least a minimum quantity of terminal arborization. For example, fibers of the accessory optic tract form connections part way along their route at the dorsal terminal nucleus and also form terminal connections at the optic tectum. If the tectum is destroyed, these fibers then show hypertrophy of their connections at the dorsal termi-

nal nucleus. 3) The retino-tectal projections tend to preserve their topographic order even if they are forced to terminate in an abnormally small area such as the residual caudal part of the superior colliculus after ablation of the superficial layers of the rostral tectum at birth.

Recently So and Schneider (93) have been studying how much of the abnormal ipsilateral sprouting occurs when the superior colliculus is removed at birth and one eye is removed at various later ages. Up to about Day 12, the abnormal connections spread all the way across the superior colliculus, but if removal is delayed until Day 14, only a small amount of spread is observed. Several hypotheses that might account for the limitation of spread by Day 14 have been tested. When the visual cortex is ablated, thus removing some of the projects to the superior colliculus, this encourages growth of the retinal projections to the colliculus. It is now possible to obtain ipsilateral connections up to Day 16, and further prolongation of the growth period will undoubtedly be possible. Such studies of factors that limit and factors that promote formation of connections in the brain may eventually be of therapeutic significance. Some cases of recovery of function after destruction of neural tracts appear to involve establishment of new pathways or strengthening of existing ones. It may be possible in the future to promote selectively the formation of new connections linking chosen brain regions.

Effects of Visual Deprivation or Distorted Visual Input on Cortical Connections and Responses

A vast amount of research beginning in the 1960s has shown changes in cortical visual receptive fields and in cortical connections when one eye of a cat or monkey is deprived of visual input during early development or when such animals are exposed to restricted and distorted visual inputs such as being exposed only to horizontal lines or only to vertical lines or small spots of light. No attempt will be made to review this extensive research in any detail here; the interested reader is referred to the reviews by Grobstein and Chow (46), Chow (14), and Lund (62, Chapter 15).

Monocular deprivation of vision was shown by Wiesel and Hubel (114) to reduce severely the number of cortical visual cells responding later to the previously occluded eye. Whereas in normal kittens most cortical units respond to either eye, in kittens in which one eye had been occluded for several weeks during early development, the cells responded almost entirely to the eye with visual experience. Later research showed a clear anatomical correlate of this effect. In Layer 4 of the primary visual cortex, connections from the lateral geniculate body are monocular, the two eyes being represented by alternate bands of connections. At other layers of the cortex, the projections from the two eyes converge so that most units represent both

eyes. When one eye has been deprived of light during early development, its bands of representation in Layer 4 are much reduced in width, and the bands representing the eye with normal experience are correspondingly broader (50). Research with the radioactive deoxyglucose method has confirmed that ocular dominance columns are present in monkey striate cortex on the day of birth, that monocular deprivation begun early in life leads to narrowing of the columns representing the deprived eye, and that such plasticity has ended by the preadolescent period (19). It now appears, however, that the observed reduction in connections from the deprived eye is not sufficient to account for the almost total failure of this eye to drive cortical cells. There remain connections, but they are inhibited by those from the stimulated eye, as we will discuss below.

A large number of reports have claimed that if a young kitten is exposed only to vertical or to horizontal stripes or to some other sort of restricted visual stimulation, then receptive fields tend to respond to the stimulation experienced and in general not to other types of stimulation (10, 49, 70). Recently it has been claimed that these results are not completely replicable (102), and further work is needed in this area. It has also been claimed that structural changes can be found in response to restricted experience. Preliminary reports by Spencer and Coleman (94) and Flood and Coleman (32) have indicated that in kittens exposed only to vertical stripes, the dendritic fields of cells in the primary visual cortex were elongated along the anteroposterior axis of the cortex; in kittens exposed to horizontal stripes, the dendritic fields were elongated along the dorsoventral axis. In the kittens with normal visual experience, the dendritic fields tended to be round, while dark-reared kittens showed smaller dendritic fields than did normal control animals.

A number of investigators have reported effects of differential early visual experience on later visual behavior. Blakemore and Cooper (10) reported dramatic effects—kittens that had been exposed only to horizontal stimuli could scarcely see vertical stimuli; they would blunder into the legs of tables and chairs in the laboratory. Hirsch (48) found less striking results. He obtained small but significant differences in thresholds for horizontal and vertical lines. Eyes exposed only to horizontal or only to vertical stimuli were inferior even in the orientation of exposure, to eyes with normal visual exposure. Such effects were reported to be long lasting, remaining months or even years after the animals had been given normal visual experience. Chow and Stewart (15) showed that the effects of restricted early stimulation, although severe, could be overcome to a large extent by long and patient training.

People with astigmatism have suffered from reduced visual input along certain visual meridians in which their vision is blurred. Since astigmatism tends to be present from very early on, such people have been involuntary

subjects in a natural experiment on differential stimulation of visual meridians. Freeman and Thibos (33) found that astigmatic subjects showed a small deficit in visual acuity along the orientation of their astigmatism and that this deficit could not be completely overcome by optimal visual correction. Their only subjects not showing such a deficit were two individuals who had been given visual correction before three years of age. Thus, human beings may incur a persisting visual deficit caused by distorted sensory input, and it may be impossible to correct this completely unless remedial efforts are undertaken early in the life of the person. Just as hard-of-hearing youngsters are being given hearing aids in their first few years to promote normal development of speech, so it may be important to give children visual corrections earlier than is now common in order to prevent permanent establishment of abnormal central visual connections.

There has been considerable speculation as to whether the developmental plasticity of the visual system has some functional value. Perhaps this simply reflects the fact that the visual system, like other parts of the nervous system, is not completely specified genetically and that evolution has occurred under circumstances that normally entail a contribution of information from the environment. Others have suggested that at least some aspects of the plasticity of the visual system may be present because genetic information may be intrinsically inadequate to create optimal connections (e.g., 46). This may be especially true in the case of acuity for binocular disparity. The adult viewer is highly sensitive to small binocular disparities, and these provide clues to depth and distance in the visual field. But in neonatal kittens, the precision of binocular representation is poor (69). In the young kitten a 3° field in one eye summates with a point stimulus in the other eye. Over a six-week period, this reduces to 0.5° in kittens that receive normal visual experience, but such a reduction does not occur unless the two eyes receive experience at the same time. Shlaer (92) raised kittens with a prism over one eye, causing a vertical displacement of a few degrees. After some weeks of this experience, the cortical representations of the visual fields of both eyes were measured without the prisms present. In prism-reared kittens, cortical cells tended to receive input from noncorresponding points in the visual fields of the two eyes, such as to compensate for the displacement that had been caused by the prism over one eye. Apparently the visual systems had been able to compensate for small discrepancies during experience, although earlier experiments had shown that no compensation was possible for a large discrepancy, such as occurs in strabismus. Precise correspondence of registration of the two eyes depends on a number of factors including the exact optics of the eyes and their positions in the eye

sockets as determined by the extraocular muscles. The two eyes have somewhat independent histories of development, so only after the system begins to function can it be determined exactly what positions on the two retinas see the same region in visual space. Thus, within a development of visual connections that is broadly specified genetically, the effects of specific experience may be required for the fine tuning of the correspondence of connections.

Plasticity in the Hippocampus

Recent research on the hippocampus shows that when one set of its input fibers is eliminated, fibers from other sources rapidly sprout new endings to occupy the vacated synaptic sites. This search for plasticity in the hippocampus was begun with young animals, presumably on the widespread premise that plasticity may be confined to early development. But it was then extended to adult animals, and evidence was found of continuing "competition" for connections even in adult brains. Let us review this research briefly.

The hippocampus is a three-layered structure of paleocortex. The relative simplicity and regularity of its structure makes it especially suitable for analysis of structure and function. In the dentate gyrus of the hippocampus, the middle layer is made up of granule cell bodies whose dendrites project up into the molecular layer where they receive projections from a variety of sources. In the inner one-quarter to one-third of the molecular layer, the part closest to the granular cell bodies, the dendrites receive afferents mainly from the contralateral hippocampus through the lateral ramus of the hippocampal commissure. The same region also receives projection from association fibers of the ipsilateral hippocampus but not as many as from the contralateral. In the outer one-half to two-thirds of the molecular layer, dendrites of the granule cells receive projections from the ipsilateral entorhinal cortex and a much less dense projection from the contralateral entorhinal cortex. Entorhinal cortex is a transitional form of cortex in the anterior part of the temporal lobe. If one of the normal afferent pathways is eliminated, then two types of reorganization may occur: (1) the spreading or sprouting of afferent fibers into areas of the dendritic field adjacent to the areas that they would normally occupy; and (2) the development of a thicker or denser afferent projection into parts of the dendritic field that these fibers would normally innervate only sparsely.

The experimental procedures used by Lynch, Stanfield, and Cotman (63)

consisted of several stages. In the experimental group, entorhinal cortex was removed unilaterally in 11-day-old rats. Time was then allowed for complete degeneration of projections from the entorhinal cortex and for the products of degeneration to be removed by phagocytosis. Then, when the rats were 91 days old, the commissural pathway to the hippocampus was transected. Five days later the rats were sacrificed to study the distribution of the newly degenerated projection from the commissure to the dentate gyrus. The pattern of degeneration was compared in the two hemispheres and also with control animals in whom entorhinal cortex had not been removed. On the side deafferented by the entorhinal lesion, the commissural fibers now spread through more than nine-tenths of the molecular layer. This broad distribution contrasted sharply with the pattern in the other hemisphere of the early-lesioned rats and with both hemispheres of the controls. In the latter, the picture was normal with the commissural projection restricted to a narrow zone just above the granule cell body. The results suggested that the normal narrow distribution is probably maintained by competition among inputs.

Lynch et al. (63) then compared effects when entorhinal cortex was removed at 11 days of age or in adults at least 100 days old. Following a recovery period of at least 150 days, the experimenters then removed dorsal hippocampus contralateral to the original entorhinal lesion. A few days thereafter the animals were sacrificed in order to determine the spread of connections from the dorsal hippocampus through the commissure to the contralateral hippocampus. The median percentage of the width of the molecular layer of the dentate gyrus occupied by the band of commissural degenerations was 52% in rats that sustained the entorhinal lesion at 11 days of age, 44% in the adult-lesioned rats, and 27% in the controls. Thus, the width of the projection increased by 90% after removal of the entorhinal projection in rat pups and by 60% after removal in adults.

Current work has extended this investigation to 24-month-old rats (89). Preliminary indications are that axonal sprouting to occupy vacated sites is slower to start in older rats than in young rats, but that, given enough time, the effect in the older animals may be substantial, although still not as great as that in the younger animals.

Changes in the density of synapses in the hippocampus in response to enriched vs. impoverished experience in postweaning rats have been reported by Altschuler (4).

EFFECTS OF DIFFERENTIAL EXPERIENCE AND TRAINING ON BRAIN MEASURES

Now let us consider how experience and training can affect the brain. The preceding sections have shown that plastic changes occur in response to

severe or harmful treatments—depriving young animals of normal sensory stimulation or transecting tracts in the brain. Such severe treatments are not necessary to induce cerebral effects. A large number of experiments, first in our laboratories at Berkeley and later in several other laboratories as well, have demonstrated that differential experience and training lead to measurable changes in the biochemistry and the anatomy of the brain. Furthermore, many of these effects occur not only in young animals but also in adults. Only a brief survey of these findings will be presented here, since several rather extensive recent reviews are available (43, 80, 81, 82).

Most of the experiments on this subject have involved placing laboratory rodents for periods lasting from a few weeks up to a few months in differential environments—environments either enriched or impoverished in comparison to the usual animal colony cages. The enriched environments have usually included both more social stimulation and a greater variety of inanimate objects than the colony cage; that is, 10 to 12 animals are placed in a large cage with a variety of stimulus objects or "toys." In some experiments, the social stimulation and the inanimate stimulation have been varied separately (80); social stimulation cannot account for the full enrichment effect (85). For impoverished experience, the subject is placed alone in a colony cage, instead of having one or two cagemates as in the standard colony situation.

Effects on Brain Weights

Typical results in terms of brain weights are these, when littermate rats are compared after exposure to the enriched condition (EC) versus the impoverished condition (IC): Rats with EC experience show greater weight of total brain by about 2-3%; this is statistically significant in most experiments. The overall brain difference is due mainly to changes in cerebral cortex which show effects of about 5% while the rest of the brain differs by only about 1-2%. A very stable measure that tends to yield highly significant differences between groups is the cortical/subcortical weight ratio. Because both cortical weight and subcortical weight are correlated with body weight, the ratio tends to eliminate the influence of body weight and to provide, in effect, a covariance on body weight. Within the cerebral cortex, the occipital area shows the largest difference between EC and IC, the occipital effect often amounting to 8% or more.

The results just described are found in experiments in which starting ages range from 25 days to over 200 days. (See Table 17.1 of Bennett, 7, for specific results with a variety of starting ages and experimental durations.) Thus these effects of differential experience can apparently be evoked at any part of the lifespan. In this respect they differ from the changes in the

visual system caused by deprivation or distorted stimulation, since these visual alterations can be produced only during a sensitive period early in development. As we will see, effects of differential experience are also found in brain-lesioned rats, and these effects may be important in recovery of function.

Interpretation of Brain Weight Effects in Terms of Cellular Changes

The occurrence of changes in weights of brain, and especially of cerebral cortex, as a function of exposure to different environments is interesting, but more refined analyses are required if we are to progress toward the level of understanding cellular functions. Fortunately a number of further observations have been made, although more types are still required. It has been found by Greenough and collaborators that neurons show greater branching of dendrites in EC rats than in their IC littermates (43). This also is a regional effect, occurring with greater magnitude in the occipital cortex than in the other cortical regions measured. Globus et al. (41) found that the number of dendritic spines per unit of length of dendrite was significantly greater in EC than in IC rats. This effect was localized even within neurons, the difference in spine density on basal dendrites amounting to 9.7% ($P < .01$), on oblique dendrites, 3.6% ($P < .05$), and no effect occurring on apical dendrites. Along with this increase in the dendritic tree, it was found that the cross section of the neuronal cell bodies was significantly larger in EC than in littermate IC rats (21). Presumably the cell body must be more active metabolically to support a larger dendritic aborization, and such an increase in biosynthetic function was supported by the finding of greater amounts of RNA in cortex of EC rats, as compared with IC littermates (7). Hydén and Rönnbäck (54) have recently reported that the incorporation of valine into brain protein of frontal, entorhinal, cerebellar, and visual cortex and the hypothalamus was much greater in rats raised in an enriched environment than in rats raised in a dark and restricted environment for 90 days beginning at 15 days of age. Enriched experience may also cause an increase in the number of glial cells to minister to the more active neurons; an increase in glial/neural counts has been reported in EC vs. IC rats (23, 103).

Putting this evidence together, we attribute the increase in cortical bulk with enriched experience mainly to the growth of neural ramifications. Presumably the interconnectedness of cortical neurons increases; this may reflect both greater redundancy (and thus greater effectiveness of some circuits) and also the establishment of novel circuits.

We should note that evidence of structural plasticity with differential experience is not limited to rats. It has also been found in laboratory mice and in gerbils (78) and in feral deermice. Unfortunately, little research of this sort has yet been done with subjects other than rodents. It would be highly desirable to extend this research to other orders of mammals, and especially to carnivores and primates. Not only would such work test the generality of the results obtained so far and indicate more clearly to what extent the conclusions might be extrapolated to human beings, but research with larger and more precisely mapped brains would permit the use of more powerful and refined techniques than are available for rodent subjects. A collaborative project to study effects of differential experience on brains of monkeys has been initiated by two psychologists, William T. Greenough of the University of Illinois and Gene P. Sackett of the University of Washington (45).

Regional Brain Changes Measured by Tomography?

Perhaps new radiological techniques, and particularly computerized axial tomography, can be applied to study changes in the conformation of specific brain regions during training. In computerized axial tomography, the head is exposed to X-rays at doses equivalent to those now used in routine diagnostic procedures, and the absorption information is fed into a computer which transforms the data into cross-section pictures of the brain and skull. As Galaburda et al. (35) have reviewed, structural asymmetries have been found between the two hemispheres in the human brain. The best-defined asymmetry in the gross configuration of the human cerebral cortex is that the planum temporale on the upper surface of the temporal lobe is significantly larger in the left hemisphere than in the right hemisphere of most subjects (39). Galuburda et al. present a computerized axial tomogram of a human brain showing asymmetry of the planum temporale (35, fig. 3, p. 855). It has been concluded that this asymmetry does not represent effects of experience, since asymmetry in the same direction is present in the newborn infant and can be observed as early as week 31 of gestation. It is not clear, however, that the magnitude of the difference between hemispheres cannot be affected by experience. In the case of the brain of the rat, the cortex differs in thickness among various regions, but the magnitude of these differences is affected by experience (22).

Hemispheric asymmetry is not limited to human beings; the great apes also show hemispheric asymmetry in the Sylvian fissure which marks the superior boundary of the temporal lobe. We would like to suggest that this powerful technique be applied to the chimpanzee and other apes that are

now receiving unprecedented amounts of training in communication in several laboratories (e.g., the laboratory of the Gardners at the University of Nevada and the laboratory of Fouts at the University of Oklahoma). Chimpanzees in these laboratories are receiving training in the use of American sign language, and training often extends through several hours a day over a period of several years. The subjects of these experiments could receive tomographic examinations at the outset of their training and at regular intervals during training. It might be possible in this way to study the course of changes at several points during the program of training, especially if comparison could also be made with the cerebral development of nontrained controls.

A few months after we wrote the paragraphs above (and also the section on preventing or alleviating mental retardation and learning disabilities) suggesting possible uses of tomography to study brain plasticity, a paper was published that made our proposals seem less farfetched than they originally might have appeared. Carlen et al. (12) found that computed tomography scans revealed cerebral atrophy in the brains of eight chronic alcoholics; the four who then abstained and showed functional improvement also showed partial reversal of the atrophy. The investigators suggested that the partial recovery may have been due to regrowth of axons and dendrites of neurons that were damaged but not killed by ethanol abuse. This study not only indicates the feasibility of such investigations, but it also provides new evidence of plasticity of human brain in gross anatomical measures.

Hypotheses to Account for Cerebral Effects of Differential Experience

We originally began to use the differential environments as a way of providing animals with differential opportunities to learn. This step was taken after we had found that giving rats formal training appeared to alter cortical acetylcholinesterase (AChE) activity (87). We hoped to test this effect of training more clearly and to enhance the magnitude of the results by providing round-the-clock opportunities for self-paced learning over a period of several weeks. When we obtained differences in cortical AChE following exposure to enriched or impoverished laboratory environments, we were inclined to attribute them to differential learning. Nevertheless, in our first publications we tested alternative hypotheses and showed that neither the greater handling of the EC rats nor their greater amount of locomotor activity could account for the observed effects (59, 87).

During the last 18 years, many alternative hypotheses have been proposed, and quite a few have been tested. There is no need to discuss most of

them at length here, because they have already been reviewed elsewhere (43, 81, 82). Let us simply note some of the frequently mentioned alternatives that research has allowed us to reject. Thereafter we will note some recent evidence that provides further support for the hypothesis that the cerebral changes are consequences of learning and memory storage.

Tests of Several Hypotheses

1. Stress is frequently offered as an explanation for the cerebral effects—either "isolation stress" in IC or the stress of information overload in EC. Neither IC rats nor EC rats show enlargement of the adrenal glands in comparison with littermate SC rats, so it is unlikely that these rather mild treatments involve appreciable amounts of stress. Furthermore, imposition of overt stress daily for 30 days on IC or EC rats did affect weights but did not alter EC vs. IC cerebral effects in measures of brain weights or AChE activity (73). Therefore, the cerebral effects of differential experience cannot be attributed to stress.

2. Hormonal mediation is probably not required for production of the cerebral effects. Not only do EC and IC rats not differ in adrenal weight/body weight or in thyroid weight/body weight, but hypophysectomy does not prevent the development of typical cerebral differences between rats in EC or IC (83).

3. Speeded maturation in an enriched environment has been suggested as a possible cause of cerebral differences between rats in EC or IC environments. This might seem able to account for some of the characteristics of EC rats such as greater weight of cerebral cortex and greater branching of dendrites. But on other measures, EC rats resemble young animals more than do IC rats. Here are two examples: The cortical/subcortical weight ratio declines with age, but it is higher in EC than in IC rats; and the RNA/DNA ratio declines after about 30 days in the rat, whereas EC causes an increase in this measure. Furthermore, we have shown that most of the EC-IC differences can be induced even when the differential treatment is initiated in adult animals, so speeded maturation is clearly not the cause of these effects.

4. Deprivation or distortion of sensory input has been demonstrated to alter aspects of perceptual functions, receptive fields of cortical neurons, and even cortical anatomy. We believe, however, that this does not provide a general model for neural events in learning or for effects of differential experience. Three criteria enable us to distinguish the effects of sensory deprivation or distortion from those of differential experience of the EC-IC sort: The effects of differential experience occur even in adults, they require di-

rect interaction with stimuli, and severe deprivation or distortion of stimulation is not required in the IC group. In contrast, the sensory effects occur only during a limited period of development, they have been reported to be produced by simple passive exposure to stimuli, and severe departures from normal stimulation are required to alter sensory development.

Effects of Self-Paced Maze Training on Cerebral Measures

In order to test directly whether specific training leads to anatomical and biochemical changes in the brain, we have recently conducted a series of experiments in which individual rats ran self-paced trials in mazes (9). The maze consisted of a plastic box inserted as a floor or story inside the larger cages that are used for the enriched condition; the box is as broad and as deep as the large cage and is 10 cm high. Food was available on the ground floor of the cage and water was available above the top of the plastic box. In order to get from food to water, the rat had to climb up into the plastic box (by a door open in one corner), traverse the box, and exit above by a door in another corner. Each rat in this condition was moved from one cage to another each day, and it found a new pattern of maze barriers in the plastic box every day over a 30-day period. We called this condition "Individual in Complex Maze" (I-CM). Each I-CM rat had a littermate assigned to a standard EC group and another in the IC environment.

Results showed that the I-CM rats differed significantly from their IC littermates in brain weights and in brain RNA (N = 70 per condition). These I-CM vs. IC effects were about half as large as the EC-IC differences obtained in the same experiments. The last several experiments (N = 26 per condition) included a stringent control condition to test whether the cerebral effects observed with I-CM might be due to the exercise of climbing into and out of the plastic box, the exposure to a series of large cages, etc. The control condition was exactly like I-CM except that the plastic box contained no barriers; this condition was therefore called "Individual in Empty Box" (I-EB). In contrast to the effectiveness of the I-CM treatment, I-EB was almost completely ineffective in altering brain values. Thus, these experiments demonstrate more clearly than heretofore that training causes significant modifications in brain measures.

RECOVERY OF FUNCTION

How may various kinds of neural plasticity contribute to recovery of function after damage to the nervous system? This question is too large for

review in this chapter, so only a few points about the subject will be made here. For a good deal of information and varied discussion of this subject, see the following books: *Plasticity and Recovery of Function in the Central Nervous System* (100), and *Recovery from Brain Damage* (30).

Sperry (96, 99) has denied that adaptive changes occur in the nervous system following injury. He has stressed that in many cases the impairment caused by brain damage persists. Where behavioral improvement occurs, he has attributed this to the animal subject or the human patient using "tricks." That is, the individual learns to substitute some other movement or function to replace the still impaired one. It is somewhat surprising to see a contrast being made between behavioral and physiological mechanisms of recovery, as if all behavior did not have a physiological basis. If learning is used to overcome a behavioral deficit, does not learning have a physiological basis, and is not evidence accumulating that learning involves plastic changes in synaptic connections?

Recovery through Reconnection

In many cases recovery does occur through restoration of the original function and not through a "trick" or substitution of other behavior. For example, many cases of aphasia show excellent recovery although extensive destruction of neurons has occurred. Polio patients have recovered manual dexterity even though postmortem examinations revealed destruction of many ventral horn cells. Animal subjects have demonstrated recovery of normally coordinated locomotion after various types of experimental lesions, including damage to motor cortex, spinal cord sensory or motor tracts, and cutting of several dorsal spinal roots.

The neural mechanisms of most of these examples of recovery of function are not yet known, but there is considerable evidence that recovery after experimental transection of some dorsal spinal roots is related to axonal sprouting from remaining roots. An early observation along this line was that of Liu and Chambers (61). They cut dorsal roots (e.g., T11-L6) unilaterally. After recovery of hind limb coordination, they examined the spinal cord anatomically to determine the spread of terminals from axons entering through an adjacent root (e.g., L7), both on the experimental side and on the normal control side. Much more extensive branching and spread of terminals was found on the affected side. Recent work relating such collateral sprouting to recovery of coordination has been done by Goldberger (42). Another mechanism that may aid such recovery is the release from inhibition of quiescent neurons or parts of neurons; evidence of such release will be presented and discussed below.

The mechanisms stated in the last paragraph may be of considerable importance in making possible recovery of function when most but not all of the fibers of a tract have been destroyed. Here are two examples of recovery that may be based on these mechanisms: Bach-y-Rita (6) has reported the excellent recovery over a period of about three years of a man who suffered a stroke at the age of 65, resulting in severe right-sided hemiplegia and loss of ability to speak or write. A postmortem examination seven years after the stroke revealed that extensive damage had occurred in the lower left pons and medulla, destroying most of the corticospinal tract. "A few fibers remained intact and these may have formed the basis of the functional reorganization." The other example is from a unpublished case of Rasmussen, cited by Bach-y-Rita (6, pp. 211-212). This is the case of a man who became paraplegic following an automobile accident, but who gradually regained complete function and was able to enlist in the U.S. Navy. He served three enlistment periods with no physical limitations and then died in a second automobile accident. Autopsy revealed that an apparently complete separation of the spinal cord at the level of T7 had resulted from the first accident; the gap was approximately 1 cm long. However, microscopic study revealed approximately 150 axon cylinders embedded in the fibrous tissue separating the two portions of the spinal cord. Thus, it is likely that recovery was obtained by the functional reorganization of the input to the cell bodies of the 150 remaining fibers, as well as the possible redirection of the axon terminals.

Experience and Training Aid Recovery of Function

A growing volume of research with animal subjects indicates that varied experience and/or specific training can reduce the impairment of function caused by brain injury. In different experimental designs, the experience may be provided before the brain damage is inflicted, or it may be given between two stages of surgery in a serial-lesion study, or it may follow the lesion. Research in this area has been reviewed by Greenough, Fass, and DeVoogd (44) and by Finger (31).

Since providing enriched experience following the lesion may offer a model for behavioral therapy, and since such experience has been shown to produce some changes in brain measures, we will review this research briefly here. Schwartz (91) removed tissue from occipital cortex of neonatal rats and then raised them either in groups of 2 to 6 in a standard colony (SC) environment or in enriched-experience cages (EC) where they lived in groups of 4 to 8 and had a variety of stimulus objects. At about 130 days of age, the rats were pretrained and then tested on the standard series of 12

mazes of the Hebb-Williams test. Both brain status (lesioned vs. intact) and environment (SC vs. EC) affected the scores significantly. Lesioned-EC rats performed as well as intact-SC rats; best of all were intact-EC, and worst were lesioned-SC. Thus, postlesion enriched experience helped overcome effects of the brain lesion. Experiments in our laboratory have since replicated and extended this finding. We found beneficial effects of enriched experience not only after neonatal lesions (117), but also when the damage was inflicted after weaning (118), and even after 100 days of age (116). Furthermore, the subjects included both male and female groups, and rats of two strains different from those of Schwartz were employed. Shorter periods of enrichment than those in Schwartz's experiment were found to yield positive effects; in one experiment, 2 hr/day of EC over a 60-day period was found to be as effective as 24 hr/day. Among the brain-injured as among the intact rats, EC led to an increase in cortical weight and in cortical RNA/DNA. Thus, it is clear that even an impaired brain responds to experience, as shown by both behavioral and cerebral measures.

A CATALOG OF PLASTIC CHANGES IN THE NERVOUS SYSTEM

In the earlier sections of this chapter, we have mentioned many ways in which neurons can change in response to demands and modifications of their environments. Here we will compile a partial catalog or roster of plastic capabilities in order to present them in one place, adding some findings not mentioned earlier. Although we will focus on changes that are known to occur during adulthood in mammals, we will start with changes that are characteristic of early development, and we will also mention some kinds of plasticity that are best known in invertebrates. Changes in developing animals or in invertebrates will be mentioned not simply for completeness but also because some processes that were once thought to be restricted to those forms have later been found to occur in adult mammals as well, and more similarities and generalities of plastic modifications among diverse animal forms will undoubtedly be discovered in the future. Although most points in this catalog are well substantiated by research, a few are speculative.

Changes Characteristic of Early Development

Early in the development of the nervous system, the cells divide, and young daughter cells often appear to remain uncommitted for a period between becoming neuroglia or neurons; neuroblasts may be able to differentiate into any of several specific types of neuron. It is sometimes said that

neurons do not divide after birth in the mammal, but in the rat most of the cerebellar neurons are produced after birth and so are some forebrain neurons. After new neurons are formed, many of them migrate considerable distances to their final sites, often passing through layers of already established cells. Only after having reached their final locations do the neurons begin to assume their characteristic and varied shapes, sending out an axon, pushing out branches and branchlets of the dendritic tree, and in some cases growing dendritic spines. Even in neurons that take up their stations prenatally, the processes of assuming the adult form often require months of postnatal growth. During this period, the growth and formation of connections of neurons in many locations can be influenced by stimulation and activation and by competition with neighboring units, as we saw in the case of development of sensory systems. For a review of migration and differentiation of cortical neurons, see Rakic (71); concerning migration and differentiation in the cerebellum, see Altman (2).

Changes Occurring in the Adult Nervous System

Now, moving on the adult organism, let us consider what kinds of plastic changes may occur in its nervous system that could subserve learning, memory, and progressive changes in behavior after disease or injury to the nervous system (i.e., both recovery of function and progressive deterioration). We will take these up in an order going from least to most: a) Functional changes that involve no changes or only minor anatomical alterations in existing units. b) Anatomical changes in parts of existing neurons—axon terminals, dendritic branching, and dendritic spines. c) The possibility of migration and specification of immature neurons and even production of new neurons in the adult brain. d) Finally, observed changes in glial cells as a function of experience should not be ignored, even though none of the hypothesized roles of glia in learning and memory has yet been established.

Plasticity Requiring Little or No Anatomical Change

Changes at Existing Synapses

Functional changes at existing synapses without any anatomical modification of synapse number or location would be sufficient to subserve many instances of learning; indeed, functional changes must suffice in the case of short-term memory where the memory can be retrieved within seconds af-

ter learning occurs and there is no time to accomplish anatomical changes. This is more or less like altering a circuit by turning switches on or off or by changing the setting of a variable resistor without altering the existing hardware. Changes in the functional properties of existing synapses during habituation to repeated stimuli have been studied in a number of preparations. In some cases the habituation persists well beyond short-term memory, lasting for hours or even days and suggesting that some long-term memories may be held in terms of chemical changes at existing synapses. For a review of research on functional changes in synapses of the much-studied invertebrate, *Aplysia*, see Kandel (57). For research on functional changes at synapses of the hippocampus of the rat during habituation, see Lynch and Wells (64) and Teyler and Alger (105).

A rather simple anatomical change at existing synapses has been suggested as a correlate of posttetanic potentiation and possibly of other functional changes. This is the observation of swelling of dendritic spines in the fascia dentata of the hippocampus after tetanic stimulation of the afferent perforant fibers (108).

Changes Between Active and Quiescent States of Neurons or Parts of Neurons

Somewhat akin to the notion of altering neural circuits by modulating the properties of existing synapses is the possibility that entire neurons or parts of neurons may be made either quiescent or active, depending on circumstances (111). Cass et al. (13) showed that two or three nerves run to the muscles in a salamander limb, each nerve occupying its own territory. These territories are not absolutely predetermined, since cutting one nerve leads to expansion of the territories of other nerves. Furthermore, the timing of this expansion was highly revealing. Two or three days after the nerve was cut, some muscle innervation appeared as a marginal extension of the territories of adjacent nerve roots. A week or two later, the adjacent innervation spread considerably further into the denervated zone. Thirty days after the section of the nerve, it grew back into its original area, and the adjacent nerves were no longer able to command these muscle fibers. Then the nerve was cut for a second time and the muscle fibers were again paralyzed, not responding to stimulation of any nerve. Three days later, however, the whole of this denervated area responded to the adjacent nerves. The investigators suggested that the rapid spread of innervation that occurred three days after cutting a nerve, either the small spread the first time or the extensive spread the subsequent time, resulted from the emergence into function of preexisting but quiescent nerve terminals.

Evidence of quiescent terminals in the spinal cord of the cat was obtained by Wall (111). He mapped the receptive fields of cells in dorsal column nuclei and selected some single cells that responded only to stimulation of the foot. Then the cord was blocked by cold in the lumbar 4 segment. Most of the cells being monitored simply lost their receptive field to peripheral stimuli but continued to show some ongoing activity. A few of the cells, however, now showed a receptive field to stimulation on the abdomen which had not been effective before. On removal of the cold block, these cells responded again to the foot but not to the abdomen. Thus, some cells have alternative inputs—the afferents that operate under normal conditions and an alternative group that can become effective as soon as the normal input is silenced or removed.

Research in the visual system has also provided results that have been interpreted in terms of inhibition of connections from a visually deprived eye by those of the normally stimulated eye. In a young cat or monkey, occluding one eye for a period of a few weeks reduced substantially the percentage of striate cortical cells that responded to stimulation of that eye. Thus, in kittens with lids of one eye sutured closed for their first 4 to 5 months, testing showed that only 0–10% of the cortical cells could be driven by stimulation of the deprived eye (58). Although there is probably some anatomical loss of connections from the deprived eye, it is probably not as great as this functional evidence might suggest. Kratz et al. (58) found that if the normally stimulated eye is removed while the anesthetized kitten is in the testing situation, the deprived eye is then able to drive 30–40% of the striate cortical units. This increase is present during the first 12 hours after enucleation; it was not possible to state exactly how promptly the recovery occurs, because it required 20 to 30 minutes for the anesthesia to wear off, and finding units is a rather slow affair. There was no difference between the percentages found in the first 12 hours or thereafter, nor was the percentage increased if the recording was done only 3 or 12 months after enucleation.

Independent evidence that some connections from the deprived eye are functionally inhibited rather than being eliminated was obtained by a quite different technique in a study conducted by Duffy et al. (26). Kittens in this experiment were monocularly deprived of vision by eyelid suture in their fourth week, and 8 months later single-unit recordings were made in primary visual cortex. Of 33 cells evaluated in 5 kittens, all responded to stimulation of the normal eye, but only one could be driven by stimulation of the amblyopic eye. Since several lines of evidence suggest that inhibition in the visual system of the cat is mediated by gamma-aminobutyric acid (GABA), the experimenters then tried to restore central responses to the deprived eye by administering a drug that blocks GABA-receptors. Within 30

seconds, 17 of the cells became responsive to the amblyopic eye and remained so for several minutes; a few more showed a delayed onset of responsiveness. During the brief duration of responsiveness, it appeared that the receptive fields for the amblyopic eye were closely similar to those found for the normal eye. These findings provide strong evidence that many connections from the deprived eye remain present and capable of normal function but are inhibited by connections from the stimulated eye.

Changes in Axon Terminals, Dendrites, and Synapses

Existing neurons in the adult mammal may form new synaptic connections under a number of circumstances, several of which have been mentioned in earlier sections. Many aspects of neurons have been demonstrated to undergo changes in the adult; these include axon terminals, dendritic branches and dendritic spines, and synaptic receptor areas. Let us note some examples of each of these kinds of changes.

If one input to a region has its flux of messages sharply reduced or if the input tract is transected, then terminals of other inputs to that region may sprout and form new functional endings. We saw examples in the case of closure of one eye during the critical period in development and in the case of removal of one input tract to the hippocampus. It may not be necessary for one set of endings to degenerate in order for sprouting of a competing set to be induced. Cajal (11) noted that nerves often grow relatively long distances to reach their target organs but do not start to sprout collateral branches until they arrive at the target location. Furthermore, the sprouting results in rather uniform innervation, without either large open spaces or areas of heavy concentration. To account for this, Cajal hypothesized that the target tissue secretes substances that promote sprouting and that the collaterals release factors that neutralize these influences. Support for these hypotheses comes from the following recent finding: Applying colchicine to one hindlimb nerve of a salamander induced sprouting into its field by the adjacent nerves, even though the colchicine did not impair conduction of impulses but only interrupted axoplasmic transport (1, 20). Presumably the colchicine prevented transport and release of factors that neutralize promotion of sprouting, and the terminals of neighboring nerves therefore responded to the signals to sprout.

A number of different kinds of axonal growth may occur, including the following: a) An existing terminal may form a second nearby synaptic contact with the same dendritic spines. b) An existing axon may form additional end boutons that make contact with other dendritic spines—so-called terminal proliferation. c) An axon may form new branches that travel for

some distance before making contact with new target cells—collateral sprouting. Lynch and Wells (64) concluded that the capacity of axons for such growth, unlike that of dendrites, becomes reduced with the onset of maturity. If this is true, then what would supply the input for the dendritic branches and dendritic spines that have been demonstrated to form in mature nervous systems? On the contrary, Lasek and Black (60) have concluded that axon terminals may always be ready to grow. They analyzed the axonal flow and turnover of axonal cytoskeletal proteins in guinea pig phrenic nerve and concluded as follows: "Our analyses of axonal transport suggest that the axon is continually supplied with the full complement of proteins necessary either for growth or for the formation of synaptic endings. The decision—to grow or not to grow—appears to depend upon cues in the axon's immediate environment and their effect on proteins which are always present in the axon" (p. 161).

Dendrites and dendritic spines have been demonstrated to respond to the stimulation of enriched experience and/or training. Thus, when rats receive postweaning exposure to enriched versus impoverished environments, neurons in the occipital cortex show increased numbers of higher-order branches of dendrites (43) and increased numbers of spines per unit of length of dendrite, especially on basal dendrites (41). In fish, isolation rearing was found to decrease the number of dendritic spines and to alter the proportion of the spine head to the shaft in the optic tectum (16). There is reason to believe that enriched experience produces these neural effects at least in part because of the increased opportunity for learning that it affords (9; 43, pp. 267-268; 82). Furthermore, some of these changes have been observed as a result of formal training. Thus Greenough (43, p. 270) has found that maze training produces similar increases in dendritic branching to those found with enriched experience. We have found that maze training produces changes in cortical weight and RNA/DNA similar to the changes caused by enriched experience (9). Rutledge (88) has reported on cats that were conditioned to associate shock to the foreleg (UCS) with electrical stimulation of the suprasylvian gyrus (CS). Stimulation of the cortex alone led to some changes in the form of terminal branches of apical dendrites and in their spine counts, and conditioning led to further changes. Rutledge has described several kinds of changes in apical dendrites and spines in these experiments (88, fig. 22.6): a) Increased length of terminal portions of apical dendrites and also new spines appearing on new surfaces of vertical dendrites; b) new small spines on new, thin, terminal twigs; c) secondary spines that form near established spines; d) new spines clearly separated from older ones; and e) increased area of synaptic contact on established spines.

Increases in the length of postsynaptic thickenings (presumably the receptor area) have been shown in synapses in the occipital cortex in enriched-environment rats vs. impoverished-environment littermates (24, 113). Enriched experience also led to increased density of synaptic contacts in the hippocampus (4).

Possibility of Differentiation and Even of Formation of New Neurons in the Adult

In some species of fish, neurons proliferate even in the adult, but this is generally thought not to be possible in adult mammals. Yet there have been some recent suggestions that even in the adult mammalian nervous system, there may be a low level of production of new neurons and/or reservoirs of storage of undifferentiated neurons. Thus, Altman and Das (3) hypothesized production of new neurons to account for increased length of the cerebral hemispheres of the rat as a consequence of enriched postweaning experience. They suggested that these neurons were formed in the ependymal layer lining the lateral ventricles. Wall (111) has speculated that some small cells in many regions of the central nervous system (e.g., in the substantia gelatinosa) "may form a pool of undifferentiated nerve cells, arrested in their progress from the germinal epithelium in the embryo and free to continue their voyage if conditions permit in the adult" (p. 361). Whether such production of new neurons or differentiation of neurons actually occurs, and if so which conditions induce these events, remains for further research to determine.

Plasticity of Glial Cells

Although it is not clear that neurons can proliferate, differentiate, and migrate in the adult mammalian brain, there is no doubt that glial cells do so readily. Abnormal glial proliferation can result in brain tumors—gliomas. The finding that brain cholinesterase (ChE) activity increased even more than did acetylcholinesterase (AChE) with enriched experience (8) led us to make counts of glia and of neurons in cortical tissue, since cholinesterase is found in glia but not in neurons. The cell counts showed a significant increase in the number of glia per unit of cortical volume in enriched-experience vs. imporverished-experience littermates (23), and this has since been confirmed by other investigators (103).

May these changes in glia be related in any direct way to mechanisms of learning and memory? Although there has long been speculation along

these lines (36, 53), no relation has yet been demonstrated. We have tended to suppose that the glial changes are consequent upon neural changes, since significant differences in ChE activity appeared only after about 8 weeks of differential experience, whereas differences in AChE activity were clearly present in 30 days or less (84). Perhaps larger neurons with more dendritic ramifications require more glial cells to minister to them. On the other hand, Lynch and Wells (64) showed that glial proliferation (on days 1-4 postlesion) preceded neural sprouting (beginning on day 5 or 6) when one input to the hippocampus was removed. They suggested that the glial reaction may actually stimulate the axonal sprouting, since it has been found in tissue cultures that glial cells can release material that stimulates the growth of neurons (68, 110). Since types of glia are diverse and the functions of glia are many and varied, it is quite possible that glial responses precede and promote neural responses in the case of brain damage, but that neural reactions are the primary ones in the case of normal learning. The evidence is still so scanty, however, that more well-planned research will be needed before any valid conclusions can be reached about possible roles of glia in brain plasticity.

RESEARCH ON NEURAL PLASTICITY: POSSIBILITIES FOR FURTHER ADVANCES AND FOR APPLICATIONS

Our survey has shown many ways in which the nervous system changes, anatomically and biochemically, even in the adult mammal. And many circumstances evoke these changes, ranging from extreme treatments such as surgical intervention or sensory deprivation to mild and natural demands such as those involved in learning and memory. Because the plasticity of the nervous system presumably evolved to meet the requirements of behavioral adaptation and adjustment, it is not surprising that behavioral techniques can inflect and modify many aspects of the nervous system.

Of course, our knowledge of the forms and the extent of neural plasticity is still fragmentary. Although investigators at the end of the last century could already glimpse some aspects of anatomical plasticity of neurons, it is only since about the 1950s that techniques have emerged allowing real progress to be made in this field. There is still much descriptive work to be done. And beyond this lies the testing of hypothesized mechanisms and the assessing of functional significance of different kinds of plasticity.

But we already have a platform from which to launch speculations, both to guide further research and to envisage possibilities of application of research findings to problems of individual and social behavior. If we could determine the factors that facilitate and limit plastic changes in the nervous

system, what are some of the conditions and problems to which we could hope to apply this knowledge? It seems to us that an imposing set of topics could be attacked in this way, including these examples: a) Enhancing intellectual ability; b) on the other side of the coin, preventing or alleviating some kinds of mental retardation and some kinds of learning disabilities; c) alleviating or retarding senile decline in intellectual abilities; and d) promoting recovery of function after damage to the nervous system. We have already touched on some of these topics, but let us take up in more detail the first two on this list.

Enhancing Intellectual Ability

Both behavioral and physiological methods are being studied to promote full growth and development of the brain and of behavioral capacities. It is, of course, a matter of debate to what extent behavioral capacity is related to cerebral development, so we might note some recent research and discussion of this question before going further. It has long been presumed by lay people that head size is related to intelligence, but empirical tests of this hypothesis have been few and far between. A recent review by Van Valen (109) reported correlations ranging from 0.08 to 0.22. Van Valen argued that when allowance is made for the fact that brain size is not directly measured by head circumference and for other problems of measurement, the true correlation between brain size and intelligence may be as high as 0.3. Jerison (56) criticized Van Valen's argument, but he presented new data indicating correlations of about 0.1 between head size and intelligence in children, when the influence of stature was partialed out. (The raw correlations, without allowing for stature, were about 0.3). Thus a positive but low correlation between brain size and intelligence is probable, based on the available data on human subjects.

Several kinds of animal studies have been conducted in an attempt to test the hypothesized relation between brain size and intelligence. In some cases, mice have been selectively bred to produce high brain weight (HBW) and low brain weight (LBW) strains from the same foundation stock (34, 75). The selection programs were successful—brain weight is a trait that shows considerable genetic determination. Some behavioral consequences of the selections were also found—the HBW strains tended to be more active than unselected or LBW strains, and the HBW mice were also superior on some but not all tests of learning. Within genetically heterogeneous stocks of mice, brain weight has also been found to correlate positively with several measures of learning ability (55). Part of the difficulty of this kind of research is that no single behavioral test yields an adequate measure of

learning ability or intelligence. [In the case of brain chemistry also, different measures correlate with different behavioral tests, and it is difficult to make overall generalizations (115).] Our own work has shown that enriched environment both increases cortical growth and leads to improved learning behavior. These cerebral and behavioral effects are parallel in a number of respects (81), but correlation does not demonstrate causality. Elsewhere we have speculated on possible reasons for evolution of responsiveness of the nervous system, even in gross morphology, to environmental demands (77). Zamenhof (122) has attempted to produce large-brained individuals by various biological treatments. For example, allowing only one rat fetus to develop (whereas there are usually ten or more) causes all of the maternal resources to be devoted to the single fetus. Such a singleton is larger than normal, and it has more brain cells than the normal rat. Preliminary behavioral evidence indicates that such "super rats" are also better in behavioral tasks than are control animals of the same stock. Zamenhof has also observed that some rats with unusually large brains occur without any experimental intervention. He argues that the factors that limit brain size are largely unknown and that it should be possible to inhibit some of them to produce large-brained (and potentially more intelligent) individuals. Zamenhof hopes that research along these lines will eventually be applicable to human beings, but in the meantime behavioral methods are available, as we will discuss shortly.

Attempts have also been made to look for relations between brain size and intelligence across species of mammals. Some investigators have argued that such an effort is misguided, since different species have evolved for different specific behavior rather than for overall ability (see Glickman, 40, for a discussion from this point of view). But others (e.g., Riddell, 72) have claimed that tests of general cross-species intelligence are possible and that they correlate positively with various indices of the "excess neurons" that a species possesses above those needed for vegetative functions.

The fact that brain size can be affected by various biological factors does not mean that behavioral factors can be neglected. Dudek (25) recently investigated whether the genetic constitution of high-brain-weight strains of mice guaranteed maximum growth of brain regardless of environment and experience. He found that the brains of the HBW strains responded just as much to enriched or impoverished environments as did the brains of LBW strains. Furthermore, the selection for brain weight may actually have been made partly on a behavioral basis, according to recent work of Hahn (47). Hahn investigated the effects of cross-fostering mouse pups (i.e., assigning HBW pups to LBW mothers and LBW pups to HBW mothers) versus in-fostering (i.e., assigning pups to foster mothers of their own strain). Cross-fostering reduced substantially the differences in brain size, whereas in-for-

stered pups showed the characteristic brain weights of their strains. Thus, there is a parental effect on brain weight in these strains. Whether it is due to behavioral differences (e.g., the HBW mothers have been observed to be more active with their pups than LBW mothers) or to other causes (e.g., the HBW mothers might have a better milk supply) is not yet known and will require further research to determine, but other work has already shown effects of differential preweaning experience on brain measures (65, 86). Furthermore, much research with human infants has shown effects of environmental stimulation and complexity on behavioral and intellectual development (51, 52, 119).

The fact that both environmental and genetic factors contribute to mental growth is also implicated in the work of Piaget and his colleagues on stages in intellectual development. A sequence of specific stages has been found, such that one stage (e.g., the stage of formal operations) cannot occur until the preceding one has been achieved (e.g., stage of concrete operations). Moreover, typical ages have been observed for the several stages, although results of all workers do not jibe completely concerning the exact ages or the fixity of the ages. A brighter child masters a stage more rapidly, but passes through the same sequence as a duller child. A rather controversial attempt has been made by Epstein (27, 28, 29) to link the Piagetian behavioral stages to alleged spurts in brain growth. According to Epstein, there are spurts in growth of brain and intellect (called "phrenoblysis") at ages 2–4, 6–8, 10–12, and 14–16, with periods of slow growth in between. While the brighter child can accomplish each mental stage more quickly, according to this viewpoint he or she must wait for the next phase of cerebral growth before beginning the next stage of mental development. But even if the brain has matured to the point where it can sustain more complex mentation, this is unlikely to occur unless the environment is sufficiently rich and challenging to promote such development.

Preventing or Alleviating Mental Retardation and Learning Disabilities

Hunt (51) has stated that there is mental retardation of social origin, occurring when the environment of a child does not provide sufficient stimulation to foster normal development. He has reviewed many studies whose results support this interpretation. Some of Hunt's own research has been done at an orphanage in Iran where children were considerably retarded in development of motor behavior and of speech. He was able to show that providing more interaction with caretakers led to significantly faster development. Different types of interaction affected different aspects of behavior. As Yarrow et al. (119) reported from their work with infants, an environ-

ment is not generally enriching or impoverishing, but rather there are specific kinds of experience that lead to development of one or another aspect of behavior. In Hunts's latest orphanage group, special attention to development of speech led to even faster progress than among American children living at home. Thus, depending upon the number of children per adult and the instructions and training given to the caretakers (who were rural women with little formal education), the same orphanage, drawing upon the same population, could produce either retarded or advanced children.

It is possible that some specific intellectual deficits and learning disabilities may be related to inadequate growth and development of certain regions of the brain. It is becoming possible to study relations between brain anatomy and behavior in living subjects by use of radiological techniques such as computerized axial tomography described above. Galaburda et al. (35) suggest that some deficiencies in language abilities may be related to the fact that the brain contains speech areas that are small in both hemispheres. They mention the case of a man diagnosed as having developmental dyslexia and whose father and brother also had similar difficulties. Preliminary study of the brain of this subject has indicated that the planum temporale on both sides is smaller than in normal brains. It has also been hypothesized that retarded development of speech and reading occurs when neither hemisphere has a clear dominance for these functions. Let us suppose that through further work of this sort certain characterisitcs of the gross anatomy of the brain can be correlated closely with difficulties in communication abilities. Suppose further that with the advance of radiological techniques, the necessary X-ray exposure can be reduced to completely nonharmful levels. It is possible then to imagine that in the future young children will be routinely examined for cerebral anatomical indications of developmental difficulties in language skills and other abilities. Remediation could then be started at early ages when it can be most effective rather than waiting for several years until frank disability is evident.

SUMMARY AND CONCLUSIONS

Recent research has demonstrated that the nervous system is plastic not only in ways hypothesized decades ago but also in ways grasped only recently, and further discoveries of this sort are inevitable. Yet this is not the general and almost unlimited plasticity that many assumed during the first four decades of this century. Both the overall layout of the nervous system and many specific connections are determined by genetic instructions. Thus, we now know far more about both plasticity and the limits of plasticity than was true a decade ago.

Many plastic changes in ramifications and connections of neurons have been demonstrated in response to severe or harmful treatments such as depriving young animals of normal sensory stimulation or transecting tracts in the brain. But such harsh treatments are not necessary to induce significant cerebral effects; mild experiences in differential environments and also formal training lead to measurable changes in the biochemistry and anatomy of the brain. Furthermore, many of these effects occur not only in young animals but also in adults. Several alternative hypotheses to account for the cerebral effects of differential experience have been ruled out by direct tests; thus the cerebral effects cannot be attributed to stress, to hormonal mediation, or to speeded maturation, nor do they follow the model of effects of sensory deprivation or distortion. Recent experiments with improved controls show more clearly than heretofore that training causes significant modification in brain measures.

A catalog of plastic changes in the adult nervous system showed that many possibilities have now been demonstrated. These include the following: a) Functional changes at existing synapses. b) Changes between active and quiescent states of neurons or parts of neurons. c) Anatomical changes in axon terminals, dendrites, dendritic spines, and synapses. d) Proliferation of glial cells. Although it has been dogma that the production and differentiation of new neurons can occur only early in life, some authors have suggested that even these events can occur to a limited extent in the adult mammalian brain.

Although our knowledge of the forms, extent, and limitations of plasticity is still far from complete, we can envisage possible applications to many conditions and problems of individual and social importance. Among these are the following: a) Enhancing intellectual ability; b) Preventing or alleviating some kinds of mental retardation and some kinds of learning disabilities; c) Alleviating or delaying senile decline in intellectual abilities; d) Promoting recovery of function after damage to the nervous system. Much can be accomplished along these lines by behavioral techniques, in some cases alone, and in other cases in conjunction with physiological techniques.

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The Comprehensive Handbook of Behavioral Medicine, Volume 1

CHAPTER 10

A Systems Approach For The Assessment And Treatment Of Memory Problems

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The differentiation of "normal" forgetting from "abnormal" memory failure and the effective treatment of memory problems are two major areas of concern for clinicians and laymen today. Although it may be relatively easy to identify classic memory dysfunction in cases related to amnesia, Korsakoff syndrome, cerebral accidents, and advanced organic brain syndrome, the differentiation of mild and moderate cases from normal functioning presents considerable difficulty. The purpose of this chapter is twofold. The first is to evaluate our current practices in processing memory complaints and in diagnosing and treating memory dysfunctions. The second is to suggest a systems approach for diagnosing and treating memory problems. Specifically, this chapter will describe the use of behavioral techniques in the treatment of memory problems.

The source of difficulty in diagnosing and treating memory problems lies in the multidimensionality of memory. In the last several decades, psychologists, neurologists, and neuropsychologists have attempted to establish models of memory and the relationship between memory and brain functions. While no model is completely satisfactory, it is now possible to identify some major characteristics of memory (70) and the factors that influence its functioning. Imagine memory as a multidimensional entity with interacting dimensions. One dimension of memory consists of dynamic processes related to the encoding, storage, and retrieval of information. Another is a theoretical definition of different information storage capacities—iconic (sensory), primary (short-term), secondary (newly learned information), and tertiary (well-learned or very familiar) memory stores. A third di-

mension defines the modality-specific properties of the incoming information—verbal, spatial, visual, auditory, olfactory, etc. The functioning of any or all of these dimensions of memory is affected by differences in the characteristics of the total person—patterns of responses to environmental stresses, changing affective states, personality and cognitive styles, health, differences in intelligence, education, socioeconomic status, and transient and stable neurological, hormonal, and physiological states. The many factors that can influence memory make it clear why the level of functional memory is fluid and subject to intra- and interpersonal variability.

Accordingly, any observed and subjectively perceived changes of memory may be attributed to changes in any one or combination of the above dimensions. Differential diagnostic instruments for memory dysfunction would be incomplete if they only assessed the performance of specific types of memory without accounting for the impact of environmental, physiological, and personal status on the individual assessed. Similarly, treatment of memory dysfunction must relate to individual differences in order to have lasting and effective results. This multidimensional model of memory should therefore be kept in mind to use as a guide to evaluate the adequacy of current practices in the diagnosis and treatment of memory problems.

It is generally agreed, from the findings reported by memory researchers and clinical investigators, that our memory capabilities decline over our lifespan (see 4, 11, 12, 22 for selective reviews). Individuals claim they can detect, and frequently register complaints about, their failing memory (53, 64). Some of the questions clinicians frequently ask regarding memory complaints are: How can these memory complaints be evaluated? Are these memory complaints real or imagined? What appropriate memory tests should be used? What memory treatment methods are most effective? And who can benefit from memory training? Answers for these questions are beginning to emerge from different research and demonstration programs evaluating various aspects of memory. The need to answer these questions is obvious; however, there are few systematic investigations linking the examination of memory complaints with their assessment and treatment.

It is difficult for patients to describe adequately their memory problems, and no comprehensive descriptive instrument is available to systematically document memory complaints. Clinicians have to depend upon their clinical judgment and experience to evaluate the validity of memory complaints. The relevance of most existing memory psychometric tests for the differential diagnosis of memory problems has been challenged (30), and existing treatment programs are only in experimental stages. Memory complaints are often considered symptoms of other dysfunctions such as depression or anxiety reactions (45). Treatment for anxiety and depression may

offer relief from these symptoms experienced with failing memory or memory complaints (52); they do not treat, however, memory difficulties that are not related to depression or anxiety. Each of these factors will be reviewed and discussed in detail in the following sections.

COMPLAINTS, DIAGNOSIS, AND TREATMENT

Memory Complaints

Clinicians and primary health care professionals are frequently confronted with complaints of failing memory, especially from the elderly. At the present there are no standardized methods of categorizing these complaints so they can be used as a basis for eventual formal evaluation and remediation. Memory problems may mean different types of difficulties to different people. When a layman complains about his memory he may be complaining about anything from absent-mindedness to learning difficulties. When clinicians refer to "memory disorders," the term usually denotes a generalized learning or cognitive breakdown (47). Experimental psychologists, on the other hand, define memory according to specific encoding, storage, and retrieval processes (70). Semantic confusion is created by differences in definitions of memory problems among professionals and laymen. Consequently, nonspecific memory complaints are generally ignored if the consequences of the complaints are judged harmless to the individual (64).

Two studies have attempted to evaluate the relationship between memory complaints and actual memory functioning. Lowenthal, Berkman, and associates (64) found that two-thirds of a 600-person community sample of persons over 60 years old tended to complain about their memory loss, and this incidence was higher in institutionalized individuals. The study also found that the frequency of memory complaints fluctuated with time, and that the severity of the complaints was not corroborated with performance on several brief formal tests of memory function.

In a more comprehensive study, Kahn, Zarit, Hilbert, and Niederehe (53) examined 153 persons 50 years and older; 113 were psychiatric patients, and 40 were normal control subjects. Perceived memory was evaluated by a 5-point scale based on the general question, "Do you have trouble with your memory?" The subjects then proceeded to describe their memory problems relative to changes in recent and remote memory. The affective conditions were evaluated by the Hamilton Rating Scale for Depression and the McNair and Laire Adjective Mood Checklist. The Face-Hand Test and the Mental Status Questionnaire were administered to evaluate memory functions.

The results indicated that larger numbers of memory complaints were associated with a greater degree of depression, while poor memory performance was associated with low scores on the tests of brain function. Complaints about memory functions were found not to be related to decreased memory functions as defined by the objective tests. These findings substantiate those reported by Perlin and Butler (79) but are not in agreement with those of Allison (1) and Williams and Jaco (100), who reported a positive relationship between depression and decreased cognitive performance.

The reasons for the discrepant results reported in these studies relating memory complaints, actual functions, and affective status are not clear. What is clear is that a better instrument should be developed to help an individual describe his memory problems. A detailed examination of self-perceived memory problems should provide an indication of affective states, specific versus generalized memory problems, the degree of debilitation in everyday function, the effect of self-perceived memory problems, self-esteem, self-worth, and motivation for change. This information could be used to select appropriate memory tests for the evaluation of specific complaints and to design individualized treatment programs.

Memory Assessment

The clinician faces three major problems in the selection of tests for the differential diagnosis of memory problems. First, there is no available memory assessment battery that defines a comprehensive pattern of functioning among the various dimensions of memory.

Second, there are few available memory tests that adequately assess everyday memory complaints and problems. Third, tests that could evaluate specific types of memory are often too time consuming to administer and thus impractical.

There are three types of memory tests—brief mental examinations, uni-or multidimensional psychometric tests, and laboratory-based information processing tests. All three types are deficient in two ways: They sample very limited aspects of the various dimensions of memory, and it is difficult to interpret the scores in a way that isolates possible causal factors for remediation.

Gross memory dysfunction in orientation, recent, and remote memory can be easily identified by either a patient interview or the administration of brief mental status examinations (e.g., 51, 67, 80). These examinations evaluate the patient's awareness of general personal (e.g., name, birthdate) and colloquial (e.g., names of presidents) information. Individuals with

mild impairment often perform satisfactorily in this type of examination. More elaborate mental status examinations are available, including evaluation of other abilities such as motor and perceptual skills (e.g., 48, 61, 81).

Brief mental status examinations could be used as preliminary screening devices to support initial clinical impressions; however, they offer limited insight into the differential diagnosis of memory problems.

The psychometric methods are often employed by clinicians using paper and pencil tests or questionnaires to isolate abnormal memory functions by comparing patient's scores with those standardized on community-dwelling normal adults or patient groups. The most widely known of these tests is the Wechsler Memory Scale (95) which contains seven subtests: personal and current information, orientation, mental control, logical memory, memory span, visual reproduction, and associate learning. Similar tests are those developed by Wells and Martin (97), Babcock and Levy (6), Cronholm and Ottoson (24), and Williams (99). Unidimensional tests that evaluate a specific aspect of memory are also available. A sample of tests for the evaluation of verbal memory has been developed by Shapiro and Nelson (88), Walton and Black (93), Kendrick, Parboosingh, and Post (55), Inglis (46), and Buschke and Fuld (16). A sample of tests for nonverbal memory has been developed by Graham and Kendall (38), Benton and Spreen (10), Zangwill (103), and Fowler (32).

The Wechsler Memory Scale is a widely used test for memory which was originally designed to detect memory deficits with brain-injured and organic patients. In subsequent research efforts utilizing the test, Cohen (21) could not differentiate psychoneurotic, psychotic, and organic patients when he controlled for age and intelligence. Parker (76) found the Wechsler test could not discriminate between brain-damaged and normal subjects with equivalent IQ scores. Prolonged institutionalization was found to produce a depressive effect on performance with both organics and psychotics (40). Further, differences in specific subtests were found to depend on attention and concentration, not on memory (94). The limitations of this test are further reviewed by Mensh (68), Newman (73), Davis and Swenson (25), Dujovne and Levy (26), Kear-Colwell (54), and Erickson and Scott (30).

One important lesson to learn from the validation research on the Wechsler test is that memory is a multidimensional entity that fluctuates and changes depending upon a multitude of factors. The notion of a single composite memory score such as the memory quotient used by Wechsler is misleading and simplistic. For the purpose of remediation, the significance of a pattern of memory performance must be judged in the context of individual differences in intellectual, experiential, environmental, motivational, and health factors. For example, two individuals may show similar deficits in a test of short-term memory ability to recall digits immediately after presen-

tation. For one individual, the difficulty may be cognitive; he may have limited experience in dealing with numbers and not know how to encode the information into memory under the conditions of the test. Teaching this person some basic mnemonic skills for numbers may be sufficient to improve this aspect of his short-term memory. The difficulty for the second individual may be due to transient environmental stresses, such as his daughter getting married next week, and he may be too distracted to attend to the digits. His performance scores might be improved if the test were repeated after the wedding. It is important to note that identification of an abnormal pattern of memory function is only one aspect of differential diagnosis; the methods of isolating the causal factors for remediation have not been adequately explored.

The information-processing tradition (62,70) originally used to investigate memory functions may be more suited to isolate difficulties in specific memory processes. This laboratory-based method evaluates the performances of various memory processes by manipulating stimulus, information, context, and state variables. Researchers concerned with isolating specific memory dysfunctions with various patient populations have employed this method quite fruitfully. The reader is referred to: Talland (89), Cermak and Butters (20), Butters and Cermak (17), and Baddeley and Warrington (7) for samples of research on memory functions in amnesic-Korsakoff patients; Inglis (47) for various memory disorders involved in cerebral injuries, Korsakoff's, and senile dementia; Botwinick and Storandt (12), Craik (22), and Fozard and Thomas (34) for reviews and studies on age-related memory changes in normal adults. It should be noted that the concomitant influences of personality, motivation, and environment on memory performances have received relatively little attention in these investigations.

One practical problem with existing memory tests is their inability to relate test performances to common memory complaints. Patients generally complain about forgetting names of associates and relatives, frequently used or just memorized telephone numbers, shopping lists, birthdates, dates of major personal events, and failure to recognize faces, streets, etc. Is the digit-span test an adequate instrument to assess memory failure for telephone numbers? Is the memory-for-design test adequate to evaluate problems of face recognition? Is the verbal paired-associate test appropriate to evaluate an inability to retrieve a name given a particular face, or to retrieve an address or telephone number given a specific name? The answers to these questions are not clear at present. It is clear that many memory tests are quite complex and have been criticized as meaningless from the patient's standpoint (11). Some test results cannot provide adequate information to confirm or reject everyday memory complaints.

A related third problem of memory testing is that clinicians frequently have limited time in which to provide a diagnosis. Brief mental status examinations provide preliminary indications, but they are insufficient to provide a reliable diagnosis. Standardized multifaceted tests are frequently too time consuming and have problems of external validity. Procedures associated with the information processing approaches may need special equipment, frequently lack face validity, are time consuming, and are not standardized. The selection of an appropriate test battery for memory testing presents a formidable problem for clinicians at present.

Memory Remediation

Although the extant knowledge of memory functions has advanced to take into account the important influences of environment, personality, cognitive style, motivation, and physiology, the state of the art in clinical memory remediation unfortunately has not advanced appreciably since the days of Simonides in 477 B.C. Simonides pioneered the "method of loci" as an aid to create a visual organization scheme to encode information into memory. This visual mnemonic method is taught today in modern memory improvement programs (19, 39, 63) and is still being evaluated as a viable memory improvement method for the young (13), for elderly adults (84), and for patient groups (78). Memory improvement methods typically emphasize the use of visual imageries to encode information. Relatively little emphasis has been placed on information-retrieval techniques (13), nor has effort toward utilizing individual differences in the design and maintenance of remediation programs been extensively evaluated.

There are three major mnemonic methods—methods of "loci," "peg," and "link." For a detailed description of these and associated procedures the reader is referred to Cermak (19), Higbee (39), and Lorayne and Lucas (63). The method of loci uses a well-learned series of geographical or architectural locations as anchors or loci in the memory scheme. One may visualize putting specific items at various locations in one's house. During recall one may "walk" through the house to retrieve these items.

The peg system is generally used to recall numbers of sequential events. Each digit is associated with a "peg," or phonetically similar familiar object. For example, one is associated with "bun," two with "shoe," three with "tree," four with "door," etc. After learning the "pegs," an individual may use the system to remember numbers or events. For instance, the number 123 may be visualized as a bun put on top of a shoe that is balanced on top of a tree. If an individual needs to mail a letter first thing in the morning, he

may want to visualize a letter placed on top of a bun flying toward a mailbox. The link method is used to remember sequential items or events by forming a chain of interacting imageries. Using the peg system to remember the number 123 is a special case of the link method.

In addition to the use of visual imageries, mnemonic methods have been developed to take advantage of the phonetic and categoric grouping systems. Professional mnemonists have developed mnemonic systems to remember everyday information such as telephone numbers, names and faces, shopping lists, stock-market information, foreign and English vocabulary, etc.

Experimental evaluation of memory techniques has mostly been confined to the facilitative effects of visual imageries, particularly with paired-associate learning tasks. Clinical tests of mnemonic methods for memorizing of everyday information (e.g., names-faces, events, etc.) are beginning to appear in the literature (59, 104). Atwood (5) and Paivio (75) demonstrated that visual imageries can facilitate memory performance in young adults. Such techniques have been shown to be beneficial in memory improvement with the community-dwelling elderly (85, 87, 91, 104), the brain injured (49, 60, 78), and the educable mentally retarded (57, 77). Failure to improve memory using visual mnemonics has also been reported. Baddeley and Warrington (7) and Jones (49) reported that amnesic patients derived no benefit from visual imageries. However, they were able to improve their memory with the aid of phonetic and taxonomic category mnemonics. The elderly tended to use verbal more than visual mnemonics, and they did not use visual mnemonics as spontaneously as the young (41, 42). Patten (78) reported that several patients with organic diseases failed to use visual mnemonics. On the other hand, these patients were reportedly not aware of their memory deficits or had no interest in improving their memory. In summary, there is overwhelming data in the literature suggesting that almost anyone can improve his memory skills with mnemonic techniques.

Inspection of the results of successful laboratory demonstrations shows unanimously that mnemonic techniques facilitate initial information acquisition and short-term retention. However, the results of a few studies evaluating the long-term effects of visual mnemonics are not clear. Thomas and Ruben (91) found that groups (25 to 65 years) with verbal instructions specifying visual mediational techniques, and with cartoons depicting specific interacting relationships, improved their associate recall better than groups with no specific instructions over an eight-month period. On the other hand, Lewinsohn, Danaher, and Kikel (60) demonstrated that subjects with visual mnemonics instruction showed improved retention 30 minutes after acquisition but not after one week. A similar lack of long-term effects was found by Glasgow, Zeiss, Berrera, and Lewinsohn (36), and Treat, Poon,

and Fozard (92). The discrepancy in results could be due to differences in mnemonic techniques, instructions, or subject samples.

The successful acquisition of mnemonic techniques is the first goal of memory skill training. The second and perhaps the more important goal is to help motivate the individual to retain the newly learned skills over time and generalize these skills to similar or novel learning situations. The lack of systematic maintenance of these techniques may account for the reported absence of long-term facilitative effect in some studies. A significant amount of work is needed to advance this area of investigation.

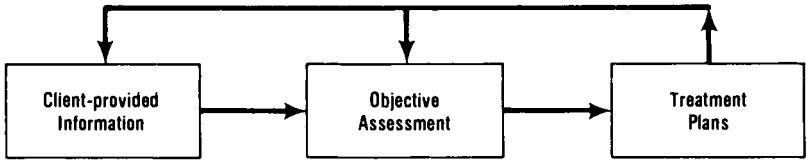
A SYSTEMS APPROACH

It is clear from the above review that a considerable amount of research is needed to advance the state of the art of effective memory diagnosis and treatment. Meanwhile, it is necessary to try to revise existing procedures and devise new ones in order to obtain accurate descriptions of memory complaints, meaningful diagnosis, and effective treatment. This section proposes a preliminary model linking self-assessment, objective evaluation, and treatment of memory problems. Particular attention is focused on behavioral procedures for the treatment of memory dysfunctions.

Table 1 presents some pertinent variables for self-assessment, objective evaluation and treatment. For individuals with specific memory complaints, the system could be used to objectify and validate the complaints, to evaluate pertinent areas that might contribute to the difficulties, and to suggest concurrent, specific memory training and other treatment alternatives. The results of the initial self-assessment and objective tests could be used as criteria for treatment effectiveness. For individuals with nonspecific memory problems, the system could be used as an overall diagnostic and treatment plan. In cases related to amnesia and brain damage, specific techniques reducing interference and consolidating memory traces have been shown to facilitate memory recall (96). For these individuals, other specific mnemonic, remedial, and supportive therapies would be needed. In cases of memory dysfunction where the etiology is related to metabolic malfunction or drug intoxication (27, 28), appropriate therapy in addition to memory training may restore confidence in memory abilities. For individuals who simply want to improve their memory, an education program about factors that would facilitate or inhibit memory, in addition to training in mnemonics with specific emphasis on retrieval and maintenance techniques, would be helpful to sustain long-term benefits.

In the preliminary self-assessment of memory problems the question arises: Can an individual give an accurate report of his memory status? Nis-

Table 1. Pertinent Variables in the Diagnosis and Treatment of Memory Problems.



1. Memory Functions	• identification and documentation of nonspecific and specific memory complaints • rank order of the relative strengths, weaknesses, and frequency of forgetting of concrete examples of different types of memory	• short mental states examination • memory tests to evaluate specific types of memory functions	• mnemonic training • retrieval plans • memory maintenance plans
2. Individual Differences	• health, medications • environmental stresses • affective states • cognitive styles • motivation to improve • coping strategies	• physical examination • neuropsychological examination • state/trait anxiety and depression evaluation • personality inventory • cognitive styles	• individualized training program • mnemonic techniques • attitude, motivation • information feedback • reinforcement • practice • counseling • medical/pharmacological intervention

bett and Wilson (74) reviewed research evidence that suggests individuals have little or no direct introspective access to higher-order cognitive processes. Mandler (65, 66), Miller (69), and Neisser (72) have proposed that we have only limited awareness of our evaluation, judgment, and problem-solving behavior. However, accurate reports may be obtained if the questions are logical, plausible, and specific, and the response possibilities are highly structured (74).

This suggests that a systematic and structured self-assessment inventory may help an individual articulate his memory problems. To do so, it should

have some nominal scaling properties so that the individual may rank order and compare his strengths and weaknesses. Specific and concrete examples which tap the various dimensions of memory described earlier should be given so that an individual can assess his memory problem according to some relative criteria. For example, the elderly frequently complain of lapses of memory for new information about names, faces, dates, or remote events. Specific examples of these complaints could be given so that the person could rank order his perceived strengths and weaknesses in terms of familiarity of information (e.g., new vs. familiar information), retrieval intervals (e.g., immediate vs. later retrieval), types of information (verbal or spatial), types of learning (e.g., incidental vs. intentional), conditions of recall (e.g., free recall vs. recall with available cues), etc.

It is not anticipated that an individual can accurately evaluate his own memory, given specific guidelines. However, this type of instrument may help to establish a dialogue between the clinician and patient through which a pattern of perceived memory problems and peripheral factors associated with the problems may be isolated. The results can be related more easily to objective measures of various memory and memory-related functions. It is possible that the low correlations found by Lowenthal and associates (64) and Kahn et al. (53) between memory complaints and "actual" memory functions may be due to either inaccurate or incomplete indices of self-report and memory performance scores, or both.

In addition to self-assessment of memory problems, self-report should contain information about the following in order to evaluate possible peripheral influences on memory difficulties : 1) Recent changes in the environment that might produce situational anxiety or depression; 2) the individual's propensity to be overly anxious or depressed; 3) existing styles and methods in learning and remembering information; 4) the reasons for wanting to improve memory; 5) the methods used in coping with memory difficulties; and 6) recent health status and medications.

Information obtained from self-assessment and report should be used as guidelines for the selection of objective tests. When appropriate, a physical examination, an evaluation of possible interactions among medications, and a neuropsychological examination may be useful in isolating possible metabolic malfunction, drug intoxication, or brain damage as reasons for memory and learning difficulties. Tests of state and trait anxiety and depression, for example, the State-Trait Anxiety Scale, the Zung Depression Scale or the Hamilton Rating Scale for Depression should be used to evaluate possible distractive effects on learning and memory. Evaluation of existing styles and techniques for remembering information may be assessed so that initial mnemonics appropriate for the individual may be employed.

The selection of appropriate memory tests to evaluate memory complaints and functions is a common dilemma among clinicians. In an excellent review of clinical memory tests, Erickson (29) concluded that collaboration among clinicians and experimental researchers is mandatory to produce meaningful and valid tests. Erickson (29) suggested a multiple-step test procedure starting with a brief "bed-side" screening instrument designed to assess verbal and spatial learning and retention, followed by different test batteries evaluating specific deficits when required.

There is a trend among memory remediation clinics to develop memory tests with meaningful stimuli which can be used to relate to everyday memory complaints. For example, a face-name recall test, a face recognition test, remembering ordered sequences of tasks, and recall of specific parts of a story are employed at the Neuropsychology Laboratory at the University of Oregon (59) to validate memory complaints of brain-damaged patients. A face recognition test was also developed and employed at the VA Hospital in Boston (2).

A naming latency procedure for identifying pictures of objects varying in familiarity, datedness, and usage was developed at the Geriatric Research, Educational, and Clinical Center, Boston VA Outpatient Clinic (82, 90). This procedure could be used to evaluate motor-perceptual speed as well as memory retrieval speed from long-term memory. The procedure is sensitive in differentiating retrieval speed from schizophrenic and normal individuals (101) as well as retrieval from various age groups (89). The frequency of "blocking" during recall of familiar information could also be evaluated. The naturalistic observation method was used by Zola, Gunilla, and Oberg (105) to evaluate recognition memory recall of patients with Korsakoff syndrome for events observed during a field trip.

A test is being developed at the Boston VA Outpatient Clinic to present information in a controlled colloquial setting. The subject is told that he is going to a get-together and will meet new acquaintances. A booklet is given to the subject depicting sequential scenes from the party in which names, faces, addresses, dates, phone numbers, general information, etc. are presented and tested in a story-related manner. The presentation rate is controlled and the information is tested at various intervals ranging from immediate test, minutes, and one week following the presentation. Questions of remote colloquial information are interspersed in the testing procedure. In this manner, verbal, spatial, new, and remote information, as well as free and recognition recall at various retrieval intervals could be evaluated.

A successful memory skill-training program should be designed with the requirements of the individual's daily needs, environment, and cognitive styles in mind. Most memory improvement programs advocate the acquisi-

tion of very similar mnemonic approaches to organize and encode information and have shown successful short-term benefits. However, in order to assure long-term benefits, it is necessary to develop a practical "retrieval plan" (14) to aid an individual during recall and a maintenance program to sustain the memory improvement over time. The following sections will delineate some experimental findings that could be incorporated in a memory training program for long-term benefits.

Any scheme that guides an individual to search through memory for specific information may be conceptualized as a "retrieval plan." The plan should tell an individual where to begin, how to proceed, and when to stop the memory search. Qualitative differences in encoding and retrieval operations were found to have significant effects on memory performances (23, 31, 43, 44). Retrieval efficiency is highest when similar encoding variables are used as retrieval cues. Since it is clearly to the advantage of the learner to know what to do during recall, a retrieval plan using relevant cues can have a marked facilitative effect (102).

Bower (14) proposed several retrieval plans for recall of memorized lists of items. The "generative rule" plan (69) proposed that the subject first generate a brief description of the memorized set followed by a series of "decision rules" distinguishing patterns that are in, as opposed to out, of memory set. The "hierachical retrieval" plan (15) proposed a category-sorting strategy, beginning the recall at the top of a hierachical structure and working recursively from the top down. The "associative retrieval" plan (3) proposed that the subject search out associative connections among list words during learning and particularly at recall. The methods of loci and peg (described earlier) have their inherent retrieval plans. Retracing one's steps through the loci and working through the sequential pegs would help the subject with recall. The appropriateness of these theoretical retrieval plans for the recall of everyday information has not been tested. These plans, along with other techniques based on experimental findings, should be tested in order to further the development of practical retrieval strategies for everyday information.

Ambiguity exists in the literature identifying successful behavioral techniques (37) for the maintenance of cognitive skills. Most techniques emphasize a "broad-spectrum" or multifaceted approach instead of single or isolated techniques (56). Experiences reported by self-help groups (35) (e.g., Alcoholics Anonymous, assertive training, physical fitness, Weight Watchers) as well as by programmed learning and behavioral modification studies suggest that five factors are important in the modification of habits. 1) The training program should inculcate in the learner the attitude that he is capable of carrying out the desirable changes in behavior and will succeed in his effort to do so. 2) The program should provide some measure of

progress so that the individual has proof that the skills do work. 3) Salient extrinsic and intrinsic reinforcers to maintain the skill should be identified for both the clinician and the learner. 4) The program should help the learner establish a routine to practice the skills periodically. 5) Individual differences should be utilized in the design of the training program.

The attitude of the subject is, first and foremost, central to the successful maintenance and outcome of the training program. The clinician's approach, instructions, and expectations, and the subject's expectancy and interpretation of "social desirability" to generate images, all were found to influence the production of mental imageries (see 98, for a review). It is possible that variability in the results of clinical and laboratory evaluation of the mnemonic techniques is due to individual differences in the instructional and treatment techniques of the clinicians.

Any apathy toward applying the mnemonic skills in everyday situations will be to the learner's disadvantage. Individuals who have experienced memory difficulties tend to be conservative and tentative in their efforts in memory recall. For example, with an elderly subject, the stereotype of impaired cognitive functioning in the aged may exacerbate the effects of memory difficulties. The subject must be convinced that his aging as such does not imply the inability to remember information; rather, it may necessitate approaching learning in what may appear to be a new manner. Korsakoff patients tend to say they can't remember if they are at all unsure of their response. Incremental confidence building is as important if not more so, as memory skill acquisition.

Positive attitude can be better maintained by appropriate feedback that the newly acquired skills are helping the learner in his daily functioning. Positive feedback will show that the skills are of constructive use to him and will strengthen the resolve to continue using the skills to his advantage. Keeping progress charts is another useful aid which helps the learner to objectify problems as well as progress. It is important for the clinician to set up a series of realistic goals and behaviors that can be attained by the learner. Success in remembering information that was perceived as difficult to remember may serve as intrinsic reinforcement to maintain memory skills. Other reward contingencies could be discussed with and incorporated by the learner so that the positive aspects of memory training can be accentuated. The clinician should also prepare the learner for the contingency of negative feedback, which could also serve as a useful indicator for the learner. Transient failure to retrieve information may indicate that the subject may not be applying the appropriate memory techniques, is too anxious, or is not paying attention during encoding or recall. It may also serve as a signal to the learner to adjust his mnemonic techniques or to practice the memory skills more frequently. Feedback and skills adjustments should

be monitored jointly by the clinician and by the patient when he is capable of self-monitoring.

Practice is an important factor for the acquisition and maintenance of physical skills, e.g., playing tennis, and cognitive skills, e.g., improving memory. It consolidates the necessary techniques by providing repeated experience which improves self-confidence. No one technique works for all subjects. Practice allows an individual to try to refine various techniques that might work best for him in different circumstances. In the study of age-related differences in simple decision time, for example, it is generally reported that the elderly are slower than the young. However, practice has been shown to reduce (83) or eliminate (71) age differences in decision time.

The potency of practice was demonstrated in a recently completed verbal learning study (92). The purpose of the study was to evaluate age differences in the carryover effects of various kinds of mnemonic training on paired-associate learning. Subjects memorized different lists of paired-associates in three sessions, with two weeks separating each session. In the second and third sessions, they were also retested on the earlier lists. For both young and elderly adults, the mnemonic groups performed significantly better than the no-instruction control group in the first session. At the end of the third session, the older subjects in the control group performed as well as those in the mnemonic groups. Some of the control-group subjects reported using organizational strategies at the end of the experiment but not at the beginning. Given sufficient opportunity, the subject may exploit organizational skills that appear useful in laboratory tasks. Thus, the incorporation of regular or self-imposed practice sessions for memory skills into a memory learning program could strengthen the patient's conviction that he can perform the memory task and maintain a desirable level of memory over a long period of time.

The considerable amount of intersubject variability and lack of long-term effect in studies evaluating the applicability of various mnemonic methods suggest that individual differences play a significant role in the successful outcome of memory training. Specific methods which have a demonstrated statistical effect may not be the appropriate and meaningful therapy technique for a particular individual. Although this criticism can be applied to most experimental tests of therapy techniques, the multidimensionality of memory may make evaluating specific techniques without taking into account relevant individual differences an almost meaningless exercise. Fortunately, results from studies examining various aspects of memory and individual differences may help to point the way toward designing preliminary training programs for a particular individual.

Among the many relevant dimensions of individual difference, cognitive style and personality, environment, and general abilities are very relevant

to designing an individualized treatment program. One dimension of cognitive style, "verbalizer and visualizer," may be germane to the preliminary selection of encoding and retrieval techniques (see 84, for a comprehensive review). Visual mnemonics, while effective for most individuals, may not work for people who prefer to use verbal or semantic encoding. The aged (41) and individuals in certain professions (e.g., psychologists and anthropologists; 86) tend to use more verbal mnemonics, and the refinement of existing verbal techniques may be sufficient for memory improvement for these individuals. Amnesic patients with bilateral damage to the temporal lobes can form interacting visual imageries; however, this technique does not seem to improve their memory. On the other hand, verbal mnemonics seemed to facilitate retrieval for the amnesic (7, 49). Left-hemisphere lobectomy patients can use verbal mnemonics to perform as well as normals (49). The above information could be useful in the design of individualized training programs for optimal results.

"Surgency" (18) is another cognitive style dimension that should be considered in the design of a training program. Fozard and Costa (33), for example, have shown that persons who are relatively "surgent" or happy-go-lucky, in comparison to sober, glum individuals are faster at retrieving information from short- and long-term memory as well as more rapid at simple decision making. Variations in the speed of cognitive processes provide an individual with an indication of the quality of his cognitive functioning; slowness of such processes is a common complaint of elderly persons.

Therapy to relieve some of the conservative attitudes, to become less rigid and more "surgent" in their responses, could be attempted for subjects who are initially hesitant or lack confidence in their effort in memory recall. Also, providing a person with information relative to understanding the conditions governing the speed of cognitive functioning may itself be of therapeutic value.

A training program should take into account the decremental influences of transient, environmentally induced anxiety and depression as well as inherent individual differences in these personality traits. Therapy and counseling aimed at the reduction and better management of these affective states should be included prior to and during the memory improvement program for all subjects who are more susceptible to affective changes.

Some adjustment in a training program should be made to take into consideration an individual's general abililties, education level, and occupation. Examples and stimuli employed in the training sessions, types of reinforcement and performance feedback, and frequency of practice should be individualized so that the training procedures will be related to and found meaningful by the individual. Outstanding work by Belbin and Bel-

bin (8) in skill training showed pedagogical techniques that allow the individual more control over the conditions of learning (coined "the discovery technique"), help the learner relate to the training procedure and to be more aware of the incremental progress.

CONCLUSIONS

We generally do not pay attention to our memory until we fail to recall specific information which we know we should remember. It is difficult to imagine the quality of our lives without this precious gift of memory. Yet we tend to take this gift for granted, and few make it a conscious habit to exercise and maintain this important function. The outline presented in this chapter is a preliminary proposal to consolidate various experimental findings into an effective system to evaluate and treat memory complaints and problems. It is necessary to set up controlled experimental programs to identify the simplest combination of treatment techniques for maximum effectiveness. Particular attention should be focused on evaluating the cost effectiveness of various treatment delivery methods, i.e., therapist-centered, therapist-administered, or self-administered programs (35) and the interaction between treatment delivery and treatment technique. Since very limited experience has been accumulated in the treatment of memory difficulties, the problems of high attrition rates and poor treatment compliance, found in most behavioral treatment programs, should be addressed. Experimental methods using single-subject designs (58), sequential presentations of various treatment components (9), and time series analysis (50) may be useful in the collection of clinical data and in the evaluation of treatment effectiveness in the clinical setting.

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The Comprehensive Handbook of Behavioral Medicine, Volume 1

CHAPTER 11

Treating The Complaint Of Insomnia: Self-Management Perspectives

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TREATING THE COMPLAINT OF INSOMNIA: A BEHAVIORAL SELF-MANAGEMENT APPROACH*

In a prospective study of risk factors in coronary heart disease, stroke, and cancer, Hammond (25) surveyed 1,064,004 persons and found that 13% of the men and 26.4% of the women complained of disturbed sleep. Williams, Karacan, and Hursch (60) surveyed 1600 individuals in Florida; 45% reported difficulty in getting to sleep (31% sometimes and 14% often). Similar results were found recently for adolescents: 37.6% reported occasional sleep problems while 12.6% reported frequent sleep disturbances occurring for one year or longer (50).

Methods of treating insomnia vary considerably. When home remedies fail, over-the-counter medications often constitute the second plan of at-

*Throughout this chapter, we refer to insomnia as "the complaint of insomnia." Insomnia is a complex problem that a person experiences, not a specific disease entity with a well-established etiology. The complaint can indicate disturbed sleep secondary to a wide variety of physiological and social/psychological problems (e.g., sleep apnea, nocturnal myoclonus, organ malfunction, chronic stress, depression, major life change events).

tack. Although these highly advertised drugs have little impact on sleep beyond placebo effects (33, 35), they can increase daytime drowsiness thus tending to encourage the person's perception that sleep is becoming worse. The person may think that more potent medications are required. Alcohol is perhaps the most common over-the-counter remedy—by itself or in combination with accepted hypnotic medications (cf. 53). Indeed, the complaint of insomnia has been implicated as one of the factors in the etiology of uncontrolled drinking problems (6); further, using alcohol in combination with certain hypnotics, especially barbiturates, can be dangerous.

After such efforts, patients may approach their physicians with the strong expectation (if not demand) that potent and effective treatments are available and can be offered.* The barbiturates may finally be losing their popularity due to research documenting 1) the rapidity with which persons fall victim to the dependence-tolerance cycle characteristic of these drugs (30, 31), and 2) the disruptions these medications cause in the sleep structure and cycle (33).

The negative effects of barbiturates and related drugs are clinically significant: REM and slow-wave sleep are suppressed, beta rhythm activity is increased, and persons attempting to withdraw abruptly often experience withdrawal reactions as well as REM rebound in the form of severe nightmares. Thus, during withdrawal a person's sleep becomes *more* disturbed than before the drug was originally prescribed. Understandably, the person concludes that his insomnia has worsened and that drugs are essential; to obtain some relief, drug use is resumed, possibly at an increased level (cf. Figure 1).

The benzodiazepines and other nonbarbiturate hypnotics (e.g., chloral hydrate, glutethidimide, methaqualone, meprobamate), although less detrimental in general, can affect sleep in ways similar to the barbiturates. On the positive side they decrease body movements, number of awakenings, minutes to sleep onset, and the number of shifts in sleep stages; increased total sleep time and Stage 2 sleep are common. However, they reduce total REM time and slow-wave sleep, and increase beta rhythm activity in the EEG. These medications can also lead to dependence and tolerance; with-

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*In a recent survey, Bixler et al. (8) asked 4358 physicians from around the country to estimate the percentage of their total patient population experiencing several sleep disorders. The total sample of physicians reported that 18.0% of these patients complained of insomnia. Estimates varied by subspecialty: General and Family Practice (2607 physicians), 15.2%; Adult Psychiatry (740 physicians), 32.6%. Obstetrical and Gynecology (319 physicians), 11.3%; Surgery (145 physicians), 18.1%; Pediatrics (64 physicians), 5.3%; Child Psychiatry (367 physicians), 19.4%. The complaint of insomnia is not uncommon to medical practitioners; solutions other than medications, however, may be scarce.

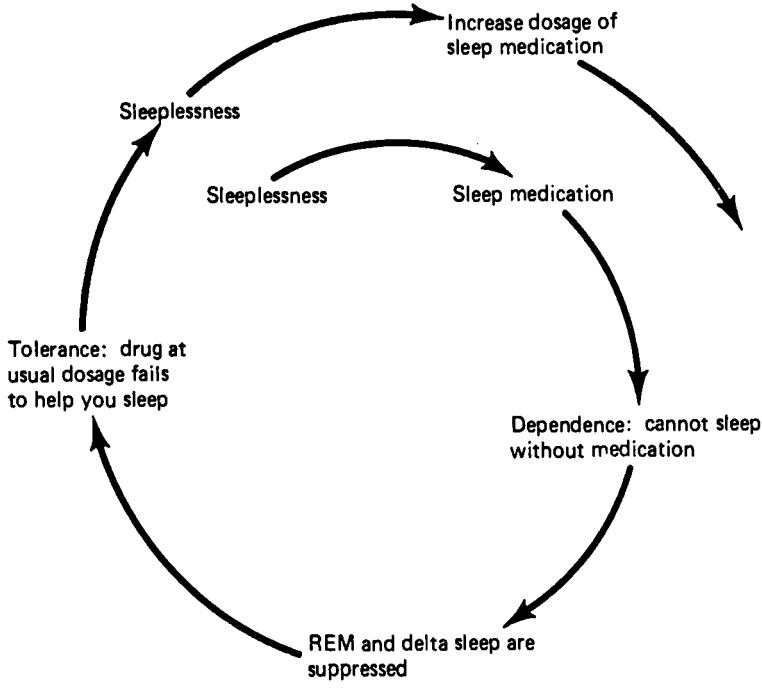

Figure 1: The vicious cycle

drawal from them can be accompanied by REM rebound and other adverse reactions (cf. 31, 35).

THE BEHAVIOR THERAPIES: A NEW ALTERNATIVE

Methods based on social learning theory (3, 55), also referred to as behavior therapy, can be effective in treating certain complaints of insomnia (cf. 9, 12, 38, 47) (cf. Table 1). Effects appear especially promising for persons 1) complaining of moderate sleep disturbances, 2) having difficulties in falling asleep initially, and 3) whose treatments are evaluated *only* by changes in self-reports of sleep. Effects have been much more modest when 1) persons with chronic and severe insomnia are treated, 2) variables other than minutes to sleep onset are used to evaluate treatment outcomes (e.g., minutes awake after sleep onset, number of awakenings), and 3) all-night sleep recordings are used in addition to self-reports (cf. Table 2).

Traub, Jencks, and Bliss (58), for example, used autogenic training: Three out of seven subjects decreased latency to sleep onset while two showed increased slow-wave sleep. Hauri and Cohen (27) reported that

Table 1.
Behavioral Treatments for the Complaint of Insomnia

Progressive Muscle Relaxation (cf. Bernstein and Borkovec, 7 Borkovec and Weerts, 13)	Tense and relax specific muscle groups (e.g., forearms, biceps, neck) systematically; focus on feelings of relaxation; usually done prior to bedtime.
Autogenic Training (cf. Traub, Jencks, and Bliss, 58; Nicassio and Bootzin, 48)	Systematically focus on specific muscle groups (e.g., arms, legs) to induce feelings of warmth or heaviness in those muscles.
Meditation (Cognitive Focusing) (Woolfolk, Carr-Kaffasan, and McNulty, 62)	Focus on single mental stimulus (e.g., word, phrease, image, or sound) imagined and subvocalized repeatedly; usually done prior to bedtime.
Biofeedback (cf. Feinstein, Sterman, and MacDonald, 21; Freedman and Papsdorf, 23; Hauri and Cohen, 27)	EMG: to relax specific muscles (usually frontalis) EEG: to increase central or occiptal alpha or theta activity; to increase sensorimotor rhythm (SMR) activity from centrofrontal cortex.
Stimulus Control (cf. Bootzin, 10, 11; Haynes, Price, and Simons, 29)	Associate bed and bedroom only with sleep; eliminate or reduce activities incompatible with sleep in bedroom, while in bed, and at bedtime; person must leave bed and bedroom if not sleeping or engaged in sexual activity.

SMR biofeedback training was effective in increasing sleep efficiency (time asleep divided by time in bed) among ten insomniacs; EMG feedback training to reduce frontalis muscle tension had no effect on ten other subjects. However, subjects who received *appropriate* feedback (EMG if they showed high muscle tension; SMR if they showed an excessive number of arousals) improved their efficiency from an average of 78.5% to 90.5%. Subjects who received *inappropriate* feedback (those with high muscle tension receiving SMR feedback; those with excessive nighttime arousals receiving EMG feedback) increased only from 80.2% to 82.3%. They concluded that both EMG and SMR feedback might be useful in certain types of insomnia if patients can be matched to an appropriate feedback mode. The other studies in Table 2 which report positive results provide hopeful leads for future research.

Our research and clinical efforts to date have been devoted to developing and evaluating nondrug strategies for treating chronic and severe sleep dis-

turbances. Three issues dominated our approach to the compliant of insomnia.

1. We have adopted the view that insomnia is a broad descriptive term usually referring to 1) physiologically documented sleep disturbances, 2) the complaint of poor sleep, and 3) impaired daytime functioning. Insomnia can arise from a variety of causes and can lead to a variety of outcomes.

2. We have been especially concerned with frequent and extended nighttime arousals, early morning awakenings, night-to-night variability in sleep duration and quality, as well as disrupted sleep cycles and decreased slow-wave sleep. These variables may be more likely to lead to the complaint of insomnia and impaired daytime functioning than extended latency to sleep onset alone (cf. 14, 22). They also appear to be less amenable to treatment than improving latency to sleep onset.

3. Research findings in behavioral treatments for insomnia are subject to criticism because all-night sleep recordings have typically not been employed to evaluate treatment programs; many studies have relied on retrospective self-reports (cf. 17). Self-report data have been questioned because they correlate only weakly with all-night physiological data (cf. 59).

Persons complaining of insomnia, for example, may tend to overestimate the amount of time to fall asleep or minutes awake during sleep on the previous night. Early in our clinical research with chronic and severe insomniacs, we recognized the need to evaluate treatments using *both* all-night physiological and self-report data. The low correlations between self-report and physiological measures may be expected because relatively independent phenomena are being assessed (cf. 42). Treatment may alter the person's perception of sleep, physiological characteristics of sleep, or both. It is essential to know which electrophysiological variables change with treatment and which are correlated with the perception of improvement and the reduction of daytime fatigue.*

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*Discussions of this issue usually give priority to all-night physiological data. According to Rechtschaffen and Kales (51) criteria, the sleep record is scored in an "all-or-none" fashion. The record is divided arbitrarily into 30-second segments and one rating (awake; non-REM stages 1, 2, 3, 4; REM; gross body movement) is given to the entire page. An entire page is given a specific rating (e.g., awake, Stage 1) if 50% or more of the page shows the brainwaves characteristic of that sleep stage. Obviously, the person could be vacillating between Stage 1 and awake, but the record could be scored asleep according to this criterion, if 50% or more of the record showed 3 to 7 cps activity. Further, sleep onset is scored when three successive pages of Stage 1 sleep are found. Again, it is conceivable that a person could show sleep onset physiologically but not experience that sleep has occurred.

Table 2. Evaluations of Behavioral Strategies for Treating Insomnia Using All-Night Sleep Recordings

Study	*Treatments*	*Results*	*Follow-Up*
Poser, Fenton, and Scotton (49)	140 trials of classical conditioning (N = 1)	No evidence of conditioned sleep.	No follow-up reported.
Traub, Jencks, and Bliss (58)	Autogenic training prior to bedtime (N = 7)	3 subjects decreased latency to sleep onset; 2 subjects increased slow-wave sleep; 2 subjects showed no change	No follow-up reported.
Feinstein, Sterman, and MacDonald (21)	1. Sensorimotor rhythm feedback (13.5 cycles per second—sensorimotor cortex) (N = 4) 2. Occiptal feedback (10 cycles per second) (N = ?) 3. Central feedback (10 cycles per second) (N = ?) 4. Central feedback (15 cycles per second) (N = ?) (25 sessions)	4 SMR feedback subjects decreased latency to sleep onset and gross body movements increased REM sleep, and improved cyclicity of sleep stages. Other groups did not show similar improvement.	No follow-up reported.

Freedman and Papsdorf (23)	1. Frontalis EMG biofeedback (N = 6) 2. Progressive Relaxation (N = 6) 3. Placebo control (N = 6) (6 sessions)	Biofeedback and Progressive Relaxation groups reduced latency to sleep onset. No other sleep EEG changes significant.	At 2 months, no differences between groups in latency via self-report.
Borkovec and Weerts (13)	1. Progressive Relaxation (N = 11) 2. Placebo (N = 11) 3. Wait control (N = 11) (4 sessions)	All groups reduced latency to Stage 1 at week 3; Progressive Relaxation group showed significantly less within group variance at week 4. No other sleep EEG changes were significant.	At 1 year, self-reported mean values for Progressive Relaxation subjects showed maintenance of improvement in latency to sleep onset; mean values for placebo subjects showed return to baseline; variances not reported.
Hauri and Good (28)	Frontalis EMG feedback (N = 10) (13–67 sessions)*	No reduction in latency to sleep onset; sleep efficiency measured (79.9% to 85.5%, $p < .04$); percent delta sleep increased (5.0% to 11.1%, $p < .05$).	No follow-up reported.
Hauri and Cohen (27)	1. SMR biofeedback (N = 10) 2. Frontalis EMG biofeedback (N = 10) 3. Frontalis EMG + theta biofeedback (N = 10) 4. No treatment control (N = 7) (15–60 sessions)†	SMR group increased sleep efficiency and minutes Stage 2 sleep; EMG + theta group increased minutes of REM sleep.	No follow-up reported.

*Subjects continued with training "until both technician and patients felt maximum benefits had been achieved" (p. 222).
†Subjects were trained to criterion or until they showed no more progress in 10 sessions.

PROBLEMS AND TREATMENTS: THEORETICAL PERSPECTIVES

It would be useful to reflect on central theoretical and treatment issues before going ahead with research and development. Two divergent (and perhaps equally plausible) strategies are open. One involves continued study of variations in current approaches (e.g., progressive muscle relaxation) in the hope that subtle variables, such as the timing and intensity of treatment, might provide the key for improving treatment outcomes. Studying individual difference variables is consistent with this strategy; persons with specific sleep complaints and certain characteristics can be matched with specific treatments.

The second approach recognizes the somewhat limited efficacy of current procedures—hypnotic medications and behavioral techniques—and questions the basic presuppositions underlying these methods. Developing other ways of viewing the problem of insomnia might conceivably provide improved foundations for more clinically effective methods.

The Hyperarousal Hypothesis

A common hypothesis forms the basis of the procedures reported in Tables 1 and 2: Disturbed sleep results from hyperarousal, especially at bedtime, due to interoceptive and exteroceptive stimulation which maintains wakefulness (37). Disturbed sleep occurs when these stimulations play on the central nervous system to maintain the waking state. Monroe's (46) seminal study comparing good and poor sleepers is often cited to support the hyperarousal hypothesis. He found that poor sleepers not only slept less well but also showed heightened arousal on a variety of physiological variables (e.g., heart rate, respiration rate) at bedtime and throughout the night. Other interpretations of these data are possible. For example, one might consider a phase lag conceptualization for insomnia. The process view of sleep suggests that sleep must be viewed in the context of the total waking REM/sleep non-REM/sleep cycle (20). Many bodily functions (e.g., respiration rate, body temperature) oscillate on an internally generated daily schedule—the circadian rhythm. Sleep disturbance may represent the desynchronization of some of these rhythms. In Monroe's data, body-temperature curves of good sleepers were observed to fall when they retired and temperatures rose toward morning. By contrast the poor sleepers' body temperatures fell at bedtime but peaked after five hours and then began falling again. Thus, disturbances in sleep throughout the night could represent abnormal fluctuations in rhythmic processes which lead to disturbed sleep.

It is also possible that central to the complaint of insomnia is marked variability in sleep from night to night. Poor sleepers, in contrast to good sleepers, show widely varying values on most sleep parameters from one night to the next (cf. 18). This marked variability may dispose certain poor sleepers to interpret their sleep as a highly unpredictable phenomenon. This uncertainty might lead poor sleepers to attribute their highly variable sleep to factors beyond their control. Thus, a person's *conceptualization* of the problem, perhaps combined with worries about general, physical, and psychological health, may be central to the complaint.

Finally, poor sleepers themselves show widely varying sleep patterns. In the Monroe (46) study good sleepers averaged 7 minutes to fall asleep and poor sleepers averaged 15 minutes; standard deviations were 7 and 15 minutes respectively. Poor sleepers averaged 73 minutes awake after sleep onset (S.D. = 60 minutes). Note the variability. The standard deviations often match the means.

Three implications seem clear: 1) Variations of progressive muscle relaxation training may help *some* persons but for reasons other than those proposed by the hyperarousal model; 2) treatments based on other theoretical models may be needed for the various kinds of disturbed sleep that are experienced; and 3) the marked variability found among poor sleepers suggests that treatment generalizations about persons complaining of insomnia may be premature.

Multiple Causes

The experience of insomnia is related to a variety of causes. Understandably, any major change or disruption in a person's life, whether brief or prolonged, can markedly influence sleep. Among physiological antecedents there is sleep apnea, in which the person experiences a prolonged period during sleep when no breathing occurs (24); nocturnal myoclonus—periodic leg twitches associated with alpha arousal throughout the night (44); restless legs syndrome—aching sensations in the legs (44); various organic problems, such as nutritional disorders, thyroid dysfunction, brain tumor, liver or kidney disorders, hypermetabolic states, and cardiovascular disease (60); and narcolepsy can lead to the experience of sleepiness and to the belief that insomnia is the cause (64). Insomnia can also be a consequence of certain drugs: Stimulants, such as caffeine in coffee, cocoa, cola beverages, and tea; alcohol when used chronically; prescription drugs for medical disorders; and some hypnotic medications, e.g., barbiturates, taken to relieve insomnia.

Beyond these problems, a variety of psychological and environmental difficulties can operate to maintain poor sleep (17): A person's perception of the problem, his beliefs about its causes, his perceived ability to take successful action to solve his difficulty; chronic depression, negative affect and low self-esteem (cf. 19, 33, 60); emotional arousal, e.g., anger, stress, exposure to stressful stimulation, and general arousal at bedtime; excessive worrying, excessive thinking, perfectionistic standards, frequent self-critical thoughts, and deficient problem-solving skills (cf. 20); a variety of other variables whose relationship to sleep remains elusive or unknown, e.g., diet and eating patterns, physical exercise, work environment, occupation, coronary prone behavior patterns, and sleep routines and rituals (cf. 12).

Insomnia Is Not A Disease

Insomnia is neither a unitary disease nor a specific disorder. Rather, as a broad descriptive term, it refers to a variety of complaints arising from a variety of sources. Therapeutic progress can sometimes occur when assumptions are simplified and the task becomes one of finding the treatment most effective for the widest variety of persons. Efforts at simplification may be premature, coming at the expense of effective treatment. Most evaluations of behavior therapy with insomnia have involved the application of a single treatment strategy such as progressive muscle relaxation training. It seems unlikely, however, that any single strategy will be effective in providing clinically significant change for many persons, given the marked heterogeneity in persons' complaints and the factors maintaining each person's individual patterns of disturbed sleep (cf. 26, 60). It may be necessary to teach persons a variety of skills and strategies related to the antecedent conditions specific to their sleep problem (17).

Lasting clinical change may require that persons become skilled in using the processes of behavior analysis and problem solving so that they can, on their own, analyze conditions related to poor sleep, try out and evaluate various solutions, and maintain gains made during intensive treatment contact.

This perspective might seem to demand only one logical alternative: Because each person is different, each cause can be treated only on a completely individualized basis. Both alternatives (treat all cases the same or treat all cases idiosyncratically) fail to recognize the middle ground suggested by social learning theory. Human behavior is expected to vary from situation to situation and from time to time, due to complex interactons of a person's behavior with environmental and cognitive variables (4, 45) (cf. Figure 2). Using this admittedly more complicated perspective, a social

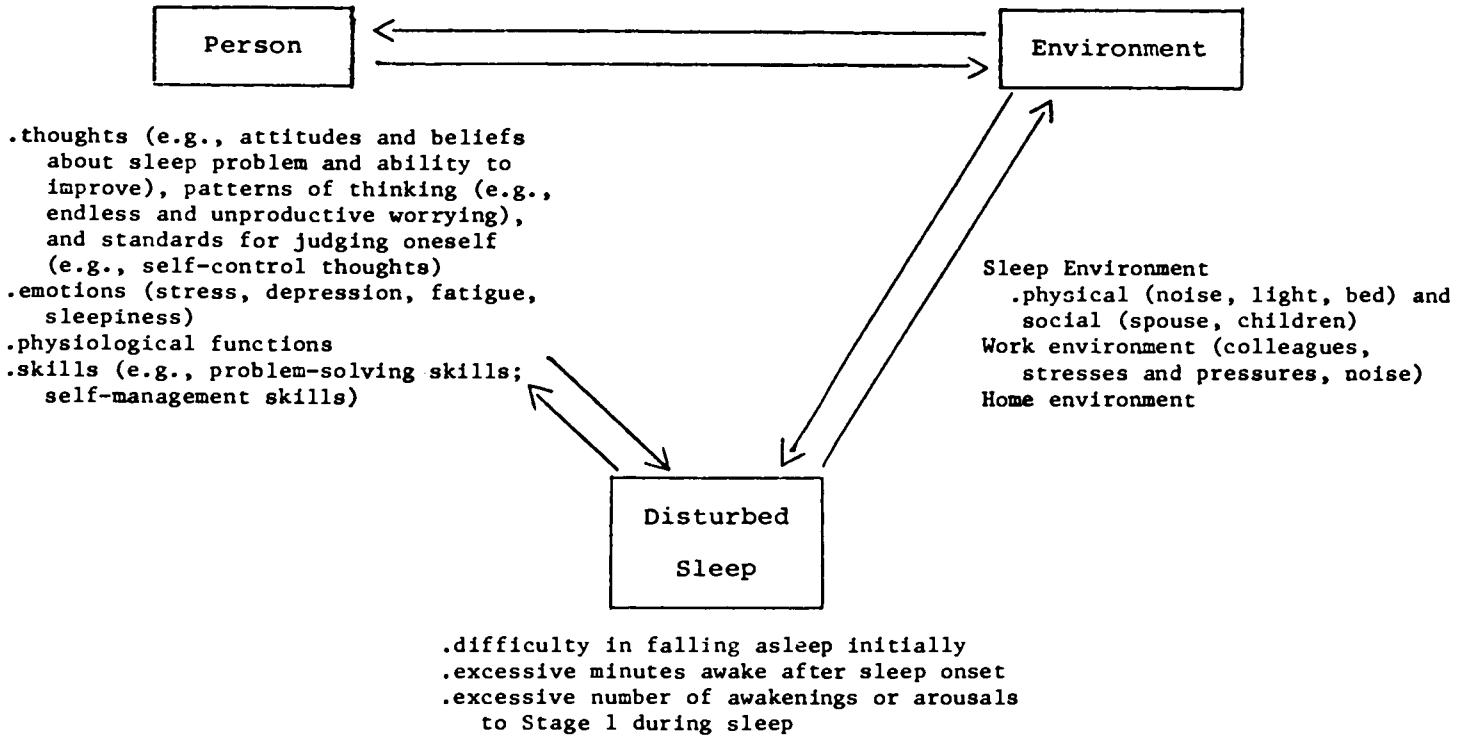

Figure 2.

The person, the environment, and disturbed sleep in a reciprocally interacting system. Any one element in the system can influence all others. Disturbed sleep, for example, can result from and also lead to problems in environment (e.g., arguments with spouse) or adverse personal reactions (depression, stress). Adequate treatment requires assessing the total system and planning a strategy to insure that all elements of the system interact to promote positive outcomes.

learning approach encourages a methodology for assessing specific interacting conditions operating in the single case, development of a basis for moving from assessment to specific treatment, and a rationale for assessing the generalized impact of treatments (cf. 40, 61, 63). Contemporary social learning theory has moved beyond the conditioning/cure paradigm (cf. 4, 55).

Bandura (5), for example, has proposed that therapies in general may be effective to the degree that they enhance a person's ability to perform specific goal-directed behaviors *and* a person's belief that the specific behaviors can be performed when needed to bring about meaningful change. Thus, if the person's insomnia is sustained by excessive worrying at 2 A.M., a client is never cured in the traditional "absence of disease" sense; rather, the client learns a variety of methods to use when needed to manage sleeping problems as they emerge in particular situations.

THE SELF-MANAGEMENT MODEL

Following the social learning perspective, our goal has been to develop treatment programs that permit assessment of a wide variety of variables potentially related to the person's poor sleep, use this assessment in the differential application of treatment strategies, and enhance the client's sense of personal efficacy for controlling those conditions associated with poor sleep. Our guiding assumption has been that because insomnia will never be cured, it is necessary to teach persons self-management skills so that they will learn and practice new living habits as well as skills for managing poor sleep in the future.*

Behavioral self-management implies more than a set number of techniques. Developing skills of personal mastery over the many influences in one's life requires a method for analyzing personal actions and environments, as well as the actions and the environments of others, along with a process for translating such analyses into specific change plans (cf. 56, 57). The difference may be characterized as follows: a person experiencing bacterial pneumonia can be given powerful antibiotics. These drugs will usually be effective regardless of the person's knowledge of their mechanisms

*Improving our conceptualizations for daytime impairment is also essential. How do concepts such as sleepiness and fatigue differ; how are they most usefully operationalized and measured? Measures of daytime performance have been primarily used in performance and simulation tests (55). We have been exploring a variety of alternative observational and self-report methods for assessing daytime functioning in the natural environment while persons are engaged in everyday routine tasks.

Table 3. A Self-Management Approach to Treating the Complaint of Insomnia

	Assessment (2–4 weeks)	All-night sleep recording, physical and neurological examination, psychiatric examination, psychometric evaluation.
		Personal, medical, sleep, and medication history.
		Sleep environment, chronic stress and tension, work demands and environments, depression, other factors (e.g., eating habits, caffeine assumption, daily schedule, exercise).
		Spouse and family interview; spouse/roomate behavioral checklist.
Concurrent	Relaxation Training (3–4 weeks)	Progressive muscle relaxation. Self-hypnosis (cognitive focusing skills). Clients use cassette tapes for home practice.
Concurrent	Cognitive Restructuring (2–3 weeks)	Basic information about sleep and sleep processes; practice using thoughts conducive to good sleep; analysis of beliefs about sleep problem; analysis of beliefs about ability to learn new skills and change behavior associated with sleep problems.
Concurrent	Problem-solving (2–4 weeks)	Analysis of behavioral, environmental, and cognitive conditions related to poor sleep; design individualized strategies for improving disturbed sleep; evaluate and modify as needed.
	Maintenance (3–6 months)	Spaced sessions to note progress, promote continued use of strategies, and identify and change other behaviors, thoughts, or environments still related to poor sleep. Increased responsibility placed on client to analyze and propose solutions, and evaluate progress.

of action. Further, these drugs do not require the person's active involvement other than to comply to the prescribed regimen for taking the medication. Physical relaxation, biofeedback, and similar procedures are sometimes conceptualized and administered in the same way. They are expected to work in a fairly automatic fashion to cure the problem behavior.

By contrast, the self-management model recognizes that a change in any behavior can seldom be achieved by means of an automatic effect produced

by any treatment technique. Instead, meaningful change which is maintained requires persons to possess a variety of skills for assessing and managing a myriad of environmental and cognitive events related to poor sleep, including the belief that they can use these skills successfully when needed (cf. 5, 17).

An overview of the treatment program we have developed to date is presented in Table 3. In this program, assessment methods seek to determine the particular nature of the sleep disturbance, to rule out insomnia secondary to a physical disorder, and to examine environmental, cognitive, and behavioral variables potentially related to poor sleep. Following this, clients are taught physical and mental relaxation skills as well as cognitive restructuring strategies. During these sessions, clients are reminded continually that these skills will be effective only if used and practiced consistently.

During cognitive restructuring, clients are introduced to the notion that solving a personal problem often requires learning to view events from different perspectives, and to practice replacing specific maladaptive thoughts and images with more adaptive and sleep-enhancing ones. Some attention is given to "means-ends" thinking and "contingency rules," especially in terms of examining beliefs about current behavior patterns in relation to short- and long-term life goals.

Problem-solving training is designed to provide clients with skills to analyze and change those conditions consistently related to poor sleep. Anticipating future problems is also highlighted. The maintenance sessions provide encouragement for continued use of these strategies and help ensure that the client has learned and can use a variety of self-management skills.

TREATING DRUG-DEPENDENT INSOMNIA: TWO CASE STUDIES

Drug-dependent insomnia is maintained by chronic use of hypnotic medication (cf. 33, 34). Withdrawal can be difficult, and even when accomplished, provides no guarantee that sleep will improve. Recently we have withdrawn clients from medications and used self-management training to improve their sleep. Two case studies are presented here to illustrate how treatment has been implemented.

Two middle-aged women, who had suffered from insomnia for a number of years, had experimented with a variety of sleep medications to improve their sleep (cf. Table 4). Mary was ingesting 600 mg of methyprylon per day initially while Paula was taking 15 mg of diazepam with 12 to 15 oz of dry wine (12% alcohol) at bedtime.

Table 4. Treatment Programs for Two Persons with Drug-Dependent Insomnia

	Mary		*Paula*
Description	Female, 53 years old, insomnia for 25 years		Female, 37 years old, insomnia for 20 years
Pretreatment Drug	600 mg methyprylon + 30 mg chlordiazepoxide at bedtime; 30 mg chlordiazepoxide t.i.d.		15 mg diazepam + 12–15 oz wine at bedtime
Withdrawal Plan	Sleep stabilized at 300 mg methyprylon + 30 mg chlordiazepoxide at bedtime; methyprylon first withdrawn by 100 mg per week; chlordiazepoxide withdrawn by 10 mg per week		Sleep stabilized at 30 mg diazepam which was decreased by 2.5 mg per week; drinking wine at bedtime was stopped
Treatment Sequence	1. Assessment (2 weeks) 2. Relaxation Training (3 weeks) and Cognitive Restructuring (2 weeks) 3. Problem-solving (4 weeks) + Maintenance (Cue-controlled relaxation)	Concurrent with withdrawal	1. Assessment (2 weeks) 2. Relaxation Training (2 weeks) and Cognitive Restructuring (1 week) 3. Problem Solving (6 weeks) + Maintenance (Anticipation training; activity planning)

Treating drug-dependent insomnia requires that persons, in addition to learning self-management skills, also withdraw from a drug at a rate that is safe, minimally disruptive to sleep, and leads to the least amount of discomfort. We prescribed relatively safe hypnotic drugs at a dosage that ensured sleep (cf. Table 4). When self-management training began, the drug was withdrawn at the rate of one therapeutic dose per week.* Clients were informed about the mechanisms of drug action and warned about side effects and reactions to withdrawal. Treatment continued until they were

*The exact amount of weekly withdrawal will vary across cases. In some, cases, ½ of one dose per week is tried. The key factor is the observed effects of the reduction to consider on the person.

completely withdrawn and sleep was maintained at a relatively acceptable level.

Specific withdrawal plans and treatment programs for Mary and Paula are presented in Table 4. Note that the treatment was varied for each client within the general program format. Drug dosage was reduced according to the rate at which the drug could be withdrawn and reasonably stable sleep maintained. Similarly, each client learned relaxation and cognitive focusing skills following assessment. However, the length of training varied in each case according to the rate at which these skills could be mastered and used successfully. Finally, the program became completely individualized during the problem-solving phase.

Mary taught 7th-grade English in a lower socioeconomic neighborhood. Because she attributed much of her poor sleep to the stresses imposed by students and administrators, she learned cue-controlled relaxation during problem-solving (cf. 17). Paula, by contrast, experienced frequent mild to moderate depression. Anticipation training (cf. 2) was taught as one method for managing her depressive experiences.

Two consecutive all-night sleep recordings were conducted on three different occasions: prior to treatment, two weeks following final withdrawal, and six months following final withdrawal. The results are presented in Table 5. The drugs had predictable effects. Both slow-wave and REM sleep were suppressed before treatment and increased to normal levels following withdrawal. Drug effects are also reflected in delayed onset to the first REM period, which was reduced following withdrawal.

A second finding was quite surprising yet significant. Both clients *reduced* total sleep time but *increased* sleep efficiency. In addition, Mary experienced fewer arousals to wakefulness or Stage 1. Both reported needing less sleep, feeling more refreshed during the day, and having increased energy in the morning.

SOME DIRECTIONS

In using the self-management model for treating insomnia the adequacy of both traditional medical remedies (e.g., hypnotics) and standard behavioral alternatives (e.g., muscle relaxation training) have been questioned. The self-management model views insomnia from a broadened perspective: Insomnia consists of a variety of complaints related to a variety of maintaining conditions for each person. The present scenario, however, is far from complete. At least five research and clinical issues require attention.

First, crucial empirical tests are needed. Although the cases discussed here were severe and chronic, the successes point at best to the promise of

Table 5. Results of All-Night Sleep Recording Data While Subjects Were on Drugs, During Withdrawal, and at 6 Months Following Treatment

	MARY			PAULA		
	Pretreatment	*Withdrawal*	*6 Months Follow-up*	*Pretreatment*	*Withdrawal*	*6 Months Follow-up*
Total Sleep Time	454.0	310.5	336.0	399.7	327.0	375.0
Latency to Sleep Onset	41.5	14.0	5.5	24.7	32.0	3.0
Minutes Awake after Sleep Onset	39.0	12.5	20.0	27.6	22.5	26.5
Sleep Efficiency	84.7	91.1	93.1	86.9	83.3	92.1
Number of Arousals	34.0	39.5	20.0	27.6	22.5	26.5
Pct. Stage 1	9.5	10.0	8.8	10.5	9.9	11.1
Pct. Stage 2	69.2	36.8	56.0	69.8	51.1	31.4
Pct. Stage 3	2.5	11.6	6.0	0.6	10.1	17.6
Pct. Stage 4	0.0	21.3	10.7	0.0	5.8	15.6
Pct. Stage REM	18.1	19.0	18.0	14.9	21.4	24.3
Onset to REM 1	129.0	75.5	66.5	209.3	98.5	123.0

Data based on the average of two consecutive laboratory sleep recordings.

the approach. Replications are needed along with rigorous experimental tests of procedures to determine their relative efficacy in comparison to other methods. The possible contribution of attention-expectancy effects and other factors conceivably related to treatment outcomes needs to be assessed. For example, in the two cases reported here, it is possible that a systematic drug-withdrawal regimen offered in a warm and supportive doctor-patient relationship *without* training in self-management skills might produce comparable results.

Second, measurement strategies require further development and increased sophistication. We have conjectured, for example, that successful treatment involves the enhancement of problem-solving skills and expectations about personal abilities to perform important therapeutic actions when needed. Major problems exist in assessing problem-solving skills as well as changes in efficacy about specific treatment procedures. In addition, the validity and reliability of all-night recordings deserve scrutiny. For example, we have found that not enough sleep recordings are made and that sleep patterns are consequently inaccurately characterized (18). We have also stressed that impairment of daytime functioning is central to the complaint of insomnia. Theoretical and empirical studies are needed to develop more adequate strategies for conceptualizing and measuring daytime experience. In the two cases presented here, we relied on client self-reports along with all-night recordings to document these changes. To date, we have found no reported studies using independent observers to document patterns and changes in the *daytime* behaviors of chronic insomniacs.

Third, hypnotic medications are easy to administer and lead to short-term reductions in the complaint. By contrast, self-management treatment is more complex and time consuming; its effects are less immediate. Alternatives are needed in delivering treatment in a way that is less time consuming, especially if they are to be used by physicians and other health professionals. For example, behavioral psychologists and physicians might work collaboratively in the delivery of treatment and in the development of cost-effective delivery systems. It also seems reasonable that audio and videotape material could be used by paraprofessional staff to provide training in basic skills, such as physical and mental relaxation (cf. 17).

Fourth, the possibility of self- and peer-administered care and treatment merits study (cf. 43). Some persons might be able to learn to self-assess and self-manage their own sleep-related behaviors without direct contact with a highly trained professional. A self-administered strategy could be more effective in enhancing a person's attributions of personal efficacy—the conviction that one can successfully do certain things to improve sleep. Peer-administered treatment programs might also prove useful. Local community-based self-help groups could be established to instruct and provide ongoing social support for disturbed sleepers.

Fifth, primary prevention concerning sleep-related problems and educational curricula for adolescents on sleep deserves attention (cf. 50). Such programs could possibly reduce the prevalence of sleep disorders among young adults and the subsequent high use of hypnotic medications. Young persons could be taught fundamental information about sleep and methods for preventing daytime sleepiness and nighttime sleeplessness (cf. 36). Learning about the detrimental effects of altering sleep-wakefulness cycles, for example, could help many persons avoid disruption in sleep, performance, and mood during the day (cf. 1, 54). A self-management-oriented curriculum could be offered in secondary schools and community colleges to teach the specific skills discussed earlier in this chapter. Such efforts might reduce the amount of hypnotic medications and alcohol used as a sleep aid, and encourage consideration of nondrug alternatives. Further, the existence of educational curricula along with self-help programs would be a step in the direction of encouraging persons to accept more personal responsibility for their own health and welfare (39).

A complaint as varied and as complex as insomnia merits a broad and open perspective to the possibilities—conceptually, methodologically, and clinically. Helping persons to help themselves deserves our attention, especially in reducing reliance on hypnotics.

ACKNOWLEDGMENTS

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Introduction To Psychoneuroendocrinology

A new field, psychoneuroendocrinology, has developed subsequent to the discovery of many behavorially active neurotransmitter substances in the central nervous system. Many of these compounds, collectively called neuropeptides, are short chains of amino acids joined together by peptide bonds. Similar substances and non-peptide compounds such as steroids which are produced throughout the body, have a wide range of hormonal properties. In addition, modified fatty acids, called pheromones, appear to be a distinct, although yet uncharacterized, group of hormonal compounds. A large number of these "neuro-hormonal" substances have behavorial effects. For example, a few micrograms of lutinizing hormone releasing hormone (LHRH) injected into a rat induces sexual lordosis; fragments of ACTH ($ACTH_{4\text{-}10}$, $ACTH_{4\text{-}7}$) markedly change the characteristics of passive and active avoidance learning in rats; and fragment $ACTH_{1\text{-}13}$ appears to facilitate attention or motivation. Vasopressin-like hormones of the posterior pituitary also have behavioral effects and an apparent effect on memory.

The possibilities for interactions between neuropeptides and behaviors are formidable. A recent review (3) lists 10 endogenous brain hormonal substances and 10 anterior and two posterior pituitary peptides; and points out the possibility that an almost infinite number of similar compounds could be endogenously synthesized from various combinations of amino acids within the brain itself. In addition, dozens of peptide-like substances that are behaviorally active are found in the gastrointestinal system, in endocrine glands, and in skeletal muscles. In most cases, a full behavioral characterization of these compounds has not been carried out and their functional significance is unknown (1, 2). Early research has demonstrated that both naturally occurring and artificially administered neuropeptides have marked effects on the acquisition, extinction, and expression of various behaviors. Many of these compounds appear to elicit behaviors or change drive states without learning or behavioral expression. For example, the gut hormone, cholecystokinen, and its analogues may induce behavioral satiety in animals; and injection of miniscule quantities of betalipoprotein

fragments, the endorphins or enkephlins, which are formed in normal brain tissue, induces various stereotyped behaviors in experimental animals. Although the significance of these compounds in normal behavioral states is unknown, their presence and the existence of receptor sites which when activated artificially produce behavioral changes may suggest that they play a key neurophysiological role in the expression of behavior.

In Chapter 12, Woods and Burchfield discuss the conditioning of endocrine responses. Using respondent techniques, many endocrine systems have been conditioned to environmental stimuli; their conditioning properties are somewhat different from other autonomic responses. This ability to be conditioned is not surprizing, since most hormones are either secreted by neurons or have secretory cells which are directly influenced by neural projections. This type of conditioning process was demonstrated when insulin shock was used to treat schizophrenic patients. Clinicians treating these patients observed that after several treatments, their patients would begin to show insulin shock-like behavior when they were wheeled into the treatment room to receive their injection of insulin. When they analyzed the patients' serum glucose, they found that the serum glucose level began to drop when they were placed in the environment in which they normally would have received a coma-inducing dose of insulin. It appeared that they had learned to respond to the treatment room by secreting insulin, and that this response was strong enough to produce a coma-like state. Woods and Burchfield make the point that conditioned insulin secretion could be an adaptive response or, on the basis of conditioning, could produce a reactive hypoglycemia with symptoms indistinguishable from an anxiety attack or a multitude of other psychiatric or physiological disease states. They point out that many types of hormones, notably those connected with stress, reproduction, and parenting can come under respondent control, and that the possibility exists that operant control can also be established. In a final part of the chapter, they speculate about pheromones, the interpersonal hormones about which very little is known.

The discovery of naturally occuriing behaviorally active hormonal substances and the observation that most hormonal systems can be conditioned at least by respondent techniques are most exciting. It can be speculated that the conditioned release of hormones could provide a breakthrough in many areas in medicine and psychology. For example, if the secretion of growth hormone could be controlled, an alternative to very expensive replacement therapy for growth hormone deficient children would be available. Similarly, individuals deficient in insulin might be maintained for some time by conditioned insulin response rather than exogenous insulin administration. More speculatively, if the naturally occurring ACTH fragments, or the vasopressin fragments that appear to enhance memory and

change the properties of learned avoidance responses are conditionable, it might be possible to improve the memory of disabled or normal individuals, enhance the retention of learned material, or possibly improve intelligence either by conditioned release or exogenous administration.

Observations in this field are phenomenological, and the technology is not available to carry out much of the research suggested by this introduction, or to implement a treatment program. Assays for many of these hormones are extremely time consuming, occasionally taking weeks or months. Without immediate feedback, their secretion could not be conditioned by any known method. However, technologies do develop, and the implications for this type of development are very interesting.

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CHAPTER 12

Conditioned Endocrine Responses

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INTRODUCTION

The secretion of most hormones is of one of two types. The first is a tonic or basal rate of secretion that occurs during normal functioning and in the absence of specific exogenous influences. Fluctuations of this basal hormone secretion due to endogenous influences that occur in synchrony with diurnal rhythms are included in this category. The second type are increments or decrements from this basal secretion in response to exogenous homeostatic or emergency needs. It is probably this latter category which is more adaptable and therefore more susceptible to modification through learning or experience.

LEARNING AND ENDOCRINE SYSTEMS

We shall begin with the qualification that most reported instances of conditioned endocrine changes are the result of classical or Pavlovian conditioning. We know of no reported instances where operant techniques have been used. Reinforcement requires rapid feedback and endocrine assays typically require too much time. On the other hand, since most autonomic responses are subject to modification through reinforcement (66), it seems reasonable to assume that endocrine secretions may be likewise operantly controlled.

The process of learning or conditioning implies a degree of neural influence over a system. The logical extension of this is that endocrine systems

which can be conditioned are subject to neural influences, a premise supported by current literature. Most hormones are either secreted by neurons, e.g., all of the hypothalamic "releasing hormones," catecholamines, vasopressin, and melatonin, or are influenced directly via neural projections onto the secretory cells as in the case of insulin and most of the gut hormones, or indirectly via the hypothalmo-hypophyseal axis as in the case of gonadal and adrenocortical steroids, and thyroid hormone. One popular theory which describes embryological origin of many endocrine tissues suggests that these hormone-secreting cells actually consist of modified neurons of neural crest origin (71, 72). After differentiation, these cells retain many properties not observed in nonneural tissues. The theory states that as certain ectodermal tissues begin to differentiate into neural crest cells, some of these cells migrate in the developing embryo to reach a final destination within other supporting tissues. These cells later comprise the peptide hormone secreting cells of the anterior pituitary, the thyroid, the parathyroids, the hormone secreting cells in the digestive tract, the endocrine pancreas etc. This theory proposes that the secretion of peptide hormones be conceptualized as a neurosecretory process analogous to the secretion of catecholamines from the adrenal medulla, which is also modified neural crest tissue. Further support for the concept that much of the traditional endocrine system may actually be a branch of the nervous system is the finding that many cells that secrete hormones have electrical properties including action potentials that are analogous to those observed in brain neurons (5,10,28,65). This close link between the endocrine and the nervous systems renders more plausible the contention that the secretion of specific hormones can be brought under stimulus control through learning.

Any meaningful discussion of conditioned hormone secretion must provide evidence that the secretion of specific hormones can be modified by neural activity. Otherwise, hormone changes elicited by specific stimuli in a learning context can be more parsimoniously attributed to other responses elicited by the stimuli and not a direct consequence of the conditioning process per se. For example, if animals are fed only once a day their diurnal clocks entrain upon the feeding time, and the secretions of several hormones become synchronized with feeding time. It should not be concluded that the secretion of each specific hormone has been conditioned unless other controls are included. Only one behavioral response may actually be conditioned, and this response may have the ability to influence a spectrum of endocrine responses secondarily. Such changes of hormone secretion are not, strictly speaking, instances of conditioned endocrine secretion. In many of the references cited below, proper controls were not included, and only inferences about possible conditioned hormone secretion can be made. In the discussion, we will indicate those experiments where appropriate controls were included.

There are several additional interpretive problems that should be mentioned before proceeding to the specific literature. The first has to do with the strictness of one's definition of learning. Very few of the cited experiments included adequate controls that would eliminate alternative explanations such as pseudoconditioning or sensitization. With a strict behavioral definition of learning, these studies are at best inconclusive and suggestive. On the other hand, if one assumes the broader position that learning reflects the ability of an animal to benefit from experience and that specific procedural paradigms and temporal associations are not critical, then a more realistic, albeit in a theoretical sense more limited, view of the capabilities of the endocrine system is possible.

An analogous problem relates to situations or circumstances in which "conditioned" changes of hormone levels have been observed. Most reported experiments describe an instance of altered hormone level, or inferred hormone level, in a group or an individual, and a natural conditioning mechanism is invoked after the fact to explain the findings. The rationale for including these reports in this review is that similar findings have frequently been observed when specific experimental protocols have been followed, suggesting common underlying mechanisms.

A final problem concerns the reported dependent variables. Many researchers have inferred changes of hormone levels from observed changes in other physiological parameters. For example, changes of catecholamines have been inferred from changes in heart rate or blood pressure, changes in insulin levels from changes in blood glucose, changes in oxytocin secretion from milk letdown, etc. In some of these instances, the measured variables were legitimate and often included bioassays for the hormones; in others, the required inferences strained credibility and are subject to ambiguity. Again, the rationale for including these experiments in this discussion is that similar experiments have occasionally been reported in which specific hormone assays have been used and similar results attained.

Therefore, in the review that follows, we have included a number of experiments that used questionable research designs and/or questionable dependent variables. Much of the work has been done with animals and can be extended to humans only by inference. As a whole, we feel that the literature supports the conclusion that most endocrine systems can be brought under stimulus control through learning.

INSULIN AND OTHER METABOLIC HORMONES: A MODEL SYSTEM

Insulin was the first peptide hormone for which a specific radioimmunoassay existed. This fact, coupled with the importance of insulin

clinically in the treatment of diabetes mellitus, has resulted in insulin being the most extensively studied hormone. This is also the case in the conditioning literature, and so insulin can serve as a model for conditioned endocrine responses.

For many years, there have been reports that changes of blood glucose level could be conditioned in dogs (3, 49), rats (2, 87, 105), and humans (54). A review of this literature has recently appeared (104). Specifically, when the administration of drugs that elicit hypoglycemia is associated with arbitrary stimuli, typically odors or sounds, over a number of trials, these stimuli begin to elicit hypoglycemia in the absence of the drug. This phenomenon, which is called conditioned hypoglycemia, has been the subject of many Pavlovian conditioning control procedures including variance of the strength of the unconditioned stimulus (105, 109), the nature of the conditioned stimulus (44, 105), the nature of the unconditioned response (98), the number and temporal parameters of the conditioning trials (2, 41, 105), investigations of extinction (105), and pseudoconditioning and sensitization controls (43). All of these experiments conclude that conditioned hypoglycemia is a true instance of classical conditioning (see 104).

More importantly for the present discussion, conditioned hypoglycemia has been shown to depend upon the conditioned secretion of insulin from the endocrine pancreas (100, 102), and the response can be eliminated when the neural link between the brain and the endocrine pancreas is compromised by vagotomy (4, 97), or the administration of parasympathetic blocking drugs (97). These experiments were among the first to suggest a functional link between the brain and the endocrine pancreas, a link which is now widely accepted (see 89, 106, 107).

The demonstration that insulin secretion can be brought under stimulus control in the laboratory when specific experimental protocols are used foreshadowed the development of a literature which suggests that conditioned insulin secretion may occur naturally in association with eating. Insulin is normally secreted in large amounts during and after a meal to cope with the influx of consumed fuels to the blood. Specific stimuli always associated with the presentation of meals are therefore likely candidates for stimuli that can elicit insulin secretion in the absence of actual food ingestion; i.e., whereas Pavlov originally showed that dogs secrete saliva upon the presentation of stimuli that predict food (70), others have shown that insulin secretion occurs at the same time. This "cephalic phase" of insulin secretion, the subject of several recent reviews (75, 99), has been shown to use the same vagal pathway as the conditioned insulin secretion produced by strict conditioning procedures (56, 92, 99). In addition, we recently demonstrated that the cephalic phase of insulin secretion can be entrained to arbitrary stimuli that reliably predict the presentation of food to rats (110). In

this experiment, rats ate only one meal per day. If either the time of day or an arbitrary odor was regularly associated with food presentation, these stimuli later elicited insulin secretion in the absence of food.

This demonstrated that the cephalic phase of insulin secretion can be brought under stimulus control. Its normal occurrence also suggests that some form of conditioning has occurred, since many food-related cues can elicit it. This response occurs when animals (92) or humans (47) sample the sweet taste of saccharin, when animals eat nonnutritive foods (91), or when they sham-eat (9, 42), and even when humans smell and see a meal but do not consume it (69, 78). Finally, in one experiment, hypnotized humans secreted insulin when they were told they were eating a desirable meal (35).

There are other indications that insulin secretion is easily learned by humans. The laboratory demonstration of conditioned hypoglycemia has most frequently been reported when large amounts of insulin were used as the unconditioned stimulus (104). There are two instances when humans are given large amounts of insulin: In the treatment of diabetes mellitus and in insulin shock therapy for certain psychotic disorders. In both instances, there are reports that stimuli present during the normal administration of the insulin later develop the ability to elicit hypoglycemia and, presumably, insulin secretion in the absence of insulin injections (46, 59). A related observation is that humans receiving regular insulin-shock treatments often become very sensitive to insulin, and that eventually very small doses produce a coma (57, 93). This possibly reflects conditioned insulin secretion that augments the administration of the exogenous insulin.

Insulin secretion is only one of many metabolic responses that animals and people make when they eat; many related hormonal responses are probably also conditionable. Pavlov was the first to demonstrate that the secretion of saliva and gastric juices could become entrained upon stimuli which were associated with food presentation (70). More recently, there have been suggestions that there is a cephalic phase of gastrin (58) and pancreatic glucagon secretion (29). Even metabolic responses that have no obvious link to the nervous system have been entrained to stimuli associated with feeding. For example, Saito and his colleagues have reported that the levels of intestinal disaccharidases and related enzymes become entrained to the time of the day that the rats normally eat (80, 81, 82), although similar enzymes in nondigestive-related organs do not have these fluctuations. Powley (75) has recently reviewed the evidence for various cephalic responses and reached the conclusion that most, if not all, of these responses are conditioned.

Since metabolic responses, especially insulin secretion, are easily conditioned, one must assume that some metabolic advantage accrues from the conditioning process. We can speculate that an anticipatory secretion of in-

sulin as animals begin to eat will tend to minimize the homeostatic imbalance created by the meal. However, the learned secretion of insulin may sometimes be maladaptive. Obese humans have been characterized as overresponding to external stimuli in their environment, especially stimuli associated with feeding (85, 86). If obese individuals oversecrete insulin in anticipation of eating, the increased insulin could exacerbate their obese condition (7, 101, 103, 108).

Since conditioned insulin secretion is presumably an adaptive response, there may be instances when it outlasts its usefulness or becomes inappropriate. Reactive hypoglycemia is a clinical syndrome characterized by oversecretion of insulin when a meal is begun which results in an uncomfortably low blood glucose level (73). Since therapies that disrupt the parasympathetic link between the brain and the endocrine pancreas help alleviate reactive hypoglycemia (12, 74, 95), it is reasonable to conclude that reactive hypoglycemia may represent an instance of an inappropriate conditioned insulin secretion.

CONDITIONING OF STRESS HORMONES

Many experiments have been reported in which one or more stress hormones have been measured in humans or animals following the presentation of stimuli formerly associated with stressful events. In some of these experiments, the stimuli were originally arbitrary and neutral, and specific experimental paradigms associating these stimuli with specific stressors, usually electric shock, were employed. In others, the experimental model of "conditioning" is more tenuous; for example, research on humans has often utilized various psychological stressors like movies or stressful interviews. The assumption is that an association has been formed by the individual in the past, and that certain mental images or situations can lead to an arousal which includes alteration of hormone secretions. It is not at all clear whether these situations reflect learning or an innate stress response. Most hormones secreted when animals are stressed are also secreted to a lesser extent when they are aroused but not necessarily stressed. Sensitization is the phenomenon of overresponsiveness to arousing stimuli. Most experimental examples of sensitization have been produced after the animals have been stressed. Most examples of "conditioned" secretion of stress hormones cited below may therefore be examples of sensitization rather than of learning.

Stress is a somewhat ambiguous term whose meaning varies from change of an organisms's normal routine to specific physiological responses to physical or psychological insults. Depending upon one's definition, many

different hormones may be considered stress hormones. With one major exception, published experiments report changes in only one or two specific hormones or their correlates in response to stress. However, when animals are stressed physically, it has been shown that an entire spectrum of endocrine changes usually occurs (51, 79). Hormones typically included in this group are those of the hypothalamo-hypophyseal-adrenocortical axis (i.e., CRF, ACTH, and the glucocorticoids), the adrenal medulla (epinephrine and norepinephrine), other anterior pituitary hormones and their various hypothalamic releasing hormones (including the growth hormone and thyroid hormone systems), vasopressin, and the hormones of the renin-angiotensin-aldosterone system. It should be clear that specific stressors probably alter specific hormones within this group and that generalizations should not be made from one hormone to another. For example, cold stressors elicit more thyroid response than other stressors (33).

Most stress-hormone experiments have used rats, monkeys, or human subjects, and a variety of different experimental models have been employed. These include simple fear conditioning in which inescapable electric shock is given to animals in a particular setting and the animals are later returned to that setting but not shocked, various kinds of active and passive avoidance learning, conditioned suppression (formerly called conditioned emotional responses or CER's), and conditioned taste aversion. Plasma levels of glucocorticoids, corticosterone in rodents and cortisol in humans, were measured in the majority of published experiments, and the general conclusion was reached that animals have elevated glucocorticoids when they are confronted with stimuli formerly associated with stress.

To cite some specific examples, glucocorticoids are elevated in simple fear conditioning situations (45), in active and passive avoidance situations (14, 17, 19, 76), in conditioned suppression (CER) (6, 13, 39, 61), and during activation of conditioned taste aversions (1, 77). Another series of studies stresses the animals by depriving them of either water or food, which is accompanied by elevated glucocorticoids. Subsequent consumption of water or food reduces these hormone levels. When specific stimuli are associated with this consumption during a learning process, these stimuli cause an apparently conditioned decrease of glucocorticoids (see 23) in the absence of food or water repletion (23, 53, 90).

Although sensitization may well account for many of these results, a couple of the experiments suggest that a true conditioning process may also occur. Coover et al. (23) included a pseudoconditioning control group which did not develop the same response as the conditioned group in a feeding-time and reduction of glucocorticoid experiment. Smotherman et al. (90) found that rats with a conditioned taste aversion to a specific flavor had elevated glucocorticoids when given a choice between that flavor and

water only if they were forced to sample a considerable amount of the flavored solution and not if they mainly sampled the water (see also 53). This implies a specific stimulus-response association rather than a more generalized arousal effect.

In addition to glucocorticoids, animals confronted with a situation in which they were formerly shocked have elevated epinephrine (as determined by a bioassay, 32) and vasopressin (also as determined by bioassay, 24). There is also one report that MSH is elevated in this situation (83). This latter finding has been interpreted in terms of the attention-enhancing qualities of MSH (67, 84). Another study reported that growth hormone levels are increased in monkeys in a situation where they were formerly shocked (14). However, there were no controls for restraint in a primate chair in this experiment, and previous work by the authors had shown that such restraint is stressful.

Perhaps the most extensive series of investigations on conditioned endocrine responses in stress-related situations has been conducted by Mason, Brady, and their colleagues on monkeys. We shall provide only a brief overview of their work here; the interested reader is referred to any of their recent reviews of this work (13, 39, 62, 63,). The basic paradigm involves creating a conflict on the part of the monkeys so that a lever-pressing response which normally produces food, also elicits electric shock. This conflict situation typically lasts for 72 hours, and is accompanied by a spectrum of endocrine changes which the researchers attribute to conditioning. Specifically, they found that glucocorticoids, epinephrine, norepinephrine, thyroid hormone, and growth hormone were all increased during the 72 hours when the conflict was present. Brady found that monkeys repeatedly placed in this conflict situation over a one-year interval gradually reduced their conditioned stress response, or habituation of an unlearned response. Brush and Levine (17) also reported that a conditioned elevation of glucocorticoids extinguished rather quickly when their rats were able to successfully avoid the shock over a one-hour interval. Ferreira et al. (32) reported rapid extinction of an elevation of epinephrine and growth hormone in primates which had occurred in response to a tone formerly associated with shock, when the tone was repeatedly presented alone.

In the experiments by Mason et al., some endocrine responses decreased rather than increased when the monkeys were in the conflict situation. Specifically, estrogen, testosterone, and pepsinogen levels decreased while insulin levels did not change. The suggestion from these experiments is that the conflict situation elicited an integrated spectrum of behavioral and endocrine responses, some of which better enable the animal to cope with the presumed stress and some of which inhibited those systems not needed at

that time. Brady et al. claim that the individual aspects of the spectrum are conditioned individually even though many of them are not independent.

As with all classical conditioning, the results with stress hormones can be interpreted as anticipatory responses that allow some degree of preparedness for the subject prior to the actual occurrence of the stressor. A number of human studies support this position. Graham et al. (36) found that humans given shocks in the lab had elevated catecholamines on entering the lab the next day, and Kurokawa et al. (48) reported that humans told they would see a stressful movie had higher levels of growth hormone before the movie began than during or after it. Similar reasoning might be used to explain the report of Greenwood et al. (37) that a medical student, told he was being injected with insulin and would be hypoglycemic, developed increased cortisol and growth hormone levels even though only saline was actually injected. It appears that there may be more of an endocrine response in anticipation of some stressors than in response to them.

There are individual differences in the anticipatory or conditioned stress response. Not all dogs had a CER in response to a shock chamber (24), and not all humans had elevated stress hormones during a stress interview (48). Most humans told they were receiving insulin but actually injected with saline had no change of hormone levels (37). The differences in individual responsivity that have been observed in experiments of this type have often been attributed to differences in personality variables (8, 13, 16, 40, 52).

An interesting theory has recently been proposed by Leshner that provides a framework for long-term conditioning of some endocrine responses and for the wide range of individual variability usually observed (50). Leshner reviewed considerable data on the hormonal responses to antagonistic encounters by rodents and monkeys and concluded that when two animals meet and establish relative dominance, they have differential endocrine responses. Although both participants have elevated glucocorticoids as a result of the encounter, the response is greater in the subordinate animal or "loser." In addition, there is a more prolonged suppression of reproductive hormones in the loser. Leshner suggests that future encounters by the loser of the original encounter are more likely to result in submissive behaviors and secretion of related hormones as a result of the earlier experience. This theory suggests that a form of endocrinological imprinting occurs during the development of animals that dictates the responses of their endocrine systems later in life. In adaptive terms, the increased tendency to submissiveness of former encounter losers renders them less subject to attack and therefore more likely to survive. Their reproductive endocrinology tends to be chronically suppressed unless they have no competitors. To generalize to humans would be inappropriate, but it is tempting to speculate

that some analogous mechanism might account for the differential responsiveness of the stress-endocrine system within individual humans.

CONDITIONING OF REPRODUCTIVE AND PARENTING HORMONES

Although considerably fewer experiments have dealt with the possibility that reproductive hormone secretion could become conditioned, there are sufficient experiments to suggest that the general pattern documented above exists in this case as well; i.e., when it is advantageous for animals or humans to anticipate certain events which elicit a change in hormone secretion, there is a high likelihood that they can learn to secrete the hormones in response to stimuli that reliably predict those events. Very little work has been done on the effects of experience on gonadotropins or gonadal steroids. One area of research in which the possibility of conditioning has not been explored is the secretion and effects of pheromones. Phermones are species-specific volatile chemicals, often very similar chemically to hormones, which are secreted from the body and which elicit responses from other members of the same species. These compounds act as intersubject hormones activating the olfactory and gustatory receptors (31, 55), and in some instances actually entering the bloodstream of the recipient animal and functioning like traditional hormones (11).

There are indications that both the rate of secretion of reproductive pheromones and the response to them by adult animals varies as a function of experience (15). Although it seems certain that maturity plays a large role in these phenomena, the fact that specific prior reproductive experiences enhance the response suggests that some form of conditioning occurs. Since there is some (albeit not too rigorously obtained in some instances) evidence for reproductive pheromones that influence human behavior (22, 30), it would seem to be a reasonable area for future research. A comprehensive review of pheromones has recently become available (88).

The conditioned secretion of gonadotropins and gonadal steroids in humans can only be inferred from a few suggestive experiments. For example, when college coeds living in pairs or small groups share a common social life, their menstrual cycles become synchronized (64, 96). In these studies, the dependent variable was the date of onset of the last menstrual period in October, the beginning of the school year, and again in the late Spring, at the end of the school year. Whereas the dates for any pair or group were random in the Fall, they were in phase in the Spring. This did not occur for randomly chosen control subjects not living together. Although one could generate several hypotheses to account for these data, including phero-

mones, facility for scheduling social functions, or whatever, it does seem clear that experience is critical for the shift, and that this repesents some type of learning.

There are also suggestive experiments for males. There is an anonymous report written by a man who lived in isolation on an island, who only came to the mainland to socialize with women every one or two weeks. He measured the weight of his beard growth every day and found that it increased significantly on the day or two prior to a trip to the mainland. He interpreted this as a conditioned anticipatory response. Since beard growth is a function of testosterone levels, he postulated that he had a conditioned release of gonadotropins and testosterone. He also measured the secretion of sebum from his sebacious glands, another response which is a direct function of testosterone levels, and found a similar synchrony. The influence of one's state of mind over this system is suggested by experiments showing changes of testosterone levels as one's self-image changes. For example, men undergoing instruction in yoga developed increased levels of testosterone which coincided with an increase of their self-evaluation (34). Similar phenomena may account for the decrease of cycling of women under various forms of psychological stress (25, 96).

Another area of potential research on conditioning endocrine levels is that of induced reflex ovulation. Many animals, such as the cat and the rabbit, ovulate only after copulation, the copulation itself evidently triggering a release of LH from the anterior pituitary (27). Although no experiment has specifically investigated the conditionability of this response, Davidson et al. (26) suggested that it probably exists and that they have observed conditioned ovulation in birds. This might have some relevance to humans since the argument has been made that humans have a tendency toward reflex ovulation.

Lactating mothers secrete pituitary hormones, oxytocin in most species, when the baby begins sucking the nipple. A few minutes later milk is let down. There is considerable evidence that stimuli associated with the process of nursing, such as the presence of the hungry baby, the cries of the baby, etc., can cause a conditioned release of the hormones with subsequent milk letdown. This is a common observation among experienced nursing human mothers (68) but is never seen in nonnursing mothers (94). In dairy cows, which are milked by machine at about the same time each day, there is a conditioned release of oxytocin when the regular milker enters the stall (21). Similarly, growth hormone, a milk release hormone important in goats, has been shown to be conditionable to arbitrary stimuli (60), as has prolactin in rats (38).

Again, these responses can be interpreted in terms of an adaptive anticipatory response that is due to learning. In all of the examples above, in-

cluding all of the different hormone systems, there are very few instances where endocrine conditioning that occurs naturally is maladaptive. It may be that inappropriate secretion of insulin in response to meal-related stimuli can cause some discomfort, or that inopportune crying of a baby can cause some discomfort to a mother, but the general tendencies seem to be more favorable. When one scans the classical conditioning literature on nonendocrine internal responses, especially the Russian literature (e.g., 18, 20), one gets the impression that virtually any response that can be influenced by the nervous system can become conditioned, and that if it is advantageous for an animal to learn such a response, including an endocrine response, it probably will.

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The Comprehensive Handbook of Behavioral Medicine, Volume I

Introduction To The Genitourinary System

We have included two chapters in this section. The first reviews the evaluation and treatment of enuresis, the second focuses on the treatment of common male and female sexual concerns. A discussion of toilet training in normal children is presented in Volume 3 of the handbook.

Doleys begins his chapter on enuresis with a review of the physiology of micturation and the prerequisite medical, clinical, and behavioral screening necessary to properly assess a client's problem. Adequate treatment may hinge on an accurate evaluation of the client; and the failure of some treatments to have an effect may occur because they are used with the wrong clients. As Doleys notes, the "urine, bell-and-pad, or Mower apparatus is probably the most thoroughly researched behavioral conditioning device, both from a standpoint of outcome and process variables." The overall results of treatments based on these apparatus are impressive: "In over 600 subjects mostly between the ages of 4 and 15, there was a 75% rate of remission with treatment durations of 5 to 12 weeks. A 41% relapse rate was reported with 68% of those subjects who were retreated to become continent."

There have been several case reports of the usefulness of behavioral techniques with two rather rare but troublesome urinary problems, "the shy bladder," characterized by avoidance of public urination and pollakuria, or excessive frequency of urination. The shy bladder symptom can provide considerable discomfort to a client who may avoid many activities and rush from one bathroom to another in hopes of finding one empty. Anderson (1) used desensitization *in vivo* to treat five patients with chronic inability to urinate in public. All patients improved after six or seven sessions. There were no relapses at one year. Pollakuria is an excessively high number of daily urinations. The problem has been successfully treated by reinforcing and shaping increased bladder capacity (2), muscle relaxation and systematic desensitization (4), reinforcing increased periods of non-urination (6), relaxation and structuring time of urination (3), and a technique which used discussing pleasant events to compete with the urge to urinate (5).

The behavior treatments of common sexual problems have not been as extensively studied or evaluated as have the behavioral therapies of enuresis, but the techniques and approaches are equally exciting. The stage for the treatment of sexual problems was set by Masters and Johnson, who have revolutionized the assessment and treatment of sexual disorders. Behavioral researchers owe them a great deal, since they have removed much of the mystery of sexual dysfunction and firmly legitimized the evaluation and treatment of sexual disorders as a scientific undertaking. Behavior therapists have jumped into the breach with a wonderful array of techniques and procedures. Annon and Robinson discuss several useful techniques in their chapter on the treatment of common male and female sexual concerns. They argue convincingly that sexual disorders are learned, and provide a classification system based on learning theory. They then discuss simple treatment approaches to many common sexual problems. As they note, educating clients about "normal" sexual function and reassuring them that their sexual activities or fantasies are not "sick" is often sufficient to overcome their problems. Other clients require more elaborate treatments and many of these treatments can be successfully used by health care practitioners.

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The Comprehensive Handbook of Behavioral Medicine, Volume I

CHAPTER 13

Enuresis

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DEFINITION AND CLASSIFICATION

Functional nocturnal enuresis is characterized by persistent wetting of the bed in the absence of urological or neurological pathology (8). There is considerable controversy over the age at which a child may be considered enuretic. Muellner (46, 47) suggests that urinary control should be established by three years of age and that potential long-term bedwetters can be identified at this time. In most instances, however, treatment is not applied to children under five years of age (15). Most investigators accept regular wetting of the bed three times a week as sufficiently frequent to initiate treatment, although treatment is often applied when the enuretic episodes are less frequent.

It is estimated that enuresis occurs in 15 to 20% of all five-year-olds (40, 49, 70). This decreases to approximately 2% in the 12 to 14 year-old age group. The incidence of enuresis among males is about 1.5 times greater than in females. Enuresis is typically divided into two categories, primary and secondary. The primary (continuous or persistent) enuretic has never achieved nocturnal continence. The secondary (discontinuous or acquired) enuretic has demonstrated nighttime continence for a period of at least six months sometime in his life. It is estimated that 85% of enuretics are of the primary type; this percentage decreases with age, and only about 50% of 12 to 14-year-old enuretics fall into this category.

Forsythe and Redmond (25) reported a study of 1129 enuretics who had not become continent with drug therapy or surgery and who had never been exposed to a urine alarm conditioning procedure. Fifty-six percent of these patients were primary enuretics, and 90% wet their beds five or more times a week in an eight-week period. Spontaneous remission occurred in

14% of the 5 to 9-year-olds, 16% of 10 to 14-year-olds, and 15% of 15 to 19-year-olds, with 3% of the original sample remaining enuretic at 20 years of age.

Although enuresis is often regarded as a sign of emotional or psychiatric disturbance, this has not been experimentally substantiated. Studies by Kolvin et al. (37), Shaffer (57), and Rutter et al. (54) did not reveal a significant correlation between enuresis and any specific psychiatric syndrome or deviant behavior pattern. Some positive correlations, however, were noted between enuresis and immature and introverted behavior in boys, familial disruptions, and behavioral deviance in females.

PHYSIOLOGY OF MICTURITION

A basic understanding of the physiology of micturition (urination) and the developmental sequence associated with the acquisition of diurnal and nocturnal urinary control helps one adequately assess the problem and devise an effective treatment procedure. Yeates (71) described five steps in the functioning of the normal mature bladder: Bladder filling by flow of urine from kidneys through the ureters; desire to void initiated by stretching and relaxing of the detrusor muscle which surrounds the bladder; inhibition of voiding by maintaining pressure on the sphincter muscles or by perineal pressure; onset of voiding following bladder fullness as a result of rhythmical contractions of bladder; and sustained bladder contractions and sphincter relaxation until the bladder is empty.

Muellner (46, 47) has described four stages in the development of the mature bladder in children: Demonstrated awareness of bladder fullness, usually between one and two years of age; ability to retain urine, noted at about three years of age; capacity to start and stop the flow of urine in midstream noted about 41/2 years of age; and ability to initiate and terminate the flow of urine at any degree of bladder fullness.

ASSESSMENT

A thorough assessment of the problem, which is a prerequisite to efficient treatment, should include a medical screening, a clinical interview, and behavioral recording (10, 16, 17). The medical screening should evaluate the patient for neurological or urological pathology. A urine culture and a urine analysis are standard procedures for ruling out infections and intrinsic kidney disease. Although some physicians also perform voiding cystourethrograms (VCU's) or intravenous pyelograms (IVP's) to further

substantiate the absence of anatomical or functional abnormalities, the standard use of these radiological and urological procedures prior to any treatment has been debated because of the discomfort to the child and the expense. There are several easily observed signs which may indicate pathology, and their presence should be followed up with radiological examinations. These include the size and velocity of urine stream, presence of diurnal dribbling, dysuria (painful urination), polyuria (frequent urination), and urgency (inability to retain) (2, 11). The presence of organic pathology does not necessarily contraindicate the use of conditioning procedures such as the urine alarm, but may imply other treatment considerations such as surgery or medication.

The clinical interview should be comprehensive and elicit information in at least five areas: Daytime and nighttime wetting; history of enuresis and previous treatment; brief medical history of family; presence of other problem behaviors; and home and family environment (15, 16, 17). Estimates of frequency of wetting episodes, retention ability, and sphincter control help determine the severity of the problem, the need for in-depth medical examination, and the achievement of developmentally important skills. Effects of toilet training, events correlated with the onset of enuresis in the secondary enuretic, and responsivity to previous treatment may indicate to the clinician procedures to avoid. When the family history is positive for renal disease or diabetes, these disorders should be specifically ruled out in the patient. The use of behavioral problem checklists aid in identifying other problems, such as noncompliance, which could interfere with treatment. Information relating to the general home and familial environment and parental attitudes is also important. Parental tolerance of wetting has been positively correlated with treatment success (45), while marital and familial difficulties have been related to failures (64).

Behavioral recordings can provide a valuable description of the wetting behavior, and a baseline against which to evaluate treatment, and an assessment of parental cooperation and motivation. A three-week recording period is recommended. The frequency of nighttime wetting, time of wetting episodes, size of wet spot, bladder capacity, and retention ability should be examined. Self-monitoring and advice-encouragement by themselves have been shown to be effective with some children (14, 19, 43, 68) and can be initiated during this three-week period.

Functional bladder capacity is defined as the amount of urine an individual will retain before voiding rather than the structural size of the bladder. Zaleski et al. (74) compared the functional bladder capacity of enuretic and nonenuretic children and found that of enuretics to be generally lower. A small functional bladder capacity may suggest the need for bladder stretching exercises (59) or retention control training (19). Two estimates of blad-

der capacity can be obtained: The maximum (MBC) and average (ABC) bladder capacity. The MBC is determined by providing the child with a relatively large amount of liquid. He is then asked to refrain from voiding as long as possible when he feels the urge. Urine output is measured for the next two voiding episodes, and the largest of these two is the MBC. The ABC is the average void calculated from recording the volume of each voiding episode over a 7 to 14-day period. The largest void recorded during this period is another way to determine the MBC. Providing parents with a measuring cylinder, data collection sheets, and written instructions facilitates accurate recording. The child's involvement and cooperation is necessary. Weekly phone contacts or brief office visits help maintain collection during the baseline period.

TREATMENT

Pharmacological

Most of the pharmacological agents which have been used to treat functional enuresis have been relatively unsuccessful. Contemporary research has focused primarily on the use of stimulants such as amphetamines and the tricyclic antidepressant imipramine (Tofranil). It was proposed that amphetamines would lower the threshold of sleep and increase the likelihood that the enuretic would be aroused by internal cues generated by bladder fullness. Research by McConaghy (42), Young (72), and Forrester et al. (24) showed amphetamines to be successful with 22%, 36%, and 23% of the subjects respectively, which is relatively less effective than conditioning treatments. In a comprehensive review of the literature, Blackwell and Currah (5) reported they could find no controlled studies that supported the use of amphetamines in the treatment of nocturnal enuresis.

Although imipramine hydrochloride (Tofranil) has been significantly more effective than other drugs, the basis for its action is still unknown. The effects of imipramine have been attributed to mood elevation; lightening of levels of sleep; its anticholinergic effect on the detrusor muscle which results in relaxation of the muscle and accommodation of larger amounts of urine in the bladder; and increases in voluntary control of the urethral sphincter (5, 38, 41).

In general, imipramine appears to be more effective than a placebo when given in dosages of 25–75 mg adjusted for age and weight. Increased urinary control is typically noted during the first week of administration in as many as 85% of the cases (58). About 30% of children achieve total control, but only 5 to 40% of these maintain continence following the withdrawal of the drug (5, 35, 58, 61). Cystometric studies have shown increased bladder

capacity and delayed voluntary detrusor contractions after taking imipramine (29). Unpleasant side effects, including dizziness, headaches, constipation, and disturbances in mood and sleep, have been reported. Although rare, fatalities from imipramine toxicity have also been noted (44, 53). Imipramine is not differentially effective with functional enuretics and its use as a screening agent may mask organic disease which could be determined by a urological evaluation. Mahoney et al. (41) concluded that imipramine was of value in bringing about temporary relief of enuresis but should be used as an adjunct to procedures whose effects have been shown to be more permanent.

Bladder Expansion–Retention Training

Some studies on the physiology of micturition in bedwetters have noted a tendency for voiding to occur in response to relatively small degrees of bladder fullness (46, 47, 66). Similarly, Zaleski et al. (74) and Starfield and Mellits (60) have found the bladder capacity of enuretics to be lower than in nonenuretics. These data suggest the possible usefulness of bladder expansion or retention training to increase the functional bladder capacity of enuretics. Increased bladder capacity permits the child to sleep through the night by holding larger amounts of urine, or results in nighttime arousal due to the increased strength of internal cues emanating from a more distened bladder.

The basic retention training of bladder expansion procedure calls for the patient to increase his intake of liquid and refrain from voiding for successively longer periods of time or until it becomes very uncomfortable. Generally, a maximum retention time of 30 to 60 minutes is used. Vincent (65, 66) described a mechanical device that facilitated the suppression of voiding by applying perineal pressure from a patient-controlled inflatable apparatus. Caution must be exercised to avoid rupturing the bladder if extreme pressure is maintained for long periods of time.

The studies in this area have provided mixed results (16, 17, 19). Starfield and Mellits (59), for example, required one holding trial a day over a six-month period. Although increased bladder capacity was positively correlated with the decreased frequency of bedwetting, only 6 of 83 children achieved continence. In some instances large increases in bladder capacity were accompanied by no change in bedwetting, and conversely, some children who showed no or small changes in bladder capacity demonstrated significant decreases in nocturnal wetting.

A variation of this procedure was introduced by Kimmel and Kimmel (36) and later studied by Paschalis et al. (50). Retention control training

(RCT) used increased liquid intake, retention following each urge to void, gradually increased retention interval, and reinforcement for successful retention during a 7 to 20-day treatment period. Seventeen of 38 children treated this way became dry. Other studies (1, 18, 19, 20, 31, 52) have not replicated this positive outcome. These studies generally noted that RCT or bladder expansion training, while producing an increase in bladder capacity, did not result in a concomitant decrease in bedwetting.

This discrepancy is perhaps best illustrated by Harris and Purohit (30) who treated 18 children for 35 days with retention training. The first 5 days were supervised by the experimenter and the remaining days by the parents. The children were given increased amounts of liquids, retention training, and reinforcement for success. Retention trials were at least once a day. At the end of training the children had an increase of 34.6% (83.3 ml) in bladder capacity and an average decrease in wetting of less than one night per week. A similar study by Allen (1), essentially replicating the Kimmel and Kimmel procedure with retention training for 90 days, reported that only 8 of a group of 14 children completed treatment and that none of these became continent.

Johnstone et al. (33) discussed the results of a "closed method" of bladder distension for children who have abnormal detrusor activity. In this procedure a balloon catheter was passed into the bladder and filled with saline. Distension of the bladder with the balloon continued for two hours. Enuresis was eliminated in 4 of 16 cases. In the 11 cases where significant increases in post-treatment bladder capacity were recorded, 3 were cured, 3 improved, and 5 unchanged at one-year follow-up. These data are slightly better that would have been expected without treatment.

In summary, these data do not support the isolated use of retention training or bladder expansion to treat nocturnal enuresis. Although Doleys (16, 17) has outlined a number of considerations that make the comparison of studies difficult, the fact remains that increases in bladder capacity have not been reliably correlated with decreases in bedwetting. Based upon the observation that many enuretics do show a low functional bladder capacity, Hunsaker (31) has proposed a two-process approach to enuresis in which retention training is used in conjunction with the urine alarm. This combination may be particularly applicable to children who display developmentally inappropriate urinary control.

Dietary-Fluid Restriction

Gerrard and his colleagues (20, 26, 27) postulated that the lower bladder capacity frequently noted in enuretic children may be due to a spastic bladder caused by the child's hypersensitivity to certain foods. In these cases, he felt enuresis could have an allergic basis similar to that found in asthmatics

where the smooth muscles of the bronchi are in spasm. This notion of enuresis as a response to food allergies was previously proposed by Bray (7). Based upon this premise, Gerrard and his colleagues compared treatment based on dietary restriction and imipramine. Milk, egg products, and a variety of juices were removed from the child's diet. Twenty-five children were treated with dietary restriction initially for two weeks, and then placed on imipramine for two weeks. Twenty-five additional children were treated in the reverse order. Imipramine was more effective than dietary restriction in reducing the frequency of nighttime wetting. Dietary restriction, whether imposed before or after imipramine, did result in significant increases in bladder capacity. Similar data are presented by Gerrard and Zaleski (26), who discuss single cases showing more direct relationships among dietary restriction, changes in bladder capacity, and wet nights.

Several points need to be made about this research. First, many of the children treated with dietary restriction alone or with imipramine had low functional bladder capacities and daytime symptoms of urgency and frequency that are consistent with the diagnosis of structural functional pathology. Many enuretics do not display these behaviors. Secondly, reduction of fluid intake that occurred with the dietary restriction may have affected the enuretic pattern. Thirdly, long-term effects were not examined. Fourthly, the question remains whether or not these children could have been treated effectively with other procedures, like a urine alarm. This line of research may help establish the relationship between bladder capacity and enuresis. It also suggests that the importance of bladder capacity for the acquisition of nighttime continence may vary among individuals.

The effect of fluid restriction, which is often used as the first step in treating enuresis, has not been well studied. One study by Hagglund (28) compared the effect of fluid restriction and nighttime awakening to that of forced fluids during the day, and to a no-treatment control group over a three-month period with follow-up to eight months. Six of 18 children in the forced-fluid group became continent as compared to none in the other two groups. Cystometric data showed a 20% increase in bladder capacity for the forced-fluid group as compared with 10% for the fluid restriction group and 9% for the control group. Hagglund suggested that increasing fluid intake resulted in the acquisition of voluntary control over bladder muscular contractions and the elimination of involuntary contractions, both of which are characteristic of nonenuretic children. Fluid restriction and nighttime awakening appeared to mitigate against the development of this type of control.

Urine Alarm

The urine alarm, bell-and-pad, or Mowrer apparatus is probably the most thoroughly researched behavioral conditioning device. Usually a sen-

sing device which is activated by the passage of urine is placed on the bed. The child is awakened by a bell, buzzer, or light stimulus, turns off the alarm, and finishes voiding in the bathroom. He then returns to sleep. "Buzzer ulcers," which were noted infrequently with earlier alarm systems, have been virtually eliminated by recent safety standards.

Early research (56) reported an 80% rate of remission using a urine alarm. These data were supported in a later paper by Jones (34), who found higher rates of relapse and more variability among studies. Other experimenters (39, 40, 63, 70) report between 80 and 90% remission and about 35% relapse. The most recent summary (19) reviewed studies conducted between 1960 and 1975. In over 600 subjects, mostly between the ages of 4 and 15, there was 75% rate of remission with treatment durations of 5 to 12 weeks. A 41% relapse rate was reported with 68% of those subjects who were retreated becoming continent. These data are confounded by a high degree of variability in criteria for dryness (7 to 28 days) and relapse between researchers.

Several features of the urine alarm procedure have been examined. Temporal contiguity between the onset of the alarm and nocturnal wetting is important (12, 51). The urine alarm was found to be superior to nighttime awakening (9), placebo tablets (68), no treatment (13), verbal psychotherapy (48, 67), imipramine (42, 72), and retention training (1). The adjunctive use of stimulant medication has not increased rates of remission or reduced treatment duration (19). However, Turner, Young, and Rachman (64) have indicated the potential utility of any medication that would produce a rapid decrease in wetting as a means of ensuring parental compliance with treatment requirements. Parental noncompliance has been cited as the most frequent cause of treatment failures (19), which can be treated only by careful instruction, feedback, and follow-up by professionals. The instructions that usually accompany commercially available alarms are inadequate and result in the frequent failure of unsupervised treatment attempts.

Some questions have arisen regarding the behavioral side effects resulting from the use of the urine alarm. A review by Shaffer (57) noted that new emotional or behavioral responses may appear temporarily in a minority of treated children. Sacks et al (55), using personality inventories, symptom checklists, and teacher rating scales, found a decrease in behavioral symptoms following the application of the urine alarm procedure.

Two modifications of the standard urine alarm procedure have been introduced to reduce the rate of relapse: Intermittent scheduling of the alarm, and overlearning. Finley et al. (23) described an automated apparatus that can be preprogrammed to operate following a given percent (usually 50 to 70%) of wetting episodes. They propose that the use of an intermittent

alarm is similar to an intermittent schedule of reinforcement. Behaviors acquired with this schedule of reinforcement should be more resistant to extinction than behaviors acquired with a continuous schedule of reinforcement. Finley et al. (23) compared an intermittent alarm (70% of wets were followed by the alarm which is paired with a light) to an alarm that sounded with each wetting, and a no-alarm treatment group over a six-week period. Both conditioning groups were superior to the no-alarm control group, and rates of relapse were significantly lower for the intermittent group, 15% vs. 44%. A subsequent study (22) reported that only 3 of 87 children treated by the intermittent procedure failed to achieve continence and only 17% relapsed! In a more recent study by Finley et al. (21) the results of treatment with a 70% intermittent variable ratio schedule in 80 enuretics is reported. Continence (14 consecutive dry nights) was acquired by 94% of the subjects completing treatment, with a mean treatment duration of 7 weeks. The overall rate of relapse was 25%, which ranged from 5.25% for 7 to 8-year-olds to 50% for 9 to 10-year-olds. Other studies (19) have confirmed the efficacy of the intermittent alarm procedure, although in most cases the results are not as markedly successful. Experimental control, population variables, apparatus, number of dropouts, and alarm schedule may contribute to this difference in outcome.

Overlearning following the initial acquisition of dryness is a second innovation in the standard urine alarm procedure. Overlearning requires the child to consume 10 to 32 ounces of fluid, depending upon age and weight, prior to bedtime after achieving dryness with the urine alarm. Young and Morgan (73) proposed that relapses were a result of the absence of generalization of conditioning to conditions of bladder fullness or bladder distension which are larger than those experienced during treatment. Increased liquid intake increases bladder fullness during the night, tests the degree of control established during conditioning, and provides an opportunity for this control to include a broad range of bladder fullness and detrusor muscle distension. The initial study of overlearning (73) reported a lower relapse rate (13% vs. 35%) for subjects who completed overlearning compared to those who were exposed to the standard urine alarm procedure without overlearning. Taylor and Turner (62) compared overlearning to continuous and intermittent alarm groups. Although the percent achieving nocturnal continence was similar in all groups, lower rates of relapse were noted for the overlearning groups (23% vs. 69% vs. 44%). Jehu et al. (32) investigated the effects of overlearning in a residential home for children. Of the 19 children in the overlearning condition, 95% achieved continence after an average of 12 weeks of treatment. Thirteen continued to be dry during a 20-month-plus follow-up. Four subjects relapsed but were successfully retreated.

Despite its apparent efficacy, there are potential disadvantages to overlearning. Increased fluid intake has been known to cause relapse in some children (73). These children seem unable to tolerate large amounts of fluid and should probably be placed on a gradually increasing regimen. Alternatively, overlearning could be imposed several weeks or months following the initial acquisition of dryness to ensure the strength of this initial response to treatment. Renewed bedwetting after a period of dryness can be upsetting to parents and child. Both should be prepared for this possibility regardless of the treatment procedure initially employed, and they should be assured that it can be achieved again through retreatment or continued treatment. It has recently been shown that drinking 16 to 20 ounces of fluid at nighttime may not be necessary for overlearning, and it may be harmful to the child. Ten to 20 ounces of fluid in the hour preceding bedtime is usually sufficient.

Azrin et al. (3, 4) have expanded the urine alarm procedure into a multifaceted treatment program referred to as dry bed training (DBT), which emphasizes the application of social contingencies. They incorporate positive practice, positive reinforcement, retention control training, nighttime awakening, negative reinforcement, and full cleanliness training into the treatment program. DBT is initiated during one night of intensive training for which the therapist spends the evening in the child's home. The child is first told about the entire program and then required to perform 20 positive practice trials which consist of lying in bed for a brief period of time, arising, going to the bathroom, displaying the appropriate toileting behavior, and then returning to bed. The positive practice trials are intended to help integrate this response, which must occur in the middle of the night, into the behavioral repertoire of the child. He is then encouraged to drink his favorite liquid, repeat the training instructions verbally, and retire for the night. Hourly awakenings occur during the night. At each of these times, the child is directed to the bathroom and is encouraged to void or inhibit urination for an additional hour depending on his age. Positive verbal reinforcement is provided for a dry bed and small amounts of liquids are given to the child to ensure that he has the opportunity and the need to void during the night. Wetting episodes are followed by full cleanliness training exercises in which the child changes his nightclothes and bedding, cleans himself, and engages in 20 positive practice trials.

After this one night of "intensive training," post-training supervision begins with full cleanliness training and positive practice whenever wetting occurs during the night as well as prior to bedtime if the child has wet the bed the previous night. Children are awakened and taken to the bathroom only once during the late or mid-evening. This nighttime awakening is then gradually faded out. Parents are strongly encouraged to reinforce the child

for dry nights and to involve other family members and friends in supporting and encouraging the child. The third phase of dry bed training, referred to as "normal routine," begins after seven consecutive dry nights. During this phase, the urine alarm is taken off the child's bed and full cleanliness training is conducted only in the morning following a wet night. Positive practice is conducted the evening following a wet night. Post-training supervision is reintroduced any time two or more wet nights occur within any seven-day recording period. Of the 24 subjects in the initial study by Azrin et al. (3), 100% achieved 14 consecutive dry nights. Reinitiation of post-training supervision was required for seven of these children. Although specific follow-up data are not given, it was suggested that none of the subjects relapsed to pretreatment rates of nocturnal enuresis during the six-month follow-up.

Doleys et al. (19) replicated the dry bed training procedure and compared it with retention-control training. Thirteen subjects had six weeks of dry bed training (longer if the parents decided to continue). Eight of the 13 children achieved continence for 14 consecutive dry nights with a mean treatment duration of 8.4 weeks. Five dropped out at various points of treatment. A two-year follow-up (69) was obtained for 12 of 13 children treated with these procedures. Parental reports indicated that five of the eight children who achieved dryness maintained continence through a two-year follow-up. Two were wetting the bed approximately 4 times per month, and 1 was wetting the bed 6 times per month. The four children who terminated treatment before dryness criteria were achieved had an average of 14.5 episodes of wetting per month. Overall, about 38% of the children who achieved dryness with this procedure relapsed.

Bollard and Woodroffe (6) replicated the DBT procedure using parents rather than trained therapists to implement the program. Their results were very similar to those of Azrin et al. (3). All of the children who had dry bed training achieved 14 consecutive dry nights. Total treatment duration was approximately 6 weeks. None of the children relapsed to pre-treatment wetting frequency during a 16-month follow-up; however, two did require reinstatement of the urine alarm. A smaller group of children were exposed to DBT without the use of the urine alarm. Although wetting was substantially reduced in this group, continence was not achieved. It appears that the urine alarm is an essential feature of DBT.

SUMMARY

The accumulated data suggests that the vast majority of children with enuresis can be classified as functionally nocturnally enuretic. This defini-

tion implies that their enuresis is occurring in the absence of any identified neurologic or urological pathology. The incidence of enuresis is known to be higher among males than females and is not usually associated with any specific behavioral or psychiatric pathology. Although many professionals recommend postponement of the treatment of the enuresis with the rationale that many enuretics become continent in time, early treatment is strongly supported as a means of avoiding embarrassing and troublesome situations for the child and his family. Intervention when the child is five or six years old is not uncommon and usually successful. There is reason to believe (21) that postponing treatment until later years may increase the likelihood of relapse or unsuccessful outcome.

Enuresis should be viewed as a complex problem requiring a multidimensional assessment which should begin with a thorough medical evaluation, a complete clinical review of other problem areas for the child or his family, and an accurate and reliable description of the patient's wetting pattern.

A variety of treatment procedures have been proposed for enuresis. Imipramine (Tofranil) appears to be the most effective pharmacological agent for the disorder. This medication does not result in a substantial percentage of enuretics becoming continent, and the relapse rate following withdrawal is above 50%. Imipramine can help produce a rapid decrease in the frequency of wetting, and build confidence in the ultimate success of treatment in the parents and the child. The continued use of a urine alarm after withdrawal of the imipramine may help reduce the high rate of relapse seen when the drug is used alone. Fluid and dietary restrictions, nighttime awakenings, baseline self-recording, and parental encouragement and reinforcement have all been shown to be somewhat effective treatments. Reports of the success of these procedures usually include small numbers of children; the effectiveness of these procedures has not been demonstrated in large numbers of children with enuresis. Bladder stretching exercises and retention control training have also been found to be successful in a limited number of cases. However, some data indicate that a few children become continent in spite of small bladder capacities while other children who learn to make large changes in their bladder capacity do not become continent. Thus, bladder stretching exercises appear to be useful only as an adjunct to conditioning-oriented procedures.

The behavioral conditioning techniques that employ a urine alarm device have been the subject of many well-controlled scientific investigations. Although the standard urine alarm, as introduced by Mowrer and Mowrer, was quite successful, dissatisfaction with high relapse rates has resulted in several modifications of their procedure. An intermittent alarm schedule

and overlearning following the initial acquisition of continence have been shown to be effective in reducing the rate of relapse while also producing a high rate of remission.

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The Comprehensive Handbook of Behavioral Medicine, Volume I

CHAPTER 14

Treatment Of Common Male And Female Sexual Concerns

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The *form* and *direction* of sexual behaviors is principally determined by sociopsychological influences (6, 9, 11, 23, 62). The influence of culture on what is considered "normal" sexual behavior can hardly be overstated. Anthropological research has repeatedly shown a wide variation in patterns of sexual behavior in different cultural groups. There is also wide variability among subgroups within a culture that depends on such factors as socioeconomic level and political or religious beliefs. The range of sexual behaviors considered appropriate when all cultures are considered encompasses virtually the entire range of sexual responses (62). Each culture has a code for "appropriate" sexual behavior, and individuals engaging in activities not endorsed by the code are usually considered "deviant." The following section will attempt to describe the role that psychological learning plays in an individual's acquiring of various sexual behaviors and attitudes within any given culture.

A PSYCHOLOGICAL LEARNING VIEWPOINT

In discussing the role of learning in human sexual behavior, Ullmann and Krasner (62) note that the range of biological stimuli and acts a person can be involved with far exceeds that which a given culture would select as appropriate. Environmental conditions affect the probability of and individual's exposure to sexually related stimuli or activities. The more frequently a person is exposed to sexual stimuli, whether or not the stimuli are

culturally "appropriate," the higher the probability some form of sexual activity will be learned. This might potentially account for the relatively lower incidence of many sexual "disorders" reported in preliterate cultures in contrast to more technologically advanced ones.

The assumption that human sexual behaviors are primarily the result of learning or conditioning has been suggested repeatedly over the past 30 years by researchers and scientists from a variety of disciplines (20, 31, 32, 45). They have postulated that these learning processes are similar to those governing other types of behavior. Hovever, few of these early formulations were supported by empirical data.

At the Reproductive Biology Foundation in St. Louis, Missouri, William Masters and Virginia Johnson (41) developed a now well-known model for sexual therapy. They postulated that human sexual response is constituted by the interaction of two separate systems, called the "biophysical" and the "psychosocial" systems. More relevent to the psychological learning theories of sexual behavior are the comparatively recent writings of a number of learning-oriented researchers, theoreticians, and clinicians (3, 4, 6, 23, 60, 62). These are carefully reasoned data-based theoretical discussions of how sexual behaviors may be acquired and treated.

In looking for a possible explanation of how and what individuals learn about sex within a given culture, we may consider the following: At birth the human, unlike nonhuman primates and other mammals, has relatively few "built-in" responses. Sex is not a basic need like food or air. If an individual human or animal does not engage in sexual behavior, it will not cease to live; however, animals must mate in order for the species to survive. From an evolutionary viewpoint, mating patterns must exist for species survival. Beach (12) suggests that the behaviors essential for reproduction are built up through learning from a *core* of unlearned stimulus-response patterns that are mediated by genetically controlled mechanisms. What does this core consist of?

Consider the newborn human. There are reports that about 50% of males are born with erections and approximately the same number of females with vaginal lubrication, the counterpart of the arousal-erection response in males. During sleep the average healthy adult male normally has an erection on the average of every 90 minutes during rapid eye movement (REM) sleep. the average healthy adult female may lubricate approximately every 90 minutes also during REM sleep. These data support the concept that sexual arousal is a natural physiological process like respiratory, bladder, and bowel functions. However, as Masters and Johnson (41) pointed out, although sexual functioning is a natural physiological process, it is unique among these processes since responsitivity in this system can be functionally denied or delayed for a lifetime.

Beach (12), making a cross-species comparison, concluded that one aspect of sexual behavior that does *not* depend on learning is the reinforcing effect of sexual climax. From a learning point of view, orgasm is a natural or unconditioned reinforcer that potentially can reinforce a wide range of sexual behaviors and attitudes through respondent and/or operant conditioning. In another cross-species comparison, he observes that "The self-stimulatory activities of male monkeys and apes are so similar to autogenital behavior in human males that we are justified in provisionally defining male masturbation as a basic primate trait." Beach goes on to point out that although masturbation in female primates has been observed, it is reported much less frequently than in males. Male genitals are external to the body and arousal leading to erection may be more easily discovered through accidental handling, fondling, or friction against environmental objects than in the female where the genitals are internal. If random behavior results in sexual arousal, the chances that the animal will demonstrate a similar behavior in the future becomes more probable. The more frequently these behaviors occur, the more often the animal pairs sexual arousal with specific environmental stimuli. Eventually, these stimuli alone may evoke an arousal response followed by sexual behavior. In this case, respondent and operant learning principles may explain animals' sexual responses and behaviors to "unusual" stimuli. Beach's (12) description of a cat that preferred to copulate with its food dish is a possible example of this type of behavior.

If this kind of behavior can be learned by nonhumans, we can presume that the same principles apply to human sexual responses and behaviors. For example, in American culture it is common during toilet training for small boys to receive positive reinforcement for handling their genitals appropiately, e.g., aiming directly into the toilet, while little girls generally learn to sit and "not to touch down there" and if they do, such as during a bath "only briefly—no lingering." With their external genitals and natural arousal responses to friction, it is not surprising that masturbation in males begins at a very early age and even leads to orgasm if prolonged, in boys as young as five months of age (32). Although the incidence may be lower, orgasm in girls has been reported at four months of age (31). Kinsey et al. concluded that masturbation is a normal and frequent behavior of male and female children. Human masturbation is often evoked and/or accompanied by fantasy and imagery which may relate to specific sexual behaviors with partners of other objects.

McGuire, Carlisle, and Young (44) suggested that frequent masturbation accompanied by fantasy may be important in the formation and shaping of sexually "deviant" behaviors. They suggest that the orgasm experienced during masturbation provides the critical reinforcing event for conditioning

the fantasy associated with masturbation. They argue that the fantasy material may be arbitrarily determined by a random experience to which the individual was subjected at some point in life. The availability of potential environmental stimuli in a given culture may shape sexual behavior at this point. For example, incidence of sexual activity with animals is reported more often in rural areas than in urban areas. The reported incidence of sexual "deviancy" is much higher among males than females, as is the reported frequency of masturbation and use of imagery during masturbation, a possible result of differences in conditioned behaviors. With the striving toward liberation in female sexuality, including masturbation and fantasy, in contemporary society, it will be interesting to observe if these differences continue to persist.

There have been numerous case descriptions supporting the masturbatory conditioning hypothesis (1, 44). Although direct experimental testing of the theory is not ethically possible successful clinical use of masturbatory conditioning has been widely reported (1, 2, 6, 16, 17, 28, 39), with occasional failures (16).

Rachman (51) and Rachman and Hodgson (52) demonstrate that it is possible experimentally to condition sexual arousal in males to previously neutral stimuli–slides of black boots–by pairing their presentation with sexually arousing stimuli, colored slides of nude women. McConaghy (43) reported conditioning penile volume change to the presentation of slides with red circles or green triangles after such presentation was repeatedly followed by pictures of female nudes for "heterosexual" subjects and male nudes for "homosexual" subjects. However, Langevin and Martin (33) had inconsistent results using similar procedures in subjects with low levels of sexual arousal.

Although orgasm should be an extremely effective reinforcer for any preceding or accompanying stimulus, Staats (59) pointed out that each sexual act is a series of potential classical conditioning trials because this series of sexual behaviors extends over a considerable length of time. Staats (58, 59, 60) proposed a motivational system for humans which he refers to as the Attitude-Reinforcer-Discriminative (A-R-D) system. Annon (1) designed and carried out a study to develop, test, and refine a conceptual scheme for understanding sexual problems and treating them from a learning theory framework. This research concluded that an initial analysis of a client's sexual problem using the A-R-D framework followed by a behavioral diagnosis of relevant behaviors was the most promising approach for ordering sexual concerns and developing therapeutic strategies. The A-R-D system has many important implications for the treatment of complex sexual disorders as well as for theoretical considerations of how various sexual behaviors and attitudes may be learned. A more detailed explanation of this system and its application to behavior is available elsewhere (3).

In summary, when dealing with sexual problems we do not have separate principles for "deviant" or "abnormal" and "normal" sexual behavior. As Ullmann and Krasner (62) have argued, rather than trying to define what is "normal," "perverted" or "deviant," we should ask, "How is any behavior learned, or how does anybody come to like or value anything?"

CLASSIFYING SEXUAL PROBLEMS

Some systems for classifying sexual problems may divide them into two categories; for example, one category is "deviant object" in which appropriate behaviors are related to inappropriate stimuli. A second category is often called "deviant response" regardless of partner "appropriateness." Eysenck and Rachman (19) proposed three categories: Inability to make an adequate sexual response to appropriate stimulation; sexual response to inappropriate stimulation; sexual response to inappropriate stimuli; and combinations of both. A more detailed system was presented by Staats and Staats (61) that included categories for behavioral deficits, inappropriate behavior, inadequate or inappropriate stimulus control, defective stimulus control of behavior, and inadequate or inappropriate reinforcing systems.

Although each classification strategy has advantages and disadvantages, none has proved very satisfactory, partly because of the ambiguity of the words "deviant," "appropriate," and "inappropriate." Ullmann and Krasner (62) suggest that categorization is difficult because the same act may be performed for different reasons due to different antecedent conditioning, and that the same person usually engages in a variety of acts which makes assignment to mutually exclusive categories difficult.

Labeling a particular sexual activity does not provide much *useful* information. We have compiled a list of more than 100 labels that have been used in the literature, with varying degrees of frequency and understanding for assumed and/or possible sexual disorders (6). There are undoubtedly many more. Even a casual glance at the list shows that one would have to be an expert in sexual terminology or a Latin and Greek scholar to make sense of many of these terms. To further confuse matters, different "experts" give the same term different meanings.

Labeling a person or his behavior is not an acceptable approach to understanding and assisting people with complex human problems, sexual or otherwise (4, 24). Labeling does not give clinically useful information about patients' thoughts or feelings in relation to their problems, and it may lead to assumptions about their thoughts or behaviors that may not be true. Labeled persons tend to be viewed as having behavioral characteristics which in fact may be seldom or never exhibited in their actual behavior, and it usually implies that a person responds or acts in the same way under all

conditions. Labeling sexual problems or behaviors does not dictate treatment. In contrast to the diagnosis "appendicitis," which dictates a fairly well-defined, though not always agreed upon surgical procedure, not one of the commonly used sexual labels dictates a well-defined set of treatment strategies.

While it is recognized that human behavior is rich, complex, and overlapping, classification systems could offer a tentative method for understanding sexual problems. For sexual counseling, a system is needed that *describes* the behavior of concern without value judgments or labels, and that still describes the relative clinical incidence of the behavior in question. As a step in this direction, the authors proposed a scheme (6) comprising four possible areas of concern: Response, Object, Behavior, and Identity (ROBI). This chapter will be limited to a discussion of the first of these areas, Response. Concerns in this area are usually viewed as a problem by the individual and his or her partner and generally include some type of inadequacy or dissatisfaction on the part of the individuals involved. The following types of sexual difficulties are included in this category.

Sexual problems related to:

1. *Response* (with opposite and/or same sex) involving:
   - A. Orgasm
     1. None, infrequent, or delayed
     2. Too soon
   - B. Arousal
     1. None or low
     2. High
   - C. Desire
     1. None or low
     2. High

THE PLISSIT MODEL

Before discussing specific procedures for assisting people with sexual concerns, the authors will present a model which they feel is useful for the conceptualization of different "levels" of treatment for sexual problems. As an aid to memory, this conceptual scheme is referred to as the PLISSIT model (3, 4) or more accurately: P-LI-SS-IT. The model has four levels of approach; each letter or pair of letters stands for a suggested method for handling sexual concerns. The four levels are the following: Permission–Limited Information–Specific Suggestions–Intensive Therapy. The first three levels are part of brief therapy, as contrasted with the fourth level, intensive therapy. The majority of sexual problems within the "Response"

category can be effectively dealt with by the first three treatment levels, P-LI-SS.

Level I—Permission

Often, people only want to know if they are normal and to be reassured that they are not "perverted," "deviant," or "abnormal." Many times they are not bothered by the specific behavior that they are engaging in, but they are bothered by the thought that there may be something "wrong" or "bad" with what they are doing. They would like a professional counselor or someone who is in a position of authority to listen to their fears, reassure them that they are not alone or unusual in their concerns, and give them permission to continue doing what they have been doing. Permission often prevents a problem from developing and, in this sense, it can be seen as a preventive measure as well as a treatment technique. Although it will not solve all sexual problems, it will resolve some and it has the advantage that it can be used in almost any setting at any time. Permission can be given for thoughts, fantasies, dreams, and feelings, as well as overt behaviors.

On the other hand, many sexual concerns can also be resolved by giving clients permission *not* to engage in certain sexual behaviors unless they choose to. For example, consider the young woman whose partner wants her to have "multiple orgasms" despite her satisfaction with the one orgasm that she experiences and unconcern that she be multiorgasmic. Giving her permission not to have multiple orgasms unless she chooses to may be very helpful. Conversely, in the case of the woman who would really like to experience multiple orgasms but is fearful or hesitant that she might then become a "nymphomaniac," permission to be multiorgasmic, if she chooses to, might be helpful. Permission-giving is most appropriate and helpful when it is directly related to the client's goals.

Limitations

It may appear that the basic assumption underlying permission-giving is that the clinician may sanction whatever sexual thought, fantasy, or behavior that an adult wishes to engage in privately or with consenting adult partners. In a very general sense this may be correct; however, there are limitations to the therapist's permission. One would not give permission for a thought, feeling, or behavior that would lead to potentially dangerous and/or illegal consequences. For example, to give a client permission to continue using rape fantasy during masturbation might set the stage for un-

desirable future consequences. The major limitations of permission-giving as treatment are determined by the clinician. The exent to which clinicians feel comfortable with and are willing to use permission-giving generally depends upon their breadth of sexual knowledge, theoretical orientation, and value system. If permission-giving is not sufficient to resolve the client's concern, then they may combine permission-giving with the second level of treatment.

Level II–Limited Information

Limited information provides clients with specific factual information relevant to their sexual concern. It may result in their continuing to do what they have been doing, or doing something different.

Limited information helps dispel sexual myths, whether they are specific such as those pertaining to genital size, or general such as the following: On the average, men and women differ markedly in their capacity to want and to enjoy sexual relations and in their fundamental capacity for responsiveness to sexual stimulation. Limited information is often helpful where there are concerns about breast and genital shape and size, masturbation, genital intercourse during menstruation, oral-genital contact, and sexual frequency and performance.

In giving limited information, it is important for the clinician to provide information directly relevant but limited to the client's concern. Robinson (54) has shown that a three-hour presentation of a wide range of sexual information has little influence on a client's behavior associated with a specific sexual problem. In contrast, presenting limited information directly related to the client's problem often produces significant changes in relevant attitudes and behaviors.

The drawbacks to limited information as treatment are essentially the same as for permission. The extent to which clinicians can use the limited information level will depend upon their breadth of knowledge. How they offer this information to their clients will depend upon their individual style and the needs of the client. As with permission-giving, the degree to which they feel comfortable with and are willing to use limited information-giving will also depend upon their theoretical orientation and their value system.

Level III–Specific Suggestions

To make appropriate specific suggestions to a client, the clinician needs a sexual *problem* history. This is not to be confused with a sexual history,

which is part of intensive therapy. At this level in intervention, it is assumed that a comprehensive sexual history is not relevant or necessary. If specific suggestions are not effective, then a complete sexual history may be a necessary first step for intensive therapy.

Guidelines for taking a sexual problem history for brief treatment are outlined below.

Sexual Problem History

1. Description of current problem.
2. Onset and course of problem.
   a. Onset. (Age, gradual or sudden, precipitating events, contingencies)
   b. Course. (Changes over time: Increase, decrease, or fluctuation in severity, frequency, or intensity; functional relationships with other variables)
3. Client's concept of cause and maintenance of the problem.
4. Past treatment and outcome:
   a. Medical Evaluation. (Specialty, date, form of treatment, results, currently on any medication for any reason)
   b. Professional help. (Specialty, date, form of treatment, results).
   c. Self-treatment. (Type and results)
5. Current expectancies and goals of treatment. (Concrete or ideal)

This outline can be structured to an interview of five minutes or five hours. For sex counselors it may be helpful to have a form printed with these sexual problem history guidelines that can be used as a general guide while interviewing, and as a record sheet for the client's responses for future reference.

Specific types of suggestions, based on the information that would be obtained in an interview based on this outline, are presented later in this chapter.

Level IV—Intensive Therapy

It is not within the scope of this chapter to describe or even to attempt to outline an intensive therapy approach to the treatment of sexual problems. For clinicians who have already received training within a particular orientation, now may be the appropriate time to initiate intensive treatment if specific suggestions do not resolve the client's problem. Annon has described a behavioral approach to the intensive treatment of sexual problems (3) elsewhere.

The PLISSIT model has a number of advantages. It may be used in a variety of settings and adapted to whatever treatment time is available. Theoretically, each level of treatment requires increasing amounts of knowledge, training, and skill on the part of the clinician. Because each level requires increasing professional experience, the model allows clinicians to treat clients up to their level of competence or comfort; and then make a referral elsewhere. Most importantly, this model provides a framework for discrimination between problems that require intensive therapy and those that respond to brief therapy. For those who teach sexual therapists, this model may offer a framework for training that can be geared to the student's level of competence. This model provides a framework within which clinicians can expand their knowledge, experience, and skills. It is also important to emphasize that this type of brief therapy is not intended to resolve all sexual problems. However, based on an ever-increasing amount of clinical and research evidence, we feel it is unethical to involve clients in expensive, long-term treatment programs without first trying to resolve their problems with brief therapy.

THE TREATMENT OF SEXUAL PROBLEMS RELATED TO RESPONSE

Problems of sexual response, with either opposite and/or same sex partners, are among the most prevalent in the American culture. Masters and Johnson (41) estimate that one-half of the marriages in the United States are threatened by some form of sexual incompatibility between partners who are experiencing difficulty in initiating or achieving mutual satisfaction in sexual relations. Others suggest the incidence may be even higher (15). Problems of response may be classified as concerns pertaining to orgasm, arousal, or desire.

Orgasm

Limited information may help resolve concerns in this area, or it may provide a groundwork upon which to base specific suggestions. For example, consider the following information related to orgasm.

There is some evidence to indicate that the experience of orgasm for males and females is essentially the same (50, 63). Some males can experience orgasm without ejaculation or possibly multiple orgasms. Although there are many theories about the process, the physiological mechanism that triggers orgasm is unknown. Many types of tactile stimulation can pro-

duce an orgasm in males and females, and dreams and fantasies can lead to orgasm without tactile stimulation. Males and females have continued to experience orgasm even though their genitals were removed or reconstructed, as in sex-change surgery, provided that they could experience orgasm prior to their surgery. Masters and Johnson (40) showed that males and females are more similar in their capacity for experience of sexual responsiveness than they are dissimilar. Unfortuantely, when it comes to treatment, males and females with identical sexual response problems are approached as if they were unrelated complaints. Treatment procedures that work for sexual response concerns may be equally effective for both sexes.

None, Infrequent, or Delayed

The incidence of never or infrequently having experienced orgasm is much higher for females (variously labeled: Frigidity, anorgasmia, or primary or secondary orgasmic dysfunction) than males (labeled: Impotence, ejaculatio retardata, ejaculatory incompetence, or retarded ejaculation). Although there may be many biological, chemical, or organic factors that influence desire or arousal patterns, there is little evidence to indicate that these factors play any direct role in a person's ability to experience orgasm. It is generally assumed that all healthy males and females can learn how to experience orgasm.

Prior to the development of direct therapeautic approaches, these problems had been considered refractory to treatment. For females, behaviorally oriented approaches to treatment are generally directed at covert therapies, such as systematic desensitization, which are aimed at reducing the anxiety associated with sexual behavior, or overt treatment such as assisting her to learn new behaviors conducive to the experience of arousal and orgasm by using some form of successive approximation to sexual response (1), or a combination of treatments.

When obtaining a sexual problem history from the female, it is important to ask if she ever experienced an orgasm under any conditions, such as self-stimulation, oral or manual stimulation by a partner, dream, or fantasy, or other means. If she has not, the most effective way to help her experience orgasm is to suggest self-exploration. The goal is not to learn to experience orgasm only through "masturbation," but to learn about her body so she can teach her partner to help her achieve sexual satisfaction. Initial suggestions usually center on creating an environment for relaxation and privacy, with attention to lighting, setting, music, or whatever she has found conducive to relaxation and arousal in the past. She may be asked to explore

her body visually with the aid of a mirror and tactually with her hands, using water and soap in the bath or shower, oils, or talcum powder in other settings. She is asked to determine what areas feel most positive or pleasurable, and to continue to explore them a little further each time to find out what rhythm and movement feels best. For women who have learned "not to touch down there," a vibrator or other objects may be helpful. She is then asked to combine exploration with fantasy of her partner. If fears about reaching orgasm are present, role playing of what she thinks an orgasm might feel like at and beyond the point she fears is suggested. Once she learns to experience orgasm with this type of stimulation, it may be important to suggest that she fantasize intercourse with her partner if this is one of her goals. Once she has learned experience orgasm consistently with some form of self-stimulation, she is ready to teach her partner what she has learned about her own responsiveness. She is now in the same position as women who report that they can experience orgasm through self-stimulation, but not with a partner.

There are a number of ways for her to transfer what she has learned to her partner. For example, she can show him—usually with his arms around her, with the light on or off depending upon their preference. Afterwards, he can show her what he has learned about himself in similar exercises. She can then successively guide him in the rhythm and form of stimulation that she has found most conducive to the experience of orgasm until he can reliably assist her in this experience. Certain positions may be helpful, such as rear entry to the vagina so that she can continue manual or vibrator stimulation to the point of orgasm. Various bridging techniques have been described, such as mutual sexual activity with the vulva near the penis with continued manual or vibrator stimulation, then entry at the point of orgasm. Gradually, entry is made earlier and earlier so that eventually penile thrusting alone leads to orgasm. Alternatively, entry can be made any time and followed by stimulation to the point of orgasm. Manual or vibrator stimulation can be slowly faded out as penile thrusting takes over. (For a more comprehensive and detailed description of these and other suggestions for women who are alone or with a partner, see Annon, 2, 4.)

There are many procedures that help females achieve orgasm, as well as a number of research studies demonstrating the efficacy of more novel approaches. Robinson (53, 54), in a controlled study with no therapist contact, demonstrated that women exposed to observational learning (modeling) through the use of videotapes acquired novel sexual behaviors and/or significantly increased sexual activities that had occurred only rarely when they entered the program. These procedures were also effective in promoting more favorable attitudes toward the specific sexual activities presented in the videotapes (7). LoPiccolo and Lobitz (38) reported excellent results in

helping women achieve their first orgasm with a nine-step program of directed masturbation combined with training the husband in sexual techniques. Similar successful results have been obtained in groups of women with similar sexual concerns (10), groups of women with different sexual concerns (46), groups of couples with mixed sexual problems (36), and groups of couples with general sexual dissatisfaction (37).

As previously mentioned, males having difficulty in experiencing orgasm are relatively rare. Some indirect approaches to treatment in the past sometimes took up to six years of continuous therapy (48). Masters and Johnson (41) reported success in their two-week intensive therapy program by using procedures somewhat similar to those mentioned for females that can be described as shaping or successive approximation. Gagliardi (22) has an interesting case report of a treatment failure using indirect techniques for 52 sessions, after which he shifted to a Master and Johnson type approach with successful results after eight additional treatment sessions. Newell (47) attempted the Masters and Johnson procedure with a male client but had to abandon it because of the wife's refusal to cooperate. He then switched his client to the use of a penile vibrator with successful results that were maintained at follow-up 18 months later. Again the authors wish to stress their viewpoint that almost any suggestion given to a female for this problem—for example, the use of a vibrator—will be appropriate for a male with a similar problem. Failure to achieve orgasm during genital intercourse is the most frequently reported problem by females, and the least frequent problem reported by males. Most interesting is that this ratio is reversed in the problem of experiencing orgasm too soon.

Too Soon

A male feeling that he has reached orgasm too soon (variously called: Impotence, ejaculatio praecox, hyperaesthesia, premature ejaculation, or even satyriasis) is one of the most commonly reported male sexual response concerns in America. For females (labeled: Hyperaesthesia or nymphomania) it is extremely rare, as it is for males who engage in same-sex behavior. The defining characteristic, "too soon", may range from "anytime before the male (or female) is ready" to "after the completion of four strokes."

Unlike erection concerns, this problem in an otherwise healthy male does not have any known physical cause. Permission and limited information can be very helpful in these cases. Letting him know that almost *any* man who is sexually deprived, particularly after prolonged sexual separation from a partner, or with a new partner for the first time, tense, very sexually

excited, or who maintains an erection for a prolonged period of time, will almost invariably ejaculate quickly, can be very reassuring and may help to change his view of the situation. He is provided information relevant to his situation as determined by the problem history, and a possible hypothesis is offered about how he may have learned to respond in this manner. This provides the framework within which to offer specific suggestions.

Discrimination learning and successive approximation suggestions are the most common direct approaches to treatment in the male. Semans's (56) "stop-start" technique using extended sessions of penile stimulation by his partner, or Masters and Johnson's (41) "squeeze" technique, where the partner applies pressure to the glans of the penis immediately prior to his report of impending ejaculation, have been equally successful. Zeiss (65) reported good treatment results by providing couples with instructional materials on the Semans techniques and minimum therapist contact. Group treatment of four couples with this problem was reportedly successful with a variation of the Semans technique (30).

These and other therapeutic suggestions, e.g., redirection of attention, use of the Valsalva maneuver, or pulling of the male's testicles away from his body at the point of impending orgasm, all have a common underlying theme. It does not seem to be the "squeeze" or the "pull" or the "stop" as much as the *increased frequency* of sexual contact leading to orgasm and ejaculation during the treatment process that helps the male learn to prolong his orgasm (4).

Typically, a man with this concern keeps putting off sexual contact with his partner, who may at the same time be avoiding contact because of past frustration. When the male finally does make contact, he is so easily aroused and highly excited that it may take only a touch, tap, or whisper to "set him off." To break this cycle, increased frequency of sexual contact with orgasms and ejaculation is recommended. This can be suggested in one of two different ways. The therapist can suggest that, if at all possible, the male and his partner take a weekend holiday away from commitments, family, and usual everyday pressures. This may be a weekend in an expensive hotel with luxury accommodations, or a weekend camping trip on a secluded beach or in an isolated forest. He is asked to forget about delaying orgasm and to engage in whatever sexual activity he and his partner enjoy, and to experience orgasm as quickly as possible. They can then rest, shower, swim, etc., then engage in more mutually enjoyable sexual activity until he has another erection, orgasm, and ejaculation. If he is worried that he may not be able to obtain an erection a second time, he is told not to worry about it since there is always another occasion and in the meantime he can use the opportunity to explore other methods of mutual gratification and pleasure that do not require an erect penis. Relieving him of pressure

for performance and directing his attention to pleasing his partner usually results in another erection. It may be suggested that he engage in sexual activity as often as possible during this weekend and enjoy any type of stimulation both partners find mutually pleasing.

Usually by the second or third ejaculation, the male finds that he is lasting longer without conscious effort. This in turn reinforces his desire to have intercourse again. Couples returning from a weekend like this generally report positive results. Their success encourages them to engage in mutual sexual activity more often, which helps maintain their progress. Mutually pleasing sexual activity eliminates many problems that are reported as "sex drive differences" or "frequency differences."

As an alternative, those who are not able to take such a weekend can explain the program to their partner and increase their frequency of sexual activity at home for a trial period of one month. This somewhat slower procedure generally has the same results.

Albert Ellis (18) has long advocated increased frequency of intercourse for premature ejaculation by suggesting to males that they "double" their frequency. If after a period of time they are not lasting as long as they would like, they are told to "double it again" and to continue to do so until it is no longer a problem.

To the best of the authors' knowledge, there are no reports of treatment for this concern with females. However, it is interesting to observe that the clinical literature over the years has often noted that when women are described as engaging in a high frequency of intercourse (labeled: Nymphomania), they also appear to experience more difficulty than usual or take longer to experience orgasm.

Arousal

The arousal response in males and females may be elicited by overt tactile stimulation and sensory input to the eyes, ears, nose, and mouth, and/or by covert stimulation in dreams, thoughts, and fantasies. This response appears to be a natural physiological process which in males includes blood volume and pressure pulse changes in the penis during erection. The comparable response in females is vagianal blood volume, pressure pulse changes, and lubrication. Researchers have measured these responses with penile strain gauges and photoplethysmographs to discover factors that elicit arousal. The data suggest that eliciting stimuli in males and females may be more similar than dissimilar (27). There are some preliminary data suggesting there may be discrepancies between objective measures and subjective report of arousal which depends on expectation in

a given situation. With their erection responses, men are usually more accurate in their reports of arousal than females with their internal reactions. There is also evidence that cognitive factors can either inhibit (25) or enhance arousal (27). The arousal response may become associated through classical and/or operant conditioning with a variety of stimuli; e.g., pictures, slides, films, and audio and videotapes related to opposite sex, same sex, children, inanimate objects, rape, etc. Objective measurement of arousal is proving very useful in treatment for initial, ongoing, and post-therapy assessment.

None or Low

There are many stimuli that may interfere with the arousal response in males or females. For example, a female engaging in painful intercourse may eventually experience involuntary contraction of the vaginal outlet which may become a classically conditioned response that may prevent penile entry. As Roen (55) has suggested, there are numerous possible organic diseases, e.g., anatomic abnormality, neurologic disease, systemic disease, accidental or surgical trauma with scarring, hormonal deficiencies, and drugs like narcotics, sedatives, alcohol, etc., that may interfere with the natural arousal response. However, it should be kept in mind that one man with an organic problem may be impotent while another man experiencing the same pathological process may have no erectile problems. Belt (13) pointed out that even some castrated, estrogen-treated males with prostatic cancer may perform sexually with little problem.

Despite the large number of possible organic causes for sexual dysfunction, most authorities agree that 90% to 95% have a psychological basis. Some suggested causes include the following: Orgasm concerns, fatigue, preoccupation, jet lag, overdrinking, criticism from a partner, boredom with a partner, an unattractive partner, worry about disease or pregnancy, guilt about masturbation, same-sex responses, unfaithfulness, anxiety associated with expected performance either by one partner or the other, etc. This type of event may be a "precipitating" event that elicits thoughts about the next sexual encounter, which in turn produces a great deal of anxiety and worry or "fear of performance," which blocks the normal arousal system and results in another failure. Once this cycle is established, it may be difficult to break. Learning-oriented approaches generally see these problems as conditioned anxiety, and treatment is usually aimed at reducing anxiety associated with approach to sexual relations.

For females who report pain during sexual relations, there are many possible physical causes which require a careful gynecological examination,

diagnosis, and appropriate medical treatment. If pain is continually associated with sexual intercourse, this may result in a classically conditioned involuntary spastic contraction of the vaginal outlet which prevents penile entry. This condition can occur with otherwise normal genitalia and may last for years with the diagnostic labels of "unconsummated marriages" and "virgin wives" (21). In taking the problem history it is essential to identify exactly the circumstances of the pain experience and where the pain is felt. For example, if the woman reports a dull pain deep inside her vagina only when she is on her back with her partner kneeling above and her ankles around his neck and at no other time, then the specific suggestion will obviously be about that position.

Before direct treatment approaches, this condition was considered highly refractory to intervention. The direct approaches usually reduce anxiety associated with the sexual response, and help the woman learn a new response using principles of successive approximation. Although good results have been reported using systematic desensitization to reduce the anxiety response (19), Haslam's (26) technique with a series of graded dilators inserted in the vagina until the woman is able to retain one the size of a normal penis, then slow insertion of her partner's penis has become a standard approach. Masters and Johnson (41) in addition have the couple take the dilators home to practice. Other therapists have the woman use her fingers or those of her partner as graded dilators. The basic procedure is similar. She first learns to relax, then practices voluntary tensing and relaxing the vaginal vault to give her a feeling of control. After trimming and cleaning her fingernails, she puts a sterile lubricating jelly on her or her partner's fingers and gradually inserts the smallest finger into her vagina while relaxing her muscles as much as possible. Gentle massage, movement, and stretching can be carried out on a regular basis until she can tolerate full finger insertion without discomfort. Larger fingers are used in order, followed by two, three, or more fingers used together. She and/or her partner continue this insertion, stimulation, and stretching until she can tolerate it without discomfort. Intercourse may then be attempted in a number of ways. For example, the male may lie on his back with the female sitting astride him. Using a liberal amount of lubrication, she can slowly move back on his erect penis, and make several partial insertions, withdrawing each time, before finally completely enveloping his penis. This may take several sessions, depending upon the pain, fear, and wishes of the couple. Slow initial movement by her, followed by slow movement of her partner while she remains still may be the next step. Eventually, slow movement by both of them is gradually increased until she is ready to proceed with full active thrusting.

Reports of pain associated with sexual intercourse that interferes with arousal in males are much less frequent than those from females. Usually

the pain is caused by physical pathology. However, Sharpe and Meyer (57) reported a case of a male who complained of unbearable pain in the tip of his penis when he engaged in sexual intercourse and ejaculation. After ruling out organic factors, they instituted a behavioral retraining program with progressively increased attention to penile sensations. At the end of treatment the patient reported no pain and increased frequency and enjoyment of intercourse which was maintained at a three-month follow-up.

Direct approaches to the treatment of males with arousal-erection concerns usually use graded sexual responses in the actual sexual situation if some degree of sexual arousal is reported, or systematic desensitization if no arousal is present. Lazarus (34, 35) has reported successful group desensitization. In one study he assigned patients randomly to different treatment groups: Group desensitization for erection concerns, and psychoanalytically derived interpretive therapy. Patients in the desensitizational group recovered, those in the other group did not. Wolpin (64) described good results with guided imagining, and Cautela (14) successfully used covert negative reinforcement to treat this problem. Other treatment programs have used multimodal treatments, such as the combined use of thought stopping, systematic desensitization, and sexual assertion; techniques of sensate focus and successive approximation of sexual responses (41); or various modifications of the Masters and Johnson procedures (42, 49).

One technique that is particularly useful for males with partial erections, or erection problems of relatively recent origin, is based on paradoxical intention. After permission and information-giving, based on the sexual problem history, the couple is given a series of homework assignments. They are told to spend as much time as possible exploring ways of stimulating or pleasing each other for a week or two and to forget about arousal, erection, and orgasm. Their sessions are to be private, nude, and to include any form of stimulation they want to experiment with, such as hands, feet, oils, whipped cream, mouths, vibrators, tongues, feathers, etc., in whatever manner is mutually agreeable. There is only one restriction: Under no circumstances are they to engage in genital intercourse.

At this point the male client usually heaves a visible sigh of relief, or he may mumble something like, "I can't get it up anyway." The clinician can take advantage of this and respond, "But you see, that's the whole point. You can take advantage of the situation by using this time to explore and experiment with different and novel ideas that you might have thought about, but never had an opportunity to try. If you should happen to have an erection, fine. Enjoy the experience, but *do not* engage in genital intercourse. Remember, your goal at that time is not arousal, erection, or orgasm. If you should happen to experience arousal, appreciate the feeling,

and if either of you feel that you would like to experience orgasm as the result of some particular form of caressing, experience it and enjoy it. However, remember, no genital intercourse. There is always another time."

Relieved of the pressure for performance, the couple is usually eager to follow the suggestions. In many cases, by the time the couple is seen again, they somewhat sheepishly report that they did not quite follow the instructions. The first session is generally reported as initially embarrasing, then fun, and finally arousing. By the second or third session, the male is usually experiencing full erections and having a difficult time refraining from genital intercourse. Many times they are so happy about the situation that they say "to hell with the instructions" and go ahead with genital intercourse. At this point and for those few cases where the couple managed to refrain from genital intercourse but now request it, the clinician can suggest that they continue on the same program for one more week unless the male has an "overpowering urge" to have genital relations, and only then, perhaps he may go ahead. (For variations on this procedure, as well as additional suggestions for this whole area, see Annon, 4, 5.)

High

A high or continual degree of arousal in males (labeled hyperaesthesia or satyriasis) and females (labeled hyperaesthsia or nymphomania) is seldom reported except with injuries or diseases of the spinal cord, which may cause persistent erection of the penis without sexual desire (priapism). In these cases medical treatment is obviously required.

Desire

None or Low

Although the physiological ability to experience arousal and orgasm may be present, the *desire* may be decreased or absent. In the male this is often labeled impotence or hypolibido; in the female it is called frigidity or hypolibido. Usually those who complain of this problem avoid sexual encounters. However, many experience some level of arousal and possibly orgasm if they do engage in sexual activity. As with arousal, constitutional or organic factors may inhibit desire as well as factors like anxiety (29), depression, interpersonal and intrapersonal conflict, traumatic sexual experiences, unwelcomed fantasies, or generalized sexual inhibition. Treatment is usually difficult, although hypnosis (8) and direct approaches like suggesting that the person temporarily refrain from any form of sexual activity but continue to engage in other activities that were at least mildly arousing in

the past, such as sharing romantic dates with a partner, reading erotic stories or books, attending romantic or erotic motion pictures with a partner, etc., as well as various exercises (4) have often been successful in resolving such concerns. There are almost no empirical studies of the factors influencing desire or which forms of treatment are most efficient. It may be hypothesized that many of these problems originate in negative consequences that occur in connection with arousal and/or orgasm experiences. Dealing directly with these experiences and this learned response may influence the cognitive desire.

High

Problems of a high degree of desire in males (labeled hyperaesthesia or satyriasis) and females (labeled hyperaestheia or nymphomania) are often discussed in the clinical literature, but no empirical studies of factors in etiology or treatments appear to be available.

CONCLUSION

The average healthy male and female generally progress through the three areas of response in a sequential manner from desire to arousal to orgasm. When each area is functioning to the satisfaction of the individual, there are no concerns. However, when one area of functioning becomes a problem, it may affect the preceding area of functioning in a hierarchical manner. This knowledge may help therapists in obtaining informaion for the sexual problem history. For example, people with concerns about orgasm, such as none or few for a female or too quickly for a male, may eventually find they are experiencing difficulty in the preceding area of arousal, perhaps a lack of lubrication and pain for the female or erection concerns for males. If this concern over arousal continues, it may then lead to problems of desire, either none or low. With this knowledge, the clinician may offer suggestions for treating areas of inadequate functioning in addition to the initial complaint offered by the patient.

Operant principles offer a partial explanation for the complaint of "different sex drives" or "different libidos" for couples. For example, if the usual sexual response pattern of desire–arousal–orgasm is functioning to the satisfaction of one partner, the probability of that person engaging in the same sexual behaviors is increased. However, if the other person has concerns in one of these areas or experiences negative consequences from

sexual activity, the probability of their wishing to engage in sexual activity decreases. This can lead the couple to blame their differences on "sex drive" rather than to look at the consequences of their sexual behavior for each other.

By using a descriptive classification scheme in conjunction with a sexual problem history instead of the usual lables, the therapist can obtain more relevant information and offer more appropriate treatment suggestions. For example, what does a male client mean when he says he is "impotent"? Is he talking of low desire, erection concerns, reaching orgasm too soon? Is the woman who says she is "frigid" complaining of low desire, no lubrication, pain with intercourse, difficulty or failure in experiencing orgasm? By dispensing with labels and using description, the therapist can make more specific treatment suggestions for specific complaints. For example, most suggestions that traditionally have been offered only to females who have never experienced orgasm through genital intercourse may also be offered to males with similar complaints.

In summary, it is hoped that the descriptive classification scheme, the sexual problem history, and the P-LI-SS-IT model that we have described will provide clinicians a framework within which they may develop and expand their knowledge, experience, and skills in the assessment and treatment of common male and female sexual concerns.

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