Autonomic Nervous System

Part II: Specific Biofeedback Applications

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**Introduction**

This module addresses specific disorders related to dysfunction of the autonomic nervous system (ANS) and the appropriate interventions for these disorders. The autonomic nervous system, composed of the sympathetic and parasympathetic divisions, is responsible for maintaining the normal functions of major physiological systems. Primary examples are the cardiovascular, respiratory, and gastrointestinal systems. Both divisions of the ANS are active in varying degrees at all times. The *sympathetic division* maintains normal levels of tone in the vasculature and also activates the cardiovascular and respiratory systems to respond to physical or psychological stress. The *parasympathetic division* makes a significant contribution to normal digestive processes and other vegetative functions.

Disorders of the autonomic nervous system can target the physiological systems normally controlled by the sympathetic or parasympathetic divisions, including the neurotransmitters and stress hormones that normally mediate transmission of neural/hormonal signals. In this module migraine headache, essential hypertension, cardiac arrhythmias, Raynaud’s disease, and dysfunctional respiration will be defined using established criteria. Medical and behavioral treatments will be discussed. Biofeedback alone or in combination with other behavioral and/or medical therapies is effective in treating each of these disorders. Indeed, scientific research has formed the basis for designing treatment protocols for these disorders.

**Migraine Headache**

The first disorder to be discussed is migraine headache. Both migraine with aura (formerly called “classic migraine”) and migraine without aura (formerly called “common migraine”) will be discussed. The less common variants of migraine are beyond the scope of this module.

**Definition and Characteristics**

A *migraine headache* is a potentially debilitating neurovascular disorder reported by 11 percent of the population, approximately three females for every male. Prior to puberty, prevalence is equal in boys and girls. First migraine usually occurs in middle childhood, adolescence, or early adulthood and less frequently in middle adulthood (Lipton & Stewart, 1993).
Frequency may be once or twice a month; some patients report two or three debilitating headaches per year. Migraine headaches are characterized by unilateral, throbbing, pulsating pain ranging in severity from moderate to severe. The pain may last anywhere from four hours to as long as three days, with the usual duration less than one day. Associated with the pain in many patients are nausea, vomiting, photophobia, and intensification during physical activity. A prodrome—characterized by increased or decreased appetite and changes in usual sleep patterns or normal moods—can occur one or two days before a migraine (Cady et al., 2002).

In about 15 percent of patients with migraine, the headache is preceded by neurological symptoms primarily visual in nature; such patients have *migraine with aura*. The aura can consist of tunnel or blurred vision, a feeling of apprehension, or floating spots in front of the eyes (*scotoma*). If the aura occurs, pain can be expected within one hour. When these warning symptoms are absent from the clinical presentation, the diagnosis is likely to be *migraine without aura* (International Headache Society, 1988; Silberstein & Rosenberg, 2000).

Migraine headaches differ from tension-type headaches in the type and frequency of pain, the influence of family history, the presence of associated features, and the patient’s response to prescription and over-the-counter drug therapy. Tension-type headaches are covered in detail in Module 4.

Migraine may adversely affect a person’s quality of life. Patients lose work or school time and cannot engage in family, social, or leisure activities. The Migraine Disability Assessment Tool (MIDAS) is a quick questionnaire useful for evaluating the effects of pain on productivity, absenteeism, and leisure activities. The composite score indicates the impact of pain on the individual’s daily life (Stewart et al., 1999). The MIDAS can be repeated during therapy to track progress.

**Physiology of Migraine Headache**

The physiology of migraine is complex and requires an in-depth background in neurophysiology that is beyond the scope of this module. However, a basic understanding of mechanisms is necessary to guide the design of therapy and to educate patients during psychophysiological therapy.
The first link in the migraine chain involves family history. Although no one specific gene has been identified, there is a strong tendency for migraines to run in families. The second link in the chain involves some type of a triggering mechanism to set off the headache. Rather than thinking of the trigger as a single event, it is more helpful to think of it as a cumulative effect of a variety of factors.

Among the many triggers of a migraine headache may be the occurrence of a tension-type headache. If an individual has experienced migraines, then any type of head pain may set the stage for the development of a migraine. Some clinicians believe that when a migraine patient has a tension-type headache, the apprehension and fear of a possible migraine may actually enhance the probability of developing a migraine. Thus one way that tension-type and migraine headaches may overlap is when the patient believes that the tension-type headache is actually the beginning of a migraine. Tension-type headaches can also play a role in causing a migraine since tension-type headaches are often related to excessive muscle activity. Contracted muscles impinge on arterioles and decrease blood flow to those muscles; the resulting ischemia is a pain producer. The combination of reduced blood supply and increased muscle activity produces a burning, throbbing pain.

Additional triggers related to the development of a migraine are dietary in nature. As a group, the foods that are most likely to trigger a migraine are referred to as vasoactive. Vasoactive foods are foods that cause the diameter of the arteries to change. Alcohol, for example, causes both vasodilation and vasoconstriction depending on the length of time since the alcohol was ingested. Caffeine is a vasoconstrictor that can lessen the pain of migraine during an acute attack. Some migraine patients report an association between the ingestion of monosodium glutamate and the onset of a migraine headache. Other patients may be sensitive to tyramine, found in such substances as aged cheeses and red wine. Still other patients may be sensitive to nitrites, used as preservatives in packaged luncheon meats.

The role played by foods in migraine genesis can best be determined by taking a thorough history and undertaking an elimination diet. By selecting certain foods and totally eliminating them for a period of time, the patient may be able to reduce migraine activity. If this decrease occurs, then the
foods in that group should be reintroduced one at a time and migraine activity should be carefully assessed. If a particular food is eaten and migraine activity increases, then that food becomes suspect and should once again be eliminated.

The following analogy can be used in explaining the effects of migraine triggers to patients. Imagine that you are an empty bucket. Above the bucket there are a number of different hoses and each hose is pouring migraine triggers into you, the empty bucket. The first hose may be various foods; the second hose may be a lack of sleep; and the third hose may represent psychological stress. As the hoses pour migraine triggers into the bucket, the bucket gradually fills. You will have your migraine whenever your bucket overflows. This analogy is useful because it points out to the patient that rarely will one particular trigger be the sole source of his or her migraines. If this were the case, the elimination of this trigger would prevent any future migraines. It is much better to see migraine as culminating from the interaction of several factors and the therapeutic goal as turning off as many hoses as possible. Improved control is possible even without shutting off all of the hoses.

Migraine headache is not primarily a vascular disorder, as previously thought, but a neurovascular disorder most likely localized in the brain nuclei that influence sensory input and cranial vessels. Several factors are major participants in the generation of migraine pain: the intracranial blood vessels and their neural connections (the trigeminovascular system); the ophthalmic branch of the trigeminal nerve; the trigeminal nucleus; and the brain stem nuclei that control the processing of signals emanating from the trigeminal system. The migrainous brain has a fundamental hyperexcitability, making it vulnerable to specific triggers such as irregular sleep, foods, and psychological stress. Neural events are associated with the dilation of blood vessels, resulting in pain and increased sensitivity to sensory stimuli (Goadsby, Lipton, & Ferrari, 2002).

The prodrome is experienced by most patients with migraine and can include mood changes, food craving, yawning, and muscle aches. The prodrome occurs one or two days or several hours before the headache. In contrast, the migraine aura is a localized neurological event experienced by 10–20 percent of migraine patients less than one hour before the pain
begins. The flashing lights or jagged lines experienced as visual aura may be explained by spreading electrical depression in the brain. This is followed by a wave of *oligemia* (reduced blood flow due to vasoconstriction) that also spreads across the cortex.

Stimulation of the trigeminal system causes the release of Substance P and other peptide chemicals that cause pain and vasodilation. Dilated blood vessels play an important role in generating the throbbing pain characteristic of migraine. Lower than normal levels of serotonin have been noted during migraine attacks, contributing to the vasodilation of cranial blood vessels and greater sensitivity of parts of the trigeminal nerve. Normally the brain stem nuclei monitor and modulate sensory input and then send signals to the cranial blood vessels to regulate their diameter. In migraine, however, this modulation may be ineffective and sensitivity to multiple stimuli is increased. Although the pain-generating process in migraine is not completely understood, it has been proposed that the *nociceptors* (pain receptors) of the intracranial structures are acutely sensitive and there is poorer functioning of the endogenous pain control pathways that normally block pain signals (Hamel, 1999). Many patients experience a *postdrome* period after the pain has resolved. Muscle pain, stiffness, problems with concentration, and fatigue lengthen the period of disability and delay the return to optimal functioning.

To set the stage for understanding the rationale for utilizing biofeedback, it is important to emphasize that migraine does not always progress through all of these stages, including the stage of severe pain. If the increased sensitivity to stimuli or the initial vasoconstriction is prevented, then the overdilation and the entire migraine episode can be prevented. The pathway from prodrome to postdrome can be interrupted at several different points.

**Drug Therapy**

Drug therapy is currently based on preventing migraine and managing acute attacks. Nonpharmacological therapies—whether they involve dietary restriction, exercise, or biofeedback and relaxation—are focused on prevention. Rarely will a nonpharmacological intervention dramatically reduce pain during an acute migraine attack.
Preventive drug therapy should be considered when attacks are frequent, increasing in frequency, or poorly controlled by acute medication. Preventive therapy may decrease the sensitivity of the brain to triggering stimuli. Types of drug therapy prescribed for daily use include such products as beta-adrenergic receptor antagonists (Inderal, Valproate), the specific serotonin reuptake inhibitors (Zoloft, Prozac), and the nonsteroidal anti inflammatory drugs (Motrin, Advil). According to Goadsby, Lipton, and Ferrari (2002), possible side-effects of the preventive medicines include drowsiness (Valproate), fatigue (Inderol), and anxiety and insomnia (Zoloft).

In an acute migraine attack, some patients respond well to nonprescription analgesics and nonsteroidal anti inflammatory drugs. Dosage should be adequate but patients should avoid using medicine more than two or three days per week. Ergot derivatives such as Cafergot were used for many years and are effective, but patients taking these drugs are plagued by side-effects. Newer drugs include the triptans; these drugs are serotonin 5-HT\textsubscript{1B/1D} receptor agonists. The first of these drugs developed was sumatriptan (Imitrex), a serotonin analogue, which may act by facilitating cranial vasoconstriction and inhibiting peripheral or trigeminocervical neural activity. The second-generation triptans (such as Zomig and Amerge) produce better pain relief and lower recurrence rates. Theoretically drugs that decrease the excitability of the trigeminal neurons or produce vasoconstriction during the pain stage of the migraine should be beneficial in relieving the pain. Drugs such as Cafergot, which consist of a combination of caffeine and ergotamine, can relieve pain because Cafergot produces vasoconstriction of the overdilated arteries. The recent popularity of the triptans can be attributed to their receptor specificity, which greatly improves effectiveness. Silberstein, Saper, and Freitag (2001) identify possible side-effects of the abortive medicines such as gastritis (nonsteroidal antiinflammatory drugs) or dizziness and sleepiness (triptans).

It is imperative, however, that medication is taken at the right time. For example, taking Imitrex every morning—indeed of having or not having a headache—can actually initiate a migraine, and taking Inderal while having a migraine can potentially make the pain much worse. Therefore timing is very important and patients must understand how to properly utilize their medications.
Biofeedback-Assisted Relaxation Therapy

Many clinical research trials have shown that biofeedback combined with relaxation is efficacious in preventing migraine with and without aura (Penzien, Rains, & Andrasik, 2002). The rationale for the use of biofeedback relies on our understanding of the pathophysiology of migraine and the role of psychological stress in the etiology of migraine. If the patient’s physiological responses to stress can be prevented, then the entire headache episode can be avoided. If stress management improves and a relaxed, calm mental state becomes a person’s “norm” rather than the exception, the oversensitivity of the brain may decrease. Relaxation therapy facilitated by temperature feedback produces vasodilation similar to that produced by the drug Inderal at the peripheral level, but there is no evidence that temperature feedback produces a systemic vasodilation (Holroyd & Penzien, 1990). Normalizing the hyperexcitable state of the brain and improving the control of pain with biofeedback-assisted relaxation may be more logical explanations than those based solely on the vascular hypothesis of migraine.

The biofeedback modality of choice in migraine prevention is temperature feedback (Blanchard et al., 1990). However, some practitioners think that temperature feedback should not be the first modality of feedback but should follow muscle feedback (SEMG). Patients find it easier to relax muscles than to warm their hands; muscle relaxation seems to be conducive to hand warming. In addition, after years of recurrent migraine attacks, patients may develop a bracing posture in which the muscles of the shoulders and neck become chronically tense—a situation that can be aggravated by stress, during which additional bracing can occur. Learning to relax these muscles decreases the risk of both migraine and tension-type headaches.

Preliminary data are encouraging for the use of neurofeedback in treating migraine headache. Because slow cortical potentials are altered in patients who suffer from migraine, these potentials can be used as a feedback signal. Learning to decrease the exaggerated negative amplitude of cortical potentials is associated with less excitability of the brain, faster habituation to stimuli, and lower arousal. Decreases in frequency and duration of headache are concurrent with lower negative slow cortical potentials (Kropp, Siniatchkin, & Gerber, 2002).
The sequencing of feedback modalities can be determined by the individual practitioner, but temperature feedback should be an integral part of any training program for patients with migraine. The goal of temperature feedback should be to make the hands as warm as possible, with a goal of 95 degrees. To help accomplish these goals, relaxation training (described in detail in Module 8) in both the clinic and at home becomes important. Home training can be accomplished through the use of relaxation audio-tapes. Training should also include the ability to hand warm while under stressful conditions and in the absence of any artificial feedback. An emphasis should be placed on efficiency such that the migraine patient can warm the hands quickly following cooling related to stress. Home trainers are available for temperature feedback to facilitate the acquisition of hand-warming skills.

**Patient Education and Counseling**

Education should accompany the biofeedback training. Using the bucket analogy described earlier, the focus of education is to turn off as many hoses as possible. Problem solving can help identify triggers and change behaviors associated with the stressor. The importance of coping with stress should be addressed in therapy sessions and the clinician should offer suggestions for more effective coping strategies.

Counseling or psychotherapy is an essential component of therapy for some patients—for example, those who have an underlying psychopathology. Frequently depression and anxiety are part of the clinical picture in migraine patients, particularly those who have experienced migraine for many years. Assessment for psychiatric disorders should be conducted by practitioners who are licensed to conduct this type of diagnostic evaluation. Where mood or anxiety disorders are identified, more extensive medical or psychotherapeutic therapy may be required in conjunction with biofeedback and relaxation therapy (Silberstein, 2001).

**Summary**

There is substantial empirical evidence that relaxation therapy, SEMG and thermal biofeedback, and cognitive-behavioral therapy are associated with clinically significant improvements in migraine headache activity (Andrasik, 1996; McGrady et al., 1999). Migraine is a prevalent and persistent problem that is not likely to respond to one individual treatment
modality. Combined medical management, patient education, and behavioral therapy will promote better control and improved quality of life for the patient with migraine.

Hypertension

Hypertension refers to high blood pressure. The most common form of hypertension is essential hypertension—that is, a high blood pressure of unknown etiology since no ongoing disease process or trauma to the cardiovascular system can account for the elevated blood pressure. Secondary hypertension, which is much less common, refers to an elevated blood pressure due to a specific disease of the kidney, adrenal glands, or thyroid (to name a few possibilities). White coat hypertension is characterized by elevated blood pressure readings in the office or clinic setting but normal blood pressure readings at home or outside the clinic.

Approximately 4 percent of adults aged 18–34 have sustained elevations in blood pressure sufficient to merit the diagnosis of essential hypertension. Individuals of African American or Hispanic ancestry are at the highest risk for developing hypertension. Lifestyle factors—such as smoking, inactivity, high salt diet, obesity, and psychological stress—confer risk to the cardiovascular system, sometimes resulting in chronically high blood pressure.

Normal Regulation of Blood Pressure

Blood pressure is regulated by a complex interaction of cardiac, vascular, neural, and endocrine factors. The two major factors are cardiac output and total peripheral resistance. Cardiac output is the product of heart rate (beats per minute) and stroke volume (the amount of blood ejected during one beat of the heart). Total peripheral resistance represents the difficulty that blood has in passing through tissues and is chiefly determined by the diameter of the arterioles. Another important factor that influences blood pressure is the volume of blood in the circulatory system. The kidney maintains long-term control of blood pressure by altering the blood volume.

As one variable (such as heart rate) moves upward, which would tend to raise blood pressure, a change in another variable may compensate. For example, an increase in heart rate might be offset by the dilation of the peripheral arteries. Whereas increased heart rate will by itself raise blood
pressure, peripheral dilation decreases resistance and thereby lowers blood pressure. If they occur simultaneously, there may be no noticeable change in pressure. However, if a number of factors controlling blood pressure all move in the direction of raising the pressure, then an increase would be expected (Vander, Sherman, & Luciano, 2001).

An interaction between muscle tension and peripheral vasoconstriction can potentially influence blood pressure. For example, the combination of excessive muscle contraction and constriction of the peripheral arteries can produce a quick elevation of blood pressure. A typical stress response—consisting of increased muscle tension, constriction of the peripheral arterioles, and increased heart rate—serves as a good example of how a combination of factors increases blood pressure. If the blood pressure elevation persists, the risk of certain life-threatening consequences increases proportionately. For example, as systolic blood pressure rises, the probability of rupturing a weakened artery increases. If the artery that ruptures happens to be a major blood supply to the heart, then a heart attack ensues. If (on the other hand) the ruptured artery provides blood to the brain, then a stroke occurs.

There is also evidence that the cardiovascular system can “learn” to maintain elevated blood pressure. In the arch of the aorta, there are specialized cells referred to as baroreceptors (pressure receptors) that play a role in maintaining a particular level of blood pressure. If an individual’s blood pressure remains high over a period of time, then baroreceptor cells recalibrate and serve to maintain this elevated pressure.

**Lifestyle Factors**

Lifestyle factors are important to consider in the development and maintenance of high blood pressure. Caffeine and nicotine increase heart rate and constrict peripheral arteries. Caffeine is present in many medications such as Excedrin. Nicotine is a potent vasoconstrictor and cardioaccelerator that has the potential for elevating blood pressure. High-sodium foods promote fluid retention, increasing blood volume and raising blood pressure. The active ingredient in black licorice is a vasoconstrictor. A sedentary lifestyle that is lacking in aerobic exercise has been associated with elevated blood pressure. Obesity, which is often associated with inactivity, can elevate blood pressure through at least two mechanisms. Simply
carrying the extra weight by itself is very likely to cause blood pressure to rise. In addition, extra weight is composed of living tissue that requires its own blood supply; the added arteries and veins increase peripheral resistance. Thus patients with essential hypertension should be assessed for contributory lifestyle factors and counseled as appropriate (Joint National Committee VI, 1997).

**Range of Blood Pressure in Essential Hypertension**

A committee of hypertension experts that meets periodically to review diagnostic criteria and treatment defined four ranges of blood pressure (Joint National Committee VI, 1997).

- Stage 1 hypertension is defined as a systolic pressure in the 140–159 mm Hg range and a diastolic pressure between 90 and 99 in a person with no other cardiovascular or endocrine problems.
- Stage 2 hypertension is based on a systolic pressure of 160–179 and a diastolic pressure between 100 and 109.
- Stage 3 and Stage 4 hypertension are defined as pressures over 180–210/110–120.

Currently many patients with Stages 1–2 and all patients with Stages 3–4 hypertension are prescribed medication. Furthermore, it is unlikely that a patient who has Stage 3 or Stage 4 hypertension and is not treated with medication will present for behavioral treatment. Should this occur, the practitioner must recommend immediate consultation with a physician.

The major classes of antihypertensives are diuretics, beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor antagonists, and calcium channel blockers. Based on a prior understanding of the physiological control systems in regulating blood pressure, a brief explanation of the effects of the hypertensives follows (see Cushman, 2001; International Society of Hypertension, 1999).

- Diuretics increase sodium and water removal by the kidneys, thus decreasing blood volume and total peripheral resistance.
- Beta-blockers reduce cardiac output.
- Angiotensin-converting enzyme inhibitors lower resistance and decrease retention of sodium.
• Calcium channel blockers inhibit blood vessel wall constriction and thereby decrease peripheral resistance.

As always, patient education is an important part of therapy. Patients should be taught to measure their own blood pressure and monitor it daily. With this information they can track trends, seasonal influences, dietary effects, and the benefits of activity. In addition, normal home pressures coexisting with elevated clinical pressures can support the diagnosis of white coat hypertension, thus decreasing the necessity for medication (Nakao et al., 2000). Repeated measurements of blood pressure, both in the clinic and at home, are essential prior to initiating drug or behavioral therapy for hypertension. In fact, research shows that daily blood pressure monitoring for one month may lower blood pressure significantly in about one-third of hypertensive patients (McGrady & Linden, 2003). Patient education should also address the use of caffeine and alcohol, smoking, body weight, and physical activity.

**Biofeedback Treatment of Hypertension**

Several biofeedback modalities are useful in reducing blood pressure and/or the need for antihypertensive medication. SEMG feedback can be used to teach the individual to relax excessively contracted skeletal muscles. SEMG feedback is important when one recognizes that strong muscle contractions have the potential for producing a state of ischemia. By teaching an individual to relax the muscles through SEMG feedback, more blood flows to the muscles, thus reducing peripheral resistance and blood pressure. However, the arteries and arterioles are surrounded by smooth muscles, and it is through the contraction of these smooth muscles that these vessels actually constrict. General relaxation of skeletal and smooth muscles can have a positive effect on blood pressure (McGrady & Linden, 2003).

Skin temperature feedback can teach the hypertensive patient to dilate the peripheral arterioles (Blanchard, 1990). Temperature training is often coupled with passive relaxation therapy to assist hypertensive patients in warming their hands to 95 degrees. After several training sessions, challenges in the form of stressful imagery or mental arithmetic are introduced and the patient practices warming his or her hands under these conditions. The patient is encouraged to be aware of hand temperatures throughout the
day and is instructed to utilize the hand-warming skills learned in the clinic setting when he or she notices a falling hand temperature. The goal is to prevent excessive vasoconstriction during stressful situations.

Despite the effectiveness of thermal feedback in facilitating decreased blood pressure in more than 50 percent of hypertensive patients who complete treatment, some researchers argue that the skin is not a major vascular bed. Because changes in hand temperature cannot fully explain decreases in blood pressure, identifying the exact mechanism that explains the beneficial effects of temperature feedback requires further research. It may be that thermal feedback facilitates the downregulation of sympathetic nervous system activity, which has major implications for lowering heart rate and blood pressure (McGrady, 1994).

The rationale for electrodermal feedback relates to the sympathetically mediated sweating response that occurs during stressful or anxiety-producing situations. Electrodermal activity fluctuates according to the volume of perspiration. By teaching the individual to reduce sweating and thereby the electrodermal response, the therapist is actually using the feedback as an indicator of sympathetic arousal. Learning to reduce electrodermal activity mediates better control over stress-related responses and indirectly lowers blood pressure (Patel & Marmot, 1988).

Heart rate or pulse feedback can also be utilized as part of a comprehensive approach to controlling elevated blood pressure. Heart rate feedback is often provided by a photoplethysmograph that reports heart rate information to the patient as a visual or auditory signal. A transducer is attached to one of the fingers such that a light is directed through the skin of the finger. This light is reflected from the underlying blood back to the transducer, generating an electrical signal. As the heart contracts and blood surges through the artery, there is a change in the reflected light that is interpreted by the instruments as a heart beat. Heart rate biofeedback is helpful for those patients who manifest their anxiety through significant elevations of heart rate. Because anxiety is often associated with elevated blood pressure, training the patient to control his or her heart rate can have a positive effect on blood pressure levels.

Direct feedback of systolic or diastolic blood pressure has also been used successfully to treat high blood pressure. Blood pressure feedback is
best combined with relaxation therapy and repeated home monitoring of blood pressure (Glasgow, Engel, & D’Lugoff, 1989). This type of feedback requires more complex instrumentation, more sessions, and a greater commitment on the part of the patient. Compared to the use of SEMG, temperature, and electrodermal feedback, few clinics are prepared to provide direct feedback of blood pressure. It must be remembered, however, that daily self-monitoring of blood pressure provides a type of direct blood pressure feedback and thereby important information for both the patient and the practitioner.

The most positive effect on blood pressure is obtained if the patient simultaneously reduces muscle tension, dilates peripheral arteries, and slows the heart rate. Through a technique called *yoking*, the biofeedback therapist can arrange a feedback signal that changes only if more than one physiological parameter moves in the right direction. For example, the pitch of a tone might drop only if muscle activity drops and arterioles dilate and heart rate drops. Thus a simple signal is used to reflect a relatively complex sequence of physiological events. The yoking technique is usually used after each modality has been mastered and requires sophisticated computerized feedback equipment.

The practitioner should consider potential interactions between the medications that the hypertensive patient is taking and biofeedback training. For example, the patient may be taking a beta-blocker such as Inderal. Since the beta-blocker slows heart rate and indirectly decreases peripheral resistance, the biofeedback therapist might erroneously conclude that the patient taking Inderal does not need relaxation therapy or temperature feedback. Although the patient taking Inderal has a slower heart rate and starting hand temperature of 92 degrees, this patient still needs to acquire the skill of lowering arousal. The therapist should provide temperature feedback to train lower arousal with a goal temperature of 95 degrees.

Clinically it is important to recognize that the correct modality cannot be prescribed in advance but must be determined as a result of an initial assessment of the individual patient. Thus the patient who tends to respond to stress with excessive muscle activity becomes a candidate for SEMG feedback, while the person with consistently cold hands may benefit most from temperature feedback. Additional therapies—such as problem solving
or anger management—can be incorporated if necessary (Linden, Lenz, & Con, 2001). Therefore an initial psychophysiological profile is recommended to determine the best choice of biofeedback and relaxation therapies. Research indicates that six types of feedback (SEMG, temperature, electrodermal, direct blood pressure, respiratory sinus arrhythmia, and heart rate) have all been used successfully to treat either essential or white coat hypertension (McGrady, Bourey, & Bailey, 2003).

Once the basic self-regulation skills have been mastered for each modality of feedback provided, it may be necessary to “desensitize” the learner to a variety of psychological stimuli associated with high blood pressure such as white laboratory coats, the doctor’s office, and the process of having the blood pressure measured (Nakao et al., 2000). Desensitizing a learner to the blood pressure apparatus can occur when self-regulation skills have been mastered under nonstressful conditions. The therapist might begin by placing the blood pressure apparatus on a table across the room from the learner while monitoring the most reactive feedback modalities. If baseline temperatures decline with the presentation of the blood pressure apparatus, then feedback and coaching are provided to restore normal hand temperatures. The apparatus is then moved progressively closer to the learner. Finally the cuff is placed on the learner’s arm (not the arm with the thermistor) and his or her blood pressure is taken. This desensitization process may need to be repeated during several biofeedback sessions before the learner is able to tolerate having his or her blood pressure taken without experiencing a significant temperature decline. The purpose of this desensitization process is to prevent a rise in blood pressure due solely to its measurement, since false “highs” may influence the physician to prescribe unnecessary medication.

A typical 8–10 session treatment protocol for a hypertensive patient comprises:

- Interview.
- Assessment of psychosocial factors relevant to blood pressure.
- Brief medical history.
- Psychophysiological profile.
- Blood pressure monitoring (clinic and self) and use of log sheets.
• Relaxation training (deep breathing and autogenic).
• Biofeedback (one or more modalities).
• Desensitization if necessary.
• Problem solving about psychological factors that increase blood pressure.

Follow-up consists of monthly refresher sessions of problem-solving and biofeedback-assisted relaxation for three to six months. The patient should expect to continue using relaxation therapy if the lower blood pressure achieved during active treatment is to be maintained for the long term (Linden, Lenz, & Con, 2001).

It is important to be sure that a comprehensive approach to blood pressure reduction is implemented by the patient. Smoking cessation, weight control, exercise, and relaxation training through biofeedback may all be needed to achieve the optimal decrease in blood pressure. Biofeedback training alone is rarely effective (Spence et al., 1999). Clinical research supports significant decreases in blood pressure with good long-term maintenance when multicomponent therapy is used in hypertensive patients. However, the patient’s physician should be consulted prior to beginning any exercise program or weight loss initiative.

**Cardiac Arrhythmias**

The pacing of the heart (beats per minute) is controlled by two electrical centers that have different levels of intrinsic excitability. Each atrium has a *sinoatrial node* with the fastest inherent excitability, and each ventricle has an *atrial ventricular node*. As the atrium fills with blood, the sinoatrial node notifies the atrial ventricular node that a supply of blood is about to arrive in the ventricle. This message is designed to ensure that each chamber of the heart waits until it is properly filled with blood before contracting and expelling the blood. Since the sinus node is typically the most excitable region of the heart, the cardiac cycle begins there. The electrical signal is transmitted through the atria, the atrial ventricular node, and then through the ventricles via the heart muscle itself instead of by an external nerve. Muscle contraction follows the passage of the electrical signal. Under normal resting conditions, the sympathetic nervous system activates
the sinoatrial node to fire at a rate of 60–90 beats per minute. Heart rates above 90 at rest are called \textit{tachycardia} and those below 60 are termed \textit{bradycardia} (Vander, Sherman, & Luciano, 2001).

\textit{Arrhythmias} are disruptions in the normal rhythm of the heart that may originate in the sinoatrial or atrial ventricular nodes, atria, or ventricles. The primary care physician or cardiologist makes the diagnosis of arrhythmia based on an electrocardiogram. Premature atrial contraction can result from a conduction problem in the atria. \textit{Atrial fibrillation} is defined as an atrial rate greater than 350 beats per minute. Heart block occurs when conduction across the atrial ventricular node region is delayed. Irregular rhythm in the ventricles is usually more serious than in the atria because the pumping action of the heart may be compromised. \textit{Premature ventricular contractions} are caused by an abnormal pacemaker—that is, an area other than the sinus node activating the heart beat. The heart skips a beat while the ventricles reestablish their normal rhythm (Harvey et al., 1992). As a result of a heart attack, muscle tissue in the heart dies and becomes scar tissue. Scar tissue does not volume conduct in the same way as healthy heart muscle, resulting in disruption of the transmission of the message. \textit{Respiratory sinus arrhythmia} (RSA) is the variation in heart rate that occurs during inspiration and expiration and has been suggested as an index of parasympathetic tone (Porges, 1986). Reduced heart rate variability is an important risk factor for mortality and life-threatening cardiac arrhythmias (Poulsen et al., 2001).

\textbf{Treatment Modalities}

Medical management of the arrhythmias is driven by the need to reduce or eliminate the beats emanating from a region other than the sinoatrial node and restore normal sinus rhythm. There are four types of therapy: implanting an artificial pacemaker, pharmacotherapy, education, and behavioral therapy.

Pacemakers are surgically implanted and calibrated to maintain a normal range of heart rate at rest and during activity. In essence, the pacemaker artificially replaces the missing message.

Antiarrhythmic drugs are divided into four classes, grouped according to their major effects on the cardiac electrical signal (Harvey et al., 1992).
• Class I drugs (e.g., lidocaine) act similarly to local anesthetics and depress excitability of cardiac tissue.

• Class II drugs (e.g., beta-blockers) suppress abnormal pacing, particularly at the atrial ventricular node.

• Class III agents (e.g., amiodarone) slow conduction of the electrical impulse.

• Class IV drugs (e.g., verapamil and digitalis) likewise slow conduction of the electrical impulse.

Education is an important part of the overall management of cardiac arrhythmia. It is crucial to explain the basics of electrical activity conduction to the patient and define the particular abnormality specific to that patient in understandable terms. Stimulants such as caffeine in any form and nicotine must be eliminated. An exercise program should be implemented after consultation with the appropriate care provider. Depending on the contribution of psychological stress or emotional illness to the problem, a counselor or psychotherapist should be included in the treatment team.

Early research has shown that it is possible to improve arrhythmias through deep breathing training and RSA feedback. Van Dixhoorn developed a treatment protocol that utilizes breath control to decrease sympathetic arousal and increase vagal (parasympathetic) activity, which in turn decreases heart rate and stabilizes cardiac rhythms (see Van Dixhoorn et al., 1987). RSA feedback promotes increased amplitude of the variability of the sinus rhythm (Lehrer, Vaschillo, & Vaschillo, 2000). Despite promising preliminary results, there are no randomized clinical trials to support the clinical use of RSA feedback in cardiac arrhythmia.

As with hypertension, the original assessment and diagnosis must be carried out by a physician. The treatment plan is best designed with careful attention to the severity of the abnormality in heart rate and rhythm, the patient’s response to medical management, and the impact of psychological stress on the etiology and maintenance of the disorder. Applied psychophysiological therapy that incorporates breathing training, relaxation, and biofeedback has the theoretical basis and potential to contribute significantly to the management of cardiac arrhythmias, but more research is needed to establish clinical efficacy.
Raynaud’s Disease

First identified by Maurice Raynaud in 1862, Raynaud’s disease is a disorder of the peripheral circulation system characterized by episodic contractions of the blood vessels (vasospasms) in the fingers or toes. The symptoms of Raynaud’s involve discoloration in the extremities and pain. The discoloration may involve a progression consisting of whiteness (palor or blanching), blueness (cyanosis), and redness (rubor or reactive hyperemia) depending on the particular stage of Raynaud’s being experienced at that time. These color changes are attributed to spasms in the arterioles of the fingers and toes, which in turn are triggered by exposure to cold such as might occur when selecting frozen foods at a supermarket. Indeed, Raynaud’s patients often wear gloves in order to avoid triggering these painful spasms. The spasms may last from minutes to hours; some researchers believe that Raynaud’s spasms can also be triggered by emotional stress, but the evidence for this is inconsistent.

Types and Prevalence

There are two major varieties of Raynaud’s: Raynaud’s disease and Raynaud’s phenomenon. Raynaud’s disease is also known as primary Raynaud’s and this label is used whenever the symptoms of Raynaud’s have no known medical cause. Raynaud’s phenomenon is the proper diagnosis to use when the symptoms of Raynaud’s are known to be secondary to some type of medical condition such as rheumatoid arthritis, systemic lupus erythematosus, scleroderma, trauma, or diseases such as carpal tunnel syndrome or thoracic outlet disorder. Raynaud’s phenomenon can also result from various drugs that may be spasm inducing. More women experience Raynaud’s phenomenon than men: the ratio varies from three to one to four to one depending on the particular study.

The prevalence of Raynaud’s disease is not well established, but estimates run from 4.3 percent of women in the southern United States to 19 percent in the United Kingdom (Silman et al., 1990). Some theorists believe that the symptoms of Raynaud’s disease are the result of excessive activity in the sympathetic nervous system. Others (historically Sir Thomas Lewis in 1929) believe that there may be some type of faulty mechanism in the digital arteries. Still other researchers believe that Raynaud’s disease results from a combination of both of these factors. Some researchers speculate on
the role of blood viscosity or other blood chemistry factors. At this point there is no definitive, widely accepted explanation for the symptoms of Raynaud’s and therefore the term “idiopathic” seems appropriate; however, more recent evidence seems to favor the Lewis hypothesis. For example, Freedman and, Baer, and Mays (1995) have recently demonstrated that a defect in the alpha 2-adrenergic receptors (but not the alpha 1 receptor) triggers vasospasm in Raynaud’s phenomenon.

**Biofeedback Treatment of Raynaud’s Disease**

Most researchers believe that biofeedback should be utilized for Raynaud’s disease rather than Raynaud’s phenomenon. Various researchers have reported on the use of thermal biofeedback and other behavioral treatments for Raynaud’s (Grove & Belanger, 1983; Rose & Carlson, 1987; Sedlacek, 1984, 1989). Since most cases of Raynaud’s are of the Raynaud’s disease type, this means that biofeedback holds some promise for most Raynaud’s patients. The modality of biofeedback most appropriate for Raynaud’s is temperature feedback with the goal of warming both the hands and feet to normal levels. There has been no research indicating that there is anything inherent in Raynaud’s disease that precludes the possibility of hand and foot warming. Indeed, clinical experience indicates that many Raynaud’s patients can successfully warm their hands to 95 degrees.

Because Raynaud’s involves excessive constriction of peripheral arteries, it is prudent to counsel the Raynaud’s patient that exposure to vasoactive effects (such as caffeine) should be minimized or eliminated. To the extent that excessive arousal in the sympathetic nervous system plays a role in Raynaud’s symptoms, stress management counseling also becomes a viable therapy although most studies have not demonstrated a stress-Raynaud’s disease link (Freedman & Ianni, 1983). Medications that produce peripheral vasoconstriction should also be minimized or avoided. Examples of these types of medications would be ergot preparations such as Cafergot and pain-killing medications that contain significant levels of caffeine.

In an elegant series of studies, Freedman (1991) and his colleagues have demonstrated that:

- Finger warming is achievable in a variety of settings.
• Finger-cooling (but not finger-warming) ability is stopped by sympathetic blockade.

• Finger-warming ability is blocked by a nonneural beta-adrenergic blocker.

The implications of these findings for thermal biofeedback are significant. For the patient with ongoing vasospasm (e.g., temperatures in the 70s), initial warming may occur mainly through a decrease in sympathetically mediated vasoconstriction. However, increases in temperature above the usual normal range (86–88 degrees) are likely to be based on a centrally mediated systemic response that does not involve the sympathetic nerve, but is probably a blood-borne hormonal adrenergic response.

In AAPB’s 1992 Standards and Guidelines (now out of print), Raynaud’s was included as a disorder appropriately treated by biofeedback. A recent multisite trial calls this conclusion into question (Raynaud’s Treatment Study Investigators, 2000), but it appears that some treatment sites did obtain meaningful results (Middaugh et al., 2001). The most extensively researched protocol combines thermal feedback with a cold stressor challenge in order to improve the training results. The cold stressor involves some way of cooling the extremities—such as the use of cold water or a stream of cold air—and then teaching the patient to quickly recover normal hand temperatures. The use of cold stressor challenges is not a standard component of many biofeedback programs, but it does appear in published studies and provides further evidence regarding the efficacy of biofeedback. Freedman, Lynn, and Ianni (1982) developed a comprehensive history questionnaire for Raynaud’s disease. It is very important that medical tests establish the diagnosis as Raynaud’s disease before beginning treatment.

Many clinicians have incorporated a procedure that is helpful when dealing with difficult cases. If you find after a reasonable number of temperature feedback sessions that hand warming is minimal, you may wish to accelerate the process by using one of the following procedures to warm the patient’s hands to approximately normal temperatures.

• Use a standard heater-type fan.

• Ask the patient to place his or her hands in warm water.

• Ask the patient to hold a cup containing a hot drink.
Then provide biofeedback designed to assist the patient in learning to slow the cooling process. It has been observed that slowing the cooling process is an easier skill to master than hand warming. Once the patient has learned to maintain 95-degree hand temperatures over an extended period of time using artificial warming, the next step is to gradually withdraw the artificial heat as the patient maintains normal peripheral vasodilation. Some clinicians have found this approach to be quite effective in their training protocols, and this has allowed them to be successful with Raynaud’s patients where other techniques have failed.

**Summary**

The proper treatment of Raynaud’s should include more than simply biofeedback. All of those factors in the patient’s life that contribute to peripheral vasoconstriction need to be explored and, if possible, brought under control. If the patient can avoid exposure to cold or stress triggers, this should be encouraged. Minimizing the use of vasoconstricting medications should also be encouraged whenever possible. The patient needs to be taught not only what to do but why these particular steps are important in controlling the symptoms.

It also needs to be stressed to the patient that this is a management technique for the symptoms of Raynaud’s, not a cure. One of the ways to manage the disorder is through the skills learned in biofeedback, but this may be insufficient for patients who require a multimodality approach that incorporates biofeedback and other procedures to maintain maximum relief from symptoms. These additional treatments may include vasodilating medication, special diets, and a modified lifestyle to avoid triggers. This modified lifestyle may include cessation of smoking since nicotine is a potent vasoconstrictor that probably contributes to the occurrence of Raynaud’s attacks.

**Respiratory Interventions**

The respiratory system is unique among physiological systems in that it is both voluntary and involuntary. This fact probably explains the predominance of breathing in meditation and healing traditions going back thousands of years. A good general reference on the relationship between

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psychological factors and breathing is *Multidisciplinary Approaches to Breathing Pattern Disorders* by Chaitow, Bradley, and Gilbert (2002).

**Respiratory Function**

The function of respiration is performed by the lungs. The lungs consist of two cone-shaped spongy organs that are contained within the pleural cavity of the thorax. At the base of the lungs lies the diaphragm, a sheet-like muscle that allows the lungs to expand and contract. When the diaphragm moves in a downward direction, the lungs expand; this is referred to as *inhalation*. When the diaphragm relaxes back to its normal position, the lungs contract; this is referred to as *exhalation*.

The right lung has three and the left lung has two lobes. An adult lung weighs approximately 1260 grams (a little under three pounds). Average adults breathe about 12–18 times per minute. An adult lung has a capacity of approximately 20 cubic inches of air with each respiration, which translates into 300 cubic feet of air every 24 hours. Each lung has tiny tubes called the *bronchi*; on entering the lung, these bronchus tubes subdivide (17 times) into smaller bronchi, which in turn subdivide into smaller bronchioles. At the end of bronchioles lie the alveoli through which air can pass in two directions. As blood circulates through the lungs, carbon dioxide moves from the blood stream into the alveoli and is then exhaled. Oxygen moves from the lungs into the blood stream where it is circulated throughout the body. Air moves to and from the lungs via the pharynx, trachea, and larynx.

The lungs contain small hairs called *cilia*. These cilia move somewhat like seaweed on the floor of the ocean. Through this wave-like motion, the cilia continually sweep debris from the lungs upward so that it can be expelled from the body. Smokers develop a smoker’s cough because tar is deposited on these cilia, which prevents normal cleansing of the lungs. The smoker must mechanically cough in order to expel contaminants that have accumulated in the lungs as a normal part of respiration. This is why smoker’s cough is particularly noticeable early in the morning: it is the only way the smoker can cleanse the lungs after a night of accumulation. The entire respiratory tract is coated with mucus as a defense against the contamination of the lungs themselves. This mucus may become thickened in conditions such as asthma and actually impede the transfer of air from the lungs into the blood stream or from the blood stream into the lungs.
There are various indexes of respiratory function, most of which are measured with a device called a spirometer. A spirometer monitors the rate of exhalation. For example, one index is called peak expiratory flow rate (PEFR). PEFR indexes the maximum amount of air that can be expelled as the patient is encouraged to exhale as strongly as possible. Forced expiratory volume measures how much air exits the lungs during each discrete second of exhalation. These respiratory indexes are used medically to evaluate respiration and can be significantly compromised by a variety of respiratory diseases.

It is also interesting to note that the nerve supply to the lungs includes both sympathetic and parasympathetic fibers from the autonomic nervous system. Thus there is a neural pathway that accounts for the effect of stress on respiratory function. Sympathetic activation produces bronchodilation (the opening of airways) while the parasympathetic branch of the ANS—through the vagus—potentiates bronchoconstriction (the closing of airways). During an attack of bronchoconstriction, as might occur in asthma, the administration of an adrenergic agonist is used to produce the bronchodilation needed to end the attack. This means that for the respiratory system, the effects of stress do not directly produce the symptom of wheezing or obstruction. Instead it is believed (but not well understood) that sympathetic activation may produce a “rebound” parasympathetic effect causing the symptoms.

**Hyperventilation**

Another pathway through which emotional or psychological factors may influence respiration is hyperventilation. Hyperventilation has a variety of different definitions, all of which reference an abnormally low supply of carbon dioxide. CO₂ can be measured noninvasively with a device known as a capnometer. The capnometer pumps a small sample of exhaled air though a gas spectrometer and gives you an estimate of arterial CO₂ that is called end tidal CO₂ (ETCO₂). Many definitions focus on rapid breathing and/or breathing excessive amounts of air with each breath. However, what is common to all definitions of hyperventilation is the deficiency in CO₂ levels.
Hyperventilation is probably produced by abnormal input through the sympathetic and parasympathetic nerves, which are known to be carriers of the stress response. The criteria for diagnosing hyperventilation vary among clinicians but it is quite common to refer to a measurement of carbon dioxide levels in the blood stream. It is well known that changes in CO₂ levels can affect a variety of emotional symptoms including anxiety, shortness of breath, dizziness, tingling in the limbs, cognitive deficits, and the like. A questionnaire called the Nijmegen Index is available to assess the frequency and severity of hyperventilation symptoms (Van Dixhoorn & Duivenvoorden 1985). On many occasions a vicious cycle develops in which there is a minimal occurrence of hyperventilation, which then triggers a slight drop in CO₂ levels; this in turn triggers an increase in anxiety, which further amplifies the rate of hyperventilation. The anxiety increases as the hyperventilation increases, and this cycle can quickly escalate into a major anxiety and/or panic attack. This is sometimes referred to as the hyperventilation syndrome (Fried, 1993).

There is a difference of opinion among various researchers on the role of hyperventilation in symptoms involving panic. Some researchers attribute a minor role to respiration in panic attacks, whereas others argue that hyperventilation is a major trigger for panic symptoms. More research is needed to resolve these differing opinions. When low ETCO₂ accompanies a high score on the Nijmegen Index, the hyperventilation syndrome is likely. Recent research using ambulatory measurement devices in panic versus other patient groups has indicated subtle abnormalities in CO₂ and other respiratory parameters in panic attack patients (e.g., Wilhelm, Trabert, & Roth, 2001a, 2001b). Frequent sighs and poor CO₂ recovery were the most notable features of these findings.

**Hypoxia and Hypocapnia**

It is important to discriminate between hypoxia and hypocapnia. Hypoxia is defined in terms of insufficient oxygen in the blood stream, whereas hypocapnia is defined in terms of insufficient levels of CO₂. Hypocapnia, therefore, is the appropriate term to describe the results of hyperventilation. Many researchers argue that both states must be included in order to completely explain the connection between inappropriate breathing and symptoms of panic.
In reviewing the literature, it is apparent that different research groups have reached entirely different conclusions regarding the roles of hypoxia and hypocapnia in panic disorder. However, it is well established that hypocapnia has widespread and profound effects on the body, both intracellularly (affecting the myocardium and sympathetic activity) and extracellularly (affecting oxygen demand and dissociation). Thus it can affect cardiac rhythms, muscle excitability, sympathetic activity, neuronal excitability, and (most important) the ability of hemoglobin—the primary carrier of oxygen to the target organs in the body—to bind with oxygen. With hypocapnia (overbreathing), the oxygen becomes overbound to the hemoglobin molecule and thus not available to the target organ (e.g., brain, heart, muscle). This is why hyperventilation can produce profound symptoms.

In addition to hyperventilation, some patients will present complaints of chest pain. Although chest pain is commonly associated with elevated levels of muscle activity and/or cardiac dysfunction, it also can be related to dysfunctional respiration. Respiratory muscles, unlike cardiac muscles, are skeletal and striated and possess no inherent rhythm. Some researchers believe that abnormal activity in the diaphragm muscle can produce symptoms of pain. This abnormal activity may involve spasms of the diaphragm or another yet-to-be-discovered function.

Relaxation and Biofeedback Treatment of Hyperventilation

With one relatively minor exception, most researchers agree that relaxation therapy is helpful in dealing with hyperventilation. The one exception is a condition referred to as relaxation-induced anxiety in which a very small number of patients experience increased anxiety on relaxing. Because the number of people experiencing relaxation-induced anxiety is quite low, the assumption can be made at the beginning of therapy that the patient will benefit from relaxation, but the therapist should watch for any signs of shift toward anxiety or panic. Some researchers have found that the cause of relaxation-induced anxiety may be a failure to appropriately reduce the respiration rate while seated in a reclined position. This leads to hyperventilation to the extent that the rate of respiration exceeds the rate appropriate for a person in a reclined position.
Biofeedback has been used to address a variety of hyperventilation problems. Biofeedback always involves the use of instruments and there are several methods available to monitor respiratory function. One of these measures nasal airflow with a thermistor. Recognizing that inhaled air will be cooler than exhaled air, it is possible by using a computer to discriminate between the temperatures of the air during the breathing cycle and generate a display for the patient showing inhalation and exhalation information. As the display rises and falls on the computer screen, the patient receives feedback regarding the nature of inhalation and exhalation. This nasal airflow technique is not in wide use by most biofeedback clinicians.

A more common approach is to utilize a strain gauge in which two straps are wrapped around the upper body, one around the upper chest and one around the area of the diaphragm. Appropriate care must be taken to respect patient modesty, especially when the training involves a male therapist and a female patient. As the patient inhales and exhales, the strain gauge records these motions and translates them into a rising and falling display on a computer screen. One line on the screen displays movement in the upper chest and the other line displays movement in the area of the diaphragm. A frequent goal in training is to teach the patient to gradually increase the movement in the area of the diaphragm while decreasing movement in the upper chest. This is equivalent to teaching diaphragmatic breathing, which has been thought to be a more effective breathing technique. For aesthetic reasons many of us are reluctant to breathe with our abdominal muscles, but this is a better and more efficient way to exchange air.

The advantage of this measurement technique over nasal airflow is that the strain gauge provides information about movement in the upper chest as well as in the diaphragm area. Thus strain gauges can be used to teach diaphragmatic breathing (whereas this is not possible with the nasal airflow techniques) and can provide accurate information regarding respiration rate. Unfortunately strain gauge measurements only provide relative amplitude data during a particular training session. Numbers cannot be compared from session to session because it is not possible to reattach the sensors in precisely the same way.
It is also possible to use SEMG feedback to monitor activity in the breathing muscles. This can be single- or multiple-channel feedback and allows a separation of feedback, as is the case with strain gauge feedback. It is up to the individual therapist to determine exactly which muscles are to be monitored and it is very important to properly attach the sensors and select the proper bandpass in order to avoid crosstalk from unwanted muscle activity.

Some researchers use what is referred to as an *inspirometer*, which consists of a piston moving within a cylinder as the individual breathes into the cylinder. The position of the piston depicts the amount of air inhaled. This device is relatively inexpensive and can be reused, but it does not provide information regarding different areas of the body and is less informative than strain gauge or muscle tension feedback.

Thus it would appear that each method for measuring respiration has its advantages and disadvantages, and it is important that the researcher/clinician select the technique that best meets the client’s needs. There are as many as a half-dozen other techniques for measuring respiration but these are relatively rare and will not be covered here.

The goal of using biofeedback for respiration is to initially correct abnormal breathing patterns, such as using chest muscles rather than muscles in the area of the diaphragm. This is followed by teaching the patient to slow the rate of respiration since this is taken as a sign of relaxation. Slow and even breathing is also a goal of many schools of meditation and there is a long history of focusing on breathing as a relaxation technique. Indeed, biofeedback may simply be a means by which the process of learning to breathe properly is achieved in a shorter period of time due to the provision of feedback with each breathing cycle. Over time and with practice, chronic hyperventilation may actually resolve and ETCO₂ may return to normal or near normal.

**Heart Rate and Pulmonary Biofeedback**

Another application related to respiration is called *resonance frequency heart rate* biofeedback. Feeding back a combination of heart rate and respiration has been shown to help the client create a “resonant” state in the ANS that may be useful in treating irritable bowel syndrome, panic disorder, asthma, or other disorders mediated by ANS dysfunction. Specialized
equipment is needed to obtain an accurate picture of the various slow wave components in the heart rate, but teaching people slow (usually about six breaths per minute), effortless breathing should help accomplish many of the goals of the biofeedback. Teach the breathing with the goal of creating increased awareness of internal rhythms; eventually clients should be able to “sense” when they are in the proper rhythm and stay there for 20–30 minutes. Jan van Dixhoorn, a Dutch cardiologist, has worked for years with cardiac rehabilitation patients and has developed a very effective system based on the premise that clients discover the optimal breathing pattern for themselves (see Van Dixhoorn, Duivenvoorden, & Poole, 1990).

When biofeedback is used for chronic obstructive pulmonary disease (COPD) such as emphysema, different studies have produced differing levels of benefit. One of the rationales for using biofeedback with COPD is that relaxation will facilitate a degree of improvement. Although some studies support this contention, other studies do not. Therefore the safest conclusion at this time is that using biofeedback to produce increased levels of awareness for COPD may be helpful for an individual patient, but does not necessarily produce benefit for all patients trained.

A more specific form of feedback for COPD (or asthma) involves respiration feedback. Results are more promising in this area since the training is specific to improved respiratory function. For example, by using strain gauges it is possible to train the asthmatic to increase PEFR levels. These increases in PEFR levels translate into more efficient breathing and can help mitigate the effects of the pulmonary disease. There is some research also showing benefit from training the patient to relax facial muscles utilizing SEMG feedback. In some patients facial muscle feedback seems to have a beneficial effect on the symptoms of asthma. Once again it is prudent to recognize that these beneficial results vary among patients.

There is also a rationale for utilizing electrodermal feedback that has been shown to be sensitive to increased levels of anxiety. As breathing becomes more labored and impaired, anxiety levels may increase, which can further impair breathing. This can produce an asthmatic attack that necessitates the use of some type of strong medication. It is possible to prevent these attacks in some patients by training the patient to move to lower levels of anxiety as the initial signs of difficult respiration first appear.
Without this training it is typical for the patient to panic at the first sign of difficult breathing, and this in turn makes the breathing even more difficult.

Paul Lehrer and his colleagues (1997) have shown that daily practice using heart rate variability and respiration biofeedback can improve lung function, reduce asthma symptoms, and allow reduction in rescue medication use. This type of biofeedback involves training the patient to breathe at a prescribed frequency that promotes resonance in the autonomic nervous system, while maintaining a “mindful” state mentally.

Perhaps one way to determine what is best for the individual patient is to conduct a psychophysiological stress profile. Through the results of the psychophysiological stress profile, it may be possible to determine exactly how the patient suffering from COPD tends to respond to stress sources. If the patient is primarily a muscle responder, then facial muscle feedback may be the modality of choice. If, however, the patient is primarily an electrodermal responder, then electrodermal feedback may make more sense for that particular patient. In the absence of specific information regarding a particular patient, it is wise to consider a comprehensive approach that includes a variety of feedback modalities in order to maximize the beneficial effects.
References


Continuing Education Appendix

A. Continuing Education Instructions

B. Learning Objectives
A. Continuing Education Instructions

Some readers have purchased this module to obtain continuing education credit for psychologists or for BCIA recertification. If you are interested in receiving continuing education credit, please read the following information and instructions.

Continuing Education for Psychologists

AAPB is approved by the American Psychological Association to offer continuing education for psychologists. AAPB maintains responsibility for the program. Credit is provided for the term of the program; i.e., one hour of independent study provides one hour of credit. This module offers four hours of credit.

Biofeedback Certification Institute of America

This independent study module is accredited by BCIA to provide four hours of Category A continuing education. If you would like more information about continuing education credit through BCIA, please see its website at www.bcia.org.

Other Continuing Education Credit

Please check with your certification/licensure provider to determine specific requirements for CE credit for nursing, physician, physical and/or occupational therapy, social workers and all other CE requirements.
Module 5: Autonomic Nervous System
Part 2 – Specific Biofeedback Applications

B. Learning Objectives

This module is designed to help you:
1. Describe the etiology of migraine headache and develop a treatment plan including assessment, session design, and follow-up.
2. Discuss the normal regulation of blood pressure and the abnormalities of normal regulation seen in hypertension.
3. Summarize the application of SEMG, thermal, and electrodermal biofeedback to lowering blood pressure in hypertension.
4. Explain the symptoms of hyperventilation in terms of the basic anatomy and physiology of the respiratory system.
5. Describe the vascular changes associated with a Raynaud’s attack and design a course of biofeedback treatment.