INTRODUCTION AND OVERVIEW

In recent years an increasing number of practitioners have been using thermal biofeedback and other related techniques as a therapy for Raynaud's disease and other vasoconstrictive syndromes. Subjects are taught to self-regulate increases in hand temperature and this ability is then employed by patients to avert or abort the vasospastic attacks associated with these disorders.

Raynaud's disease is a functional disorder of the cardiovascular system involving interruption of local blood flow, usually in the fingers, due to paroxysmal vasospasm. The vasospastic attacks are usually the result of exposure to cold, although they can also be precipitated by emotional upsets. When attacks occur, the fingers are cold and numb; there is a loss of manual dexterity and frequently there is considerable pain, usually requiring an interruption of ongoing activities. While this condition is not often severely incapacitating, Lewis (1949) estimated that in its mildest forms it affects approximately 20% of young individuals. Current medical treatment can be characterized, at best, as providing only partial relief, and it frequently involves undesirable side effects.

Contributing Committee Members for this report are as follows: John R. Erbeck, Clarence H. Hartman, Norman L. Stephenson, and Richard Surwit.

Address all correspondence to Dr. Edward Taub, Institute for Behavioral Research, 2429 Linden Lane, Silver Spring, Maryland 20910.
Related vasoconstrictive disorders that have also been thought amenable to biofeedback therapy include acrocyanosis, posttraumatic Raynaud’s phenomenon, scleroderma and other collagen-related vasospastic disease processes, causalgia, posttraumatic reflex sympathetic dystrophy, pernio syndrome, and trench foot. Thermal biofeedback therapy has been applied to some of these conditions, although it has not been used as frequently as with Raynaud’s disease.

Experimentation in the area and clinical case reports have almost uniformly indicated a positive result in the use of thermal biofeedback and related techniques as a therapeutic approach to Raynaud’s disease and some other vasoconstrictive disorders. Research has not yet indicated the extent to which these positive outcomes are due to a specific increase of blood flow in the hands or to some more general factor, such as relaxation and reduced sympathetic nervous system outflow, which could be induced by means other than temperature self-regulation. However, whatever the answer to this important question, it is clear that thermal biofeedback has a markedly beneficial effect in the treatment of Raynaud’s disease and some other peripheral vasoconstrictive syndromes. Given the relative lack of effectiveness of chemotherapy and surgical intervention, thermal biofeedback must therefore be considered a useful addition to the therapies available for treatment of these conditions.

BACKGROUND AND RESEARCH

Target Pathological Conditions

Raynaud’s Disease. Raynaud’s disease is a peripheral vascular disease in which paroxysms of cutaneous vasospasm occur, usually in the fingers. The vasospastic attacks can be precipitated by emotional upsets (Graham, 1955; Graham, Stern, & Winokur, 1958; Mittelmann & Wolff, 1939) but are usually precipitated by exposure to cold. Attacks may last for minutes to hours, and in their complete form have three stages indicated by characteristic color changes: (1) pallor or a white color of the skin caused by reduction or complete cessation of cutaneous blood flow, (2) cyanosis or a blue color of the skin, (3) hyperemia or a red color of the skin due to a rebound state of excessive vasodilatation. The attacks result in cold, numb hands and are frequently painful. In the progressive or advanced stages of the disease, the phenomenon may be so severe and frequent that it is disabling. Exposure to a slightly cool environment and almost any emotional stress may precipitate episodes; consequently, even the warmer weather of the
summer season may afford little relief. The trophic changes in the involved parts may interfere with normal use of the extremity.

Raynaud's disease is approximately five times more common in women than in men (Fairbairn, Juergens, & Spittell, 1972), and Lewis (1949) has estimated that in its mildest forms it affects approximately 20% of young individuals. While the syndrome may be associated with primary vascular disease or be secondary to various other conditions, in its idiopathic form no type of therapy has been very satisfactory. Reserpine given by either oral or intraarterial routes may bring symptomatic relief, and vasodilator drugs taken immediately before exposure to cold may provide some protection from attacks. Other agents such as phenobarbital and aspirin are also used. For many patients, use of the vasodilators and catecholamine depleting agents used in chemotherapy results in side effects that are more annoying than the disease itself. Consequently, treatment often emphasizes or is confined solely to avoidance of low environmental temperatures, emotion-producing situations, and chemical agents that may aggravate attacks—in effect, no treatment at all. Radical management involving sympathectomy is sometimes employed in severe cases. However, even this procedure ensures against later recurrences in only approximately 50% of the cases. Besides, sympathectomy is a major surgical procedure that has permanent adverse consequences and some risk.

The physiology of Raynaud's disease is not completely understood. Even the nature of the circulatory derangement is still the subject of some disagreement. Whether the sympathetic nervous system is overreactive, as originally suggested by Raynaud (1888), or a local fault exists in the digital vessels, as later asserted by Lewis (1929), is still debated. Many workers have suggested (e.g., Lewis, 1938; Mendlowitz, 1954; Peacock, 1958) that there are two types of Raynaud's disease, one with and one without digital vascular obstruction. Exaggerated vasomotor tone is found largely in the latter group and it is only these cases that may be considered idiopathic.

The increased vasomotor tone is apparently due to heightened sympathetic neural discharge (Mendlowitz & Naftchi, 1959); there may also be an increased sensitivity of the peripheral vasculature to norepinephrine (Peacock, 1959; Jamieson, Lubdroom, & Wilson, 1971), although there is still disagreement on both points (Birnstingl, 1971; Burch & Phillips, 1963; Kontos & Wasserman, 1969). Halpern, Kuhn, Shaftel, Samuels, Shaftel, Selman, and Birch (1960) have suggested that the basic mechanism in Raynaud's disease involves a disruption of equilibrium between catecholamines and the "buffering" action of serotonin, aggravated by a monoamine oxidase deficiency; however, this view has not met with complete agreement.

Although it is known that during a vasospastic attack blood is initially absent from the capillaries, it is not known whether the spasm occurs at the
arterial, arteriolar, or capillary level. Recent work by Coffman and Cohen (1971) indicates that in Raynaud's disease patients there is a decrease in arteriovenous shunt flow, as well as in capillary flow, below that of normal individuals during whole-body cooling. In the 1930s, several investigators (Lewis & Pickering, 1931; Fatherree & Allen, 1938) suggested that the innervation of digital vessels is different in Raynaud's patients from that of normals. Other investigators believe, in contrast, that the disease involves a general sympathetic hyperactivity. Whether either or both of these contentions is correct, the evidence is strong that there are neurogenic factors involved in Raynaud's disease. The importance of psychogenic factors is also unequivocal (Craig, 1944; Grace & Graham, 1952; Mittelmann & Wolff, 1939; Neumann, Lhamon, & Cohn, 1944; Peacock, 1958). A common element in both neurogenic and psychogenic factors might be a hyperactivity or hyperreactivity of the sympathetic nervous system.

**Other Vasospastic Disorders.** A number of other pathological conditions involve a hyperreactivity of the peripheral vasculature (Fairbairn et al., 1972) and are therefore potentially amenable to therapeutic approach through the use of temperature self-regulation. *Acrocyanosis,* for example, is related to Raynaud's disease and is characterized by painless and persistent coldness and cyanosis of the distal parts of the extremities. It can be distinguished from Raynaud's disease in that the associated vasospasm tends to be persistent rather than intermittent and is as easily precipitated by other stressors as by cold. Its incidence is at least as great as that of Raynaud's disease (Fairbairn et al., 1972), and it tends to be more incapacitating. *Posttraumatic Raynaud's phenomenon* sometimes occurs among persons who use pneumatic and vibratory tools; among those whose occupation requires frequent squeezing of tools and parts of machine tools, such as mechanics, lathe operators, and farmers; and among typists and pianists. The vasospastic attacks sometimes become so severe that individuals must give up their trade or profession. *Scleroderma and other collagen-related vasospastic disease processes* usually begin with sclerotic changes in the skin and subcutaneous tissues of the fingers, which may then progressively spread to involve the backs of the hands, forearms, face, and chest. They are usually associated with vasomotor hypersensitivity to cold (Raynaud's phenomenon). While many authorities are of the opinion that this is not the primary causative factor, vasomotor disturbances are certainly a prominent symptom.

*Causalgia* is a neurovascular syndrome occurring after injury to or operation on the extremities. The chief symptom is an agonizing, burning pain. The clinical picture also includes alternating episodes of vasoconstriction and vasodilatation. In addition, there tends to be persistent coldness, cyanosis, and occasionally Raynaud's phenomenon, edema, atrophy of the
skin, hyperesthesia, and paresthesia. Posttraumatic reflex sympathetic dystrophy is probably essentially the same entity as causalgia, except that the latter is initiated by peripheral nerve injury while in the former there is no demonstrable injury to a major peripheral nerve. It is believed by many authorities that some portion of the pain in both conditions is due to the vasomotor disturbances.

Pernio syndrome is a nonfreezing type of cold injury produced by exposure to low environmental temperatures. It includes acute and chronic chilblains, trench foot, and immersion foot. All these conditions appear to involve the operation of a similar mechanism, which has as its outstanding feature a change in the normal reaction of the peripheral blood vessels to cold. Chronic pernio involves the appearance of recurring and chronic cutaneous lesions in susceptible individuals, following repeated exposure to the cold. The lesions produce a burning, itching sensation, and frequently become ulcerative. Trench foot and immersion foot frequently occur in wars fought under conditions of extreme cold. In severe cases, there is a residual hypersensitivity to even cool environments that constitutes a long-term disabling injury for many veterans.

Rationale for the Use of Thermal Biofeedback Therapy for Raynaud’s Disease and Other Vasoconstrictive Syndromes

During vasospastic attacks there is an interruption of circulation in the capillary beds of the digits and a resulting marked decrease in skin temperature. Lewis (1938) found that vasospasm in the hand could be relieved and circulation restored by local warming. Since a local warming effect can be self-induced as a result of learning to self-regulate hand temperature, it seems reasonable that training to carry out this task could provide a useful therapeutic approach to Raynaud’s disease.

The symptoms in Raynaud’s disease are episodic. Thus, in order for self-regulation to be effective in these cases, it need be carried out only for short periods of time—just long enough to terminate or prevent an attack. This is a simpler requirement to place on a self-regulation task than the control of some chronic condition, which would involve continuous self-regulation over long periods of time.

Many investigators have shown that normal individuals can learn to self-regulate increases in hand temperature. Among the first workers in the field were Lisiria (1965), Green, Sargent, and co-workers (Green, Green, & Walters, 1970; Sargent, Green, & Walters, 1972, 1973), Roberts and co-workers (Roberts, Kewman, & Macdonald, 1973; Roberts, Schuler, Bacon, Zimmermann, & Patterson, 1975), and Taub and co-workers (Taub, 1977;
Taub and Emurian, 1971, 1976). Experimental results with normal subjects provide considerable basis for optimism with respect to the value of the technique as a therapy for Raynaud's disease. For example, normal subjects have displayed the following phenomena relevant to the use of thermal biofeedback for Raynaud's disease (summarized in Taub, 1977): (1) the ability to maintain self-regulated increases in hand temperature for periods up to 42 minutes (the longest period tested); (2) retention of the ability for periods up to 1 year, (3) performance of the task during such concurrent activities as participation in a bridge game, (4) performance of the task without feedback after overtraining. All of these abilities would be required if thermal biofeedback is to have practical significance, and the data indicate that people do have them. In addition, (5) performance when subjected to cold stress was found to be equal or superior to that at normal room temperature, even after vasoconstriction had occurred following a 25-minute exposure to cold. Raynaud's disease, of course, involves a hyperreactivity to cold.

Research

In the early 1970s, temperature or photoplethysmographic feedback was employed to treat five published individual cases of Raynaud's disease or allied conditions in four different laboratories, two cases in one laboratory (Shapiro & Schwartz, 1972) and one case in three others (Jacobson, Hackett, Surman, & Silverberg, 1973; Peper, 1972; Surwit, 1973). Each of the studies was undertaken on a pilot basis and therefore the results can provide only suggestive evidence. However, the report in each case was positive, and taken as a whole, the work was certainly promising.

In further research, Taub, Howell, Slattery, and Prandoni (described in Taub, 1977) reported on work with seven patients with confirmed diagnoses of idiopathic primary Raynaud's disease. They found that Raynaud's disease patients could learn temperature self-regulation as easily as a matched group of unaffected subjects. At times, these patients began a session with their hand temperature either at, or only 1-2° F above, ambient room temperature, indicating that there was little or no blood flow in the skin. With training, they were frequently able to increase hand temperature into the normal range.

Four of the Raynaud's disease patients, who began training in the winter, were frequently exposed to cold environments and other situations conducive to vasospastic attacks. These patients kept daily records of their condition. After approximately 20 sessions, each reported a greatly decreased incidence of such attacks and an ability to reduce the severity of
incipient attacks, or even to prevent them, by employing, without feedback, the technique learned in this laboratory. (A fifth patient, who was also trained in winter, took elaborate measures to avoid exposure to cold, which could precipitate vasospastic attacks, while the remaining two subjects did not begin their training until warm weather had begun.)

Four of the subjects were subjected to a whole-body cold challenge by circulating cold water through tubing that lined the inside surface of a "cold suit" they were required to wear. In control sessions, normal subjects typically exhibited a drop of 5–8° F in finger temperature during a 10-minute cold challenge (suit inlet temperature at 60° F) when asked to simply sit quietly and not temperature self-regulate. Raynaud's patients exhibited even larger drops in temperature. Temperature self-regulation conferred considerable protection against hand temperature decreases in the four patients tested. One of the patients was able to prevent hand temperature decrease entirely, while another was able to increase hand temperature during the cold challenge.

Stroebel (unpublished results) has conducted a study involving approximately 80 patients with primary idiopathic Raynaud's disease, the largest series to date. The patients were subjected to cold-stress tests and were asked to keep diaries of the frequency and severity of vasospastic attacks before, during, and after training. During training the subjects learned to self-regulate increases in hand temperature and were instructed in a technique for inducing relaxation. The ability to carry out these procedures helped many patients avert vasospastic attacks during cold-stress tests in the laboratory after training. In addition, a large proportion of the patients reported major decreases in the number and severity of vasospastic incidents. Many of the patients who had frequent vasospastic attacks prior to training were virtually free of these incidents during an 18-month follow-up period. Patients with scleroderma and other types of secondary Raynaud's phenomenon exhibited improvements as good as that of patients with primary Raynaud's disease.

Two other studies that are as yet unpublished provide confirmatory results. Freedman, Lynn, Ianni, and Hale (1978) studied 6 patients with primary idiopathic Raynaud's disease and 4 patients with Raynaud's phenomenon secondary to some other condition. At the time of this writing, the patients had been followed for 3–12 months after thermal biofeedback instruction. All 10 patients were found to employ strategies developed during biofeedback training to dramatically reduce vasoconstrictive attacks. Two of the patients were found to be completely symptom-free, while 5 others were nearly so. May and Weber (1977) worked with 4 primary Raynaud's disease patients and 4 patients with secondary Raynaud's phenomenon. A daily record of the number of vasospastic attacks of 5 of these subjects occurring during the first 2 weeks of training was compared with
those occurring during the last 2 weeks of training. A significant reduction of symptoms occurred in all five cases.

In the past, autogenic training has been employed for the relief of symptoms in various vasoconstrictive syndromes, including Raynaud's disease. Autogenic training is a technique in which individuals learn to increase hand temperature through the use of self-suggestive phrases that are repeated silently (e.g., "My hands are heavy and warm"). Though no feedback is presented to the subject, the technique is similar to thermal biofeedback in that individuals do learn to self-regulate their hand temperature. The results with this technique have been generally positive, but there has been considerable variability between patients (summarized in Luthe and Shultz, 1969, p. 79). Recently Surwit, Pilon, and Fenton (1977) used an autogenic traininglike procedure with patients suffering from primary idiopathic Raynaud's disease. All trained subjects demonstrated a significant ability to maintain finger skin temperature in the presence of a cold-stress challenge and also reported significant reductions in both frequency and intensity of vasospastic attacks. A second group was also run in this study in which biofeedback training was added to the autogenic traininglike procedure. The authors report that the treatment outcome for this group was no better than for the group receiving autogenic training alone. However, there was no independent evidence in this experiment that the subjects had in fact developed the ability to increase hand temperature on the basis of the feedback. Since evidence of this nature was not obtained, it is not possible to interpret this aspect of the results.

It is of interest to note that three case studies have been reported in which self-regulation of increases in hand temperature has been of value in reducing the pain of angina pectoris and the medication required to control it (Hartman, 1978). However, this report must be considered only suggestive until more evidence is obtained.

In summary, thermal biofeedback and associated techniques have been shown to be very promising as a therapeutic approach to Raynaud's disease and allied vasoconstrictive disorders. Treatments currently available for this disease are generally unsatisfactory, because of either minimal effect, unpleasant side effects, or radical nature. In contrast, temperature self-regulation has two major advantages. First, long-term use has not been observed to have any secondary consequences, and certainly no undesirable side effects, in either normal subjects or Raynaud's disease patients. Second, since the vasospastic attacks are episodic, the patient need employ the technique only when attacks threaten to occur; no continuous regimen is necessary. Consequently, thermal biofeedback can appropriately be considered a useful alternative or adjunctive therapy for Raynaud's disease and related vasoconstrictive syndromes.
SUGGESTIONS FOR FUTURE RESEARCH

1. This report summarizes research involving approximately 130 patients with pathological conditions of the peripheral vascular system. It would be of value to have additional research in order to increase the number of subjects for whom there are relevant data.

2. More long-term follow-up than is currently available is needed.

3. The most obvious means of obtaining information about Raynaud’s disease symptoms is the recording in a diary of incidence and severity of attacks. Diary data, however, are subjective, and therefore subject to various undesirable errors resulting from, for example, faulty memory or a desire on the part of the subject either to appear in a favorable light or to conform to the expectations of the experimenter. The research of Stroebel, of Surwit, and of Taub, and their co-workers, has included cold-stress tests where the capability of subjects to prevent vasospastic attacks through the use of temperature self-regulation is observed directly under laboratory conditions. It would be important for future work to obtain objective verification of the efficacy of thermal biofeedback through this or similar means.

However, a demonstration in the laboratory that temperature self-regulation can be used to avert vasospastic attacks does not necessarily mean that this can be accomplished in a nonlaboratory setting. Information relating to this issue can be obtained by means of diary keeping. Thus optimal experimental procedure would involve the use of both techniques to quantify treatment outcome.

4. Although the evidence that thermal biofeedback eliminates symptoms in patients with Raynaud’s disease is reasonably good, the mechanism by which this effect is achieved is unknown. Raynaud’s disease is known to be a labile entity that is importantly influenced by psychogenic factors. It is recognized that the experimenter–patient interaction and other potential placebo effects can significantly affect the outcome. Future research should be designed to control for these effects in as rigorous a fashion as possible. One simple technique for approaching this problem is to ascertain whether the ability to self-regulate hand temperature, as determined by laboratory tests, is correlated with symptom reduction. A second, and more conclusive, technique for determining whether a specific or general effect is involved would be to include one or more placebo-attention control groups in the experimental protocols. The treatment in these groups would be designed to promote relaxation in a subject and confidence that he could control his symptoms; however, the subject would receive no training in a technique that could be expected to specifically alter blood flow to the hand.
REFERENCES


Lewis, T. Experiments relating to the peripheral mechanism involved in spasmodic arrest of the circulation in the fingers, a variety of Raynaud’s disease. *Heart*, 1929, 15, 7-101.

Lewis, T. The pathological changes in the arteries supplying the fingers in warm-handed people and in cases of so-called Raynaud’s disease. *Clinical Science*, 1938, 3, 287-319.


(Received July 25, 1978)