Physical Agents for Physical Therapists

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This book deals with the clinical applications of physical agents in the management of patients' problems with pain, strength or coordination. The text concentrates on recent advances, resulting from clinical and laboratory research, which permit more logical use of nerve and muscle stimulating currents, cold, heat, diathermy, ultraviolet light and ultrasonic energy. Aside from its obvious worth in the classroom, practicing physical therapists will find the book to be a valuable reference for confirming clinical experience and for establishing guidelines for more effective treatment.

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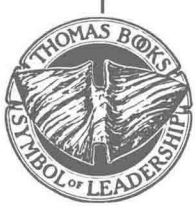

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PHYSICAL AGENTS FOR PHYSICAL THERAPISTS

PREFACE

THIS book deals with the clinical application of physical agents. It is intended as a text for the baccalaureate-level physical therapy student. It is assumed that the student has a reasonable background in undergraduate-level biology, chemistry, pathology, and physics and that the material contained herein will be supplemented by appropriate lectures and demonstrations.

The reason for writing the book is that nowhere in recent years has any physical therapist made an attempt to bring together and apply the advances in physiology and electronics which have evolved from clinical or laboratory research. These advances permit more logical use of cold, heat, nerve and muscle stimulating currents, diathermy, and ultraviolet and ultrasonic energy in helping the patient to be relieved of pain and to regain independence in activities of daily living.

A wealth of material has been reported in various journals within the last several decades. The literature cited in the book is in no way complete or exhaustive. It has been chosen as representative of developing new concepts of disease and treatment, or as confirming older concepts that have withstood the test of time.

Sections of the various chapters dealing with treatment technics do not spell out details that are more effectively learned within practice classes. Technics are given in detail when the described procedure can lead to improved patient care.

Chapters on electronic instrumentation design, hazards, and safety requirements reflect the major changes in federal regulations which are presently or soon to be in effect. Conceptual knowledge of modern apparatus design and of advances in physiological rationale for choice of physical agent to alleviate a problem should minimize the occasion for the physical therapist to become involved in malpractice suits because of use of defective equipment or because of misapplication of good equipment and/or technics.

J.E.G.
T.C.K.

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PHYSICAL AGENTS FOR PHYSICAL THERAPISTS

Chapter 1

PAIN

PERHAPS the simplest definition of pain is that "it is a hurt that we feel."[1] The standard medical dictionary definition of pain is that it is a more or less localized sensation of discomfort, distress, or agony, resulting from the stimulation of specialized nerve endings.[2]

If a physical therapist wishes to be useful in the relief of pain, he needs to know as much as possible about the biophysical effects of physical agents. If a patient is limited in his activities of daily living because of pain, or because he perceives his pain as a major factor during his waking hours, it is unlikely that he will make much progress in regaining control over range of motion, use of assistive devices, or gaining in strength until such time as the pain has been relieved, or at least reduced to a tolerable level.

Regular administration of the appropriate physical agent can be highly effective in relieving pain in properly selected patients. Physical agents may also be used in conjunction with medication and/or postsurgically. Frequently, the patient will derive greater benefit from therapeutic exercise if the exercise is preceeded by a carefully selected physical agent.

CAUSES OF PAIN

Causes of pain are myriad. Quite often there will be abnormal activity of muscle, peripheral circulation, and/or the nervous system, either as a cause or the result of the patient's perception of pain.

Spasm may be defined as an abnormal and continuing hyperactivity of any type of contractile tissue, such as skeletal, smooth, or cardiac muscle. Although far less common than spasm in either

skeletal or smooth muscle, the cardiac muscle spasm, which is a dominant feature in true angina pectoris, is one of the most severe forms of pain. If this abnormal cardiac muscle contraction can be relaxed, the accompanying pain will be diminished.

When pain is present, there is often concomitant local circulatory impairment. One known cause of such impairment is skeletal muscle spasm, which can significantly limit venous return. Such flow deficit can give rise to additional pain because the deficit leads to inadequate local removal of metabolic waste products.

Vasospasm is the abnormal continuing hyperactivity of smooth muscles in the walls of blood vessels. It is of particular importance in arterial vessels with a diameter of a few millimeters. The ratio of muscle to other tissues is especially high in arterial vessels of small diameter. Vasospasm can be triggered by peripheral or central irritation of sympathetic nerves, or by edema formation. In all cases, vasospasm leads to the reduction or cessation of arterial flow. Significant reduction gives rise to pain, and in extreme cases to irreversible damage, in the tissues supplied by the malfunctioning vessels. Organic occlusive peripheral vascular diseases can cause severe chronic impairment of circulation and give rise to steadily increasing pain over weeks and months.

Furthermore, diminished circulation, either arterial or venous, can give rise to skeletal or cardiac muscle spasm because of inadequate local nutrition or ineffective removal of waste products from the local area served by the malfunctioning vessels.

The nervous system can also play a major role in pain production. The sensory portions of the nervous system must be functioning and have functioning connections to the spinal cord and on up to higher (cortical) levels before the patient can be aware of noxious stimuli. The rare infant with a congenital lesion such that he cannot perceive pain seldom survives his infancy. He dies as a consequence of injury or disease of which no one is aware. Skeletal muscle hyperactivity, vasospasm, and nervous system malfunction can all occur without the patient feeling pain if pain pathways to the cortex are interrupted by trauma or disease. The abnormal activity without pain can be just as much or more of a handicap to activities of daily living. Abnormal muscular hyper-

activity without pain is commonly seen in the chronic phase of the traumatic paraplegic or quadriplegic patient. In such patients, the primary lesion is usually in the spinal cord; more rarely, it occurs within the cranial vault. Usually, the peripheral nervous system remains largely intact, but because of edema, anatomical cord severance, or other lesion in the cord, pain pathways are interrupted. Because of neuroanatomical relationships within the spinal cord, pain and other sensation is more apt to be lost than is motor nerve control over skeletal muscle or sympathetic nerve control over smooth muscle in blood vessel walls.

CLASSIFICATION OF PAIN

The clinical physician usually classifies pain into three major categories. One type is often called a fast-onset, pricking type of pain. A second category is frequently described as of slow onset, leading to a burning sensation. The third type is commonly pictured as a deep, dull ache. This ache is frequently remote from the causal site and usually is due to the dysfunction of some portion of the viscera. Mountcastle[3] has published in monograph form a very useful review of pain and pain pathways from the sensory physiologist's point of view. It now appears certan that the fast, pricking pain is most often transmitted from the periphery into the central nervous system by delta axons, which are 4 to 6 microns in diameter, whereas the smaller, unmyelinated C fibers normally transmit pain stimuli leading to a burning sensation. It is this latter type of pain with which the physical therapist is most often concerned, pain of slow onset, great persistence, and accompanied by powerful responses, both physiological and psychological. The third general category of pain awareness, visceral or aching pain, is more apt to be a life-threatening indicator and is not often amenable to physical therapy procedures.

Abnormal functioning of local areas of skeletal muscle, circulation, and the nervous system are very much interrelated. Each abnormality can be a cause of pain; each can be the result of noxious stimuli. Each can be a part of the vicious cycle of pain —spasm—more pain—more spasm. Under some circumstances, appropriate administration of the most suitable physical agent

can be the best means to interrupt that vicious cycle and thereby achieve cumulative, lasting pain relief.

The psychological aspects of pain are somewhat more elusive and controversial than the physiological components. The psychological aspects can be conveniently considered on the basis of the importance that the subject attaches to his pain. Sternbach[1] expressed these variables quite well when he wrote,

> It is not pain which is mental or physical, functional or organic, psychic or somatic, but our ways of thinking about pain and the systems of terms we use to describe pain which may be so dichotomized. All pain can be described in both languages, the psychological and the physiological. Pain itself is not one or the other. But because pain can be described in both mental or physical terms, pain is a truly psychosomatic concept. From this point of view, all pain is real; and all pain is also psychosomatic since both mental and physical descriptions are possible.

The key to how intense or how minimal is an individual's response to pain is probably previous conditioning plus present distraction. Either can serve to increase or decrease anxiety and thus to increase or decrease pain.

THEORIES OF PAIN AWARENESS

There have been many theories published about possible mechanisms and pathways for pain perception and pain relief. Possibly the one that comes the nearest to affording a reasonable working hypothesis in accounting for all presently known aspects of pain and pain relief is that presented by Melzack and Wall[4] in 1965. Their gate control theory proposed a neural mechanism in the spinal cord which can increase or decrease the flow of pain impulses from the periphery to the brain. When other sensory input is minimal, pain awareness is great. When other sensory input is equal to or greater than pain input, awareness of the latter is diminished. This gate control theory can well account for the concept of counter irritation, which is very useful in attempting to explain why judicious intermittent use of heat, cold, ultrasound, and nerve stimulating currents can relieve pain. By 1974, Melzack[5] was able to state and defend the hypothesis that the

neospinothalamic system selects and modulates sensory input in such a fashion as to permit the discriminative dimension of pain. He further stated that activation of reticular and limbic structures through the paramedial ascending system underlies the motivational drive and unpleasant affect that trigger the organism into action. Lastly, Melzack presented evidence that neocortical or higher central nervous system processes (suggestion, anxiety, and attention) exert control over activity in both discriminative and motivational systems (Fig. 1-1).

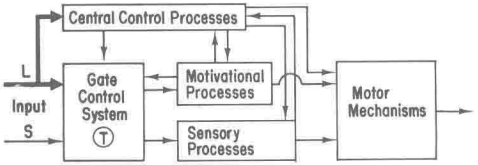

Figure 1-1. Schema for demonstrating the gate control theory of pain. L — large, fast-transmitting axons. S — small, slow-transmitting axons. T — transmission cephalad from spinal cord after sorting. Motivational processes include central intensity monitoring. Sensory processes include spatiotemporal analysis. From International Symposium on Pain (Advances in Neurology, Volume 4), p. 526. Editor, John J. Bonica. © 1974 by Raven Press, New York.

As physical therapists, our primary concern with pain and pain relief is to add sufficient energy to the patient to provide sufficient increase in nonpainful sensory input to override or counterbalance the painful sensory input, or to transiently diminish the ability of axons to conduct. The problem frequently is how to drive a physiologically acceptable quantity of energy to the needed depth without damaging intervening tissues.

KNOWN EFFECTS OF CLINICALLY APPLICABLE HEAT AND COLD

When cold is applied to a patient for the purpose of relieving pain, the objective is to lower tissue temperature within physiological limits. Fortunately, altering the temperature of cutaneous nerves can do much to reduce hyperactivity of underlying contractile tissue. Miglietta[6] showed that application of cold to the

skin can bring about significant reduction of deep muscle spasm within one minute. However, Wolf and Basmajian[7] have demonstrated that even when intense cold is applied to a small area of skin, it takes an application time of at least five minutes before there will be a 1°C reduction in muscle temperature at a depth of 5 cm. In earlier experiments more nearly approaching standard clinical application of cold to a local area, Bierman[8] demonstrated that cold must be applied for thirty minutes before there is a similar drop in deep muscle temperature. Abramson et al.[9] have published evidence that there is always vascoconstriction with a drop in muscle temperature.

Reviews of clinical cryotherapy[10] and of the physiological effects of heat and cold[11] indicate that much of the measurable effect of conductive and/or radiant heating and of conductive cooling is an indirect response due to the stimulation of cutaneous nerves. Effects of these agents on skeletal muscle and subcutaneous circulation are due more to such indirect effects than to primary energy absorption or release by subcutaneous tissues.

Voluminous literature has developed over the years demonstrating that when cutaneous thermal receptors are stimulated by heat, there is an increase in nerve conduction velocity, an increase in cutaneous circulation brought about by local cutaneous release of histamine (a potent vasodilator) or histaminelike substances, and a further increase in cutaneous circulation as warmed blood reaches the vasomotor regulators within the central nervous system. There is also reduction of skeletal muscle tension as motor nerve impulses diminish in number in response to cutaneous thermal stimulation.

When cold is applied to the skin, with techniques suitable for relieving pain, there is reduction in cutaneous nerve conduction velocity, vasodilation followed by vasoconstriction, and reduction in skeletal muscle tension. It is interesting to note that both increase and decrease in sensory nerve conduction velocity, as well as both cutaneous vasodilation and vasoconstriction, can lead to relaxation of underlying muscle. Any standard text on electromyographic techniques[12] will give ample data indicating that both