The Use of Splints in the Treatment of Joint Stiffness: Biologic Rationale and an Algorithm for Making Clinical Decisions

The purposes of this article are (1) to discuss the rationale for using splints to increase range of motion (ROM) and (2) to describe an algorithm that can guide therapists' clinical decisions when splints are used to treat patients who have limited ROM. The primary rationale for using splints is to apply relatively long periods of tensile stress to shortened connective tissues to induce tissue lengthening through biologic remodeling. The process of remodeling is contrasted with more temporary mechanical phenomena that occur in biologic tissues. The proposed algorithm guides the use of splints based on measurements of pain and ROM. We describe three variables of splint use that may be adjusted: frequency, duration, and intensity. The relative importance of each of these variables is discussed. The algorithm is not joint or injury specific and requires continual modification of splint use based on a patient's response to treatment. Deciding which patients are appropriate for end-range splinting and deciding when to discontinue splint use are also discussed. [McClure PW, Blackburn LG, Dusold C. The use of splints in the treatment of joint stiffness: biologic rationale and an algorithm for making clinical decisions. Phys Ther. 1994;74:1101–1107.]

Philip W McClure
Lisa G Blackburn
Carol Dusold

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Splints are used for many purposes in rehabilitation.[1] Among these purposes is the application of splints to hold joints at or near their end-range position in order to increase range of motion (ROM). These splints may be static,[2] dynamic,[3] or static-progressive.[4] Static splints are those that hold the joint at some constant position. Dynamic splints are those that incorporate some elastic component such as springs or rubber bands that exert a force on the joint but allow for some change of joint position. Static-progressive splints are simply static splints that include a component that allows adjustments in joint position in relatively small increments. Many authors have described the use of splints for increasing ROM at various joints, including those of the hand,[1] elbow,[3] shoulder,[2] and knee.[3] There is a growing body of literature supporting the use of splints and other end-range devices (such as continuous passive motion machines and traction applied via a pulley system) to provide low-load prolonged stress to shortened periarticular tissues that limit ROM.[2–7] Despite this literature, there appears to be no generalized model or algorithm to guide the clinician when making decisions relating to splint usage by patients with limited ROM. Rather, various isolated joint- or injury-specific protocols are described that are not readily applicable to other conditions.[5,7] Although

PW McClure, PT, OCS, is Assistant Professor, Department of Orthopedic Surgery and Rehabilitation, MS 502, Hahnemann University, Broad and Vine Sts, Philadelphia, PA 19102 (USA). Address all correspondence to Mr McClure.

LG Blackburn, PT, is Staff Physical Therapist, National Rehabilitation Hospital, 102 Irving St NE, Washington, DC, 20010.

C Dusold, PT, is Staff Physical Therapist, Associated Physical Therapists of Milwaukee, Milwaukee, WI 53233.

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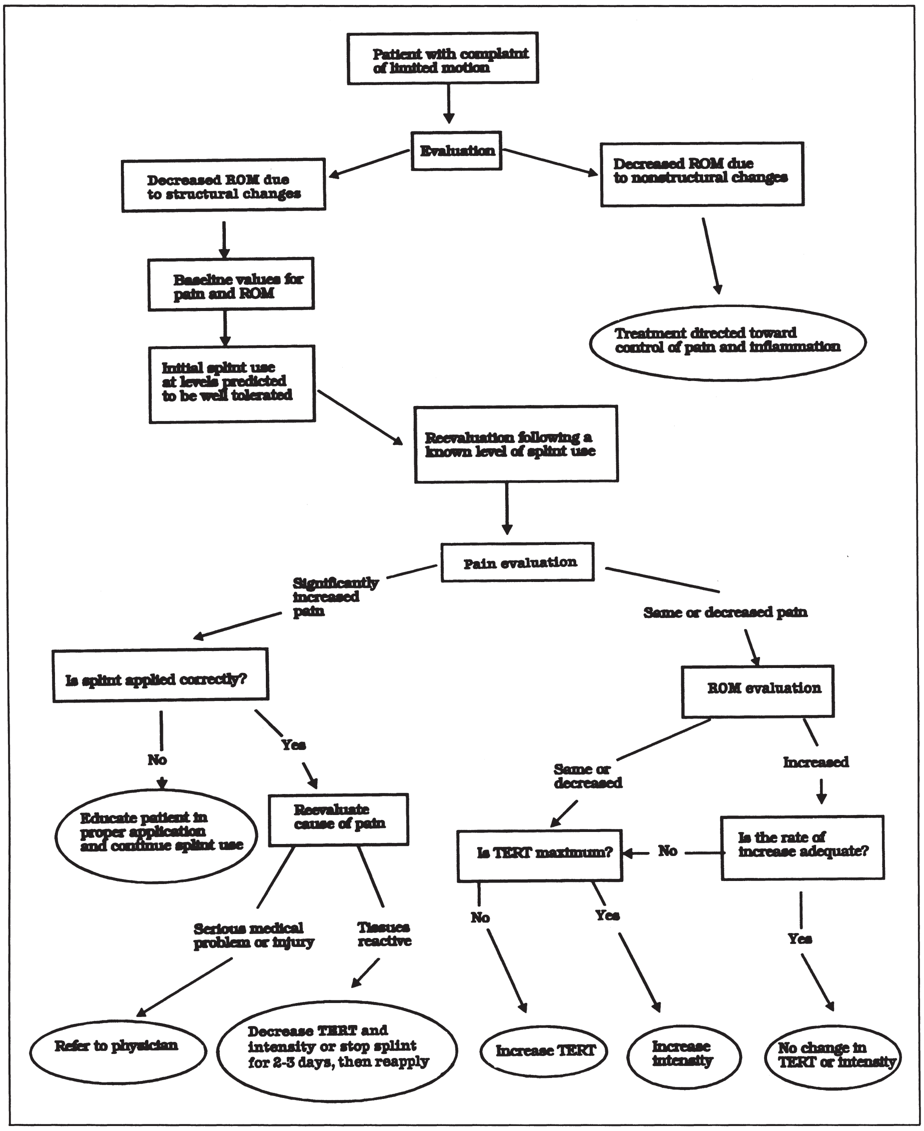

Figure. *An algorithm to guide the use of splints in the treatment of joint stiffness. Intensity reflects the amount of force applied. The intensity of a dynamic splint is increased by adding tension; for a static splint, the joint angle would be increased. TERT=total end-range time (frequency×duration of splint wearing time daily); ROM=range of motion.*

numerous splints have been described and are available commercially, no unified model exists to guide the proper clinical application of these devices. Therefore, we have developed an algorithm (Figure) to guide clinical decisions relating to the use of splints for increasing ROM.

The general purpose of this article is to discuss the use of splints for the treatment of limited ROM. Specifically, we will (1) review the rationale for using splints to increase ROM and (2) describe an algorithm that can guide therapists' clinical decisions when splints are used to treat patients who have limited ROM. The proposed algorithm is not joint or injury specific, but is more universal and calls for problem solving and continual modification of splint use based on a patient's response to treatment.

Rationale for Using Splints: Biologic Remodeling

The primary rationale supporting the use of splints for increasing ROM is the biological principle that periarticular connective tissues (PCTs) will remodel over time in response to the type and amount of physical stress they receive.[8–10] Remodeling refers to a physical rearrangement of the connective tissue extracellular matrix (fibers, cross-links and ground substance). There is strong evidence that the process of remodeling is mediated by the fibroblasts[8,9] in response to physical forces. Remodeling is a biologic phenomenon that occurs *over long periods of time* rather than a mechanically induced change that occurs within minutes.

There is normally a balance between degradation and synthesis of collagen and other extracellular components of connective tissue such as glycosaminoglycans (GAGs).[8,10,11] This balance allows maintenance of the necessary structural and mechanical characteristics of the tissue required for normal use. When the normal pattern of use is altered significantly (primarily by increased or decreased tensile loading), morphological, biomechanical, and biochemical changes occur in the PCTs.[8,10–12] Frost[9] has termed this general phenomenon "structural adaptation to mechanical usage," and the reader is referred to his work for a more complete explanation of his theoretical model.

Periarticular connective tissues are designed to withstand tensile stress. If these tissues are deprived of tensile stress by being immobilized in a shortened position, predictable changes will occur. Collagenous tissues deprived of tensile stress exhibit decreased amounts of collagen, GAGs, and water, as well as an increase in the amount of reducible cross-links.[8,11] Immobilized tissues also become shorter, weaker, and less stiff.[8,10–15] The decrease in tissue stiffness should be distinguished from an increase in joint stiffness[11,14] (increased resistance to motion) found with immobilization. Increased joint stiffness occurs despite actual weakening of PCTs because these tissues assume a shorter length and therefore exert greater resistance to a given amount of joint motion. These detrimental changes associated with immobilization are worse if the tissues have been traumatized.

Woo and associates[11] proposed a mechanism for the development of limited joint ROM or contracture. They theorized that with immobilization, loss of GAGs and water allows new intermolecular and intramolecular cross-links to be formed within the PCT, which limits the extensibility of the tissue. Other investigators[16] suggest that connective tissues may shorten by the process of contraction. The contractile protein actin has been identified in fibroblasts and implicated in the process of ligament contraction.[16] Others[17] believe the myofibroblast, a cell that resembles a smooth muscle cell, may be the primary mediator of connective tissue contraction. Another mechanism by which joint motion may be limited is adhesion formation. An adhesion is simply scar tissue that has formed between tissues that normally move relative to one another.[18] Adhesions have been identified as an important source of limited motion in many common clinical problems.[19,20] Abnormal cross-links, PCT contraction, and adhesion formation may all play a role in formation of a joint contracture.

Muscle also exhibits morphologic changes when immobilized in a shortened position. Muscle accommodates to a shortened position by losing sarcomeres, and therefore the muscle itself becomes shorter.[21] The connective tissue within a muscle also undergoes shortening with immobilization in a shortened position.[22] Length-associated changes in muscle have been reviewed extensively,[23,24] and a complete discussion of these concepts is beyond the scope of this article. Regardless of the actual mechanism, the result of joint immobilization is a decrease in the functional length of the PCTs and muscles that have been held in a shortened position. This decrease in length causes limited ROM.

Many authors[15,25,26] have found that subjecting periarticular tissues to controlled tensile loading will result in length changes as well. Frost[9] theorizes that there exists a "minimal effective strain" for a given tissue and that when this level of strain is exceeded, biologically mediated changes occur. In general, collagenous tissues respond to increased tensile loading by increasing synthesis of collagen and other extracellular components.[8–10,15,25] The collagen is oriented parallel to the lines of stress, and tensile strength is increased.[15,25,26] Muscles subjected to prolonged positioning in a lengthened position will become lengthened by adding sarcomeres.[21]

Studies using both animal and human models provide evidence that controlled tensile stress applied cyclically or statically leads to remodeling of periarticular tissue. Arem and Madden[26] used a rat model to study the effects of prolonged periods of tensile stress (6 hours per day) applied for 4 weeks to healing scar tissue. When the stress was applied to 3-week-old scars, the scars were markedly elongated as compared with controls. When the same amount of tensile stress was applied to 14-week-old scars, however, essentially no scar elongation was observed. These data suggested that following trauma, scar tissue formation is influenced by mechanical stress but the scar becomes less amenable to change over time.

In two clinical studies,[5,7] the effects of low-load prolonged stress (via prolonged positioning in a traction apparatus) were directly compared with the effects of more intense, brief forms of stress for gaining ROM. Light et al[5] studied elderly subjects with long-standing knee flexion contractures. They compared a low-load prolonged stress applied with a modified Buck's traction apparatus with passive stretching applied manually. Rizk et al[7] studied subjects with adhesive capsulitis of the shoulder. They compared low-load prolonged stress applied to the shoulder by a pulley system with more traditional active and passive exercises. The data from these studies indicated ROM improved more for those subjects receiving the low-load prolonged stress.

To summarize, the primary basis for using splints to increase ROM is that by holding the joint at or near its end-range over time, therapeutic tensile stress is applied to the restricting PCTs and muscles. This tensile stress induces remodeling of these tissues to a new, longer length, which allows increased ROM.

Remodeling Versus Transient Physical Changes

Remodeling can be contrasted with other processes that lead to a more transient increase in ROM. Because periarticular tissues are viscoelastic, they demonstrate creep, stress-relaxation, and preconditioning.[27] When noninjurious loads (ie, within the elastic limit) are applied to tissues, either statically or cyclically, tissues readily demonstrate a transient lengthening (creep or preconditioning) or a decrease in the amount of force required to hold a given length (stress-relaxation). These phenomena occur within a short period of time, typically a few minutes, and are thought to be highly dependent on the viscous component of the tissue.[27] Some authors[28] have described these phenomena as producing a permanent, plastic change in tissue length. Although this may be the case, we believe that unless a tissue is deformed beyond its elastic limit and therefore injured, length increases due to viscoelastic phenomena are only temporary.[9,29,30]

This controversy may exist because mechanical studies demonstrating creep, stress-relaxation, and preconditioning are usually performed with tissues isolated from the body and length changes are only monitored for a short period following release of the load.[31] Mechanical changes, therefore, tend to remain after forces are removed, and these changes are characterized as being permanent or plastic. When applying forces to tissues in living humans, however, the tissue remains in a closed, fluid-filled homeostatic biologic system. Here, the contents and organization of the extracellular matrix remain under the influence of cellular control. Therefore, a purely mechanically induced change may be less likely to remain once external forces are removed. Brand has noted that "any elongation of tissue accomplished by stretch will shorten again once the force is relaxed."[30(p1097)] Frank et al[29] have similarly stated that following short-term stretching procedures, "ligaments return to prestretch lengths."

Changes in ROM due to viscoelastic phenomena can be easily demonstrated with procedures that are typically applied for brief periods, such as joint mobilization and other passive stretching techniques. We believe, however, that these stretching procedures share a significant drawback in that they can only be applied for a short time. Therefore, tissues that have been temporarily preconditioned or "stretched out" are permitted to return to prestretch lengths. The major advantage of using splints to increase ROM is that forces are applied for longer periods of time and as a result are more likely to induce a change in tissue length by remodeling. We consider the total amount of time the joint is held at or near an end-range position, or "total end-range time" (TERT),[32] as the most critical variable in determining the amount of tensile stress applied to a joint with limited ROM. The concept of TERT will be discussed in detail within the context of the algorithm.

Determining Appropriate Patients

An important clinical issue is determining which patients are appropriate candidates for end-range splinting. In general, patients with joints that have structural changes in the periarticular tissues producing limited motion are appropriate for splinting. These structural changes include shortening of capsule, ligament, or muscle as well as adhesion formation. These changes generally result from a combination of inflammation and immobilization.[8,10,11] Findings that suggest limited ROM due to these structural changes include the following:

1. A history of trauma followed by immobilization.[8,10]

2. A history of restricted motion greater than 3 weeks.[8,10]

3. Loss of passive ROM in a capsular pattern.[33] (The loss of motion will be in a specific, predicted pattern for that specific joint.)

4. A capsular end-feel.[33] (A *capsular end-feel* is defined as a firm halt to passive movement with only a slight degree of give with further force.)

We believe that if either of the first two findings is present, the likelihood of structural changes is high. Cyriax[33] suggests that loss of motion in a capsular pattern indicates pathology involving the entire synovium and capsule as opposed to other processes not directly involving these tissues that may limit motion. We believe, however, that it is possible for problems such as protective muscle guarding to limit motion in a capsular pattern. The ability to distinguish a capsular end-feel from other types of end-feel may have questionable reliability. We believe, therefore, that the presence of a capsular pattern and capsular end-feel may be helpful in confirming the presence of periarticular structural changes, but that their presence alone is not conclusive evidence of such changes.

In patients with central nervous system deficits, it may be helpful to distinguish between a muscle/tendon unit that has shortened due to loss of sarcomeres and a muscle that is actively contracting due to excessive reflex activity. Structural shortening due to loss of sarcomeres results from immobilization in a shortened position,[21] and this situation can be reversed by holding the muscle in a lengthened position over time.[21] Patients with structural shortening of muscle should demonstrate decreased ROM even if active muscle contraction is inhibited. Patients with inappropriate muscle activity only (ie, without structural shortening due to sarcomere loss) should demonstrate normal ROM if muscle contractions are inhibited. Splinting is clearly appropriate for patients with structural shortening or those in whom structural shortening is anticipated. A discussion of the merits of splinting for the treatment of disorders involving reflex activity is beyond the scope of this article. Tardieu and Tardieu[34] provide a more detailed discussion of this issue.

In contrast, patients may also have limited ROM not attributable to structural changes in the periarticular tissue. Most often there is active inflammation, and the patients report significant pain and exhibit protective muscle guarding. There may also be other signs of inflammation such as swelling, redness, and local temperature elevation around the joint. The treatment of actively inflamed joints can involve modalities directed at decreasing inflammation and pain rather than applying end-range stress. It is also possible to simultaneously have both structural changes and an acute inflammatory response. Patients with acute inflammmation are not appropriate for end-range splinting because prolonged end-range stress would risk increasing the inflammatory response.

Explanation of the Algorithm

The decision to use a splint and the initial variables of splint use (frequency, duration, and intensity) should be based on the patient's history and examination. In general, we try to use the splint initially in a manner that we are confident will be well tolerated. That is, we prefer to "understress" the joint with the initial trial of splinting and subsequently increase the stress in a controlled manner. This approach not only avoids unnecessary pain but also gives the patient a positive first experience with use of the splint, which improves compliance. We typically will see the patient within 2 days following initiation of splinting and can therefore increase use of the splint if necessary.

The algorithm (Figure) describes the process of decision making during reevaluation of the patient at each visit. The frequency of reevaluation is dependent on the nature of the condition. Initially, we typically monitor splint use two to three times per week. With joints that have well-established, long-standing stiffness, the frequency of visits may be decreased to as little as once every 2 or 3 weeks. The response of the joint and periarticular tissues to initial splint use is judged based on two variables: pain and ROM. We believe these variables are adequate clinical indicators of tissue reactivity or degree of inflammation. That is, measurements of pain and ROM reflect the degree of inflammation in the articular and periarticular structures. For example, a highly reactive (inflamed) joint would lead to increased pain and decreased ROM. A nonreactive joint would not exhibit increased pain and would not lose motion in response to end-range stress via a splint. Other indicators of inflammation such as swelling and increased temperature could also be used. The basic premise of the algorithm is that measurements of pain and ROM may be utilized to determine the optimal frequency, duration, and intensity of splint application.

The Principle of Total End-Range Time and Adjustments in Splint Usage

The basic aim of splinting is to apply tensile stress to tissues that are restricting motion. We have found it helpful to think of the total amount of stress being applied as the "dose" in much the same way "dose" is used in regard to medication. In treating structural joint stiffness, we believe clinicians should adjust the dose of tensile stress until a therapeutic result (increased ROM) is achieved. An insufficient dose of stress will have no therapeutic effect, whereas an excessive dose will produce complications such as pain and inflammation. Three factors should be considered when calculating the "dose" or total amount of stress delivered to a tissue: intensity, frequency, and duration.

Flowers and Michlovitz[32] have coined the term "total end-range time," which is calculated by multiplying the frequency and duration of time spent at end-range daily. For example, a patient using an end-range splinting device in four sessions per day for 30 minutes each session has a TERT of 120 minutes. Intensity, which is the amount of force applied, is usually limited by the patient's pain tolerance. For static splints, intensity may be adjusted by changing the splint angle. For dynamic splints, intensity may be adjusted by changing the tension in the elastic component of the device, typically a spring or rubber band.

Because remodeling is a biologic process that occurs over long periods of time, we believe that frequency and duration of the applied stress are the key factors to maximize and that intensity is less important. Increasing intensity is secondary because the use of high forces may injure tissue and result in an inflammatory response and subsequent fibrosis.[35] The amount of force necessary to cause tissue injury is probably dependent on the rate of application, the amount of time the force is applied, and the temperature and initial mechanical state of the tissue.[36] Studies on isolated tissue suggest that strains beyond 2% cause permanent damage to connective tissues. It is impossible, however, to know the degree of strain placed on a given tissue during a clinical procedure.

We initially set the intensity such that the patient can wear the splint without increased pain for at least 20 minutes. Although initial settings of frequency and duration may vary, we typically start with a TERT of 1 hour per day. This TERT may be achieved by using four 15-minute sessions or two 30-minute sessions.

As shown in the algorithm, we consider the TERT to be the key factor to be modified when using splints to increase ROM. If the patient fails to demonstrate a satisfactory increase in ROM, we believe the first goal should be to increase the TERT. Maximum TERT will be different for each patient and is often dictated by circumstances, such as a job or other responsibilities, which may prevent a patient from increasing TERT. For example, a patient using a splint to increase ROM in a dominant hand may be obligated to a job that requires frequent use of that hand. The TERT may therefore be limited to what can be accomplished in the evening and during sleep. In this example, the intensity should be increased once maximum TERT is achieved. Intensity may be increased by increasing tension in dynamic splints or by increasing joint angle position in static splints.

If a patient shows a satisfactory increase in ROM with splint use, no relative change in use is necessary. In patients using static splints, increases in the joint angle may be needed as ROM gains occur in order to maintain the same relative intensity. Occasionally, a patient may demonstrate increased ROM but at a rate less than expected or preferred. For example, a patient may gain 5 degrees of motion over 2 weeks, but the therapist may decide, based on experience, that a greater increase should have been achieved. In this example, splint use (TERT or intensity) should be increased according to the algorithm.

Pain Evaluation

Pain may be measured by several methods. We prefer to use the visual analogue scale (VAS). A VAS consists of a line, typically 10 cm, anchored at each end by phrases such as "no sensation of pain" and "the worst pain imaginable." The patient's pain is quantified by measuring the distance between one end of the scale and the patient's own mark representing his or her level of pain. There is evidence that VAS measurements of pain can be reliable and valid.[37,38] We are unaware of studies that show how much of a change in pain represents a "significant" change. Based on our clinical experience, we consider a change greater than 2 cm to be meaningful.

If it is determined that pain increases, we contend the therapist should reevaluate the patient to determine the source of this pain. One source of increased pain could be improper splint application. Improper application of the splint would require further patient education regarding the proper wearing of the splint.

In the case of correct splint application coupled with increased pain, further evaluation is required. Most likely, the joint tissues are simply reactive to the stress applied by the splint. This reactivity would result in decreasing the splint intensity or the wearing time, or both, or complete elimination of the splint for 2 or 3 days to allow the condition to subside. The patient should then be reevaluated according to the algorithm to determine which changes in splint use (frequency, duration, intensity) should occur. We prefer to decrease intensity first because that most typically decreases pain. If this change does not decrease the pain, wearing time is decreased as necessary.

Less commonly, evaluation of a patient with increased pain may reveal the presence of a significant medical problem or injury. For example, a patient may develop an infected wound site, or may have experienced an overt trauma such as a fall and a new injury may be suspected. In these cases, the patient should be referred back to the physician.

Range of Motion Evaluation

We generally choose one or two specific motions that are most limited or most important for function to monitor ROM. We believe passive ROM is a more appropriate measure than active ROM because the goal of end-range splinting is to increase ROM by lengthening tissues that restrict motion passively. Active ROM may be limited due to impaired motor function and not directly affected by splinting. The amount of change in ROM necessary to decide that a true change has actually occurred (rather than simply measurement error) is a function of measurement reliability. A change of more than 5 degrees in goniometric ROM measurements is often considered a reflection of an actual change in ROM rather than simply measurement error.[39–41] This number may be slightly higher for some joint motions where goniometric reliability is less and slightly lower where reliability is high. In general, measurements are more reliable when repeated by a single tester than when different testers are used.[40] Therefore, clinicians should be aware of their own measurement reliability in order to accurately discern whether a change in ROM has truly occurred.

We advocate that joint tissues should be preconditioned prior to measuring ROM because the degree of preconditioning may affect ROM. Preconditioning is the phenomenon in which increases in tissue deformation occur when a given load is applied cyclically. A tissue is said to be preconditioned when tissue deformation reaches a steady state and continued cyclic loading produces no additional deformation.[27] Because the therapist does not know whether a patient's periarticular tissues are preconditioned when the patient first arrives at the clinic, preconditioning these tissues before measuring ROM is an attempt to standardize the conditions for measurement. We believe ROM measurements are more reliable and meaningful, and therefore more comparable, when taken with the periarticular tissues preconditioned or "warmed up" rather than "cold."[32] Preconditioning procedures will be different for each joint, but in general may consist of various forms of active ROM exercise, passive ROM exercise, and joint mobilization, which may be combined with deep-heating modalities. In our experience, the preconditioned state is achieved after 10 to 15 minutes of some type of exercise emphasizing the end-range position.

Deciding to Discontinue Splinting

There are two primary reasons to discontinue the use of end-range splints. The first is that the ROM goals have been achieved and no further increase in ROM is necessary. A special case may exist where the ROM goals have been achieved but the therapist believes continued splinting may be necessary to prevent a loss of ROM. An example would be a patient who has had a surgical release of a Dupuytren's contracture and splinting is used prophylactically. The second reason would be when the splint has been used with maximal TERT and intensity for a prolonged period of time without an increase in ROM. The length of time necessary to determine that no further gains are possible is essentially unknown and is probably dependent on the specific problem. Our experience is that some joints with well-established, long-standing stiffness may take as long as 2 months of continual splint use to demonstrate changes in ROM.

Summary and Conclusions

We have described the rationale for applying low-load prolonged stress via splints for the treatment of joint stiffness. We also have proposed an algorithm to guide the clinician when using splints for this purpose. The strengths of this algorithm are that it may be used with virtually any end-range splinting device and that it is not limited to a specific joint or diagnosis.

This algorithm is not meant to provide a specific protocol to be strictly followed; rather it serves as a guideline for decision making based on continual reevaluation by the clinician. The algorithm, therefore, allows for individual modifications, which may be tailored to a specific patient problem. This process also calls for active participation by the patient to determine appropriate variables for splint use, which may increase compliance and be therapeutic in itself. Successful use of this process has been documented.[2,6] We believe this process may also be useful as a guideline in future research studies relating to the efficacy of splinting for increasing ROM.

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