ELEVENTH NATHALIE BARR LECTURE

The Source of Our Strength

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This lecture is dedicated to Dr. William E. Burkhalter, 1928–1992, my mentor and my friend, in appreciation of his life, an example of excellence in ethics, clinical practice, and teaching.

I am honored to have the privilege of addressing you, my colleagues, and some of my dearest friends in editorial style, and to have my family, and my staff here with me to share this most special moment of my professional life. I thank this society for allowing me the opportunity to say what is on my mind.

Life is made supportable mainly by two things; love and work. (Edward Robb Lewis)

My life has been richly blessed with both. I am keenly aware of the fact that without the love of my family, especially my husband, I could have produced nothing beyond income. Creative energy is most easily tapped by a heart that feels loved.

My work has been my passion. The joy and satisfaction of caring for a patient from the early stages of wounding to a point of physical recovery and emotional equilibrium have provided me with perspective and a sense of purpose. Most of us in this room have a great appreciation for the strength and sensitivity of the human hand, and a respect for the depth of the human spirit and its ability to overcome adversity. We gain strength from the knowledge that our work and our science make a difference in our patients' lives and thus the world at large.

My message today is inspired by the part of me that is really a part of us all: the love of our science and our work, and by the many special people who have touched our professional souls *And to whomsoever much is given, of him shall much be required; and to whom they commit much, of him will they ask more.*[1] We are all enriched by:

- Those who came before us and paved our way.
- Those who inspire us with their strong sense of what is right and who teach us not to compromise.
- Those who expect more of us than we think possible.
- Those who question us and thus make us grow.
- Those who find merit in our work.
- Those who bring perspective to life and make us laugh.

New Age Science

I don't know
I don't care
It doesn't matter anyway

I promised my friends that I would talk about science today. I toyed with the idea of throwing this slide up, inviting you all to the bar for a drink, and sitting down. No doubt the talk most of you would have preferred. That idea probably would have most effectively captured our collective mood of despair and feelings of loss of control over our current professional situation. Our feelings run deep as we are now asked by American business to lower our scientific and ethical standards.

If you've come to this meeting without feeling depressed, I have to think that you are either on Prozac or having some kind of out-of-body experience.

Let's define the problem and look at a few solutions.

While there is no question that some reorganization is desperately needed in American healthcare, it seems that, for the most part, the healthcare people have been left out of the decision-making loop, or at the very least are feeling like this health care revolution is beyond their control. It also appears that both business and medicine alike have forgotten that there is one major flaw in a system in which non-medical persons are making the decisions regarding the amount and quality of health care that can be delivered to the American people. And that is that there is only one group of profes-

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sionals that can deliver that care. When we get to the point that the CEO, MBA, or high-school-graduate insurance clerk can replace a heart valve, remove a brain tumor, save a child with bacterial meningitis, care for a burn victim, discover a cure for AIDS, or treat a complex hand case, then we can allow ourselves to feel out of control and maybe then it will be okay for us to roll over. But for now, it's only the persons who have trained from 5 to 16 years who can provide the care for that same CEO or MBA when he gets into trouble with his health.

Argue for your limitations and sure enough they're yours.[2]

Theoretically, the *new order* of American healthcare affectionately known to us as *managed health care* will provide affordable, quality health care with good functional outcomes in a reasonable time to the American public. Contributing factors that have led to these reforms are a shrinking pool of health care dollars, alleged over-utilization of services and fraud, defensive medicine practices, pressure from business and the federal government to control costs, inaccessible health care for some populations (40 million Americans are uninsured), and the use of treatments that are not supported by research or outcome studies.[3]

The root of many of these problems can be traced to medicine.

Inspired in part by business fed up with rising medical costs, the *new order medicine* entrepreneurs have infiltrated medicine and have turned health care into a corporate battlefield increasingly governed by the promise of stock market wealth, incentives that reward minimal care, and a brand of aggressive competition that is alien to doctors and scientists.[4] Thinking that they understand us, these entrepreneurs advertise to the *uncommitted*, and try to apply the principles of the business world to the scientific world.

While business claims that managed health care is the solution to rising medical costs, we as health care providers recognize that the *new order* is not without problems that accompany its solutions. The delivery of care is now being determined by a corporate business model instead of a medical model with profit-driven incentives, excess monies spent on administration, denial or delay of critically necessary medical services, and restrictions of certain providers from certain networks.

Monies are not necessarily saved by the patient or employer but are instead redistributed to corporate managed care companies. Some insurers are negotiating discounts with health care providers but not passing those savings on to policy holders or patients responsible for making co-payments, and it is the insurer, not the patient or employer, who appreciates the savings. This practice has now generated federal class action suits against insurers and managed care organizations in 24 states.[5] Rapid member growth and lower reimbursement to providers have combined to yield huge profits to insurance companies.[4] While we are worried about getting coverage for dressings and silicone for our patients and caring for the indigent patient with no way to pay, the salaries for the CEOs of these managed health care companies and insurance companies are increasing at incredible rates. As one example, Ronald Compton, Chairman of Aetna Life and Casualty, received a 485% raise in compensation last year to $6.6 million, while Aetna stock rose 46.9% last year.[6] In March of this year, *The Wall Street Journal* reported that "nine of the biggest publically traded HMO's were sitting on $9.5 billion in cash with no place to spend it."[7]

There are ethical concerns with the *new order*. Some providers as well as adjusters are receiving bonus incentives for denial of care. Capitated care will only work for the primary care physician who is able to limit diagnostic testing and visits to the specialist. Conflict of interest issues are raised when managed care organizations and hospital corporations merge, as with the recently proposed merger of Blue Cross/Blue Shield of Ohio and Columbia HCA Healthcare Corporation.[8] And, closer to home, some therapists are concerned as they are forced to deal with profit-driven, corporate-owned facilities that may require them to bill unfairly or render inappropriate services.

The shift of all liability to the provider within most managed care contracts is frightening. Indemnification clauses and clauses that restrict the medical decision-making authority of the provider leave the provider vulnerable to legal action while gag rules interfere with the provider's responsibility to work as the patient's advocate.[9] In the March issue of *Florida Physicians Alert*, Florida doctors were warned that, while economic incentives used by some managed care organizations provide network physicians with financial reasons not to properly treat, refer, and hospitalize patients, few managed care organizations have been successfully sued for cost containment failures, and that physicians will bear most, if not all, of the fault and resultant liability in medical negligence cases.[10]

William Garrison, president of the Health Insurance Association of America, tells us that "guarding information helps to hold down costs." Dr. Melvin Kirschner of Van Nuys, California, tells us that "doctor's enforced silence puts the patient at risk."[11]

Clearly, the soul of business and the soul of medicine are separated by many miles of uncommon ground.

Perhaps the most chilling effect of the shift of values in the era of managed care is taking place in the area of medical research. Investigative or experimental treatment will not be covered by managed care organizations. In January, *Time* reported that the *new medicine, by its nature abhors complexity and innovation. Managed care companies fear that spending money on research will create a competitive disadvantage.*[4] Funding for medical schools and teaching institutions is jeopardized, putting medical research at risk. Dr. Michael Johns, Dean of Medical Faculty at the John Hopkins School of

medicine, and Dr. Herb Pardes, Dean of Medical Faculty at Columbia University, state that academic medicine is the best tool that we have for advancing health care. They warn the American public against its demise.[11]

As research is not funded, *soft science* will find its way into the literature, and unbiased study and critical thinking will fall victim to the *new order*. Outcome studies which we so desperately need to support our treatments, may become a part of the trend toward *soft science* if the results of these studies and their methods are not subject to strong peer review. I can just envision the marriage of outcome studies provided by a large rehabilitation corporation and a group of insurance adjusters. This combination could well give new meaning to the terms *scientific analysis, statistical significance,* and *ethics.* Double-blind study may come to mean *let's both look the other way.*

At first glance, it may appear that medicine has been no match for business. Some providers have succumbed. We disappoint ourselves as we observe that the focus of health care providers has shifted from science and ethics to business, reimbursement, and contract negotiations. Even those of use who want to participate in research and provide the highest quality of care are now in a position of just trying to survive and maintain enough income to support our staff and our practices.

The reality of our current practice situation is that most of us barely have the energy to get through a clinic day in which we are forced to treat a large number of patients who have often been referred late, too often by primary care physicians who have misdiagnosed and mistreated the patient. We then have to clear a treatment plan through a 20-year-old insurance clerk with no medical training or an insurance doctor who probably wouldn't be an insurance doctor if he had the clinical skills or personal desire to practice medicine. We then have to struggle with the means of bringing complex cases to recovery with a limited number of visits for patients whose insurance will often not cover critical items such as wound dressings, with no margin allowed for variables such as patient personality, patient intelligence level, or poor surgical skills. We have to deal with the growing attitude of entitlement amongst our patients—the feeling that the provider is responsible for getting them better and for absorbing the cost if insurance is not available or will not pay. We are evaluated by our outcomes, which are in part determined by a treatment course that is determined by an insurance adjuster or a managed care contract. Between the calls we get from adjusters, employers, rehabilitation nurses, and lawyers, the paperwork requirements, and the time spent reviewing managed care contracts for clauses that require us to accept all the risks in managing a case, we are lucky to find our way to lay hands on our patient.

So there probably aren't too many of you who want to hear me talk about the relationship of interleukin I and PIP joint inflammation.

But I'm going to do it anyway because I believe that our professional self-esteem and the joy of practice are unquestionably related to scientific curiosity, ethics, and good clinical results. And good clinical results are dependent on one thing—good science. And if we weaken our stand in the areas of ethics, clinical treatment, and scientific investigation to fit into the *new order* of American healthcare, then we will surely be left with no defense against American business.

If we allow our standards to become sub-standard, the next generation of therapists will assume that sub-standard is standard.

We must stand firm on these issues for our patients and for our cherished profession.

Truth is not negotiable. (John Madden, MD)

At first glance it appears that the *new order* requires less of our clinical and scientific skills, when in reality much more will be required of us. Our diagnostic skills will be tested as we receive more patients from primary care physicians and walk-in clinics. Our clinical skills will be challenged as we are asked to solve complex treatment problems in a limited amount of time. Our negotiating skills will be proportionate to our clinical knowledge and to the results of our outcome studies. Our knowledge is critical to our role as patient advocate; to obtain the proper care for our patient, we must use it to teach those who will listen and to intimidate those who won't.

Perhaps one solution to this dilemma is to improve our results and treatment times by taking a closer look at our management of healing tissues.

Could it be that some of our protocols are based on habit instead of science?

As therapists we have the unique opportunity to observe and feel the patient's tissues from day to day throughout the wound-healing process and to observe the tissues' response to a number of variables: wound and surgical trauma, infection, inflammation, response to stress, and to modalities. Our clinical observations will have a different slant than those of the surgeon or engineer and are a fertile source of research ideas. We are a critical link between basic science, medical engineering, and clinical treatment. How much more could we contribute if we spent more time learning from basic science and engineering and making a connection between that knowledge and what we observe from day to day?

Good clinical investigation is the touchstone of good surgical research now, just at it was with Lister and Halsted. The surgical investigator must be a bridgetender, channeling knowledge from biologic science to the patient's bedside and back again. (Francis Moore, MD)

I thought I would offer some thoughts that I have on making the connection between clinical observation and basic science studies in four areas: carpal tunnel, Dupuytren's contracture, distal joint inflammation with proximal injury, and the re-

paired flexor tendon which might make us rethink some of our protocols in these areas. *You teach best what you most need to learn.*[2]

CARPAL TUNNEL

We observe clinically that limiting finger motion is sometimes required to decrease the symptoms of carpal tunnel syndrome, and that in a number of cases, wrist control splinting alone does not offer relief of pain. This is especially true in our manual laborers with well-developed lumbricals[12] or with the anxious and often elderly patients who attempt to improve their symptoms by continually flexing their digits. These same patients will find relief of symptoms if the MP joints are splinted in extension. We observe clinically that wrist position is critical, and that subtle changes in position by as much as 20 degrees can alter symptoms.

What connection can we make between these clinical observations and basic science studies?

Intratunnel pressures are the lowest with the wrist position near neutral,[13] and most specifically at 2 degrees of wrist flexion and 3 degrees of ulnar deviation.[14] Intratunnel pressures are further relieved by finger positions that pull the lumbricals up out of the carpal tunnel. Several studies published in the last two years examine the dynamic relationship of the lumbrical muscles and the carpal tunnel and the resulting increase in intratunnel pressures associated with finger flexion movements which pull the lumbricals into the carpal tunnel increasing pressures by decreasing space[12,15-17]

The four lumbricals take their origin from the flexor digitorum profundi as the latter cross the palm.[18] Anatomical studies have demonstrated that the lumbrical muscles originate distal to the carpal tunnel with the fingers held in extension but that all four lumbrical muscles lay within the carpal canal when the fingers are actively flexed.[12,19,20] As a composite fist is made, the FDP tendons pull the proximal portion of the lumbricals into the carpal canal.

Lumbrical incursion has been studied in four finger positions.[19] The lumbrical muscle origins were found to be an average of 7.8 mm distal to the carpal tunnel with full finger extension, 14 mm into the tunnel with 50% finger flexion, 25.5 mm with 75% flexion, and 30 mm with 100% flexion. The lumbrical muscles were distal to the proximal aspect of the hook of the hamate *only* for the position of full digital extension and the position of 50% finger flexion.[19]

This information is important clinically because the hook of the hamate has been found to be the most constrictive portion of the carpal canal.[15,20,21] Therefore, lumbrical incursion to this level could likely have the greatest effect on median nerve compression.[16] This MRI and schematic of the carpal tunnel at the level of the hook of the hamate demonstrate the crowding of the flexor tendons at this level.[20] Note the transverse carpal ligament extending between the hook of the hamate and the tubercle of the trapezium. It has been demonstrated that the median nerve is compressed and flattened to the greatest degree at the level of the hook of the hamate.[22] So with finger flexion greater than 50%, this already crowded area becomes even more crowded as the lumbricals move in to take up more space and apply more pressure to the median nerve. It is easy to see how repetitive finger flexion exercises can increase median nerve symptoms.

These same researchers studied carpal tunnel pressures in the same four finger positions and found that a progressive and linear increase in carpal tunnel pressure was noted for each degree of finger flexion if the lumbricals were intact.[16] Pressures did not change if the lumbricals were excised in any finger position. A greater amount of change in pressure was recorded between the 75% and 100% flexed positions.[16]

Another recent study measured pressure within the carpal tunnel during nine functional positions of the hand and wrist[23] and supports the work of Cobb et al.[16] Intratunnel pressures exceed normal pressures[24-26] by >200 mm Hg upon making a strong fist in normal subjects, and in fact making a fist increased the intratunnel pressure significantly more than variations of either wrist flexion or extension in normal subjects.[23]

The clinical implication from review of these recent studies may be that wrist control splinting alone may not be sufficient to reduce pressures in the carpal canal in *some* patients, and that splinting should be designed to pull the lumbricals out of the carpal canal, and to discourage the patient from working the digits into greater than a 50% fist position with some cases.

We make the clinical observation that some therapy techniques designed for strengthening the hand or stretching connective tissues appear instead to increase median nerve symptoms.

The application of externally applied forces to the palm in cadaver hands increases carpal tunnel pressure and the magnitude of that pressure change is dependent on the location of the applied force.[27] It has been demonstrated that 1 kg of external force will increase carpal tunnel pressure by 103 mm Hg if applied over the flexor retinaculum, 37 mm Hg over the hypothenar region, and 75 mm Hg over the thenar area adjacent to the distal aspect of the carpal tunnel. The highest pressures are generated by pressure applied in the midline of the palm adjacent to the hook of the hamate (mean 136 mm Hg).[27]

Perhaps we should take a closer look at the use of hand grippers, theraputty, some work simulation tools, dyna-splints and progressive static splinting for the wrist, and the effects of cast pressures on median nerve compression to insure that our treatments are not contributing to median nerve compression.

We observe clinically that postoperative management following open carpal tunnel release is complicated by greater scar tenderness when the incision crosses the wrist than when the incision is limited to the palm and that treatment time and expense will nearly double in these cases.

In a study of predictors of return to work following carpal tunnel release, Katz et al.[28] demonstrated that persistent symptoms and scar tenderness most strongly correlated with failure to return to work. While other demographic predictors were defined in this study, the authors concluded that the work disability at 6 months after CTR is 23% and the principal predictor is clinical outcome of symptom relief and scar tenderness.[28]

Is there information in the literature to explain our clinical observation? Should the length of the incision be a variable within our outcome studies? Can we make our surgeons who use the long incisions aware of the additional cost in terms of treatment time, expense, and lost work with these patients when compared with incisions that stay within the boundaries of the palm? Perhaps there is a clue in this next study.

Cassidy et al.[29] have recently suggested that there is an anatomic basis for the increased tenderness of incisions which transverse the area from 5 mm proximal to the wrist flexion crease to 10 mm distal to this crease. In a cadaver study of ten hands nerve density was measured within the dermis, between the epidermis and superficial fascia, and between the superficial fascia and the transverse carpal ligament to assess whether a variation in nerve density exists in the region of standard carpal tunnel release. The results of this study indicate that subcutaneous nerve density peaks in the region extending from 5 mm proximal to the wrist crease to 10 mm distal to the wrist crease, averaging twice the number of nerves seen proximally or distally.[29]

Early suture removal following open carpal tunnel which results in even minor dehiscence results in increased scar tenderness and lengthened therapy. This problem is seen often in my practice with certain physicians who allow their technicians to remove sutures between 7 and 10 days before being examined by the physician. A point so simple as this, which violates the most basic principles of wound biology and incision line tensile strength, costs the patient and employer time and money and results in poorer outcomes for the therapist. Clinical observation, the basic rules of biology, and comparative outcome studies that look at this variable give us the support that we need to insist that carpal tunnel sutures stay in longer, sometimes as long as three weeks when the palmar skin is thick and calloused.

DUPUYTREN'S CONTRACTURE

Clinically, we recognize the importance of keeping dermal wounds tension free. Traumatic surgical technique, sutures that are tied too tightly, or poorly planned incisional design may lead to tissue necrosis, wound spreading, or hypertrophy.[30,31] Excessive tension at the wound site may cause necrosis by jeopardizing local blood supply.[30,32] This is basic biology.

Why then is it common practice to splint postoperative Dupuytren's cases with all digtal joints in full extension as early as postoperative day 2 or 3?

Clinically, we observe that there is a dose–response relationship between excessive stress and inflammation, and between inflammation and fibrosis. Why then do therapists, even hand therapists, push for end range of motion when the tissues are inflamed, and provide their patients with therapy toys that stress the tissues?

I have observed that, following Dupuytren's fasciectomy, patients who are splinted and exercised in such a fashion as to keep tension off the palmar wound experience fewer problems with hypertrophic scars and recurrence than do patients who are immediately splinted in full extension and exercised in a more vigorous fashion. Over the past 4 years I have altered my postoperative treatment for Dupuytren's by splinting the MP joints in 30 to 40 degrees of flexion, the PIP joints in full extension allowing full gentle flexion postoperatively, but disallowing full extension at the MP level until the end of the second week of wound healing when the fibroblast activity is beginning to slow. This technique, almost without fail, results in less inflammation, softer, better organized scars, good range of motion, and minimal recurrence. Regaining MP extension is never a problem.

It is reported in the literature that recurrence after Dupuytren's surgery is common and occurs in more than 50% of cases after fasciectomy.[33] Cellular and genetic aspects of the disease have been investigated as researchers try to explain the pathogenesis and recurrence of the disease,[34–49] but I have found no references that suggest that wound site tension or prolonged postoperative inflammation from aggressive therapy or forceful dynamic splinting may have an effect on recurrence.

Are there any clues in the basic science literature that might correlate postoperative tension, inflammation, and disease recurrence? Should we adjust our postoperative approach to address the issues of minimizing inflammation, and encouraging maximum nutrition to the tissues to facilitate a better cellular response instead of a quest to obtain full extension early on?

Consider these studies:

The effect of inflammation: Wiseman has suggested that there is a dose-dependent relationship between local inflammation, the number of macrophages, and thus the number of fibroblasts which are active in a wound.[50]

The macrophage, a critical cell mediator of inflammation, is responsible for phagocytosis and secretory products such as interleukin I that enhance fibroblast activity.[51,52]

Macrophage-mediated growth factors *may* provide the initial stimulus for the progression of Dupuytren's disease, and a correlation has been made between macrophage numbers and the presence of myofibroblasts in the palmar fascia of patients with Dupuytren's disease.[34]

Could excess mechanical stress from splinting and exercise great enough to inflame the tissues have an effect on the number of macrophages at the wound site and thus the number of working fibroblasts at the wound site?

The myofibroblast is thought to have a major role in the development of Dupuytren's disease.[36,38,49,53] Myofibroblast-rich foci have been found in the subcutis, dermis, and right up to the edge of the epidermal boundary and may explain the high recurrence rate of Dupuytren's disease after fasciectomy.[37,43] It has been suggested that this specialized fibroblast could be the agent that regulates the palmar fascia.[39,54]

These specialized fibroblast cells have been shown to have contractile properties,[35] and there is some speculation regarding myofibroblast activity and wound-edge tension.[55]

What is our role in preventing wound site tension? Can our treatments have an effect on the development of postoperative fibrosis?

The effect of local blood supply: How critical is the local blood supply, and what relationship does it have to fibrotic tissue reaction or Dupuytren's recurrence? The digital arteries in a digit that has been contracted over a period of time will shorten—when the contracture is released and the digit is brought to full extension these vessels will be under tension, and blood flow to the digit will be diminished. Do we compromise local nutrition and stimulate an adverse cellular response by splinting under tension in the early wound-healing phases? Do venous and lymphatic congestion associated with edema alter cellular activity?

Hypoxia is known to be a stimulus to fibroblasts in tissue culture.[56] Some investigators suggest that fibroblast proliferation may be induced by local hypoxia.[34,44,57] There is evidence that ischemia increases oxygen-derived free radical production,[44,57,58] which then may stimulate fibroblasts, causing proliferation of Dupuytren's disease.[44]

Several investigators have found the presence of occluded capillaries in Dupuytren's nodules and cords.[34,42] Microvascular changes may be a common pathway in the development of fibrotic lesions.[41]

Perhaps closer attention to the basic rules of wound healing will yield better results, fewer complications, and lower recurrence rates, with Dupuytren's patients. We should observe the tissue effects of nonphysiologic application of stress through excessive exercise and forceful splinting. Any therapeutic approach that increases inflammation or decreases circulation is to be condemned, as the possibility exists it will stimulate negative cellular response, which could conceivably lead to increased fibrosis or recurrence.

DISTAL JOINT INFLAMMATION WITH PROXIMAL INJURY

It is often the uninjured joints distal to the site of injury that become the biggest rehabilitation problem. We have all observed this phenomena with DIP stiffness following PIP joint injury or PIP stiffness following Colles' fracture, most commonly in postmenopausal women. Here is an example of a distal radius fracture 4 weeks out of fixation with excellent wrist motion but limited PIP joints. Are there biochemical studies that might explain distal joint inflammation with more proximal injury? Could we interrupt the development chemically or mechanically by better controlling inflammation if we were more alert to the diathesis?

There are studies to suggest that it may not be necessary to have a wound in order for fibrosis to occur at a joint; alterations of the microenvironment may produce the same effect.[58-60] Capillary congestion, as noted previously, may be a sufficient stimulus. Nonfibroblast cells may contribute to fibrous tissue formation.[59,61] Diegelman et al. note that the production of collagen by nonmesodermal cells in culture is perhaps one of the best examples of the way in which an altered environment can stimulate collagen expression.[59]

We are again reminded of the importance of seeing a patient early in the wound-healing process to control edema and to start gentle motion to minimize the effects of altered nutrition to the uninvolved joints. We should think in terms of the biochemical effects of injury and management, and not just in terms of range of motion.

ACTIVE TENSION FOLLOWING FLEXOR TENDON REPAIR

And, finally, a few thoughts about the flexor tendon injury. One of our greatest challenges in practice continues to be the problem of reestablishing functional gliding of the repaired zone I and II flexor tendon without creating gap formation or rupture at the repair site during the first 3 weeks of wound healing. The shift in postoperative management has come full circle from active motion in 1912,[62] 1917,[63] and 1923[64] to immobilization, passive motion, and, in this past decade, the shift again to controlled active motion immediately following repair. The shift to active motion, inspired by inconsistent clinical results with passive motion programs[65-67] and questions regarding true tendon excursion with passive motion[68-72] has support in a number of biochemical and clinical studies.[73]

The most convincing case for active motion has just been published by investigators who have determined in the experimental model that breaking strength is improved and cellular activity enhanced when both *motion* (passive tendon movement through the synovial sheath) and *tension* (active tension or stress) are applied to a flexor tendon repair site when compared with the use of only *motion*, only *tension*, or the use of neither.[74]

Recently developed active motion programs have been dependent on stronger repair techniques with increased suture material.[75-82] Favorable results have been reported, but these repairs have not been widely accepted because many surgeons feel that they are too bulky and technically difficult. Active motion with conventional suture (modified Kessler with epitenon)[82-85] has not been recommended by most surgeons[71,72,80,86] because the ten-

sile strength of these repairs has been considered inadequate based on previous studies of internal tension forces with digital flexion.[87,88]

A close review of the literature and careful clinical observation may raise some questions about these assumptions.

What effect does extra suture material have on a tendon's ability to glide? Consider these recently published studies.

The mechanical interactions of tendon loading and motion between the FDS and FDP and the distal edge of the A2 pulley have been studied.[89] These authors have demonstrated that there is a narrowing of the tunnel formed by the FDS through which the FDP passes if the FDS is loaded proximally, creating a situation similar to the "Chinese finger trap." With load, the FDP at this level also changes shape and narrows as it moves through Camper's chiasma.[89] *We must ask our surgeons what effect the additional bulk of extra suture will have on the ability of both the FDP and the decussation to change shape as they interact with proximal tension or loading at the level of Camper's chiasma.*

The amount of suture material and the increase in resistance to tendon gliding have been studied in cadaver tendon.[90] These investigators have demonstrated that the *work of flexion*[90-92] is increased in direct proportion to the amount of suture material used in the repair.[90] Note that the repairs designed to tolerate the forces of active motion—i.e., Silverskiolds mesh sleeve,[80] Savage 6-strand,[79] and Aoki's internal and dorsal tendon splints[93]—also markedly increase the resistance to tendon gliding and the *work of flexion. We must ask our surgeons what effect these heavier repairs have on the drag a tendon encounters as it glides proximally with active tension and how much the additional suture increases internal tendon forces at the repair site.*

These two studies[89,90] suggest that, while increased suture material may strengthen a tendon repair site, it may also affect the tendon's ability to change shape with load[89] and may increase the resistance to flexor tendon gliding,[90] which will elevate internal tendon tension at the tenorrhaphy. We should also remember that the more complex repairs require more handling by the surgeon and may also result in more tendon edema which also will increase the *work of flexion.*

While most surgeons feel that conventional repair (i.e., a modified Kessler with an epitendinal suture) will not tolerate active tension, we have all observed that it is the patients who "cheat" within their passive programs by providing some active tension to these repairs who often enjoy the best functional outcomes. We note that our colleagues from the British Isles, and indeed some of us in this country, who employ active programs with these repairs report excellent results and rupture rates that are similar to those with passive programs.[94-99]

Perhaps our surgeons should direct more attention to the definition of "active motion" and to a more precise application of force through specific joint angle and external load,[100] Perhaps they should recognize that our contribution to the problem of tendon gliding can reach beyond splint making, and protocols which define forces in terms of "light active motion" or "place and hold" exercise.

Perhaps, though, we first have to recognize how much we can contribute of we understand the basic principles of engineering that allow us to calculate internal tendon forces mathematically[100-103]; and how much we can contribute if we understand the added resistance to tendon gliding created by the effects of wound healing,[104,105] suture material,[90] and pulley.[106-108] Perhaps we need to know as much about the tensile strength of each tendon repair that we work with as our surgeons know. How can we, in fairness to our patients, apply active stress to a tendon repair site without having some working knowledge of the forces imposed on that repair and if those forces will be tolerated by that repair? Are we satisfied to accept a referral from a physician that reads *zone 2 flexor tendon repair, "place and hold" technique O.K.*? Would you dare to move that tendon without knowing exactly what kind of repair was employed, if an epitendinal suture was used, or if the repair would glide under the pulley?

Do we think in numbers as we work with our tendon cases?

My good friend Dr. John Madden reminds us of the words of the famous physicist Lord Kelvin: "I often say that when you can measure what you are speaking about and express it in numbers you know something about it; but when you cannot measure it, when you cannot express it in numbers, your knowledge is of a meagre and unsatisfactory kind; it may be the beginning of knowledge, but you have scarcely in your thoughts advanced to the stage of science."

The future of flexor tendon management will depend on the combined efforts of the surgeon, the engineer, and the therapist. In this next decade we may find that is not extra suture material that is the answer to applying active tension at a flexor tendon repair site, but perhaps so simple a concept as reversing the order of suture placement with conventional repair.[109,110] Placing the epitendinal suture before the core suture will make the repair better aligned, and it will slip through the pulley with less resistance, and adds 22% tensile strength.[110]

We may find that the engineer will be able to determine the numbers that have thus far eluded us regarding the resistance to tendon glide in vivo that is caused by edema, hematoma, swollen tendon, and tight pulley. They are now beginning the development of a stress transducer, projected to be a micro-sized sensor about the size of a period on a typewriter, that can be implanted and left in a repaired tendon to measure its tensile forces throughout the wound healing stages.[111,112]

Perhaps we as therapists will reach a level of sophistication in our postoperative management programs by improving our knowledge of biomechanics, tendon repair technique, and tendon healing that we will have a working knowledge of the number of tendon repair tensile strengths and of the internal tendon forces imposed on that repair with our split geometry and application of stress

through exercise. Perhaps we are the missing link in the successful management of flexor tendon repair.

We have much to learn about the management of healing tissues and much to contribute to the disciplines of hand surgery and hand therapy through our unique observations. The *new order* of American healthcare is asking for better, faster results. There will be no time or money for those who practice generic hand therapy. Take a closer look at your management of the healing tissues and focus as much on *why* as you do on *how*. Some of the answers to today's political dilemmas may be found in our current understanding of basic science. One thing is certain: relaxing ethical, clinical, and scientific standards will not be the answer.

In 1970 Richard Bach wrote a story about a seagull.[113]

More than anything else, Jonathan Livingston Seagull loved to fly. While most of the other gulls were just bothering to learn the simplest facts of flight—how to get from shore to food and back again—Jonathan spent his days in fierce concentration, learning to stall, dive, learning more about speed than the fastest gull alive. He worked at the very peak of his ability, ignoring the pleas of the rest of the flock and of his parents to stop his foolish quest of challenging the seagull rules of flight and survival.

He figured out that short wings were the answer to speed, that "the wing strain at a hundred and forty miles per hour wasn't nearly as hard as it had been before at seventy, and with the faintest twist of his wingtips he could ease out of a dive and shoot above the waves, a gray cannonball under the moon."

But Jonathan's research was not appreciated. He was cast out of gull society, banished to a solitary life on the Far Cliffs, branded as recklessly irresponsible for violating the dignity and tradition of the gull family. Jonathan lived the rest of his days alone, sorrowful that the other gulls refused to open their eyes and see, but he was not sorry for the price that he had to pay.

For he had touched excellence in his learning.

He learned that *the gull sees farthest who flies highest.*

He found *that we choose our next world through what we learn in this one. Learn nothing, and the next world is the same as this one, all the same limitations and lead weights to overcome.*

That each of us is in truth an idea of the Great Gull, an unlimited idea of freedom. Everything that limits us we have to put aside . . . that the flight of ideas can be as real as the flight of wind and feather.

His love for learning, his passion for the science of flight were the wind beneath his wings.

Maybe we have something to learn from Jonathan Livingston Seagull.

It has been an honor and a pleasure to deliver the Nathalie Barr lecture. I have had many long walks on the beach this summer reflecting on these words and on the love that I have for the people who have inspired them.

As with Jonathan Livingston Seagull. . .

You are the wind beneath my wings.

The lecture concluded with a short film crediting those special people in my personal and professional life who have inspired this lecture. Instrumental: *The Wind Beneath My Wings, Larry Henley and Jeff Silbar, 1982 Warner House of Music and WB Gold Music Corp.*

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