Medical Progress

Ununited Lower Limb Fractures

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Nonunion is a fairly common complication of fracture management, with an overall rate of about 3% for the skeleton as a whole and 9% for the tibia. High-energy injury fractures have a nonunion rate as high as 75%. Other factors that may lead to nonunion are inappropriate treatment, infection, and preexisting disease.

The diagnosis of nonunion is based largely on clinical examination. Plain radiographs and tomograms, computed tomograms, and contrast imaging may be used to confirm nonhealing. Radionuclide imaging can help determine the presence of infection, an impaired blood supply, or impaired osteogenic activity at the fracture site.

The treatment of ununited fractures is based on the principles of good fracture management: adequate immobilization, asepsis and soft tissue cover, osteoconduction (bone contact), osteoinduction (stimulation of bone growth), and metabolic well-being. New modalities for osteoinduction are promising adjuncts to standard treatment, the autogenous bone graft, but conclusive proof of efficacy in humans does not yet exist.

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Fracture nonunion is a relatively common problem faced by orthopedic surgeons. A nonunion is defined as a fracture that shows no radiographic or clinical evidence of healing over a specified time period. The time period when a fracture is considered a nonunion depends on the bone involved and the expected healing time for fractures of that particular bone. There are two basic types of nonunion. In a fibrous nonunion, fibrous tissue has grown across the fracture site but does not provide sufficient stability for function. In the second type, a synovial pseudarthrosis—a false joint with its own synovial lining—develops.

In the lower limb, nonunion can occur in any bone but most commonly occurs in the tibia and, to a lesser extent, the femur.[1] In this discussion we will concentrate on the diagnosis and treatment of tibial and femoral nonunions, although the principles apply to nonunions in general.

The overall rate of nonunion for the skeleton has been estimated at 3%.[2] In the tibia, however, the nonunion rate is much higher, having been reported to be as high as 9% for unselected cases[3] and 75% for displaced, open, comminuted fractures.[4] One of the major determining factors in the development of nonunion is the degree of soft tissue injury and interruption of osseous blood supply at the time of the initial injury. High-energy injuries can often lead to nonunion because of the degree of soft tissue damage. Thus patients who sustain lower extremity fractures as a result of motor vehicle accidents and, most commonly, motorcycle accidents are at risk for having nonunion. In an ongoing review of cases of nonunion at our institution, about 60% of tibial nonunions occurred in fractures that resulted from motor vehicle accidents, with most of those being motorcycle accidents. Similarly, 30 of 39 patients in a study by Gershuni and Pinsker were injured as a result of a motor vehicle accident.[5] Other populations at risk include the elderly and patients with an impaired ability to heal caused by metabolic or nutritional deficiencies. In this review, we will discuss the diagnosis, pathogenesis, and treatment of fracture nonunion in the lower limb.

Causes and Pathogenesis

The basic cause of an ununited fracture is the injury that produced the fracture. Fractures that are open, involve extensive soft tissue trauma, and involve bone loss or segmentation all carry a high risk of impaired union.[6] In general, these fractures result from situations with high-energy dissipation such as motor vehicle accidents or falls from a height.

Other factors that may have a causal relationship to nonunions are the treatment method, infection, and preexisting disease.[7] Open procedures for the internal fixation of closed fractures may inhibit healing by further devitalizing the bone, distracting the fragments, or getting them infected. Treatment methods that provide insufficient stability may inhibit union, and uncontrolled infection will devitalize tissue with a similar result.

Diseases that may decrease the rate of union fall into three major categories: metabolic or endocrine, congenital, and psychiatric, including chemical dependency.[8] Metabolic and endocrine diseases known to retard bone formation include diabetes mellitus, thyroid disease, parathyroid disease, rickets and osteomalacia, estrogen deficiency, Cushing's disease, Paget's disease, renal disease, and malabsorption syndromes. Congenital syndromes such as mucopolysaccharidoses and some dysplasias may cause delayed healing. Patients with psychiatric disorders often have difficulty complying with any treatment program; and chemical substances such as alcohol, tobacco, other street drugs, heparin, corti-

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costeroids, and anticoagulants may retard healing directly or through an associated nutritional deficiency.

The pathogenesis of nonunion can in most cases be related to one or more of the following six factors: bony instability, a lack of bone contact, impaired osteogenesis, an insufficient blood supply, poor nutrition, and infection. The importance of identifying the pathogenic factors for each case will become apparent in the discussion of treatment.

If gross motion is allowed at a fracture site, a true pseudarthrosis may form. Small amounts of motion, such as might occur in a well-applied plaster cast, can actually stimulate fracture healing; but if motion exceeds the strain limits of the healing tissue, it will be disrupted, inhibiting union. Those fractures close to joints are at particular risk for nonunion because they are difficult to immobilize, especially if the joint is stiff and all motion is translated to the fracture site.

Bone apposition is important for fracture healing. Cases in which apposition is not maintained, in which there is segmental bone loss, and where soft tissue is interposed between bone ends will probably proceed to an established nonunion.[9]

Patients with diseases or metabolic problems that retard osteogenesis, such as diabetes, can expect delayed union and should be cautioned that their chance of nonunion is relatively high. The stimulation of osteogenesis might be considered earlier in this group of patients than in those in whom normal healing responses are expected.

Dead bone does not heal well. If large amounts of bone are devitalized during the injury or if vascular disease is present, revascularization and healing will proceed slowly, if at all. The rate of nonunion is extremely high in such cases.

Nutritional deficiency, whether from a primary disease or inadequate intake, has a profound effect on healing in general, and fractures are not exempt. While calcium deficiency probably does not have a great effect on fracture healing, protein plays an important role. A nutritional assessment is a routine part of our evaluation of patients with delayed fracture healing.

Diagnosis

The diagnosis of fracture nonunion is based on a combination of clinical and radiographic findings. The clinical hallmark of nonunion in a lower extremity is persistent pain at the fracture site that is aggravated by weight bearing. On physical examination, gross motion of the fracture site confirms the diagnosis of nonunion. Often, however, an examiner must rely on more subtle findings because a nonunion can exist in the absence of gross motion. For example, a fracture that has been instrumented with orthopedic hardware can be stable on examination without bony union. In addition, fibrous tissue can grow across a fracture site and provide sufficient stability to make motion difficult or impossible to detect by examination. Other clinical signs include tenderness to palpation and pain with stress testing. To do a stress test, the examiner grasps the limb on either side of the fracture site and applies stress to the fracture. If this maneuver elicits discomfort, the examiner should expect incomplete bone healing. Some authors also claim that a decrease in sound transmission can be detected across the fracture site in a nonunion by placing a tuning fork at one end of the fracture and a stethoscope at the other,[10] and others have used strain gauges to measure fracture stiffness.[11]

The next step in the diagnosis of nonunion is the plain radiograph. Radiographs must be taken in at least two planes—anteroposterior and lateral—and oblique views are often helpful. These radiographs will help delineate the type of nonunion present. In a hypertrophic nonunion, abundant callus is present, but it fails to bridge the fracture gap. In an atrophic nonunion, there is an absence of callus formation. The bone ends are sclerotic appearing, and the medullary canals are obliterated, with a persistent wide gap at the fracture site.

Even with plain radiographs, the status of healing can be difficult to assess (Figure 1). Several other procedures have been used in this situation. These include plain tomography, computed tomography (CT), osteomedullography, and radionuclide scanning. Because plain radiographs are a two-dimensional image of a three-dimensional object, there may be apparent osseous bridging where none exists. This is especially the case in spiral fractures in which bony overlap can appear to represent healing. Both tomograms and CT scans can make it easier to assess such cases, providing three-dimensional information.

Osteomedullography is a little-used but potentially valuable diagnostic technique. To do this test, dye is injected into the medullary canal of the metaphysis distal to the fracture site. A tourniquet is inflated at the fracture site, and the extremity is imaged under fluoroscopy. In a positive test, evidence of vascular continuity across the fracture site is seen. A positive test indicates that the fracture will most likely heal without surgical intervention, but it does not correlate with the rapidity of healing.[12]

Radionuclide scanning has been recommended to delineate physiologic from pathophysiologic healing. Results have been mixed. Scans using technetium Tc 99m methylene diphosphenate have shown the presence of a synovial pseudarthrosis.[13] In addition, some authors have used radionuclide scans to determine whether the fracture has the biologic

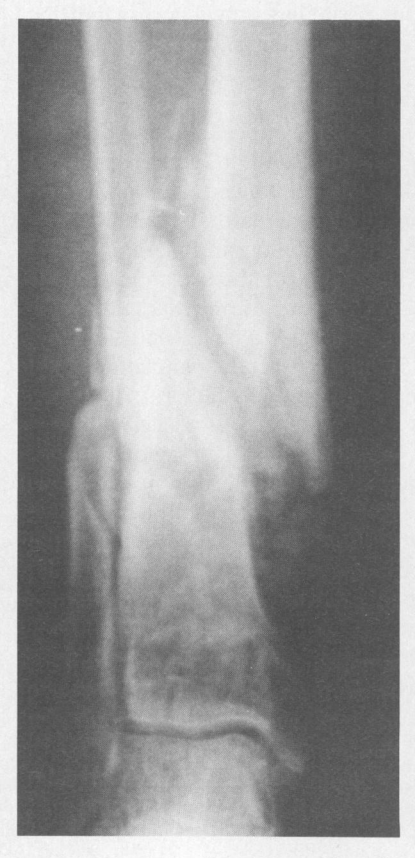

Figure 1.—The radiophotograph shows a tibial nonunion. Note the callus apparently filling the fracture gap. Tomograms revealed the absence of a bony bridge.

ability to respond to a specific therapy such as electrical stimulation. With mature nonunions, radionuclide scans can identify large hypovascular areas that have no potential for healing. In such cases, operative intervention is needed.

In a case of nonunion, the possibility of infection must be considered. An increase in activity at the fracture site on the radionuclide scan is consistent with both bony healing and infection. Infection at the fracture site can also be a cause of persistent pain and contribute to the nonunion. Scans using leukocytes tagged with indium 111 may help differentiate subclinical osteomyelitis in these cases.[14]

Treatment

Principles

The treatment of ununited fractures generally follows the principles of basic fracture care: providing adequate mechanical stability in a satisfactory biologic environment. Those factors mentioned previously that are responsible for the creation of the nonunion, once identified, are modified to allow healing to proceed. Pathogenic factors invariably cause deficiency in one or more of the following treatment principles:

- Adequate immobilization
- Asepsis and adequate soft tissue cover (blood supply)
- Osteoconduction
- Osteoinduction
- Metabolic, including nutritional, well-being.

Immobilization

Adequate immobilization is that which provides sufficient bony stability to allow vascular invasion and soft tissue metaplasia within the nonunion. In some cases, stabilization alone can promote union. Depending on the fracture type, stability may be achieved best by external or internal methods. Nonunions that are inherently stable generally require limited immobilization provided by a cast, splint, or orthosis. This external method has the advantage of being noninvasive but the disadvantage of immobilizing adjacent joints, with residual stiffness and prolonged rehabilitation.

External fixation, a semi-invasive technique using pins placed into bone percutaneously and clamped to an external frame, can provide excellent stability.[15] Various frame types and configurations are available. Some allow compression, distraction, or axial loading at the fracture site. Advantages of this method include skeletal fixation, adjustability, and soft tissue access. Disadvantages include surgical risks, expense, a possibility of pin tract infection, and the necessity for removal. Early results using an external fixation technique developed by Ilizarov and co-workers in Siberia for cases involving bone loss or infection are encouraging.[16] This device uses fine percutaneous wires under tension attached to a complex frame that not only provides stability but also may be used to move and lengthen bone fragments. This technique is currently available at only a few centers in the western states.

Internal fixation of a fracture or nonunion is a third method of achieving stability and reduction. Pins, screws, plates, intramedullary devices, or a combination are used in a variety of constructs that vary from very flexible, with little inherent stability, to very secure, allowing full function including weight bearing. Pins and screws are generally flexible, while plates, depending on the type and fracture construct, are less flexible. Intramedullary devices are at the more secure end of the spectrum. Although all methods have relative advantages, the ultimate goal of invasive stabilization is to provide satisfactory alignment with fixation sufficient to allow full function. Pins and screws alone require limited surgical exposure, reducing injury to the blood supply, and will control alignment but usually require adjunctive external immobilization to protect the fixation. Plates can produce excellent stability and alignment but require extensive soft tissue dissection and clearly produce avascular areas adjacent to and under the plate. A plate can also prevent fracture ends from impacting and, if rigid, shield the bone from normal stresses that influence strength and remodeling. Intramedullary nails generally provide excellent stability and allow fracture impaction with the transmission of load through the healing bone, augmenting remodeling (Figure 2).[17,18] Intramedullary devices, however, destroy all or part of the endosteal blood supply of the bone shaft that, in a normal bone, accounts for about two thirds of the total supply. The use of these devices in a nonunion often prolongs healing. Implanting metal devices, because of the associated devascularization, is not recommended with a preexisting infection.

Asepsis and Soft Tissue Cover

With a high percentage of nonunions coming from open fractures and fractures with extensive soft tissue trauma, infection and a marginal blood supply are present in correspondingly high numbers. If present, infection must be treated with thorough debridement and aggressive local care.[19] Antibiotics administered through a tube or continuous irrigation systems may be more useful than parenteral antibiotics because of poor vascularity. Resecting infected bone is desirable in terms of curing the infection but may not be possible without leaving large bony defects that require long

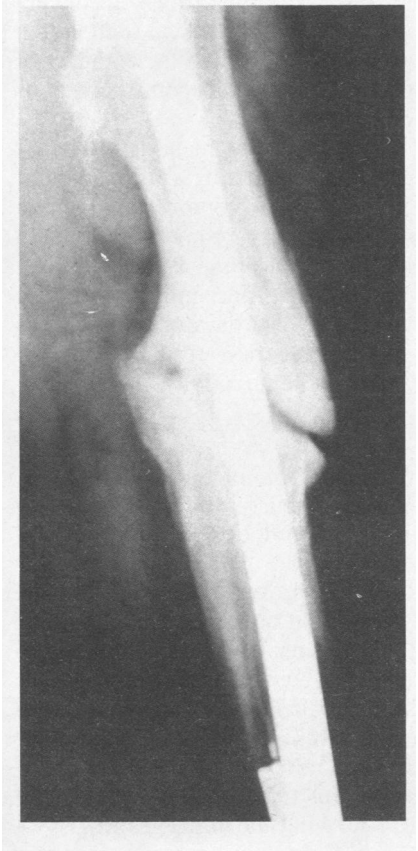

Figure 2.—A femoral nonunion is treated with intramedullary stabilization. An implantable bone growth stimulator is seen distally.

and complex procedures for reconstruction. A subacute or chronic infection in a bone showing nonunion may be made quiescent with aggressive local care, allowing the treatment to proceed largely as in an uninfected case. When the infection cannot be controlled and all attempts at achieving union have failed, amputation should be considered. We have had many patients, disabled by an infected nonunion, who were able to get on with their lives and to be productive members of society following amputation. Although amputation should be mentioned as a treatment and a possible end result of the treatment of a complex nonunion, it should never be seriously considered until the patient has thoroughly weighed the alternatives, is completely familiar with the procedure, and requests it.

If soft tissue loss is present, the blood supply to the healing area is severely impaired. Covering exposed bone at the site of nonunion with well-vascularized soft tissue will assist the treatment of infection and maximize the chances of healing. With relatively small wounds where a healthy bed of granulation tissue can be produced with aggressive local care, healing by secondary intention—which can take a long time—or split-thickness skin grafting may be all that is needed. With larger tissue defects, coverage is achieved with local pedicle muscle flaps or free vascularized musculocutaneous transfers.[20] Nearly all large medical centers now have the capability of doing free vascularized tissue transfer, and no comprehensive treatment program for nonunion should be without this option.

Osteoconduction

To heal, a fracture must have a scaffold that new bone can use to bridge and heal the gap between bone ends. In normal fracture healing, the scaffolding is usually provided by the fracture callus or direct bony apposition. No scaffolding is present in a nonunion, so it must be provided. Osteoconduction is the term used for this purely mechanical scaffolding. Fractures with bony defects larger than half the diameter of the bone often lose osteoconduction and become nonunions because the callus cannot "jump the gap."

The most straightforward way of augmenting osteoconduction is to ensure direct bony contact by axial loading of the nonunion with weight bearing or compression using a fixation device. This step alone may promote healing. In cases where bony apposition is not possible, a scaffolding may be provided by interposing a bone graft or synthetic biocompatible materials such as hydroxyapatite or porous ceramics.[21] While all of these materials provide osteoconduction, only bone grafts have biologic activity.

Osteoinduction

Osteoinduction is defined as biologic stimulation that causes bone to form. If all previously discussed treatment principles are followed and union does not occur, osteoinduction should follow.

At present the only clinically proved method to stimulate bone formation, the so-called gold standard, is grafting with autogenous bone.[4,21,22] The results of all other techniques must be compared with those of autogenous bone grafting in controlled human trials. Thus far, no other technique has been shown to equal or better this method. Boyd and associates reported 262 cases of nonunion of the tibial shaft, 65% of which resulted from open fractures, treated with autogenous cancellous bone grafts.[1] After the initial grafting, 88% healed. Additional grafting improved the success rate to 93%. Nonunion in their series took, on average, about nine months to heal after grafting. Autogenous grafting with cancellous bone from the iliac crest remains our initial treatment of choice for most cases of nonunion.

For patients with nonunion with large bone defects, free vascularized autogenous bone grafts, often with adjoining soft tissue transfer, are a consideration.[23,24] Sources of bone for these transfers include fibula, iliac crest, and rib. Although these grafts tend to heal well, long periods of time are required for them to remodel to sufficiently withstand the large loads in the lower limb. Stress fractures through the graft can develop during this time.

In those patients who require repeated grafts, sources of autogenous bone may disappear. Allograft bone, now readily available from many sources, is an alternative. Although the biologic activity of allograft relative to autograft bone has not been confirmed, evidence is mounting that the stimulation is comparable.[22] Allograft bone is available in fresh frozen and freeze-dried forms. Irradiation or ethylene oxide is sometimes used to enhance sterility, and all material and donors are rigorously tested for infection. Despite the precautions, the use of allograft bone still has the disadvantage of a risk of biologic and chemical contamination and of reaction to foreign tissue. Advantages are that maximum amounts of bone can always be used and graft donor site morbidity—pain, infection, soft tissue damage—is eliminated.

Records of the electrical stimulation of fracture healing exist as early as the 19th century. In 1841 Harthsorne reported on the use of electricity in the treatment of pseudarthrosis.[25] Garrett in 1866 claimed success in the treatment of nonunion by applying a direct current at the fracture site through gold needles.[26] In 1953 Yasuda published a report on the relationship between electricity and bone formation, noting that a current passed through bone elicited new bone formation in the area of the cathode.[27] Becker and colleagues confirmed this work, showing that bone when stressed had an electrical potential proportional to the applied stress and that bone under compression was electronegative while the opposite was true for bone under tension.[28] On the basis of these findings, Bassett and co-workers implanted direct current devices in dog femurs and found bone formation around the negative electrode.[29] Many other investigators have shown the effect of small electrical currents on osteogenesis.

Reports of the electrical stimulation of bone grew rapidly. The majority, using a variety of electrical techniques, were encouraging.[30] Of 119 articles reviewed by Spadaro, only 6 described negative or equivocal results.[31] Brighton summarized the findings of these reports as follows:

- Electrically induced osteogenesis shows a dose-response curve in which current levels of less than 5 μA delivered through a cathode do not produce osteogenesis, current levels of 5 to 20 μA produce progressively increasing amounts of bone formation, and currents of greater than 20 μA begin to cause cellular necrosis;
- Electricity can accelerate fracture healing in laboratory animals;
- Given the proper criteria, electricity can induce bone formation in the absence of trauma;
- The reaction at the cathode results in oxygen consumption and hydroxyl radical formation; and
- Constant direct current is more effective in producing bone than pulsed direct current.[32]

Three types of electrical stimulation are available. The first involves the direct stimulation of tissue through electrodes that are either percutaneously inserted or implanted through a surgical incision. The second method was developed as an alternative to surgical implantation and involves the use of electromagnetic fields applied externally through coils strapped to the limb. The third is also noninvasive and uses a sinusoidal electrical field applied through electrodes attached to the skin. Paterson and associates reported 86% healing in a series of 47 patients with delayed union and 37 with nonunion using one to three implants of a direct current stimulator.[33] In a series of our patients with nonunion treated with the implantable stimulator, 68% healed after one implant and 87% healed with two or three implants. Bassett and colleagues reported a series of 127 patients with tibial nonunions treated with pulsed electromagnetic fields with a healing rate of 81%.[34] Other authors report healing rates of 64% to 71% using this method,[35] and 68% healed in our series. Brighton and Pollack, using the electrical fields, cite a 77% success rate in healing cases of nonunion.[36] We have no experience with this method.

At our center, electrical stimulation is reserved for those patients in whom adequate bone grafting procedures have failed or who have a contraindication to grafting. In recalcitrant cases, an implantable stimulator is sometimes used in conjunction with a bone graft (Figure 2), recognizing that a second surgical procedure will be necessary in 6 to 12 months to remove the current generator. In patients who refuse surgical treatment, cannot tolerate an operation for medical reasons, or whose local skin condition substantially increases the risk of complications, pulsing electromagnetic field stimulation is used. Compliance is essential and must be considered when choosing patients for this method. Noninvasive electrical stimulation with a fracture gap of greater than 5 mm or with a synovial pseudarthrosis has a poor success rate.

Mechanical stimulation methods to heal nonunions are probably effective, although evidence is still inconclusive. The earliest, and perhaps most convincing, method of mechanical stimulation is repetitive axial loading, such as weight bearing.[37] In the presence of adequate bending stability, weight bearing appears to accelerate the fracture healing process if impaction and load transfer through the bone is allowed, such as in a walking cast. That this same process will induce healing of a nonunion is implied but not yet proved. Early reports of mechanical stimulation through ultrasound claim success but have not been verified.[38]

Urist and co-workers have succeeded in purifying a protein from bovine bone that stimulates bone formation, bone morphogenic protein.[39] Studies of this substance carried out in animals are encouraging; as availability increases, it may become a viable alternative to autogenous bone grafting.

Metabolic Well-Being

The final intervention we can provide is metabolic. Adequate caloric intake, especially of protein, is absolutely essential for adequate fracture healing.[40] Mineral intake is less critical but should meet the minimum daily requirements. More than half of our patients with nonunions have had dietary deficiencies, making nutritional analysis and augmentation an integral part of the treatment program.

Chemical dependency often leads to nutritional deficiency and must be addressed as well. Medications that retard bone formation and healing should be eliminated if possible. These include corticosteroids, nonsteroidal anti-inflammatory drugs, anticoagulants, chelating agents, and heparin.

Summary

Nonunion is a common complication of fracture management in the lower limb, especially of those fractures that result from high-energy trauma such as motorcycle accidents. A knowledge of the pathogenesis of this complication aids in the diagnosis and treatment. Although increasingly sophisticated diagnostic modalities are becoming available, none have yet replaced a clinical examination for assessing fracture union.

The best treatment of nonunion is prevention. Good fracture management coupled with early recognition and rectification, if possible, of those factors that retard healing will minimize the likelihood of the complication. When nonunion does occur, the basic criteria of adequate stability, asepsis and an adequate blood supply, osteoconduction, osteoinduction, and metabolic well-being must be met. Optimum management dictates that it be individually tailored to each case. Autogenous bone grafting remains the procedure of choice for stimulating bone growth, although stimulation by electrical means or other bioactive materials may, in some cases, be useful adjuncts or even alternatives.

The final message is the one too often ignored: do not neglect the nutritional status of the patient. No treatment, even the most sophisticated, can replace the body's innate ability to heal itself given sufficient raw materials.

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Book Review

The Western Journal of Medicine *does not review all books sent by publishers, although information about new books received is printed elsewhere in the journal as space permits. Prices quoted are those given by the publishers.*

Asthma, and What You Can Do About It

Milton Millman, MD. Tidal Press, PO Box 1969, San Diego, CA 92112, 1988. 204 pages.

The author states that "patients and the general public usually lack sufficient medical information about asthma to judge whether or not they are getting the best treatment currently available." He attempts to provide the missing information in a language readily understandable by those with limited medical knowledge. Dr Millman, with associates Frank Millman, Ira Goldstein, William Grundon, and Alex Mercandetti, has produced a book that is as easy to understand as possible, with simplified explanations. This is done in four parts: Introduction to Asthma and Allergy; Diagnosis; Treatment; and Methods for Avoiding Allergens; along with a glossary and references. The book is written primarily for patients, parents of children who have allergies, and others who must work with this subject on a day-to-day basis. The 19 chapters contain numerous easily understood illustrations.

Asthma, and What You Can Do About It is not designed to be a self-treatment manual but is to serve as a home reference that would enhance the instructions and information provided by the treating physician. Of particular help is the section on environmental control of allergens. In the next edition of this book, Dr Millman should emphasize important rehabilitation factors such as summer camps and physical exercise programs for asthmatics, as well as information concerning asthmatics in the school system.

My own patients and I think that this book is quite suitable for the asthmatic patient and his or her family. Its proper use can serve to help prevent the need for hospital stays when the information provided is coordinated with the recommendations of the private physician. This book contains much information that when properly studied will allow the reader to understand that there is more to asthma than is realized by most physicians or the general public.

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