Preface

For over 30 years, the diabetic Charcot foot has both intrigued as well as perplexed me. Indeed, it was a patient with this disorder during my student clinical rotations in 1975 that set me on the course that my career has taken. At that time, Charcot joints were still considered to be very rare and usually associated with tertiary syphilis as Jean-Martin Charcot himself had first described in his landmark paper of 1868. In 1883 he also published the first cases involving the foot, calling this deformity “pied tabétique.” Since Jordan’s first mention of a neuropathic diabetic patient’s “rather typical, painless Charcot joint” of her ankle, diabetes mellitus has increasingly become the disease with which this severely destructive bone and joint disorder is associated. Aside from J-M Charcot himself, the person who contributed the most to our modern understanding of this entity was Sydney Eichenholtz. His classic “Charcot Joints” monograph published in 1966 brought this disorder to the forefront of medicine and orthopedics at a time when the incidence of tabes dorsalis was declining. Although his was a review of experience with just 68 patients (including only 12 with diabetes mellitus), he vividly illustrated the various presentations and natural history of joint affections of the upper and lower extremities and offered his now widely adopted classification scheme based on clinical and radiographic parameters. A chapter was devoted to the neuropathic foot wherein he described the common changes noted in Charcot feet associated with diabetes, leprosy, and alcoholic neuropathy including their natural history and the frequency with which foot ulceration complicates this condition. Indeed, there is really nothing new under the sun.

In the last several decades there has been a literal explosion of interest in the Charcot foot from all corners of the globe. This enigmatic complication of diabetic peripheral neuropathy (or other causes of neuropathy) is indeed a complex process, and one that very often places the limb at risk of amputation. A significant risk factor for both ulceration and amputation, even severe instability can lead to limb loss when conservative or surgical measures fail to adequately address the deformity. While conservative care remains the mainstay of therapy, surgical treatment of Charcot feet and ankles has assumed a more important role in the comprehensive management of this condition. Unfortunately, there are no prospective trials as of yet comparing these two approaches to provide guidance in selecting appropriate treatment regimens for a given stage or pattern of involvement.

The most exciting advances have come in the last decade when the underlying nature of bone physiology, especially that associated with osteolysis, has been elucidated. The discovery of pro-inflammatory cytokines, nuclear transcription factor kappa beta (NF κ beta), RANK, RANK-L and OPG along with other modulatory cytokines and peptides have lead to a better understanding of the destructive processes at work in the bone dissolution stages of Charcot joints. This has lead to new pharmacologic approaches aimed at modifying (mitigating) the osteoclastogenesis that drives the active osteolytic activity that is the hallmark of acute Charcot osteoarthropathy. In the near future there will likely be discovered a genetic profile that predisposes persons with peripheral neuropathy to develop osteoarthropathy. This will not only allow us to be more pre-emptive in our efforts to diagnose this complication during its earliest prodromal stage (Stage 0), but may also lead to novel pharmacologic therapies aimed at a more rapid resolution of the acute

inflammatory stage. As it is now, preservation of the architecture of the foot will always be the final goal.

This text is an attempt to provide the reader with the most current information available on the pathogenesis, natural history, and management of the diabetic Charcot foot. Many of the world's experts have come together to present their unique perspectives on this disorder. It is our hope that this work can become a valuable resource for those less familiar with the peculiarities of this often misunderstood complication of peripheral neuropathy. I am indebted to each author for the contribution of their time, efforts, and expertise.

Notwithstanding, Eichenholtz's own words are as relevant today as they were in 1966: "One must therefore surmise that the final word has not been written on the pathogenesis of Charcot joints. No one theory fits all cases; each theory fits some cases." Indeed, much is yet to be learned about the underlying nature of this condition as well the optimal ways to manage it during the various stages of its presentations. Charcot himself was very prescient when he concluded his famous 1868 manuscript with the words "Sera continué."

"To be continued..."

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Acknowledgement

This text is the culmination of many years of interest in this fascinating disorder that, unfortunately, has caused so much heartache and limb loss for too many of my patients. Consequently, I am obliged to acknowledge my own as well as the tens of thousands of persons around the globe that have suffered not only from the diabetic Charcot foot, but osteoarthropathy caused from peripheral neuropathy of any etiology. Along the way there have been many colleagues and mentors that contributed to a growing understanding and interest in this condition to whom credit is due. Most of them are authors in this book. I would especially like to acknowledge two physicians who played a major role in the early development of my interest in the Charcot foot: Martin Bleckman, MD and George Kozak, MD. Both of these superb physicians took a young podiatrist under their wings and gave him the opportunity to flourish. I have been and always will be indebted to them. To my good friends and colleagues Larry Harkless and Andrew Boulton I credit with imbuing me with the need for critical thinking and an academic approach to the practice of medicine. They have both influenced me more than they can imagine, and for that I am grateful. To Lee Sanders, a friend, colleague, and coauthor who has explored this subject with me over two decades, thank you for your keen insights and eloquence. I am also indebted to the many students and residents that I have had the opportunity to work with over the years from Boston to Des Moines to Phoenix. It is an honor to train young physicians and a source of great pride as their accomplishments far outweigh any of those that I can claim. A special word of thanks goes to Vicki Armstrong, library assistant at the medical library of the Phoenix VA Health Care System. She graciously provided me with any and all requests for articles used during the research for this project, no matter how difficult they were to obtain.

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