REFLEX SYMPATHETIC DYSTROPHY

CURRENT MANAGEMENT OF PAIN

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REFLEX SYMPATHETIC DYSTROPHY

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SERIES EDITOR FOREWORD

Painful disorders following injury of peripheral nerves, bones and other soft tissues have occurred from the earliest times of human existence. Ambroise Pare was called upon to treat the persistent pain experienced by King Charles IX which was caused by a lancet wound. The pain was persistent, diffuse and associated with contracture of muscles. The king could neither flex nor extend his arm for a month until the pain finally disappeared.

Weir Mitchell, G.R. Moorehouse, and W.W. Keene produced a monumental treatise in 1864 titled "Gunshot Wounds and Other Injuries of Nerves," which contained an account of symptoms and signs of peripheral nerve injuries as observed in Unionist Soldiers. After 1864, however, little mention of this condition was made during peacetime until a spate of articles appeared again after World War One and Two.

With the formation of societies such as International Association for the Study of Pain, renewed interest has been shown in understanding the mechanisms and management of pain syndromes. Pain caused by sympathetic disorders has always caught the fancy of clinicians, and yet confusion exists as to the etiology and proper treatment of reflex sympathetic dystrophy. Many new names have been proposed for these syndromes; recent ones include sympathetically or non sympathetically maintained pain.

Taxonomy of The International Association for the Study of Pain lists causalgia and reflex sympathetic syndromes as two distinct entities. All clinicians seem to feel that pain relieved by a diagnostic sympathetic block should be labeled as causalgia or reflex sympathetic dystrophy. Similarly, numerous therapeutic modalities have been proposed. They all center around sympathetic denervation of some sort, pharmacologically, chemically, or surgically. In spite of a great advance in our understanding of pain mechanism in the last quarter century, we are no closer to improving the outcome of patients with severe reflex sympathetic dystrophy. Etiology and incidence is

still unclear. Diagnosis is made late and treatment is not standardized. Clinicians who treat causalgia and reflex sympathetic dystrophy have different treatments based upon their background and experience, rather than on the mechanism of the syndrome itself.

The time is opportune now to gather some unbiased thoughts on RSD and clear the air. Our editors, in particular Michael Stanton-Hicks, need to be congratulated for organizing a timely symposium on the subject and inviting international experts to discuss the pathophysiology and treatment of RSD. What follows in this monograph is a clear, concise presentation and discussion of nomenclature, etiology, incidence, mechanism, treatment, and outcome of RSD.

I have no doubt that the readers will find this new information useful in management of patients with RSD in their daily practice.

PREFACE

The syndrome of Reflex Sympathetic Dystrophy is one long recognized clinically by those providing treatment for chronic pain. Despite this, basic research has been sparse with little support from clinical studies to clarify our understanding of the syndrome or reveal its pathophysiology. While many clinical investigators have added their own diagnostic points and new terminology, confusion rather than consensus now prevails.

Provocative enquiry by recent clinical researchers like P. W. Nathan and J. J. Bonica challenged conventions of the day encouraging much of the momentum in study, which has led to the Workshop and material appearing in this text.

To discuss the syndrome RSD, clinicians and basic scientists drawn from 9 countries gathered at Schloss Rettershof, Kelkheim in West Germany last Fall. In keeping with its present description as a triad of autonomic, motor and sensory disturbances in an extremity following a precipitating event, the participants reviewed RSD against all of the other descriptions that are now assembled under the term sympathetically maintained pain. There was general agreement that the sympathetic nervous system is variably involved with the generation and maintenance of the clinical phenomena of RSD but that the syndrome is probably aneurologic disease.

The charge of the Workshop was an attempt to develop a statement that might more clearly define the syndrome of RSD, provide minimal diagnostic criteria and screening tests as well as confirmatory laboratory methods and to offer guidelines for future epidemiological, basic and clinical research. While the material listed in the table of contents accurately reflects the topics discussed, it may not belie the differing points of view that were expressed throughout the Workshop; the greatest difficulty being what should be included under the term RSD.

We hope also that clinicians will be encouraged to maintain outcome audits of their cases and also that therapists will focus their treatment on multidisciplinary management techniques. At the very least it is hoped that this meeting and its text will consolidate and coordinate efforts of those working in the field of pain and rehabilitation, for patients with posttraumatic painful disorders. More immediate tangible consequences of this Workshop include the formation of a special interest section with the IASP, under whose sponsorship the Workshop was held, and submissions for a redefinition of RSD terminology of the Taxonomy of Pain. A synthesis of these ideas and a suggested definition of RSD can be found at the end of the text.

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Section I

GENERAL CONSIDERATIONS

1

REFLEX SYMPATHETIC DYSTROPHY: CLINICAL FEATURES

Stephen H. Butler

INTRODUCTION

In order to discuss the clinical presentation of the disorder Reflex Sympathetic Dystrophy (RSD), a definition of the term is necessary.

The term was first used by Evans in 1946 (5), with reappearance over the next few years in a series of articles in the surgical literature. It was further popularized in the pain literature by Bonica (1), and came to indicate a spectrum of previously distinct syndromes. They have in common, regional pain, vasomotor and integumentary findings of varying severity, up to and including causalgia, a distinct entity following nerve injury. RSD has been defined by the International Association for the Study of Pain (IASP) as: Continuous pain in a portion of an extremity after trauma which may include fracture but does not involve a major nerve, and is associated with sympathetic hyperactivity (14). Although causalgia is left as a separate syndrome, this is arguable since the symptomatology and clinical presentations of the conditions overlap, as do their treatments, and possibly also their pathophysiology.

A review of the clinical syndromes which Bonica (1) grouped under the umbrella term "minor reflex sympathetic dystrophies" gives insight into the clinical presentation of the disorder or syndrome known as RSD:

Sudeck's atrophy
traumatic arthritis
minor causalgia
posttraumatic osteoporosis