Introduction

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Reflex sympathetic dystrophy (RSD) is a complex regional pain syndrome and probably first described by Ambroise Paré in the 16th century. Sudeck reported in 1900: "Es handelt um eine acute Entzündung des Handgelenks, mit frühzeitig auftretender Steifigkeit und Schmertzhaftigkeit der Fingergelenke und sehr bald—nach mehreren Woche—stark ausgesprochener Atrophie des ganzen Handskelettes" (Sudeck 1900). Since then, the commonest name for this pain syndrome has been Sudeck atrophy. During the centuries, the thoughts about the pathogenesis, definitions and therapies have changed and they still change. Many names have been given to this syndrome and related conditions. More than 30 different names in the French literature, more than 80 different names in the Anglo-Saxon literature and more than 50 were found in the German literature. The Dutch literature has 15 names for this syndrome. Many are related to a supposed pathogenesis (e.g., reflex sympathetic dystrophy, paralyse d'ordre reflexe), to the clinical signs (e.g., swollen atrophic hand, Stauungsatrophie), to diagnostic findings like those on radiographs (e.g., Sudecksche Knochenatrophie, osteotrophie traumatique) or to the inciting event (e.g., rheumatisme neurotrophique, posttraumatic dystrophy) (Veldman 1995, Kurvers 1997).

The causative event of RSD may vary considerably, including a minor strain, a contusion, a fracture, frostbite, a myocardial infarction and cancer. Sometimes it arises spontaneously or the causative event was so small that the patient cannot even remember it.

RSD has many clinical symptoms such as pain, edema, discoloration, hyperhidrosis, etc. (Veldman 1995). The intensity of the pain is disproportionate to the causative event. This suggests a behavioral or psychological component. Several authors have expressed their ideas about the involvement of psychological or psychosocial aspects in the developing of RSD or in consequence of it (Houdenhove van et al. 1992, Lynch 1992, Bruehl et al. 1996).

Risk factors for RSD are coexisting diseases such as hyperlipidemia, diabetes mellitus, hemiplegia or alcoholism. Women between the ages of 40 and 70 are more at risk than others. In the pathogenesis of RSD, two main theories exist: involvement of the sympathetic nervous system or an abnormal inflammatory tissue reaction (Goris 1985, Jänig and Stanton-Hicks 1996). The involvement of the sympathetic system is assumed to be sympathetic overactivity in the affected limb. This assumption is based upon the observation that skin blood flow, and consequently the temperature, are reduced in the affected limb in many patients and that blocking the sympathetic efferent pathway to the affected limb relieves these symptoms (Bonica 1990). Others hypothesized that peripheral interactions between efferent sympathetic and afferent nociceptive pathways maintain a central vicious circle, accounting for vascular, sensory and motor disturbances. The theory about the abnormal inflammatory tissue reaction was described in 1982 by Fantone and Ward. According to that theory, hydroxyl radicals play an important role in the inflammatory processes. The hydroxyl radicals are produced by activated phagocytes or by ischemia. Excessive production of the radicals leads to destruction of healthy tissues, which may lead to RSD. Positive results of treatment with scavengers substantiate this theory (Goris 1985, Veldman 1995).

The diagnosis is mostly made on the basis of clinical criteria, but no uniformly defined criteria exist (Amadio et al. 1991, Jänig and Stanton-Hicks 1996). The International Association for the Study of Pain (IASP) published in 1994 a revised taxonomic system for RSD (Merskey and Bogduk 1994). "Complex Regional Pain Syndrome, type I", earlier described as RSD, is a syndrome that usually develops after a noxious event, is not limited to the distribution of a single nerve and is apparently disproportionate to the inciting event. It is associated at some point with evidence of edema, changes in skin blood flow, abnormal sudomotor activity in the region of pain or allodynia or hyperalgesia.

Many physicians believe that the diagnosis of RSD can be made only when additional investigations have been performed, such as a nuclear scan, thermography, testing of the sympathetic system or radiography. Evidence for this belief is lacking, however.

More than 50 treatments are described in the literature for RSD patients. These recommended treatments can be divided into a few subgroups: physical therapy, drugs applied to the skin (DMSO), drugs administered regionally (guanethidine), drugs adminis-

tered systemically (corticosteroids), drugs administered at the level of the spinal cord (opiates), nerve blocks with local anesthetics, surgical procedures, anesthesiological procedures, neurosurgical procedures (thalamotomy), electrical neurostimulation and miscellaneous (radiation therapy, acupuncture) (Kurvers 1997). Most authors claim success rates of about 60–70%. This claim conflicts with the finding that a lot of patients frequently go from doctor to doctor (medical shopping), specialists included. Individual patients may, over the years, undergo many types of treatment, including sympathectomy, protracted physical and/or occupational therapy and/or pharmacotherapy.

In other cases, RSD may lead to chronic invalidity or to amputation of the affected limb. In "Reflex Sympathetic Dystrophy; Current Management of Pain" by Stanton-Hicks et al. (1990), it was concluded that there was a lack of knowledge in the efficacy of treatment, time-dependent symptoms of RSD and psychologic and psychosomatic aspects of RSD." Clinically, as a rehabilitation specialist working in a university hospital, a referral center for patients with complex hand-pathology, I also found little knowledge concerning the efficacy of treatments, time-dependent symptoms and the psychologic and psychosomatic aspects of RSD.

In order to inform RSD patients adequately, extensive knowledge concerning the long-term outcome of RSD is necessary. Until now, only a few studies (Atkins et al. 1990, Field et al. 1992, Inhofe and Garcia-Moral 1994, Subarrao and Stillwell 1981) have described the long-term outcome results, but in only one study (Bickerstaff and Kanis 1994) were the instruments for measuring the impairments described. Consequently, quantification of the extent of the impairments in RSD in other studies is almost impossible. Disabilities and handicap are mentioned only briefly (Field et al. 1992, Subarrao and Stillwell 1981). To quantify the extent of impairments, disabilities and handicaps in RSD, a long-term follow-up study was performed to analyze this outcome in terms of impairments, perceived disabilities and handicaps, defined by us as general health and vocational outcome.

The objective of the long-term follow-up study described in this Supplement was to identify impairments resulting from RSD of the upper extremity and to analyze the relationship between impairment and disability in RSD patients. The study group consisted of a referred sample of 65 RSD patients with clinical signs involving the upper extremity. RSD developed after fractures of the wrist or hand in 29 patients (45%) or after a carpal tunnel release in 9 patients (14%). The mean interval between the diagnosis of RSD and the follow-up was 5.5 (3–9) years (SD = 0.76). The main outcome measurements were the impairments measured by standard physical examination. Activity of daily life (ADL) and pain were assessed on a visual analogue scale (VAS). Pain was assessed directly before and after the physical examination and pain perceived in the week before examination was determined. Significant differences in impairments were found between the affected and the unaffected sides ($p<0.05$). According to the AMA guides, the impairments found should not cause disabilities. Significant correlations were found between VAS-ADL and VAS-pain in the last week prior to evaluation ($r = 0.65$) and full-fist grip-strength ($r = -0.55$). Pain was the most disabling factor. It was found that an early diagnosis and early treatment did not lead to less impairment or disability, as was suggested in the literature. Another objective of the long-term follow-up study described in this Supplement was to describe the general health, long-term vocational outcome and psychosocial aspects, such as social life events (SLE), around the inciting event of RSD, and to describe the psychological history of the 65 patients mentioned earlier. General health and long-term vocational outcome were assessed by means of a general health questionnaire (RAND-36) and a structured interview, respectively. SLE (life-change unit > 35) was present in 32 patients (49%). A psychological (or psychiatric) history was found in 22 patients (34%). In total, 60% of the patients had a SLE and/or a psychological history. The pain scores of the RAND-36 of the RSD patients were significantly higher than in a RAND control group. In total, 17 patients (26%) had to change profession due to RSD. Nearly 30% of the patients had to stop their work for more than one year. The results show a high coincidence between RSD and interacting psychosocial disorders, which may play a role in intensifying and prolonging the symptomatology of RSD.

Frequently RSD patients are referred for medical examination to evaluate treatment or to make a report for disability payments from insurance companies. During this examination, impairments, such as muscle strength (and range of motion), are assessed. Repeated measurements of muscle strength (and range of motion) usually result in differences in results (Low 1976). In this study, the three different grip-strengths were measured in 29 upper extremity RSD patients. We used the generalizability theory to assess the extent of disagreement or differences (errors in measurements) within or between observers and interactions between observer-session and repetition of the measurements (Brennan 1992). The aims of our study were to identify the sources of variation in grip-

strength, to analyze the smallest detectable differences (SDD) and to determine the reliability of upper extremity grip-strength tests in RSD patients. The main sources of variation in measurement errors were observer, patient/observer interactions and patient/session/observer interaction and the random error. We found that the generalizability theory is a good tool for estimating the sources of measurement error. Clinical examinations for muscle strength measurements as a part of a total clinical examination—for example, a disability payment or worker's compensation in case of RSD patients—should be done in more than one session and on more than one occassion.

The general conclusion of the studies described in this Supplement is that RSD tends to become a chronic pain syndrome and that social life events and psychological dysfunction play an important role in the pathogenesis of RSD. In "chronic" RSD, pain and loss of full-fist grip-strength are the most evident impairments, of which pain is the most disabling factor. Finally, the interaction patient-observer causes considerable variation in measuring impairments, such as grip-strength during medical examinations.

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