AN INVESTIGATION OF THE ROLE OF CLONIDINE IN THE TREATMENT OF REFLEX SYMPATHETIC DYSTROPHY

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INTRODUCTION

The possible central and spinal mechanisms involved in the transmission of the pain of reflex sympathetic dystrophy (RSD) are discussed in detail in this volume. This study is designed to investigate the possible mechanisms involved in the transmission of pain of RSD at the postganglionic synapse. Guanethidine has been used successfully to treat the pain of RSD as described by Hannington-Kiff (Chapter 12) and (1). This pain relief has been shown to be associated with sympathetic blockade as measured by an increase in skin blood flow, and abolition of the vasoconstrictor ice response (2). Thus the pain relief achieved with this technique is believed, in part, to be a result of sympathetic blockade at the postganglionic synapse, indicating a possible role for the synapse in pain transmission.

RSD most commonly results from trauma in the periphery, and so it is reasonable to assume that some "cause" of the pain may also be in the periphery. This study investigated the role of clonidine, an alpha 2 agonist, given via an intravenous regional (Bier's block) in the treatment of pain in this patient group. Thus it was possible to compare the effect of guanethidine, (a false transmitter of noradrenaline) to clonidine, (blocking presynaptic release of noradrenaline) in the same patient.
PATIENTS AND METHODS

Twenty-two patients agreed to participate in the study, which had approval of the local Ethics Committee. There were 13 females and 9 males. The diagnosis of RSD was made in these patients on clinical grounds, and confirmed by 24 hours of pain relief following guanethidine block, suggesting an involvement of the sympathetic nervous system in the transmission of each patient's pain. Nineteen patients had RSD of various causes (Table 1), 2 had Rheumatoid Arthritis (RA), and 1 had Post-Herpetic Neuralgia (PHN). The mean age of the patients was 54 years with a range of 30-87 years. The mean duration of pain was 45 months with a range from 3 to 240 months. After previous positive responses to guanethidine block, clonidine 150ug in 10ml of physiological saline was compared in a double-blind random fashion to 10ml of physiological saline given intravenously as a Bier's block (4) to the affected limb, on 2 separate occasions at least a week apart. The ice response to skin blood flow was measured by venous occlusion plethysmography (3,5) with a mercury strain gauge on the thumbs or great toes before and after each injection. Pain intensity, pain relief, and mood were assessed by using the visual analogue scale before and after the injection, and again half an hour later just prior to discharge. Blood pressure was also measured manually at the same times, and any other comments by the patient about either injection were recorded. The effect of each injection on the somatic nervous system was assessed before discharge by sensory (pinprick) and motor (power) testing.