Reply:

Dr. Berry raises a very important point in stating that Reflex Sympathetic Dystrophy Syndrome (RSDS) can be mimicked by certain conditions of emotional or neuropsychiatric origin. We also have patients in our practices which have similar complaints and signs as those quoted by him; and the importance of a complete history cannot be overstated.

The International Association for the Study of Pain has addressed this issue recently.[1] In changing the name to Complex Regional Pain Syndrome, Type 1 (RSDS) they have tried to recognize that not all patients with signs of autonomic instability have RSDS. In defining CRPS they have stated "A term describing a variety of painful conditions following injury which appears regionally having a distal predominance of abnormal findings, exceeding in both magnitude and duration the expected clinical course of inciting event often resulting in significant impairment of motor function, and showing variable progression over time".

In defining CRPS Type 1 (RSDS): 1) Type 1 is a syndrome that develops after an initiating noxious event. 2) Spontaneous pain or allodynia/hyperalgesia occurs, is not limited to the territory of a single peripheral nerve, and is disproportionate to the inciting event. 3) There is, or has been, evidence of edema, skin blood flow abnormality, or abnormal sudomotor activity in the region of the pain since the inciting event. 4) This diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction.

In the patients that Dr. Berry describes there is an emotional component involved, and so by the above reasoning, 4) above would therefore exclude CRPS as the diagnosis.

In the case of both of our patients,[2] there was indeed a history of emotional disturbance in the remote past, but psychiatrists or psychologists had been involved and were satisfied that their emotional or psychiatric state had no bearing whatsoever on their current conditions. Secondly, the clinical picture was such that CRPS Type 1 (RSDS) was the only diagnosis that could fit. Both had severe allodynia, massive edema, vasomotor changes, sudomotor changes, dystrophic changes, and major changes on the bone scan showing an underlying inflammatory component. In addition, both had responded (albeit temporarily) to sympathetic blockade and alpha 1 receptor blockade suggesting a sympathetically maintained syndrome picture. Both had severe allodynia which continued despite full sensory and motor blockade under continuous spinal anaesthesia which definitely could not be on the basis of any emotional or psychiatric component.

The bone scan is a very useful diagnostic procedure in cases of doubt. As a general rule, a scan showing decreased uptake of Te^{99} on all three phases would be more suggestive of a primary disuse origin for symptoms, whereas an inflammatory component and generalized increased uptake would be more suggestive of CRPS Type 1.

The more difficult cases are those with both a "true" CRPS Type 1 (RSDS) picture, and in addition an emotional/psychiatric component. It is our practice to treat both simultaneously, and it is indeed important to recognize that both can occur simultaneously in the same patient.

1. Stanton-Hicks M, et al. Reflex Sympathetic Dystrophy: changing concepts and taxonomy. Pain 1995; 63: 127-133.

2. Becker WJ, Ablett DP, Harris CJ, Dold ON. Long Term Treatment of Intractable Relex Sympathetic Dystrophy with Intrathecal Morphine. Can J Neurol Sci 1995; 22: 153-159.

David P.J. Ablett FRCPC
Department of Anaesthesia
Calgary General Hospital
Calgary, Alberta

To the Editor:

Re: Environmental Exposures in Elderly Canadians with Parkinson's Disease. S. Chaturvedi, et al. (Can J Neurol Sci 1995; 22: 232-234)

It was with great interest I read the article[1] on Environmental Exposures in Elderly Canadians with Parkinson's Disease by S. Chaturvedi, T. Ostbye, A.J. Stoessl, H. Merskey and V. Hachinski in the Canadian Journal of Neurological Sciences. The association with an environmental toxin, especially solvents and Parkinson's Disease, has been postulated for many years.[2] I have suggested a possible association with environmental toxins and Progressive Supranuclear Palsy,[3] and I think this recent finding lends support to the environmental toxin theory,[4] immediate or delayed. I should like to add an interesting case study to this article. I saw a lady, (one of the thirteen ex-subjects)[3] who suffered from progressive supranuclear palsy. This lady's husband, whom I interviewed as she could not talk, suffered from Parkinson's Disease. This man had no family history of neurological disorder and both parents had lived well into their seventies. He was an organic chemist and worked in the Far East for Proctor and Gamble after World War II. He and his wife spent many hours travelling in this job, and they both were exposed to solvents at take-off and landing when their plane would be fumigated with aerosol insecticide. Both he and his wife were exposed repeatedly over an eight to ten year period. The only difference was that his wife, who had a flying phobia, would use alcohol as a tranquillizer. He abstained. Many years later she, with no family history of any neurological illness either, began in her early sixties to suffer from and eventually was diagnosed with progressive supranuclear palsy. He later was diagnosed with Parkinson's disease. Could this interesting case study be an example of an association with exposure to solvents and of delayed toxic reaction/action?

This concept has been elucidated more definitively by Tetrud[4] and commented on with another case report.

Much work remains, but this recent work lends further support to some of the original observations of Yosio.[6] The relationship to an environmental toxin should be pursued.

E.McCrank, M.D.
London, Ontario

1. Chaturvedi S, Østbye T, Stoessl AV, Merskey H and Hachinski V. Environmental exposures in elderly Canadians with Parkinson's disease. Can J Neurol Sci 1995; 22: 232-234.

2. Barbeau A, Roy M, Bernier G, Campanella G, Paris S. Ecogenetics of Parkinson's disease: prevalence and environmental aspects in rural areas. Can J Neurol Sci 1987; 14 (1): 36-41.

3. McCrank E. (Letter to the Editor) PSP risk factors. Neurology 1990; 40: 1637.

4. Tetrud JW, Langston JW, Irwin I, Snow B. Parkinsonism caused by petroleum waste ingestion. Neurology 1994; 44: 1051-1063.

5. McCrank E. (Letter to the Editor) Parkinsonism secondary to petroleum exposure. Neurology 1995; 45(11): 2112.

6. Yosio U, Skata E. A case of progressive supranuclear palsy – neurological findings and etiology. J Otolaryngol 1978; 7: 409-414.

https://doi.org/10.1017/S0317167100038932 Published online by Cambridge University Press