

## IS REFLEX SYMPATHETIC DYSTROPHY (RSD)-DYSTONIA AN ORGANIC DISEASE?

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One of the most difficult syndromes that face both movement disorder and pain specialists is the combination of reflex sympathetic dystrophy (RSD), now preferentially called complex regional pain syndrome (CRPS) and dystonia. The syndrome is not rare. Its nature is not known, and it is very controversial since some physicians think these patients have an organic disorder (Bhatia et al., 1993) and some think these patients are all psychogenic (Verdugo and Ochoa, 2000). And, in part because of the confusion, treatment is typically not satisfactory. Therefore, it is more than academic to try to resolve the controversy.

CRPS has two subtypes, CRPS-I, formerly known as reflex sympathetic dystrophy (RSD), and CRPS-II, formerly known as causalgia. These are pain syndromes as a consequence of peripheral trauma, with CRPS-II including a nerve injury. The pain can be severe and is associated with a variable complex of other manifestations including allodynia, edema, color change (red, white or blue), hyper- or hypohidrosis, and dystrophy of the body part. The disorder begins focally and can spread. The nature of the disorder thought to be originally mediated by sympathetic overactivity, and while this may be the case in some circumstances, it is clearly not present in others. Hence, the earlier term of RSD was changed to avoid an inaccurate implication. Some of these patients are thought to be psychogenic. There are many types of dystonia, and one type appears to be post-traumatic in origin (Jankovic, 2001). Trauma as a trigger is controversial, but there are enough cases with dystonia on coming sufficiently close in time and somatotopically related to the injury that most investigators consider this a genuine phenomenon. It might be noted that other movement disorders can also arise from apparently similar trauma such as tremor, segmental myoclonus and tics, and there is no explanation for the variation. The dystonia in these patients tends to be fixed in nature rather than action induced, which is more common for the primary dystonias (Schrag et al., 2004). It can spread to other body parts from the site of origin. Its nature is not understood at all, and is controversial since some of these patients are thought to be psychogenic.

Hence CRPS and post-traumatic dystonia are kindred conditions. Both can be triggered by trauma, but are relatively uncommon consequences of it. The trauma might well be mild, and it would not be completely unfair to say that everyone experiences some type of trauma every day. Indeed, there is some evidence that sometimes the trauma is even an internal lesion. This by itself has been controversial, but more commonly the trauma is more substantial. For example, a Colles fractures or tibial fractures are common causes. In any event, it should not be surprising that CRPS and dystonia might occur together. Features of similarity, in addition to trauma, are the relatively rapid onset, female preponderance, and the possibility of spread.

It seems logical to think that there might be an underlying predisposition of the nervous system in certain people that would predispose them to an abnormal response to trauma. There is some evidence for decreased inhibition and abnormal sensory function as factors in dystonia (Hallett, 2006a), and these may be true also for CRPS. However, there is an overlap of the two syndromes only in a minority, so the predisposition or some other feature of the physiology would have to differ.

Looking at trauma in more detail, trauma certainly can give rise to various intensities and durations of pain. The pain might lead to disuse and reduced mobility. In some circumstances, the body part might be splinted, and there is some evidence to suggest that this might be a particularly important precedent. Trauma could give rise to nerve injury and neuropathy, the latter including small fiber neuropathy.

Trauma as well can give rise to a psychogenic reaction, perhaps via anxiety. It is fair to point out that not only is the pathophysiology of CRPS and dystonia unknown; the pathophysiology of psychogenicity is also obscure (Hallett, 2006b; Hallett et al., 2006). The basic notion is that a psychic trauma becomes converted to a physical symptom. A psychogenic reaction to trauma and pain, perhaps with anxiety, could well lead to disuse.

Both an organic response to trauma and a psychogenic response to trauma can have secondary consequences as a result of central nervous system plasticity. The nervous system reacts to any change. Most of the time, such change is beneficial and adaptive, but change can also be maladaptive. A clear example of a maladaptive change is phantom pain. It is also possible that the occupational cramps result from repetitive activity. Blepharospasm is often preceded by local eye disease, and this might be a good analogy for the relationship between local trauma and induction of dystonia. Moreover, the abnormal predisposition mentioned earlier could be related to the chances that plastic changes might go awry.

Perhaps some tentative primitive conclusions can be drawn. Someone getting this syndrome might have an underlying predisposition to react badly to trauma, and loss of inhibition could be central to this. Trauma acts a trigger that could act directly or by several indirect means including a psychogenic reaction. An abnormal response ensues, either sensory or motor or both, and this can be engrained (and further distorted) by maladaptive plasticity. Hence the end result looks similar if a purely organic route or if mediated in part by a psychogenic mechanism. If it is the case that a high percentage of these patients are psychogenic, we should try to understand why psychogenicity tends to cause this particular syndrome.

### References

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