

Are symptoms of restless legs syndrome generated in the periphery of the nervous system or are they born centrally?

The clinical case “Restlessness in right upper limb as sole presentation of restless legs syndrome”^[1] presented by Gupta and colleagues in this volume and issue of the *Journal of Neurosciences in Rural Practice* refers us to an as-yet debated issue on the pathophysiology of restless legs syndrome (RLS): Are the neuroanatomical sites of generation of the unpleasant, annoying symptoms of the disease peripheral or central?

Karl A. Ekbom, who initiated the studies (1945) on RLS^[2,3] was also one of the first researchers to describe RLS in amputees (1961). In his paper,^[4] through a clear and logical reasoning, he hypothesized that the symptoms were generated in the periphery of the nervous system. The question is still unanswered for most modern researchers of RLS.^[5] However, many insights derived from clinical observations of RLS also influence us to assume that RLS symptoms are generated in the periphery: 1) In amputees, frequently, symptoms are ‘phantom’, that is, felt as if generated from the leg that is missing, 2) if RLS patients move or stretch their legs they experience some relief (seems logical because if the symptoms were central, the restlessness would not cease with movement), 3) massaging the affected limb or applying cold pads lead frequently to amelioration of symptoms (very little probability that such commonplace nonpharmacological measures would alleviate the symptoms if they were generated centrally), and 4) in some subjects submitted to total knee arthroplasty, RLS may ensue only after the surgery and only in the knee articulation with the prosthesis device.^[6,7] Also, the mechanisms underpinning RLS pathophysiology are considered unknown; however,

recently we theorized that the RLS symptoms are secondary to an imbalance between two forces, the hypothalamus-pituitary-thyroid axis and the dopaminergic system,^[8] a hypothesis based on sufficient circumstantial evidence.

RLS may be primary or secondary to a number of conditions including peripheral neuropathies. Gupta and colleagues describe a rather interesting case of a woman who initiated her restless symptoms in the right upper limb. However, the assumption that their case is one of primary RLS must be cautiously treated. RLS is a disease of deranged sensibility, and not of motor impairment. So, efforts to search for peripheral neural impairment might be misleading, as for instance when one studies a possible case of thoracic outlet syndrome clinically, and when only sensitive symptomatology is being observed. One important principle of the physiology of the somatosensory system (principle of the labeled lines) is that when a nerve fiber is stimulated at any point on its path to the sensory cortex, the stimulus evokes symptoms exactly as if they were coming from the territory nourished by the nerve fiber.^[9] As the patient in this case has a cervical rib, only a clinical study might not be sufficient for a precise diagnosis of the case. Is it a case of primary RLS or a secondary one? Would not be possible that the neural fibers, coming from the cutaneous territory where the patient feels the symptoms, were stimulated on their passage through the cervical rib region? Ancillary studies such as electromyography and image studies perhaps could have helped toward a more precise diagnosis: Was the case of Gupta and colleagues’ a primary or secondary one? Another important point to remember is that their patient does not have any close relative suffering from RLS, which makes the diagnosis of a primary RLS a bit more difficult.

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