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The Pain-Adaptation Model: Remembering James P. Lund

The sudden passing in December, 2009, of our dear colleague and friend, James P. Lund, BDS, PhD, a brilliant thinker and scientist, astute observer, passionate critic, and tireless patient advocate, has triggered this reflection on a topic for which he expressed great excitement—the relationship between movement and pain, notably in the context of human persistent pain disease.

Jim's lasting contributions were much broader than the research into the relationship between movement and pain. His PhD dissertation already marked a classic scientific discovery with the conclusive demonstration of the existence of a central neural pattern generator, located in the brainstem, underlying rhythmic, masticatory jaw movements. The further demonstration that jaw reflexes are gated as a function of jaw gape and the directionality of jaw movement represented another major advance for the field. However, among his many lasting contributions to the body of knowledge, not to omit the more recent extension of his interest into clinical trials, there is little doubt that we remember Dr Lund most for his passionate presentations of the Pain-Adaptation Model.

Dr Lund was the chief architect of the Pain-Adaptation Model, which redefined the role of pain in clinical presentations, including persistent muscle pain conditions such as masticatory muscle pain, back pain, and fibromyalgia.[1] The model challenged and displaced the prevailing Vicious Cycle Theory[2] that was directing care for many pain conditions. More recently, Murray and Peck proposed another model that included elements of both.[3]

Lund's Pain-Adaptation Model, an abstract representation of biological processes, presented the general principles that govern the function of muscle at the α-motoneuron and system's levels in situations of muscle pain in humans. It was formulated to be testable for validity. Based upon experimental data, using the intramuscular infusion of hypertonic saline to induce and maintain a constant level of pain, the model predicted that in the presence of pain, α-motoneurons when acting as agonists during muscle work are inhibited while they are facilitated when acting as antagonists; this in turn results in less forceful contraction, or the limitation of the range of motion of the body part in pain, respectively. On the other hand, at rest, the effect of pain on α-motoneurons or resting muscle activity would be negligible. Twenty years after the introduction of the Pain-Adaptation Model, the scorecard capturing the presence or absence of matches between the model's prediction and actual observations remains good, although there have been reports of contradictory findings (for review and discussion, see reference 3). However, Dr Lund was adamant that the few studies presenting higher electromyographic activity at rest in humans were limited to observations gained in the face and not the truncal musculature, suggesting that pain-related expressions linked to the action of nonmasticatory facial muscles likely contaminated the surface electromyographic recordings originating from the muscles of mastication, located beneath the mimetic muscles.

Reports that failed to demonstrate a reduction of bite force during chewing or evidence of antagonistic co-contraction during the opening phase of the chewing cycle, studies that appeared to challenge the Pain-Adaptation Model, included chewing tasks of food boli that required low bite forces and/or a small degree of mouth opening. Such task features did not present a physical challenge as antagonistic co-contraction is a function of jaw gape, becoming more pronounced with wider mouth openings.

Although pain exerts a strong influence on α-motoneuron pools as mentioned above, it has become clear that the same holds true for mood based upon a more recent body of electrophysiological and neuropharmacological data. For example, akinesia has been associated with stroke, dementia, depression, and chronic pain, while hyperkinesias are linked to mania and the effect of medications, such as antidepressants, benzodiazepines, calcium-channel blockers, and others. Outward signs of emotions are observable in the action of truncal, respiratory, vocal, and mimetic muscles. Referring to Darwin, Dr Lund loved to cite the statement of face, voice, respiration, and body postures and gestures "betraying our true feelings despite our efforts to conceal them." Pain, fear, grief, anxiety, and exhaustion are not only expressed in the action of facial muscles but may include masticatory muscles

at low activity levels as well. In fact, up to our very last discussion of the matter, Dr Lund was resolute that variations in motor function are the effect of pain and not its cause, and that the specific effect of pain and effects of pain-related mood explain much of the variance in motor function associated with painful disease.

Thank you, Jim. You have been the "conscience" of the orofacial sensorimotor fields and your intellect and work has stimulated your colleagues and students and will continue to do so for years to come. More importantly, it benefitted patients, challenging senseless treatments by shifting the emphasis onto the importance of managing pain. Your sudden and unexpected death constitutes a profound loss that is hard to "chew" and swallow.

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