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CORR Insights®: Stretching After Heat But Not After Cold Decreases Contractures After Spinal Cord Injury in Rats

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Where Are We Now?

Joint contractures restrict patient mobility [1, 12] and can be disabling, but are the focus of little clinical research. They can limit an individual's autonomy, reduce quality of life, and due to their chronicity, impose a large burden on the healthcare system [7].

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In a previous study [4], patients with rheumatoid arthritis identified the lack of complete joint mobility as their second-most-troublesome problem after pain [4]. Consistently, the second preference for improvement of health for these patients was mobility, with emphasis on walking and handling activities [9].

The current experimental animal study evaluated several therapeutic approaches that sought to restore full ROM in a rat model. They found that preconditioning with heat alone before stretching increased knee ROM in extension.

Still, our patients' calls for effective treatments to reverse joint contractures remain largely unanswered. The sparsity of research activities and conflicting findings on joint contractures mean that it is hard to apply evidenced-based approaches to treatment. Epidemiological studies are few

[2, 6] and report a wide range of prevalence of joint contractures (between 20% and 80%) in nursing homes, hospitals, and the outpatient community. Few causal factors are known and the most-studied are immobility and genetics [3, 5]. The lack of use of the full amplitude of a joint ROM is the common factor associated with the development of a joint contracture [1]. The fact that joint contractures also occur with a number of common but chronic and heterogeneous conditions, such as osteoarthritis, further complicates the study of etiology.

Where Do We Need To Go?

Joint contracture is notoriously difficult to study. There is no consensus on the definition of a joint contracture, on what constitutes a clinically important amount of reversibility or response to treatment. Different settings and a diverse array of data collection methods used by investigators both contribute to our currently limited understanding of many aspects of joint

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contractures including prevalence, etiology, and pathophysiology.

Good care can be defined by sustained stretching, ROM exercises, dynamic bracing, serial casting, and/or serial splinting [1]. The response to rehabilitation varies and is difficult to compare across studies due to the lack of standardized clinical endpoints and quantitative data on ROM. We need those clinical endpoints and data to measure reversibility in terms of gains made in ROM and in response to treatment and physical disability. Additionally, in order to resolve opposing viewpoints on the potential for reversibility, we should determine whether joint contractures are fully reversible [8, 12]. We can do this by comparing ROM measured with a goniometer and the Functional Independence Measure score recorded before and after treatment.

We still cannot identify in all cases, the anatomical structures limiting the natural ROM, the range missing in degrees, and the extent by which function is impaired by a joint contracture. Structures including muscles, tendons, ligaments, and joint capsules have different mechanical properties and respond differently to immobility and to mechanical stimulation. The identification of molecular-level influences on the mechanical properties of joint tissues will provide deeper insight into pathogenesis.

How Do We Get There?

Connective tissue around synovial joints, known as the joint capsule, responds to extended periods of immobility and limits ROM [10, 11]. The application of cutting-edge methods to measure genome-wide expression in the capsule led to the identification of biochemical pathways altered during the progression of a flexion contracture [13]. These identified molecular changes represent potential drug targets and offer new avenues for the design of treatment for joint contracture. Additionally, increased knowledge and awareness will likely result from discussions between clinicians and scientists in the forum of international conferences focusing on joint contractures. For this, the involvement of professionals such as orthopaedic associations in combination with patients' associations will be instrumental to force research investments by grant-funding agencies. To my knowledge, the only international conference with a focus on joint contractures took place in Montreal in 2005 and was organized by our group at the Bone and Joint Research Laboratory. It is now time for an update.

In order to determine which kinds of contractures are more responsive to treatment, and what constitutes a clinically important improvement, clinical studies will need to examine the

initiation, progression, and severity of the joint contracture, along with the causal factors and quantitative measures of ROM. Providing measures of ROM in degrees before, during, and after treatment will establish standards for treatments. The use of animal models to mimic the clinical presentation of joint contractures will be instrumental to furthering our knowledge on the subject. The selection of relevant controls will be essential in order to draw conclusions with clinical relevance. Intense basic research aimed at elucidating the molecular mechanisms involved will serve at identifying targeted strategies for treatment.

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