Arthrogenic Muscle Inhibition: 20 Years On

Arthrogenic muscle inhibition (AMI) was a phrase that was seldom used 20 years ago, and it certainly had little real impact on clinical decision making and practice. Muscle dysfunction directly stemming from a change in joint sensory information and its integration in the central nervous system was a poorly understood phenomenon. After a couple decades of work in this area, AMI is a regularly used phrase in research circles as well as clinical practice. It is better understood as a consequence of every joint injury, and it has become a part of the theoretical foundation in understanding joint reinjury and joint disease. In the last 22 years, AMI has appeared in over 2800 peer-reviewed publications as we seek to better understand the mechanisms, consequences, and possible interventions. There is still a large mountain to climb as we seek to understand these important factors. In 2000,1 we proposed that AMI plays a central role in propagating joint injury. Twenty-two years of evidence suggests that is does, indeed, play an important role in impeding joint injury rehabilitation, changing long-term motor strategies and loading, and altering joint tissues.

The 2000 review, Arthrogenic Muscle Inhibition: A Limiting Factor in Joint Injury Rehabilitation,1 provided a theoretical framework for possible mechanisms by which joint injury produced muscle dysfunction. It also offered some ideas for its consequences and potential interventions. We have learned a great deal since those educated guesses were made. In this special section, we hope to provide a summary of what we have learned: an update on the mechanisms that drive AMI,2 how joint pain influences and complicates AMI,3 the relationship of Gamma loop dysfunction to AMI,4 the AMI patterns associated with specific joint injuries,5-7 and a clinical perspective of the implications of AMI.8,9 We will also introduce a few original research pieces with AMI as the central theme. It is extremely satisfying to see what we have learned, but it is also sobering to realize how much work there is still yet to do. It was the physicist, John Archibald Wheeler, who wisely stated, “We live on an island surrounded by a sea of ignorance. As our island of knowledge grows, so does the shore of our ignorance.”

Although much has been learned about AMI in the last 20-plus years, there is much more to learn. If we are to make a real difference in clinical practice and patient outcomes, we still have a lot of work to do. A few examples of areas where we need more data include the following.

Although much has been learned about the underlying physiology of AMI, the picture of how it changes over time is not settled. More information about the natural history of AMI and its transition into muscle activation failure is needed.

Clinical tools to measure AMI must be developed. The measures used in laboratory studies thus far are difficult to obtain, require substantial training to measure correctly, are not very portable, and may be cost prohibitive. A simple tool that utilizes wearable technologies and/or already available clinical tools would be ideal to help clinicians focus interventions in the correct area.

More information about the effects of early interventions for prevention of long-term dysfunction is needed. The plethora of outstanding work examining the long-term effects of inhibition on brain activity has evolved because of the long-standing deficits caused by unresolved AMI. If early interventions removed the factors causing long-term deficits, the need for these longer term interventions could become obsolete.

More information about how current early exercise regimens encourage compensatory changes will help focus interventions and allow the necessary changes to clinical practice to obtain optimal outcomes. Are we restoring normal recruitment and activation patterns in early therapeutic exercise, or are we creating compensatory strategies? Are the compensatory strategies problematic or beneficial to the patient? The answers to these questions are key to how we approach intervention strategies.

The development of more and better intervention tools/strategies is essential to increase clinical implementation and improve outcomes.

Our hope is that 20 years from now, we will be able to erase the long-term consequences of AMI and improve the health and well-being of individuals who suffer from joint injury.

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References


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