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Letter to the Editor

Management of intrinsic minus posture in a patient with poststroke spasticity: The role of nerve block

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The intrinsic minus hand is a hand deformity characterized by metacarpophalangeal joint hyperextension with proximal interphalangeal joint and distal interphalangeal joint flexion resulting from an imbalance between strong extrinsic muscles (extensor digitorum communis, flexor digitorum profundus, and flexor digitorum superficialis) and weak intrinsic muscles.[1] In patients with poststroke spasticity, muscle hypertonia often presents in various common postural patterns.[2] Although it is not very common, the intrinsic minus hand can present in a stroke.[3] In a patient with poststroke spasticity, the intrinsic minus hand can be due to either contracture or muscle overactivity. Spastic postures can sometimes be associated with a deformity that can be fully corrected, which means that the affected joint can be moved passively or actively throughout its entire range. Typically, slow-passive movements allow for this correction, while fast movements can lead to a temporary catch in the joint, making movement less smooth and possibly resulting in a distinct endpoint. At the other end of the spectrum is musculotendinous retraction, where the muscle cannot be further lengthened to achieve full range of motion (ROM). Although the term "contracture" is used for this second state, physical examination alone may not definitively determine whether a joint is fully correctable or not. Spastic muscle overactivity can also sometimes prevent the muscle from reaching its full length.[4] In such cases, nerve blocks may be beneficial.

Nerve blocks in spasticity management can be deemed "a 'dying art' that merits revival."[5] Nerve blocks can be categorized into three main categories: diagnostic, prognostic, and therapeutic. Diagnostic nerve blocks with anesthetics are useful for assessing various aspects. The diagnostic nerve blocks help determine whether a true muscle contracture is present rather than a deformity that can be corrected. They also allow the strength of antagonist muscles to be assessed in cases where the ROM increases due to muscle paralysis and cessation of spastic muscle overactivity.[6] If no improvements are observed with diagnostic nerve blocks, it is improbable that a more permanent procedure (e.g., botulinum toxin, phenol block, cryoneurolysis, and surgical neurotomy) would yield any benefits.[6]

A 72-year-old patient with poststroke spasticity with an intrinsic minus hand who complained about being unable to wear a resting splint, use their hand in daily activities, and move their fingers was evaluated for spasticity management (Figure 1a). A General Electric LogiqP5 ultrasound device (GE Healthcare, Milwaukee, Wisconsin, USA) with a high-frequency linear transducer (7-12 MHz) and 23-gauge Sterican 60-mm (B. Braun Medical Inc., Melsungen, Germany) needle were used for all blocks. First, an ultrasound-

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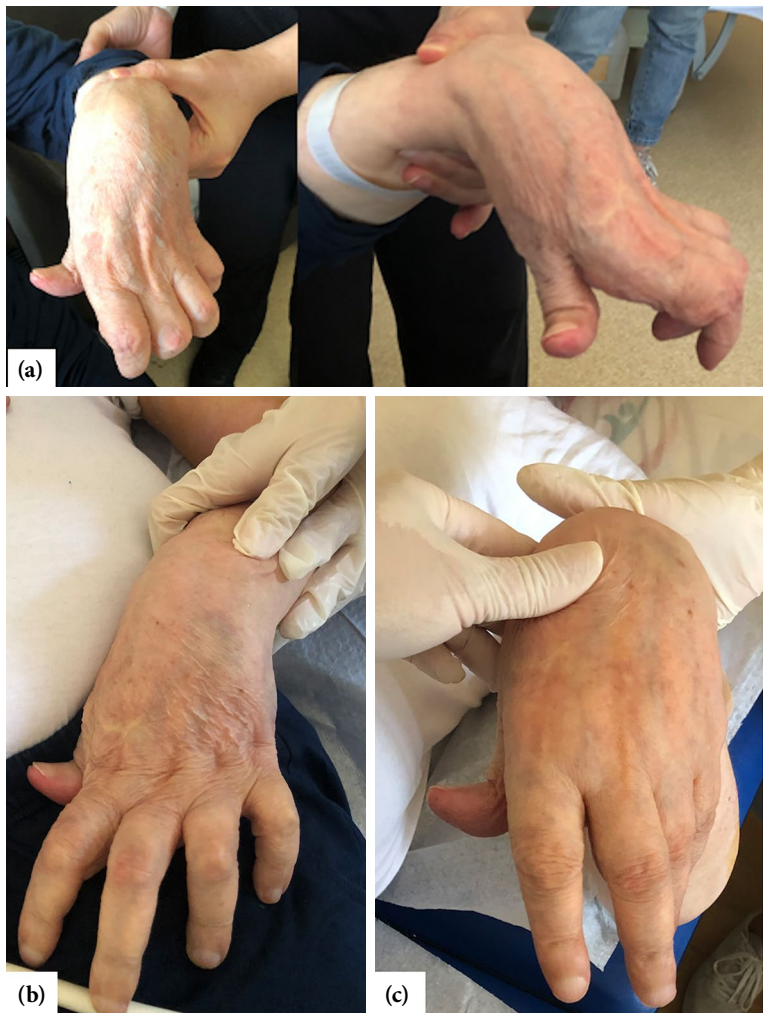

Figure 1. **(a)** Intrinsic minus hand deformity appearance before diagnostic nerve blocks. **(b)** Appearance of fingers after median nerve block at the forearm level. **(c)** Appearance of fingers after radial nerve block at the Arcade of Frohse level.

guided distal median nerve block was performed with 2 mL of 2% lidocaine below the elbow to affect the flexor digitorum superficialis and profundus. After the block, complete resolution of finger flexor spasticity and full ROM in the extensor direction in the fingers was achieved (Figure 1b). Subsequently, diagnostic nerve block for the radial nerve was performed with 2 mL of 2% lidocaine at the arcade of Frohse level, and it was observed that finger extensor spasticity was resolved; full ROM was achieved in the flexor direction of the fingers (Figure 1c). In addition, the patient was able to start voluntary finger extension and flexion. Although a more permanent chemoneurolysis intervention was initially considered due to the gain of full ROM and selective motor control, a botulinum toxin application was selected instead of phenol nerve block to avoid dysesthesia symptoms that may result from phenol nerve block since the radial and median nerves are both mixed motor and sensory nerves. Accordingly, botulinum toxin application to the extensor digitorum communis, flexor digitorum profundus, and flexor digitorum superficialis muscles was planned.

Neurolysis using nerve blocks with phenol or alcohol or cryoneurolysis and the use of diagnostic nerve blocks with anesthetics before these procedures to predict the outcome of spasticity management are niche practices. Although they are very feasible and helpful, they are not adequately implemented in most spasticity assessments.[6] With this case, we would like to draw attention to the marvelous role of diagnostic nerve blocks in poststroke spasticity management in

an unusual hand posture. In conclusion, diagnostic nerve blocks should be performed before further permanent chemoneurolysis procedures to avoid unsuccessful outcomes.

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