

## Mechanisms of neuroplasticity in chronic axonal polyneuropathy: therapeutic potential and functional constraints – case report

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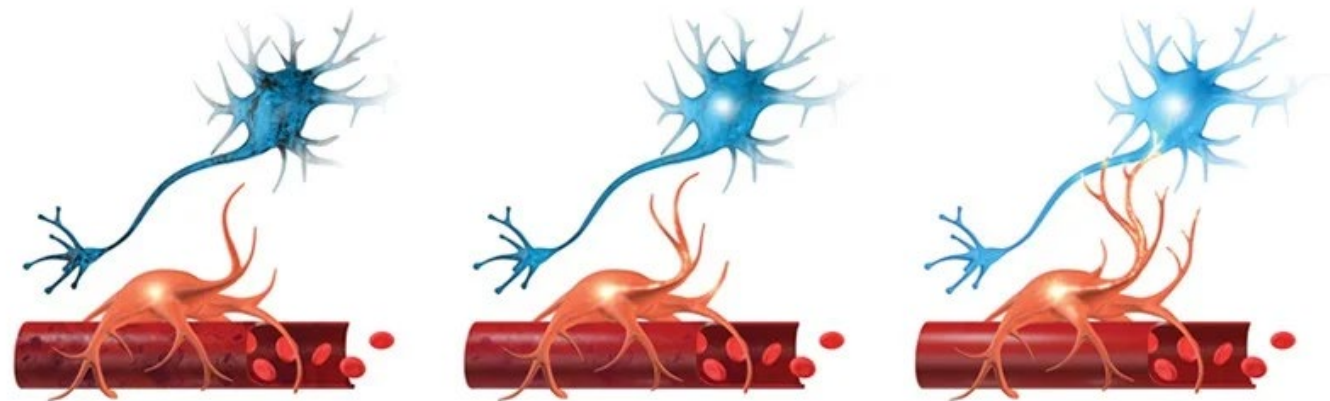
## Disclosure statement

**I have no actual or potential conflict of interest in relation to this program/presentation.**

## Introduction

Chronic axonal polyneuropathy represents one of the greatest challenges in rehabilitation medicine, characterized by distal axonal degeneration and, consequently, the loss of functional motor units. Although the etiology is peripheral, the impact of the disease is systemic, leading to a profound reorganization of the entire nervous system.

Neuroplasticity defines the nervous system's ability to reorganize itself structurally and functionally in response to injury. Medical rehabilitation can facilitate these mechanisms, contributing to the patient's functional recovery.



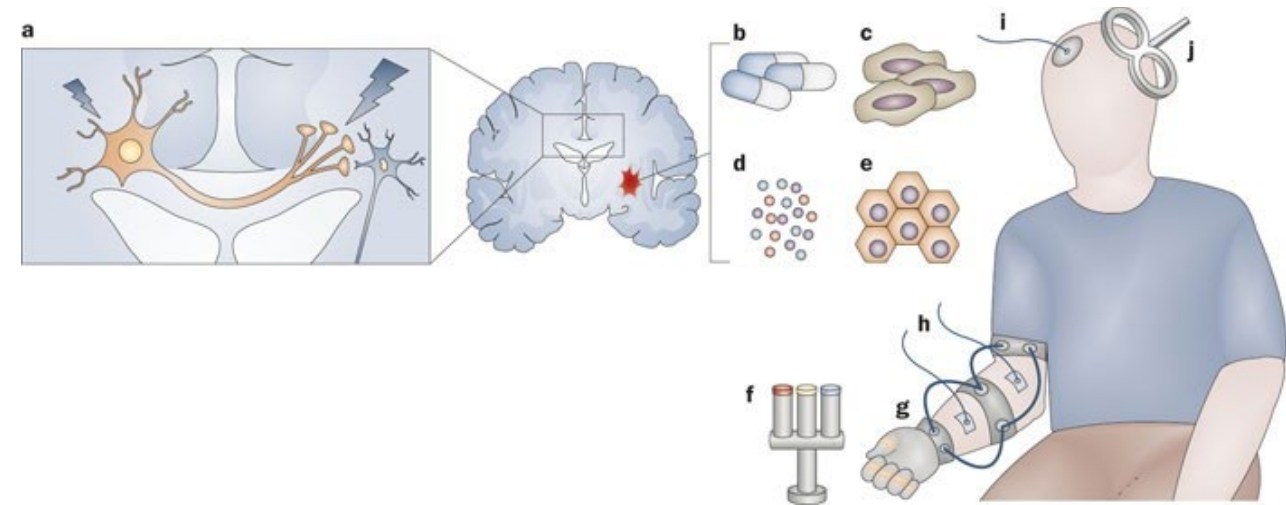
## Purpose of the presentation

- Highlighting the role of neuroplasticity in the recovery of patients with axonal polyneuropathy
- Assessing the effectiveness of rehabilitation interventions
- Identifying functional and therapeutic limitations

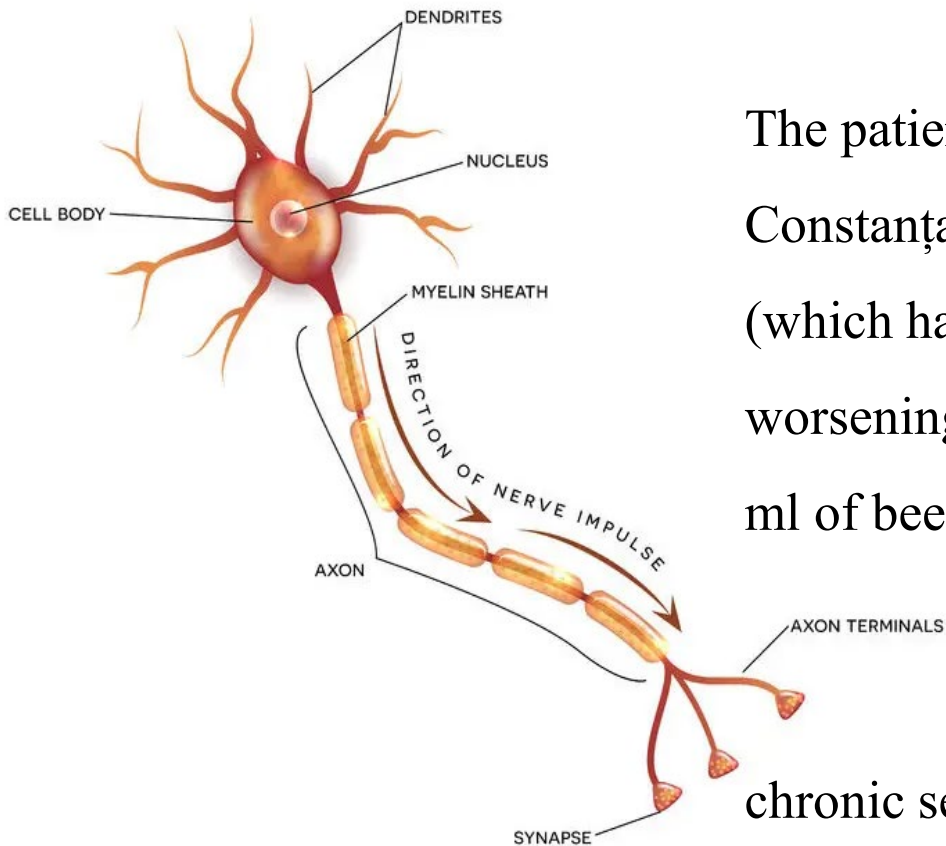


## Case presentation – demographic data and medical history

Patient NC, a 75-year-old woman, with a history of severe chronic sensory-motor axonal polyneuropathy (onset approximately 1 month ago), cerebrovascular disease, distal flaccid tetraparesis, stage III hypertension, a urinary tract infection caused by Klebsiella, a Bosnick renal cyst, uterine leiomyoma, and dyslipidemia. Given this multimorbid condition, the impact on recovery is negative.



## History of symptoms



The patient presented on December 25, 2025, at the Emergency Department of Constanța Hospital, complaining of bilateral paresthesia in the lower limbs (which had begun approximately 3 weeks earlier and was progressively worsening); she reported daily alcohol consumption (100 ml of spirits and 330 ml of beer)

During her hospitalization, she underwent a series of tests and investigations (CT, MRI, EMG) that led to a diagnosis of severe chronic sensory-motor axonal polyneuropathy, and she was transferred to the Techirghiol Balnear and Rehabilitation Sanatorium on January 15, 2026

## Clinical examination



- The patient is conscious, cooperative, oriented in time and space, and in good general condition;
- No signs of meningeal inflammation and no nystagmus
- No swallowing difficulties, no urinary incontinence
- Locomotor dysfunction; standing and walking are impossible; transfers performed with assistance from another person; dependence in activities of daily living
- Predominantly crural tetraparesis (left lower limb 1/5 MRC and right lower limb 3/5 MRC; left upper limb 3/5 MRC and right upper limb 4/5 MRC)
- Bilateral distal MS atrophy
- Bilateral Achilles tendon reflexes absent; patellar, biceps, and triceps reflexes present
- Bilateral symmetrical superficial plantar paresthesia and hypoesthesia

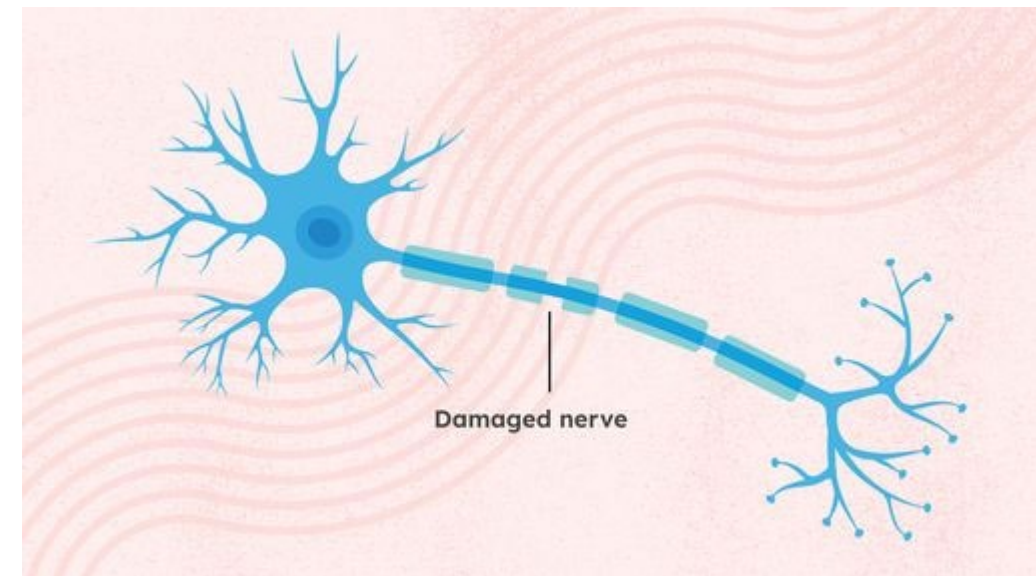
## Diagnosis

Based on the clinical presentation, neurological examination, and EMG results, the primary diagnosis was established:

severe chronic sensory-motor axonal polyneuropathy

In this case, the severity of axonal damage is the primary limiting factor, significantly reducing the capacity for nerve regeneration.

Consequently, functional recovery becomes largely dependent on compensatory mechanisms, particularly those of a central nature.



## Therapeutic goals



- Improved muscle blood flow
- Increased muscle strength
- Improved motor control
- Increased functional independence
- Reintegration into daily activities

## Therapeutic strategy

How can we stimulate neuroplasticity in a 75-year-old female patient with multiple vascular comorbidities and severe motor impairment?

### 👉 **Multisensory and motor stimulation**

kinesiotherapy

hydrokinetotherapy

### 👉 **Neuromuscular facilitation**

electrostimulation

diadynamic currents

### 👉 **Trophic and vascular modulation**

galvanization

massage



## Treatment plan

Medical rehabilitation was tailored to the patient's cardiovascular condition and included:

### ➤ **Hydrokinetic therapy:**

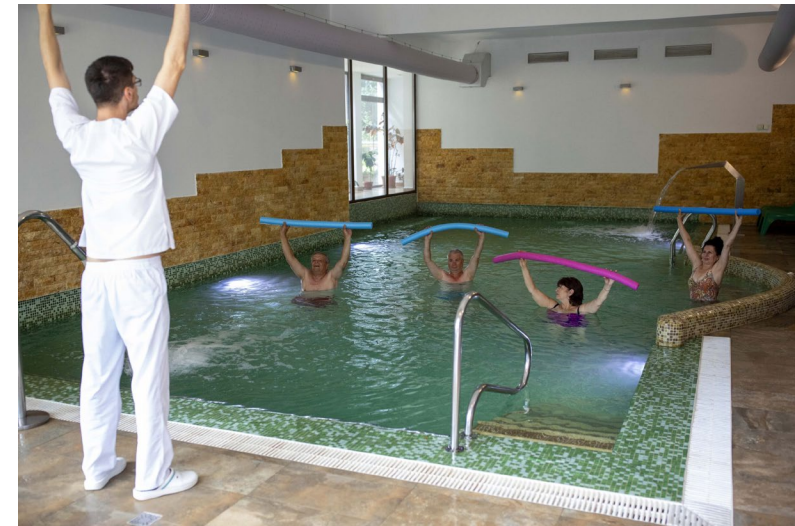
Effect on Plasticity: Provides the brain with positive motor feedback. Successfully performing a movement in water strengthens efferent motor pathways

### ➤ **Electrostimulation:**

Essential in axonal polyneuropathy to counteract denervation atrophy.

Mechanism: Replaces the absent or weak nerve impulse, forcing the contraction of muscle fibers.

Effect on Plasticity: Maintains the neuromuscular junction viable and sends retrograde signals to the anterior horn of the spinal cord



## Treatment plan

- **Galvanization and Diadynamic Current:** Modulation of the Environment
  - Galvanization improves local trophism through vasodilation and ionic balance.
  - Diadynamic currents act on the pain threshold and the autonomic nervous system.
  - Address vascular and inflammatory constraints.
  - Effect on Plasticity: Reducing sensory "noise," (pain, paresthesia), allows useful motor signals to be processed more clearly by the cortex.



## Treatment plan

### ➤ Therapeutic Massage:

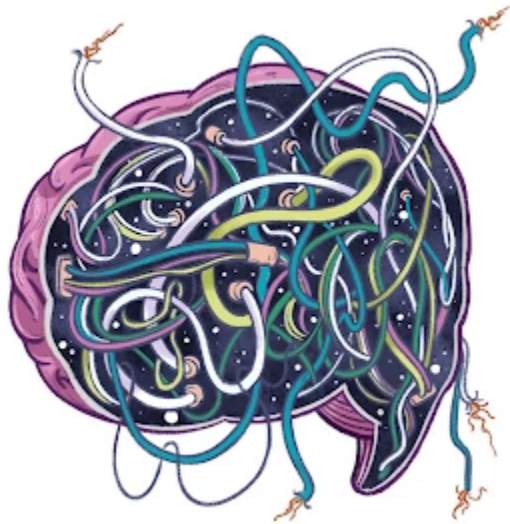
- to stimulate superficial and deep skin mechanoreceptors.

### ➤ Physical Therapy:

- Exercises based on neurofacilitation
- Functional electrostimulation (Bioness 300) – applied selectively to denervated muscle groups
- exercises to tone the upper and lower limbs,
- exercises to re-educate gait coordination using the R-force device
- grasping exercises using the Gloreha glove
- Functional vibrotactile stimulation - applied distally to the calves to reactivate receptors and improve proprioception



## Mechanisms of neuroplasticity



**CAN THE  
BRAIN BE  
REWIRED?**

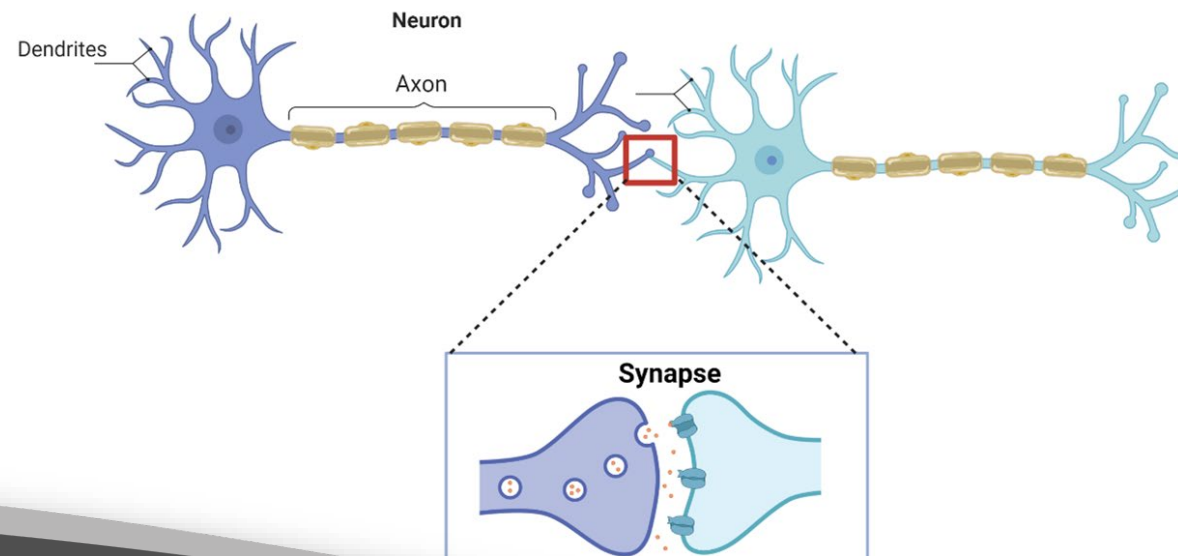
- From the perspective of neuroplasticity, these interventions can contribute to the adaptation of the central nervous system
- Repetition of movements supports the consolidation of synaptic connections, sensory stimulation facilitates the recalibration of cortical representations, and muscle activation leads to the recruitment of alternative neural circuits.
- Therefore, rehabilitation can be viewed not merely as a peripheral intervention, but as an active process of retraining the nervous system.

## Results

However, despite the implementation of a comprehensive treatment program, the patient's progress did not meet expectations.

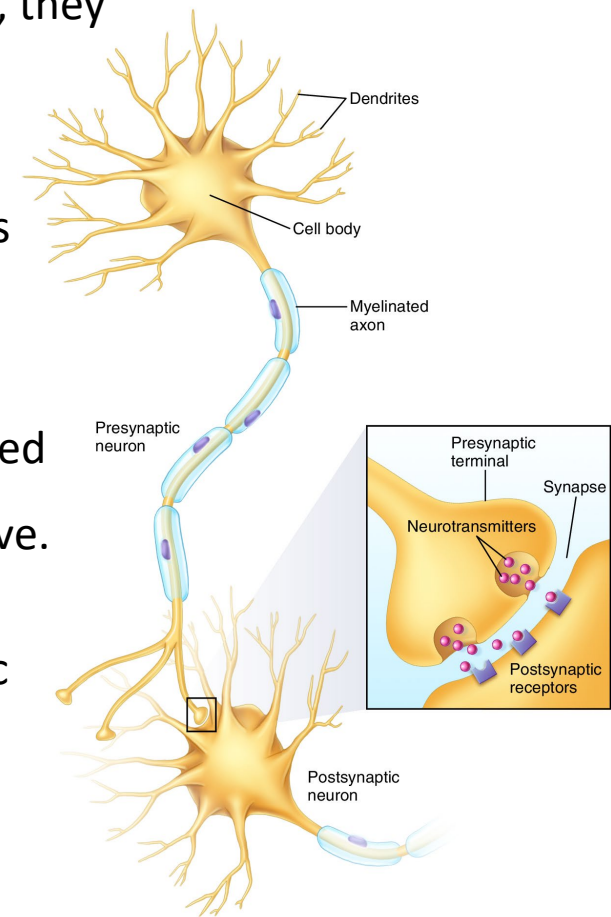
Only a slight improvement in muscle strength and motor control was observed, with no significant impact on her level of functional independence.

Progress was slow and limited; the Functional Independence Measure (FIM) score increased from 22 to 25



## Functional limitations

- This suboptimal outcome suggests that, although neuroplasticity mechanisms are present, they cannot fully compensate for the severity of structural damage.
- Particularly in geriatric and multimorbid settings, the nervous system's adaptive capacity is reduced, and the response to therapy becomes variable and limited.
- The main factors that have negatively influenced the course of the disease include advanced age, the severity of axonal degeneration, multiple comorbidities, and low functional reserve.
- These factors must be taken into account when establishing the prognosis and therapeutic goals.



## Conclusions

- Neuroplasticity is an essential compensatory mechanism in recovery
- Recovery in axonal polyneuropathy is not solely dependent on peripheral nerve regeneration
- The severity of axonal damage and comorbidities are limiting factors
- Multimodal therapy can transform structural limitations into functional adaptations
- The final outcome depends on the balance between the patient's biological potential and therapeutic intervention



The brain doesn't heal the nerve, but it learns to function despite it

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## Thank you for your attention!

