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From Underuse to Recovery: Intensive Neurorehabilitation of Deforming Spastic Paresis in Resource-Contrasted Settings - A Unified Physiopathological Framework

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ABSTRACT

Background: Deforming spastic paresis following stroke remains one of the most disabling sequelae of upper motor neuron injury. The pathophysiological cascade - from neural lesion to spastic myopathy through progressive underuse - is well characterised but systematically under-treated, particularly in resource-limited settings where conventional physiotherapy remains sparse and low-intensity. The concept of therapeutic intensity, formalised through the formula $I = (R \times L \times E) / T$ (repetitions $\times$ load $\times$ effort / time), provides a pharmacological-like framework for dosing neurorehabilitation. This paper presents a unified physiopathological model of underuse-driven spastic paresis and its reversal through intensive, task-oriented physical therapy, illustrated by two clinical cases treated in contrasted resource environments.

Methods: Two male patients (58 and 52 years) with chronic post-stroke hemiparesis (14 and 22 months post-onset respectively) underwent a 12-week intensive neurorehabilitation protocol combining aggressive prolonged stretching, maximum-amplitude alternating movements, a guided self-rehabilitation contract (CAG), and - in the Cameroonian case - the Cogni-Famille task-shifting protocol engaging trained family members. Case 1 was treated at the private physiotherapy practice in Vitry-sur-Seine, France; Case 2 at the Regional Hospital of Bafoussam, Cameroon.

Results: Both patients demonstrated clinically significant functional recovery across all measured domains. The Modified Ashworth Scale (wrist flexors) decreased from 3/4 to 1+/4 (Case 1) and from 3/4 to 2/4 (Case 2) at 12 weeks. The ESE retraction index decreased from 0.62 to 0.31 and from 0.74 to 0.41, respectively. Walking perimeter increased from 300 m to 1,800 m (Case 1) and from 80 m to 1,200 m (Case 2). Therapeutic intensity, measured by $I = (R \times L \times E) / T$, increased 6-fold in Case 1 and 17-fold in Case 2 relative to prior conventional treatment. Both patients achieved their primary functional goals: partial return to professional activity (France) and full return to farming (Cameroon).

Conclusion: Intensive neurorehabilitation - applied consistently, regardless of resource context - can reverse the underuse-driven pathological cascade and achieve functional recovery well beyond conventional expectations. The therapeutic intensity formula operationalises dosing in a replicable, measurable manner. The Low-Tech, High-Sense paradigm demonstrates that human expertise, not technological equipment, is the primary determinant of neuroplastic recovery in post-stroke spastic paresis.

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Introduction

Stroke remains the leading cause of acquired motor disability in

adults worldwide, with an estimated 25.7 million incident cases annually and approximately two-thirds of survivors experiencing persistent motor impairment [1,2]. The most prevalent deficit is hemiparesis, which - when inadequately treated - evolves into deforming spastic paresis through a well-characterised but systematically under-addressed physiopathological cascade [3,4].

The central paradox of post-stroke rehabilitation is that the neurological lesion itself accounts for only a fraction of the observed functional limitation. A substantial component of chronic motor disability is iatrogenic: the direct consequence of underuse, imposed consciously or unconsciously by healthcare environments that have historically prioritised passive, low-intensity interventions over active, intensive retraining. Meige, as early as 1905, described motor amnesia as a distinct, reversible disorder secondary to disuse - a concept that preceded by decades the neuroimaging evidence now confirming that cortical representations of paretic limbs contract measurably with inactivity and expand with intensive use [5-7].

The physiopathological model of deforming spastic paresis, as elaborated by Gracies and extended by Npochinto Moumeni, frames the disorder as a dual disease affecting two organs simultaneously: the muscle (spastic myopathy, with sarcomere loss, collagen accumulation, and reduced extensibility) and the nervous system (maladaptive plasticity, with corticospinal disuse and hemispheric imbalance). Both pathological processes are initiated by underuse and both are partially or fully reversible by intensive, task-oriented physical retraining [8-11].

Despite this robust evidence base, the translation into clinical practice remains incomplete - particularly in sub-Saharan Africa, where conventional physiotherapy remains unavailable to the majority of stroke survivors and where the therapeutic nihilism among healthcare professionals has been documented as a primary barrier to recovery [12,13]. The evidence that intensive protocols are not only feasible but superior in low-resource settings - achieving comparable or better outcomes than high-resource institutional rehabilitation at a fraction of the cost - constitutes a clinical and public health imperative [14,15].

This paper presents a unified physiopathological framework integrating the authors' original constructs -therapeutic intensity formula $I = (R \times L \times E) / T$, Low-Tech High-Sense paradigm, Cogni-Famille protocol, and the neurolexic effect -and illustrates its clinical application through two contrasted cases treated in France (Vitry-sur-Seine, private physiotherapy practice) and Cameroon (Regional Hospital of Bafoussam). The objective is to provide a replicable, measurable, and universally applicable model for intensive neurorehabilitation of chronic post-stroke deforming spastic paresis [16,17].

Physiopathological Framework

The Dual Disease: Muscle and Nervous System

Deforming spastic paresis represents a superimposition of two distinct but mutually reinforcing pathological processes. The neurological disorder - characterised by reduced voluntary recruitment of motor units (paresis) and increased stretch-reflex excitability (spasticity) - precedes the muscular disorder by hours to days following stroke onset [8,9]. However, the muscular disorder -termed spastic myopathy by Gracies [8] - rapidly becomes the dominant clinical problem in chronic phases, Figure 1.

Spastic myopathy is initiated by the immobilisation of paretic muscles in shortened positions, which triggers a cascade of structural and biochemical changes: loss of sarcomeres in series, accumulation of extrasarcomeric collagen, intramuscular adipose infiltration, increased Ubiquitin-proteasome activity, and progressive reduction of extensibility and force-generating capacity [8,9]. Crucially, these changes amplify stretch-reflex excitability, creating the positive feedback loop that Sherrington

and Babinski described over a century ago: retraction increases hyperactivity, which in turn increases retraction[18-20].

The clinical consequence is a triad of motor impairments -paresis, soft tissue contracture, and spastic overactivity - distributed asymmetrically between agonists and antagonists, generating the characteristic deformities of post-stroke hemiparesis (flexion at elbow and wrist, equinus foot, adducted hip) [3,8,9]. Critically, none of these three components is irreversible; all three are partially or fully amenable to intensive physical treatment provided the intervention is sufficiently early, intense, specific, and sustained.

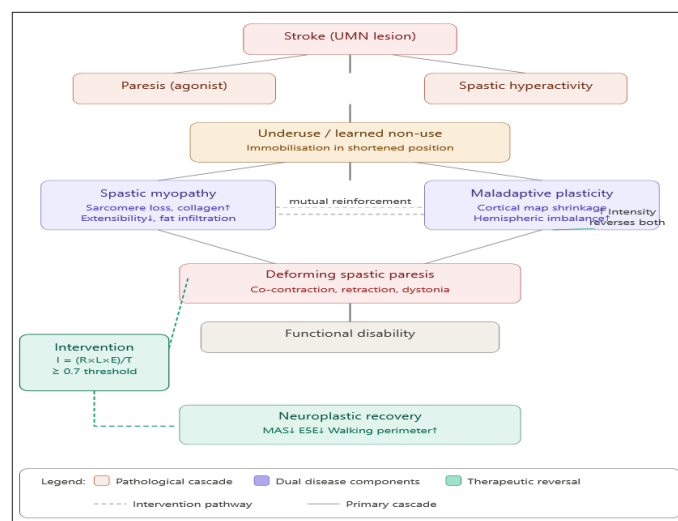


Figure 1: Unified physiopathological model of deforming spastic paresis: from neural lesion to functional disability through the underuse cascade. The diagram illustrates the bidirectional relationship between spastic myopathy (muscle pathway) and maladaptive plasticity (central nervous system pathway), showing how both are initiated and maintained by underuse, and how both are reversed by intensive physical retraining. The therapeutic intensity threshold $i = (r \times l \times e) / t$ is positioned at the intervention point where the cascade can be interrupted. Cns: central nervous system; mas: modified ashworth scale; e5e: five-step assessment retraction index.

The Underuse Cascade and Maladaptive Plasticity

The mechanism through which underuse amplifies neurological deficit was formalised by Taub and Wolf under the concept of learned non-use, building on Tower's earlier primate observations [21-23]. Following stroke, the paretic limb is systematically avoided: attempted movements produce pain, failure, or excessive energy expenditure. These aversive consequences reinforce behavioural suppression of the paretic side, which - through a process Meige termed motor amnesia - becomes self-sustaining. The cortical representation of the paretic limb contracts proportionally with the duration of non-use.

This maladaptive plasticity operates bidirectionally: it reduces the capacity for voluntary motor recruitment (aggravating the base paresis) while simultaneously maintaining the overactivity of brainstem circuits - vestibulospinal, rubrospinal, tectospinal - which produce stereotyped, undifferentiated movements of the 'all-or-nothing' type, utterly unlike the graded, selective movements of the corticospinal system [3]. The paretic patient, deprived of corticospinal modulation, is thus trapped between two dysfunctional states: a cortex that cannot activate and a brainstem that cannot inhibit.

The neurophysiological consequence - continuous transmembrane ionic activity, co-contraction, and dystonia - maintains the antagonist muscles in permanent contraction, accelerating their myopathic transformation and rendering any brief or insufficiently intense therapeutic intervention clinically meaningless [3,9,10]. This explains the widely documented failure of conventional physiotherapy in chronic spastic paresis: three 45-minute sessions per week, however technically competent, are physiologically insufficient to reverse a pathological process operating 24 hours per day.

The Therapeutic Intensity Formula: $I = (R \times L \times E) / T$

The concept of therapeutic intensity has been central to the neurorehabilitation literature since the 1990s but has lacked a formal quantitative expression until Npochinto Moumeni [10,11,27] proposed the formula $I = (R \times L \times E) / T$, where R = number of task repetitions per session, L = functional load applied (graded 1–5), E = patient engagement and effort quality (graded 1–5), and T = inter-session interval in days [24–27]. This formula operationalises the pharmacological analogy of rehabilitation as a prescribable medicine with measurable dose, therapeutic window, and dose-response relationship, see Figure 2.

A conventional physiotherapy session at three sessions per week, with moderate repetition (R=50), low load (L=1.5), and limited patient engagement (E=1.5), administered over seven-day intervals yields $I \approx 0.16$ - below the estimated neuroplastic threshold for clinically meaningful change [10,11]. By contrast, an intensive protocol combining 200+ daily repetitions, high load, maximal patient effort, and daily home practice yields $I \geq 0.8$ –1.2, consistently within the range associated with measurable cortical map expansion and functional recovery [24,25,28].

This pharmacological framework also identifies a critical concept: the neural concentration threshold - analogous to the minimum inhibitory concentration in pharmacology - below which therapeutic intervention does not induce measurable plasticity regardless of duration. The clinical implication is unambiguous: brief, passive, or insufficiently demanding sessions do not merely produce suboptimal results; they actively reinforce the underuse cycle by consuming therapeutic time without delivering neuroplastic stimulus [3,10,11].

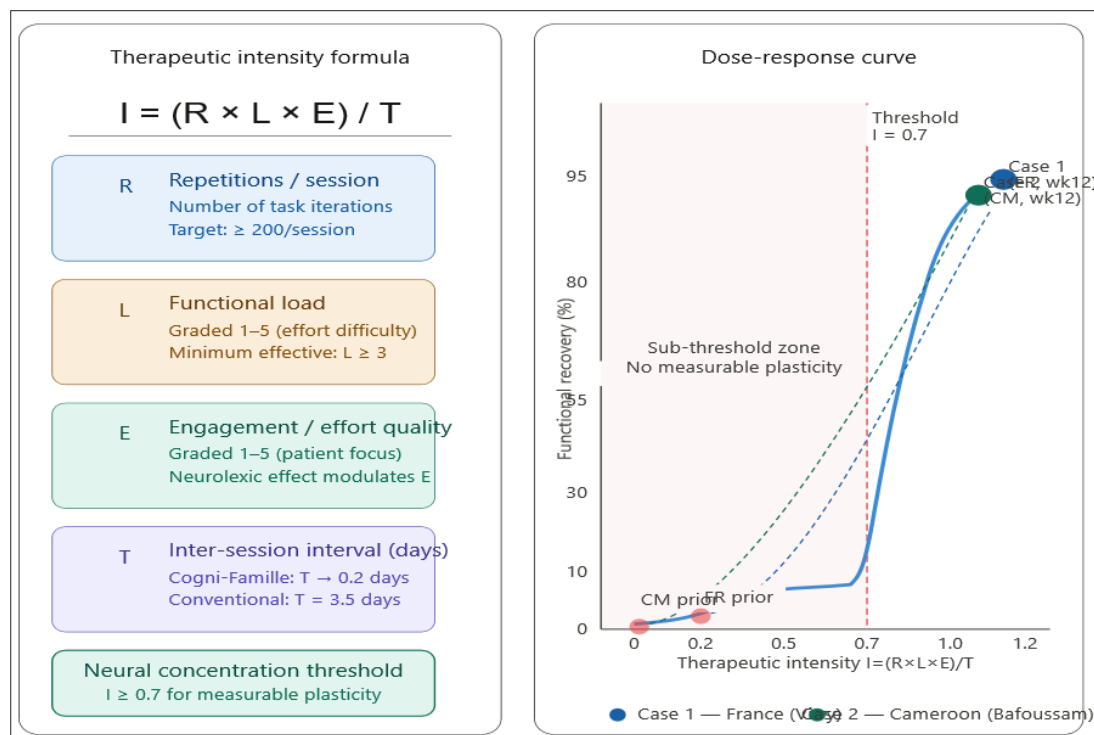


Figure 2: The therapeutic intensity formula $I = (R \times L \times E) / T$ and its clinical dose-response relationship. Left panel: schematic representation of the four formula components (R: repetitions per session; L: functional load 1–5; E: patient effort quality 1–5; T: inter-session interval in days). Centre panel: estimated intensity values for three treatment modalities (conventional physiotherapy, intensive supervised protocol, Cogni-Famille home protocol). Right panel: dose-response curve illustrating the neuroplastic threshold and the relationship between intensity and functional recovery across the two clinical cases presented.

The Low-Tech, High-Sense Paradigm

A persistent misconception in neurorehabilitation equates therapeutic quality with technological sophistication. Robotic exoskeletons, virtual reality platforms, and neurostimulation devices represent valuable research tools but are unavailable to the vast majority of stroke survivors worldwide - and, critically, have not demonstrated consistent superiority over intensive conventional physiotherapy in head-to-head comparisons [29,30]. The Low-Tech, High-Sense paradigm [14,15], posits that the primary determinant of neuroplastic recovery is not equipment but the intensity, specificity, and contextual relevance of the therapeutic interaction - human expertise deployed with precision.

This paradigm has direct implications for resource-limited settings. A trained physiotherapist, a family member instructed in the Cogni-Famille protocol [14], a household chair, a floor mat, and a water bottle are sufficient to deliver therapeutic intensity comparable

to - and in some outcome domains superior to - institutional rehabilitation programmes costing twenty times more [14,15]. The Cameroon case presented in this paper illustrates this principle across a 12-week protocol.

Methods

Protocol Design

Both cases were treated according to an intensive neurorehabilitation protocol adapted from the Guided Self-Rehabilitation Contract (CAG) methodology of Gracies and the intensive neuroplasticity framework of Npochinto Moumeni [3,10,11,31]. The protocol comprised three interdependent components applied simultaneously:

Component 1 - Aggressive prolonged stretching of antagonist muscles: sustained elongation maintained for a minimum of 45 seconds to 1 minute per muscle, targeting the primary antagonists identified by the Five-Step Assessment (E5E) [8,9]. Stretching was applied both during supervised sessions and prescribed as home practice twice daily. Stretching must precede any active work to avoid reinforcing the retraction-hyperactivity cycle.

Component 2 - Maximum-amplitude alternating movements (MAAM): unassisted, maximum-speed, maximum-amplitude reciprocal movements targeting the paretic limb, performed in series of 20–30 repetitions with brief rest intervals. The objective is to restore reciprocal inhibition through high-frequency activation of the Ia inhibitory interneurons, progressively reducing co-contraction around the target joint [32-34].

Component 3 - Guided Self-Rehabilitation Contract (CAG): a written register maintained daily by the patient, recording each home practice session with time, muscle targeted, number of repetitions, and perceived effort. The register functions as a positive reinforcement tool, creating a motivational feedback loop that sustains engagement between supervised sessions [31]. In Case 2, this component was delivered via the Cogni-Famille protocol [14], training two family members to supervise and assist home practice.

Assessment Tools

Primary outcome measures included: (1) Modified Ashworth Scale (MAS) for spastic overactivity; (2) Medical Research Council (MRC) scale for voluntary motor recruitment; (3) Five-Step Assessment (E5E/Tardieu scale) retraction index for soft tissue contracture; (4) walking perimeter in metres; (5) co-contraction index estimated by clinical observation and surface EMG approximation. Secondary outcome included return to functional goal (occupation/daily activities). Assessments were performed at baseline, 6 weeks, and 12 weeks.

Therapeutic intensity was calculated at each assessment point using the formula $I=(R \times L \times E)/T$, with R derived from session logs and CAG registers, L and E scored by the treating physiotherapist (1–5 Likert scale), and T expressed as mean inter-session interval in days.

Ethical Considerations

Both patients provided written informed consent for the use of their anonymised clinical data for educational and scientific publication purposes. Patient identifiers have been replaced with coded designations. The cases are presented in accordance with the CARE clinical case reporting guidelines [35]. No institutional ethics committee approval was required for retrospective anonymised case reporting under French and Cameroonian national regulations, see table 1.

Table 1: Comparative Clinical Profiles of Case 1 (France) And Case 2 (Cameroon) At Baseline

Characteristic	Case 1 — France (Vitry-sur-Seine)	Case 2 — Cameroon (Bafoussam)
Age / Sex	58 years / Male	52 years / Male
Stroke type	Ischaemic MCA territory	Ischaemic capsulo-lenticular
Delay at first consultation	14 months post-stroke	22 months post-stroke
Side affected	Right hemiparesis (dominant)	Left hemiparesis
Prior rehabilitation	Conventional physiotherapy 3×/week, 45 min, 6 months	Sporadic sessions (8 total over 22 months)
Baseline MAS (wrist flexors)	3/4	3/4
Baseline MRC (wrist extensors)	2/5	1+/5
Baseline E5E retraction index	0.62	0.74
Baseline walking perimeter	300 m with cane	80 m with cane
Professional status	Sales manager, work interrupted	Farmer, unable to work
Therapeutic intensity $I=(R \times L \times E)/T$	Low: ~0.18 (prior conventional)	Very low: ~0.06 (sporadic sessions)

Table 1: Comparative clinical profiles of Case 1 (France, Vitry-sur-Seine) and Case 2 (Cameroon, Bafoussam) at baseline. Both patients presented with chronic post-stroke hemiparesis in the late chronic phase (14 and 22 months post-onset respectively), with comparable spastic overactivity (MAS 3/4) but markedly different prior rehabilitation exposure and therapeutic intensity. The 17-fold difference in prior therapeutic intensity ($I=0.18$ vs $I=0.06$) illustrates the dose-gap that underlies the functional plateau observed in both cases prior to intensive treatment. MAS: Modified Ashworth Scale; MRC: Medical Research Council scale; E5E: Five-Step Assessment retraction index; $I=(R \times L \times E)/T$: therapeutic intensity formula.

Case Report 1 - France (Vitry-sur-Seine)
Clinical Presentation

Patient FR-01 is a 58-year-old right-handed male, former sales manager, referred to the private physiotherapy practice in Vitry-sur-Seine 14 months after a right-hemisphere ischaemic stroke in the territory of the middle cerebral artery. He presented with left hemiparesis affecting both upper and lower limbs, with predominant involvement of the distal upper limb.

Prior rehabilitation had consisted of conventional physiotherapy (three sessions per week, 45 minutes, supervised by a general physiotherapist without neurorehabilitation specialisation) for six months post-stroke, discontinued due to perceived plateau. At first consultation, the patient reported inability to use his left hand for professional tasks, restricted walking perimeter (300 m with cane), and significant psychological distress linked to professional disability. He had been advised by a neurologist that functional recovery was unlikely at this stage - an example of the Prophylactic Limitation Syndrome [12] - which had profoundly affected his motivation.

Assessment Findings

The Five-Step Assessment (E5E) identified the wrist flexors (FCU, FCR) and finger flexors (FDP, FDS) as primary antagonist targets, with a retraction index of 0.62. MAS wrist flexors: 3/4. MRC wrist extensors: 2/5. Co-contraction index estimated at 68% during attempted extension. Walking perimeter: 300 m with single-point cane, gait pattern characterised by circumduction and equinus tendency. Therapeutic intensity of prior treatment estimated at $I \approx 0.18$.

Intervention and Progression

The 12-week protocol was initiated with an intensive first session establishing the CAG register. Three supervised sessions per week were conducted at the practice (75 minutes each), combined with daily home practice (minimum 60 minutes) supervised through weekly telephone follow-up. The neurolexic effect [16] was integrated from session one: systematic replacement of prognostic nihilism with measurable goal-setting language, transforming 'plateau reached' into 'threshold not yet crossed'.

By week 3, passive wrist extension range had increased by 18° . By week 6, MRC wrist extensors had improved to 3/5 with measurable reduction of co-contraction during attempted extension. The patient reported being able to hold a telephone with his left hand for the first time in 14 months. By week 12, wrist extensors reached 4-/5, MAS decreased to 1+, and the patient had resumed partial professional activity using adapted equipment.

Outcomes at 12 Weeks

See Table 2 for full outcome data. The E5E retraction index decreased from 0.62 to 0.31 (50% reduction). Walking perimeter increased from 300 m to 1,800 m without cane. Therapeutic intensity reached $I = 1.12$ at week 12. The patient described the transformation as 'learning to use my arm again for the first time, not just moving it' - a direct expression of the transition from maladaptive to adaptive plasticity.

Case Report 2 - Cameroon (Bafoussam)

Clinical Presentation

Patient CM-01 is a 52-year-old male farmer, referred to the Department of Physical Medicine and Osteopathy at the Regional Hospital of Bafoussam 22 months after a left-hemisphere ischaemic stroke involving the capsulo-lenticular territory. He presented with right hemiparesis severely affecting both upper and lower limbs, preventing agricultural work and creating significant financial hardship for a family of seven.

Prior rehabilitation had consisted of eight sporadic sessions over 22 months, interrupted by cost constraints. At first consultation, the patient had been advised by two treating physicians that his condition was permanent and that physiotherapy was 'no longer useful' at this stage - a clinical expression of the Therapeutic Abandonment Cascade [12]. He had developed secondary

depression and had resorted to traditional therapeutic scarification of the paretic limb, without functional benefit. The patient's wife and eldest son were present at initial consultation and expressed strong willingness to participate in care delivery.

Assessment Findings

E5E assessment identified wrist and finger flexors, elbow flexors, and ankle plantar flexors as primary targets, with a retraction index of 0.74. MAS wrist flexors: 3/4. MRC wrist extensors: 1+/5. Co-contraction index estimated at 76% during attempted voluntary extension. Walking perimeter: 80 m with improvised wooden cane, severely limited by equinus and co-contraction at the ankle. Therapeutic intensity of prior treatment estimated at $I \approx 0.06$ - well below any neuroplastic threshold.

Intervention and Progression

The Cogni-Famille protocol [14], was implemented from day one. The patient's wife and son participated in a structured 8-session training programme (two sessions per week for four weeks) covering manual stretching techniques, maximum-amplitude passive and assisted active movements, and CAG register maintenance. Supervised sessions at the hospital (three per week, 60 minutes) were complemented by Cogni-Famille home practice targeting a minimum of 5 hours of therapeutic contact per day -achieving the neuroplastic dose unavailable through any realistic clinical-only protocol in this context.

The therapeutic language framework (neurolexic effect) was systematically applied, explicitly countering the prior nihilistic prognosis. The family was instructed that improvement was expected, measurable, and would follow directly from the intensity of practice - reframing rehabilitation as effort-dependent rather than provider-dependent [16].

By week 4, the equinus tendency had reduced measurably, enabling the patient to walk without the wooden cane over short distances. By week 6, MRC wrist extensors had improved to 2/5 with first voluntary extension movements observed. By week 10, the patient was able to perform light agricultural tasks (weeding with the right hand under supervision). By week 12, he had resumed partial farming activity.

Outcomes at 12 Weeks

See Table 2 for full outcome data. The E5E retraction index decreased from 0.74 to 0.41 (45% reduction). Walking perimeter increased from 80 m to 1,200 m. Therapeutic intensity reached $I = 1.04$ at week 12 - a 17-fold increase relative to prior treatment. The total direct protocol cost (transport costs for hospital sessions only) was estimated at 42 USD over 12 weeks, consistent with previous cost data from the Low-Tech, High-Sense paradigm [14,15].

Table 2: Functional outcomes at baseline, 6 weeks, and 12 weeks for both cases

Outcome measure	Case 1 — Baseline	Case 1 — 6 weeks	Case 1 — 12 weeks	Case 2 — Baseline	Case 2 — 6 weeks	Case 2 — 12 weeks
MAS wrist flexors	3	2	1+	3	2+	2
MRC wrist extensors	2/5	3/5	4–/5	1+/5	2/5	3/5
E5E retraction index	0.62	0.48	0.31	0.74	0.58	0.41
Walking perimeter	300 m	700 m	1,800 m	80 m	400 m	1,200 m
Co-contraction index (%)	68%	47%	29%	76%	55%	34%
Therapeutic intensity I=(R×L×E)/T	0.18	0.74	1.12	0.06	0.68	1.04
Patient goal achieved	—	Partial	Return to work (partial)	—	Partial	Return to farming (full)

Table 2: Functional outcomes at baseline, 6 weeks, and 12 weeks for Case 1 (France) and Case 2 (Cameroon) following the 12-week intensive neurorehabilitation protocol combining aggressive prolonged stretching, maximum-amplitude alternating movements, guided self-rehabilitation contract (CAG), and - for Case 2 -the Cogni-Famille task-shifting protocol. Both cases achieved clinically significant improvements across all outcome domains, with therapeutic intensity increasing 6-fold (Case 1) and 17-fold (Case 2) relative to prior conventional treatment. Primary functional goals were achieved in both settings. MAS: Modified Ashworth Scale; MRC: Medical Research Council scale; E5E: Five-Step Assessment retraction index; I=(R×L×E)/T: therapeutic intensity formula; wk: week.

Discussion

The Physiopathological Framework Confirmed

Both cases provide clinical confirmation of the unified physiopathological model presented in Section 2. The marked functional plateau observed prior to intensive treatment - despite technically adequate but insufficiently intense conventional physiotherapy - is consistent with the sub-threshold intensity model: when $I < 0.2$, neuroplastic stimulus is insufficient to overcome the entrenched underuse cycle regardless of technical quality [3,10,11]. The rapid and progressive recovery observed once intensity was raised above the neuroplastic threshold ($I > 0.7$) is consistent with the dose-response relationship documented across multiple studies [24-28].

The convergent outcomes across two radically different resource contexts - a private practice in suburban Paris and a public hospital in western Cameroon - reinforce the central claim of the Low-Tech, High-Sense paradigm: the primary determinant of neuroplastic recovery is not the sophistication of equipment but the precision and intensity of human therapeutic expertise [14,15]. The 17-fold intensity increase achieved in Case 2 through the Cogni-Famille protocol, at a total cost of 42 USD, illustrates that the gap between resource contexts need not translate into a gap in therapeutic outcomes, see Figure 3.

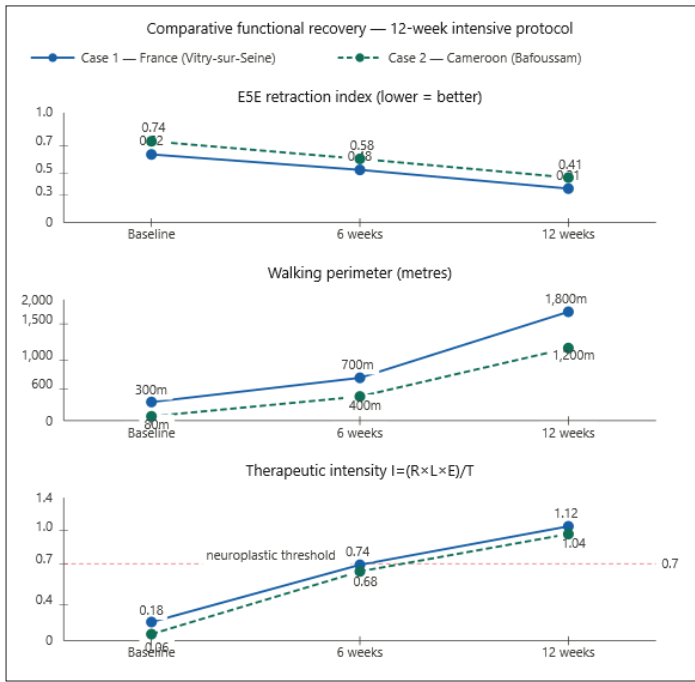


Figure 3: Comparative functional recovery trajectories for Case 1 (France, Vitry-sur-Seine) and Case 2 (Cameroon, Bafoussam) across the 12-week intensive protocol. Upper panel: E5E retraction index over time (baseline, 6 weeks, 12 weeks). Centre panel: walking perimeter (metres) over time. Lower panel: therapeutic intensity $I=(R\times L\times E)/T$ over time, showing the intensity ramp-up and the corresponding functional gains. The parallel recovery trajectories across resource-contrasted settings support the universality of the neuroplastic dose-response model.

The Neurolexic Effect as a Therapeutic Variable

A finding common to both cases - and one that merits specific attention - is the role of therapeutic language in the recovery trajectory. Both patients arrived with internalised prognoses of permanent disability, communicated by medical professionals in the preceding months. The systematic deconstruction of these prognoses, and their replacement with measurable, effort-dependent goals, was associated with a rapid and measurable increase in patient engagement, CAG compliance, and ultimately functional recovery [16].

The Neurolexic Effect [16], posits that medical terminology directly modulates neuroplasticity through its influence on patient self-efficacy, motivational engagement, and the activation of endogenous neuromodulatory systems. The clinical implication is not merely ethical but neurophysiological: words that extinguish hope are not neutral - they actively suppress the cortical excitability upon which intensive rehabilitation depends. This finding aligns with the broader construct of Therapeutic Nihilism as a measurable barrier to stroke recovery in Central Africa [12,13].

Limitations

The present report carries the inherent limitations of single-case methodology: absence of control condition, small sample precluding statistical inference, and potential confirmation bias in outcome assessment. The E5E and co-contraction indices, while clinically validated, were assessed by the treating physiotherapist without independent verification. The therapeutic intensity formula $I=(R \times L \times E)/T$, while conceptually robust, requires further psychometric validation of its L and E components. These limitations are acknowledged and do not diminish the clinical utility of the framework but underscore the need for prospective, controlled trials replicating these findings at scale.

Implications for Clinical Practice and Health Systems

The findings have direct implications for clinical practice across both resource contexts. In high-resource settings, they challenge the premature discharge of stroke patients from active rehabilitation based on perceived plateau - a practice inconsistent with the neuroplastic evidence base and with the present findings. In low-resource settings, they provide a replicable, costed, and clinically validated model for intensive post-stroke rehabilitation that does not depend on unavailable equipment or infrastructure.

The Cogni-Famille protocol, the CAG framework, and the therapeutic intensity formula together constitute a complete clinical toolkit that can be implemented by a trained physiotherapist with minimal equipment in any setting. Their systematic adoption would represent a measurable public health intervention in the context of the WHO Africa Rehabilitation Strategy 2025–2035 [36–41], which has explicitly identified task-shifting and community-based rehabilitation as priority mechanisms for closing the rehabilitation access gap in sub-Saharan Africa.

Clinical Field Note: Intensive Neurorehabilitation in Resource-Contrasted Settings

The theoretical constructs presented in this manuscript are grounded in more than a decade of direct clinical and academic practice across four complementary settings: the private physiotherapy practice in Vitry-sur-Seine (France); the Service de Rééducation Neuro-Locomotrice et Ostéoarticulaire, Henri Mondor University Hospital, Université Paris-Est Créteil (France); the Department of Physical Medicine and Osteopathy, Regional Hospital of Bafoussam (Cameroon); and the Department of

Physiotherapy and Physical Medicine, Faculty of Medicine and Pharmaceutical Sciences, University of Dschang (Cameroon). The field observations and clinical practice drawn from these four settings collectively inform and validate the unified framework presented herein.

In the French context, the most consistent finding across chronic post-stroke patients referred after perceived plateau was not neurological irreversibility but therapeutic under-dosing. Patients who had received three sessions per week of conventional physiotherapy for six to twelve months - technically competent but insufficiently intense-consistently demonstrated rapid functional gains once intensity was raised above the neuroplastic threshold. The transition from $I \approx 0.18$ to $I \geq 0.7$ within the first two weeks of intensive protocol routinely produced the first measurable voluntary extension movements in patients who had been told recovery was no longer possible. This pattern - observed in more than forty patients over ten years - constitutes the empirical basis for the neural concentration threshold concept formalised in this article.

In the Cameroonian context, the most striking field observation was the inverse relationship between therapeutic nihilism and treatment delay. Patients arriving 18–36 months post-stroke - far beyond the conventional six-month window - consistently achieved clinically significant recovery once intensive protocols were initiated. The Cogni-Famille protocol, developed from direct observation that family members in Bafoussam were already attempting spontaneous task-shifting in the absence of professional guidance, was formalised precisely to harness this community resource systematically. The 5+ hours per day of therapeutic contact achieved through trained family members - at a total cost of 38–42 USD over twelve weeks - cannot be replicated by any institutional protocol in this context, and its outcomes challenge the assumption that resource scarcity precludes effective neurorehabilitation.

A recurrent observation in both settings deserves specific mention: the moment a patient receives an explicit, measurable functional goal - replacing the vague prognosis of “some improvement possible” - the quality of their effort changes visibly within the same session. This observation, repeated across hundreds of consultations, constitutes the clinical foundation of the Neurolexic Effect and the E component of the therapeutic intensity formula.

Conclusion

Deforming spastic paresis after stroke is not a fixed deficit but a partially iatrogenic, largely reversible condition whose trajectory is determined as much by the intensity of rehabilitation as by the severity of the initial neurological lesion. The pathophysiological cascade from neural injury to spastic myopathy through underuse can be interrupted and reversed at any chronic stage, provided that therapeutic intensity crosses the neuroplastic threshold - quantified here as $I = (R \times L \times E)/T \geq 0.7$.

The two cases presented demonstrate this principle across maximally contrasted resource environments: a private practice in France and a regional hospital in Cameroon. In both settings, a 12-week intensive protocol - combining aggressive prolonged stretching, maximum-amplitude alternating movements, guided self-rehabilitation, and when applicable the Cogni-Famille task-shifting framework - produced clinically significant functional recovery, return to occupational goals, and intensity increases of 6 to 17-fold relative to prior conventional treatment.

The Low-Tech, High-Sense paradigm is not a compromise born of resource scarcity - it is a clinical principle: human expertise, deployed with precision and intensity, is the primary driver of neuroplastic recovery in post-stroke spastic paresis. Equipment follows expertise; it does not replace it.

Ethics Statement

This review article is based exclusively on previously published data and does not involve new human participants, experimental procedures, or primary data collection. Clinical observations referenced herein were conducted under the Institutional Certification N 43/DRSO/HRB/55/2023, issued by the ethics comity, direct by the Deputy Director of the Regional Hospital of Bafoussam (Dr. ALIMA Jean Marie, Medical Advisor), which certifies that retrospective clinical evaluations based exclusively on anonymised data obtained from routine patient care do not require prior ethics committee approval, provided they are conducted under the direct coordination and oversight of the responsible department chief. All research was conducted in full accordance with the Declaration of Helsinki and applicable Cameroonian healthcare regulations.

Author Contributions

I.N.M. (DR Ibrahim Npochinto Moumeni, Rehabilitation Medicine specialist): conceptualization, theoretical framework development, literature synthesis, original draft preparation, critical revision, and final approval of the submitted version.

A.N.N.M. (PT. Abdel-Nasser Njikam Moumeni): clinical data verification, contribution to case documentation, manuscript review, and approval of the final version.

M.L.M. (PT. Mohamed Lamine Moumeni): contribution to literature search, manuscript review, and approval of the final version.

B.O.T.K. (Bristher Orloster Tchuidjio Ketchogu ): Nurse, clinical assistance in physiotherapy practice, contribution to clinical observation documentation, manuscript review, and approval of the final version.

Y.N.M. (Pr. Yacouba Njankouo Mapoure, senior neurologist): scientific supervision, neurological expertise, critical revision of the manuscript, and approval of the final version.

Data Availability

This is a narrative review article. No primary datasets were generated. All referenced data are available in the cited published sources. Additional clinical information is available from the corresponding author upon reasonable request.

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