

Lifting protective inhibition: the EEG signature of a “released” muscle

EEG pilot study (subject Barbara) — two muscles, two opposite starts, one common landing

Level of evidence : Observed in-house (frontal EEG signal) · Hypothesis in progress (protective inhibition lifted by Q-Theta) — pilot, n = 1

Study type	Exploratory EEG pilot, within-subject, without Q vs with Q
Participant	One subject (Barbara), one session
Measure	Muse EEG (Athena), frontal sensors AF7/AF8 — frozen signal removed
Muscles	Deltoid and latissimus dorsi
Q circuit	Q-Theta
Protocol	9 rapid isometric resisted contractions / muscle, without then with Q-Theta
Data read	Mean frontal activation (EEG index) — force judged by manual testing
Status	EEG signal = observation · “inhibition lifted” reading = hypothesis to be validated

Summary

Targeted EEG study (distinct from the Kinvent force series). Working hypothesis: a muscle that tests weak is not necessarily injured — it may be under a protective neurological inhibition. On two muscles of one subject (deltoid, latissimus dorsi), frontal activation (Muse EEG, AF7/AF8) starts from opposite levels — deltoid low (0.95), latissimus high (1.91) — and both converge toward a median zone (~1.5) with Q-Theta: the low one rises (+72%), the high one falls (–25%). Proposed reading: a cortical NORMALIZATION (neither over-guarding nor under-connection). Explicitly unproven hypothesis — this document also names the confound (regression to the mean) and the protocol that will tell them apart.

1. Background and objective

A muscle that tests weak is not necessarily damaged. It may be held back: the nervous system places a protective inhibition on it, and while that is there, the muscle tests weak regardless of will. It is not a force problem, but an access problem.

How to tell an injured muscle from a held-back one? Test it nine times in a row. A truly pathological muscle fatigues and weakens further; a muscle merely under inhibition stays weak but stable — it does not worsen. It is this “stable” profile that can be “released.”

The aim of this measurement: to look, via EEG, at what the cortex does during this test — without then with a Q-Theta circuit.

2. Method

Targeted EEG protocol:

- Participant: one subject (Barbara), one session.
- Measure: Muse EEG (Athena), frontal sensors AF7/AF8 — frozen-signal segments removed.
- Muscles: deltoid and latissimus dorsi.

- Protocol: 9 rapid isometric resisted contractions per muscle, without circuit then with Q-Theta in contact.
- Data read: mean frontal activation per condition (unitless EEG index).
- Conditions: without Q-Theta, then with Q-Theta — fixed order.
- Important: force was judged by manual testing (not instrumented). The analysis here is on the EEG signal, not on an instrument-measured force.

3. Results — the EEG signature

OBSERVATION Mean frontal activation — two muscles, two opposite starts, one common landing.

Muscle	Without Q-Theta	With Q-Theta	Reading
Deltoid	0.95	1.63	low start → rises (+72%)
Latissimus dorsi	1.91	1.44	high start → falls (-25%)

The two muscles start from very different frontal activation levels — the deltoid low (0.95), the latissimus high (1.91) — and both converge toward a median zone (~1.5) with Q-Theta.

So it is not “more activation” or “less activation” as a rule: it is a move toward a common landing point, from two opposite directions.

4. Reading under the protective-inhibition hypothesis

HYPOTHESIS Interpretive reading, unproven — under the protective-inhibition hypothesis.

Latissimus dorsi — the over-guarding would release. High starting activation: the cortex “stands guard” over a muscle it keeps inhibited. With Q-Theta, activation drops (-25%): once coherence is restored, the brain would no longer need to over-monitor — it would lower its guard. Less cortical effort for a muscle that itself becomes strong again.

Deltoid — the connection would re-establish. Low starting activation: the cortex–muscle link would be “under-fed.” With Q-Theta, activation rises (+72%): the brain would reinvest the link, the time to restore an effective command.

The common thread: Q-Theta would not push the cortex in a single direction, but bring it back toward a median functioning — neither over-guarding nor under-connection. A normalization, consistent with the idea of a safety being lifted rather than a forced stimulation.

5. The 9-contraction sign

An EEG-independent support. The 9-contraction protocol is not just repetition: it is a diagnostic test. A truly injured muscle fatigues and gives way more as it is solicited; a muscle merely under inhibition stays weak but STABLE — it does not worsen.

Here, the muscles tested weak but without worsening across the 9 repetitions: the clinical sign points to inhibition, not a lesion. This is a line of evidence distinct from the EEG signal, and it points the same way.

6. What could disprove it — regression to the mean

The point to lock down, and it is serious: the “high goes down, low goes up” pattern is also exactly what a simple regression to the mean would produce — a statistical phenomenon in which extreme values mechanically tend toward the center on a second measurement, with no biological cause.

Until this confound is ruled out, the observed convergence does not prove a neurological normalization. To settle it, one must PRE-REGISTER the predicted direction of each muscle — from its starting state — BEFORE applying Q-Theta. Predicting correctly and repeatedly distinguishes a true normalization from a statistical artifact.

A second limitation to name plainly: force here was judged by manual testing, not measured on an instrument. That is weaker than the rest of the series (dynamometer). The “force” side of this measurement therefore needs to be instrumented.

7. Validation protocol

To turn this appealing hypothesis into a defensible result:

- Pre-register the per-muscle prediction (rises / falls) from its starting activation, BEFORE applying Q-Theta.
- Instrument force (dynamometer) and document stability across the 9 repetitions (the “no worsening = inhibition” signature).
- Look at the temporal dynamics second by second: re-connection predicts a peak then a plateau; released over-guarding predicts a sustained drop.
- Replicate across several muscles and subjects, classified by starting state (over-guarded vs under-connected).
- Ideally, measure cortico-muscular coherence (EEG–EMG) as a direct marker of the brain–muscle link.

8. Limitations

- $n = 1$, two muscles, one session, fixed order.
- Force judged by manual testing (not instrumented); short EEG windows; frontal sensors only (AF7/AF8).
- The “inhibition lifted → normalization” reading is a mechanistic hypothesis, coherent but unproven; the regression-to-the-mean confound is not ruled out.
- The 9-contraction sign (stable = inhibition) is an independent support, but on a single subject.

9. Conclusion

An elegant EEG signature — two muscles with opposite starts converging toward the middle under Q-Theta — and a coherent hypothesis: a protective inhibition being lifted rather than a forced stimulation. The clinical 9-contraction sign supports the idea of a brake, not a lesion. But we present it for what it is: the observed pattern could also come from a simple regression to the mean, and force was not instrumented. Proof will require a pre-registered prediction, instrument-measured force, and replication. Framing: an exploratory pilot study, to be validated, with no medical claim.

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Source: Muse EEG (Athena), frontal sensors AF7/AF8, 9 contractions/condition, deltoid and latissimus dorsi. Unaudited internal data. Reading under the protective-inhibition hypothesis. Not a medical claim.