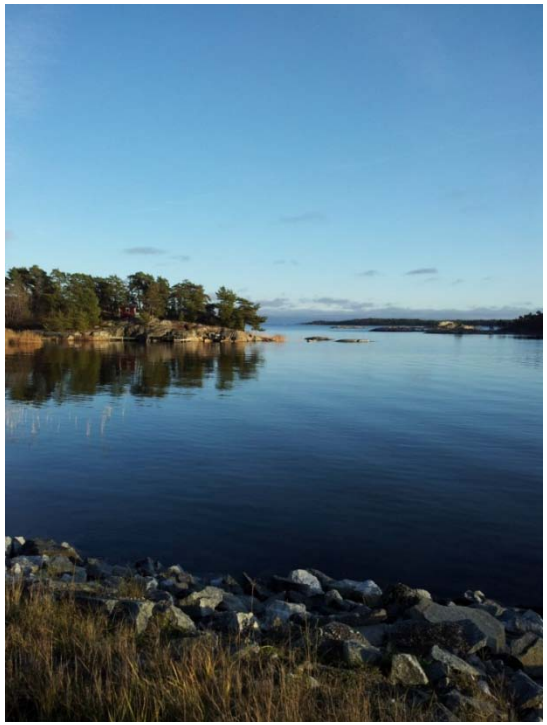
Development of spasticity after stroke – preliminary results from the Prohand Study



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Recovery of hand function after stroke

- A majority of patients with stroke experience impaired function in the upper limb (UL) — Welmer et al.2008; Houwkink et al. 2013
- ~ 50% regain full function — Kwakkel et al. 2003
- ~ 20-40% develop UL spasticity depending on time since stroke onset — Lundström et al. 2010; Opheim et al. 2014
- The role of spasticity for recovery remains unclear

The Prohand Study

The overall aim

– identify key determinants for recovery of hand function after stroke by applying newly developed hand function measures together with MRI measurements of connectivity in brain networks

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Aim of this study

The specific aims

– investigate the longitudinal changes of hand spasticity profiles and the relation between hand function recovery and the presence of an elevated neural component

Research methods and material

Design: Longitudinal explorative study

1st timepoint (T1) at 2-6 weeks

2nd timepoint (T2) at 3 months

3rd timepoint (T3) at 6 months

Subjects: 32 patients with 1st ever stroke

Setting: University Department of Rehabilitation Medicine Danderyd Hospital

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Research methods and material

OUTCOME MEASURES

- Neuroflexor — Neural, Elastic and Viscous components
- Modified Ashworth Scale (0-5)
- Passive range of motion (pROM)
- Fugl-Meyer Assessment for upper extremity (FMA-UE)
  - motor domain, reflexes items excluded (0-60)
  - domain for Joint Pain (0-24)

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Defintitions: Recovery: Δ FMA-UE motor score (T3 minus T1)

Spasticity: > 3,4N according to the Neuroflexor

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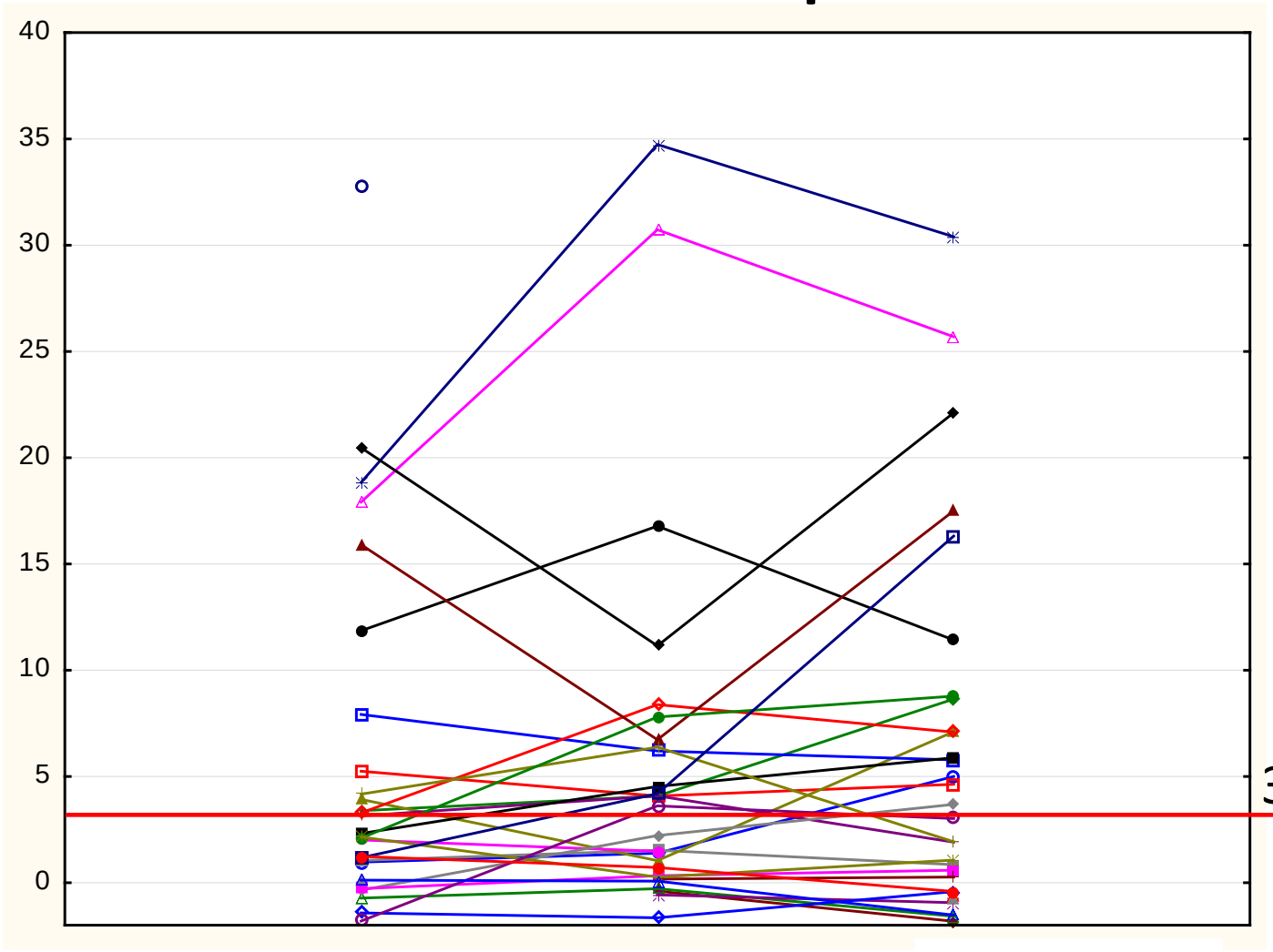
Patient characteristics (*n*=32)

Clinical variables		T1 Median (IQR)
Time after stroke, days		22 (17-29)
Age, years		58 (50-62)
Female / Male		12 (38) / 20 (62)*
Stroke type	Ischaemic	21 (66)*
	Haemorrhagic	11 (34)*
NIHSS		4 (3-11)
Barthel Index		75 (45-100)

* *n* (%)

Neural Component



Change over time: p=0.03 (rANOVA)

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Number of patients above cut-off – correspondance with MAS (*n*=32)

NeuroFlexor Variables	T1 *n* (%)	T2 *n* (%)	T3 *n* (%)
Neural component (NC)	11 (34)	15 (48)	15 (48)
NC **above** cut off / **Pos**. MAS	6	10	12
NC **below** cut off / **Pos**. MAS	5	5	3

(*n*=32)

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Neural Component at T1 – Spearman Correlations (r^s)

Clinical variables	T1	T2	T3
pROM	-0.42	-0.44	-0.52
FMA-UE motor score	-0.45	-0.40	-0.44
FMA-pain	-0.18	-0.39	-0.61

(n=32)

all values = $p < 0.05$ except for FMA-pain at T1

Neural Component at T1 – Spearman Correlations (r^s)

Clinical variables	T1	T2	T3
pROM	-0.42	-0.44	-0.52
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Repeated measures ANOVA

Dependent variables	Within factor	p-values
pROM	TIME	< 0.02
FMA-UE motor score		< 0.000

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Neural Component at T1 – Spearman Correlations (r^s)

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Repeated measures ANOVA

Dependent variables	Within factor	p-values	Predictor variable
pROM	TIME	< 0.02	NC at T1
FMA-UE motor score		< 0.000	(above/below cut off)

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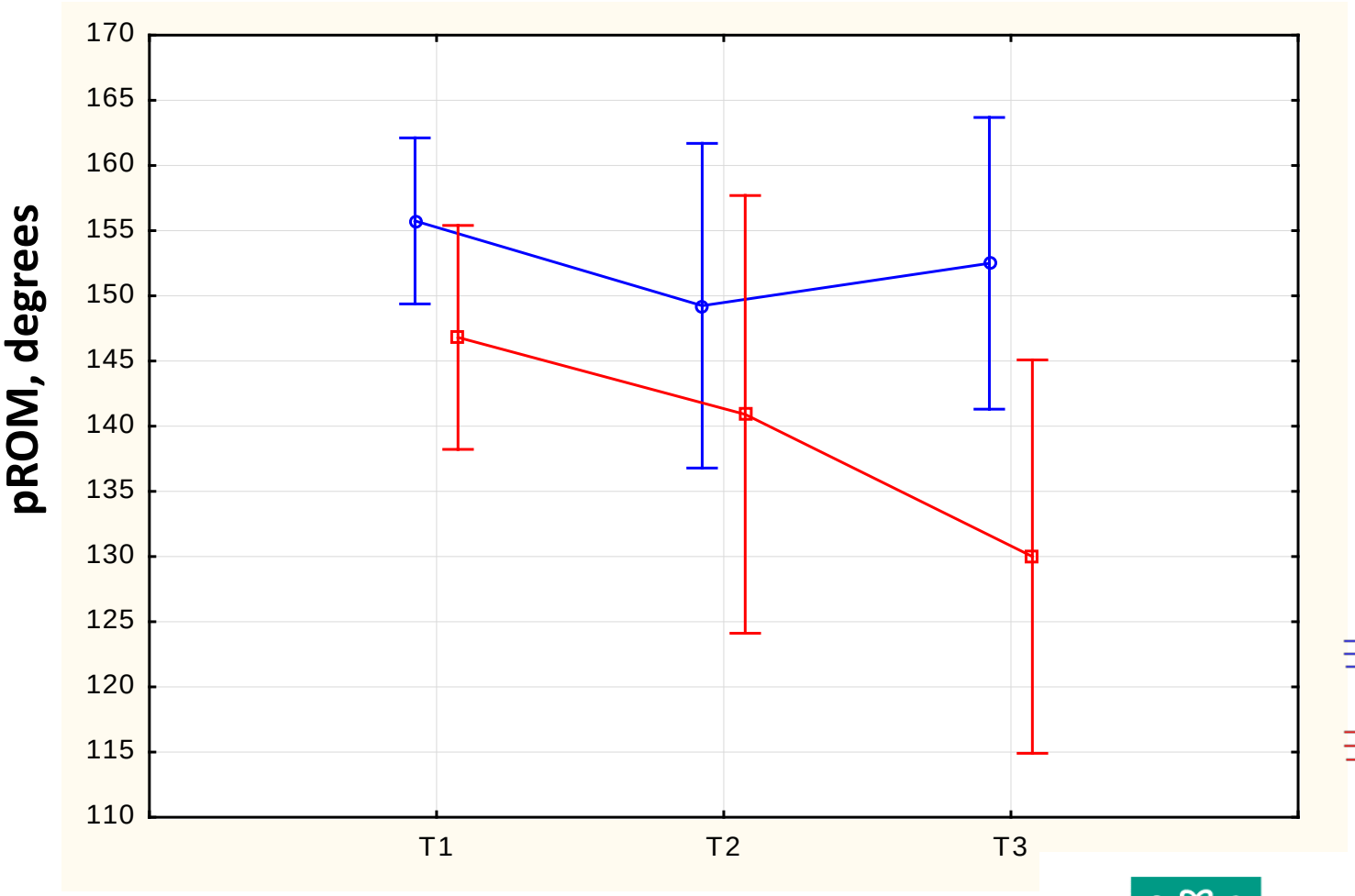
pROM over time - Neural Component interaction



Interaction p=0.06 (rANOVA)

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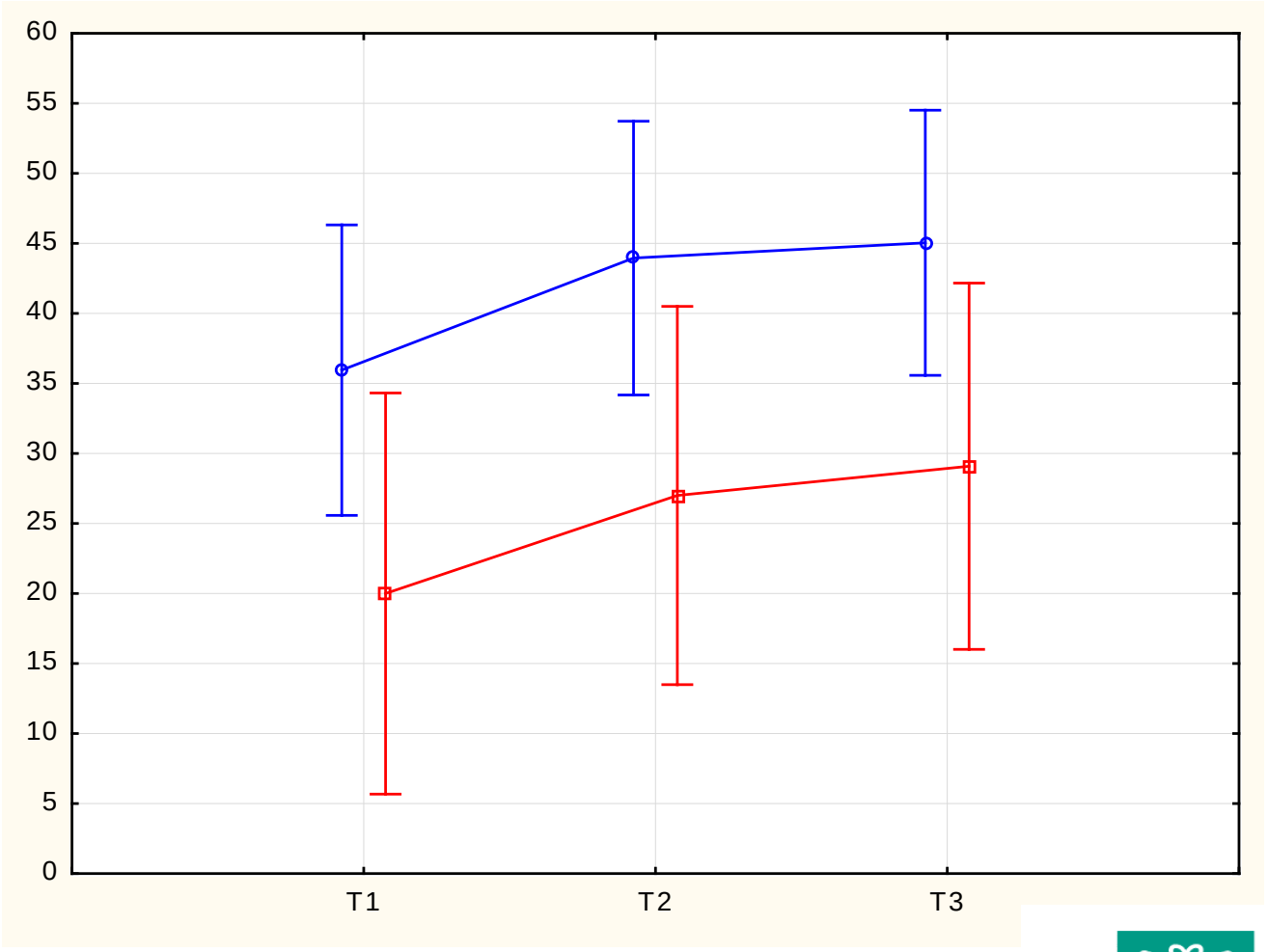
Motor recovery over time - Neural Component interaction



Interaction p=0,93 (rANOVA)

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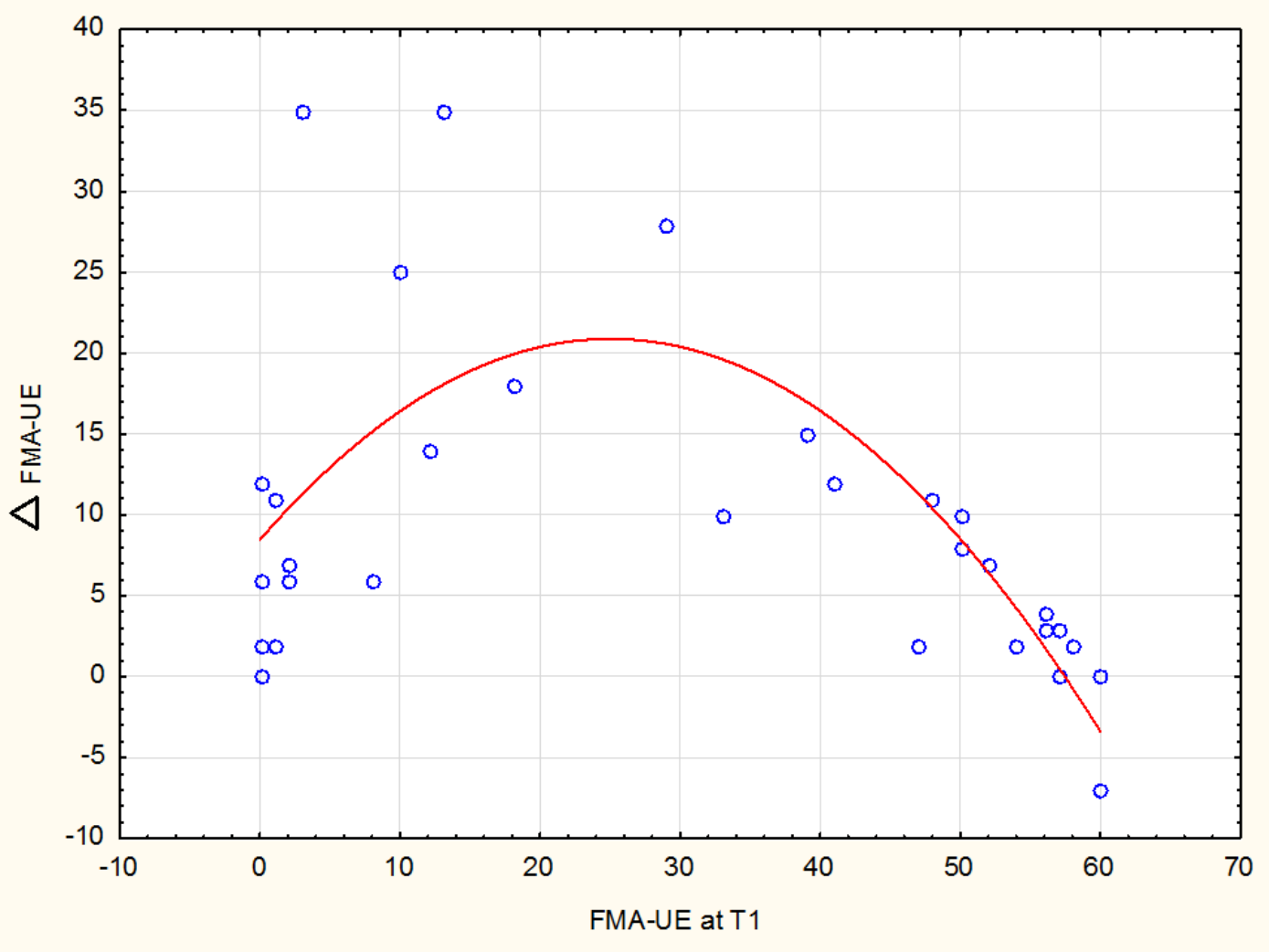
Motor recovery, does spasticity matter?



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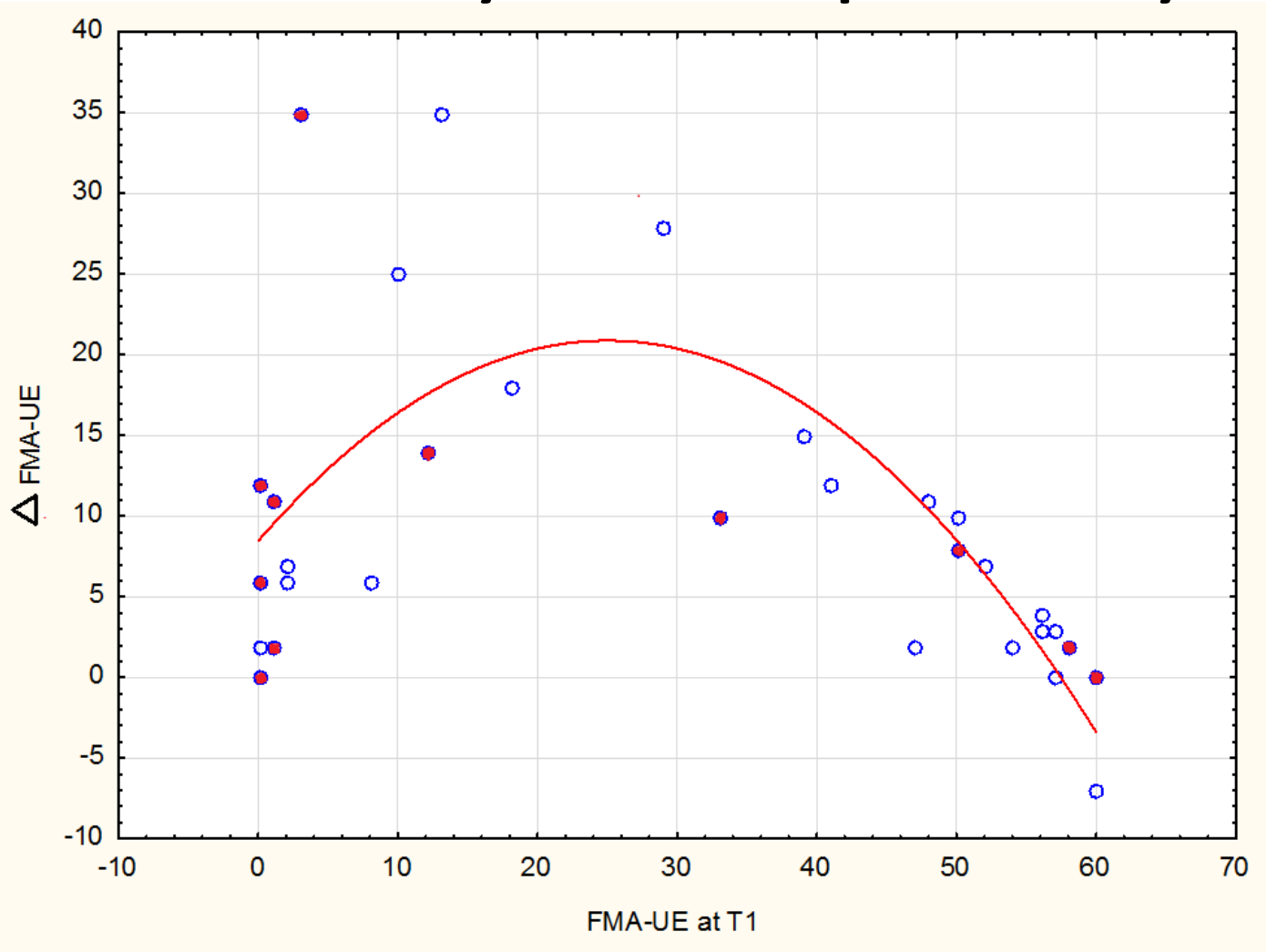
Motor recovery, does spasticity matter?



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Conclusion

- 34% of the patients had early spasticity
- Quantitative (NF) and clinical (MAS) measurements of spasticity were not congruent, especially not in the early phase
- Clinical variables correlated with early spasticity
- Clinical variables pROM and FMA-UE motor score changed over time but did not show a significant interaction with early spasticity
- High inter-individual variability in motor recovery

Thank You!

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