#### Electrophysiological approaches to compression and entrapment neuropathies

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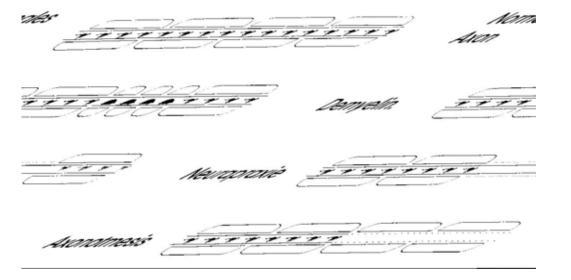


# Aims of EDX testing in nerve compression/entrapment

- Provides information on:
  - Localization
  - Motor, sensory or mixed involvement subclinical nerve damage
  - Axonal, demyelinating injury or both
  - Severity can be assessed
    - Neuropraxia;axonotmesis; neuronotmesis
  - Prognosis can be estimated
- Effect of treatments may be quantitated

## Type of compression

- Acute compression conduction block axonal loss
- Chronic entrapment demyelination axonal damage



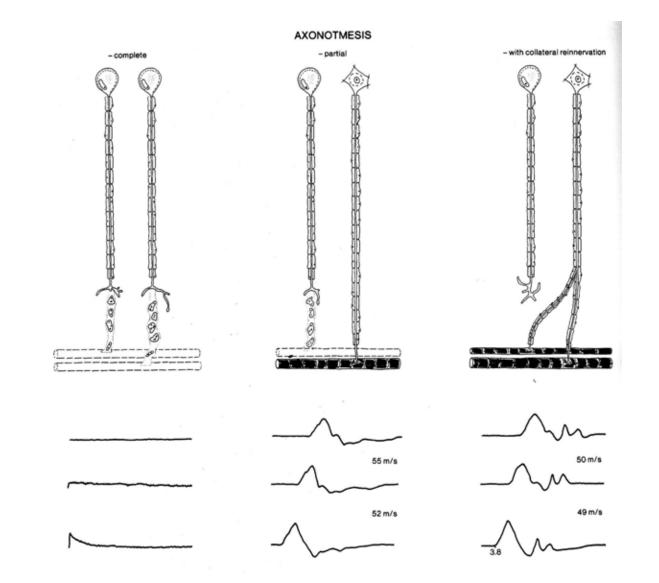
# Techniques

- Neurography
  - Nerve conduction studies (motor/sensory)
    - Conduction block
    - myelin problems
  - F-wave studies
    - proximal conduction
- Electromyography
  - loss of motor units
  - axonal degeneration and regeneration
  - time course of an axonopathy
  - early reinnervation

#### Pathophysiology of nerve lesion Axonotmesis

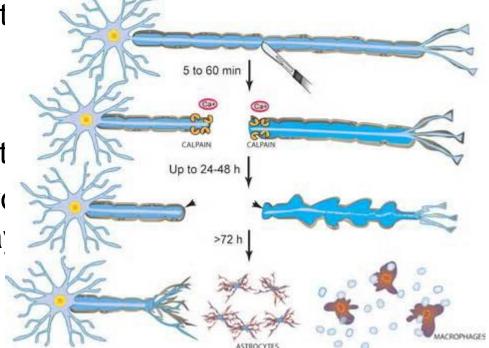
- Disruption of the axon and myelin sheath
- Supporting and connective tissue spared
- Recovery
  - May be minimal, incomplete or full
  - Dependent upon severity and length of nerve injured
  - Via axonal sprouting and regeneration
    - Growth rate of about 1-3 mm/day
  - In partial axonotmesis collateral reinnervation starts very early
  - Crush, stretch most common causes
  - Slow recovery, usually months

# Seddon types



# Neuronal Degeneration

- Distal to Injury Site
  - Follows axonal transection
  - Due to isolation of distal nerve segment from cell body
  - Wallerian degenerat
- Proximal to Injury Site
  - Degeneration back t
  - If very proximal, chreat transected axon mag



# EDX findings

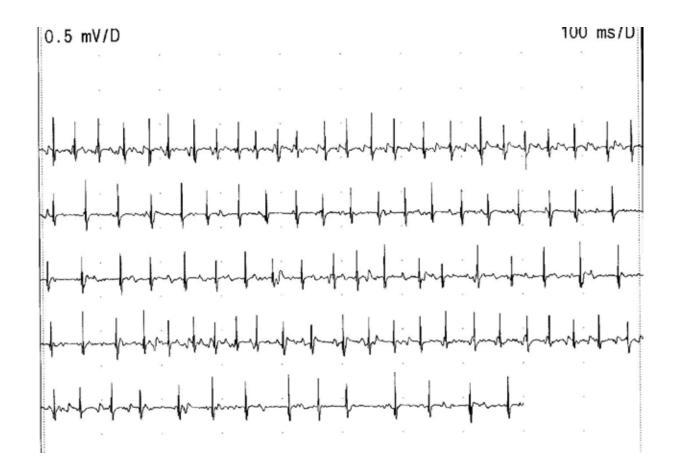
- Conduction studies are not specific and usually not useful to localize the damage
- Concentric needle EMG is essential in the diagnosis of an axonopathy
- It is helpful
  - to localize the lesion
  - to estimate to time course
- Findings depend on the severity (number of axons affected) timing of the entrapment (new, repeated)

# Time course of EMG findings in (partial) axonopathies

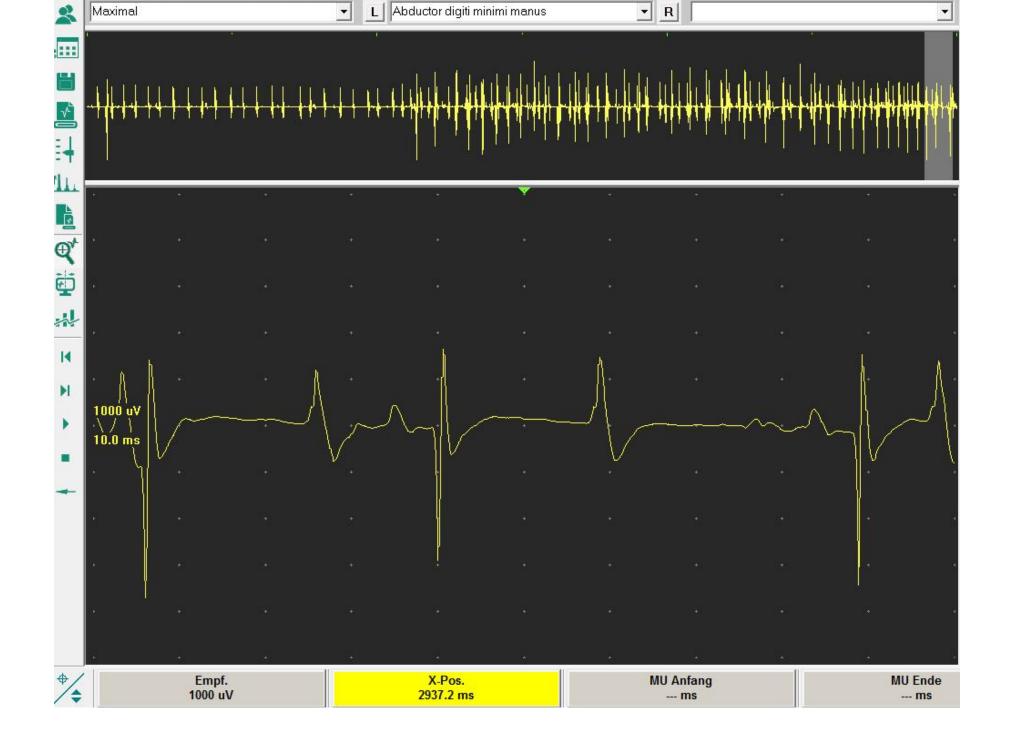
week	fibs	amplitude	polyphasic	satellite	discharge frequency
0-2	-	normal	none	none	>20Hz
3-6	++	normal	none	none	>20Hz
6-12	++	normal	few	yes	>20Hz
12-36?	+	increasing	many	rare	(>20Hz)
old	(+)	high	few	none	variable

Within the first 2 weeks conduction block and axonopathy have the same EMG findings

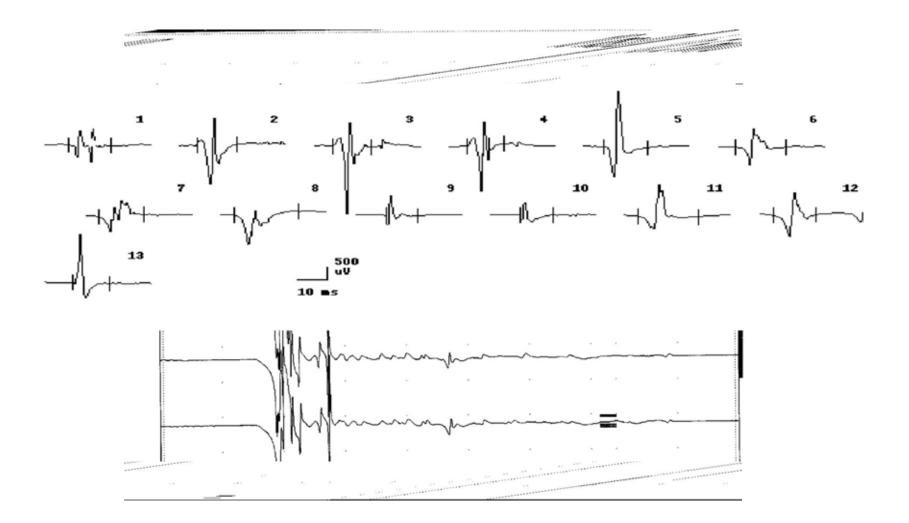
# MUP discharge frequency



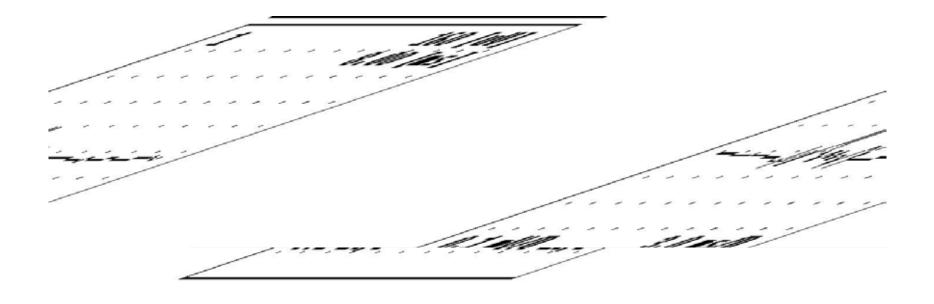
Discharge frequency does never differ between cb and axonopathy



# Partial axonopathy collateral reinnervation



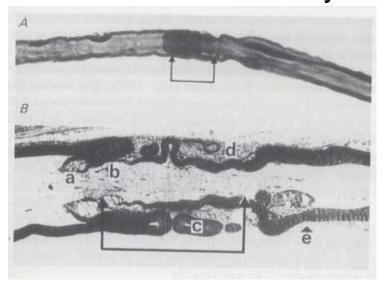
# Total axonopathy early reinnervation

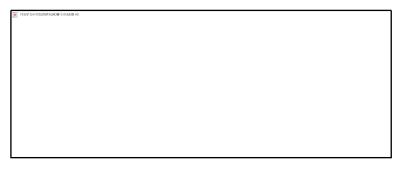




## Conduction block

- Nerve conduction is normal proximal and distal to the compression but there is no conduction across the lesion
- Axonal damage can be differentiated after 1 week by conduction velocity studies after 2 weeks by EMG too





Ochoa 1972

Pathophysiology of nerve lesion Neuroapraxia – conduction block

- Mildest type of nerve compression
- usually transient disruption of nerve function
- Ischemic, metabolic or microstructural abnormalities
- Axonal integrity maintained
- REVERSIBLE failure of nerve conduction
- Most often compressive/ischemic
- Normalizes within hours to weeks (months)

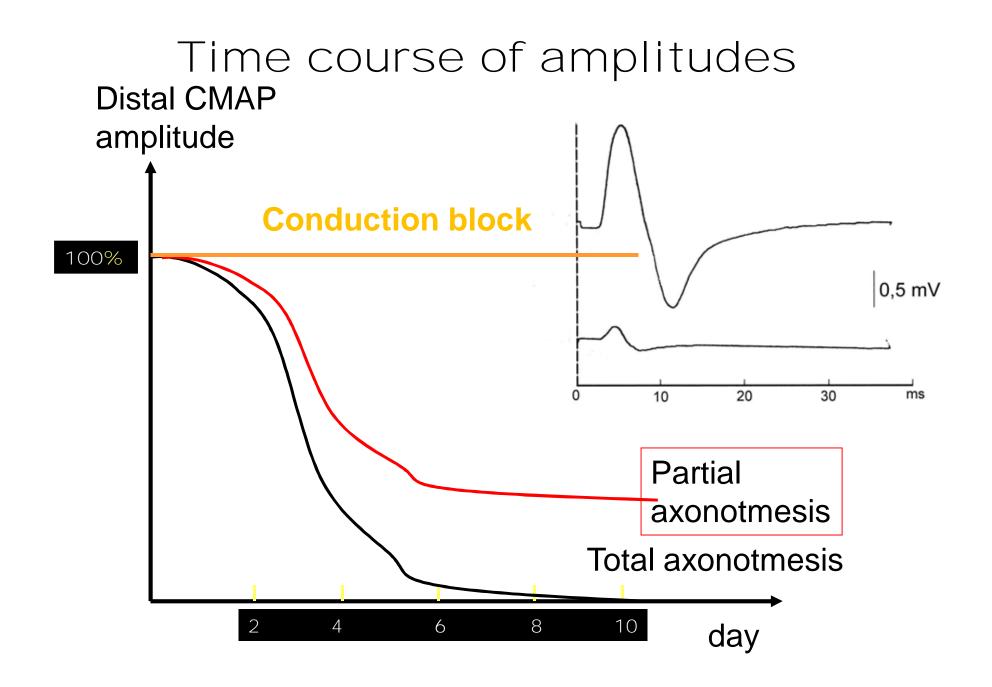
### Conduction block

- Acute due to compression
  - e.g. radial nerve peroneal nerve
- Special situations: distal and very proximal conduction block
- "chronic" conduction block in patients with MMN
- Hereditary liability to pressure palsy

# partial conduction block

- Clinical signs of weakness can only be found when >60% of nerve fibres are damaged
- Using EDX it may be found earlier
- Motor CB are better defined than sensory CB
- Short segment stimulation is the most reliable procedure
- EMG is normal, no fibs, normal MUP, except discharge rate of motor units may be >20/s

(as it is in weak muscles due to conduction block)



# Criteria

	No signs of temporal dispersion	CMAP duration increases by <30%	CMAP duration increases by 30-60%
	Definite CB	Possible CB	Possible CB
Median nerve	50%	40-49%	>50%
Ulnar nerve	50%	40-49%	>50%
Radial nerve	n.d.	>50%	>60%
Peroneal nerve	>60%	50-59%	>60%
Tibial nerve	>60%	50-59%	>60%

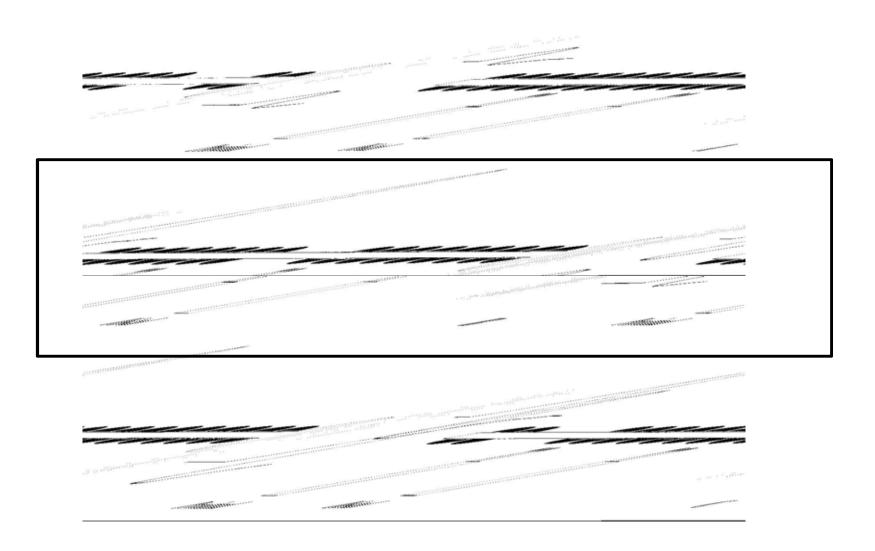
Amplitude >20% of LLN

# Pitfalls

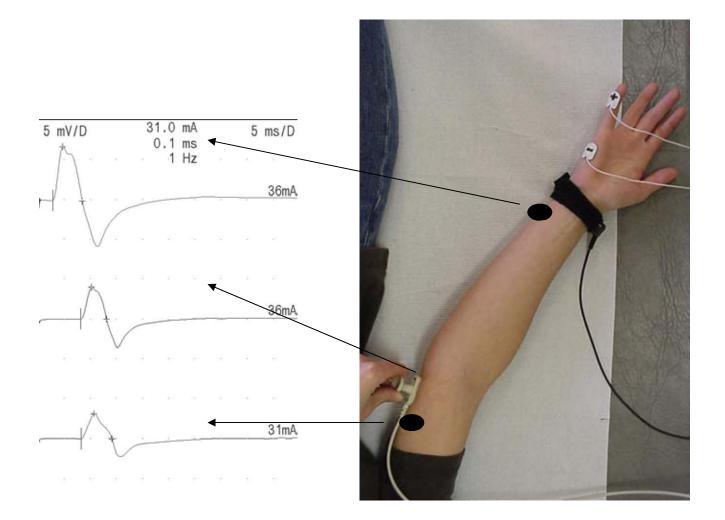
- Anatomical Localisation anastomosis
- Differentiation between conduction block and axonopathy
- Technical

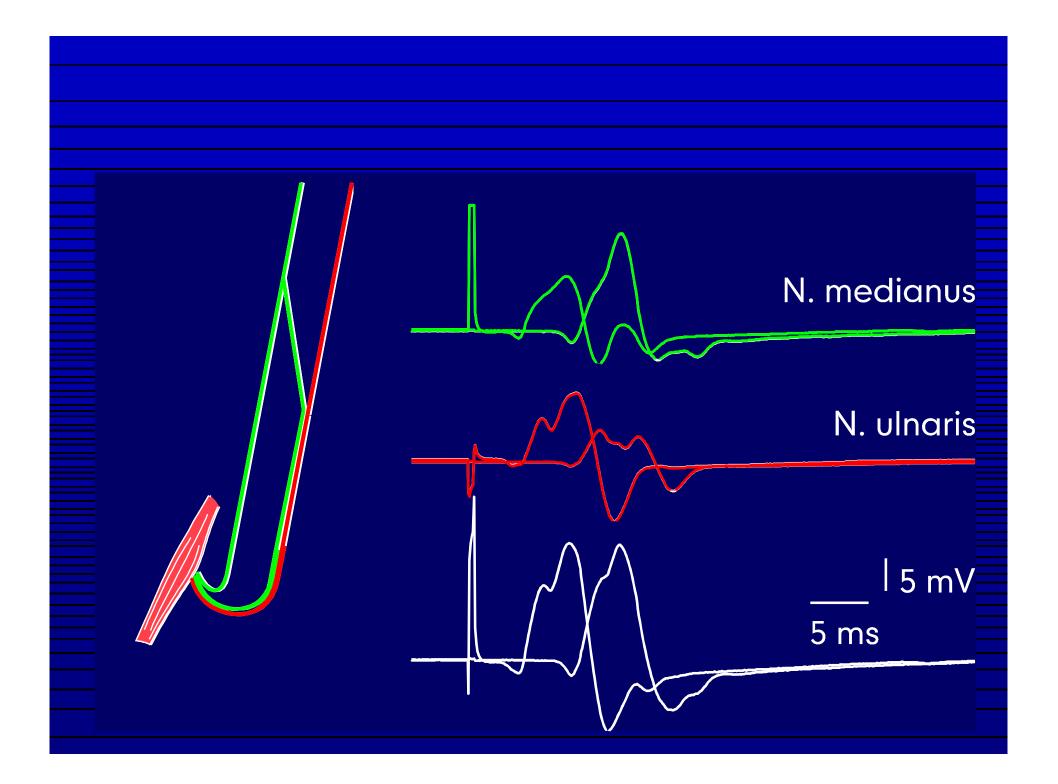
submax stimulation measurement error temperature latency measurement amplification positive deflections of the potential

# Pitfalls due to the distal localisation of a conduction block

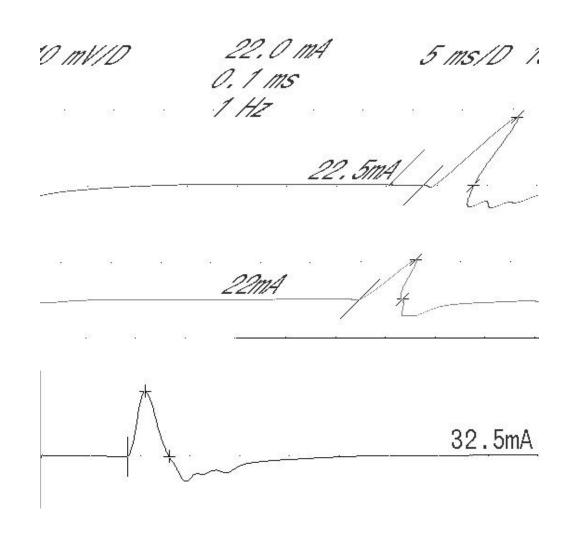


#### Conduction block?





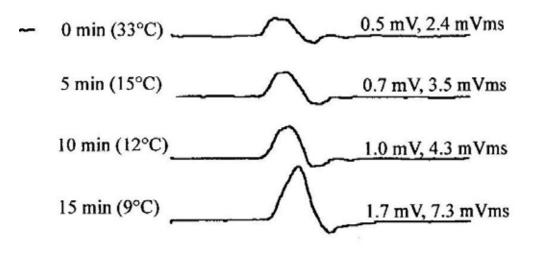
#### Conduction block?



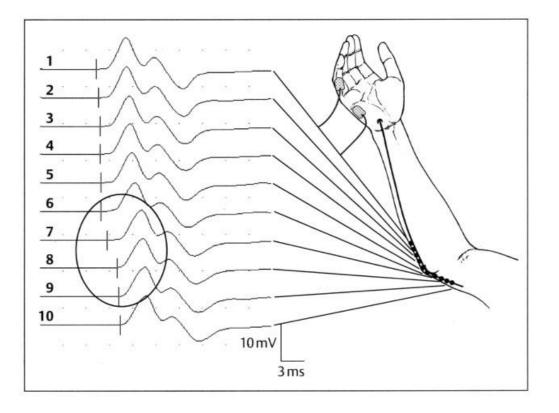
# Conduction block depends on temperature

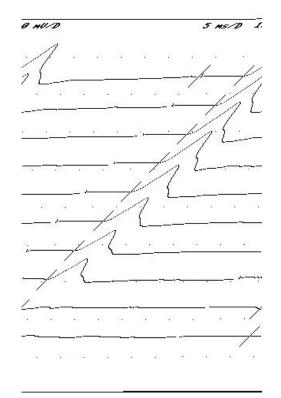
**Popliteal Fossa Stimulation** 





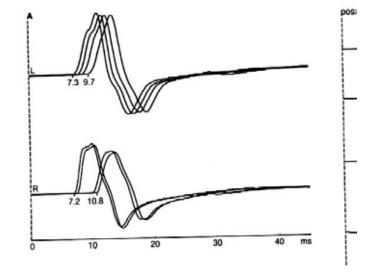
# Short segment stimuation Inching at the ellbow

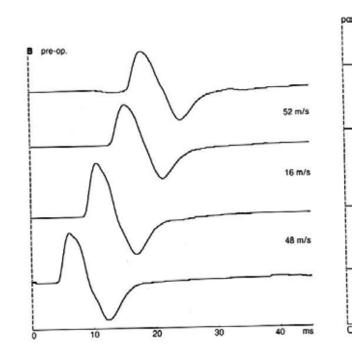




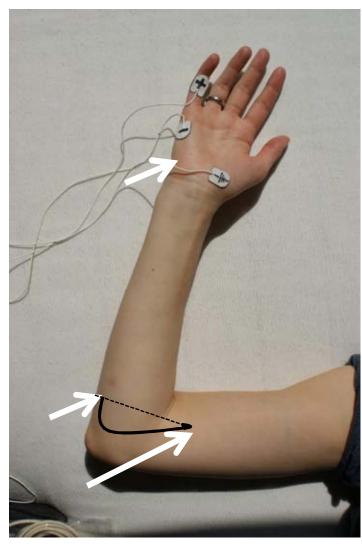


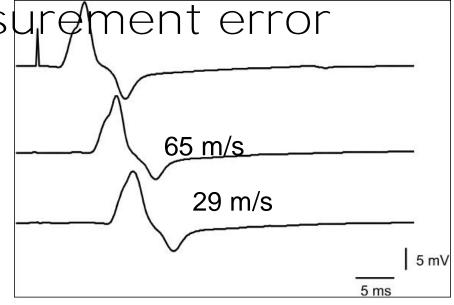


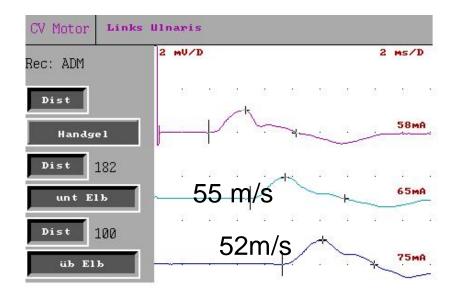




# Length measurgment error







# Pitfalls in sensory neurography

- Stimlus intensity too low (submaxima)
- Stimulus intensity too high
- Wrong position of the electrodes
- Temperature
- Anomaly of the innervation
- Not affected in proximal conduction block
- Electrical atrefacts

# Chronic entrapments

• Due to repeated denervation and renervation of the nerve segent:

Segmental slowing of nerve conduction velocity prolonged distal motor latency

- Temporal dispersion and therefore low amplitudes
- Worst case: very complex configuration of the CMAP but normal conduction velocity
- May be combined with axonal findings

#### Conduction block or not?

Amplitude Area CV mV mVs m/s 5 ms/0 5 mV/D 3,5 11,1 50 50 2,9 8,1 69mA 32 1,2 5,1

# Problems of sensory NCS

- Range of reference values is very wide
- Short segment studies are necessary to show conduction blocks
- loss of SNAP may due to axonal loss pronounced demyelination

# Indications

- Conduction block (only) technique to figure it out total or partial multifocal
- Chronic nerve compression
- Differentiation between plexopathy and radiculopathy
- Demyelinating neuropathies
  pattern of affected nerves