

Welcome and Introduction to Restorative Neurology

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External Facilitation for Restoration of Movement of Drop Wrist in Stroke Subject

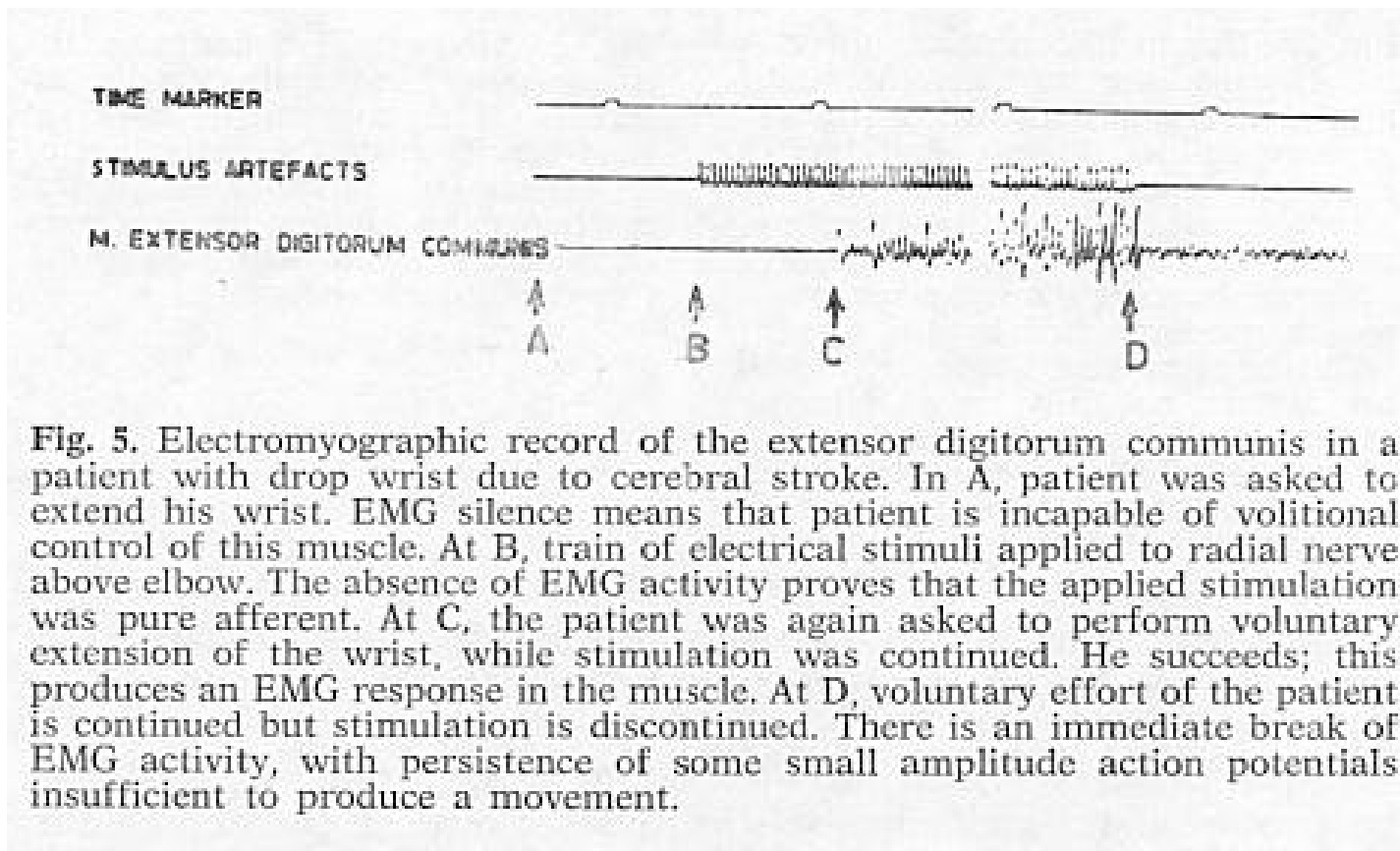


Fig. 5. Electromyographic record of the extensor digitorum communis in a patient with drop wrist due to cerebral stroke. In A, patient was asked to extend his wrist. EMG silence means that patient is incapable of volitional control of this muscle. At B, train of electrical stimuli applied to radial nerve above elbow. The absence of EMG activity proves that the applied stimulation was pure afferent. At C, the patient was again asked to perform voluntary extension of the wrist, while stimulation was continued. He succeeds; this produces an EMG response in the muscle. At D, voluntary effort of the patient is continued but stimulation is discontinued. There is an immediate break of EMG activity, with persistence of some small amplitude action potentials insufficient to produce a movement.

Ljubljana, 1957

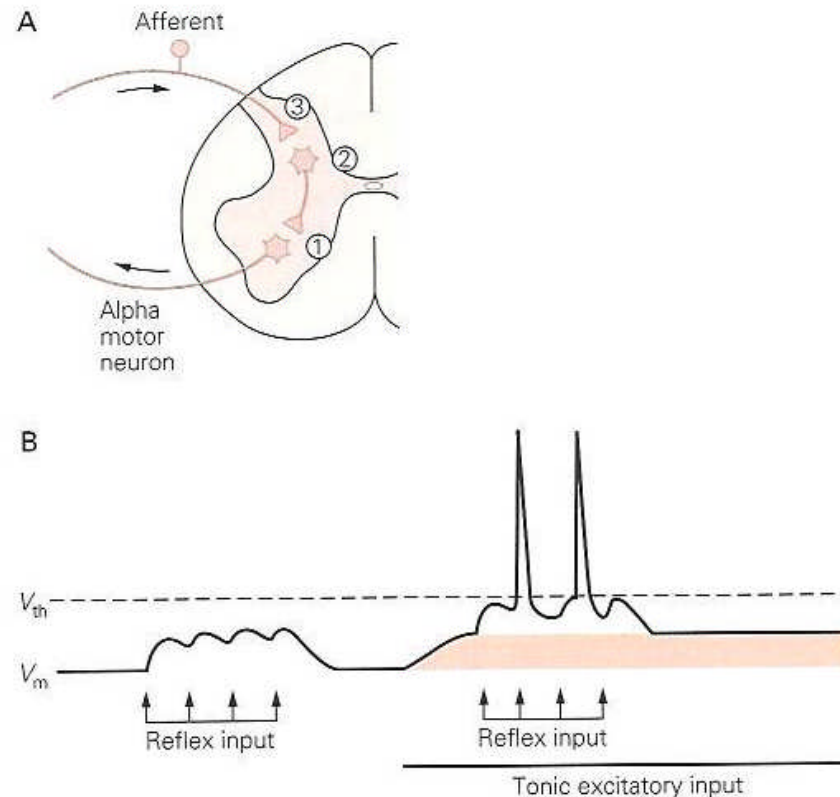


Figure 36-8 The strength of a spinal reflex can be modulated by changes in transmission in the reflex pathway.

A. A reflex pathway can be modified at three sites: (1) alpha motor neurons, (2) interneurons in polysynaptic pathways, and (3) afferent axon terminals. Transmitter release from the primary afferent fibers is regulated by presynaptic inhibition (see Chapter 14).

B. An increase in tonic excitatory input maintains depolarization in the neuron (**shaded**) and enables an otherwise ineffective input to initiate action potentials in the neurons (V_{th} = threshold voltage; V_m = membrane potential).

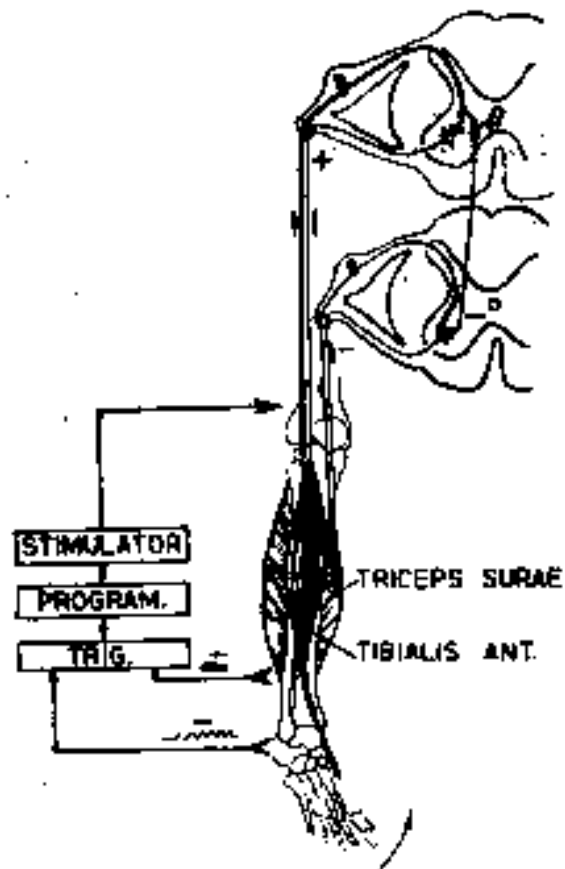
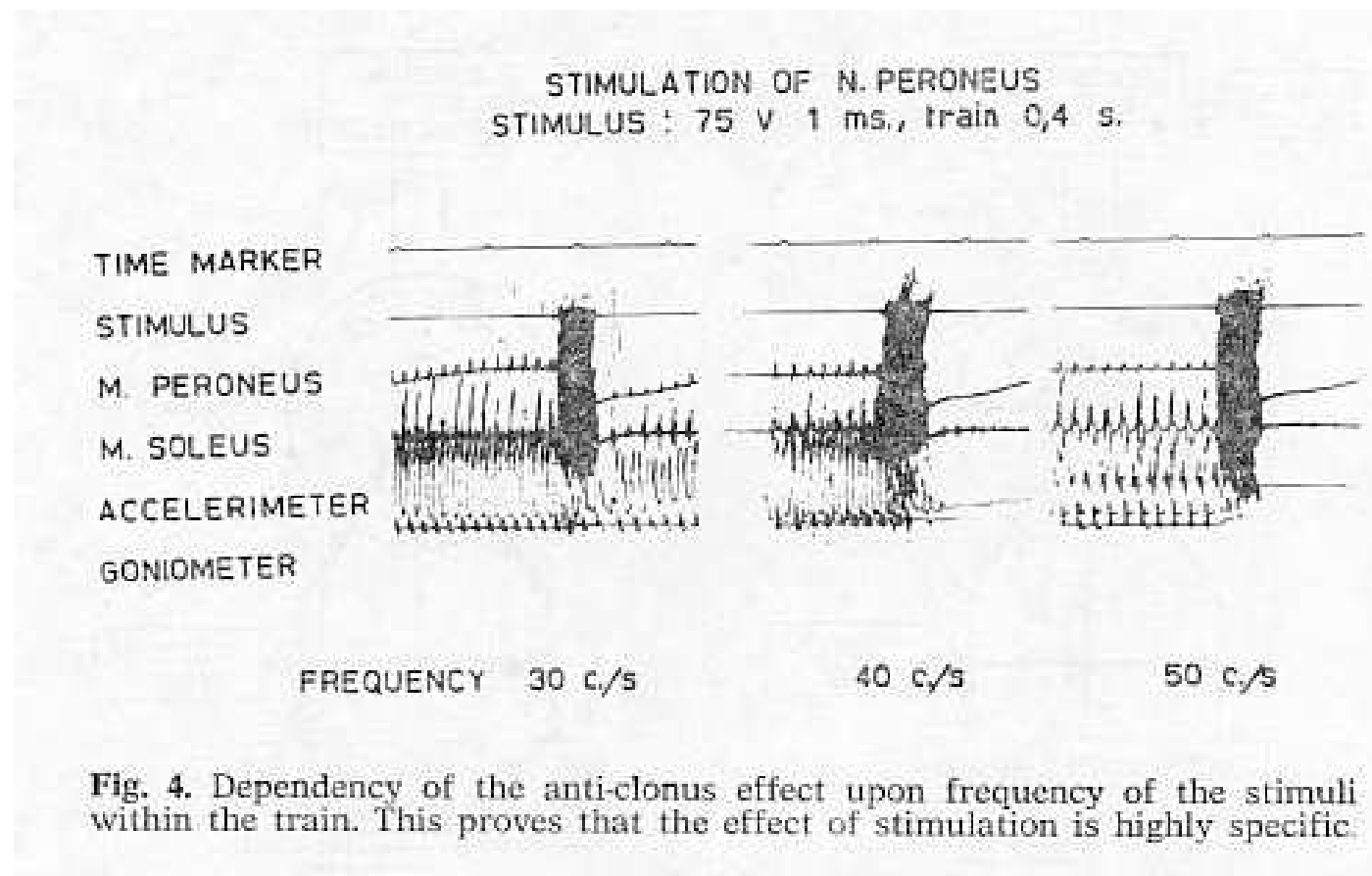


Fig. 8. Anti-clonus model. Clonus in triceps surae (detected by electro-mechanical or bioelectrical clonus detector) triggers stimulator, which delivers stimuli to afferent fibres of peroneal nerve. These evoke slight contraction in the tibialis anterior muscle, and at the same time inhibit clonic activity in antagonistic triceps surae muscle.

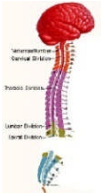
Ljubljana, 1960



Ljubljana, 1960

Summary of Observations

- 1. There is a neurocontrol of upper motor neuron paralysis
- 2.
- 3.
- 4.



sEMG of “spontaneous” spasm

[50 μ V Lt. gluteus max.

[50 μ V Lt. quadriceps

[50 μ V Lt. biceps

[50 μ V Lt. gluteus med.

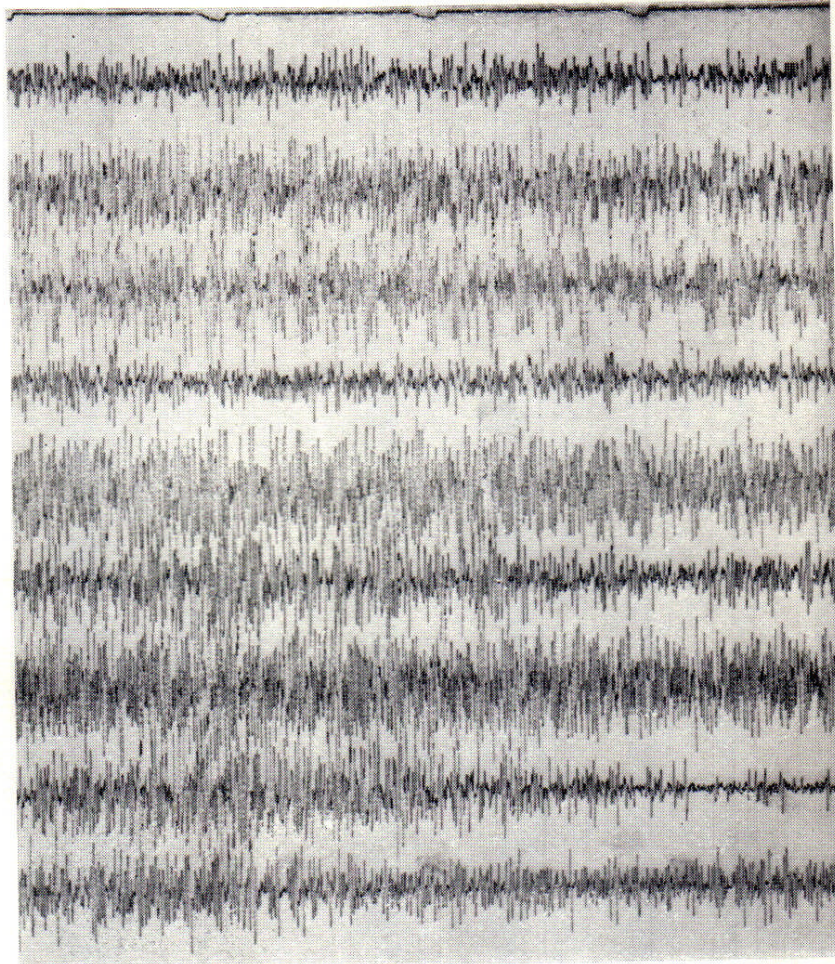
[50 μ V Lt. adductor

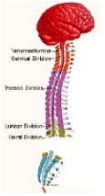
[50 μ V Rt. quadriceps

[50 μ V Rt. tibialis ant.

[50 μ V Rt. gastrocnemius

[50 μ V Rt. adductor





Early event of TJ reciprocal inhibition

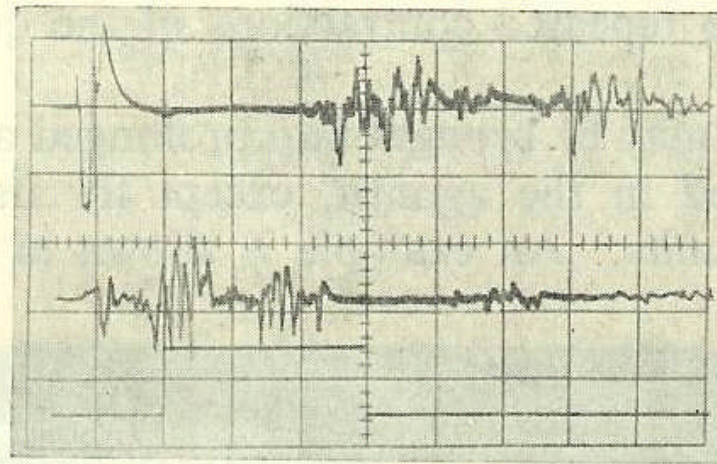
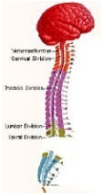


FIG. 3.—Electromyogram recorded on the oscilloscope of knee-jerk in a patient. Top channel, rectus femoris, lower channel, gracilis. The leg starts moving forwards at the point where the marker moves upwards and it has returned to its former position when the marker returns to the baseline. Each gridline on x-axis, 50 m.sec.; each gridline on y-axis, 0.2 mV.



TJ response and after discharge response

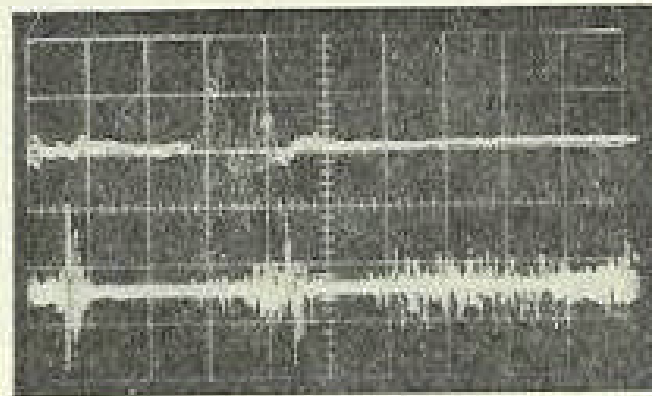
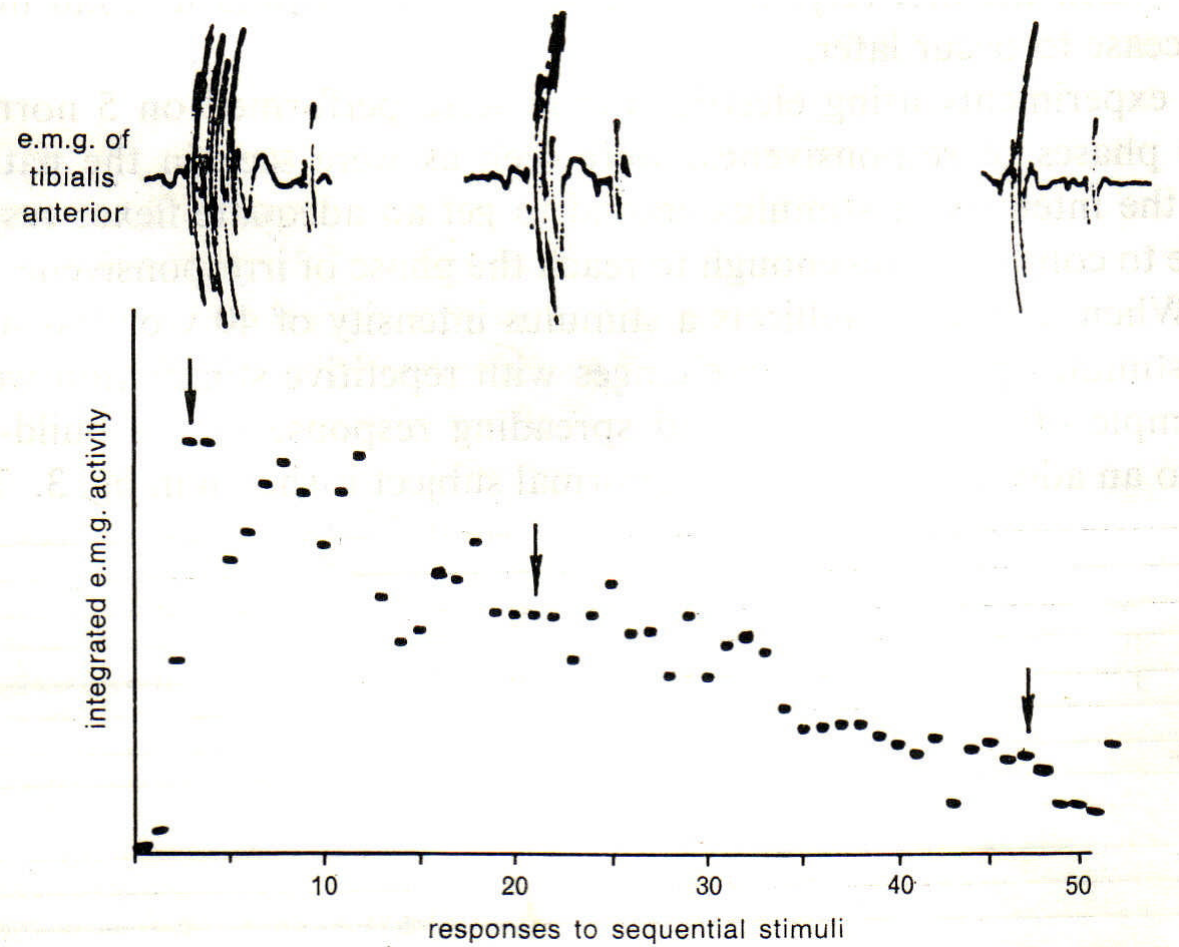
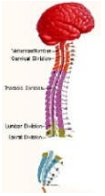
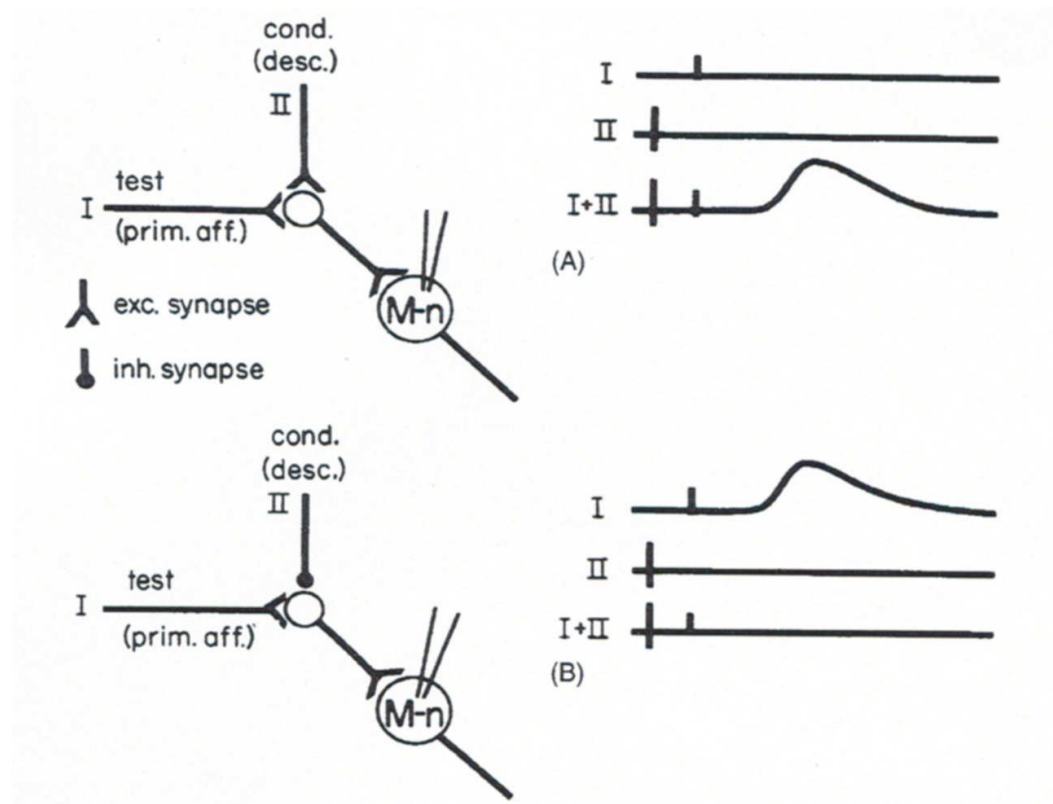


FIG. 2.—Electromyogram recorded on the oscilloscope of ankle-jerk in a patient, recording from gastrocnemius. Top channel, 3 jerks superimposed; lower channel a single jerk. Each gridline on x-axis, 10 m.sec. in upper record 50 m.sec. in lower record; each gridline on y-axis, 2 mV.

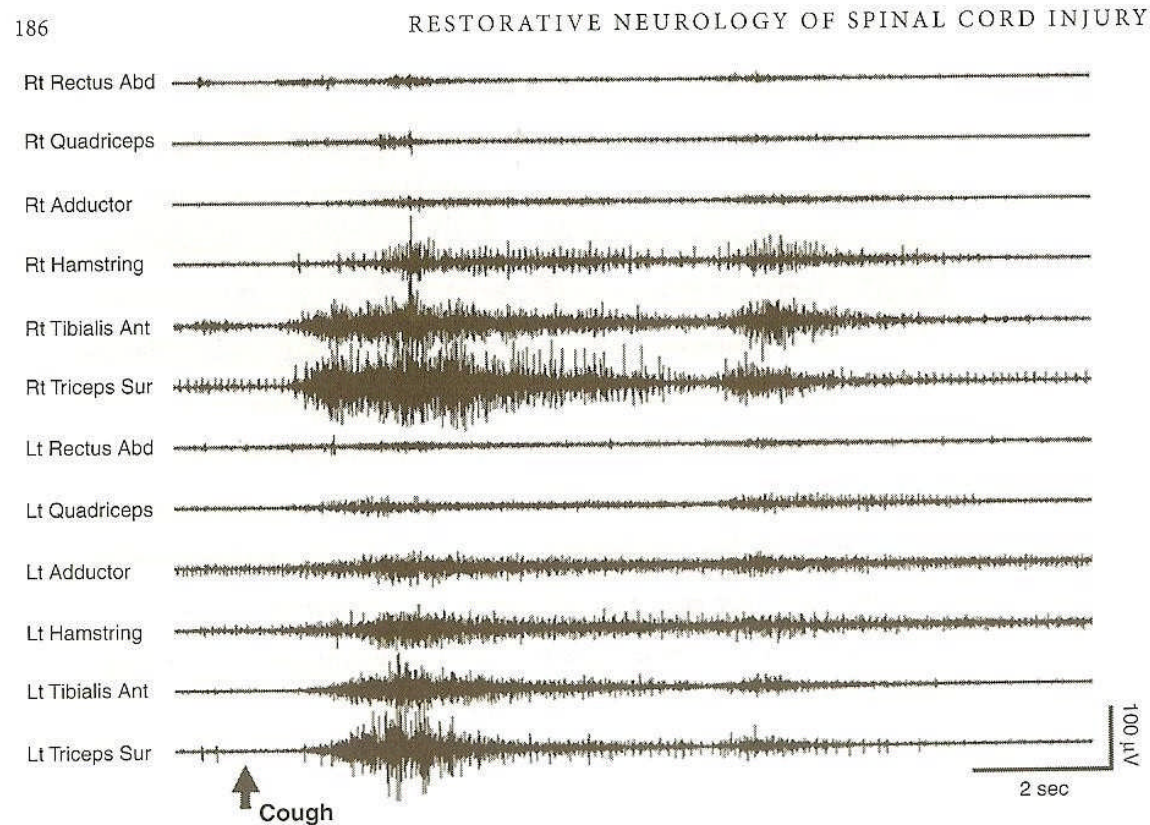
Habituation of repetitive plantar withdrawal reflex





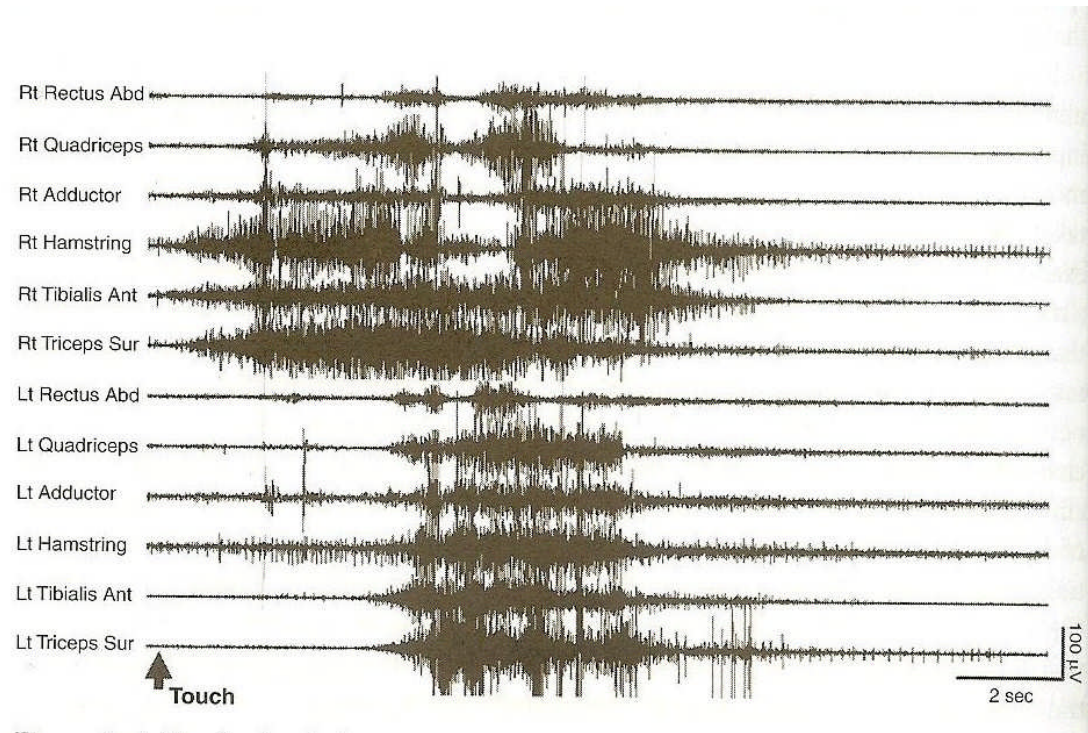
Convergence onto interneurons of reflex pathways

Spasm response to complex input



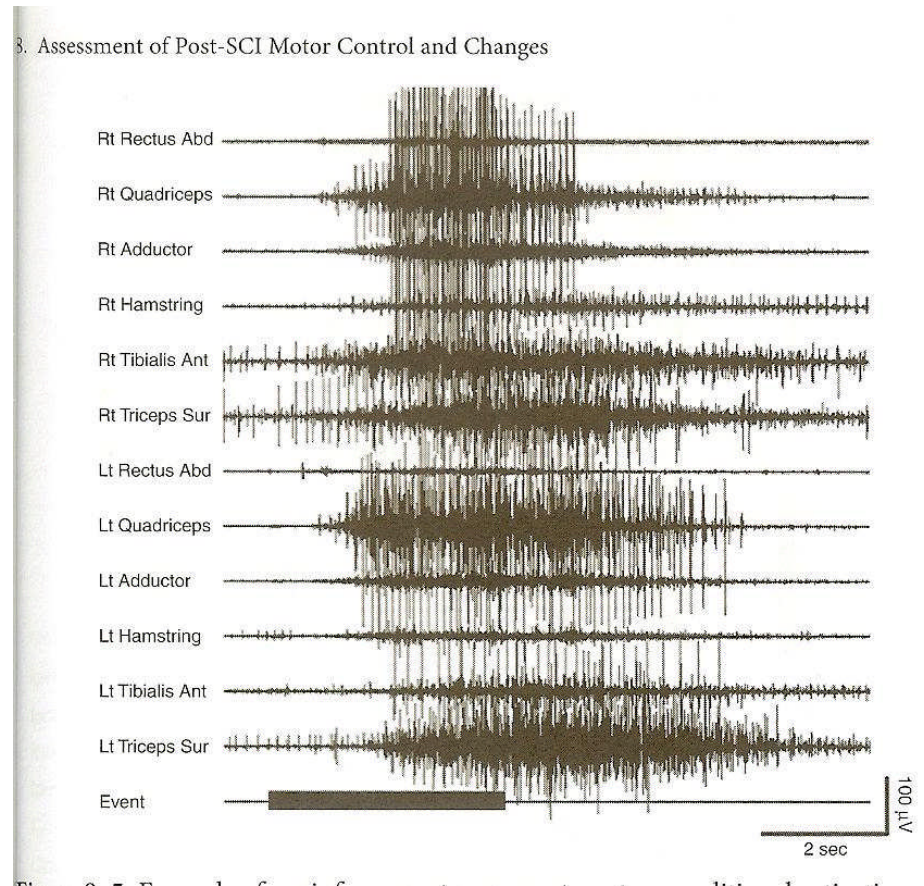
Spasms often occur without the stretch of any muscle, but rather in response to such provocations as coughing or touch.

Spasm response to simple input



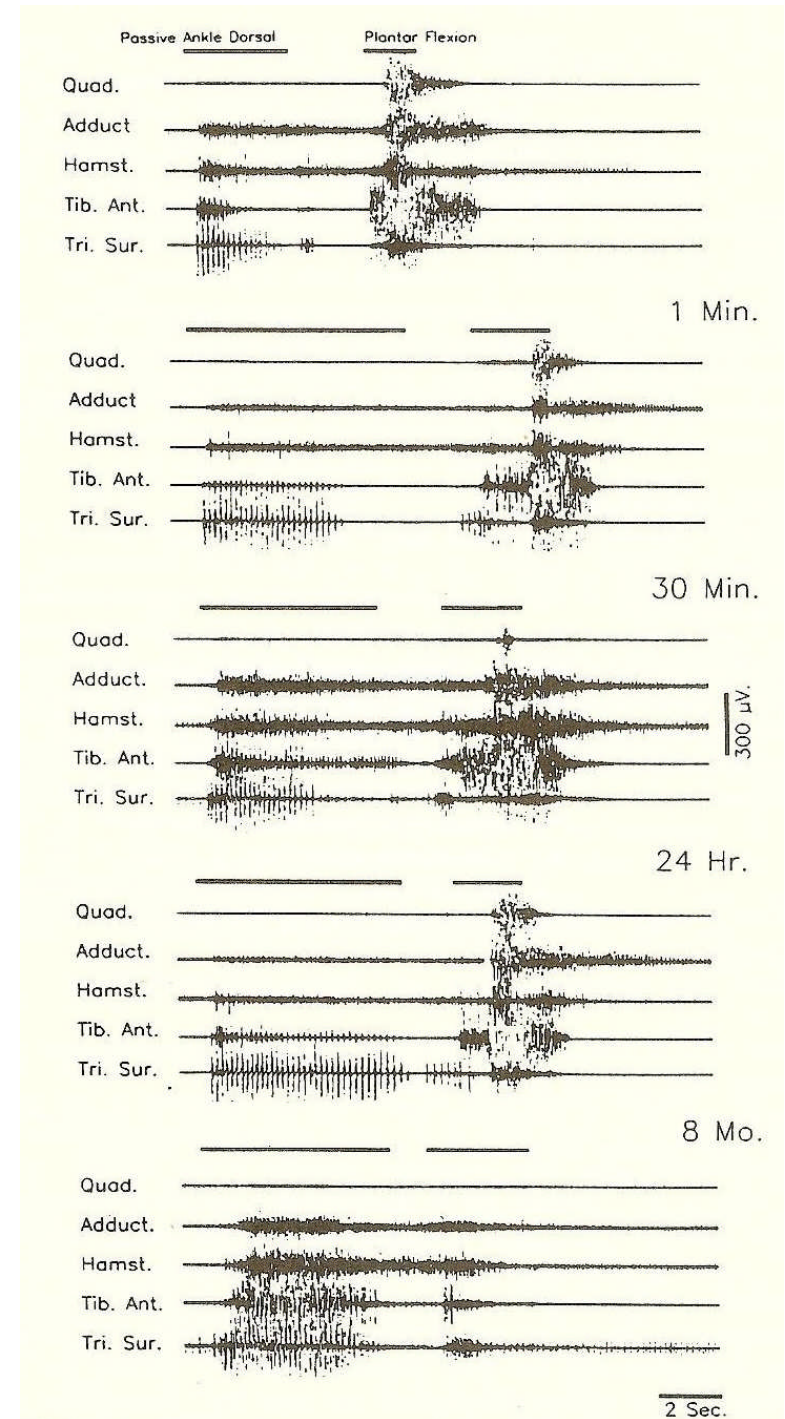
Spasms often occur without the stretch of any muscle, but rather in response to such provocations as coughing or touch.

Spasm response to reinforcement maneuver



Example of a reinforcement response to a strong volitional activation of muscles innervated from above the spinal cord injury level in a person with C6 AIS-C SCI. The shoulder shrug task replaces the classic Jendrassik maneuver in persons with weakened grip.

Consistency of “spastic” response to volitional dorsi- /plantarflexion



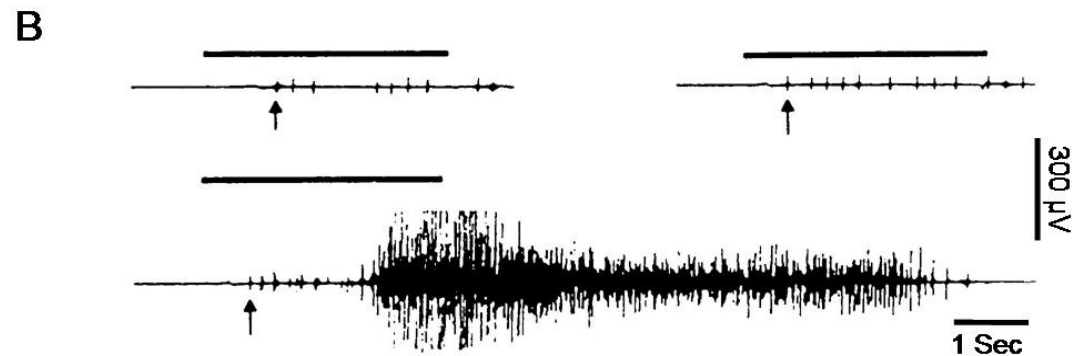
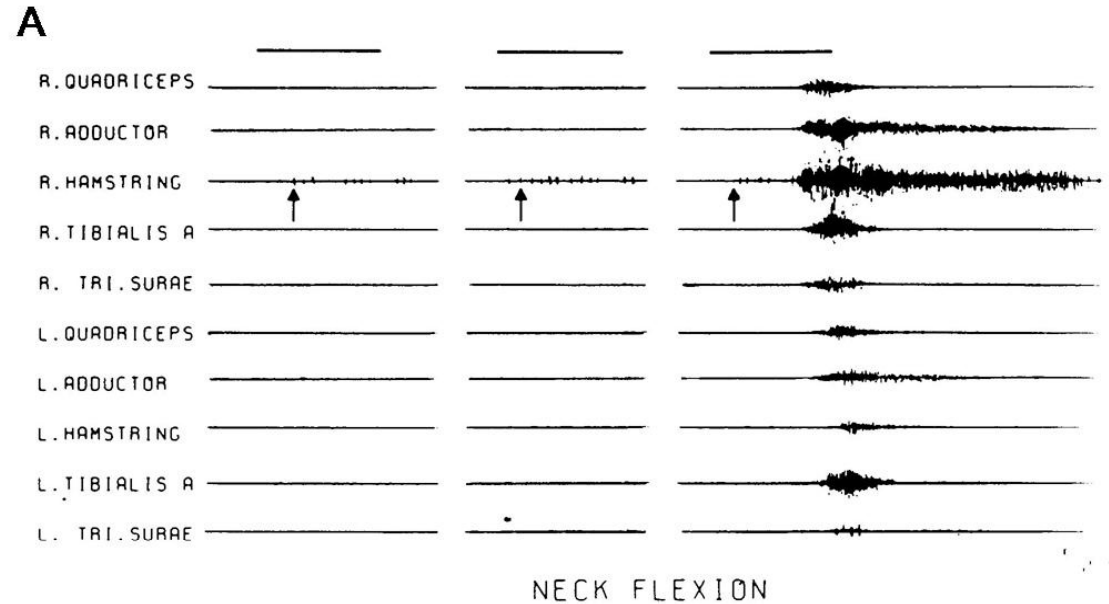
Spasticity and Spasms



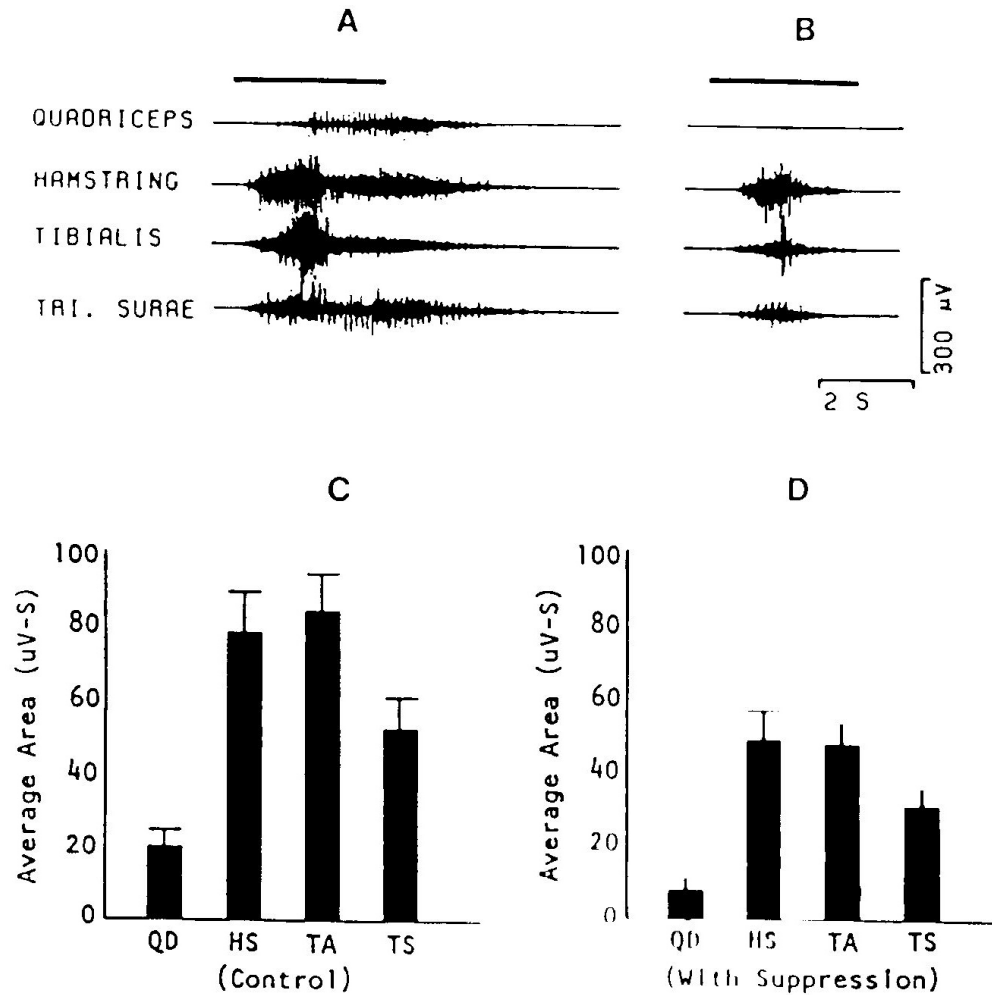
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- 2. Neurocontrol of spasticity
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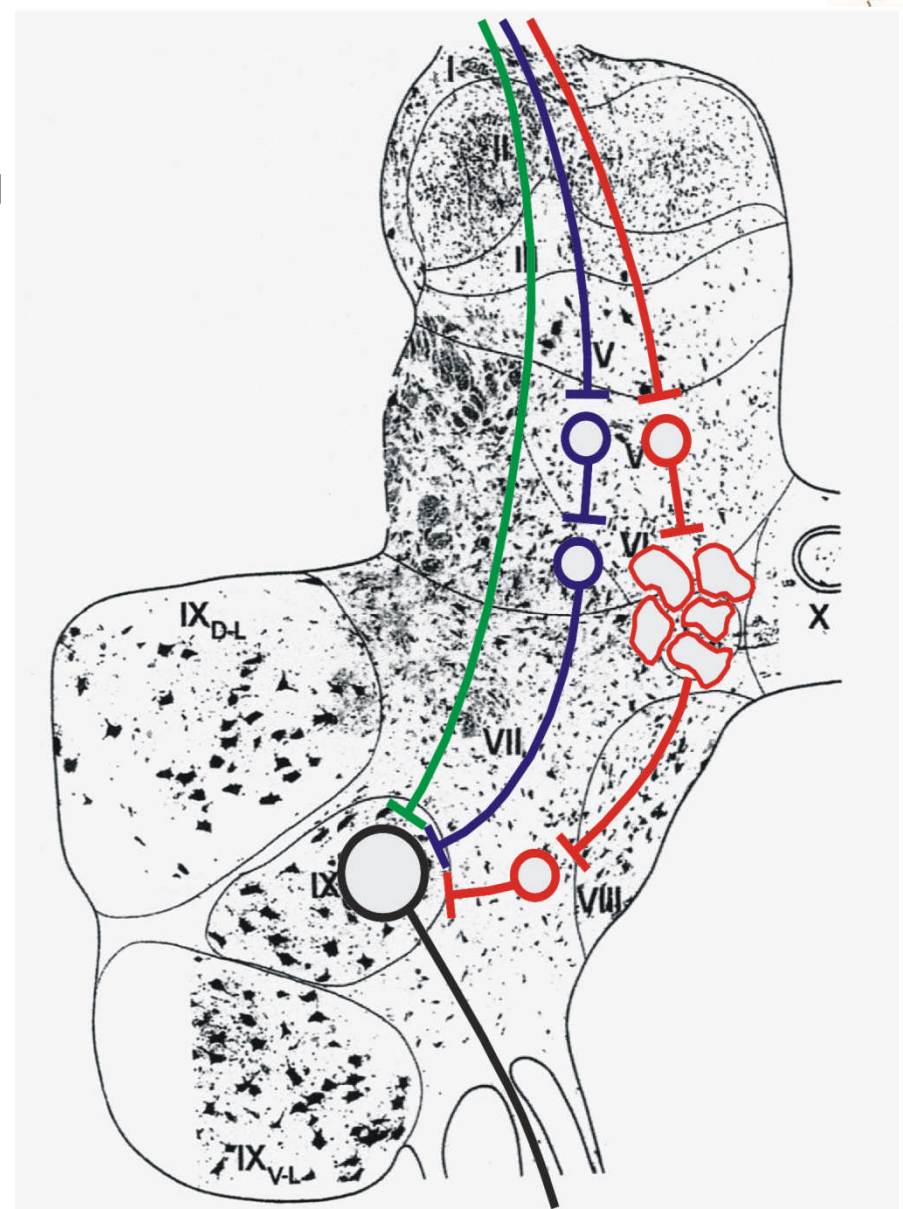
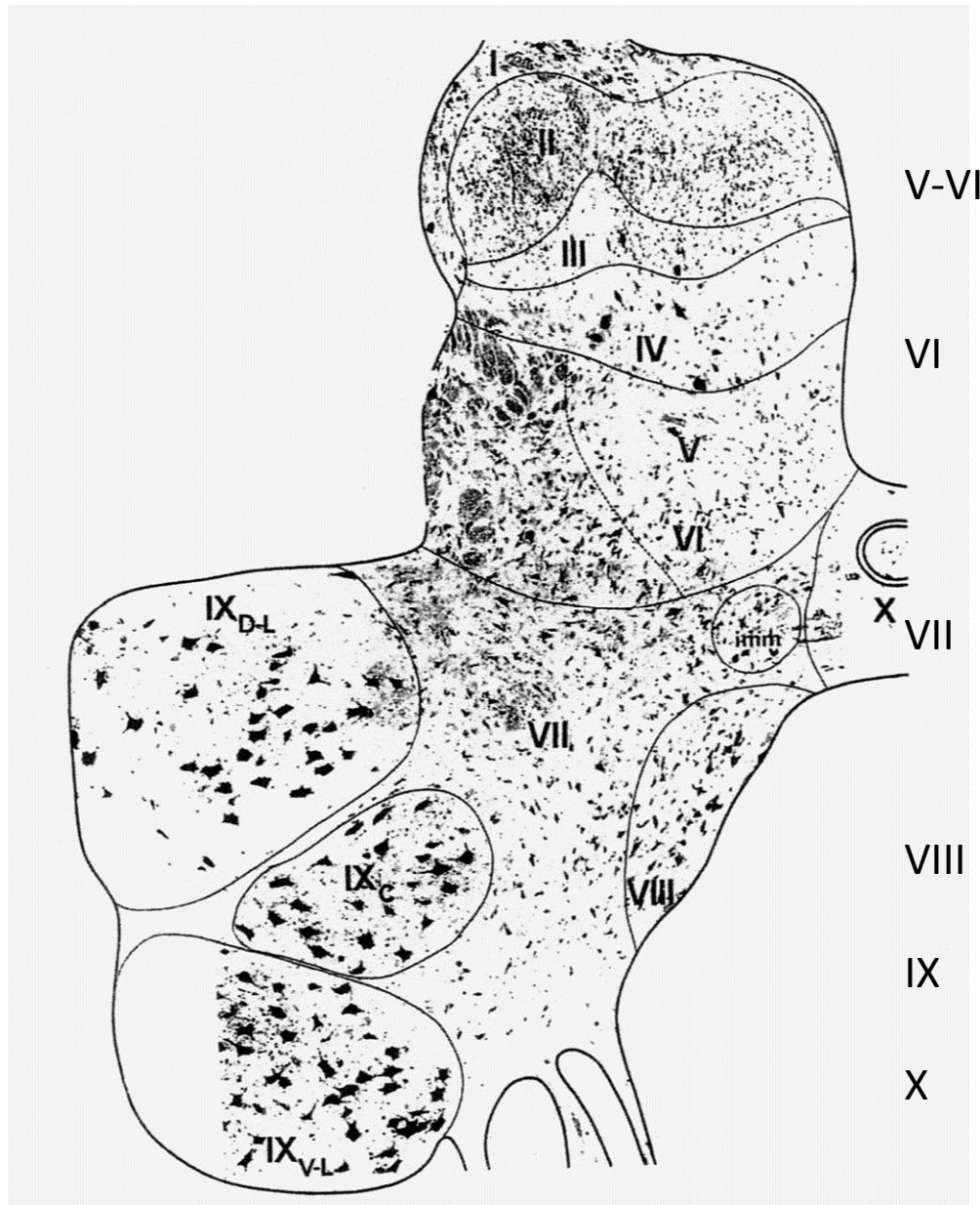
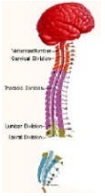
Residual Excitatory Motor Control in Clinically Paralyzed SCI



Residual Inhibitory Motor Control in Clinically Paralyzed SCI



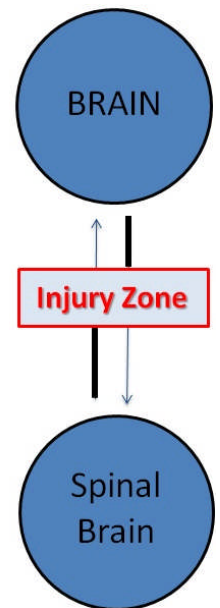
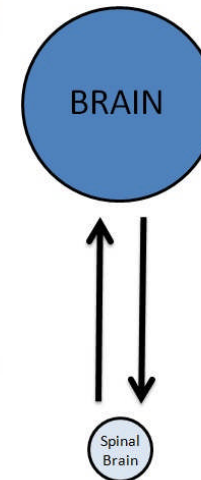
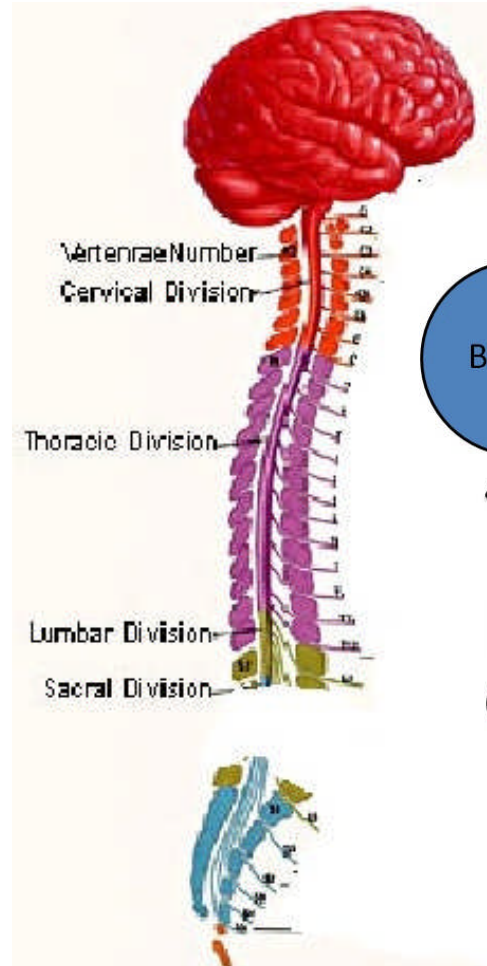
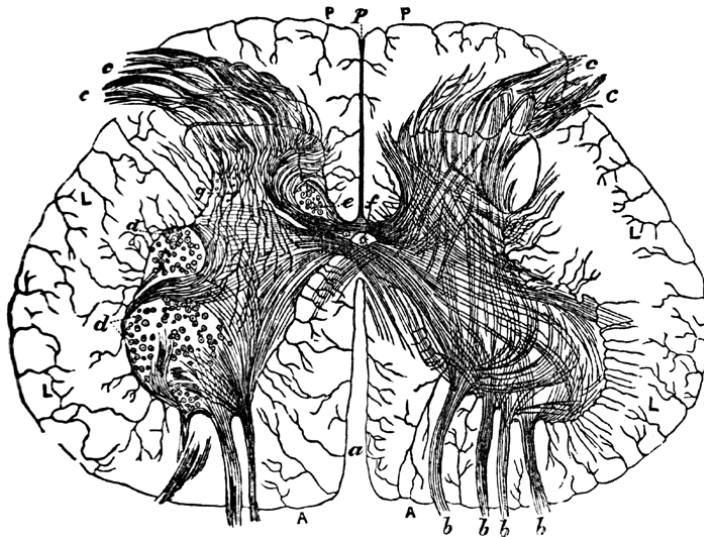
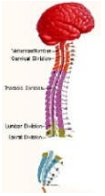
Testing segmental activity by TJ and WR



Summary of Observations

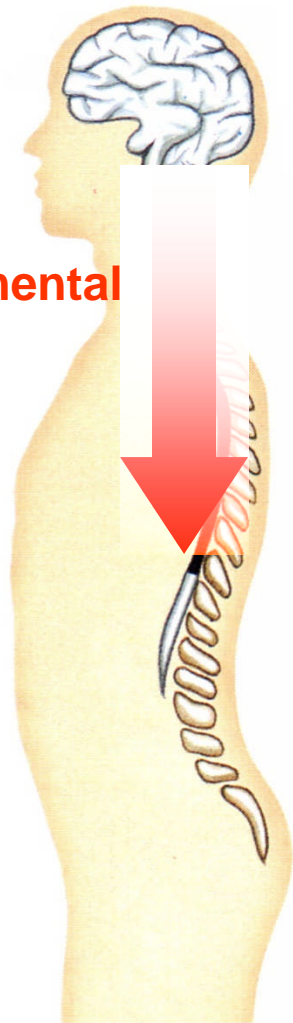
- 1. There is a neurocontrol of upper motor neuron paralysis
- 2. Neurocontrol of spasticity
- 3. There is residual brain control and influence
- 4.

From historical illustration of spinal cord network to conceptual modeling of evolution of spinal brain by severity of lesion



Mimicking brain stem control of the lumbar network

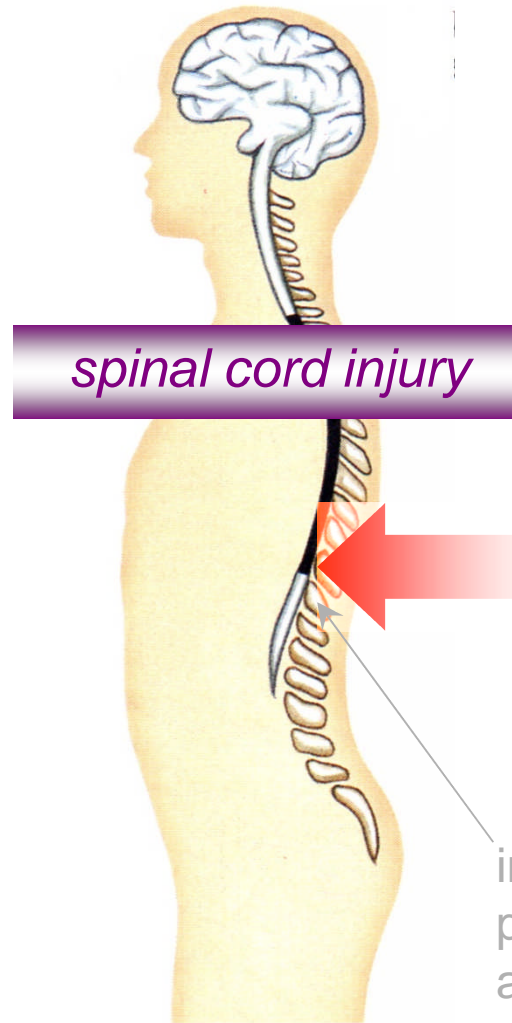
tonic
suprasegmental
drive



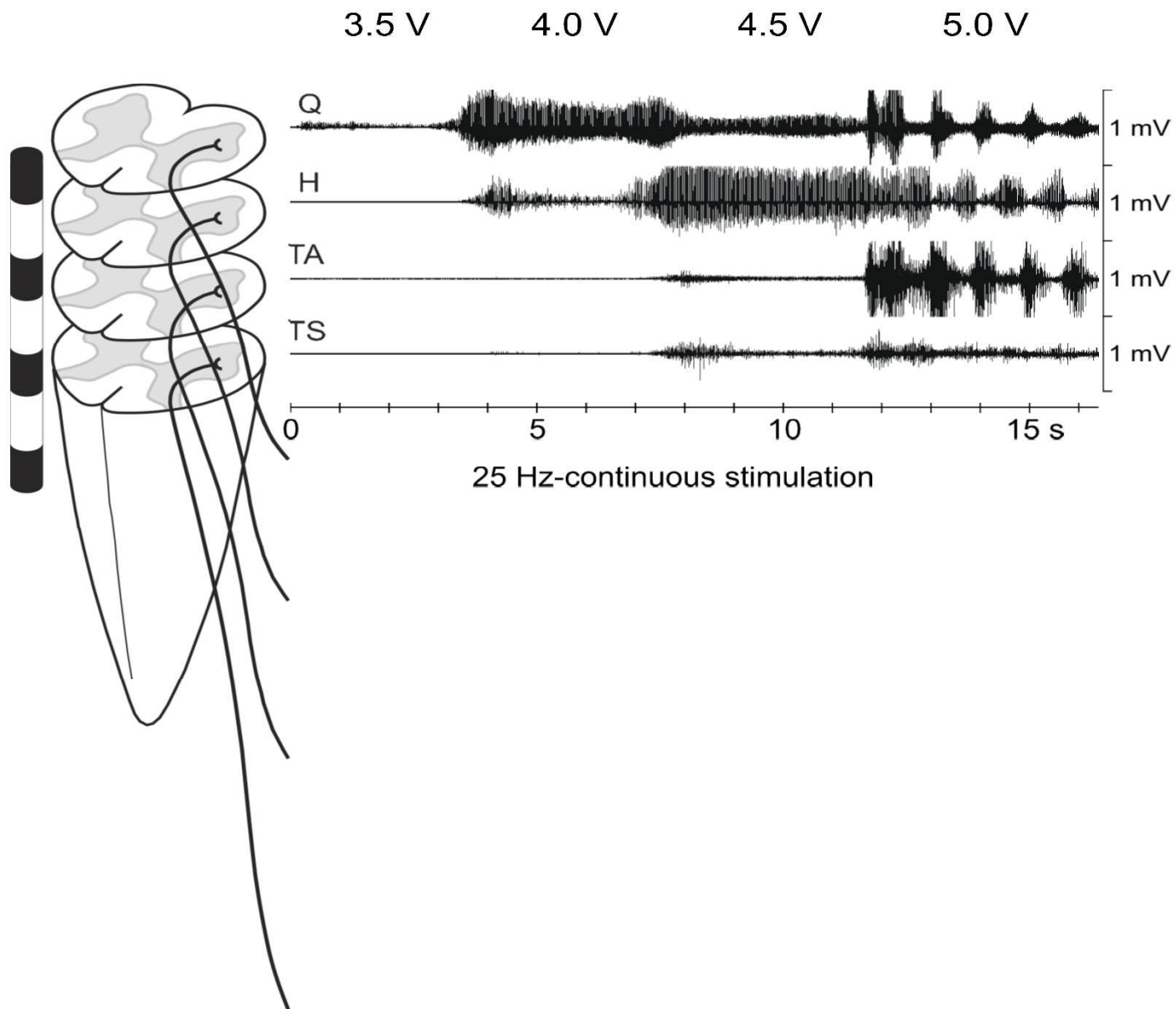
spinal cord injury

spinal cord
stimulation
(SCS)

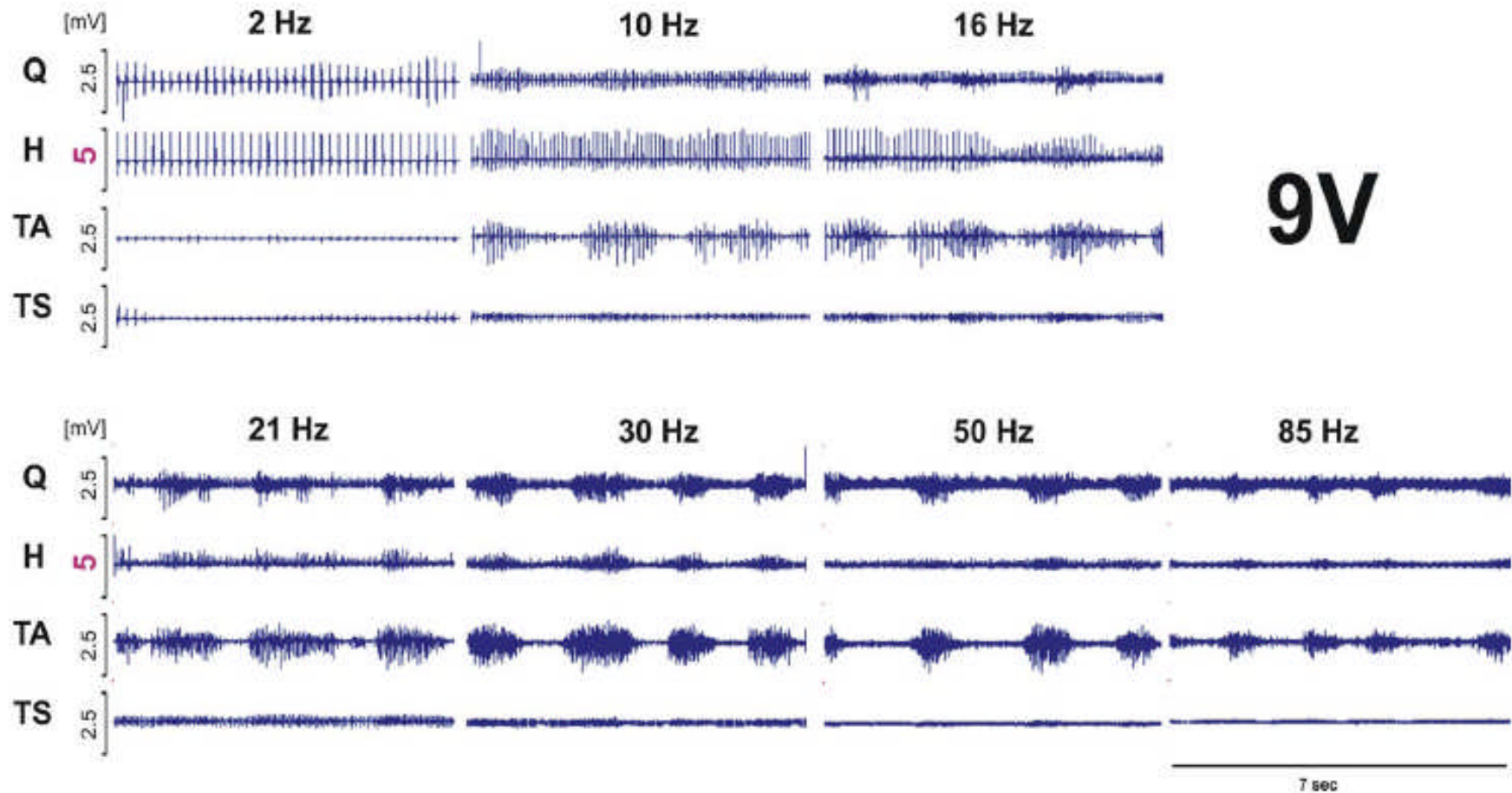
intermittent
phasic
afferent input



Effect of increased input from posterior roots to output



Constant high level of stimulation, but different frequencies



Summary of Observations

- 1. There is a neurocontrol of upper motor neuron paralysis
- 2. Neurocontrol of spasticity
- 3. There is residual brain control and influence
- 4. Lumbar cord brain



Fig. 3.

