NEURAL CONTROL OF MUSCLES DURING GAIT IN HEALTHY SUBJECTS AND IN AMBULATORY SPINAL CORD INJURY PATIENTS

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ABSTRACT

In order to outline central nervous system mechanisms, which control muscle innervation during gait, EMG activity and force basograms of 10 healthy adults (1 female, 9 males, ages 17-34) and 12 spinal cord injury patients (2 females, 10 males, age 16-58, levels C3-Th12, average onset 58 months) were studied. The patients with intact neurocontrol mechanisms, who could walk on short distances, had an impaired suprasegmental influence below the motor and sensory incomplete lesions. Tibialis anterior muscle, normally active when unloaded, was used to assess the centrally preprogrammed control, while soleus, the antigravity muscle, was a monitor for the spino-bulbar-spinal and segmental stretch reflexes. Amplified EMG signals were full wave rectified and filtered with a time constant 3 ms, which did not obscure the latency of segmental reflex after foot contact. Special force shoes provided the ground reaction forces and phases of stride. The data were sampled with 200 Hz and averaged around the foot-floor contact over at least 30 strides. EMG activity before the foot contact and in corresponding latency windows after it, was studied together with the force, showing either hypo- or hyperactivity with diminished preprogrammed control and suprasegmental reflexes in the spinal cord injury patients.

INTRODUCTION

Three CNS mechanisms were demonstrated in the neural control of muscles during gait. Central preprogrammed control, descending from the brain, depends mainly to the vestibular influences and is only related to the phase of stride (1), while a polysynaptic spino-bulbar-spinal functional stretch reflex (2) and a monosynaptic segmental stretch reflex (3, 4) are directly triggered by the contact of foot with the floor.

Neural control can be seriously altered in patients with the upper motor neurone lesions (5, 6, 7). External activation of movements interacts with the residual neurocontrol of these patients. Therefore, it became important to study the neurocontrol mechanisms of gait in addition to its biomechanical quantification. Human gait, as a repetitive sequence of movements which must also adapt itself to sudden changes, can hardly be analysed on a basis of the single stride. Therefore, computer

averaging of several EMG channels during one stride was implemented including the necessary signal conditioning.

For this purpose, an EMG system was designed (8). It enables recording and signal conditioning of 18 EMG channels for computer analysis together with the biomechanical data. The number of EMG channels were chosen to cover the main trunk and lower limb muscles and give possibility for expansion to the upper limb muscles on both sides. With recently developed extensive data acquisition and data processing software packages, EMG activity of 10 muscle groups (paraspinal, quadriceps, hamstring, tibialis anterior, and triceps surae muscles on both sides) were on-line sampled together with force shoe and force crutch data and then processed for the gait of 10 healthy adults and 12 ambulatory spinal cord injury patients.

In this paper, suprasegmental and segmental components of the neurocontrol of ankle flexors and extensors were studied during the gait of healthy adults and incomplete spinal cord injury patients. Ambulatory spinal cord injury patients were chosen as a model with intact suprasegmental and segmental mechanicms and impaired suprasegmental influence below the level of lesion.

METHODS

EMG activity and force basograms of 10 healthy adults (one female, nine males, ages 17-34) and 12 spinal cord injury patients (two females, 10 males, age 16-58, levels C3-T12, average onset 56 months) were studied. The patients, who could walk on short distances, had an impaired suprasegmental influence below the motor and sensory incomplete lesions. Tibialis anterior muscle, normally active when unloaded, was used to assess the central preprogrammed control, while soleus, the antigravity muscle, was a monitor for the suprasegmental and segmental stretch reflexes.

Amplified surface EMG signals were full wave rectified and filtered with a time constant 3 ms, which did not obscure the latency of segmental reflex after foot contact. To avoid electrical and mechanical artefacts, the EMG system was divided to small local preamplifiers with electrodes and to separated amplifiers with signal conditioners (8). Specially designed local preamplifiers (gain: 900, frequency range: 5 - 1200 Hz, noise: 6 uV) with In Vivo Metric Ag-AgCl surface electrodes on 10 cm cables and amplifiers with signal conditioners (AC coupled inputs, 5 - 1000 Hz frequency range at -3 dB point and >6 dB/okt. slope, gains 1, 2 and 5, full wave rectification and 3 ms smoothing) were used together with special force shoes, which provided the ground reaction forces and phases of stride. The data were on-line sampled with 200 samples per second and later averaged over several strides for each subject. EMG activity before the foot contact and in the corresponding latency windows after it (35 - 45 ms for the segmental stretch reflex and 120 - 180 ms for the suprasegmental functional stretch reflex), was studied together with the force basogram.

RESULTS

In healthy adults walking with their normal velocity (Figures 1, 2, 3), the tibialis anterior, as a flexor muscle, was active from the end of stance through the initial swing, decreased through the swing and abruptly increased before the heel contact, indicating the centrally preprogrammed control. It reached its peak discharge shortly after the heel contact and ended after the flat-foot.

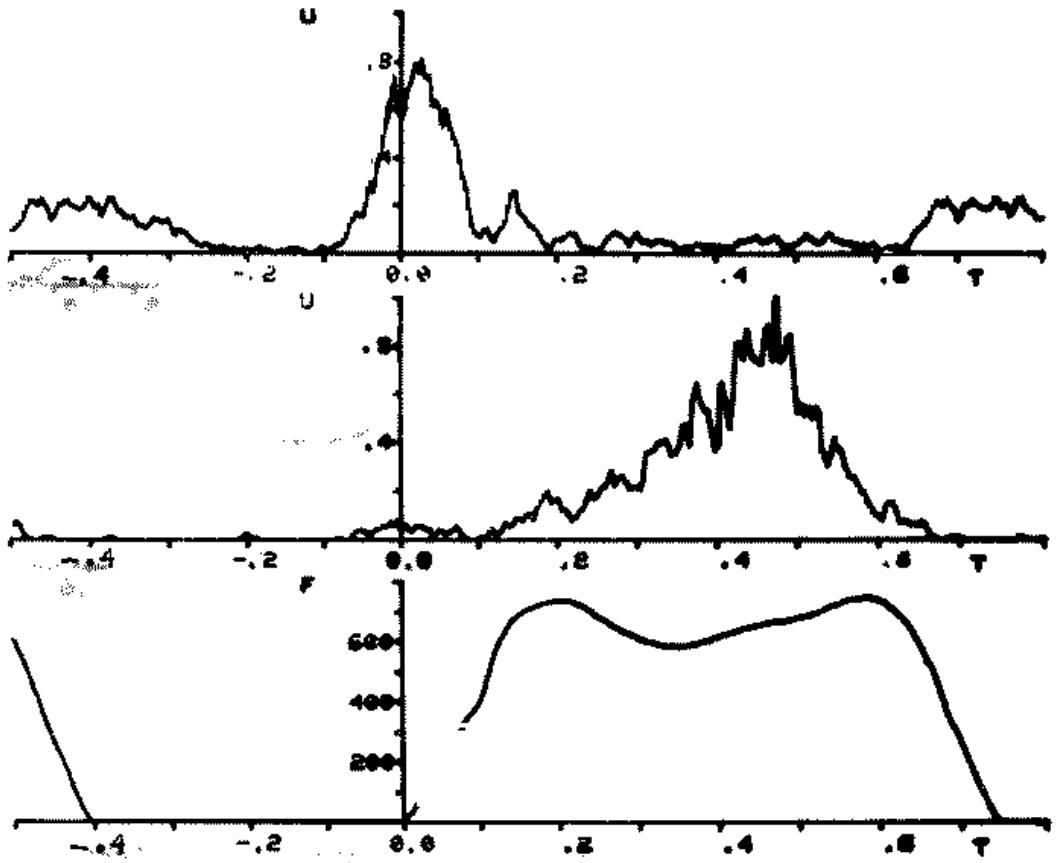

Figure 1 Average left tibialis anterior (top diagram) and left soleus activity (middle diagram) in milivolts with the average left vertical force in Newtons (lower diagram) during 33 strides of a healthy, 17 year old male walking with normal speed. Time axes are given in seconds.

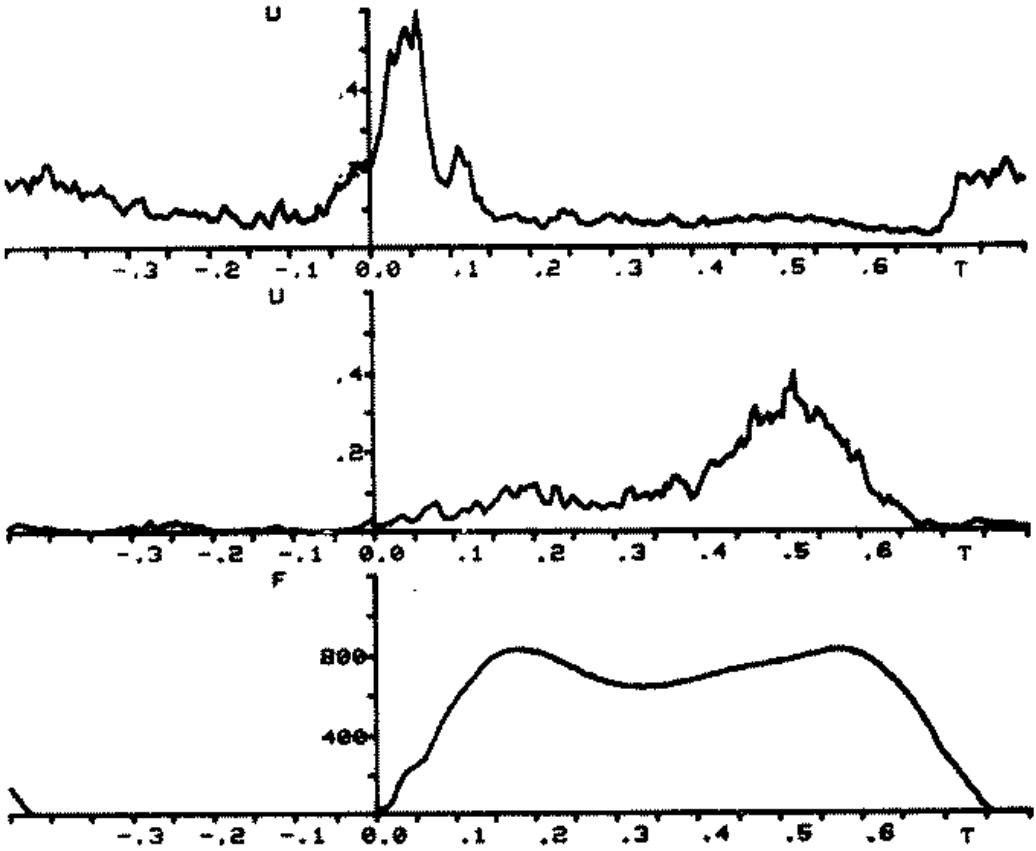

Figure 2 Average right tibialis anterior (top diagram) and right soleus activity (middle diagram) in milivolts with the average right vertical force in Newtons (lower diagram) during 36 strides of a healthy, 28 year old male walking with normal speed. Time axes are given in seconds.

Oppositely, the soleus, as an antigravity extensor muscle, was active exclusively during the single limb support in the stance phase. It started its increasing activation approximately 120 ms after the heel contact in the latency and shape of suprasegmental functional stretch reflex.

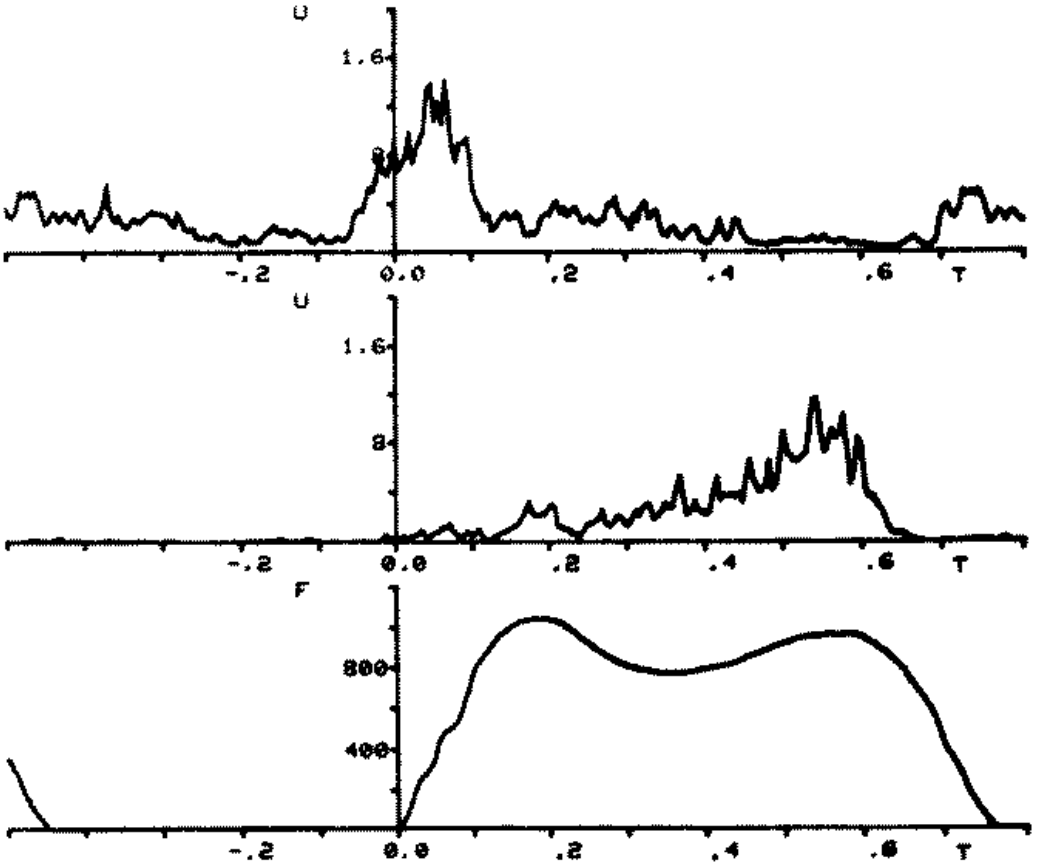

Figure 3 Average right tibialis anterior (top diagram) and right soleus activity (middle diagram) in milivolts with the average right vertical force in Newtons (lower diagram) during 28 strides of a healthy, 39 year old male walking with normal speed. Time axes are given in seconds.

There was no distinct discharge in the soleus muscle within the 35 - 45 millisecond latency window after the heel contact, which would imply the segmental stretch reflex activity in the normal speed gait of the healthy adults.

In most patients (Figures 4, 5, 6) the patterns were replaced by a diminished tibialis anterior activity in the terminal swing and low tonic coactivation of both muscles, starting before the foot-floor contact.

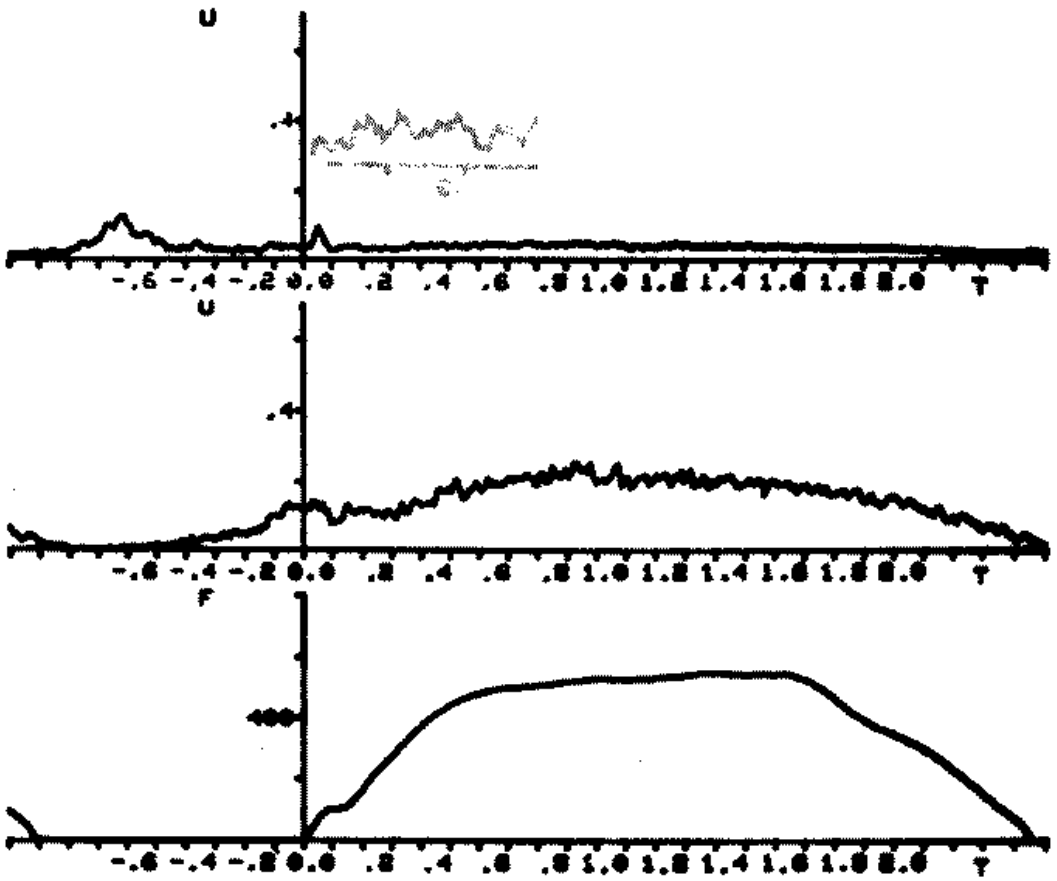

Figure 4 Average right tibialis anterior (top diagram) and right soleus activity (middle diagram) in milivolts with the average right vertical force in Newtons (lower diagram) during 30 strides of an 18 year old, C5-C6 incomplete spinal cord injury female 21 months post onset of injury, who was walking with her chosen speed. Time axes are given in seconds.

The patient from Fig. 4 displayed low tonic activity of both muscles, with only slight peak of the tibialis anterior in the mid- swing and soleus activation starting before the heel contact, without evidence of the reflex activity

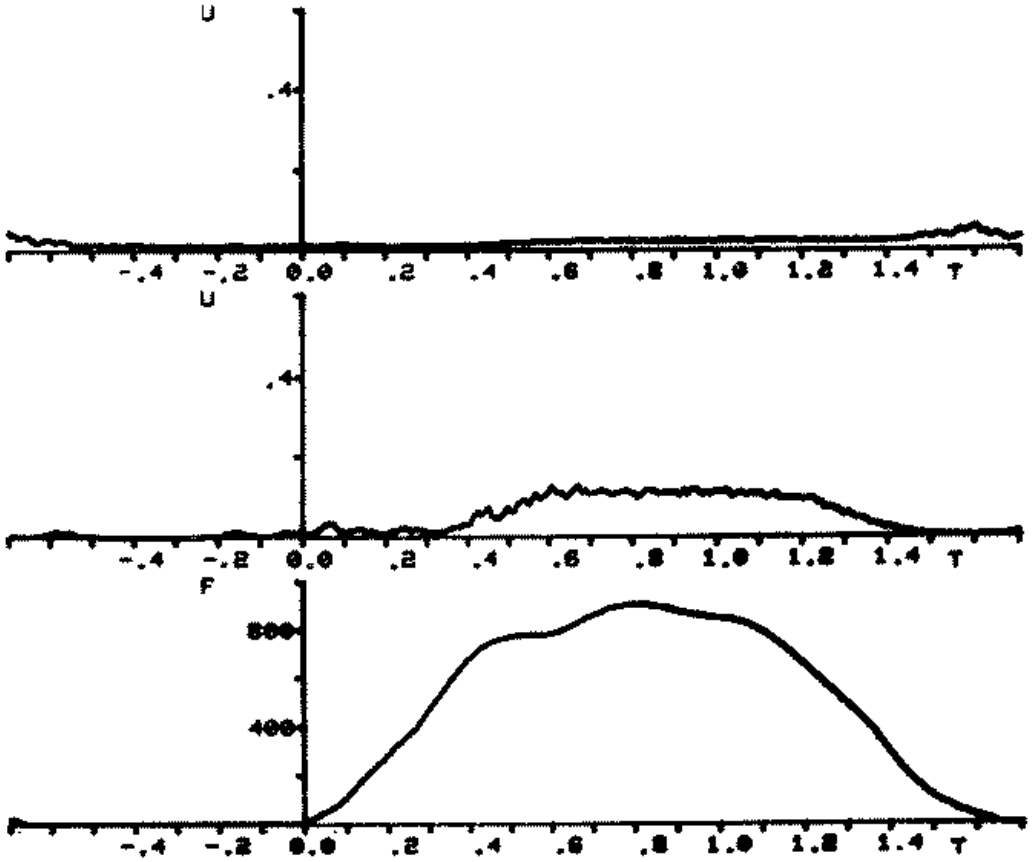

Figure 5 **Average** right tibialis **anterior** **(top** diagram) and right soleus activity (middle diagram) in milivolts with the average right vertical force in Newtons (lower diagram) during 39 strides of a 42 year old, C3-C4 incomplete spinal cord injury male 6 years **post** onset of injury, who was walking with **his chosen speed. Time axes are given in seconds.**

In the patient from Fig. **5,** activity of **the tibialis anterior** muscle was practically absent throughout the **entire** stride period, with a very slight discharge at the **terminal stance** and initial swing. The soleus muscle displayed **low tonic** activation during the single limb support, however, there were no **distinct** change either in **the** 35 - 45 ms or 120 - 180 ms **latency windows. All neurocontrol components were drastically suppressed.**

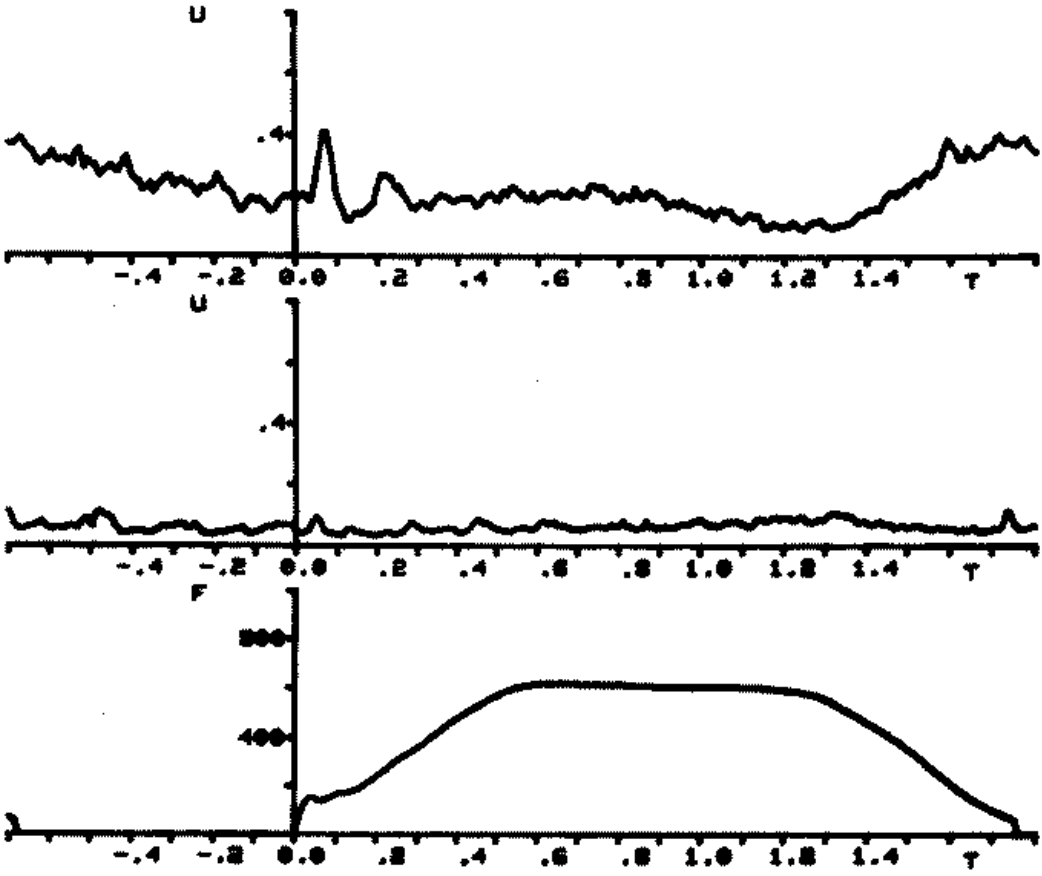

Figure 6 **Average left tibialis anterior (top diagram)** and left soleus activity (middle diagram) in milivolts with the average left vertical force in Newtons (lower diagram) during 29 strides of a 28 year old, C5-C6 incomplete spinal cord injury male 7 months post onset of injury, who was walking with his chosen speed. Time axes are given in seconds.

The patient from Fig. 6, was recorded only 7 months after injury. He also displayed a sustained tonic coactivation in both the tibialis anterior and soleus muscles. However, **the activity** was stronger in the tibialis anterior, with a moderate increase **in the** terminal stance. There was no increase prior to the heel contact, indicating the centrally preprogrammed control. **The** soleus activity did not change during the entire stride, **giving so no** evidence of either segmental or suprasegmental reflexes.

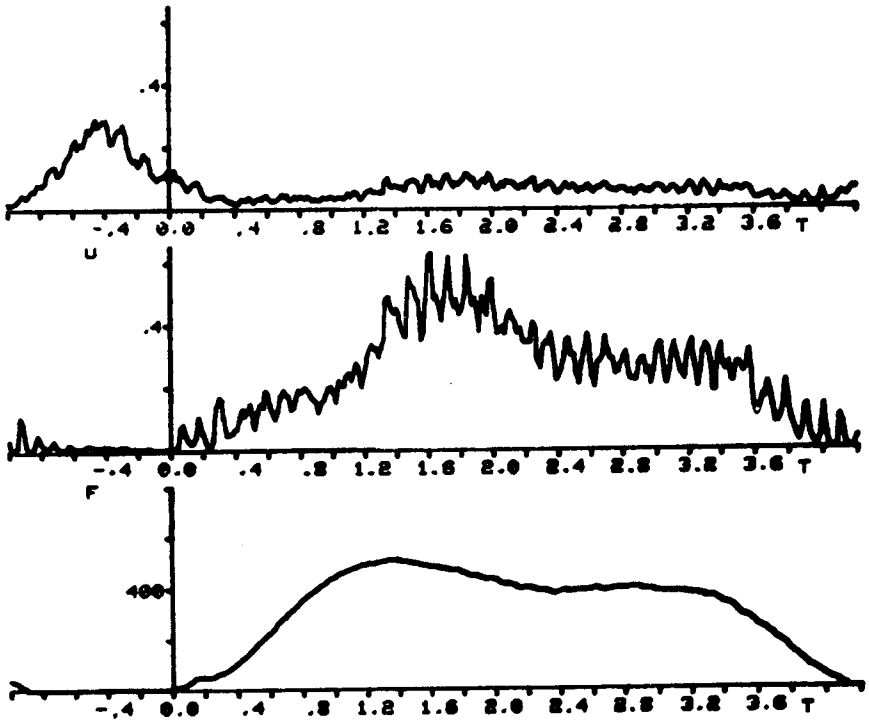

Figure 7 Average right tibialis anterior (top diagram) and right soleus activity (middle diagram) in milivolts with the average right vertical force in Newtons (lower diagram) during 26 strides of a 26 year old, T4-T5-T6 incomplete spinal cord injury male 8 years post onset of injury, who was walking with his chosen speed. Time axes are given in seconds.

Some partients showed partly increased tonic activity, as the one from Fig. 7. In this patient, both the tibialis anterior and soleus patterns were shifted towards the swing phase. There was an evidence of diminished central preprogrammed control in the tibialis anterior and of the suprasegmental reflex with superimposed clonogenic bursts of partly released segmental activity.

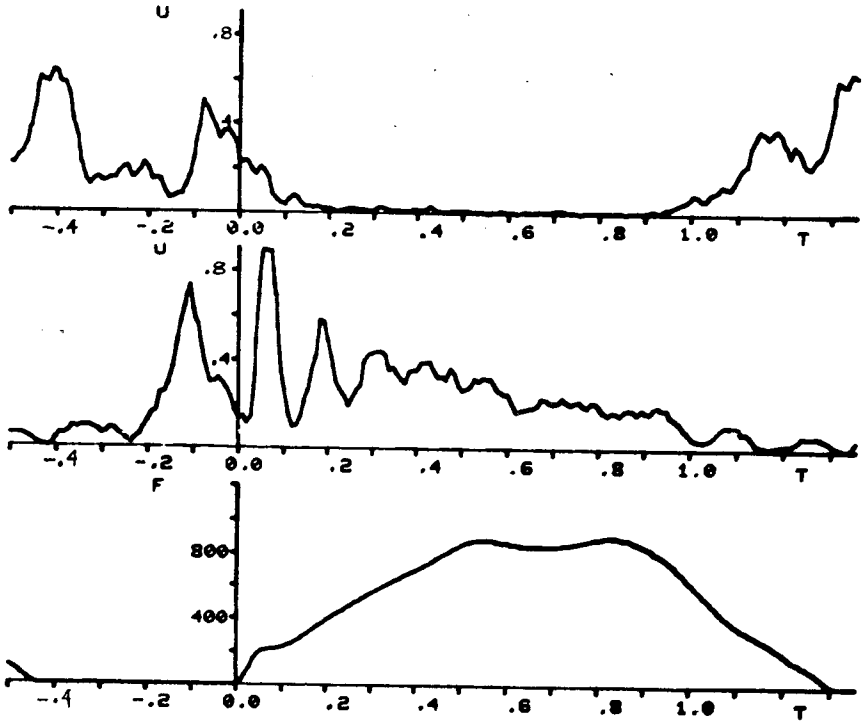

Figure 8 Average left tibialis anterior (top diagram) and left soleus activity (middle diagram) in milivolts with the average left vertical force in Newtons (lower diagram) during 49 strides of a 46 year old, T12 incomplete spinal cord injury male 6.5 years post onset of injury, who was walking with his chosen speed. Time axes are given in seconds.

Another patient (Fig. 8) showed an increased tonic tibialis anterior activity in the initial and terminal swing, the latter partly implying the central preprogrammed control. However, the simultaneous coactivation of soleus muscle before the foot-floor contact showed its alteration. Initially strong superimposed clonogenic bursts in the soleus, which decreased after the flat foot, partly evidenced a presence of segmental stretch reflex activity. There was no obvious suprasegmental reflex control.

CONCLUSION

The purpose of this study was to compare healthy and impaired neurocontrol. In ambulatory spinal cord injury patients, the gait is influenced by the segmental and impaired suprasegmental control. Such patients could either mainly have used a released segmental stretch reflex or the residual suprasegmental control, diminished by the segmental reflex activity. In the observed patients, the presumed released segmental activity did not prevail, the suprasegmental functional stretch reflex was suppressed, while the diminished preprogrammed mechanisms from the brain mainly controlled their severely disabled activity during ambulation.

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