# Proton Magnetic Resonance Spectroscopic Changes of the Primary Motor Cortex and Supplementary Motor Area in Hemiparetic Patients with Corticospinal Tract Injury due to Deep Intracerebral Hematoma

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## ABSTRACT

This study was conducted to investigate the metabolic changes in the motor and motor association cortices following axonal injury in the internal capsule that was caused by deep intracerebral hematoma. Using 1H MRS, the authors studied the primary motor cortices (M-1) and supplementary motor areas (SMA) of 9 hemiparetic patients with documentable hemiparesis of varying severity. To measure the M-1 and SMA biochemical changes, 4 separate single volumes of interest (VOIs) were located bilaterally in the affected and unaffected hemisphere (AH and UH). The M-1/SMA NAA/Cr ratios of the AH and UH in patients, and the AH and normal volunteers were compared. The NAA/Cr ratios of the M-1 and SMA in AH, and the SMA in UH were significantly lower than those of normal volunteers. These 1H MRS findings indicate that axonal injury in the descending motor pathway at the level of internal capsule could induce metabolic changes in the higher centers of the motor pathway.

## INTRODUCTION

Early studies of 1H MRS in stroke have mostly investigated ischemic stroke, and they have shown an increased lactate and decreased NAA within the stroke lesion [1]. However, attempts to determine whether the magnitude of neuronal damage, as measured by NAA loss, correlates with the disability and impairment in ischemic stroke patients have brought forth inconsistent results [2]. There has been no previous 1H MRS study that has investigated the metabolic changes in the higher motor cortex following intracerebral hematoma in hemiparetic humans. The aim of this study was to evaluate the local metabolic changes for the primary motor cortex (M-1) and supplementary motor area (SMA) in the affected hemisphere (AH) and unaffected hemisphere (UH), according to their axonal injuries at the level of the internal capsule.

## MATERIALS AND METHODS

The M-1/SMA of the AH and the UH were studied using 1H MRS on 9 patients (4 men and 5 women; mean age 54 yr, range 30-70 yr, right-handed) with documentable hemiparesis of varying severity. In all cases, the hemiparesis was caused by deep intracerebral hematoma in the putamen and the thalamus because these locations are directly adjacent to the internal capsule, and an intracerebral hematoma in these areas mostly brings about hemiparesis as a neurologic sequele. The 1H MRS study was performed on alert patients with definite hemiparesis of the extremities contralateral to the AH. To control the possible 1H MRS spectral change, we excluded those patients that had undergone any surgical intervention or if they had a major systemic illness, such as uremia. The results were compared with 1H MRS studies performed on 10 normal control volunteers. The mean duration of the study was approximately 1 yr, with a range of 7 days to 1 yr after development of intracerebral hematoma.

## RESULTS

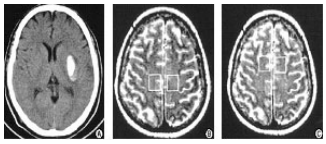

*Fig.1. CT scan of a hemiparetic patient. Voxel site for 1H MRS acquisition in the primary motor cortex (M-1) and supplementary motor area (SMA).*

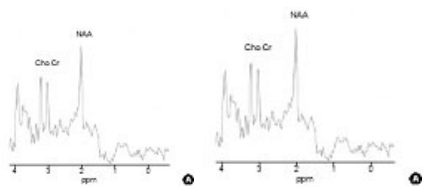


*Fig 2. Typical spectra of 1H MRS in the chosen voxel. obtained from the M-1 cortex in the affected and unaffected hemispheres. (A) M-1 in affected hemisphere. (B) M-1 in unaffected hemisphere.*

The NAA/Cr ratios of M-1 in the AH and UH were 1.08±0.12, 1.50±0.17, respectively. There were significant differences between the AH and UH in the NAA/Cr ratios of M-1 ($p<0.05$). The NAA/Cr ratios of M-1 measured in the AH and controls were 1.08±0.12, 1.37±0.12, respectively. There was also significant differences between the AH and controls in the NAA/Cr ratios of M-1 ($p=0.01$). However, the NAA/Cr ratios of M-1 measured in the UH showed no differences when compared with the ratios of normal volunteers ($p=0.115$). These results showed that the NAA/Cr ratios of the AH were significantly decreased when compared with those of the UH and normal volunteers and therefore, the metabolic changes had occurred in the high cortical region ipsilateral to the injured hemispheres. However, the NAA/Cr ratios of M1 in the AH showed no significant correlation with the severity of arm weakness, leg weakness, or the duration of hemiparesis. The NAA/Cr ratios of the SMA measured in the AH and UH were 1.18±0.15, 1.18±0.27, respectively. There was no difference between the AH and UH in the NAA/Cr ratios of the SMA ($p=0.980$). However, when compared with the SMA of the controls, the NAA/Cr ratios of SMA in the AH and UH showed significant differences ($p<0.05$). In terms of Cho/Cr ratios, no significant differences were found between the following; the M1 cortices of the AH and UH ($p=0.316$), the SMAs of the AH and UH ($p=0.561$), the M-1 cortex and SMA of the AH ($p=0.745$), and the M-1 cortex and SMA of the UH ($p=0.289$). No significant differences were found when comparing the Cho/Cr ratios of each cortex with those of the normal volunteers. The NAA/Cr and Cho/Cr ratios of the M1 and SMA in the control group did not show any difference between right and left hemispheres.

## DISCUSSION

In summary, we sought to determine whether 1H MRS is able to detect the metabolic changes of the M-1 and SMA that follow pyramidal tract injury after intracerebral hemorrhage. We found that the M-1 in the AH has lower NAA/Cr values than in the M-1 in the UH and in normal volunteers. In addition, the SMA also showed bilateral lower NAA/Cr values in both the AH and UH than in normal volunteers. Though we cannot precisely document the exact meaning of these lower NAA/Cr ratios, a presumed retrograde degeneration or functional deactivation may play a role in these NAA losses in M-1 and SMA due to motor tract injury. Further experiments should include longitudinal studies of patients with motor deficit after intracerebral hematomas, and whether there is a reversible component to this NAA loss.

## REFERENCES

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