Reorganization of the Cortical Control of Movement due to Radiation Necrosis: Evidence from fMRI of the Supplementary Motor Area.

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Introduction:

Recent publications have demonstrated neural plasticity and reorganization in strokes, AVM's and tumors (1). The study of cortical reorganization in the brain directly invaded by a pathological process is complicated by physiological factors such as morphologically and functionally abnormal blood vessels, which limits the accuracy of fMRI (2, 3). In terms of reorganization of the cortical control of movement, this limitation of fMRI can be overcome by analyzing the supplementary motor area (SMA) which is functionally related to motor control, yet, anatomically far removed from the primary motor cortex (PMC). Therefore, the accuracy of fMRI of the SMA will not be affected in a pathological process that invades the PMC, but not the SMA. This approach has successfully demonstrated cortical reorganization in tumor invasion of the PMC (4, 5). In the current report, we demonstrate cortical reorganization of motor control due to radiation necrosis.

Method/Materials:

A 63-year-old man had a left convexity atypical meningioma resected three years prior. Following focal radiation and stereotactic radiosurgery, the patient developed enhancement and edema in the right (contralateral) parietal lobe, including the post-central gyrus (sensory cortex) and extending into the precentral gyrus (PMC) but not into the expected location of the SMA. The differential diagnosis was disease progression vs radiation necrosis. The patient had numbness and minimal clumsiness, but no weakness in the left hand. Pre-operative routine MR and BOLD fMRI scans using a finger tapping paradigm (gradient echo EPI, TR/TE =4000/60 msec, 21 slices, 128x128 matrix) were obtained on a 1.5T scanner. fMRI data was analyzed using AFNI and co-registered to the T1-weight images. The volumes of activation were determined for both the PMC and SMA for multiple thresholds (cross-correlation-R values).

Results:

Pathology demonstrated radiation necrosis. The volume of activation in the SMA was greater on the side with the radiation necrosis for all r-values tested ($p < 0.017$) (Fig 1, 2 and 3). There was no significant difference in the volume of activation of the PMC between the two sides (Fig. 1).

Discussion:

These findings appear to support the contention that as radiation necrosis damages the PMC, the SMA takes on a more active role in the cortical control of movement. The SMA is important for the planning and execution of movements even when the tasks are simple. The SMA is also associated with the initiation of movement, motor programming and planning, readiness to move, motor learning, complexity of the movement, and responsiveness to internal cueing of movements. fMRI studies in normals have shown that the volume of activation in the SMA during bilateral tasks is almost identical to those during a unilateral movement (6). Therefore, in the normal setting, both sides of the SMA appear equally involved in unilateral hand movement. Since our results demonstrated an asymmetry in the SMA's, the finding in normals gives further credence that reorganization of the cortical control of movement involving the SMA has occurred. What remains unclear is the exact role of the SMA. Is there increased activation because one needs more "planning" from the SMA to achieve the same movement or is SMA taking on the function of the PMC? Work on non-human primates suggests that the SMA may actually assume some of the function of the PMC (7, 8).

Conclusion:

As radiation necrosis damages the PMC, there is fMRI evidence for increased activation in the SMA implying reorganization of the cortical control of movement. This appears to occur in a manner similar to the consequences of invasion of the PMC by glial tumors.

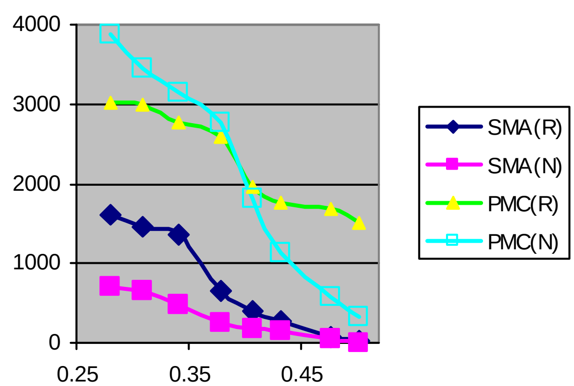

Fig. 1: Volume of activation (mm) vs r-values

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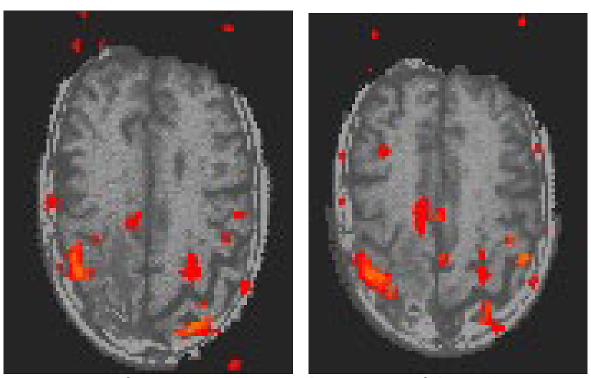

Fig.2: BOLD fMRI shows increased activation of the right SMA.

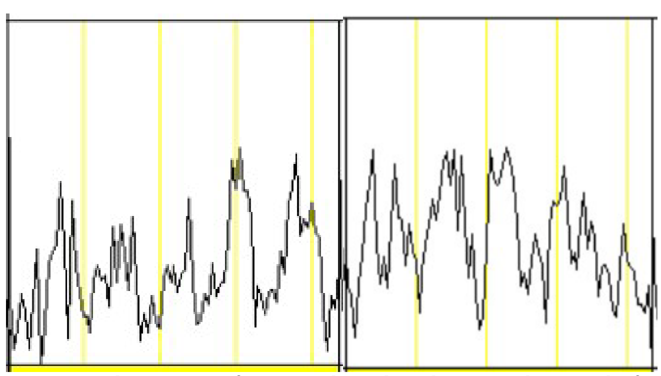

Fig.3: Signal-time curves from typical voxels in the SMAs shown in the fig. 2.