

Panel: Unusual Aphasias: Subcortical Aphasia

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The notion of "subcortical" aphasia, i.e., aphasia related to lesions demonstrated by C.T. scan in the basal ganglia, thalamus, internal or external capsules has gained wide acceptance (Basso, Sala and Farabola, 1987). While the evidence for thalamic lesions as a cause of speech disturbances is substantial (Crosson, 1984), the evidence that lesions in these other deep structures can cause aphasia is far less compelling. Particularly noteworthy is the paucity of clinico-pathological correlation: there is only one pathologically verified case (Barat *et al.*, 1981) of aphasia in association with an isolated lesion in the basal ganglia. A recent experience with a case of "subcortical" aphasia led us to review the clinical and radiological evidence for this entity.

REPORT OF CASE

The patient was a 56-year-old Filipino man in previously good health, except for treated hypertension. He had learned English in grammar school, has served for 20 years in the U.S. Navy, and was presently working as a janitor. He wrote with his right hand, but used his left hand primarily in work, threw a ball with his left hand and kicked a ball with his left foot. On the afternoon of admission he suddenly developed right arm weakness and numbness quickly followed by weakness and numbness in both legs. He fell without losing consciousness. He noted that his speech was slurred and he had trouble finding words. Within 30 minutes his strength had returned, but he continued to suffer from numbness of the right hand and difficulty finding words.

On examination in the emergency room, the patient was alert and without evident weakness or reflex asymmetry. There was a mild sensory deficit, without neglect or apraxia, in the right hand. The patient's cranial nerves were intact. His speech was slow. He had obvious difficulty finding words, difficulty naming, and made frequent paraphasic errors. His ability to repeat spoken words was poor and he could perform only single-step commands well.

The next morning, the patient was noted to have severe right-left confusion without finger agnosia. He could name some pictured items. He was able to read written commands but could not perform them. He could repeat short sentences, but failed on longer ones. He could print his name and address, and draw circles and squares. His performance on complex commands and mental arithmetic was poor.

A C.T. scan demonstrated focal areas of diminished attenuation in the posterior limb of the internal capsule, lenticular nucleus, corona radiata, posterior periventricular white matter and the subcortical white matter in the posterior parietal lobe, all on the left side. The cortex appeared to be spared. A cerebral arteriogram revealed an occlusion of the left middle cerebral artery within the M-1 segment just distal to the origins of the anterior and lenticulostriate arteries. The Sylvian vessels on the left filled slowly via retrograde collaterals from the anterior cerebral artery. A <sup>133</sup>xenon blood flow study showed grossly diminished blood flow to the

left parietal cortex as well as subcortical areas on the left. An EEG performed 11 days after the patient's stroke revealed prominent slowing over the left hemisphere with a delta focus in the left temporal region.

The patient's speech improved steadily in the hospital. Two weeks after admission he received an overall severity rating of 3 on the Boston Diagnostic Aphasia Examination (BDAE; Goodglass and Kaplan, 1976). His major deficit was in auditory comprehension, with a Z score of -.5. He continued to be troubled with right-left confusion and difficulty remembering longer sentences.

#### CONCLUSIONS

This case and those described by Olsen et al. (1983, 1986) demonstrate that aphasias in association with lesions which appear to be confined to the deep subcortical structures are in fact often also associated with profound abnormalities in cerebral blood flow to the cortical speech areas. These abnormalities cannot be explained by the very mild decrements in cortical metabolism described by Metter et al. (1983) in patients with subcortical lesions. Partial ischemia of the cortex can result in functional impairment without demonstrable morphological damage (Heiss, 1983), resulting in an "ischemic penumbra" (Olsen, 1986) around a cerebral infarction. This penumbra is highly variable, depending on the extent of collateral blood flow, and tends to quickly shrink as collateral blood vessels expand to accommodate increased flow. These characteristics thus explain the variability (Basso et al., 1987) and usual rapid recovery (Damasio et al., 1982) of "subcortical" aphasias.

"Subcortical" aphasia was first established as a distinct clinical entity in two simultaneous reports (Naeser et al., 1982 and Damasio et al., 1982). In the 9 cases reported by Naeser only 1 patient underwent arteriography: that patient had a complete occlusion of the left internal carotid artery. Of the 11 cases reported by Damasio, 1 patient suffered his aphasia after trauma to the left carotid artery. No vascular studies were reported for the other cases, but this group later reported on 2 cases (one of whom was aphasic) of proximal middle cerebral artery occlusion with resultant subcortical infarction (Adams et al., 1983). The remaining case reports demonstrating "subcortical" aphasia (see Basso et al., 1987 for a summary) have all relied on C.T. scans for clinico-pathological correlation.

Occlusion of the internal carotid or middle cerebral arteries may cause watershed infarctions in the basal ganglia and deep white matter visible on C.T. scan, but are also often associated with cortical infarctions which are difficult to detect with routine C.T. sections (Wodarz, 1980). Moreover, the absence of visible cortical infarction on C.T. scan does not guarantee cerebral blood flow adequate for normal functioning. Olsen et al. (1986) reported on 18 cases with subcortical infarctions, of which 8 were aphasic. The aphasic patients all demonstrated substantially decreased cerebral blood flow to the cortical speech areas, while the nonaphasic patients had normal cerebral blood flow to these areas.

"Subcortical" aphasia may exist, but if so it is probably quite rare. To establish it as a clinical entity will require evidence that cortex which appears normal on C.T. scan is indeed adequately perfused to permit normal function. In the absence of such data, it is premature to revise our theories of the anatomy of language.

## REFERENCES

- Adams, H.P., Damasio, H.C., Putnam, S.F., and Damasio, A.R. Middle cerebral artery occlusion as a cause of isolated subcortical infarction. Stroke, 14, 948-952, 1983.
- Barat, M., Mazaux, J.M., Bioulac, B., Giroire, J.M., Vital, C.L., and Arne, L. Troubles du langage de type aphasique et lesions putamino-caudees. Revue Neurologique (Paris) 137, 343-356, 1981.
- Basso, A., Della Salla, S., and Farabola, M. Aphasia arising from purely deep lesions. Cortex, 23, 29-44, 1987.
- Crosson, B. Role of the dominant thalamus in language: A review. Psychological Bulletin, 3, 491-517, 1984.
- Damasio, A.R., Damasio, H., Rizzo, M., Varney, N., and Gersh, F. Aphasia with nonhemorrhagic lesions in the basal ganglia and internal capsule. Archives of Neurology, 39, 15-20, 1982.
- Goodglass, H. and Kaplan, E. The Assessment of Aphasia and Related Disorders. Philadelphia, PA: Lea and Febiger, 1976.
- Metter, E.J., Riege, W.H., Hanson, W.R., Kuhl, D.E., Phelps, M.E., Squire, L.R., Wasterlain, C.G., and Benson, D.F. Comparison of metabolic rates, language and memory in subcortical aphasias. Brain and Language, 19, 33-47, 1983.
- Olsen, T.S., Larsen, B., Herning, M., Skriver, E.B., and Lassen, N.A. Blood flow and vascular reactivity in collaterally perfused brain tissue. Stroke, 14, 332-341, 1983.
- Olsen, T.S., Bruhn, P., and Oberg R.G.E. Cortical hypoperfusion as a possible cause of subcortical aphasia. Brain, 109, 393-410, 1986.
- Wodarz, R. Watershed infarctions and computed tomography. A topographical study in cases with stenosis or occlusion of the carotid artery. Neuroradiology, 19, 245-248, 1980.