

Mean plasma DOPA and ventricular HVA in 4 patients with advanced Parkinson's disease. Note the small rise in CSF HVA at 4 hours.

tion found in our patients probably reflects chronic levodopa therapy and pretreatment with carbidopa.

References

- Moussa Z, Raftopoulos C, Przedborski S, Hildebrand J. Lack of changes in ventricular cerebrospinal fluid concentrations of homovanillic acid following acute challenge with levodopa. Ann Neurol 1992;31:113–114
- Olanow CW, Gauger LL, Cedarbaum JM. Temporal relationships between plasma and cerebrospinal fluid pharmacokinetics of levodopa and clinical effect in Parkinson's disease. Ann Neurol 1991;29:556–559

Acquired Selective Signature Dysgraphia

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A case of acquired dysgraphia is reported which selectively affected signature performance but not other writing abilities. A 35-year-old right-handed man suffered a head injury in a street assault, resulting in multiple skull fractures with bone splinters in the left parietal area. The neurological examination was entirely normal at that time, except for a 3-cm compression without significant parenchymal damage at the parietal lobe, as revealed by a computed tomographic scan (Fig, A). The bone fragments were removed surgically and replaced by an acrylic plate. A small epidural hematoma was appreciated during surgery. Shortly after discharge, when the neurological examination was normal, the patient reported a selective but severe signature dysgraphia when attempting to sign checks, which were refused by his banks (Fig, B). He therefore had to change his signature for all bank accounts and identification documents. After three years he was still







(A) Computed tomographic brain scan. (B) Signature before (bottom) and after (top) brain damage.

unable to sign with his previous signature, but no further neurological deficit has been appreciated. He shows normal drawing, word-finding, reading, right-left orientation, and calculation ability. Indeed, he can write normally even his isolated name (which was part of the old signature, Fig, B), but only if he does not attempt to trigger a "signature signal." We believe this phenomenon may be of some localizing value, and supports an anatomical distinction between this and other dysgraphias [1-3].

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References

- Friedland J. Development and breakdown of written language. J Commun Disord 1990;23:171–186
- Croisile B, Laurent B, Michel D, Trillet M. Pure agraphia after deep hemisphere haematoma. J Neurol Neurosurg Psychiatry 1990;53:263-265
- Soma Y, Sugishita M, Kitamura K, et al. Lexical agraphia in the Japanese language. Pure agraphia for Kanji due to left posteroinferior temporal lesions. Brain 1989;112:1549-1561