Dysarthria results from lesions in the central nervous system, the peripheral nervous system, or both. Therefore, dysarthria is associated with a wide variety of neurological pathologies and presents in a variety of forms. In a given clinical neurological picture, disturbance of speech is usually relegated towards the bottom of the list of symptoms; its presence may be acknowledged but granted little importance for diagnosis and usually for the rehabilitation process as well. The purpose of this chapter is to present a clinical case study illustrating the potential importance of dysarthria in the differential diagnosis of a neurological condition. Our patient was diagnosed with Binswanger’s disease (BD), but the diagnosis did not match the dysarthria or some aspects of the clinical neuropsychological symptomatology. Subsequently, and in part due to the dysarthric signs, the diagnosis of amyotrophic lateral sclerosis (ALS) was added.

CASE STUDY: FIRST DIAGNOSIS

LS, a 71 year old female, was referred for outpatient speech-language pathology services on the basis of reduced intelligibility and swallowing difficulties. The patient carried the diagnosis of BD. In the nine months preceding her visit, the patient reportedly experienced two episodes of degradation in neurologic function. One of these episodes, presumably a TIA, occurred five months prior to her visit. At that time, the family reported that the patient’s speech was slurred; however, her physician described her speech as normal and her language as relevant. An examination report two months later mentioned normal gait and normal cranial nerve functions. The report also mentioned a CT scan showing mild hyperdensities of the periventricular white matter that were more prominent in the frontal regions and reportedly “of doubtful clinical significance”. No other lesions were observed and the sulci appeared normal.

Approximately one month later, that is, two months prior to her arrival in the speech-language pathology service, an MRI of the head was conducted. The T2 weighted axial images of the brain demonstrated multiple confluent high signal intensity foci of the periventricular white matter and of the centra semiovalia consistent with multiple deep white matter infarcts. The foci of increased signal intensity in the centra semiovalia are clearly visible in Figure 1 as extensive subcortical white areas. There was no evidence of
neoplasm, subdural hematoma or communicating hydrocephalus. A slight lateral ventricle enlargement was noted. The diagnosis of BD was made at that time.

Binswanger’s disease, or subcortical arteriosclerotic encephalopathy (Olszewski, 1962), is characterized by bilateral and symmetric demyelination of the periventricular white matter. Later in the course of the disease, the demyelination invades the entire centrum semiovale (Weisberg, Gerard, and Stasio, 1988). The cerebral cortex and the associated U-fibers are typically spared in BD. Lacunar infarcts in the basal ganglia and the pons may be present, but patients usually show no signs of brain stem infarction (Rosenberg, Kornfeld, Stovring, and Bicknell, 1979; Salomon, Yates, Burger, and Heinz, 1987). Moreover, there are no senile plaques or neurofibrillary tangles (De Reuck, Crevits, De Coster, Sieben, and vander Eecken, 1980).

Until recently, the diagnosis of BD could only be made confidently at autopsy. With the advent of modern imaging techniques, BD can be diagnosed in vivo (Ma, Lundberg, Lilja, and Olsson, 1992). Revesz, Hawkins,