

*Short communications***Cerebral blood flow in thalamic aphasia****A. M. Fasanaro<sup>1</sup>, D. L. A. Spitaleri<sup>1</sup>, R. Valiani<sup>1</sup>, A. Postiglione<sup>2</sup>, A. Soricelli<sup>3</sup>, L. Mansi<sup>3</sup>, and D. Grossi<sup>4</sup>**<sup>1</sup>Divisione di Neurologia, Ospedale A. Cardarelli, Via A. Cardarelli, I-80131 Napoli, Italy<sup>2</sup>Istituto di Medicina Interna e Malattie dismetaboliche, II. Facoltà di Medicina e Chirurgia, Napoli, Italy<sup>3</sup>Cattedra di Medicina Nucleare, Istituto di Scienz Radiologiche, II. Facoltà di Medicina e Chirurgia – Servizio di Medicina Nucleare, Istituto Nazionale dei Tumori, Fondazione Pascale, Napoli, Italy<sup>4</sup>Clinica Neurologica, II. Facoltà di Medicina e Chirurgia, Napoli, Italy

**Summary.** A 59-year-old man is reported, who became aphasic after left thalamic infarction, shown by CT. His speech was fluent, with reduced voice volume, impaired auditory and reading comprehension, verbal paraphasias but intact repetition skills. A single photon emission computed tomography (SPECT) scan to measure regional cerebral flow (rCBF) showed a reduction of flow in the parietotemporal areas of the left hemisphere. It is suggested that thalamic aphasia could result from structural subcortical damage with a homolateral functional cortical deficit leading to the specific aphasic disturbance.

**Key words:** Thalamus – Aphasia – rCBF – SPECT**Introduction**

Language disturbance following thalamic lesions, first described by Déjerine and Roussy [4] and Hillemand [7], has been confirmed by anatomoclinical studies, as well as by neuropsychological evaluation in thalamotomy patients [3].

The disorders can be grouped into extrapyramidal deficits, deficits in word access, and comprehension defects. Thalamic stimulation performed during stereotaxic surgery has indicated that the posterior part of the left thalamus (the pulvinar and the ventrolateral nucleus) plays a definite role in language [9, 14], so that an aphasic disorder occurs if this region is damaged.

More recent observations have been reported by Cappa and Vignolo [1], Mazaux and Orgogozo [11] and Puel et al. [15] in subjects with spontaneous pathology (haemorrhagic or ischaemic strokes). The clinical features of thalamic aphasia, as appears from these reports, resemble in some aspects the semantic aphasia described by Luria [10] for damage of the associative parietal cortex, and the dynamic aphasia described as arising from lesions of the premotor frontal cortex, and the transcortical aphasias [1]. From a pathophysiological viewpoint it has been hypothesized that the thalamic lesion implies a disturbance of the focusing and alerting of neuronal complexes in the posterior temporal and parietal lobes, as the pulvinar projects extensively to these regions [5, 16]. It is, therefore, of interest to investigate such patients by dynamic and structural methods, in order to see whether or not dynamic changes could be related to language impairment.

We report the results obtained in a patient with thalamic aphasia due to an ischaemic stroke, where the regional blood flow was investigated by single photon emission computed tomography (SPECT) using xenon 133 (<sup>133</sup>Xe) [8].

**Case report**

The patient under study was a 59-year-old, right-handed, male farm worker, who had received primary education. No relevant previous disease was reported. The patient was admitted after sudden onset of right-sided weakness and difficulty in speaking. The initial examination showed a mild right-sided hemiparesis with homolateral hypoaesthesia and no visual field defects; the patient was alert and cooperative. There was

**Table 1.** Results of the aphasia examination test (Battery of Neuropsychological Centre of the University of Milan)

<b>A. Oral expression</b>		Fluent, dysprosodic speech with verbal paraphasias, stereotypy, of limited communicative efficacy	
Picture naming	60%	Correct	
Automatic series	100%	Correct	
<b>B. Comprehension</b>			
Words	40%	Correct	
Words of the same semantic group	20%	Correct	
Sentences	70%	Correct	
<b>C. Repetition</b>			
Letters	90%	Correct	
Words	100%	Correct	
Neologisms	90%	Correct	
Sentences	100%	Correct	
<b>D. Reading aloud</b>			
Letters	60%	Correct	
Words	50%	Correct	
Neologisms	0%	Correct	
Sentences	0%	Correct	
Reading comprehension	10%	Correct	
<b>E. Writing</b>			
Spontaneous	0%	Correct	
Dictation	0%	Correct	
Copying	0%	Correct	

a paucity of spontaneous speech; when prodded, however, the patient produced almost fluent speech with stereotypy, neologisms, verbal and phonemic paraphasias. Voice volume was reduced and dysprosody was evident. He was able to understand very simple commands such as “close your eyes”, but failed in more complicated commands. Repetition was normal; naming of objects was impaired; he could no longer read or write. Attempts at figure drawing and copying produced illegible marks that did not resemble the model; he was, however, able to group similar figures.

About 20 days after the onset he was subjected to a complete aphasia examination test (aphasia test of the University of Milan), which demonstrated a profile consistent with transcortical sensory aphasia (Table 1). Furthermore, the general neuropsychological examination showed the presence of constructional apraxia and of long-term memory impairment. EEG showed slow rhythms in the parietotemporal areas.

CT scan of the brain showed an ischaemic area confined to the left thalamus (Fig. 1). A SPECT scan to measure regional cerebral flow (rCBF) was carried out using a brain dedicated

32 phototubes SPECT scanner (Tomomatic 32, Medimatic, Copenhagen) and <sup>133</sup>Xe gas (20 mCi/l). Measurements of rCBF (ml/min per 100 g) were performed in one axial slice (+5 cm from the orbitomeatal line). After the inhalation for 1 min of <sup>133</sup>Xe and a 3-min period of exhalation, the rCBF was calculated according to specially designed algorithms [2]. The SPECT scan revealed a reduction of rCBF in the parieto-temporal areas of the left hemisphere (Figs. 2, 3).

The patient was discharged on the 30th hospital day without any improvement in his language abilities. Three months after discharge a second aphasia test was performed, which showed an improvement of naming and comprehension as well of reading tasks. Writing and drawing abilities were still severely impaired.

Discussion

The present case and others from the literature indicate that a left thalamic lesion is followed by an aphasic picture characterized by dysarthric, dysprosodic non-fluent speech with diminished volume of voice, absence of phonemic paraphasias, presence of verbal paraphasias, preserved verbal repetition with impairment of aural and reading comprehension. We conclude that the profile of the aphasia suggests a sensory transcortical aphasia, in agreement with previously reported cases [1, 6].

The evaluation of cerebral blood flow by SPECT in our patient showed a significantly low perfusion in the parietotemporal region of the left hemisphere; this area is the one expected for transcortical sensory aphasia, and it is congruent with the EEG findings. These data are in agreement with the hypothesis that structural thalamic damage is associated with a functional deficit in the homolateral cortical regions. Furthermore, the data are in agreement with the findings obtained in patients with subcortical lesions where the metabolic activities have been investigated employing positron emission tomography using the fluoro-deoxyglucose-F18 method [12, 13]. Indeed these patients showed diminished metabolic activ-

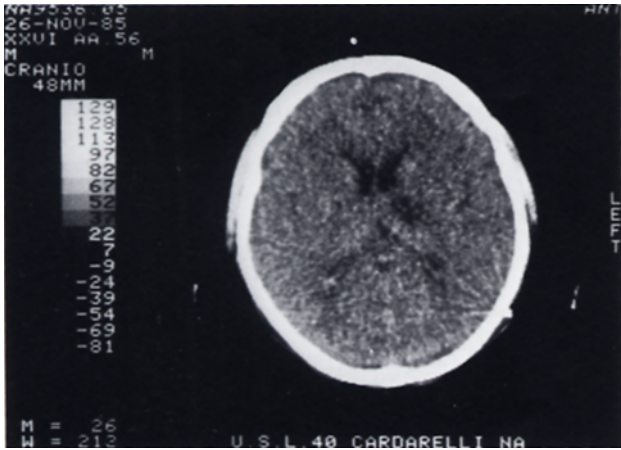
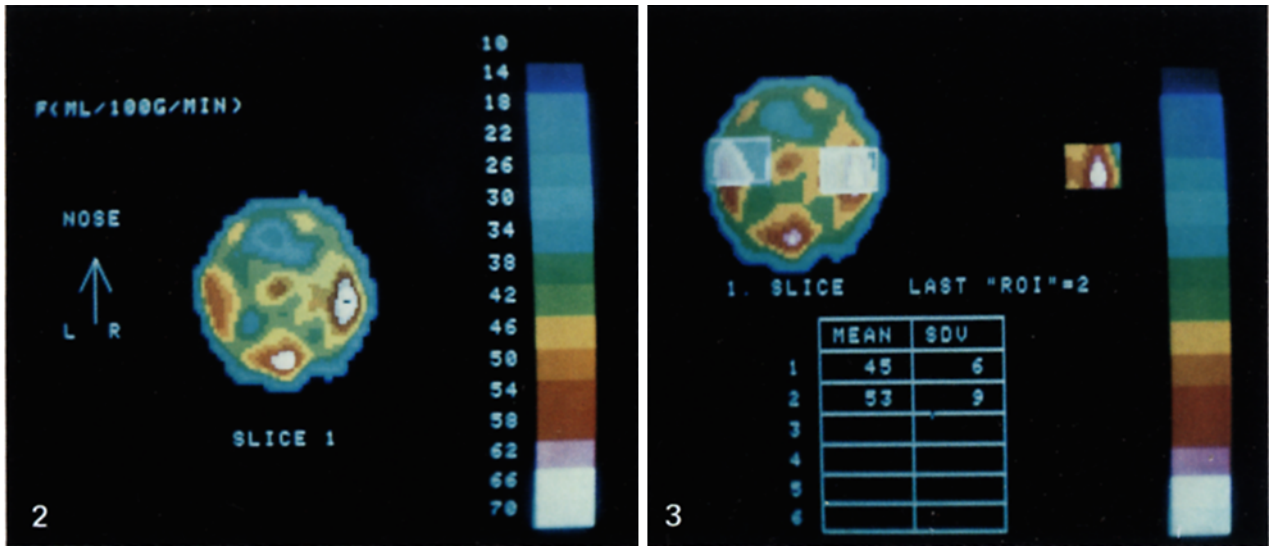


Fig.1. CT scan showing an area of decreased density in the capsular-thalamic region of the left hemisphere



Figs.2, 3. Cerebral blood flow (CBF) in a patient with thalamic aphasia following an ischaemic stroke. Reduction of CBF (>15%) is observed in the left temporal region as compared with the right temporal lobe

ity in cortical regions, while the CT scan demonstrated only a subcortical homolateral lesion. Hence thalamic aphasia can result from subcortical damage leading to the extrapyramidal signs, and from a functional cortical deficit leading to a specific aphasic disturbance.

It seems possible that following structural thalamic damage a disturbance of thalamocortical connections would occur, producing an intrahemispheric vertical disconnection, from which an aphasic syndrome could follow. The analogy between transcortical aphasia and thalamic aphasia suggests that in both cases the connections between areas involved in languages, not the areas themselves, are damaged.

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