

NOTE

APHASIA DOES NOT ALWAYS FOLLOW LEFT THALAMIC HEMORRHAGE: A STUDY OF FIVE NEGATIVE CASES

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INTRODUCTION

Case reports on thalamic aphasia have consistently focused on positive cases. The occurrence of aphasia associated with an acute hemorrhagic or ischemic lesion of the left thalamus has been repeatedly stressed (see Mohr, 1983) all through neurological literature, from the scattered clinical observations of earlier studies (for a review see Lecours and Lhermitte, 1979) to the welter of case-reports of the last two decades. The clinical picture of this "thalamic aphasia" is not uniform, but in most cases a mixed transcortical picture, characterized by preserved repetition, with variable impairment of expression and auditory comprehension, has been described (see Cappa and Vignolo, 1983; Crosson, 1984).

On the other hand, while left thalamic lesions unaccompanied by aphasia in right-handed subjects are an occurrence for the clinician, a surprisingly small number of such "negative" cases are found in literature and they are even fewer if well-studied cases, i.e. patients submitted to a formal language examination in the acute or "recent" period post stroke are considered. Graff-Radford, Damasio, Yamada et al. (1985) described ten cases in which Computerized Tomography (CT) - assessed ischemic lesions were confined to the left thalamus; language performance was investigated by the Multilingual Aphasia Examination (Benton and Hamsher, 1978). All three patients with antero-lateral thalamic infarctions were aphasic. Six patients had an infarction involving the lateral thalamus and the posterior limb of the internal capsule: of these, three, examined three to 15 days after stroke, had mild language difficulties, not conforming to any aphasic syndrome. The remaining three, tested at intervals of three days, one month and five months after stroke, did not show any aphasic disorder. One patient, with a posterior lateral thalamic infarction, associated with a posterior cerebral artery ischemic lesion, was not aphasic three months after stroke. On these grounds the authors suggest that intrathalamic localization of the ischemic lesion is a crucial factor for the occurrence of aphasia. Thalamic hematomas are less suited for

localization than infarcts, as they are usually large lesions which tend to involve, directly or due to mass effects, the whole thalamus and neighbouring structures. A few clinical series of thalamic hemorrhages (Miller Fisher, 1959; Walshe, Davis and Miller Fisher, 1977) suggest that aphasia is a frequent but not an invariable feature of left-sided lesions. No signs of aphasia following vascular thalamic lesions were found in a recent study (Wallesch, Kornhuber, Brunner et al., 1983), but the patients, who had rather small ischemic or hemorrhagic thalamic lesions, were examined months to years post-stroke. These data suggest that the time interval between stroke and neuropsychological assessment is a crucial factor in anatomo-clinical correlation studies.

In the present paper we describe five patients with thalamic hemorrhage, who did not display any sign of aphasia as assessed by a standard examination given within one month after stroke.

MATERIAL AND METHODS

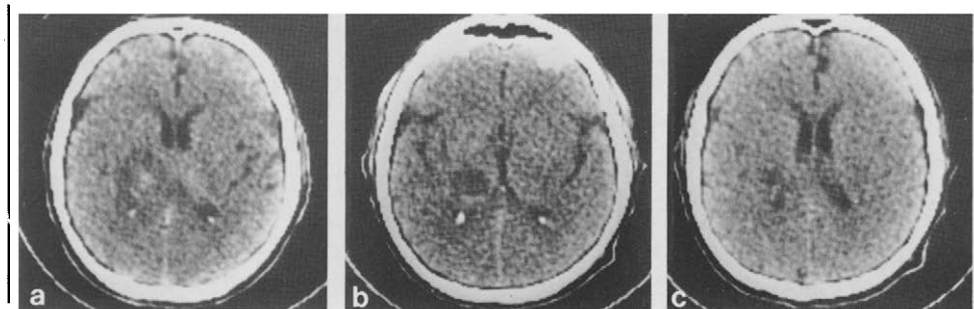
Subjects

Five right-handed patients (four men and one woman) were examined in the first month after a thalamic hemorrhage. Mean educational level was 10.2 years of schooling (range 5-17), mean age was 61.4 years (range 49-74). The lesion was documented by means of the CT scan and the site was checked by referring to the atlas by Matsui and Hirano (1978).

Case I

A 53 year old right-handed man with 13 years of schooling was hospitalized on December 17th, 1980. The day before he had suddenly noticed a loss of strength in his right limbs and difficulty in articulation of speech. On neurological examination he had a right hemiparesis, dysarthria and hypoesthesia in the area of the right fifth cranial nerve. The CT-scan showed a left hemorrhagic area with oedema involving the posterior thalamus (Figure 1).

Fig. 1 — *Case I.* (a) CT performed 12 days after stroke: hyperdense lesion, surrounded by an hypodense area, involving the posterior thalamus, the posterior limb of the internal capsule and encroaching on the posterior lateral lenticular nucleus. (b, c) CT performed 26 days after stroke: hypodense area involving the posterior thalamus.



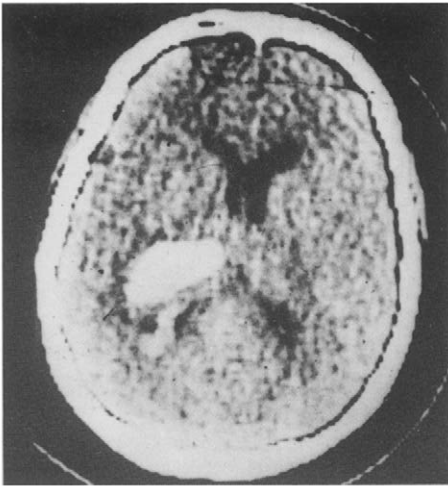


Fig. 2 — Case II. CT performed 2 days after stroke. Hyperdense area involving the posterior thalamus and the posterior limb of the internal capsule; limited extension to subcortical white matter.

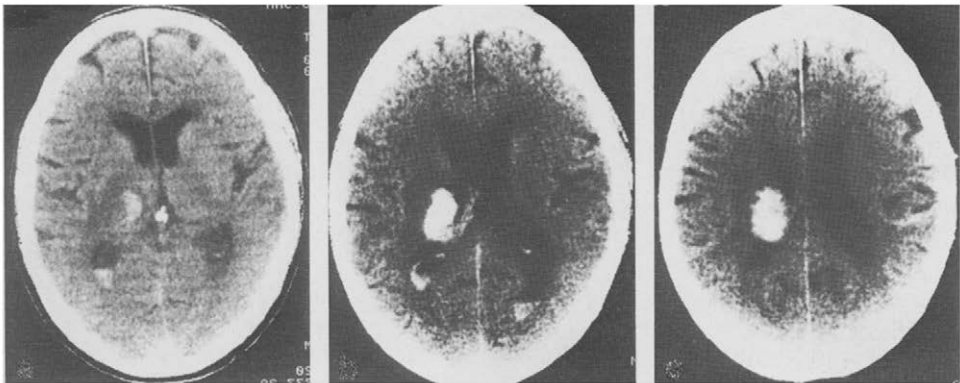


Fig. 3 — Case III. CT performed the same day of the stroke. Hyperdense area involving the posterior two thirds of the thalamus and the posterior limb of the internal capsule; limited extension to subcortical white matter.

Case II

A 49 year old right-handed man with 13 years of schooling was hospitalized on November 2nd, 1980. The night before, he had felt numbness and loss of strength in the right leg. On neurological examination he had a right hemiparesis more pronounced in the arm with increased deep tendon reflexes and Babinski sign. A somatosensory defect was present on the right side. The CT-scan showed an hyperdense area involving the posterior thalamus (Figure 2).

Case III

A 66 year old right-handed man with 17 years of schooling was hospitalized on March 23rd, 1984, when he suddenly noticed a loss of strength in the right limbs. Neurological examination revealed a right hemiparesis, less pronounced in the leg. The CT-scan showed a left hemorrhage involving the posterior two-thirds of the thalamus (Figure 3).

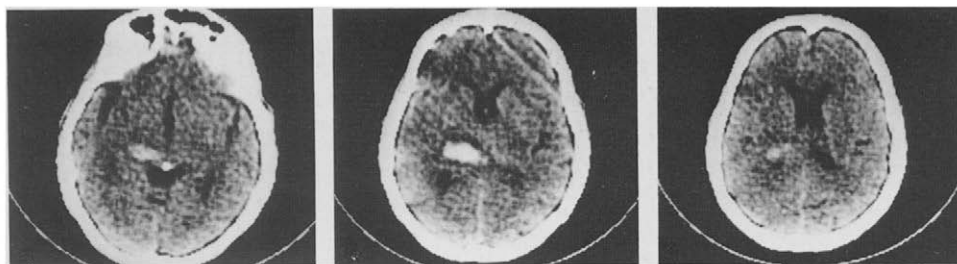


Fig. 4 — Case IV. CT performed one day after stroke. Hyperdense area confined to the posterior thalamus and the posterior limb of the internal capsule.



Fig. 5 — Case V. CT performed the same day of the stroke. Hyperdense area limited to the posterior thalamus and the posterior limb of the internal capsule.

Case IV

A 74 year old right-handed woman with 5 years of schooling was hospitalized on October 3rd, 1984. The day before she had suddenly noticed a loss of strength first in the right arm, then in the right leg. Neurological examination showed that the right arm was plegic, the right leg was paretic and there was a doubtful right homonymous hemianopia. The CT-scan showed a left posterior thalamic hemorrhage (Figure 4).

Case V

A 65 year old right-handed man with 8 years of schooling was hospitalized on September 24th, 1985. The day before, he had suddenly noticed a loss of strength in his right leg, followed by a similar sensation in the right arm. The neurological examination showed a right hemiplegia with hyperreflexia and hypoesthesia. He had severe dysprosodia and dysarthria. The CT-scan showed a left hemorrhage of the posterior thalamus and of the internal capsule.

Language Examination

The presence or absence of aphasia was assessed by means of a standard language examination carried out at our Aphasia Unit (Basso, Capitani and

Vignolo, 1979), supplemented with the shortened version of the Token Test (De Renzi and Faglioni, 1978), which has a cut off score adjusted for educational level of 29/36. This language examination investigates oral expression, auditory verbal comprehension, writing and reading. With the exception of oral and written description subtests (see below), scores are expressed in percentage of correct responses.

Oral expression

Description: the patient has to describe as accurately as possible how he would shave using a safety razor (if a man) and how she would cook spaghetti (if a woman). Communication is scored 0 (none), 1 (poor), 2 (sufficient), 3 (good).

Naming: the patient is asked to name 20 realistic pictures of common objects.

Automatic sequences: the patient is required to recite numbers from one to ten, days of the week, months of the year.

Auditory verbal comprehension

Sentence comprehension: the patient is asked to carry out 10 oral commands.

Word comprehension: the patient is asked to point to pictures on oral command, on a display of 20 pictures. In the second part of this subtest the patient is asked to point to pictures on oral command, on a display of 20 pictures, which are semantically related in groups of four.

Repetition

The patient is required to repeat letters, syllables, words, nonwords and sentences.

Writing

Description: the patient is asked to write a letter to a relative or a friend, giving news about his/her present condition. Score is analogue to oral expression.

Naming: the patient is asked to write the name of 20 pictures.

Dictation: the patient is required to write down auditorily presented letters, syllables, words, nonwords and sentences. This section also assesses copying of words and transcription from print to script.

Reading

Reading aloud: the patient is requested to read aloud letters, syllables, words, nonwords and sentences.

Word comprehension: the patient is given 20 written words and is asked to point to the corresponding pictures, on a 20 item display; in the second part of this subtest the patient is given 20 semantically related words.

Sentence comprehension: the patient is asked to carry out 10 written commands.

RESULTS

The language performances of the five patients are summarized in Table I.

TABLE I

Language Examination of Five Patients with Left Posterior Thalamic Hemorrhage

Case	I	II	III	IV	V
Stroke onset/test interval (days)	21	30	17	28	30
Oral expression					
— Description of an event	3/3	3/3	3/3	3/3	3/3
— Naming	.95	.95	1.0	.85	.85
— Automatic sequences	1.0	1.0	1.0	1.0	1.0
Auditory comprehension					
— Words	1.0	1.0	1.0	1.0	1.0
— Semantically related words	.90	.95	1.0	.75	.85
— Oral commands	.90	.80	1.0	.80	1.0
— Token Test	29/36	34/36	31/36	27/36	30/36
Repetition					
— Letters	1.0	1.0	1.0	.80	1.0
— Syllables	.90	1.0	1.0	.70	.70
— Words	1.0	1.0	1.0	.95	1.0
— Sentences	1.0	1.0	1.0	1.0	1.0
— Nonwords	.90	.80	1.0	.50	1.0
Written expression					
— Signature	+	+	+	+	+
— Naming	.90	.95	1.0	*	.85
— Writing a letter	2/3	2/3	3/3	*	*
Reading comprehension					
— Words	1.0	1.0	1.0	.90	1.0
— Semantically related words	.90	.90	1.0	.75	.80
— Written commands	.80	1.0	1.0	.80	1.0
Reading aloud	1.0	1.0	1.0	1.0	1.0
Dictation					
— Letters	1.0	1.0	1.0	*	*
— Syllables	1.0	.60	1.0	*	*
— Words	1.0	.60	1.0	*	*
— Sentences	1.0	.80	1.0	*	*

*The patient refused to write using the left hand.

DISCUSSION

We have described five patients with a CT-assessed left thalamic hemorrhage. None of them was aphasic on a standard language examination performed within the first month after stroke. Case IV, though clinically nonaphasic, had a Token Test score marginally inferior to the cut off.

All patients suffered lesions which predominantly involved the pulvinar and the posterior parts of the thalamus, suggesting the possibility of a differential role of thalamic nuclei in language functions. Evidence obtained from cases of thalamic infarction appears to support this hypothesis, arguing for a main role of the ventrolateral and anterior nuclei. There was sparing of the posterior region in the aphasic patients reported by Davous, Bianco, Duval-Lota et al. in 1984 (neuropathological examination), by Archer, Ilinsky, Goldfader et al. in 1981 (stereotactic reconstruction from CT scan) and by Gorelick, Hier, Benevento et al. in 1984 (CT-assessed lesion). In the six aphasic patients of Graff-Radford et al. (1985) two lesion sites were found to be associated with language disturbances:

mild, aspecific disorders were shown in the case of the three infarctions involving the lateral thalamus and the posterior limb of the internal capsule, while clear-cut aphasia was present in the three patients suffering from anterolateral thalamic infarctions.

These findings appear to suggest an association of ventrolateral and anterior thalamic infarctions with aphasia. However, the data from hemorrhagic lesions confined to portions of the thalamus conjure up a less definite anatomic-clinical correlation pattern. Ciemins (1970) described an aphasic patient with an autopsically ascertained hemorrhagic lesion involving the left posterior thalamus, while aphasia was absent in the two patients with comparable CT damage reported by Hirose, Kosoegawa, Saeki et al. (1985). Unfortunately no formal language examination was reported in these patients. Mohr, Watters and Duncan (1975) reported a case with an anterior thalamic hematoma and aphasia. However, in the retrospective autopsy series reviewed in the same paper, there was one patient with aphasia and posterior thalamic hemorrhage, and another patient without aphasia with hemorrhage of the anterior thalamus. Sameral, Wright, Sergay et al. (1976)'s case, one aphasic patient of Reynolds, Turner, Harris et al. (1979)'s series and one case of Mazaux and Orgogozo (1982)'s series showed aphasia associated with a CT-assessed hemorrhage involving the posterior half of the thalamus.

A comparison of the present findings with the data of the literature is in order. While our study indicates a good correlation between posterior thalamic hemorrhage and absence of aphasia, a number of previous observations does not allow us to draw a clear-cut conclusion. Lesion site *per se* does not always account for the presence or absence of aphasia in the individual case. The same holds true for patients with left capsulo/putaminal ischemic or hemorrhagic lesions (Cappa, Cavallotti, Guidotti et al., 1983; Puel, Demonet, Bonafè et al., 1984). In a large series of subcortical lesions, Puel et al. (1984) have shown that there is no clear cut correlation between lesion site and type of aphasia. It may be worth reminding that the same inconsistency has been found in patients with right hemispheric subcortical lesions as far as contralateral spatial neglect is concerned (Vallar and Perani, in press): 50% of patients with thalamic lesions and 70% of those with lenticular lesions did not show signs of impairment on an exploratory task given within one month after stroke. Noninvasive imaging techniques for measuring cerebral blood flow and metabolism may be a helpful supplement to traditional anatomoclinical correlations in neuropsychology. Studies using Positron Emission Computed Tomography have shown metabolic depression in cerebral areas which appeared undamaged at the CT examination (Metter, Wasterlain, Kuhl et al., 1981). Patients with CT-assessed subcortical vascular lesions may show blood flow reduction in the ipsilateral cortex not only in an acute phase (Olsen, Larsen, Herning et al., 1983), but also one month to one year after stroke (Perani, Gerundini, Di Piero et al., 1985). Olsen, Bruhn and Öberg (1984) have recently shown that aphasic patients with CT-assessed subcortical lesions have a low cortical blood flow, while non aphasics have no detectable blood flow abnormalities. Future research should consider *both* the site and size of the subcortical lesion *and* the functional cortical disfunction as relevant factors in determining the presence/absence of aphasic disorders.

ABSTRACT

Five right-handed patients suffering from a recent CT-assessed left posterior thalamic hemorrhage are reported. In all patients a standard language exami-

nation performed within one month after stroke onset did not reveal aphasic disturbances. While the present series argues for the role of intrathalamic lesion localization, a review of the literature suggests that lesion site *per se* does not always account for the presence or absence of aphasia in the individual case. The importance of reduced blood flow and metabolism in cortical areas which appear undamaged on CT scan is briefly discussed.

REFERENCES

- ARCHER, C.R., ILINSKY, I.A., GOLDFADER, P.R., and SMITH, K.R. Aphasia in thalamic stroke: CT-stereotactic localization. *Journal of Computer Assisted Tomography*, 5: 427-432, 1981.
- BASSO, A., CAPITANI, E., and VIGNOLO, L.A. Influence of rehabilitation on language skills in aphasic patients. A controlled study. *Archives of Neurology*, 36: 190-196, 1979.
- BENTON, A.L., and HAMSHER, K. *Multilingual Aphasia Examination*. Iowa City: Department of Neurology, University of Iowa Hospitals, 1978.
- CAPPA, S.F., CAVALLOTTI, G., GUIDOTTI, M., PAPAGNO, C., and VIGNOLO, L.A. Subcortical aphasia: two clinical-CT scan correlation studies. *Cortex*, 19: 227-241, 1983.
- CAPPA, S.F., and VIGNOLO, L.A. CT scan studies of aphasia. *Human Neurobiology*, 2: 129-134, 1983.
- CIEMINS, V.A. Localized thalamic hemorrhage. A cause of aphasia. *Neurology*, 20: 776-781, 1970.
- CROSSON, B. Role of the dominant thalamus in language: a review. *Psychological Bulletin*, 96: 491-517, 1984.
- DAVOUS, P., BIANCO, C., DUVAL-LOTA, A.M., DERCONDO, J., VEDRENNE, C., and RONDOT, P. Aphasie par infarctus thalamique paramédian gauche. Observation anatomo-clinique. *Revue Neurologique*, 140: 711-719, 1984.
- DERENZI, E., and FAGLIONI, P. Normative data and screening power of a shortened version of the Token Test. *Cortex*, 14: 41-44, 1978.
- GORELICK, P.B., HIER, D.B., BENEVENTO, L., LEVITT, S., and TAN, W. Aphasia after left thalamic infarction. *Archives of Neurology*, 41: 1296-1298, 1984.
- GRAFF-RADFORD, N.R., DAMASIO, H., YAMADA, T., ESLINGER, P.J., and DAMASIO, A.R. Nonhaemorrhagic thalamic infarction. Clinical, neuropsychological and electrophysiological findings in four anatomical groups defined by computerized tomography. *Brain*, 108: 485-516, 1985.
- HIROSE, G., KOSOEGAWA, H., SAEKI, M., KITAGAWA, Y., ODA, R., KANDA, S., and MATSUHARA, T. The syndrome of posterior thalamic hemorrhage. *Neurology*, 35: 998-1002, 1985.
- LECOURS, A.R., and LHERMITTE, F. *L'Aphasie*. Paris: Flammarion, 1979.
- MATSUI, T., and HIRANO, A. *An Atlas of the Human Brain for Computerized Tomography*. Tokyo: Igaku-Shoin, 1978.
- MAZAUX, J.M., and ORGOGOZO, J.M. Étude analytique et quantitative des troubles du langage par lésion du thalamus gauche: l'aphasie thalamique. *Cortex*, 18: 403-416, 1982.
- METTER, E.J., WASTERKAUB, C.G., KUHLE, D.E., HANSON, W.R., and PHELPS, M.E. ¹⁸FDDG Position Emission Computed Tomography in a study of aphasia. *Annals of Neurology*, 10: 173-183, 1981.
- MILLER FISHER, C. The pathologic and clinical aspects of thalamic hemorrhage. *Transactions of the American Neurological Association*, 84: 56-59, 1959.
- MOHR, J.P., WATTERS, W.C., and DUNCAN, G.W. Thalamic hemorrhage and aphasia. *Brain and Language*, 2: 3-17, 1975.
- MOHR, J.P. Thalamic lesions and syndromes. In A. Kertesz (Ed.), *Localization in Neuropsychology*. New York: Academic Press, 1983, Ch. 11, pp. 269-293.
- OLSEN, T.S., BRUHN, P., and OBERG, G. Cause of aphasia in stroke patients with subcortical lesions. *Acta Neurologica Scandinavica, Suppl.*, 69: 311-312, 1984.

- 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.
- PERANI, D., GERUNDINI, P., DI PIERO, V., SAVI, A., CARENZI, M., VANZULLI, A., DEL MASCHIO, A., LENZI, G.L., and FAZIO, F. Remote cortical effects in patients with ischemic lesions of subcortical structures studied with 123HIPDM and SPECT. *Journal of Nuclear Medicine and Allied Sciences*, 29: 129-130, 1985.
- PUEL, M., DEMONET, J.F., CARDEBAT, D., BONAFÉ, A., GAZOUNAUD, Y., GUIRAUD-CHAUMEIL, B., and RASCOL, A. Aphasies sous-corticales. Étude neurolinguistique avec scanner X de 25 cas. *Revue Neurologique*, 140: 695-710, 1984.
- REYNOLDS, A.F., TURNER, P.T., HARRIS, A.B., OJEMANN, G.A., and DAVIS, L.E. Left thalamic hemorrhage with dysphasia: a report of five cases. *Brain and Language*, 7: 62-73, 1979.
- SAMERAL, A., WRIGHT, T.L., SERGAY, S., and TYLER, H.R. Thalamic hemorrhage with speech disorder. *Transactions of the American Neurological Association*, 101: 283-285, 1976.
- VALLAR, G., and PERANI, D., The anatomy of unilateral neglect after right hemisphere stroke lesions. A clinical/CT-scan correlation study in man. *Neuropsychologia*, (in press).
- WALLESCH, C.W., KORNUBER, H.H., BRUNNER, R.J., KUNZ, T., HOLLERBACH, B., and SUGER, G. Lesions of the basal ganglia, thalamus and deep white matter: differential effects on language functions. *Brain and Language*, 20: 286-304, 1983.
- WALSHE, T.M., DAVIS, K.R., and MILLER FISHER, C. Thalamic hemorrhage: a computed tomographic-clinical correlation. *Neurology*, 27: 217-222, 1977.

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