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Ideomotor Apraxia in Left Thalamic Hemorrhage: Discrepancy between Clinical Course and SPECT

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We present a patient who developed severe ideomotor apraxia (IA) and subcortical aphasia after a hemorrhage involving the posterior part of the left thalamus and the posterior limb of the internal capsule. The cerebral blood flow (CBF) of the left hemisphere as measured by 99Tc-HM-PAO SPECT was initially diminished as compared to the right hemisphere. The apraxia and aphasia eventually resolved. Despite this clinical improvement CBF of the left hemisphere worsened. Our findings do not support the view that apraxia and aphasia following lesion of deep subcortical structures is due to cortical derangement induced by disruption of unspecific activating thalamo-cortical pathways. The results call for caution in the functional interpretation of perfusion deficits detected by SPECT.

Introduction

Ideomotor apraxia (IA) is usually associated with lesions of the left cerebral hemisphere affecting the inferior parietal lobule (Heilman *et al.*, 1982), the temporo-frontal connections, especially the fasciculus arcuatus (Benson, 1973), and the prefrontal area. In these cases, apraxia is bilateral. Lesions confined to the corpus callosum may lead to left sided unilateral apraxia (Liepmann, 1905; Geschwind, 1965; Watson and Heilman, 1983). Contrary to these well known lesion sites, IA has rarely been reported in subcortical vascular lesions. It was then usually mild and limited to body-part-as-object errors (BPO) (Alexander and LoVerme, 1980; Basso *et al.*, 1980; Agostoni *et al.*, 1983; Hungerbühler *et al.*, 1984). De Renzi *et al.* (1986), however, recently described five patients with severe IA due to thalamic, putaminal, or capsular vascular lesions on the left side. Thus, they considered these structures critical for gestural organization, i.e. praxis.

Several authors concluded from their SPECT and PET studies that aphasia and neglect with subcortical lesions were due to distant "cortical" effects, i.e. that either a disruption of activating thalamo-cortical pathways or cortical hypofusion induced a functional deficit or cortical areas, thereby leading to aphasia or neglect (Metter *et al.*, 1981; Baron *et al.*, 1986; Skyhøj *et al.*, 1986; Perani *et al.*, 1987; Vallar *et al.*, 1988). In ideomotor apraxia due to a subcortical lesion however, CBF has never been studied. We report a

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patient with severe IA, oral apraxia, and subcortical aphasia after left thalamic hemorrhage in whom we performed serial SPECT studies. We assumed that if the apraxia was due to a functional deficit of cortical areas, then SPECT would not only demonstrate a perfusion deficit of the left hemisphere, but such a deficit should correlate with the recovery of the patient in subsequent SPECT studies.

Case Report

A 60-year-old right-handed woman was admitted with an acute right hemiplegia and an inability to speak. Her medical history was unremarkable except for moderate arterial hypertension and hyperlipidemia. A CT-Scan performed the same day (Fig. 1*a*) showed a hemorrhage of the posterior part of the left thalamus with involvement of the posterior limb of the internal capsule and probable compression of the putamen. The hemorrhage extended into the tegmentum of the midbrain.

On neurological examination 3 days post onset, there was a right hemiplegia and hemihypaesthesia including the face, a visual extinction

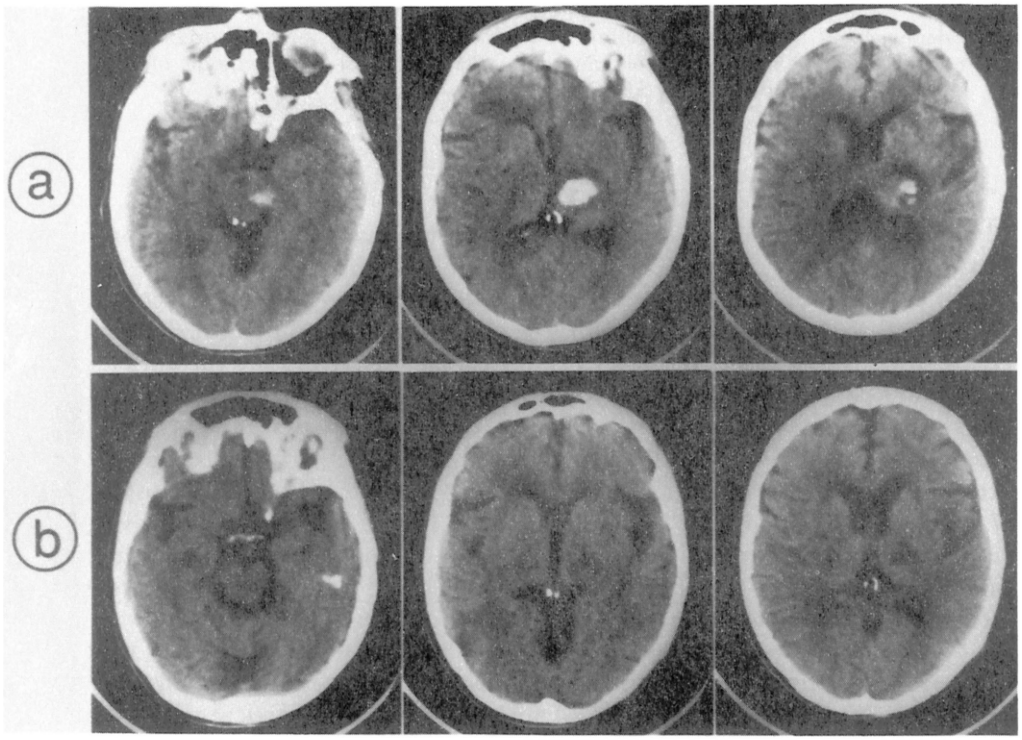

FIG. 1. (*a*) CT scan on day 1, showing a fresh hemorrhage in the left posterior thalamus and posterior limb of the internal capsule, extending into the midbrain tegmentum. (*b*) CT scan on day 60 with complete resolution of the hematoma, leaving only a small hypodense area in the lateral part of the thalamus and posterior limb of the internal capsule.

phenomenon for the right half field but no hemispatial neglect. There was a right central Horner's sign, a conjugate upbeat-nystagmus and an upward gaze palsy, which disappeared within 2 weeks. On mental status examination she was alert but she could neither initiate speech spontaneously, nor when asked to repeat, sing or count. Auditory comprehension was moderately impaired. She correctly and reliably executed two step verbal commands. Reading comprehension, however, was severely impaired, as tested by single word-object matching and written commands. Writing and copying of a drawing of a flower with the left hand were impossible. Tactile object recognition with the left hand and visual object recognition were both intact, as evidenced by correct object-object picture matching. Figural memory was preserved. There was no ideational or limb-kinetic apraxia of the left arm. However, there was severe ideomotor apraxia of the face, the left arm and the left leg, which included spatial and sequential errors (Goodglass and Kaplan, 1983; Rothi *et al.*, 1988). Thus when asked to put out her tongue, she grasped her chin with the left hand and shook it to and fro, then pulled her tongue out to the front teeth. When asked to show the use of a hammer, she made a wiping movement on the table with the open hand. If asked to show the use of a key, she made repetitive circling movements on the table with the tip of her index finger. The performance neither improved if the object was shown to the patient, nor upon imitation. She, however, recognized the correct movement easily if the examiner performed some related gestures from which she had to choose. When given the real object she performed the appropriate movements flawlessly.

On the fourth day, the first brain SPECT was performed (Fig. 2*a*).

While the motor and sensory deficits persisted, language remarkably improved and on day 11, the patient started to utter spontaneously simple sentences. By day 16 she could name objects put into her left hand and correctly repeat longer sentences. In the same period, praxis greatly improved. She correctly showed her teeth or tongue on request and was able to pantomime the use of objects such as a key, a toothbrush, a hammer or a screwdriver. However, this was not true for all items, i.e. when asked to pantomime taking off her glasses, she still wiped her eyes with the hand. She also became able to copy the drawing of a flower and to write with the left hand.

After the marked clinical improvement (Fig. 3), a second brain SPECT was performed on day 19 (Fig. 2*b*).

Over the next 5 months, the hemiplegia improved so that the patient could perform proximal arm movements and walk with a cane without help. Severe right hemisensory disturbances, however, persisted. Except for a mild dysarthria spontaneous speech became fluent without paraphasias. Repetition, auditory and reading comprehension, as well as naming normalized completely. Writing with her left hand was slow, but no aphasic errors were noted. During the same period, apraxia almost completely disappeared. Three weeks after the stroke, only BPO errors were observed which became subsequently rarer and praxis became quasi normal.

A CT-Scan on day 60 showed complete resolution of the hematoma

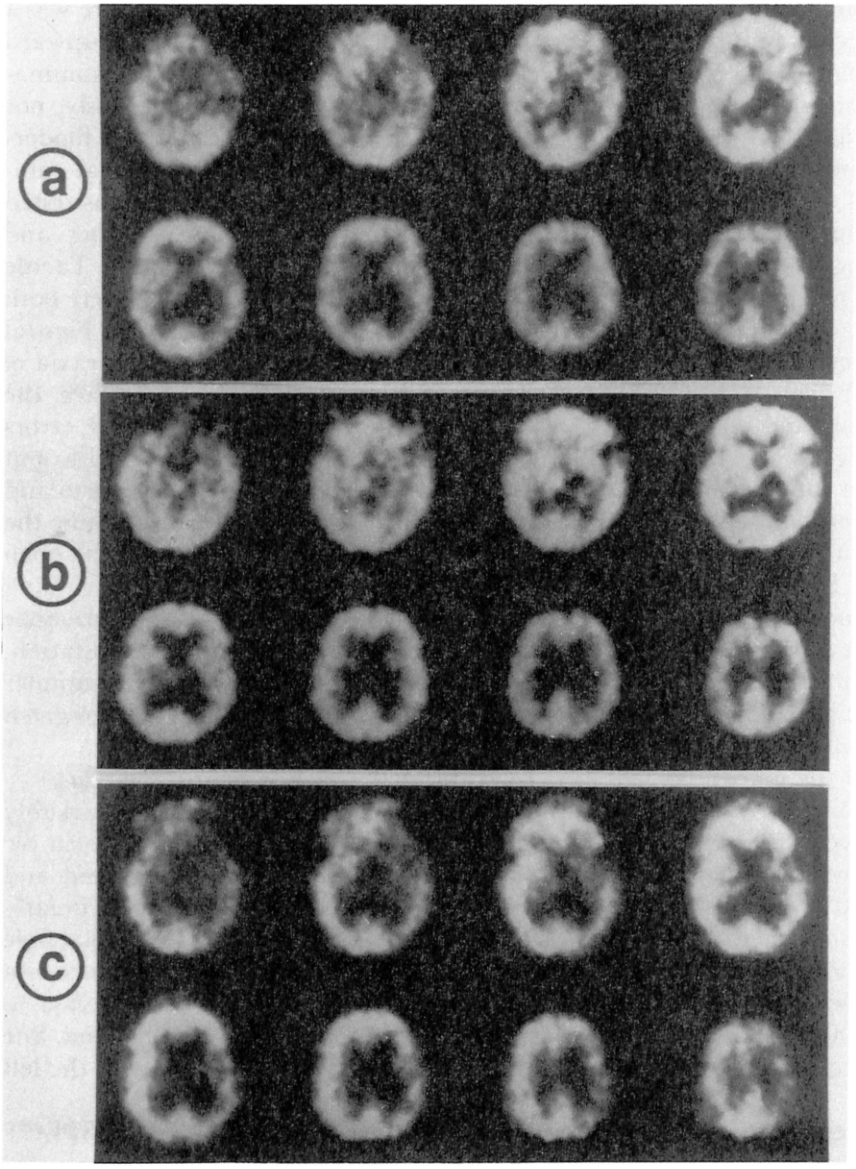

FIG. 2. SPECT studies on (*a*) day 4, showing a marked perfusion deficit in the area of the left thalamus and posterior part of the basal ganglia, as well as a hypoperfusion of the whole cortical mantle of 9% as compared to the right hemisphere, (*b*) day 19, with increase of the perfusion deficit of the left hemisphere (14%), despite marked clinical improvement, and (*c*) day 143, with virtually unchanged perfusion in spite of further clinical improvement. The orientation of the SPECT images corresponds to the CT scan in Fig. 1, i.e. the right side of the images corresponds to the left side of the brain.

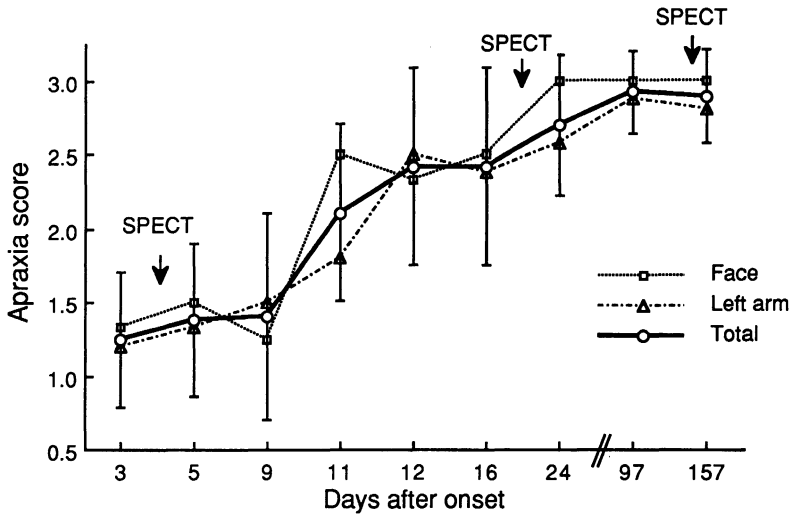

FIG. 3. Course of improvement of oral and ideomotor apraxia as measured by an apraxia-score (Kertesz and Ferro, 1984) (3 = good performance, 2 = impaired but recognizable, 1 = poor performance, 0 = no response, unrecognizable or unrelated gesture), with respect to the time from onset of the symptoms. The time of the SPECT studies on the 4th, 19th, and 143rd day is indicated.

without evidence of a vascular malformation or a tumor. Only a small hypodense area was evident in the lateral part of the thalamus and the posterior limb of the internal capsule (Fig. 1*b*).

With respect to the clinical improvement of the hemiplegia and the virtual disappearance of apraxia and aphasia, a third SPECT was performed on day 143 (Fig. 2*c*).

Methods of Investigation

Apraxia

Ideomotor and oral apraxia were tested for with the usual items (Goodglass and Kaplan, 1983; Heilman and Rothi, 1985). The motor responses of the patient were scored immediately after the examination as proposed by Kertesz and Ferro (Kertesz and Ferro, 1984): 3 = good performance, 2 = impaired but recognizable performance, 1 = poor, only approximate performance or performance with object only, and 0 = no response, unrecognizable or unrelated gesture). The clinical course of apraxia is shown in Fig. 3.

SPECT

The patient underwent three SPECT perfusion studies. Fifty-five MBq of 99mTc-HMPAO was administered intravenously. The injection was carried

out in a quiet and darkened room, with the patient supine and her eyes closed. All three studies were performed with a rotating single head camera (Siemens Orbiter 7500) using an astigmatic collimator (Neurofocal^{R}). Data acquisition followed the standard protocol with a 360° rotation, employing the step and shoot mode with 128 increments, extending over an examination time of about 35 min. The raw data were fed into a dedicated computer system (Siemens Micro-Delta) and, after reconstruction done over night (VAX 750 or Micro-Vax II), the study was filtered in x, y and z directions with a three-point operator. Slices of 6 mm each were generated in a 64 × 64 matrix which covered the whole cerebrum and the upper half of the cerebellum. For a comparison of left to right perfusion, a semiautomated computer program utilizing doubled (= 12 mm) slice was employed. The outer borders of the cerebrum and inner borders of the grey matter were delineated using eliptic sections. The areas on both sides between the frontal and occipital pole were subdivided into six equidistant regions of interest. The count rates obtained from these regions were displayed, and the interhemispheric ratios were calculated. Normal (±2SD) thresholds are 0·87 to 1·12 (Buell *et al.*, 1988).

Results

As shown in Fig. 3, the initially very low apraxia-score for both ideomotor and oral apraxia improved markedly between days 9 and 12. Oral apraxia normalized by day 24 and ideomotor apraxia almost reached a steady state from this day on with only occasional use of body-part-as object errors (BPO).

Table 1 shows the perfusion of the left hemisphere in relation to the right hemisphere for the regions separately calculated.

Day 4: (Fig. 2*a*) In addition to a marked perfusion deficit in the area of the left thalamus and posterior part of the basal ganglia, there was hypoperfusion of the whole cortical mantle on the left side involving both frontal and occipital aspects, i.e. within all three main vascular territories. The perfusion deficit of the left hemisphere was calculated as 9% as compared to the right hemisphere.

TABLE 1. *Percentage of the perfusion of the left hemisphere in relation to the right hemisphere as measured by SPECT for the regions separately calculated*

Area	*Day 4*	*Day 19*	*Day 143*
Frontal	94	88	90
Fronto-precentral	89	91	84
Central	90	84	82
Temporo-parietal	92	85	83
Parieto-occipital	87	85	85
Occipital	90	78	91
Total	91	86	86

Day 19: (Fig. 2*b*) While the hypoperfusion of the deep structures around the left thalamus was unchanged, the perfusion deficit of the left hemisphere as a whole had increased to 14% as compared to the right hemisphere.

Day 143: (Fig. 2*c*) The average of the perfusion deficit of the left hemisphere remained at 14% as compared to the right hemisphere.

Discussion

Recently a number of well-documented cases have shown that lesions of the left thalamus, internal capsule and basal ganglia can cause severe ideomotor apraxia (De Renzi *et al.*, 1986). Our patient complements the literature and was additionally studied with serial SPECT. She suffered from an intracerebral hemorrhage involving mainly the posterior thalamus and the posterior part of the internal capsule. The ideomotor apraxia included severe spatial and sequential errors during the first 2 to 3 weeks, while in later examinations, only BPOs were found, which persisted to a mild degree at the time of the last examination at 5 months. The subcortical aphasia showed a similar improvement with only a minimal residual dysarthria.

The mechanism leading to apraxia with deep subcortical lesions is unclear. De Renzi (1986) suggested that it was due to a disruption of "basal ganglia-thalamo-cortical" loops. Such a mechanism, long ago conjectured for certain types of aphasia by Marie (1906), has recently been reconsidered to explain aphasia following subcortical lesions (Damasio *et al.*, 1982; McFarling *et al.*, 1982). The clinical course of our patient with rapid improvement within 2 weeks and subsequently slower improvement, is consistent with the idea of the blocking of thalamo-cortical pathways and their release by a diminution of the surrounding edema and subsequent resolution of the thalamic hemorrhage. The CT scan performed after 60 days supports this assumption as it shows complete resolution of the hematoma leaving only a small hypodense area in the lateral part of the thalamus and the posterior limb of the internal capsule (Fig. 1*b*). However, given the close relationship of "specific" and "unspecific" activating pathways (Brodal, 1981), the CT scan does not allow the distinction between a lesion affecting fibers that are specific for praxis and a lesion affecting unspecific fibers that are supposed to activate cortical centers involved in praxis. We endeavoured to clarify this issue in our patient by means of SPECT.

SPECT was performed initially and subsequently twice after significant clinical improvement of the praxia and aphasia had occurred. The initial SPECT showed a hypoperfusion involving the whole cortical mantle of the left hemisphere. Several PET studies of patients with aphasia or neglect with subcortical lesions have reported ipsilateral cortical hypometabolism which was either attributed to a disruption of unspecific activating thalamo-cortical pathways (Metter *et al.*, 1981; Baron *et al.*, 1986) or to cortical hypoperfusion (Skyhøj *et al.*, 1986). In both cases, the aphasia or neglect was considered due to cortical dysfunction. A similar conclusion was drawn from

SPECT studies of patients with subcortical aphasia (Perani *et al.*, 1987). If either of these hypotheses were adequate for our patient, i.e. if there really was a causal relationship between the hypoperfusion and the apraxia and aphasia, the improvement of the perfusion deficit of the left hemisphere and the clinical recovery should follow a parallel course. This clearly was not the case. The control SPECTs 19 and 143 days after the event showed the relative perfusion deficit of the left hemisphere to have increased despite a marked clinical improvement. This stands at odds with the findings of Vallar *et al.* (1988) who found a positive correlation between recovery from aphasia and improvement of the cortical perfusion in their patients studied with SPECT. However, a positive correlation does not prove a causal relationship between the neuropsychological deficit and the cortical hypoperfusion. Negative cases such as ours in whom cortical hypoperfusion and the recovery of higher "cortical" functions do not correlate call for caution in assuming such a causal relationship of the neuropsychological deficit under consideration and the cortical hypoperfusion as evidenced by SPECT.

The mechanisms leading to cortical hypoperfusion as detected with SPECT or PET in subcortical lesions are not known. The notion of a disruption of unspecific activating thalamo-cortical pathways (Metter *et al.*, 1981; Baron *et al.*, 1986) cannot account for the evolution of the hypoperfusion in our patient since such pathways would be expected to recover as well as "specific" pathways. The matter seems even more complicated as Rango *et al.* (1989) recently found in a SPECT investigation of nine patients with acute hemispheric ischemic strokes, that SPECT agreed particularly well with the clinical findings within the first 48 hours but not by day 10 or 30. In some of their patients, the evolution of the hypoperfusion evidenced by SPECT was similar to our patient in that the SPECTs at day 10 and 30 showed a more severe hypoperfusion of the damaged hemisphere than the initial SPECT. They concluded that "this may reflect tracer characteristics or aspects of recovery from cerebral ischemia that are still not fully understood".

In summary, our study shows that severe ideomotor apraxia may emanate from a deep subcortical lesion. The SPECT studies performed in our patient do not support the view that apraxia and aphasia following damage to subcortical structures is due to distant "cortical" effects as thought to be measured by hypoperfusion in SPECT. As long as the mechanisms leading to perfusion changes evidenced by SPECT are not fully understood, SPECT results have to be interpreted with caution as regards their functional significance.

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