RIGHT MOTOR NEGLECT ASSOCIATED WITH DYNAMIC APHASIA, LOSS OF DRIVE AND AMNESIA: CASE REPORT AND CEREBRAL BLOOD FLOW STUDY

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Abstract—A 30-year-old right-handed man had right motor neglect, amnesia, aphasia and loss of drive following bilateral thalamic and subthalamic infarctions. Serial resting cerebral blood flow (CBF) measurements with either Xenon 133 inhalation or positron emission tomography at 1, 8 and 10 months post-onset showed a widespread and long-lasting low CBF in the cortex. An additional CBF measurement, during motor tasks, showed a marked interhemispheric asymmetry in the pattern of activation: whereas left hand movement resulted in a CBF increase in contralateral superior rolandic and prerolandic areas, no significant regional CBF increase in contralateral superior rolandic areas during voluntary movement of the previously neglected side points to a disruption of cortico-subcortical pathways subserving motor activation. The pathophysiology of aphasia, loss of drive and amnesia as well as their relationships to motor neglect, may also be discussed on the basis of thalamo-cortical disconnections.

INTRODUCTION

MOTOR NEGLECT (MN) was initially described by CRITCHLEY (1953) [14] as a lack of spontaneity affecting motor function, following expanding lesion within the parietal lobe. This manifestation was further specified by CASTAIGNE *et al.* (1970, 1972) [11, 12] so that the main features of MN include: (1) unilateral and spontaneous reduction of the motricity without motor deficit; (2) decreased withdrawal motor reaction to pain; (3) inappropriate position of the limbs left in uncomfortable position; (4) reversibility of these symptoms after verbal stimulation. This syndrome is more often than not part of the non-dominant hemisphere syndrome [2, 6, 7, 59, 60] but may occur rarely as a pure condition without other deficits suggestive of spatial or sensory neglect. These isolated cases, along with experimental studies in non-human primates [58], have allowed to hypothesize a primary defect of programming and planning movement [25] independent of sensory and spatial neglect [54]. The lesions underlying unilateral MN have been ascribed to various topographic sites, most in the right cerebral hemisphere: parieto-temporal cortex [10, 25], frontal cortex [11, 25], anterior limb of the internal capsule [45, 56], internal capsule and lenticulostriate structures [54], thalamus [5, 24, 47] and ventrolateral nucleus of the thalamus [63]. All these structures

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are thought to be concerned in the planning, programming and organizing of movement [25] and thus would comfort the hypotheses described above. Here, we report a clinical case of right MN caused by left thalamic lesions, without sensory or spatial neglect, but associated with profuse neuropsychological deficits. As neuropsychological symptoms following thalamic lesions are thought to reflect cortical dysfunction [7, 16, 60], each deficit could be interpreted in terms of specific thalamocortical dysconnections.

The measurement of CBF in the present case has shown a diffuse and remote cortical low flow reflecting a probable hypometabolism. Such hypometabolism, following thalamic lesions, has been previously demonstrated [4, 36] and interpreted in terms of interruption of neural connections, reminiscent of the concept of diaschisis [17, 37]. An additional regional CBF measurement, during left and right sensorimotor activation tasks, showed a marked interhemispheric asymmetry in the pattern of activation.

CASE PRESENTATION

A right-handed 30-year-old man born in Turkey suddenly had a diffuse and severe headache and then became comatose for 24 hr. After that time, when the patient was again completely conscious, he was found to have a lack of spontaneous movement of the right limbs. His gait was severely disturbed by constant deviation to the right; he dragged the right leg and did not swing the right arm; he fell often. Moreover, he frequently hit objects with his right side but no attempt was made to avoid them. Spontaneously, when he was sitting he left his right arm limp next to the body, and when he was lying the leg and the arm were left in uncomfortable positions behind the body or hanging out of the bed. Surprisingly, spontaneous 'placing reactions' such as putting the hand on the footboard of the bed, on the wall, or crossing the legs, were sometimes but rarely executed perfectly, in contrast to the paucity of movement of the right limbs. In usual bimanual motor activities such as eating, or dressing, he failed totally to use the right hand or merely made the beginning of the gesture. Painful stimuli to the right hemibody caused no, or a very light, withdrawal reaction, whereas pain was normally felt. Following verbal stimulation, when the patient was asked with insistence to contract his limbs, the motor strength was symmetrical and without segmental deficit. Deep tendon reflexes were symmetrical and the plantar reflexes flexor. A comment from the examiner to indicate an inappropriate or uncomfortable position of the arm and leg was usually sufficient to lead the patient to rectify it. On the other hand, improvement of gait and gestures needed continuous and sustained encouragements and, even so, there was not full disappearance of the neglect. When the patient was asked to put his right index-finger to his nose or ear, a definite dysmetria was noted as he fell behind or beside the target. Examinations performed at different periods never showed sensory deficit or extinction. Somatosensory evoked potentials after stimulation of the median nerves were asymmetrical since the latest parietal wave (42 msec) was not recorded on the left side. There was no hemianopsia or visual extinction. No visuo-spatial neglect was found on the following tasks: complex image description, placement of cities on the map of Turkey, tridimensional constructive praxia (Benton), clock and room drawing. The Rey figure revealed a mild constructive apraxia. Verbal dichotic listening gave normal results. The patient's behaviour was remarkable: he was extremely inert, spending much time doing nothing but watching television. He showed a total lack of concern regarding his presence in hospital and seemed unaware of his own illness. This impassive state was easily abolished by external stimulation: for instance, he would perform neuropsychological tasks with reasonably sustained attention. No frontal signs were detected in the Porteus maze test and the Raven progressive matrices: he performed normally the sequence of three consecutive hand movements and had no grasping or imitation syndrome. A dynamic aphasia was present including loss of initiation of speech, reduced but coherent narration, with some word-finding difficulties and profoundly reduced verbal fluency: he said three words in 1 min in the verbal fluency subtest of the French version of the Boston diagnostic aphasia examination (BDAE). A moderate anomia was definitely present since 55/105 was the score he obtained at the confrontation naming test of the BDAE. Although these abnormalities existed in French and Turkish alike, the interpretation of the French language disorders must be tempered. Reading aloud seemed fluent in Turkish; the patient was French-illiterate. Comprehension and repetition were unimpaired. We never observed agrammatism, phonemic paraphasia, perseveration or articulatory disturbances

Finally, this patient exhibited marked memory impairment consisting in anterograde amnesia with total disorientation both for place and time, loss of learning ability and retrograde amnesia as, for instance, he could not remember his own job or his present address. The remote memory was also severely impaired since he could not recall either the date of his immigration to France 7 years before or his birthday. The Weehsler memory scale showed an MQ of 40 and the scores of the different subtests were: subtest 1: 4, subtest 2: 2, subtest 3: 0, subtest 4: 1, subtest 5: 1, subtest 6: 2, subtest 7: 0. The long-term memory for visual material (visual reproduction = 2) as well as verbal material (logic memory = 1) was disturbed. The score of the digit span was 4.

Evolution

Motor neglect began to improve approx. 4 weeks post-onset. Two months later, clinical examination still revealed dragging of the right leg, imbalance of the arm and slight deviation of gait but normal bimanual motor activity. Repeated motor examinations performed during the next 8 months showed the full disappearance of motor neglect although the patient still complained of a heavy and lazy right hand and clumsiness.

Behavioural abnormalities remained severe and his performances in language and memory tests remained unchanged. Ten months after the onset of his disease, a marked decrease in spontaneous activity and poor affect were still present. He had to be stimulated by his wife into day-to-day actions, to wake up, wash and dress, eat and even to switch on the television. Obsessive-compulsive behaviour was absent. This abulia was still associated with dynamic aphasia and memory impairment although a slight improvement was observed. Mild disturbances were observed in the BDAE confrontation naming test (score 75/105), but he totally failed twice at 2-monthly intervals in the verbal fluency subtest. The memory score on Wechsler's scale was 61. Disorientation for place and time had disappeared. He was able to remember his last visit to hospital and to recognize the medical staff.

Magnetic resonance imaging and angiography

The magnetic resonance imaging (MRI) was performed 2 months after the onset of symptoms. T1 (TR = 600 msec; TE = 9 msec) and T2 (TR = 3273 msec; TE = 80 msec) weighted sequences were used in coronal and axial planes (parallel to orbito-meatal line); only T1 weighted sequences were used in the sagittal plane. Sequential slices (5 mm) were scanned in the thalamic region. We attempted to localize the MRI abnormalities, above all in the sagittal plane, using a three-dimensional atlas of the human brain [52]. Small size lesions exhibiting hypointense signals on T1 weighted images and hyperintense signal on T2 weighted images, consistent with infarctions, were seen in the left thalamic and subthalamic regions. The largest, a paramedian lesion, extended medially from the inferior and external part of the dorsomedial nucleus (DM) to the internal part of the ventrolateral nucleus (VL) laterally. Between these limits, the intralaminar nuclei were probably involved and included the parafascicular and the centromedian nuclei, but the anterior pole of the thalamus was preserved (Figs 1, 2, 5 and 7). A second lesion, located in the posterior and lateral thalamus, involved the pulvinar (Figs 3, 6 and 7). Subthalamic lesions were noted in the vicinity of the substantia nigra and subthalamic nucleus (Fig. 2).

On the right side, numerous dotted lesions were observed in the medial part of the thalamus and subthalamic region. At least two of them were probably located in the DM (Figs 4 and 5). The precise localization of the others proved difficult.

Cerebral angiography revealed a total occlusion of the distal portion of the basilar artery.

Measurements of cerebral blood flow (CBF)

CBF was measured 4 weeks, 8 months and 10 months post-onset, under resting condition: the patient lay with eyes closed and ears plugged. The first and third measurements were carried out using the Xenon 133 (¹³³Xe) inhalation technique, the third measurement also included activation trials (see protocol). The second investigation included a combined measurement of CBF and cerebral oxygen consumption, and was carried out at 8 months post-onset using positron emission tomography (PET).

Xenon 133 procedure. The CBF measurement with the Xenon 133 (¹³³Xe) inhalation technique [33, 40] employed a device with 32 detectors positioned over both hemispheres (Novocerebrograph). CBF was measured from the washout curves by estimation of the initial slope index (ISI) [40]. The procedures of measurement during the resting state and activation trials, were identical to those previously reported in a similar case [45]. The positioning of the scanning probes is illustrated on Fig. 8.

Protocol of the activation paradigm. The patient was submitted to three CBF measurements with the ¹³³Xe inhalation technique: rest, left hand activation, right hand activation, with a 1 hr interval between each study. The activation measurements were made over an 11 min period by having the patient feel different objects with the same hand, each for 30 sec, for a total number of 22 objects. Adequate palpation of the objects was ascertained by direct observation of the patient during the examination and adequate object identification by having the patient enumerate some of the objects felt after the measurement had been completed, despite his memory disturbance. Seven healthy, right-handed control subjects, five men and two women aged from 37 to 80 years (average age, 62.4 years) underwent the same protocol for comparison. The patient and control subjects performed identically.

Statistical analysis. For the patient, as asymmetry indices observed previously between homologous probes in 11 control subjects in rest condition (average age, 31.2 years) were 5.16% + 3.5%, interhemispheric ISI differences, comparing each probe to its contralateral homologous probe were considered significant P < 0.05 if above 13% (mean + 2 SD).

An index of immediate reproductibility (second CBF/first CBF \times 100) has been previously calculated in 12 control subjects (average age, 33.4 years). The values were 102.2 + 2.2% and 103.5 + 4.9% for mean ISI and regional ISI, respectively. So mean ISI differences between baseline and activation were considered significant P < 0.05 if above 6.9% and regional ISI differences between baseline and activation were considered significant P < 0.05 if above 14.5% and P < 0.01 if above 19%.

Results in control subjects were studied using the Student's t-test on matched series.

PET scanning. PET images were obtained on the LETI TTV01 seven-slices time-of-flight tomograph, which has a resolution of $13 \times 13 \times 12$ mm at full width at half maximum. According to the principles of the oxygen-15 steady state inhalation technique [18, 28], oxygen-15-labelled gases ${}^{15}O_2$ and ${}^{15}CO_2$ were administered sequentially to provide two separate data sets which were incorporated to produce functional maps of CBF, oxygen extraction fraction (OEF) and cerebral oxygen consumption (CMRO₂). Serial blood samples were collected through a catheter placed in a radial artery at the onset of the study. Correction for attenuation was made using germanium-68/gallium-68 transmission scans. Of the seven scanned planes, five (OM + 10 to OM + 70) were examined by placing 3 cm² circular regions of interest (ROI) over cortical areas and the thalami. Following a protocol detailed elsewhere [48], ROI values were averaged to compute regional absolute values and right/left ratios of CBF, OEF and CMRO₂ prediction limits derived from 15 normal control subjects (average age, 44.5 years).

RESULTS

The patient

The results of the first and third CBF measurements are shown in Table 1. Regional percentage increase during activation vs baseline is graphically presented in Figs 9 and 10.

The first mean CBF, measured 4 weeks post-onset, was symmetrical. ISI values (47.2 and 47.4) were low for the patient's age as the normal mean ISI values for 8 normal subjects (aged 26–33, average age, 30.5 years) were 58.2 + 4.2, very close to those established by MATSUDA *et al.* (1984) [29]. No regional hypoperfusion area was detected, except in the right superior prerolandic area (probe 5: -14.8%).

The PET scan performed at 8 months showed a diffuse decrease of about 30% in cortical CBF and CMRO₂ throughout both hemispheres. This 30% decrease just failed to achieve statistical significance in reference to the control subjects, older than the patient. The OEF was normal. The analysis of cortical regions (frontal, parietal, central and temporal cortices) detected neither perfusional nor metabolic asymmetries, whereas the left thalamus exhibited a significant reduction in both CBF and CMRO₂ relative to the right thalamus.

The 10 month ¹³³Xe measurement still gave low flow values without mean interhemispheric asymmetry. The mean values (47.4 and 47.4) were identical to those recorded during the first CBF measurement. Left regional hypoperfusion was detected in the frontal (probes 1 and 2) and parietal (probes 13 and 15) areas.

During the activation study of the right hand, both left and right mean CBF values were unchanged (+3.7%; +0.8%). With respect to the rCBF values (Fig. 9), slight increases occurred, in the left anterior frontal (probe 1) and parietal (probe 13) areas, but not in the rolandic (probe 8, +8%) and prerolandic (probe 5, 0%) areas. No rCBF increases were seen in the right hemisphere.

During the activation study of the left hand, the values for right and left mean CBF again failed to significantly change (+5.7%; +0%). rCBF (Fig. 10) increased markedly in the following right hemisphere territories: superior prerolandic (probes 2, +22% and 5, +30%) and superior rolandic areas (probe 8, +28%) corresponding to the primary sensorimotor cortex for the hand. Significant increases in rCBF occurred in the following left hemisphere areas: anterior frontal (probe 1), middle prerolandic (probe 4) and middle rolandic (probe 7) zones.

Control subjects

Results are shown in Table 2. Percentage increase during activation vs baseline is graphically presented on Figs 11 and 12.

At rest, the average ISI was 41.1 in the left and 41.8 in the right, without significant asymmetry.



Figs. 1–3. MRI left sagittal T1 weighted slices scanned every 5 mm from the midline, showing a large anterior thalamic infarct (Figs 1 and 2), subthalamic lesions (Fig. 2), and a pulvinar infarct (Fig. 3).

Fig. 4. MRI right sagittal T1 weighted slice scanned near the midline, showing punctate medial thalamic infarcts.

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Figs 5, 6. MRI coronal T2 weighted slices scanned with a 10 mm interval, showing the left paramedian thalamic infarct and right punctate medial thalamic lesions (Fig. 5) and an infarct in the pulvinar (Fig. 6).

Fig. 7. MRI axial T2 weighted slice, showing two lesions sited in the pulvinar and paramedian thalamus, respectively.



Fig. 8. Position of the scanning probes.

Table 1. Patient's first CBF measurement, 4 weeks after the onset of symptoms and third CBF measurement 10 months after the onset of symptoms: ISI values at rest and during activation

	Rest				Activation					
	(4 weeks)		(10 months)		Right	hand	Left hand			
Condition	LH	RH	ĹĦ	RH	LH	RH	LH	RH		
Mean CBF	47.2	47.4	47.4	47.4	49.1	47.8	47.3	50.1		
Probes										
1	53	50	44	55	51*	53	54**	57		
2	44	49	49	41	47	46	48	50**		
3	47	47	51	51	50	53	50	56		
4	48	51	45	50	50	56	53**	56		
5	46	54	42	41	47	41	45	52**		
6	50	47	47	51	50	47	47	52		
7	48	50	47	48	53	53	56**	52		
8	49	49	48	43	52	48	51	55*		
9	43	47	48	45	47	48	47	44		
10	47	48	51	52	51	48	48	51		
11	47	45	50	47	52	51	55	45		
12	47	44	48	48	48	46	42	49		
13	45	47	45	53	52*	51	43	52		
14	44	43	48	52	47	46	48	49		
15	46	46	45	38	41	39	35	39		
16	50	42	50	43	49	39	36	42		

ISI increases during activation vs rest: *0.01 < P < 0.05; **P < 0.01; LH = left hemisphere; RH = right hemisphere.

Motor activation tasks resulted, whichever hand was used, in a bilateral and roughly symmetrical increase in the mean hemispheric flow values. With respect to the regional values, the highest increase was situated over the superior rolandic areas (probe 8) contralateral to the hand in action: left hemisphere-right hand, +22% (P<0.01); right hemisphere-left hand, +19% (P<0.01). In each control subject taken individually, motor hand activation resulted in a significant rCBF increase in the contralateral superior rolandic area. Furthermore, the regional values increased diffusely on both sides.

DISCUSSION

The particular interest of our patient was two-fold: clinically, the constellation of signs and symptoms surrounding his motor neglect; and haemodynamically, the disturbances in



Fig. 9. Patient; CBF following activation of the right hand. Graphic presentation of ISI changes. Each number signifies the percentage increase during activation vs resting conditions. White: not significant; hatched: 0.01 < P < 0.05; black: P < 0.01.



Fig. 10. Patient; CBF following activation of the left hand. Same remarks as Fig. 10.

cerebral perfusion associated with his MN. These two aspects will be discussed in turn. While this patient had a spectrum of neuropsychological deficits (dynamic aphasia, abulia and amnesia), it should be emphasized that he presented with pure MN, in the absence of spatial and sensory neglect. As such, this is a relatively rare case which would speak against the hypothesis that the motor component of neglect is a function of hemispheric hypo-arousal [21]. Another aspect of this patient's MN should be underlined: the duration of the motor abnormalities which persisted for the 2 months following the ictus. These motor signs were severe, mimicked a hemiplegia, and only improved transiently following sustained verbal commands.

The normal transience of, and functional recovery from, MN has been explained by the rapid intervention of alternative pathways [25], either from the healthy ipsi- [54] or contralateral hemispheric tissue. Delayed recuperation from MN, after callosotomy in monkey, might suggest interhemispheric mechanism of recovery [61]. Accordingly, language motivation and its role in motor intention could be of crucial importance [25]. In the present case, dynamic aphasia (defined as a paucity of spontaneous language) and MN were not only associated but equally striking. The failure of the mechanisms for the arousal or activation of cortical language, subserved in part by the thalamus [30, 32, 39], might

	Left hemisphere				Right hemisphere							
	Rest		Act, right hand		Act. left	Act. left hand		est	Act. right hand		Act. left hand	
Р	Х	SD	x	SD	Х	SD	х	SD	x	SD	Х	SD
1	43.1	4.91	46.6	6.50	46.1	4.10	41.6	8.00	46.0**	7.72	47.00***	6.38
2	40.9	4.11	46.3*	6.60	45.3***	5.99	38.7	8.90	43.4	8.12	45.1*	7.47
3	42.4	5.56	48.1*	9.79	48.7***	7.27	45.9	6.82	50.6*	8.98	45.7***	17.75
4	42.9	6.39	49.3***	6.05	49.0***	4.55	43.9	5.98	49.1***	8.63	48.1**	6.74
5	41.7	7.67	48.3**	7.61	47.3**	4.64	42.1	6.79	48.5***	7.48	48.4***	5.50
6	42.0	6.19	46.7***	8.46	46.7***	7.95	43.9	7.10	49.1**	7.47	51.0***	7.79
7	41.4	7.63	46.4**	8.48	46.1**	7.52	41.9	6.44	47.6***	7.87	48.0***	6 38
8	39.0	8.97	46.3***	7.80	43.6**	7.63	41.3	6.37	45.4***	8.32	48.6***	5.59
9	37.8	8.23	42.4	12.34	46.1**	7.52	40.7	9.69	42.4	9.86	44.7*	10.53
10	40.3	8.61	46.3*	9.71	47.6**	7.61	42.4	9.60	46.4*	9.31	47.9***	8.05
11	42.7	10.26	48.4***	10.56	50.4**	13.10	43.3	12.19	46.1	10.37	49.0	10.91
12	41.7	10.10	45.9**	11.19	47.6**	11.87	41.7	8.32	47.7***	10.22	48.9***	8.88
13	39.9	7.03	46.7**	8.54	46.4*	8.38	41.4	7.37	47.0***	9.92	47.6***	6.95
14	38.4	5.62	43.0	6.43	44.1**	5.55	39.6	6.73	44 4*	7 23	46 7***	6.42
15	40.4	6.29	45.9***	9.41	46.9***	6.87	39.3	10.97	43.3***	9.48	44.0**	9.17
16	41.3	11.18	43.6**	10.75	46 7**	10.26	38.9	10.95	44.2	13.29	46.3*	12.4
M	41.1	6.81	46.2***	8.35	46.8***	7.29	41.8	7.08	46.6***	8.08	47.7***	7.06

Table 2. Control subjects (n=7): mean regional ISI values at rest and during motor activation tasks

ISI are given in ISI units as mean (X) and standard deviation (SD). The *P* values for paired *t*-test comparing the difference (activation vs resting) are as follows: *0.02 < P < 0.05; **0.01 < P < 0.02; ***P < 0.01.

P = probe; M = hemispheric mean; Act. = activation.



Fig. 11. Controls; CBF following activation of the right hand. Same remarks as Fig. 10.

explain the severity of the MN in this patient. This hypothesis would be supported by the patient's minimal reactivity to strenuous verbal stimulation.

Other than the language-related disorders, this patient presented with behavioural abnormalities that consisted of apathy, lack of initiative and total disregard for his condition. Similar abnormalities have been reported following bilateral lesions of the lentiform nuclei [1, 23, 26, 27] which, in one case, extended unilaterally to the anterior nuclei of the thalamus [27]. The PET studies performed in this cohort of patients demonstrated a relative prefrontal hypometabolism [27] interpreted as a 'deafferentation' process following a bilateral disruption of the 'association circuit' that includes the caudate nucleus, pallidum, substantia nigra along with the ventralanterior and dorsomedial nuclei of the thalamus [27]. Within



Fig. 12. Controls; CBF following activation of the left hand. Same remarks as Fig. 10.

this hypothetical scheme, the present case may have had an interruption of the association circuit, centred on the dorsomedial thalamic nuclei with its dense projections to the prefrontal cortex [10]. Like the MN, this patient's impressive abulia could only be ameliorated by continuous external stimulation.

The third major neuropsychological deficit of this patient was a global and devastating amnesia. Verbal and visuo-spatial memory loss coexisted. No significant recovery was evidenced over a 10-month period. These features are consistent with bilateral thalamic lesions [3, 12, 13, 20, 38, 46, 49, 57, 62]. However, unlike the deactivated processes of dynamic aphasia and abulia, the associated memory deficit shed little light on our understanding of MN.

The bilateral and diffuse trend to decrease in cortical blood flow concords with more extensive studies on the cerebrovascular consequences of uni-[4] and bilateral [29] thalamic lesions. The trend to low perfusion was unchanged after 10 months, which paralleled the findings at PET scan. The lack of modification of CBF values was associated to a persisting neuropsychological deficit. Following subcortical lesions, the severity of aphasia or neglect has been correlated to the degree of cortical hypoperfusion [37] which apparently recovers *pari passu* improvements in the neuropsychological and clinical status [55].

But the most striking and instructive CBF abnormalities were seen, not in the resting state, but during the performance of the manual sensorimotor tasks. Left hand movements activated the right rolandic and prerolandic areas; identical execution with the right hand failed to change the homologous areas in the left hemisphere. Simple motor activity is known to increase flow in the contralateral primary sensorimotor cortex [19, 22, 34, 41] and both supplementary motor areas (SMA) [19, 35, 42, 53], the latter not being measurable with our 133 Xe technique. When manual activity is combined with object manipulation, there is a recruitment in premotor areas [40], evidenced by increased CBF. It has been argued that the premotor activation represents the sensory guidance of the more complex tasks that involve motor manipulation [43, 44]. In a previous case of pure and transient left-sided MN, object manipulation failed to activate CBF in the contralateral rolandic and prerolandic cortices [45], even after the resolution of the MN syndrome. Based on our present and previous case, we postulate that the failure of hand movement to activate focal CBF is a 'functional deficit' that is characteristic of MN. This abnormality, which persists despite normal motor performance and relatively normal cortical flow, might be explained by one or two mechanisms: either there is a partial cortical activity (normal rolandic resting flow) enabling sufficient motor execution; or ipsi- and/or contralateral alternative circuits that prime the cortex for motor execution have been recruited. These two cases of MN are clearly distinguished from the situations of permanent hemiparesis and aphasia of subcortical origin in which there is a permanent, steady state oligaemia [50, 51].

A syndrome of neglect has been described in the non-human primate. As with the present case, cortical metabolism is relatively preserved although significant decreases were noted in the caudate nucleus, putamen, pallidum, superior colliculus and dorsomedial nucleus of the thalamus [15]. This aspect would be worthy of investigation in MN patients with strictly unilateral subcortical lesions. Even though PET scan showed a left thalamic hypometabolism, the resolution of the camera was such that this hypometabolism could be of structural and/or functional origin.

This MN patient is noteworthy for two specific reasons. Firstly, the MN was of unusual severity and duration and paralleled the dynamic aphasia and abulia. Though difficult to dissect the relative importance in one patient, it is not improbable that these neuropsychological deficits, involved in activation, play a role in the recovery from MN. Secondly, our CBF data indicates that the recovery from MN is much less complete than that suggested from the objective evaluation of the motor performance. Indeed, the long-lasting failure of motor tasks to evoke a cortical activation might subserve the persistent complaint of MN patients that previously automatic activity became voluntary movements and expressed in comments such as 'I have to think before I move my hand' [25, 45].

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