Primary Somatosensory Cortex Activation Is Not Altered in Patients With Ventroposterior Thalamic Lesions
A PET Study

Philippe Remy, MD, PhD; Mônica Zilbovicius, MD, PhD; Pierre Cesaro, MD, PhD; Pierre Amarenco, MD; Jean-Denis Degos, MD; Yves Samson, MD

Background and Purpose—We know remarkably little about the mechanisms underlying cortical activation. Such mechanisms might be better understood by studying the effect of well-localized lesions on the cortical activations in simple paradigms.

Methods—We used $H_2^{15}$O and positron emission tomography to measure regional cerebral blood flow (rCBF) at rest and during hand vibration in 7 patients with unilateral thalamic lesion involving the ventroposterior (VP) somatosensory thalamic relay nuclei. We compared the results with those obtained in 6 patients with thalamic lesions sparing the VP nuclei and 6 healthy controls.

Results—The patients with VP lesions had a selective hypoperfusion at rest in the ipsilesional primary sensorimotor cortex (SM1). This hypoperfusion was significantly correlated with the degree of contralateral somatosensory deficit. Despite this deafferentation, the ipsilesional SM1 was normally activated by the vibration of the hypesthetic hand.

Conclusions—The fact that a lesion of the somatosensory thalamic relay nuclei alters the rCBF at rest in SM1 but not its activation by hand vibration indicates that the mechanism of cortical activation is complex, even in the case of simple sensory stimulation. In addition, a dissociation may occur between obvious neurological deficits and apparently normal activation patterns, which suggests that activation studies should be interpreted cautiously in patients with focal brain lesions. (Stroke. 1999;30:2651-2658.)

Key Words: cerebral blood flow • somatosensory cortex • thalamus • tomography, emission computed • vibration

Positron emission tomography (PET) and functional MRI are useful for mapping the functions of the human brain because they reveal the activation of precisely localized and often distributed cortical areas during sensory, motor, or cognitive tasks.1,2 However, we still know remarkably little about the mechanisms underlying cortical activation. For example, it seems obvious that moving the right hand would activate motor cortical areas in the left hemisphere, but it remains unknown how these areas are selectively activated when a subject decides to move. Yet, it has been recently shown that this process may be disrupted in many diseases, because abnormal patterns of cortical activations have been described in patients recovering from focal brain lesions3–7 and in a variety of diseases, ranging from Parkinson’s disease and dystonia8–10 to schizophrenia and autism.11,12 However, to interpret these abnormal activations it is necessary to better understand what leads to the activation of a specific cortical area. One way to address this issue is to investigate how well-localized lesions modify the cortical activations in simple paradigms.

One of the most simple activation paradigm studied is unilateral hand vibration, which normally results in a strong and robust activation of the contralateral primary sensorimotor cortex (SM1).13–17 These activations may simply reflect the processing of information conveyed to specialized somatosensory cortical areas by the ascending somatosensory pathways. A major subcortical relay of these pathways is the ventroposterior (VP) group in the thalamus, which receives many extralemniscal and almost all lemniscal ascending fibers.18–22 Accordingly, a lesion involving the VP nuclei may impair the somatosensory cortical activations during contralesional hand vibration. To investigate this issue, we studied the effects of thalamic lesions on the cortical activations induced by hand vibration in 2 groups of patients whose lesion involved or spared the specific somatosensory relays in the thalamus (ie, the VP group). Our results do not fit with the proposed model and suggest that cortical somatosensory activation do not depend on the VP integrity and may thus involve more complex and parallel systems.

Received August 9, 1999; final revision received September 24, 1999; accepted September 24, 1999.
From the CEA, Service Hospitalier Frédéric Joliot (P.R., M.Z., Y.S.), Orsay; Service de Neurologie, CHU Henri-Mondor (P.R., P.C., J-D.D.), Créteil; Service de Neurologie, CHU Lariboisière (P.A.), Paris; Urgences Cérébro-Vasculaires, CHU Pitie-Salpêtrière (Y.S.), Paris, France.
Correspondence to Dr Philippe REMY, CEA, Service Hospitalier Frédéric Joliot, 4, Place du Général Leclerc, 91401 Orsay Cedex, France. E-mail remy@shfj.cea.fr
© 1999 American Heart Association, Inc.
Stroke is available at http://www.strokeaha.org

2651
Subjects and Methods

Subjects

These studies were approved by the local Ethical Committee for Human Studies, and all subjects gave their informed consent. Handedness was assessed with the Edinburgh inventory.²³

Patients

We studied 13 right-handed patients aged 25 to 83 years, with a unilateral thalamic lesion (stroke in 12 subjects and abscess in 1) demonstrated by CT scan or MRI. They were divided into 2 groups according to the presence (n=7; 62±14 years old) or absence (n=6; 46±12 years old) of a lesion of the VP nuclei of the thalamus. The existence of a lesion of the VP thalamus was supported by the following criteria: (1) a somatosensory deficit contralateral to the thalamic lesion, and (2) the reconstruction of the lesion obtained from individual MRI performed on the day of the PET examination (see below). As shown in Figure 1, the VP was involved in the lesion in all patients with a contralateral somatosensory deficit (patients 1 through 7). The 6 patients who had no somatosensory deficit but, rather, cognitive symptoms had lesions of more anteromedial parts of the thalamus that always spare the VP (Figure 2).

Control Subjects

Six healthy right-handed male volunteers aged 20 to 40 years (mean±SD, 29±8 years) were studied. All had a normal clinical examination and a normal brain MRI.

Clinical Examination

On the day of the PET study, all patients had a bedside examination carried out by a single examiner (P.R.) who used a fixed protocol.¹⁷ The Canadian Neurological Scale²⁴ score was used to measure the degree of motor deficit. In addition, we used a standardized scale to determine the degree of permanent somatosensory deficit.¹⁷ The total score ranges from 0 to 70, with increasing values indicating increasing deficit. The following somatosensory modalities were tested. (1) Discriminative touch (0–21): This included (a) measuring stereognosia (0–3), by placing 3 common objects successively in each hand of the subjects with each incorrect recognition scored as 1 point; (b) graphesthesia (0–6), for which 3 numbers were drawn on the forearm and 3 others on the thigh and each error in recognizing the number was scored as 1 point; and (c) 2-point discrimination (0–12), assessed using Weber’s compasses. Two different areas were tested for the upper limb (fingertip of the third finger and forearm) and lower limb (dorsal foot and anterior part of the thigh). The first distance at which 2 points were discriminated on at least 3 different trials was measured. This distance was then compared for each
cutaneous area tested with the measures obtained in the intact side, and a percent difference was determined as follows: \(100 \times \text{impaired side} - \text{normal side})/\text{normal side}\). For each site examined, 1, 2 or 3 points were scored if this percentage exceeded 30%, 50%, or 100%, respectively. (2)

Vibration and proprioception (0 –24):
The subject had to detect a vibratory stimulus applied with a tuning fork at 3 different levels on the upper and the lower limb. For each of these trials, the patients scored 1 point if the sensation was reported to be diminished compared with the contralateral (intact) side and 2 points if they did not detect the vibration. In addition, 1 point was scored if the vibrator used for the PET stimulation was felt with less intensity in the hypesthetic hand than in the normal hand and 2 points if the vibration was not perceived. The perception of passive movements was assessed by asking the patients to identify the directions of 5 up-down movements applied to the second finger and first toe. Each error was scored as 1 point. (3) Temperature (0 – 6):
The thermal sense was assessed with 2 different test tubes filled with cold water (\(<5^\circ\text{C}\)) or warm water (\(>40^\circ\text{C}\)). The subject had to recognize the temperature (“cold” or “warm”). Three trials were performed for each limb. The score corresponds to the number of errors.

Pin-prick (0 –10):
For each limb 5 stimulations were performed. The subject had to identify the stimulus (pin or prick). The score corresponds to the number of errors.

Pain (0 – 4):
1 point was scored if the patient had transient pain in the hemibody contralateral to the brain lesion; 2 points if the pain was permanent; 3 if the pain increased during hand vibration; and 4 in the case of severe central pain, such as complete Déjerine-Roussy syndrome. (6) Paresthesia (0 –5):
Paresthesia was scored as 1 if transient and 2 if continuous, for each limb. One point was added if paresthesia occurred to the face.

The subjects were blindfolded during all testing. Each hemibody was similarly tested, and the score is given for 1 side.

Brain Imaging Studies
These studies were performed 1 to 37 months (mean±SD, 11±10 months; n=13) after the occurrence of the lesion. This delay did not significantly differ between the groups (with somatosensory deficit, 10±9 months; without somatosensory deficit, 13±12 months).

Brain imaging studies consisted of MRI and PET images acquired on the same day for each subject. The detailed information obtained from the MRI allowed us to perform an individualized anatomic localization of brain regions for the functional PET image analysis for each subject.17,25

Anatomic Images
MR images were obtained with a 0.5-T MR imager (MRMAX, General Electric). Each subject was positioned so that MRI axial slices were parallel to the bicommissural line (AC-PC),26 which was verified on a midsagittal image. Skin marks were applied where indicated by the MR imager positioning laser. Contiguous T1-weighted 3-mm-thick axial slices and 5-mm-thick coronal T2-weighted slices were then obtained throughout the entire brain.

Functional Images
Scanning was performed with a LETI-TTV03 tomograph (CEA), the characteristics of which have been described elsewhere.27 Briefly, this scanner collects 7 parallel transaxial planes, 9 mm thick and 12 mm apart, with a reconstructed 7-mm in-plane resolution (full width at half maximum).27 We used the skin marks drawn on the subjects’ face in the MR imager to ensure an exact repositioning of the head in the PET session. In addition, the crossed laser beams permanently attached on the PET tomograph allowed us to monitor the subjects’ head position throughout the examination. During each PET scan, the room was darkened and the only ambient noise was the cooling system. The effects of radiation attenuation by the head were corrected using a transmission scan collected during exposure to a \(^{68}\text{Ge}\)germanium source. In each condition, an rCBF study was performed with a method derived from the \(^{15}\text{O}\) autoradiographic method, except no arterial catheters were used.28 We injected an intravenous bolus of 3 mL of saline containing 2960 to 3700 MBq \(^{15}\text{O}\)-labeled water, and an 80-second scan was initiated when the tracer bolus entered the brain. The first frame showing the arrival of...
radioactivity in the brain was identified. From this time point the radioactive counts for the ensuing 80 seconds were summed to generate rCBF images. There was a 15-minute interval between each rCBF measurement.

Tasks
Three different conditions were studied: (1) rest (subjects were asked to remain in a resting awake state), (2) vibration of the right hand, and (3) vibration of the left hand. The order of conditions (2) and (3) was counterbalanced across subjects. One of the 6 controls received stimulation only on the left hand. The stimulations were performed by a single examiner using a vibrator that produces 2-mm-amplitude movements with a frequency of 130 Hz (Daito), and involved all the fingers. The subjects were asked not to grasp the vibrator, but instead to let the examiner maintain their fingerpads in contact with the vibrator. To avoid any surprised reaction to the task, subjects

<table>
<thead>
<tr>
<th>TABLE 1. Patients With VP Thalamic Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
</tr>
<tr>
<td>Age/Sex</td>
</tr>
<tr>
<td>Lesion</td>
</tr>
<tr>
<td>Side</td>
</tr>
<tr>
<td>Type</td>
</tr>
<tr>
<td>Canadian Neurological Scale score (0–15)</td>
</tr>
<tr>
<td>Somatosensory Score</td>
</tr>
<tr>
<td>Discriminative touch (0–21)</td>
</tr>
<tr>
<td>Graphesthesia (0–6)</td>
</tr>
<tr>
<td>Stereognosis (0–3)</td>
</tr>
<tr>
<td>2-point discrimination (0–12)</td>
</tr>
<tr>
<td>Vibration/position sense (0–24)</td>
</tr>
<tr>
<td>Tuning fork (0–12)</td>
</tr>
<tr>
<td>Viberator (0–2)</td>
</tr>
<tr>
<td>Position sense (0–10)</td>
</tr>
<tr>
<td>Lemniscal score (0–45)</td>
</tr>
<tr>
<td>Temperature (0–6)</td>
</tr>
<tr>
<td>Pin-prick (0–10)</td>
</tr>
<tr>
<td>Pain (0–4)</td>
</tr>
<tr>
<td>Extralemniscal score (0–20)</td>
</tr>
<tr>
<td>Paresthesia (0–5)</td>
</tr>
<tr>
<td>Total Somatosensory Score (0–70)</td>
</tr>
</tbody>
</table>

Hem indicates hemorrhage; Isch, ischemic stroke; and Lac, lacunar stroke. The Canadian Neurological Scale score measures the motor deficit. The somatosensory score is described in Subjects and Methods; increasing values indicate increasing deficit.

<table>
<thead>
<tr>
<th>TABLE 2. Patients With Anteromedial Thalamic Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
</tr>
<tr>
<td>Age/sex</td>
</tr>
<tr>
<td>Lesion</td>
</tr>
<tr>
<td>Side</td>
</tr>
<tr>
<td>Type</td>
</tr>
<tr>
<td>Canadian Neurological Scale score (0–15)</td>
</tr>
<tr>
<td>Aphasia</td>
</tr>
<tr>
<td>Confusion</td>
</tr>
<tr>
<td>Total somatosensory score (0–70)</td>
</tr>
</tbody>
</table>

Hem indicates hemorrhage; Isch, ischemic stroke; and Lac, lacunar stroke. The Canadian Neurological Scale score measures the motor deficit. The somatosensory score is described in Subjects and Methods.

*In patient 9, the stroke was discovered on CT scan performed for hypertension and headache.
were acclimated to the vibrator before the PET session, and stimulation began 30 seconds before tracer injection.\textsuperscript{17}

\textbf{Data Analysis}

\textbf{Regions of Interests}

We focused our interest on the primary sensorimotor cortex and used an individual anatomic analysis to take into account the anatomic variability between brains of different subjects.\textsuperscript{30} PET and MRI data were transferred to a VAX computer (Digital Equipment Corporation) and the MRI-PET images put in register as described previously.\textsuperscript{17,25} Briefly, 3 digitized 3-mm-thick axial slices of the MRI were combined to obtain images with the same thickness as the PET slices (9 mm). MRI and PET were coregistered with isodensity contours and custom software allowing in-plane translation and rotation. Using this software, MRI isocountours were then superimposed on PET images in all slices. The accuracy of this registration was assessed by checking that a successful superimposition was simultaneously obtained at all brain levels. Such a simultaneous superimposition cannot be achieved if registration error exceeds 5 millimeters in any of the $x$, $y$, and $z$ axes. This procedure was repeated for each set of rCBF images to rule out head movements between rCBF measurements.

The primary sensorimotor cortex (SM1) was a priori defined in each hemisphere for analysis. In addition, a global cortical area was defined in each hemisphere, including all cortical areas that were not analyzed for their involvement in sensorimotor function (we excluded the parietal cortex). This global cortical area (mainly temporal, frontal, and occipital cortex) was defined as a cortical ribbon having a 14-mm thickness. All regions of interest (ROIs) were drawn on individual MR images and copied onto corresponding PET images. Their boundaries were determined according to the identification of the main sulci.\textsuperscript{17,25,29,31,32}

\textbf{Data Analysis}

Since the change in local tissue radioactivity is linearly related to blood flow, relative changes in tissue activity are taken to indicate relative changes in blood flow.\textsuperscript{28} One should note, however, that the relationship between rCBF increase and neuronal activation is not necessarily linear.\textsuperscript{33} To correct for intercondition changes in the global activity of the brain, all PET images were normalized so that the statistical significance of this asymmetry was analyzed with a Student $t$ test. Finally, we made a correlation analysis between clinical scores and the rCBF parameters.

\begin{figure}
\centering
\includegraphics[width=\textwidth]{fig3.png}
\caption{Degree of asymmetry of SM1 activation in the 3 groups of subjects. The asymmetry between response to contralateral hand vibration obtained in the normal and lesioned hemispheres ($A_{\text{Lm}}$, normal—lesioned side) is indicated for the 3 groups of subjects. In controls, the right-left side was calculated. Bars indicate the mean. No marked asymmetry was observed between normal and lesioned hemisphere responses, even in patients having a VP lesion with a contralateral somatosensory deficit.}
\end{figure}

\textbf{Results}

\textbf{Activations Induced by Hand Vibration}

\textbf{Control Subjects}

In control subjects, hand vibration strongly and significantly ($P<0.001$, Student $t$ test) activated the contralateral SM1 when compared with rest: left SM1, $+12.8 \pm 4.6\%$; right SM1, $+12.7 \pm 4.0\%$. The asymmetry between activation obtained in left and right SM1 was $0.0 \pm 4.5\%$ (range $-6.8\%$ to $+5.5\%$, $n=5$; Figure 3).

\textbf{Patients}

\textbf{Patients With VP Lesion}

The vibration of the hypesthetic hand significantly activated the contralateral SM1 ($+9.2 \pm 6.2\%$, $P<0.01$). SM1 activation in the lesioned hemisphere did not significantly correlate with any of the somatosensory scores. More specifically, the vibratory score was not significantly correlated with the asymmetry of response in SM1 ($A_{\text{Lm}}$, $r=0.48$, $P=0.29$) or with the amplitude of activation in SM1 in the lesioned hemisphere ($r=-0.15$, $P=0.76$). In addition, there was no correlation between the level of activation in SM1 and the degree of rCBF asymmetry measured at rest ($r=0.27$, $P=0.57$).

The vibration of the normal hand of these patients induced comparable activations in the contralateral SM1 ($+9.3 \pm 3.9\%$, $P<0.001$). The activations were thus symmetrical in the normal and lesioned hemisphere, because the amplitude of asymmetry in SM1 ($A_{\text{Lm}}$) was $0.1 \pm 4.8\%$ (range $-7.4\%$ to $+6.5\%$). This asymmetry was comparable with that observed in controls ($0.0 \pm 4.5\%$, range $-6.8\%$ to $+5.5\%$; Figure 3 and 4).
Patients Without VP Lesion

In patients without somatosensory deficit, the SM1 activation induced by contralateral hand vibration was significant in the lesioned \((+13.5 \pm 4.4\% , P < 0.001)\) and in the normal \((+12.1 \pm 5.6\% , P < 0.01)\) hemispheres.

Between-Group Comparisons

No significant difference or interaction was found when comparing the activations observed in SM1 of the different groups, using an ANOVA with the group as intersubject variable and the hemisphere as intrasubject variable \((F_{2,15} = 1.3, P = 0.3)\). In addition, since the SD value of the activation of SM1 in the lesioned hemisphere is higher than in the other groups, we compared these SD values with an F test. This test demonstrated no significant difference in the SD values of SM1 activation between patients with VP lesion and patients without VP lesion \((F_{5} = 1.82)\) or controls \((F_{5} = 1.99)\).

Finally, in controls and in the normal hemisphere of the patients, there was no correlation between age and SM1 activation induced by hand vibration \((d f = 17, r = -0.3, P = 0.2)\).

Asymmetry at Rest

We found no significant asymmetry at rest in the control subjects. The patients with VP lesion had a slight rCBF asymmetry in the cortical ribbon \((AI_{\text{rest}} = 2.7 \pm 1.5\% , P < 0.01\), Student t test\), and a marked asymmetry in SM1 \((7.3 \pm 5.2\% , P < 0.01)\). The patients without VP lesion had a marked rCBF asymmetry in the cortical ribbon \((AI_{\text{rest}} = 6.6 \pm 2.1\% , P < 0.001)\). The comparison of asymmetries between the 2 groups of patients showed a significant interaction between patients and regions \((F_{1,11} = 15.2, P < 0.005)\), because the AIrest was higher in the SM1 region of patients with VP lesion than in the patients without VP lesion, whereas the reverse was found for the cortical ribbon \((7.3 \pm 5.2\% \text{ and } 1.4 \pm 3.4\% , \text{ respectively})\).

The rest asymmetry in SM1 of patients with VP lesion was significantly correlated with the vibration-proprioception subscore of the somatosensory scale \((d f = 5, r = 0.96, P < 0.0005\); Figure 5\), the lemniscal score \((r = 0.85, P = 0.01)\), and the total score of somatosensory deficit \((r = 0.80, P = 0.03)\).

Discussion

The main result of this study is that patients with thalamic lesions involving the VP thalamic somatosensory relays have a normal activation of somatosensory cortical areas during the vibration of the contralesional, hypesthetic hand. The activation of SM1 is symmetrical to the activation during the vibration of the normal hand in the same subjects and lies in the range of activations observed in controls or in patients with thalamic lesions outside the VP somatosensory relays. The normal response to hand vibration in patients with VP lesion occurs despite the deafferentation of somatosensory cortical areas demonstrated by the clinical somatosensory deficit and the decreased rCBF observed in the somatosensory cortical areas during the resting state. These findings will be discussed below.

In this study, the thalamic lesions appear to have little effect on the rCBF SM1 activation provoked by hand vibration but induce different patterns of rCBF abnormalities at rest in the 2 groups of patients. The patients without VP lesion had a widespread cortical hypoperfusion ipsilateral to the thalamic lesion, as evidenced by a significant rCBF asymmetry in the cortical ribbon. This widespread effect is a
well-known consequence of thalamic lesions involving non-
specific activating thalamic nuclei,35–37 which were likely
damaged in these patients. This widespread cortical hypoper-
fusion contrasts with the selective hypoperfusion at rest of the
ipsilesional SM1 found in the patients with VP lesion. The
latter indicates the local reduction of the synaptic activity at
rest in this region, confirming its partial deafferentation from
the specific somatosensory thalamic inputs due to the VP
lesion.38–40 Accordingly, we found a significant correlation
between the SM1 rCBF asymmetry at rest and the somato-
sensory deficit in these patients.

Despite the clear-cut effect on rCBF at rest, the thalamic
lesions appear to have little effect on the rCBF cortical
activations induced by hand vibration. The SM1 activation
was normal in the 2 groups of patients, even in the patients
with VP lesion during the vibration of the hypesthetic hand,
which was much less perceived than the vibration of the
normal hand. Despite this clear asymmetry of perception, the
level of activation was symmetric in both hemispheres of
patients with VP lesion (Figures 3 and 4). Thus, the interrup-
tion of the somatosensory input at the VP level alters the
perception of vibration and the synaptic activity at rest in
SM1 but does not impede the activation of this region by
hand vibration. These results might be limited by the lack of
absolute rCBF quantitation, since this normal activation is
associated with a lower absolute rCBF in this region both
during rest and hand vibration. However, the subjects with
VP lesion still have a preserved ability to activate their
ipsilesional SM1 despite the lesion of the somatosensory
relays at the thalamic level. One possible explanation is that
the SM1 region is only partially deafferentated. Although
somatosensory evoked potentials could not be performed in
our patients to assess the amount of residual connectivity
between VP thalamus and SM1, it is evident that none of the
patients had a complete anesthesia. However, this hardly
explains the normal SM1 activation, because the level of this
activation did not correlate with the score of somatosensory
deficit. For example, patient 7, who had the most severe
deficit of vibration/position sense (score 21/24), still had a
7.7% increase of blood flow in SM1 during vibration of the
hypesthetic hand. In addition, the SM1 rCBF increase in-
duced by hand vibration is symmetrical in both hemispheres
of patients with VP lesion despite the clear asymmetry of
perception. These results are finally in line with those of a
previous report41 which show that the complete section of the
lemniscal pathways at the spinal cord level does not abolish
the SM1 activation induced by vibration. Taken together,
these data indicate that the cortical activation caused by
contralateral vibration does not require the integrity of the
well-identified VP thalamocortical circuitry. They suggest
that other ascending systems may play a role in specific
cortical activation. In line with this hypothesis, we recently
reported17 that SM1 activation induced by hand vibration is
abolished in patients with tactile extinction who present
considerably fewer somatosensory deficits than patients with
VP lesion. Thus, even in this simple paradigm, cortical
activation emerges as a complex phenomenon that might be
viewed as a cortical “gating” mechanism: the light-up of
specialized cortical areas will allow them to process the flow
of information conveyed by modality-specific thalamic relay
nuclei. Although hypothetical, this view is consistent with the
effects of selective attention on the level of cortical activa-
tion, which are now established in different sensory activation
paradigms, including hand vibration.14,42,43 Little is known
about the underlying neuroanatomic bases of these activation
mechanisms. Here, some parallel lemniscal ascending path-
ways44 might have been spared by the VP lesion. Alterna-
tively, the extralemniscal system might be involved, since,
unlike the lemniscal system, it sends divergent fibers (ie,
spino-reticulothalamic and spinomesencephalic) to extrathal-
lamic targets, which were not damaged in our patients. But
how some of these collaterals are able to activate specific
somatosensory cortical maps— through either dedicated
pathways or interactions with different cortical activating
systems—remains to be investigated. This will become an
important field of clinical research, since understanding
the mechanisms of cortical activation may be a major step to link
localization and function in the human brain. Here for
example, the preserved activation of SM1 contrasting with its
hypoperfusion at rest might be an important cue to understand
the dissociation between preserved awareness and decreased
perception of somatosensory stimuli, which is a typical
clinical feature of patients with VP lesion.

Acknowledgments
The authors thank Ken Moya, Dr V. Biousse, Dr F. Bolgert, Pr J.-C.
Willer, M. Crouzel, L. Laurier, and B. Martins for their help.

References
1. Frackowiak RSJ, Friston KJ. Functional neuroanatomy of the human
brain: positron emission tomography: a new neuroanatomical technique.
The functional anatomy of motor recovery after stroke in humans: a study with
4. Weiller C, Ramsay SC, Wise RSJ, Friston KJ, Frackowiak RSJ. Indi-
vidual patterns of functional reorganization in the human cerebral cortex
5. Weder B, Seitz RJ. Deficient cerebral activation pattern in stroke
K, Woods RP, Noth J, Diener HC. Recovery from Wernicke’s aphasia: a
S, Chain F, Rancurel G, Samson Y. Recovery from non-fluent aphasia
after melodic intonation therapy: a PET study. Neurology. 1996;47:
1504–1511.
8. Playford ED, Jenkins IH, Passingham RE, Nutt J, Frackowiak RSJ,
Brooks DJ. Impaired mesial frontal and putamen activation in Parkinson’s
disease: a positron emission tomography study. Ann Neurol. 1992;32:
151–161.
9. Tempel LW, Perlmutter JS. Abnormal cortical responses in patients with
10. Ceballos-Baumann AO, Passingham RE, Marsden CD, Brooks DJ. Motor
Effects of auditory stimulation on regional cerebral blood flow in autistic
Dopaminergic modulation of impaired cognitive activation in the anterior
13. Fox PT, Burton H, Raichle ME. Mapping human somatosensory cortex
Primary Somatosensory Cortex Activation Is Not Altered in Patients With Ventroposterior Thalamic Lesions: A PET Study
Philippe Remy, Mônica Zilbovicius, Pierre Cesar, Pierre Amarenco, Jean-Denis Degos and Yves Samson

Stroke. 1999;30:2651-2658
doi: 10.1161/01.STR.30.12.2651
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1999 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/30/12/2651

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/