BRAIN GLUCOSE METABOLISM IN BORDERLINE PERSONALITY DISORDER

JOSÉ MANUEL DE LA FUENTE,*† SERGE GOLDMAN,+ ETIENNE STANUS,* CORO VIZUETE,‡ IGNACIO MORLÁN,§ JULIO BOBES¶ and JULIEN MENDLEWICZ*

*Department of Psychiatry, Erasme Hospital, Free University of Brussels, Belgium; †PET/Biomedical Cyclotron Unit, Erasme Hospital, Free University of Brussels, Belgium; ‡Sanatia Psychiatric Clinic, Brussels, Belgium; §Faculty of Computer Science, Basque Country University, San Sebastián, Spain; ¶Faculty of Medicine, Oviedo University, Spain

(Received 30 June 1995; revised 5 June 1996; accepted 2 December 1996)

Summary—We searched for regional cerebral metabolic disturbances in patients with borderline personality disorder (BPD). Ten inpatients with BPD, no current DSM-III-R Axis I diagnosis and free of any psychotropic substances, were compared with 15 age-matched control subjects using positron emission tomography with 2-deoxy-2-[18F]fluoro-D-glucose and semiquantitative analysis of regional glucose metabolic activity. We found relative hypometabolism in patients with borderline personality disorder at the level of the premotor and prefrontal cortical areas, the anterior part of the cingulate cortex and the thalamic, caudate and lenticular nuclei. This study shows significant cerebral metabolic disturbances in patients with borderline personality disorder. These metabolic disturbances, which are similar to some of those described in other psychiatric entities, may help to understand the characteristic clinical aspects of this disorder. © 1997 Elsevier Science Ltd.

Introduction

Borderline personality disorder (BPD) as defined in 1980 by the DSM-III criteria (APA, 1980) has compiled a series of patients formerly diagnosed as having emotionally unstable character, hysteroid dysphoria, pseudoneurotic schizophrenia, etc. (Gunderson et al., 1981). Patients with BPD present affective or psychotic symptoms, impulsiveness, episodic behavioral dyscontrol, pathological responses to psychological stress and chronic social malfunctioning. BPD can be identified with good reliability (Gunderson et al., 1981; Spitzer et al., 1979).

From a phenomenological point of view, this syndrome has been associated with several psychiatric and organic pathological conditions including affective disorders (Akiskal et al., 1985; Carroll et al., 1981; Pope et al., 1983; Schultz et al., 1989), schizophrenia (Gunderson et al., 1975, 1979; Schultz et al., 1989), schizoaffective psychoses (Andrulonis et al., 1982), atypical psychoses (Andrulonis et al., 1982; Mitsuda & Fukuda, 1974) and epilepsy (And-
Table 1
Clinical Characteristics of the BPD Patients

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>DSMIIIR score</th>
<th>Social adaptation DIB score</th>
<th>Disturbed behavior DIB score</th>
<th>Disturbed affect DIB score</th>
<th>Psychotic features DIB score</th>
<th>Interpersonal relationship disturbed DIB score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>6</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>7</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>7</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>7</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>7</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>7</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>6</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td>7</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

DIB = Diagnostic interview for borderlines; HRDS = Hamilton Depression Rating Scale; w = women; m = men.


The pathophysiological processes involved in the BPD syndrome are not well understood. Noradrenergic and endogenous opiate dysfunction (Van der Kolk, 1987), serotonin (Brown et al., 1982; Coccaro et al., 1989; Hollander et al., 1994) and dopamine (Bonnet & Redford, 1982; Lucas et al., 1987) system dysfunction have been proposed as possibly involved in BPD.

Although the biological bases of this clinical entity have largely been discussed in the literature (for a review see Korzekwa et al., 1993), only a few introductory results on the use of positron emission tomography (PET) with 2-deoxy-2-[18F] fluoro-D-glucose (FDG), have been reported in BPD (De La Fuente et al., 1992, 1993, 1994; Goyer et al., 1994). This is in contrast to the great bulk of PET data described for other psychiatric disorders. As some of these pathological conditions already studied with PET share common symptoms and are thought to be associated to BPD, we believe it interesting to study regional cerebral metabolism in BPD using PET with FDG because it may offer clues to a better understanding of this disorder.

Methods

Patient data

Ten patients (eight women and two men, mean age=34.2; range 24 to 45) fulfilling the DSM-IIIIR criteria for BPD and with a score of at least 7 in the Gunderson and Kolb’s diagnostic interview for borderlines (Gunderson et al., 1981) were recruited. Exclusion criteria were: current DSM-IIIIR axis I disturbances, abnormal physical or neurological examinations, abnormal standard biological blood tests, abnormal electrocardiogram, history of brain lesion, convulsion or generalized seizure or standard EEG traits of epilepsy. Depressed patients were excluded only if they met the DSM-IIIIR criteria for major
depression. A 24-item Hamilton depression rating scale was obtained on the day of the PET. Lifetime diagnosis of major depression, recurrent brief depression (Angst et al., 1990), alcohol and drug abuse, and suicide attempts were recorded (Table 1).

The patients underwent a washout period for any psychotropic drugs for at least 10 days before the PET (15 days for tricyclic antidepressants and monoamino-oxidase inhibitor agents). Nine BPD patients were withdrawn from: benzodiazepines \( n = 3 \); amitriptyline \( n = 2 \); phenelzine \( n = 1 \); alcohol \( n = 2 \); alcohol plus benzodiazepines \( n = 1 \). No patient had taken neuroleptics during the 2-month period before the PET. The washout period was strictly verified by regular plasma screening. Informed consent was obtained from all subjects and use of PET with FDG for the study of mental disorders has been approved by the Ethical Committee of our hospital.

The 15 age-matched control subjects (seven women and eight men, mean age = 30.7; range 20 to 38) were free of somatic and psychiatric pathology as assessed by the Schedule for Affective Disorders and Schizophrenia (SADS) (Spitzer & Endicott, 1978). All subjects were right-handed as determined by the Edinburgh handedness inventory (Raczkowski et al., 1974).

**PET methodology**

Local cerebral glucose uptake was studied on the patients and the normal subjects in a supine resting state with eyes closed and ears unplugged in a room with dimmed light, no conversation, and low ambient noise mostly from the scanner gantry fans. Subject fasted for at least 2 hours before scanning. The patient's head was positioned within the field of view of the camera so that the cantho-meatal line was parallel and 55 mm below the middle slice of the scan. Subject immobility was checked by the alignment of three crossed laser beams with lines drawn on the subject's face.

The PET tomograph was a CTI-Siemens 933/08-12, Knoxville, TN. The 15 6.75-mm-
thick adjacent slices (eight direct and seven crossed slices) provided, covered the entire brain. In-plane spatial resolution (full width at half maximum) was about 5 mm.

Approximately 5 mCi of FDG were injected IV and a 20-minute scan was acquired 40 minutes post-injection. PET images were corrected for attenuation using transmission scan data.

One trained technician unaware of the diagnosis, manually delineated on the PET slices regions of interest (ROI) representative of the major brain structures following a template based on the human brain stereotaxic atlas of Talairach & Tournoux (1988) (Table 2).

Table 2
ROI Templates and ROI/ROIeb Values in Controls and BPD Patients (Mean ± SD)

<table>
<thead>
<tr>
<th>Region, Brodmann area(s) and side</th>
<th>Distance</th>
<th>Controls</th>
<th>BPD</th>
<th>t-test(t =)b</th>
<th>p =</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal 6 L</td>
<td>+50</td>
<td>1.35 ± 0.16</td>
<td>1.24 ± 0.09</td>
<td>2.05</td>
<td>ns</td>
</tr>
<tr>
<td>Frontal 6 R</td>
<td>+50</td>
<td>1.39 ± 0.16</td>
<td>1.23 ± 0.08</td>
<td>2.76</td>
<td>0.01</td>
</tr>
<tr>
<td>Parietal 7 L</td>
<td>+50</td>
<td>1.14 ± 0.14</td>
<td>1.12 ± 0.07</td>
<td>0.46</td>
<td>ns</td>
</tr>
<tr>
<td>Parietal 7 R</td>
<td>+50</td>
<td>1.19 ± 0.15</td>
<td>1.12 ± 0.07</td>
<td>1.30</td>
<td>ns</td>
</tr>
<tr>
<td>Parietal 40 L</td>
<td>+50</td>
<td>1.21 ± 0.12</td>
<td>1.16 ± 0.08</td>
<td>1.04</td>
<td>ns</td>
</tr>
<tr>
<td>Parietal 40 R</td>
<td>+50</td>
<td>1.24 ± 0.11</td>
<td>1.21 ± 0.10</td>
<td>0.78</td>
<td>ns</td>
</tr>
<tr>
<td>Superior cingulate/limbic 24 L</td>
<td>+35</td>
<td>1.31 ± 0.15</td>
<td>1.20 ± 0.12</td>
<td>1.99</td>
<td>ns</td>
</tr>
<tr>
<td>Superior cingulate/limbic 24R</td>
<td>+35</td>
<td>1.33 ± 0.19</td>
<td>1.20 ± 0.13</td>
<td>1.78</td>
<td>ns</td>
</tr>
<tr>
<td>Frontal 9 L</td>
<td>+35</td>
<td>1.37 ± 0.12</td>
<td>1.26 ± 0.09</td>
<td>2.32</td>
<td>0.02</td>
</tr>
<tr>
<td>Frontal 9 R</td>
<td>+35</td>
<td>1.42 ± 0.13</td>
<td>1.28 ± 0.09</td>
<td>3.07</td>
<td>0.005</td>
</tr>
<tr>
<td>Posterior cingulate/limbic 29-31 L</td>
<td>+20</td>
<td>1.44 ± 0.17</td>
<td>1.38 ± 0.08</td>
<td>1.07</td>
<td>ns</td>
</tr>
<tr>
<td>Posterior cingulate/limbic 29-31 R</td>
<td>+20</td>
<td>1.46 ± 0.16</td>
<td>1.39 ± 0.09</td>
<td>1.32</td>
<td>ns</td>
</tr>
<tr>
<td>Frontal 10-46 L</td>
<td>+8</td>
<td>1.37 ± 0.09</td>
<td>1.25 ± 0.08</td>
<td>3.37</td>
<td>0.002</td>
</tr>
<tr>
<td>Frontal 10-46 R</td>
<td>+8</td>
<td>1.42 ± 0.09</td>
<td>1.28 ± 0.12</td>
<td>3.13</td>
<td>0.004</td>
</tr>
<tr>
<td>Frontal 10-32 L</td>
<td>+8</td>
<td>1.35 ± 0.10</td>
<td>1.24 ± 0.10</td>
<td>2.67</td>
<td>0.01</td>
</tr>
<tr>
<td>Frontal 10-32 R</td>
<td>+8</td>
<td>1.38 ± 0.12</td>
<td>1.24 ± 0.10</td>
<td>3.07</td>
<td>0.005</td>
</tr>
<tr>
<td>Caudate L</td>
<td>+8</td>
<td>1.38 ± 0.05</td>
<td>1.24 ± 0.10</td>
<td>4.55</td>
<td>0.0001</td>
</tr>
<tr>
<td>Caudate R</td>
<td>+8</td>
<td>1.45 ± 0.06</td>
<td>1.27 ± 0.14</td>
<td>4.46</td>
<td>0.0002</td>
</tr>
<tr>
<td>Thalamus L</td>
<td>+8</td>
<td>1.38 ± 0.12</td>
<td>1.26 ± 0.14</td>
<td>2.21</td>
<td>0.03</td>
</tr>
<tr>
<td>Thalamus R</td>
<td>+8</td>
<td>1.44 ± 0.14</td>
<td>1.30 ± 0.14</td>
<td>2.57</td>
<td>0.01</td>
</tr>
<tr>
<td>Temporal 42-22 L</td>
<td>+8</td>
<td>1.20 ± 0.12</td>
<td>1.23 ± 0.10</td>
<td>-0.70</td>
<td>ns</td>
</tr>
<tr>
<td>Temporal 42-22 R</td>
<td>+8</td>
<td>1.24 ± 0.10</td>
<td>1.23 ± 0.11</td>
<td>0.34</td>
<td>ns</td>
</tr>
<tr>
<td>Lenticular nucleus L</td>
<td>-1</td>
<td>1.48 ± 0.09</td>
<td>1.35 ± 0.16</td>
<td>2.59</td>
<td>0.01</td>
</tr>
<tr>
<td>Lenticular nucleus R</td>
<td>-1</td>
<td>1.53 ± 0.13</td>
<td>1.38 ± 0.15</td>
<td>2.71</td>
<td>0.01</td>
</tr>
<tr>
<td>Temporal 21 L</td>
<td>-1</td>
<td>1.21 ± 0.09</td>
<td>1.20 ± 0.07</td>
<td>0.28</td>
<td>ns</td>
</tr>
<tr>
<td>Temporal 21 R</td>
<td>-1</td>
<td>1.25 ± 0.11</td>
<td>1.17 ± 0.09</td>
<td>1.93</td>
<td>ns</td>
</tr>
<tr>
<td>Ventral striatum L</td>
<td>-8</td>
<td>1.34 ± 0.11</td>
<td>1.25 ± 0.15</td>
<td>1.66</td>
<td>ns</td>
</tr>
<tr>
<td>Ventral striatum R</td>
<td>-8</td>
<td>1.38 ± 0.12</td>
<td>1.25 ± 0.14</td>
<td>2.34</td>
<td>0.02</td>
</tr>
<tr>
<td>Anterior cingulate/limbic 25 L</td>
<td>-16</td>
<td>1.21 ± 0.10</td>
<td>1.10 ± 0.11</td>
<td>2.68</td>
<td>0.01</td>
</tr>
<tr>
<td>Anterior cingulate/limbic 25 R</td>
<td>-16</td>
<td>1.25 ± 0.13</td>
<td>1.11 ± 0.09</td>
<td>2.77</td>
<td>0.01</td>
</tr>
<tr>
<td>Frontal 11 L</td>
<td>-16</td>
<td>1.32 ± 0.10</td>
<td>1.30 ± 0.14</td>
<td>0.45</td>
<td>ns</td>
</tr>
<tr>
<td>Frontal 11 R</td>
<td>-16</td>
<td>1.36 ± 0.12</td>
<td>1.29 ± 0.13</td>
<td>1.21</td>
<td>ns</td>
</tr>
<tr>
<td>Temporal inf L</td>
<td>-24</td>
<td>0.89 ± 0.08</td>
<td>0.89 ± 0.05</td>
<td>-0.19</td>
<td>ns</td>
</tr>
<tr>
<td>Temporal inf R</td>
<td>-24</td>
<td>0.91 ± 0.05</td>
<td>0.90 ± 0.05</td>
<td>0.28</td>
<td>ns</td>
</tr>
</tbody>
</table>

R = right; L = left; ns = not significant (p > .05).
*ANOVA for repeated measures, group × region interaction: p = 0.0001.
bTwo-tailed unpaired student t-test, df= 23 except for regions with missing values; ventral striatum L and R (df= 21), cingulate-24 L and R (df= 22).

cEach ROI is delimited with a horizontal plane corresponding to a section of the atlas of Talairach & Tournoux (1988). The distance in millimeters is measured horizontally from the anterior commissure-posterior commissure line (+ : up, - : down).
In each ROI, the average count rate among all pixels was obtained. We did not perform local metabolic rates of glucose determination, which would have required arterial blood sampling. PET without arterial sampling for glucose metabolism quantification, seemed less traumatic for this kind of psychiatric patients and appeared acceptable to them. To obtain semiquantitative evaluation of regional glucose metabolism, we calculated the ratio of the average count rate in each ROI divided by the average count rate in the ipsilateral hemicerebellum (ROI/ROIcb).

Since the autoradiographic method of local metabolic rates of glucose determination uses images obtained at the same time as in our study, the ratios (ROI/ROIcb) calculated using local count rates are identical to the ratios that would have been calculated using local metabolic rates of glucose.

The cerebellum was chosen as region of reference since its cortex is a gray matter structure located outside the multiple hemispheric regions studied. We checked for possible influence of this choice by comparing the ratio ROIhem/ROIcb in the two groups with ROIhem corresponding to a ROI, which includes the whole hemisphere at the level of the thalamus and lenticular nucleus (Figure 1).

Statistical differences in regional glucose metabolism between the groups were evaluated with analysis of variance (ANOVA) for repeated measures and with two-tailed unpaired Student's t-test.

*Figure 1.* Regions of interest delimited in a horizontal plane at the level of the thalamus and lenticular nucleus situated 8 mm above the anterior commissure-posterior commissure line.
Results

The ROIhem/ROIcb indexes were not different between the two groups (mean ± SD): BPD, 0.99 ± 0.06, controls, 1.01 ± 0.06 on the left side (t = 0.76, df = 23, p = 0.45) and BPD, 0.98 ± 0.07, controls, 1.02 ± 0.06 on the right side (t = 1.40, df = 23, p = 0.17).

The semiquantitative ROI/ROIcb showed differences between BPD and controls in some cortical and subcortical areas: ANOVA for repeated measures showed a significant interaction group × region (F = 2.27, df = 33, p = 0.0001). Post-hoc t-tests showed lower ROI/ROIcb in the BPD patients at the level of the frontal premotor cortex on the right side (area 6), the prefrontal association cortex (area 9) bilaterally, the mesial and anterior prefrontal cortex bilaterally (areas 10–32 and 10–46), the anterior part of the cingulate cortex bilaterally, the ventral portion of the striatum on the right side, the caudate nucleus bilaterally, the lenticular nucleus bilaterally and the thalamus bilaterally (Table 2).

Discussion

Our study offered evidence of metabolic cortical and subcortical disturbances in a sample of unmedicated BPD patients without current DSM-IIIR axis I diagnoses compared with controls. Relative hypometabolism was found in the dorsolateral part of the frontal cortex, in the anterior cingulate cortex, the basal ganglia and the thalamus.

No significant differences were found between BPD patients and controls in the frontal premotor cortex (area 6) on the left side, the inferior part of the frontal cortex, the parietal cortex, the temporal lobes, and the ventral portion of the striatum on the left side.

We noted that all normalized values calculated with the cerebellum as region of reference were greater on the right side in the normal subjects. Such a trend has already been observed in another group of normal controls (Hagman et al., 1990). This was not found in the BPD patients.

Although in our study, patients and control subjects were not matched for gender, previous studies failed to demonstrate an effect of gender on regional distribution of glucose metabolism in the resting state (Baxter et al., 1987; Tyler et al., 1988). Differences related to gender have been described when a performance task is applied (Andreason et al., 1993). We chose the resting state for the uptake condition that has been shown to be stable in psychiatric patients and controls in FDG-PET experiments for both absolute and relative metabolic measurements (Bartlett et al., 1988, 1991).

Frontal lobes

The prefrontal cortex constitutes one of the three association cortices. It is thought to be implicated in some aspects of the cognitive behavior and in complex motor actions. Our study has shown bilateral relative hypometabolism in BPD at the level of the prefrontal and premotor cortical regions.

The frontal lobe has been extensively studied with PET in relation to psychiatric disorders. Alcoholism (Volkow et al., 1994), unipolar and bipolar depression (Baxter et al., 1985; Biver et al., 1994), obsessive–compulsive disorder (Martinot et al., 1990), anxiety disorders (Reivich et al., 1983) and even borderline personality disorder (Goyer et al., 1994) have been associated in some studies with frontal hypometabolism. Multiple authors have found
frontal hypometabolism in schizophrenic patients (for a review see Bachneff, 1991). Kaplan et al. (1993) have found a negative correlation between FDG metabolism and reality distortion in schizophrenia in right Brodman's area 6, a region that was relatively hypometabolic in our group of BPD patients. Therefore, this metabolic disturbance in BPD might be a correlate of the characteristic reality distortion often presented by BPD patients in the form of chronic feelings of depersonalization and unreality.

Impulsiveness is a leading characteristic of BPD patients. This trait might also be related to the hypofrontality found in this study. Lesions in the prefrontal cortex of primates are known to produce behavioral disinhibition (Fuster, 1980) and frontal brain injury in humans can cause personality changes (Silver et al., 1987; Stuss & Benson, 1986a, 1986b). Some of these changes are remarkably similar to certain BPD symptoms. Moreover, the frontal cortex is the main source of input to midbrain serotonergic neurons of the dorsal raphe nucleus (Mayberg et al., 1990) and serotonin system dysfunction is thought to be involved in the production of impulsiveness in BPD (Brown et al., 1982; Coccaro et al., 1989; Hollander et al., 1994). A diminished frontal metabolism could parallel impaired serotonergic function and be therefore related to the impulsiveness.

Although our BPD patients had no current axis I DSM-III-R co-diagnosis, they show depressive feelings, anxiety and brief psychotic symptoms as part of the BPD syndrome. Moreover some of our patients had a lifetime prevalence of major depressive disorder and recurrent brief depression. Therefore the question arises about the possible implication of these phenomena in the development of hypofrontality in our patients. It has been suggested (Bromfield et al., 1992) that the frontal hypometabolism found in different depressive states may be related to depressive thought and cognition rather than to the full depressive syndrome. It has also been proposed that hypofrontality might be a non-specific finding, not linked to a particular mental illness (Bachneff, 1991) but rather reflecting the cognitive deficits found in some mental states characteristic of several psychiatric illnesses (Buchsbaum et al., 1986; Dolan et al., 1993).

**Basal ganglia**

The caudate nucleus and the putamen are the largest component of the basal ganglia, they receive input mainly from the whole cerebral cortex and the intralaminar nuclei of the thalamus and they project to the frontal cortex via the pallidum and thalamus. The putamen is concerned with motor control and the caudate is associated with cognitive functions and behavioral control through the lateral orbitofrontal circuit. The ventral portion of the striatum is related to limbic functions.

Hypometabolism of the basal ganglia has been found in patients with schizophrenia (Buchsbaum et al., 1982; Sedvall et al., 1984; Sheppard et al., 1983), unipolar and bipolar depression (Baxter et al., 1985; Buchsbaum et al., 1986), generalized anxiety disorder (Wu et al., 1991a), obsessive compulsive disorder (Martinet et al., 1990), alcoholism (Volkow et al., 1994), and in total sleep deprivation conditions in normal subjects (Wu et al., 1991b).

We found bilateral relative hypometabolism in the caudate and lenticular nuclei and on the right side of the ventral striatum in BPD. This, along with the hypometabolism found in frontal and thalamic regions, suggested that the fronto-striatal-pallidal-thalamic-frontal circuitry was altered in our patients since all their main components were hypometabolic.
The striatum is the main target of the dopaminergic ascending system. A dopamine-related production of symptoms in BPD has been suggested (Bonnet & Redford, 1982; Lucas et al., 1987). The striatal glucose hypometabolism in BPD could reflect a change in dopaminergic activity as suggested by studies on acute use of cocaine (London et al., 1990) and amphetamine (Wolkin et al., 1987) in humans.

It must be mentioned that the striatal hypometabolism in our patients could also reflect the influence of associated psychopathology or past substance abuse. Three patients in our group had a lifetime diagnosis of alcohol abuse and hypometabolism of these structures, which persisted after detoxification, and has been described in alcoholic patients (Volkow et al., 1994).

Thalamus

The thalamus is a major constituent of the circuitry connecting the basal ganglia and the cerebral cortex. Virtually all pathways transmitting sensory information to the cerebral cortex make connections in the thalamus, which then projects to specific sensory areas of the cerebral cortex.

Hypometabolism in the thalamus has been described in schizophrenia (Tamminga et al., 1992; Wiesel et al., 1987), bipolar depression (Baxter et al., 1985), obsessive compulsive disorder (Martinot et al., 1990) and total sleep deprivation conditions in normal subjects (Wu et al., 1991b).

The bilateral thalamic hypometabolism found in our BPD patients might be related to disturbances in the cortical-striatal-pallidal-thalamic circuitry already suggested by the prefrontal and striatal hypometabolism.

Limbic regions

The anterior part of the cingulate gyrus, has been found to be hypometabolic in our BPD patients. This area is part of the limbic association cortex. It receives projections from the higher-order sensory areas and projects to other cortical regions including the prefrontal cortex. It also projects to the caudate nucleus and the ventral portion of the striatum (Roland, 1992). This provides a pathway by which emotions can affect motor planning. Cingulate hypometabolism, in parallel to prefrontal and striatal hypometabolism, could be related to impulsiveness, a main characteristic of BPD, which clearly represents a disturbed control of motor acts influenced by emotions.

Tamminga et al. (1992) found low metabolic rate of glucose in the anterior cingulate cortex in schizophrenia raising the hypothesis of a “psychosis circuit”. This could be of interest in BPD patients since their anterior cingulate appeared to be hypometabolic and they show brief psychotic features. The anterior cingulate is also associated with pain representation (Roland, 1992). This is important to notice since the propensity of BPD patients for automutilation may be related to a distinctive relationship to pain.

The anterior cingulate is rich in opiate receptors (Roland, 1992) and the endogenous opiate system has been postulated to be altered in BPD (Van der Kolk, 1987). This alteration could be reflected as an hypometabolic state.
In conclusion, this PET study demonstrated that cerebral metabolic disturbances are present in a sample of unmedicated BPD patients. It also confirmed that low glucose metabolism in the frontal cortex, the thalamus and the basal ganglia is a finding common to different psychiatric states. Other metabolic and biological studies are needed to confirm our results and to advance in the knowledge of BPD.

References


