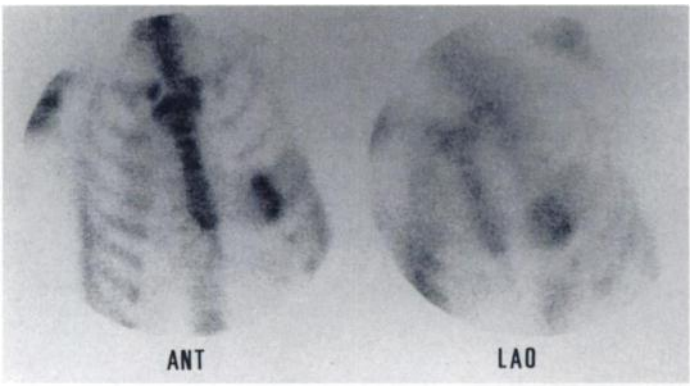

FIGURE 2. Technetium-99m-pyrophosphate cardiac scintigraphy shows a markedly positive image located on the anteroapical wall of the left ventricle. ANT= anterior view; LAO = left anterior oblique view.

cases of pericardial effusion and, less commonly, cardiac rupture, have also been reported (*1*).

Acute myocardial infarction due to electrocution is certainly a rare complication. Sometimes, the diagnosis cannot be definitively made because the electrocardiographic signs are nonspecific and CK-MB elevation can be related to skeletal muscle necrosis, and echocardiographic disorders do not persist. In some necropsy series (*4*), myocardial necrosis has been demonstrated, revealing a clear correlation between the CK-MB elevation and the myocardial damage. These necropsy studies confirmed that electrocution was the cause of the myocardial infarction in cases of no previous coronary artery disease. Electrical shock may produce necrotic bands in the nonstriated muscles of the coronary arteries and also arterial thrombosis.

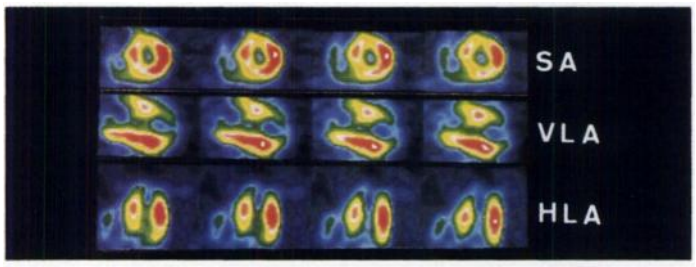

FIGURE 3. Thallium-201 SPECT scan at rest shows perfusion defects on the anteroapical and septal-wall. HLA = horizontal long-axis; SA = short-axis; VLA = vertical long-axis.

The right coronary artery, because it is closest to the thoracic wall, is the most vulnerable to electrocution.

In our patient, electrocardiographic evolution and myocardial enzyme elevation were characteristic of an anterior myocardial infarction. Technetium-99m-pyrophosphate scintigraphy confirmed acute myocardial damage with a clear segmental anteroapical location. Some cases with segmental defects on ^{201}Tl scintigraphy have been described. Additionally, a case of a positive image on ^{99m}Tc-pyrophosphate scintigraphy after radiofrequency application has been reported (*5*). Electrical defibrillation has been reported to induce positive cardiac images with ^{99m}Tc-pyrophosphate scintigraphy (*6*), but these are diffuse and rare (*7*). In our patient, echocardiography showed only mild hypokinesia in the anterior wall in contrast with ECG, cardiac enzymes and scintigraphic images. Although coronary arteriography was not performed in our patient, the findings may represent an occlusion-reperfusion phenomenon in the left descending anterior coronary artery.

CONCLUSION

Although arrhythmias following electric shock are not unusual, this case reports a previously healthy young man who developed classic findings of myocardial infarction following such an episode. Although coronary angiography data are lacking, the myocardial perfusion defect corresponds to a distal LAD occlusion.

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ECT Treatment and Cerebral Perfusion in Catatonia

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A 40-yr-old woman with a diagnosis of schizoaffective disorder developed catatonia in the context of a depressive episode. A dramatic decrease in perfusion of the inferior frontal, posterior temporal and parietal lobes bilaterally and in posterior frontal lobes corresponding to the motor cortices was noted on the ^{99m}Tc-HMPAO SPECT scan obtained in the acute phase. The most dramatic decreases compared to normal control subjects were observed in the left parietal and left motor cortices. The patient was treated with a five-treatment course of electroconvulsive therapy (ECT), which resulted in a complete resolution of catatonia and some resolution of her symptoms of depression. The repeat HMPAO-SPECT scan showed improved perfusion in all areas. The most dramatic increases occurred in the left parietal and left motor cortices. Decreased perfusion in motor and parietal cortices could be state-specific to catatonia. Thus, SPECT imaging may be a useful method for monitoring catatonia treatment response.

Key Words: catatonia; SPECT; electroconvulsive therapy
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The term "catatonia" describes a syndrome that involves dramatic changes in the level and quality of psychomotor

activity (*1*). As defined in DSM-IV, catatonia involves at least two of the following: (a) motoric immobility as evidenced by catalepsy (including waxy flexibility) or stupor, (b) excessive purposeless motor activity, (c) extreme negativism or mutism, (d) peculiarities of voluntary movement as evidenced by posturing, stereotyped movements, prominent mannerisms or prominent grimacing and (e) echolalia or echopraxia. Historically, catatonia was associated with major psychosis, specifically schizophrenia, and in the DSM-IIIR, catatonia was only described as a subtype of schizophrenia (*2*). However, the eventual recognition that it could be a state phenomenon representing motor dysfunction that generally resolves with treatment led to calls for more broad definition of catatonia (*3*). In the DSM-IV (*4*), catatonia is described in association with mood disorders, schizophrenia or medical illness under the subheading "with catatonic features."

The mechanism and neurochemistry of catatonia remain unclear, although it is recognized as associated with dysfunction of the brain's motor regulation centers, most notably in the frontal lobe (*1*). The involvement of dopaminergic systems has been postulated where there are defects in the relationship of the mesolimbic and mesostriatal pathways to the frontal lobe and brainstem (*1*). Functional brain imaging could be helpful in clarifying the mechanism of catatonia. To test the hypothesis that catatonia is a state phenomenon, it would be essential to demonstrate changes in regional cerebral blood flow (rCBF) or regional glucose metabolism (CMRglu) with resolution of catatonic syndrome. Below is a case report of a woman with schizoaffective disorder with catatonic features, whose SPECT scan showed significant changes in CBF as her catatonia resolved with electroconvulsive therapy (ECT) treatment (*5*).

CASE REPORT

Patient

A 40-yr-old black woman with a 25-yr history of schizoaffective disorder and hypothyroidism was brought to the emergency room by her mother after she became stiff, nearly mute and had stopped eating and drinking. She had a history of both manic and depressive episodes and had been hospitalized on many occasions, including three times in the last year. Once, 2 yr before this admission, she was admitted for "catatonic depression" and was successfully treated with ECT. For several months before and at the time of admission, she was taking thiothexene 40 mg qd, valproate 250 mg bid, clonazepam 1 mg qhs, benztropine 1 mg bid and levothyroxine 100 mcg qd. Before her admission, she was able to work as a security staff member of her residence. She had no history of substance abuse and no history of psychiatric illness in her family.

On admission, she displayed severe psychomotor retardation, negativism and waxy flexibility. She was at times stuporous and incontinent of urine and feces. She did not exhibit stereotypy, automatic obedience, echolalia or echopraxia. She was nearly mute, with greatly increased latency of speech and very low volume and decreased speech rate. Her mood was somewhat depressed, and her affect was flat. She was alert and oriented to person, place, time and current events. She was not delusional and was not hallucinating.

Her Hamilton Rating Scale for Depression (HRSD, 6) score was 14, her total Scale for the Assessment of Negative Symptoms (SANS, 7) score was 24 (affective flattening −5, alogia −4, avolition and apathy −4, anhedonia −5, attention −5), and she refused to comply with Mini-Mental State Examination (MMSE, 8). Her CBC, SMA-20, thyroid function tests, B12 and folate levels were within normal limits. Beta-hCG, RPR and hepatitis B tests and urine toxicology screen were negative. Her valproate level was 88.3.

She initially refused ECT. Instead, she was treated with thiothexene 30 mg qd, valproate 250 mg bid. Her clonazepam was changed to alprazolam 0.5 mg TID, with minimal response. She subsequently consented to ECT treatment, and after five treatments, her symptoms of catatonia and, to a lesser degree, her symptoms of depression improved. Her rigidity, waxy flexibility and negativism resolved. Her speech regained normal rate, tone and volume. She described her mood as "good," and her affect was bright. Her HRSD score was 13, SANS score was 16 (affective flattening −3, alogia −4, avolition −3, anhedonia −4, attention −2) and her MMSE score was 29. The patient was later discharged in improved condition.

Imaging Protocol

The patient was scanned two times using a single-head SPECT camera with a low-energy, high-resolution collimator (FWHM 7.5 mm at 10 cm depth). Her scans were compared with those of a group of healthy control subjects. The subjects were hospital employees, 5 men and 3 women, mean age 36.1, who did not have a history of psychiatric or neurological disorders and were not on psychotropic medications. The subjects were injected with 740 MBq ^{99m}Tc-HMPAO while lying supine in a dimly lit room with eyes open. Image acquisition began 20–60 min postinjection and lasted 45 min during a 360° rotation of the gamma camera in a 128 × 128 matrix.The data were prefiltered with a Gaussian filter, cutoff frequency 0.42, filter order 18. Reconstructions were performed in the transaxial, coronal and sagittal planes using a Butterworth filter with attenuation correction applied. The transaxial plane was reconstructed parallel to the canthomeatal line. The SPECT image was then viewed on a video-display terminal and consisted of transaxial, coronal and sagittal image sets. Cortical and subcortical regions of interest (ROIs) were symmetrically defined in each hemisphere. Cerebellar ROIs were selected in the middle portion of each cerebellar hemisphere. Cortical-to-cerebellar perfusion ratios were established semiquantitatively. Images were analyzed by two readers who were blind to the subjects' identities.

RESULTS

The first SPECT scan was performed 5 days after the patient's admission and before the first ECT treatment. The first study showed that the patient, when compared to control subjects, had significantly decreased perfusion in the frontal (left inferior frontal −21% decrease right inferior frontal 15% decrease), posterior temporal (15% bilaterally) and parietal cortices (left 25%, right 18%), as well as in the posterior frontal lobes corresponding to motor cortex (left 29%, right 20%) (Table 1). The changes were bilateral but were most pronounced in the left motor and left parietal lobes (Fig. 1). The second SPECT scan was obtained 2 days after her fifth ECT (Fig. 2). It showed that most of the previous areas of decreased perfusion in the frontal, posterior temporal, parietal and motor cortices had improved and appeared normal (Table 1). However, some areas of decreased perfusion, namely the right motor cortex (13% decrease) and the right parietal cortex (11% decrease), improved only partially.

DISCUSSION

Changes in rCBF in psychiatric patients were shown to occur in several psychiatric disorders such as schizophrenia (*9*), MDD (*10*), other varied disorders (*11*) and in healthy subjects (*12*). In some instances, changes in rCBF correlated with symptom severity. Such is the case in primary depression, in which decreased prefrontal perfusion was reported to worsen with the severity of depression (*13*). However, many of these differences are detectable only on group data and are not visible on individual scans for diagnostic purposes (*10,13*).

TABLE 1
Perfusion Ratios of Catatonic Patient before and after ECT Treatment and of the Control Group

ROIs	Controls Mean	Controls s.d.	Catatonia before	Catatonia after	% Change
Cingulate	0.92	0.06	0.82 (10)*	0.85 (7)	3
			Right hemisphere		
Inferior frontal	0.76	0.08	0.55 (21)	0.70 (6)	15
Frontal	0.84	0.07	0.66 (18)	0.81 (3)	15
Superior frontal	0.82	0.07	0.77 (5)	0.75 (7)	−2
Motor cortex	0.81	0.06	0.61 (20)	0.68 (13)	7
Anterior temporal	0.82	0.07	0.76 (6)	0.78 (4)	2
Posterior temporal	0.72	0.10	0.57 (15)	0.68 (4)	11
Parietal	0.83	0.11	0.65 (18)	0.72 (11)	7
Occipital	0.78	0.08	0.67 (11)	0.75 (3)	8
Basal ganglia	0.86	0.11	0.86 (0)	0.80 (6)	−6
Thalamus	0.86	0.09	0.82 (4)	0.87 (−1)	5
			Left hemisphere		
Inferior frontal	0.77	0.09	0.62 (15)	0.78 (−1)	16
Frontal	0.78	0.07	0.70 (8)	0.81 (−3)	11
Superior frontal	0.85	0.04	0.71 (14)	0.75 (10)	4
Motor cortex	0.79	0.07	0.50 (29)	0.72 (7)	22
Anterior temporal	0.76	0.05	0.71 (5)	0.68 (8)	−3
Posterior temporal	0.70	0.08	0.55 (15)	0.67 (3)	12
Parietal	0.83	0.08	0.58 (25)	0.80 (3)	22
Occipital	0.80	0.09	0.70 (10)	0.75 (5)	5
Basal ganglia	0.82	0.05	0.79 (3)	0.79 (3)	0
Thalamus	0.80	0.09	0.82 (−2)	0.81 (−1)	−1

*Catatonia "before" and "after" perfusion values are accompanied by the percent difference from the control subjects.

The SPECT scans of our patient are notable in that the decreased rCBF pre- and post-treatment are significant enough to be easily detected in an individual patient. Except for hypofrontality, this patient's rCBF pattern in the acute state nor changes in rCBF with ECT treatment are consistent with those reported in literature for patients with MDD undergoing ECT (*14,15*) or for patients with schizophrenia (*5,16,17*). Specifically, rCBF in the anterior cortical regions in patients with MDD was reported to decrease after ECT treatment. We surmise that most of the changes in the scans are due to catatonic symptoms and their resolution rather than depression, psychosis or the ECT effects. Hypofrontality was reported to be associated with negative symptoms in patients with schizophrenia (*16,17*), dementia of Alzheimer's type and strokes (*18,19*). Partial improvement in this patient's frontal perfusion could therefore be related to the partial improvement of her negative symptoms.

The areas of hypoperfusion seen in this patient's SPECT scan during acute catatonia and before treatment were visualized in the frontal, motor, posterior temporal and temporal cortices and parietal cortices bilaterally. The decrease in perfusion was most pronounced and the treatment response was most dramatic in the bilateral frontal, left motor and left parietal cortices (Table 1). These areas are somewhat different from those reported in the older neurological lesion/deficit studies that implicated frontal lobe, basal ganglia and brainstem involvement (1, review). In fact, in our patient, perfusion in basal ganglia and thalamus was normal and did not change with treatment. The areas of decreased perfusion reported here are more consistent with those recently published in functional imaging studies (*19–22*) of catatonic schizophrenia. While intuitively, it seems logical that motor neurons will be involved in the motor slowing of catatonia, the decreased perfusion seen in our patient's motor cortex during an acute catatonic episode and return to normal perfusion with treatment is a confirmation of such involvement.

In six clinically stable patients with catatonic schizophrenia, a [^{123}I]iodoamphetamine SPECT study and ^{18}F-2-fluoro-2-deoxy-D-glucose PET study showed both decreased rCBF and glucose utilization to the dorsal frontal and parietal lobes (*20,21*). This pattern was specific to those patients and distinguished them from patients with other schizophrenic subtypes (*20*). Although no correlation analyses were performed, Satoh et al. (*20*) reported that there seemed to be an association between the severity of catatonic symptoms and decreased frontoparietal hypoperfusion in treated and partially treated

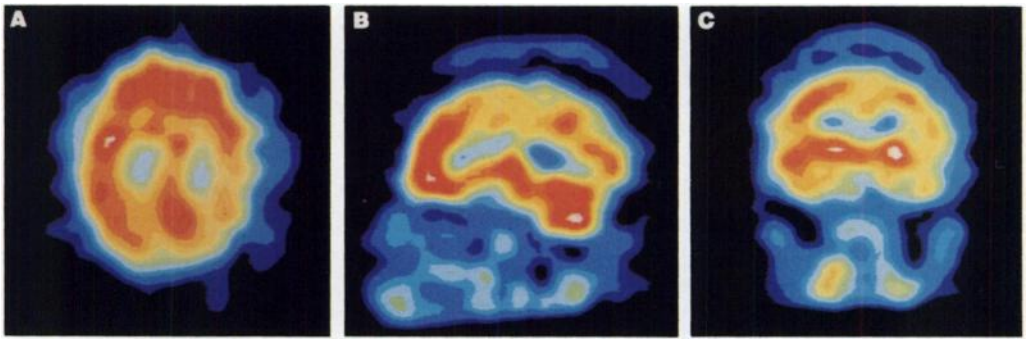

FIGURE 1. Technetium-99m-HMPAO SPECT transverse (A), saggital (B) and coronal (C) sections before (9/15/95) the ECT treatment. All sections pass through the posterior frontal cortex.

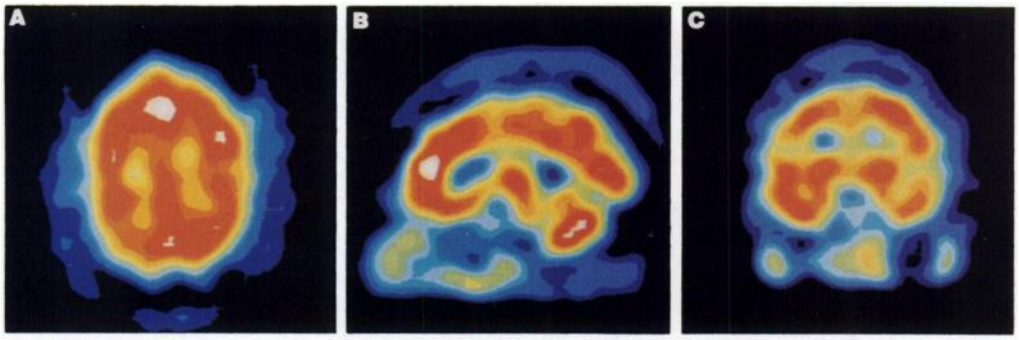

FIGURE 2. Technetium-99m-HMPAO SPECT transverse (A), saggital (B) and coronal (C) sections after (10/20/95) ECT treatment. All sections pass through the posterior frontal cortex.

patients with catatonic schizophrenia. Since rCBF changes with resolution of catatonic state were not assessed in these reports, at this juncture it is not possible to determine whether perfusion patterns reported for patients within catatonic schizophrenia by Satoh et al. were trait- or state-specific.

CONCLUSION

Resolution of hypoperfusion in the frontal and parietal cortices with resolution of acute catatonia in a patient with schizoaffective disorder indicates that these changes may be specific to the catatonic condition rather than to the diagnosis of catatonic schizophrenia. Thus, this finding supports the hypothesis that catatonia is a state phenomenon rather than a syndrome confined to catatonic schizophrenia. Further significance of this case lies in demonstrating the potential usefulness of SPECT as a clinical tool in psychiatry.

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Identification of Severe Right Ventricular Dysfunction by Technetium-99m-Sestamibi Gated SPECT Imaging

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An 84-yr-old man with previous anterior wall myocardial infarction presented with shortness of breath and palpitations. His symptoms were attributed to myocardial ischemia, and he was referred for a stress ^{99m}Tc-sestamibi SPECT imaging study with gating. The images showed minimal left ventricular ischemia, but a dilated and hypokinetic right ventricle suggested pulmonary pathology as the probable etiology of his presenting symptoms. A subsequent ventilation perfusion study was consistent with the diagnosis of multiple pulmonary emboli. Thus, ^{99m}Tc-sestamibi SPECT imaging with gating provides information about right ventricular perfusion and function, enhancing the clinical utility of stress myocardial perfusion imaging.

Key Words: gated SPECT; technetium-99m-sestamibi; right ventricle

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Stress radionuclide myocardial SPECT perfusion imaging is an important noninvasive modality for assessing patients with known or suspected coronary artery disease (*1–4*). The high

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