mechanism of the tumor uptake has been investigated but not identified clearly yet. Further basic and clinical investigation is necessary to understand the behavior of this promising radiopharmaceutical.

REFERENCES

1. Tonami N, Shuke N, Yokoyama K, et al. Thallium-201 SPECT in the evaluation of suspected lung cancer. *J Nucl Med* 1989;30:997–1004.
2. Tonami N, Hisada K. Clinical experience of tumor imaging with ^{201}Tl-chloride. *Clin Nucl Med* 1977;2:75–81.
3. Kaplan WD, Takvorian T, Morris JH, Rumbaugh CL, Connoly BT, Atkins HL. Thallium-201 brain tumor imaging: a comparative study with pathologic correlation. *J Nucl Med* 1987;28:47–52.
4. Ancri D, Bassett JY, Lonchampt MF, et al. Diagnosis of cerebral lesions by ^{201}Tl. *J Nucl Med* 1978;128:417–422.
5. Black KL, Hawkins RA, Kim KT, Becker DP, Lerner C, Marciano D. Use of ^{201}Tl SPECT to quantitate malignancy grade of gliomas. *J Neurosurg* 1989;71:342–346.
6. Mountz JM, Stafford-Schuck K, McKeever PE, Taren J, Beierwaltes WH. Thallium-201 tumor/cardiac ratio estimation of residual astrocytoma. *J Neurosurg* 1988;68:705–709.
7. Kim KT, Black KL, Marciano D, et al. Thallium-201 SPECT images of brain tumors: methods and results. *J Nucl Med* 1990;31:965–969.
8. Westera G, Gadze A, Horst W. A convenient method for the preparation of ^{99m}Tc(V) dimercaptosuccinic acid (^{99m}Tc(V)-DMSA). *Int J Appl Radiat Isot* 1985;36:311–312.
9. Blower PJ, Singh J, Clarke SEM. The chemical identity of pentavalent ^{99m}Tc-dimercaptosuccinic acid. *J Nucl Med* 1991;32:845–849.
10. Ohta H, Yamamoto K, Endo K, et al. A new imaging agent for medullary carcinoma of the thyroid. *J Nucl Med* 1984;25:323–325.
11. Ohta H, Endo K, Fujita T, et al. Imaging of soft tissue tumors with ^{99m}Tc(V)-dimercaptosuccinic acid, a new tumor seeking agent. *Clin Nucl Med* 1984;9:568–573.
12. Ohta H, Endo K, Fujita T, Konishi J, Torizuka K, Horiuchi K, Yokoyama A. Clinical evaluation of tumor imaging using ^{99m}Tc(V)-dimercaptosuccinic acid, a new tumor seeking agent. *Nucl Med Commun* 1988;9:105–116.
13. Hirano T, Otake H, Yoshida I, Endo K. Primary lung cancer SPECT imaging with pentavalent ^{99m}Tc-DMSA. *J Nucl Med* 1995;36:202–207.
14. Lamki L, Shearer R. Technetium-99m-DMSA uptake by metastatic carcinoma of the prostate. *J Nucl Med* 1985;25:733–734.
15. Hirano T, Tomiyoshi K, Ying Jian Zhang, Ishida T, Inoue T, Endo K. Preparation and clinical evaluation of ^{99m}Tc-DMSA for tumor scintigraphy. *Eur J Nucl Med* 1994;21; 82–85.
16. Watkinson JC, Allen SJ, Laws DE, Lazarus CR, Maisey MN, Clarke SEM. The pharmacokinetics and biodistribution of ^{99m}Tc(V)-dimercaptosuccinic acid in an animal tumor model. *J Nucl Med* 1992;32:1235–1238.

Fasting Improves Discrimination of Grade 1 and Atypical or Malignant Meningioma in FDG-PET

Uwe Cremerius, Roland Bares, Joachim Weis, Osama Sabri, Michael Mull, J. Michael Schröder, Joachim M. Gilsbach and Udalrich Buell

Departments of Nuclear Medicine, Neuropathology, Neuroradiology and Neurosurgery, Aachen University of Technology, Germany

We investigated the use of PET with 2[^{18}F]fluoro-2-deoxy-D-glucose (FDG) to discriminate between atypical or malignant and grade 1 meningiomas. The influence of fasting state and high-dose corticosteroid medication was analyzed retrospectively. **Methods:** Preoperative PET scans of 75 patients with suspected diagnosis of intracranial meningioma were evaluated using standardized uptake values (SUV) and tumor-to-contralateral gray matter ratios (TGR) of FDG uptake. Fifty-one of 75 patients fasted before the PET scan, and 27 of 75 patients were studied under high-dose corticosteroid medication. Eighteen tumors had recurred. PET results were compared to histopathological grading. **Results:** PET correctly identified 8/9 atypical or malignant meningiomas and 58/66 grade 1 meningiomas using TGR and a threshold of 1.05 in primary meningioma and 0.85 in tumor recurrence. This corresponds to a specificity of 0.88 for the detection of higher tumor grading. Specificity was significantly higher in fasting compared to nonfasting subjects (0.96 versus 0.73; $p < 0.025$). SUV quantification lead to a reduced specificity of 0.77 at the same level of sensitivity. The only false-negative PET finding occurred in a recurrent meningioma, which had been operated on four times before. **Conclusion:** Overnight fasting before injection is needed to improve the diagnostic accuracy of FDG-PET for noninvasive metabolic grading of meningioma. Hyperglycemia in nonfasting patients and in diabetic patients may lead to overestimation of meningioma grading.

Key Words: meningioma; grading; PET; fluorine-18-FDG

J Nucl Med 1997; 38:26–30

Meningiomas are the most common benign intracranial tumors. They represent about 15% of all primary intracranial tumors. Meningiomas are not always curable. The rate of recurrence depends on the completeness of removal, the site of the tumor and its biological aggressiveness (*1*). In a large series, recurrences were found in 7% of grade 1 meningiomas, in 35% of grade 2 and 70% of grade 3 tumors (*2*). Glucose consumption of intracranial meningioma assessed by PET using 2[^{18}F]fluoro-2-deoxy-D-glucose (FDG) has been proposed as an index of tumor aggressivity and probability of recurrence by Di Chiro et al. (*3*).

Recent studies have been published that addressed the effect of blood glucose levels on FDG uptake in cancer (*4,5*). Little, however, is known about the influence of metabolic factors on FDG uptake in intracranial tumors and brain tissue. Various investigators have stressed the importance of the fasting state for PET scans using FDG in oncology (*4,6*). It is not known whether patients in PET studies related to neuro-oncology should also fast before the scan. Additionally, the effect of medication, which may influence glucose metabolism, has not yet been systematically studied. A large number of meningioma patients preoperatively receive high-dose corticosteroids to reduce peritumoral edema. Corticosteroids are known to stimulate gluconeogenesis in hepatic tissue (*7*) and may thus have an effect on FDG uptake similar to nutritive glucose.

The aim of this study was to evaluate the use of FDG-PET as a noninvasive metabolic assessment of intracranial meningiomas compared to histopathological grading. We also analyzed the influence of fasting state, high-dose corticosteroid medication and previous surgical treatment.

METHODS

Patients

Seventy-five patients (51 women, 24 men; mean age 58 yr; range 14–81 yr) with the neuroradiological diagnosis of suspected meningioma were examined by FDG-PET within 21 days before neurosurgery. Informed consent was obtained from all patients. The diagnosis was proven histologically in all patients. According

Received Nov. 29, 1995; revision accepted May 29, 1996.

For correspondence or reprints contact: Uwe Cremerius, MD, Department of Nuclear Medicine, RWTH Aachen, Pauwelsstr. 30, D-52057 Aachen, Germany.

to the new WHO classification of brain tumors (*8*), 66 meningiomas were classified grade 1, six grade 2 (atypical) and three grade 3 (malignant) by an experienced neuropathologist. In 57 patients, meningioma resection was performed for the first time; 18 tumors had recurred after previous surgery. PET studies were performed after overnight fasting in 51 patients and 3–5 hr after breakfast in the residual 24 patients. Twenty-seven patients received high-dose corticosteroid therapy (dexamethasone 8–48 mg/day) to reduce distinct peritumoral edema seen on CT or MRI scans; 20/27 patients under corticosteroid therapy had been fasting overnight before the PET scan. There were two patients with known insulin-dependent diabetes mellitus: both were studied in the fasting condition and without corticosteroid. The study was approved by the local ethics committee.

PET Imaging

PET was performed using an ECAT 953/15 scanner (Siemens-CTI, Knoxville, TN), which allows simultaneous acquisition of 15 contiguous cross-sectional slices with a 3.375-mm slice thickness. Axial and in-plane resolution of the device was 6.5 mm FWHM under routine conditions. Transmission scanning was performed with a retractable ^{68}Ge ring source to correct for photon attenuation either before the injection (in 39 patients) or the following day using a specially designed headholder (*9*) and flexible masks to ensure a reproducible position of the head (in 36 patients). This repositioning procedure had been used before in our institution for PET versus MRI fusion. Maximum spatial error was reported to be within 3 mm (*9*). Two adjacent bed positions were needed to cover the tumors as well as contralateral gray matter at the level of basal ganglia as a reference. Approximately 10 million true counts/plane were acquired during a typical 15-min transmission scan. Emission scans were started 30 min after injection of 125–330 MBq FDG (delivered from the Institute of Radiochemistry, Research Center Jülich). The two bed positions were scanned sequentially three times, with a scanning time of 5 min per bed position and scan. Transversal images were reconstructed with 128 × 128 matrix size using a Hanning filter for filtered backprojection and a cutoff frequency of 0.5 nyquist. Scatter correction was not available. Static images of corresponding planes were summed up to reduce time-dependent changes of radioactivity concentrations between the two bed positions, but no slices were added. Coronal and sagittal slices were generated to improve visual tumor identification but were not used for quantitative evaluation.

Circular regions of interest (ROIs) were drawn within the borders of the tumor region on three adjacent slices with a diameter of at least 18 mm and the average radioactivity concentration was determined. All meningiomas could be clearly identified as hypo- or hypermetabolic lesions compared to gray and/or white matter. CT and/or MRI scans were available for comparison in each case. Rectangular ROIs manually adjusted to the outer brain contour were used to cover a representative part of contralateral temporoparietal gray matter in three adjacent slices. The average radioactivity concentration was used for further evaluation.

FDG uptake was measured using standardized uptake values (SUV) as follows: SUV = radioactivity concentration in ROI [Bq\ml]\injected dose [Bq] per body weight [g]. The radioactivity concentration was corrected for calibration and decay. Relative uptake was determined as the tumor-to-gray matter ratio (TGR).

Plasma glucose levels were routinely measured immediately before FDG injection. In 37 patients, plasma insulin levels before injection were also determined.

For statistical evaluation of differences between patient subgroups, the Mann-Whitney U-test was used. The Spearman rank test was used to prove significance of bivariate correlation.

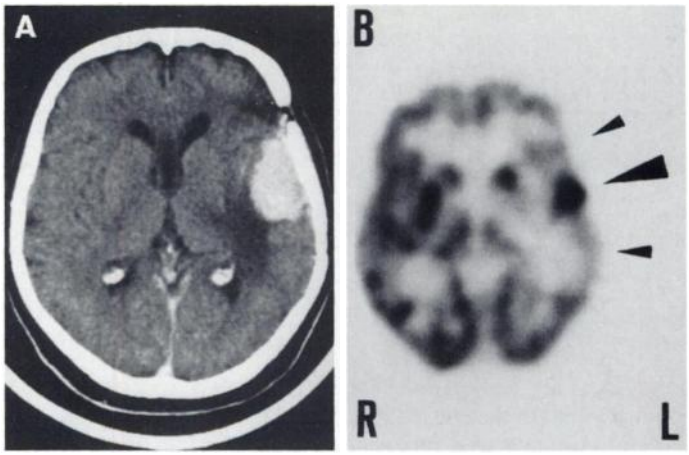

FIGURE 1. Corresponding contrast-enhanced CT (A) and PET (B) scans of Patient 3 who had tumor recurrence 8 yr after removal of a convexity meningioma. Marked contrast-enhancement and perifocal edema can be seen on the CT scan. PET scan shows intense focal tumoral FDG uptake (SUV = 9.3; TGR = 1.27; large arrowhead) and significantly reduced uptake of peritumoral gray matter (smaller arrowheads). WHO tumor classification: grade 3.

Significance of different specificities between various subgroups was evaluated by Fisher's exact test.

RESULTS

All meningiomas, with the exception of two patients, showed a homogeneous FDG uptake. In 59/75 (79%) patients, tumor uptake was below contralateral gray matter values and gray matter uptake was exceeded in 16/75 (21%) meningiomas (Figs. 1 and 2).

By using a threshold of 5.5 for SUV as a discriminating parameter, 8/9 tumors with higher grading were correctly detected, while grade 1 meningiomas were correctly classified in 51/66 patients for a specificity of 0.77. With relative FDG uptake (TGR) and a cutoff level of 0.85, one atypical meningioma was also misclassified, and a specificity of 0.74 was calculated. Due to the low number of grade 2 and 3 meningiomas in the patients examined, positive predictive values were low, but excellent negative predictive values were achieved. By using separate thresholds for TGR of 1.05 in primary meningioma and 0.85 in recurrent meningioma, a higher specificity of 0.88 was achieved at the same level of sensitivity (Table 1).

Specificities were also calculated separately in fasted and nonfasted patients as well as in patients with and without

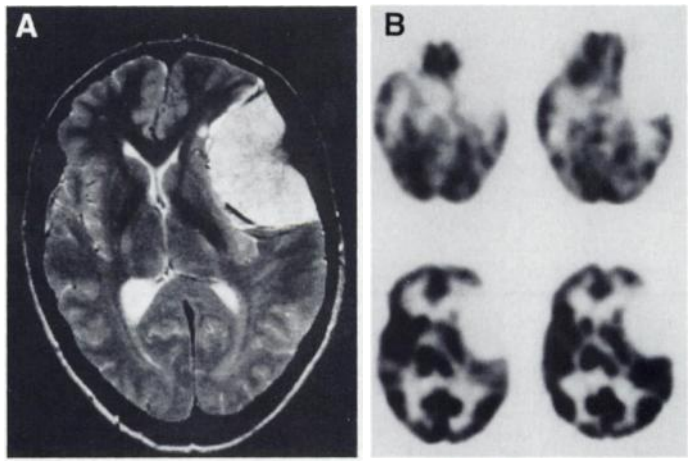

FIGURE 2. Corresponding MR (A) and PET (B) scans of a patient with a large grade 1 meningioma of the left lateral sphenoid. The tumor is seen as a defect area with homogeneous low FDG uptake compared to gray matter (SUV = 3.1; TGR = 0.36).

TABLE 1
Diagnostic Accuracy of SUV and TGR for Detection of Atypical or Malignant Meningioma

Parameter	Threshold	TP	FN	TN	FP	Sens.	Spec.	PPV	NPV
SUV	5.5	8/9	1/9	51/66	15/66	0.89	0.77	0.35	0.98
TGR	0.85	8/9	1/9	49/66	17/66	0.89	0.74	0.32	0.98
TGR	0.85/1.05*	8/9	1/9	58/66	8/66	0.89	0.88	0.5	0.98

*In primary/recurrent meningioma.
TP = true-positive; FN = false-negative; SUV = standardized uptake value. TGR = tumor-to-gray matter ratio. TN = true-negative; FP = false-positive; Sens. = sensitivity; Spec. = specificity; PPV = positive predictive value; NPV = negative predictive value.

corticosteroid medication (Table 2). Higher specificity was usually found in the subgroups of fasted patients and of patients without corticosteroid medication, but the difference was significant only in TGR, when different cutoff levels for primary and recurrent meningioma were used. Thus, histopathological grading of meningioma was overestimated by PET only in 2/44 fasted patients but in 6/22 nonfasted patients ($p < 0.025$).

Recurrent tumors were significantly smaller than meningiomas without previous surgical treatment. Plasma glucose levels were similar in both groups. Reference gray matter in patients with tumor recurrence showed significantly higher SUV. SUV of recurrent tumors did not differ from primary meningiomas. None of the patients with a recurrent grade 1 tumor received corticosteroid medication. For those patients without dexamethasone therapy, the difference between gray matter SUV of primary and recurrent meningiomas became insignificant, but gray matter SUV of recurrences remained higher. TGR in recurrences was lower and the range was smaller than in primary tumors (Table 3).

Table 4 shows the influence of fasting and high-dose corticosteroid treatment on patient plasma glucose and plasma insulin levels as well as on SUV of reference gray matter and grade 1 meningiomas. Fasting patients exhibited significantly lower plasma glucose and insulin levels compared to nonfasting patients, while in patients under corticosteroid medication, only insulin levels were significantly higher. SUV of gray matter was significantly reduced in patients under corticosteroid treatment. No effect of fasting or corticosteroid therapy was seen on tumoral SUV of grade 1 meningiomas.

Regression analysis was performed between FDG uptake (SUV) and plasma glucose level. The two parameters were significantly correlated in gray matter with a rank correlation coefficient of $r_s = -0.55$ ($p < 0.001$) but not in grade 1 meningioma (Fig. 3).

Table 5 gives clinical data of all patients in whom higher meningioma grading was correctly predicted and of the nine patients in whom histopathological grading was over- or underestimated by FDG-PET using TGR and separate thresholds for primary and recurrent tumors.

TABLE 2
Influence of Metabolic Conditions on Specificity of SUV and TGR for Detection of Atypical or Malignant Meningioma

Condition	SUV	TGR*	TGR†
Fasting	0.77	0.77	0.96‡
Nonfasting	0.77	0.68	0.73‡
No corticosteroid	0.79	0.81	0.91
Corticosteroid	0.74	0.61	0.83

* Threshold 0.85 for all patients.
† Threshold 0.85 for recurrent meningioma and 1.05 for primary meningioma.
‡Significant for $p < 0.025$ (Fisher's exact test).

DISCUSSION

The highest glucose utilization rates among 17 intracranial meningiomas were found in an atypical and a papillary meningioma by Di Chiro et al. in 1987 (*3*). Both tumors would have been classified grade 2 or 3 according to the new WHO classification. In a recent study, our group evaluated the relationship between FDG uptake and biological aggressiveness of meningioma in 60 patients. We found significantly elevated FDG uptake in grades 2 and 3 compared to grade 1 meningiomas in tumors of high cellularity and in those with an elevated Ki-67 proliferation index (*10*). Enhanced FDG uptake in some meningiomas was correlated to monosomy of the short arm chromosome one, a specific cytogenetic anomaly that had been previously found in a variety of malignant tumors (*11*). These results encouraged us to evaluate the diagnostic accuracy of FDG-PET for preoperative detection of atypical or malignant meningioma in patients with suspected meningioma.

In most FDG-PET studies of intracranial tumors, an absolute quantification of the metabolic rate for glucose according to Sokoloff et al. (*12*) and Phelps et al. (*13*) is performed. This approach, however, needs a priori estimates for kinetic constants, which are not known for individual tumors. Di Chiro and Brooks (*14*) stressed the importance of visual interpretation of PET scans and the limitations of quantification. While in some tumors such as hepatocellular carcinomas, a correlation between the in vivo measurement of rate constants by FDG-PET and the in vitro assessment of hexokinase activity was reported (*15*), similar information is still lacking in meningioma. In our study, quantitative tumor analysis was limited to standardized uptake values [(SUV), also called differential uptake ratio (DUR)] and TGR of FDG uptake. We felt that arterial blood sampling should not be included in a noninvasive method, and

TABLE 3
Clinical and PET Data of Patients with Primary and Recurrent Grade 1 Meningioma*

Parameter	Primary meningioma (n = 53)	Recurrent meningioma (n = 13)
Tumor volume (m)	32 ± 30‡	18 ± 14‡
Plasma glucose (mmol/liter)	5.1 ± 1.6	5.2 ± 0.9
SUV (tumor)	4.9 ± 1.7	5.0 ± 1.6
SUV (gray matter)	6.9 ± 1.8‡	8.2 ± 1.5‡
SUV (gray matter)†	7.4 ± 1.8	8.2 ± 1.5
TGR	0.74 ± 0.28	0.61 ± 0.14
TGR†	0.69 ± 0.25	0.61 ± 0.14

*Mean ± s.d.
†Only in patients without corticosteriod therapy (n = 30/13).
‡$p < 0.025$ (Mann-Whitney U-test).

TABLE 4
Metabolic Data* and SUV* in Different Metabolic Conditions

Condition	Plasma glucose (mmole/liter)	Plasma insulin (mU/liter)	SUV of gray matter	SUV of meningioma†
Fasting (n = 51)	5.0 ± 1.5‡	10.5 ± 7.9§	7.2 ± 1.8	4.9 ± 1.5
Non-fasting (n = 24)	5.4 ± 1.0‡	23.0 ± 14.6§	7.0 ± 2.0	5.0 ± 1.9
No corticosteroid (n = 48)	5.0 ± 1.5	8.7 ± 7.5§	7.7 ± 1.7§	4.9 ± 1.4
Corticosteroid (n = 27)	5.2 ± 1.2	17.2 ± 11.6§	6.2 ± 1.7§	5.0 ± 2.0

*Mean ± s.d.
†Only grade 1 tumors.
‡Significant for $p < 0.025$.
§Significant for $p < 0.01$ (Mann-Whitney U-test).

PET protocols should be as simple as possible to be practical as a routine procedure.

One major aim of our study was to find out whether metabolic factors significantly interfere with parameters of tumor aggressivity correlated to elevated FDG uptake. Histopathological grading was chosen as the parameter of tumor aggressivity because it is still the basis of clinical decision making.

By using different thresholds for primary and recurrent tumors, TGRs were more specific in the detection of atypical or malignant meningioma than SUVs at the same level of sensitivity. With glucose consumption of normal unaffected gray matter being a rather stable parameter, the use of TGR may be regarded as a simple way to correct for variations of input function, which is not included in the SUV. However, TGR does not correct for variations of plasma glucose level, because FDG uptake of grade 1 meningioma and gray matter were influenced in a different way by hyperglycemia. A similar observation was reported in glioblastoma studied by FDG-PET before and after glucose loading (*16*). This may explain why specificity of TGRs was superior to SUVs only in fasting patients.

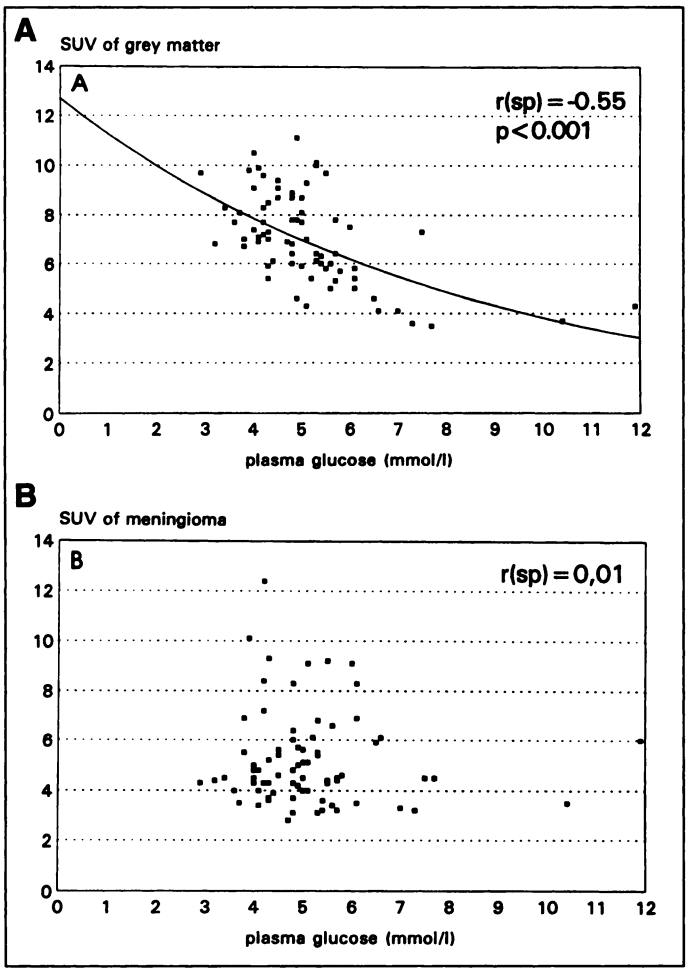

FIGURE 3. Regression analysis of FDG uptake (SUV) versus plasma glucose level for reference gray matter (A) and grade 1 meningioma (B). SUV and plasma glucose are correlated in gray matter ($p < 0.001$; Spearman's rank correlation) but not grade 1 meningioma.

Fasting improved specificity of tumor-gray matter ratios substantially. Six of eight false-positive findings occurred in patients who had not been fasting before the study; four of them had also received dexamethasone (12–48 mg/day). One of the remaining two patients (Patient 14) had known insulin-dependent diabetes mellitus, leading to hyperglycemia and hyperinsulinemia during the PET study.

As mentioned above, constant glucose consumption of reference contralateral gray matter is a prerequisite for a good reliability of TGR values. Many conditions such as cerebrovascular disease, dementia, cerebral activation during the PET scan, site of the dominant hemisphere and others may probably interfere with this demand. It is interesting to note in this context that we observed significantly higher SUVs in reference gray matter of patients with recurrent grade 1 meningiomas than in primary ones. This difference was partly explained by the lack of a corticosteroid therapy in the recurrence group, but a difference remained after eliminating this influence. A possible explanation might be that under normal conditions cerebral functions are more randomly distributed on both hemispheres, whereas after surgical treatment, the contralateral hemisphere may become more dominant. In our opinion, this justifies the use of different thresholds for TGR of 1.05 in primary and 0.85 in recurrent tumors. Interestingly, a lower cutoff level of 0.60 has been reported recently for optimal differentiation of low-grade from high-grade gliomas by FDG-PET and TGRs (*17*).

The only false-negative finding in our study occurred in a recurrent parasagittal meningioma with a tumor volume of 14 ml. Of the atypical or malignant meningiomas, this was the only patient who had undergone multiple (four) previous operations at the tumor site. Histological examination revealed tumor tissue containing surgical threads and extensive reactive inflammatory infiltration, probably leading to an underestimation of tumor cell metabolism.

CONCLUSION

FDG-PET imaging of unselected patients with suspected intracranial meningioma yielded a high negative but only low positive predictive value in the differentiation of atypical or malignant meningioma subtypes, if the patient's metabolic

TABLE 5
Clinical Data and PET Findings in Patients with Atypical or Malignant Meningioma and/or Elevated FDG Uptake*

Patient no.	Age (yr)	Sex	Subtype/ Grade (WHO)	Previous surgery†/ Month ago	Site/Volume (ml)‡	Fasting/ Diabetes/ Dexa. (mg/d)	Plasma glucose (mmole/liter)	Plasma insulin (mU/liter)	SUV	TGR	PET finding versus grading§
1	66	F	Fibrobla/2	No/–	CPA/7	Yes/No/24	6.6	na	6.1	1.47	TP
2	68	F	Transitio/2	Yes/35	Parasg/28	No/No/–	4.2	11.0	7.2	0.86	TP
3	66	F	Meningo/3	Yes/96	Convex/18	Yes/No/–	4.3	4.4	9.3	1.27	TP
4	71	M	Sarcoma/3	Yes/10	Convex/26	Yes/No/8	4.8	6.4	6.4	0.92	TP
5	70	F	Meningo/2	No/–	Convex/47	Yes/No/48	6.5	13.7	5.9	1.29	TP
6	70	M	Meningo/3	Yes/19	Convex/40	No/No/–	4.2	10.6	12.4	1.30	TP
7	19	M	Meningo/2	No/–	Parasg/130	Yes/No/48	4.9	28.8	5.7	1.22	TP
8	62	F	Papillary/2	No/–	Convex/50	Yes/No/–	4.2	3.6	8.4	1.10	TP
9	55	M	Meningo/2	Yes (4×)/7	Parasg/14	Yes/No/–	4.3	na	5.2	0.61	FN
10	71	F	Psammo/1	No/–	FB/8	No/No/–	6.0	na	9.1	1.17	FP
11	57	M	Fibrobla/1	No/–	Convex/57	No/No/12	7.7	na	4.5	1.27	FP
12	64	F	Fibrobla/1	No/–	HT/52	No/No/12	6.1	na	6.9	1.41	FP
13	69	M	Transitio/1	No/–	Convex/29	Yes/No/–	5.2	5.1	6.1	1.14	FP
14	65	M	Fibrobla/1	No/–	Convex/74	Yes/Yes/–	11.9	32.3	6.0	1.40	FP
15	68	M	Fibrobla/1	No/–	Convex/65	No/No/16	6.1	21.5	8.3	1.42	FP
16	64	F	Fibrobla/1	No/–	Convex/85	No/No/48	5.6	52.8	6.6	1.32	FP
17	55	F	Meningo/1	Yes/84	Sphenoid/7	No/No/–	5.1	na	9.1	0.94	FP

*Tumor-gray matter ratio (TGR) > 1.05 for primary or > 0.85 for recurrent meningioma.
†Meningioma resection.
‡Measured by CT or MRI scan.
§Using TGR.
Dexa = dexamethason; fibrobla = fibroblastic; transitio = transitional; meningo = meningothelial; psammo = psammomatous; convex = convexity; parasg = parasagittal; CPA = cerebello-pontine angle; FB = fronto-basal; HT = hiatus tentorii; na = not available; TP = true-positive; FN = false-negative; FP = false-positive.

condition during the PET scan was not clearly defined. Diagnostic accuracy in our study was mainly limited by false-positive PET findings (i.e., high FDG uptake also found in some histopathologically benign meningiomas). Retrospective analysis identified the influence of previous surgery and of the patient's fasting state on FDG uptake of reference gray matter as important factors leading to an impairment of the results. Acceptable diagnostic accuracy was only reached in patients who fasted before the PET scan. In this group, tumor uptake compared to contralateral gray matter of more than 1.05 in primary meningioma and more than 0.85 in recurrent meningioma predicted higher histopathological grading in 6/8 patients. The clinical role of FDG-PET for noninvasive metabolic grading of meningiomas and other intracranial tumors should be further evaluated, taking into account the patient's metabolic condition.

ACKNOWLEDGMENTS

We thank the personnel of the nuclear medicine department and Mrs. I. Lossau for their cooperation. The study was supported by grant DFG Bu 682/1-1 from the German Research Society.

REFERENCES

1. Black PM. Brain tumors (second of two parts). *N Engl J Med* 1991;324:1555–1564.
2. Maier H, Oefner D, Hittmair A, Kitz K, Budka H. Classic, atypical and anaplastic meningioma: three histopathological subtypes of clinical relevance. *J Neurosurg* 1992;77:616–623.
3. Di Chiro G, Hatazawa J, Katz DA, Rizzoli HV, De Michele DJ. Glucose utilization by intracranial meningiomas as an index of tumor aggressivity and probability of recurrence: a PET study. *Radiology* 1987;164:521–526.
4. Lindholm P, Minn H, Leskinen-Kallio S, et al. Influence of blood glucose concentration on FDG uptake in cancer—a PET study. *J Nucl Med* 1993;34:1–6.
5. Wahl RL, Henry CA, Ethier SP. Serum glucose: effects on tumor and normal tissue accumulation of 2[^{18}F]fluoro-2-deoxy-D-glucose in rodents with mammary carcinoma. *Radiology* 1992;183:643–647.
6. Bares R, Klever P, Hellwig D, et al. Pancreatic cancer detected by positron-emission-tomography with ^{18}F-labeled deoxyglucose: method and first results. *Nucl Med Commun* 1993;14:596–601.
7. Hierholzer K, Schmidt RF. Pathophysiology in man. Weinheim, Germany: VCH Publishing Co.; 1991.
8. Kleihues P, Burger PC, Scheithauer BW. The new WHO classification of brain tumors. *Brain Pathol* 1993;3:255–268.
9. Kaiser HJ, Sabri O, Wagenknecht G, et al. A method of correlating and merging cerebral morphology and function by a special headholder. *Nuklearmedizin* 1994;33:123–126.
10. Cremerius U, Striepecke E, Henn W, et al. FDG-18 PET in intracranial meningioma versus grading, proliferation index, cellular density and cytogenetical analysis. *Nuklearmedizin* 1994;33:144–149.
11. Henn W, Cremerius U, Heide G, et al. Monosomy 1p is correlated with enhanced in vivo glucose metabolism in meningiomas. *Cancer Genet Cytogenet* 1995;79:144–148.
12. Sokoloff L, Reivich M, Kennedy C, et al. The ^{14}C-deoxyglucose method for the measurement of local cerebral glucose utilization: theory, procedure and normal values in the conscious and anesthetized albino rat. *J Neurochem* 1977;28:897–916.
13. Phelps ME, Huang SC, Hoffmann EJ, et al. Tomographic measurement of local cerebral glucose metabolic rate in humans with [^{18}F]2-fluoro-2-deoxy-D-glucose: validation of method. *Ann Neurol* 1979;6:371–388.
14. Di Chiro G, Brooks RA. PET quantification: blessing and curse. *J Nucl Med* 1988;29:1603–1604.
15. Okazumi S, Isono K, Enomoto K, et al. Evaluation of liver tumors using fluorine-18-fluorodeoxyglucose PET: characterization of tumor and assessment of effect of treatment. *J Nucl Med* 1992;33:333–339.
16. Ishizu K, Sadato N, Yonekura Y, et al. Enhanced detection of brain tumors by [^{18}F]fluorodeoxyglucose PET with glucose loading. *J Comput Assist Tomogr* 1994;18:12–15.
17. Delbeke D, Meyerowitz C, Lapidus RL, et al. Optimal cutoff levels of fluorine-18-fluorodeoxyglucose uptake in the differentiation of low-grade from high-grade brain tumors with PET. *Radiology* 1995;195:47–52.