Exhibit H

Perelman School of Medicine
University of Pennsylvania

Ruben C. Gur, PhD
Professor, Departments of Psychiatry, Radiology & Neurology
Director, Brain Behavior Laboratory and the Center for Neuroimaging in Psychiatry

PATIENT'S NAME:	Freddie Owens
DATE OF BIRTH:	9/25/1974
DATE OF REPORT:	9/2/2016
INTEGRATION BY:	Ruben C. Gur, PhD
REFERRED BY:	Hank Ehlies

RE: NEUROBEHAVIORAL ASSESSMENT OF MR. OWENS

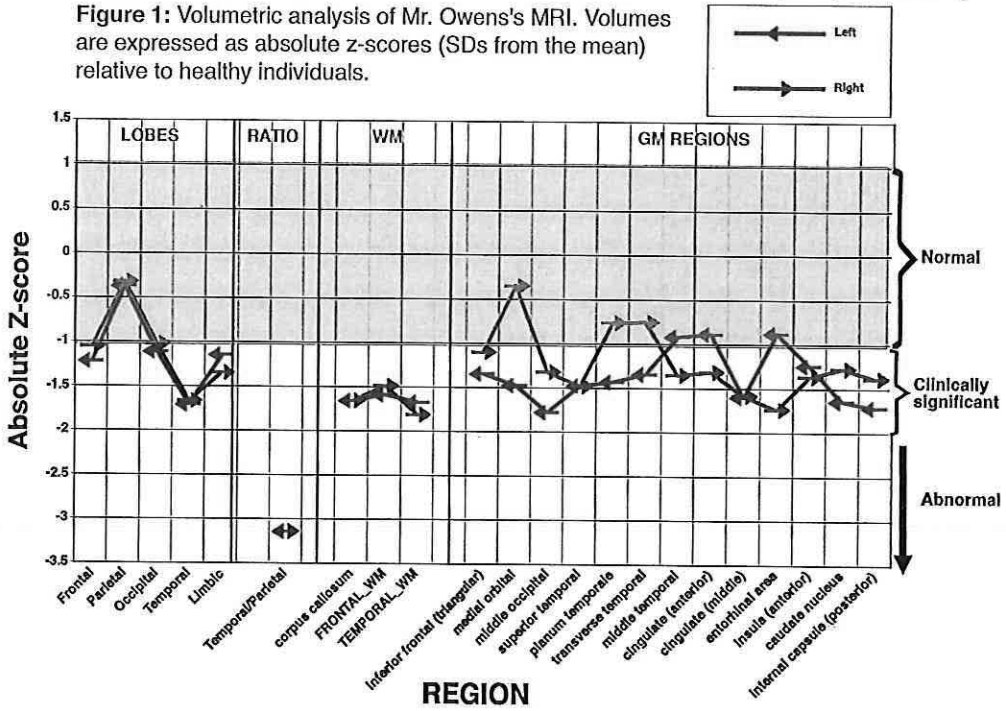
Background

Mr. Owens was referred for a comprehensive neurobehavioral assessment, comprising of analyses of structural and functional neuroimaging. The following studies were performed on Mr. Owens at the Medical University of South Carolina in Charleston, South Carolina, and I have reviewed and summarized them in this report: volumetric analysis of magnetic resonance imaging (MRI), and quantitative analysis of positron emission tomography (PET). Notably, the analyses conducted here are not designed to replace a routine clinical reading of the scans by a neuroradiologist or a nuclear medicine physician, which should reveal any potentially life-threatening finding such as a tumor, arteriovenous malformation or stroke, as well as any focal findings such as multiple sclerosis plaques or extensive local gliosis. Instead, the aim is to find and document evidence for more diffuse tissue loss, such as results of mild traumatic brain injury, toxin exposure, or regions that show abnormalities in resting state ("default mode" activity.) Diffuse volume loss is not usually detectable by visual inspection of the images generated by the scan, and require the application of standard software to the actual data that generate the images in order to quantify volume – in the case of MRI – or cerebral metabolic rates for glucose – in the case of PET.

Results of Magnetic Resonance Imaging (MRI): Volumetric structural analysis

The MRI was examined quantitatively by Christos Davatzikos, PhD, via delineation of regions of interest (ROI) assisted by a deformable registration algorithm[1] **(Figure 1)**. Regions showing a reduction in volume of > 1 standard deviations (SD) below normal, and their corresponding contralateral structures, are displayed below.

Figure 1: Volumetric analysis of Mr. Owens's MRI. Volumes are expressed as absolute z-scores (SDs from the mean) relative to healthy individuals.



These results show that the overall volume of Mr. Owens' brain is in the average to below average range, with the frontal, temporal, and limbic lobes being below average (see profile of LOBES in Figure 1). Such variability among lobes is abnormal, as indicated by the temporal-to-parietal lobe ratio, which exceeds 3 SDs below normal (see under RATIO in Figure 1). Frontal and temporal lobe white matter volume is reduced, as well as the corpus

[1] Yangming Ou, Aristeidis Sotiras, Nikos Paragios, Christos Davatzikos, "DRAMMS: deformable registration via attribute matching and mutual-saliency weighting," Medical Image Analysis, 2011;15, 622-639. Ou, Y., Akbari, H., Bilello, M., Da, X., Davatzikos, C., "Comparative Evaluation of Registration Algorithms in Different Brain Databases With Varying Difficulty: Results and Insights," IEEE Trans. On Medical Imaging, 2014; 33, 2039-2065.

10th Floor Gates Pavilion | 3400 Spruce Street | Philadelphia, PA 19104 | 215.615.3604 | Fax: 215.662.7903 | gur@upenn.edu

callosum, all bilaterally. Some volume reduction is also seen in specific gray matter regions, including frontal, occipital and temporal cortex as well as limbic and striatal regions.

Results of Positron Emission Tomography (PET)

The study examined the regional distribution of cerebral glucose metabolic activity using fluorine-18 labeled deoxyglucose (FDG). The technical quality of the scan was excellent. Dr. Andrew Newberg read the study clinically: "The scan results demonstrate mild to moderately decreased metabolism in the white matter tracts particularly on the right, the limbic structures such as the hippocampus and amygdala, the pons, uncus, and the temporal poles. Several cortical areas also have mildly to moderately increased metabolism including the anterior cingulate, caudate nuclei, supramarginal gyrus, visual cortices, putamen, right posterior cingulate, angular gyrus, superior parietal lobe, and precuneus. There is also moderately increased metabolism throughout a number of right frontal lobe structures including the right dorsal medial cortex, medial frontal, inferior frontal, dorsolateral prefrontal cortex, and sensorimotor cortex." (Dr. Newberg's report of July 26, 2016). These PET scans are displayed in **Figures 2 and 3**.

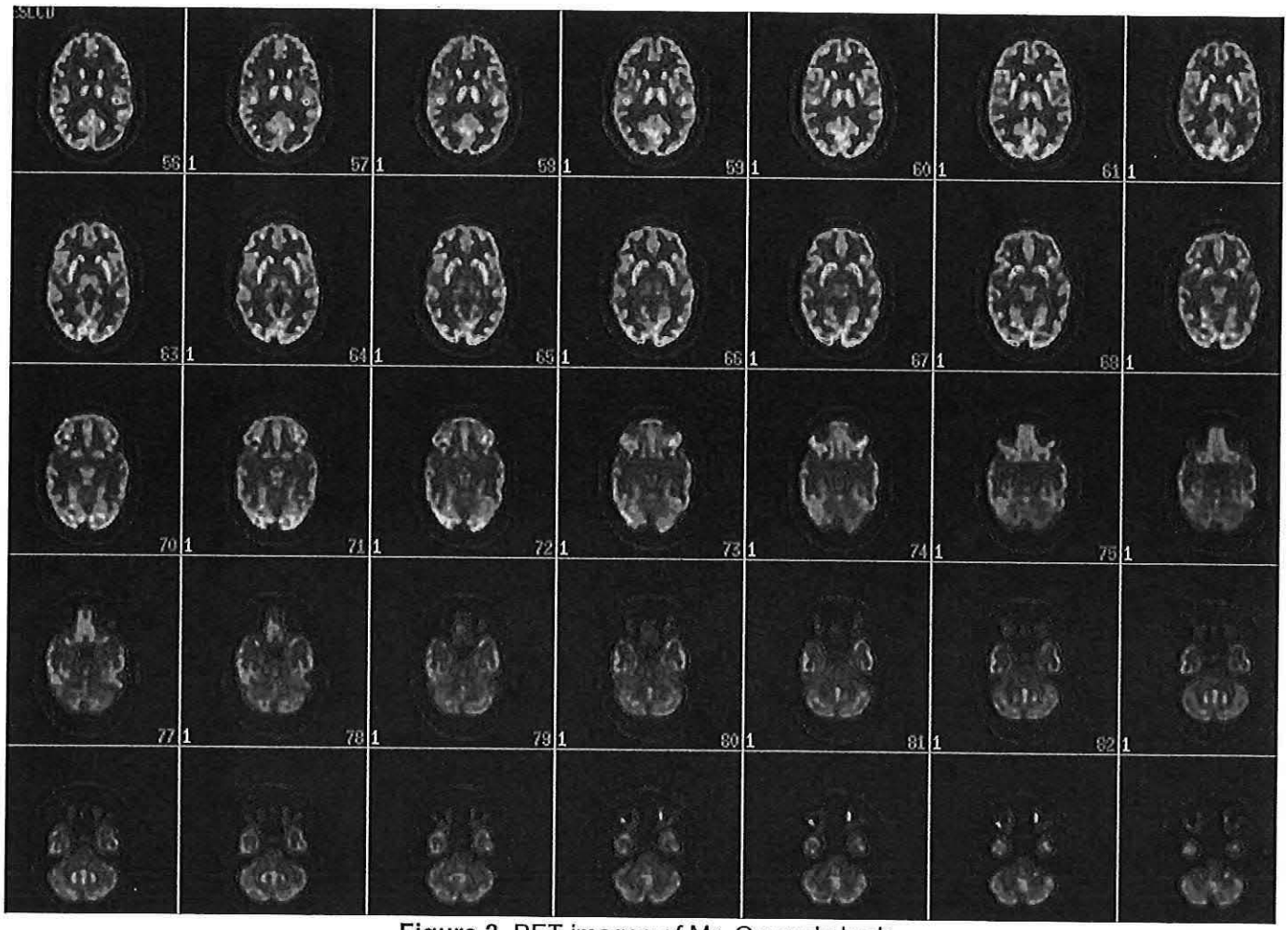


Figure 2. PET images of Mr. Owens's brain

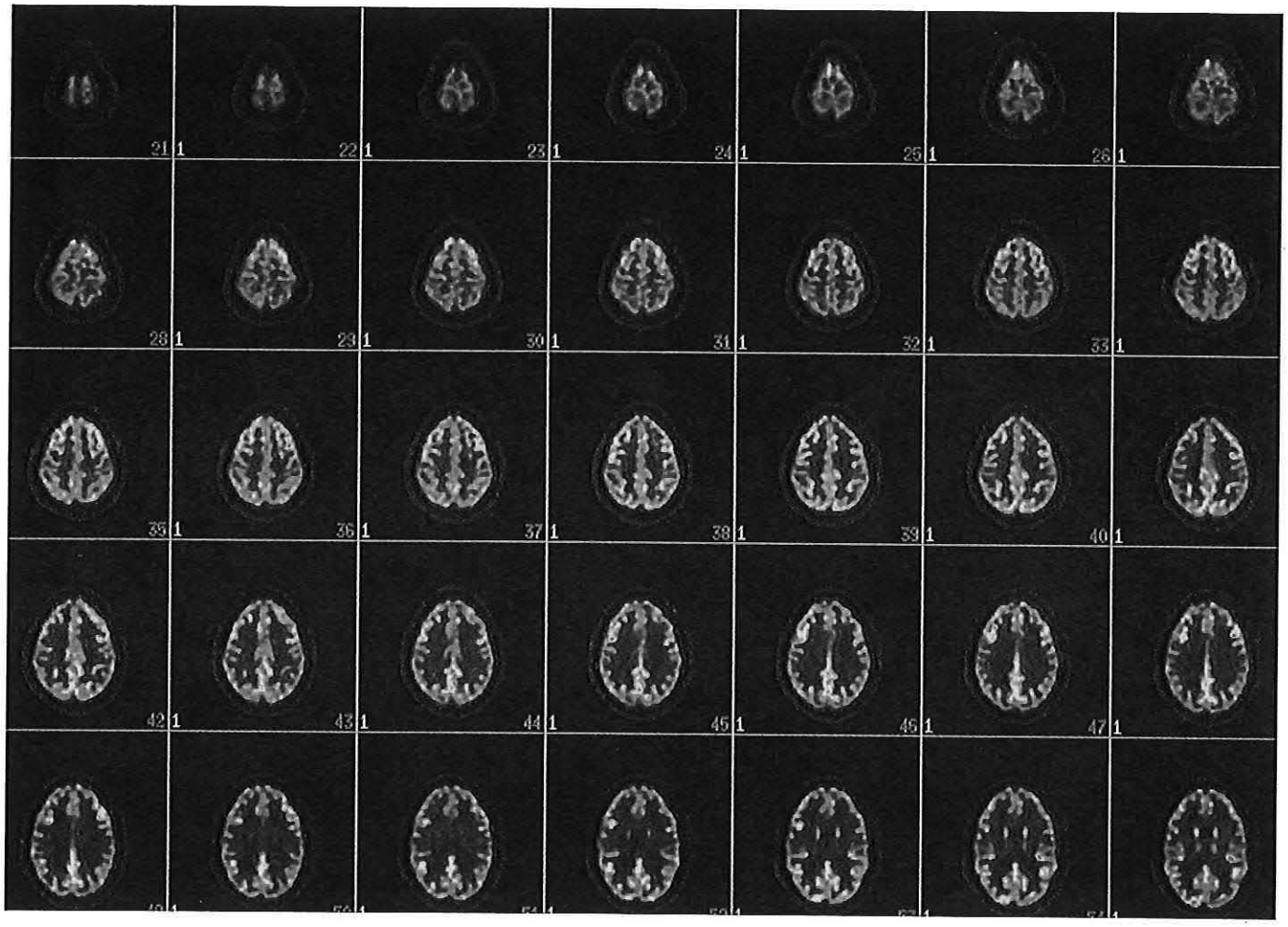
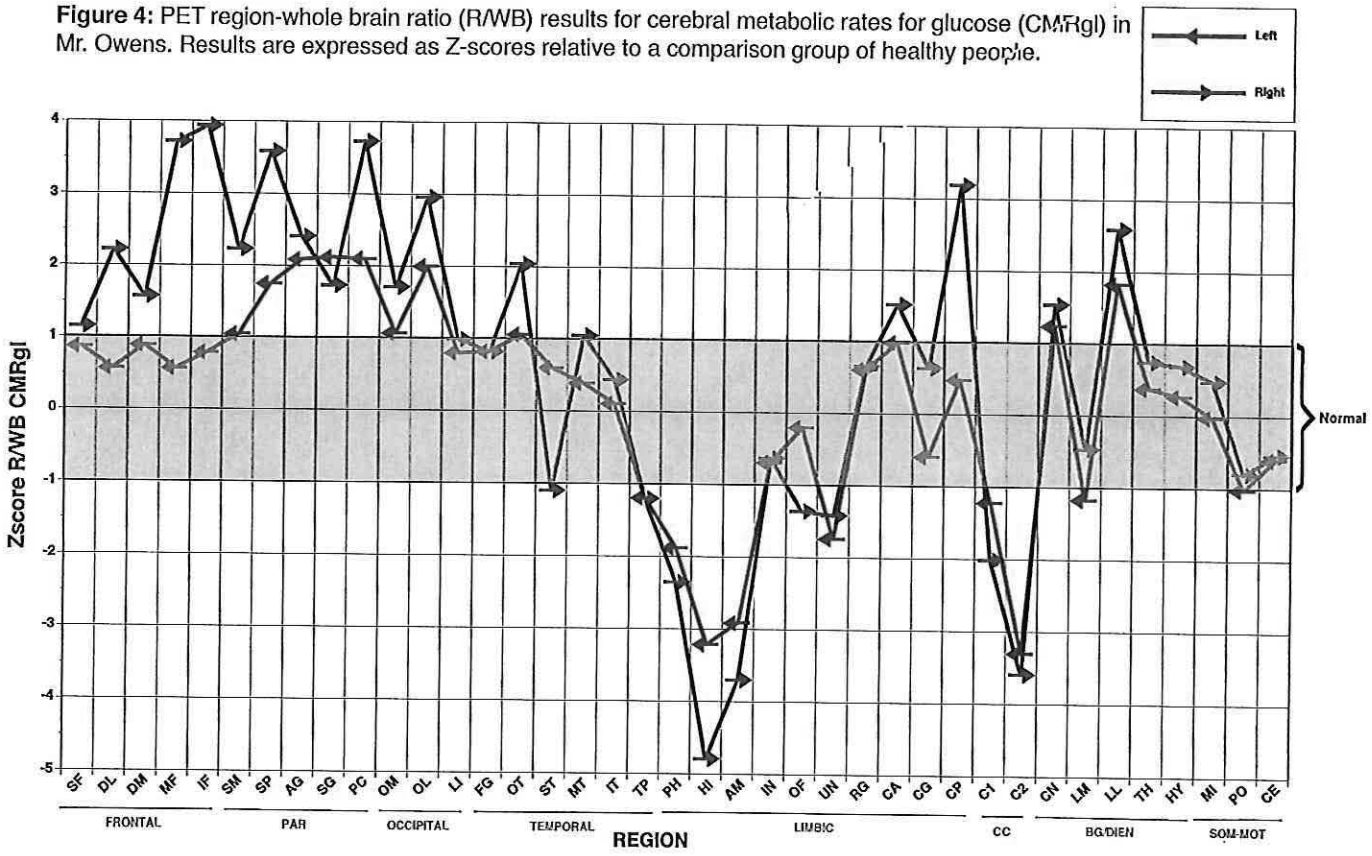


Figure 3. PET images of Mr. Owens's brain

The PET study was subjected to a quantitative analysis using a standard regions of interest (ROI) approach.[2] The quantitative analysis of cerebral metabolic rates relative to whole brain (**Figure 4**) supports Dr. Newberg's clinical reading and points to more specific sets of regions that show abnormal glucose uptake. The analysis indicated relative decreases in 9 regions (see key for region abbreviations in **Figure 4**): TP, PH, HI, AM, right OF, UN, C1, C2, and left LM (when hemisphere not indicated, it is bilateral). Seventeen (17) areas show relative increases in metabolism: right SF, right DL, right DM, right MF, right IF, right SM, SP, AG, SG, PC, right OM, OL, right OT, right CA, right CP, CN, and LL.

[2] Gur RC, Mozley LH, Mozley PD, Resnick SM, Karp JS, Alavi A, Arnold SE, and Gur RE. (1995). Sex differences in regional cerebral glucose metabolism during a resting state. *Science*. *267*:528-531.

Figure 4: PET region-whole brain ratio (R/WB) results for cerebral metabolic rates for glucose (CMRgl) in Mr. Owens. Results are expressed as Z-scores relative to a comparison group of healthy people.



SF = Superior Frontal; DL = Dorsal Prefrontal - Lateral; DM = Dorsal Prefrontal - Medial; MF = Mid-Frontal; IF = Inferior Frontal; SM = Sensorimotor; SP = Superior Parietal; AG = Angular Gyrus; SG = Supramarginal Gyrus; PC = Precuneus; OM = Occipital cortex, Medial; OL = Occipital cortex, Lateral; LI = Lingual Gyrus; FG = Fusiform Gyrus; OT = Occipital Temporal; ST = Superior Temporal; MT = Mid-Temporal; IT = Inferior Temporal; TP = Temporal Pole; PH = Parahippocampal Gyrus; HI = Hippocampus; AM = Amygdala; IN = Insula; OF = Orbital Frontal; UN = Uncus; RG = Rectal Gyrus; CA Cingulate Gyrus = Anterior; CG = Cingulate Gyrus - genu; CP = Cingulate Gyrus - Posterior; C1 = Corpus Callosum - Anterior; C2 = Corpus Callosum - Posterior; CN = Caudate Nucleus; LM = Lenticular - Medial [Globus Pallidus]; LL = Lenticular - Lateral [Putamen]; TH = Thalamus; HY = Hypothalamus; MI = Midbrain; PO = Pons; CE = Cerebellum.

These results indicate a pattern of hypo-activation in the limbic system (parahippocampal gyrus, hippocampus, and amygdala) and corpus callosum. At the same time there is hyper-activation across cortical regions and in the cingulate gyrus (anterior and posterior), caudate nucleus, and globus pallidus. Notably, the most striking areas of hypermetabolism are all on the right hemisphere (mid-frontal and inferior frontal gyri, superior parietal gyrus, precuneus, and posterior cingulate gyrus, which exceed 3 SDs higher in activation than normal). The hippocampus, amygdala, and posterior corpus callosum are near or exceeding 3 SDs below normal, exhibiting moderate to severe hypoactivation.

Summary and Conclusions

Results of structural and functional imaging of Mr. Owens's brain converge to show abnormalities indicating brain damage. These abnormalities are in regions that are very important for regulating emotions and behavior.

Several components of the emotion regulation system show dysfunction in Mr. Owens: (1) the amygdala, which activates in response to stressful or threatening environmental stimuli; (2) the insula, which moderates the experience of anxiety in response to threatening stimuli; and (3) the cingulate region, which resolves conflicts between top-down cognitive information and bottom-up emotional instincts.

The abnormalities observed in the frontal regions of Mr. Owens's brain would indicate diminished executive functions such as abstraction and mental flexibility, planning, moral judgment, and emotion regulation, moderating limbic arousal, and especially impulse control. The volume loss in the temporal lobe would portend memory deficits, and the white matter abnormalities would interfere with efficient communication among brain regions.

A most relevant finding is the reduced metabolism in the hippocampus and amygdala, combined with hyper-metabolism in cortical regions. Abnormally high metabolism in cortical regions most likely indicates compensatory hyper-vigilance of the

"default mode" brain state. Such a pattern of activation indicates that Mr. Owens has an inappropriately high level of cognitive control over emotion processing and experience. Furthermore, reduced activity in emotion-related limbic regions, as is the case here, could result in "kindling" or sudden hyper-excitability. A damaged amygdala will misinterpret danger signals and when excited it will issue false alarms that require intact frontal components of the limbic system for modulation. In Mr. Owens's case, the cortex is already at a hyper-activated state. When Mr. Owens's amygdala becomes activated, his frontal lobe would be unable to exercise control as a normal frontal lobe would, because his "thinking brain" is not only damaged but is already operating at full capacity in its hyper-vigilant state. The situation is analogous to a car with weak brakes that are already engaged when it begins to race. The frontal lobe is unable to do its job and act as the brakes on the primitive emotional impulses emanating from the amygdala when the limbic system reaches its activated stage (the Klüver-Bucy syndrome).

There's also low metabolism in the corpus callosum, which can lead to deficits in integrating verbal reasoning and analytic processing modes of the left hemisphere with intuitive, integrative, and affect-related processing modes of the right hemisphere. Such white matter dysfunction would also impair speed of processing across all cognitive and affective domains.

The etiology of these abnormalities is difficult to determine and requires clinical evaluation and integration with history. However, the abnormalities observed are consistent with several causes, including traumatic brain injury and fetal alcohol exposure. This brain profile is also consistent with that of someone with epilepsy or a seizure disorder.

Thank you for the opportunity to participate in Mr. Owens's evaluation. Please let me know if you have questions or need further elaboration or analysis.

Sincerely,

Ruben C. Gur, PhD
Professor, Departments of Psychiatry, Radiology & Neurology
Director, Brain Behavior Laboratory and the Center for Neuroimaging in Psychiatry