Neuroscience

Among the major trends that I see emerging in neuroscience are β-amyloid imaging, with 26 abstracts on that topic presented at this meeting and a growing number of presentations on dopamine transporter (DAT)/vesicular monoamine transporter (VMAT) imaging, with 25 abstracts. Small animal imaging has made a surprising impact in neuroscience with 22 abstracts. We are seeing an increasing variety of neuroreceptor radioligands, but ^{18}F-FDG PET studies still predominate, with the largest number of abstracts at this meeting.

The main areas of research activity—the big 4—are cognitive decline/dementia, psychiatric disorders, Parkinson disease (PD), and brain tumors. We are also seeing interesting clusters of work in drug mechanisms, drug/alcohol abuse, epilepsy, traumatic brain injury (TBI), and what is called "chemo brain" or the cognitive effects of chemotherapy.

β-Amyloid Imaging

Chet Mathis, the codeveloper at the University of Pittsburgh with William Klunk of Pittsburgh Compound B (PiB), received the 2010 Aebersold award at this meeting. The first study was performed in 2002, with the first publication in 2004. Today we are beginning to see the results of longitudinal studies with this compound in large numbers of patients. One such study came from Villemagne et al. [383] from the Austin Hospital, University of Melbourne, National Aging Research Institute, and National Mental Health Institute (all in Melbourne, Australia) and the Commonwealth Scientific and Industrial Research Organisation Preventative Health National Research Flagship (Brisbane, Australia), who reported on "Longitudinal assessment of Aβ burden and cognition with ^{11}C-PiB PET in aging and Alzheimer's disease." They looked at 100 healthy elderly volunteers, 65 individuals with mild cognitive impairment (MCI; which implies a high risk of Alzheimer disease [AD]), and 40 patients with AD. They defined a normal cutoff range for a negative scan and found that 33% of normal elderly people have amyloid deposits in the brain. For those with MCI, this figure was 68%, in accord with the estimated 60% of people with this condition expected to develop AD. For those already clinically diagnosed with AD in the study, 98% had positive scans. Looking at serial studies in this cohort, it is now clear that amyloid builds up extremely slowly. In those with a positive scan among the normal controls, the rate of increase is about 1%/y. In those with MCI this rate appears to be about 2%/y, and in early AD about 3%—however, in later scans it seems to plateau. It is not good to have amyloid in the brain. These studies show that healthy elderly controls with amyloid in the brain had a 14% risk of developing objective MCI or AD within 2 y compared with only a 1% risk for elderly controls with negative scans. Those who already had objective cognitive impairment but were not demented but had positive scans had a 66% chance of being clinically diagnosed as demented within 2 y, compared with only 5% for those with objective cognitive impairment but negative scans.

So ^{11}C-PiB, on which there were 12 abstracts at the meeting, is making great inroads into our understanding and has huge potential for early diagnosis of AD. However, it is not widely available. I am pleased to report that 3 ^{18}F-labeled tracers are in phase 3 clinical trials, and we expect the first of these to reach clinical practice within 2 y. At this meeting 11 abstracts focused on the Bayer Schering product ^{18}F-florbetaben (or AV-1), 4 abstracts on the Avid product ^{18}F-florpiramine (or AV-45), and 1 abstract on the GE product ^{18}F-flutemetamol (or 3'-F-PIB).

Christopher Rowe, MD

Sabri et al. [384] from the University of Leipzig (Germany), Helios Hospital Berlin-Buch (Germany), Charité-Campus Benjamin Franklin (Berlin, Germany), Ludwig-Maximilian University (Munich, Germany), Bayer Healthcare (Berlin, Germany), and Molecular Neuro-Imaging (New Haven, CT) reported on a "Multicentre phase 2 trial on florbetaben for β-amyloid brain PET in Alzheimer's disease." The study included 150 participants. The scan had 80% sensitivity and 90% specificity in distinguishing AD from normal controls, compared against the results of clinical diagnosis. Clinical diagnosis is far from perfect—so we do would not want to see 100% agreement here. These figures are quite encouraging.

Villemagne et al. [1786] from the Austin Hospital (Melbourne, Australia), Bayer Schering Pharma (Berlin, Germany), and the Mental Health Research Institute (Melbourne, Australia), reported on "^{18}F-florbetaben PET imaging in the differential diagnosis of dementia." Figure 43 shows extensive uptake in cortex in patients with AD. In those with frontotemporal dementia the scans are negative, with only nonspecific white matter binding, similar to controls. These scans have excellent clinical potential for distinguishing frontotemporal dementia from AD. No uptake is seen in pure vascular dementia or PD, but intermediate grades of uptake are seen in MCI and dementia with Lewy bodies.

Ong et al. [438] from the Austin Hospital (Melbourne, Australia), Bayer Schering Pharma (Berlin, Germany), and the Mental Health Research Institute (Melbourne, Australia), reported on "Assessment of Aβ deposition in mild cognitive impairment with ^{18}F-florbetaben PET." Although the data are preliminary, results with this tracer are looking very much like those with PiB. Twenty-one subjects have reached the 1-y follow-up endpoint, with 5 of 11 participants in the florbetaben-positive group and none of the 10 participants in the florbetaben-negative group progressing to AD.

An extremely important paper, "Florbetapir (^{18}F-AV-45) PET imaging of β-amyloid plaque is highly correlated with histopath-

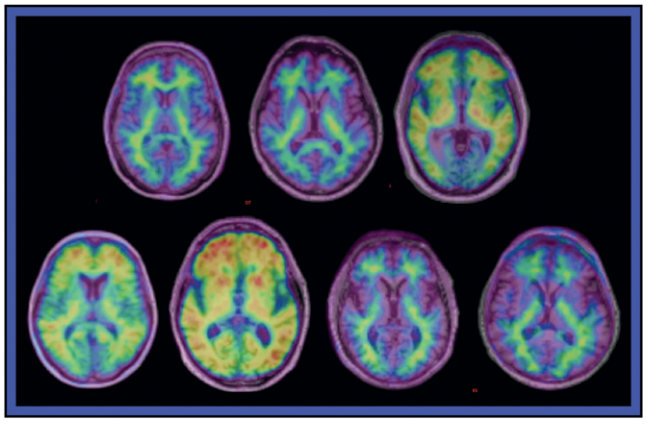

FIGURE 43. ^{18}F-florbetaben PET in differential diagnosis of dementia in: (top row, left to right) healthy control, Parkinson disease, dementia with Lewy bodies; and (bottom row, left to right): mild cognitive impairment, Alzheimer disease, frontotemporal lobe disease, and pure vascular dementia.

ologic assays at autopsy," was presented by Mintun et al. [387] from Washington University (St. Louis, MO), representing the members of the AV-45-A07 Study Group. In this phase 3 study, participants with life expectancies of <6 mo were asked to volunteer for a PET scan and to donate their brains after death. A 10-min PET scan was performed 50 min after injection, was read visually, and cortical-to-cerebellar ratios were calculated. These results were compared with post mortem quantitation of amyloid. Figure 44 shows the 6 results that have been presented so far. Note that 1 person, who was diagnosed with AD in life and whose scan was negative, was found at post mortem to have no evidence of AD. All studies showed an excellent correlation between the amount of tracer uptake on the brain scans and immunohistochemical assessment of amyloid burden.

β-amyloid imaging, then, has a very high sensitivity for AD and should be a useful tool to assist in early diagnosis of this condition. It appears to accurately distinguish AD from frontotemporal dementia and can predict progression from MCI to AD. It can also detect preclinical AD in apparently healthy elderly individuals, although the mechanisms and factors governing timing or "lag phase" to dementia still must be fully elucidated. The ability to detect preclinical AD may allow early intervention to prevent or significantly delay the dementia of AD once effective therapy has been developed.

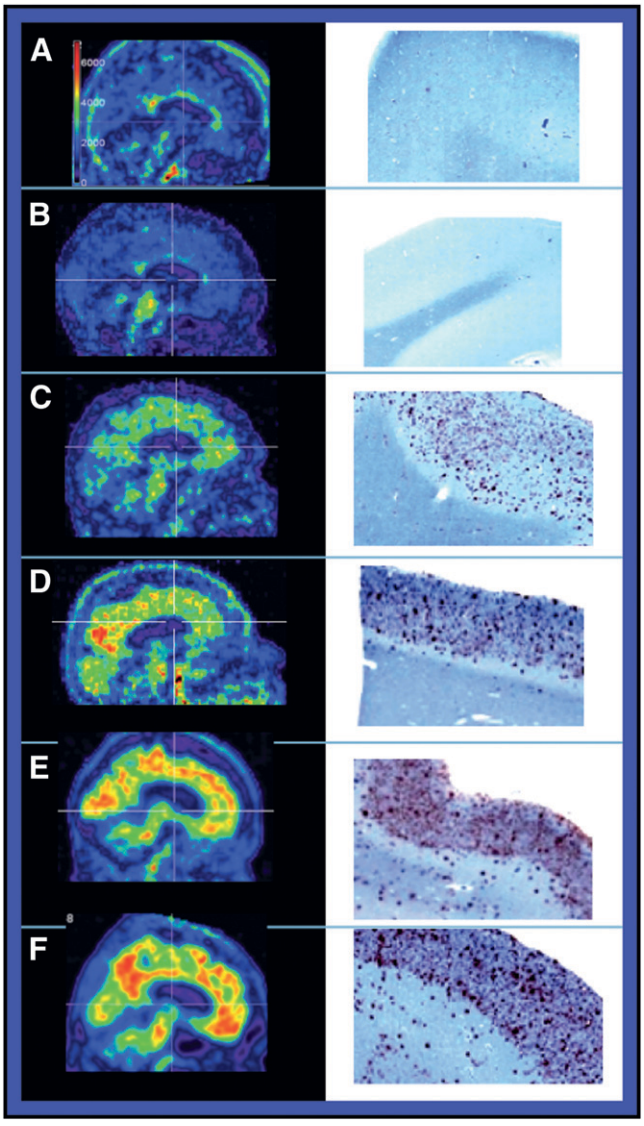

FIGURE 44. Florbetapir PET (left column) and amyloid staining at autopsy in: (A) mild cognitive impairment; (B, D–F) Alzheimer disease; and (C) Parkinson disease.

AD is a challenging problem. It currently affects 25% of those aged 85 and older. The cost of AD in the United States is $172 billion/y. By 2050, 16 million Americans will have AD, and the cost will be $1 trillion per year. Molecular neuroimaging will play an important role in fighting the enormous health problem of AD. This will be a major growth area for nuclear medicine—in fact, my prediction is that in the future people who are worried about AD, who have subjective memory complaints, who are carriers of genes (eg, APO e4) that increase the risk of AD, who have a family history of AD, or who test themselves on serial cognitive testing (such as that available on the Internet) and note a decline over time, will come either for β-amyloid imaging or cerebrospinal fluid analysis. If found to be positive for brain amyloid, they will be put on drugs that have been developed to reduce β-amyloid and prevent AD. Of course, these drugs currently do not exist, but many candidates are in clinical trials.

^{18}F-FDG PET neuroimaging is still very important and, in my opinion, underutilized in this country. In my center, we use quantitative techniques and find that it adds significantly to the clinical management of patients. Thiele et al. [441] from Philips Research North America (Briarcliff Manor, NY), the University of Washington (Seattle), and Philips Research Europe (Hamburg, Germany) reported on "Prediction of cognitive decline in patients with mild cognitive impairment: observer-independent voxel-based classification of FDG PET." They looked at 140 patients with MCI who are participating in the AD Neuroimaging Initiative (ADNI) study. The researchers used the 3D-SSP Neurostat program and a multivariate automatic classifier of PET data to predict who would progress to AD. At baseline, all participants had Mini-Mental Status Examination (MMSE) scores ≥ 25 and underwent baseline ^{18}F-FDG PET. At 2-y follow-up and repeat MMSE, patients were classified as stable (MMSE ≥ 25; 99 individuals) or progressive (MMSE < 25; 41 individuals). They found that PET data analysis predicted 81% correctly.

The ADNI, which is currently running in the United States, has 800 individuals who are undergoing serial MR imaging every year. Four hundred of these individuals are having ^{18}F-FDG PET imaging every year, and 95 are having PiB scans every year. These data, along with clinical data and other information, are available on a Web site (www.lonu.ucla.edu/ADNI/) for any researcher to access, download, and analyze. A study with which I am closely involved, the Australian Imaging Biomarkers and Lifestyle study of aging, also has placed 290 subjects with MR and PiB scans on this Web site.

Drzezga et al. [381] from the Massachusetts General Hospital/Harvard Medical School (Boston), Howard Hughes Medical Institute (Chevy Chase, MD), and the Technische Universität (Munich, Germany) reported on the "Relation between metabolism, functional connectivity, and β-amyloid burden in predementia stages of Alzheimer's disease." These researchers used MR imaging to look at the effect of amyloid in the brain in individuals who were either normal or had very early-stage MCI. MR imaging can show which parts of the brain have the greatest number of connections to other parts. Areas of high connectivity

are called cortical hubs. Functional MR imaging maps whole-brain connectivity to identify these cortical hubs. These authors found that even in individuals who are asymptomatic but have amyloid in the brain, hypometabolism and disrupted connectivity can be detected in corresponding areas (Fig. 45). So even a little bit of amyloid in the brain is not a good thing.

So what can an individual do at the present time if he or she is found to have amyloid in the brain? The current line is that one should keep the body and mind regularly exercised and address cardiovascular risk factors aggressively (because these are also risk factors for dementia and AD). Do these strategies work? Förster et al. [1822] from Ludwig-Maximilian University (Munich), Technische Universität (Munich), University Rostock, Johann Wolfgang Goethe University (Frankfurt), and University Mainz (all in Germany) reported on "Effects of a stage-specific cognitive intervention on brain metabolism in patients with aMCI and mild AD." They looked at the effect of mental stimulation programs on patients with MCI and early AD. On ^{18}F-FDG PET the placebo group showed a significant decline in metabolism over time, whereas those receiving the cognitive intervention showed little or no decline. So this is encouraging evidence that these programs may work.

DAT/VMAT Imaging

Seven SPECT and 8 PET abstracts focused on DAT imaging at this meeting, with 10 PET abstracts on VMAT imaging. Figure 46 is an ^{18}F VMAT compound, ^{18}F-AV-133, from Avid Radiopharmaceuticals (Philadelphia, PA), showing uptake in a normal healthy elderly man on the left and a patient with mild PD on the right. I like to describe what is seen as "rabbits in the brain," with the "head" and the "ears" (the caudate nucleus) above the "body" of the bunny (the putamen). In even the mildest forms of PD we see dramatic loss of the body of the rabbit.

Villemagne et al. [442] from the Austin Hospital, the Howard Florey Institute of Experimental Physiology and Medicine, and the Mental Health Research Institute (all of Melbourne, Australia); Tohoku University (Sendai, Japan); and Avid Radiopharmaceuticals and the University of Pennsylvania (both of Philadelphia) reported on "Differential diagnosis of dementia with Lewy bodies from Alzheimer's disease using ^{18}F-AV-133, a novel PET tracer for VMAT2." They showed that this tracer is very accurate in distinguishing between the 2 diagnoses (Fig. 47). Dementia with Lewy bodies is the second most commonly

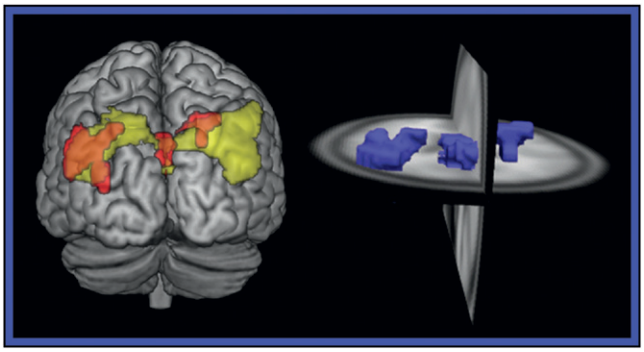

FIGURE 45. Overlap of hypometabolism and disrupted connectivity in mild cognitive impairment. Left: Areas of hypometabolism are represented in yellow; areas of connectivity disruption are represented in orange. Right: 3D-rendered cutaway of overlap (blue).

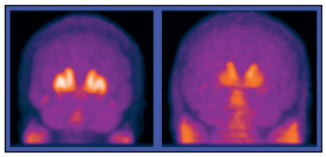

FIGURE 46. ^{18}F-AV-133 PET vesicular monoamine transporter imaging in: (left) a healthy elderly man; and (right) an individual with mild Parkinson disease (PD). The characteristic "rabbit" sign is seen in healthy individuals, but only the "head" and "ears" remain in those with PD.

diagnosed cause of dementia at autopsy but is diagnosed in only about 40% of cases in life.

Kotagal et al. [55] and the group from the University of Michigan (Ann Arbor) have been using these techniques and compounds, such as ^{11}C-PMP AChE (for cholinergic innervation) and ^{11}C-DTBZ (for VMAT or dopaminergic innervation), to look at various clinical aspects of PD and to assess how these factors interact. They reported that "REM sleep behavior disorder (RBD) is associated with cortical cholinergic deficits in Parkinson disease without dementia." They studied RBD, also known as "acting out dreams" and associated with an increased risk of developing PD and dementia in PD, in 42 patients with PD without dementia. They found an association between RBD and cholinergic but not dopaminergic innervation, suggesting that RBD is associated with cortical cholinergic loss that may herald the development of dementia (Fig. 48).

^{18}F-FDG can still play a role in movement disorders. Tang et al. [1782] from the Feinstein Institute for Medical Research (Manhasset, NY), New York University School of Medicine (New York), and Stanford University School of Medicine (CA) reported on "Accurate differential diagnosis of parkinsonism by pattern analysis of metabolic imaging data." This group used pattern recognition in 167 patients presenting with parkinsonism. Participants underwent ^{18}F-FDG PET imaging and were followed for 2.6 y before a final clinical diagnosis was made. At final diagnosis, 96 had idiopathic PD (IPD), which responds well to dopaminergic treatment. Forty-one had multisystem atrophy (MSA) and 30 had progressive supranuclear palsy (PSP), neither of which responds well to currently available treatments. The automated imaging classification results found characteristic metabolic patterns for idiopathic PD, with hypermetabolism in the globus pallidus, thalamus, and cerebellum, with mild hypometabolism in frontal and parietal cortex. In MSA, they found hypometabolism in the striatum and the cerebellum. In PSP they found hypometabolism in the frontal cortex. The specificities for these patterns were in the high 90% range (Fig. 49).

Other techniques for distinguishing dementias are available. La Fougère et al. [610] from the University of Munich (Germany)

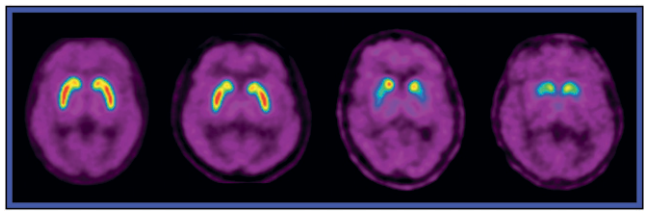

FIGURE 47. ^{18}F-AV133 PET images in: (left to right) a healthy individual and patients with Alzheimer disease, dementia with Lewy bodies, and Parkinson disease.

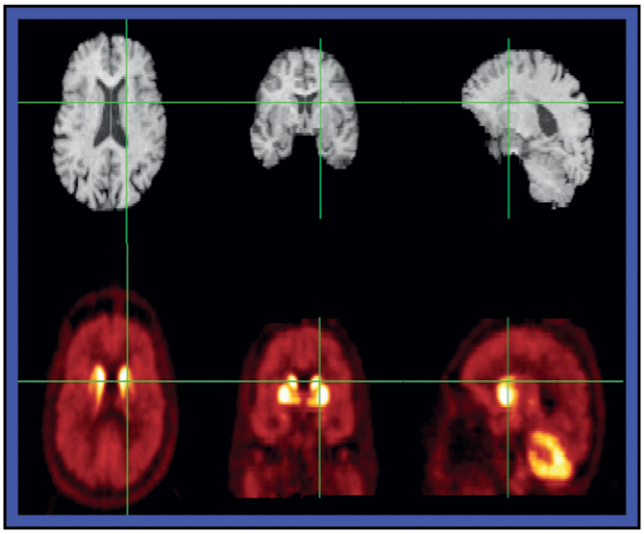

FIGURE 48. ^{11}C-PMP PET imaging in patients with Parkinson disease without dementia showed an association between rapid-eye movement sleep behavior disorder and cortical cholinergic deficits.

reported on "Differentiation of idiopathic and non-idiopathic parkinsonian syndromes with ^{18}F-desmethoxyfallypride." This ^{18}F-labeled D_2 receptor ligand has widespread clinical potential. In the more severe parkinsonian syndromes (MSA and PSP), the striatum is destroyed and D_2 receptors are lost. In IPD only the input from the midbrain is damaged and the striatal neurons are intact, so the D_2 receptors are preserved. In fact, the D_2 receptors upregulate in early PD, whereas in MSA and PSP they are lost. La Fougère et al. showed very high specificity and sensitivity (96% and 87%, respectively) for distinguishing IPD (with a good prognosis) from non-IPD syndromes with poor prognoses (Fig. 50). Their conclusion was that ^{18}F-desmethoxyfallypride could be easily used in routine clinical practice for this type of differential diagnosis.

PET/MR Neuroimaging

Figure 51 shows one system under evaluation from Siemens (Erlangen, Germany), in which the PET component is an insert that goes inside the 3T MR system. Herzog et al. [411] from the Institute of Neuroscience and Medicine (Jülich, Germany) reported on studies with this device in "Simultaneous FET-PET and fMRI in brain tumors using a PET/MRI hybrid scanner." The PET was used to define the extent of glioma, with the fMR imaging defining the extent of vital primary motor cortex. In Figure 52, both the extent of the tumor and the extent of brain activation (elicited by finger tapping) were assessed during the same acquisition. The neurosurgeon with this technology can now confidently perform a wide excision of this tumor, staying well clear of the vital motor center. Figure 52 also provides examples of the resolution of PET images acquired within the MR scan.

Some physical and technical challenges remain to be sorted out for PET/MR, particularly attenuation correction. Fei et al. [81] from Emory University (Atlanta, GA) are making progress in this regard and reported at this meeting on "MRI-based attenuation correction and quantification tools for combined MR/PET." Figure 53 is an upside-down brain phantom. The image shows PET with

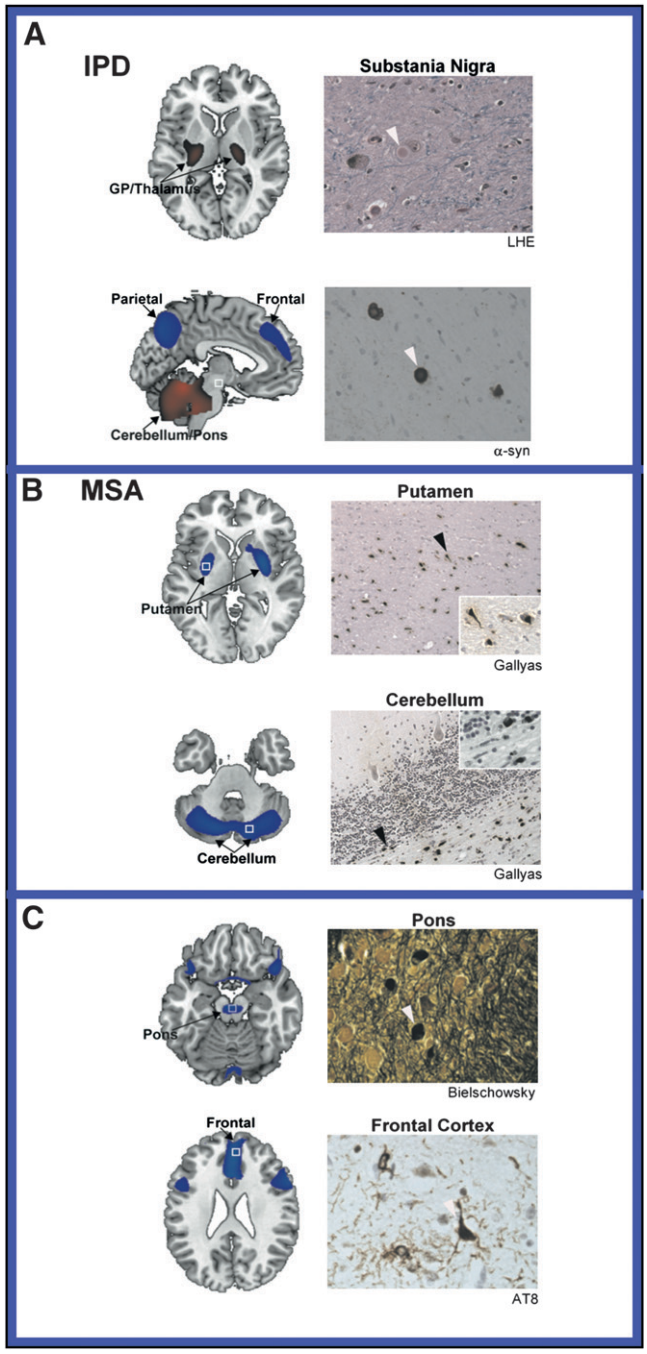

FIGURE 49. Automated imaging classification results found characteristic metabolic patterns of ^{18}F-FDG uptake on PET for: (A) idiopathic Parkinson disease; (B) multisystem atrophy; and (C) progressive supranuclear palsy. The ability of this technique to provide accurate differential diagnoses was confirmed with histochemical studies (right).

an HRRT camera with standard attenuation correction, the MR/PET without attenuation correction, and an image using one of the MR-based attenuation techniques, which looks quite satisfactory.

MicroPET

In the past I was skeptical about microPET, because mice have tiny brains compared with humans—but I have had to eat

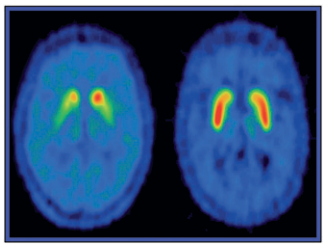

FIGURE 50. ^{18}F-desmethoxyfallypride PET differentiation of nonidiopathic (left) and idiopathic (right) Parkinsonian syndromes.

my words, because microPET is now finding a major place in neuroscience. Rominger et al. [105] from the University of Munich (Germany) reported that "Alpha$_2$-adrenergic drugs modulate the binding of ^{18}F-fallypride to dopamine $D_{2/3}$ receptors in striatum of living mouse." They looked at the effects of drugs on dopamine release in the striatum of the living mouse. Figure 54 shows the uptake of fallypride in the striatum of mouse brain. When the A_2-antagonist is administered, there is a release of dopamine, which washes off the fallypride. The resulting image is part of the first in vivo evidence for adrenergic modulation of dopaminergic release in mice. This may be relevant to actions of certain antipsychotic medications via adrenergic receptors and lead to improvements in drug development.

Brain Tumors

Brain tumors have been a focus of neuroimaging for many years. At this meeting papers were presented on research with ^{18}F-FDG, ^{11}C-methionine, ^{11}C-acetate, ^{11}C-choline, ^{18}F-FET, and ^{18}F-fluorodopa. Why has this array of tracers not advanced into clinical practice? Langen et al. [497] from the Institute of Neuroscience and Medicine (Jülich, Germany) and Aachen University Hospital (Germany) reported on the "Prognostic value of early ^{18}F-fluorethyltyrosine PET after radiochemotherapy in glioblastoma multiforme." They showed that the survival rate was much better in those who showed a response to chemotherapy

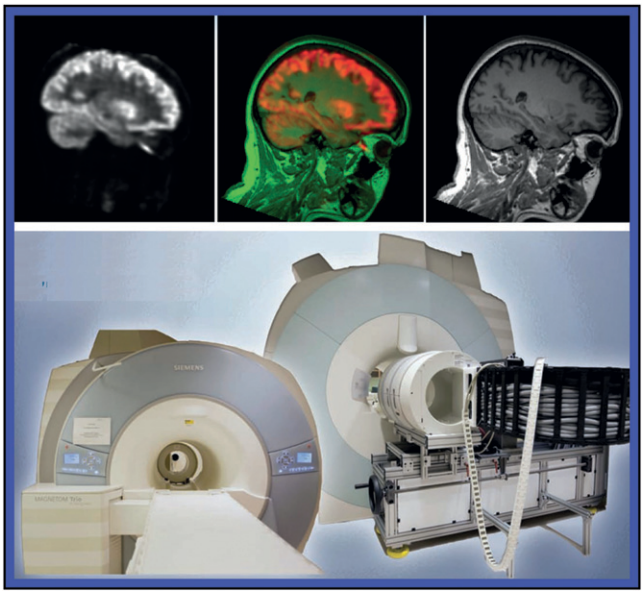

FIGURE 51. Siemens PET/MR system, with PET component as an insert inside the 3T MR unit, with test images above.

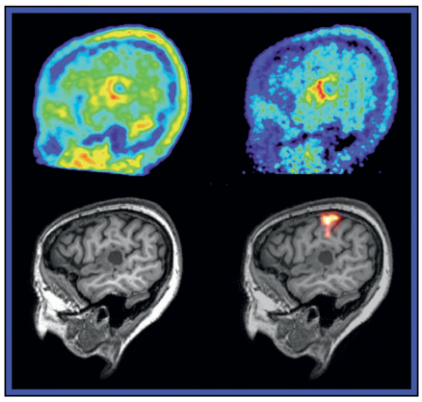

FIGURE 52. T1 and functional MR images acquired simultaneously with ^{18}F-FET PET images. Left column: (top) HR+ at 20–40 min after injection; (bottom): T1 MR image. Right column: (top) MR/PET image at 65–95 min after injection; (bottom): T1 MR plus functional MR during right finger tapping.

based on ^{18}F-FET PET. This tracer, however, is not yet making inroads into clinical practice, because we have a deficiency of data on its effectiveness and relative costs. The paper by Walter et al. [1801] from the University of California, Los Angeles on the "Impact of F-DOPA PET/CT imaging on the management of patients with brain tumors: The referring physician's perspective," was the sole presentation in the entire neuroscience stream at this meeting that looked at management impact. These authors issued pre- and postscan questionnaires to clinicians in 28 patients with suspected recurrence after MR imaging. They defined management change as a change in treatment modality and/or a switch from no treatment to treatment. Results showed significant management changes in more than 20% of patients who underwent ^{18}F-F-DOPA PET/(CT) imaging.

This is only the first step—it changed management, but did it improve the outcomes for patients? What we need and what is the essential and final step in translational research to accelerate basic discoveries into clinical practice is more comparative effectiveness research. This was the main theme of Henry Wagner's Highlights lecture last year. We need to see more of this in neuroscience, where the data are particularly lacking. We need to know more about the costs of our investigations compared with other investigations. We need to know about management change and whether such change actually benefits patients.

Traumatic Brain Injury and Neuroinflammation

Interesting findings on TBI were presented at this meeting. Much interest focuses on this area because mild TBI from blasts is the most common injury currently afflicting veterans returning

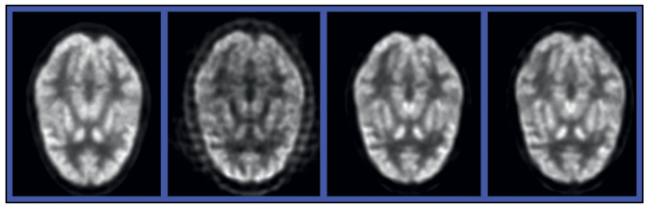

FIGURE 53. Images acquired with (left to right): a high-resolution research tomography PET, MR/PET with no attenuation correction, MR/PET with transmission-based attenuation correction, and MR/PET with MR-based attenuation correction.

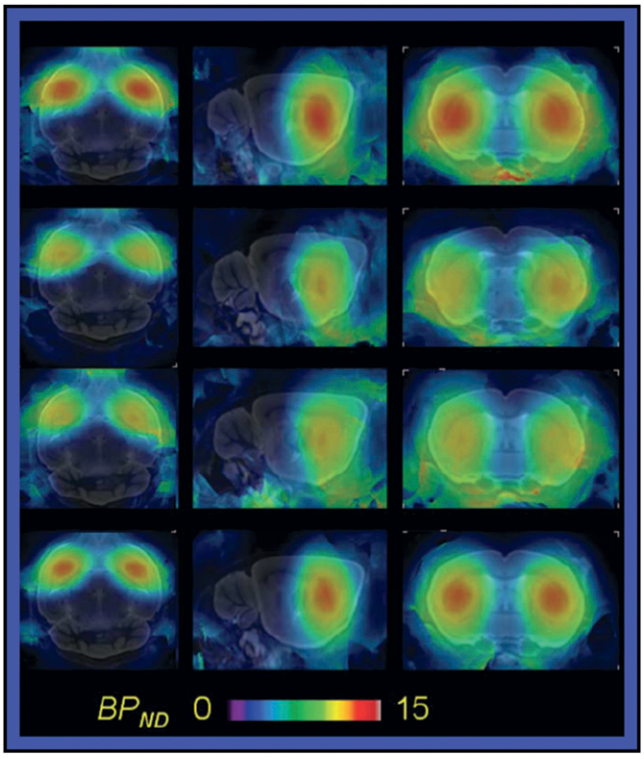

FIGURE 54. MicroPET images showing modulation of ^{18}F-fallypride binding to dopamine $D_{2/3}$ receptors in striatum of living mouse by administration of α_2-adrenergic drugs, including: (second row) yohimbe α_2-antagonist; (third row) RX821002 α_2-antagonist; (bottom row) clonidine α_2-antagonist. Top row: administration of saline as a control.

from military conflicts. Cross et al. [1813] from the University of Washington, the Northwest Network Mental Illness Research Education and Clinical Center, and Veterans Affairs Puget Sound Health Care (all in Seattle) reported on "Metabolic deficits in blast-exposed veterans with mild traumatic brain injury as revealed by FDG PET." In people with normal MR images but with symptoms of mild TBI, a significant reduction in metabolism was seen in the cerebellum and thalamus (Fig. 55). This pattern

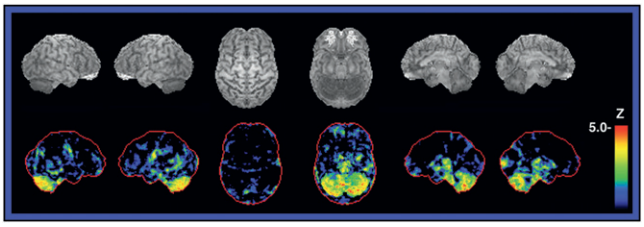

FIGURE 55. Template MR views (top) and Z-score maps of cerebral glucose metabolism differences (bottom) between individuals with mild traumatic brain injury (TBI) and normal individuals. Metabolism was decreased in cerebellum (−7.3%), thalamus (−6.4%), pons (−6.1%) and medial temporal lobe (−4.7%) in mild TBI. Top: template MR views. Views are (from left to right): right lateral, left lateral, superior, inferior, right medial, and left medial.

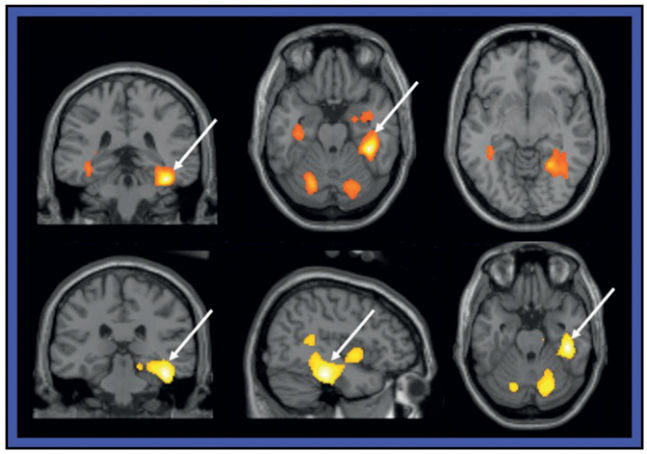

FIGURE 56. Effect of chemotherapy on regional metabolism in the brain. An increase in metabolism was noted in the medial temporal regions in postmenopausal women on aromatase inhibitor therapy. Bottom row shows differences between cancer group and controls in the study.

was seen consistently in mild TBI, regardless of blast events or time interval between injury and imaging.

Interesting research continues to focus on neuroinflammation ligands. Although we have had an ^{11}C ligand for many years, this has been unsatisfactory for most purposes. Work is progressing on better tracers for neuroinflammation, including ^{18}F-labeled compounds. Eberl et al. [33] from the Royal Prince Alfred Hospital and the Australian Nuclear Science and Technology Organisation Radiopharmaceutical Research Institute (both in Sydney, Australia) reported on "Quantitative analysis of the peripheral benzodiazepine receptor ligand ^{11}C-PBR170 in baboons with PET"; Dickstein et al. [1804] from the National Institute of Mental Health (Bethesda, MD) reported on "Comparison of 2 radioligands for PET imaging of translocator protein in human brain: [^{11}C]PBR28 and [^{18}F]PBR06"; and Carson et al. [355] from Yale University (New Haven, CT) and GlaxoSmith-Kline (London, UK) reported on "Comparison of 3 F-18 TSPO ligands in non-human primates." These groups are developing ^{18}F-labeled compounds that look very promising for neuroimaging, and I expect that over the next few meetings clinical studies will begin to appear.

The effect of chemotherapy in the brain is an area of interest. Romanowicz et al. [1804] from the University of Pennsylvania (Philadelphia) and the Gdansk Medical University (Poland) reported on "Effect of systemic cancer treatment on brain metabolism assessed by FDG PET." The authors described glucose metabolism as decreased bilaterally in the amygdala of patients treated with interferon for melanoma, and this was concordant with reports on the emotional impact of interferon therapy. Silverman et al. [1803] reported on "Changes in regional brain metabolism following aromatase inhibitor therapy in postmenopausal women: neuroscience summary." They noted an increase in metabolism in the medial temporal regions (Fig. 56).

Summary

Molecular neuroimaging will continue to have an important role in drug development and basic discovery in neuroscience.

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First MICoE Young Investigator Award Winners

Three abstracts were selected for recognition as winners of the newly initiated Molecular Imaging Center of Excellence (MICoE) Young Investigator Awards at the SNM Annual Meeting in Salt Lake City, UT. Seven abstracts were selected for presentation at the Young Investigators (YI) Symposium on June 6, and winners were announced at the MICoE Business meeting the next day. Those recognized include: (1) First place: Hongguang Liu, PhD, from the Molecular Imaging Program at Stanford, Department of Radiology and Bio-X Program, Stanford University CA), for "Noninvasive molecular imaging of radioactive tracers using optical imaging techniques." (2) Second place: Ambros Beer, MD, from Nuclear Medicine, Technical University of Munich (Germany), for "Correlation of $\alpha_v\beta_3$ expression, glucose metabolism, and tissue diffusivity by multimodality MR and PET imaging in cancer patients." The runner-up was I-Chih Tan, PhD, from the Center for Molecular Imaging, The Brown Foundation Institute of Molecular Medicine, The University of Texas Health Science Center (Houston), for "NIR fluorescence imaging of response to therapy in normal and lymphedema subjects."

Other abstracts selected for presentation at the YI session were: "In vivo multiplexed optical imaging with radiation luminescence excited quantum dots," also presented by Liu; "Mesenchymal stem cell therapy for peripheral arterial disease: In vivo monitoring and tracking with noninvasive imaging" by Yingli Fu, PhD, from the Johns Hopkins School of Medicine (Baltimore, MD); "Near-infrared fluorescent imaging of prostate cancer using integrin $\alpha_2\beta_1$ targeted peptide probes" by Chiun-wei Huang, from the University of Southern California (Los Angeles); and "Development of an activatable fluorescent probe for specific imaging of MT1-MMP expression in tumors" by Takashi Temma, from the Graduate School of Pharmaceutical Sciences, Kyoto University (Japan).

Outgoing MICoE President Henry VanBrocklin, PhD, and incoming President Carolyn J. Anderson, PhD, present Ambros Beer, MD, with the second place award.

Congratulations to these young investigators for their innovative work.

Next year all young researchers will have the opportunity to request that their abstract be considered for the Molecular Imaging Young Investigator Awards.

Craig Levin, PhD
Chair, MICoE Grants and Awards Task Force

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Clinical applications in neurodegenerative disease are set to expand substantially—through better use of ^{18}F-FDG, through ^{18}F-amyloid ligands that I expect to be in clinical practice within 2 y, and through DAT ligands for SPECT that should be available very soon, with VMAT ligands for PET to follow.

I believe our field will play an evolving role in brain tumor management. Although the future is bright, clinical acceptance and routine utilization will depend on comparative effectiveness research. This has not yet been fully embraced by the molecular imaging community, but I am hopeful that in coming years, we will begin to see the results of this sort of research moving discovery into clinical practice.

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