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BRAIN IMAGING

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The *AJNR* has been at the forefront of the imaging revolution that has taken place over the past 20 years. It has served as one of the primary sources for data critical to the progress of neuroimaging. I have chosen to categorize brain imaging arbitrarily into several sections and summarize some of the advances specifically related to our journal. It has been an amazing 20 years!

Techniques

It was clear quite early in its course that MR imaging could have a profound effect on the depiction of blood flow (1, 2). Indeed, Alvarez et al (3) in 1986 first described the MR imaging appearance of the cessation of blood flow in the internal carotid artery. Among the many comparison studies reported was that of Litt et al (4), who compared conventional angiography with 2D time-of flight (TOF) angiography. They observed good agreement between the results of MR angiography (MRA) and the standard of reference, conventional angiography. These data began the implementation of what is now the commonly accepted use of MRA as a primary diagnostic tool for extracranial occlusive vascular disease.

Ross et al (5) in 1990 compared intracranial MRA with intra-arterial digital subtraction angiography (DSA) for the evaluation of intracranial aneurysms. They concluded that MRA could reveal intracranial aneurysms as small as 3–4 mm and was a promising method for noninvasive screening of patients at risk for aneurysms.

Lewin and Laub (6) performed comparison studies of TOF techniques for intracranial MRA and suggested tailoring intracranial MRA for maximal diagnostic value. They also provided an analysis of the difficulties associated with these techniques, highlighting problems with slow flow on 3D TOF studies. They confirmed what Ruggieri et al (7) reported; ie, to maximize flow-related enhancement, the imaging volume should be perpendicularly oriented to inflowing blood, with an optimized flip angle and TR. Additionally, to reduce signal loss from flow-induced phase cancellation, constant-velocity flow-compensation gradients, combined with the shortest TE, could minimize intravoxel phase dispersion. Thin sections with small voxels reduced signal loss from phase shifts by limiting the effective range-of-motion–induced phase shifts across the small voxel.

One of the first articles that reported the use of fluid-attenuated inversion recovery (FLAIR) appeared in the *AJNR* in 1992 (8). Harbingers of its current use, the images clearly showed the utility of the pulse sequence. In 1986, LeBihan (9) showed that MR pulse sequences could be sensitized to perfusion and diffusion. One of the earliest and most exciting articles employing diffusion appeared in the *AJNR* in 1990. In it, Moseley et al (10), using an animal model of stroke, reported that diffusion-weighted imaging was more sensitive to early stroke than was conventional T2-weighted imaging. Chien et al (11) extended these observations to patients with stroke and indicated that diffusion imaging may improve tissue specificity and enable differentiation between various types of tissue damage. Tsuruda et al (12) in 1990 implemented the technique to distinguish arachnoid cysts from epidermoid tumors. In commentaries, Henkelman (13) in 1990 noted that diffusion-weighted MR imaging was here to stay, and Fisher and Sotak in 1992 (14) concluded a new era for the very early evaluation of ischemic cerebrovascular disorders appeared to be dawning. These reports correctly predicted the widespread use of diffusion-weighted imaging as an important diagnostic tool.

Functional MR (fMR) imaging represents an important technique for enhancing our understanding of how the brain works. Yetkin et al (15) reported on the application of fMR imaging for noninvasively determining the proximity of eloquent brain to focal lesions. Roberts and Rowley (16) argued that a combination of magnetic source and fMR imaging provided the best results for mapping the sensorimotor cortex. Hedera et al (17) and Lee et al in 1998 (18) reported unexpectedly negative results, failing to identify activated visual cortex and motor cortex, respectively. An editorial by Bryan and Kraut recognized the importance of these negative results, admonishing us to ''be skeptical of what [we] see and make nothing of what [we] don't see'' (19).

It was clear rather early that contrast agents would have a major role in the evaluation of brain diseases. Claussen et al (20) in 1985 demonstrated that tumor tissue could be differentiated from perifocal edema with the use of noncontrast T2-weighted imaging and that gadolinium would increase the potential of MR imaging. Graif et al (21) in the same year reported that comparison of MR with CT showed a greater degree of contrast enhancement on MR images in a group of patients with malignant cerebral tumors.

The technique for determining CBF by xenon-enhanced CT, originally reported by Meyer et al in 1981 (22), has been subsequently discussed and evaluated in a number of articles published in the *AJNR* that sought to improve and validate the technique (23–25).

In 1992, Aoki et al (26) suggested that 3D CT angiography (CTA) could be performed routinely and was helpful for surgical planning by revealing the anatomy of cerebral aneurysms and sur-

rounding structures. Evaluation of CTA in the journal clearly aided in improving the methodology (27, 28).

Stroke and Hemorrhage

The diagnosis of acute stroke prior to MR imaging was based on the clinical presentation in concert with a CT scan that excluded other pathology. Using an acute stroke model, Brant-Zawadzki et al (29) in 1986 reported the potential for increased sensitivity with MR imaging. Bryan et al (30) reported that MR imaging had increased sensitivity for the early detection (within 24 hours) of cerebral infarction and the better definition of the extent of the infarct compared with CT. Yuh et al (31) in the same issue of the *AJNR* argued that signal changes may not be reliable within the first 8 hours and observed that vascular abnormalities, when present, were the most reliable and earliest findings. They also theorized that morphologic changes and early parenchymal enhancement preceded signal changes. They indicated that paramagnetic contrast medium might provide additional data for detection and evaluation of acute ischemia. This confirmed the reports of Elster and Moody (32, 33) identifying arterial enhancement. Crain et al (34) embellished the notion of enhanced MR imaging of cerebral ischemia, reporting a variety of enhancement patterns in the acute phase, which they concluded reflected underlying pathophysiology and could have prognostic significance.

Moody et al (35) found focal abnormalities in terminal arterioles and capillaries among patients and dogs who had undergone cardiopulmonary bypass. They proposed that these findings could be the anatomic correlate of the neurologic deficits or intellectual dysfunction seen in at least 24% of patients after cardiopulmonary bypass. With alkaline phosphatase staining, they found acellular fatty material in the microvasculature of patients who died shortly after cardiopulmonary bypass (36). Steinberg et al (37), using MR and cerebrospinal fluid enzymes, confirmed that brain damage can result from cardiopulmonary bypass. In a commentary, Moody (38) suggested that the heart surgery performed at Stanford, under hypothermia with low blood flow and continuous systemic blood pressure in the 50- to 70-mm Hg range, may decrease the microembolic injury to the brain.

In an elegant scientific investigation, Moody et al (33) in 1990 studied vascular anatomy, employing a refined histochemical technique for alkaline phosphatase that labeled the microvasculature and preserved the background neuropil. They found six different patterns of intraparenchymal afferent blood supply to the brain, and suggested that some of these patterns offered protection to certain brain regions, while leaving others vulnerable in cases of anoxia or hypoperfusion. The work provided an explanation of variations in the brain's response to such injury.

The term *leukoaraiosis* was first used by Hachinski et al (39) in relation to periventricular white matter hypodensities. Braffman et al correlated MR findings of brain specimens with gross and microscopic histopathology in an attempt to provide MR criteria to differentiate lacunar infarction from Virchow-Robin spaces (40) and elucidate the etiology of white matter hyperintensity (41). Hendrie et al (42) noted the correlation between foci of increased intensity on T2-weighted images and aging unrelated to cognitive function or cerebrovascular risk factors. Using xenon CT, Kobari et al (43) in 1990 measured local blood flow in a variety of patient groups and concluded a correlation existed between diffuse cerebral hypoperfusion, cognitive impairment, and leukoaraiosis. Bryan et al (44) in a large patient cohort found that the prevalence of subclinical cerebrovascular disease reflected by MR imaging was of a magnitude greater than clinically suspected, and that disease increased with age and was more prevalent among blacks.

The term *venous congestive encephalopathy* was proposed in the *AJNR* by Willinsky et al in 1994 (45) for patients who present with neurologic deficits caused by venous hypertension. Periventricular venous collagenosis with stenosis or occlusion of deep cerebral veins has been associated with leukoaraiosis (46). Hurst et al (47) found that venous hypertensive encephalopathy secondary to dural arteriovenous fistulas could cause progressive dementia. The journal has published its share of vascular syndrome findings, including familial arteriopathic leukoencephalopathy (48), primary antiphospholipid sndrome (49), and leukoencephalopathy in cerebral amyloid angiopathy (50).

The significance of a hyperdense middle cerebral artery (MCA) on CT has been thoroughly debated in the journal (51–54). Bozzao et al (55) suggested that the appearance of hypodensity revealed by CT soon after embolism onset was strongly predictive of hemorrhagic transformation. In an *AJNR* commentary, Pessin et al (56) argued that the occurrence of hemorrhagic infarction alone does not necessarily imply a serious complication. von Kummar et al (52) expanded this outcome correlate and stated that early hypodensity on CT scans occuring with acute infarction was a predictor of ischemic brain damage. If it encompassed 50% or more of the MCA territory, then there was an 85% positive predictive value for fatal outcome.

Schwartz et al (57) used diffusion imaging to indicate that the edema of hypertensive encephalopathy (as seen in a variety of diseases, including pre-eclampsia-eclampsia, lupus nephritis, and with immunosuppressive drug therapy) has primarily vasogenic origin from lack of autoregulation. They suggested that therapy should aim to lower blood pressure and prevent hemorrhage. Nonaneurysmal perimesencephalic subarachnoid hemorrhage was first reported in 1985 by van Gijn et al (58). In 1991, CT and MR patterns differentiating this entity from aneurysmal rupture was published in the

AJNR (59) and correlated with an excellent prognosis.

The complex MR appearance of hemorrhage generated a great deal of discussion and controversy since the initial publication of its imaging characteristics at 1.5 Tesla (60) and the *AJNR* has been an active forum for much of this discussion. Edelman et al (61) employed gradient-echo imaging to improve detection of susceptibility effects and sensitivity to hemorrhage. Zimmerman et al (62) in 1988 illustrated the imaging appearance of acute hemorrhage at 0.5 Tesla. The role of magnetic susceptibility in the appearance of hypointensity seen on T2-weighted images was corroborated by a histologic biochemical study in rats (63) and in vivo magnetization transfer (MT) measurements (64). Hayman et al (65) suggested the role of hemoglobin immobilization by clot structure or red cell contraction in acute hematoma and disputed the results of in vitro MT and relaxation rate measurements (60). Janick et al (66) refined the understanding of how various oxidation states of intracellular and extracellular hemoglobin and protein concentration contribute to the MR appearance of hemorrhage. Boyko et al (67) described T1 shortening from processes unrelated to hemorrhage (calcification and laminar necrosis).

Infection and Inflammation

Braun et al (68) recognized that finding a ring pattern on unenhanced CT scans could increase specificity of the enhanced images, particularly related to structural lesions such as brain abscess. MR features of pyogenic abscesses were reported in 1989 and included edema, central necrosis, extraparenchymal spread, and peripheral high intensity on T1-weighted images (69). MR imaging was reported to be superior to CT for detecting the full extent of inflammation and assessing the resolution of abscess. The journal reported the difficulties with CT in the diagnosis of subdural empyema (70), but by 1989 the diagnosis could be made early and accurately with MR imaging (71).

By the early 1980s, it was known that HIV was neurotrophic (72). The *AJNR* and other radiologic journals (73–77) reported a plethora of imaging findings in AIDS patients. Flowers et al (78) determined that MR imaging was valuable for evaluating encephalopathy in AIDS patients. Cohen et al (79) in 1992 noted that MR findings are normal to minimally abnormal during early stages of HIV infection, subtle neuropsychologic abnormalities do not correlate with MR, and there may be a prominence of adenoidal tissue in patients without opportunistic infections.

Wehn et al (80) in 1989 first observed dilated perivascular spaces in cryptococcal infections, Tien et al (81) and Mathews et al (82) extended the MR observations regarding this AIDS-related infection, including its lack of enhancement and underestimation of the extent of disease. Coccidioidomycosis was revealed to show widespread cisternal and cervical meningeal enhancement and ventricular enlargement (83). HIV patients were particularly susceptible to brain abscess.

MT ratio (MTR) measurements proved useful for distinguishing progressive multifocal leukoencephalopathy (PML) from HIV encephalitis (84). PML had lower MTR values—much lower than regions involved with HIV encephalitis. Thallium 201 brain single-photon-emission CT (SPECT) could be employed in the imaging of AIDS patients to differentiate CNS lymphoma from infectious lesions, such as toxoplasma encephalitis (85). Increased intense uptake was associated with lymphoma, whereas toxoplasma encephalitis had no uptake. Kim et al (86) suggested that T1-weighted imaging revealed that tuberculoma had hyperintense and hypointense rims that corresponded to collagenous fibers and inflammatory infiltrate, respectively. The *AJNR* featured imaging findings of many other infectious and inflammatory diseases, including Wegener granulomatosis (87), Lyme disease (88, 89), chronic fatigue syndrome (90), neurosyphilis (91, 92) aspergillosis (93), Whipple's disease (94), St. Louis encephalitis (95), Japanese encephalitis (96), malaria (97) and Rocky Mountain spotted fever (98).

White Matter Disease

Horowitz et al (99) reported that the ovoid lesion on MR images might increase specificity for the diagnosis of MS and that this lesion was the MR correlate of ''Dawson's finger.'' MS lesions could have a diverse appearance, including rings on MR images that are high-intensity with T1-weighted imaging and low-intensity with T2-weighted imaging (100). T2 shortening was reported to be present in MS patients in the thalamus, putamen (101), cortex, and adjacent subcortical white matter (102).

Two investigations reported contrast enhancement decreases over disease duration that reflect clinical classification (103, 104). Grossman et al (105) suggested that proton spectroscopy could lead to better categorization of MS lesions than contrast enhancement could and that demyelination had a longer course than contrast enhancement. Guttmann et al (106) characterized the temporal evolution of MS lesions.

In an animal model of Wallerian degeneration, Lexa et al (107) proved that application of the MTR measure was more sensitive for the early detection of degeneration than was conventional MR imaging and that temporal changes revealed by the MTR corresponded to histologic phases of Wallerian degeneration. The MTR was suggested to be a robust measure (108) and reliable method for determining MS lesion age (109). Hiehle et al (110) drew attention to T1 hypointense lesions and their low MTRs and concluded that, based on MTR data, T1 hypointense lesions represented the most demyelinated MS lesions. Loevner et al (111) confirmed this observation and suggested that T1-weighted imaging might be useful for characterizing MS le-

sions. van Buchem et al (112) combined a computer software program and MT to produce global MTR histograms for estimating whole-brain disease burden in MS. Phillips et al (113) reported that the MTR histogram was a better indicator of global disease burden than T2 was of lesion volume.

Metabolic and Toxic States

Xiong et al (114) reported the MR findings in toluene abuse, including loss of gray-white matter, periventricular white matter abnormality, decreased size of the corpus callosum, and hypointensity intensity in the thalami. Methanol can produce putaminal necrosis and hemorrhage as well as peripheral white matter lesions (115) and other regions involved in severe intoxication include the caudate nucleus, pontine tegmentum, and optic nerves (116). Ethylene glycol toxicity affects the thalamus and pons (117). MR imaging reveals that organic mercury poisoning (Minamata disease) affects the calcarine area, cerebellum, and postcentral gyri, and these regions are responsible for the characteristic manifestation of the disease, including constriction of the visual fields, ataxia, and sensory disturbance (118). Cyclosporin A produced reversible changes in the occipital region (119).

Degenerative Diseases

In 1986, George et al (120) showed that leukoencephalopathy was linked to the aging process and was observed in both ''normal'' and cognitively impaired individuals who had no other evidence of vascular disease. De Leon et al (121), in a 3-year longitundinal study of patients with Alzheimer's disease, found that changes in ventricular size reflected clinical changes. Holodny et al (122) reported that dilatation of the perihippocampal fissures could be a sensitive and specific marker for distinguishing Alzheimer's disease from normal-pressure hydrocephalus. Bradley et al (123) called attention to the relationship between CBF and CSF circulation and to the association of ischemic periventricular lesions in patients with normal-pressure hydrocephalus and in elderly patients with communicating hydrocephalus. As would be expected, the results of these fresh ideas were debated in the journal (124, 125).

The MR imaging appearance of acute lesions in the Wernicke-Korsakoff syndrome, including reversible involvement of the dorsal medial thalamic nuclei and periaqueductal region (126, 127), were first described in the journal. Kato et al (128) used MT measurements to detect pyramidal tract lesions in amyotrophic lateral sclerosis.

The imaging findings in chronic acquired hepatic failure (increased signal intensity in the basal ganglia, pituitary gland, and mesencephalon surrounding the red nuclei on T1-weighted images) were characterized by Brunberg et al (129). We have even seen the ''eye-of-the-tiger'' in Hallervorden-Spatz disease (central hypointensity within a hyperintense rim surrounded by hypointensity on T2-weighted images in the globus pallidus [130]).

Trauma

Several investigators (131–133) reported imaging characteristics of extracerebral collections secondary to traumatic brain injury. McCluney et al (134) called attention to the position of the cortical veins in differentiating subdural hygroma from atrophy. In a blinded comparison of CT and MR imaging, Orrison et al (135) argued that CT and MR were complementary in the evaluation of acute head trauma. Gentry et al (136) reported that corpus callosal injuries were more frequent than expected, with an associated high incidence of diffuse axonal injury. Mittl et al (64), using T2*-weighted imaging, found evidence of diffuse axonal injury in some patients with mild head injuries in whom CT findings were normal. They suggested that these lesions might be responsible for some aspects of the postconcussive syndrome. Blatter et al (137) performed brain volumetric quantitation in traumatic brain injury, and suggested that these measures might be predictive of cognitive outcome. Bigler et al (138) focused on hippocampal and temporal horn volume and reported that, in the subacute phase after brain trauma, the volume of the temporal horn might correlate with intellectual outcome and that of the hippocampus with verbal memory function.

Pituitary Region

A number of investigators studied the pituitary with CT and MR imaging and described its normal appearance (139, 140), the changes in adolescents and preadolescents (141), and the utility of contrast-enhanced MR imaging for localizing microadenomas (142–145). This small region has had more than its share of reports and controversy. Mark et al (146) in 1984 suggested that high intensity in the posterior sella as shown on T1-weighted images might represent fat. Fujisawa et al (147–149) theorized that the signal intensity was related to the functional status of the hypothalamoneurohypophyseal axis and the signal was secondary to neurosecretory granules. Using phospholipid vesicles, Kucharczyk et al (150) in an elegant experiment modeled the signal intensity and concluded that the MR imaging characteristics could be explained by the phospholipid vessicles. Using fat suppression, Mark et al (146) suggested that the high-intensity signal could have more than one source.

Tien et al (151) proposed that MR imaging could reveal central diabetes insipidus. Moses et al (152) concluded that T1-weighted MR imaging might be able to reveal the difference between central diabetes insipidus (absent posterior pituitary bright spot) and primary polydipsia (bright spot present). Lundin et al (153) reported serial changes in macroprolactinomas resulting from long-term bromo-

criptine therapy, including increasing T2 values over time.

Dynamic MR imaging showed abnormalities of the hypophysial vasculature in lymphocytic hypophysitis (154). Using classical radiologic-pathologic correlation, Sartoretti-Schefer et al (155) were able to separate adamantinous MR findings from squamous-papillary craniopharyngiomas, and Masayuki et al (156) provided MR imaging criteria for the diagnosis of Rathke cleft cysts.

Neoplasms

The detection of metastatic disease has received considerable attention in the journal. In 1990, Sze et al (157) recommended contrast enhancement for the detection of brain metastasis. In 1992, Yuh et al (158) suggested high-dose (0.3 mmol/kg) gadolinium for detection of early or small metastases. Higher contrast doses were judged to be better than delayed imaging with standard contrast doses (159). Sze et al (160) provided confirmation of the beneficial effects of triple dose in cases of equivocal findings or solitary metastasis. In a commentary in the *AJNR*, Ginsberg and Lang (161) argued for postcontrast MT imaging rather than triple-dose gadolinium. Elster and Chen in 1992 (162) concluded that nonenhancing white matter abnormalities have a low probability of representing metastatic disease.

It would be too difficult to enumerate all of the articles on particular brain neoplasms. Suffice to say that the *AJNR* literature runs the spectrum from investigations of astrocytomas (163) to xanthoastrocytomas (164). Proton MR spectroscopy was deemed a reliable technique for grading gliomas when combinations of metabolites were statistically compared (165).

Complications of radiation injury to the brain and the differentiation from recurrent or residual tumor are important clinical issues. The journal published an excellent review of this subject in 1991 (166, 167). Schwartz et al (168) suggested that dual-isotope SPECT with ^{201}TI and ^{99m}Tc-HMPAO may be useful for differentiating sites of tumor from radiation changes in patients treated for malignant glioma. In a provocative paper, Ricci et al (169) suggested that flurodeoxyglucose (FDG) positron emission tomography (PET) may not be as useful as previously indicated (170) for differentiating recurrent tumor from radiation necrosis.

Neuropsychiatry

The ability of MR imaging to define structural abnormalities may elucidate behavioral abnormalities. Andreasen in an *AJNR* commentary stressed a new alliance between neuropsychiatry and neuroradiology (171). Degreef et al (172) detected an increased prevalence of a cavum septum pellucidum, cavum vergae, and partial callosal agenesis in schizophrenics, suggesting that these might be an important substrate in this disorder. Seidenwurm et al (173) studied subjects with extremely violent behavior with FDG PET and found decreased temporal lobe metabolism was correlated with limbic abnormalities seen at electrophysiologic and neuropsychiatric evaluation. Using MR spectroscopy, Gonzalez et al (174) quantified brain lithium. They found variability in brain versus serum levels among patients with bipolar disease. Using fMR imaging, Sunshine et al (175) studied patients with attention deficit disorder. In this preliminary study, they detected additional areas of activity in this patient cohort.

Epilepsy

Imaging findings in postictal patients include transient cerebral swelling and enhancement (167, 176). In a cohort of patients with histologically verified mesial temporal sclerosis, Meiners et al (177) found increased hippocampal signal intensity and decreased gray-white demarcation in the temporal lobe to be the most specific MR imaging features of this lesion. Oppenheim et al (178) observed that complete loss of digitations in the hippocampal head was a sensitive and specific marker of mesial temporal sclerosis. Cheon et al (179) reported that visual assessment was slightly superior to MR volumetry. Using pathologic confirmation as a standard of reference in a large patient cohort with temporal lobe epilepsy, Lee et al (180) found that MR imaging yielded 93% sensitivity and 83% specificity in detecting hippocampal/amygdalar abnormalities. The presence of hippocampal atrophy correlated with the duration of seizures.

The Future

Neuroradiology has been key in advancing our understanding of the pathophysiology of disease and improving the sensitivity to its detection and extent as well as the specificity to particular pathogens. I am struck by how far we have come in the past 20 years in elucidating neurologic diseases. There is, however, still a long road ahead!

MR imaging has become the primary technique for probing the brain and will continue to have that role in the future. Molecular imaging with various MR, SPECT, or PET probes may further our understanding of brain metabolism and function. There is no question that we will have a more profound understanding of how the brain works as functional imaging becomes more refined.

We will clearly move to faster imaging techniques, stronger gradients, and higher field-strength magnets. Resolution will improve, and we may eventually evolve to image true in vivo pathology. High-resolution spectroscopy will be commonplace and that biochemical window will be invaluable as a predictor of outcome. We will use it to assess the effect of disease on the entire

brain. Quantitative methods will readily enable assessment of tumor or disease burden and treatment outcome. These methods will make MR imaging a primary outcome measure in neurologic disease.

It is critical that neuroradiology be substantively involved in the continued development of imaging science. Unless neuroradiology commits itself to performing significant scientific investigation, the specialty could be dismantled. Without scientific progress, there will be no specialty. Other specialties have stronger commitments to basic science and training programs are designed in part to preparing physician scientists. We need to see this as a challenge to our fundamental existence and support all aspects of the science of neuroimaging.

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