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Is a Picture Worth a Thousand Words? Neuroimaging in the Courtroom

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I. INTRODUCTION

Neuroimaging has advanced our understanding of how the living brain operates, providing structural and functional images of both healthy and diseased brains. This technology pervades today's society, particularly affecting the legal arena. Some judges argue that scientific evidence, which offers insight into the offender's mental state, is crucial because it is the only means of determining whether an offender's punishment is proportional to his crime.[1] Other judges argue that "objective" evidence does not "wholly determine the controversy," and focus instead on their duty as gatekeepers to independently evaluate scientific evidence. If courts use brain images to make their culpability determinations more objective and sound, these images must meet pertinent legal standards and shed light on medical conditions. For neuroimaging to meet these legal and medical standards with scientific integrity, scientists must convincingly correlate the dynamic images in a person's brain with the way the person is thinking or acting at that moment.

With respect to understanding the relationship between the brain and certain behaviors, the state of scientific knowledge is nascent, but promising. The more complex and specific the behavior examined, the more speculative the connection. For example, violence is a multifactorial and socially driven behavior that is not likely reducible to a unitary brain function or region. At present, there is no specific area of the brain to which aggression and violence are singularly ascribed. Brower and Price stated, "The evaluation of research on the neurobiology of violence demands conceptual clarity, along with careful analysis of methods and data to prevent misunderstanding and

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[1] Stanford v. Kentucky 1989, 492 U.S. 361, 402-404 (1989) (discussing the proportionality of punishment).

possible abuse of the results."[2] Currently, attorneys rely on psychologists, psychiatrists, and neurologists to offer integrated expert testimony regarding illness and behavior. This testimony varies among experts. While "scientific" brain imaging appears to offer greater objectivity,[3] current brain imaging techniques, by themselves, may be no more objective than the modalities that came before them.

Some of the problems associated with using new medical technology in the legal arena stem from the different modes of inquiry each discipline uses. Scientists begin by asking questions, collecting as complete a set of relevant information as possible, experimenting with variables, applying statistical odds, and only then drawing conclusions. Because the state of scientific knowledge changes rapidly, sometimes there are multiple contradictory scientific views of a given issue, all of which are potentially credible. These controversies help fuel scientific progress.

Lawyers, unlike scientists, are advocates, and therefore operate within a different paradigm. They gather facts to support a particular theory, attempting to reconcile facts contradictory to that theory in their client's favor. The law counts on legal adversaries to present contrary theories and thereby allow the truth to emerge.

With these cautions in mind, we can still describe the contribution brain imaging may make on certain neurologic illnesses and in the presence of other neurologic data. This Article will review some of the current research on the attempt to use functional imaging to construct a neuropathologic diagnosis for certain behaviors. This article will present a continuum, ranging from situations in which neuroimaging may contribute to the explanation of aggressive or criminal behavior, to situations in which neuroimaging is currently too speculative to provide a solid footing.

The goal of this Article is to elucidate the role of neuroimaging in the courtroom from a neurologic perspective. This article begins with a brief commentary on historical uses of neurologic evidence in criminal cases and early scientific attempts to create a "science" of criminality. What follows will be a review of the brain anatomy that is relevant to these neuroimages and a simple explanation of the imaging techniques involved. The remainder and bulk of this Article will present instances in which the imaging of certain brain insults may contribute to the understanding of criminal or violent activity. The conditions selected for this section on "solid ground" are a representative sample of such injuries or illnesses, but are not an exclusive list of these conditions. Finally, this Article will discuss emerging (and as yet theoretical) attempts to use functional imaging to identify brain substrates for specific undesirable traits or activities.

II. HISTORICAL SEARCH FOR THE NEUROANATOMY OF BEHAVIOR

While the brain has some role in producing violent behavior, it is not clear whether there is or ever will be a linear, identifiable relationship between the

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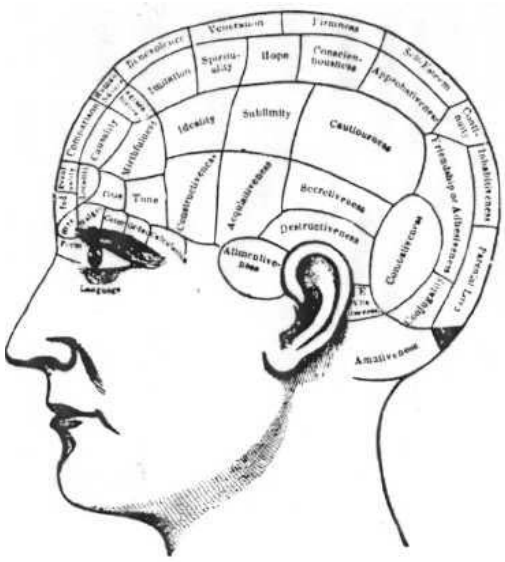
[2] Montgomery C. Brower & Bruce H. Price, *Epilepsy and Violence: When is the Brain to Blame?*, 1 EPILEPSY & BEHAV. 145, 146 (2000).

[3] Richard Restak, *See No Evil. The Neurological Defense Would Blame Violence on the Damaged Brain*, SCIENCES July-Aug. 1992, at 16, 16-21.

two. Early religious explanations characterized behavior as the product of spirits, gods, demons, or the devil.[4] These explanations occurred in the context of religious tenets that defined morality and formed a latticework for societal behavioral norms. It was widely accepted that mind and body were a duality, thereby relegating explanations of behavior to an ethereal realm. As recently as a century ago people believed the brain was an organ that, given its gray and unimpressive appearance, could not possibly be responsible for sublime human actions.[5]

These concepts underwent drastic revision both during and after the Enlightenment, and with the decriminalization of anatomical dissection.[6] For instance, Franz Joseph Gall, a physician in the early nineteenth century, challenged the duality of mind and body with his assertion that the brain was the organ of the spirit.[7] Gall did careful and laborious examinations and dissections of the brain and skull, and extrapolated his theories regarding neurologic function from his postmortem findings.[8] He developed a topographical model of the brain based on this research, and asserted that the brain was divided into separate components, each performing a distinct function (see Figure 1).[9]

Figure 1.



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[4] Dines Bhugra, *Religion and Mental Health*, *in* Psychiatry and Religion: Context, Consensus, and Controversies 1-3 (1996).

[5] *See* Julie M. Fenster, Mavericks, Miracles, and Medicine 190 (2003).

[6] *Id.*

[7] *Id.* at 192.

[8] *Id.* at 190.

[9] *Id.* at 192.

[10] Phrenology.com, Phrenological Map, http://www.phrenology.com/phrenology.jpg (last visited July 2, 2007).

Gall believed that the brain's components and their functions were reflected in raised areas on the human skull and that one could ascertain brain function by examining these bumps.[11] He posited that these raised areas would reveal how and where an individual's brainpower was allocated; the greater the skull space dedicated to a particular part of the brain, the greater its faculty.[12] Gall extended his study of the skull to the features of the human face and asserted that these features revealed an individual's attributes.[13] Gall's brain studies were an attempt to classify a relatively unknown organ. He was among the first to associate brain anatomy with brain function, and although phrenology eventually became discredited, Gall remained committed to his classification of the human brain and the groundbreaking notion that science could link areas of the brain with the tasks they perform.[14]

Nineteenth-century Italian physician Cesare Lombroso, known as the father of modern criminology, later used phrenology to spearhead a major shift in criminal investigation.[15] In 1872, while performing an autopsy on a notorious criminal, Lombroso was struck with inspiration: "I seemed to see all at once, standing out clearly illumined as in a vast plain under a flaming sky, the problem of the nature of the criminal, who reproduces in civilized times characteristics, not only of primitive savages, but of still lower types as far back as the carnivores."[16] This theory became known as Atavism, or the re-emergence of regressed evolutionary traits in modern criminals.[17] It was a radical departure from the thinking of the time and drew heavily from both the work of Charles Darwin and evolutionary theory.[18] Lombroso believed that some individuals were "born" criminals; they were throwbacks to an earlier evolutionary stage, and these individuals could be identified by studying their physical features (see Figure 2).[19]

Lombroso differentiated these "born" criminals from those criminals who committed crimes in a state of passion and believed that the latter could be rehabilitated.[20] However, those with the indelible marks of the born criminal were beyond such help, he posited, and should be adjudicated based on their features rather than their crimes.[21] Lombroso used the "scientific" foundation of his work to marginalize earlier enlightened theories of punishment as "abstract, unscientific, and out of touch with the facts."[22] Lombroso's theories were widely accepted and became the basis for much of Italian law at that time.[23]

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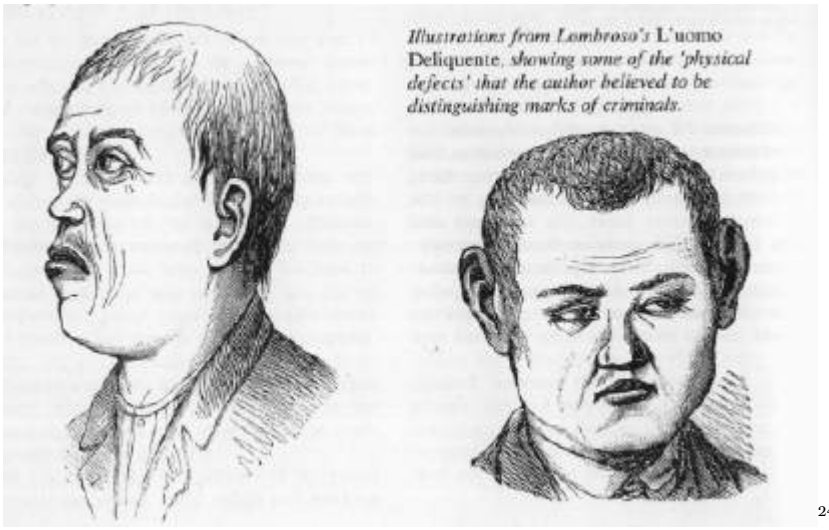
[11] Fenster, *supra* note 7, at 192.
[12] *Id.*
[13] *Id.* at 191-192.
[14] *Id.* at 197.
[15] Mary Gibson, Born To Crime: Cesare Lombroso and the Origins of Biological Criminology, 19 (2002).
[16] *Id.* at 20.
[17] *Id.* at 11.
[18] Stephen Jay Gould, The Mismeasure of Man, 124 (1st ed. 1981).
[19] Gibson, *supra* note 15, at 23.
[20] *Id.*
[21] *Id.* at 27.
[22] *See id.* at 26.
[23] *See id.* at 210-241.

Figure 2.



[24]

As scientific understanding of the human brain progressed, scientists rejected the mind/body duality of the past and instead studied the brain in an effort to understand precisely how this mass of nerve cells produced thought, emotion, and action. Before physicians could image or observe the brain in action, they inferred functional anatomy from observing behavioral changes caused by observable trauma to the brain. The famous index case for this type of injury was that of Phineas Gage.[25] In 1848, Gage, a construction crew foreman, was laying railroad tracks using a three-foot spike to tamp down dynamite.[26] A premature explosion propelled the spike into his face, through his brain, and out the top of his head (see Figure 3).[27] Miraculously he survived, but with a distinct change in his personality. Whereas prior to the accident he was efficient and industrious, afterwards he was behaviorally different.[28] Dr. John Harlow, Gage's personal physician, wrote an extensive account of these changes. Though Gage showed no deficits in speech, movement, or language, he was:

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[24] Crimeculture.com, Victorian Detective Fiction, http://www.crimeculture.com/Contents/VictorianCrime.html (last visited July 2, 2007) (reprinted with permission).

[25] Antonio R. Damasio, Descartes' Error 3 (1994); Hanna Damasio et al., *The Return of Phineas Gage: Clues About the Brain from the Skull of a Famous Patient*, 264 Sciences 1102, 1102-1105 (1994).

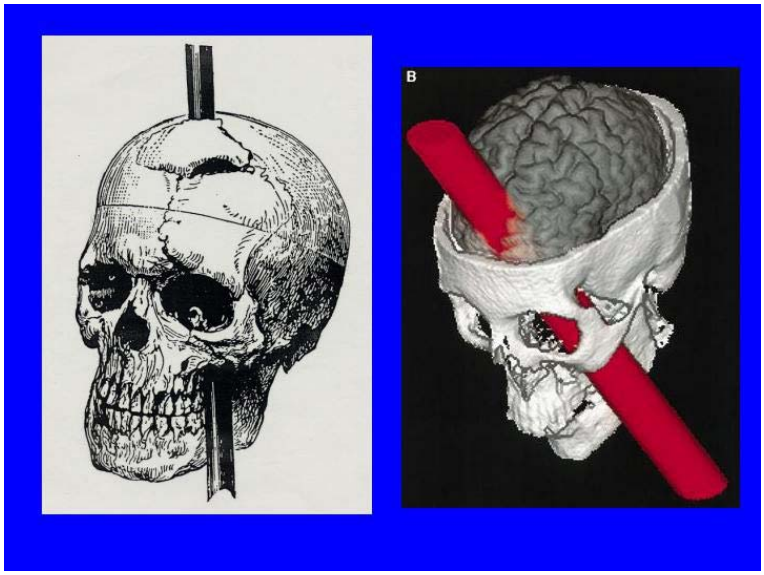
[26] Hanna Damasio et al., *supra* note 25, at 1102.

[27] *Id.*

[28] *Id.* at 1102.

> fitful, irreverent, indulging at times in the grossest profanity which was not previously his custom, manifesting but little deference for his fellows, impatient of restraint or advice when it conflicts with his desires, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operation, which are no sooner arranged than they are abandoned . . . . A child in his intellectual capacity and manifestations, he has the animal passions of a strong man.[29]

Figure 3.



[30]

The case of Phineas Gage established that the brain and personality are intimately related, and that damage to the human frontal cortex can cause a dramatic change in behavior, but not cognition. As his family and peers observed, "Phineas Gage is no longer Phineas Gage." The case of Phineas Gage began the study of localizing the part of the brain that houses our "humanity."

One hundred and fifty years elapsed between Phineas Gage's elucidating accident and the first use of brain imaging in the courtroom. In 1981, John Hinckley attempted to assassinate President Ronald Reagan. His defense team requested permission to introduce CAT scan images of his brain as part of an effort to establish the presence of mental illness.[31] The judge allowed the jury to see Hinckley's CAT scan, which suggested abnormal brain shrinkage.[32]

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[29] ANTONIA DAMASIO, *supra* note 25, at 8.

[30] *Id.* at 1104 (reprinted with permission).

[31] Jennifer Kulynych, *Psychiatric Neuroimaging Evidence: A high tech crystal ball?* , 49 STAN. L. REV. 1249, 1252 (1997).

[32] *See id.*

Thus began the link between brain imaging and the explanation of criminal behavior that is the subject of this Article.

III. RELEVANT NEUROANATOMY

A brief overview of the relevant neuroanatomy will help clarify useful substrates for brain imaging. The Central Nervous System (CNS) is responsible for coordinating a body's interaction with the outside world. It receives sensory input, organizes information, and directs responses. The CNS comprises the brain and the spinal cord. The primary cellular building block of the CNS is the neuron. A neuron is a cell that receives chemical communication and converts it into an electrical impulse. That impulse then travels down the length of the neuron to be re-converted into a chemical message that is delivered to the next neuron in the chain. In order to help preserve the electrical impulse that travels down the neuron, the neuron is covered in a sheath called myelin that performs a function similar to the insulation around telephone and cable wires. Much of what we know about function of the brain comes from either animal studies (ablating areas of the rodent or primate brain) or human lesions (either tumors or injuries).

The brain can be rudimentarily divided into the brainstem (responsible for basic functions of life, e.g. respiration), the cerebellum (which primarily coordinates movement), the limbic system, and the cerebrum, which involves both cortical and subcortical components (see Figure 4).[33] The cerebellum, cerebrum, and limbic systems are lateralized, with left and right sides. The two hemispheres of the cerebrum are connected by a dense bridge known as the corpus callosum. The two areas of greatest interest for our purposes are the limbic region and the cerebral cortex, which have been the subject of many advances in understanding the mechanisms of behavior.[34] The limbic region is among the oldest and most primitive evolutionary part of the CNS (see Figure 5).

The limbic region is intimately involved with regulation of emotion, memory, motivation, and autonomic and endocrine function. It is a source for integrating primary sensations, emotions, and memories, and in this way has been implicated in evolutionary survival functions.[35] While there is debate about what comprises the limbic brain, this Article assumes it includes the amygdala, hypothalamus, cingulate gyrus, temporal, and prefrontal cortices.[36] The amygdala infuses emotional valence into memories. The hypothalamus receives information concerning the internal state of the body and orchestrates endocrine (hormonal) responses through its control of the pituitary gland, known as the master gland of the body. The hypothalamus plays a significant role in predatory aggression. In animal studies, destruction

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[33] *See* James D. Fix, HIGH YIELD NEUROANATOMY (1995).

[34] *See generally*, Jeffrey L. Cummings & Michael S. Mega, NEUROPSYCHIATRY AND BEHAVIORAL NEUROSCIENCE, 7-19 (2003) (summarizing the neurobiological basis of behavior).

[35] R.B. CHRONISTER & S.G.P. HARDY, *The Limbic System*, *in* FUNDAMENTAL NEUROSCIENCE, 443, 444 (Duane E. Haines, M.D., ed. 1997).

[36] *See* W.A. Weiger & D.M. Bear, *An Approach to the Neurology of Aggression*, 22 J. PSYCHIATRIC RESEARCH 85, 98 (1988).

of part of the hypothalamus can produce an appearance of "extreme anger and marked sympathetic arousal."[37]

Figure 4.

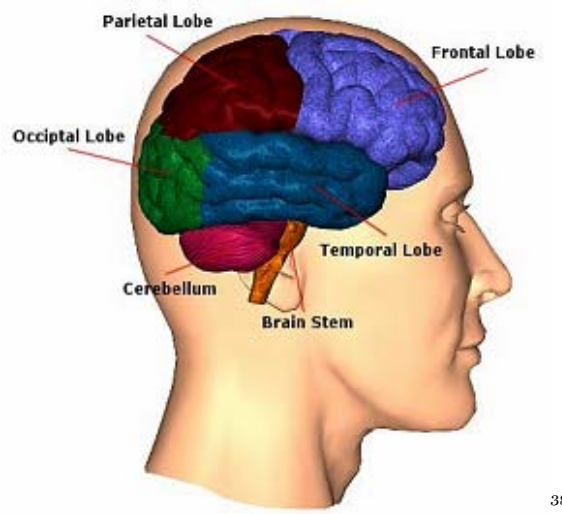

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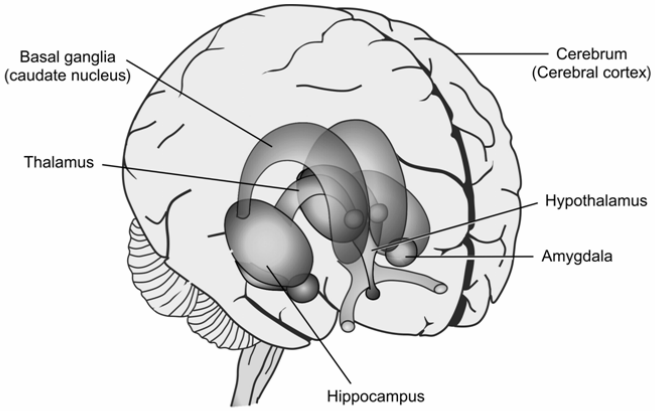
Figure 5.



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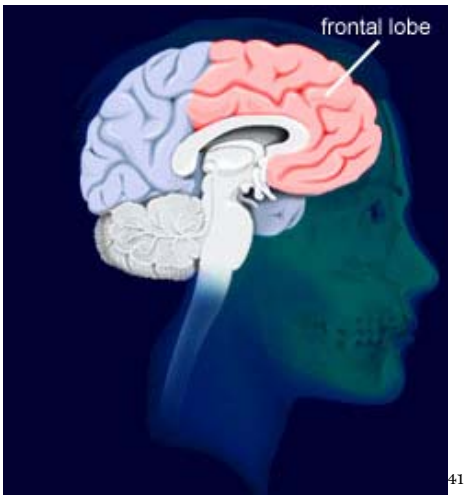
[37] David Bear, *Neurological Perspectives on Aggressive Behavior*, J. NEUROPSYCHIATRY, Spring 1991, at S3, S4.

[38] James Knupp, The Human Brain: Mysteries of the Human Brain, http://www.wright.edu/academics/honors/institute/brain/mysteries.html (last visited July 2, 2007) (reprinted with permission).

[39] This image depicts inner structures of the human brain, including the limbic system. Organisation for Economic Cooperation and Development, Understanding the Brain—

The frontal lobe is the most recently developed and highly evolved portion of the human brain. The prefrontal cortex is responsible for our ability to reason, plan, and sequence ideas (see Figure 6).[40]

Figure 6.



[41]

The prefrontal contex has been subdivided into five frontal-subcortical circuits, which subserve cognition, behavior, and movement. For our purposes, two of these circuits, the dorsolateral prefrontal circuit and the orbital frontal circuit, are the most relevant. The dorsolateral prefrontal circuit governs executive functions, including the ability to plan and maintain attention, problem solve, learn new information, retrieve memories, sequence the order of events, and adaptively change cognitive and behavioral sets. The orbital frontal circuit connects frontal monitoring functions to the limbic system. This circuit governs appropriate responses to social cues, empathy, social judgment, and interpersonal sensitivity. Dysfunction in this circuit can lead to aggression, irritability, disinhibition, and improper social behavior.[42]

IV. BRAIN IMAGING

Structural neuroimaging modalities, such as computer-assisted tomography and magnetic resonance imaging (MRI), have revolutionized the practice of medicine in recent decades. They offer an objective, non-invasive, quantifiable image, which can provide useful information particularly when

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Towards a New Learning Science, http://www.oecd.org/dataoecd/50/26/15355602.gif (last visited July 2, 2007) (reprinted with permission).

[40] *See e.g.*, Monte S. Bucksbaum, *Frontal Cortex Function*, 161 Am. J. Psychiatry 2178 (2004).

[41] Brain Explorer, Glossary, Frontal Lobe, http://www.brainexplorer.org/glossary/frontal_lobe.shtml (last visited July 2, 2007) (reprinted with permission).

[42] Bruce H. Price et al., *The Compartmental Learning Disabilities of Early Frontal Lobe Damage*, 113 Brain 1383, 1388 (1990).

the clinical examination may otherwise be normal. Functional imaging techniques were developed after the advent of structural neuroimaging and show great promise for both clinical use and neuroscience research. These modalities include positron emission tomography (PET), single photon emission computer tomography (SPECT), and functional magnetic resonance imaging (fMRI).

A. Structural Brain Imaging

Structural brain images include Computed Axial Tomography (CAT) and Magnetic Resonance Imaging (MRI). CAT scans obtain images of the brain by taking multiple x-ray images from different angles and fusing them to show a cross sectional image of the brain. The images are reconstructed using computer programs, which also allow for digital manipulation of the images and the ability to view brain structure along different planes.[43] MRI uses a powerful magnet to obtain its images, rather than a conventional x-ray. The basis of MRI is the magnetic property of the studied cells. The scanner uses its magnet to align the nuclei of the cells' atoms.[44] The strength of the magnet determines the quality of the image obtained. Because not all tissues respond to the magnetic field equally, once the magnetic field is halted, the cells return to their original pre-magnetized form at different rates.[45] In the process of their realignment to pre-magnetized states, they generate a signal that is converted into an image by computer. An image obtained using CAT scans or MRI is by definition static, a snapshot of the structure of the brain at that moment.

B. Functional Brain Imaging

While there are myriad techniques that produce real-time images of brain activity, the commonly available techniques are Functional MRI (fMRI), Positron Emission Tomography (PET), and Single Photon Emission Computerized Tomography (SPECT). fMRI uses the properties of blood oxygenation to obtain a dynamic picture of function,[46] taking advantage of the brain's need for oxygen-rich blood to perform necessary tasks. Oxygenated blood reacts to the magnet differently than deoxygenated blood, which allows for the real-time quantitative monitoring of brain metabolic activity.[47] Manipulating this contrast creates a more dynamic "picture" of the area of study because it can image metabolic activity over time. PET and SPECT scanning do not rely on the use of a magnetic field; rather they image regional blood flow, blood volume, and metabolism. They are most advantageous for studying neurochemistry. They use radiographic material that is injected into

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[43] Gordon M. Shepherd, *Information Processing in Dendrites*, *in* Fundamental Neuroscience 393 (Michael J. Zigmond et al. eds,, 1999).

[44] Martin A. Goldstein & Bruce H. Price, *Magnetic Resonance Imaging*, *in* Essentials of Neuroimaging for Clinical Practice, 21, 22 (Darin Dougherty et al. eds., American Psychiatric Publishing 2004).

[45] *See id.* at 25.

[46] *See* Robert Turner et al., *Functional Magnetic Resonance Imaging of the Human Brain: Data Acquisition and Analysis*, 123 Experimental Brain Research, 5, 6 (1998).

[47] Robert A. Novelline, Squire's Fundamentals of Radiology 602 (6th ed. 2004); Shepherd, *supra* note 43, at 393.

the subject and taken up by the brain.[48] The degradation of the radioactive material produces visible light that is converted into an image.[49] That image demonstrates blood flow while subjects perform tasks.[50]

C. Limitations: A Cautionary Note

There are limits to the "objectivity" of neuroimaging. Brain imaging is the product of a complex set of techniques, subjective decisions, technical choices, and informed interpretations. Scientists, technicians, and clinicians decide the level of detail they will use to scan the brain. They must determine what types of imaging should be ordered, how thick or thin the slices should be, the degree of clarity, the difference in contrast between types of tissue, and how the signal should be filtered from background noise.

Other factors in the process of brain imagining complicate the results. Brain structure varies greatly within the normal population, as does the extent to which the brain compensates for pathology. Significant mechanical variables include the limits of magnet strength, spatial resolution, and the inter-rater reliability of the interpreters. A brain abnormality does not necessarily imply dysfunction. Most current data allows only correlation, not causality, to be inferred. To draw valid conclusions about brain imaging, one must determine whether the imaging technique is sensitive, accurate, reliable, valid, and reproducible. These conclusions also depend on what is being measured and what the measurements mean. A structural MRI scan is unable to accurately predict even the age or gender of a patient. None of these tests (either structural or functional neuroimages) has independent predictive value.

PET, SPECT, and fMRI reveal changes in blood flow that are presumed to reflect changes in local brain cell activity. They essentially represent statistical maps of the probability of a change in local blood flow correlated with some task being carried out by the subject. Although they are described as "real-time" brain images, the temporal resolution of the hemodynamic response is several seconds while events relevant to information processing are at least three orders of magnitude faster.[51] Similarly, PET, SPECT, and fMRI spatial resolution is measured in millimeters while nerve cells and axons are three orders of magnitude smaller. [52]

Furthermore, the utility of functional imaging depends on the questions the subject is being asked. Scientists and clinicians must decide what type of tasks test subjects should perform, how precisely the experiment should be set up, what kind of individuals to include in the test group, and how to define the control population. Data are reconstructed by using a set of mathematical assumptions that are framed by an experimental context or design. Such reconstructions involve statistical analyses of comparisons. These steps are not standardized from one technology to the next, or from one machine or laboratory to the next. The more detailed the study, the more complex the

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48 Novelline, *supra* note 47, at 603.
49 *See* Shepherd, *supra* note 43, at 393.
50 *Id.* at 393.
51 Colin Blakemore, *Harveian Oration*, 366 Lancet 2035, 2035-2059 (2005).
52 *Id.*

interpretation of data. Each person's brain in these detailed studies is mathematically "squeezed" to appear the same size, which may give the false impression that an individual is abnormal, when actually the person is merely not average. Even if the finding is abnormal, it may not imply dysfunction.

In summary, these techniques offer a visual advantage in both quantifying and qualifying types of brain activity. However, they present a unique danger because of the appearance of scientific neutrality. The computer-generated analysis of imaging data presents the same problems with standardization as those encountered with other medical modalities. While this poses few diagnostic difficulties in the medical arena, it may pose serious problems in the legal arena. With these cautions in mind, this Article proceeds to cases where imaging is instructive.

V. NEUROPATHOLOGY AND VIOLENCE - THE SOLID GROUND

A. Dementia

Dementia, defined as a disabling deterioration of previous intellectual function, is the result of brain diseases such as Alzheimer's disease and Cerebrovascular disease.[53] In general, dementia impairs memory, language, perception, and visual skills and most notably for the purposes of this Article, may also affect judgment, abstraction, and problem solving skills. Common behavioral manifestations include agitation, aggression, dysphoria, disinhibition, paranoid delusions, hallucinations, and aberrant motor behavior, all of which can translate into violence. While the various forms of dementia have different types and locations of brain abnormality, traditional structural CAT and MRI scans play a significant role in locating and characterizing such pathology. For example, the MRI of a patient with Alzheimer's disease may show atrophy and enlargement of the ventricles and cortical sulci. PET and SPECT scans are sometimes used to measure reduced tissue perfusion or reduced metabolic brain activity in such instances. But do these modalities offer salient links to behavior which might inform the legal process?

1. Public Domain Case: Vincent Gigante

The case of Vincent Gigante is instructive for both the diagnosis of dementia and the complexities of providing expert testimony. Authorities alleged that Mr. Gigante directed an organized crime family in New York.[54] He was a fixture in his Greenwich Village neighborhood for many years, and was often seen walking the streets in his bathrobe and slippers muttering to himself.[55] It was widely believed that he was feigning mental illness in order to provide evidence for an insanity defense should he be arrested.[56] His "act" seemed to belie the fact that he was directing one of the most powerful

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[53] David Mylan Kaufman, Clinical Neurology for Psychiatrists, 123, 127 (W.B. Saunders ed. 5th ed. 2001).

[54] Peter Maas, Underboss 185-86 (HarperCollins Publishers 1997).

[55] *Id.*

[56] Richard E. Vatz & Lee S. Weinberg, *Getting Away With Murder: Vincent Gigante and Exculpatory Psychiatry*, USA Today Magazine, Mar. 2004, at 26, 27.

organized crime families of New York.[57] During his trial, the defense presented a number of renowned experts to testify that Mr. Gigante was not competent to stand trial.[58] In addition to the possibility of psychosis, there were indications of vascular dementia evidenced by neuropsychological testing.[59] PET scans were presented to bolster the clinical diagnosis of dementia but were rejected due to lack of baseline studies and the limited number of controls.[60] Jonathan Brodie, a psychiatrist and expert in the field of neuroimaging, felt that the PET data was inconclusive and did not support a diagnosis of vascular dementia. Testifying as a technical advisor for the judge on the case, Brodie further argued that the clinical picture was inconsistent with the diagnosis of dementia.[61]

Mr. Gigante was found guilty and subsequently signed a statement "admitting" that he had been faking mental illness in order to avoid conviction.[62] After the trial there was a barrage of media criticism of the scientific expert testimony questioning how so many experts could have been fooled.[63] William Reid, a past president of the American Academy of Psychiatry and the Law, and one of the experts retained by the defense, offered some insight into the complexities of the case. He described new "evidence" presented to the experts after Gigante was found competent to stand trial and guilty of the charges.[64] Tape-recorded conversations seemed to indicate that Gigante had considerable intellectual ability and prosecutors threatened to charge family members with obstruction of justice unless Gigante admitted his deception.[65]

The wide variance among expert opinions in the Gigante case aptly highlights the fact that judges and juries face a complex task when assigning weight to conflicting scientific testimony. They are expected to listen to complex testimony often given in technical jargon and are presented with contradictory expert opinion and must extract those points pertinent to the case. In trials with intense media involvement, the task of remaining "objective" is rendered even more difficult.

2. New Case: Vascular Dementia, Substance Abuse, Stroke

The case of JR reflects an instance when adequate neuroimaging might have contributed to a superior resolution of criminal charges.[66] JR was a 50-

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[57] *Id.* at 27.

[58] *Id.* at 27-28.

[59] Andy Newman, *Gigante Says He Was Crazy . . . Like a Fox*, N.Y. TIMES, Apr. 8, 2003, at D1.

[60] Brickford Y. Brown, et al., *Are We Out of the Gray Area Yet? Recent Developments in the Use of PET and SPECT Scans to Prove Causation and Injury in Toxic Tort Litigation*, http://www.morankikerbrown.com/CM/Articles/Articles67.asp.

[61] Personal communication with Dr. Brodie as well as a letter written to the Judge in this case, written by Dr. Brodie and furnished to the authors.

[62] Newman, *supra* note 59.

[63] *Id.* Testimony Before Texas State House of Representatives Committee on Criminal Jurisprudence Re: HB 614, Michael Welner, April 8,2003, *available at* http://www.forensicpanel.com/aboutus/pressroom/2003_04_08_texastestimony2.htm.

[64] William H. Reid, *Expert Evaluation, Controversial Cases, and the Media*., 9 J. PSYCHIATRIC PRACTICE 388, 388-90 (2003).

[65] *Id.*

[66] This information derives from Dr. Edersheim's clinical practice.

year-old woman charged with manslaughter. It was alleged that she pushed a hospital orderly to the ground, resulting in an intracranial hemorrhage and death. JR had a history of insulin dependent diabetes and substance abuse. At age 49, she was found unconscious at her home with cocaine paraphernalia nearby. She demonstrated several cardinal signs of severe brain injury (abnormal language, right sided weakness, impaired responsiveness to external stimuli) and was diagnosed with a left-sided cerebrovascular infarct (stroke). A CAT scan indicated the presence of a small area of tissue damage deep in the brain and an electroencephalogram (EEG, which measures electrical activity spontaneously generated by the brain) showed slowed background rhythm with intermittent rhythmic slowing, which is often seen in patients with generalized brain dysfunction. There is no record of any subsequent neuroimaging. Initial neuropsychological testing documented widespread and severe cognitive impairments. Repeat neuropsychological testing confirmed persistent neurological injuries, including widespread cognitive deficits ("likely involving bilateral cortical and subcortical structures, particularly frontal systems"), functional illiteracy, and impaired auditory comprehension. JR showed profound deficits in memory and information processing and had poor self-control, thought to be a manifestation of damage to the frontal lobes of her brain, which mediate behavior and planning. Her mood was volatile, ranging from elevated to agitated and tearful, and shifted quickly without warning. She became dysphoric, belligerent, and aggressive and allegedly pushed a hospital employee, who sustained head injuries resulting in his death.

JR's attorney requested an independent medical examination to determine whether JR possessed the competency required to stand trial. The examiner offered his opinion that JR suffered from severe cognitive deficits and that her brain injury was sufficient to cause a personality disorder, with volatile emotions, disinhibition, and the propensity to become frustrated and aggressive. While the judge agreed that JR was currently not competent to stand trial, he remanded JR to the custody of a state forensic unit indefinitely in order to determine whether she could be restored to competency. JR remains in custody seven years after this ruling, and has had approximately eight subsequent independent forensic evaluations for competency to stand trial. There is no indication that JR has had serial neurologic examinations to clarify whether her dementia is progressive or stable, which is particularly relevant in the context of a vascular dementia. Adding structural brain imaging to this neurological data might have resolved the issue of whether JR would ever regain sufficient cognition to participate in a trial, allowing for the final resolution of her charges and appropriate treatment.

3. New Case: Pick's Disease[67]

FK was a 58-year-old gentleman with no previous psychiatric history. He had emigrated from Europe to the United States, obtained degrees in engineering, and was from a socially prominent family. Over the course of a

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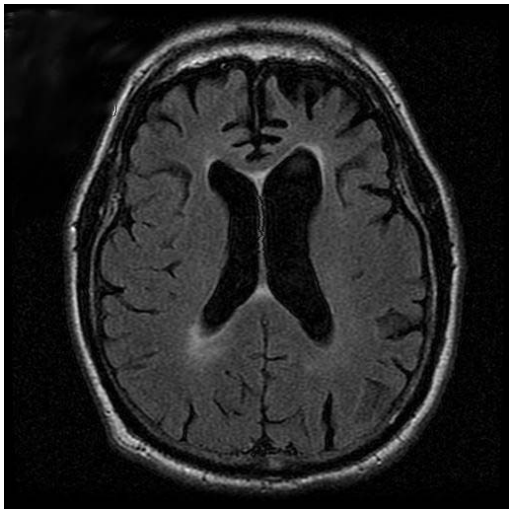
[67] The case that follows was described to the authors by one of their colleagues, derived from their colleague's clinical experience.

two-year period, FK's behavior deteriorated. Specifically, he exhibited gross changes in his social behavior. He began spitting compulsively and fondling himself, and was eventually arrested for open and gross lewdness due to public urination. FK's charges included child endangerment because he was apprehended within a short distance of a school. Neuro-psychological testing demonstrated significant deficits on tests of executive functioning, suggesting pathology in his frontal lobes. Neuroimaging confirmed atrophy of the frontal lobes (see Figure 7).

Figure 7.



[68]

The neurologist determined that FK was suffering from Pick's Disease, an uncommon form of dementia that causes personality changes, loss of social skills, loss of intellectual function, and speech disturbances. This case has not yet been adjudicated. It is likely that his neurological problems, well documented by multiple complementary modalities, will determine the eventual resolution of his charges.

4. Research: The Neuroimaging of Dementia

Diagnostic imaging is being used with greater accuracy in confirming the diagnosis of Alzheimer Dementia (AD),[69] as well as Frontotemporal Dementia (FTD) and Vascular Dementia. Some genetically determined forms of AD are identifiable by structural changes that predate the emergence of clinical symptoms.[70] The development of AD can be predicted with the improving accuracy of functional scans, which document a reduction of glucose

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[68] This image, taken during the course of Dr. Price's clinical practice, depicts an axial brain MRI scan demonstrating selective atrophy of the frontal lobes.

[69] K. Kantarci & C.R. Jack Jr., *Neuroimaging in Alzheimer Disease: An Evidence-Based Review*, 13 NEUROIMAGING CLINICS N. AM. 197, 205-06 (2003).

[70] Jose Masdeu et al., *Neuroimaging as a Marker of the Onset and Progression of Alzheimer's Disease*, 236 J. NEUROLOGICAL SCI. 55, 55 (2005).

metabolism in certain areas of the brain.[71] Teipel and colleagues describe a method for analyzing the portion of the brain believed to be the epicenter of the loss of acetylcholine, a neurotransmitter (chemical message mediator) thought to be instrumental in the pathology of AD.[72] Frontotemporal Dementia (FTD), of which Pick's Disease is a subset, has been important in forensic contexts because of the behavioral changes that often signify the disorder's presenting symptoms.[73] Miller and colleagues have done extensive work on this disease and its implications for behavior.[74] They compared twenty-two FTD subjects with twenty-two AD subjects and found a significant correlation between FTD and anti-social behavior.[75] Diagnoses were made clinically and confirmed with SPECT scans.[76] Mendez demonstrated that such patients suffered a loss of insight best described as a "lack of concern."[77] PET and SPECT scans confirmed the hypometabolism and hypoperfusion within these patients' prefrontal regions.[78]

Both of the above examples highlight an important point. Patients who are neurologically impaired by progressive dementia typically are not restorable to competency. The persistence of criminal charges limits the options for resolution of the cases. Neuroimaging evidence might facilitate a superior disposition of these cases, e.g. civil commitment or guardianship.

B. Traumatic Brain/Frontal Lobe Injury

About 1.5 million people each year in the United States sustain a traumatic brain injury (TBI).[79] Leading causes include motor vehicle accidents, falls, assaults, recreational accidents, and warfare.[80] Frontal, anterior temporal, and limbic regions of the brain are particularly vulnerable and relevant to behavioral changes. The emergence of psychiatric disorders related to TBI are likely due to a complex interplay of factors including the nature, location, and severity of the neurological injury, the age at which it occurs, premorbid personality and cognition, pre-existing psychiatric illness,

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[71] Lisa Mosconi, *Brain Glucose Metabolism in the Early and Specific Diagnosis of Alzheimer's Disease: FDG-PET Studies in MCI and AD*, Eur. J. Nuclear Med. & Molecular Imaging 486, 505 (2005).

[72] Stefan J. Teipel et al., *Measurement of Basal Forebrain Atrophy in Alzheimer's Disease Using MRI*, 128 Brain 2626, 2626-44 (2006); Marina Boban et al., Letter to the Editor, *Nucleus Subputaminalis: Neglected Part of the Basil Nucleus of Meynert*, 129 Brain E42; Helmut Heinsen, et al., Response, *Response to Boban et al: Computer-Assisted 3D Resconstruction of Basalis Complex, Including the Nucleus Subtaminalis (Ayala's Nucleus)*, 129 Brain E43.

[73] M.F. Mendez et al., *Functional Neuroimaging and Presenting Psychiatric Features in Frontotemporal Dementia*, 77 J. Neurology, Neurosurgery, & Psychiatry 4, 4 (2006).

[74] *See* Bruce L. Miller et al., *A 34-year-old Man with Progressive Behavioral and Language Disturbance*, 170 Neurology 68, 68-74 (2007).

[75] Bruce L. Miller et al., *Aggressive, Socially Disruptive and Antisocial Behavior Associated with Fronto-Temporal Dementia*, 170 Brit. J. Psychiatry 150,150-154 (1997).

[76] *Id.* at 151.

[77] Mario F. Mendez et al., *Loss of Insight and Functional Neuroimaging in Frontotemporal Dementia*, 17 J. Neuropsychiatry & Clinical Neurosci. 413, 413 (2005).

[78] *Id.* at 413.

[79] CDC Injury Center, Traumatic Brain Injury in the United States: A Report to Congress (1999), *available at* http://0-www.cdc.gov.mill1.sjlibrary.org/ncipc/tbi/tbi_congress/TBI_in_the_US.PDF.

[80] *Id.*

substance abuse history, family psychiatric history, educational level, occupational status, coping strategies, age, stressors and support systems. The possibility of psychological and financial gains motivates some claims regarding TBI and psychiatric symptoms. Structural MRIs can often detect acutely diffuse axonal injury, small hemorrhages, edema, or contusions that characterize TBI. The typical profile of patients with TBI-related psychosis includes a median latency to onset of one year and the presence of delusions and hallucinations.[81] As demonstrated in the cases presented below, a confluence of data, including cognitive and behavioral changes in the wake of TBI as well as correlative MRI, EEG, and neuropsychological evaluations, offer the best evidence for a causal relationship.

1. Public Domain Case: GK[82]

GK was a 31-yr-old right-handed man who was diagnosed with numerous psychiatric disorders including atypical psychosis, schizophrenia, and antisocial or borderline personality disorders. When he was seven days old, he had brain surgery to remove excess fluid in order to relieve pressure on the brain and avoid possible mental retardation. Post-operatively, GK was noted to have an irregular collection of fluid in the left frontal lobe, however the intracranial pressure had stabilized and he recovered. By age eight, he began to show serious behavioral difficulties. He did not respond to parental discipline, always sought immediate gratification, and blamed his difficulties on others. He was irresponsible, tended to wander, and consistently fell under the influence of other oppositional children. Under firm guidance and after two school transfers, he was able to graduate from high school and joined the Marine Corps. He was dishonorably discharged after six weeks.

Over the next 10 years, GK was hospitalized twenty-seven times in psychiatric institutions, and imprisoned eight times on charges of assault, forgery, grand larceny, drug involvement, and lewd behavior. Some examples of his inappropriate behaviors were as follows. While walking by a gas station GK saw an unattended taxi with keys in the ignition. He jumped in and drove off, ripping the hose from the gas pump, only to be captured several blocks away. He escaped from a locked psychiatric ward after being reprimanded by a ward attendant. He scratched that attendant's car with broken glass, signed his own name, and re-entered the ward. When confronted, he denied his involvement. GK was charged with arson of two public buildings. He was sexually promiscuous, often trading cigarettes for oral sex, and masturbated in public. Neither individual psychotherapy nor trials of multiple psychotropic agents were effective. An MRI revealed bilateral frontal lobe lesions with greater involvement of the left side (see Figure 8).

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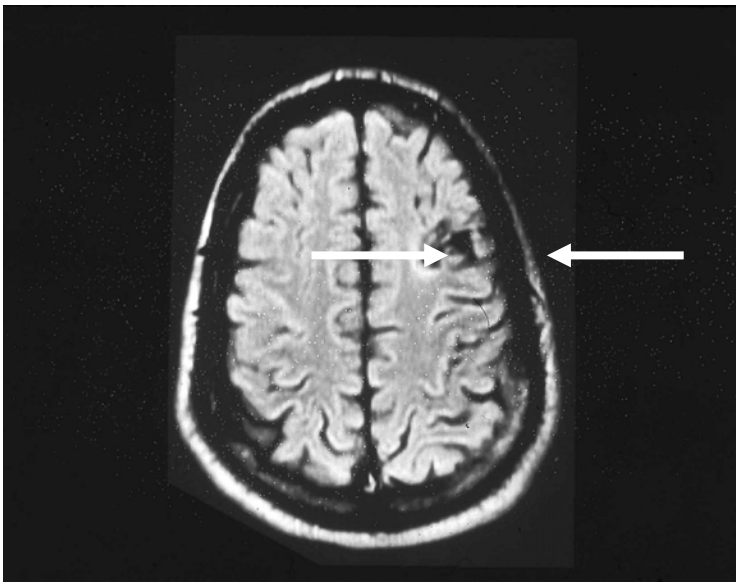
[81] E.M. Warriner & D. Velikonjad, *Psychiatric Disturbances after Traumatic Brain Injury: Neurobehavioral and Personality Changes*, 8 CURRENT PSYCHIATRY REP. 73, 74 (2006).

[82] Bruce H. Price et al., *supra* note 42, at 1383-1384 (1990). The following section is substantially derived from Dr. Price's personal clinical experience, partially relayed in an article that he jointly wrote for BRAIN.

Figure 8.

 [83]

As one might predict given his behavioral profile, GK was lost to neurological follow-up. The resolution of his legal cases is unknown.

2. Public Domain Case: TH[84]

At the age of fifteen, TH suffered a major head injury after a skateboarding accident. He required two surgeries, the second of which involved a frontal lobe resection, and was in a coma for six weeks (see Figure 9). After the accident, neighbors stated TH was never the same. "As an adult . . . he had the mind of a 12 year old. He would wander into homes uninvited, pester people on the street, and ride his bike on neighbors' lawns." His father lamented his inability to get his son help since noting the changes in him following the accident. Several years after his accident, TH, encouraged by his friends, lit a box of firecrackers in a crowded fireworks store. The ensuing fire killed nine people and seriously injured eleven. During his arraignment, TH "giggled like a delighted child and mugged for the cameras." While being photographed coming into the courtroom he joked, "Let me do my hair first." As the charges were being read, TH protested his innocence and hurled obscenities at the judge. He pleaded not guilty by reason of insanity. TH was found incompetent to stand trial and was remanded to a state mental health facility where he still resides.

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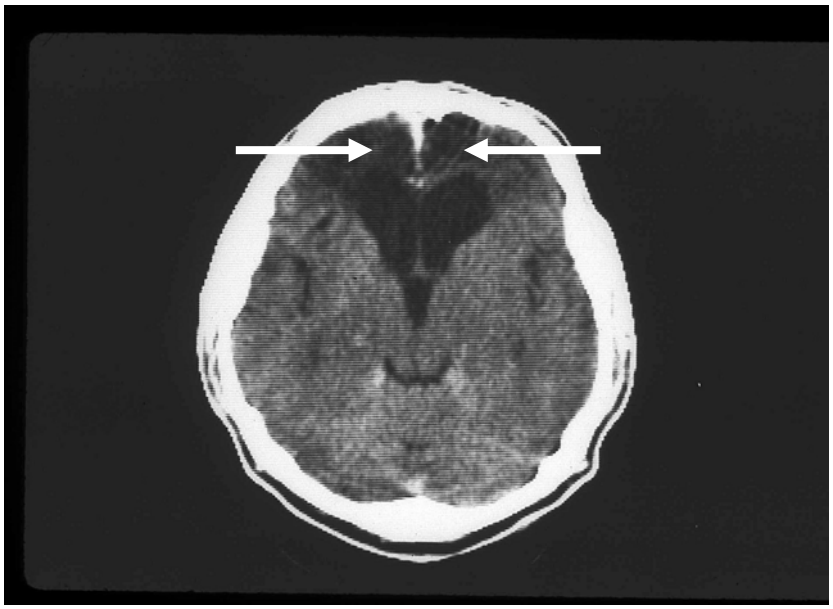
[83] *Id.* This image, taken during the course of Dr. Price's clinical practice, depicts an axial brain MRI scan demonstrating left frontal lobe damage in GK. By convention, the left brain appears on the examiners right. The axial plane is the view from the patient's feet upward into the brain.

[84] *See* Mark Williams, *Man who Started Fire Had Had A Lobotomy*, SOUTH COAST TODAY, Jun. 6, 1996, available at http://archive.southcoasttoday.com/daily/07-96/07-06-96/a03wn022.htm.

Figure 9.

 [85]

3. Research: The Brain Imaging of TBI

There is a multitude of neuropsychological effects and behavioral disturbances that can result from TBI.[86] The frontal lobes, as has been shown, are particularly vulnerable and relevant to behavioral changes.[87] GK provides an example of an injury at a young age that prevents the appropriate maturation of executive function as mediated by the frontal lobes. TH's case is illustrative of an injury suffered after the development of these skills but with subsequent loss, similar to Phineas Gage. Many studies using structural and functional imaging elucidate the diffuse axonal injury that characterizes TBI.[88] Neuropsychological tests are more sensitive in detecting deficits, but Ichise and colleagues demonstrated that imaging techniques can identify

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[85] This image, taken during the course of Dr. Price's clinical practice, depicts an axial brain CAT scan demonstrating bilateral frontal lobe trauma. CAT scans are generally limited to axial orientation only. MRI scans can image in any orientation: axial, coronal, or sagittal.

[86] J.E. Max et al., *Personality Change Disorder in Children and Adolescents Following Traumatic Brain Injury*, 6 J. Int'l Neuropsychological Soc'y 279, 279 (2000); Dan J. Stein & Fredrick G. Moeller,*The Man Who Turned Bad*, CNS Spectrums 88, 90 (2005).

[87] M.C. Brower & B.H Price, *Neuropsychiatry of Frontal Lobe Dysfunction in Violent and Criminal Behavior: A Critical Review*, 71 J. Neurology, Neurosurgery, & Psychiatry 720, 720 (2001); Nora D. Volkow & Laurence Tancredi, *Neural Substrates of Violent Behaviour: A Preliminary Study with Positron Emission Tomography*, 151 Brit. J. Psychiatry 668, 670-72 (1987).

[88] Bruce Lee & Andrew Newberg, *Neuroimaging in Traumatic Brain Imaging*, 2 NeuroRx: J. Am. Soc'y for Experimental NeuroTherapeutics 372, 377 (2005); Thomas W. McAllister et al., *Neuroimaging Findings in Mild Traumatic Brain Injury*, 23 J. Clinical Experimental Neuropsychology 775, 775(2001).

abnormalities in these patients.[89] This is especially true of functional imaging since many patients may demonstrate significant deficits on neuropsychological tests but have normal structural MRIs. Fontaine and colleagues describe studies that correlated functional imaging (SPECT and PET respectively) with diagnoses made using clinical exams and neuropsychological tests.[90] In addition to aiding in the diagnosis, these images may also help to improve understanding of specific symptoms exhibited and to guide treatment options.[91] It is important to note that often the degree of impairment cannot be ascertained by clinical examination or neuroimaging standing alone. It is essential to correlate the data from different diagnostic modalities including history, clinical examination, brain imaging, and neuropsychological testing.

C. Seizure Disorders/Epilepsy

The use of the EEG in the courtroom has a controversial past. While the understanding of seizure activity in the brain has progressed markedly since the 1960s, there is little evidence that a seizure disorder alone can explain planned, complicated, sequenced, and directed violent behavior.[92] There are, however, instances in which chronic, uncontrolled seizure disorders and their neuropathological effects have combined with other brain injuries to establish a link between neuropathology and criminal acts.[93] To the extent that functional imaging can capture the nature of the seizure activity and the neuroanatomical changes that may have resulted, it may play a significant role in establishing this link.

1. Public Domain Case: Jack Ruby

Historically, neurological data have been given great evidentiary weight, often before the scientific basis warranted this degree of confidence. A well-known example of scientific prematurity was the use of EEG evidence in the 1960s. In 1964, Jack Ruby went on trial for the murder of Lee Harvey Oswald. His defense team presented EEG evidence purporting to show that he had a seizure disorder and argued that this disorder caused him to shoot Oswald.[94] The use of EEG evidence occurred more than a decade before neuroimaging became an available diagnostic modality. At issue was an allegedly abnormal electrical rhythm ("bursts of notched waves with moderate voltages of a frequency of 5 to 6 cycles per second lasting for 5 to 10 seconds most clearly seen in the mid-temporal regions"), which is now known

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[89] Masanori Ichise et al., *Technetium-99m-HMPAO SPECT, CT, and MRI in the Evaluation of Patients with Chronic Traumatic Brain Injury: A correlation with Neuropsychological Performance*, 35 J. Nuclear Med. 217, 217 (1994).

[90] A. Fontaine et al., *Functional Anatomy of Neuropsychological Deficits after Severe Traumatic Brain Injury*, 53 Neurology 1963, 1963 (1999).

[91] B. Levine et al., *In Vivo Characterization of Traumatic Brain Injury Neuropathology with Structural and Functional Neuroimaging*, 23 J. Neurotrauma 1396, 1411 (2006).

[92] Mario F. Mendez, *Postical Violence and Epilepsy*, 39 Psychosomatics 478, 478 (1998).

[93] *Id.*

[94] Lawrence Zelic Freeman, *Forensic Psychiatry*, 120 Am. J. Psychiatry 708 (1965).

to be a normal variant in the general population.[95] Many of the most prominent psychiatrists, psychologists, and neurologists of the day testified for and against the proposition that Ruby suffered from "psychomotor seizures" and whether, if present, these seizures rendered him unable to conform his conduct to the requirements of the law.[96] The defense team ultimately failed in its attempt to exculpate Ruby on the basis of this seizure disorder, but the approach ushered in a new era regarding the use of emerging knowledge of the brain as a defense to criminal behavior.

2. New Cases: Viral Encephalitis, Intractable Seizure Disorder, Pyromania

The case of PR[97] illustrates an instance in which structural and functional neuroimaging, in combination with other diagnostic modalities, contributed significantly to the resolution of a criminal case. PR was a twenty-five-year-old male charged with homicide in connection with a fire that he allegedly set in a warehouse. Prior to his arrest, PR's childhood and early adulthood were characterized by continuous psychiatric, medical, and behavioral difficulties. At the age of two, he contracted Herpes Simplex Virus (HSV) encephalitis (a brain virus) that resulted in four hours of continuous seizure activity and subsequent coma. After his discharge from the hospital, PR exhibited uncharacteristic behavioral disturbances, including random aggression and rage. He also demonstrated serious learning disabilities and information processing difficulties in early childhood and was diagnosed with speech and language impairments. PR's aggression continued throughout early childhood and adolescence and included violent outbursts, running away, and self-mutilation while at home and school.

Frequent educational and psychological testing demonstrated PR had a below average IQ and severely impaired memory, learning and language abilities. Between the ages of two and twelve, he also demonstrated symptoms of an ongoing seizure disorder but was inconsistently treated with anti-seizure medications. At approximately age thirteen, his aggression began to decrease in severity and frequency, coincident with an increase in complex partial seizure activity and the beginning of fire setting behaviors. PR reported a consistent fascination and preoccupation with setting and watching fires. His seizures became more frequent and intense, and included vomiting, dizziness, watery eyes, agitation, "lost time", fatigue, inability to concentrate, and mood swings with uncontrollable crying. He also exhibited automatisms, or stereotyped involuntary movements, such as lip smacking.

During the four years prior to his arrest, PR's seizure disorder became intractable, resulting in up to eight seizures daily despite multiple anticonvulsant medications. He received a comprehensive diagnostic workup that included a battery of neuropsychological tests. PR scored in the below average ranges on intelligence testing and in the severely to moderately impaired range on specific tests of memory, learning, language, attention, concentration, and executive ability. Brain MRI showed bilaterally abnormal hippocampi (temporal lobe structures) with scarring and shrinkage of these

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95 *Id.* at 709.
96 *See id.* at 709.
97 This information derives from Dr. Edersheim's clinical practice.

structures. The data from the neuropsychological testing and MRI were corroborative, as Herpes Simplex Virus has a predilection for frontal and temporal structures. Continuous EEG monitoring showed left temporal lobe epilepsy and his treatment team considered resection of the left temporal lobe for seizure control. However, both depth electrode placement (right hippocampal, left hippocampal and right parietal foci) and SPECT imaging (bilateral mesiotemporal hypoperfusion interictally, left temporal and right frontoparietal hyperperfusion or activation ictally) revealed multifocal bilateral disease, which precluded resection.

At trial, an independent medical expert retained by PR's attorney offered the opinion that PR's fire setting was the result of the interaction between his neurological deficits and his underlying psychiatric illness. The expert diagnosed him with an impulse control disorder (pyromania), multiple cognitive deficits, and an intractable seizure disorder. The evaluator further testified that PR's impaired neurologic functioning interacted with his urges to set the fire in a manner that rendered him unable to control his impulses or anticipate their consequences. Although careful not to overstate direct links between brain pathology and behavior, the expert testified that this case presented an unusual wealth of neurological evidence supporting this link, all obtained with state of the art diagnostic modalities very close to the time of the offense. PR was found not guilty by reason of insanity and was remanded to a state psychiatric facility. In light of the permanent and intractable nature of his neurologic deficits, it is unlikely that PR's condition will improve sufficiently to allow him to live in an unsupervised setting.

3. Research: Seizure Disorders and Aggression

A controversial area of research into aggression has been in the area of epilepsy.[98] With some exceptions, it is difficult to make a direct correlation between aggression and seizures. Marsh and Krauss presented several case examples of violence or aggression committed in the context of a seizure disorder.[99] They subdivided the stages of seizures (pre-seizure, during the seizure, post-seizure, between seizures) and described the potential for aggression during each phase.[100] During a seizure and between seizures, directed aggression is extremely rare.[101] The gold standard for seizure diagnosis is EEG. Neuroimaging may confirm the presence of a lesion that explains the findings on an EEG.[102] Some studies of aggressive patients with documented seizure disorders demonstrate lesions in the temporal lobe.[103] Additionally, fMRI results have correlated temporal lobe lesions with an individual's impaired fear processing, a feature associated with aggression in

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[98] Brower & Price, *supra* note 2, at 145.

[99] Laura Marsh & Gregory Krauss, *Aggression and Violence in Patients with Epilepsy*, 1 EPILEPSY & BEHAV. 160, 160 (2000).

[100] *Id.* at 161-166.

[101] *Id.*

[102] L. Bonilha et al., *Medial Temporal Lobe Atrophy in Patients with Refractory Temporal Lobe Epilepsy*, 74 J. NEUROLOGY, NEUROSURGERY, & PSYCHIATRY 1627, 1627 (2003).

[103] L. Tebartz van Elst et al., *Affective Aggression in Patients with Temporal Lobe Epilepsy: A Quantitative MRI Study of the Amygdala*, 123 BRAIN 234, 234 (2003).

animal models.[104] J.M. Tonkonogy presented five cases of patients with identifiable lesions and histories of aggressive and impulsive behavior.[105] In several of these patients, lesions were associated with seizure disorders captured on EEG recordings.[106] Neuroimaging alone is not sufficient to make the diagnosis of temporal lobe epilepsy (internationally classified as complex partial seizures), because a substantial portion of patients with the diagnosis have normal MRIs.[107] The use of corroborative data is essential for associating seizures with violent activity.

D. Mass Lesions

Mass lesions in the brain are tumors that result from the overgrowth of cells. The brain is a delicate organ housed in a closed cranium, and even small tumors may displace and compress normal brain tissue. There are myriad physical symptoms accompanying such lesions, depending on where they occur in the brain, their rate of growth, and on what structures they impinge. For the purposes of this Article, however, it is important to note that some tumors can cause significant changes in communication, comportment, memory, judgment, and emotional regulation. In this regard, tumors are sometimes implicated in legal actions.

1. Public Domain Case: Charles Whitman[108]

In the early morning hours of August 1, 1966, Charles Whitman, a twenty-four-year-old former Marine and University of Texas student, murdered his wife and mother. Later that morning he purchased ammunition and a shotgun and climbed the bell tower at the University of Texas. Along the way he murdered two more people. Once atop the tower he opened fire on the people below killing eleven before being shot and killed by the police. In a note composed prior to his rampage, he wrote: "Lately, (I can't recall when it started) I have been the victim of many unusual and irrational thoughts." He complained of headaches and asked that a post mortem be performed to elucidate his "mental illness." He had sought psychiatric help during which he revealed a fantasy to shoot people from the bell tower. The autopsy revealed a small tumor whose role in his actions has since been debated. The original pathology report described the tumor as a glioblastoma multiforme, the size of a walnut, protruding from beneath the thalamus, impacting the hypothalamus, extending into the temporal lobe and compressing the amygdaloid nucleus. This type of tumor is highly malignant and aggressive. Neurologists continue to debate the significance and accuracy of the

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[104] Francesca Benuzzi et al., *Impaired Fear Processing in Right Mesial Temporal Sclerosis: A fMRI Study*, 63 Brain Research Bulletin 269, 270 (2004).

[105] Joseph M. Tonkonogy, *Violence and Temporal Lobe Lesion: Head CT and MRI Data*, 3 J. Neuropsychiatry 189, 189 (1991).

[106] *Id.* at 190.

[107] Aaron A. Cohen-Gadol, et al., *Normal Magnetic Resonance Imaging and Medial temporal Lobe Epilepsy: The Clinical Syndrome of Paradoxical Temporal Lobe Epilepsy*, 102 J. Neurosurgery 902, 902 (2005).

[108] Clayton Stapleton, *"It's Coming from the Tower"*, http://www.whatwasthen.com/uttower.html.

conclusions of the pathologists in the case, highlighting the divergence of opinion that can exist among experts.

2. New Case: Craniopharyingioma

JC was a fifty-eight-year-old male without a history of mental disorder.[109] As a clearing officer for a brokerage house, JC was responsible for receiving funds and transferring or depositing them for the client companies. He successfully performed this duty for many years and was well respected within his field. This changed dramatically when he began collecting the checks, many of which were worth tens of thousands of dollars, and placing them in a drawer in his desk. JC made no attempt to cash these checks or divert them to his own accounts. There is no evidence that JC displayed any other significant psychiatric or behavioral changes. He finally sought medical attention at the urging of his regular squash partner, who noticed that if he hit the ball into the upper left corner of the court, JC would consistently miss the return.

An MRI revealed a craniopharyngioma, a tumor near the pituitary gland, situated in the deep brain in close proximity to the optic nerve (see Figure 10). The tumor was pressing on the optic nerve, which accounted for JC's loss of vision in his squash game. Upon resectioning the tumor, JC's visual and behavioral abnormalities resolved. He was puzzled and embarrassed by his actions and sought to provide restitution for any financial damages caused by his failure to properly route the funds. No criminal charges were filed. However, he was sanctioned by the Securities Exchange Commission and his securities license was permanently revoked.

3. Research: Mass Lesions

Several important studies have reinforced the link between the presence of a mass in the brain and its role in causing behavioral dysregulation. Nakaji and colleagues presented two case reports of aggressive and antisocial behavior showing dramatic improvement after the successful resection of temporal lobe tumors.[110] His first case serves as an example for the role a tumor can play in creating aggressive behavior. The patient was a thirteen-year-old boy with a seizure disorder since the age of five. An MRI demonstrated a small right midline temporal lobe tumor that did not appear to be increasing the pressure inside the cranium. This condition, if left unchecked, can lead to severe brain injury and death. His treatment team made the decision to manage his seizures with medication rather than surgery. In addition to experiencing continued seizure activity, the patient was extremely aggressive and antisocial. He required frequent hospitalizations and was restrained for up to six hours per day. His behavior included bizarre suicide attempts (such as trying to ride his skateboard on the freeway) and violence against others. He was referred for neurosurgery and underwent a successful resection of the tumor. Subsequent to the operation

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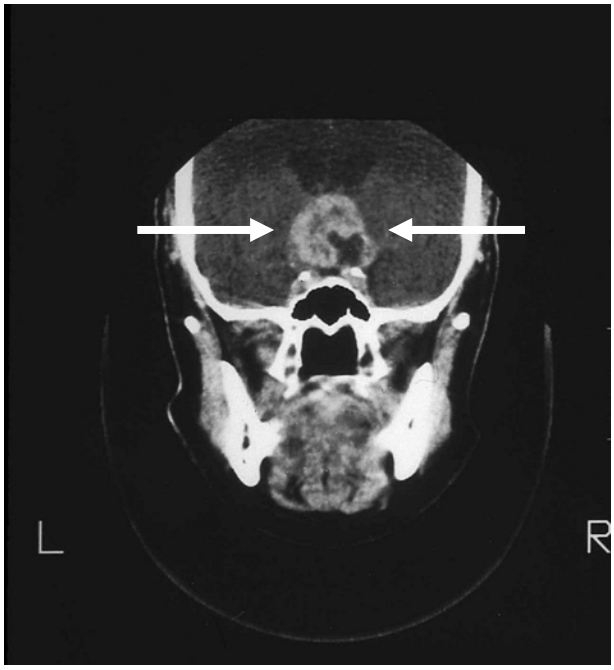
[109] The case that follows was described to the authors by one of their colleagues, derived from their colleague's clinical experience.

[110] Peter Nakaji et al., *Improvement of Aggressive and Antisocial Behavior after Resection of Temporal Lobe Tumors*, 112 PEDIATRICS, 430, 430 (2003).

he was successfully weaned off of his anti-epileptic medication and there was no resumption of the aggressive and violent behavior.

The assertion that a structural lesion can "cause" a violent act has been debated extensively. Relkin and colleagues presented a case of a sixty-five-year-old man who murdered his wife after she scratched his face in a fight.[111] He had no previous history of mental illness or violence. After an extensive work-up that found no significant abnormalities on neuropsychological testing, an MRI revealed a large left-sided arachnoid cyst that had likely been growing for many years. Areas adjacent to the cyst were found to have decreased metabolism on PET scanning, a finding used to support the theory that he was not responsible for his actions that day. The medical and legal communities have debated this case in a broad-based analysis published in the 1996 Seminars in Clinical Neuropsychiatry. Many of the issues raised in that debate are reflected in this Article. While there is discussion within the scientific and medical communities about the potential effects of tumors on brain function, it is altogether different to make the leap to legal standards of criminal responsibility.[112]

Figure 10.



[113]

[111] N. Relkin et al., *Impulsive Homicide Associated With an Arachnoid Cyst and Unilateral Frontotemporal Cerebral Dysfunction*, 1 Seminars Clinical Neuropsychiatry 172, 183 (1996).

[112] S.J. Morse, *Brain and Blame*, Seminars Clinical Neuropsychiatry 222, 235 (1996).

[113] This image, taken during the course of Dr. Price's clinical practice, depicts a coronal brain MRI scan demonstrating a craniopharyngioma. The coronal plane is the patient to examiner "face-to-face" view.

VI. THE FAR REACHES—FROM DIAGNOSIS TO IMAGE

The cases discussed above present only a sampling of situations in which a neurological impairment may be implicated in behavioral abnormalities. But what of the role neuroimaging may play in moving beyond identifying illnesses that directly account for acts of aggression? What about using the technology to image criminality and violence? Although there are several scientific initiatives that have attempted to capture these states, traits, and thoughts, they present the most speculative and ethically problematic uses for functional imaging in the courtroom. At the outset, the concepts underlying some of this research are vague and fail to integrate the current understanding of the biologic, psychiatric, and social underpinnings of violence and crime. For example, there is no illness, injury, or anatomical locus to correlate with the most reliable risk factors for criminal behavior (youth, gender, socioeconomic status, and previous violent acts). There are no normative data that account for contributing factors such as substance abuse, previous head injury, psychiatric or neurological treatment, and the impact of medications. In addition, violence and crime are not unitary concepts and social scientists have made significant progress categorizing violence into meaningful subtypes, with different degrees of aggression, different social determinants, and different precipitants. While these new areas of research are too nascent to play a reliable role in the courtroom, it is possible that some of these subjects may be fruitful areas for understanding the link between brain structures and feeling states.

A. Psychopathy

Several research teams are attempting to localize the brain areas responsible for antisocial behavior and psychopathy, diagnoses that are risk factors for criminal behavior. Psychopathy is defined as a personality disorder characterized by superficiality, glibness, lack of empathy or remorse, and is associated with difficulties in processing and producing affective material.[114] A brain imaging protocol that purports to identify these individuals would certainly have far-reaching implications for the criminal justice system. Thus far, studies have been able to show abnormalities in the structural MRIs of psychopaths including decreased pre-frontal gray matter, decreased posterior hippocampal volume, and increased callosal white matter.[115] Additionally, functional imaging has demonstrated a decrease in frontal and temporal lobe perfusion and metabolism in these individuals.[116] Disrupted limbic structures

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[114] K.A. Kiehl et al., *Limbic Abnormalities in Affective Processing by Criminal Psychopaths as Revealed by Functional Magnetic Resonance Imaging*, 50 Biological Psychiatry 677, 677 (2001).

[115] *See* L. Bassarath, *Neuroimaging Studies of Antisocial Behavior*, 46 Can. J. Psychiatry 728, 729 (2001); J.L. Bufkin & V.R. Luttrell, *Neuroimaging Studies of Aggressive and Violent Behavior; Current Findings and Implications for Criminology and Criminal Justice*, 6 Trauma, Violence, & Abuse 2005 176, 181 (2001); M.P. Laakso et al., *Psychopathy and the Posterior Hippocampus*, 118 Behav. Brain Res. 187, 188 (2001); S. Pridmore et al., *Neuroimaging in Psychopathy*, 39 Austl. & N.Z. J. Psychiatry 856, 858-59 (2005); A. Raine et al., *Reduced Prefrontal Gray Matter Volume and Reduced Autonomic Activity in Antisocial Personality Disorder*, 57 Archives Gen. Psychiatry 119, 123 (2000).

[116] *See* A. Raine et al., *Brain Abnormalities in Murderers Indicated by Positron Emission Tomography*, 42 Biological Psychiatry 495, 498-501 (1997); A. Raine et al.,

(amygdala/hippocampal formation, parahippocampal gyrus, ventral striatum, and anterior/posterior cingulated gyri) have also been postulated to account for the affective abnormalities that define psychopaths.[117] The data from the various studies are not uniform and methodological problems exist including recruitment of subjects, use of controls, and the social, economic, and biological complexities of violence. However, there do seem to be some consistent anomalies that require further investigation.

B. Lying

The history of the polygraph, which indexes alterations in breathing, blood pressure, and perspiration as the physiologic accompaniments of deception, is an informative and cautionary story.[118] Used for almost a century, its reliability is, at best, eighty-five percent with a false positive rate of up to twenty-five percent.[119] Since 1923, polygraph results have been inadmissible as evidence in most federal courts. The 1988 Employee Polygraph Protection Act also criticized polygraphs, and a 2003 National Research Council report concluded that polygraphs were bad science and needed replacing. Despite these criticisms, approximately 40,000 polygraphs per year continue to be performed in the United States by insurance companies, government and law enforcement agencies, and others.[120] Such technology, once introduced, is difficult to retract, despite contrary evidence that the result is more harmful than beneficial.[121]

Several new studies have posited that MRIs can be successfully used to identify brain changes in individuals who fabricate information. This information could benefit both civil and criminal litigation.[122] Yang and colleagues have demonstrated an increase in prefrontal white matter and a reduction in gray/white ratios in the brains of pathological liars.[123] Lee and colleagues have also implicated the prefrontal lobe along with parietal regions in the production of feigned memory impairment.[124] Kozel and colleagues have designed studies to test the use of fMRI in detecting deception.[125] This team designed a method for reproducibly illustrating that certain regions of

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Reduced Prefrontal and Increased Subcortical Brain Functioning Assessed Using Positron Emission Tomography in Predatory and Affective Murderers, 16 Behavioral Sci. & L. 319, 319-20 (1998). J. Tiihonen et al., *Single-Photon Emission Tomography Imaging of Monoamine Transporters in Impulsive Violent Behaviour*, 24 Eur. J. of Nuclear Med. 1253, 1257-58 (1997).

[117] Kiehl et al., *supra* note 118, at 682.

[118] Symposium, *Will brain imaging be [sic] lie detector test of the future?*, Harv. U. Gazette, Feb. 8-14, 2007 at 1.

[119] *Id.*

[120] *See, e.g.*, George W. Maschke & Gino J. Scalabrini, *The Lie Behind The Lie Detector*, Chapter 4 (4th digital edition), *available at* http://antipolygraph.org/pubs.shtml.

[121] *Id.*

[122] Jennifer Wild, *Brain Imaging Ready to Detect Terrorists, say Neuroscientists*, 437 Nature 457, 457 (2005).

[123] Y. Yang et al., *Prefrontal White Matter in Pathological Liars*, 187 Brit. J. Psychiatry 320, 323 (2005).

[124] T.M. Lee et al., *Neural Correlates of Feigned Memory Impairment*, 28 Neuroimage 305, 311-12 (2005).

[125] A. Kozel et al., *Detecting Deception Using Functional Magnetic Resonance Imaging*, 58 Biological Psychiatry 605, 611 (2005).

the brain were activated when an individual lied. The variations in an individual's brain activity documented during periods of truth telling and lying demonstrate that such differences can be discerned.

These studies, while promising from a neurobiological and research standpoint, can be misleading when applied to the criminal justice system. Most lie detection devices now available, like the polygraph and fMRI, do not detect the lie itself but the anxiety underlying it. Furthermore, the current state of lie detection technology relies upon the subject's attitude toward untruth.[126] The technology may be defeated by people who don't care that they are lying, don't feel that they are lying, or have been trained to lie. Motor movements during the imaging can also obscure the results. There is very little about being in a functional MRI scan that is natural, rendering it difficult to replicate the real world condition of lying during this test procedure.

The ethical implications are even more sobering than the technical complexities.[127] Analysis depends on the type of lie being told, whether it is dangerous or harmless, the likelihood of a false positive result, and the voluntary nature of the scan. Will there ever be a test that can definitively separate truth from deception with good science and legal relevance? Both psychopathy and deception are germane to the criminal justice system. More study and refinement are needed before these techniques can be applied in a non-research context. Although promising, we doubt that the current iteration of fMRI scans could ever be a "stand alone" technology without supplemental evidence.

C. Homicide/Violent Crime

Several studies have demonstrated the presence of a range of neurological and neuropsychological anomalies in subjects with a history of violence or criminality. Lewis and colleagues have done extensive studies on adult and juvenile death row inmates.[128] His work elucidates a number of abnormalities including a high rate of head injury, seizures, and developmental pathology, such as mental retardation and learning disabilities. Raine and colleagues performed studies consistent with Lewis's research, finding a correlation between early neuromotor deficits and violence.[129] These studies demonstrate that some people who commit violent acts have detectable irregularities in their brains. Discerning the causality of different signs and symptoms, however, requires significantly more analysis.

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[126] *Lure Of Lie Detectors Spooks Ethicists*, 441 Nature 918, 919 (2006).

[127] *See id.*; *Neuroethics Needed*, 441 Nature 907, 907 (2006).

[128] *See* D.O. Lewis et al., *Neuropsychiatric, Psychoeducation, and Family Characteristics of 14 Juveniles Condemned to Death in the United States*, 145 Am. J. Psychiatry 584 (1988); D.O. Lewis et al., *Ethics Questions Raised by the Neuropsychiatric, Neuropsychological, Educational, Developmental, and Family Characteristics of 18 Juveniles Awaiting Execution in Texas*, 32 J. Am. Acad. Psychiatry & L. 408 (2006); D.O. Lewis et al., *Psychiatric, Neurological, and Psychoeducational Characteristics of 15 Death Row Inmates in the United States*, 143 Am. J. Psychiatry 838 (1986).

[129] A. Raine et al., *High Rates of Violence, Crime, Academic Problems and Behavioral Problems in Males with Both Early Neuromotor Deficits and Unstable Family Environments*, 53 Archives Gen. Psychiatry 544, 548 (1996).

Raine and colleagues have also done numerous PET scan studies on murderers who plead not guilty by reason of insanity or were found incompetent to stand trial.[130] In one study they examined potential differences between "affective" murderers (those aggressive acts that are defined by impulsive, reactive acts), and "predatory" murderers (where planned and opportunistic aggression is the norm).[131] They posited that limbic structures, those parts of the brain which are more primitive and reactive, might produce the hot-blooded, affective emotions necessary for murder, while the frontal lobes, which house executive judgment and forethought, would be the regions that curtail those murderous impulses.[132] To that end, they proposed that PET would demonstrate reduced activity in the frontal lobes of affective but not predatory murderers.[133] These findings have supported this Article's preliminary theory.

Another study by Raine and colleagues involved PET scans of the brains of defendants found not guilty by reason of insanity (NGRI).[134] The study compared 41 patients charged with homicide and found NGRI to age-matched controls.[135] The NGRI group showed reduced metabolism in the frontal lobe areas as well as other regions that may point to a neurological predilection for violence.[136] Other studies have found changes in the temporal lobes.[137] While PET scans are informative, EEGs demonstrate deficits in places PET scans do not.[138] This comparison highlights the need to appreciate the limitations of each modality and to use varied sources to reach an accurate conclusion from the available data.

VII.CONCLUSION

Ultimately, we encounter the same moral and methodological problems predicting violence with neuroimaging as we do trying to predict violence with other clinical instruments. In most instances, violence is a complex, multifactorial, and socially driven behavior that will likely not be reducible to a unitary brain function or region.[139] Studies on violence are most often retrospective and anecdotal. The gold standard for scientific study is prospective, controlled, and blinded studies, presenting a great challenge in devising methods of prospectively testing violence in an ethical fashion. The Aspen Neurobehavioral Conference, composed of experts from diverse fields such as neurology, neuropsychology, psychiatry, evolutionary psychology, medical ethics, and law, produced a consensus statement concerning the

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[130] *See* A. Raine et al., *Reduced Prefrontal and Increased Subcortical Brain Functioning Assessed Using Positron Emission Tomography in Predatory and Affective Murderers*, *supra* note 116, at 321.

[131] *Id.* at 322.

[132] *Id.* at 329.

[133] *Id.* at 329-30.

[134] A. Raine, et al., *Brain Abnormalities in Murderers Indicated by Positron Emission Tomography*, *supra* note 116, at 496.

[135] *Id.*

[136] *Id.*

[137] Lisa M. Gatzke-Kopp et al., *Temporal Lobe Deficits in Murderers: EEG Findings Undetected By PET*, 13 J. Neuropsychiatry & Clinical Neuroscience 486, 486 (2001).

[138] *Id.*

[139] Brower & Price, *supra* note 2, at 145.

neurobehavioral aspects of violence.[140] They noted that a simple correlation between brain dysfunction and a violent act is rarely possible. "Violence occurs in a social context, and other concurrent factors such as emotional stress, poverty, crowding, alcohol and other drugs, child abuse, and social disintegration of the family are involved."[141]

Data from fMRI, SPECT, and PET scans can be referenced and presented in dazzling multimedia displays that may inflate the scientific credibility of the information presented. Imaging, available in brilliant colors, with its apparent simplicity and vividness, accompanied by exotic names for brain regions, can prove irresistible to many defense attorneys, judges, and jurors.[142] Sophisticated clinicians may present oversimplified, contradictory testimony that may be met with considerable skepticism by judges, juries, and society in general. The psychiatric or neurological expert who uses brain imaging in the courtroom must be careful to acknowledge the limitations of the technology, to resist inflating the meaning of the images, and to be more circumspect and less definitive than retaining counsel might prefer.

Society has not yet reached a consensus as to whether, as a matter of morality or legality, neurological explanations should lead to exculpation. Even if we were to connect the theoretical dots between neuropathology and behavior, this correlation may not be legally or morally relevant. These complicated questions of free will, responsibility, and agency are far from being resolved. No image to date can identify thoughts or ascribe motive. Images cannot distinguish thought from deed and have little, if any, predictive power.

What can we conclude regarding the use of structural and functional imaging technology in the courtroom? Science is so uncharted at present that it is best to limit the legal use of neuroimaging to the elucidation of known structural correlates of neurological insult and the resulting inability to control behavior. These include instances in which the correlation between brain injury and behavior is specifically identified, well researched, and supported by other diagnostic modalities. The technical information derived from a brain image should be given added weight in assisting the fact finder in reaching a decision when it is complementary to other specific and neurologic data presented. Neuroimaging should not stand alone.

Science is not currently helpful in determining structural correlates of predatory violence or cognitive constructs such as psychopathy. The use of this kind of speculative neuroimaging in the courtroom presents methodological and ethical problems that currently outweigh any probative value. We concur with Helen Mayberg, a highly regarded neuroimaging researcher, who in 1992 commented that the current evidence does not support the use of brain imaging technology to "predict neurological, psychiatric, or behavioral deficits."[143] The use of scans to justify behavior after

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[140] Christopher M. Filley et al., *Toward an Understanding of Violence: Neurobehavioral Aspects of Unwarranted Physical Aggression: Aspen Neurobehavioral Conference Consensus Statement*, 14 Neuropsychiatry, Neuropsychology, & Behav. Neurology 1 (2000).

[141] Id. at 2.

[142] Paul S. Appelbaum, *Policing Expert Testimony: The Role of Professional Organizations*, 53 Psychiatric Services 389, 390 (2002).

[143] Helen S. Mayberg, *Functional Brain Scans as Evidence in Criminal Court: An Argument for Caution*, 37 J. Nuclear Med. 18, 18 (1996).

crimes have been committed lead to "unsupportable conclusions [that attempt to] link neurophysiological parameters (such as blood flow or metabolism [in the case of PET scans]) to defendant's judgment, insight, or motives association with a commission of a crime."[144] Such suppositions are generally anecdotal and lack rigorous peer review and scientific evidence.

As recently as 20 years ago, not many could have predicted the current achievements of brain imaging and, in particular, functional imaging. The ability to perform in-vivo clinical-functional-anatomic correlative studies with objectivity and unparalleled sensitivity will create powerful new opportunities and challenges. The next 20 years may well witness even more impressive advances in the understanding of healthy and diseased brain anatomy and its relevance to human behavior. These advances may allow earlier detection of disease, response to injury, and evaluation of therapeutic interventions. Different brain imaging techniques and other modalities may converge in a complementary way. However, in the final analysis, criminal responsibility may be more of a moral question than scientific one. Even with further advances, neuroscience will supplement but not entirely supplant existing criteria of responsibility within moral and legal domains.

Our current attempts to map a brain topography for complex behavior is not unlike the work begun by Gall and Lombroso, albeit couched in far more advanced technological terms.[145] Their efforts were performed with personal and scientific integrity. In fact, Lombroso's study of brain pathology shows remarkable consistency with what we currently know.[146] Scientific communities at the time enthusiastically embraced the works of Lombroso and Gall without a larger perspective on the social implications of this research. The social aspect of both men's ideas was manipulated for non-scientific purposes. History has demonstrated that it is very difficult to distance ourselves sufficiently from the work we do, to appreciate the social context and limitations of the work. In conclusion, we offer a cautionary statement from the late Stephen Jay Gould, the eminent biologist and author: A contemporary note: Lombroso's brand of criminal anthropology is dead, but its basic postulate lives on in popular notions of criminal genes or chromosomes. These modern incarnations are worth about as much as Lombroso's original version. Their hold on our attention only illustrates the unfortunate appeal of biological determinism in our continuing attempt to exonerate a society in which so many of us flourish by blaming the victim.[147] This holds true for neuroimaging as well.

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[144] Soc'y of Nuclear Med. Brain Imaging Council, *Ethical Clinical Practice of Functional Brain Imaging*, 37 J. Nuclear Med. 1256, 1257 (1996).

[145] *See* James R. Blair, *Neurobiological Basis of Psychopathy*, 182 Brit. J. Psychiatry 5 (2003). *See also* T.B. Benning, *Neuroimaging Psychopathy: Lessons from Lombroso*, 183 Brit. J. Psychiatry 563, 564 (2003).

[146] A. Chio et al., *Cesare Lombroso, Cortical Dysplasia, and Epilepsy*, 61 Neurology 1412 (2003).

[147] Stephen Jay Gould, Ever Since Darwin: Reflections in Natural History 224 (W.W. Norton & Co. 1977).