

Guide to Mental Health Mitigation

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GUIDE TO MENTAL HEALTH MITIGATION

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1. Introduction

Through an often-painful process of trial and error over the past thirty years or so, capital defense attorneys have learned that we, as lawyers, have primary responsibility for ensuring that our clients' disabilities and impairments are accurately identified and understandably explained. Winning the right to state-paid expert assistance for our almost-always-indigent clients was a crucial step in this process. *Ake v. Oklahoma*, 470 U. S. 68 (1985). But recognizing the issues that require expert assessment, and identifying the kinds of expertise that a given case requires, is the job of defense counsel: it can't be delegated to anyone else. And this means that defense counsel must have a basic knowledge of mental health issues, and of the myriad physical and psychiatric conditions that can and do afflict our clients.

Part of our job, of course, is to learn our clients' stories in great detail, and from as many different sources as possible. But another part involves recognizing the possible meanings of what we are uncovering as we probe. For example:

- When we find episode after episode of our client's bad judgment, impulsivity and a lifelong pattern of seeming inability to learn from his mistakes, are we to conclude (as have most of the teachers, social workers, police and probation officers, prosecutors, judges, and even defense attorneys that the client has encountered up until now) that's he just an exasperating and self-centered person? Or might we actually be witnessing the behavioral stigmata of a biological catastrophe—prenatal exposure to

alcohol—that occurred before he was even born, as innocent and as helpless as a human being can be, and that left him to cope (or not) with undiagnosed Fetal Alcohol Effect for every minute of his life on earth?

- When three sets of juvenile and adult prison mental health professionals labeled the client with the diagnoses of conduct disorder and antisocial personality disorder, were they describing the client as he was, or had they simply failed (due to lack of information, time, care, or objectivity) to recognize that the client suffered from an organic mental illness such as childhood bipolar disorder, or was exhibiting symptoms of untreated post-traumatic stress disorder? Or both?

These examples could be multiplied endlessly. The point is simply this: a mental health professional is unlikely to answer these questions accurately and reliably unless we, as lawyers, know to ask them.

And that is the point of this Guide. It is designed as an overview and introduction to the mental health issues most often encountered in the process of understanding our clients and telling their stories. It is meant as a gateway for attorneys into specific issues that are important as mitigation, any one of which should be pursued through outside sources and experts if it is relevant to your client.

This Guide cannot stand alone. It is intended to provide people involved in capital defense a doorway through which to gain access to and begin to develop necessary

knowledge about some of the issues that are important to mitigation. Specifically, this Guide focuses on issues that have a direct connection to mental health impairments and disorders. It recognizes that each of our clients are charged with conduct that lies at the outer extreme of human behavior, and it assumes, as a plausible working hypothesis, that each client has impairment in some realm or another.

This is a hypothesis which your investigation will explore and test as your work on the case progresses. Figuring out the specific what, how, and why of your client's impairment is the most difficult task faced by counsel, a task that requires you to research and study scientific and mental health issues that may be new to you and difficult to understand. This is an unavoidable part of the terrain in defending people charged with capital crimes. Because mental illness usually cannot be detected and understood by untrained people, the first task in developing mitigation is not to prematurely reach conclusions about our clients' functioning, to continue spending a lot of time with your client while collateral information is developed, and to allow your hypotheses to grow and change as you gain more information. Your hypotheses about the what, how, and why of your client's impairment will be tested repeatedly as you gather information. Some initial ideas will be ruled out by the evidence you gather, some may be modified and sharpened, and some new hypotheses will develop that you did not foresee. At the heart of this effort is the goal of understanding your client as fully as possible. How your client's brain works and the social context that has shaped his or her behaviors – both chosen and not chosen – is a critical component of the inquiry into, and shaping of, mitigation.

This Guide focuses on behavior. As a consequence, it looks closely – and sometimes technically – at the processes that drive behavior: the human brain and the disorders of the brain which affect behavior. The Guide's technical sections may be frustrating to read at first. But their purpose is to provide in one place a wide range of materials and ideas that have proven useful in cases over many years of collective experience; to provide capital counsel with better tools to prepare the investigation, choose experts, and talk to experts rather than defer passively to experts; to provide a baseline from which to recognize areas of further scientific and medical research with which defense teams must become familiar; and, to allow defense teams to better develop hypotheses about impairments that may be present in a client's case and to go about investigating those hypotheses.

Each section of this Guide draws on social science and medical knowledge that is current and litigation ideas and themes that are well-established in order to assist you in the preparation and litigation of your case. Although often discussed in terms of mitigation, these issues may apply equally to guilt phase defenses, competence to stand trial, the admissibility of your client's statements, and many other matters that have to do with how your client perceives and interacts with other people and with his surroundings.

As should become clear throughout, it is not possible to begin your investigation into these issues too early, nor can you delegate responsibility for developing mitigation. Capital cases require teams of people to effectively develop and present the facts necessary to achieve a verdict less than death. We hope this introduction to the mental health

issues most often encountered in capital cases will assist you and your team as you begin this process.

2. Record Gathering and Mitigation

Gathering records is a critical step in every case, but in capital cases it takes on a scope and importance not usually found in non-capital cases. Historical records can often be the difference between winning and losing a case. In a capital case, record gathering is often described as one of the most significant factors in how a defense team must prepare a case. Social and family history records may answer many questions which are otherwise difficult to deal with:

- a) when did your client first begin to have certain types of symptoms or display certain types of behaviors?
- b) what mental or physical illness symptoms are documented and diagnosed prior to your client's indictment?
- c) what mental or physical illness symptoms did the client's family members have that helped shape your client's life?
- d) what other family members have shown symptoms or been found to have medical or psychiatric conditions?
- e) what witness, who knew the client before his/her arrest, can come testify about specific parts of the client's life?
- f) how can you prove that your client is not malingering symptoms of mental illness that he/she currently appears to have?
- g) what evidence, other than people who

know your client, will your expert rely on in forming an opinion?

As important as being prepared to answer these questions is, it is also from these records that you will begin to develop the picture of the story that you are going to tell. Records shape the story you can tell and provide you external corroboration to support the reliability of that story as told by witnesses and the records. Records are the foundation upon which a competent and thorough social history is built.

In capital cases, you must start with the goal that your team will gather every piece of paper, no matter how irrelevant you might think it will be, that relates to your client and your client's family. Sometimes, this will seem futile, as when you are waiting for the third time at the School Board office seeking records you have been told do not exist. However, going in person to obtain records often is the difference between getting them and not getting them, and having a particular document may be the difference between life and death.

Among other issues recently addressed in *Wiggins v. Smith*¹ is that records open the door to further investigation. Social and family history records will include the names and identifying information for potential witnesses as well as lead you to further records and a better development of your client's story.

The first step in getting records is to obtain a signed authorization for the release of records by the individual who has the authority to get you access to that record. As

¹ 539 U.S. 510 (2003).

a result of HIPPA, the requirements for what the release says may be different for different types of records, but sample releases are available from the Project.

Most importantly though, there is no short-cut or checklist of records which can lead you to the answer - it is through a process of no stone unturned record gathering that you may find just the piece of paper that leads you to a witness who tells your client's story and saves his/her life.

Client and client's family records that offer insight into and evidence of mental illness include:

- adoption records
- bankruptcy filings
- birth records
- civil commitment records
- civil and criminal court files for multiple generations of family
- death records (death certificates, autopsy and coroner reports)
- dental records
- employment records (social security, performance evaluations, medical evaluations, etc.)
- family court records
- foster care records
- immigration applications and documents
- jail and custody records
- juvenile court and juvenile prison records
- law enforcement and police contact records (local police departments, sheriff's departments, BOP, FBI, DEA, INS, Border Patrol, etc.)
- marriage certificates
- medical records (clinics, hospitals, family doctors, tests, reports, etc.)
- mental health reports (tests, raw data, reports, doctor notes, etc.)
- military records
- prison records
- probate records for family members
- probation and parole records
- records on prior offenses (including prior attorney files)
- school records
- selective service records
- social service agency records (food stamps, disability, AFDC, food stamps, WIC, etc.)
- Social Security earned income reports
- unemployment records
- worker's compensation records

3. Overview of brain-behavior relationships

The relationship between the brain, brain functioning and behavior is extremely complicated but some working knowledge of the relationship is important for the defense team to competently investigate and prepare. Unfortunately, there is no simple way to provide an overview of the brain, its parts and how they work. This chapter is technical and difficult to read, but contains information that is the scientific foundation for the rest of the workbook which is less technically presented. This chapter is included here because, while “complexity” is not compelling in a jury presentation, the defense team, including counsel, must have a substantial understanding of the relationship as it plays out in your case so that the team can identify, work with and prepare, and most effectively use lay witnesses and experts about how the pieces fit together to make up the story. Defense teams must make the effort to understand meaningfully the technical and scientific underpinnings of mental illness and brain functioning so that they can work effectively with experts and be able to translate the information for a lay jury.

The overview that follows seeks to provide a baseline of technical information much of which can be further researched in numerous textbooks. It attempts to set out the framework in which behavior can be scientifically conceptualized and to provide the framework in which your investigation will take place. Chapters that follow refer back to some of this information.

The adult human brain, of course, results from an extended developmental process that begins during the third week of gestation, just after conception. The precursor cells that

initiate the developmental process that leads to the adult brain are present just after conception, which helps explain why some disorders and diseases, such as fetal alcohol syndrome, begin in utero.

Brain cells, known as neurons, begin to coalesce and develop into the central nervous system approximately the end of the first month of gestation; this process of coalescing and developing of neurons into the central nervous system lasts until the sixth month of pregnancy. Each neuron has an axon which transmits information to other neurons and a dendritic tree which integrates information coming into the neuron from thousands of other neurons. Each dendritic tree can have several thousand branches, each connecting to other neurons, muscles or glands at a synapse. A synapse is the junction between one neuron and the next, and also between a neuron and muscle. These three components form the primary structural building blocks of the brain.

Information is transferred across the synaptic gap by means of neurotransmitters which are chemical agents that pass across the synaptic gap between neurons. The neural structures which connect neurons (axons) are covered by a fatty tissue sheathing, known as myelin, which facilitates passage of electrical signals. The linkages between neurons change over time by a constantly occurring, natural process of reorganization, with some connections being culled and new ones being built. Similarly, neurotoxic exposure or any of a number of injuries or insults to the brain may cause permanent damage to the connections within the brain’s structures. Thus, injury or damage to any of these component parts of the brain might result in behavioral abnormality.

The central nervous system comprises the spinal cord and brain; the peripheral nervous system comprises the nerves that run throughout the rest of the body, carrying information back and forth from the body to the brain. Most of the attention here is on the central nervous system except for instances where symptoms are most easily seen in the peripheral nerves.

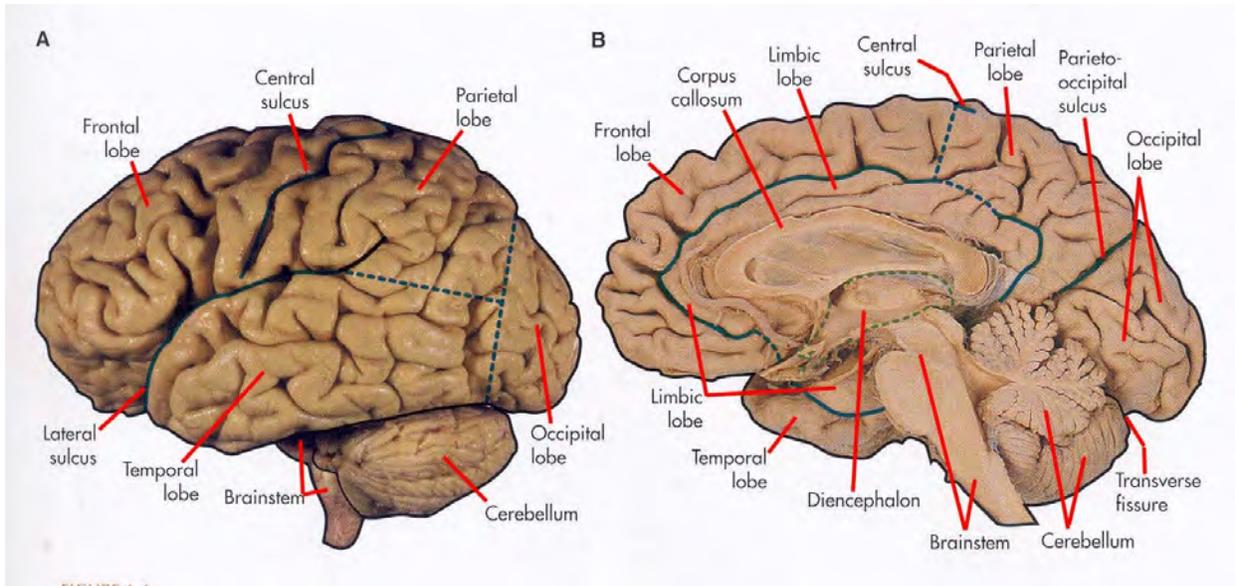
A way of viewing the brain-behavior relationship is that the normally functioning brain creates an action (e.g., speaking, moving, acting, thinking), interprets the response in the environment to that action, and then adjust or acts again as that sensory information that comes back into the brain. In the scientific literature, sensory input caused by our own actions is termed refference; sensory input caused by the external environment is termed exafferance. As refference or exafferance comes back to the brain through the senses, the person adapts to that new information and adjusts. For instance, if you feel hungry, a sensation triggered by the central nervous system, you might go to your refrigerator and open the door. Depending on what you see, you might eat something. If you open the refrigerator and it is empty, you might look somewhere else for food. This is a sensory-environment feedback loop.

As common as this process is, happening for even the most minute of actions, it is also extremely complex. Every behavior involves this complex neurochemical and neuroelectrical process that draws on many parts of the brain. It is less the size of the brain (an average adult brain weighs 1,400 grams) than the complexity of the connections among the various parts of the brain that allows the normal human to function and

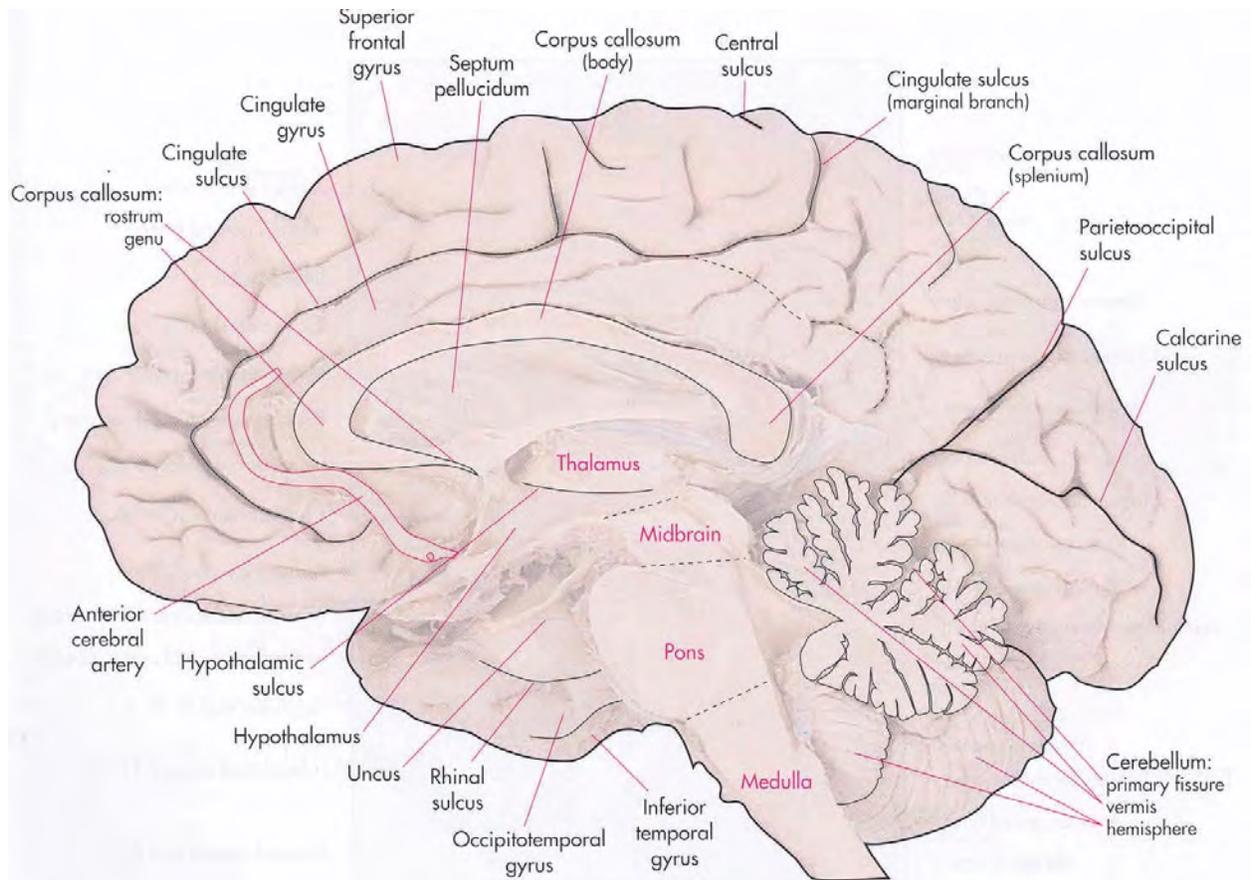
behave. Although too complicated to address here, an overview of how the brain performs, especially this conception of a never-ending interaction between the organism and its environment, is the scientific basis for nearly all mitigation evidence and investigation in one way or another.

The reason to have some insight into the complexity of this process is that at every point, a minor defect or impairment can result in dramatic behavioral maladaptation. Physical, chemical, or electrical impairments in the brain can so dramatically alter this process as to lead to seriously disordered behavior.

Below are some overview images of the brain:



Lateral and medial view of brain. Reprinted with permission from Nolte and Angevine (2000) *The Human Brain in Photographs and Diagrams* 2nd ed., St. Louis: Mosby, Inc. p.1



Medial view of sagittally hemisected brain. Reprinted with permission from Nolte and Angevine (2000) *The Human Brain in Photographs and Diagrams* 2nd ed., St. Louis: Mosby, Inc. p.8.

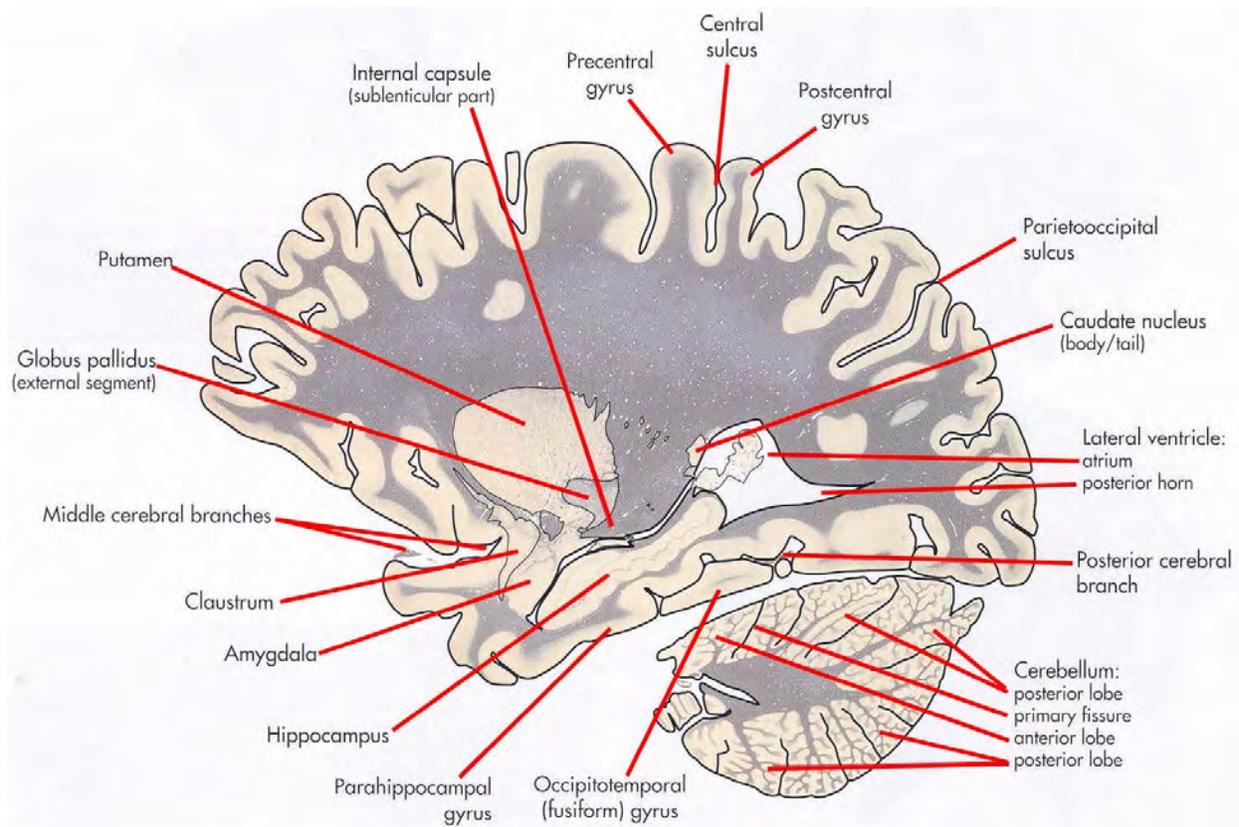
Although different areas of the brain are said to control various functions, it is still important to remember throughout this discussion that the brain works as an integrated system: the various pieces of the brain reach out to and connect with the entire organism and other parts of the brain, processing information both coming in and going out through an intensely complex array of interactions within the brain.

A. Some Key Parts of the Brain Structure:

The medial views of the brain (above) shows many primary parts of the brain. The hindbrain consists of the brain stem, cerebellum and the pons. These are the oldest (in an evolutionary sense) parts of the brain, and link the rest of the nervous system to the brain. They play important roles in the most primitive functions of the brain (e.g., blood pressure, respiration, heart beat, mediates posture, motor reflexes, coordination and modifying output from other parts of the brain), control alertness and have some role in sensory processing and perception.

Just above the hindbrain are a series of structures that play critical roles in memory, sensory perception, motor function, arousal, attention, autonomic functions, emotional expression and seem to play the central role in integrating the flow of information within the brain (thalamus and hypothalamus). Also in this area are the basal ganglia which are the base of the cerebrum, the most evolved and largest part of the brain. Memory, learning, cognitive flexibility, emotional state and mood disorders occur when there is damage to the basal ganglia. These behavioral changes can

also result from disruptions to the control and flow of information. The basal ganglia affect complex motor functions and reach from the cerebrum to the frontal lobes. The corpus callosum (above the basal ganglia) connects the left and right hemispheres of the brain and is crucial to the communication between the hemispheres. The cerebral cortex is the outer layer of the brain and appears to be involved in mediating most complex behaviors.



View passing longitudinally through the hippocampus. Reprinted with permission from Nolte and Angevine (2000) *The Human Brain in Photographs and Diagrams* 2nd ed., St. Louis: Mosby, Inc. p.94

The frontal lobes compose about a third of total brain area in humans. Just behind the frontal lobes is the motor cortex which controls complex motor activity and reflexes. Just behind the motor cortex is the parietal lobe which includes the somatosensory cortex. The back of the brain holds the occipital lobes.

Occipital lobes: The occipital lobes are involved in visual perception, recognition of emotional state, inability to recognize objects, inability to recognize faces or emotional content of faces.

Parietal lobes: The parietal lobes are responsible for processing touch and the

integration of visual, tactile and auditory input. These lobes also relate to drawing, writing and constructional tasks.

Temporal lobes: The temporal lobes contain the primary auditory functions, but also play a critical role in language formulation and comprehension.

Frontal Lobes: The frontal lobes control executive functions: inhibition of movement and behavior, judgment, planning, assessing options and consequences, intentionality, complex decision making. Executive functions are sometimes defined as real-world adaptability. They are the newest evolutionary part of the brain and have the greatest number

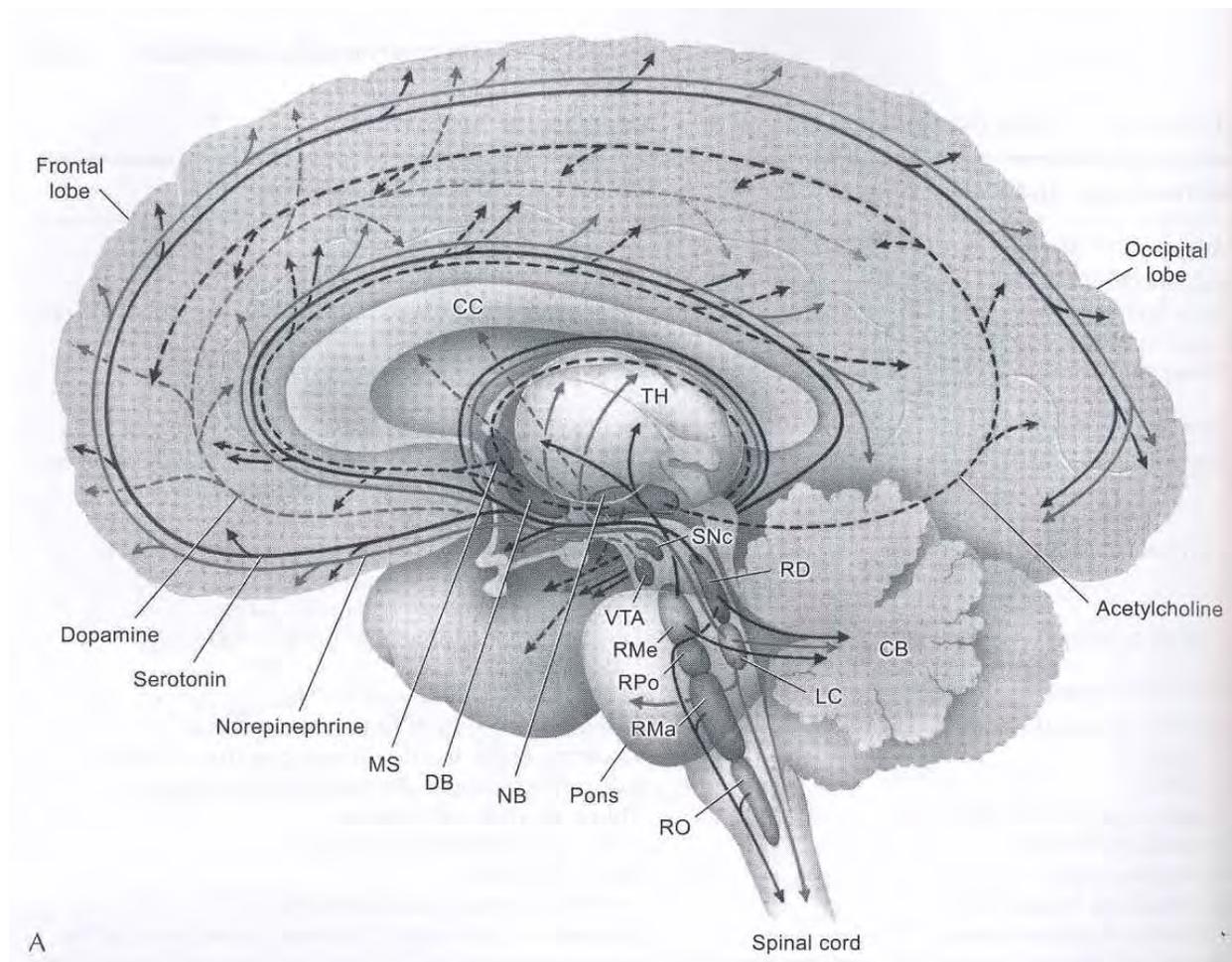
of connections to other parts of the brain. The frontal lobes are also involved in motor functions, language processing, and mental flexibility (initiating, stopping and adjusting behavior). The frontal lobes are the part of the brain that most distinguishes humans from other mammals.

Limbic System: The major structures of the limbic system are the hippocampus, the amygdala and the uncus. In brief, the limbic system has everything to do with emotional responses to sensory stimuli, links perception and memory, encodes visual and auditory sensations into the memory, encodes emotional tags onto memories, retrieves memories, and originates protective drive states (fear, fight or flight, autonomic responses to perceived danger).

All of these parts of the brain rely on neurochemical and neuroelectric mechanisms to communicate and interact. This makes the

neurochemicals of the brain crucial to the normal functioning of the organism. When a signal is sent from one part of the brain or body to another, it is transmitted by electrical activity through nerves or neurons, but where the message needs to be continued on to the next nerve, muscle or neuron, the signal is converted to a chemical (in the brain, a neurotransmitter) and passed on (across the synaptic gap), where it is re-converted to an electrical signal by the next neuron. For even the most minor or common action, this process occurs.

As mentioned above, disruptions in chemical or electrical systems in the central nervous system, including over- or under-abundance of neurochemicals, can result in serious disordered behavior. An overview of the chemical systems and the primary pathways these neurochemicals move through are below:



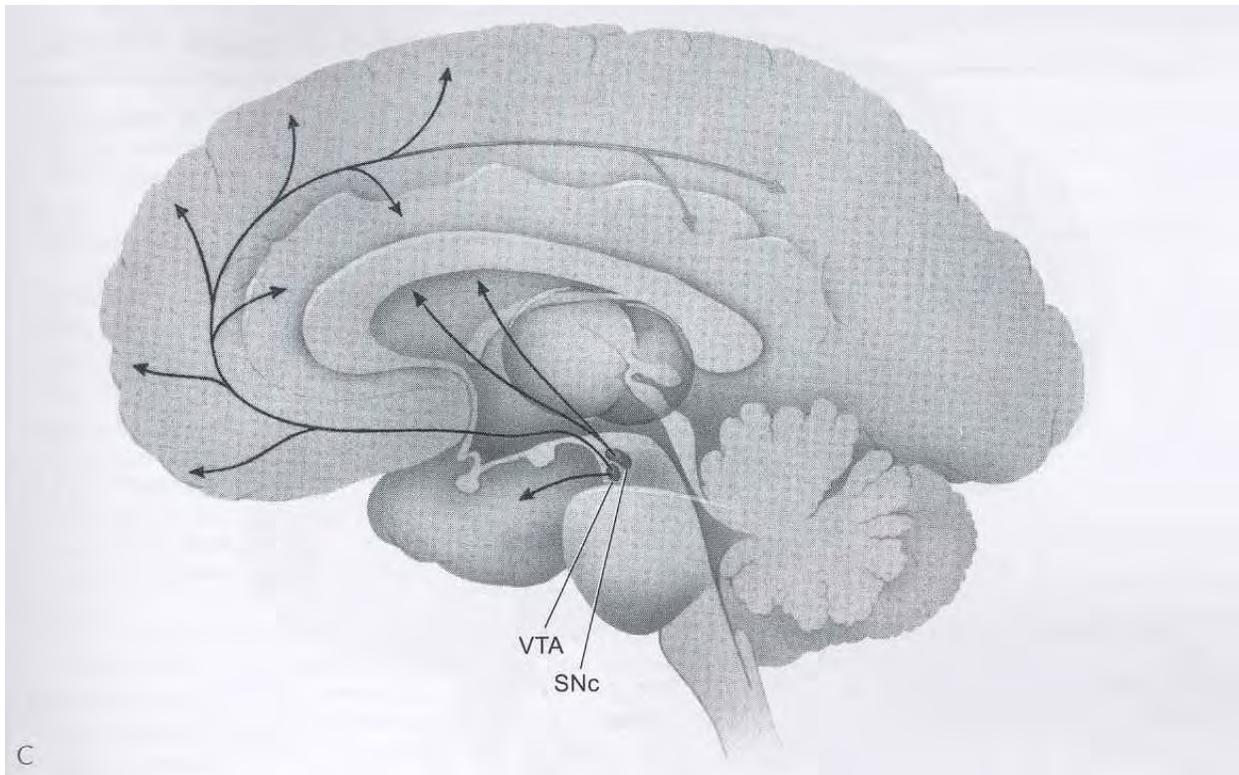
A
Neurochemical pathways in the brain. Reprinted with permission from Devinsky and D'Esposito (2004) *Neurology of Cognitive and Behavioral Disorders*, Oxford: Oxford University Press, p. 106.

Some key neurochemicals which are recognized to directly affect behavior are:

Serotonin (5-HT): which is ubiquitous in the brain, although receptors are more densely distributed in basal ganglia, amygdala and hippocampus. Accumulation of serotonin may be involved in impulse control disorders. It also appears involved in psychotic disorders and depression.

Dopamine: which is especially prominent in the limbic system and basal ganglia (e.g.,

death of dopamine neurons in the basal ganglia appears to cause Parkinson's disease). It has a role in regulating neuroendocrine secretions, regulation of locomotor activity, emotion and affect. Dopamine receptors are the primary target for many antipsychotic medications known as neuroleptics. Note that the primary dopamine pathway runs through the frontal lobes.



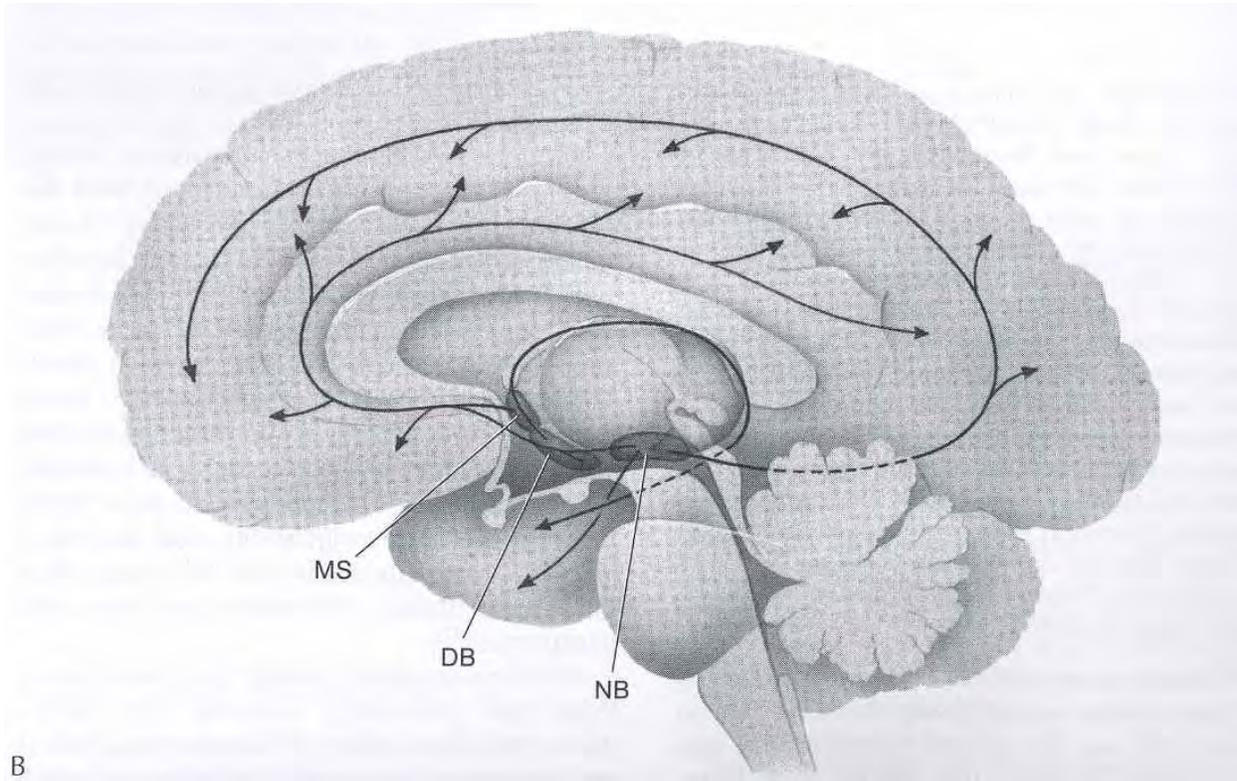
Dopamine pathway. Reprinted with permission from Devinsky and D'Esposito (2004) *Neurology of Cognitive and Behavioral Disorders*, Oxford: Oxford University Press, p.107.

Glutamate: which is especially prominent in the frontal lobes. Glutamate is regarded as an excitatory neurotransmitter. When dopamine is reduced, glutamate pathways become hyperactive.

role in stimulating brain maturation.

Acetylcholine: which is especially prominent in the limbic system (especially the hypothalamus) and reaches into the frontal lobes. Cholinesterase inhibitors (see Pesticides Section 5c below) administered directly to the brain cause otherwise docile animals to exhibit aggressive and predatory behavior.

GABA (gamma aminobutyric acid): which is also ubiquitous in the brain and central nervous system. It is especially prominent in the frontal lobes. GABA is generally an inhibitory neurotransmitter, but it also plays a



B
Acetylcholine system. Reprinted with permission from Devinsky and D'Esposito (2004) *Neurology of Cognitive and Behavioral Disorders*, Oxford: Oxford University Press, p.106.

B. More specific overview of selected parts of the brain

1) *The Frontal Lobes*: Elkhonon Goldberg, a well-known neurologist has said: “The frontal lobes are to the brain what a conductor is to an orchestra, a general to an army, the chief executive officer to a corporation. They coordinate and lead other neural structures in concerted action. The frontal lobes are the brain’s command post.”² This is why so much attention in capital cases focuses on the functioning of the frontal lobes. The frontal lobes perform the tasks with which the law is most concerned.

The neural developmental process in humans leaves the frontal lobes till last. Thus, myelination (the process by which the connective structures of the neurons are coated with myelin to allow for proper electrical conduction) does not complete in the frontal lobes until early adulthood (around eighteen to twenty years old). Until completed, the frontal lobes do not work efficiently or properly (hence, the erratic and disinhibited behavior of teenagers).

The frontal lobes appear highly involved when a person is given a novel stimulus, but when that stimulus is repeated (e.g., when a person is familiar with a task), the frontal lobes no longer activate.

² Goldberg, E. (2001). *The Executive Brain* New York: Oxford University Press at p.2.

Current research has identified seven

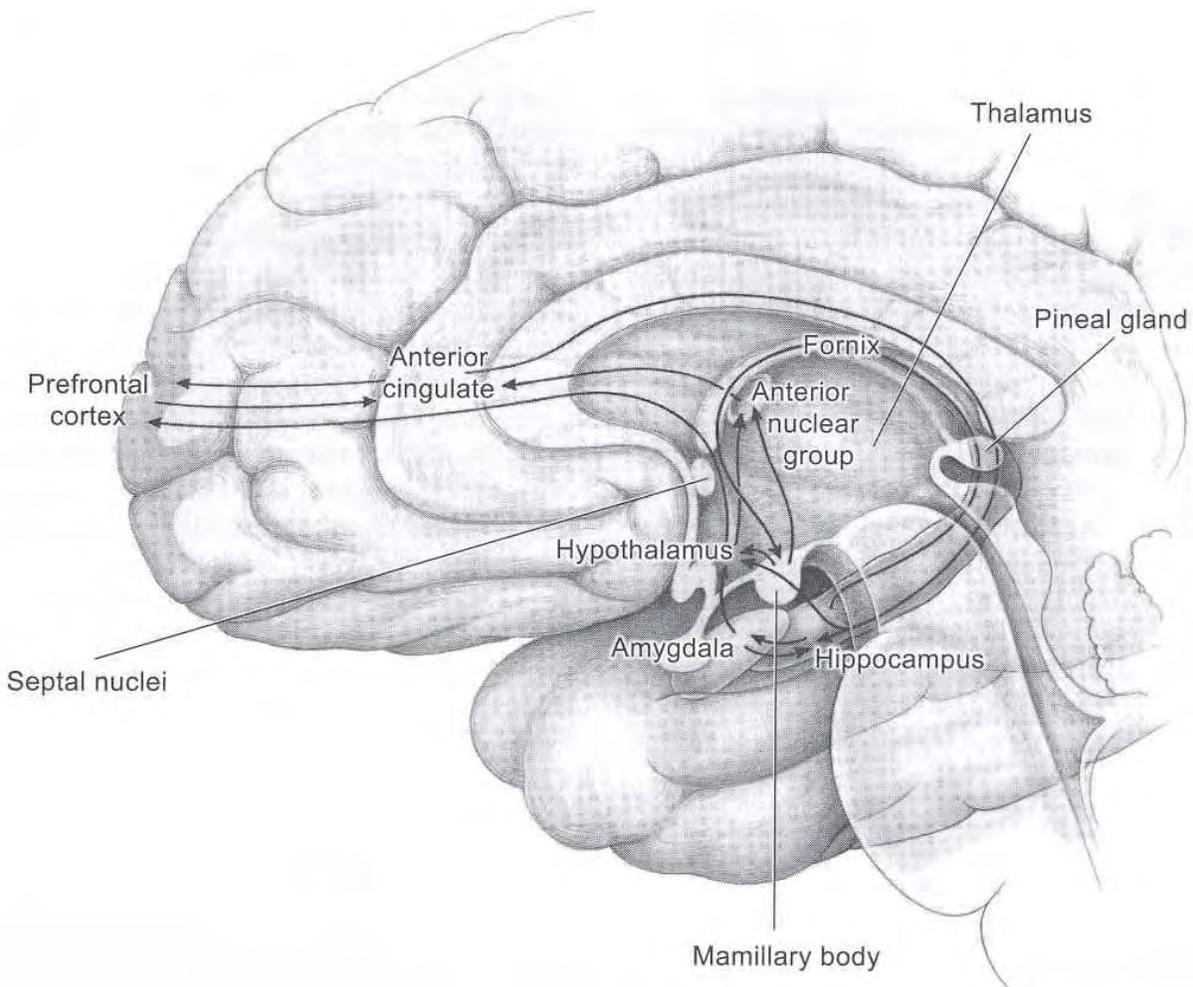
frontal-cortical circuits. These circuits are basically information/stimuli loops within the frontal lobes that take in information from all regions of the brain, evaluate it, and send output signals back. The circuits are the mechanism by which the frontal lobes regulate behavior. Dysfunction in one of these loops causes very specific behavioral deficits, although your task is really to document frontal lobe damage rather than specific damage to a loop. Nevertheless, the importance of the seven loops helps to explain why people with frontal lobe damage do not all exhibit exactly the same behaviors. Variations in behavior will occur depending on which loops or portions of the brain the loop incorporates have suffered damage or are no longer functioning properly.³ Similarly, some psychiatric illnesses appear to be circuit related as well (for instance, mania and

psychosis).

In terms of behavior, it is clear that frontal lobe dysfunction is related to attention and working memory, difficulty shifting attention from one stimulus to another, reduction of memory span, deficits in the ability to self-monitor, diminished planning and problem solving ability, inflexibility in thinking, inhibition of behaviors, and visuospatial impairments.

2) *The Limbic System*: The components of the brain that make up the limbic system are, in an evolutionary sense, some of the oldest in the brain. The limbic system helps to regulate emotion, memory, motivation, instinctual behaviors and social relations, and therefore, disorders or damage which make this system unbalanced can have dramatic behavioral manifestations. The limbic system is also physically and chemically “wired” to the frontal lobes.

³ Lichten, D.G. and Cummings, J.L. (2001). Frontal-Subcortical Circuits in Psychiatric and Neurological Disorders New York: The Guilford Press.



Principal structures of the limbic system. Reprinted with permission from Devinsky and D'Esposito (2004) *Neurology of Cognitive and Behavioral Disorders*, Oxford: Oxford University Press, p.334.

The hippocampus and amygdala are particularly important in the coding and retrieval of emotional memories. That means that this region of the brain is especially affected by exposure to traumatic events. Brain imaging of people with PTSD suggest that they have decreased hippocampal volume (smaller brain mass) and excessive amygdala activation. This likely explains the persistent fight-or-flight responses and the host of other behavioral changes seen in people with PTSD.

The hippocampus is one of the locations where perception and memory interact. The hippocampus is involved in memory storage and temporal dating of the memory. Verbal memory, visual memory, auditory memory, recognition and recall all appear to rely on a properly functioning hippocampus.

The amygdala is involved in object recognition, sense of smell and provides an emotional tag to the memory that is stored. It

is also directly involved in many core survival mechanisms. Perhaps more importantly for trauma and behavior though, the amygdala is critical for fear conditioning, such that threatening stimuli are coded in the amygdala and re-exposure to them prompts heightened response. The amygdala also plays a critical role in determining the emotional content of visual information and interacts with the frontal lobes in responding to that stimulus.

C. Finally, there are basically four types of injury or causes of damage to consider when developing your case in mitigation.

1) *In utero injury*: innumerable injuries or illnesses can occur during gestation, some of which cause developmental changes, others of which cause malformation of the central nervous system. For clients with little or no prenatal care, records which document such problems may be limited, but certain types of exposure to toxins (e.g., alcohol, pesticides, metals) can be proven by careful interviewing and record gathering.

2) *Genetic predispositions*: we are learning more about how to recognize diseases which have genetic components to them, and some central nervous system illnesses are now well-recognized to have genetic components (e.g., schizophrenia, alcoholism). These must be proven by multi-generational family history and record gathering which can be used to demonstrate the patterns of illness across the generations.

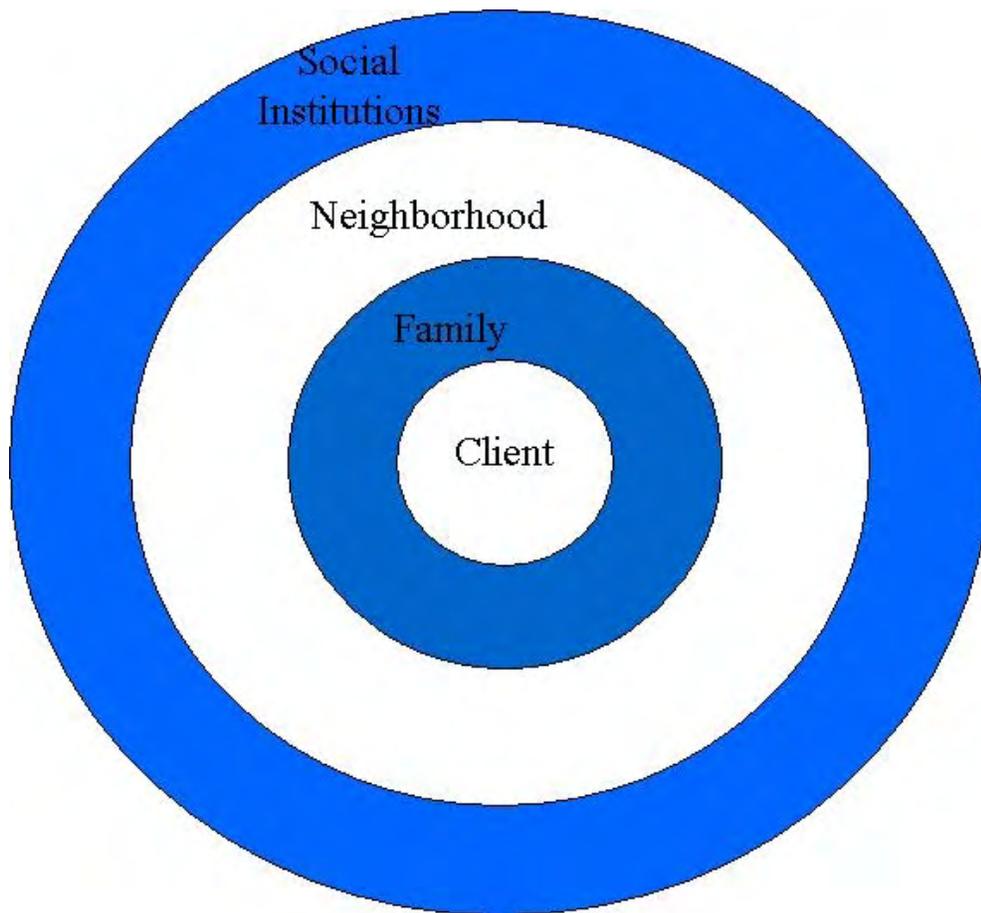
3) *Physical injury*: both intentional and unintentional are quite common. Again, medical record availability will depend on access to health care, but interviewing will certainly assist in obtaining corroborative information on physical injuries.

4) *Toxicological injury*: includes intentional and unintentional exposure to chemicals or agents which alter central nervous system function (e.g., lead, alcohol). Exposures may be community-wide or individual.

4. The brain-behavior relationship in social context

The chemical, electrical and structural processes of the central nervous system play out in social context and therefore will have to be understood in that social context. The easiest model to conceptualize the social context of behavior comes from social science research and is known as the ecological-transactional model.⁴ This model (despite its

name) is a straightforward way of organizing both your investigative tasks and your presentation to the jury.



Ecological model adapted from Bronfenbrenner 1979.

⁴ Bronfenbrenner, U. (1977). The Ecology of Human Development Cambridge, MA: Harvard University Press.

The advantage of adopting well-recognized scientific models such as this is that you will have an additional tool for defending your expert's opinion and your themes to the finder of fact. This research is important for a number of reasons, not the least of which is that it helps you answer the question of why your client did something that others with a similar experience did not do. Your goal in answering such a question (lots of people are abused, they don't all kill someone) is to differentiate your client from the general pool of people by demonstrating differences in both the quantity and quality of factors. You want to tell the story of your client's life in the context of his/her family, neighborhood and interaction with social institutions, each of which was a piece of shaping and narrowing the path which was available for your client.

The ecological-transactional model helps you do this by placing your client within his/her family dynamic in the neighborhoods he/she was raised and lived in, examining the social environment of his/her life and the social institutions with which he/she had contact, and helping you consider how each of these parts of your client's life was affected by the other parts. This in turn helps you to investigate and prove how each of your client's experiences in the world narrowed the available options your client had and how others with some similar experiences had some area of life that allowed that person, unlike your client, to escape.

As the diagram above shows, the model consists of four levels: individual, family, neighborhood, and institutions.

This model helps you keep information organized in ways that is useful as your case develops and more information is gathered.

For instance, some people get confused as to whether an abused child (and the symptoms exhibited by an abused child) is about the child's behavior or about the family dysfunction including abusive caregivers. The ecological model can help you organize your thinking and your evidence about the relationship between the child and his/her family, without having to attribute family dysfunction to the child who is least responsible for it and least able to alter it. Child abuse can be understood and presented as the responsibility of the abuser rather than the abused.

The ecological transactional model is not just an information organizing tool however. It is a well-established social science theory which has extensive theoretical and methodological support. Here, we present an outline of selected mitigation-related issues and where within an ecological model they belong:

A) *Individual*: the individual issues are the most obvious in litigation. Much of the rest of this mitigation workbook focuses on individual issues. The prosecution seeks to shape all the evidence to be about choices that your client made as though he/she chose from millions of options, free to do as he/she pleased. You will be situating your client's behavior within a framework that allows the jury to understand how your client ended up on the path he/she did and how at every step along the way, options were limited and shaped by both individual factors (e.g., his/her neurologic and psychiatric functioning) as well as family, neighborhood and social institutional factors.

B) *Family*: The influence of families on children is significant, especially when that influence is predominantly negative. Research

on the role of family factors in juvenile delinquency began at least seventy years ago.⁵ Research has identified a host of factors that are important in shaping how children behave. Some of these are whether the child: witnesses family violence, is physically or sexually abused, is subject to abandonment or neglect (e.g., adopted or placed in foster care or simply ignored in the home), or exposed to substance abuse by family members (e.g., sees its use, is introduced to using).

The key to understanding family dynamics will be good interviewing techniques which must start from the premise that open-ended questions will gain information no one could ever have thought to ask. Keep in mind that although you may be asking about your client's childhood, the family dynamics are still active and each member of the family may still have relationships to protect or emotionally charged relationships that still shape how they act. You are seeking information about both specific incidents that occurred as well as trying to gain an understanding of how the family unit functioned.

One other family issue which gets less attention is parentification. Parentification is a clinical term which refers to a situation in which a child must take on the responsibilities of an adult care-giver, either because of abandonment or illness usually. There is a large amount of research on the psychological harm this causes the child who must take on the role of parenting.

C) *Neighborhood/community*: An increasingly strong body of research points to

⁵ Healy, W. and Bronner, A.F. (1936). New light on delinquency and its treatment. New Haven, CT: Yale University Press.

“social cohesion” or “community efficacy” as a critically important factor in determining the potential for criminal activity. Collective efficacy is defined as working trust and shared willingness and capacity for people in a neighborhood to intervene informally (exercise non-coercive, informal social control) in neighborhood activities to promote social good. Neighborhood research has found that concentrated disadvantage and residential instability explain 70% of neighborhood variation in how willing people are to help their neighbors, intervene on their behalf or protect other people's children.⁶

A recent summary of neighborhood research finds that

the evidence is solid on the ecological differentiation of American cities along socio-economic and racial lines, which in turn corresponds to the spatial differentiation of neighborhoods by multiple child, adolescent, and adult behaviors. These conditions are interrelated and appear to vary in systematic and theoretically meaningful ways with hypothesized social mechanisms such as informal social control, trust, institutional resources and routines, peer-group delinquency, and perceived disorder. An important take-away of our assessment is that these and other neighborhood-level mechanisms can be measured reliably with survey, observational, and archival approaches.⁷

⁶ Sampson, R.J., Raudenbush, S.W. and Earls, F. (1997). Neighborhoods and Violent Crime: A Multilevel study of collective efficacy Science 277:918-24 (Aug 15).

⁷ Sampson, R.J., Morenoff, J.D. and Gannon-Rowley, T. (2002). Assessing “neighborhood effect:” Social processes and new directions in research Annual

Neighborhood processes can and should be treated as ecological or collective phenomena rather than as individual-level perceptions or traits. Collective efficacy is a measure of informal social control and mutual dependence, where people believe that members of their community will assist them when they are in need. For instance, a community survey found that where there were lower than expected rates of child abuse there was higher reported satisfaction with neighborhoods.⁸ They suggested that neighborhood, and community member perception of their neighborhood, was an important factor in child maltreatment. Fundamental isolation is theorized to be the inverse of community efficacy, with families and individuals isolated from support. In practice this means that families and individuals who are fundamentally isolated are separated from neighborhood institutions and the mechanisms of informal social control. They doubt that the neighborhood will act to assist and protect its members.

Thus, key neighborhood issues include: social isolation, both of the family within a geographic region but also of the neighborhood itself, sometimes caused by language differences or the length of time since immigration and/or acculturation processes; density of poverty and disadvantage, because it appears to matter an enormous amount on behavioral outcomes if a poor family also resides in a neighborhood where everyone else is poor; collective efficacy; exposure to community violence; proximity to neurotoxin producing facilities

Review of Sociology 28(1) 443-78.

⁸ Garbarino, J. and Sherman, D. (1980). High-risk neighborhoods and high-risk families Child Development 51:188.

and exposure to toxins; and access to resources, such as public transportation or medical care.

D) *Social Institutions*: The threshold question about the functioning of a social institution is: can and does the institution identify, within its particular expertise, those people to whom it is charged with providing services. For instance, if a person went into a police station and reported that he had just committed a crime, it seems reasonable to expect that the police would recognize that dealing with this person fell within their socially defined and expected role. The secondary question is can and did the institution provide adequate and competent services. For each social institution that your client came into contact with, you will want to assess these questions.

A third question about social institutions is whether, within the institution or in your client's attempts to gain access to the institution, barriers to equality existed. For instance, a person denied a job on the basis of race is denied equal opportunity. This is an institutional barrier to equality because the discrimination is acted out against an individual, but exists as a result of bias against a class of people which the institution enforced.

The social institutions with which our clients most come in contact are juvenile detention and foster programs, schools, medical and psychiatric institutions, and prisons. The barriers to equality of opportunity and outcome are most usually the result of race discrimination and poverty.

Race and culture: This model is a standard sociological model which can assist in understanding and structuring both the

investigation and the presentation of your client's story. Race and cultural issues cut across each of the four levels of the model, acting in specific instances on the daily life of your client. Cultural issues may help explain specific behaviors, views of the world or the client's relationship to other participants in the crime. For instance, some clients have deeply held cultural views about opening the family to outside view or presentation and may at first be hesitant to allow mitigation investigation. Cultural experts can be helpful to defense teams in planning how to deal with some cultural issues as they relate to working with your client. However, cultural issues may also have a direct impact on your client's behaviors. For instance, some client's may have acted at the simply request of an elder without having considered the consequences of the action because of cultural training which instilled a world view of how to behave.

To make sense of your client's life experience, and then to present it to the jury, you have to understand how specific race and cultural issues played a role. You will have

develop an understanding of how culture played a role in each of the four levels of the ecological model. For instance, a bi-racial client will have a very different experience with racism at each of the levels (individual, family, neighborhood and institutions) than will a recent immigrant from Asia. The stories may be equally compelling, but must be investigated with sensitivity.

To do this successfully, you will likely need to find an expert to assist you. This may also prove helpful in conducting a mental health assessment because cultural issues can play an enormous role in understanding mental illness and behavior. Not the least of the issues related to culture and race are that people of color with mental illness are systematically under-diagnosed and under-treated; have less access to, and availability of, mental health services; and receive poorer quality of mental health care.⁹

⁹ Surgeon General's Report. Mental Health: Culture, Race, Ethnicity - Supplement (2001). Washington, D.C.: U.S. Government Printing Office.

5. Selected Neurological Disorders

A. Mental Retardation

In 2002, the US Supreme Court ruled that mentally retarded people cannot be executed *Atkins v. Virginia*, 536 U.S. 304 (2002). It held, in part:

Mentally retarded persons frequently know the difference between right and wrong and are competent to stand trial. Because of their impairments, however, by definition they have diminished capacities to understand and process information, to communicate, to abstract from mistakes and learn from experience, to engage in logical reasoning, to control impulses, and to understand the reactions of others (at 318).

The *Atkins* Court recognized that the deficiencies of the mentally retarded “do not warrant an exemption from criminal sanctions, but they do diminish their personal culpability.” The death penalty is, of course, reserved for the limited group of people whose culpability is the greatest. In recognition of these same problems, Congress enacted a similar prohibition to that enunciated in *Atkins* when it reinstated the federal death penalty in 1988 and again when it expanded the death penalty law in 1994. Thus, although Congress has long barred the execution of the mentally retarded, the *Atkins* Court has now offered an argument that it would be unconstitutional to force a mentally retarded person through capital proceedings because of the inherent threat to the reliability and fairness of those proceedings.

Mental retardation is a pervasive, developmental disability. In 2002, AAMR

slightly revised the definition.¹⁰ The revision is better suited for application in forensic evaluations in death penalty cases. A person with mental retardation is someone who manifests “a disability characterized by significant limitations both in intellectual functioning and in adaptive behavior as expressed in conceptual, social, and practical adaptive skills.” The intellectual impairment must be documented in the individual’s real-world experiences and at the same time in broad categories of adaptive impairment.

At this time, however, it remains unclear how the slight differences between the definitions in the DSM-IV-TR¹¹ and the 2002 AAMR Manual will play out in specific cases. It seems advisable for now to address both definitions when discussing how your client meets the MR criteria for diagnosis.

The three-prong definition of mental retardation that requires 1) significant limitation in intellectual functioning; 2) significant impairment in adaptive functioning; and 3) onset before age 18, is well established in the medical and scientific literature. Because the differences between DSM-IV-TR and AAMR 2002 can be significant in some cases, it is important to think through the diagnostic differences in your case before seeking an opinion from an expert.

IQ Scores: The details of testing are

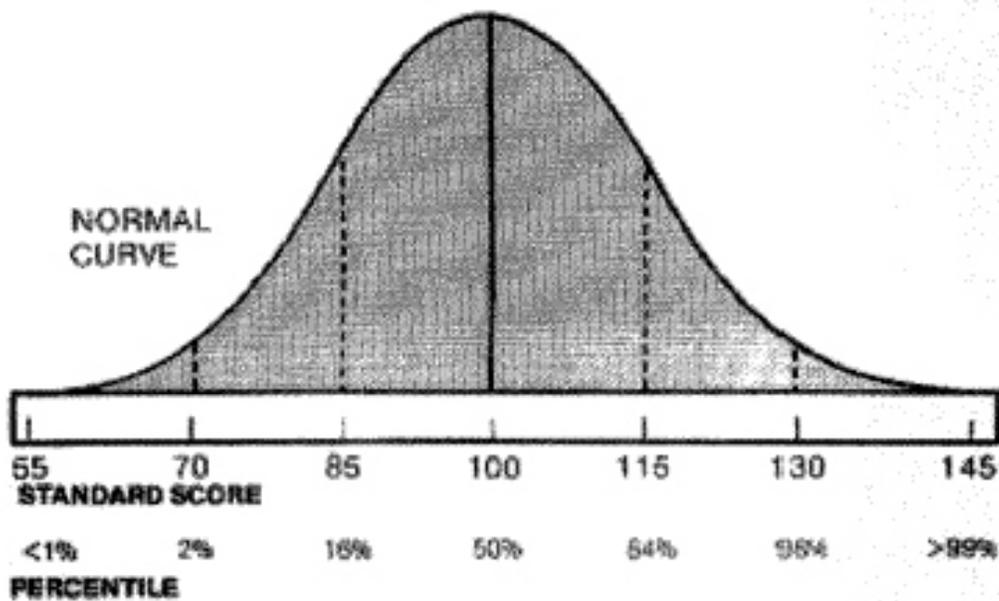
¹⁰ AAMR Mental Retardation: Definition, Classification, and Systems of Supports 10th Ed. (2002) Washington, D.C.: AAMR. Hereinafter, AAMR Manual.

¹¹ American Psychiatric Association (2000). Diagnostic and Statistical Manual of Mental Disorders, 4th Ed. Text Revision. Washington, D.C.: APA. Hereinafter, DSM-IV-TR

discussed later, but for now, the assessment of intellectual functioning requires testing be done to assess IQ. The upper boundary of mental retardation cannot be stated with complete precision in terms of IQ scores (meaning that the upper limit is a guideline rather than a bright line rule). Generally, it will encompass everyone with a score of 70 or below, and additionally some individuals with

scores in the low 70s (and even mid-70s), depending on the nature of the testing information.

As seen on the bell curve below, approximately 2% of people are mentally retarded, meaning your client functions at a level worse than 98% of the general population:



Levels of Severity of Mental Retardation

Levels	IQ	% of M R Population
Mild	50-55 to 70-74	85
Moderate	35-40 to 50-55	10
Severe	20-25 to 35-40	3.5
Profound	20-25	1.5

The DSM-IV-TR definition was adopted based on the 1992 AAMR Manual which has not been replaced. It is likely that the next version of DSM will adopt the new definition, but until it is published, there are slight differences between the two standards. The DSM-IV-TR defines the IQ score component of MR as “an IQ of approximately 70 or below” and notes that measurement error on the specific IQ test should be considered. This means that on a test with a measurement error of 5 points such as the WAIS-III, a score of 70 actually represent a range of 65 to 75. This is sometimes referred to as a confidence interval and can be understood to mean that a true IQ of 70 is reflected (with 95% confidence) by a test score IQ between 65 and 75. The confidence interval refers to the boundary around the test score within which the true IQ sits. Other IQ tests will have specific measurement errors which must be found in the test manual in order to interpret the confidence interval.

In *Atkins*, the Supreme Court noted that “an IQ between 70 and 75” and below is “typically considered the cutoff IQ score for the intellectual function prong of the mental retardation definition.”¹² This language appears to have been based on a recognition of the measurement error and confidence intervals for IQ tests.

The AAMR 2002 adopts a standard on IQ tests that the person scored two standard deviations below the mean on the assessment instrument and taking the standard error of the instrument into account. On the WAIS-III, 70 is the score which is two standard deviations below the mean of 100, reflecting the same general standard as the DSM-IV-TR. However, other assessment instruments have

other standard deviations, so a higher or lower score may actually reflect performance two standard deviations below the mean.

Both the DSM and AAMR have been attempting to push the discussion of MR away from hard cut-off numbers on IQ instruments. Each definition uses language which describes no upper limit on IQ score above which MR cannot be diagnosed, but each also suggests the range of 70 to 75 or below, the language adopted by *Atkins*.

This upper boundary of IQs for use in classification of mental retardation is flexible to reflect the statistical variance inherent in all intelligence tests and the need for clinical judgment by a qualified psychological examiner. As DSM-IV-TR comments: “Thus it is possible to diagnose Mental Retardation in individuals with IQs between 70 and 75 who exhibit significant deficits in adaptive behavior.”¹³

Adaptive functioning: Adaptive behavior is the component of the definition that requires that the intellectual impairment have produced real-world disabling effects on the individual’s life. The purpose of this element is to assure that the individual is not merely a poor test-taker, but rather, is a truly disabled individual. In conjunction with the age-of-onset requirement, it also provides a check against allegations that the mental retardation is malingered.

Again the definition varies slightly as to how to measure adaptive functioning although all the definitions require a significant deficit. This is the area in which attorneys must do substantive independent factual development. School records, youth authority records, social

¹² 122 S.Ct. at 2245 n.5

¹³ DSM-IV-TR at pp. 41-42

service records, family assessment records, juvenile assessments by law enforcement or medical professionals, lay witness observations and family history to explore multi-generational patterns of impairment are essential to substantiate a claim of mental retardation. No determination of MR can be made without this extensive social and family history work.

According to the DSM-IV-TR, adaptive functioning is measured by deficits or impairments in two of the following ten areas: communication, self-care, home living, social/interpersonal skills, use of community resources, self-direction, functional academic skills, work, leisure, health, and safety.

The AAMR 2002 definition modified this standard to better address the conceptual approach to mental retardation and the provision of services and support to people with MR. The definition requires significant deficits in the areas of social, conceptual or practical functioning. Social skills are said to include: interpersonal, self-esteem, responsibility, gullibility, naivete, following rules, obeying laws and avoiding victimization. Conceptual skills are said to include: language, reading and writing, money concepts and self-direction. Practical skills are said to include: daily living activities, instrumental activities of daily living, occupational skills and maintaining safe environments.¹⁴ Significant deficits in this context means a score of two standard deviations below the mean on an instrument that measures all the areas of adaptive functioning. AAMR recognizes that no such instrument currently exists but the point seems to be to stress that the assessment of adaptive dysfunction must be quantitative rather than

based on impressions of functioning. The AAMR Manual does review a number of currently available adaptive functioning assessment instruments.

The assessment of adaptive functioning will continue to rely heavily on social history information and the anecdotal information elicited through competent investigation. Social history information needs to be woven into the assessment of adaptive functioning.

A diagnosis of MR requires clinical judgment. This means that your judge should not make such a determination without expert opinion. This also means that you will need to provide an expert with extensive social and family history information which you develop pre-authorization.

Client records and information: Relevant information may appear in school records and psychological assessments conducted during adolescence and childhood, including custodial assessments; medical evaluations, hospitalizations, counseling referrals and previous assessments; reports of seizures, fever, infections, failure to thrive, physical or emotional abuse, head injury, and ingestion of toxic substances; previous testing, both raw testing data and reports prepared (either medical or forensic); prior criminal proceedings in which the client may have discussed legal rights or cognitive impairments; disability applications or social service contacts; nutritional information; information about day-to-day functioning (going to the store, doing homework, dealing with conflict, ability to care for self (hygiene, food, work history), relationships with neighborhood peers, reading a map or taking public transportation alone; memory problems; suggestibility, independence, ability to abstract information.

¹⁴ AAMR Manual at p.82

Family records and information: Relevant information may be found in the client's parents health records; parents' level of intellectual functioning; whether the mother used drugs or alcohol during pregnancy; whether the mother used home remedies to self-medicate illnesses during pregnancy; mother's age when pregnant; birth complications; whether there is a pattern of low intellectual functioning in siblings, or extended family; social service contacts with the family. These records may contain information that allows you to demonstrate the adaptive functioning deficits; that supports the genetic-familial patterns of impaired cognitive functioning and which bolster the finding of mental retardation by providing possible etiologies for MR.

Neighborhood records and information: For a client you suspect to be mentally retarded, neighborhood information may include, exposure to neurotoxic agents which cause MR. These include, at least, heavy metals such as lead, but may also include numerous other toxic agents in the environment if the client was exposed in sufficient quantity (see dose-response section below); level of support available to the client from social networks in the neighborhood or social institutions; positive influences from mentors or role models (or lack thereof); economic stability and status.

The historical component of MR also makes it almost impossible to fake. While some clients might be able to intentionally perform poorly on an IQ test, no one can retrospectively fabricate the historical pattern required for diagnosis. In fact, the vast majority of MR clients will seek to hide their impairments not make themselves appear worse (see below). You must develop facts that demonstrate developmental delays at

critical periods of development of language, physical movement, personality, independence, and cognition and academic stages. Social history development is the basis for documenting the adaptive functioning component of MR, and will also provide you the details with which to explain MR to a jury.

Working with a mentally retarded client: Working with MR clients can be very challenging, especially working with a high functioning MR individual. Despite popular conceptions, most mentally retarded clients do not appear different than any other client. Most MR clients will not have facial or other physiological malformations, they will not be unable to speak or always be non-responsive to questions, they will not always demonstrate peculiar physical movements, and they are likely not going to be docile and trusting of you during the first interviews.

Working with an MR client requires that you spend a significant amount of time with him/her. Your first task, as with all clients, is to build trust and rapport. You might feel frustrated because the client only wants to talk with you about something that seems irrelevant to your work on the case (sports, for instance), but this repetitive talking on a subject is a hallmark of MR. Spending time with an MR client will allow you to see and understand:

- 1) how the client is compliant with your requests even when he/she does not understand what you are asking;
- 2) how the client attempts to minimize impairments by talking about a subject he/she feels competent in (sports);
- 3) how the client makes sense of issues you raise and whether over time, he/she is able to progress to or beyond a

- rudimentary understanding of an issue;
- 4) how difficulty expressing thoughts and emotions may at first appear to be callousness or indifference but actually reflects impairment;
 - 5) how words are created by the client (e.g., trickster to describe the behavior of the police during interrogation) which convey a sense of being overwhelmed and taken advantage of; and,
 - 6) how an ability to use certain words or phrases masks the client's inability to comprehend meaningfully the concepts behind the phrase.

Mentally retarded (as well as mentally ill) clients require the defense team to spend a great deal of time with them. High functioning mentally retarded clients in the criminal justice system will already have well-developed techniques for hiding the scope of their functioning and impairment. These clients probably will be able to hold a conversation and pay attention, but repeated visiting will begin to show you areas of conversation where the client is comfortable and returns to when he/she does not understand other areas.

Masking is the process by which mentally retarded people attempt to distract from or hide from view their true level of functioning.¹⁵ Many clients are successful at masking. Many clients are able to talk for hours on a single subject and appear to have extensive knowledge so long as the conversation remains within the area they have comfort. For instance, one client was able to recite backwards and forwards (starting at any point) the kings and queens of

Europe over hundreds of years and their relationships to each other. This area of competence must be explored to assess the depth and width of the client's knowledge. This client was not able to calculate change from a dollar he would get back after buying a 60 cent soda. Mental retardation does not require that your client have no mental functioning at all, simply that your client is seriously impaired compared to the norm in cognitive and adaptive function.

Masking is an important concept for two reasons: first, because it means that you will have to work harder to recognize mental retardation in your client. Second, your client might face charges of malingering or lying as a result of those normal processes and you will need to carefully document how your client masks and other examples to counter the charges of malingering.

The mentally retarded client is likely to be a very poor historian and may not be self-aware of his/her strengths or weaknesses. Some reasons why mentally retarded clients are poor historians include:

- a) an inability to recall details (impaired memory);
- b) inability to organize concepts and understand how things relate to each other;
- c) limited ability to communicate (receptive and expressive language deficits);
- d) masking illness (attempt to hide the extent of the disability);
- e) confabulation (filling in details to portray a coherent story despite not having actual knowledge of details provided)
- f) passivity, compliance and deference (likely to agree with interviewer in effort to please);

¹⁵ Edgerton, R. (1993). The Cloak of Competence 2nd Ed. Berkeley: University of California Press.

- g) rigidity in the face of contradictory evidence; and,
- h) lack of comprehension.

Given these features of mental retardation, development of accurate facts to prove up mental retardation requires very detailed interviewing of lay witnesses on the facts of pre-offense functioning as discussed above. In addition, you will need to retain a mental health expert with particular skills and training on mental retardation.

B. Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effect (FAE)

Maternal alcohol consumption during pregnancy has important negative consequences for fetal development. Research suggests that there is no safe level of consumption, although as with most toxins, increased dose leads to increased neurological deficits. FAS results from intrauterine exposure to alcohol and has well-recognized central nervous system, physiological, cognitive and behavioral symptoms. FAE is a related syndrome, sometimes called alcohol-related neuro-developmental disorder. Originally thought to be less severe than FAS, it is now thought to be different only in the expression of the facial stigmata of FAS. FAS results from maternal drinking of approximately 2 drinks per day while pregnant, although no intake of alcohol during pregnancy is considered to be without consequences for the developing fetal brain. FAS is also associated with failure to thrive in infancy and family history will assist in identifying patterns in the family which support a hypothesis of intrauterine alcohol exposure.

FAS is a leading cause of mental retardation and cognitive impairment.

Approximately half of FAS children are mentally retarded, but nearly all have serious cognitive, attention and behavioral problems. Autopsy findings have indicated significantly reduced brain size in FAS, as documented in newborns as well as older children and adults. FAS children have extreme difficulty with:

1. abstract reasoning and judgment
2. executive functions (ability to coordinate, plan and carry-out appropriate responses)
3. perceiving social cues
4. processing speeds and diminished attention
5. learning
6. inhibition of impulses

Although first observed in childhood, these functional deficits persist throughout life and appear to worsen over time when untreated or undiagnosed.

FAS also has three areas that serve as physical markers: 1) intrauterine and/or postnatal growth retardation; 2) central nervous system impairment; and 3) a pattern of facial characteristics that includes short palpebral fissures (eye slits), elongated midface, flattened philtrum, thin upper lip, flattened maxilla, epicanthal folds, and minor ear anomalies. Some of these features may occur in normal people, but it is the pattern that defines FAS. Some of the facial patterns do not persist beyond adolescence, changed by pubescent growth. Other physical symptoms which are common (but not always present) in FAS include: heart defects, minor hand anomalies, malformed or misaligned teeth, myopia and hearing loss.

People with FAE will likely not have the facial pattern associated with FAS, but may have equally serious behavioral and cognitive

symptoms. FAE is likely caused by intrauterine exposure to alcohol in smaller amounts than causes FAS or possibly varying amounts of alcohol at specific developmental periods during pregnancy.

Factual development of FAS/FAE requires assessment of the physiological, cognitive and behavioral patterns that are markers of intrauterine exposure to alcohol both at the time of trial and more usefully, in social and family history records. It is particularly important to note in the records patterns of family substance use as well as alcohol related deaths. Similarly, records may contain crucial information on developmental milestones, physiological symptoms noted in childhood and early photographs which will show facial patterns of abnormalities that have disappeared in adulthood. Additionally, you will need percipient witnesses to discuss your client's mother's alcohol intake during pregnancy. Many witnesses may be reluctant to discuss these facts, but you must get them. Although your client's siblings may be helpful, better witnesses on this issue are the people who went out drinking with the client's mother or caretakers who were present when she came home from drinking.

Physical and sexual abuse are also strongly associated with FAS/FAE – meaning that those with FAS/FAE report exceptionally high rates of being abused. This may make your investigation more difficult because you will also need to be gathering evidence of abuse, often committed by the same witnesses (e.g., caregivers who become frustrated with your client's disabilities).

MRI imaging and autopsy findings seem to indicate that intrauterine exposure to alcohol damages the corpus callosum, basal ganglia, hippocampus, and cerebellum,

although imaging research on FAS/FAE is relatively new. Neuropsychological testing has demonstrated cognitive impairments as well as consistently reduced frontal lobe function greater than that explained by reduced IQ.

FAS/FAE will ultimately be an explanatory model that you can use to help the jury understand your client. You will have to be prepared to deal with the common response that: “well, my mother drank while pregnant with me” The answer to this will have to be developed during your investigation as to how FAS related to other deficits that your client experienced and other problems your client's mother may have had that enhanced the impairment your client suffered. Nevertheless, FAS/FAE is a casual explanation of functioning (see Section 7 on causation) that shows the origins of your client's impaired functioning rests in the family and social dynamics in which he/she was conceived and raised.

C. Pesticide Exposure

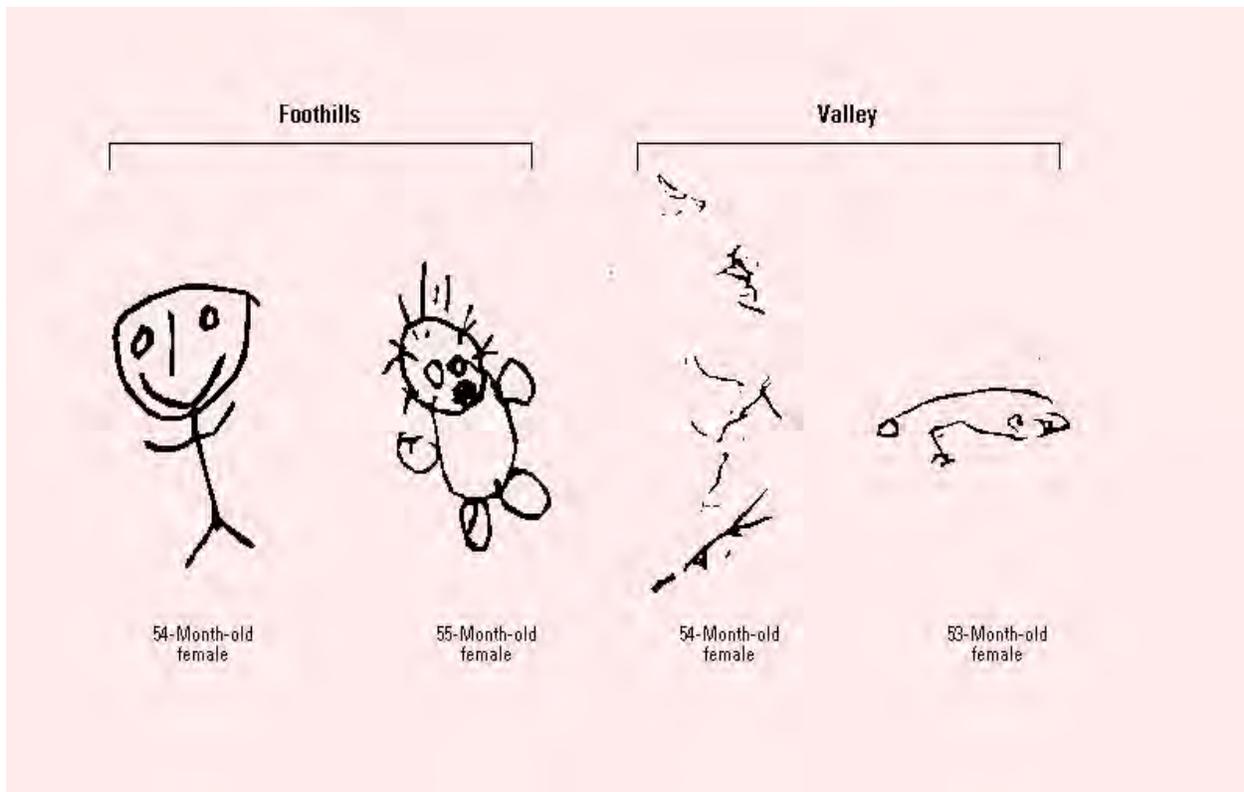
The widespread use of pesticides in urban and rural areas has made exposure to them an issue in many cases. A pesticide, which is a poison, is a compound with the purpose of controlling or destroying any pest. In general, pesticides kill insects by attacking the central and/or peripheral nervous systems. There are three primary classes of insecticides in use throughout the world: organochlorines, organophosphates and carbamates. Almost every pesticide affects the central and/or the peripheral nervous system directly or indirectly. Pesticides work by interrupting or destroying the interactive processes of the nervous system, including functioning of the neurochemical and neuroelectric systems of the brain. The mechanism by which

pesticides disrupt normal functioning are slightly different by class of pesticide and as a result exposure to each class has slightly different short and long-term consequences and symptoms.

At sufficiently high dose levels, pesticide-induced effects may result in transient changes or permanent neuronal dysfunction (see causation section below). Environmental exposures, like exposure to pesticides, are part of the explanation and etiology of neurological impairment, dysfunction and behavior changes. Pesticide exposure alters the way in which your client's brain works and may initiate otherwise inexplicable behaviors. Pesticide exposure evidence also

fills out the social history picture of your client's life to differentiate him/her as an individual, and to provide necessary information to mental health experts.

Human exposure occurs by three pathways: ingestion, inhalation or absorption. These mean, a child or adolescent may be exposed by the water supply, by what is sprayed in the home or school or at playgrounds, from neighborhood industry or proximity to hazardous waste sites, by air and food supply contamination, by places that children play, by contaminants brought home on the clothes of the parents from work or by fires and industrial accidents.



Drawing of age-matched children, comparing pesticide exposed children (on right) to non-exposed children (on left), controlling for socio-demographic factors. Drawings on left by less exposed children; drawings on right from highly exposed children. Reprinted with permission of EHP: Guillette, E.A., Meza, M.M., Aquilar, M.G., Soto, A.D. and Garcia, E. (1998) Environmental Health Perspectives 106(6) 347-53.

In young children, from in utero to adolescence, the human body is less able to process out pesticides compared to adults experiencing the same exposure. The absorption in children is more than 70% compared to absorption in adults of 30% of the chemical agent. In addition, because the central nervous system is not fully developed until after adolescence, exposures prior to and during adolescence alter the development and functioning of the brain to a greater degree. The enhanced susceptibility of the young human brain to damage from these chemicals has been well-recognized by toxicology for decades.

Symptoms that result from exposure depend upon a number of factors:

- 1) the specific agent to which your client was exposed (type of exposure)
- 2) the duration of exposure (quantity of exposure)
- 3) the amount of agent ingested, absorbed, inhaled (quality of exposure)
- 4) the susceptibility of your client to symptoms based on physiological, genetic and psychiatric functioning and predisposition.

To investigate pesticide exposure issues,

you will need to obtain detailed information on each type of compound your client was exposed to, how often the exposure occurred, the context of the exposure (in a field picking crops or playing in a hazardous waste site), the symptoms observed at the time of exposure and the pathway by which your client was exposed. Many clients will not know what compounds he/she was exposed to, only how he/she felt and symptoms they experienced. You will have to uncover evidence of which compounds from other sources. Much of this information will, by necessity, come from percipient witnesses, for instance, farm owners who purchased and arranged for spraying of fields in which your client worked. However, in addition to the social and family history records and interviews related to your client, you must also search for local, county, state and federal regulatory agency records (e.g., EPA, HUD, Fire Departments, departments of pesticide regulation), local newspapers, civil suits against local industries, local medical clinics and medical professionals, and local agriculture extension departments of local universities.

Overall, symptom patterns to investigate include:

Physiological	Cognitive	Behavioral
Eye twitching Eyelid twitching Pinpoint pupils (constriction) Skin rashes Nausea Vomiting Diarrhea Abdominal pain Weakness in extremities or generalized Eye irritation and redness Headaches Dizziness Sweating Salivation Chest tightness Blurred vision Pressure in the head Muscle twitches or tremors Aching in joints Unconsciousness	Reduced vigilance Attention deficits Psychomotor retardation Impaired memory function Reduced comprehension Depression Confusion Speech difficulty (slurring) Difficulty formulating thoughts Intelligence decline Excessive dreaming Developmental Delays Developmental retardation Cognitive deficits Brain function decrease	Depression Mental confusion Slowing of performance Impairment of judgment Schizophrenic-like reactions Irritability Temper outbursts Aggressive behavior not previously observed Belligerence not previously observed Psychosis Hyper-excitability Emotional lability Anxiety Paranoia Increased excitability and agitation Extreme/disproportionate response to stimuli

Pesticides, like almost all neurotoxic agents, cause both short-term (acute) and long-term (chronic) central nervous system damage. Acute exposure refers to a poisoning event where the client suffers immediate symptoms. Chronic exposure generally refers to a sub-symptomatic exposure that repeats or extends over time. It is possible that a person can be repetitively acutely exposed as well. Acute exposure places the brain into a state of “uncontrollable” over-stimulation or over-excitation. This causes a short term physiological and behavioral changes, as well as permanent brain dysfunction.

A number of additional issues arise when investigating exposure to pesticides. First, most of the research that has been done to date has examined single exposures to a single pesticide. This does not reflect the real-world

exposure of most people who are instead exposed to numerous types and kinds of pesticides at once. This is important because many of these compounds interact with each other in the human body and cause a toxicity as much as a thousand times as great as either compound alone. For instance, while malathion is considered comparatively safe, when exposed to malathion and parathion at the same time, the effect of the malathion is enhanced nearly 400 times because parathion inhibits the enzymes that break down malathion in the human body.

Second, the severity of symptom patterns (including brain dysfunction) may also be potentiated by other injuries or impairments to the brain. For instance, physical child abuse which results in brain trauma appears to potentiate the effect of neurotoxic agents like

pesticides. In effect, more than simply being an additional injury on a list, pesticides potentiate the existing damage, resulting in a significantly more impaired functioning.

Third, kindling effects refer to a sort of priming that occurs when a person is exposed to pesticides for a period of time, then removed from the exposure and subsequently re-exposed. Because pesticides causes chemical and electrical changes in the brain, it appears that upon re-exposure, the brain quickly returns to its familiar response, which may be more severe than the current exposure level would suggest. This is a kindling effect.

Finally, after sufficient factual development, it is possible to use neurological and neuropsychological tests to assess the impact of pesticide exposure. Neuropsychological batteries have been developed to specifically test for neurotoxic exposures. The World Health Organization has published a recommended battery for assessing neurotoxic exposure. These batteries utilize existing neuropsychological tests, but the examiner should have specialized training to interpret the tests for

neurotoxicity. Similarly, the neurologic examination is not especially different for assessing neurotoxicity (although some additional types of tests may be indicated, such as nerve conductance testing), but the examiner should have experience and training to assess the results.

D. Metal Exposure

Mercury, lead, cadmium, arsenic, selenium, manganese, and aluminum, all metals, are widely used in common products. Exposure to each of them causes serious cognitive and behavioral problems which persist throughout life after exposure. As a result of their widespread use, people come into contact with these metals quite frequently, usually at very low doses. For some, a low level dose-exposure appears to have no measurable, lasting effect. However, for all metals at high levels of exposure, and for lead and mercury at any exposure level, significant cognitive and behavioral effects will be observed. Significant exposure to lead or mercury in utero or in early childhood can cause mental retardation as well.

Symptoms Associated with Metal Exposure

Acute Exposure Symptoms	Chronic Exposure Symptoms
<p style="text-align: center;"> abdominal colic constipation vomiting headache lightheadedness dizziness forgetfulness anxiety depression irritability muscle and joint pain seizure coma increased intra-cranial pressure parathesia nightmares confusion emotional lability mood swings </p>	<p style="text-align: center;"> Persistent cognitive deficit Decline in IQ score Impaired Attention Decline in visuo-spatial functioning Impaired Memory Reduced Reaction time Impaired Executive Functioning Mood Alterations </p>

1) *Lead*: Recent research indicates that there is no safe level of lead exposure, with even the smallest amounts, at 1 microgram per deciliter of blood, ingestion in childhood results in lifelong decreases in IQ and increases in behavior problems.

Lead has been recognized as causing neurological damage for at least 150 years, yet industry was slow to remove lead from places which exposed people to lead's dangers. Lead water pipes were used until the 1920s, lead paint was used in household indoor paint until the 1960s, it was used in cans for food and drinks until the 1970s, and in gasoline well into the 1970s. Lead persists in the soil of many urban neighborhoods in significant amounts. Lead is still found in solder, batteries, paint, pipes, ceramic glazes, and roofing materials. Lead is still used in many

folk remedies in some Asian and Latino communities. Lead exposure disproportionately affects poor and urban people.¹⁶ Although its use is now limited in many products, lead is still extensively used in industrial production.

Lead crosses the placental barrier and poses a threat to normal development in utero. As with other neurotoxic agents, children are more susceptible to exposure and symptoms because of a combination of behaviors and the developmental stages of the brain. Children often put things in their mouths and chew on

¹⁶ Brody, D.J. et al (1994). Blood lead levels in the US population: Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991) Journal of the American Medical Association 272(4) 277-83.

things that adults may not (e.g., lead paint chips which have a sweet taste). Once exposed, lead is stored in skeletal bones.

Even at exceptionally low levels of exposure, lead causes:

- decreased IQ and cognitive functioning
- heightened distractibility and shortened attention span
- impulsivity
- inability to inhibit inappropriate responses to stimuli
- poor vigilance
- inability to follow simple and complex sequences of directions
- deficits in changing response strategy

These impairments, which often begin in childhood from lead exposure, persist across the lifespan of the exposed person. Lead has a diffuse affect on the central nervous system, reducing synaptic counts, neuron density, mitochondrial membrane development, and neurotransmitter and enzyme function. As a result, low level lead exposure, even without overt symptoms can result in cognitive, developmental, and behavioral deficits and delays.

Almost all jurisdictions (county or local) have lead abatement programs. These programs are usually an excellent source of community exposure information. Some of these programs have created zip-code based exposure risk maps that are very helpful exhibits.

2) *Mercury*: Mercury is commonly used in batteries, paint, radios, thermometers, calculators, cosmetics, jewelry, dental care (although, rarely in the US currently), and various manufacturing processes. Mercury was also used widely in the US as a fungicide

for many years to treat seeds (in some countries it is still in use). Exposure to mercury is also common for people whose diets are high in fish as a result of the bio-accumulation of mercury in fish which passes onto humans on ingestion. Methyl mercury, one of two types of mercury (the other being inorganic mercury), is extremely damaging to the brain because a large amount of ingested, inhaled or absorbed mercury crosses the blood-brain barrier and builds up in the brain, with approximately 10% of methyl mercury body burden being found in the brain. As with lead, there is really no safe level of exposure although EPA currently uses an exposure limit of .1 microgram per kilogram of weight per day.

Mercury appears to be the only metal that biomagnifies, meaning higher in the food chain shows higher amounts of mercury per body weight and heightened effects. Mercury is also quite mobile, carried in water, air and soil. Epidemiological evidence of the effects on humans come from two large scale poisoning incidents (Minamata, Japan 1953 to 1959; and Iraq 1971).

Methyl mercury also crosses the placental barrier and children are at heightened risk for symptoms and exposure. Children who are exposed (unlike adults) show language and memory deficits. Acutely exposed adults also have certain hallmark symptoms that involve peripheral neuropathy, muscle tremoring, gait disturbance and ataxia, visual field constriction and hearing loss.

Like lead, mercury causes a variety of long-term problems:

- decreased IQ and cognitive functioning
- gait and balance problems

- impulsivity and agitation
- inability to inhibit inappropriate responses to stimuli
- poor vigilance
- inability to follow simple and complex sequences of directions
- deficits in changing response strategy
- mood swings

Childhood and *in utero* exposure effects persist across life-span. *In utero* exposure in humans has been shown to cause severe developmental abnormalities, including neurological abnormalities, delays in developmental milestones, sensory and behavioral maladjustment that persists. An extensive literature on human methyl mercury exposure and outcomes exists for both acute and chronic exposures.

Testing for Metals: First, you must have developed facts from independent sources that effectively prove the quality and quantity of the exposure before you consider medical testing for metals. Second, you must have neuropsychological testing performed before considering medical testing. Neuropsychological batteries have been developed to specifically test for neurotoxic exposures. These batteries utilize existing neuropsychological tests, but the examiner should have specialized training to interpret the tests for neurotoxicity. Since the evidence you are seeking for mitigation is both the facts of exposure and how your client was exposed, as well as evidence of the behavioral and cognitive symptoms of exposure, it is not enough nor often useful to use medical tests to quantify exposure. However, in some cases, when carefully considered with your experts and based on overwhelmingly strong neuropsychological and corroborative factual evidence, quantifying exposure may incrementally add to your case.

For lead, it is possible to use long-bone x-ray techniques to assess quantity of lead exposure that occurred earlier in life. If the exposure is very close in time to when you test, blood-lead levels can be helpful.

For mercury and lead, a pattern of deficits has been identified that can be observed with MRI imaging. For lead, diffuse neuronal damage is expected which suggests functional as well as structural imaging (see Section 9 below). For mercury, the key areas of the brain damaged by methyl mercury appear to be the visual cortex, cerebellar vermis and hemispheres, and the postcentral cortex.

E. Organic Solvents

Industrial solvents are ubiquitous. The term “organic solvents” refers to a group of chemical compounds or mixtures that are used for extracting, dissolving or suspending non-water soluble materials. Solvents are used in many manufacturing processes, as well as in dyes, polymers, plastics, textiles, inks and pharmaceuticals. These solvents have been known since the early 1970s to be neurotoxic, meaning that they have been known since then to cause damage to the central (brain) and peripheral nervous systems.

Studies of chronic exposure in workers has demonstrated that organic solvents cause peripheral neuropathy, which are disorders of the peripheral nervous system, and mild toxic encephalopathy that persists for many months and years following the cessation of exposure. Chronic exposure has also been shown to cause neurobehavioral changes in workers, including impaired judgment, impaired concentration and impaired memory. Chronically exposed workers have been shown to experience fatigue, irritability, memory impairments, sustained alteration in

mood, emotional instability, diminished impulse control, and deterioration in cognitive functioning.

These effects are caused by the pharmacological properties of solvents in the human body. Research on the behavioral sequelae of solvent exposure began in the 1950s, beginning with studies on the psychological functioning of exposed subjects. In the 1960s, this research was extended to examine in a systematic manner the behavioral consequences observed in both chronically and acutely exposed workers. Evidence from this early work has been confirmed over decades of continuing research. Additionally, research over this period has examined the effect of specific solvents in the human body as well as potentiating effects from multiple, simultaneous exposures.

Solvents as a class have some common effects in the human body because of the mechanism of action in the body. The somatic and mental changes noted above occur as the result of degeneration of the myelinated nerve fibers and axonal swelling. In brief, solvents deteriorate the functioning of the central and peripheral nervous system by breaking down the integrity of the system at its smallest component parts. The deterioration in central nervous system function is often permanent and irreversible. Further, solvents preferentially accumulate in lipid-rich tissues, including the central nervous system, which both explains the significant effect in the central nervous system and results in accumulation in the body.

Toluene inhalation is well documented to cause dysfunction of the central nervous system. The exposed worker will experience impaired cognitive and neuromuscular

function. Chronic exposure to toluene results in permanent damage. The sequelae include ataxia, tremors, seizure activity, paranoid psychosis, hallucinations, nystagmus and impaired speech, hearing and vision. These changes can range from mild alterations to severe depending on dose. These effects were well documented by the 1960s. Toluene, found in numerous consumer products such as glue and gasoline, is one of the toxic agents that causes brain damage when people “huff” substances (see Section 6f below).

Benzene is an extremely toxic solvent. In the early 1970s, the National Institute for Occupational Safety and Health (NIOSH) had already promulgated standards to minimize and control worker exposure to benzene. One strategy they recommended was to stop using Benzene in favor of other less toxic solvents. Another recommended strategy was that workers be given personal protective equipment, such as respirators and skin protection. Inhalation of Benzene fumes is more significant than skin contact, although both pose a serious risk. Benzene’s toxicity was first noted in the medical literature in the 1920s and 1930s. These included reports of deaths from acute exposure and physiologic symptoms of chronic exposure. By the 1950s, the effects of inhalation were well-documented to include giddiness, headache, nausea, depressed respiration, ataxia and in severe cases, seizures and loss of consciousness.

Chloroform targets the central nervous system, having a depressive effect. Exposure results in ataxia, decreased coordination and an anesthetic effect. In fact, chloroform has been used historically as an anesthetic for surgery. Chronic inhalation results in decreased concentration, depression, irritability, and possible psychotic episodes

including hallucinations. The neurotoxicity of chloroform is well documented beginning in the 1940s.

Chorotheane (TCE) exposure has permanent effects on the central and peripheral nervous system. The sequelae of such exposure include fatigue, ataxia, difficulty concentrating, memory impairments and increased irritability and anxiety. Employers are required to use mechanical and educational methods to reduce worker exposure to TCE because of its known toxicity. As with Benzene, these methods are recommended to include substituting another, less toxic solvent for TCE, and requiring engineering and personal protective equipment. Knowledge of TCE's toxicity has also been known for decades.

Acetone is also quite toxic. The sequelae of exposure are similar to those of other solvents. Exposed workers first report lightheadedness and headache. Acetone also causes declines in neurological and neurobehavioral functioning. One unique feature of acetone is that it potentiates the effect of other solvents when inhaled and contacted together. Thus, the effect of Benzene, in the case of hand washing with Benzene and Acetone (a common industrial practice), is enhanced when contacted along with Acetone. These effects were well known by the 1970s.

Hexane is known to result in severe peripheral neuropathy. Exposure is most commonly by inhalation. Sequelae of chronic exposure include weakness, numbness, an anesthetic effect, and motor system dysfunction. Hexane exposure can result in a permanent decrease in electrical velocity within nerves, a slowing of normal processing. Research documenting the short and long term

effects of worker exposure to Hexane was being published in the 1970s.

Xylene exposure can result in memory impairments, decreases in reaction time, and ataxia. Research also indicates numerous physiologic effects and pronounced neurologic effects from chronic exposure. These effects may include hyper-reactivity to stimuli, loss of motor function, and behavioral alterations. Repeated, low-level exposure can cause permanent impairments. These effects were widely recognized by the 1970s.

Currently, occupational health regulations, promulgated by both state and federal agencies, require safety equipment for workers who come in contact with solvents. The first line of safety equipment is mechanical, including proper ventilation and filtering systems. Workers must also be given personal protective equipment, including respirators to protect against inhalation and covering for skin to protect against absorption. Living near industrial plants will also expose people to solvents, usually through inhalation but also through contamination of groundwater and soil.

Testing for solvent exposure is similar to that for pesticides. A pattern of neuropsychological deficits can be observed on testing. Similarly, investigative approaches to suspected solvent exposure are similar to pesticide and metal exposure investigation.

F. Learning Disabilities/Language Deficits

Language and learning impairments may seem minor in relation to other forms of mitigation, but when present, they have an enormous impact on how your client has experienced the world, how others have perceived him/her and the likelihood of

psychiatric and behavioral problems. Possibly more importantly, language and learning deficits are likely to be your first hint of your client's neurological problems. During your interviews with your client, you should be paying careful attention to how your client talks, the types of information your client seems able to integrate into other concepts and your client's ability to express ideas. Many language and learning impairments will be recognizable as small or peculiar language usage, speech oddities, repetition in speech patterns or a need for you to discuss the same concept repeatedly.

Language and learning impairments often begin in childhood. Language impaired children have long been recognized to be at increased risk for psychiatric disorders. Further, significant overlap exists between observed behavior problems in childhood and language and learning deficits.

Language and learning problems usually reflect broader brain functioning impairments. These problems usually involve receptive language, the way in which words are heard and understood; expressive language, being the way in which concepts are formed and how words are articulated; and, language processing in the brain, cognition or comprehension. After spending time with your client, these types of deficits may be the easiest to observe and paying careful attention to how your client speaks and responds may give you the first hints as to how your client functions.

Empirical research indicates that about one third of children referred by teachers or parents for psychiatric evaluation specifically because of observed behavior problems, actually suffered from unsuspected language and learning deficits. That means, although

sent for intervention because of acting out, once assessed, it turned out these children had learning and language deficits that caused the behavior problems and which could be treated.

In the studies, when mothers did not know that their children had a language problem, they rated their own children higher on delinquency scales. Where the language impairment was known, the behaviors appeared less significant to the child's mother. When the language impairment was not known to the teachers, they tended to rate children who have language and learning impairments as having severe behavior problems, including considering them to be aggressive, inattentive and overactive.

Other research demonstrates that kids who refuse to go to school were significantly more likely than controls to have language and learning disabilities. These findings demonstrate the co-occurrence of school refusal and language and learning impairments. Longitudinal studies of children referred for language deficits have found a long-term increase in anxiety, attention and social relations behavior problems, but not in conduct disturbance or anti-social behavior. These problems often persist into adulthood and will help you to explain how your client saw him/herself. Even if your client no longer has apparent language problems, he/she may have internalized beliefs based on growing up with these impairments.

All of this research points to the importance in how children are perceived: as delinquent and aggressive or with a treatable neurological condition. It also points to the very real need to undertake extensive investigation, especially if your client at first blush appears to have behaviors that constitute

conduct disorder. When not aware of the language and learning deficits, teachers and parents appear to hold children responsible for negative behavioral problems. This finding tends to lend support to the hypothesis that language and learning deficits may underlie attention, delinquency, truancy and aggressive acts as identified by teachers and parents. These are the “bad acts” that constitute conduct disorder but may in fact be symptoms of neurological dysfunction.

Approximately 13% of state prisoners and 7% of federal prisoners self-report having speech or learning problems. A much higher percentage of juvenile delinquents (35%) are estimated to have learning disabilities. Social and family records (especially school records) and neuropsychological testing will assist you in figuring out if your client has language and learning deficits.

G. Mild Traumatic Brain Injury

Traumatic brain injury (TBI, sometimes referred to as closed head injury) refers to a specific type of injury that is quite common in the United States. Somewhere around 1.5 million people seek medical care each year for head injury. TBI generally refers to a blow to the head that does not pierce the skull, although fracturing may occur. The most common forms of TBI result in diffuse damage to the brain where the force of injury causes a shearing effect or bruising as the brain collides with the inside of the skull.

DSM-IV-TR does not contain a diagnostic category for TBI, although there is a related category, post-concussive syndrome, which was studied for inclusion but determined to be insufficiently supported by empirical research. Determining whether your client has suffered traumatic brain injury depends on four critical

factors: 1) your client lost consciousness for a period of time; 2) your client has (or had) a loss of memory for events either just before and/or just after the injury; 3) your client had an alteration in mental state following the injury; and 4) your client suffered focal, meaning located in a specific place, neurological impairment which can be either permanent or transient.

The problem in many cases is that insufficient documentation exists to answer these four questions. Record gathering is essential since any post-incident reporting of severity will be challenged. However, most of our clients will have suffered what is termed “mild” TBI and will not have sought medical care following head injury because: a) the injury resulted from physical abuse which the care-taker is trying to keep from public view; b) access to medical care is limited and/or ability to pay for care is limited and therefore not sought; c) the injury occurred during substance use and is not reported or discounted because of the presence of substances; d) the client had insufficient social support and was unable to get to a doctor or did not recognize and was not told the severity of the injury until later; or e) the client simply did not go to the doctor. However, if contemporaneous documentation exists, here are the most common techniques for assessing the four critical factors:

A. Level of Consciousness: The standard technique for assessing a person’s level of consciousness at the time of an accident is the Glasgow Coma Scale (GCS). Total score ranges from 3 to 15 based on whether the person’s eyes are open, open in response to verbal command, open in response to pain, or not responsive; whether the person can move in response to verbal command or level of response to a variety of painful stimuli; and

whether the person can verbally respond to simple orientation questions. Alteration in the level of consciousness rather than a specific cut-off on the GCS should be noted, but a GCS score between 13 and 15 is indicative of mild traumatic brain injury.

B. Loss of Memory: The duration of loss of memory is much less important than whether or not any occurred. It is often difficult to assess this since the evaluator usually has little external information about the incident and has no way to assess confabulation or missing details.

C. Change in Mental Status: The obvious signs of this are disorientation or confusion, but may range across a fairly broad spectrum of problems. Most commonly, emergency response personnel simply ask the brain injured person for a self-assessment. Sometimes the alterations are severe and

persistent, most apparent in mood, affect and behavior.

D. Neurological deficits: Assessed by neurologic and/or neuropsychological evaluation. Lack of specific deficits may describe the severity of the injury, not determine the presence or absence of injury.

One key to proving TBI's importance to your client's life is to document the before and after behavior patterns your client exhibited. Your social and family history investigation should be geared (on this issue) to documenting very specific changes in emotion, responses to stimuli and behavior. To do this, you must develop a clear picture of how your client was before the injury and the course of changes in behavior that stem from the injury.

Common Symptoms of TBI

Physical	Behavioral
Headaches Dizziness Nausea Vertigo Noise intolerance Sleep disturbance Blurred or double vision Physical or mental fatigue Decline in coordination/motor function Enhanced startle response	Memory impairment Slowed mental processing Difficulty maintaining train of thought Diminished concentration Increased distractibility Emotional lability Anxiety Depression Substance use Libido changes Low frustration tolerance/agitation Poor impulse control Disinhibition

Adapted from Murrey¹⁷

¹⁷ Murrey, GJ (2000). The Forensic Evaluation of Traumatic Brain Injury Boca Raton, FL: CRC Press.

The important component here though remains that you must document a change (before and after injury) if you plan to argue that the TBI caused your client's behavior (see Section 7 below).

Along with behavior changes, people with TBI are significantly more likely to use substances, to be diagnosed with psychiatric illnesses such as depression, post-traumatic stress disorder, and anxiety, and to have decreased adaptive functioning as evidenced by a noted decline compared to pre-injury ability. Depending on the region of the brain affected, there may also be other problems, such as movement disorders, speech or vision problems, cognition impairments or executive function deficits. Not all regions of the brain will be affected by an injury, thus a person could very likely maintain full cognitive function while losing executive function.

A favorite approach of prosecutors is to suggest that since the TBI occurred some amount of time earlier, your client should be over it. With treatment and extensive rehabilitative services, the majority of TBI patients appear to recover most functioning. A point you should consider making to the jury is that your client did not have access to those services that would have assisted in recovery. Research indicates that psychiatric symptoms associated with TBI persist in about half of people with TBI. Approximately twenty-five percent of TBI sufferers meet the criteria for PTSD six months following the incident. TBI is strongly associated with substance use, especially alcohol. However, nearly 50% of people were intoxicated at the time of injury – meaning you must establish onset of substance abuse or changes in usage for your client. The effect of substances on an injured brain may also be enhanced compared

to non-injured users. Behavioral effects from TBI also persist and may be permanent (up to thirty years).¹⁸ It is estimated that around 15% of mild TBI patients have long-term symptoms.

Evidence or reports of TBI do not change the approach to testing discussed below (see Section 9). Neuropsychological and neurology examinations are still necessary. It is essential that your expert have evidence to substantiate the TBI prior to testing and interpreting testing data. Some additional tests, following administration of standard testing, may be indicated based on social and family history information.

H. Temporal Lobe Epilepsy

Temporal lobe epilepsy (TLE) is a rare condition in which seizure activity originates in the amygdala, uncus and hippocampal regions of the temporal lobe. Another way to say this is that there is abnormal electrical activity in the temporal lobe. Approximately .2% of people are thought to have TLE and the condition is very difficult to diagnose. The seizures are rarely, if ever, grand mal and are more typically partial seizures involving just a portion of the brain. The behaviors associated with TLE make it very appealing as a mitigator, although proving its presence remains quite difficult.

Behavior changes brought about by seizure activity are usually described in relation to the seizure itself: the ictal state (during seizure), postictal (the days and weeks following seizure); and interictal (the period between seizures which defines baseline

¹⁸ Bernstein, DM (1999). Recovery from mild head injury Brain Injury 13(3) 151-72.

functioning). Seizures may be known to the person only through headache, visual aura, confusion or loss of consciousness.

The observable behaviors of the ictal state and the postictal state are very similar, and include non-reflex movement without volition (chewing, swallowing, rubbing the hands, walking or running), hypoactivity and drowsiness, depression with flat affect, confusion (including confusion about the period of seizure), emotional lability, memory dysfunction, increased anxiety, altered social interactions and psychosis. Typically, psychosis begins after a number of years of seizure activity and onset may follow 2 to 72 hours after seizure.

Interictal behavior, that is the “normal state” behaviors that are brought on by seizure activity include psychosis, paranoia, intrusive and repetitive thoughts, dissociative fugue states, increased aggression, mood changes, anxiety and a feeling of impending disaster, hallucinations (vivid sensory experiences), feelings of *deja vu*, and somatic problems.

Other common interictal symptoms are: hyper-moralism (religious conversions, great attention to rules, inability to distinguish major and minor infractions, a desire to punish offenders, metaphysical interests), verbosity and tangentiality (pedantic, constant talking with a tendency towards explaining every detail, speech which branches away from a direct line of thought, excessive background information), hypergraphia (extensive and detailed writings, diaries containing details of every day events, autobiographical writing), heightened emotionality, periodic elation or euphoria (sometimes viewed as grandiosity), depression, irritability, altered sexual interest and libido, obsessionism (ritualized behaviors, compulsion to detail, excessive

orderliness), dependence, humorlessness, and passivity.¹⁹

Diagnosis of TLE, as with all conditions, must first look to historical information such as brain injury, gestational toxicity, birth complications, childhood infections, delayed milestones, learning disabilities or endocrine disruption (e.g., exposure to toxins). It is very difficult to diagnose TLE because the patterns of symptoms shown by sufferers are not always the same. Additionally, some of the symptoms resemble psychiatric illnesses and some of the symptoms also respond to psychiatric medication. Thus a person experiencing psychosis as a result of TLE may be misdiagnosed and prescribed an antipsychotic medication, and antipsychotic medication will be beneficial in reducing the psychotic symptoms even though the cause of the symptoms has not been identified.

Testing with EEG can be uncertain unless seizure activity occurs during the test. Since the seizure activity originates in the deep structures of the brain, nasopharyngeal leads which are placed well inside the nasal passages are necessary. However, placing the leads into the nasal passages can be very uncomfortable and upsetting for many clients and counsel should be sure to prepare the client carefully for the discomfort. Functional imaging may be useful, although it remains uncertain.

I. Degenerative Basal Ganglia Disorders

Parkinson's Disease, Huntington's Disease, Wilson's Disease and Fahr's Disease

¹⁹ Bear, D (1979). The temporal lobes: An approach to the study of organic behavior changes, in Handbook of Behavioral Neurology ed. Gazzaniga, M. New York: Plenum Press.

are all degenerative disorders of the basal ganglia. These are all diseases that progressively affect the deep structures of the brain, primarily the basal ganglia. Each is associated with movement disorders as well as cognitive and psychiatric symptoms. Each is also an inherited, degenerative disorder, meaning that familial patterns will be observed and that over time the symptoms worsen significantly. No current treatment can halt or reverse the progressive decline in functioning, and end-stage for these diseases is a near total inability for self-care. For this workbook's purposes, these four conditions are considered as a group, although if you suspect one of them to be present, it will be essential to determine the specific symptoms associated with each as they are similar but not identical diseases.

The basal ganglia connect to the thalamus and frontal lobes. These connections mean that damage or impairment of the basal ganglia can result in a variety of disease symptoms. The most easily recognized are the movement disorders (chorea, dystonia, myoclonus, or parkinsonism); however, basal ganglia diseases are also accompanied by mood disorders (depression, bipolar and/or suicidal thoughts), dementia, and personality change (irritability, aggression or apathy), often including violent homicidal and suicidal behavior (apathy and aggression are not usually seen in the same patient). Psychosis can also occur, with hallucinations and delusions.

In the early stages of these diseases, people show marked increases in hostility, irritability and disinhibited aggression, and behaviors such as assault, arson, and homicide are often found. Irritability can take different forms. One form is an increase in the baseline level of irritability punctuated by more severe

outbursts. The irritable responses become exaggerated in intensity and duration. In another form, people are not necessarily irritable in general, but become agitated when their requests are not met immediately, no matter how inappropriate. These people often "perseverate relentlessly on a single desire or idea and become progressively more irritable when it is not indulged."²⁰ Personality changes may present prodromally.

In addition, sexual disinhibition, hypersexuality, and paraphilias are commonly observed. These personality changes have been shown to often lead to marital breakdown, in which the reasons for separation mainly relate to aggressive, violent, and abusive behavior associated with the disease.²¹

Interestingly, Gulf War Syndrome (GWS) affects the basal ganglia in much the same way as Huntington's Disease, although it is not degenerative. GWS's behavioral symptoms are almost identical to those found in the basal ganglia disorders.

Although treatment is not available to reverse the degeneration, it is possible to effectively manage the behavioral

²⁰Ranen, NG (2000). Huntington's Disease, in Psychiatric management in neurological disease ed. E.C. Lauterbach. Washington, D.C.: American Psychiatric Press.

²¹ Lauterbach, EC, ed. (2000). Psychiatric management in neurological disease Washington, D.C.: American Psychiatric Press; Lauterbach, EC et al (1998) Neuropsychiatric correlates and treatment of lenticulostratial diseases: A review of the literature and overview of research opportunities in Huntington's, Wilson's, and Fahr's diseases Journal of Neuropsychiatry and Clinical Neuroscience 10(3) 249-66; Harper, PS (1994) Huntington's Disease London: W.B. Saunders Co.

manifestations. Improvement in irritability can usually be seen if the person is relieved of responsibilities and where unexpected changes are minimized. Underlying causes or triggers of outbursts can be removed at least to some degree. These triggers are usually such things as hunger, pain, frustration with failing abilities, or minor unexpected changes in routine. Since incarceration may accomplish much of this, the behavioral aspects of these diseases can be easily managed.

J. Autism-spectrum disorders

Autism is a neurological disease that affects a person's ability to communicate, to form relationships and interact with others appropriately and to respond appropriately to the immediate environment. It is a disease which is increasingly diagnosed: between 1987 and 1998, California experienced a 273% increase in annually reported cases and the increase cannot be explained only by changing reporting criteria. Autism-spectrum (from high-functioning to severely disabled) disorders are an important cause of mental retardation, although high functioning autistic people and people with Asperger Syndrome are likely to have IQ scores well above 70. Most capital cases in which autism will be an issue are likely to involve high-functioning autism (HFA) or Asperger Syndrome (AS).

Although still debated among experts, for this workbook's purposes, these will generally be considered as autism-spectrum disorders (ASD) except where noted. Often times, High-functioning autism and Asperger's are considered the same disorder. However, they are separate diagnostic disorders in DSM-IV-TR and are distinguished based on whether early cognitive and language impairments are present (they are present in autism, not in Asperger's) and whether there is inappropriate

interest in parts of objects (autism) or all encompassing pursuit of an interest to which the individual devotes inordinate amounts of time. DSM-IV-TR notes that differentiating these two conditions may be difficult.

Autism is a developmental and behavioral disorder which is diagnosed (see DSM-IV-TR for complete diagnostic criteria and for Asperger's criteria) based on:

1) impairments in social interaction: impairment in the use of non-verbal behaviors such as eye contact, facial expressions, posture or gestures related to social interaction; failure to develop age appropriate relationships with peers; lack of social or emotional reciprocity; or a lack of seeking to share enjoyment or interests with others;

2) impairments in communication: delay in normal developmental milestones related to language; impairment in ability to initiate or maintain conversations; stereotyped or repetitive use of language; developmentally inappropriate or non-varied play or interaction;

3) restricted repetitive and stereotyped patterns of behavior: preoccupation with a pattern of behavior that is abnormal in intensity or focus; inflexible adherence to non-functional routines or rituals; repetitive or stereotyped motor mannerisms; or persistent preoccupation with parts of objects; and,

4) onset prior to 3 years of age.

The skill or behavior is not described as totally absent, but rather impaired. Thus, at any level of the functioning spectrum of ASD, portions of the skill or behavior may be present but if impaired significantly, the criteria may be met.

Higher functioning people with ASD may focus repetitively on a specific element of their world, such as the type of car driven by every person they know or about how weather maps are made and what they mean. High functioning people with ASD may have extensive knowledge on a single or handful of topics and the breadth of the details known on this subject may at first appear very impressive. It is important to probe this knowledge to see how broad it is and whether the preoccupation with a single or handful of subjects has interfered with the person's ability to understand the larger meaning or context of the subject matter, as in, an inability to see the forest for the trees. Thus, a knowledge of the kings and queens of England may on its face appear impressive, but a high functioning person with ASD will likely have a limited ability to explain the processes by which power and political intrigue have shaped the passing of the crown.

Investigation of ASD requires very good historical records and interviewing of percipient witnesses who can describe the behaviors of your client in very good detail. Because onset is very young, it will be important to thoroughly investigate early childhood in an effort to document behaviors specific to your client's developmental progress. Even without being able to completely substantiate the onset prior to age 3 (average age of diagnosis for ASD is 6 years old), the pattern of behaviors and the symptoms related to ASD are potentially powerful mitigation evidence.

Since ASD likely includes impairment in nonverbal behavior, this is the first line of investigation: you should be looking for such odd movements or postures or gestures during every interview with the client. Similarly, odd use of words, repetitive behaviors or

comments and inappropriate or odd responses to simple emotional discussion may become apparent to you as you spend time with the ASD client. Errors in word meaning and impaired usage of words in proper social context usage is often seen as well. Also unusual for many of our clients, the Asperger's child may be exceptionally verbally precocious.

Some of the behaviors of ASD often appear to be obsessive-compulsive disorder (OCD) because of the repetitive nature of the some of the manifestations of the disorder. Since people with ASD often display ritualistic and repetitive behaviors, this may appear at first to be OCD. The behaviors of attention deficit disorder (ADD or ADHD) also overlap to some degree with ASD and should be differentiated. Similarly, ASD can often be mistaken for schizotypal or schizoid personality disorder in its lack of empathy, impaired social skills and inappropriate social interactions. ASD symptoms are often more severe in these terms than schizoid personality symptoms. The significance of differentiating them is that ASD is a clear neurological disorder, with a number of potential causes while schizoid personality disorder is a personality disorder that is likely to be used to describe quality of your client's character rather than to be seen as a condition with which your client lives.

Along with the usefulness of explaining the pattern of deficits to the jury which persist throughout life, ASD can also help explain behaviors that the prosecution may attempt to convert to the criteria for conduct disorder (see antisocial personality disorder section below). ASD symptoms in childhood usually include tantrums and emotional volatility behaviors which the prosecution may attempt to convert into indicia of life-long bad acts

and conduct disorder. ASD may affirmatively explain that the cause of these behaviors is not volitional or bad temper foretelling future criminal activity, but rather a specific neurological disease. Additionally, behaviors often seen in childhood include sleep disorders (trouble getting to sleep, staying asleep, getting enough sleep) and serious difficulties interacting with others such that children with ASD often are very isolated, even from family members with whom they live.

ASD can help explain the cause of unusual or odd behaviors as manifest throughout life. Documenting the pattern of behavior over time is essential. Further, people with ASD respond well to highly structured and predictable environments – it is one of the keys to treatment and remediation – and this argument may be especially helpful in a

penalty phase.

Substantial evidence supports the conclusion that people with ASD have executive functioning deficits. Many people with ASD appear to perseverate more, have difficulty with cognitive flexibility and planning on neuropsychological tests of executive function. Research suggests that executive function deficits are common features of those with ASD but not necessarily causative of ASD.

Brain imaging research suggests that people with ASD have brain abnormalities in the amygdala and hippocampus and frontal regions, however there is no evidence to support a role for functional imaging in the clinical diagnosis of ASD.

6. Selected Psychiatric Disorders

It is conceptually useful to separate psychiatric disorders from neurologic or medical disorders, although they certainly overlap and co-exist. We address here the psychiatric disorders most frequently found in capital clients: psychotic and delusional disorders, mood disorders and dissociative disorders.

A. Schizophrenia is a major psychotic disorder. Schizophrenia is estimated to occur in 1-2% of the population. It is characterized by the presence or absence of a number of symptoms involving hallucinations, ideation, delusions, loose associations, flat or inappropriate affect, disorganized behavior, and impaired attention, concentration, motivation and/or judgment. Schizophrenia is noted for heterogeneity in the symptom patterns across time for the individual and between individuals. The symptoms of schizophrenia are generally discussed in two categories, positive symptoms (referring to an excess or distortion of normal function) and negative symptoms (referring to a restriction in range or intensity of normal function).

The onset of schizophrenia is typically in late adolescence or early adulthood, although signs and symptoms may begin to appear in very early childhood. This is termed the prodromal phase, which is a slow and steady increase in symptoms and severity of symptoms towards active phase schizophrenia. Some people have abrupt onset of psychotic symptoms, but most have a slow progression towards psychosis. The majority of people also have acute phases of illness mixed with periods of stability or remission, often described as waxing and waning of symptoms.

All of these changes are important for your social and family history investigation. Although onset of the first psychotic episode is very important, your investigation should seek evidence of the prodromal period. Evidence from this period is crucial for accurate diagnosis of schizophrenia. Similarly, the waxing and waning of symptoms will likely mean that different witnesses have different experiences with your client – will have seen your client in different phases of the illness. This is also important for correct diagnosis and you will want to create a careful timeline to document the pattern of illness.

Positive Symptoms	Negative Symptoms
<ul style="list-style-type: none"> Delusions (unfounded/unrealistic beliefs) Unusual thought content (fantastic or bizarre) Hallucinations (in any sensorium) Suspiciousness/Persecution Grandiosity (exaggerated self-opinion) Disorganized speech (language or thought) Grossly disorganized behavior 	<ul style="list-style-type: none"> Affective flattening Emotional withdrawal Lack of spontaneity Reduced thought/speech productivity Disturbance of volition Poor rapport/lack of empathy Poverty of speech

Similarly important for your investigation is the substantial research on genetic predisposition to schizophrenia. Part of your factual development will necessarily look for multi-generational patterns of mental illness in your client's family. First-degree biological relatives of schizophrenics have 10 times the risk for schizophrenia as the general population. Keep in mind that this is a predisposition rather than a genetic trait, meaning that although at heightened risk, not every biologically related family member will develop recognizable symptoms, illness may skip generations or be inconsistent within generations.

Although more rare, schizophrenia does begin in childhood for some people. Childhood onset of schizophrenia seems to begin gradually, often preceded by developmental disturbances and lags in speech and language. Children with schizophrenia often have visual or auditory hallucinations and have paranoid and bizarre beliefs. Other symptoms seen in childhood include problems paying attention, impaired memory and reasoning, speech impairments, inappropriate or flattened affect, inappropriate expression of emotion, poor social skills, and depressed mood. Such children may laugh at a sad event, make poor eye contact, and show little body language or facial expression. Misdiagnosis of schizophrenia in children is very common.

The brains of teens with early onset schizophrenia appear to be progressively damaged by what researchers call a "back-to-front wave" of neuronal damage. This loss of working brain tissue begins in the back of the brain (in the perception processing areas), and over about 5 years reaches to the frontal areas (executive functioning). Longitudinal research suggests that children who later develop schizophrenia have

neuromotor, receptive language and cognitive developmental delays starting, in some cases, as early as three years old. These children also had emotional and interpersonal problems, but at similar rates to children who developed other psychiatric illnesses.²² Recent brain imaging research²³ has found that over time (measured longitudinally between ages 4 and 22), childhood-onset schizophrenics have a significant loss of total brain volume, driven by drops in frontal gray matter and total gray matter, compared to healthy controls.

The more common pattern of onset is one of an extended prodromal period, with a psychotic incident in the late teens to late twenties. After onset of the first psychotic episode, the acute phase of illness, a period of stabilization and finally a stable period often follow. The acute phase is marked by florid symptoms (hallucinations and/or delusions) and severely disorganized thinking. During stabilization, the symptoms decrease in severity. Symptoms in the stable phase are less severe, sometimes not present, although non-psychotic symptoms such as depression or anxiety may appear.

The popular image of schizophrenia is of someone wandering the streets aimlessly, mumbling or talking incoherently, dirty and unkempt, and acting quite bizarre. It is exceedingly unlikely that this is what you will see when you meet your client. If your client is in an acute phase of illness when you first

²² Cannon, M. et al (2002). Evidence for early-childhood, pan-developmental impairment specific to schizophreniform disorder Archives of General Psychiatry 59:449-56.

²³ Gogtay, N., Giedd, J. and Rapoport, JL (2002). Brain development in healthy, hyperactive, and psychotic children Archives of Neurology 59:1244-8.

meet him/her, you may observe disorganized thinking, tangential thinking, periods of distraction or what appear to be short fugue states which may be auditory or visual hallucinations. In an acute state, your client might not be able to differentiate your voice from hallucinatory voices. In a less severe state, your client may still hear hallucinatory voices but can recognize them as such. More than likely, your client will also be experiencing unusual physical sensations. Speech patterns will also be odd in acute stages, rambling and unedited commentary that sometimes seems pointless or unfiltered skipping from thought to thought. Your client may explain this as his/her brain moving faster than he can talk and a self-perception of enhanced mental acuity. At the same time, the person may be easily distracted, unable to tell important from unimportant stimuli. Sometimes this seems like a lack of interest in the agenda for your interview (at times viewed as arrogance or grandiosity), but it is rather an important symptom of which you will need to take note.

You will also want to listen carefully for delusional and especially paranoid delusional thinking. Delusions are fixed false beliefs or ideas developed to explain events or experiences which often incorporate a kernel of truth. Since the symptoms of schizophrenia include abnormal perceptions and sensations, your client will struggle to explain his/her experience of the world to you. Delusions are false ideas with which the client seeks to explain the world. Some delusions may be very complex and some appear at first to be at least partially accurate reflections of your client's current life. To be delusional, these thoughts do not need to be completely false or totally irrational.

Anosognosia is the clinical term for lack

of awareness of one's own illness (insight). This is a critical concept for understanding schizophrenia, but more importantly for developing techniques for working with your schizophrenic client. This lack of insight is often coupled with a compulsion to disprove any evidence of illness. In many situations, this leads to confabulation to explain observations and facts that are contradicted by strongly held beliefs. Somewhere between 60 and 80 percent of schizophrenics deny being mentally ill. Lack of insight need not be comprehensive. Some schizophrenics will be aware of one component of illness but not others. Lack of insight appears to be related to neuropsychological impairments rather than denial or defensiveness.²⁴

Confronting your client's delusions is, at best, pointless and is likely counter-productive to your client's interests. Sometimes, frustration with a client's inability to see simple facts as you present them, or to turn simple facts into convoluted conspiracies, creates a desire to confront the delusional nature of the client's beliefs. This desire comes from our own confusion about how mental illness works and forgetting that symptoms of mental illness are not stubbornly held-to ideas, but a disease state which the client cannot "think" his/her way out of. Confrontation of your client's beliefs will create a break-down in your relationship and likely push the client towards incorporating you and the defense team into his/her delusional system of beliefs. Instead, you must listen to your client carefully, demonstrate that you have heard and understand his/her concerns, empathize with how difficult daily life is (especially living

²⁴ Amador, X and Shiva, AA (2000) Insight into schizophrenia Civil Rights Law Journal 10(3) 401-15.

with delusional beliefs), and build trust that you will work together to address these problems.

As with all psychiatric disorders, social history will assist in determining whether each of the criteria established by DSM-IV-TR are met sufficient for diagnosis. The DSM-IV-TR Criteria for Schizophrenia are:

A. Characteristic symptoms: Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated):

- 1) delusions
- 2) hallucinations
- 3) disorganized speech (e.g., frequent derailment or incoherence)
- 4) grossly disorganized or catatonic behavior
- 5) negative symptoms, i.e., affective flattening, alogia, or avolition

Note: Only one Criterion A symptom is required if delusions are bizarre or hallucinations consist of a voice keeping a running commentary on the person's behavior or thoughts, or two or more voices conversing with each other.

B. Social/occupational dysfunction: For a significant portion of the time since the onset of the disturbance, one or more major areas of functioning such as work, interpersonal relations, or self-care are markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, failure to achieve expected level of interpersonal, academic, or occupational achievement).

C. Duration: Continuous signs of disturbance persist for at least 6 months. This 6 month period must include at least 1 month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of disturbance may be manifested by only negative symptoms or two or more symptoms listed in Criterion A present in attenuated form (e.g., odd beliefs, unusual perceptual experiences).

D. Schizoaffective and Mood Disorder exclusions: Schizoaffective Disorder and Mood Disorder with Psychotic Features have been ruled out because either 1) no Major Depressive, Manic, or Mixed Episodes have occurred concurrently with the active-phase symptoms; or 2) if mood episodes have occurred during active-phase symptoms, their total duration has been brief relative to the duration of the active and residual periods.

E. Substance/general medical condition exclusion: The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

F. Relationship to a Pervasive Developmental Disorder: If there is a history of Autistic Disorder or another Pervasive Developmental Disorder, the additional diagnosis of Schizophrenia is made only if prominent delusions or hallucinations are also present for at least one month (or less if successfully treated).

DSM-IV-TR subtypes include paranoid type, disorganized type, catatonic type, residual type.

Schizophrenia research has dramatically advanced in recent years both by brain imaging and careful empirical study. Findings have consistently shown alterations in the prefrontal cerebral cortex, cerebellum and temporal cortex. This research points to a dysfunction of the pathways connecting the cerebral cortex, the thalamus and the cerebellum that leads to thought disorder and behavioral symptoms. According to this model, multiple different factors including genetic predisposition and environment contribute to neural dysfunction, the cognitive dysfunction emerges, and the person begins to have symptoms. Neuroimaging studies correlate abnormalities of brain frontal and mesolimbic regions in schizophrenic subjects with deficits in emotional processing and sensory hallucinations.²⁵

Other neurological abnormalities such as perioral dyskinesias and dystonic posturing that are often seen in schizophrenia, may indicate a malfunction of the basal ganglia and thalamus. Frequent blinking and grimacing are also believed to be related to hyperactivity of the dopaminergic system, a critical neurotransmitter system involved in schizophrenia. Another well-documented neurological deficit associated with schizophrenia is olfactory dysfunction, which is related to damage to brain pathways also involved in emotional response and the experience of pleasure.

National Institutes of Mental Health scientists recently reported that the excess dopamine activity found in schizophrenia may be driven by a defect in the prefrontal cortex,

the brain's executive control center.²⁶ This would confirm the primary role that frontal lobe dysfunction plays in behavioral abnormalities seen in schizophrenia.

Schizophrenia is a severe mental illness, but that does not mean it occurs alone. Rather, many people with schizophrenia also suffer depression, suicidal ideation and attempts, anxiety, substance use and report being victims of violence at rates higher than non-mentally ill people.

Treatment issues are first and foremost litigation issues. Although difficult, you will have to assess the effect on your litigation of numerous issues such as, motions to gain appropriate treatment for your client, to oppose forced medication of your client, for a non-testifying expert who will assist your team in illness management and client relations, or for extensions of time because of the special needs of your client and the effect of his/her mental illness on defense team functioning, investigation and preparation.

Some of these issues, such as forced medication in most situations, you will lose outright because of bad precedent, but filing the motion may be useful as a litigation strategy to educate the judge, gain needed assistance or time, or simply to establish a way of talking about your client's mental illness in court that he/she can agree with rather than oppose.

One issue is whether to seek medication for your client. To assist in this decision, it is helpful to know a little about current

²⁵ Sawa, A and Snyder, SH (2002). Schizophrenia: Diverse approaches to a complex disease Science 296:692-5 26 April 2002.

²⁶ Meyer-Lindenberg, A et al (2002) Reduced prefrontal activity predicts exaggerated striatal dopaminergic function in schizophrenia Nature Neuroscience 5(3) 267-71.

antipsychotic medications that are commonly used to treat schizophrenia. In the 1950s, chlorpromazine was discovered to be beneficial in ameliorating the positive symptoms of schizophrenia and allowing patients to live moderately well in the community. Chlorpromazine and those that work like it are called “neuroleptics.” The side effects were quite serious, causing Parkinson’s like symptoms because neuroleptics block dopamine receptors. Neuroleptics are referred to as “conventional” or “typical” medications.

In the 1990s, a second type of medication came into widespread use, the “atypical” or “novel” antipsychotics: clozapine (trade name: Clozaril), risperidone (trade name: Risperdal), olanzapine (trade name: Zyprexa), quetiapine (trade name: Seroquel), and ziprasidone (trade name: Geodon). The atypicals have fewer and less debilitating side effects, and they relieve both the positive and negative symptoms of schizophrenia. Clozapine appears to work better than the other atypicals for most people, with fewer negative side effects. One of the unique features of Clozapine is that it works on both dopamine and serotonin, and this dual neurochemical blocking is suspected to result in its better performance.

B. Other Psychotic Disorders: Along with schizophrenia, a number of other psychotic and delusional disorders are recognized. Many of these are less helpful and less well identified in historical records, often being mis-diagnosed. Alternatively, some of these conditions may be diagnosed in historical records but may reflect diagnoses based on too little historical information. Rather than discuss each in detail, what follows is simply some definitional information that may help you do further research where these diagnoses appear in your case. Most of these conditions

are defined in reference to the criteria which denote schizophrenia:

1) Delusional Disorder is characterized by at least 1 month of non-bizarre delusions without other active-phase symptoms of schizophrenia. Delusional disorder is a less severe illness in terms of number of symptoms, but may be no less debilitating to your client;

2) Brief Psychotic Disorder is characterized by symptoms that last more than 1 day but less than 1 month. Some people have unsuccessfully tried to argue that the nature of a crime is proof that the client suffered a brief psychotic disorder at the time of the offense. The evidence for this had better be overwhelmingly clear if you intend to make such an argument, and based almost entirely on collateral sources of information;

3) Schizophreniform Disorder has the same symptom pattern as schizophrenia but diagnoses people whose symptom persistence does not meet the duration requirements. The course of illness is shorter than in schizophrenia and there is no need to show changes in social functioning. Duration of schizophreniform illness is longer than brief psychotic disorder but shorter than schizophrenia;

4) Schizoaffective Disorder requires that all the criteria of schizophrenia and major depression, manic or mixed episode are met during the same period of illness. As noted in DSM-IV-TR, distinguishing between Schizoaffective and schizophrenia and mood disorders can be quite difficult. The difference is essentially the duration of mood symptoms (in schizophrenia, they are of shorter duration);

5) Psychotic Disorder not Otherwise Specified is for people who have psychotic presentations but do not meet the criteria for any other illness;

6) Psychotic disorder due to a medical condition includes: dementia, delirium, brain tumor or certain illnesses which have delusional and/or hallucinatory features; and,

7) Psychotic disorder due to substances may result from either prescribed medication or illicit drugs which can cause psychosis – for instance, phencyclidine (PCP) is recognized to mimic schizophrenic psychosis.

Each of these will have behavioral symptoms that resemble schizophrenia, although, by definition, the severity and persistence is likely to be less severe.

C. Mood disorders: Mood disorders consist of a number of psychiatric illnesses such as major depression, bipolar disorder and anxiety. They may be either episodic or recurrent, for instance an episode of depression versus major depressive disorder. Each of the mood disorders requires significant impairment in normal functioning and is marked by significant behavioral symptoms.

1. *Major depressive disorder*: Depression is a major cause of disability and suicide in the United States. Nearly twenty percent of people will suffer from depression during some period of their lifetime. For some people, major depression, the most severe form of the illness, is a chronic illness. Onset of major depression can be at any age.

Depression results from a combination of early life experiences, genetic predisposition, and environmental factors. Early life events

such as physical or sexual abuse significantly increase the likelihood that a person will develop major depression. Depression has a strong familial pattern (genetic predisposition).

Associated with depression are disturbances in a number of neurotransmitter systems, at least MAOA (monoamine), norepinephrine and serotonin. The research evidence and symptom patterns suggests that each of these systems is involved in major depression. It appears that major depression is best understood as a common end-point for a variety of underlying problems.

Depressed mood and anhedonia (the inability to experience pleasure) are the hallmark symptoms of depression, but depression has numerous additional somatic markers: sadness, loss of interest, anxiety, irritability, a sense of hopelessness, attention and concentration impairments, and suicidal thoughts. Similarly, a host of physical symptoms often accompanies depression: fatigue or lethargy, sleep problems, headache, gastrointestinal problems, appetite changes, and general body aches and pains.

Major depression is defined as a period of at least two weeks during which the person suffers depressed mood or a loss of interest or pleasure in nearly all activities. Severe major depression may have psychotic features.

The DSM-IV-TR criteria for major depressive disorder include two possible types: single episode or recurrent. The criteria are essentially the same, except for the presence of a single episode versus presence of two or more episodes, and the symptoms must not be better accounted for by, or superimposed on, schizophrenia, schizoaffective disorder, schizophreniform

disorder, delusional disorder, or psychotic disorder. There must be an absence of any manic episode as well. Finally, the severity and context of the depression has to be determined.

The DSM-IV-TR Criteria for Major Depressive Episode are:

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure. Note: do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.

1. depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). Note: In children and adolescents, can be irritable mood.

2. markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others)

3. significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. Note: In children, consider failure to make expected weight gains.

4. insomnia or hypersomnia nearly every day

5. psychomotor agitation or retardation nearly every day (observable by others, not

merely subjective feelings of restlessness or being slowed down)

6. fatigue or loss of energy nearly every day

7. feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)

8. diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)

9. recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

B. The symptoms do not meet the criteria for a Mixed Episode.

C. The symptoms cause clinically significant distress or impairment in social, occupational, or some other important areas of functioning.

D. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hypothyroidism).

E. The symptoms are not better accounted for by Bereavement, i.e., after the loss of a loved one, the symptoms persist for a period of longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

As the criteria set out, factual proof to

substantiate the diagnosis requires information from collateral witnesses as to symptoms. Major depressive disorder also requires longitudinal evidence of the disorder and familial patterns of depression are useful, all of which will be part of the comprehensive social and family history work.

The most difficult aspect of major depression at trial tends to be convincing the jury that, in this particular client, the condition was severe and significantly different than the sad feelings every one has experienced at some point in time. Depression as a general diagnosis tends to be a difficult diagnosis from which to explain criminal behaviors.

Whether you can make it the centerpiece of your mitigation case or not, however, major depression is very important in helping you figure out how to work with your client, how to explain specific behavioral symptoms the jury may observe (e.g., appearing to lack remorse by failing to respond to evidence) and your client's ability to participate in his/her defense.

2. *Bipolar disorder*: Sometimes referred to as manic depression, bipolar disorder affects approximately 3% of people in the United States, and its symptom pattern has been described consistently for centuries. Subtle variations and complex behavior patterns make it difficult to diagnose at times and even more difficult to predict the course and outcome.

Bipolar is a serious but treatable disease of the brain that causes extreme shifts in mood, energy and functioning. Men and women are equally likely to develop this disabling illness that typically emerges in adolescence or early adulthood. However, new research suggests that childhood onset is more common than

previously thought, although often misdiagnosed because the adult criteria do not accurately describe the behavioral symptoms experienced by children. Cycles, or episodes, of depressive and/or manic symptoms typically recur if untreated and may become more frequent, often disrupting work, school, family and social life.

A number of different symptom patterns make up various types of bipolar disorder, but essentially they all contain periods of mania (abnormally and persistently elevated or irritable mood that lasts at least a week and includes grandiosity, decreased need for sleep, pressured talking, flights of ideas, distractibility, increased goal directed activity, excessive involvement in pleasurable activities and social or occupational impairment) or hypomania (abnormally and persistently elevated or irritable mood that lasts at least four days and includes grandiosity, decreased need for sleep, pressured talking, flights of ideas, distractibility, increased goal directed activity, excessive involvement in pleasurable activities without social or occupational impairment), usually intermixed with depression (the patterns vary as to how often each of the manic and depressive states occur and last, and it is possible to have bipolar without having a major depressive episode). Most clinicians and researchers agree that the DSM-IV-TR criteria are overly restrictive, leading to under-diagnosis. It is likely that DSM-V will lower the number of days of mania or hypomania required for diagnosis.

Again, social history information and descriptions of the following types of symptoms over time will assist in diagnosis:

Manic Symptoms	Depressive Symptoms
<ul style="list-style-type: none"> * Severe changes in mood—either extremely irritable or overly silly and elated * Overly-inflated self-esteem; grandiosity * Increased energy * Decreased need for sleep—ability to go with very little or no sleep for days without tiring * Increased talking—talks too much, too fast; changes topics too quickly; cannot be interrupted * Distractibility—attention moves constantly from one thing to the next * Hypersexuality—increased sexual thoughts, feelings, or behaviors; use of explicit sexual language * Increased goal-directed activity or physical agitation * Disregard of risk—excessive involvement in risky behaviors or activities 	<ul style="list-style-type: none"> * Persistent sad or irritable mood * Loss of interest in activities once enjoyed * Significant change in appetite or body weight * Difficulty sleeping or oversleeping * Physical agitation or slowing * Loss of energy * Feelings of worthlessness or inappropriate guilt * Difficulty concentrating * Recurrent thoughts of death or suicide

Bipolar disorder (meaning both the manic and the depressive aspects) may wax and wane over time. As with major depression, longitudinal evidence of mania and depression are required and collateral witnesses to the behavioral symptoms are critical as people are often unaware of how unusual their behavior is (especially during the manic phases of the illness).

The medications that have been found to help control bipolar disorder include mood stabilizers, antidepressants, and anti-psychotic medicines. Mood stabilizers include lithium, divalproex sodium (Depakote) and

carbamazepine (Tegretol). In high doses these medications can stop mania. They are also used to minimize or prevent episodes of mania or depression. The medications must be taken continuously and usually for life.

Antidepressants include fluoxetine (Prozac), sertraline (Zoloft), paroxetine (Paxil), bupropion (Wellbutrin), nefazodone (Serzone), venlafaxine (Effexor), and others. These medications should generally be used along with mood stabilizers. It is generally not appropriate to take antidepressants without mood stabilizers. Antidepressants are used mainly to treat acute major depression rather

than bipolar generally. Once the depressive episode is controlled, mood stabilizers are recommended to prevent future depression.

Surprisingly, electroconvulsive therapy (ECT, previously known as electroshock) has once again become a common and apparently successful tool in the treatment of bipolar disorder. More traditional therapy (cognitive-behavioral) has also proven useful in treating bipolar disorder.

Bipolar disorder, when you are able to sufficiently document the behaviors over time that allow for its diagnosis, is important as a mitigator because it may explain a variety of otherwise negative appearing behaviors: grandiosity, irresponsibility, oversexualization, disregard of self or others by engaging in risky behaviors. Make sure that you can convincingly factually substantiate the presence of a manic episode at the correct period of time before attempting to make use of bipolar to explain any specific incident.

3. *Anxiety disorders*: these disorders are a set of conditions that include: panic attacks, post-traumatic stress (see below, Section E), obsessive-compulsive disorder, generalized anxiety and acute stress disorder. Panic attacks are present in many of the anxiety disorders. A panic attack is defined by a discrete period of intense fear or discomfort in the absence of any real danger that is accompanied by a number of somatic (physiological) or cognitive symptoms. These conditions are included here because they have important effects on behavior and are often quite disabling.

Although not fully understood, current research points to the hippocampus and the amygdala as critical to the neuroanatomy of fear and anxiety. As discussed above, the

amygdala plays an essential role in the acquisition of conditioned fear and the expression of innate and learned fear responses. Through connections to other parts of the brain, the amygdala has a role in automatic and involuntary behavioral responses, which triggers fight or flight responses. Direct electrical stimulation of the amygdala precipitates fearful and panic-like responses.

Additionally, the amygdala and hippocampus connect to the cortex and are involved in the elaboration of contextual information, interpreting the environments associated with conditioned responses, and storing this information in memory. This is one of the important feedback loops that appears to exert an inhibitory effect on the amygdala. Therefore, dysfunction of the prefrontal cortex would lead to a disinhibition of the amygdala.

As with all the other conditions discussed in this manual, the key issues for mitigation related to anxiety disorders are to establish through the social and family history investigation the onset and course of the illness. This will allow you both to tell the story of how this illness shaped your client's life, as well as develop the facts necessary to substantiate the diagnosis.

D. Dissociative disorder refers to a state in which the individual's normally integrated mental functions of consciousness, memory, identity and/or perception are disrupted or severed from each other. Dissociation is a criterion subset for a number of other psychiatric disorders such as the dissociative response to trauma, but a "Dissociative Disorder" is found only when dissociative symptoms occur outside the presence of those conditions.

Dissociation is often described by those who have experienced it as a floating above and/or looking down on oneself (depersonalization) or as a feeling that one's surroundings and the events are unreal (derealization). It may also be experienced as an absence, a period for which there is no memory or explanation (amnesia). A client who describes an experience in any of these ways is likely describing dissociation. You must determine whether the dissociation is caused by an underlying psychiatric illness or meets the criteria for Dissociative Disorder by means of a thorough social and family history investigation.

A common misunderstanding of dissociation is that the dissociated person is unable to act or do things, but a better description is that the person has an absence from conscious awareness of his/her actions, perhaps understood as avolitional activity. This has been termed "dissociated control," a state in which the person can perform learned tasks that require little executive functioning, like dialing a phone number, but those actions take place outside the full functioning of the cognitive system. In effect, lower systems of function are dissociated from higher functions and the complex interactions that should occur within the brain breakdown.

This is an appealing concept, particularly in cases where a client maintains he/she knows nothing about the offense but physical evidence appears to strongly implicate him/her. Some experts have opined that a client experienced dissociation at the time of the offense based on clinical interviewing in which the client appeared unable to recall any specific details of the offense. On cross-examination and in rebuttal through prosecution witnesses, this opinion is nearly impossible to sustain credibly if the opinion is

based solely on clinical interview of the client. As with other conditions, you must establish the reliability of this assertion through collateral sources, for instance, through witnesses who spoke with your client while he dissociated, by a history of dissociation – preferably documented by a mental health expert prior to the initiation of litigation, by documenting specific events that trigger dissociation in your client and which occurred at the time of the offense.

Even with substantial evidence of dissociation, you must be prepared for a rebuttal argument that each action your client took leading up to and during the offense is evidence of volition. This type of rebuttal evidence is sometimes called "forensic behavioral analysis" and consists of a prosecution expert performing a narrative crime reconstruction for the jury. Some prosecution experts have used this technique with unfortunate effectiveness, recasting the dissociated control behaviors, which are properly considered avolitional, as intentional, planning activity. Yet, being right that your client acted avolitionally is not enough and you must be prepared for this type of rebuttal. If, in the end, the jury is left with a complicated psychological explanation for behaviors on the one hand, and a simple series of bad, volitional acts on the other, your client is in trouble.

Dissociation as a useful concept in criminal defense settings is probably most useful when attached to an underlying psychiatric condition which causes it (e.g., trauma) rather than as a stand-alone explanation for behavior.

E. Trauma and PTSD: Trauma and the consequences of trauma (one of which can be post-traumatic stress disorder) are not

synonymous concepts in planning for your mitigation presentation. Instead of meaning the same thing, they are related in a causal fashion: the short and long-term consequences of trauma result from an exposure to a life-threatening traumatic experience. Physical and sexual abuse trauma is usually perpetrated by a family member, but may also be committed by a stranger. Witnessed violence may also be between family members or in the neighborhood. While the research literature has usually focused on a single incident of trauma (witnessing a severe beating), our clients tend to have been chronically exposed to the most serious forms of trauma – repeated physical beatings, multiple sexual assaults or witnessing many friends shot and killed.

The exposure to trauma is an important mitigator because the event itself represents a critical moment in your client's life. "Trauma" basically refers to an experience outside the normal during which the person perceives his/her life to be at risk. Regardless of the long-term consequences of this event, the event itself is important in telling your client's story. For instance, while a client who witnessed his mother stab his father to death will have long-term symptoms related to this traumatic event, your mitigation story must include both descriptions of the event and the description of the changes observed in your client following the event. The event is a compelling and important fact to be presented. Social history investigation is the basis for the story-telling regarding the event of being exposed to trauma regardless of whether that event is sexual abuse at the hands of a stranger, observing friends shot death, being physically beaten by a parent. The event itself is part of your client's story.

Exposure to such trauma has both short and long-term consequences. Trauma has

shaped your client's understanding of every event in his/her client's life because it reshapes how a person sees and experiences the world. You must help to explain how this happened in your client's life. For instance, traumatized children who do not receive treatment often experience difficulty: relating to other people, trusting other people, regulating feelings, forming close bonds, developing a sense of self-worth, believing in or planning of the future, learning and concentrating, and maintaining physical health.

However, the list of symptoms does not even begin to tell the story of how dramatic the impact of trauma can be on day-to-day experience of life. For instance, for people who develop hypervigilance, one of the common symptoms of exposure to trauma, every moment of every day becomes as though he/she is sitting on the edge of their chair, waiting for the other shoe to drop. They can get no respite from being in a heightened state of wariness, senses over-attuned to even the most minor of emotional or sensory stimuli. Hypervigilance results from a central nervous system that, as a result of trauma, has become conditioned to a state of over-excitement. It is a constant state that shapes decisions every day decisions that most people give no thought to at all – where to sit in a restaurant to maximize flight or self-defense, how to maintain physical integrity in a crowded bus, how facial expressions and minor movements may hold significant meaning as to another person's intent. It is a never-relaxed attention paid to the even the remotest of environmental stimuli that most people ignore throughout the day. No decision, and in fact, no experience, is not shaped by trauma for those exposed.

The persistence of the symptoms of

trauma across the life-span means that your client's childhood experience has shaped his/her life-experience. This is the story of trauma in your client's life you need to tell.

Clients, like most trauma survivors, will under-report or minimize the extent of the trauma. This occurs for a number of reasons including:

1. the event is embarrassing or painful to re-live and/or to risk having others know;
2. the person who inflicted the trauma may still have an important role in your client's life and/or the enforced silence within the family or among the group is still in effect; and,
3. the normal psychological processes that kept the client alive through the trauma are still in place and the client has impaired memory of the event, actively or passively uses the process of denial and avoidance, or dissociates rather than being able to talk about the event.

This means, that your investigation must pursue information about trauma carefully and by developing rapport with both the client and the family. Family dynamics are often shaped by abuse and your investigation will have to assess how best to navigate the dynamics to uncover the information which you need.

Long-term Consequences:

In addition to telling the story of the trauma, in many cases you will also need to tell the story of the long-term consequences. One such consequence is often post-traumatic stress disorder (PTSD). Chronic or single incident trauma must be documented in order to find PTSD. Inversely, the experience of

trauma does not mean your client suffers from PTSD. Current research suggests that approximately 25% of people exposed to a life-threatening experience will develop PTSD.²⁷ However, for those who do develop PTSD, and do not receive treatment, symptoms of PTSD will be long-lasting. The essential features of PTSD result from changes that occur following the exposure to a life threatening event. Thus, the DSM-IV-TR criteria begin with a direct personal experience of an event that actually or is perceived to threaten the life of the person and then move to persistent behavioral changes:

A. The person has been exposed to a traumatic event in which both of the following were present:

1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others

2) the person's response involved intense fear, helplessness, or horror. [Note: In children, this may be expressed instead by disorganized or agitated behavior]

B. The traumatic event is persistently reexperienced in one (or more) of the following way:

1) recurrent and intrusive distressing recollections of the event, including images, thoughts or perceptions. [Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed]

²⁷ Carlson, E (1997) Trauma assessments New York: The Guilford Press.

2) recurrent distressing dreams of the event. [Note: In children, there may be frightening dreams without recognizable content]

3) acting or feeling as if the traumatic event were recurring (includes sense of reliving the experience, illusions, hallucinations, or dissociative flashback episodes, including those that occur on awakening or when intoxicated). [Note: In young children, trauma-specific reenactment may occur]

4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event

5) physiological reactivity on exposure to internal or external cues the symbolize or resemble an aspect of the traumatic event

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

1) efforts to avoid thoughts, feelings, or conversations associated with the trauma

2) efforts to avoid activities, places, or people that arouse recollections of the trauma

3) inability to recall an important aspect of the trauma

4) markedly diminished interest or participation in significant activities

5) feeling of detachment or estrangement from others

6) restricted range of affect (e.g., unable to have loving feelings)

7) sense of foreshortened future (e.g., does not expect to have a career, marriage,

children, or a normal life span)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

1) difficulty falling or staying asleep

2) irritability or outbursts of anger

3) difficulty concentrating

4) hypervigilance

5) exaggerated startle response

E. Duration of the disturbance (symptoms in B, C, and D) is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

The key features described here must be proven before a diagnosis of PTSD can be made. It is not enough (even if your expert tells you otherwise) that the traumatic event is documented. To be diagnosed with PTSD, your client must meet these criteria. Your investigative efforts should be geared towards uncovering the behavioral and psychological changes brought about by the exposure to trauma even if your client does not currently meet these criteria.

Although not incorporated into the DSM-IV-TR criteria, the concept of chronic or multiple trauma as different than single incident trauma is very important. The DSM suggests that each incident be considered separately, but clinical experience and research indicates that people exposed to repeated life-threatening events or who are in an environment that poses a repetitive threat are at increased risk of a more severe form of PTSD. This is a helpful way to organize your thinking about trauma and this distinction is important for understanding the consequences

of trauma and for reading the scientific literature on trauma.

Some debate has surrounded PTSD (especially in post-conviction litigation) because it was adopted into DSM only in 1980 (with DSM-III). Innumerable experts have been cross-examined on the issue of whether PTSD was simply added to DSM-III for political reasons or whether it actually represents an illness. There is a grain of truth in these charges – intense lobbying to get PTSD included in DSM as a distinct diagnostic category by Vietnam era veterans and by mental health workers who treated them did occur – but the notion that the diagnosis resulted from lobbying is completely erroneous.

First, DSM diagnoses were subjected to extensive field testing prior to being adopted into DSM. The DSM Sourcebooks (available from the American Psychiatric Press) report on these field trials, the statistical analyses and the comparisons between types of evidence that was relied upon prior to adoption of diagnostic categories and criteria.

Second, PTSD, as a psychological phenomenon, has been documented in the medical and scientific literature since at least 1870. In the United States, major discussion of trauma followed the American Civil War. In 1866, John Erichsen, a professor of surgery at the University College Hospital in London published a series of lectures on railway spine, a concept which first brought together the post-traumatic symptoms that would later become PTSD. Railway spine referred initially to somatic symptoms and subsequently to the psychological symptoms suffered by people in railway accidents. With the dramatic expansion of rail travel in the mid-1800s, the incidence of people involved

in rail accidents increased enormously.²⁸

By the end of the 1880s, a French doctor named Jean-Martin Charcot had written extensively on trauma in male patients and coined the term that persisted throughout the first and second world wars: traumatic neuroses. British and French militaries studied and “treated” war neuroses throughout WWI.

In 1940, Emanuel Miller edited a book entitled *The Neuroses of War*. Miller writes “rich clinical experiences and considered opinions have been derived from the 1914-1918 war which should be placed at the service of the present military medical officers so that they shall see the size of the problem, the character of the cases, the aetiological factors and the methods for treatment.”²⁹ Despite the current literature on PTSD, by the end of WWII, post-traumatic stress disorders were well-documented and well-established in the medical and scientific literature.

Having said that, there is also no doubt that the studies of Vietnam veterans that began in the 1970s were more sophisticated and subtle scientifically, and produced an extensive body of literature on the mechanisms and causes of traumatic stress.

The best approach to understanding PTSD is that the psychological and behavioral characteristics of the disorder are, at the outset, normal and healthy responses to extraordinary circumstances. PTSD is “a set

²⁸ Micale, M.S. and Lerner, P eds. (2001) *Traumatic Pasts: History, psychiatry and trauma in the modern age, 1870-1930* Cambridge: Cambridge University Press.

²⁹ Miller, E. ed. (1940) *The Neuroses War* New York: The MacMillan Co. at p.vii.

of maladaptive emotional, behavioral, and cognitive problems, which are rooted in the original adaptive response to a traumatic event.”³⁰ Thus, where dissociation may be a quite serious social problem for your client as an adult, in response to childhood physical abuse, it was a biologically effective mechanism that kept your client alive. However, what was adaptive functioning in the crucible of the event becomes maladaptive when it persists beyond the event, denying the person a return to normalcy.

In short, the systems of the brain that regulate arousal, vigilance, affect, behavior attention, movement, sleep and startle are reactivated at inappropriate times following traumatic stress. Reactivation can occur in response to a normal stimulus (car backfiring) or during sleep or at any point in time.

Three primary types of trauma are often present in the early life of clients: physical abuse, child sexual abuse and witnessing violence. Each of these types of exposure to violence has been demonstrated to dramatically increase the risk for developing PTSD. Further, most of the research on traumatic events has focused on single incident trauma (a single episode of life-threatening violence exposure), whereas most of our clients have multiple exposures, sometimes chronic and persistent exposure. This type of chronic trauma, for instance, a physically abusive parent who controls the child’s environment for many years, can result in “complex” PTSD. The complexity refers to the multiplicity of arenas in a person’s life that are impaired as a result of this type of

exposure to violence. Along with childhood exposure to violence, adulthood chronic and persistent exposure to life-threatening experiences (such as combat) will also be more likely to cause complex PTSD.

Since not all people exposed to the same type of traumatic event develop PTSD, some attention has been given to what differentiates those people who do from those who do not. In short, a mixture of resiliency and risk factors account for much of the difference. Resilience factors tend to include: access to resources for immediate treatment and intervention, a stable (and non-violent) home environment, individual factors like cognitive abilities, external support systems (presence of a mentor, strong peer group affiliation) and exposure to little or no neighborhood violence. The presence of these factors simply makes it more likely compared to their absence that the trauma will not result in PTSD. For example, a child sexually abused by a neighbor who has strong familial ties, access to resources and treatment and good teachers is less likely to develop PTSD than a child whose parents are the abusers and keep the abuse secret through coercive control over the child.

Long-term consequences of childhood trauma:

- 1) Psychological distress, including: heightened anxiety or depression; fearfulness, paranoia, and hypervigilance; a foreshortened sense of future; psychosis, dissociation, numbing, and denial; impaired sense of self and heightened risk for suicide and self-harm;
- 2) Impaired social relations and inability to interact appropriately with others;
- 3) Significant changes in brain

³⁰ Perry, B.D. et al (1995) Childhood trauma, the neurobiology of adaptation, and “use-dependent” development of the brain: How “states” become “traits” *Infant Mental Health Journal* 16(4) 271-91 at p.278.

development, including smaller brain volume, hippocampal and amygdala damage, left hemisphere abnormalities (memory and verbal ability), limbic system dysfunction;

4) Academic and intellectual problems, including significantly lower IQ compared to non-abused people;

5) Somatization, including: heightened arousal, sleep disturbances, elevated startle responses, out-of-proportion response to stimulus, abnormal neurochemical functioning (affecting serotonin, GABA, catecholamine, cortisol, dopamine, epinephrine and adrenalin);

6) Memory impairments, both related to the trauma and more generalized impairment seen on neuropsychological testing;

7) A variety of information processing impairments may persist: intrusive memories of the traumatic event and disrupted social perception (inability to differentiate between happy and angry faces)³¹;

8) An inability to modulate physiological responses, such that the person likely over-responds to situations (heart rate spikes and fight-or-flight response are activated in a situation where a non-abused person would not have these responses). This includes the conditioned responses to specific stimuli such that the person re-enacts behaviors when exposed

to stimuli that trigger autonomic nervous system arousal;

9) Childhood and adolescent sexual abuse also causes adult sexual dysfunction, and child physical abuse may as well under certain circumstances.

These symptoms do not always occur and may occur without the diagnostic criteria necessary for a PTSD diagnosis. To some degree, nearly all trauma-exposed people have some degree of long-term symptoms, but the severity and manifestation of the symptoms varies for each individual and by the type of trauma.

Taken together, these long-term symptoms change behavior in specific contexts. Some people have attempted to use the dissociative features of PTSD to allege a lack of intent during the crime. It will be a very rare case where retrospective evidence will provide sufficient evidence of dissociation at the moment of an offense. Prosecutors have mostly been successful at poking holes in the evidence that a specific client was in a dissociative state at the moment of the offense. This does not mean you should not consider it, but rather that you must carefully consider the strength of your evidence as you make strategic decisions about the role of mental health in your case.

Additionally, prosecutors have been successful at turning evidence of abuse into aggravation by agreeing that the abuse occurred, but arguing that now your client is “damaged” and was permanently changed by the abuse, and that the abuse has made your client a future danger. You must work to explain why abuse is mitigation and prepare to present additional information to counter these attempts to convert mitigation into

³¹ Pollack, S.D. and Kistler, D.J. (2002) Early experience is associated with the development of categorical representations for facial expressions of emotion *PNAS* 99(13) 9072-6.

aggravation.³²

Association of trauma with other psychiatric conditions: PTSD is under-diagnosed in people with serious mental illness. In numerous studies, the prevalence of current symptoms of PTSD in psychiatric populations ranges between about 30% and 45%, but in those same patients, documentation of PTSD in medical records runs as low as 2%.³³ People with psychiatric illnesses are significantly more likely to have been abused as children and seriously mentally ill people who have been abused as children have more severe psychiatric symptoms.³⁴ In a very large study of severely mentally ill patients, one third of women and over a third of men with serious mental illness reported sexual or physical assaults within the previous year.³⁵

The behaviors associated with PTSD often resemble antisocial behaviors. Although the two conditions might overlap, it is essential that you and your expert consider whether the behaviors are a result of PTSD or antisocial personality disorder (ASPD). If the behaviors result from PTSD, ASPD is not an appropriate

³² See: Crocker, P (1999) Childhood abuse and adult murder: Implications for the death penalty North Carolina Law Review 77 NCL Rev 1143.

³³ Mueser, K.T. et al (1998) Trauma and posttraumatic stress disorder in severe mental illness Journal of Consulting and Clinical Psychology 66(3) 493-9.

³⁴ Mueser, K.T. et al (2002) Trauma, PTSD, and the course of severe mental illness: an interactive model Schizophrenia Research 53:123-34.

³⁵ Goodman, L.A. et al (2001) Recent victimization in women and men with severe mental illness: prevalence and correlates Journal of Traumatic Stress 14(4) 615-32.

diagnosis (not even a co-morbid diagnosis) and should be ruled out. To make this assessment, you must provide your expert with detailed descriptions from witnesses to the behaviors in question.

Major depression, polysubstance abuse and generalized anxiety very often co-occur with PTSD and should be explored. Some people will develop psychiatric illnesses other than PTSD in response to trauma. Although less common, persistent psychosis and transient dissociative states also follow exposure to traumatic events.

Polysubstance abuse is significantly likely to occur in people who have been traumatized, and traumatized people who meet the criteria for a PTSD diagnosis are at increased risk of substance use. Empirical research supports the notion that substance use is a coping strategy which is often quite an effective, although maladaptive, response to trauma. Traumatized people are thought to use drugs and alcohol to regulate distressful emotional and social experiences and recurrent images of the trauma. That is, people self-medicate to deal with the physiological and psychological consequences of the trauma.³⁶ To present this argument, you must have evidence of the age of onset of the substance use in relation to the trauma (the trauma must precede the substance use or changes in substance use temporally).

Traumatic brain injuries can often result from physical abuse, especially abuse suffered as a child. In cases of physical abuse in childhood, consideration should be given to

³⁶ Kilpatrick, D.G. et al (2000) Risk factors for adolescent substance abuse and dependence: Data from a national sample Journal of Consulting and Clinical Psychology 68(1) 19-30.

whether, along with psychiatric symptoms, your client suffered physiological injury to the head and brain.

Exposure to traumatic events early in life can also cause changes in the development of the brain. Changes have been documented in the limbic system (specifically, the hippocampus and amygdala). In addition, because stressful events provoke enormous neurochemical and neuroelectric activity, it appears that the brain become excessively sensitive to re-activation of the specific systems involved in stress responses. This is an important issue for linking adult behaviors to childhood abuse, since the excessive sensitivity created in childhood may make an adult act in objectively unreasonable ways. Similarly, the development stage a person is at when exposed to trauma has an impact on the persistence and severity of symptoms.

Answering the question: “Why your client and not every other person who was abused?” At some point in the presentation of trauma, you will face the question of why your client, but not every person who is abused, killed. You must be prepared to answer this question factually based on a confluence of factors that exposed your client to the most severe forms of trauma and deprived your client of access to resources that would have assisted in overcoming the trauma. This is a model of risk and resilience in which your client is at the upper end of the risk/exposure continuum and at the bottom end of the resiliency/intervention continuum.

Along with scouring social and family history records for evidence and subtle indicators of abuse (for instance, unexplained absences from school, refusal to change clothes for gym, complaints of stomach problems or urinary infections), evidence will

need to be gained by interviewing. Gaining evidence on trauma requires first and foremost establishing rapport and asking open-ended questions.

Experts trained in trauma assessment will use a variety of clinical interview techniques and structured instruments to assess the presence and severity of PTSD. It is not appropriate nor useful to use psychological personality instruments to assess PTSD (e.g., MMPI or MCMI) as they are not reliable or valid for such purposes. Clinical interview for trauma and PTSD can only take place after sufficient rapport has been established and at some point during the course of interviewing, each of the DSM criteria and known symptoms must be addressed. It is important that your client be asked about traumatic experiences in appropriate and non-judgmental ways, and that the questions get at the experience of the event as well as the client’s interpretation of their meaning. Clinical interviews must be based on open-ended questioning. As the criteria for PTSD make clear, PTSD is a condition whose very nature leads the sufferer to minimize or deny traumatic exposure or refuse to discuss specific details of the trauma. To overcome this, the interviewer must establish rapport and ask questions in specific, appropriate ways. Untrained interviewers should be discouraged from asking your client about trauma, because unskilled questioning on emotionally charged topics such as abuse almost always elicits denials because of the shame, embarrassment or fear of re-experiencing. Once the denial is elicited, it becomes tremendously more difficult to create an interview environment which allows you to uncover the information you need.

Among the instruments a qualified expert might use are the Structured Clinical

Interview for DSM-IV Dissociative Disorders Revised (SCID-R) or the Clinician-Administered PTSD Scale (CAPS).

F. Polysubstance abuse: Alan Leshner, former Director of the National Institute on Drug Abuse concluded in 1997: “Addiction is a brain disease.” This is true because drug use changes the functioning of the brain, meaning the brains of drug users are different than the brains of non-users, and because the craving and dependence observed with repetitive use results from specific pathways in the brain. Addiction and use are not the same, of course, since addiction refers to someone who has a compulsion to continue use.

Addiction is a disease of compulsion, and therefore not voluntary. Addiction is defined by a loss of control, where the user does it despite the negative consequences that coincide with use. Craving is another component of addiction wherein getting/using the drug consumes the addicts’ thoughts. Craving is dysphoric, agitating, and feels very bad. Addiction is a chronic, relapsing disease. The natural history of the disease is illustrated by the progressive loss of control over use, so the loss of control occurs more rapidly as the disease progresses.

Nearly all substances of abuse work on the same pathway in the brain, with some important differences discussed below: the mesolimbic reward system. Long term use of nearly all substances causes pervasive changes in both brain function and brain structure, including changes in metabolic activity, neurochemical receptor activity and availability, and responsiveness to stimuli.

Drugs and alcohol have long been associated with violent crime, but most people continue to view substance use as a willful act

that simply makes worse whatever other crimes are associated with being on drugs. Surveys have indicated that nearly 80% of incarcerated people have used drugs or alcohol, many of them during the time of their offense.³⁷ The litigation strategy for explaining substance use must dually explain that your client’s substance use began for a reason other than self-enjoyment and that once begun, addiction processes took away the volitional nature of the use. Substantial scientific evidence exists to indicate:

- 1) many people who later become addicted are introduced to substance use by an older sibling or peer, often times in the context of physical or sexual abuse;
- 2) many people with otherwise untreated or under-medicated psychiatric illness, language or learning disabilities, exposure to violence, brain dysfunction or numerous medical conditions, self-medicate with illicit drugs, prescribed medication and/or alcohol in an effort to control their emotional experience of the world; and,
- 3) once initiated, drugs and/or alcohol alter the way in which the brain functions; all of them create short-term alterations in function, but each also (to a varying degree) causes changes in brain function and/or brain structure that persists across life-span.

The first task of investigating substance use is to obtain a very detailed drug history: when your client began use of any drug or

³⁷ National Center on Addiction and Substance Abuse at Columbia University: Behind Bars: Substance abuse and America’s prison population 1998.

alcohol, who introduced your client on that first occasion, when and how much of each substance was used, how often, where did your client obtain it, what was the quality or purity of the substance, how did your client deal with periods of withdrawal or inability to procure. You want to obtain extensively detailed information of your client's substance use over the course of his/her life following that first use.

In addition to creating a substance use timeline, you will want a parallel timeline of childhood abuse, trauma exposure, mental illness and brain injury symptoms to explore onset and course of mental illness in relation to substance use. You should not rely on your client's self-report of the temporal relationship between substance use and onset of mental illness or brain dysfunction (or, for that matter of substance use as most people under-report their own use), but you will want to start with your client and then develop collateral evidence.

Further, because substance use has been demonstrated to be strongly linked to parental and familial use, you will want to develop information on the multigenerational use of substances within the family that may have an environmental and/or genetic influence on your client's use.

Substance abuse is strongly associated with childhood abuse, neglect and trauma. People who suffer abuse, neglect and/or trauma are significantly more likely to use drugs and alcohol. The leading view as to why this is suggests that people seek to self-medicate the symptoms that follow such trauma or seek to avoid every day life and dealing with the consequences and experience of the abuse. Especially in cases where treatment for the trauma was not available, it

is more likely that the traumatized person will seek a means for dealing with the day to day consequences of trauma and abuse. The critical task for the investigation is to demonstrate that the onset of the substance use followed the trauma.

DSM-IV-TR defines intoxication and abuse of each kind of substance (a different criteria set for each type of substance which can be abused or to which a person can become dependent – see DSM-IV-TR for each set of criteria). All of the important substances which people use (see below) have the capacity to create dependence. The common criteria across all substances are that the person have recently used the substance, that the use causes significant impairment or distress, that the person experiences repeated social or interpersonal problems as a result of the use, and that the person experiences some sort of craving (physiological or not) to seek continuing use.

Most drug use researchers consider the notion of physiological dependence versus psychological dependence to be an obsolete distinction based on a decades old lack of understanding about how substance use alters brain function. The hallmark of this approach depended on the presence of withdrawal symptoms when a person was detoxified. However, it is now generally accepted that all major substances of abuse produce withdrawal symptoms and all have specific effects within the brain when a person goes through the detoxification process. Evidence of withdrawal is very important to corroborate use and to assess the extent of addiction. Along with documenting use patterns, you will want to document the onset of withdrawal symptoms when developing the substance use history (especially if part of your mitigation case relates to substance use at the time of the

offense or time of arrest).

1. *Methamphetamine (meth)* is a stimulant drug that became significantly cheaper and easier to use in the early 1980s. Meth can be smoked, injected or ingested. Acute effects of meth last between four and twenty four hours (depending on dose and quality). Like all stimulant drugs, meth forces the release of the body's own natural stimulants without reason or demand from the body, that is, without reasonable need for the energy. Meth's principle biochemical action on the brain is to mimic and increase the effects of epinephrine (adrenaline) and to prompt the massive release of dopamine into the brain.

Meth damages dopamine terminals and transporters in the brain, causing a dysregulation of the dopamine system. This appears to be most significant in the orbitofrontal cortex. Current research suggests that this dysregulation explains both the loss of control and the craving pattern associated with meth use. Chronic meth use causes frontal lobe brain damage (reduced neuronal activity and density).

The initial effect of the release of excess stimulating chemicals is euphoric. However, activation of the sympathetic nervous system produces a "fight or flight" behavior pattern in which intoxicated individuals misinterpret, overreact, and misjudge stimuli. Methamphetamine keeps the chemicals circulating by blocking the normal re-absorption, so the stimulant effects are both exaggerated and prolonged.

High doses or prolonged use of meth can cause toxic psychosis. These symptoms are almost always the result of high potency meth used either intravenously or by inhalation (smoking) and occur more often among

chronic users. Psychosis occurs when the vague drug-induced fears crystallize into a fixed delusional system. Typically, the psychosis includes delusions and/or hallucinations occurring in the absence of intact reality testing, sometimes with disorganization of speech and behavior. The delusions (erroneous beliefs involving distortion or exaggeration of thought) suffered by methamphetamine users are paranoid delusions, meaning that the individual believes he is being watched, persecuted, or attacked when he is not. The auditory hallucinations (distortions or exaggeration of perception) most often reported are vague noises, voices, and occasional conversations with the voices. Speech is pressured, tangential, and fragmented reflecting internal disorganization.

Amphetamine psychosis can be expected to have several substantive effects on behavior; these can include euphoria or affective blunting, changes in sociability, hypervigilance, interpersonal sensitivity, anxiety, tension, or anger, stereotyped (repetitive) behaviors, impaired judgment, or impaired social or occupational functioning. Cognitive abilities are similarly affected, including paranoia and hallucinations, compounding the severe irritability and poor impulse control. As a consequence of the drug-induced psychotic state, the individual is unable to separate fact from fantasy (psychosis), and is subject to irrational fear (paranoia) that can trigger responsive (and impulsive) behavior from him. Events occur, and behaviors are seen that would not occur if it were not for methamphetamine intoxication.

Even in very low doses, methamphetamine produces a variety of significant physiological responses that are a consequence of the increase of epinephrine (adrenaline) in the

system: pulse and blood pressure increase; the pupils dilate, and finally, body temperature rises. Methamphetamine increases motor and speech activity as well as nervousness and irritability. Alertness and excitement increase, and consequently, in high doses the drug produces prolonged periods of wakefulness, even when the individual is physiologically exhausted.

Meth-impaired neuropsychological function does not appear to recover following an extended period of abstinence, meaning that damage caused by chronic meth use is permanent.

2. *Phencyclidine (PCP)*: PCP is a dissociative anesthetic agent developed in the late 1950s as a surgical anesthetic (it was subsequently banned from use). PCP has come in and out of widespread use a number of times in the last twenty years. It is commonly used with other substances (e.g., marijuana is sometimes dipped in PCP). It is generally reported that people use PCP to achieve the feelings of numbness and dissociation (e.g., as a self-medication). Ketamine, another dissociative anesthetic drug, works in much the same way as, and with similar effects to, PCP.

PCP intoxication causes a psychosis that is indistinguishable from schizophrenia. Psychosis is the threshold effect of PCP and people who are intoxicated with PCP are often unpredictable and volatile. In some people, PCP acts as a sedative, although for most people it causes an extreme agitation.

Along with psychosis, low doses of PCP cause mood fluctuations, distortions in thinking, memory loss, impaired judgment, agitation, impaired perception, disorientation and hallucinations. Psychosis can last from

approximately twenty four hours up to six weeks from low dose usage of PCP.

Chronic use of PCP causes permanent alterations in mood and behavior as well as in brain functioning. PCP binds to opioid receptors which are very prevalent (high density of receptors) in the hippocampus and frontal cortex. This binding blocks the neurotransmitter NMDA (N-methyl-D-aspartate), a subtype receptor for the excitatory neurotransmitter glutamate. This explains the behavioral effects observed.

3. *Cocaine*: Cocaine is a stimulant. Cocaine can be taken by any of a number of routes (ingestion, inhalation, absorption) and reaches the brain relatively quickly (by inhalation more quickly than intravenous or intranasal).

Chronic cocaine use produces impairments in learning and memory, visuospatial ability, mental flexibility, processing speed and ability to abstract (frontal-subcortical related functions). Chronic use of cocaine has been shown to cause functional and structural changes in the brain, primarily in the frontal cortex and basal ganglia. Chronic use also can cause a variety of medical problems (seizures, optic neuropathy, intracerebral or subarachnoid hemorrhage, myocardial infarction, ischemia, brain atrophy). Cocaine also produces axonal degeneration (see Section 3 above), especially in areas very high in acetylcholine receptors.

Chronic use may also cause toxic psychosis. Cocaine users develop tolerance which usually leads to the use of increasing amounts to accomplish a similar intoxication. This in turn leads to a greater rate of degeneration in brain function and a greater likelihood of developing psychotic symptoms.

Cocaine induced psychosis is characterized by paranoia with ideas of reference, delusions, fear, anxiousness, impaired cognitive functioning, impaired judgment, inappropriate responses to stimuli, impaired impulse control, and hallucinations.

Cocaine, like methamphetamines, works by raising the amount of dopamine released in the brain. It then blocks the process by which dopamine would normally be taken out of the system, resulting in an on-going stimulation.

Chronic cocaine use also causes a kindling effect, which means the brain develops an anticipatory posture and is primed for further dosing. Cocaine use following the development of this kindling state is often seen to have a heightened impact (in contrast to tolerance).

4. *Alcohol*: More than any other substance, alcohol is involved in a tremendous number of injuries and deaths in the United States. Ethyl alcohol is a psychoactive drug that is just as powerful as the more notorious illegal drugs. Physiologically, alcohol produces a general and nonselective depression of the central nervous system. Although it is not completely clear how alcohol works, it appears that alcohol first depresses the neurons in the brain stem which control the higher centers of the cerebral cortex. Thus, alcohol first affects the cerebrum which in turn controls complex human behavior. The result of low doses of alcohol is therefore impaired perception, thought, judgment, organization, and fine motor processes.

Alcohol produces disinhibition in behavior. With higher doses of alcohol disorientation increases; impairment of judgment and distortion of thought increase in severity. As the user's blood alcohol level

increases the individual becomes progressively incapacitated; first fine motor function, and then, gross motor function is affected. The consequence of very high doses of alcohol in the body is the suppression of respiration and finally, death.

Chronic alcohol ingestion may irreversibly destroy nerve cells leading to permanent impairment of cognition, memory, and motor control. Chronic use of alcohol can also produce a specific dementia called "Korsakoff's syndrome."

The long term consumption of alcohol has psychological consequences for the user. Prolonged drinking produces anxiety and depression. All of the symptoms associated with depression and anxiety: insomnia, irritability, palpitations often appear. Drinking temporarily relieves these symptoms increasing the difficulty of maintaining sobriety in the long term user. Drinking alcohol also exacerbates any pre-existing depression and/or anxiety from which the individual suffered.

Alcohol occasionally produces amnesia or blackouts. The amnesia is anterograde: a failure to make new memories. During a blackout, the individual has relatively intact remote and immediate memory, but experiences a specific short-term memory deficit for which he is unable to recall events that happened 5 or 10 minutes before. Because other intellectual faculties are well preserved, they can perform complicated acts and appear normal to the casual observer.

Because many jurors will be familiar with the effects of alcohol, it can be extremely difficult to convince jurors of the significance of the effects related to alcohol use. Alcohol use alone tends to not be a particularly

compelling mitigator, but as with many conditions, it may be important to telling your client's story and humanizing him/her. It may also have an interactive effect with psychiatric and neurologic illnesses.

5. *Marijuana*: Marijuana is generally believed to have the most limited long-term effects on the brain and to be the most benign of illicit drugs. Recent research has sought to challenge this view because marijuana appears to affect the stress and reward systems in the brain in much the same way as heroin, although not as successfully as heroin. Nevertheless, there is an insubstantial body of research on brain damage from long-term marijuana use.

THC is the active ingredient in marijuana and it passes from the lungs into the bloodstream, and then to the brain. THC connects cannabinoid receptors in the brain. Many cannabinoid receptors are found in the parts of the brain that influence pleasure, memory, thought, concentration, sensory and time perception, and coordinated movement. They are most common in the cerebellum, hippocampus, cerebral cortex (especially the cingulate, frontal, and parietal regions), and the basal ganglia.

Marijuana does cause cognitive, sensory and perception, mood and motor alterations in the period immediately following use. Also in the short-term, most users show lowered levels of aggression. Marijuana may also cause long-term memory impairment, although the evidence is not conclusive.

The "gateway" drug argument (that marijuana use naturally leads to use of harder drugs) may be employed in certain circumstances, although you are better off developing evidence of contextual reasons to

explain changes your client's use patterns. Like alcohol, marijuana alone has not been a particularly compelling mitigator, but is important to investigate and consider as part of the overall picture of your client. It may also have an interactive effect with psychiatric and neurologic illnesses.

6. *Heroin*: Somewhere between thirty seconds and two minutes after injection (or inhalation), heroin crosses the blood-brain barrier (ingested or snorted takes slightly longer). Heroin is converted to morphine and binds rapidly to opioid receptors in the brain. The opioid receptors are normally used by endorphins (a sort of endogenous morphine). Endorphins relieve stress and pain. Morphine has an analgesic and sedating effect in the body.

Pure heroin, which is a white powder with a bitter taste, is rarely sold on the streets. Most illicit heroin is a powder varying in color from white to dark brown. The differences in color are due to impurities that have been left from the manufacturing process or the presence of additives. Another form of heroin known as "black tar" heroin is available most often in the western and southwestern U.S. This heroin, which is produced in Mexico and Hawai'i, may be sticky like roofing tar or hard like coal, and its color may vary from dark brown to black. The color and consistency of this type of heroin result from the crude processing methods used to illicitly manufacture this substance.

Users typically report feeling a surge of pleasurable sensation, a rush. The intensity of the rush is a function of how much drug is taken, the purity of what is taken and how rapidly the drug enters the brain and binds to the natural opioid receptors. Heroin is particularly addictive because it enters the

brain so rapidly.

The intense rush is followed by sedation (which causes the “nodding” effect often observed). The pupils constrict (miosis) and the purity of the heroin is reported to effect how small the pupil becomes (purer heroin leads to greater constriction). A number of hormones are released in response to heroin in the body.

Heroin is very addictive because, as with endorphins, the brain signals for continuing supply of morphine once exposed. Tolerance does occur and increasing amounts of heroin are required to achieve a similar euphoric sensation. Heroin withdrawal is well enough known to have become the focus of numerous popular portrayals in movies and books.

7. *Inhalants (Glue/Gasoline)*: The term inhalant refers to any of almost a thousand different commercially available products that can be sniffed or smelled and which have an intoxicating effect. Inhalant of organic solvents produces a temporary stimulation and reduced inhibitions before the central nervous system (CNS) depressive effects begin causing dizziness, slurred speech, unsteady gait, and drowsiness. Impulsiveness, excitement, and irritability may also occur, along with hallucinations, and delusions. Users report experiences of euphoria culminating in a short period of sleep. Delirium with confusion, psychomotor clumsiness, emotional instability, and impaired thinking are seen. The intoxicated state may last from minutes to an hour or more.

Symptoms associated with inhalant use include belligerence, apathy, impaired judgment, and impaired functioning in work or social situations, dizziness, drowsiness,

slurred speech, lethargy, depressed reflexes, general muscle weakness, and stupor. Nearly all inhalants produce anesthesia, a loss of sensation and even unconsciousness.

Chronic use causes long-lasting damage to the brain, including damage to the protective sheath (myelin) around certain nerve fibers in the brain and peripheral nervous system. This extensive destruction of nerve fibers is clinically similar to that seen with neurological diseases such as multiple sclerosis. The neurotoxic effects of prolonged inhalant abuse include neurological syndromes that reflect damage to parts of the brain involved in controlling cognition, movement, vision, and hearing. Cognitive abnormalities can range from mild impairment to severe dementia. Other effects can include difficulty coordinating movement, spasticity, and loss of feeling, hearing, and vision.

One of the reasons that inhalant use causes such severe brain damage is that the substances that are inhaled to gain the sense of euphoria also include quite toxic solvents and metals. Huffing gasoline used to be a major cause of lead poisoning. Severity of brain damage depends on the specific substance inhaled.

Toluene sniffing (also known as huffing), which has historically been disproportionately prevalent in the Latino and Native American communities, causes this euphoric feeling as a result of changes wrought in the dopamine system. Toluene is a solvent found in many commonly abused inhalants including airplane glue, paint sprays, and paint and nail polish removers. The damage to the brain is diffuse and pervasive from huffing toluene.

8. *MDMA (ecstasy)*: Ecstasy (sometimes referred to as X) is widely used as a

recreational drug. X is a synthetic, psychoactive drug with both stimulant (amphetamine-like) and hallucinogenic (LSD-like) properties. In the past few years, a great deal of attention has been focused on X, largely because of its use by middle-class, suburban teenagers.

X targets brain serotonin. The serotonin system plays a direct role in regulating mood, aggression, sexual activity, sleep, and sensitivity to pain. For people who take MDMA at moderate to high doses, depletion of serotonin may be long-term, but the research on long-term brain damage is currently confused due to laboratory testing errors in the primary research conducted. Persistent deficits in serotonin would likely be responsible for long-term behavior effects that some users report. New research has now found that X also has a dramatic effect on dopamine in the brain.

Many of the risks users face with MDMA use are similar to those found with the use of cocaine and amphetamines: psychological difficulties (including confusion, depression, sleep problems, drug craving, severe anxiety, and paranoia) and physical symptoms (including muscle tension, involuntary teeth clenching, nausea, blurred vision, rapid eye movement, faintness, and chills or sweating).

Research on the long-term effects of X use is still very new and uncertain, but tends to show learning and memory impairments that persist as well as impulsivity.

Dual Diagnosis (Substance use and Psychiatric Illness): As mentioned elsewhere, there is a very high co-occurrence of psychiatric illness and polysubstance use and neurologic illness and polysubstance use. It is essential to establish, during the development

of the social and family history, the onset of both substance use and symptoms of neurologic and psychiatric illnesses. It is important not to confuse this co-occurrence with one or the other disorder, but at the same time, symptoms of one condition (e.g., psychiatric illness) must not be inaccurately considered in support of another condition (e.g., substance use) unless causation is dually occurring. For diagnostic purposes, it is not enough to determine that a symptom occurred and the person was taking drugs and suffered a psychiatric illness. Rather, to properly diagnose, it would be important to assess whether it was either the drugs or the psychiatric illness or both that caused the symptom.

G. Personality disorders: DSM-IV-TR states that a “Personality Disorder is an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual’s culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment.”³⁸

Simply put, personality disorders are traits which compose the core, definitional characteristics of your client. Under the DSM multiaxial diagnostic system, the difference between Axis I, often referred to as clinical disorders, and Axis II, often referred to as personality disorders, might best be understood to be the difference between a state and a trait: a state being conditions that could remit (or wax and wane) and a trait being a core definitional characteristic of the organism; or more clearly, it is the difference between defining a condition a person has compared to defining who a person is.

³⁸ DSM-IV-TR at p.685.

Since 1980 when DSM III instituted the multiaxial system, Axis I has been defined generally as comprising “clinical” disorders and Axis II the “personality” disorders. This distinction is crucial to understanding what happens when Axis II diagnoses are brought into court because rather than being conditions which might be effectively treated or which may in fact change over time, the personality disordered individual is being described for the merit of his or her character and the essential nature of his or her personhood. Therefore, rather than a story about a person who has certain experiences, it is a story of the quality of the person predicated on the notion that they are socially disordered.

In short, the reason that personality disorders do not assist you in presenting mitigation evidence is because such presentation asks the jury to judge the merit of your client’s character, rather than the more complex story concerning how and why your client has come to this situation.

In the pre-DSM-III period (before 1980), clinical descriptions of personality disorders focused on traits rather than behavior. Personality disorders under this view were juxtaposed against a non-theoretical notion of normalcy (the well-adjusted person). There was little empirical work on whether these personality types existed, whether clinicians were identifying the same conditions as deviant or whether the disorders reflected communal concerns or the concerns of a few. For instance, much of the early work on personality disorders focused on immigrant groups (Italians, Irish and European Jews) and the desire to “treat” them in such a way as they became “normal.” The criteria for diagnosis were based on personality traits such as selfishness, lack of remorse, or incapacity for loyalty, seen in the clinical

interview.

In 1980, the promulgation of DSM-III changed some of this, shifting the focus to observable behaviors rather than traits. This change was purportedly brought on by empirical research. However, extensive debate remains about the empirical support for the criteria and diagnoses on Axis II, particularly those in Cluster B.

DSM-III separated the clinical (Axis I) and personality (Axis II) disorders onto separate Axes. The move to a separate axis occurred in 1980 and was explained as an effort to keep clinicians from ignoring the personality disorders and to highlight them. DSM-III defined the personality disorders as “inflexible and maladaptive and cause either significant functional impairment or subjective distress.” It suggests that combinations or constellations of traits constitute a personality disorder, but only when they are inflexible, maladaptive or cause significant functional impairment.

As a general rule, personality disorders are not helpful to your case. Many clients have been previously diagnosed with a personality disorder and you are likely to have to deal with the appropriateness of that diagnosis versus your current evidence for mental illness. In all situations, comprehensive social history evidence is necessary for your mental health expert to make a determination about the appropriateness of diagnosing a personality disorder.

DSM-IV-TR recognizes that personality disorders should be considered subsidiary to the clinical disorders. Since the criteria may often overlap between clinical and personality disorders, DSM-IV-TR cautions that a “Personality Disorder should be diagnosed only when the defining characteristics ... do

not occur exclusively during an episode of an Axis I disorder.”³⁹ Thus, Axis I disorders must be considered and ruled-out before the expert concludes your client suffers from a personality disorder: “The enduring pattern is not better accounted for as a manifestation or consequence of another mental disorder.” In short, Axis I clinical disorders trump the personality disorders. If a clinical disorder is present and explains the behaviors, no personality diagnosis should be made. Further, the enduring pattern must not occur solely as a result of substance abuse or medical condition (neurological disease or head trauma), and when the personality changes occur as a result of extreme stress, post-traumatic stress must be considered.

Cluster A Personality Disorders: Paranoid Personality Disorders include three types: paranoid, schizoid and schizotypal. In general, these are like lesser included conditions of clinical disorders. They are defined by a pervasive pattern of distrust, paranoia and suspiciousness. People with these conditions are said to appear odd or eccentric. Cluster A disorders must not have occurred exclusively during the course of schizophrenia, mood disorder with psychotic features, or psychotic disorder. Therefore, like the personality disorders overall, every effort must be made to develop facts which will allow the expert to properly evaluate the presence of a clinical disorder that rules out the personality disorder.

Cluster B Personality Disorders: Often called the dramatic disorders, Cluster B disorders include Antisocial Personality Disorder, Borderline Personality Disorder, Histrionic Personality Disorder and

Narcissistic Personality Disorder. Serious conceptual and legal problems related to Cluster B are discussed below in the section on Antisocial Personality Disorders. In short, there is poor evidence currently that four disorders that make up Cluster B are in fact distinct disorders that can be accurately identified. As with all the personality disorders, clinical disorders must be ruled out prior to diagnosis.

Cluster C Personality Disorders: Cluster C disorders include Avoidant Personality Disorder, Dependant Personality Disorder, Obsessive-Compulsive Disorder and Personality Disorder Not-Otherwise-Specified. People with these conditions are said to appear anxious or fearful. The same rule-out provisions apply to these disorders as well.

³⁹ DSM-IV-TR at p.688.

7. Overview of causation issues (brain-behavior focus)

A crucial piece of telling the story of why the jury should care about brain-behavior relationships in your client is explaining, either explicitly or implicitly that the brain impairment altered the way in which your client behaved. It is critical to do more than simply state that your client has some form of brain impairment or brain damage. You must both explain how it altered your client's behavior and experience, and why the jury should care. Some brain impairments may have no relationship to behavior. For brain impairment to be successfully mitigating, you should present evidence that links the impairment to behavior. You do not have to prove that the brain injury caused the specific crime, but you must explain how the impairment changed the way in which your client understood his environment, was able to respond to unexpected changes in the environment, or perhaps undermined the client's ability to choose another course of action. This is referred to as causation: that the brain impairment caused disordered behaviors.

The key issues that must be developed to make such an argument include:

A) *Strength of Association (e.g., injury clearly related to behavior)*: Technically, strength of association refers to a mathematical measurement. A number of techniques are available to assess the strength of association depending on the type of data and the techniques used for gathering it. Strength of association cannot be accurately measured for a single individual in a single situation. It is a measure that requires a significant number of people be studied carefully. However, a piece of your argument

will rely on what is known about the strength of the association between the type of impairment your client has, and the type of behavior in which he has engaged. To do this, you will need to understand the scientific literature specific to your case as it relates to strength of association.

For instance, if your client had never engaged in bad behavior before suffering an injury to his frontal lobes, but after suffering the injury engaged in erratic and impulsive behaviors, you would argue that a very strong association exists between the injury and the behavior. You would also be able to put forward a great deal of scientific evidence which supports (based on empirical research) a more generally recognized relationship between frontal lobe damage and changes in behavior. In this way, you are arguing that the strength of association between your client's brain impairment and his behavior is very strong.

B) *Biological Plausibility (e.g., injury to this area would cause these problems)*: Another component then is to be able to articulate how the specific injury causes the specific problem. Often, this is an area attorneys prefer to defer to experts to explain. Doing so, however, generally leads to two problems for your case: either a very technical description that the jurors cannot understand or a discussion that loses the focus on storytelling. To articulate biological plausibility in a way that helps your case, you must be able to explain the details of what the injury is and what is known about those behaviors, and link that information directly to the experience of your client.

For instance, evidence about the frontal lobes and the testing used to assess whether your client has frontal lobe impairment must

be linked to telling the story of how life is experienced by someone who is unable to properly regulate impulsivity. The clinical literature has many stories about such people. One story is about a man who, following frontal lobe damage, drove to work every day in precisely the same way. He drove the exact same route, the exact same speed. In fact, this is the sort of post-injury rehabilitation that traumatic head injury patients should receive - information on how to manage every day life given the new experience of a brain that now works differently. For quite some time this man was able to get to work without incident. One day, a road construction crew had blocked off the lane in which he drove every day for repairs. Unable to adjust to the new information that he saw right in front of him, the man drove directly into the blocked off area, without braking or changing speed. He was unable to adjust to the changed environment because of the damage to his frontal lobes.

Biological plausibility works in reverse as well. It is not plausible that an injury to the frontal lobes alone would change a person's ability to perform on a math test. You do not want to be in a position of arguing contrary to the scientific evidence about the injury.

C) Time Sequence (e.g., behavior starts after injury): Time sequence is the piece of causation with which most people are familiar. Most lawyers know to look for pre-injury evidence that contradicts the argument that the bad behavior began only when the brain injury occurred. Thus, if the client has been an arsonist since the age of twelve, but suffered a head injury at the age of twenty-five, it is not plausible that the injury caused client to start fires.

This is not to say that the injury did not

alter the client's behavior or interact with a pre-existing set of circumstances, it simply means that causation should not be implied if the behavior precedes the injury.

D) Dose-response Relationship (e.g., more is worse): Toxicologists are fond of saying that the dose makes the poison. By this, they mean that in small enough quantities, almost any substance can be taken into the body and handled by the normal functioning of the organism. Some poisons, cyanide for instance, have a very steep dose-response relationship. At very small quantities, the body responds very little. As the dose increases only marginally, the body quickly begins to respond quite significantly. Very small doses of cyanide can kill a person within a few minutes. Cigarette smoke, on the other hand, has a very shallow dose-response relationship. Illnesses, although serious and often lethal, occur from smoking over long periods of time. In the scientific literature, unlike in your client's life, the dose-response evidence will be presented while controlling for all other variables. Researchers try to minimize the interaction between the poison of study and other poisons or the context of its delivery. Your client does not experience the world in that way though and you have to explain to the jury that it is not only a specific dose of one poison, but that dose interacting with a plethora of other factors which you will also explain. This may make for complicated science, but it is the heart of your story.

Dose-response relationships are crucial to causation discussions. If a person is exposed to limited quantities of cigarette smoke, and the scientific evidence indicates that only long-term exposure causes illness, you will have to demonstrate that your client was exposed to a significant enough dose to cause the behaviors of interest.

Another example that many attorneys are familiar with is a situation where all the siblings in a family were severely physically abused. Prosecutors often argue that if abuse made people kill, all of these siblings would have killed, but only your client has so it is not the abuse that matters. Dose-response is one way in which to address this issue in some situations where your client was singled out for a quality of abuse that was substantively different than his siblings (higher dose) or where a specific quality (low intellectual functioning) about your client interacted with the abuse in a way that made it different for him than for his siblings.

E) *Confounding (e.g., making sure it is the injury, not something else)*: The worst possible situation for an attorney is to have not considered a simpler and better explanation than the one being offered to the jury. Confounding occurs when researchers are looking at one thing, but forget to look at something else that might be causing the behaviors or cannot tell between the first thing and the alternative. Lawyers have this same problem. Much of the penalty phase is trying to convince a jury that the human sitting before them arrived where he is as the result of simple, understandable things. Prosecutors try to argue that evil or complete disregard for others led your client to murder, whereas you are trying to explain the inevitability that could have led anyone to your client's position.

If you are offering scientific or medical evidence, you must make sure that what you are suggesting cannot be undone by a simpler explanation. For instance, some people have suggested that having children causes grey hair. If a study were to be done, it is true that most people who have had children do get grey hair. A much greater percentage of

people without children do not have grey hair. Thus, children cause grey hair. It is a story that could be told. However, the simpler and clearly better explanation is that people get grey hair as they age. Since most people do have children, those with children in this study will be older than those without children. Therefore, age and not children are a better explanation for grey hair. In this silly example, age confounds the study of greying hair leading to a false conclusion. You must guard against confounding in making arguments about causation.

8. Biopsychosocial history and Experts

Biopsychosocial history does not just serve the task of pointing the way to deficits and hardships your client has suffered, but also serves the mission of fleshing out the client as a human being with a full range of human qualities, with potential to grow and become better, and with experiences and qualities that draw out empathy not just sympathy.

Defense counsel bear primary responsibility for providing mental health experts with a complete and accurate biopsychosocial history. Generally, mental health experts do not gather and develop a criminal defendant's biopsychosocial history. The experts need the defendant's history and cannot perform a professionally competent and reliable evaluation without it, but they do not put the history together themselves. In a clinical setting, psychiatric social workers perform this task. In criminal cases, defense counsel is responsible for developing the history.

Without a complete history, experts should inform defense counsel that they cannot conduct an evaluation or form an opinion. However, some experts will try to do an evaluation anyway – based on whatever skimpy (and probably) inaccurate history can be gleaned from the client in a clinical interview, on the clinical interview itself, and perhaps on a battery of psychological tests. Evaluations conducted on this basis – without a complete biopsychosocial history – are likely to be inaccurate and more likely than not damaging to your case. Most people are not able to remember completely their life histories, will remember events inaccurately, and will omit critical facts such as incidents of head trauma. Certain important areas of

information – such as prenatal history, whether their mother drank or took drugs during the pregnancy, early developmental issues, injuries, illness, and exposure to environmental toxins and genetic predispositions to illnesses – may not be known to or by the defendant.

A defendant's narrative about his or her life provides important information but not nearly all the information that is needed for a complete biopsychosocial history. Especially in the capital case context, where the prosecution is likely to challenge the truthfulness of your client's self-report, you must obtain corroborating information from collateral sources. Only through thorough gathering of records (e.g., maternal and paternal medical records, pregnancy and birth records, medical and mental health records, school records, social welfare agency records, military and employment records, criminal records, environmental toxin reports, and community-based dysfunction records (such as incidents of violence in the neighborhood)), interviewing of scores of people (parents, grandparents, siblings, knowledgeable extended family members, teachers, previous health care providers, friends, coworkers, military buddies and commanders, police officers, jail and prison personnel and fellow inmates, co-perpetrators in criminal offenses), and analysis of all this information can a complete biopsychosocial history be developed. This process must be undertaken in each capital case.

Studies conducted over the past few years involving post-trial interviews of jurors from capital trials have established what many defense lawyers have already learned from experience: juries find lay witnesses far more credible than experts in relation to mental health issues. Accordingly, when evidence is

presented on mental health issues, defense counsel must structure the presentation so as to establish the defendant's life history and relevant behaviors and emotions through life history witnesses and other lay witnesses (e.g., co-defendants, crime scene witnesses, jail witnesses, family members, neighborhood friends). Defense counsel also must present expert testimony to provide information to the jury that is beyond the expertise of lay people, but expert testimony must be tailored to the lay testimony and provide an interpretation of the defendant's history, behaviors, and emotions as established by lay witnesses.

When an expert testifies, counsel bears considerable responsibility for the effectiveness of the expert's testimony. There is no way to catalogue completely all the elements of effective expert testimony. However, there are some that can be identified as consistently important in each case:

1. Experts need to know, without hesitation, the data, their conclusions, and the reasoning process by which they reached their conclusions. This seems obvious. However, many experts are extremely busy and, in trying to accommodate the need for their services, take on too much work. Unless counsel stays in close touch with the experts, there is a risk that they will show up unprepared or under-prepared.

2. Experts need to use common, non-technical, simple direct language. There will invariably be technical terms and terms of art that need to be used and explained. However, they must be explained and they cannot be relied on in testimony without frequent re-explanation. The persuasiveness of an expert's testimony cannot be made to be dependent on lay jury's abilities to master technical and specialized information.

3. Experts' opinions must be grounded continually in the facts. All the potentially relevant facts must be taken into account. The facts supporting the opinion and the facts contrary to the opinion must be addressed. This principle applies to both of the reasoning processes the expert employs (e.g., the process that leads to clinical conclusions), and the process that leads to forensic conclusions.

4. Experts must present their testimony through short answers to focused questions. Narrative answers to broad questions are ineffective.

5. Experts must highlight and emphasize the important facts about the client and relate their opinions to those facts. Their opinions must be presented as an aid to understanding the facts, not as the primary facts.

Mitigation evidence, because it is about the telling of the life story of your client, must focus on the onset and course of illness and impairment. Behaviors at the age of four may not forecast later behavior directly, but they may tell a story about how you client experienced the world, a story that the jury can understand, sympathize with and ultimately empathize with. Increasingly, neurologic and psychiatric illnesses and impairments cannot be diagnosed without an accurate and thorough multigenerational social and family history; but more importantly, no compelling mitigation case can developed without such a social and family history because you, and subsequently the jury, must come to understand the life of the client. There is absolutely no substitute for accomplishing this work, and counsel must play a leading role in getting it done and understanding its meaning.

9. Brain Function Testing

No mental health investigation or litigation can be brought without the involvement of testing. Unfortunately, many people simply hire an expert to “assess” the client. This will result, in almost every case, in personality testing. Figuring out your client’s personality function is the least helpful type of mental health evidence you can obtain, may distract from uncovering evidence of major mental illness, and may bring into your case negative evidence about your client that was not previously in your case. To avoid this, you need to carefully choose your expert(s) and then work closely with the expert in developing a testing protocol based on the social and family history evidence that you have developed, and based on the theory of your case and the type of evidence that will be beneficial to your case.

No testing protocol works effectively without a comprehensive social and family history. As Adolph Sahs, an eminent neurologist, is reported to have taught, “If you have thirty minutes to see a patient, spend twenty-eight minutes on history, two minutes on the examination, and no time on the skull X-ray or EEG.”⁴⁰ Similarly: “It may come as a surprise to many that it is generally not the sophisticated imaging studies of the brain nor the complicated neurophysiological testing (bioelectrical testing) that typically leads to the most information about neurologic condition. Rather, it is the history which actually gives the practitioner the most

diagnostic information.”⁴¹ You must have a comprehensive social and family history prior to determining what tests to give and you must provide your expert with this information before he/she can interpret any testing data with reliability.

The key issue in testing is to understand what type of mental impairment or dysfunction you are seeking to substantiate through tests so that you choose the correct type of testing protocol. To do this, you will have to:

- become familiar with the various types of testing and the scientific literature that describes what different types of testing are capable of doing reliably;
- interact with experts in a process that is reciprocal: do not abdicate to experts decisions about what tests should be given, what background evidence they need or the process of evaluation. Make sure your expert has prior testing which is already in your case;
- generate hypotheses about your client’s functioning that you can test;
- understand the cultural, language, education, age and gender issues that will effect the reliability of any test given and its interpretation.

You will need to participate in an open exchange with your expert about these issues. Keep in mind that mental health experts probably do not know the legal standards

⁴⁰ As cited in Caplan, L and Hollander, J (2001) The Effective Clinical Neurologist Boston: Butterworth/Heinemann at p.25.

⁴¹ Starzinski, D.T. The Forensic Neurological Assessment of Traumatic Brain Injury, in Murrey, G.J. ed. (2000). The Forensic Evaluation of Traumatic Brain Injury Boca Raton, FL: CRC Press at p.24.

related to your litigation, so you need to work with them on what the standards are for the different types of legal issues you are pursuing.

Different types of testing require different types of experts. If you simply hire a forensic expert, you will get whatever preferences that person has for testing. Instead, your defense team will need to look for the right type of expert and you may consider hiring a mental health consultant who will work with your team in making these decisions but will not testify.

Below is a table which lists the main types of testing. Tests for mental functioning can be considered in three broad categories: neurological tests, neuropsychological tests and psychological tests. Although to some degree each type produces some evidence of how your client functions, the first two types:

- provide significantly different ways of talking about your client;
- have significantly different scientific reliability and validity;
- will open your case to differing types of rebuttal and cross-examination.

Neurological Testing	Neuropsychological Testing	Personality Testing
<p>Brain Imaging</p> <p>Structural Imaging</p> <ul style="list-style-type: none"> ▶ CT ▶ MRI <p>Functional Imaging</p> <ul style="list-style-type: none"> ▶ fMRI ▶ PET ▶ SPECT ▶ EEG ▶ qEEG <p>Physical Exam</p> <ul style="list-style-type: none"> ▶ Babinski and Grasp ▶ Snout and Suck ▶ Gait and Posture ▶ Motor Reflexes ▶ Visual Tracking and Eye Movement ▶ Vision, Smell and Hearing ▶ Physical Injury/Malformations ▶ Strength ▶ Lateral Dominance ▶ Nerve Conductance 	<p>Halstead-Reitan Battery Category</p> <p>Tactual Perception</p> <p>Seashore Rhythm</p> <p>Speech-Sounds Perception</p> <p>Tapping</p> <p>Trail Making A and B</p> <p>Aphasia Screening</p> <p>Sensory Perceptual</p> <p>Luria-Nebraska Motor Function</p> <p>Rhythm</p> <p>Tactile Function</p> <p>Visual Function</p> <p>Receptive Speech</p> <p>Writing/Reading/Arithmetic</p> <p>Memory</p> <p>Intellectual Processes</p> <p>Other Key Tests</p> <ul style="list-style-type: none"> ▶ Wisconsin Card Sort ▶ Go - No Go ▶ Continuous Performance Tests ▶ Rey-Osterreith Complex Figure ▶ Wechsler Memory Scale ▶ WAIS or WISC ▶ Wide Range Achievement Test (WRAT) ▶ malingering tests 	<p>Common Personality Instruments</p> <ul style="list-style-type: none"> ▶ MMPI (Minnesota Multiphasic Personality Inventory) ▶ Rorschach ▶ TAT (Thematic Apperception Test) ▶ PCL-R (Psychopathy Checklist - Revised) ▶ MCMI (Millon Clinical Multiaxial Inventory) ▶ CBCL (Child Behavior Checklist) <p>Common Screening Instruments</p> <p>Bender-Gestalt</p> <p>Mini-Mental Status Exam</p>

Neuropsychological Batteries: Neuropsychological assessment measures various aspects of cognitive functioning, including intelligence, academic functioning, attention and concentration, verbal and visual memory, language functioning, visual spatial functioning, motor abilities, sensory-perceptual processing, abstract reasoning and

executive functioning (such as planning, self-monitoring, inhibition of impulses, and mental flexibility). Neuropsychology is a subspecialty of psychology that requires unique training and clinical experience, and has its own professional organizations, journal, credentialing and ethical guidelines. You must retain an expert with the qualifications to

administer and interpret neuropsychological exams.

There are two types of approaches to neuropsychological testing: standardized batteries and flexible batteries. The two primary standardized batteries are the Halstead-Reitan Neuropsychological Battery and the Luria-Nebraska. The Halstead-Reitan is widely used and research has consistently shown it to be the most reliable such battery in differentiating impaired from non-impaired people.

Flexible batteries vary according to the person administering the examination and therefore each examination will vary as to reliability and validity depending on the specific tests given. Additionally, even some well-qualified experts choose odd combinations of tests when doing flexible batteries which may not fully explore your client's functioning. If your expert insists on a flexible battery, you must participate in the test selection with your expert to insure that key areas of brain function are assessed. The recommended technique is to use the Halstead-Reitan battery, followed by flexible testing to follow-up indications of impairment based on results from the Halstead-Reitan.

The Halstead-Reitan battery consists of a number of different tests selected by the examiner on the basis of the reason for the referral. However, the battery is normally constructed around a core of five tests: (1) The Category Test; (2) The Tactual Performance Test; (3) The Seashore Rhythm Test; (4) The Speech Sounds Perception Test; and (5) The Finger Oscillation or Finger Tapping Test. Other tests should be added to augment the core battery, including the Trail Making Test, Wisconsin Card Sorting Test and the Rey-Osterreith Complex Figure Test.

Administration of the entire battery typically requires from six to eight hours. Interpretation of the Halstead-Reitan should include scores of each test as well as at least one of the index scores (Impairment Index, General Neuropsychological Deficit Scale, Average Impairment Rating) which can be calculated to provide an indication of overall impairment.

These additional instruments, given in combination with the Halstead-Reitan, will provide significant information about frontal lobe function: Wisconsin Card Sort Test, Rey-Osterreith Complex Figure Test, Go/No-Go and Continuous Performance tests, Trails A and B.

Some experts, most often clinical psychologists without specialized training in neuropsychology, use screening tests to determine whether to conduct more neuropsychological testing (e.g., Bender-Gestalt or Trails Test alone). None of the screening tests is reliable and by definition will test only certain functions of the brain. No determination about brain function should be based on these instruments. You should discourage any expert you hire from giving these screening tests and instead retain a trained and qualified neuropsychologist to administer a complete battery.

Finally, remember that your expert will interpret the pattern of functioning across all the tests – your client will almost certainly not perform identically badly or well across the entire battery. It is essential to understand this pattern and not become over-focused on one or two tests.

Neurological Examinations: The standard neurological physical examination is so well established in the literature that there is little

to say about it. Properly performed, the physical examination tests each of the cranial nerves and reflexes. Neurological exams will generally include the following: a mental status assessment, cranial nerve assessment (a test for each of the twelve cranial nerves), motor system testing (including muscle strength and tone, coordination and gait), reflex testing, and sensation testing.⁴²

Neurological testing for frontal lobe damage has come under attack by prosecutors in a number of cases, however, primarily because of the role of frontal lobe impairment in unrestrained behaviors. Prosecutors sometimes challenge the specificity of neurological tests in determining frontal lobe damage. Frontal lobe dysfunction can and should be assessed by neurological examination as well as neuropsychological testing. Both types of tests are important and can provide corroborating evidence of frontal lobe damage. Assessment of reflexes, gait, posture, muscle tone and olfactory disturbances provide reliable evidence of frontal lobe damage.⁴³

Academic tests: The most widely utilized test for assessing academic level is the WRAT (Wide Range Achievement Test). Tests of academic level do not provide much information specifically about brain function,

but they are important for interpreting neuropsychological testing that has education-level norms and adjustments. The WRAT provides information on actual educational level in arithmetic, spelling and reading.

Intelligence tests: Nearly everyone has heard of IQ testing, but it is essential to remember it is a measure of only one part of the brain. The most widely used intelligence test is the Wechsler Adult Intelligence Scale - III (WAIS, for ages 16-89) or the Wechsler Intelligence Scale for Children -III (WISC, for ages 6-16) or the Wechsler Preschool and Primary Scale of Intelligence - Revised (WPPSI, for ages 3-7). Many other tests for measuring intelligence are available (Stanford-Binet, Raven, Shipley or Kaufman), although the WAIS is more widely used). These tests are essential to assess functioning, but take on heightened importance in cases where you suspect mental retardation.

WAIS-III is the most widely used and probably the most widely accepted in terms of reliability and validity. The components of the WAIS-III include verbal subtests (vocabulary, similarities, arithmetic, digit span, information, comprehension and sequencing) and performance subtests (picture completion, digit symbol-coding, block design, matrix reasoning, picture arrangement, symbol search and object assembly). The test-taker is given scores on each subtest, overall scores for verbal and performance tests, and single combined IQ score which includes all subtest scores. Overall, the WAIS-III assesses verbal comprehension, working memory, perceptual organization and information processing speed. Significant differences between verbal and performance scores (more than 15 points) may be indicative of organic brain damage, but no conclusion can be based on this discrepancy alone, it should only be

⁴² Devinsky, O and D'Esposito, M (2004) Neurology of Cognitive and Behavioral Disorders Oxford: Oxford University Press; Caplan, L and Hollander, J (2001) The Effective Clinical Neurologist Boston: Butterworth/Heinemann.

⁴³ Devinsky, O and D'Esposito, M (2004) Neurology of Cognitive and Behavioral Disorders Oxford: Oxford University Press; Damasio, A (1979) The Frontal Lobes, in Clinical Neuropsychology Heilman, K.M. and Valenstein, E. eds. New York: Oxford University Press.

used to prompt further neuropsychological testing of brain functioning.

Objective personality tests: These include the Minnesota Multiphasic Personality Inventory II (MMPI-II) and the Millon Clinical Multiaxial Inventory III (MCMI-III) which are probably the most common of the objective personality tests. Objective here refers to the administration of the exam and means the test is standardized rather than open to clinical interpretation (as in the subjective tests). These tests use standardized questions and can be computer scored. Results appear on a chart and in some cases a computerized narrative which provides interpretation based on character traits associated with the scores. At their best, these tests group your client with other people who have answered questions in a similar way. The manual carefully advises, however, that an elevated scale is insufficient to diagnose a person, meaning, for instance, that an elevated schizophrenia scale does not mean the person is schizophrenic. These tests are widely used and many mental health professionals find them helpful in clinical practice. However, in a forensic setting, personality tests produce unreliable evidence and evidence that simply addresses your client's character and worth. These tests do not help you explain your client's life or experiences in an effective way.

Additionally, recent research that has re-evaluated these instruments in light of admissibility questions suggests significant problems in reliability and validity. Writing about the MCMI-III, Rogers et al commented that the MCMI-III had fundamental problems

in scientific validity and error rates.⁴⁴ Similarly, researchers have questioned the forensic use of the MMPI-II because of problems of validity and reliability when used in forensic settings. Further, numerous questions have been raised in the literature about the scoring procedures, computerized scoring and interpretative cookbooks that are often used to analyze the MMPI-II.⁴⁵

Particularly on the MMPI and MCMI, the setting of the assessment can have a dramatic impact on how a person answers the questions. None of the objective personality tests has been normed or assessed for use in a custodial setting. The MMPI is not normed for use with people with IQ's under 80. Some psychiatric disorders may severely skew the results of these tests (e.g., trauma or psychotic disorders)

Subjective personality tests: These tests (e.g., the Rorschach, thematic apperception test, draw-a-person) are unreliable under any circumstances, clinical or forensic. Literature reviews have suggested that these tests have insufficient scientific merit and you should not administer them to your client.⁴⁶ Nevertheless, many mental health experts like these tests and have mistaken beliefs about the

⁴⁴ Rogers, R., Salekin, R.T. and Sewell, K.W. (1999) Validation of the Millon Clinical Multiaxial Inventory for Axis II disorders: Does it meet the *Daubert* standard? Law and Human Behavior 23(4) 425-43.

⁴⁵ Senior, G. and Douglas, L. (2001) Misconceptions and misuse of the MMPI-2 in assessing personal injury claimants NeuroRehabilitation 16:203-13; Allard, G. and Faust, D. (2000) Errors in scoring objective personality tests Assessment 7(2) 119-29.

⁴⁶ Lilienfeld, S.O., Wood, J.M. and Garb, H.N. (2000) The scientific status of projective techniques Psychological Science in the Public Interest 1(2) 27-66.

strengths and weaknesses of them or mistaken beliefs about the experts own special skills in making interpretations. Subjective personality tests offer the tester the greatest degree of interpretative freedom to the person administering them and are clearly the least reliable and scientifically weakest.

Malingering tests: An assessment of malingering is essential (see Section 11). As well as correctly interpreting the neuropsychological battery itself for evidence of malingering, a standard neuropsychological examinations should probably include tests that assess for malingering (e.g., Rey-15, Rey Word Recognition Test, Portland Digit Recognition Test, Dot counting, Symptom Validity Test). Each of these tests assess the degree of effort and theoretically, each can be completed without error by most unimpaired people with even severely demented patients performing in the chance error range on symptom validity tests. Malingering can also be detected by scoring patterns on some of the standard neuropsychological batteries. Experts should use these tests as well as clinical judgement based on affect and behavior during the testing sessions to assist in reaching determinations about malingering.

Finally, there are a number of increasingly popular, stand-alone instruments which are designed to assess malingering. These include the Test of Memory Malingering (TOMM) and the Structured Interview of Reported Symptoms (SIRS). These tests may be useful to counter prosecution allegations of malingering under certain circumstances.

Even seriously ill people may feign symptoms and it is important that the degree of exaggeration and intent be assessed as best the examiner can do so.

Brain Imaging

EEG is a test of brain function based on electrical activity. If given during seizure activity, it is tremendously useful. Standard EEG's use leads (which monitor electrical activity) on the outside of the head which is not particularly efficient for assessing certain parts of the brain. Nasopharyngeal leads (leads placed deep inside the nasal passages) are more effective but uncomfortable and often not available. qEEG measures brain function in the same way, but uses computerized analysis to compare the data to known standards. It provides information on more subtle forms of dysfunction.

PET scans measure glucose uptake and blood flow by marking glucose with a radioactive agent and tracking how the marked glucose is used in the brain. PET images are analogous to CT and MRI, but demonstrate function rather than structure. PET demonstrates areas of normal and abnormal energy utilization. PET provides excellent resolution and very precise images of the brain's functioning.

MRI (magnetic resonance imaging) and fMRI (functional MRI) use the same basic technology to produce images of the brain. MRI provides excellent structural images by subjecting the brain to a magnetic force (which aligns atomic nuclei) and then sending radio wave pulses through the brain which are absorbed by some nuclei and change the energy state of nuclei. If you are looking for lesions or scar tissue or malformation, MRI is very good. In general, MRI is superior to CT with a few exceptions (e.g., calcification, subarachnoid lesions, skull fractures). fMRI assesses the oxygenation status of hemoglobin in the brain. This test is given while the client is performing certain types of tasks (motor

tasks or experiencing sensory stimulus). This provides an excellent and precise image of the brain in action. Resolution of the image using fMRI is by far the most subtle and detailed of any imaging technique. This technique also allows for repeated images over a period of time so that assessment can be made of the brain at rest and during performance of tasks. Interpretation of the images remains debated by experts.

MRS (Magnetic Resonance Spectroscopy) works on a similar technology to MRI but provides a better image of neurometabolism and neurochemical functioning. MRS is very good for examining NAA (N-acetyl aspartate), CH (Choline) and Lactate in the brain.

SPECT (Single Photon Emission Computed Tomography), like PET measures blood flow and glucose uptake but the images are not as finely detailed as PET and are often more difficult to interpret. SPECT resolution is poor compared to other techniques and it provides less robust information.

CT (Computer assisted tomography) imaging is cheap and more labs have the technology. CT is effectively an X-ray of the brain structure and allows for assessment of the integrity of the blood-brain barrier (e.g., stroke, tumors, inflammation and some neurological disorders). It is useful for bone structure abnormalities and calcification. CT scans are not particularly sensitive (meaning, many people with brain dysfunction will appear normal on CT scan). It is also reported to be less effective than MRI for brain stem, cerebellum and temporal lobe imaging.

Summary of testing issues: Although there may be exceptions to the rule, the rule is that you will want neuropsychological and physical neurological testing in nearly every

case. The reliability and validity of these types of testing are generally well-established and well-documented. For instance, since at least the 1970s, standard neurology textbooks have recommended physical examination of reflexes to assess for frontal lobe brain damage.⁴⁷ Similarly, there is an extensive literature on neuropsychological testing.⁴⁸ Work with your expert to determine what neuropsychological tests are appropriate and be sure to be prepared to defend the reliability and validity of the tests you have decided to have administered as the prosecution may well challenge you on admissibility.

The types of evidence which neurological and neuropsychological testing provides are the most directly useful because they address both your client's impairment and how your client functions in the world. These types of testing allow you to frame your client's mental functioning in the context of how he/she functioned in the world.

You may have psychological testing from prior evaluations of your client. You will have to deal with this evidence and should come up with a plan for addressing this evidence if it is in your case. As a rule, you do not want additional psychological testing because the character evidence it produces will neither help you tell your client's story nor clarify your client's circumstances. In short, it will not assist your case.

The question of obtaining brain imaging in

⁴⁷ Heilman, K.M. and Valenstein, E. eds. (1979) Clinical Neuropsychology New York: Oxford University Press.

⁴⁸ See: Lezak, M.D. (1995) Neuropsychological Assessment, 3rd Ed. New York: Oxford University Press.

your case may be more complicated. Structural imaging provides a view of the physical make-up of the brain. Structural imaging does not provide information on how the brain functions. Structural imaging of a corpse may show no malformations and it would not be possible to tell that the brain was dead, only that it was physically intact. Functional imaging provides images of specific types of systems in the brain (for instance, glucose uptake).

Some additional issues with brain imaging: Despite the apparent appeal of a picture that tells your story, brain imaging often leads people to believe there are shortcuts to factual mitigation. In fact, quite the opposite is true: to make effective use of brain imaging, you must provide the neuroradiologist with extensive historical information and you must have undertaken neuropsychological tests prior to the imaging. No competent decision can be made as to whether to do imaging without having first undertaken an extensive work-up. Prior to making a decision about imaging, you and your expert must discuss whether the evidence of brain impairment will be bolstered by imaging. To do this, you and your expert must have completed a multi-step process:

1. Compiled a complete and comprehensive, multi-generational family, medical and social history;
2. With your expert, you should have detailed discussions about the physiological, neurological and psychiatric systems involved in your client's functioning;
3. With your expert, begin the process of assessing diagnostic criteria and defining a psychiatric diagnosis. At this stage, this

should be a hypothesis generating exchange with your expert;

4. Neuropsychological testing should be undertaken based on the prior three steps. This generally should include a complete Halstead-Reitan Neuropsychological Battery and appropriate additional tests based on the above steps;

5. With your expert, assess the neuropsychological findings in the context of all the other information you have obtained.

At this point, you are ready to consider whether brain imaging will be of benefit in your case. Keep in mind that while the pictures are interesting and can offer demonstrative evidence to the jury, it also has serious limitations such as:

a) brain imaging risks distracting the jury from the truth of your presentation which is a story about your client and how the brain impairments caused or influenced certain behaviors;

b) brain imaging does nothing to humanize your client and risks engaging the jury in technological issues that undermine your effort to re-focus the jury on the human being sitting next to you;

c) brain imaging is not better than neuropsychological examinations for localizing brain impairment and does not allow you to discuss behavioral functioning as well as neuropsychological testing does. Neuropsychological assessment permits a description of the behavioral outcome of the deficits which brain imaging is currently unable to do;

d) brain imaging of people with major mental illnesses more often than not appear normal. MRI scans of 6200 psychiatric inpatients at McLean Hospital found positive findings, meaning an abnormal MRI image, in only 1.6% of patients;⁴⁹

e) as a result of normal differences between brains, brain imaging will almost certainly result in a battle of expert opinion which differs on the question of whether the image is abnormal or normal except in situations where a lesion is present;

f) affect during testing may cause changes in the results, which means that brain imaging of clients with psychiatric illnesses might not be as reliable as needed. Repeated testing is likely to demonstrate differences in the individual if the affect changes between test periods, for instance, if the person is very depressed during one test period and euphoric during the next as is possible with a client with bi-polar disorder;

g) medication effects are serious but not well-known at this point in time for prescribed as well as self-administered drugs. Recent research even found changes in brain function among a group of patients given placebo rather than medication;⁵⁰

h) significant variations occur based on a person's age. Normal brain function changes with age and can be reflected in imaging, but it is also true that on some tasks, the young use one hemisphere and adults use both hemispheres of the brain;

i) very little is known about brain plasticity which means that no one can say what a brain should look like in an adult who suffered a childhood traumatic brain injury – the brain may adapt or repair itself in some situations and not in others or may adapt by “recruiting” parts of the brain to do tasks not typically associated with that task;

j) except in certain instances, brain imaging does not address the question of etiology of behaviors, meaning, while brain imaging has the potential to show a specific way in which the brain is malfunctioning or malformed, it cannot answer the question, in most cases, of why or how the brain got that way;

k) many very serious brain diseases cannot be identified with confidence by current technology (such as, Parkinsons, Alzheimers, migraine, depression, bipolar disorder, epilepsy, or many psychotic disorders);

l) most of the brain imaging technologies are very new, and as a result some serious questions about how to interpret what is being observed remain. The state of the science does not support diagnosis of brain impairment or dysfunction solely based on brain imaging. While specificity is high for a handful of conditions (e.g., temporal lobe epilepsy, Alzheimer's disease, schizophrenia), sensitivity is very low for brain imaging of these conditions

⁴⁹ Rauch, S.L. and Renshaw, P.F. (1995) Clinical neuroimaging in psychiatry Harvard Review of Psychiatry 2:297-312.

⁵⁰ Mayberg, H.S. et al (2002) The functional neuroanatomy of the placebo effect American Journal of Psychiatry 159(5) 728-37.

and unknown for many more common conditions (e.g., depression). This means that if the image resembles a pattern known to be schizophrenia or Alzheimer's disease or temporal lobe epilepsy, the individual has that disorder; but if the brain image appears different from the standard for those few illnesses, it does not mean the individual is normal. Some people with those illnesses will have brain images that do not appear typical for the condition. This dramatically increases the risk that brain imaging in criminal cases will undermine your other evidence of brain dysfunction or impairment when in fact you have correctly assessed your client to be impaired;

m) the images which you want to show the jury are usually composite images or reconstructions of data; for instance Blood Oxygen Level Dependent (BOLD) fMRI relies on measuring oxygenated versus deoxygenated hemoglobin some number of seconds after the neural activity occurs; in most cases, those data are then compared to statistical norms of a small number of people developed by the Montreal Neurologic Institute; many of these comparisons use "spatial normalization" software to make comparisons; some of the most commonly used software programs conform the data in such a way as to actually mask or hide lesions that should appear; other of the common software programs conform the brain to a standardized map (spatial normalization) which means the image does not directly correspond to what the individual's brain actually looks like;

n) brain imaging measures, at least theoretically, brain activity; however, research shows that people asked to

repeatedly perform the same task use different areas of their brains when the task is novel than after time; imaging technology cannot, at this time, differentiate between efficiency of brain function and deficit; similarly, and probably more importantly, poor performance or disorganized performance in most cases will correlate with more brain activity not less - meaning that a damaged area of the brain may "recruit" other parts of the brain to help with tasks that a normal brain would more simply perform; on the image, this will appear to be enhanced brain activity rather than deficit and is very difficult to correctly interpret;

o) finally, scan patterns, no matter what the pattern indicates, do not predict behavior. As with other evidence, many people who have no behavior problems do have brain images that appear abnormal while others, with severe behavior problems have scans that appear within the normal range.

Many jurisdictions have held certain types of brain imaging inadmissible under Daubert, although the courts have almost always made these determinations on the issue of "fit" rather than reliability. Many of these criticisms are accurate given the state of the science; for instance, based solely on brain imaging, no opinion could reasonably attempt to explain behavior.

A few of the issues can be understood by some simple concepts:

Normal Brain Variance: Intra-individual variance refers to changes in the how the brain responds to tasks during repeated tests. There will be a range of results for each individual

across multiple imaging tests even when the individual's brain is "normal." What "normal" means is a range that fits a probabilistic, normal bell curve. It is not currently possible to reach conclusions about normality or abnormality unless it is significantly outside range of normal results, and it is not yet possible to know what the precise parameters of the range are. The best example of this comes from one of the innovative bits of information that imaging has provided: the normal brain, when shown novel information responds differently than when the information is not novel.

Inter-individual variance refers to differences in the imaging of normal brains and also the similarity between images of abnormal people with vastly different impairments. In short, healthy people's brain look different, often significantly so. Normally functioning individuals may have brains that look very different from each other and it is often difficult to interpret the brain image as normal variance or so different as to constitute abnormality.

Inter-group variance refers to differences between normal and abnormal brain images. Although some images allow for firm conclusions, many images will not because there is currently not certainty about what a schizophrenic brain looks like compared to a depressed brain.

Fit: Daubert and its progeny establish two key gate-keeping roles for judges: assessing the reliability of the test and assessing case fit. While reliability has been the focus of most law review articles and a great deal of commentary, most judicial opinions to date have focused on fit. Many cases where brain imaging has been excluded never address the issue of reliability, excluding the tests based

on whether the science answers the particular question in the specific case. Thus, *US v. Mezvinsky*, 206 F.Supp.2d 661 (E.D.Pa. 2002) rejected PET scans because of case fit (the experts agreed that the impairment shown on the PET did not address the question of whether Mezvinsky was lying: "both [experts] agreed that no study exists that links diminished capacities in various parts of Mezvinsky's brain to any specific disorder there is, therefore, no evidence that Mezvinsky's PET-identified brain abnormalities had any pertinence to his capacity to deceive....") and in *Jackson vs. Calderon*, 211 F.3d 1148 (9th Cir. 2000) the court found that PET was not generally accepted to diagnose PCP abuse and that the PET image could not answer the legal question of intent ("No evidence was introduced that the PET scan proves that Jackson was unable to premeditate or form a specific intent at the time of the shooting. The PET scan evidence could, at best, only establish that Jackson suffered some PCP-induced brain abnormality, the effect of which on Jackson's capacity for higher thought is not demonstrated.").

Little question remains as to whether brain imaging accurately measures what it is supposed to (e.g., PET scans accurately and reliably measure blood-glucose uptake in the brain). The question defense counsel must be prepared to answer is case-fit and whether the visual image of blood-glucose uptake is sufficiently related to the mental health issue.

Technological variance: fMRI and PET scans currently provide the best functional images available. If your client suffers from schizophrenia or has been poisoned with a pesticide, functional imaging is more likely to be of assistance than structural imaging.

If your client suffered traumatic brain injury or metal poisoning, structural imaging with MRI or CT may provide better evidence.

Different laboratories use different technology, meaning that all PET scans or all MRI scans are not created equal. You will have to investigate the quality of the technology (based primarily on type of equipment, age of equipment, and experience of technicians) before choosing a laboratory to perform the imaging.

You must participate in providing direction to the imaging team about what evidence you are seeking. Different types of impairment may require different images and you cannot rely on sending someone for a scan to accomplish your needs. You will have to figure out what images you need and make sure the lab you have chosen can and does obtain them.

Not all readers of brain images (neuroradiologists or neuropsychiatrists) are equally trained. Do not allow a written report from a technician you have not retained and spoken with about your case.

Brain imaging can be useful in confirming evidence of mental dysfunction or impairment that is factually developed through other investigation. Unfortunately, there is no simple answer to the question of which type of brain imaging will show brain damage compared to other imaging techniques.

If you do undertake brain imaging, you are almost guaranteed rebuttal by a prosecution expert. You must prepare for this and plan with your expert to be available for rebuttal. You must also follow-up testing by uncovering additional information that bolsters and corroborates the findings,

including: lay witnesses to specific head injuries or trauma; lay witnesses to specific behaviors and onset/course of illness; additional expert witnesses (if etiology is fetal alcohol, may need FAS expert); and prior examining professionals who can testify as fact witnesses to what they observed during evaluations which preceded criminal prosecution.

10. Dealing with Evidence of Anti-Social Personality Disorder

Antisocial personality disorder (ASPD) is a very commonly diagnosed condition for people who have ever been in contact with the criminal justice system. It was once considered a “death” diagnosis because once a prosecutor argued its presence, a death verdict was the result. As with other aggravating evidence, however, ASPD can be dealt with and defeated based on thorough investigation and litigation.

One of the arguments some people have attempted to make in relationship to anti-social personality disorder is that since it may be related to particular ways in which the brain operates, it should be considered mitigating. That is, since there is some evidence that anti-social personality disorder has a genetic component and the brains of anti-social personality disordered people may appear different on certain types of brain imaging, this should be considered to be evidence in mitigation. It is not.

In some ways, every experience could be considered brain related, but it must make sense and matter for it to be mitigation. For instance, if you are walking down the street and stub your toe, it could be argued that this is a brain injury. Certainly the pain you felt was located in the response within your brain, and in fact, if the nerves between your toe and brain had been severed prior to stubbing your toe, you would not have felt that pain. No one could reasonably think that this was a good way of explaining stubbing your toe. Similarly, a pervasive pattern of bad acts probably does relate to the functioning of the brain, but why should a juror consider that sympathetic? If in fact there is evidence that brain dysfunction underlies ASPD, your client

is better off if you investigate and present that underlying brain dysfunction rather than converting that evidence into a personality disorder.

The purpose of mitigation is to argue for the client’s life. ASPD undermines this quite simply by dehumanizing the client and distancing the jury from the client, creating a perception that the client is defective and less than human. As the Ninth Circuit Court of Appeals held when asked to consider that ASPD has a genetic basis and is not the fault of the individual: “It is highly doubtful that the sentencing court would have been moved by information that Landrigan was a remorseless, violent killer because he was genetically programmed to be violent, as shown by the fact that he comes from a family of violent people, who are killers also.... although Landrigan’s new evidence can be called mitigating in some slight sense, it would also have shown the court that it could anticipate that he would continue to be violent.”⁵¹

ASPD also undermines any effort to individualize the client by placing that person into a criminal group: nearly 80% of prisoners are estimated to meet the criteria for ASPD. Further, the definition of ASPD states: “Only when antisocial personality traits are inflexible, maladaptive, and persistent and cause significant functional impairment or subjective distress do they constitute Antisocial Personality Disorder.”⁵² It is the inflexible, maladaptive and persistent trait aspects of the definition, combined with enduring, pervasive and inflexible pattern of

⁵¹ Landrigan v. Stewart, 272 F.3d 1221 (9th Cir 2001)

⁵² DSM-IV-TR at pp.705-6)

behavior that defines all the personality disorders that makes personality disorders in general, and ASPD in particular, useless as mitigation and often affirmatively harmful to your client.

ASPD offers no way in which to tell a compelling story as to how the client's life unfolded and how he is more than the sum of the actions that resulted in the capital murder charge. Instead, the story that ASPD tells is of an inevitable pathway towards capital murder: bad behavior in childhood leading through various types of increasingly aggressive criminality to capital murder. It is not possible to tell the ASPD story in a way which is mitigating because the DSM criteria establish the story as one in which crime, aggression and a disregard for others combine to create a persistent and pervasive pattern of failing to conform to social norms and the law. It is further a story in which bad acts are chosen repeatedly, where those acts result from a volitional and cognitively astute process of free will. In this context, even the story of how a person comes to be this way (violence in the home, genetic predisposition) becomes meaningless when articulated as volition and choice.

Diagnostic Criteria: Even a casual review of the criteria provides a sense of why this diagnosis is helpful to prosecutors even if all they can do is ask your expert about each of the criteria:

Diagnostic Criteria for Antisocial Personality Disorder (DSM-IV-TR)

A. There is a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years, as indicated by 3 (or more) of the following:

- (1) failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest
- (2) deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure
- (3) impulsivity or failure to plan ahead
- (4) irritability and aggressiveness, as indicated by repeated physical fights or assaults
- (5) reckless disregard for safety of self or others
- (6) consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations
- (7) lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated or stolen from another

B. The individual is at least age 18 years

C. There is evidence of Conduct Disorder with onset before age 15

D. The occurrence of antisocial behavior is not exclusively during the course of Schizophrenia or a Manic Episode

What's wrong with ASPD: Antisocial personality disorder is part of Cluster B of the Axis II personality disorders (discussed more generally above). Cluster B conditions are often referred to as "dramatic" disorders (because they reflect acting out behaviors). Cluster B comprises antisocial, borderline, histrionic and narcissistic. Under DSM-II, antisocial personality disorder was thought to have a prevalence of about 30% in prison populations. The move to place personality disorders onto their own Axis and the shift from trait-based to behavior-based criteria drastically increased the prevalence of the

condition to 75-80% in the prison population, leading some to suggest that ASPD was simply a codification of criminal behavior, no longer predicated on a personality structure.⁵³

Other than a collection of bad acts, serious question remains as to whether there is any diagnostic reliability or validity to ASPD. The scientific literature points out numerous, on-going diagnostic problems with ASPD, starting by recognizing that hundreds of thousands of combinations of subcriteria behaviors can lead to the same diagnosis without any relationship between them. This means, all subcriteria are weighted equally, such that armed robbery and rape are equal diagnostically to truancy). They also note that there is very poor diagnostic reliability between experts when they assess ASPD.

Some researchers have also commented that mental health clinicians have no particular expertise in assessing deviation from social norms. This is an often ignored criticism, that the training and expertise of psychologists and psychiatrists renders them no more able than a lay person to opine on “deviance” and to assess the pattern of deviant social behavior. This is critically important in the forensic diagnosis of antisocial personality disorder because under the guise of a psychological classification of disease, experts often diagnose social deviance as a psychological disorder based solely on personal opinion or previous adjudication rather than independent expertise and training. Personal dislike of the patient or moral judgments about the patient’s behavior may result in an ASPD diagnosis, but: “If so, the diagnosis may present a

⁵³ Wulach, J.S. (1983) Diagnosing the DSM-III Antisocial Personality Disorder Professional Psychology: Research and Practice 14(3) 330-40.

pseudo-scientific facade for value judgments.”⁵⁴

The more recent versions of DSM (i.e., DSM-III-R, DSM-IV, and DSM-IV-TR) have stayed with observable behavior criteria with minor modifications (such as dropping certain criteria to better differentiate between ASPD and other Cluster B personality disorders). DSM-IV added a handful of conditions which should be ruled out prior to a diagnosis of ASPD. These include: that the behavior is not better accounted for by another mental disorder – meaning that an Axis I diagnosis “trumps” a personality disorder for diagnostic significance; that the behaviors are not the direct physiological effect of substance abuse – including drug seeking behavior or addiction maintenance behavior, intoxication or withdrawal, and criminal behavior directly caused by the substance abuse; that the social and cultural context of the person is not the cause of the behavior – this was meant to address the excessive diagnosis of personality disorders in the poor and appears to include cultural beliefs as well as poverty; and, that when personality change occurs as the result of external stressors it should not be considered as a Cluster B disorder – as in, when trauma caused a change in personality the diagnosis of PTSD should be considered.

Because the DSM-IV-TR has stayed with the observable behavior criteria, the problems with the model have not improved. First, the empirical research provides support to the idea that personality disorders are more

⁵⁴ Weinstock, R. and Nair, M. (1984) Antisocial personality - diagnosis or moral judgment? Journal of Forensic Sciences 29(2) 557-65; Rogers, R., Dion, K.L. and Lynett, E. (1992) Diagnostic validity of antisocial personality disorder Law and Human Behavior 16(6) 677-89.

appropriately assessed on a continuum, where people might be viewed as incrementally further from “normal” in some areas and not others.⁵⁵ DSM-IV-TR discusses this idea, but the criteria for diagnosis remain static and dichotomous (subject does or does not meet criteria). This approach to diagnosis is not supported by any empirical findings but which is clinically pragmatic.

Do professionals diagnose the same behaviors the same way? A total lack of discriminant validity currently undermines the Cluster B disorders. Discriminant validity means that the disorder can be accurately and consistently identified and distinguished from other disorders. There is tremendous diagnostic overlap which some people have inaccurately referred to as co-morbidity but which is in fact a failure to systematically describe distinct and identifiable conditions.

Many people qualify for more than one personality disorder based on the same or similar behaviors or based on slight variation in the interpretation of the behavior by a clinician. DSM-IV-TR cautions: “Other Personality Disorders may be confused with Antisocial Personality Disorder because they have certain features in common.”⁵⁶ This overlap is recognized as a failure of the science: “Substantial evidence of the lack of exclusiveness is provided by studies of diagnostic overlap mentioned previously. Overlap is often misleadingly referred to as comorbidity. However, comorbidity refers to the co-occurrence of distinct diagnoses and there is no evidence that personality disorder diagnoses are distinct in this sense. When

⁵⁵ Livesley, W.J. ed. (2001) Handbook of Personality Disorders New York: The Guilford Press.

⁵⁶ DSM-IV-TR at p.705.

applied to personality disorder, the term ‘comorbidity’ simply obscures a fundamental flaw in the system.”⁵⁷

In a review of the existing literature on “comorbidity” among Axis II disorders under DSM-III-R, researchers found that only 4 of 196 cases of Borderline Personality Disorder (BPD) did not have a comorbid personality disorder – meaning that nearly every person in these studies diagnosed with BPD was also diagnosed with another personality disorder. Eighty two (82) percent of antisocial diagnoses also were diagnosed with another personality disorder in the studies reviewed.⁵⁸

Does ASPD relate to a mental illness? Unlike schizotypal personality disorder which clearly relates to the Axis I mental disorder of schizophrenia, ASPD has no corresponding Axis I illness. This suggests that personality disorders are not in fact mental illnesses as currently defined. In addition, the fact that DSM-IV-TR includes a “trumping” of Axis II by Axis I disorders in diagnosis implies a recognition that Axis II -- especially Cluster B -- identifies character flaws as opposed to mental illness where mental illness is considered a condition that a person has rather than a core definitional characteristic of the organism.

Dr. Stephen Hart, a leading forensic psychologist, has recently written: “As do the mental health professions, however, the law

⁵⁷ Livesley, W.J. ed. (2001) Handbook of Personality Disorders New York: The Guilford Press at p.18.

⁵⁸ Widiger, T.A. et al (1991) Comorbidity among Axis II disorders, in Personality Disorders: New Perspectives on Diagnostic Validity ed. by Oldham, J.M. at p168.

often distinguishes between mental illness and personality disorder. Mental illness can be defined as acute and severe disturbances in psychological functioning - that is, disorders falling on Axis I of the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). In contrast, personality disorders can be defined as chronic disturbances of character or social relations, disorders falling on Axis II of DSM-IV..... If the personality disorder is not severe enough to be a mitigating factor in forensic decision making it may be considered, somewhat ironically, an aggravating factor, something that can be used to argue for harsher punishment or imposition of long-term social controls.”⁵⁹

Hart’s argument is basically that if volition and cognition are not impaired by ASPD, which almost every forensic consultant concludes, and since it is unlikely that any personality disorder under the current definitions will impair volition and cognition, it is not mitigating and may be viewed as aggravating. “[W]hereas mental illness is generally considered a mitigating factor in forensic decision making, personality disorder generally is considered to be an aggravating factor.”⁶⁰ Hart comments on capital sentencing and ASPD saying that it denotes a lack of good character (lack of mitigation) and elevated risk for future dangerousness (aggravation).

⁵⁹ Livesley, W.J. ed. (2001) Handbook of Personality Disorders New York: The Guilford Press at p.556.

⁶⁰ Livesley, W.J. ed. (2001) Handbook of Personality Disorders New York: The Guilford Press at p.560.

Is ASPD a unique condition? The construct validity of the current system has yet to be established (construct validity meaning that the definition of the condition is unique, identifiable and recognized by multiple clinicians as the same condition). Although most clinicians agree that such conditions exist in their patients, the evidence that what the clinician is observing constitutes a taxon (a unique and identifiable condition) is currently non-existent. The current catalogue of features observed clinically or self-reported by the patient as the basis for diagnosis is unsupported by empirical evidence.

Can ASPD be ruled out? The multiple diagnoses model that the multi-axial system implies has failed to get clinicians to adequately rule out, assess or differentiate clinical disorders (Axis I conditions and mental retardation) from personality disorders. Although DSM-IV adopted a small number of conditions which should be considered when diagnosing ASPD, there is no requirement for differential diagnosis. For instance, if a mental disorder is present or is the cause of the behaviors, do not diagnose the antisocial behaviors – differential diagnosis is a term applied to the process of weighing and assessing conditions and symptoms before arriving at a single diagnosis.

The context of behavior: The current criteria completely ignore the context of behaviors. Thus, although lying to avoid physical or sexual abuse is specifically acceptable, other behaviors which derive specifically from external stimuli (as opposed to internal personality structure) are not considered according to the criteria. The empirical research is clear that for many people diagnosed with personality disorders, at the very least, family and environment (including, density of poverty, access to

services, peer group behaviors, family behavior and structure, and neighborhood factors) are causative rather than internal personality structure. Some researchers have looked at changes wrought in personality by these external events which at least acknowledges a change in personality structure rather than the “bad seed” implied by others.

This remains a contested area of research. For example, child abuse has been alleged to cause bad behavior in adulthood. In fact, the research indicates that, untreated and unremediated, being subjected to child abuse may place a person at higher risk for coming into contact with law enforcement and other behaviors which jeopardize health. One key distinction here is between “causation” and “increased risk.” A second key issue is whether being abused as a child changes the personality structure of the child – renders the child unable to emotionally relate to others normally – or whether child abuse is like a medical condition which can be identified, altered (placing the child in a safe environment) and remediated or treated. From a defense perspective, child abuse may present both a way in which to humanize the client as an individual who has experienced specific events as well as explain certain behaviors that have followed from the abuse (post-traumatic stress disorder). From a prosecution perspective, child abuse has been argued to cause irreparable changes to the child who, now being an adult, is antisocial and will engage in criminal activity.

The key issue here is that the context of the behavior matters, both in origin (is it innate to the person or the result of stressors and external stimuli?) and in outcome (does the child run away from the abuse or is the child running away to thwart parental

supervision). “Antisocial behavior may arise from a multiplicity of causes of which personality pathology is only one. Conflating traits and behavior in the measurement of psychopathy means that it is impossible to infer that personality pathology drives antisocial behavior”⁶¹ Cicchetti and Rogosch described this as equifinality (multiple pathways to a similar outcome) and multifinality (similar pathways to different outcomes).⁶² Each concept demands careful attention to the ecological context of the behavior.

Recently reported longitudinal research (which confirms earlier research) compares a birth cohort over time, dividing it into two groups which exhibit behavior problems: an early onset and persistent over time group (LCP) and an adolescent onset and short-lived group (AL). The theory is basically that the LCP group starts antisocial behaviors early and continues across life-span. The AL group engages in similar antisocial behaviors, but they desist relatively quickly (meaning, the behaviors observed in adolescence for both the LCP and AL groups is delinquent). Moffitt and Caspi describe this cohort and find that the LCP group is more likely to come from dysfunctional families (younger mothers, harsh or inconsistent discipline, family conflict, mentally ill mothers, more changes in parental caregivers, poverty, as well as neurocognitive deficits, hyperactivity and fighting and peer rejection as assessed by

⁶¹ Cooke and Michie (2001) Refining the construct of psychopathy: Towards a hierarchical model Psychological Assessment 13(2) 171-88.

⁶² Cicchetti, D. and Rogosch, F.A. (1996) Equifinality and multifinality in developmental psychopathology Development and Psychopathology 8:597-600.

parents and teachers).⁶³ One way to understand this research is that the LCP children are in a social and family environment which places them at significantly heightened risk for behavior problems and simultaneously undermines the resiliency factors. This supports the notion that the context of behavior is crucial both to the development of the behaviors of concern and that many of those behaviors reflect social adjustment problems rather than an innate personality deficit.

Do the historical behaviors that make up ASPD tell us anything about the present or future? Research provides limited evidence that the current personality disorder categories predict treatment success or behavior. Most of the research indicates a failure to develop successful treatment for all of the Cluster B conditions. However, the treatment outcome research has been thoroughly irrelevant to the death penalty context, in part because there is evidence that most people diagnosed with ASPD adjust well to the structured environment of prison. Thus, the treatment outcome research has primarily looked at how well the “treated” person does discharged from prison or the hospital (in the community, usually without resources), a situation which does not apply to penalty phase determinations.

Does ASPD meet Daubert? “A consensus seems to be emerging among clinicians and researchers that there are fundamental problems with the DSM classification of personality disorders that require radical

⁶³ Moffitt, T.E. and Caspi, A. (2001) Childhood predictors differentiate life-course persistent and adolescent-limited antisocial pathways among males and females Development and Psychopathology 13:355-75.

change.”⁶⁴ This includes a consensus concerning the need to radically change the atheoretical approach with respect to etiology, diagnoses, treatment of personality disorders. How exactly this will play out in DSM-V is unclear.

Conduct Disorder: One of the key diagnostic requirements of ASPD is that conduct disorder existed prior to age 15. Many evaluators ignore this requirement and it is an important technique for defeating ASPD.

Diagnostic Criteria for Conduct Disorder (DSM-IV-TR)

A. A repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated, as manifested by the presence of three (or more) of the following criteria in the past 12 months, with at least one criterion present in the past 6 months:

Aggression to people and animals

- (1) often bullies, threatens or intimidates others
- (2) often initiates physical fights
- (3) has used a weapon that can cause serious physical harm to others
(e.g., a bat, brick, broken bottle, knife, gun)
- (4) has been physically cruel to people
- (5) has been physically cruel to animals
- (6) has stolen while confronting a victim
(e.g., mugging, purse snatching, extortion, armed robbery)

⁶⁴ Livesley, W.J. ed. (2001) Handbook of Personality Disorders New York: The Guilford Press at p.9.

(7) has forced someone into sexual activity

Destruction of Property

(8) has deliberately engaged in fire setting with intention of causing serious damage

(9) has deliberately destroyed others' property (other than by fire setting)

Deceitfulness or theft

(10) has broken into someone else's house, building or car

(11) often lies to obtain goods or favors or avoid obligations (i.e., "cons" others)

(12) has stolen items of non-trivial value without confronting victim

(e.g., shoplifting, but without breaking and entering; forgery)

Serious Violations of Rules

(13) often stays out at night despite parental prohibitions, beginning before age 13

(14) has run away from home overnight at least twice while living with parent or parental surrogate (or once for a lengthy period)

(15) is often truant from school beginning before age 13

B. The disturbance in behavior causes clinically significant impairment in social, academic or occupational functioning.

C. If the individual is age 18 years or older, criteria are not met for Antisocial Personality Disorder.

ASPD is one of the very few disorders in DSM that requires childhood onset and which cannot be diagnosed without specific historical markers of the condition. This has meant that the CD validity and reliability,

especially as retrospectively assessed, is a critical area of concern.

"Many antisocial behaviors emerge in some form over the course of normal development."⁶⁵ Thus, the key differentiation between behaviors and disorder are frequency and intensity of the behaviors.

As diagnosed, conduct disorder is "likely to be the end product of complex multifactorial processes operating within and outside a given individual, but the relevant causal processes are likely also to differ across individuals."⁶⁶ For some, antisocial behaviors may be internally driven (a mental disorder), but for others those behaviors derive from extrinsic, environmental factors. There is no basis for the assertion that a mental disorder underlies the behavior defined as conduct disorder.⁶⁷ Rather, many children thought to have conduct disorder could more accurately be diagnosed with an Axis I mental illness.⁶⁸

Longitudinal research indicates that conduct disorder in childhood relates to numerous problems in adulthood: psychiatric

⁶⁵ Kazdin, A.(1995) Conduct Disorders in Childhood and Adolescence 2nd ed. Thousand Oaks, CA: at p.8.

⁶⁶ Richters, J. and Cicchetti, D. (1993) Mark Twain meets DSM-III-R: Conduct disorder, development, and the concept of harmful dysfunction Development and Psychopathology 5:5-29 at p.15.

⁶⁷ Richters, J. and Cicchetti, D. (1993) Mark Twain meets DSM-III-R: Conduct disorder, development, and the concept of harmful dysfunction Development and Psychopathology 5:5-29 at p.21.

⁶⁸ Lewis, D.O. et al (1984) Conduct disorder and its synonyms: Diagnosis of dubious validity and usefulness American Journal of Psychiatry 141(4) 514-9.

symptoms, occupational difficulties, physical illness, criminal contacts (often resulting from substance abuse), social isolation and lowered educational attainment.⁶⁹ This suggests that some of the symptoms associated with conduct disorder may be markers of above noted conditions, each of which could be treated or remediated in childhood or adulthood (as opposed to the CD diagnosis which does not respond well to treatment or remediation).

Triad assessments in childhood (firesetting, bedwetting and animal cruelty): Although DSM requires a diagnostic finding of conduct disorder in order to diagnose ASPD, some clinicians continue to rely on the “triad” of childhood problems as the basis for predicting future behavior problems. Historically, psychiatrists suggested that enuresis (bedwetting) was an aggressive act of destruction and hate.⁷⁰ (see, Hellman and Blackman 1966; MacDonald 1963). Firesetting was observed more often in delinquent boys than non-delinquent boys and has been associated with enuresis. Animal cruelty was also associated anecdotally with aggression.

By the early 1970s, empirical research was already demonstrating that the link between the triad behaviors and violence was substantially weak. One such study concluded that behaviors other than the triad were more

predictive of adulthood violence, and that none of the behaviors of childhood adequately predicting future violence: “As already noted, the early-warning signs of violence defined in this study are behavior patterns that appear in every child at some time. How, then, is it possible to be certain that a child exhibiting these behaviors will later commit violence? It is not.”⁷¹

In a 1984 study, the triad was found to be a maladaptive response to abuse and family dysfunction rather than a predictor of antisocial behavior. Twice as many physically abused children exhibited triad behaviors compared to non-abused children and four times as many sexually abused as non-sexually abused children exhibited triad behaviors. More neglected children also showed triad behaviors. Drug abusing mothers and criminal fathers also had children who were much more likely to exhibit triad behaviors. The researchers concluded that triad behaviors “represent a nonspecific maladaptive response to a malignant home environment” and conclude that: “This study found no evidence to support the predictive value of the triad for adult criminal outcome.”⁷²

Nevertheless, some experts continue to rely on the presence of triad behaviors as the basis for diagnosis.

Keys to challenging ASPD: As the foregoing suggests, there are a number of

⁶⁹ Kazdin, A.(1995) Conduct Disorders in Childhood and Adolescence 2nd ed. Thousand Oaks, CA: at p.69.

⁷⁰ Hellman D.S. and Blackman N. (1966) Enuresis, firesetting and cruelty to animals: A triad predictive of adult crime American Journal of Psychiatry 122(2) 1431-5; MacDonald, J.M. (1963) The threat to kill American Journal of Psychiatry 120(2) 125-30.

⁷¹ Justice, B., Justice, R. and Kraft, I.A. (1974) Early-warning signs of violence: Is a triad enough? American Journal of Psychiatry 131(4) 457-9 at p.458.

⁷² Prentky, R.A. and Carter, D.L. (1984) The predictive value of the triad for sex offenders Behavioral Sciences and the Law 2(3) 341-54.

ways to challenge ASPD successfully. One strategy is to challenge the conduct disorder finding that must be made. Many times, prosecution experts do not bother to detail the criteria upon which your client has been determined to be ASPD. In situations where the social and family history records support your contention that there was no CD – where the pervasive pattern of bad acts does not exist or is better explained by an Axis I disorder or the behaviors only occurred in response to cultural or external pressures or when onset of the behaviors follows injury or mental illness – you will have to challenge the expert directly, preferably before the expert testifies through in limine motions that challenge the case fit and diagnostic reliability of ASPD in your case.

Remember that the behaviors that make up the CD criteria are extremely heterogenous, describing many behavior patterns which are not alike. The term for this, equifinality, means that similar outcomes (the pattern of behaviors) arise from many different pathways or causes.⁷³ Attention must be given to the pathway and to whether some pathways provide evidence for an alternative diagnostic picture. Some pathways may better explain the behavior than a persistent pattern of bad acts.

Children with organic brain dysfunction are often mis-diagnosed with CD. Brain damage, whether genetic or from injury or insult may better explain a child's behavior, behaviors that could result in a CD diagnosis unless explained to be the result of brain damage. Similarly, the behaviors associated

with many psychiatric illnesses (e.g., psychotic disorders in childhood, attention deficits, traumatic stress, language and learning disabilities, exposure to violence) each may resemble conduct disorder and may lead to mis-diagnosis. Such differential diagnosis must be encouraged and can only be based on thorough family and social history evidence.

Substance abuse as well should be considered prior to diagnosing CD or ASPD. Since the conditions can (and often will be said to) co-exist, you must carefully document the onset and course of the behaviors. You want to be able to argue that the bad acts resulted from substance abuse rather than substance abuse being simply another marker of antisocial behaviors. The issue here is all about which came first. When the bad acts begin in late adolescence, it is more likely, according to the empirical literature, that substance abuse is causing the behavior problems. Early onset of behavior problems, starting at ages 2 to 3 years old, better supports a picture of antisocial conduct, although the vast majority of early onset children desist by mid-adolescence. The small percent that persist appear to have a greater degree of negative external factors influencing them (family, neighborhood, and institutional) and a higher rate of neurological deficits.⁷⁴

Therefore, you must examine the alleged behaviors for an explanation other than CD which both defeats the diagnosis of CD, and forms the baseline of your mitigation story. You will undertake the same process for the adult criteria of ASPD, seeking to better

⁷³ Cicchetti, D. and Rogosch, F.A. (1996) Equifinality and multifinality in developmental psychopathology Development and Psychopathology 8:597-600.

⁷⁴ Hinshaw, S.P. and Lee, S.S. (2002) Conduct and Oppositional Defiant Disorders in Mash, E.J. and Barkley, R.A. eds. Child Psychopathology 2nd ed New York: The Guilford Press.

explain the truth behind the behaviors.

Rule-out provisions: Another key to defeating ASPD is the rule-out provision (criterion D): “The occurrence of antisocial behavior is not exclusively during the course of Schizophrenia or a Manic Episode.” If your client suffers one of these illnesses, you must spend sufficient time to assess whether the bad behaviors occurred during the course of the illness, in which case, ASPD cannot be diagnosed. Similarly, if the behaviors are the specific result of trauma or PTSD, DSM-IV-TR requires that PTSD be considered and a strong case can be made that those behaviors are better interpreted as traumatic stress related rather than antisocial. You must also seek to do this with all other clinical disorders as well.

Some experts insist on diagnosing ASPD along with other conditions (essentially arguing that your client is mentally ill, but he is also a jerk). You must talk to your experts about how this will be used in litigation before they write a report stating an opinion. Sometimes, you will have to walk your own expert back through the social and family history to discuss why ASPD is inappropriate. In some cases, you will not be able to have that expert testify.

Sometimes, the records that you gather or that are provided in discovery, will contain prior diagnoses of ASPD. After preparing your family and social history case, you must attempt to meet with and discuss the prior diagnosis with the assessing doctors. Bring them the evidence that shows a better explanation and guide them through all the new evidence that demonstrates a better explanation.

To conclude, ASPD is not helpful to your

case. You may have to deal with it from prior evaluations, from prosecution experts or even from your own expert. You can deal with it by finding and documenting better explanations for the behaviors that make up ASPD.

11. Dealing with evidence of malingering

Allegations of ‘malingering’ are often how the prosecution seeks to counter mental health evidence. The concept of malingering is simply: “...the intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives, such as avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution or obtaining drugs.”⁷⁵ Boiled down, it is a medicalized description of your client as a liar and manipulator. DSM-IV-TR does set out some conditions under which malingering is more likely to occur, although it does not have specific criteria as do other conditions; the best evidence available suggests that the DSM-IV-TR indices lead to wrong malingering determinations four out of five times in a forensic setting.⁷⁶ Additionally, one of the indices is antisocial personality disorder and there is no support in the scientific literature that people with ASPD malingering more or less than anyone else.

‘Malingering’ is something you will almost certainly deal with, and it is certainly something you must prepare for, if you are presenting mental health evidence in any phase of your case. An expert may make a determination that your client is malingering based on clinical interview, psychological assessment instruments, or a combination. Clinical interview based determinations of

malingering have not been demonstrated to be particularly reliable, at least in part because they are subject to a host of bias problems, including: the clinical background and experience of the interviewer; information (or lack thereof) provided to the interviewer prior to assessment (such as social history information that contextualizes behaviors); political, social or cultural views of the interviewer (including beliefs about poor people and people of color, as well as “retainer bias” – the increased likelihood of findings supporting the side who pays the expert); and beliefs about malingering itself. It is important to obtain the notes and scoring information that underlie an expert’s determination of malingering so that you can assess precisely how the determination was made, what questions it is based on and potential bias in the interviewer.

Another key to countering malingering evidence is to be prepared to present onset and course of illness evidence. Malingering charges amount to an allegation that your client is making himself appear ill because of the criminal charges against him/her. Your first counter to this is to present extensive, independent evidence of the illness prior to the initiation of criminal proceedings. The social and family history evidence which you have gathered to build your mental health presentation is the basis for this counter-argument. Corroboration of behavioral and physiological symptoms over the course of your client’s illness, presented by means of records and percipient witnesses can be used to make apparent that this specific illness is neither feigned nor newly alleged.

Further, you must evaluate the assessment of malingering and the concept of malingering as it is being applied to your client. Below is an introduction to basic concepts related to

⁷⁵ DSM-IV-TR at p.739.

⁷⁶ Rogers, R. (1997) Clinical assessment of malingering and deception, 2nd ed. New York: The Guilford Press; Rogers, R. (1990) Development of a new model classificatory model of malingering Bulletin of the American Academy of Psychiatry and the Law 18(3) 323-333.

malingering. No one really knows how prevalent malingering is, which leads some psychologists to suggest its presence in nearly every case in which they consult. Some forensic evaluators have been taught to diagnose malingering whenever a person in a forensic setting endorses very rare or non-standard symptoms. If this is the case, you have to work with your client to understand why those items were endorsed (e.g., your client actually has the rare symptoms, did not understand the question, endorsed it because it was similar to another symptom and the client was told to say yes if the symptom was more likely than not). You should also discuss with the client when the symptoms in question were first experienced, if they are specific to some contextual fact (e.g., being in custody) and if the symptom waxes and wanes or is constant. Following this, you again need to carefully review medical and psychological records (and likely re-interview witnesses) to find instances where the client endorsed these symptoms prior to arrest or to find evidence that supports the onset described by your client.

Next, you should consider whether the symptoms that led to a malingering assessment resulted from something other than an intent to deceive. Rogers⁷⁷ has suggested a number of response styles and conditions which lead clients in forensic settings to appear to be malingering when in fact they are not or a better explanation is available for his/her answers:

A) defensiveness about mental illness or trauma: refers to conscious denial or

minimizing of symptoms in an effort to make oneself look less ill. Sometimes this will result in a client endorsing peculiar or contradictory symptoms. For some clients, endorsing or rejecting odd symptoms may also result from a social desirability, meaning an attempt to please the examiner or to appear less odd in the examiner's eyes;

B) irrelevant response: resulting from the client not engaging in the interview process or not understanding questions or words being used. This may include random answering of questions on objective tests like the MMPI or a lack of comprehension sufficient to interpret the questions;

C) honest responses admitting peculiar or rare symptoms: as noted above, many of our clients actually have very rare symptoms or very rare combinations of symptoms, some are in very unusual settings, and some experience the world in very unusual ways – this will lead them to honestly endorse items that are rare (endorsing rare items being one of the primary measures of malingering).

Finally, there may be differences in the severity of endorsing or feigning odd symptoms. A client could easily provide feigned responses to one set of questions and answer honestly throughout the rest of the interview. Similarly, some answers may simply be unreliable rather than intentionally false, and other answers may be distorted or out of proportion but not provided with an intent to deceive. The severity and scope of malingering should also be assessed by you in cooperation with your experts.

Some psychiatric illnesses are well-known to have as a component “confabulation.” Confabulation is not malingering although it may be confused for it. Confabulation is a

⁷⁷ Rogers, R. (1997) Clinical assessment of malingering and deception, 2nd ed. New York: The Guilford Press.

normal process in which a person fills in details, often coming to fully believe those details, which they do not actually recall, did not actually experience or do not actually know. This is a normal process where, for instance, people do recall some specific details of an event but not enough details to make the re-telling of the story coherent. That person may fill in details to allow the story to make sense. This is a type of confabulation. Similarly, people who were psychotic during an event may later recognize that their experience of that event no longer makes sense and he/she will alter the details to cover over the psychotic features that no longer seem correct. The process of confabulation does not contain the intent to deceive necessary for a finding of malingering.

Sometimes, your client actually has malingered some symptoms. In these cases, your affirmative presentation must rely on sources other than the client for descriptions of symptom patterns and behavior. If your client has truly malingered symptoms, you cannot allow your expert to rely on anything gained from clinical interview. This puts significantly more pressure on your record gathering and lay witnesses, but you have little choice and must not open your expert to cross-examination that will create an impression that the expert relied on false and intentionally misleading information provided by your client.

Psychological tests used to assess malingering: As mentioned above, some assessments of malingering are based on clinical interviews in which a clinician disbelieves or discounts your client's self-reporting. Other assessments will be made based on how your client scores on structured interviewing scales, stand alone psychometric tests or psychometric tests which are part of

broader assessment batteries.

By far, the most common technique used is to give the client personality instruments like the MMPI-2 (see discussion above). Do not give your client the MMPI-2 to try to rule out malingering. The MMPI-2 is not a rule out instrument and you will almost certainly create a host of additional problems in your case by giving the MMPI-2.

Although you should not administer any of these personality instruments to your client, you are likely to have to deal with them in relation to malingering either because the prosecution has them administered or because prior evaluations in the client's records include MMPI scores. The MMPI-2 has been used countless times as an assessment of malingering. At its best, the MMPI-2 should only be used to generate hypotheses about the individual, not to diagnose. Thus, despite the prevalence of its use in determinations of malingering, it is not a reliable tool with which to make such claims.

Originally, the method of detecting malingering was known as the "F minus K Index" or the Dissimulation Index. This method relied on subtracting a subject's score on the K scale from his or her score on the F scale leaving the clinician with a number that, if high enough, was said to suggest the presence of malingering. The cut-off number selected was usually greater than 9 although there was a great deal of debate about the appropriate cut-off, which illustrated the ineffectiveness of this method. It is well established and repeatedly published in the literature (at least since 1989) that the use of the F minus K Index is not reliable for detecting malingering.

Currently, some clinicians rely on an

elevation on the F scale. The F scale captures the items that have been answered in unusual or atypical ways. An elevated F scale is more likely to indicate confusion, acute distress, or exposure to trauma. Additionally, as noted above, paying insufficient attention over the course of the test, the custodial context of test taking, your client's mood when taking the test, his/her having true experiences out of the norm, or a host of other problems (including a panoply of mental illnesses – mentally ill patients endorse F-scale items at 3 times the rate of non-ill people) may lead a client to honestly endorse items in unusual or atypical directions. As a result, although perhaps of some use in clinical settings, the F scale is unreliable in criminal proceedings.

Other scales that have been used to assess malingering include the L, K and CS scales. The L scale is comprised of items that, when a significant number are endorsed in a particular direction, suggest that the person may be attempting to make him/herself look better (more virtuous, fewer faults). The K scale measures defensiveness and elevation on this scale may also indicate that the subject is attempting to represent themselves in an artificially favorable light. Finally, the “cannot-say” scale (denoted as a “?” or CS) refers to the number of items that a person either refuses to answer or answers both true and false. There are a number of reasons why someone may answer questions that will lead to elevations on the L and K scales (for instance, wanting to be liked, fear, embarrassment as well as trauma, mood during the testing, conditions of confinement among many others).

The CS scale captures questions not answered. It is generally accepted to interpret a test as valid with a cut off as great as 29 on this CS scale. A person with a great deal of

indecision might have a hard time answering questions, might not understand the questions, might be unclear as to how to answer true or false to a question, might have intended to go back to it, may have decided not to answer for some other reason. Similarly, all the reasons referred to in relation to F, L and K may apply, but here the client chooses not to provide information.

High scores on the F, 2, 4, 6 and 8 scales (as a pattern) are sometimes used to allege malingering as well. However, the research is quite clear that elevations in these scales occur commonly in abused people and in victims of trauma and posttraumatic stress disorder sufferers generally. The concurrent elevation on scales 6 and 8 is indicative of the hypervigilance and loss of trust characteristic of traumatized patients. To counter the prosecution's use of such a pattern, you must put forward a factual basis for a better interpretation of the scales.

Finally, there is a standard test taking bias in which all people tend towards answering “true” when forced to choose between true and false (and when not feeling strongly either way). The scales which seek to assess malingering are generally structured to raise malingering scores when the person answers “true.”⁷⁸ Therefore, some questions which are said to indicate malingering will simply be endorsed because of standard test taking bias and has nothing at all to do with malingering. You will have to take the prosecution's test scores to your client and go over each of the questions with him/her to figure out if this type of bias occurred.

⁷⁸ See, Rogers, R. (1997) Clinical assessment of malingering and deception, 2nd ed. New York: The Guilford Press.

Most of the research on the MMPI-2 and malingering has used subjects who were told to malingering and compared them to controls (people not told to malingering). Although perhaps necessary, this is not a reliable means of determining how well the MMPI-2 works at the assessment of malingering. No single cut-off score on any combination of scales has been established as reliable and valid for determining malingering on the MMPI-2. This means that you must find out what cutting score was used by the expert alleging your client was malingering and then look at the psychological research to see whether that cutoff is supported or not.

Most neuropsychologists will assess malingering as part of the standard test battery. Standard neuropsychological examinations should include tests that assess for malingering (e.g., Rey Word Recognition Test, Portland Digit Recognition Test, Dot counting, Symptom Validity Test). Each of these tests assess the degree of effort and theoretically, each can be completed without error by most unimpaired people (severely demented patients perform in the chance error range on symptom validity tests). Malingering can also be detected reliably by scoring patterns on some of the Halstead-

Reitan neuropsychological battery. Essentially, the expert looks for atypical patterns of answers on memory and concentration, or significant scoring differences on tests that measure the same abilities. Experts will use these tests as well as clinical judgment based on affect and behavior during the testing sessions to assist in reaching determinations about malingering. You should discuss this with the neuropsychologist you are working with on the case.

Finally, there are a number of increasingly popular, stand-alone instruments which are designed to assess malingering. These tests might be useful to countering prosecution allegations of malingering under very rare circumstances. None of these instruments “rule-out” malingering (at best, they offer a probability that the client is not malingering), although they may provide limited indication that at least on this test the client tried. Further, they tend to over-include people into the malingering category. The best of these is the Structured Interview of Reported Symptoms (SIRS), which is a 172 item structured interview, using eight primary scales to assess feigning.

SIRS Primary Scales: Composition and Description

Scale	Items	Alpha	Reliab	Description of Scale
RS	8	.85	.98	Symptoms infrequently endorsed by genuine patients
SC	10	.83	.97	Pairs of symptoms uncommon in genuine patients
IA	7	.89	.96	Fantastic and very atypical symptoms--unlikely to be true
BL	15	.92	.95	Disproportionate number of obvious symptoms
SU	17	.92	.96	Disproportionate number of everyday problems
SEL	32	na	1.00	Overall proportion of symptoms higher than found in genuine patients
SEV	32	na	1.00	Disproportionate number of symptoms with unbearable severity
RO	11	.77	.91	More discrepancies in observable behavior than genuine patients

Dr. Richard Rogers, who developed the SIRS, suggests that when standardized testing suggests malingering, the expert should go back and re-interview the client before making a determination. In effect, he argues that these tests should be used for hypothesis generation, not diagnosis. As with all these instruments, you must get the raw testing data and carefully review the cutting scores used. Rogers recommends that any scale in the definite feigning range or three or more scales in the probably feigning range or total score in the feigning range allows a determination of malingering.

It is essential to keep in mind that these stand alone tests tend to over-include people in the malingering category – they have low sensitivity and high specificity, meaning they tend to identify all malingerers but also many non-malingerers. This speaks against using these instruments affirmatively because even when your client is truthful, he/she is at risk of being grouped into the malingering category.

Finally, The Schedule of Affected Disorders and Schizophrenia (SADS) was developed by the National Institutes of Mental Health as a structured instrument to assess presence and severity of major mental illness. It was designed as a research tool, but because of its focus on symptoms of psychiatric illness and differential diagnosis, it may be useful in some cases because it is more likely to detect and properly note rare symptoms, combinations of symptoms, symptom severity and non-intentional or random symptom endorsement. The SADS is not currently a standard instrument in forensic practice, in part because it takes a long time to administer and because most forensic experts are not trained to administer and interpret it.

In conclusion, malingering and the assessment of malingering will become the focal point of your case if the prosecution can successfully change the focus from your story to their story. You can avoid this in a number of ways, all of which depend on: the breadth and sufficiency of your social and family history investigation, your cooperation with your expert and preparation for the evaluations your team does, and your preparation for the prosecutions rebuttal evidence. Do not have your client tested with personality instruments like the MMPI-2 to disprove malingering – it cannot do that and you risk creating bad facts unnecessarily. Do talk with your neuropsychologist about how malingering will be assessed during the neuropsychological testing that you will have performed.

SOME VERY SELECTED, GENERAL RESOURCES

1. Web-based resources

Free, searchable database of medical research:

<http://www.ncbi.nlm.nih.gov/entrez/query.fcgi>

Useful summaries of medical information and news

<http://www.medscape.com/>

Brain Image pages

<http://www.med.harvard.edu/AANLIB/home.html>

<http://www.brainconnection.com/>

Mental health advocacy organizations and professional organizations

<http://www.unl.edu/ap-ls/> (American Psychology-Law Society)

<http://www.emory.edu/AAPL/> (American Academy of Psychiatry and the Law)

<http://www.nami.org/> (National Alliance for the Mentally Ill)

<http://www.apa.org/> (American Psychological Association)

<http://www.psych.org/> (American Psychiatric Association)

<http://www.aamr.org/> (American Association on Mental Retardation)

<http://www.thearc.org/> (Mental Retardation Advocacy Organization)

<http://nanonline.org/index.shtml> (National Academy of Neuropsychology)

Brain Injury

<http://www.biausa.org/> (Brain Injury Association)

Schizophrenia

<http://www.mhsource.com/narsad/>

<http://www.nimh.nih.gov/publicat/schizmenu.cfm>

Bipolar

<http://www.manicdepressive.org/>

Trauma related web-sites

<http://www.istss.org/>

<http://www.ncptsd.org/>

<http://www.dartmouth.edu/dms/ptsd/>

<http://www.trauma-pages.com/>

Child Abuse and Trauma

<http://www.calib.com/nccanch/> (National Clearinghouse on Child Abuse Information)

Drugs

<http://www.metrich.com/slang/slang.htm> (Lists of drug slang)
<http://www.pharma-lexicon.com/>

FAS/FAE

<http://depts.washington.edu/fadu/>
<http://www.cdc.gov/nceh/cddh/fashome.htm>
<http://www.nofas.org/main/index2.htm>

2. Books and Articles

General, non-technical books on mental illness

William Styron (1990) Darkness Visible New York: Vintage Books (depression)
Andrew Solomon (2002) Noonday Demon New York: Scribner (depression)
Michael Dorris (1989) The Broken Cord New York: Harper and Row (FAS)
Eve LaPlante (2000) Seized (epilepsy)
Dorothy Alison (1993) Bastard out of Carolina New York: Plume (Trauma)
Cathy Crimmins (2000) Where is the Mango Princess? New York: Alfred Knopf (brain injury)

The Brain

Devinsky, O and D'Esposito, M (2004) Neurology of Cognitive and Behavioral Disorders Oxford: Oxford University Press.

Goldberg, E (2001) The Executive Brain New York: Oxford University Press.

Pincus, JH and Tucker, GJ (1985) Behavioral Neurology 3rd Ed. New York: Oxford University Press.

Mental Retardation

American Association on Mental Retardation (AAMR) Mental Retardation: Definition, Classification, and Systems of Supports, 10th Ed. (2002) Washington, D. C.

Edgerton, Robert (1993). The Cloak of Competence Berkeley: University of California Press

Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effect (FAE)

Streissguth, AP, et al. (1996) Understanding the occurrence of secondary disabilities in clients with fetal alcohol syndrome (FAS) and fetal alcohol effects (FAE) (Final Report) Fetal Alcohol and Drug Unit, University of Washington School of Medicine, Centers for Disease Control and Prevention Grant No. RO4/CCR008515.

Streissguth, AP et al. (1991) Fetal alcohol syndrome in adolescents and adults. Journal of the American Medical Association 265(15) 1961.

Streissguth, AP, et al. (1985) Natural history of the fetal alcohol syndrome: A 10-year follow-up of eleven patients. The Lancet Vol. II for 1985, No. 8446 p.85.

Streissguth, AP (1977) Maternal drinking and the outcome of pregnancy: Implications for child mental health American Journal of Orthopsychiatry 47(3) 422.

Pesticide/Metal/Solvent Exposure

Ecobichon, D ed. (1988) Occupational Hazards of Pesticide Exposure Philadelphia: Taylor and Francis.

Ecobichon, D and Joy, R (1994) Pesticides and Neurological Diseases 2nd Ed. Boca Raton: CRC Press.

Feldman, R (1999) Occupational and Environmental Neurotoxicity Philadelphia: Lippincott-Raven.

Greater Boston Physicians for Social Responsibility (2000) In Harm's Way: Toxic Threats to Child Development Boston: PSR.

National Research Council (2000) Toxicological Effects of Methylmercury Washington, D.C.: National Academy Press.

Wright, DA and Welbourn, P (2002) Environmental Toxicology Cambridge: Cambridge University Press.

Mild Traumatic Brain Injury

Murrey, GJ (2000). The Forensic Evaluation of Traumatic Brain Injury Boca Raton, FL: CRC Press.

Schizophrenia/Psychotic Disorders/Bipolar Disorder

Amador, X (2000) I am not sick, I don't need help New York: Vida Press.

Jamison, KR (1995) An Unquiet Mind New York: Knopf.

Papolos, D and Papolos J (2002) The Bipolar Child (revised edition) New York: Broadway Books.

Torrey, EF et al. (1994) Schizophrenia and Manic-Depressive Disorder New York:

BasicBooks/HarperCollins.

Torrey, EF (1995) Surviving Schizophrenia 3rd Ed. New York: HarperCollins.

Vinogradov, S ed. (1995) Treating Schizophrenia San Francisco: Jossey-Bass.

Trauma and PTSD

Briere, JN (1992) Child Abuse Trauma: Theory and treatment of the lasting effects Newbury Park: Sage Publications.

Buka, S et al. (2001) Youth exposure to violence: Prevalence, risks, and consequences American Journal of Orthopsychiatry 71(3) 298-310.

Carlson, E (1997) Trauma assessments New York: The Guilford Press.

Crocker, P (1999) Childhood abuse and adult murder: Implications for the death penalty North Carolina Law Review 77 NCL Rev 1143.

Herman, J. L. (1992) Trauma and Recovery New York: Basic Books.

Hunter, M. (1990) Abused Boys: The Neglected Victims of Sexual Abuse. Massachusetts: Lexington Books.

Malinosky-Rummell, R and Hansen, DJ (1993) Long-term consequences of childhood physical abuse Psychological Bulletin 114(1) 68-79.

Polusny, MA and Follette, VM (1995) Long-term correlates of child sexual abuse: Theory and review of the empirical literature Applied and Preventive Psychology 4:143-166.

Rossmann, BBR and Rosenberg, MS (1998) Multiple victimization of Children New York: Hawthorne Press.

van der Kolk, BA, et al. (1997) Traumatic Stress Washington, D.C.: American Psychiatric Press.

Polysubstance abuse

Leshner, AI (1998) Addiction is a brain disease - and it matters National Institute of Justice Journal 237:2-6, Washington, D.C. NIJ.

Majewska, MD ed. (1996) Neurotoxicity and Neuropathology Associated with Cocaine Abuse NIDA Research Monograph 163, Rockville, MD.

National Center on Addiction and Substance Abuse at Columbia University: Behind Bars: Substance abuse and America's prison population 1998.

Personality disorders

Kazdin, A (1995) Conduct Disorders in Childhood and Adolescence 2nd ed. Thousand Oaks, CA

Livesley, WJ ed. (2001) Handbook of Personality Disorders New York: The Guilford Press.

Mash, EJ and Barkley, RA eds. (2002) Child Psychopathology 2nd Ed. New York: The Guilford Press.

Neuropsychology and Neurological Testing

Caplan, L and Hollander, J (2001) The Effective Clinical Neurologist Boston: Butterworth/Heinemann.

Groth-Marnat, G ed. (2000) Neuropsychological Assessment in Clinical Practice New York: John Wiley and Sons.

Kaufman, AS and Lichtenberger, EO (1999) Essentials of WAIS-III Assessment New York: Wiley and Sons.

Lezak, MD (1995) Neuropsychological Assessment, 3rd Ed. New York: Oxford University Press.

Rogers, R (1997) Clinical assessment of malingering and deception, 2nd Ed. New York: The Guilford Press.

Sbordone, RJ and Saul, RE (2000) Neuropsychology for Health Care Professionals and Attorneys 2nd Ed. Boca Raton: CRC Press.

Sweet, JJ ed. (1999) Forensic Neuropsychology Lisse: Swets and Zeitlinger Publishers.

Other Books on Mental Illness

Saddock, BJ and Saddock, VA (2000) Kaplan and Sadock's Comprehensive Textbook of Psychiatry, 7th Ed. Philadelphia: Lippincott, Williams and Wilkins.

Torrey, EF (1997) Out of the Shadows: Confronting America's Mental Illness Crisis New York: John Wiley and Sons.