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Visual Hallucinations Differential Diagnosis Annotated Decision Tree

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Abstract

Differential diagnosis of the etiology of visual hallucinations is challenging. Although visual hallucinations can be symptomatic of psychiatric disorder, they more commonly indicate neurological or medical disorders, sensory impairment, or substance intoxication or withdrawal. Accurate diagnosis and treatment is crucial given that misdiagnosis and incorrect treatment intervention can have profound consequences. The purpose of this paper is to summarize the most prevalent causes of visual hallucinations, review the DSM-5 hallucination decision tree, and provide an annotated visual hallucination differential diagnosis decision tree.

Keywords: Visual hallucination, differential diagnosis, decision tree
Visual Hallucinations Differential Diagnosis Decision Tree

While I was training as a psychology graduate student on a neuropsychology practicum, a patient presented for a neuropsychological evaluation with a personal history of visual hallucinations. The referral question was to assess his current level of cognitive functioning and to make a differential diagnosis. Rule outs included major depression with psychotic features and dementia with Lewy bodies. If he had depression with psychotic features, psychiatry would likely begin a trial of antipsychotic medication in addition to his antidepressant medication. If it was determined to be dementia with Lewy bodies certain antipsychotics may be contraindicated. Ultimately, it was determined that the patient had serotonergic syndrome after mixing antidepressant medications with over-the-counter herbal supplements. He had initially denied taking supplements, but subsequently acknowledged doing so after neuropsychological testing.

Visual hallucinations can be indicative of a wide range of disorders, both psychiatric and non-psychiatric in nature. Visual hallucinations are one potential feature of psychosis, and a psychology trainee should be well versed in the various non-affective and affective psychiatric disorders that most commonly give rise to them, such as schizophrenia, bi-polar disorder, and depression. However, a wide range of neurologic and medical conditions can present with visual hallucinations, including seizures, metabolic or endocrine disorders, and infection, and these may require immediate medical attention and specialized medical knowledge to adequately diagnose. Visual hallucinations may also be related to substance intoxication or withdrawal, which may or may not be comorbid with underlying psychiatric and/or non-psychiatric conditions. Visual hallucinations may also be related to sleep disturbances. They may also fall into the realm of normal experience, particularly if occurring immediately before or after sleep, or following the death of a loved one (Teeple, Caplan, & Stern, 2009).
Examining the potential etiologies of visual hallucinations regarding the case above made me wish for a systematic visual hallucination differential diagnostic decision tree. This desire prompted the idea for this paper; to present a beginning framework and questions to guide a psychology trainee through the steps of what to consider at each decision tree juncture. This experience also prompted me to examine the process, potential for misdiagnosis, and logical errors that may occur with differential diagnosis.

Diagnosis itself has many potential functions. Diagnosis should be valid and more importantly useful (Kendall & Jablensky, 2003). Diagnosis has the greatest utility when it informs evidence-based treatment options, identified in a timely manner (McGorry & van Os, 2013). Diagnosis may be used to label or to identify a condition or set of symptoms, provide shorthand for clinicians to communicate with each other, and help with billing codification and payment. Accurate diagnosis may help to promote understanding of the disorder and the potential course of the disorder. Diagnosis may impact a patient's access to care. Diagnosis may also be used to stigmatize.

Diagnoses from the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, 2013), and to a lesser extent the ICD-10 (International Statistical Classification of Diseases and Related Health Problems, Tenth Edition, 1990), often are used in a top down approach. A clinician starts with the suspected diagnosis or diagnoses and then compares the presentation, and if possible the course of the presentation and response to treatment. The top down approach although often used, is not recommended because it is subject to confirmation bias and assumes reliability within categories, which is often not the case.

Starting with a symptom, such as visual hallucinations, leads to a bottom to top approach to diagnosis. In theory one would consider all of the possible etiologies of the symptom and
work upwards towards differential diagnosis in a systematic manner attempting to recognize the pattern of the symptom as it relates to potential diagnoses. The bottom-to-top approach would not be useful if the symptom was common such as a headache, fever, or low mood. However, with a symptom that is rare this approach is useful.

Beside this limitation, the bottom-to-top approach has several benefits and drawbacks. This approach creates the potential for improved diagnostic accuracy. In addition, increased awareness and understanding of the mechanisms underlying varying diagnoses with similar symptomology, could potentially inform future research and treatment. Downsides include the amount of additional time and expense it could take to make a diagnosis, which in many cases may not be an available luxury. Another downside is the knowledge base required to approach a diagnosis in this manner. Expecting a single clinician to have a substantial fund of knowledge of all the potential diagnoses related to visual hallucination that occur across various fields and disciplines is unrealistic.

Thus, creating a single decision tree is challenging. Given that visual hallucinations can initially present to providers in many medical and mental health disciplines, including psychiatry, psychology, neurology, neuropsychology, emergency medical services, primary care, nursing, and assisted living, the interplay of these fields should be considered when making a diagnosis. These field specialties each have their own approaches to problem solving and different lenses through which to view differential diagnosis. Doctors may refer to psychiatry, neuropsychology or psychology when the patient’s presentation does not fit the expected clinical picture, just as neuropsychology and psychology may refer a patient to neurology if the clinical picture is unclear and does not fit the expected pattern of presentation for a psychiatric disorder. Respect for the bounds, capabilities, limitations, ethical considerations, and awareness of the
assumptions of each field is warranted. However, a basic understanding of alternative explanations for visual hallucination and the prevalence rates of those disorders may help to lead to more accurate differential diagnosis and treatment. Therefore, information regarding the most prevalent etiologies of visual hallucinations, and prevalence rates in the general population as well as base rates in other more specific populations will be indicated when available.

The hallucination decision tree of the DSM-5 Handbook of Differential Diagnosis (see Figure 1.0) (First, 2014) is used as an initial decision tree framework as applied to visual hallucinations. In this paper I will address the value and utility of the model, as well as the potential errors in clinical judgment, assessment, treatment, diagnosis, and logic this model may inadvertently perpetuate when applied to visual hallucinations. Questions regarding what to consider at each juncture of the decision tree will also be presented.

**Prevalence Rates and Differential Diagnosis**

It is important to remember the medical school saying attributed to Dr. Theodore Woodward, “When you hear hoof beats, think horses not zebras” (Sotos, 2006, p1). As clinicians, we increase our vulnerability to misdiagnosing if we do not sufficiently consider base rates and acknowledge the limits of our clinical judgment (Karson & Nadkarni, 2013). In addition we are vulnerable to making rare diagnoses rather than seeing the more common diagnoses due to the availability heuristic; the phenomenon in which that which is more memorable stays in mind more than that which is more probable and because “the striking and novel stays longer in mind” (Sotos, 2006). We are also likely to look for a psychiatric condition rather than a medical condition with psychiatric features due to the availability of our training and fund of knowledge in psychology compared with the limited scope of our medical knowledge.
Prevalence rates do not conclusively tell if the individual patient has the diagnosis. Diagnosis depends on whether the individual patient has the disorder, not on a statistic based on the group (Harvey et al., 1979). However, given that there are usually alternate diagnoses to which the patient’s symptoms may fulfill diagnostic criteria, the clinician needs to remember that among those potential diagnoses, zebras and horses exist and may even be considered potential rule-outs, particularly if the presentation of the course of the disorder is not as expected. Prevalence rates may serve to remind the clinician what is statistically common in the group studied; they may indicate whether if one is looking at a zebra or a horse. Put differently, in a Bayesian analysis, an oddity in presentation or history changes the situation and requires a different base rate. Thus, the patient above not having taken supplements would have put him in a group where the base rate for the ultimate diagnosis was much lower than it was for the group that taking supplements put him in. If you are in Tanzania, think zebras not horses.

Given the low prevalence/base rate of visual hallucinations, as well as the low base rates of the many etiologies behind them, caution is advised regarding base-rate fallacy. A discussion of base rates, Bayes theorem, base rate fallacy, and Sutton’s law is in order. Regarding base rates, also called prevalence, it is important that the base rate/prevalence is the “naturally occurring rate of a condition in a population” (American Psychological Association, 2007, p. 103). Base-rate fallacy is a decision-making error in which information about rate of occurrence in a population (base rate information) is ignored or not given proper weight” (American Psychological Association, 2007, p. 103). For example, if I told you we were going to a business retreat island vacation and 90% of those on the island were psychologists and 10% were morticians, one might assume a gentleman on the island with an interest in dead bodies was one of the morticians and downplay the fact that 90% of the people are psychologists. “Bayes’
Theorem is a formula derived from probability theory that relates two conditional probabilities: the probability of the event A, given that even B has occurred, \( p(A/B) \), and the probability of event B given that event A has occurred, \( p(B/A) \). It serves as a basis for linking prior and antecedent probabilities” (American Psychological Association, 2007, p. 105). The formula is as follows:

\[
p(A \mid B) = \frac{p(B \mid A) p(A)}{p(B \mid A) p(A) + p(B \mid \sim A) p(\sim A)}.
\]

Suppose that a new blood test has been discovered that correctly identifies 90% of those with schizophrenia as having it, but falsely identifies those without schizophrenia as having it 10% of the time. This sounds like a fairly good test, right? What do you intuitively assume the probability that a person actually has schizophrenia given a positive test result? For simplicity’s sake, assume that the prevalence rate of schizophrenia is 1% and \( A \) = an individual has schizophrenia and \( B \) = a positive test result. The equation would be

\[
(.90)(.01)/(.01)(.90) + (.99)(.10).
\]

The result would be an 8.3% chance of actually having schizophrenia with a positive test result (because the a priori probability is so low). Thus, intuitive judgment may get one into trouble with diagnosis. When the prevalence rate is so low and the diagnostic gold standard is not clear, there is incredible room for incorrect diagnosis.

Clinicians also need to remember Sutton’s law, which states that a clinician should conduct assessment regarding the most obvious diagnosis first, balancing it against the potential of risk to the patient of not addressing another less common diagnosis (Rytand, 1980). In the differential diagnosis of the etiology of visual hallucinations, one needs to remember there are many zebras and a few horses, with the added concern that several of the diagnoses are potentially fatal.

Regarding visual hallucinations, suppose the case of a 23 year old patient presenting at 2 o’clock in the morning at an ER in New Orleans during Mardi Gras; statistically the visual
hallucinations are likely substance related, but not necessarily. Diagnosis could also be the onset of schizophrenia, a head injury, infection, or one of many other etiologies. However, if the main presenting feature is visual hallucinations without auditory hallucinations the odds are again very low that it is schizophrenia in a non-inpatient environment, and presumably even lower in a non-inpatient environment that is also a substance rich environment. For example, consider the prevalence rates for visual hallucinations of patients with schizophrenia are between 16-72%. The higher rates are gleaned from studies of patients in inpatient units, the lower rates are from studies of those with a diagnosis in the general population, and if visual hallucinations are present, auditory hallucinations are generally co-occurring, although not necessarily at the same time. Given the prevalence of schizophrenia is estimated to be 0.7% according to the DSM-5 that would mean the prevalence rate for an individual to present with a symptom of visual hallucinations is very low. In contrast between 7-25% of people presenting with psychotic features for the first time are determined to have substance/medication use or withdrawal in various populations. Also, considering the 23 year-old hallucinating at Mardi-Gras, it would be prudent to know first if the patient had used substances. Then one would rule-out the most likely life threatening causes by utilizing the simplest and most accurate measure, which has the lowest false positive rate. This spotlights another consideration: the base rate for diagnostic validity and specificity within a population.

Regarding diagnostic validity and specificity, for the point of this discussion I am going to move from the Mardi-Gras example and consider Alzheimer’s disease. The gold standard of diagnostic accuracy for Alzheimer’s disease is diagnosis based on autopsy findings. Not an ideal method of diagnostic accuracy when one hopes a patient to survive. This highlights the importance of balancing patient safety and diagnostic accuracy. However, prior to autopsy with
what specificity and sensitivity may diagnosis be made? The answer is so low as to recognize that it is generally diagnosed as probable Alzheimer’s disease.

Considering, psychological diagnoses with what specificity and validity is a diagnosis of schizophrenia made? What is the gold standard of diagnosis? What are the sensitivity and specificity of diagnostic measures? There have been multiple attempts to identify biological markers and early identifiers of schizophrenia and there is to date no gold standard of diagnosis (American Psychiatric Association, 2013). What are the base rates for false positives, false negatives, and diagnostic accuracy? As psychologists we diagnose based on what we believe we know and what we have learned as psychologists about symptom criteria that diagnoses describe, as well as what we know about the patient. We often take a history (psychiatric, social, and medical) of the patient and perhaps the patient’s family during a formal or informal interview, and if possible we may collect collateral information. From this information, clinicians attempt to determine what happened or changed prior to onset (e.g., was the onset acute or slow and insidious). Then we rule out likely alternate causes. Then clinicians compare the cluster of symptoms to the diagnostic cluster of symptoms accepted at the time as the diagnostic definition and determine whether what has been observed or reported fits the diagnosis. Clinicians may use psychological and neuropsychological assessment for diagnostic clarification as well. After diagnosis, if clinicians remain in contact with the patient, which may be a big if given many patient populations and clinical settings, clinicians the attempt to determine if the course of the disorder is as expected and responds to treatment as expected, or does not.
Neuropsychological/Psychological Assessment

As stated above, as a part of differential diagnosis clinicians may use psychological assessment and/or neuropsychological assessment measures. The DSM-5 hallucination diagnostic decision tree lists neuropsychological performance as a factor to consider at several diagnostic junctures. Neurocognitive domains that may be assessed with neuropsychological assessment include complex attention, executive function, learning and memory, expressive and receptive language, perceptual motor abilities, and social cognition. Valuable data may be gained from testing to help inform the level of functioning and diagnosis, when compared to the patient’s history and current level of functioning. However, regarding neuropsychological assessment it is important to remember the diagnostic validity and specificity of the assessment measures used.

Regarding the degree to which a measure tests what it is claiming to test (validity), the probability that a test accurately identifies those who do have the specific diagnosis (specificity) and the degree to which the test accurately identifies a negative diagnosis for those who do not have the diagnosis (American Psychological Association, 2007), it is important to remember the conclusion of the 1996 Neuropsychology Assessment panel:

No neuropsychological tests have been shown to have consistent diagnostic validity. Some tests accurately distinguish between two or three diseases when samples of patients with these diseases are assessed, but no study has shown that neuropsychological tests have positive predictive values when a wide variety of disorders are tested” (593).
Neuropsychological tests provide data that can be used as part of the diagnostic process, but no neuropsychological test supersedes the judgment of the clinician. Tests may be used to help quantify or describe a current level of function, but not to predict.

In addition, researchers often mistake “the null hypothesis in research designs and group statistics (e.g. means, standard deviations, correlation coefficients, etc.) for research that directly quantifies how well (or how poorly) our tests actually quantify individuals” (Smith et al, p 40). This is said not to undercut the utility and value of neuropsychological testing in differential diagnosis, but to reiterate that group norms of expected performance of patients with a given disorder may be very similar to patterns of performance of groups of patients with other disorders as well, or even groups without identified disorders. Also, part of neuropsychological testing performance looks at deviation from the norm the individual has from the population, and what is expected given the patient’s age and education. Individual testing performance can be influenced by anxiety, sleep deprivation, sickness, medication, pain, and many other variables. Even in “normals” being tested there is significant deviation from the norm by one standard deviation. As stated by Binder, Grant and Iverson:

Abnormal performance on some proportion of neuropsychological tests in a battery is psychometrically normal. Abnormalities do not necessarily signify the presence of acquired brain dysfunction because low scores and large intra-individual variability often are characteristic of healthy adults. We recommend that test battery developers provide data on the amount of variability in normal samples and also provide base rate tables with false positive rates that can be used clinically when interpreting test performance (2009, p.31).

For example the base rate for a 25-point score discrepancy on the Working Memory
Index for someone with an IQ score in the 90-109 range on the Wechsler Adult Intelligence Scale, Forth Edition (WAIS –IV )is 10.7%. Another study addressing flexible neuropsychological batteries found “most (73%) of the healthy older adults had one or more scores at or below the 10th percentile and 37% had one or more scores at or below two standard deviations from the mean” (Binder, Iverson, & Brooks, 2009, p. 31; Palmer et al., 1998). Again, this is stated not to diminish the potential utility of testing, but to remind clinicians of what assessment can and cannot do.

**Visual Hallucination**

At the first branch of the DSM-5 Hallucination Decision Tree one is to determine whether the symptom is a hallucination. Hallucination is defined in the DSM-5 as “a perception-like experience with clarity and impact but without the external stimulation of the relevant sensory organ” (American Psychiatric Association, 2013, p. 822). The DSM-5 further clarifies the definition by stating that visual hallucinations are to be distinguished from misperceptions or misinterpretations of an external event as occur in illusions. This definition does not sufficiently take into account attribution error, nor does it truly clarify what a visual hallucination is. Perception, imagination, and hallucination are identical in that they may all be classified as perception behaviors. They all involve the same behavior of seeing. However, in perception that which is seen is in front of you; in imagination it is not and you know it is not; in hallucination it is not but you think it is. The same behavior of seeing is happening whether the scene is there or not (Karson, 2006). Patients with insight regarding their hallucinations are often hesitant to tell providers of their hallucinatory experiences for fear of being seen as having a psychiatric condition (Shea, 1998). Also, if a patient readily acknowledges that what was seen was not there, would this not be classified as a visual aberration instead of a visual hallucination?
Conversely, patients without insight are likely to not report hallucinations because they are unaware their experiences are hallucinatory. It is up to the provider to observe if a patient is acting as they are responding to a visual hallucination and to question about visual hallucinations in a manner that is open and reduces perceived stigma about hallucinations (Zuckerman, 2010; Shea, 1998). Providers must also consider whether there is potential gain to be obtained by the report and consider the possibility of malingering in such cases (Lezak, Howieson, Loring, Hannay, & Fischer, 2004).

Visual hallucinations with formed objects such as people are called complex hallucinations. Those with unformed images such as auras, light flashes, or patterns are called simple hallucinations (Moore & Puri, 2012). Visual hallucinations that occur during the daytime and do not occur immediately before or after sleep have a lifetime prevalence rate of 3.2% in the general population (Ohayon, 2000). Hallucinations that occur while falling asleep (hypnogogic) or waking (hypnopompic) are considered “within the range of normal experience” (American Psychiatric Association, 2013, p. 88). Almost one-third of the general population may experience complex visual hallucinations while in the state proceeding or following sleep (Ohayon, 2000). However, although complex hallucinations affect normal populations, they also occur in pathological condition (Manford & Adermann, 1998). Visual hallucinations have a bimodal distribution prevalence with respect to age. Psychosis related visual hallucinations occur more often in late adolescence and early adulthood. Visual hallucination related to neurodegenerative disorders, and eye disease occur more frequently in elderly populations (Waters et al., 2014).

Visual hallucinations may be a symptom of non-affective and affective psychiatric disorders, such as schizophrenia, bi-polar disorder, or depression (American Psychiatric Association, 2013). Often patient report of visual hallucinations does generate psychiatric
consultation, even though visual hallucinations “are not pathognomonic of a primary psychiatric illness” (Teeple et al., 2009, p. 26). Although a feature of psychosis and often considered psychiatric in nature, the etiology of visual hallucinations is greatly varied, as are the appropriate treatments and interventions.

Visual hallucinations are frequently indicators of a number of neurologic and medical conditions such as seizures, metabolic or endocrine disorders, and infection, as well as substance intoxication or withdrawal, rather than indicative of psychiatric disorder (Cummings & Miller, 1987; Duwe & Turetsky, 2002; Hall, Popkin, Devaul, & Faillace, 1978; Sacks, 2013; Shea, 1998; Teeple et al., 2009). Hallucinations due to neurological or medical disorders often are distinguishable from schizophrenia spectrum disorders by having a higher prevalence of prominent visual hallucinations, and a lower prevalence of bizarre behavior, thought disorder, rapid speech, and negative symptoms (Cornelius et al., 1991). However, the prevalence of visual hallucinations is higher than previously believed in psychiatric conditions (Waters et al., 2014). If and how visual hallucinations are etiologically related in psychosis, neurodegenerative disorders and eye disease remains unclear (Waters et al., 2014).

There are multiple mimics of psychiatric dysfunction that should be considered when a patient initially presents with visual hallucinations (Teeple et al, 2009; Shea, 1998; Duwe & Turetsky, 2002). When a patient presents with a visual hallucination, one should suspect organic, medical or toxin etiologies (Shea, 1998). Disturbances in sleep are linked to visual hallucinations, even if a formal diagnosis of sleep disorder or narcolepsy is not present. Visual hallucinations linked to sleep disturbance are also found in Parkinson’s disease, PTSD, peduncular seizures, Lewy Body dementia, stoke, migraine, epilepsy, Charles Bonnet syndrome, and schizophrenia (Manford & Adermann, 1998).
Accurate diagnosis of the etiology of visual hallucinations can be difficult, especially given similar presentations between disorders, co-morbid conditions, and the low prevalence rates of many of the potential etiologies. Prompt medical evaluation is recommended with the initial presentation of psychotic features, particularly if onset is acute with no prior history of hallucination or other psychotic features (Hall, Popkin, Devaul, & Faillace, 1978; Shea, 1998). Proper diagnosis and treatment is crucial, as misdiagnosis of those with visual hallucinations can have profound and even life threatening consequences. Even with careful psychiatric interviewing, medical examination and diagnosis, misdiagnosis does happen. A clinician should be aware of treatment effectiveness and what does not fit the clinical and psychological picture and re-evaluate (Shea, 1998). For example, schizophrenia and narcolepsy can have similar symptom presentations and can be difficult to differentially diagnose (Talih, 2011). Studies have indicated that patients with narcolepsy have been misdiagnosed with schizophrenia, placed in psychiatric hospitals and treated with anti-psychotic drugs; in fact, antipsychotic drugs may increase the psychotic features, including visual hallucinations in patients with narcolepsy. An incorrect diagnosis can contribute to a cycle of psychiatric symptoms, hospitalization, decreased quality of life, and economic and societal impact continued in some cases for years until the correct diagnosis was identified and the incorrect treatment ceased. This is just one example of the potential profound impact of misdiagnosis.

Given that neuropsychological testing, neurological imaging, lab tests and psychological testing all have their limitations regarding validity, specificity, and base rates of inaccurate diagnosis, and that norms for testing are based on the group norms, the psychiatric interview is an important diagnostic tool. The interview should obtain pertinent information regarding the patient’s age, substance/medication use and/or discontinuation, medical history, psychiatric
history, presence, onset, type of onset, behavioral, psychiatric, mood, or cognitive symptoms, estimated pre-morbid functioning, family medical, psychiatric and neurological histories. Attribution error and malingering should also be considered.

Questions to be considered at this juncture are:

1. Is the event described a hallucination, misperception, illusion, or imagination?
2. Is there attribution error on the part of the patient or clinician?
3. Is the hallucination hypnagogic or hypnopompic or not?
4. What type of hallucination is it, complex or simple?
5. Is insight intact? If so, how did the person identify the hallucination or visual aberration?
6. Is the hallucination disturbing to the patient?
7. What is the context of the hallucination?
8. What was the duration of the hallucination?
9. What does the patient believe the consequences of the hallucination are?
10. Are other hallucinations or delusions present?
11. Are there negative symptoms?
12. Is there potential gain or secondary gain?
13. Is this the first time a visual hallucination has presented? If not, what were the previous hallucinations and in what context?

**Substance/Medication Induced Visual Hallucinations**

The next juncture of the decision tree asks the clinician to determine whether the visual hallucination is due to the physiological effects of a substance/medication. Substance or medication use and/or withdrawal symptoms are statistically a likely cause of visual
hallucinations. Substance use among patients presenting with first episode of psychosis is two times that of the general population (Barnett et al., 2007). Substance use is present in the majority of people presenting with first episode psychosis (Barnett et al., 2007), and “between 7-25% of individuals presenting with a first episode of psychosis in different settings are reported to have substance/medication induced psychotic disorder,” with the higher rates reported in emergency room settings (American Psychiatric Association, 2013). However, it is possible that people with new onset psychotic disorders are more likely to use substances.

Distinguishing between substance-related psychotic symptoms and primary psychotic illness is critical, because each requires different treatment. Some cases may require treatment with medication and medication may be contraindicated in other cases. Studies indicate that visual hallucinations were more common with substance related psychotic symptoms and that negative symptoms of psychosis were more frequently related with primary psychosis. However, studies also indicate, "psychotomimetic drug use may precipitate a chronic schizophrenic illness" (Caton et al., 2005, p. 143). According to the DSM-5 substance or medication induced psychotic disorder has unknown prevalence rates in the general population. Studies have indicated a prevalence rate of diagnosed substance induced psychotic disorder in the general population of 0.43% (Peralta et al., 2007). However, one should note that the statistic for those with diagnosed substance induced psychotic disorder does not include those with substance withdrawal, substance withdrawal delirium, substance intoxication, or substance intoxication delirium, all which may present with visual hallucinations. In my first year as a practicum student I noticed a patient in his 50’s looking around the room while I was speaking to him following surgery. When I asked him what he saw, he described seeing a mouse. There was no mouse, but he was unaware that he was beginning to have hallucinations related to alcohol withdrawal. When
admitted to the hospital for surgery he had denied regular alcohol use. In fact he denied alcohol use until the unreality of the hallucination symptoms, the course of treatment and potential risks of alcohol withdrawal were described to him. Fortunately, when the patient was faced with potential medical complications his wife provided his heavy alcohol use history, and treatment with benzodiazepines ensued.

Besides denial of substance use or withdrawal patients and/or providers may be unaware of or fail to research substance/medication interaction (Zuckerman, 2010), or the interaction may be unknown. For example, suppose that you have a patient taking 16 prescribed medications, as well as herbal supplements and vitamins; it is likely that given all the permutations of substance interaction there are no available studies researching someone taking those 16 medications and additional supplements. The interactions and side effects may be unknown, particularly in an older patient who is medically compromised. Patients may also not take their medication as prescribed. Given these variables it is important for the clinician to keep substance/medication use or withdrawal in mind even after the initial consultation, particularly if the clinical picture remains unclear and psychological data does not fit the pattern of hallucinations and behavior. Symptoms of substance/medication intoxication and withdrawal include autonomic hyperactivity, pupillary dilation, nystagmus, sweating, increased hand tremor, insomnia, nausea, hallucinations, psychomotor agitation, anxiety, generalized tonic seizures, impaired judgment, and confusion.

Drugs and medications associated with visual hallucinations include street drugs such as alcohol, cocaine, PCP, methylenedioxymethamphetamine (Ecstasy), amphetamine, mescaline, d-lysergic acid diethylamide (LSD), opioids, and cannabis; psychotropic medications such as benzodiazepines, L-dopa, dopaminergic, neuroleptic, anti-cholinergic, serotonergic, sedative,
anxiolytic, tricyclic antidepressants, benztropine, narcotics; non-psychotropic medications such as digoxin, glucocorticoids, amantadine, cimetidine, ranitidine, sildenafil, beta-blockers, clarithromycin; and over the counter drugs such as ephedrine and phenylpropanolamine (Liu, Volpe, & Galletta, 2001; APA, p. 482). Substance/medication related visual hallucinations are one of the few horses in the diagnostic decision tree and combined with medical implications they should be a primary consideration and rule-out.

Alcohol is the most commonly used drug associated with visual hallucinations. It is estimated that 12.4% of adult men and 4.9% of adult women have alcohol substance use disorder. It is also estimated that approximately 50% of middle-class individuals with alcohol use disorder experience full alcohol withdrawal syndrome and more than 80% of those with alcohol use disorder who are hospitalized or homeless may experience withdrawal. Transient visual, auditory, and tactile hallucinations may accompany withdrawal. Hallucinations may occur outside of delirium. Less than 10% of those will develop alcohol withdrawal delirium or withdrawal seizures (American Psychiatric Association, 2013). However, the answer to the question as to whether or not substance/medication use or withdrawal is involved can be more difficult to determine than one may initially anticipate. This determination is important because withdrawal from alcohol can be fatal for heavy drinkers, and if necessary, medical treatment should begin as soon as possible.

Even before we have considered other substances such as stimulants, hallucinogens, opioids, sedatives, cannabis, and other medication, it is of note that it is statistically more likely for substance/medication involvement than psychiatric disorder alone. Also important statistically is the higher prevalence of substance use by young adults (American Psychiatric Association, 2013). The prevalence rate of substance abuse among people with severe mental
illness is significantly higher than that in the general population. Diagnostic accuracy is particularly critical in the onset of a psychiatric disorder, at a time when the clinical picture may be clouded by substance use (Caton et al., 2005). Diagnostic stability is also a question. Those diagnosed with drug-induced psychosis generally fall into one of two categories. Either the patients are discharged with no follow up for several years or they have a longer hospital stay, are referred to log-term follow-up care and have a change in diagnoses (Komuravelli, Poole, & Higgo, 2011). In addition to diagnostic accuracy, substance use history is also important because it is strongly correlated with non-compliance to psychiatric treatment (Weiss, Smith, Hull, Courtney, & Huppert, 2002).

Regarding this juncture of the decision tree, it is important to consider:

1. Which substances/medications have been used or discontinued?
2. Are signs of autonomic disturbance, agitation, tremor, and other indicators of withdrawal apparent?
3. Is medical referral necessary due to risk to patient?
4. If substances are related, is there also a co-morbid psychiatric condition?
5. You may not be getting the truth regarding substance use, continue to watch for signs of substance involvement, even if initially denied.
6. Substance use/withdrawal related hallucinations often are visual, tactile and auditory and are usually transient, but may persist.
7. Prevalence rate amongst the general population in various settings is 7-25%.
8. The prevalence rate of substance use among young adults is higher, and prevalence rate of medication use among older adults is higher.
9. Substance use is much more frequent among those with primary psychiatric disorders than the general population.

10. Medications such as antipsychotic and psychotomimetic medications may be contraindicated.

**Visual Hallucinations Due to Physiological Effects of Medical Condition**

The next juncture of the DSM-5 hallucination decision tree asks the clinician to determine if the hallucination is due the physiological effects of a medical condition and then to consider whether the patient presents with fluctuating attention and awareness, indicating delirium, or with neurocognitive impairment in at least one cognitive domain such as complex attention, executive function, social cognition, perceptual motor ability, language, learning and memory. The DSM-5 has reclassified Dementia, Delirium, Amnestic, and Other Cognitive Disorders as Neurocognitive Disorders (NCD). Visual hallucinations related to physiologic conditions may range from simple to complex and the patient may or may not have insight that the hallucination is a hallucination and not real. As with determining whether a hallucination is substance or medication related, determining if a hallucination is due to a medical condition is crucial as some of the conditions associated with visual hallucinations are life threatening and prompt treatment is imperative (Shea, 1998; Teeple et al., 2009).

**Delirium**

The clinician is first asked to determine whether the patient is experiencing delirium. Delirium is a medical condition and can indicate life threatening medical disorders. Delirium is defined as a “disturbance of attention or awareness that is accompanied by a change in baseline cognition that cannot be better explained by existing or evolving neurocognitive disorder” (American Psychiatric Association, 2013, p. 599). The change develops over a short period of
time from a few hours to a few days and may fluctuate over the course of a day (American Psychiatric Association, 2013). However, delirium is not always acute in onset and it is possible for onset to be insidious (Shea, 1998).

In the general population the prevalence of delirium is 1-2%, but it is as high as 14% in those over 85 years of age. The prevalence of delirium in patients at hospital admission ranges from 14%-24%. Prevalence rates for delirium during hospitalization range from 6%-56% in general hospital settings and 70%- 87% of older individuals in intensive care. Prevalence rates for delirium at the end of life are as high as 83% for all individuals.

Delirium may precede stupor, coma, and death if underlying causes are not treated. Mortality rates among those hospitalized with delirium are as high as 40%. Deliriums characterized by vivid hallucinations, delusion, language disturbance, and agitation must be distinguished from psychotic disorders (American Psychiatric Association, 2013, p.600). Estimations of prevalence of visual hallucinations among delirious patients are as high as 75% (Cummings & Miller, 1987). The presence of visual hallucinations should prompt a provider to strongly consider organic origins or delirious states. Fear and anxiety are often present with atypical affect. Orientation and short-term memory are frequently impaired and illogical thought or loosening of associations may appear. Delirious patients may frequently identify someone unfamiliar, such as a new doctor, as being someone familiar. Deliria tend to fluctuate and often worsen at night, which is called “sundowning” (Shea, 1998, p. 326). Delirium may be difficult to distinguish from other neurocognitive disorders, particularly in individuals with unrecognized neurocognitive disorders. Furthermore, a delirium may be superimposed on another neurocognitive disorder. However, delirium is often recognized by acuteness of onset, fluctuation of course, and often disturbance in the sleep/wake cycle (American Psychiatric
Association, 2013). Common causes of delirium are infection, metabolic disorders, neurologic disorders, post-operative sequelae, and substance/medication intoxication or withdrawal. Infections can be intracranial such as encephalitis of meningitis, or systemic, such as urinary tract infection, pneumonia, septicemia, typhoid and malaria. Common metabolic disorders include electrolyte imbalance, hyperglycemia, hypoglycemia, hypoxia, hypercarbia, anemia, abnormal levels of calcium or magnesium, vitamin-B deficiency, liver or kidney disease, and endocrine disorders such as hyperthyroidism, hypothyroidism, and adrenal disorders. Neurologic disorders that may cause delirium include trauma, seizures, stroke, hypertensive crisis, subarachnoid hemorrhage, and vasculitits (Shea, 1998). Even in patients with known psychotic disorder, if their psychotic presentation generally presents in similar manner and then presents in an atypical manner, additional etiology should be considered, because delirium warrants aggressive medical evaluation and this may be overlooked in patients with chronic psychiatric conditions. The provider should especially attend to significant problems with attention and fluctuating levels of consciousness (Shea, 1998).

Considerations at this juncture include:

1. Does the patient meet criterion for delirium with disturbance in attention and awareness?
2. Is immediate medical referral and intervention necessary for the safety of the patient?
3. Has this type of delirium occurred before and in what circumstances?
4. What is the course of the appearance of psychiatric dysfunction?
5. Are there co-morbid conditions?
6. Is this the appropriate time to diagnose psychiatric dysfunction or should the course of the delirium be assessed?

7. Review medical/psychiatric records if possible to help rule out probable medical causes as, well as psychiatric causes.

8. Assess patient’s performance with a mini-mental status exam or other quick to administer and score repeatable measure, so that scores can be compared to each other over a short time period.

9. Even if the patient has a psychiatric history, if the symptoms are different and consistent with delirium, rule-out delirium.

**Mild and Major Neurocognitive Disorders**

According to the DSM-5, “The Neurocognitive Disorders (NCDs) are unique among DSM-5 categories in that there are syndromes for which the underlying pathology, and frequently the etiology as well, can potentially be determined” (p. 591). This statement brings forth several questions. First if the etiologies are able to be determined, why not label them as such using the appropriate medical code that is already in place versus using two to three codes with additional specifiers? The question is also raised as to the utility of such general diagnoses as Mild NCD. The diagnosis of Mild NCD seems highly sensitive, not specific, and without a reliable gold standard. All that is required to meet diagnostic criteria is (a) evidence of a modest decline from a previous level of performance in one cognitive domain (complex attention, executive function, learning and memory, language, perceptual–motor, or social cognition) based on concern from the individual, a knowledgeable informant, or a clinician, and (b) that the mild decline in cognitive function be preferably documented by neuropsychological testing, or in its
absence, by another quantified clinical assessment (American Psychiatric Association, 2013). Questions of what constitutes modest decline and other quantified clinical instrument are raised, given that the base rates for “normals” to have declines of one standard deviation is psychometrically normal. The clinician is then to specify if possible whether the NCD is due to: Alzheimer’s disease, frontotemporal lobar degeneration, Lewy body disease, vascular disease, traumatic brain injury (TBI), substance/medication use, HIV infection, Prion disease, Parkinson’s disease, Huntington’s disease, another medical condition, multiple etiologies or unspecified etiology. Finally the diagnoses of Major and Mild NCD contains rule outs that the cognitive deficits are not better explained by another mental disorder. What if there is a co-morbid mood disorder that occurs? What if there are symptoms of mood disorder within the medical diagnosis? What if a patient with a mood disorder does have impairment in cognitive domains? Does that not merit charting as a neurocognitive disorder, especially given the general nature of the NCD diagnosis and the lack of appropriate cognitive functioning specifiers of the mental disorders? Finally the DSM-5 hallucination decision tree does not consider the possibility of visual hallucinations due to a medical condition without neurocognitive disorder. The DSM-5 even recognizes that “the differential diagnosis between normal cognition and mild NCD, as between mild and major NCD is challenging because the boundaries are inherently arbitrary” (p.610). Major NCD is comparable to dementia of the DSM-IV and prevalence rate at 65 years of age is estimated at 1-2% and as high as 30% by age 85 (p.608). The DSM-5 states that estimates for Mild NCD are comparable with the previous prevalence estimates of mild cognitive disorder in the DSM-IV of “2-10% at age 65 and 5-25% by age 85” (p. 608). However, since Mild NCD is so close to normal functioning as to be difficult to differentiate, prevalence rates could be quite high. Given the change in diagnostic criteria from dementia there are no
prevalence rates available for the general Mild NCD. Which again raises the question of the utility of the diagnosis of Mild NCD.

Considerations at this juncture include:

1. Is there a medical condition that better explains the psychiatric symptom of visual hallucination?
2. Has the patient had recent medical care?
3. Is a referral recommended?
4. Is neuropsychological testing necessary or is medical diagnostic testing necessary?
5. Does the patient’s medical or psychiatric diagnostic history identify a potential etiology?
6. Is this psychiatric presentation consistent with previous diagnosis and previous psychiatric symptoms if any?
7. Are there other neurologic symptoms?
8. Are there any psychiatric symptoms?
9. Are psychiatric symptoms consistent with a psychiatric diagnosis, or inconsistent, potentially indicating alternate diagnosis?
10. As with other junctures of the decision tree consider age and the prevalence rate for the most likely conditions given the patient’s presentation and age.

Visual Hallucinations and Medical Disorders

An awareness of medical diagnoses that mimic psychiatric disorders and may present with visual hallucinations is important to keep in mind, in addition to the physiological and
medical etiologies of delirium. This list is in no way complete, but rather a short list of the medical diagnoses that most often present with visual hallucinations. The prevalence rates of these disorders will be presented. Disorders to be discussed include Lewy body disease, Parkinson’s disease, Alzheimer’s disease, vascular dementia, narcolepsy, Huntington’s disease, Creutzfeldt-Jakob disease, migraine, epilepsy, seizure, stroke, and traumatic brain injury, as well as vision loss related, retinal pathology, and Charles-Bonnet syndrome (Cummings & Miller, 1987; Liu, Volpe, & Galetta, 2010; Teeple et al., 2009). The diagnoses below, which often present with visual hallucinations, are not to be confused with the diagnosis of psychotic disorder due to a general medical condition. The diagnosis of psychotic disorder due to a medical disorder “is generally not diagnosed” if reality testing regarding the hallucinations is maintained or the patient can appreciate that the hallucinations are part of a medical condition. The prevalence rate of psychotic disorder due to medical condition is estimated at 0.22% (Perala et al., 2007).

**Neurological disorders**

*Parkinson’s Disease*

Prevalence rates for Parkinson’s disease are 0.3-0.4% for the general population, up to 4.5% for the population of those over age 85 (Blin et al., 2015). Prevalence rates for visual hallucinations among those diagnosed with Parkinson’s disease are between 6-40% depending on those studied. Lower rates are indicated in the general population and higher rates in medical and assisted living settings, as well as at later stages of the disease. Prevalence of mild neurocognitive disorder (Mild NCD) is 27% among those diagnosed with Parkinson’s disease. Visual hallucinations related to Parkinson’s disease may be due to the disease process, medication effects, or sleep disturbance (Liu et al., 2010).

*Lewy Body Disease*
In the general elderly population prevalence rate estimates for neurocognitive disorder with Lewy bodies range from 0.1%-5.0%. This rate is higher in men than in women by a ratio of 1.5:1. Lewy Body disease accounts for 1.7-35% of all dementia cases. Lesions known as Lewy bodies are present in 20%-35% of autopsy confirmed dementias. Core features of the disease include two out of three of the following features: recurrent well formed visual hallucinations, fluctuating cognition with changes in attention and alertness, spontaneous features of parkinsonism with the onset of cognitive decline (American Psychiatric Association, 2013, p. 619). It is estimated that over 77% of patients with Lewy Body dementia have visual hallucinations (Del Ser et al., 2000, p. 1034). Hallucinations may become disabling and lead to nursing home placement (Liu et al., 2010). Accurate diagnosis is essential because up to 50% of those with NCDLB have neuroleptic drug sensitivity. Other symptoms of Lewy Body disease include orthostatic hypotension, autonomic dysfunction, transient loss of consciousness, urinary incontinence, syncope, repeat falls, auditory and non-visual hallucinations, delusions, apathy, REM sleep disturbance, and depression (American Psychiatric Association, 2013; Del Ser et al., 2000). The onset of Lewy Body dementia is insidious in the sixth to ninth decades of life. Most cases occur in the seventh decade of life and the course of the disease progression is usually between five to seven years (American Psychiatric Association, 2013). Lewy Body dementia is distinguished from Parkinson’s dementia by disease course and onset. In Lewy Body dementia the onset of cognitive decline precedes the motor symptoms of Parkinson’s disease by at least one year. It can also be difficult to differentiate Lewy Body dementia from Alzheimer’s disease as these diseases are frequently is co-morbid, especially in the oldest age groups (American Psychiatric Association, 2013)

*Alzheimer’s Disease*
Prevalence of visual hallucinations among patients with Alzheimer’s disease is between 3-33%. Visual hallucinations account for 85% of the hallucinations associated with this disorder. According to the 2014 Alzheimer’s Association report, it is estimated that one in nine people over the age of 65 has Alzheimer’s disease. Of those affected, two-thirds are women (Alzheimer's Association, 2014). In the context of Alzheimer’s disease, visual hallucinations are generally associated with increased cognitive decline and poor prognosis. Delusions, paranoia, and auditory hallucinations may also be present (American Psychiatric Association, 2013).

Treatment options for Alzheimer’s disease are limited. Neuroleptic medications may be used for Alzheimer’s patients with visual hallucinations, but anticholinergic medications may be contraindicated should be monitored (Liu et al., 2010).

**Stroke**

The Stroke Association reports that an estimated one percent of those diagnosed with stroke may experience psychotic features of hallucinations or delusions. Visual hallucinations following a stroke generally start within a few days of the stroke. The hallucinations may subside in a few weeks, but they may also be present for years. Peduncular visual hallucinations are generally related to mid-brain injury and may be complex, vivid and lifelike. Stroke related visual hallucinations are often co-occur with sleep disturbance, ataxia, and cognitive disturbance (Liu et al, 2010).

**Migraine**

Visual hallucinations with migraine are usually simple and consist of aura, spectra, squiggles, dots, prisms, halos, or flashing lights usually followed by a headache. Auras are the most common and occur in approximately one-third of migraine patients. These typically last from a few minutes to an hour, but may last for up to a week. Vaso-constriction induced cortical
ischemia were originally thought to cause auras, but recent research has suggested neuronal
dysfunction from cortical depression. Complex visual hallucinations of people and animals, and
visual distortions such as size distortions of one’s own body or surroundings do occur with
migraines, albeit rarely (Liu et al., 2010).

_Epilepsy/Seizures_

Visual hallucinations are not uncommon in patients with epilepsy. Studies indicate that
among patients with occipital lobe epilepsy 60% have simple hallucinations (Liu et al., 2010).
Visual field defects are associated with occipital lobe epilepsy, but patients may be unaware of
their deficit. Complex hallucinations are more frequently associated with temporal lobe epilepsy
but are reported by 10% of patients with occipital lobe epilepsy. Between 16-18% of patients
with temporal lobe epilepsy have visual hallucinations. However, hallucinations in temporal lobe
epilepsy are more likely to involve other senses than those of the occipital lobe. Visual
hallucinations associated with parietal lobe epilepsy are uncommon (Liu et al., 2010).

_Narcolepsy_

The prevalence of narcolepsy in the general population is 0.01-0.18% (Talih, 2011).
Narcolepsy usually affects those between ages 5-55, but it is most prevalent among those in their
20s (Liu et al, 2010). Narcolepsy may be overlooked and misdiagnosed for years before the
correct diagnosis is made (Cummings & Miller, 1987) and the appropriate treatment given
(Duwe & Turetsky, 2002; Talih, 2011). Patients with narcolepsy may view themselves as having
a psychiatric disorder prior to correct diagnosis. Patients have reported not realizing that their
hallucinations were related to a sleep disorder since they could occur during the day where
patients were unaware that they were momentarily going in and out of sleep states (Sacks, 2013;
Cummings & Miller, 1987).
Visual hallucinations occurring in narcolepsy are generally complex and often associated with auditory and tactile hallucinations. The duration of these visual hallucinations is variable, ranging from a few seconds to minutes. They often occur regularly: for some on a daily basis. Insight is usually, but not always intact. When insight is compromised, hallucinations may be misattributed to some other cause in order to make sense of them (Sacks, 2013). Patients may appear to have unusual, magical thinking, or delusional thinking as well (Ohayon, 2000; Sacks, 2013; Szucs, Jansky, Hollo, & Migleczi, 2003). Symptoms associated with narcolepsy include hypnogogic hallucinations, excessive daytime cataplexy, sleepiness, or sleep paralysis (Szucs et al., 2003; Talih, 2011).

There are tests available to identify narcolepsy, including measurement of hypocretin levels in cerebrospinal fluid, huford leukocyte antigen (HLA) typing, and mean sleep latency testing (MSLT). Treatment for narcolepsy typically includes wake promoting agents and central nervous system stimulants. Selective serotonin reuptake inhibitors and serotonin norepinephrine reuptake inhibitors may be used when cataplexy is a part of the clinical picture. However, antipsychotic drugs are often contraindicated in the treatment of narcolepsy (Talih, 2011, p.31).

**Vision Impairment and Visual Hallucinations**

Complex visual hallucinations with insight occur among those with acquired visual impairment. These hallucinations are not related to chronological age and the individuals may be cognitively intact (Menon, 2005) and psychologically normal (Teunisse, Zitman, Cruysberg, Hoefnagles, & Verbeek, 1996). Many of the patients with visual impairment associated with hallucinations have reported that they would not have told their physicians about having hallucinations, if not asked directly, for fear of being thought to have a psychiatric illness or dementia (Holroyd, Rabins, Finkelstein, Nicholson, Chase, & Wisniewski, 1992; Menon, 2005).
Training physicians to educate patients and ask in a manner that does not suggest psychiatric illness may be helpful (Holroyd et al., 1992). Patients found reassurance in learning that their visual hallucinations did not represent additional pathology (Menon, 2005).

**Macular Degeneration**

Estimated prevalence rate for visual hallucinations amongst those with age-related macular degeneration is 13%. Significant variables include bilateral vision loss, particularly with visual acuity of 20/60 or worse, living alone, history of stroke, and lower scores on cognitive testing (Holroyd et al., 1992).

**Retinal Disease**

Prevalence rate for visual hallucinations in patients with retinal disease is 15%. These hallucinations last from a few seconds to minutes (Scott, Schein, Feuer, & Fostien, 2001).

**Charles Bonnet Syndrome**

Charles Bonnet syndrome is characterized by complex release hallucinations in psychologically normal individuals. The release hallucination is a spontaneous visual response, which may be due to lack of inhibitory input such as seen with hallucinations by prisoners of war who experienced sensory deprivation. Charles Bonnet syndrome has been used as a catchall term for diagnosing those with release visual hallucinations who were considered psychologically normal. Most patients with diagnosed Charles Bonnet syndrome have incurred vision loss. Those with Charles Bonnet syndrome typically have macular degeneration, cataracts, diabetic retinopathy, glaucoma, or corneal disease (Liu et al., 2010). The hallucinations experienced may last from a few seconds to hours. Estimated prevalence rates among those who have visited an ophthalmologist is less than 0.5% (Shiraishi, Terao, Ibi, Nakamura, & Tawara, 2003). The estimated prevalence rate among those with low vision is between 12.8%-17.5% for complex
visual hallucinations and 50% for simple visual hallucinations (Reichert, Series, & Storkey, 2013).

**Conversion Disorder**

At this juncture the DSM-5 diagnostic decision tree for hallucinations asks the clinician to consider conversion disorder. However, there are no prevalence rates available for transient conversion disorder. The incidence for persistent conversion symptoms is estimated to be 2-5/100,000 or 0.00002% to 0.00005%. Dissociative symptoms are more common in the disorder. Although visual hallucinations may occur in conversion disorder, they are not listed as a primary symptom. Visual symptoms such as tunnel vision are listed as possible diagnostic features. Comorbid conditions common with conversion disorder include depressive disorders, anxiety disorders, somatic symptom disorder, personality disorders, neurologic disorders and other medical conditions. A history of trauma is also common with conversion disorder. Conversion disorder is more common in women. Ruling out neurological disease, somatic symptom disorder, factitious disorder, dissociative disorder, body dysmorphic disorder, and panic disorder is part of differentially diagnosing conversion disorder. Given the low incidence rate, other potential diagnoses should be considered first.

**Culturally Sanctioned Visual Hallucinations**

The next juncture asks the clinician to determine if a hallucination is culturally sanctioned. For example in the United States visual hallucinations as a part of a grief response are not considered pathological. Grief hallucinations occur across cultures. Visual hallucinations of “seeing” the deceased are usually brief. Prevalence rates for hallucinations related to grief response are estimated from 10% to 41%; Rates are higher among those who are widows or widowers after age 40. These rates are between 23% to 41% for college students. Prevalence rates of visual
hallucinations are higher in those with pathological conditions such as PTSD, Charles Bonnet syndrome, or reactive psychosis (Collerton, Mosimann, & Perry, 2015). Clinicians should weigh the degree to which a patient is having these hallucinations. Is it a brief vision of the loved one as a potential part of the psychological process of mourning, or is the person having long periods of imagining their loved one there with them, unaware that they are not actually with them? Just because the subject matter of the hallucination may be culturally sanctioned does not mean the etiology behind the hallucination is not pathological.

**Psychotic Disorders**

Visual hallucinations occur in psychiatric disorder, including schizophrenia spectrum disorders, bi-polar disorder with psychotic symptoms, depression with psychotic symptoms, and brief psychotic disorder. In psychiatric disorders, auditory hallucinations are more common than visual hallucinations, and visual hallucinations are generally, but not always accompanied by auditory or tactile hallucinations, although not necessarily simultaneously. The duration of hallucinations in psychiatric disorder is variable. The lifetime prevalence for all psychotic disorders is between estimated between 3- 3.5% (Perala et al., 2007).

**Affective Disorders with Psychotic Features**

The next two junctures of the decision tree ask the clinician to determine whether the symptoms occur in the context of a manic or depressed mood. Estimated prevalence rates for Bipolar I Disorder with psychotic features (which may or may not include visual hallucinations) is 0.24%. The estimated prevalence rate for Major Depressive Disorder with psychotic features is 0.35% (Perala et al., 2007). The estimated prevalence rate for visual hallucinations among patients diagnosed with affective disorders with psychotic features is 15% (Waters et al., 2014).
The junctures for affective psychotic disorders are listed in the decision tree before schizophrenia spectrum disorders. However, it is important to remember that prevalence rates indicate that visual hallucinations with these disorders are statistically less common than visual hallucinations with schizophrenia spectrum disorders.

**Schizophrenia Spectrum Disorders**

The next junctures asks the clinician to consider nonaffective/schizophrenia spectrum psychiatric disorders by assessing length of time psychotic symptoms have been present, and by assessing for delusions, disorganized speech, grossly disorganized or catatonic behavior, and negative symptoms of schizophrenia spectrum disorders.

**Schizophrenia.** Schizophrenia has a lifetime prevalence rate in the general population of 0.3% to 0.87%. Research has indicated some variance in rates by ethnicity and race (American Psychiatric Association, 2013; Perala et al., 2007). Between 16% to 72% of individuals diagnosed with schizophrenia have visual hallucinations: the higher percentages are from studies of individuals on inpatient units (Mueser, Bellack, & Brady, 1990) and often occur with auditory hallucinations. Comparatively, 74% of people with a diagnosis of schizophrenia experience auditory hallucinations (Sartorius, Shapiro, & Jablensky, 1974). International studies indicate that culture impacts hallucination prevalence rates: auditory hallucinations are universally more common among those diagnosed with schizophrenia across cultures with prevalence rates from 66% to 90.8%. Visual hallucinations were far less prevalent with rates between 3.9% to 53.9%. The highest prevalence rates for both types of hallucinations were found in Nigeria and Ghana (Bauer et al., 2011, p. 322).

Onset is typically in the late 20s for females and early to mid 20s for males. “Late onset cases can still meet diagnostic criteria for schizophrenia, but it is not yet clear whether this is the
same condition as schizophrenia diagnosed prior to mid-life” (American Psychiatric Association, 2013, p. 103). Males tend to have more negative symptoms than females. Impaired cognition is common even when other symptoms are not active. Neuropsychological research has indicated abnormalities associated with schizophrenia, but none diagnostic. No laboratory or psychometric tests conclusively identify schizophrenia (American Psychiatric Association, 2013). Medical comorbidity of patients with a diagnosis of schizophrenia should be considered. Studies have indicated that some patients' mental symptoms are caused or exacerbated by undiagnosed medical conditions (Jeste, Gladsjo, Lindamer, & Lacro, 1996).

**Schizoaffective.** Schizoaffective disorder has a lifetime prevalence rate of 0.3%. As with schizophrenia, hallucinations are more commonly auditory, but may be visual. The general course is two months of auditory hallucinations or delusions followed by a depressive period. It is important to remember that part of the diagnosis is a major mood episode of either bipolar or depressive type. Differentiating schizoaffective disorder from mood disorder with psychotic features can be challenging. The distinguishing criteria are major mood episode present the majority of the duration of active and residual symptoms, as well as two or more weeks of delusions or hallucinations when no major mood episode is present. Onset is generally in early adulthood but can occur anywhere from adolescence to late life. Females are diagnosed with schizoaffective disorder more frequently than males and have more depressive symptoms (American Psychiatric Association, 2013).

**Schizophreniform.** Incidence of schizophreniform disorder is low. In the United States it is “possibly five-fold less than schizophrenia” with an estimated prevalence rate of 0.07% (American Psychiatric Association, 2013, p. 98). Approximately one-third of those diagnosed with schizophreniform disorder will recover in 6 months. “The majority of the remaining two-
thirds will eventually receive a diagnosis of schizophrenia or schizoaffective disorder”
(American Psychiatric Association, 2013, p. 98). Information on prevalence rates of visual hallucinations related to schizophreniform disorder is lacking.

**Other Schizophrenia Spectrum and Other Psychotic Disorders**

At this juncture the clinician is asked to consider other schizophrenia spectrum and psychotic disorders. However, no other criteria are suggested. In this diagnosis the clinician is to document and specify the presenting psychotic symptom. The clinician is to also document why full criteria are not met. Unspecified schizophrenia spectrum and other psychotic disorders are vague as to diagnostic criteria and the DSM-5 states that this diagnosis is made when a clinician chooses “not to specify the reasons that schizophrenia spectrum or other psychotic disorder criteria are not met.” Prevalence rates for these diagnoses are unavailable. These diagnoses lack diagnostic clarity and their utility is limited, outside of documentation.

**Brief Psychotic Disorder**

At this juncture the clinician is asked to consider brief psychotic disorder if symptoms are present more than one day but less than one month. Brief psychotic disorder accounts for 9% of cases of first-onset psychosis. This disorder may occur at any age, but may be more common in the 30s and in patients with personality disorders or personality disorder traits.

Important considerations regarding possible psychiatric related visual hallucinations include:

1. Age of the patient.
2. Does age of onset of symptoms suggest schizophrenia?
3. Is there a history of auditory hallucinations?
4. Are there negative symptoms?
5. Are there delusions?

6. Is there disorganized speech?

7. Is there disorganized or catatonic behavior?

8. Does the patient have insight regarding the hallucinations?

9. If the patient does not have insight what is the response to having the event labeled a hallucination?

10. Is the onset of symptoms: acute or insidious?

11. What is the duration of the symptoms?

12. Do the symptoms occur within or outside of mood disorder?

13. Are the hallucinations hypnopompic or hypnogogic?

14. What occurred before onset of symptoms, any changes?

15. If the patient has a history of affective or nonaffective psychiatric disorder, are the symptoms different this presentation?

16. Substance/medication use and discontinuation history

17. Medical history of the patient

18. Psychiatric history of the patient

19. Patient’s family medical, neurologic, and psychiatric history

20. History of head injury

21. Trauma history

22. Educational history

23. Social and work history

24. Is the patient an accurate historian?

25. Is psychological or neuropsychological testing suggested?
Additional Psychiatric Considerations

Trauma related flashbacks should be distinguished from visual hallucinations. PTSD has high comorbidity with other psychiatric disorders. If visual hallucinations are present other medical, psychotic, and personality disorders should be considered (American Psychiatric Association, 2013).

Hallucinations Not Covered Above

At this juncture the DSM-5 decision tree asks the clinician to consider diagnoses not covered earlier in the diagnostic decision tree, without any guidance as to possible etiologies except to reconsider a schizophrenia disorder or psychotic disorder if there is clinically significant impairment or distress. Even though the visual hallucinations may be one symptom of psychosis they “are not pathognomonic of a primary psychiatric illness” (Teeple et al., 2009, p. 26), even if clinically significant and causing distress. Note that there are no guidelines as to what defines “clinically significant” or “distress.” The logic at this juncture is somewhat circular and is prone to attribution error given the lack of diagnostic sensitivity or specificity, and does not acknowledge that the etiology of the majority of psychiatric conditions is unknown (American Psychiatric Association, 2013). Also the utility of diagnoses at this juncture is limited. The decision tree does not allow for the consideration of referral to another medical specialist and does not allow for lack of knowledge of the etiology of the visual hallucination.

The alternate diagnosis or lack of diagnosis at this point is “nonpathological,” which according to the decision tree indicates that the visual hallucination does not cause significant clinical impairment or distress. This may or may not be true. For example, a patient may not have distress regarding visual hallucinations, but that patient may have a medical or psychiatric
Considerations at the final junctures include:

1. Review data for what could have been missed. Could the hallucination be related to unknown or unrecognized medical disorders, psychiatric disorders, or substance/medication use or withdrawal?
2. Are there accompanying neurologic symptoms?
3. Is a medical referral in order?
4. Is referral to another clinician in order?
5. Is the hallucination related to sleep disturbance?
6. What does the visual hallucination mean to the patient?
7. What is the function of the hallucination?
8. What is the context of the hallucination?
9. Inform and educate the patient regarding possible medical, substance/medication, metabolic, ophthalmologic, psychiatric, and neurologic etiologies.
10. Inform the patient that you do not know the cause.
11. Consider possible secondary gain.

**Conclusion**

Differential diagnosis related to the symptom of visual hallucinations can be challenging given the wide variety of underlying etiologies, potential health risks, and consequences associated with both the underlying etiology, as well as potential damage of misdiagnosis and incorrect treatment. Although visual hallucinations are generally perceived to be a symptom of psychiatric disorder, they are more commonly associated with neurological or medical disorders,
sensory impairment, or substance intoxication or withdrawal. In this paper I summarized the
most prevalent causes of visual hallucinations, reviewed the DSM-5 hallucination decision tree,
and provided an annotated visual hallucination differential diagnosis decision tree. It is in no way
inclusive of all the causes of visual hallucinations, underlying mechanisms, or treatment options.
The causes of visual hallucinations are too numerous, and the treatment options too varied to
cover in the scope of this paper. However, it is my hope that this paper may serve as a reminder
of those causes, which are both psychiatric and non-psychiatric so that trainees like myself may
be more aware and open to diagnoses outside of our training to help limit attribution error related
to psychiatric diagnosis. Also I hope it serves as a reminder to re-examine diagnosis when the
course of the symptoms is not as expected and response to treatment is not as expected.

Information related to visual hallucination prevalence is limited and unavailable for many
disorders. This may be in part due to the fact the many patients do not report visual
hallucinations out of fear of being seen as having psychiatric disorder. Also, data may be limited
by clinicians assuming that visual hallucinations are psychiatric or organic in nature depending
on the specialty and diagnostic frame of the clinician. The data that is available is from multiple
studies over many decades. During that time diagnostic criteria have been altered and additional
diagnoses have been discovered. For example, it is likely that some individuals studied as having
schizophrenia in older studies may now be recognized as having had a stroke or dementia with
Lewy bodies. It is important to remember that diagnoses change and that a diagnosis often is
used in a top to bottom approach, which may lead to attribution error. Keeping in mind
prevalence rates is one way to help check attribution error. However, it is likely that studies of
prevalence rates may also be influenced by attribution error and diagnostic error.
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Figure 1.0. DSM-5 Handbook of Differential Diagnosis Decision Tree for Hallucinations
2.6 Decision Tree for Hallucinations

- **Occurs only in the context of an episode of elevated, expansive, or irritable mood accompanied by increased energy**
  - Yes: **MANIC EPISODE WITH PSYCHOTIC FEATURES in BIPOLAR I DISORDER [3.3.1]**
  - No:
    - **Occurs only in the context of an episode of depressed mood or diminished interest or pleasure accompanied by characteristic depressive symptoms**
      - Yes: **MAJOR DEPRESSIVE EPISODE WITH PSYCHOTIC FEATURES in MAJOR DEPRESSIVE DISORDER [3.4.1], BIPOLAR I [3.3.1], or BIPOLAR II [3.3.2] DISORDER**
      - No:
        - **Hallucinations last for 1 month or more**
          - Yes: **History of Major Depressive or Manic Episodes**
            - Yes:
              - **During an uninterrupted period of illness, psychotic symptoms concurrent with mood episodes**
                - Yes: **SCHIZOPHRENIA [3.2.1] (plus comorbid BIPOLAR I [3.3.1], BIPOLAR II [3.3.2], or MAJOR DEPRESSIVE DISORDER [3.4.1]) DISORDER if history of Major Depressive or Manic Episodes**
                - No: **Duration at least 6 months**
                  - Yes: **SCHIZOPHRENIA [3.2.1] (plus comorbid BIPOLAR I [3.3.1], BIPOLAR II [3.3.2], or MAJOR DEPRESSIVE DISORDER [3.4.1]) DISORDER if history of Major Depressive or Manic Episodes**
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                    - Yes: **SCHIZOPHRENIA [3.2.1] (plus comorbid BIPOLAR I [3.3.1], BIPOLAR II [3.3.2], or MAJOR DEPRESSIVE DISORDER [3.4.1]) DISORDER if history of Major Depressive or Manic Episodes**
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                      - No: **Duration at least 6 months**
                        - Yes: **SCHIZOPHRENIA [3.2.1] (plus comorbid BIPOLAR I [3.3.1], BIPOLAR II [3.3.2], or MAJOR DEPRESSIVE DISORDER [3.4.1]) DISORDER if history of Major Depressive or Manic Episodes**
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                          - No: **Duration at least 6 months**
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                              - No: **Duration at least 6 months**
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                                  - Yes: **SCHIZOPHRENIA [3.2.1] (plus comorbid BIPOLAR I [3.3.1], BIPOLAR II [3.3.2], or MAJOR DEPRESSIVE DISORDER [3.4.1]) DISORDER if history of Major Depressive or Manic Episodes**
                                  - No: **Duration at least 6 months**
                                    - Yes: **SCHIZOPHRENIA [3.2.1] (plus comorbid BIPOLAR I [3.3.1], BIPOLAR II [3.3.2], or MAJOR DEPRESSIVE DISORDER [3.4.1]) DISORDER if history of Major Depressive or Manic Episodes**
                                    - No: **Duration at least 6 months**
                                      - Yes: **SCHIZOPHRENIA [3.2.1] (plus comorbid BIPOLAR I [3.3.1], BIPOLAR II [3.3.2], or MAJOR DEPRESSIVE DISORDER [3.4.1]) DISORDER if history of Major Depressive or Manic Episodes**
                                      - No: **Duration at least 6 months**
                                        - Yes: **SCHIZOPHRENIA [3.2.1] (plus comorbid BIPOLAR I [3.3.1], BIPOLAR II [3.3.2], or MAJOR DEPRESSIVE DISORDER [3.4.1]) DISORDER if history of Major Depressive or Manic Episodes**
                                        - No: **Duration at least 6 months**
                                          - Yes: **![](https://via.placeholder.com/150.png?text=SCHIZOPHRENIA%20[3.2.1]%20(plus%20comorbid%20BIPOLAR%20I%20[3.3.1],%20BIPOLAR%20II%20[3.3.2],%20or%20MAJOR%20DEPRESSIVE%20DISORDER%20[3.4.1])%20DISORDER%20if%20history%20of%20Major%20Depressive%20or%20Manic%20Episodes)" width="150" height="150""