EIGHTH JUDICIAL DISTRICT COURT
CLERK OF THE COURT
REGIONAL JUSTICE CENTER
200 LEWIS AVENUE, 3rd Fl.
LAS VEGAS, NEVADA 89155-1160
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Steven D. Grierson
Clerk of the Court

Brandi J. Wendel
Court Division Administrator

July 22, 2015

Tracie Lindeman
Clerk of the Court
201 South Carson Street, Suite 201
Carson City, Nevada 89701-4702

RE: STATE OF NEVADA vs. DIPAK K. DESAI
S.C. CASE: 64591
D.C. CASE: C265107-1

Dear Ms. Lindeman:

Pursuant to your Order Denying Motion and Directing District Court Clerk to Transmit Document, dated July 17, 2015, enclosed is a certified copy of the Independent Medical Evaluation dated April 14, 2013 in the above referenced case. If you have any questions regarding this matter, please do not hesitate to contact me at (702) 671-0512.

Sincerely,
STEVEN D. GRIERSON, CLERK OF THE COURT

Heather Ungermann, Deputy Clerk

15-22801

CONFIDENTIAL

David Palestrant MD
Neurocritical Care and Vascular Neurology
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Los Angeles, CA

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Independent Medical Evaluation

State of Nevada vs. Depak Kantilal Desai

Date of Report: 4/14/2013

Records reviewed:

- Nevada Imagining Center Radiologist reports, dated October 5, 2007; November 2, 2007; February 25, 2009; and July 2, 2010 (bate stamp 154-158; 194-195; 324-360; 383-386; 396-398; 406-407)
- UCLA medical records, dated July 13, 2008 through April 14, 2009 (bate stamp 152-153; 191-193; 196-197; 380-382 ;235-262; 269-271)
- Assessment of Neurocognitive Processing Evaluation, completed by Dr. Kinsora, dates of evaluation March 9, 2009; March 10, 2009; March 12, 2009, and July 13, 2009. (bate stamp 131-151; 170-175; 399-405; 301-309)
- UCLA Health System, Outpatient Speech Pathology Clinic, Aphasis Assessment reports, dated July 22, 2008; and April 13, 2009. (bate stamp 163-169; 392-395; 597-603)
- Nevada State Board of Medical Examiners, Order Scheduling Follow-up Status Conference; Requiring Additional Medical Examination, Testing and Therapy; Continual Requirements for Interim Reporting and Documentation of any Significant Changes in the Medical Condition of the Respondent as it Relates to the Preparation and Presentation of his Answer to the Charges and Complaint on File in These Administrative proceedings; and Maintaining Prohibition of the Practice of Medicine until Further Order, dated April 15, 2009 (bate stamp 227-232; 289-296)
- Summerlin Hospital medical records and CD's, dated June 1, 2009 and November 21, 2009 (bate stamp 387-388)
- Speech Therapy Center of Excellence, Speech and Language Evaluation, dated June 4, 2009 (bate stamp 389-391; 605-607)
- Intake documents and Psychiatric Evaluation completed by Dr. Roitman, dated June 12, 2009 (bate stamp 159-162; 198-224)
- Pre-Commitment Evals from Dr. Bradley, dated February 7, 2011 and Dr. Krelstein , dated February 6, 2011

15-22801

- Lakes Report dated September 20, 2011
- MRI Brain w/o Contrast completed by Dr. Mazzu dated February 24, 2013
- Summerlin Hospital, 2/24/13- through 3/1/13 (519; MRS and CTS on four CD's with reports)
- Cardiology Records/Reports from Dr. Watoo dated 10/16/13 and 11/15/12
- Desert Neurology Reports from Dr. Veerapan dated 3/27/12 and 9/25/12
- Nevada Imaging Center Reports and MRI (CD) from Dr. Orrison dated 10/16/12
- UCI Brain Imaging Center Reports (J. Wu, M.D.), 11/21/11 PET, MR, and Reports on one CD
- UCLA Aphasia Evaluation dated 11/20/12
- Thomas E. Bittker, MD, 11/1/12 and 12/5/12 Independent Neuropsychiatric

David Palestrant MD, Background:

I was asked to perform and independent medical evaluation of Dr. Dipak Kantilal Desia. I am currently a full time Neuro Intensivist and Vascular Neurologist practicing at Cedars Sinai Medical Center in Los Angeles where I am part of the Neurology and Neurosurgery teaching faculty. I currently hold board certifications in Neurology, Vascular Neurology, Neurocritical Care and Critical Care Medicine.

My opinions in regards Dr. Desai are based on my clinical experience as an active treating Vascular Neurologist and Neurointensivist. In addition, I am also the former director of the Stroke and Neurocritical care programs as Cedars Sinai Medical Center. I teach Neurology and Neurosurgery residents as well as Neurocritical care fellows, lecture nationally and I'm involved in clinical research trials. These professional capacities require that I have a current in-depth knowledge and understanding of stroke presentation and treatment, as well as functioning of the human brain. Please see attached CV.

The review of the medical records is for IME purpose only and do not imply a doctor patient relationship. My opinions expressed are based on the documents available to me at the time of this review. All of my opinions have been rendered with a reasonable degree of medical probability, and are subject to change should any new information regarding the case be forth coming.

Medical History:

Dr. Desai is a right-handed male, with a history of hypertension and hyperlipidemia, whose pertinent medical history goes back to 1987 when he suffered an acute myocardial infarction and a cardiac arrest. At that time he underwent coronary artery bypass grafting (CABG). All indications are that after the CABG he was stable from a cardiac perspective. Follow-up cardiac evaluations by Dr. Wattoo including

cardiac echo cardiograms (ECHO) demonstrated a diminished ejection fraction with anterior septal and apical hypokinesis consistent with the old myocardial infarction. At various times, transthoracic ECHOs have shown a possible thrombus in the left ventricle, but this was not clearly substantiated on trans esophageal ECHOs (TEE).

Acute stroke (9/29/07), Taiwan:
In September of 2007, shortly after boarding at plane in Taiwan, he developed sudden onset of left arm and leg weakness and slurred speech. He was admitted to a hospital in Taiwan. His initial CT scan demonstrated only an old left cerebellar stroke. A cardiac ECHO had mild left ventricular impairment with no clear thrombus, and hypokinesis of the anterior septal and apical areas. Carotid duplex studies were negative and a transcranial Doppler showed potential right middle cerebral artery narrowing. His initial left-sided weakness improved as did his slurred speech, and at time of discharge he still had weakness of the left hand and wrist. The cause of the stroke was felt to be a pure motor lacunar syndrome. He was discharged home on Aspirin and Clopidogrel.

Nevada Medical Imaging 10/5/07, 11/2/07, 2/5/08 :
On return to Nevada Dr. Desai underwent a series of repeat brain MRI, MRA and MRV scans. The first study was interpreted as having sub acute areas of ischemia involving the right posterior frontal and temporal lobes with small involvement of the right posterior frontal and parietal subcortical white matter. The magnetic resonance venogram (MRV) demonstrated a significant decrease in the size of the right transverse and sigmoid sinus with irregularity which was interpreted as being consistent with sinus occlusive disease (blood clots in one of the veins of the brain). Based on this, a diagnosis of venous sinus thrombosis and subsequent venous strokes was made.

On my review of the imaging studies, there are multiple small ischemic strokes in the locations described which appear to be embolic and not venous in origin. In addition, there is no evidence of venous sinus occlusion, but rather, the right sigmoid and transverse sinus is congenitally small which is a normal variant.

Follow-up MRI imaging on November 2, 2007 and February 5, 2008, show improvement in the areas of stroke and the small right sigmoid and transverse sinus.

UCLA admission for acute stroke July 13, 2008:
On July 13, 2008, while visiting family in Los Angeles, Dr. Desai became acutely confused, complained of right visual field loss, and was not following commands for 10-15 minutes. In the emergency room, according to the Neurologists evaluation, he was oriented times 4, language was fluent and he had intact repetition and was able to follow two-step but not three-step commands but was unable to name. He was unable to interpret a picture or read sentence and his overall NIH stroke scale score was 1, which is a very mild deficit.

An MRI showed marked restriction of diffusion within left mesial temporal, posterior mesial temporal regions, including the hippocampal head, body and tail and inferior left temporal lobe, as well as the amygdaloid nucleus, consistent with an acute stroke. The old right hemispheric and cerebellar strokes were also seen. A magnetic resonance angiogram (MRA) demonstrated a cutoff of the left posterior cerebral artery, and an irregular M2 branch of the left middle cerebral artery. An MRV demonstrated no signs of venous sinus thrombosis. A TEE on July 16/08 had decreased left ventricular systolic function, wall motion abnormalities consistent with multi-vessel coronary artery disease with extensive anteroseptal-apical infarction and excessive trabeculation in the apex, doubt but could not exclude thrombus. Aortic atherosclerosis was also noted. No definitive source for the stroke was found.

At time of discharge he continued to have a right upper quadrant visual cut, was having difficulty with naming and memory, remembering only 2/3 objects at 3-5 minutes with significant prompts. He expressed frustration and complained of depression during the admission and was evaluated by Psychiatry and started on Remeron. Plavix was switched to Aggrenox for secondary stroke prevention and he was started on a high dose statin (Lipitor) and discharged home.

UCLA Speech Pathology follow-up 7/22/08 and 4/13/09:

Summary of pertinent findings:
First visit was on July 22, 08. During that visit his wife assisted him with the intake form. They reported that since the stroke he was unable to practice medicine, complete complex organizational tasks, and remember names of objects or people. He was able to independently dress and feed himself.

Summary of findings: Mild to moderate receptive aphasia and moderate to severe expressive aphasia. Auditory comprehension yielded poor performance with complex yes/no questions and multi-step commands. The patient's comprehension improves with repetition and visual cues from environment... it was also noted that the patient demonstrated signs of possible auditory agnosia.... The patient would make educated guesses or deductive reasoning to provide an answer. These answers were 90% correct, but patient admitted less than 100% comprehension... Verbal expression yielded significant word finding deficits and utterances consisting of single word or 2 word phrases, which contain frequent semantic and phonemic mimic paraphrasias... The responses he provided where appropriate to topic but brief and output. Reading comprehension was suitable for simple sentences, but gave patient more difficulty as the sentences increased in complexity and length.... Written expression testing proved that patient could write his name and address with reasonable stability... The patient is very motivated to improve his ability to communicate, indicating he would be a successful candidate for speech/language therapy.

Follow-up April 13/09: At this visit, the patient independently completed his intake questionnaire and reported that he continued to have difficulty with memory for distant events and people, and difficulty with organization. He was not able to resume prior household responsibilities such as fixing meals, work, finances and food shopping due to deficits in executive functioning. He continued to experience episodes of anomia when interacting with his family throughout the day.

Summary of findings: The patient presents with mild expressive aphasia. Auditory comprehension presents grossly within normal limits. The patient performed well on all the subsets during the evaluation; however, it is unknown how the patient would present if given more complex auditory stimuli. No longer demonstrates signs of auditory agnosia. Verbal expression yields mild word finding deficits... the patient is primarily responsive during informal conversation. The responses he provides are appropriate to the topic, but brief in output. Reading comprehension is accurate for simple and complex sentences, as well a paragraph length material. The patient's written description of the cookie theft picture demonstrates one verbal paraphrasic error, and decreased grammatical content... the patient preformed within normal limits on all cognitive-linguistic subtests. One instance of difficulty comprehending a spoken direction was noted, which the patient needed clinician modeling to complete and decreased content in the story recall, as sentences were grammatically sparse. The recall of details was correct and actually improved after the 20 minute delay. Overall the patient had made significant improvement language function since previous evaluation. The patient is very motivated to improve his ability to communicate, indicating he would be a successful candidate for speech/language therapy.

Summerlin Hospital admission June1, 2009:

Dr. Desai was admitted to Summerlin hospital for inability to speak which lasted 1 hour and 15 minutes. He then returned to his baseline function. He was evaluated by Dr. Veerappan, a Neurologist, after the episode resolved. On his exam, Dr. Desai was oriented x 3, with slightly reduced high mental function. MRI on June, 1 2009, was of low quality but demonstrated a new right occipital hyperdensity involving the calcarine cortex on flair imaging, but less clearly defined on diffusion imaging, consistent with an acute or sub acute right occipital stroke. This stroke also has an embolic appearance. A follow-up MRI on 7/2/09, more clearly demonstrated this lesion, which appeared sub acute at that point. An ultrasound of the carotids on June 1, 2009 shows no significant stenosis. The MRA of the brain shows a small left vertebral artery, possibly smaller than on prior studies and bilateral middle cerebral artery narrowing greater on the left than the right. Dr. Desai was diagnosed with a TIA, and as he was doing well, he was discharged home with no change in his medication regimen which included Aggrenox, Ramipril, Coreg , Tramadol and Lipitor.

Assessment of Neurocognitive Processing Evaluation, was completed by Dr. Kinsora PhD. Dates of evaluation 3/9/09, 3/10/09, 3/12/09, 7/13/09:

Summary of pertinent findings:
From the initial intake: Dr. Desai was able to give a social and personal history. Pertinent subjective complaints noted; possible depression, emotionally labile, fatigued, not feeling hungry, handwriting smaller, right hemianopia better, decreased sense of smell, word findings in Hindi and English difficulties, wife indicates problems with comprehension, loss of topographical sense, difficulty staying focused, memory impaired- forgets conversations, difficulty picturing who someone is talking about, he forgets tasks sets out to do, forgets who familiar people are. Frustrated and tearful at times and has difficulty recognizing people, but improving.

Behavioral observations: emotionally labile and tearful, frequent self-derogatory comments, but otherwise cooperative, polite and accommodating.

Test results and summary: Indeterminate. Effected by lack of established measure of effort for patients with medial temporal/occipital and hippocampal damage. Need for additional info on the damage. Notes presence of depression.

Regards to malingering: Measure of effort were in severely impaired range. Normally would indicate sub-optimal effort, however no normative reference for his type of brain injury. Consistent between measures, except in one area, usually multiple inconsistencies if suboptimal effort.

Pattern of performance consistent with severity of injury: Unclear wants to further investigate brain scans.

Symptoms are compared with similar patients: not clear

Additional threats to validity of testing: depression

Initial Findings in a Nutshell: Performance on the tests was of indeterminate validity since some of the performance was so poor. This examiner needs to determine if the severity of the damage to medial temporal, hippocampal and anterior occipital regions are such that his performance is plausible. Thus, additional information is needed by the examiner. Severe depression is present that is confounding the clinical picture. He would have difficulty-assisting counsel currently, just based on his depression, if genuine. Treatment is recommended.

Follow up:
Dr. Kinsora notes he reviewed the imaging and testing and those findings are consistent with mesial temporal lobe damage. He notes significant problems with confrontational word finding, lexical and language- based memory processing, and some aspects of executive control. In addition, milder deficits, and presumed

relative declines in performance exist across multiple cognitive domains. He notes his performance is consistent with individuals who have suffered moderate medial temporal lobe damage. Patients with similar damage demonstrate problems with autobiographical memory. These individuals demonstrate deficits in the area of memory processing and in many cases language processing. A substantial subgroup also exhibits visual processing deficits. Formal evaluation of these deficits is next to impossible. Agrees with treatment of depression approach by Dr. Roitman.

Findings in a nutshell: Performance is likely valid and consistent with degree and location of brain damage. Deficits are widespread, but most pronounced in the areas of word finding, memory and executive control. Depression continues to be significant, but becoming manageable. He is likely competent based on NRS 178.400, but in the borderline range with regard to assisting counsel. He can be considered "impaired in his ability to assist counsel", but not clearly " unable to assist counsel".

Speech therapy Center of Excellence, Speech and language evaluation, speech center of excellence Los Vegas, 6/4/09:

Summary of pertinent findings: Moderate expressive aphasia with cognitive linguistic deficits. Speech characteristics include higher-level word finding difficulties, difficulties with formulating thoughts. Cognitive deficits include reduced immediate memory with reduced auditory retention. On exam, mainly expressive issues. Auditory, visual/ reading and speech language testing are good. Immediate memory poor (54%), recent memory 98%, temporal orientation 98%, recall of information 59%, auditory processing 98%.

Norton Roitman MD, psychiatrist 6/12/09, 6/1/7/09, 7/2/09:

To paraphrase the psychosocial history Dr. Roitman obtained: Dr. Desai has undergone stresses due to the loss of his role in the family, his perception of damaged reputation in the community and his inability to pursue his practice. Due to his new cognitive deficits he is unable to pursue his past interests, has trouble comprehending and his days are now empty. He tries remembering names and faces by making lists, and finds only repetition allows him to retain information. Comprehension is poor for both visual and written material, as is his distant and recent memory, and he cannot keep track of things he used to comprehend. He becomes demoralized and cannot stop thinking of his losses. Through hours of pray he has achieved substantial fluency. He finds it humiliating he cannot perform as a leader of his family and thinks of himself as a burden. He cannot apply himself to complex tasks, and when he reads he cannot comprehend what words mean in their groupings.

On his exam: He was well dressed and groomed... his ability to give chronological history was impaired... especially to events prior and subsequent to 2008 CVA. He was non specific in his answers to questions about the nature, duration, exacerbating and palliating factors that effected his various symptoms, and returns

to his regret, humiliation that others would find him" stupid" and loss of his cognitive powers...vocabulary and language skills were reasonable good.

Dr. Roitman diagnosed Dr. Desai with post stroke depression, adjustment disorder with depression due to life events and logical demoralization secondary to loss of cognitive function. Recommends an approach of first trying to help with cognitive function then treat depression, and to this end recommended first Ritalin then Namenda and more rehabilitation therapy.

June 17, 09 Dr. Roitman, follow-up: Stimulant showed modest augmentation for patients mental efforts... additional support for task oriented rehabilitative efforts was given as well as resources for auditory materials.

July 2, 09 Dr. Roitman, follow-up: Shows an extended attention as determined by his ability to read GI specialty articles... periods of depression considerably reduced. He values the Ritalin... he was advised to use association memory to recall names and faces. His periods of depression are considerably reduced likely correlate with increased function and therapeutic effects. Discussed his ability to reapply himself neuropsychological tests... return appointment in one month

No further follow-up notes seen

Competency evaluation, Shera D. Bradley PHD, licensed psychologist, Oct 25/ 2010 and Jan 31/ 2011:

Summary of pertinent findings:
Dr. Desai's memory deficits were exaggerated; based on the sum of the data obtained from her evaluation including records, including records, interview data and results of the TOMM. He scored lower than chance and significantly lower than normative data, indicative of malingering. In addition she reported pockets of personal memory loss.

Conclusion: In conclusion, though Dr. Desai has documented medical problems, it is this evaluator's opinion that he did not put forth his full effort during this evaluation. It would likely benefit the court for Dr. Desai to be observed at a forensic facility that can provide appropriate medical care and competency restoration treatment.

Michael S Krelstein MD, Psychiatry- General and Forensic, Competency to Stand Trial Evaluation, 2/6/2011:

Summary of pertinent findings:
Daily functional status: He frequently awakens throughout the night to use the toilet. In the morning, he showers, brushes his teeth and uses the restroom, without help. Soon thereafter he drinks a cup of tea and attends to his prayers... he is able to eat a meal prepared by his wife... watches TV at night. On his exam, well groomed,

no obvious motor problems, speech fluent, no dysarthria- had processing delays, anomia, paraphrasias, mood alexithymic, responses truncated. Surrounding circumstances of his charges –comported surprise, confusion and befuddlement of the basic elements.

MacArthur Competence Assessment Tool- Criminal Adjudication (MacCat-CA) Low score understanding (11% percentile), Reasoning (2.2% percentile), Appreciation (0.3% percentile). During the exam some evidence for dissimilation indicating malingering, on other hand some perseveration and provided non bizarre rational for the wrong answers. Neurobehavioral Cognitive Status examination (cognistat) test 5 major areas – Language, construction, memory, calculation and reasoning. LOC and orientation assessed independently. Scored in moderate to severe impaired in all areas. Montreal Cognitive Assessment (MoCa): scored poorly in all domains.

Summary- Not competent to stand trial
Conclusion... demented and procedurally incompetent man with objective neurologic findings to support cognitive deterioration... has not received aggressive neurocognitive rehab, neuro-cognitive enhancers and treatment for a secondary "post-stroke" depression. And there remains an element of dissimulation and/ or purposeful symptom embellishment, though such does not account for the bulk of his impairment... consider admission to Lake's Crossing for aggressive treatment and more comprehensive neuro-cognitive testing.

Lake's crossing admission date 3/24/11, Dates of evaluation 7/7/11 and 8/25/11

Summary of pertinent findings:

A. Steven J Zuchowski MD, Diplomat in Forensic Psychiatry

In summary- Dr. Desai is alleging both total amnesia for the events surrounding his allegations and a severe ongoing problem with cognitive functioning. It is unclear to what extent these problems are authentic but it is clear that his real deficits are not as severe as he claims. In spite of these alleged deficits, however, it is my opinion that Dr. Desai does have the present ability to understand the nature of the criminal charges against him, understand the nature and purpose of the court proceedings and aid and assist counsel in his defense...

Key observations during time at Lake crossing:
1) Selective past memory recall.
2) Navigated the environment- avoided certain patients, went for walks, watched TV, fell into the routine, interacted appropriately with peers and staff. Some conversations with forensic staff indicating he had some understanding of economics and politics and some medical knowledge. He also denied depression and did not want to take any mood stabilizing or cognitive enhancing medications.

Opinion regarding malingering:
Poor cooperation with testing....Normal day- day functioning, better than claimed deficits....No evidence of new stroke or progression of old stroke... Discrepancy between demonstrated abilities with individuals he knew as his evaluator's vs. others he did not know...Given lack of cooperation, exact scope of cognitive deficits cannot be known.

Opinion regarding amnesia:
Exact scope cannot be known, however some degree of authentic amnesia is possible. By Wilson criteria, amnesia itself insufficient to render incompetent.

Opinion regarding ongoing cognitive deficits:
Because of failure to cooperate fully, it's impossible to know exact extent of current cognitive deficits. Word finding difficulties have been consistent and likely authentic. Does not limit ability to stand trial. His working memory appears heavily embellished. Deficits to the extent he claims would not allow a person to function to the extent he did in the hospital milieu.

B) Sally Farmer PHD: multiple date of evaluation 3/24 - 8/25/11

Summary of findings: ...demonstrated ability to understand nature of criminal charges against him, nature and purpose of the courts proceedings, and to aid and assist his counsel in his defense at any time during the proceedings with a reasonable degree of rational understanding.

Pertinent observations:
...Dr. Desai quickly learned the rules and schedule in place at the facility. He has been able to find his room without difficulty, remembers when various events are scheduled to occur, and recognizes the faces of most staff members and peers... during formal evaluation sessions, he has provided accurate reposes to questions posed in the legal process class he attends. As noted earlier, he has held intelligent conversations with staff members about current events, demonstrating both the ability to recall information learned earlier in his life and the capacity to retain knowledge he gained from watching news programs on TV. Early in his hospitalization, he computed the distance forensic staff members walk while completing their rounds, and he has displayed some degree of retained medical knowledge with nursing personnel. On one occasion, he brought his own pen to the legal process class; he remembered that a test would be given during that period and did not like the pens provided by the instructor, so he brought his own... Dr. Desai has demonstrated his ability to engage in reciprocal, rational conversations and work cooperatively with others throughout his Lake's Crossing hospitalization; he has the capacity to do so with an attorney as well.

Testing:
... Mini mental status exam (MMSE) - done twice, score 13 first and 15 second in moderate cognitive impairment range. Patients in this range are unable to live

independently, have problems with basic activities... Inventory of Legal knowledge: score most similar to person instructed to malinger... TOMM: score 60%, score much lower than would be expected, suggest again attempt to show him self more impaired than he is. (Second person to find this)

C) Lindell Bradley MD 9/2/11

Again assessment is malingering, and exact extent of cognitive issues unknown

4) Tom Durante Clinical Social Worker 3/24/11

Pertinent finding: Able to give quite extensive personal history but blanks on entire professional history after he left New York after residency. Reports not recalling his medical specialty.

Desert Neurology, (Dr. Veerappan, MD)3/27/12 and 9/25/12 reports:

Summary of pertinent findings:
3/27/12: Follow-up for stroke. Medications, Aspirin, Lipitor, Ramipril and Vitamin C. Patient's exam: patient alert and oriented x3, speech, cognition, mood and affect are in normal limits. Diagnosis is aphasia, memory loss and hypertension.

9/25/12: Complaining of problems with memory. He often gets frustrated that he has poor recall and keeps repeating himself. Goes into a room not knowing what he went in for. Wants to pray but unable to find the light to perform the puja. Extremely forgetful of names. Afraid to get out house because of sever memory problems and fear and anxiety. Withdrawn and visibility depressed. Developed pain and increased weakness in the left hand and forearm. Physical exam: Once again alert and oriented x3, speech, cognition, mood and affect within normal limits. Short term memory loss. Left arm with pronator drift and decreased power. Diagnosis: Aphasia, memory loss hypertension, hemiplegia, hemiparesis and questionable new stroke with slight increase in the weakness of the left arm.

UCLA aphasia evaluation, Jennifer H Bullaro SLP, 11/20/12:
Summary of pertinent findings:
Impression: The patient demonstrates signs/symptoms consistent with diagnosis of aphasia. He is able to understand simple questions with reasonable accuracy. His auditory comprehension deteriorates with increased complexity. Spoken expression is halting and filled with paraphrasias and circumlocutions. The patient requires encouragement to attempt to communicate. Today, the patient was most successful during picture description tasks. The contextual information provided by the picture help both the patient and his listener.

National Outcome Measure (NOM) used and level:

Spoken language comprehension Level 2- consistent with maximal clues, able to follow simple directions, respond to simple yes/no questions in context, and respond to simple words or phrases related to personal needs

Spoken language: level 4 – The individual is successfully able to initiate communication using spoken language in simple, structured conversations in routine daily activities with familiar communication partners. The individual usually requires moderate cueing, but is able to demonstrate use of simple sentences [i.e. semantics, syntax and morphology] and rarely uses complex sentences/messages.

Reading: level 4 -The individual reads words and phrases related to routine daily activities, and words that are less familiar, longer, and more complex. The individual usually requires moderate cueing to read sentences of approximately 5-7 words. Prognosis for improved language function: Prognosis for improved language function through therapy is poor given the amount of time since the patient's neurological insult and his progress to date.

Thomas E. Bittker, MD 11/1/12 and 12/5/12, Independent Neuropsychiatric Assessment

Summary of pertinent findings 11/1/12: Deficits in intellectual performance, ability to retain and recall information, thought organization and adaptive capacities. Currently reliant on his wife from much of his executive function. Will interfere with ability to aid and assist counsel, largely coincident to his memory deficits and his inability to intergrade new information... Dr. Desai's mini mental status exam, in brief, confirms the findings of significant deficits as related in Dr. Kinsora's and Dr. Krelstein's reports... according to the patient's wife has been progressive deterioration in his functioning... Also notes depression not treated and further treatment trial warranted... no aggressive efforts at rehabilitation and recommends neurocognitive and speech therapy.

Summary of pertinent findings 12/5/12: The finding on the speech pathology center is consistent with the psychological testing of Dr. Thomas Kinsora, is consistent with competency evaluation of Michael S. Krelstein in his report filed February 6, 2011, the MRI study of the brain by Anthony Bruno, MD of 6/13/11, the positron emission topography study of 11/21/2007, and the reports of Dr. Joseph Wu 10/24/12. In addition, they confirm the findings in my own neuropsychiatric examination. On the basis of all of the above, I can state with reasonable degree of medical certainty that Dr. Desai's potential to recapture sufficient cognitive functioning to permit him to be competent to stand trial is remote. In addition, the UCLA study of November 20, 2012 indicates no improvement in Dr. Desai from their assessment at the time of his initial evaluations at UCLA. Consequently, it is unlikely that further rehabilitation interventions will show significant promise in restoring Dr. Desai' s mental capacity sufficiently to permit him to stand trial.

University of California Irvine MRI brain, PET and DTI imaging 10/24/12, Dr. Wu:

Dr. Desai shows significant abnormalities of cerebral head imaging from 11/21/11 which showed metabolic asymmetries with left frontal and temporal cortex lower than right. They are decreases in medial orbital frontal metabolism. They are metabolic asymmetries with right parietal cortex lower than the left. There is left-sided metabolic cross cerebellar diaschisis decrease.

He also showed significant abnormalities on MRI DTI, which showed significant decrease in fractional and anisotropy in corpus callosum... Both the PET and the MRI DTI findings are consistent with Dr. Bitters findings are impaired behavioral and cognitive function and make it more medically probable that Dr. Desai has significant impairment in higher order understanding and judgment.

Cardiology records (D Wattoo, MD) 10/16/12 and 11/15/12:

Summary of pertinent findings:
10/16/12: Patient for 20 years and a friend. 62 year old with atherosclerosis, with an anterior wall MI complicated by cardiac arrest and cardiogenic shock requiring CABG, EF has varied between 43-47%. Medications: ramipril, persantine 75mg, aspirin.
Chief complaint: Headache and dizziness in afternoons with SBP > 150. Increased Ramipril over the phone. On exam states he is headache free, feeling otherwise the same. Plan was to increase ramipril to 2.5mg bid, goal systolic blood pressure 120. ECHO to evaluate EF and LVH. Of note the patient was able to provide some of his own symptoms to the physician.

ECHO 10/23/12- Ejection Fraction 45-50%, with Left ventricular hypertrophy, left atrium mildly dilated, Right ventricle normal, decreased relaxation, apical echo density seen highly suspicious of clot. If clinically indicated consider TEE

11/15/12 Hand written report of TEE - EF 35%, apical severe hypokinesis no evidence of clot. Notes from TEE procedure, patient able to understand post op instructions

Hospitalization Summerlin Hospital Medical Center 2/24/13 to 3/1/13:

Dr. Desai was brought to the emergency room after being found in the kitchen confused with a dazed look. In the Emergency department, his NIH stroke scale was 10 (moderate deficit), with aphasia, left arm weakness, partial gaze palsy, and dysarthria. Per Dr. Veerappan, the treating Neurologist, he was following to visual cues and verbal commands. There was no expressive language component, and a mild left arm drift. Initial head CT was negative. MRI of the brain showed left hemispheric multifocal infarcts. The infarcts were patchy, small and appear embolic. The largest were the left premotor and the left parietal strokes. At the time of admission he was on Ramipril, Lipitor and Aggrenox.

Work up demonstrated bilateral MCA stenosis worse on the left than right on MRA. Carotids ultrasounds showed mild atherosclerosis. Transthoracic ECHO had no source for stroke. EEG was negative. Telemetry and an EKG all show sinus bradycardia. He was started on Depakoate for possible seizures and then changed to Lamictal due to intolerance of the Depakoate. Discharge medications: Agrennox, Ramipril, Atorvastatin, and Lamicatal.

As best at I can tell from the notes, he continued to have an expressive aphasia at time of discharge, but was ambulating.

Pertinent evaluations:
Physical Therapy (PT) note 2/27/13 Birt Patrick
Summary of pertinent findings:
Notes inconsistencies in patient presentation making accurate assessment very difficult. Notes not speaking but able to ambulate, sit in chair... upon verbal cues for specific tasks (finger to nose, toe tapping, standing hip adduction), PT was met with blank stares; verbal cues, tactile cues, and PT demonstration needed to be repeated with patient ultimately demonstrating an inability to complete tasks. It was noted however, that patient was able to easily remove eyeglasses and place on bed without difficulty, sit to stand with no external assist by PT or use of arms to push from armrest of chair; upon entry to room, patient had nasal cannula in for supplemental 02. PT mentioned this and patient was able to remove cannula without any difficulty, dysmetria or clues to do so.... Patient was also able to wipe arm in specific spot IV was leaking, brush teeth, rinse/spit without spilling any water or toothpaste... grossly symmetric strength... Plan is to continue to try identify a reliable pattern or consistent impairment so that this can be specifically addressed...

Speech therapy evaluations
In summary demonstrate sever expressive aphasia and moderate receptive language impairment.

Pertinent Imaging:

Note: Italics are my notes on the individual scans. All the imaging studies available were personally reviewed.

Nevada Imaging Center MRI brain with and without contrast 10/5/07

Conclusion: Findings of sub acute ischemia involving the right posterior frontal and temporal lobes with involvement of the right posterior frontal and parietal subcortical white matter. There is flow restriction on the diffusion-weighted images with mild contrast enhancement consistent with sub acute ischemia. No arterial abnormalities are noted on the MRI of the same day and the dynamic real time MRA

sequences demonstrate a significant decrease in the size of the right transverse and sigmoid sinuses with irregularity consistent with sinus occlusive disease. These findings are most consistent with a venous infarction

Nevada Imaging Centers MRA brain 10/5/07
1) No arterial abnormalities
2) Findings of right transverse and sigmoid sinus occlusive disease. There is irregularity and narrowing of the right transverse and sigmoid sinuses without evidence of complete occlusion. Correlation with the MRI of the brain on the same day demonstrates findings most consistent with sub acute venous infarctions on the right.

MRA neck 10/5/07
MRA of the neck within normal limits

Sub acute ischemia with small strokes involving the right posterior frontal and right temporal parietal areas and centrum semiovale. There is good contrast filling of the right transverse and sigmoid sinus. MRA shows a small left vertebral artery and bilateral MCA narrowing left greater than right. MRA neck negative.

Nevada Imaging Center MRA and MRV head 11/2/07
Improvement in the appearance of the right transverse sinus compared to the previous MRI examination with findings again identified, consistent with venous sinus occlusive disease on the right without evidence of obstruction

Again no venous occlusion disease seen, right transverse sinus smaller than left

Nevada Imaging Center MRI brain 11/2/07
1. Minimal change in appearance of the MRI examination when compared to the previous examination 10/5/07.
2. Persistent abnormal increased signal intensity is noted on the flair and T2 weighted sequences involving predominantly the right posterior frontal and right posterior temporal lobes. There is improvement in the flow restriction identified on the diffusion weighted sequences consistent with resolution in the flow restriction identifies on diffusion weighted sequences consistent with resolution of the areas of acute ischemia and residual sub acute ischemia within the right centrum semiovale.

Small right temporal and parietal and small right centrum semi-ovale strokes, not acute, small cortical right frontal strokes, appear embolic. No sign of venous sinus thrombosis

Nevada Imaging Center MRI, MRA and MRV brain 2/5/08
Conclusion:

Improvement when compared to the previous MRI examination 11/2/07 with findings consistent with resolution of the prior acute ischemia and minimal residual evidence of prior cerebral ischemia involving the right posterior temporal lobes. Correlation with the MR venogram performed on the same day demonstrates continues improvement in the appearance of the right transverse sinus with no evidence of transverse sinus occlusion the current study

MRA and MRV 2/5/08
Continued improvement in the appearance of the MRV with no evidence of venous occlusive disease on the current examination

MRA 2/5/08 – left MCA smaller caliber, otherwise unremarkable
MRI 2/5/08 – right sided strokes less apparent and resolving
MRV 2/5/08- no change right smaller transverse sinus

UCLA MRI brain with and without contrast and with perfusion 7/13/08

Marked restriction of diffusion within left mesial temporal, posterior mesial temporal regions, including the hippocampal head, body and tail and inferior left temporal lobe, as well as the amygdaloid nucleus. Perfusion abnormality involving the left posterior cerebral artery and encompassing the area and diffusion abnormality as well as additional parenchyma of the parasagittal left occipital lobe, left calcarine region and left hippocampal tail and fornix. Gradient echo demonstrates intraluminal thrombosis within the left posterior cerebral artery
MRA neck negative
MRA head ... cutoff 4.5mm distal to the origin of the left posterior cerebral artery....
Irregularity of the M2 branch of the left MCA.
MRV unremarkable

UCLA
MRI brain: 7/13/08 acute left mesial temp stroke, positive DWI. Old right hemispheric strokes. Old left cerebellar stroke. No right sided hippocampal lesions
7/13/08 MRV- small right transverse sinus
7/13/08 MRA mild left MRA and right MCA stenosis not different from 2013, narrowing of left vertebral artery but joins basilar, cut off of left PCA
7/13/08 MRA neck with no significant stenosis

Nevada Imaging Center MRI brain with and without contrast – 2/25/09

Impression
1) Minimal residual abnormal signal intensity identifies involving the right frontal, posterior frontal, and temporal regions related to prior venous infarction.
2) Persistent findings consistent with old inferior left cerebellar infarction
3) New focal areas of abnormal signal intensity correlating to changes on the MRI and CT studies from July 2008 indicative of left posterior cerebral artery

distribution infarction with encephalomalacia involving the medial temporal lone on the left and the anterior occipital and posterior thalamus

Resolving right sided lesions, new small left mesial temporal hyperdensity on flair, extending from hippocampus back, amygdala largely preserved, does not extend to cortical surface or insula. Minimal thalamic involvement

Summerlin Hospital 6/1/09
CT head: Encenphalomalacia in the left PCA distribution, left cerebellar hemisphere, right parietal lone likely related to prior ischemia.

MRI brain: No evidence for acute or subacute infarct. Multiple areas of previous infarct... No acute findings.

MRA head:
1. No intracranial evidence of stenosis
2. Diminutive left anterior cerebral artery. Otherwise, normal MRA.

Carotid Ultrasound—no significant stenosis

6/1/09 – small left vertebral artery – smaller than prior, bilateral MCA narrowing left greater than right, increased compared to 7/08.
6/1/09 MRI – new right occipital hyperdensity seen on flair, not clear on DWI but poor image quality, left mesial temp hyperdensity, improved, old right hemispheric strokes.

MRI brain with and without contrast 7/2/10
1) Findings consistent with multiple areas of resolving cerebral infarction
2) ... questionable extension of right medial occipital infarction

Right right sub acute occipital stroke, larger than month prior, left mesial temp stroke improved.

Nevada Medical Imaging Center MRI brain 10/15/12
1) Findings remain consistent with multiple old areas of cerebral infarction
2) Persistent abnormal signal intensity consistent with encephalomalacia, microcystic encephalomalacia and/or gliosis involving the right frontal, temporal, and occipital regions consistent with the clinical history of prior venous infarction
3) Persistent abnormal signal intensity consistent with encephalomalacia, microcystic encephalomalacia and or gliosis involving the left medial temporal, anterior occipital, and posterior thalamic areas consistent with old cerebral infarctions

4) Persistent findings consistent with old inferior left cerebellar infarction

10/15/12 MRI brain- Characteristic finding of old strokes involving areas of the right hemisphere including the occipital lobe and left mesial temporal lobe. No new lesions seen.

University of California Irvine MRI brain, PET and DTI imaging 10/24/11, Dr. Wu

Dr. Desai shows significant abnormalities of cerebral head imaging from 11/21/11 which showed metabolic asymmetries with left frontal and temporal cortex lower than right. They are decreases in medial orbital frontal metabolism. They are metabolic asymmetries with right parietal cortex lower than the left. There is left-sided metabolic cross cerebellar diaschisis decrease.

He also showed significant abnormalities on MRI DTI which showed significant decrease in fractional and anisotropy in corpus callosum... Both the PET and the MRI DTI findings are consistent with Dr. Bitters findings are impaired behavioral and cognitive function and make it more medically probable that Dr. Desai has significant impairment in higher order understanding and judgment.

MRI 11/21/11- Old left mesial temporal stroke, old right small occipital stroke, old right hemispheric strokes, right hippocampus spared
Dr. Wu reports from the PET scan and MRI DTI images essentially demonstrate low blood flow in the areas of known damage to his brain and decreased function of the circuits associated with these lesions. This indicates that he has brain damage, but does not quantify the degree or extent. In my opinion, it does not provide much more information over and above what his other imaging studies have already told us. What is interesting to note, though not mentioned in the report, is the extent to which his blood flow is actually normal in large areas of the brain.

CT head 2/26/13
1. Sub acute small non-hemorrhagic left posterior frontal-parietal cortical infarct.
2. Old areas of infarction involving left temporal lobe, right occipital lobe, right frontal lobe, and, left cerebellum

MRI brain without contrast 2/24/13
1) Multifocal signal abnormalities identified within the left cerebral hemispheres with areas of abnormal cortical signal in the left frontal, parietal, occipital and temporal lobes all with corresponding restricted diffusion. These have the appearance of small acute multifocal infarcts. These correspond to MCA territory distribution as well as watershed infarcts.
2) Encephalomalacia consistent with remote infarcts in the right frontal, occipital, left temporal and cerebellar hemispheres

MRA head 2/25/13

Extensive intracranial atherosclerotic changes especially involving the bilateral middle cerebral artery territories left worse than right

Carotid duplex 2/25/13
Mild atherosclerotic disease without severe stenosis

2/24/13 MRI multiple small cortical and subcortical strokes in left the MCA distribution. Left post parietal/ occipital and left premotor being the largest. Residual left mesial temporal encephalomalacia, continued right mesial occipital encephalomalacia. Other strokes unchanged in right hemisphere

2/25/13 MRA moderate diffuse left m1 narrowing, left vertebral artery ends in PICA, hypoplastic left A1

Carotid US 2/25/13
Mild atherosclerotic disease without severe stenosis

Discussion

Dr. Desai has suffered a serious of ischemic strokes,
1) Time unknown, small left cerebellar stroke.
2) 2007, small strokes involving the right hemisphere.
3) 2008, a moderates size stroke involving the left mesial temporal lobe and its structures, and a small portion of the thalamus.
4) 2009, a right small occipital stroke.
5) 2013, multiple small left hemispheric stroke involving the frontal, parietal, occipital and temporal regions.

Briefly, his first stoke involving the cerebellum was asymptomatic. From the 2007 stroke the residual symptoms were some mild residual left sided weakness. After the third 2008 stroke, Dr. Desai reported that he suffered from profound retrograde and anterograde amnesia, had trouble with comprehension and speech production, inability to follow and complete complex tasks, some visual changes, depression, and progressive worsening of cognition. The 2009 stroke appears to have been asymptomatic. His most recent stroke has reportedly caused a worsened aphasia with a further decrease in speech output and comprehension, but with no new significant motoric effects.

The human brain is highly complex, functioning through intricate networks of nerves and their respective fibers. Through an understanding of many of these networks and their anatomic locations, Neurologists and Neurosurgeons are able to diagnose patients' symptoms, localize the symptoms origins in the brain and predict deficits when brain damage occurs. Some of these networks are extremely localized others much more diffusely distributed.

Language function:
Language function, in the majority of right-handed individuals, is located in the left side of the brain. Aphasia is the term used to describe a neurologic disturbance of speech, and encompasses both the ability to produce and understand speech. The left hemisphere can then be further subdivided, with different regions being more important for speech expression, comprehension, reading, writing and math. The ability to name objects and find words is distributed throughout the left hemisphere language areas. Dr. Desai suffered a stroke in the left hemisphere effecting the left mesial temporal lobe, and part of the thalamus in 2008. Though this location is not considered a primary speech center, it does have some speech function carrying fibers involved in speech, and involvement in integrating different visual, auditory and speech inputs. This explains Dr. Desai's word finding difficulties, and some of his problems with comprehension, difficulty with language processing and contextual confusion. His most recent strokes are small but again involved the left hemisphere, this time the parietal and frontal areas predominantly.

Memory function:
As mentioned Dr. Desai did suffer a stroke to the medial temporal lobe. The medial temporal lobe contains structures deeply involved in memory such as the amygdala and hippocampus, which are part of two crucial memory circuits, the Papez circuit and the ventral amygdalofugal pathway. These structures and circuits are found in duplicate on both the left and right side of the brain. They serve to regulate what information to store and not to store, but do not store the actual memories themselves. Therefore these circuits are more involved in the creation of new memories. Old and processed memories are stored diffusely through the brain often close to the location in the brain that is responsible for the particular function the memory involves. For exam, visual memories are stored close to the visual cortex(1,2).

Memory can be divided into distinct forms. Implicit memories occur without specific knowledge or recollection of how they were learned and include diverse mental capacities such as classic conditioning, motor skills and mental procedures. Explicit memory is the conscious recall of events, experiences and information and can be further subdivided, into episodic memory and semantic memory. Episodic memory is the recall of personal experiences. Semantic memory is the body of facts, principles and rules that makeup our general world of knowledge, which is consciously recalled without reference to a personal experience. Often in life these memories run concurrently. Damage to the Papez circuit more heavily damages episodic memory(1,2).

Though initially believed that both hippocampal formations functioned identically, it's now recognized that each has some unique function. The left likely has more involvement in terms of memory involving speech and visual spatial information. The right side is more involved with of non-verbal patterned material, such as geometric or tonal patterns. Damage to bilateral medial temporal lobes cause profound deficits in explicit memory. These patients have essentially total

anterograde memory loss and extensive retrograde amnesia as well, extending over several years. Very rarely strokes involving the left PCA territory can have memory impairments as profound as bilateral lesions initially but then improve over up to one year. Some patients can be left with deficits involving verbal memory but studies on patients with left PCA distribution strokes found only mildly to moderately impaired long-term verbal memory (2-4).

Amnestic syndromes are neurologic syndromes due to a process that damages the medial temporal lobes, especially the amygdala and hippocampus and portions of the thalamus, or the connections to these structures.

Several features characterize the amnestic syndrome:

1) Relative preservation of attention, concentration, visual spatial skills, language, motivation, complex perceptual abilities, and general intellectual abilities.
2) Preserved abilities to retain information for short periods, so called immediate memory.
3) Impaired registration of new information, so called anterograde amnesia. This deficit in learning does not extend to all types of material for example; skill based learning such as how to use tools is generally unaffected.
4) Variable deficits in recall of memory within certain interval before the onset of the amnestic state, so called retrograde amnesia. Amnestic syndromes represent problems with consolidation; the process whereby information is held in a temporary, transient form is converted into long-term storage. Amnestic patients maintain the ability to place information into temporary stores, and therefore have immediate recall, but have difficulty and consolidating that information. This difficulty with consolidation extends back no more than 1-2 years(1).

Stroke recovery:
All strokes, no matter what the size, if not fatal demonstrate some degree of improvement over time, often profound improvement. This improvement is contingent on the location of the stroke in the brain, the eloquence of the area involved, the size of the stroke (more recovery with smaller strokes), premorbid functioning, the patient's age, the ability for other areas of the brain to take over, rehabilitation and the plasticity of the brain.

As to the legitimate physical and psychological manifestations of Dr Desai's stroke Prior to the 2013 stroke:

Dr. Desai's claimed degree of neurologic dysfunction and neuropsychiatric testing performances between 2009-2013, are far worse than would be expected and not corroborated by the extent and anatomic distributions of his strokes. In my opinion there has been a significant amount of embellishment of his symptoms and incomplete efforts on neuropsychiatric testing. What the exact actual underlying extent of his neurologic deficits were at this time is almost impossible to tell, given the extent of the embellishment. However, based on the locations of the strokes,

testing done with what appears to be better efforts such at the Speech and Language evaluation in April 2009, and observations of his behavior when not being formally tested his baseline deficits likely included; some trouble with verbal expression and word finding, some mild difficulty with association tasks like contextualizing, integrating and processing speech inputs and following complex commands, and possibly some visual spatial difficulties as well. Ability to read, including complex paragraphs and write, was intact. At worst he would have had partial verbal memory disturbances more involved with forming new long-term memories and some degree of retrograde amnesia for events up to 2 years before the 2008 stroke, but beyond this he should have been able to recall most past events with not much disturbance. Logical thinking would remain intact as well as many higher order executive functions.

Evidence for embellishment of symptoms:

1) Veracity of the Neuropsychiatric testing:

Dr. Desai has undergone extensive neuropsychiatric testing performed by multiple practitioners. All these studies require active patient participation, without which, the results become dubious. Some of the examiners have noted that his results have at times been consistent with malingering and random guessing but at other times slightly better than random guessing. At times there was a lack of consistency in results of the same tests between examiners with no new lesions in the brain to explain the difference.

In addition, his scores were inordinately low, with a significant disconnect between his daily functional status and the degree of impairment registered on the testing. With Cognistat and MoCA testing, which assess intellectual functioning in multiple cognitive domains, he was impaired in almost all categories. Strokes affect defined vascular territories in the brain and are expected to have associated discrete deficits and not impair all cognitive spheres unless the strokes are far more wide spread than was the case in Dr. Desai.

2) Worsening performance on testing over time:

Testing done in the initial months after the stroke show better results than later, with no new brain lesions that would explain this deterioration.

As mentioned all strokes, no matter what the size demonstrate some degree of improvement over time. Based on the patient's history, it seems that at times he symptoms actually worsened. Indeed Speech and language testing done 9 months after the 2008 stroke at UCLA demonstrated significant improvement compared to the testing done in the immediate month following the stroke. From this point on, further gains would have been expected for the next 3 -9 months. However speech testing performed later at UCLA in 2012, showed actual regression though none of the MRI scans have lesions to explain this regression.

The only new brain lesion seen during imaging at this time was the right occipital stroke in 2009, which would not have caused any deterioration in speech function. Expected deficits from this stroke would have been a partial left visual field cut, more likely in the central visual fields, with no effect on language or memory. Indeed, in the time period when this new stroke occurred examiners, such as Dr. Roitman, reported that his symptoms actually improved with recommended therapy. This stroke also predates his admission to Kings Crossing were much of the discordance was observed between his claimed disability and actual functional status.

3) Discordance between functional status and testing

On some testing, such as a Mini-Mental status exam, he scored in the range of 13-16, indicating a moderate dementia. A person in this score range would most likely require 24-hour supervision. However, as documented by many of the examiners, he was able to take care of many of his activities of daily living, such as like dressing himself, toileting, feeding himself, following a daily routine and navigating around his familiar environments.

As demonstrated by his time at Lake's Crossing, he was able to successfully and quickly learn to independently navigate a new environment, set up new routines and engage and disengage appropriately individuals in his environment. A person scoring as low as he was on the neuropsychiatric assessments would not have been able to do this.

4) Discordance in memory loss expected from the stroke and claimed memory loss.

As discussed some degree of memory disturbance is possible from a mesial temporal lesion, but his memory issues he professed were far more severe than expected based on his brain damage, and reports on similar patients.

Most striking was his selective amnesia for his professional life and his legal issues. Where as for many of his examiners he gave a history on multiple personal events and current symptoms, he claimed to have very vague recall about his entire professional career, the people he worked with and at times claimed to have no recollection of his professional life from the time he left New York. He even claimed not to remember his medical specialty. His professional career would have been a major aspect of his life story and one that would be deeply embedded in his memory. At times however he did appear to slip up and indicate memory related to his career, for example, for Dr. Roitman he expressed a loss of standing in the community, and reported he was reading Gastroenterology journals. He also showed some medical understanding to the nurses at Lake's Crossing.

As mentioned, his retrograde amnesia at worst would be expected to extent out only 2 years from the stroke, and if present would be apparent for all explicit memory.

It is possible when asked questions about old memories he would have some difficulty integrating the answers with his verbal output or understanding the question or have mild confusion surrounding events or retrieving some aspects of the memory. But the complete amnesia he reported for large specific parts of his personal history is not possible as no lesion or disease of the brain could account for this very selective loss of memory.

If anything, anterograde amnesia should have been more pronounced than retrograde amnesia. Even here there are inconsistencies. During testing he was unable to complete simple short-term memory tasks. As discussed patients with severe amnestic syndromes can remember things in the short term but not consolidate them for long-term memory. Nor was his ability to form new long terms memories fully impaired. He was clearly able to form new memories in non-testing environments. He rapidly fell into routine at King's Crossing, he learned who to avoid, arrived in time for meals and group and found and navigated walking routes.

All indicators are that Dr. Desai's amnestic syndrome was far less significant than claimed. As profound a retrograde amnestic syndrome that Dr. Desai claimed, would require a severe dementia to be present with diffuse brain injury to many other neurological faculties and would render him essentially dependent for all activities of daily living.

6) Inconsistencies in reported and observed functional status:

Formal neuropsychiatric testing and physician evaluation take place within a discreet time period. For a person embellishing symptoms, a limited time frame works in their favor in terms of presenting a plausible consistent exam. The institutionalization at Lake's Crossing hospital provided for long-term observation, in a new environment and the ability to observe for inconsistencies, and should therefore be viewed as the most credible exam.

Without reiterating the Lake's Crossing reports, there were marked discrepancies between his daily functional ability, and his interactions when not being formally evaluated and his performance on formal evaluations. He very successfully learned to quickly navigate a new environment, fell into the daily routine, initiated his own new exercise routine, avoided more menacing patients, and placed himself in safe situations when feeling threatened. When talking to staff members he felt were not observing him, he apparently had very high-level sophisticated conversation. From his formal testing, he should have had problems with all most all of these activities.

In some respects his ability to embellish his symptoms for secondary gain speaks to very high-level executive functioning and planning. The observed embellishment demonstrated an ability to realize secondary gain and conceive and maintain a plan over time. All things a person with severe memory and cognitive impairment could not do.

7) Are there other potential causes for deterioration in function?

There was certainly an element of depression in Dr. Desai, and the depression itself could account for a portion of the cognitive issues, a so-called pseudo dementia. When under the treatment of Dr. Roitman, he did seem to have response to treatment, but its unclear if this treatment continued. Unfortunately he did not demonstrate consistency in the treatment for his depression.

How much the depression contributed to his poor testing performance and symptoms is unclear. One could relate some of the non-stroke associated deficits to depression, if it were not for the significance evidence of malingering.

A dementing process other than stroke should also be considered to explain his symptoms, however again the above inconsistencies in his testing and history call this into question. Additionally, PET imaging done in 2012 does not show a pattern consistent with usual dementing diseases such as Alzheimer's. Lastly, global dementing processes such as Alzheimer's effects recent memory first followed by older memories.

Neurologic symptoms after February 2013 stroke:

Dr. Desai presented on February 24, 2013 with what looks like a shower of small embolic strokes in the left middle cerebral artery distribution. The biggest of these strokes involve the left parietal area and the area near the supplementary motor cortex. According to the medical records his motoric function is good but he is having significant word finding difficulty and also difficulty with comprehension (receptive aphasia). Once again members of his treatment team have noticed inconsistencies between his observed functional ability and his performance during formal examination. This was apparent in the exams performed by the physical therapist at Summerlin Hospital.

Based on the images performed and his pre-existing medial temporal lobe damage, his current described aphasia is possible with the new lesions seen on MRI. Having not currently examined the patient my opinion is limited as to what the exact extent his current cognitive and speech problems are real versus embellished. These new strokes are however small in size and involve limited segments of the speech areas in the brain, and like with all strokes, the expectation is that the symptoms will improvement over time. Memory and executive function should not be affected by these new strokes. Whether he will get back to his real, non-embellished premorbid functional status is unclear at this point, but I do expect him to make significant gains. There is an excellent chance based on the size of the strokes that he could have near complete recovery from these strokes and get back very close to where he was prior to February 2013. Most recovery from stroke occurs in the first 3-9 months but can continue for up to 18 months out.

Given his prior history of embellishment, I would be cautious about claims as to the degree of his aphasia in the future. A claim of little improvement should be viewed with suspicion and carry a very high burden of proof.

Cause of the Strokes:

Dr. Desai has suffered a series of ischemic stroke (strokes due to lack of blood flow to the brain), which appear embolic in nature. Embolic strokes are strokes due to blood clots that either form in the heart or from atherosclerotic plaques in proximal blood vessels, that then migrate forward, blocking distal blood vessels. One can tell that these are embolic strokes, by the fact that the strokes are mainly cortical, some wedge shaped, involve multiple vascular distribution and in the 2008 stroke an actual blood vessel occlusion was seen. The emboli originated either in the heart or aortic arch. At various times, a potential thrombus was identified on cardiac transthoracic ECHO, however, this was never clear cut, and follow-up TEEs did not show a thrombus, except for the TEE at UCLA in 2008, that had questionable signs of a thrombus, but the cardiologist reading this study felt it was doubtful. From my review of the records I did not see any long term heart rhythm monitoring, such as a Holter monitor, looking for paroxysmal atrial fibrillation, an arrhythmia that strongly predisposes to stroke, occurs often in patients with pre existing heart disease, and that may not be found on short term cardiac monitoring.

His MRA studies of the brain also suggest narrowing of the intracranial blood vessels, so called intracranial atherosclerosis. This represents another possible cause for his strokes and is associated with a high reoccurrence rate even in appropriately treated patients. Going against intracranial stenosis as the etiology of his strokes is the fact that the degree of narrowing is not severe and not clearly progressive over this 5 year period on follow-up MRAs.

Having a succession of strokes over a 5-year period raises the question of medication non-compliance but also an incorrect therapeutic strategy. Even with appropriately secondary preventive treatment, stroke reoccurrence is possible. From the record review, Dr. Desai did not always follow through on rehabilitation recommendations or treatment for depression, but there was no concerns raised about non-compliance with other medication. I would surmise that he is either developing intermittent clots in his heart or has undiagnosed paroxysmal atrial fibrillation, and would likely benefit from more intense anti-coagulation such as with Coumadin; a preventive strategy that has not yet been tried on him. It may be worthwhile for him to be under the care of a Vascular Neurologist.

In Summary:

Dr. Desai has suffered a series of ischemic strokes since 2007, the most significant in 2008, effected his mesial temporal lobe which left him with expressive speech difficulties, and mild difficulties with comprehension, integration and contextualizing of speech. He had some degree of anterograde amnesia and mild retrograde amnesia. The extent of his cognitive deficits including his claimed profound selective memory loss, is unexplained by his strokes, and is more likely related to malingering and to a lesser extent depression. His new strokes in February 2013 involve the speech cortex, with a resultant expressive and receptive aphasia. Again questions of some degree of embellishment of the symptoms have been raised. Memory should not be further compromised by the new strokes. However these strokes are small and it's my expectation that he will make significant gains and return close to his level of function prior to February 2013. Most of his gains in neurologic function will be seen in the first 9 months, but full recovery can take up to 18 months.

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1. Brazis PW, Masdeu JC, Biller J. Localization in Clinical Neurology. LWW; 2012.

2. Godefroy O. The Behavioral and Cognitive Neurology of Stroke. Cambridge University Press; 2013.

3. Szabo K, Förster A, Jäger T, Kern R, Griebe M, Hennerici MG, et al. Hippocampal lesion patterns in acute posterior cerebral artery stroke: clinical and MRI findings. Stroke. 2009 Jun;40(6):2042–5.

4. Dobbins IG, Kroll N, Tulving E, Knight RT. Unilateral medial temporal lobe memory impairment: type deficit, function deficit, or both? Neuropsychologia. 1998.

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Education:

1996	MD, University of Arizona College of Medicine, Tucson, AZ
1992	BA Political Science; BS Molecular and Cellular Biology, University of Arizona, Tucson, AZ

Postdoctoral Training:

2001-2003	Fellow, Critical Care, Neurocritical Care and Stroke, University of California San Francisco, San Francisco, CA
1997-2001	Resident, Combined Internal Medicine and Neurology Program, University of Arizona Health Sciences Center, Tucson, AZ
1996-1997	Intern in Medicine, Columbia-Presbyterian Medical Center, New York, NY

Medical Board Certification:

2012	Board Certification Neurocritical care - current
2009	Board Certification Vascular Neurology- current
2005	Board Certified Critical Care Medicine- current
2003	Diplomat, American Board of Psychiatry and Neurology, current, recertified 2/13
2001	Board Certified Internal Medicine, expired

Licensure:

2011	Colorado State Medical License- Active
2011	Nebraska State Medical License- Active
2004	New York State Medical License- Inactive
2001	California Medical License- Active
2000	Arizona Medical License- Active
1997	USMLE Part III
1995	USMLE Part II
1994	USMLE Part I

Employment:

2011-present	Neuro Critical Care and Stroke Neurology, Cedars Sinai Medical Center Private Practice
2011- present	Banner Health Arizona, Telemedicine ICU per diem, Santa Monica, CA
2007-2011	Director of Neuro Critical Care and Director of Stroke Program, Cedars-Sinai Medical Center

2006-2007	Director of Neurocritical Care and Stroke Services, Sutter East Bay Neuroscience Center, Eden Medical Center, Castro Valley, CA
2005-2006	Assistant Clinical Professor of Neurology, Columbia University, New York, NY
2004	Critical Care Attending, Kaiser Permanente Vallejo, CA
2001-2003	Urgent Care, Veteran Affairs Medical Center, San Francisco, CA
1992-1992	Research Assistant. Imarx Pharmaceutical Company, Tucson, AZ
1990-1991	Research Assistant, Department of Radiology, University of Arizona College of Medicine, Tucson, AZ
1990	Internship, Public Defenders Office, Tucson, AZ
1988-1990	Primary Data Collector, Department of Respiratory Sciences, University of Arizona College of Medicine, Tucson, AZ

Volunteer Activities:

2009	Teaching, Indiana Institute for Global health, Moi University Hospital, Eldoret Kenya
2004 and 2003	Neurology Instructor, Health Volunteers Overseas, Uganda
1992-1995	Co-Chair, Students for a Broader Perspective
1992-1993	Member, Health Policy Student Interest Group (HPSIG)
1992-1994	Nogales Pediatric Clinic, Commitment to Underserved People (CUP)
1992-1995	Refugee Clinic, CUP

Awards and Honors:

2010	Friends of Nursing Award, Cedars Sinai Medical Center
2001	Dean's List for Excellence in Teaching by a House Officer, University of Arizona College of Medicine
2000	Chief Resident, Department of Neurology, University of Arizona College of Medicine
1999	House Officer Educator of the Year Award, University of Arizona College of Medicine; Alpha Omega Alpha Honor Medical Society
1998	Dean's List for Excellence in Teaching by a House Officer, University of Arizona College of Medicine
1988-1992	Dean's List, Honorable Mention, University of Arizona
1996	Neurology Medical Student of the Year; Phillip Dew Award for Excellence in Primary Care; Merck Manual Award for Individual Accomplishment, co-winner
1995	Class Rank 2 of 100, Objective Structured Clinical Examination (OSCE). University of Arizona College of Medicine
1992-1996	Academic Honors in the following courses: Behavioral Neurology; Epilepsy; Internal Medicine; Psychiatry; Neurology; Surgery Subspecialties; Pediatrics; Family and Community Medicine; Preparation for Clinical Medicine; Gross Anatomy; Molecular and Cell Biology; and Histology, University of Arizona College of Medicine

Professional Societies:

2006-present	American Academy of Neurology
2006-present	American Heart Association
2003-present	Neurocritical Care Society
2002-present	Society of Critical Care Medicine

Teaching:

2007-present	Attending physician, Department of Neurology and Neurosurgery, Cedars-Sinai Medical Center
2005-2006	Assistant Clinical Professor of Neurology, Columbia University College of Physicians and Surgeons
2004 and 2003	Neurology Instructor, Mulago University Hospital, Kampala, Uganda
2002	Instructor, Fundamental Critical Care Support Course, Society of Critical Care Medicine

2002	ACLS Instructor, American Heart Association
1998	Small Group Leader, Introduction to Neurosciences Course for 1st Year Medical Students
1995	Clinical Case Study Facilitator, 3rd Year Medical Student Neurology Clerkship
1995	Instructor, Physical Diagnosis Course for 1st Year Medical Students

Committees:

2011	National Quality Forum: Cardiovascular/ Diabetes Technical Advisory Panel, Arlington VA
2010-present	ELOS committee, Cedars-Sinai Medical Center
2009-present	Neurology Residency Committee, Dept. of Neurology Cedars Sinai Medical Center
2009	AHA region IV Brain 2 Peer Review Committee
2009-Present	Board member, World Neurology Foundation
2008-2009	Co-Chair Critical Care Committee, Cedars-Sinai Medical Center
2008	National Quality Forum: National Voluntary Consensus Standards for the Prevention and Management of Stroke Across the Continuum of Care, Steering committee, Arlington VA
2008-present	Neurointerventional Oversight Committee, Cedars-Sinai Medical Center
2007-2009	Neuro Critical Care Implementation Committee, Cedars-Sinai Medical Center
2007-present	Critical Care Committee, Cedars-Sinai Medical Center
2005-2006	Quality Assurance Committee, New York Presbyterian Hospital, Columbia University Medical Center

Research Experience:

2010-2011	Co-Investigator: ARgatroban T-PA Stroke Study – ARTSS, multi-center trial, NIH funded
2010-Present	A phase II, dose escalation, single center study on the effects of early propranolol on heart rate, blood pressure, and cerebral perfusion pressure in subjects who present with moderate to severe traumatic brain injury, Cedars Sinai Medical Center
2010-2011	Co-Investigator: Post-Traumatic Vasospasm: A prospective study looking at the outcomes from hemodynamically significant post-traumatic vasospasm detected by transcranial Doppler ultrasound (TCD) and Diffusion Weighted (WD)/Hemodynamically-Weighted (HW) MRI
2010-2011	Co-Investigator: Clot Lysis: Evaluating Accelerated Resolution of Intraventricular Hemorrhage Phase III, multi-center trial, NIH funded
2010-2011	Co-Investigator: SAPPHIRE registery
2008-2011	Neurology site PI, Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis (SAMMPRIS), multi-center trial, NIH funded
2007	Site PI, The Insulin Resistance Intervention after Stroke trial (IRIS), multi-center trial, NIH funded
2007	Site PI, Prevention regimen for effectively avoiding second strokes (PRoFESS), multi-center trial, Boehringer Ingelheim
2005	PI, Family conflict and withdrawal of care, Critical Care Neurology, Columbia University
2005	PI, Family conferences and the extent of education regarding family member's care and prognosis, Critical Care Neurology, Columbia University
2002	Heat shock proteins 60 and 72 in post-traumatic CSF samples, Department of Anesthesia, University of California San Francisco (PI: JF Pettit, MD)
1993	*Emergency department geriatric patient survey*, Department of Emergency Medicine, University of Arizona College of Medicine (PI: Elaine Roussesau, PhD)
1992	*Collective action case study: The African National Congress (ANC)*, Senior Undergraduate Independent Study, Department of Political Science, University of Arizona (Advisor: Paul Buchanan, PhD)
1990-1992	*MRI oral GI contrast agent development*, Department of Radiology, University of Arizona College of Medicine and Imarx Pharmaceutical Company (PI: Evan Unger, MD)
1990-1992	*MRI and Ultrasound oral and Intravenous GI contrast agent developmentt using Novel Polymer Contrast Agent, and Liposomal Contrast Delivery Systems*. Department of

	Radiology, University of Arizona College of Medicine. Imarx Pharmaceutical Company. (PI: Evan Unger, MD).
1988-1990	*Indoor Air Pollution Epidemiological Study*. Department of Respiratory Sciences, University of Arizona College of Medicine.

Independent Reviewer:

2011- present	Medical Legal expert witness and IME
2010- present	Expert Reviewer, Medical Board of California
2007- present	Stroke and Neuro Critical Care program evaluations

Selected Invitational Presentations:

1. **Palestrant D.** Update in Neurocritical Care, Society of Hospitalist Medicine, San Diego, CA April 2012
2. **Palestrant D.** Hypothermia after Cardiac Arrest, Cardiology Grand Rounds, Cedars Sinai Medical Center, Los Angeles, CA, Feb 2012f
3. **Palestrant D.** Acute Stroke Treatment-Best Data for Treatment Algorithms, Symposium on Neurovascular and Carotid Artery Disease 2011, Santa Monica, CA Jan 2011
4. **Palestrant D.** Neuro ICU Finances, Neuro Critical Society Annual Meeting, San Francisco, Sept. 2010
5. **Palestrant D.** Hypothermia, Pulmonary and Critical Care Conference, Providence Medical Center, Seattle WA, Sept 2010
6. **Palestrant D.** Neuro Monitoring, Pulmonary and Critical Care Conference, Providence Medical Center, Seattle WA, Sept 2010
7. **Palestrant D.** Building a Stroke Program, AHA GWTG Executive Leadership Event, Palm Springs CA April 2010
8. **Palestrant D.** TIA is a medical Emergency, New Data New Options, Advances in Neurology and Neurosurgery, Marina Del Rey, Feb 21010
9. **Palestrant D.** TIA-Evaluation and Work-up, Symposium on Neurovascular Disease, Santa Monica, Ca Jan 2010
10. **Palestrant D.** Advances in Neurointensive care, Symposium on Neurovascular Disease, Santa Monica, Ca Jan 2010
11. **Palestrant D.** Intracranial Monitoring To Improve Patient Outcomes, Rehabilitation Topics in the Management of Brain Injuries, Los Angeles, CA September 2009
12. **Palestrant D.** Pathophysiology of Traumatic Brain Injury, Rehabilitation Topics in the Management of Brain Injuries, Los Angeles, CA, September 2009
13. **Palestrant D.** Intracranial Monitoring To Improve Patient Outcomes, CSMC Neurosurgery Grand Rounds, Los Angeles, CA, September 2009
14. **Palestrant D.** Cerebral PbO2 Monitoring, Society of Neurointerventional Surgery, Boca Rotan, Florida, July 2009
15. **Palestrant D.** Neurologic Evaluation of Neurosurgery ICU Patients, SICU Teaching Conference, Cedars-Sinai Medical Center, Los Angeles, CA, July 2009
16. **Palestrant D.** Therapeutic Hypothermia For The ED, ED Grand Rounds - Cedars-Sinai Hospital, Los Angeles, CA, July 2009
17. **Palestrant D.** Recurrent Stroke Prevention (National Stroke Association.), Stroke Neurology Grand Rounds, Cedars-Sinai Hospital, Los Angeles, CA, May 2009
18. **Palestrant D.** Stroke: Code Brain The Cedars-Sinai Medical Center Stroke Program, Cedars-Sinai Medical Center, Los Angeles, CA, May 2009
19. **Palestrant D.** Update on Management of TIAs, Advances in Neurology & Neurosurgery, Marina del Rey, CA, February 2009
20. **Palestrant D.** Massive Stroke: Medical Management vs Hemicraniectomy, Advances in Neurology & Neurosurgery, Marina del Rey, CA, February 2009
21. **Palestrant D.** Neurologist View of International Work in Africa, Advances in Neurology & Neurosurgery, Marina del Rey, CA, February 2009
22. **Palestrant D** Stratifying Stroke Patients for Treatment in the Emergency Department, Cedars-Sinai Medical Center Symposium on Neurovascualr Disease 2009, Santa Monica CA, January 10 2009

23. **Palestrant D** Development of Stroke Centers: Resources, Patient Care and Outcomes, Cedars-Sinai Medical Center Symposium on Neurovascualr Disease 2009, Santa Monica CA, January 10 2009
24. **Palestrant D** Brain tissue oxygen monitoring and cerebral microdialysis, Pulmonary Critical Care Grand Rounds, Cedars Sinai Medical Center, November 2008
25. **Palestrant D** Acute Stroke Therapy, West Africa Medical Conference, Liberville, Gabon, November 2008
26. **Palestrant D** Management of Massive Stroke, Division of Neurology Grand Rounds, Cedars Sinai Medical Center, July 2008
27. **Palestrant D** Update on Acute Stroke Management at CSMC, Dept. of Medicine Grand Rounds, Cedars Sinai Medical Center, August 2008
28. **Palestrant D.** Neuro-Critical Care of Massive Stroke, Get with the Guidelines Workshop, American Stroke Association, Cedars-Sinai Medical Center, Los Angeles CA, May 2008
29. **Palestrant D.** Cerebral PbO2 and Microdialysis, Strictly Trauma Conference, Monterey CA, October 2006
30. **Palestrant D.** Cerebral Microdialysis and Multimodality Neuro Monitoring, Neurosurgical Critical Care Course, Congress of Neurological Surgeons, Chicago, IL, October 2006
31. **Palestrant D.** Mechanical Ventilation. 3rd New York Symposium on Neurological Emergencies and Neurocritical Care, New York, NY, May 2006
32. **Palestrant D.** Acute and ICU Management of Massive Stroke. Cerebrovascular Critical Care Course, Congress of Neurological Surgeons, Boston, MA, October 2005
33. **Palestrant D.** Hypothermia for Massive Stroke. Neurology Grand Rounds, Mayo Clinic Scottsdale, Scottsdale, AZ, July 2005
34. **Palestrant D.** B12 Deficiency. Internal Medicine Grand Rounds, Makerere University Medical School, Kampala Uganda, November 2003
35. **Palestrant D.** Update in ICP Management. Neurology/Neurosurgery Grand Rounds, University of California Irvine, Irvine, CA, March 2003
36. **Palestrant D.** Update in ICP Management. Clinical Stroke Conference, Stanford University, Palo Alto, CA, January 2003
37. **Palestrant D.** Aggressive Risk Reduction Strategies in Cerebrovascular Diseases. Hong Kong Stroke Symposium and Workshop 2002, Hong Kong, Republic of China, December 2002
38. **Palestrant D.** Update on Acute Stroke Therapy. Hong Kong Stroke Symposium and Workshop 2002, Hong Kong, Republic of China, December 2002
39. **Palestrant D.** Neurological Critical Care. Fundamentals of Critical Medicine (FCCM) course, University of California San Francisco, San Francisco, August 2003
40. **Palestrant D.** Uremic Encephalopathy. Grand Rounds, Department of Neurology, University of Arizona, Tucson AZ, April 2001

Original Papers:

1. Mukherjee D, Patil CG, **Palestrant D.** Amantadine for severe traumatic brain injury. N. Engl. J. Med. 2012Jun.21;366(25):2427-authorreply2427-8.

2. Miller CM, **Palestrant D.** Distribution of delayed ischemic neurological deficits after aneurysmal subarachnoid hemorrhage and implications for regional neuromonitoring. Clin Neurol Neurosurg. 2012Jul.;114(6):545-9.

3. Drazin D, Jeswani S, Shirzadi A, Choulakian A, Alexander MJ, **Palestrant D**, et al. Anterior Spinal Artery Syndrome in a Patient with Vasospasm Secondary to a Ruptured Cervical Dural Arteriovenous Fistula. J Neuroimaging. 2011Dec.30.

4. Miller CM, **Palestrant D**, Schievink WI, Alexander MJ. Prolonged transcranial Doppler monitoring after aneurysmal subarachnoid hemorrhage fails to adequately predict ischemic risk. Neurocrit Care. 2011Dec.;15(3):387-92.

5. Rincon F, Gordon E, Starke RM, Buitrago MM, Fernandez A, Schmidt JM, Claassen J, Wartenberg KE, Frontera J, Seder DB, **Palestrant D**, Connolly ES, Lee K, Mayer SA, Badjatia N. Predictors of long-term shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. J Neurosurg. 2010 Apr 2.
6. Ley EJ, Park R, Dagliyan G, **Palestrant D**, Miller CM, Conti PS, Margulies DR, Salim A. In vivo effect of propranolol dose and timing on cerebral perfusion after traumatic brain injury. J Trauma. 2010 Feb;68(2):353-6.
7. Schievink WI, **Palestrant D**, Maya MM, Rappard G. Spontaneous spinal cerebrospinal fluid leak as a cause of coma after craniotomy for clipping of an unruptured intracranial aneurysm. J Neurosurgery Nov 14, 2008
8. Badjatia N, Kowalski RG, Schmidt JM, Voorhees ME, Claassen J, Ostapkovich ND, Presciutti M,Connolly ES, **Palestrant D**, Parra A, Mayer SA. Predictors and clinical implications of shivering during therapeutic normothermia Neurocrit Care. 2007;6(3):186-91.
9. Wartenberg KE, **Palestrant D**. Cerebral Sinus Thrombosis. ArchNeurol. 2006 Sep; 63 (9):1332
10. Frontera JA, **Palestrant D**. Acute trismus associated with Foix-Marie-Chavany syndrome. Neurology. 2006 Feb 14;66(3):454-5.
11. **Palestrant D**, Frontera J, Mayer S. Treatment of Massive Stroke. Curr Neurol Neurosci Rep. 2005 Nov;5(6):494-502
12. Connolly ES Jr, Lavine SD, Meyers PM, **Palestrant D**, Parra A, Mayer SA. Intensive care unit management of interventional neuroradiology patients. Neurosurg Clin N Am. 2005 Jul;16(3):541-5, vi.
13. A. A. Jarquin-Valdivia, M.D., R.D.M.S., J. McCartney, R.V.T., **D. Palestrant, M.D.,** S. C. Johnston, M.D., PhD., D. Gress, M.D.: The Thickness of the Temporal Squama and its Implication for Transcranial Sonography. Journal of Neuroimaging, accepted
14. Shen D, Fritz T, Wu G, Kulik B, **Palestrant D**, and Unger E. Block co-polymeric magnetic resonance contrast agents. *Investigative Radiology*, 1994, *Suppl. 29*, 217-219.
15. Unger E, Fritz T, **Palestrant D**, Mekam T, and Granstrom P. Preliminary evaluation of iron phytate (inositol hexaphosphate) as a gastrointestinal MR contrast agent. *Journal of Magnetic Resonance Imaging*, 1993, *3(1)*, 119-124.

Book Chapters:

1. **David Palestrant MD,** Management of Traumatic Brain Injury, Neurohospitalist Medicine, release date November 2011,Cambridge University Press, September 2011
2. **David Palestrant MD,** Stroke Pocketcard Set, 2009, Born Bruckmeier Pub
3. **David Palestrant MD,** E. Sander Connolly JR, Subarchnoid Hemorrhage, Neurobiology of disease, 2007 Elsevier Academic Press, P. 265-270
4. **David Palestrant MD**, David Bonovich MD, Cerebral Aneurysms, Critical Care Secrets Third Edition, 2003 Hanley and Belfus INC. P 337-341.
5. **David Palestrant MD**, David Bonovich MD, Head Trauma, Critical Care Secrets Third Edition, 2003 Hanley and Belfus INC. P 355-361.

Conference Proceedings and Abstracts:

1. Katja Wartenberg, Michael Schmidt, Andres Fernandez, Jan Claassen, Noeleen Ostapkovich, Neeraj Badjatia, **David Palestrant**, Augusto Parra, Stephan Mayer. Multi territorial Symptomatic Vasospasm after Subarachnoid Hemorrhage: Predictors, Associated Complications, and Impact on Outcome. Oral Abstract Presentation, International Stroke Conference, Feb, 2007
2. Jennifer Frontera, Michael Schmidt, Andres Fernandez, Neeraj Badjatia, **David Palestrant**, Augusto Parra, Stephan Mayer. Symptomatic Vasospasm: Predictors and Effect on Outcome After Subarachnoid Hemorrhage. Poster Abstract Presentation, International Stroke Conference, Feb, 2007
3. Neeraj Badjatia, Michael Schmidt, Augusto Parra, **David Palestrant**, Jennifer Frontera, Katja Wartenberg, Noeleen Ostopkovich, ES Connolly, Stephan Mayer. Prediction of cognitive impairment and quality of Life after subarachnoid hemorrhage with the Short Orientation-Memory-Concentration test. Poster abstract presentation, International Stroke conference, Feb 2006

4. Unger E, Shen D, Wu G, Fritz T, Rose C, New T, and **Palestrant D.** Novel alkylated complexes for MR contrast agents. Presented at the Society of Magnetic Resonance in Medicine 10th Annual Meeting, August 10-16, 1991.
5. Fritz T, Unger E, New T, Kulik B, **Palestrant D**, and Tilcock C. Clearance of liposomal gadolinium complexes: In vivo decomplexation studies. Presented at the Society of Magnetic Resonance in Medicine 10th Annual Meeting, August 10-16, 1991.
6. Unger E, Shen D, Wu G, Fritz T, Kulik B, **Palestrant D**, Blatner G, Crowell M, and New T. Nanogels: Novel paramagnetic particulate MR contrast agents. Presented at the Society of Magnetic Resonance in Medicine 10th Annual Meeting, August 10-16, 1991.